Atlas on
Clinical
Ophthalmology

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Samar K Basak
MD DNB
Disha Eye Hospitals and Research Centre
Barrackpore, Kolkata 700120
West Bengal, India
e-mail: disha@cal2.vsnl.net.in
Phone: 91-33-25946193 Fax: 91-33-25928106

JAYPEE BROTHERS
MEDICAL PUBLISHERS (P) LTD
New Delhi

Published by

Jitendar P Vij
Jaypee Brothers Medical Publishers (P) Ltd
EMCA House, 23/23B Ansari Road, Daryaganj
New Delhi 110 002, India
Phones: +91-11-23272143, +91-11-23272703, +91-11-23282021, +91-11-23245672
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  Nagpur 440 009, Phone: +91-0712-3945220 e-mail: jpnagpur@rediffmail.com

Atlas on Clinical Ophthalmology

First Edition: **2006**

ISBN 81-8061-645-2

Typeset at JPBMP typesetting unit
Printed at Gopsons Papers Ltd.

to

the *three* of

"The Four Pillars of DISHA"

Dr Debasish Bhattacharya

for his leadership and vision

Dr Tushar K Hazra

for his dedication and knowledge

and

Dr Tushar K Sinha

for his enthusiastic support in lighter moods

Acknowledgements

I am extremely grateful to the following colleagues and friends all over India for their contribution of some excellent photographs which have been included in different chapters of this book.

Alay S Banker, Banker's Retina Clinic and Laser Centre, Ahmedabad, Gujarat
FIGURES: *1.30.1, 2.42.1, 12.18.6, 12.31.1, 12.31.3, 12.31.4 and 12.33.1*

Arnab Biswas, Anandalok Hospital, Kolkata, West Bengal
FIGURES: *1.1.2, 1.6a.1, 1.6a.2, 1.21.1, 1.22.4, 1.30.2, 1.39.1, 1.53.4, 1.57.1, 1.57.3, 1.61.1, 1.67.1, 1.67.2, 1.68.3, 1.68.4, 1.69.1, 1.70.3, 1.72.4, 2.21.10, 2.33.1, 2.34.1, 2.35.2, 2.44.3, 12.2.1, 14.1.8, 14.2.2, 14.3.3, 14.6.5, 14.8.1, 14.10.1, 14.10.2, 14.11.3, 14.13.2, 14.13.3, 14.15.1 to 14.15.3, 14.23.5 to 14.23.8, 14.24.1, 14.24.3, 15.15.4 and 15.15.5*

Arup Bose, EMLC, Chinsura, Hooghly, West Bengal
FIGURES: *1.19.1, 1.75.1, 2.42.4, 3.64.6, 3.64.7, 6.13.2 and 8.46.10*

Daljit Singh, Daljit Singh Eye Hospital, Amritsar, Punjab
FIGURE: *7.7.12*

Debdas Mukherjee, BKG Eye Institute, Malda, West Bengal
FIGURE: *3.71.5*

Kasturi Bhattacharya, Sri Sankaradeva Nethralaya, Guwahati, Assam
FIGURES: *14.5.1, 14.11.1, 14.12.2, 14.15.2, 14.15.6, 14.22.4, 14.26.1 and 14.26.2*

M Srinivasan, Aravind Eye Care System, Madurai, Tamil Nadu
FIGURES: *3.7.1, 3.14.8 and 3.18.1*

Pak Sang Lee, Institute of Ophthalmology, University College of London, London, UK
FIGURE: *2.10.2*

Paramita Sanatani, Athena Eye Care, Kolkata, West Bengal
FIGURES: *1.39.3, 12.30.1 to 12.30.3, 15.3.1, 15.11.3, 15.11.4, 15.12.2, 15.17.1, 15.17.2, 15.19.1 and 15.20.1*

Partha Biswas, BB Eye Foundation, Kolkata, West Bengal
FIGURES: *7.9.2 and 8.37.9*

Rajesh Fogla, Apollo Eye Hospital, Hyderabad, Andhra Pradesh
FIGURES: *3.18.3 and 16.15.4*

Rakhi Bandyopadhayay, Regional Institute of Ophthalmology, Kolkata, West Bengal
FIGURES: *1.41.8, 14.8.3, 14.14.1, 14.23.1 to 14.23.3 and 14.23.10*

Sarbjit Singh, Shamsher Singh Eye Hospital, Khanna, Punjab
FIGURES: *5.9.9, 8.42.7 and 12.3.1*

Santosh Honavar, LV Prasad Eye Institute, Hyderabad, Andhra Pradesh
FIGURES: *7.9.4, 12.69.1 to 12.69.4 and 14.3.1*

Community Eye Health Journal, Vol.18, No. 53, March 2005, International Centre for Eye Health, London School of Hygiene and Tropical Medicine, London, UK
FIGURES: *2.10.2, 2.15.1 and 2.15.2*

Eyetech Industries, Kolkata, West Bengal
FIGURE: *6.2.6*

Implant House, Kolkata, West Bengal
About 50 percent of the FIGURES in Chapter—12

Susrut Eye Foundation & Research Centre, Kolkata, West Bengal
FIGURES: *1.22.7, 1.22.8, 12.9.3, 12.17.5, 12.17.6, 12.18.1 to 12.18.5, 12.51.1 to 12.51.4, 12.74.1 and 12.74.2*

Secretarial Assistance

I also express my sincere thanks to the following staff of our hospital for helping me time-to-time actively to procure the photographs and other technical assistance during compilation of this book.

Tuhin Nandy **Mita Chakraborty**
Utpal Sarkar **Debabrata Maji**

Preface

Today, ophthalmic sciences are developing at an incredibly rapid pace. In the era of subspecialty and superspecialty in fields of ophthalmology, it is very difficult to take the role of a comprehensive ophthalmologist. Indeed, it is now lacking all over the world. Even for the specialist ophthalmologist, clinical diagnosis is important for the different parts of the eye other than his/her area of interest. On the other hand, for a comprehensive ophthalmologist, provisional clinical diagnosis is very important for referral services of his/her patients to a specialist ophthalmologist.

This book is intended to create an ophtha-photographic bridge between the common ophthalmologist and the specialist ophthalmologist. It deals with various common and uncommon ophthalmic diseases and disorders through beautiful color photographs.

The structure of this book is unique. Each chapter deals with a single part of the eye. The book starts from the diseases of the eyelids, penetrates deeper and deeper, gradually progresses towards the diseases of the retina and ends with ocular injuries. There are concise textual outlines of major and minor signs side-by-side for each disease or disorder.

I am optimistic and hopeful that this book will be a useful companion to the postgraduate trainees, fellow ophthalmologists, comprehensive ophthalmologists as well as specialist ophthalmologists. It will serve as a ready reference during most of the clinical situations in a busy clinic. I must confess that there may be some omission and commission on my part during compiling and editing the photographs. And I hope these lacunae will be corrected in future with due suggestions and criticism from the students, teachers and colleagues from different parts of the world.

I extend my sincere gratitude to my friend, Dr Debasish Bhattacharya, Director, Disha Eye Hospitals and Research Centre, who has silently approved and supported me from his heart for writing this book. I also take this opportunity to acknowledge all my wonderful colleagues and associates of my institute for their constant support and encouragement during collection of photographs for this voluminous book.

I gratefully acknowledge my old sweetheart Dr Bani and my lovely children, Soham and Sohini, for their abundant support, for their patience and understanding, and for their caring love during the rigors of completing this book. Finally, I express my sincere gratitude to my publisher and all his technical staffs for helping me in the publication of this wonderful book.

Samar K Basak

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- Depigmentation of periocular skin
- Ankyloblepharon
- Nodular hemangiomas

CONGENITAL LID CONDITIONS

Epicanthus

- *Most common* congenital lid condition
- Unilateral or bilateral
- May give rise to *pseudo-convergent squint*
- Semilunar vertical skin folds at the medial canthi running between two eyelids **(Fig 1.1.1)**
- Four types:
  - *epicanthus tarsalis:* fold is most prominent in the upperlid **(Fig 1.1.2)**
  - *epicanthus palpebralis:* skin fold in equally distributed in the upper and lower eyelids **(Fig 1.1.3)**
  - *epicanthus inversus:* fold is most prominent in the lower eyelid **(Fig 1.1.4)**
  - *epicanthus supraciliaris:* skinfold covering the medical canthus, extends high up to the eyebrow
- *Treatment:* by plastic repair

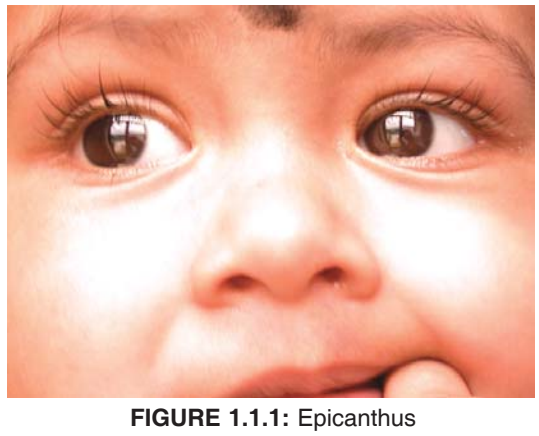


FIGURE 1.1.1: Epicanthus

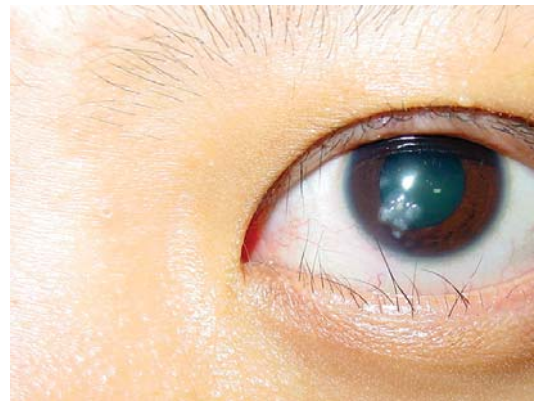


FIGURE 1.1.2: Epicanthus tarsalis

FIGURE 1.1.3: Epicanthus palpebralis

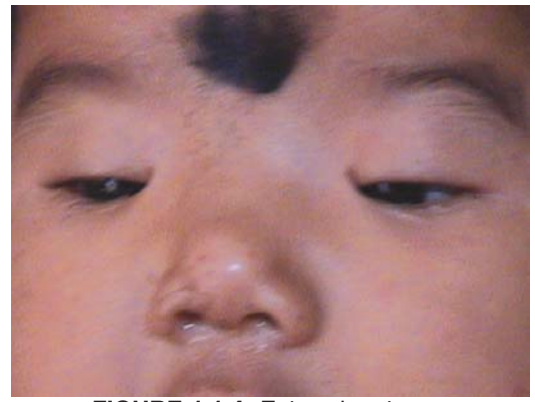


FIGURE 1.1.4: Epicanthus inversus

Telecanthus

- Increased distance between medial canthi (**Fig 1.2.1**)
- due to long abnormal medial canthal tendon
- May also give rise to *pseudo-convergent squint* (**Fig 1.2.2)**
- *Should not be confused* with hypertelorism (**Fig 1.2.3**) in which there is wide separation of bony orbits

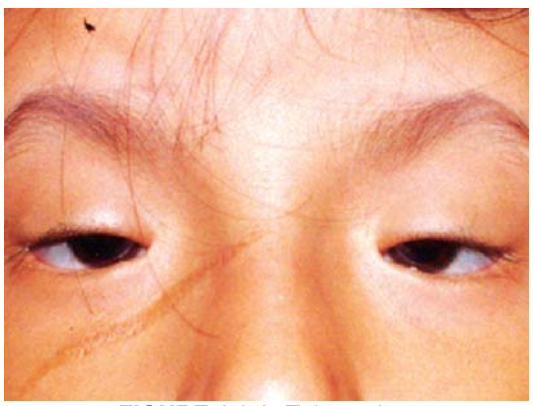


FIGURE 1.2.1: Telecanthus

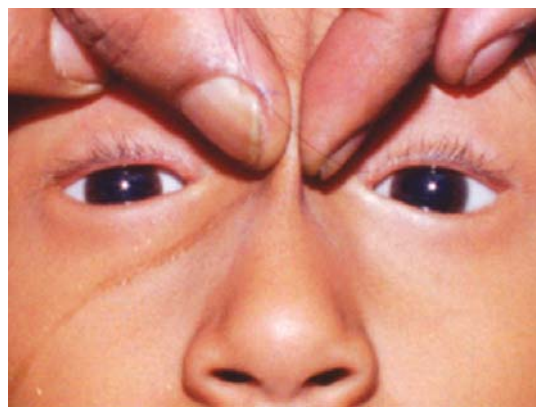


FIGURE 1.2.2: Telecanthus-pseudo-convergent squint

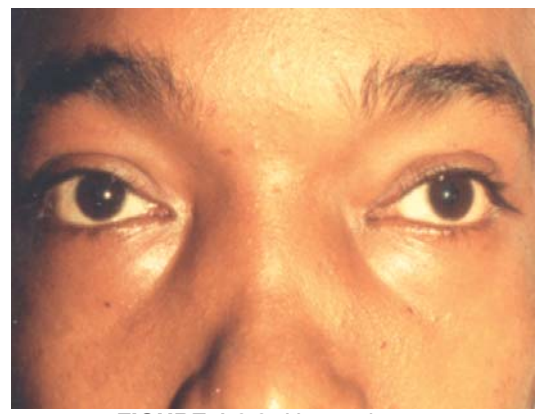


FIGURE 1.2.3: Hypertelorism

Coloboma of the Eyelid

- A notch or defect of the lid margin
- Unilateral (**Fig 1.3.1**) or bilateral (**Fig 1.3.2**); upper or lower
- *Upper lid coloboma*: at the junction of middle and inner thirds
- Not associated with systemic defects
- *Lower lid coloboma*: at middle and outer thirds junction
- Associated with Treacher Collins syndrome (**Fig 1.3.3**)
- May be acquired in traumatic cases (**Fig 1.3.4**)
- *Treatment:* urgent plastic repair at a very early age to prevent exposure keratitis

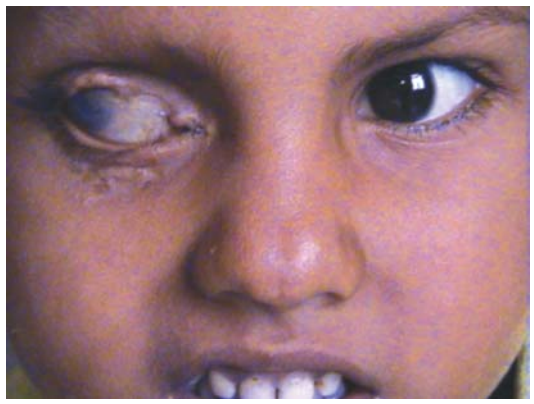


FIGURE 1.3.1: Lid coloboma

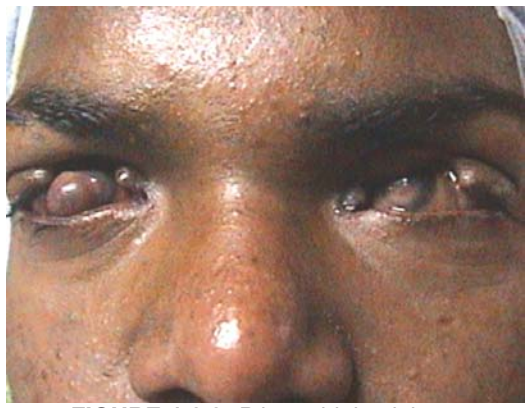


FIGURE 1.3.2: Bilateral lid coloboma

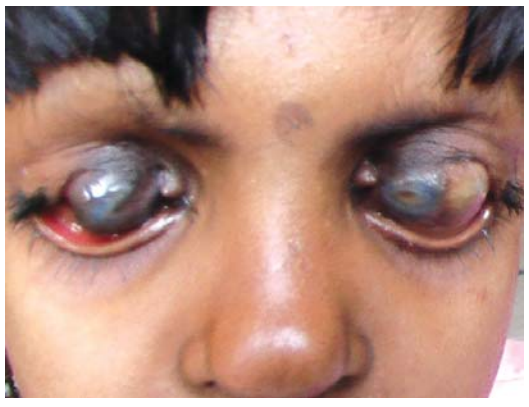


FIGURE 1.3.3: Bilateral lid coloboma

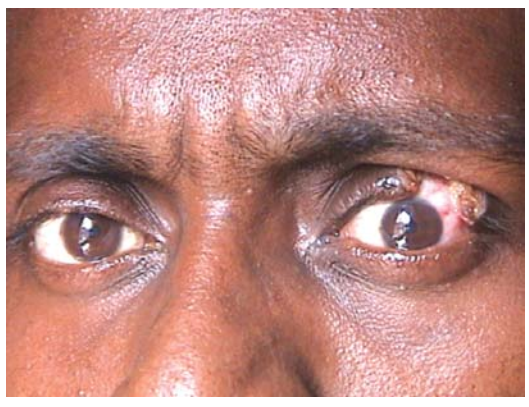


FIGURE 1.3.4: Acquired coloboma of left upper lid

Distichiasis

- Hereditary and congenital condition
- Extra posterior row of cilia, occasionally present in all four lids
- Partial (**Fig 1.4.1**) or complete (**Fig 1.4.2**)
- They occupy the position of meibomian gland orifices
- Eyelashes may irritate to cause corneal epithelial defects
- May be also seen in Stevens-Johnson syndrome—*acquired distichiasis* (**Fig 1.4.3**)
- *Treatment:* by cryotherapy or excision with grafting

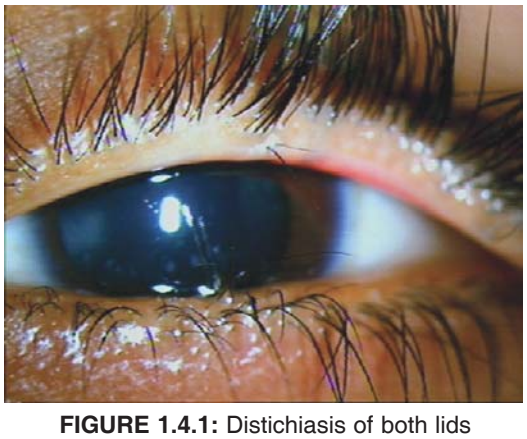


FIGURE 1.4.1: Distichiasis of both lids

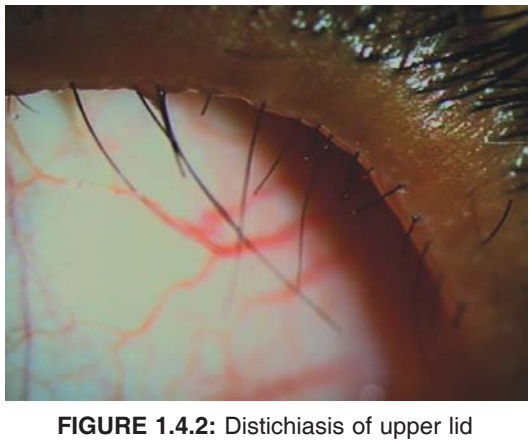


FIGURE 1.4.2: Distichiasis of upper lid

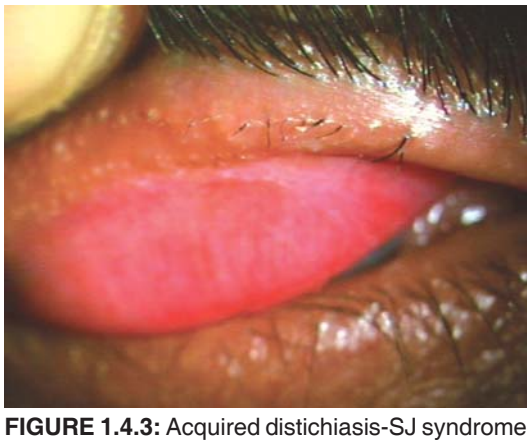


FIGURE 1.4.3: Acquired distichiasis-SJ syndrome

Blepharophimosis Syndrome

- Autosomal dominant **(Fig 1.5.1)**
- Syndrome consists of bilateral (**Fig 1.5.2**)
  - narrowing of vertical and horizontal palperbral apertures
  - telecanthus
  - inverse epicanthus folds
  - lateral ectropion and moderate to severe ptosis
- *Treatment:* plastic reconstruction of lids, along with bilateral brow suspension for ptosis

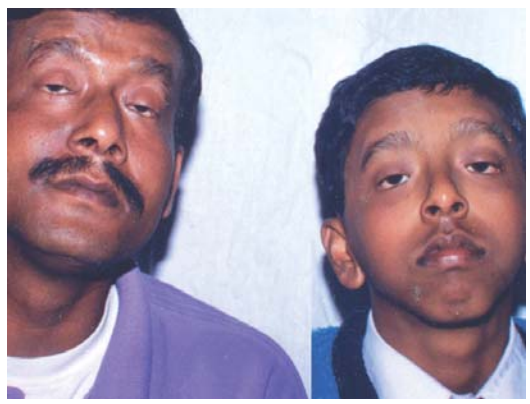


FIGURE 1.5.1: Blepharophimosis syndrome

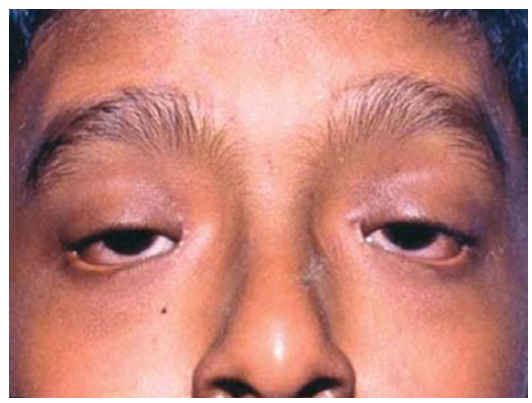


FIGURE 1.5.2: Blepharophimosis syndrome

Epiblepharon

- Extra fold of skin in the lower lid with in turning of eyelashes (**Fig 1.6.1**)
- Nasal 1/3rd is most commonly affected
- *Treatment:* plastic repair if necessary to prevent recurrent infection

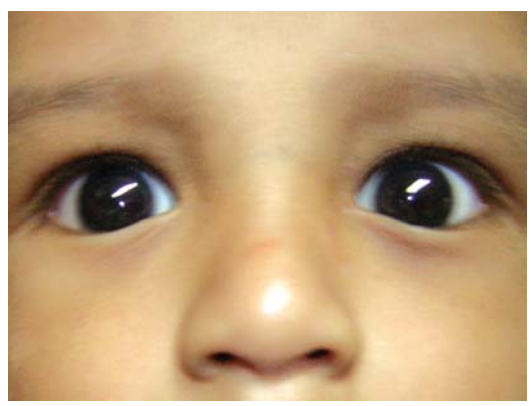


FIGURE 1.6.1: Epiblepharon

Euryblepharon

- Rare, congenital bilateral, not so serious condition
- Palperbral apertures are larger than normal **(Figs 1.6.1a and b)**
- Excessive watering may be a problem due to more exposure
- *Treatment:* no treatment for most of the cases; lateral tarsorrhaphy for symptomatic cases

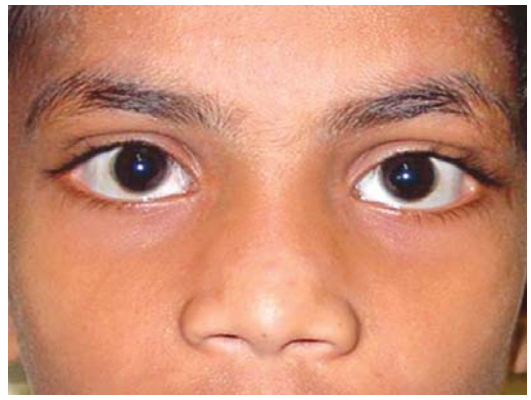


Figure 1.6.1a: Euryblepharon

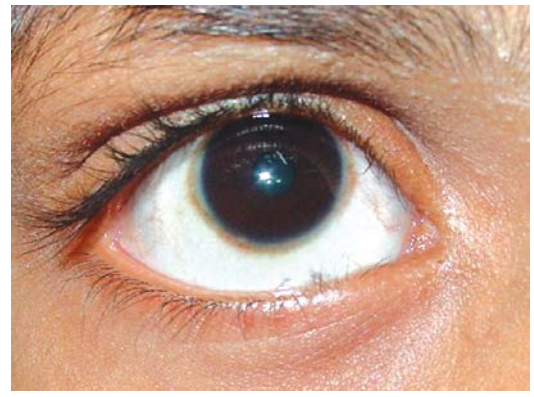


Figure 1.6.1b: Euryblepharon

EYE LASH ABNORMALITIES

Trichiasis

- Inward misdirection of eyelash(es) which irritate the cornea and/or conjunctiva (**Figs 1.7.1 and 1.7.2**)
- When associated with entropion—called *pseudo-trichiasis* (**Fig 1.7.3**)
- *Treatment:* temporarily by epilation; permanently by electrolysis, cryotherapy, or argon laser cilia ablation
  If more cilia are involved: Operative procedure as entropion is most effective

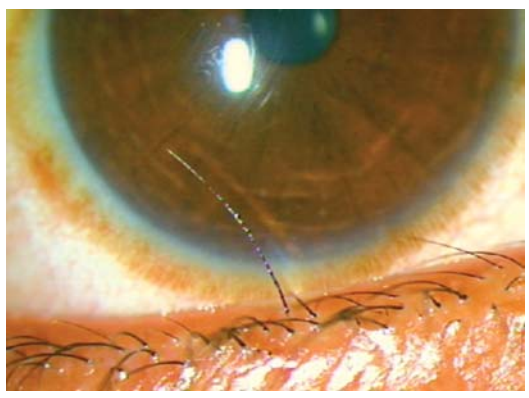


FIGURE 1.7.1: Trichiasis

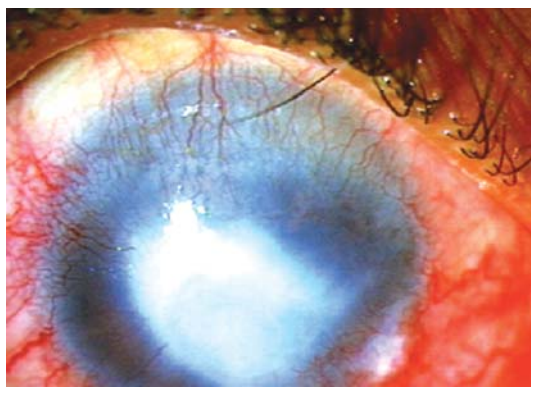


FIGURE 1.7.2: Trichiasis with corneal ulcer

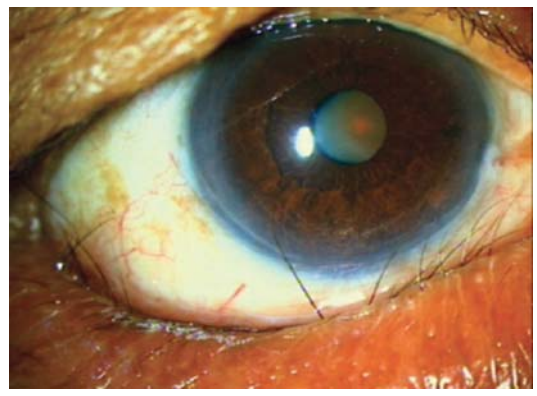


FIGURE 1.7.3: Pseudo-trichiasis in entropion

Eyelash Ptosis

- Downward drooping of upper lid eyelashes (**Fig 1.8.1**)
- Congenital or may be seen after use of prolong latanoprost eye drop

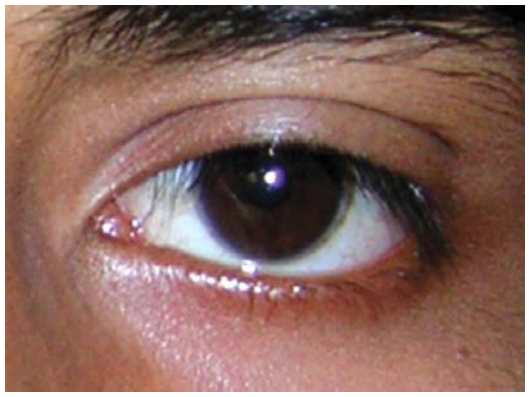


FIGURE 1.8.1: Eyelash ptosis

Lash in the Punctum

- An uncommon phenomenon which may cause a pricking sensation on blinking
- Mostly seen in lower punctum (**Fig 1.9.1**), may be in the upper punctum (**Fig 1.9.2)**
- *Treatment:* simple removal of the offending eyelash

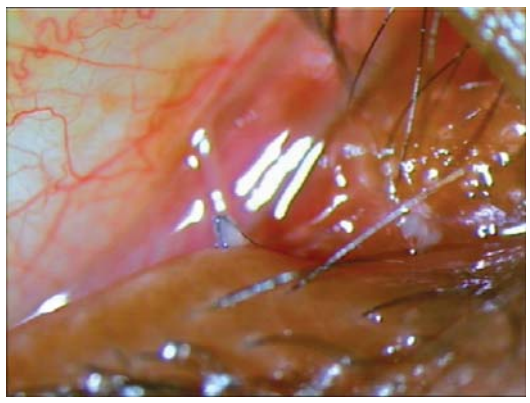


FIGURE 1.9.1: Eyelash in lower punctum

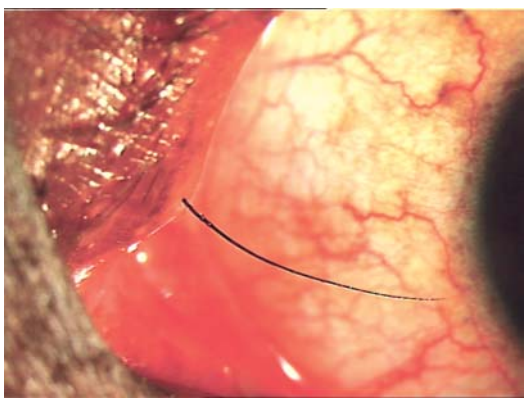


FIGURE 1.9.2: Eyelash in upper punctum

Eyelash in the Anterior Chamber

- Rare occurrence after a penetrating injury (**Fig 1.10.1**)
- May cause iridocyclitis or implantation cyst

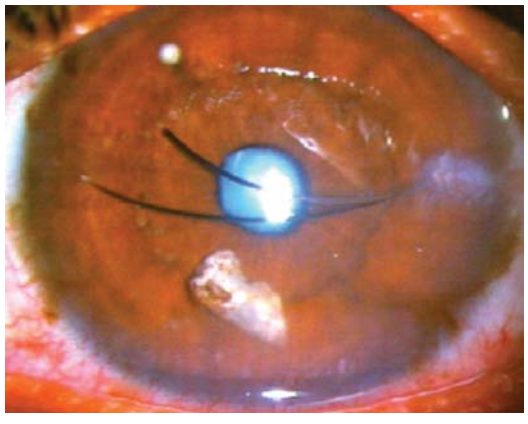


FIGURE 1.10.1: Eyelash in anterior chamber

Metaplastic Eyelash(es)

- Not so rare situation which may cause redness and irritation (**Fig 1.11.1**)
- May be seen in cicatricial lid condition like chemical burn, SJ syndrome, ocular cicatricial pemphigoid or radiational injury
- Can also cause corneal ulcer (**Fig 1.11.2**)
- *Treatment:* removal of eyelash surgically

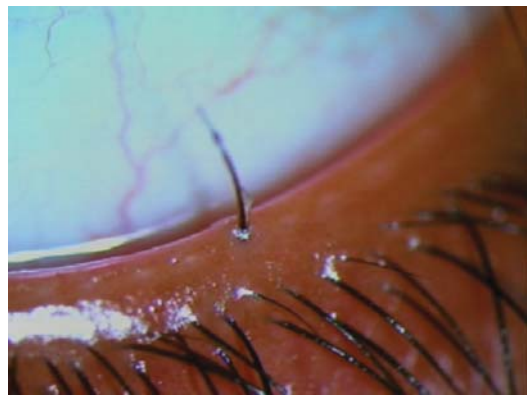


FIGURE 1.11.1: Metaplastic eyelash

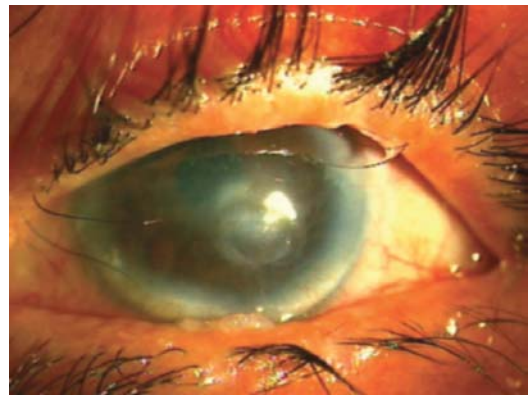


FIGURE 1.11.2: Metaplastic eyelash with healed ulcer

Poliosis

- Whitening of eye lashses; partial or total
- *Causes:*
  - aging (**Fig 1.12.1**)
  - albinism (**Fig 1.12.2**)
  - sympathetic ophthalmia
  - VKH syndrome (**Fig 1.12.3**)
  - Waardenburg syndrome
- *Treatment:* no specific treatment

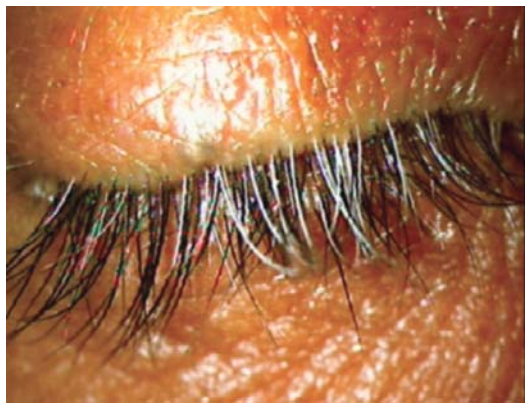


FIGURE 1.12.1: Poliosis-aging

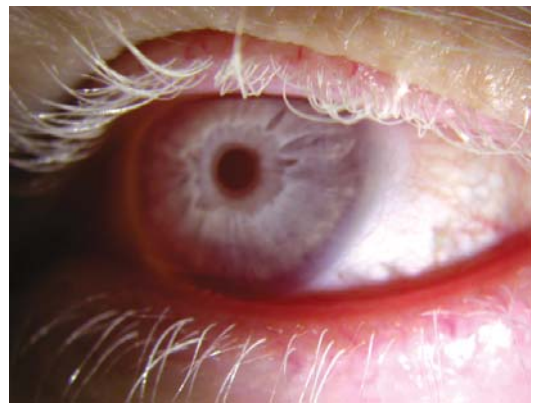


FIGURE 1.12.2: Poliosis-albinism

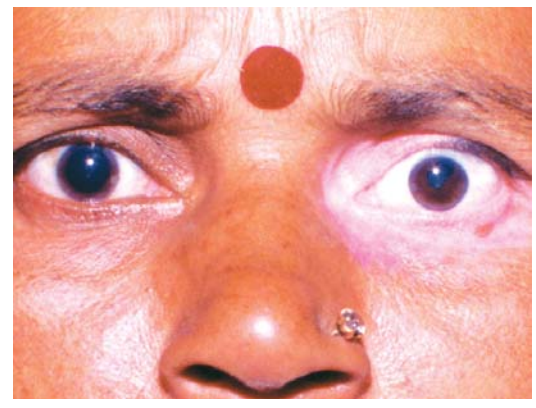


FIGURE 1.12.3: Poliosis-VKH syndrome

Madarosis

- Partial or complete loss of eyelashes
- *Causes:*
  - chronic blepharitis (**Fig 1.13.1**)
  - burns (**Fig 1.13.2**)
  - trichotillomania
  - generalized alopecia
  - myxoedema
  - leprosy (**Fig 1.13.3**)
- *Treatment:* of the cause

FIGURE 1.13.1: Madarosis-blepharitis

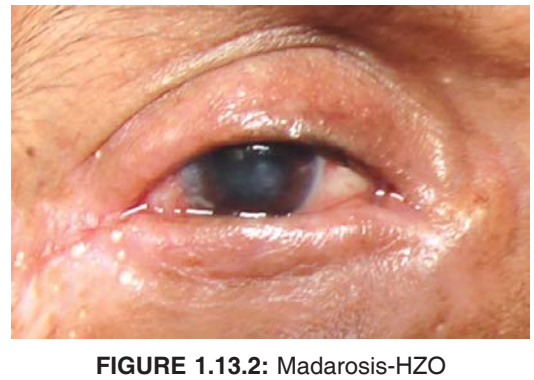


FIGURE 1.13.2: Madarosis-HZO

FIGURE 1.13.3: Madarosis-leprosy

Brittle Eyelash

- Rare, bilateral condition
- Eyelashes break with simple rubbing or with slightest manipulation (**Figs 1.14.1 and 1.14.2**)
- Associated with congenital ectodermal dysplasia

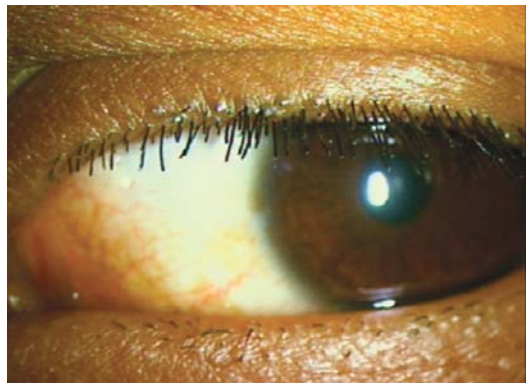


FIGURE 1.14.1: Brittle eyelash

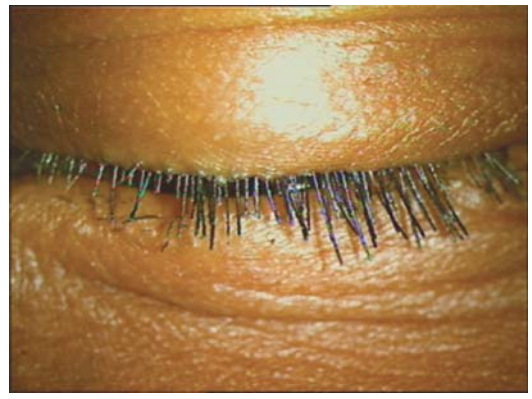


FIGURE 1.14.2: Brittle eyelash

Trichomegaly

- Excessively long and dense luxuriant eyelashes (**Figs 1.15.1 and 1.15.2**)
- May be associated with newer antiglaucoma medication, like bimatoprost
- *No treatment* is required

FIGURE 1.15.1: Trichomegaly

FIGURE 1.15.2: Trichomegaly

Matting of the Eyelashes

- Few eyelashes are stuck together
- Mostly seen in upper eyelids
- *Causes:*
  - acute conjunctivitis (**Fig 1.16.1**)
  - blepharitis (**Fig 1.16.2**)
  - bacterial corneal ulcer (**1.16.3**)
  - use of *eye* ointment
- *Treatment*: of the cause

FIGURE 1.16.1: Matting of lashes-conjunctivitis

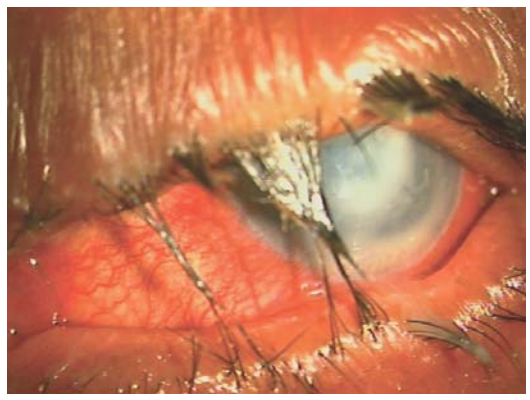


FIGURE 1.16.2: Matting of lashes-blepharitis

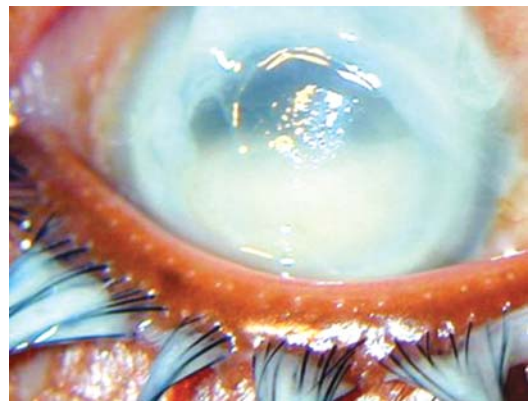


FIGURE 1.16.3: Matting-bacterial corneal ulcer

ENTROPION

Inward turning of the eyelids towards the globe

Congenital Entropion

- Rare, may be associated with microphthalmos/anophthalmos
- May be associated with epiblepharon (**Fig 1.17.1**)
- Medial 1/3 is commonly involved (**Fig 1.17.2**)
- *Treatment:* excess skin may be removed with resection of tarsus

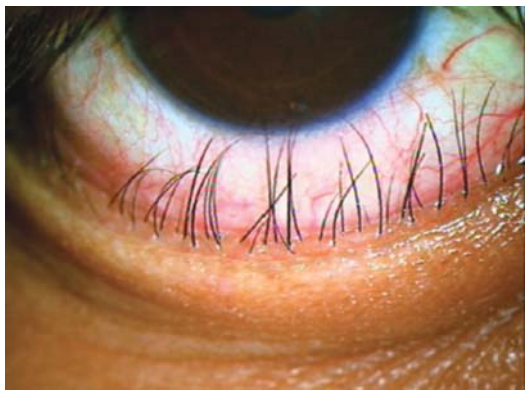


FIGURE 1.17.1: Congenital entropion

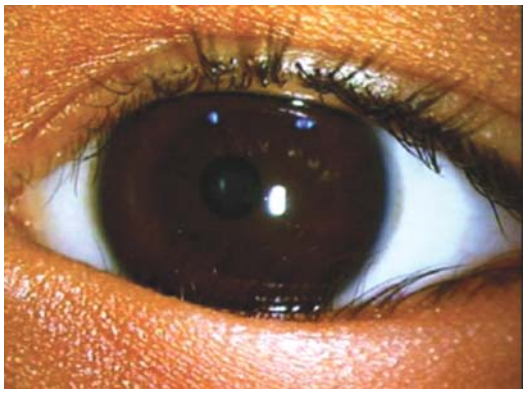


FIGURE 1.17.2: Congenital entropion with epiblepharon

Involutional (senile) Entropion

- Involutional or senile entropion is most common and affects the lower lid only (**Figs1.18.1 and 1.18.2**). Can be easily corrected by simple digital pressure
- Very rarely in both eyelids (**Fig 1.18.3**)
- It is caused by horizontal lid laxity and over-riding of pre-septal part of orbicularis
- *Treatment:* temporary – adhesive tape, cautery, transverse lid everting suture, etc.
  permanent: Weis's procedure, horizontal lid shortening, tucking of inferior lid retractors, etc.

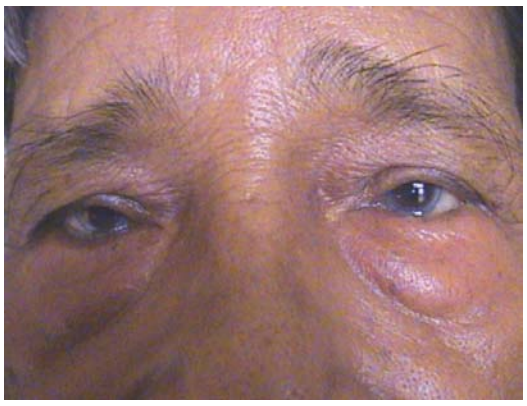


FIGURE 1.18.1: Senile entropion-both eyes

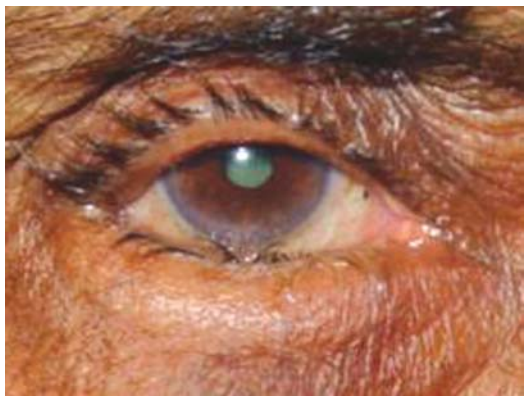


FIGURE 1.18.2: Senile entropion

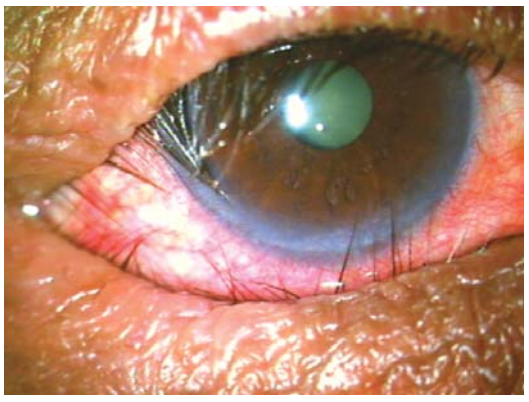


FIGURE 1.18.3: Senile entropion-both lids

Cicatricial Entropion

- Due to scarring of the palpebral conjunctiva
- It usually affects the upper lid (**Fig 1.19.1**) and cannot be corrected by digital pressure
- *Causes:* chemical burn, trachoma, Stevens-Johnson syndrome, ocular pemphigoid, etc.
- *Treatment:* tarsal wedge resection, tarsal fracture, etc.

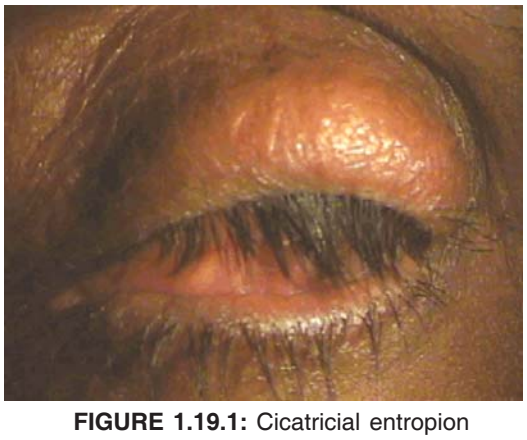


FIGURE 1.19.1: Cicatricial entropion

Acute Spastic Entropion

- Associated with blepharospasm, mainly affects the lower lids (**Fig 1.20.1**)
- *Causes:* chronic conjunctivitis, keratitis and postoperative
- *Treatment*: adhesive tape and removal of the cause

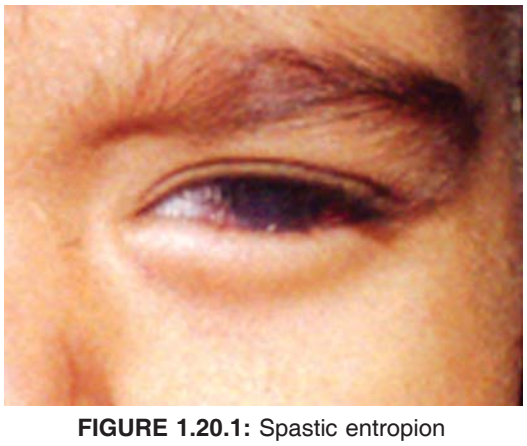


FIGURE 1.20.1: Spastic entropion

ECTROPION

Outward turning of the eyelid away from the globe

Involutional (senile) Ectropion

- Age-related condition which affects the lower lid
- It is the commonest form (**Fig 1.21.1**)
- Due to excessive horizontal eyelid length with weakness of the preseptal part of orbicularis. Laxity of medial canthal tendon is marked
- *Treatment:* medial conjunctivoplasty, Bick's procedure, horizontal lid shortening, etc.

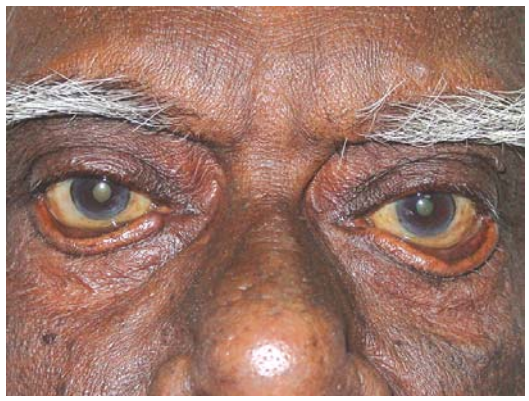


FIGURE 1.21.1: Senile ectropion

Cicatricial Ectropion

- Contracture of the skin and underlying tissues of the lower eyelid or rarely upper lid (**Fig 1.22.1**)
- *Causes*: chemical (**Fig 1.22.2**) or thermal burn (**Fig 1.22.3**), trauma/lacerated injury (**Fig 1.22.4**)
- *Treatment:*
  - excision of the scar with a skin graft to the raw area
  - lengthening of vertical shortening by Z-plasty

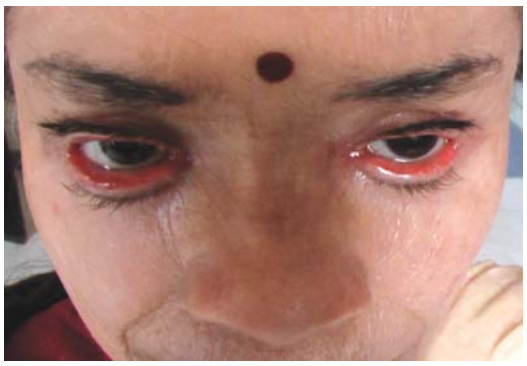


FIGURE 1.22.1: Cicatricial ectropion of all four lids

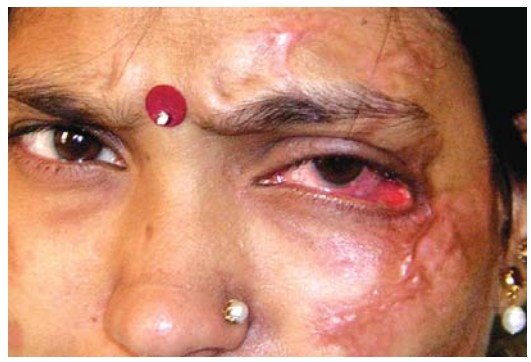


FIGURE 1.22.2: Cicatricial ectropion-chemical injury

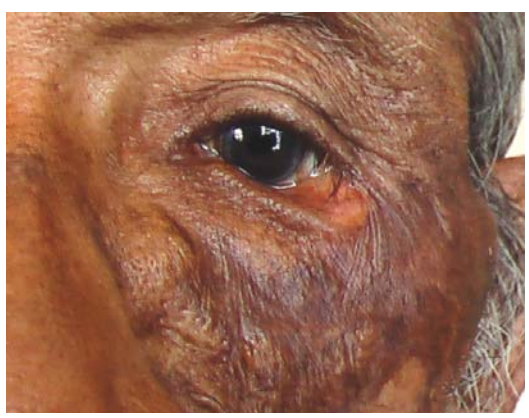


FIGURE 1.22.3: Cicatricial ectropion-thermal injury

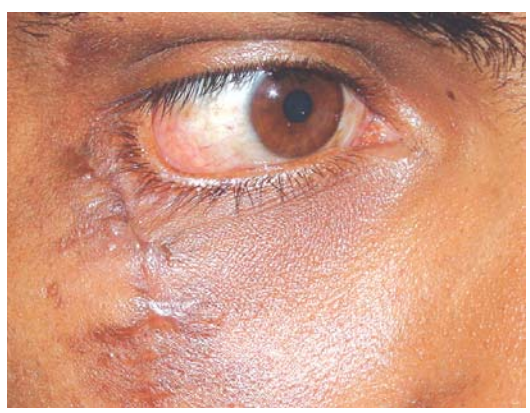


FIGURE 1.22.4: Cicatricial ectropion-traumatic

Paralytic Ectropion

- Caused by paralysis of orbicularis and associated with lagophthalmos (**Fig 1.23.1**)
- Incomplete blinking and epiphora
- May cause corneal ulcer in severe cases (**Fig 1.23.2**)
- *Treatment:*
  mild cases: tear substitute to prevent corneal drying
  severe cases: lateral tarsorrhaphy, lateral canthoplasty

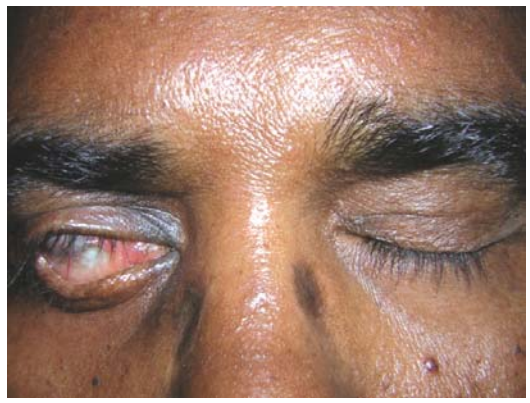


FIGURE 1.23.1: Paralytic ectropion

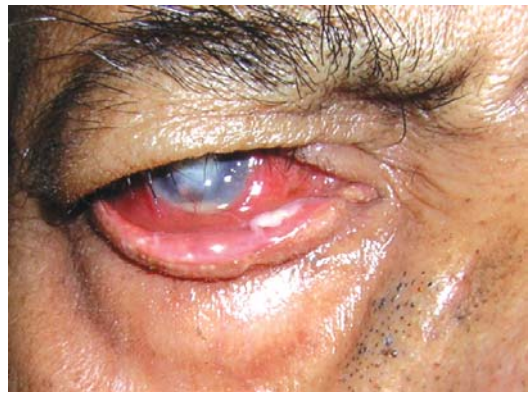


FIGURE 1.23.2: Paralytic ectropion with corneal ulcer

Mechanical Ectropion

- It is just a sequel to a swelling of the lower eyelid, *e.g.* a tumor, lid edema, or a large chalazion (**Fig 1.24.1**)
- *Treatment:* can be corrected easily

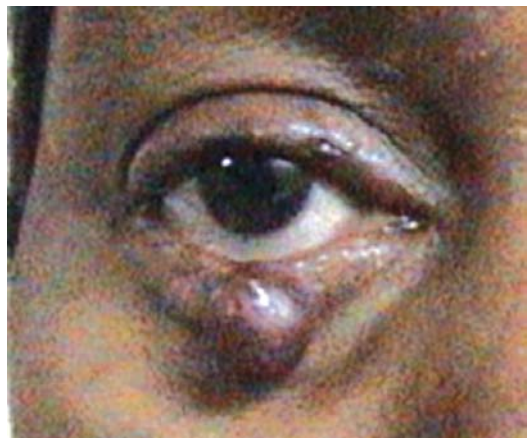


FIGURE 1.24.1: Mechanical ectropion

INFLAMMATORY LID CONDITIONS

Acute Contact Dermatitis

- Unilateral (**Fig 1.25.1**) or bilateral (**Fig 1.25.2**) condition, caused by hypersensitivity to topical medication, hair dyes, cosmetics, etc.
- Edema, erythema, vesiculation and later on crusting (**Fig 1.25.3**)
- *Treatment:* withdrawal of the irritants, antihistaminics and/or corticosteroids

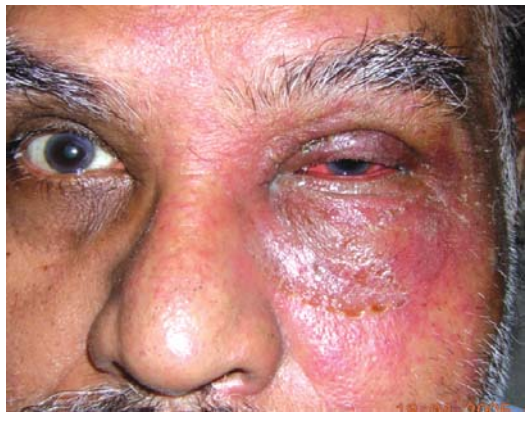


FIGURE 1.25.1: Acute contact dermatitis

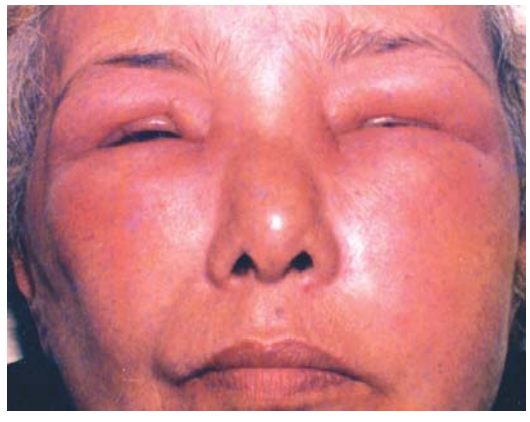


FIGURE 1.25.2: Bilateral acute contact dermatitis

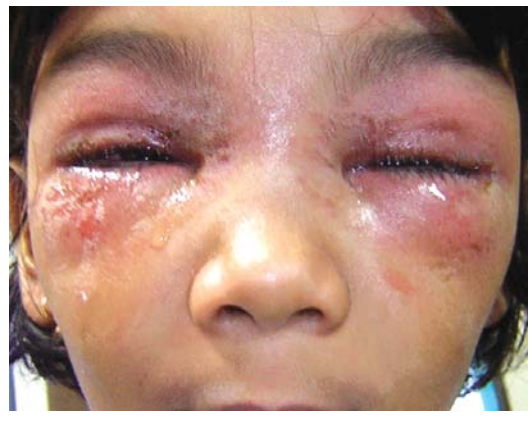


FIGURE 1.25.3: Bilateral severe contact dermatitis

Chronic Contact Dermatitis

- Caused by chronic irritants, e.g. topical medication, cosmetics, spectacles frame
- Thickening and crusting of the skin (**Fig 1.26.1**)
- *Treatment:* identification of offending agent and rectify it, antibiotic-steroid ointment

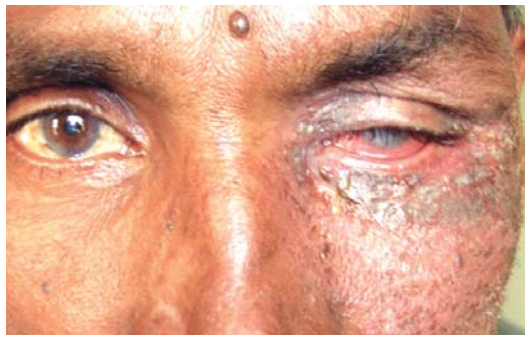


FIGURE 1.26.1: Chronic contact dermatitis

Atopic Dermatitis of the Lid

- Uncommon and more generalized with skin condition
- Eyelids are thickened and fissured (**Fig 1.27.1**)
- Associated with atopic keratoconjunctivitis

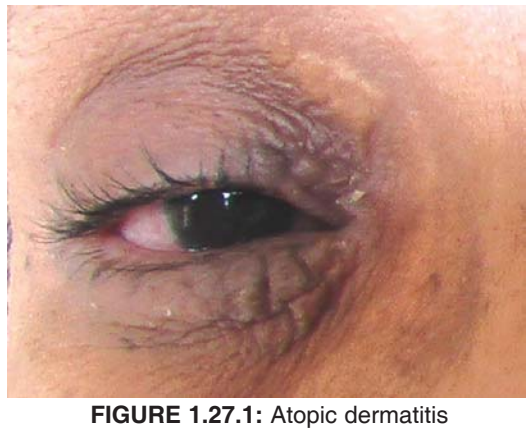


FIGURE 1.27.1: Atopic dermatitis

Angular Blepharoconjunctivitis

- Unilateral or bilateral infection, caused by *Moraxella*
- Frequently associated with conjunctivitis (**Fig 1.28.1**)
- Fissuring, maceration, erythema and scaling of one or both canthi (**Fig 1.28.2**)
- *Treatment:* oxytetracycline eye ointment and zinc oxide containing eye ointment

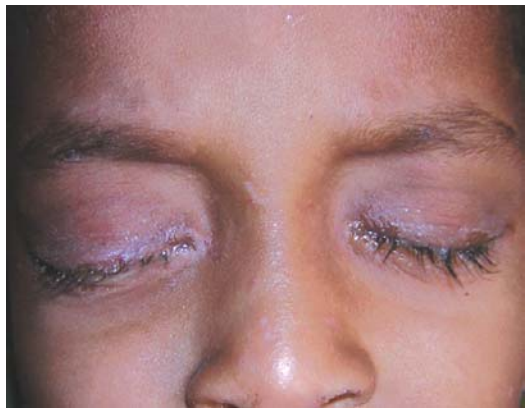


FIGURE 1.28.1: Angular blepharoconjunctivitis

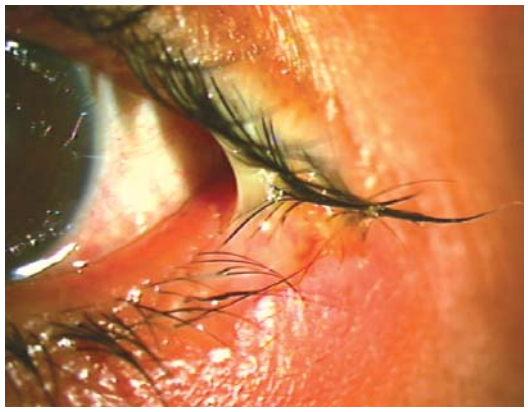


FIGURE 1.28.2: Angular blepharoconjunctivitis

Primary Herpes Simplex of Lids

- Uncommon, unilateral condition, may be associated with immune deficiency states
- Crops of small vesicles, ruptures and crust formation, may be with secondary infection (**Fig 1.29.1**)
- Healing without scarring by seven days
- May be associated with acute follicular conjunctivitis and keratitis
- *Treatment:* topical acyclovir ointment

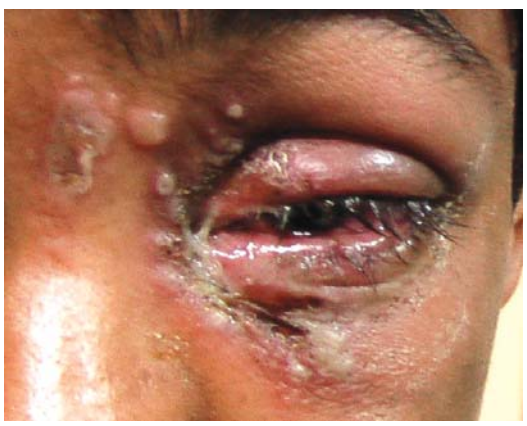


FIGURE 1.29.1: Primary herpes simplex of lids

Herpes Zoster Ophthalmicus

- More common unilateral condition, may be severe in immuno-compromised conditions
- Painful maculo-papular rash involving the first division of trigeminal nerve (**Fig 1.30.1**)
- Development of vesicles, pustules, and ulceration with crusting (**Fig 1.30.2**)
- Periorbital edema, secondary infection may lead to bacterial cellulitis
- *Treatment:* high dose of oral acyclovir, investigation to find out the cause of immune deficiency

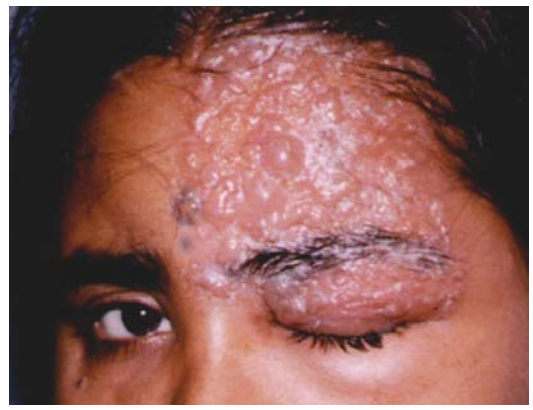


FIGURE 1.30.1: Herpes zoster ophthalmicus

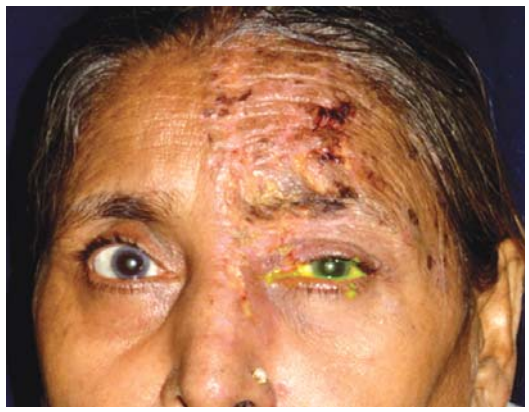


FIGURE 1.30.2: Herpes zoster ophthalmicus

Blepharitis

- Sub-acute or chronic inflammation of the eyelids
- Mostly in children and usually bilateral
- Associated with seborrhea (dandruff) of the scalp
- *Squamous blepharitis:*
  - hyperemia of lid margins
  - white dandruff-like scales on the lid margins (**Fig 1.31.1**)
  - falling of eyelashes (madarosis)
  - thickening of the lid margins (tylosis) (**Fig 1.31.2**)
- *Ulcerative blepharitis:*
  - soreness of the lid margins (**Fig 1.31.3**)
  - loss of eyelashes
  - yellow crust at the root of the eyelashes with matting (**Fig 1.31.4**)
  - small ulcers at the base of the crust (**Fig 1.31.5**)
  - marginal keratitis is a common association
- *Treatment:* lid hygiene (lid scrub), antibiotic-steroid ointment, systemic tetracycline/doxycycline, treatment of dandruffs, etc.

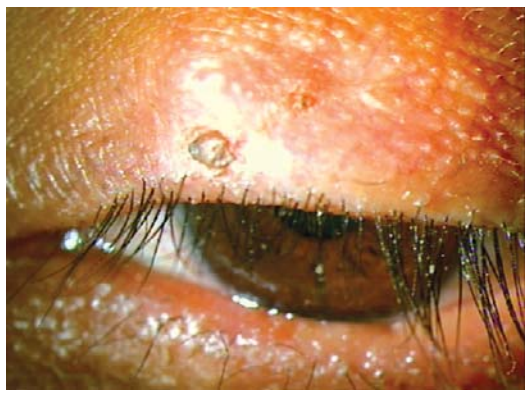


FIGURE 1.31.1: Squamous blepharitis

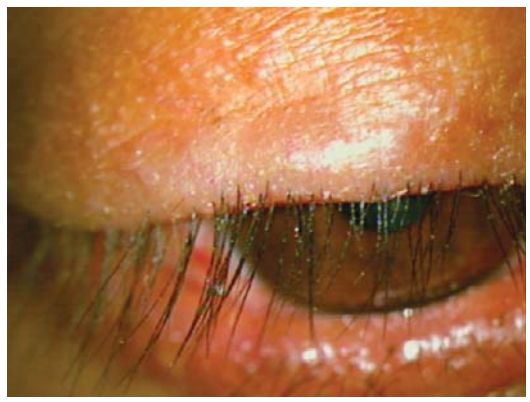


FIGURE 1.31.2: Squamous blepharitis-tylosis

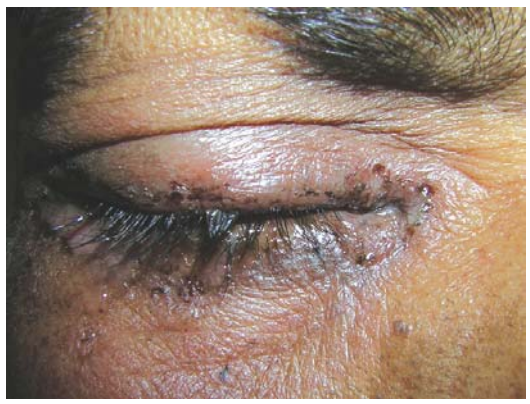


FIGURE 1.31.3: Ulcerative blepharitis

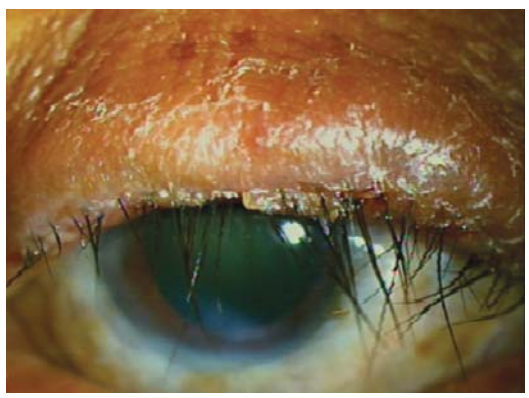


FIGURE 1.31.4: Ulcerative blepharitis-madarosis

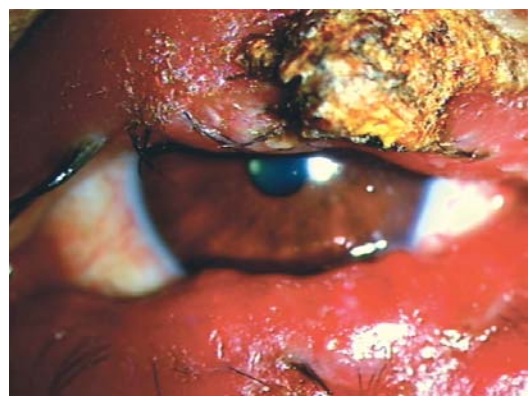


FIGURE 1.31.5: Ulcerative blepharitis-madarosis

Meibomianitis

- Chronic infection of the meibomian glands
- Occurs in the middle age
- White, frothy secretion on the eyelid margins and at the canthi (seborrhea) (**Fig 1.32.1**)
- Vertical yellowish streaks shining through the conjunctiva (**Fig 1.32.2)**
- Blocked meibomian ducts (**Figs 1.32.3 and 1.32.4**)
- Thick secretion on expression ('tooth paste sign') (**Fig 1.32.5**)
- *Treatment:* tarsal (vertical lid) massage, steroid-antibiotic ointment, systemic doxycycline, etc.

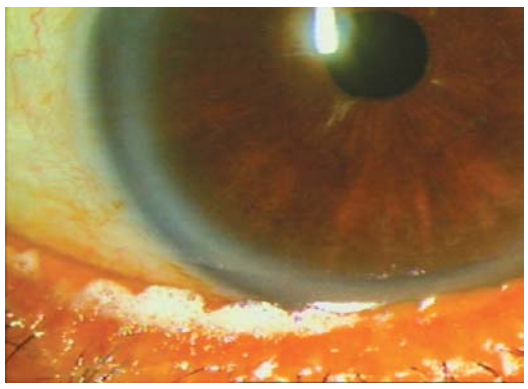


FIGURE 1.32.1: Meibomian seborrhea

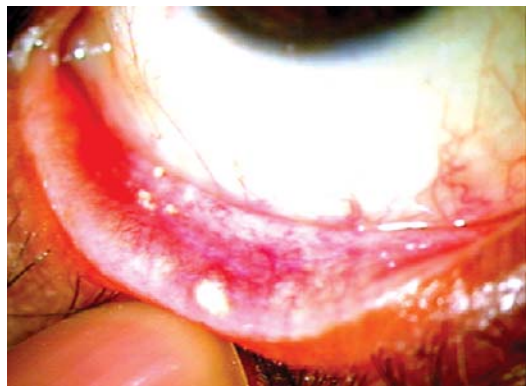


FIGURE 1.32.2: Meibomianitis-vertical streak

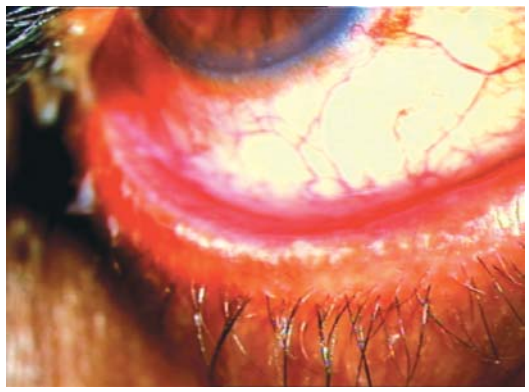


FIGURE 1.32.3: Meibomianitis-blocked ducts

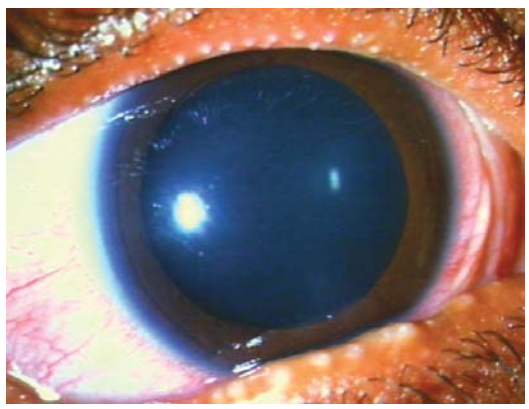


FIGURE 1.32.4: Meibomianitis-blocked ducts

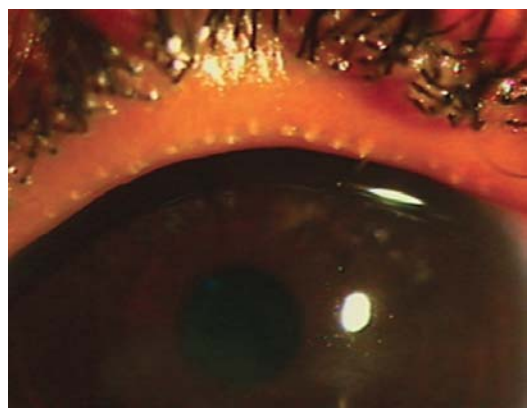


FIGURE 1.32.5: Meibomianitis-tooth paste sign

Preseptal Cellulitis

- Unilateral erythema and edema with tenderness involving the upper eyelid (**Fig 1.33.1**)
- May lead to lid abscess (**Fig 1.33.2**)
- *D/D with orbital cellulitis* (**Fig 1.33.3**)
  - no proptosis
  - normal visual acuity, ocular movement and papillary reactions
- *Treatment:* systemic antibiotics, analgesics, hot compress and topical antibiotics

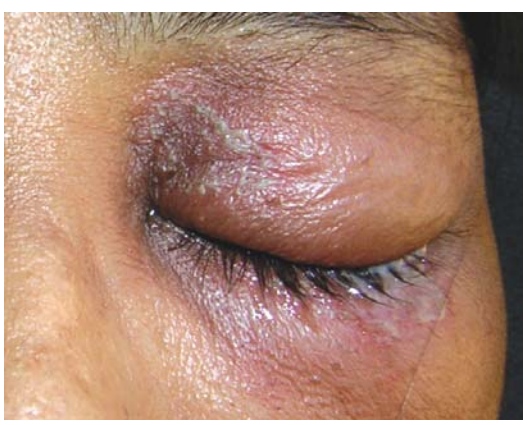


FIGURE 1.33.1: Preseptal cellulitis

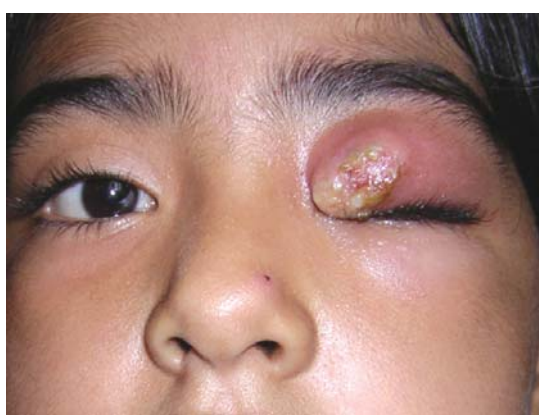


FIGURE 1.33.2: Lid abscess

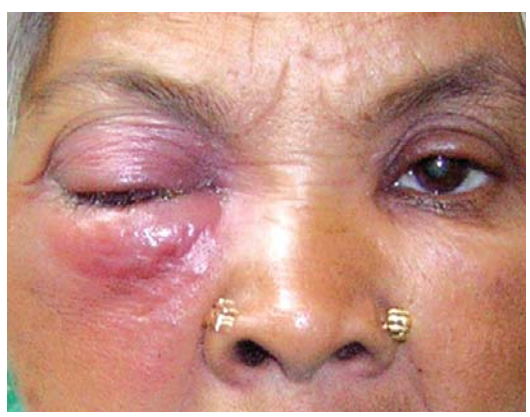


FIGURE 1.33.3: Orbital cellulitis

External Hordeolum (stye)

- Acute suppurative inflammation of the follicle of an eyelash **(Fig 1.34.1)**
- A swollen area at the lid margin
- May be associated with preseptal cellulitis and conjunctival chemosis **(Fig 1.34.2)**
- A whitish, round , raised pus point at eyelash root
- *Treatment:* hot compress, systemic analgesics, topical antibiotics, epilation of offending eyelash

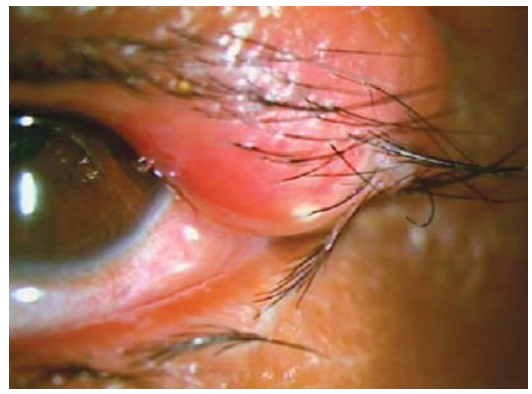


FIGURE 1.34.1: External hordeolum

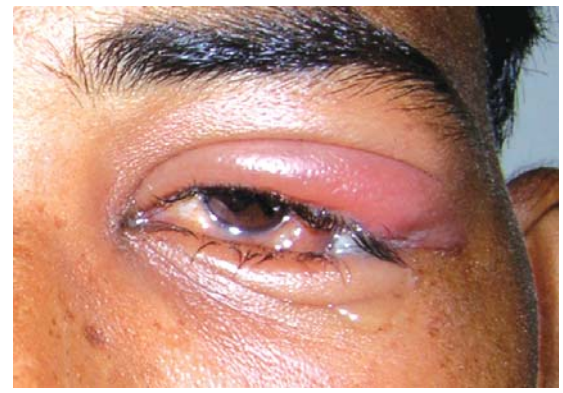


FIGURE 1.34.2: External hordeolum with preseptal cellulitis

Internal hordeolum (inflamed chalazion)

- Unilateral acute infection of the meibomian gland
- Tender, diffuse, inflamed swelling within the tarsal plate
- The swelling is away from the lid margin (**Fig 1.35.1**)
- Pus-point away from the eyelash root (**Fig 1.35.2**)
- May be associated with preseptal cellulitis
- *Treatment:* treatment of acute infection followed by incision and curettage of the chalazion later on

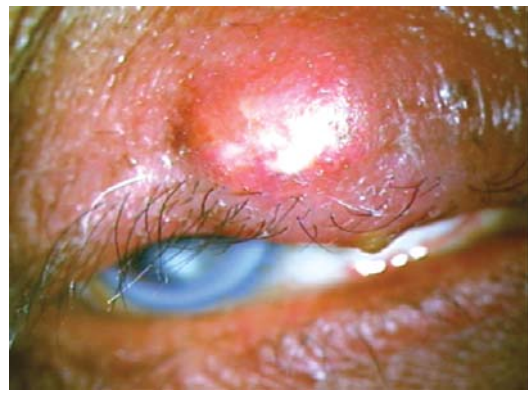


FIGURE 1.35.1: Internal hordeolum

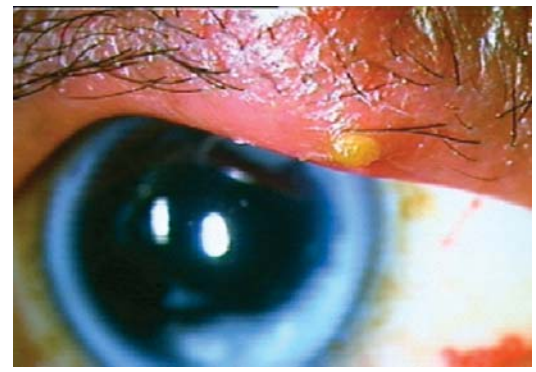


FIGURE 1.35.2: Internal hordeolum

Chalazion

- A chronic non-specific inflammatory granuloma of the meibomian gland
- Painless nodular swelling of the eyelid (**Fig 1.36.1**)
- Tarsal conjunctiva underneath the nodule is velvety red or purple and slightly elevated (**Fig 1.36.2**)
- May be single or multiple **(Figs 1.36.3 and 1.36.4)**
- It may turn into 'marginal chalazion' (**Fig 1.36.5**)
- *Treatment:* steroid-antibiotic ointment for small chalazion and 'incision and curettage' for large one

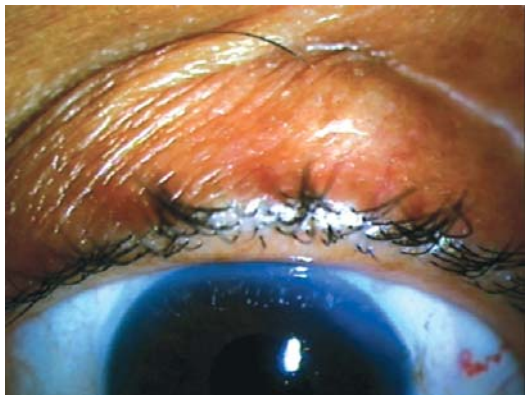


FIGURE 1.36.1: Chalazion

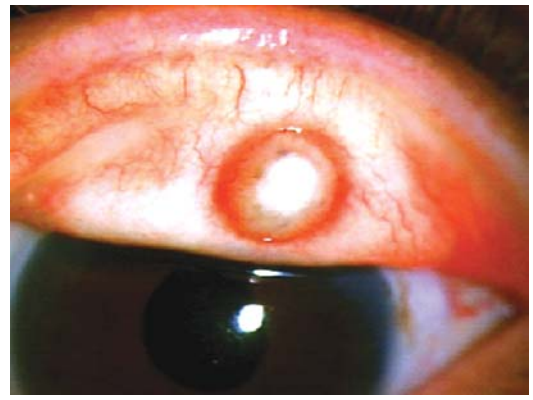


FIGURE 1.36.2: Chalazion-conjunctival surface

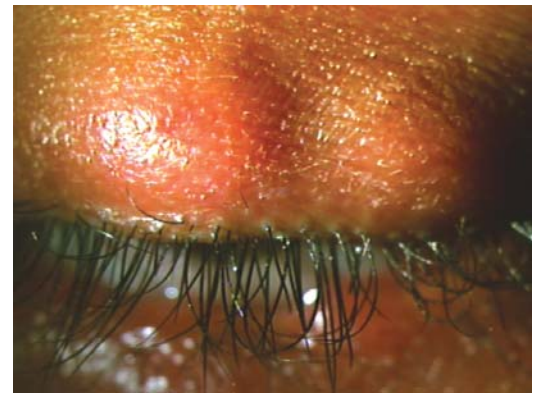


FIGURE 1.36.3: Multiple chalazion

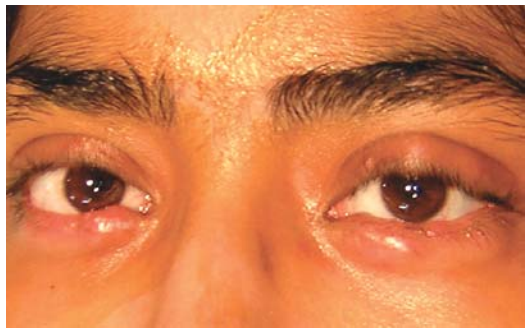


FIGURE 1.36.4: Multiple chalazion of all four lids

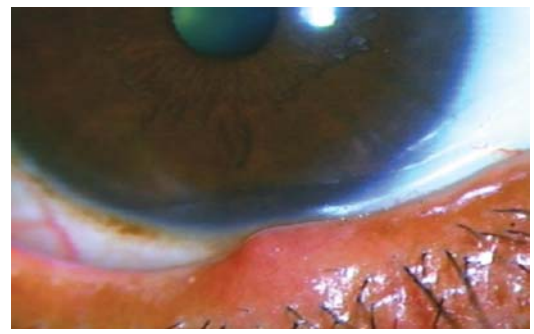


FIGURE 1.36.5: Marginal chalazion

PTOSIS: DROOPING OF UPPER LID

Ptosis

- Drooping of the upper eyelid
- Unilateral (**Fig 1.37.1**) or bilateral (**Fig 1.37.2**), and partial or complete
  - *mild ptosis:* 2 mm (**Fig 1.37.3**)
  - *moderate ptosis:* 3 mm (**Fig 1.37.4**)
  - *severe ptosis:* 4 mm or more (**Figs 1.37.5 and 1.37.6**)

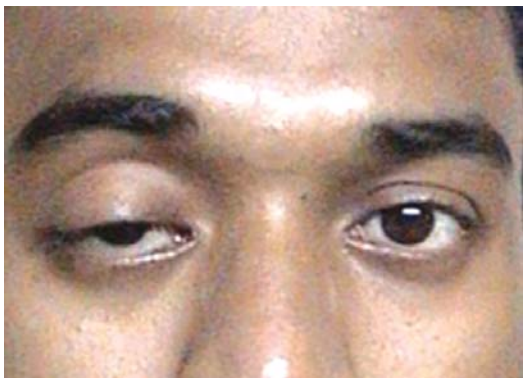


FIGURE 1.37.1: Unilateral ptosis

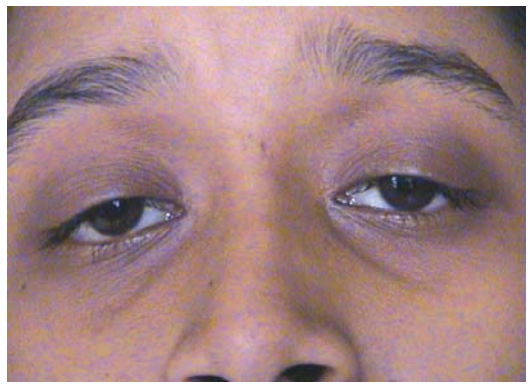


FIGURE 1.37.2: Bilateral ptosis

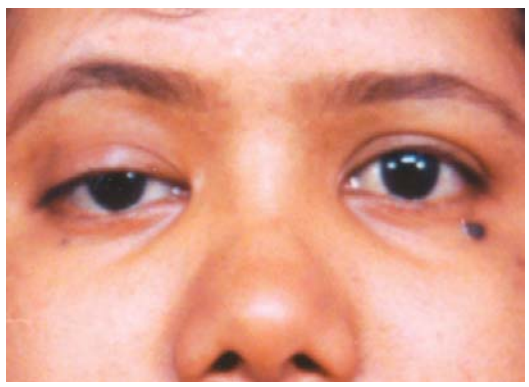


FIGURE 1.37.3: Mild ptosis

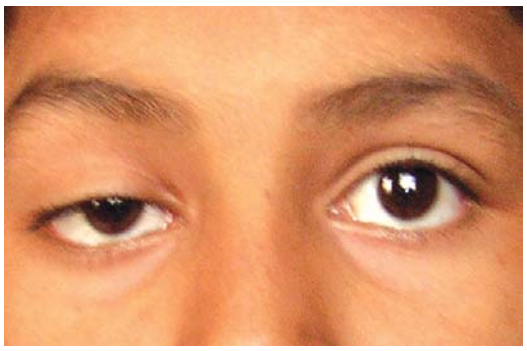


FIGURE 1.37.4: Moderate ptosis

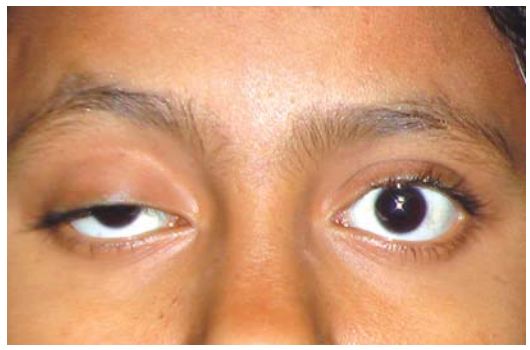


FIGURE 1.37.5: Severe ptosis

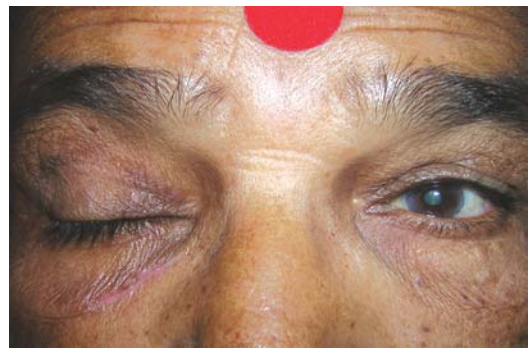


FIGURE 1.37.6: Severe complete ptosis

Synkinetic Ptosis

- *Marcus-Gunn jaw winking phenomenon:* Retraction of the ptotic eyelid with ipsilateral jaw movement **(Figs 1.38.1 and 1.38.2)**
- Sometimes, amount of ptosis increases with jaw movement—*a reverse Marcus-Gunn pheno-menon* **(Figs 1.38.3 and 1.38.4)**
- *Misdirected third nerve:* retraction of upper lid with various ocular movements

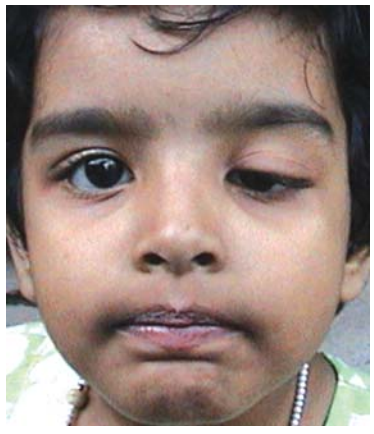


FIGURE 1.38.1: Marcus-Gunn phenomenon

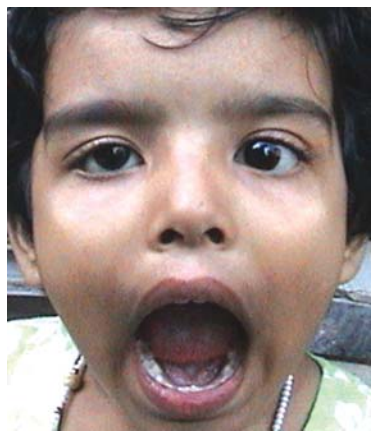


FIGURE 1.38.2: Marcus-Gunn phenomenon

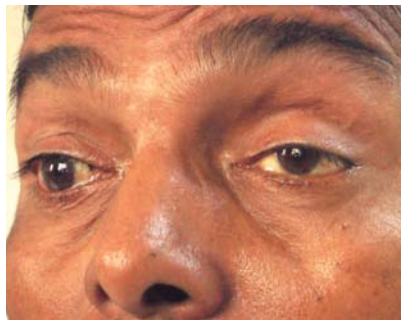


FIGURE 1.38.3: Reverse Marcus-Gunn phenomenon

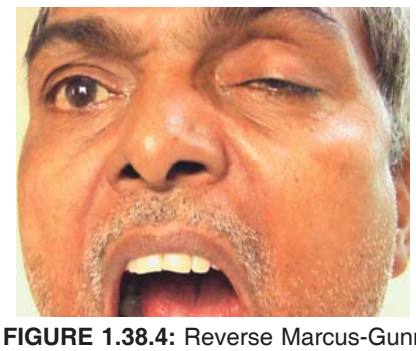


FIGURE 1.38.4: Reverse Marcus-Gunn phenomenon

Pseudoptosis

- Anophthalmos **(Fig 1.39.1)**, microphthalmos and phthisis bulbi **(Fig 1.39.2)**
- Due to hypotropia **(Fig 1.39.3)**
- Dermatochalasis

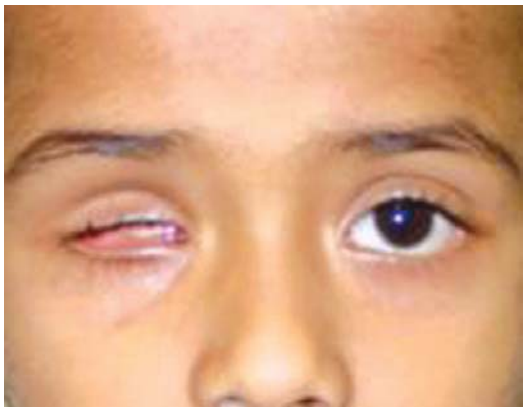


FIGURE 1.39.1: Pseudoptosis-anophthalmos

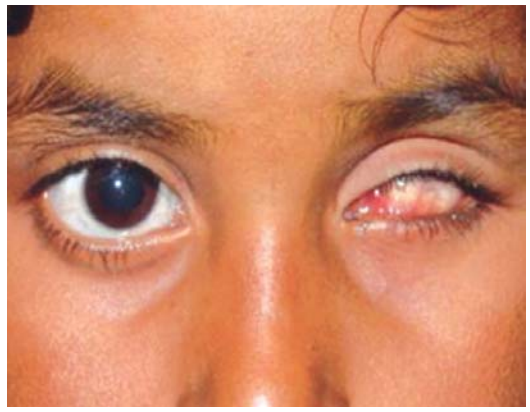


FIGURE 1.39.2: Pseudoptosis-phthisis bulbi

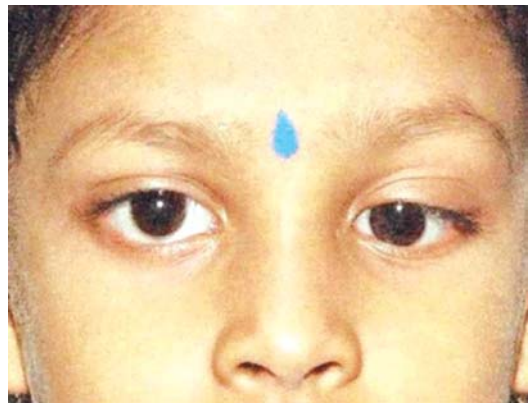


FIGURE 1.39.3: Pseudoptosis-left hypotropia

Congenital Ptosis

- Unilateral or bilateral with varying severity – mild, moderate or severe **(Figs 1.40.1 and 1.40.2)**
- May be simple or with other anomalies like "jaw-winking phenomenon"
- May be associated with blepharophimosis syndrome **(Fig 1.5.2)**
- *Treatment:* depends on severity, early intervention is required in severe ptosis to prevent amblyopia

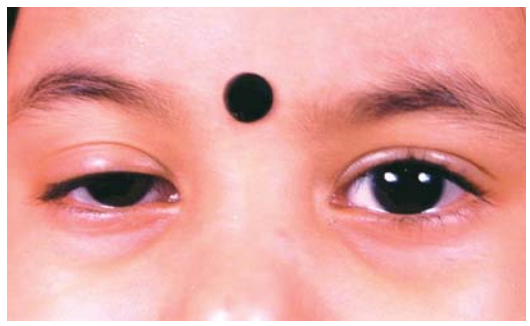


FIGURE 1.40.1: Unilateral congenital ptosis

FIGURE 1.40.2: Bilateral congenital ptosis

Acquired Ptosis

- *Neurogenic:* third nerve palsy **(Fig 1.41.1)** or Horner's syndrome **(Fig 1.41.4)**
- *Myogenic*
  - *myasthenia gravis:* Tensilon (edrophonium) or Prostigmine (neostigmine) test—a positive test means improvement of ptosis with intravenous injection **(Figs 1.41.2 and 1.41.3)**
  - *ocular myopathy* **(Fig 1.41.5)**
  - *senile ptosis*
- *Traumatic* **(Fig 1.41.6)**
- *Mechanical* **(Figs 1.41.7 and 1.41.8)**

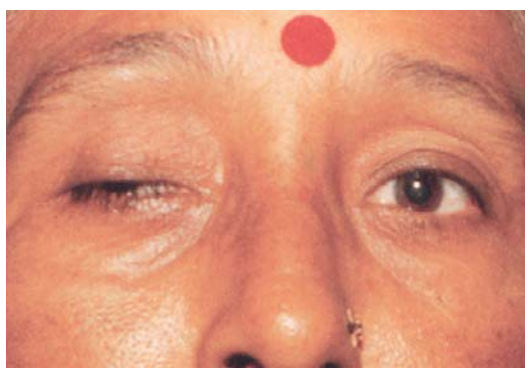


FIGURE 1.41.1: Third nerve palsy

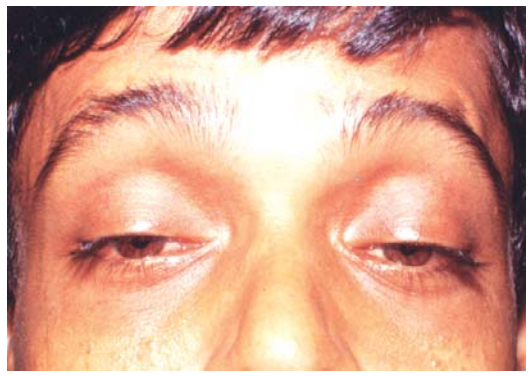


FIGURE 1.41.2: Myasthenia gravis

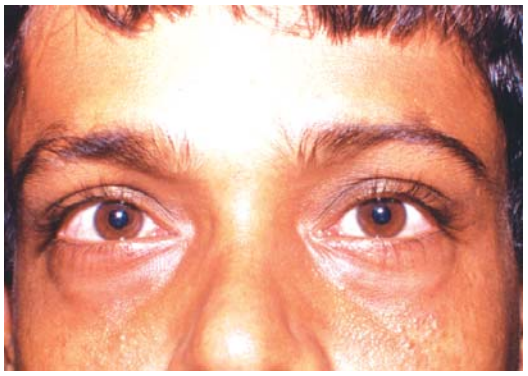


FIGURE 1.41.3: Myasthenia gravis-after injection

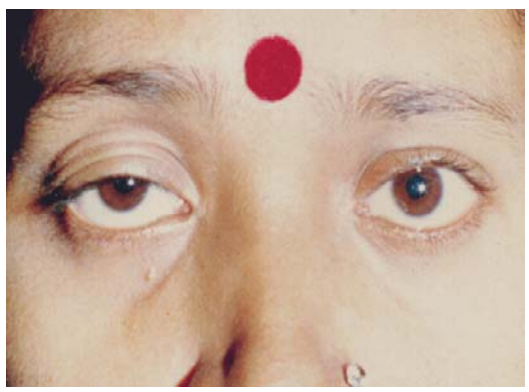


FIGURE 1.41.4: Horner's syndrome

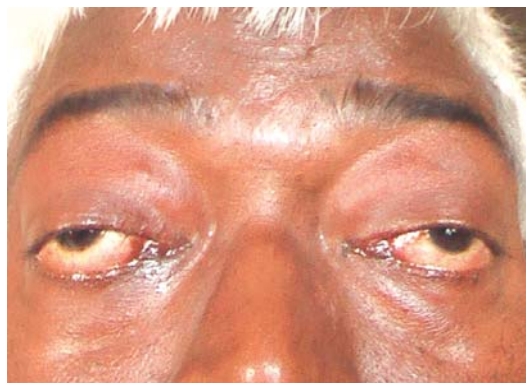


FIGURE 1.41.5: Myogenic ptosis-ocular myopathy

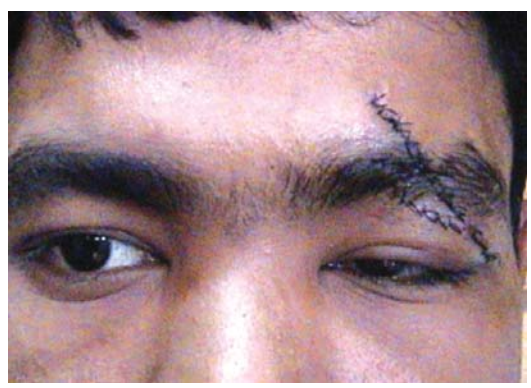


FIGURE 1.41.6: Traumatic ptosis

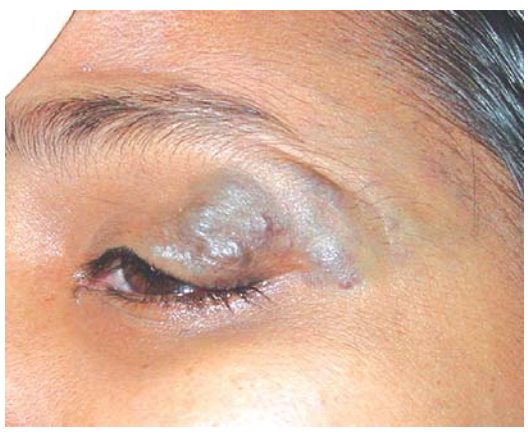


FIGURE 1.41.7: Mechanical ptosis-hemangioma upper lid

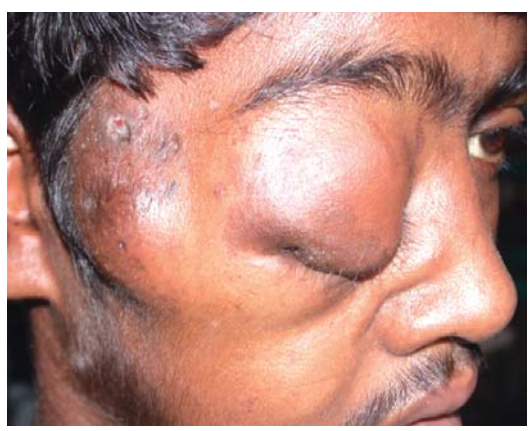


FIGURE 1.41.8: Mechanical ptosis-plexiform neurofibroma

Senile (Aponeurotic) Ptosis

- Common unilateral or bilateral ptosis caused by defect in levator aponeurosis **(Fig 1.42.1)**
- Good levator function
- Absent or high upper-lid crease
- Thinning of upper-lid above the tarsal plate
- Deep upper supratarsal sulcus (**Fig 1.42.2**)
- *Treatment*: surgical correction in severe cases

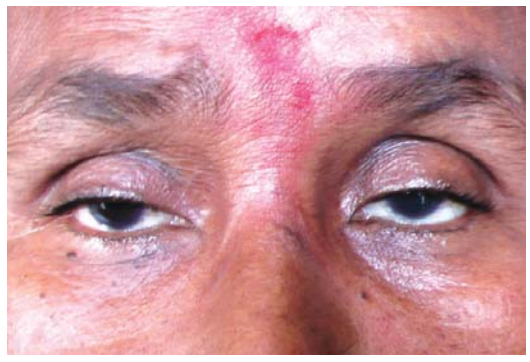


FIGURE 1.42.1: Senile ptosis

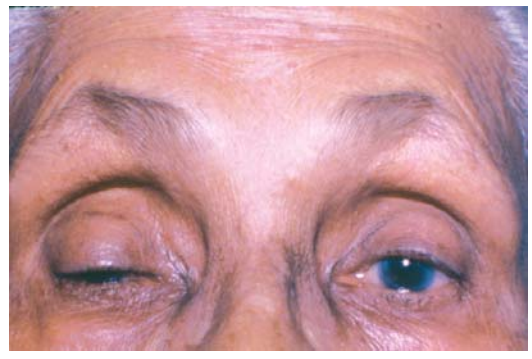


FIGURE 1.42.2: Senile ptosis-deep supratarsal sulcus

Lid Retraction

- Unilateral or bilateral retraction of the upper lid or some times both lids in the primary position
- *Causes:* thyroid eye diseases **(Fig 1.43.1)**, neurogenic **(Fig 1.43.2)**, surgical overcorrection **(Fig 1.43.3)**, phenylephrine eye drops **(Fig 1.43.4)**, hydrocephalus, etc.
- *Treatment*: directed towards the cause

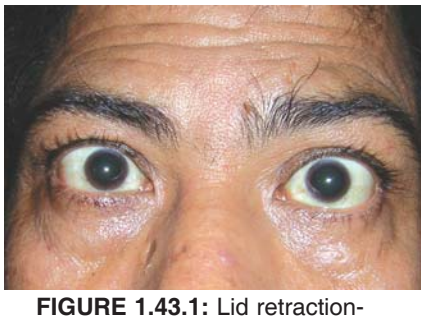


FIGURE 1.43.1: Lid retraction-thyroid eye diseases

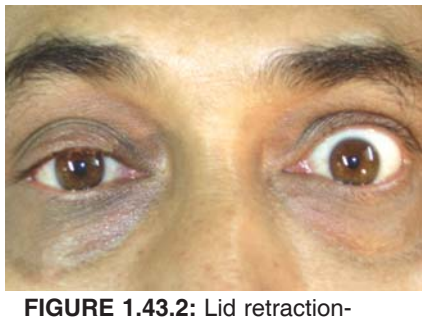


FIGURE 1.43.2: Lid retraction-neurogenic

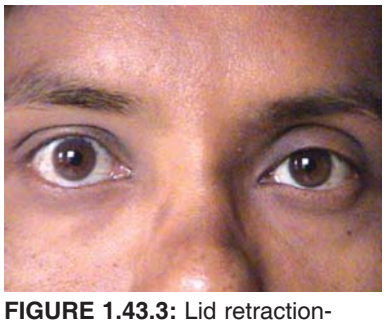


FIGURE 1.43.3: Lid retraction-surgical overcorrection

FIGURE 1.43.4: Lid retraction-pharmacological

OTHER LID CONDITIONS

Chemosis or Lid Edema

- Diffuse edematous swelling of the eyelids
- May be associated with conjunctival chemosis (**Fig 1.44.1**)
- *Causes:*
  - blepharitis (**Fig 1.44.2**)
  - conjunctivitis (**Fig 1.44.3**)
  - dacryocystitis (**Fig 1.44.4**)
  - dacryoadenitis (S-shaped lid margin) (**Fig 1.44.5**)
  - simple allergy (insect bite or urticaria) (**Fig 1.44.6**)
  - Post-surgical
  - Inflammatory orbital diseases (**Fig 1.44.7**)
- *Treatment:* directed towards the cause

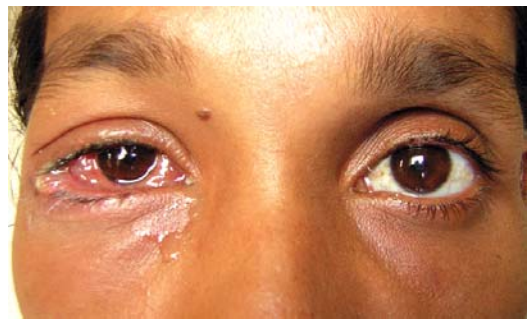


FIGURE 1.44.1: Blepharo-conjunctival chemosis

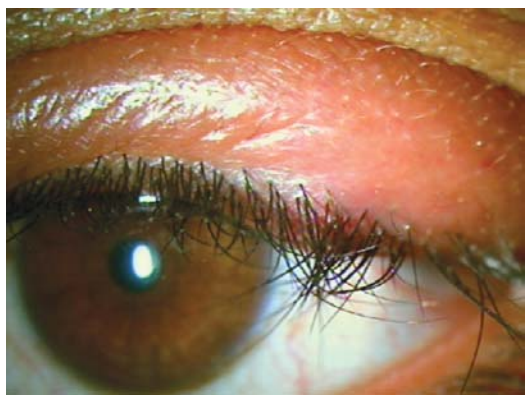


FIGURE 1.44.2: Chemosis of lid-blepharitis

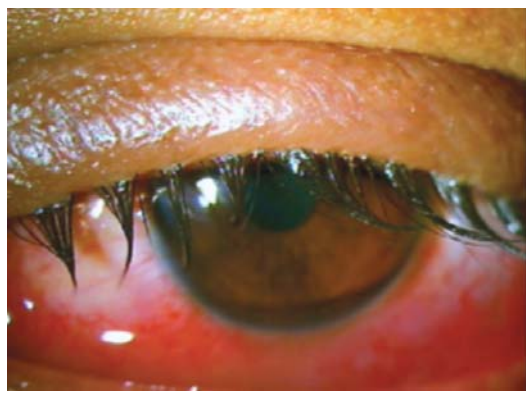


FIGURE 1.44.3: Chemosis of lid-conjunctivitis

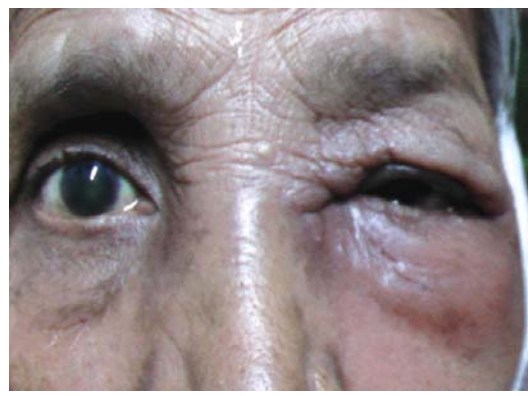


FIGURE 1.44.4: Chemosis of lid-acute dacryocystitis

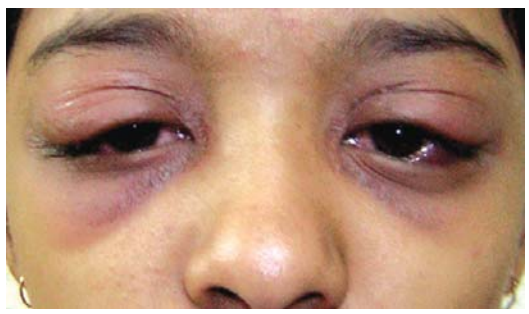


FIGURE 1.44.5: Chemosis of lid-dacryoadenitis

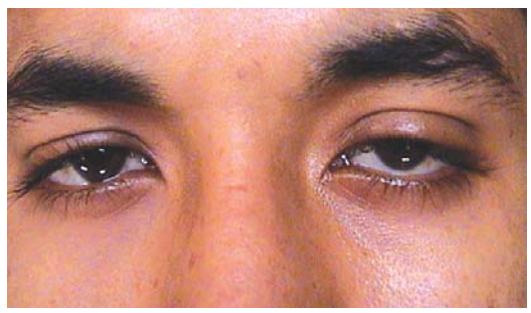


FIGURE 1.44.6: Chemosis of lid-insect bite left eye

FIGURE 1.44.7: Chemosis of lid-inflammatory orbital diseases

Lagophthalmos

- Inadequate closure of the upper eyelid (**Fig 1.45.1**)
- Associated ectropion of lower eyelid
- Dryness of the lower part of the bulbar conjunctiva and cornea (**Fig 1.45.2**)
- May cause exposure keratitis and frank corneal ulceration
- *Treatment:* artificial tears, tarsorrhaphy and lid-load operation

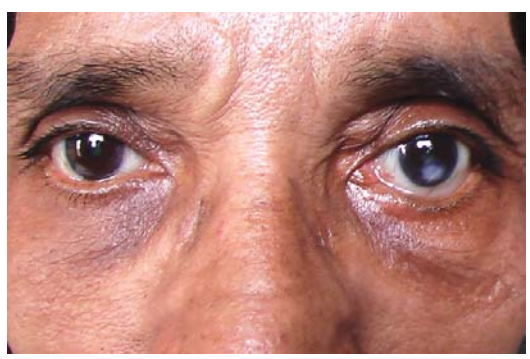


FIGURE 1.45.1: Lagophthalmos

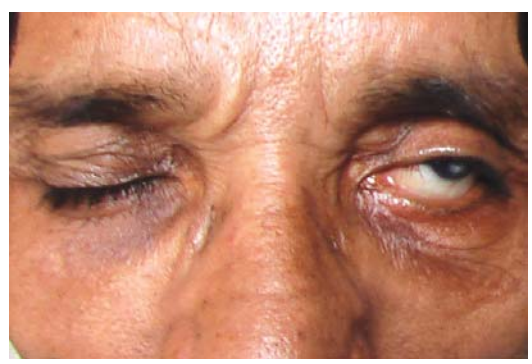


FIGURE 1.45.2: Lagophthalmos-exposure keratitis

Blepharochalasis

- Younger individuals
- Usually unilateral
- Starts at puberty with an intermittent angio-neurotic oedema (**Fig 1.46.1**) and redness of the lid
- Skin hangs down over the upper eyelid (**Fig 1.46.2**)
- *Treatment:* towards allergic problems

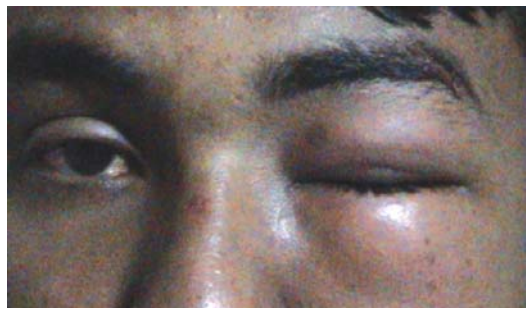


FIGURE 1.46.1: Blepharochalasis

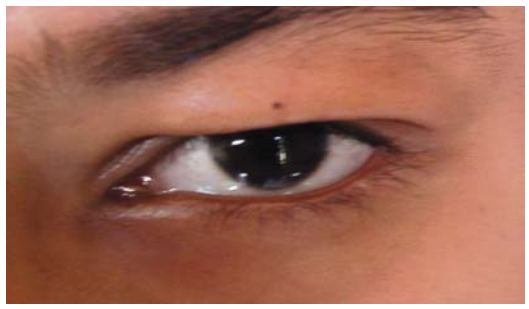


FIGURE 1.46.2: Blepharochalasis

Dermatochalasis

- Usually bilateral
- Loss of skin elasticity
- Prolapse of the fat, mainly nasally in the upper lid **(Fig 1.47.1)**
- Lower lid is also involved with bagginess **(Fig 1.47.2)**
- *Treatment:* cosmetic surgery is useful, but a recurrence is common

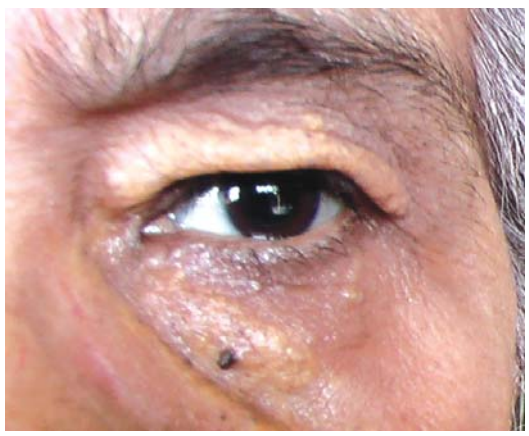


FIGURE 1.47.1: Dermatochalasis

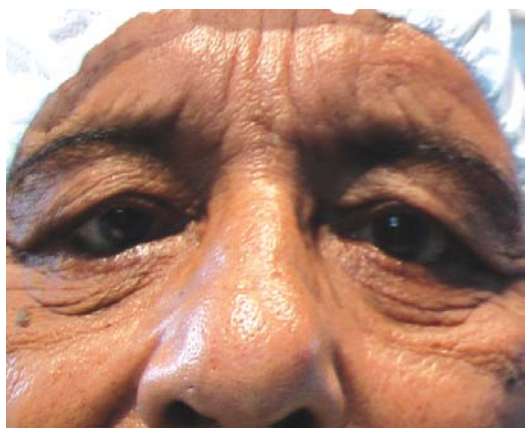


FIGURE 1.47.2: Dermatochalasis

Floppy Eyelid Syndrome

- Floppy, easily reversible upper eyelid with papillary conjunctivitis **(Fig 1.48.1)**
- Chronically red and irritable eye
- Typically obese and with sleep apnea syndrome
- *Treatment:* artificial tears, antibiotic ointment at night, correct sleep posture and lid taping

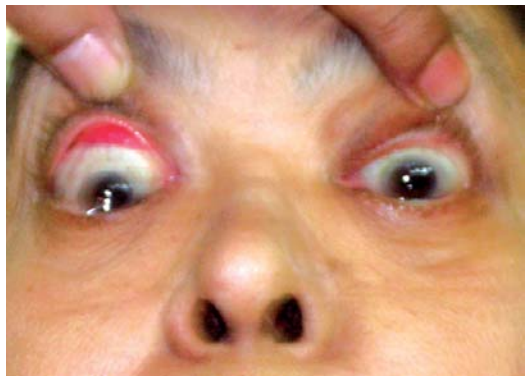


FIGURE 1.48.1: Floppy eye lid syndrome

Symblepharon

- Adhesion of lid with the globe as a result of adhesion between bulbar and palpebral conjunctiva
- *Causes:* chemical burn **(Fig 1.49.1)**, trauma thermal **(Fig 1.49.2)**, burn **(Fig 1.49.3)**, ocular pemphigoid **(Fig 1.49.4)**, Stevens-Johnson syndrome **(Fig 1.49.5)**, trachoma, etc.
- May be anterior, posterior or total
- May be in the form of small band **(Fig 1.49.6)** or frank broad adhesion
- *Treatment:* radical excision of scar tissue along with diseased conjunctiva and conjunctival autograft
  - amniotic membrane transplantation may be helpful; *Prevention* by sweeping a glass rod, and symblepharon ring

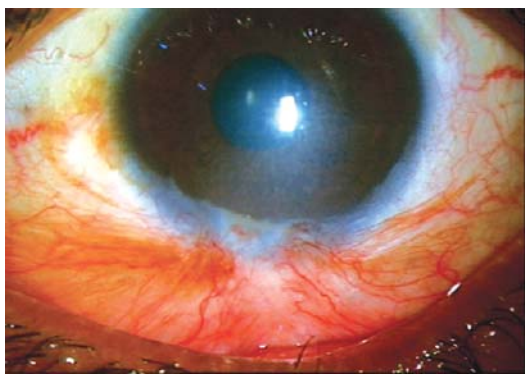


FIGURE 1.49.1: Symblepharon-chemical burn

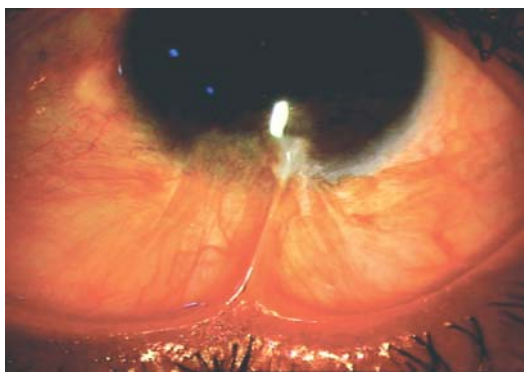


FIGURE 1.49.2: Symblepharon-traumatic

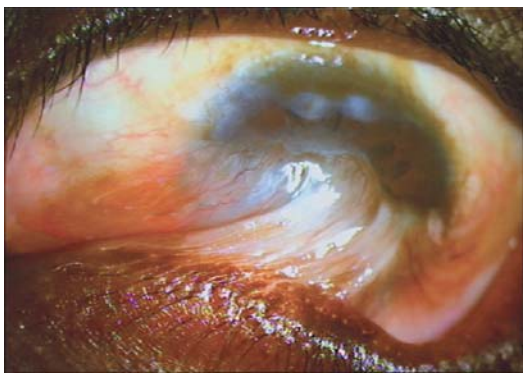


FIGURE 1.49.3: Symblepharon-thermal burn

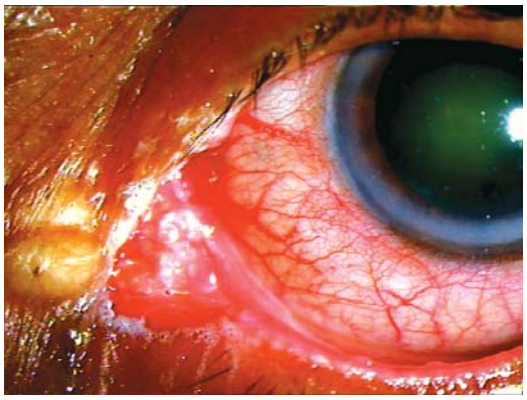


FIGURE 1.49.4: Symblepharon-ocular pemphigoid

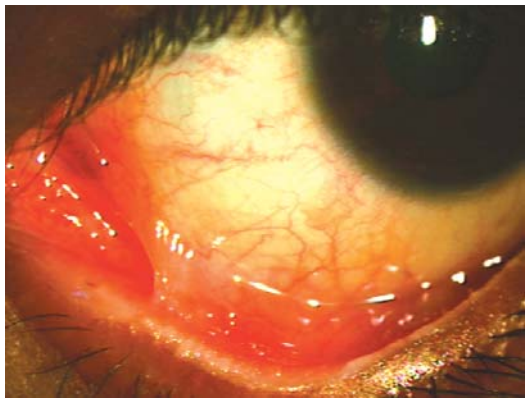


FIGURE 1.49.5: Symblepharon-SJ syndrome

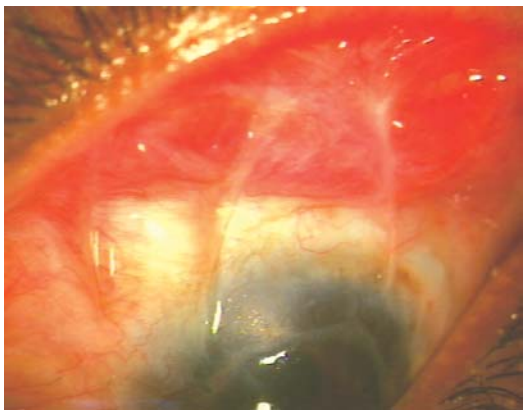


FIGURE 1.49.6: Symblepharon-chemical burn

Essential Blepharospasm

- Spontaneous in older patient
- Involuntary tonic, spasmodic, bilateral contraction of orbicularis oculi (**Fig 1.50.1**)
- Other muscles of face may involve simultaneously
- *Treatment:* botulinium toxin, alcohol injection
- *Reflex blepharospasm* may occur in superficial corneal problems (**Fig 1.50.2**) and abolished by topical anesthesia

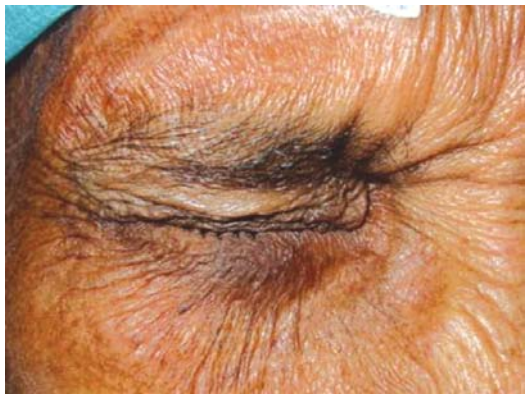


FIGURE 1.50.1: Essential blepharospasm

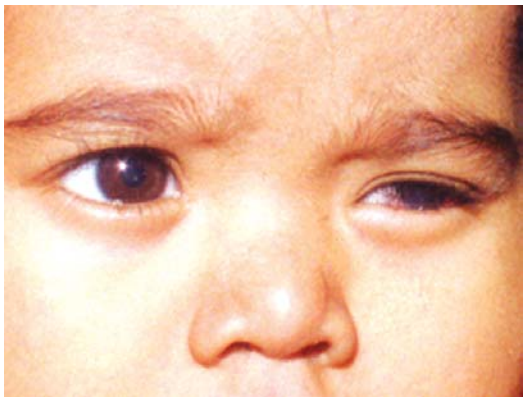


FIGURE 1.50.2: Reflex blepharospasm

Ecchymosis of the Eyelids

- Occurs after a blunt trauma **(Fig 1.51.1)**
- Painful edema with variable degree of Ecchymosis
- Called 'Panda bear' sign **(Fig 1.51.2)** when both lids are involved
- *Treatment:* no active treatment is required

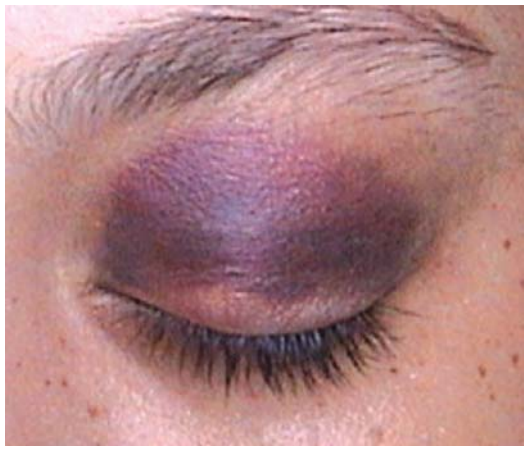


FIGURE 1.51.1: Ecchymosis of lid

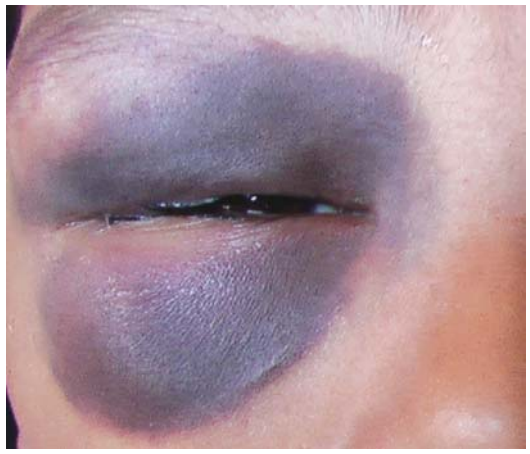


FIGURE 1.51.2: Black eye-Panda bear sign

Phthiriasis Palpebrum

- Infestation of eyelashes with crab louse "*phthirus pubis*" and its ova, called nits
- Typically affects children and young female
- The lice are adherent to the skin **(Fig 1.52.1)** and nits stuck to the lashes **(Fig 1.52.2)**
- *Treatment:* cotton pellet soaked in pilocarpine eye drop applied over the eyelashes for few minutes then the lice can be easily removed
  - *simultaneous treatment* for body louse infestation

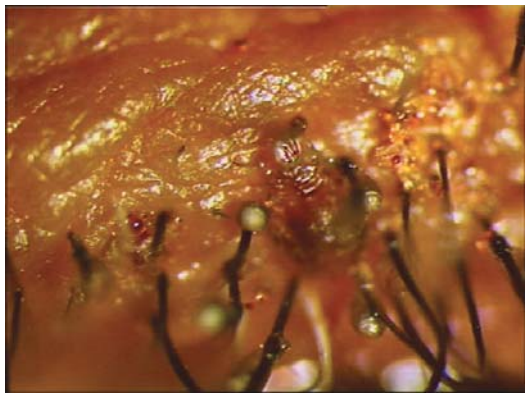


FIGURE 1.52.1: Phthiriasis palpebrum

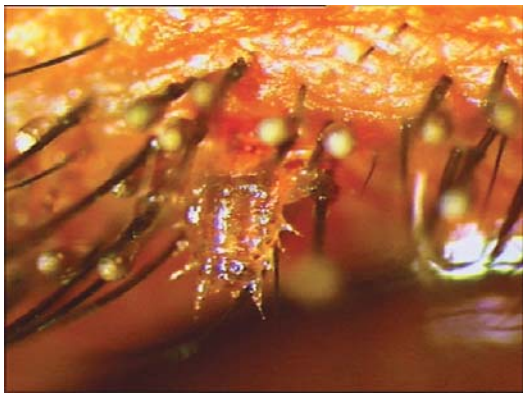


FIGURE 1.52.2: Phthiriasis palpebrum with nits

BENIGN LID CONDITIONS

Xanthelasma

- Raised, yellow plaques, most commonly found at the inner portion of the upper eyelid **(Fig 1.53.1)** rarely in lower lid **(Fig 1.53.2)**
- Often symmetrical and grow slowly
- Eventually it spreads all the four lids **(Figs 1.53.3 and 1.53.4)**
- May be associated with familial hypercholesterolemia
- Produce only a cosmetic defect
- *Treatment:* cosmetic surgery

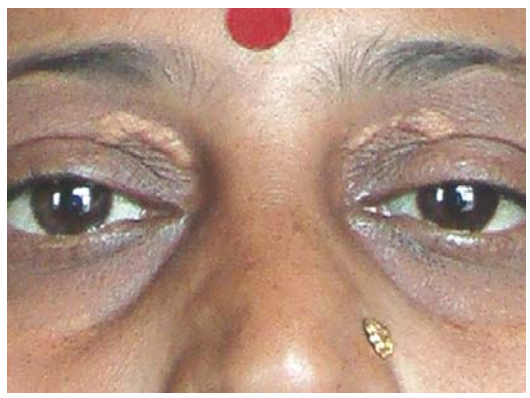


FIGURE 1.53.1: Xanthelasma-upper lids

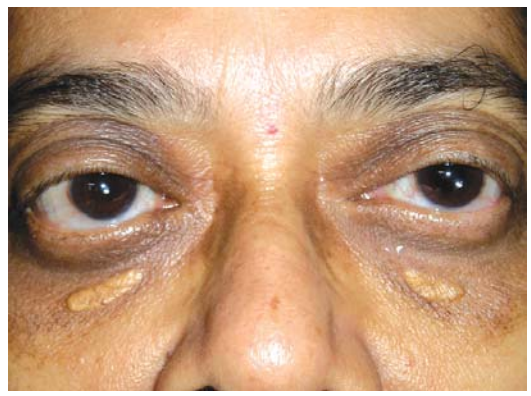


FIGURE 1.53.2: Xanthelasma-lower lids

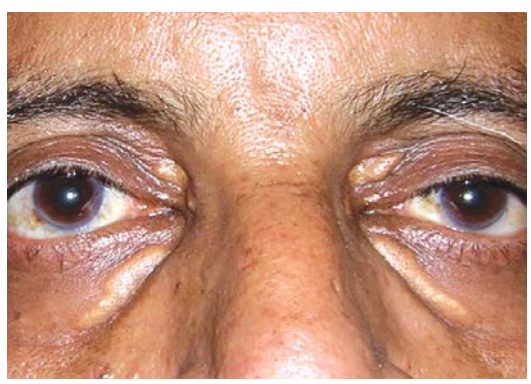


FIGURE 1.53.3: Xanthelasma-all four lids

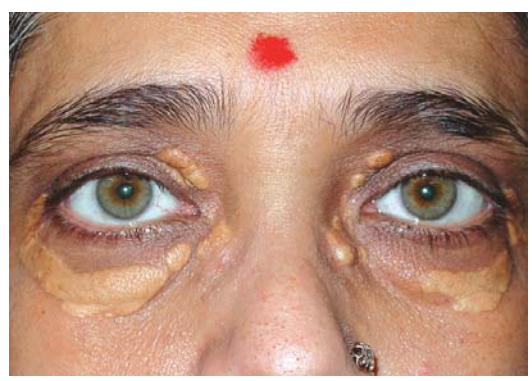


FIGURE 1.53.4: Xanthelasma-circular pattern

Capillary Hemangioma (Strawberry naevus)

- Develops soon after birth, then grows for 6 month to 1 year
- May involute spontaneously in some cases
- Irregular, raised, bright red lesion (**Figs 1.54.1 and 1.54.2**)
- It blanches on pressure and may swell on crying
- Associated with similar skin lesions elsewhere
- *Treatment:* hypertonic saline/corticosteroids injection and laser therapy

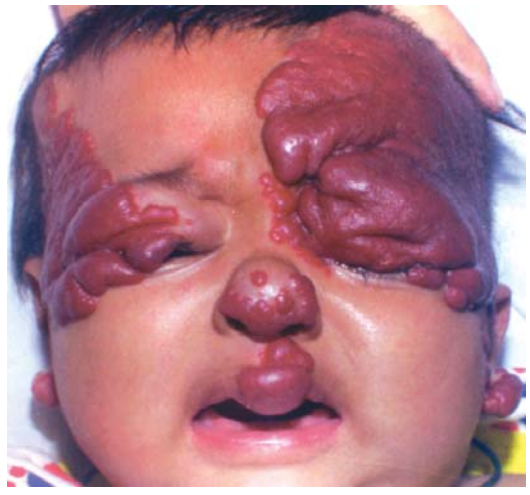


FIGURE 1.54.1: Capillary (strawberry) hemangioma

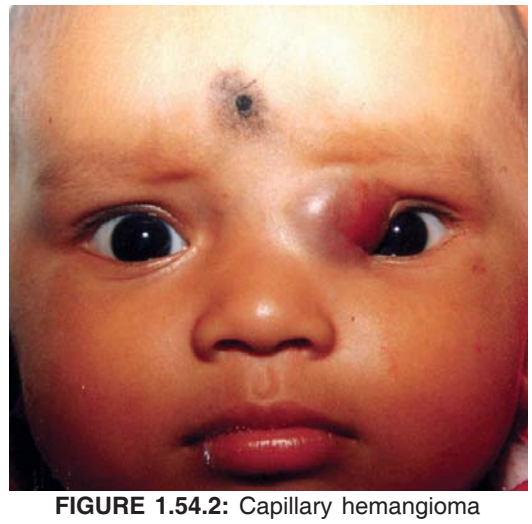


FIGURE 1.54.2: Capillary hemangioma

Port-wine Stain (naevus flammeus)

- Congenital bilateral or unilateral lesion
- Sharply demarcated red to purple patch along the first and second divisions of the fifth nerve (**Fig 1.55.1**)
- Lesion does not blanch on pressure
- Smaller lesion does not have any implication (**Fig 1.55.2**)
- Larger lesion may be associated with Sturge-Weber syndrome (choroidal hemangioma with glaucoma, hemangioma of leptomeninges) (**Fig 1.55.3**)
- *Treatment:* laser therapy and treatment of glaucoma if present

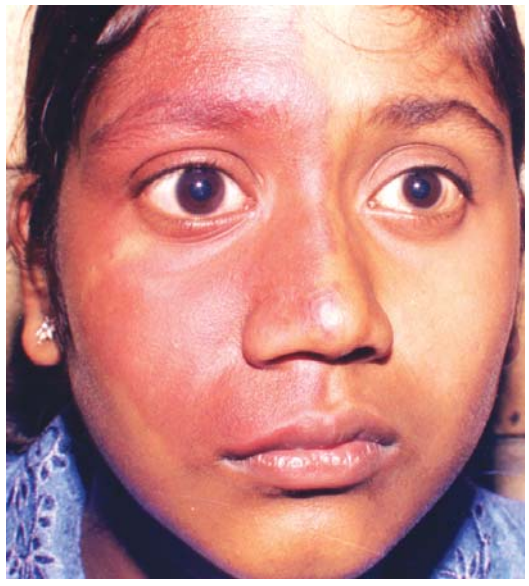


FIGURE 1.55.1: Port-wine stain

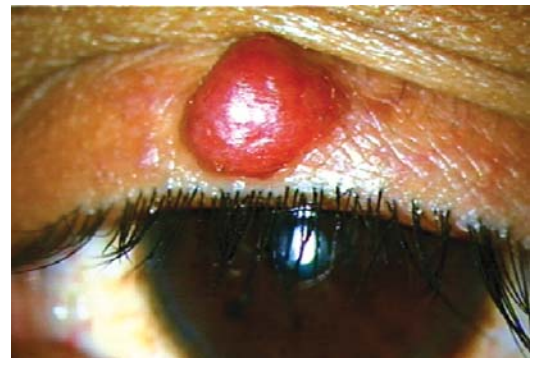


FIGURE 1.55.2: Small naevus flammeus

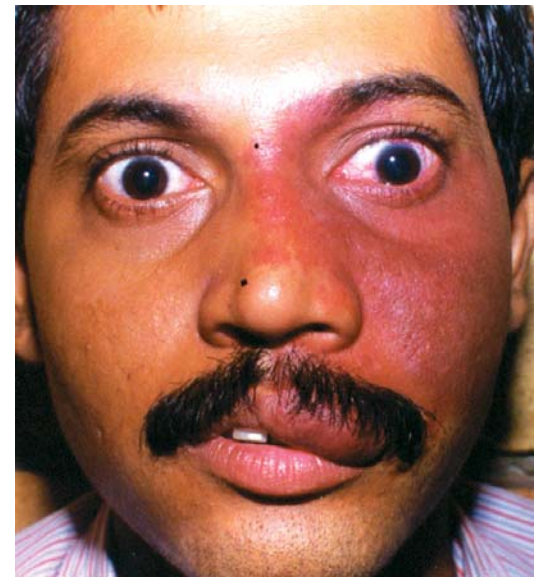


FIGURE 1.55.3: Port-wine stain-Sturge-Weber syndrome

Molluscum Contagiosum

- Unilateral or bilateral lesion with single or multiple lesion
- In immunodeficiency conditions, it may be more severe and confluent, often with other parts of body
- Small, pale, yellowish-white umbilicated lesions (**Fig 1.56.1**)
- Ulceration in severe cases
- May be a presenting feature in AIDS (**Fig 1.56.2**)
- Associated with keratitis or follicular conjunctivitis (**Fig 1.56.3**)
- *Treatment:* investigations for immune deficiency states, chemical cautery in some cases

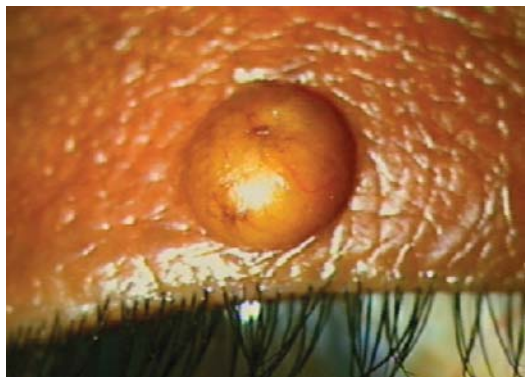


FIGURE 1.56.1: Molluscum contagiosum

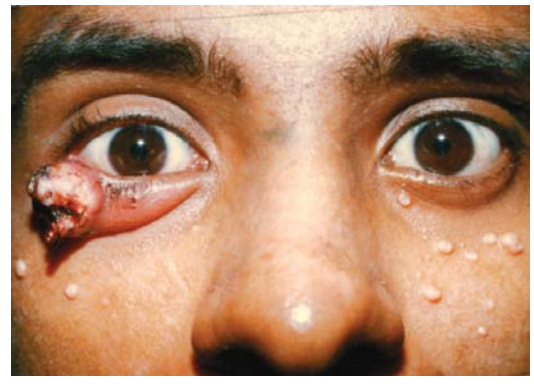


FIGURE 1.56.2: Molluscum contagiosum in AIDS

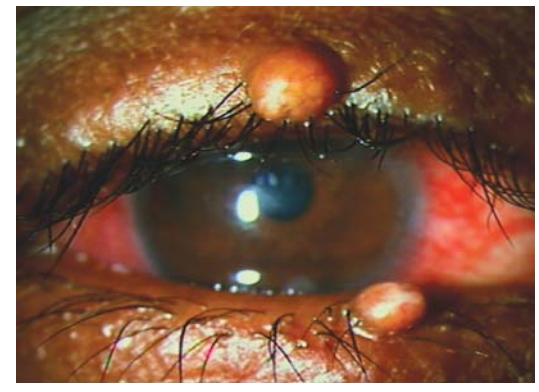


FIGURE 1.56.3: Molluscum contagiosum-keratitis

Sebaceous Cyst

- Bilateral and multiple cysts are more common
- They vary in shape and size
- Solitary cyst is more common near the canthus (**Fig 1.57.1**)
- Yellowish-white color and cyst is filled up with inspissated sebaceous secretion, often large in size (**Figs 1.57.2 and 1.57.3**)
- *Treatment:* excision for cosmetic reason only

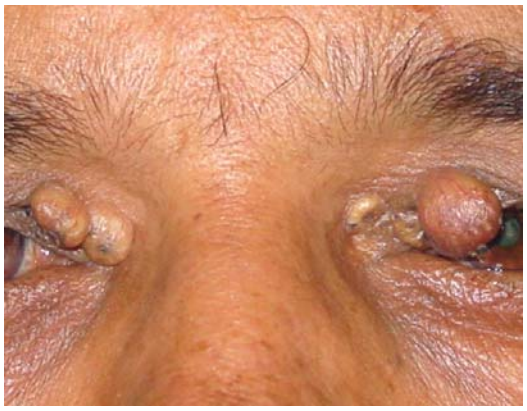


FIGURE 1.57.1: Sebaceous cyst

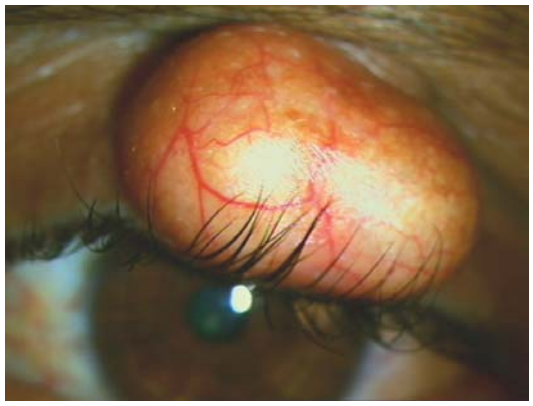


FIGURE 1.57.2: Large sebaceous cyst

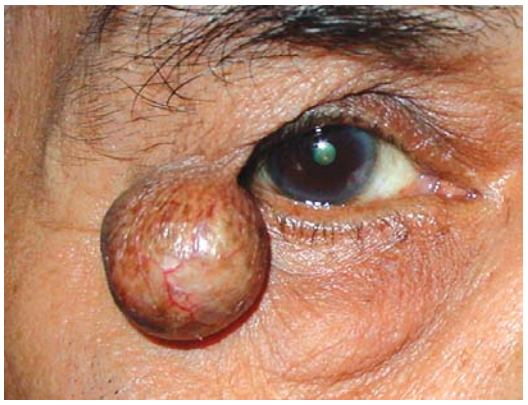


FIGURE 1.57.3: Large sebaceous cyst

Keratoacanthoma

- Rare, fast-growing benign tumor
- Firm, pinkish nodule with roled out edge and a keratin filled crater (**Figs 1.58.1 and 1.58.2**)
- *Treatment:* excision in case of large lesion

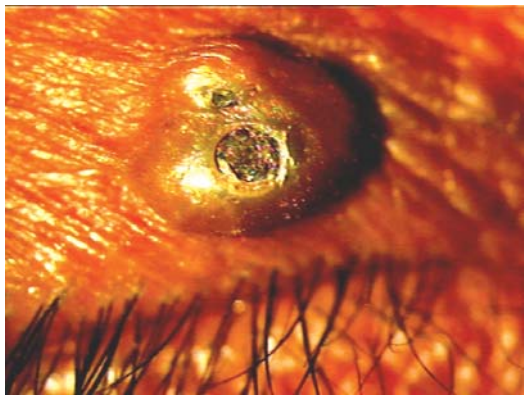


FIGURE 1.58.1: Keratoacanthoma

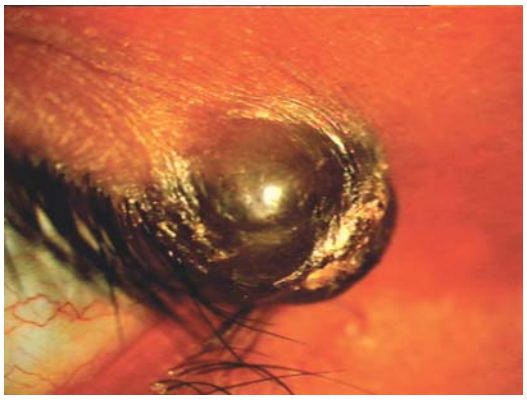


FIGURE 1.58.2: Keratoacanthoma

Keratic Horn

- A rare benign condition
- Hyperkeratotic horn-like lesion which protrudes from skin surface **(Fig 1.59.1)**
- *Treatment:* excision if necessary

FIGURE 1.59.1: Keratic horn

Squamous Cell Papilloma (viral wart)

- Most common benign tumor of the lid
- Pedunculated or sessile lesion with a characteristic irregular raspberry like surface (**Figs 1.60.1 and 1.60.2**)
- *Treatment:* excision if necssary

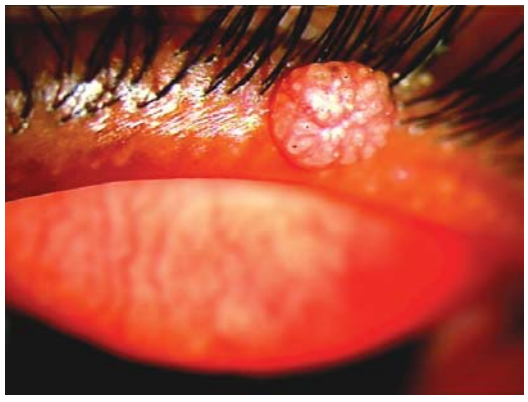


FIGURE 1.60.1: Squamous cell papilloma

FIGURE 1.60.2: Squamous cell papilloma

Basal Cell Papilloma (seborrheic keratosis)

- Common in middle and elderly people
- Discrete, round, brownish or blackish lesion with varrigated surface (**Fig 1.61.1**)
- It may have papillomatous appearence (**Fig 1.61.2**)
- *Treatment:* excision if necessary in severe cases

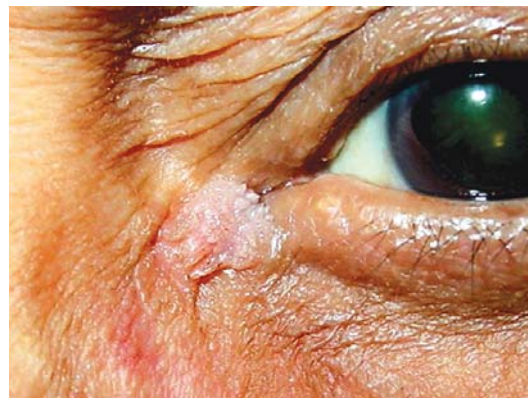


FIGURE 1.61.1: Basal cell papilloma

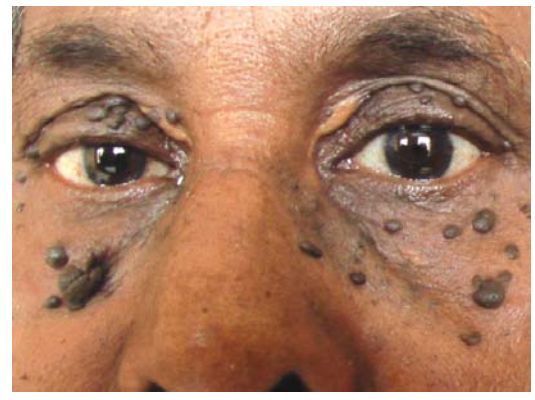


FIGURE 1.61.2: Multiple basal cell papilloma

Oculodermal Melanocytosis (naevus of Ota)

- Rare, congenital condition
- Bluish-gray discoloration of the skin affecting the 5th nerve **(Fig 1.62.1)**
- Associated conjunctival melanosis
- Hyperpigmentation of the iris (heterochromia)

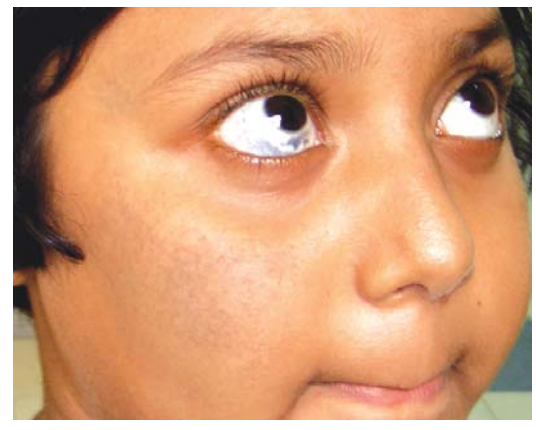


FIGURE 1.62.1: Oculodermal melanocytosis

Acquired Naevus

- *Three types:* junctional **(Fig 1.63.1)**, compound **(Fig 1.63.2)** and dermal **(Fig 1.63.3)**
- Elevated or flat lesions with variable degree of brown to black pigmentation
- Lash may protrude through this lesion **(Fig 1.63.4)**
- May be kissing type in any variety **(Fig 1.63.5)**
- *Treatment:* surgical excision for cosmetic reason

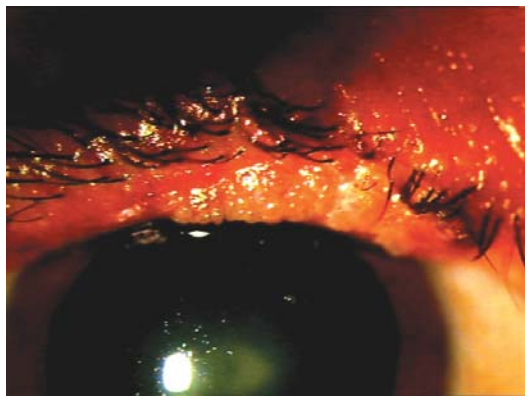


FIGURE 1.63.1: Junctional naevus

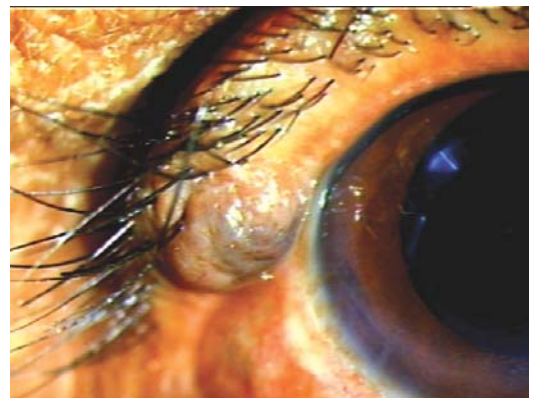


FIGURE 1.63.2: Compound naevus

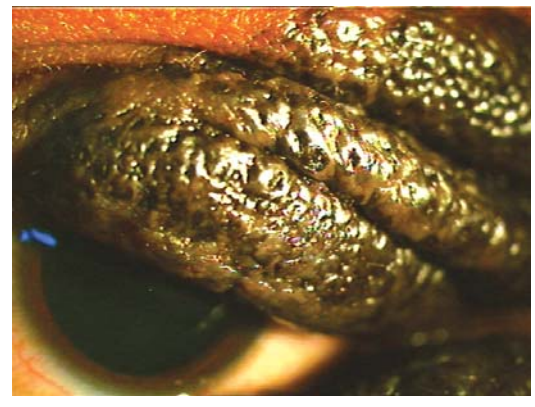


FIGURE 1.63.3: Dermal naevus

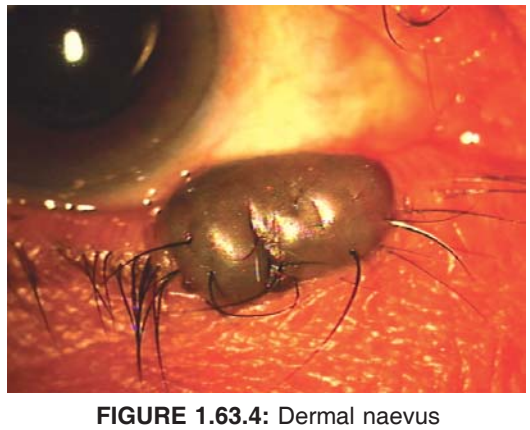


FIGURE 1.63.4: Dermal naevus

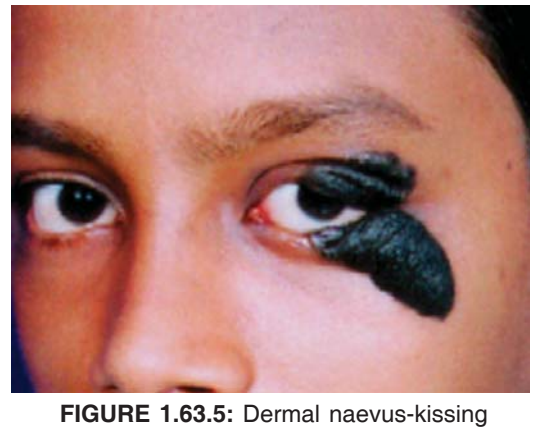


FIGURE 1.63.5: Dermal naevus-kissing

Milia

- Small, multiple, round, superficial cysts **(Fig 1.64.1)**
- It tends to occurs in crops and often bilateral
- No treatment is required

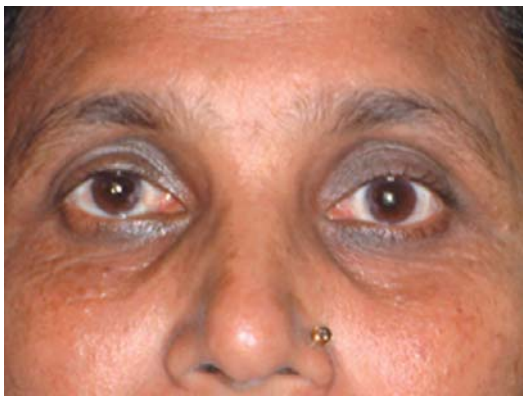


FIGURE 1.64.1: Milia

External Angular Dermoid

- Not to rare condition, present since early infancy
- Smooth, subcutaneous, firm, slow growing swelling most frequently located just below the lateral eyebrow **(Figs 1.67.1 and 1.67.2)**
- May be associated with bony orbital defect
- *Treatment*: excision of the mass, internal extension should be dissected carefully

Cyst of Moll

- Very common, painless, chronic, transparent cystic nodule just on the lid margin containing serous secretion **(Fig 1.65.1)**

Cyst of Zeis

- Similar kind of cystic swelling on the external aspect of lid margin
- It contains oily secretion

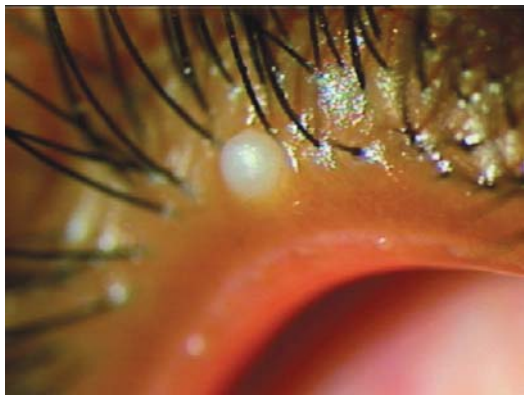


FIGURE 1.65.1: Cyst of Moll

Epidermal Inclusion Cyst

- Rare condition, due to trauma or after surgery
- Solitary, firm slowly progressive subepithelial nodule **(Fig 1.66.1)**
- Most commonly found in upper lid, may be mistaken as a chalazion
- *Treatment:* removal of cyst from skin surface

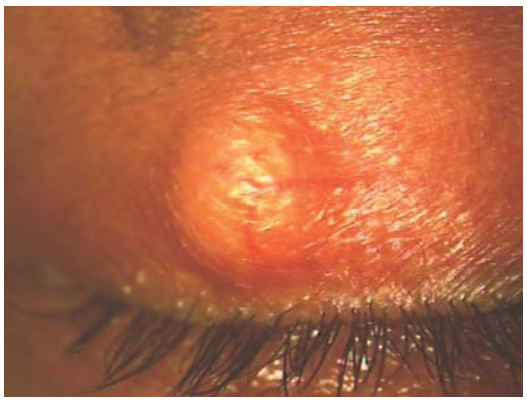


FIGURE 1.66.1: Epidermal inclusion cyst

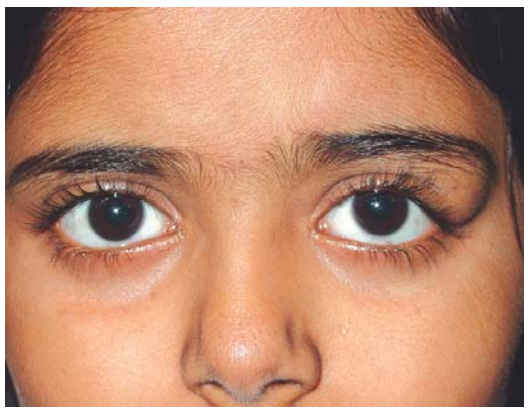


FIGURE 1.67.1: External angular dermoid

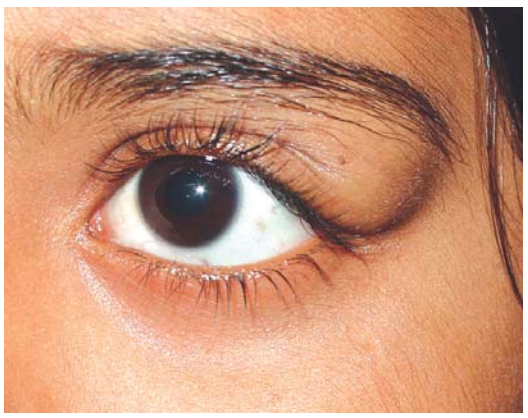


FIGURE 1.67.2: External angular dermoid

MALIGNANT LID CONDITIONS

Basal Cell Carcinoma (rodent ulcer)

- Most common malignant tumor of the eyelid
- The lower lid is more commonly involved, specially near the inner canthus **(Fig 1.68.1)**
- Sometimes in upperlid **(Fig 1.68.2)**
- The edges are raised (rolled out edges) and indurated **(Fig 1.68.3)**

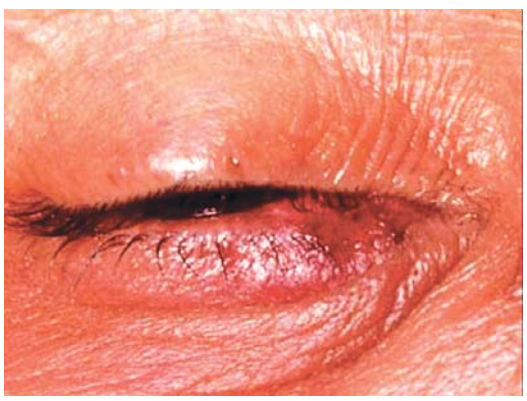


FIGURE 1.68.1: Basal cell carcinoma-ulcerative

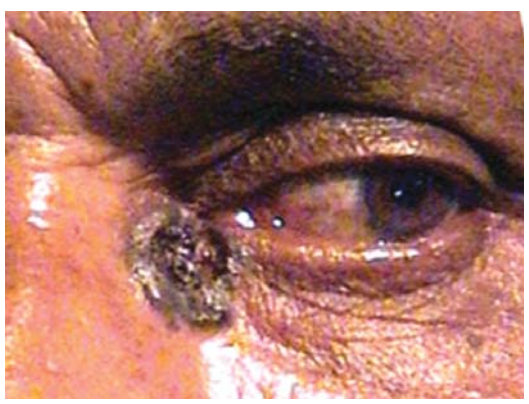


FIGURE 1.68.2: Basal cell carcinoma-sclerosing

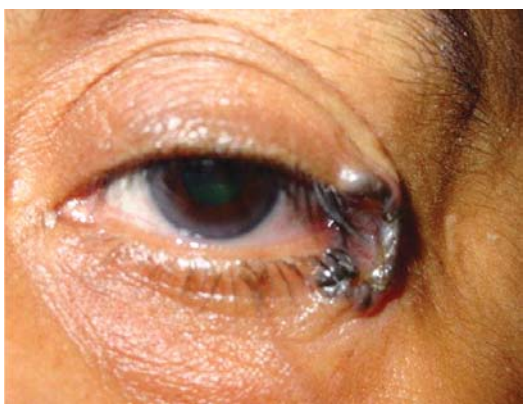


FIGURE 1.68.3: Basal cell carcinoma

- Ulcer spreads very slowly **(Fig 1.68.4)**
- Ulcer may erode the lid margin like a bridge **(Figs 1.68.5 and 1.68.6)**
- It does not metastasize to lymph
- *Treatment:* surgical excision, radiotherapy

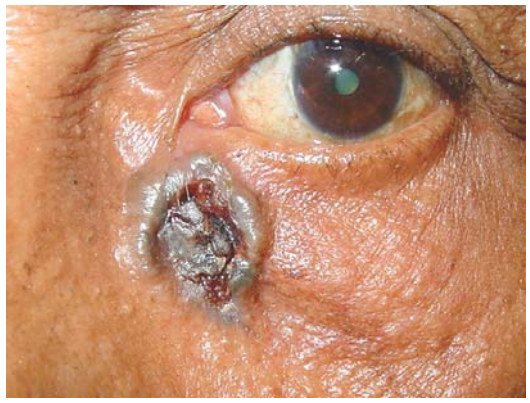


FIGURE 1.68.4: Basal cell carcinoma-noduloulcerative

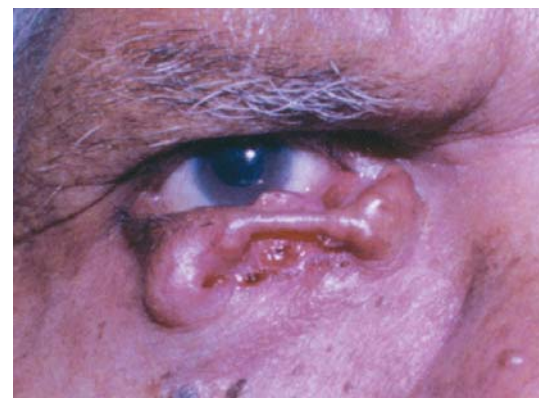


FIGURE 1.68.5: Basal cell carcinoma-bridge lesion

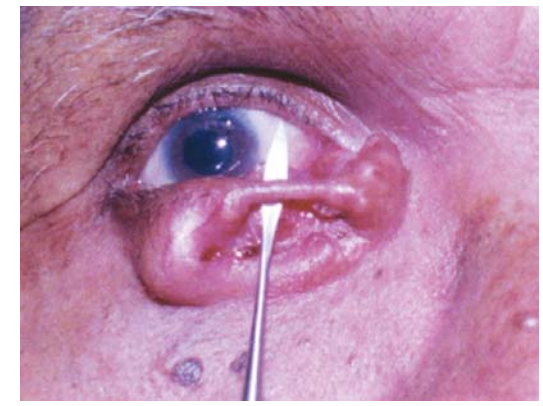


FIGURE 1.68.6: Basal cell carcinoma-bridge lesion

Squamous Cell Carcinoma

Second most common malignancy of the eyelid

- Appears as a nodule or an ulcerative lesion **(Fig 1.69.1)**, or a papilloma (**Fig 1.69.2**)
- Sometimes the growth covers the whole eye ball (**Fig 1.69.3**)
- Growth rate is faster than basal cell carcinoma
- It metastasizes into the regional lymph nodes
- *Treatment*: radical surgery with reconstruction

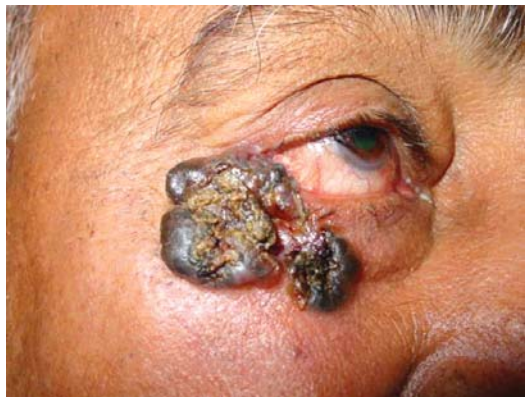


FIGURE 1.69.1: Squamous cell carcinoma-ulcerative

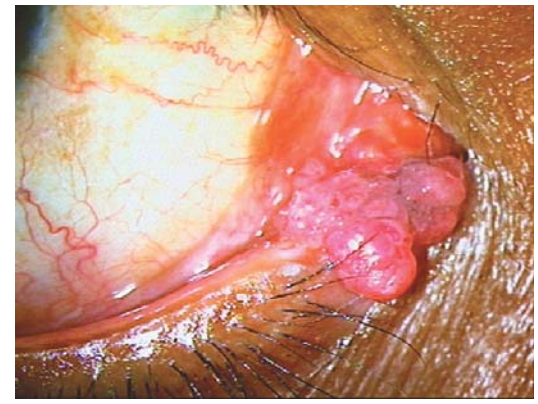


FIGURE 1.69.2: Squamous cell carcinoma-papilloma

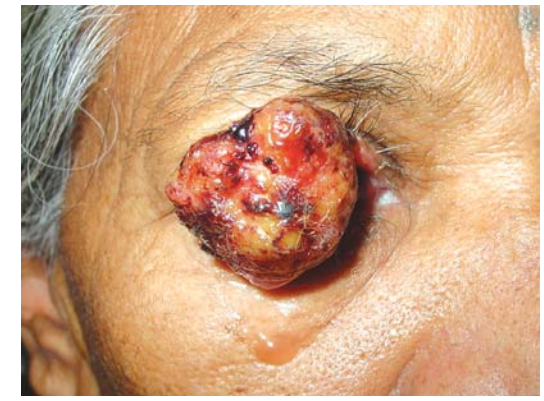


FIGURE 1.69.3: Squamous cell carcinoma-nodular

Meibomian Gland Carcinoma

- Appears as a discrete, yellow, firm nodule which is sometimes incorrectly diagnosed as 'recurrent chalazion' (**Figs 1.70.1 and 1.70.2**)
- Wide-spread metastasis is common in large tumor **(Figs 1.70.3 to 1.70.5)**
- Prognosis is poor
- *Treatment*: radical excision with reconstruction of the involved lid

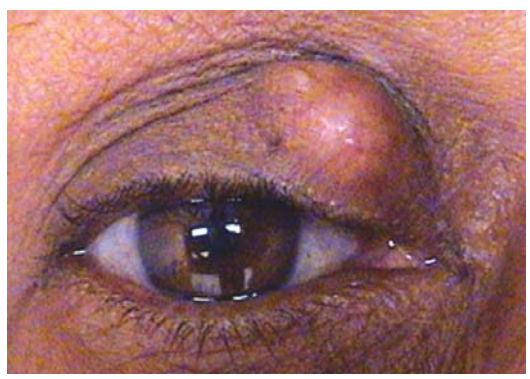


FIGURE 1.70.1: Meibomian carcinoma

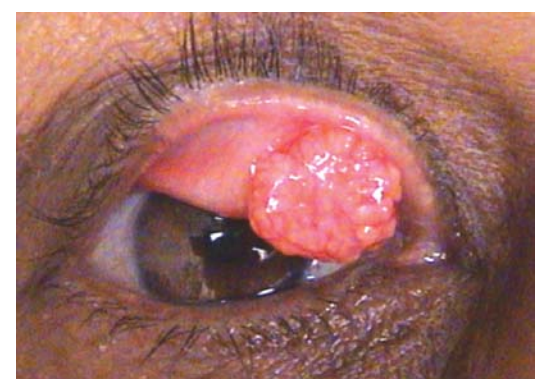


FIGURE 1.70.2: Meibomian carcinoma

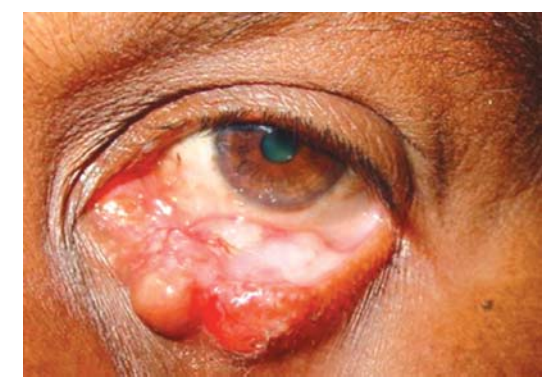


FIGURE 1.70.3: Meibomian carcinoma

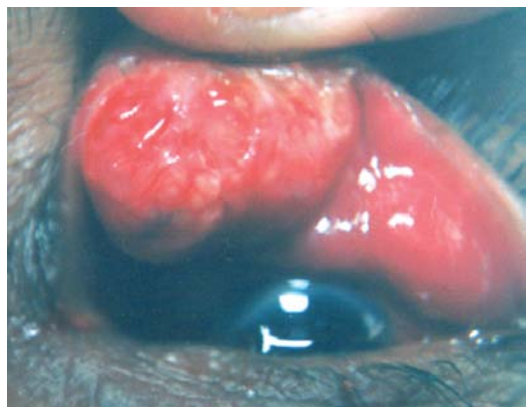


FIGURE 1.70.4: Meibomian carcinoma-large

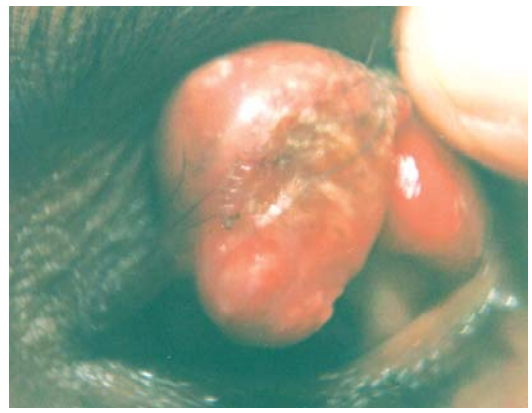


FIGURE 1.70.5: Meibomian carcinoma-large

Carcinoma of Gland of Zeis

- Affects elderly patients
- Discrete nodular slow growing tumor on the lid margin **(Fig 1.71.1)**
- May be associated with loss of local eyelashes

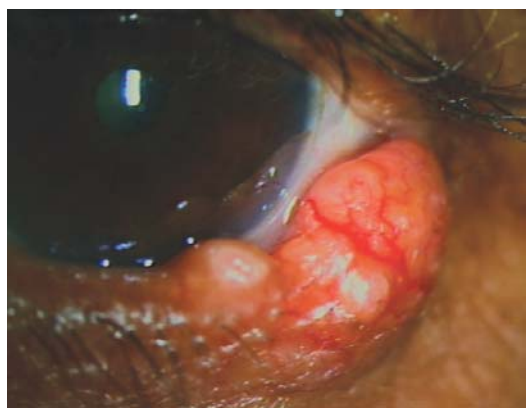


FIGURE 1.71.1: Carcinoma of gland of Zeis

OTHER MALIGNANT LID CONDITIONS

Lentigo Malignum

- Affects elderly patients
- Preinvasive stage of malignant melanoma
- Pigmented macular lesion may transform to nodular lesion **(Fig 1.72.1)**

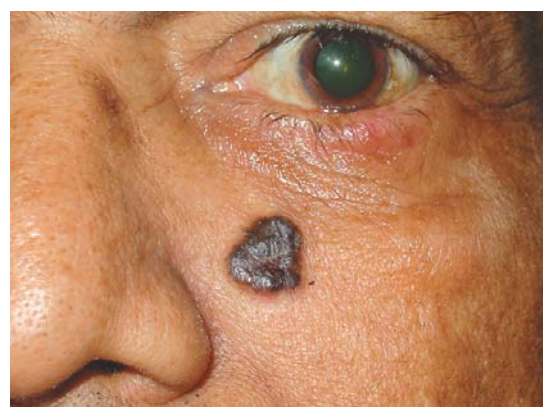


FIGURE 1.72.1: Lentigo malignum

Nodular Melanoma

- May arise *de novo* or from pre-cancerous melanoma **(Fig 1.73.1)**
- Large nodule with irregular pigmentation **(Fig 1.73.2)**
- May show rapid growth with break down of epithelium
- *Treatment*: radical excision or exenteration

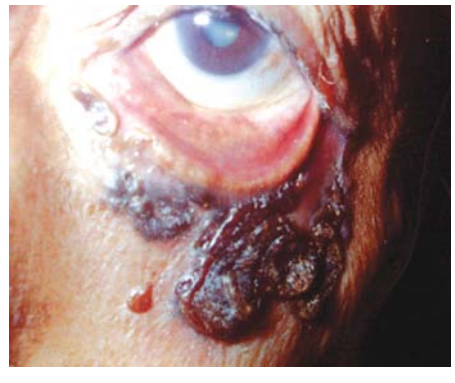


FIGURE 1.73.1: Precancerous melanoma

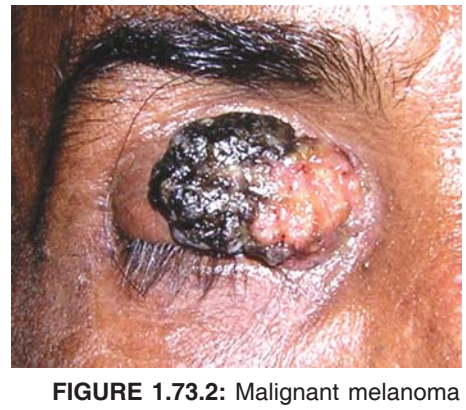


FIGURE 1.73.2: Malignant melanoma

MISCELLANEOUS LID CONDITIONS

Baggy Eyelids

- Usually bilateral, age related condition
- Due to orbital fat herniation through the weakened orbital septum **(Fig 1.74.1)**
- Initially, fat-pockets herniate into the medial aspect of the upper lid, then lower lids **(Fig 1.74.2)**
- *Treatment*: cosmetic surgical correction, but recurrence is common

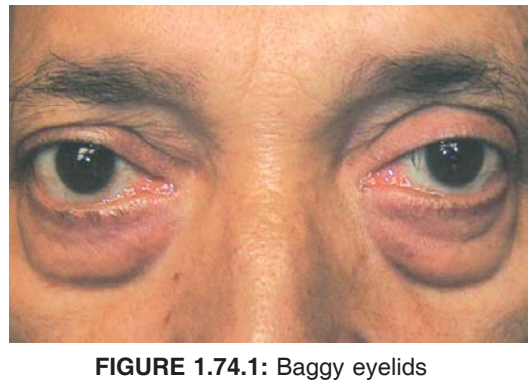


FIGURE 1.74.1: Baggy eyelids

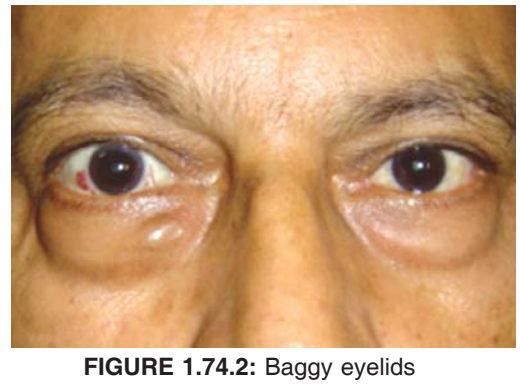


FIGURE 1.74.2: Baggy eyelids

Depigmentation of Periocular Skin

- Rare, unilateral condition
- *Causes*
  - VKH syndrome **(Fig 1.75.1)**
  - sympathetic ophthalmia
  - drug induced; like topical sparfloxacin, latanoprost, *etc*. **(Fig 1.75.2)**

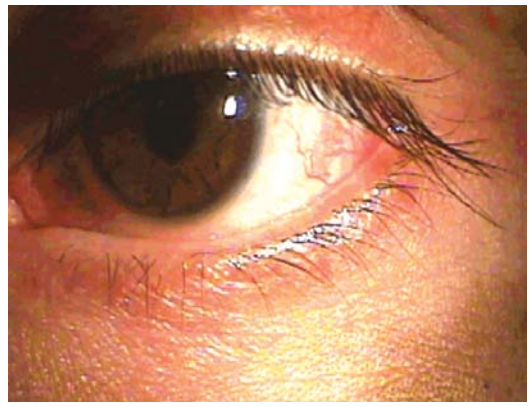


FIGURE 1.75.1: Periocular pigmentation-VKH

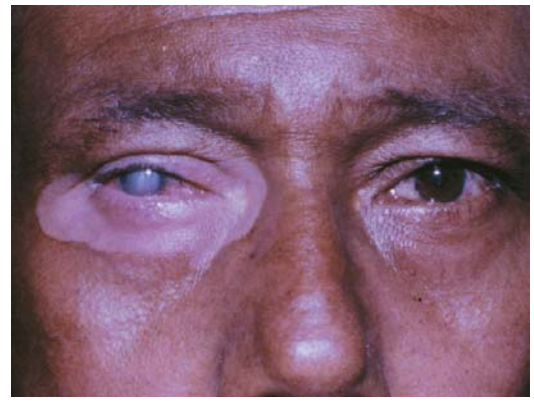


FIGURE 1.75.2: Periocular pigmentation-drug induced

Ankyloblepharon

- Adhesion between upper and lower eyelids
- May be partial or complete
- *Causes*
  - congenital **(Fig 1.76.1)**
  - acquired: due to chemical burn **(Figs 1.76.2 and 1.76.3)**
- Eye ball is usually disorganized

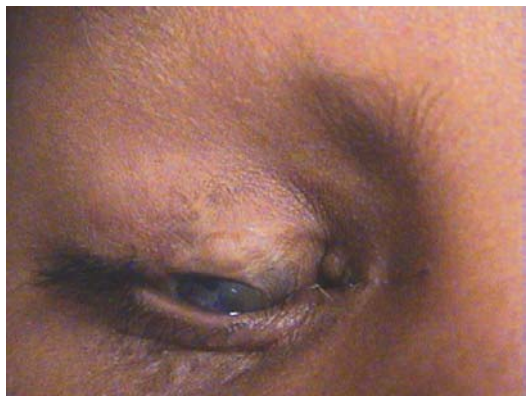


FIGURE 1.76.1: Ankyloblepharon-congenital

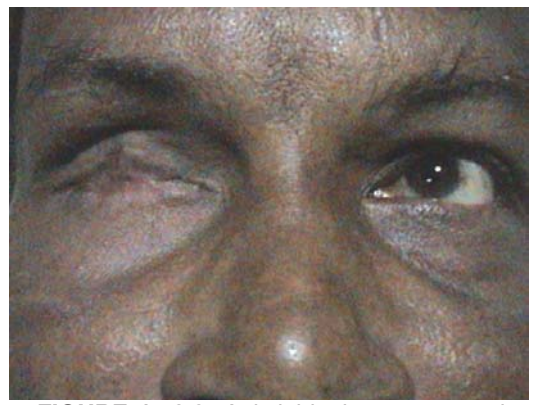


FIGURE 1.76.2: Ankyloblepharon-acquired

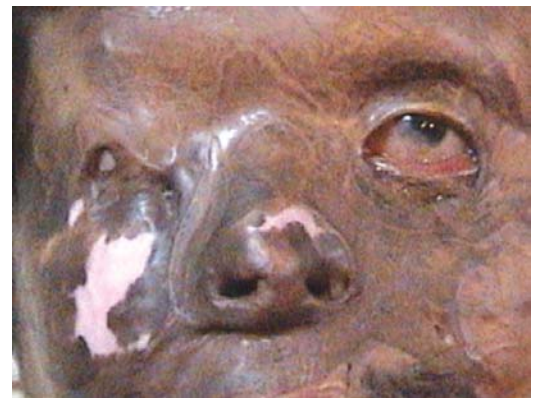


FIGURE 1.76.3: Ankyloblepharon-severe acid burn

Nodular Hemangiomas

- Localized solitary nodular lesion just away from the lid margin **(Figs 1.77.1 to 1.77.3)**
- Bright red in color
- *Treatment:* excision and histopathological examination which confirms the diagnosis

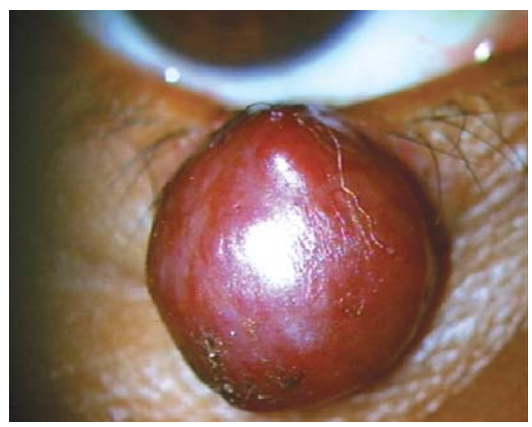


FIGURE 1.77.1: Capillary hemangioma-isolated nodular

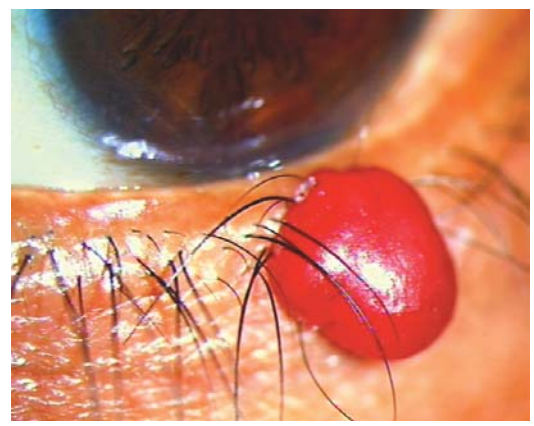


FIGURE 1.77.2: Capillary hemangioma-isolated nodular

2 Diseases of the Conjunctiva

SIGNS OF CONJUNCTIVAL DISEASES

- Hyperemia of the conjunctiva
- Congestion
- Subconjunctival hemorrhage
- Edema (chemosis)
- Follicles
- Papillae
- Membranes
- Pre-auricular lymphadenopathy

ACUTE CONJUNCTIVITIS

- Acute muco-purulent conjunctivitis
- Purulent conjunctivitis
- Ophthalmia neonatorum
- Membranous conjunctivitis
- Angular conjunctivitis
- Acute follicular conjunctivitis

CHRONIC CONJUNCTIVITIS

- Simple chronic conjunctivitis
- Trachoma

ALLERGIC CONJUNCTIVITIS

- Phlyctenular conjunctivitis
- Vernal conjunctivitis (keratoconjunctivitis)
- Giant papillary conjunctivitis

CONJUNCTIVAL DEGENERATIONS

- Concretion (lithiasis)
- Pinguecula
- Pterygium

BENIGN LESIONS

- Conjunctival cysts
- Epibulbar limbal dermoid
- Dermolipoma (lipodermoid)
- Choriostomas

MALIGNANT LESION

- Conjunctival intra-epithelial neoplasia
- Invasive squamous-cell carcinoma

PIGMENTED LESIONS

- Flat superficial pigmentation
- Benign epithelial melanosis
- Benign subepithelial melanocytosis
- Simple naevus
- Melanocytoma
- Pre-cancerous melanosis
- Malignant melanoma

OTHER CONJUNCTIVAL CONDITIONS

- Subconjunctival hemorrhage
- Conjunctival xerosis
- Bitot's spot
- Keratoconjunctivitis sicca
- Mucus fishing syndrome
- Lid imbrication syndrome
- Ocular cicatricial pemphigoid
- Stevens-Johnson syndrome
- Superior limbic keratoconjunctivitis
- Vascular malformations
- Non-Hodgkin lymphoma
- Amyloidosis of the conjunctiva
- Conjunctivochalasis
- Conjunctival granulomas
- Xerodermal pigmentosa

SIGNS OF CONJUNCTIVAL DISEASES

Hyperemia of the Conjunctiva

- Passive dilatation of the conjunctival blood vessels (**Fig 2.1.1**)
- Without exudation or cellular infiltration
- Mainly occur due to irritation
- Only redness, no other symptoms (**Fig 2.1.2**)
- Temporary blanching is noted with 1 in 1000 epinephrine solution

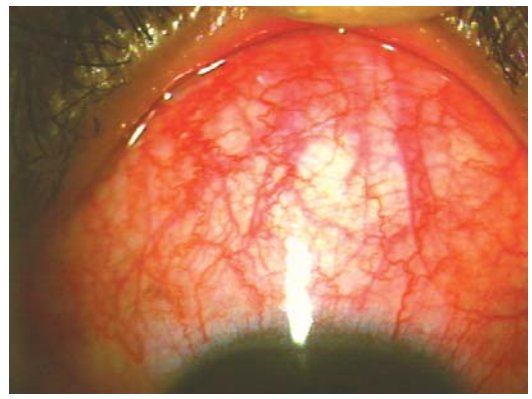


FIGURE 2.1.1: Conjunctival hyperemia

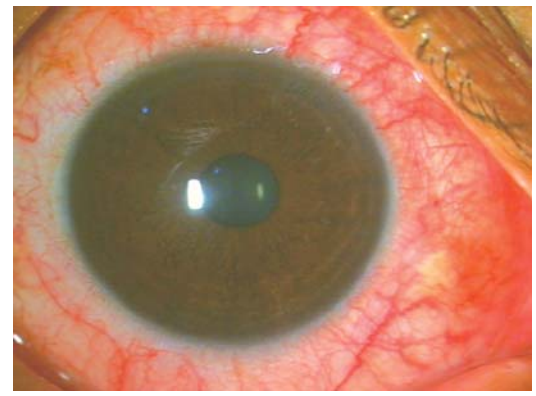


FIGURE 2.1.2: Conjunctival hyperemia

Congestion

- *Conjunctival congestion:*
  - bright red, superficial vessels more intense at fornices (**Fig 2.2.1**)
  - branched dichotomously (**Fig 2.2.2**)
  - seen in conjunctivitis
- *Ciliary congestion:*
  - deeper vessels, dusky red in color and seen mostly at limbus
  - arranged in radial fashion
  - seen in keratitis **(Fig 2.2.3)**, iridocyclitis **(Fig 2.2.4)** or angle closure glaucoma (**Fig 2.2.5**)

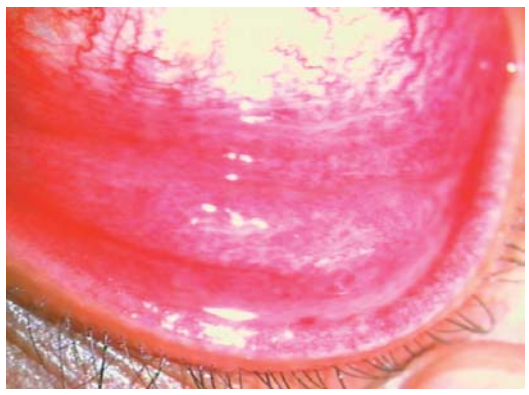


FIGURE 2.2.1: Conjunctival congestion

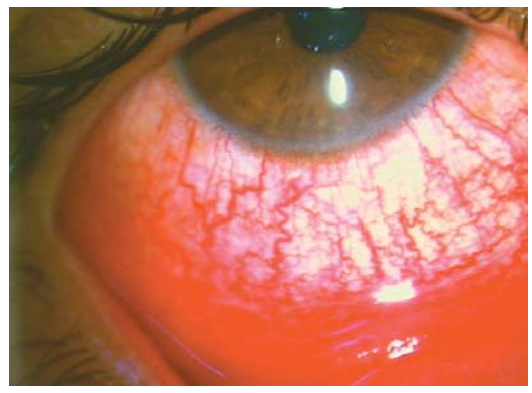


FIGURE 2.2.2: Conjunctival congestion

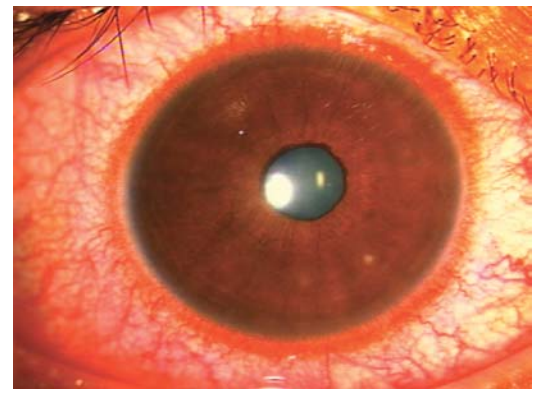


FIGURE 2.2.3: Ciliary congestion—keratitis

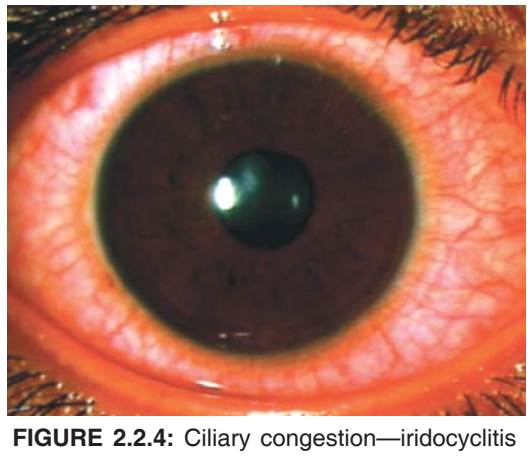


FIGURE 2.2.4: Ciliary congestion—iridocyclitis

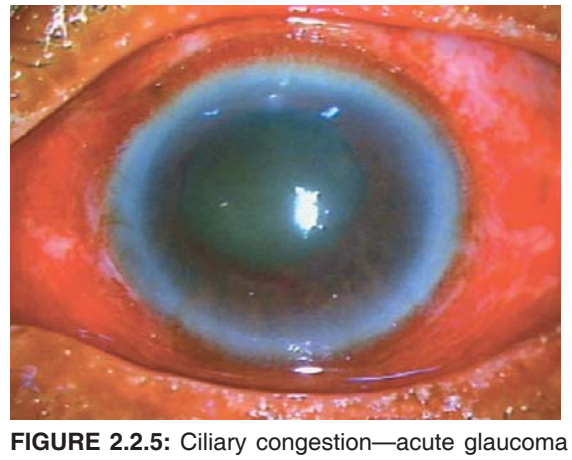


FIGURE 2.2.5: Ciliary congestion—acute glaucoma

Subconjunctival Hemorrhage

- Acute hemorrhagic conjunctivitis (*Picorna* virus) (**Fig 2.3.1**)
- Adenoviral conjunctivitis
- Bacterial conjunctivitis (*Pneumococcus* or *Haemophilus* spp.)
- It appears as petechial (**Fig 2.3.2**) or frank subconjunctival hemorrhage

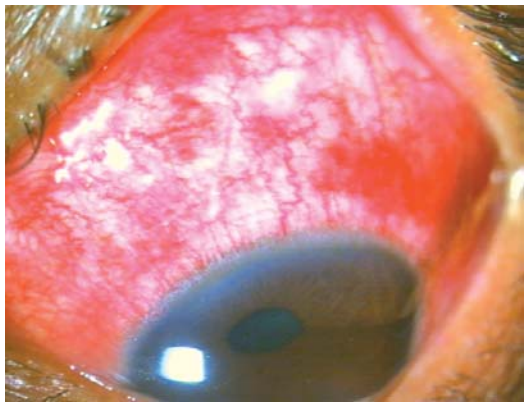


FIGURE 2.3.1: Subconjunctival hemorrhage—conjunctivitis

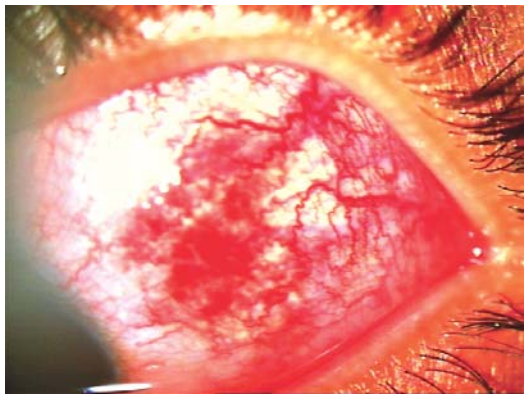


FIGURE 2.3.2: Subconjunctival petechial hemorrhage

Edema (chemosis)

- Large quantity of the exudates causes ballooning of the bulbar conjunctiva **(Fig 2.4.1)**
- Lids are often edematous
- Seen in severe bacterial conjunctivitis, orbital inflammation **(Fig 2.4.2)** and acute allergic inflammation **(Fig. 2.4.3)**

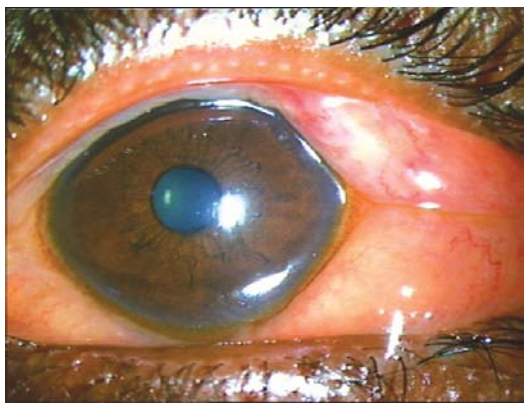


FIGURE 2.4.1: Chemosis of the conjunctiva

FIGURE 2.4.2: Chemosis—orbital inflammation

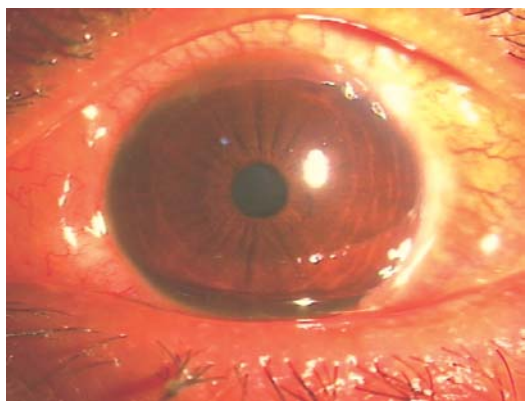


FIGURE 2.4.3: Chemosis—allergic conjunctivitis

Follicles

- Round swellings, (0.5-2 mm in diameter)
- Each follicle is encircled by tiny blood vessels **(Fig 2.5.1)**
- Mostly seen in fornices and palpebral conjunctiva **(Fig 2.5.2)**
- Seen in follicular conjunctivitis, trachoma, toxin or drug-induced

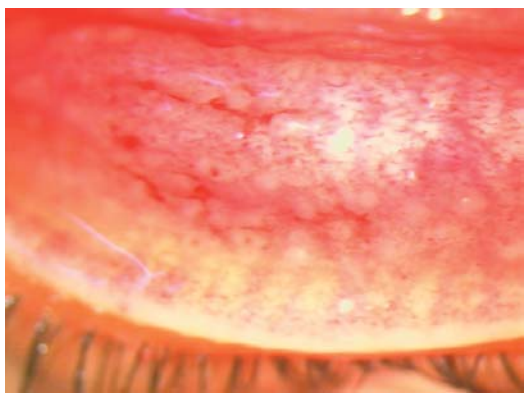


FIGURE 2.5.1: Conjunctival follicles

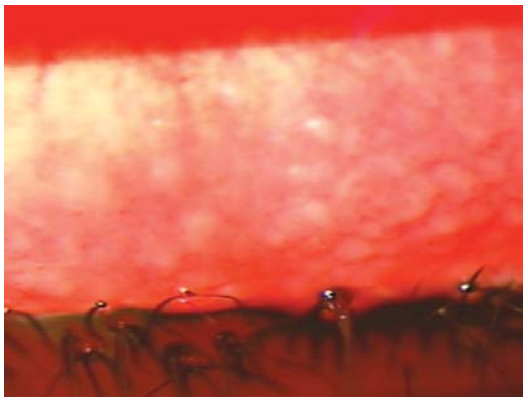


FIGURE 2.5.2: Conjunctival follicles

Papillae

- Vascular structures invaded by inflammatory cells with hyperplasia of the normal system of vascularization (**Fig 2.6.1**)
- They are hyperemic flat-topped elevations on the tarsal conjunctiva from simple papillary hyperplasia to giant papillae
- Seen in allergic conjunctivitis, trachoma and giant papillary conjunctivitis
- Small papillae along the medial aspect of upper tarsal conjunctiva is a normal finding
- *Grades of papillae:*
  - *Grade 0* = Normal tarsal conjunctiva, no papilla (**Fig 2.6.2**)
  - *Grade 1* = Multiple small papillae with smooth velvety appearance (**Fig 2.6.3)**
  - *Grade 2* = Micropapillae, each with a diameter of 0.3 -1.0 mm (**Fig 2.6.4**)
  - *Grade 3* = Giant papillae, each with a diameter of >1.0 mm (**Fig 2.6.5**)
  - *Grade 4* = Large enormous protruding papillae (**Fig 2.6.6**)

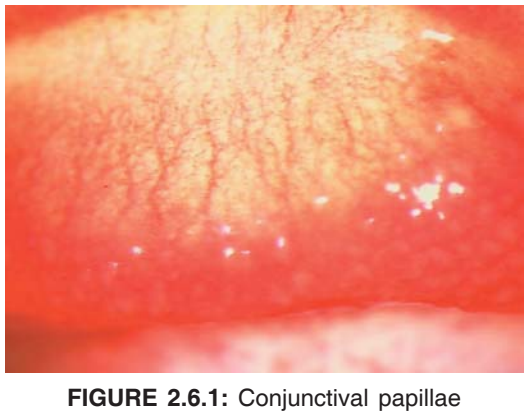


FIGURE 2.6.1: Conjunctival papillae

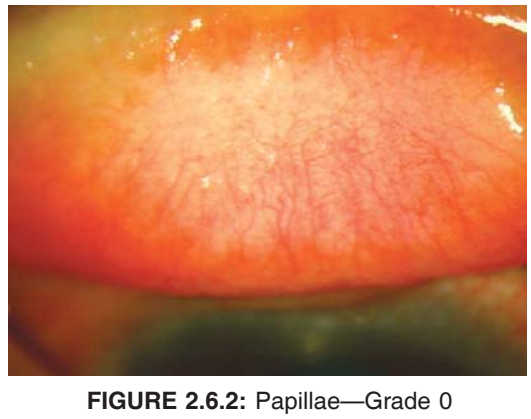


FIGURE 2.6.2: Papillae—Grade 0

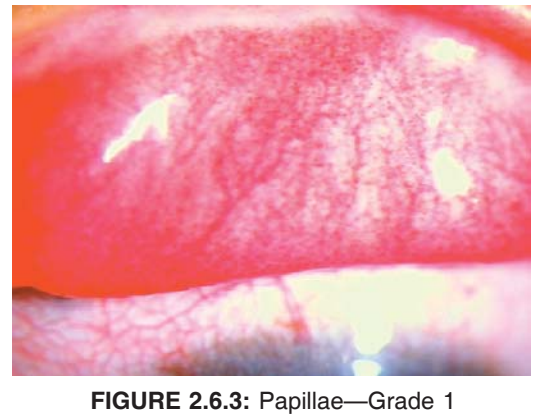


FIGURE 2.6.3: Papillae—Grade 1

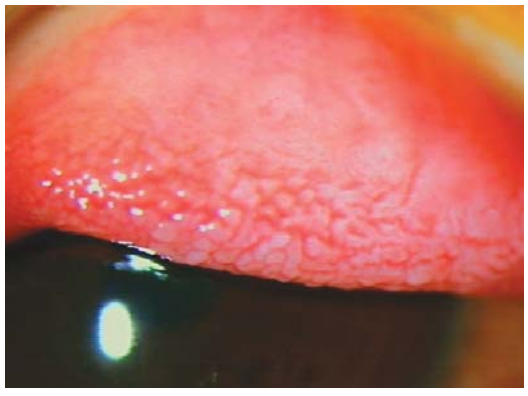


FIGURE 2.6.4: Papillae—Grade 2

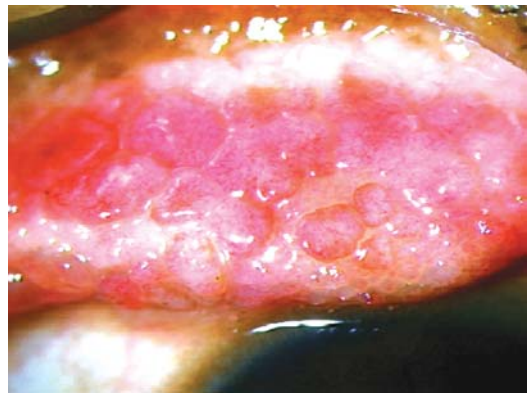


FIGURE 2.6.5: Papillae—Grade 3

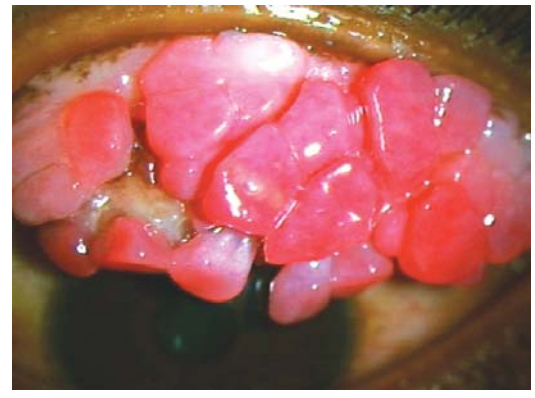


FIGURE 2.6.6: Papillae—Grade 4

Membranes

- *True membranes:* (**Fig 2.7.1**)
  - seen in certain bacterial conjunctivitis, especially in diphtheria
  - attempt to remove the membrane may cause bleeding
- *Pseudo-membranes:* (**Fig 2.7.2**)
  - coagulated exudates loosely adherent to the inflamed conjunctiva **(Fig 2.7.3);** can be easily peeled off without bleeding
  - seen in adenoviral infection, vernal conjunctivitis and other inflammation

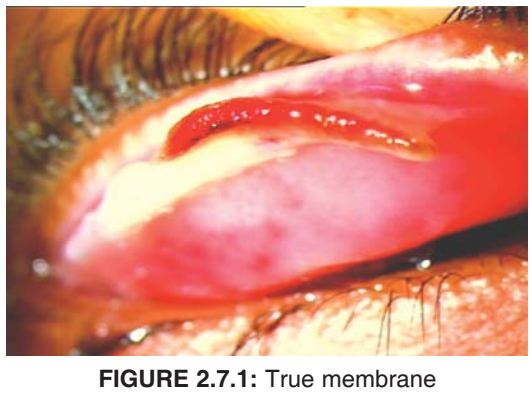


FIGURE 2.7.1: True membrane

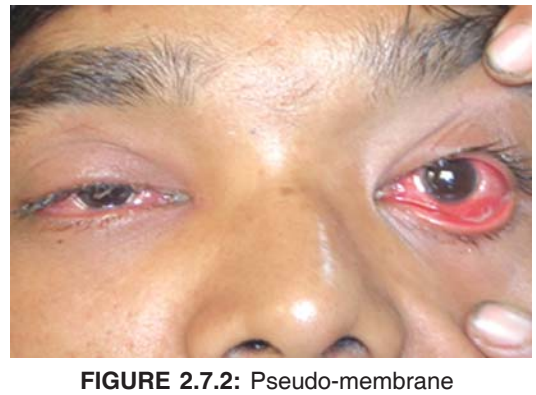


FIGURE 2.7.2: Pseudo-membrane

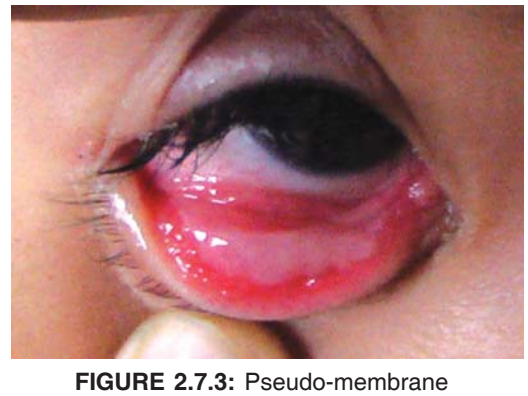


FIGURE 2.7.3: Pseudo-membrane

Pre-auricular Lymphadenopathy

- Mostly seen in *Viral* and *Chlamydial* infection of conjunctiva and sometimes, it may be tender

ACUTE CONJUNCTIVITIS

Acute Muco-purulent Conjunctivitis

- Marked hyperemia, lid edema, matting of the eyelashes **(Fig 2.8.1)**
- Chemosis of the conjunctiva, petechial hemorrhage
- Muco-purulent discharge or flakes of muco-pus **(Fig 2.8.2)**
- May be associated with acute blepharitis **(Fig 2.8.3)**
- *Treatment:*
  - frequent eye wash with lukewarm water
  - broad-spectrum antibiotic eye drops and antibiotic ointment at night
  - prevention of contamination and spread of disease

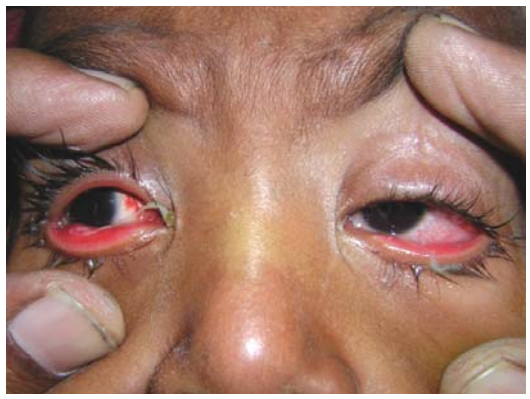


FIGURE 2.8.1: Acute mucopurulent conjunctivitis

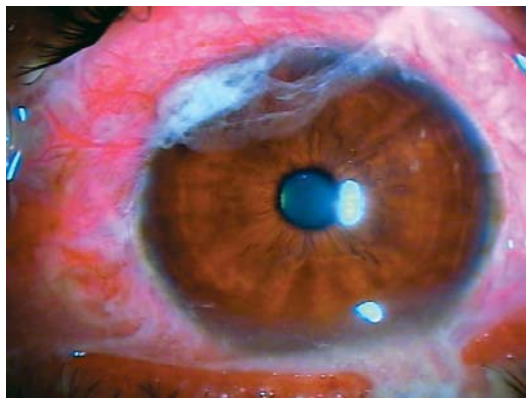


FIGURE 2.8.2: Acute mucopurulent conjunctivitis

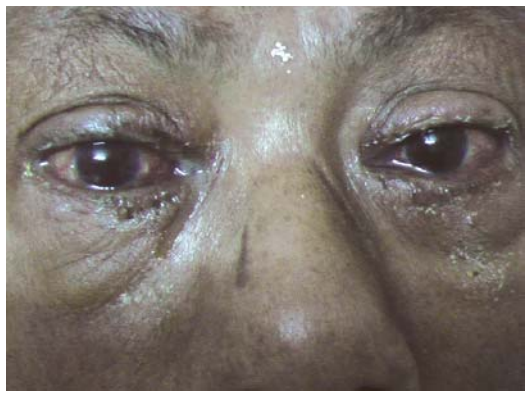


FIGURE 2.8.3: Acute blepharoconjunctivitis

Purulent Conjunctivitis

- Copious purulent discharge
- Right eye is commonly involved (**Fig 2.9.1**)
- Lid edema and crusting of eyelashes
- Conjunctival chemosis with our without membrane formation **(Fig 2.9.2)**
- Cornea may be involved in severe cases
- *Treatment:*
  - patient should be kept in isolation
  - frequent irrigation of eyes with lukewarm normal saline
  - penicillin (1 in 10,000) or ciprofloxacin eye drop hourly
  - ciprofloxacin eye ointment at night
  - systemic antibiotic, if necessary

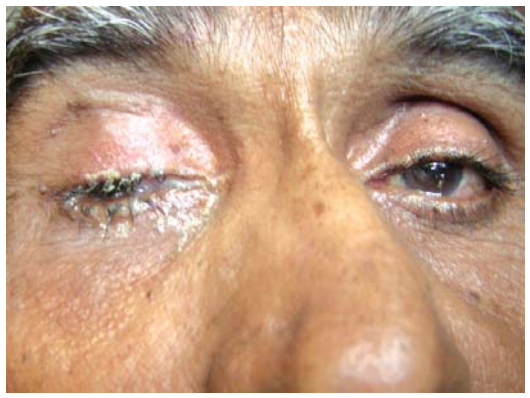


FIGURE 2.9.1: Purulent conjunctivitis RE

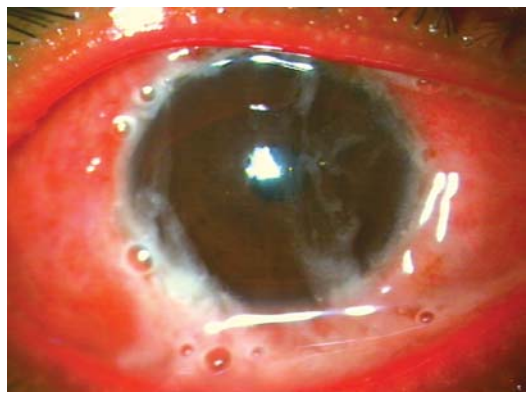


FIGURE 2.9.2: Purulent conjunctivitis

Ophthalmia Neonatorum

- In *Gonococcal* infection, hyperacute purulent conjunctivitis **(Fig 2.10.1)**
- In other cases, it is a catarrhal, or muco-purulent conjunctivitis (**Fig 2.10.2**)
- *Treatment:*
  - ciprofloxacin eye drop—hourly
  - ointment at night
  - for *Chlamydia*—sulphacetamide (10%) eye drop—4 times daily
  - *prevention by:* proper antenatal care, asepsis during delivery, antibiotic eye drop

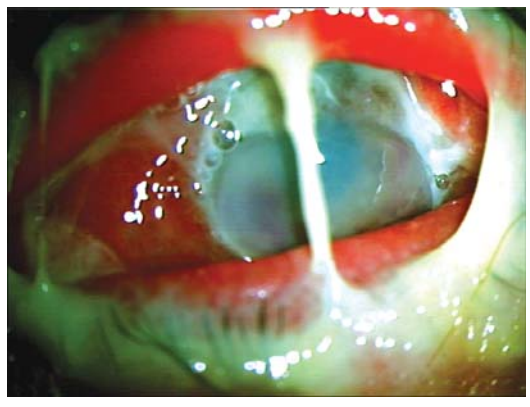


FIGURE 2.10.1: Purulent neonatal conjunctivitis

FIGURE 2.10.2: Neonatal gonococcal conjunctivitis

Membranous Conjunctivitis

- Edema of the lids (**Fig 2.11.1**)
- Mucopurulent or sanious discharge
- Thick white or grayish-yellow membrane on the palpebral conjunctiva **(Fig 2.11.2)**
- Bleeding is very common on removal of the membrane
- Symblepharon may occur in late stage
- *Treatment:* isolation, crystalline penicillin, anti-diphtheric serum, erythromycin eye ointment

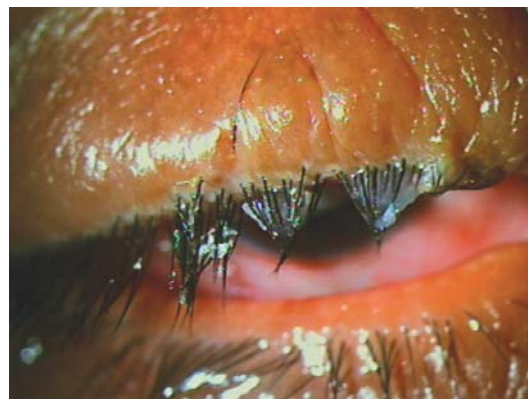


FIGURE 2.11.1: Membranous conjunctivitis

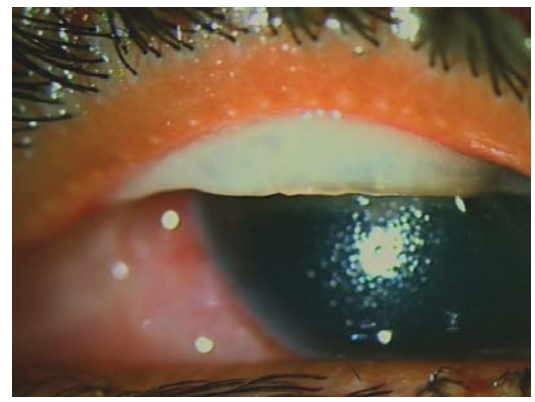


FIGURE 2.11.2: Membranous conjunctivitis

Angular Conjunctivitis

- Conjunctival inflammation is limited to inner-marginal strip (**Fig 2.12.1)** especially at the outer or inner canthi
- Excoriation of the skin at the outer or inner canthi
- Congestion of adjacent bulbar conjunctiva
- *Treatment:* oxytetracycline (1%) eye ointment and zinc oxide containing eye drop

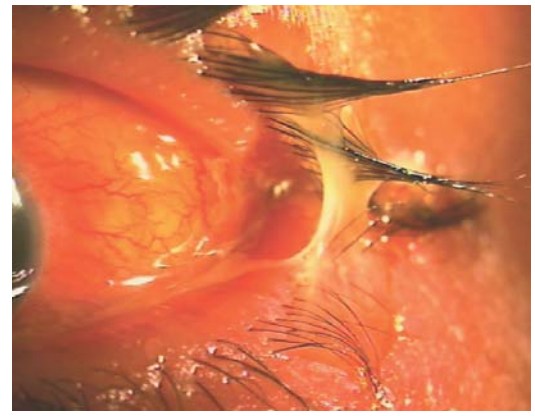


FIGURE 2.12.1: Angular conjunctivitis

Acute Follicular Conjunctivitis

- Inflammation of the conjunctiva with appearance of follicles (**Figs 2.13.1 and 2.13.2)**
- Always a tendency of corneal involvement, the typical lesion is a kerato-conjunctivitis and petechial hemorrhage **(Fig 2.13.3)**
- *Inclusion conjunctivitis:* associated with superficial punctate keratitis (SPKs) and pannus formation
- *Epidemic keratoconjunctivitis:* always associated with subepithelial infiltrates and SPKs **(Fig 2.13.4)**
- *Pharyngoconjunctival fever:* SPKs are rare
- *Acute herpetic keratoconjunctivitis:* Follicles are usually large and SPKs with small dendritic lesion on the cornea
- *Treatment:* astringents eye drop, sulphacetamide in inclusion conjunctivitis and acyclovir in herpetic infection; tear substitutes in corneal involvement

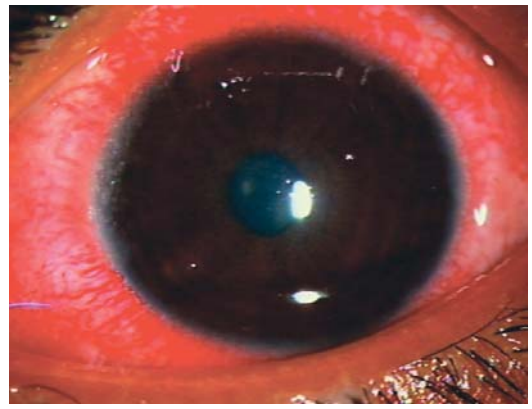


FIGURE 2.13.1: Acute follicular conjunctivitis

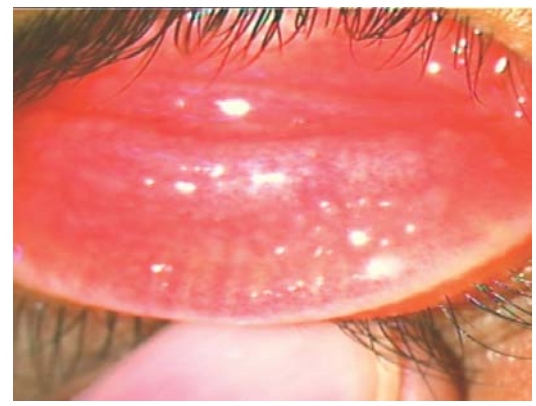


FIGURE 2.13.2: Acute follicular conjunctivitis

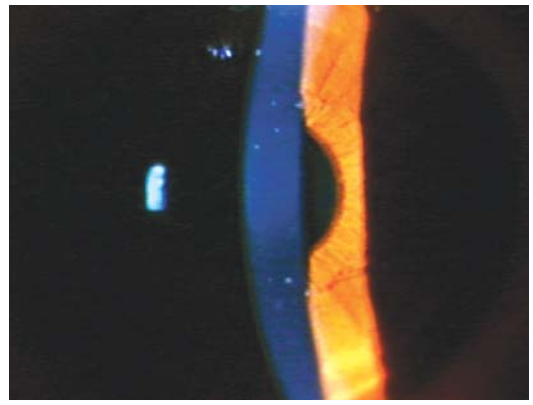


FIGURE 2.13.3: Follicular conjunctivitis—corneal involvement

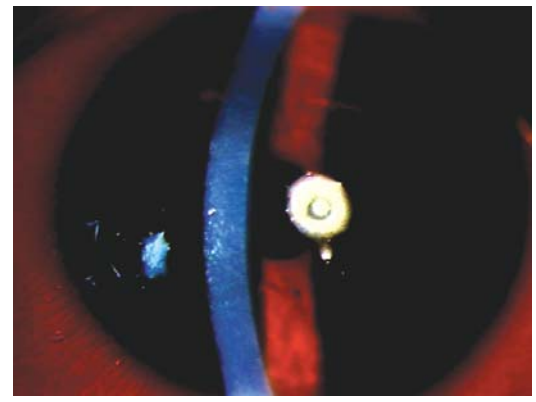


FIGURE 2.13.4: Epidemic keratoconjunctivitis

CHRONIC CONJUNCTIVITIS

Simple Chronic Conjunctivitis

- Redness, more in the evening
- Lower fornix is congested
- Thick frothy discharge with palpebral congestion (**Fig 2.14.1**)
- Associated with meibomianitis
- *Treatment:* antibiotic drop and ointment, treatment of meibomianitis

FIGURE 2.14.1: Chronic conjunctivitis

TRACHOMA

Chronic inflammation of the conjunctiva and the cornea, characterized by the presence of follicles and papillary hypertrophy of the conjunctiva, with pannus formation over upper part of the cornea caused by *Chlamydia trachomatis*

The features are:

- Bulbar congestion
- Velvety papillary hypertrophy **(Fig 2.15.1)**
- *Follicles*—mostly seen in upper tarsal conjunctiva **(Fig 2.15.2)**
- *Pannus*—mainly seen at the upper limbus and upper part of cornea **(Figs 2.15.3 and 2.15.4)**
- *Herbert's pit* at the limbus—**(Fig 2.15.5)** pathognomonic
- *Scarring of upper tarsal conjunctiva* **(Fig 2.15.6)**
- *A blinding condition* mainly due to trichiasis, entropion and subsequent corneal opacification
- included in *Vision—2020 program*
- *Treatment:* sulphacetmide (20% or 30%)—4 times daily, tetracycline eye ointment at night; oral tetracycline or azithromycin
- *Prevention:* 'SAFE' strategy to prevent trachoma blindness:
  - S = Surgery for trichiasis and entropion
  - A = Antibiotics
  - F = Facial cleanliness
  - E = Environmental sanitation

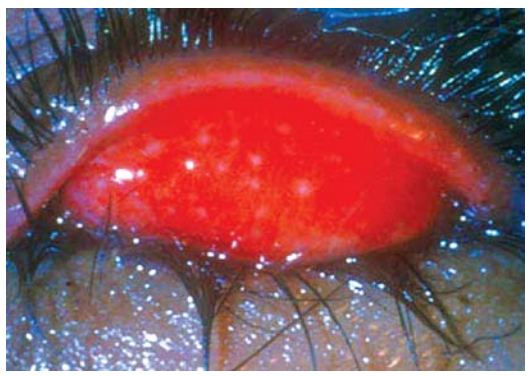


FIGURE 2.15.1: Trachoma—papillary hypertrophy and follicles

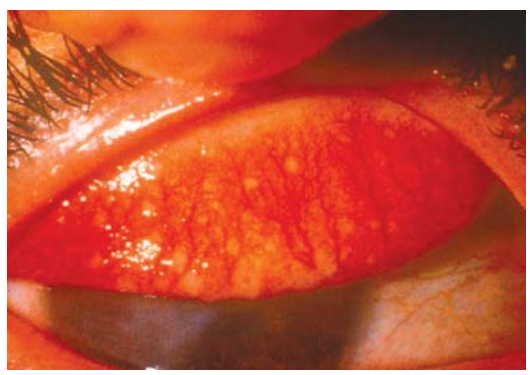


FIGURE 2.15.2: Trachoma follicles

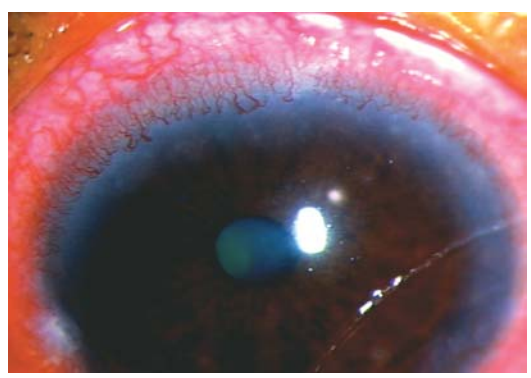


FIGURE 2.15.3: Trachomatous pannus

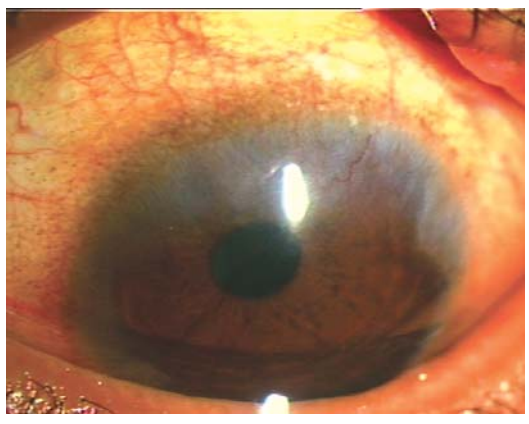


FIGURE 2.15.4: Healed pannus

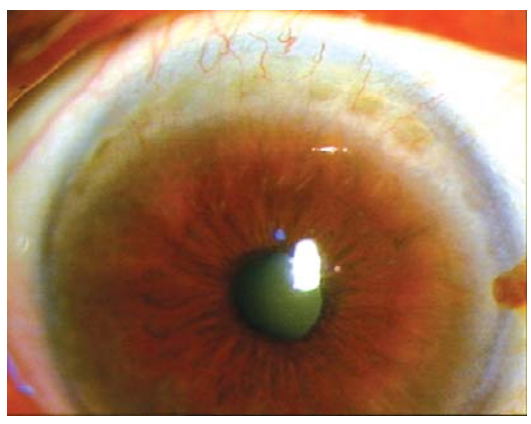


FIGURE 2.15.5: Herbert's pit

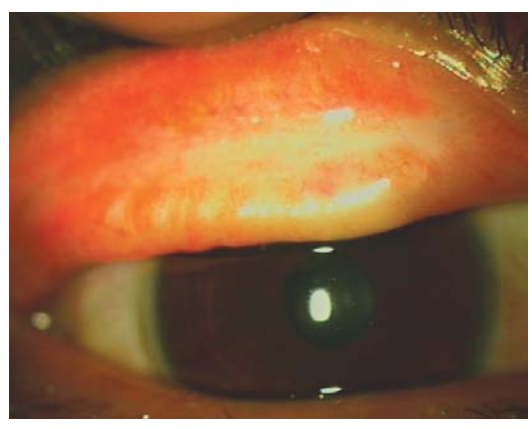


FIGURE 2.15.6: Trachoma scar

ALLERGIC CONJUNCTIVITIS

Phlyctenular Conjunctivitis

- Allergic conjunctivitis caused by endogenous bacterial toxin
- Characterized by bleb or nodule formation near or at the limbus in young children (**Fig 2.16.1**)
- Pinkish-white or gray in color, 1-3 mm in diameter
- Localized bulbar congestion surrounding the nodule (**Fig 2.16.2**)
- Multiple, recurrent phlycten may be seen in tuberculous infection (**Fig 2.16.3**)
- Bleb may rupture to form phlyctenular ulcer which may spread towards cornea leading to fascicular ulcer (**Fig 2.16.4**)
- It may be *three types:*
  - *Phlyctenular conjunctivitis:* when the conjunctiva alone is involved
  - *Phlyctenular keratoconjunctivitis*: when at the limbus, and involves both the conjunctiva and cornea
  - *Phlyctenular keratitis:* when cornea alone is involved (rare)
- *Treatment:* corticosteroid eye drops, investigation and general measures

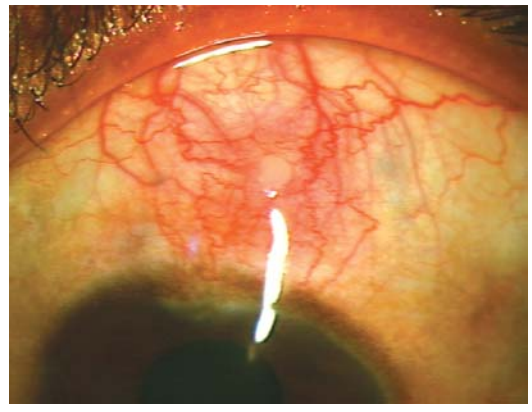


FIGURE 2.16.1: Conjunctival phlycten

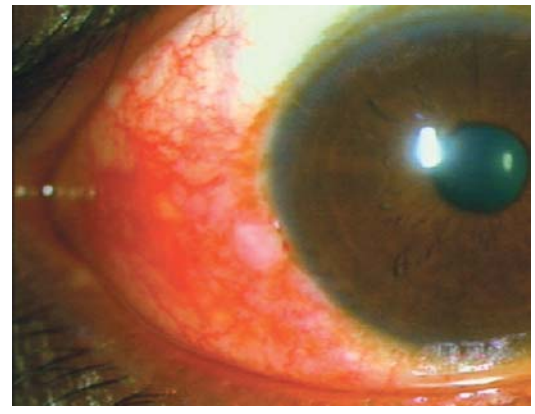


FIGURE 2.16.2: Limbal phlycten

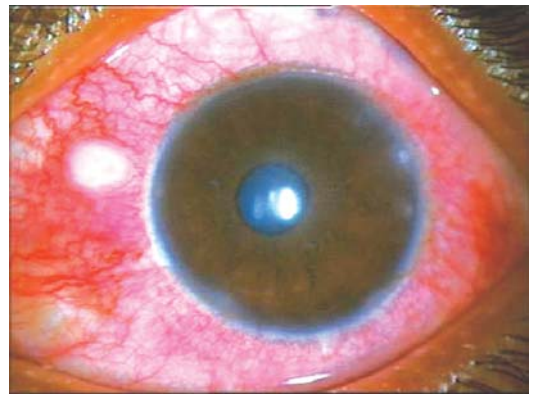


FIGURE 2.16.3: Multiple phlycten

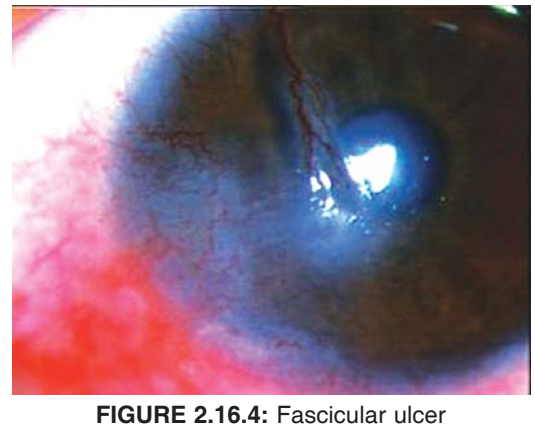


FIGURE 2.16.4: Fascicular ulcer

Vernal Conjunctivitis (keratoconjunctivitis)

- Bilateral, recurrent, seasonal allergic conjunctivitis in children caused by exogenous allergens
- Presents in one of the three forms: Palpebral, bulbar and mixed
- *Palpebral:*
  - *Cobble-stone appearance* **(Fig 2.17.1)** of papillary hypertrophy of the palpebral conjunctiva
  - The bluish-white papillae are separated by connective tissue septa
- *Bulbar:*
  - Multiple, small, nodule-like gelatinous thickening around the limbus, mostly at the upper **(Fig 2.17.2)**
  - Typical conjunctival pigmentation **(Fig 2.17.3)**—may be seen in dark pigmented races
  - Discrete superficial spots, called Horner-Tranta's dots **(Fig 2.17.4)** and micropannus **(Fig 2.17.5)** around the upper limbus
- Epithelial micro-erosions-leading to corneal ulceration *(shield ulcer)* **(Fig 2.17.6)**
- *Pseudo-gerontoxon*—resembles an arcus senilis with appearance of '*cupid's bow*' **(Fig 2.17.7)**
- Patients with vernal catarrh also have a higher incidence of keratoconus
- To be differentiated for atopic conjunctivitis **(Fig 2.17.8)**
- *Treatment:* topical corticosteroids, antihistaminics and mast-cell stabilizer eye drop

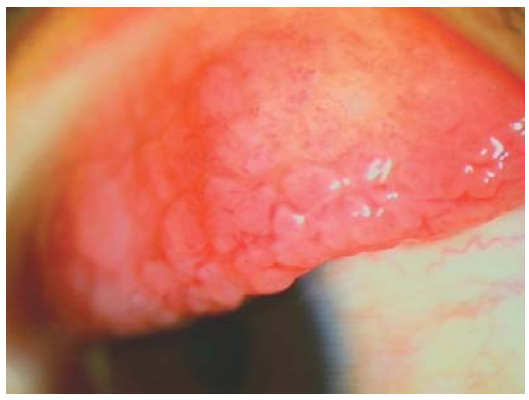


FIGURE 2.17.1: Cobble stone papillae

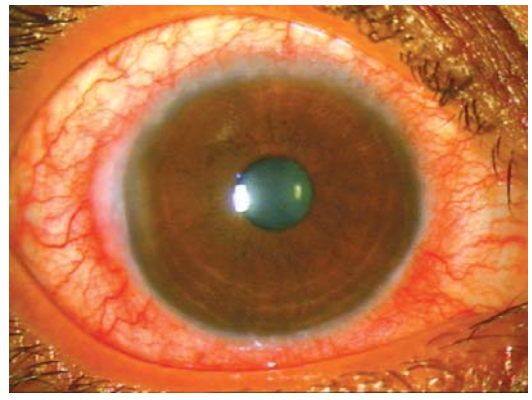


FIGURE 2.17.2: Vernal conjunctivitis—limbal

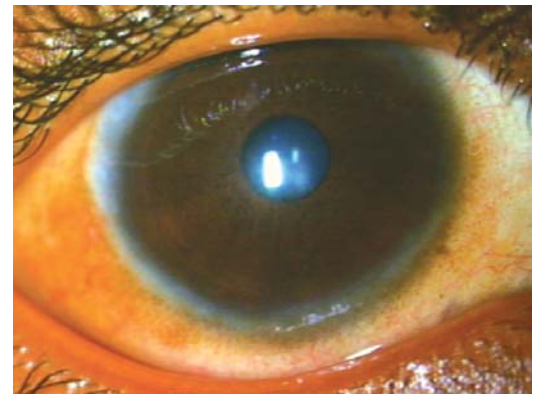


FIGURE 2.17.3: VKC—conjunctival pigmentation

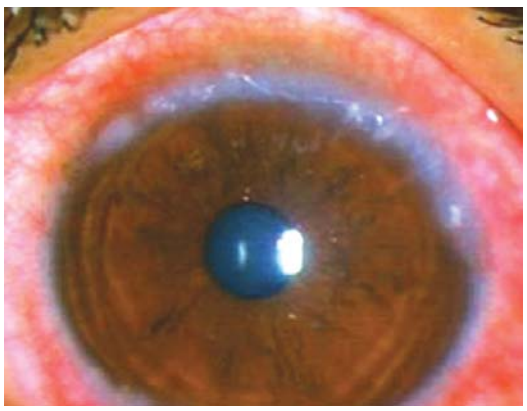


FIGURE 2.17.4: Horner Tranta's dots

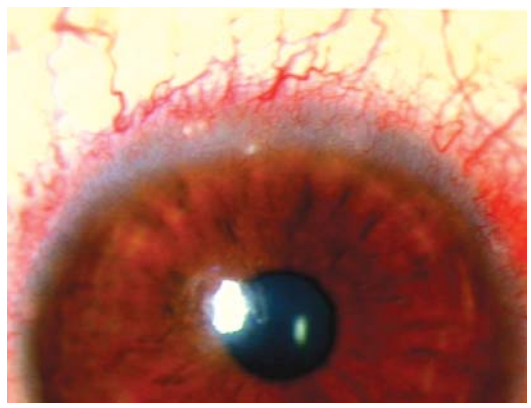


FIGURE 2.17.5: Limbal micropannus

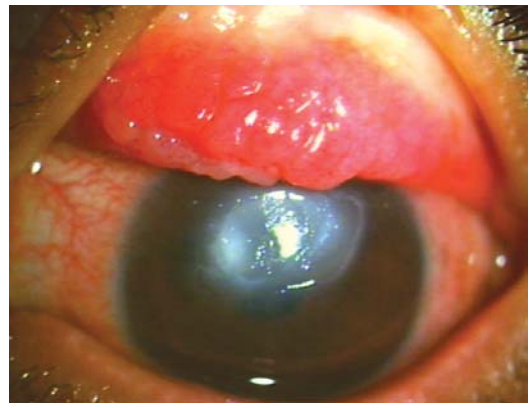


FIGURE 2.17.6: VKC- shield ulcer

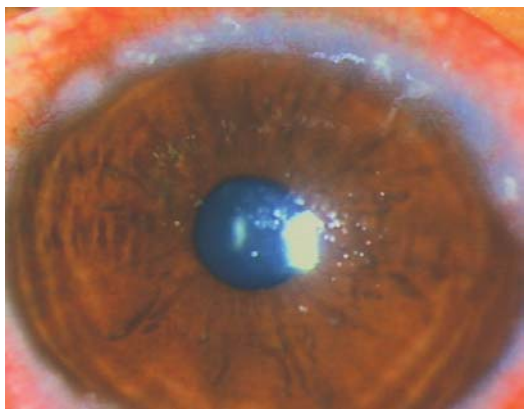


FIGURE 2.17.7: VKC- Cupid's bow

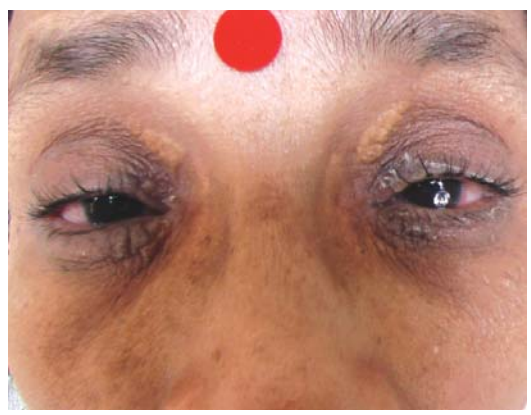


FIGURE 2.17.8: Atopic blepharoconjunctivitis

Giant Papillary Conjunctivitis

- Foreign body associated allergic conjunctivitis with characteristic giant papillae (>1 mm)
- Seen among soft contact lens wearer, artificial eye wearer and post-operative patient with protruding ends of monofilament nylon sutures
- Clinical picture is similar to palpebral type of vernal conjunctivitis with presence of giant papillae (**Fig 2.18.1**)
- *Treatment:* same as vernal conjunctivitis and avoidance of offending agents

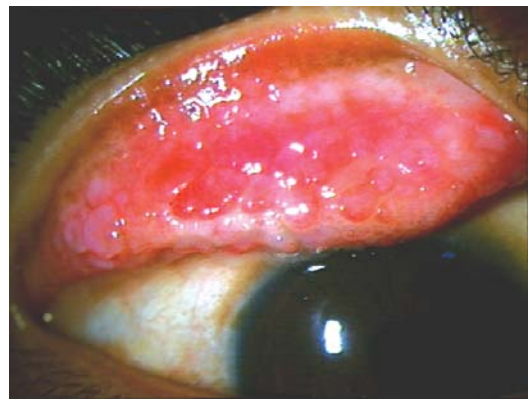


FIGURE 2.18.1: Giant papillary conjunctivitis

CONJUNCTIVAL DEGENERATIONS

Concretion (lithiasis)

- Minute hard yellow / white spots in the upper palpebral conjunctiva (**Fig 2.19.1**)
- They are not calcium deposits, but inspissated mucus and degenerated cells
- Sometimes, they may project and irritate the cornea
- *Treatment:* may be evacuated with a sharp needle

FIGURE 2.19.1: Conjunctival concretions

Pinguecula

- Yellowish, triangular deposit on the conjunctiva near the limbus at the palpebral aperture (**Fig 2.20.1**)
- The apex is towards the cornea
- Affects the nasal side first, then the temporal
- Symptomless and does not require any treatment
- Sometimes, it may be inflamed (**Fig 2.20.2**) and treated with topical corticosteroids

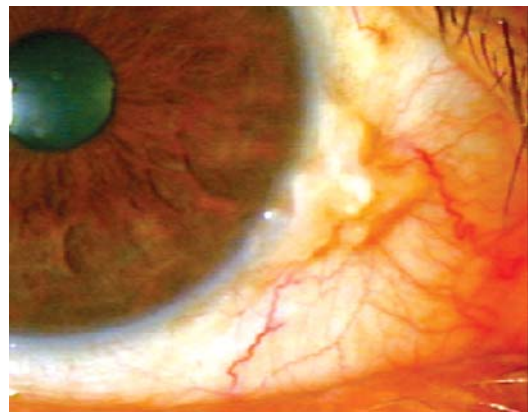


FIGURE 2.20.1: Pinguecula

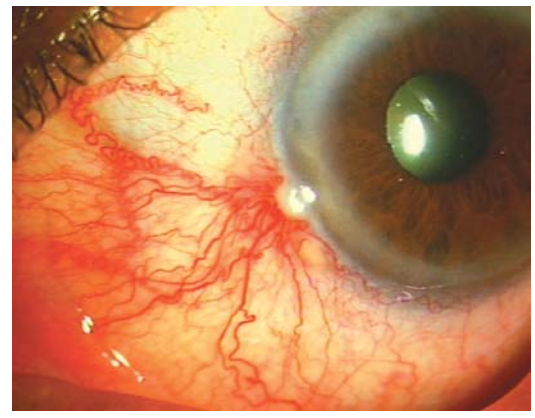


FIGURE 2.20.2: Inflamed pinguecula

Pterygium

- Sub-conjunctival tissue proliferates as a triangular wing-shaped tissue-mass to invade the cornea, involving the Bowman's membrane and the superficial stroma
- Usually bilateral, may be asymmetrical, among elderly individual
- Usually involves the nasal limbus with variable extension onto the cornea (**Fig 2.21.1**)
- Rarely it affects the temporal limbus alone (**Fig 2.21.2**)
- It may be progressive (fleshy) and stationary (atrophic) types
- *Progressive (fleshy)* (**Fig 2.21.3**)
  - thick, fleshy with prominent vascularity
  - increasing in size and encroaching towards the center of cornea
  - opaque infiltrative spot is seen, known as the 'cap'
  - *Stocker's line*—is seen on the corneal epithelium in front of the apex (**Fig 2.21.4**)
- *Atrophic (stationary)* (**Fig 2.21.5**)
  - thin, attenuated with poor vascularity
  - no opaque spot (cap) is seen
- Other types
  - *Primary double pterygium:* Both nasal and temporal limbus are involved (**Fig 2.21.6**)

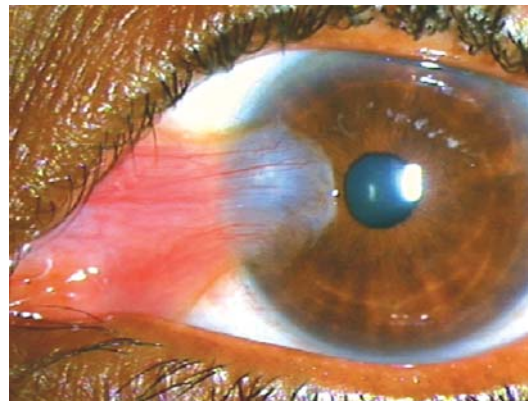


FIGURE 2.21.1: Pterygium

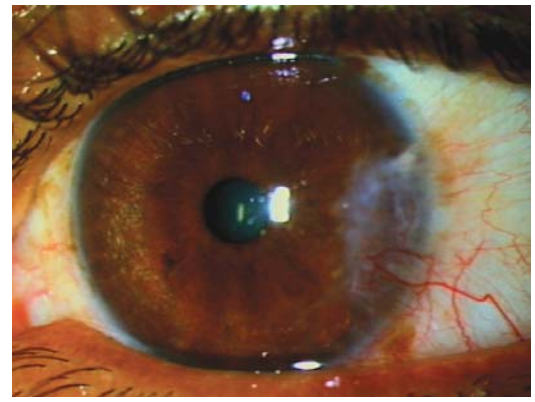


FIGURE 2.21.2: Temporal pterygium

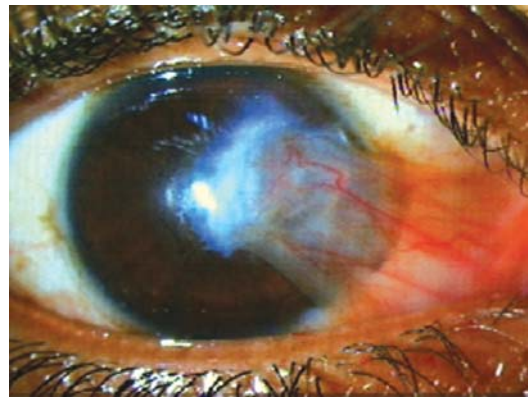


FIGURE 2.21.3: Progressive pterygium—cap

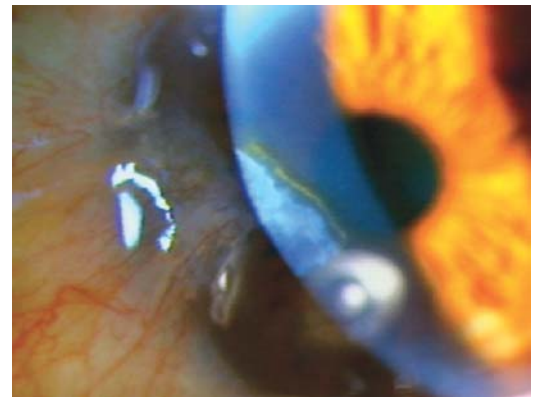


FIGURE 2.21.4: Stocker's line

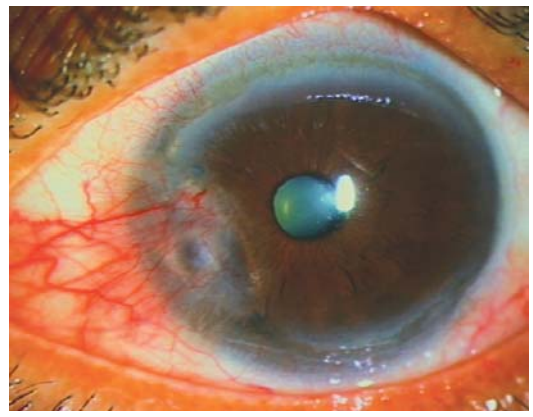


FIGURE 2.21.5: Atrophic pterygium

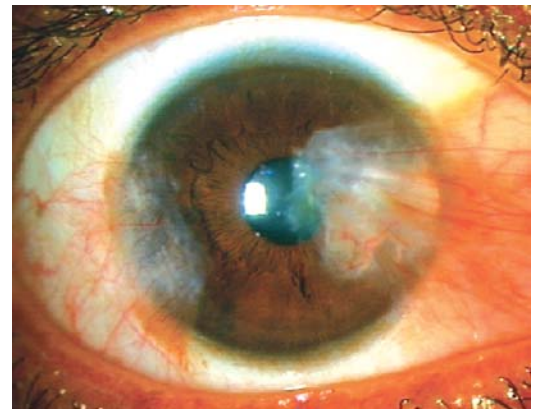


FIGURE 2.21.6: Double headed pterygium

- *Recurrent pterygium:* More scarring and some times more wide (**Fig 2.21.7**)
- *Malignant pterygium:* Recurrent pterygium with symblepharon formation associated with restriction of ocular movement on opposite side (**Figs 2.21.8 and 2.21.9**)

- *Treatment:* excision of pterygium (subconjunctival dissection) with conjunctival limbal autograft (CLAU) is the treatment of choice. Treatment of the bare sclera by MMC is another option
- *Pseudo-pterygium*
  - adhesion of a fold of conjunctiva to the peripheral cornea
  - usually unilateral, stationary, and at any meridian (**Fig 2.21.10**)
  - a probe can be passed easily beneath the neck of the pterygium *(Probe test)*
  - *Treatment:* simple excision

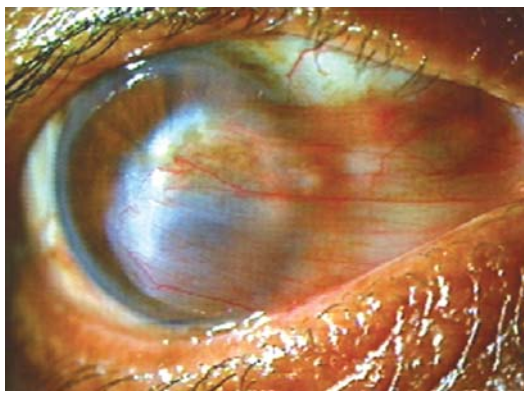


FIGURE 2.21.7: Recurrent pterygium

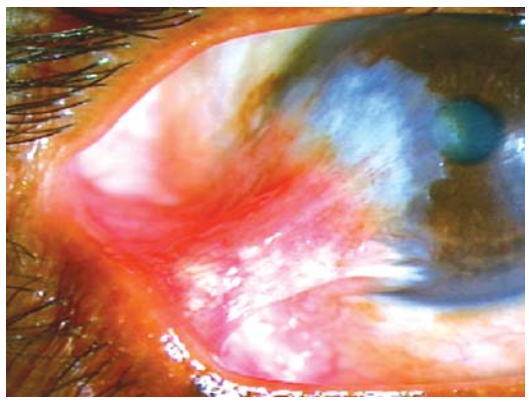


FIGURE 2.21.8: Malignant pterygium—symblepharon

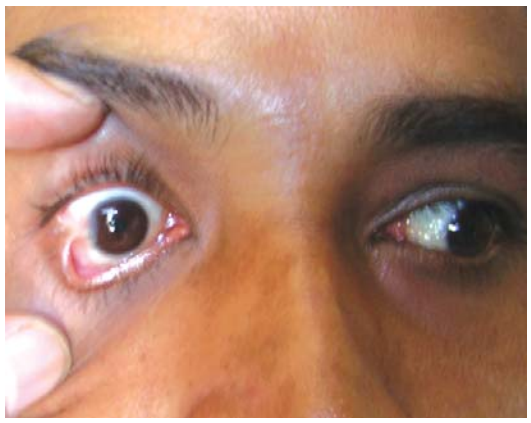


FIGURE 2.21.9: Malignant pterygium

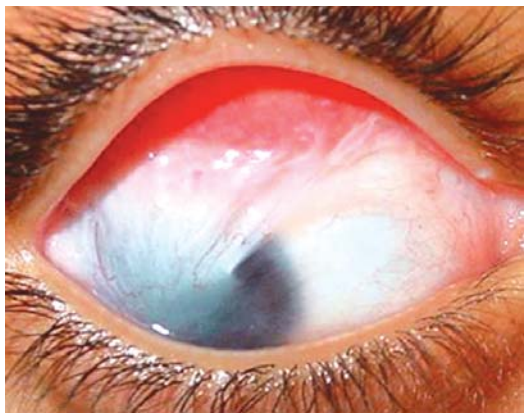


FIGURE 2.21.10: Pseudo-pterygium

BENIGN LESIONS OF THE CONJUNCTIVA

Conjunctival Cysts

- *Lymphangiectasis:* appears as dilatation of lymph vessels **(Figs 2.22.1 and 2.22.2)**
- *Retention cysts:* occur due to the obstruction of the ducts of accessory lacrimal gland of Krause **(Figs 2.22.3 and 2.22.4)**

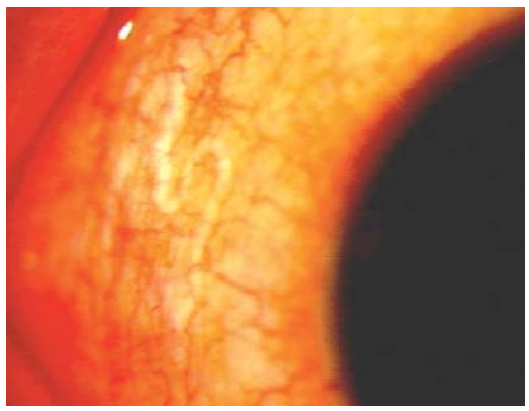


FIGURE 2.22.1: Lymphangiectasis

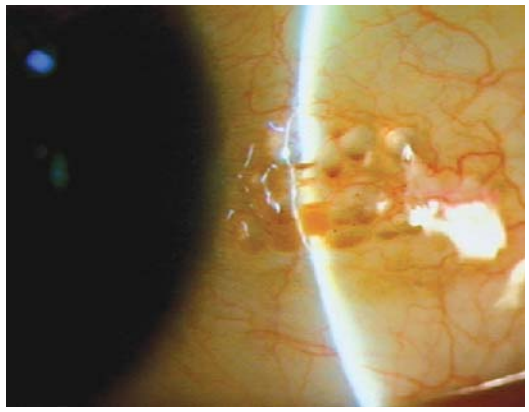


FIGURE 2.22.2: Lymphangiectasis

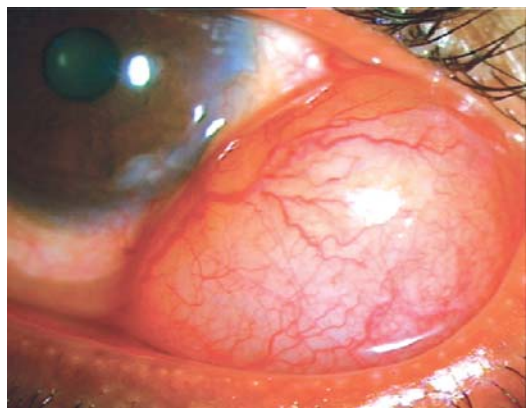


FIGURE 2.22.3: Retention cyst

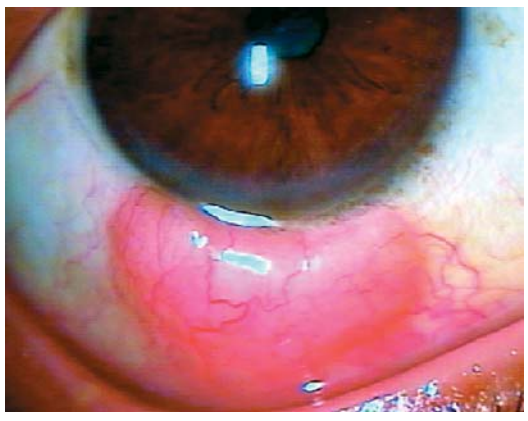


FIGURE 2.22.4: Retention cyst

- *Implantation cysts:* occur due to the implantation of conjunctival epithelial or after an operation or trauma **(Figs 2.22.5 and 2.22.6)**

- *Parasitic cysts:* occur rarely due subconjunctival cysticercus or hydatid cyst **(Figs 2.22.7 and 2.22.8)**
- *Treatment:* simple excision in most cases

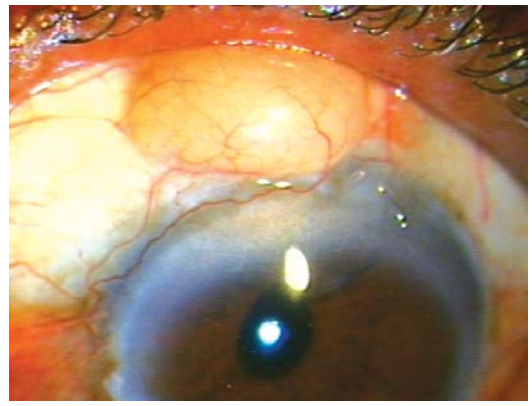


FIGURE 2.22.5: Implantation cyst

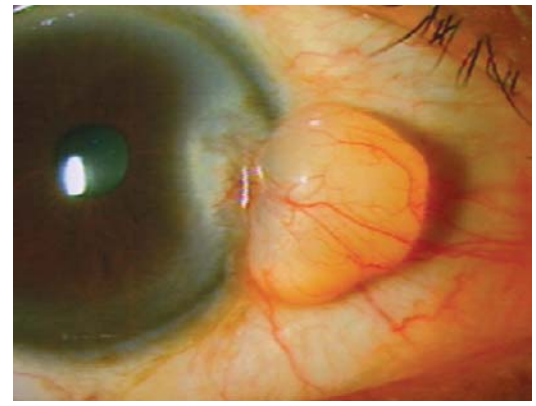


FIGURE 2.22.6: Implantation cyst

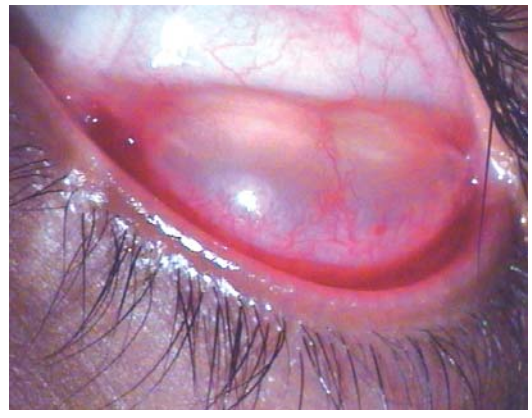


FIGURE 2.22.7: Parasitic cyst—cysticercosis

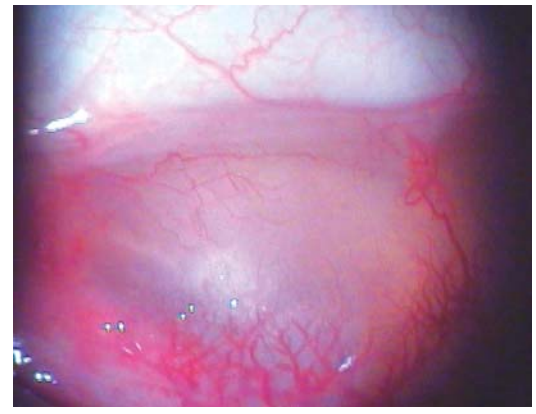


FIGURE 2.22.8: Subconjunctival cysticercus scolex

Epibulbar Limbal Dermoid

- Congenital lesion, either isolated or with systemic association
- Solid, smooth, round white masses most frequently at the limbus, especially in lower-outer quadrant (**Fig 2.23.1**)
- May occur in any quadrant (**Figs 2.23.2 and 2.23.3**) or away from the limbus (**Fig 2.23.4**)

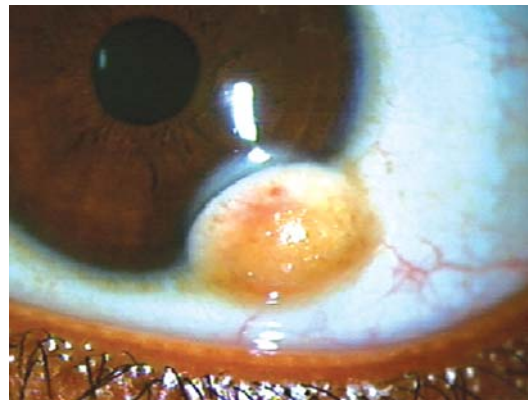


FIGURE 2.23.1: Epibulbar limbal dermoid

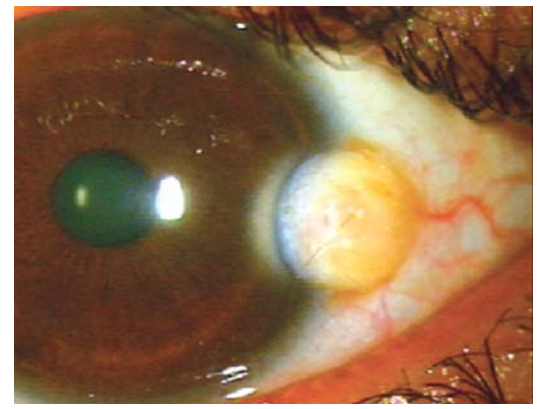


FIGURE 2.23.2: Limbal dermoid—temporal

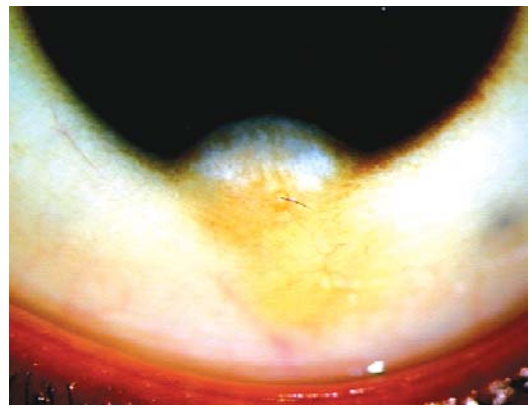


FIGURE 2.23.3: Limbal dermoid—inferior

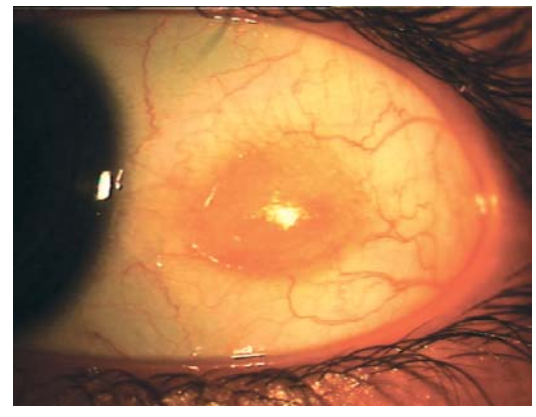


FIGURE 2.23.4: Dermoid away from the limbus

- Large lesion may also involve the cornea (**Fig 2.23.5**)
- In some cases, they are associated with *Goldenhar's syndrome* (Preauricular skin tags, vertebral anomalies and hemifacial hypoplasia) which may be unilateral or bilateral (**Figs 2.23.6 and 2.23.7**)
- Consist of skin with sebaceous glands and hair
- *Treatment:* excision of the mass with or without lamellar keratoplasty

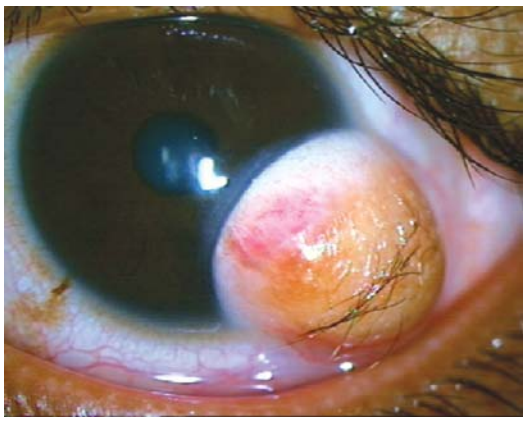


FIGURE 2.23.5: Dermoid—corneal involvement

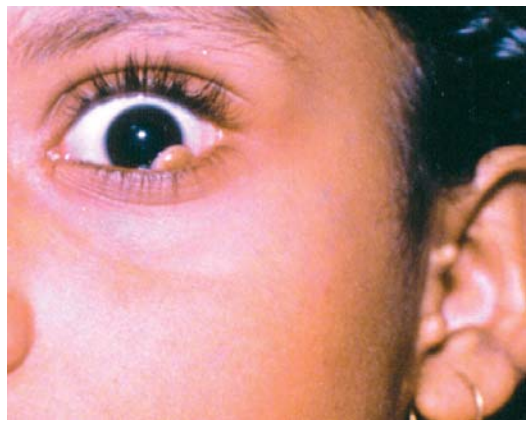


FIGURE 2.23.6: Goldenhar's syndrome—unilateral

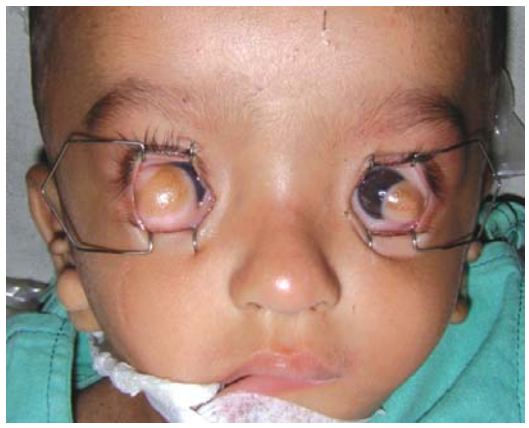


FIGURE 2.23.7: Goldenhar's syndrome—bilateral

Dermolipoma (lipodermoid)

- Large, soft, yellow, movable subconjunctival masses, located at the outer canthus or at the limbus (**Fig 2.24.1**)
- The lesions extend beyond superior fornix and impossible to visualize the posterior limit (**Fig 2.24.2**)
- Extra skin tag with hair may hang from the outer canthus (**Fig 2.24.3**)
- Sometimes, it may be associated with Goldenhar's syndrome (**Fig 2.24.4**)
- *Treatment:* excision of the mass

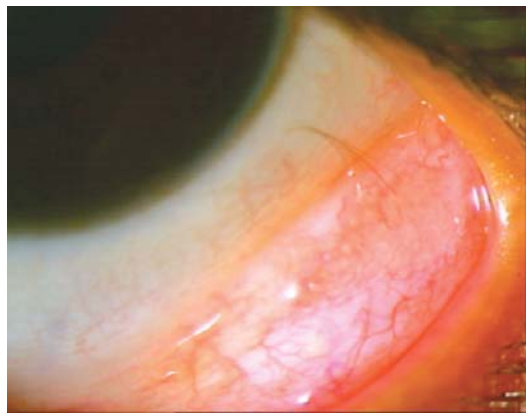


FIGURE 2.24.1: Dermolipoma

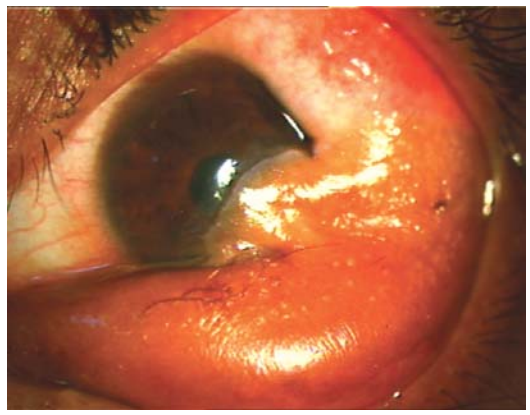


FIGURE 2.24.2: Large dermolipoma

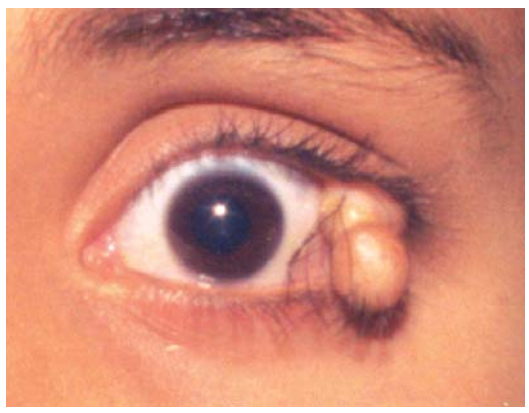


FIGURE 2.24.3: Dermolipoma with skin tag

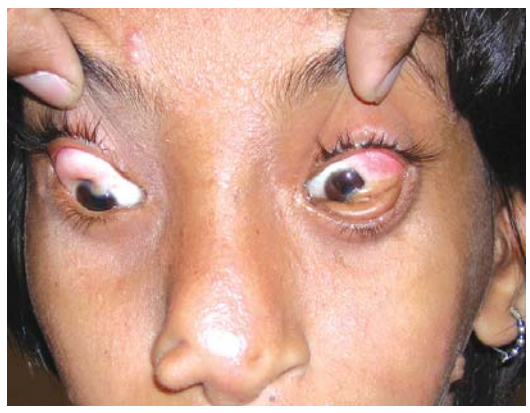


FIGURE 2.24.4: Bilateral dermolipoma—Goldenhar's syndrome

Choriostomas

- Similar to dermoid but has sharper edge and located just outside the limbus (**Fig 2.25.1**)
- More fleshy and more vascularized (**Fig 2.25.2**)
- It contains mature bone
- Very rarely other ectopic tissues may be seen, e.g. cartilage, lacrimal gland, fat, smooth muscles, etc.
- *Treatment:* excision of the mass with or without lamellar keratoplasty

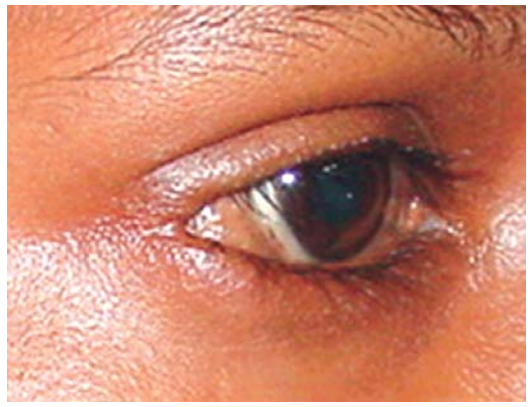


FIGURE 2.25.1: Choriostoma

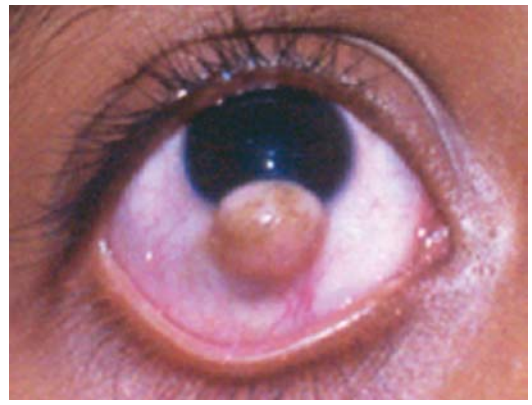


FIGURE 2.25.2: Choriostoma

MALIGNANT LESION

- Conjunctival intra-epithelial neoplasia (carcinoma-in-situ)
- Rare unilateral premalignant condition
- Seen in elderly individuals with fair complexion
- Previously called- Bowen's disease, intraepithelial epithelioma or dyskeratosis
- Slightly elevated, fleshy mass with tuft of blood vessels at the limbus (**Fig 2.26.1**)
- May involve the adjacent cornea (**Fig 2.26.2**)
- It may be *fleshy gelatinous* (**Fig 2.26.3**), *leukoplakic type* (**Fig 2.26.4**) *or papillary* type **(Fig 2.26.5)**
- *Treatment:* excision of the mass with triple cryo-thawing of the dissected area

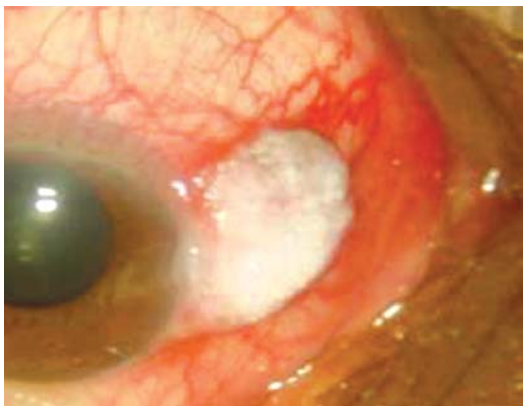


FIGURE 2.26.1: Conjunctival intraepithelial neoplasia

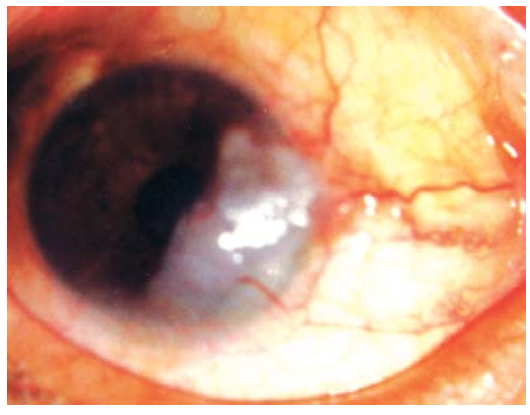


FIGURE 2.26.2: CIN—corneal involvement

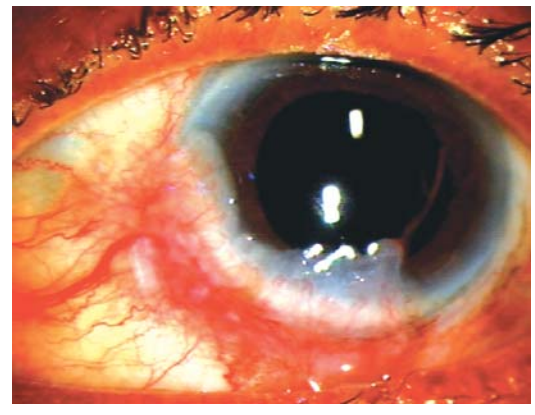


FIGURE 2.26.3: CIN—gelatineous type

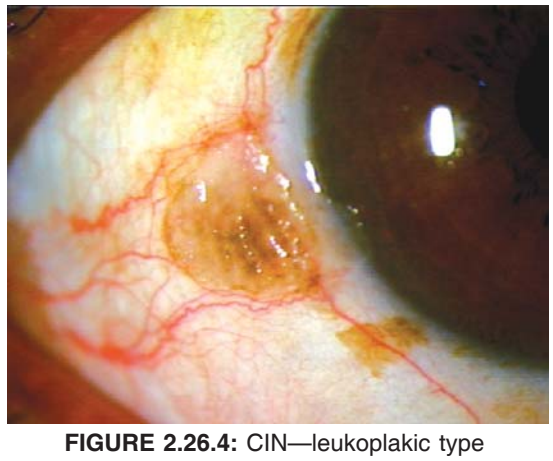


FIGURE 2.26.4: CIN—leukoplakic type

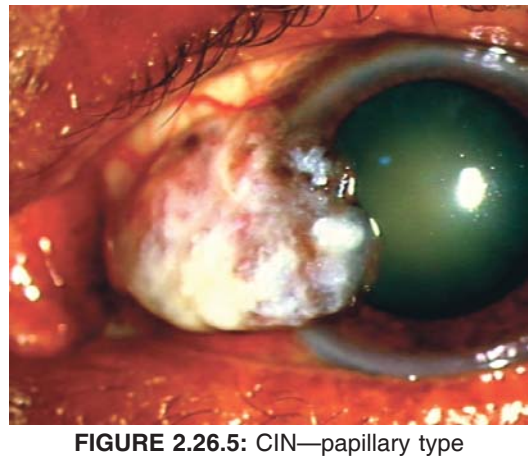


FIGURE 2.26.5: CIN—papillary type

Invasive Squamous-cell Carcinoma

- Slow growing, locally invasive tumor at the limbus (**Fig 2.27.1**)
- It arises from papilloma or carcinoma-in-situ (**Fig 2.27.2**)
- Reddish-gray fleshy mass with broad base, and characterized by deep invasion into the stroma with fixation to the underlying structure **(Fig 2.27.3)**
- Frequently associated with large feeder vessels **(Fig 2.27.4)**
- It may also involve the adjacent cornea **(Figs 2.27.5 and 2.27.6)**
- Rarely it may arise from fornix **(Fig. 2.27.7)** or from caruncle **(Fig. 2.27.8)**
- *Treatment:* excision of the mass with triple cryo-thawing of the dissected area with peroperative MMC

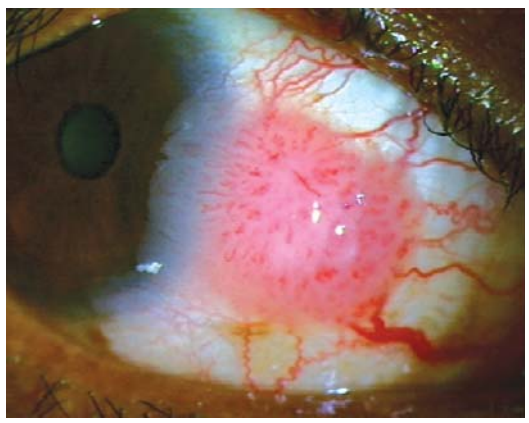


FIGURE 2.27.1: Invasive squamous-cell carcinoma

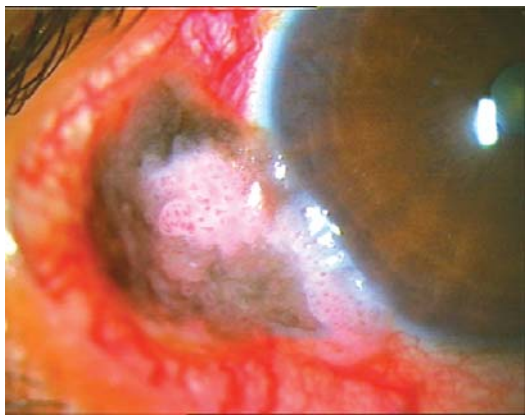


FIGURE 2.27.2: CIN to squamous-cell carcinoma

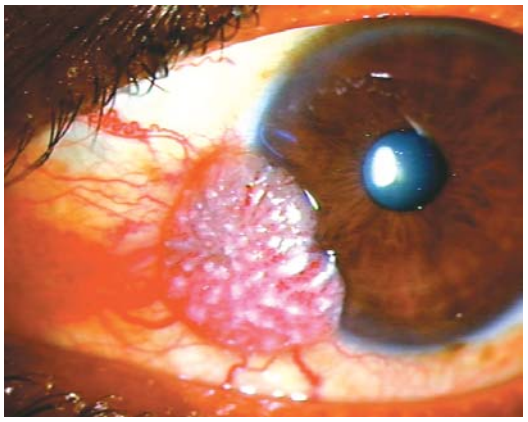


FIGURE 2.27.3: Invasive squamous-cell carcinoma

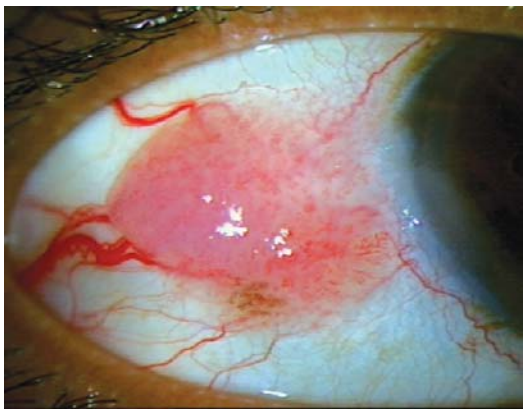


FIGURE 2.27.4: Feeder vessels in CIN

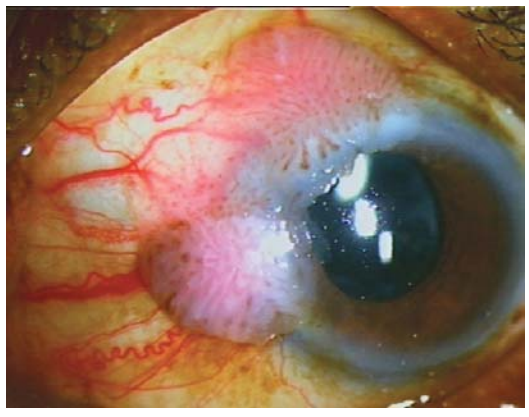


FIGURE 2.27.5: Corneal involvement

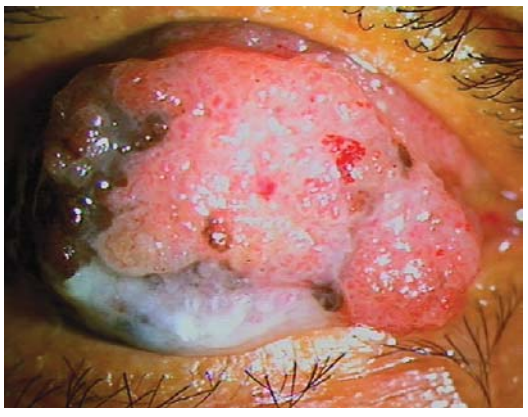


FIGURE 2.27.6: Severe corneal involvement

FIGURE 2.27.7: Squamous-cell carcinoma—from fornix

FIGURE 2.27.8: Squamous-cell carcinoma—from caruncle

PIGMENTED LESIONS

Flat Superficial Pigmentation

- These are common, congenital, small focal lesions
- The *causes are*: conjunctival freckles, melanosis around Axenfeld loop (an intra-scleral nerve loop 4 mm away from limbus) and melanosis around anterior ciliary artery (**Figs 2.28.1 and 2.28.2**)
- No treatment is necessary

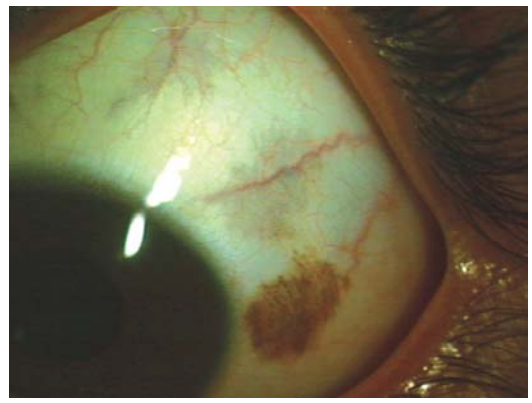


FIGURE 2.28.1: Flat superficial pigmentation

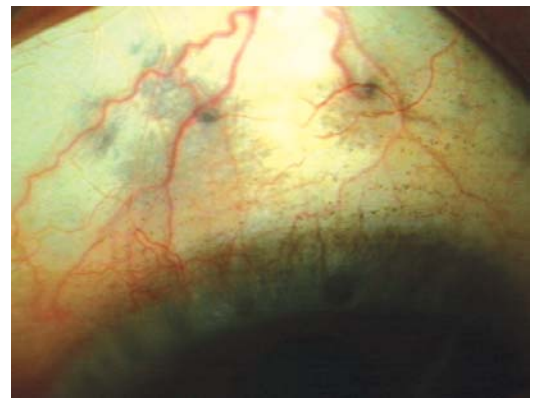


FIGURE 2.28.2: Flat superficial pigmentation

Benign Epithelial Melanosis

- Bilateral, yellowish-brown or brownish-black patches most prominent at the limbus (**Fig 2.29.1**)
- It is also present at the interpalpebral areas and gradually fades towards fornix
- Lesions can be easily moved on the globe
- No treatment is necessary

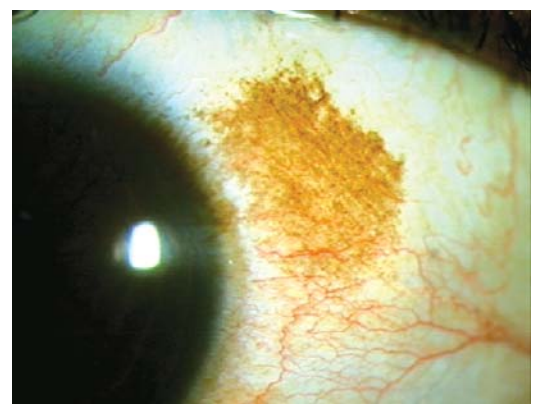


FIGURE 2.29.1: Benign epithelial melanosis

Benign Subepithelial Melanocytosis

- Rare, unilateral congenital condition with a slate blue-gray discoloration **(Fig 2.30.1)**
- It affects the episcleral and scleral tissue and can not be moved
- It may affect the skin and mucous membrane in the distribution of fifth nerve
- There may be isolated *melanocytosis oculi*, or *Oculodermal melanocytosis* (naevus of Ota) which involves both the skin and globe (**Fig 2.30.2**)
- No treatment is necessary

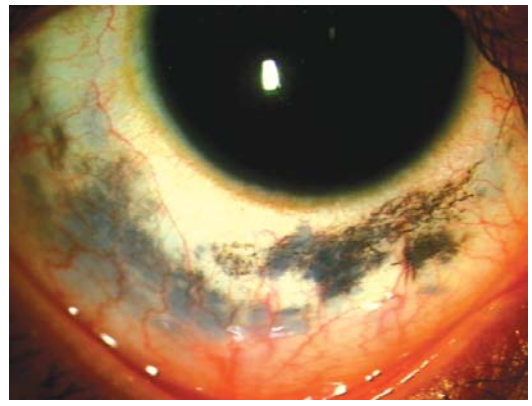


FIGURE 2.30.1: Subepithelial melanocytosis

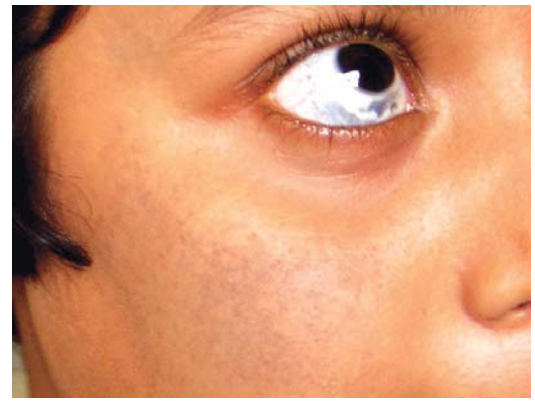


FIGURE 2.30.2: Naevus of Ota

Simple Naevus

- More common benign tumor
- Single, sharply demarcated, flat or slightly elevated lesion (**Fig 2.31.1**)
- Has predilection for the limbus (**Fig 2.31.2**) plica (**Fig 2.31.3**), caruncle (**Fig 2.31.4**) or lid margin

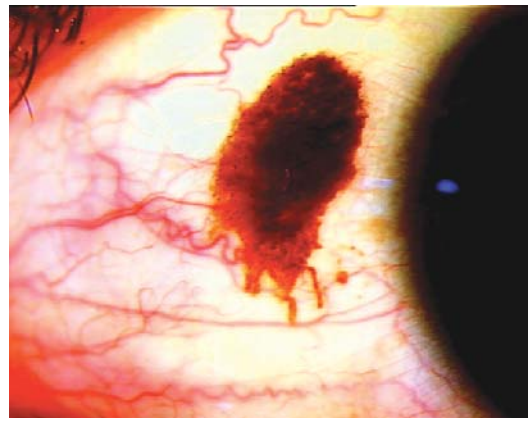


FIGURE 2.31.1: Simple naevus

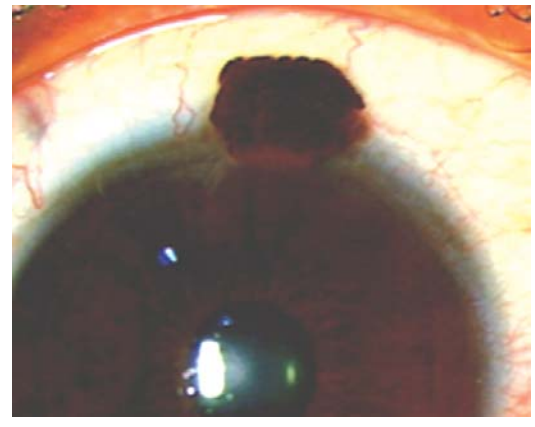


FIGURE 2.31.2: Simple naevus—limbal

- Most naevi have a tan or brown color (**Fig 2.31.5**) , and 25% are non-pigmented (**Fig 2.31.6**)
- Tends to enlarge or darken during puberty or pregnancy
- *Treatment:* usually not necessary, but some times for cosmetic reason

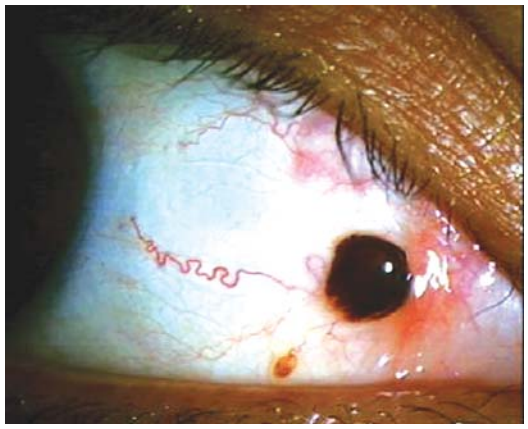


FIGURE 2.31.3: Simple naevus—at plica

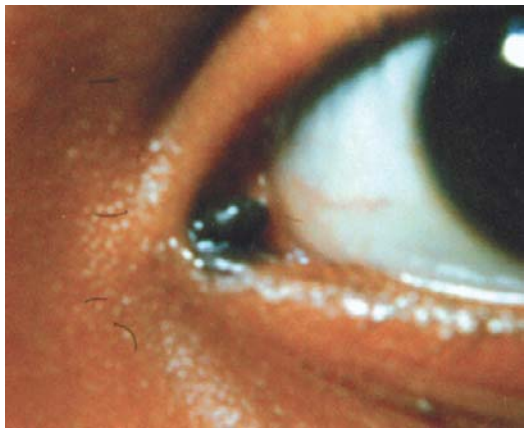


FIGURE 2.31.4: Simple naevus—at caruncle

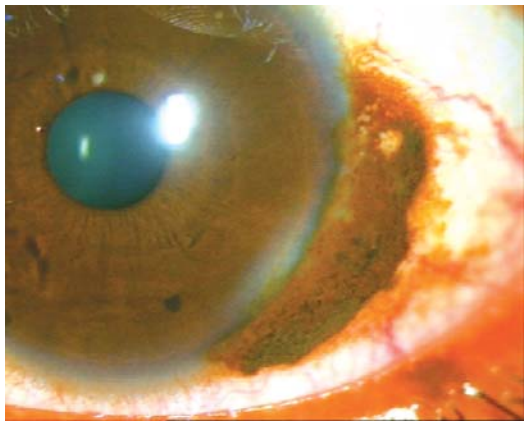


FIGURE 2.31.5: Simple naevus—limbal

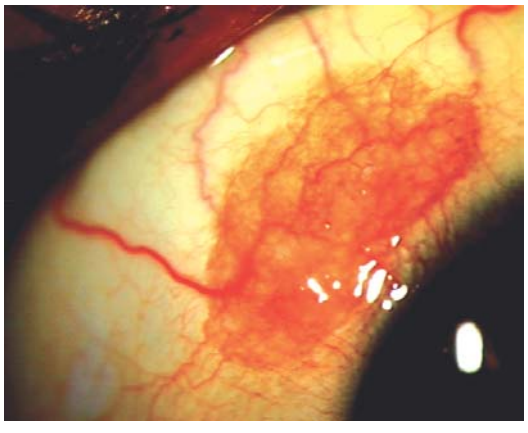


FIGURE 2.31.6: Simple naevus—nonpigmented

Melanocytoma

- Very rare, congenital benign tumor
- Black, slowly growing mass which does not move freely over the globe (**Fig 2.32.1)**
- Tends to enlarge during puberty
- *Treatment:* not necessary

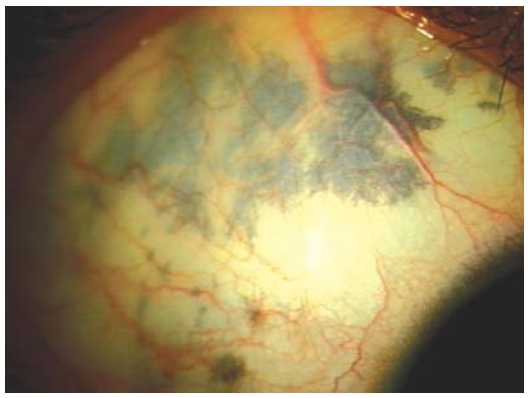


FIGURE 2.32.1: Conjunctival melanocytoma

Pre-cancerous Melanosis

- Small pigmented tumor which spread as a diffuse patch of pigmented lesion (**Fig 2.33.1**)
- Mostly occur in elderly patients
- 20% cases it proceeds to frank malignancy (**Figs 2.33.2 and 2.33.3**)
- *Treatment:* excision in suspected cases

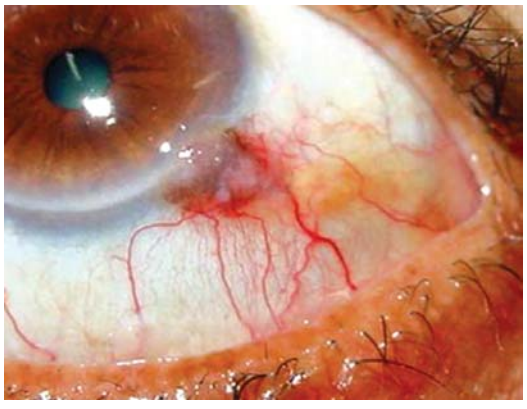


FIGURE 2.33.1: Precancerous melanosis

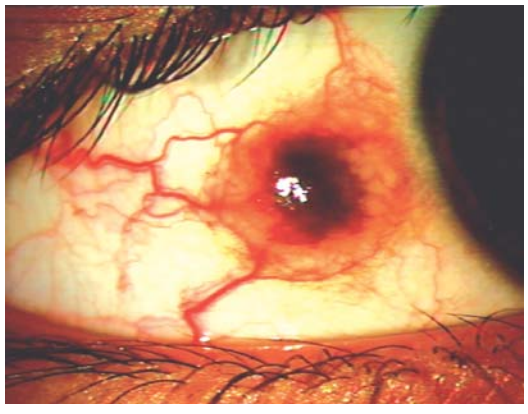


FIGURE 2.33.2: Precancerous melanosis—malignant change

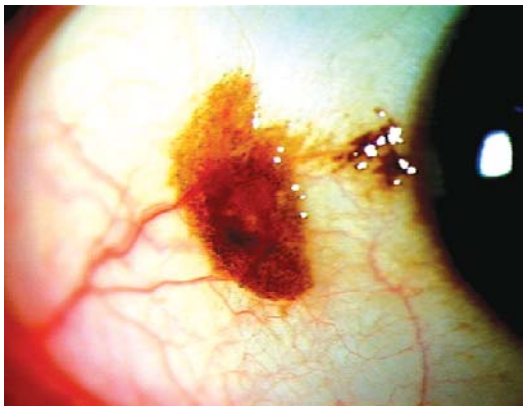


FIGURE 2.33.3: Precancerous melanosis—malignant change

Malignant Melanoma

- May be pigmented or non-pigmented lesion (**Fig 2.34.1**) which affects elderly people
- Elevated lesion can occur in any part of conjunctiva but has a predilection for limbus (**Fig 2.34.2**)
- Larger lesion may involve cornea, adjacent part of eyelid and orbit (**Figs 2.34.3 and 2.34.4**)
- Metastasis is common
- *Treatment:* excision of the mass

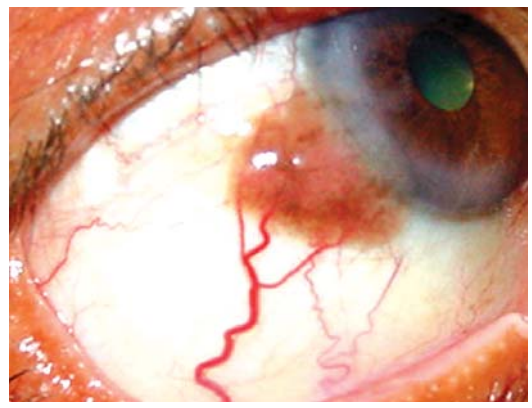


FIGURE 2.34.1: Amelanotic melanoma

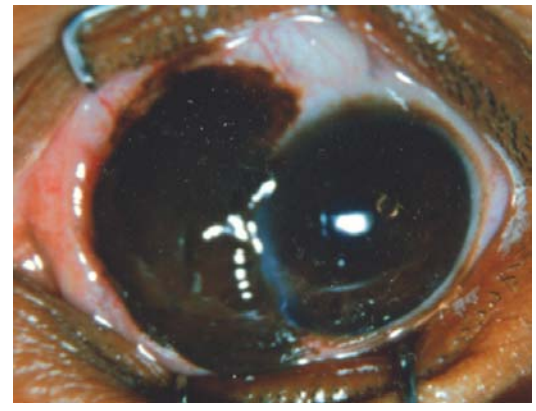


FIGURE 2.34.2: Conjunctival melanoma

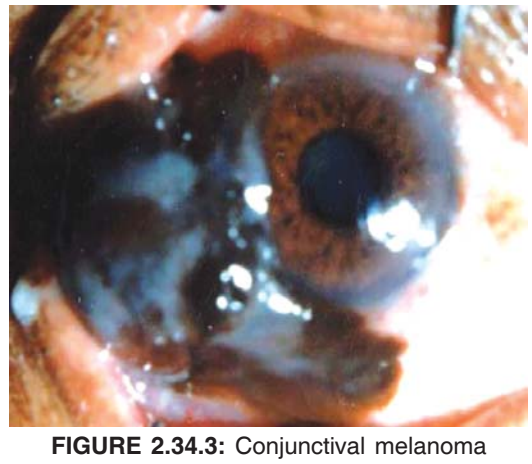


FIGURE 2.34.3: Conjunctival melanoma

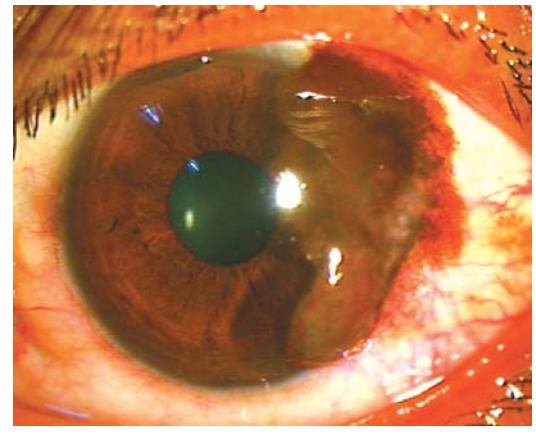


FIGURE 2.34.4: Conjunctival melanoma—corneal extension

OTHER CONJUNCTIVAL CONDITIONS

Subconjunctival Hemorrhage

- Rupture of a conjunctival blood vessel causes a bright red, sharply delineated area surrounded by the normal appearing conjunctiva
- Usually unilateral (**Fig 2.35.1**), but it may be bilateral (**Fig 2.35.2**) when precipitated by some straining factor like, whooping cough
- May be associated with conjunctivitis (**Fig 2.35.3**) or post surgical trauma (**Fig 2.35.4**)

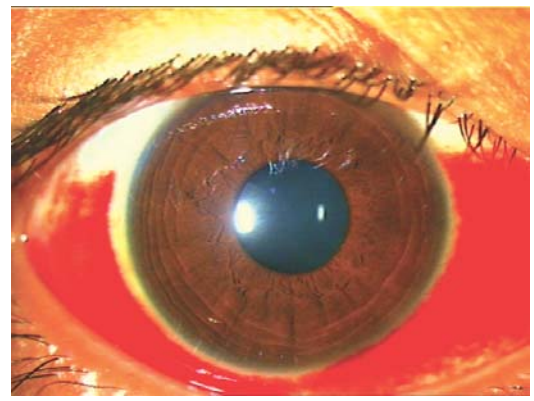


FIGURE 2.35.1: Subconjunctival hemorrhage—unilateral

FIGURE 2.35.2: Bilateral subconjunctival hemorrhage—whooping cough

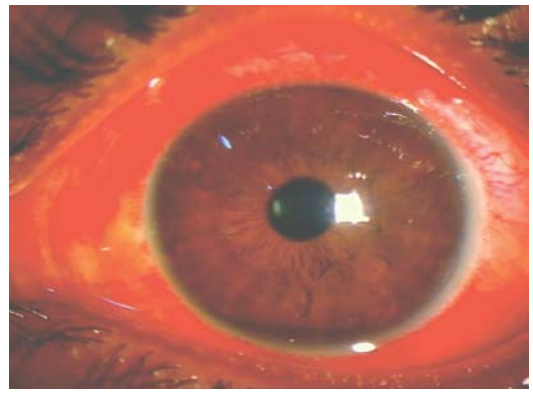


FIGURE 2.35.3: SC hemorrhage—conjunctivitis

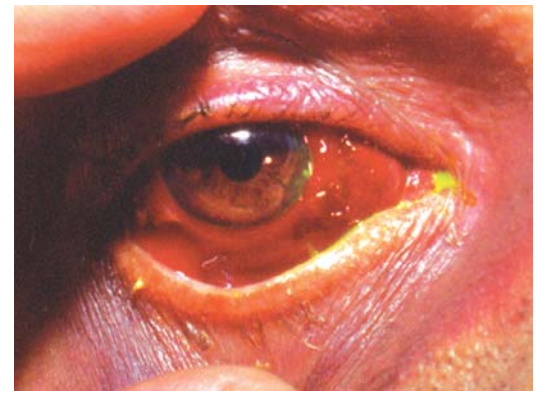


FIGURE 2.35.4: SC hemorrhage—postsurgical

- Traumatic hemorrhage may be associated with hyphema, chemosis or ecchymosis of lid (**Fig 2.35.5**)
- When caused by head injury—the posterior limit is not visible (**Fig 2.35.6**)
- *Treatment:* only assurance and astringents

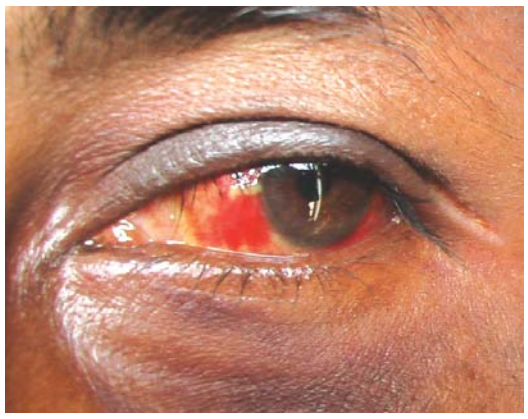


FIGURE 2.35.5: SC hemorrhage—trauma

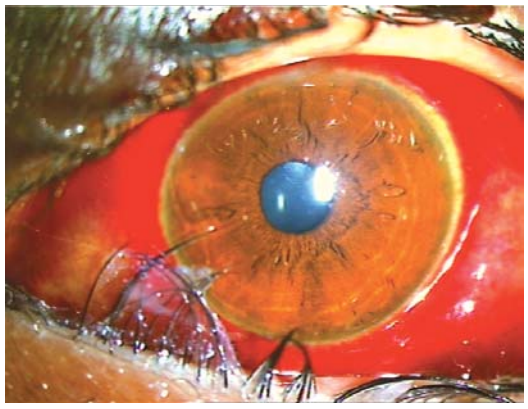


FIGURE 2.35.6: SC hemorrhage—head injury

Conjunctival Xerosis

- Due to vitamin-A deficiency with associated PEM (**Fig 2.36.1)**
- Burns, pemphigoid, diphtheria, β-blocker or following prolonged exposure, etc
- Conjunctiva is pale, lustureless and with folds and variable pigmentation (**Figs 2.36.2 and 2.36.3**)
- *Treatment:* active prompt vitamin-A supplementation

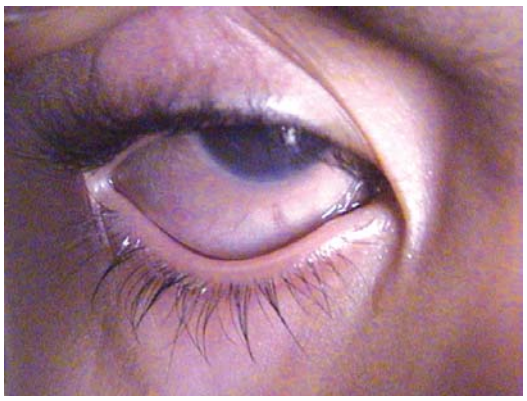


FIGURE 2.36.1: Xerosis—vitamin A deficiency

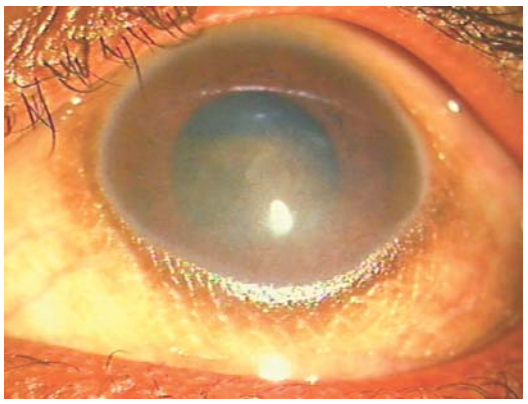


FIGURE 2.36.2: Conjunctival xerosis

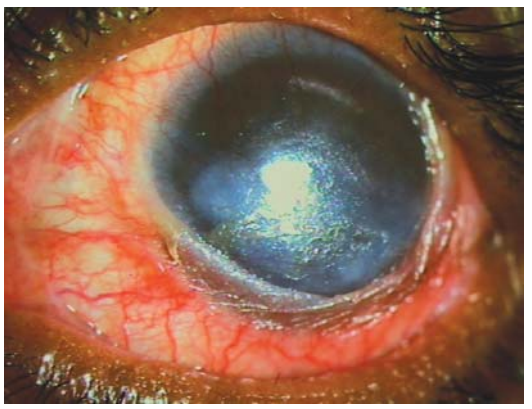


FIGURE 2.36.3: Conjunctival xerosis

Bitot's Spot

- Bilateral lesion in young children, associated with vitamin A deficiency
- Appears as triangular patch, with base towards limbus and usually on the temporal side (**Fig 2.37.1)**
- It may be cheesy or foamy in appearance (**Figs 2.37.2 and 2.37.3)**
- Associated with conjunctival xerosis and conjunctival folds
- Isolated Bitot's spot with pigmentary changes represents an area of old squamous metaplasia **(Fig 2.37.4)**
- *Treatment:* active prompt vitamin-A supplementation

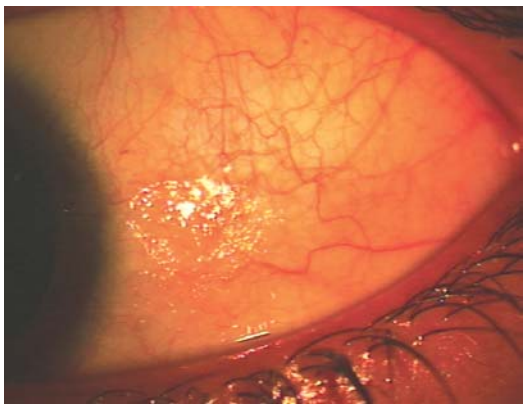


FIGURE 2.37.1: Bitot's spot

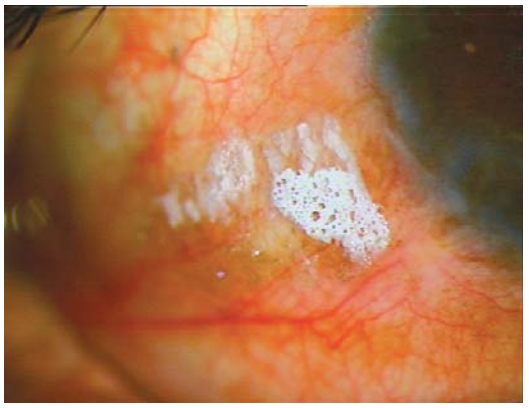


FIGURE 2.37.2: Bitot's spot—foamy

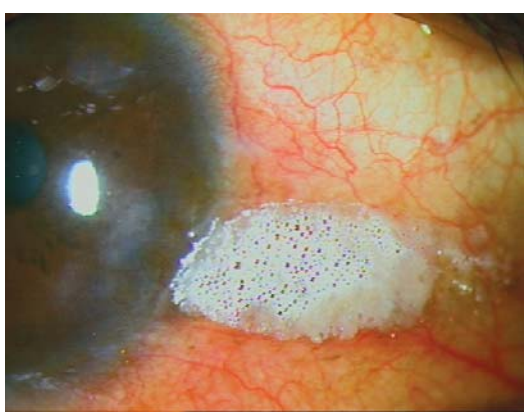


FIGURE 2.37.3: Bitot's spot—foamy

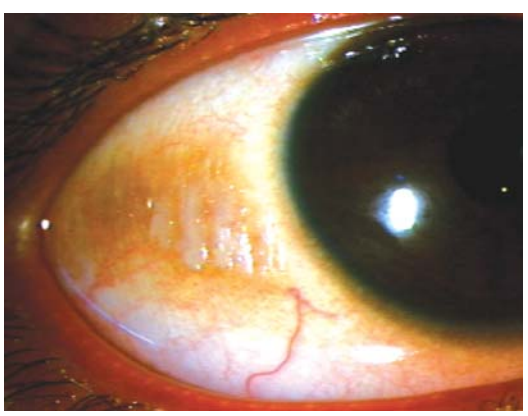


FIGURE 2.37.4: Old Bitot's spot—cheesy

Keratoconjunctivitis Sicca

- Very common bilateral condition, leading to dry eye and ocular surface disorders
- Reduced or absent tear meniscus height (**Fig 2.38.1**)
- Corneal filaments and mucus plaques
- Positive fluorescein **(Fig 2.38.2)** or Rose Bengal staining of interpalpebral conjunctival area in a triangular fashion **(Fig 2.38.3)**
- Corneal staining is also present associated with other corneal changes (***See Chapter 3***)
- *Treatment:* tear substitutes, topical cyclosporine (0.05%) or 'soft steroids', punctal occlusion, etc.

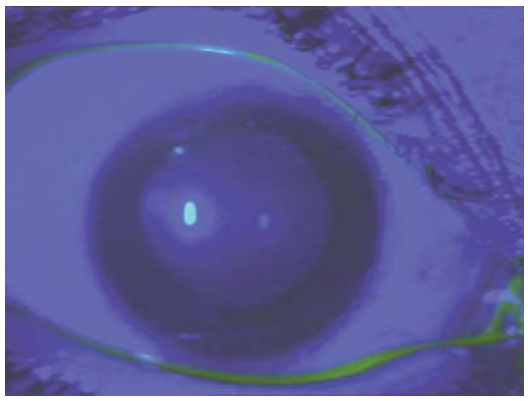


FIGURE 2.38.1: Low tear meniscus height

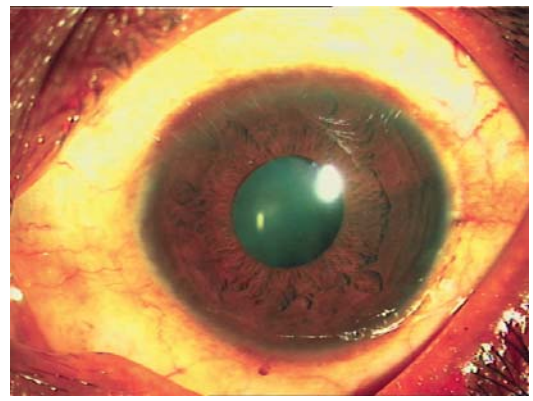


FIGURE 2.38.2: KCS—fluorescein stain

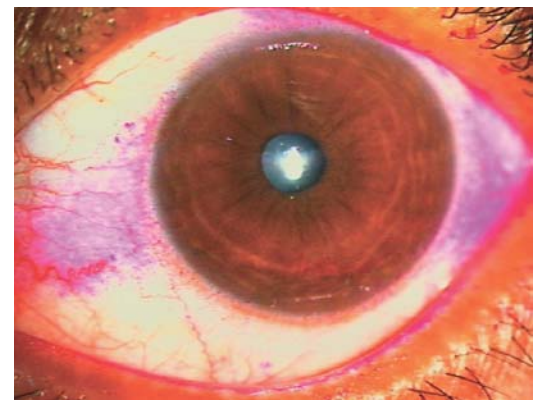


FIGURE 2.38.3: KCS—Rose Bengal stain

Mucus Fishing Syndrome

- Rare unilateral or bilateral self trauma to the conjunctiva
- Excessive mucus and isolated conjunctival areas staining with Rose Bengal (**Fig 2.39.1**)
- *Treatment:* reassurance

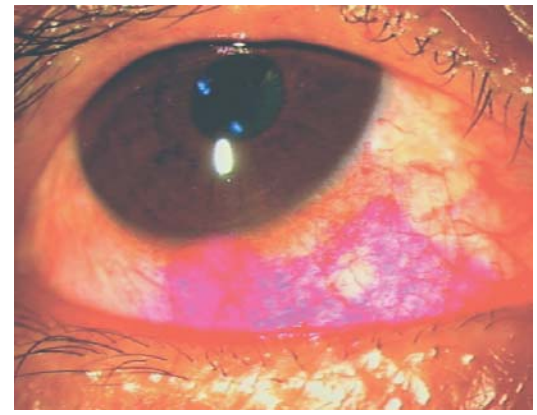


FIGURE 2.39.1: Mucus fishing syndrome

Lid Imbrication Syndrome

- Rare condition caused by over-riding of the lower lid by the upper lid (**Fig 2.40.1**)
- Staining of upper tarsus with Rose Bengal

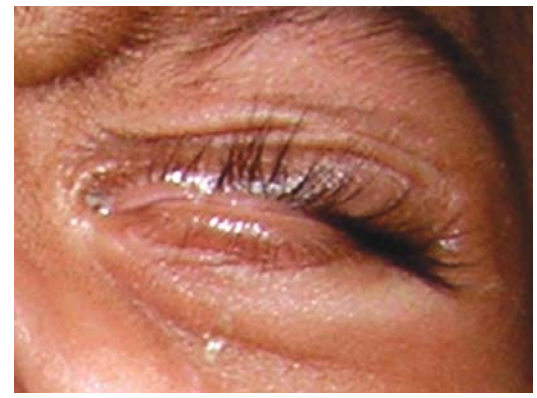


FIGURE 2.40.1: Lid imbrication syndrome

Ocular Cicatricial Pemphigoid

- Conjunctival involvement is seen in majority
- Associated oral mucosal lesions
- Conjunctival inflammation with fine subepithelial fibrosis
- Conjunctival shrinkage with shortening of inferior fornix (**Fig 2.41.1**)
- Loss of plica semilunaris outline
- Medial symblepharon followed by total symblepharon formation (**Figs 2.41.2 and 2.41.3**)
- Keratinization, vascularization and persistent epithelial defect of the cornea (**Figs 2.41.4 and 2.41.5**)
- Trichiasis and metaplastic eyelashes and total obliteration of fornices (**Fig 2.41.6**)
- *Treatment:* tears substitutes, removal of metaplastic lashes, etc.

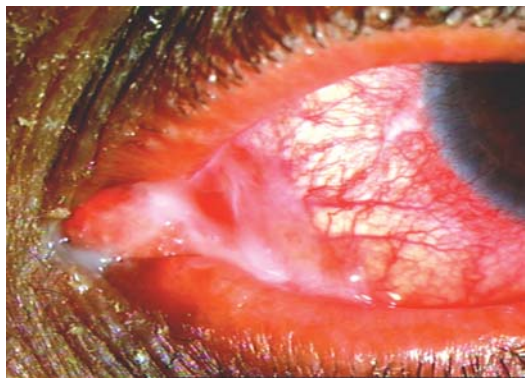


FIGURE 2.41.1: Ocular cicatricial pemphigoid

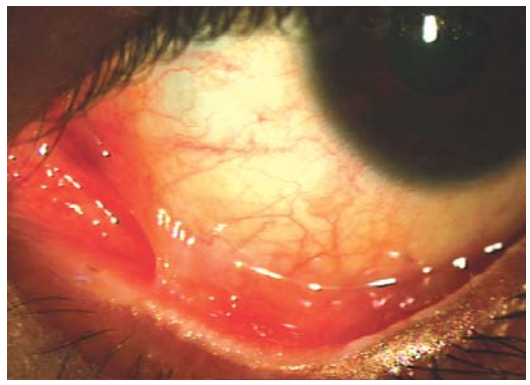


FIGURE 2.41.2: OCP—Medial symblepharon

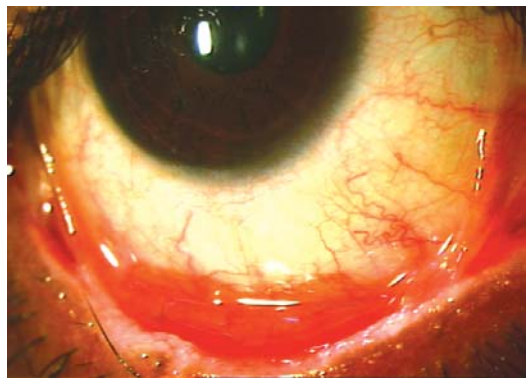


FIGURE 2.41.3: OCP—increasing symblepharon

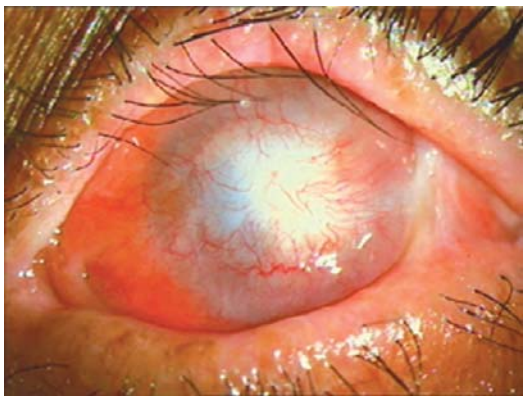


FIGURE 2.41.4: OCP—vascularization

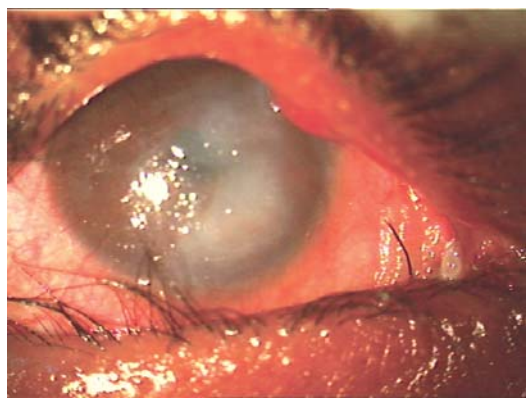


FIGURE 2.41.5: OCP—epithelial defect

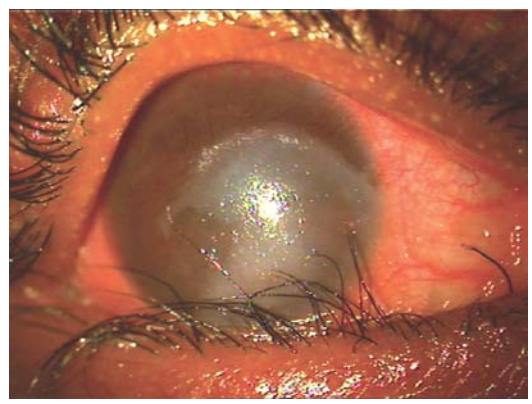


FIGURE 2.41.6: OCP—obliteration of the fornix

Stevens-Johnson Syndrome

- Muco-cutaneous vesiculobullous disease caused by a hypersensitivity reaction to certain drugs **(Fig 2.42.1)**
- Conjunctiva is involved in 50% of cases
- Muco-purulent conjunctivitis with membrane or pseudo-membrane formation (**Fig 2.42.2**)
- Oral mucous membrane lesions (**Figs 2.42.3 and 2.42.4**)
- Secondary scarring of the conjunctiva and lid margins—with trichiasis (acquired distichiasis), symblepharon (**Figs 2.42.5 and 2.42.6**) and obliteration of the fornices

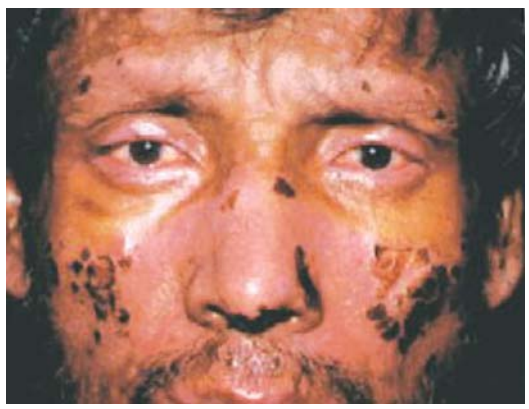


FIGURE 2.42.1: Stevens-Johnson syndrome—skin involvement

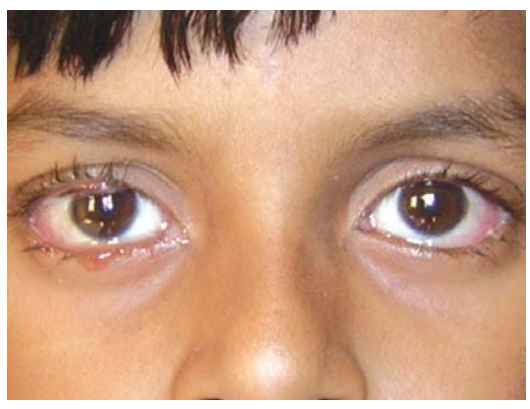


FIGURE 2.42.2: Stevens-Johnson syndrome—ocular lesion

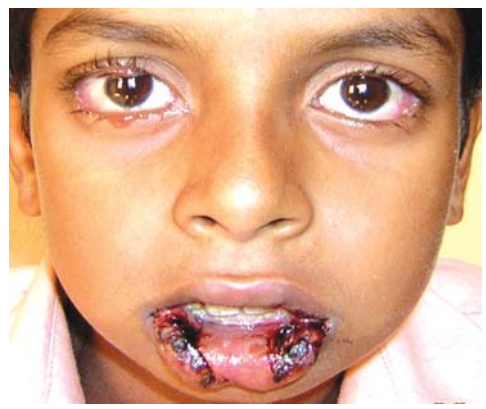


FIGURE 2.42.3: SJ syndrome—oral lesion

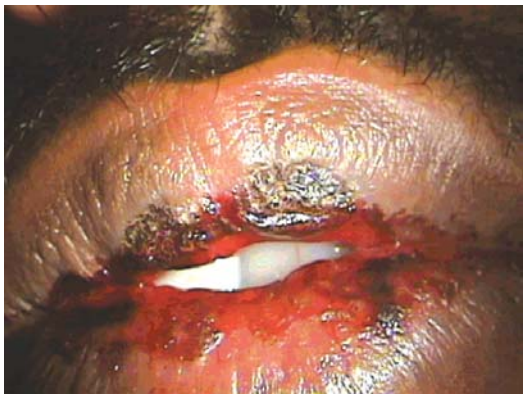


FIGURE 2.42.4: SJ syndrome—oral lesion

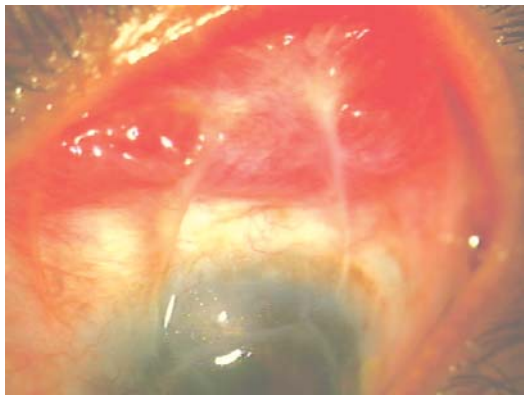


FIGURE 2.42.5: SJ syndrome—symblepharon

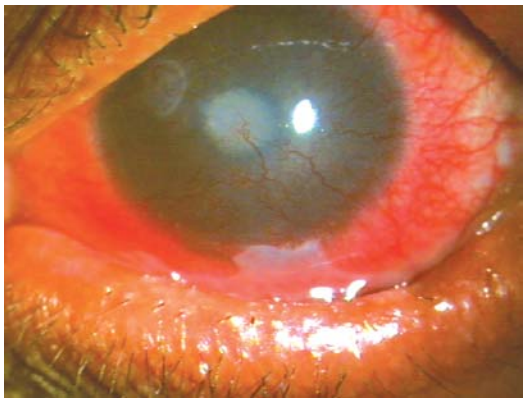


FIGURE 2.42.6: SJ syndrome—symblepharon

Superior Limbic Keratoconjunctivitis

- Bilateral keratoconjunctivitis, sometimes associated with thyroid dysfunction
- Papillary hypertrophy and thickening of superior tarsal conjunctiva **(Fig 2.43.1)**
- Hyperemia and thickening of superior bulbar conjunctiva **(Fig 2.43.2)**
- Positive Rose Bengal staining of upper part
- Filaments in adjacent cornea
- *Treatment:* tears substitutes, topical 0.05% cyclosporine, excision of upper bulbar conjunctiva

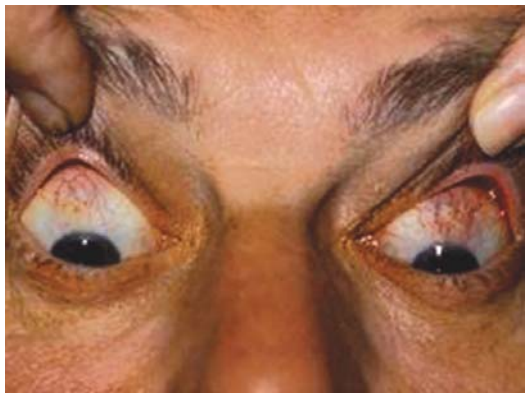


FIGURE 2.43.1: Superior limbic keratoconjunctivitis

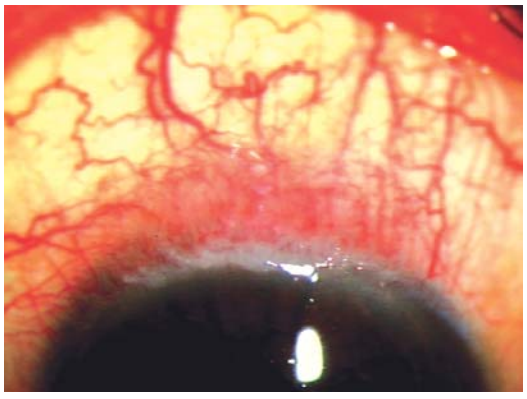


FIGURE 2.43.2: Superior limbic keratoconjunctivitis

Vascular Malformations

- *Capillary hemangioma*
  - uncommon, and may be associated with hemangiomas of lid or orbit **(Figs 2.44.1 to 2.44.3)**
  - bright red lesion of variable size, which blanches on pressure
  - may bleed following trivial trauma, or spontaneously
  - responsible for bloody tears

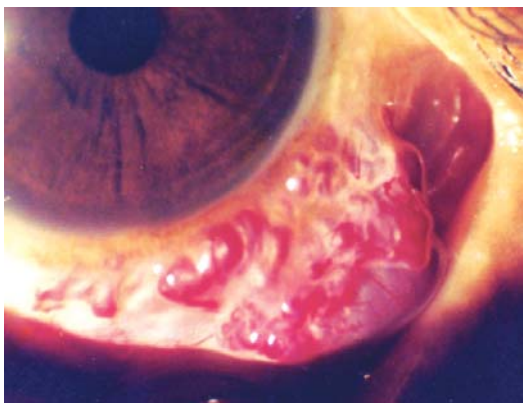


FIGURE 2.44.1: Vascular malformation—conjunctival hemangioma

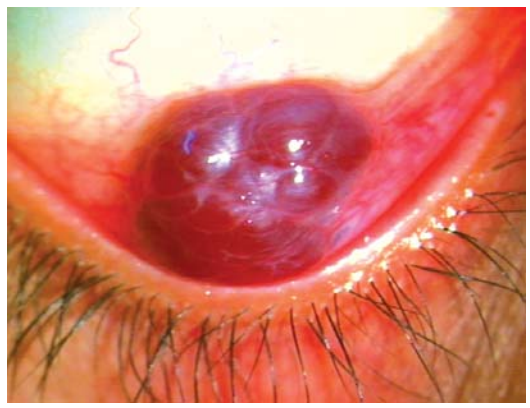


FIGURE 2.44.2: Vascular malformation—conjunctival hemangioma

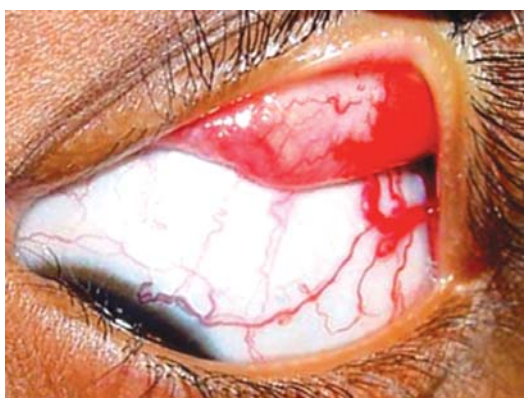


FIGURE 2.44.3: Vascular malformation—conjunctival hemangioma

- *Caput medusae*
  - Perilimbal dilated and tortuous blood vessels **(Fig 2.44.4)**
  - associated with cavernous sinus thrombosis or carotico-cavernous fistula
- *Telangiectasis*
  - dilated and tortuous blood vessels on the bulbar conjunctiva **(Fig 2.44.5)**
  - *Cause:* hematological disorder, metabolic disorders, Sturge-Weber's syndrome, etc.
  - May bleed spontaneously and responsible for bloody teres

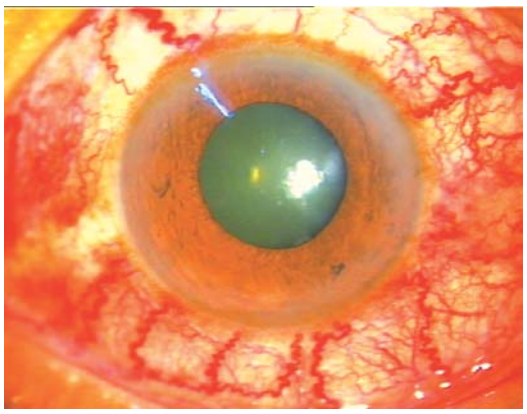


FIGURE 2.44.4: Vascular malformation—caput medusae

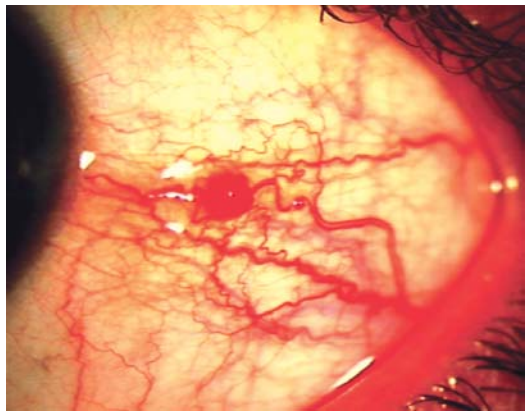


FIGURE 2.44.5: Vascular malformation

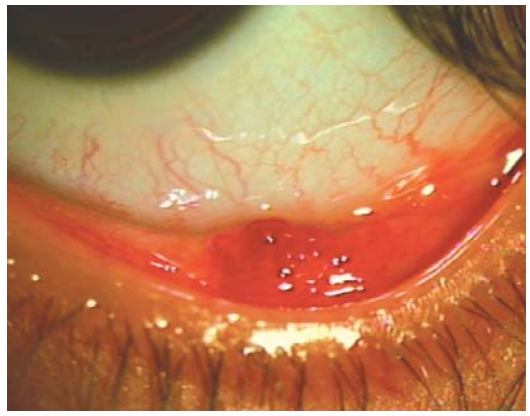


FIGURE 2.44.6: Vascular malformation

Non-Hodgkin Lymphoma

- single or multiple lesion, bilateral in 20% cases
- smooth fleshy subconjunctival infiltrate which may take the shape of a large mass
- usually located in upper or lower fornix **(Figs 2.45.1 and 2.45.2)**
- to be differentiated histopathologically from *Reactive lymphatic hyperplasia* **(Figs 2.45.3 and 2.45.4)** which is similar in appearance

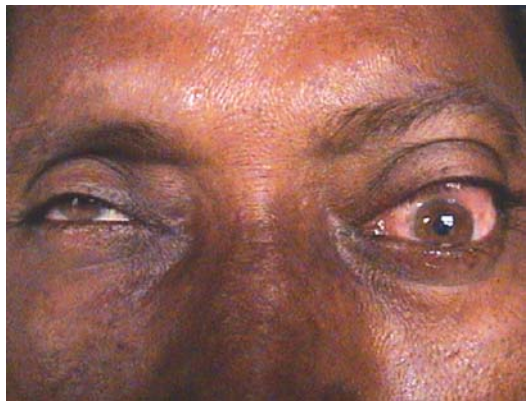


FIGURE 2.45.1: Non-Hodgkin lymphoma

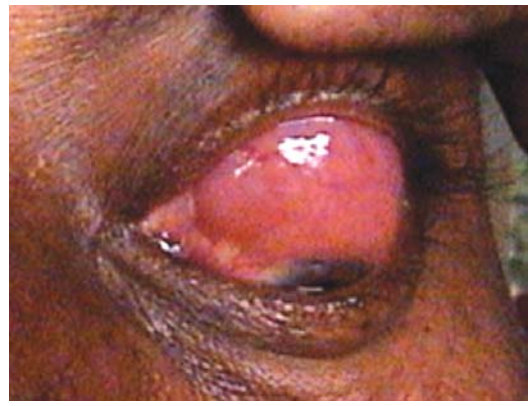


FIGURE 2.45.2: Non-Hodgkin lymphoma

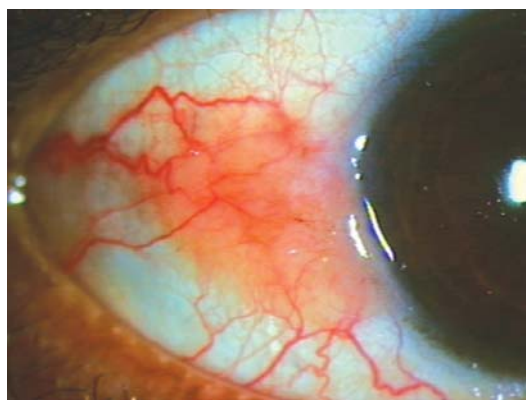


FIGURE 2.45.3: Reactive benign lymphoid hyperplasia

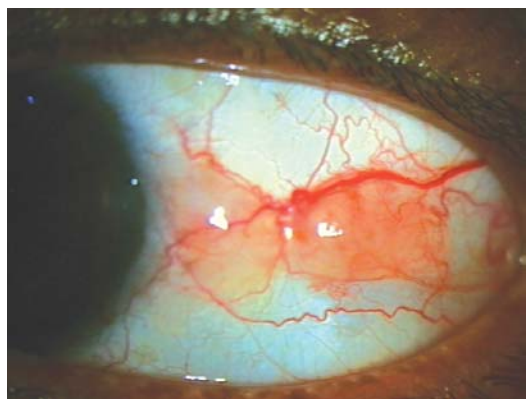


FIGURE 2.45.4: Reactive benign lymphoid hyperplasia

Amyloidosis of Conjunctiva

- Very rare condition may be associated with systemic amyloidosis
- Yellowish white lesion on the palbebral conjunctiva **(Figs 2.46.1 and Fig 2.46.2)**
- Confirmation by excision and histochemical examination

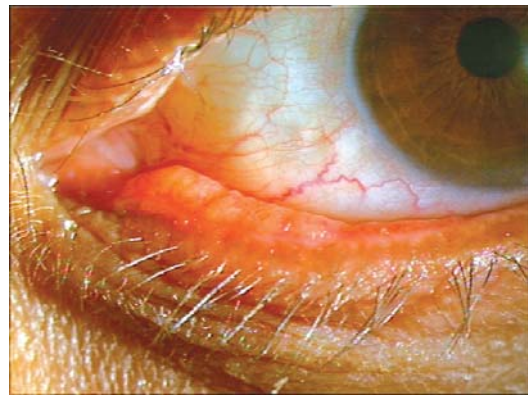


FIGURE 2.46.1: Conjunctival amyloidosis

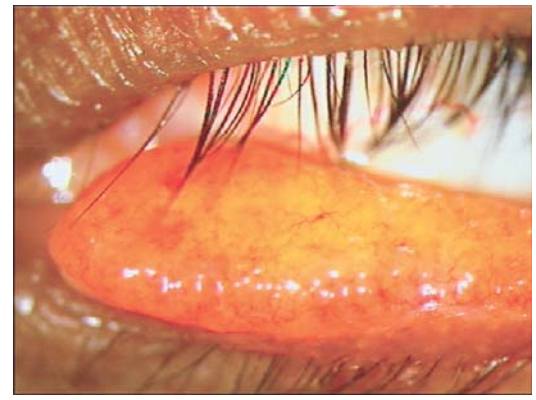


FIGURE 2.46.2: Conjunctival amyloidosis

Conjunctivochalasis

- Loose conjunctiva in old age may appear as conjunctival folds mainly in the inferior fornix **(Figs 2.47.1 and 2.47.2)**
- May be responsible for watering and recurrent conjunctivitis

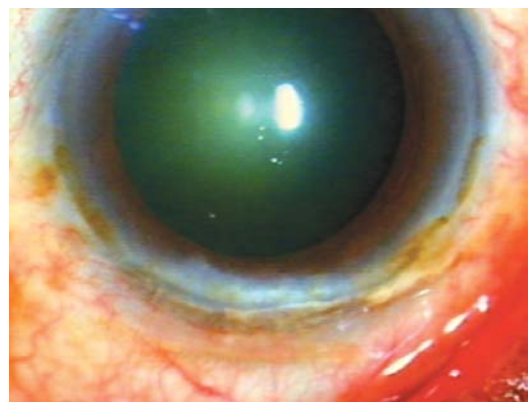


FIGURE 2.47.1: Conjunctivochalasis

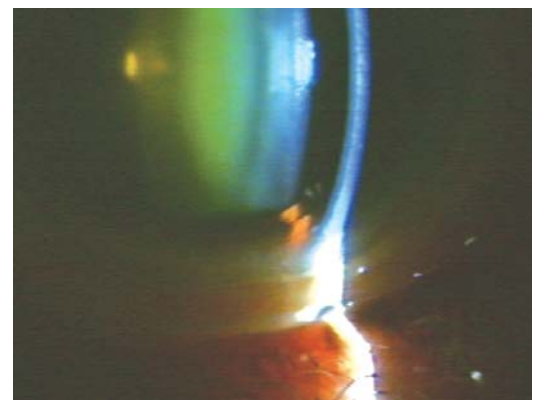


FIGURE 2.47.2: Conjunctivochalasis

Conjunctival Granulomas

- *Burst chalazion*: on tarsal conjunctiva **(Fig 2.48.1)**
- *Pyogenic granuloma*: most commonly occurs after conjunctival surgery—like, pterygium **(Fig 2.48.2)** or symblepharon **(Fig 2.48.3)**
- *Rhinosporidiosis*: rare fungal infection usually after a foreign body in the conjunctiva **(Figs 2.48.4 and 2.48.5)**

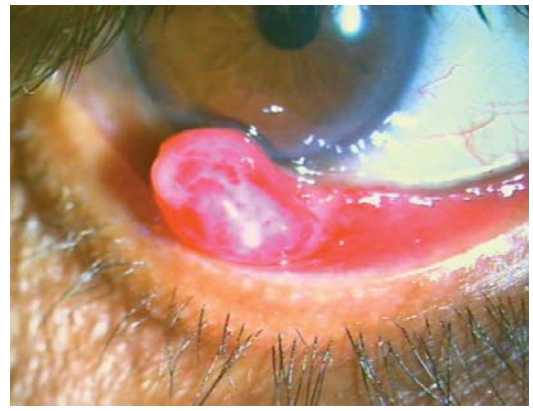


FIGURE 2.48.1: Granuloma-burst chalazion

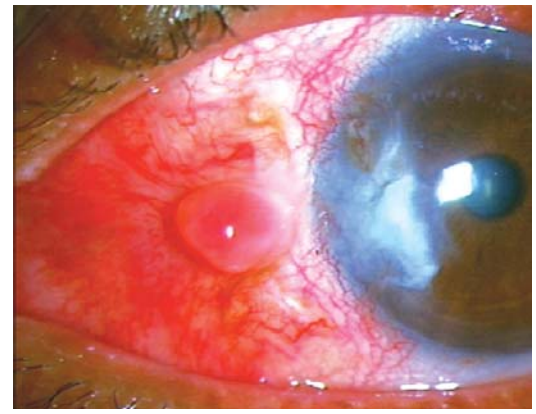


FIGURE 2.48.2: Granuloma—after pterygium operation

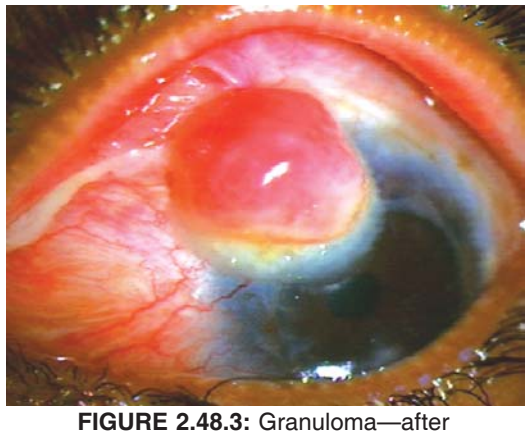


FIGURE 2.48.3: Granuloma—after symblepharon operation

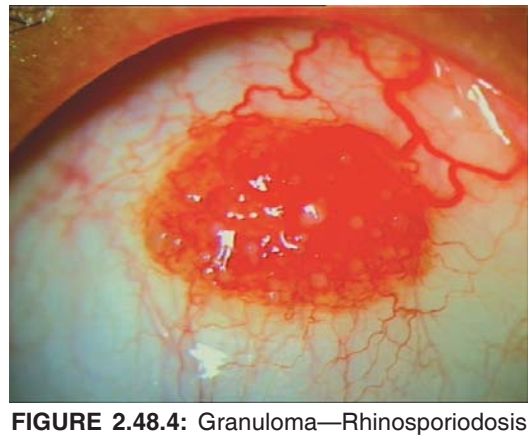


FIGURE 2.48.4: Granuloma—Rhinosporiodosis

FIGURE 2.48.5: Granuloma—Rhinosporiodosis

Xerodermal pigmentosa

- Rare, recessively inherited condition
- Photosensitive skin lesions with multiple cutaneous malignancies
- Recurrent conjunctivitis and scarring leading to conjunctival xerosis
- Corneal xerosis and clouding in late stage **(Fig 2.49.1)**
- *Treatment:* tear substitutes and protection from ultraviolet light

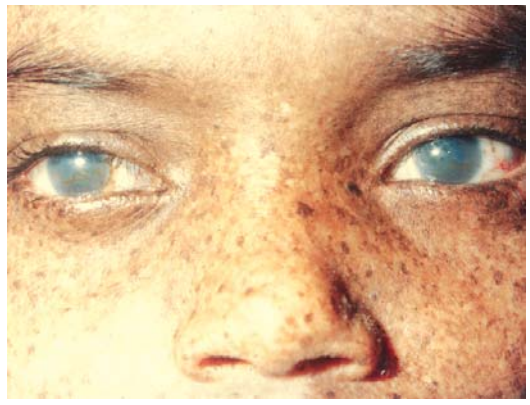


FIGURE 2.49.1: Xeroderma pigmentosa

3 Diseases of the Cornea

PIGMENT DEPOSITION IN THE CORNEA

- Iron
- Melanin
- Copper
- Other pigments

CONGENITAL CONDITIONS

- Microcornea
- Nanophthalmos
- Buphthalmos
- Sclerocornea
- Posterior embryotoxon
- Cornea plana
- Microphthalmos
- Megalocornea
- Keratoglobus
- Peters' anomaly (posterior keratoconus)
- Keratectasia

CORNEAL EDEMA

SUPPURATIVE KERATITIS

- Bacterial keratitis/ulcer
- Perforated corneal ulcer
- Corneal abscess
- Fungal keratitis (ulcer)
- Acanthameba keratitis
- Sequela of healed corneal ulcer

VIRAL KERATITIS

- Herpes simplex viral (HSV) keratitis
- Primary infection
- Recurrent HSV keratitis
- Dendritic keratitis
- Geographical or ameboid keratitis (ulcer)
- Stromal necrotic keratitis
- Metaherpetic keratitis (trophic ulcer)
- Disciform keratitis
- Herpes zoster ophthalmicus

OTHER KERATITIS (ULCER)

- Lagophthalmic (exposure) keratitis
- Neurotrophic keratitis
- Atheromatous ulcer
- Marginal keratitis
- Phlyctenular keratitis
- Interstitial keratitis
- Punctate epithelial erosions
- Punctate epithelial keratitis
- Superficial punctate keratitis of Thygesons
- Superior limbic keratoconjunctivitis
- Shield ulcer in vernal keratoconjunctivitis
- Sclerosing keratitis
- Acute stromal sclerokeratitis

PERIPHERAL THINNING AND DEGENERATIONS

- Mooren's ulcer
- Terrien's marginal degeneration
- Pellucid marginal degeneration
- Marginal ulcers associated with systemic collagen vascular disorders

CORNEAL DEGENERATIONS

- Arcus senilis (gerontoxon)
- Band-shaped keratopathy
- Salzmann's nodular degeneration
- Spheroidal degeneration (climatic droplet keratopathy)
- White limbal girdle of Vogt

CORNEAL DYSTROPHIES

ANTERIOR DYSTROPHIES (Epithelium and Bowman's Membrane)

- Map-dot-fingerprint dystrophy
- Meesmann's dystrophy
- Reis-Buckler's dystrophy

STROMAL DYSTROPHIES

- Granular dystrophy
- Macular dystrophy
- Lattice dystrophy
- Avellino (granular-lattice) dystrophy
- Central (Schnyder) crystalline dystrophy
- Congenital hereditary stromal dystrophy

POSTERIOR DYSTROPHIES

- Posterior polymorphous dystrophy
- Cornea guttata
- Fuch's endothelial dystrophy
- Congenital hereditary endothelial dystrophy

ECTATIC DYSTROPHY: KERATOCONUS

OTHER CORNEAL DISORDERS

- Striate keratopathy
- Keratoconjunctivitis sicca
- Vortex keratopathy
- Prominent corneal nerves
- Descemet's folds and wrinkles
- Corneal dellen
- Keratitis medicomentosa
- Iridocorneal endothelial (ICE) syndrome
- Corneal tumors
- Corneal abrasion
- Filamentary keratopathy
- Crocodile shagreen
- Descemet's tear
- Descemet's detachment
- Corneal signs in vitamin-A deficiency
- Tunnel abscess
- Sclero-corneal cyst

PIGMENT DEPOSITION IN THE CORNEA

Iron

Keratoconus *Fleischer's ring*	**(Fig 3.1.1)**	Epithelium
Old opacity *Hudson-Stahli line*	**(Fig 3.1.2)**	Epithelium
Pterygium *Stocker's line*	**(Fig 3.1.3)**	Epithelium
Filtering bleb *Ferry's line*	**(Fig 3.1.4)**	Epithelium
Siderosis	**(Fig 3.1.5)**	Stroma
Blood staining	**(Fig 3.1.6)**	Stroma
	(Fig 3.1.7)	

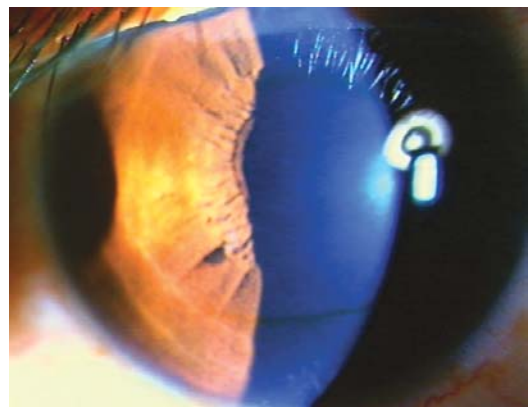


FIGURE 3.1.1: Fleischer's ring

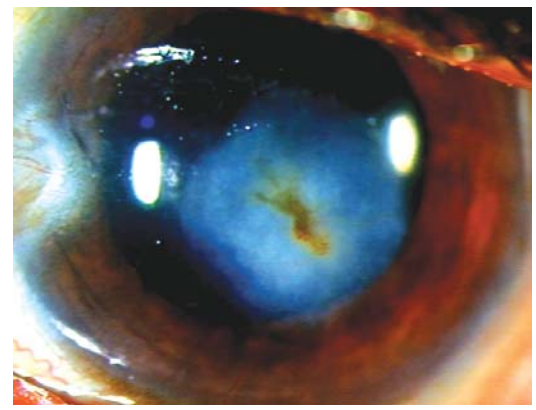


FIGURE 3.1.2: Hudson-Stahli's line

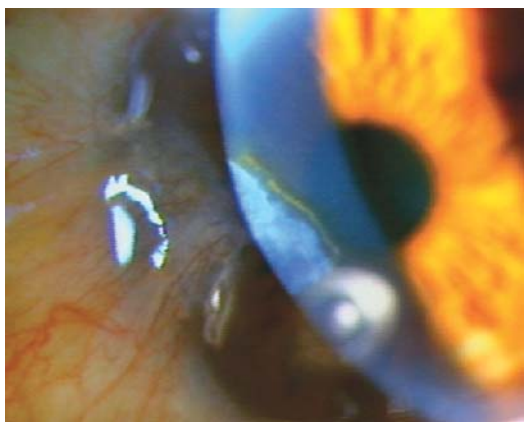


FIGURE 3.1.3: Stocker's line

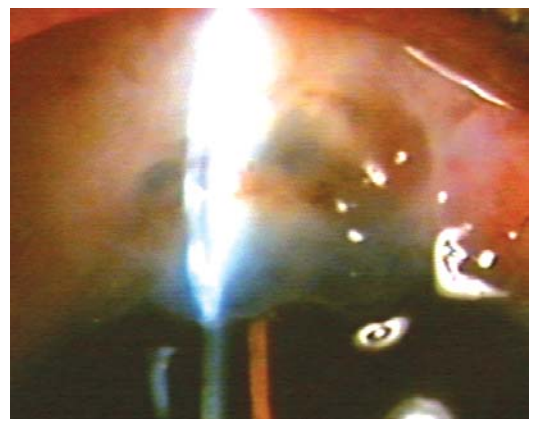


FIGURE 3.1.4: Ferry's line

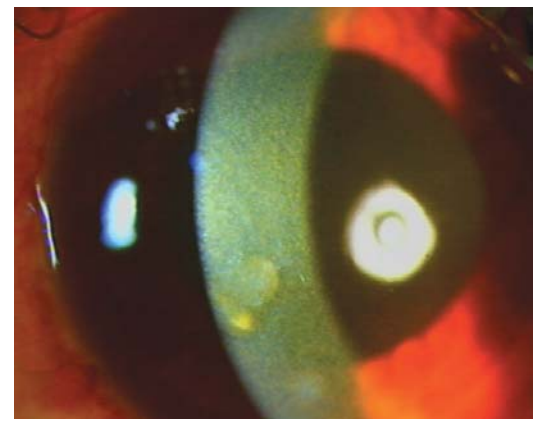


FIGURE 3.1.5: Cornea in siderosis

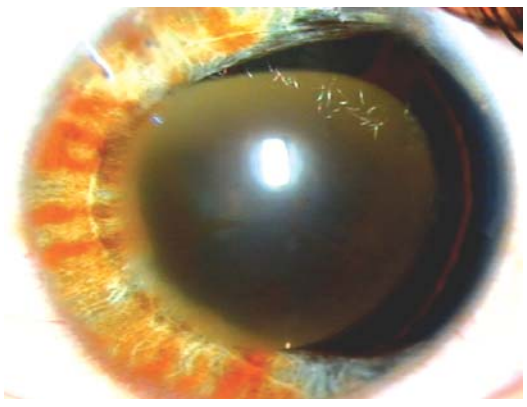


FIGURE 3.1.6: Blood staining of cornea

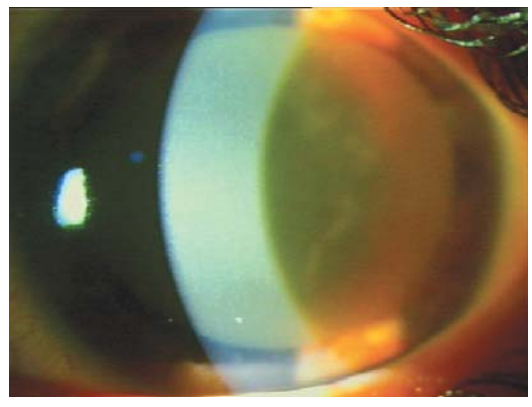


FIGURE 3.1.7: Blood staining of cornea

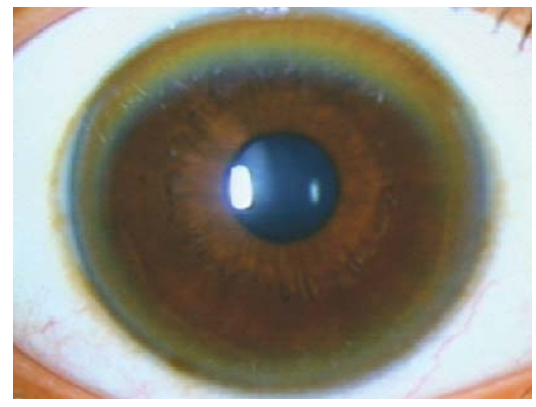


FIGURE 3.1.8: Kayser-Fleischer ring

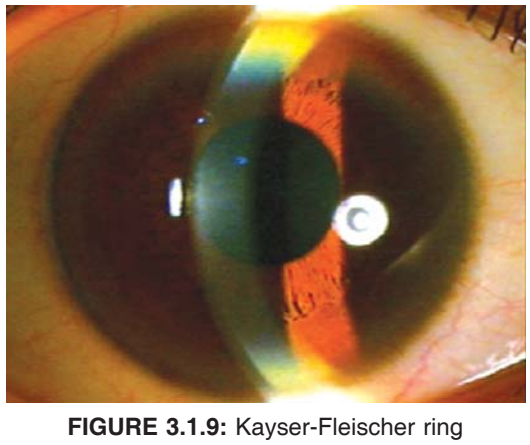


FIGURE 3.1.9: Kayser-Fleischer ring

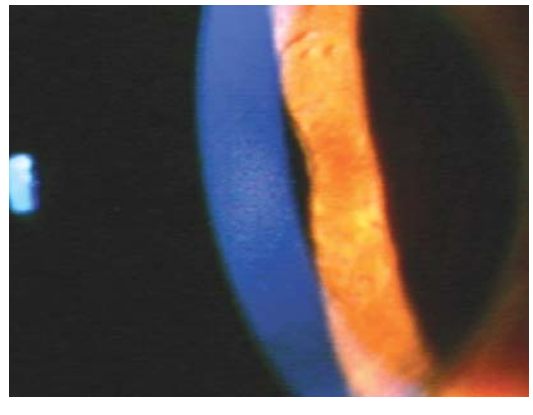


FIGURE 3.1.10: Krukenberg's spindle

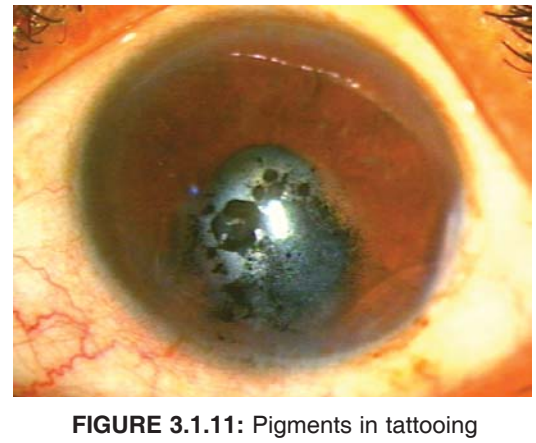


FIGURE 3.1.11: Pigments in tattooing

Copper

Wilson's disease Descemet's membrane
Kayser-Fleischer ring **(Figs 3.1.8 and 3.1.9)**

Melanin

Pigment dispersion syndrome Endothelium
Krukenberg's spindle **(Fig 3.1.10)**

Other Pigments

Corneal tattooing **(Fig 3.1.11)** Anterior stroma

CONGENITAL CONDITIONS

Microcornea

- Congenital, unilateral or bilateral condition **(Fig 3.2.1)**
- Corneal diameter is 10 mm or less
- Shallow anterior chamber
- Other ocular dimension is normal

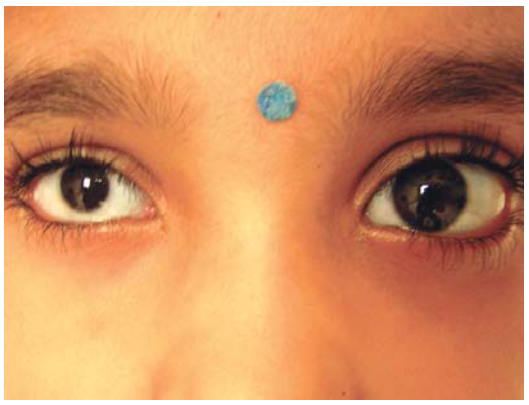


FIGURE 3.2.1: Microcornea in microphthalmos—right eye

Microphthalmos

- Unilateral or bilateral congenital abnormality in which the axial length is reduced
- In bilateral cases it may be symmetrical or asymmetrical **(Fig 3.3.1)**
- Visual acuity is usually poor and depends upon the associated anomalies
- Microphthalmos may be colobomatous or non-colobomatous
- Non-colobomatous microphthalmos may be associated with PHPV, cyst or it may occur in isolation **(Fig 3.3.2)**
- Colobomatous microphthalmos is associated with systemic syndrome or may occur in isolation

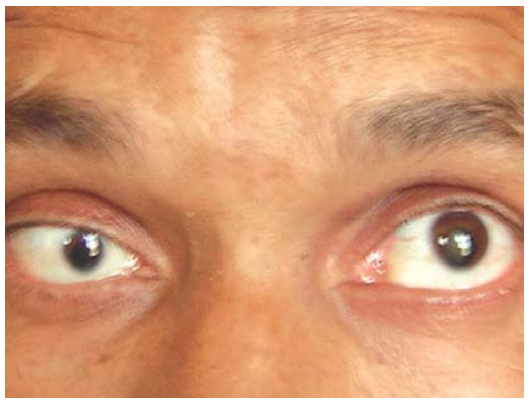


FIGURE 3.3.1: Bilateral colobomatous microphthalmos

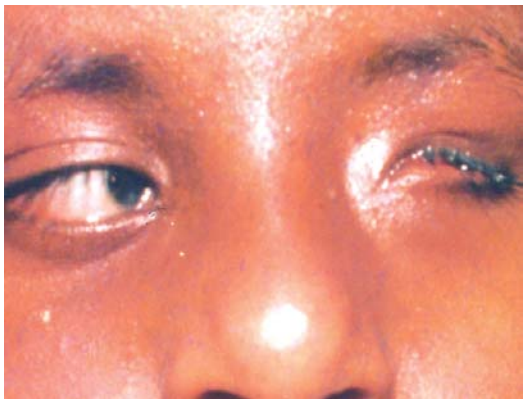


FIGURE 3.3.2: Microphthalmos and anophthalmos

Nanophthalmos

- Uncommon, congenital, bilateral condition, with small globe in all dimensions (*nano* means 'dwarf')
- Anatomical eye is grossly normal **(Fig 3.4.1)**
- Very high hypermetropia, axial length is less than 20 mm **(Fig 3.4.2)**
- Shallow anterior chamber
- Reduced anterior corneal diameter with thick sclera
- Fundus showed a crowded disk with vascular tortuosity and macular hypoplasia

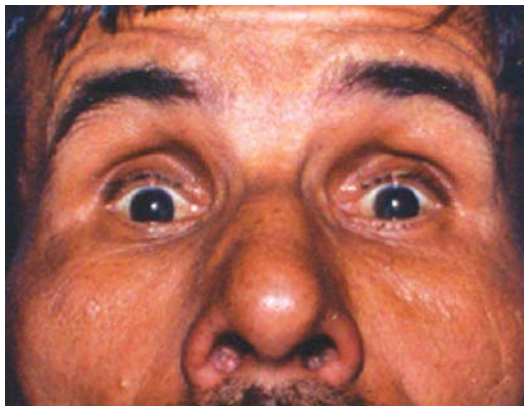


FIGURE 3.4.1: Nanophthalmos

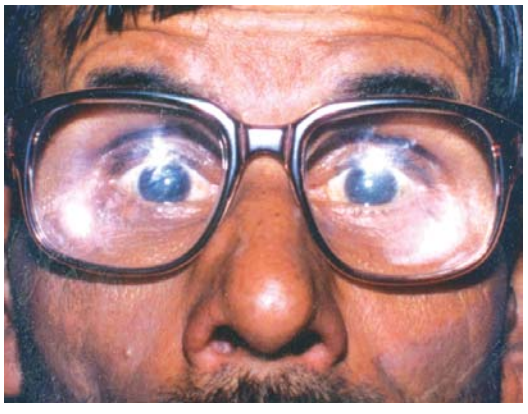


FIGURE 3.4.2: Nanophthalmos

Megalocornea

- Rare, congenital, bilateral condition **(Fig 3.5.1)**
- Corneal diameter is more than 13 mm
- Very deep anterior chamber
- High myopia and astigmatism with good visual acuity
- Normal intraocular pressure
- May be associated with lens subluxation

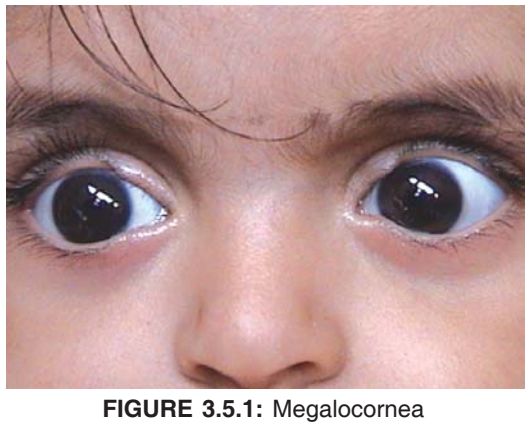


FIGURE 3.5.1: Megalocornea

Buphthalmos

- Unilateral or bilateral condition in which the eyeball is large due to stretching as a result of increased intraocular pressure within first three years of life
- Large cornea with variable scarring **(Fig 3.6.1)**
- Very deep anterior chamber
- Horizontal tear of Descemet's membrane (Haab's striae)
- *See Chapter: 9*

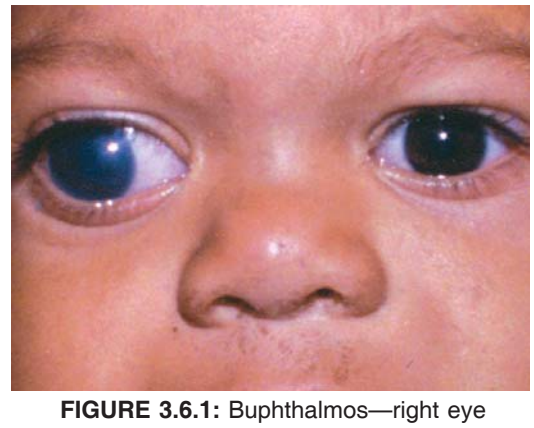


FIGURE 3.6.1: Buphthalmos—right eye

Keratoglobus

- Congenital bilateral very rare condition
- Mid-peripheral thinning resulting in protrusion or bulging of whole cornea, with an appearance of globular shape **(Fig 3.7.1)**
- Very deep anterior chamber
- Acute hydrops in extreme cases
- May be associated with other systemic abnormalities

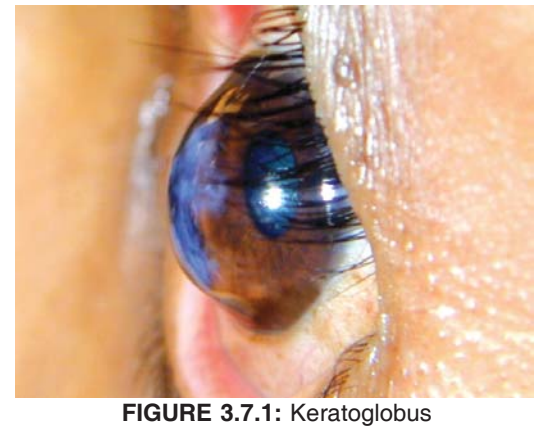


FIGURE 3.7.1: Keratoglobus

Sclerocornea

- Rare, congenital, bilateral condition
- Opacification and vascularization of peripheral or entire cornea **(Fig 3.8.1)**
- Cornea appears smaller if the scleralization is only peripheral **(Fig 3.8.2)**
- May be associated with microphthalmos, blue sclera or cornea plana

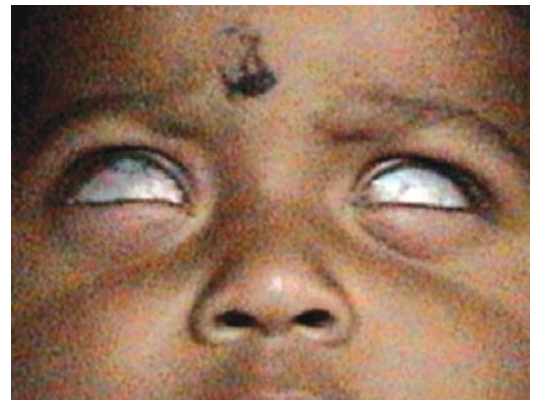


FIGURE 3.8.1: Sclerocornea—total

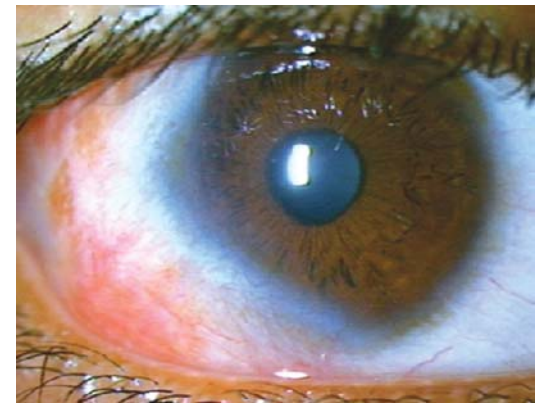


FIGURE 3.8.2: Sclerocornea—partial

Peters' Anomaly (Posterior Keratoconus)

- Central corneal opacity is due to defect in embryogenesis
- Incomplete separation of the lens from the surface ectoderm
- In milder form, it only causes posterior keratoconus **(Figs 3.9.1 and 3.9.2)**
- But in most cases, it is associated with anterior polar cataract, iris adhesion, angle abnormalities, or even secondary glaucoma **(Fig 3.9.3)**

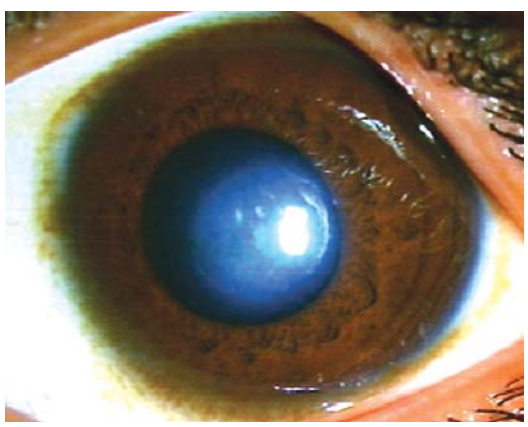


FIGURE 3.9.1: Posterior keratoconus

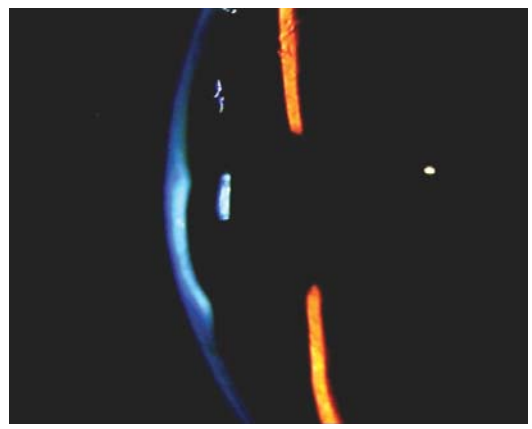


FIGURE 3.9.2: Posterior keratoconus

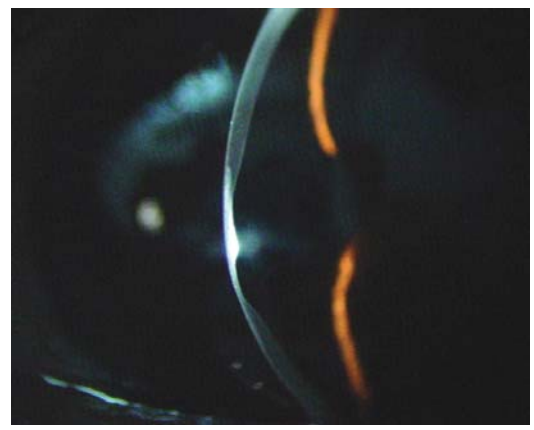


FIGURE 3.9.3: Peters' anomaly

Posterior Embryotoxon

- An unusual prominence of Schwalbe's line which is the peripheral termination of Descemet's membrane **(Fig 3.10.1)**
- Appears as a ring opacity or in part in the deeper layer of the cornea **(Fig 3.10.2)**

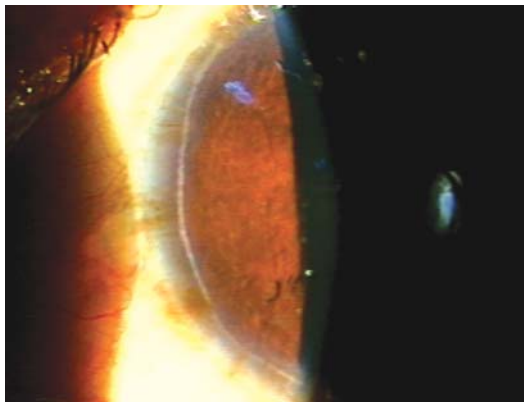


FIGURE 3.10.1: Posterior embryotoxon

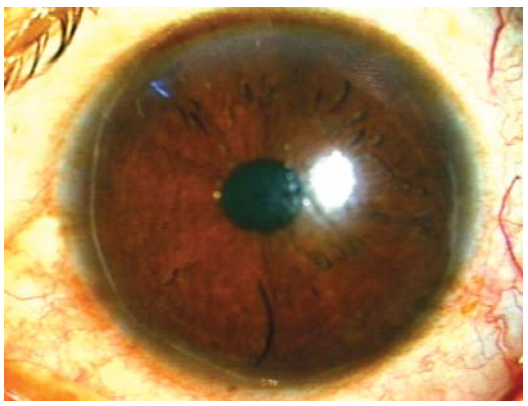


FIGURE 3.10.2: Posterior embryotoxon

Cornea Plana

- Rare, congenital, bilateral condition
- Severe decrease in corneal curvature **(Fig 3.11.1)**
- High hypermetropia
- Shallow anterior chamber **(Fig 3.11.2)**
- May be associated with microcornea and sclerocornea

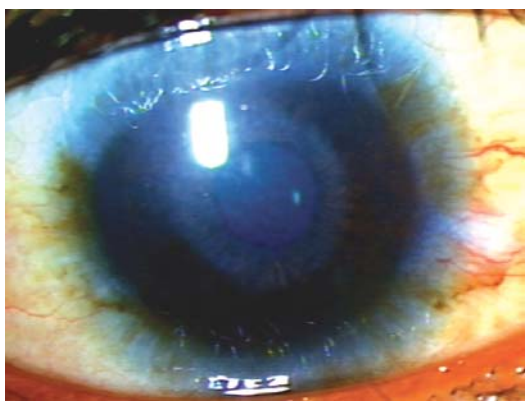


FIGURE 3.11.1: Cornea plana

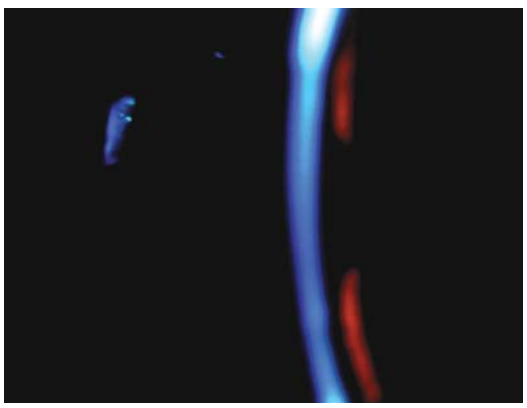


FIGURE 3.11.2: Cornea plana

Keratectasia

- Rare, usually unilateral condition, probably due to intrauterine keratitis or maternal vitamin-A deficiency followed by perforation
- Severe corneal ectasia beyond the eyelid with opacification and vascularization **(Fig 3.12.1)**

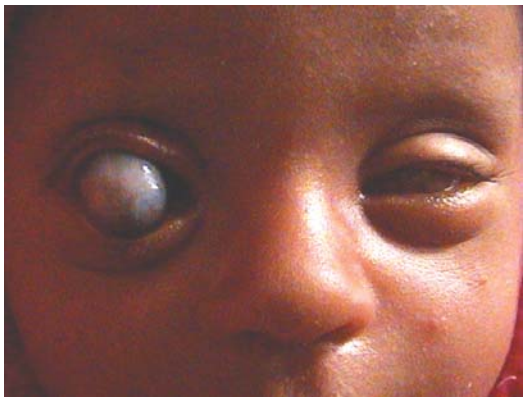


FIGURE 3.12.1: Keratectasia

Corneal Edema

- Associated with increased in corneal thickness due to accumulation of fluid **(Fig 3.13.1)**
- Variable degree of loss in corneal transparency **(Fig 3.13.2)**
- Edema may be focal or generalized **(Figs 3.13.3 and 3.13.4)**, may be with epithelial defect **(Fig 3.13.5)**
- In long-standing cases, the epithelium tends to be raised into large vesicles or bullae, leading to bullous keratopathy **(Figs 3.13.6 and 3.13.7)**
- *Important causes:* inflammation, trauma, increased IOP, endothelial dysfunction as in Fuchs' dystrophy, hypoxia of the cornea as in contact lens wearer
- *Treatment:* hypertonic saline, thin conjunctival flap, anterior stromal puncture and BCL, penetrating keratoplasty

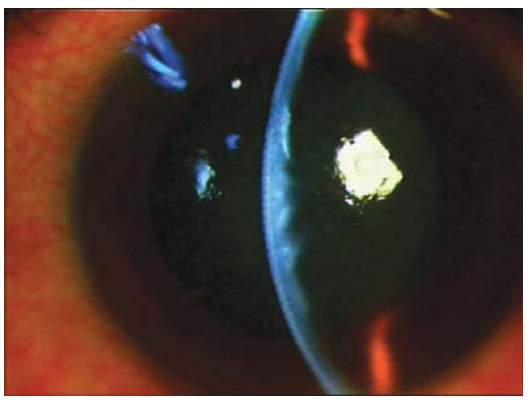


FIGURE 3.13.1: Corneal edema—increased thickness

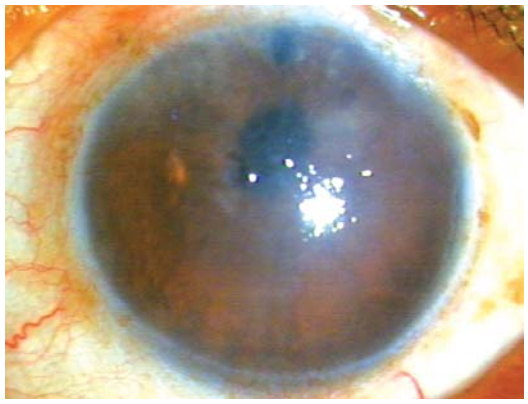


FIGURE 3.13.2: Corneal edema

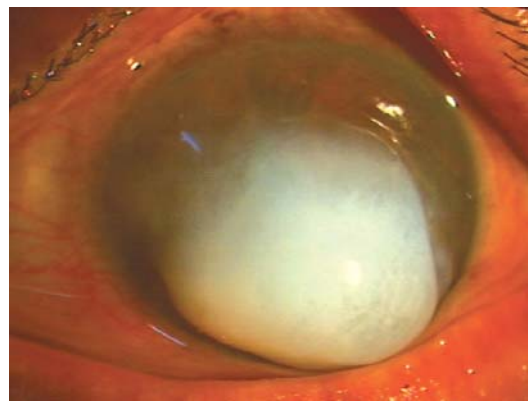


FIGURE 3.13.3: Corneal edema—focal

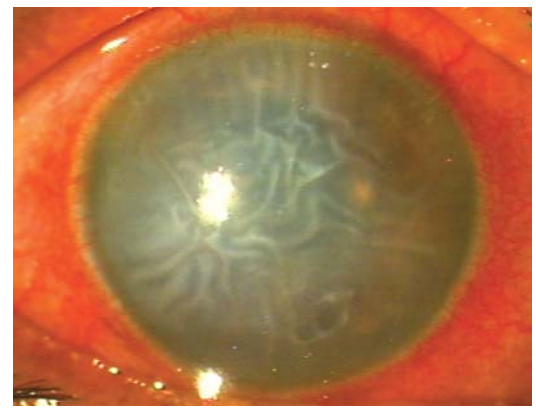


FIGURE 3.13.4: Corneal edema—generalized

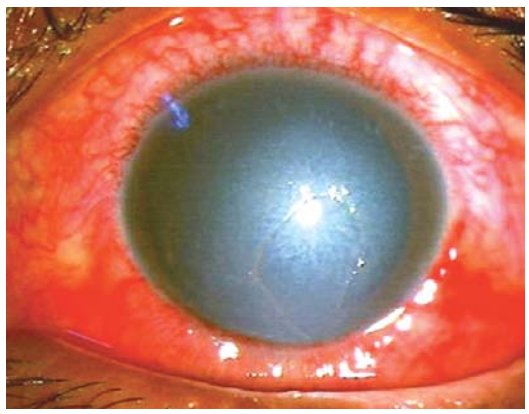


FIGURE 3.13.5: Corneal edema and epithelial defect—chemical burn

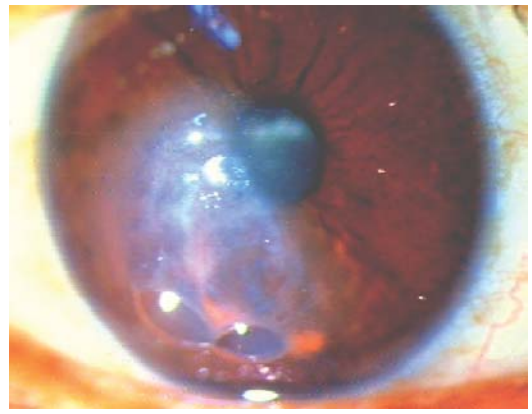


FIGURE 3.13.6: Bullous keratopathy

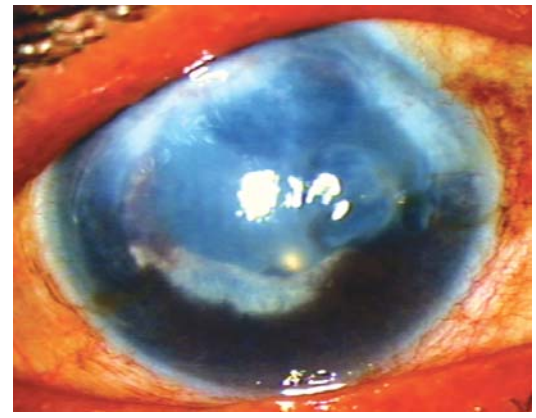


FIGURE 3.13.7: Bullous keratopathy

SUPPURATIVE KERATITIS

- Exogenous infection is most common and almost always associated with some kind of trauma
- Sometimes associated with underlying other ocular factors, e.g. corneal erosion, bullous keratopathy, herpetic keratitis, lagophthalmos, dry eyes, chronic dacryocystitis, etc.
- Other factors like, use of topical steroids, diabetes, immunecompromise states are also important
- May be *bacterial*, *fungal* or *acanthamebal*

Bacterial Keratitis/Ulcer

- The most frequent causative agents are *Staphylococcus aureus*, *Pseudomonas aeruginosa* and *Streptococcus pneumoniae*
- Signs vary with the severity and to some extent on causative agent
- Lid edema, marked ciliary congestion
- Epithelial breakdown followed by stromal suppuration
- Ulcer usually starts as a grayish-white circumscribed infiltration **(Figs 3.14.1 and 3.14.2)**
- Edema of the surrounding tissue

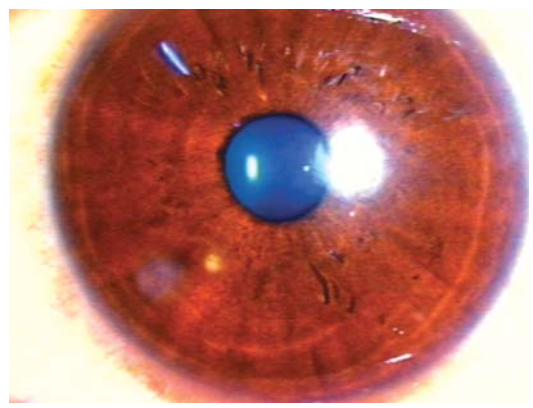


FIGURE 3.14.1: Bacterial ulcer—infiltration

FIGURE 3.14.2: Bacterial ulcer—infiltration

- Margins are over hanging and the floor is covered by necrotic material
- Overall look of the ulcer is wet **(Fig 3.14.3)**
- Hypopyon may be present in variable amount with flat border **(Fig 3.14.4)**
- Secondary anterior uveitis and glaucoma in some cases
- Specific features:
  - *Staphylococcal keratitis:* well-defined grayish-white or creamy stromal infiltrate which may progress to form dense stromal abscess. The surrounding cornea is relatively clear **(Figs 3.14.5 and 3.14.6)**
  - *Pneumococcal keratitis:* spread superficially with a serpiginous leading edge and associated with severe anterior uveitis with hypopyon formation. The surrounding cornea is relatively clear **(Fig 3.14.7)**
  - *Pseudomonas keratitis:* rapidly spreading, melting suppurative lesion associated with hypopyon and greenish mucopurulent discharge. 'Ground glass' appearance of the surrounding cornea **(Fig 3.14.8)**
  - *Enterobacteriaceae (E coli, Proteus* or *Klebsiella):* shallow ulcer with pleomorphic grayish-white necrotic areas. Sometimes they produce ring shaped corneal infiltrates **(Fig 3.14.9)**
  - *Nocardia keratitis:* multiple anterior stromal pin-head like infiltrations, typically arranged in the form of a ring, called the wreath pattern **(Fig 3.14.10)**

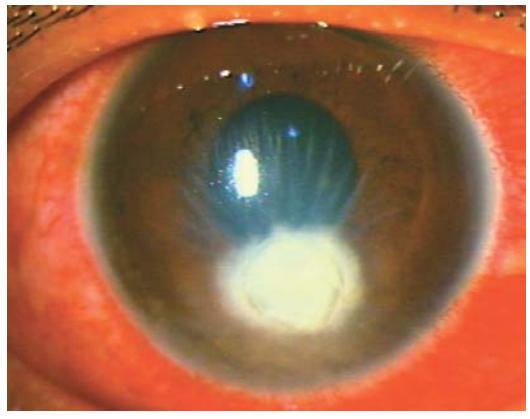


FIGURE 3.14.3: Bacterial ulcer

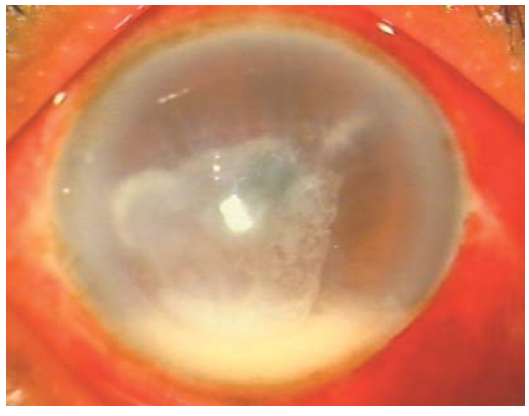


FIGURE 3.14.4: Bacterial ulcer

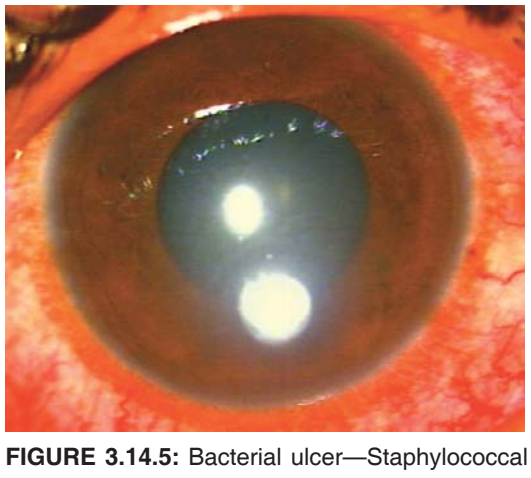


FIGURE 3.14.5: Bacterial ulcer—Staphylococcal

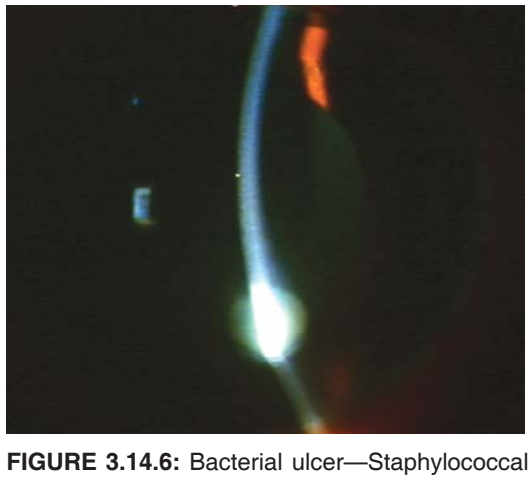


FIGURE 3.14.6: Bacterial ulcer—Staphylococcal

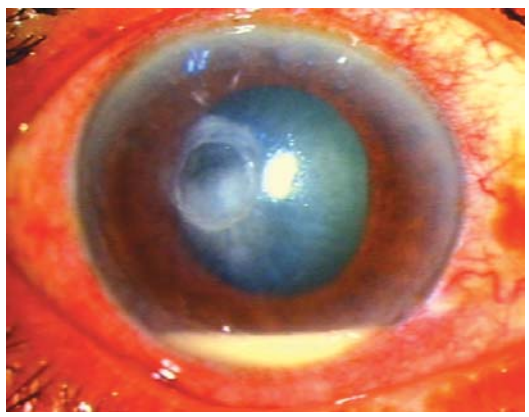


FIGURE 3.14.7: Bacterial ulcer with hypopyon—Pneumococcal

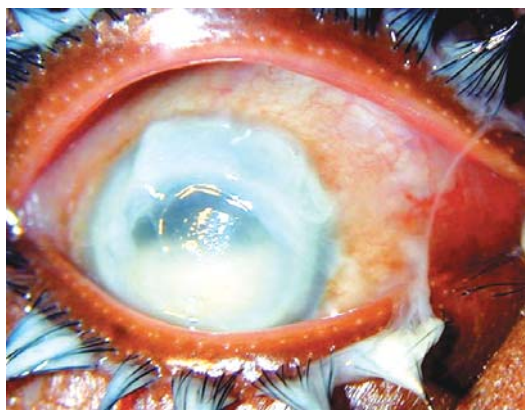


FIGURE 3.14.8: Bacterial ulcer—Pseudomonas

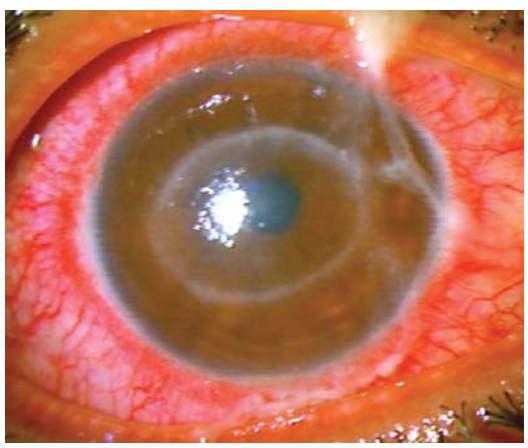


FIGURE 3.14.9: Bacterial ulcer—Enterobacteriaceae

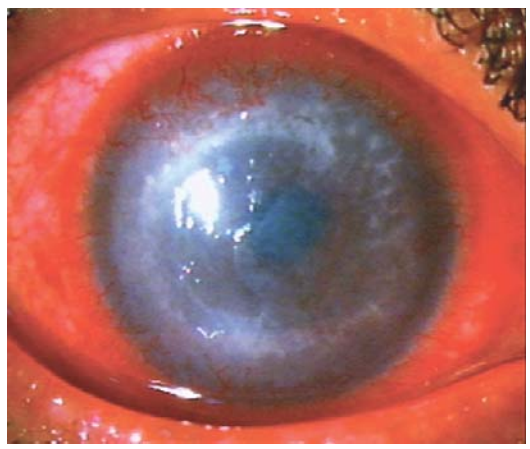


FIGURE 3.14.10: Nocardia keratitis

Perforated Corneal Ulcer

- Corneal thinning and perforation may occur in any type of corneal ulcer in advance cases **(Figs 3.15.1 and 3.15.2)**
- Prior to that *Descemetocele, i.e.,* herniation of the elastic Descemet's membrane as a transparent vesicle **(Fig 3.15.3)**
- Other types of presentation
  - *corneal fistula:* small perforation without iris prolapse **(Figs 3.15.4 and 3.15.5)**. A positive Seidel's test confirms the diagnosis **(Figs 3.15.6 and 3.15.7)**
  - *iris prolapse:* If the perforation is large peripheral or paracentral **(Figs 3.15.8 and 3.15.9)**
  - *pseudocornea formation* **(Fig 3.15.10)**
  - *perforation with extrusion of intraocular contents* **(Figs 3.15.11 and 3.15.12)**
  - *panophthalmitis* **(Figs 3.15.13 and 3.15.14)**

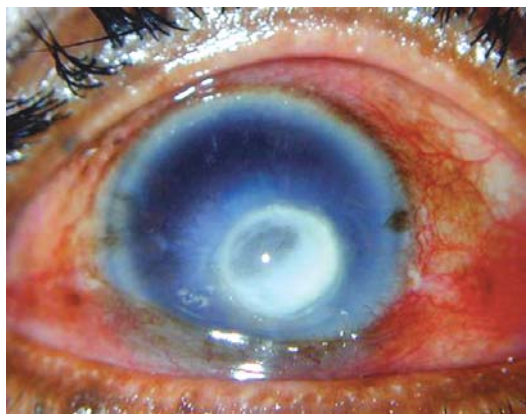


FIGURE 3.15.1: Bacterial ulcer—thinning

FIGURE 3.15.2: Bacterial ulcer—perforation

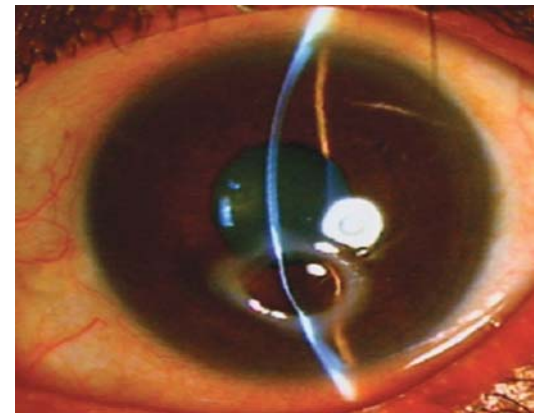


FIGURE 3.15.3: Descemetocele

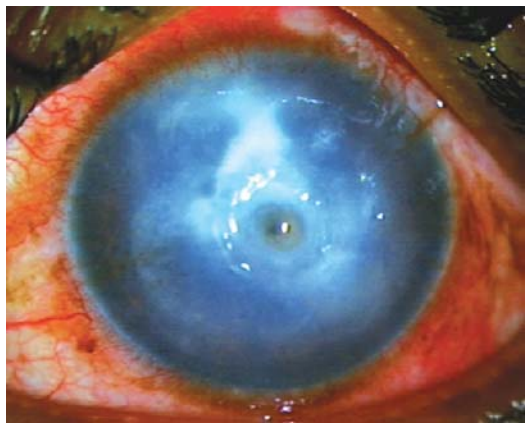


FIGURE 3.15.4: Corneal fistula

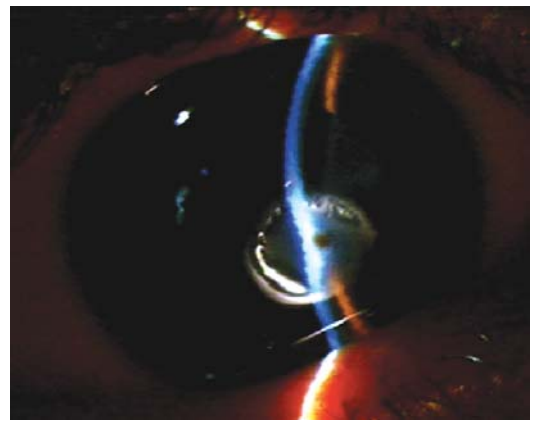


FIGURE 3.15.5: Corneal fistula

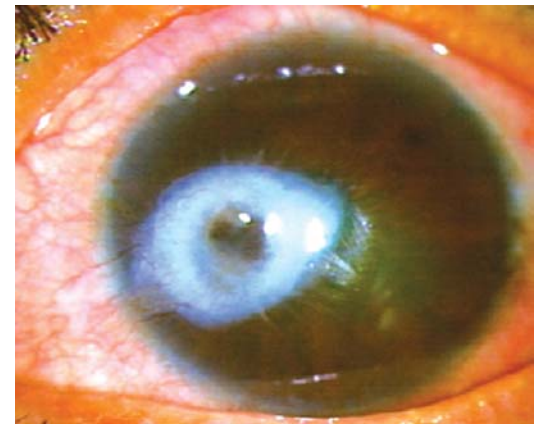


FIGURE 3.15.6: Corneal fistula

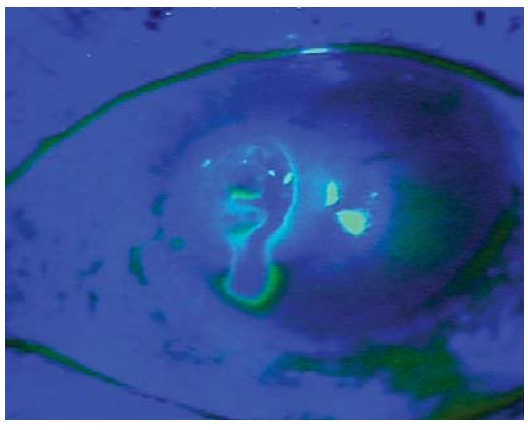


FIGURE 3.15.7: Corneal fistula—Seidel's test positive

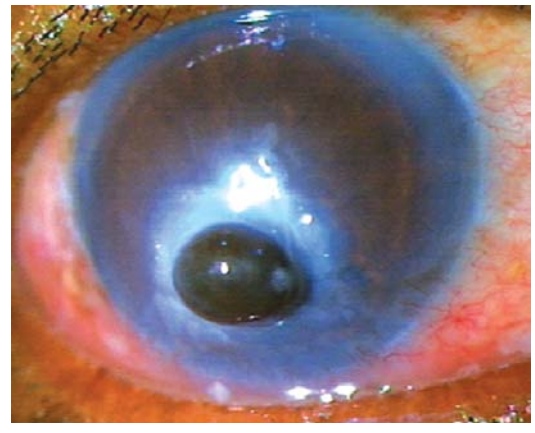


FIGURE 3.15.8: Perforation—iris prolapse

FIGURE 3.15.9: Perforation—iris prolapse

FIGURE 3.15.10: Perforation—pseudocornea

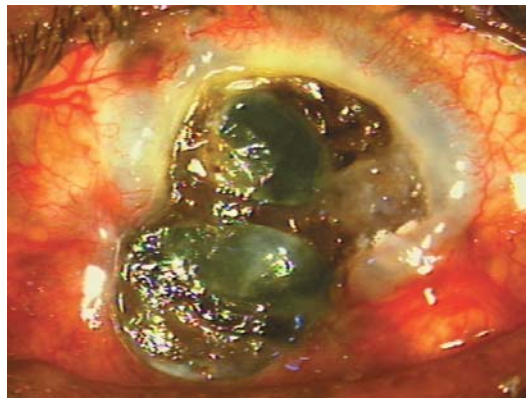


FIGURE 3.15.11: Perforation—extrusion of contents

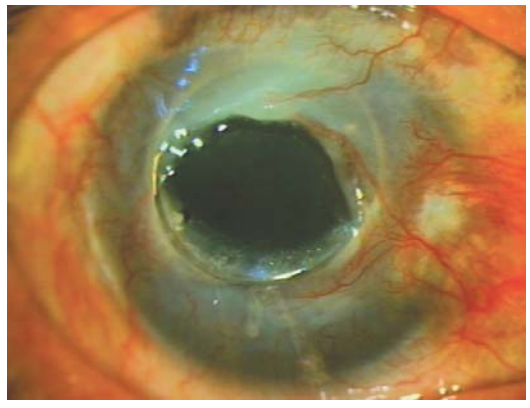


FIGURE 3.15.12: Perforation—extrusion of contents (IOL)

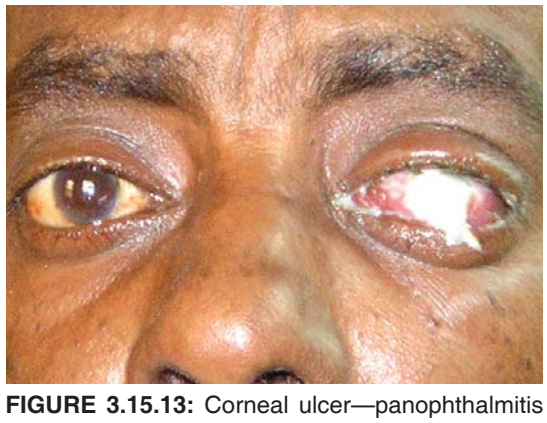


FIGURE 3.15.13: Corneal ulcer—panophthalmitis

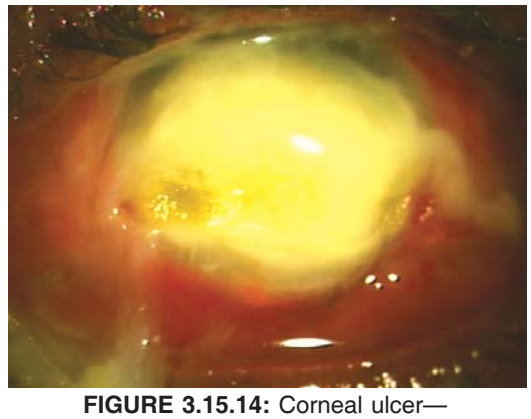


FIGURE 3.15.14: Corneal ulcer—panophthalmitis

Corneal Abscess

- Localized suppuration in the deeper stroma under intact corneal epithelium **(Figs 3.16.1 and 3.16.2)**
- Usually associated with Staphylococcal infection

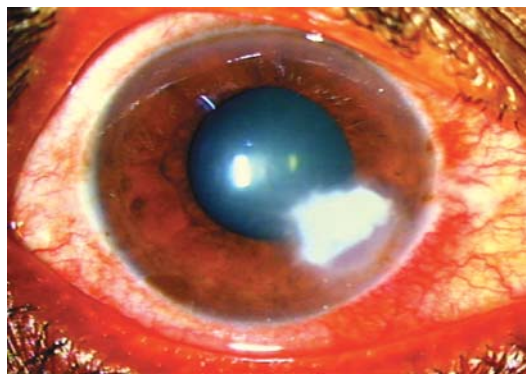


FIGURE 3.16.1: Corneal abscess

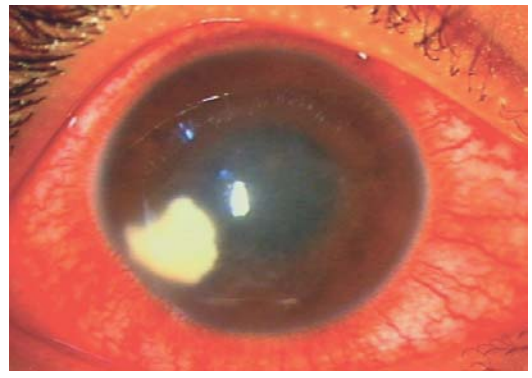


FIGURE 3.16.2: Deep corneal abscess

Fungal Keratitis or Corneal Ulcer

- The causative agent may be *Filamentous* fungi or *Yeast (Candida)*
- *Filamentous fungi* are more common in tropical agricultural areas, whereas *Candida* frequently affects the immunocompromised individual and post PK patient
- Typically preceded by ocular trauma often trivial in nature
- Mainly by agricultural and vegetable matters
- Dry looking, yellowish-white lesion with indistinct margin **(Fig 3.17.1)**
- Delicate, feathery, finger-like projections into the adjacent stroma **(Fig 3.17.2)**
- Overlying epithelium is elevated and may be intact **(Fig 3.17.3)**

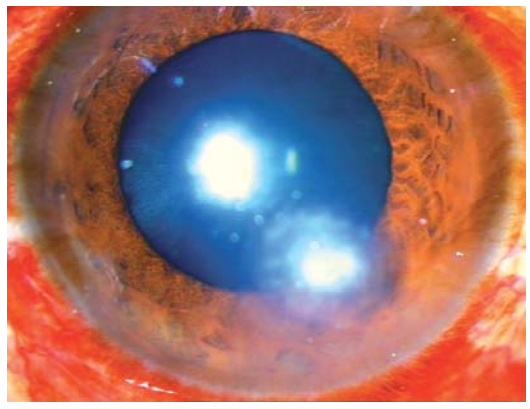


FIGURE 3.17.1: Fungal keratitis

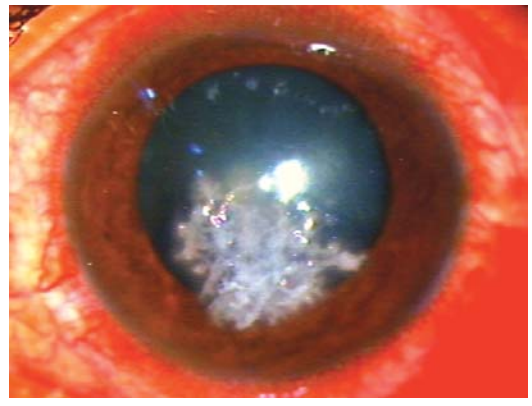


FIGURE 3.17.2: Fungal keratitis—feathery edge

- Massive dense hypopyon which is immobile with convex upper border **(Fig 3.17.4)**
- May also cause ring infiltrations **(Fig 3.17.5)**
- Secondary bacterial infection (mixed infection) is common in some cases **(Fig 3.17.6)**
- Slowly progressive stromal destruction may lead to corneal perforation with its sequelae **(Fig 3.17.7)**
- *Candida keratitis:* gray-white infiltrate (often as collar-button abscess) similar to bacterial ulcer **(Figs 3.17.8 to 3.17.10)**
- *Filamentary keratitis:* typical feathery appearance with finger-like projection and satellite lesion **(See Fig 3.17.2 and Fig 3.17.11)**
- *Dematecious fungal keratitis:* often produces pigments on the surface of the ulcer **(Figs 3.17.12 and 3.17.13)**

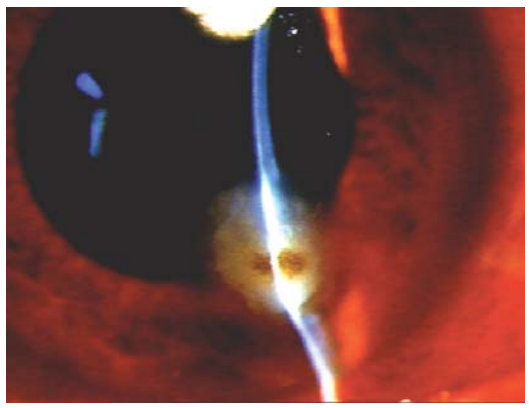


FIGURE 3.17.3: Fungal keratitis—elevated surface

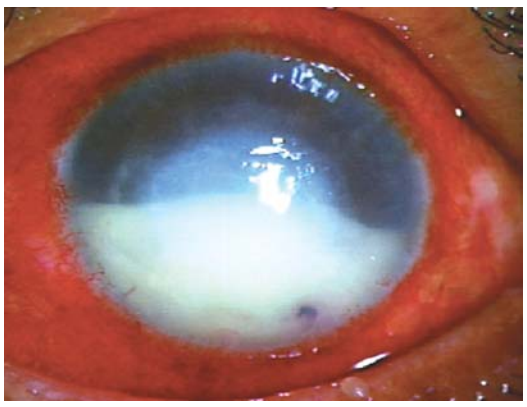


FIGURE 3.17.4: Fungal ulcer—convex hypopyon

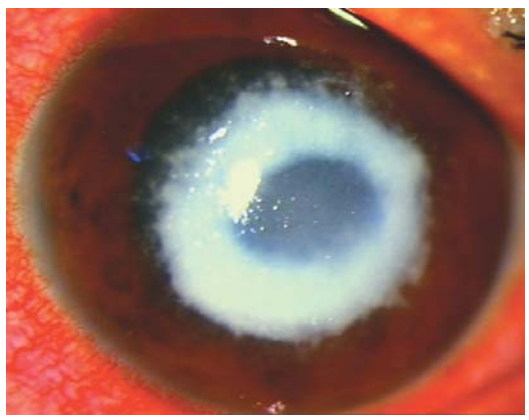


FIGURE 3.17.5: Fungal ulcer—ring infiltration

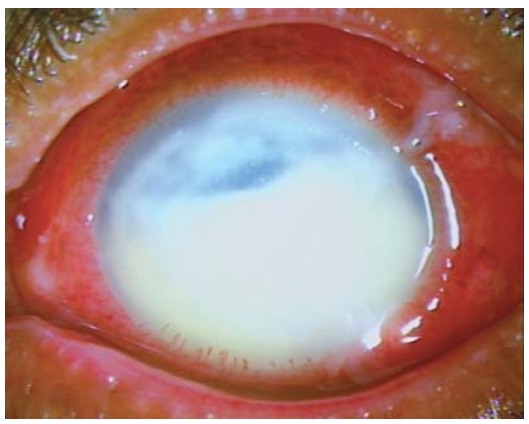


FIGURE 3.17.6: Fungal ulcer—mixed bacterial infection

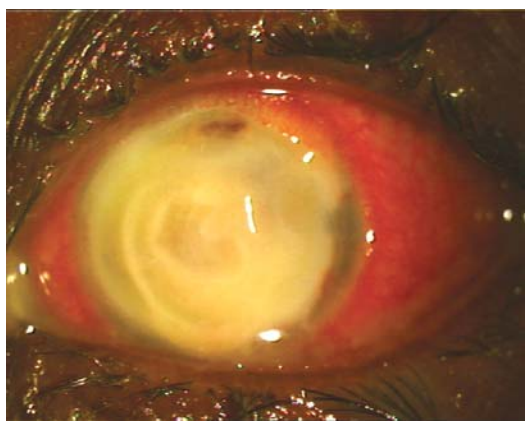


FIGURE 3.17.7: Fungal ulcer—severe

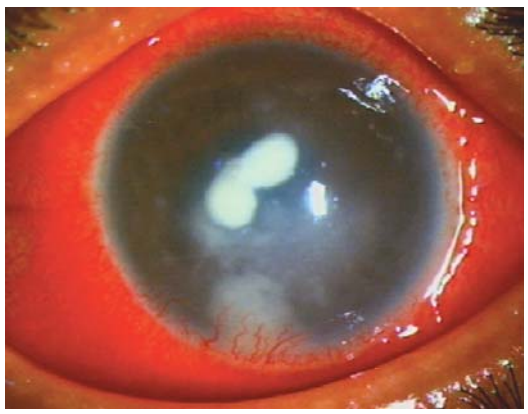


FIGURE 3.17.8: Candida keratitis—immunocompromised

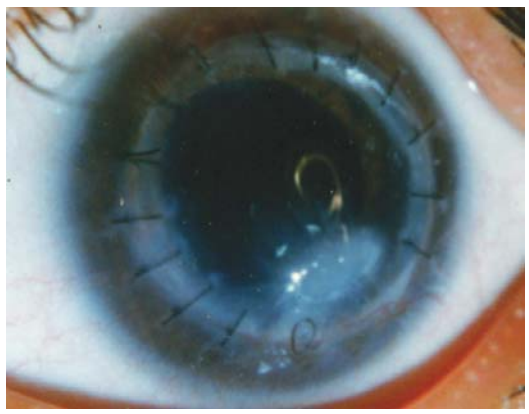


FIGURE 3.17.9: Candida keratitis—post PK

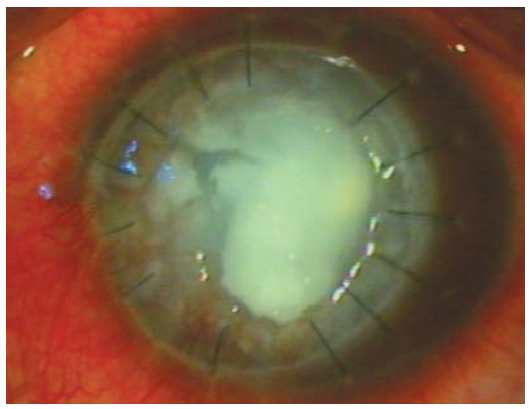


FIGURE 3.17.10: Candida keratitis—post PK

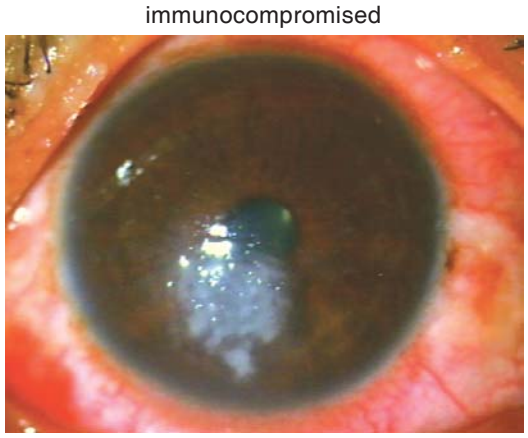


FIGURE 3.17.11: Filamentary fungal keratitis

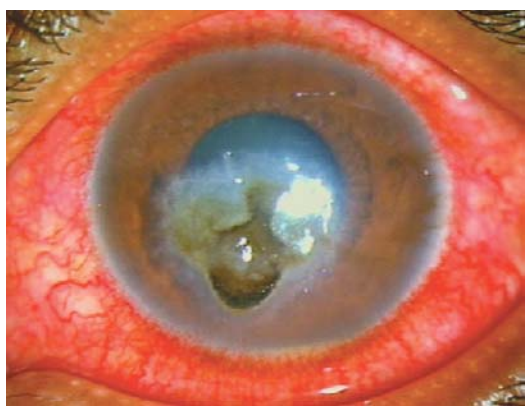


FIGURE 3.17.12: Fungal ulcer—Dematecious

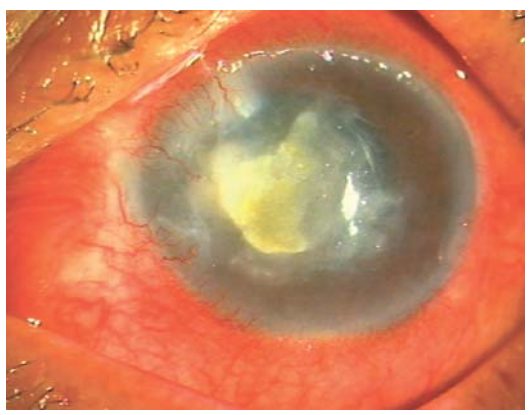


FIGURE 3.17.13: Fungal ulcer—Dematecious

Acanthameba Keratitis

- Very rare unilateral keratitis that typically affects the soft contact lens wearer
- Non-specific stromal infiltrates, not responding to usual antibacterial or antifungal
- Radial keratoneuritis
- Progressive chronic stromal keratitis with recurrent breakdown of corneal epithelium **(Fig 3.18.1)**
- Paracentral ring-shaped ulcer or abscess is the hallmark of advanced infection **(Figs 3.18.2 and 3.18.3)**
- Stromal necrosis and thinning which may lead to perforation

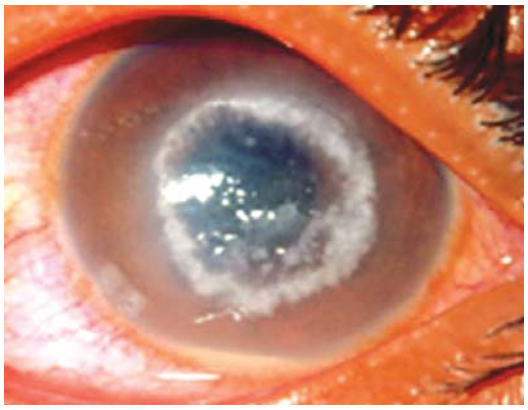


FIGURE 3.18.1: Acanthameba keratitis

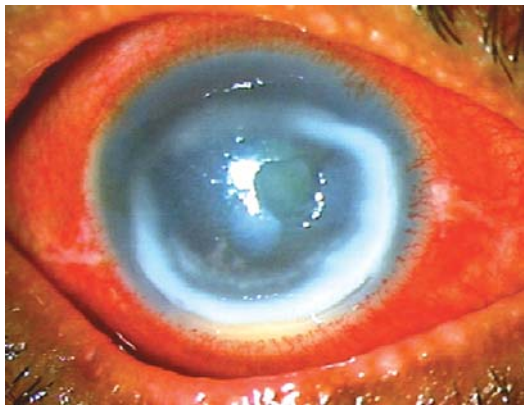


FIGURE 3.18.2: Acanthameba keratitis—ring infiltrates

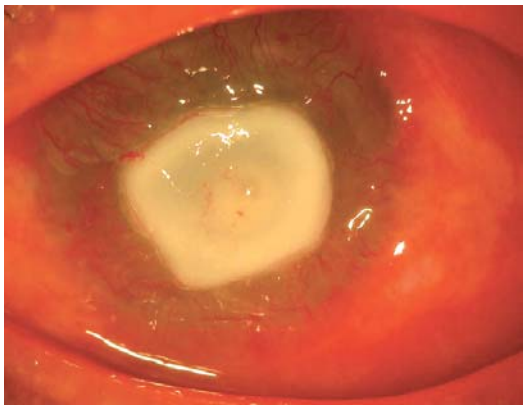


FIGURE 3.18.3: Acanthameba keratitis—stromal necrosis

Sequela of Healed Corneal Ulcer

- Any corneal ulcer heals by the formation of opacity like:
  - *nebula* **(Fig 3.19.1)**
  - *macula* **(Figs 3.19.2 and 3.19.3)**
  - *nebulomacula* **(Fig 3.19.4)**
  - *leukoma* **(Figs 3.19.5 and 3.19.6)**
  - *adherent leukoma* **(Fig 3.19.7)**
  - with or without vascularization **(Figs 3.19.8 and 3.19.9)** and degenerative changes **(Fig 3.19.10)**
- Ectatic cicatrix **(Fig 3.19.11)**
- *Anterior staphyloma*: partial or total **(Figs 3.19.12 and 3.19.13)**
- Phthisis bulbi **(Fig 3.19.14)**

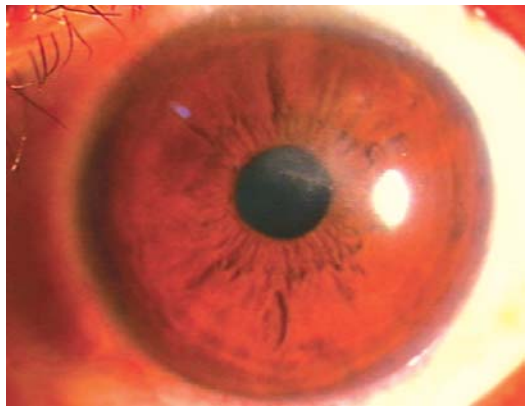


FIGURE 3.19.1: Nebular corneal opacity

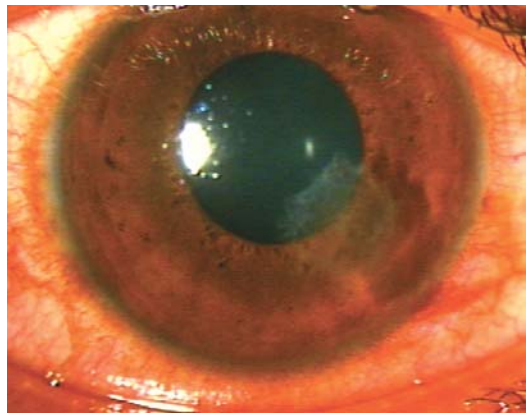


FIGURE 3.19.2: Macular corneal opacity

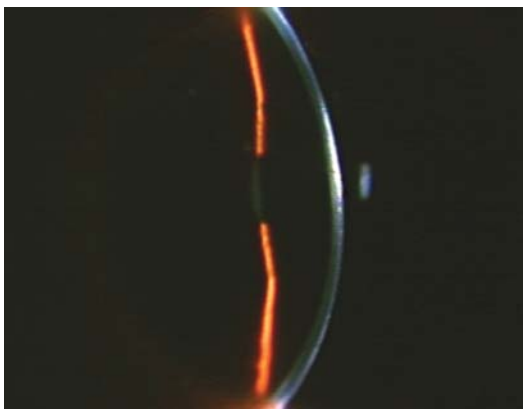


FIGURE 3.19.3: Macular corneal opacity

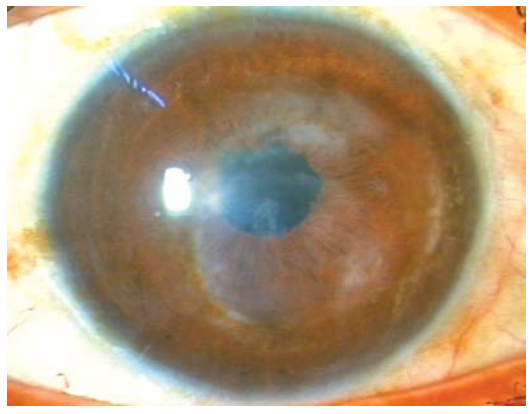


FIGURE 3.19.4: Nebulomacular opacity

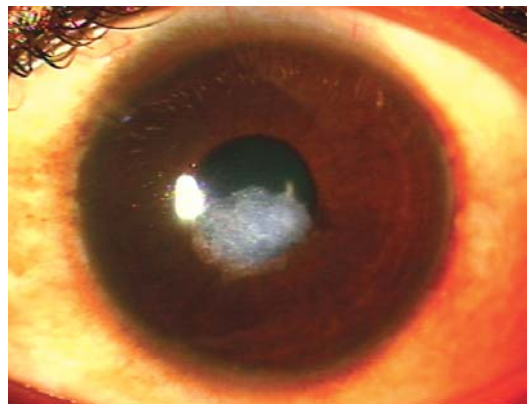


FIGURE 3.19.5: Corneal leukoma

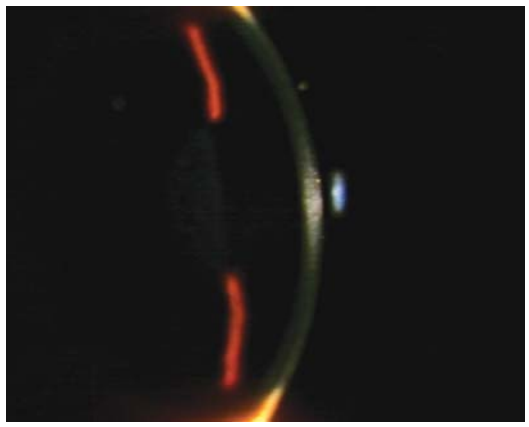


FIGURE 3.19.6: Corneal leukoma

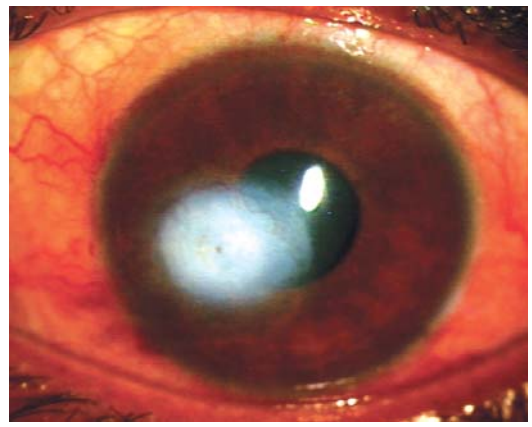


FIGURE 3.19.7: Adherent leukoma

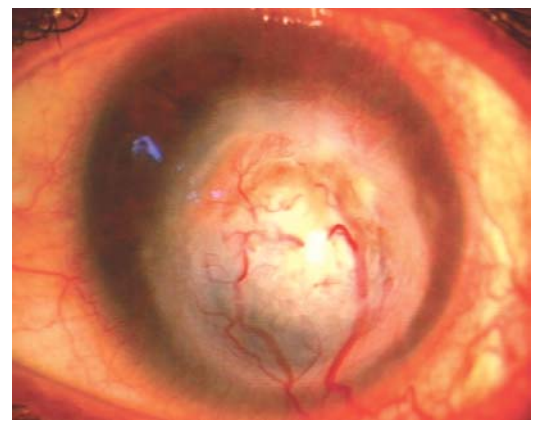


FIGURE 3.19.8: Vascularized adherent leukoma

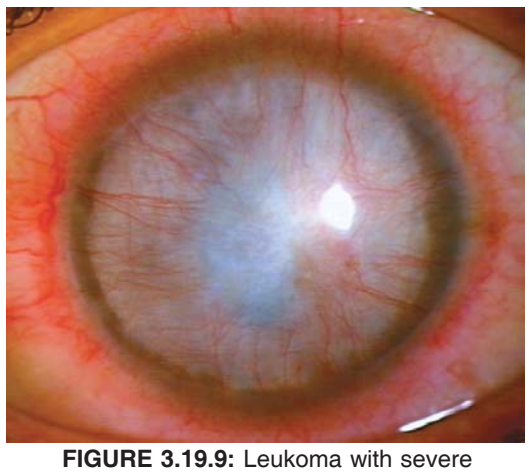


FIGURE 3.19.9: Leukoma with severe vascularization

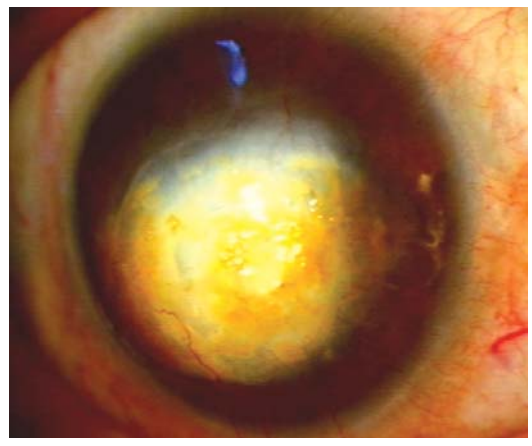


FIGURE 3.19.10: Leukoma with degenerative changes

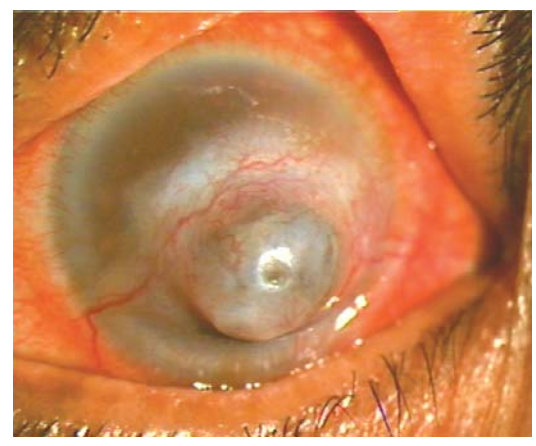


FIGURE 3.19.11: Healing—ectatic cicatrix

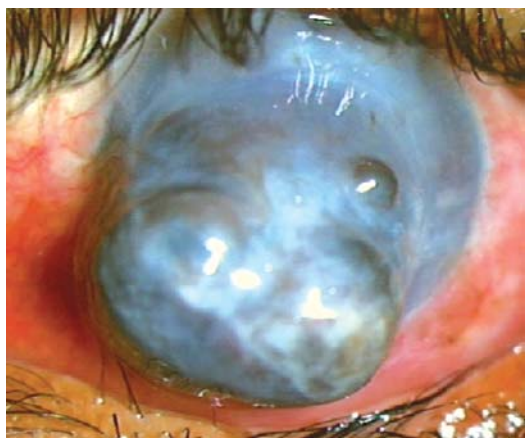


FIGURE 3.19.12: Anterior staphyloma—partial

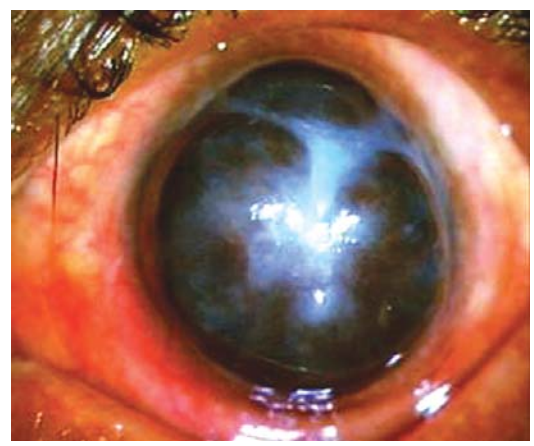


FIGURE 3.19.13: Anterior staphyloma—total

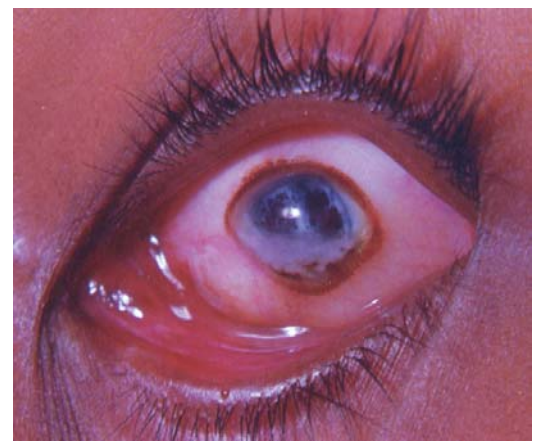


FIGURE 3.19.14: Phthisis bulbi

VIRAL KERATITIS

Herpes Simplex Viral (HSV) Keratitis

- Infection with herpes simplex virus (HSV) is extremely common
- The majority of the patients present with recurrent lesion

Primary Infection

- Usually subclinical or may present with mild ocular problem
- Typically, it occurs between 6 months to 5 years of age
- The main lesion is acute follicular conjunctivitis
- Fine epithelial keratitis may be present, which sometimes progresses into dendritic figure **(Fig 3.20.1)**
- Vesicular eruptions and edema of the lids
- It seldom causes serious ocular problem
- *Treatment:* acyclovir eye ointment—5 times daily for 2-3 weeks

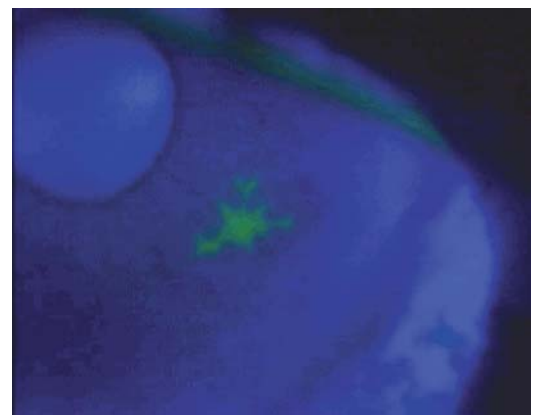


FIGURE 3.20.1: Primary HSV dendrite

Recurrent HSV Keratitis

- The virus travels down along the sensory division of the fifth cranial nerve to affect the target tissues
- Associated with some predisposing factors

Dendritic Keratitis

- Initially starts as superficial punctuate erosion which coalesce
- They send out lateral branches with knobbed ends, to form *'dendritic'* or *'tree-like'* figure and this is *pathognomonic* **(Fig 3.21.1)**
- These are single or multiple branching ulcerated epithelial lesions with raised edges and terminal knob-like projection **(Figs 3.21.2 and 3.21.3)**
- Corneal sensation is diminished
- Bed of the ulcer stains with fluorescein **(Fig 3.21.4)**
- The swollen diseased cells at the margin take up Rose Bengal stain **(Fig 3.21.5)**
- *Treatment:* debridement, topical antiviral agent (acyclovir), mild cycloplegic, oral acyclovir to prevent recurrence

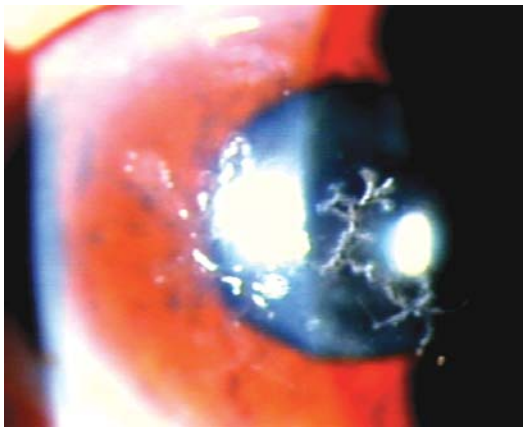


FIGURE 3.21.1: HSV—dendritic keratitis

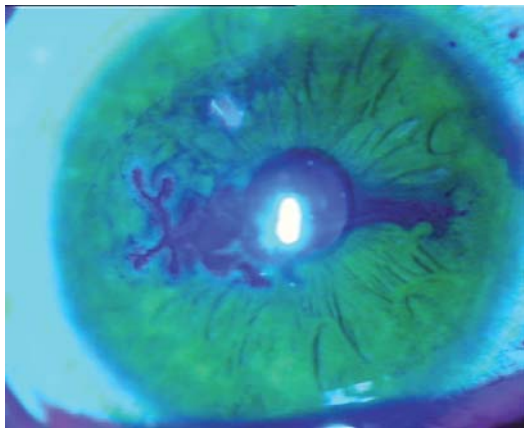


FIGURE 3.21.2: Dendritic keratitis—single

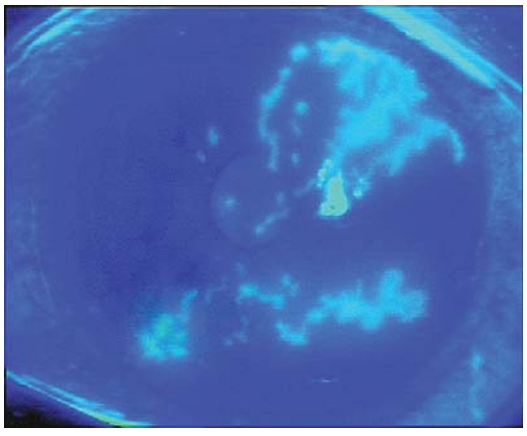


FIGURE 3.21.3: Dendritic keratitis—multiple

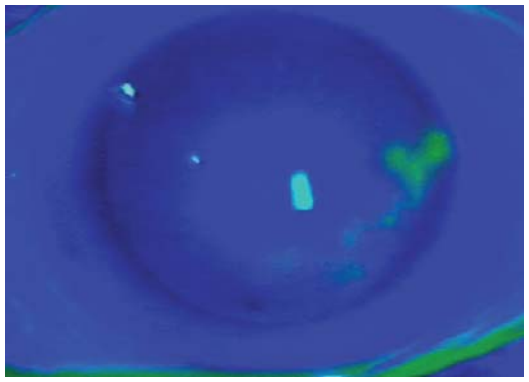


FIGURE 3.21.4: Dendritic keratitis—fluorescein stain

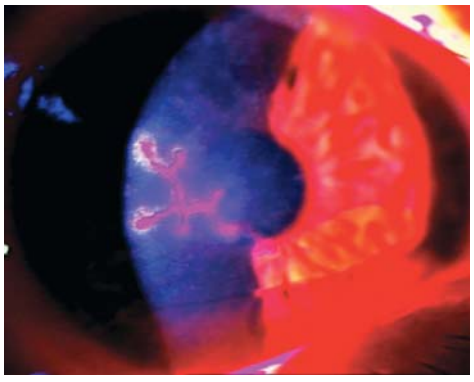


FIGURE 3.21.5: Dendritic keratitis—Rose Bengal stain

Geographical or Ameboid Keratitis (Ulcer)

- Larger epithelial lesion
- Typical *'geographical'* **(Fig 3.22.1)** or *'ameboid'* **(Fig 3.22.2)** configuration
- May occur as a continued enlargement of dendritic keratitis
- Likely to occur following inadvertent use of topical steroids
- *Treatment:* similar to dendritic keratitis

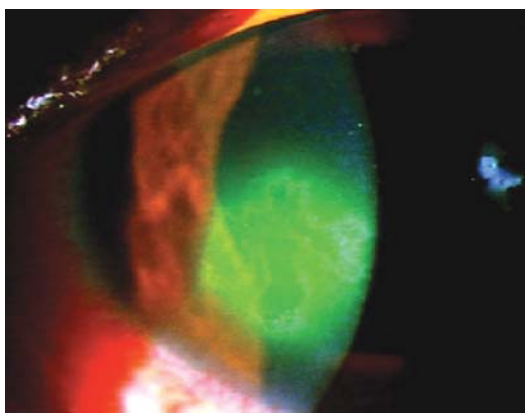


FIGURE 3.22.1: HSV—geographical keratitis

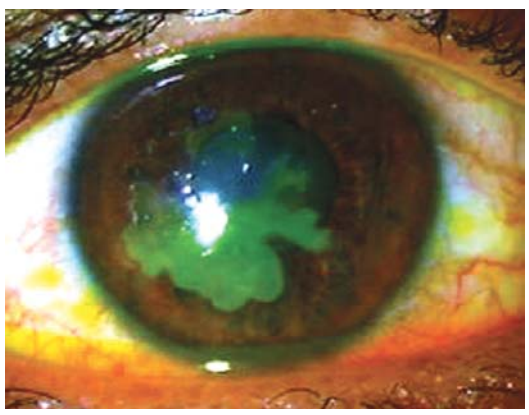


FIGURE 3.22.2: HSV—ameboid keratitis

Stromal Necrotic Keratitis

- Caused by active viral invasion and destruction in immunocompromised individuals
- Cheesy and necrotic appearance of the stroma **(Fig 3.23.1)**
- May be associated with epithelial breakdown and anterior uveitis
- Vascularization, scarring and even perforation may occur at this stage **(Fig 3.23.2)**
- *Treatment:* oral acyclovir, topical antiviral, cycloplegic and judicious use of topical steroids

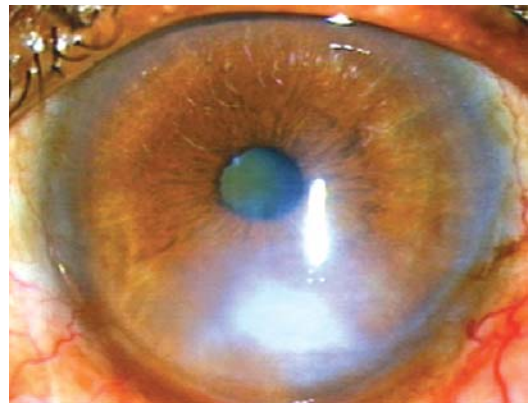


FIGURE 3.23.1: HSV—stromal necrotic keratitis

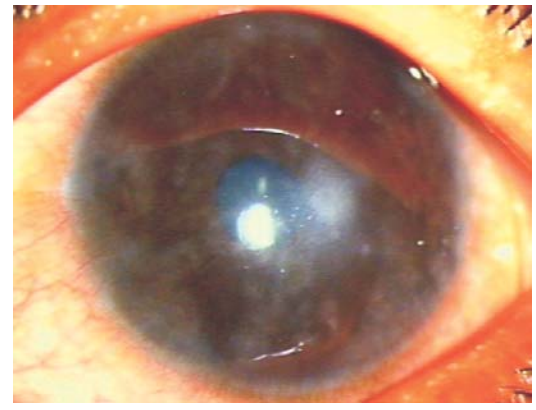


FIGURE 3.23.2: HSV—stromal necrotic keratitis

Metaherpetic Keratitis (Trophic Ulcer)

- Due to persistent defects in the basement membrane
- Margin is gray and thickened due to heaped-up epithelium **(Figs 3.24.1 and 3.24.2)**
- Not an active viral disease
- *Treatment:* artificial tears and bandage contact lens (BCL)

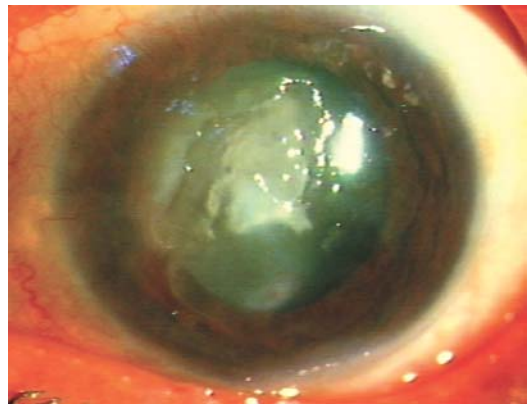


FIGURE 3.24.1: HSV—metaherpetic keratitis

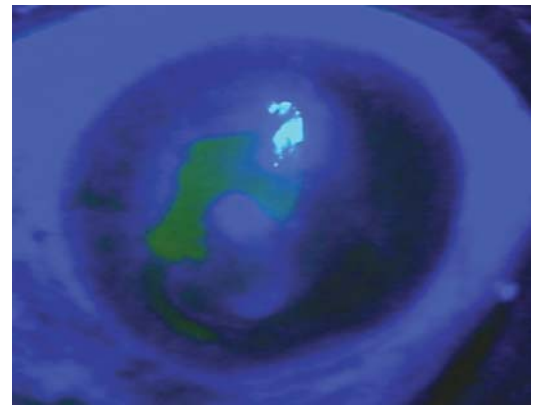


FIGURE 3.24.2: HSV—metaherpetic keratitis

Disciform Keratitis

- Deep keratitis with disk-like edema, an immunogenic reaction to HSV or less commonly with varicella-zoster antigen
- *In milder form,* focal central stromal edema with fine KPs **(Figs 3.25.1 and 3.25.2)**
- *In severe form,* round or oval diffuse areas of stromal and epithelial edema **(Figs 3.25.3 and 3.25.4)**
- Small KPs localized to involved endothelium
- Presence of Descemet's folds and increased central corneal thickness
- Focal bullous keratopathy in some cases
- *Wessely's immune ring* surrounding the edema in long-standing cases **(Fig 3.25.5)**
- *Treatment:* topical steroids (full strength or diluted) with acyclovir eye ointment in equal frequency and cycloplegic

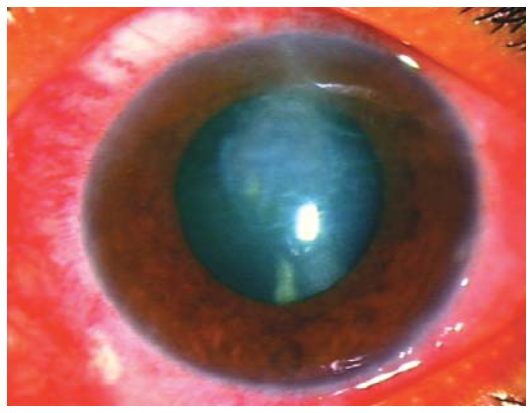


FIGURE 3.25.1: HSV—disciform keratitis

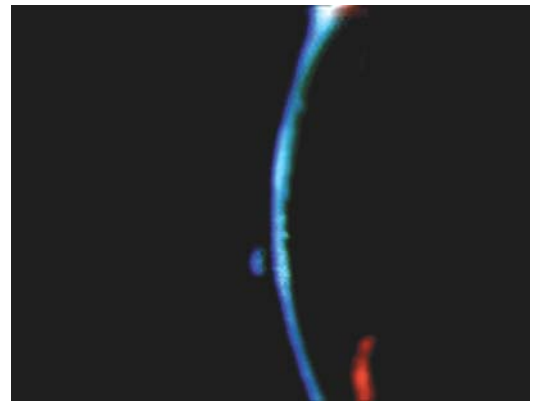


FIGURE 3.25.2: Disciform keratitis—Descemet's folds

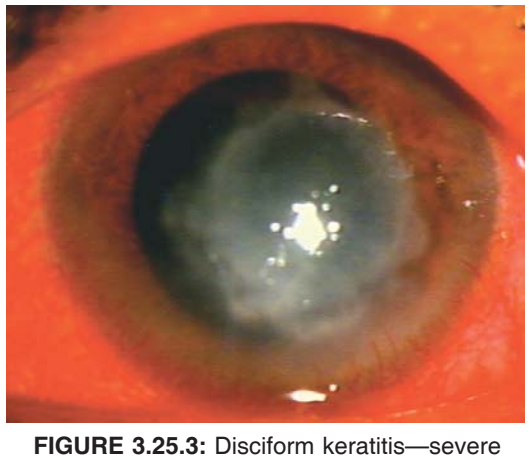


FIGURE 3.25.3: Disciform keratitis—severe

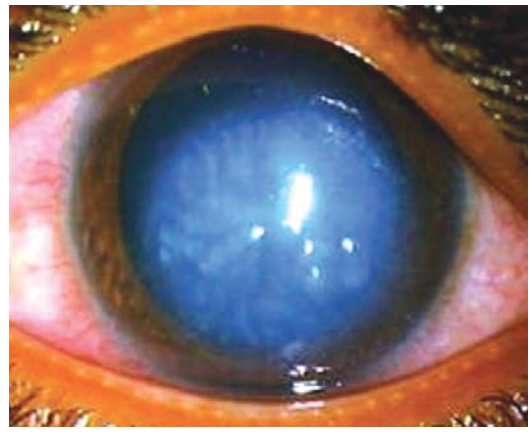


FIGURE 3.25.4: Disciform keratitis—bullous changes

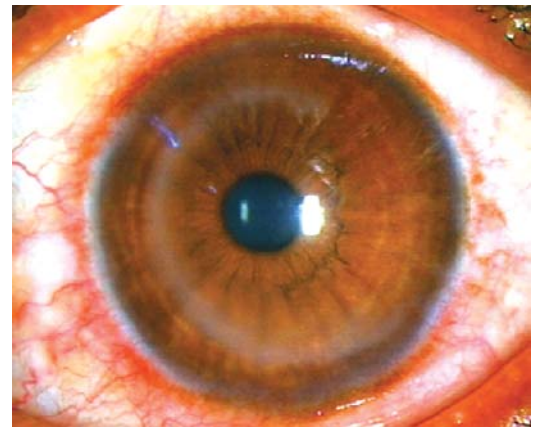


FIGURE 3.25.5: Wessely's immune ring

Herpes Zoster Ophthalmicus (HZO)

- Caused by varicella-zoster virus affecting the elderly people
- More common in immunocompromised hosts
- Vesicular eruptions around the eye, forehead and scalp
- Severe pain along the ophthalmic division of fifth cranial nerve
- *Hutchinson's rule:* when the tip of the nose is involved, the eye will also be involved, since both are supplied by the nasociliary nerve **(Fig 3.26.1)**
- Ocular lesions may be *acute, chronic or recurrent*
- *Acute ocular lesions*
  - *Lids:* redness, edema and vesicular eruptions
  - *Cornea:*
  - punctuate epithelial keratitis
  - *micro-dendrites:* small, fine, multiple dendritic or stellate lesions **(Fig 3.26.2)**
  - *nummular keratitis:* multiple granular lesions surrounded by a halo of stromal haze **(Figs 3.26.3 and 3.26.4)**
  - sensation may be diminished
  - disciform keratitis **(Fig 3.26.5)**
  - *Iris:* Acute iridocyclitis with hyphema and patches of iris atrophy **(Fig 3.26.6)**
  - *Neuro-ophthalmological:* Optic neuritis and cranial nerve palsies—affecting the 3rd (commonest), 4th and 6th nerves **(Fig 3.26.7)**
- *Chronic ocular lesion:*
  - ptosis due to scarring of the lid
  - trichiasis, entropion and lid notching
  - scleritis, nummular keratitis, ocular surface instability, etc.
- *Recurrent lesion:* mucus plaque keratitis, neuroparalytic keratitis or secondary glaucoma
- *Treatment:* oral acyclovir (800 mg 5 times daily for 7 days), topical steroids in presence of keratitis or iridocyclitis, systemic corticosteroids in neuro-ophthalmologic problems

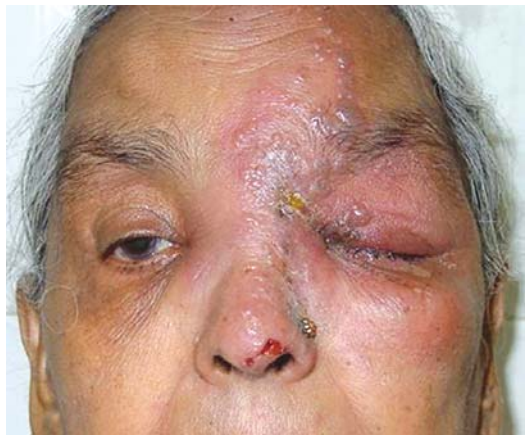


FIGURE 3.26.1: Herpes zoster ophthalmicus

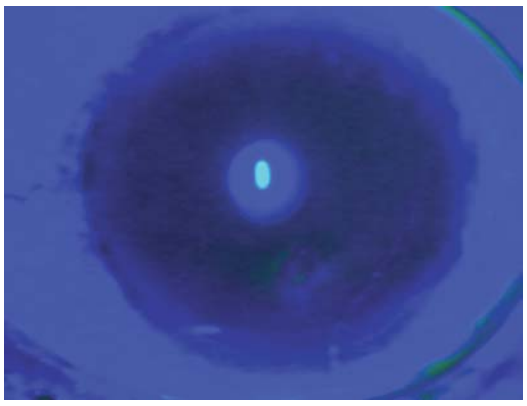


FIGURE 3.26.2: HZO—microdendrites

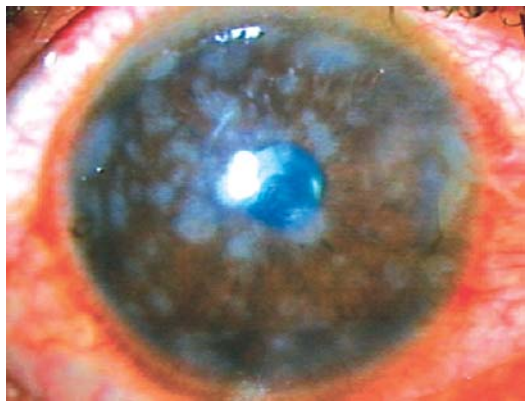


FIGURE 3.26.3: Nummular keratitis

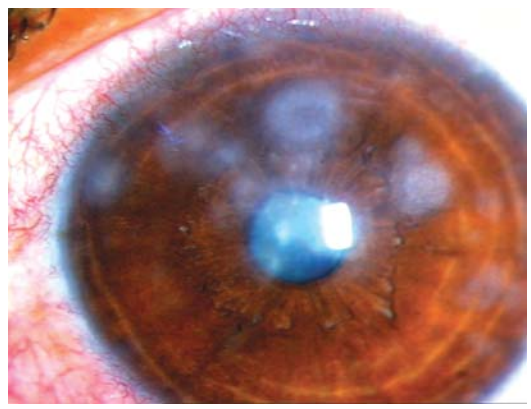


FIGURE 3.26.4: Nummular keratitis

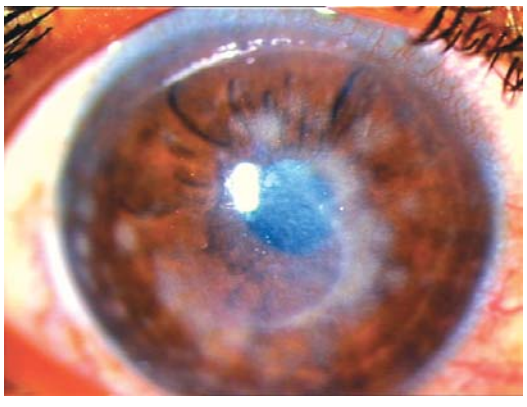


FIGURE 3.26.5: HZO—disciform keratitis

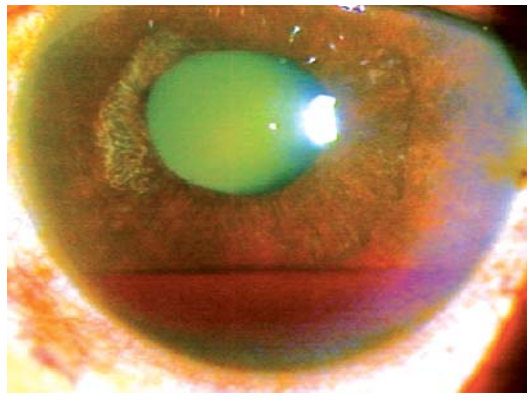


FIGURE 3.26.6: HZO—uveitis with hyphema

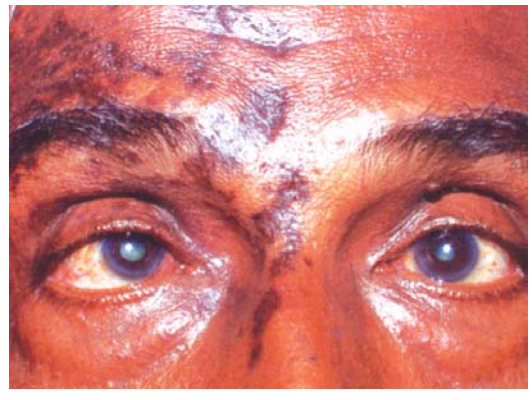


FIGURE 3.26.7: HZO—lateral rectus palsy

OTHER KERATITIS (ULCER)

Lagophthalmic (Exposure) Keratitis

- Due to exposure of cornea
- Owing to dryness and desiccation, the lower third of epithelium is cast off and the raw area is invaded by microorganism
- Seen in facial palsy **(Fig 3.27.1)**, leprosy **(Figs 3.27.2 and 3.27.3)** proptosis and thyroid exophthalmos, comatose patient, or after prolonged anesthesia
- Corneal lesion ranges from minimum epithelial erosions to severe ulceration
- *Treatment:* lid taping, tarsorrhaphy, lid-load operation and treatment of the cause

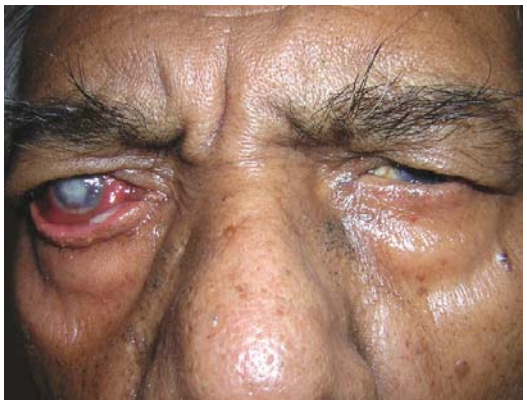


FIGURE 3.27.1: Lagophthalmic keratitis

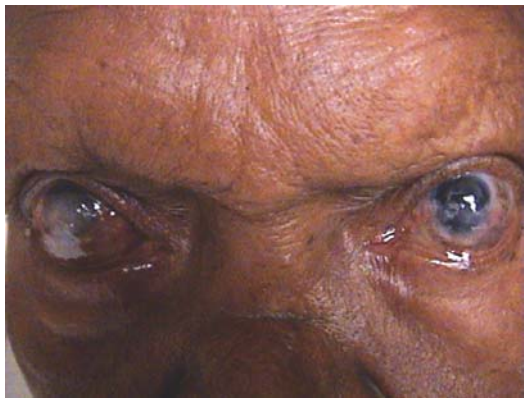


FIGURE 3.27.2: Lagophthalmic keratitis—leprosy

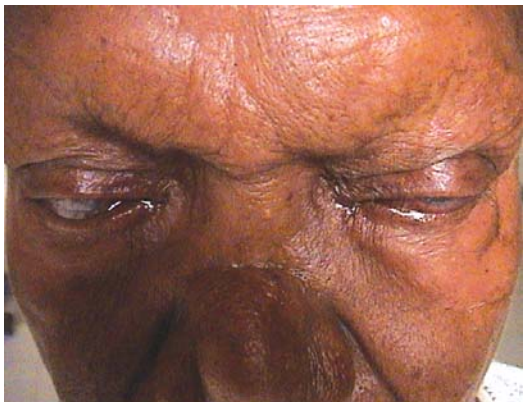


FIGURE 3.27.3: Lagophthalmic keratitis—leprosy

Neurotrophic Keratitis (NTK)

- Occurs in anesthetic cornea which alters the metabolic activity of the epithelium
- Mostly seen after HSV and HZV keratitis and also in diabetes
- Punctate epithelial erosions involving the interpalpebral area
- Edema and exfoliation of the epithelial cells followed by central ulceration **(Fig 3.28.1)**
- *Treatment:* ointment and patching, in severe cases, tarsorrhaphy for several months

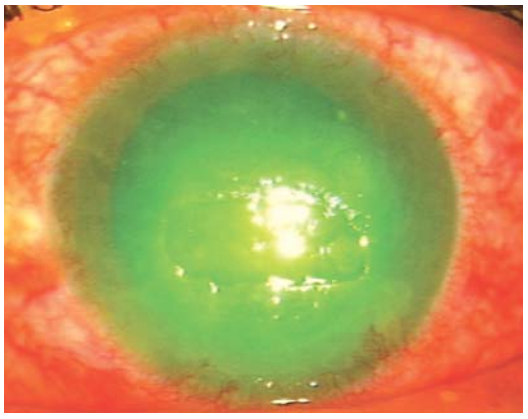


FIGURE 3.28.1: Neurotrophic keratitis

Atheromatous Ulcer

- Develops over an old leukoma with degenerative changes **(Fig 3.29.1)**
- Ulcer progresses rapidly with little tendency to heal
- Easily gets infected and perforation may occur
- *Treatment:* ointment and patching, BCL and tear substitutes

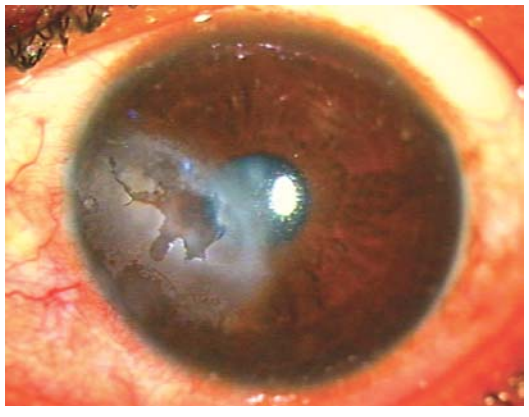


FIGURE 3.29.1: Atheromatous ulcer

Marginal Keratitis

- Caused by hypersensitivity reaction to staphylococcal exotoxin
- Prevalent in patients having chronic staphylococcal blepharitis.
- Subepithelial infiltrates at the periphery, mostly at 4-8 o'clock position, or at 10-12 o'clock position **(Fig 3.30.1)**
- Separated from the limbus by a clear zone of cornea **(Fig 3.30.2)**
- Lesions spread circumferentially and with superficial vascularization **(Fig 3.30.3)**
- *Treatment:* topical corticosteroids, steroid-antibiotic ointment and simultaneous treatment of blepharitis

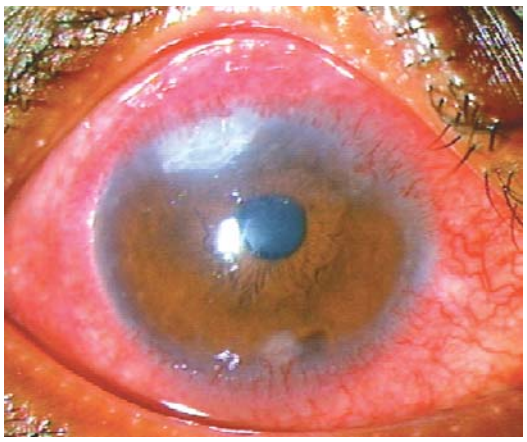


FIGURE 3.30.1: Marginal keratitis

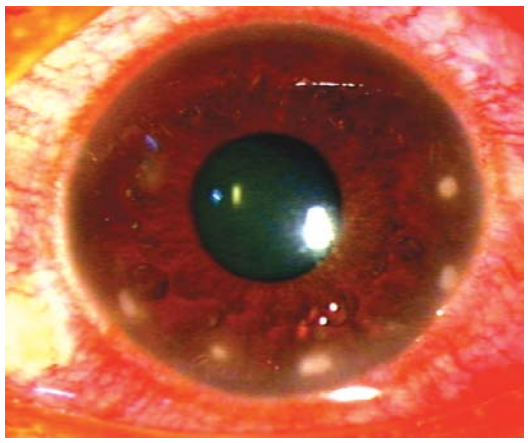


FIGURE 3.30.2: Marginal keratitis

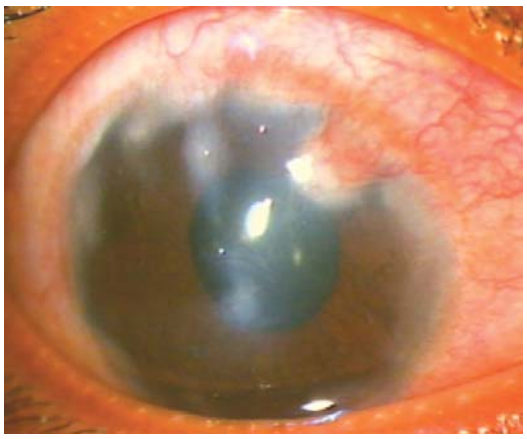


FIGURE 3.30.3: Marginal keratitis—vascularization

Phlyctenular Keratitis

- Predominantly affects the children
- Corneal phlycten is a gray nodule, slightly raised above the surface, and a phlyctenular ulcer is yellowish-white in color **(Fig 3.31.1)**
- May resolve spontaneously or may extend towards the center of cornea
- *Fascicular ulcer:*
  - phlyctenular ulcer slowly migrates from the limbus towards the center of the cornea in a serpiginous way
  - carries leash of blood vessels which lie in a shallow gutter formed by the ulcer **(Fig 3.31.2)**
  - formation of corneal opacity which is densest at it apex
- Multiple phlycten may be associated with phlyctenular pannus and may coalesce to form a ring ulcer
- *Treatment:* topical corticosteroids, cycloplegic and topical antibiotic

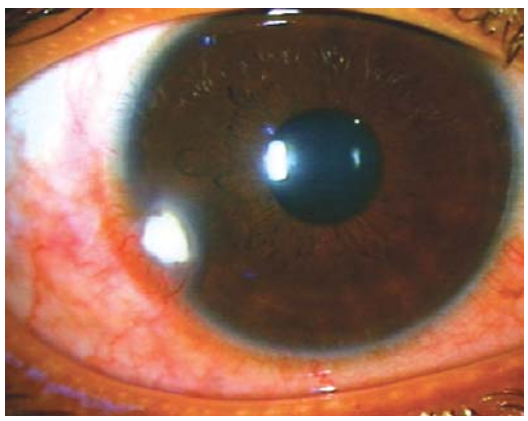


FIGURE 3.31.1: Phlyctenular keratitis

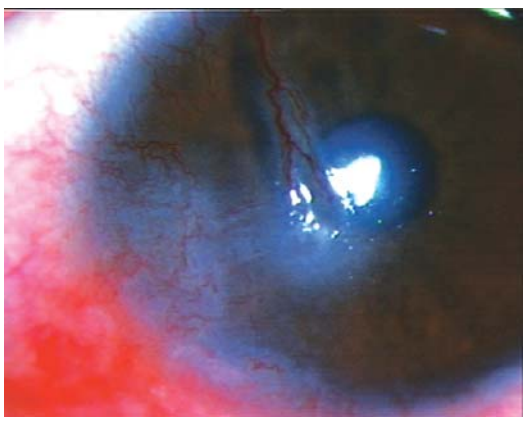


FIGURE 3.31.2: Phlyctenular keratitis—fascicular ulcer

Interstitial Keratitis

- Inflammation of the corneal stroma without primary involvement of epithelium or endothelium
- Rare, bilateral condition with diverse etiology, like, syphilis, tuberculosis, or Cogan syndrome
- Vascularized, mid-stromal, non-suppurative inflammation, giving a 'ground glass' **(Fig 3.32.1)** appearance
- In inactive stage, there is variable stromal scarring with ghost (non-perfused) vessels **(Fig 3.32.2)**

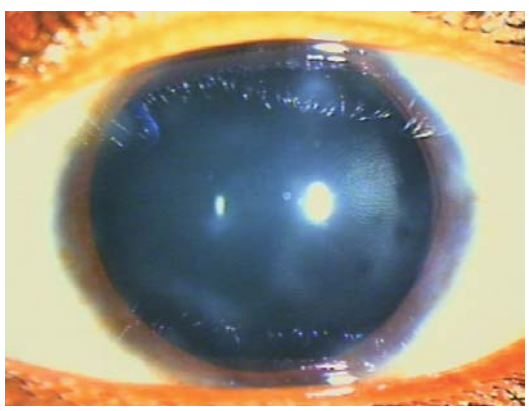


FIGURE 3.32.1: Interstitial keratitis

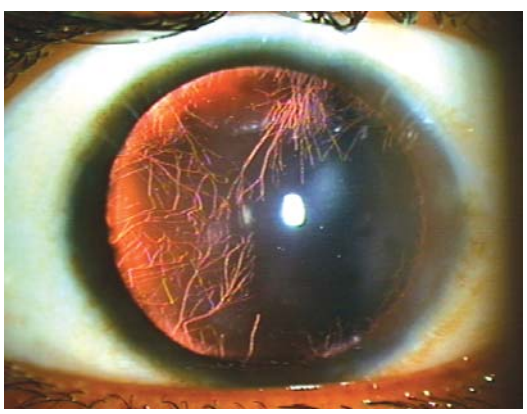


FIGURE 3.32.2: Interstitial keratitis—ghost vessels

- There is always an associated iridocyclitis
- *Hutchinson's triad:* interstitial keratitis, permanent deafness and Hutchinson's teeth—may be seen in patient with congenital syphilis
- *Treatment:* systemic penicillin, topical corticosteroids and cycloplegic

Punctate Epithelial Erosions (PEE)

- Very common nonspecific corneal epithelial lesions seen in variety of corneal diseases
- *Causes:* keratoconjunctivitis sicca **(Fig 3.33.1),** meibomianitis, contact lens wearers, foreign body in subtarsal sulcus, caterpillar hair, keratitis medicomentosa **(Fig 3.33.2)**, photokeratitis, etc.
- Tiny, grayish-white, slightly depressed dots scattered in different fashion on the cornea
- represent the area of epithelial discontinuity
- stain with fluorescein but not with Rose Bengal **(Fig 3.33.3)**
- *Treatment:* tear substitutes and others are directed towards the cause

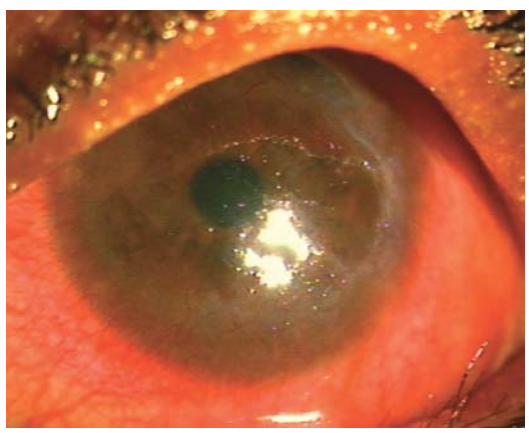


FIGURE 3.33.1: Punctate epithelial erosions—dry eye

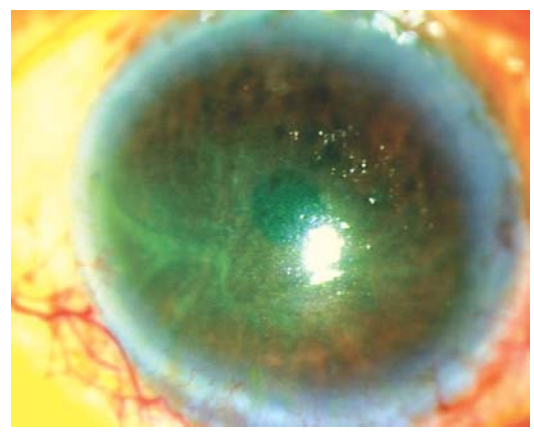


FIGURE 3.33.2: PEE—keratitis medicomentosa

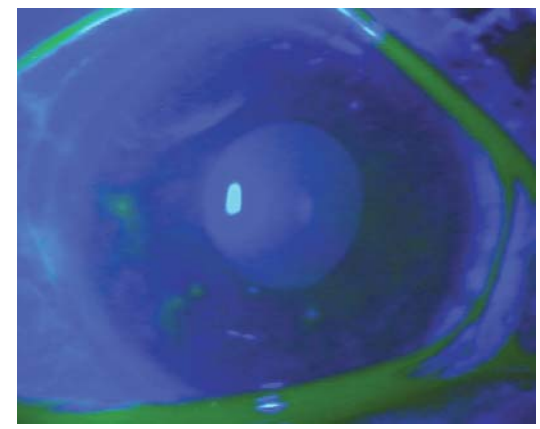


FIGURE 3.33.3: PEE—fluorescein stain

Punctate Epithelial Keratitis (PEK)

- Punctate epithelial lesions scattered all over the cornea
- Usually seen after acute follicular conjunctivitis of viral origin or after HZV infection **(See Figs 2.13.1 and 2.13.3)**
- Epithelial opacities appear as raised gray dots, scattered all over the cornea
- Sometimes, they extend into the Bowman's membrane and superficial stroma **(Fig 3.34.1)**
- Lesions stain poorly with fluorescein, but turn bright red with Rose Bengal
- *Treatment:* tear substitutes and dilute topical steroids

Superficial Punctate Keratitis of Thygesons

- Uncommon, usually bilateral, idiopathic condition
- Round, oval or stellate conglomerations of grayish-white distinct dots which are intraepithelial **(Fig 3.35.1)**
- May be associated with mild subepithelial haze
- Conjunctiva is not involved
- *Treatment:* tears substitutes

Superior Limbic Keratoconjunctivitis (SLK)

- Papillary hypertrophy of the superior tarsal conjunctiva
- Edema and thickening of the conjunctiva at the superior limbus **(Fig 3.36.1)**
- Superior cornea shows punctate epithelial erosion and filaments which stain with Rose Bengal
- *See Chapter: 2* ***(See Figs 2.43.1 and 2.43.2)***

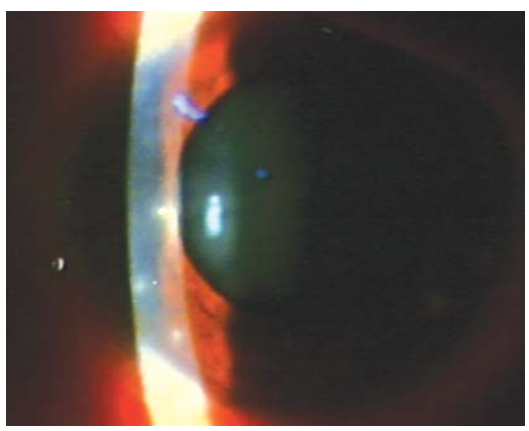


FIGURE 3.34.1: Punctate epithelial keratitis

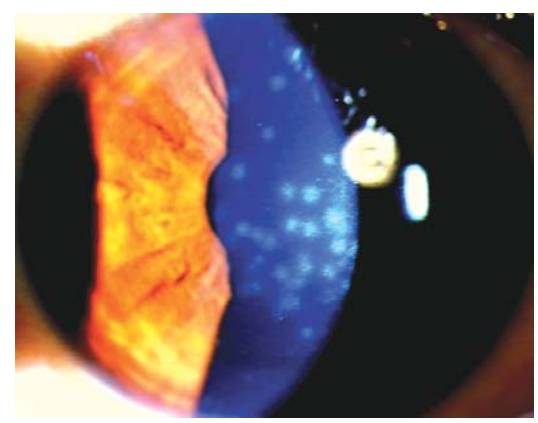


FIGURE 3.35.1: Superficial punctate keratitis of Thygesons

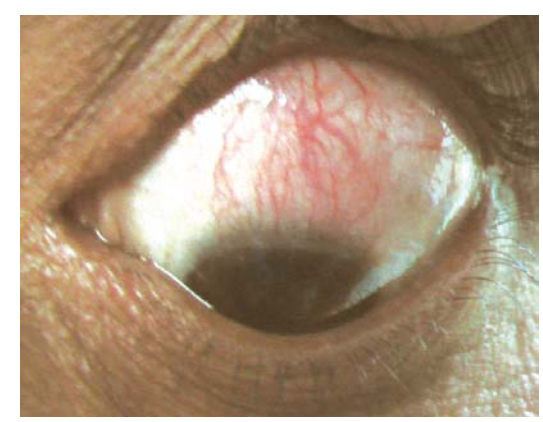


FIGURE 3.36.1: Superior limbic keratoconjunctivitis

Shield Ulcer in Vernal Keratoconjunctivitis

- Occurs occasionally in patients with severe vernal catarrh
- Superior oval elevated lesion with grayish opacification of the bed **(Fig 3.37.1)**
- Better to be designated as plaque **(Fig 3.37.2)**
- Other signs of vernal conjunctivitis
- *Treatment:* scraping or dissection of the plaque followed by BCL, or amniotic membrane graft and supratarsal injection of triamcinolone

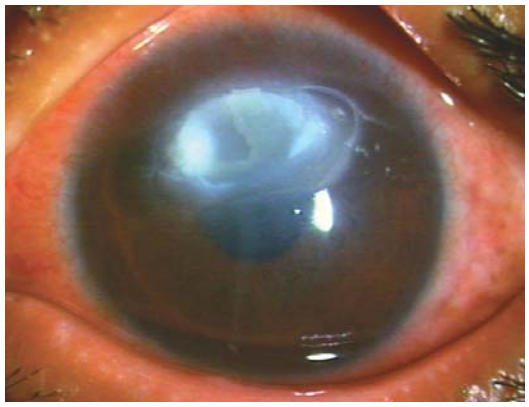


FIGURE 3.37.1: Shield ulcer—vernal keratoconjunctivitis

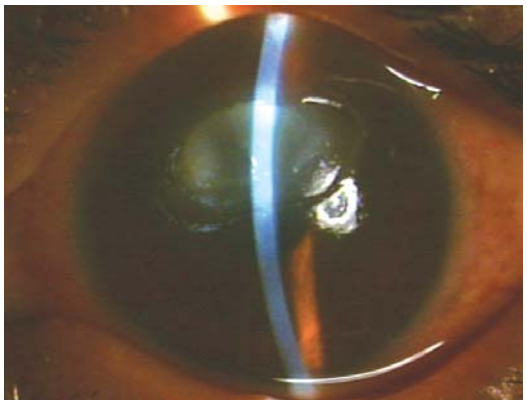


FIGURE 3.37.2: Shield ulcer—palque

Sclerosing Keratitis

- Rare chronic condition may occur in isolation or with scleritis
- May be associated with collagen diseases
- Gradual peripheral stromal thickening and opacification **(Fig 3.38.1)**
- Vascularization and lipid deposition may occur in late stage **(Fig 3.38.2)**
- *Treatment:* systemic and topical steroids and investigations

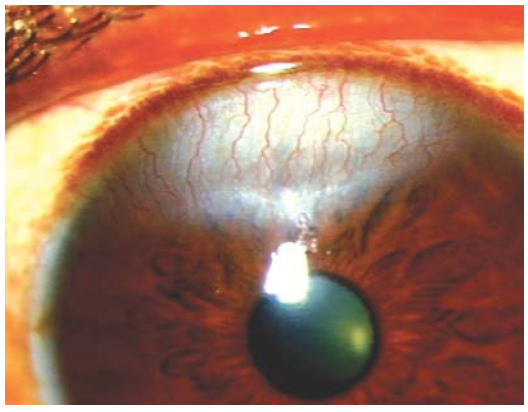


FIGURE 3.38.1: Sclerosing keratitis

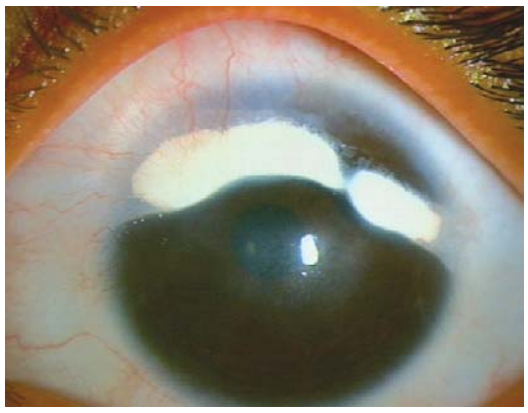


FIGURE 3.38.2: Sclerosing keratitis

Acute Stromal Sclerokeratitis

- Acute stromal keratitis is usually associated with non-necrotizing scleritis **(Fig 3.39.1)**
- Peripheral, mid- or anterior stromal infiltration adjacent to the scleral inflamed area **(Fig 3.39.2)**
- May be associated with breakdown of epithelium
- *Treatment:* systemic and topical steroids, systemic antimetabolites in selective cases

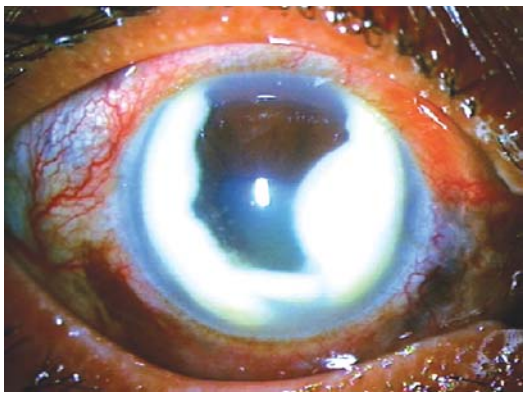


FIGURE 3.39.1: Acute stromal sclerokeratitis—severe

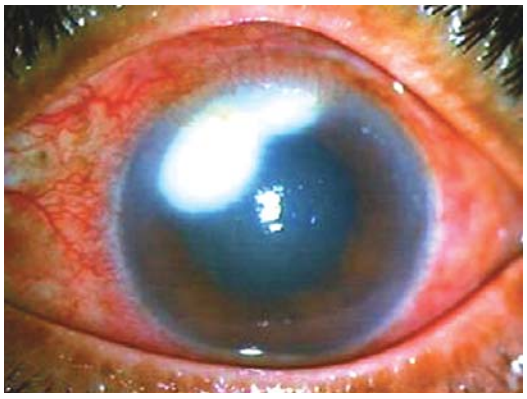


FIGURE 3.39.2: Acute stromal sclerokeratitis

PERIPHERAL THINNING AND DEGENERATIONS

This is a group of disorders, characterized by degenerative thinning and/or melting of the corneal periphery

Mooren's Ulcer

- Chronic progressive peripheral ulcer of unknown etiology
- Due to an ischemic necrosis resulting from vasculitis of the perilimbal vessels
- Two types: *limited form*—unilateral and affects elderly people, and *progressive form*—bilateral, relentlessly progressive and affects younger people
- Starts at the interpalpebral area, then spread slowly undermining the epithelium and superficial stroma
- Advancing border of the ulcer is having an overhanging edge **(Fig 3.40.1)**
- Later it involves the entire circumference of the cornea, and also spreads towards the center **(Fig 3.40.2)**
- Healing takes place from the periphery, and the healed area becomes vascularized, thinned and opaque **(Fig 3.40.3)**
- Perforation may occur with minor trauma
- *Treatment:* topical corticosteroids, cycloplegic, peritomy, glue and BCL, systemic immunosuppressant, lamellar sclerocorneal graft in extreme cases

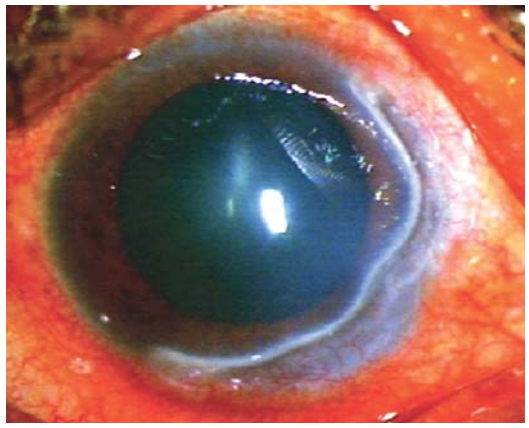


FIGURE 3.40.1: Mooren's ulcer

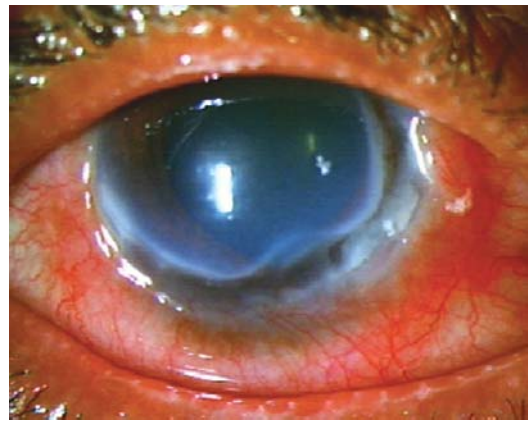


FIGURE 3.40.2: Mooren's ulcer—overhanging edge

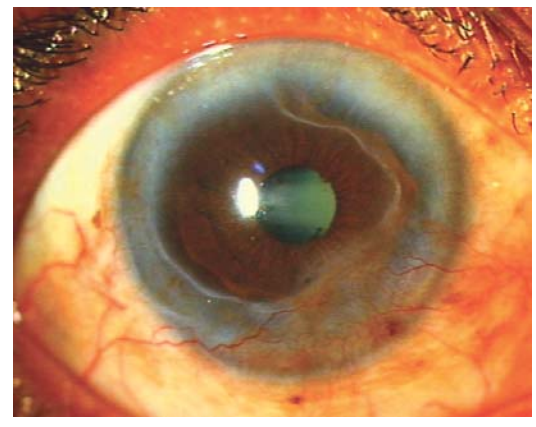


FIGURE 3.40.3: Mooren's ulcer—healing

Terrien's Marginal Degeneration

- Rare, bilateral slowly progressive marginal degeneration mostly affecting the adult males
- Lesion starts as fine yellow-white punctate stromal opacities at the upper part of the cornea. Sharp edge towards the center becomes demarcated by yellow-white lipid deposits **(Fig 3.41.1)**
- In some cases, it may be in lower peripheral cornea **(Fig 3.41.2)**
- Eventually, thinning to form a peripheral gutter **(Figs 3.41.3 and 3.41.4)**
- Overlying epithelium is intact and vascularization is prominent
- Vision usually reduce due to high corneal astigmatism
- Perforation may occur in 15 percent of the patients
- *Treatment:* RGP contact lens may be helpful, deep lamellar sectorial keratoplasty in severe cases or peripheral PK in perforation

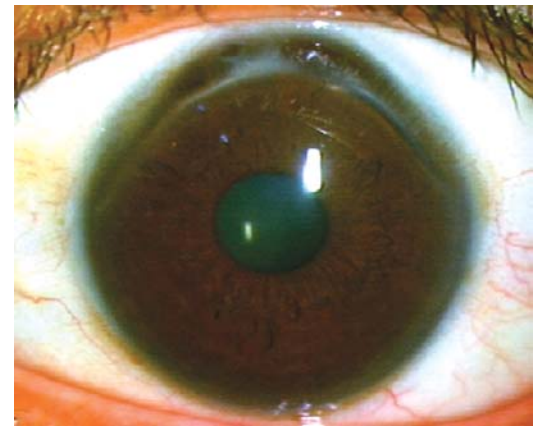


FIGURE 3.41.1: Terrien's marginal degeneration—superior

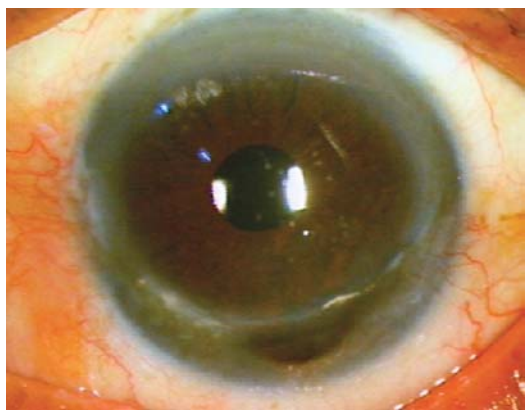


FIGURE 3.41.2: Terrien's degeneration—inferior

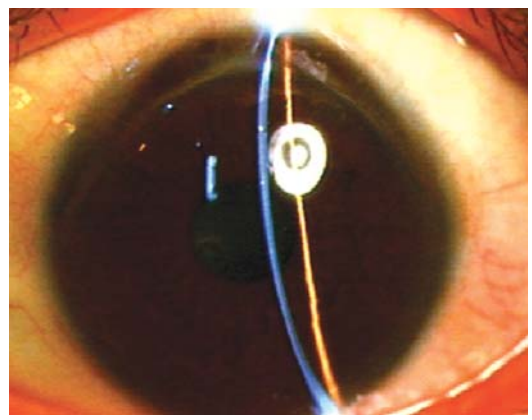


FIGURE 3.41.3: Terrien's degeneration—marginal gutter—upper

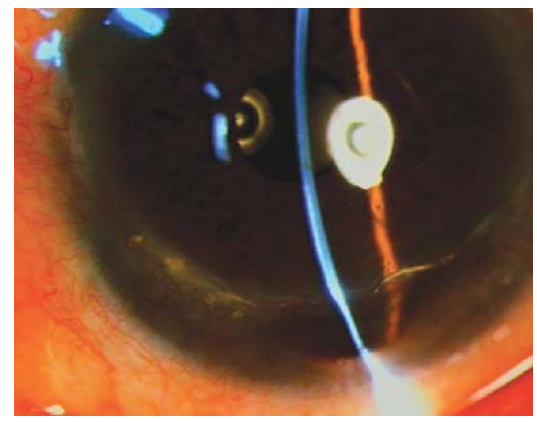


FIGURE 3.41.4: Terrien's degeneration—marginal gutter—lower

Pellucid Marginal Degeneration

- Rare, bilateral, slowly progressive marginal thinning (degeneration) of the cornea between 20-40 years of age
- Thinning involves only the inferior cornea with ectasia just above the area of thinning, giving rise the appearance of keratoconus **(Figs 3.42.1 and 3.42.2)**

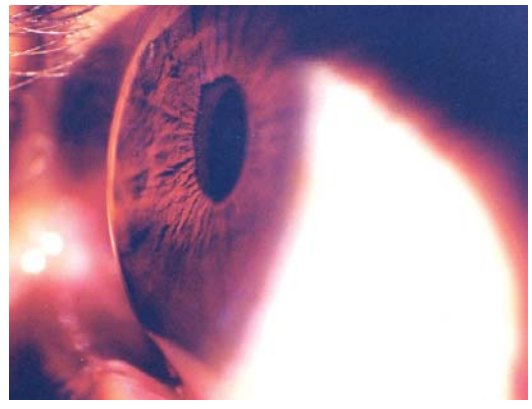


FIGURE 3.42.1: Pellucid marginal degeneration

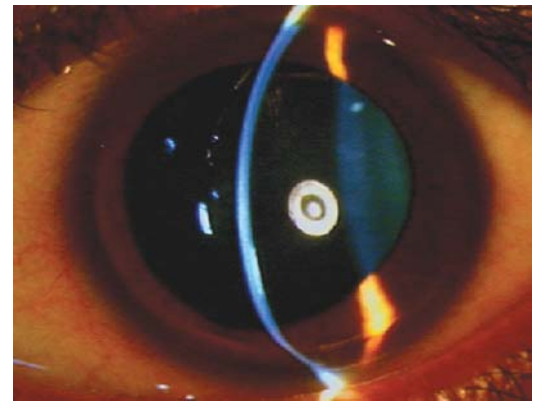


FIGURE 3.42.2: Pellucid marginal degeneration

In keratoconus, thinning involves at the center and the maximum ectasia is within the area of thinning

- Rarely, involves the superior cornea **(Figs 3.42.3 and 3.42.4)**
- Fleischer's ring does not occur
- But, PMD may be complicated by acute hydrops **(Fig 3.42.5)**
- No vascularization (as in Mooren's or Terrien's), or no lipid degeneration (as in Terrien's)
- *Treatment:* correction of astigmatism by RGP contact lens, patch tectonic graft

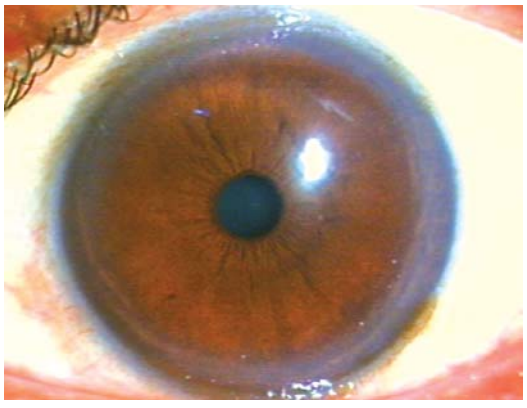


FIGURE 3.42.3: Atypical PMD—superior

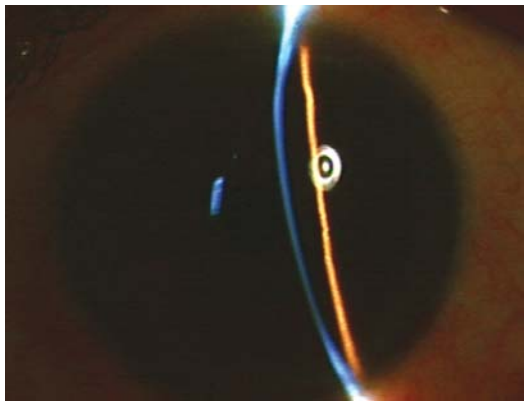


FIGURE 3.42.4: Atypical PMD—superior

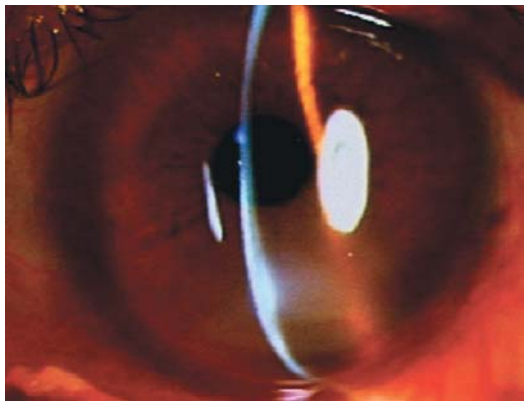


FIGURE 3.42.5: PMD—acute hydrops

Marginal Ulcers Associated with Systemic Collagen Vascular Disorders

- Seen in rheumatoid arthritis, systemic lupus erythematosus, polyarteritis nodosa or Wegener's granulomatosis
- Ulceration and thinning **(Figs 3.43.1 and 3.43.2)**
- Peripheral keratolysis **(Fig 3.43.3)**, sclerosing keratitis **(Fig 3.43.4)**, 'contact lens cornea', secondary KCS, etc.
- Uveitis, AION, etc. are the other ocular manifestations
- *Treatment:* systemic and topical corticosteroids, tear substitutes immunosuppressive agents, peritomy or peripheral keratoplasty

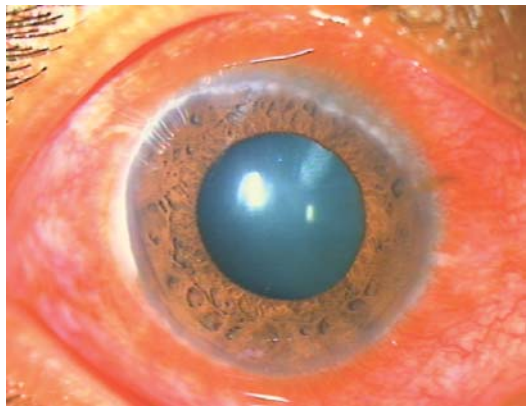


FIGURE 3.43.1: Peripheral keratitis—collagen disorders

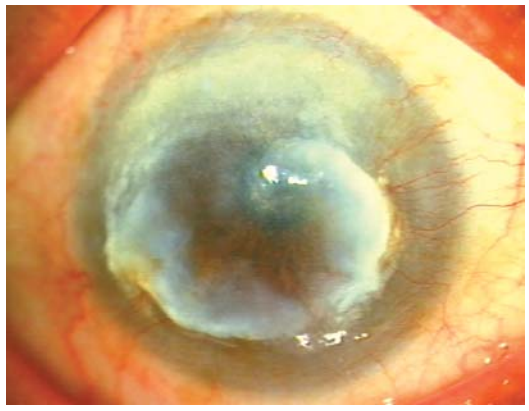


FIGURE 3.43.2: Peripheral keratitis—collagen disorders

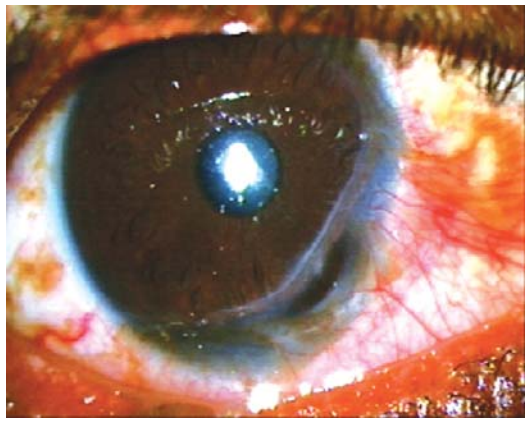


FIGURE 3.43.3: Peripheral thinning in collagen disorders

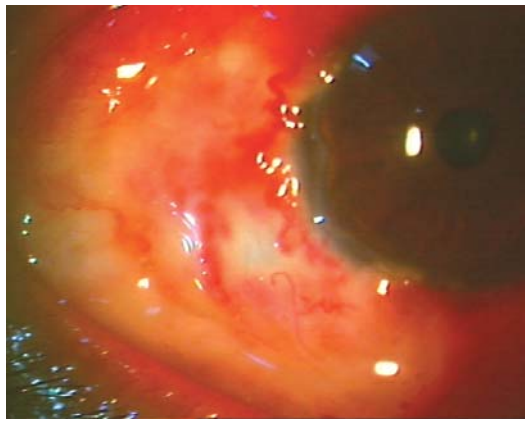


FIGURE 3.43.4: Peripheral ulcerative keratitis with scleritis

CORNEAL DEGENERATIONS

Arcus Senilis (Gerontoxon)

- Bilateral lipid degeneration of the peripheral cornea, affects the elderly persons
- Starts in the superior and inferior perilimbal cornea
- Then progresses circumferentially to form a white band of 1 mm wide **(Figs 3.44.1 and 3.44.2)**
- Peripheral sharp edge is separated from the limbus by a clear zone which may become thin to form senile furrow **(Figs 3.44.3 and 3.44.4)**
- When it occurs below the age of 40 years, it is called *arcus juvenilis* (anterior embryotoxon), which may be associated with systemic hyperlipidemia **(Fig 3.44.5)**
- *Pseudogerontoxon:* seen in vernal conjunctivitis
- *Treatment:* no treatment is required

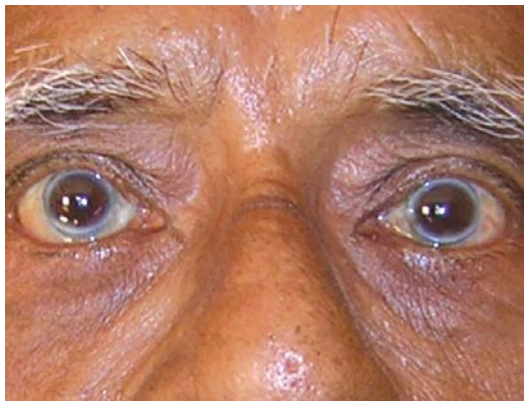


FIGURE 3.44.1: Arcus senilis

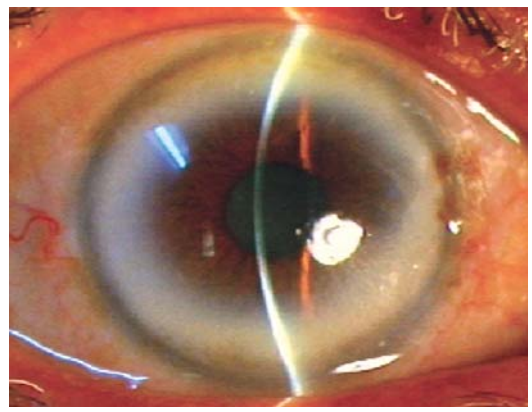


FIGURE 3.44.2: Arcus senilis—severe

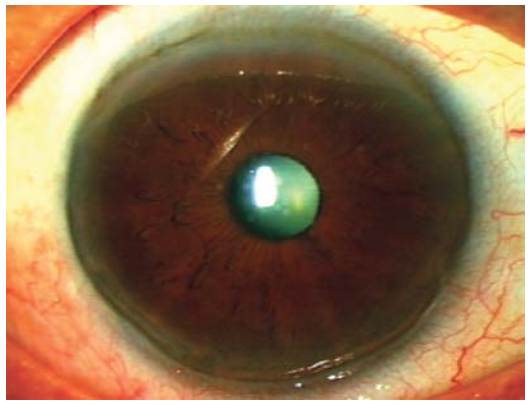


FIGURE 3.44.3: Arcus senilis—furrow degeneration

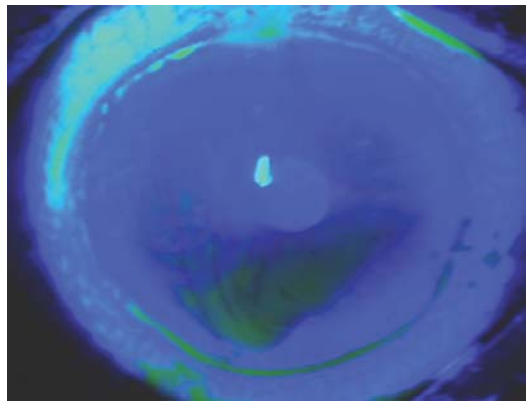


FIGURE 3.44.4: Arcus senilis—furrow degeneration

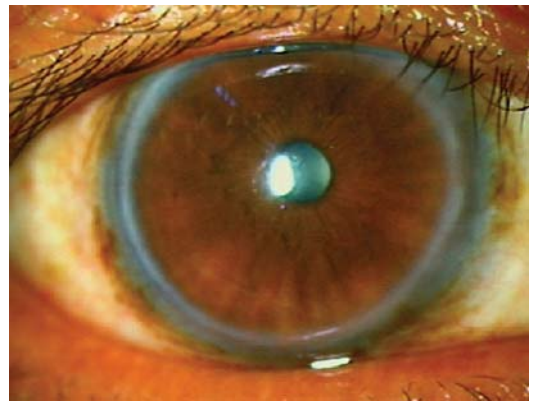


FIGURE 3.44.5: Arcus juvenilis

Band-shaped Keratopathy (BSK)

- Caused by deposition of calcium salts in the subepithelial layer
- Characterized by a horizontal band-shaped opacity
- Calcific band is largely at the palpebral fissure, separated from limbus by a clear zone **(Fig 3.45.1)**
- Band begins in the periphery at 3 and 9 o'clock position, then it affects the central area **(Figs 3.45.2 and 3.45.3)**
- Epithelium eventually becomes irregular and breaks down to form ulcer **(Fig 3.45.4)**
- Spheroidal degeneration may occur in addition **(Fig 3.45.5)**
- *Common causes*: iritis in JRA, chronic iridocyclitis, phthisis bulbi, absolute glaucoma, etc.
- Other *secondary calcific degenerations* occur in long-standing corneal opacity or other disorder **(Figs 3.45.6 and 3.45.7)**
- *Treatment:* scraping of the epithelium, treatment with di-sodium EDTA, and lamellar keratoplasty in severe cases

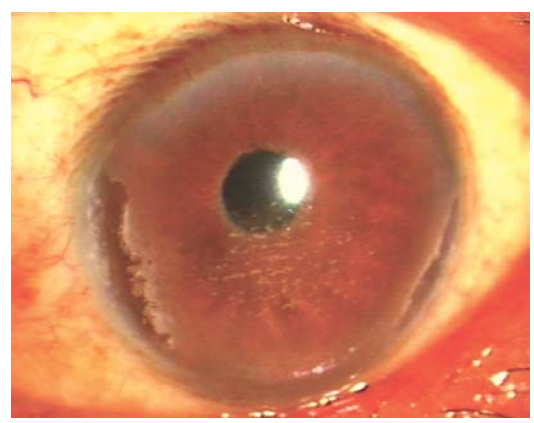


FIGURE 3.45.1: Band-shaped keratopathy

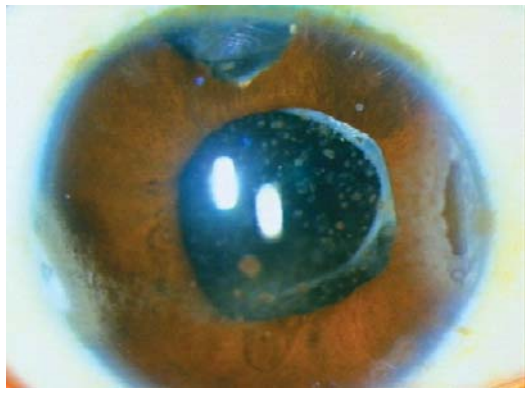


FIGURE 3.45.2: Band-shaped keratopathy

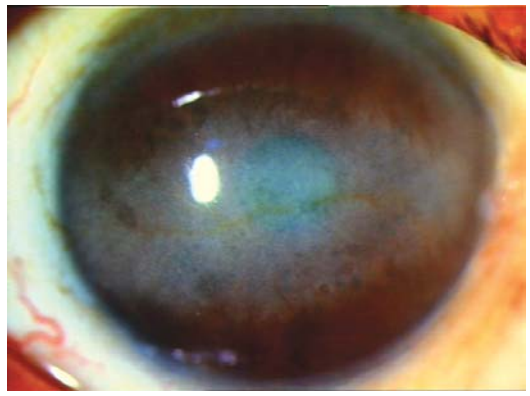


FIGURE 3.45.3: BSK—Hudson-Stahli line

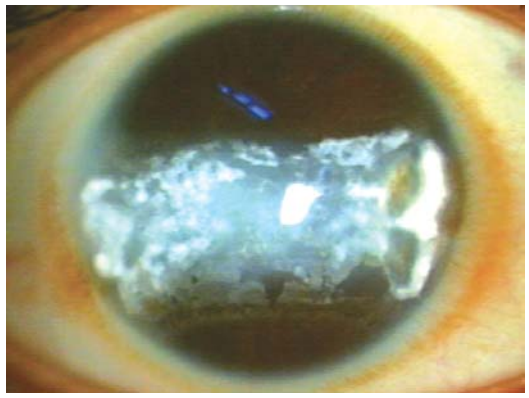


FIGURE 3.45.4: Band-shaped keratopathy

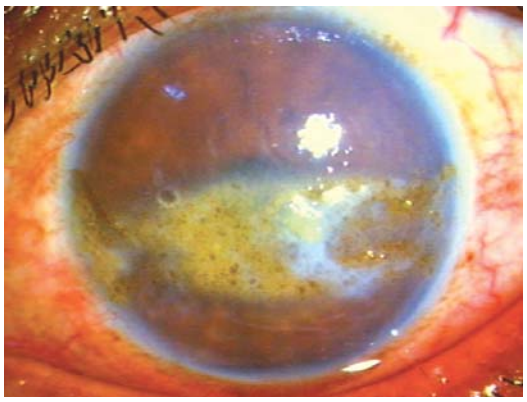


FIGURE 3.45.5: BSK with spheroidal degeneration

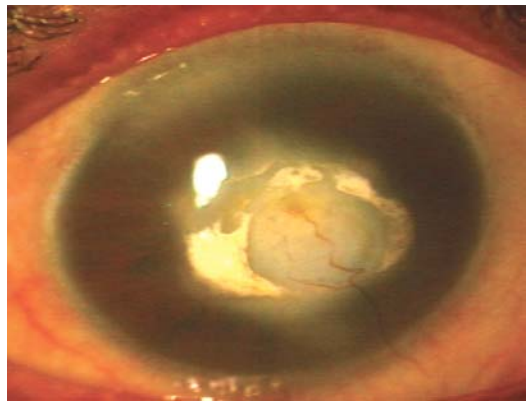


FIGURE 3.45.6: Secondary calcific degeneration

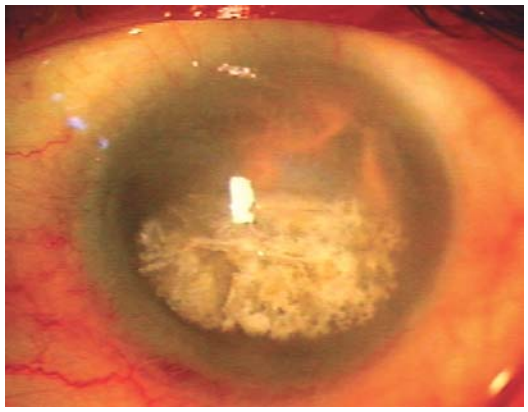


FIGURE 3.45.7: Secondary calcific degeneration

Salzmann's Nodular Degeneration

- Uncommon, and mostly unilateral condition
- Occurs as a late sequel of corneal diseases like phlyctenulosis, trachoma, chronic keratitis or vernal keratoconjunctivitis
- Elevated subepithelial bluish-gray nodules (1-9 in number), in a scarred cornea **(Fig 3.46.1)**
- They arise from the superficial stroma and surrounded by iron-pigment deposits **(Fig 3.46.2)**
- *Treatment:* usually not necessary, lamellar keratoplasty in case of central lesion

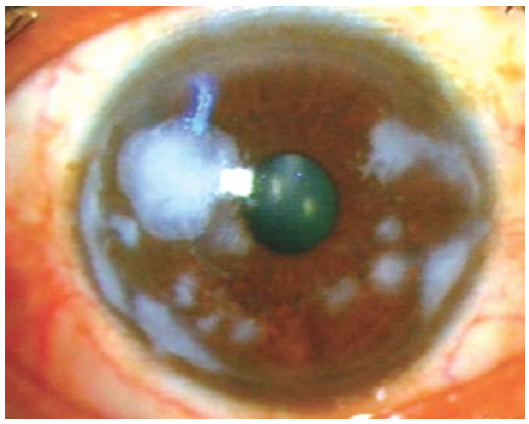


FIGURE 3.46.1: Salzmann's nodular degeneration

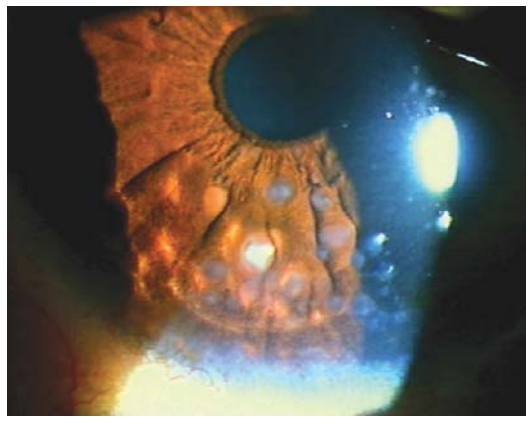


FIGURE 3.46.2: Salzmann's nodular degeneration

Spheroidal Degeneration (climatic droplet keratopathy)

- Rare, bilateral primary condition which typically affects outdoor worker **(Figs 3.47.1 and 3.47.2)**
- May also occur secondary to other corneal disorders, like corneal opacity **(Fig 3.47.3)**, BSK **(See Fig 3.45.5)**, Fuchs' endothelial dystrophy and lattice dystrophy **(See Fig 3.54.9)**
- Initially, there are small amber colored granules in the superficial stroma and conjunctiva, mainly in the interpalpebral area
- Lesions then spread centrally and coalesce to become more dense
- *Treatment:* lamellar or penetrating keratoplasty

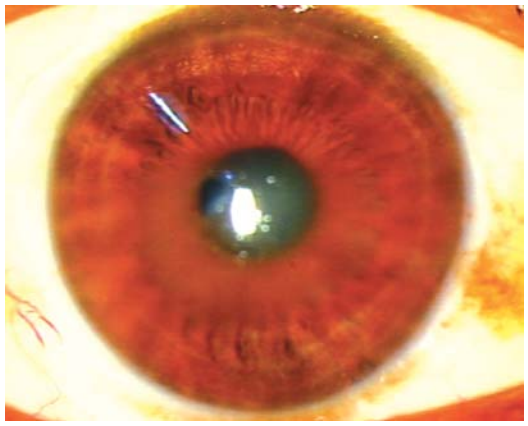


FIGURE 3.47.1: Spheroidal degeneration—primary

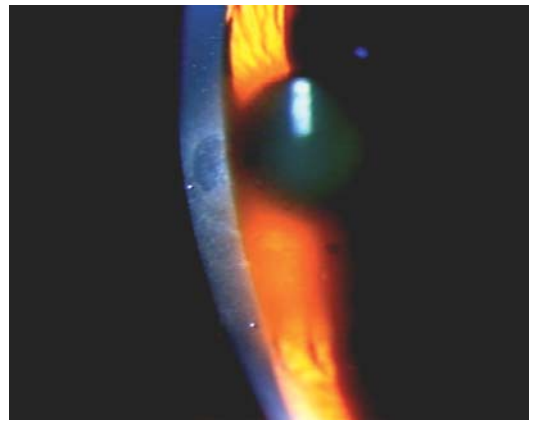


FIGURE 3.47.2: Spheroidal degeneration—primary

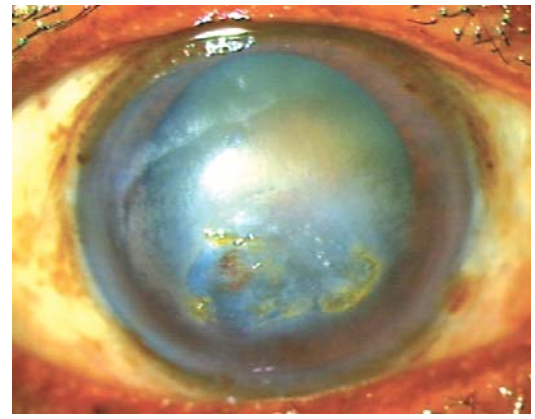


FIGURE 3.47.3: Spheroidal degeneration—corneal opacity

White Limbal Girdle of Vogt

- Very common, bilateral, innocuous age-related condition
- Chalky white, crescentic linear opacities along the nasal and temporal limbus found at the interpalpebral area **(Fig 3.48.1)**
- No treatment is required

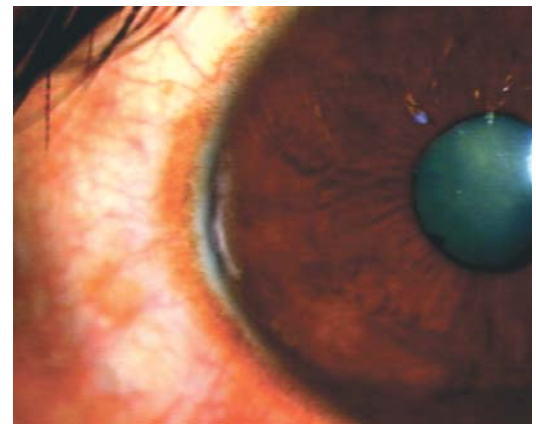


FIGURE 3.48.1: White limbal girdle of Vogt

CORNEAL DYSTROPHIES

ANTERIOR DYSTROPHIES (Epithelium and Bowman's Membrane)

- Probably, the more common dystrophies, but frequently misdiagnosed due to variable presentation
- Most of the patients remain asymptomatic, but the others develop recurrent corneal erosions
- All are autosomal dominant inheritance

Map-dot-fingerprint Dystrophy (Cogan's microcystic)

- Variety of microcysts, dots, fingerprint or map-like epithelial lesions **(Figs 3.49.1 and 3.49.2)**
- May occur singly or in combination
- Patient may present with signs of bilateral recurrent corneal erosions

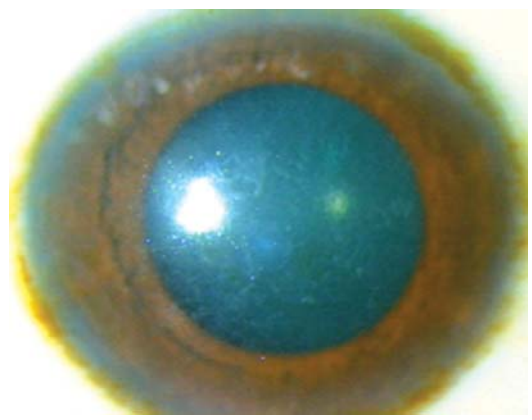


FIGURE 3.49.1: Map-dot-fingerprint dystrophy

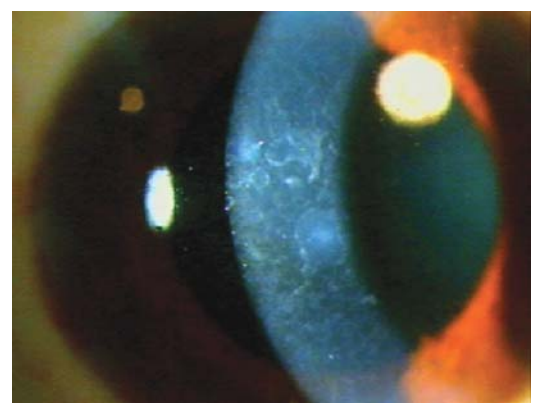


FIGURE 3.49.2: Map-dot-fingerprint dystrophy

Meesmann Dystrophy

- Very rare and innocuous condition
- Tiny epithelial cysts all over the cornea and more numerous in interpalpebral areas **(Fig 3.50.1)**
- Best visible by retro-illumination, otherwise they appear gray

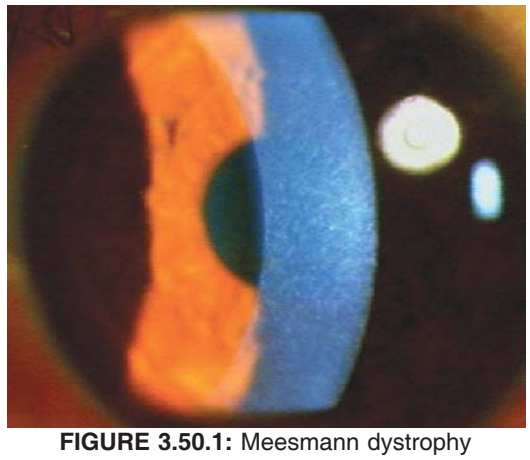


FIGURE 3.50.1: Meesmann dystrophy

Reis-Buckler's Dystrophy

- Relatively common, bilateral condition
- Reduced visual acuity in second or third decade
- Ring-shaped subepithelial opacities giving 'honeycomb' appearance **(Figs 3.51.1 and 3.51.2)**
- Entire cornea is affected with more involvement of the central area **(Figs 3.51.3 to 3.51.5)**
- *Treatment:* lamellar or deep anterior lamellar keratoplasty

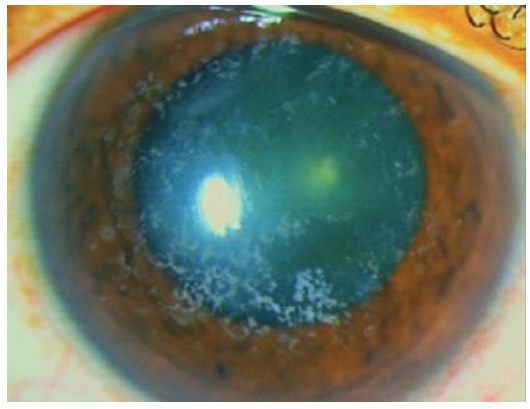


FIGURE 3.51.1: Reis-Buckler dystrophy

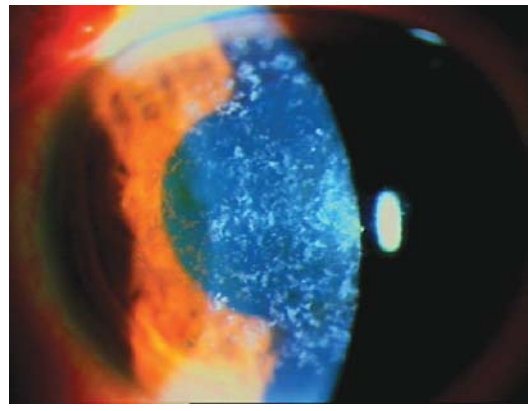


FIGURE 3.51.2: Reis-Buckler dystrophy

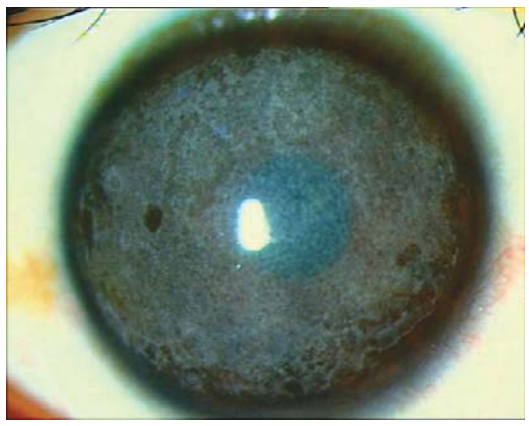


FIGURE 3.51.3: Reis-Buckler dystrophy

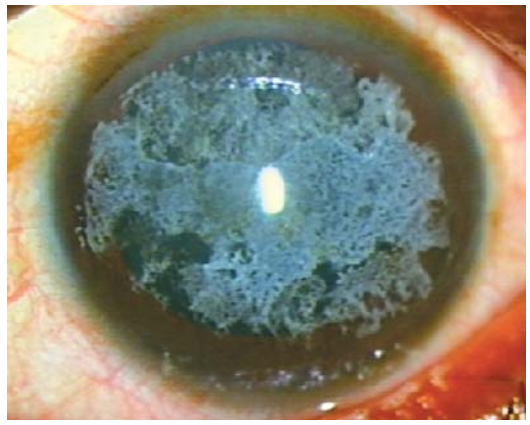


FIGURE 3.51.4: Reis-Buckler dystrophy—severe

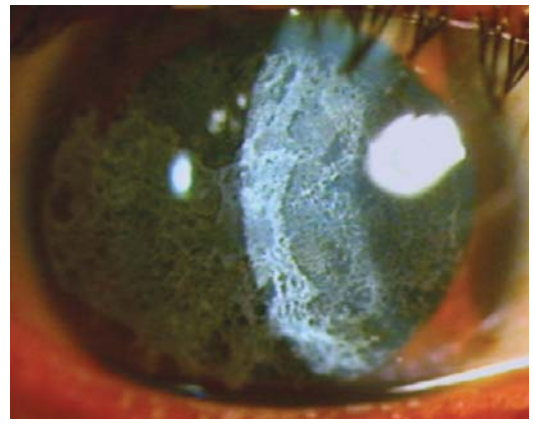


FIGURE 3.51.5: Reis-Buckler dystrophy—severe

STROMAL DYSTROPHIES

Granular Dystrophy

- Relatively common, autosomal dominant, bilateral disease
- Starts around puberty and progresses slowly
- Lesions appear as discrete, crumb-like white granules within the anterior stroma of the central cornea **(Figs 3.52.1 and 3.52.2)**
- With time the lesions become larger and more numerous and extend into the deeper stroma **(Figs 3.52.3 and 3.52.4)**
- Stroma in between the opacities and the peripheral cornea remains clear **(Figs 3.52.5 and 3.52.6)**
- Visual acuity usually remains good
- *Treatment:* deep anterior lamellar keratoplasty or penetrating keratoplasty

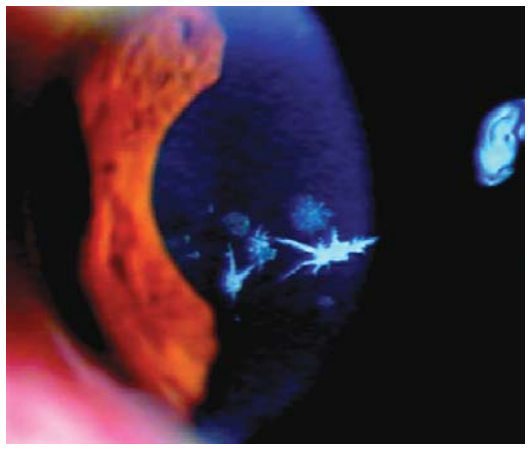


FIGURE 3.52.1: Granular dystrophy

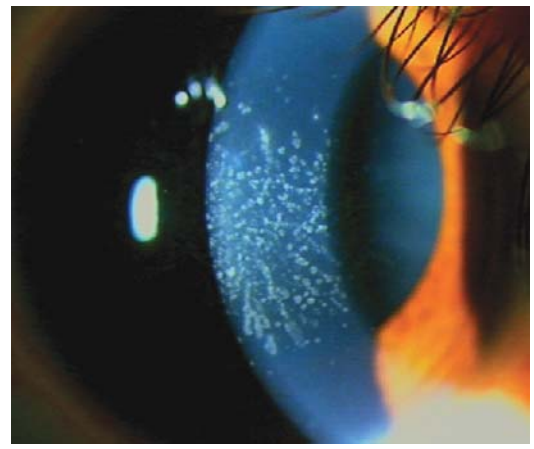


FIGURE 3.52.2: Granular dystrophy

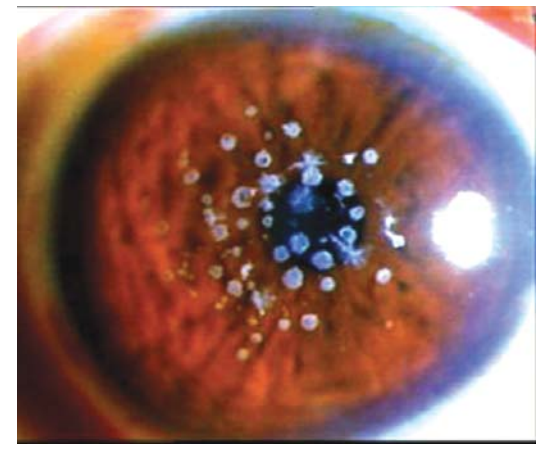


FIGURE 3.52.3: Granular dystrophy

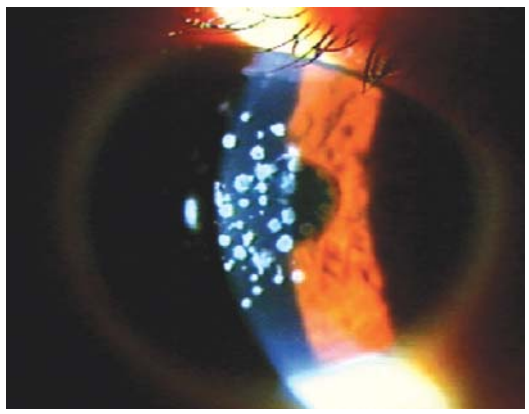


FIGURE 3.52.4: Granular dystrophy

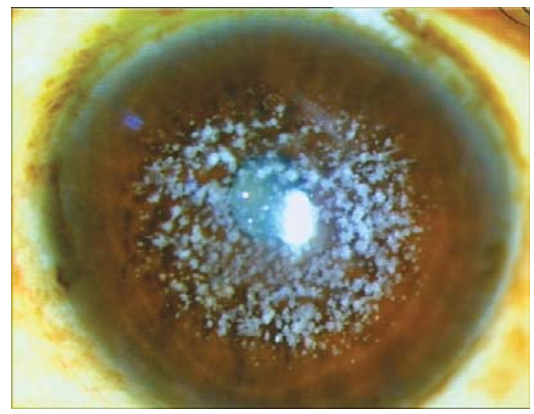


FIGURE 3.52.5: Granular dystrophy

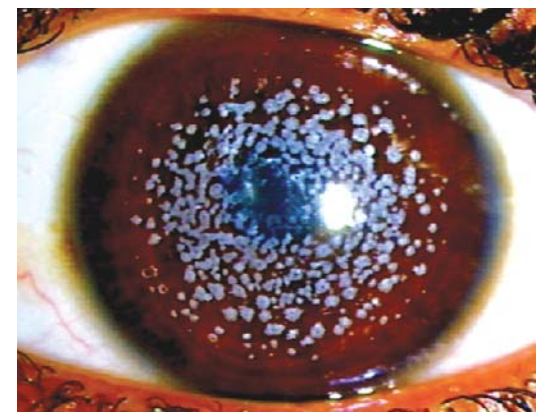


FIGURE 3.52.6: Granular dystrophy

Macular Dystrophy

- Rare, bilateral disease with autosomal recessive inheritance
- *Three types:*
  - *Type I:* presents in childhood with recurrent erosion
  - *Type II:* presents in second decade with mild erosion
  - *Type III:* presents in infancy with severe erosive attacks
- Significant impairment of vision at an early stage
- Central, focal, gray-white poorly defined opacities in cloudy stroma **(Fig 3.53.1)**
- Lesions involve the entire thickness of stroma and also extend upto the limbus **(Fig 3.53.2)**
- *Treatment:* penetrating keratoplasty; deep anterior lamellar keratoplasty in selected cases

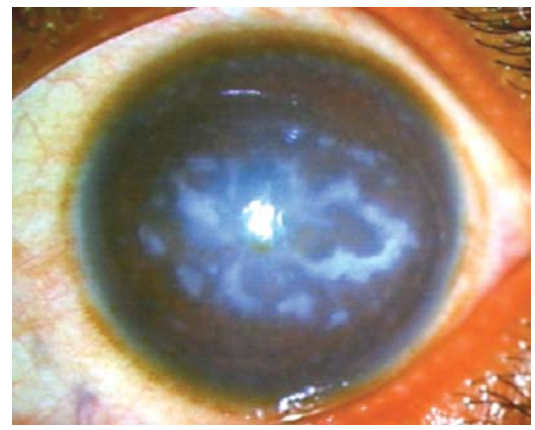


FIGURE 3.53.1: Macular dystrophy—type I

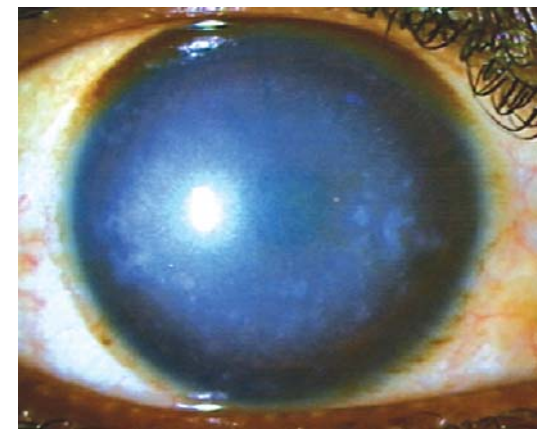


FIGURE 3.53.2: Macular dystrophy—type II

Lattice Dystrophy

- Uncommon, bilateral condition with mixed inheritance
- Also *three types:*
  - *Type I*: autosomal dominant; fine branching spider-like refractile lines which interlace and overlap at different levels within the stroma **(Figs 3.54.1 to 3.54.3)**
  - *Type II:* autosomal dominant associated with systemic amyloidosis; thicker lattice lines and less numerous **(Figs 3.54.4 and 3.54.5)**
  - *Type III:* autosomal recessive; lattice lines coarser than type I and may extend upto the limbus **(Figs 3.54.6 and 3.54.7)**
- With time, a diffuse corneal haze develops
- Visual acuity may be significantly impaired by 30-40 years of age
- Recurrent erosions **(Fig 3.54.8)** and secondary spheroidal degeneration **(Fig 3.54.9)** are common
- *Treatment:* penetrating keratoplasty, recurrence in the graft is common

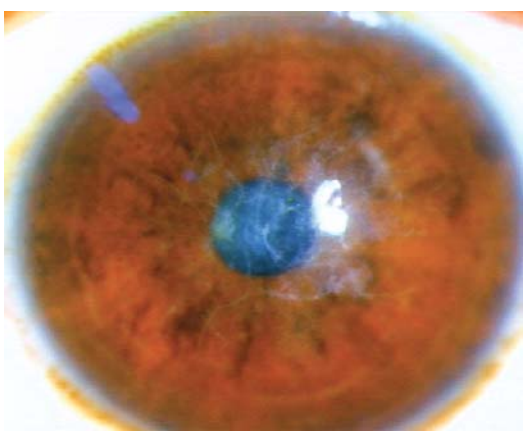


FIGURE 3.54.1: Lattice dystrophy—type I

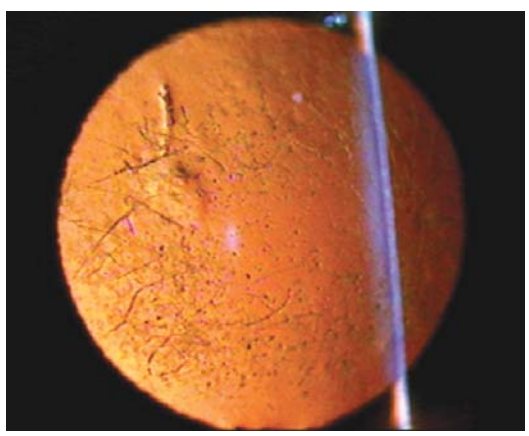


FIGURE 3.54.2: Lattice dystrophy—type I

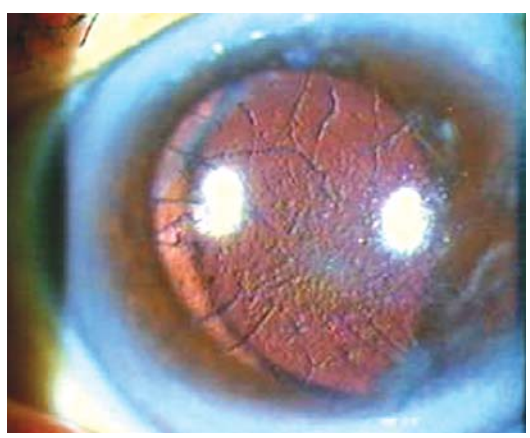


FIGURE 3.54.3: Lattice dystrophy—type I

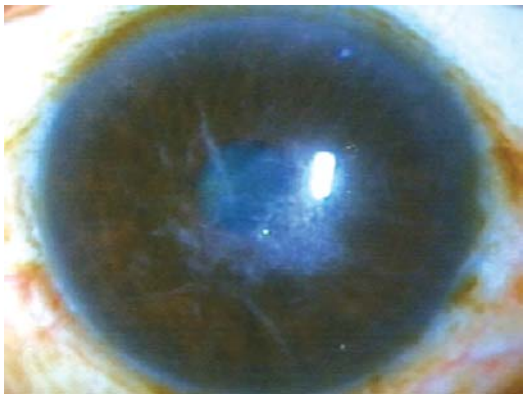


FIGURE 3.54.4: Lattice dystrophy—type II

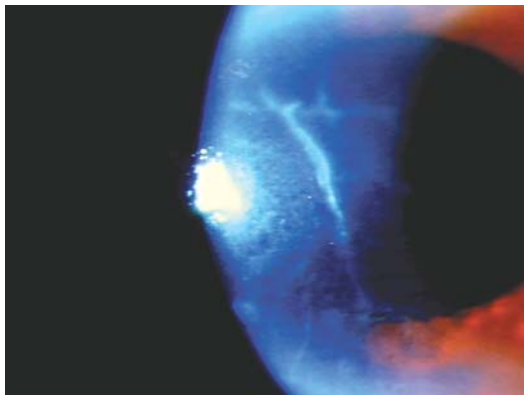


FIGURE 3.54.5: Lattice dystrophy—type II

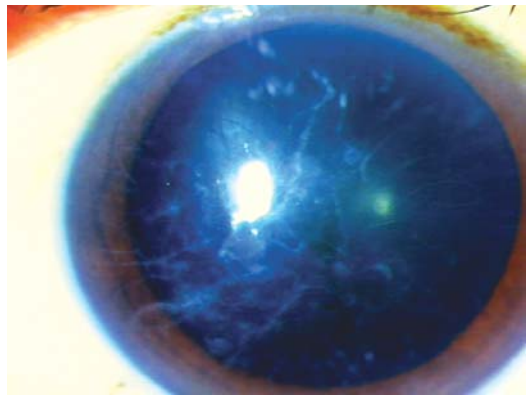


FIGURE 3.54.6: Lattice dystrophy—type III

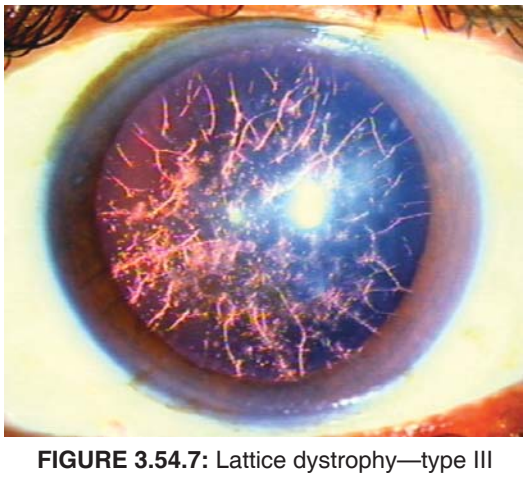


FIGURE 3.54.7: Lattice dystrophy—type III

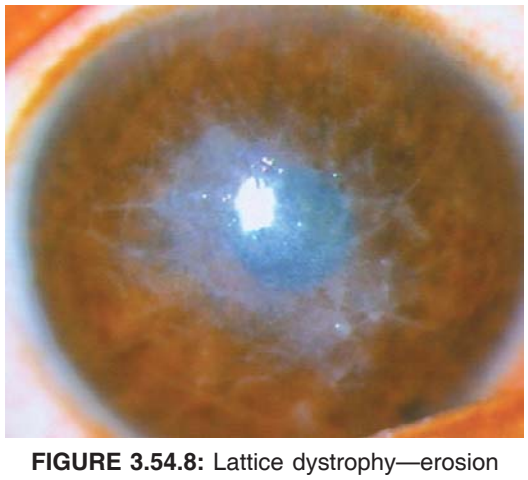


FIGURE 3.54.8: Lattice dystrophy—erosion

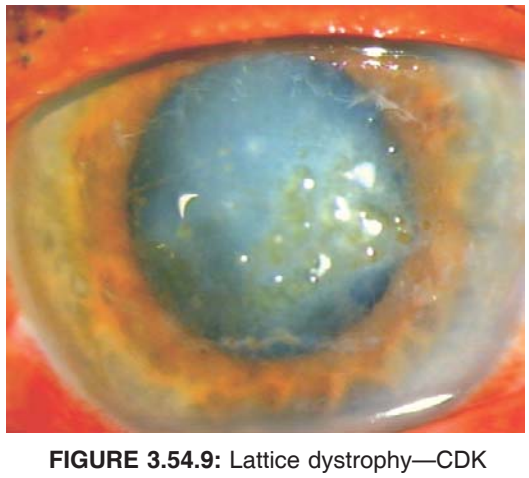


FIGURE 3.54.9: Lattice dystrophy—CDK

Avellino (granular-lattice) Dystrophy

- Very rare, autosomal dominant
- Anterior stromal dystrophy is granular and posterior stromal lesions suggestive of lattice dystrophy **(Fig 3.55.1)**

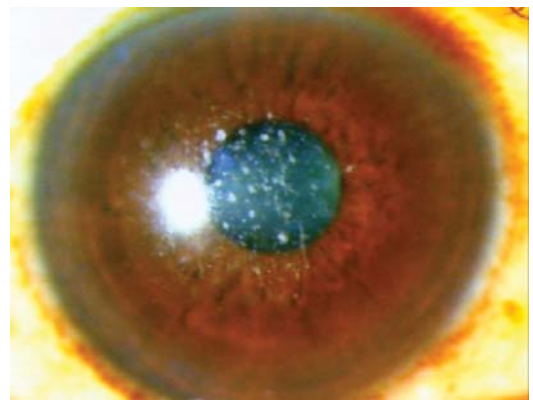


FIGURE 3.55.1: Avellino dystrophy

Central (Schnyder) Crystalline Dystrophy

- Very rare with autosomal dominant inheritance
- Needle-shaped crystalline lesions involving the central stroma
- Associated with diffuse central stromal haze

Congenital Hereditary Stromal Dystrophy

- Very rare, bilateral condition with autosomal dominant inheritance
- Usual manifestation during infancy
- Corneal clouding without any edema **(Figs 3.56.1 and 3.56.2)**
- Associated nystagmus and squint
- To be differentiated from CHED and congenital glaucoma
- *Treatment:* Penetrating keratoplasty at the earliest

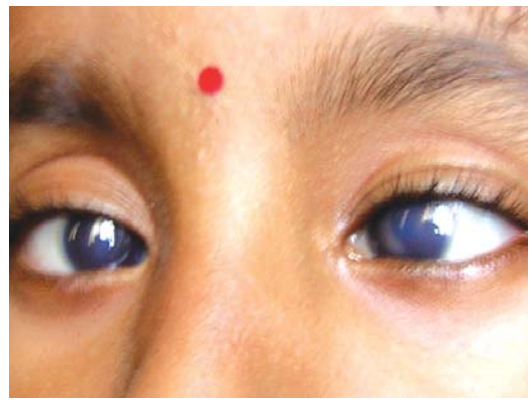


FIGURE 3.56.1: Congenital hereditary stromal dystrophy

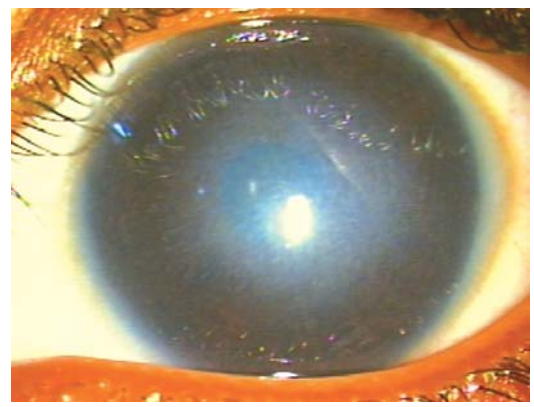


FIGURE 3.56.2: Congenital hereditary stromal dystrophy

POSTERIOR DYSTROPHIES

Posterior Polymorphous Dystrophy

- Uncommon, bilateral condition
- Vesicular, band-like, or geographical lesions on the posterior surface of the cornea **(Figs 3.57.1 to 3.57.3)**
- Lesion may be easily overlooked
- May cause corneal edema

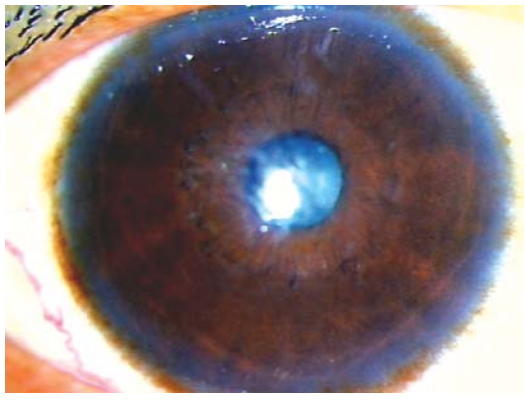


FIGURE 3.57.1: Posterior polymorphous dystrophy

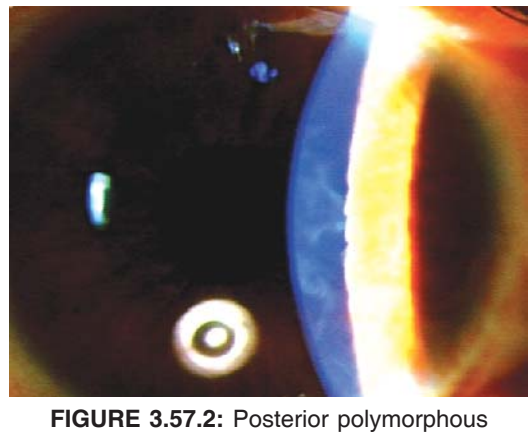


FIGURE 3.57.2: Posterior polymorphous dystrophy

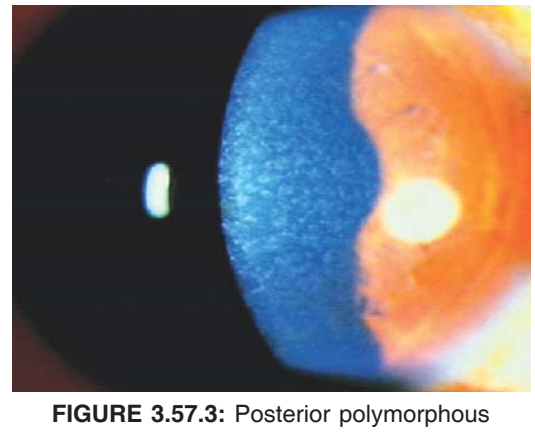


FIGURE 3.57.3: Posterior polymorphous dystrophy

Cornea Guttata

- A common aging process resulting in focal accumulation of excrescences on the posterior surface of the Descemet's membrane
- They disrupt the normal endothelial mosaic **(Fig 3.58.1)**
- With confluent lesions, they appear as dark spots or 'beaten metal' appearance **(Figs 3.58.2 and 3.58.3)**
- May be seen in early stage of Fuchs' dystrophy

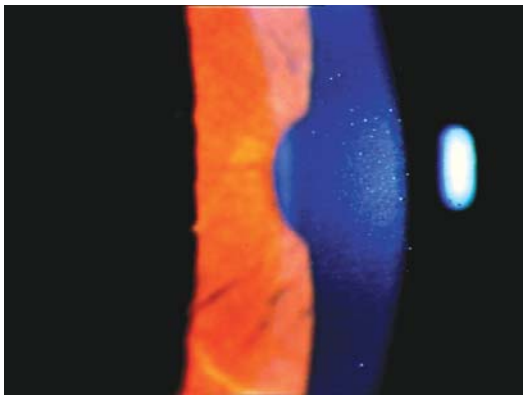


FIGURE 3.58.1: Cornea guttata

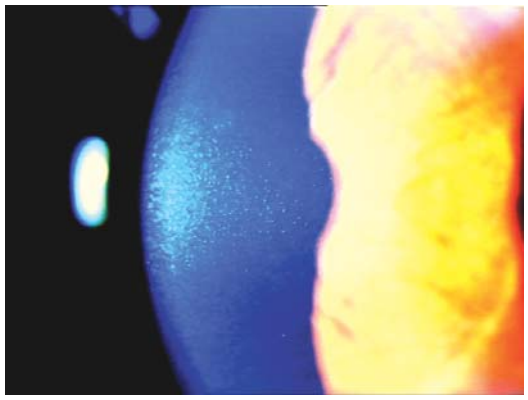


FIGURE 3.58.2: Cornea guttata—beaten metal appearance

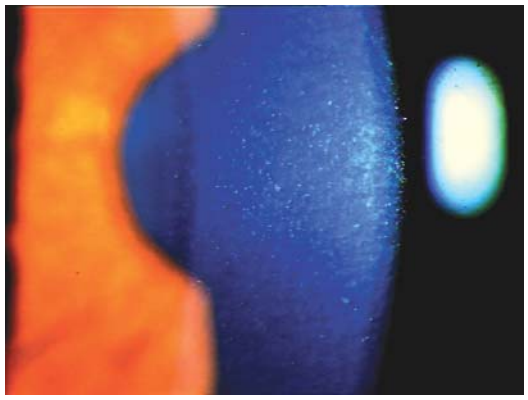


FIGURE 3.58.3: Cornea guttata—beaten metal appearance

Fuchs' Endothelial Dystrophy

- Relatively common, bilateral condition with autosomal dominant inheritance
- Slowly progressive, and more common in elderly female
- Central corneal guttata without any symptom which gradually spreads towards periphery **(Figs 3.59.1 and 3.59.2)**
- Stroma becomes edematous with endothelial decompensation **(Fig 3.59.3)**
- Epithelial edema gradually develops with impairment of vision **(Fig 3.59.4)**
- Ultimately, bullous keratopathy develops with severe symptoms **(Figs 3.59.5 to 3.59.7)**
- Gradually, scarring occurs with vascularization **(Fig 3.59.8)**
- *Treatment:* hypertonic saline, bandage contact lens and ultimately PK

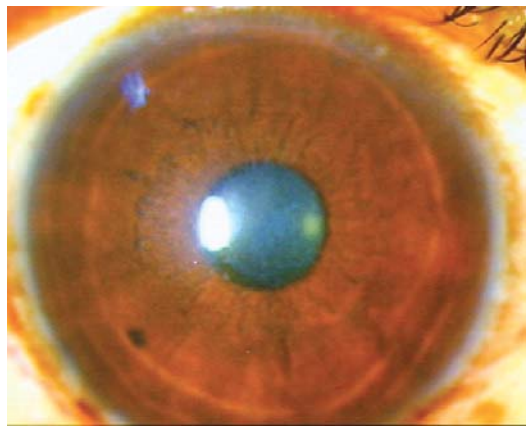


FIGURE 3.59.1: Fuchs' dystrophy—cornea guttata

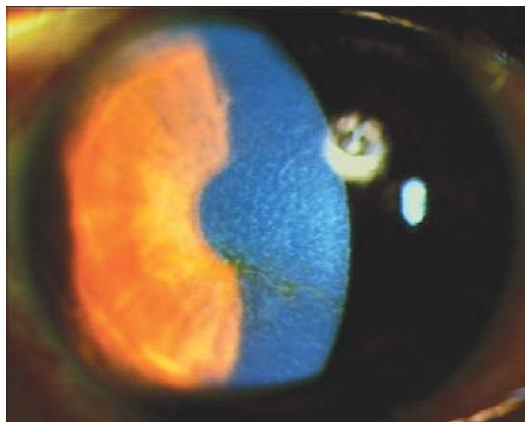


FIGURE 3.59.2: Fuchs' dystrophy—cornea guttata

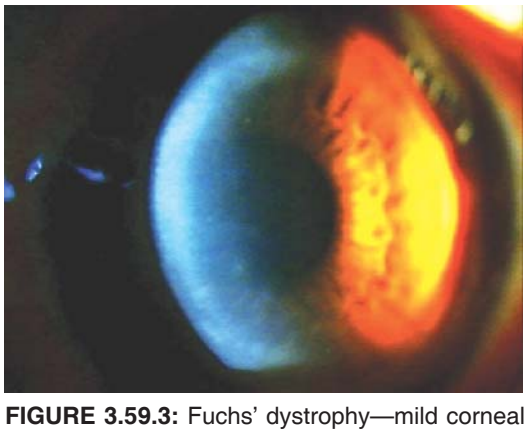


FIGURE 3.59.3: Fuchs' dystrophy—mild corneal edema

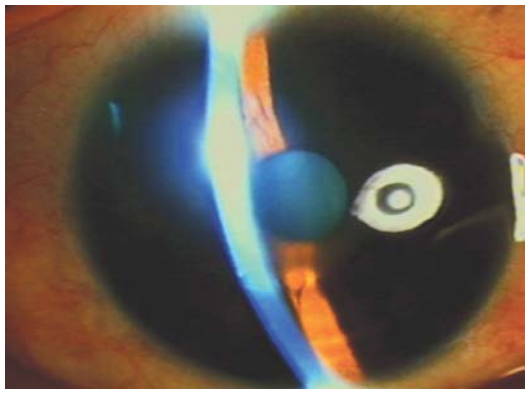


FIGURE 3.59.4: Fuchs' dystrophy—moderate corneal edema

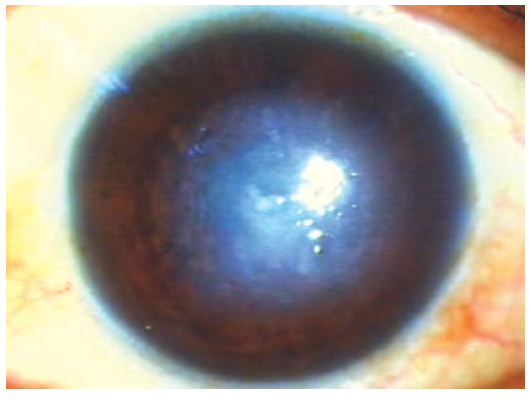


FIGURE 3.59.5: Fuchs' dystrophy—severe corneal edema

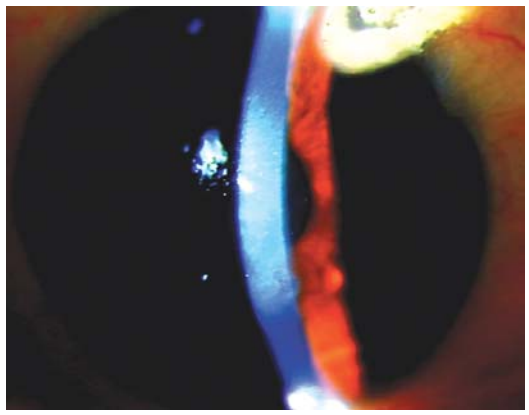


FIGURE 3.59.6: Fuchs' dystrophy—severe corneal edema

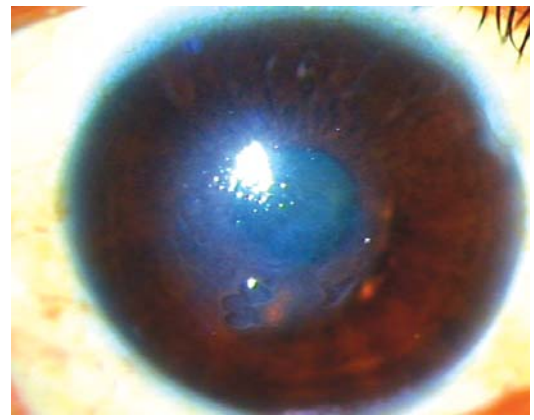


FIGURE 3.59.7: Fuchs' dystrophy—bullous keratoplasty

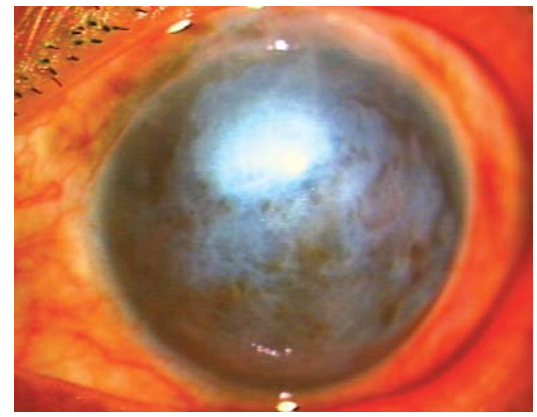


FIGURE 3.59.8: Fuchs' dystrophy—scarring

Congenital Hereditary Endothelial Dystrophy

- Rare, autosomal dominant inheritance
- Bilateral corneal edema occurs any time during first decade **(Figs 3.60.1 and 3.60.2)**
- Infantile form should be differentiated from congenital glaucoma
- *Treatment:* penetrating keratoplasty as early as possible

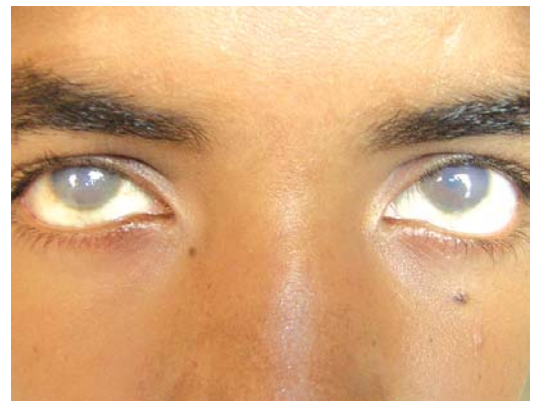


FIGURE 3.60.1: Congenital hereditary endothelial dystrophy

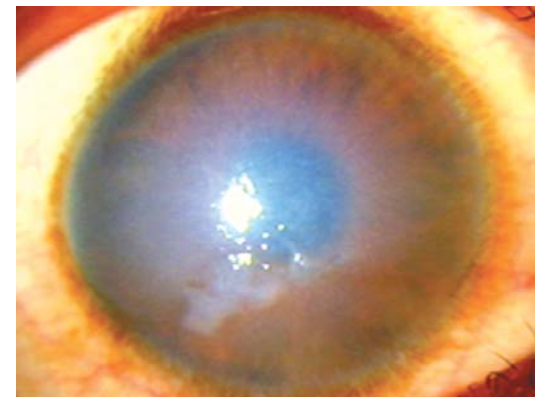


FIGURE 3.60.2: Congenital hereditary endothelial dystrophy

ECTATIC DYSTROPHY: KERATOCONUS

- Bilateral conical protrusion of the central part of the cornea with thinning **(Fig 3.61.1)**
- Starts around puberty and slowly progressive
- May be associated with vernal conjunctivitis, ectopia lentis or Down's syndrome
- Irregular retinoscopic reflex and high irregular myopic astigmatism
- Abnormal *oil-droplet* red reflex
- Thinning of the central cornea with protrusion just below and nasal to the center
- *Munson's sign:* bulging of lower lids in down gaze **(Fig 3.61.2)**
- *Vogt's lines (striae):* vertical folds at the level of deep stroma and Descemet's **(Fig 3.61.3)**
- *Fleischer's ring:* epithelial iron line at the base of the cone **(Fig 3.61.4)**
- *Rizutti's sign:* corneal reflection on the nasal limbus when light is thrown from the temporal limbus **(Figs 3.61.5 and 3.61.6)**

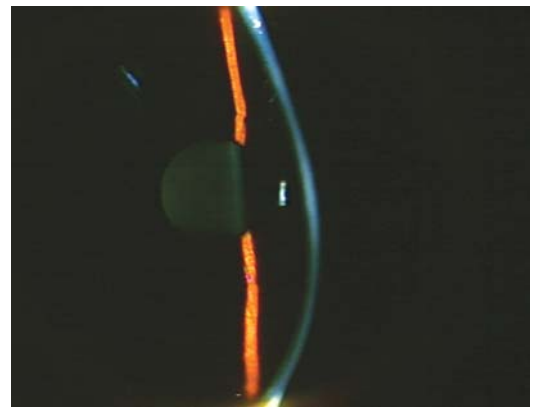


FIGURE 3.61.1: Keratoconus

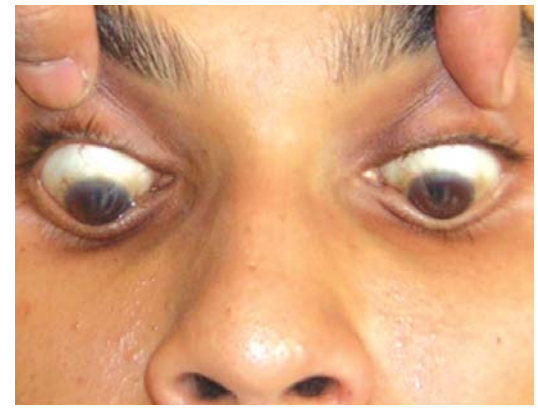


FIGURE 3.61.2: Keratoconus—Munson's sign

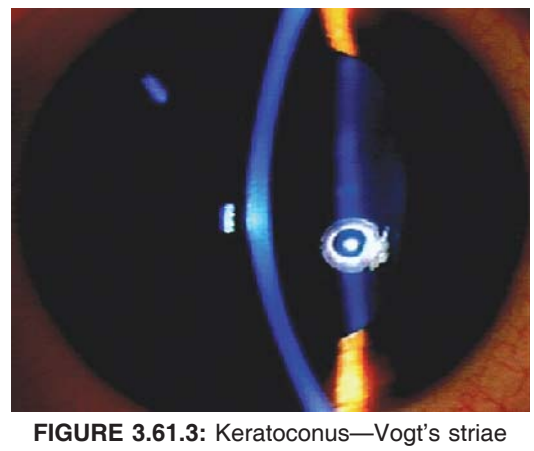


FIGURE 3.61.3: Keratoconus—Vogt's striae

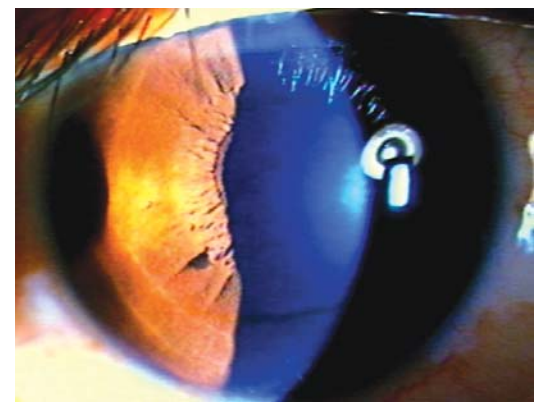


FIGURE 3.61.4: Keratoconus—Fleischer's ring

- *Prominent corneal nerves* **(Fig 3.61.7)**
- *Acute hydrops:* sudden corneal edema due to acute seepage of the aqueous into the **(Fig 3.61.8)** corneal stroma and epithelium resulting from rupture of Descemet's membrane
- Variable degree of apical corneal scarring **(Fig 3.61.9)**
- *Treatment:* spectacles, RGP contact lenses, penetrating keratoplasty or deep anterior lamellar keratoplasty

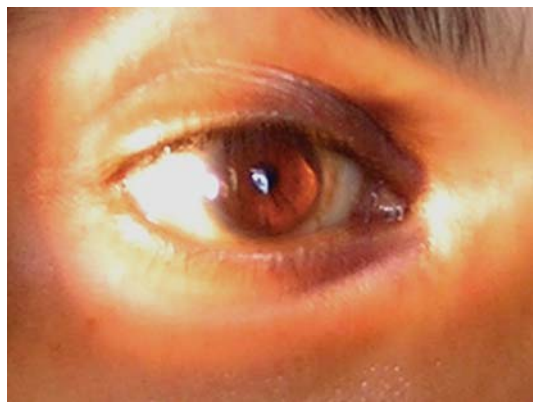


FIGURE 3.61.5: Keratoconus—Rizutti's sign

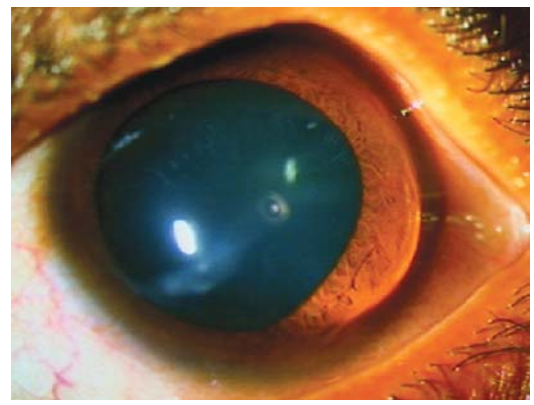


FIGURE 3.61.6: Keratoconus—Rizutti's sign

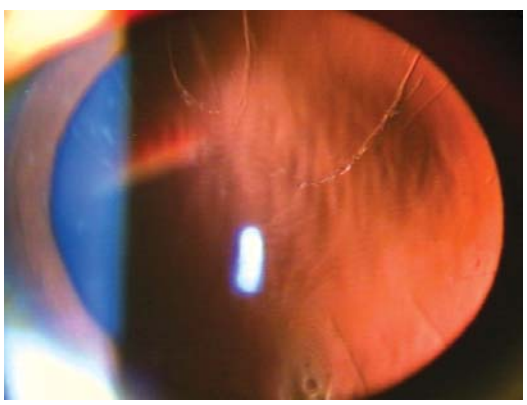


FIGURE 3.61.7: Keratoconus—prominent corneal nerve

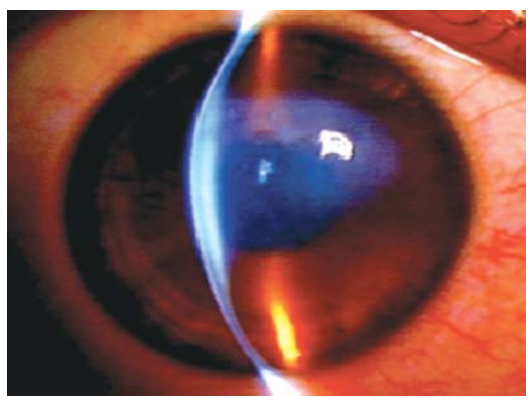


FIGURE 3.61.8: Keratoconus—acute hydrops

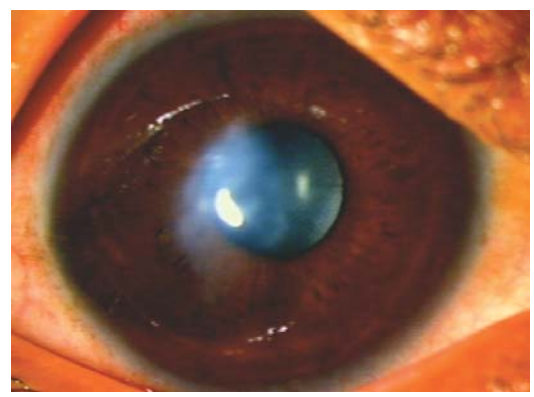


FIGURE 3.61.9: Keratoconus—apical scarring

OTHER CORNEAL DISORDERS

Striate Keratopathy (keratitis)

- Unilateral corneal edema with folds in the Descemet's membrane usually after a cataract surgery, or tight suturing on the cornea or at limbus
- Appear as delicate gray lines in the deeper cornea **(Figs 3.62.1 and 3.62.2)**
- Disappear spontaneously as the wound heals
- Sometimes, they persist and end with corneal decompensation **(Figs 3.62.3 and See 3.13.4)**
- *Treatment:* of corneal edema and penetrating keratoplasty in corneal decompensation

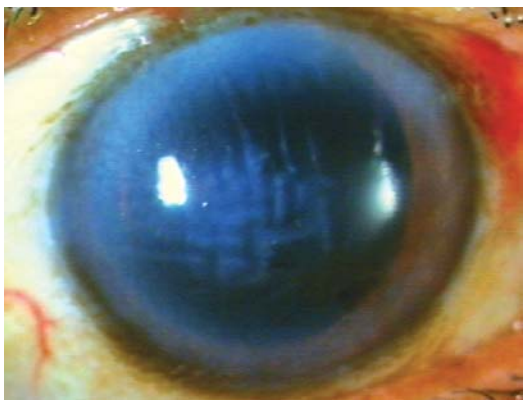


FIGURE 3.62.1: Striate keratopathy

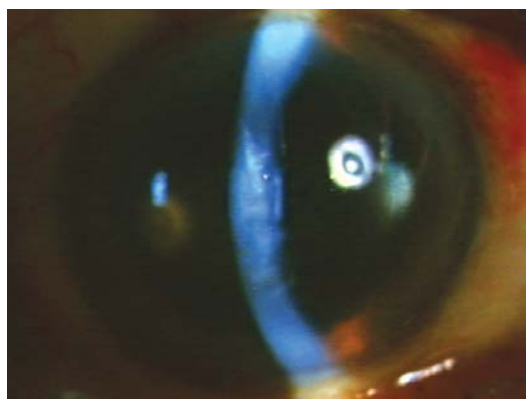


FIGURE 3.62.2: Striate keratopathy

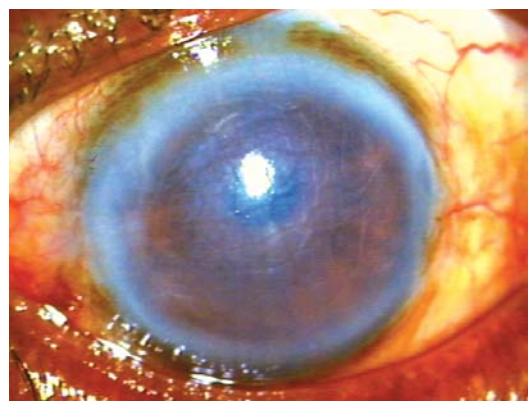


FIGURE 3.62.3: Striate keratopathy—decompensation

Corneal Abrasion

- Common, unilateral condition, usually associated with trauma
- Epithelial defect can be easily diagnosed by diffuse illumination **(Fig 3.63.1)**
- But the margins are distinctly visible after fluorescein staining **(Fig 3.63.2)**
- *Treatment:* antibiotic ointment and patching

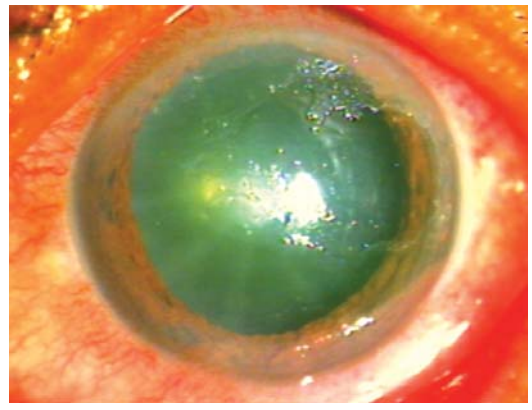


FIGURE 3.63.1: Corneal abrasion

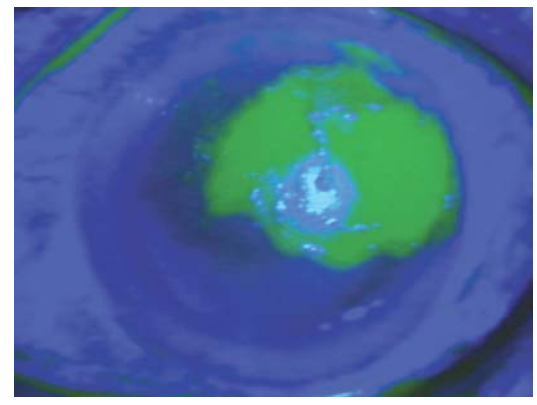


FIGURE 3.63.2: Corneal abrasion—fluorescein stain

Keratoconjunctivitis Sicca (KCS)

- Very common bilateral condition, may occur in isolation or in some systemic diseases
- Mucus debris or plaque **(Fig 3.64.1)**
- Decreased tear meniscus height **(Fig 3.64.2)**
- Reduced tear film break-up-time
- Staining of interpalpebral area with Rose Bengal in triangular fashion
- Superficial punctate keratitis stained with Rose Bengal **(Figs 3.64.3 to 3.64.5)** and Lissamine green **(Fig 3.64.6)**
- Filamentary keratopathy stained with fluorescein **(Fig 3.64.7)**
- Delen and corneal thinning leading to Descemetocele formation **(Fig 3.64.8)**
- Vascularization in severe cases **(Figs 3.64.9 and 3.64.10)**
- *Treatment:* tear substitutes, topical cyclosporine (0.05%), soft steroids, punctal occlusion, etc.

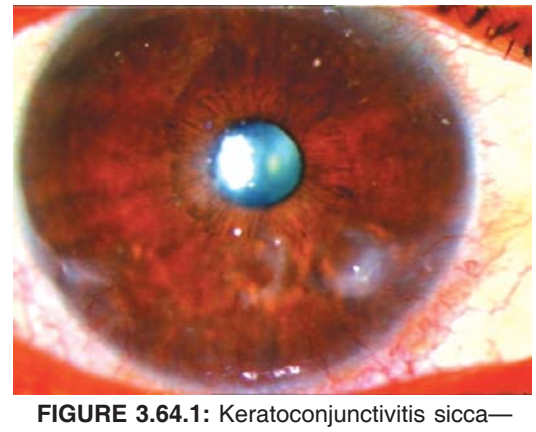


FIGURE 3.64.1: Keratoconjunctivitis sicca—mucus debris

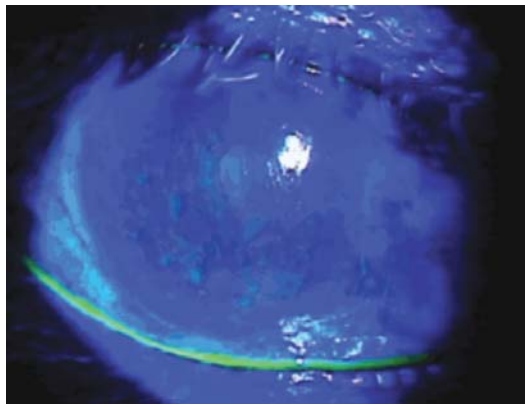


FIGURE 3.64.2: KCS—low tear meniscus height

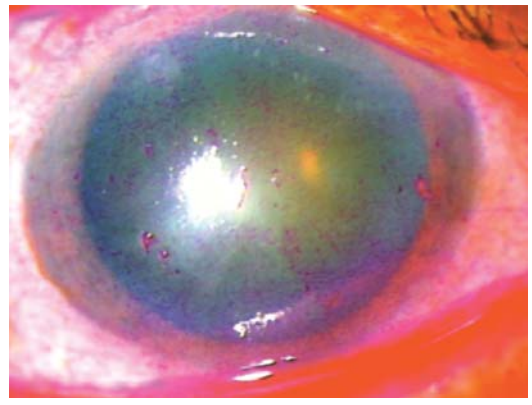


FIGURE 3.64.3: KCS—Rose Bengal stain

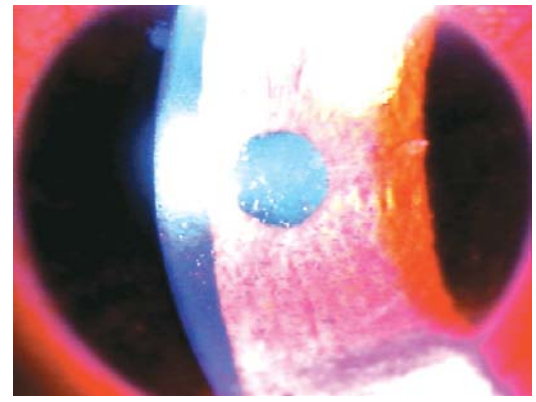


FIGURE 3.64.4: KCS—Rose Bengal stain

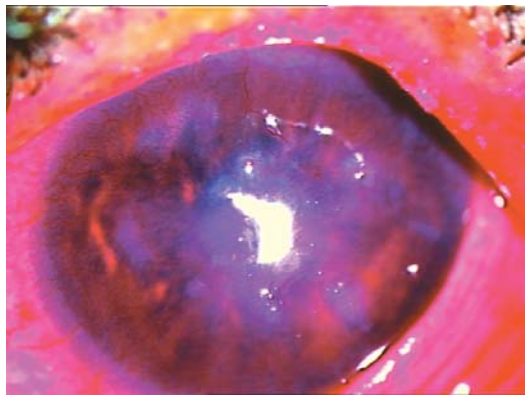


FIGURE 3.64.5: KCS—Rose Bengal stain

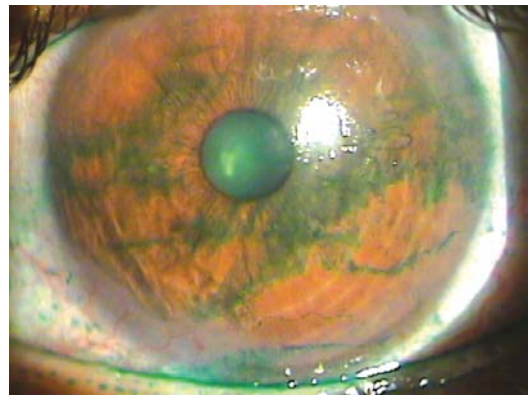


FIGURE 3.64.6: KCS—Lissamine green stain

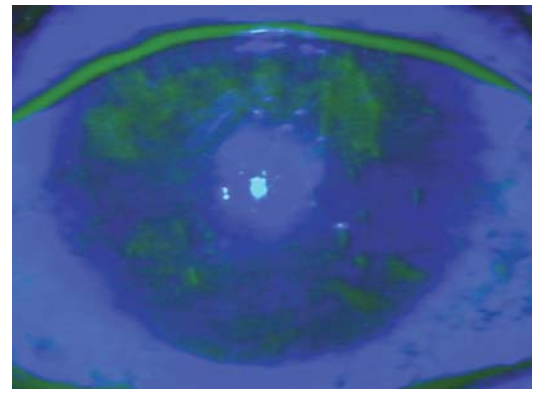


FIGURE 3.64.7: KCS—fluorescein stain

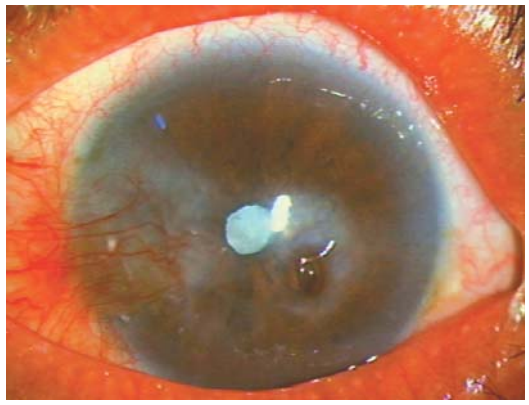


FIGURE 3.64.8: KCS—Descemetocele

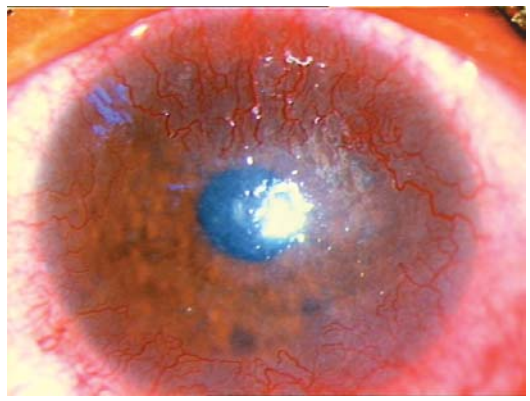


FIGURE 3.64.9: KCS—vascularization

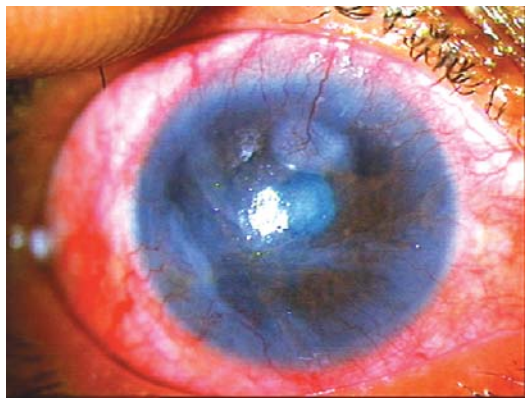


FIGURE 3.64.10: KCS—vascularization

Filamentary Keratopathy (keratitis)

- Formation of epithelial threads (filaments) on the cornea
- Filaments adhere to the cornea by one end, while the other moves freely **(Fig 3.65.1)**
- *Causes are:* keratoconjunctivitis sicca, superior limbic keratoconjunctivitis, recurrent erosions or following cataract surgery
- Beautifully stained by fluorescein **(Figs 3.65.2 and 3.65.3)**
- *Treatment:* removal of filaments by scraping then BCL, artificial tears

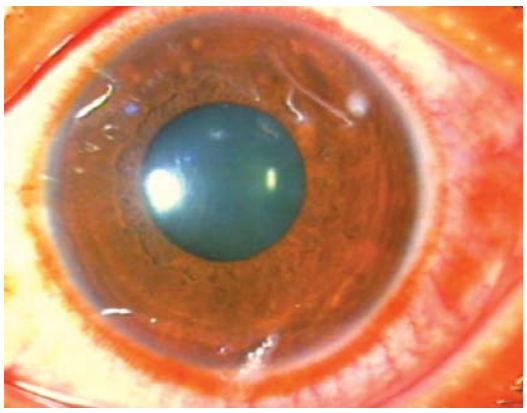


FIGURE 3.65.1: Filamentary keratopathy

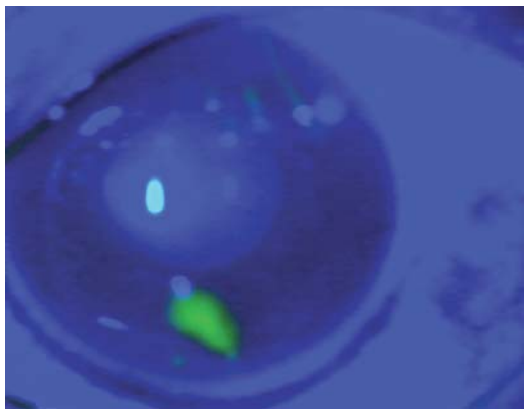


FIGURE 3.65.2: Filamentary keratopathy—fluorescein stain

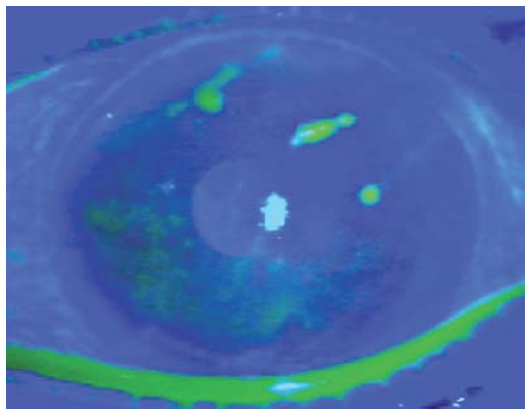


FIGURE 3.65.3: Filamentary keratopathy—fluorescein stain

Vortex Keratopathy

- Bilateral condition more commonly caused by a variety of oral medicines and also in Fabry's disease
- *Drugs are*: hydroxychloroquine, amiodarone, indomethacin, tamoxifen, chlorpromazine, etc.
- Appear as grayish or golden minute epithelial deposits arranged in a vortex fashion **(Fig 3.66.1)**
- They start at a point below the pupil and swirling outwards without involving the limbus **(Fig 3.66.2)**
- *Treatment:* withdrawal of the specific drug

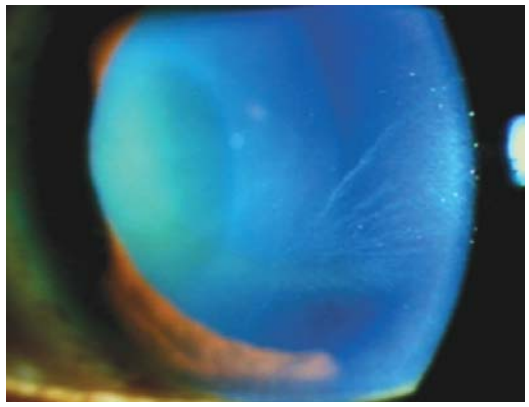


FIGURE 3.66.1: Vortex keratopathy

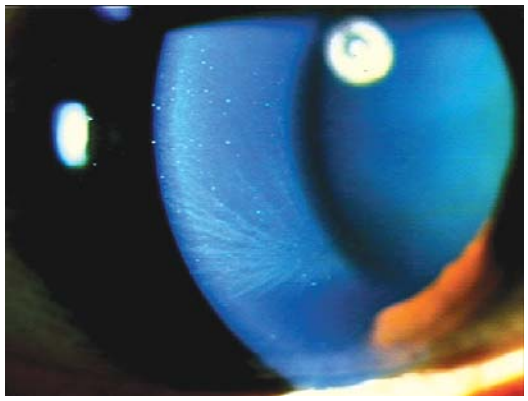


FIGURE 3.66.2: Vortex keratopathy

Crocodile Shagreen

- Uncommon, bilateral, innocuous condition
- Grayish-white polygonal opacities separated by clear spaces **(Fig 3.67.1)**
- *Anterior crocodile shagreen*—involves anterior two-third of stroma and more frequent than *posterior* type **(Fig 3.67.2)**

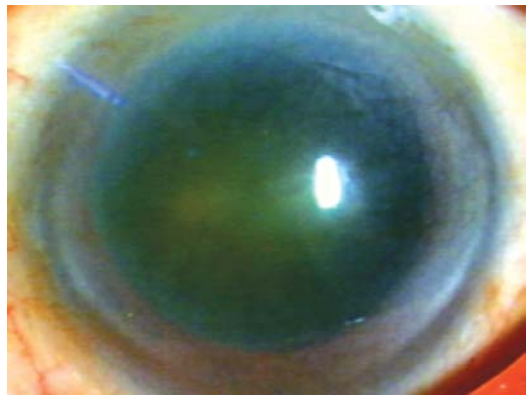


FIGURE 3.67.1: Crocodile shagreen

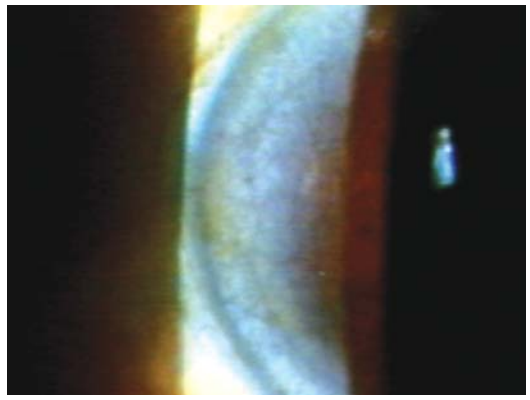


FIGURE 3.67.2: Crocodile shagreen

Prominent Corneal Nerves

- Associated with a variety of ocular and systemic conditions **(Fig 3.68.1)**
- *Causes are:* neurofibromatosis, leprosy, primary amyloidosis, keratoconus, acanthameba keratitis, failed corneal graft, etc.

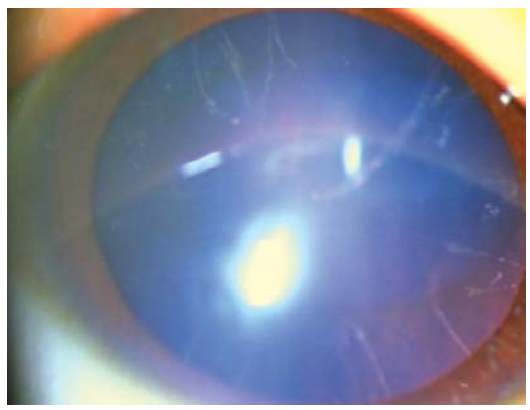


FIGURE 3.68.1: Prominent corneal nerve

Descemet's Tear

- *Causes are:*
  - *birth trauma*—vertical tear **(Figs 3.69.1 to 3.69.3)**
  - *infantile glaucoma:* horizontal tear (Haab's striae) **(Fig 3.69.4)**
  - *keratoconus/keratoglobus:* elliptical tear in acute hydrops **(Figs 3.69.5 and 3.69.6)**
  - *surgical trauma:* any direction **(Fig 3.69.7)**
- *Treatment:* observation and no active treatment

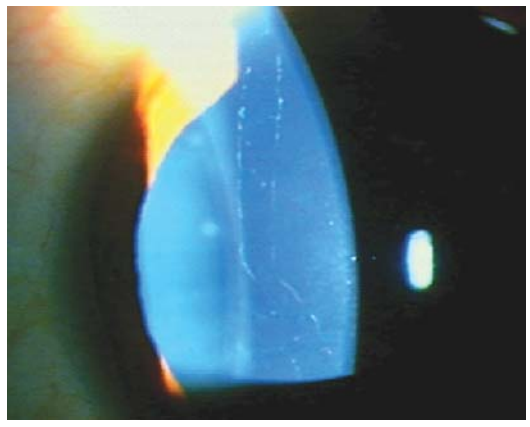


FIGURE 3.69.1: Descemet's tear—birth trauma

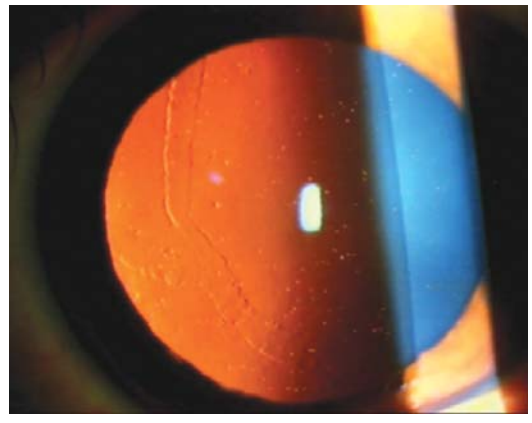


FIGURE 3.69.2: Descemet's tear—birth trauma

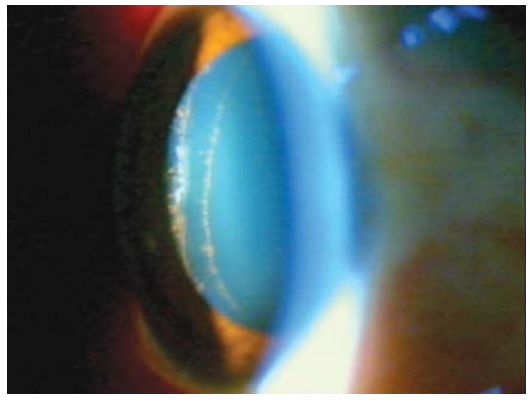


FIGURE 3.69.3: Descemet's tear—birth trauma

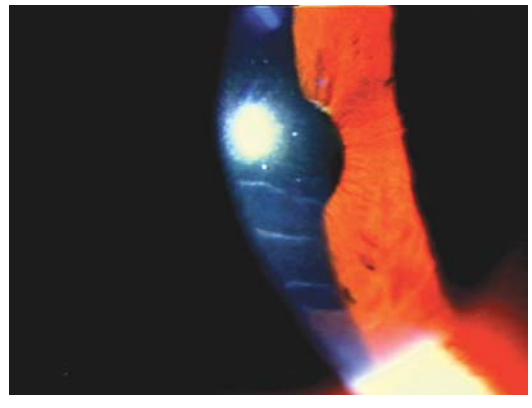


FIGURE 3.69.4: Descemet's tear—Haab's striae

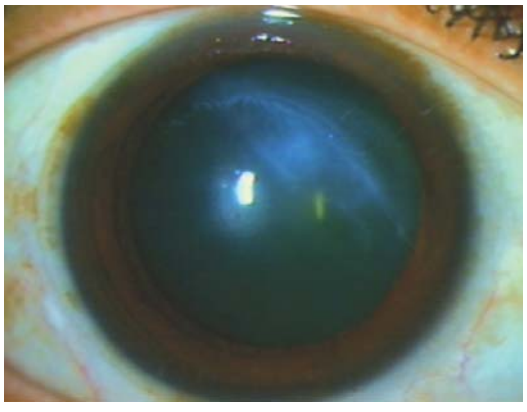


FIGURE 3.69.5: Descemet's tear—keratoconus

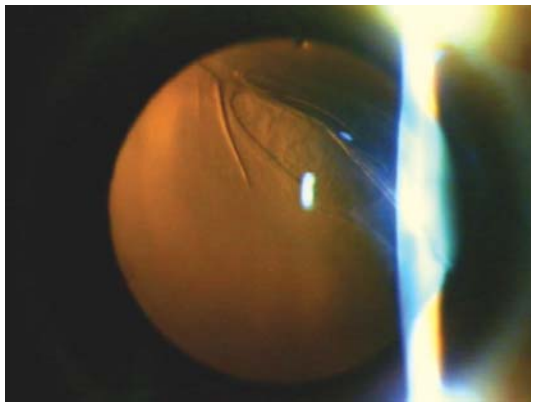


FIGURE 3.69.6: Descemet's tear—keratoconus

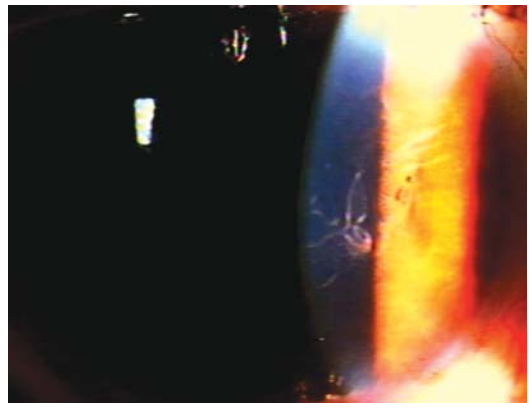


FIGURE 3.69.7: Descemet's tear—surgical

Descemet's Folds and Wrinkles

- Associated with a variety of ocular conditions and best visible under retro-illumination
  - surgical **(Fig 3.70.1)** or blunt trauma **(Fig 3.70.2)**
  - chemical burn **(Fig 3.70.3)**
  - ocular hypotony **(Fig 3.70.4)**
  - disciform keratitis **(Fig 3.70.5)**
  - congenital syphilis, etc.

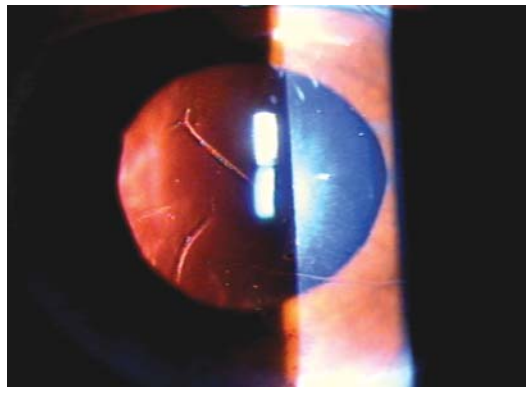


FIGURE 3.70.1: Descemet's fold—surgical

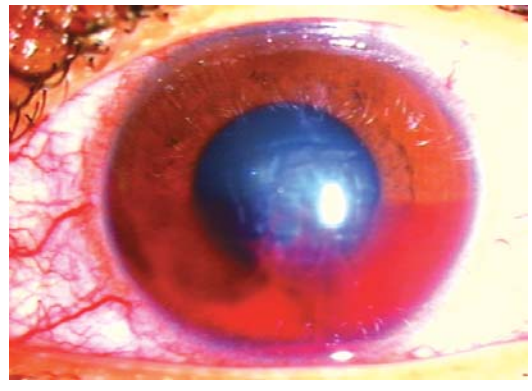


FIGURE 3.70.2: Descemet's fold—blunt trauma

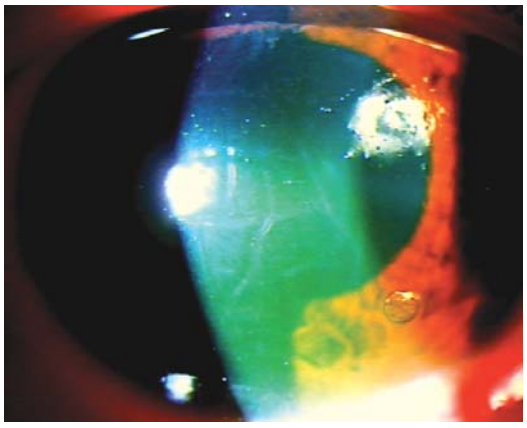


FIGURE 3.70.3: Descemet's fold—chemical

FIGURE 3.70.4: Descemet's fold—hypotony

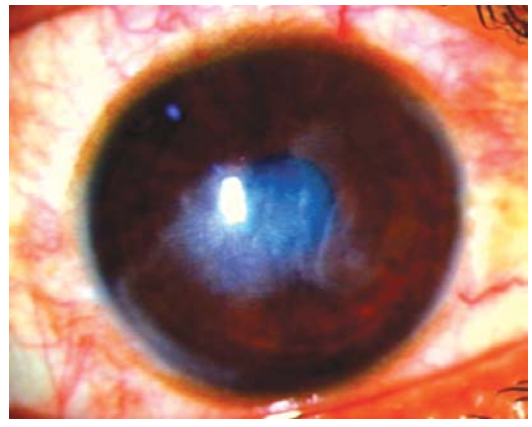


FIGURE 3.70.5: Descemet's fold—disciform keratitis

Descemet's Detachment

- Detachment of Descemet's membrane is mainly caused by surgical trauma **(Fig 3.71.1)**
- It may be partial or total
- Associated with diffuse **(Figs 3.71.2 and 3.71.3)** or localized corneal edema **(Fig 3.71.4**
- Sometimes cornea remains surprinsingly clear even after large Descemet's detachment **(Fig 3.71.5)**
- *Treatment:* in total detachment, re-surgery to reattach the membrane

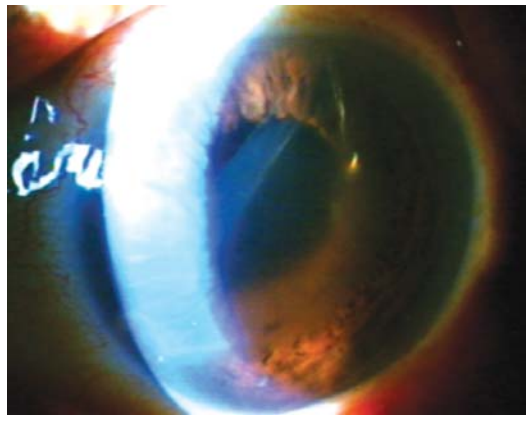


FIGURE 3.71.1: Descemet's detachment—curled

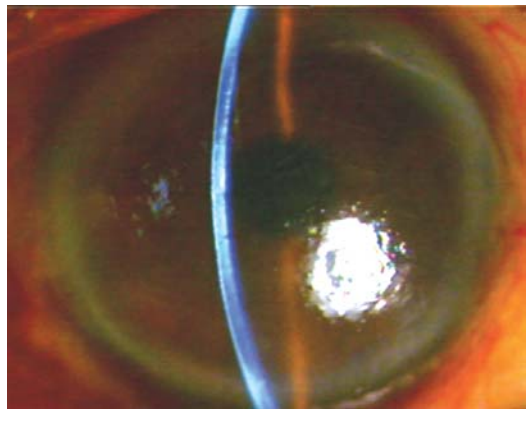


FIGURE 3.71.2: Descemet's detachment—diffuse edema

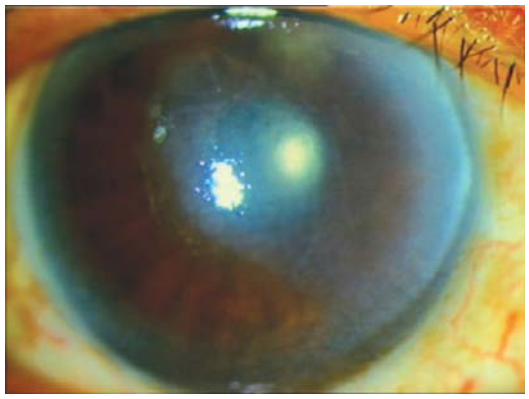


FIGURE 3.71.3: Descemet's detachment—diffuse edema

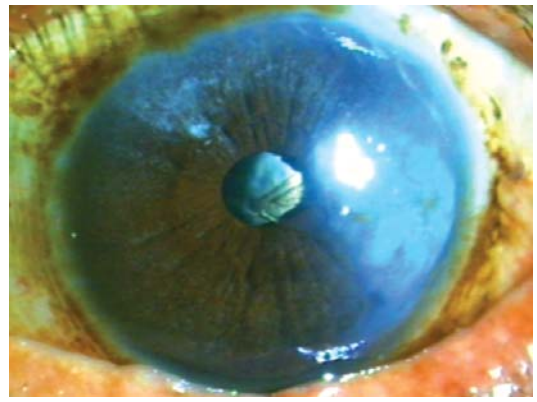


FIGURE 3.71.4: Descemet's detachment—localized corneal edema

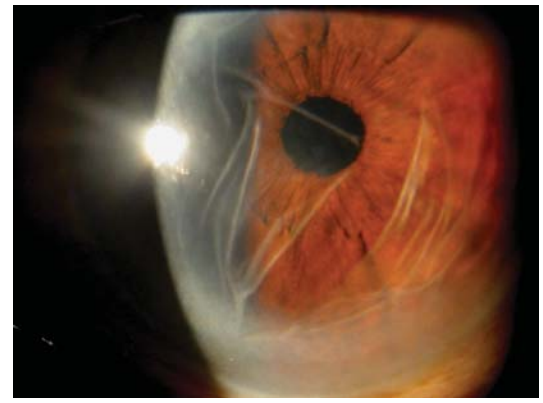


FIGURE 3.71.5: Descemet's detachment—clear cornea

Corneal Dellen

- Common, unilateral, symptomless condition
- May be seen in KCS **(Figs 3.72.1 and 3.72.2)**, adjacent to pterygium or other nodular limbal lesion **(Fig 3.72.3),** or after tight suturing
- Saucer-shaped thinning secondary to localized dry areas and stromal dehydration
- Epithelium is intact and does not stain with fluorescein, but pooling may be present **(Fig 3.72.4)**
- *Treatment:* tear substitutes and treatment of the cause

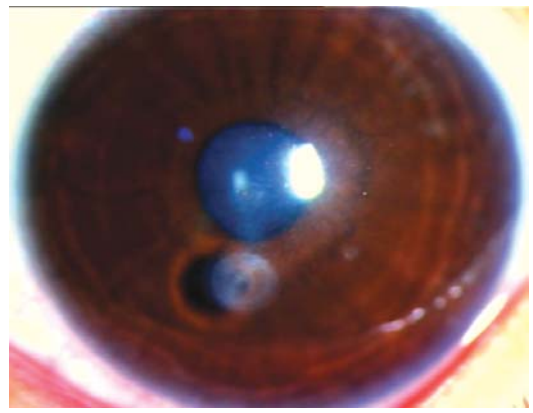


FIGURE 3.72.1: Corneal dellen—KCS

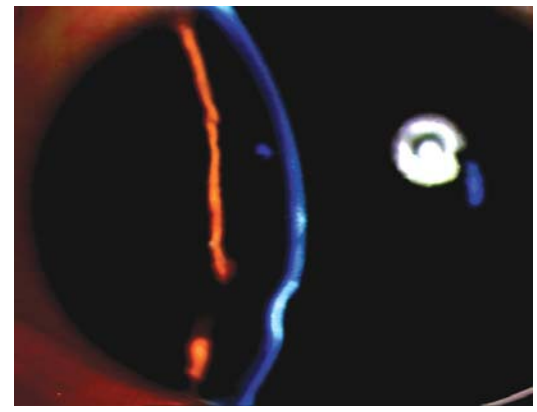


FIGURE 3.72.2: Corneal dellen—KCS

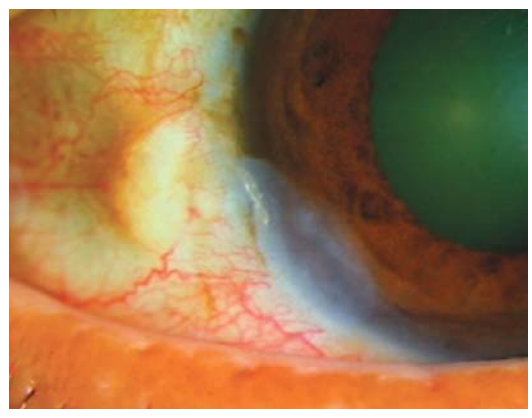


FIGURE 3.72.3: Corneal dellen—limbal lesion

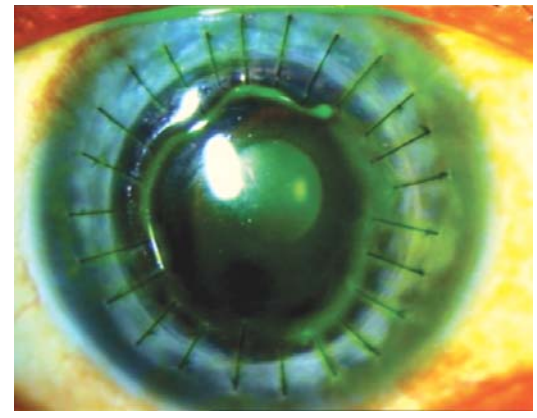


FIGURE 3.72.4: Corneal dellen—fluorescein dye pulling

Corneal Signs in Vitamin-A Deficiency

- Vitamin A deficiency diseases (VAD) is a blinding disorder mainly due to corneal involvement
- Mostly occurs in malnourished children between 6 months to 4 years of developing countries
- *Corneal xerosis (X2)*
  - Hazy lustuerless, dry cornea, mainly in the inferior part **(Figs 3.73.1 and 3.73.2)**

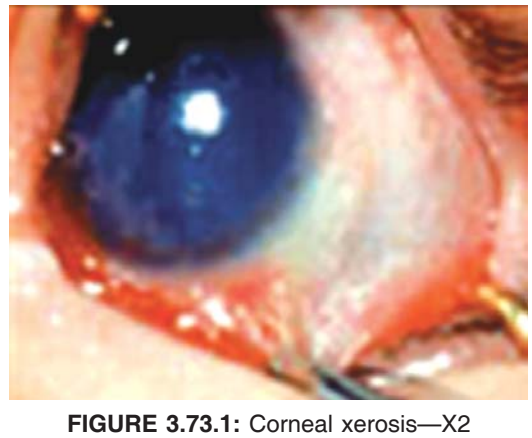


FIGURE 3.73.1: Corneal xerosis—X2

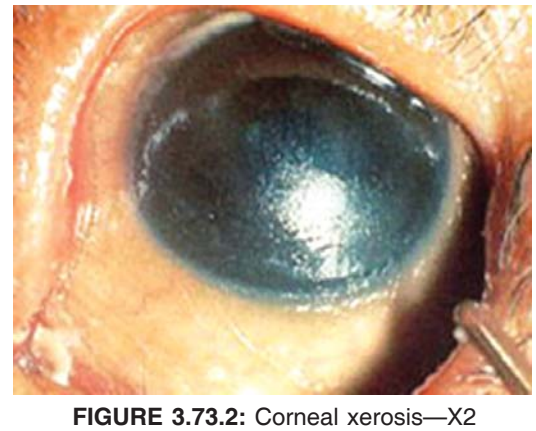


FIGURE 3.73.2: Corneal xerosis—X2

- *Corneal ulceration (keratomalacia) (X3A, 3B)*
  - round, oval, punched out defects, surrounded by xerotic cornea
  - X3A: when <1/3 cornea is involved
  - X3B: when >1/3 cornea is involved
  - perforation may occur within 24 hours with pseudocornea and anterior staphyloma formation **(Fig 3.73.3)**
- Xerophthalmic scar (Xs)
  - healed sequelae of prior cornea involvement
  - typically inferior in location **(Fig 3.73.4)**
  - includes nebula, macula, leukoma, adherent leukoma, etc. **(Fig 3.73.5)**
- *Treatment:*
  - a medical emergency
  - massive dose of vitamin A
  - treating malnutrition and underlying systemic illness
  - prophylaxis vitamin A for the community

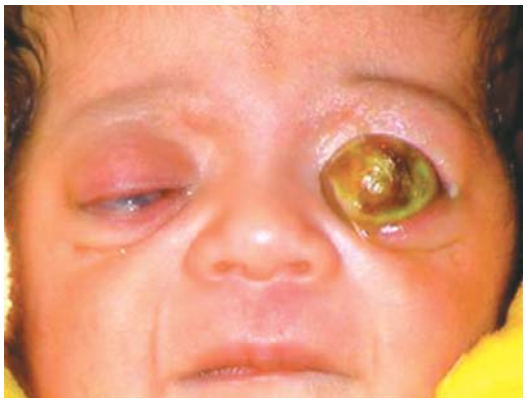


FIGURE 3.73.3: Keratomalacia RE and pseudocornea with anterior staphyloma LE

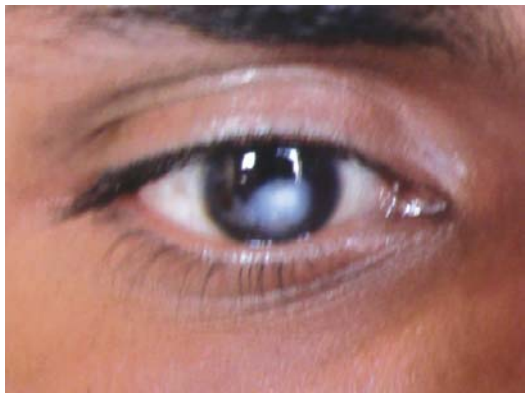


FIGURE 3.73.4: Xerophthalmic scar—Xs

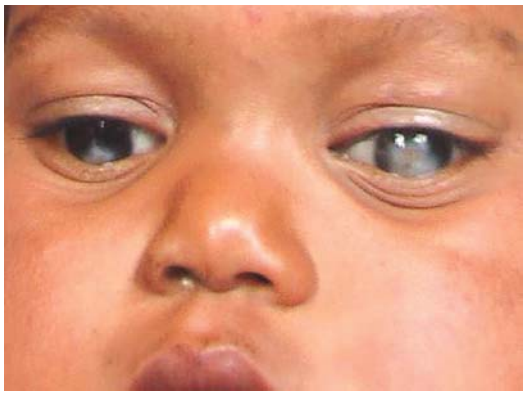


FIGURE 3.73.5: Xerophthalmic scar—Xs

Keratitis Medicomentosa

- Not so uncommon, due to preservatives or drug itself
- Initial presentation is superficial punctate keratitis, scattered all over the cornea **(Fig 3.74.1)**
- Later, patient may present with dry eye with other signs **(Figs 3.74.2 and 3.74.3)**
- *Treatment:* withdrawl of specific drug, preservative-free artificial tears

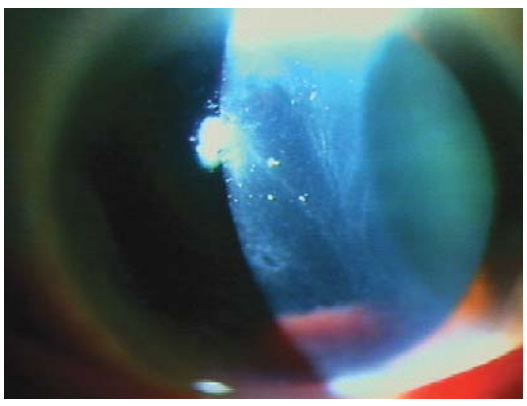


FIGURE 3.74.1: Keratitis medicomentosa

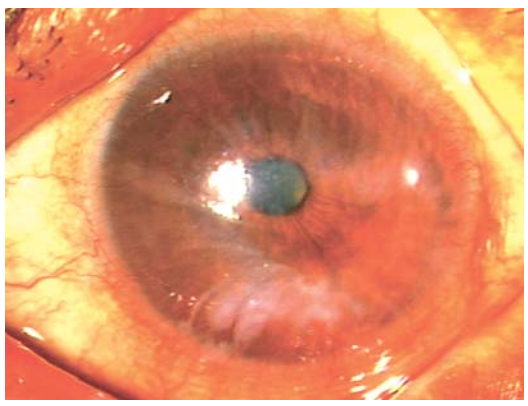


FIGURE 3.74.2: Keratitis medicomentosa—dry eye

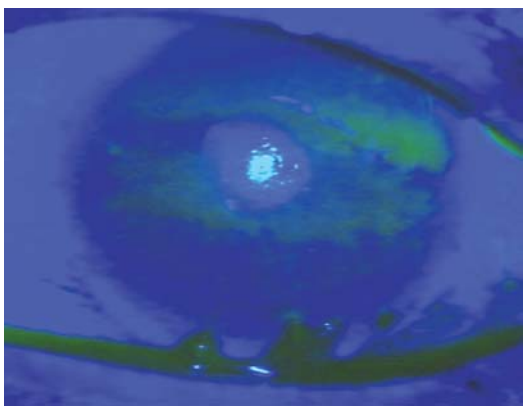


FIGURE 3.74.3: Keratitis medicomentosa—dry eye

Iridocorneal Endothelial (ICE) Syndrome

- Cornea is mainly affected in Chandler syndrome
- Endothelium is replaced by Descemet's membrane like material causing corneal edema and secondary glaucoma ***(See Chapter: 9)***

Tunnel Abscess

- Associated with infiltrations within the tunnel in phacoemulsification surgery **(Fig 3.75.1)**
- Variable degree of anterior chamber reactions
- May progress into frank endophthalmitis **(Fig 3.75.2)**
- *Treatment:* exploration of tunnel, scooping out the materials for culture and sensitivity, and tunnel wash with vancomycin or amphotericin B
- Prognosis is usually poor

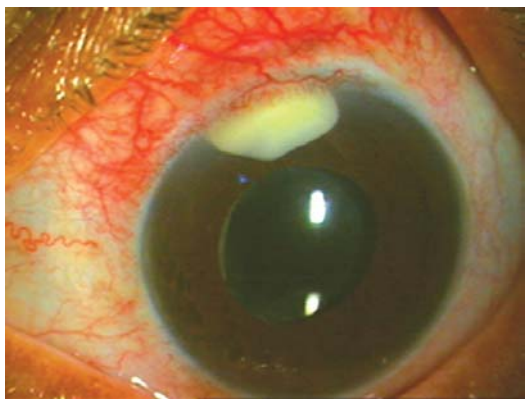


FIGURE 3.75.1: Tunnel abscess—post-phacoemulsification

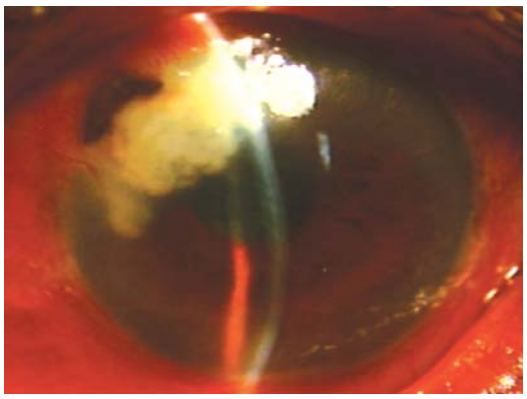


FIGURE 3.75.2: Tunnel abscess—post-cataract surgery

Sclerocorneal Cyst

- Very rare, unilateral condition, occurs after a minor trauma
- Intralamellar sclerocorneal cysts which contains turbid fluid **(Fig 3.76.1)**, and may cause *pseudohypopyon* **(Figs 3.76.2 and 3.76.3)**
- *Treatment:* lamelar dissection to remove the cyst with or without lamellar keratoplasty

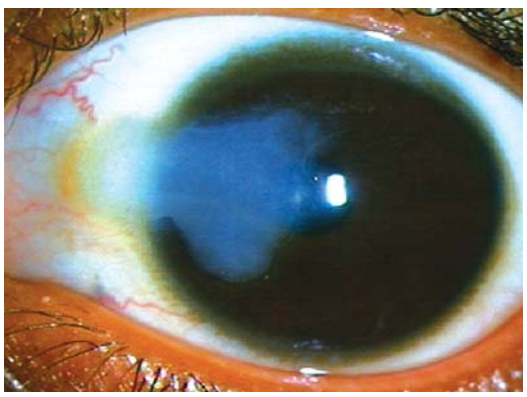


FIGURE 3.76.1: Sclerocorneal cyst

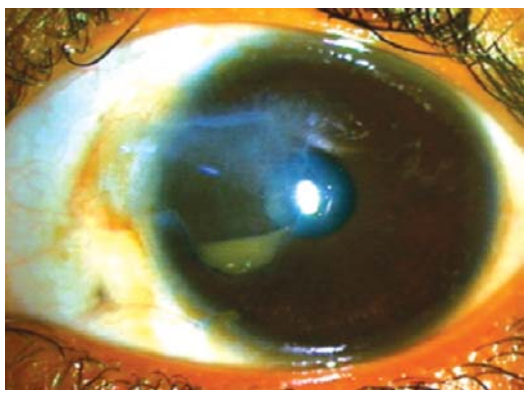


FIGURE 3.76.2: Sclerocorneal cyst

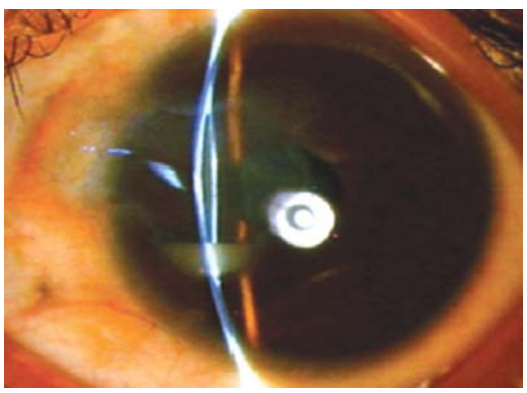


FIGURE 3.76.3: Intracorneal pseudohypopyon

Infectious Crystalline Keratopathy

- Uncommon, but specific presentation
- Usual organism is *Streptococcus viridans*, rarely by fungus
- *Predisposing factors:* prolonged topical corticosteroids and penetrating keratoplasty
- Linear white branching crystalline deposits in the anterior stroma without inflammation **(Fig 3.77.1)**
- Should be differentiated from *ciprofloxacin deposits* during treatment of corneal ulcer **(Fig 3.77.2)**
- *Treatment:* careful scraping of the deposits and proper antibiotics

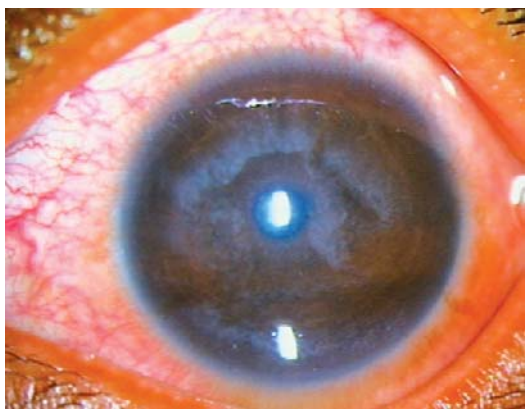


FIGURE 3.77.1: Crystalline kerotopathy

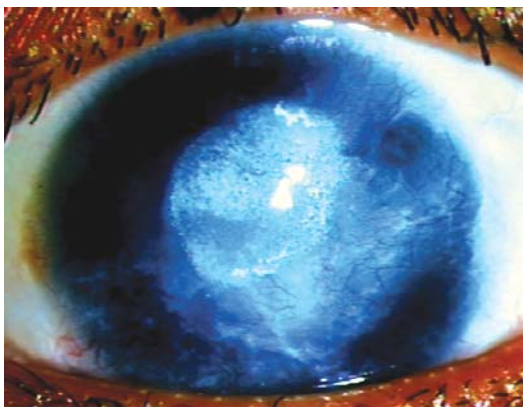


FIGURE 3.77.2: Ciprofloxacin deposits

Corneal Tumors

- Primary corneal tumors are very rare
- Mainly they are the extension of conjunctival tumors, like—
  - limbal dermoid which may cover almost whole cornea, called *central dermoid* **(Fig 3.78.1)**
  - multiple limbal dermoid **(Fig 3.78.2)**
  - invasive squamous cell carcinoma of the conjunctiva **(Fig 3.78.3)**
  - pigmented lesion of the conjunctiva **(Fig 3.78.4)**
- *Treatment:* excision along with conjunctival mass

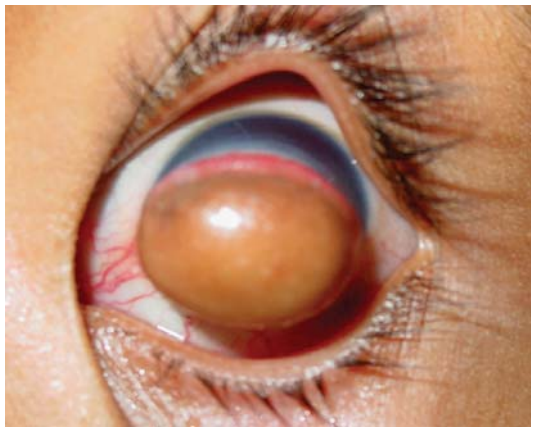


FIGURE 3.78.1: Central corneal dermoid

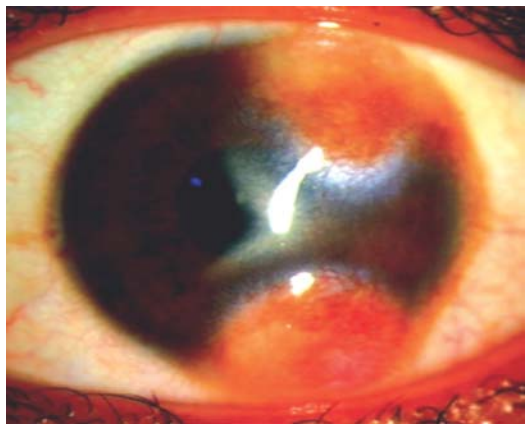


FIGURE 3.78.2: Limbal dermoid on cornea

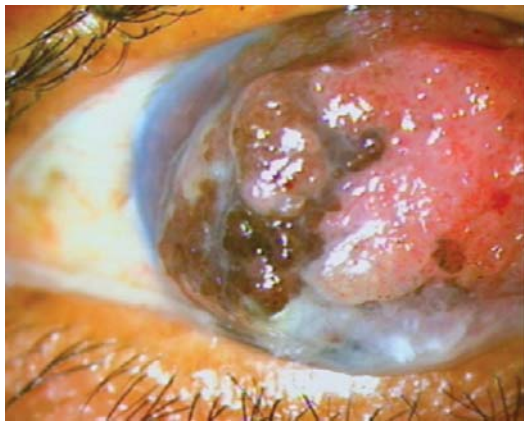


FIGURE 3.78.3: Extension of conjunctival squamous cell carcinoma

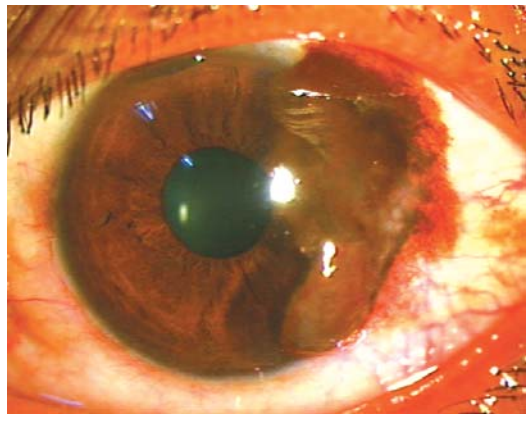


FIGURE 3.78.4: Conjunctival melanoma on cornea

4 Diseases of the Sclera

BENIGN CONDITIONS OF THE SCLERA

- Blue sclera
- Focal discoloration of sclera

INFLAMMATION OF THE SCLERA

- Episcleritis
- Scleritis
- Anterior scleritis
- Anterior necrotizing scleritis with inflammation
- Anterior necrotizing scleritis without inflammation (scleromalacia perforans)
- Posterior scleritis
- Scleral abscess
- Surgically induced necrosis of the sclera (SINS)

BENIGN CONDITIONS OF THE SCLERA

Blue Sclera

- Sclera appears more blue than white
- Due to increased visibility of the underlying uveal pigment through the thinned sclera **(Fig 4.1.1)**
- *Causes:* osteogenesis imperfecta, buphthalmos **(Fig 4.1.2)**, keratoglobus, congenital high myopia, following diffuse scleritis **(Fig 4.1.3),** oculodermal melanocytosis **(Fig 4.1.4)** ciliary staphyloma, Marfan's and Ehler-Danlos syndrome

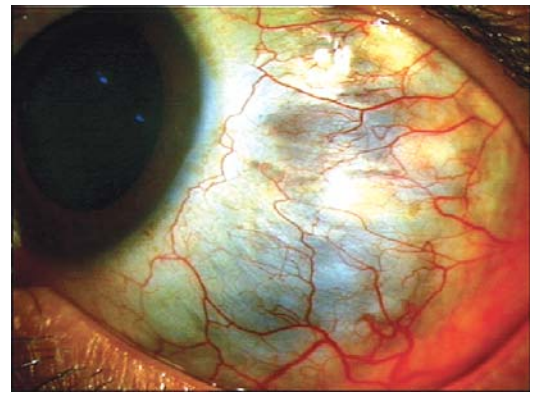


FIGURE 4.1.1: Blue sclera

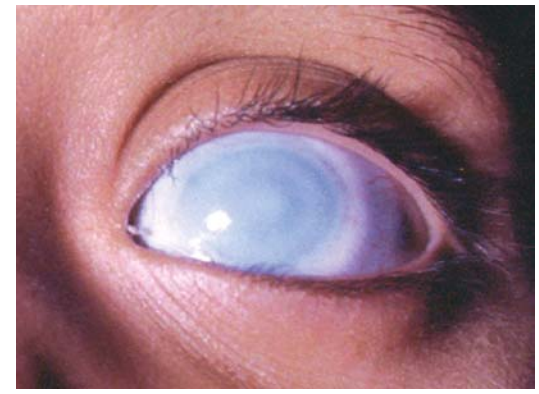


FIGURE 4.1.2: Blue sclera—buphthalmos

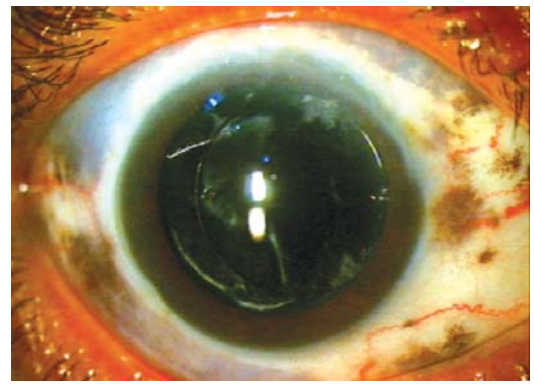


FIGURE 4.1.3: Blue sclera—diffuse scleritis

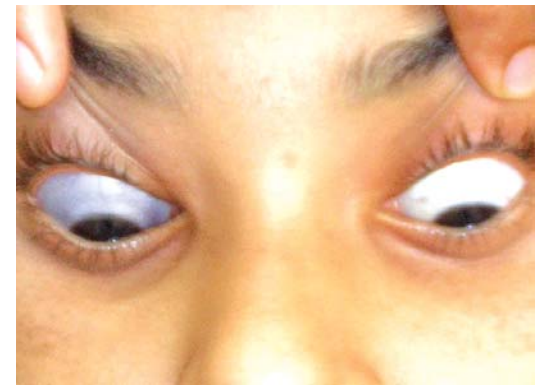


FIGURE 4.1.4: Blue sclera—oculodermal melanocytosis

Focal Discoloration of the Sclera

- Localized discoloration (blue or brown-black) of the sclera seen in variety of conditions
- *Causes:* healed focal scleritis **(Fig 4.2.1)**, equatorial staphyloma **(Fig 4.2.2)**, long-standing metallic foreign body, alkaptonuria (pigmentation at the insertion of horizontal recti) **(Fig 4.2.3)** or in extreme old age **(Fig 4.2.4)**

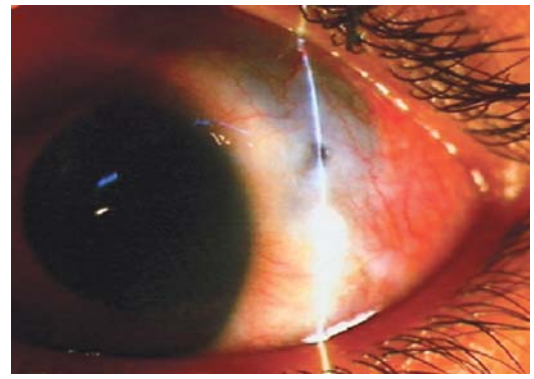


FIGURE 4.2.1: Focal discoloration—healed scleritis

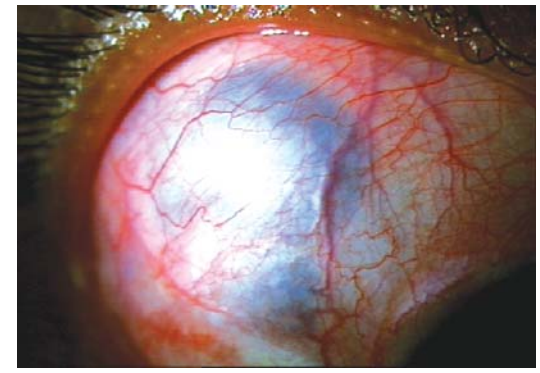


FIGURE 4.2.2: Focal discoloration—equatorial staphyloma

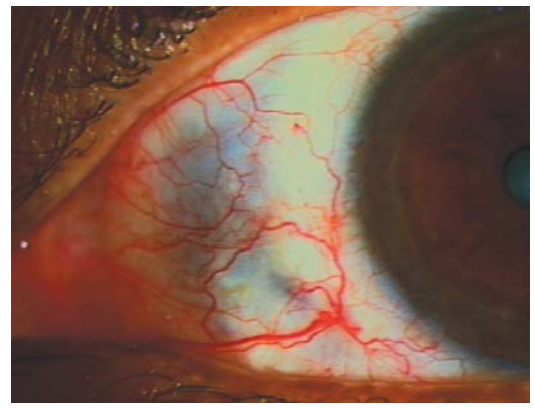


FIGURE 4.2.3: Focal discoloration—alkaptonuria

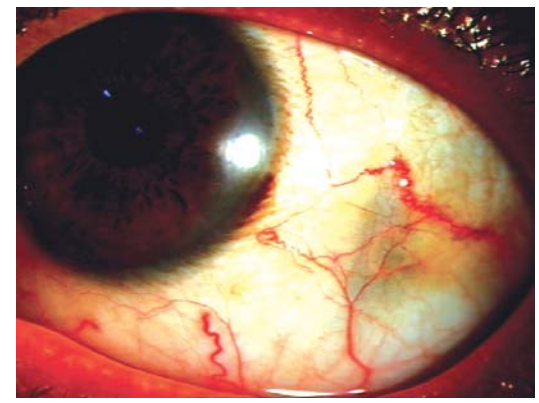


FIGURE 4.2.4: Focal discoloration—old age

INFLAMMATION OF THE SCLERA

Episcleritis

- Benign inflammation of the episcleral tissue, not so serious

May be *simple* or *nodular*

- *Simple episcleritis*:
  - sectorial or diffuse redness involving the middle episcleral vessels (**Figs 4.3.1 and 4.3.2)**
  - blanching effect on instillation of phenylepinephrine
- *Nodular episcleritis:*
  - purple solitary nodule with surrounding injection which can be moved over the sclera
  - situated 2-3 mm away from the limbus **(Fig 4.3.3)**
- *Treatment:* oral anti-inflammatory agents, dilute topical corticosteroid, or non-steroidal eye drops.

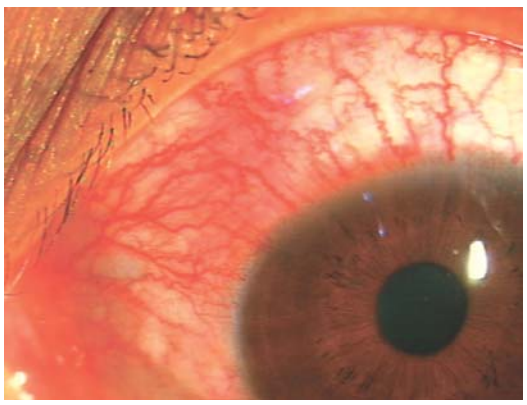


FIGURE 4.3.1: Simple episcleritis

FIGURE 4.3.2: Simple episcleritis

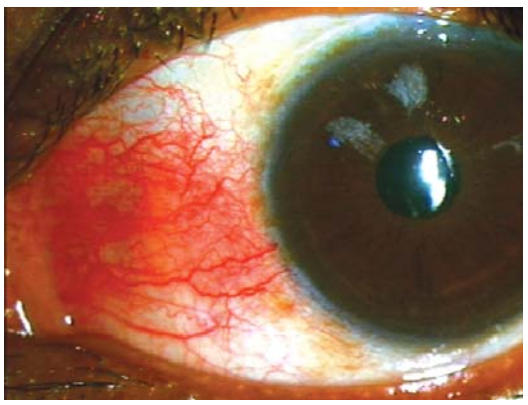


FIGURE 4.3.3: Nodular episcleritis

SCLERITIS

Anterior Scleritis

- Inflammation of the sclera which is more serious than episcleritis
- Often bilateral and frequently in women
- Associated with systemic collagen disorders in 50% of cases
- No blanching on phenylepinephrine
- It may be *diffuse*, *nodular* or *necrotizing*
- *Diffuse anterior scleritis:*
  - involves either a segment or the entire anterior sclera **(Fig 4.4.1)**
  - diffuse redness and distortion of pattern of deep episcleral vascular plexus
  - variable episcleral and conjunctival congestion **(Fig 4.4.2)**
- *Nodular scleritis (non-necrotizing):*
  - extremely tender, usually solitary **(Fig 4.5.1)** or multiple **(Figs 4.5.2 and 4.5.3)** firm immobile nodule separated from the overlying congested episcleral tissue

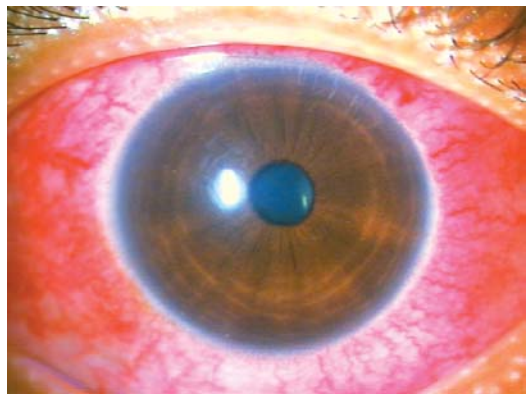


FIGURE 4.4.1: Diffuse anterior scleritis

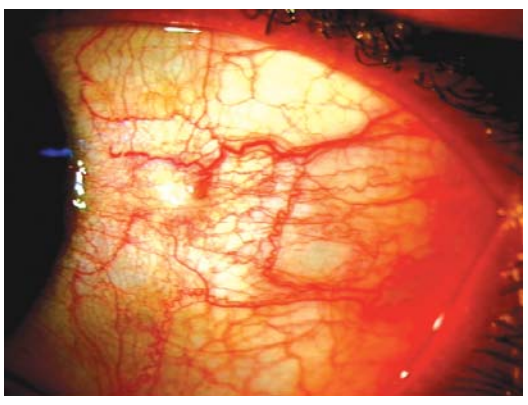


FIGURE 4.4.2: Diffuse anterior scleritis

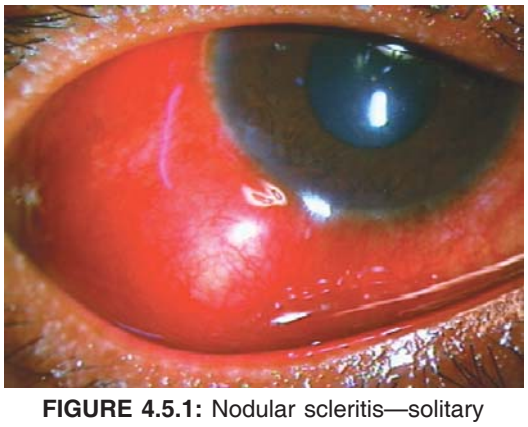


FIGURE 4.5.1: Nodular scleritis—solitary

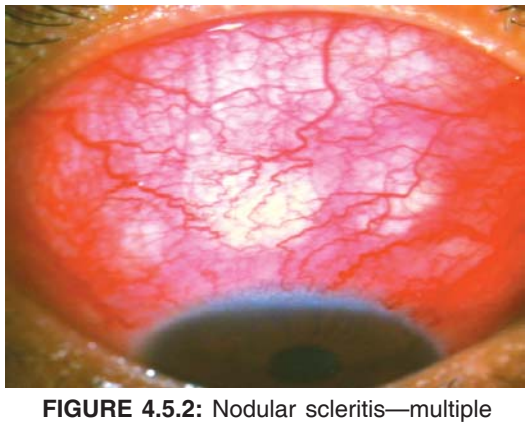


FIGURE 4.5.2: Nodular scleritis—multiple

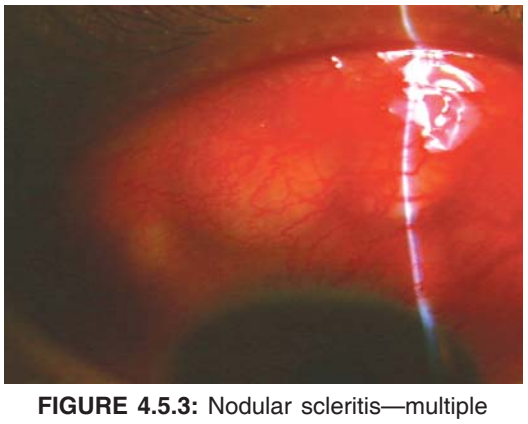


FIGURE 4.5.3: Nodular scleritis—multiple

- *Anterior necrotizing scleritis with inflammation:*
  - avascular patches appearing in the episcleral tissue with scleral necrosis and melting **(Fig 4.6.1)**
  - marked thinning of the sclera with increased visibility of underlying uvea **(Fig 4.6.2)**
  - associated anterior uveitis and severe keratitis in some cases **(Fig 4.6.3)**
- *Anterior necrotizing scleritis without inflammation (scleromalacia perforans):*
  - classically seen in patient with longstanding rheumatoid arthritis
  - painless, and starts as a white necrotic patch in the normal sclera
  - eventually, extreme scleral thinning with exposure and bulging of underlying uvea **(Fig 4.7.1)**
- *Treatment:* systemic and topical corticosteroids, non-steroidal anti-inflammatory agents, immunosuppressive (antimetabolites) in severe and unresponsive cases; and investigations for systemic collagen disorders.

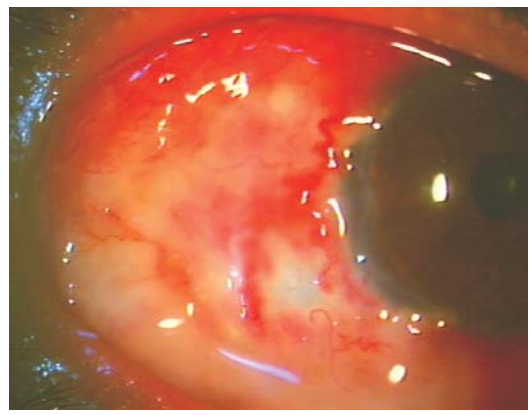


FIGURE 4.6.1: Necrotizing scleritis with inflammation

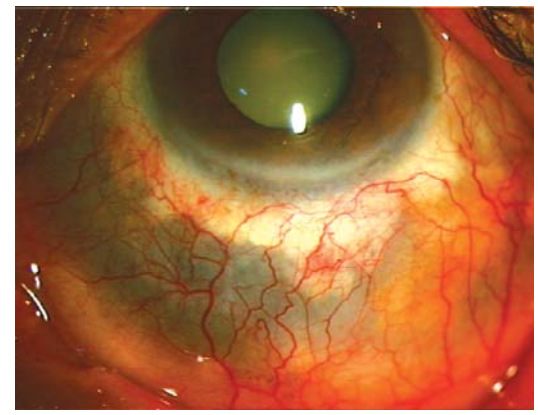


FIGURE 4.6.2: Necrotizing scleritis—thinning

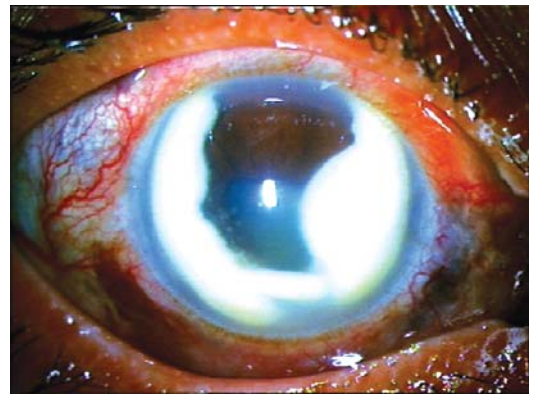


FIGURE 4.6.3: Necrotizing scleritis—associated keratitis and uveitis

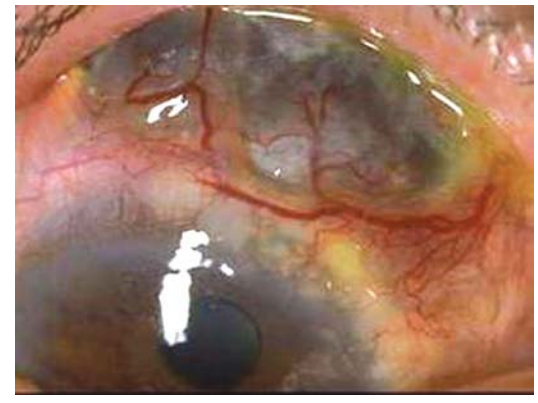


FIGURE 4.7.1: Scleromalacia perforans

Posterior Scleritis

- The signs depend upon site of maximum involvement primarily
- May be associated with anterior scleritis
- Inward extension gives rise to '*uveal effusion syndrome*'—choroiditis, choroidal effusion, exudative retinal detachment, macular edema **(Fig 4.8.1)**
- Outward extension into the orbit gives rise to proptosis with extraocular muscle involvement
- *Treatment:* systemic and topical corticosteroids, non-steroidal anti-inflammatory agents; immunosuppressive (antimetabolites) in severe and unresponsive cases

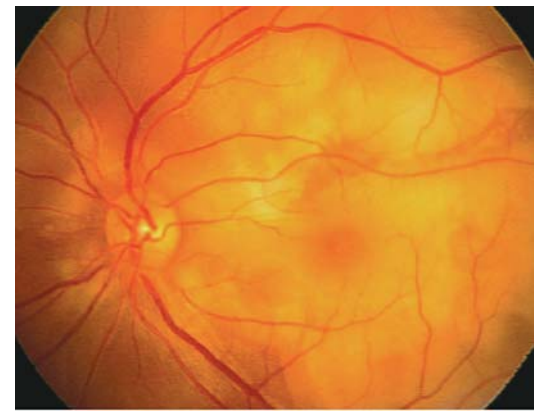


FIGURE 4.8.1: Posterior scleritis

Scleral Abscess

- Rare, unilateral condition
- Usually traumatic, but may rarely occur in tuberculous infection
- Nodular yellowish-white lesion, with mild to moderate pain **(Fig 4.9.1)** and localized congestion
- *Treatment:* simple incision and drainage of abscess with culture sensitivity and antibiotics

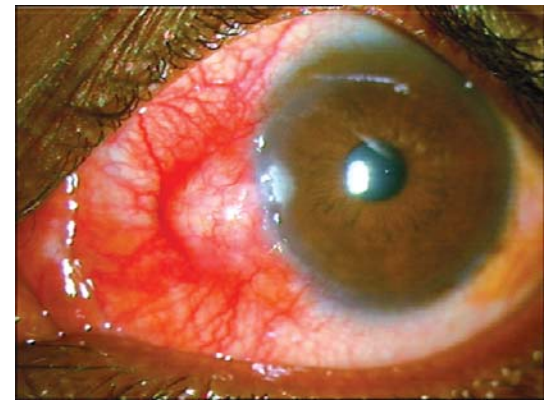


FIGURE 4.9.1: Scleral abscess

Surgically Induced Necrosis of the Sclera (SINS)

- Rare postoperative immune-mediated necrosis of sclera
- May be triggered by excessive cautery, use of antimetabolities during surgery, etc.
- *May be noticed after:*
  - cataract surgery **(Figs 4.10.1 and 4.10.2)**
  - pterygium surgery **(Figs 4.10.3 to 4.10.5)**
  - vitreoretinal surgery **(Fig 4.10.6)**
- *Treatment:* topical and systemic steroids, systemic immune-suppressants and scleral patch graft in extreme situation

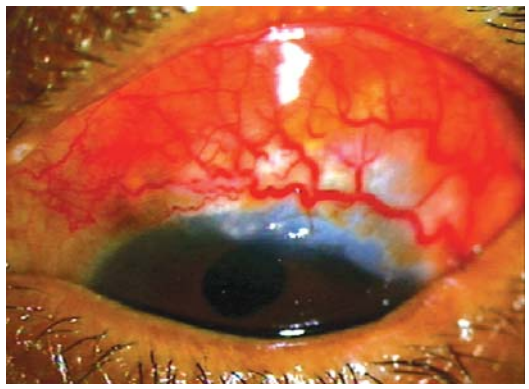


FIGURE 4.10.1: Surgically induced necrosis of sclera

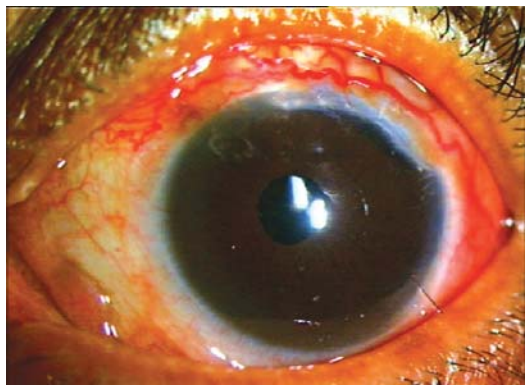


FIGURE 4.10.2: SINS—post-cataract surgery

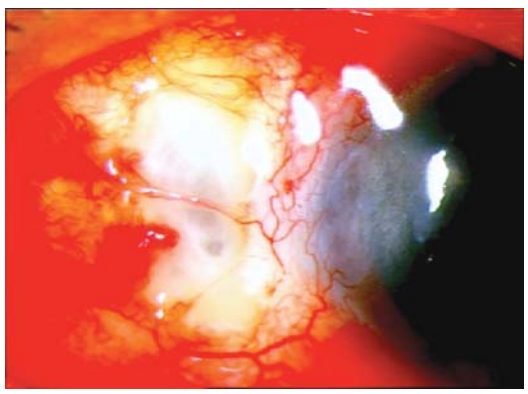


FIGURE 4.10.3: SINS—post-pterygium surgery

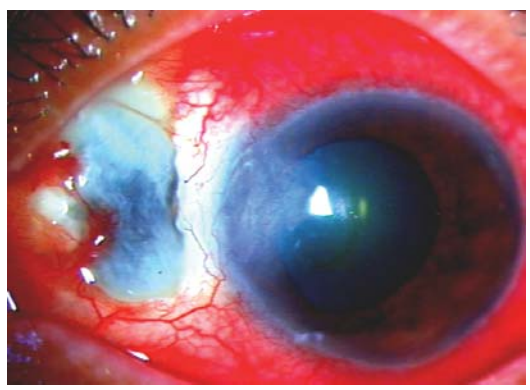


FIGURE 4.10.4: SINS—post-pterygium surgery

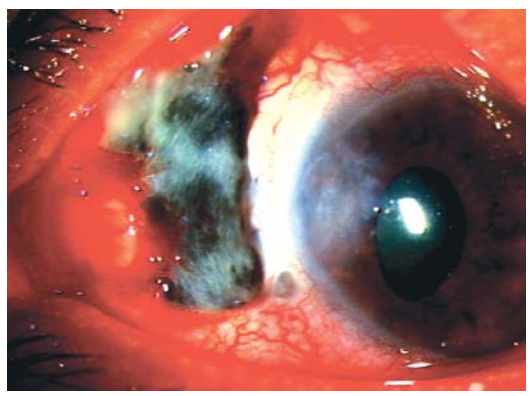


FIGURE 4.10.5: SINS—post-pterygium surgery

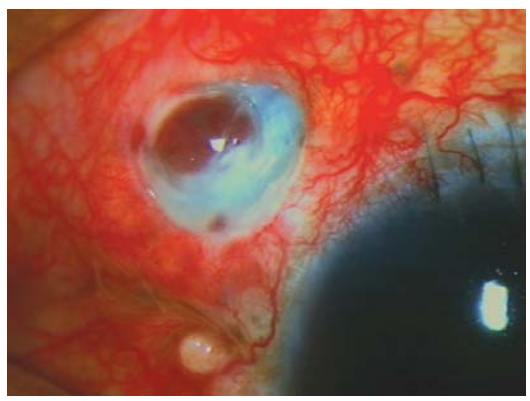


FIGURE 4.10.6: SINS—post-VR surgery

5 Abnormalities in the Anterior Chamber

ANTERIOR CHAMBER

ABNORMAL DEPTH

- Shallow anterior chamber
- Deep anterior chamber
- Irregular anterior chamber

ABNORMAL CONTENTS

- Blood (hyphema)
- Pus (hypopyon)
- Pseudohypopyon
- Albuminous materials
- Other abnormal contents

ANTERIOR CHAMBER

- Space between the cornea and the iris
- Contains crystal clear aqueous humor
- Central depth is approximately 2.5: mm, and uniformly and gradually shallower towards periphery **(Fig 5.1.1)**
- Depth varies in normal condition and also in diseases **(Figs 5.1.2 to 5.1.4)**

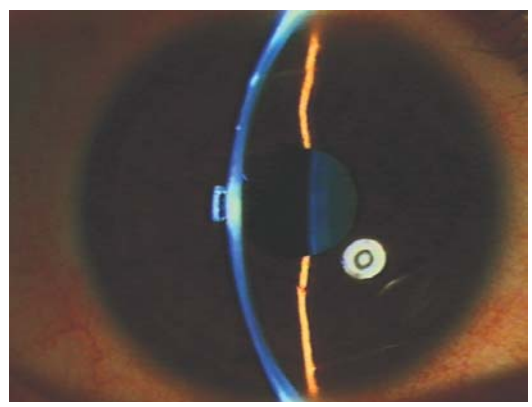


FIGURE 5.1.1: Normal depth AC

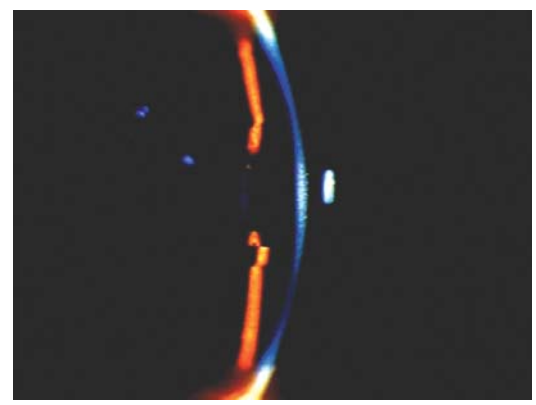


FIGURE 5.1.2: Mild shallow AC

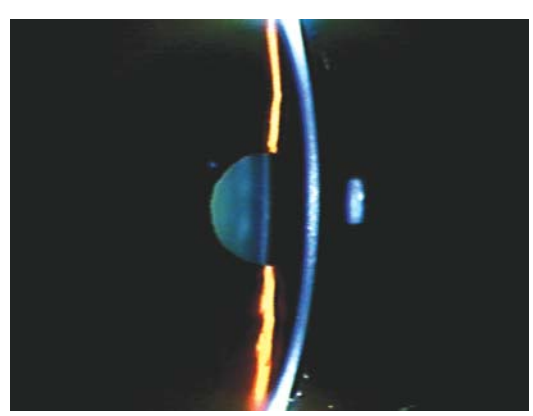


FIGURE 5.1.3: Moderately shallow AC

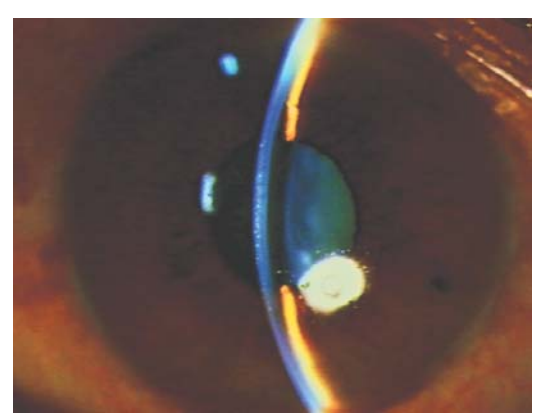


FIGURE 5.1.4: Very shallow AC

ABNORMAL DEPTH

Shallow Anterior Chamber

- *Causes:*
  - cornea plana **(Fig 5.2.1)**
  - hypermetropia **(Fig 5.2.2)**
  - intumescent cataract **(Fig 5.2.3)**
  - pupillary block glaucoma **(Figs 5.2.4 and 5.2.5)**
  - over filtration of filtering bleb **(Fig 5.2.6)**
  - choroidal detachment
  - wound leak after intraocular surgery
  - penetrating injury **(Fig 5.2.7)**
  - malignant glaucoma **(Fig 5.2.8)**

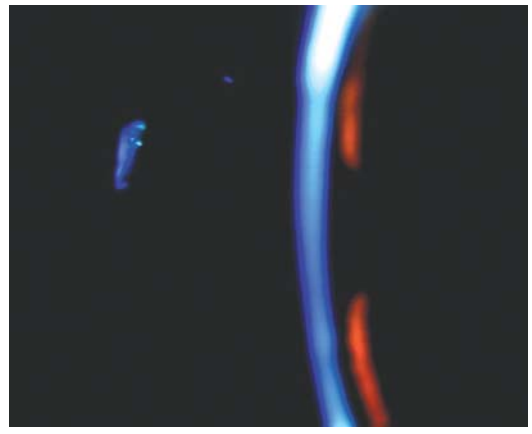


FIGURE 5.2.1: Shallow AC—cornea plana

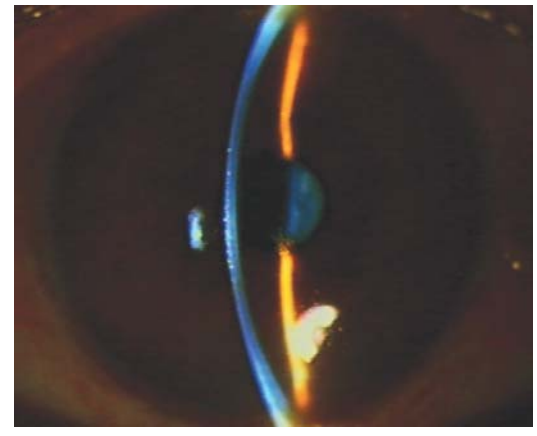


FIGURE 5.2.2: Shallow AC—hypermetropia

FIGURE 5.2.3: Shallow AC—intumescent cataract

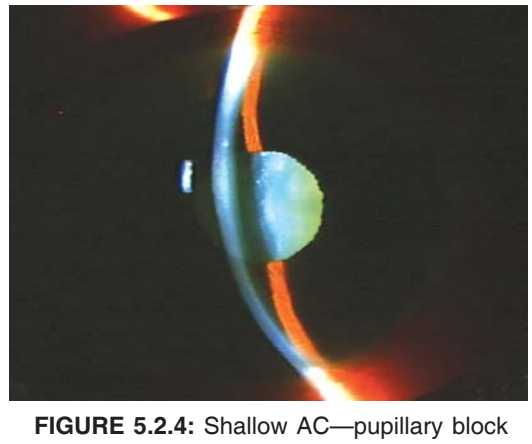


FIGURE 5.2.4: Shallow AC—pupillary block glaucoma

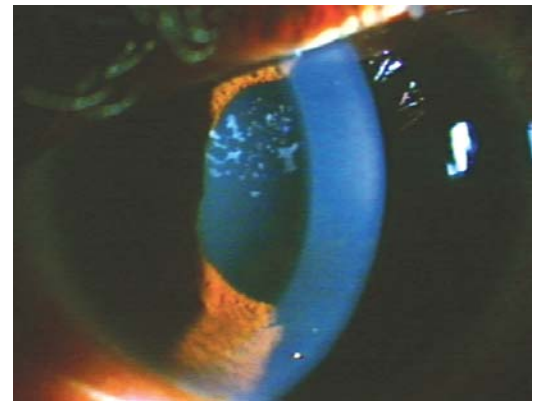


FIGURE 5.2.5: Shallow AC—pupillary block glaucoma

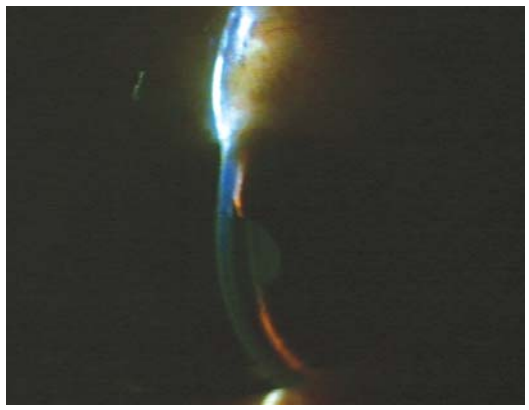


FIGURE 5.2.6: Shallow AC

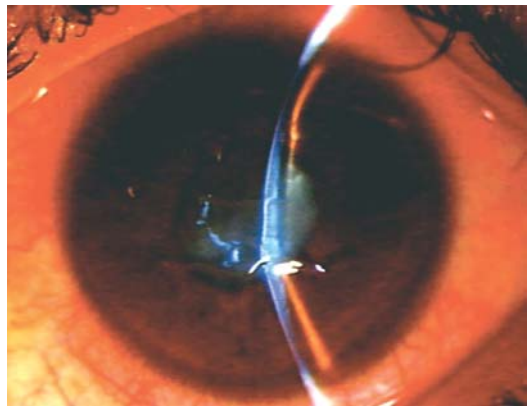


FIGURE 5.2.7: Shallow AC penetrating injury

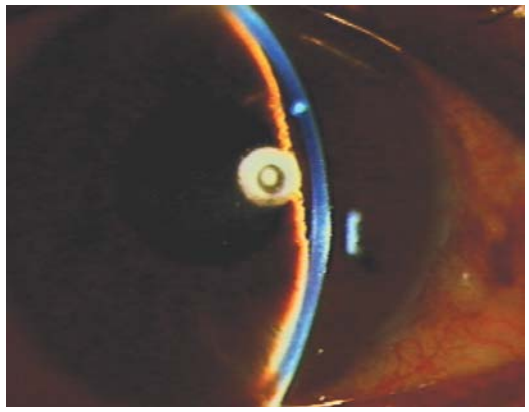


FIGURE 5.2.8: Shallow AC—malignant glaucoma

Overfiltering Bleb
Deep Anterior Chamber

- *Causes:*
  - myopia **(Fig 5.3.1)**
  - aphakia, pseudophakia **(Fig 5.3.2)**
  - megalocornea **(Fig 5.3.3)**
  - keratoglobus, keratoconus **(Fig 5.3.4)**
  - buphthalmous
  - posterior dislocation of the lens

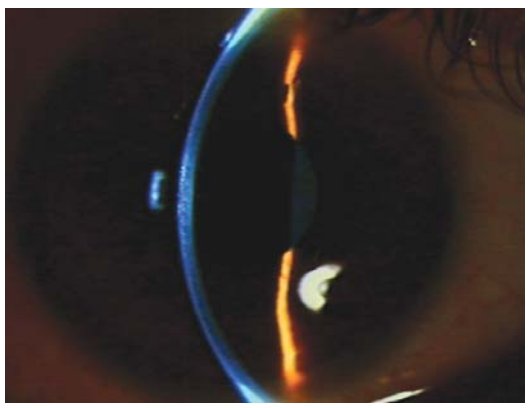


FIGURE 5.3.1: Deep AC—myopia

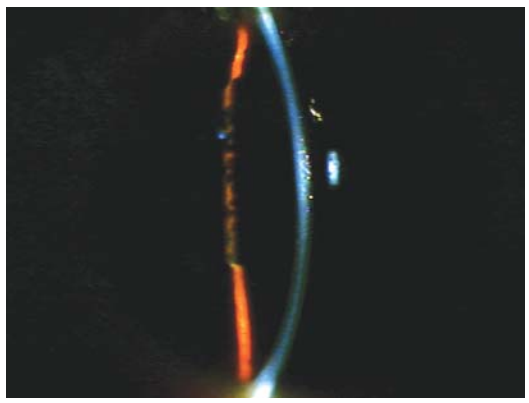


FIGURE 5.3.2: Deep AC—pseudophakia

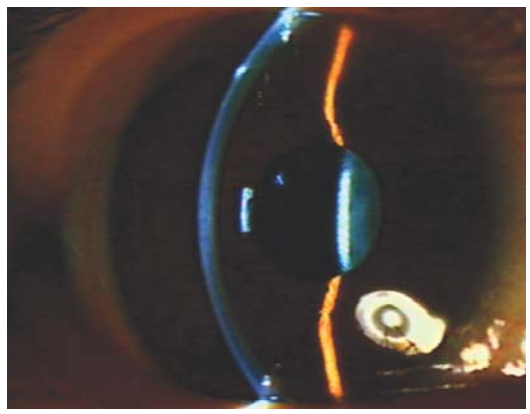


FIGURE 5.3.3: Deep AC—megalocornea

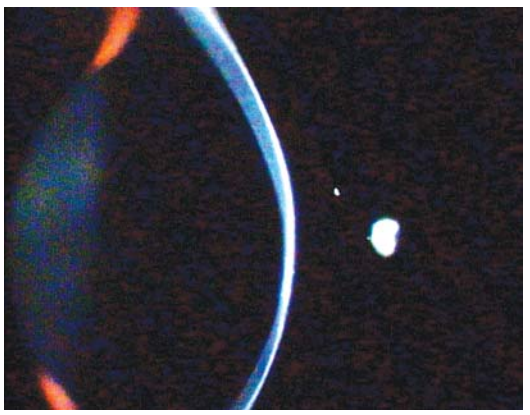


FIGURE 5.3.4: Deep AC—keratoconus

Irregular Anterior Chamber

- *Causes:*
  - ectopia lentis **(Figs 5.4.1 and 5.4.2)**
  - iris bombe: funnel shaped **(Figs 5.4.3 and 5.4.4)**
  - adherent leucoma
  - iris cyst or tumor **(Fig 5.4.5)**
  - penetrating injury with iris prolapse **(Fig 5.4.6)**
  - angle recession
  - pellucid marginal degeneration **(Fig 5.4.7).**

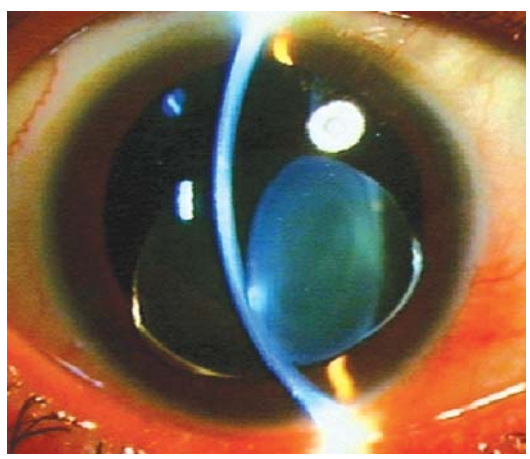


FIGURE 5.4.1: Irregular AC—ectopia lentis

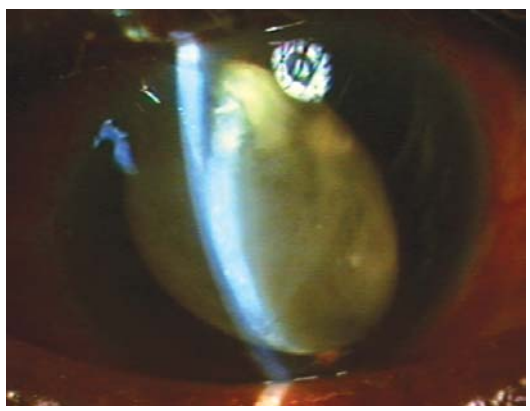


FIGURE 5.4.2: Irregular AC—subluxated lens

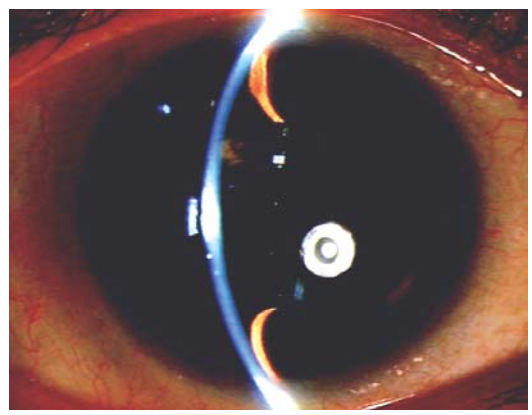


FIGURE 5.4.3: Funnel shaped AC—iris bombe

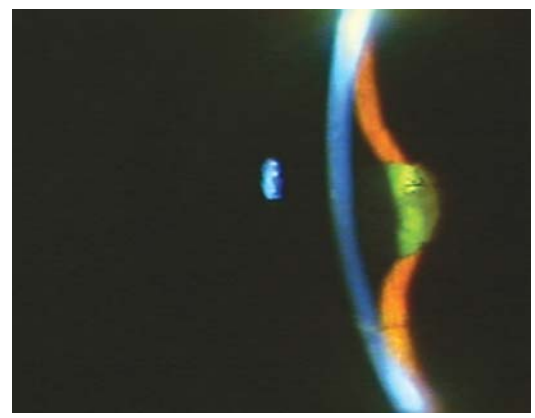


FIGURE 5.4.4: Funnel shaped AC —iris bombe

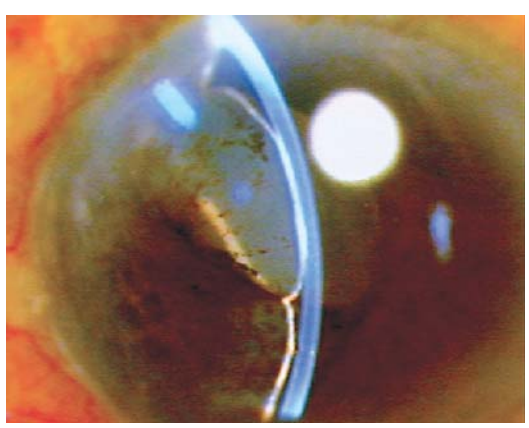


FIGURE 5.4.5: Irregular AC—iris cyst

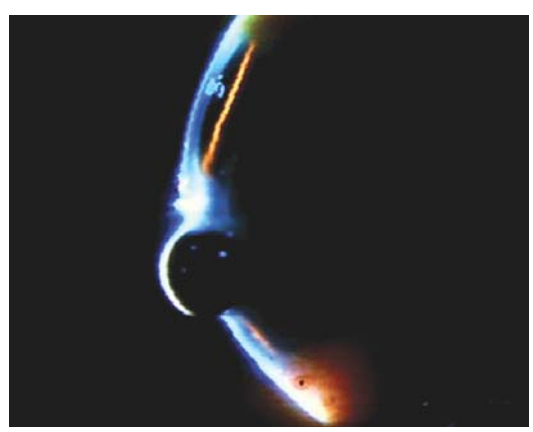


FIGURE 5.4.6: Irregular AC—iris prolase

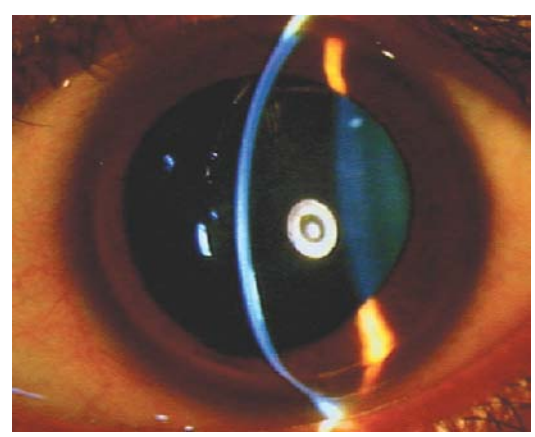


FIGURE 5.4.7: Irregular AC—pellucid merginal degeneration

ABNORMAL CONTENTS

Blood (hyphema)

- Traumatic **(Figs 5.5.1 and 5.5.2)**
- Postoperative **(Fig 5.5.3)**
- Herpetic iridocyclitis
- Spontaneous

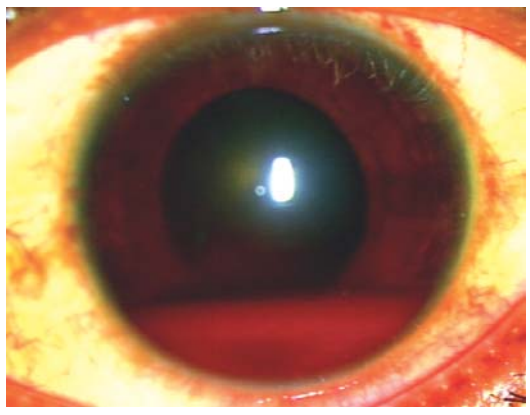


FIGURE 5.5.1: Traumatic hyphema

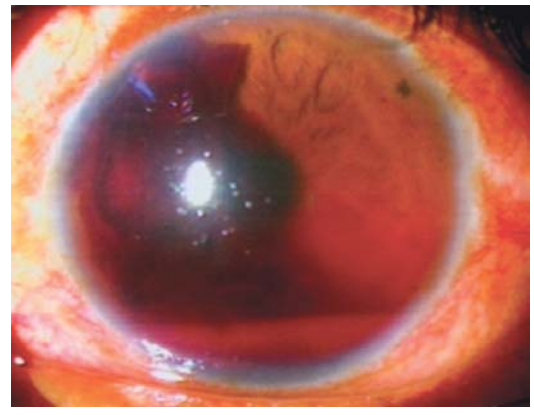


FIGURE 5.5.2: Traumatic—cotted blood

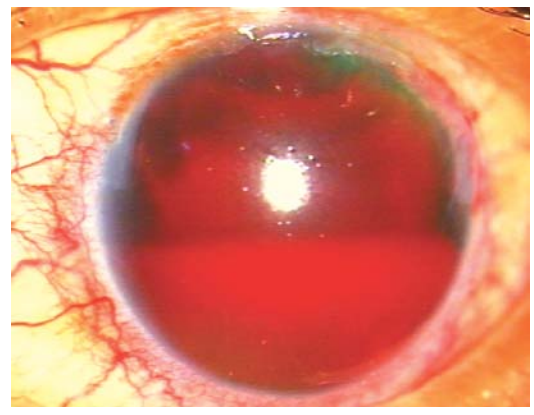


FIGURE 5.5.3: Postoperative hyphema

Pus (hypopyon)

- Corneal ulcer **(Figs 5.6.1 to 5.6.3)**
- Acute iridocyclitis **(Fig 5.6.4)**
- Endophthalmitis **(Figs 5.6.5 and 5.6.6)**
- Panophthalmitis **(Fig 5.6.7)**

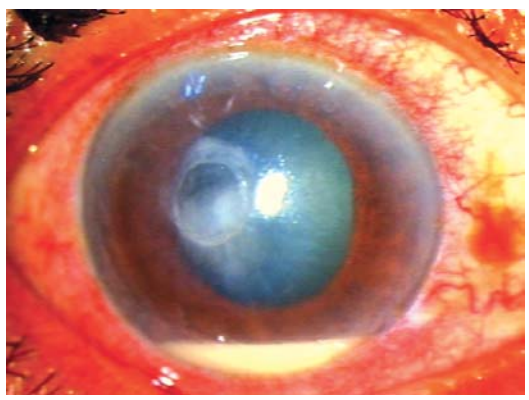


FIGURE 5.6.1: Mild hypopyon—corneal ulcer

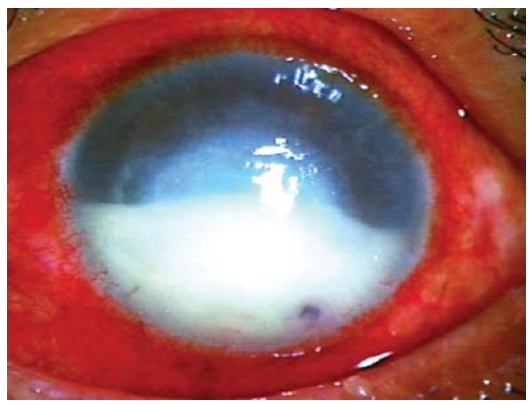


FIGURE 5.6.2: Moderate hypopyon—corneal ulcer

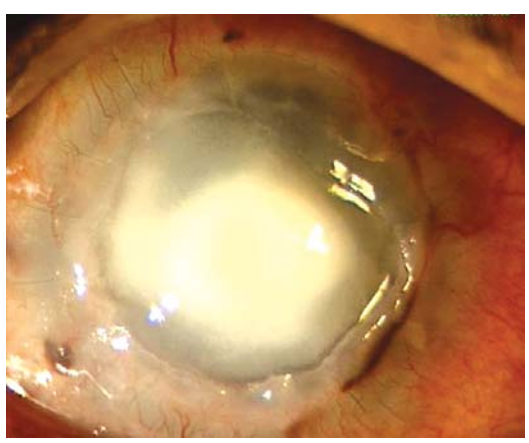


FIGURE 5.6.3: Severe hypopyon—corneal ulcer

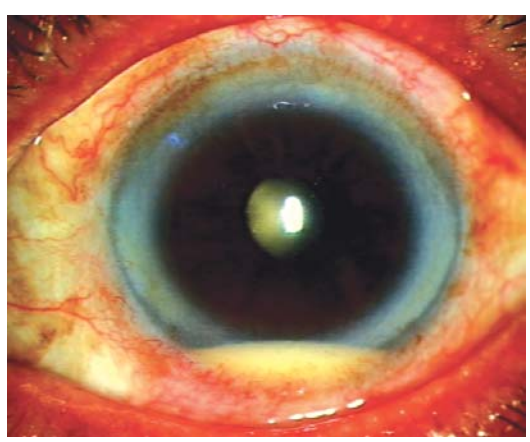


FIGURE 5.6.4: Hypopyon—acute iridocyclitis

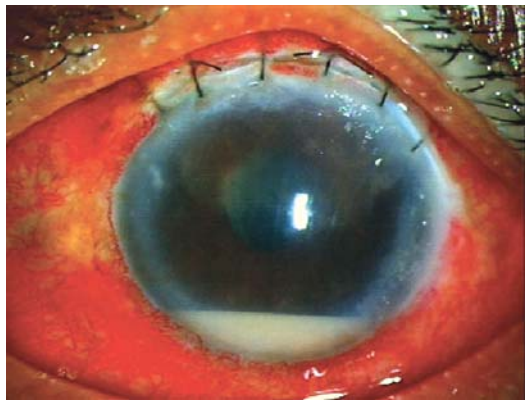


FIGURE 5.6.5: Hypopyon—endophthalmitis

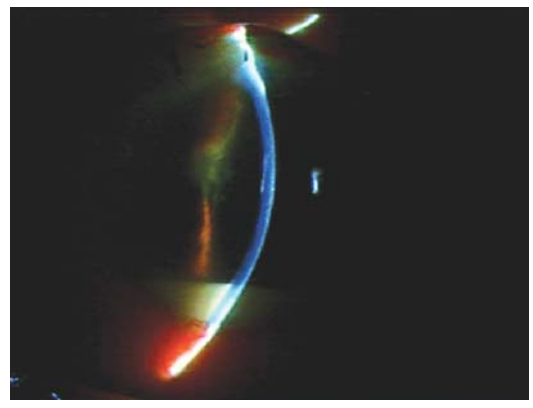


FIGURE 5.6.6: Hypopyon—endophthalmitis

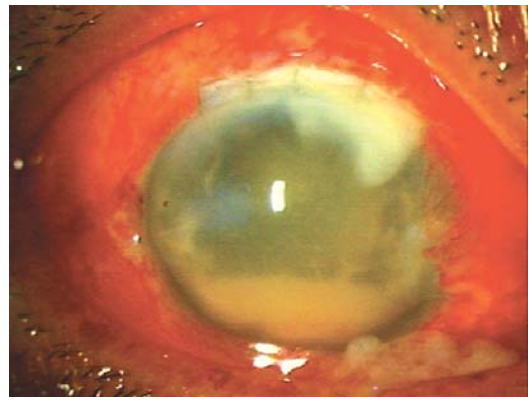


FIGURE 5.6.7: Hypopyon—panophthalmitis

Pseudohypopyon

- Silicone oil—*inverse hypopyon* **(Figs 5.7.1 and 5.7.2)**
- Malignant cells **(Fig 5.7.3)**
- Liquefied milky cortex in hypermature morgagnian cataract **(Fig 5.7.4)**

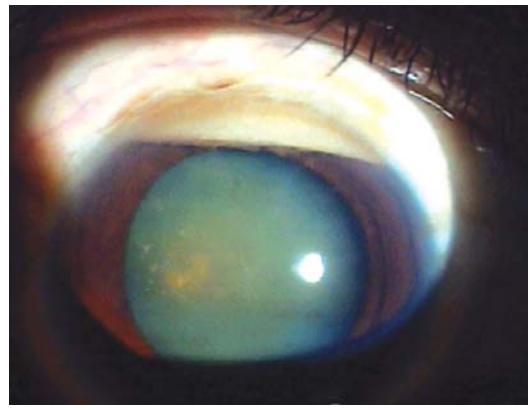


FIGURE 5.7.1: Inverse hypopyon—emulsified silicone oil

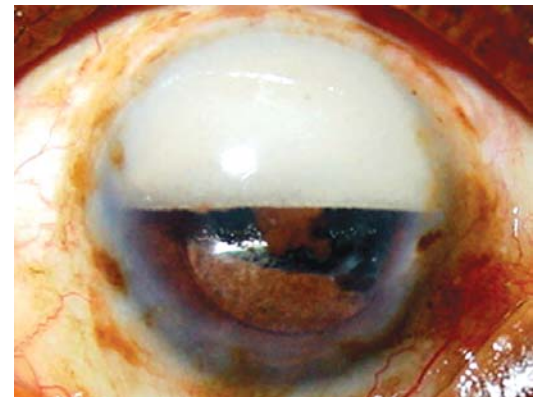


FIGURE 5.7.2: Inverse hypopyon—emulsified silicone oil

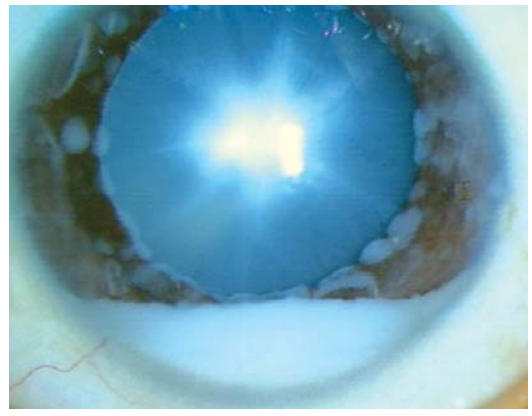


FIGURE 5.7.3: Pseudohypopyon—retinoblastoma

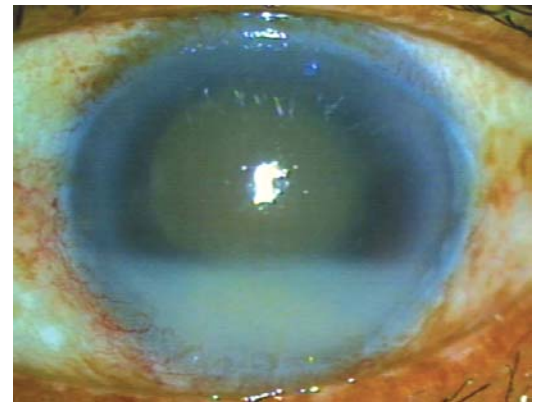


FIGURE 5.7.4: Pseudohypopyon—liquefied cortex

Albuminous Materials

- Aqueous flare in iritis **(Figs 5.8.1 and 5.8.2)**
- Fibrinous exudates in iritis **(Fig 5.8.3)**
- Aqueous cells in iritis **(Fig 5.8.4)**
- Dense exudates in endophthalmitis **(Figs 5.8.5 and 5.8.6)**

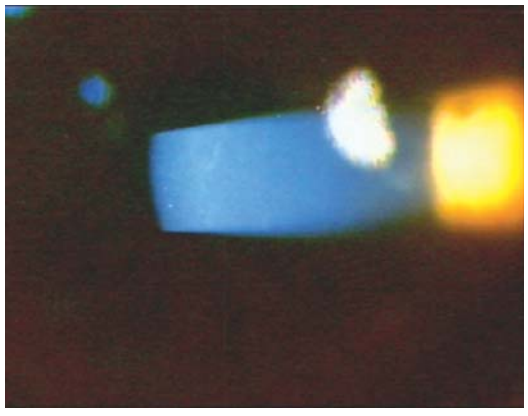


FIGURE 5.8.1: Aqueous flare—iritis

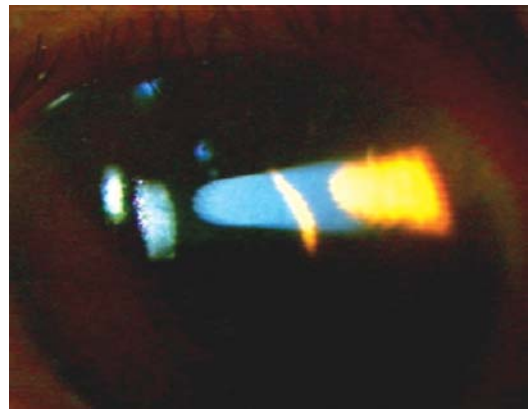


FIGURE 5.8.2: Aqueous flare—iritis

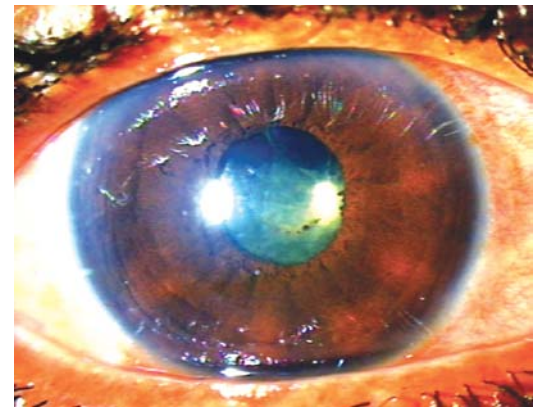


FIGURE 5.8.3: Fibrinous exudate—iritis

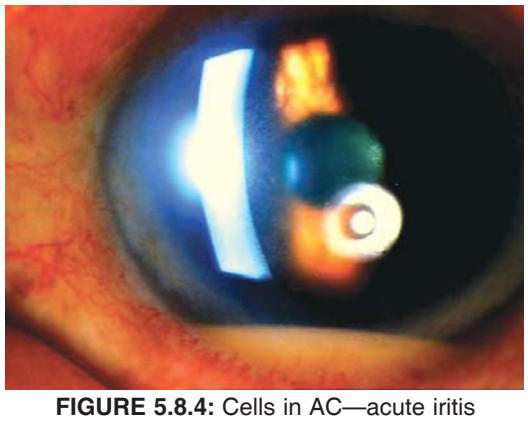


FIGURE 5.8.4: Cells in AC—acute iritis

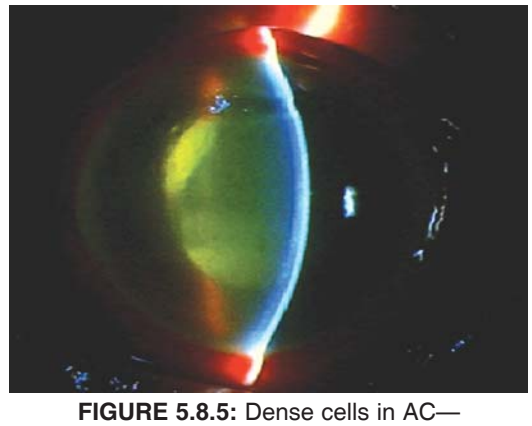


FIGURE 5.8.5: Dense cells in AC—endophthalmitis

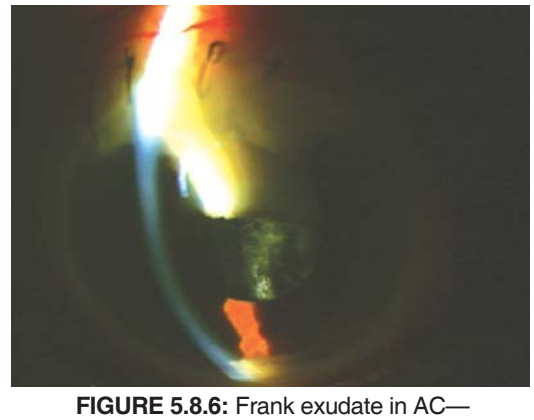


FIGURE 5.8.6: Frank exudate in AC—endophthalmitis

Other Abnormal Contents

- Crystalline lens in AC **(Fig 5.9.1)**
- Anterior chamber IOL **(Fig 5.9.2)**
- Lens cortical matter in AC **(Fig 5.9.3)**
- Lens fragment in AC **(Fig 5.9.4)**
- Vitreous in AC **(Figs 5.9.5 and 5.9.6)**
- Air bubble in AC **(Figs 5.9.7 and 5.9.8)**
- Silicone oil in AC **(Figs 5.9.9 to 5.9.11)**
- Foreign body in AC **(Figs 5.9.12 and 5.9.13)**
- Eyelash in AC **(Fig 5.9.14)**
- Fungal granuloma in AC **(Figs 5.9.15 and 5.9.16)**
- Parasite dead or live **(Figs 5.9.17 to 5.9.19)**
- Cholesterolosis bulbi **(Figs 5.9.20 and 5.9.21)**
- Milky liquefied lens cortex **(Fig 5.9.22)**
- Pseudoexfoliative material **(Fig 5.9.23)**
- Caterpillar hair **(Fig 5.9.24)**
- Epithelial cyst (pearl) in AC **(Fig 5.9.25)**

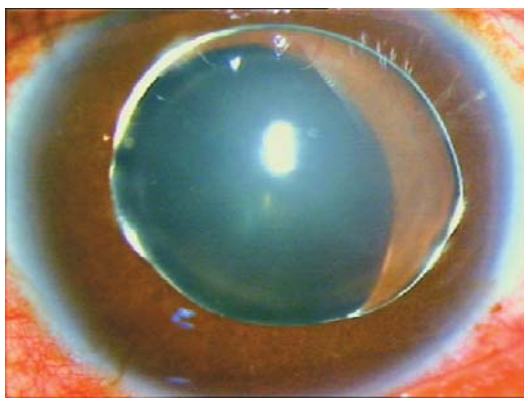


FIGURE 5.9.1: Crystalline lens

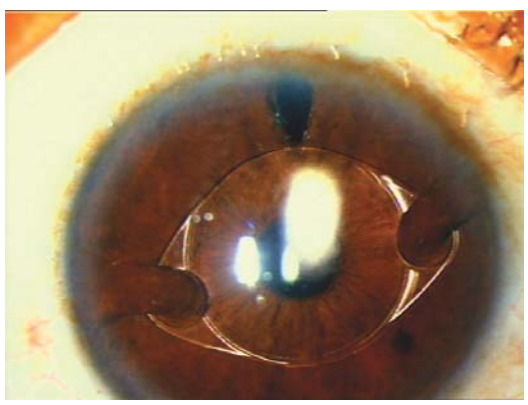


FIGURE 5.9.2: Anterior chamber IOL

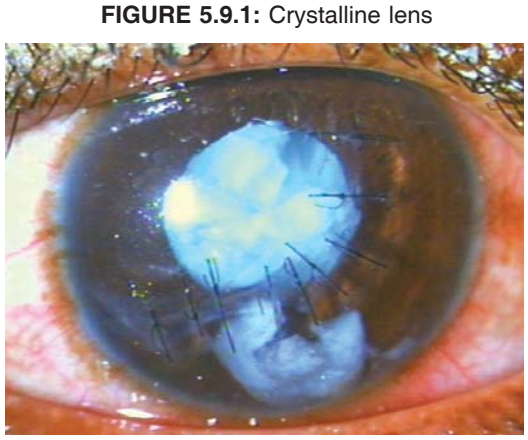


FIGURE 5.9.3: Lens cortex

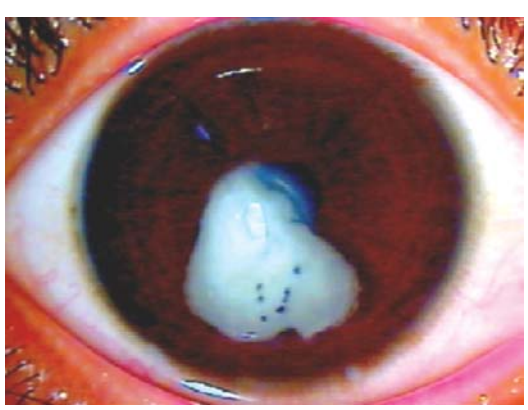


FIGURE 5.9.4: Lens fragment

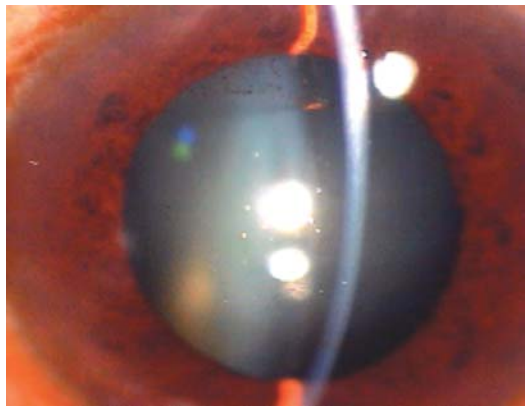


FIGURE 5.9.5: Vitreous in subluxation

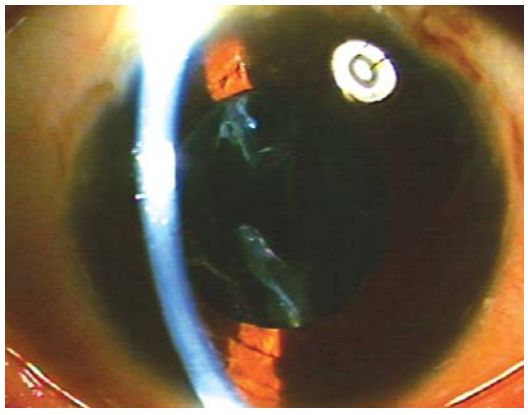


FIGURE 5.9.6: Vitreous in aphakia

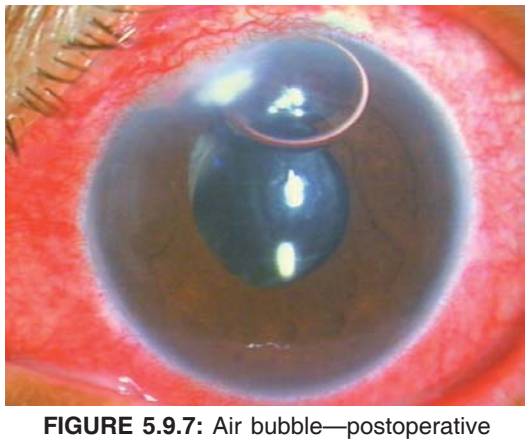


FIGURE 5.9.7: Air bubble—postoperative

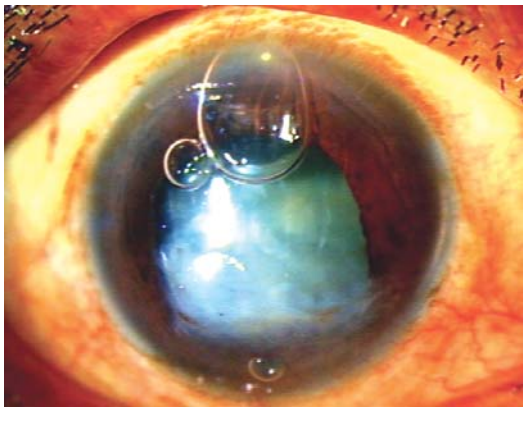


FIGURE 5.9.8: Air bubble—penetrating injury

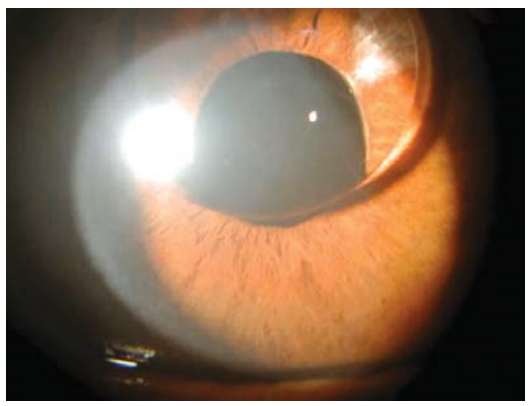


FIGURE 5.9.9: Silicone oil —single bubble

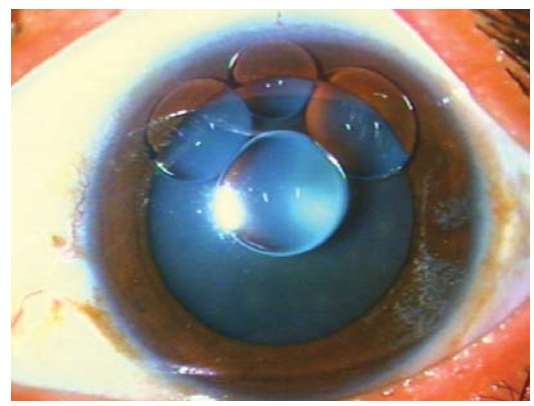


FIGURE 5.9.10: Silicone oil—multiple bubbles

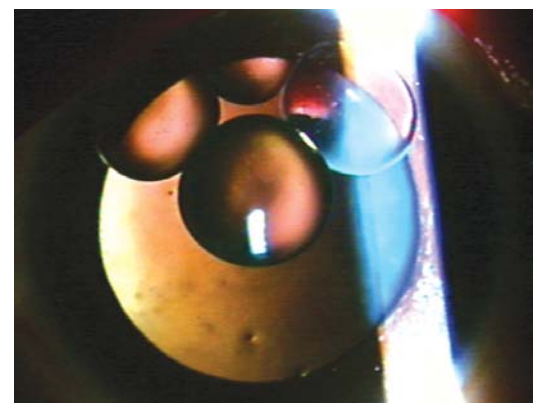


FIGURE 5.9.11: Silicone oil —multiple bubbles

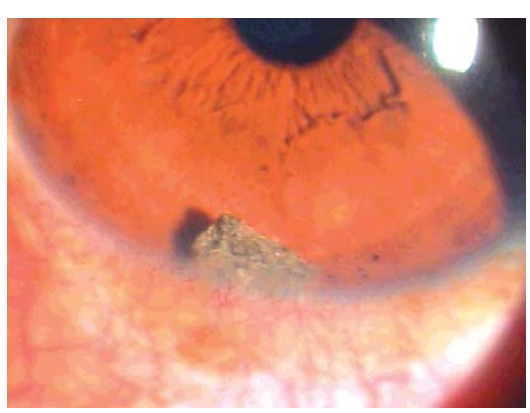


FIGURE 5.9.12: Foreign body

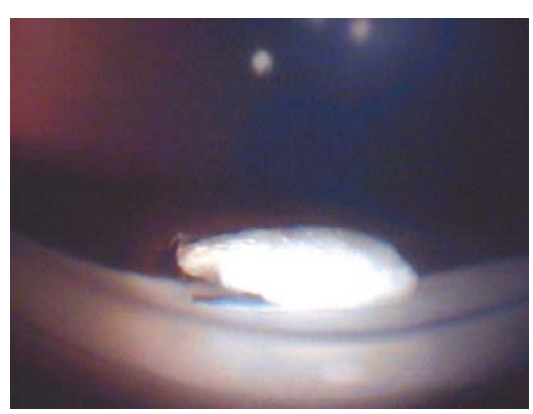


FIGURE 5.9.13: Foreign body in angle of AC

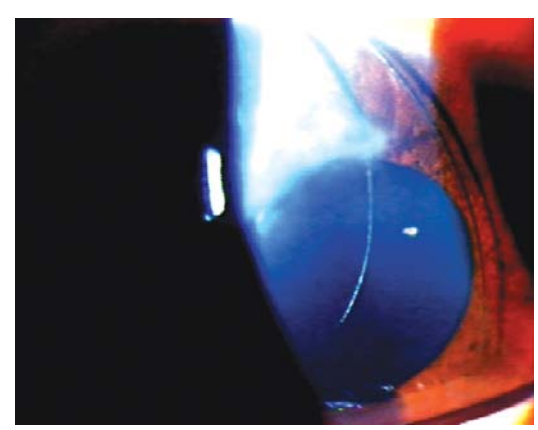


FIGURE 5.9.14: Eyelash

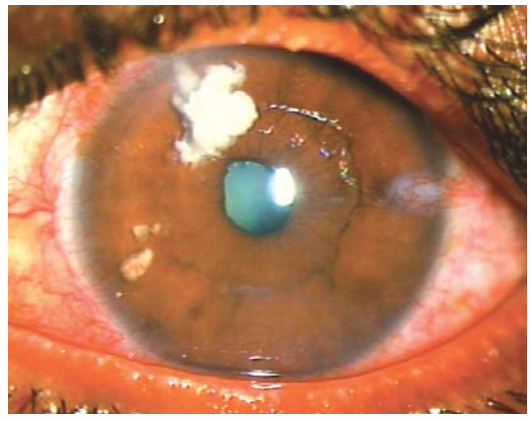


FIGURE 5.9.15: Fungal granuloma

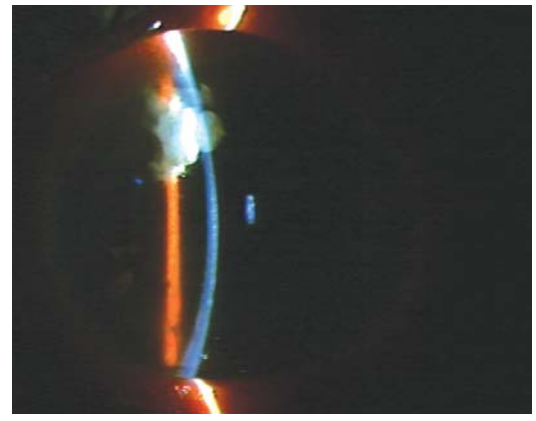


FIGURE 5.9.16: Fungal granuloma

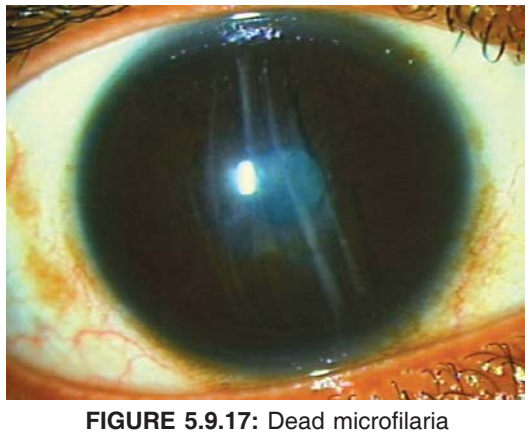


FIGURE 5.9.17: Dead microfilaria

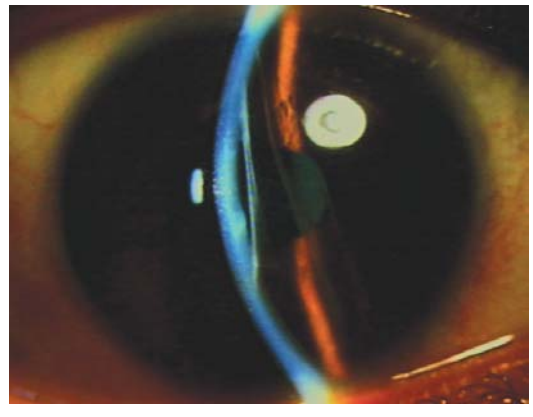


FIGURE 5.9.18: Dead microfilaria

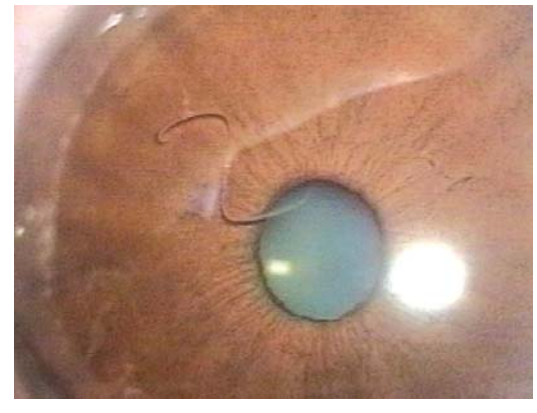


FIGURE 5.9.19: Live microfilaria

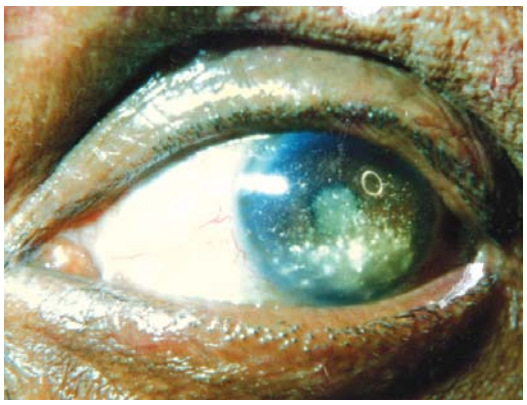


FIGURE 5.9.20: Cholesterolosis bulbi

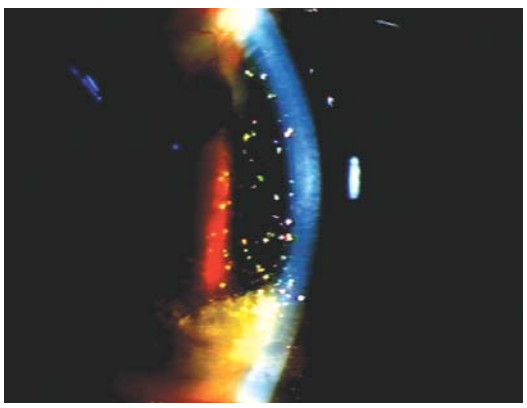


FIGURE 5.9.21: Cholesterolosis bulbi

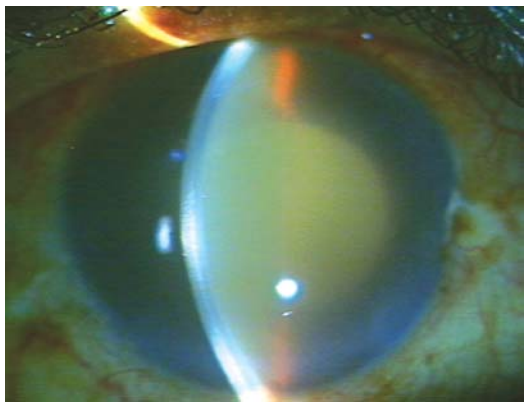


FIGURE 5.9.22: Milky liquefied lens cortex

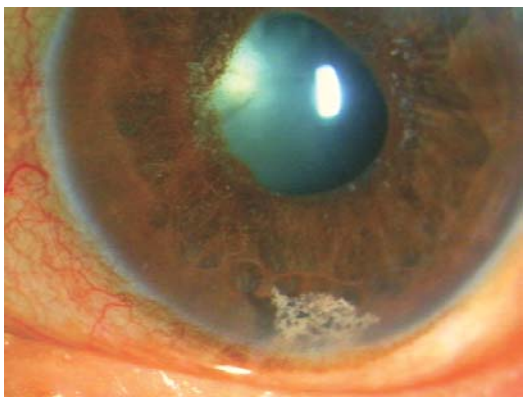


FIGURE 5.9.23: Pseudoexfoliative materials

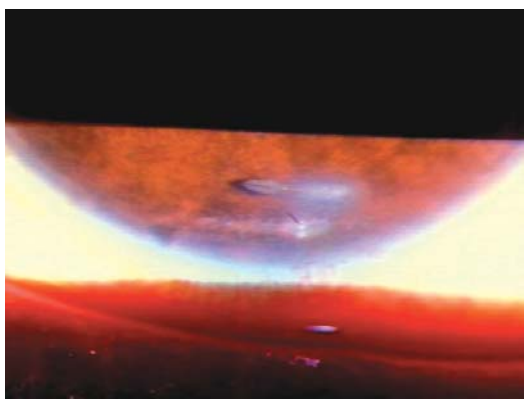


FIGURE 5.9.24: Caterpillar hair

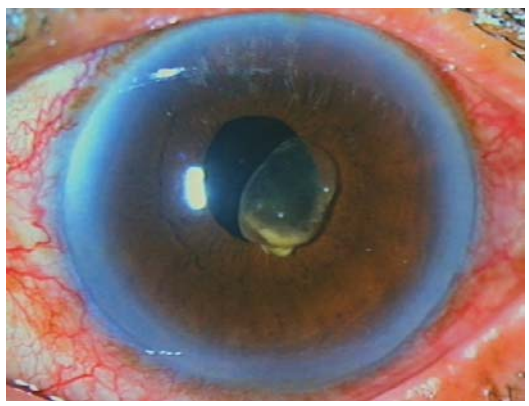


FIGURE 5.9.25: Epithelial cyst

6 Diseases of the Uvea

CONGENITAL CONDITIONS

- Aniridia
- Coloboma
- Albinism
- Heterochromia iridum
- Ectropion uveae

ANTERIOR UVEITIS (IRIDOCYCLITIS)

- Aqueous flare
- Anterior chamber
- Iris nodules
- Posterior synechia

CHOROIDITIS

- Toxoplasmosis
- Toxocariasis
- Vogt-Koyanagi-Harada (VKH) syndrome
- Sympathetic ophthalmitis
- Birdshot retinochoroiditis
- Endophthalmitis
- Panophthalmitis

IRIDO-CORNEAL ENDOTHELIAL (ICE) SYNDROMES

- Progressive essential iris atrophy
- Iris-naevus syndrome (Cogan-Reese)
- Chandler's syndrome

OTHER UVEAL DISEASES

- Iridoschisis
- Iris atrophy
- Rubeosis iridis
- Primary choroidal sclerosis
- Iris cysts
- Gyrate atrophy of the choroid
- Chorioderemia
- Angioid streaks
- Acute posterior multifocal placoid pigment epitheliopathy
- Serpiginous (geographical) choroidopathy
- Choroidal detachment

BENIGN UVEAL LESIONS/ NODULES/ MASS

- Iris floccules
- Iris freckles
- Lisch nodules (spots)
- Brushfield spots
- Iris pearls
- Iris mammiliations
- Iris naevus
- Choroidal hemangioma
- Benign melanocytoma (naevus)
- Choroidal osteoma

MALIGNANT UVEAL TRACT LESIONS

- Metastatic deposits of retinoblastoma
- Malignant melanoma of iris
- Medullo-epithelioma (diktyoma)
- Malignant melanoma of the ciliary body
- Malignant melanoma of the choroid
- Metastatic carcinoma of uveal tract

CONGENITAL CONDITIONS

Aniridia

- Whole of the iris is appeared to be missing on external examination
- Ciliary processes and suspensory ligaments of the lens are visible **(Figs 6.1.1 and 6.1.2)**
- Subluxation of lens and secondary glaucoma in 25 percent of the cases **(Fig 6.1.3)**
- Aniridia and Wilms' tumor are associated with deletion of the short arm of chromosome-11 (Miller's syndrome)

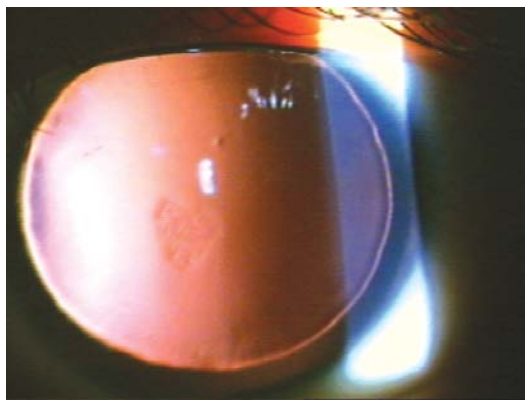


FIGURE 6.1.1: Aniridia

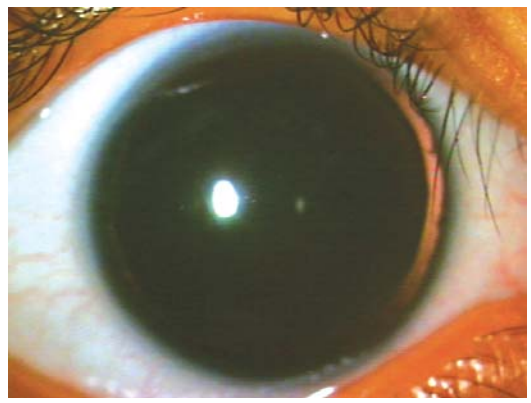


FIGURE 6.1.2: Aniridia

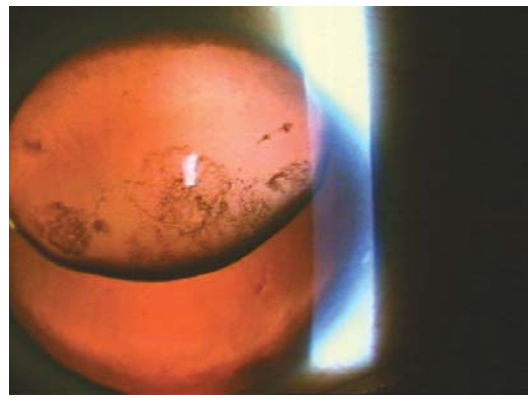


FIGURE 6.1.3: Aniridia

Coloboma

- Development imperfect closure of the fetal fissure
- Lower nasal sector of the eye
- Coloboma may be typical or atypical
- Extends from the pupil to the optic nerve **(Figs 6.2.1 and 6.2.2)**
- May stop short of the optic nerve, iris or else or even partitioned by islands of normal tissue
- *Typical coloboma of the iris*
  - pear-shaped with broad base towards the pupillary margin
  - it may be complete **(Fig 6.2.3)** or incomplete **(Fig 6.2.4)**
- *Atypical coloboma of the iris*
  - isolated coloboma with or without lens coloboma which occurs at any meridian **(Fig 6.2.5)**
  - may be surgical **(Fig 6.2.6)**

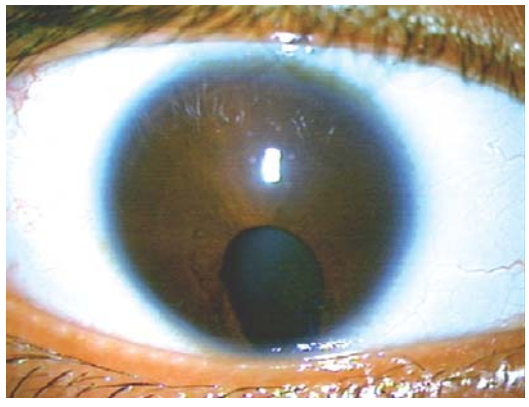


FIGURE 6.2.1: Typical coloboma

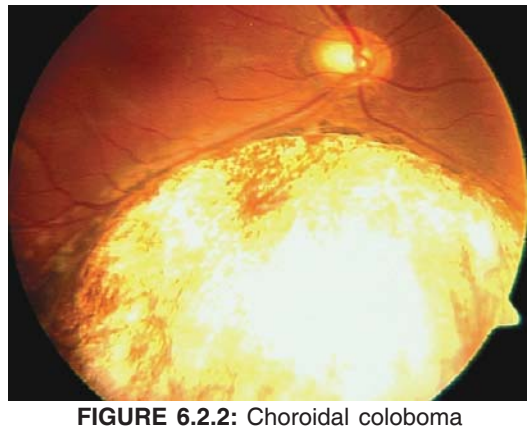


FIGURE 6.2.2: Choroidal coloboma

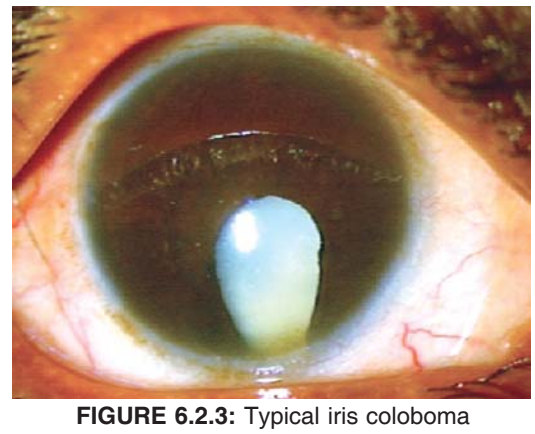


FIGURE 6.2.3: Typical iris coloboma

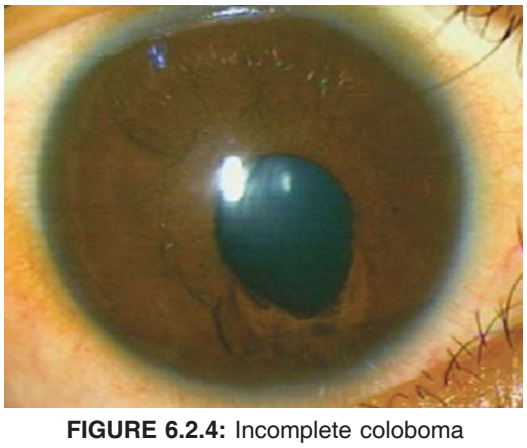


FIGURE 6.2.4: Incomplete coloboma

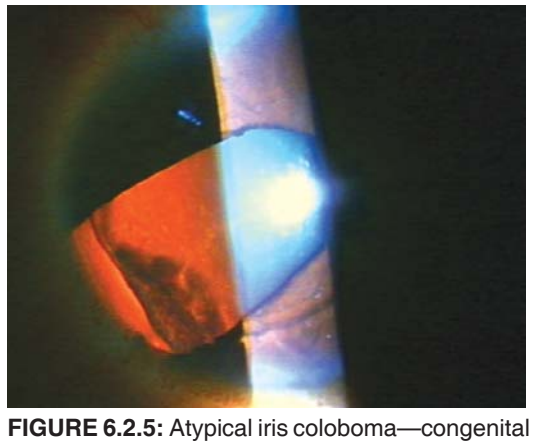


FIGURE 6.2.5: Atypical iris coloboma—congenital

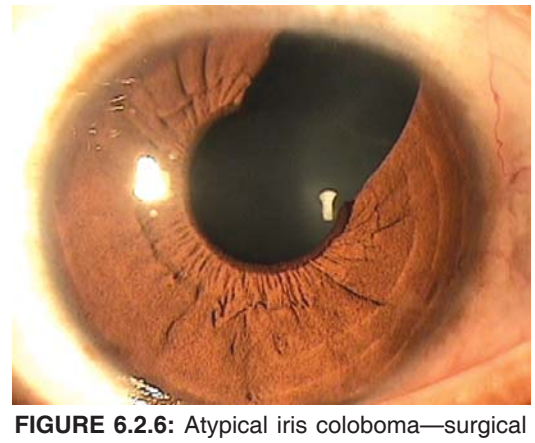


FIGURE 6.2.6: Atypical iris coloboma—surgical

Albinism

- Hereditary disorder in which there is absence or reduction of melanin pigmentation throughout the body **(Fig 6.3.1)**
- *Two main types*: oculocutaneous and ocular
- Iris looks pink and translucent, owing to lack of pigment **(Figs 6.3.2 and 6.3.3)**
- Fundus appears orange-pink in color **(Fig 6.3.4)**. The retinal and choroidal vessels are difficult to differentiate **(Fig 6.3.5)**
- *Partial albinism*
  - absence of pigment is limited to the choroids and retina
  - iris is brown or blue colored

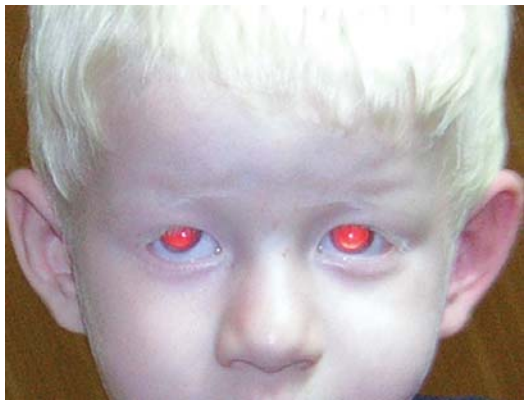


FIGURE 6.3.1: Oculocutaneous albinism

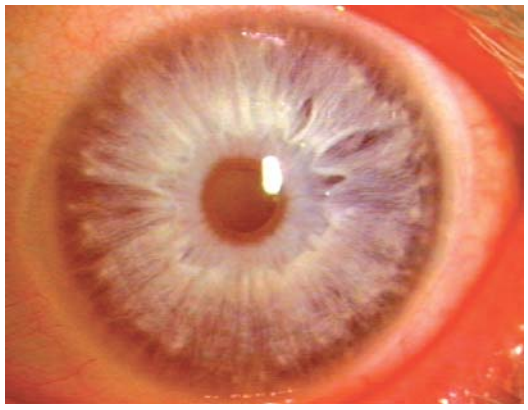


FIGURE 6.3.2: Albinism—pink iris

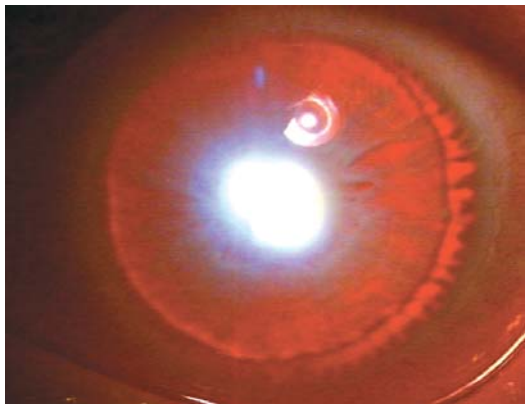


FIGURE 6.3.3: Albinism—iris transillumination

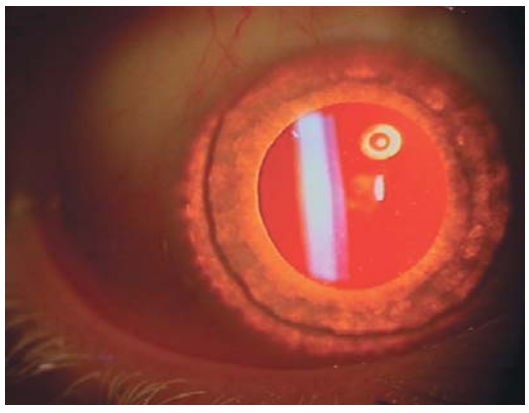


FIGURE 6.3.4: Albinism—iris transillumination

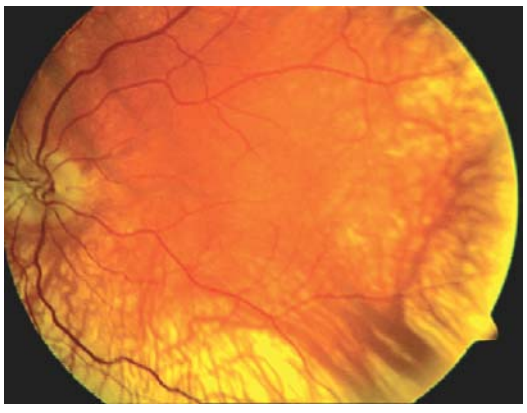


FIGURE 6.3.5: Albinism—fundus

Heterochromia Iridum

- Two irides show a significant difference in color **(Figs 6.4.1 and 6.4.2)**
- *Heterochromia iridis*: usually a sector, shows difference in color from the remainder **(Fig 6.4.3)**
- *Hypochromic* **(Figs 6.4.4 and 6.4.5)**
  - *eye* with lighter-colored iris is abnormal
- *Hyperchromic* **(Figs 6.4.6 and 6.4.7)**
  - iris on the side of the disease is darker than its fellow

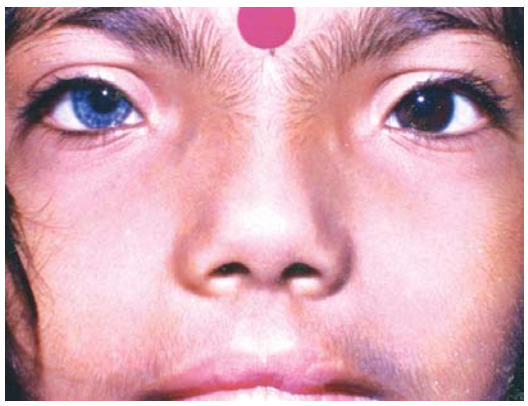


FIGURE 6.4.1: Heterochromia

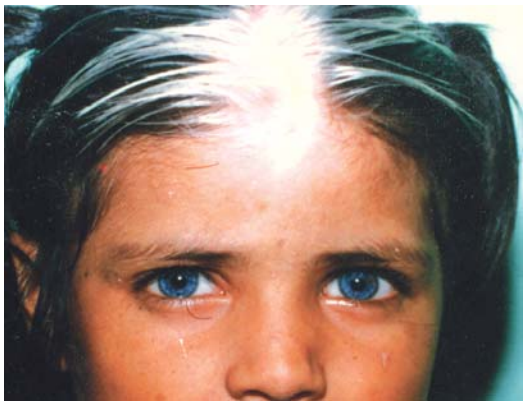


FIGURE 6.4.2: Heterochromia—Waardenberg syndrome

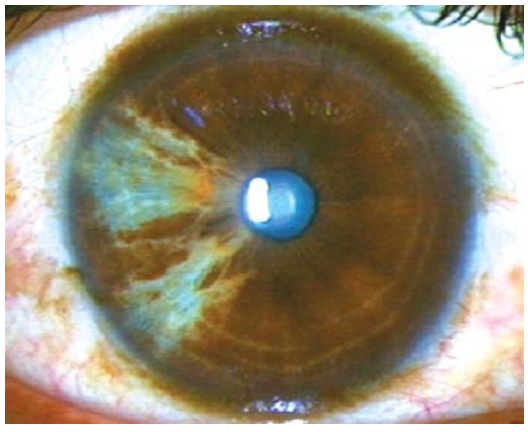


FIGURE 6.4.3: Heterochromia iridis

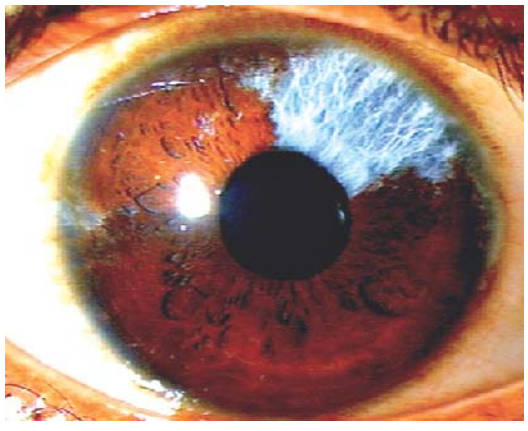


FIGURE 6.4.4: Hypochromic heterochromia

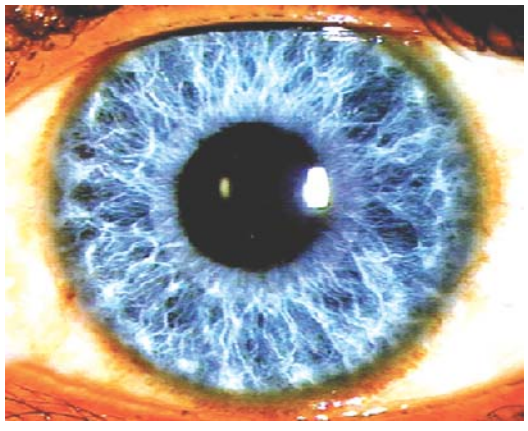


FIGURE 6.4.5: Hypochromic heterochromia

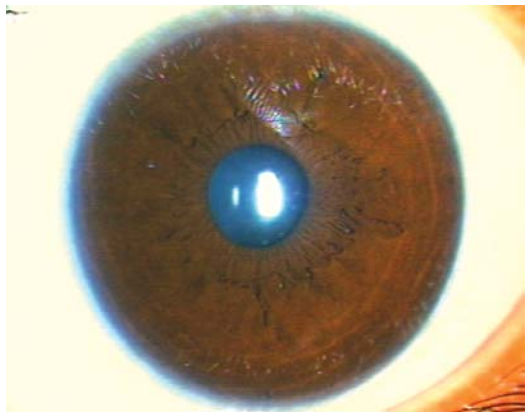


FIGURE 6.4.6: Hyperchromic heterochromia—normal eye

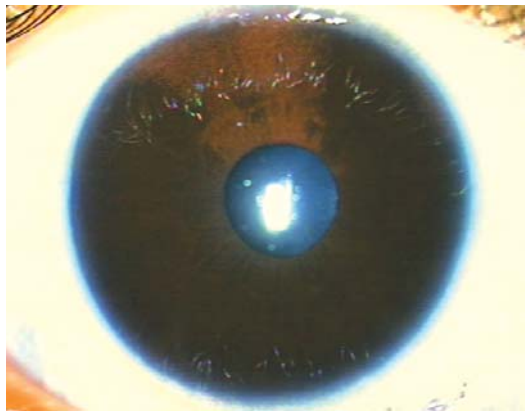


FIGURE 6.4.7: Hyperchromic heterochromia

Ectropion Uveae

- Pigmented iris surface appears anteriorly, especially along the pupillary border
- *Causes:*
  - isolated congenital condition: non-progressive **(Fig 6.5.1)**
  - Axenfeld-Rieger's anomaly **(Fig 6.5.2)**
  - neurofibromatosis **(Fig 6.5.3)**
  - rubeosis iridis **(Fig 6.5.4)**
  - iris coloboma **(Fig 6.5.5)**
  - siderosis bulbi **(Fig 6.5.6)**
- Patients should be looked for glaucoma

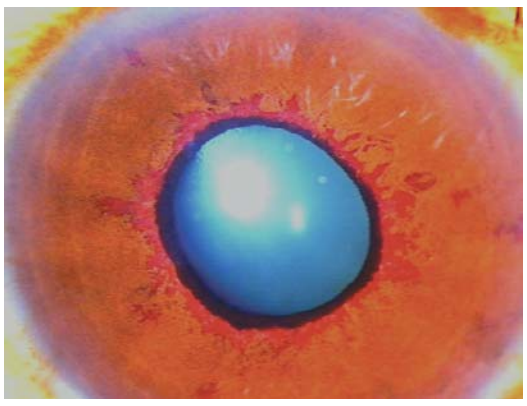


FIGURE 6.5.1: Ectropion uveae—congenital

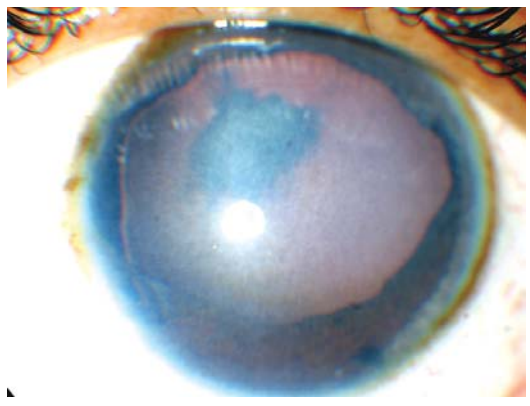


FIGURE 6.5.2: Ectropion uveae—AR syndrome

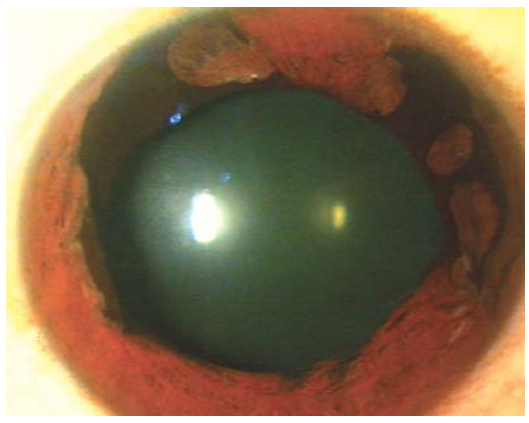


FIGURE 6.5.3: Ectropion uveae—neurofibromatosis

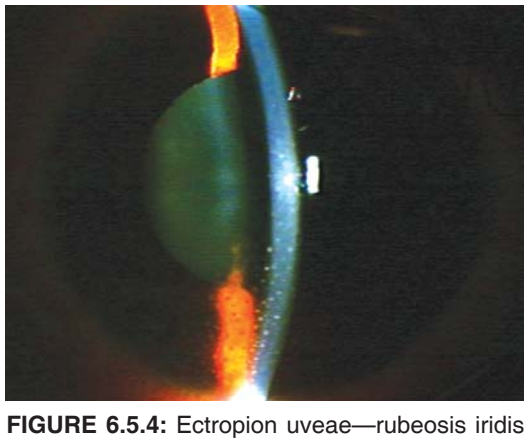


FIGURE 6.5.4: Ectropion uveae—rubeosis iridis

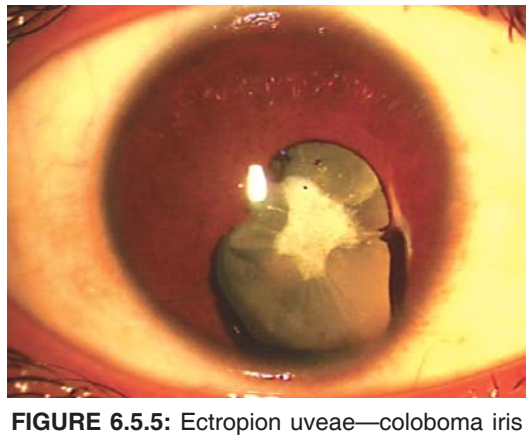


FIGURE 6.5.5: Ectropion uveae—coloboma iris

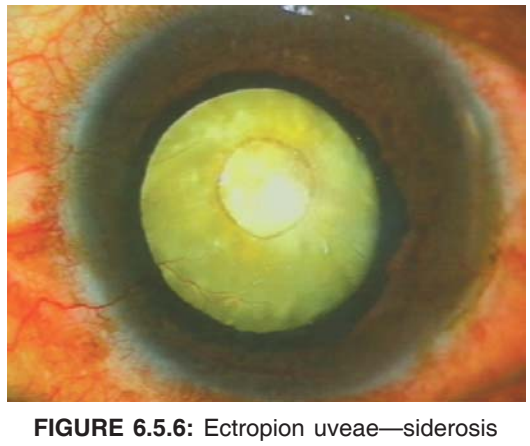


FIGURE 6.5.6: Ectropion uveae—siderosis

ANTERIOR UVEITIS (IRIDOCYCLITIS)

- May be acute nongranulomatous **(Fig 6.6.1)** or chronic granulomatous **(Fig 6.6.2)** type
- Circum-corneal (ciliary) congestion **(Fig 6.6.3)**
- Keratic precipitates (KPs) –They are cellular deposits on the corneal endothelium
  - *Distribution*—They arrange in a base-down triangular area at the lower part of the cornea (Arlt's triangle) **(Fig 6.6.4)**
  - In Fuchs' cyclitis, they are scattered throughout the endothelium
  - *Size:* KPs may be small **(Fig 6.6.5)**, medium **(Fig 6.6.6)**, large **(Fig 6.6.7)** and 'mutton fat' **(Fig 6.6.8)** types
  - Small and medium size KPs are due to deposition of lymphocytes and plasma cells and they are seen in *nongranulomatous* or acute uveitis
  - Large KPs are due to deposition of macrophage and epithelioid cells, and they are seen in *granulomatous* uveitis (as in tuberculosis or sarcoidosis)
  - *Age:* KPs may be fresh or old **(Fig 6.6.9)**

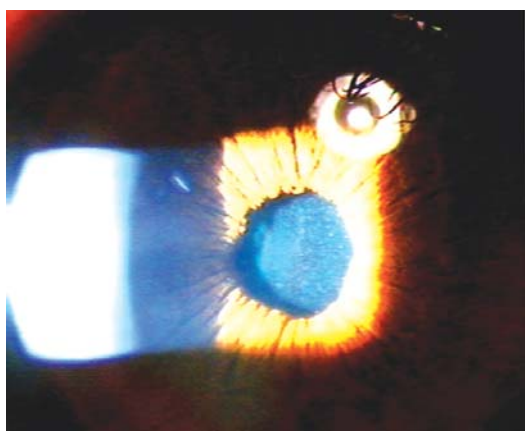


FIGURE 6.6.1: Acute nongranulomatous type

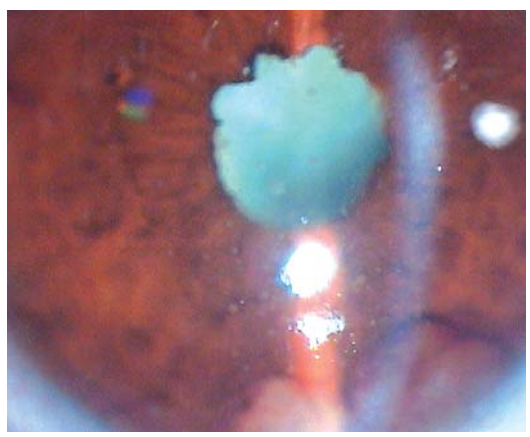


FIGURE 6.6.2: Chronic granulomatous uveitis

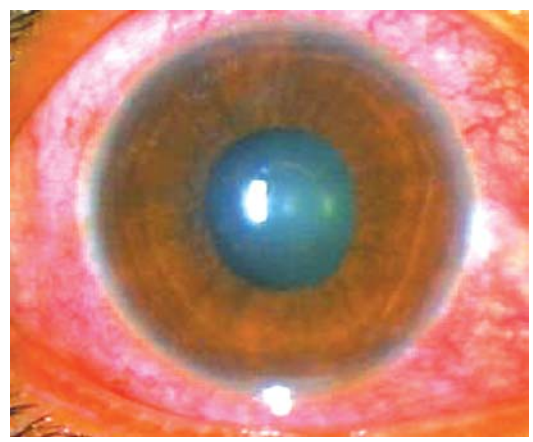


FIGURE 6.6.3: Iridocyclitis—ciliary congestion

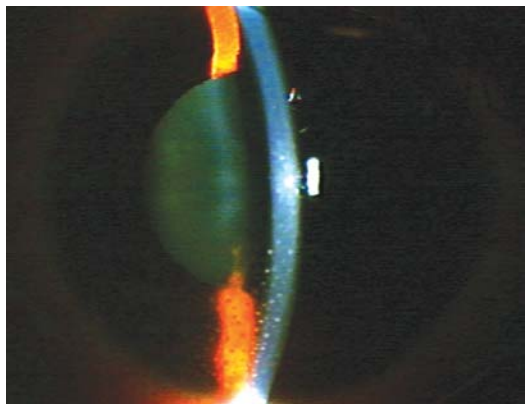


FIGURE 6.6.4: KPs—Arlt's triangle

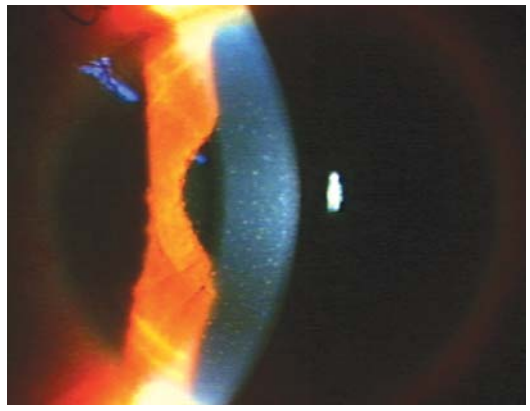


FIGURE 6.6.5: Small KPs

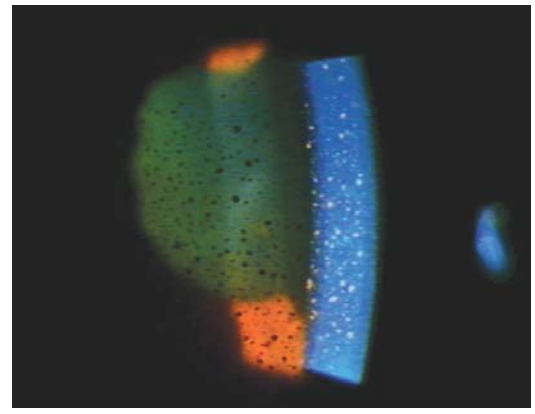


FIGURE 6.6.6: Medium KPs

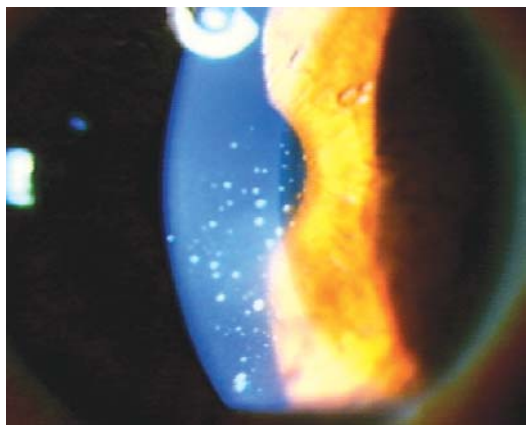


FIGURE 6.6.7: Large KPs

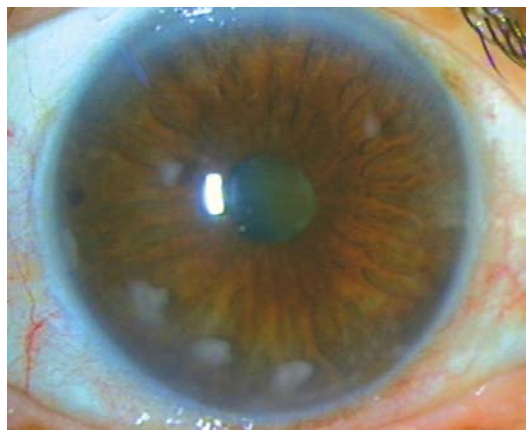


FIGURE 6.6.8: Mutton fat KPs

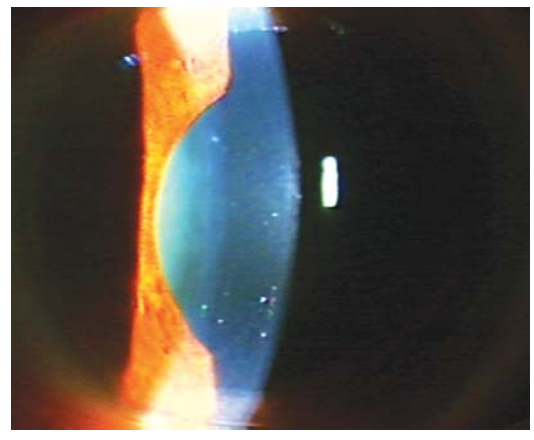


FIGURE 6.6.9: Old KPs

Aqueous Flare

- Proteins leaks through the damaged capillaries, causing a Tyndall effect **(Figs 6.7.1 and 6.7.2)**

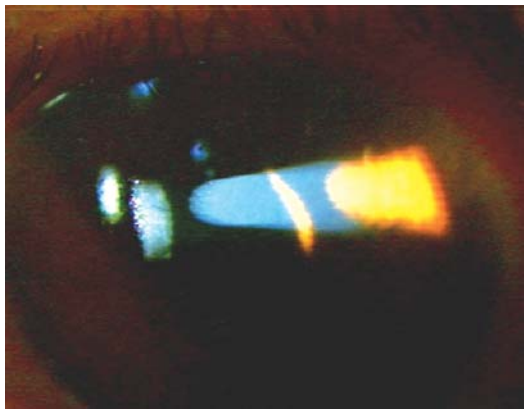


FIGURE 6.7.1: Aqueous flare

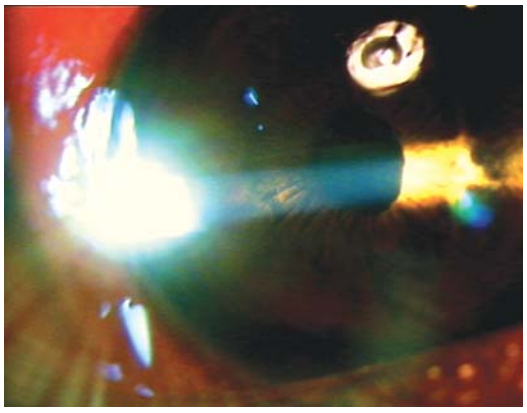


FIGURE 6.7.2: Aqueous flare

Anterior Chamber

- *Aqueous cells* **(Fig 6.8.1)**
- *Hypopyon:* classically seen in Behçet's syndrome or infectious cases (herpetic uveitis) **(Figs 6.8.2 and 6.8.3)**
- *Hyphema in herpetic uveitis* **(Fig 6.8.4)**
- *Deep and irregular:* in posterior synechiae **(Fig 6.8.5)**
- *Funnel-shaped:* in iris bombe **(Fig 6.8.6)**

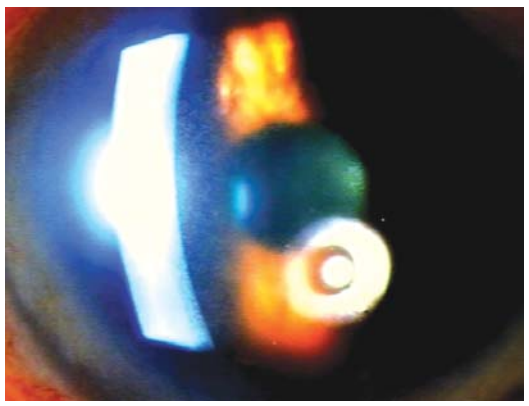


FIGURE 6.8.1: Aqueous cells

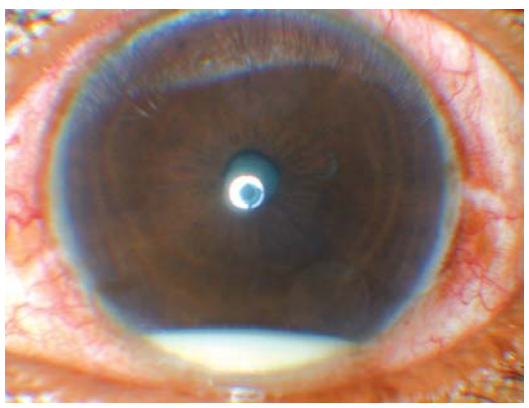


FIGURE 6.8.2: Acute iridocyclitis—hypopyon

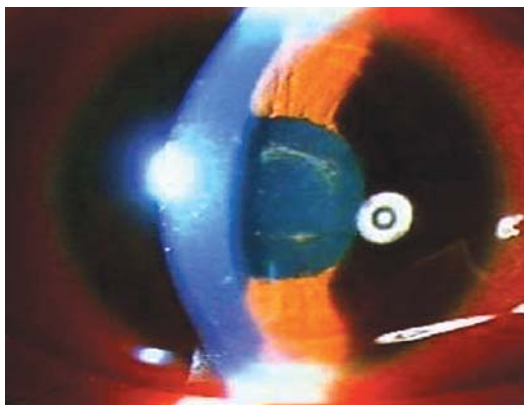


FIGURE 6.8.3: Acute iridocyclitis—hypopyon

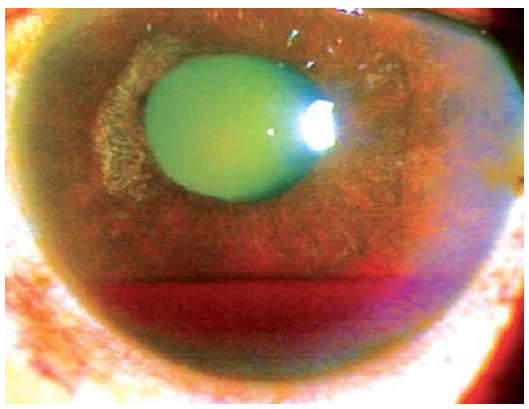


FIGURE 6.8.4: Herpetic iridocyclitis—hyphema

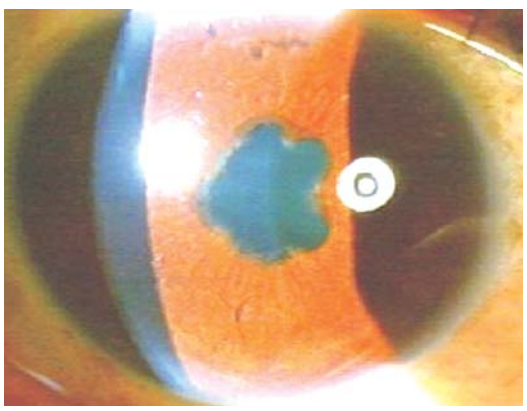


FIGURE 6.8.5: Acute iridocyclitis—posterior synechiae

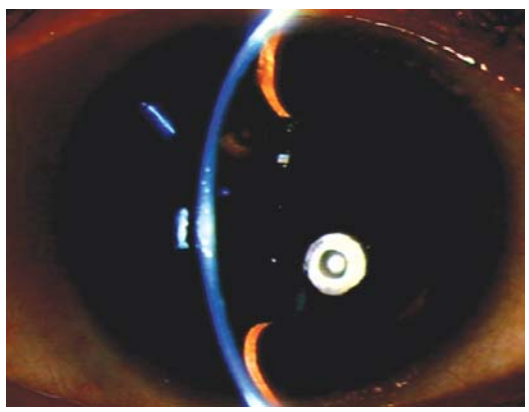


FIGURE 6.8.6: Acute iridocyclitis—iris bombe

Iris Nodules

- *Koeppe's nodules*: at the pupillary border and smaller in size **(Fig 6.9.1)**
- *Busacca's nodules*: on the surface of the iris, away from the pupil **(Fig 6.9.2)**

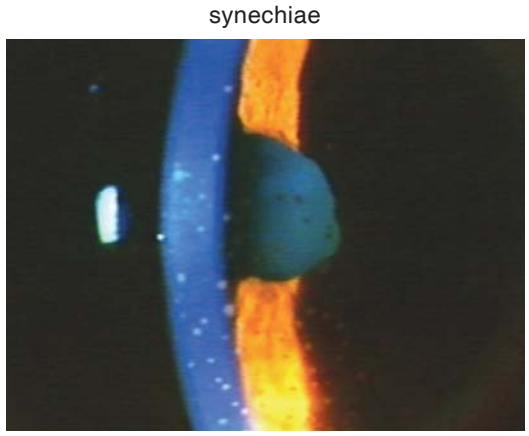


FIGURE 6.9.1: Koeppe's nodules

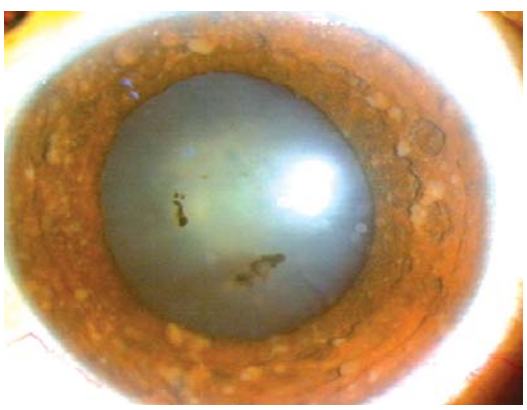


FIGURE 6.9.2: Busacca's nodules

Posterior Synechia

- Ring (annular) synechiae **(Fig 6.10.1)**
- Irregular or 'festooned pupil' **(Figs 6.10.2 and 6.10.3)**
- *Occlusio pupillae* **(Figs 6.10.4 and 6.10.5)**
- Complicated cataract **(Fig 6.10.6)**
- Band-shaped keratopathy (BSK) **(Fig 6.10.7)**; more common in children with juvenile rheumatoid arthritis, and chronic iridocyclitis
- Membrance formation **(Fig 6.10.8)**
- Pigments on anterior lens surface **(Fig 6.10.9)**

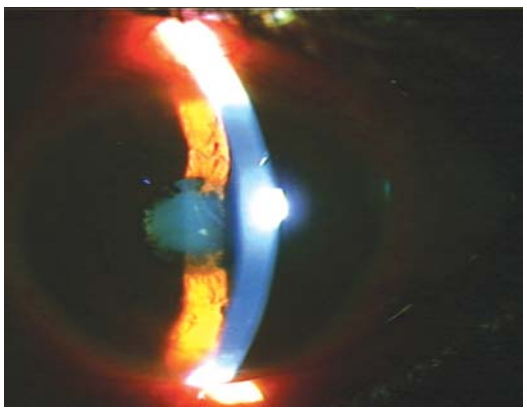


FIGURE 6.10.1: Ring synechiae—iris bombe

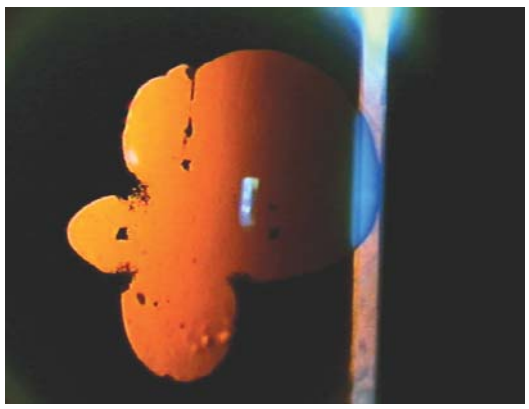


FIGURE 6.10.2: Irregular synechiae—festooned pupil

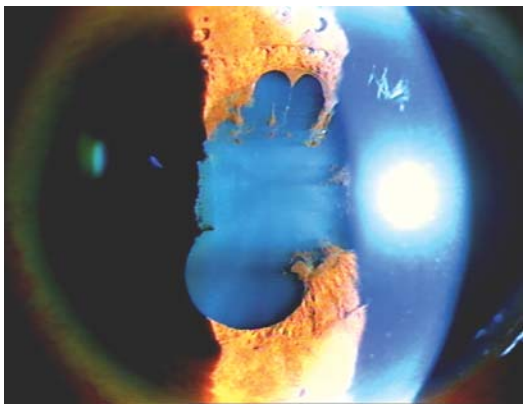


FIGURE 6.10.3: Festooned pupil

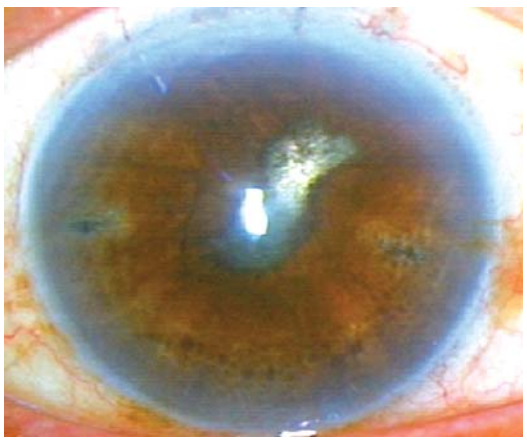


FIGURE 6.10.4: Occlusio pupillae

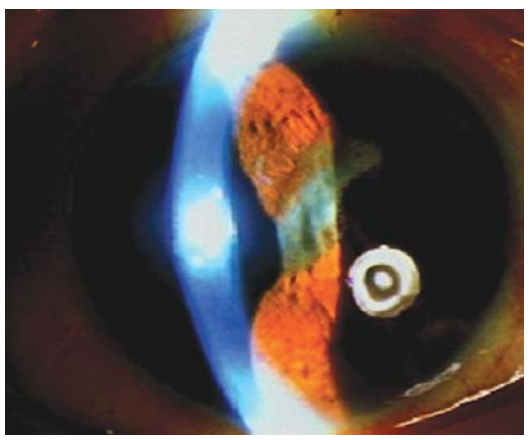


FIGURE 6.10.5: Occlusio pupillae—iris bombe

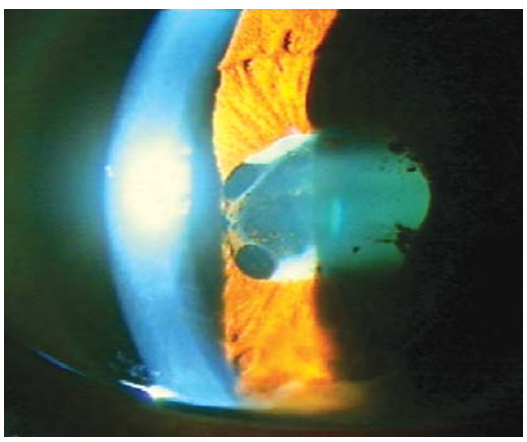


FIGURE 6.10.6: Healed Iritis—complicated cataract

FIGURE 6.10.7: Chronic iridocyclitis and BSK in JRA

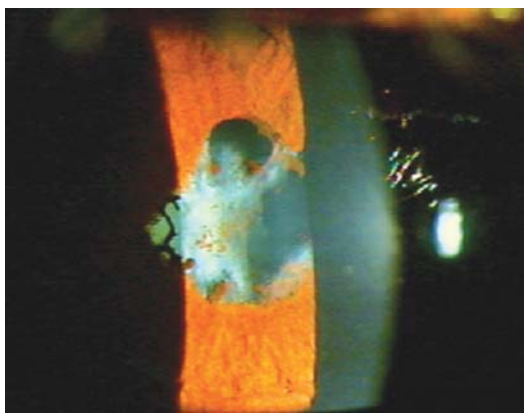


FIGURE 6.10.8: Healed uveitis—membrane formation

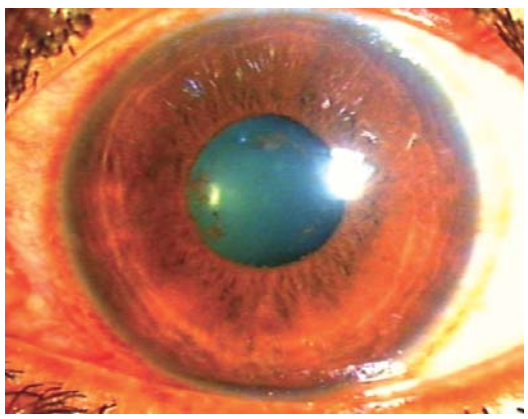


FIGURE 6.10.9: Healed uveitis—pigments on anterior lens surface

Choroiditis

- *Active lesions:* well-defined yellowish-white patches of chorioretinal inflammation
- *Inactive lesions:* well-defined white patches of chorioretinal atrophy with pigmented margins **(Figs 6.11.1 to 6.11.3)**
- *Perivasculitis:* periphlebitis is more common **(Fig 6.11.4)**
- Called 'candle-wax dripping', seen in sarcoidosis **(Fig 6.11.5)**

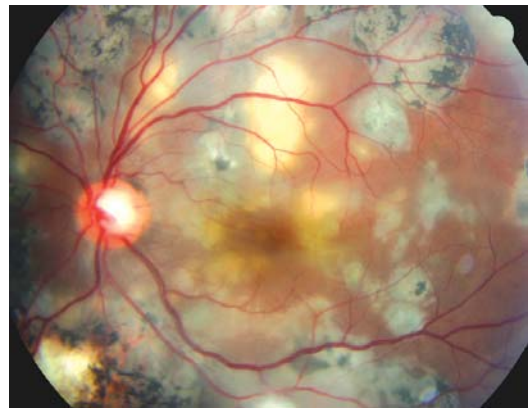


FIGURE 6.11.1: Choroiditis

FIGURE 6.11.2: Choroiditis—central

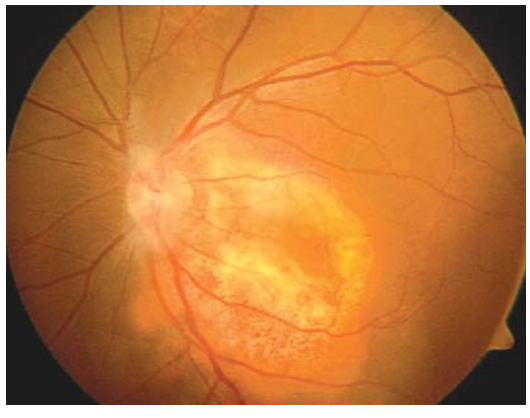


FIGURE 6.11.3: Choroiditis—juxtapapillary

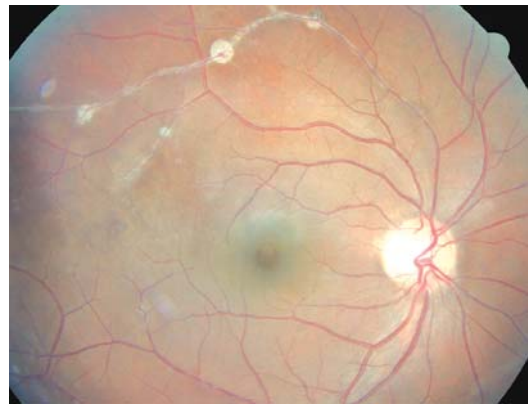


FIGURE 6.11.4: Perivasculitis

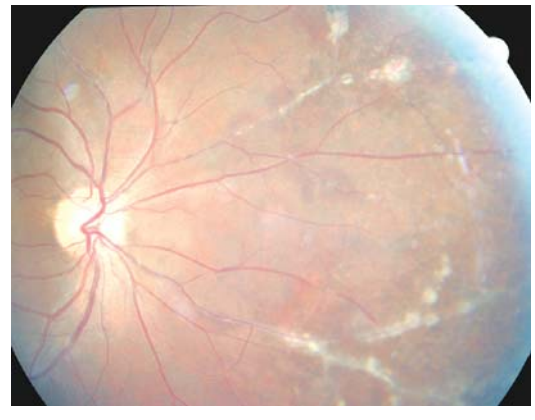


FIGURE 6.11.5: Perivasculitis—candle wax drippings

Toxoplasmosis

- Bilateral healed chorioretinal scars **(Figs 6.12.1 and 6.12.2)**, may be unilateral **(Fig 6.12.3)**
- *Focal necrotizing retinitis*-is a 'satellite lesion' adjacent to the edge of the old scar **(Fig 6.12.4)**
- *In reactivation*, white or yellowish white lesions with fluffy indistinct edges visible at the postequatorial fundus—"headlight in fog" appearance **(Fig 6.12.5)**

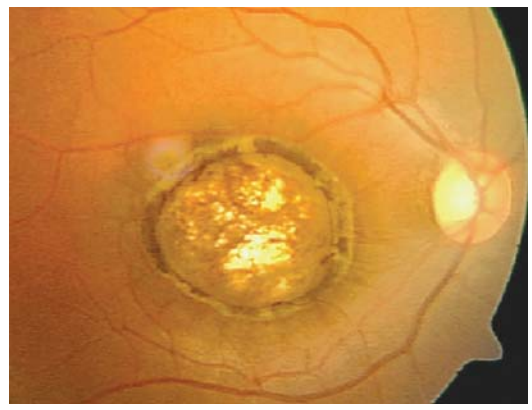


FIGURE 6.12.1: Toxoplasma choroiditis scar—bilateral

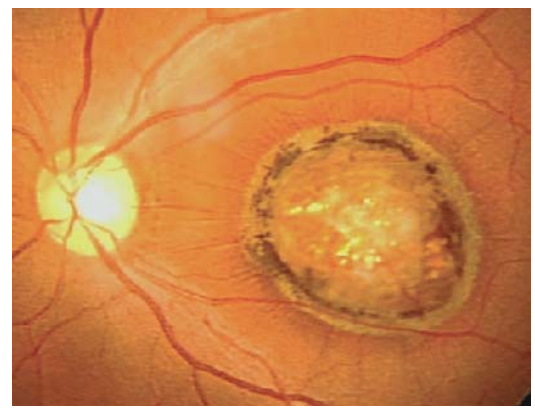


FIGURE 6.12.2: Toxoplasma choroiditis scar—bilateral

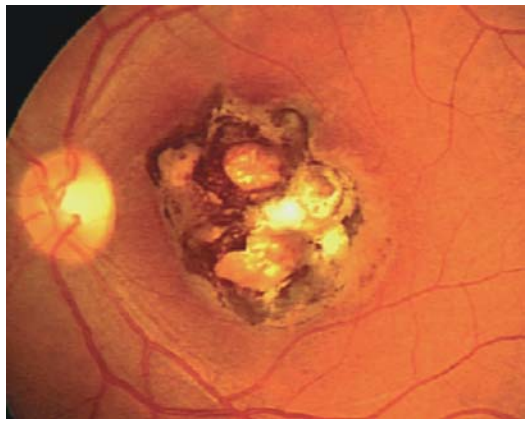


FIGURE 6.12.3: Toxoplasma choroiditis scar—unilateral

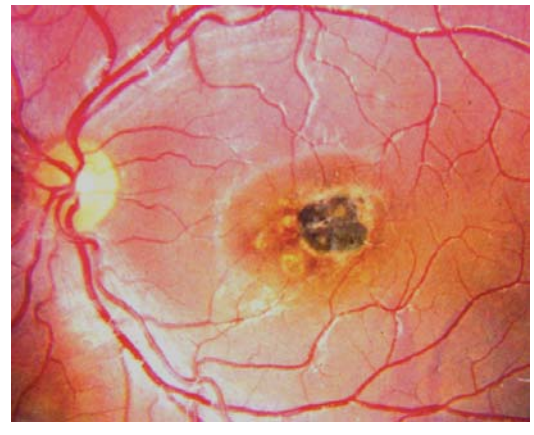


FIGURE 6.12.4: Toxoplasma scar—satellite lesion

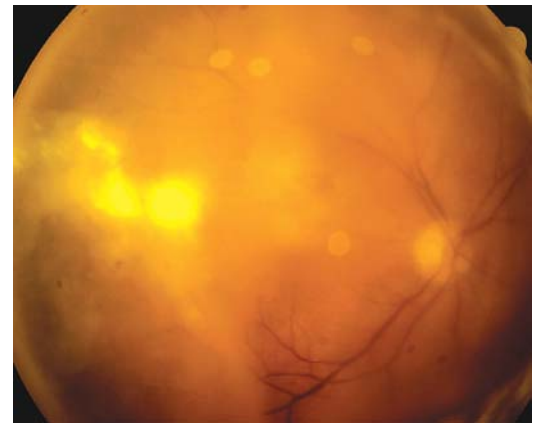


FIGURE 6.12.5: Toxoplasma reactivation—"head light-in fog"

Toxocariasis

- Chronic endophthalmitis, vitreous clouding and severe cyclitic membrane formation **(See Fig 7.9.8)**
- *See Chapter: 7*

Vogt-Koyanagi-Harada (VKH) Syndrome

- Rare, idiopathic condition affects Asians, especially the Japanese
- Alopecia, vitiligo and poliosis **(Fig 6.13.1)**
- Chronic granulomatous anterior uveitis **(Fig 6.13.2)** with multifocal choroiditis
- Multiple sensory retinal detachments may be with exudation (Harada's) **(Fig 6.13.3)**
- Prognosis is fairly good

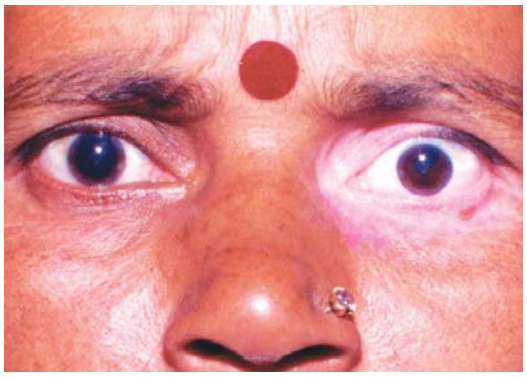


FIGURE 6.13.1: Vogt-Koyanagi-Harada syndrome

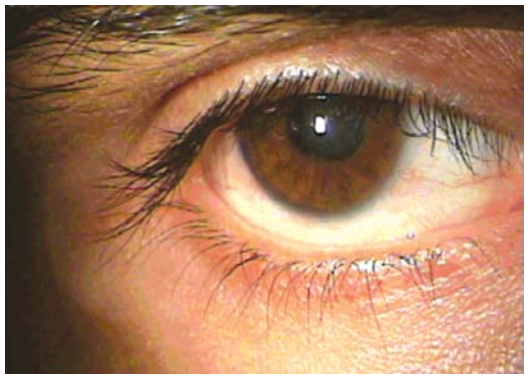


FIGURE 6.13.2: VKH syndrome—chronic granulomatous anterior uveitis

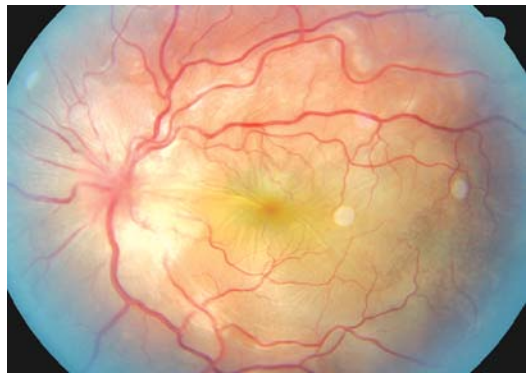


FIGURE 6.13.3: VKH syndrome—exudative retinal detachment

Sympathetic Ophthalmitis

- In the 'exciting eye'—there is ciliary congestion and evidence of initial insult
- In the 'sympathizing eye'—typical granulomatous uveitis with mutton-fat KPs, iris nodules
- Dalen-Fuchs' nodules scattered throughout posterior pole **(Fig 6.14.1)**

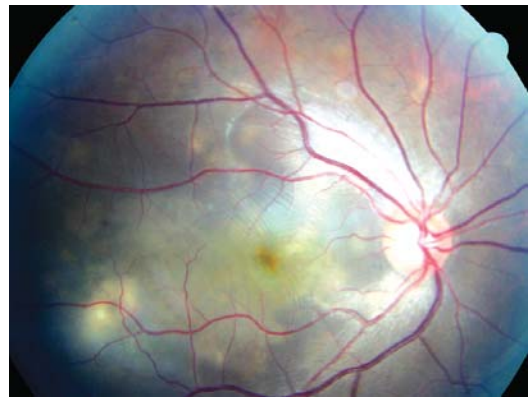


FIGURE 6.14.1: Sympathetic ophthalmia—Dalen-Fuchs' nodules

Birdshot Retinochoroiditis

- Uncommon, usually a bilateral condition
- Creamy-yellow, deep ovoid spots of moderate size with indistinct edges, radiate from the optic disk towards the equator **(Figs 6.15.1 and 6.15.2)**
- Prognosis is often guarded

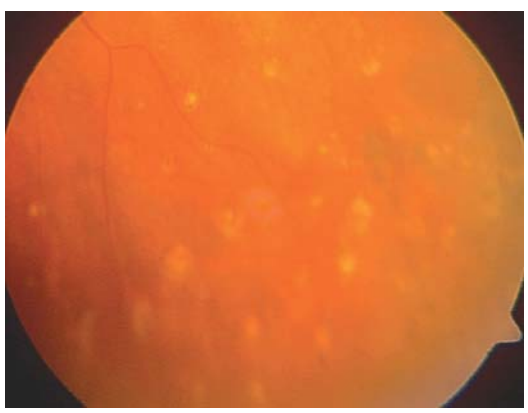


FIGURE 6.15.1: Birdshot retinochoroiditis

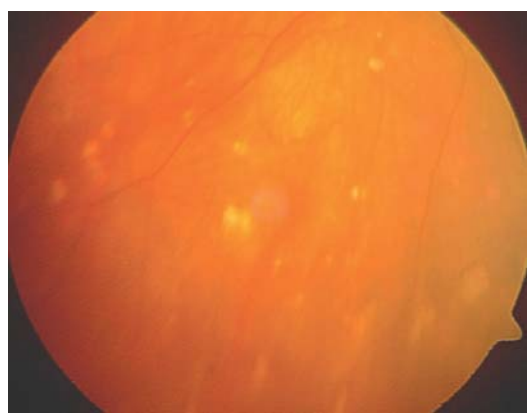


FIGURE 6.15.2: Birdshot retinochoroiditis

Endophthalmitis

- *Exogenous*
  - following penetrating injury **(Fig 6.16.1)**
  - following intraocular surgery **(Fig 6.16.2)**
  - following wound leak or bleb infection (after glaucoma surgery) **(Fig 6.16.3)**
- *Endogenous*
  - septic emboli (metastatic endophthalmitis) **(Fig 6.16.4)**
- *Toxic (sterile)*
  - toxic reaction to the chemicals used during surgery **(Fig 6.16.5)**

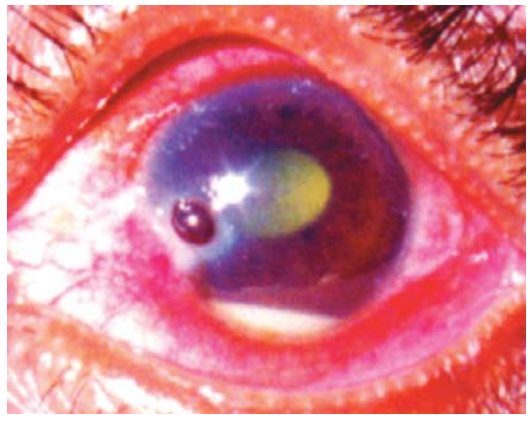


FIGURE 6.16.1: Endophthalmitis—penetrating injury

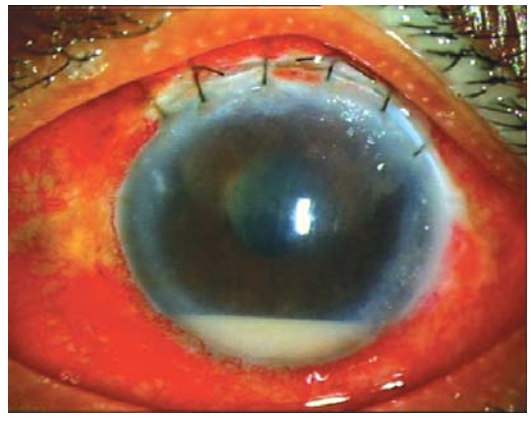


FIGURE 6.16.2: Endophthalmitis—cataract surgery

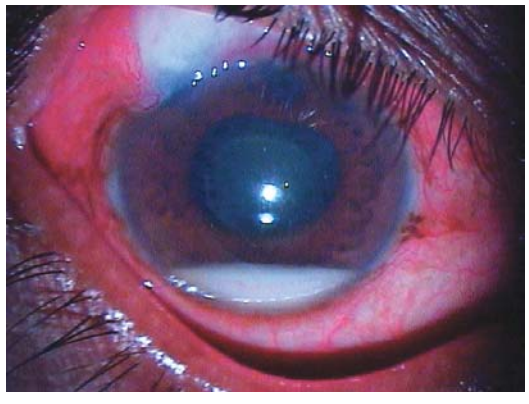


FIGURE 6.16.3: Blebitis—endophthalmitis

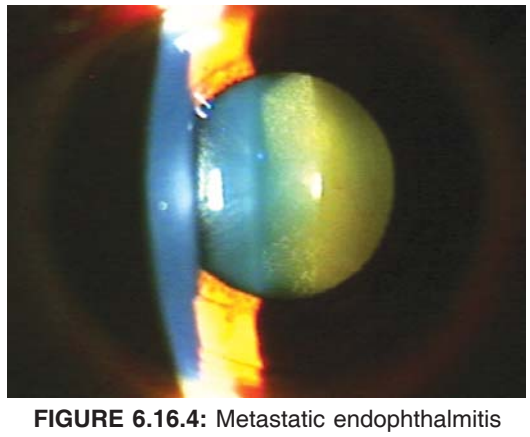


FIGURE 6.16.4: Metastatic endophthalmitis

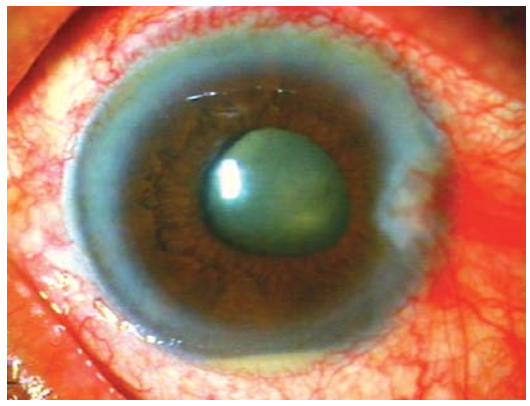


FIGURE 6.16.5: Toxic endophthalmitis

Panophthalmitis

- Marked lid edema
- Conjunctival chemosis and congestions
- Anterior chamber is full of pus
- Following corneal ulcer **(Fig 6.17.1)**, penetrating injury or post-surgical **(Fig 6.17.2)**

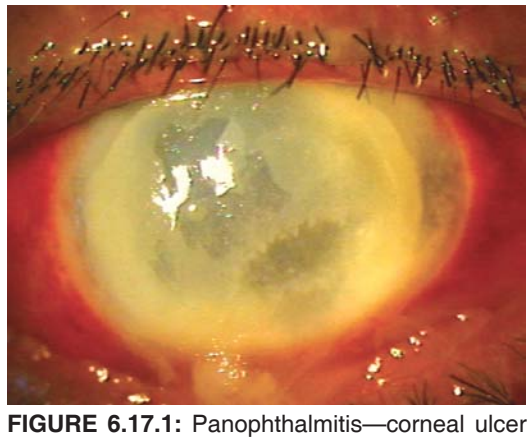


FIGURE 6.17.1: Panophthalmitis—corneal ulcer

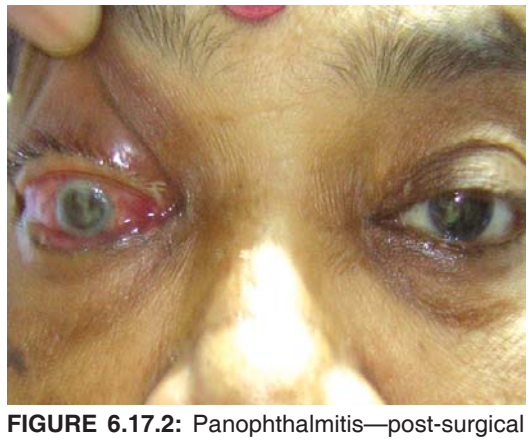


FIGURE 6.17.2: Panophthalmitis—post-surgical

IRIDO-CORNEAL ENDOTHELIAL (ICE) SYNDROMES

- Rare, unilateral condition, typically affects women and associated with secondary intractable glaucoma
- After metaplasia, Descemet's membrane-like material covers the anterior iris surface and angle of the anterior chamber, causing iris anomalies and secondary glaucoma

Progressive Essential Iris Atrophy

- Patchy iris atrophy with partial or complete hole formation **(Fig 6.18.1)**
- Corectopia
- Pseudopolycoria, and gradual enlargement of the iris holes **(Fig 6.18.2)**
- Broad peripheral anterior synechia **(Fig 6.18.3)**

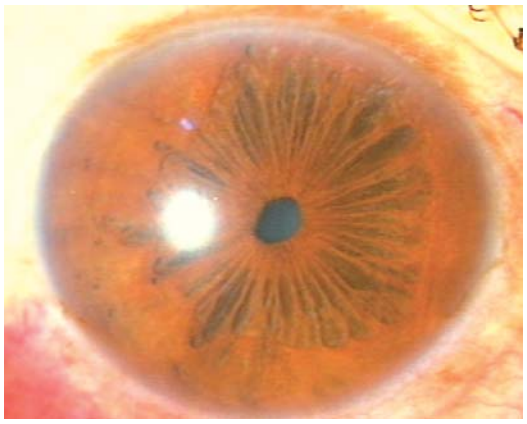


FIGURE 6.18.1: Essential iris atrophy

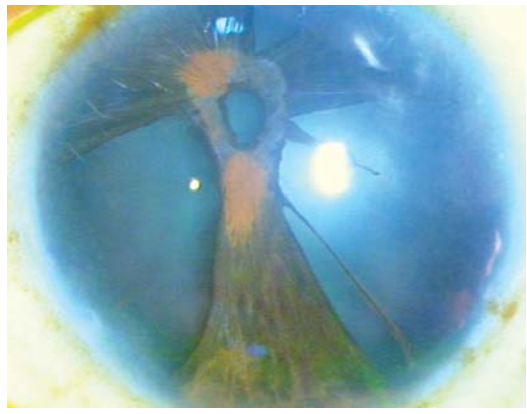


FIGURE 6.18.2: Essential iris atrophy—pseudopolycoria

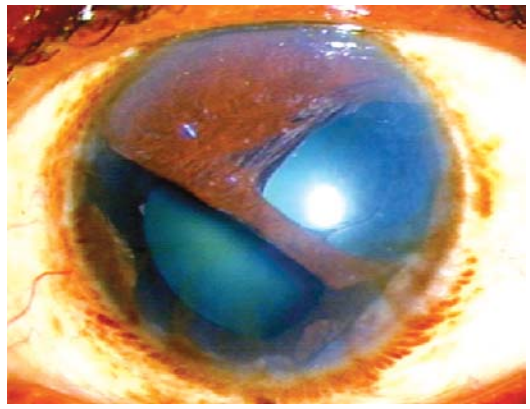


FIGURE 6.18.3: Essential iris atrophy—broad PAS

Iris-naevus Syndrome (Cogan-Reese)

- Dark-brown pigmented nodules in the iris stroma as small woolen-spherules **(Fig 6.19.1)**
- Peripheral anterior synechiae and secondary glaucoma may also occur **(Fig 6.19.2)**
- Endothelial decompensation appears later

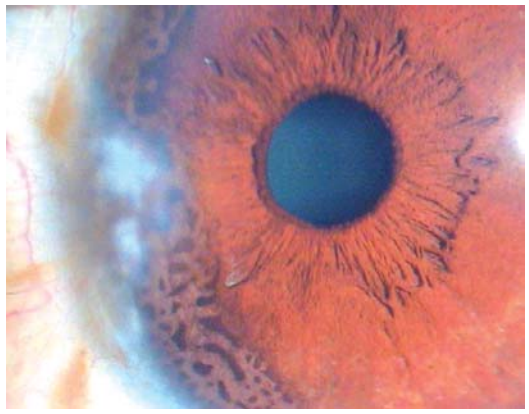


FIGURE 6.19.1: Cogan-Reese syndrome

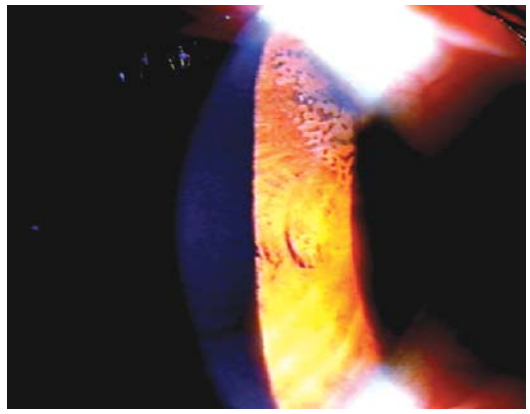


FIGURE 6.19.2: Cogan-Reese syndrome—PAS

Chandler's Syndrome

- Corneal endothelium appears 'beaten-silver' appearance **(Fig 6.20.1)**
- With gradual endothelial disturbances, corneal edema develops
- Iris atrophy is minimal

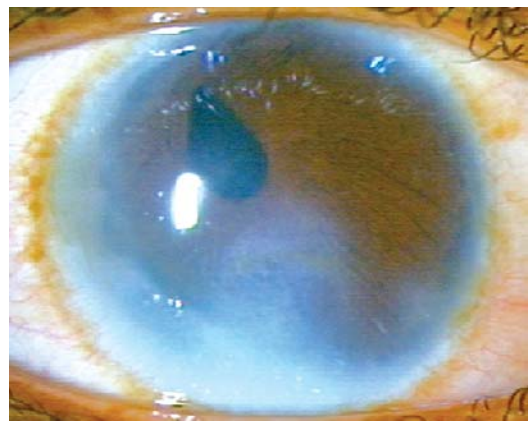


FIGURE 6.20.1: Chandler's syndrome

OTHER UVEAL DISEASES

Iridoschisis

- Senile degenerative condition of the iris
- Large dehiscences appear on the anterior mesodermal layer of the iris **(Fig 6.21.1)** and strands of tissue may float into the anterior chamber **(Fig 6.21.2)**
- *Treatment* is not necessary

FIGURE 6.21.1: Iridoschisis

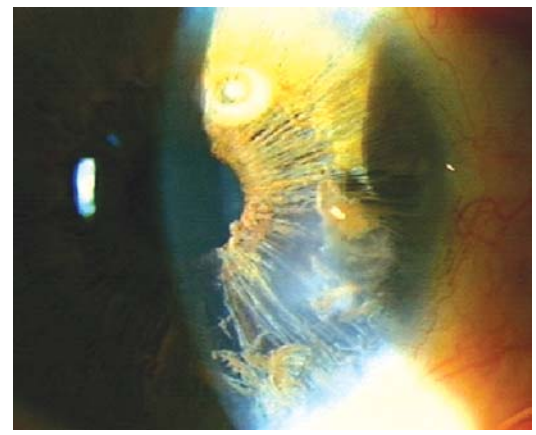


FIGURE 6.21.2: Iridoschisis

Iris Atrophy

- May be diffuse or sectorial
- *Diffuse type* occurs in old age, post-cataract surgery or other anterior segment surgeries **(Figs 6.22.1 and 6.22.2)**
- *Sectorial type* found in—herpetic iritis **(See Fig 6.8.4)**, angle closure glaucoma **(Fig 6.22.3)**, after surgery or trauma **(Fig 6.22.4)**

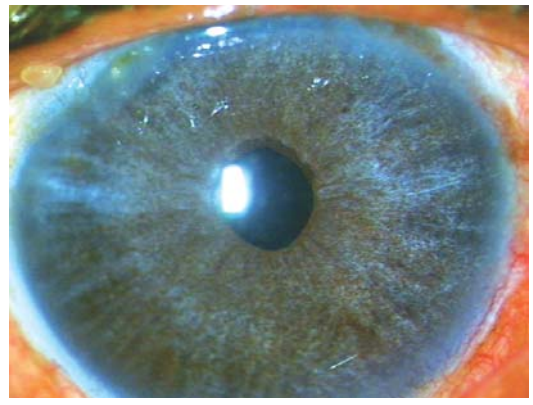


FIGURE 6.22.1: Iris atrophy—diffuse

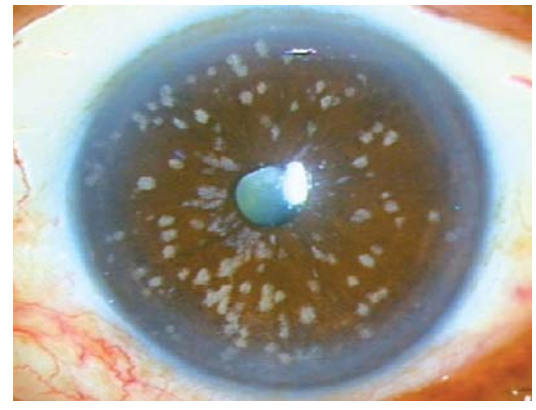


FIGURE 6.22.2: Iris atrophy—moth-eaten iris

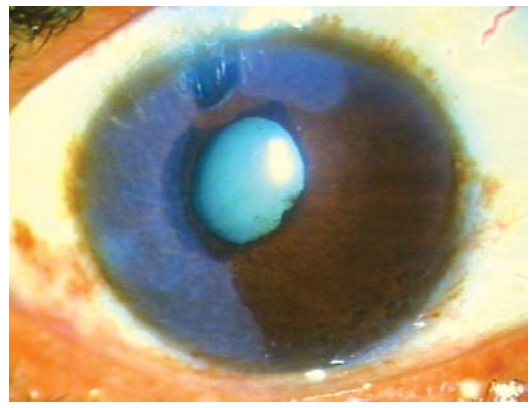


FIGURE 6.22.3: Iris atrophy—sectorial

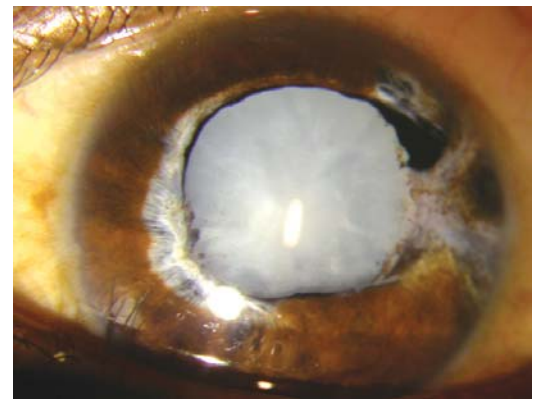


FIGURE 6.22.4: Iris atrophy—sectorial

Rubeosis Iridis

- Start as tiny dilated capillaries at the pupillary border
- Radial iris neovascularization **(Fig 6.23.1)**
- Followed by irregularly distributed network of new vessels on the iris surface and the stroma **(Fig 6.23.2)**
- New blood vessels and associated fibrous tissue may cover angle and the trabecular meshwork

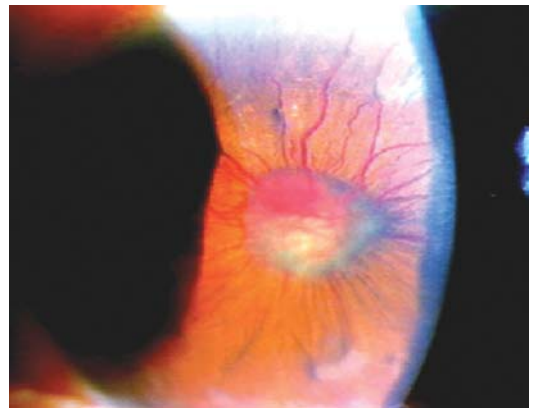


FIGURE 6.23.1: Rubeosis iridis

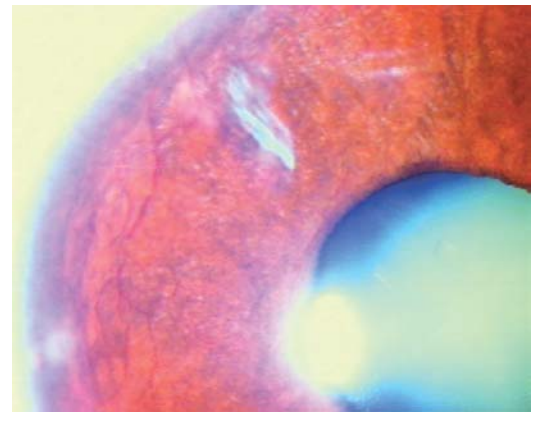


FIGURE 6.23.2: Rubeosis iridis

- Cause peripheral anterior synechiae and intractable neovascular glaucoma **(Fig 6.23.3)**
- Ectropion uveae is common in late stage **(Fig 6.23.4)** and new vessels may spread over the anterior surface of the lens **(Fig 6.23.5)**
- *Treatment:* pan-retinal photocoagulation; anterior-retinal cryocoagulation in presence of opaque media

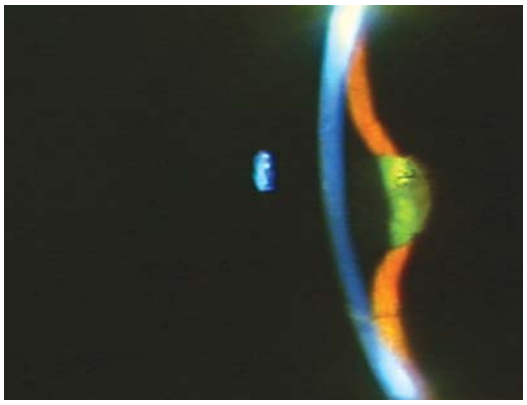


FIGURE 6.23.3: Rubeosis iridis—PAS—iris bombe

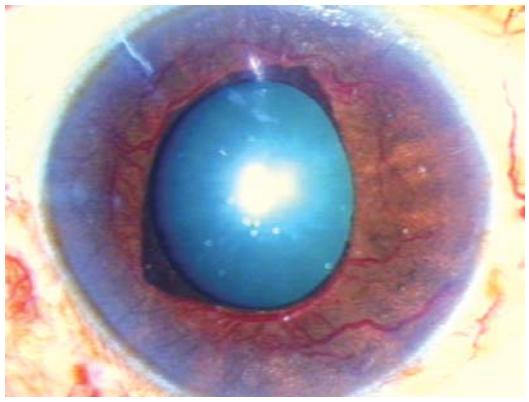


FIGURE 6.23.4: Rubeosis iridis—ectropion uveae

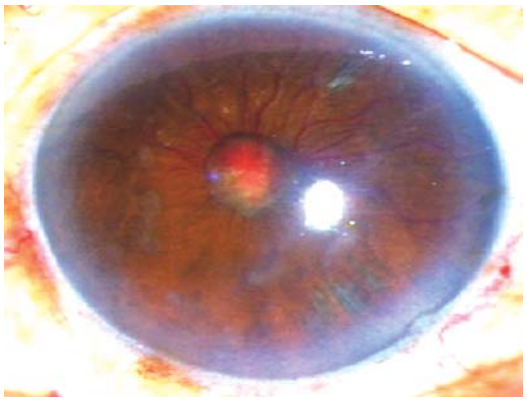


FIGURE 6.23.5: Rubeosis iridis—new vessels on lens surface

Primary Choroidal Sclerosis

- Diffuse atrophy of the RPE and chorio-capillaries
- 'Tessellated' appearance of the fundus
- It occurs in *two forms*
  - diffuse or generalized sclerosis **(Fig 6.24.1)**
  - localized sclerosis: affects the central or circum-papillary region **(Fig 6.24.2)**

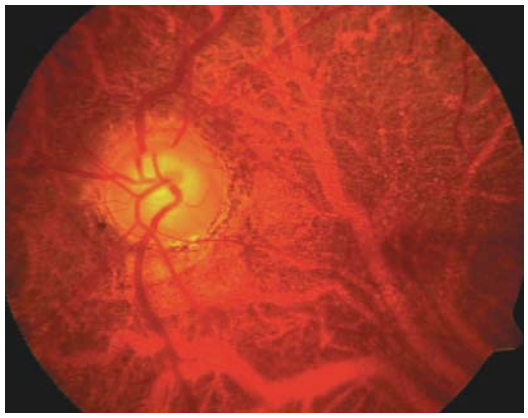


FIGURE 6.24.1: Choroidal sclerosis—diffuse

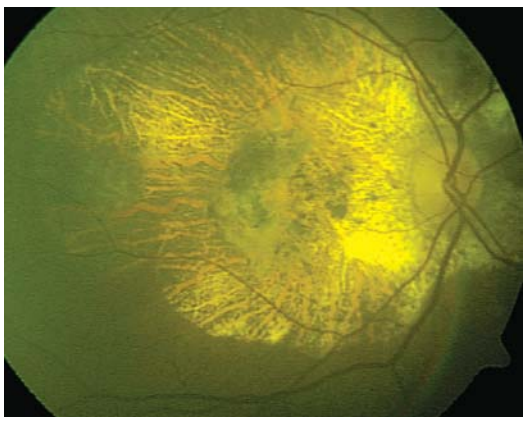


FIGURE 6.24.2: Choroidal sclerosis—localized

Iris Cysts

- Iris cysts are not very uncommon, and they may be primary or secondary
- *Primary iris cysts:* are stromal and occur in young children. It is filled with clear fluid and may often touch the cornea **(Fig 6.25.1)**
- *Post-surgical iris cysts:* a form of epithelial down growth and occur following cataract surgery **(Fig 6.25.2)**
  - cysts lie on the anterior surface of the iris, and grayish-white in color and filled with clear fluid with anterior clear wall **(Fig 6.25.3)**
- *Treatment*: surgical excision or sometimes YAG laser deroofing of the cyst

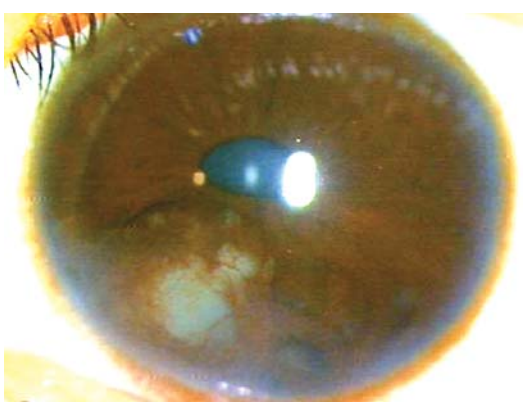


FIGURE 6.25.1: Iris cyst—primary

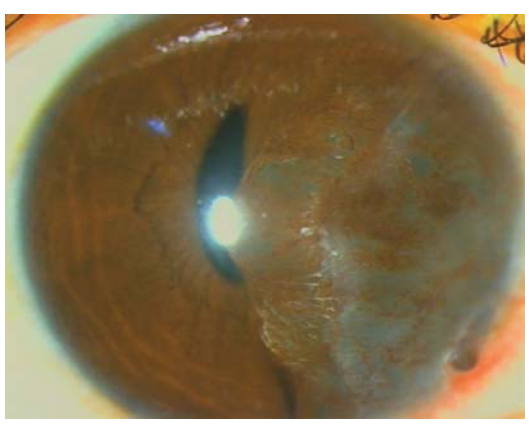


FIGURE 6.25.2: Iris cyst—post-surgical

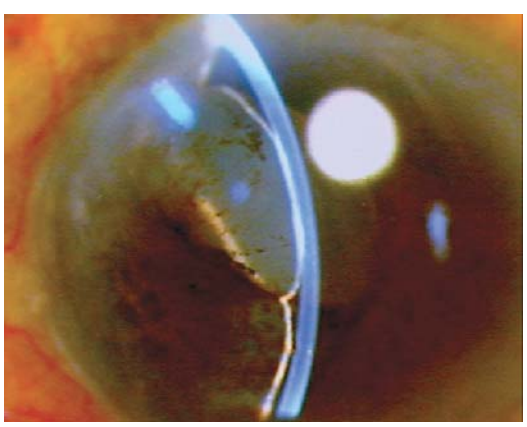


FIGURE 6.25.3: Iris cyst—clear fluid

Gyrate Atrophy of the Choroid

- Deficiency of ornithine ketoacid amino-transferase enzyme, resulting in hyper-ornithinemia
- Scalloped to circular patches of chorioretinal atrophy in the far and mid-retinal periphery **(Figs 6.26.1 and 6.26.2)**

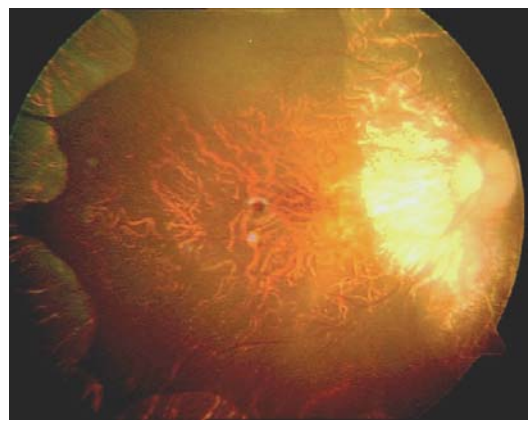


FIGURE 6.26.1: Gyrate atrophy of the choroid

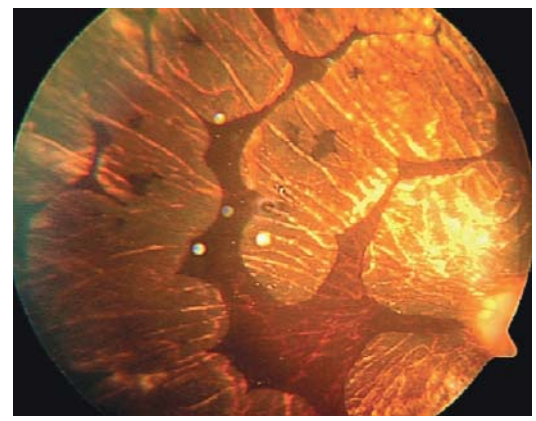


FIGURE 6.26.2: Gyrate atrophy of the choroid

Chorioderemia

- Rare, X-linked recessive inheritance
- Diffuse mottled depigmentation of the RPE **(Figs 6.27.1 and 6.27.2)**
- In late stage, extensive RPE and choroidal atrophy with sparing of fovea

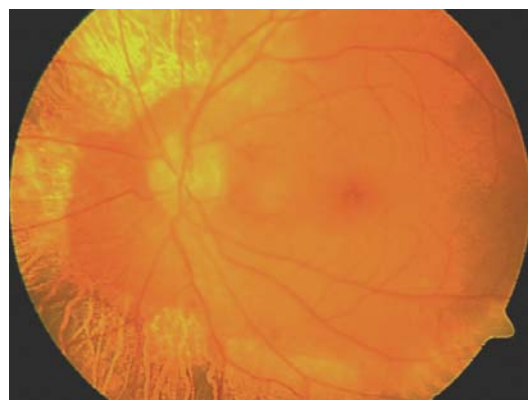


FIGURE 6.27.1: Choroideremia

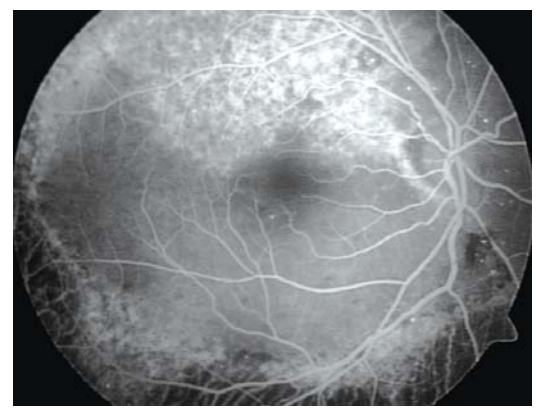


FIGURE 6.27.2: Choroideremia

Angioid Streaks

- Angioid streaks are irregular and jagged network of red to brown lines, mainly seen in the central fundus **(Fig 6.28.1)**
- Lesions are approximately the width of a retinal vessel, which may thus resemble 'angioid'
- But, the streaks are darker, have an irregular contour with serrated edges, and tend to terminate abruptly **(Fig 6.28.2)**

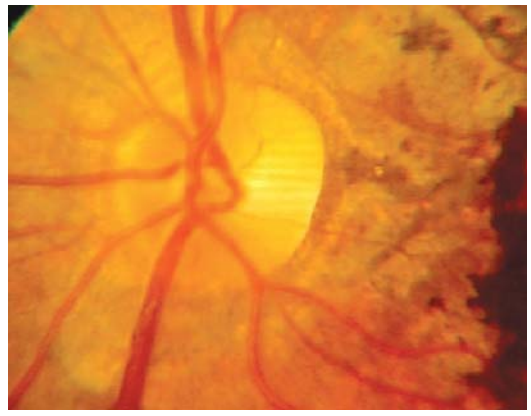


FIGURE 6.28.1: Angioid streaks

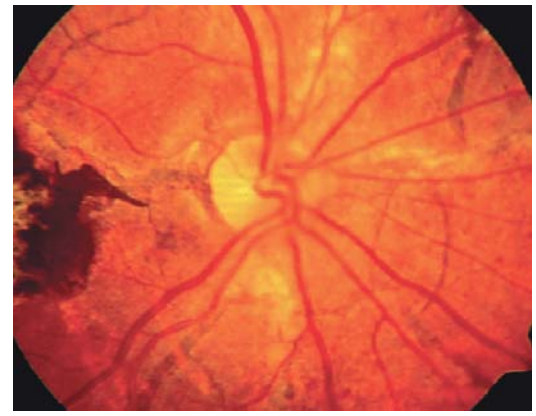


FIGURE 6.28.2: Angioid streaks

Acute Posterior Multifocal Placoid Pigment Epitheliopathy (APMPPE)

- Typical deep, cream-colored placoid lesions, involving the posterior pole within the equatorial region **(Figs 6.29.1 and 6.29.2)** *(See also Chapter 12)*

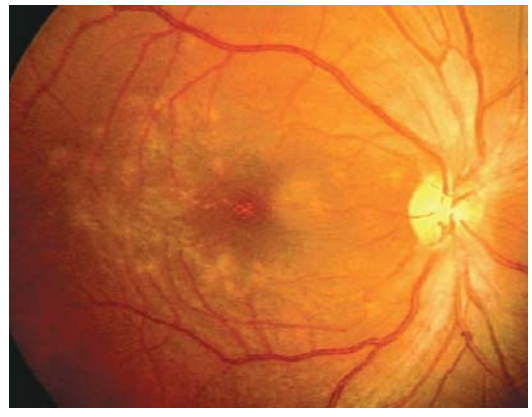


FIGURE 6.29.1: APMPPE

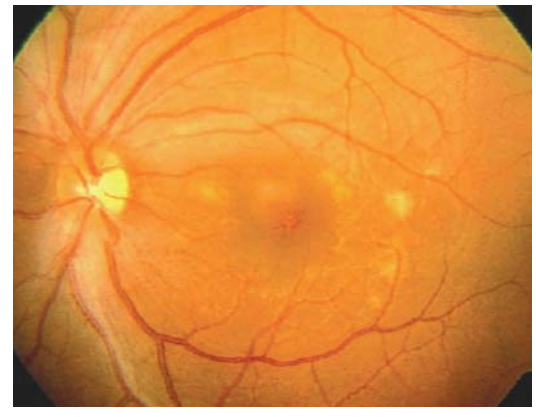


FIGURE 6.29.2: APMPPE

Serpiginous (geographical) Choroidopathy

- Lesion usually starts around the optic disk, and spreads outwards in all directions
- Cream-colored opacities with hazy border without any inflammatory cells in the vitreous **(Fig 6.30.1)**
- Fresh acute lesions usually arise as extensions from old scars, and successive attacks result in serpiginous extension of the destruction process peripherally from the peripapillary area **(Fig 6.30.2)**

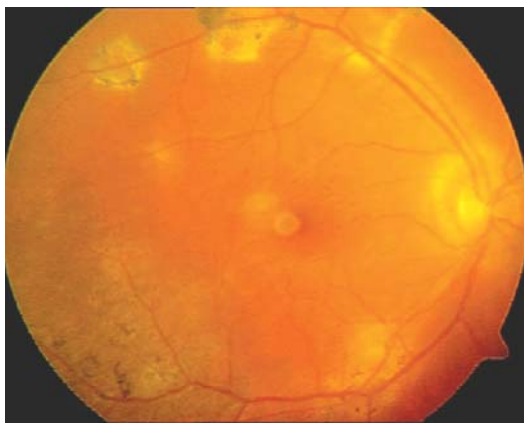


FIGURE 6.30.1: Serpiginous geographical choroidopathy

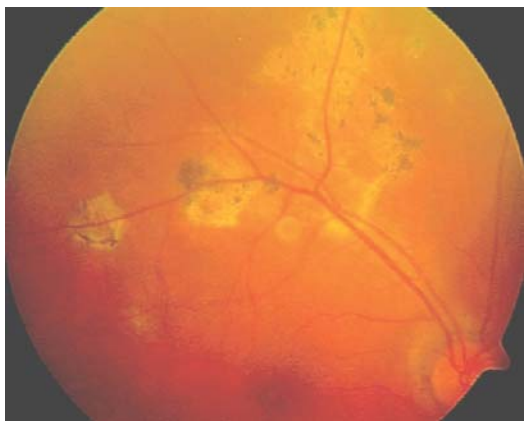


FIGURE 6.30.2: Serpiginous geographical choroidopathy

Choroidal Detachment

- Means separation of the choroids from the sclera
- Shallow anterior chamber
- Shows smooth, elevated, dark-brown bullous lesions, which are more prominent on the temporal and nasal sides **(Figs 6.31.1 and 6.31.2)**

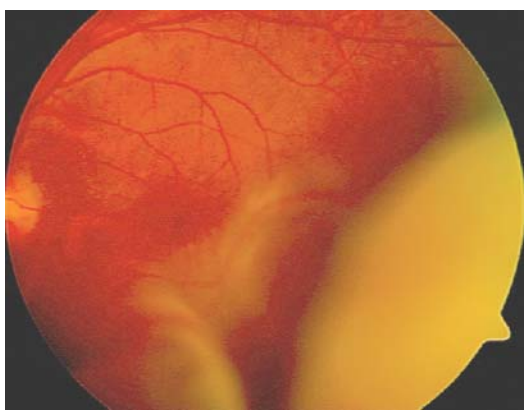


FIGURE 6.31.1: Choroidal detachment

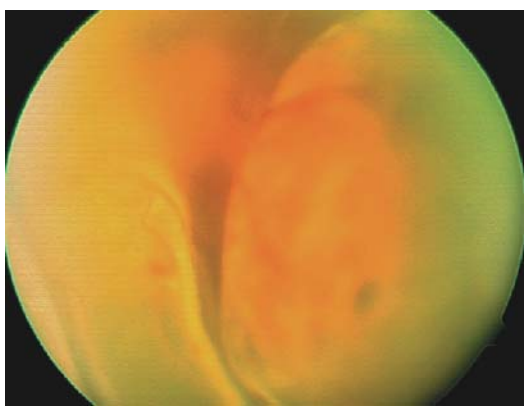


FIGURE 6.31.2: Choroidal detachment and retinal detachment

BENIGN UVEAL LESIONS/ NODULES/ MASS

Iris Floccules

- Bilateral, uncommon condition
- Pupillary tags or tiny solid pigmentary nodules around the pupil **(Fig 6.32.1)**

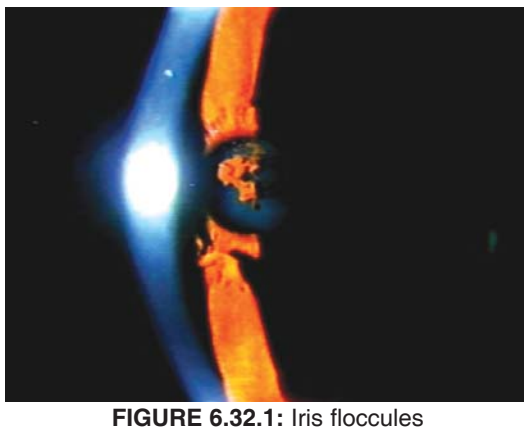


FIGURE 6.32.1: Iris floccules

Iris Freckles

- Bilateral tiny naevi on the iris surface
- Mainly seen in adults, rarely in children
- Multiple, discrete colorful spots on the anterior iris surface **(Fig 6.33.1)**

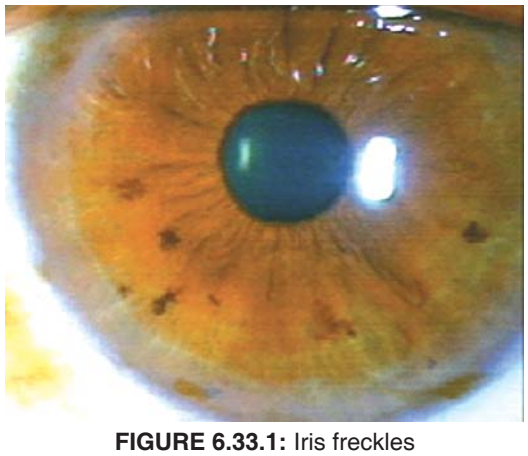


FIGURE 6.33.1: Iris freckles

Lisch Nodules (spots)

- Bilateral melanocytic hamartomas found in patients with neurofibromatosis after puberty **(Fig 6.34.1)**
- Small brown or yellowish brown dome-shaped spots **(Fig 6.34.2)** or nodules **(Fig 6.34.3)** on the anterior surface

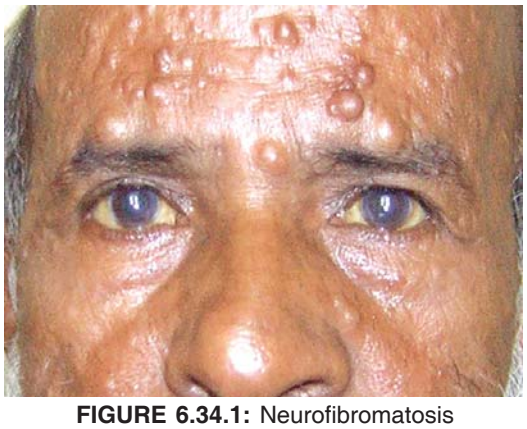


FIGURE 6.34.1: Neurofibromatosis

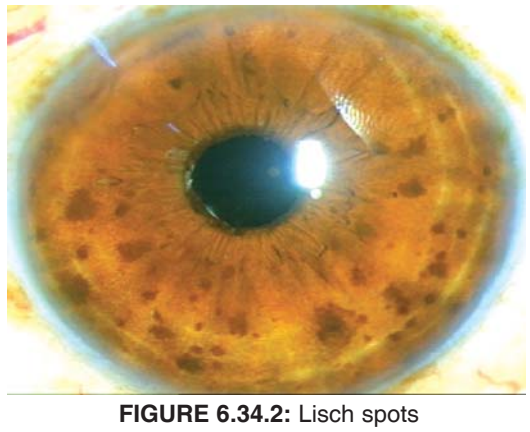


FIGURE 6.34.2: Lisch spots

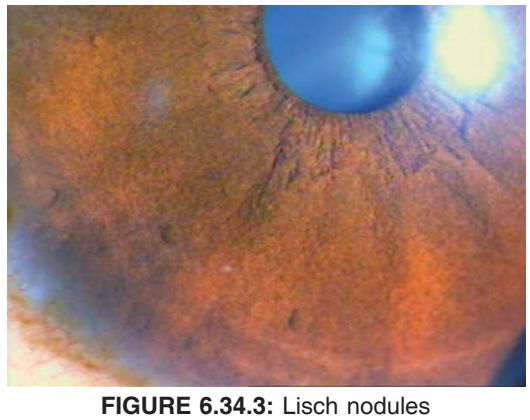


FIGURE 6.34.3: Lisch nodules

Brushfield Spots

- Bilateral, and are usually found in Down's syndrome, may be seen in general population **(Fig 6.35.1)**
- Tiny yellowish or white spots arranged in a ring at the junction of middle and outer third of iris surface

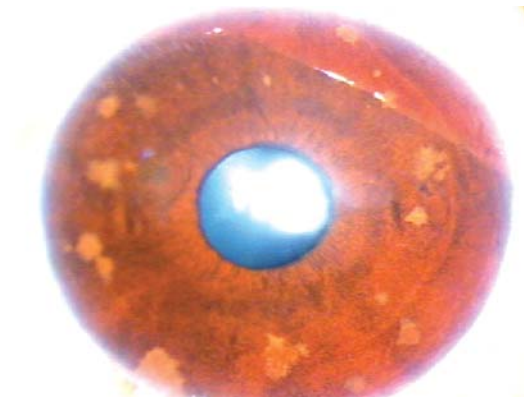


FIGURE 6.35.1: Brushfield spots

Iris Pearls

- Bilateral small or large, pearl-like clear nodules on the iris surface and pupillary margin **(Fig 6.36.1)**
- May be seen in lepromatous leprosy
- May drop into AC and eventually disappear

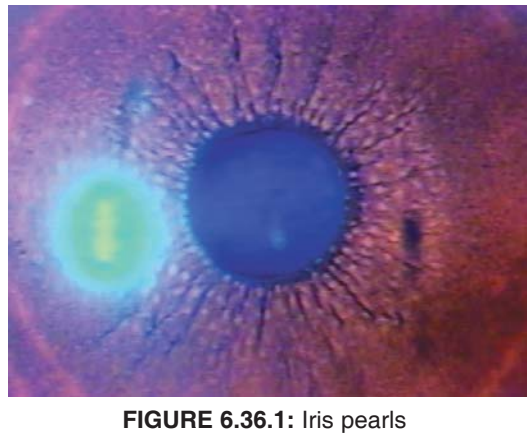


FIGURE 6.36.1: Iris pearls

Iris Mammiliations

- Unilateral or bilateral rare condition
- Tiny, smooth, villiform lesions which are regularly spaced **(Fig 6.37.1)**
- May found in normal people or with some syndromes

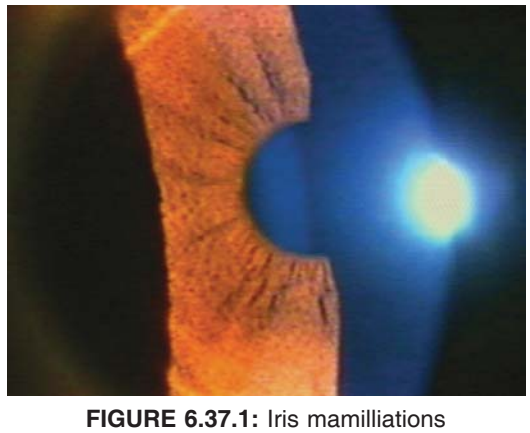


FIGURE 6.37.1: Iris mamilliations

Iris Naevus

- Common, usually unilateral
- Elevated or flat, localized, discrete, small pigmented mass **(Fig 6.38.1)**
- Usually does not distort the pupil
- May cause heterochromia iridis **(Fig 6.38.2)**

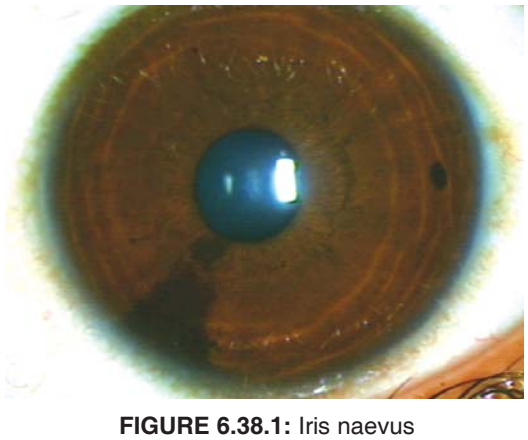


FIGURE 6.38.1: Iris naevus

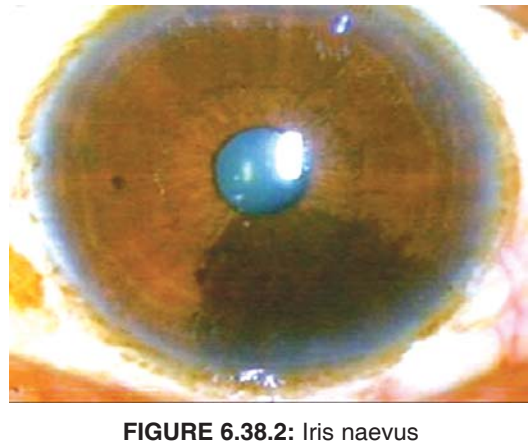


FIGURE 6.38.2: Iris naevus

Choroidal Hemangioma

- In 50 percent cases associated with skin angioma, as in Sturge-Weber syndrome **(Fig 6.39.1)**
- Appears as a dome-shaped or diffuse, reddish-orange lesion mostly at the posterior pole

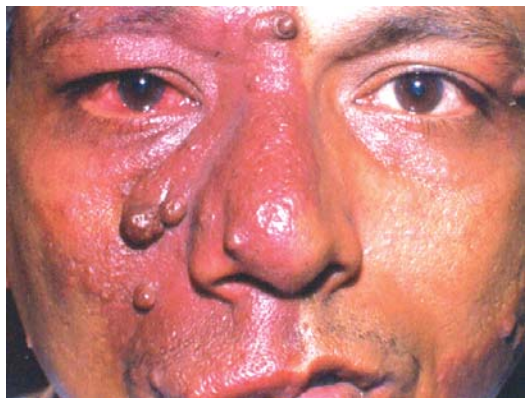


FIGURE 6.39.1: Sturge-Weber syndrome

Benign Melanocytoma (naevus)

- Typical benign melanomas of the choroid are flat or slightly elevated, oval or circular state-gray lesions **(Fig 6.40.1)**
- Occur most frequently at the posterior half of the fundus
- *Treatment:* not indicated, except the patient should be followed up regularly with serial photographs

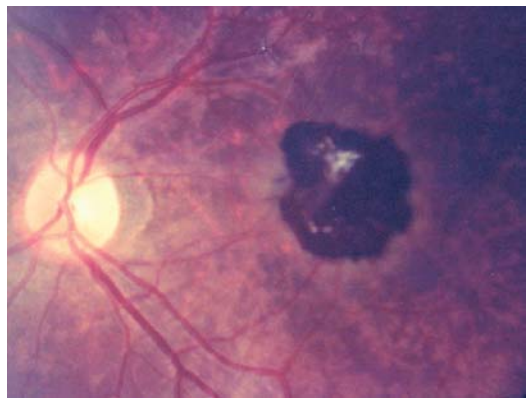


FIGURE 6.40.1: Benign melanocytoma of choroid

Choroidal Osteoma

- Very rare, juxtapapillary lesion, that typically affects the young females
- Yellowish-orange lesion with scalloped borders adjacent to the optic disk **(Fig 6.41.1)**

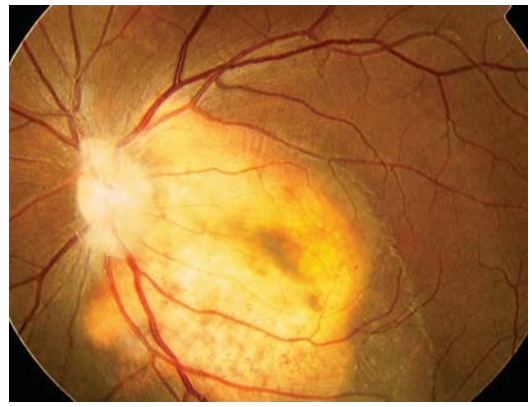


FIGURE 6.41.1: Choroidal osteoma

MALIGNANT UVEAL TRACT LESIONS

Metastatic Deposits of Retinoblastoma

- Rare presentation of retinoblastoma
- Pale-white multiple iris nodules, often associated with pseudo-hypopyon **(Fig 6.42.1)**

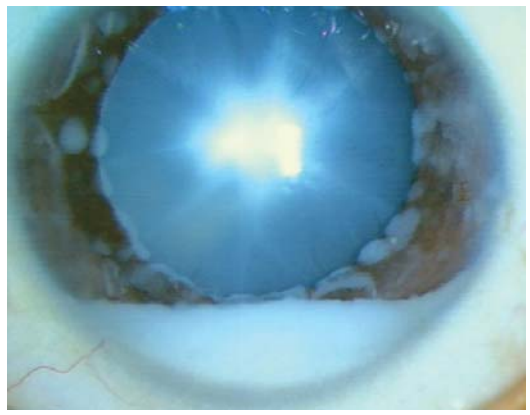


FIGURE 6.42.1: Retinoblastoma—metastatic deposits

Malignant Melanoma of the Iris

- Very rare, slow-growing tumor with relative low malignant potential
- Noticed as a brown or non-pigmented mass on the iris surface, usually located in the inferior half of the iris **(Fig 6.43.1)**
- May start at the angle of AC **(Fig 6.43.2)**
- Pupil is distorted; ectropion uveae and secondary lens opacities are seen
- May extend into the anterior chamber angle giving rise to secondary glaucoma **(Fig 6.43.3)**

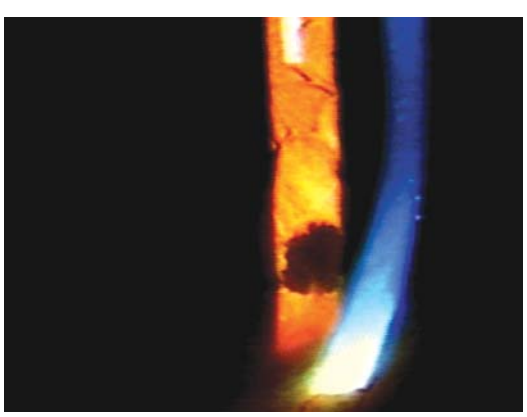


FIGURE 6.43.1: Malignant melanoma—iris

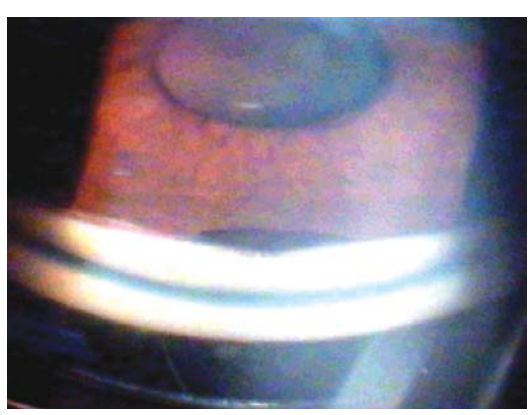


FIGURE 6.43.2: Iris melanoma—angle

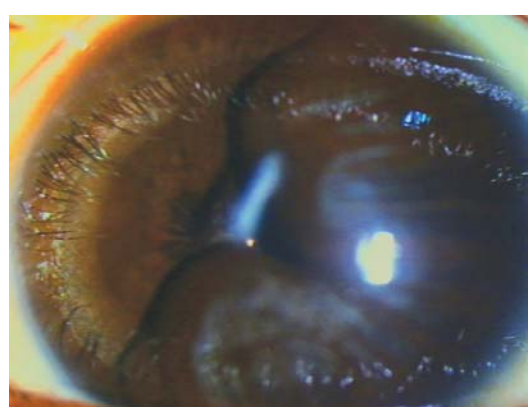


FIGURE 6.43.3: Malignant melanoma—iris

- *Forward extension of the ciliary body melanoma*
  - may not be confined to inferior half **(Figs 6.43.4 and 6.43.5)**
  - examination after dilatation confirms the diagnosis

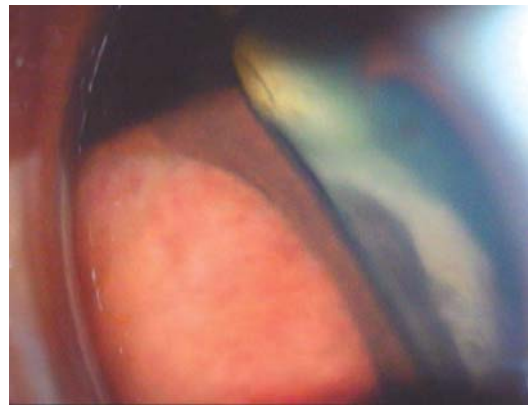


FIGURE 6.43.4: Ciliary body melanoma

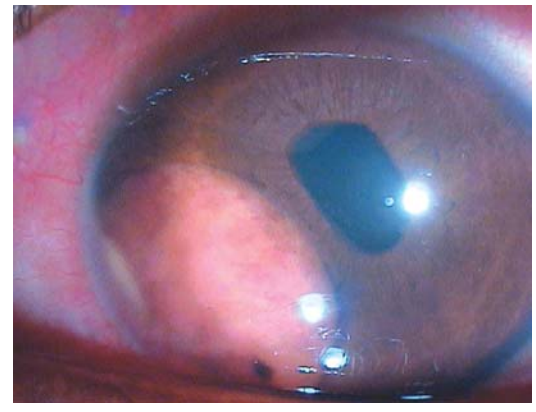


FIGURE 6.43.5: Ciliary body melanoma—extension

Medullo-epithelioma (diktyoma)

- Extremely rare, arises from the non-pigmented epithelium of the ciliary body
- Presents with a white pupil (leukocoria), secondary cataract and secondary glaucoma

Malignant Melanoma of the Ciliary Body

- Ciliary body melanoma is more common than iris melanoma
- *May present:*
  - as subluxation with secondary glaucoma
  - as dilated episcleral blood vessels ('sentinel vessels')
  - as a diffuse mass around the ciliary body **(Fig 6.44.1)**
- *Treatment:* by enucleation (for large tumors) and local resection (for small tumors)

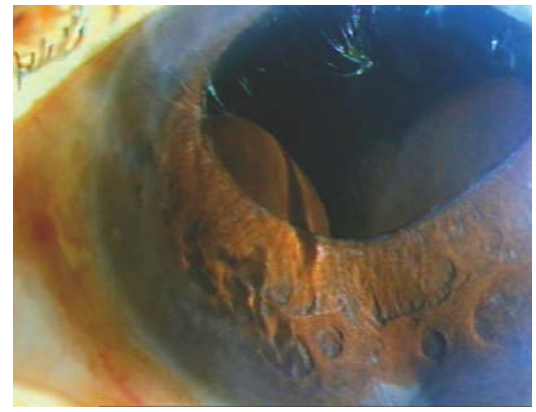


FIGURE 6.44.1: Ciliary body melanoma

Malignant Melanoma of the Choroid

- Chief symptoms result from the exudative retinal detachment with secondary glaucoma
- Typically appears as a pigmented and elevated oval mass **(Fig 6.45.1)**
- As the tumor grows, a brown exudative detachment results **(Figs 6.45.3 to 6.45.5)**
- *Treatment*: small melanoma can be treated by laser **(Fig 6.45.2),** for large melanoma—enucleation

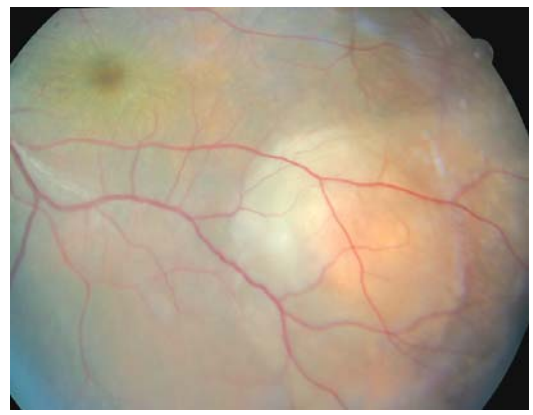


FIGURE 6.45.1: Choroidal melanoma

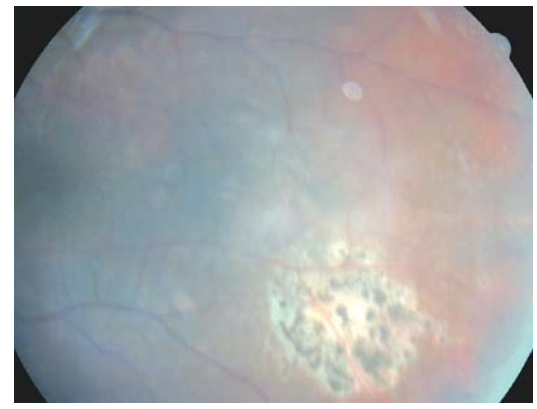


FIGURE 6.45.2: Choroidal melanoma—post-laser

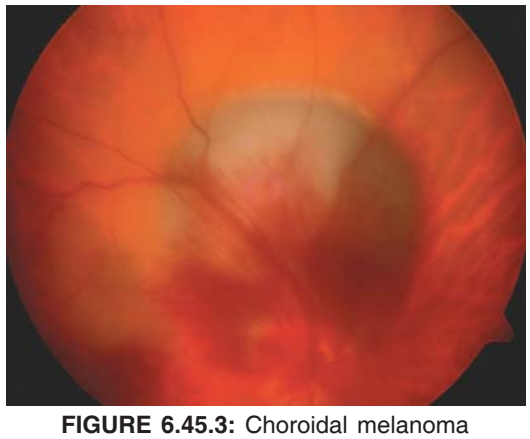


FIGURE 6.45.3: Choroidal melanoma

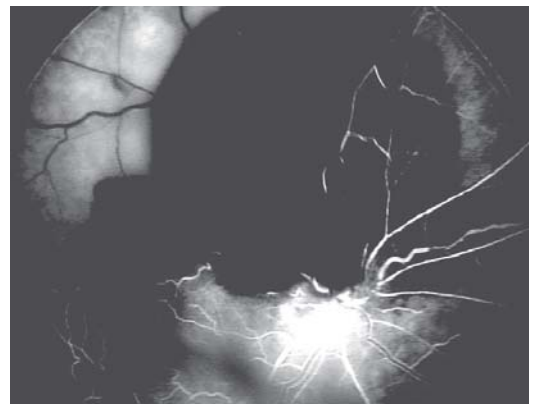


FIGURE 6.45.4: Choroidal melanoma—FFA

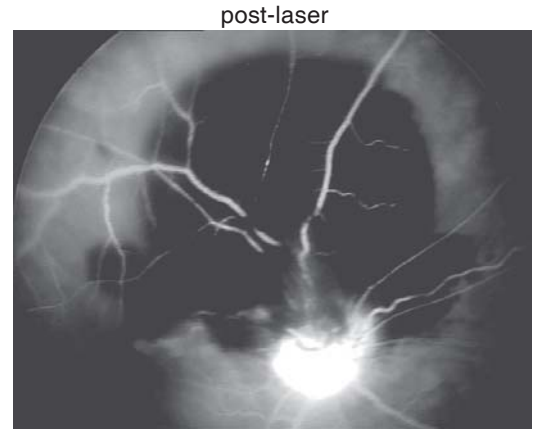


FIGURE 6.45.5: Choroidal melanoma—FFA

Metastatic Carcinoma of Uveal Tract

- *Most frequent primary sites:* bronchus in males, and breast in females
- May deposit on iris, ciliary body or choroid **(Fig 6.46.1)**
- Typically, appear as solitary or multiple, creamy-white, placoid or oval lesions which infiltrate laterally **(Figs 6.46.2 and 6.46.3)**
- Careful examination of the other *eye* is important as bilateral metastases are common
- *Treatment:* must be directed to the primary disease. Enucleation is contraindicated unless the *eye* is painful and blind

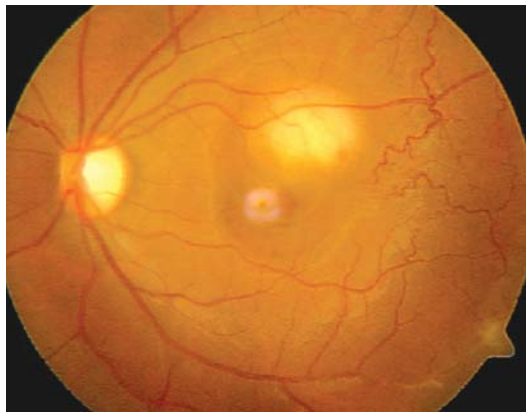


FIGURE 6.46.1: Metastatic carcinoma of choroid— from breast

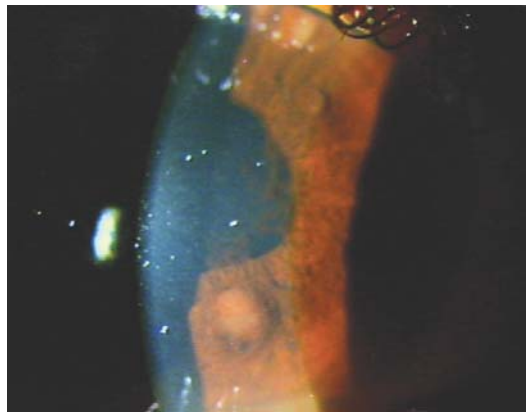


FIGURE 6.46.2: Metastatic carcinoma of iris— from bronchus

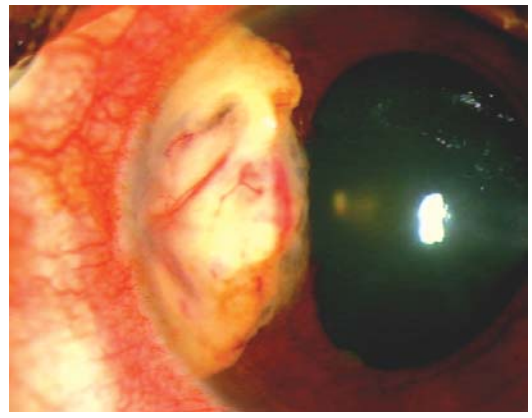


FIGURE 6.46.3: Metastatic carcinoma of iris— from breast

7

Pupil and its Abnormalities

PUPILLARY ANOMALIES

- Acoria
- Corectopia
- Polycoria
- Persistent pupillary membrane
- Small (miotic) pupil
- Large (mydriatic) pupil
- Abnormalities in shape of the pupil
- Anisocoria

LEUKOCORIA OR WHITE PUPILLARY (AMAUROTIC CAT'S EYE) REFLEX

- Congenital cataract
- Retinoblastoma
- Retinopathy of prematurity
- Toxocara endophthalmitis
- Persistent hyperplastic primary vitreous (PHPV)
- Retinal dysplasia
- Coats' disease
- Choroidal coloboma

PUPILLARY ANOMALIES

Acoria

- Means absence of pupil
- *Congenital*: very rare, bilateral or unilateral condition (**Fig 7.1.1**)
  - anterior chamber remained formed by aqueous drained via micro-openings in the pupillary area
- *Acquired*: also very rare and unilateral **(Fig 7.1.2**)
  - patient ultimately develops hypotony and atrophic bulbi

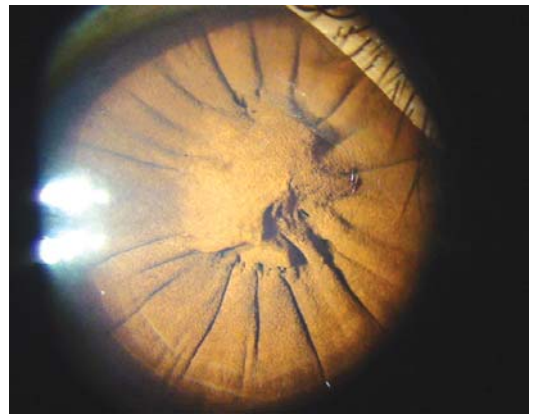


FIGURE 7.1.1: Acoria—congenital

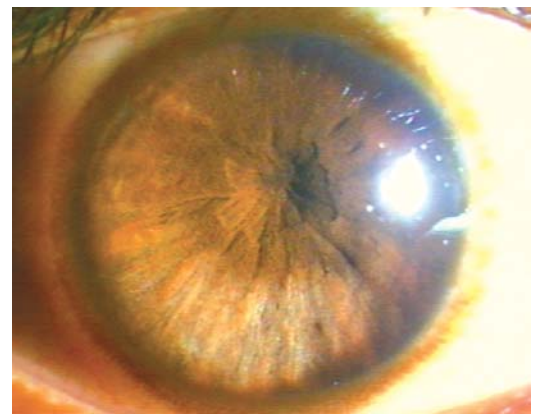


FIGURE 7.1.2: Acoria—acquired

Corectopia

- Displacement of the pupil from its normal position, usually more to the nasal side **(Figs 7.2.1 to 7.2.4)**
- May be associated with ectopia lentis **(Fig 7.2.5)**, coloboma iris **(Fig 7.2.6)**, irido-corneal endothelial (ICE) syndrome **(Fig 7.2.7)**, after trauma or after cataract surgery **(Fig 7.2.8)**

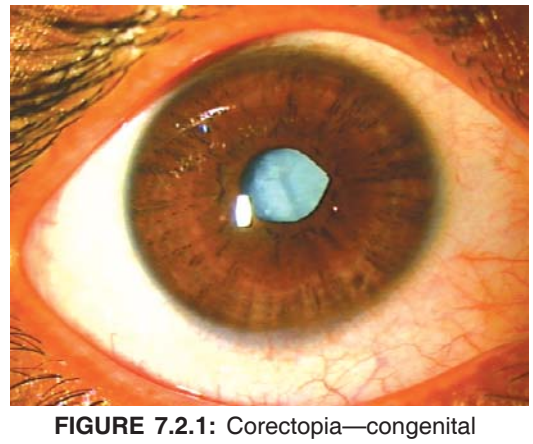


FIGURE 7.2.1: Corectopia—congenital

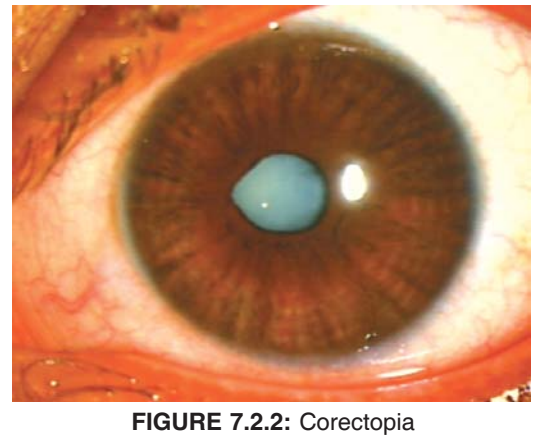


FIGURE 7.2.2: Corectopia

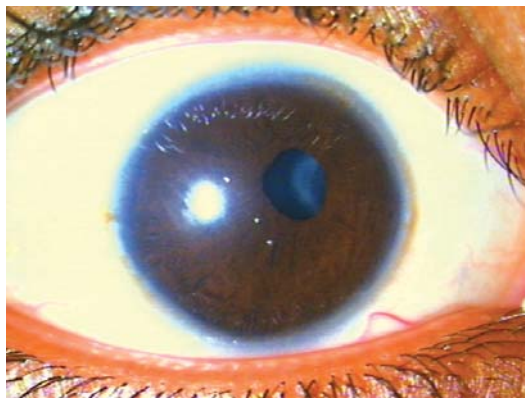


FIGURE 7.2.3: Corectopia—congenital

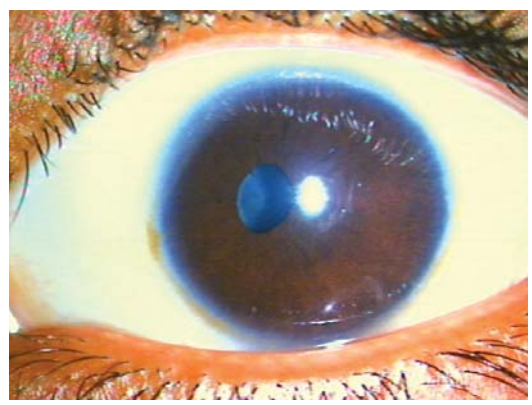


FIGURE 7.2.4: Corectopia—congenital

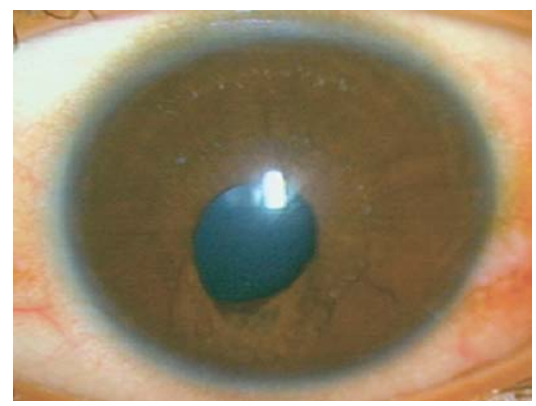


FIGURE 7.2.5: Corectopia—ectopia lentis et pupillae

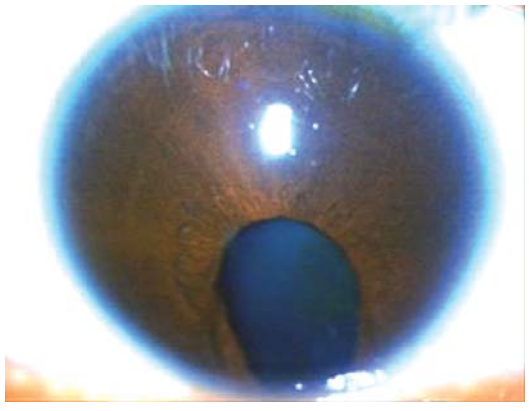


FIGURE 7.2.6: Corectopia—iris coloboma

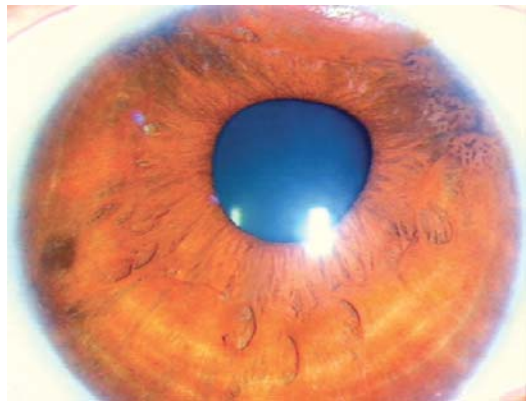


FIGURE 7.2.7: Corectopia—ICE syndrome

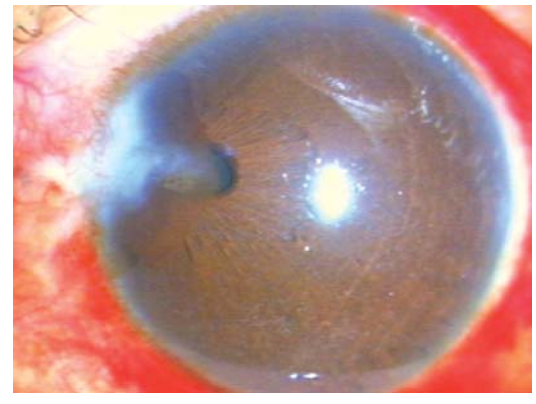


FIGURE 7.2.8: Corectopia—post-traumatic

Polycoria

- Multiple pupils
- *True polycoria:* extremely rare, multiple pupils, each having a sphincter muscle
- *Pseudopolycoria:* not uncommon, found in ICE syndrome—mostly in essential iris atrophy **(Figs 7.3.1 to 7.3.4)**, trauma and after surgery **(Fig 7.3.5)**

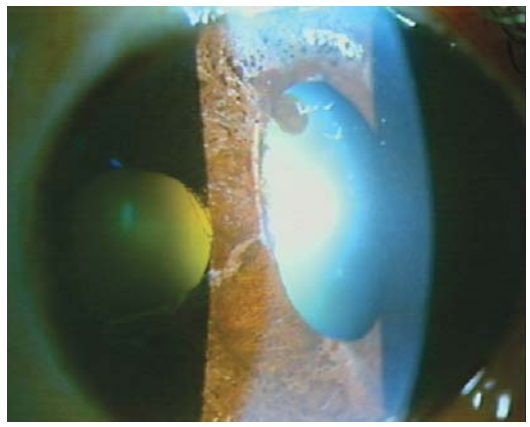


FIGURE 7.3.1: Pseudopolycoria—ICE syndrome

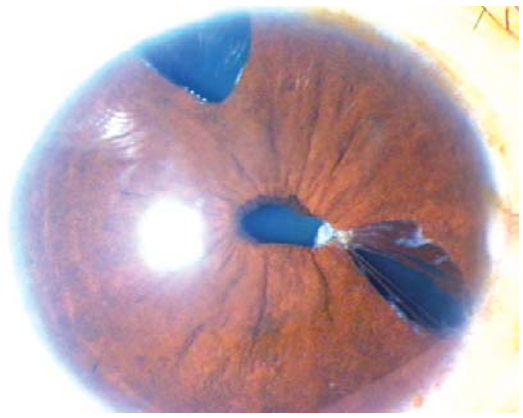


FIGURE 7.3.2: Pseudopolycoria—ICE syndrome

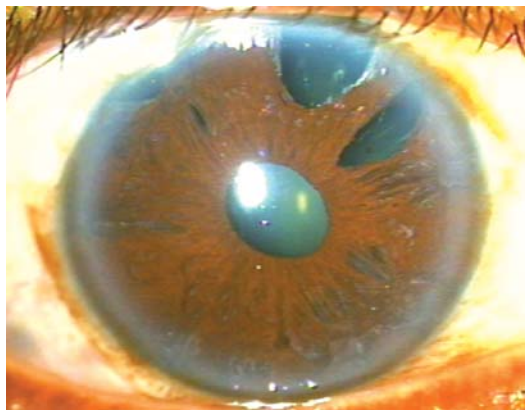


FIGURE 7.3.3: Pseudopolycoria

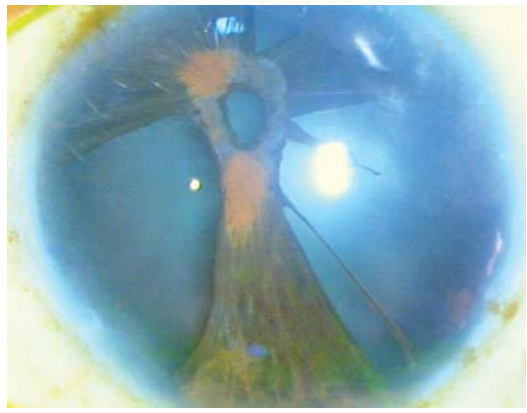


FIGURE 7.3.4: Pseudopolycoria—ICE syndrome

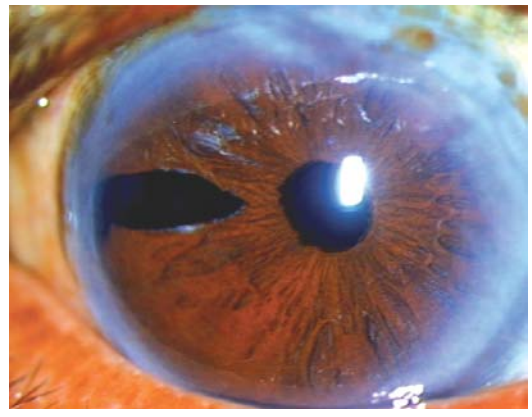


FIGURE 7.3.5: Pseudopolycoria—surgical

Persistent Pupillary Membrane

- Is the continued existence of the part of anterior vascular sheath of the lens, a fetal structure, which normally disappears shortly before birth **(Fig 7.4.1)**
- Appears as fine strands of membrane, running inwards from the collarette inserting into the anterior lens capsule **(Fig 7.4.2)**
- Usually, it does not interfere with vision
- Rarely, it may block the pupil, and surgical intervention is then necessary **(Figs 7.4.3 and 7.4.4)**

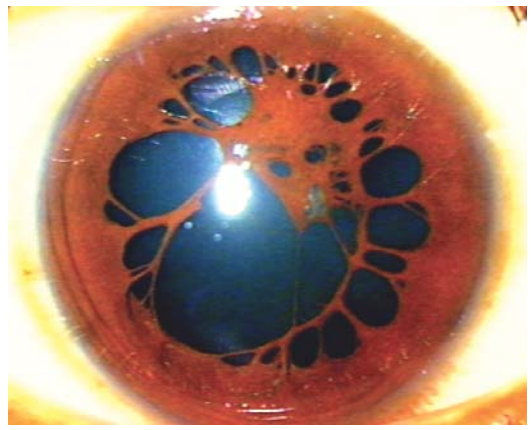


FIGURE 7.4.1: Persistent pupillary membrane

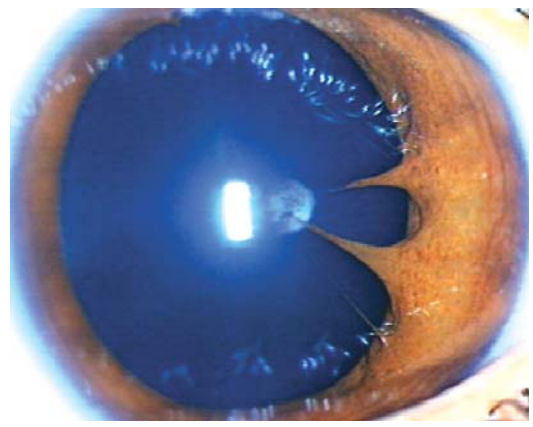


FIGURE 7.4.2: Persistent pupillary membrane

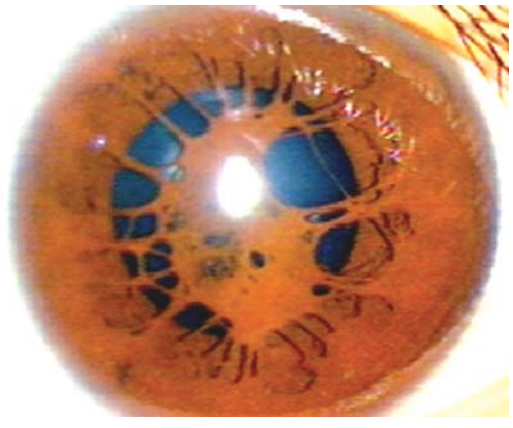


FIGURE 7.4.3: Persistent pupillary membrane—blocking the pupil

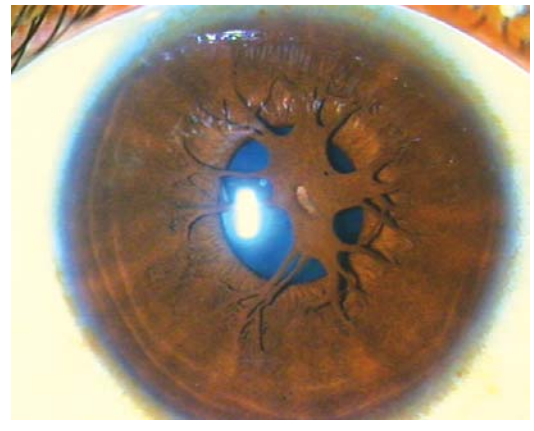


FIGURE 7.4.4: Persistent pupillary membrane—blocking the pupil

Small (miotic) Pupil

- Normally it varies between 2-4 mm, but depends on level of illumination
- Pupil size less than 2 mm
- *Causes:*
  - extreme of ages **(Fig 7.5.1)**
  - in bright light
  - opium/morphine addict **(Fig 7.5.2)**
  - pontine hemorrhage
  - healed iridocyclitis **(Fig 7.5.3)**
  - use of miotics (e.g., pilocarpine) **(Fig 7.5.4)**
  - pseudoexfoliation **(Fig 7.5.5)**
  - during sleep

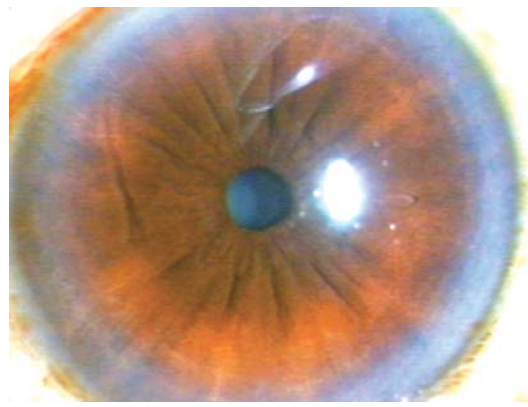


FIGURE 7.5.1: Miotic pupil—aging

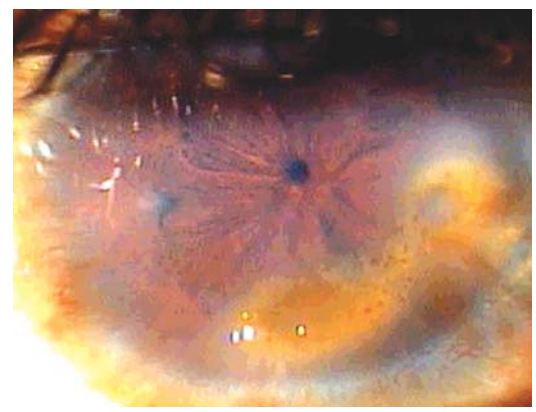


FIGURE 7.5.2: Extreme miosis—opium addict

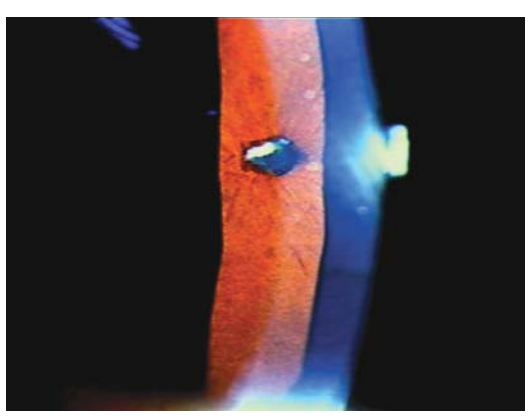


FIGURE 7.5.3: Miosis—healed iritis

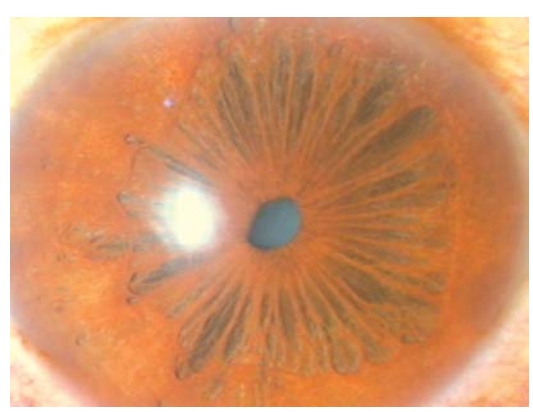


FIGURE 7.5.4: Miosis—pilocarpine

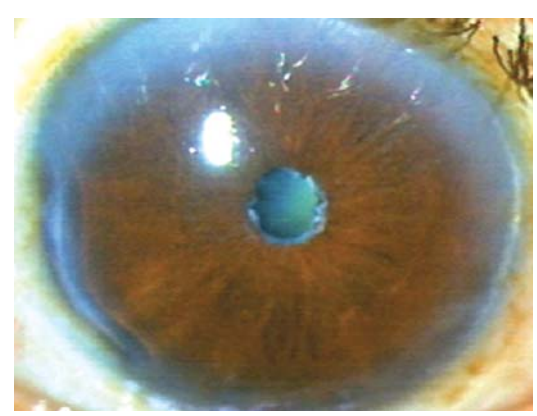


FIGURE 7.5.5: Miosis—pseudoexfoliation syndrome

Large (mydriatic) Pupil

- Pupil size is 6 mm or more
- Causes:
  - in dark
  - optic atrophy **(Figs 7.6.1 and 7.6.2)**
  - absolute glaucoma **(Fig 7.6.3)**
  - comatose patient/head injury
  - third nerve palsy
  - post-traumatic **(Fig 7.6.4)**
  - use of mydriatics **(Fig 7.6.5)**

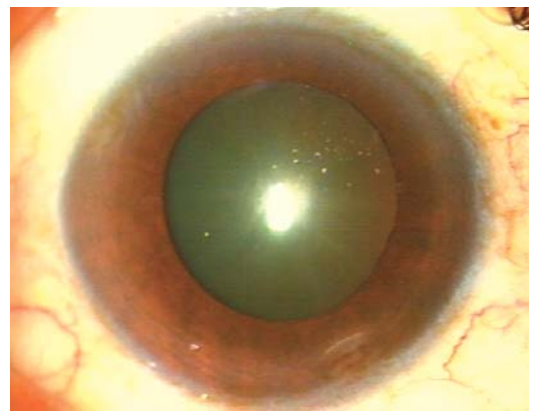


FIGURE 7.6.1: Mydriasis—optic atrophy

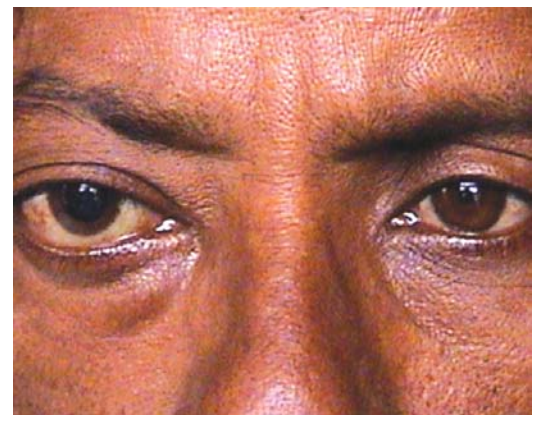


FIGURE 7.6.2: Mydriasis—optic atrophy

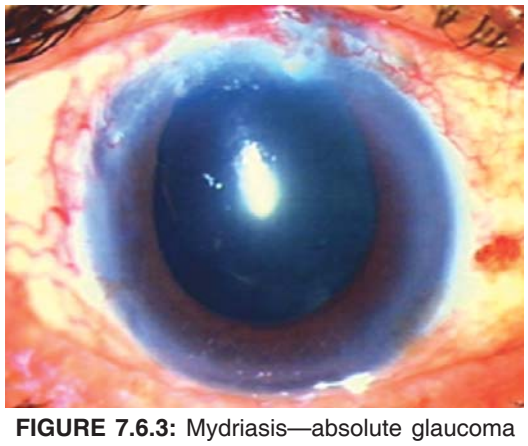


FIGURE 7.6.3: Mydriasis—absolute glaucoma

FIGURE 7.6.4: Mydriasis—post-traumatic with subluxation

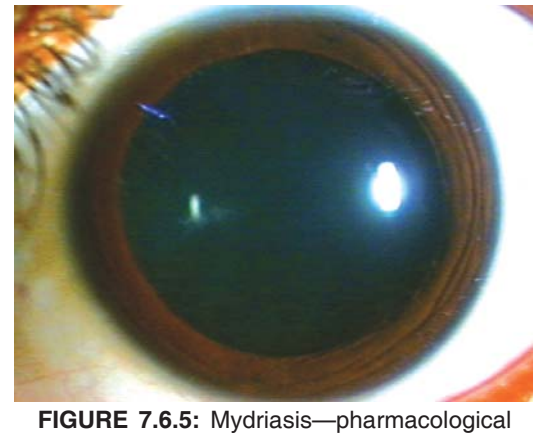


FIGURE 7.6.5: Mydriasis—pharmacological

Abnormalities in Shape of the Pupil

- *Irregular:* iritis **(Fig 7.7.1)** or post-traumatic, surgical sphincterotomy **(Fig 7.7.2)**
- *D-shaped:* iridodialysis **(Fig 7.7.3)**
- *Boat- or Hammock-shaped:* vitreous loss in cataract surgery
- *Pear-shaped:* incarceration of iris with the wound—traumatic or after surgery **(Figs 7.7.4 and 7.7.5)**
- *Festooned:* iridocyclitis **(Fig 7.7.6)**
- *Inverted pear-shaped:* coloboma *(inferonasal)* of the iris **(Fig 7.7.7),** penetrating injury with iris prolapse**(Fig 7.7.8)**
- *Mid-dilated and oval:* acute ACG **(Fig 7.7.9)**
- *Elliptical, triangular or square*: IOL-optic capture **(Figs 7.7.10 to 7.7.12)**
- *Updrawn pupil:* after cataract surgery **(Fig 7.7.13)**
- *Slit-like*: Axenfeld-Reiger's syndrome **(Fig 7.7.14)** and IOL-optic capture **(7.7.15)**
- *Large semicircular and up:* complete iridectomy in cataract surgery **(Fig 7.7.16)**

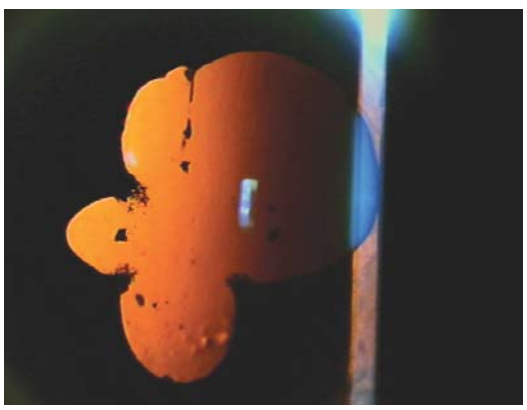


FIGURE 7.7.1: Irregular pupil—iritis

FIGURE 7.7.2: Irregular pupil—sphincterotomy

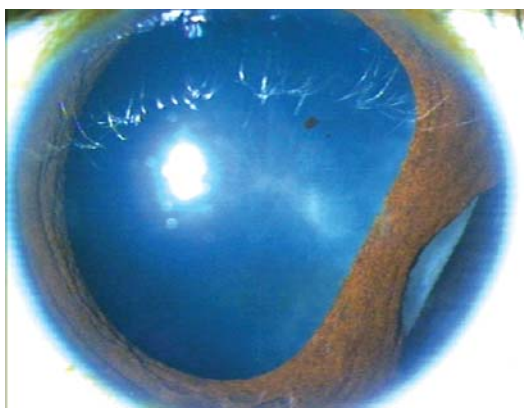


FIGURE 7.7.3: D-shaped pupil—iridodialysis

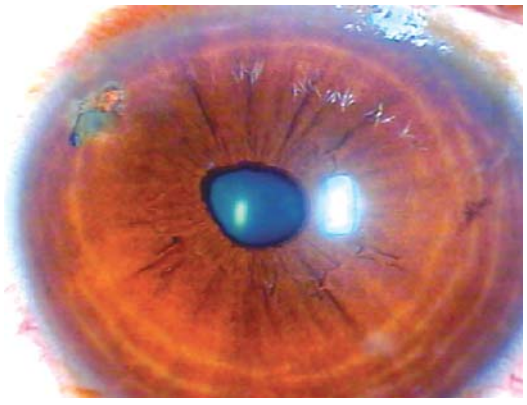


FIGURE 7.7.4: Pear-shaped pupil—iris trauma

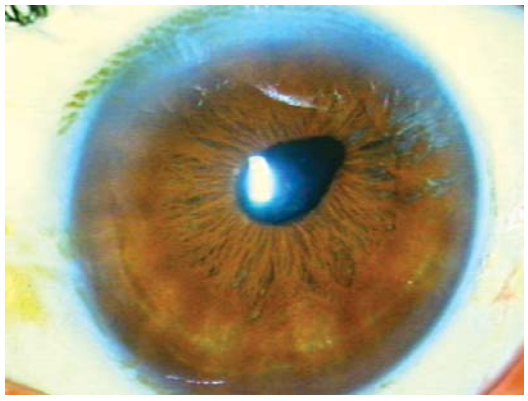


FIGURE 7.7.5: Pear-shaped pupil—post-surgical

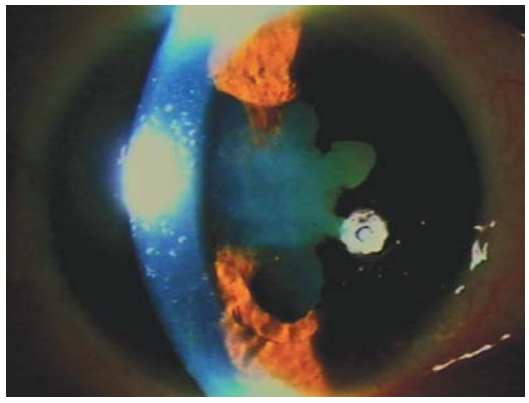


FIGURE 7.7.6: Festooned pupil—iridocyclitis

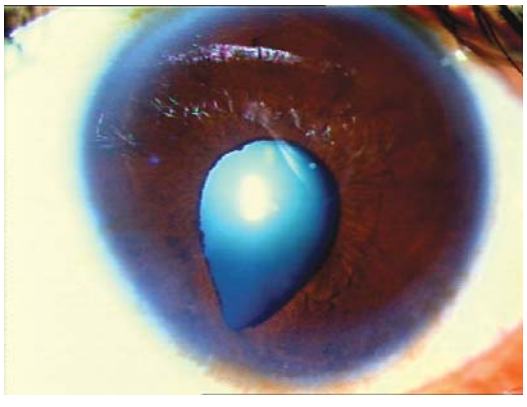


FIGURE 7.7.7: Inverted pear-shaped—incomplete coloboma

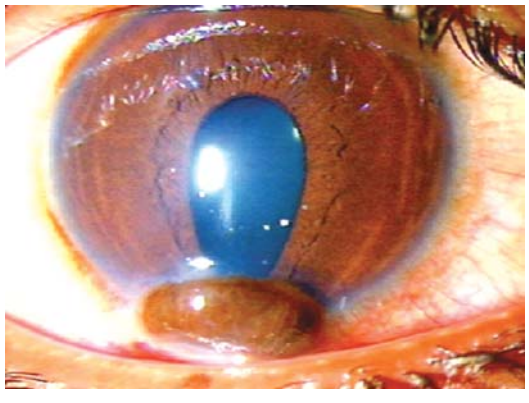


FIGURE 7.7.8: Inverted pear-shaped—perforating injury

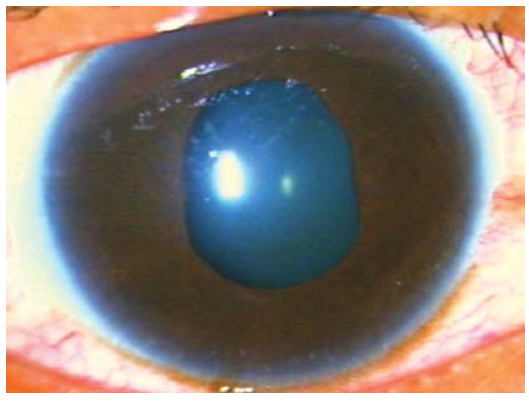


FIGURE 7.7.9: Oval pupil—acute attack ACG

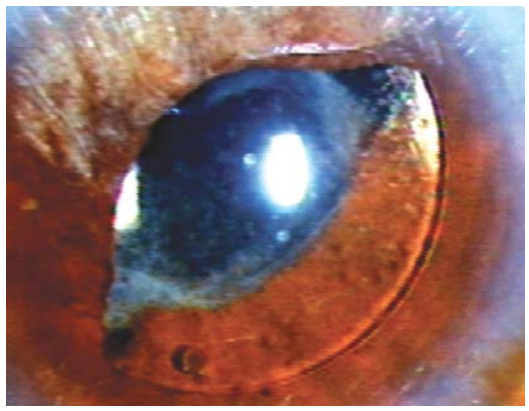


FIGURE 7.7.10: Elliptical pupil—IOL optic catch

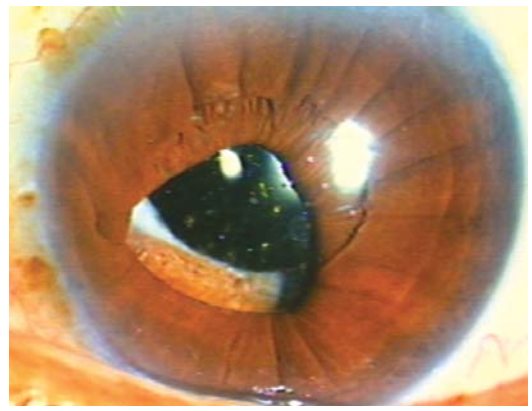


FIGURE 7.7.11: Triangular pupil—iris catch

FIGURE 7.7.12: Square pupil—IOL optic catch

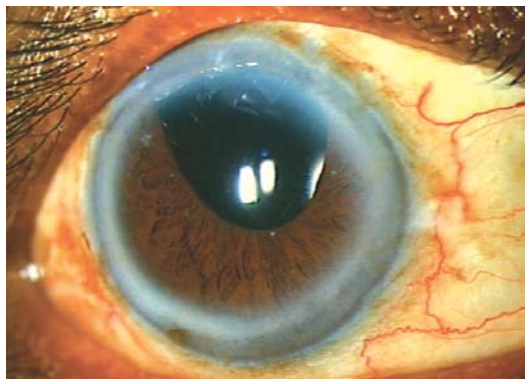


FIGURE 7.7.13: Updrawn pupil—cataract surgery

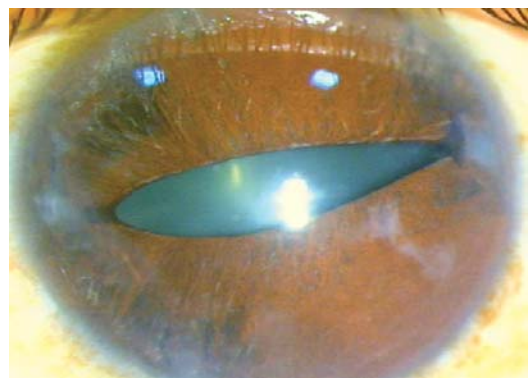


FIGURE 7.7.14: Slit-like pupil—Axenfeld Reiger's syndrome

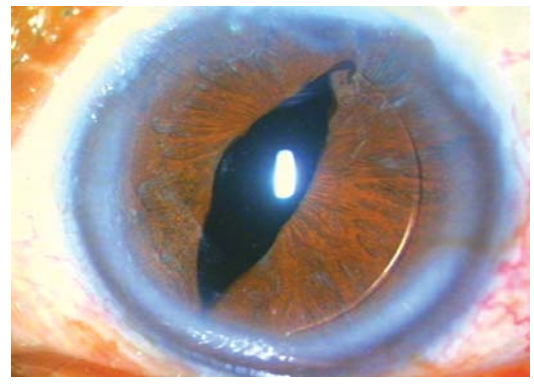


FIGURE 7.7.15: Slit-like pupil—IOL optic capture

Anisocoria

- Unequal size of the pupil between two eyes
- Pupil of one eye is normal, and the other eye is either miotic or mydriatic
- *Causes:*
  - Horner's syndrome
  - Adie's pupil
  - 3rd nerve palsy **(Fig 7.8.1)**
  - use of miotic or mydriatic in one eye **(Fig 7.8.2)**
  - springing pupil **(Fig 7.8.3)**
  - optic atrophy in one eye

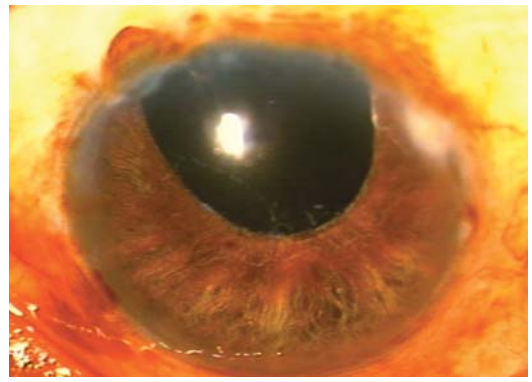


FIGURE 7.7.16: Pupil after complete iridectomy in aphakia

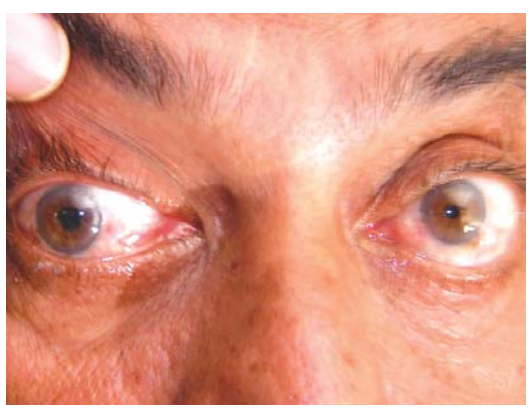


FIGURE 7.8.1: Anisocoria—third nerve palsy

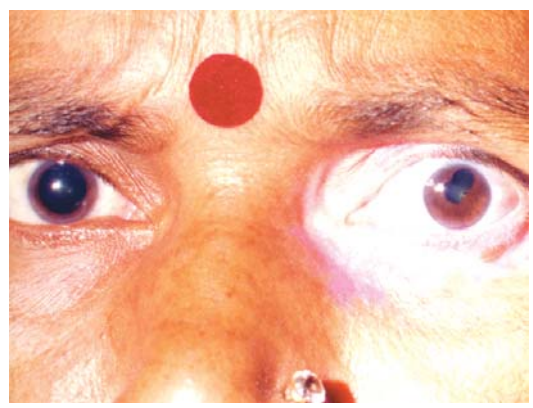


FIGURE 7.8.2: Anisocoria—iritis

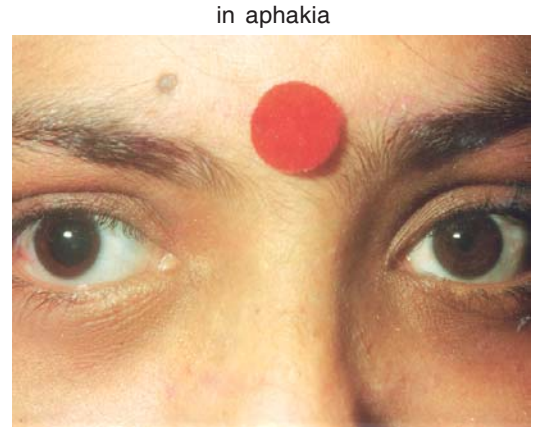


FIGURE 7.8.3: Anisocoria—springing pupil

LEUKOCORIA OR WHITE PUPILLARY (AMAUROTIC CAT'S EYE) REFLEX

- Relatively common condition
- May be unilateral or bilateral
- *Common causes:* congenital cataract, retinoblastoma, retinopathy of prematurity, toxocara endophthalmitis, persistent hyperplastic primary vitreous, retinal dysplasia, Coats' disease, choroidal coloboma, etc.
- ***Congenital cataract***
  - unilateral or bilateral **(Fig 7.9.1)**
  - opacity in the lens clearly indicates the presence of cataract **(Fig 7.9.2)**
  - discussed in *Chapter: 8*
- ***Retinoblastoma***
  - unilateral, progressive, malignant condition
  - usual age at diagnosis is 18 months **(Figs 7.9.3 and 7.9.4)**
  - no inflammatory sign in anterior segment
  - ophthalmoscopy shows, a pearly-white mass with presence of secondary calcification **(Fig 7.9.5)**
  - lens is usually transparent
  - intraocular pressure is high
- ***Retinopathy of prematurity***
  - prematurity and low birth weight with history of prolonged exposure to oxygen
  - bilateral in 100 percent of cases **(Fig 7.9.6)**
  - first noted in neonatal period
  - presence of tractional retinal detachment **(Fig 7.9.7)**
  - Intraocular pressure is normal
- ***Toxocara endophthalmitis***
  - unilateral with history of contact with pet cat or dog
  - presentation is between 2 to 9 years of age
  - signs of inflammation in anterior segment and vitreous **(Fig 7.9.8)**
  - retinal detachment, low intraocular pressure and eventually, the eye may be phthisical **(Figs 7.9.9 and 7.9.10)**

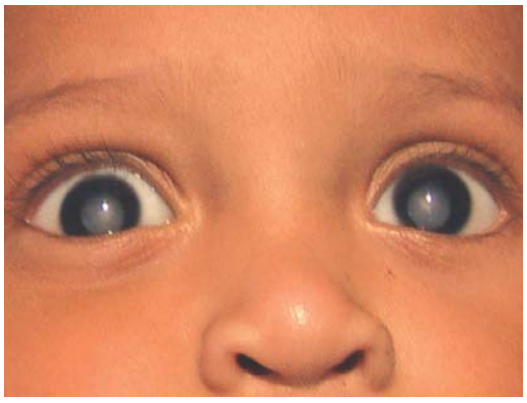


FIGURE 7.9.1: Leukocoria—congenital cataract

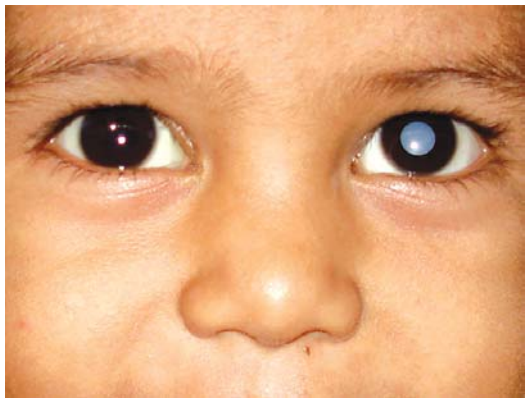


FIGURE 7.9.2: Leukocoria—congenital cataract—RE operated

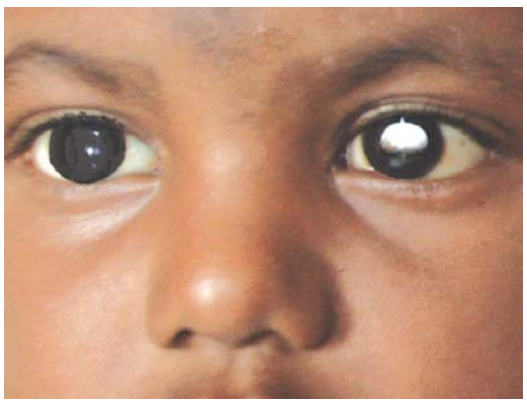


FIGURE 7.9.3: Leukocoria—retinoblastoma

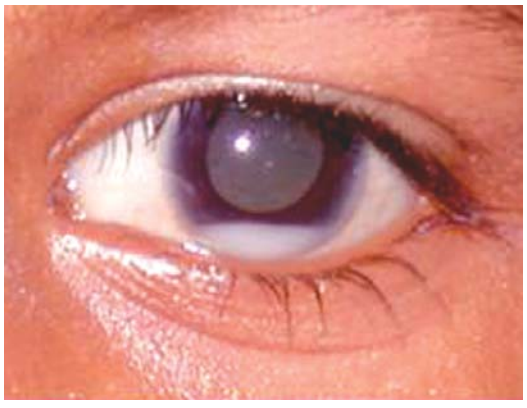


FIGURE 7.9.4: Leukocoria—retinoblastoma—pseudohypopyon

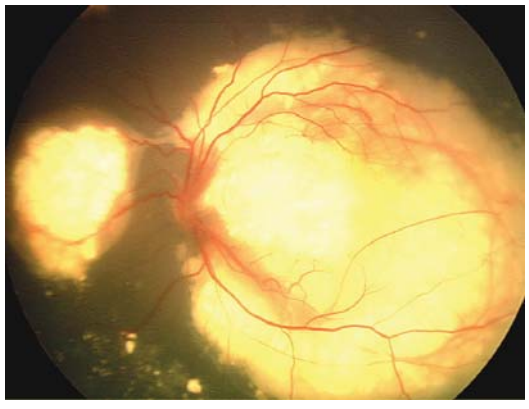


FIGURE 7.9.5: Leukocoria—retinoblastoma—endophytic lesion

FIGURE 7.9.6: Leukocoria—retinopathy of prematurity

- *Persistent hyperplastic primary vitreous (PHPV)*
  - usually unilateral and first noted in neonatal period
  - associated with microphthalmos and cataract
  - elongated ciliary processes are visible through the dilated pupil **(Figs 7.9.11 and 7.9.12)**
  - intraocular pressure may be high
- *Retinal dysplasia*
  - unilateral or bilateral, usually present at birth
  - pink or white retrolental mass
  - microphthalmic eye with shallow anterior chamber and elongated ciliary processes
  - associated with severe systemic abnormalities
- *Coats' disease*
  - unilateral, occurs primarily in older boys
  - large areas of retinal or subretinal exudates with cholesterol crystals
  - dilated and tortuous retinal blood vessels at the posterior pole
  - exudative detachment as a retrolental mass occurs eventually **(Figs 7.9.13 and 7.9.14)**
- *Choroidal coloboma*
  - unilateral or bilateral, present at birth
  - leukocoria is only with a large choroidal coloboma with microphthalmos
  - inferonasal in location
  - fundus shows, the defect is at the embryonic fissure, with shiny-white sclera **(See Fig 6.2.2)**

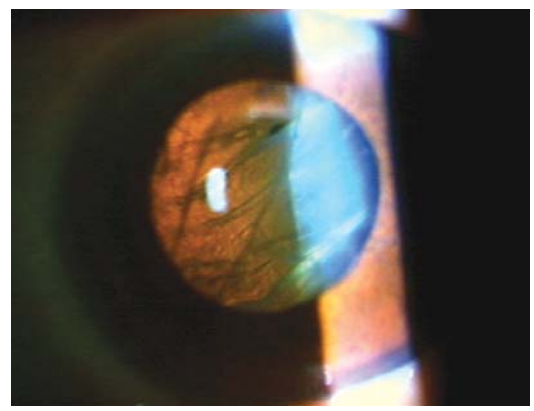


FIGURE 7.9.7: Leukocoria—retinopathy of prematurity

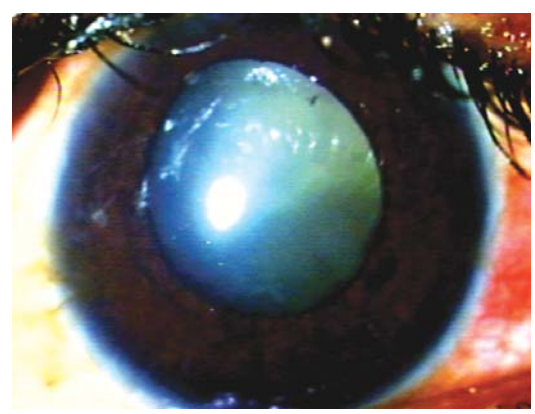


FIGURE 7.9.8: Leukocoria—toxocara endophthalmitis

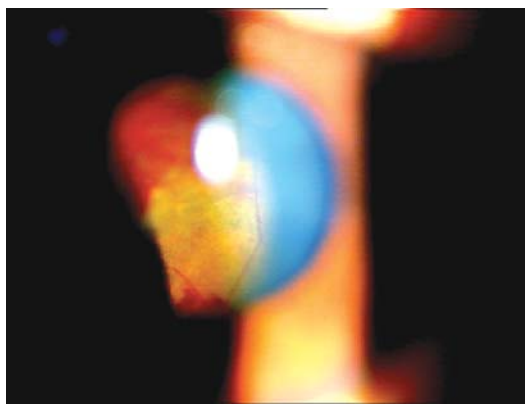


FIGURE 7.9.9: Leukocoria—toxocara endophthalmitis

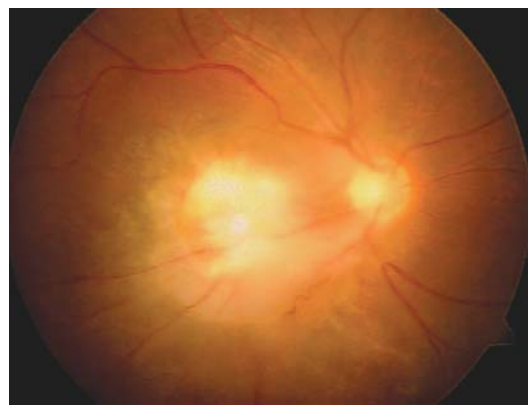


FIGURE 7.9.10: Leukocoria—toxocara endophthalmitis

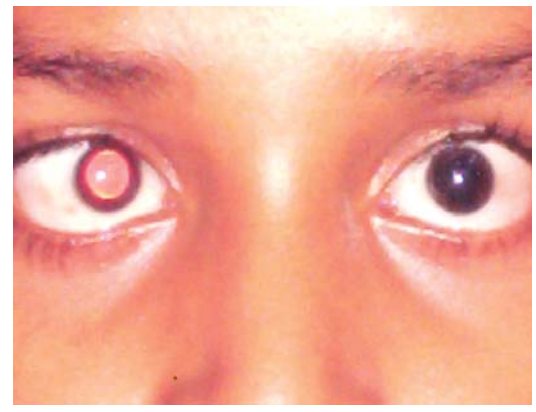


FIGURE 7.9.11: Leukocoria—PHPV

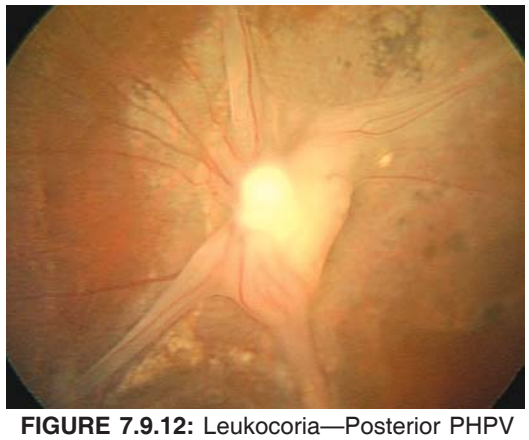


FIGURE 7.9.12: Leukocoria—Posterior PHPV

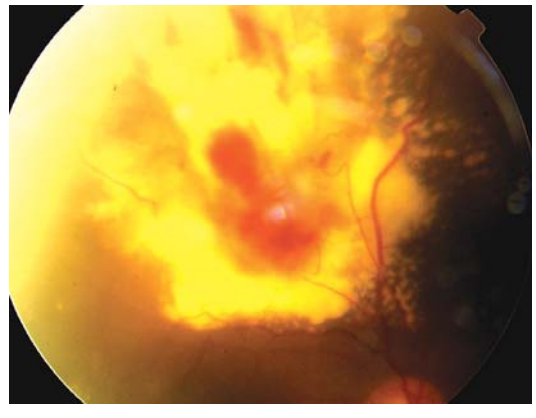


FIGURE 7.9.13: Leukocoria—Coats' disease

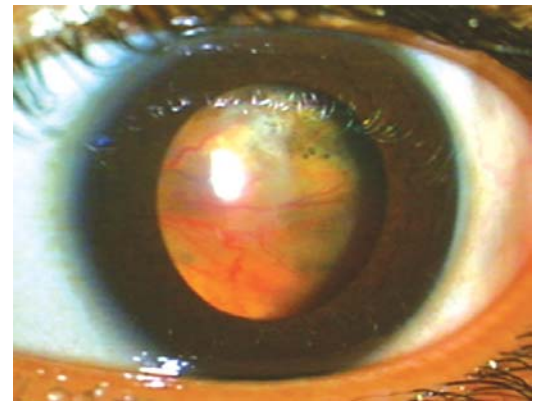


FIGURE 7.9.14: Leukocoria—Coats' disease

8 Diseases of the Lens

CONGENITAL CONDITIONS

- Coloboma of the lens
- Anterior lenticonus
- Posterior lenticonus
- Spherophakia (microspherophakia)
- Ectopia lentis
- Marfan's syndrome
- Homocystinuria
- Weill-Marchesani syndrome

LENS-INDUCED OCULAR DISEASES

- Phacolytic glaucoma
- Phacomorphic glaucoma
- Phacotoxic uveitis

CATARACT

- Lental opacity

CONGENITAL OR DEVELOPMENTAL CATARACT

- Anterior polar cataract
- Posterior polar cataract
- Coronary cataract
- Zonular (lamellar) cataract
- Central pulverulent cataract
- Rubella cataract
- Sutural cataract
- Blue-dot cataract
- Other types of congenital/developmental cataract

SENILE (ADULT) CATARACT

- Cortical cataract
- Stages of cortical cataract
- Cupuliform cataract (posterior subcapsular cataract)
- Nuclear cataract
- Other types of adult cataract

SPECIFIC CATARACT ENTITIES

- Complicated cataract
- Diabetic cataract
- Galactose cataract
- Traumatic cataract
- Glaukomfleckens

OPACITIES IN PSEUDOPHAKIA

- Anterior capsular opacification/fibrosis
- Posterior capsular opacification (PCO) or 'After Cataract'
- Soemmering's ring
- Cortical matter behind the IOL
- Inflammatory plaques
- Posterior capsular folds
- Pseudophakic opacities/defects

DISPLACEMENT OF THE CRYSTALLINE LENS AND IOLs

- Subluxation of the crystalline lens
- Dislocation of the crystalline lens
- Displacement of the IOL
- Miscellaneous pseudophakic conditions

APHAKIA

CONGENITAL CONDITIONS

Coloboma of the Lens

- Rare unilateral or bilateral condition, may occur with other typical colobomatous defects of the uveal tract **(Fig 8.1.1)** or in isolation **(Figs 8.1.2 and 8.1.3),** called atypical coloboma
- Notching of the lens at the inferior equator or other area with absence of zonules **(Fig 8.1.4)**

FIGURE 8.1.1: Coloboma of the lens

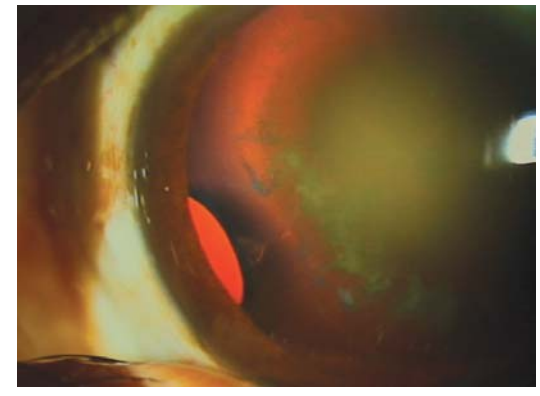


FIGURE 8.1.2: Coloboma of lens—atypical

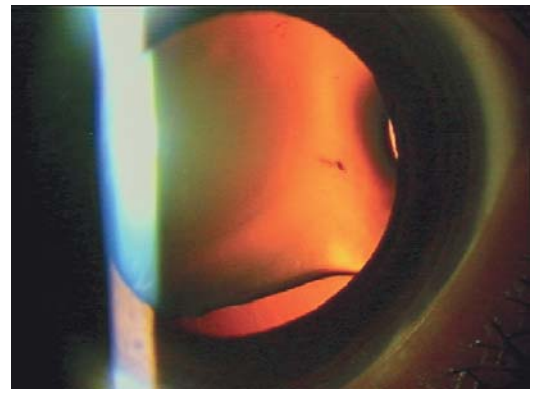


FIGURE 8.1.3: Double coloboma of lens—atypical

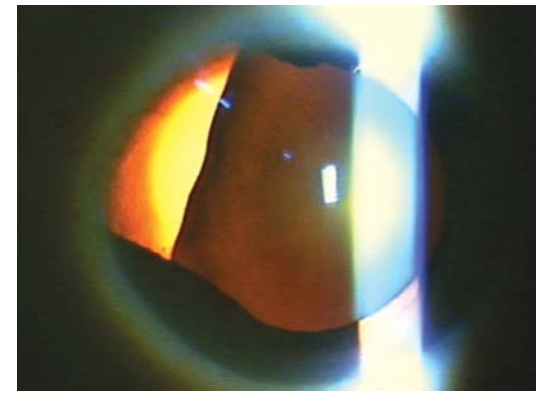


FIGURE 8.1.4: Coloboma of lens with atypical iris coloboma

Anterior Lenticonus

- Rare bilateral condition, often associated with Alport's syndrome
- Anterior conical projection at the center of the lens **(Fig 8.2.1)**
- Oil-droplet sign on distant direct ophthalmoscopy
- High lenticular myopia
- May be associated with lenticular opacification

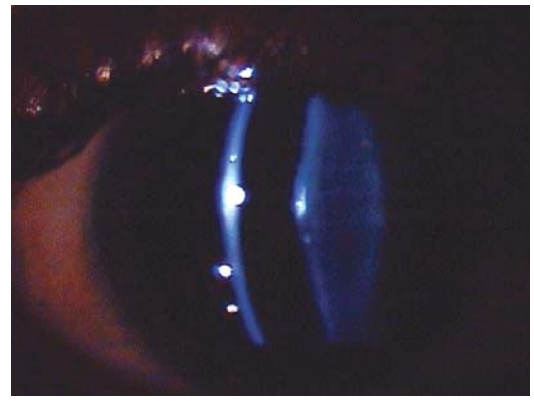


FIGURE 8.2.1: Anterior lenticonus

Posterior Lenticonus

- Rare bilateral condition, may be associated with Lowe's syndrome
- Posterior conical or globular (lentiglobus) bulge in the axial zone of the lens (**Fig 8.3.1**)
- May be associated with lenticular opacification

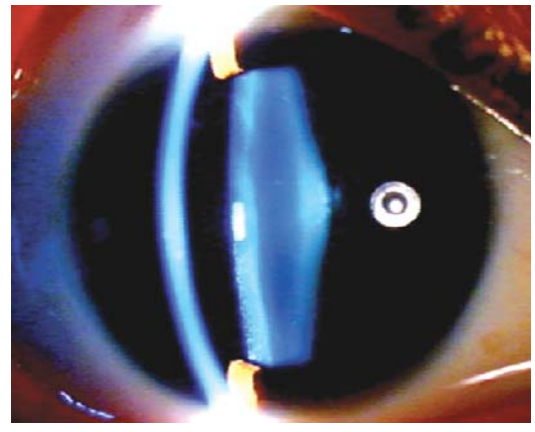


FIGURE 8.3.1: Posterior lenticonus

Spherophakia (microspherophakia)

- Smaller diameter lens with spherical shape (**Fig 8.4.1**)
- Subluxation of the lens is common (**Fig 8.4.2**)
- Lenticular myopia
- Pupillary block glaucoma and this glaucoma is aggravated by miotics and relieved by mydriatics (called *'inverse glaucoma'*)
- Associated with Weill-Marchesani syndrome

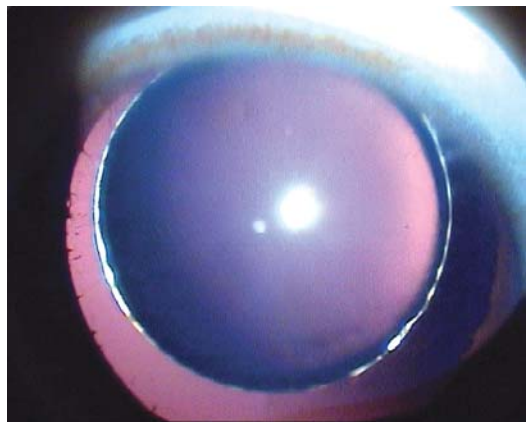


FIGURE 8.4.1: Spherophakia

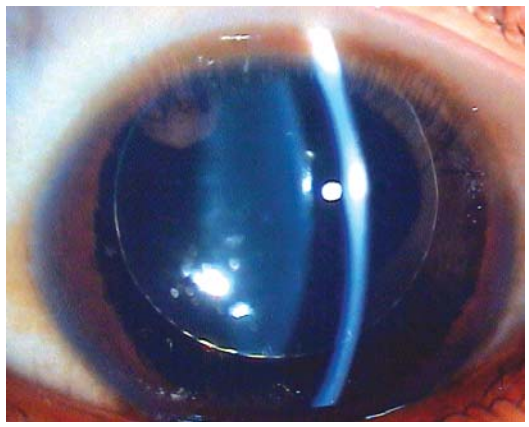


FIGURE 8.4.2: Spherophakia with subluxation

Ectopia Lentis

- Ectopia lentis is a congenital or developmental bilateral subluxation or dislocation of the lens **(Fig 8.5.1)**
- Diagnosis should be confirmed after full dilatation of the pupil **(Figs 8.5.2 and 8.5.3)**
- Edge of the displaced lens appears in the pupillary area as a dark or golden crescent **(Fig 8.5.4)**
- Displaced lens may touch the cornea **(Fig 8.5.5)**
- They are mainly hereditary in nature
- *Causes of ectopia lentis :*
  - *ectopia lentis et pupillae:* pupil and lens are displaced in opposite direction
  - Marfan's syndrome
  - homocystinuria
  - Weill-Marchesani syndrome
  - Ehlers-Danlos syndrome
  - hyperlysinemia
  - familial ectopia lentis
  - sulphite oxidase deficiency

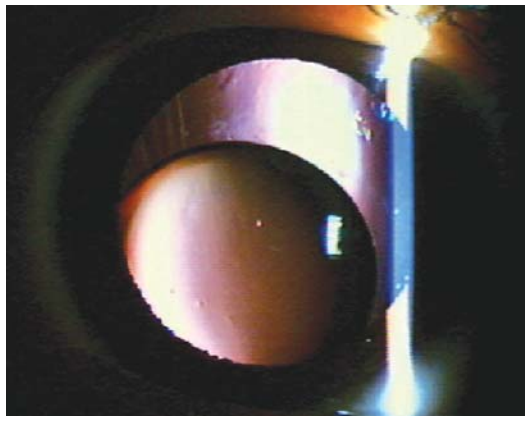


FIGURE 8.5.1: Ectopia lentis

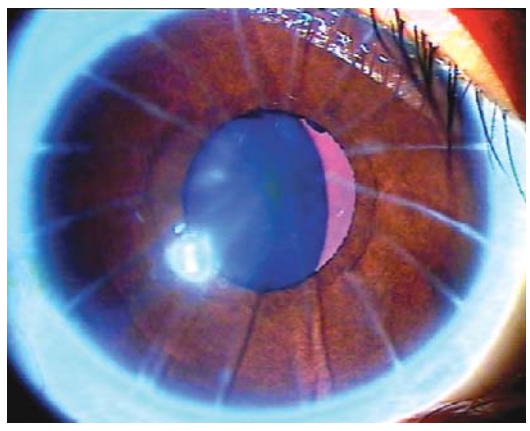


FIGURE 8.5.2: Ectopia lentis—after dilatation

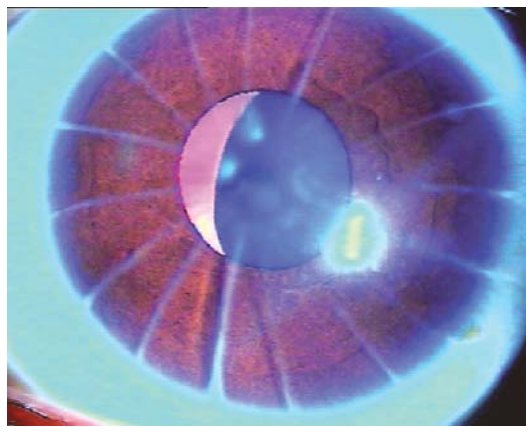


FIGURE 8.5.3: Ectopia lentis—after dilatation

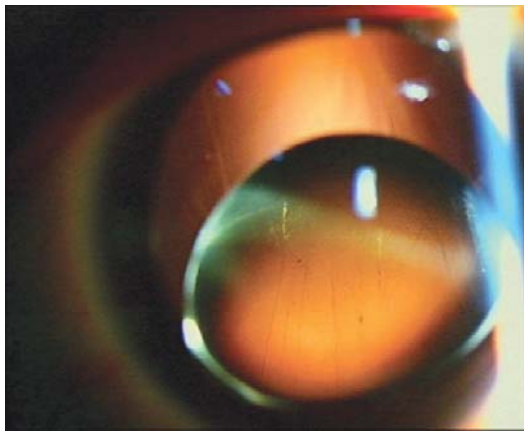


FIGURE 8.5.4: Ectopia lentis—golden crescent

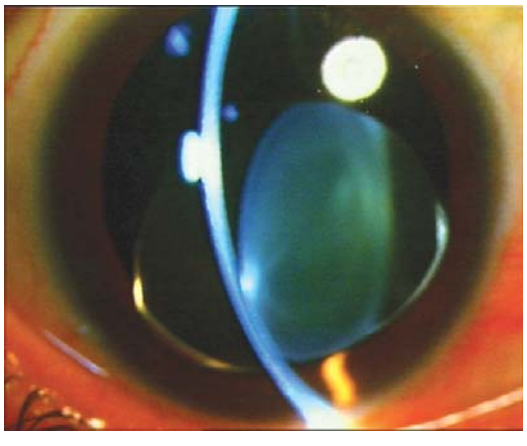


FIGURE 8.5.5: Ectopia lentis—corneal touch

Marfan's Syndrome

- Autosomal dominant, a multi-system mesodermal dysplasia
- Lens is typically subluxated in upward and inward direction **(Fig 8.6.1)**
- May be subluxated in any quadrant (**Figs 8.6.2 and 8.6.3**)
- Iris hypoplasia causes poor pupillary dilatation (**Fig 8.6.4**)
- *Systemic features:*
  - arachnodactyly (spider fingers) (**Fig 8.6.5**)
  - long extremities (**Fig 8.6.6**)
  - hyper-extensibility of the joints
  - cardiovascular anomalies

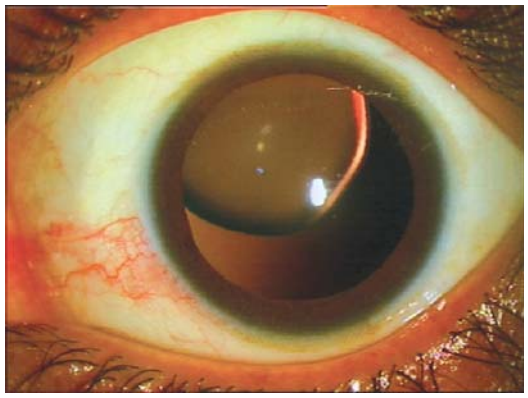


FIGURE 8.6.1: Marfan's syndrome

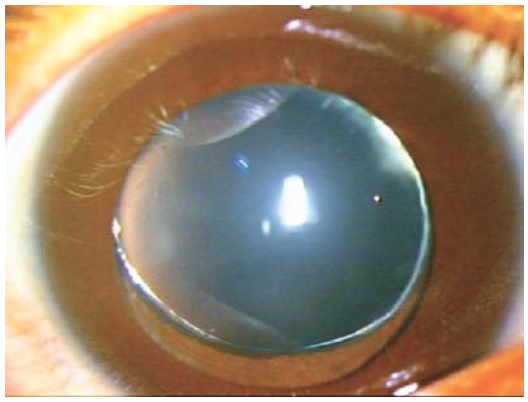


FIGURE 8.6.2: Upward subluxation in Marfan's syndrome

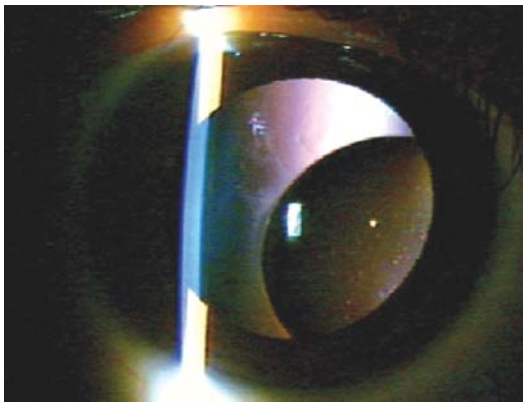


FIGURE 8.6.3: Marfan's syndrome—downward subluxation

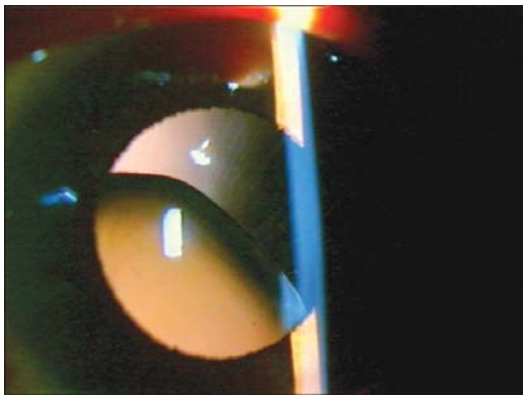


FIGURE 8.6.4: Poor pupillary dilatation

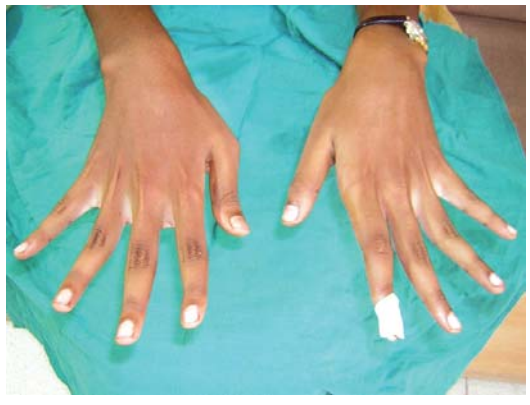


FIGURE 8.6.5: Marfan's syndrome—arachnodactyly

FIGURE 8.6.6: Marfan's syndrome—long extremities

Homocystinuria

- Autosomal recessive, an inborn error of metabolism
- Inability of convert methionine to cystine
- Lens displacement is typically downward and outward **(Figs 8.7.1 and 8.7.2)**
- Diagnosis is confirmed by urine test with sodium nitroprusside
- Anesthetic hazards during operation

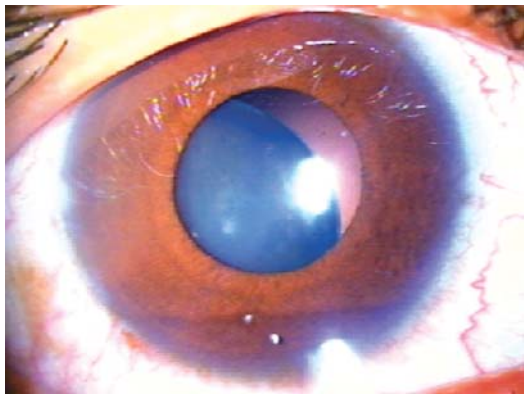


FIGURE 8.7.1: Ectopia lentis—homocystinuria

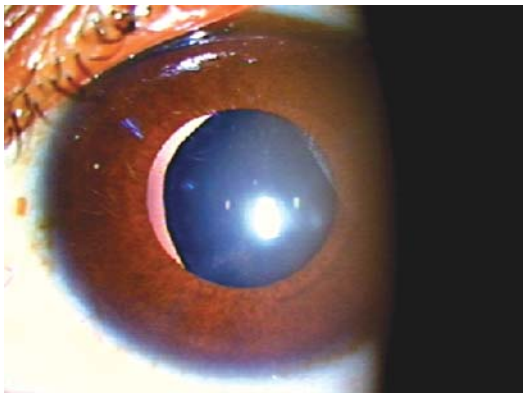


FIGURE 8.7.2: Ectopia lentis—homocystinuria

Weill-Marchesani Syndrome

- *Autosomal recessive:* a mesodermal dysplasia
- Short stature, stubby fingers **(Fig 8.8.1)**
- Microspherophakia, which may be subluxated, usually downward **(Figs 8.8.2 and 8.8.3)**

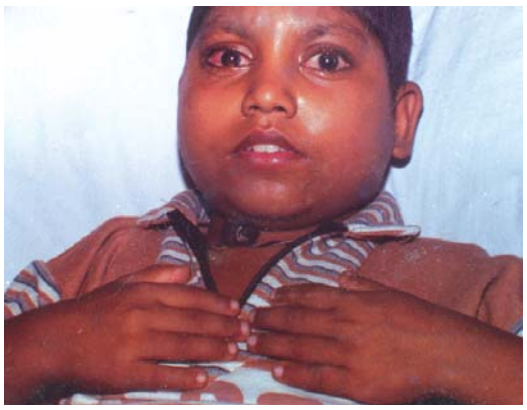


FIGURE 8.8.1: Weill-Marchesani syndrome

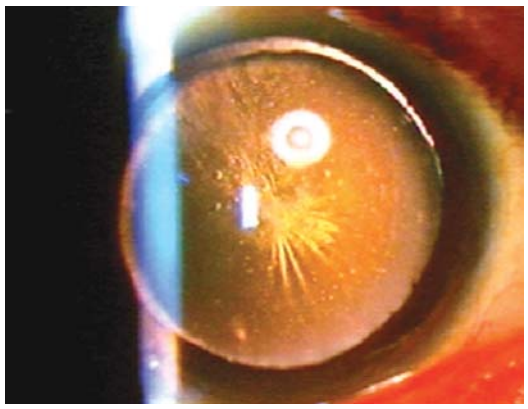


FIGURE 8.8.2: Weill-Marchesani syndrome—Downward subluxation

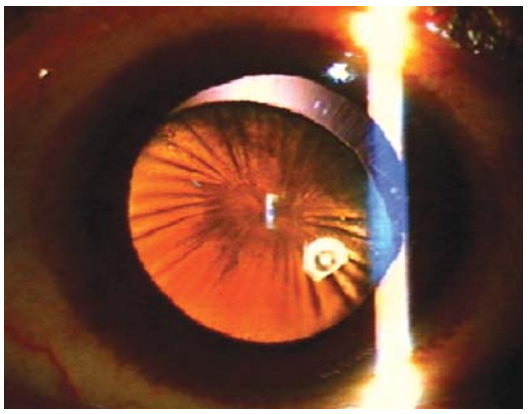


FIGURE 8.8.3: Weill-Marchesani syndrome—Downward subluxation

Treatment of Ectopia Lentis

- Spectacles or contact lenses are used to correct the optical defects through the phakic part
- Pars plana lensectomy with vitrectomy with scleral fixation IOL is better than other surgical means **(Fig 8.9.1)**
- In milder degree of subluxation—ECCE with capsular tension ring (CTR) with PCIOL **(Fig 8.9.2)**

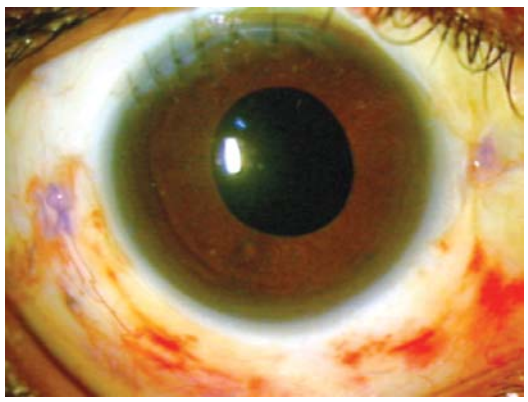


FIGURE 8.9.1: Scleral—fixation PCIOL

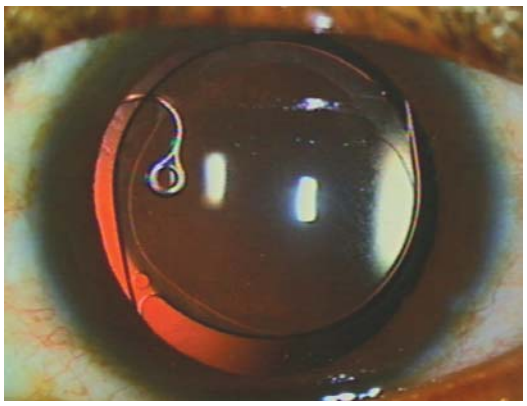


FIGURE 8.9.2: Ectopia lentis—PCIOL with CTR

LENS-INDUCED OCULAR DISEASES

Phacolytic Glaucoma

- In some hypermature cataract the capsule leaks, and large phagocytes filled with lens material obstruct the trabecular meshwork **(Figs 8.10.1 and 8.10.2)**
- Secondary open-angle glaucoma
- *Treatment:* glaucoma must be controlled medically first and then the lens has to be extracted urgently with PCIOL

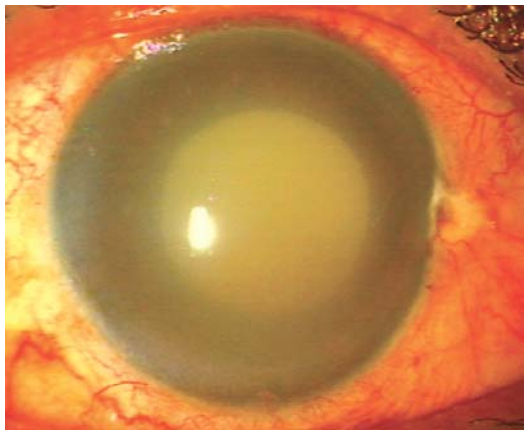


FIGURE 8.10.1: Phacolytic glaucoma

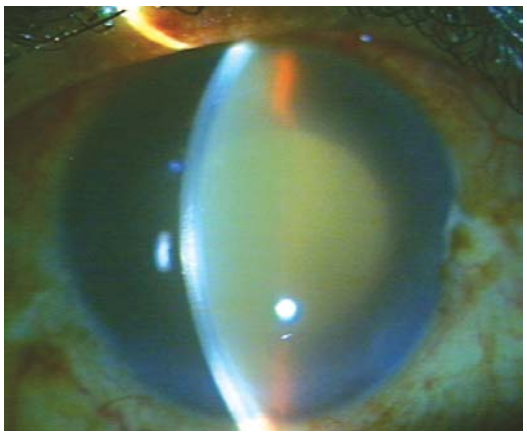


FIGURE 8.10.2: Phacolytic glaucoma—deep AC

Phacomorphic Glaucoma

- Rapid swelling of the lens in intumescent cataract **(Fig 8.11.1)**
- Anterior chamber becomes shallow **(Fig 8.11.2)**
- May cause a secondary angle-closure glaucoma
- *Treatment:* urgent control of glaucoma and extraction of lens with PCIOL

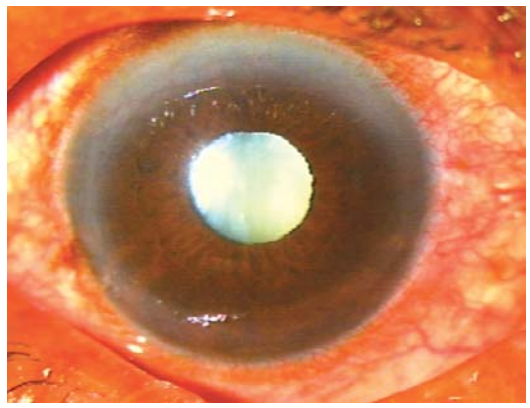


FIGURE 8.11.1: Phacomorphic glaucoma

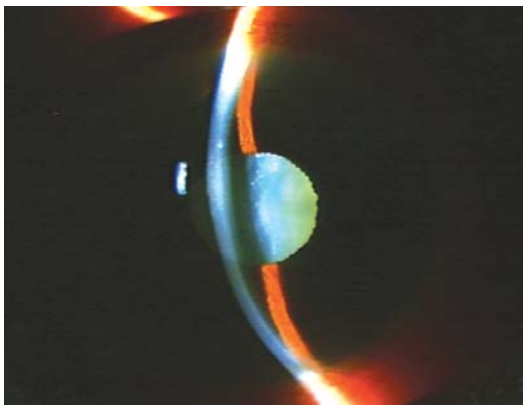


FIGURE 8.11.2: Phacomorphic glaucoma—shallow AC

Phacotoxic Uveitis

- Lens proteins are relatively poor antigens
- Sometimes, a granulomatous uveitis may develop and may be with secondary glaucoma **(Fig 8.12.1)**
- *Treatment:* high dose of systemic and topical corticosteroids and cycloplegic
- *Phaco-anaphylactic uveitis*
  - dislocation of lens nucleus or nuclear fragments in the vitreous cavity during an extracapsular extraction or phacoemulsification
  - causes severe granulomatous uveitis

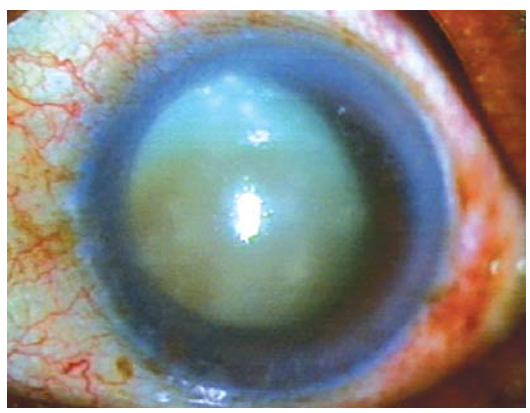


FIGURE 8.12.1: Phacotoxic uveitis

CATARACT

Any opacity of the lens or its capsule, causing visual impairment, is called cataract (**Figs 8.13.1 and 8.13.2**)

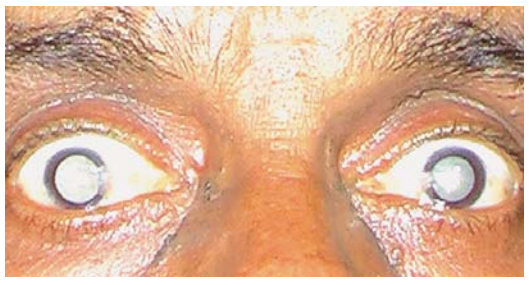


FIGURE 8.13.1: Bilateral senile cataract

FIGURE 8.13.2: Developmental cataract

Lental Opacity

- Many congenital opacities are stationary **(Fig 8.14.1)**
- Even in senile cataract the opacities may remain localized for many years
- Not associated with visual impairment **(Fig 8.14.2)**
- It should be better termed as *lental opacity*

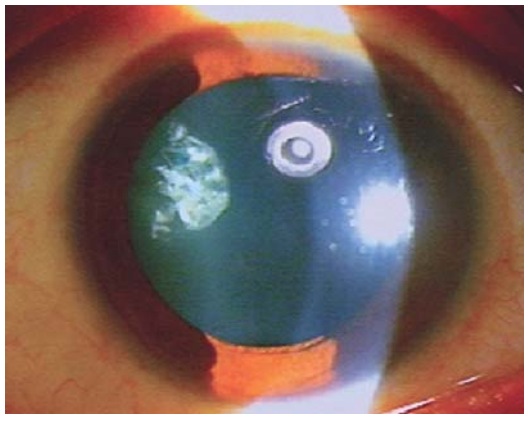


FIGURE 8.14.1: Isolated lental opacity

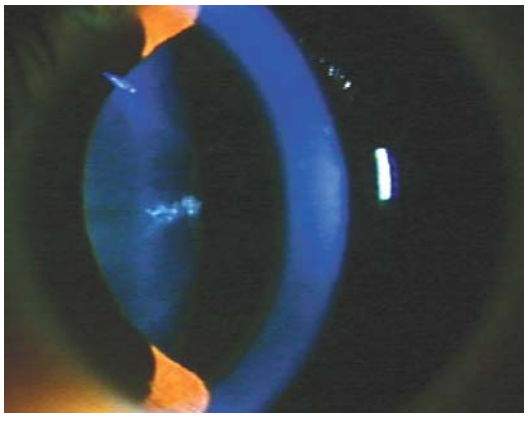


FIGURE 8.14.2: Lental opacity

CONGENITAL OR DEVELOPMENTAL CATARACT

- Presents at birth (**Fig 8.15.1**), or develops within first few years after the birth (**Fig 8.15.2**)
- Some congenital opacities escape detection at birth
- 1/3rd hereditary, 1/3rd idiopathic and 1/3rd are associated with some systemic problems
- Idiopathic variety may be unilateral or bilateral, whereas others are usually bilateral
- One type of opacity may coexist with other type
- Half of the eyes with congenital cataract have some other ocular anomalies

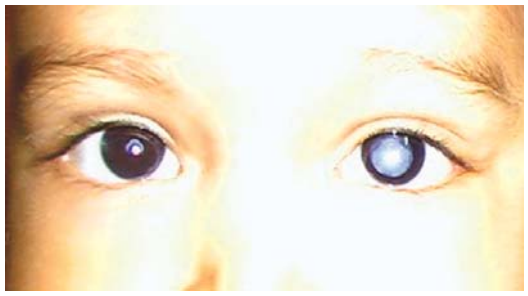


FIGURE 8.15.1: Congenital cataract

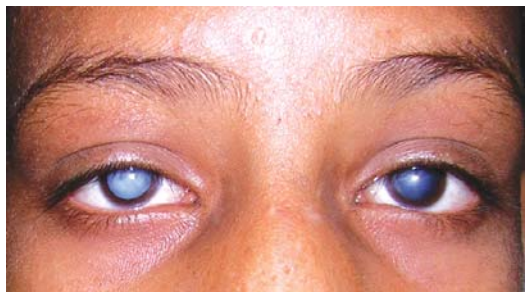


FIGURE 8.15.2: Developmental cataract

Anterior Polar Cataract

- Rare, usually bilateral and sometimes hereditary
- The opacity involves the capsule or both capsule and anterior cortex **(Fig 8.16.1)**
- May be pyramidal and may project into the anterior chamber *(pyramidal cataract)* **(Fig 8.16.2)**
- Usually associated with Peters' anomaly **(Fig 8.16.3)**, posterior keratoconus or persistent pupillary membrane **(Fig 8.16.4)**
- May be associated with other type of developmental cataract **(Fig 8.16.5)**
- *Treatment:* Most of the cases do not require any treatment

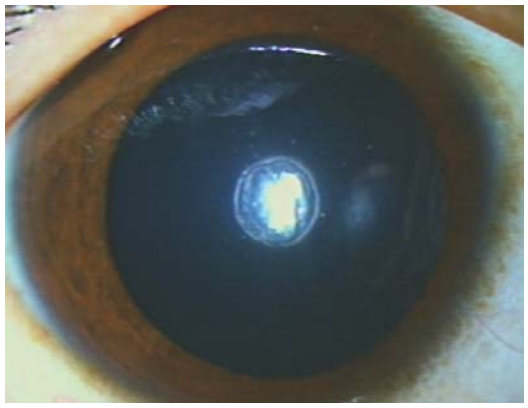


FIGURE 8.16.1: Anterior polar cataract

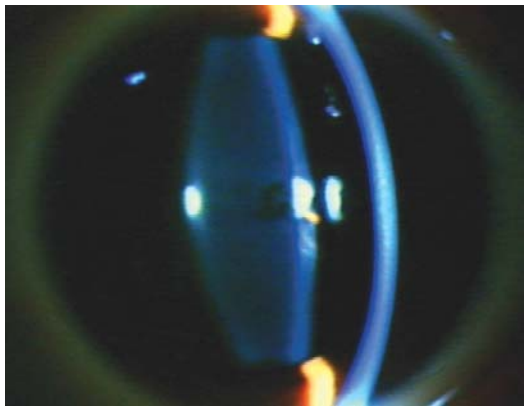


FIGURE 8.16.2: Anterior polar cataract—pyramidal

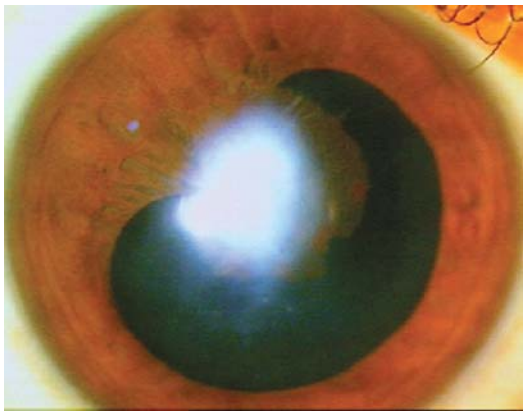


FIGURE 8.16.3: Anterior polar cataract—Peters' anomaly

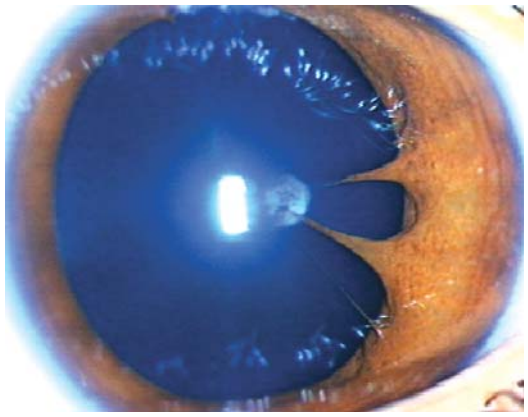


FIGURE 8.16.4: Anterior polar cataract—persistent pupillary membrane

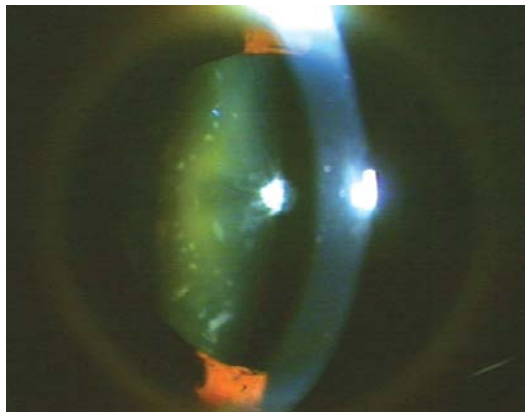


FIGURE 8.16.5: Anterior polar cataract with coronary cataract

Posterior Polar Cataract

- Opacity may involve only the posterior capsule **(Figs 8.17.1 and 8.17.2)**

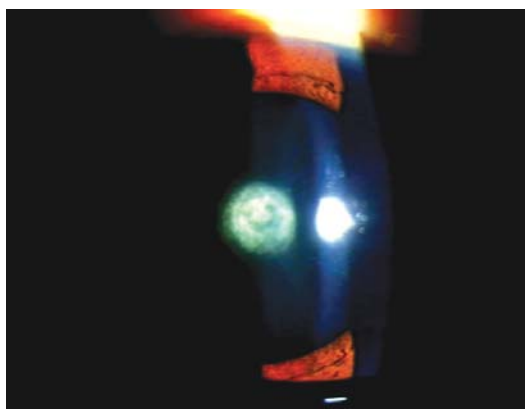


FIGURE 8.17.1: Posterior polar cataract

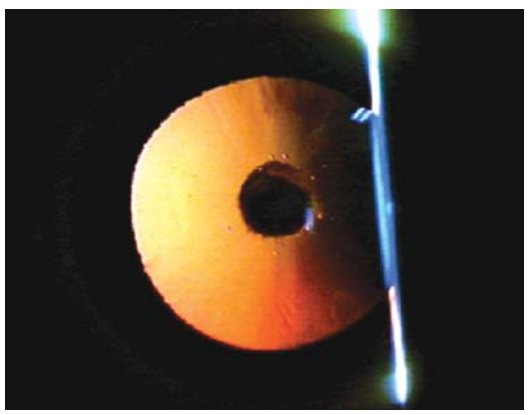


FIGURE 8.17.2: Posterior polar cataract

- Sometimes, it forms a plaque on the posterior cortex with onion-ring appearance **(Figs 8.17.3 and 8.17.4)**
- Few dense dots are seen within the opacity and at the outer ring **(Fig 8.17.5)**
- Sometimes associated with a frank gap in the posterior capsule **(Fig 8.17.6)**
- May be associated with residue of the attachment of hyaloid artery on the posterior lens capsule as small dots—*Mittendorf's dot* **(Fig 8.17.7)**
- May be with persistent hyperplastic primary vitreous

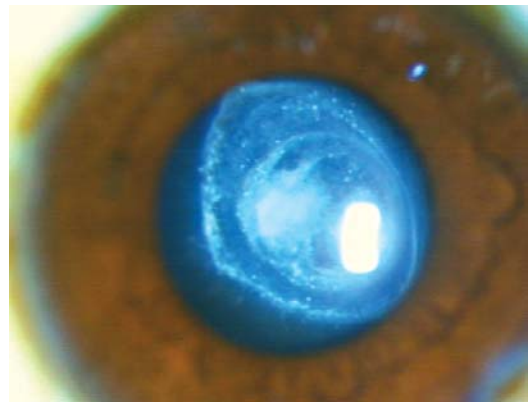


FIGURE 8.17.3: PPC—onion-ring appearance

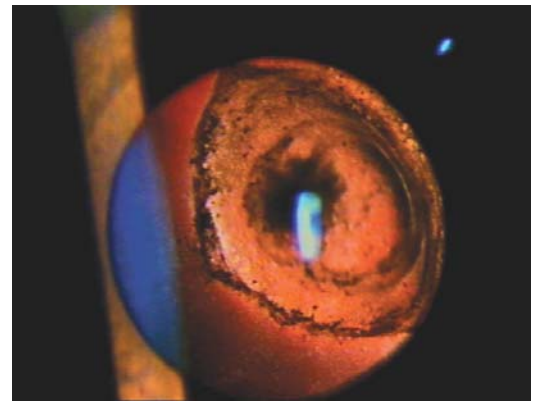


FIGURE 8.17.4: PPC—onion-ring appearance

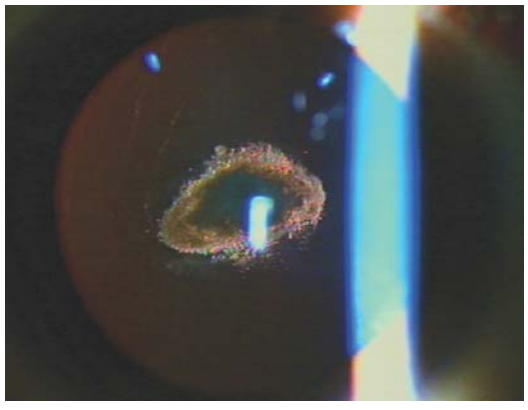


FIGURE 8.17.5: PPC—white dots at outer ring

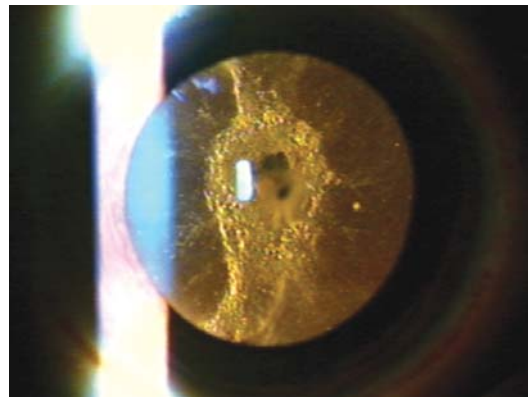


FIGURE 8.17.6: PPC—gap in posterior capsule

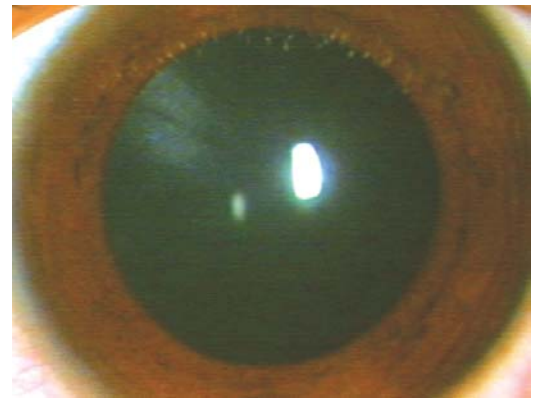


FIGURE 8.17.7: Mittendorf's dot

Coronary Cataract

- Usually sporadic, developmental cataract, occurring at puberty
- Appears as a 'corona' or club-shaped opacities, near the periphery of the lens cortex **(Fig 8.18.1)**
- They are hidden by the iris, dilatation of the pupil is essential **(Fig 8.18.2)**
- Does not interfere with vision
- May coexist with other type of opacity

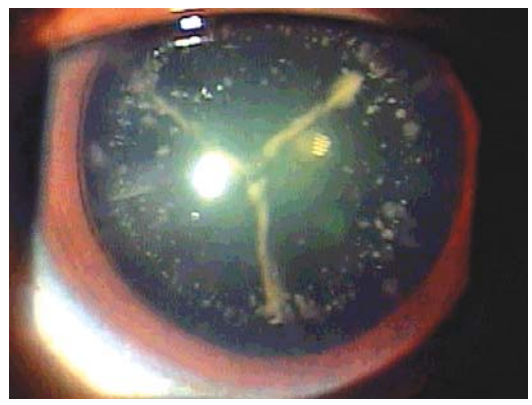


FIGURE 8.18.1: Coronary and sutural cataract

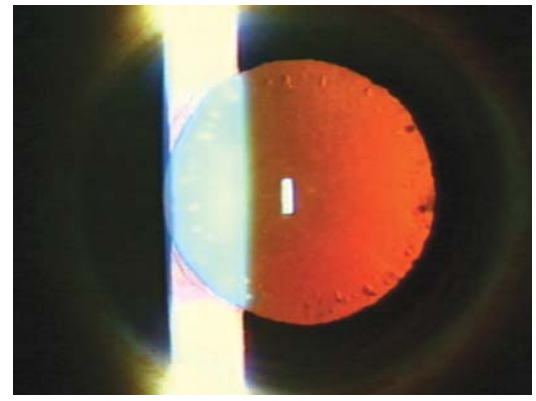


FIGURE 8.18.2: Coronary cataract

Zonular (lamellar) Cataract

- Commonest type of developmental cataract presenting with visual impairment
- Usually dominant, but may be recessive
- Consists of concentric, sharply demarcated zone (lamellae) of opacities surrounding a clear nuclear core, and enveloped by the clear cortex externally **(Figs 8.19.1 and 8.19.2)**

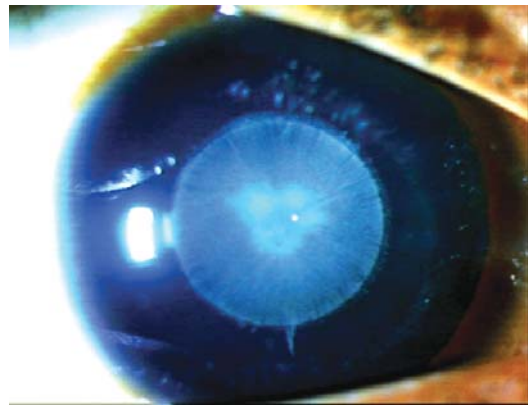


FIGURE 8.19.1: Zonular cataract

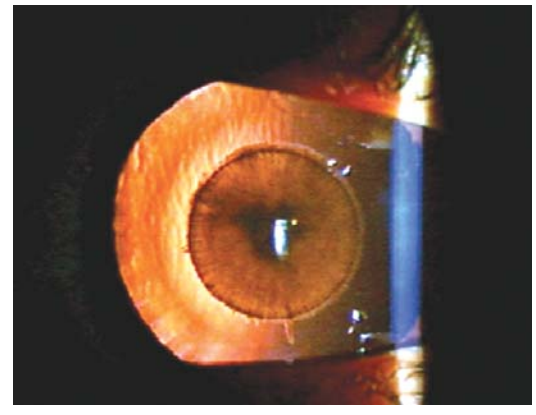


FIGURE 8.19.2: Zonular cataract

- Linear opacities, like spokes of a wheel (called *riders*) **(Figs 8.19.3 and 8.19.4)** that extend outwards toward the equator—and it is pathognomonic

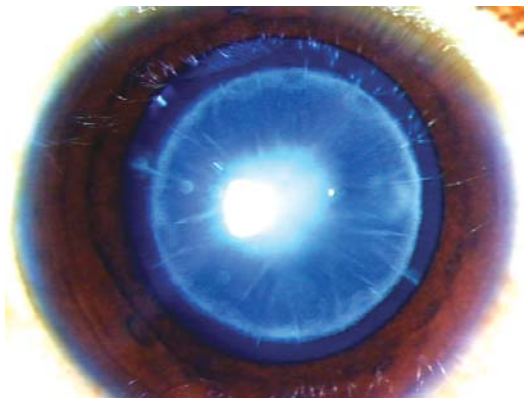


FIGURE 8.19.3: Zonular cataract—riders

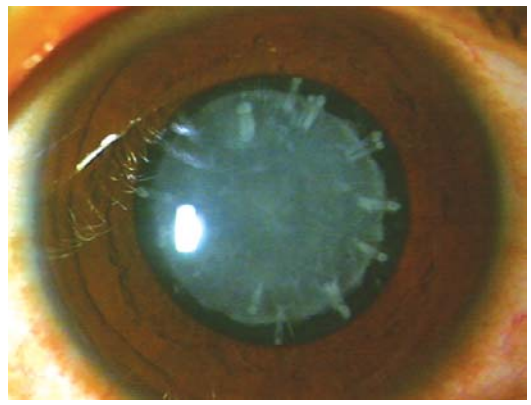


FIGURE 8.19.4: Zonular cataract—riders

Central Pulverulent Cataract

- Rare, nonprogressive cataract with dominant inheritance
- Spheroidal opacity, 2-4 mm in diameter within the lens nucleus, with a relatively clear center **(Figs 8.20.1 and 8.20.2)**

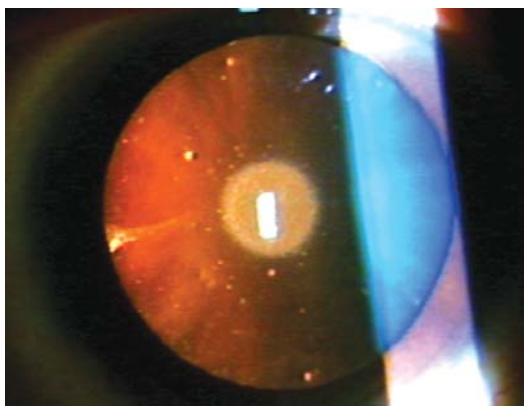


FIGURE 8.20.1: Central pulverulent cataract

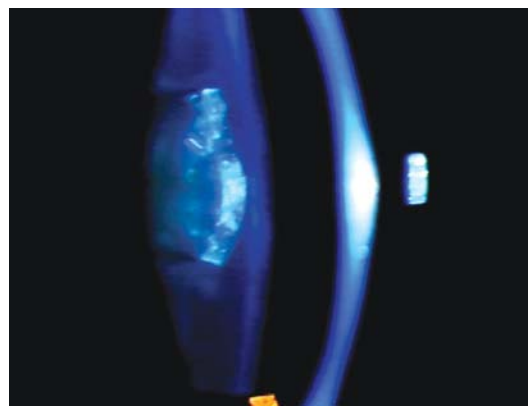


FIGURE 8.20.2: Central pulverulent cataract

Rubella Cataract

- Cataract is originally nuclear and progresses to become total (pearly white in color)
- Associated with microphthalmos, nystagmus, strabismus, glaucoma and pigmentary retinopathy
- Maternal history is very important
- Removal of such cataract frequently provokes an intense uveitis or endophthalmitis

Sutural Cataract

- Rare, bilateral cataract, usually does not interfere with vision
- X-linked recessive inheritance, with males has significant opacities than females
- Opacities follow the course and shape of anterior or posterior Y-sutures of the lens (**Fig 8.21.1**)
- May be associate with other types of congenital cataract **(Figs 8.21.2 and See Figs 8.18.1 and 8.22.2)**
- Usually no treatment is necessary

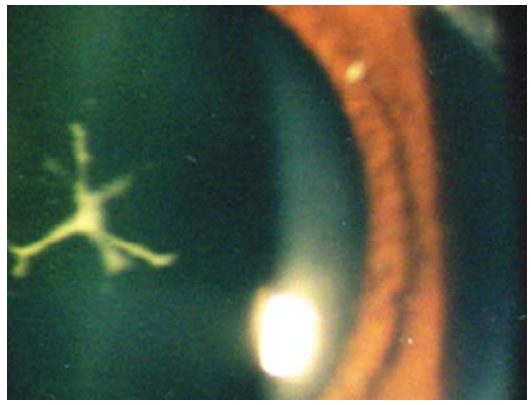


FIGURE 8.21.1: Sutural cataract

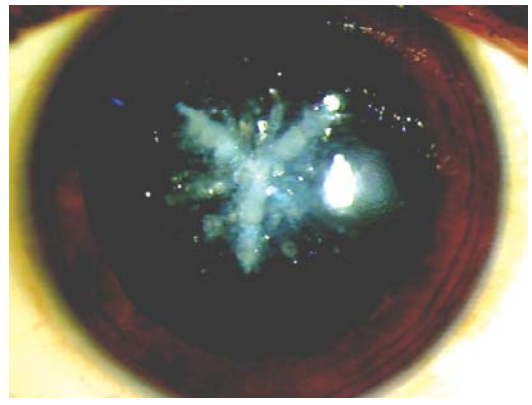


FIGURE 8.21.2: Sutural cataract

Blue Dot Cataract

- Bilateral, rather common, and innocuous **(Fig 8.22.1)**
- Usual detection is during routine ophthalmological examination
- No treatment is necessary
- May coexist with other type of congenital cataract **(Figs 8.22.2 and 8.22.3)** and then it may require treatment

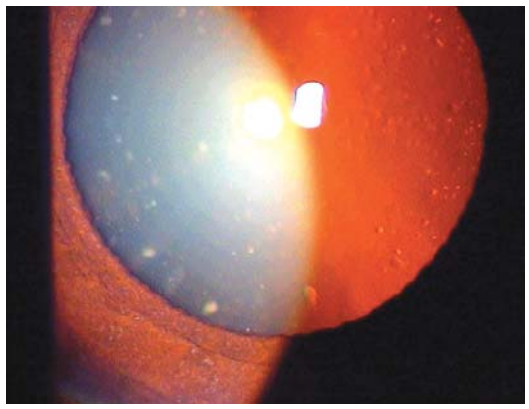


FIGURE 8.22.1: Blue dot cataract

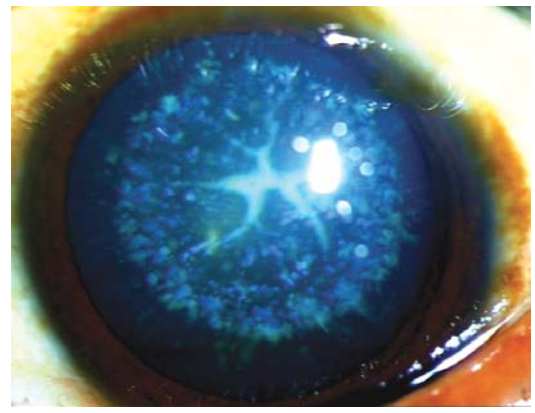


FIGURE 8.22.2: Blue dot and sutural cataract

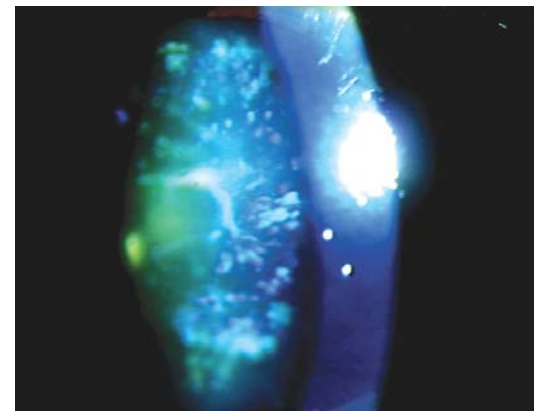


FIGURE 8.22.3: Blue dot and sutural cataract

Other Types of Congenital/Developmental Cataract

- Coraliform cataract **(Figs 8.23.1 and 8.23.2)**
- Floraliform cataract **(Figs 8.23.3 and 8.23.4)**
- Spoke-like opacities **(Fig 8.23.5)**
- Star-like opacities **(Fig 8.23.6)**
- Epicapsular pigmented stars **(Figs 8.23.7 and 8.23.8)**

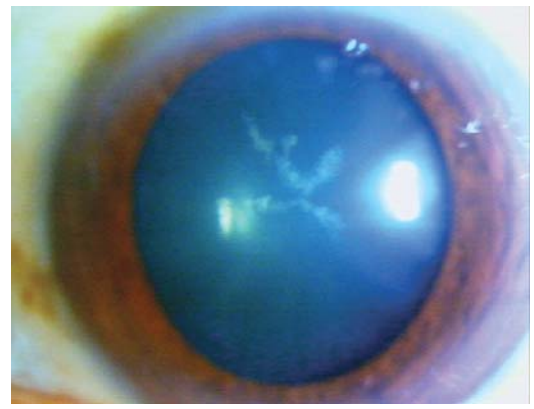


FIGURE 8.23.1: Coraliform cataract

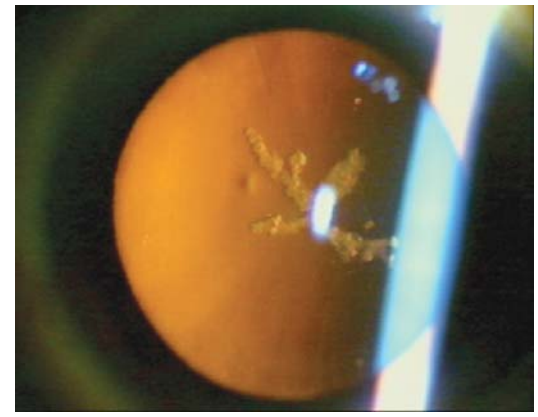


FIGURE 8.23.2: Coraliform cataract

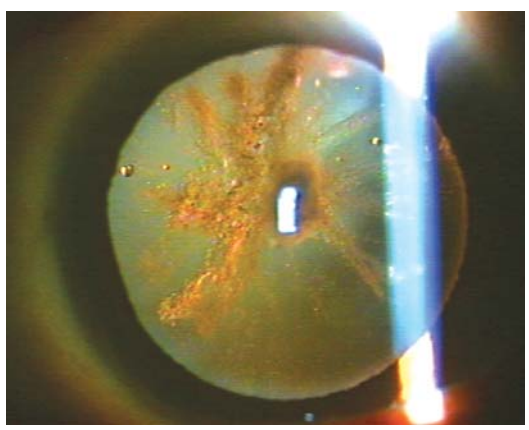


FIGURE 8.23.3: Floraliform cataract

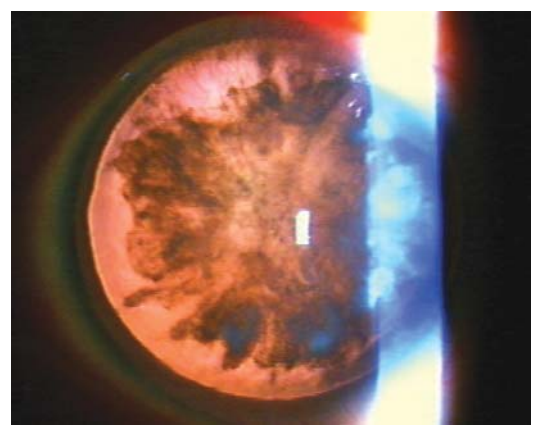


FIGURE 8.23.4: Floraliform cataract

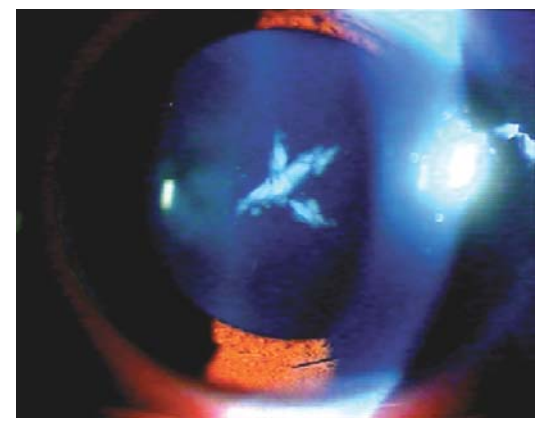


FIGURE 8.23.5: Spoke-like cataract

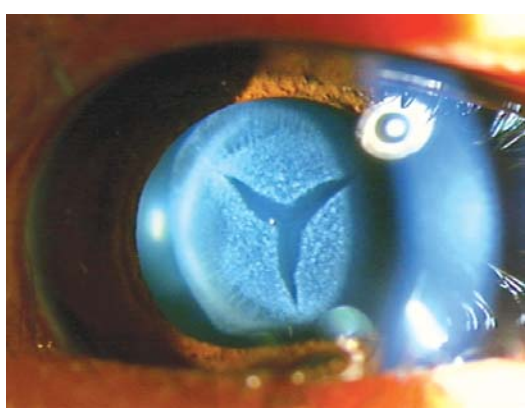


FIGURE 8.23.6: Star-like cataract

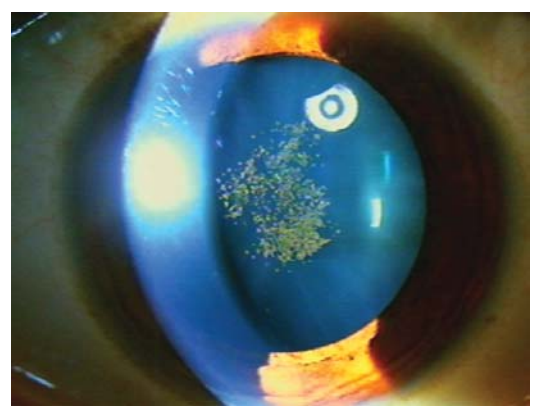


FIGURE 8.23.7: Epicapsular pigmented stars

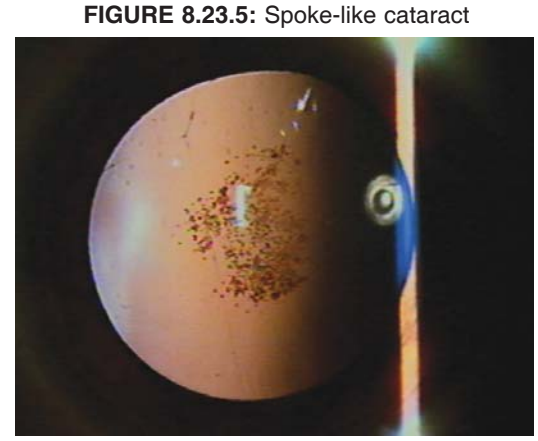


FIGURE 8.23.8: Epicapsular pigmented stars

SENILE (ADULT) CATARACT

- Cortical or soft cataract
- Nuclear or hard cataract
- Cortico-nuclear or mixed cataract

Cortical Cataract

- *Cuneiform:* It starts as wedge-shaped spokes of opacity at the periphery and gradually encroaches towards center **(Figs 8.24.1 and 8.24.2)**
- *Cupuliform cataract:* opacity appears in the posterior cortex just beneath the capsule and gradually it forms a dense opacity **(Figs 8.24.3 and 8.24.4)**

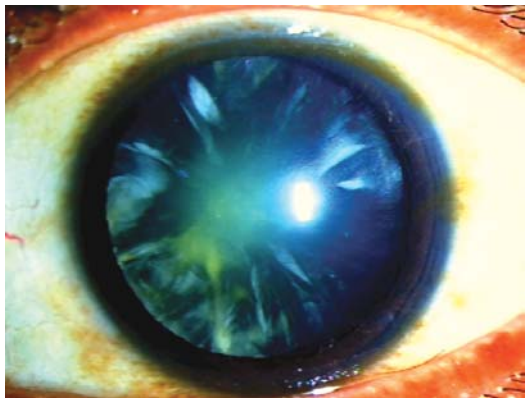


FIGURE 8.24.1: Cuneiform cataract

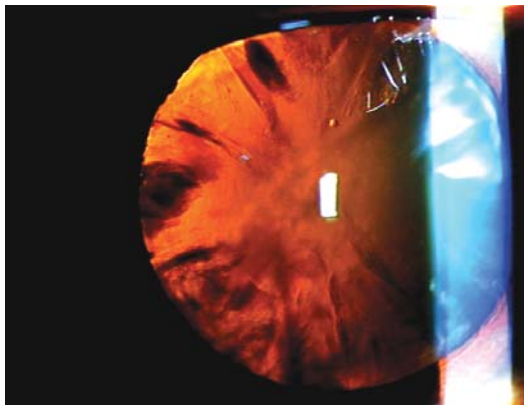


FIGURE 8.24.2: Cuneiform cataract

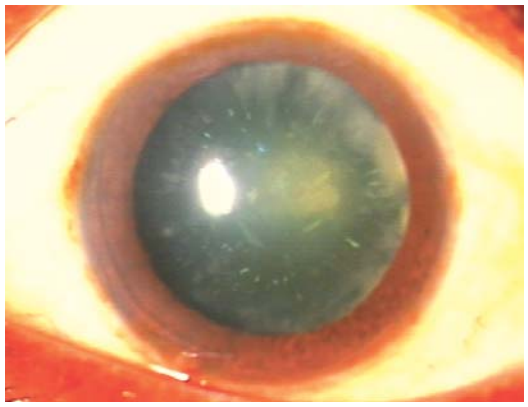


FIGURE 8.24.3: Cupuliform cataract

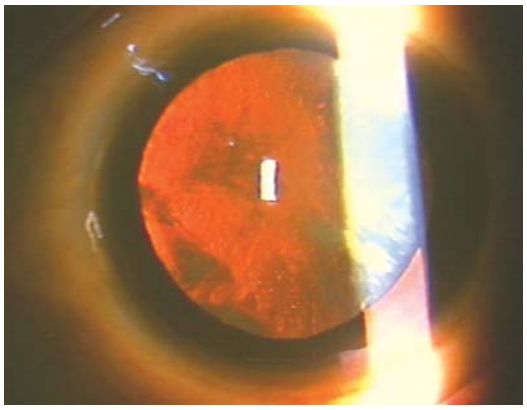


FIGURE 8.24.4: Cupuliform cataract

Stages

Immature stage

- Lens is grayish or grayish white in color
- Iris shadow is present **(Figs 8.25.1 and 8.25.2)**

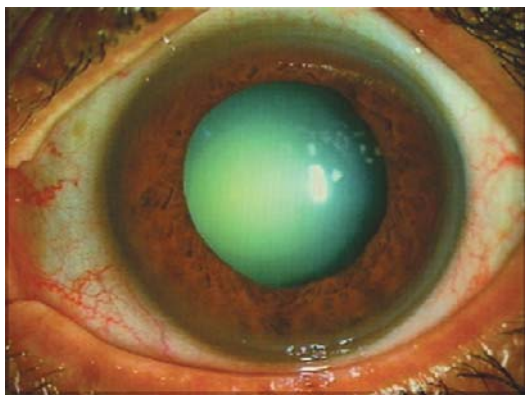


FIGURE 8.25.1: Immature cataract

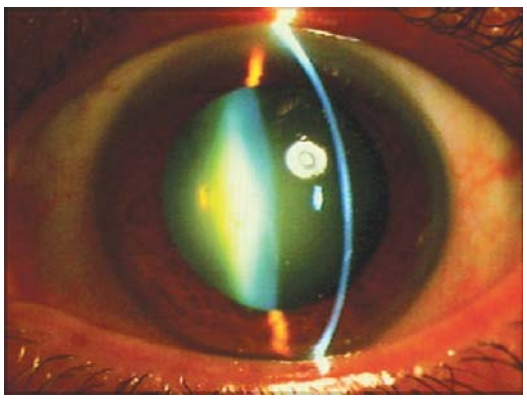


FIGURE 8.25.2: Immature cataract

Intumescent stage

- Lens becomes swollen due to progressive hydration of the cortical fibers (**Fig 8.26.1**)
- Anterior chamber is shallow (**Fig 8.26.2**)
- Chance of secondary angle-closure glaucoma

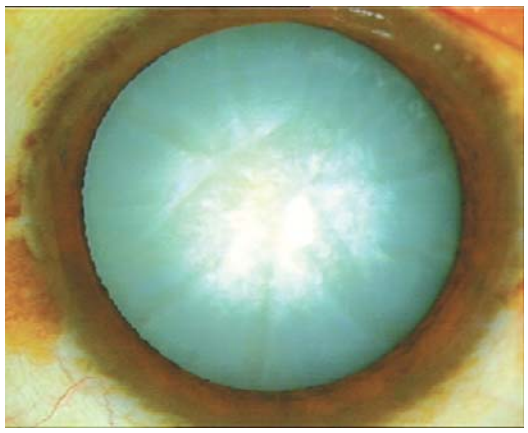


FIGURE 8.26.1: Intumescent cataract

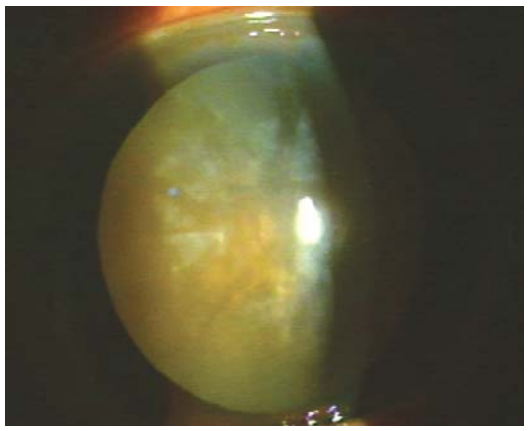


FIGURE 8.26.2: Intumescent cataract

Mature stage

- Lens is white or pearly white in color **(Fig 8.27.1)**
- No iris shadow
- In many developing countries, patient may present with bilateral mature cataract **(Fig 8.27.2)**

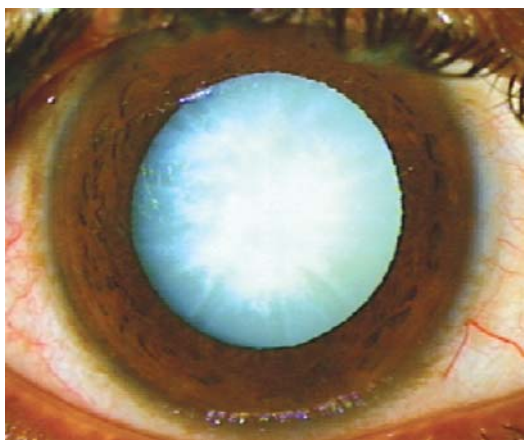


FIGURE 8.27.1: Mature cataract

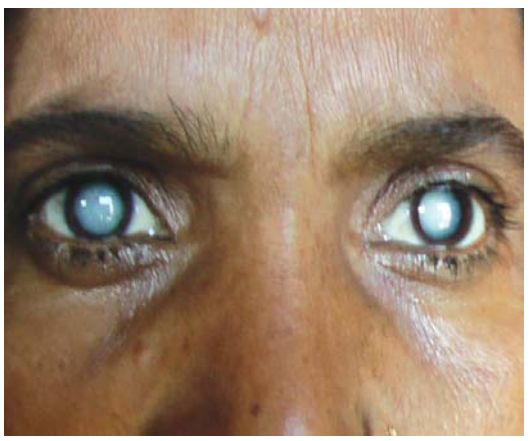


FIGURE 8.27.2: Bilateral white mature cataract

Hypermature stage

- Cortex becomes disintegrated and then liquefied, or transformed into a pultaceous mass
- *Morgagnian cataract*
  - cortex becomes fluid and the brown nucleus may sink at the bottom within the lens capsule **(Fig 8.28.1)**
  - fluid is milky-white in appearance
  - a semi-circular line above the nucleus, which may change its position **(Fig 8.28.2)**
  - capsular fibrosis occurring some cases **(Fig 8.28.3)**
- *Sclerotic cataract*
  - more and more inspissated, and shrunken in appearance, due to loss of fluid **(Fig 8.28.4)**
  - lens is more flat and yellowish-white in appearance
  - calcific deposits in some part of the capsule

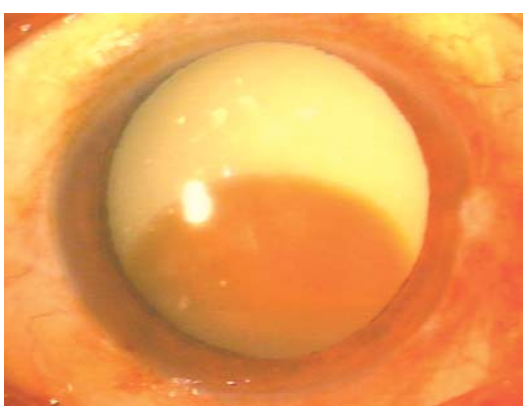


FIGURE 8.28.1: Hypermature cataract—Morgagnian

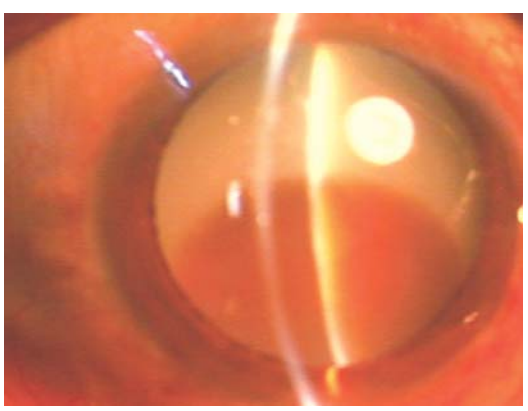


FIGURE 8.28.2: Hypermature cataract—Morgagnian

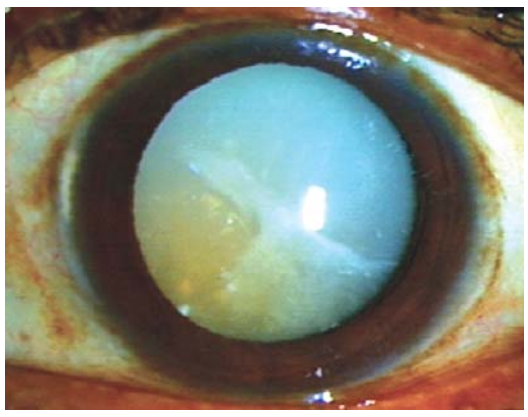


FIGURE 8.28.3: Morgagnian cataract—capsular fibrosis

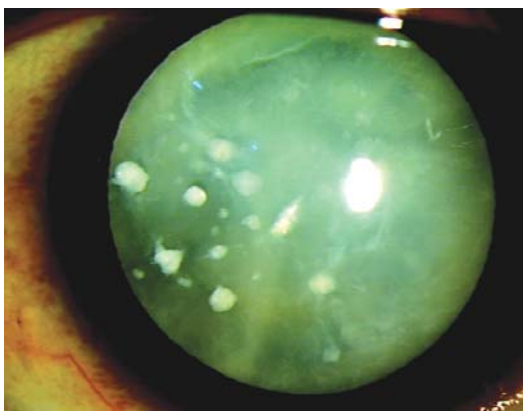


FIGURE 8.28.4: Hypermature cataract—sclerotic

Cupuliform Cataract (posterior subcapsular cataract)

- Starts in the axial region of the posterior cortex (**Fig 8.29.1**)
- Slowly progresses to involve the entire posterior cortex
- Marked diminution of vision, as the opacity is near the nodal point of the eye
- Opacity is best judged by a slit-lamp, and with dilated pupil (**Fig 8.29.2**)

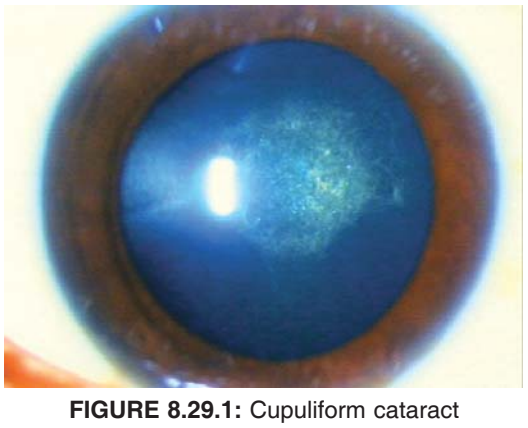


FIGURE 8.29.1: Cupuliform cataract

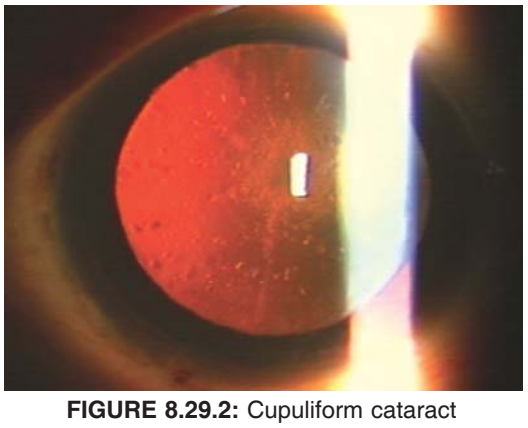


FIGURE 8.29.2: Cupuliform cataract

- Appears as dirty yellowish-white layer in the posterior cortex (**Figs 8.29.3 and 8.29.4**)
- Patients with posterior cortical cataract always see better in darkness (dawn or dusk)

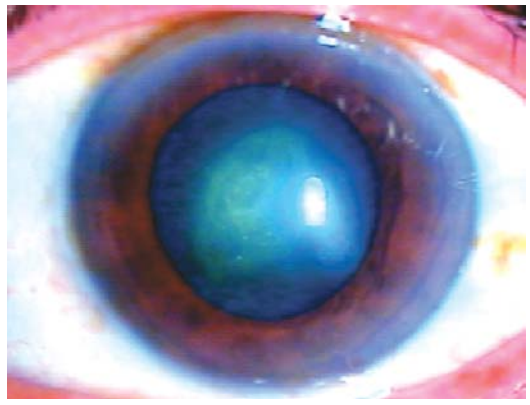


FIGURE 8.29.3: Dense cupuliform cataract

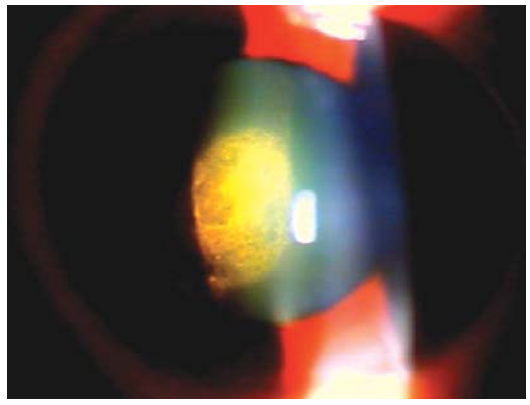


FIGURE 8.29.4: Dense cupuliform cataract

Nuclear Cataract

- Tends to occur earlier than cortical variety
- More commonly seen in degenerative myopia, post-vitrectomy and diabetes
- Varies in density and color which gradually spreads towards the cortex
- With time the lens becomes yellow **(Fig 8.30.1)**, amber **(Fig 8.30.2)**, brown *(cataracta brunescens)* **(Figs 8.30.3 and 8.30.4)**, or black *(cataracta nigra)* **(Fig 8.30.5)**
- May be associated with cortical type and called, corticonuclear cataract **(Figs 8.30.6 and 8.30.7)**
- Opacity is better appreciated in dilated pupil

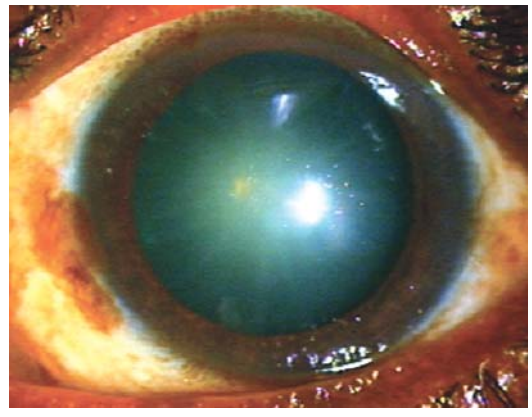


FIGURE 8.30.1: Nuclear cataract

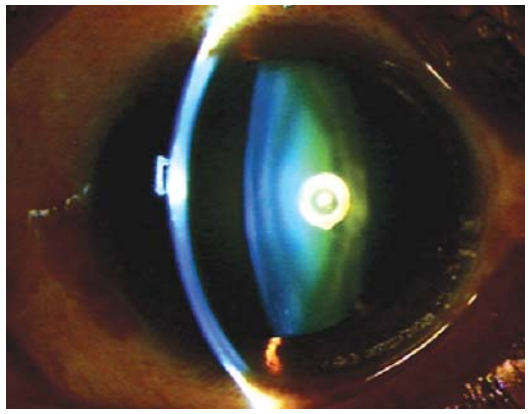


FIGURE 8.30.2: Nuclear cataract and PSC

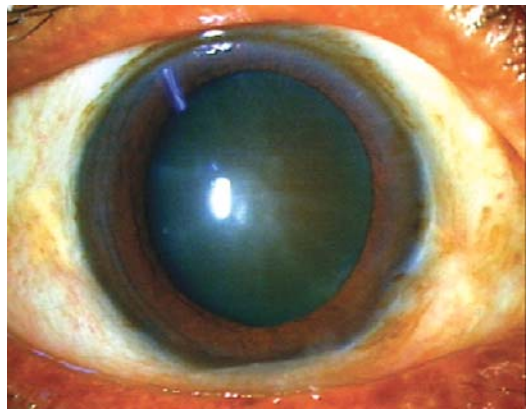


FIGURE 8.30.3: Brown cataract

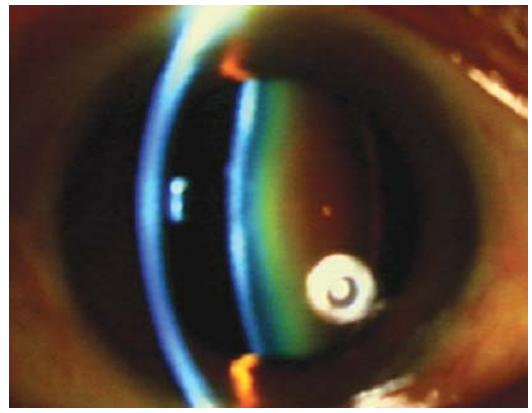


FIGURE 8.30.4: Brown cataract

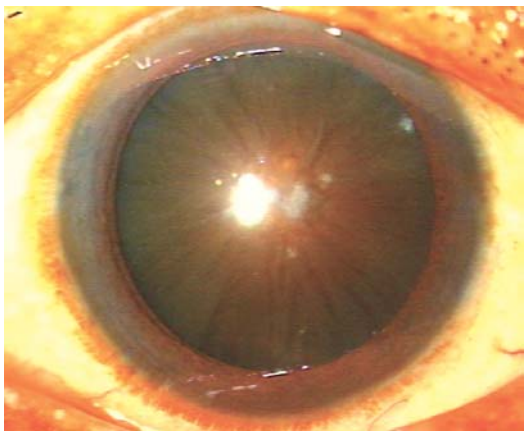


FIGURE 8.30.5: Black cataract

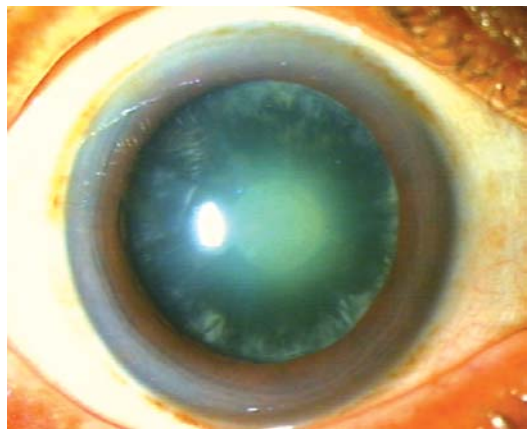


FIGURE 8.30.6: Corticonuclear cataract

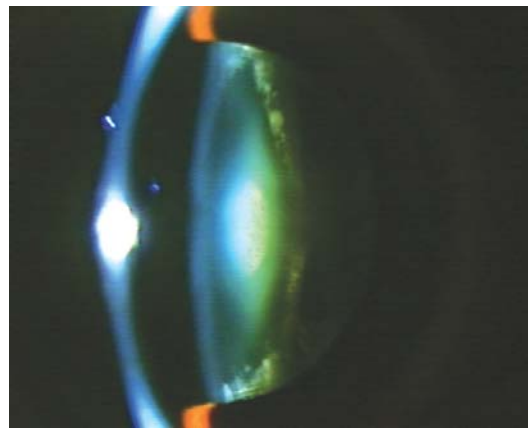


FIGURE 8.30.7: Corticonuclear cataract

Other Types of Adult Cataract

- *Christmas tree cataract:*
  - uncommon age-related cataract
  - polychromatic needle like opacities in the deeper cortex and nucleus **(Fig 8.31.1)**
  - may be present with other opacities **(Fig 8.31.2)**
- *Anterior subcapsular deposits:*
  - bilateral, fine granular opacity in the anterior cortex **(Figs 8.31.3 and 8.31.4)**
  - usually associated with certain drugs like, chlorpromazine
- *Star-shaped cataract:*
  - another uncommon age-related cataract
  - deep corticonuclear star-shaped opacities with visual disturbances **(Figs 8.31.5 and 8.31.6)**

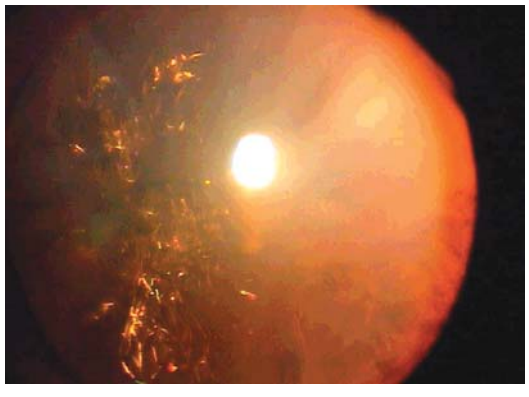


FIGURE 8.31.1: Christmas tree cataract

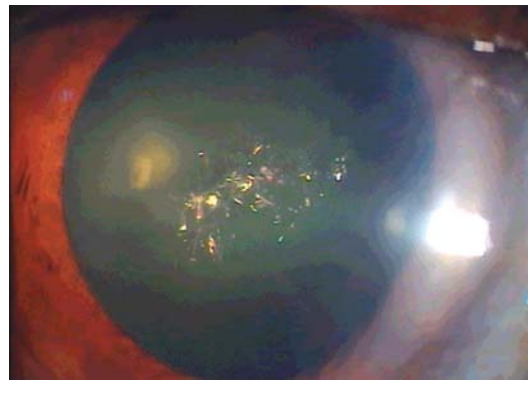


FIGURE 8.31.2: Christmas tree cataract

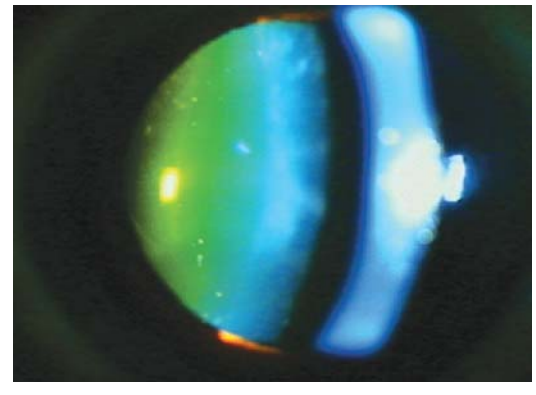


FIGURE 8.31.3: Anterior subcapsular deposit

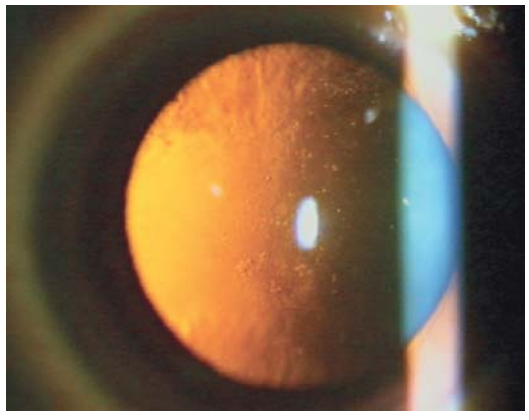


FIGURE 8.31.4: Anterior subcapsular deposit

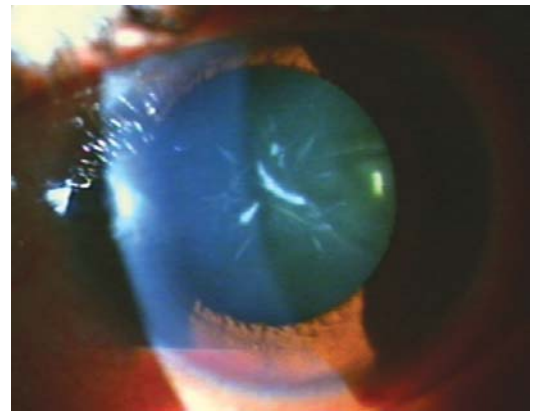


FIGURE 8.31.5: Star-shaped cataract

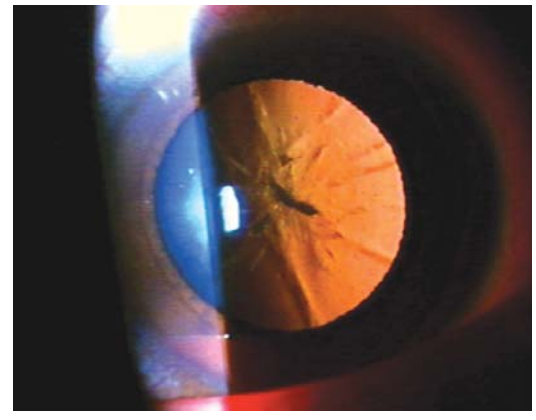


FIGURE 8.31.6: Star-shaped cataract

Surgical Techniques in Senile Cataracts

- *Intracapsular cataract extraction (ICCE):* obsolete nowadays
- *Extracapsular cataract extraction (ECCE):*
- *Manual (stitchless) small incision cataract surgery (MSICS):* popular in most developing countries because of cost-effectiveness
- *Phacoemulsification*: preferred method with implantation of foldable intraocular lens (IOL) in-the-bag

SPECIFIC CATARACT ENTITIES

Complicated Cataract

- Results from disturbances in lens metabolism in inflammatory or degenerative diseases
- *Causes:* iridocyclitis, degenerative myopia, retinitis pigmentosa, retinal detachment, etc.
- Opacity usually commences in the axial region of posterior cortex *(posterior cortical cataract)* **(Fig 8.32.1)**

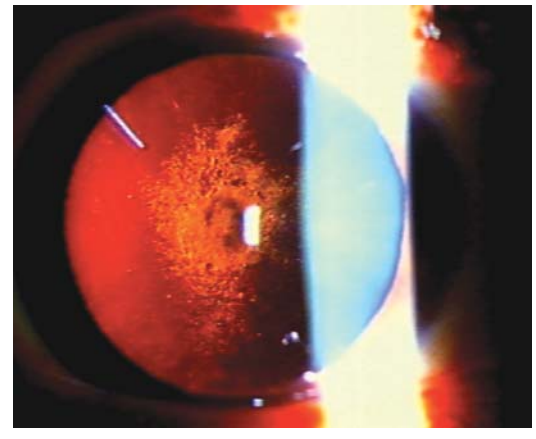


FIGURE 8.32.1: Complicated cataract—posterior cortical cataract

- Appears as grayish-white opacity with irregular border extending towards the equator and in oblique illumination it gives bread-crumb appearance **(Figs 8.32.2 and 8.32.3)**
- Shows a characteristic rainbow display of colors, the polychromatic luster **(Fig 8.32.4)**
- Vision is much impaired as the opacity is near the nodal point of the eye

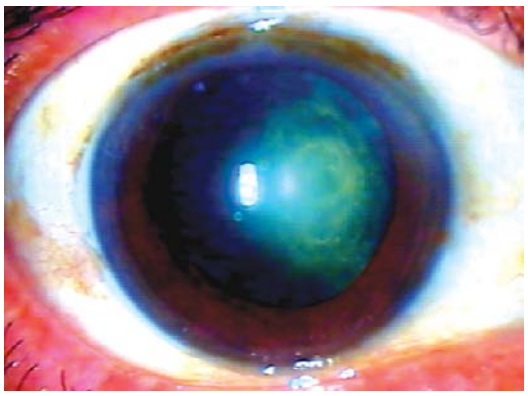


FIGURE 8.32.2: Bread crumb appearance

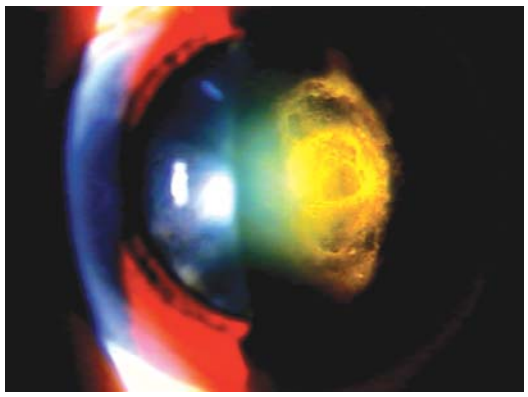


FIGURE 8.32.3: Bread crumb appearance

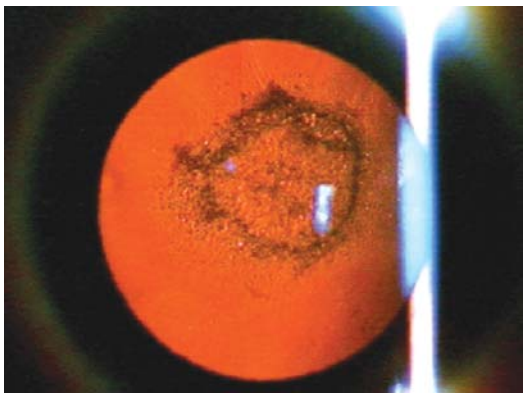


FIGURE 8.32.4: Polychromatic luster

Diabetic Cataract

- Early onset of nuclear cataract
- Posterior and anterior subcapsular opacities of varying degree
- *True diabetic cataract:*
  - more common in uncontrolled juvenile diabetics
  - bilateral cortical cataract
  - consists of minute white dots of varying size like 'snow flakes' and are usually called 'snow-storm cataract' **(Figs 8.33.1 and 8.33.2)**
- *Treatment:* as in adult cataract, but associated retinopathy often reduces the visual outcome

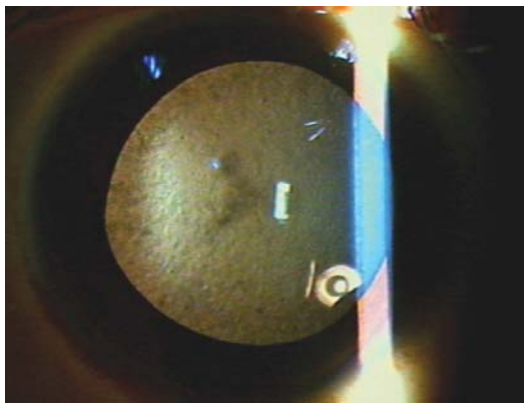


FIGURE 8.33.1: Diabetic cataract—snow flakes

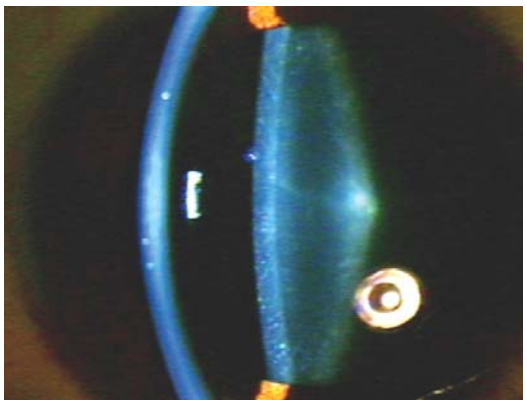


FIGURE 8.33.2: Diabetic cataract—snow flakes

Galactose Cataract

- Rare, bilateral, recessively inherited condition
- *Classical galactosemia:* about 75 percent of the sick infants with classical galactosemia develop bilateral 'oil-droplet' lental opacities which may progress to maturity within a few months
- *Galactokinase deficiency:* milder type and only associated with cataract in healthy child

Traumatic Cataract

- Due to concussion, penetrating or other type of injuries to the eyes
- *Heat cataract:*
  - produced by prolonged exposure to infra-red rays, and occurs in industry
  - among glass-blowers in glass factories *('glass-blower's cataract')* **(Fig 8.34.1)**
  - associated 'exfoliation' *(true exfoliation)* of the lens capsule

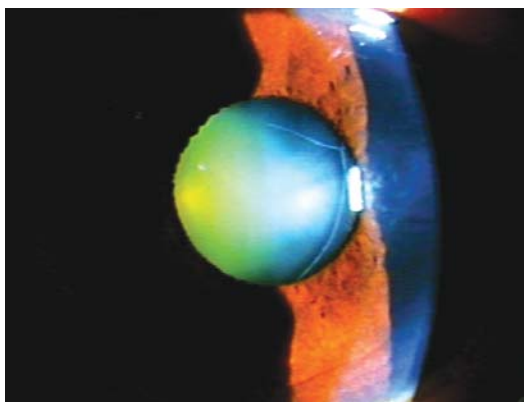


FIGURE 8.34.1: True exfoliation—cataract

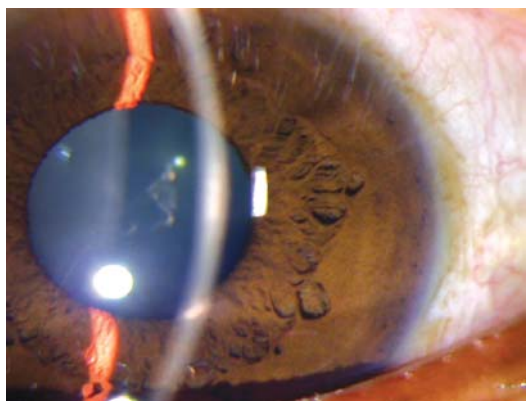


FIGURE 8.34.2: True exfoliation

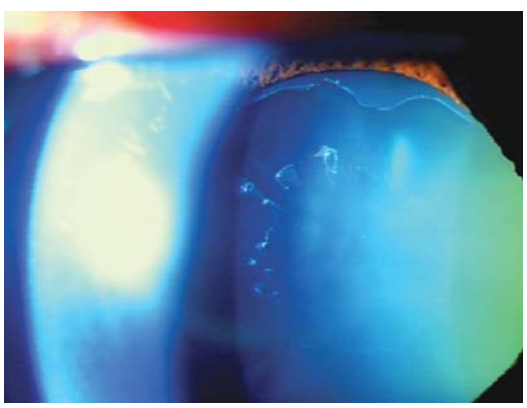


FIGURE 8.34.3: Pseudoexfoliation

  - lamella of the capsule may be curled up in the pupillary area as large sheets **(Fig 8.34.2)**
  - must be differentiated from cataract with pseudoexfoliation **(Fig 8.34.3)**
- *Concussion cataract:*
  - after a blunt trauma
  - initially early rosette **(Fig 8.34.4)** then late rosette (**Fig 8.34.5**) cataract and later on total cataract
  - may be with subluxation (**Fig 8.34.6**)
- *Penetrating injury:*
  - after a penetrating injury **(Fig 8.34.7)**
  - may be associated with capsular rupture
  - fluffy white cortical matter in the anterior chamber **(Fig 8.34.8)**
  - white cataract in most cases
  - with or without intraocular foreign body **(Fig 8.34.9)**
  - may be associated with Siderosis bulbi **(Fig 8.34.10)**
- *Radiation cataract:* associated with exposure to radiation therapy **(Fig 8.34.11)**
- *Electric cataract:* associated with high voltage current or hit by lightening **(Figs 8.34.12 and 8.34.13)**

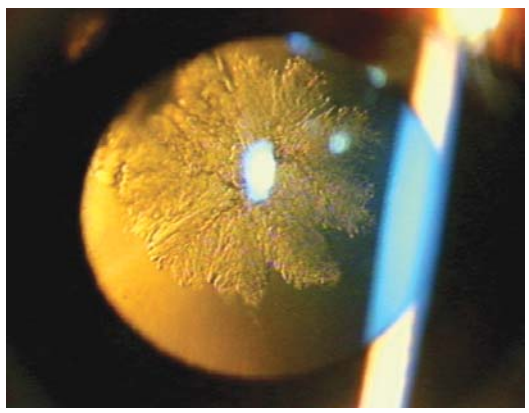


FIGURE 8.34.4: Early rosette cataract

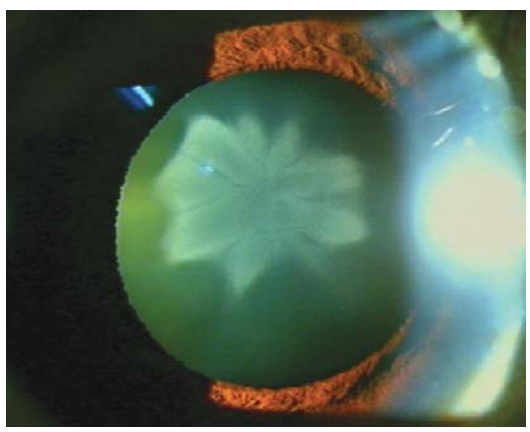


FIGURE 8.34.5: Late rosette cataract

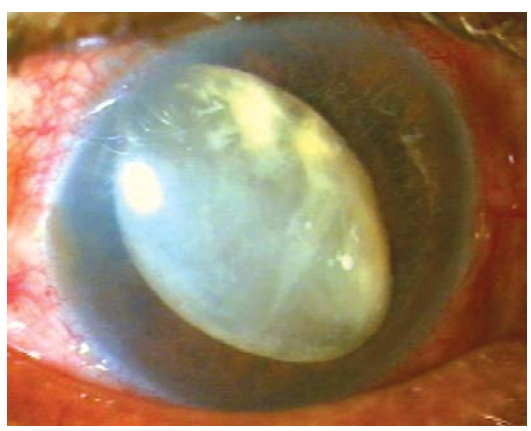


FIGURE 8.34.6: Traumatic cataract—subluxation

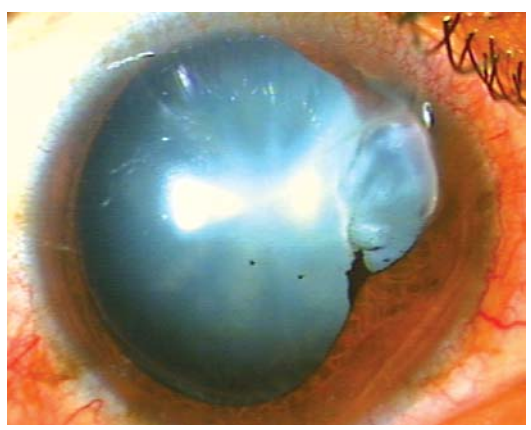


FIGURE 8.34.7: Traumatic cataract after penetrating injury

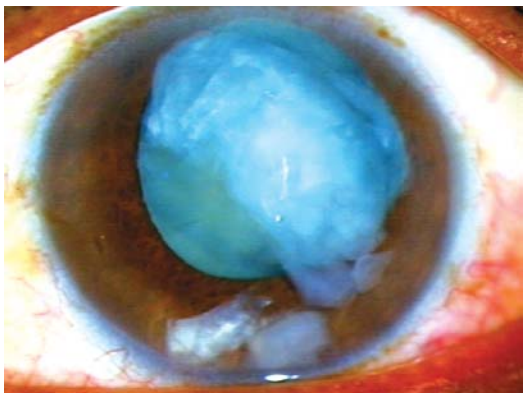


FIGURE 8.34.8: Traumatic cataract with fluffy cortex

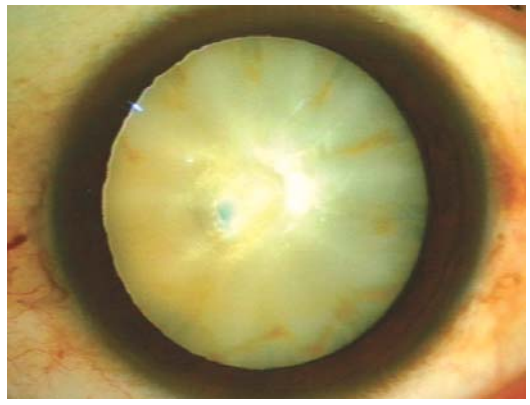


FIGURE 8.34.9: Traumatic cataract with lenticular FB

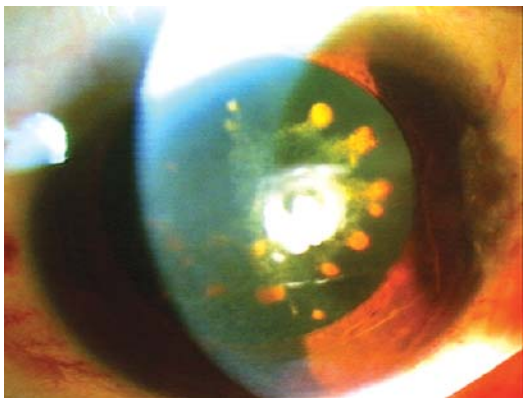


FIGURE 8.34.10: Traumatic cataract with siderosis

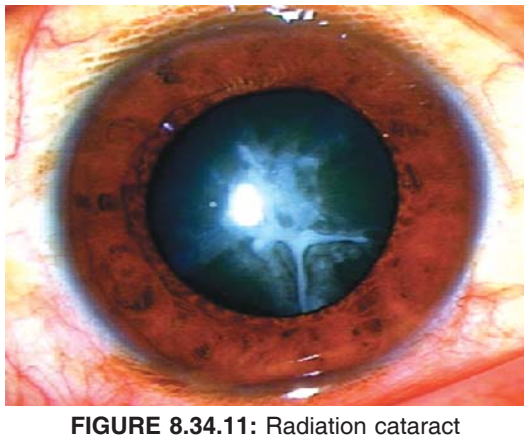


FIGURE 8.34.11: Radiation cataract

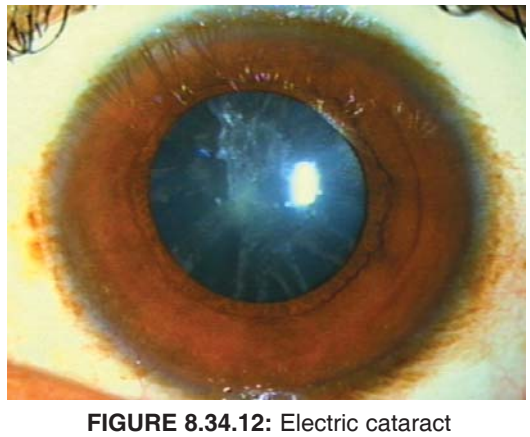


FIGURE 8.34.12: Electric cataract

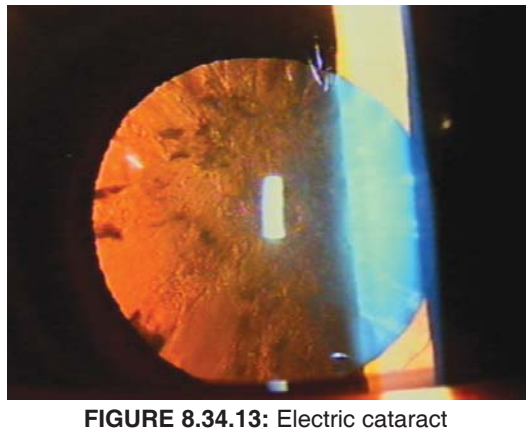


FIGURE 8.34.13: Electric cataract

Glaukomfleckens

- Not so uncommon, usually unilateral condition
- Follows after an acute attack of angle closure glaucoma
- Grayish-white opacity in the pupillary zone which resembles 'spilled milk' **(Fig 8.35.1)**
- Associated with iris-sphincter atrophy **(Fig 8.35.2)**

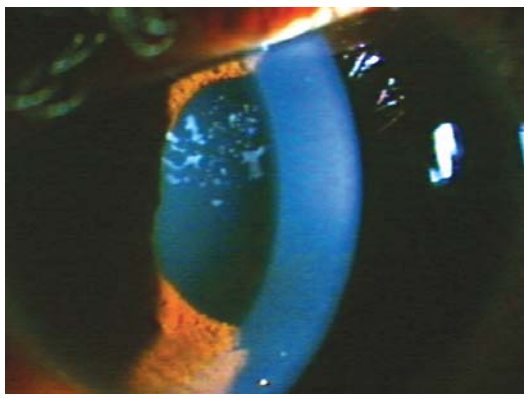


FIGURE 8.35.1: Glaukomfleckens

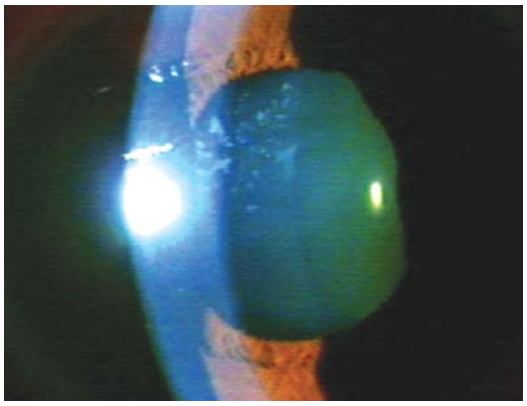


FIGURE 8.35.2: Glaukomfleckens—after PACG attack

OPACITIES IN PSEUDOPHAKIA

Anterior Capsular Opacification/Fibrosis

- Usually appears within 3 to 6 months after the surgery **(Fig 8.36.1)**
- Fibrosis may be associated with or without capsular phimosis **(Figs 8.36.2 and 8.36.3)**
- Capsulorhexis may be small or presence of signs of iridocyclitis **(Fig 8.36.4)**
- May be associated with posterior capsular opacification **(Fig 8.36.5)**
- *Treatment:* in extreme cases YAG laser anterior capsulotomy may be required

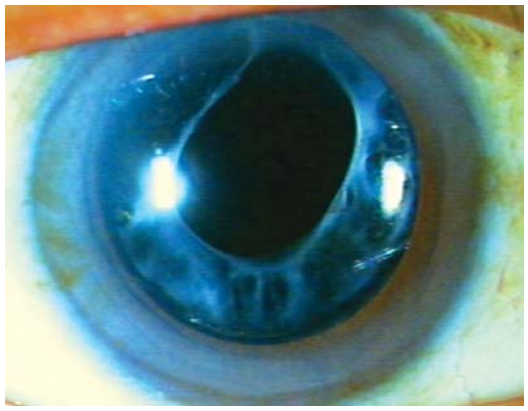


FIGURE 8.36.1: Anterior capsular opacification

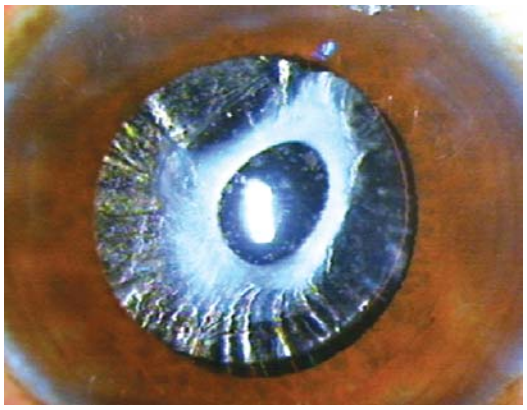


FIGURE 8.36.2: ACO with phimosis

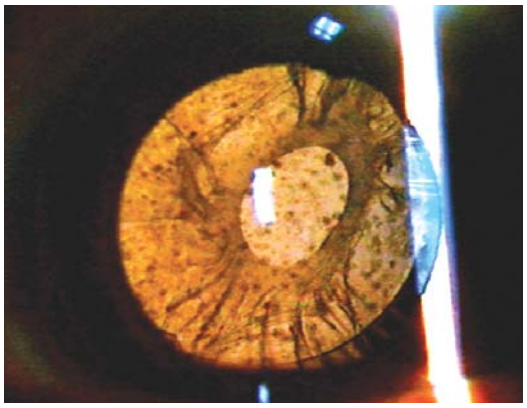


FIGURE 8.36.3: ACO with phimosis

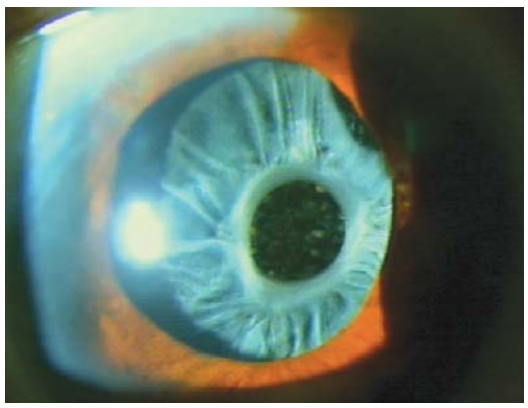


FIGURE 8.36.4: Capsular phimosis with PCO

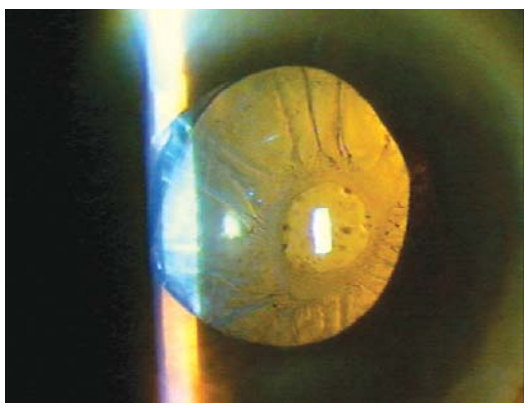


FIGURE 8.36.5: Capsular phimosis with PCO

Posterior Capsular Opacification (PCO) or 'After Cataract'

- In adults between 1 to 35 percent cases after surgery
- In infant may be upto 100 percent cases
- Membranous white irregular opacity with or without Elschnig's pearl
- Significantly reduces the vision
- After extracapsular cataract extraction
- *Posterior capsular fibrosis:*
  - membranous, white capsular fibrosis formed by the remnants of anterior and posterior capsules of the lens **(Figs 8.37.1 and 8.37.2)**
  - causes less disturbances in vision

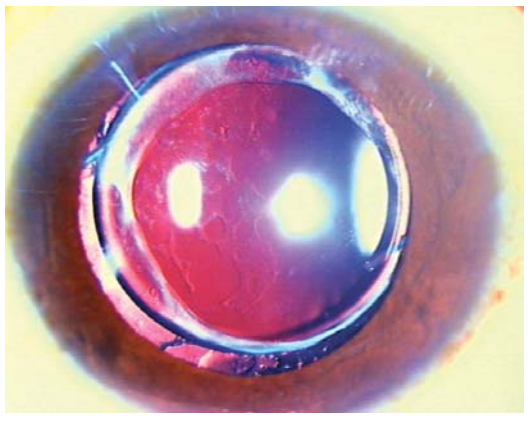


FIGURE 8.37.1: Early fibrous PCO

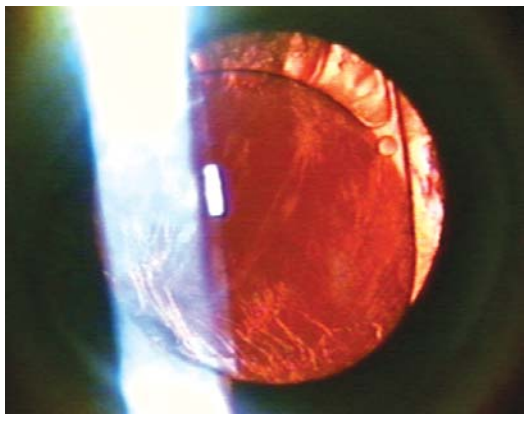


FIGURE 8.37.2: Fibrous PCO

- *Elschnig's pearls*
  - subcapsular cubical cells proliferate and instead of forming lens fibers, they develop into large balloon-like cells which fill the papillary aperture **(Figs 8.37.3 and 8.37.4)**
  - this balloon lens-cell looks like pearl, and is known as Elschnig's pearl **(Fig 8.37.5)**
  - more common in young patients
  - appear after several months or years
  - causes more disturbances in vision **(Fig 8.37.6)**
- *Treatment:*
  - *YAG-laser capsulotomy* **(Figs 8.37.7 and 8.37.8)**
  - in case of children and young adults, even after YAG-laser capsulotomy the Elschnig's pearls continue to grow and form a *pearl necklace* around the capsulotomy opening **(Figs 8.37.9 to 8.37.11)**

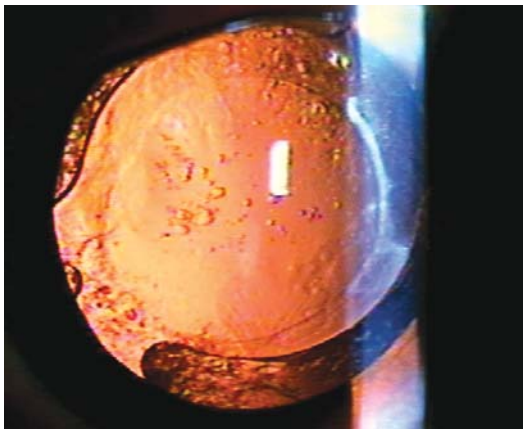


FIGURE 8.37.3: PCO—Elschnig's pearl—early

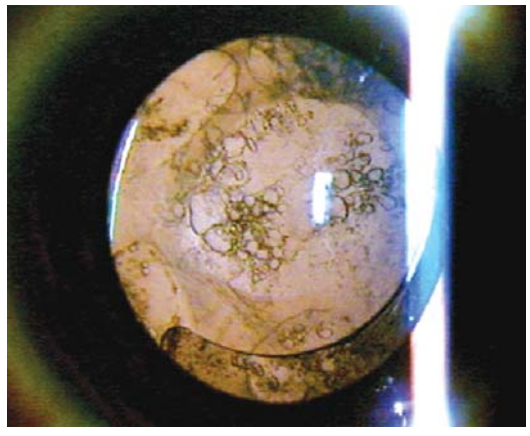


FIGURE 8.37.4: PCO—Elschnig's pearl

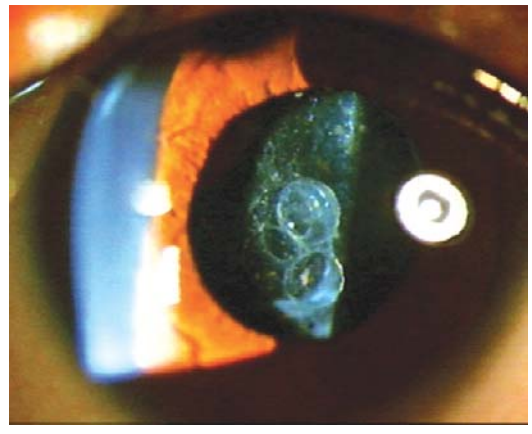


FIGURE 8.37.5: PCO—Elschnig's pearls

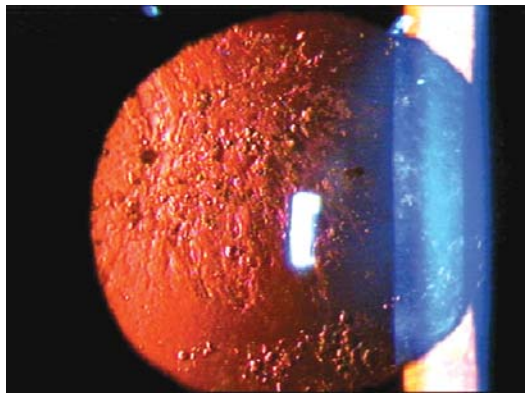


FIGURE 8.37.6: PCO—Elschnig's pearl

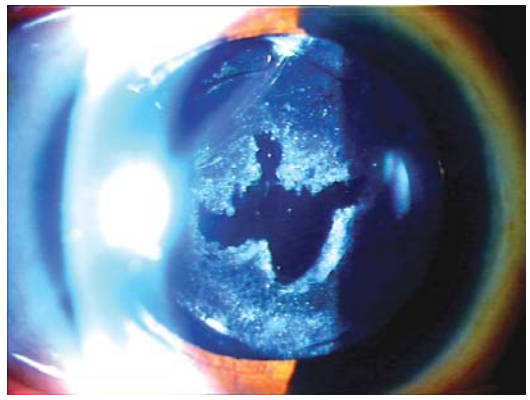


FIGURE 8.37.7: YAG capsulotomy in PCO

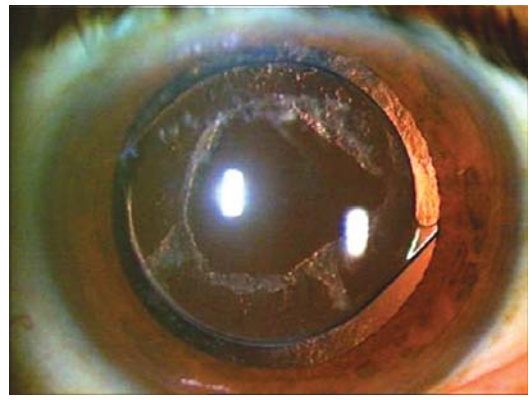


FIGURE 8.37.8: YAG capsulotomy in PCO

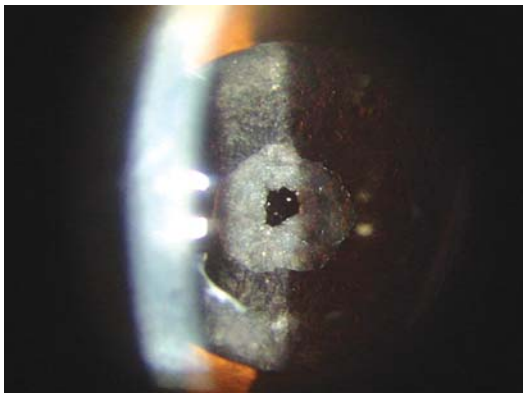


FIGURE 8.37.9: Pearl necklace after YAG capsulotomy

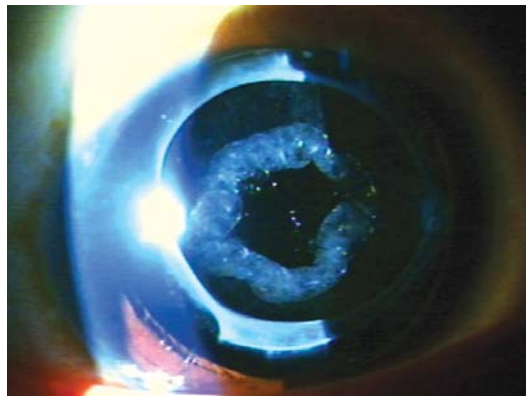


FIGURE 8.37.10: Pearl necklace after YAG capsulotomy

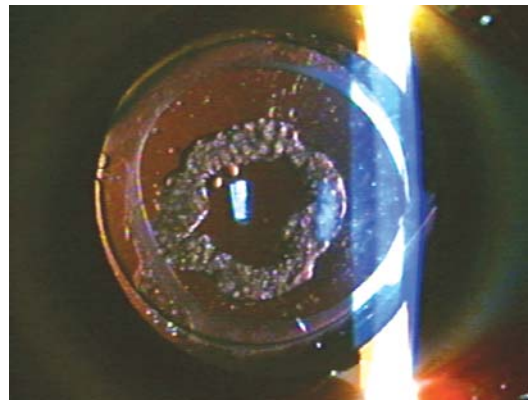


FIGURE 8.37.11: Pearl necklace after YAG capsulotomy

Soemmering's Ring

- Doughnut-like white ring behind the iris **(Fig 8.38.1)**
- Formed by the lens fibers enclosed between the two layers of lens capsule at the equatorial region
- Found in some cases where the peripheral cortex not cleaned properly

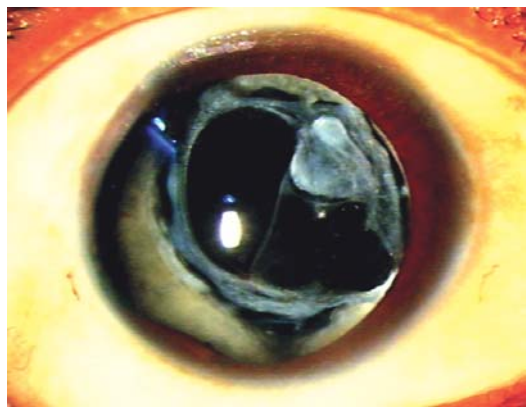


FIGURE 8.38.1: Soemmering ring

Cortical Matter Behind the IOL

- Associated with improper cleaning of posterior sheet of cortex during surgery **(Fig 8.39.1)**
- *Treatment*: to be removed surgically as early as possible

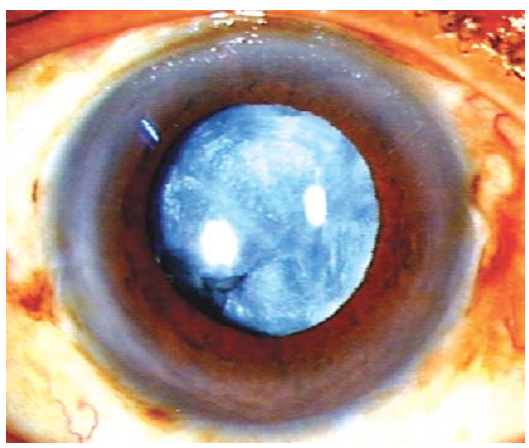


FIGURE 8.39.1: Cortical matter behind IOL

Inflammatory Plaques

- Associated with chronic iridocyclitis in some cases of aphakia **(Fig 8.40.1)** or pseudophakia **(Fig 8.40.2)**
- Localized fluffy white thickening of the capsule which are often pigmented **(Fig 8.40.3)**
- *Treatment:* YAG laser capsulotomy

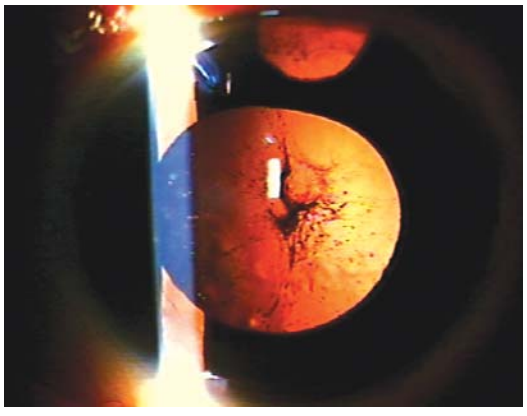


FIGURE 8.40.1: Pigmentary PCO in aphakia

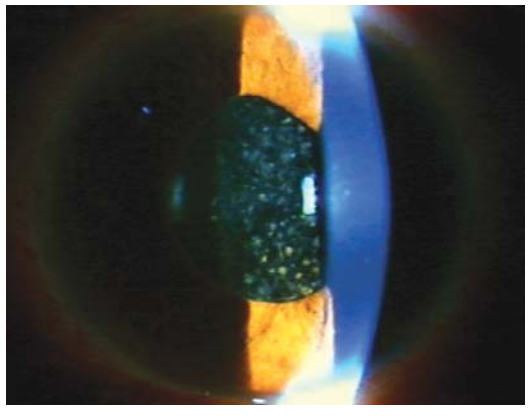


FIGURE 8.40.2: Inflammatory deposits in pseudophakia

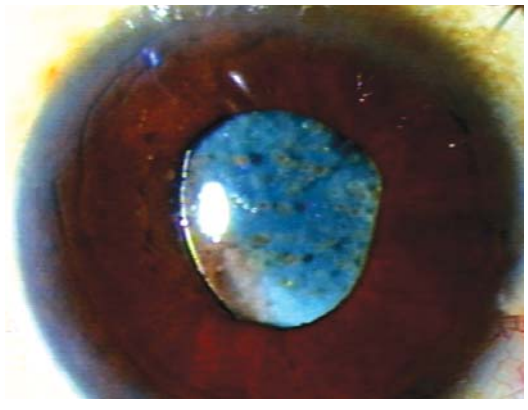


FIGURE 8.40.3: Dense PCO—inflammatory

Posterior Capsular Folds

- Seen in some cases of pseudophakia, due to abnormality in IOL- size placed in-the-bag
- Capsule is stretched in one direction (parallel to the haptics) with capsular folds **(Fig 8.41.1)**
- May give rise to Maddox rod effects
- Sometimes, may create star folds **(Fig 8.41.2)**

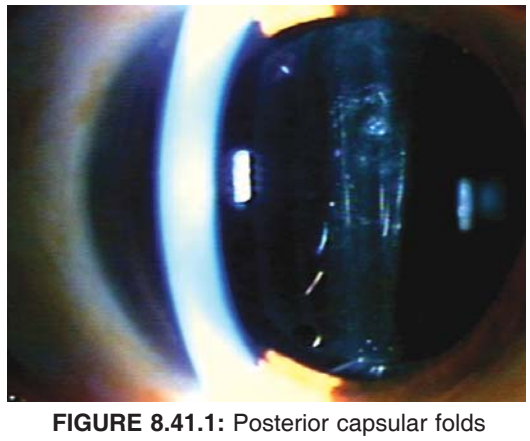


FIGURE 8.41.1: Posterior capsular folds

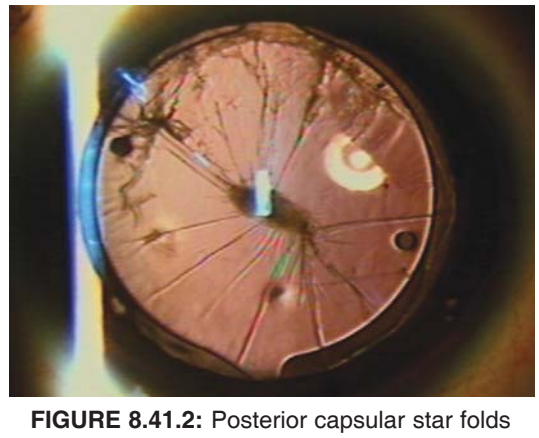


FIGURE 8.41.2: Posterior capsular star folds

Pseudophakic Opacities/Defects

- *Inflammatory precipitates:* associated with postoperative uveitis **(Figs 8.42.1 to 8.42.3)**
- *Pigments deposition*: associated with iritis **(Fig 8.42.4)**
- *Pitting:* seen after inadvertent injury to the IOL optic by YAG laser **(Fig 8.42.5)**
- *Opacification:* seen in some cases of hydrophilic foldable IOL **(Figs 8.42.6 and 8.42.7)**
- *Blue discoloration:* seen in hydrophilic foldable IOL after trypan blue capsulorhexis
- *Cracking of IOL optic:* may be seen in any type of IOL **(Figs 8.42.8 and 8.42.9)**
- *Yellow coloration:* with yellow blue-blocker IOLs
- *Multiple rings:* normal with multifocal IOL **(Fig 8.42.10)**

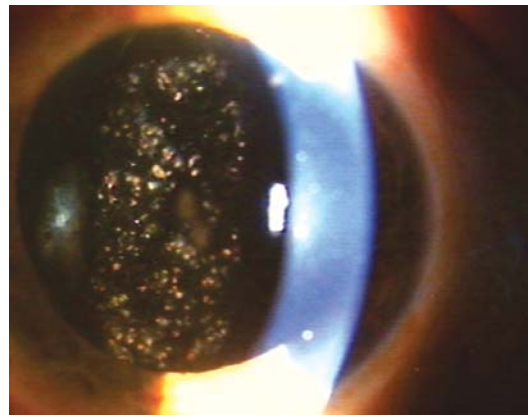


FIGURE 8.42.1: Inflammatory deposit

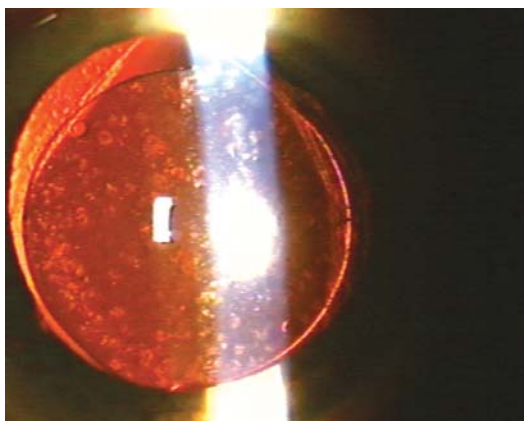


FIGURE 8.42.2: Inflammatory plaques in pseudophakia

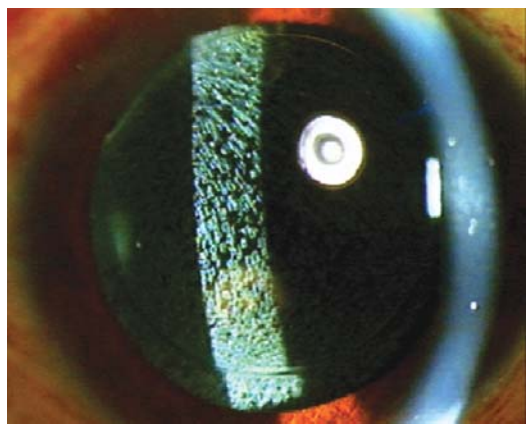


FIGURE 8.42.3: Inflammatory deposits on IOL surface

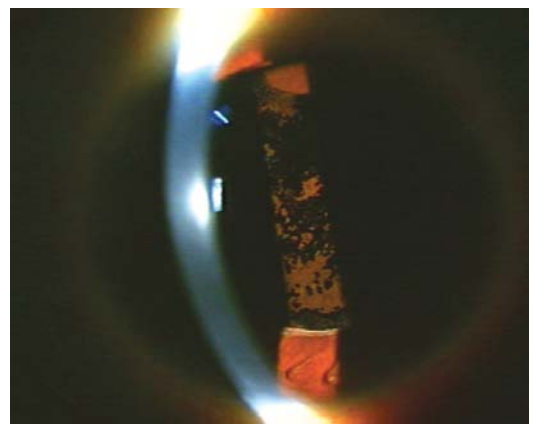


FIGURE 8.42.4: Pigment deposition on IOL

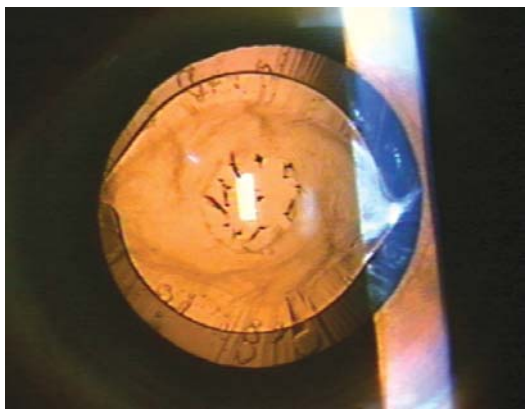


FIGURE 8.42.5: IOL pitting after YAG capsulotomy

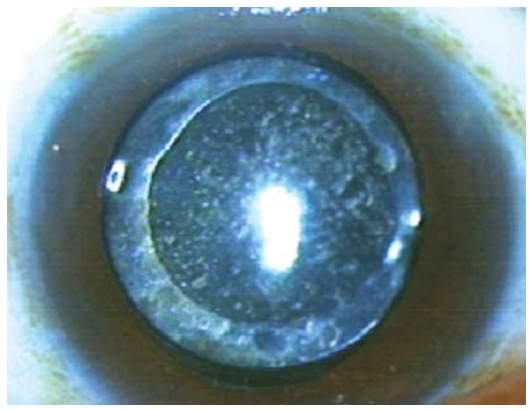


FIGURE 8.42.6: Opacification—hydrophilic IOL

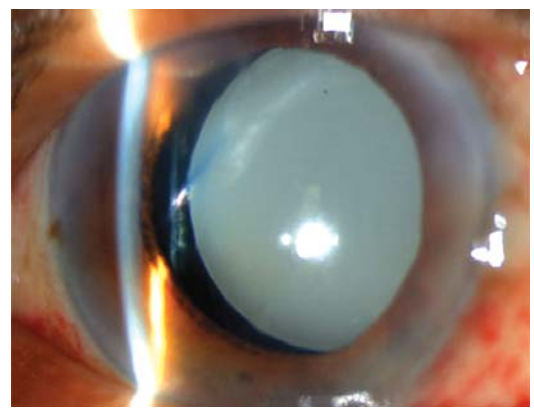


FIGURE 8.42.7: Opacification—silicone IOL

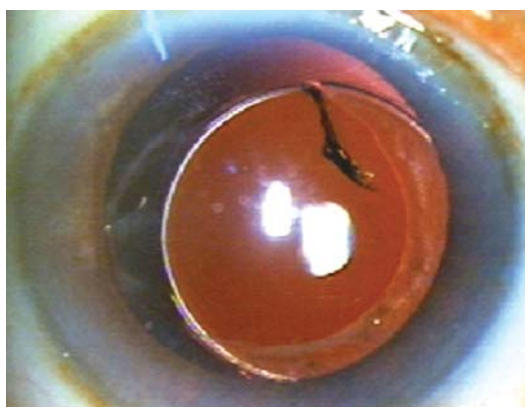


FIGURE 8.42.8: IOL—optic crack

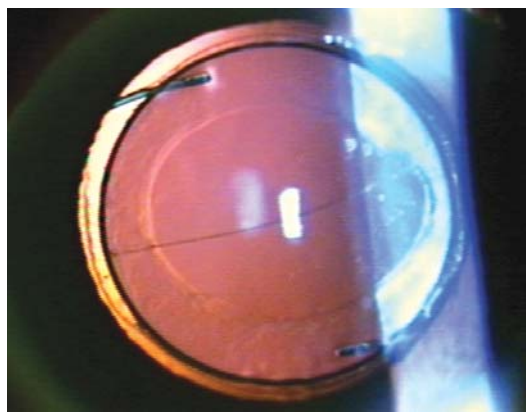


FIGURE 8.42.9: IOL—optic crack

FIGURE 8.42.10: Multiple ring—IOL optic

DISPLACEMENT OF THE CRYSTALLINE LENS AND IOLs

- There may be subluxation or dislocation of the crystalline lens and IOLs
- *Causes:*
  - *congenital ectopia lentis* (already discussed)
  - *acquired:* traumatic, degenerative, syphilis, hypermaturity, anterior uveal tumor, etc.
  - *for IOL:* capsulo-zonular problems during operation, trauma or spontaneous

Subluxation of the Crystalline Lens

- A portion of the supporting zonules is absent, and the lens lacks support in that quadrant
- Diagnosis should be confirmed after full dilatation of the pupil **(Figs 8.43.1 and 8.4.3.2)**
- Edge of the lens is visible as a golden crescentic line in oblique illumination **(Fig 8.43.3)**
- May be associated with vitreous prolapse in the anterior chamber **(Fig 8.43.4)**

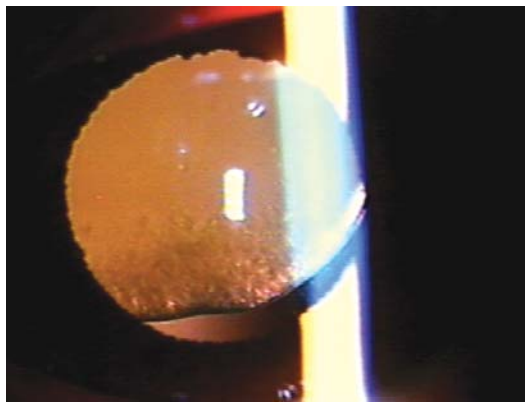


FIGURE 8.43.1: Subluxation of lens

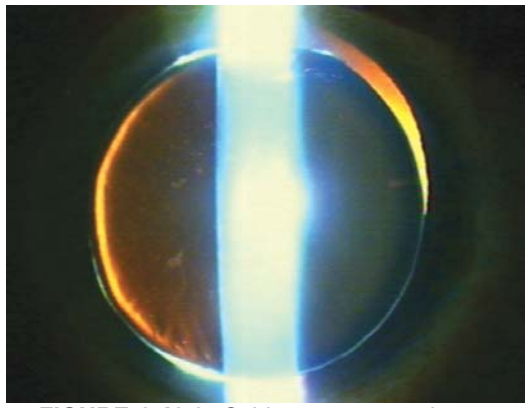


FIGURE 8.43.2: Subluxation—360 degree

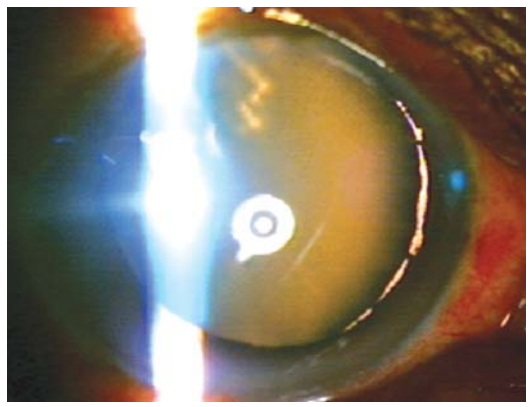


FIGURE 8.43.3: Subluxation—golden ring

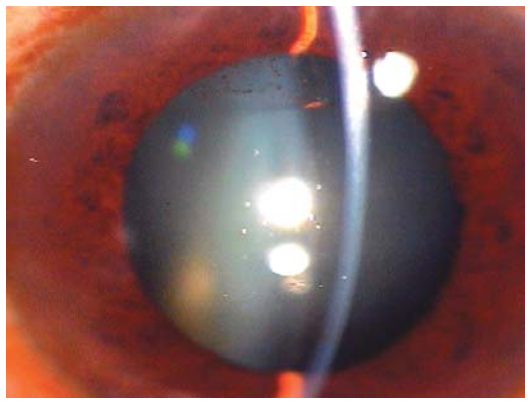


FIGURE 8.43.4: Subluxation—vitreous in AC

Dislocation of the Crystalline Lens

- Crystalline lens is completely unsupported by the zonular fibers
- Displaced from the pupillary area
- Presence of signs of aphakia
- Associated with other signs in traumatic cases
- Lens dislocation may be anterior, posterior or rarely subconjunctival **(Fig 8.44.1)**
- *Anterior dislocation:*
  - lens is dislocated into the bottom of the anterior chamber
  - appears as an 'oil-globule' due to total internal reflection **(Fig 8.44.2)**
  - cataractous lens may also dislocate anteriorly **(Fig 8.44.3)**
- *Posterior dislocation:*
  - lens can be seen as translucent or opaque mass in the vitreous cavity **(Fig 8.44.4)**
  - may be fixed or mobile (wandering) in the vitreous

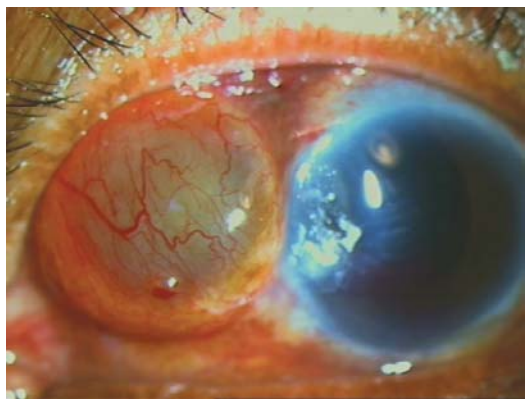


FIGURE 8.44.1: Subconjunctival dislocation of lens after trauma

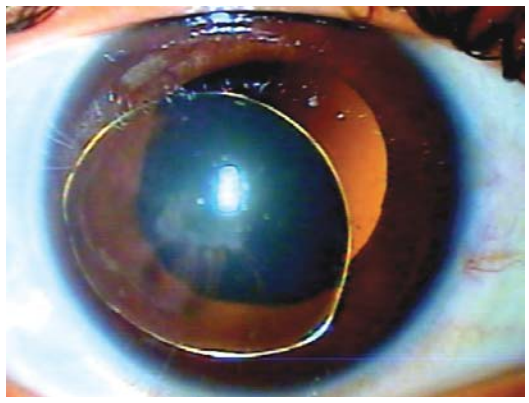


FIGURE 8.44.2: Anterior dislocation of lens

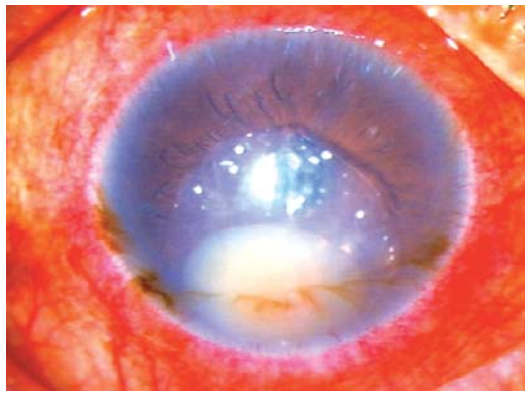


FIGURE 8.44.3: Anterior dislocation of cataractous lens

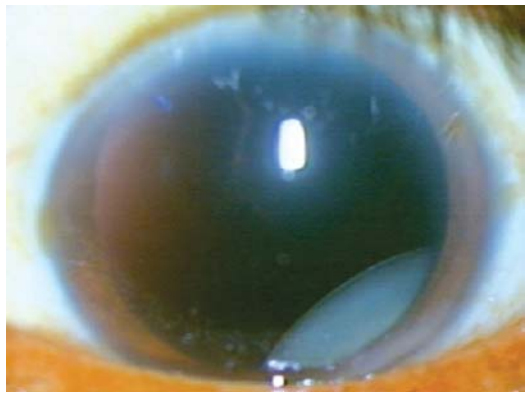


FIGURE 8.44.4: Dislocation of lens in vitreous

Displacement of the IOL

- Ideally, it should be well-centered and preferable in-the-bag **(Figs 8.45.1 and 8.45.2)**
- Displacement usually associated with posterior capsular rent of variable degree
- May be displaced downwards (sunset sign) **(Fig 8.45.3)**, upwards **(Fig 8.45.4)** or sideways **(Fig 8.45.5)**
- Diagnosis is usually obvious after full dilatation of the pupil
- Sometimes only the haptic is visible in the pupillary area **(Fig 8.45.6)**
- An IOL may be dislocated in the vitreous and the pupillary aperture remains clear
- A decentration may occur without a PC rent **(Fig 8.45.7)**
- Rarely may dislocate in the subconjunctival space after a trauma, called *pseudophacocele* **(Fig 8.45.8)**

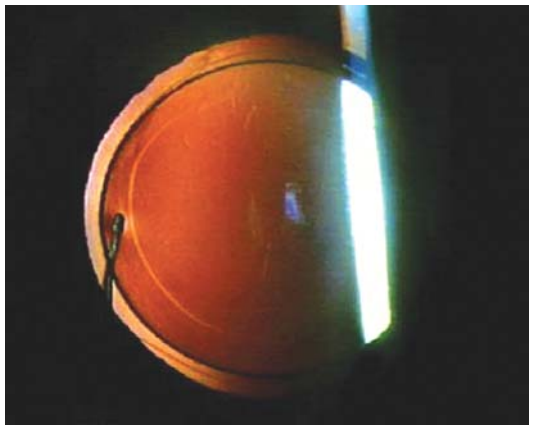


FIGURE 8.45.1: Foldable IOL in-the-bag

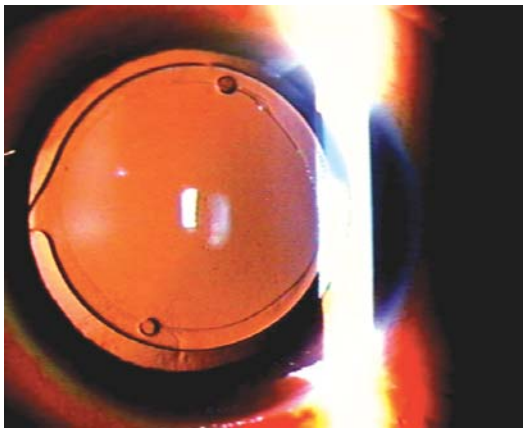


FIGURE 8.45.2: PMMA IOL in-the-bag

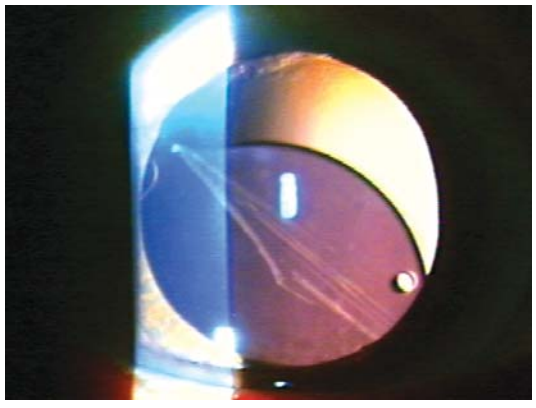


FIGURE 8.45.3: IOL—downward displacement—sunset sign

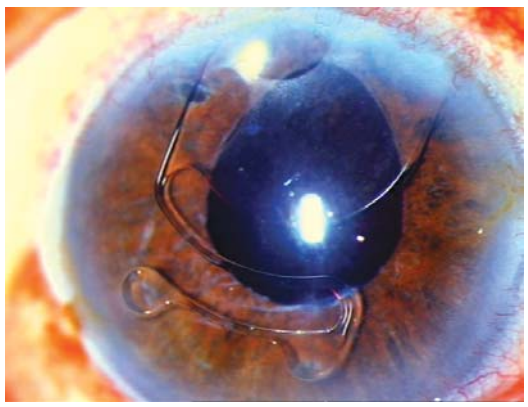


FIGURE 8.45.4: IOL—upward displacement of AC IOL

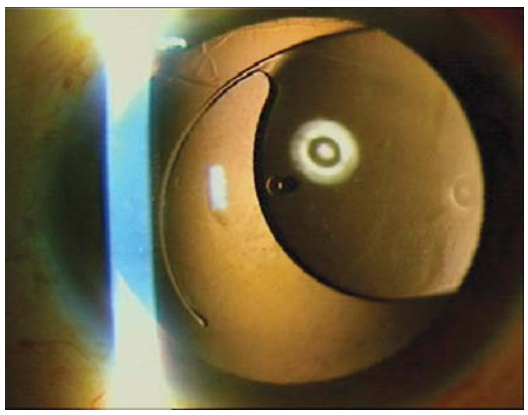


FIGURE 8.45.5: IOL—sideway displacement

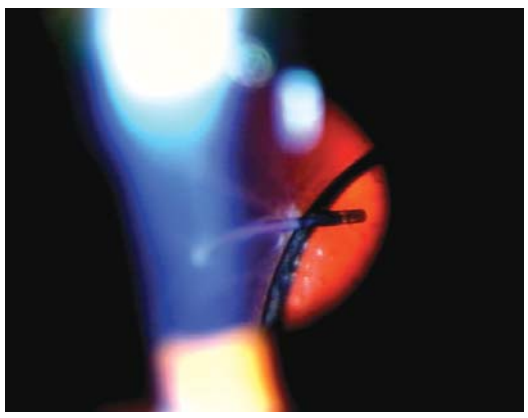


FIGURE 8.45.6: IOL dislocation—haptic visible

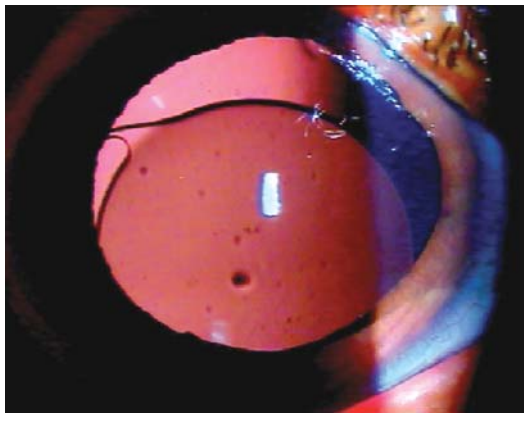


FIGURE 8.45.7: Decentration of IOL

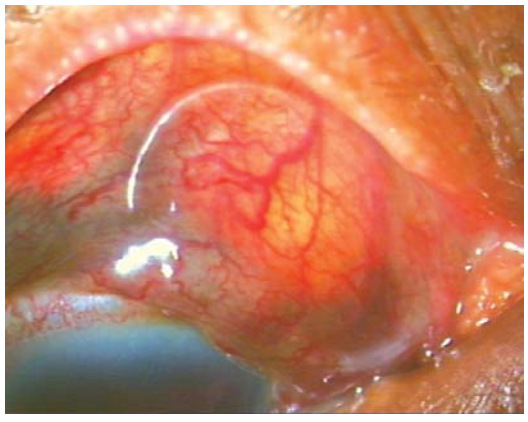


FIGURE 8.45.8: Subconjunctival dislocation of IOL—pseudophacocele

Miscellaneous Pseudophakic Conditions

- *Iris capture:* associated with both anterior and posterior chamber IOL **(Fig 8.46.1)**
- *PC IOL in AC:* usually gives rise to pseudophakic bullous keratopathy **(Figs 8.46.2 and 8.46.3)**
- *Haptic in tunnel:* in AC or Scleral fixation IOL **(Figs 8.46.4 and 8.46.5)**
- *PC rent and well-centered IOL in-the-bag* **(Fig 8.46.6)**
- *Cotton fiber behind the IOL* **(Fig 8.46.7)**
- *One haptic in anterior chamber* **(Figs 8.46.8 and 8.46.9)**
- *Unfolded haptics of foldable IOL* **(Fig 8.46.10)**

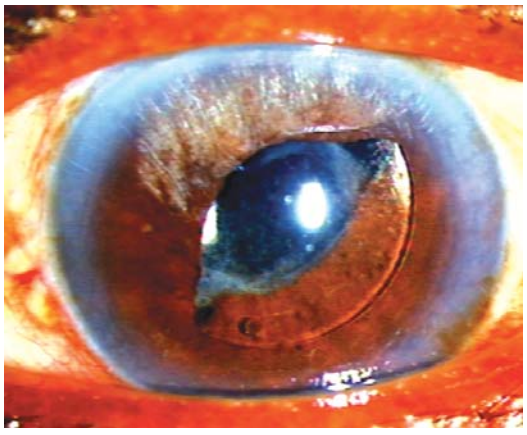


FIGURE 8.46.1: Iris capture of IOL

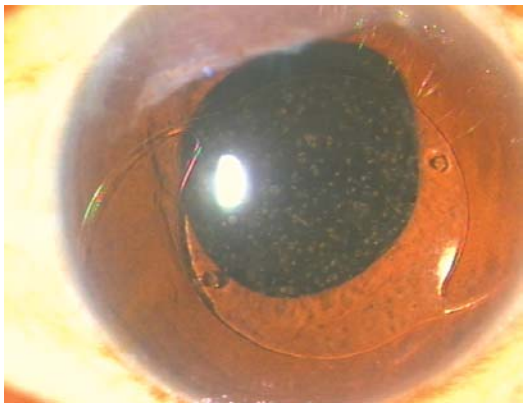


FIGURE 8.46.2: PC IOL in AC

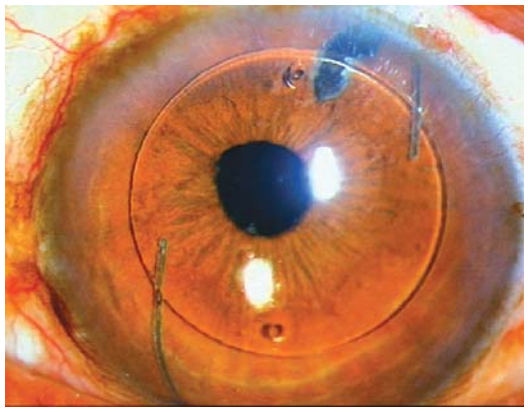


FIGURE 8.46.3: PC IOL in AC

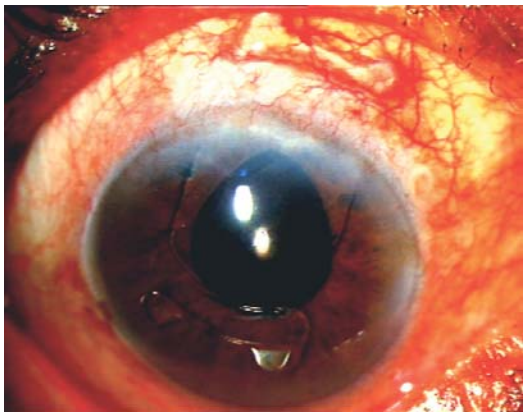


FIGURE 8.46.4: Haptic in tunnel

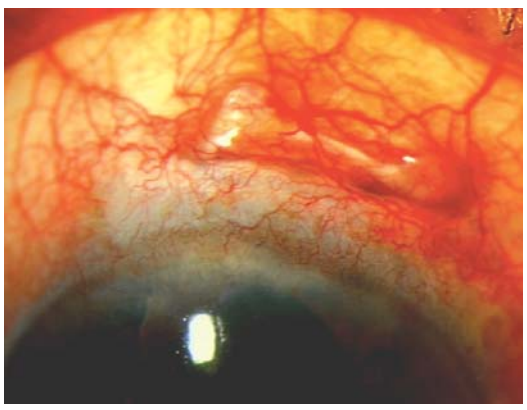


FIGURE 8.46.5: Haptic in tunnel

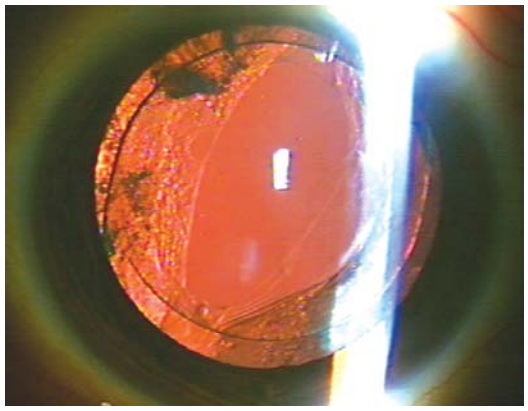


FIGURE 8.46.6: PC rent and well-centered in-the-bag IOL

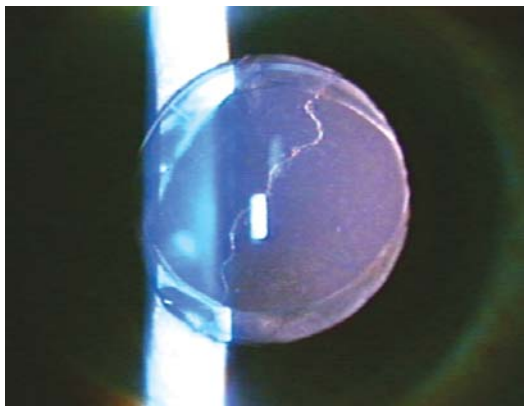


FIGURE 8.46.7: Cotton fiber behind the IOL

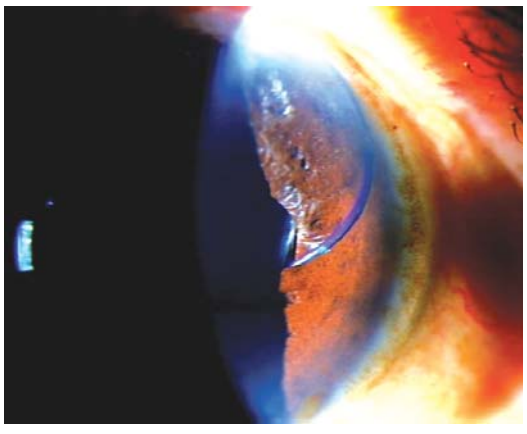


FIGURE 8.46.8: One haptic in AC

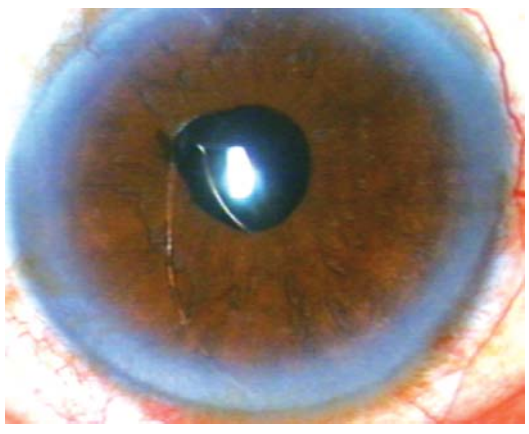


FIGURE 8.46.9: One haptic in AC

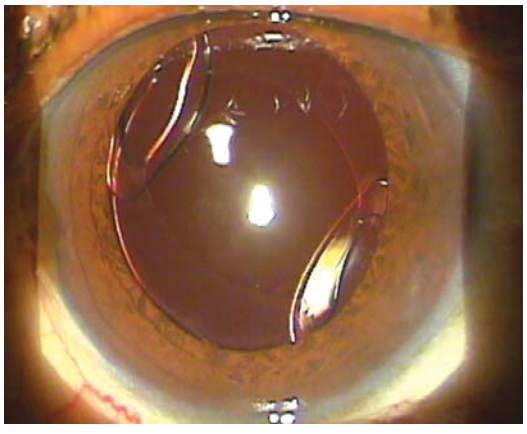


FIGURE 8.46.10: Unfolded haptic of foldable IOL

APHAKIA

- Commonest cause—surgical **(Fig 8.47.1)**, and then traumatic **(Figs 8.47.2 to 8.47.4)**, though sometimes spontaneous absorption occurs **(Fig 8.47.5)**
- Deep anterior chamber
- Jet black pupil
- Tremulousness of iris
- Associated peripheral iridectomy
- *Treatment:* Aphakic glasses, contact lens or secondary anterior chamber or scleral fixation IOL

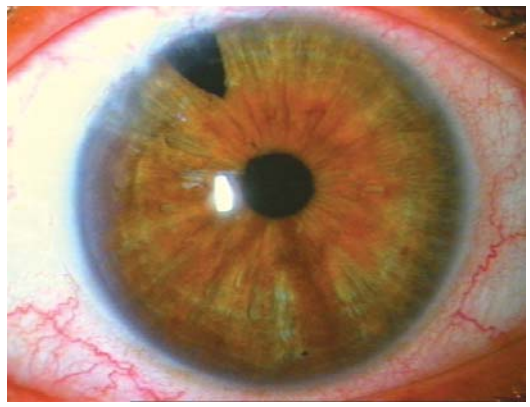


FIGURE 8.47.1: Good surgical aphakia

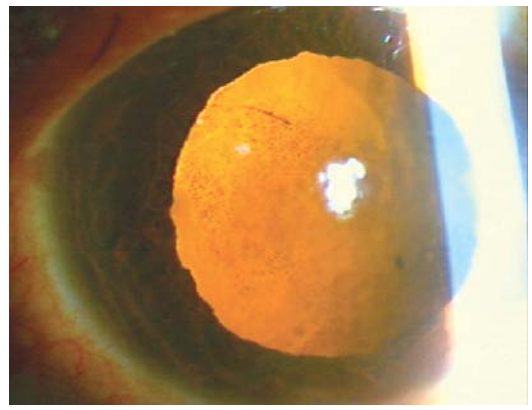


FIGURE 8.47.2: Traumatic aphakia

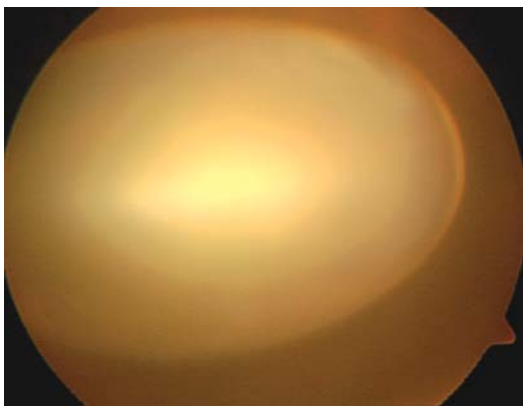


FIGURE 8.47.3: Traumatic aphakia—lens in the vitreous

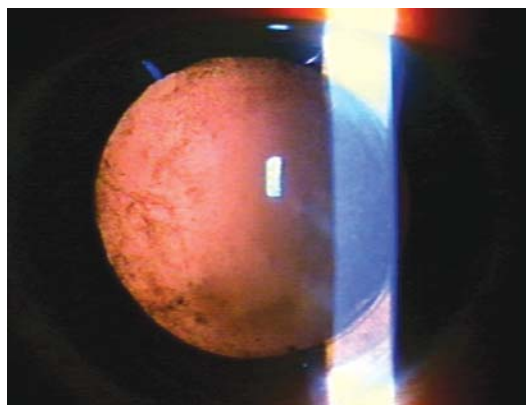


FIGURE 8.47.4: Traumatic aphakia

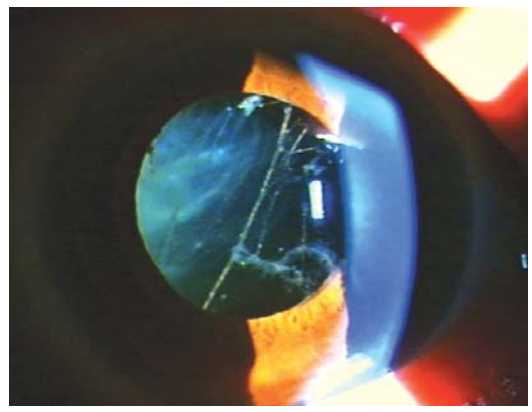


FIGURE 8.47.5: Aphakia—spontaneous absorption

9

Glaucomas

CONGENITAL OR INFANTILE GLAUCOMA (BUPHTHALMOS)

- Primary congenital glaucoma
- Secondary developmental glaucoma

PRIMARY ANGLE CLOSURE GLAUCOMA

- Acute congestive attack
- Chronic congestive stage
- Stage of absolute glaucoma

• SLIT-LAMP GRADING OF ANGLE (HERICK)

PRIMARY OPEN ANGLE GLAUCOMA

- Cupping of the optic disk

SECONDARY GLAUCOMAS

- Glaucoma capsulare
- Phacomorphic glaucoma
- Phacolytic glaucoma
- Lens-particle glaucoma
- Glaucoma associated with ectopia lentis
- Pigmentary glaucoma
- Inflammatory secondary glaucoma
- Glaucoma associated with trauma
- Glaucoma following intraocular surgery
- Malignant (ciliary block) glaucoma
- Neovascular glaucoma
- Glaucoma in iridocorneal endothelial syndrome
- Other causes of secondary glaucoma

FILTERING BLEB ABNORMALITIES

- Normal functioning bleb
- Failed filtering bleb
- Cystic filtering bleb
- Multilocular filtering bleb
- Overhanging bleb
- Overfiltering filtering bleb
- Leaking filtering bleb
- Blebitis (bleb infection)

CONGENITAL OR INFANTILE GLAUCOMA (BUPHTHALMOS)

Primary Congenital Glaucoma

- Congenital or infantile glaucoma is due to simple outflow obstruction
- Rare, unilateral **(Fig 9.1.1)** or bilateral **(Fig 9.1.2)** condition
- Autosomal recessive inheritance
- Forty percent cases are true congenital and 50 percent are infantile **(Fig 9.1.3)**
- Boys are more affected than girls
- Eyeball becomes enlarged, if the IOP becomes elevated prior to age of three years
- Cornea is enlarged, globular and steamy **(Fig 9.1.4)**
- Horizontal curvilinear lines are seen on the back of the cornea, known as Haab's striae **(Fig 9.1.5)**
- Blue discoloration of sclera **(Fig 9.1.6)**
- Deep anterior chamber
- Cupping of the disk
- *Treatment:* examination under anesthesia; goniotomy, trabeculotomy., or trabeculectomy and trabeculotomy, and visual rehabilitation

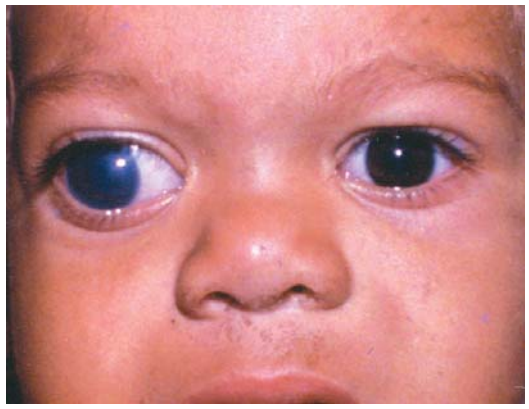


FIGURE 9.1.1: Primary congenital glaucoma

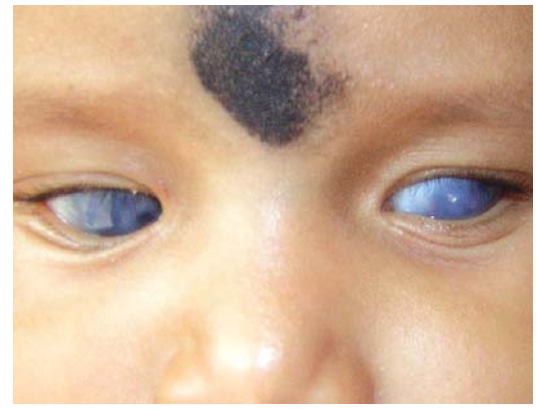


FIGURE 9.1.2: Primary congenital glaucoma—bilateral

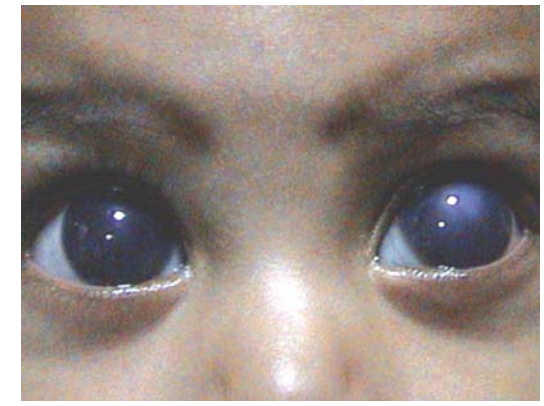


FIGURE 9.1.3: Primary infantile glaucoma

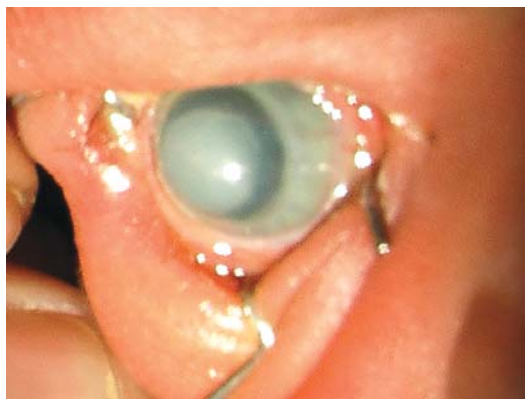


FIGURE 9.1.4: Congenital glaucoma—steamy cornea

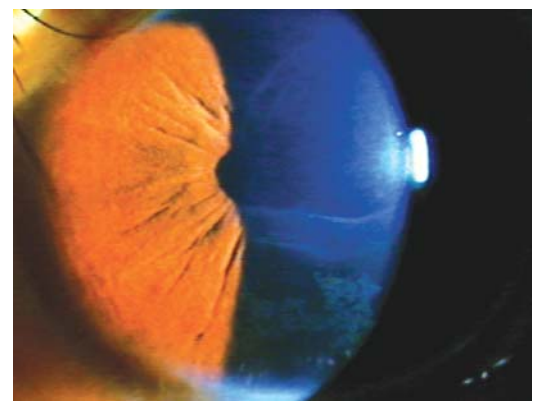


FIGURE 9.1.5: Congenital glaucoma—Haab's striae

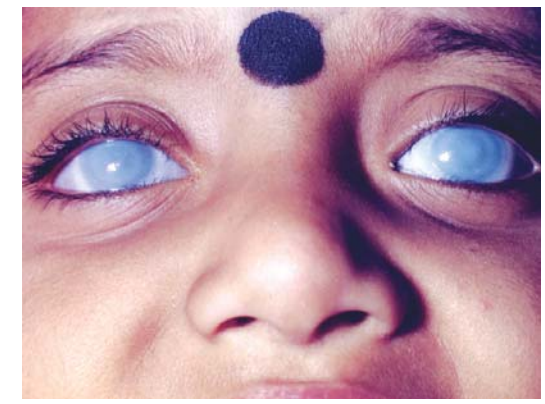


FIGURE 9.1.6: Congenital glaucoma—blue sclera

Secondary Developmental Glaucoma

May be associated with other systemic diseases (secondary form): like

- Aniridia **(Fig 9.2.1)**
- Rubella syndrome
- Sturge-Weber syndrome **(Fig 9.2.2)**
- Neurofibromatosis

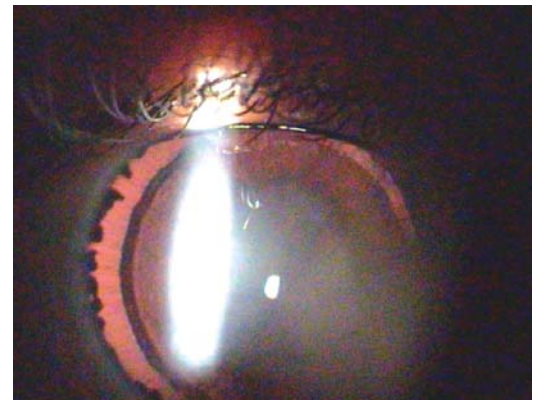


FIGURE 9.2.1: Aniridia

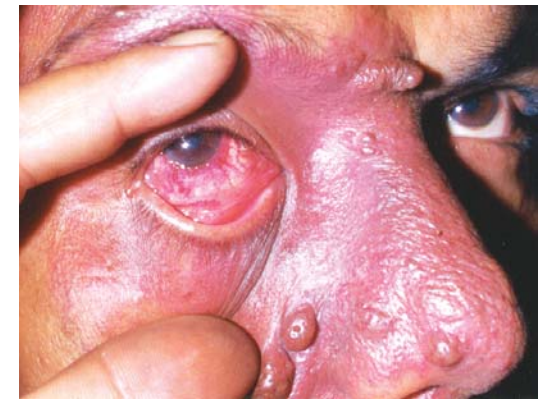


FIGURE 9.2.2: Sturge-Weber syndrome

- Mesodermal dysgenesis:
  - Axenfeld-Rieger's anomaly **(Fig 9.2.3)**,
  - Peters' anomaly **(Fig 9.2.4)**, etc.
- Manifestation occurs in early childhood or later
- Presence of other systemic signs
- *Treatment:* same as primary type

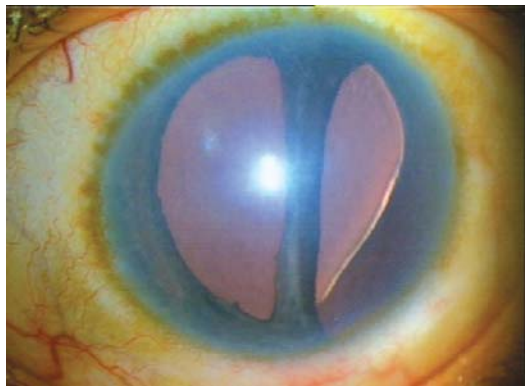


FIGURE 9.2.3: Axenfeld-Reiger's anomaly

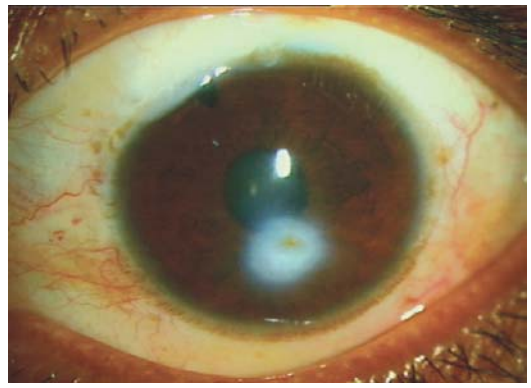


FIGURE 9.2.4: Peters' anomaly—operated

PRIMARY ANGLE CLOSURE GLAUCOMA

Primary angle closure glaucoma (PACG) is an acute, sub-acute or chronic glaucoma due to obstruction of the aqueous outflow, solely caused by closure of the angle by the peripheral iris

- Typically, the eye is hypermetropic, with shallow anterior chamber and narrow angle **(Fig 9.3.1)**

Acute Congestive Attack

- circumcorneal ciliary congestion **(Fig 9.3.2)**
- steamy and insensitive cornea
- shallow anterior chamber
- pupil is mid-dilated and oval **(Fig 9.3.3)**
- iris shows atrophic changes adjacent to the sphincter muscle
- *glaukomfleckens* **(Fig 9.3.4)** are small grayish-white anterior sub-capsular opacities occur in the pupillary zone-diagnostic of previous attack of angle-closure glaucoma
- peripheral anterior synechiae develop **(Fig 9.3.5)**, mostly in the upper part of the angle, but gradually spread around the whole circumference

Chronic Congestive Stage

- angle becomes slowly and progressively closed
- creeping angle closure **(Fig 9.3.6)**
- variable degree of cupping of the disk

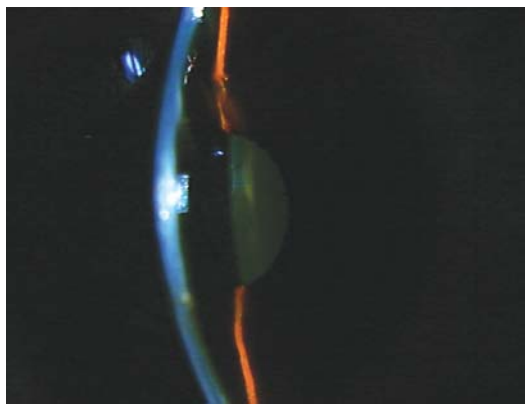


FIGURE 9.3.1: Hypermetropic eye—shallow anterior chamber

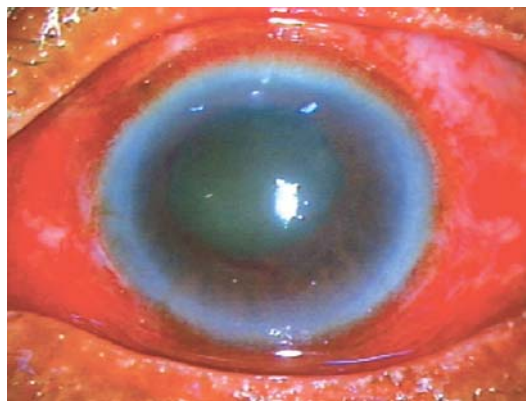


FIGURE 9.3.2: PACG—acute attack—ciliary congestion

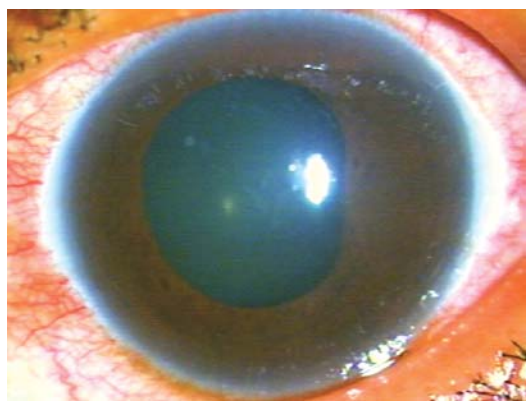


FIGURE 9.3.3: PACG—acute attack—oval pupil

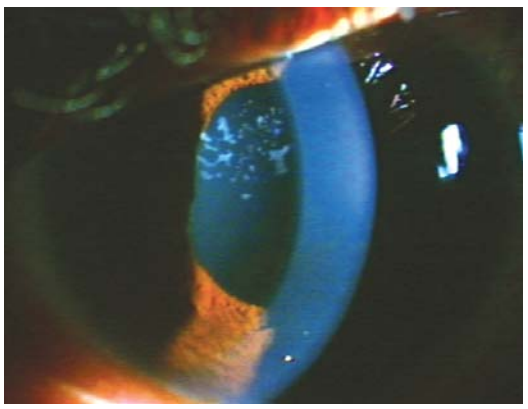


FIGURE 9.3.4: PACG—acute attack—glaukomfleckens

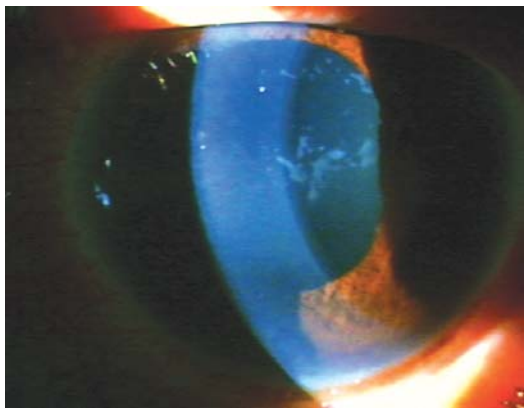


FIGURE 9.3.5: PACG—PAS formation

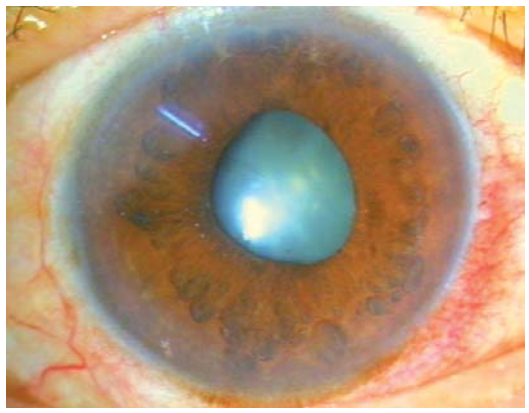


FIGURE 9.3.6: PACG—chronic angle closure

Stage of Absolute Glaucoma

- reddish-blue zone surrounding the limbus, due to dilated anterior ciliary vein
- cloudy cornea, may be bullous changes
- very shallow anterior chamber
- iris atrophy with ectropion uveae **(Fig 9.3.7)**
- dilated and grayish pupil

- *Treatment of ACG is always surgical*
  - pilocarpine (2%) eyedrop, oral acetazolamide, IV mannitol, etc. initially,
  - followed by YAG laser peripheral iridotomy **(Fig 9.3.8)** or surgical PI in initial stage
  - if the PAS is more—trabeculectomy is the treatment of choice
  - *Treatment:* of fellow eye YAG laser PI

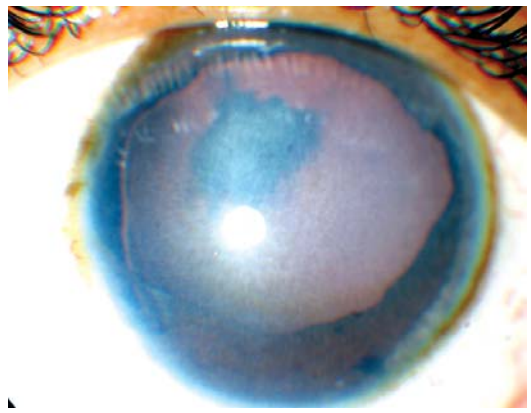


FIGURE 9.3.7: Absolute glaucoma

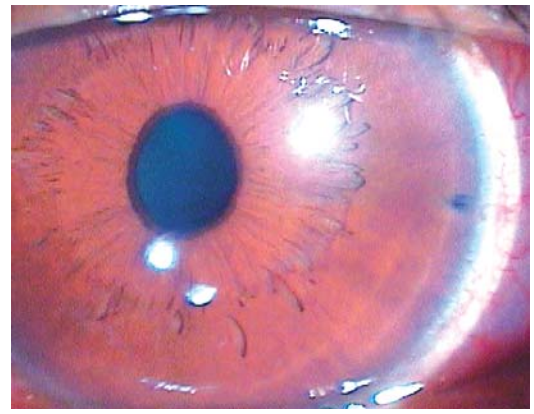


FIGURE 9.3.8: YAG PI opening

SLIT-LAMP GRADING OF ANGLE (van HERICK)

- Used with a fair accuracy
- Useful when a gonioscopy is difficult to perform
- Depth of the 'peripheral anterior chamber' (PAC) is estimated by comparing it to the adjacent 'corneal thickness' (CT) 1 mm inside the limbus
- *Four grades:*
  - *Grade 4*: PAC $\geq$ 1CT = wide open angle **(Fig 9.4.1)**
  - *Grade 3*: PAC = 1/4th to ½ CT = open angle **(Fig 9.4.2)**
  - *Grade 2*: PAC = 1/4th CT = moderately narrow **(Fig 9.4.3)**
  - *Grade 1*: PAC < 1/4th CT = extremely narrow **(Fig 9.4.4)**

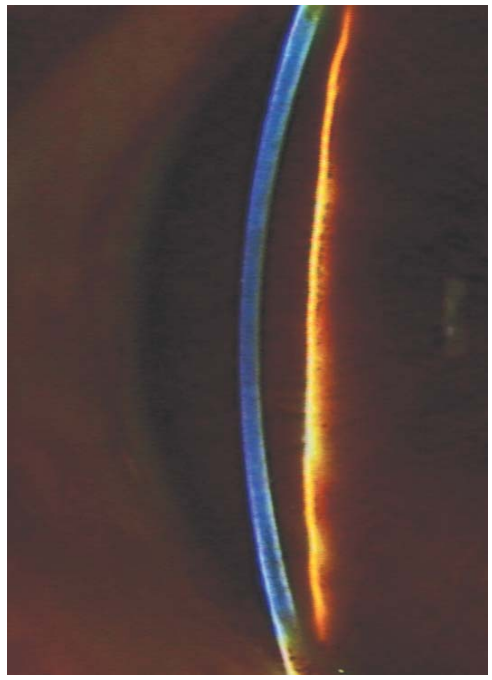


FIGURE 9.4.1: van Herick grading—Grade 4

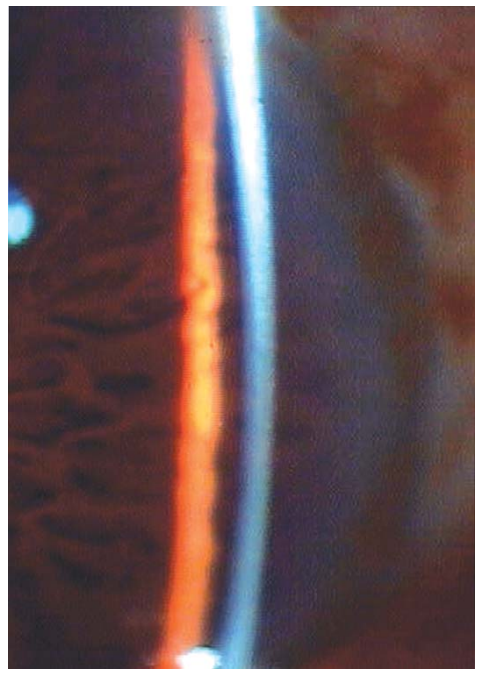


FIGURE 9.4.2: van Herick grading—Grade 3

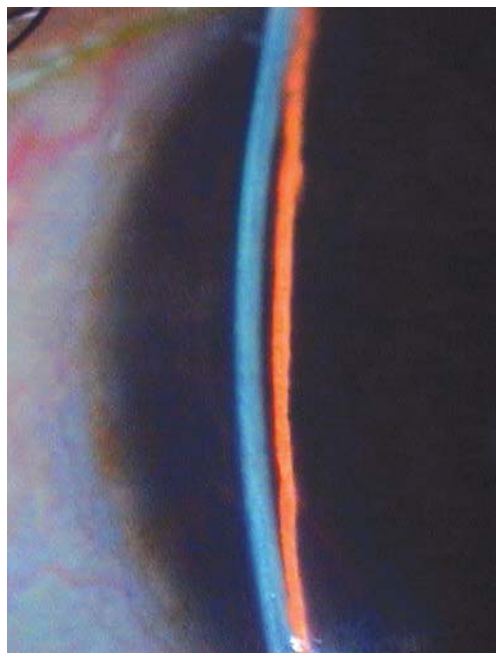


FIGURE 9.4.3: van Herick grading—Grade 2

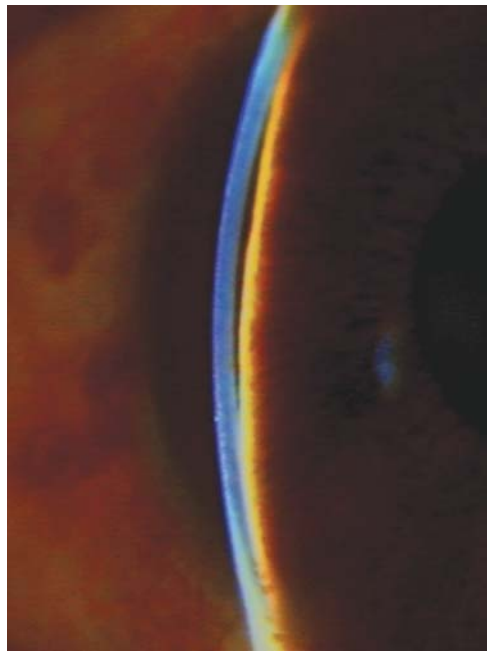


FIGURE 9.4.4: van Herick grading—Grade 1

PRIMARY OPEN ANGLE GLAUCOMA

- Chronic, bilateral slowly progressive glaucoma with typical cupping of the optic disk and characteristic field changes
- Usually older people, 40 years and above
- Positive family history in 15-20 percent cases
- Some people are corticosteroids responder
- Eyeball otherwise looks normal
- *Classical triad:* raised IOP, cupping of the disk **(Figs 9.5.1 and 9.5.2)** and classical field defects
- *Treatment: primarily medical*
  - *medical:* pilocarpine, beta blockers, carbonic anhydrase inhibitors, latanoprost, brimonidine, etc. used alone or in combination
  - *laser:* argon laser trabeculoplasty
  - *surgical:* trabeculectomy, non-penetrating deep trabeculoplasty
  - *combination therapy*

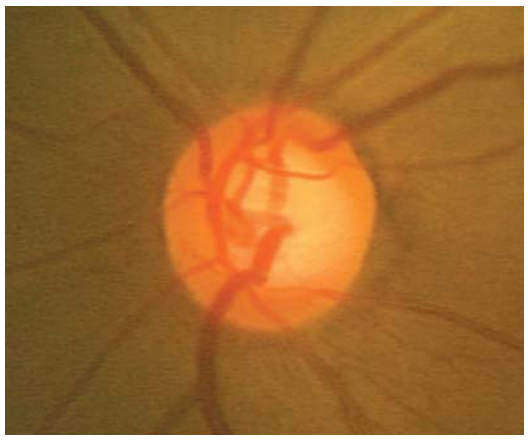


FIGURE 9.5.1: Glaucoma cupping

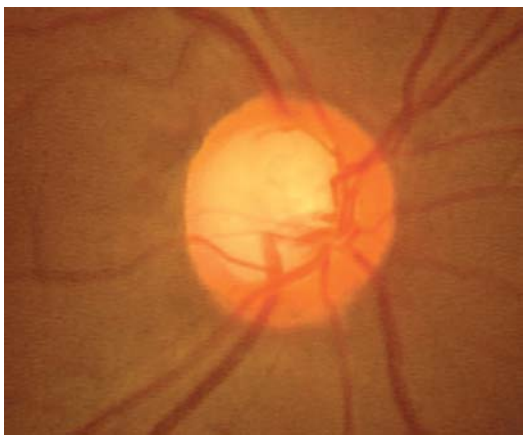


FIGURE 9.5.2: Glaucoma cupping

Cupping of the Optic Disk

- *Asymmetry of the cupping* (C:D ratio difference more than 0.2) **(Figs 9.6.1 and 9.6.2)**
- Cupping starts as focal enlargement *(notching)* at the inferotemporal quadrant **(Fig 9.6.3)**. It may be superior or both **(Figs 9.6.4 and 9.6.5)**
- *Bayonetting sign*: double angulations of the blood vessels, pass sharply backwards and then turn along the steep wall of the excavation before angling again onto the floor of the cup **(Fig 9.6.6)**

FIGURE 9.6.1: Glaucoma cupping—asymmetry

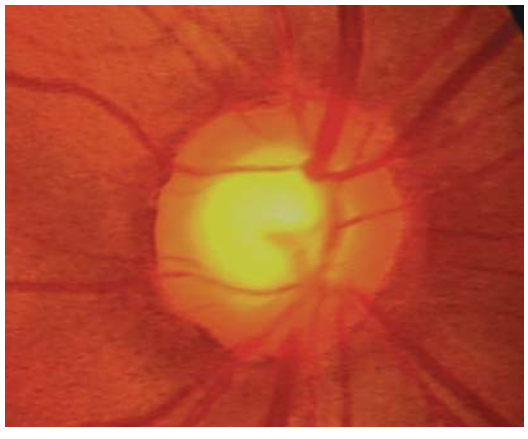


FIGURE 9.6.2: Glaucoma cupping—asymmetry

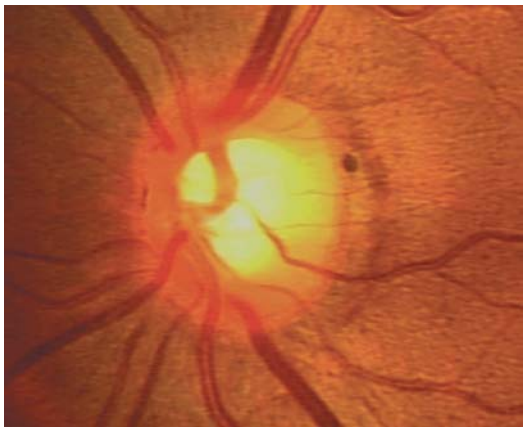


FIGURE 9.6.3: Glaucoma cupping—inferior notching

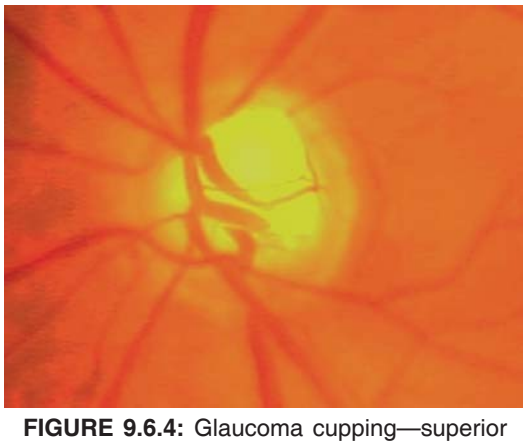


FIGURE 9.6.4: Glaucoma cupping—superior notching

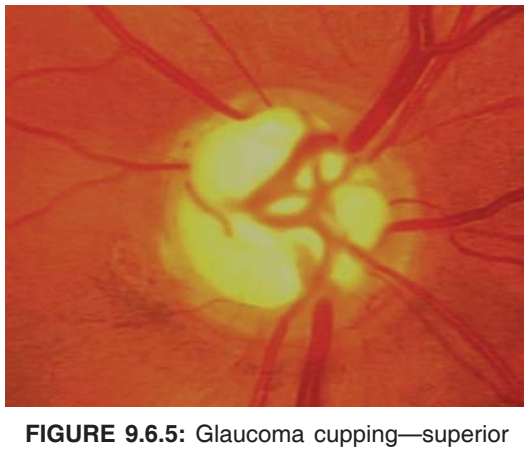


FIGURE 9.6.5: Glaucoma cupping—superior and inferior notching

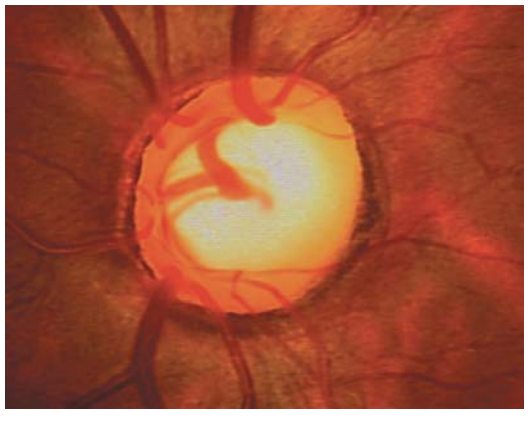


FIGURE 9.6.6: Glaucoma cupping—bayonetting sign

- *Thinning* of the neural rim **(Figs 9.6.7 and 9.6.8)**
- *Nasal shifting* of retinal blood vessels **(Fig 9.6.9)**
- *Over passing* of blood vessels **(Fig 9.6.10)**
- *Splinter hemorrhages* at the disk margin **(Fig 9.6.11)**
- *Baring of circumlinear blood vessels* may be seen at the disk margin **(Fig 9.6.12)**
- *Near total cupping* **(Fig 9.6.13)**
- *Total cupping:* it appears as a white disk with loss of all neural rim, and bending of all retinal vessels at the margin of the disk—called *'bean-pot cupping'* **(Figs 9.6.14 and 9.6.15)**
- *Total pallor of the disk*, as there is glaucomatous optic atrophy **(Figs 9.6.16 and 9.6.17)**
- Visible upto the margin of the disk *'laminar dot sign'* **(Fig 9.6.18)**

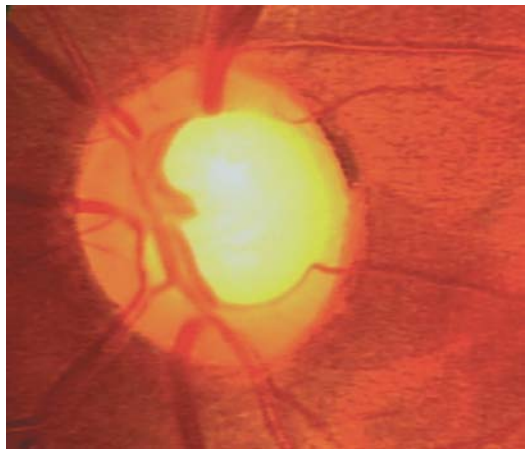


FIGURE 9.6.7: Glaucoma cupping—neuro-retinal rim thinning

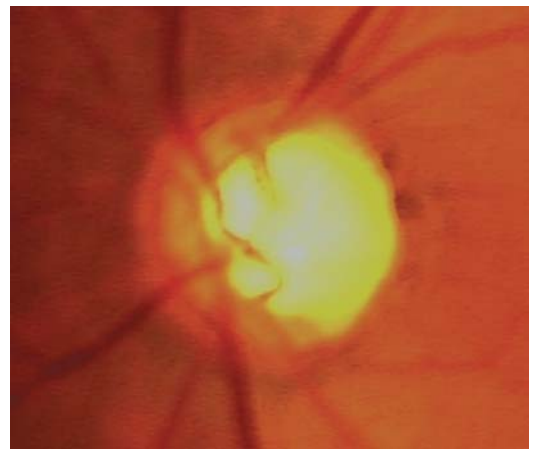


FIGURE 9.6.8: Glaucoma cupping—neuroretinal rim thinning

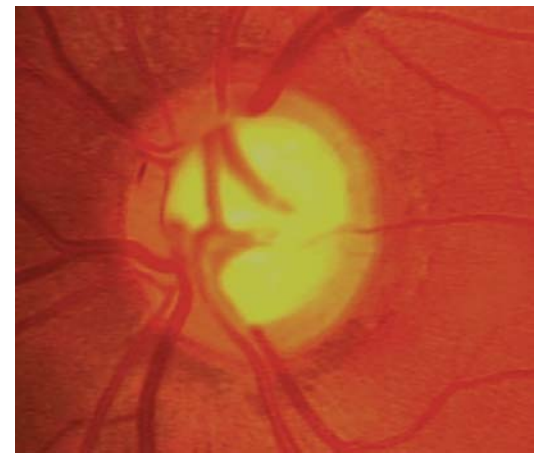


FIGURE 9.6.9: Glaucoma cupping—nasal shifting of the blood vessels

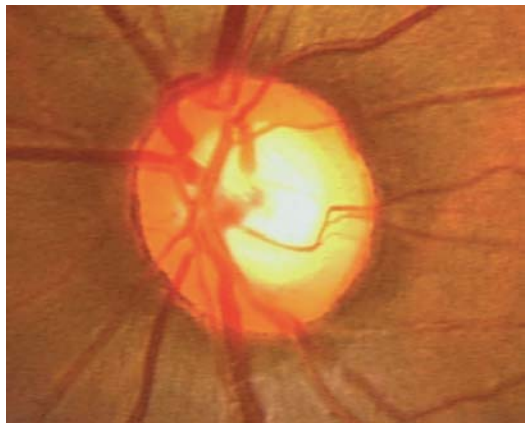


FIGURE 9.6.10: Glaucoma cupping—overpassing phenomena

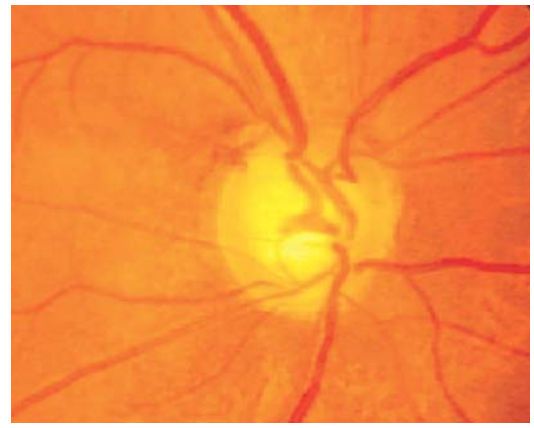


FIGURE 9.6.11: Glaucoma cupping—splinter hemorrhage

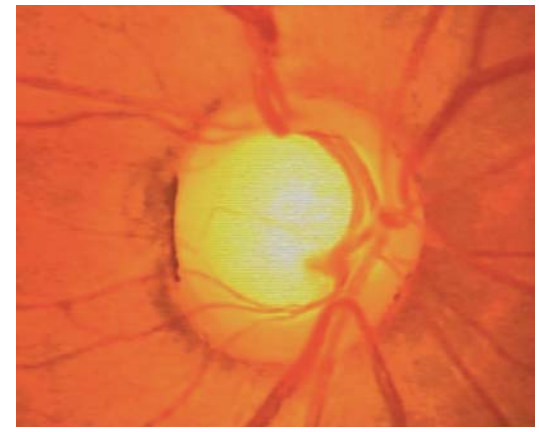


FIGURE 9.6.12: Glaucoma cupping—baring of blood vessels

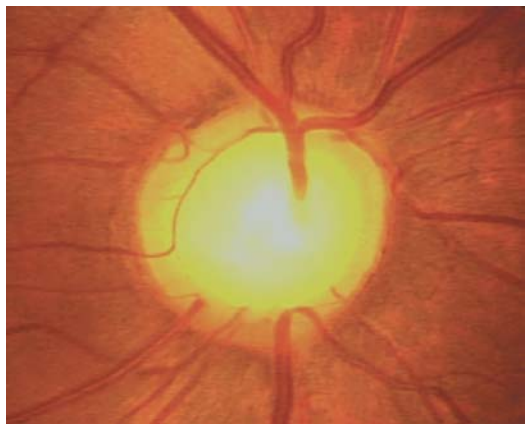


FIGURE 9.6.13: Glaucoma cupping—near total

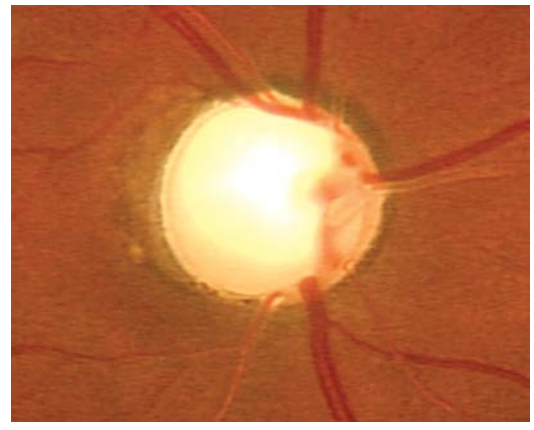


FIGURE 9.6.14: Glaucoma total cupping—bean pot cupping

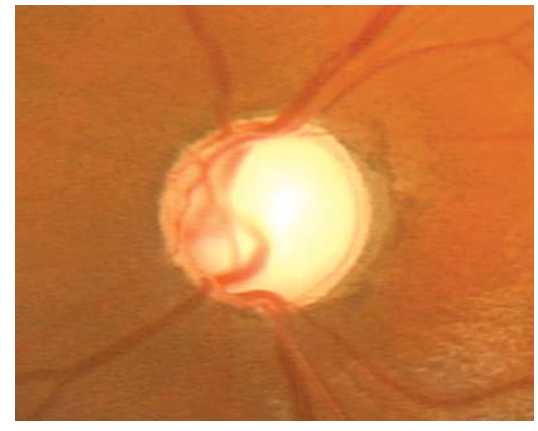


FIGURE 9.6.15: Glaucoma total cupping—bean pot cupping

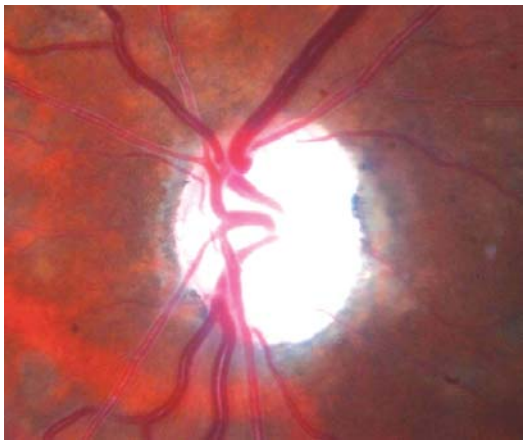


FIGURE 9.6.16: Glaucoma cupping—total pallor

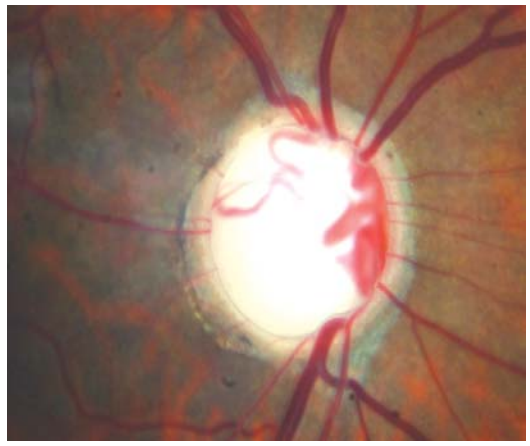


FIGURE 9.6.17: Glaucoma cupping—total pallor

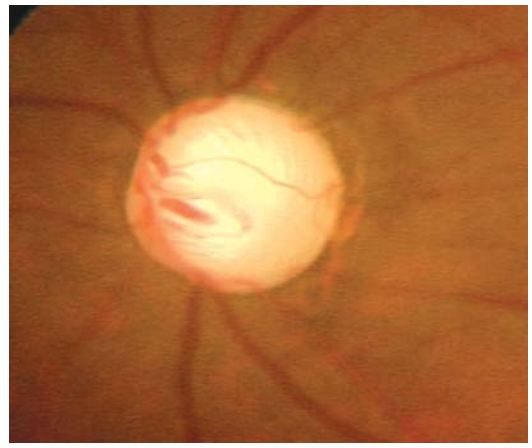


FIGURE 9.6.18: Glaucoma cupping—laminar dot sign

SECONDARY GLAUCOMAS

Glaucoma Capsulare

- Secondary glaucoma with pseudoexfoliation syndrome
- Deposition of fibrillar basement membrane like material blocking the trabecular meshwork
- White exfoliative materials on the anterior lens capsule **(Fig 9.7.1)**, pupillary margin and angle of the anterior chamber **(Fig 9.7.2)**
- *Treatment:* same as POAG

FIGURE 9.7.1: Pseudoexfoliation of lens capsule

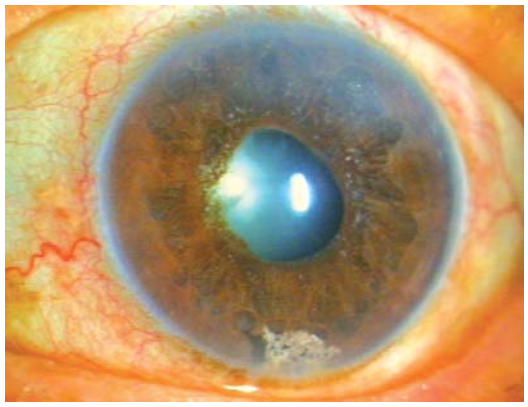


FIGURE 9.7.2: Pseudoexfoliative glaucoma

Phacomorphic Glaucoma

- A swollen intumescent cataract **(Fig 9.8.1)** may cause secondary angle closure glaucoma
- Shallow anterior chamber **(Fig 9.8.2)**
- Picture like angle closure glaucoma
- *Treatment:* urgent reduction of IOP followed by cataract surgery with or without trabeculectomy

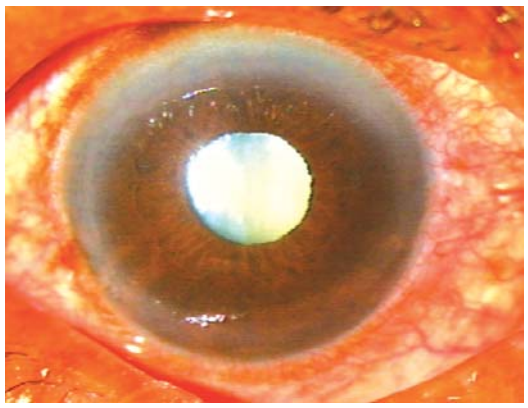


FIGURE 9.8.1: Phacomorphic glaucoma

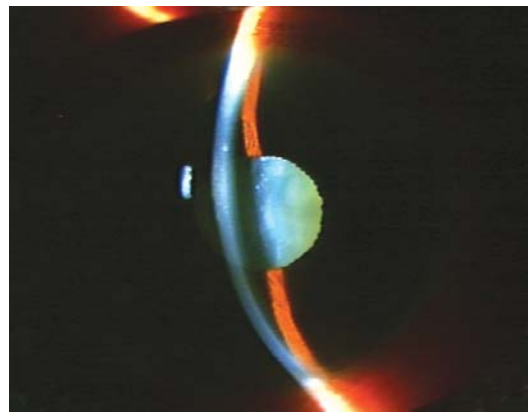


FIGURE 9.8.2: Phacomorphic glaucoma—shallow AC

Phacolytic Glaucoma

- Secondary open angle glaucoma due to micro-leak of lens capsule in a hypermature cataract
- IOP-rise is due to obstruction of trabecular meshwork by macrophages which ingest the lens protein
- Deep or normal anterior chamber with turbid aqueous **(Figs 9.9.1 and 9.9.2)**
- *Treatment:* urgent reduction of IOP medically followed by cataract surgery with or without trabeculectomy

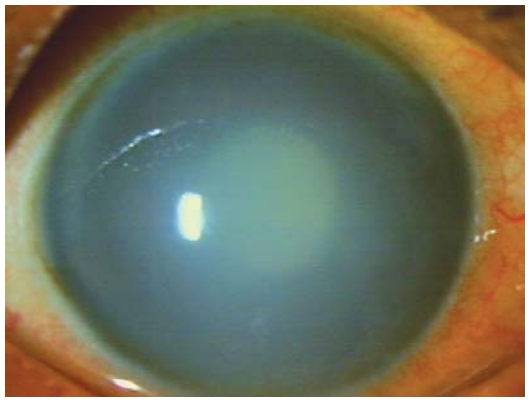


FIGURE 9.9.1: Phacolytic glaucoma—milky cortex in AC

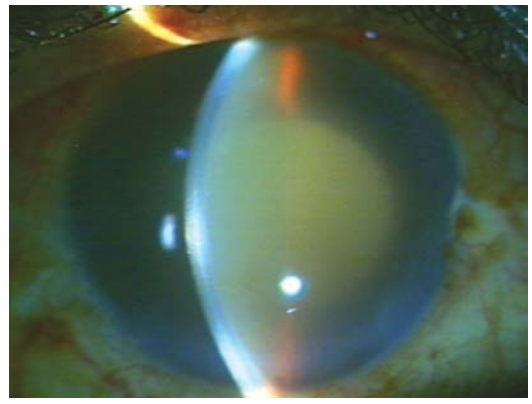


FIGURE 9.9.2: Phacolytic glaucoma—turbid aqueous

Lens-particle Glaucoma

- Typically following ECCE, or after penetrating injury of the lens **(Fig 9.10.1)**
- Due to direct obstruction of trabecular meshwork by lens particles or associated trabeculitis **(Fig 9.10.2)**
- *Treatment*: surgical removal of lens materials

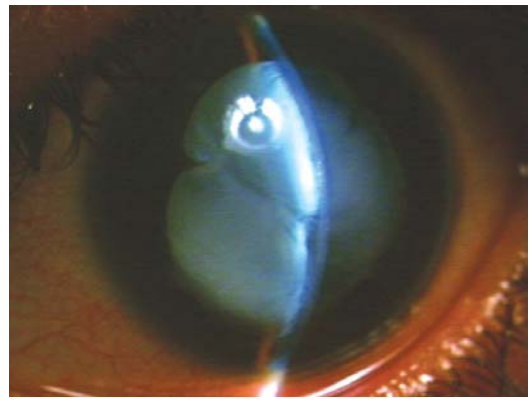


FIGURE 9.10.1: Lens matter glaucoma

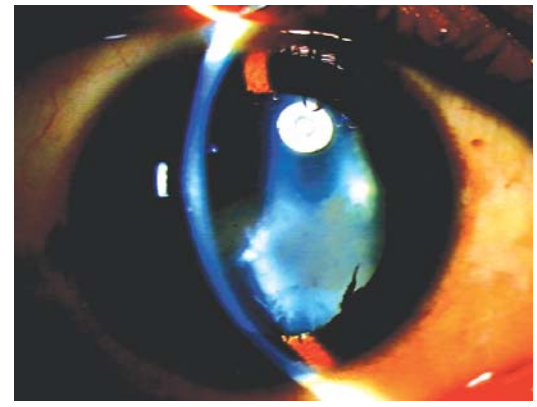


FIGURE 9.10.2: Lens-particle glaucoma

Glaucoma Associated with Ectopia Lentis

- Secondary glaucoma may develop in ectopia lentis or with subluxated or dislocated lens **(Figs 9.11.1 and 9.11.2)**
- Caused by pupillary block or by developing PAS
- *Treatment:* extraction of lens with anterior vitrectomy with peripheral iridectomy or trabeculectomy

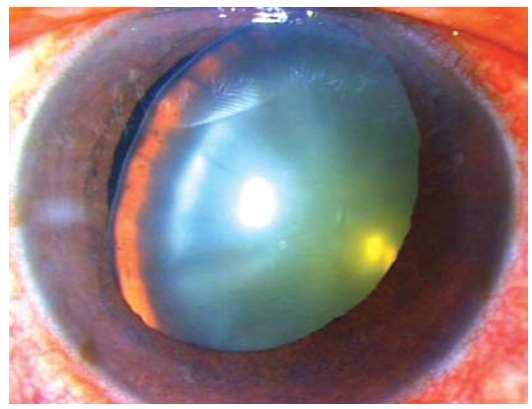


FIGURE 9.11.1: Glaucoma with subluxation

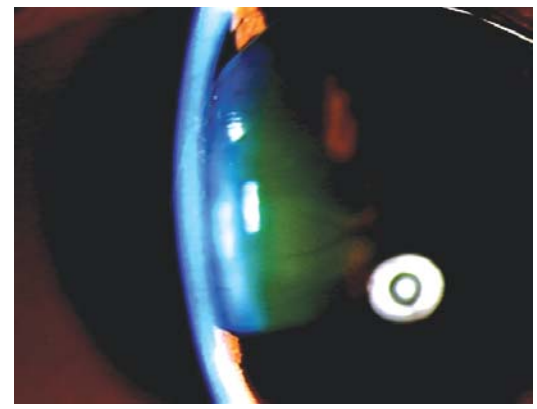


FIGURE 9.11.2: Glaucoma with subluxation—pupillary block

Pigmentary Glaucoma

- Pigment dispersion occurs throughout the anterior segment
- Loss of pigments from the iris—with positive iris transillumination
- Deposition of pigment on the corneal endothelium in a vertical line called *Krukenberg's spindle* **(Fig 9.12.1)**
- Accumulation of pigment along the Schwalbe's line, especially inferiorly, as a dark line—*Sampaolesi's line* **(Fig 9.12.2)**
- *Treatment:* same as POAG

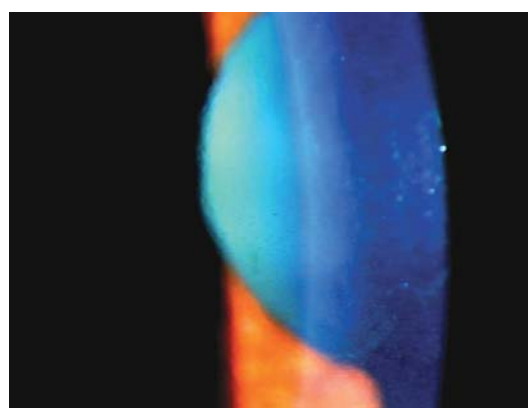


FIGURE 9.12.1: Pigmentary glaucoma—Krukenberg's spindle

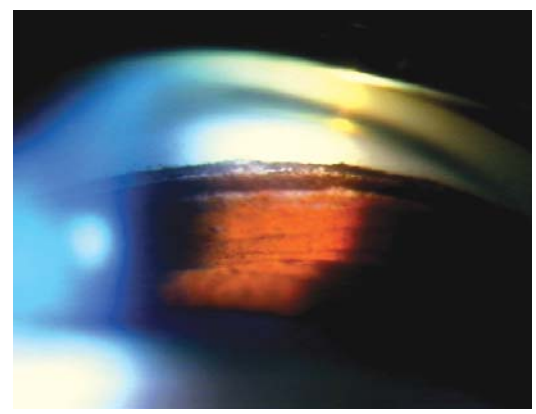


FIGURE 9.12.2: Pigmentary glaucoma—Sampaolesi's line

Inflammatory Secondary Glaucoma

- Associated with inflammation of other structures
- *Causes:*
  - iridocyclitis **(Figs 9.13.1 and 9.13.2)**
  - Posner-Schlossman syndrome (Glauco-mato-cyclitic crisis) **(Fig 9.13.3)**
  - corneal ulcer with hypopyon **(Fig 9.13.4)**
  - adherent leukoma
- *Treatment:* medical and if necessary surgical

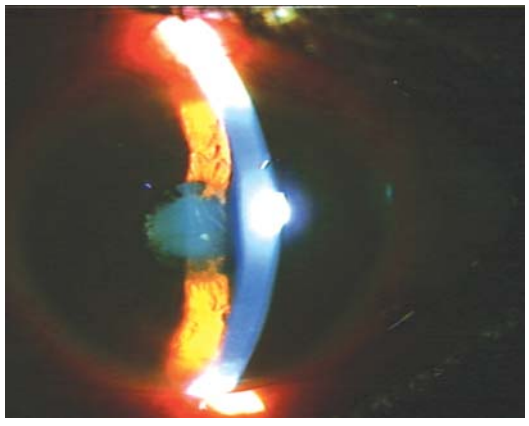


FIGURE 9.13.1: Glaucoma with iridocyclitis—iris bombe

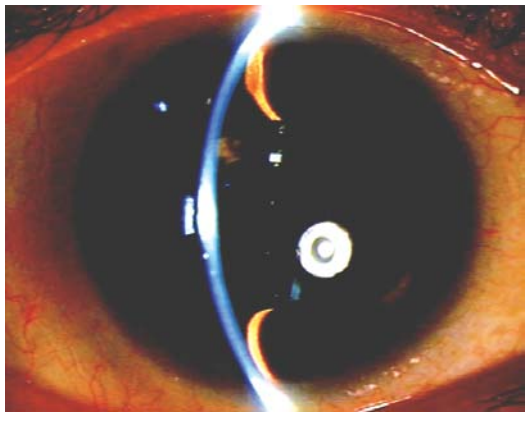


FIGURE 9.13.2: Glaucoma with iridocyclitis—iris bombe

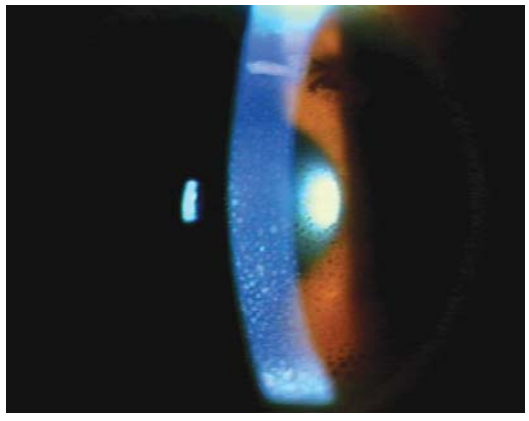


FIGURE 9.13.3: Glaucoma with iridocyclitis—PS syndrome

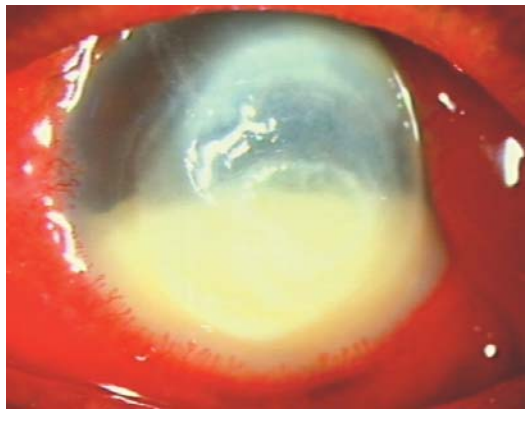


FIGURE 9.13.4: Glaucoma with hypopyon corneal ulcer

Glaucoma Associated with Trauma

- *Blunt injury:* more than one mechanism are involved
  - hyphema **(Fig 9.14.1)**
  - subluxation or dislocation of lens
  - angle recession **(Fig 9.14.2)**
- Penetrating injury
- *Chemical injury:* both by acid or alkali burn
- *Treatment:* antiglaucoma medication, but no miotics

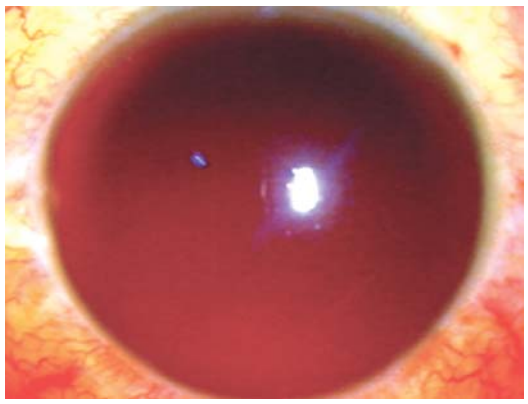


FIGURE 9.14.1: Traumatic hyphema—glaucoma

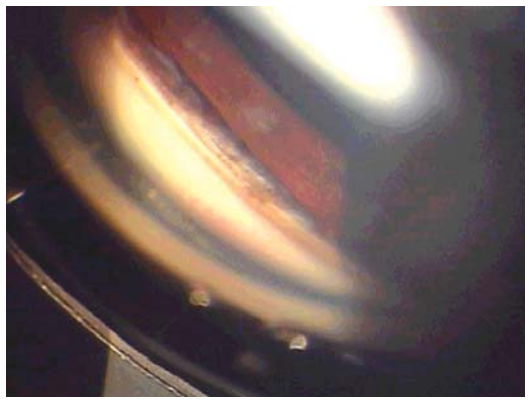


FIGURE 9.14.2: Angle recession glaucoma

Glaucoma following Intraocular Surgery

- *Pseudophakic:* hyphema, iridocyclitis, pupillary block or steroid induced
- *Aphakic:* pupillary block, hyphema, vitreous in AC, iris cyst **(Fig 9.15.1)**, etc.
- *Post keratoplasty:* tight suturing **(Fig 9.15.2)**
- Post vitreoretinal surgery
- *Treatment:* antiglaucoma medication and treatment of the cause

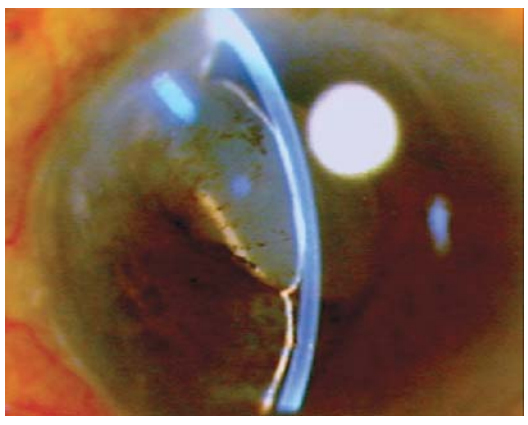


FIGURE 9.15.1: Glaucoma—iris cyst

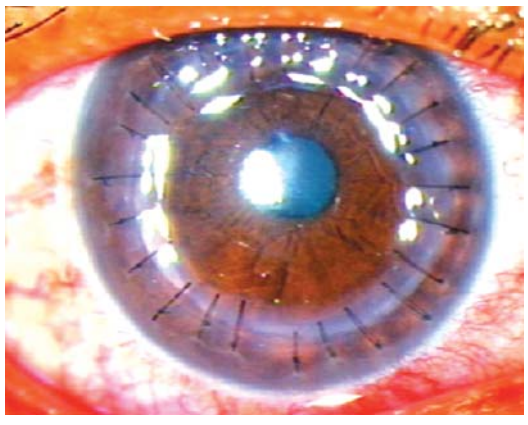


FIGURE 9.15.2: Glaucoma—tight suturing in PK

Malignant (ciliary-block) Glaucoma

- Total shallowing (both central and peripheral) of anterior chamber **(Fig 9.16.1)**
- Poor response to conventional antiglaucoma medication
- *Two types:*
  - cilio-lenticular block: after trabeculectomy
  - cilio-vitreal block: after cataract operation (mainly after ICCE) **(Fig 9.16.2)**
- *Treatment:*
  - USG-localization of vitreous pockets
  - lens extraction in phakic cases
  - YAG laser hyaloidotomy in pseudophakic cases
  - vitrectomy in aphakic cases

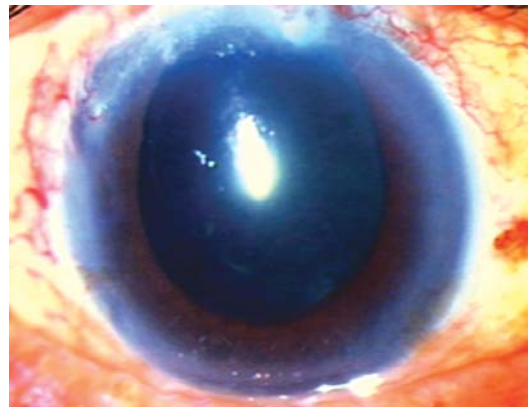


FIGURE 9.16.1: Malignant glaucoma

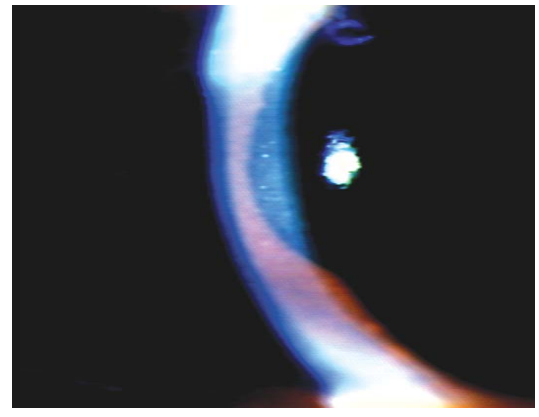


FIGURE 9.16.2: Malignant glaucoma—ciliovitreal block

Neovascular Glaucoma

- Secondary glaucoma due to rubeosis iridis and neovascularization of the angle
- *Three stages:*
  - *preglaucomatous:* rubeosis iridis **(Fig 9.17.1)**
  - *open angle glaucoma:* due to intense neovascularization at the angle
  - *angle closure glaucoma:* due to gonio-synechia and PAS formation **(Fig 9.17.2)**
- *Treatment:* prophylactic panretinal photo-coagulation, anterior retinal cryopexy and cyclophotocoagulation

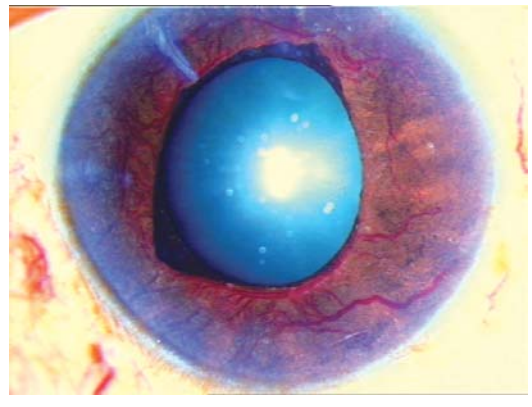


FIGURE 9.17.1: Glaucoma—rubeosis iridis

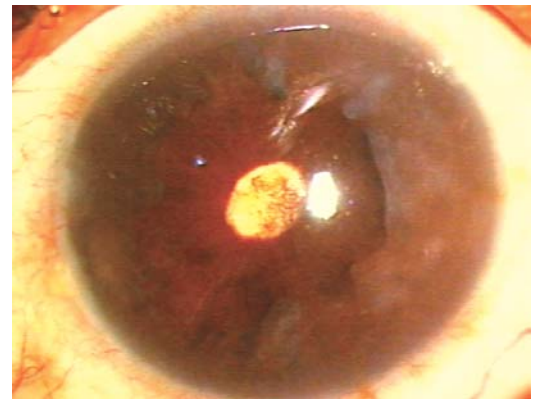


FIGURE 9.17.2: Neovascular glaucoma—total PAS

Glaucoma in Iridocorneal Endothelial Syndrome

- Angle is blocked by Descemet membrane like material
- Peripheral angle synechia leading to secondary angle-closure glaucoma
- *Essential iris atrophy:* glaucoma is more common **(Figs 9.18.1 and 9.18.2)**
- *Cogan Reese syndrome:* glaucoma is common **(Fig 9.18.3)**
- *Chandler's syndrome:* glaucoma is least common **(Fig 9.18.4)**

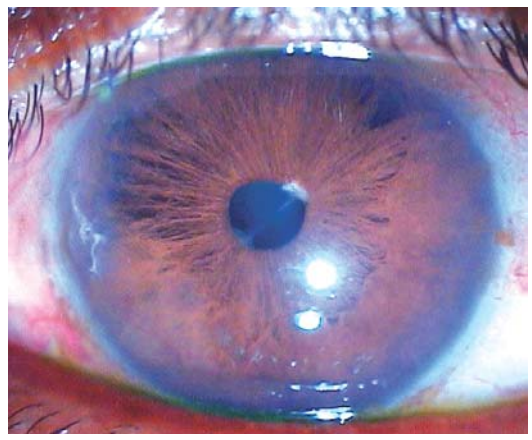


FIGURE 9.18.1: Glaucoma—essential iris atrophy

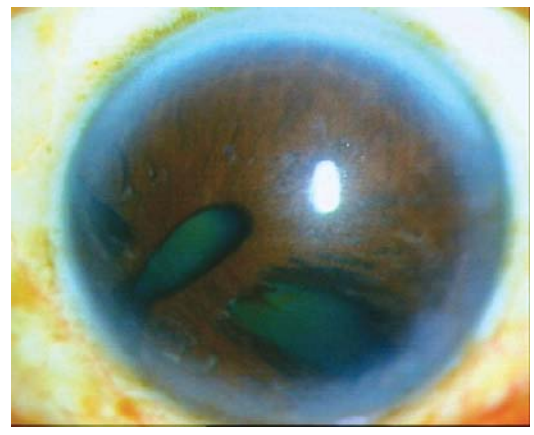


FIGURE 9.18.2: Glaucoma—essential iris atrophy

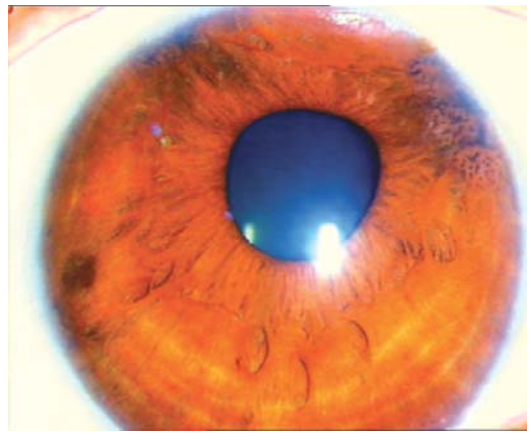


FIGURE 9.18.3: Glaucoma—Cogan Reese syndrome

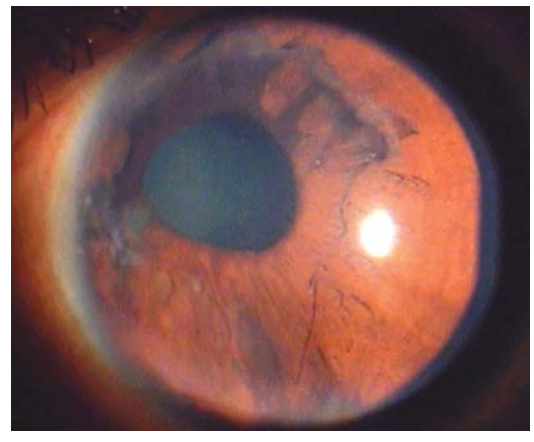


FIGURE 9.18.4: Glaucoma—Chandler syndrome

Other Causes of Secondary Glaucoma

- *Intraocular tumor*: retinoblastoma, malignant melanoma or ciliary body tumor
- Steroid induced glaucoma
- Hemorrhagic glaucoma
- *Raised episcleral venous pressure:* superior venacaval syndrome, orbital varices **(Fig 9.19.1)** or carotico-cavernous thrombosis **(Fig 9.19.2)**
- *Epidemic dropsy glaucoma:* by consuming adulterated mustard oil by *Argemone mexicana*

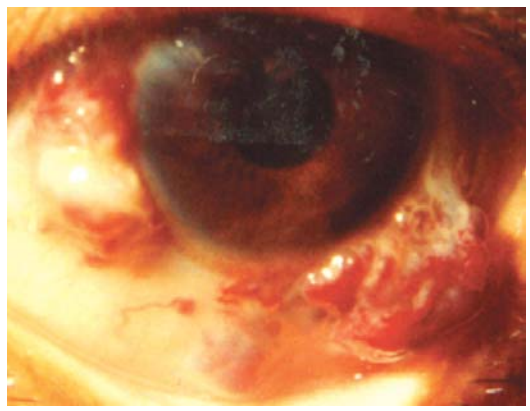


FIGURE 9.19.1: Raised episcleral venous pressure

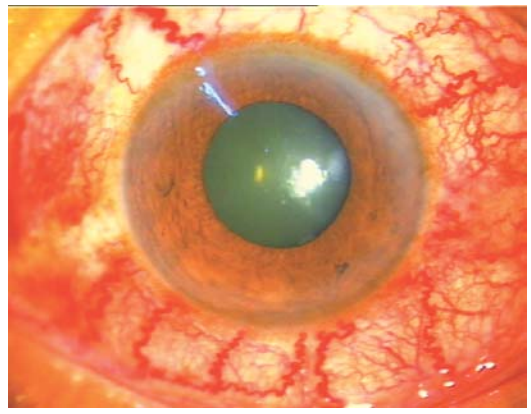


FIGURE 9.19.2: Raised episcleral venous pressure

FILTERING BLEB ABNORMALITIES

Normal functioning bleb (Fig 9.20.1)

Failed filtering bleb (Fig 9.20.2)

Cystic filtering bleb (Fig 9.20.3)

Multilocular filtering bleb (Figs 9.20.4 and 9.20.5)

Overhanging filtering bleb (Figs 9.20.6 and 9.20.7)

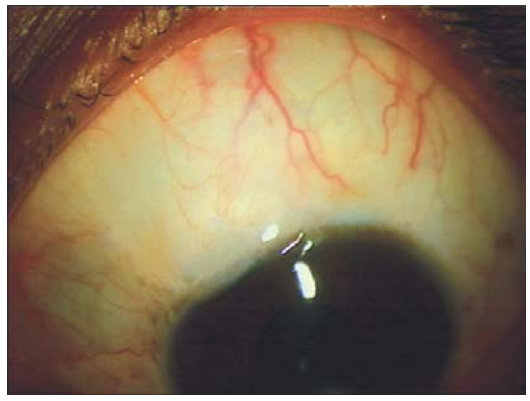


FIGURE 9.20.1: Normal functioning bleb

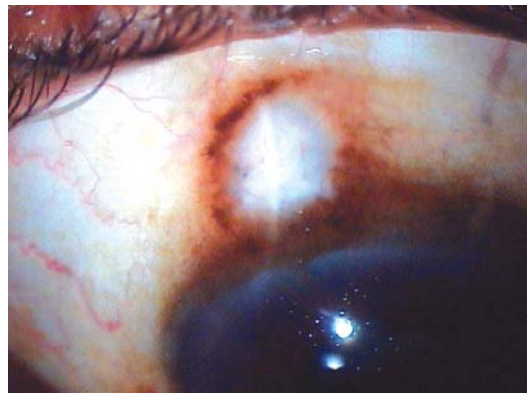


FIGURE 9.20.2: Failed filtering bleb

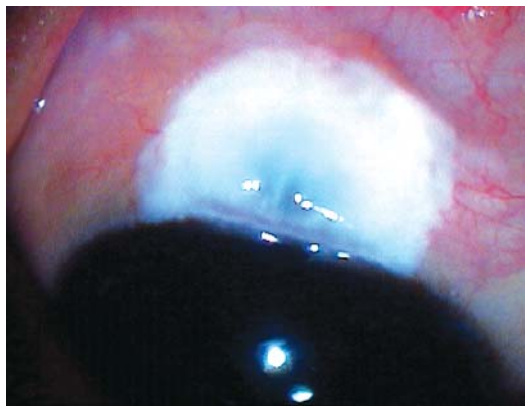


FIGURE 9.20.3: Cystic filtering bleb

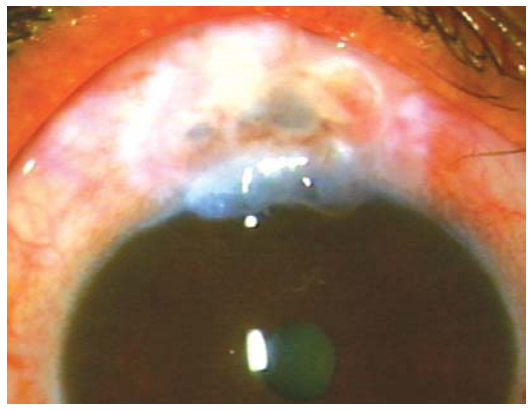


FIGURE 9.20.4: Multilocular filtering bleb

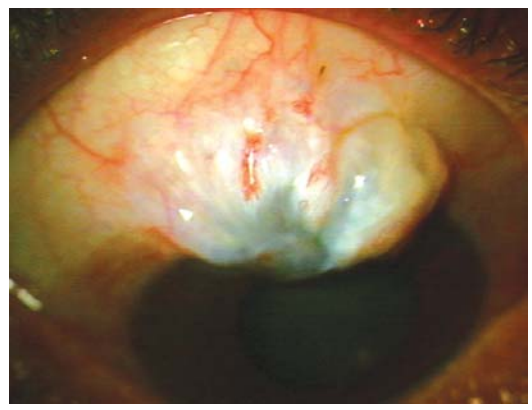


FIGURE 9.20.5: Multilocular filtering bleb

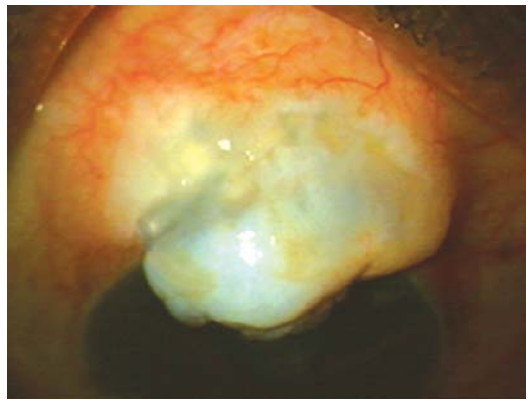


FIGURE 9.20.6: Overhanging filtering bleb

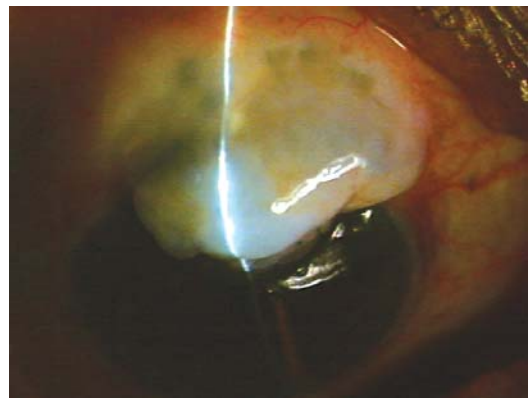


FIGURE 9.20.7: Overhanging filtering bleb

Overfiltering Bleb

- Shallow anterior chamber **(Figs 9.20.8 and 9.20.9)**
- Corneal folds due to hypotony **(Fig 9.20.10)**
- Choroidal folds and hypotonic maculopathy **(Fig 9.20.11)**
- *Treatment:* repair of bleb to reduce overfiltration

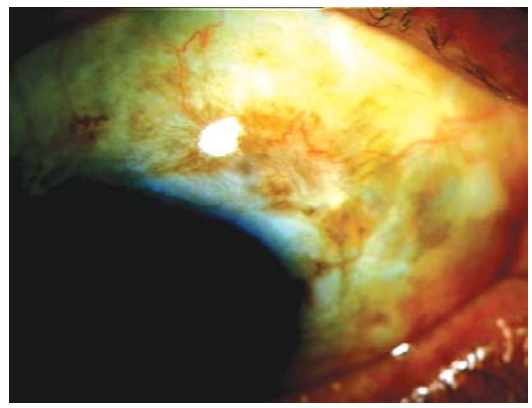


FIGURE 9.20.8: Overfiltering bleb

FIGURE 9.20.9: Overfiltering bleb—shallow AC

FIGURE 9.20.10: Overfiltering bleb—hypotony—corneal folds

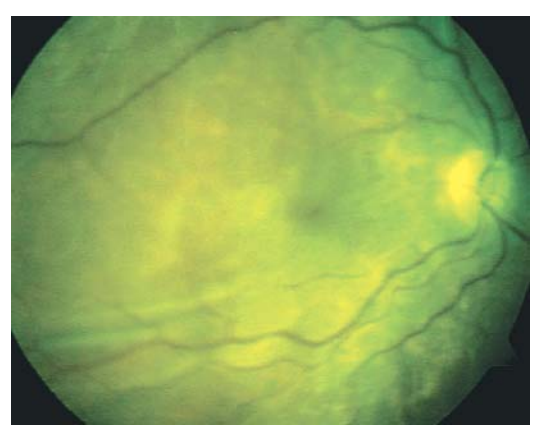


FIGURE 9.20.11: Overfiltering bleb—hypotony—choroidal folds

Leaking Filtering Bleb

- Shallow anterior chamber **(Fig 9.20.12)**
- Corneal and fundal changes may be present
- Positive Seidel's test **(Fig 9.20.13)**
- *Treatment:* repair of bleb

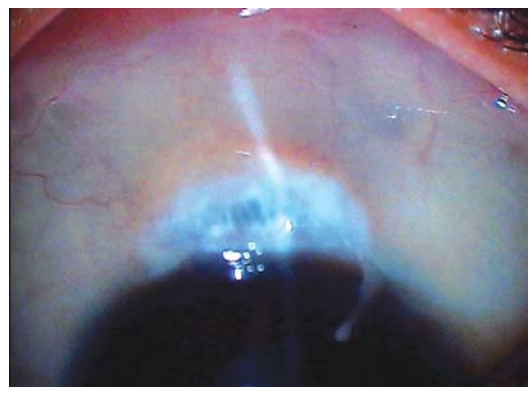


FIGURE 9.20.12: Leaking filtering bleb

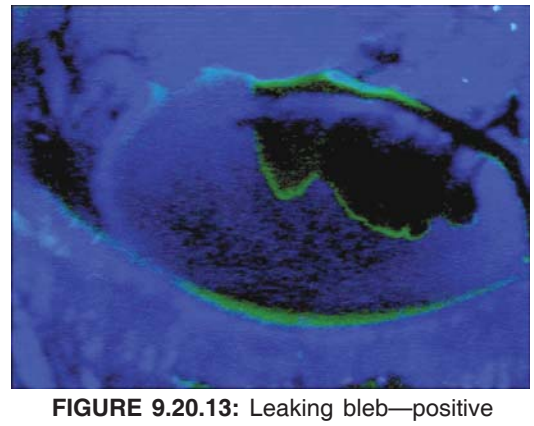


FIGURE 9.20.13: Leaking bleb—positive Seidel's test

Blebitis (bleb infection)

- Suppurative infection of bleb mainly by bacteria
- Bleb appears yellowish white in color with surrounding congestion **(Fig 9.20.14)**
- Anterior chamber reaction and hypopyon **(Fig 9.20.15)**
- *Treatment:* systemic and topical antibiotics with surgical revision of bleb

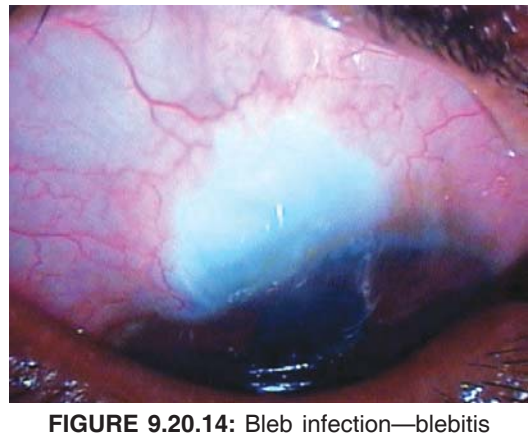


FIGURE 9.20.14: Bleb infection—blebitis

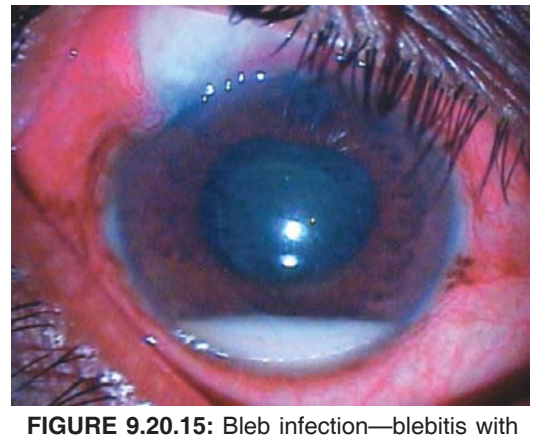


FIGURE 9.20.15: Bleb infection—blebitis with hypopyon

10 Diseases of the Vitreous

VITREOUS OPACITIES

- Muscae volitantes
- Asteroid hyalosis
- Synchisis scintillans
- Persistent hyperplastic primary vitreous
- Vitreous cells
- Vitreous hemorrhage

MISCELLANEOUS VITREOUS OPACITIES

- Pigment cells
- Cotton ball exudates
- Parasite in the vitreous
- Foreign bodies in the vitreous
- Vitreous prolapse

VITREOUS OPACITIES

- Normally, under slit-lamp it appears optically clear
- Many natural or foreign substances may be suspended in the vitreous cavity **(Figs 10.1.1 and 10.1.2))**
- *Endogenous*: e.g. vascular remnants, leukocytes, RBCs, tumor cells, pigments, calcium or cholesterol crystals, etc.
- *Exogenous*: e.g. parasite, foreign particles.

Muscae Volitantes

- Unilateral or bilateral minute fly-like or cobweb opacities best perceived by the patient against white or bright light background
- Represent physiologic remnants of primitive hyaloid vascular system
- Reassurance is the only treatment

Asteroid Hyalosis

- An involutional, usually bilateral condition that affects aged patients
- Appear as numerous, white, round or discoid bodies suspended throughout, or in a portion of the solid vitreous **(Fig 10.2.1)**
- Represent calcium soap crystals from degeneration of vitreous fibrils **(Fig 10.2.2)**
- Symptomless and do not require treatment

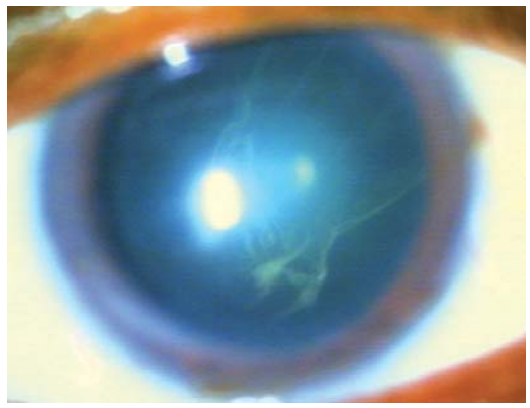


FIGURE 10.1.1: Vitreous opacity

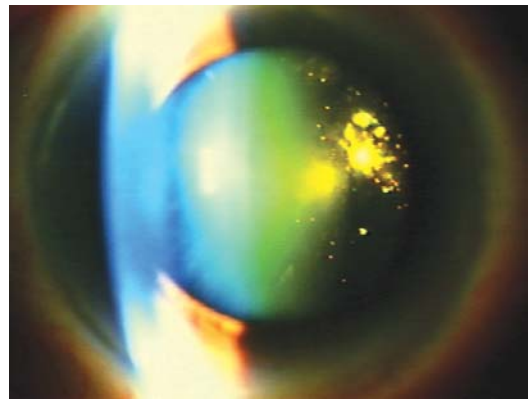


FIGURE 10.1.2: Vitreous opacity

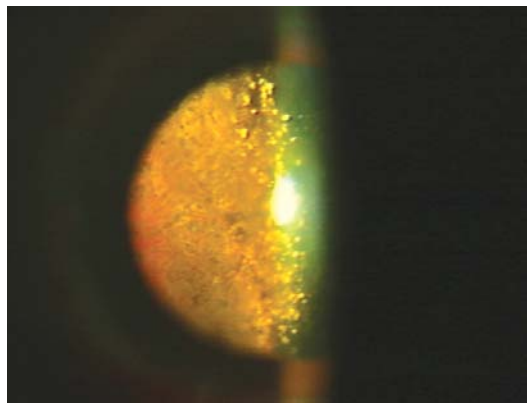


FIGURE 10.2.1: Asteroid hyalosis

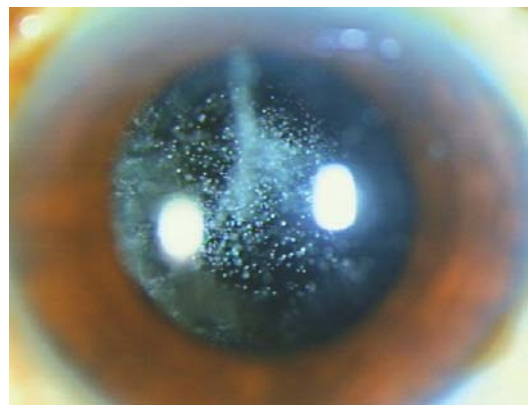


FIGURE 10.2.2: Asteroid hyalosis

Synchisis Scintillans

- Unilateral condition, with previous history of vitreous hemorrhage or inflammation
- Crystals appear as golden or multi-colored glittering particles which settle at the bottom of the vitreous cavity **(Fig 10.3.1)**
- Can be thrown upwards by the ocular movements, to form a 'golden shower' **(Fig 10.3.2)**
- After ICCE, the crystals may be seen in the anterior chamber also **(Fig 10.3.3)**
- No effective treatment

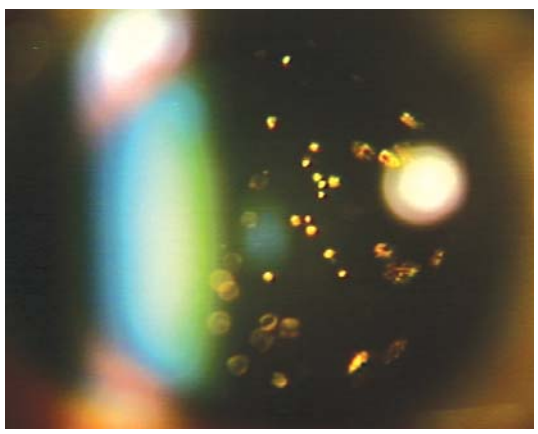


FIGURE 10.3.1: Synchisis scintillans—anterior vitreous

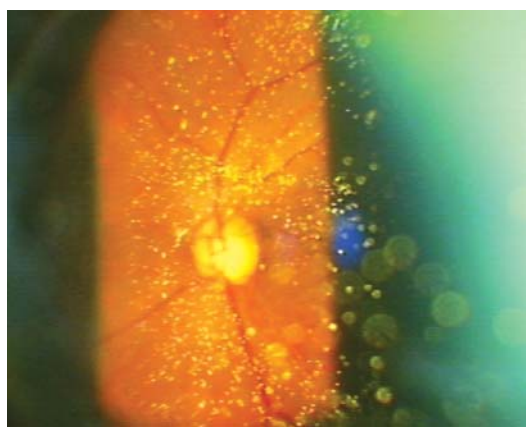


FIGURE 10.3.2: Synchisis scintillans—posterior vitreous

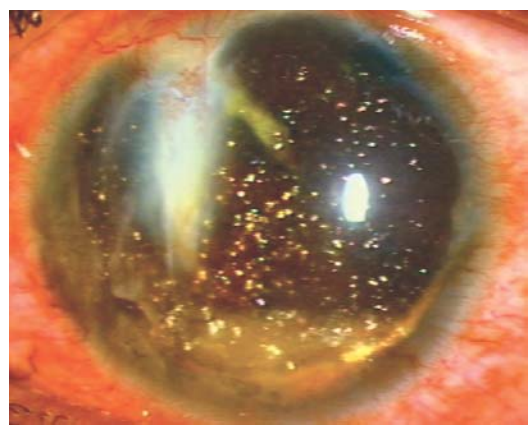


FIGURE 10.3.3: Synchisis scintillans—anterior chamber

Persistent Hyperplastic Primary Vitreous

- Unilateral condition, and the affected eye is smaller than the fellow eye due to faulty development
- White pupillary reflex is noticed in the full-term infant, shortly after birth **(Fig 10.4.1)**
- Under slit-lamp, a yellowish-white mass may be visible in the anterior vitreous **(Fig 10.4.2)**
- Associated microphthalmos, cataract, or long ciliary processes
- Must be differentiated from other causes of white pupillary reflex ***(See Chapter: 7)***
- *Treatment:* lensectomy and vitrectomy as early as possible

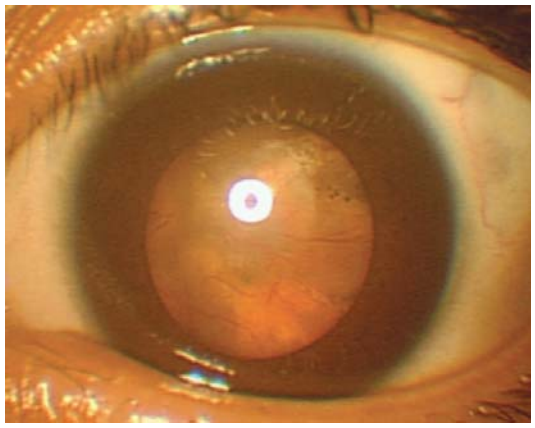


FIGURE 10.4.1: Persistent hyperplastic primary vitreous

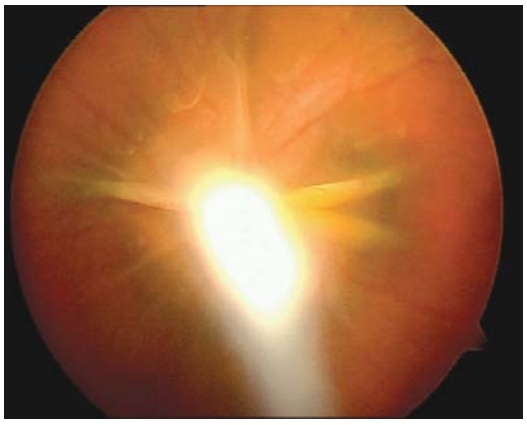


FIGURE 10.4.2: Persistent hyperplastic primary vitreous

Vitreous Cells

- *Iridocyclitis*: retrolental cells along with aqueous cells, especially when cyclitis is predominant **(Figs 10.5.1 and 10.5.2)**
- *Intermediate uveitis*: different degree of vitreous cells and inferior peripheral vitreous snow-banking
- *Infective endophthalmitis*: cells or frank exudates in the vitreous. May be acute in case of penetrating injury **(Fig 10.5.3)** and post-surgery **(Figs 10.5.4 and 10.5.5)**
- *Posterior uveitis*: cells are most dense adjacent to the lesions

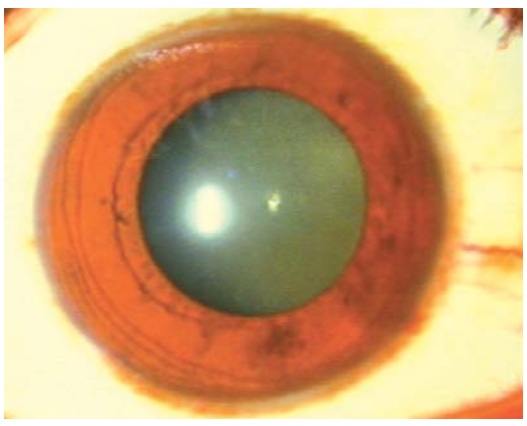


FIGURE 10.5.1: Vitreous cells in iridocyclitis

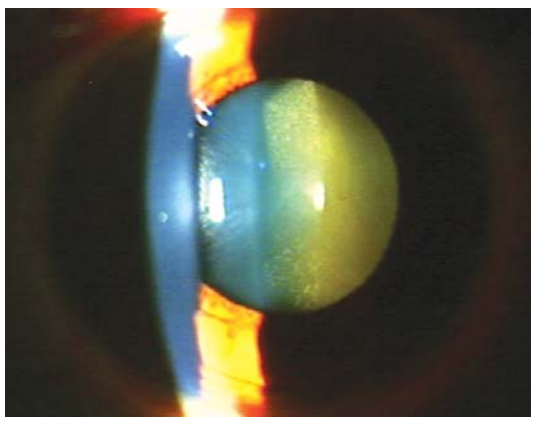


FIGURE 10.5.2: Vitreous cells in iridocyclitis

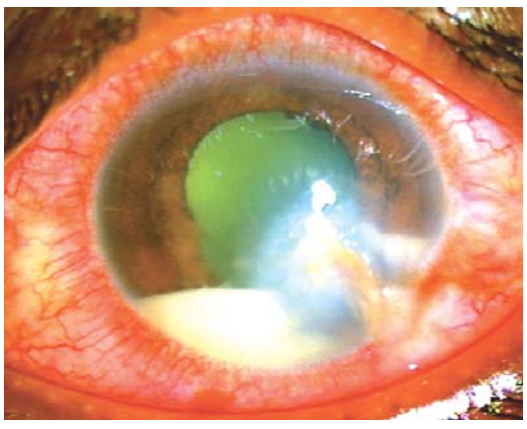


FIGURE 10.5.3: Endophthalmitis—penetrating injury

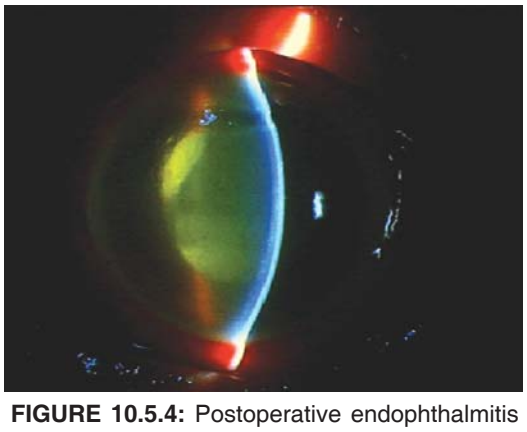


FIGURE 10.5.4: Postoperative endophthalmitis

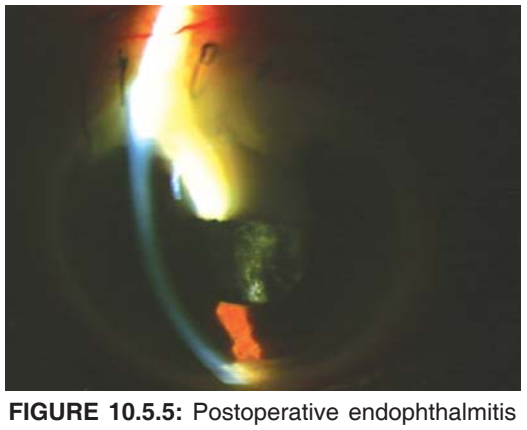


FIGURE 10.5.5: Postoperative endophthalmitis

Vitreous Hemorrhage

- May occur as a preretinal or an intravitreal phenomenon **(Figs 10.6.1 and 10.6.2)**
  - *retinal break*: Traumatic or by vitreous traction
  - *proliferative retinopathies*: rupture of newly formed blood vessels **(Fig 10.6.3)**
  - acute posterior vitreous detachment (PVD)
  - bleeding disorders
  - periphlebitis or Eales' disease **(Fig 10.6.4)**
  - avulsion of the optic nerve
- May be seen as minute red colored or dark spots, as red mass, or with no fundal view
- Organized vitreous hemorrhage may appear clotted with membrane formation **(Figs 10.6.5 and 10.6.6)**
- *Treatment:* investigation, medical and /or pars plana vitrectomy with endo-laser photocoagulation

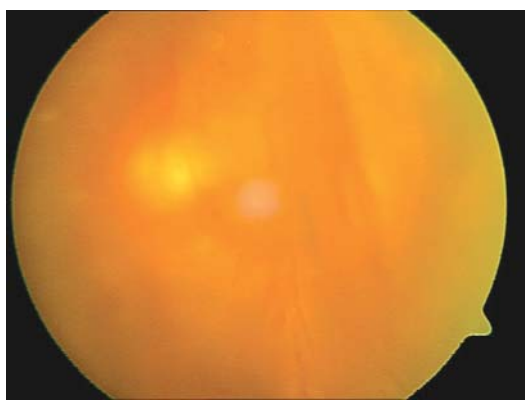


FIGURE 10.6.1: Vitreous hemorrhage

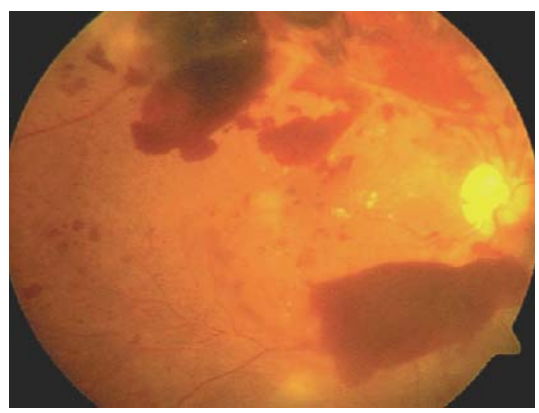


FIGURE 10.6.2: Vitreous hemorrhage—PDR

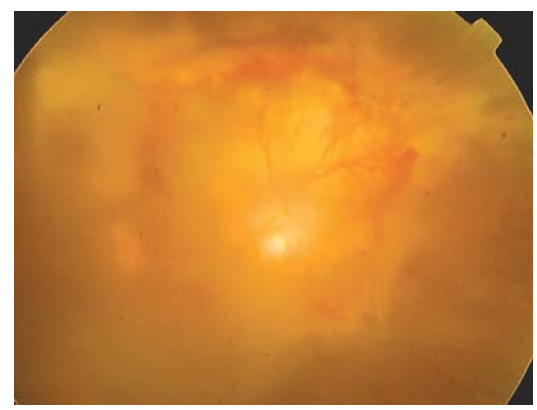


FIGURE 10.6.3: Vitreous hemorrhage—NVE

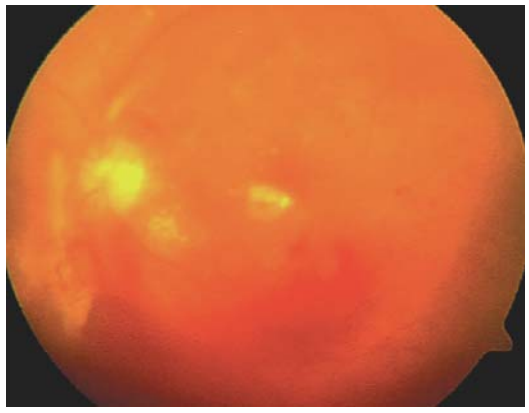


FIGURE 10.6.4: Vitreous hemorrhage—Eales' disease

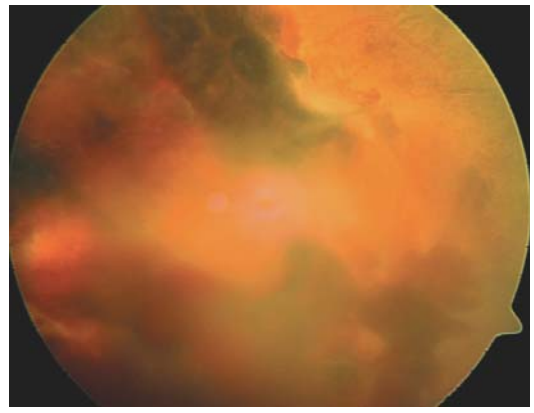


FIGURE 10.6.5: Organized vitreous hemorrhage

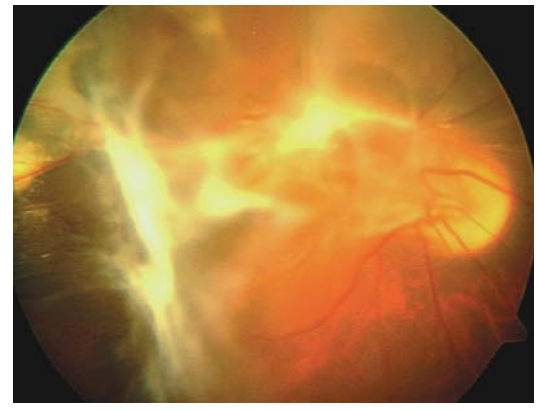


FIGURE 10.6.6: Vitreous hemorrhage—membrane formation

MISCELLANEOUS VITREOUS OPACITIES

Pigment Cells ('tobacco dust')

- Consist of macrophages containing retinal pigment epithelial cells
- Mainly visible in the anterior vitreous **(Figs 10.7.1 and 10.7.2)**
- *Causes:* retinal tears with PVD, rhegmatogenous retinal detachment, trauma, excessive retinal cryopexy

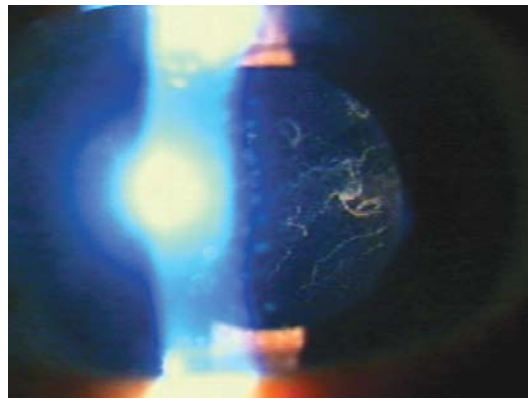


FIGURE 10.7.1: Vitreous membrane with tobacco dust

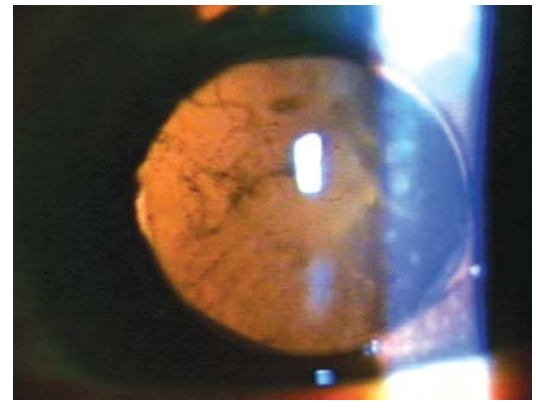


FIGURE 10.7.2: Vitreous membrane with tobacco dust

Cotton Ball Exudates

- Seen in intermediate uveitis
- Sarcoidosis
- Candidiasis **(Figs 10.8.1 and 10.8.2) (See Figs 12.30.2 and 12.30.3)**

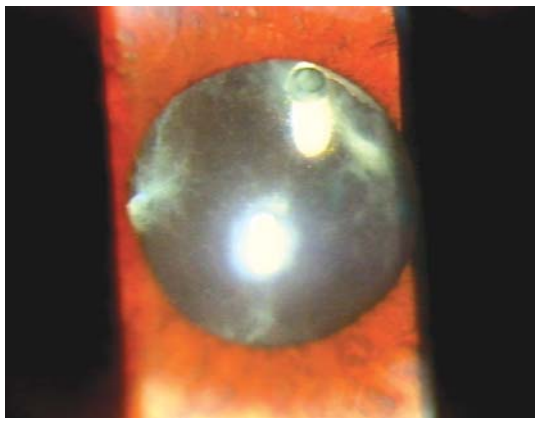


FIGURE 10.8.1: Fungal exudates in endophthalmitis

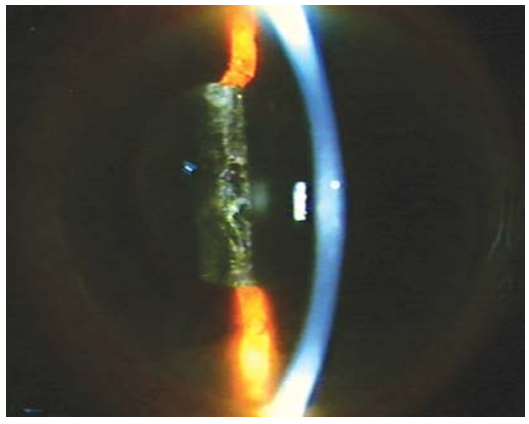


FIGURE 10.8.2: Fungal exudates in endophthalmitis

Parasite in the Vitreous

- May be live or dead parasite
  - Cysticercosis **(Figs 10.9.1 and 10.9.2)**
  - Gnathostomiasis **(Figs 10.9.3 and 10.9.4)**

FIGURE 10.9.1: Cysticercus in the vitreous

FIGURE 10.9.2: Cysticercus in the vitreous

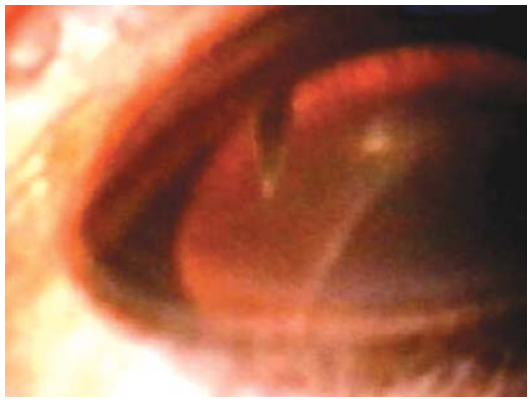


FIGURE 10.9.3: Gnathostoma in the vitreous

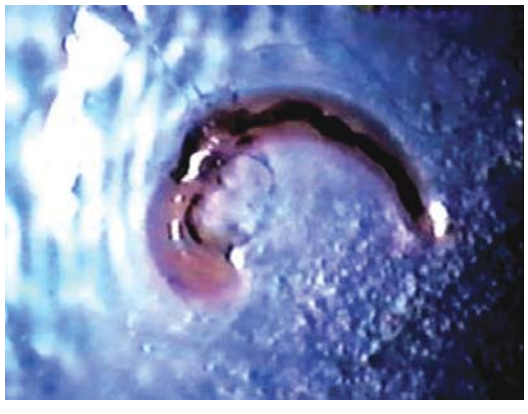


FIGURE 10.9.4: Gnathostoma in the vitreous

Foreign Bodies in the Vitreous

- Metallic **(Fig 10.10.1)**
- Intravitreal triamcinolone injection **(Fig 10.10.2)**
- Silicone oil, may be emulsified **(Fig. 10.10.3)**
- Air bubble and other gasses **(Fig 10.10.4)**

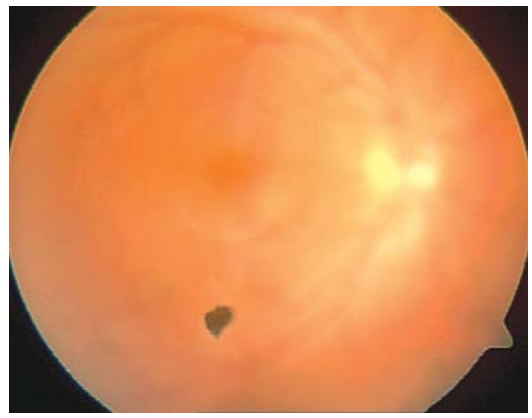


FIGURE 10.10.1: Metallic foreign body in the vitreous

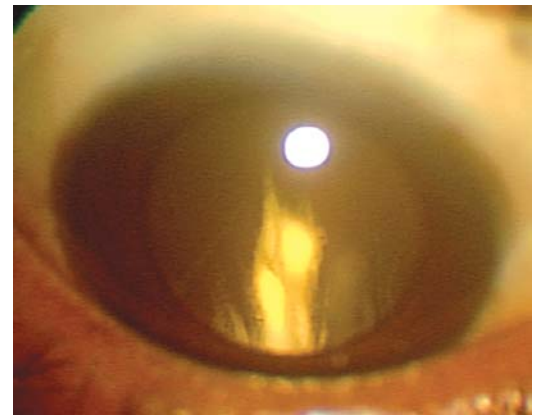


FIGURE 10.10.2: Intravitreal triamcinolone

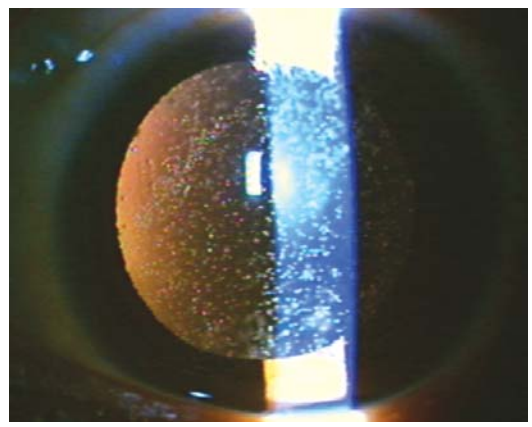


FIGURE 10.10.3: Vitreous opacities—emulsified silicone oil

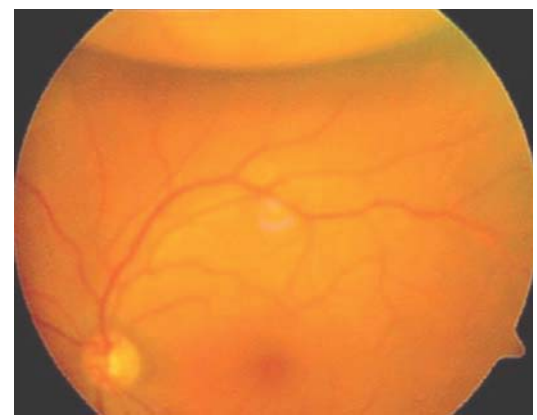


FIGURE 10.10.4: Gas bubble in the vitreous

Vitreous Prolapse

- After rupture of the posterior lens capsule (in ECCE), the anterior vitreous may herniate into the anterior chamber to fill it completely **(Fig 10.11.1)**
- Vitreous also herniates into anterior chamber in subluxation or dislocation of lens **(Figs 10.11.2 and 10.11.3)**
- Direct contact of the vitreous with other structures, such as the cornea or the angle **(Fig 10.11.4)**
- Incarceration of the vitreous into the operative wound
- *Treatment:* anterior vitrectomy along with other main procedures

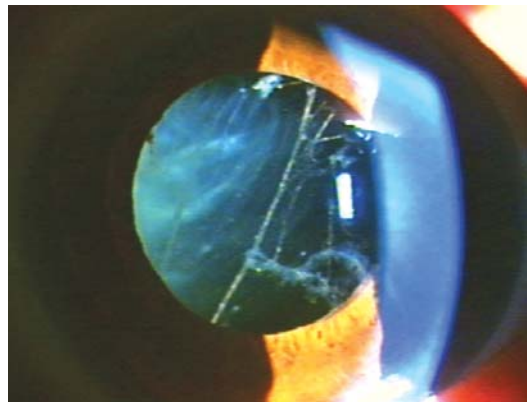


FIGURE 10.11.1: Vitreous prolapse in AC—aphakia

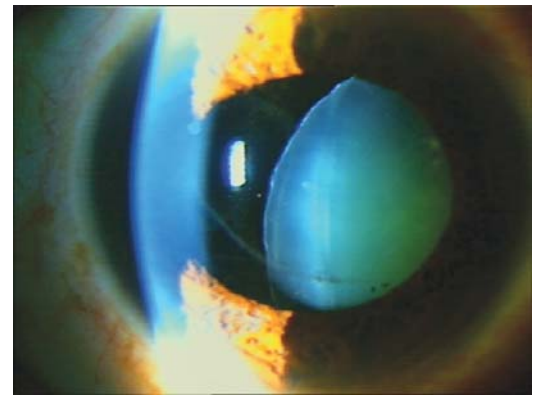


FIGURE 10.11.2: Vitreous prolapse in AC—subluxated lens

FIGURE 10.11.3: Vitreous prolapse in AC—dislocated lens

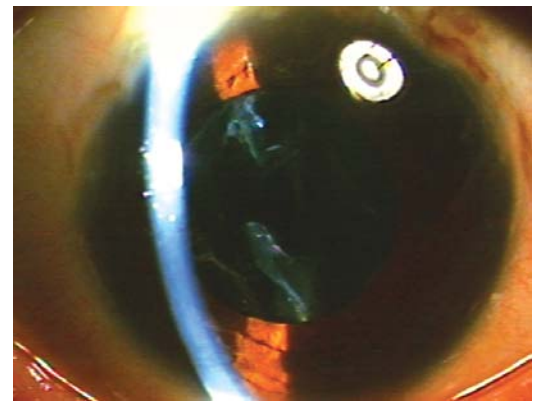


FIGURE 10.11.4: Vitreous prolapse in AC—after PC rent

11 Diseases of the Optic Nerve

CONGENITAL ABNORMALITIES OF OPTIC NERVE

- Myelinated nerve fibers
- Persistent hyaloid artery
- Drusen
- Conus
- Optic disk pit
- Coloboma of the optic disk
- 'Morning glory' syndrome
- Small disk: high hypermetropia
- Hypoplastic optic disk
- Tilted disk
- Large disk: myopia
- Large disk (megalopapilla)
- Disk with large cup
- Glaucomatous optic disk

VASCULAR ANOMALIES

- Disk collaterals
- New vessels on the disk
- Optico-ciliary shunts
- Disk hemorrhage
- Dragged vessels

OPTIC NEURITIS

- Papillitis
- Retrobulbar neuritis
- Neuroretinitis
- Anterior ischemic optic neuropathy

OTHER CAUSES OF UNILATERAL DISK EDEMA

- Central retinal venous occlusion
- Central vasculitis (papillophlebitis)
- Long-standing hypotony
- Posterior scleritis

PAPILLEDEMA

- Early papilledema
- Established (acute) papilledema
- Chronic papilledema
- Atrophic papilledema

OTHER CAUSES OF BILATERAL DISK EDEMA

- Malignant hypertension

OPTIC ATROPHY

- Primary optic atrophy
- Secondary optic atrophy
- Consecutive optic atrophy
- Glaucomatous optic atrophy
- Temporal pallor

PRIMARY OPTIC DISK TUMORS

- Optic disk capillary hemangioma
- Optic disk cavernous hemangioma
- Optic disk melanocytoma
- Astrocytoma
- Optic nerve glioma and meningioma

INFILTRATIVE LESIONS

CONGENITAL ABNORMALITIES OF OPTIC NERVE

Myelinated Nerve Fibers

- Myelin sheaths of the optic nerve fibers cease normally at the lamina cribrosa
- Congenital condition, present 1 percent of normal population
- Bilateral in 20 percent cases
- Appear as white patches with radial striations (feathery) at peripheral edges **(Fig 11.1.1)**
- Usually peripapillary or sometimes peripheral and isolated **(Figs 11.1.2 to 11.1.4)**
- Myelin sheaths disappear in optic atrophy
- *No treatment* is required

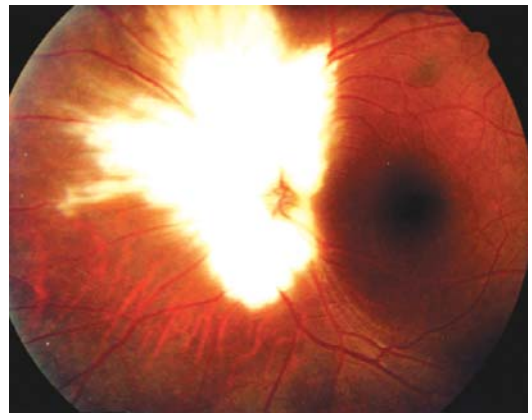


FIGURE 11.1.1: Myelinated nerve fibers

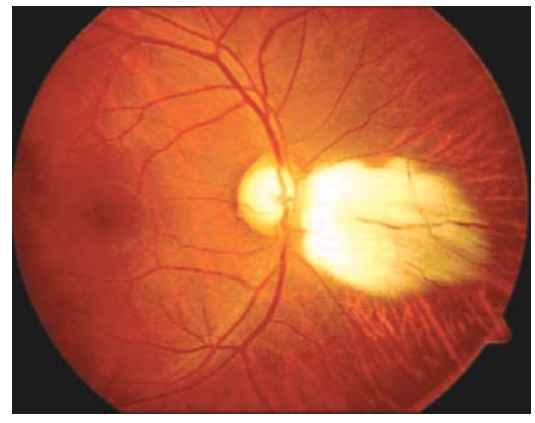


FIGURE 11.1.2: Myelinated nerve fibers

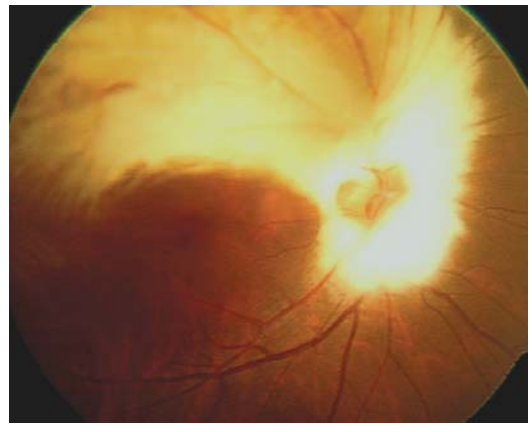


FIGURE 11.1.3: Myelinated nerve fibers

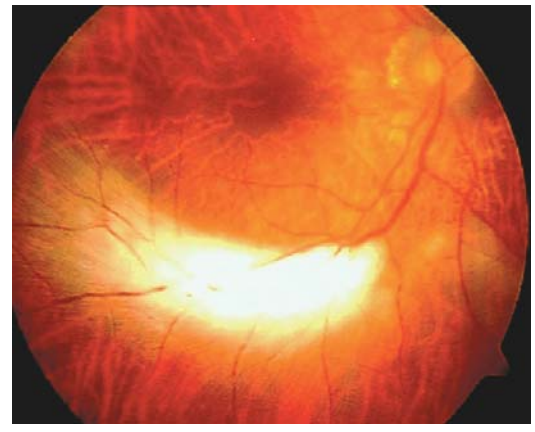


FIGURE 11.1.4: Myelinated nerve fibers—peripheral

Persistent Hyaloid Artery

- A short stub of this vessel projects into the vitreous cavity from the center of the optic disk and is surrounded by a small mass of glial tissue—called Bergmeister's papilla **(Fig 11.2.1)**
- May project into the vitreous cavity **(Figs 11.2.2, and 11.2.3)**
- Sometimes, it attaches to the posterior capsule of the lens (Mittendorf's dot)
- Failure of the hyaloid artery of regress cause persistent primary vitreous, which may proliferate to PHPV as a retrolental white mass **(Fig 7.9.11)**
- *Treatment* is required in extreme cases

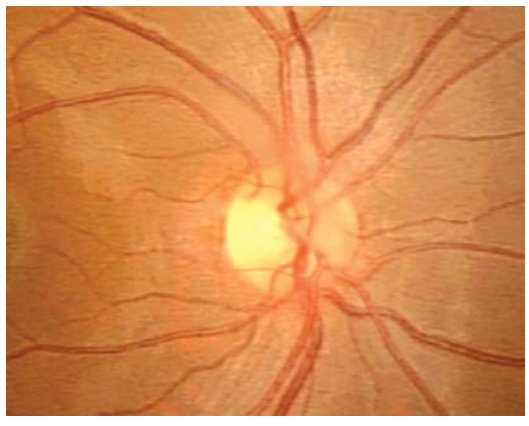


FIGURE 11.2.1: Bergmeister's papilla

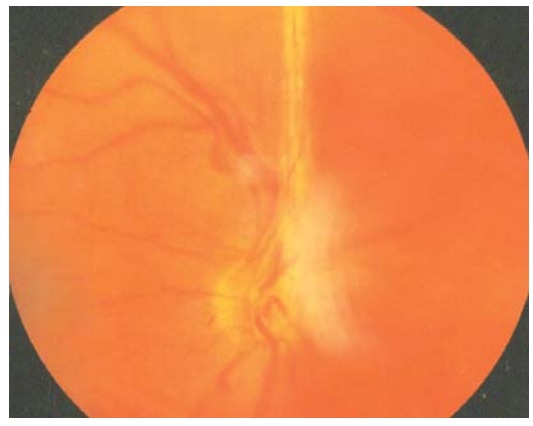


FIGURE 11.2.2: Persistent hyperplastic primary vitreous

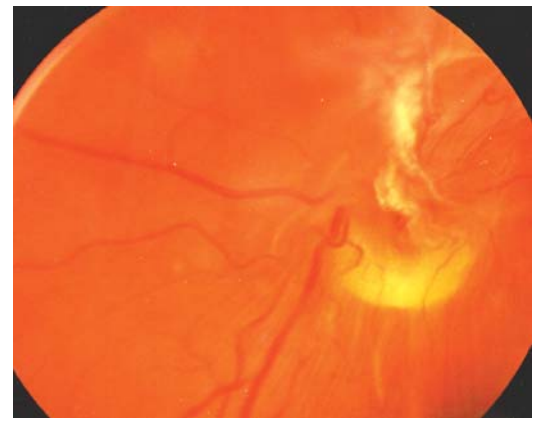


FIGURE 11.2.3: Persistent hyperplastic primary vitreous

Drusen

- *Common drusens*—are laminated, nodular, and may give the disk margins a blurred appearance (pseudo-papilloedema) **(Figs 11.3.1 and 11.3.2)**
- Rarely causes visual disturbances
- Second type is *giant drusens*, which are astrocytic hamartomas that occur in patient with tuberous sclerosis **(Fig 11.34.2)**

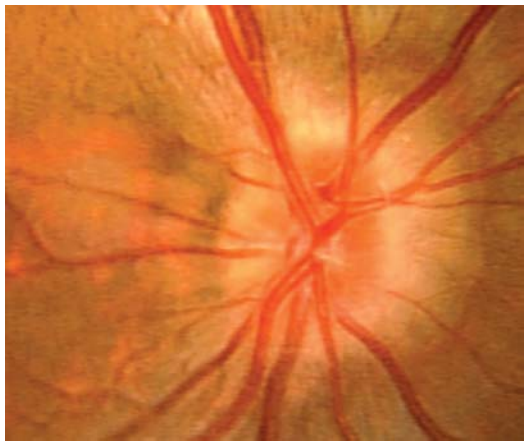


FIGURE 11.3.1: Optic nerve drusen

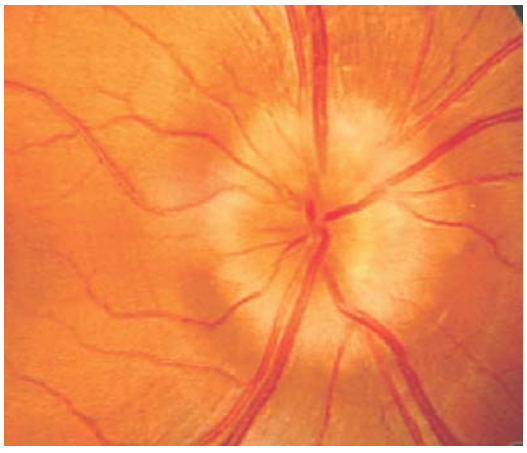


FIGURE 11.3.2: Optic disk drusen

Conus

- Congenital optic disk cresent
- A large, white, semilunar area of sclera, seen adjacent to the disk, in the region of primitive retinal fissure (infero-nasal) **(Figs 11.4.1 to 11.4.3)**
- Often associated with visual field defect
- A myopic crescent has a similar appearance, and is located at the temporal side of the disk **(Fig 11.4.4)**

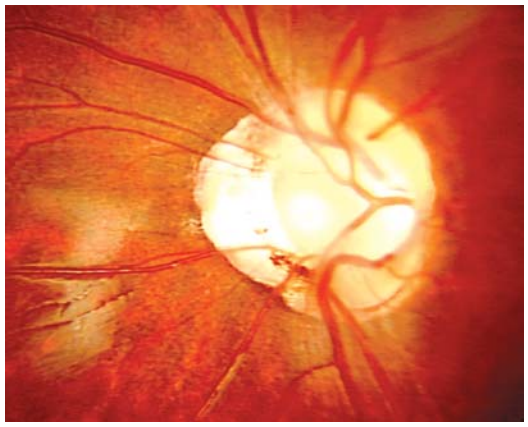


FIGURE 11.4.1: Optic disk conus

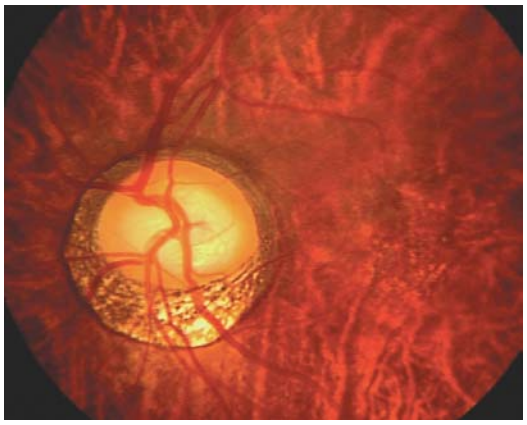


FIGURE 11.4.2: Optic disk conus

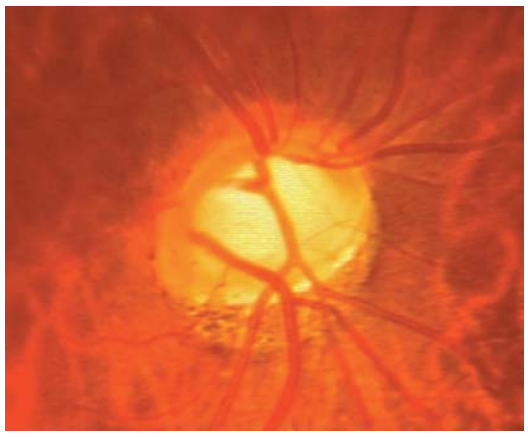


FIGURE 11.4.3: Optic disk conus

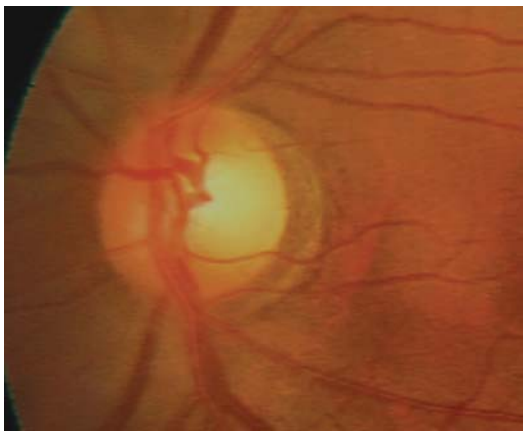


FIGURE 11.4.4: Myopic temporal crescent

Optic Disk Pit

- Isolated congenital unilateral condition
- Round or oval depression, most frequently involve the temporal margin **(Figs 11.5.1 and 11.5.2)**
- A central pit is less common

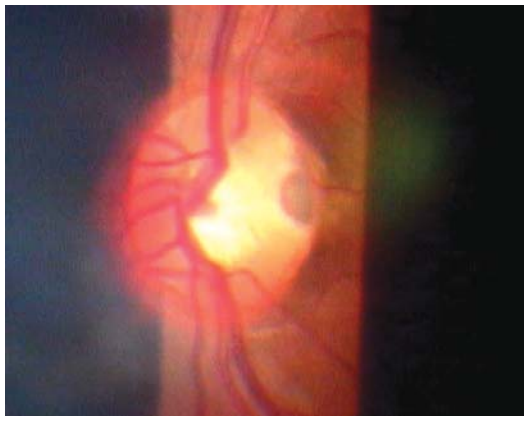


FIGURE 11.5.1: Congenital optic disk pit

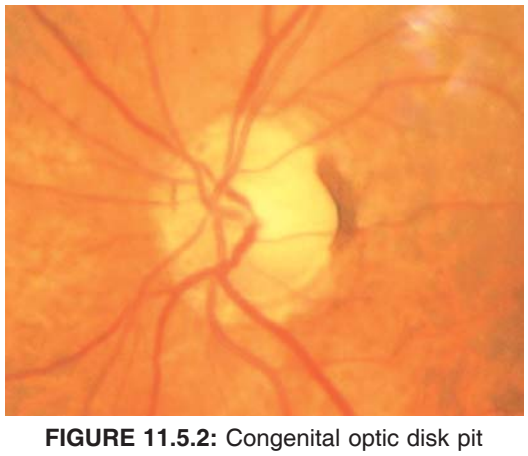


FIGURE 11.5.2: Congenital optic disk pit

Coloboma of the Optic Disk

- Rare, is usually unilateral, but bilateral optic disk colobomata occur as an autosomal dominant hereditary defect **(Figs 11.6.1 and 11.6.2)**
- Large disk with inferior excavation with glistening appearance of underlying sclera **(Figs 11.6.3 to 11.6.5)**
- May be associated with typical coloboma of other parts **(Fig 11.6.6)**
- Juxtapapillary coloboma may appear as '*double optic disk*' or with isolated colobomas as '*triple optic disk*' **(Figs 11.6.7 and 11.6.8)**

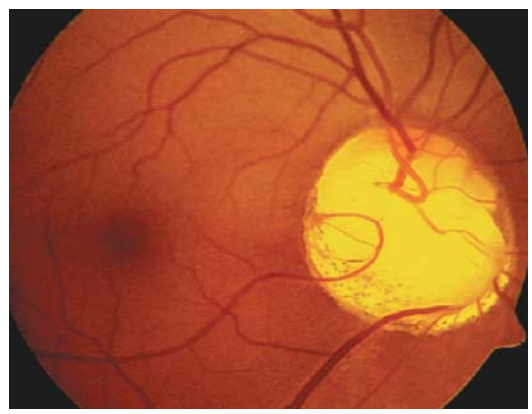


FIGURE 11.6.1: Optic disk coloboma

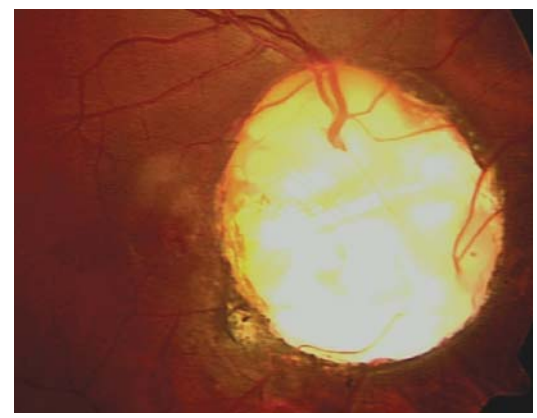


FIGURE 11.6.2: Optic disk coloboma

FIGURE 11.6.3: Optic disk coloboma

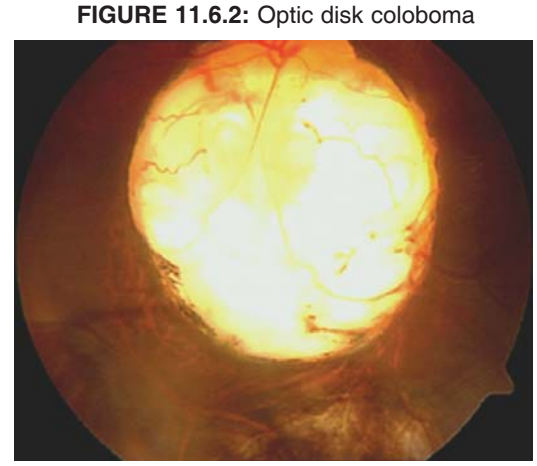


FIGURE 11.6.4: Optic disk coloboma

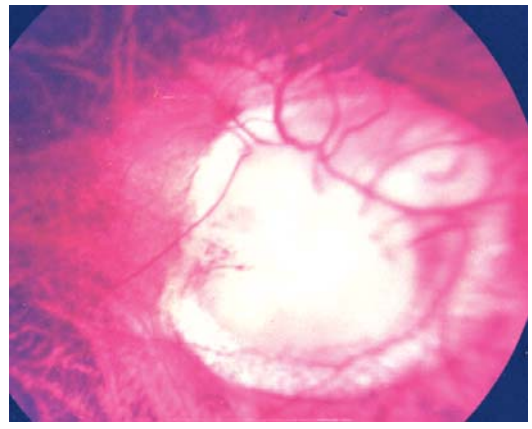


FIGURE 11.6.5: Optic disk coloboma

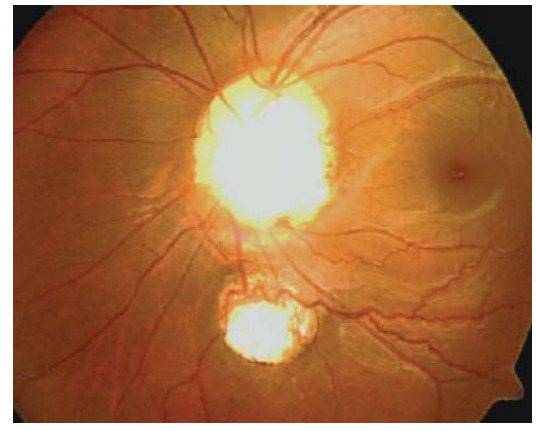


FIGURE 11.6.6: Optic disk coloboma with isolated retinochoroidal coloboma

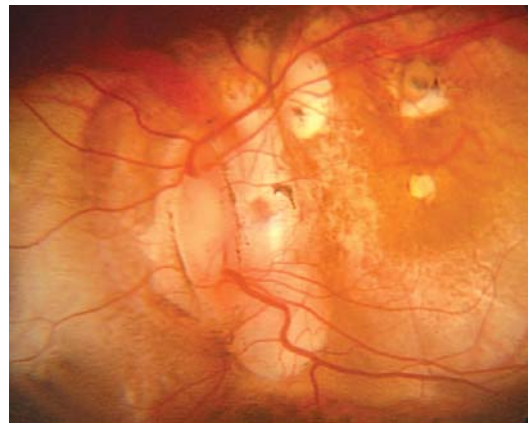


FIGURE 11.6.7: Juxtapapillary coloboma—'double disk' appearence

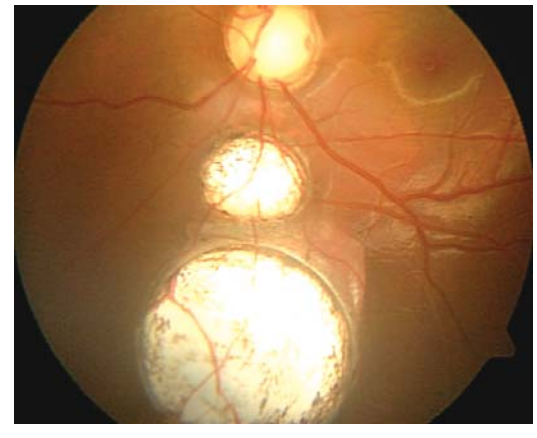


FIGURE 11.6.8: Isolated colobomas—'triple disk' appearance

'Morning Glory' Syndrome

- Usually unilateral, congenital and with poor visual acuity
- Disk looks large with core of white tissue **(Fig 11.7.1)**
- Vessels have an abnormal distribution emerging only around the edges in a spoke-like fashion **(Fig 11.7.2)**
- Surrounded by an annular choroidal ring **(Fig 11.7.3)**
- May be associated with other ocular abnormalities

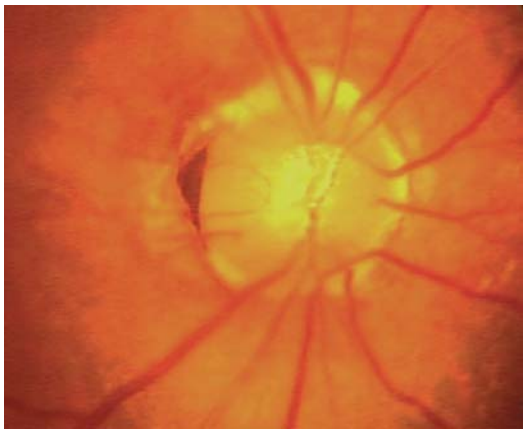


FIGURE 11.7.1: Morning glory syndrome

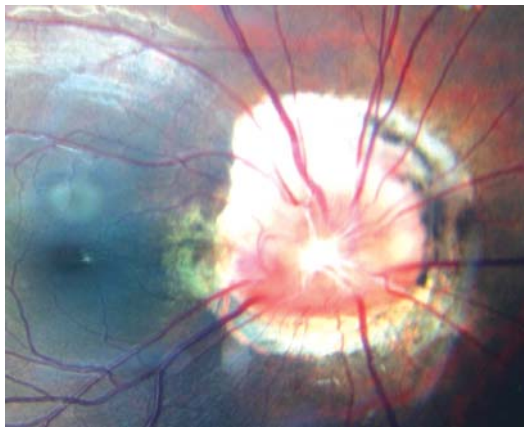


FIGURE 11.7.2: Morning glory syndrome

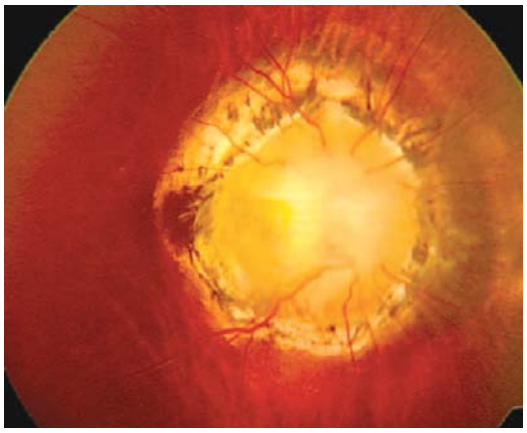


FIGURE 11.7.3: Morning glory syndrome

Small Disk: High Hypermetropia

- Disk is more pink and slightly elevated with small or absent cup **(Fig 11.8.1)**
- Crowding and tortuosity of normal retinal vessels
- Should be differentiated from papilledema as the disk margin is blurred

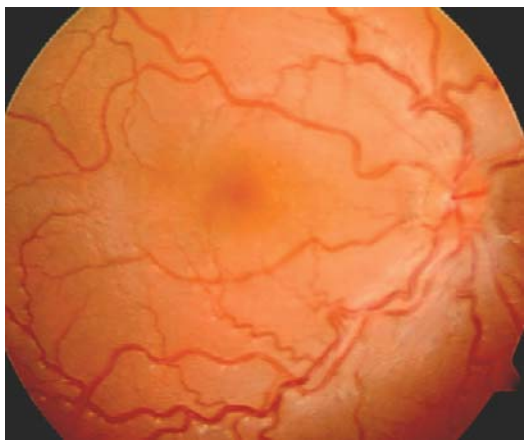


FIGURE 11.8.1: Small disk with tortuous vessels —hypermetropia

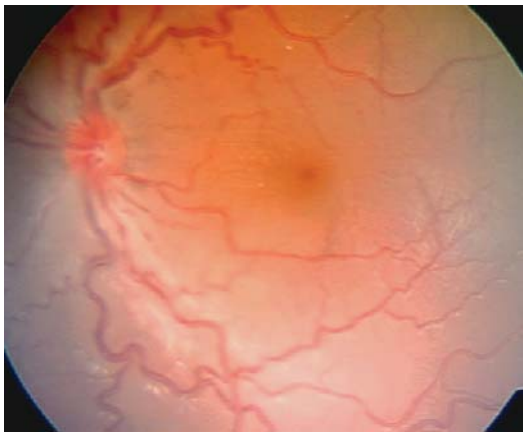


FIGURE 11.8.2: Small disk with tortuous vessels—hypermetropia

Hypoplastic Optic Disk

- Failure of the axons of the ganglionic cells to develop or to reach the disk causes a small hypoplastic optic disk **(Fig 11.9.1)**
- Unilateral or bilateral congenital condition
- Typical appearance consists of a small gray optic disk surrounded by a yellowish peripapillary halo of hypopigmentation (*double-ring sign*) **(Fig 11.9.2)**
- Outer ring corresponds to the margin of normal disk
- Blood vessels are of normal caliber

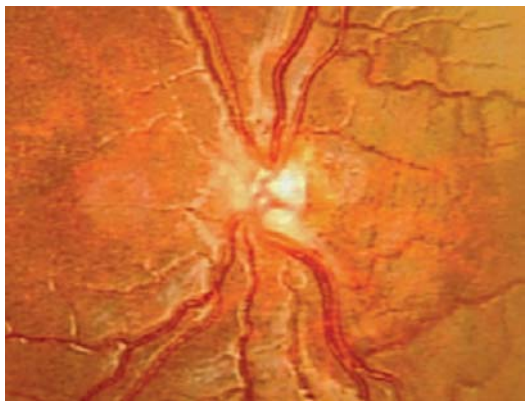


FIGURE 11.9.1: Hypoplastic optic disk

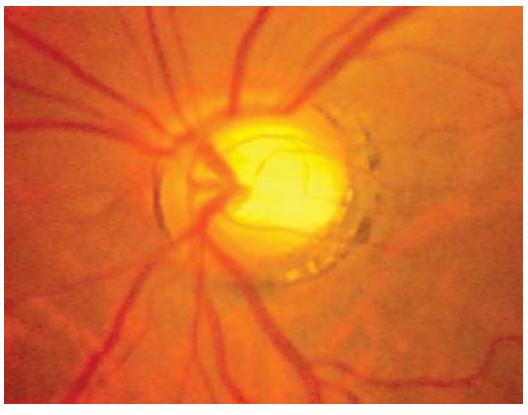


FIGURE 11.9.2: Double-ring sign

Tilted Disk

- Is due to an oblique entrance of the optic nerve into the globe
- Congenital and usually bilateral
- Appearance of the disk is extremely oval or 'D-shaped', with the vertical axis directed obliquely **(Fig 11.10.1)**
- Hypopigmentation of inferonasal fundus and peripapillary atrophy **(Figs 11.10.2 and 11.10.3)**
- *Situs inversus*: temporal blood vessels deviate nasally before turning temporally **(Figs 11.10.4 and 11.10.5)**
- May be associated with myopia, and sometimes with temporal field defect

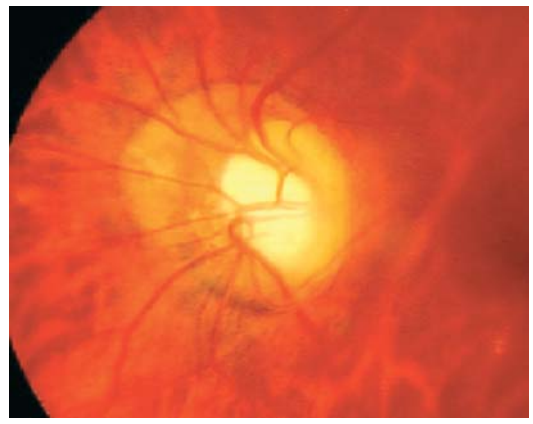


FIGURE 11.10.1: Tilted optic disk

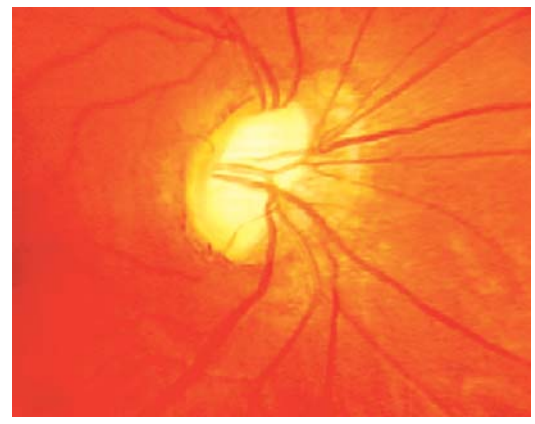


FIGURE 11.10.2: Tilted optic disk

FIGURE 11.10.3: Tilted optic disk

FIGURE 11.10.4: Tilted disk-situs inversus optica

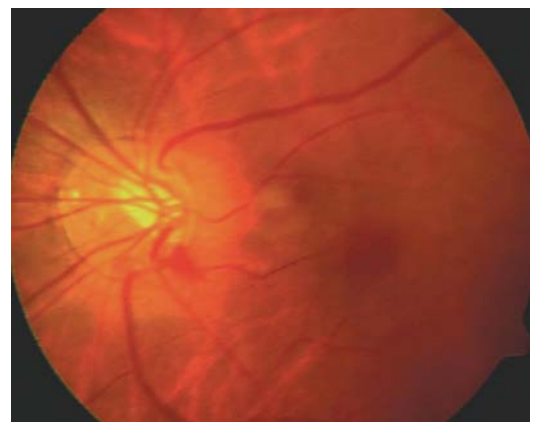


FIGURE 11.10.5: Tilted disk-situs inversus optica

Large Disk: Myopia

- Disk is larger than normal, with normal or large cup
- Temporal **(Fig 11.4.3)** or annular crescent **(Figs 11.11.1 and 11.11.2)**
- Should be differentiated from glaucomatous cupping
- May be associated with posterior staphyloma in very high myopia
- ***See Chapter: 12***

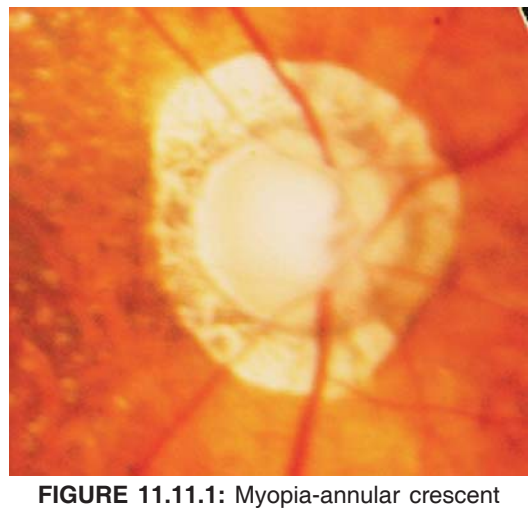


FIGURE 11.11.1: Myopia-annular crescent

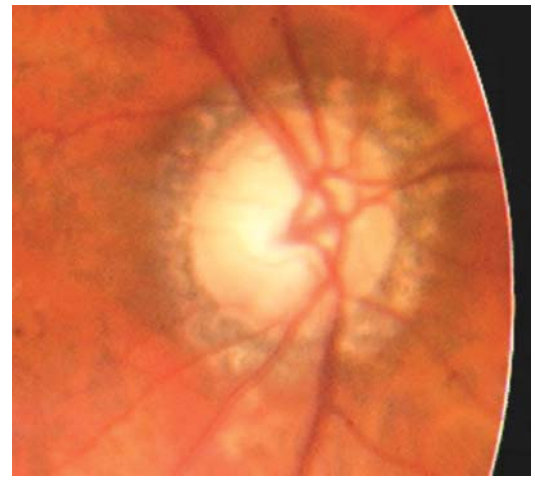


FIGURE 11.11.2: Myopia-annular crescent

Large Disk Megalopapilla:

- Very rare, congenital unilateral condition
- Disk diameter 2.0 mm or more **(Figs 11.12.1 and 11.12.2)**
- Reduced distance between foveola and temporal edge of the disk
- Blood vessels appear normal

FIGURE 11.12.1: Megalopapilla

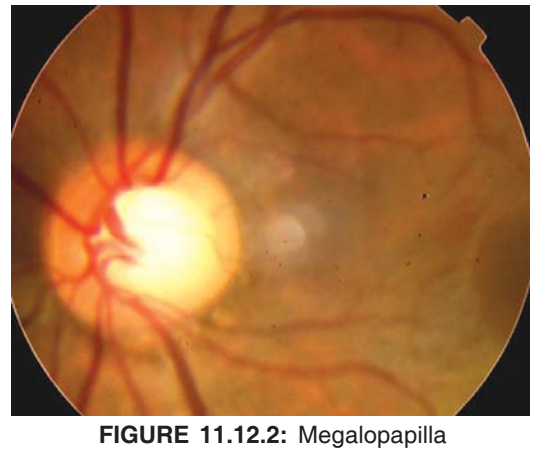


FIGURE 11.12.2: Megalopapilla

Disk with Large Cup

- Normal cups are of *three types*
  - small dimple cup
  - punched out cup
  - cup with temporal slopping
- Normal cup:disk ratio is 0.3 or less **(Fig 11.13.1)**
- A cup:disk ratio > 0.4 is present in 2-5 percent of normal population **(Figs 11.13.2 and 11.13.3)**
  - bilateral and symmetrical **(Figs 11.13.4 and 11.13.5)**
  - absent of notching
  - peripapillary striation of nerve fiber layer can be seen in most cases

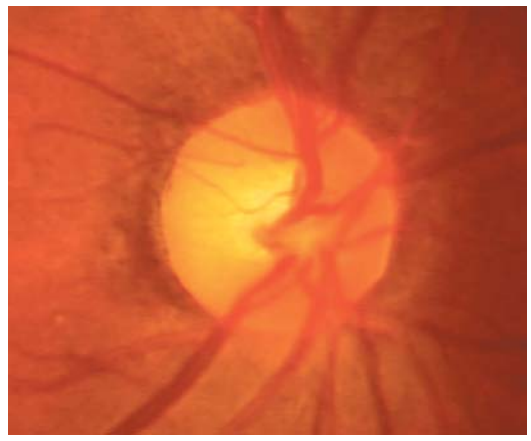


FIGURE 11.13.1: Normal cup

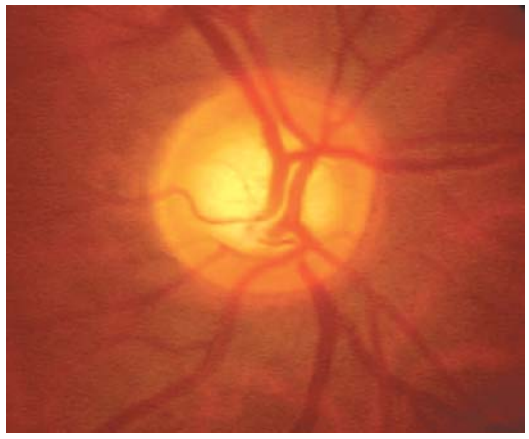


FIGURE 11.13.2: Large cup

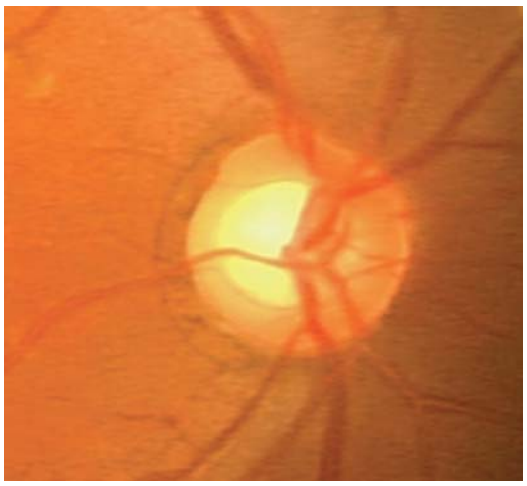


FIGURE 11.13.3: Large cup- myopic crescent

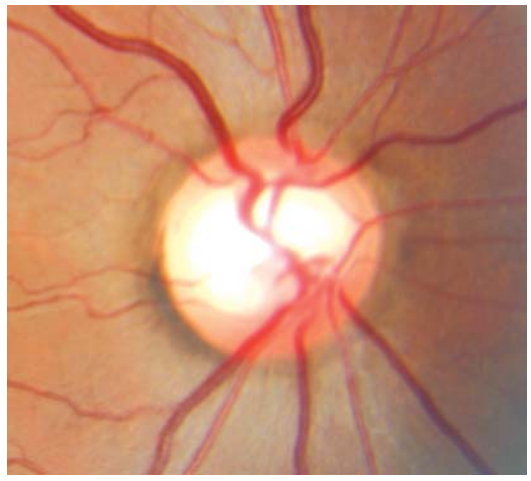


FIGURE 11.13.4: Large cup—symmetrical

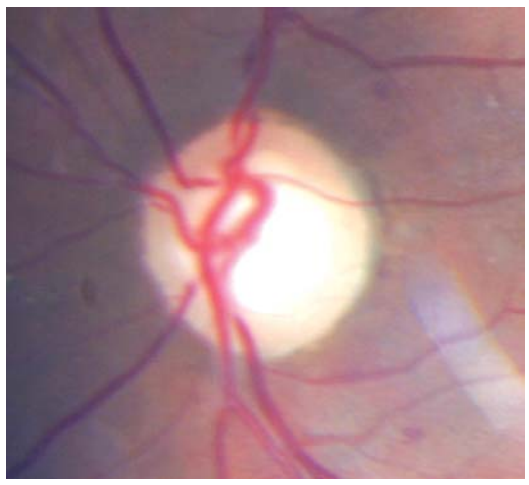


FIGURE 11.13.5: Large cup—symmetrical

Glaucomatous Optic Disk

(See Chapter: 9)

VASCULAR ANOMALIES

Disk Collaterals

- Common shunts that develop within the exiting vascular system
- They are distended flat vessels that start and end on the disk surface **(Figs 11.14.1 and 11.14.2)**
- *Commonest cause*: central retinal venous occlusion

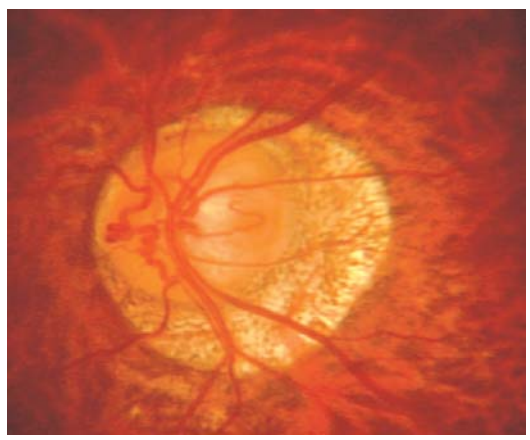


FIGURE 11.14.1: Disk collaterals

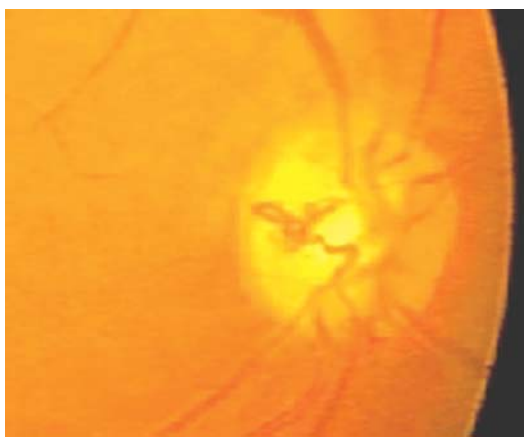


FIGURE 11.14.2: Disk collaterals

New Vessels on the Disk (NVD)

- Very common and associated with areas of capillary drop out
- Unilateral or bilateral, depending upon the cause
- Lace-like fine vessels, may be flat or elevated **(Fig 11.15.1)**
- Vessels may extend to the peripapillary region **(Figs 11.15.2 and 11.15.3)**
- Gliosis may be present in variable amount
- *Causes*: proliferative diabetic retinopathy, retinal venous occlusion (CRVO/ BRVO), central retinal vasculitis, retinal ischemia, etc.

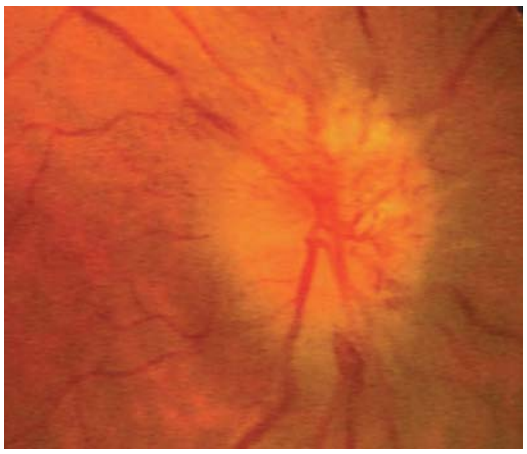


FIGURE 11.15.1: Neovascularization of the disk

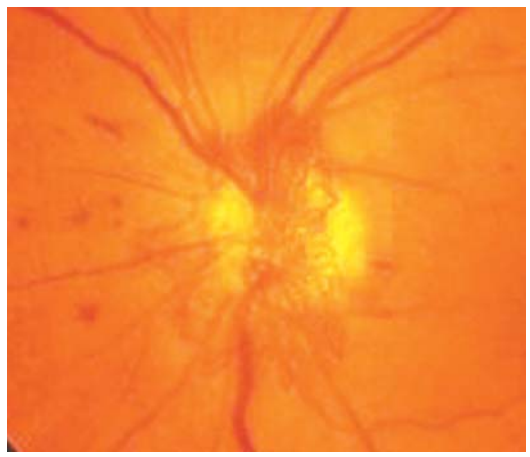


FIGURE 11.15.2: Neovascularization of the disk

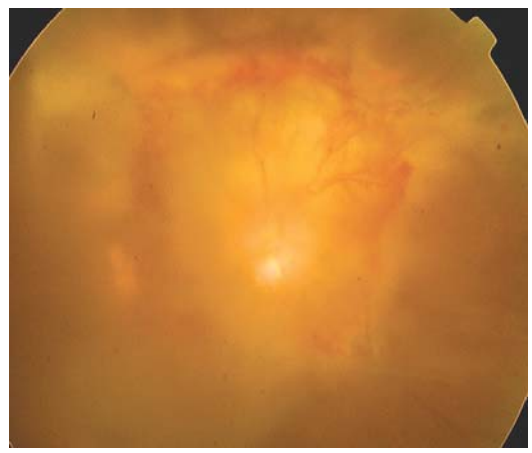


FIGURE 11.15.3: Massive neovascularization of the disk

Optico-ciliary Shunts

- Rare, unilateral anastomosis between the retinal and choroidal blood vessels
- Vessels hook from the center of the cup to the disk-retina junction
- *Causes*: optic nerve sheath meningioma (25% cases), rarely in optic nerve glioma and papilledma

Disk Hemorrhage

- Seen as splinter hemorrhage over the disk and at disk-retina junction **(Figs 11.16.1 to 11.16.3)**
- *Causes*: papilledema, anterior ischemic optic neuropathy (AION), optic neuritis, open angle glaucoma, diabetic papillopathy, acute PVD, etc.

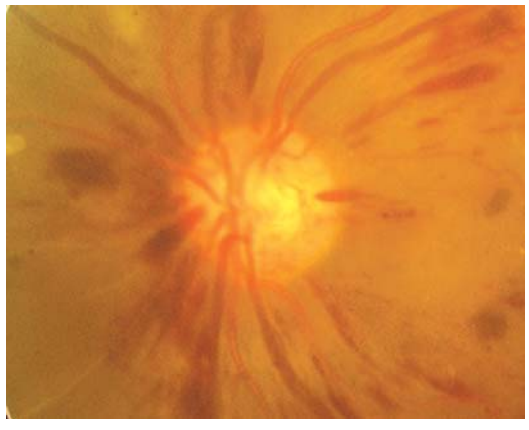


FIGURE 11.16.1: Disk hemorrhage

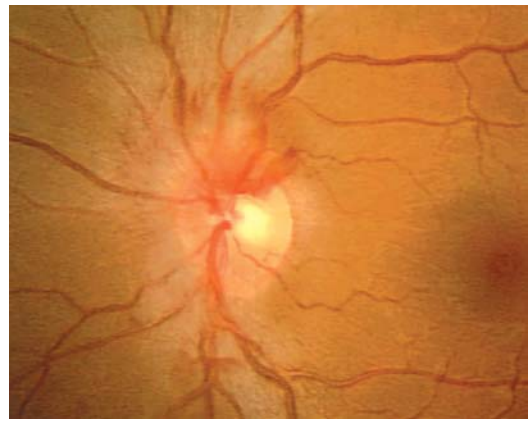


FIGURE 11.16.2: Disk hemorrhage

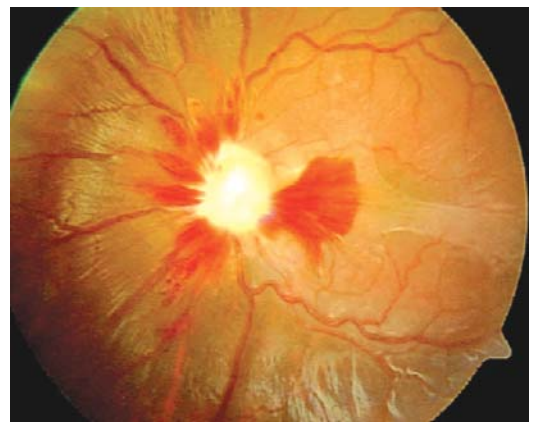


FIGURE 11.16.3: Disk hemorrhage-post traumatic

Dragged Vessels

- Seen in proliferative retinopathies: e.g. PDR, sickle cell retinopathy, ROP, toxocara granuloma and certain hamartomas
- Dragging may be of any direction, but temporal dragging is common **(Figs 11.17.1 to 11.17.3)**

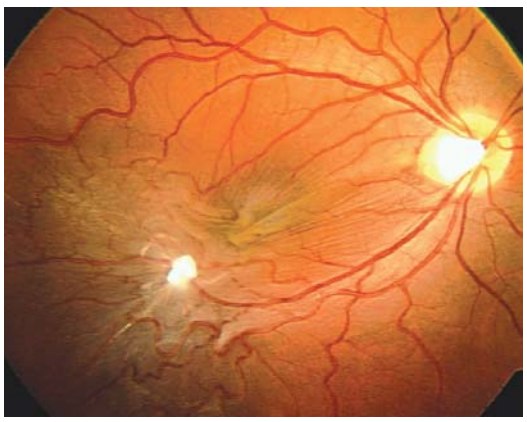


FIGURE 11.17.1: Dragged vessels

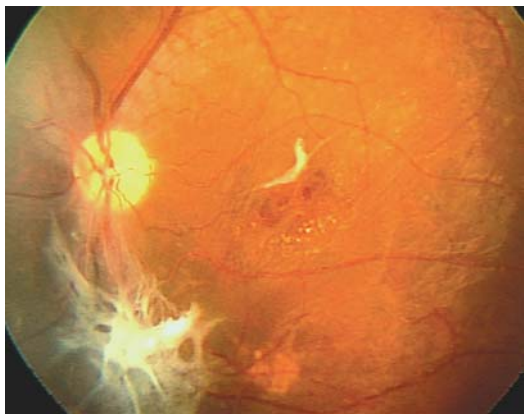


FIGURE 11.17.2: Dragged vessels

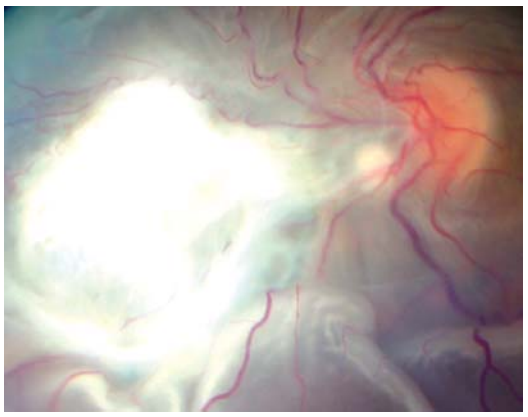


FIGURE 11.17.3: Dragged vessels

OPTIC NEURITIS

Papillitis

- Optic neuritis is an inflammatory or demyelinating disorder of the optic nerve (from the optic disk to the lateral geniculate body)
- Idiopathic type, or from demyelination, typically affects the patients between 20-40 years of age; but post-viral type typically occurs in children.
- It may be *papillitis* (optic neuritis proper), *retrobulbar neuritis* and *neuroretinitis*
- ***Signs of Papillitis*** –
  - sudden visual loss and pupillary signs
  - disk edema with obliteration of the physiological cup
  - hyperemia and blurring of the disk margin **(Figs 11.18.1 and 11.18.2)**
  - hemorrhages on the disk **(Fig 11.18.3)**
  - slit-like defect in nerve fiber layer **(Fig 11.18.4)**
  - inflammatory cells into the adjacent posteriovitreous

Retrobulbar Neuritis

- here, the optic disk, and retinal nerve-fiber layer are normal, as the site of involvement is behind the globe
- visual loss and pupillary sign are important

Neuroretinitis

- never associated with demyelinating diseases
- signs of optic neuritis
- macular star in addition **(Fig 11.18.5)**
- juxta-papillary exudates may be seen

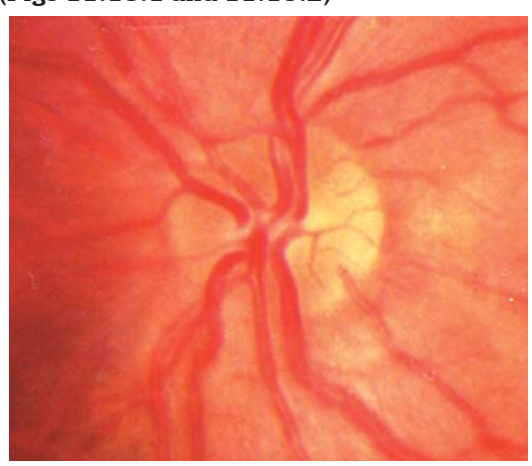


FIGURE 11.18.1: Optic neuritis

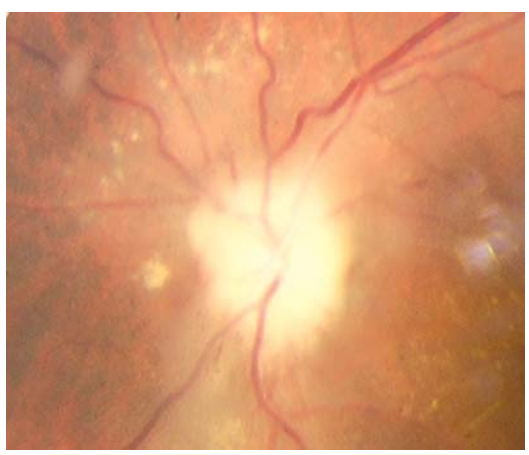


FIGURE 11.18.2: Optic neuritis

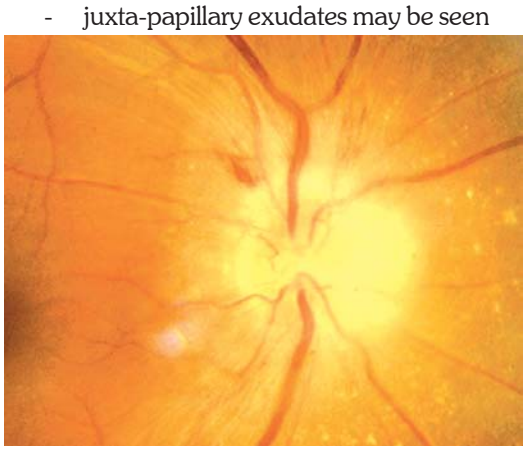


FIGURE 11.18.3: Optic neuritis—disk hemorrhage

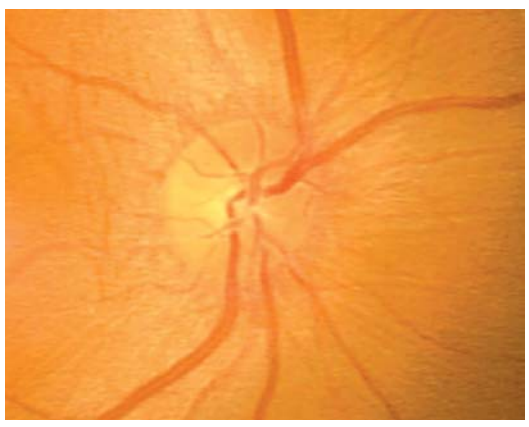


FIGURE 11.18.4: Optic neuritis-slit defect of NFL

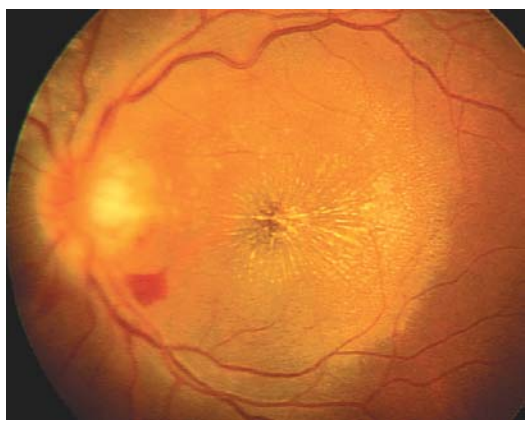


FIGURE 11.18.5: Neuroretinitis—macular star

Anterior Ischemic Optic Neuropathy (AION)

- *Two types*: non arteritic AION and arteritic AION
- *Non arteritic AION*:
  - non arteritic AION is a segmental or generalized infarction of the anterior part of the optic nerve, caused by the occlusion of short posterior ciliary arteries
  - usually unilateral, aged patient between 60-65 years
  - pale, sectorial (usually upper part) disk edema which may be surrounded by splinter hemorrhages **(Figs 11.19.1 and 11.19.2)**
  - associated with sudden altitudinal hemianopia (usually lower field)
- *Arteritic AION*
  - caused by giant cell arteritis, more elderly people
  - unilateral severe loss of vision
  - a swollen, usually diffuse white or pale disk, with splinter hemorrhages around **(Fig 11.19.3)**
  - with time, the entire optic disk becomes pale **(Fig 11.19.4)**
  - fellow eye is frequently affected
  - tender nodular temporal arteries and in severe cases, may be scalp necrosis

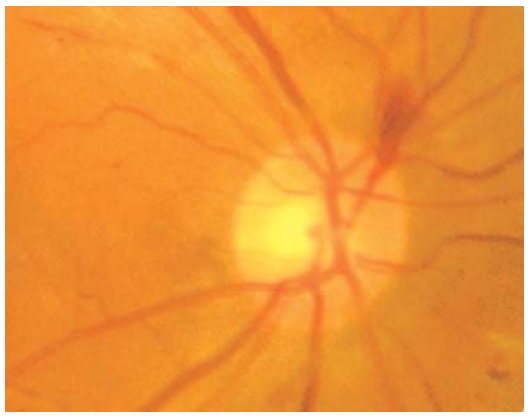


FIGURE 11.19.1: AION-nonarteritic type

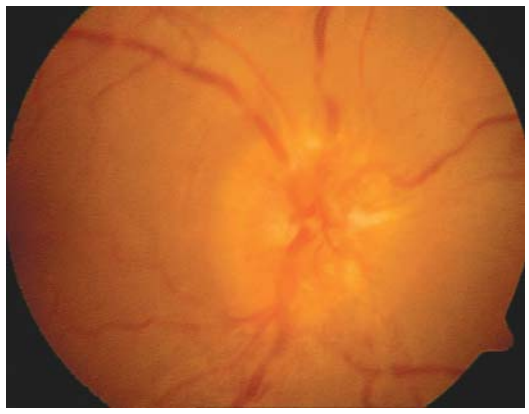


FIGURE 11.19.2: AION-nonarteritic type

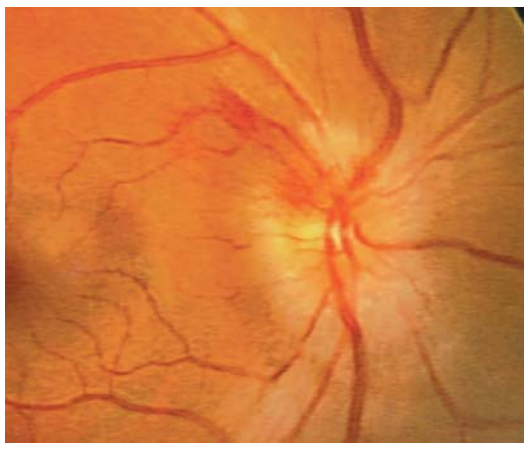


FIGURE 11.19.3: AION-arteritic type

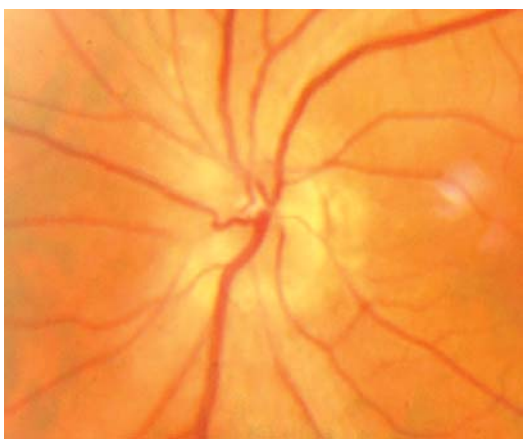


FIGURE 11.19.4: AION-arteritic type

OTHER CAUSES OF UNILATERAL DISK EDEMA

Central Retinal Venous Occlusion

- Unilateral condition, with moderate to severe visual loss in elderly patients
- Disk edema with splinter hemorrhages on the disk and peripapillary area **(Fig 11.20.1)**
- Venous engorgement and wide spread retinal hemorrhages **(Fig 11.20.2)**
- Soft exudates may be present

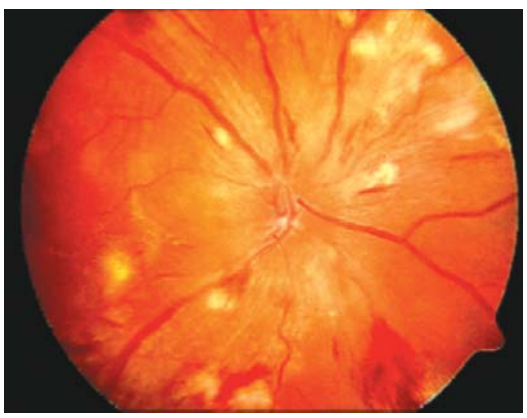


FIGURE 11.20.1: Central retinal venous thrombosis

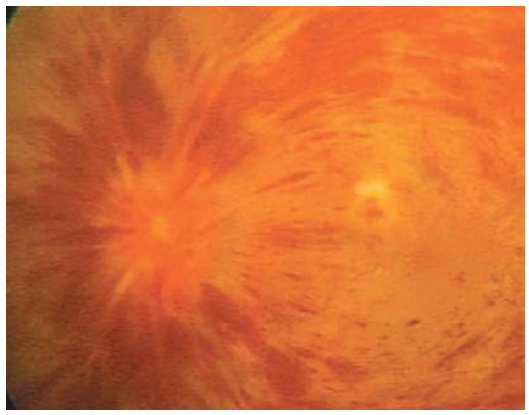


FIGURE 11.20.2: Central retinal venous thrombosis

Central Vasculitis (papillophlebitis)

- Similar to CRVO, but it affects in younger, otherwise healthy individuals **(Fig 11.21.1)**
- Variable disk edema, venous engorgement, peripapillary and retinal hemorrhages **(Fig 11.21.2)**
- May be associated with macular star **(Fig 11.21.3)**
- Prognosis is better than CRVO

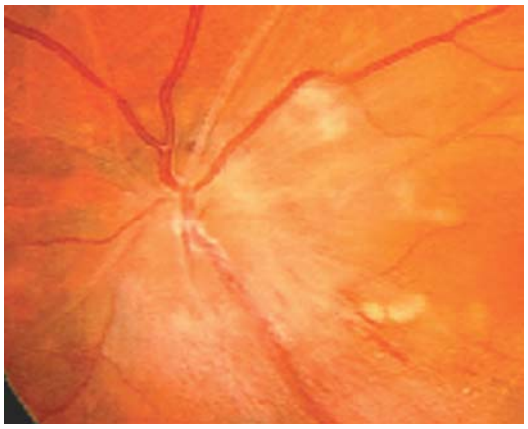


FIGURE 11.21.1: Central vasculitis

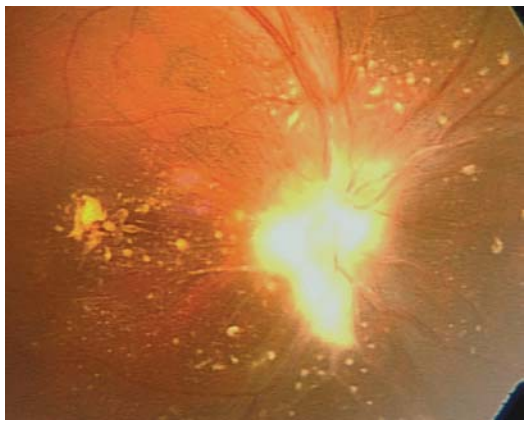


FIGURE 11.21.2: Central periphlebitis

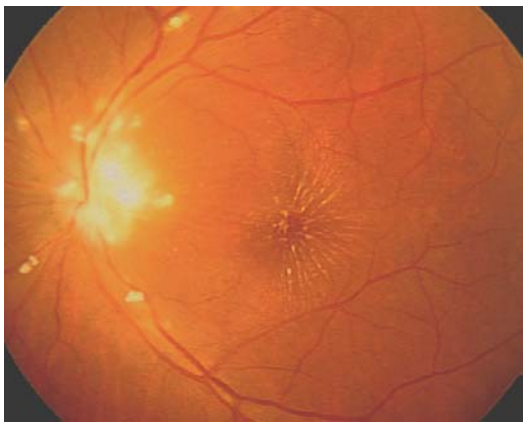


FIGURE 11.21.3: Central vasculitis-macular star

Long-standing Hypotony

- Seen after glaucoma filtration surgery, (overfiltration) prolonged wound leak, chronic anterior uveitis, choroidal detachments, etc. **(Figs 11.22.1 and 11.22.2)**

FIGURE 11.22.1: Over filtering bleb—hypotony

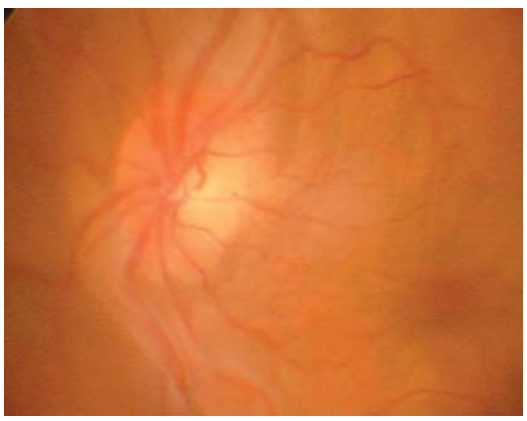


FIGURE 11.22.2: Disk edema-long standing hypotony

Posterior Scleritis

- Unilateral or bilateral condition with some systemic disease
- Disk edema without hemorrhage **(Fig 11.23.1)**
- Cells in the posterior vitreous

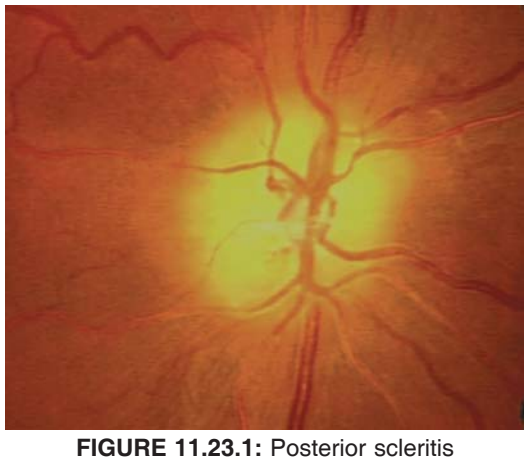


FIGURE 11.23.1: Posterior scleritis

PAPILLEDEMA

- Papilledema is the bilateral, non-inflammatory passive swelling of the optic disk, produced by raised intracranial tension (ICT)
- Unilateral papilledema with optic atrophy on the other side suggests a frontal lobe tumor or olfactory meningioma of the opposite side—the *Foster-Kennedy syndrome* **(Figs 11.24.1 and 11.24.2)**
- Signs of papilledema depend on duration and its severity
- *Four stages*: early, established, chronic and atrophic

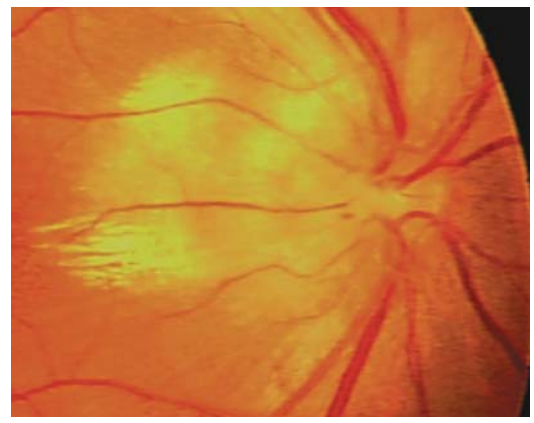


FIGURE 11.24.1: Foster-Kennedy syndrome

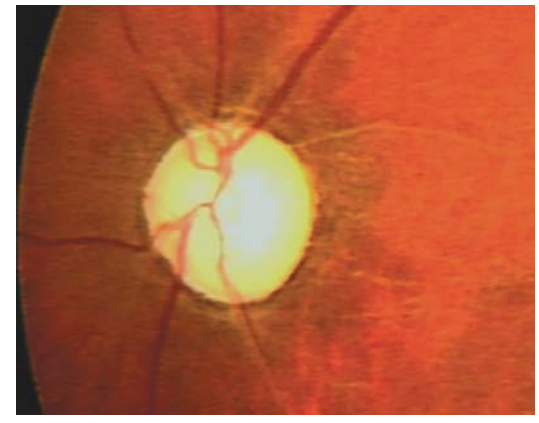


FIGURE 11.24.2: Foster-Kennedy syndrome

Early Papilledema

- Disk hyperemia, elevation and preservation of optic cup **(Fig 11.24.3)**
- Blurring of the nasal sector of disk margin first, then the superior and inferior **(Fig 11.24.4)**
- Splinter hemorrhages at or just off the disk-margin
- Absent spontaneous venous pulsation (also absent in 20 percent of general population)

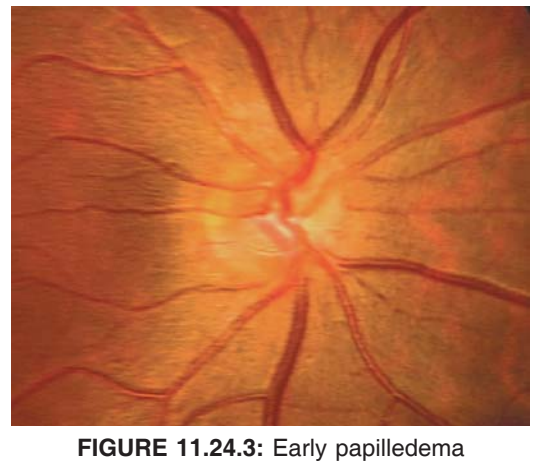


FIGURE 11.24.3: Early papilledema

FIGURE 11.24.4: Early papilledema

Established (acute) Papilledema

- Disk elevation is marked
- Entire disk margins become indistinct and central cup is obliterated **(Figs 11.24.5 and 11.24.6)**
- Venous engorgement, flame-shaped hemorrhage and peripapillary edema **(Fig 11.24.7)**
- *Patton's line*—peripapillary circumferential retinal folds due to accumulation of fluid **(Figs 11.24.8 and 11.24.9)**

FIGURE 11.24.5: Established papilledema

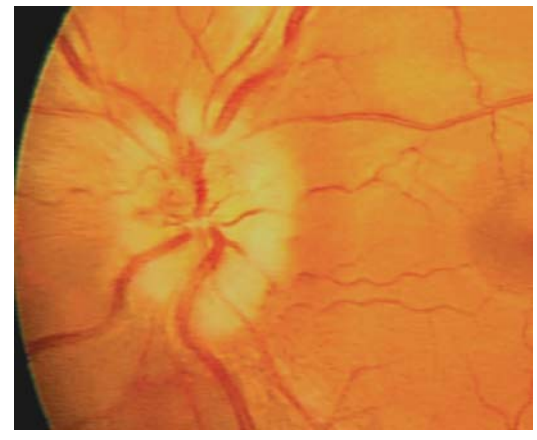


FIGURE 11.24.6: Established papilledema

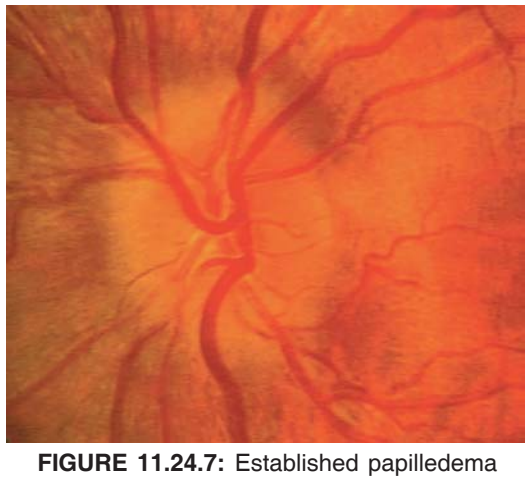


FIGURE 11.24.7: Established papilledema

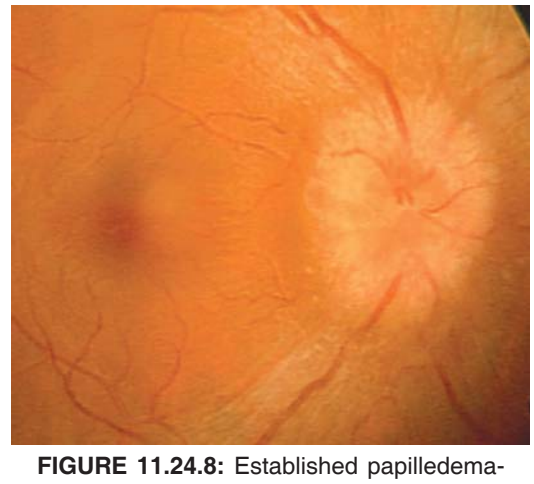


FIGURE 11.24.8: Established papilledema-Patton's line

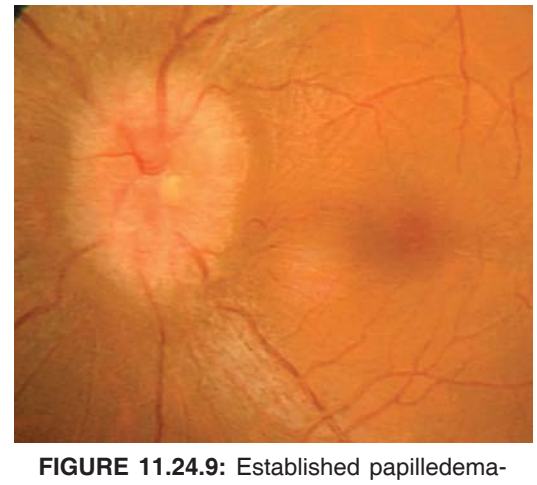


FIGURE 11.24.9: Established papilledema-Patton's line

Chronic Papilledema

- Hemorrhagic and exudative components gradually resolve **(Figs 11.24.10 and 11.24.11)**
- Optic disk appears as a '*champagne cork*'-like elevation **(Figs 11.24.12 and 11.24.13)**
- Optico-ciliary shunt may be visible
- A macular star may be present

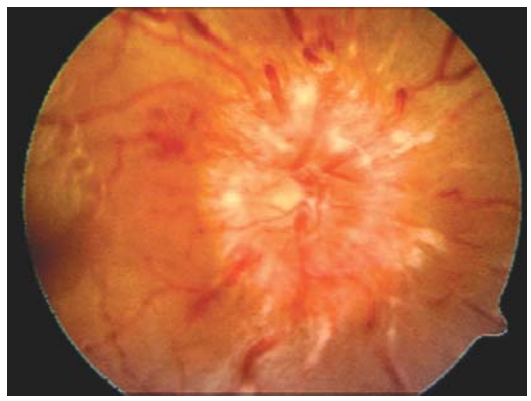


FIGURE 11.24.10: Chronic papilledema

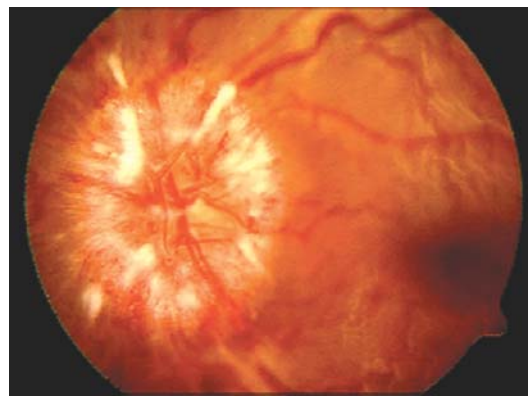


FIGURE 11.24.11: Chronic papilledema

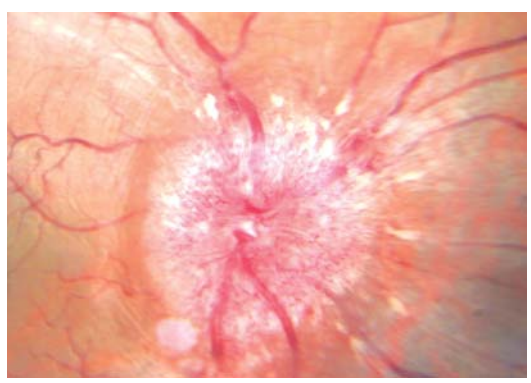


FIGURE 11.24.12: Chronic papilledema-champagne cork appearance

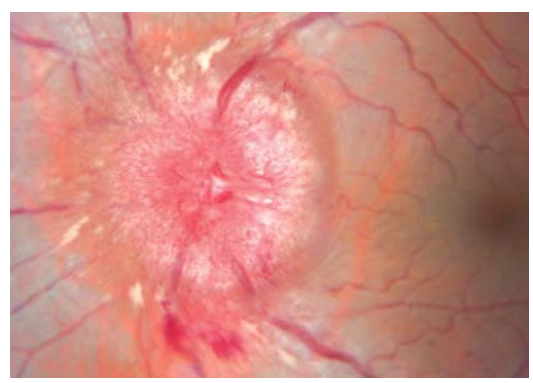


FIGURE 11.24.13: Chronic papilledema-champagne cork appearance

Atrophic Papilledema

- Dirty-white appearance of the optic disk, due to reactive gliosis—leading to secondary optic atrophy **(Fig 11.24.14)**
- Retinal vessels are attenuated with perivascular sheathing **(Fig 11.24.15)**
- Peripapillary pigmentary changes

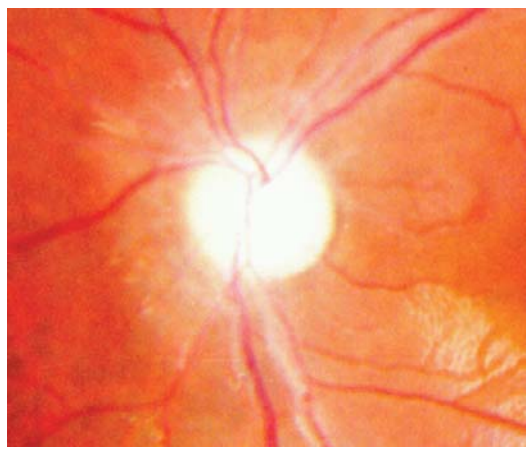


FIGURE 11.24.14: Atrophic papilledema

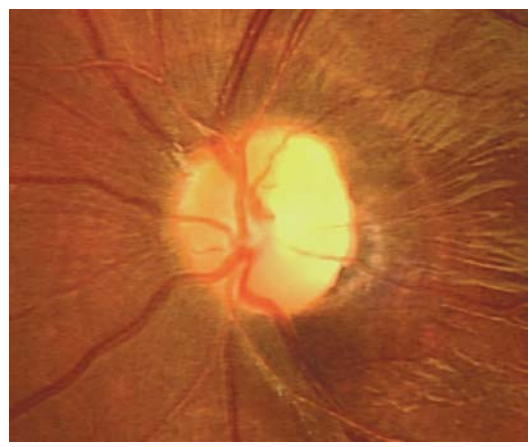


FIGURE 11.24.15: Atrophic papilledema

OTHER CAUSES OF BILATERAL DISK EDEMA

- **Malignant Hypertension**
  - presence of severe rise in blood pressure
  - more in secondary hypertension
  - bilateral disk edema, flame-shaped hemorrhage, soft exudates and a macular star **(Figs 11.25.1 and 11.25.2)**
  - variable amount of hypertensive retinal changes
  - peripheral pale areas of choroidal infarcts
- Compressive thyroid optic neuropathy
- Acute methyl alcohol toxicity
- Bilateral optic neuritis

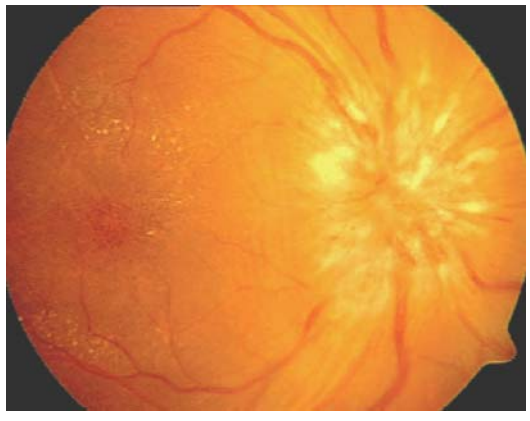


FIGURE 11.25.1: Disk edema—malignant hypertension

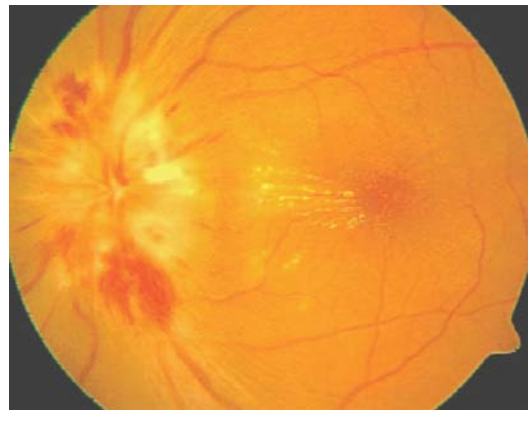


FIGURE 11.25.2: Disk edema—malignant hypertension

OPTIC ATROPHY

- Degeneration of optic nerve fibers with loss of their myelin sheaths, and characterized by the pallor of the optic disk
- 'Pallor' of the disk is not due to atrophy of the nerve fibers, but due to loss of vascularity owing to obliteration of the disk capillaries
- *Three types:* primary, secondary and consecutive

Primary Optic Atrophy

- Pallor of the disk, with white or gray color, without significant gliosis
- Disk is flat, margin is sharply defined, and peripapillary retina is normal looking **(Fig 11.26.1)**
- Reduction in number of small vessels crossing the disk **(Fig 11.26.2)**
- Thinning of nerve fiber layer

Secondary Optic Atrophy

- Preceded by edema of the optic nerve head
- Dirty-gray color with blurred margins
- Cup is full, and lamina cribrosa is obscured **(Fig 11.27.1)**
- Narrowing of the blood vessels with sheathing **(Fig 11.27.2)**
- Seen in papilledema, optic neuritis or after AION

Consecutive Optic Atrophy

- Yellowish-waxy pallor of the disk **(Fig 11.28.1)**
- Disk margins are less sharply defined **(Fig 11.28.2)**
- Marked narrowing or even obliteration of arterioles **(Figs 11.28.3 and 11.28.4)**
- Seen in retinitis pigmentosa, after extensive panretinal photocoagulation, disseminated choroiditis, etc.

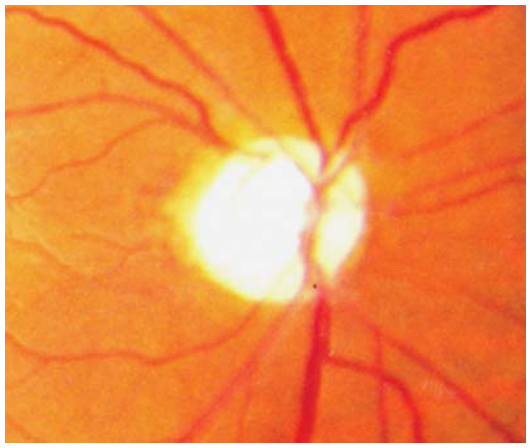


FIGURE 11.26.1: Primary optic atrophy

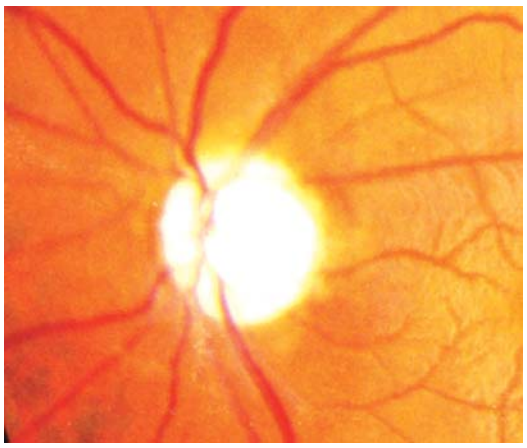


FIGURE 11.26.2: Primary optic atrophy

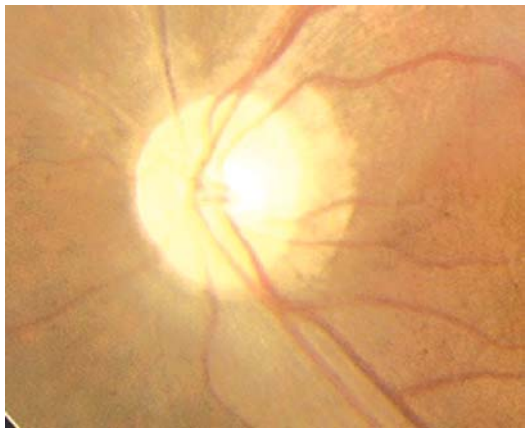


FIGURE 11.27.1: Secondary (postneuritic) optic atrophy

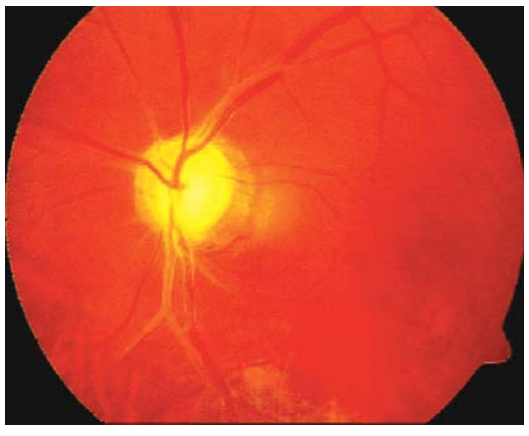


FIGURE 11.27.2: Secondary (postneuritic) optic atrophy

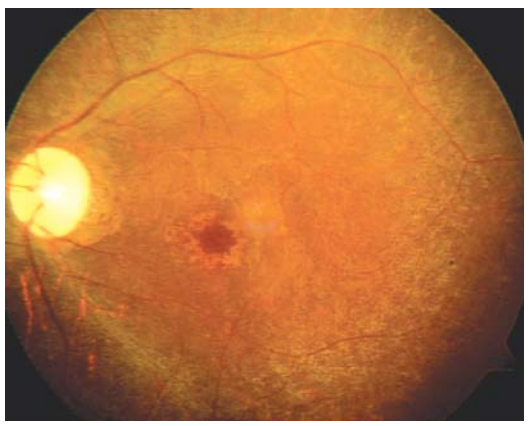


FIGURE 11.28.1: Consecutive optic atrophy

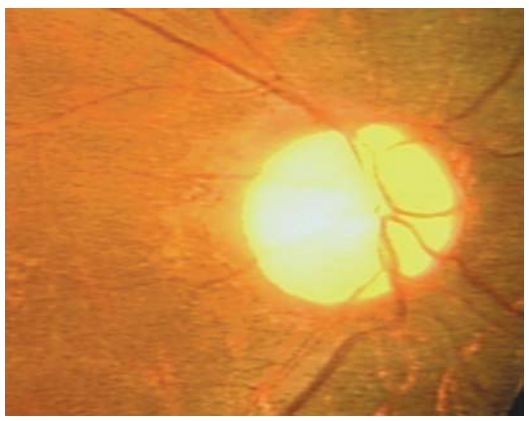


FIGURE 11.28.2: Consecutive optic atrophy

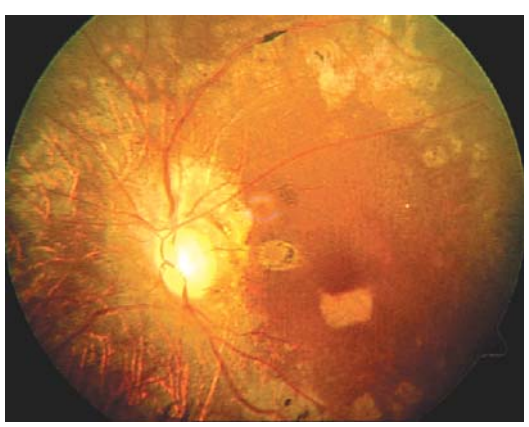


FIGURE 11.28.3: Consecutive optic atrophy

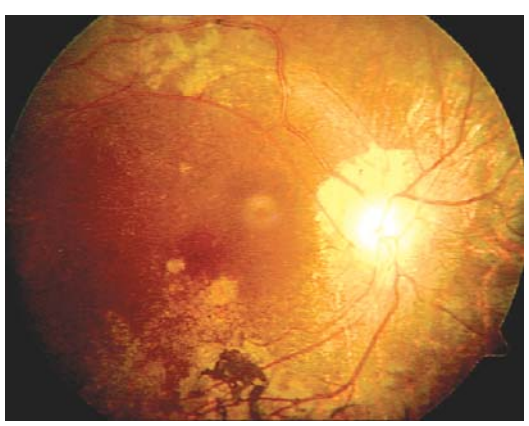


FIGURE 11.28.4: Consecutive optic atrophy

Glaucomatous Optic Atrophy

- Associated with total cupping of the disk **(Figs 11.29.1 and 11.29.2)**

(See Also Chapter: 9)

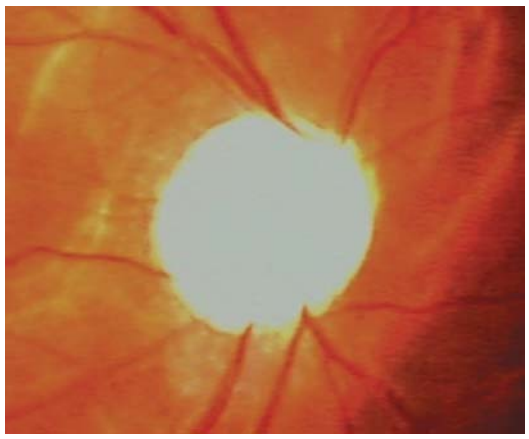


FIGURE 11.29.1: Glaucomatous optic atrophy

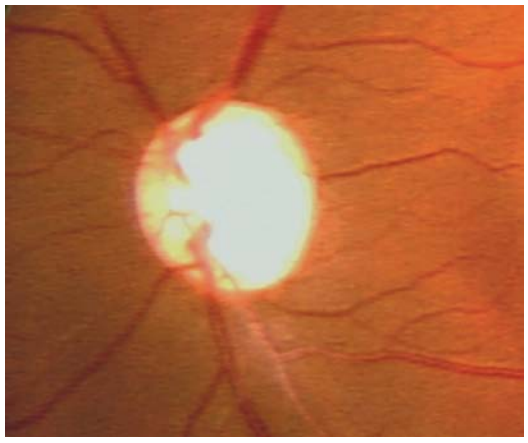


FIGURE 11.29.2: Glaucomatous optic atrophy

Temporal Pallor

- Usually a bilateral condition
- A form of partial optic atrophy, involves a loss of temporal fibers including the papillo-macular bundles **(Figs 11.30.1 and 11.30.2)**
- Temporal side is normally relatively pale, because the retinal vessels emerge from the nasal side and the temporal side is normally less vascular

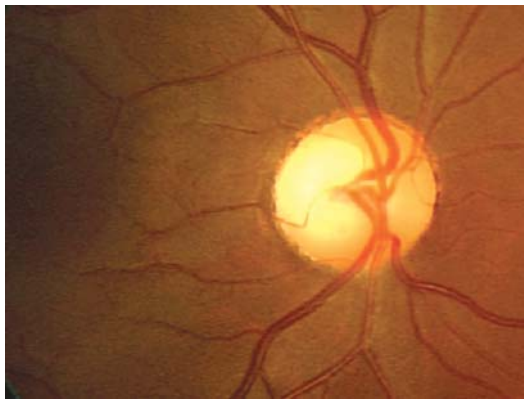


FIGURE 11.30.1: Temporal pallor

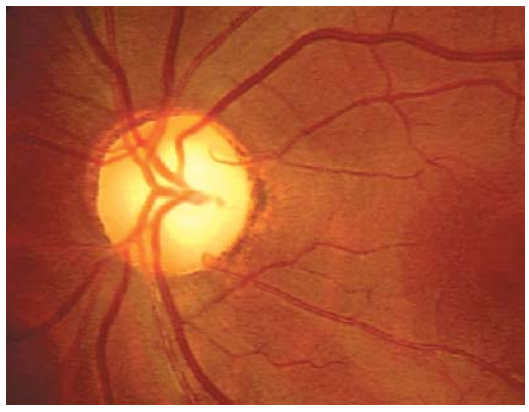


FIGURE 11.30.2: Temporal pallor

PRIMARY OPTIC DISK TUMORS

Optic Disk Capillary Hemangioma

- Rare, unilateral, may also involve the retina
- 25 percent patients have von-Hippel-Lindau syndrome
- Orange-red lesion of the optic disk with macular hard exudates **(Figs 11.31.1 to 11.31.3)**
- Peripheral retinal angiomas or grossly dilated blood vessels

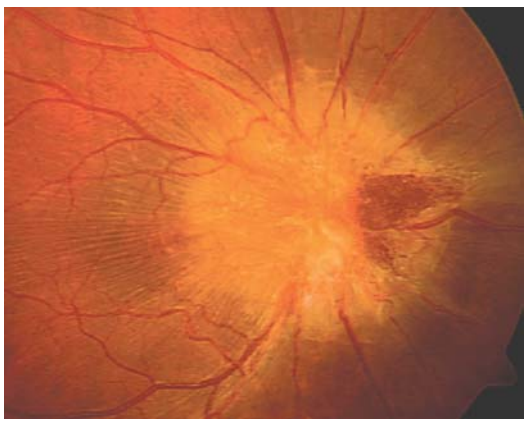


FIGURE 11.31.1: Optic disk hemangioma

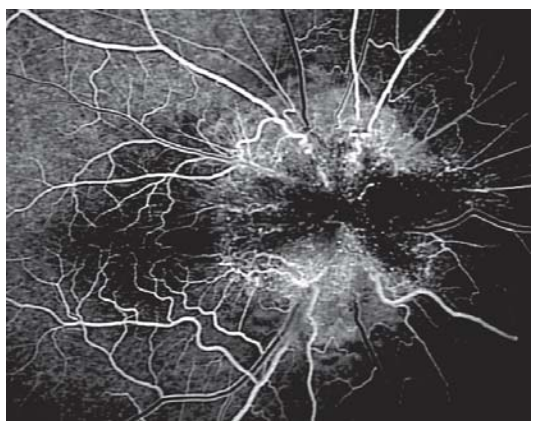


FIGURE 11.31.2: Optic disk hemangioma

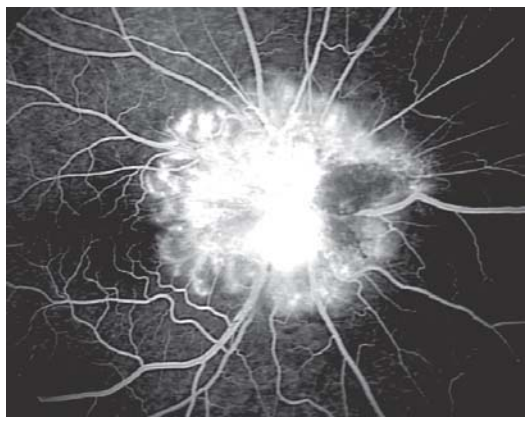


FIGURE 11.31.3: Optic disk hemangioma

Optic Disk Cavernous Hemangioma

- Very rare, unilateral benign tumor
- May also involve the adjacent retina
- Saccular aneurysm-like lesion with dark blood vessels at the disk **(Figs 11.32.1 and 11.32.2)**

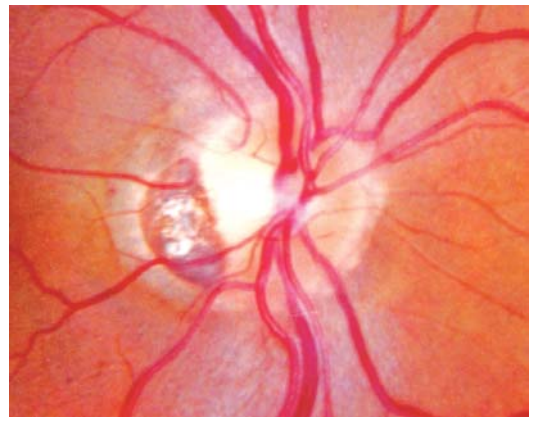


FIGURE 11.32.1: Optic disk hemangioma-cavernous

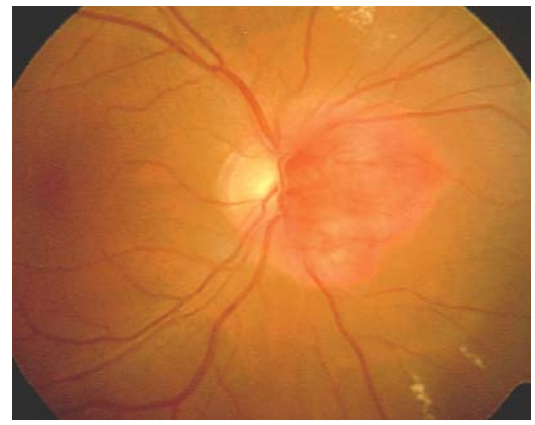


FIGURE 11.32.2: Optic disk haemangioma—involvement of adjacent retina

Optic Disk Melanocytoma

- Rare unilateral, benign tumor in pigmented races
- Pigmented disk lesion frequently located eccentrically **(Figs 11.33.1 and 11.33.2)**
- May induce disk swelling **(Figs 11.33.3 and 11.33.4)** and interfere with vision **(Figs 11.33.5 and 11.33.6)**

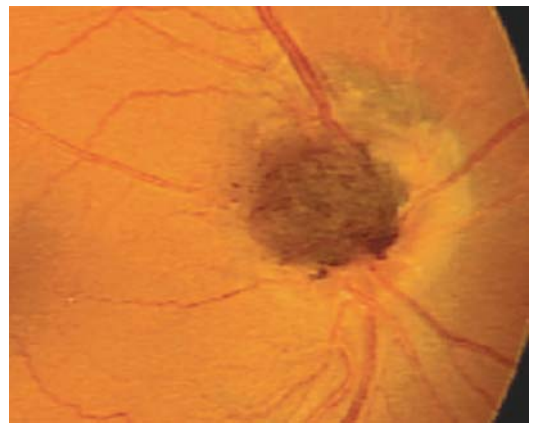


FIGURE 11.33.1: Optic disk melanocytoma

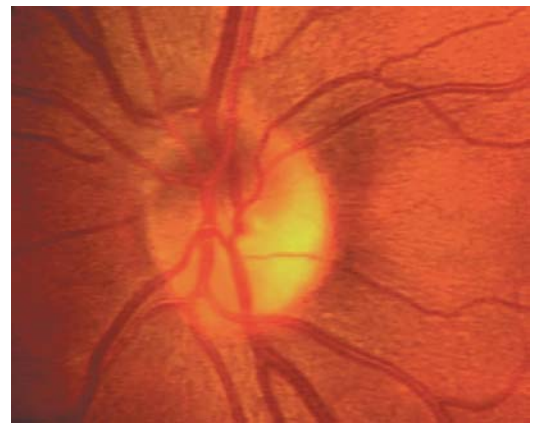


FIGURE 11.33.2: Optic disk melanocytoma

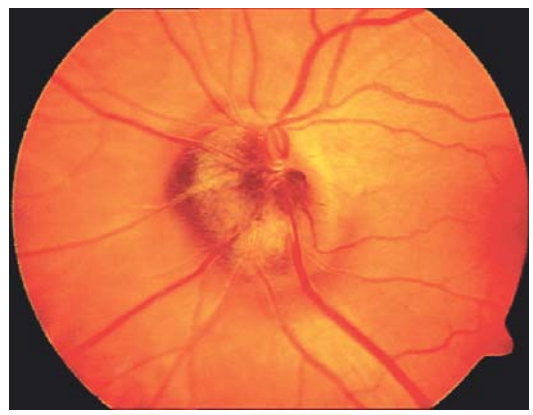


FIGURE 11.33.3: Optic disk melanocytoma

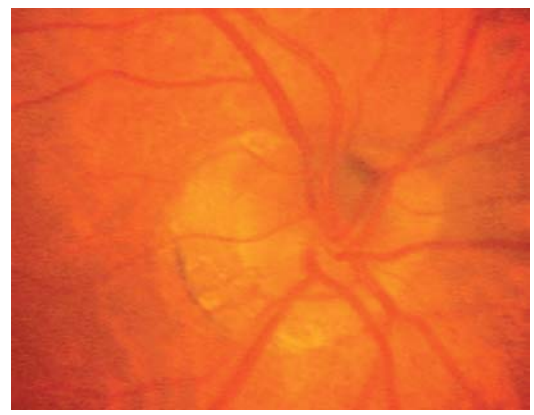


FIGURE 11.33.4: Optic disk melanocytoma

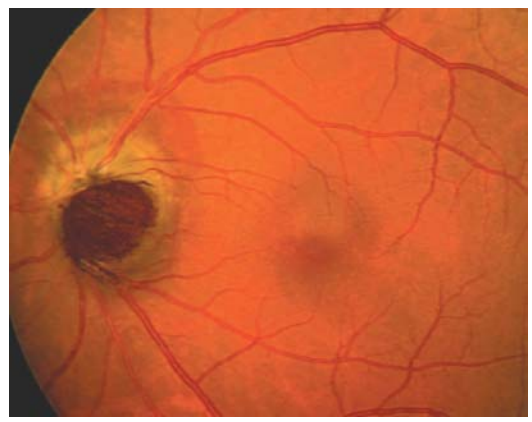


FIGURE 11.33.5: Optic disk melanocytoma

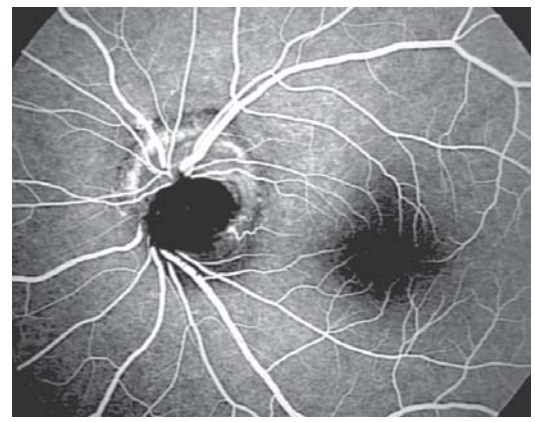


FIGURE 11.33.6: Optic disk melanocytoma—FFA

Astrocytoma

- Unilateral giant drusen, a benign condition
- Frequently associated with tuberous sclerosis or *epiloia* **(Fig 11.34.1)**
- Semitransparent, mulberry-like lesion that displays autofluorescence **(Fig 11.34.2)**

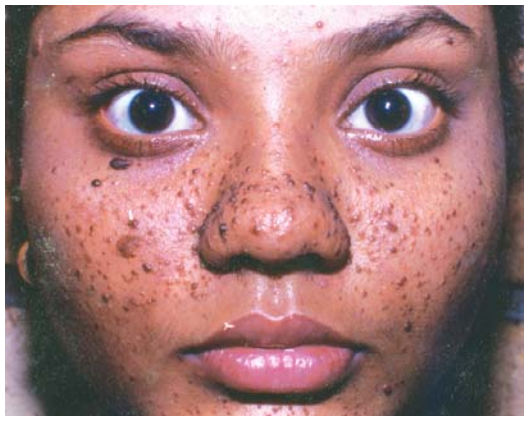


FIGURE 11.34.1: Tuberous sclerosis

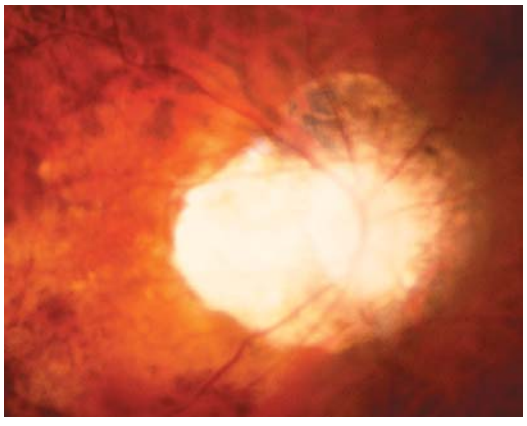


FIGURE 11.34.2: Optic disk astrocytoma

Optic Nerve Glioma and Meningioma

- They present as proptosis and *see Chapter: 14*
- In late stage there is pallor of the disk and primary optic atrophy
- Long standing visual impairment, a pale swollen disk, and optico-cillary shunt vessels is pathognomonic of optic nerve sheath meningioma

INFILTRATIVE LESIONS

Retinoblastoma, choroidal melanoma, leukemia, metastatic carcinoma **(Fig 11.35.1)**, toxocara infection **(Fig 11.35.2)**, cytomegalovirus (CMV) infection **(Fig 11.35.3)**, etc.

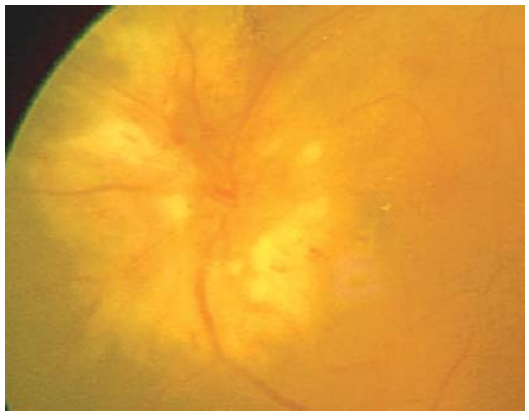


FIGURE 11.35.1: Optic disk infiltration-metastatic deposits

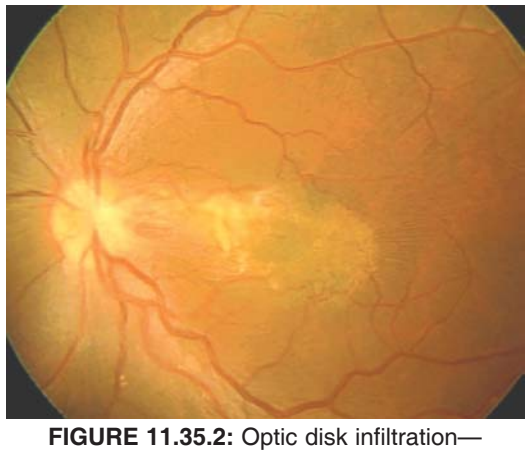


FIGURE 11.35.2: Optic disk infiltration—toxocara

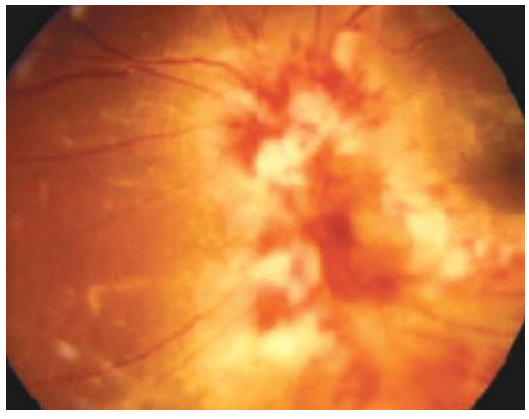


FIGURE 11.35.3: Optic disk infiltration-CMV retinitis

12 Diseases of the Retina

CONGENITAL RETINAL DISORDERS

- Myelinated nerve fibers
- Phakomatosis
- Albinism
- Retinochoroidal coloboma

RETINAL VASCULAR DISORDERS

- Central retinal artery occlusion
- Central retinal vein occlusion
  - Non-ischemic CRVO
  - Ischemic CRVO
- Branch retinal occlusion
- Preretinal hemorrhage
- Roth spot
- Subretinal hemorrhage
- Sub-RPE hemorrhage
- Choroidal hemorrhage
- Neovascularization
- Collaterals
- Retinal hemangiomas
- Retinal vasculitis
- Frosted branch angitis

RETINAL EXUDATES

- Hard exudates
- Macular stars
- Soft exudates or cotton wool spots
- Subretinal exudates
- Coats' disease

INFLAMMATORY RETINAL LESIONS

- Multiple evanescent white dot syndrome
- Punctate inner choroidopathy
- Birdshot retinochoroidopathy
- Acute posterior multifocal placoid pigment epitheliopathy
- Multifocal choroiditis with panuveitis
- Disseminated choroiditis
- Serpiginous choroidopathy
- Focal toxoplasmosis
- Toxocariasis
- Candidiasis
- Cytomegalovirus retinitis
- Acute retinal necrosis
- Progressive outer retinal necrosis
- Vogt-Koyanagi-Harada's syndrome
- Sympathetic ophthalmia
- Tuberculous choroiditis

MACULAR LESIONS (MACULOPATHIES)

HEREDITARY MACULOPATHIES

- Stargardt's macular dystrophy
- Central areolar choroidal dystrophy
- Best's vitelliform macular dystrophy
- Hereditary cone dystrophy (Bull's eye maculopathy)
- Myopic maculopathies
- Dry age-related macular degeneration

EXUDATIVE MACULOPATHIES

- Cystoid macular edema
- Central serous retinopathy
- RPE detachment
- Wet age-related macular degeneration
- Macular hole

OTHER MACULOPATHIES

- Cellophane maculopathy
- Commotio retinae (Barlin's edema)
- Crystalline maculopathy

MULTIPLE FLECKED RETINAL LESIONS

- Familial dominant drusen (Doyne's honeycomb dystrophy)
- Fundus flavimaculatus
- Fundus albipunctatus
- Benign flecked retina syndrome
- Pattern macular dystrophy
- Hard drusens
- Soft drusens
- Cuticular (basal-laminer) drusens

PIGMENTARY RETINOPATHY

- Typical retinitis pigmentosa
- Atypical retinitis pigmentosa

RETINAL DETACHMENT

- Rhegmatogenous retinal detachment
- Associated predisposing factors
- Tractional retinal detachment
- Exudative retinal detachment

HYPERTENSIVE RETINOPATHY

DIABETIC RETINOPATHY

- Nonproliferative diabetic retinopathy
- Diabetic maculopathy
- Proliferative diabetic retinopathy
- Advanced proliferative diabetic retinopathy

RETINOPATHY OF PREMATURITY

RETINOBLASTOMA

MISCELLANEOUS RETINAL CONDITION

- Idiopathic polypoidal choroidal vasculopathy
- Familial exudative vitrioretinopathy
- Stickler's disease
- Idiopathic parafoveal telangiectasia
- Retinal cyst

CONGENITAL RETINAL DISORDERS

Myelinated Nerve Fibers

- Myelinated (opaque) nerve fibers is a congenital condition **(Fig 12.1.1)**
- Present 1 percent of normal population
- Appear as white patches with radial striations at peripheral edges - feathery appearance ***(See also Chapter: 11)***
- Usually peripapillary or sometimes peripheral and isolated **(Fig 12.1.2)**
- No treatment is required

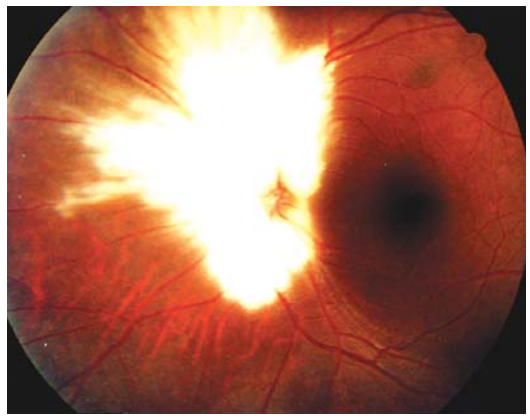


FIGURE 12.1.1: Myelinated nerve fibers

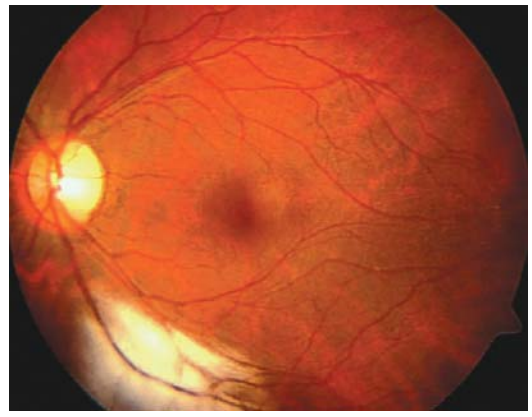


FIGURE 12.1.2: Myelinated nerve fibres

Phakomatosis

- A group of conditions (hamartomas) in which there are congenital, disseminated, usually benign tumors of the blood vessels or neural tissues
- Often ocular, cutaneous, and intracranial in location
- *Neuro-fibromatosis* (von Recklinghousen's disease)
  - most common type, with typical subcutaneous nodules and café-au-lait spots **(Fig 6.34.1)**
  - iris nodule- Leish's nodule **(Fig 6.34.3)**
  - plexiform tumors of lids with ptosis thickened corneal nerves **(Fig 14.24.3)**
- *Tuberous sclerosis* (Bourneville's disease)
  - *diagnostic triad* are epilepsy, mental retardation and adenoma sebaceum **(Fig 12.2.1)**
  - called '*epiloia*': *eip* (epilepsy), *loi* (low IQ), *a* (adenoma sebaceum)
  - ocular lesion: astrocytoma optic disk or retina proper in 50 percent cases **(Fig 12.2.2)**
- *Angiomatosis retinae* (von Hipple-Lindau's disease)
  - a reddish, slightly elevated tumor is seen in the retina which is nourished by dilated large retinal artery and vein **(Figs 12.2.3 and 12.2.4)**
- *Encephalo-trigeminal angiomatosis* (Sturge-Weber's syndrome)
  - port-wine stain along the distribution of the trigeminal nerve **(Figs 12.2.5 and 12.2.6)**
  - choroidal hemangioma which may result in a congenital glaucoma

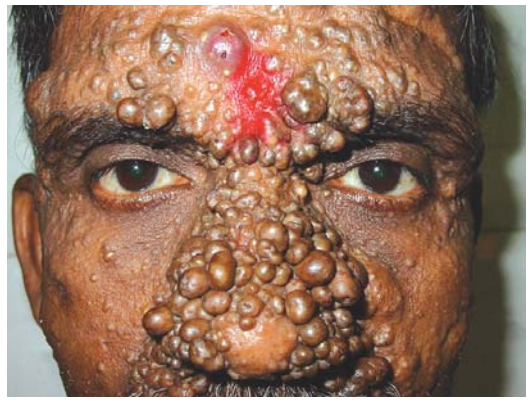


FIGURE 12.2.1: Tuberous sclerosis—adenoma sebaceum

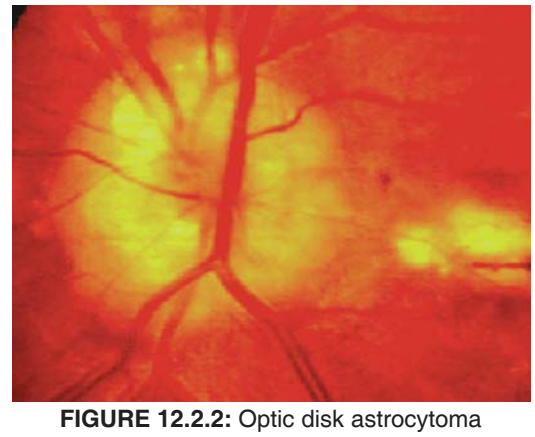


FIGURE 12.2.2: Optic disk astrocytoma

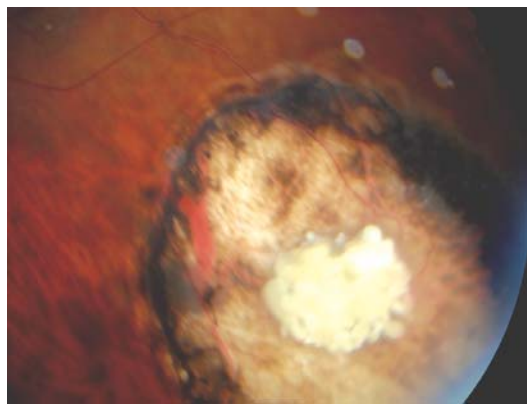


FIGURE 12.2.3: Retinal astrocytoma

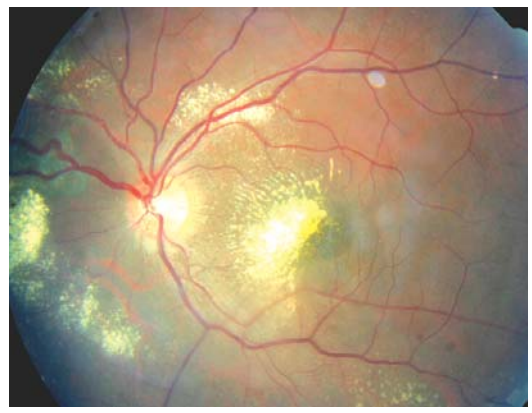


FIGURE 12.2.4: Angiomatosis retinae

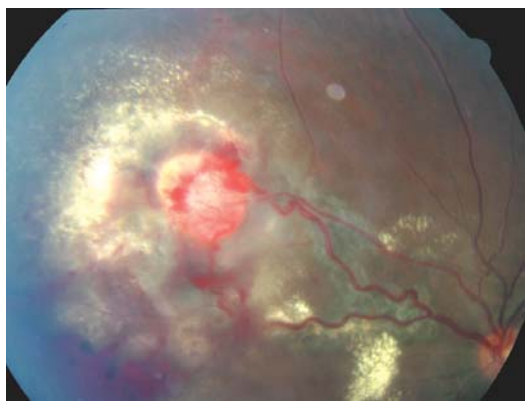


FIGURE 12.2.5: Angiomatosis retinae

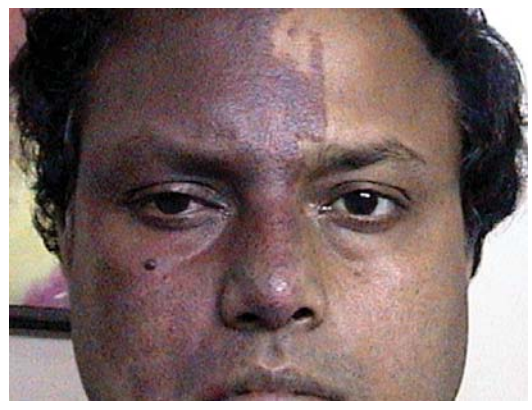


FIGURE 12.2.6: Sturge-Weber syndrome

Albinism

- Pink fundal glow **(Fig 12.3.1)**
- Generalized pallor of the fundus with visible choroidal vasculature **(Fig 12.3.2)**
- Positive iris transillumination
- For futher details ***See Chapter: 5***

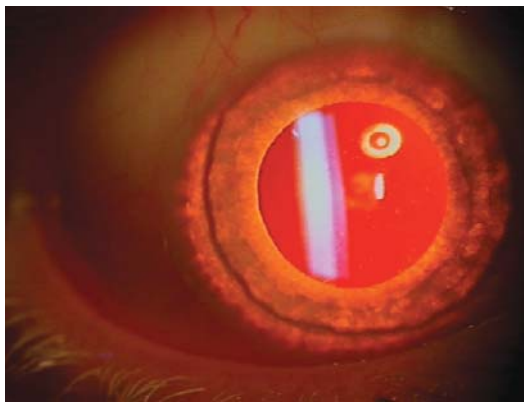


FIGURE 12.3.1: Albinism—pink fundal glow

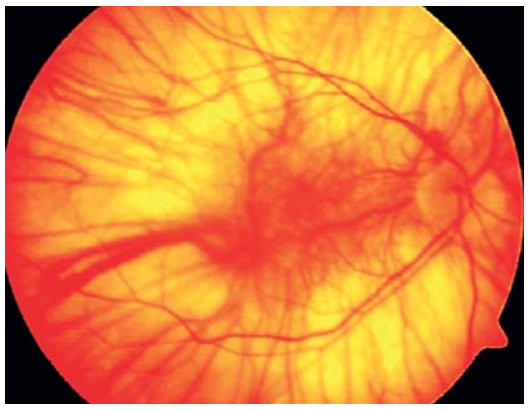


FIGURE 12.3.2: Albino fundus—generalized pallor

Retinochoroidal Coloboma

- Large oval, or semicircular sharply-defined white area inferior to the optic disk **(Fig 12.4.1)**
- Sometimes, it may include the disk
- May appear as isolated coloboma with a bridge of normal retinochoroidal tissue in between, giving rise to "double disk" **(Fig 12.4.2)** or "triple disk" **(Fig 12.4.3)** appearance
- May be associated with total coloboma

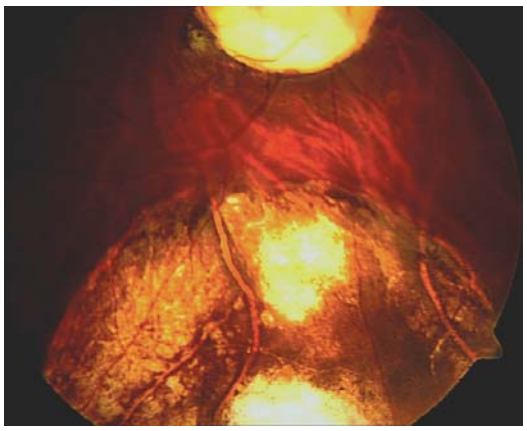


FIGURE 12.4.1: Retinochoroidal coloboma

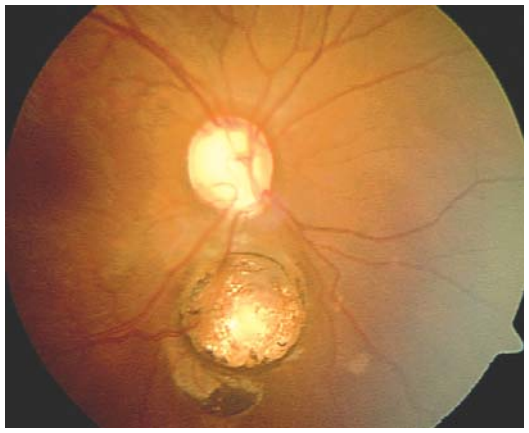


FIGURE 12.4.2: Retinochoroidal isolated coloboma—'double disk' appearance

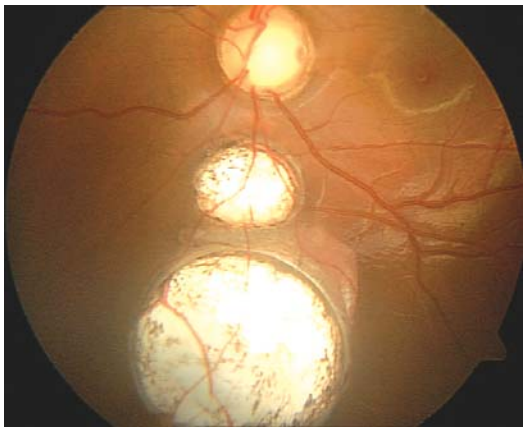


FIGURE 12.4.3: Retinochoroidal isolated colobomas—'triple disk' appearance

RETINAL VASCULAR DISORDERS

Central Retinal Artery Occlusion (CRAO)

- Painless, sudden loss of vision
- Occlusion may affect the central retinal artery (CRAO) itself
- A peripheral branch (arteriole), when the effect is localized
- Retina loses its transparency, and becoming opaque and milky-white, especially around the posterior pole **(Fig 12.5.1)**
- A cherry-red spot appears at the fovea **(Figs 12.5.2 and 12.5.3)**

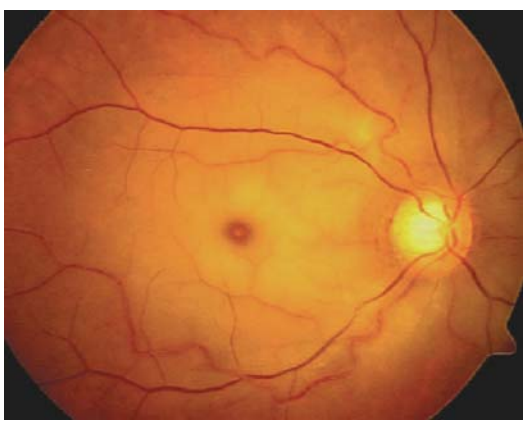


FIGURE 12.5.1: Central retinal artery occlusion

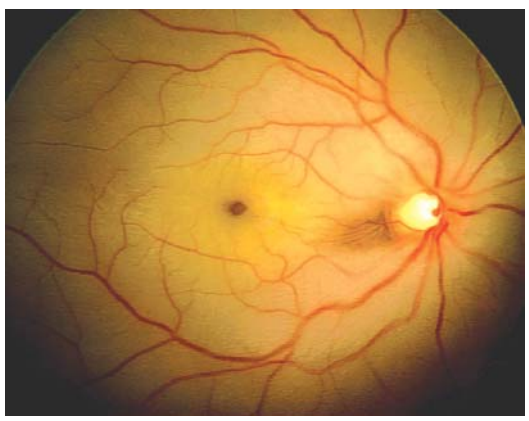


FIGURE 12.5.2: CRAO—cherry red spot

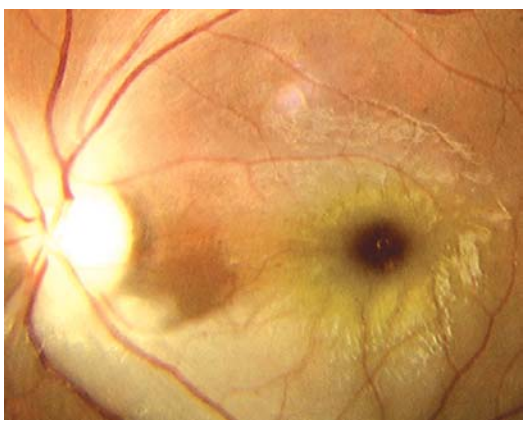


FIGURE 12.5.3: Cherry red spot

- 'Cattle-track' appearance in incomplete obstruction
- Branch occlusion occurs at a bifurcation and usually by an embolus **(Fig 12.5.4)**
- An atheromatous embolus may be visible as a refractile body (Hollenhorst plaque) within the artery **(Fig 12.5.5)**
- In presence cilio-retinal artery (25% cases), macula may often spared **(Figs 12.5.6 and 12.5.7)**
- After a week or so retina appears normal with optic atrophy with pale disk **(Fig 12.5.8)**
- *Treatment:* emergency digital massage, paracentesis, I/V acetazolamide, CO_2 inhalation, etc.
- Prognosis is invariably poor, if the obstruction is more than 6 hours

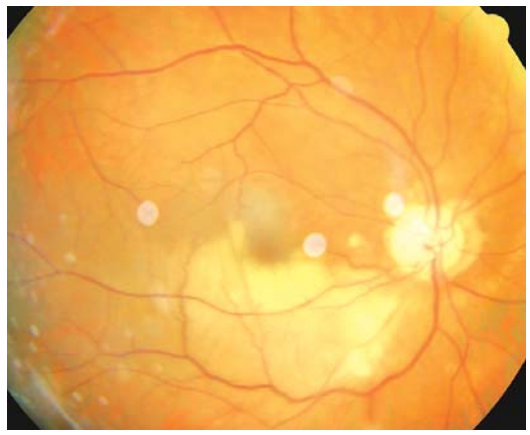


FIGURE 12.5.4: Branch retinal artery occlusion

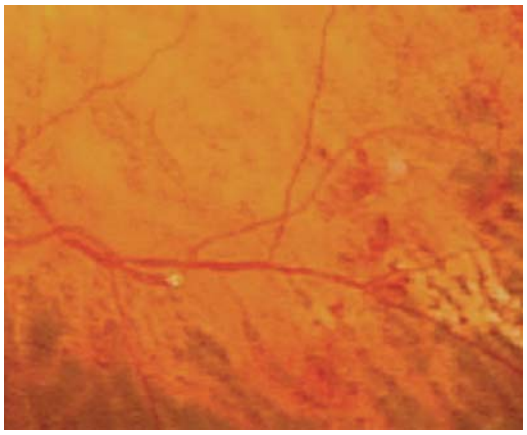


FIGURE 12.5.5: Hollenhorst plaque

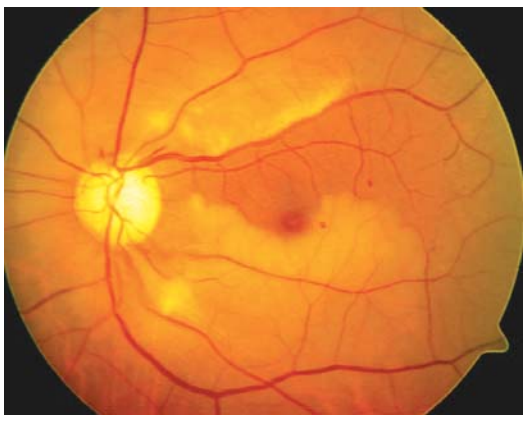


FIGURE 12.5.6: CRAO—patent cilioretinal

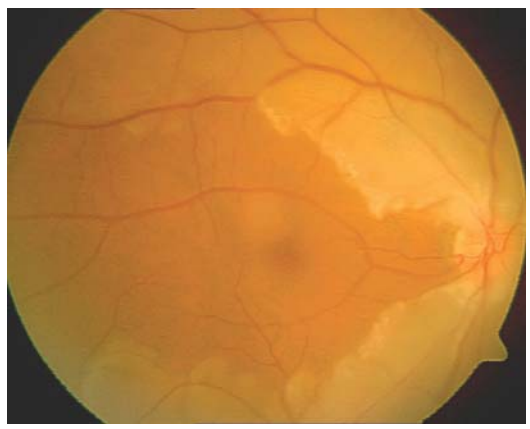


FIGURE 12.5.7: CRAO—patent cilioretinal

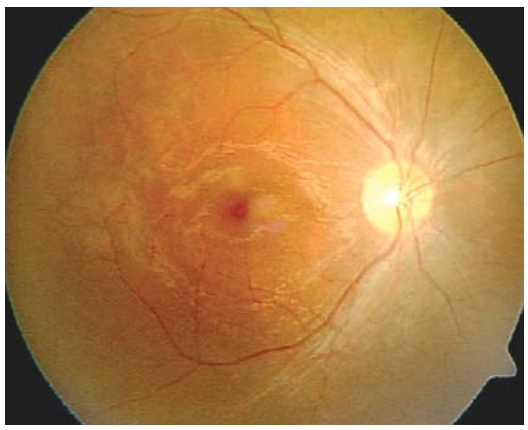


FIGURE 12.5.8: CRAO—optic atrophy

Central Retinal Vein Occlusion (CRVO)

- Obstruction of the central retinal vein, with sudden loss of vision
- Associated with systemic hypertension, diabetes, raised IOP, hypercellularity of blood, or central periphlebitis
- *Two types: Non-ischemic CRVO and ischemic CRVO*

Non-ischemic (CRVO)

- Mild tortuosity and dilatation of all branches of the central retinal vein **(Figs 12.6.1 to 12.6.4)**
- 'Dot' and 'blot'; and 'flame' shaped hemorrhages are seen throughout all four quadrants of the retina
- Sclerosed veins may be seen in old CRVO **(Fig 12.6.5)**
- *Treatment:* treatment of predisposing factors

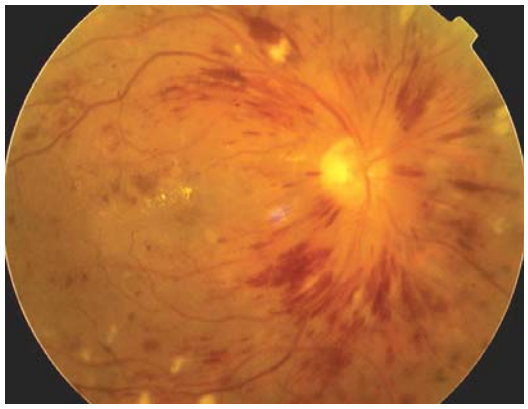


FIGURE 12.6.1: Non-ischemic CRVO

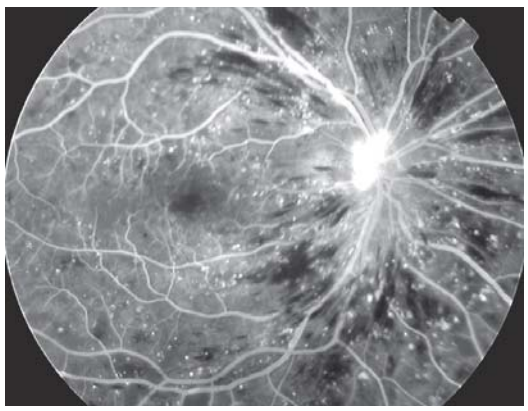


FIGURE 12.6.2: Non-ischemic CRVO

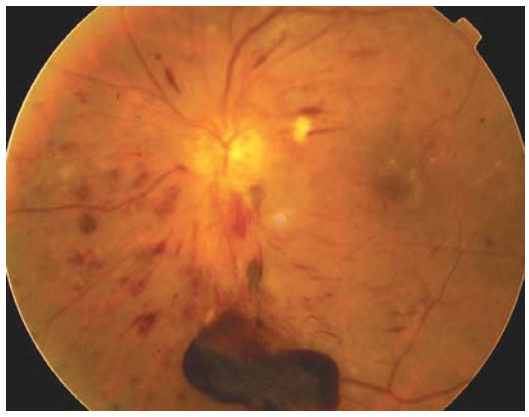


FIGURE 12.6.3: Non-ischemic CRVO—preretinal hemorrhage

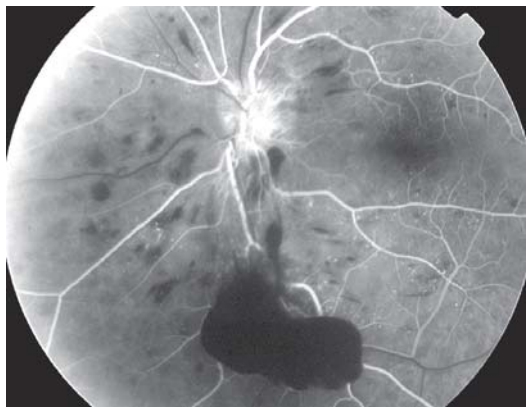


FIGURE 12.6.4: Non-ischemic CRVO—preretinal hemorrhage

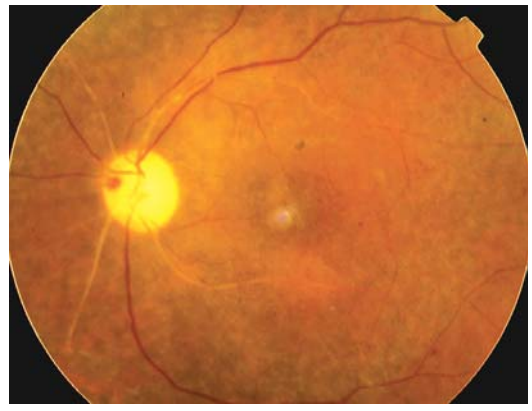


FIGURE 12.6.5: Old CRVO—sclerosed veins

Ischemic (CRVO)

- Marked tortuosity and engorgement of the retinal veins
- Massive superficial flame-shaped and deep blotchy hemorrhages throughout the fundus **(Figs 12.7.1 and 12.7.2)**
- Cotton-wool exudates are common
- Optic disk is swollen and hyperemic **(Fig 12.7.3)**
- Macular edema and hemorrhages
- Sometimes, called 'Blood and Thunder' fundus **(Fig 12.7.4)**
- *Treatment:* patient should be followed up closely to prevent rubeosis iridis and neovascular glaucoma

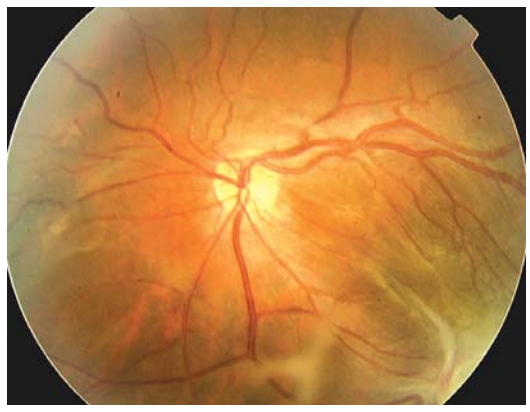


FIGURE 12.7.1: Ischemic CRVO

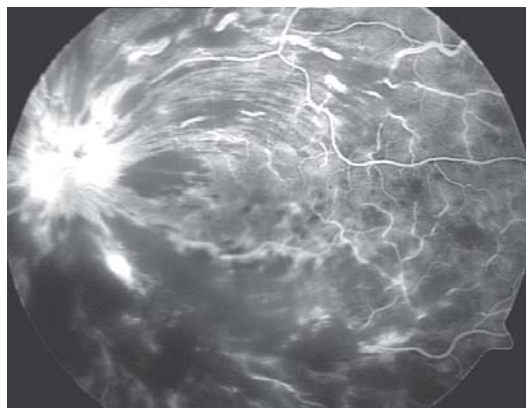


FIGURE 12.7.2: Ischemic CRVO

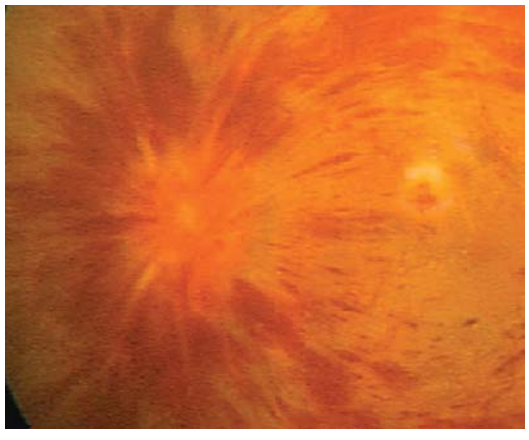


FIGURE 12.7.3: CRVO—disk edema

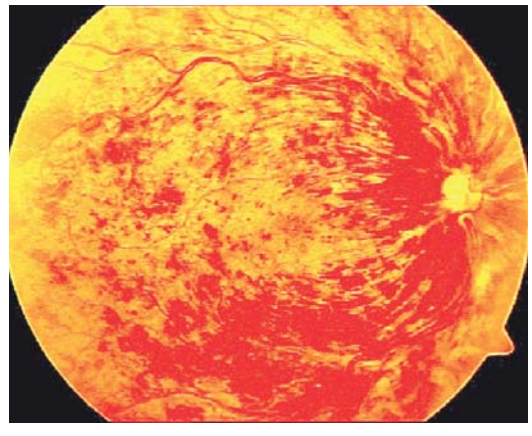


FIGURE 12.7.4: CRVO—blood and thunder fundus

Branch Retinal Vein Occlusion

- BRVO may occur near the optic disk and involves a major quadrant of the retina **(Figs 12.8.1 and 12.8.2)**

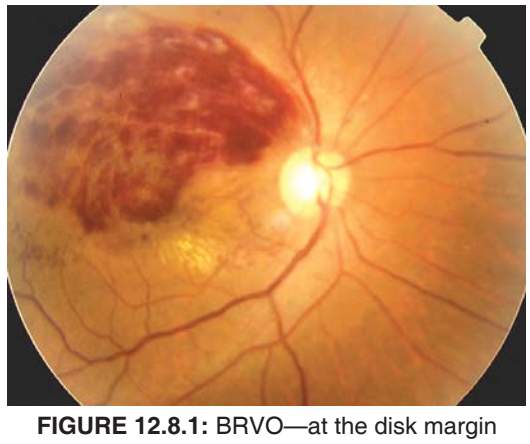


FIGURE 12.8.1: BRVO—at the disk margin

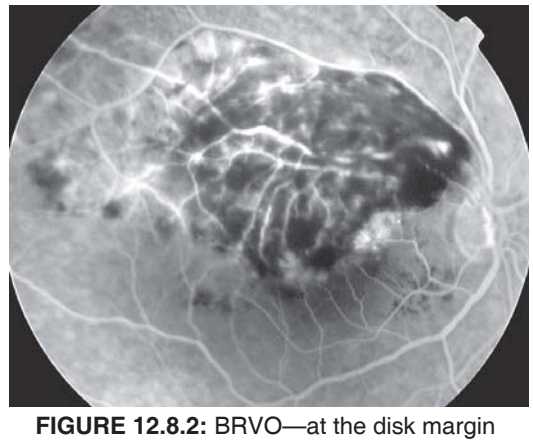


FIGURE 12.8.2: BRVO—at the disk margin

- Occur at a peripheral crossing with an artery **(Fig 12.8.3)**
- Blockage of the superior temporal vein frequently involves the macula **(Fig 12.8.4)**
- Even with inferior temporal vein, macula may also involved **(Fig 12.8.5)** which may be bilateral in rare situation **(Figs 12.8.6 and 12.8.7)**
- Ophthalmoscopically, the affected part of the retina, drained by the obstructed vein shows:
  - dilated and tortuous veins
  - flame-shaped, and 'dot' and 'blot' hemorrhage in the affected quadrant.
  - edema and cotton-wool spots
- Sometimes there is *hemiretinal venous occlusion*, involving the upper half or lower half of retina **(Figs 12.8.8 to 12.8.10)**
- There may be *tributary venous occlusion* **(Figs 12.8.11 to 12.8.13)**
- In case of old BRVO their may be sclerosis of blood vessels with formation of collaterals **(Fig 12.8.14)** and sometimes epiretinal fibrovascular membrane formation
- *Treatment:* fundus fluorescein angiography (FFA) to know the macular perfusion status and later macular grid photocoagulation or to treat neovascularization

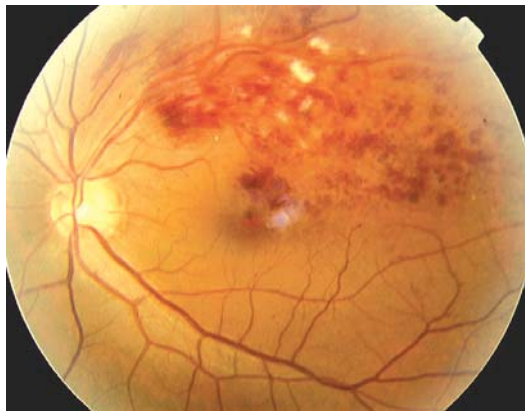


FIGURE 12.8.3: BRVO—at the AV crossing

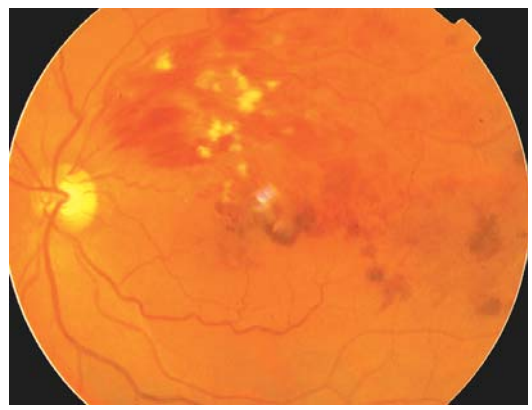


FIGURE 12.8.4: ST BRVO—macular involvement

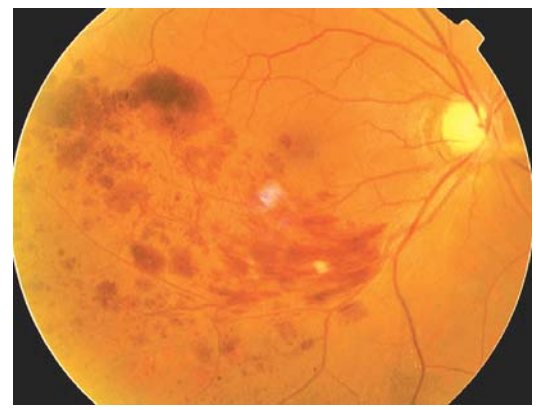


FIGURE 12.8.5: IT BRVO—macular involvement

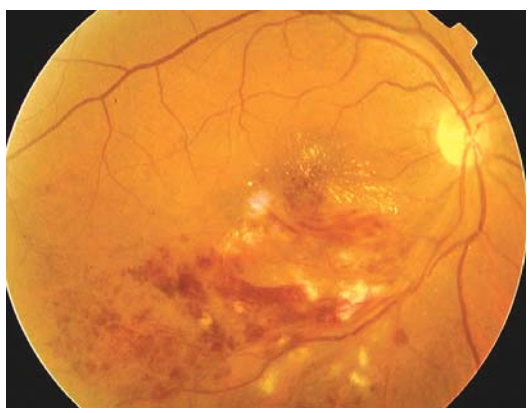


FIGURE 12.8.6: Bilateral inferior temporal BRVO

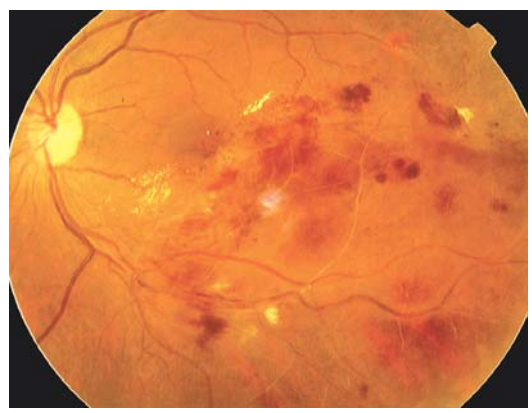


FIGURE 12.8.7: Bilateral inferior temporal BRVO

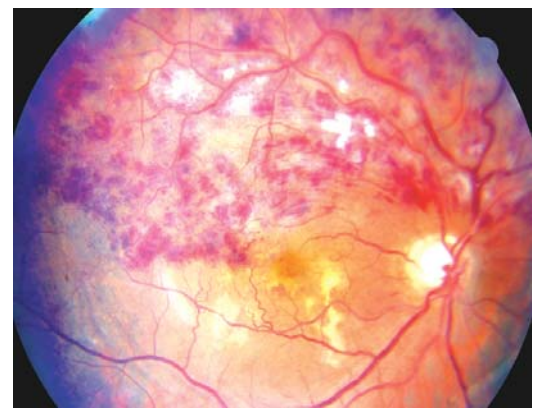


FIGURE 12.8.8: Hemiretinal venous occlusion

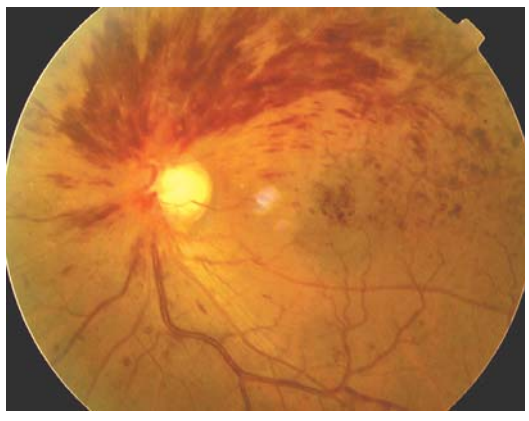


FIGURE 12.8.9: Hemiretinal venous occlusion

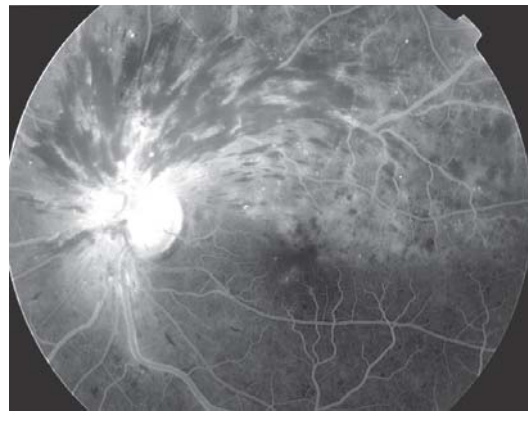


FIGURE 12.8.10: Hemiretinal venous occlusion

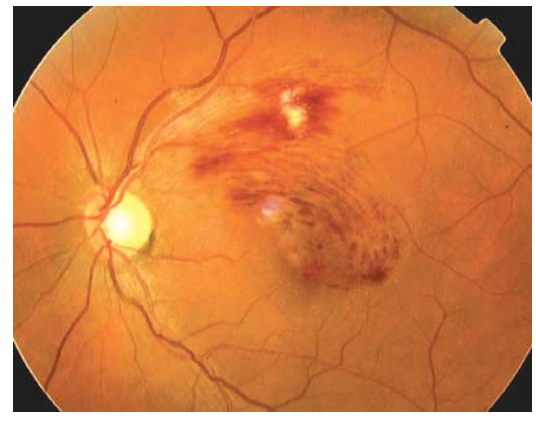


FIGURE 12.8.11: ST BRVO—tributary

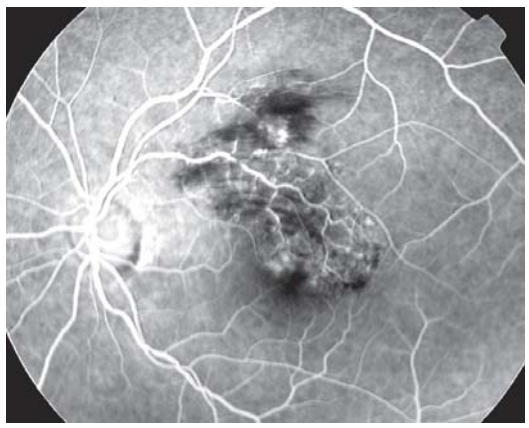


FIGURE 12.8.12: ST BRVO—macular involvement

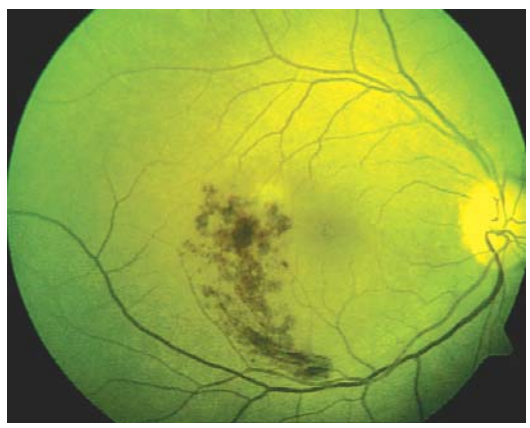


FIGURE 12.8.13: Tributary BRVO—IT

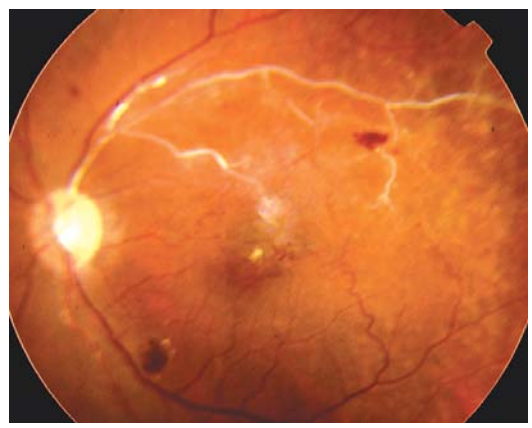


FIGURE 12.8.14: ST BRVO— sclerosed vessels

Preretinal Hemorrhage

- Usually solitary and located at the posterior pole obscuring the visualization of underlying retinal vessels **(Figs 12.9.1 and 12.9.2)**
- Initially round **(Fig 12.9.3)**, but later turn into *boat-shaped hemorrhage* due to settling by gravity **(Fig 12.9.4)**
- Large hemorrhage may break into the vitreous cavity spontaneously
- Absorption occurs from the top with yellow-white diskoloration **(Figs 12.9.5 and 12.9.6)**
- Fundus fluorescein angiogram (FFA) reveals blocking of retinal fluorescence **(Figs 12.9.7 and 12.9.8)**
- *Causes:* trauma, Valsalva retinopathy, proliferative retinopathies, Terson's syndrome (with subarachnoid hemorrhage)
- *Treatment:* laser hyaloidotomy to drain the blood into the vitreous cavity **(Figs 12.9.9 and 12.9.10)**

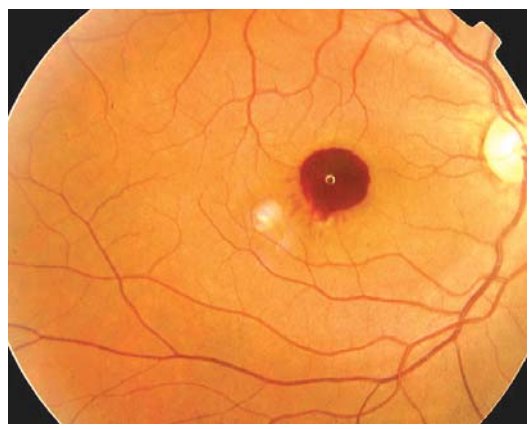


FIGURE 12.9.1: Preretinal hemorrhage

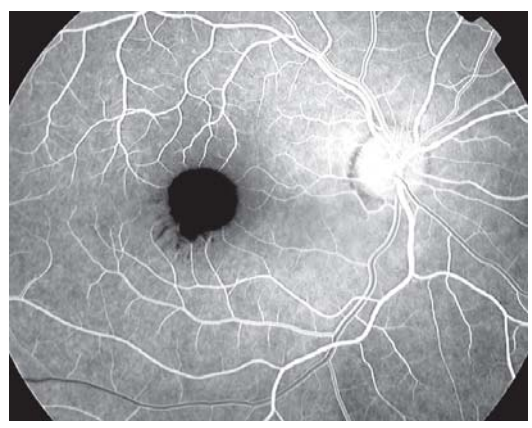


FIGURE 12.9.2: Preretinal hemorrhage

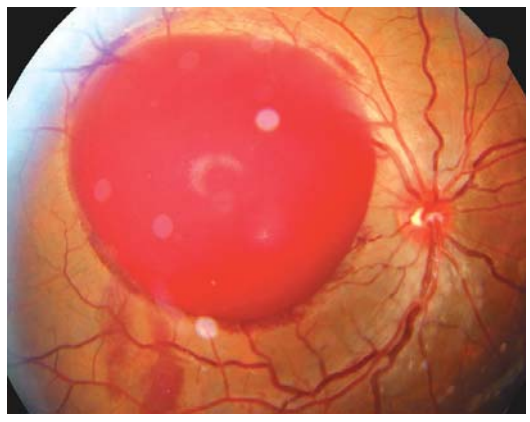


FIGURE 12.9.3: Preretinal hemorrhage1

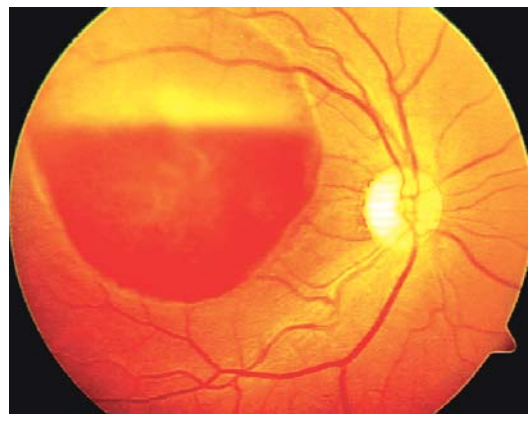


FIGURE 12.9.4: Preretinal hemorrhage

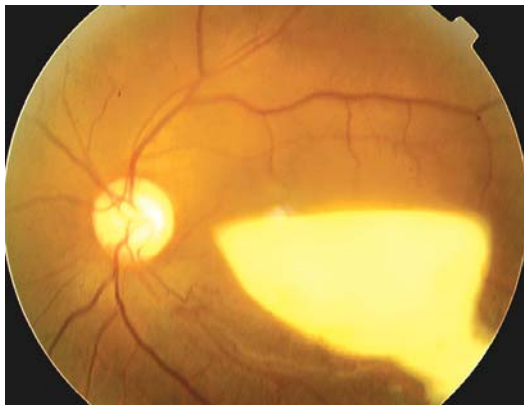


FIGURE 12.9.5: Preretinal hemorrhage—absorbing

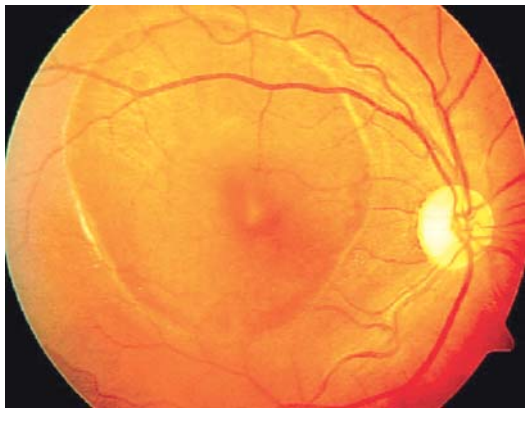


FIGURE 12.9.6: Preretinal hemorrhage—absorbed

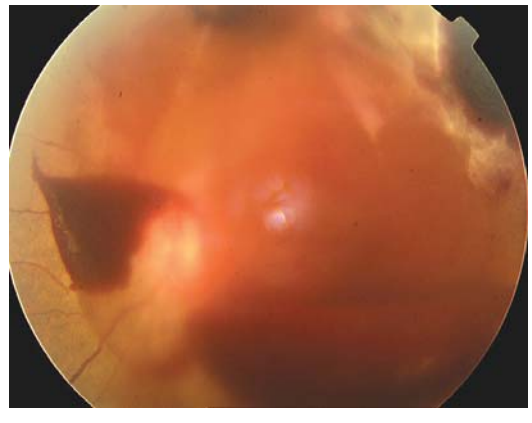


FIGURE 12.9.7: Preretinal hemorrhage—large

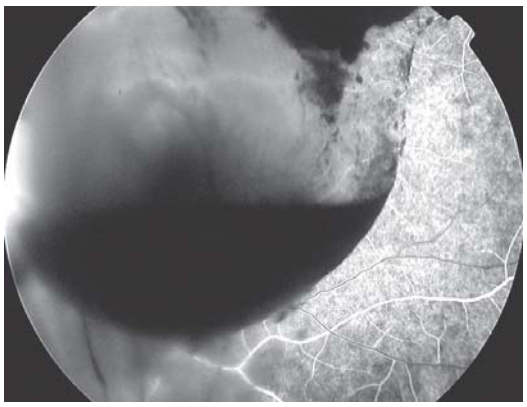


FIGURE 12.9.8: Preretinal hemorrhage—FFA

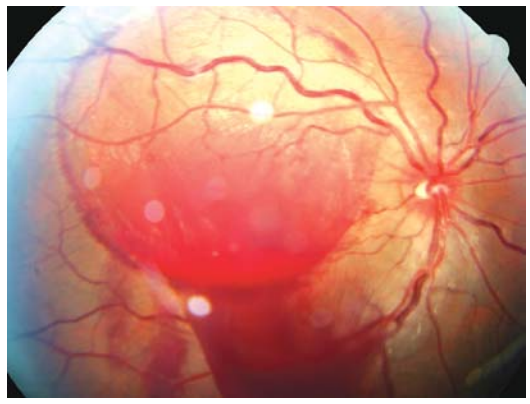


FIGURE 12.9.9: Preretinal hemorrhage—YAG hyaliodotomy

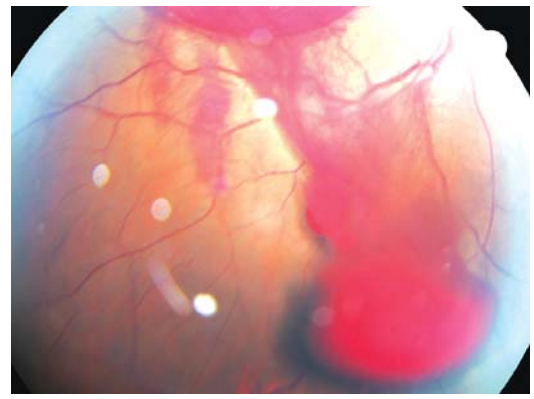


FIGURE 12.9.10: Preretinal hemorrhage—YAG hyaliodotomy

Roth Spot

- Retinal hemorrhage with white center
- *Causes:* leukemia **(Fig 12.10.1)**, severe anemia **(Figs 12.10.2 and 12.10.3)**, dysproteinemias, subacute bacterial endocarditis, HIV retinopathy

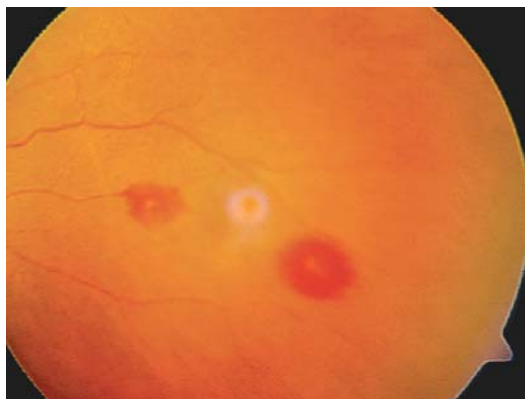


FIGURE 12.10.1: Roth spot—leukemia

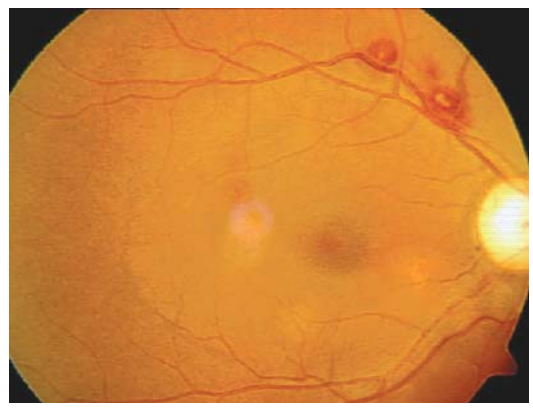


FIGURE 12.10.2: Roth spot—severe anemia

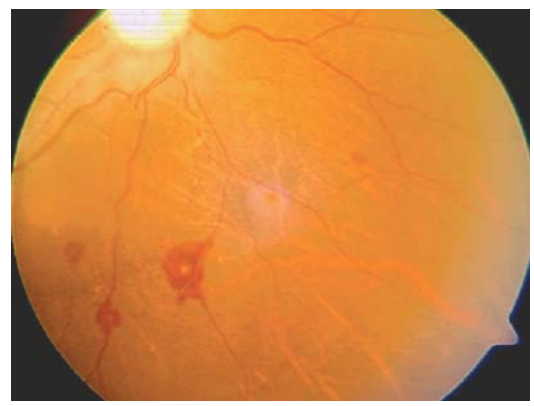


FIGURE 12.10.3: Roth spot—severe anemia

Subretinal Hemorrhage

- Blood is localized between the photoreceptors layer and retinal pigment epithelium (RPE) layer
- Large, bright red area with indistinct outline **(Fig 12.11.1)** and the overlying retina is slightly elevated with visible retinal blood vessels **(Figs 12.11.2 and 12.11.3)**
- May be associated with sub-RPE hemorrhage in some cases
- *Causes:* blunt trauma, choroidal neovascularization, ruptured retinal macroaneurysm, etc.

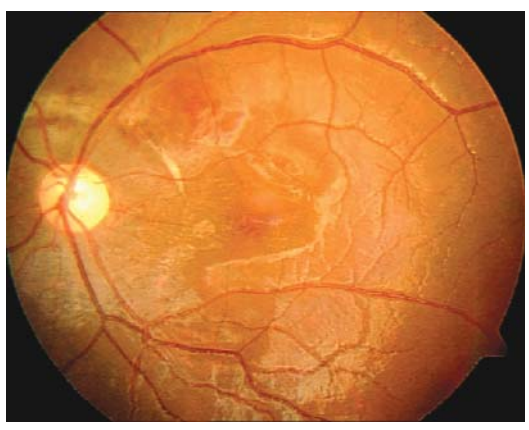


FIGURE 12.11.1: Subretinal hemorrhage—post traumatic

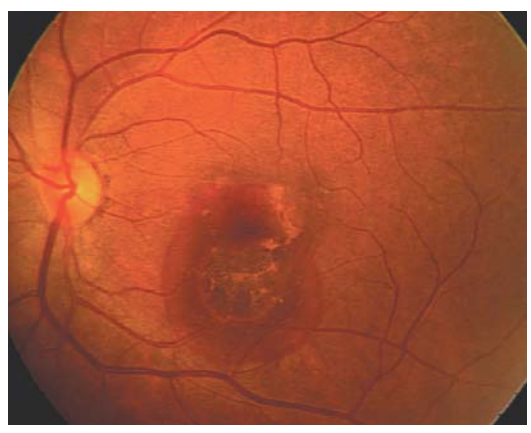


FIGURE 12.11.2: Subretinal hemorrhage

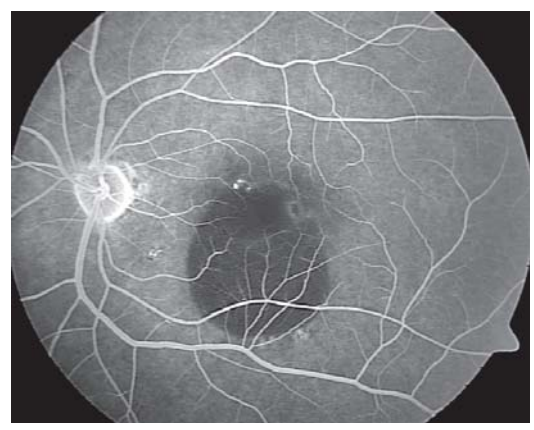


FIGURE 12.11.3: Subretinal hemorrhage—FFA

Sub-RPE Hemorrhage

- Blood from choroid seeps into the space between the Bruch's membrane and RPE
- Solitary, usually very dark-red with well defined outline **(Figs 12.12.1 and 12.12.2)**
- May be multiple in some cases **(Figs 12.12.3 and 12.12.4)**
- FFA shows blocked background choroidal fluorescence
- *Commonest cause:* subretinal choroidal neovascular membranes (SRNVMs)

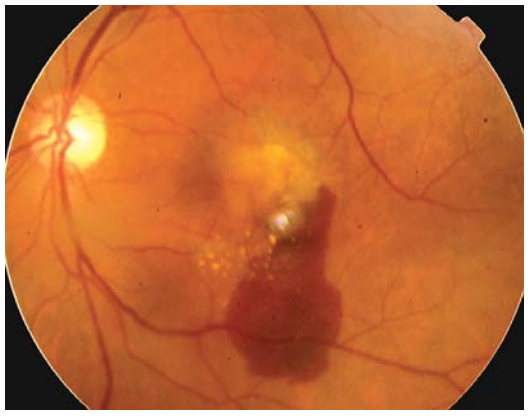


FIGURE 12.12.1: Sub-RPE hemorrhage

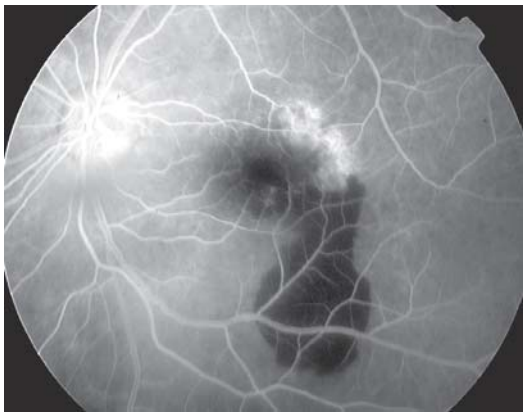


FIGURE 12.12.2: Sub-RPE hemorrhage

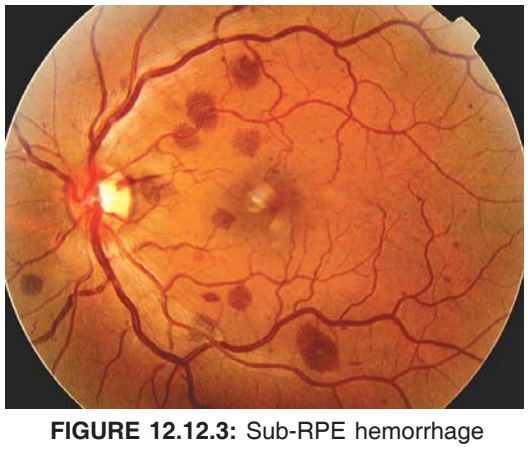


FIGURE 12.12.3: Sub-RPE hemorrhage

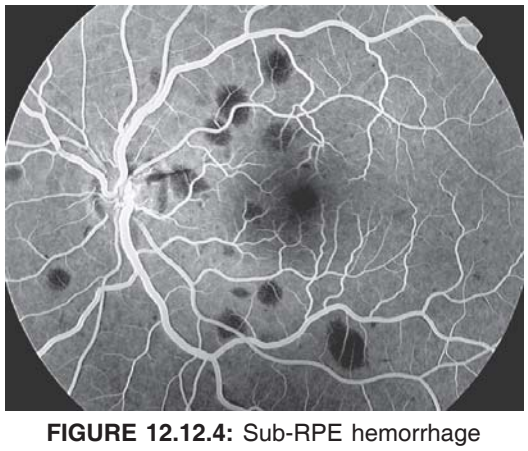


FIGURE 12.12.4: Sub-RPE hemorrhage

Choroidal Hemorrhage

- Dark-red, almost black, with well defined outline **(Figs 12.13.1 and 12.13.2)**
- *Causes:* blunt trauma, often associated with choroidal tear; during drainage of subretinal fluid
- In cases of severe trauma, there may be combination of subretinal sub-RPE and choroidal hemorrhage (**Fig 12.13.3)**

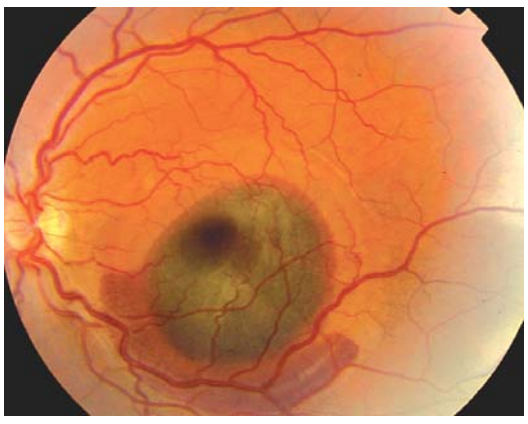


FIGURE 12.13.1: Choroidal hemorrhage

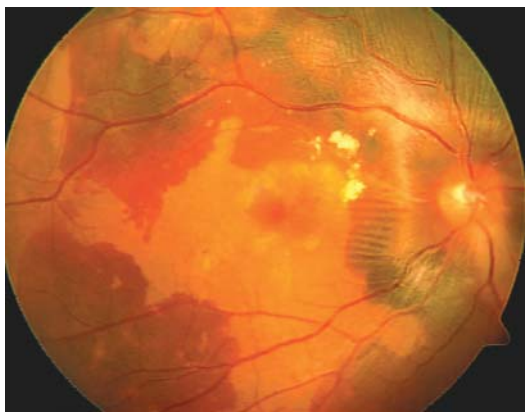


FIGURE 12.13.2: Choroidal hemorrhage—with RD

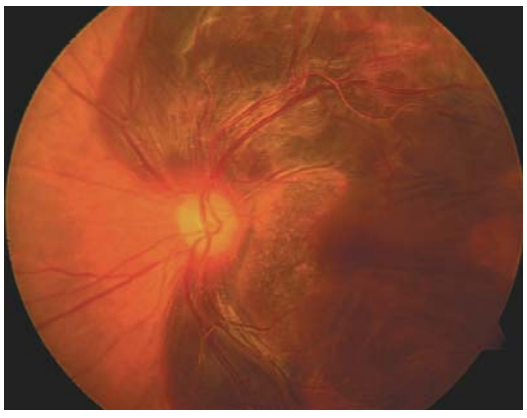


FIGURE 12.13.3: Extensive subretinal, sub-RPE and choroidal hemorrhage in trauma

Neovascularization

- May involve the optic nerve head, called neovascularization disk (NVD) **(Figs 12.14.1 and 12.14.2)** and when it involves central retina, called neovascularization elsewhere (NVE) **(Figs 12.14.3 and 12.14.4)**
- Both NVD and NVE may be seen in the same eye (**Fig 12.14.5**)
- May involve peripheral retina and takes the configuration of "sea fan" **(Figs 12.14.6 to 12.14.8)**
- New vessels often bleed, resulting retinal or vitreous hemorrhage
- New vessels may be flat, elevated or mixed and associated with variable degree of fibrosis
- When fibrous tissue contracts, it may result tractional retinal detachment **(Figs 12.14.9 and 12.14.10)**
- *Causes:* PDR, CRVO or BRVO, retinal vasculitis, Eales' disease, ROP, sickle cell retinopathy, etc.

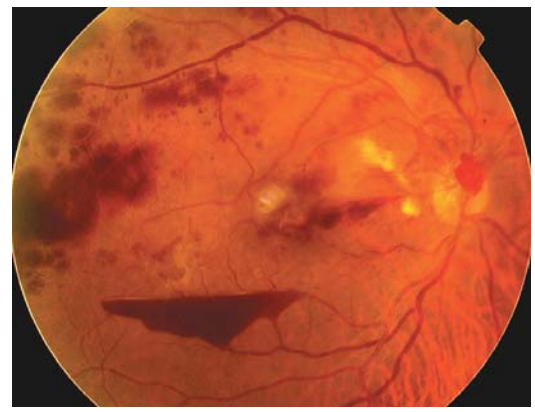


FIGURE 12.14.1: NVD—preretinal hemorrhage

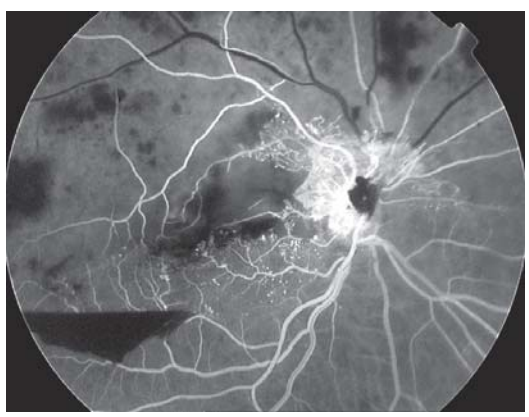


FIGURE 12.14.2: NVD—preretinal hemorrhage

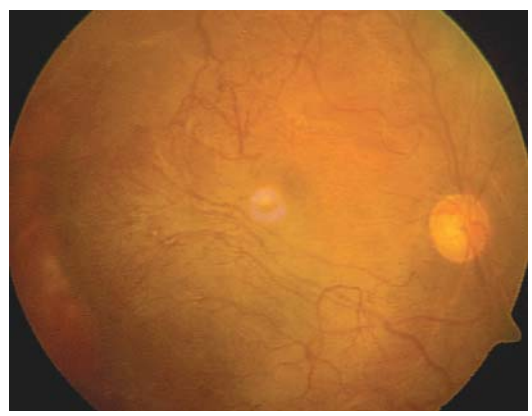


FIGURE 12.14.3: Extensive NVE

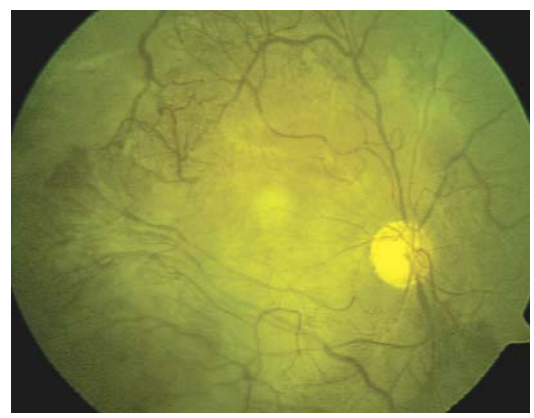


FIGURE 12.14.4: Extensive NVE

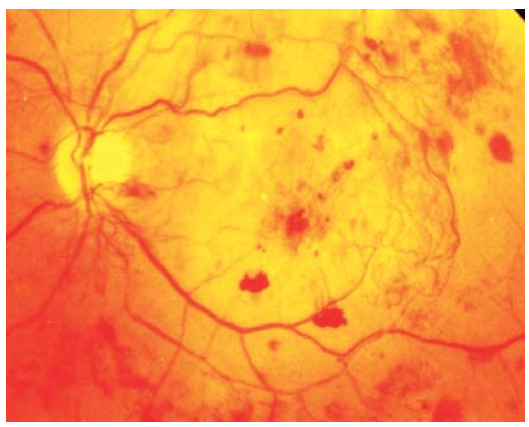


FIGURE 12.14.5: NVD and NVE—PDR

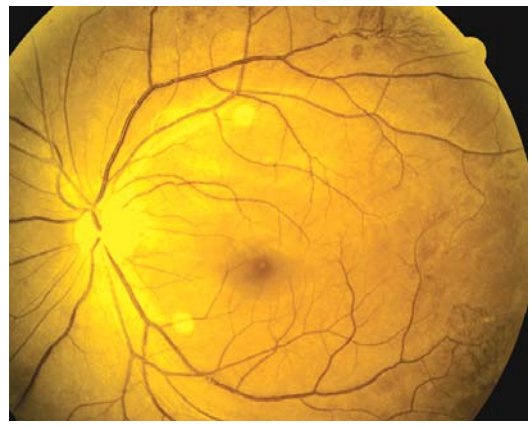


FIGURE 12.14.6: NVE—sea fan neovascularization

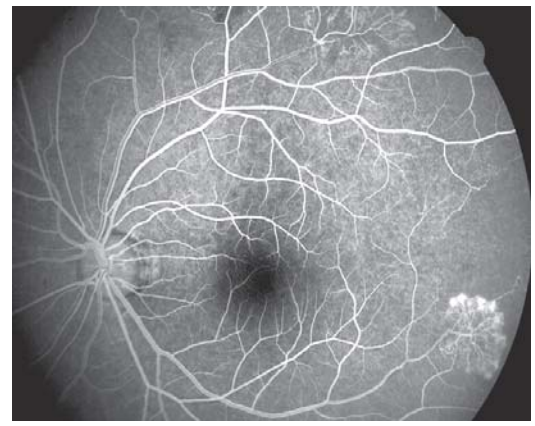


FIGURE 12.14.7: NVE—sea fan neovascularization

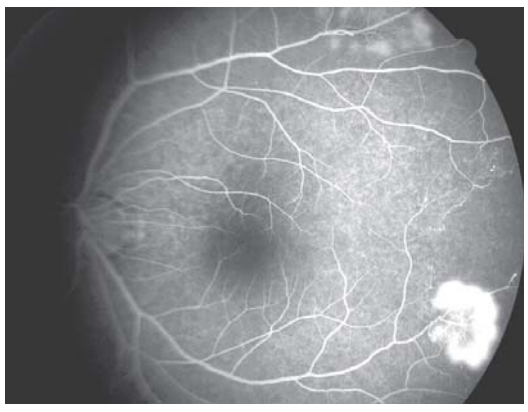


FIGURE 12.14.8: Sea fan neovascularization

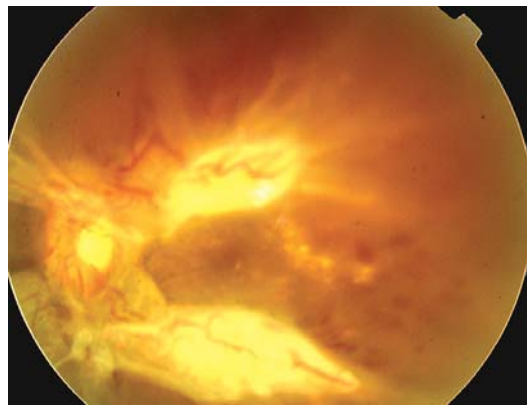


FIGURE 12.14.9: Neovascularization—tractional RD

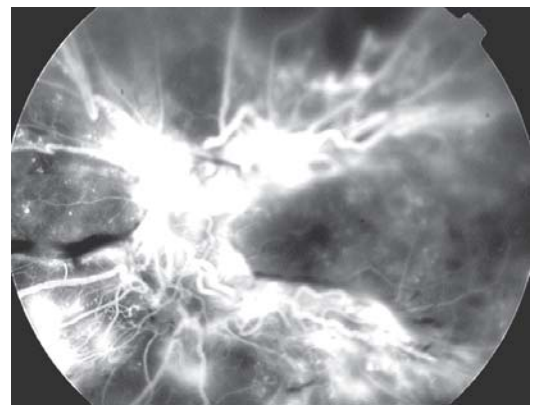


FIGURE 12.14.10: Neovascularization—tractional RD

Collaterals

- Are dilated pre-existing capillaries which occur in response to vascular insult **(Figs 12.15.1 and 12.15.2)**
- These shunts are acquired communication between:
  - *artery and vein,* as in diabetic retinopathy
  - *artery and artery*, as in BRVO
  - *vein and vein,* as in BRVO **(Figs 12.15.3 and 12.15.4)** or CRVO
- Larger in calibre than that of neovascularization and never cross the major retinal vessels
- Can occur in the disk **(Figs 12.15.5 and 12.15.6)** or elsewhere like neovascularization
- Collaterals *should not be lasered* as they are physiological
- Collaterals and neovascularization may exist together **(Figs 12.15.7 to 12.15.9)**

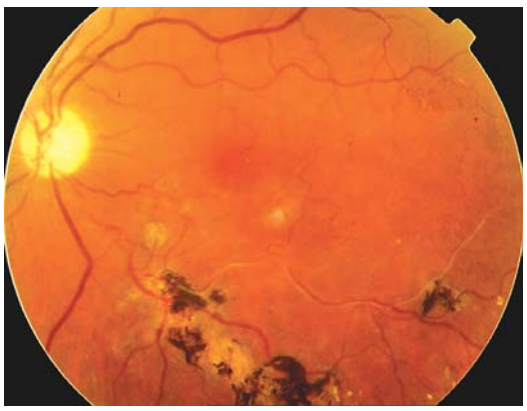


FIGURE 12.15.1: Collaterals

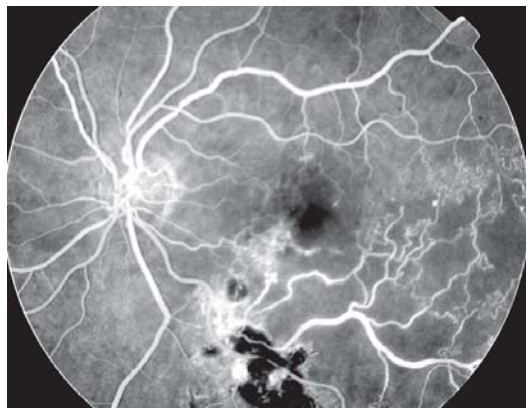


FIGURE 12.15.2: Collaterals

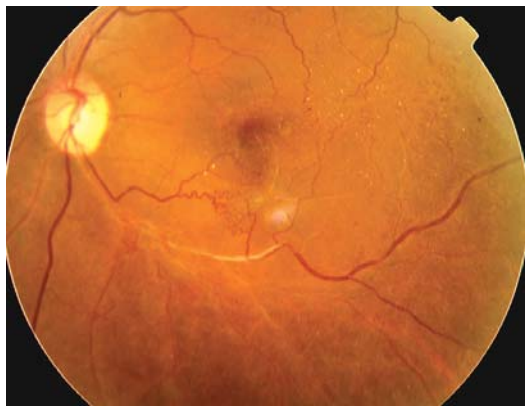


FIGURE 12.15.3: Collaterals—BRVO

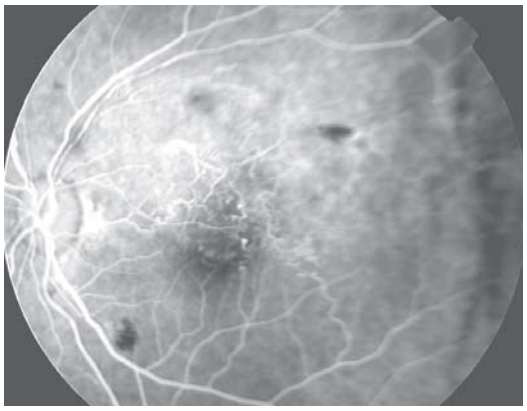


FIGURE 12.15.4: Collaterals—BRVO

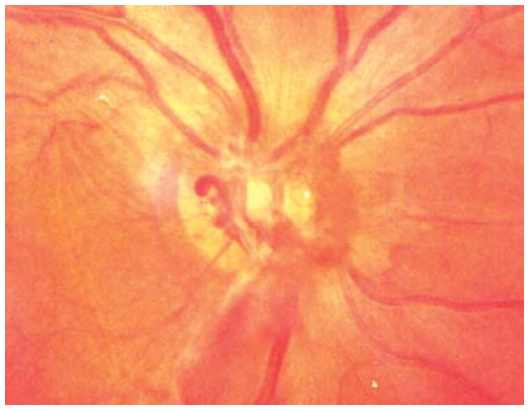


FIGURE 12.15.5: Collaterals—optic disk

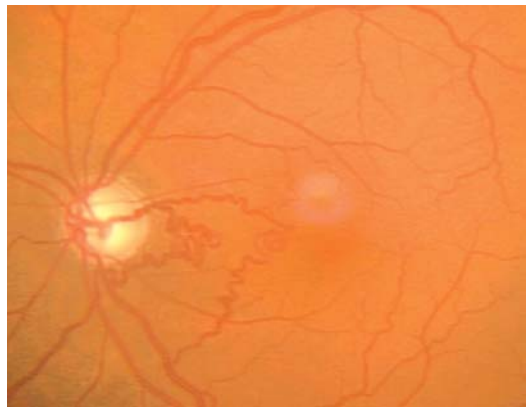


FIGURE 12.15.6: Collaterals—optic disk

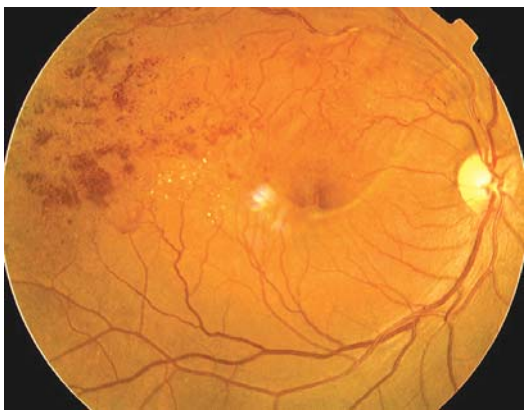


FIGURE 12.15.7: Collaterals and neovascularization

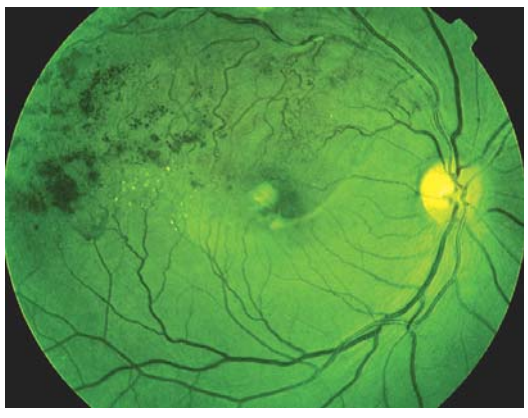


FIGURE 12.15.8: Collaterals and neovascularization

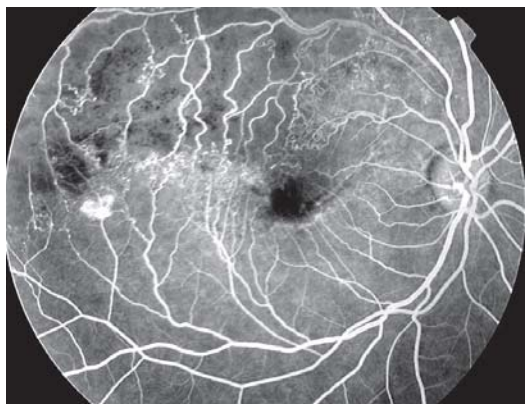


FIGURE 12.15.9: Collaterals and neovascularization

Retinal Hemangiomas

- Uncommon, often multiple, benign lesion
- May be associated with von Hippel-Lindau syndrome **(Figs 12.2.3 and 12.2.4, Fig 12.16.1)**
- Small to large orange-red nodular lesion associated with dilated and tortuous blood vessels **(Figs 12.16.2 and 12.16.3)**
- Often associated with hard exudates

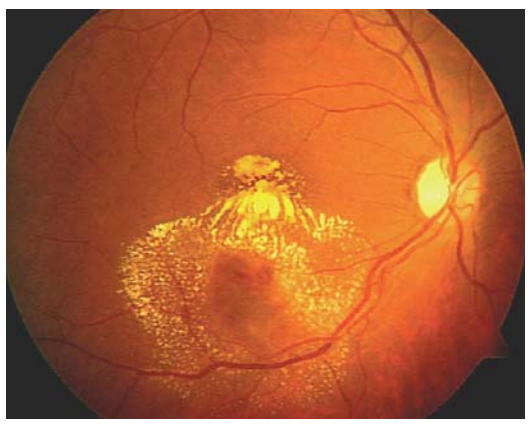


FIGURE 12.16.1: Retinal angioma

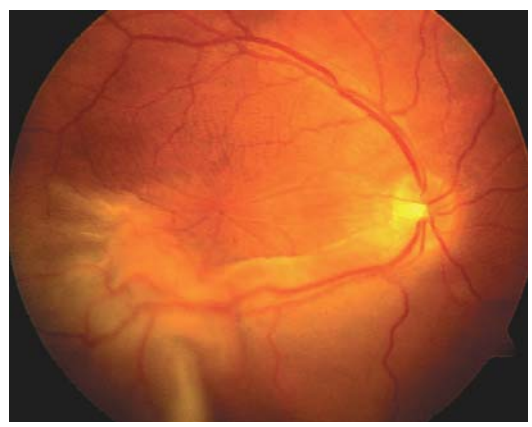


FIGURE 12.16.2: Retinal angioma—angiomatosis retinae

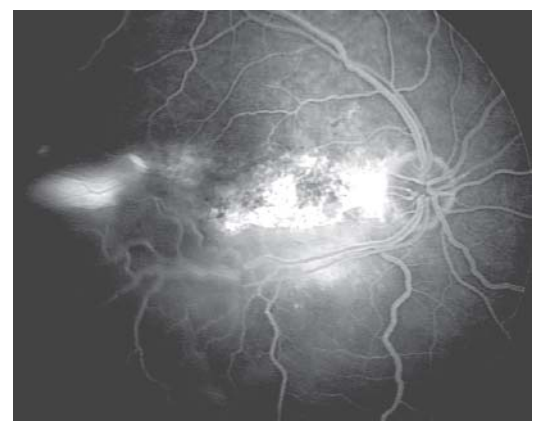


FIGURE 12.16.3: Retinal angioma—angiomatosis retinae

Retinal Vasculitis

- More commonly involves retinal vein (periphlebitis), rarely the arterioles (periorteritis)
- Active vasculitis appears as fluffy white perivascular cuffing better visible on FFA **(Figs 12.17.1 and 12.17.2)**
- Severe periphlebitis may give rise to CRVO or BRVO like pictures **(Figs 12.17.3 and 12.17.4)**
- Periphlebitis in sarcoidosis may appear as 'candle wax dripping' around the veins **(Figs 12.17.5 and 12.17.6)**
- Periarteritis may block a branch arteriole
- *Causes:*
  - *periphlebitis:* sarcoidosis, Eales' disease, intermediate uveitis, CMV retinitis, frosted branch angitis, etc.
  - *periarteritis:* collagen disorders like, SLE, polyarteritis nodosa, etc.

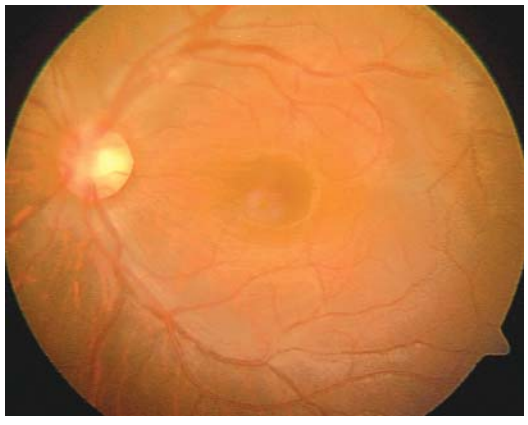


FIGURE 12.17.1: Vasculitis

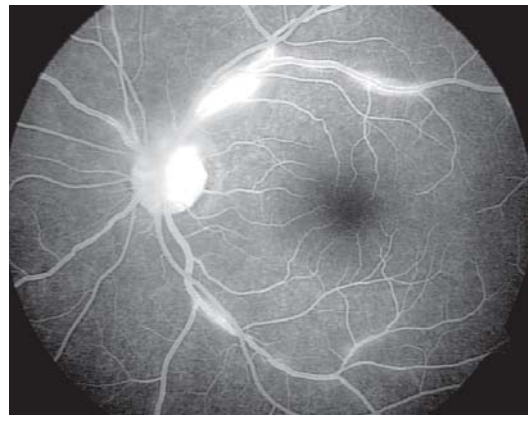


FIGURE 12.17.2: Vasculitis

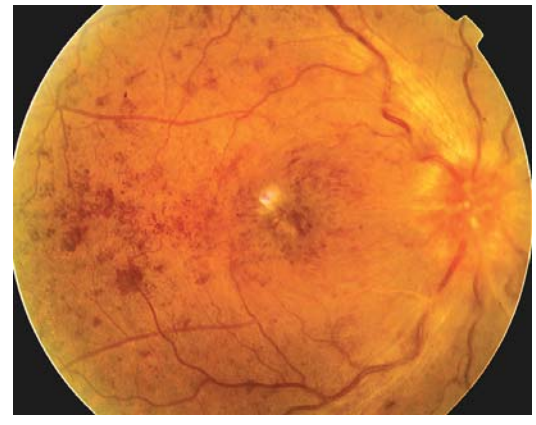


FIGURE 12.17.3: Vasculitis—CRVO

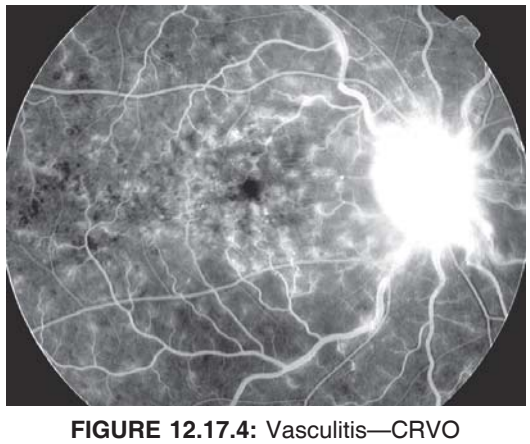


FIGURE 12.17.4: Vasculitis—CRVO

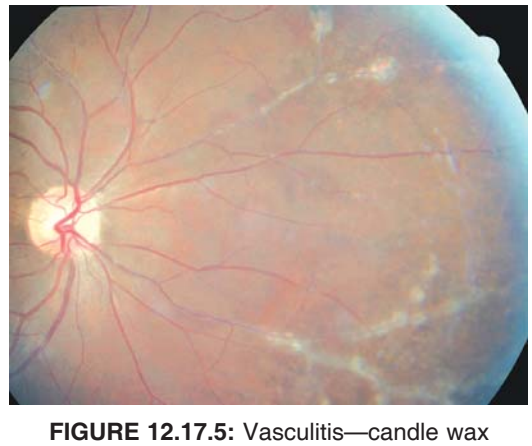


FIGURE 12.17.5: Vasculitis—candle wax dripping

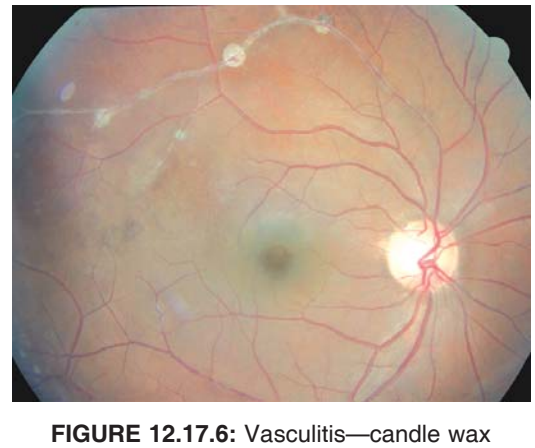


FIGURE 12.17.6: Vasculitis—candle wax dripping

Frosted Branch Angitis

- Uncommon, unilateral or bilateral acute inflammation of retinal vessels
- Sheathing of blood vessels with appearance of frosted branch of a tree **(Figs 12.18.1 and 12.18.2)**
- FFA findings confirm the diagnosis **(Figs 12.18.3 to 12.18.5)**
- May be associated with CMV retinitis **(Fig 12.18.6)**
- *Causes:* CMV retinitis, rubella, renal transplant patients on immunosuppressants, etc.

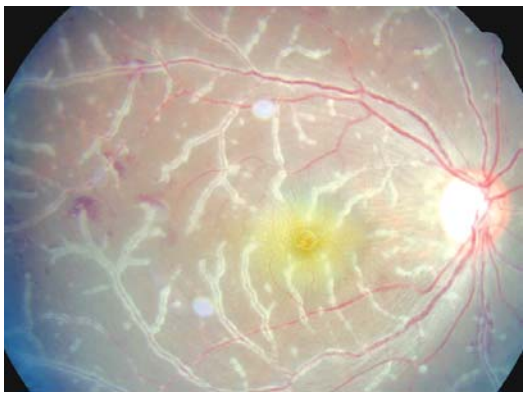


FIGURE 12.18.1: Frosted branch angitis

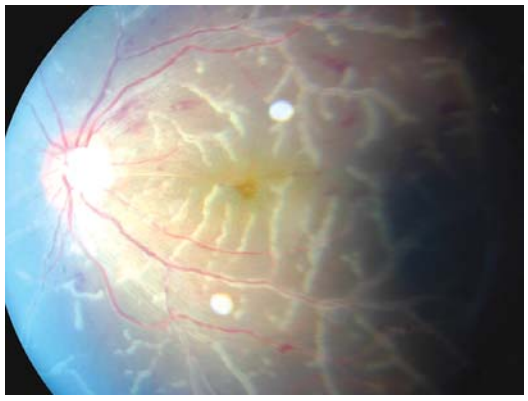


FIGURE 12.18.2: Frosted branch angitis

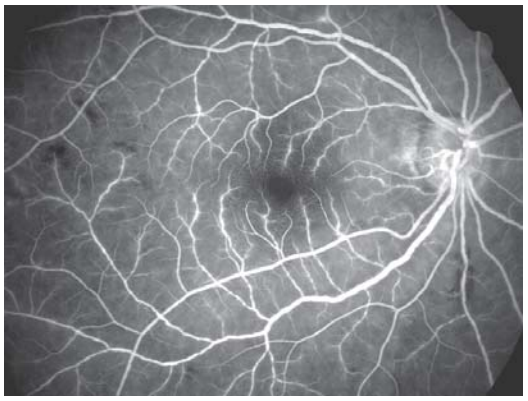


FIGURE 12.18.3: Frosted branch angitis—FFA

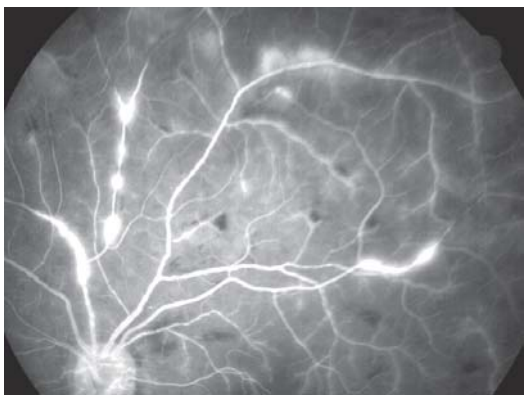


FIGURE 12.18.4: Frosted branch angitis—FFA

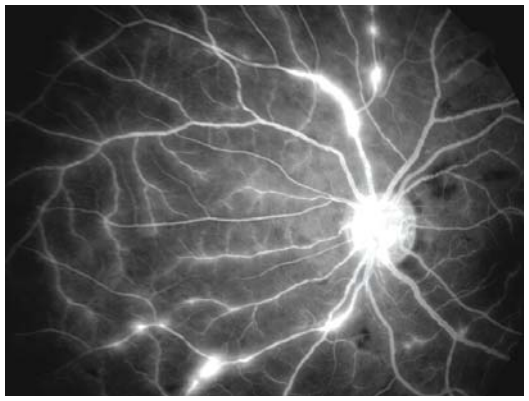


FIGURE 12.18.5: Frosted branch angitis—FFA

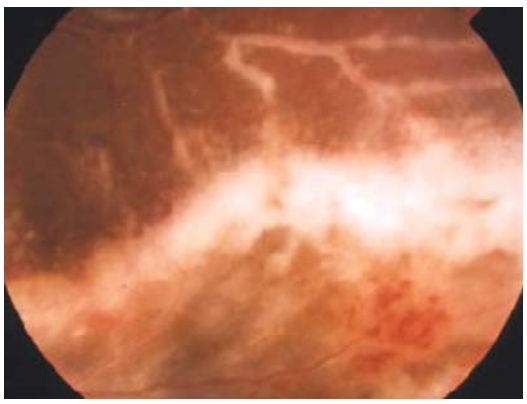


FIGURE 12.18.6: Frosted branch angitis

RETINAL EXUDATES

Hard Exudates

- Yellow-waxy deposits or plaques with fairly distinct margins
- Most frequently seen in the posterior pole
- Vary in size and configuration
  - *very few* **(Fig 12.19.1)**
  - *isolated clumps* **(Figs 12.19.2 and 12.19.3)**

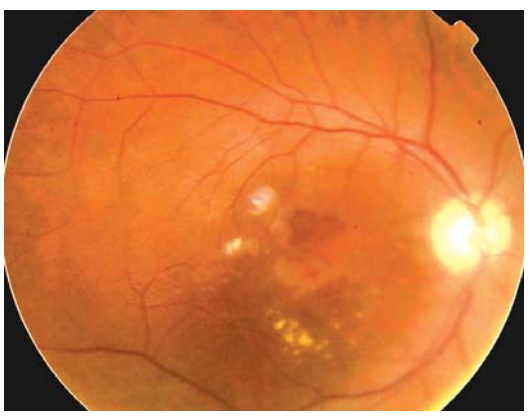


FIGURE 12.19.1: Hard exudates—very few

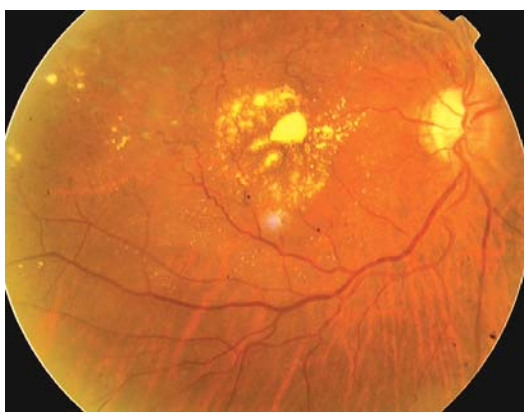


FIGURE 12.19.2: Hard exudates—isolated clumps

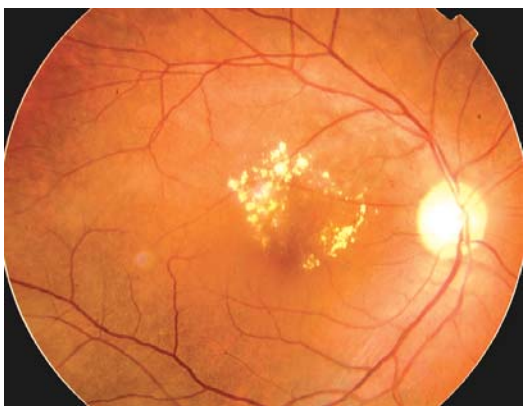


FIGURE 12.19.3: Hard exudates—isolated clumps

- *ring-shaped* **(Figs 12.19.4 and 12.19.5)**
- *circinate pattern* around a leaking capillary **(Figs 12.19.6 to 12.19.8)**
- *may be with subretinal exudates* **(Fig 12.19.9)**

• *Causes:* diabetic retinopathy, old BRVO, retinal macroaneurysm or hemangiomas

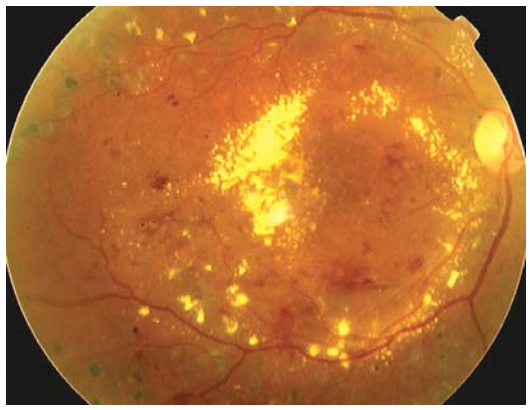


FIGURE 12.19.4: Hard exudates—ring-shaped

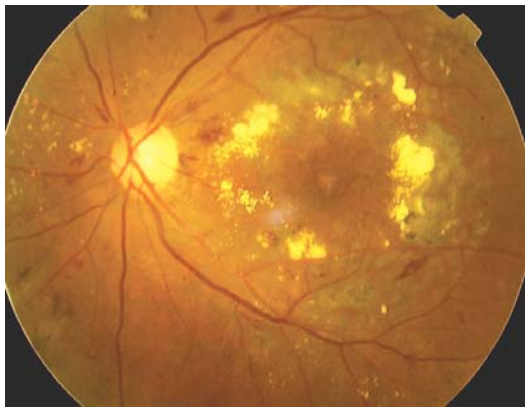


FIGURE 12.19.5: Hard exudates—ring-shaped

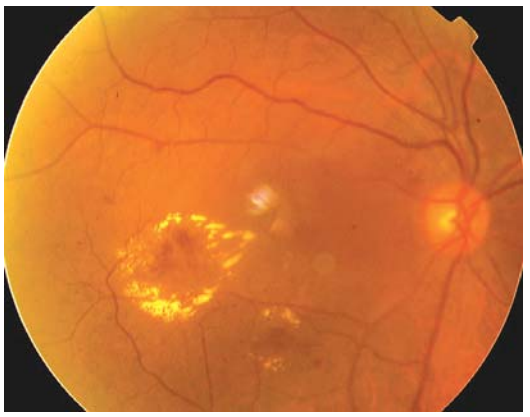


FIGURE 12.19.6: Hard exudate—circinate pattern

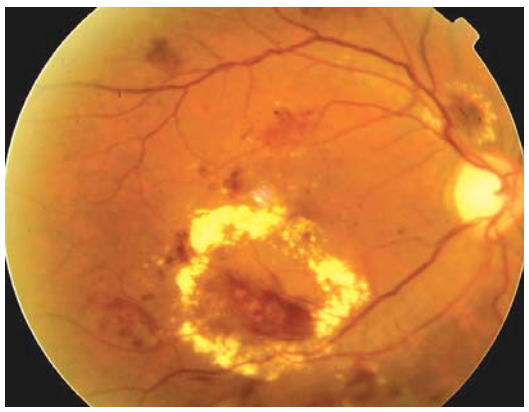


FIGURE 12.19.7: Hard exudate—circinate pattern

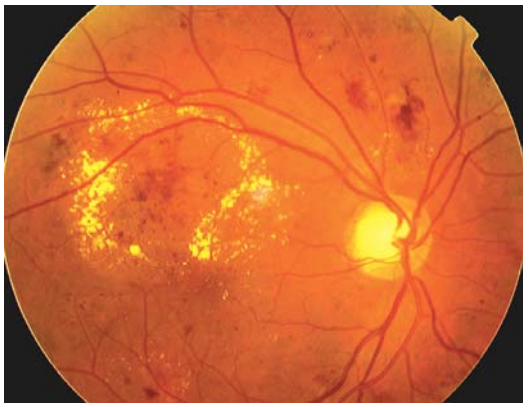


FIGURE 12.19.8: Hard exudates—circinate pattern

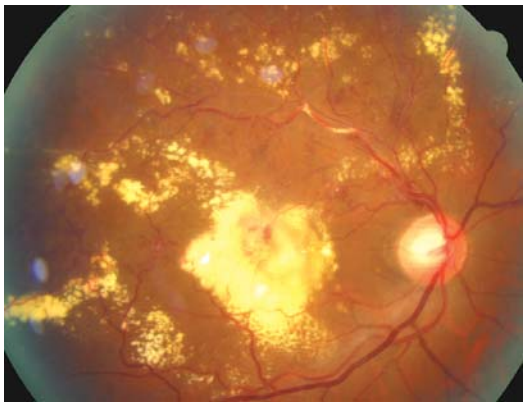


FIGURE 12.19.9: Hard exudates with subretinal exudates

Macular Stars

- Hard exudates around the macula often take the shape of a macular star
- May be incomplete **(Figs 12.20.1 to 12.20.3)** or complete **(Figs 12.20.4 and 12.20.5)**
- Sometimes, associated with optic disk swelling **(Fig 12.20.6)**
- *Causes:* papilledema, hypertension, neuroretinitis, peripapillary capillary angiomas

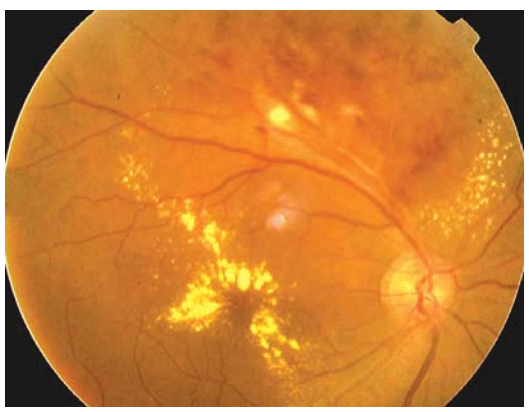


FIGURE 12.20.1: Macular star—incomplete

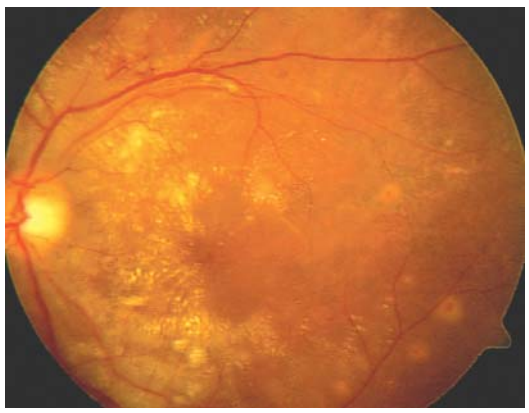


FIGURE 12.20.2: Macular star—incomplete

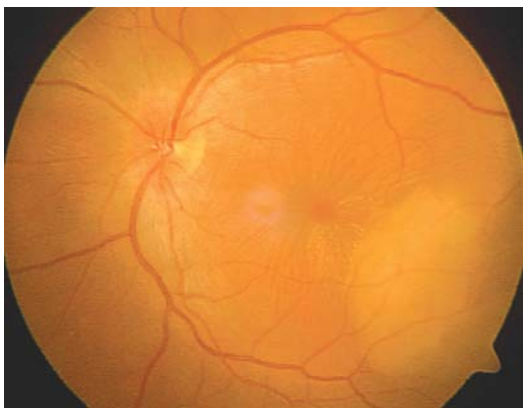


FIGURE 12.20.3: Macular star—incomplete

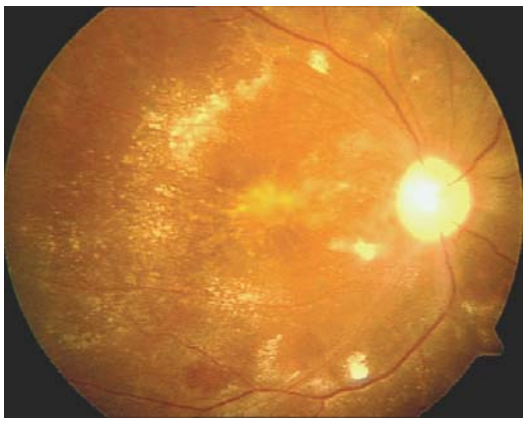


FIGURE 12.20.4: Macular star—complete

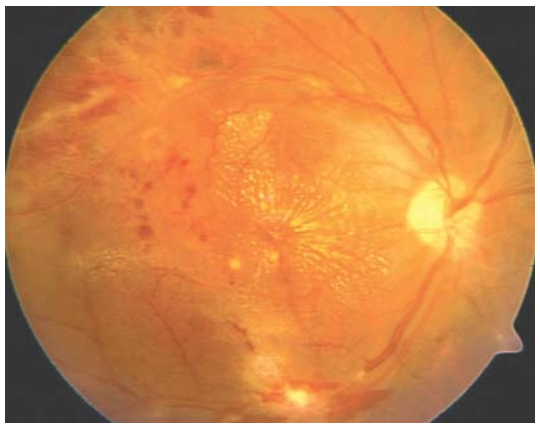


FIGURE 12.20.5: Macular star—complete—BRVO

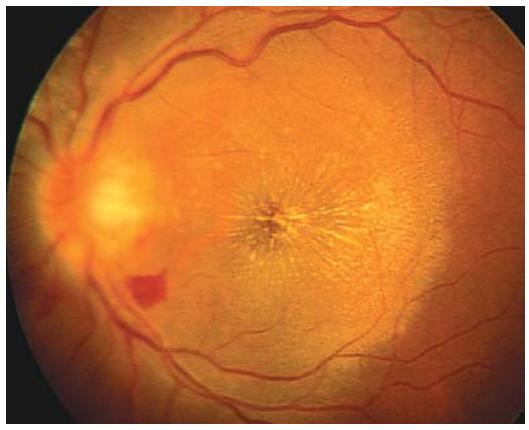


FIGURE 12.20.6: Macular star—complete—neuroretinitis

Soft Exudates or Cotton-Wool Spots

- White, cotton-wool like spots with indistinct margins **(Figs 12.21.1 and 12.21.2)**
- May obscure the underlying small blood vessels **(Figs 12.21.3 and 12.21.4)**
- Localised accumulation of axoplasmic debris in the nerve fiber layer as a result of ischemia
- May also be associated with hard exudates **(Fig 12.21.5)**
- *Causes:* CRVO or BRVO, hypertensive retinopathy, preproliferative diabetic retinopathy, HIV, microangiopathy, anemia, *etc.*

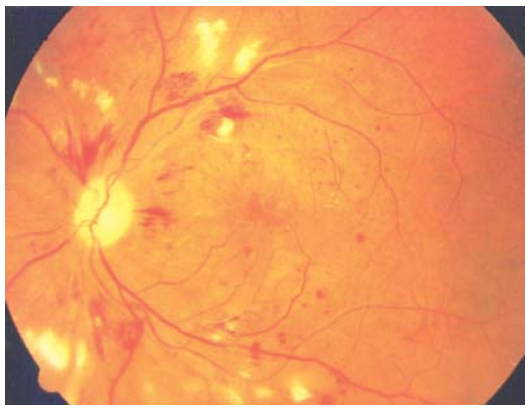


FIGURE 12.21.1: Soft exudates

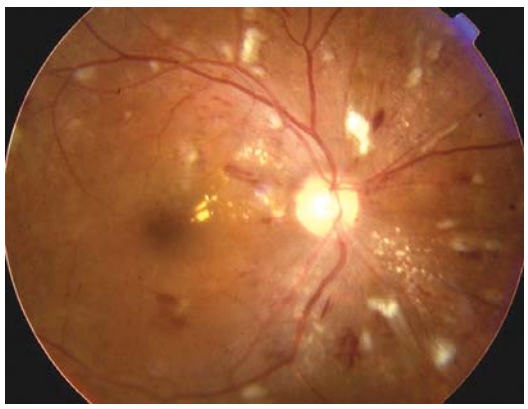


FIGURE 12.21.2: Soft exudates

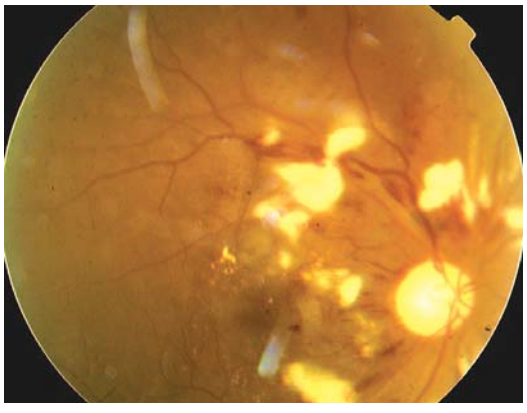


FIGURE 12.21.3: Soft exudates

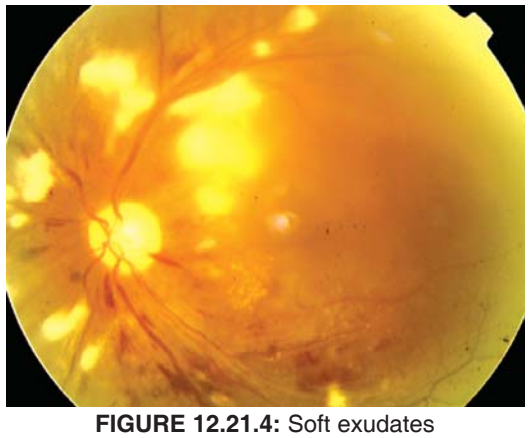


FIGURE 12.21.4: Soft exudates

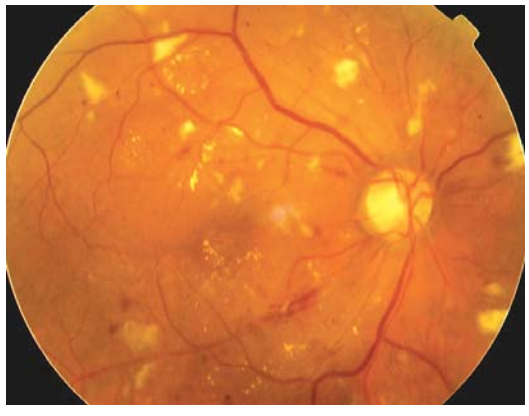


FIGURE 12.21.5: Soft and hard exudates

Subretinal Exudates

- Yellowish-white, exudative lesions with indistinct margin in the central or peripheral retina **(Figs 12.22.1 and 12.22.2)**
- Frequently associated with elevation of overlying retina **(Figs 12.22.3 and 12.22.4)**
- *Causes:* chronic leakage from SRNVMs, Coats' diseases, toxocariasis

FIGURE 12.22.1: Subretinal exudates

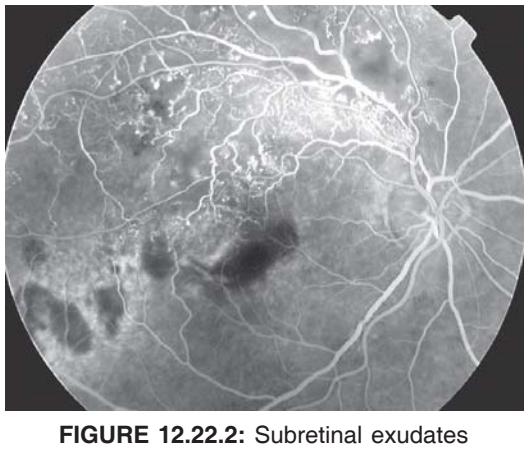


FIGURE 12.22.2: Subretinal exudates

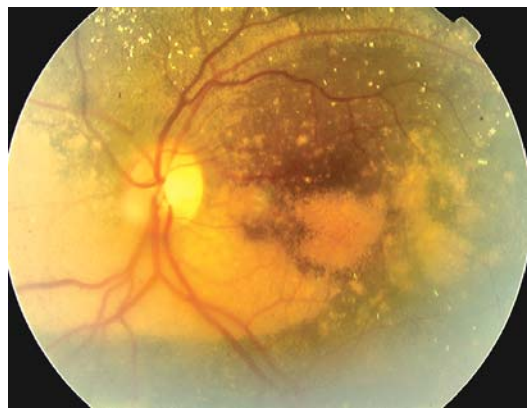


FIGURE 12.22.3: Subretinal exudates

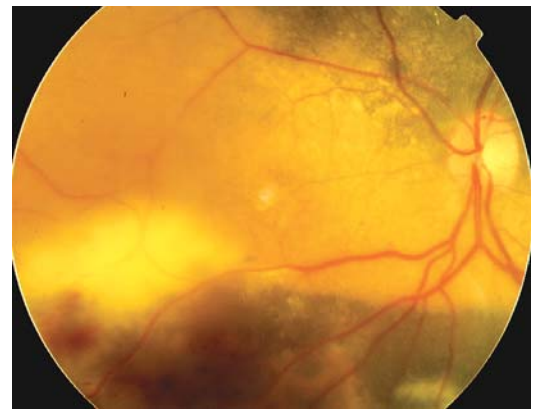


FIGURE 12.22.4: Subretinal exudates

Coats' disease

- It usually affects boys between 18 months to 18 years of age
- Main symptoms are decreased visual acuity and a 'white pupillary reflex'
- Yellowish-white exudative patches are seen behind the retinal blood vessels **(Figs 12.23.1 and 12.23.2)**
- Blood vessels have a tortuous course with aneurysms, fusiform dilations, and loop formations (**Fig 12.23.3**)

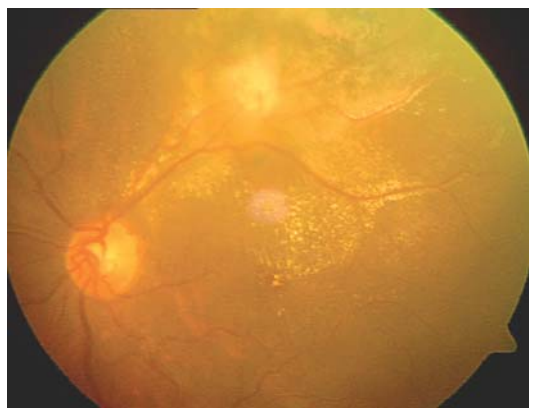


FIGURE 12.23.1: Coats' disease

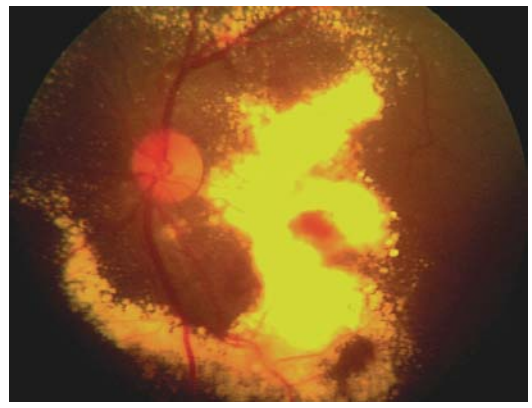


FIGURE 12.23.2: Coats' disease

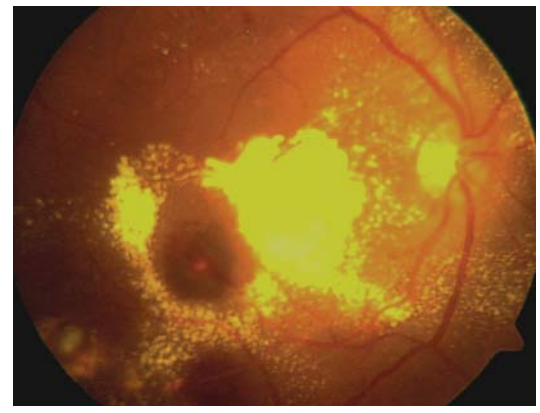


FIGURE 12.23.3: Coats' disease

INFLAMMATORY RETINAL LESIONS

Multiple Evanescent White Dot Syndrome

- Rare, unilateral condition affects the young women
- Mild vitritis in the posterior vitreous
- Multiple, very small white dot like lesion at the posterior pole and midperiphery **(Fig 12.24.1)**
- They are at the level of RPE and clearly visible on FFA **(Figs 12.24.2 and 12.24.3)**
- Prognosis is always good

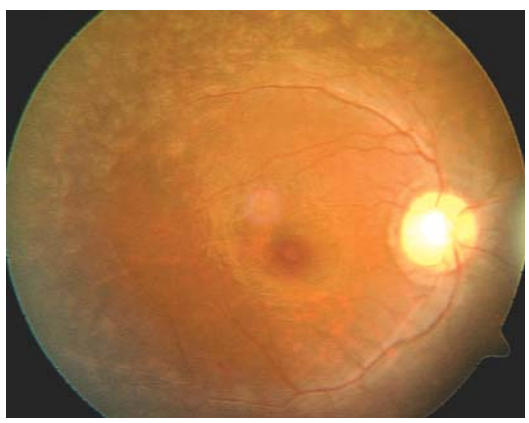


FIGURE 12.24.1: Multiple evanescent white dot syndrome

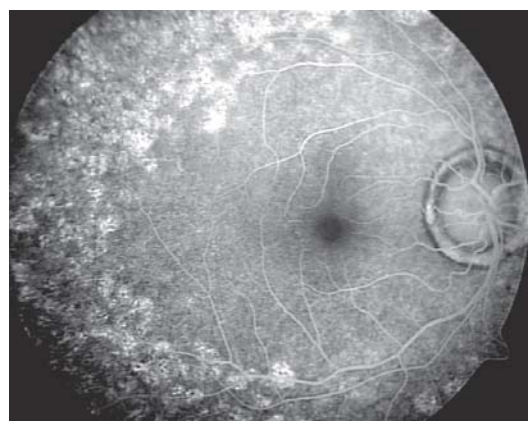


FIGURE 12.24.2: MEWDS—FFA

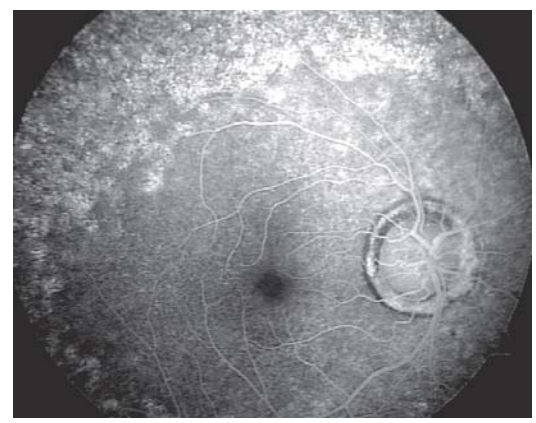


FIGURE 12.24.3: MEWDS—FFA

Punctate Inner Choroidopathy (PIC)

- Uncommon, bilateral condition affects young ladies with myopia
- No sign of vitritis
- Small yellowish-white indistinct choroidal lesions at the posterior pole **(Figs 12.25.1 to 12.25.3)**
- May be associated with a serous retinal detachment
- Prognosis is always guarded

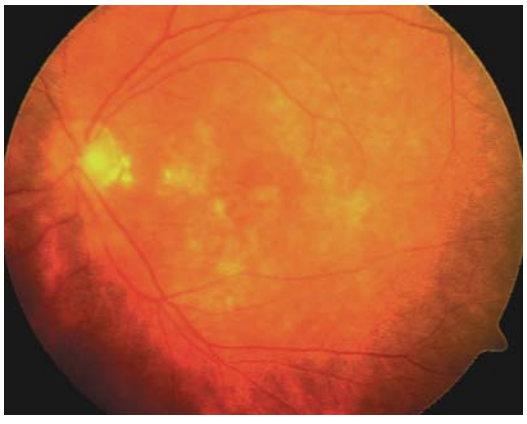


FIGURE 12.25.1: Punctate inner choroidopathy

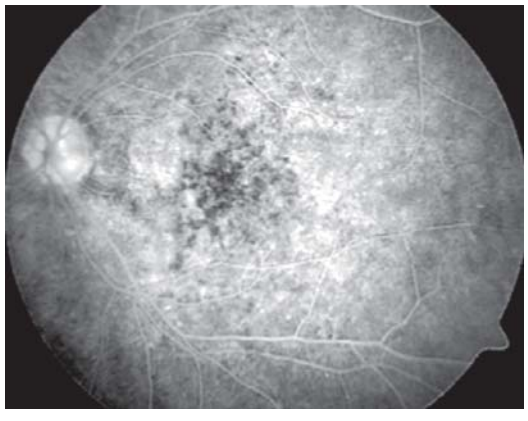


FIGURE 12.25.2: Punctate inner choroidopathy

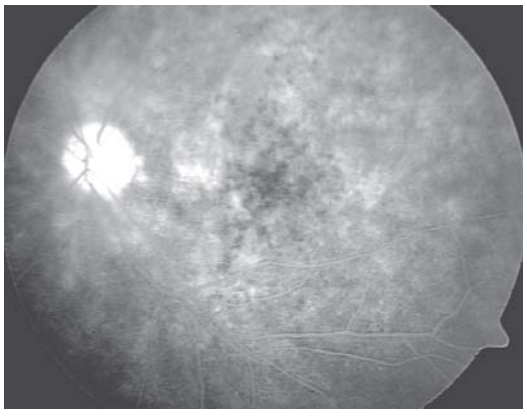


FIGURE 12.25.3: Punctate inner choroidopathy

Birdshot Retinochoroidopathy

- *See Chapter: 6*

Acute Posterior Multifocal Placoid Pigment Epitheliopathy (APMPPE)

- Large cream-colored, placoid lesions at the posterior pole and midperiphery **(Fig 12.26.1)**
- Located at the level of RPE, appreciated by FFA: initial hypofluorescence followed by late diffuse staining **(Figs 12.26.2 and 12.26.3)**

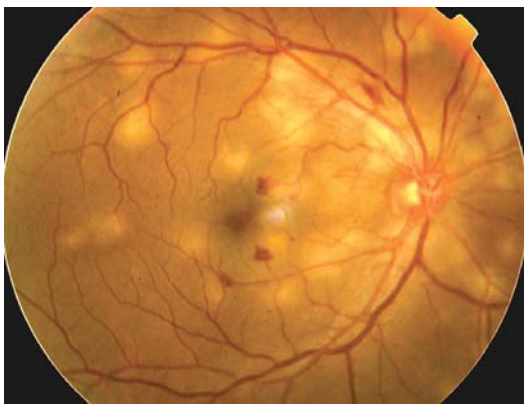


FIGURE 12.26.1: APMPPE

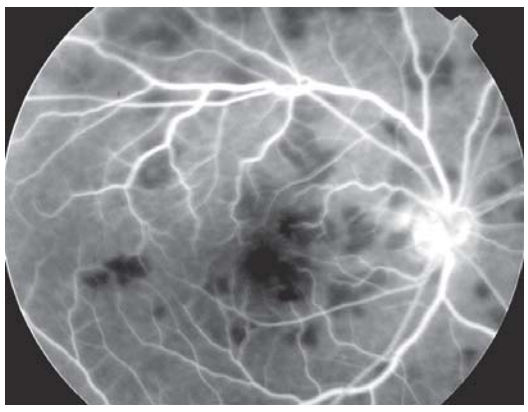


FIGURE 12.26.2: APMPPE on FFA

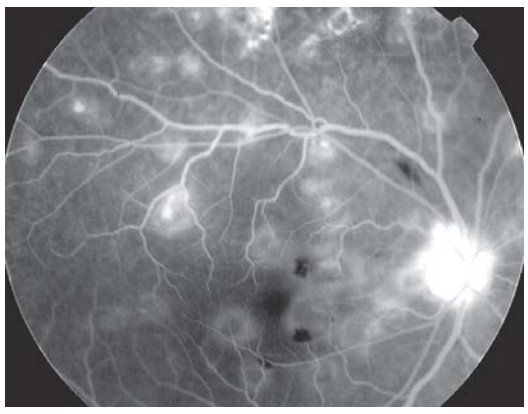


FIGURE 12.26.3: APMPPE on FFA

Multifocal Choroiditis with Panuveitis

- Unilateral or bilateral uncommon condition, affects the middle-aged women
- Often recurrent in nature
- Associated anterior uveitis
- Small, diskrete, fresh and old lesions at mid-periphery and posterior pole **(Fig 12.27.1)**
- Variable areas of chorioretinal atrophy
- Lesions are more accurately visible on FFA **(Figs 12.27.2 to 12.27.5)**

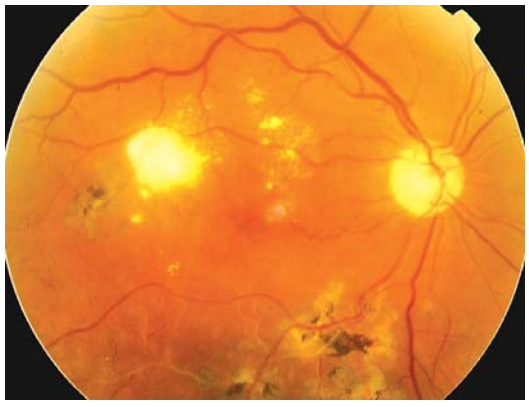


FIGURE 12.27.1: Multifocal choroiditis with panuveitis

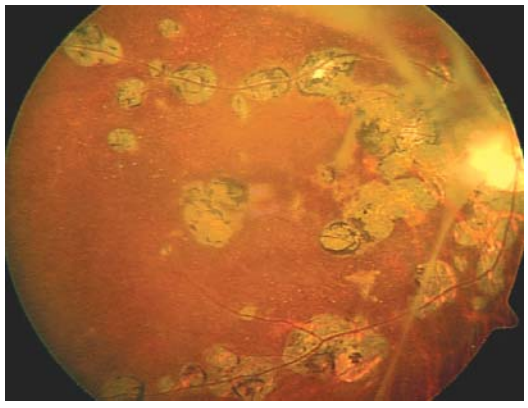


FIGURE 12.27.2: Active multifocal choroiditis—bilateral

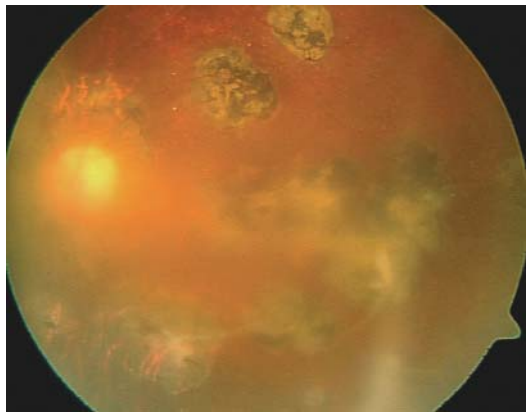


FIGURE 12.27.3: Active multifocal choroiditis—bilateral

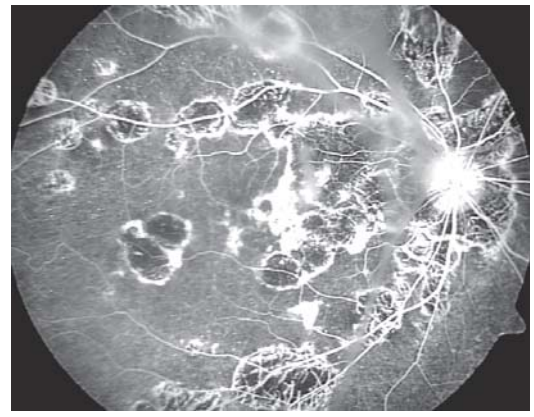


FIGURE 12.27.4: Active multifocal choroiditis—bilateral

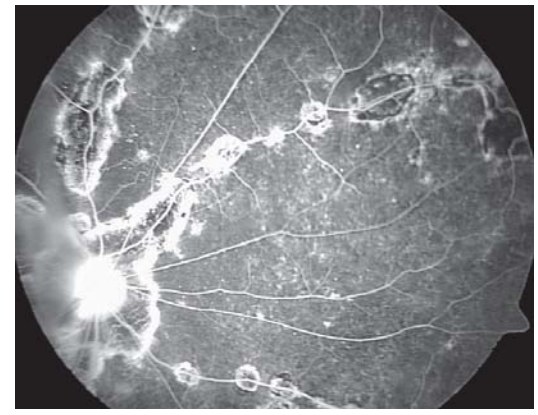


FIGURE 12.27.5: Active multifocal choroiditis—bilateral

Disseminated Choroiditis

- Unilateral or bilateral lesions, may occur at any age
- Multiple chorioretinal yellowish-white lesions scattered all over the fundus **(Figs 12.28.1 and 12.28.2)**
- Associated with mild vitritis
- FFA shows typical lesions of initial hypofluorescence followed by hyperfluorescence **(Figs 12.28.3 and 12.28.4)**
- Prognosis is fair

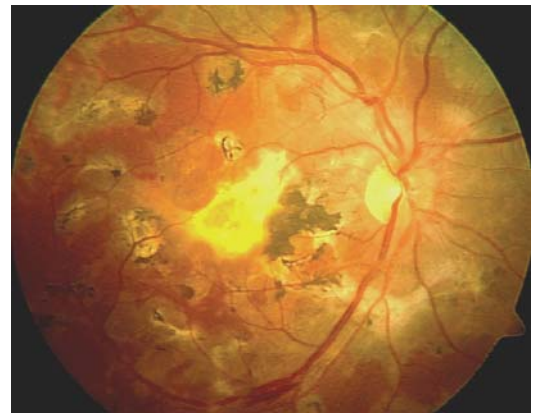


FIGURE 12.28.1: Disseminated choroiditis

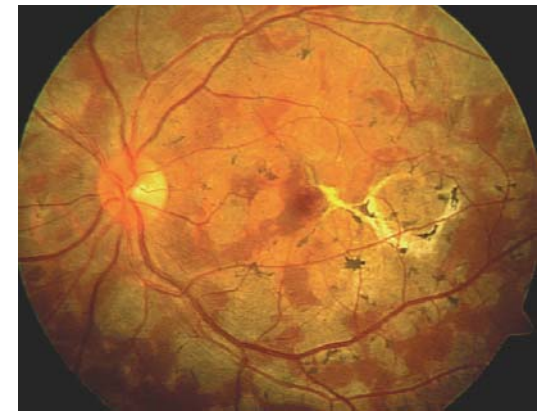


FIGURE 12.28.2: Disseminated choroiditis

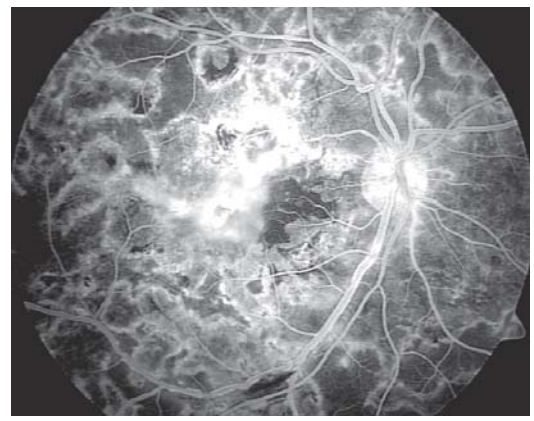


FIGURE 12.28.3: Disseminated choroiditis

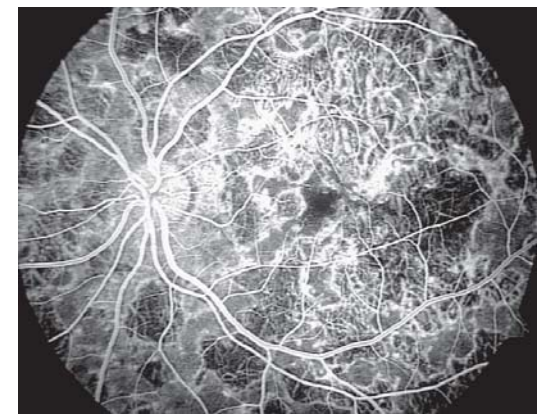


FIGURE 12.28.4: Disseminated choroiditis

Serpiginous Choroidopathy

- *See Chapter: 6*

Focal Toxoplasmosis

- Unilateral, protozoan infection caused by *Toxoplasma gondii*
- Typically affects young individual between 15 to 30 years of age
- Moderate to severe vitritis
- Focal retinitis adjacent to the edge of old lesion **(Figs 12.29.1 and 12.29.2)**
- May be with cystoid macular edema
- In acquired toxoplasmosis, it may not be associated with old scar

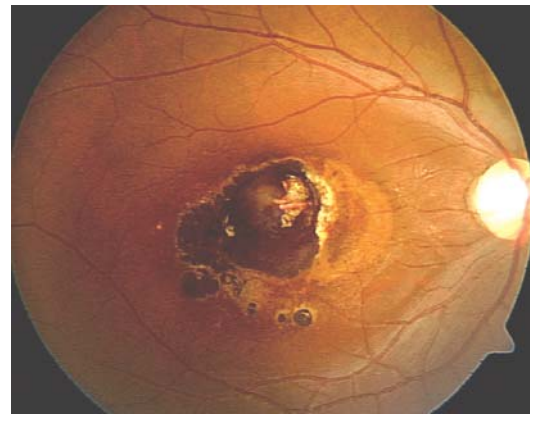


FIGURE 12.29.1: Toxoplasmosis—old scar

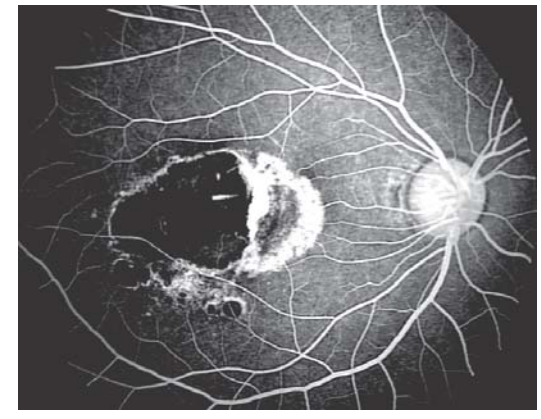


FIGURE 12.29.2: Toxoplasmosis—old scar

- Extensive late leakage on FFA **(Figs 12.29.3 and 12.29.4)**
- May be associated with anterior granulomatous uveitis

FIGURE 12.29.3: Toxoplasmosis—reactivation other eye of same patient

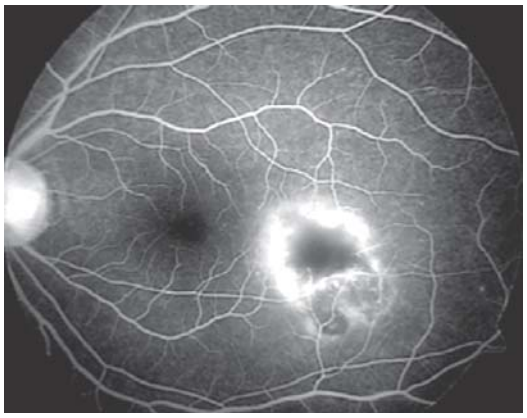


FIGURE 12.29.4: Toxoplasmosis—reactivation

Toxocariasis

- *See Chapter: 11*

Candidiasis

- Rare, unilateral or bilateral fungal infection, typically affects drug addicts, or patients on some kind of immunosuppressants
- Solitary or multiple deep retinal infiltrates **(Fig 12.30.1)**
- Multiple cotton ball opacities in the vitreous with vitritis **(Figs 12.30.2 and 12.30.3)**
- Prognosis is usually poor

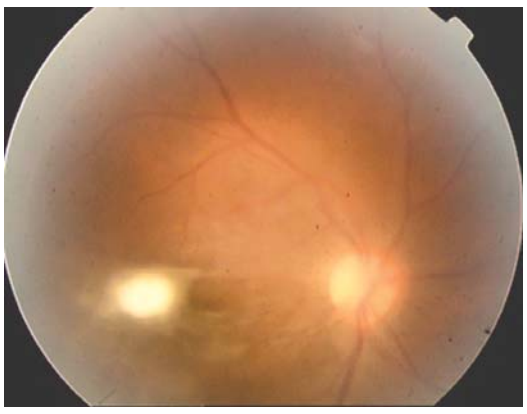


FIGURE 12.30.1: Candidiasis—retinal lesion

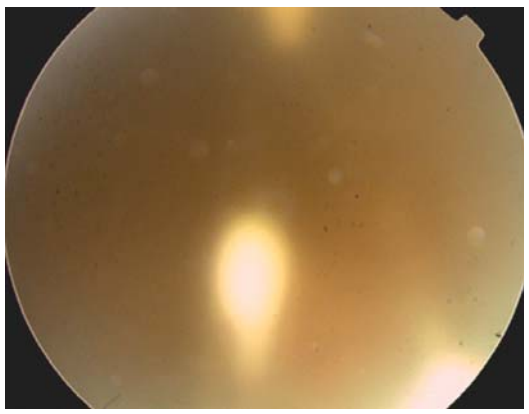


FIGURE 12.30.2: Candidiasis—cotton ball opacity in the vitreous

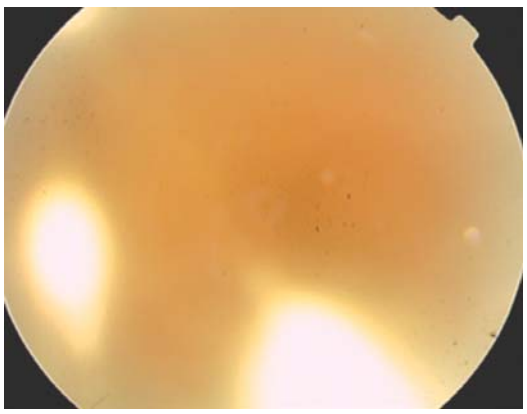


FIGURE 12.30.3: Candidiasis—cotton ball opacities in the vitreous

Cytomegalovirus (CMV) Retinitis

- Twenty five percent of patients of AIDS suffer from CMV retinitis and bilaterality in 50 percent cases
- Yellowish-white areas of retinal infiltration with advancing brush-like border **(Fig 12.31.1)**
- Slowly progressive and typically start at the posterior pole and spread along the vascular arcades
- Hemorrhages in the midst of retinitis
- Diffuse atrophy in late stage
- *Other fundal changes in AIDS patients:*
  - *retinal microvasculopathy* as evident by cotton wool spots, superficial and deep hemorrhage **(Fig 12.31.2)**
  - immunocomplex deposits in the pre-

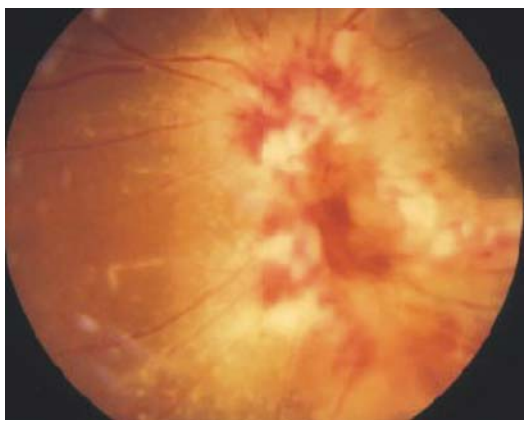


FIGURE 12.31.1: CMV retinitis

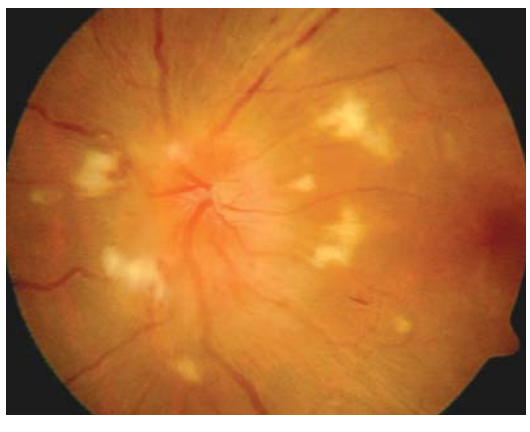


FIGURE 12.31.2: Retinal microvasculopathy—HIV

capillary arterioles
- central retinal venous occlusion **(Fig 12.31.3)**
- immune-recovery uveitis **(Fig 12.31.4)**

- *Other causes of CMV retinitis:* patient on cytotoxic chemotherapy or renal transplant patient

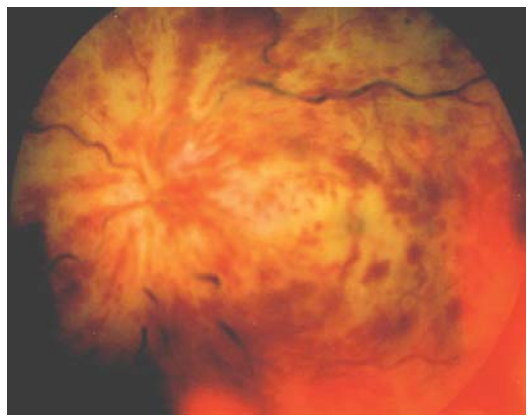


FIGURE 12.31.3: CMV retinitis—CRVO

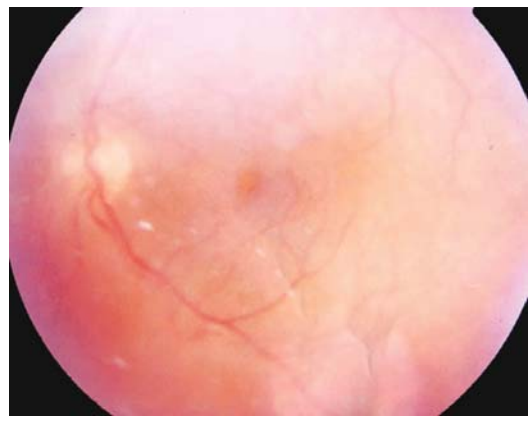


FIGURE 12.31.4: Immuno-recovery uveitis

Acute Retinal Necrosis

- Extremely rare, devastating necrotizing retinitis affects otherwise healthy subjects
- Anterior uveitis and vitritis is common
- Confluent yellowish-white patches with sheathing and occlusion of blood vessels **(Fig 12.32.1)**
- Most eyes develop multiple retinal holes with rhegmatogenous retinal detachment
- Progression is faster and macula often gets spared

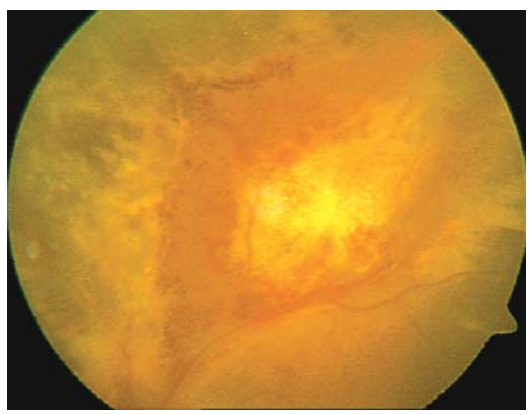


FIGURE 12.32.1: Acute retinal necrosis—CMV retinitis

Progressive Outer Retinal Necrosis (PORN)

- PORN is a severe variant of acute retinal necrosis in patients with AIDS
- May be bilateral in 70 percent of cases
- Deep multifocal lesions giving rise to peripheral necrotizing retinitis **(Fig 12.33.1)**
- No occlusive vasculitis
- Very rapid progression with involvement of the optic disk and macula

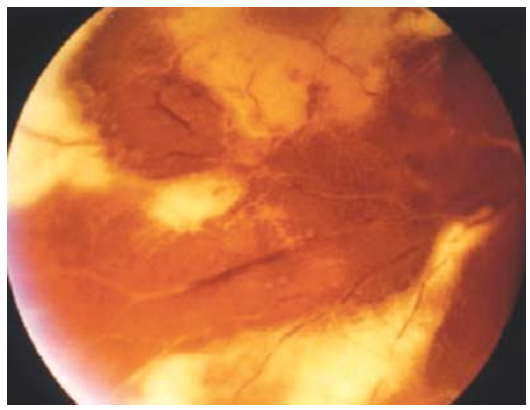


FIGURE 12.33.1: Progressive outer retinal necrosis

Vogt-Koyanagi-Harada Syndrome

- *See Chapter: 6*

Sympathetic Ophthalmia

- *See Chapter: 6*

Tuberculous Choroiditis

- Not so uncommon cause of uveitis specially in developing countries
- *May appear in many ways:*
  - chronic granulomatous anterior uveitis
  - multifocal central or midperipheral choroiditis **(Figs 12.34.1 and 12.34.2)**
  - vasculitis or periphlebitis
  - occasionally as solitary choroidal granuloma **(Figs 12.34.3 to 12.34.5)**

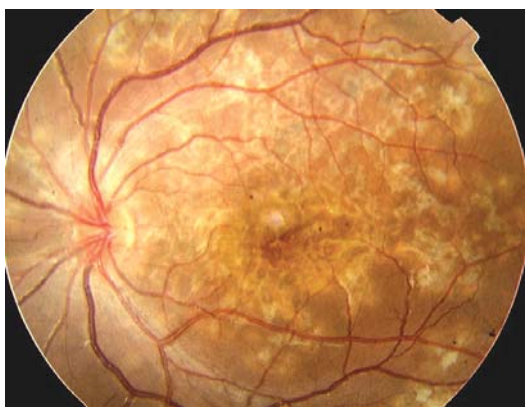


FIGURE 12.34.1: Active central multifocal choroiditis

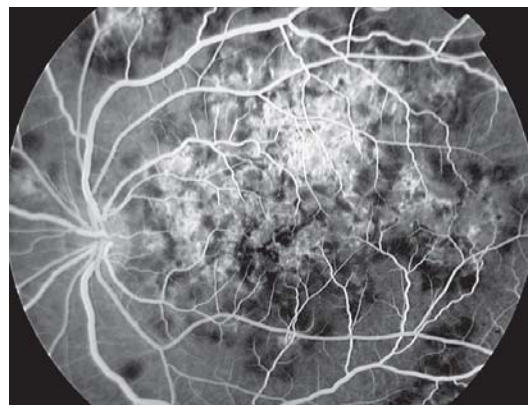


FIGURE 12.34.2: Active central multifocal choroiditis

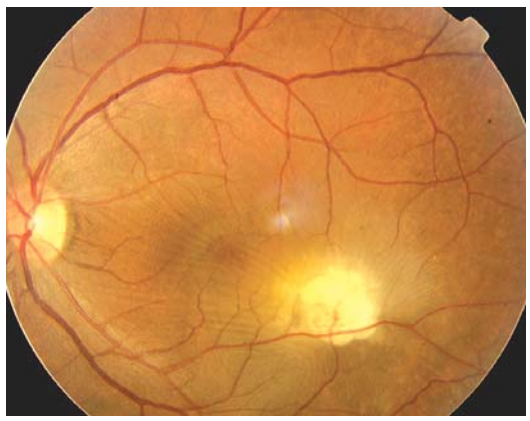


FIGURE 12.34.3: Active tubercular granuloma

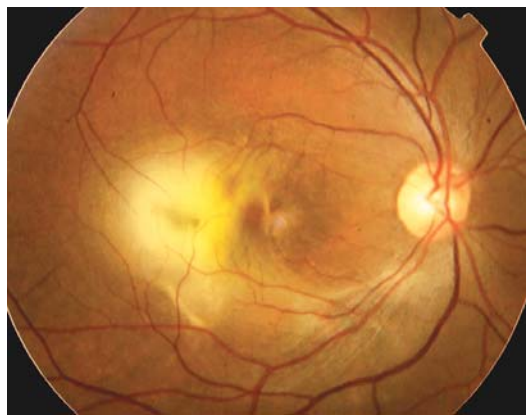


FIGURE 12.34.4: Tubercular granuloma

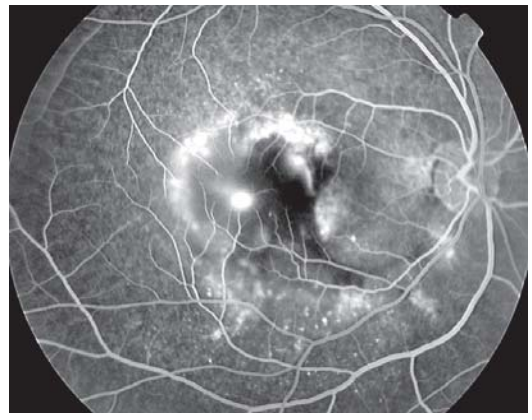


FIGURE 12.34.5: Tubercular granuloma—FFA

MACULAR LESIONS (MACULOPATHIES)

HEREDITARY MACULOPATHIES

Stargardt's Macular Dystrophy

- Rare, bilateral, recessive condition starts around 14 to 16 year
- May result in severe visual loss within five years
- *Macula may appear as:*
  - isolated atrophic maculopathy **(Figs 12.35.1 and 12.35.2)**
  - atrophic maculopathy with fish tail-shaped flecks **(Figs 12.35.3 to 12.35.6)**
  - with diffuse flecks around the posterior pole **(Figs 12.35.7 and 12.35.8)**
  - beaten metal appearance
- Prognosis is always poor

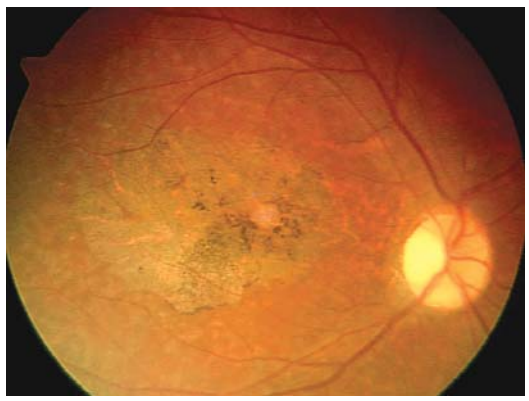


FIGURE 12.35.1: Stargardt's atrophic maculopathy

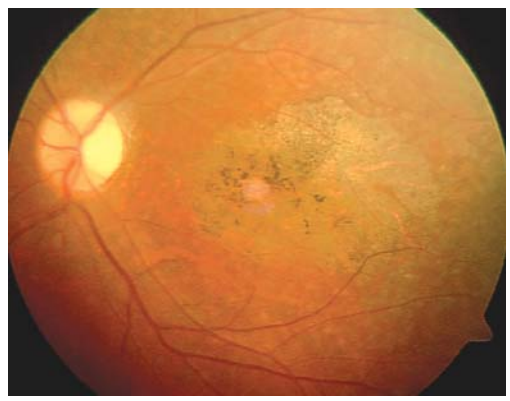


FIGURE 12.35.2: Stargardt's atrophic maculopathy

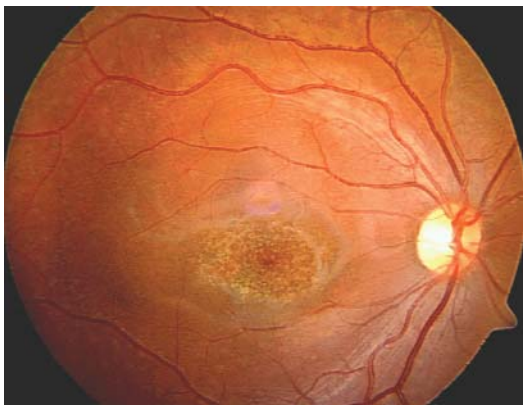


FIGURE 12.35.3: Stargardt's atrophic maculopathy with fish tail-shaped flecks

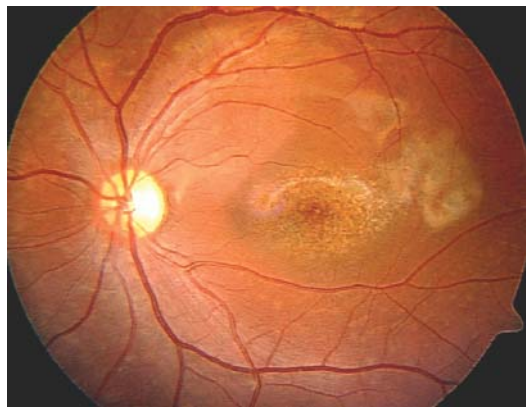


FIGURE 12.35.4: Stargardt's atrophic maculopathy with fish tail-shaped flecks

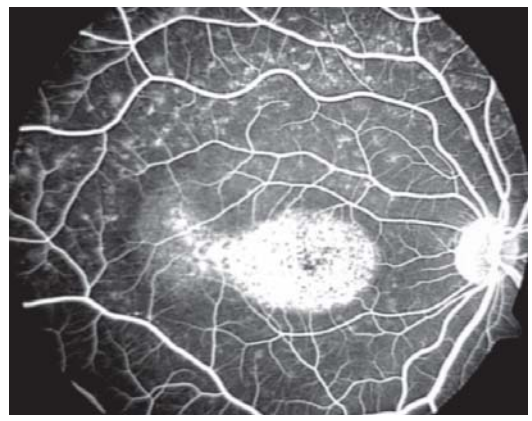


FIGURE 12.35.5: Stargardt's atrophic maculopathy with fish tail-shaped flecks

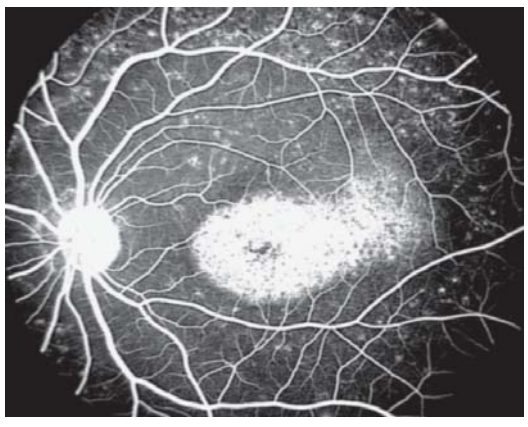


FIGURE 12.35.6: Stargardt's atrophic maculopathy with fish tail-shaped flecks

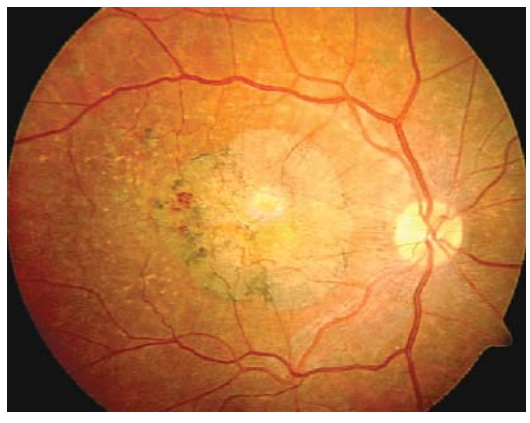


FIGURE 12.35.7: Stargardt's maculopathy with flecks

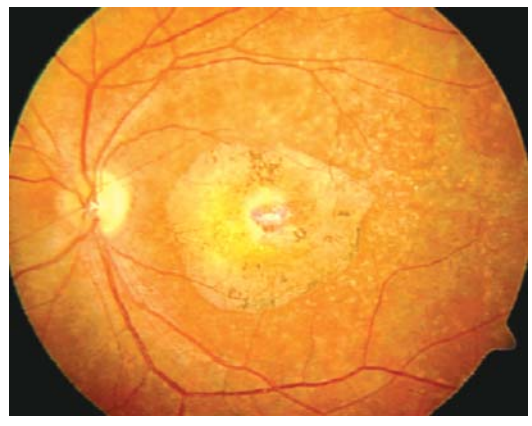


FIGURE 12.35.8: Stargardt's maculopathy with flecks

Central Areolar Choroidal Dystrophy

- Rare, bilateral condition with dominant inheritance
- Loss of central vision at around 50 to 60 years
- Well circumscribed, bilateral atrophic macular lesion of 1 to 3 disk diameter **(Figs 12.36.1 and 12.36.2)**
- Not associated with drusen

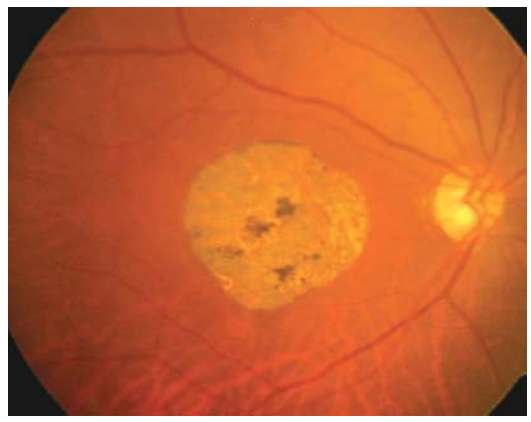


FIGURE 12.36.1: Central areolar choroidal dystrophy

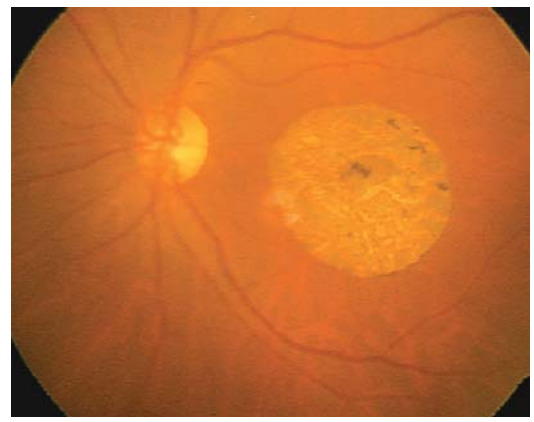


FIGURE 12.36.2: Central areolar choroidal dystrophy

Best's Vitelliform Macular Dystrophy

- Very rare, dominant hereditary condition starts in childhood
- Usually bilateral, but often asymmetrical
- Clinically, it has the *following stages*:
  - *vitelliform stage:* egg yolk-like macular lesion **(Fig 12.37.2)**
  - *pseudohypopyon stage:* with partial absorption
  - *vitelliruptive stage:* scrumbled egg macular lesion **(Fig 12.37.1)**
  - *end stage:* with atrophic maculopathy and diskiform scarring
- Family member may be diagnosed early in *pre-vitelliform stage* by EOG
- Prognosis is always poor

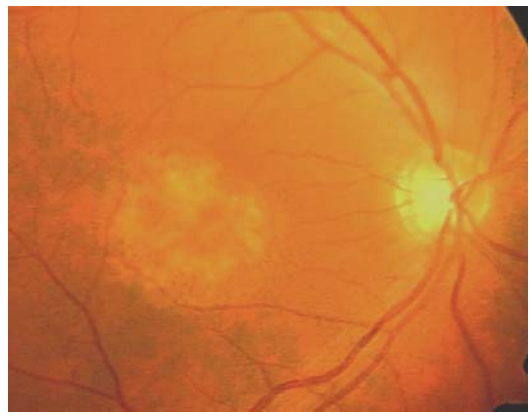


FIGURE 12.37.1: Vitelliform dystrophy—scrumbled egg appearance

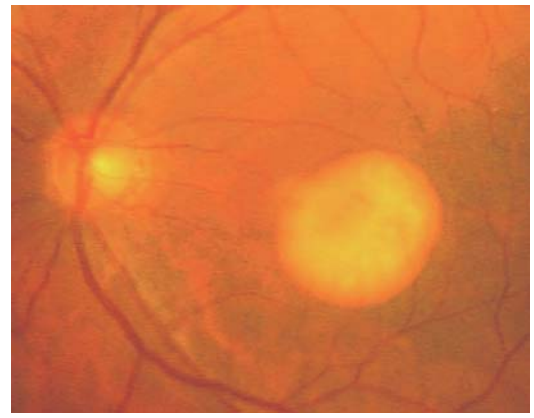


FIGURE 12.37.2: Vitelliform dystrophy—egg yolk lesion

Hereditary Cone Dystrophy (Bull's eye maculopathy)

- Very rare bilateral macular dystrophy which presents as Bull's eye maculopathy
- Autosomal dominant or X-linked recessive
- Presents between first to third decade
- *Bull's eye macular lesions*, with golden reflex **(Figs 12.38.1 to 12.38.4)**
- vascular attenuation and temporal pallor may be present
- *Other causes of Bull's eye maculopathy:*
  - *chloroquine toxicity:* dose-dependent and occurs after prolonged use, especially if used for rheumatoid arthritis; associated retinal vascular attenuation and may not be reversible **(Figs 12.38.5 to 12.38.8)**
  - *late stage of Stargardt's disease* **(Figs 12.38.9 and 12.38.10)**
  - *inverse retinitis pigmentosa*

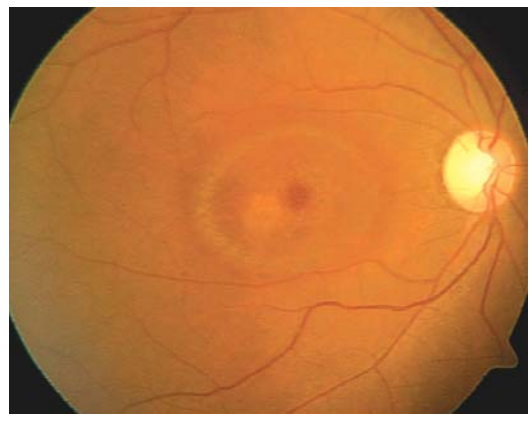


FIGURE 12.38.1: Bull's eye maculopathy—cone dystrophy

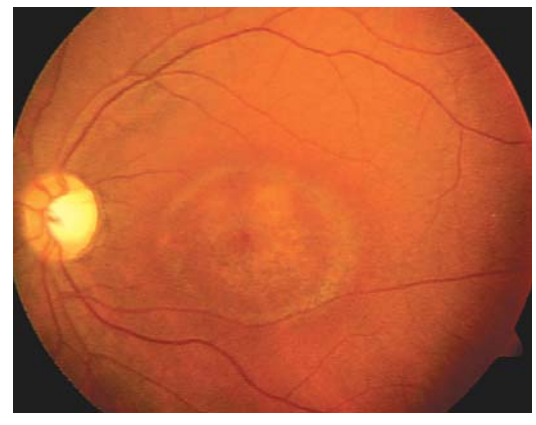


FIGURE 12.38.2: Bull's eye maculopathy—cone dystrophy

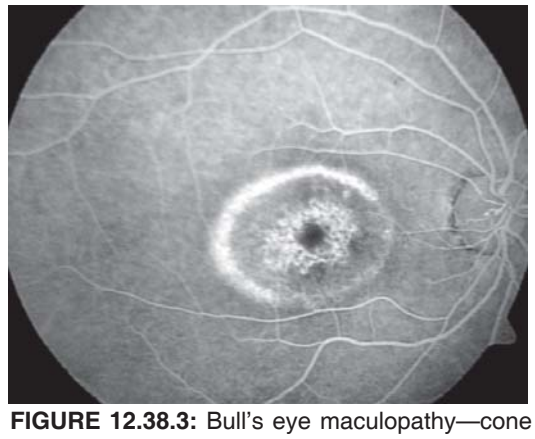


FIGURE 12.38.3: Bull's eye maculopathy—cone dystrophy

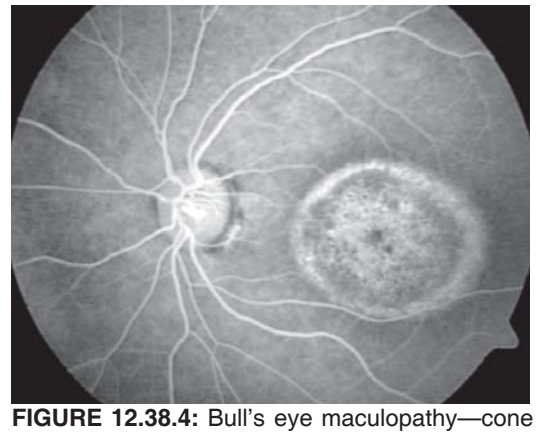


FIGURE 12.38.4: Bull's eye maculopathy—cone dystrophy

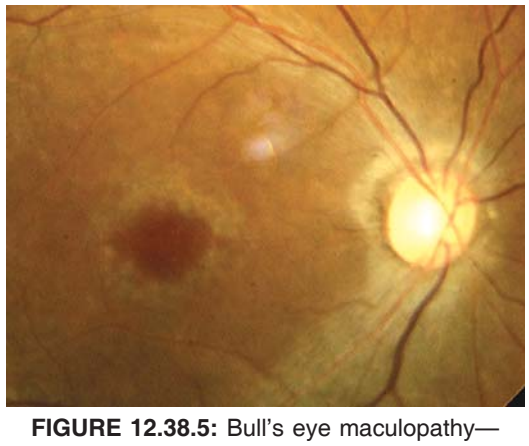


FIGURE 12.38.5: Bull's eye maculopathy—chloroquine toxicity

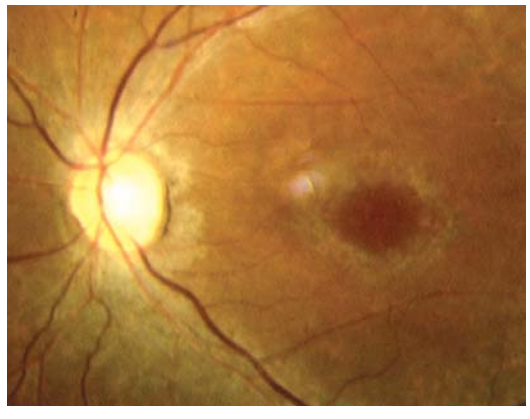


FIGURE 12.38.6: Bull's eye maculopathy—chloroquine toxicity

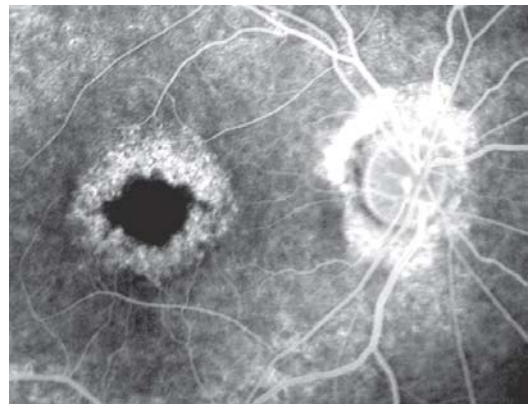


FIGURE 12.38.7: Bull's eye maculopathy—chloroquine toxicity

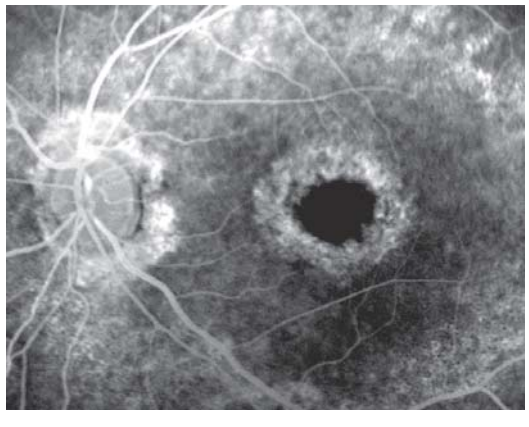


FIGURE 12.38.8: Bull's eye maculopathy—chloroquine toxicity

FIGURE 12.38.9: Bull's eye maculopathy—Stagardt's disease late stage

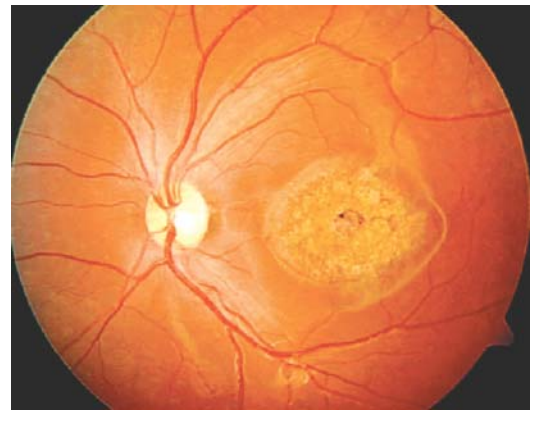


FIGURE 12.38.10: Bull's eye maculopathy—Stagardt's disease late stage

Myopic Maculopathies

- Common, bilateral, often asymmetrical macular lesions in adults with high and pathological myopia
- Lesions are usually progressive
- *Changes are:*
  - lacquer cracks at posterior pole **(Fig 12.39.1)**
  - atrophic maculopathy **(Figs 12.39.2 and 12.39.3)**
  - macular hemorrhage with choroidal neovascularization **(Figs 12.39.4 and 12.39.5)**
  - macular hole with or without retinal detachment **(Fig 12.39.6)**
  - Fuchs' pigmented spot at fovea **(Figs 12.39.7 and 12.39.8)**
  - posterior staphyloma **(Figs 12.39.9 and 12.39.10)**
- Overall gross tessellation of entire fundus including the macular region **(Fig 12.39.11)**
- Peripheral degenerative lesions are also common in myopia

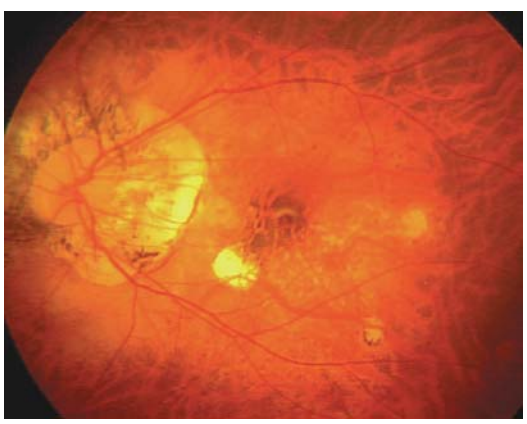


FIGURE 12.39.1: Myopic maculopathy—lacquer cracks

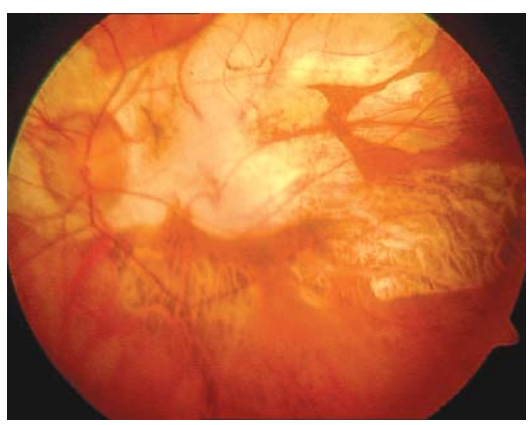


FIGURE 12.39.2: Myopic atrophic maculopathy

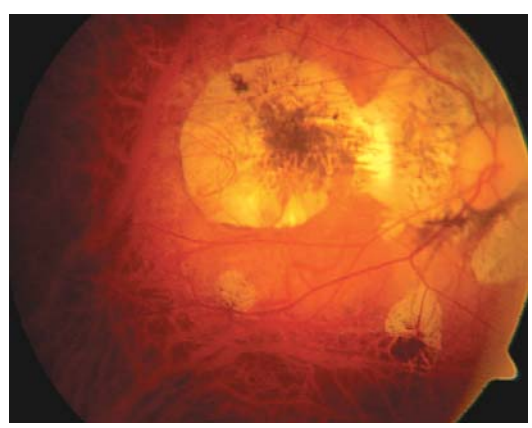


FIGURE 12.39.3: Myopic atrophic maculopathy

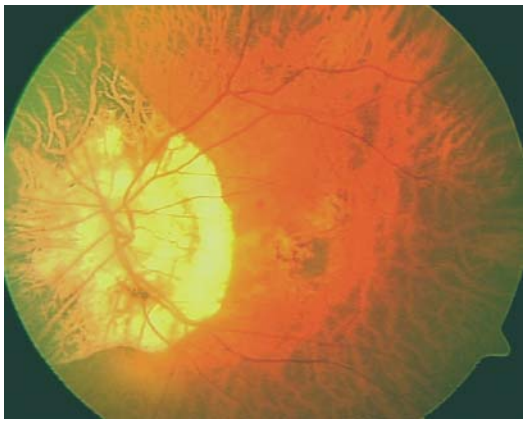


FIGURE 12.39.4: Myopic maculopathy—hemorrhage from CNV

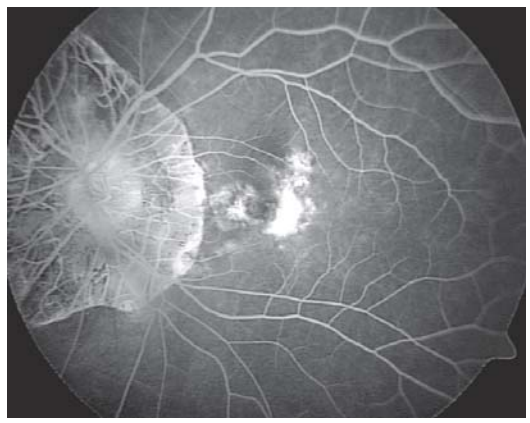


FIGURE 12.39.5: Myopic maculopathy—hemorrhage from CNV

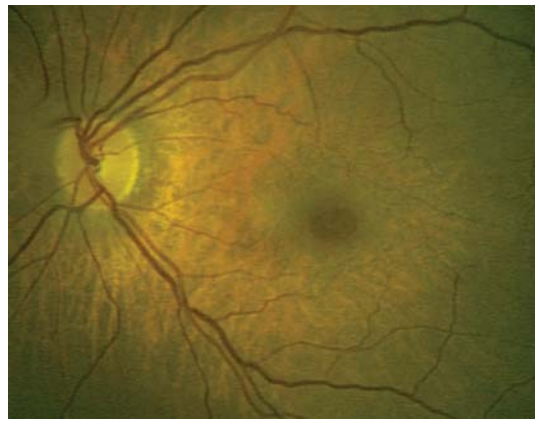


FIGURE 12.39.6: Myopic macular hole

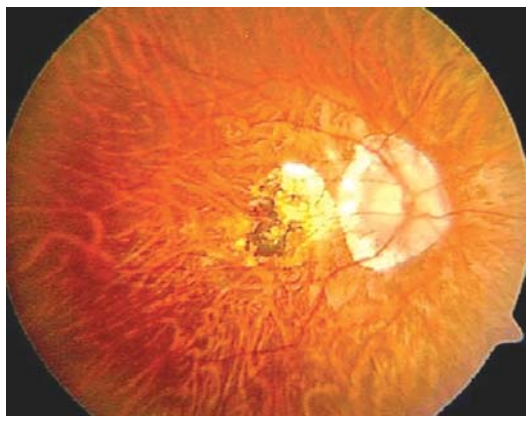


FIGURE 12.39.7: Myopic maculopathy—Fuchs' spot

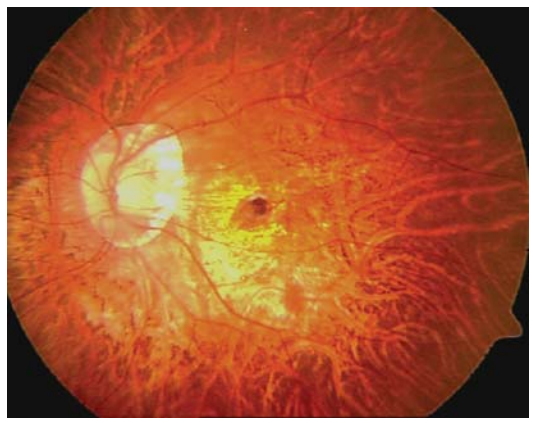


FIGURE 12.39.8: Myopic maculopathy—Fuchs' spot

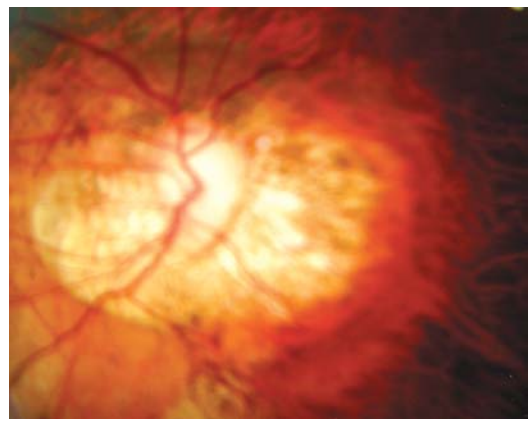


FIGURE 12.39.9: Myopic maculopathy—posterior staphyloma

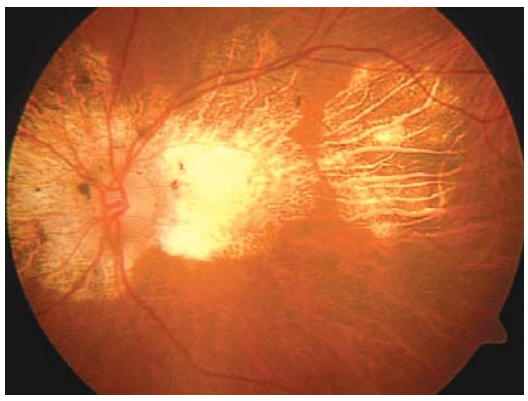


FIGURE 12.39.10: Myopic maculopathy—posterior staphyloma

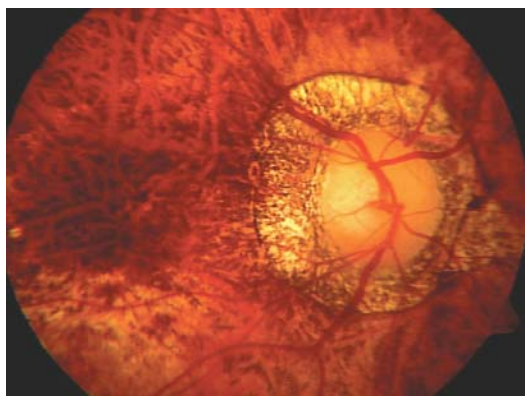


FIGURE 12.39.11: Myopic maculopathy — tessellated fundus

Dry Age-Related Macular Degeneration

- Dry ARMD is very common, bilateral condition, often bilateral
- Most common form of macular degeneration
- Affecting the patient above sixty years of age
- Associated with soft drusen **(Figs 12.40.1 and 12.40.2)**

FIGURE 12.40.1: Dry AMD—geographical atrophy

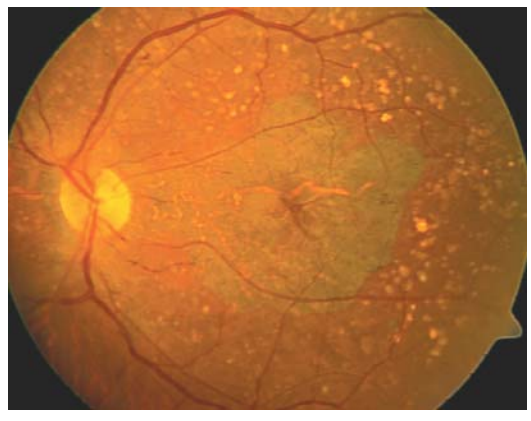


FIGURE 12.40.2: Dry AMD geographical atrophy

- Sharply-demarcated areas of macular hypopigmentation or depigmentation
- Visible underlying choroidal vessels (geographical atrophy) **(Figs 12.40.3 and 12.40.4)**
- FFA shows widespread window defect with late staining

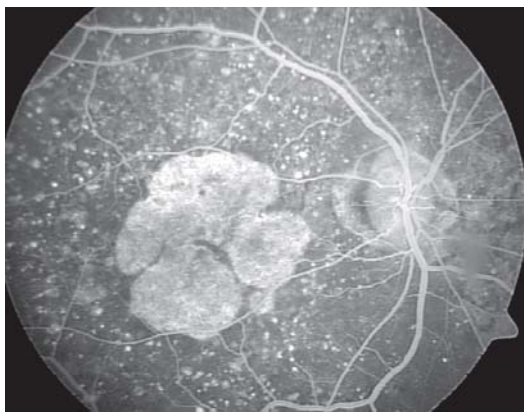


FIGURE 12.40.3: Dry AMD—geographical atrophy

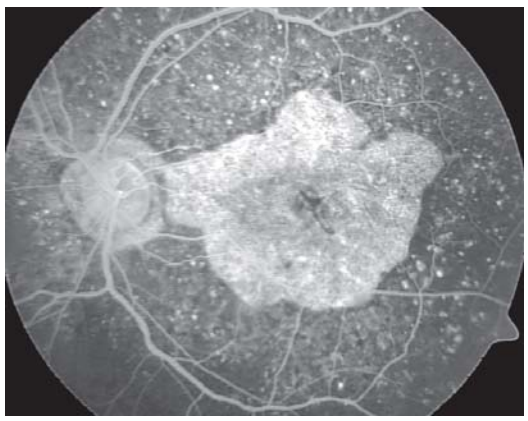


FIGURE 12.40.4: Dry AMD—geographical atrophy

EXUDATIVE MACULOPATHIES

Cystoid Macular edema

- Cystoid macular edema (CME) is an accumulation of fluid in the outer plexiform (Henle's) layer and inner nuclear layer of the retina, in the macular region
- Shows an irregularity and blurring of the foveal reflex **(Fig 12.41.1)**
- Macula is wrinkled, edematous, and may show multiple small cystic changes
- FFA shows typical 'flower-petal' or 'spoke' pattern of leakage at the fovea during the late phase **(Figs 12.41.2 and 12.41.3)**
- *Causes:* cataract surgery (Irvine-Gass syndrome), chronic iridocyclitis, intermediate uveitis, YAG capsulotomy, retinitis pigmentosa, diabetes, etc.

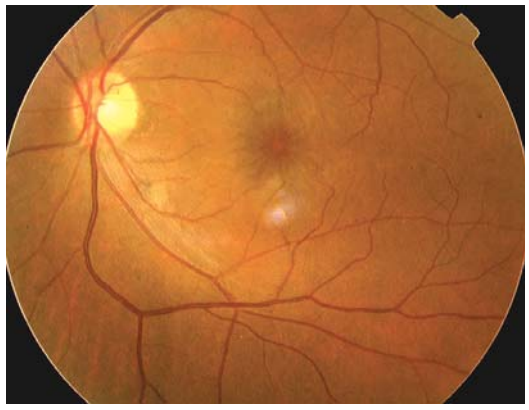


FIGURE 12.41.1: Cystoid macular edema

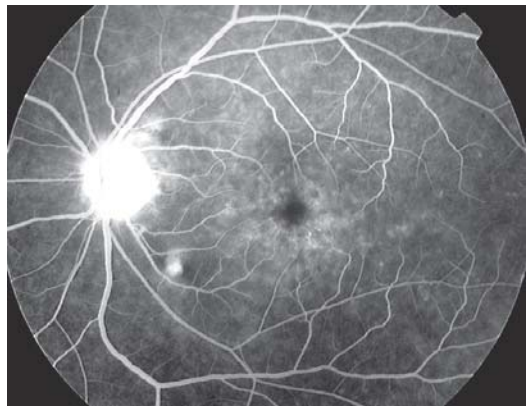


FIGURE 12.41.2: Cystoid macular edema

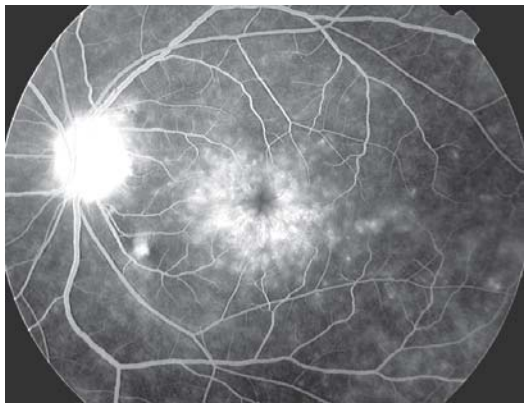


FIGURE 12.41.3: Cystoid macular edema

Central Serous Retinopathy

- Is actually the detachment of the neurosensory retina from the RPE by serous fluid
- Characterized by sudden shallow elevation of the neuroretina in the macular area with indistinct margin
- Macula appears as an oval or circular dark swelling, about the size of the optic disk, often with glistening ring-reflex **(Fig 12.42.1)**

FIGURE 12.42.1: Central serous retinopathy

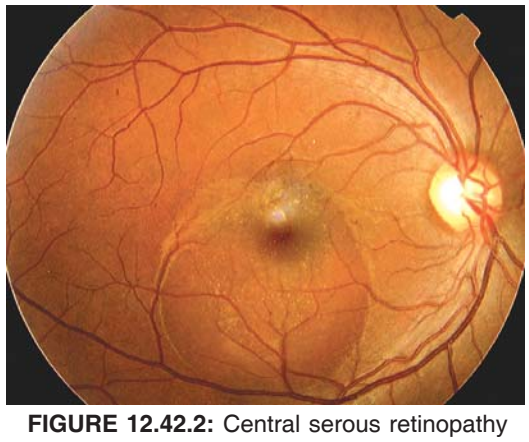


FIGURE 12.42.2: Central serous retinopathy

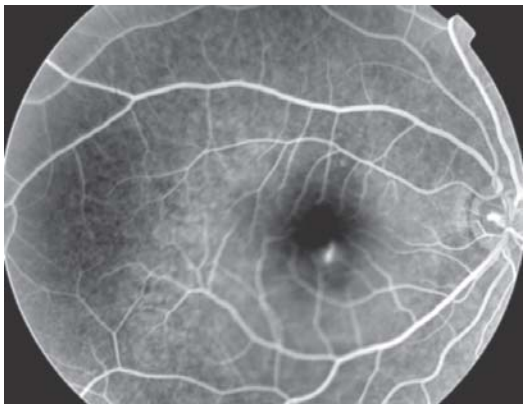


FIGURE 12.42.3: CSR—smoke-stack pattern on FFA

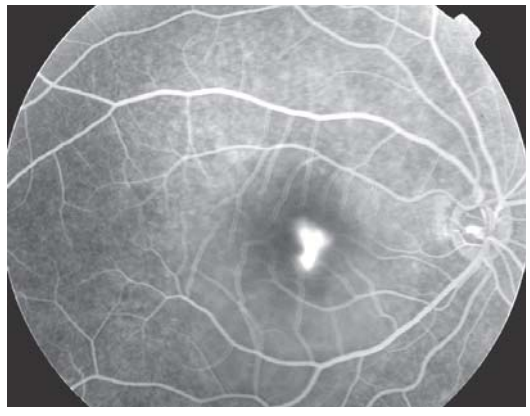


FIGURE 12.42.4: CSR—smoke-stack pattern on FFA

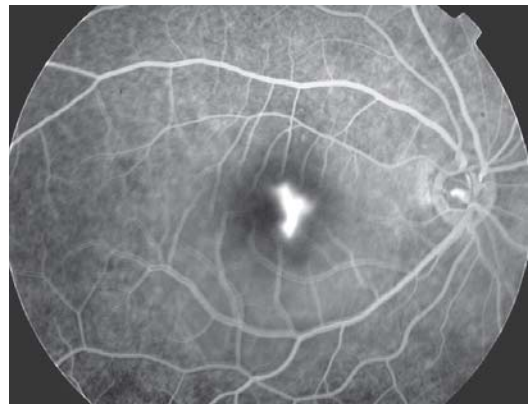


FIGURE 12.42.5: CSR—smoke-stack pattern on FFA

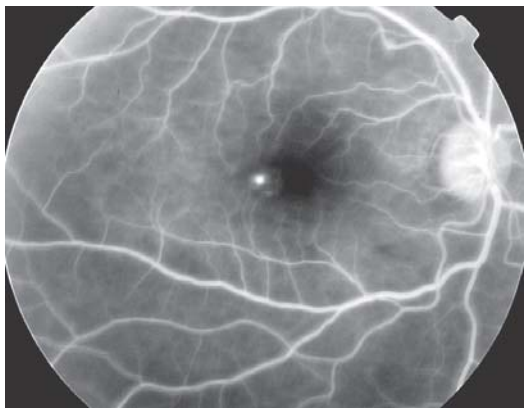


FIGURE 12.42.6: CSR—ink-blot pattern on FFA

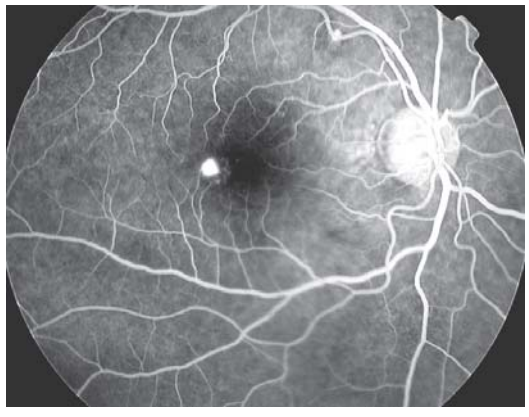


FIGURE 12.42.7: CSR—ink-blot pattern on FFA

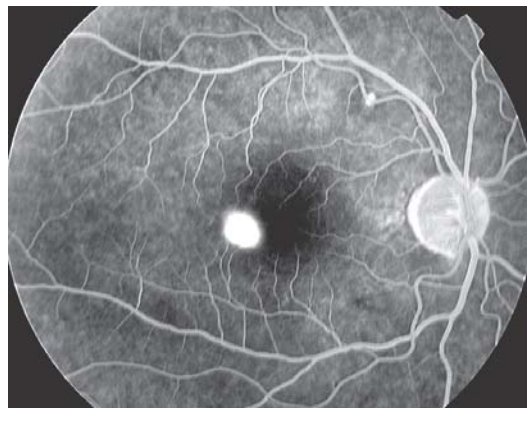


FIGURE 12.42.8: CSR—ink-blot pattern on FFA

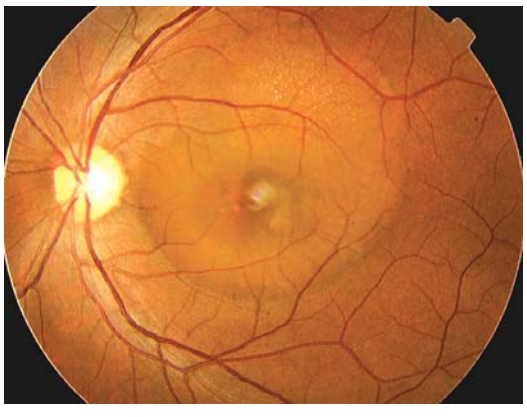


FIGURE 12.42.9: CSR—extra foveal

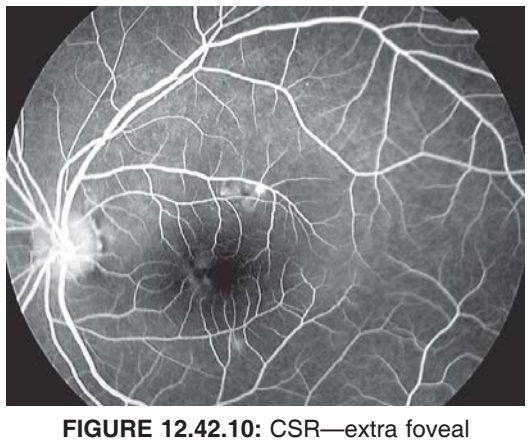


FIGURE 12.42.10: CSR—extra foveal

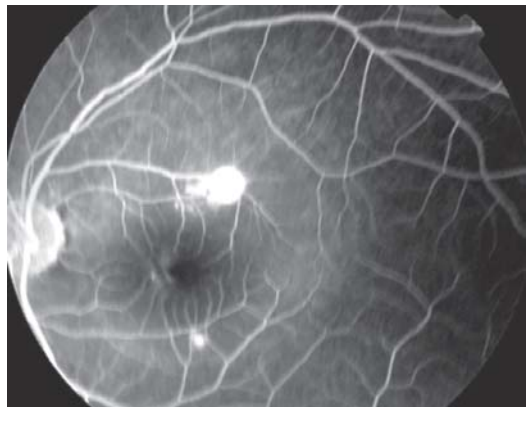


FIGURE 12.42.11: CSR—extra foveal

- Sometimes, tiny subretinal precipitates may be visible **(Figs 12.42.2)**
- FFA shows characteristic 'smoke-stack'-pattern leak **(Figs 12.42.3 to 12.42.5)** or sometimes gradual 'ink-blot'-pattern leak **(Figs 12.42.6 to 12.42.8)** under the neuroretina
- Occasionally, there may be atypical and extrafoveal **(Figs 12.42.9 to 12.42.11)** o,r multiple leak CSR
- Healed CSR may leaks profusely in a different ways**(Figs 12.42.12 and 12.42.13)**

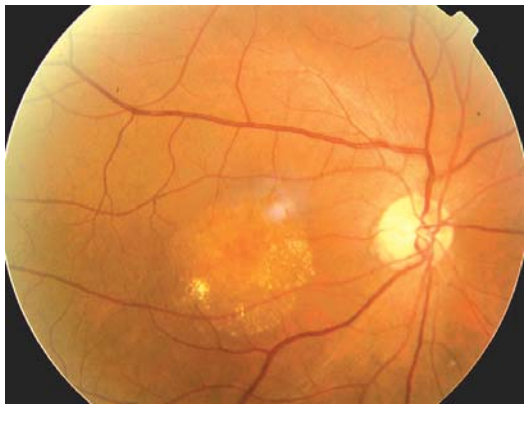


FIGURE 12.42.12: Healed CSR

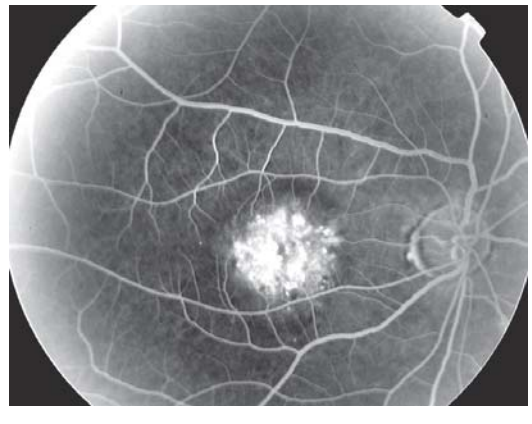


FIGURE 12.42.13: Healed CSR on FFA

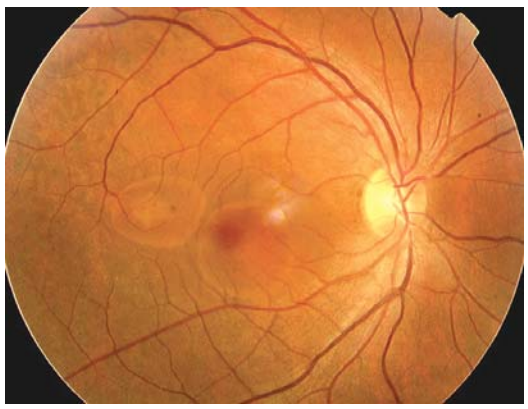


FIGURE 12.43.1: RPE detachment—bilateral

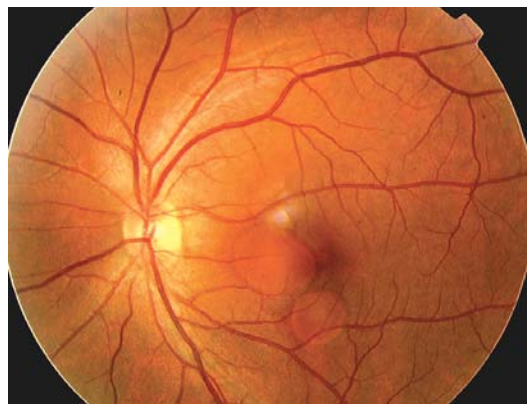


FIGURE 12.43.2: RPE detachment

RPE Detachment

- Separation of RPE from the Bruch's membrane
- Unilateral sharply demarcated dome-shaped lesions of varying size at the posterior pole **(Figs 12.43.1 and 12.43.2)**
- FFA shows area of hyperfluorescence which increases in intensity but not in size in late phase **(Figs 12.43.3 to 12.43.6)**
- There may be hemorrhage inside the detached area **(Fig 12.43.7)**

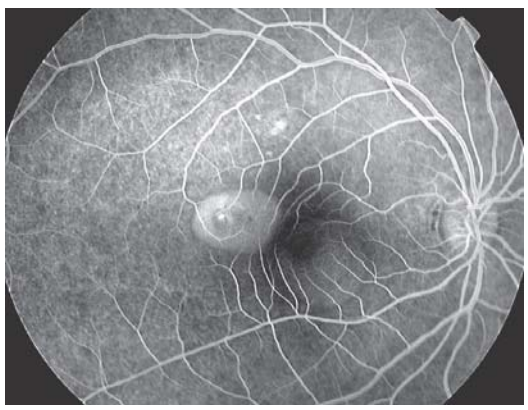


FIGURE 12.43.3: PED—FFA finding

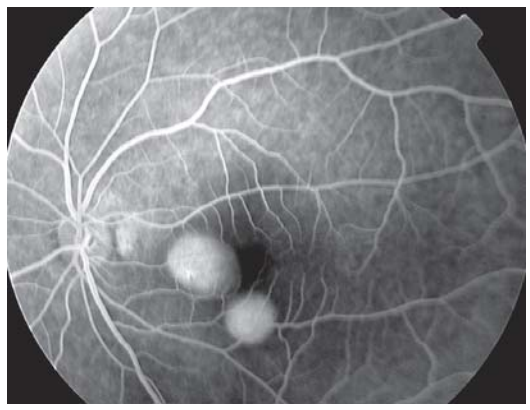


FIGURE 12.43.4: PED—FFA finding

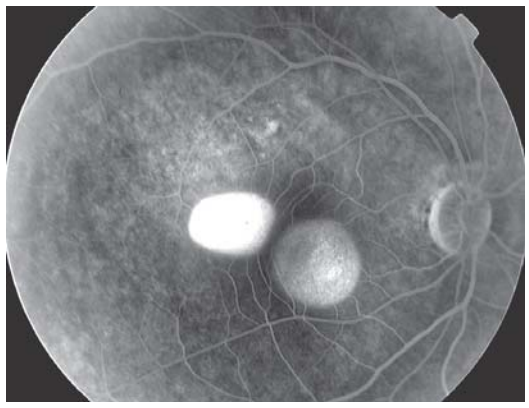


FIGURE 12.43.5: PED on FFA

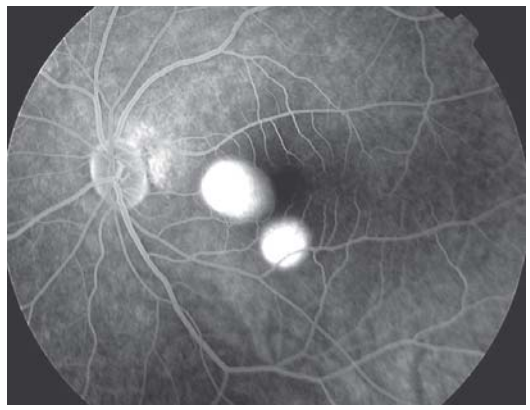


FIGURE 12.43.6: PED on FFA

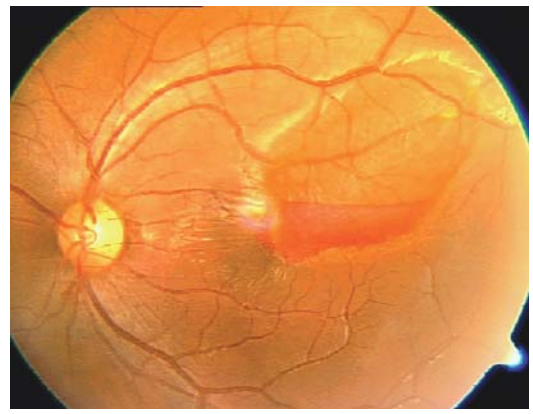


FIGURE 12.43.7: Hemorrhagic PED

Wet Age-related Macular Degeneration

- Wet ARMD is a common bilateral disease affecting the elderly people
- Characterized by formation or subretinal neovascular membranes (SRNVMs) at the macula
- SRNVMs consists of proliferations of fibrovascular tissue from the choriocapillaries **(Figs 12.44.1 to 12.44.4)**
- Clinically appears as pinkish-yellow slightly elevated subretinal lesion of variable size **(Fig 12.44.5)**
- FFA of SRNVMs shows characteristic lacy pattern during the early phase, followed by hyperfluorescence and late leakage **(Figs 12.44.6 to 12.44.8)**
- Extensive subretinal macular fibrosis occurs in end stage **(Figs 12.44.9 and 12.44.10)**

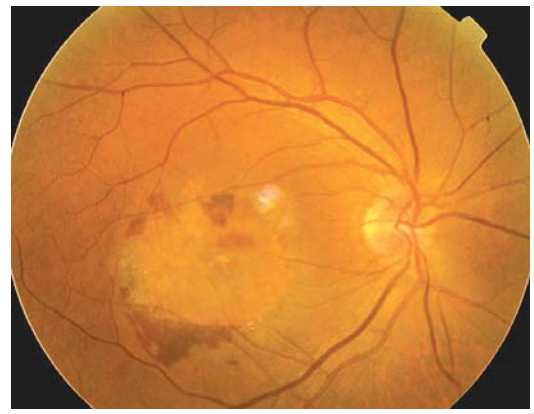


FIGURE 12.44.1: Wet AMD

FIGURE 12.44.2: Wet AMD—bleeding SRNVMs

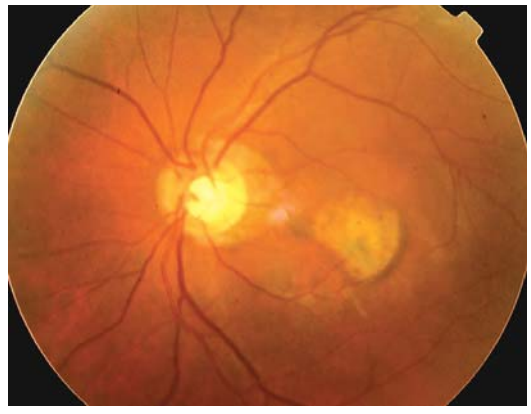


FIGURE 12.44.3: Wet AMD—classical SRNVMs

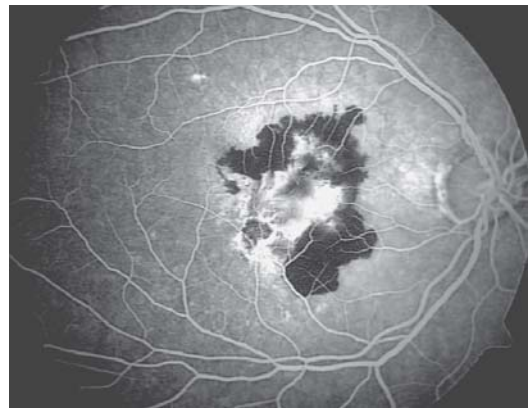


FIGURE 12.44.4: Wet AMD—bleeding SRNVMs

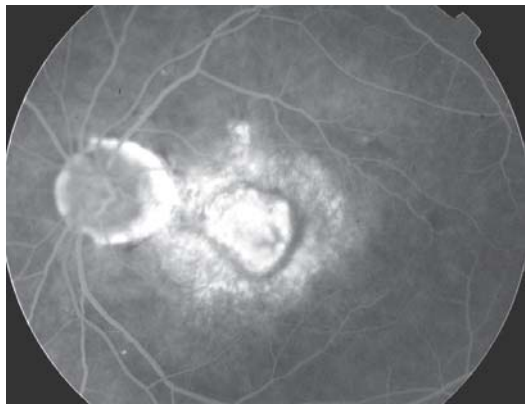


FIGURE 12.44.5: Wet AMD—classical SRNVMs

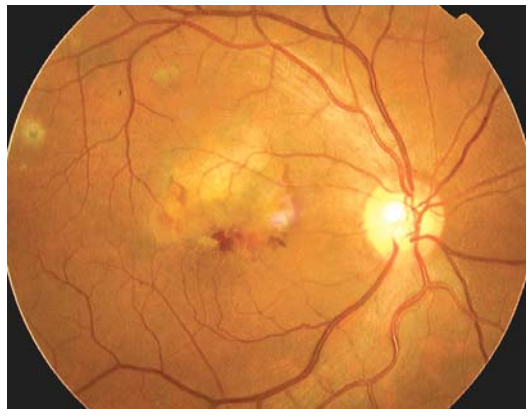


FIGURE 12.44.6: Wet AMD—SRNVMs

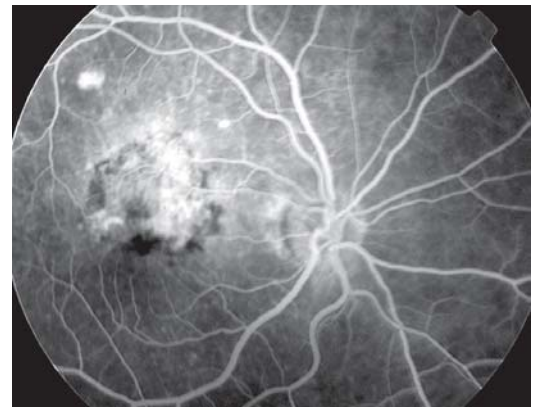


FIGURE 12.44.7: Wet AMD—SRNVMs—FFA

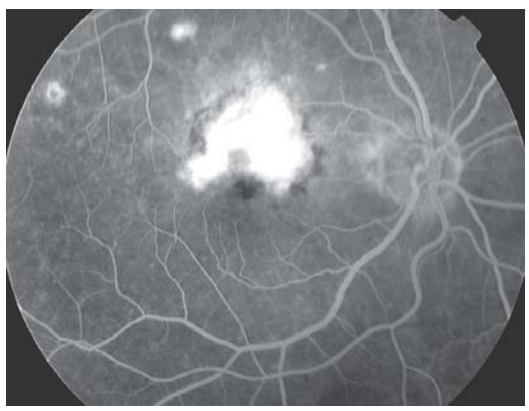


FIGURE 12.44.8: Wet AMD—SRNVMs—FFA

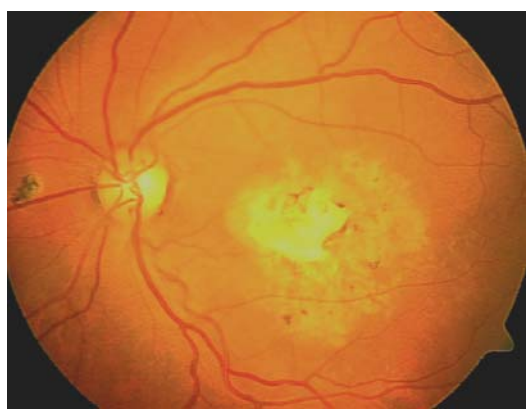


FIGURE 12.44.9: Wet AMD—scarring

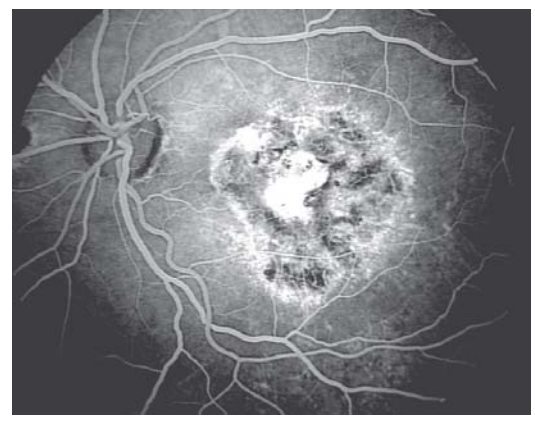


FIGURE 12.44.10: Wet AMD—scarring

Macular Hole

- May be full thickness or lamellar
- *Idiopathic type:*
  - a common age-related condition with bilateral affection in 15 percent cases
  - *Stage I:* yellow foveolar spot with loss of depression
  - *Stage II:* central round foveal defect with an elevated retinal rim **(Fig 12.45.1)**
  - *Stage III:* central round foveal defect with a central smaller operculum **(Fig 12.45.2)**
  - *Stage IV:* central round foveal defect with complete separation of operculum **(Fig 12.45.3)**
  - multiple yellow deposits at the level of RPE within the hole **(Fig 12.45.4)**
  - FFA shows a corresponding circular zone of hypofluorescence **(Figs 12.45.5 and 12.45.6)**
- *Other types:*
  - macular hole in myopia
  - traumatic macular hole **(Fig 16.10.3)**
  - lamellar hole after prolonged CME
- *Pseudohole:* diskontinuity of an epiretinal membrane over the macula **(Fig 12.45.7)**

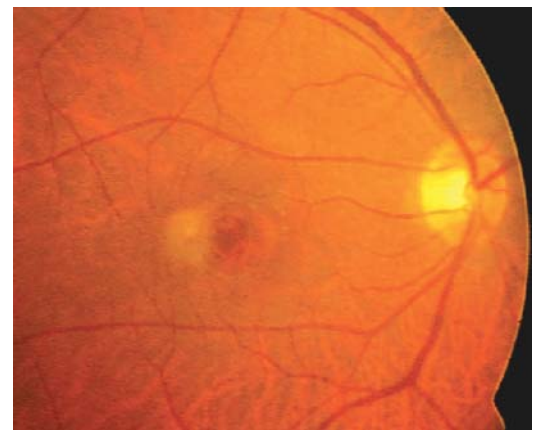


FIGURE 12.45.1: Macular hole—stage II

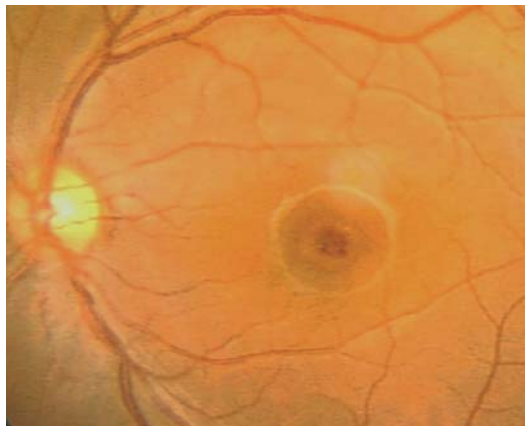


FIGURE 12.45.2: Macular hole—stage III

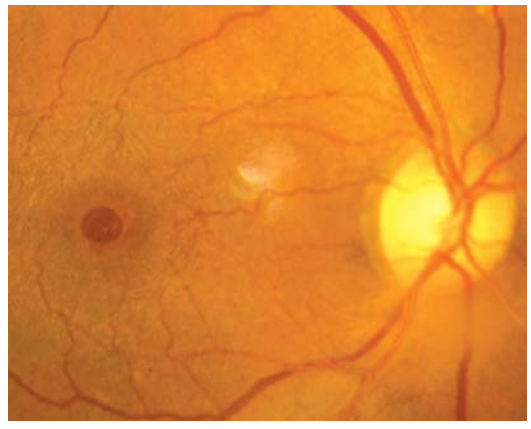


FIGURE 12.45.3: Macular hole—stage IV

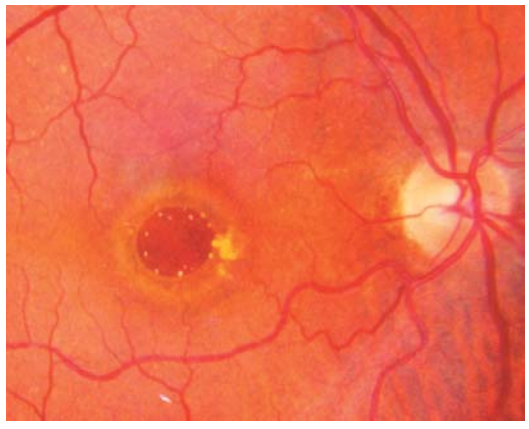


FIGURE 12.45.4: Macularhole—yellow deposits

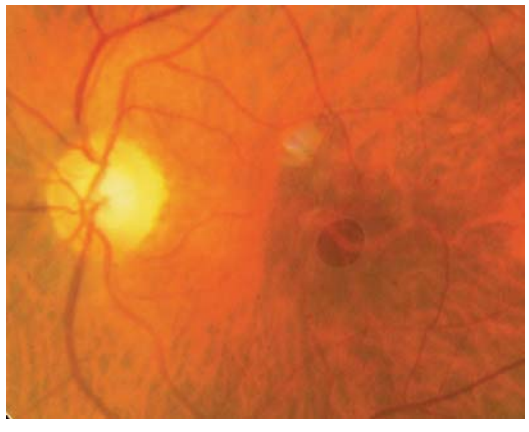


FIGURE 12.45.5: Macular hole

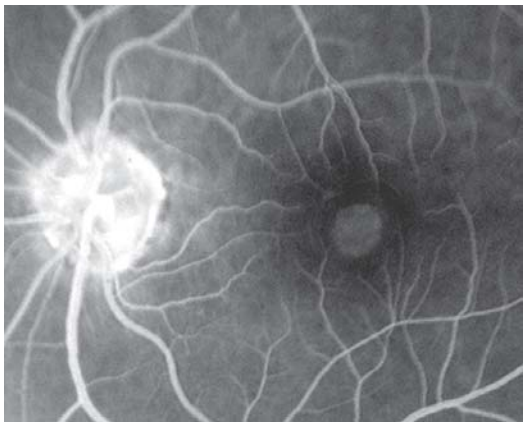


FIGURE 12.45.6: Macular hole FFA

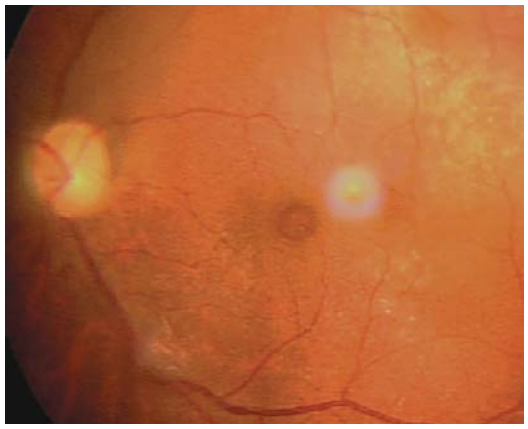


FIGURE 12.45.7: Macular pseudohole

OTHER MACULOPATHIES

Cellophane Maculopathy

- Preretinal macular fibrosis caused by contraction of *epiretinal membrane* over the macula
- Appears as a transparent membrane with fine retinal striation **(Fig 12.46.1)**
- Associated vascular tortuosity is best appreciated by FFA **(Fig 12.46.2)**
- Later the membrane becomes more opaque with more wrinkling **(Figs 12.46.3 to 12.46.5)**

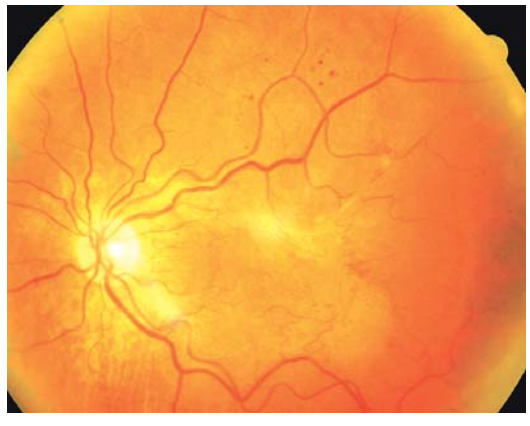


FIGURE 12.46.1: Cellophane maculopathy—epiretinal membrane

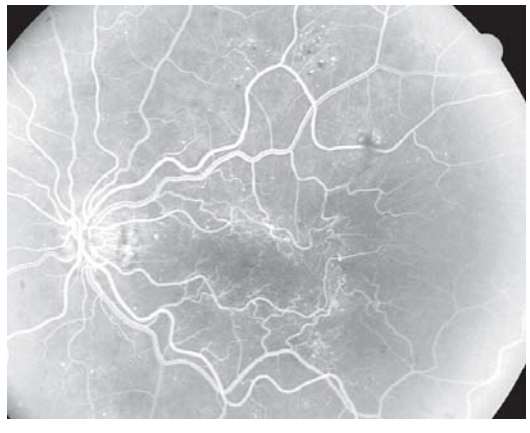


FIGURE 12.46.2: Epiretinal membrane—FFA

FIGURE 12.46.3: Cellophane maculopathy

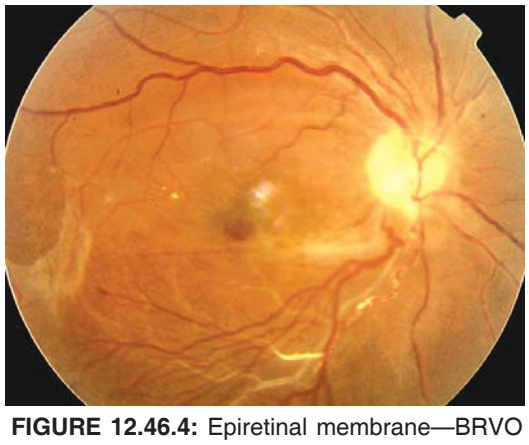


FIGURE 12.46.4: Epiretinal membrane—BRVO

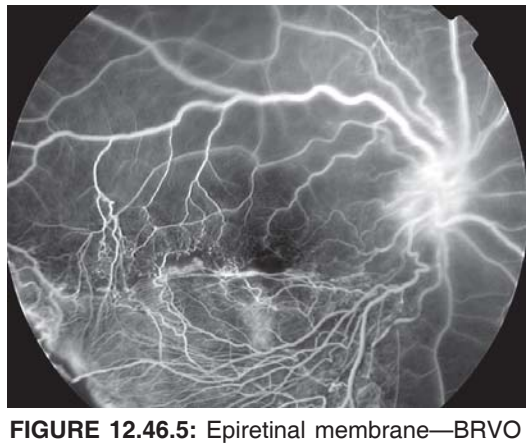


FIGURE 12.46.5: Epiretinal membrane—BRVO on FFA

Commotio Retinae (Berlin's edema)

- Localized macular edema, often with cherry red spot **(Fig 12.47.1)**
- Caused by blunt trauma
- May be associated with disk hemorrhage or retinal hemorrhage **(Fig 12.42.2)**

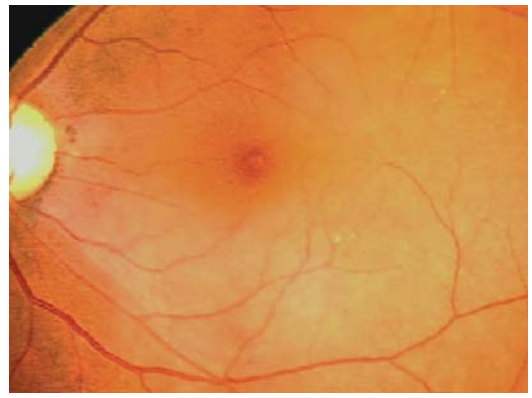


FIGURE 12.47.1: Commotio retinae—Berlin's edema

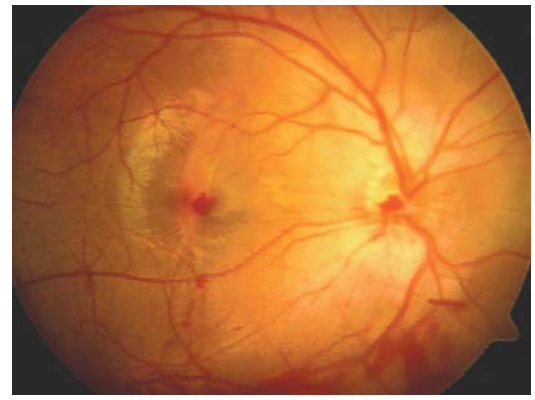


FIGURE 12.47.2: Retinal edema—cricket ball injury

Crystalline Maculopathy

- Multiple tiny glistening crystals at the posterior pole and parafoveal region
- May be drug-induced like, tamoxifen, canthaxanthin, or talc **(Figs 12.48.1 and 12.48.2)**
- May be also seen in metabolic disorder like, cystinosis

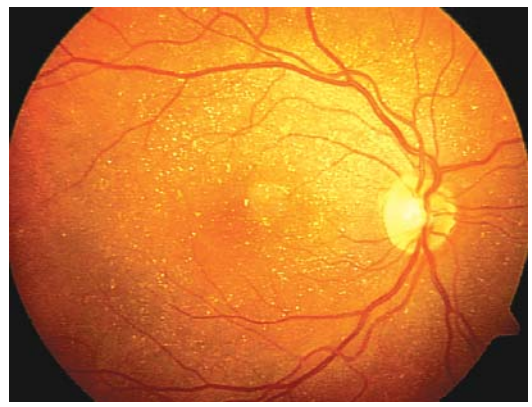


FIGURE 12.48.1: Crystalline maculopathy—tamoxifen

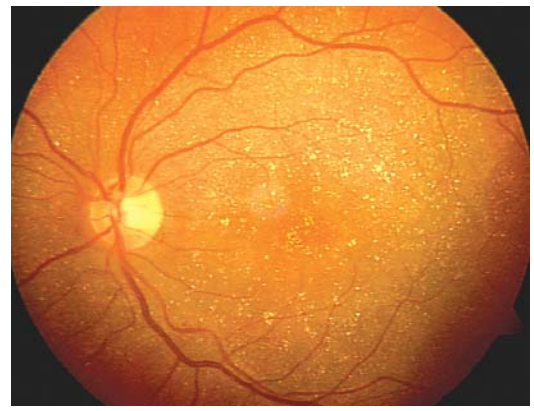


FIGURE 12.48.2: Crystalline maculopathy—tamoxifen

MULTIPLE FLECKED RETINAL LESIONS

Familial Dominant Drusen (Doyne's honeycomb dystrophy)

- Rare condition with dominant inheritance, affects the younger individuals
- Relatively large, diskrete nodular drusen mainly affects the area either temporal to the fovea or nasal to the disk **(Figs 12.49.1 and 12.49.2)**
- Often in a symmetrical distribution
- With time they merge together and become confluent with an appearance of honeycomb
- In late stage pigmentary changes occur with loss of central vision

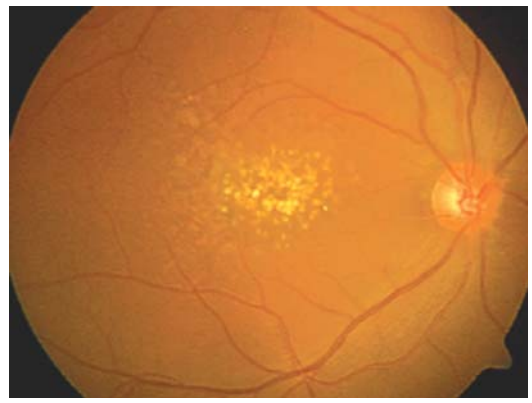


FIGURE 12.49.1: Familial drusens

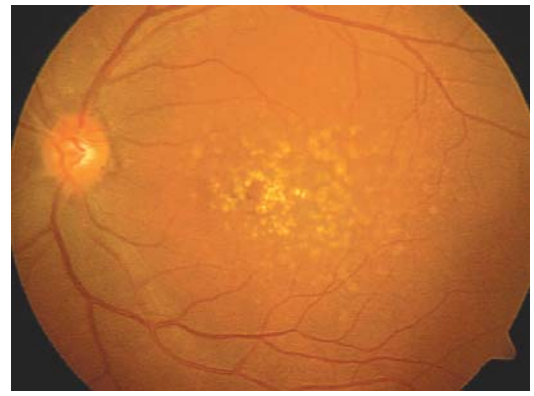


FIGURE 12.49.2: Familial drusens

Fundus Flavimaculatus

- Rare bilateral condition with recessive inheritance
- Yellowish-white fish-tail like flecks at the posterior pole and midperiphral retina **(Figs 12.50.1 and 12.50.2)**
- Flecks may occur in isolation or with maculopathy, as in Stargardt's disease **(Figs 12.35.7 and 12.35.8)**
- FFA shows hyperfluorescence due to atrophic changes

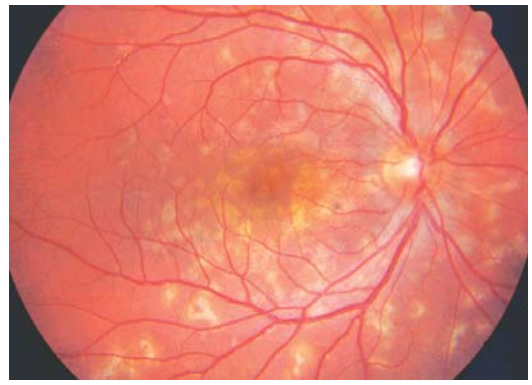


FIGURE 12.50.1: Fundus flavimaculatus

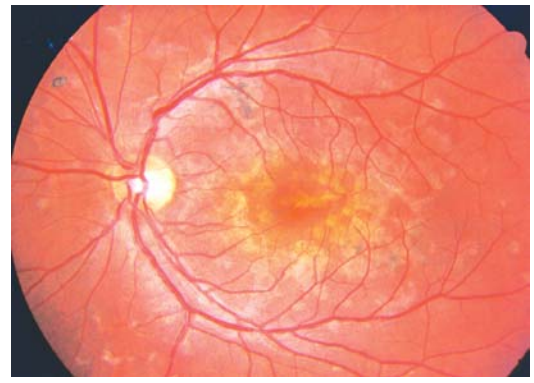


FIGURE 12.50.2: Fundus flavimaculatus

Fundus Albipunctatus

- Rare, bilateral recessively inherited condition with congenital stationary night blindness
- Multiple tiny yellowish-white dots from posterior pole to the periphery **(Figs 12.51.1 and 12.51.2)**
- Macula is spared and visual acuity remains normal

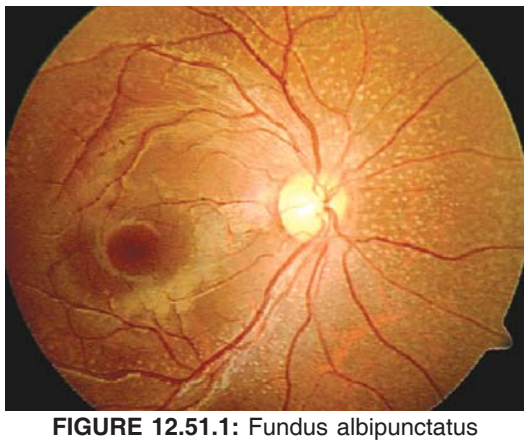


FIGURE 12.51.1: Fundus albipunctatus

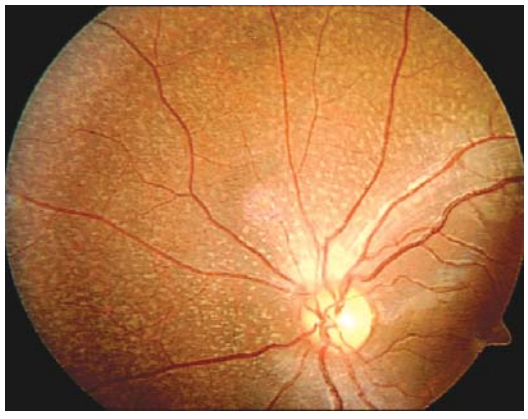


FIGURE 12.51.2: Fundus albipunctatus

Benign Flecked Retina Syndrome

- Very rare, bilateral condition
- Multiple yellowish-white flecks all over the fundus
- Similar to fundus flavimaculatus, but the macula is spared **(Figs 12.52.1 and 12.52.2)**
- FFA shows the apparent lesions **(Figs 12.52.3 and 12.52.4)**
- Prognosis is excellent with normal vision

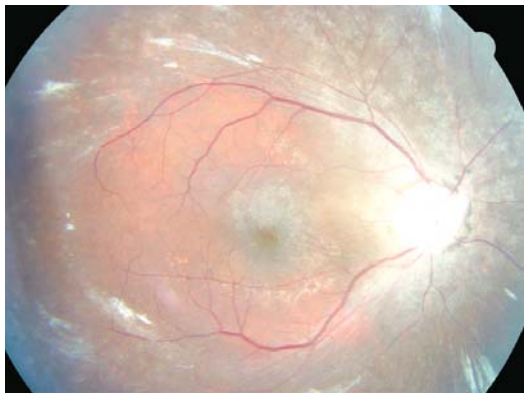


FIGURE 12.52.1: Flecked retina syndrome

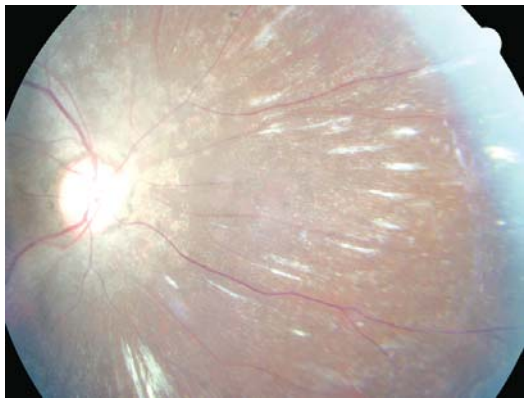


FIGURE 12.52.2: Flecked retina syndrome

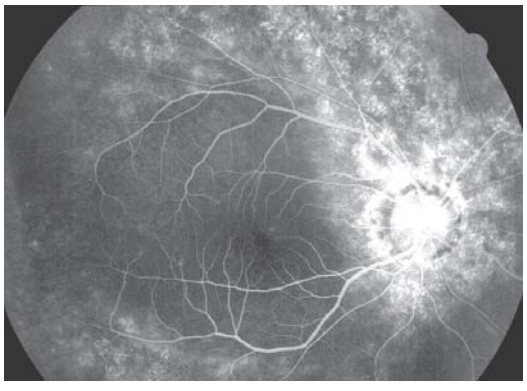


FIGURE 12.52.3: Flecked retina syndrome

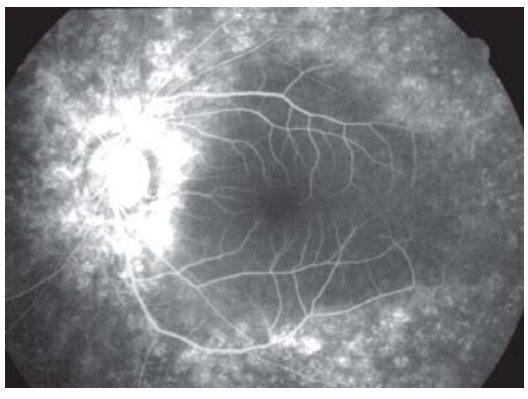


FIGURE 12.52.4: Flecked retina syndrome

Pattern Macular Dystrophy

- Very rare, bilateral condition with dominant inheritance
- Slowly progressive symmetrical lesions with various geometrical configurations **(Figs 12.53.1 and 12.53.2)**
- Prognosis is relatively good

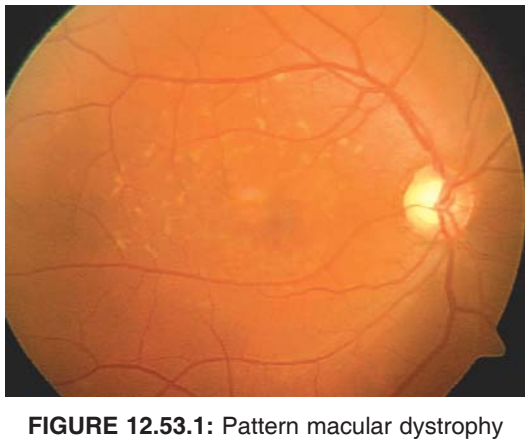


FIGURE 12.53.1: Pattern macular dystrophy

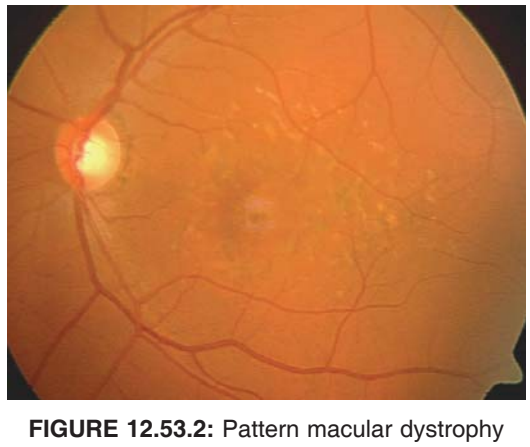


FIGURE 12.53.2: Pattern macular dystrophy

Hard Drusens

- Very common age-related condition
- Usually not associated with subsequent development of macular degeneration
- Multiple, bilaterally symmetrical, small, diskrete, yellow-white, slightly elevated lesions at the posterior poles of both fundi **(Figs 12.54.1 to 12.54.4)**
- *Calcified drusen:* secondary calcification in long standing cases gives them glistening-white appearance with conspicuous margin **(Figs 12.54.5 to 12.54.8)**
- *Hard and Calcified drusen* may exist together **(Figs 12.54.9 and 12.54.10)**
- FFA shows multiple RPE-window defects with hyperfluorescence spots, and late staining of the drusen

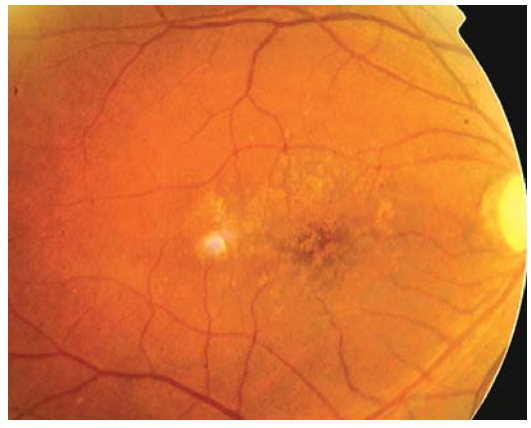


FIGURE 12.54.1: Hard drusens

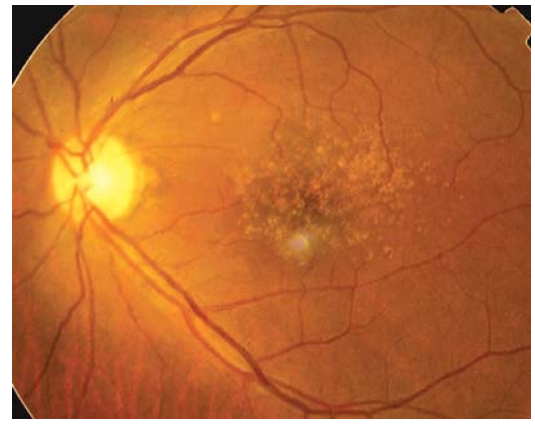


FIGURE 12.54.2: Hard drusens

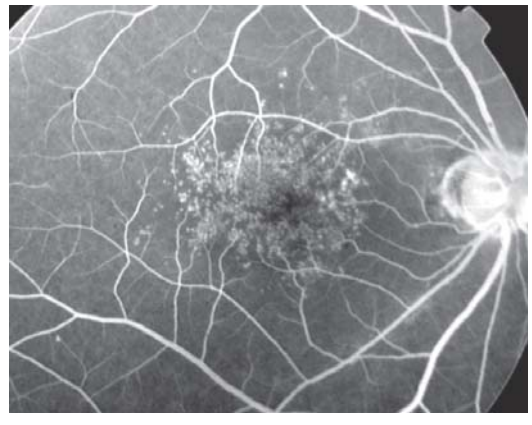


FIGURE 12.54.3: Hard drusens on FFA

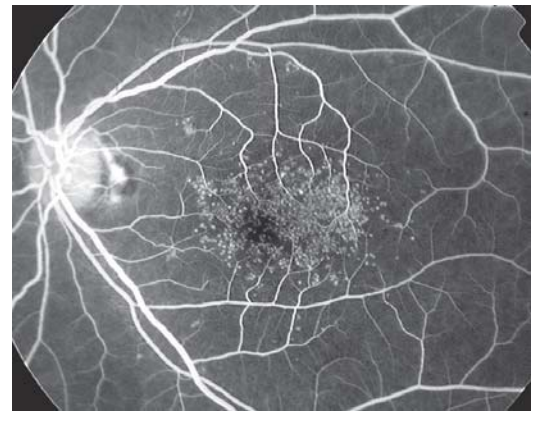


FIGURE 12.54.4: Hard drusens on FFA

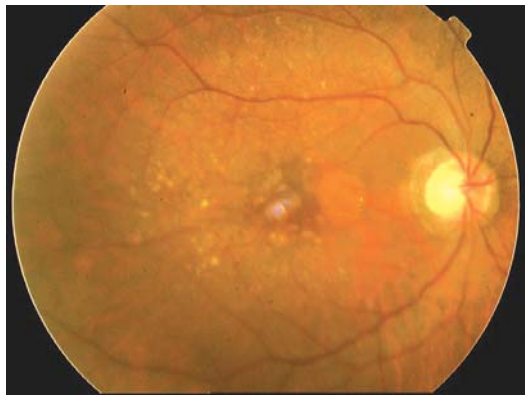


FIGURE 12.54.5: Hard and calcified drusens

FIGURE 12.54.6: Hard and calcified drusens

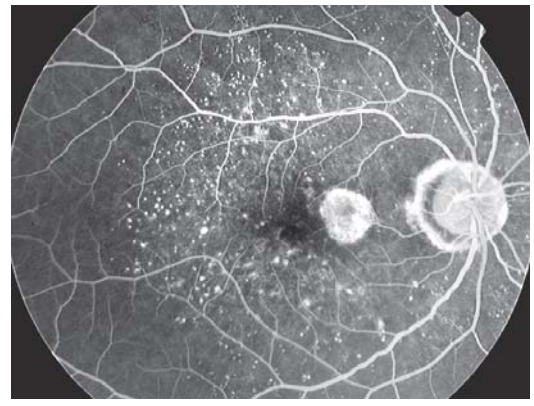


FIGURE 12.54.7: Hard and calcified drusens on FFA

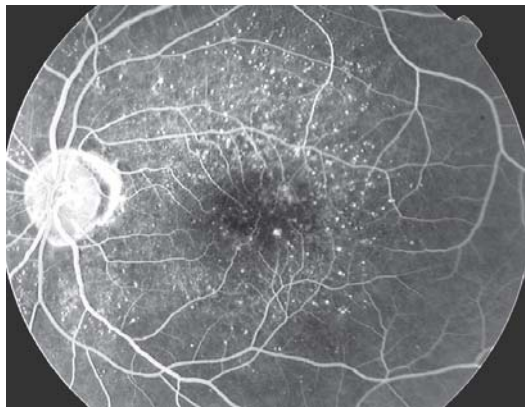


FIGURE 12.54.8: Hard and calcified drusens on FFA

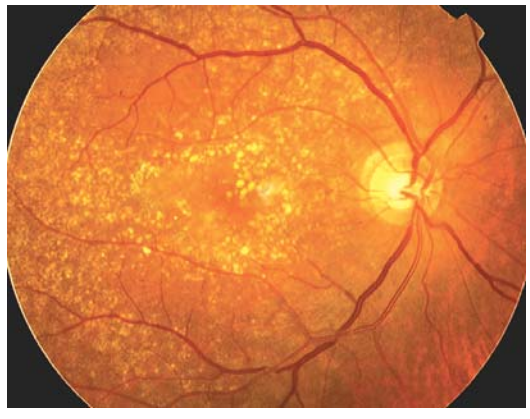


FIGURE 12.54.9: Hard and calcified drusens

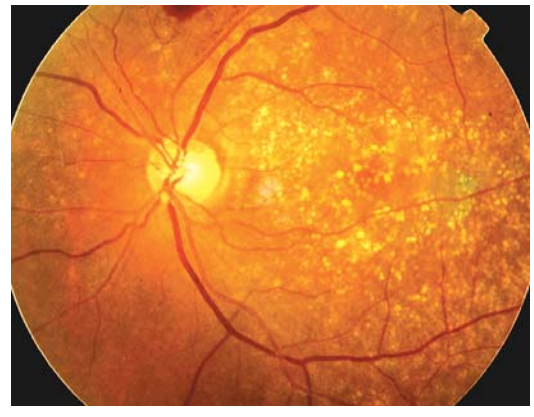


FIGURE 12.54.10: Hard and calcified drusens

Soft Drusens

- Common age-related lesions associated with increased risk of ARMD
- Are larger than hard drusen with indistinct edges **(Figs 12.55.1 and 12.55.2)**
- Become confluent with time **(Fig 12.55.3)**
- Lesions are often asymmetrical and secondary RPE changes are quite common
- FFA shows hyperfluorescence
- Sometimes, they may be found along with hard drusen

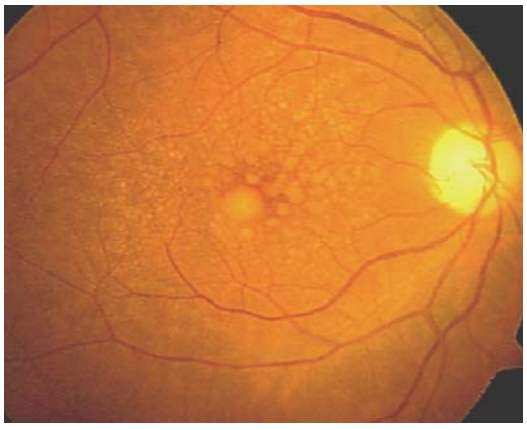


FIGURE 12.55.1: Soft drusens

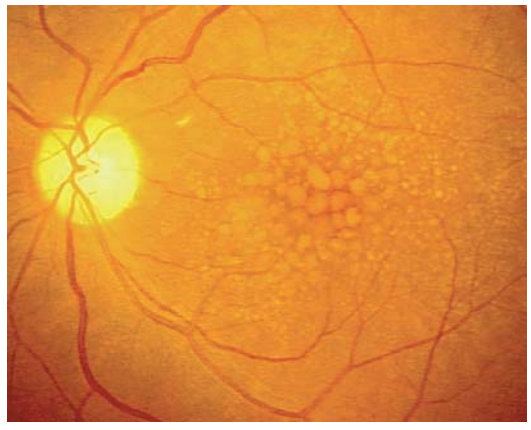


FIGURE 12.55.2: Soft drusens

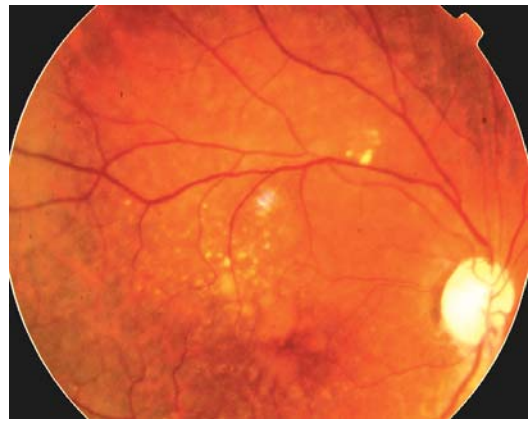


FIGURE 12.55.3: Soft drusens—confluent

Cuticular (Basal-laminer) Drusens

- Rare kind of drusens affects the middle aged person
- Bilaterally symmetrical, innumerable, small uniform, diskrete round slightly elevated yellowish subretinal lesions all over the posterior pole **(Figs 12.56.1 and 12.56.2)**
- 'Starry night' or 'Milky way' appearance of the fundus which can be appreciated better on FFA
- In late stage, there are pigmentary changes in the macula

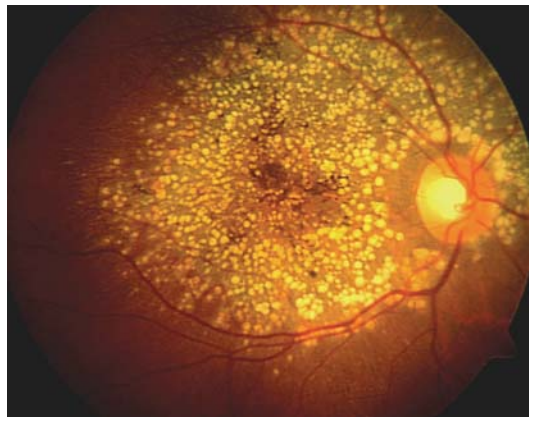


FIGURE 12.56.1: Cuticular drusens

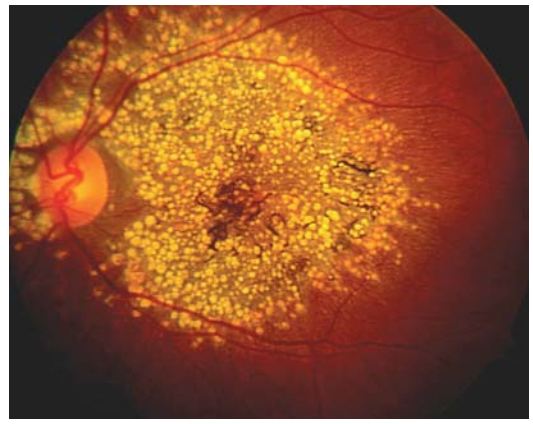


FIGURE 12.56.2: Cuticular drusens

PIGMENTARY RETINOPATHY

Typical Retinitis Pigmentosa

- Typical RP is a bilateral, symmetrical, progressive diffuse pigmentary retinal dystrophy which predominantly affecting the rods **(Figs 12.57.1 and 12.57.2)**
- Patients usually present in the second decade of life and ultimately have severe visual loss in later life

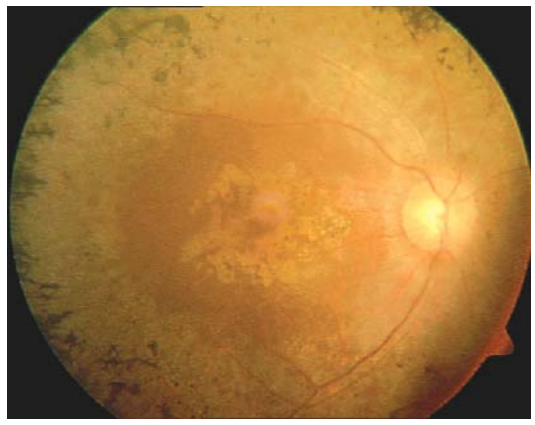


FIGURE 12.57.1: Retinitis pigmentosa

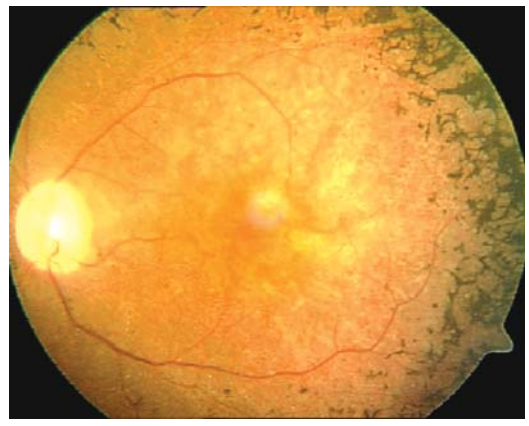


FIGURE 12.57.2: Retinitis pigmentosa

- *Classical triad of RP:*
  - bony-spicule pigmentation **(Fig 12.57.3)**
  - arteriolar attenuation and
  - waxy-pallor of the optic disk
- Pigmentary changes are typically perivascular, and have a bone-spicule appearance, which are observed mostly at the equatorial region of the retina **(Fig 12.57.4)**
- In late stages of the disease, the unmasking of the larger choroidal vessels gives the fundus a tessellated appearance
- Cystoid *macular oedema* **(Figs 12.57.5 and 12.57.6)**, *cellophane or atrophic maculopathy* **(Fig 12.57.7)** may be present upto 70 percent of the patients with RP

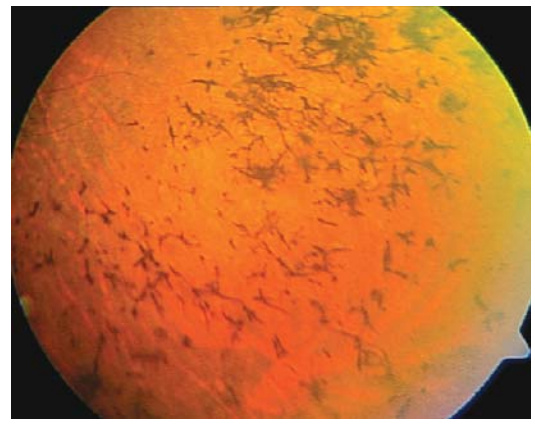


FIGURE 12.57.3: Retinitis pigmentosa

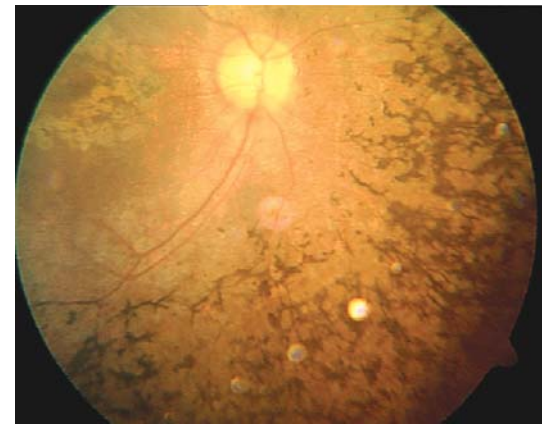


FIGURE 12.57.4: Retinitis pigmentosa

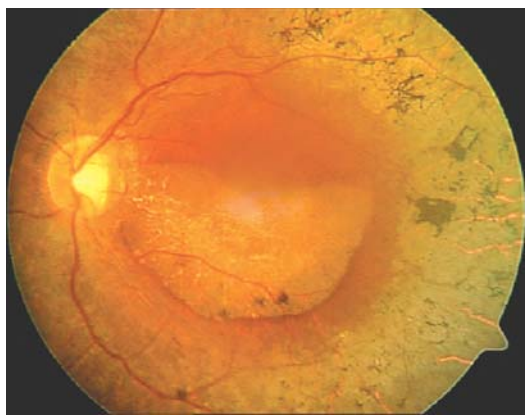


FIGURE 12.57.5: Retinitis pigmentosa—CME

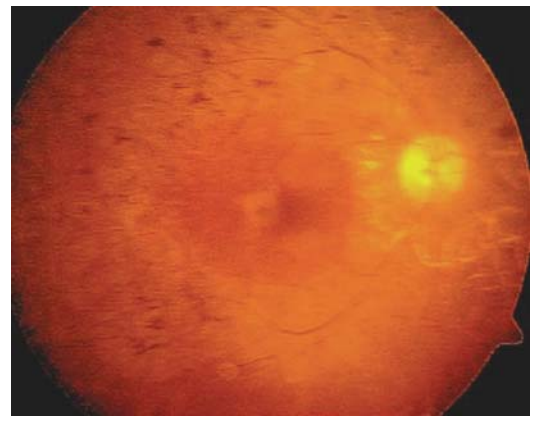


FIGURE 12.57.6: Retinitis pigmentosa—CME

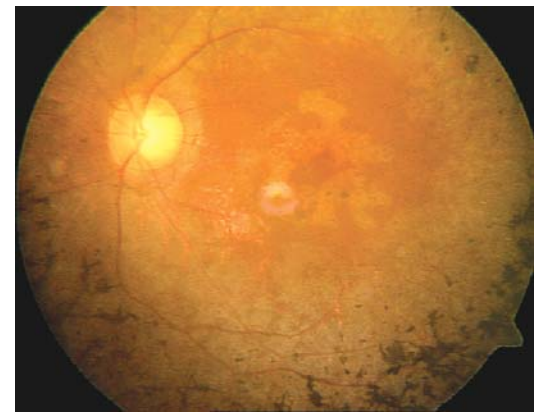


FIGURE 12.57.7: Retinitis pigmentosa—atrophic maculopathy

Atypical Retinitis Pigmentosa

- *Retinitis punctata albescens:* characterized by multiple scattered white dots, mostly between the posterior pole and the equator. Other findings are similar to typical RP **(Figs 12.58.1 to 12.58.3)**
- *Retinitis pigmentosa sine pigmento:* presence of arteriolar attenuation and waxy-pallor of the disk with subnormal ERG
- *Unilateral retinitis pigmentosa* **(Figs 12.58.4 and 12.58.5)**

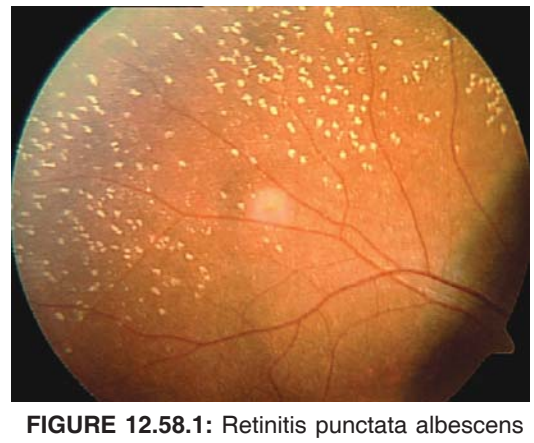


FIGURE 12.58.1: Retinitis punctata albescens

FIGURE 12.58.2: Retinitis punctata albescens

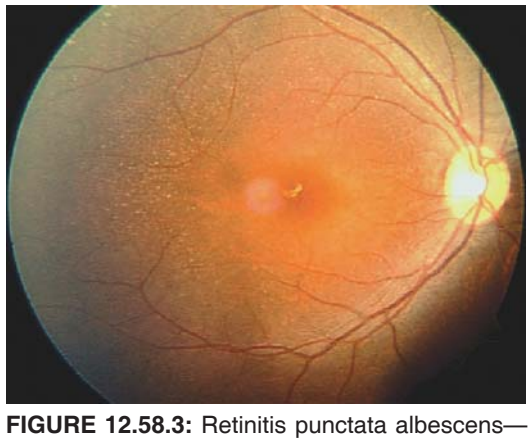


FIGURE 12.58.3: Retinitis punctata albescens—CME

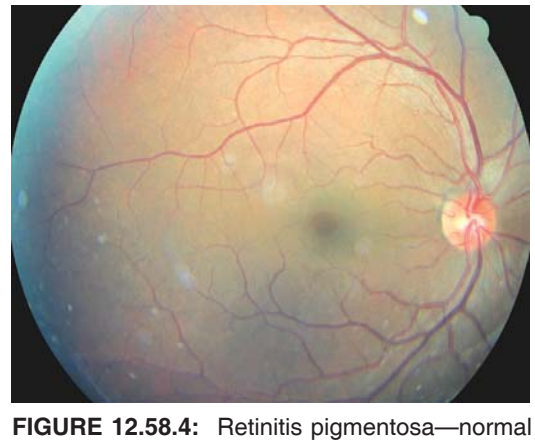


FIGURE 12.58.4: Retinitis pigmentosa—normal eye

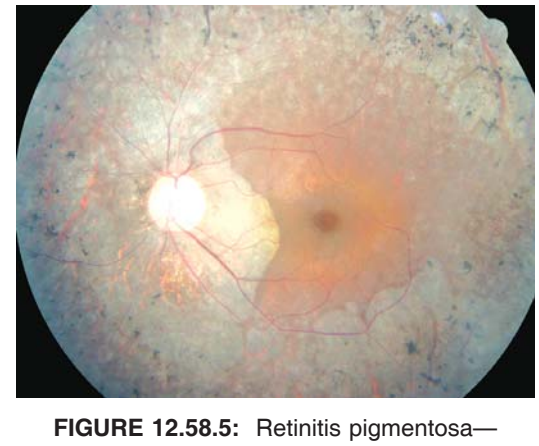


FIGURE 12.58.5: Retinitis pigmentosa—unilateral

- *Sectorial retinitis pigmentosa:* one-quadrant or one half (usually inferior) of the fundus **(Figs 12.58.6 and 12.58.7)**
- *Pericentricor central retinitis pigmentosa:* RP, except that the pigmentary changes are confined to central or pericentral area, sparing the periphery **(Figs 12.58.8 and 12.58.9)**
- *With systemic association*
  - *Laurence-Moon-Biedl syndrome:* RP, mental retardation, obesity, polydactyly and hypogonadism **(Fig 12.58.10)**
  - *Refsum's disease:* RP, peripheral neuropathy, deafness, ataxia and ecthyosis
  - *Bassen-Kornzweig syndrome:* RP, ataxia, acanthocytosis, fat malabsorption
  - *Usher syndrome:* RP and sensory-neural deafness
  - *Cocayne's syndrome:* RP, dwarfism, bird-like face, ataxia, mental retardation and premature aging
  - *Kearn-Sayre syndrome:* RP, ocular myopathy (CPEO) and heart block

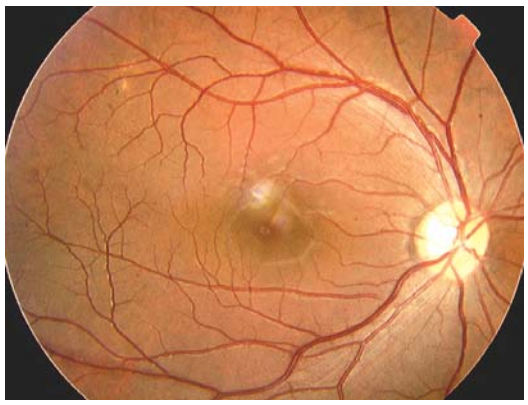


FIGURE 12.58.6: Retinitis pigmentosa—normal eye

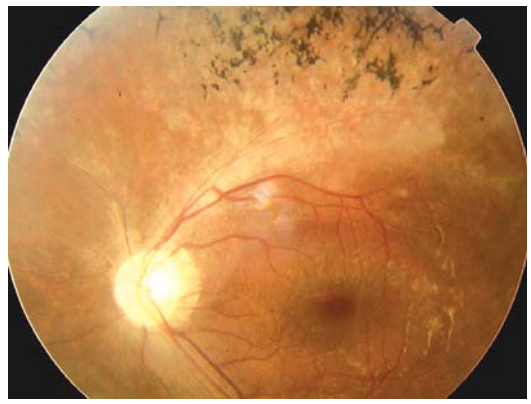


FIGURE 12.58.7: Retinitis pigmentosa—unilateral sectorial

FIGURE 12.58.10: Retinitis pigmentosa—LMB syndrome

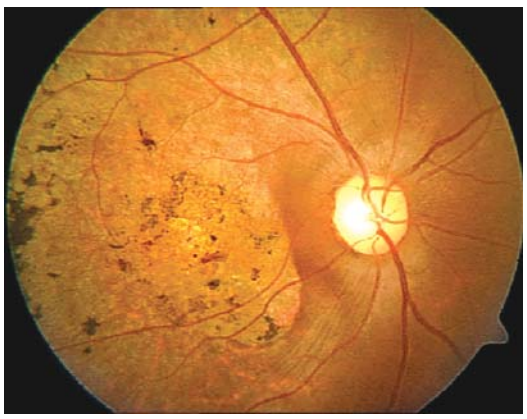


FIGURE 12.58.8: Retinitis pigmentosa—central

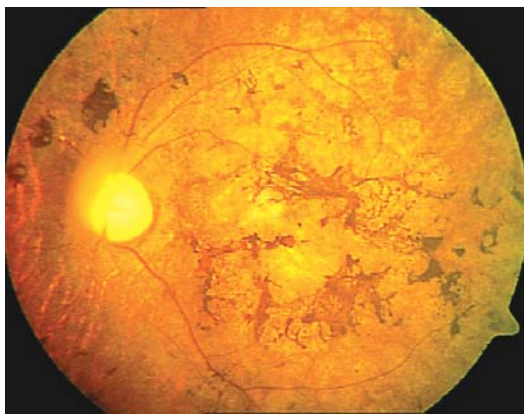


FIGURE 12.58.9: Retinitis pigmentosa—central

RETINAL DETACHMENT

Rhegmatogenous Retinal Detachment

- Detached retina is slightly opaque, convex and corrugated in appearance with loss of underlying choroidal pattern **(Fig 12.59.1)**
- Blood vessels appear darker than normal attached retina **(Fig 12.59.2)**
- Detached retina undulates freely with ocular movements **(Figs 12.59.3 to 12.59.6)**

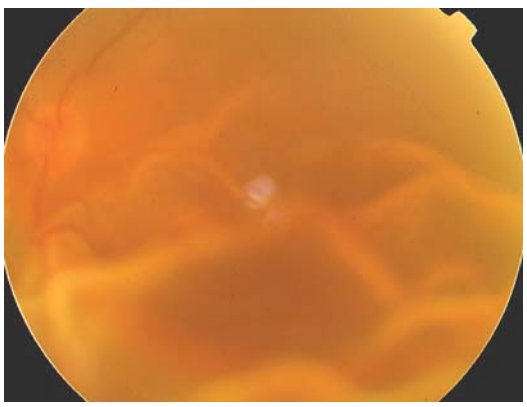


FIGURE 12.59.1: Rhegmatogenous retinal detachment

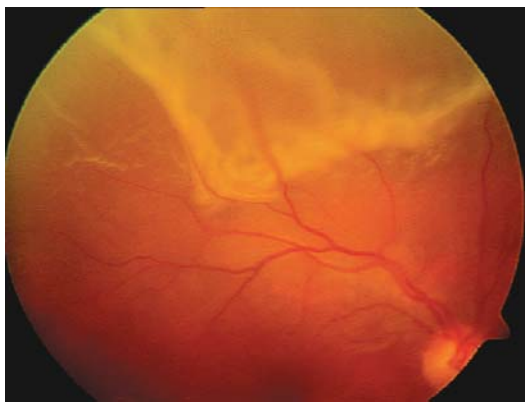


FIGURE 12.59.2: Rhegmatogenous retinal detachment—superior RD—macula on

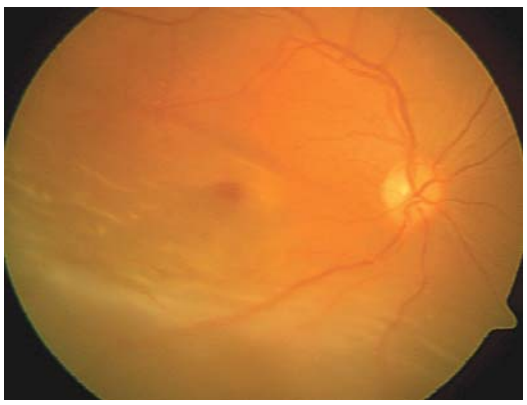


FIGURE 12.59.3: Rhegmatogenous retinal detachment

- Vitreous shows 'tobacco dust' in the anterior vitreous, with posterior vitreous detachment
- Breaks appear as red areas (holes or tears) of diskontinuity mainly in the peripheral retina
- Sometimes, associated with choroidal detachment **(Figs 12.59.7 and 12.59.8)**
- *In long standing cases:*
  - retinal thinning with demarcation lines **(Fig 12.59.9)**
  - secondary intraretinal cyst and fibrosis **(Fig 12.59.10)**
  - proliferative vitreoretinopathy (PVR) develops depending upon the duration of the detachment **(Fig 12.59.11)**
- Retinal dialysys may occur sometimes **(Fig 12.59.12)**
- If untreated, RDs become total and eventually gives rise to complicated cataract, chronic uveitis, hypotony and phthisis bulbi

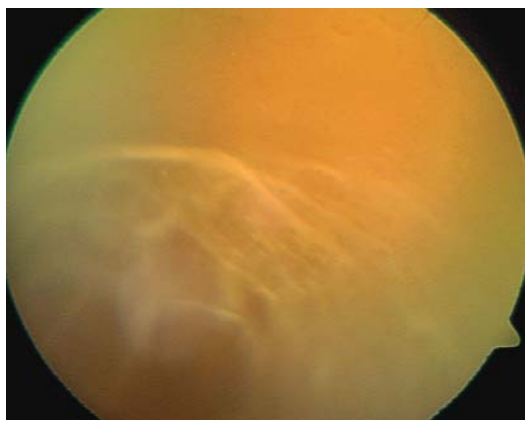


FIGURE 12.59.4: Rhegmatogenous retinal detachment

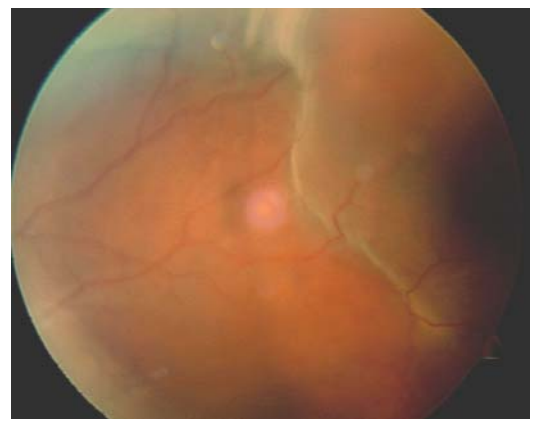


FIGURE 12.59.5: Rhegmatogenous retinal detachment

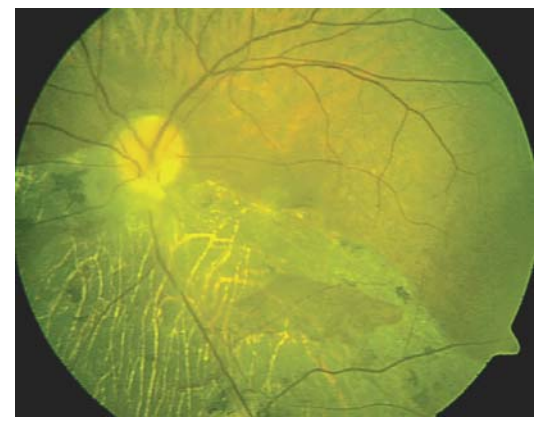


FIGURE 12.59.6: Rhegmatogenous RD—inferior

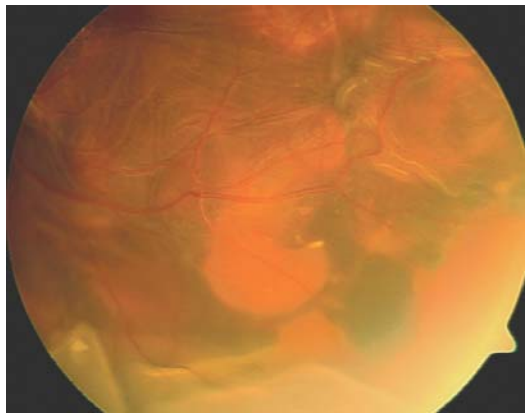


FIGURE 12.59.7: Rhegmatogenous RD—choroidal detachment

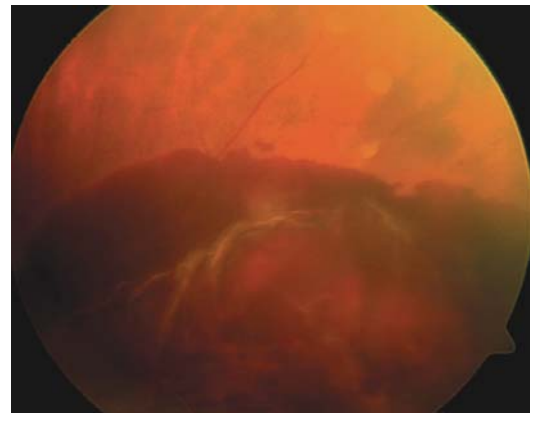


FIGURE 12.59.8: Rhegmatogenous RD—choroidal detachment

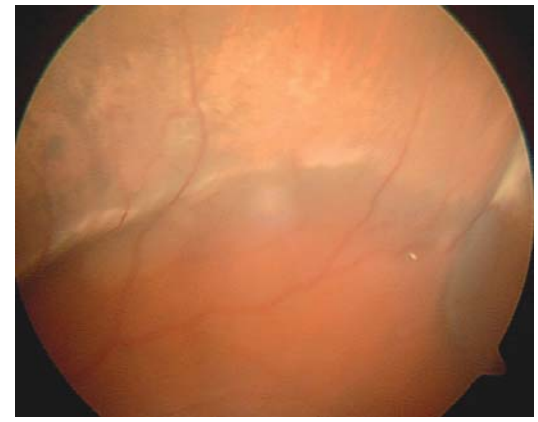


FIGURE 12.59.9: Rhegmatogenous RD—demarcation line

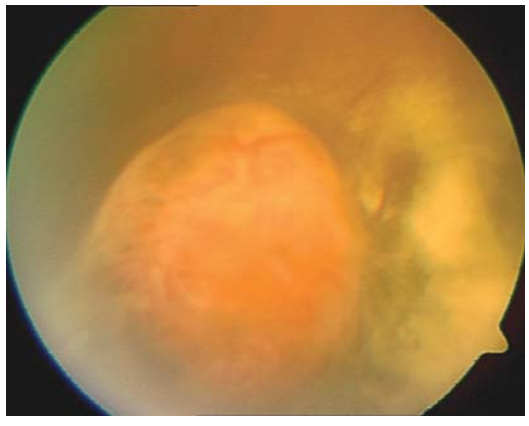


FIGURE 12.59.10: Rhegmatogenous RD—intraretinal cyst

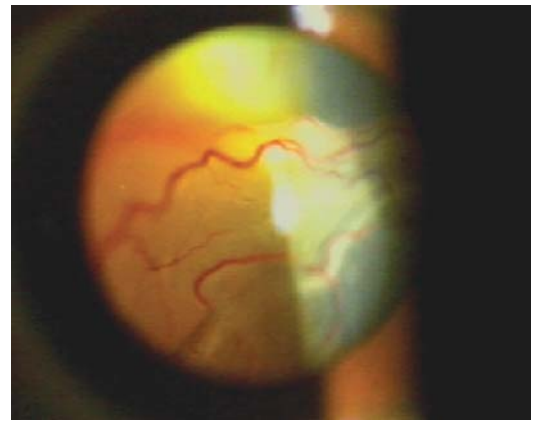


FIGURE 12.59.11: Retinal detachment—PVR changes

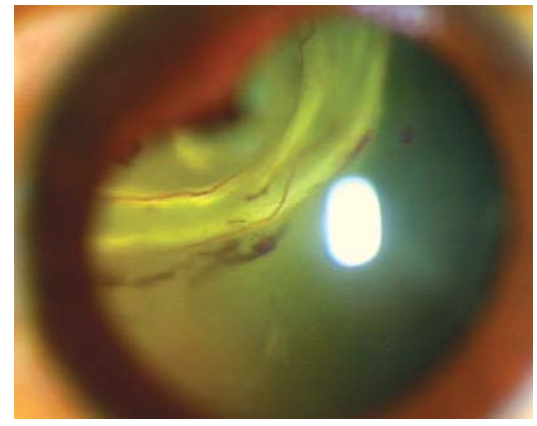


FIGURE 12.59.12: Rhegmatogenous RD—retinal dialysis

Associated Predisposing Factors

- *Lattice degeneration:* peripheral, sharply demarcated, spindle shaped arborising white lines arranged in a lattice pattern, often with variable pigmentation
- *Snail-tract degeneration:* peripheral, sharply demarcated tightly packed white snow flecks bands which appears frosted
- *Retinal holes:* a small break, usually circular, without any vitreous traction **(Fig 12.60.1)**
- *Retinal tear:* a retinal break, usually horse-shoe shaped, with vitreous tractional element. A tear is more dangerous than a hole **(Fig 12.60.2)**
- There may be giant retinal tear **(Fig 12.60.3)**

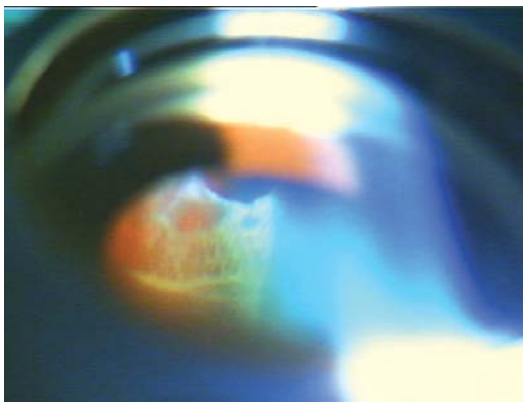


FIGURE 12.60.1: Rhegmatogenous RD—hole

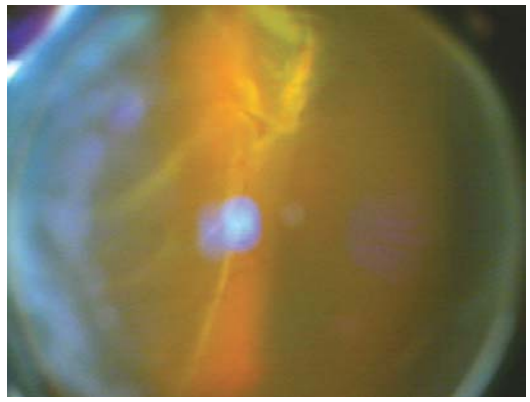


FIGURE 12.60.2: Rhegmatogenous RD—horse-shoe tear

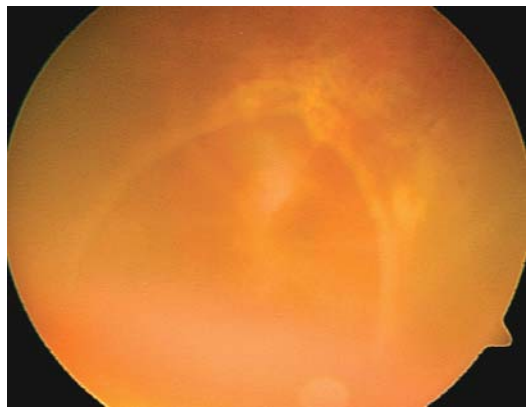


FIGURE 12.60.3: Rhegmatogenous RD—giant retinal tear

Tractional Retinal Detachment

- Configuration of detached retina is concave
- Shallow detachment which seldom extends beyond equator
- Highest elevation of retina occurs at the site of vitreoretinal traction
- Mobility of detached retina is severely reduced
- Retinal breaks are usually absent
- *Causes:* proliferative retinopathies **(Figs 12.61.1 and 12.61.2)**, penetrating injuries **(Fig 12.61.3)** and severe intraocular inflammation **(Figs 12.61.4 and 12.61.5)**

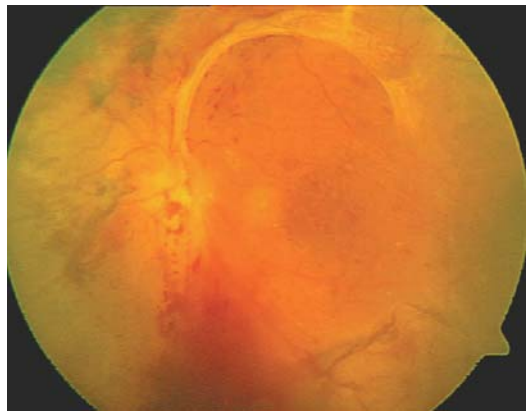


FIGURE 12.61.1: Tractinal RD—diabetic

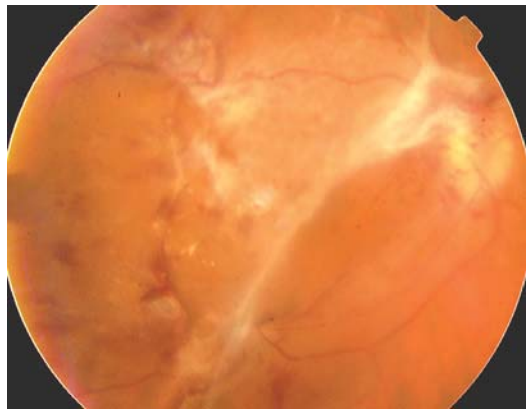


FIGURE 12.61.2: Tractional RD—PDR

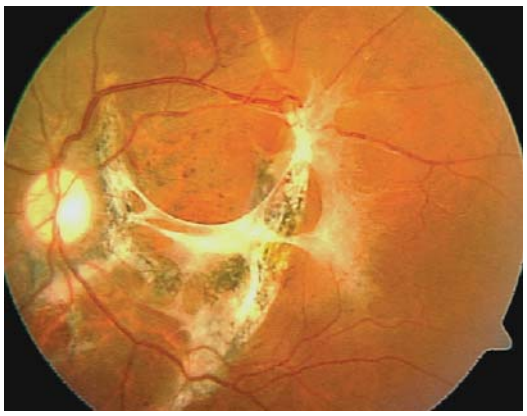


FIGURE 12.61.3: Tractional RD—traumatic

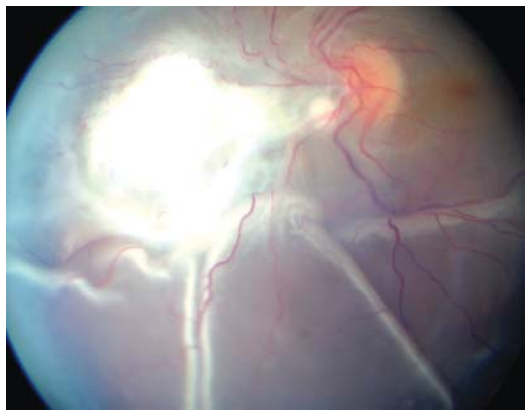


FIGURE 12.61.4: Tractional RD—inflammatory

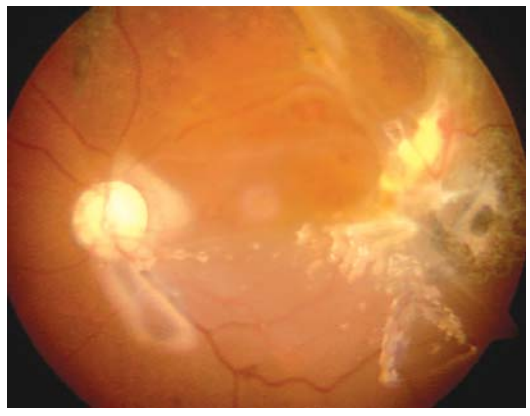


FIGURE 12.61.5: Tractional RD—inflammatory

Exudative Retinal Detachment

- RD configuration is very much convex
- Detached retina is smooth, non-corrugated and bullous **(Fig 12.62.1)**
- May even touch the back of the lens in severe cases
- Marked retinal mobility
- Shifting of fluid is the hallmark of exudative detachment due to force of gravity
- Retinal breaks are absent
- Obvious ocular pathology is frequently evident **(Fig 12.62.2)**
- *Causes:* choroidal tumors, VKH syndrome, posterior scleritis, hypertension, toxemia of pregnancy, uveal effusion syndrome, etc.

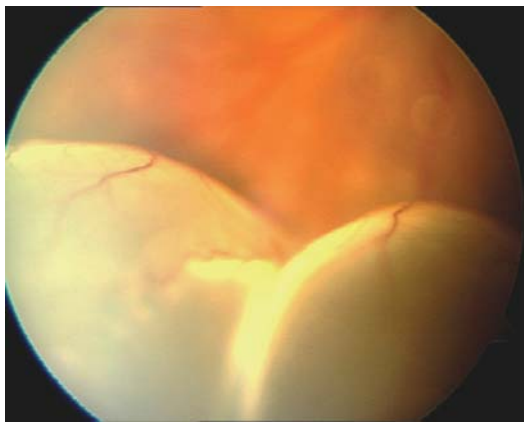


FIGURE 12.62.1: Exudative RD—bullous detachment

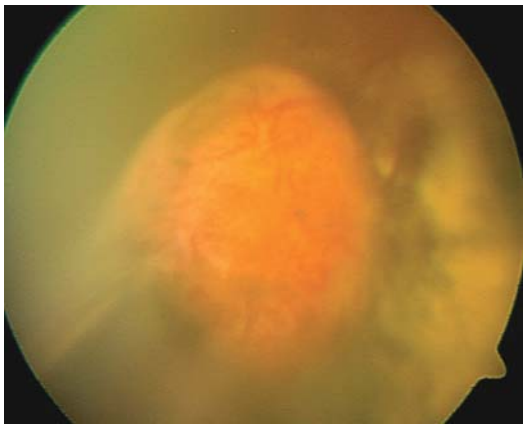


FIGURE 12.62.2: Exudative RD—choroidal melanoma

HYPERTENSIVE RETINOPATHY

- Focal retinal edema, retinal hemorrhage and hard exudates in chronic hypertension
- Hard exudates often deposit around the macula as macular star **(Figs 12.63.1 and 12.63.2)**
- Cotton-wool patches in acute or severe hypertension
- Papilledema including the neuroretinal edema may occur in malignant hypertension **(Figs 12.63.3 and 12.63.4)**
- Various types of arteriovenous crossing changes
- Associated choroidal changes like, RPE changes, pigment deposition along the blocked choroidal vessels, Elschnig's spot, etc.
- Exudative bullous detachment in severe cases

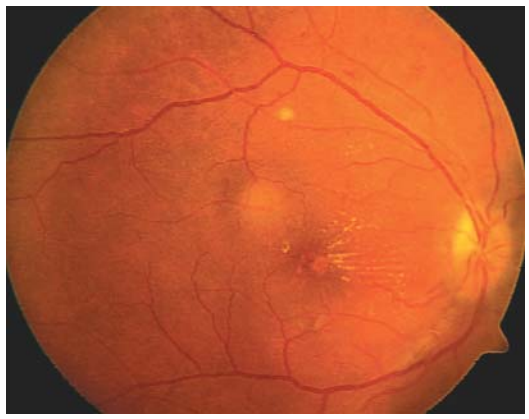


FIGURE 12.63.1: Hypertensive retinopathy

FIGURE 12.63.2: Hypertensive retinopathy—macular star

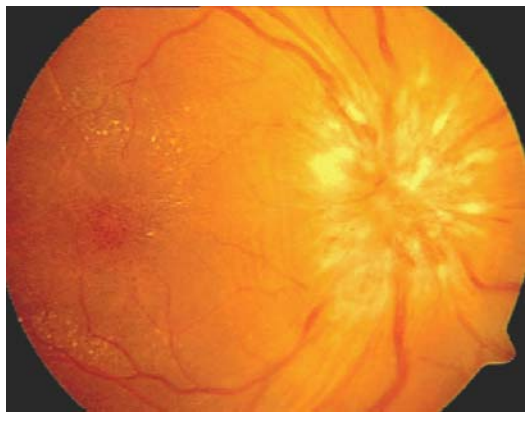


FIGURE 12.63.3: Malignant hypertension

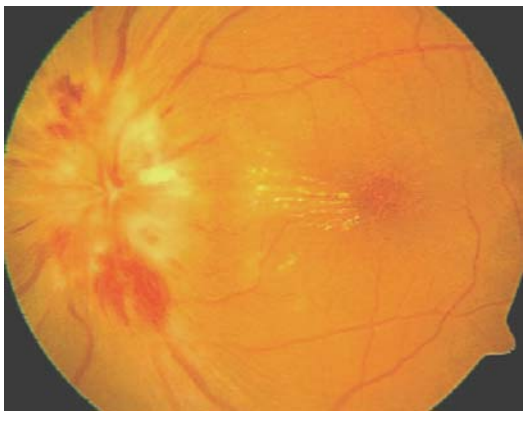


FIGURE 12.63.4: Malignant hypertension

DIABETIC RETINOPATHY

Non-proliferative Diabetic Retinopathy (NPDR)

- Microaneurysm, vascular changes, like looping **(Figs 12.64.1 to Figs 12.64.3)**, beading, etc.
- Small 'dot' and 'blot' hemorrhage **(Figs 12.64.3 and 12.64.4)**
- Intraretnal microvascular abnormalities (IRMA)
- Hard exudates **(Fig 12.64.5)**
- Further classified as mild, moderate, severe and very severe NPDR

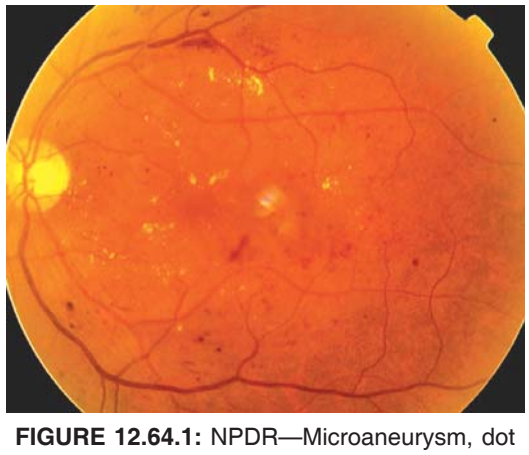


FIGURE 12.64.1: NPDR—Microaneurysm, dot and blot haemorrhage

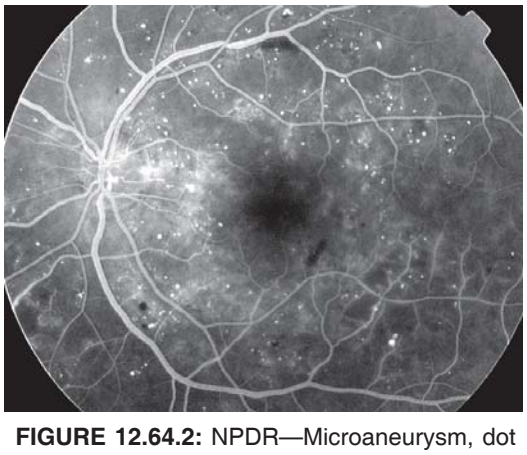


FIGURE 12.64.2: NPDR—Microaneurysm, dot and blot haemorrhage—FFA

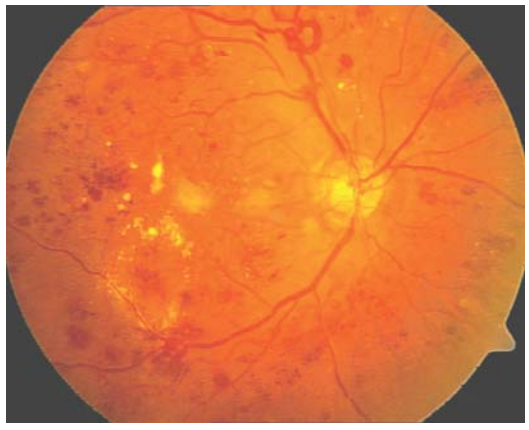


FIGURE 12.64.3: NPDR—looping, dot and blot hemorrhage

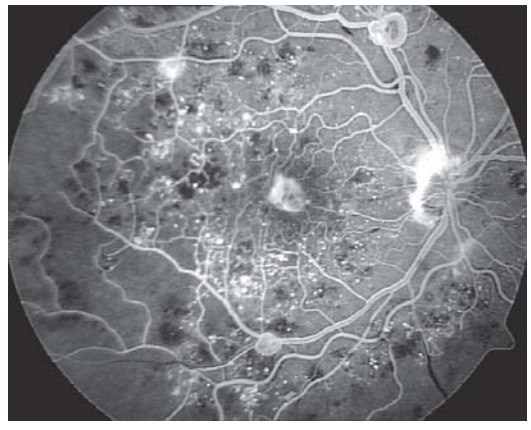


FIGURE 12.64.4: NPDR—looping, dot and blot hemorrhage

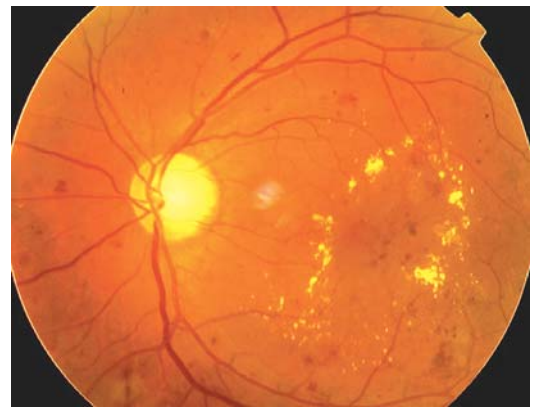


FIGURE 12.64.5: NPDR—hard exudates

Diabetic Maculopathy

- *Clinically significant macular edema (CSME) is defined as:*
- Retinal edema or thickening at or within 500 μm of the center of fovea
- Hard exudates at or within 500 μm of the center of fovea **(Figs 12.65.1 and 12.65.2)**
- Area of retinal thickening of 1 dd size (1500 μm) or at least part of which within 1 dd of the center of fovea **(Figs 12.65.3 to 12.65.5)**

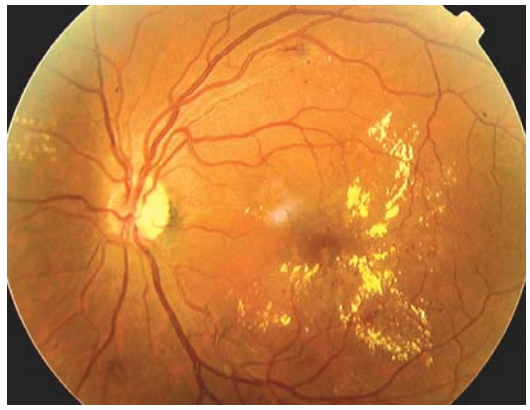


FIGURE 12.65.1: NPDR—CSME

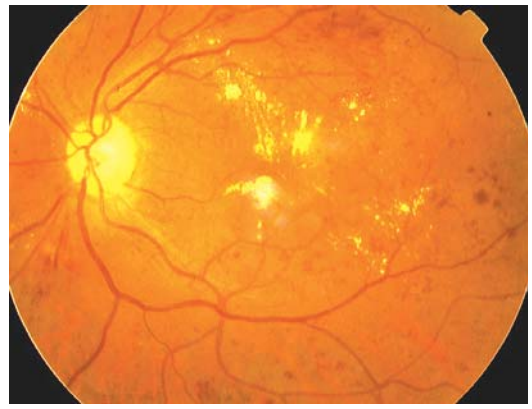


FIGURE 12.65.2: NPDR—CSME

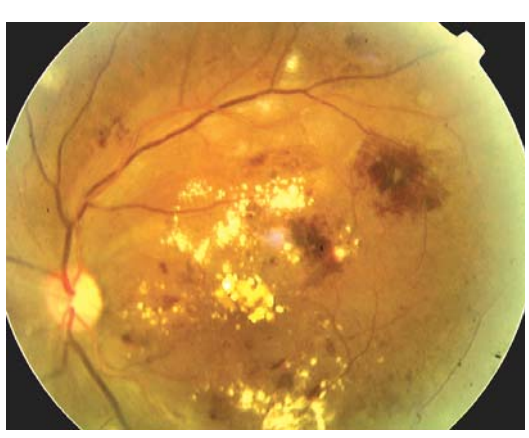


FIGURE 12.65.3: NPDR—CSME

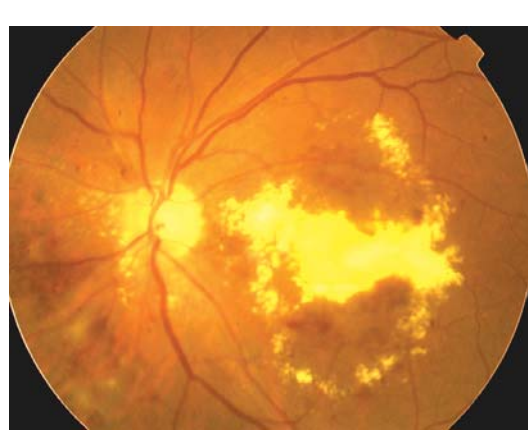


FIGURE 12.65.4: NPDR—CSME

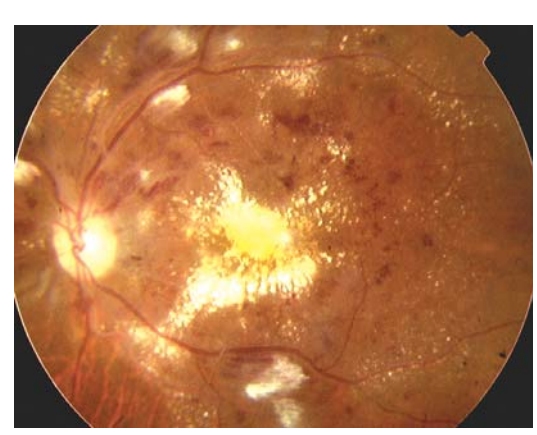


FIGURE 12.65.5: NPDR—CSME

Proliferative Diabetic Retinopathy (PDR)

- Affects 5 percent of diabetic population and more with Insulin dependent diabetes mellitus (IDDM)
- Neovascularization is the hallmark of PDR **(Figs 12.66.1 and 12.66.2)**
- New vessels at the disk (NVD), or along the course of major blood vessels, NVD-'new vessels elsewhere'

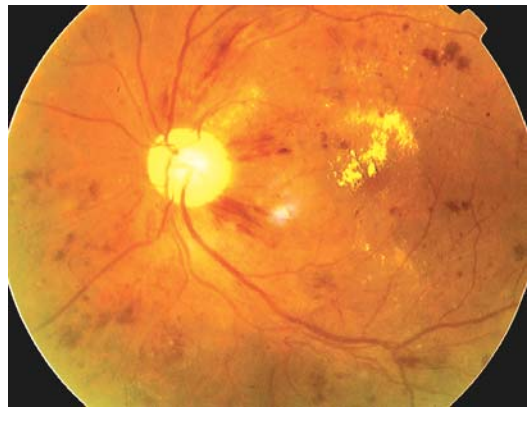


FIGURE 12.66.1: Early PDR

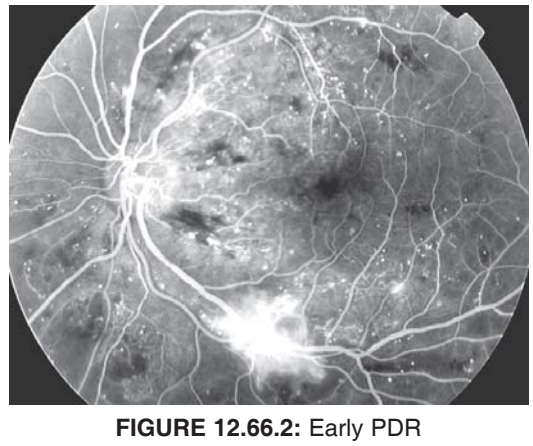


FIGURE 12.66.2: Early PDR

- Posterior vitreous detachment
- Hemorrhage may occur in the form of intraretinal, preretinal and vitreous hemorrhage **(Figs 12.66.3 and 12.66.4)**

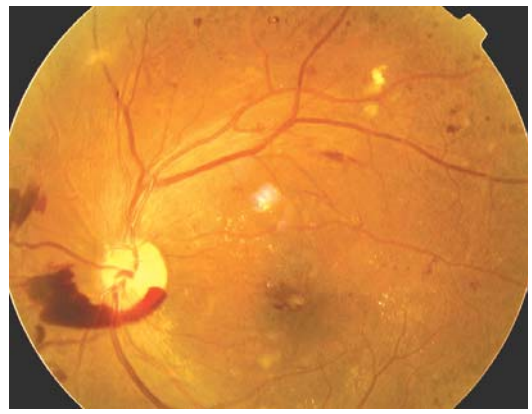


FIGURE 12.66.3: Late PDR

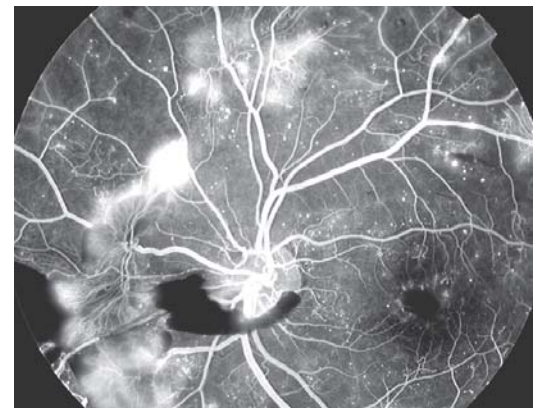


FIGURE 12.66.4: Late PDR

Advanced Proliferative Diabetic Retinopathy (Advanced PDR)

- Central or branch retinal venous occlusion **(Figs 12.67.1 and 12.67.2)**
- Recurrent vitreous hemorrhage **(Figs 12.67.3 and 12.67.4)**
- Tractional retinal detachment **(Figs 12.14.9 and 12.14.10)**
- Brunt out proliferative diabetic retinopathy or ultimately featureless retina **(Fig 12.67.5 to 12.67.8)**
- Neovascular glaucoma

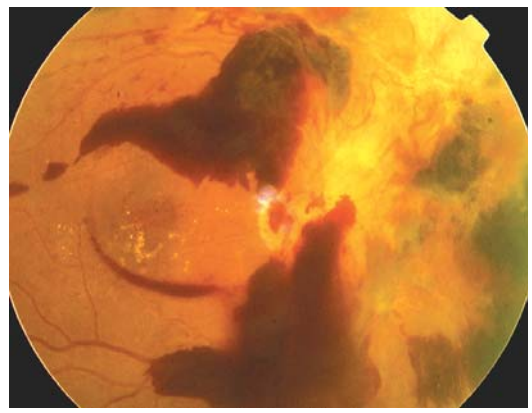


FIGURE 12.67.1: Advanced PDR—CRVO with preretinal hemorrhage

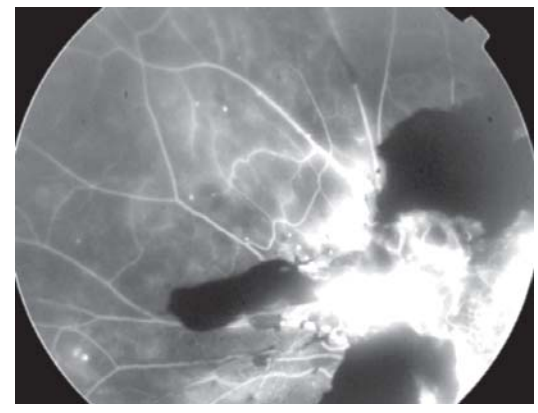


FIGURE 12.67.2: Advanced PDR—CRVO with preretinal hemorrhage

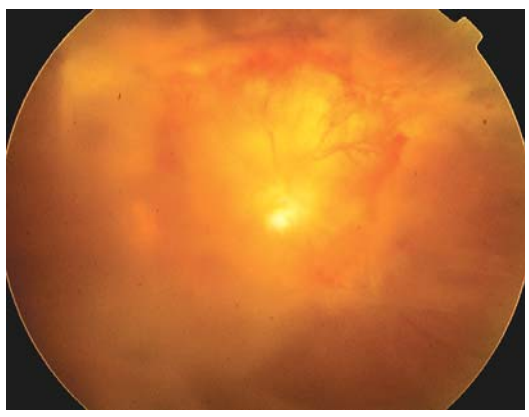


FIGURE 12.67.3: Advanced PDR—vitreous hemorrhage

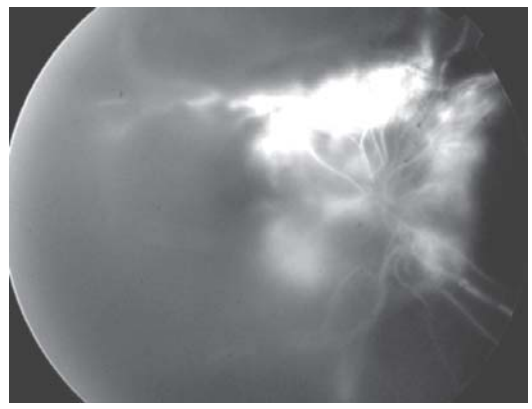


FIGURE 12.67.4: Advanced PDR—vitreous hemorrhage

FIGURE 12.67.5: Advanced PDR—featureless retina

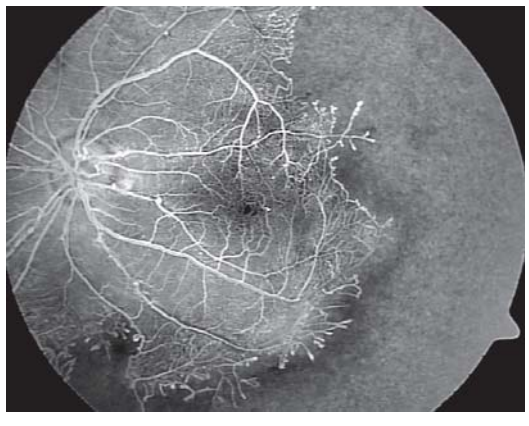


FIGURE 12.67.6: Advanced PDR—featureless retina

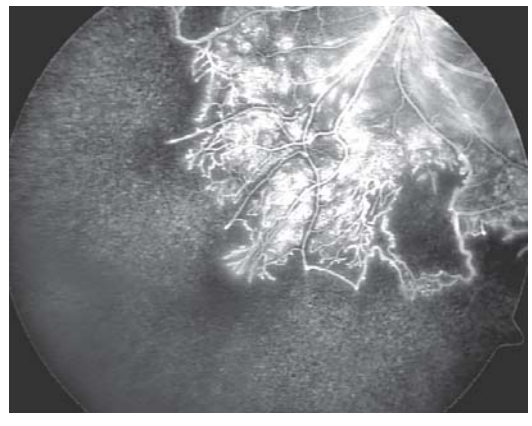


FIGURE 12.67.7: Advanced PDR—featureless retina

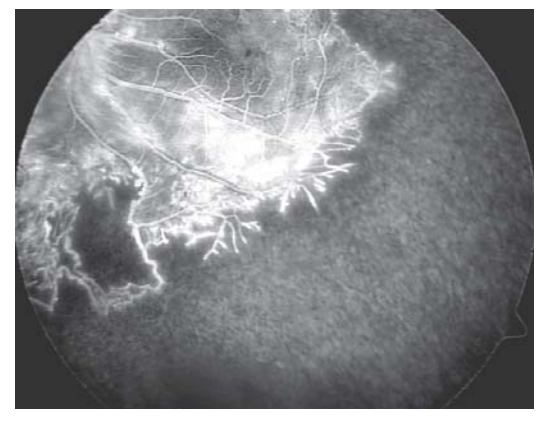


FIGURE 12.67.8: Advanced PDR—featureless retina

RETINOPATHY OF PREMATURITY (ROP)

- Occurs some weeks after birth, in premature infants (less than 1500 gm birth weight), who have been given high concentration of oxygen during the first 10 days of life
- Appearance of hazy white patches in the temporal peripheral retina **(Figs 12.68.1 and 12.68.2)**
- Milder forms result in tractional bands at upper temporal quadrant producing an ectopic macula or dragged optic disk **(Figs 12.68.3 to 12.68.5)**
- In severe cases, fibrovascular tissue proliferates to form a mass behind the lens – causing *pseudoglioma and leukocoria (See Chapter: 7)*
- Eventually tractional retinal detachment occurs and the vision is usually lost

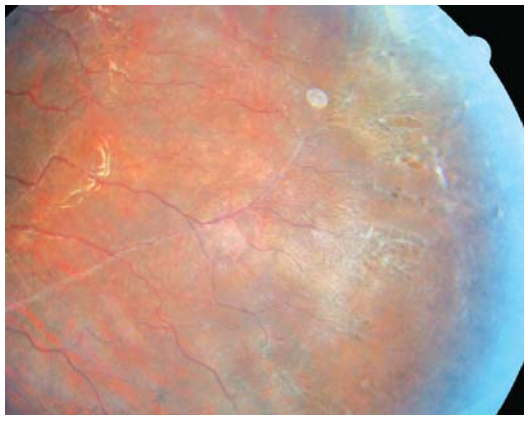


FIGURE 12.68.1: Retinopathy of prematurity

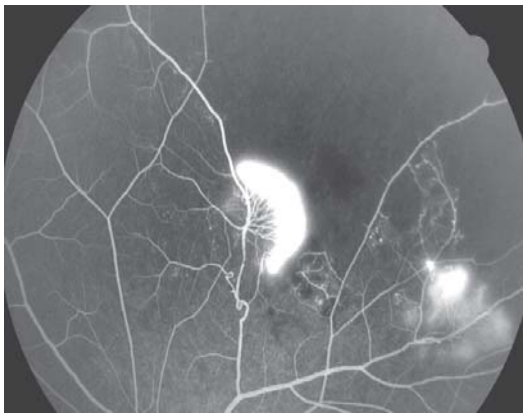


FIGURE 12.68.2: Retinopathy of prematurity

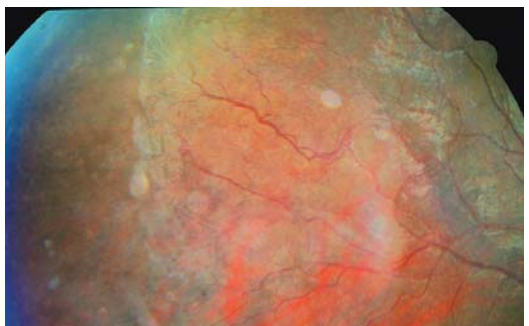


FIGURE 12.68.3: ROP—Sea fan neovascularization

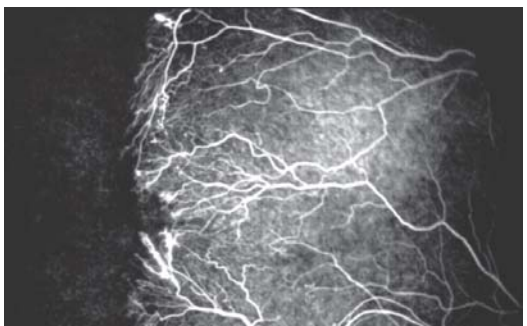


FIGURE 12.68.4: ROP—Sea fan neovascularization

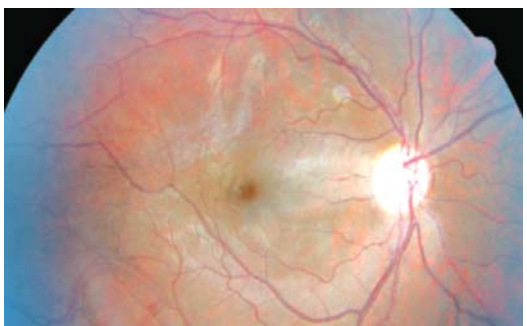


FIGURE 12.68.5: Retinopathy of prematurity—ectopic macula

RETINOBLASTOMA

- Malignant intraocular tumor originating in the outer nuclear layer
- Average age of presentation is 18 months, and 20 percent cases are bilateral
- Leukocoria or 'amaurotic cat's eye reflex' – the most common mode of presentation in 60 percent of cases **(Fig 12.69.1)**
- Squinting of the eye-the second most common mode of presentation (20%)
- Secondary glaucoma which may be associated with Buphthalmos **(Fig 12.69.2)**
- In neglected cases, there is orbital involvement, leading to proptosis **(See Figs 14.13.1 to 14.13.3)**

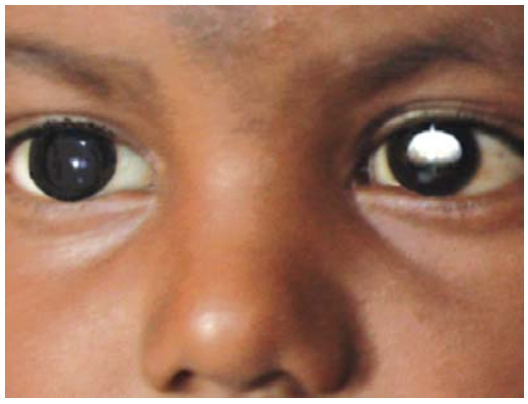


FIGURE 12.69.1: Retinoblastoma—leukocoria

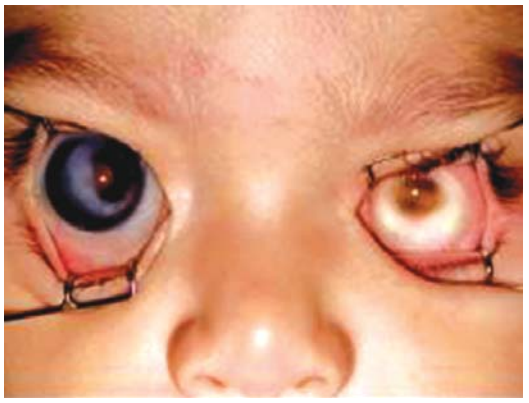


FIGURE 12.69.2: Retinoblastoma—buphthalmos and phthisis bulbi

- Endophthalmitis or anterior uveitis may also be a presenting feature which may lead to atrophy of the globe **(See Fig 5.3.7)**
- *Endophytic type* is most common
  - white or pearly-pink colored mass with sharp margin projects into the vitreous cavity **(Figs 12.69.3 to 12.69.5)**
  - presence of calcium deposits giving an appearance 'cottage cheese' **(Fig 12.69.6)**
  - multiple seedling may occour in vitreous cavity
- *Exophytic type* gives rise to exudative retinal detachment and the tumor itself is difficult to visualize
- Should be differentiated from other causes of leukocoria ***(See Chapter: 7)***
- *Treatment:* Enucleation, radiotherapy, chemotherapy, photocoagulation or cryotherapy and in extreme cases exenteration of orbit

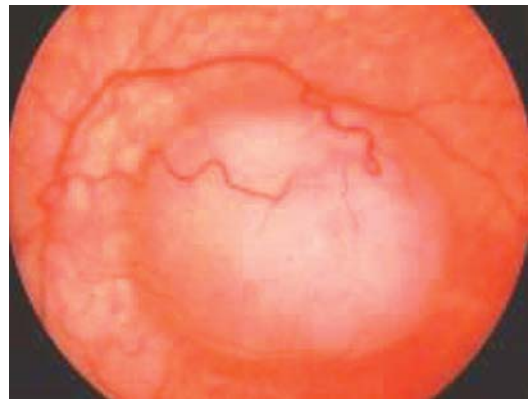


FIGURE 12.69.3: Large endophytic RB

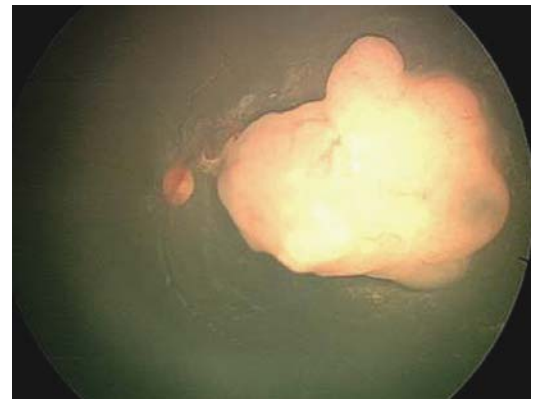


FIGURE 12.69.4: Large endophytic RB

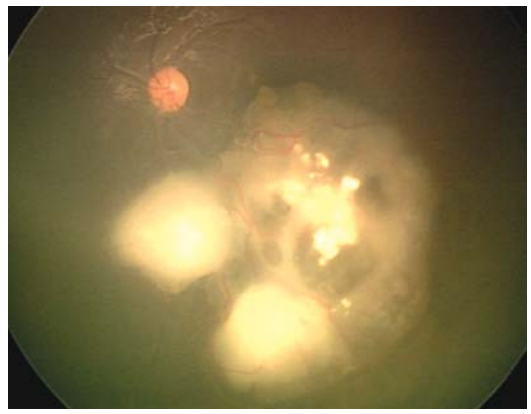


FIGURE 12.69.5: Large endophytic retinoblastoma

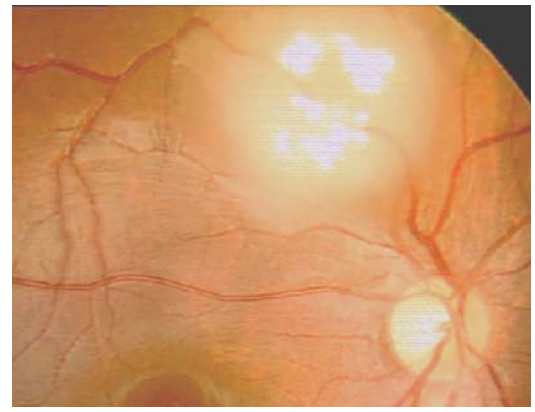


FIGURE 12.69.6: Retinoblastoma—cottage cheese appearance

MISCELLANEOUS RETINAL CONDITION

Idiopathic Polypoidal Choroidal Vasculopathy

- Rare, unilateral condition which affects the darker women
- IPCV consists of dialated innner choroidal vessels, at the macula or juxta papillary region which causes repeated episode of subretinal or sub-RPE hemorrhage
- Clinically, orange-red subretinal nodules are seen in the posterior poll which represents polyps **(Fig 12.70.1)**
- Multiple areas of hemorrhagic PED surrounded by hard exudates **(Fig 12.70.2)**
- FFA shows stippled hyperfluorescence in the region of polyp which becomes fuzzy in the late stage **(Figs 12.70.3 and 12.70.4)**
- But an ICG angiography is more diagnostic
- Prognosis is usually good

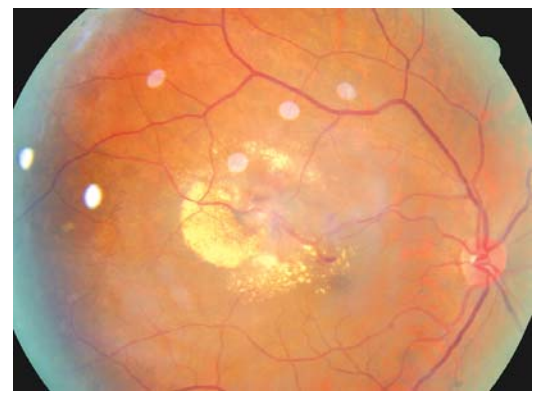


FIGURE 12.70.1: Idiopathic polypoidal choroidal vasculopathy

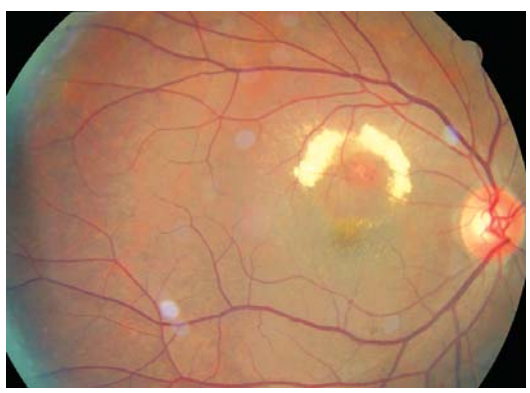


FIGURE 12.70.2: Idiopathic polypoidal choroidal vasculopathy

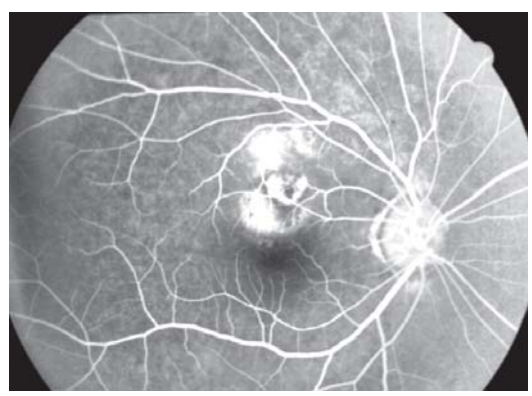


FIGURE 12.70.3: Idiopathic polypoidal choroidal vasculopathy—FFA

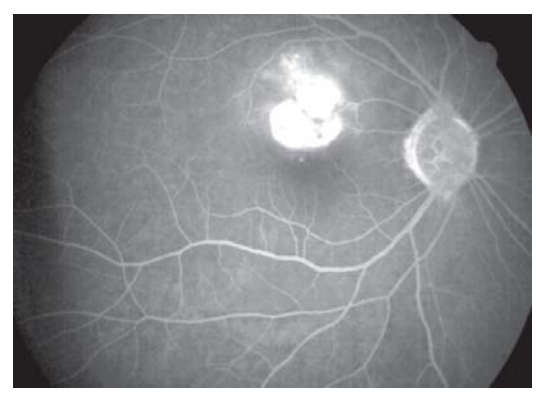


FIGURE 12.70.4: Idiopathic polypoidal choroidal vasculopathy—FFA

Familial Exudative Vitreoretinopathy

- Rare, dominant inherited condition, similar to retinopathy of prematurity
- Condition is not associated with prematurity or low birth weight babies
- Dragged retinal vessels from optic disk with an abrupt termination
- Equatorial area retina has a scalloped edge **(Figs 12.71.1 to 12.71.3)**

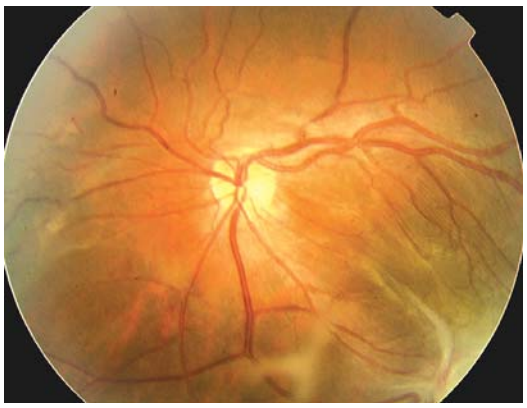


FIGURE 12.71.1: Familial exudative vitreoretinopathy —dragged retina

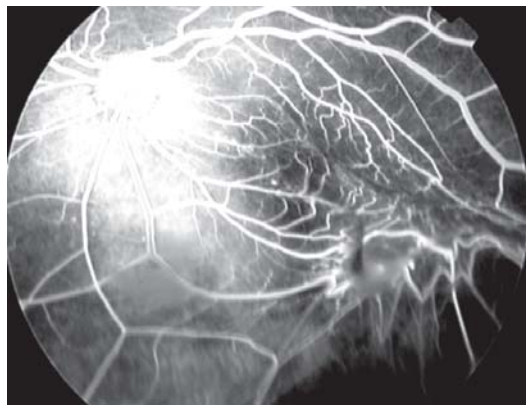


FIGURE 12.71.2: Familial exudative vitreoretinopathy—FFA

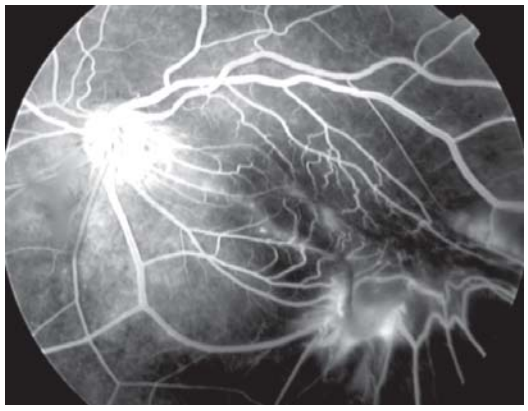


FIGURE 12.71.3: Familial exudative vitreoretinopathy—FFA

Stickler's Disease

- Autosomal dominant with marfanoid habitus
- Lattice-like radial perivascular pigmentation with overlying chorioretinal atrophy **(Figs 12.72.1 to 12.72.4)**
- Associated vitreous degeneration with liquefaction, myopia and cataract
- General orofacial and skeletal anomalies
- Increased risk of retinal detachment
- Periodic checkup is necessary to treat retinal breaks early

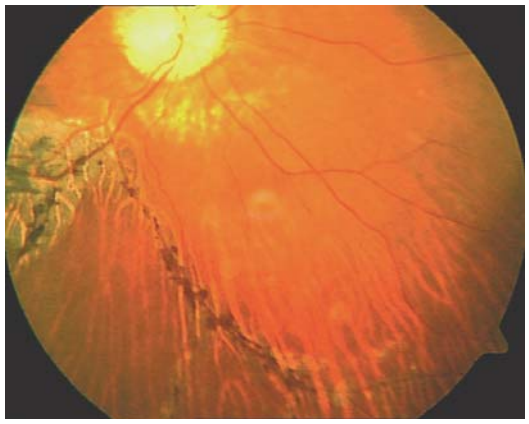


FIGURE 12.72.1: Stickler's disease

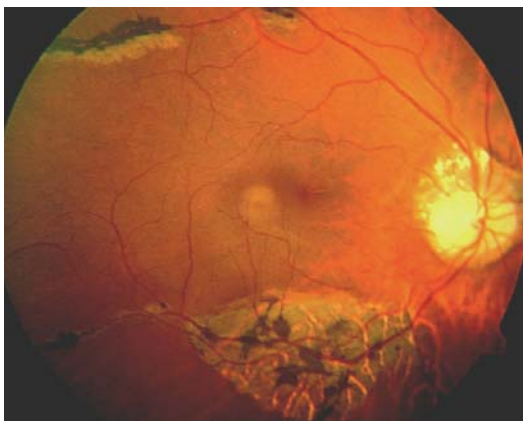


FIGURE 12.72.2: Stickler's disease

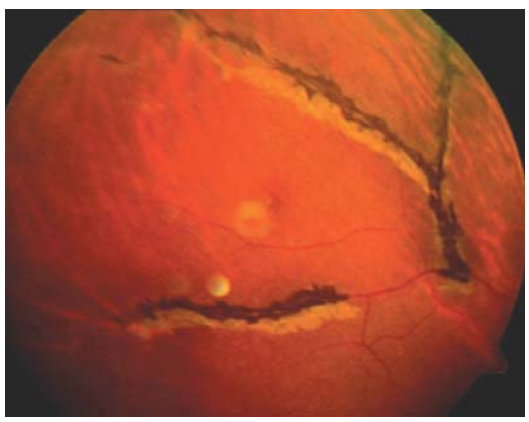


FIGURE 12.72.3: Stickler's disease

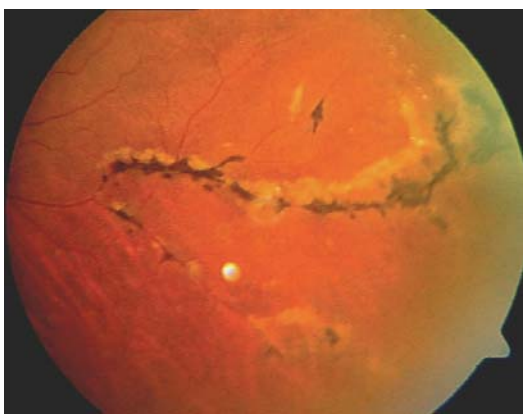


FIGURE 12.72.4: Stickler's disease

Idiopathic Parafoveal Telangiectasia

- Rare, unilateral or bilateral condition, may be congenital or acquired
- Presence of focal microaneurysm or saccular dilatation of some pattern of the parafoveal capillary network **(Figs 12.73.1 and 12.73.2)**
- May also involve the area temporal to fovea with the formation of SRNVMs
- Entire network may be involved in late stage and in some cases progressive obliteration of perifoveal capillary network
- FFA shows the pathology clearly with late staining **(Figs 12.73.3 and 12.73.4)**

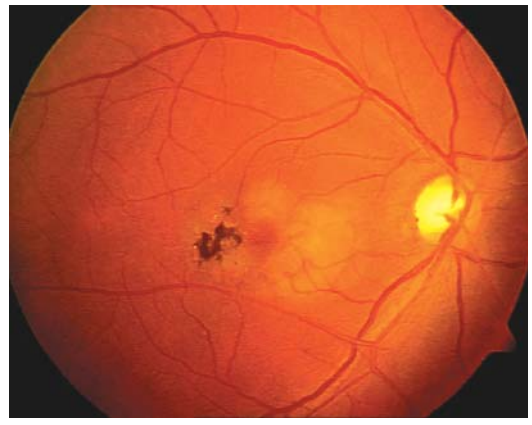


FIGURE 12.73.1: Idiopathic parafoveal telangiectasia

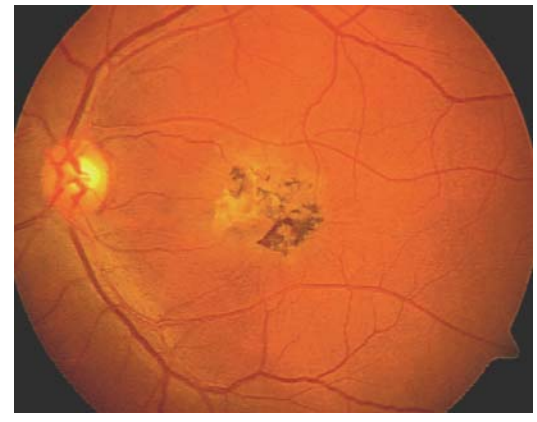


FIGURE 12.73.2: Idiopathic parafoveal telangiectasia

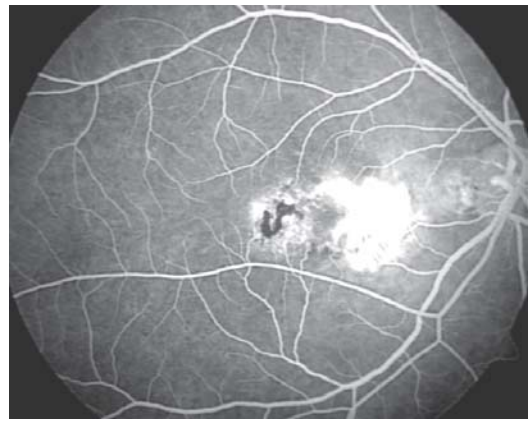


FIGURE 12.73.3: Idiopathic parafoveal telangiectasia—FFA

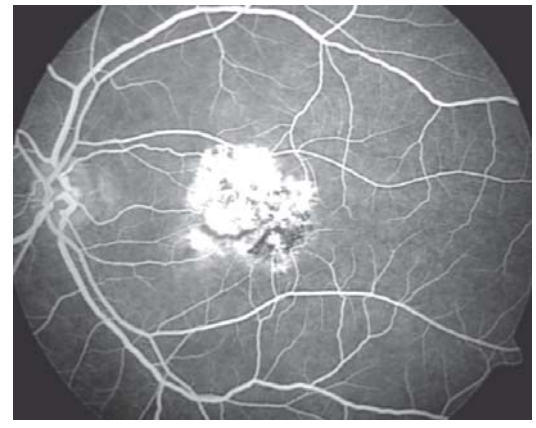


FIGURE 12.73.4: Idiopathic parafoveal telangietasia

Retinal Cyst

- May be subretinal or intraretinal cyst
- Intraretinal cysts form in case of old retinal detachment
- Subretinal cysts are caused by parasite like, subretinal *Cysticercosis* **(Figs 12.74.1 and 12.74.2)**
- May cause subretinal fibrosis, secondary pigmentary changes and tractional retinal detachment **(Fig 12.74.3)**

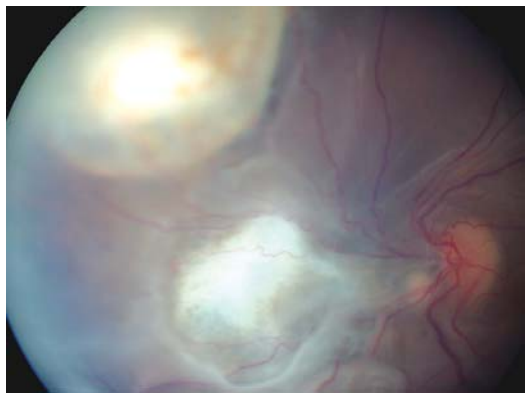


FIGURE 12.74.1: Subretinal cysticercosis

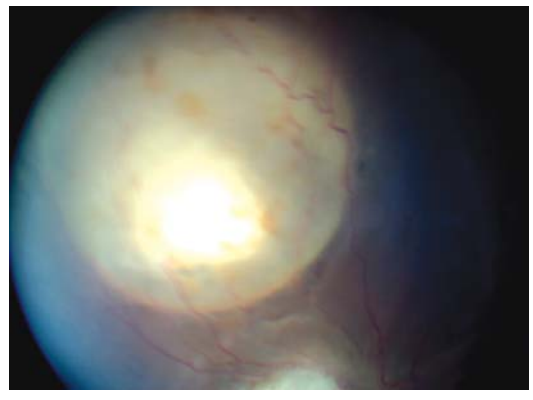


FIGURE 12.74.2: Subretinal cysticercosis

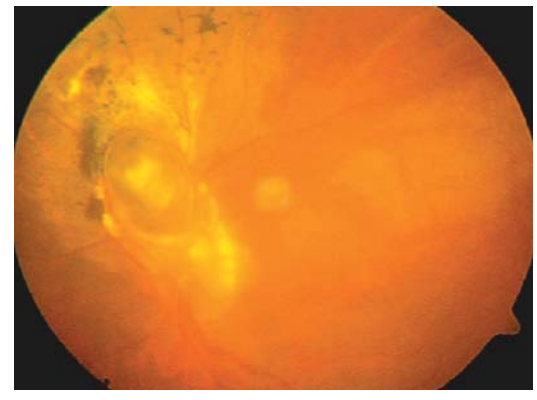


FIGURE 12.74.3: Subretinal cysticercosis—fibrosis and TRD

13 Diseases of the Lacrimal Apparatus

LACRIMAL GLAND DISEASES

- Acute dacryoadenitis
- Chronic dacryoadenitis

EPIPHORA

- Mechanical obstruction
- Lacrimal pump failure
- Chronic conaliculitis
- Occlusion of puncta or canaliculus

DACRYOCYSTITIS

- Acute (on chronic) dacryocystitis
- Chronic dacryocystitis
- Tumors of the lacrimal sac

RARE LACRIMAL CONDITIONS

- Lacrimal fistula
- Accessory lacrimal punctum
- Accessory lobe of lacrimal gland

LACRIMAL GLAND DISEASES

Acute Dacryoadenitis

- Associated with systemic diseases like mumps, influenza or glandular fevers
- Characterized by acute local pain, swelling and tenderness **(Fig 13.1.1)**
- Drooping of the outer part of upper lid with an S-shaped curve **(Fig 13.1.2)**
- *Treatment:* usually self limiting, but systemic antibiotics and local hot compress are required in some cases

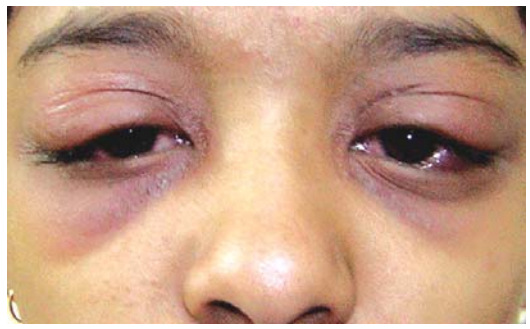


FIGURE 13.1.1: Bilateral acute dacryoadenitis

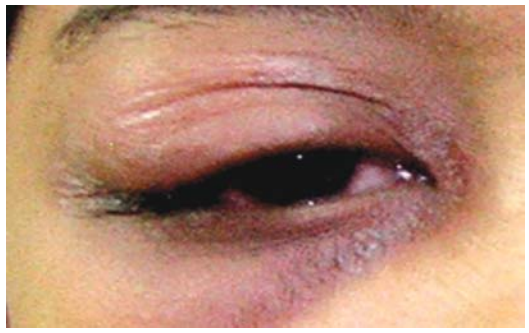


FIGURE 13.1.2: Acute dacryoadenitis—S-shaped lid

Chronic Dacryoadenitis

- *Mikulicz syndrome:* characterized by a symmetrical swelling of the lacrimal and salivary glands of both sides
  - may occur as a part of sarcoidosis, Hodgkin's disease or one of the lukemias
- *Dacryops:* cystic swelling in the upper fornix due to retention of secretion owing to blockage of one of the lacrimal ducts **(Fig 13.2.1)**
- *Lacrimal glands tumors*
  - relatively rare and show a marked resemblance to the parotid gland tumors
  - appears as hard, nodular, and slightly mobile lumps
  - proptosis is downwards and medially **(Fig 13.2.2)**
  - benign mixed tumors are the majority and have excellent prognosis
  - carcinoma has rapid onset with involvement of lymph nodes, needs radical surgery after needle biopsy or exenteration of the orbit

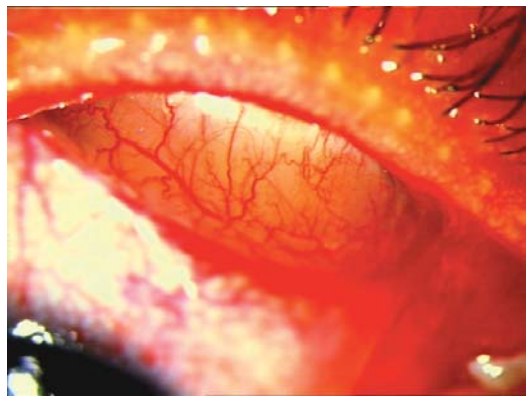


FIGURE 13.2.1: Dacryops

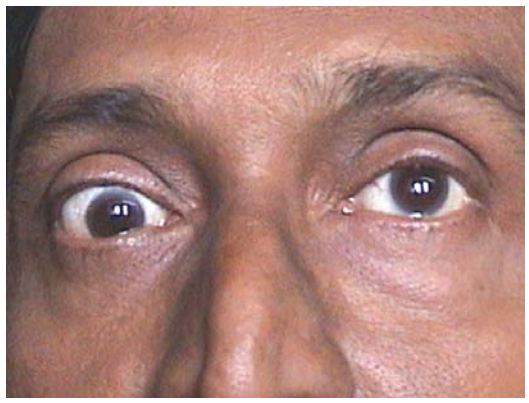


FIGURE 13.2.2: Lacrimal gland tumor

EPIPHORA

- Watering is associated with blurring of vision and continuous discomfort caused by tears running down the cheek
- In all cases it is necessary to differentiate it from *lacrimation* which is due to excessive secretion of tears
- *Epiphora* is due to defective drainage of tears

Mechanical Obstruction

- Congenital absence or occlusion of puncta
- Punctal atresia or stenosis **(Fig 13.3.1)**
- Foreign body in the puncta **(Fig 13.3.2)**
- Malposition of puncta **(Figs 13.3.3 and 13.3.4)**
- Congenital dacryocystitis
- Chronic dacryocystitis
- Neoplasm of lacrimal sac
- Nasal pathology—like, polyps, tumors of inferior meatus of nose

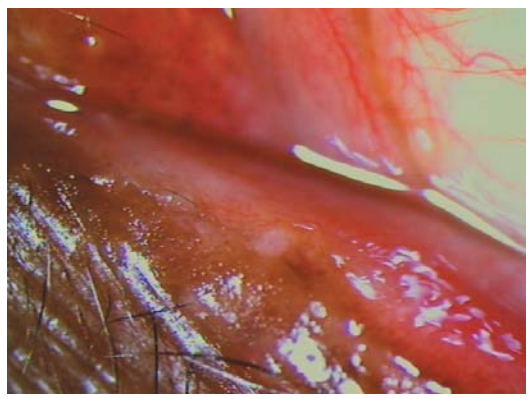


FIGURE 13.3.1: Punctal stenosis

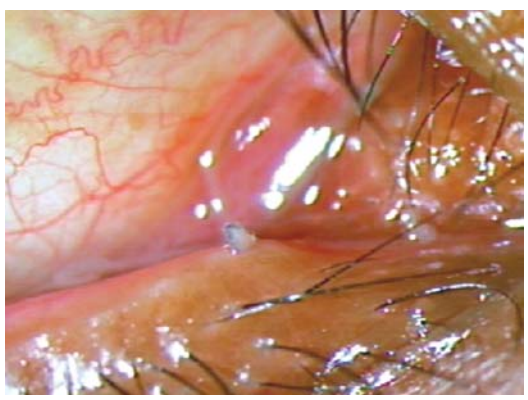


FIGURE 13.3.2: Eyelash in lower punctum

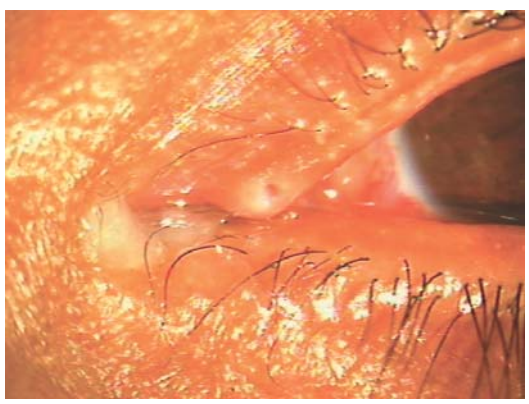


FIGURE 13.3.3: Lacrimal punctal malposition

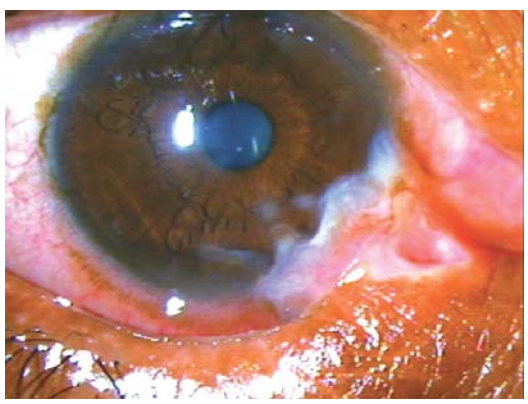


FIGURE 13.3.4: Lacrimal punctal malposition

Lacrimal Pump Failure

- Lower lid laxity
- Weakness of orbicularis, as in Bell's palsy **(Fig 13.4.1)**

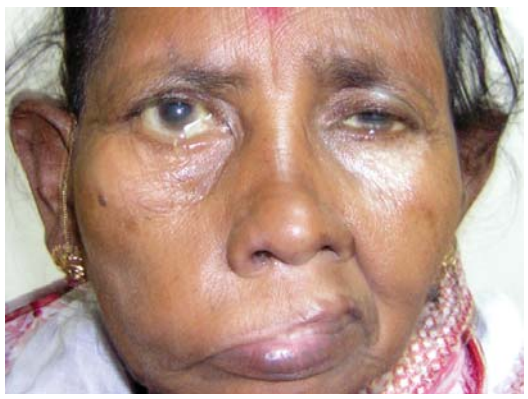


FIGURE 13.4.1: Lacrimal pump failure—Bell's palsy

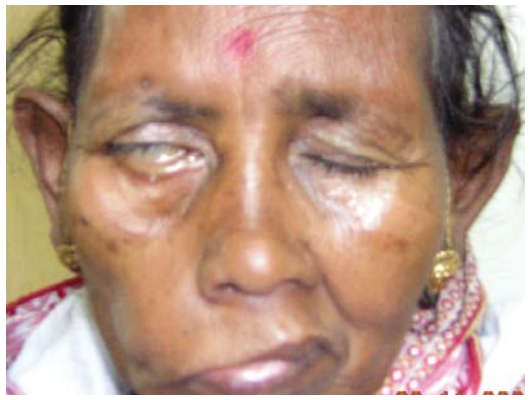


FIGURE 13.4.2: Lacrimal pump failure—Bell's palsy

Chronic Canaliculitis

- Commonly caused by *Actinomyces israelii* (streptothrix)
- Chronic infection causes discharge, a pouting punctum and concretions within the canaliculi **(Fig 13.5.1)**
- Sometimes with stone formation, called *dacryolith*
- *Treatment:* linear incision to remove the dacryolith

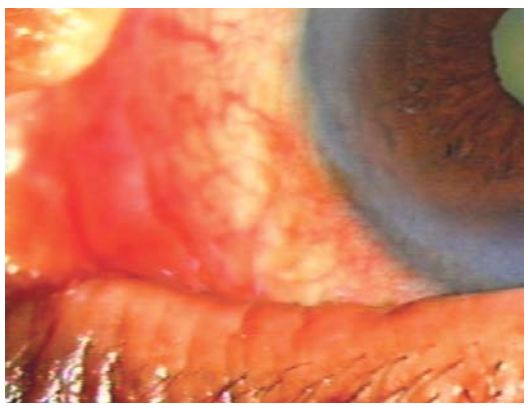


FIGURE 13.5.1: Chronic canaliculitis

Occlusion of Puncta or Canaliculus

- Punctal stenosis **(Fig 13.3.1)**
- Foreign body (eyelash—commonest) **(Fig 13.6.1)** or concretion
- Scarring of lower punctum **(Fig 13.6.2)**
- *Drug induced:* like prolonged use of IDU eye drops **(Fig 13.6.3)**
- *Iatrogenic:* punctal plug for treating dry eye conditions **(Fig 13.6.4)**

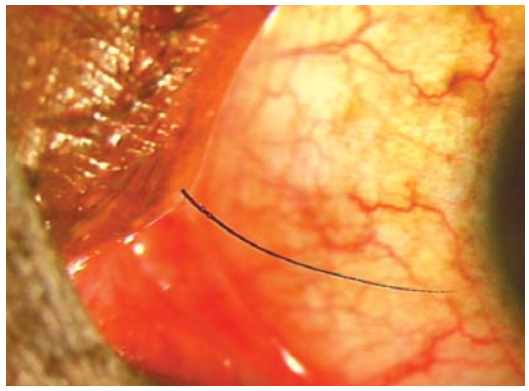


FIGURE 13.6.1: Eyelash in lower punctum

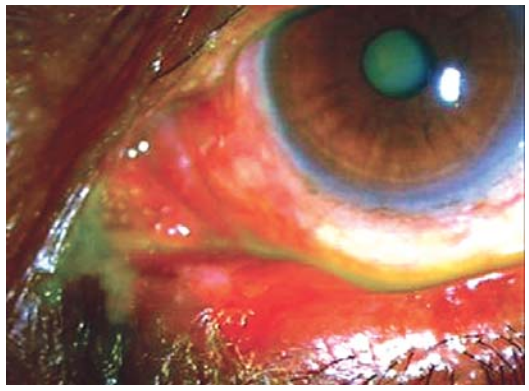


FIGURE 13.6.2: Scarring of lower punctum

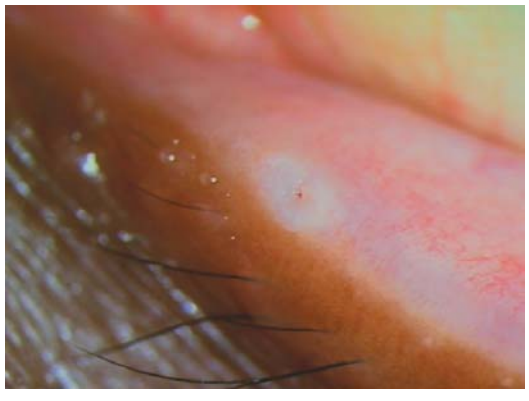


FIGURE 13.6.3: Punctal stenosis—IDU toxicity

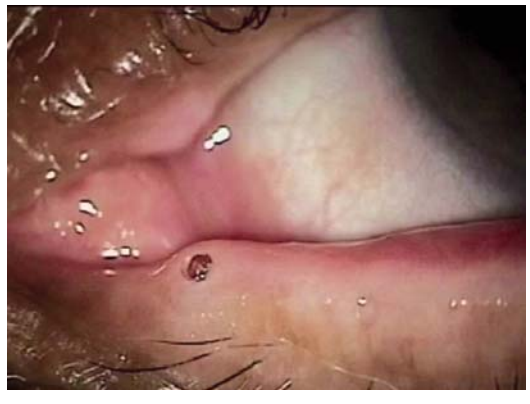


FIGURE 13.6.4: Punctal plug

DACRYOCYSTITIS

Acute (on chronic) Dacryocystitis

- Suppurative inflammation of the lacrimal sac, with an associated cellulitis of the overlying tissues **(Figs 13.7.1 and 13.7.2)**
- *Lacrimal abscess* **(Fig 13.7.3)** with scar formation **(Fig 13.7.4)**
- May often with perforation of the skin just below palpebral ligament, leading to formation of *lacrimal fistula* **(Fig 13.7.5)**
- *Treatment:* hot compress, systemic antibiotics, drainage of abscess if necessary, and the acute phase subsides, dacryocystorhinostomy (DCR) or dacryocystectomy (DCT) operation

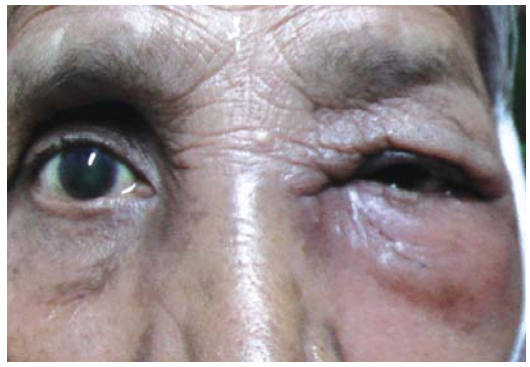


FIGURE 13.7.1: Acute dacryocystitis

FIGURE 13.7.2: Acute dacryocystitis

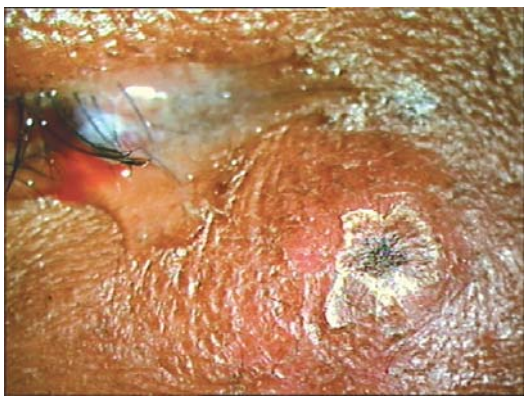


FIGURE 13.7.3: Lacrimal abscess

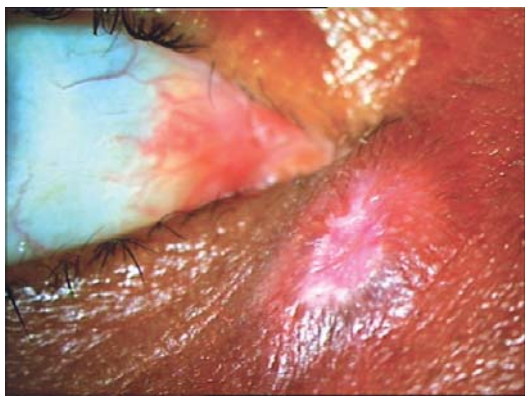


FIGURE 13.7.4: Lacrimal abscess-scar formation

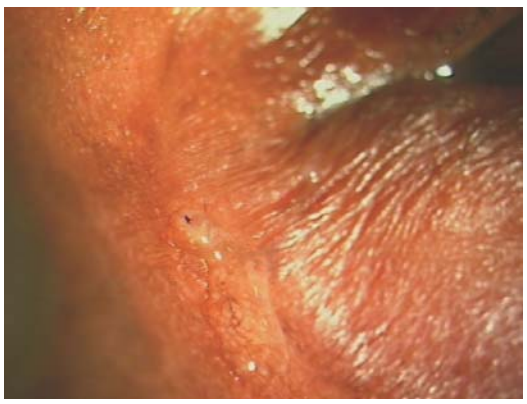


FIGURE 13.7.5: Lacrimal fistula

Chronic Dacryocystitis

- Most frequently in new-born infants and middle-aged women
- Chronic muco-purulent discharge at inner angle of the eye **(Fig 13.8.1)**
- Regurgitation of the pus or muco-pus through the puncta, on pressure over the sac region
- In long-standing cases, there is an extraordinary dilatation and thinning of the lacrimal sac, called *mucocele* **(Fig 13.8.2)**, or *pyocele* **(Fig 13.8.3)** of the lacrimal sac
- Hypopyon corneal ulcer: mostly due to *pneumococci* **(Fig 13.8.4)**
- *Treatment:*

  Congenital cases:
  - hydrostatic sac massage and antibiotic eye drop
  - probing and syringing usually at 10-12 months
  - if fails, DCR operation

  Adult cases:
  - dacryocystorhinostomy (DCR) or dacryocystectomy (DCT) operation

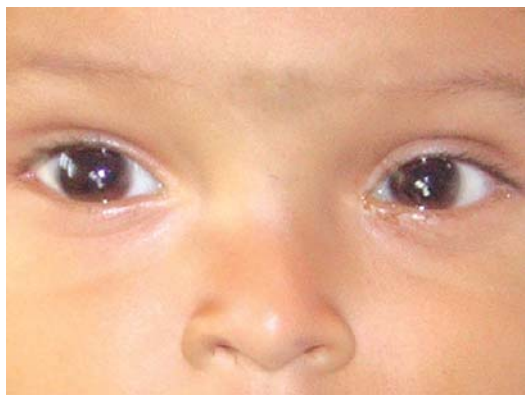


FIGURE 13.8.1: Congenital dacryocystitis LE

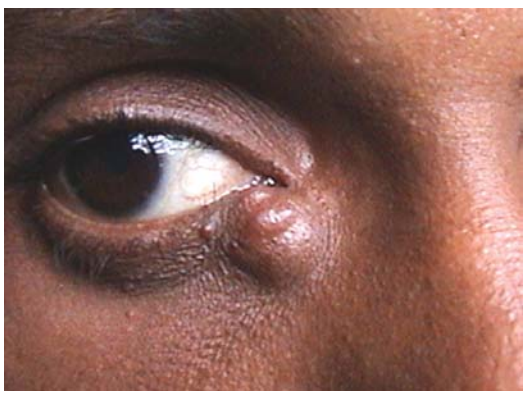


FIGURE 13.8.2: Mucocele of the lacrimal sac

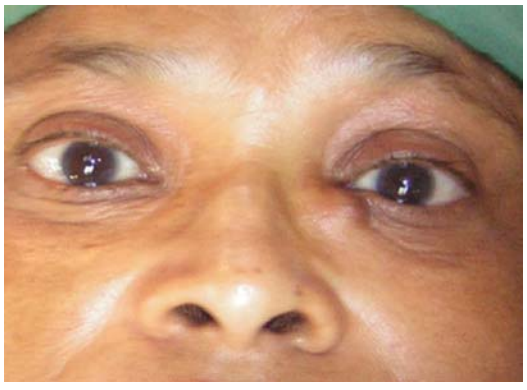


FIGURE 13.8.3: Pyocele of the lacrimal sac

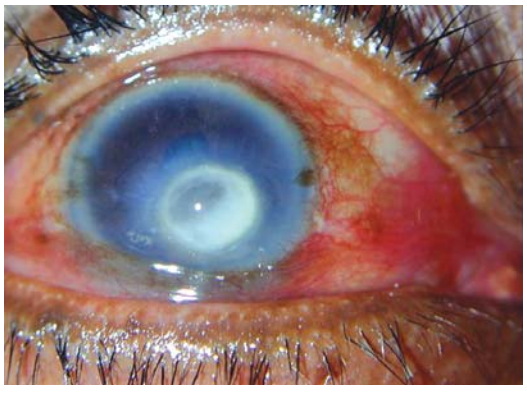


FIGURE 13.8.4: Hypopyon corneal ulcer

Tumors of the Lacrimal Sac

- Tumors of the lacrimal sac are extremely rare and represent a potentially life-threatening condition
- *Triad of malignancy* are:
  - mass below the medial palpebral ligament
  - chronic dacryocystitis that irrigates freely, and
  - regurgitation of bloody mucopus
- *Treatment:* dacryocystectomy followed by radiation

RARE LACRIMAL CONDITIONS

Lacrimal Fistula

- Rare, unilateral or bilateral condition
- May occur in congenital **(Figs 13.9.1 and 13.9.2)** or acquired **(Fig 13.9.3)** form
- Causing watering from a separate opening on the skin just below the medial canthal ligament
- Acquired from had a history of acute dacryocystitis in the past
- *In congenital form:* usually no treatment is required
- *In adult form:* fistulectomy along with DCR or DCT

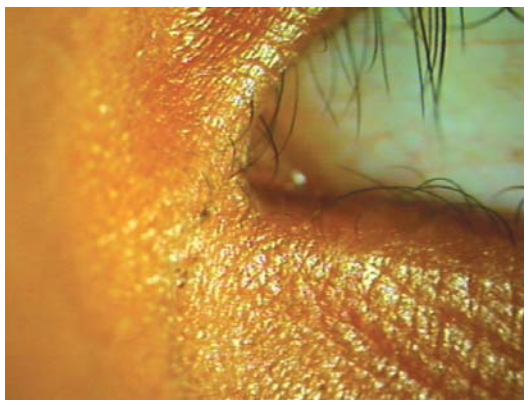


FIGURE 13.9.1: Congenital lacrimal fistula

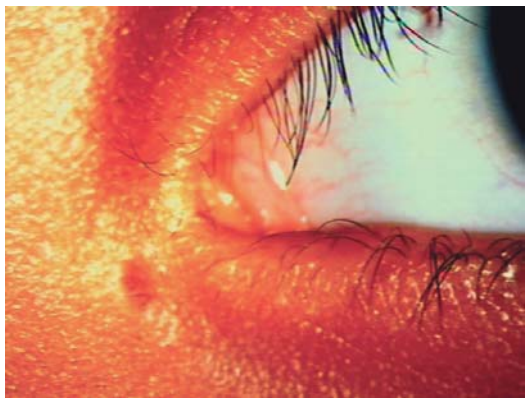


FIGURE 13.9.2: Congenital lacrimal fistula

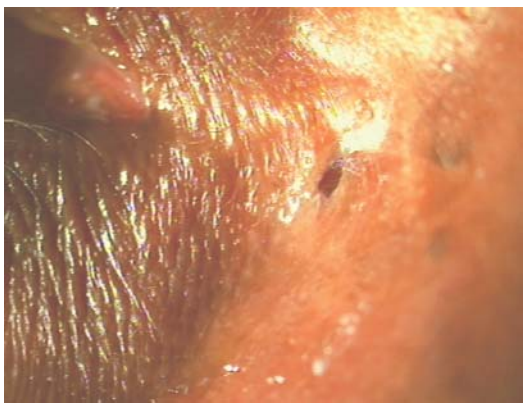


FIGURE 13.9.3: Acquired lacrimal fistula

Accessory Lacrimal Punctum

- Rare, congenital **(Fig 13.10.1)** or acquired **(Fig 13.10.2)** condition
- Seen medial to the original punctum of lower lid
- In acquired cases it may be due to trauma
- No treatment is required

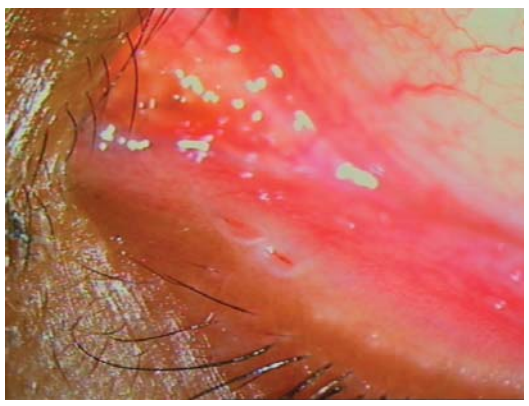


FIGURE 13.10.1: Accessory lacrimal punctum-congenital

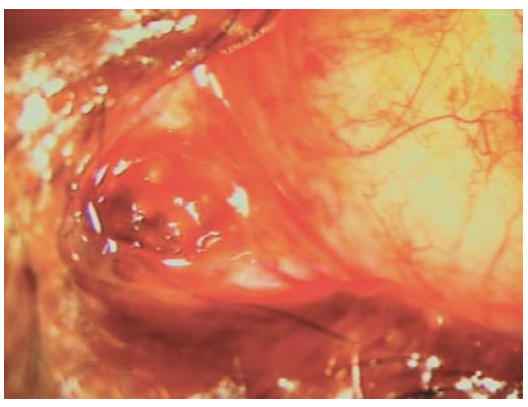


FIGURE 13.10.2: Accessory lacrimal punctum-acquired

Accessory Lobe of Lacrimal Gland

- Visible sometimes at the superolateral fornix **(Fig 13.11.1)**
- To be differentiated from dermolipoma **(Fig 13.11.2)** and orbital fat prolapse **(Figs 14.25.1 and 14.25.2)**
- Does not require any treatment

FIGURE 13.11.1: Accessory lacrimal gland lobe

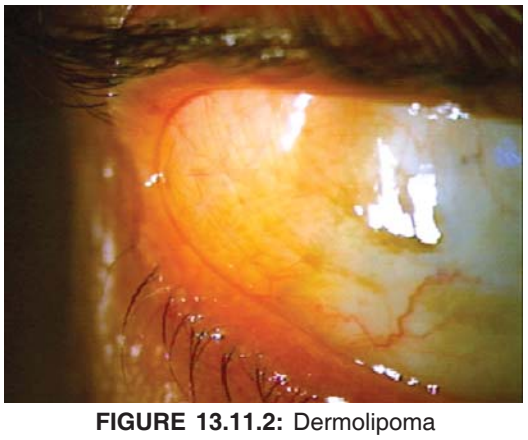


FIGURE 13.11.2: Dermolipoma

14 Diseases of the Orbit

CONGENITAL/DEVELOPMENTAL BONY ANOMALIES

- Craniosynostosis
- Orbital dysplasia
- Anophthalmos or extreme microphthalmos
- Cryptophthalmos
- Orbitopalpebral (colobomatous) cyst

PROPTOSIS

- Pseudoproptosis
- Enophthalmos

PROPTOSIS IN CHILDREN

- Craniosynostosis (shallow orbit)
- Orbitopalpebral (colobomatous) cyst
- Capillary hemangioma
- Orbital cellulitis
- Lymphangioma
- Glioma of the optic nerve
- Rhabdomyosarcoma
- Orbital extension of retinoblastoma
- Acute leukemia

OTHER RARE ORBITAL TUMORS IN CHILDREN

- Juvenile xanthogranuloma

PROPTOSIS IN ADULTS

- Inflammatory orbital diseases (orbital pseudotumor)
- Orbital cellulitis in adults
- Thyroid associated ophthalmopathy
- Cavernous hemangioma
- Orbital varix
- Meningioma of optic nerve
- Carotidcavernous fistula
- Cavernous sinus thrombosis

OTHER RARE CAUSES OF PROPTOSIS IN ADULTS

MISCELLANEOUS ORBITAL LESIONS

- Orbital rim lesions
- Orbital fat prolapse
- Luxatio bulbi
- Contracted socket

CONGENITAL/DEVELOPMENTAL BONY ANOMALIES

Craniosynostosis

- Follows premature closure of one or more cranial sutures
- Thereby, a complete arrest of bone growth perpendicular to the closed suture
- Compensatory growth of the cranium in other diameters which causes the typical shape of the skull
- *Common features are*:
  - bilateral proptosis due to shallow orbit
  - esotropia or exotropia
  - chemosis of conjunctiva, corneal exposure
  - papilledema due to increased intracranial tension
  - optic atrophy
- *Treatment:* by craniotomy or orbital compression to reduce CSF pressure and papilledema
- ***Craniofacial dysostosis (Crouzon)***
  Brachycephaly: (clover leaf skull) premature closure of all sutures
  - combined with hypoplasia of the maxilla **(Fig 14.1.1)**
  - often hereditary in nature **(Fig 14.1.2)**

  Ophthalmic features:
  - widely separated eyeballs (hypertelorism)
  - shallow orbits with proptosis **(Fig 14.1.3)**
  - conjunctival chemosis **(Fig 14.1.4)**
  - corneal problems due to exposure
  - divergent squint
  - optic atrophy
- ***Mandibulofacial dysostosis (Treacher-Collins)***
  - hypoplasia of the zygoma and mandible.
  - indistinct inferior orbital margin
  - coloboma (notching) of the lower lid
  - anti-mongoloid slanting
- ***Median facial cleft syndrome***
  - hypertelorism with telecanthus
  - divergent squint
  - cleft nose, lip and palate **(Figs 14.1.5 and 14.1.6)**
  - V-shaped frontal hair line (Widow's peak)
- ***Oxycephaly syndactyle (Apert)***
  - tower skull with flat occiput
  - hypertelorism, shallow orbits and proptosis
  - syndactyle
  - mental retardation
- ***Hypertelorism***
  - increased separation of the eyes
  - separated orbits, and broad nasal bridge **(Fig 14.1.7)**
  - divergent squint, telecanthus and anti-mongoloid slanting
  - should be differentiated from *true telecanthus* **(Fig 14.1.8)**

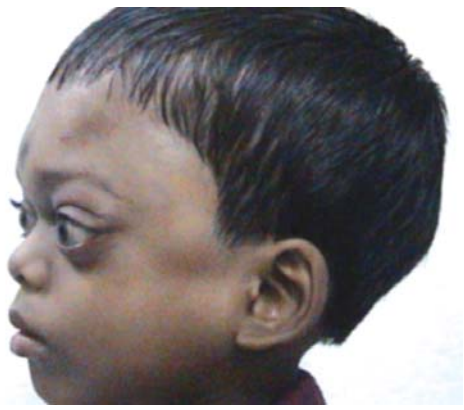


FIGURE 14.1.1: Cruzon's syndrome

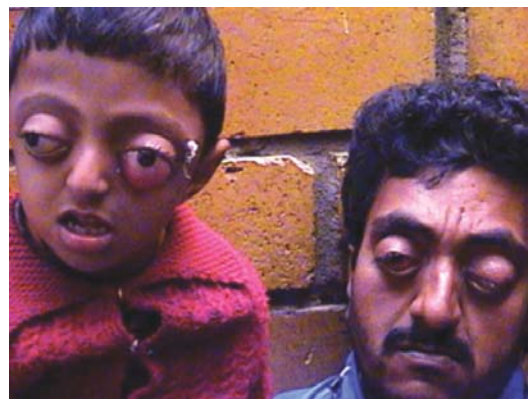


FIGURE 14.1.2: Cruzon's syndrome

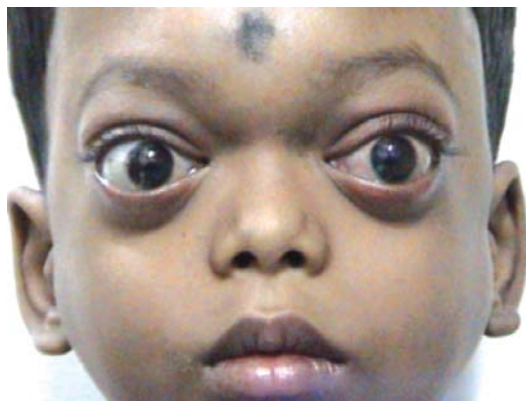


FIGURE 14.1.3: Cruzon's syndrome

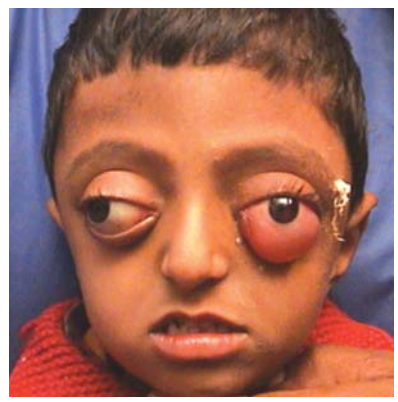


FIGURE 14.1.4: Cruzon's syndrome

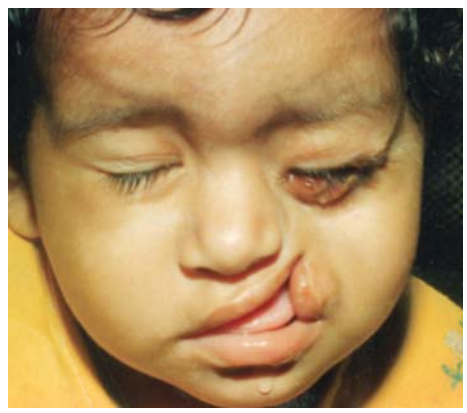


FIGURE 14.1.5: Median facial cleft syndrome

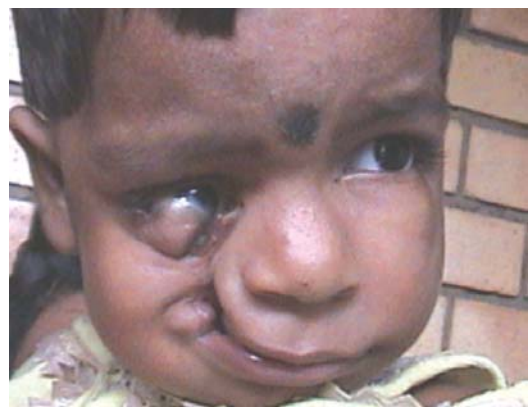


FIGURE 14.1.6: Median facial cleft syndrome

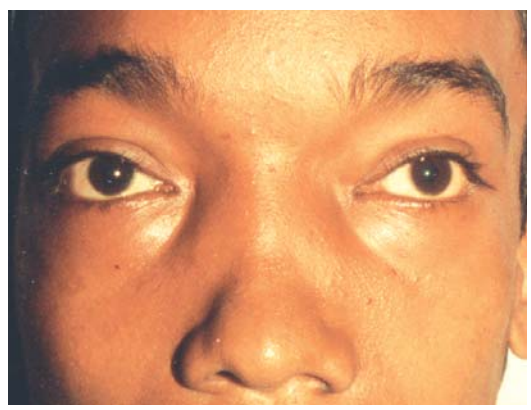


FIGURE 14.1.7: Hypertelorism

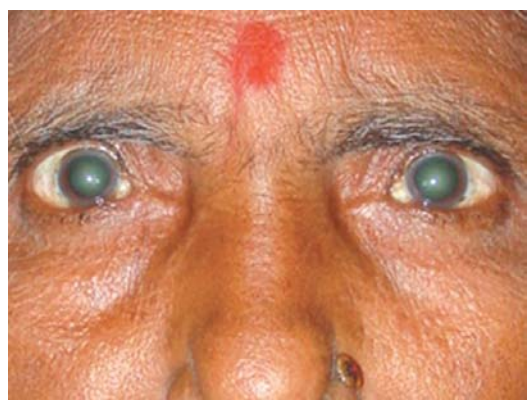


FIGURE 14.1.8: Telecanthus

Orbital Dysplasia

- Asymmetry of levels of orbital margins, especially the inferior **(Figs 14.2.1 and 14.2.2)**
- May be associated with hypertelorism, hemifacial microsomia, Goldenhar's syndrome, etc.

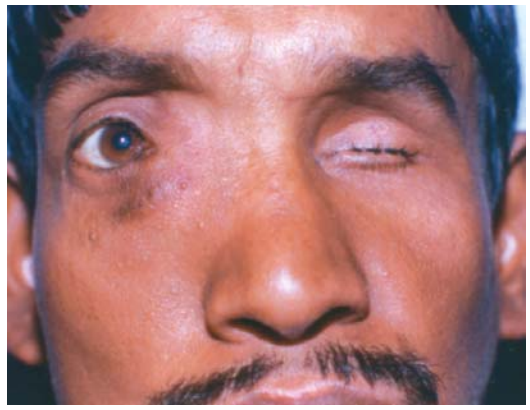


FIGURE 14.2.1: Orbital dysplasia

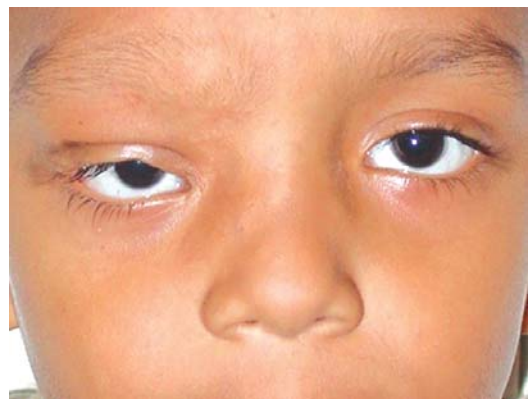


FIGURE 14.2.2: Orbital dysplasia

Anophthalmos or Extreme Microphthalmos

- Under development of bony orbit due to extreme microphthalmos or anophthalmos **(Fig 14.3.1)**
- Facial asymmetry is common
- Features of anophthalmic socket **(Fig 14.3.2)**
- *Oculodigital sign* (a blind child with eye-poking sign) may be present **(Fig 14.3.3)**
- To be differentiated from acquired anophthalmos

Cryptophthalmos

- Sometimes, associated with an extremely rare condition—*cryptophthalmos* **(Fig 14.3.4)**
  - hidden eyeball behind the non-formed eyelids
  - no conjunctival sac
  - malformed globe and orbit
  - facial asymmetry

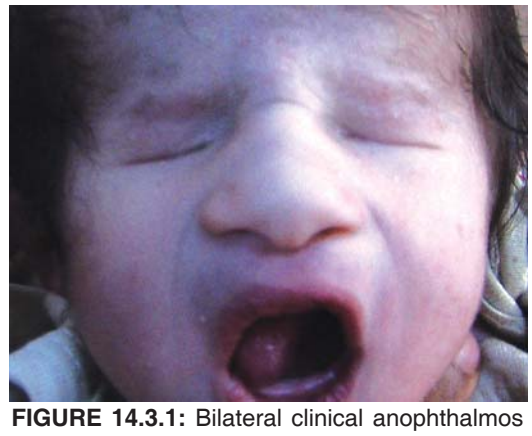


FIGURE 14.3.1: Bilateral clinical anophthalmos

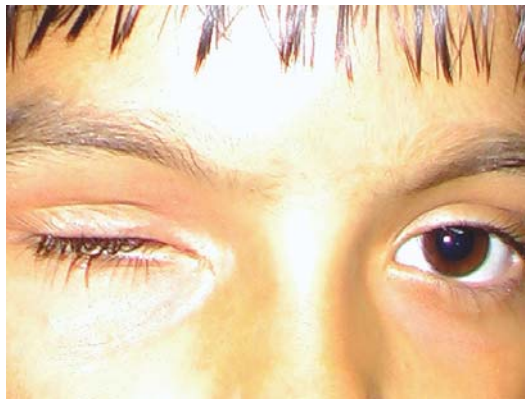


FIGURE 14.3.2: Anophthalmic socket

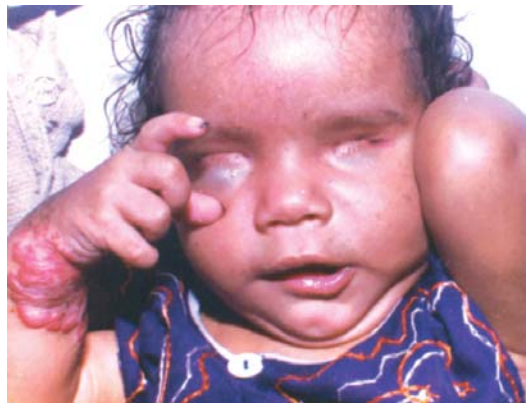


FIGURE 14.3.3: Anophthalmic socket—oculodigital sign

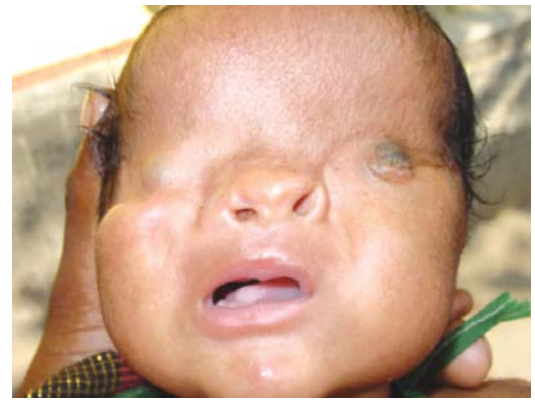


FIGURE 14.3.4: Cryptophthalmos

Orbitopalpebral (colobomatous) Cyst

- Usually associated with microphthalmos with coloboma **(Figs 14.4.1 and 14.4.2)**
- May be with normal but deformed globe **(Fig 14.4.3)**
- Classical site of the cyst is inferonasal, which corresponds to fetal fissure
- Bluish in color with upward displacement of eyeball **(Fig 14.4.4)**
- *Congenital cystic eyeball* is another variation of congenital developmental defect of the whole eyeball **(Fig 14.4.5)**

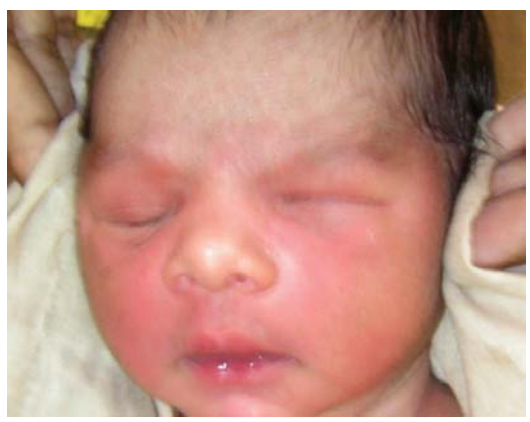


FIGURE 14.4.1: Orbitopalpebral cyst in LE

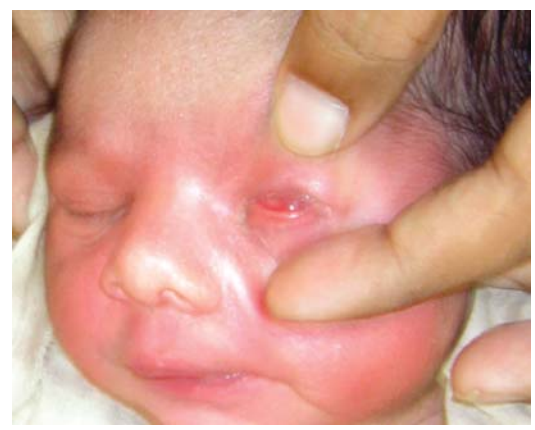


FIGURE 14.4.2: Orbitopalpebral cyst

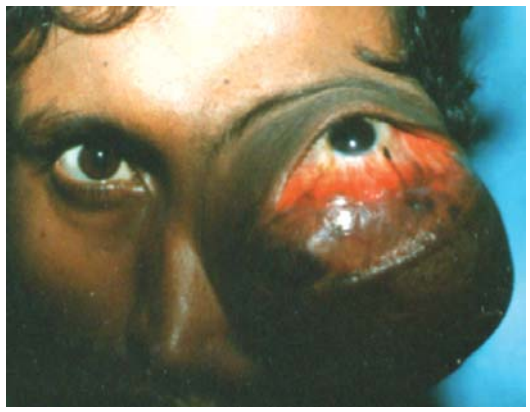


FIGURE 14.4.3: Orbitopalpebral cyst—large

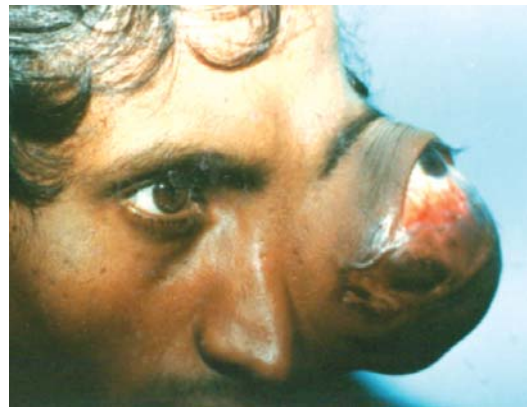


FIGURE 14.4.4: Orbitopalpebral cyst—large

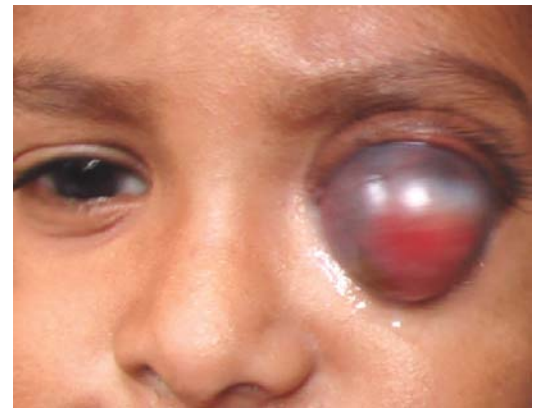


FIGURE 14.4.5: Congenital cystic eyeball

PROPTOSIS

- Forward protrusion of the eyeball much beyond the orbital margin **(Fig 14.5.1)**
- It may be dynamic, called *exophthalmos* (as in thyroid ophthalmopathy) or due to passive pushing, by a retro-orbital mass, called *proptosis*
- Most readily assessed clinically by standing behind and viewing the eyes from above **(Fig 14.5.2)**
- Usually measured by Hertel's exophthalmometer or by a plastic ruler
  - *normal value:* less than 20 mm (average = 16 mm)
  - *in proptosis:* 21 mm or more or a difference >2mm between two eyes
- Rule out the possibilities of *pseudoproptosis* and *enophthalmos*

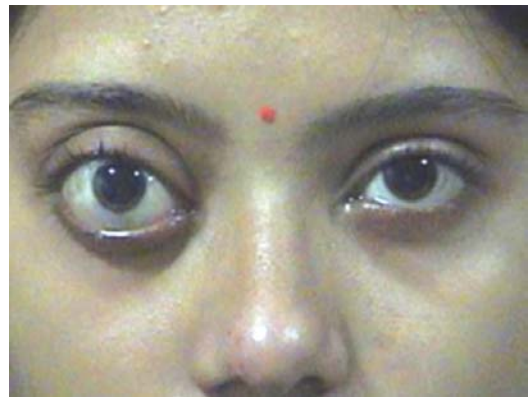


FIGURE 14.5.1: Proptosis

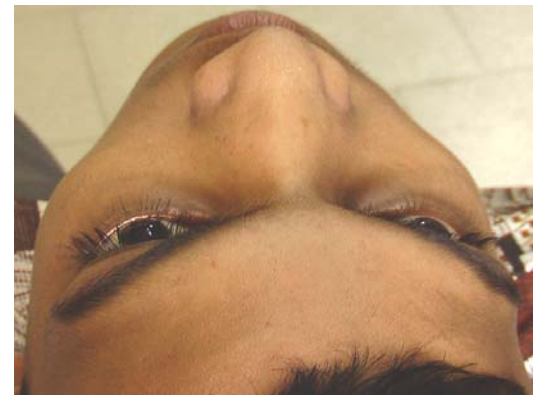


FIGURE 14.5.2: Proptosis—viewing from above

Pseudoproptosis

- One eye may apparently look to be larger than the fellow eye
- *Causes:*
  - unilateral high axial myopia **(Fig 14.6.1)**
  - unilateral buphthalmos **(Fig 14.6.2)**
  - pseudocornea or anterior staphyloma **(Figs 14.6.3 and 14.6.4)**
  - retraction of eyelid of one eye **(Fig 14.6.5)**
  - enophthalmos of opposite eye **(Figs 14.6.6 and 14.6.7)**

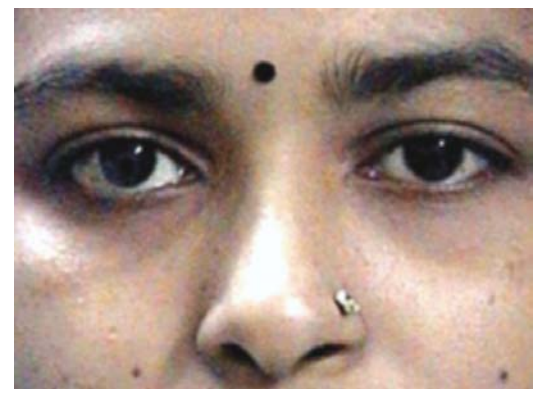


FIGURE 14.6.1: Pseudoproptosis—myopia RE

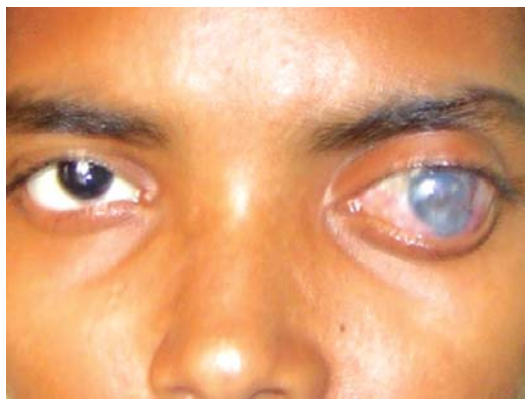


FIGURE 14.6.2: Pseudoproptosis—buphthalmos LE

FIGURE 14.6.3: Pseudoproptosis—pseudocornea RE

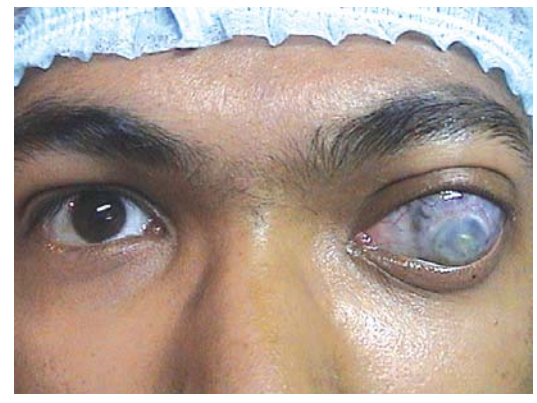


FIGURE 14.6.4: Pseudoproptosis—total staphyloma LE

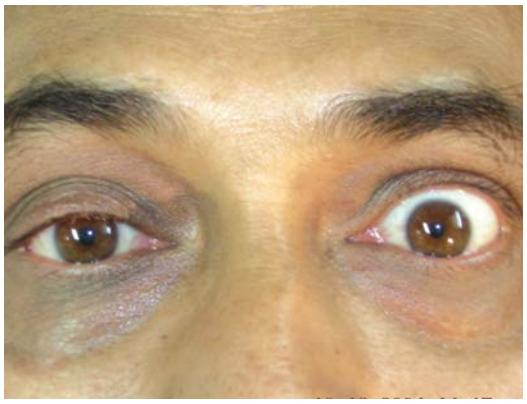


FIGURE 14.6.5: Pseudoproptosis—lid retraction LE

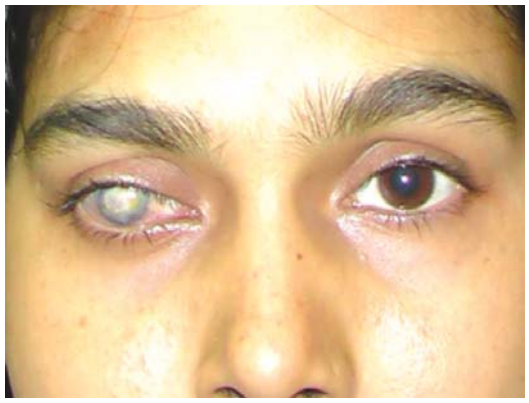


FIGURE 14.6.6: Pseudoproptosis—enophthalmos RE

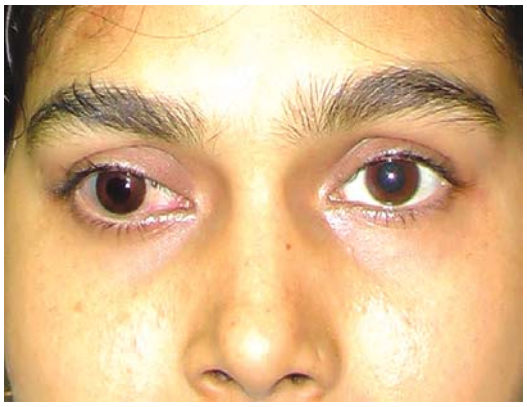


FIGURE 14.6.7: Enophthalmos—after cosmetic CL

Enophthalmos

- A condition in which eyeball is recessed within the orbit
- About 25 percent of patients with enophthalmos are initially misdiagnosed as having contralateral proptosis or ipsilateral ptosis
- *Causes:*
  - phthisis bulbi, unilateral or bilateral **(Figs 14.7.1 to 14.7.3)**
  - microphthalmos or anophthalmos
  - surgical anophthalmos **(Fig 14.7.4)**
  - blowout fracture **(See Fig 16.12.1)**
  - Horner's syndrome

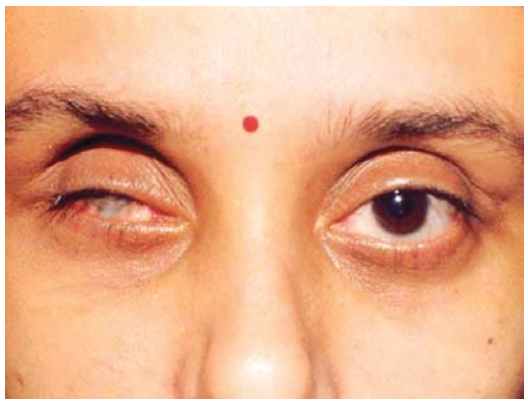


FIGURE 14.7.1: Enophthalmos—phthisis bulbi

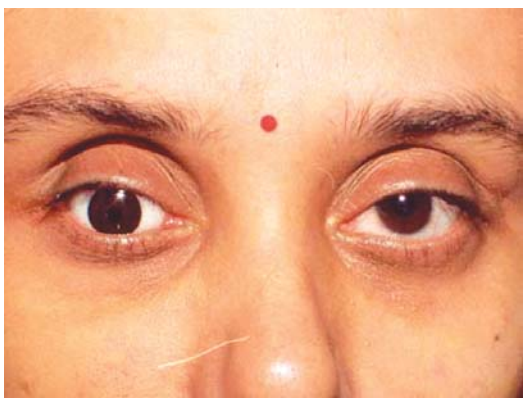


FIGURE 14.7.2: Enophthalmos—after prosthesis

FIGURE 14.7.3: Bilateral enophthalmos

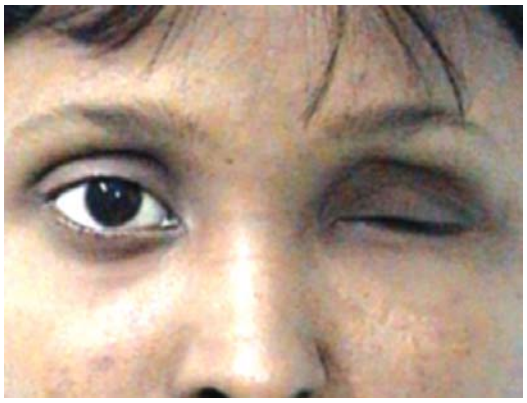


FIGURE 14.7.4: Surgical enophthalmos

PROPTOSIS IN CHILDREN

Craniosynostosis (shallow orbit)

- *Already discussed earlier*

Orbitopalpebral (colobomatous) Cyst

- *Already discussed earlier*

Capillary Hemangioma

- Present at birth or early infancy **(Fig 14.8.1)**
- Slowly growing unilateral proptosis due to hemangiomatous mass in the upper anterior orbit **(Figs 14.8.2 and 14.8.3)**
- Becomes engorged, and may enlarge when the baby cries
- May have similar lesion in eyelids or elsewhere

FIGURE 14.8.1: Capillary hemangioma

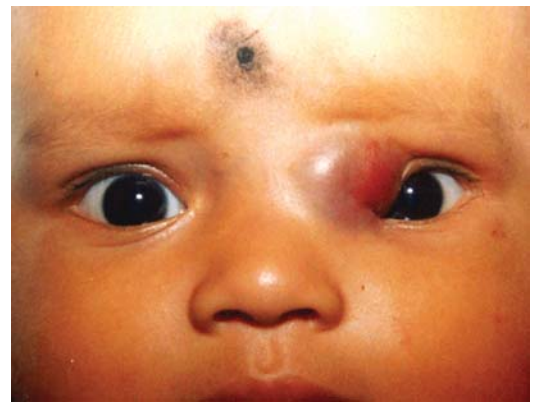


FIGURE 14.8.2: Capillary hemangioma

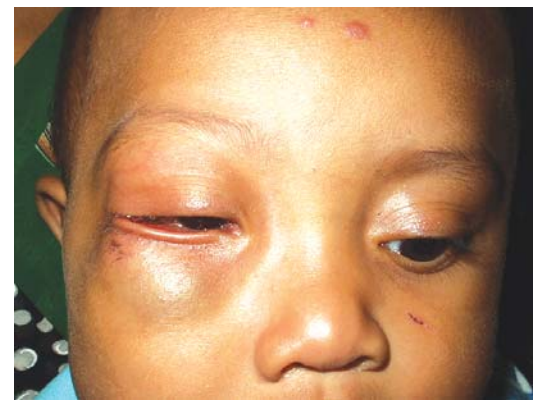


FIGURE 14.8.3: Capillary hemangioma

Orbital Cellulitis

- Usually child or young adult
- Ethmoidal sinusitis — is the commonest cause
- *Preseptal cellulitis*
  - acute periorbital swelling and redness
  - conjunctival chemosis
  - fluctuating mass signifies abscess formation **(Fig 14.9.1)**
- *Orbital cellulitis*
  - very sick child with rise in temperature
  - sudden onset, rapidly growing unilateral proptosis with severe pain and chemosis of lids **(Fig 14.9.2)**
  - eyeball is displaced laterally and downwards **(Fig 14.9.3)**
  - diplopia due to limitation of ocular movements
  - chemosis and congestion of conjunctiva **(Fig 14.9.4)**
- *Treatment:* systemic antibiotics and analgesics, hot compress, topical antibiotics and drainage of orbital abscess if necessary

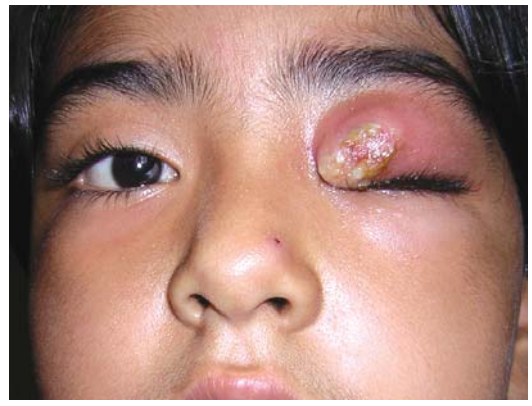


FIGURE 14.9.1: Preseptal cellulitis

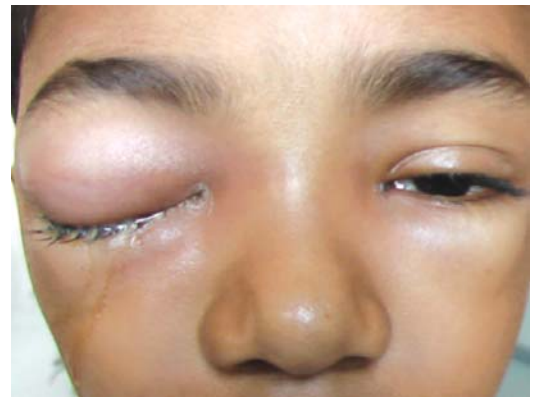


FIGURE 14.9.2: Orbital cellulitis

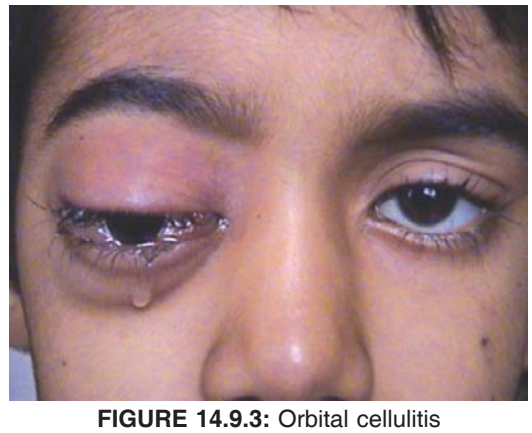


FIGURE 14.9.3: Orbital cellulitis

FIGURE 14.9.4: Orbital cellulitis—chemosis

Lymphangioma

- Benign tumor between 1 to 15 years
- May occur in isolation or be combined with lid or conjunctival lesions
- Soft bluish mass mainly in the superior orbit, which may remain stationary **(Fig 14.10.1)**
- May also involve the sinuses or oropharynx **(Fig 14.10.2)**
- Periodic sudden enlargement with upper respiratory tract infection, or with spontaneous bleeding
- Visual and motility disturbances are common

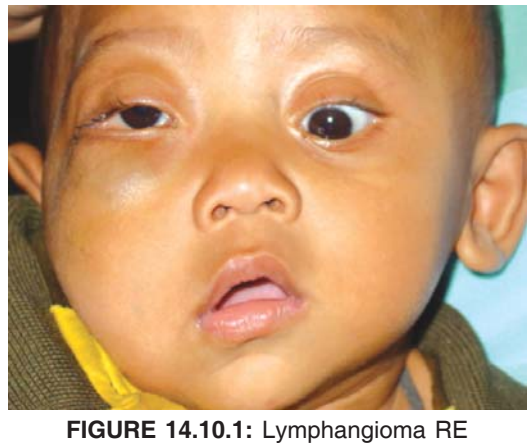


FIGURE 14.10.1: Lymphangioma RE

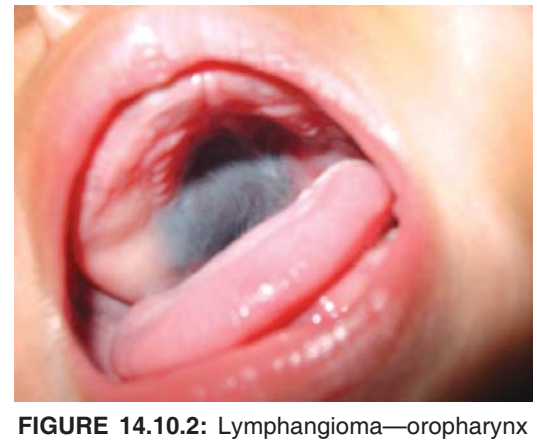


FIGURE 14.10.2: Lymphangioma—oropharynx

Glioma of the Optic Nerve

- Ectodermal benign tumor of the optic nerve
- Most prevalent in childhood between 2 to 8 years
- 50 percent cases may be associated with neurofibromatosis
- Presents as unilateral proptosis with significant visual impairment
- Slow growing unilateral proptosis which is axial **(Figs 14.11.1 and 14.11.2)**
- Marcus Gunn pupil and optic atrophy and divergent squint **(Fig 14.11.3)**
- Enlargement of optic foramen in X-ray is present in 90 percent cases

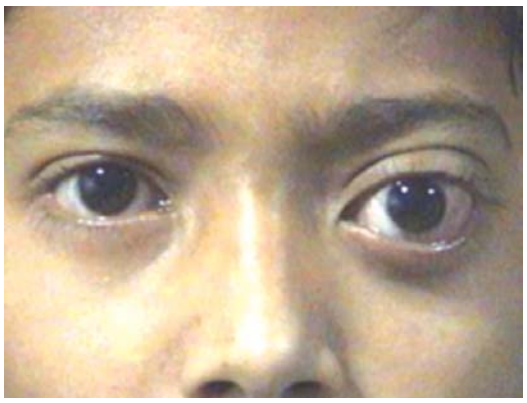


FIGURE 14.11.1: Optic nerve glioma LE

FIGURE 14.11.2: Optic nerve glioma RE

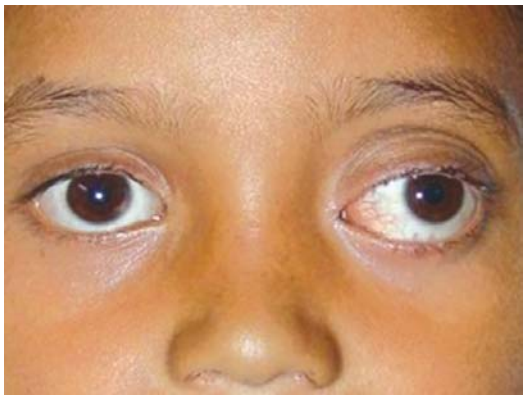


FIGURE 14.11.3: Optic nerve glioma LE

Rhabdomyosarcoma

- Most common primary malignant orbital tumor in children
- Occurs at about 7 years of age and more in boys
- Tumor arises from the striated muscles
- Rapidly growing proptosis with chemosis of conjunctiva and lids **(Fig 14.12.1)**
- Typically, a mass is palpable in the supero-nasal quadrant
- In late stage, it may extend into the cranium **(Fig 14.12.2)**
- *Treatment:* combination of radiotherapy and chemotherapy; in unresponsive cases, orbital exenteration may be required

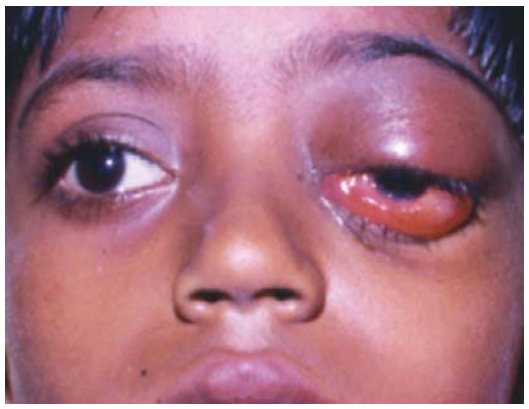


FIGURE 14.12.1: Rhabdomyosarcoma

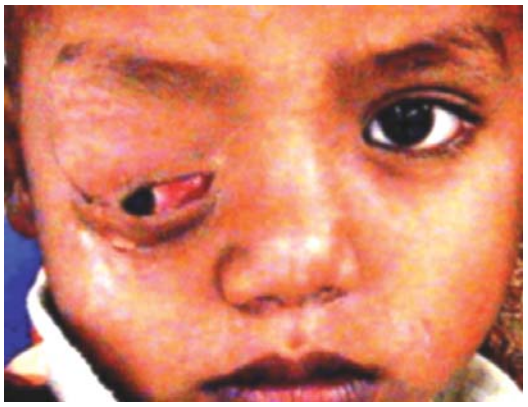


FIGURE 14.12.2: Rhabdomyosarcoma—late stage

Orbital Extension of Retinoblastoma

- In neglected cases, patient with retinoblastoma may present with proptosis due to direct orbital extension **(Figs 14.13.1 and 14.13.2)**
- The child looks toxic in these cases **(Fig 14.13.3)**
- *Treatment:* exenteration followed by radiotherapy
- Prognosis is extremely poor

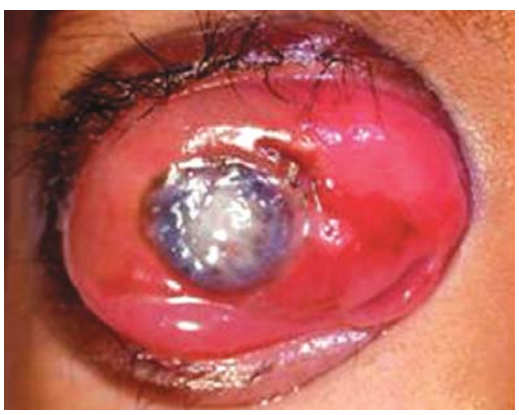


FIGURE 14.13.1: Orbital extension of retinoblastoma

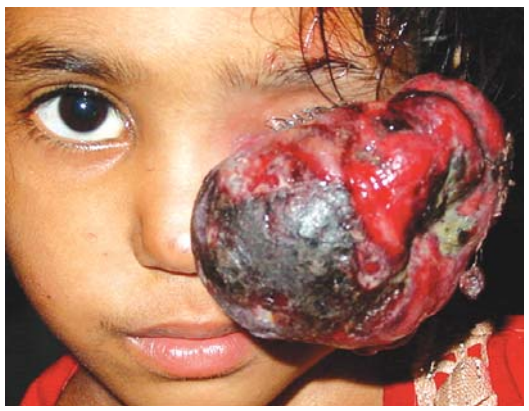


FIGURE 14.13.2: Orbital extension of retinoblastoma—massive

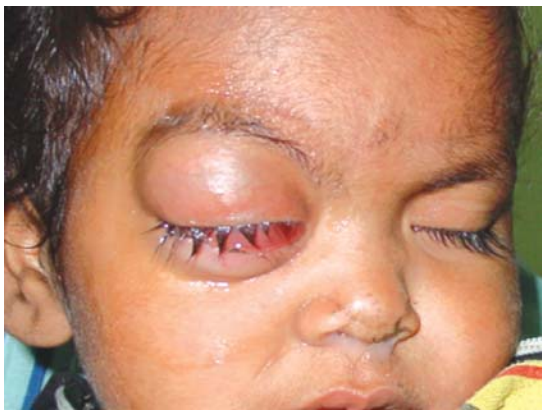


FIGURE 14.13.3: Orbital extension of retinoblastoma toxic child

Acute Leukemia

- Most frequently occurs between 5 to 8 years of age
- Orbital soft tissue involvement in *chloroma* is not associated with abnormal peripheral blood smear which occurs more in African and Asian children
- Unilateral or bilateral rapidly progressive proptosis **(Fig 14.14.1)**
- May be associated with ecchymosis and chemosis of the lids

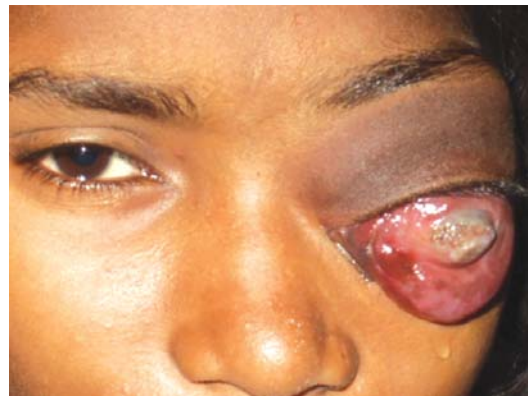


FIGURE 14.14.1: Acute leukemia

OTHER RARE ORBITAL TUMORS IN CHILDREN

- *Metastatic neuroblastoma:* sudden, rapidly growing proptosis with superolateral orbital mass
- *Metastatic Ewing's sarcoma:* rapidly growing proptosis with ecchymosis and chemosis
- *Metastatic Wilms' tumor:* associated with aniridia
- *Fibrosarcoma:* history of previous radiotherapy to orbit for retinoblastoma **(Fig 14.15.1)**
- *Sinus tumors involving the orbit:* osteogenic sarcoma, Burkitt's lymphoma, etc.
- *Enchondromatosis:* very rare, islands of cartilage in metacarpals and phalanges; hemangiomas and enchondromas of orbit gives rise to proptosis (**Figs 14.15.2 and 14.15.3**)
- *Dermoid cyst in the orbit* **(Fig 14.15.4)**
- *Orbital teratoma:* very rare, congenital benign orbital tumor may present with proptosis, rarely turn into malignant germ cell tumor even after resection **(Figs 14.15.5 and 14.15.6)**

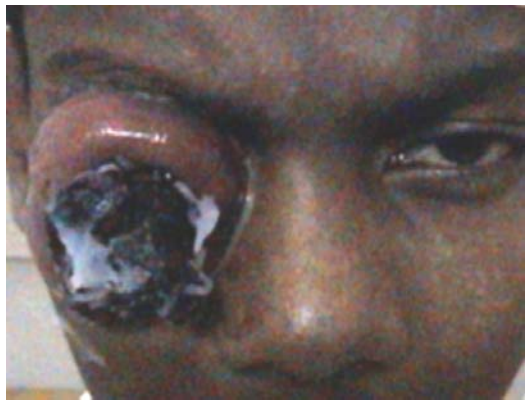


FIGURE 14.15.1: Metastatic fibrosarcoma

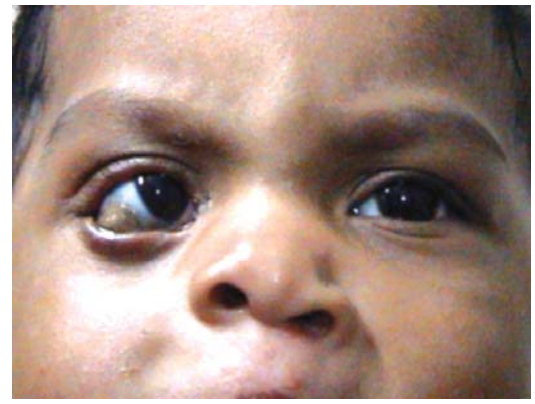


FIGURE 14.15.2: Proptosis—enchondromatosis

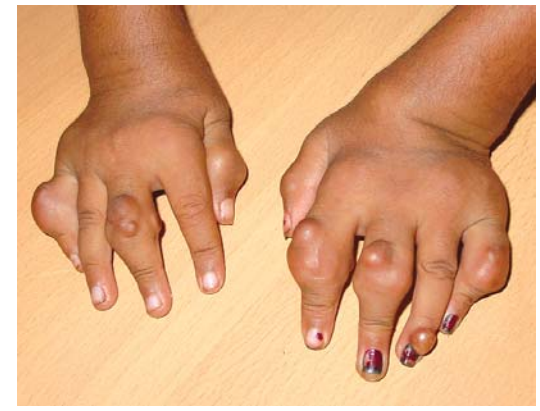


FIGURE 14.15.3: Enchondromatosis—metacarpals and phalanges

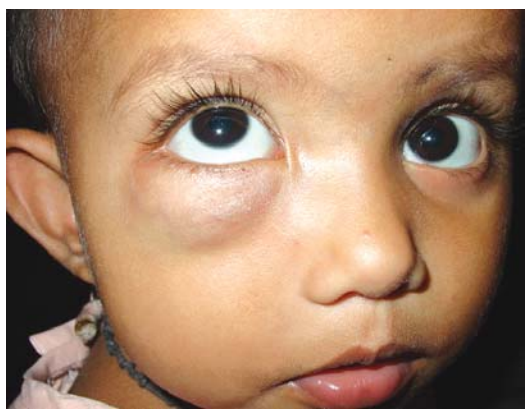


FIGURE 14.15.4: Proptosis—dermoid cyst

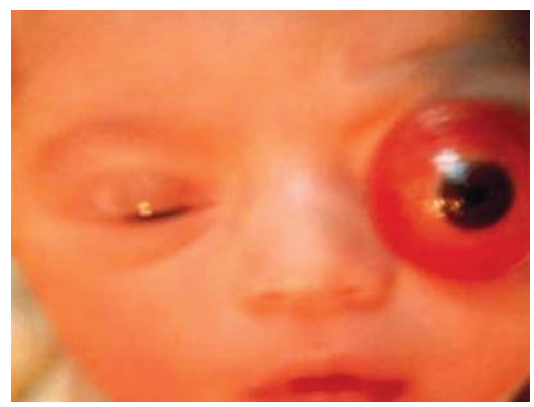


FIGURE 14.15.5: Orbital teratoma—4 day-old-child

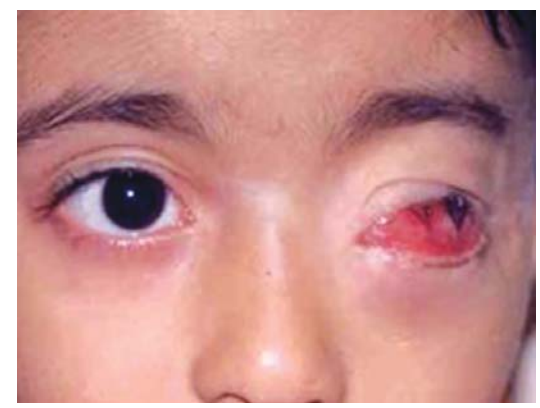


FIGURE 14.15.6: Orbital teratoma—recurrence after 5 years of removal

Juvenile Xanthogranuloma

- Very rare, benign infantile condition
- Unilateral proptosis, often associated with strabismus
- Cutaneous lesions, heterochromia and spontaneous hyphema may be present

PROPTOSIS IN ADULTS

Inflammatory Orbital Diseases (orbital pseudotumor)

- Idiopathic, non-neoplastic, non-microbial space-occupying periocular lesion, which may simulate an orbital neoplasm *(pseudotumor)*
- Typical affects the males between 20 to 50 years
- Unilateral axial proptosis with variable chemosis of the lids and ptosis **(Fig 14.16.1)**
- Conjunctival chemosis and congestion **(Figs 14.16.2 and 14.16.3)**
- Limitation of extraocular movements and diplopia
- To be differentiated from thyroid ophthalmopathy and orbital cellulitis
- *Treatment:* observation, systemic corticosteroids, and cytotoxic agents in resistance cases

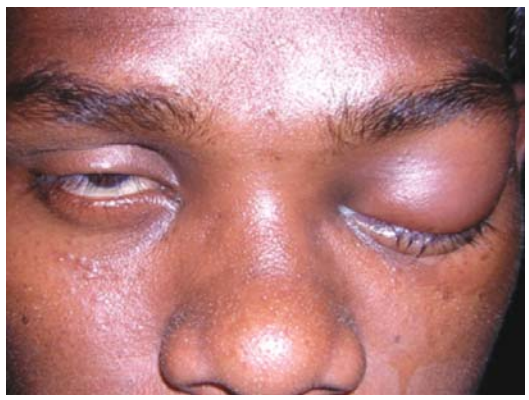


FIGURE 14.16.1: Inflammatory orbital diseases

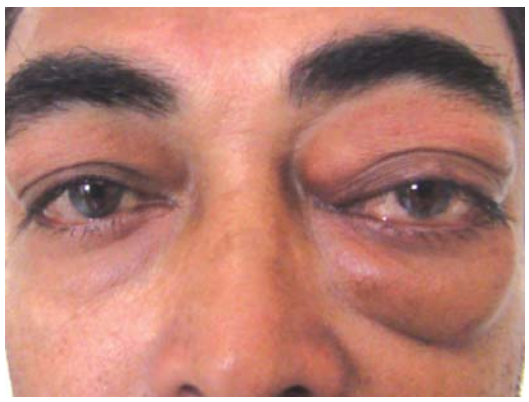


FIGURE 14.16.2: Inflammatory orbital diseases

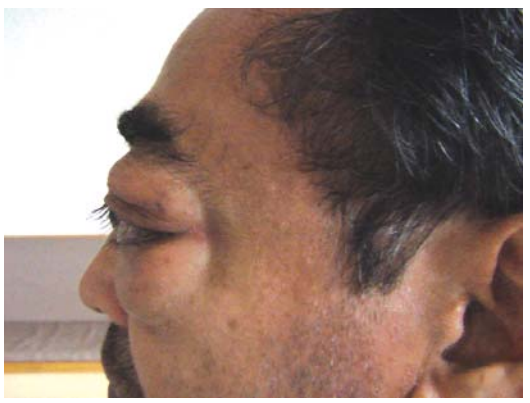


FIGURE 14.16.3: Inflammatory orbital diseases

Orbital Cellulitis in Adults

- More often from infection of adjacent structures like, dacryocystitis, internal hordeolum, dental infection
- May be post-traumatic or post-surgical
- Painful, unilateral rapid proptosis with severe lid edema **(Fig 14.17.1)**
- To be differentiated from preseptal cellulitis and lid abscess **(Fig 14.17.2)**
- Limitation of ocular movements

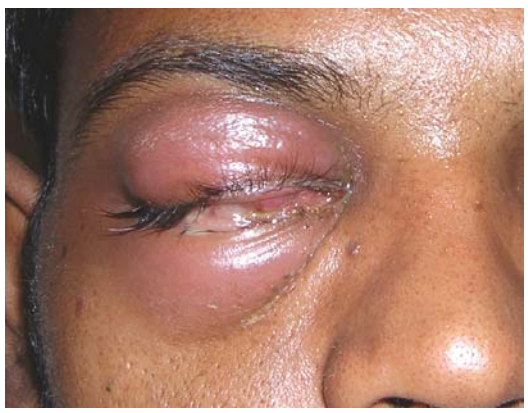
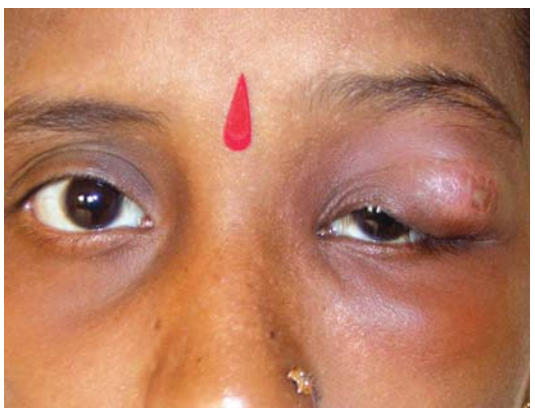


FIGURE 14.17.1: Orbital cellulitis—adult



FIGUGRE 14.17.2: Preseptal cellulitis

Thyroid Associated Ophthalmopathy

- Most common cause of proptosis in adults
- Unilateral **(Figs 14.18.1 and 14.18.2)** or bilateral chronic slow growing axial proptosis **(Figs 14.18.3 and 14.18.4)**
- Retraction of the eyelid, lid lag (**Fig 14.18.5)**
- Hyperemia of conjunctiva near horizontal rectus muscles (**Fig 14.18.6)**
- Extraocular muscle involvement (most common – inferior rectus, then medial rectus, least common – lateral rectus) with restriction of movements

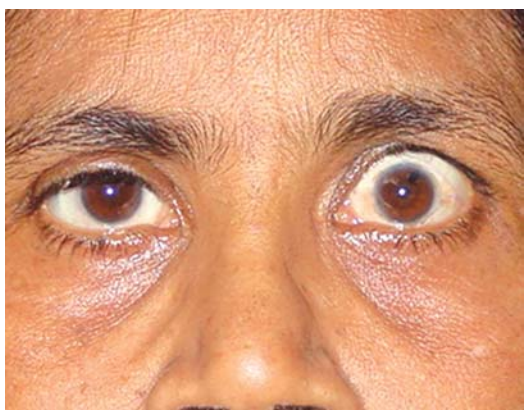


FIGURE 14.18.1: Thyroid exophthalmos—unilateral

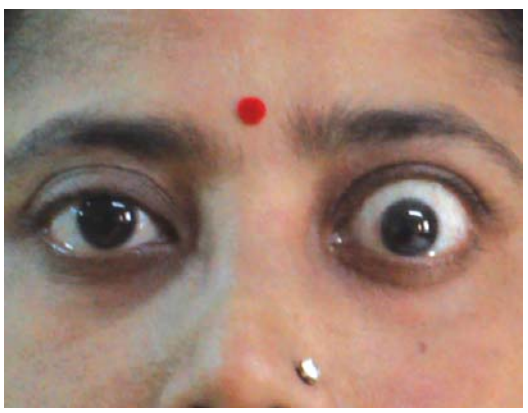


FIGURE 14.18.2: Thyroid exophthalmos—unilateral

- Conjunctival chemosis and severe congestion in advance cases
- Superior limbic keratoconjunctivitis
- Corneal involvement like, exposure keratopathy or dry eye in late stage
- Thyroid swelling may be obvious in some cases (**Fig14.18.7**)
- Compressive optic neuropathy disk edema, and chorioretinal folds in late stage **(Fig 14.18.8)**
- *Treatment:* antithyroid drugs, artificial tears, systemic corticosteroids or cytotoxic agents, lateral tarsorrhaphy and orbital decompression in severe cases

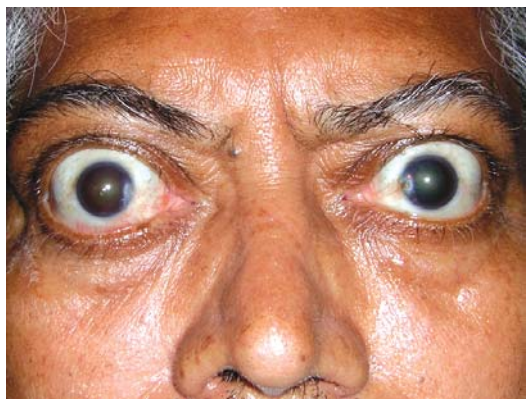


FIGURE 14.18.3: Thyroid exophthalmos

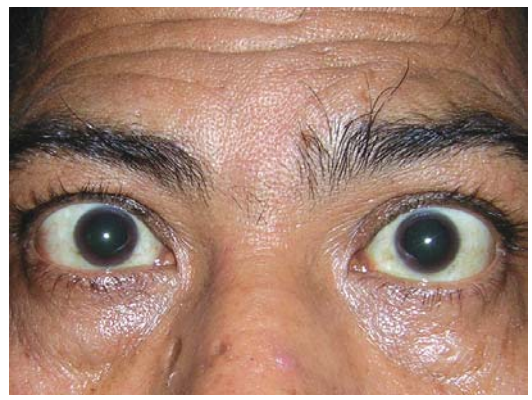


FIGURE 14.18.4: Thyroid exophthalmos

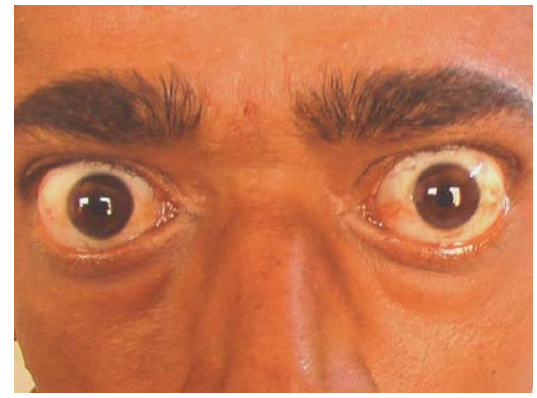


FIGURE 14.18.5: Thyroid exophthalmos

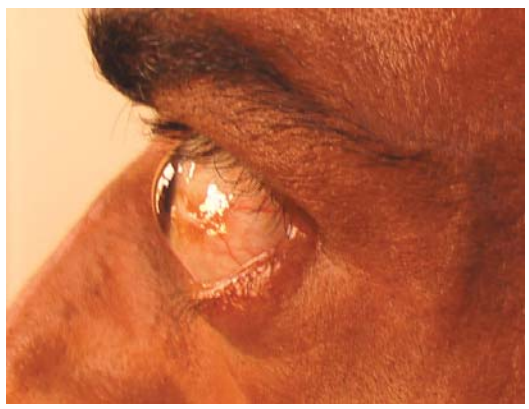


FIGURE 14.18.6: Thyroid exophthalmos

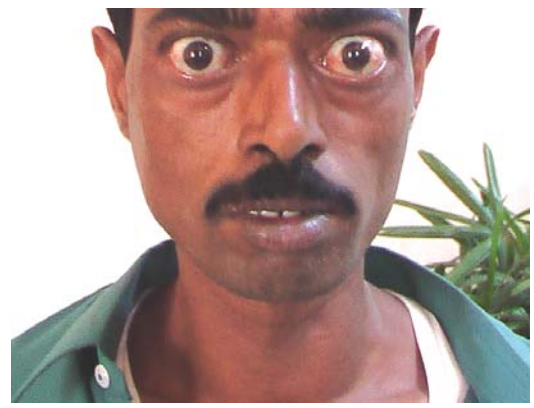


FIGURE 14.18.7: Thyroid exophthalmos—thyroid swelling

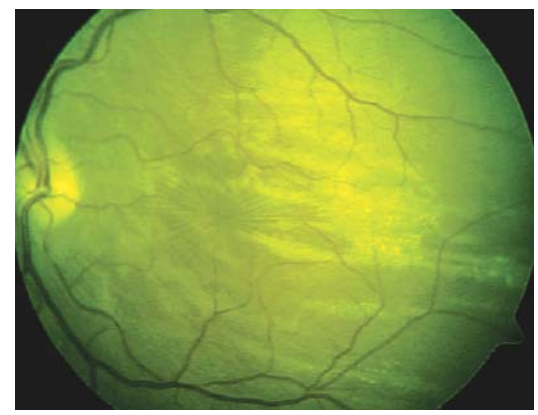


FIGURE 14.18.8: Thyroid ophthalmopathy—choroidal folds

Cavernous Hemangioma

- Most common benign tumor in adults
- Presents between 30 to 50 years of age
- Unilateral, slowly progressive proptosis which is usually axial **(Figs 14.19.1 and 14.19.2)**
- May be associated with chemosis of the conjunctiva **(Fig 14.19.3)**
- Usually not associated with optic nerve compression unless it is at the orbital apex

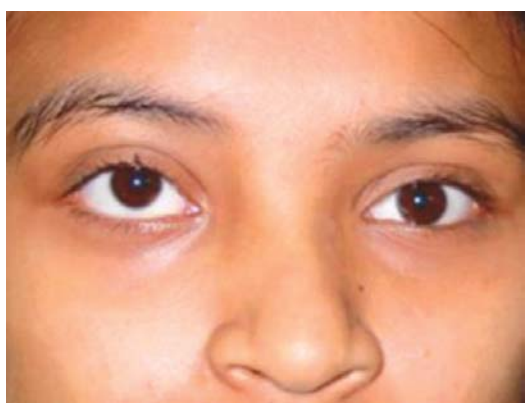


FIGURE 14.19.1: Cavernous hemangioma

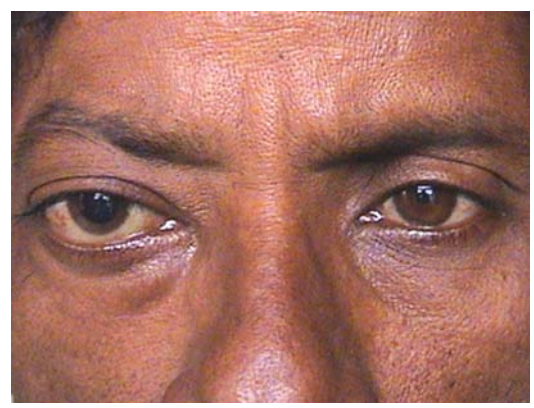


FIGURE 14.19.2: Cavernous hemangioma

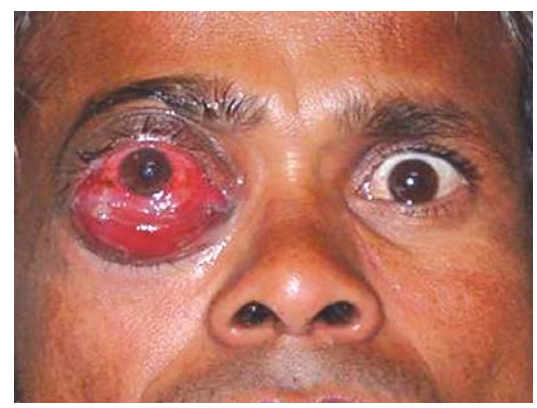


FIGURE 14.19.3: Cavernous hemangioma

Orbital Varix

- Usually unilateral, vascular malformation, can occur at any age
- Non pulsatile, intermittent axial proptosis, not associated with a bruit **(Fig 14.20.1)**
- Proptosis may be accentuated or precipitated by dependent head posture or by performing Valsalva maneuver **(Figs 14.20.2 to 14.20.4)**
- Associated vascular lesion of the eyelids or conjunctiva **(Figs 14.20.5 and 14.20.6)** is a common finding

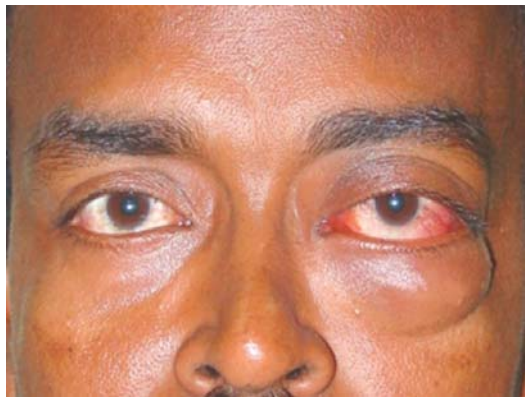


FIGURE 14.20.1: Orbital varix

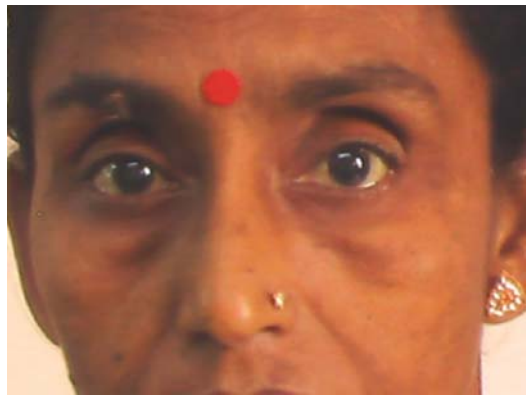


FIGURE 14.20.2: Orbital varix LE

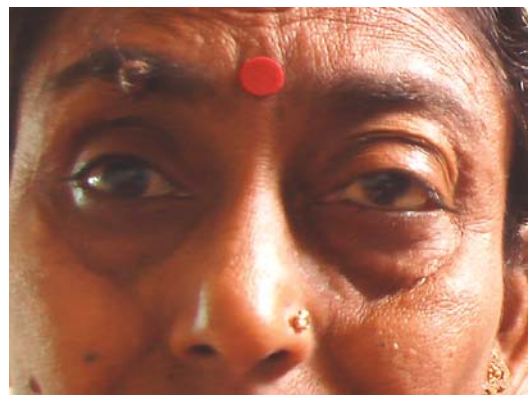


FIGURE 14.20.3: Proptosis LE—just after head down position

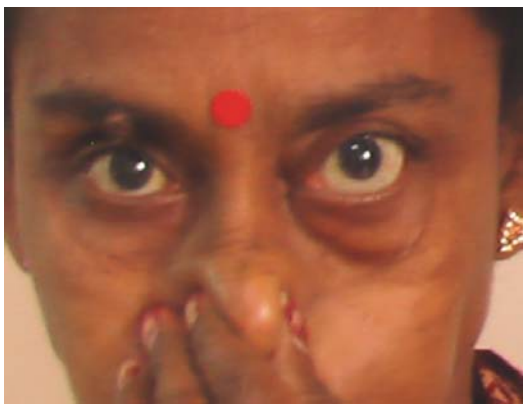


FIGURE 14.20.4: Orbital varix—proptosis LE with Valsalva maneuver

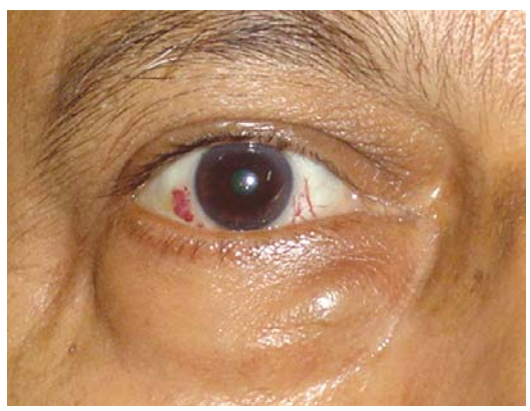


FIGURE 14.20.5: Orbital varix—vascular malformation of the lid

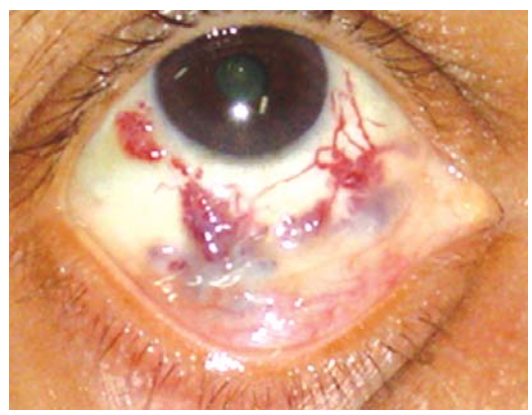


FIGURE 14.20.6: Orbital varix—vascular malformation of the conjunctiva

Meningioma of Optic Nerve

- Occurs predominantly in the middle-aged women
- Ocular features are related to the site of primary involvement by the tumor
- *Tuberculum sellae meningioma:* compress the optic chiasma
- *Sphenoidal-ridge meningioma:*
  - slowly growing, painless down and out proptosis **(Fig 14.21.1)**
  - fullness of temporal fossa **(Fig 14.21.2)**
  - visual impairment due to optic nerve dysfunction
- *Optic nerve-sheath meningioma:* slowly-growing unilateral axial proptosis **(Fig 14.21.3)**
  - *the triad* of long-standing visual impairment, a pale swollen optic disk, and opticociliary shunt vessels is virtually pathognomonic
- *Treatment:* surgical excision of the tumor by lateral orbitotomy

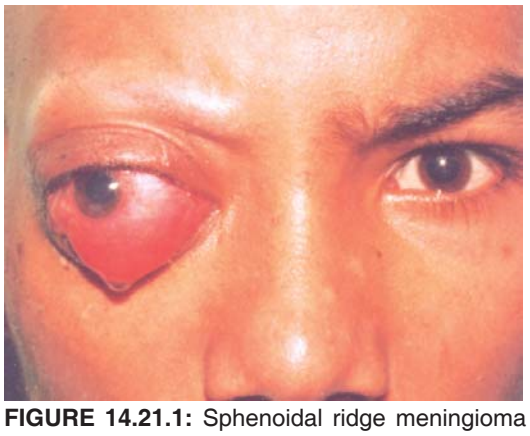


FIGURE 14.21.1: Sphenoidal ridge meningioma

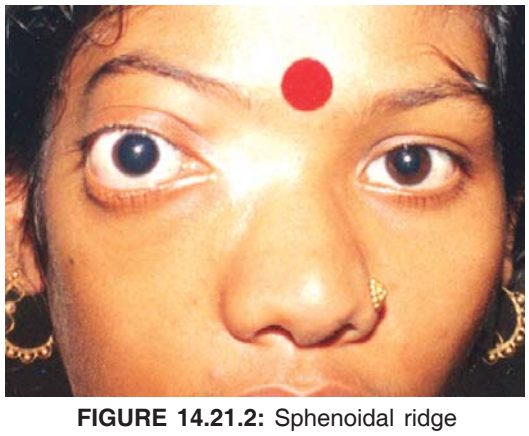


FIGURE 14.21.2: Sphenoidal ridge meningioma—temporal fossa fullness

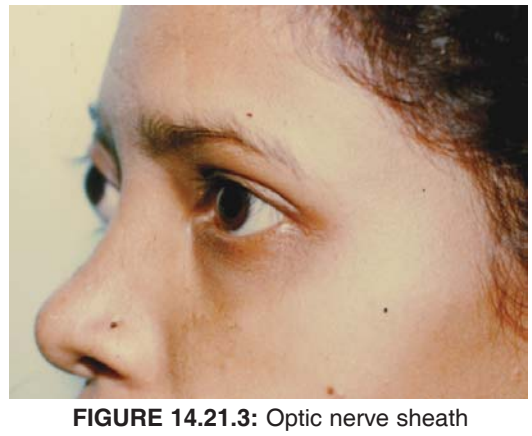


FIGURE 14.21.3: Optic nerve sheath meningioma right side

Carotid Cavernous Fistula

- Results from an abnormal communication between the cavernous sinus and the internal carotid artery, giving the classical picture of a pulsating exophthalmos **(Figs 14.22.1 and 14.22.2)**
- May be traumatic or spontaneous
- Marked unilateral, sudden pulsatile proptosis
- Chemosis **(Fig 14.22.3)**, redness and dilation of the episcleral blood vessels *(caput medusae)* **(Figs 14.22.4 and 14.22.5)**
- Ophthalmoplegia due to involvement of the 3rd, 4th and 6th nerve **(See Fig 15.14.1)**
- Retinal venous congestion with hemorrhage—CRVO in extreme cases **(Fig 14.22.6)**
- Raised IOP, due to elevated episcleral venous pressure
- Anterior segment ischaemia may develop in some patients
- *Treatment:* intracavernous surgery and balloon catheter embolization

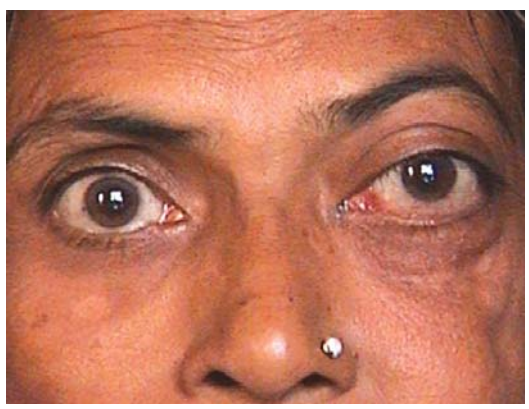


FIGURE 14.22.1: Caroticocavernous fistula

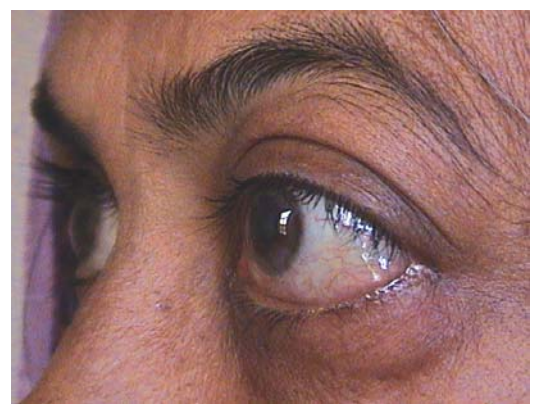


FIGURE 14.22.2: Carotico cavernous fistula

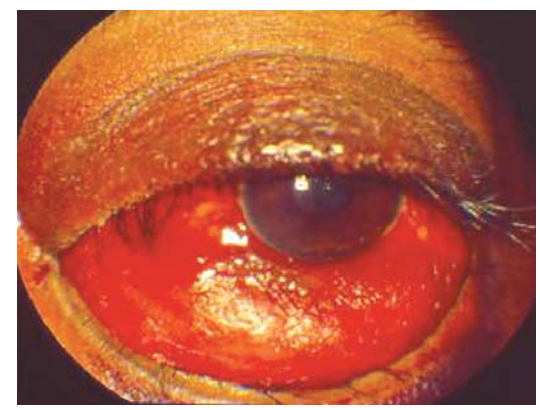


FIGURE 14.22.3: Caroticocavernous fistula—conjunctival chemosis

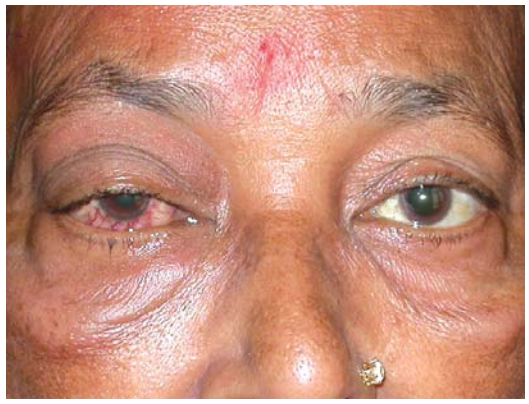


FIGURE 14.22.4: Caroticocavernous fistula

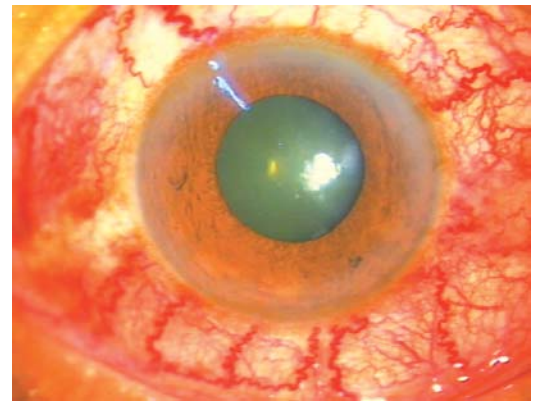


FIGURE 14.22.5: Caput medusae in same patient (Fig. 14.22.4)

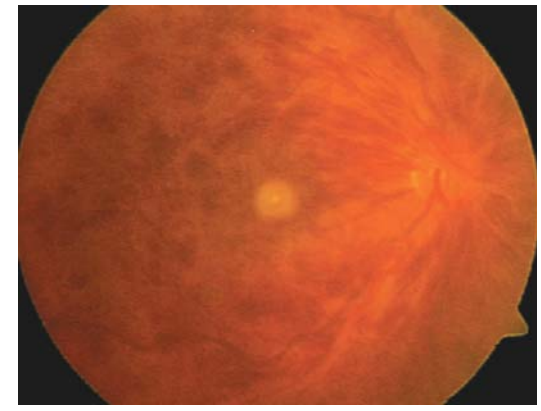


FIGURE 14.22.6: CRVO in same patient (Fig. 14.22.4)

Cavernous Sinus Thrombosis

- An acute thrombophlebitis with violent onset
- Features are almost similar to caroticocavernous fistula
- Edema of the mastoid region indicating back-pressure in the mastoid emissary vein—a *pathognomonic sign*
- *Treatment:* intensive intravenous antibiotic and anticoagulants

OTHER CAUSES OF PROPTOSIS IN ADULTS

- **Lymphoid Tumors**
  - usually in old age
  - unilateral or bilateral with orbital puffiness **(Fig 14.23.1)**
  - rubbery consistency on palpation
  - associated conjunctival extension

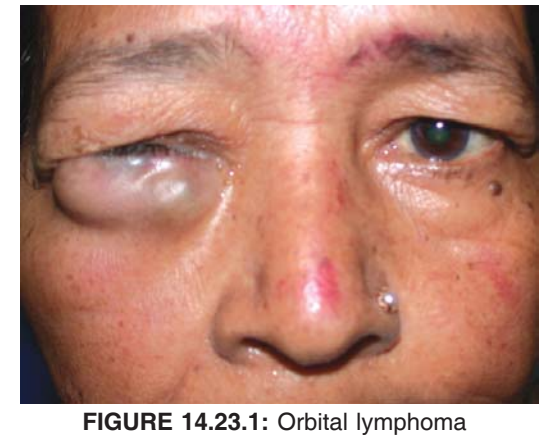


FIGURE 14.23.1: Orbital lymphoma

- **Optic nerve glioma**
  - as in children
  - slow growing axial proptosis with visual loss **(Fig 14.23.2)**
- **Intraorbital dermoid**
  - slow growing unilateral axial or non-axial proptosis **(Fig 14.23.3)**
- **Pleomorphic adenoma of lacrimal gland**
  - downward and medial proptosis **(Fig 14.23.4)**
- **Metastatic tumors**
  - relatively rapid onset proptosis
  - often with pain and diplopia
  - severe chemosis and conjunctival congestion
  - *primary site*: bronchus, prostate, breast or gastrointestinal tract **(Fig 14.23.5)**
  - extension from the paranasal sinuses **(Figs 14.23.6 to 14.23.8)**
  - leukemic deposits also occur in adults **(Figs 14.23.9 and 14.23.10)**

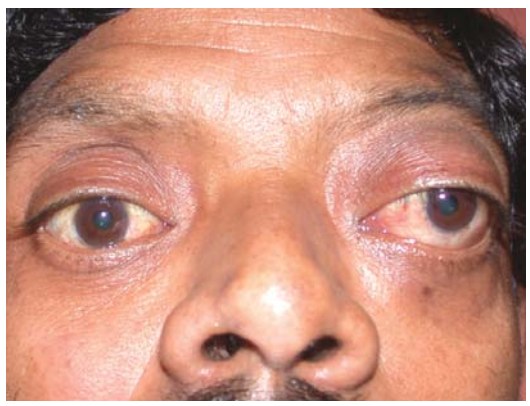


FIGURE 14.23.2: Glioma optic nerve—adult

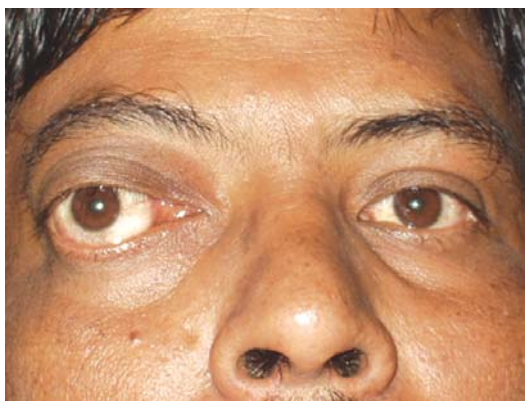


FIGURE 14.23.3: Intraorbital dermoid

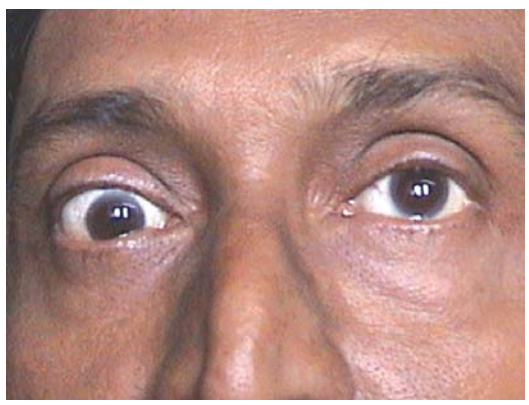


FIGURE 14.23.4: Proptosis RE—pleomorphic adenoma of lacrimal gland

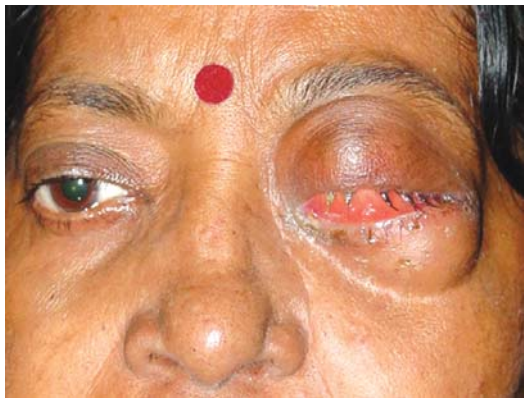


FIGURE 14.23.5: Metastatic deposits—breast carcinoma

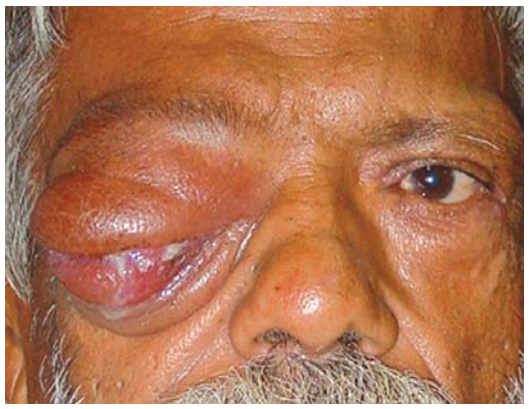


FIGURE 14.23.6: Proptosis—from ethmoidal sinus carcinoma

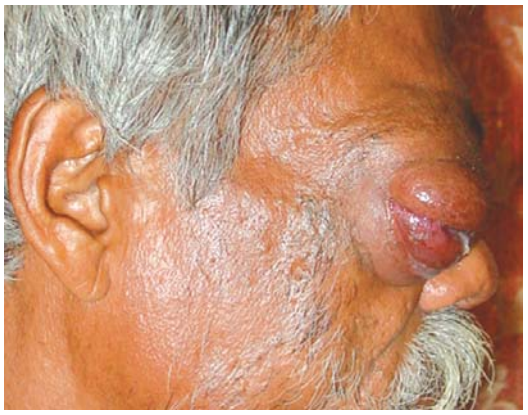


FIGURE 14.23.7: Proptosis—from ethmoidal sinus carcinoma

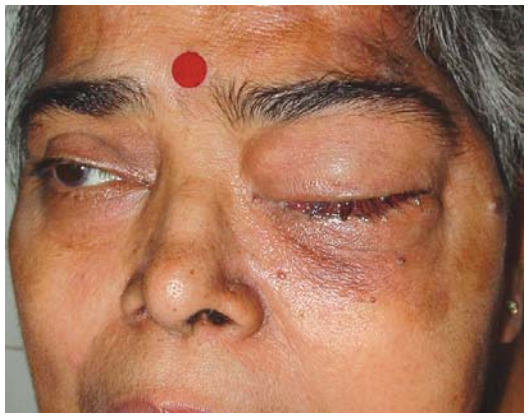


FIGURE 14.23.8: Proptosis—from maxillary sinus carcinoma

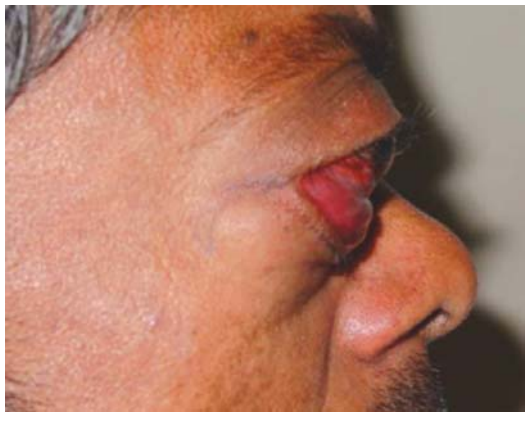


FIGURE 14.23.9: Metastatic deposits—leukemia

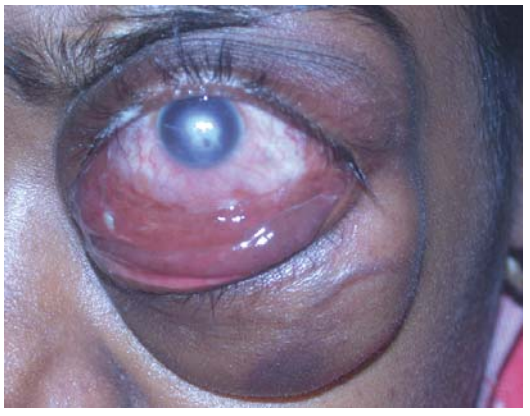


FIGURE 14.23.10: Metastatic deposits—leukemia

MISCELLANEOUS ORBITAL LESIONS

Orbital Rim Lesions

- Almost always unilateral and may be any meridian
- Associated with bony defects
- *Causes:*
  - *external angular dermoid*: mostly at the upper and outer quadrant **(Fig 14.24.1)**
  - subperiosteal lesions
  - *cavernous hemangioma* **(Fig 14.24.2)**
  - fracture orbital margin
  - *plexiform neurofibromatosis* **(Fig 14.24.3)**

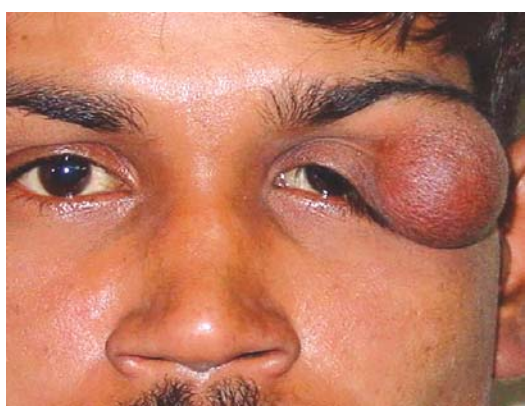


FIGURE 14.24.1: External angular dermoid

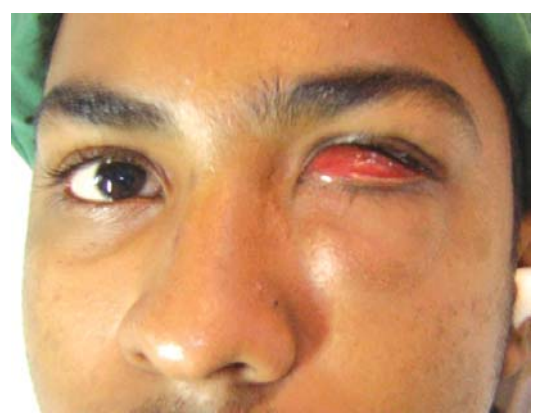


FIGURE 14.24.2: Cavernous hemangioma

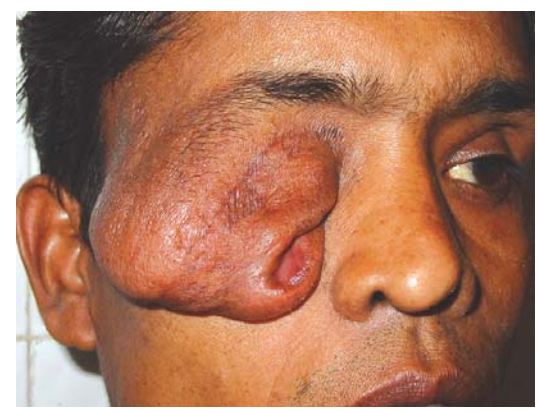


FIGURE 14.24.3: Plexiform neurofibromatosis

Orbital Fat Prolapse

- Not so uncommon, occurs in old age
- Unilateral or bilateral, often mistaken as lymphoid hyperplasia
- Associated with defects in orbital septum and baggy eyelids **(Figs 14.25.1 and 14.25.2)**

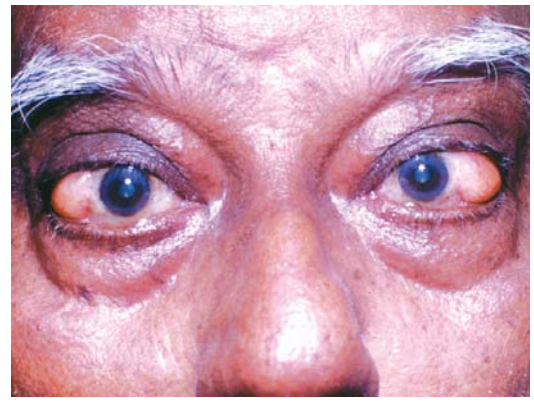


FIGURE 14.25.1: Prolapsed orbital fat—baggy lids

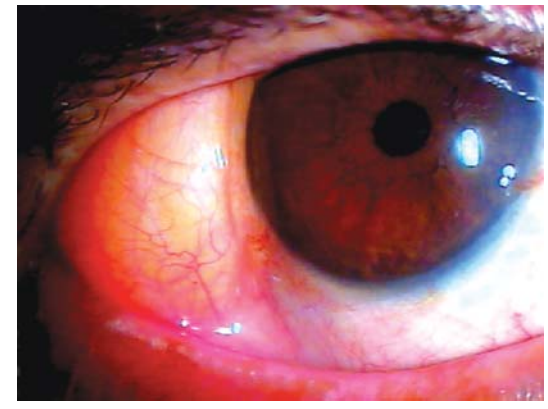


FIGURE 14.25.2: Prolapsed orbital fat

Luxatio Bulbi

- Extremely rare, usually unilateral
- Whole globe is luxated out of the orbital cavity **Fig 14.26.1)**
- Severe chemosis of the conjunctiva, sometimes with anterior segment ischaemia
- In cases of delay, there may be exposure keratitis with eventual corneal ulcer **(Fig 14.26.2)**
- *Causes:* blunt trauma, malignant thyroid exophthalmos, psychiatric problem (*oedipism*), etc.
- *Treatment:* required urgently to save the eye-ball

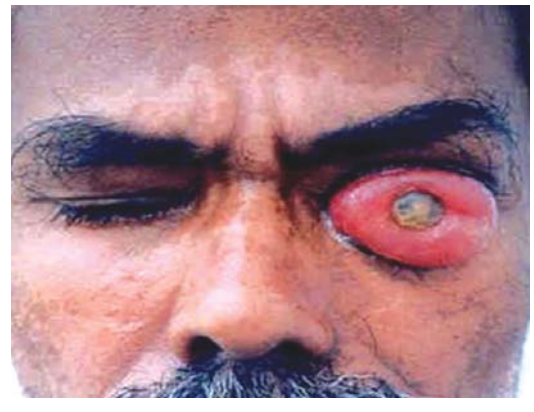


FIGURE 14.26.1: Luxatio bulbi—malignant thyroid exophthalmos

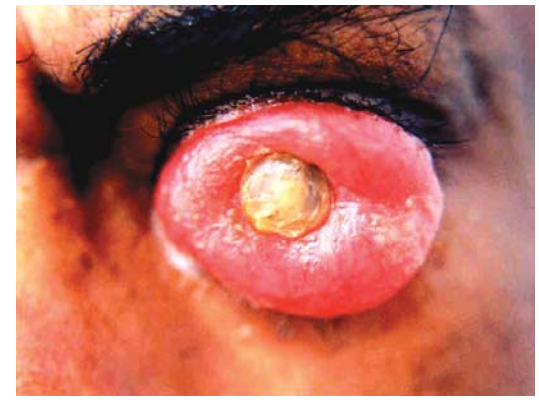


FIGURE 14.26.2: Luxatio bulbi—severe chemosis

Contracted Socket

- Occurs due to chronic inflammation, trauma, prior ocular surgeries, cicatricial ocular pemphigoid, etc.
- Resulting in conjunctival shortage, leading to shallowing of the fornices and inability to wear the artificial shell **(Fig 14.27.1)**
- *Types:*
  - *Grade I:* shallowing of inferior fornix **(Fig 14.27.2)**
  - *Grade II:* shallowing of both inferior and superior fornices **(Fig 14.27.3)**
  - *Grade III:* formation of symblepharon bands **(Fig 14.27.4)**
  - *Grade IV:* gross contracture of whole socket **(Fig 14.27.5)**
  - *Grade V:* wet, discharging foul socket
- *Treatment:* reconstructive procedures like, fornix forming sutures, buccal mucous membrane graft and dermis fat graft, etc. **(See Fig 14.20.5)**; *exposed orbital implant:* not uncommon with recurrent orbital infection **(Fig 14.27.6)**

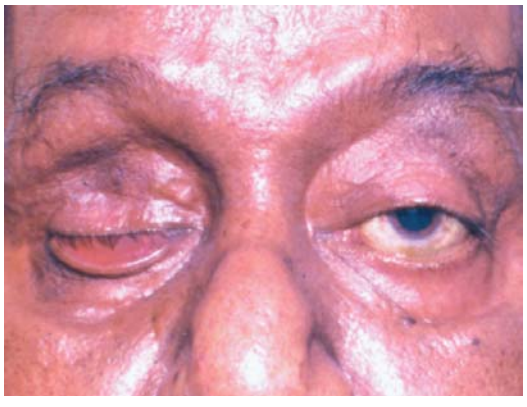


FIGURE 14.27.1: Contracted socket RE

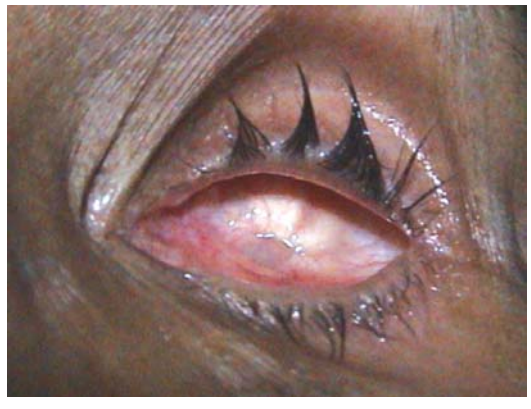


FIGURE 14.27.2: Contracted socket—grade I

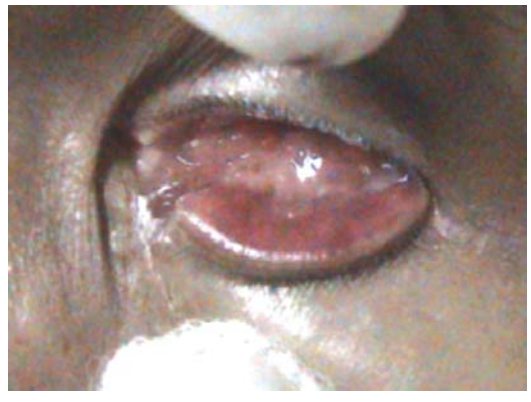


FIGURE 14.27.3: Contracted socket—grade II

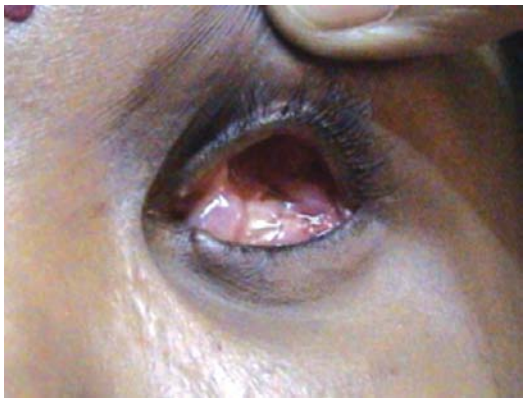


FIGURE 14.27.4: Contracted socket—grade III

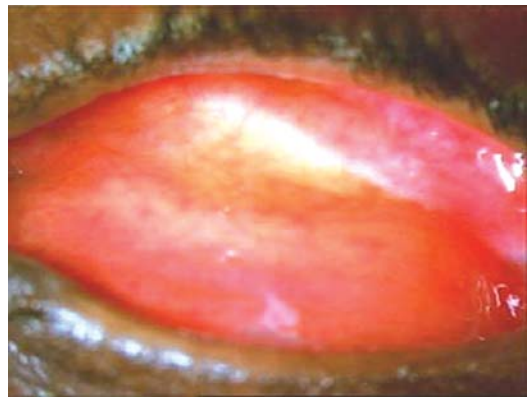


FIGURE 14.27.5: Contracted socket—grade IV

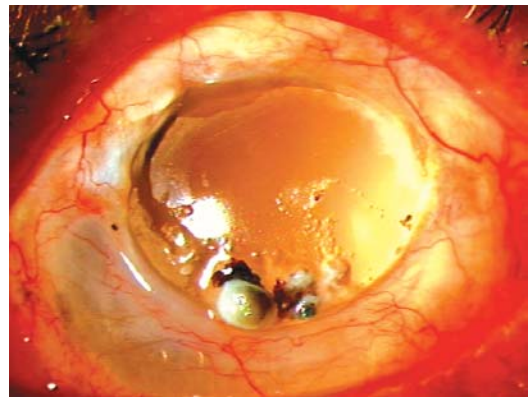


FIGURE 14.27.6: Exposure of orbital implant

15 Ocular Motility Disturbances and Squint

APPARENT SQUINT (PSEUDO-STRABISMUS)

CONCOMITANT SQUINT

- Convergent concomitant squint (esotropia)
- Divergent concomitant squint (exotropia)
- Alternating squint
- Vertical squint
- Inferior oblique overaction

PARALYTIC SQUINT

- Total ophthalmoplegia
- External ophthalmoplegia
- Third (Oculomotor) nerve palsy
- Fourth (Trochlear) nerve palsy
- Sixth (Abducens) nerve palsy

MISCELLANEOUS OCULAR MOVEMENT DISORDERS

- Duane's retraction syndrome
- Superior oblique tendon-sheath syndrome (Brown's syndrome)
- Double elevator palsy
- Strabismus fixus

APPARENT SQUINT

(PSEUDO-STRABISMUS)

- Here, the visual axes are in fact parallel, but the eyes seem to have squint.
- *Causes:*
  - *prominent epicanthic folds:* may simulate a convergent squint **(Fig 15.1.1)**
  - *telecanthus:* also simulate a convergent squint **(Fig 15.1.2)**
  - *hypertelorism:* may simulate a divergent squint
  - *hypermetropia:* an apparent divergent squint due to large angle *kappa*
  - *myopia:* an apparent convergent squint due to smaller or even negative angle *kappa*
- Squint in these conditions may be easily excluded by checking the relative position of corneal light reflections and also by other means

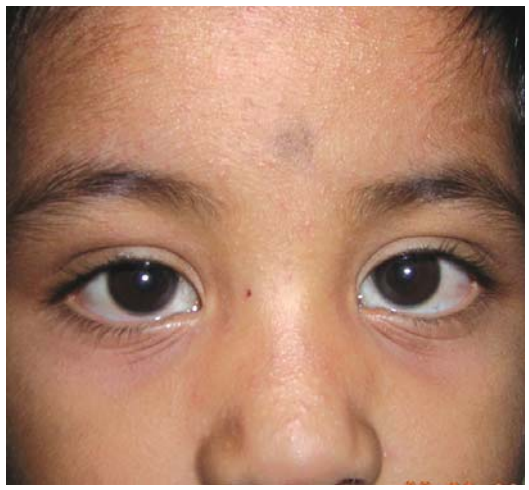


FIGURE 15.1.1: Epicanthus—pseudo-convergent squint

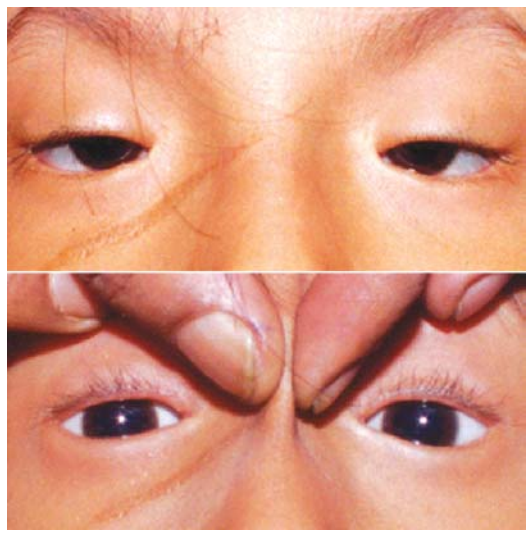


FIGURE 15.1.2: Telecanthus—pseudo-convergent squint

CONCOMITANT SQUINT

- Angle of deviation remains the same in all directions of gaze, irrespective of eyes of fixation
- May be *monocular* or *alternating*
- In 85% cases the squint is *monocular,* in the sense one eye habitually takes up fixation and the other eye is squinting eye

Convergent Concomitant Squint (esotropia)

- One eye always deviated inwards
- Typically develops in early childhood before the binocular reflexes are firmly established
- Amblyopia is a frequent association
- *Types of concomitant esotropia:*
- *Infantile esotropia*
  - develops before the age of 6 months **(Figs 15.2.1 and 15.2.2)**
  - large and stable angle
  - may have alternate or cross fixation **(Fig 15.2.3)**
  - normal refraction for age
  - poor potential for binocular vision
  - inferior oblique overaction may be present initially or develop later **(Fig 15.2.4)**

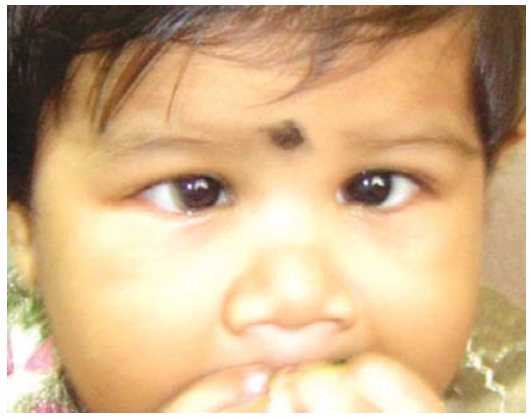


FIGURE 15.2.1: Infantile esotropia

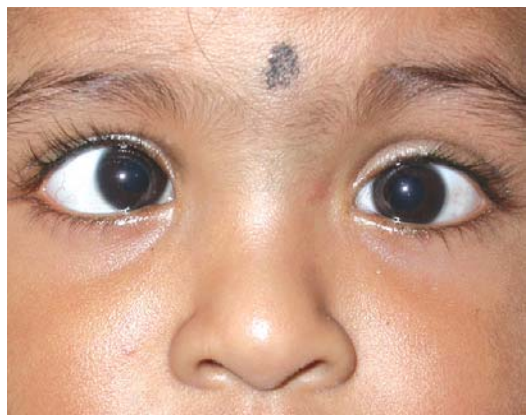


FIGURE 15.2.2: Infantile esotropia

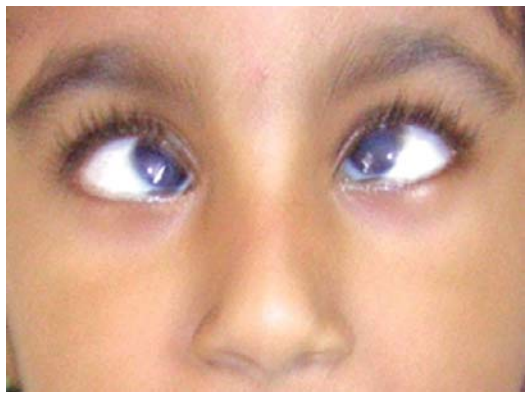


FIGURE 15.2.3: Infantile esotropia—cross fixation

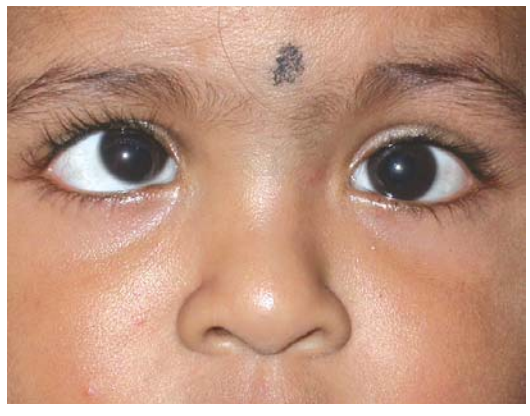


FIGURE 15.2.4: Infantile esotropia—IO overaction

- *Accommodative esotropia*
  - *fully accommodative*: spectacles fully correct the deviation **(Fig 15.3.1)**
  - *partially accommodative*: spectacles partially correct the deviation **(Fig 15.3.2)**
  - onset is between 2 and 3 years, rarely earlier
  - hypermetropia
  - atropine refraction is a must

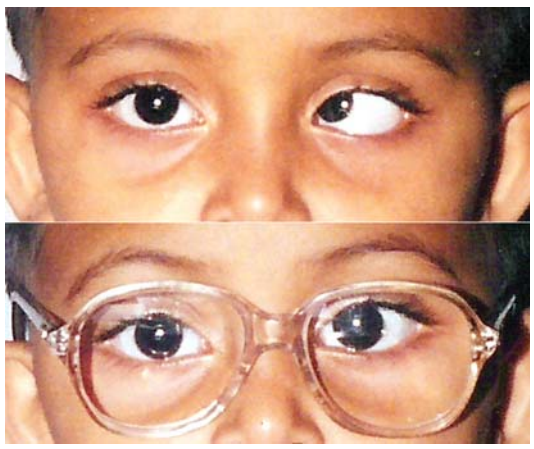


FIGURE 15.3.1: Fully accommodative convergent squint

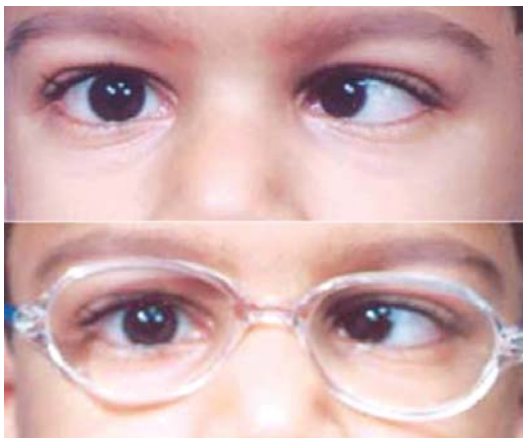


FIGURE 15.3.2: Patially accommodative convergent squint

- *Non-accommodative esotropia*
  - onset during childhood, but after 6 months of age
  - insignificant refractive error
  - no excess accommodative element
  - angle of deviation same for distance and near fixation **(Figs 15.4.1 and 15.4.2)**
- *Non-refractive accommodative esotropia*
  - onset between 6 months and 3 years
  - with high AC/A ratio, with increased accommodative convergence
  - normal near point of accommodation
  - no significant refractive error
  - straight eyes for distance, but esotropia for near
- *Sensory esotropia*
  - caused by unilateral reduction in visual acuity at an early age which interferes or abolishes fusion
  - other eye remains normal
  - *causes:* congenital cataract, corneal opacity, coloboma, microphthalmos, etc. **(Figs 15.5.1 and 15.5.2)**

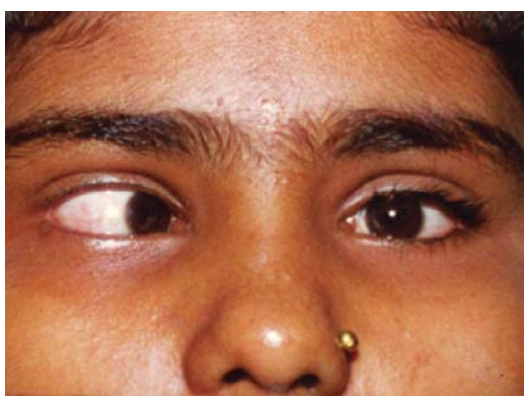


FIGURE 15.4.1: Non-accommodative convergent squint

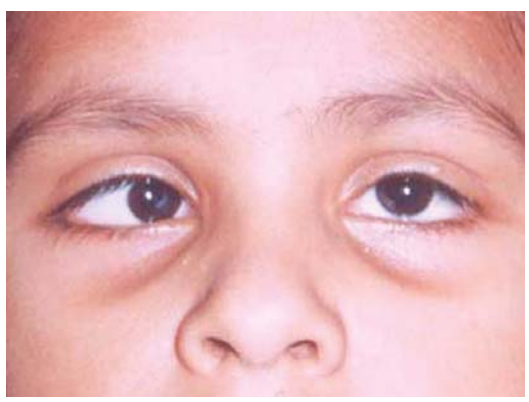


FIGURE 15.4.2: Non-accommodative convergent squint

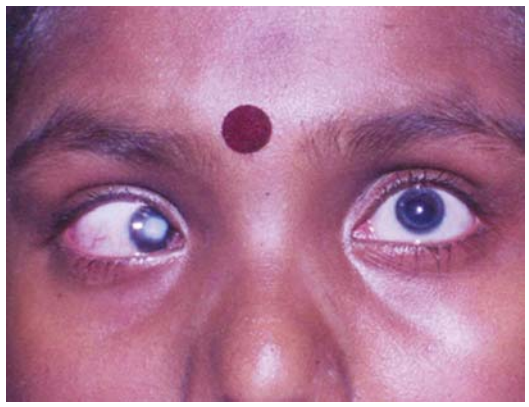


FIGURE 15.5.1: Sensory esotropia

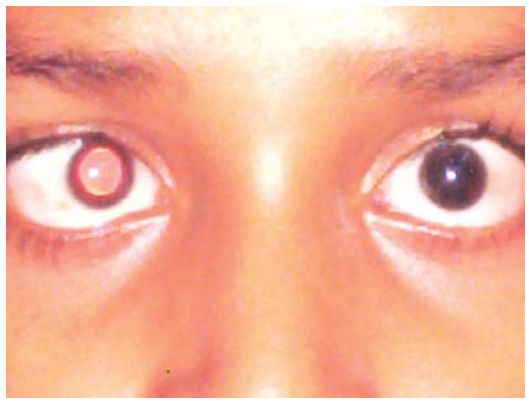


FIGURE 15.5.2: Sensory esotropia

- *Consecutive esotropia*
  - occurs after surgical overcorrection of an exotropia **(Fig 15.6.1)**

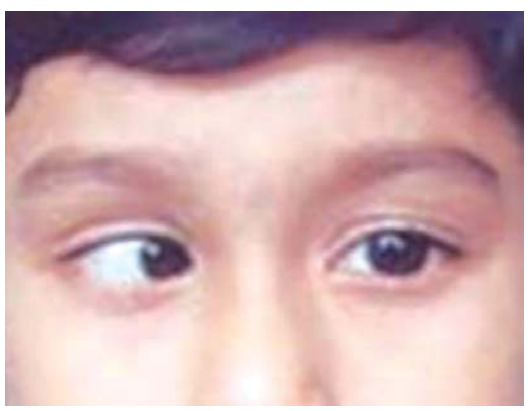


FIGURE 15.6.1: Consecutive esotropia

Divergent Concomitant Squint (exotropia)

- One eye always deviates outwards
- More likely to be latent or intermittent than esotropia
- Constant exotropia is usually seen in older patients
- Amblyopia is less common
- *Types:*
- *Congenital exotropia*
  - much less common than congenital esotropia
  - commonly seen in craniofacial anomalies like, Cruzon's syndrome **(Fig 15.7.1)**, hypertelorism **(Fig 15.7.2)**
- *Basic exotropia*
  - initially intermittent **(Fig 15.8.1)** then becomes constant **(Figs 15.8.2 to 15.8.4)**
  - angle of deviation is more for distant fixation than near
  - myopia may be a common association
- *Sensory exotropia*
  - caused by uniocular sensory visual deprivation
  - most commonly by cataract, optic atrophy, after injury, etc. **(Figs 15.9.1 to 15.9.3)**
- *Consecutive exotropia*
  - following overcorrection of a convergent squint

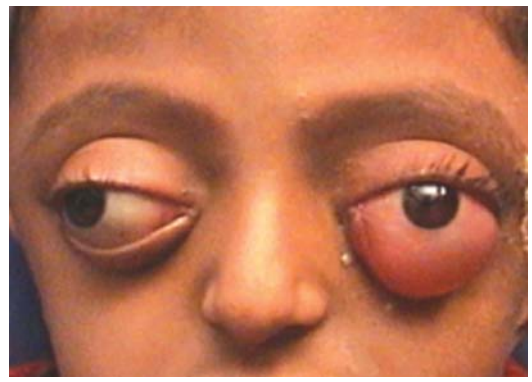


FIGURE 15.7.1: Cruzon's syndrome

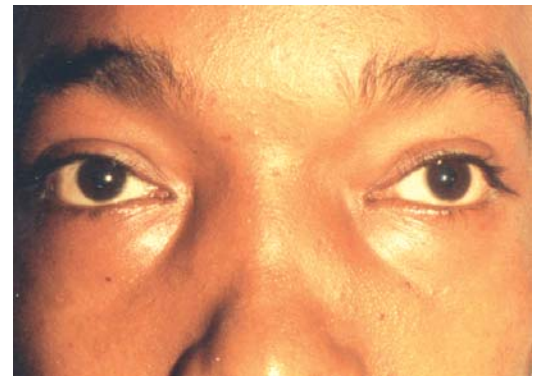


FIGURE 15.7.2: Hypertelorism

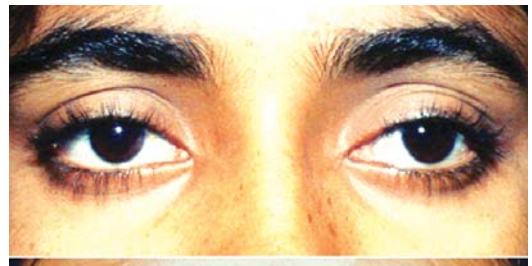


FIGURE 15.8.1: Intermittent exotropia

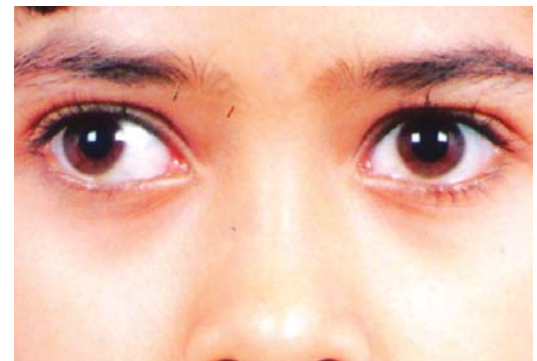


FIGURE 15.8.2: Basic exotropia

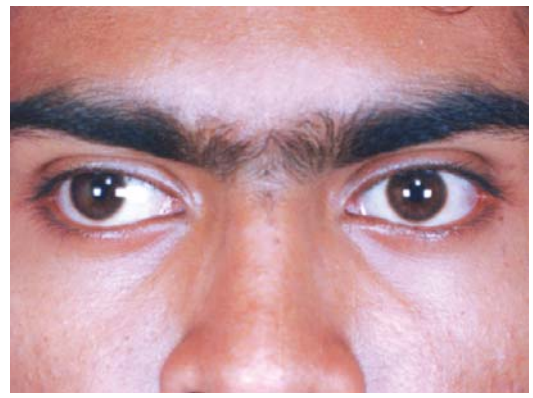


FIGURE 15.8.3: Basic exotropia

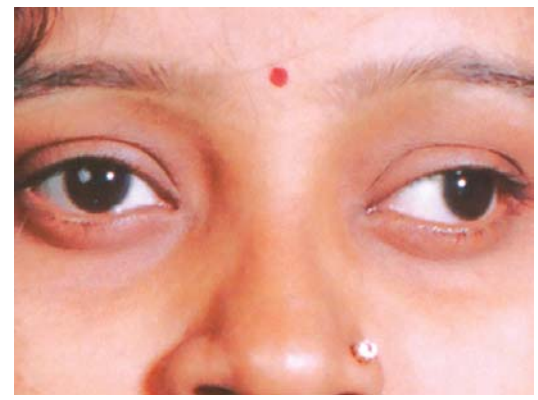


FIGURE 15.8.4: Basic exotropia

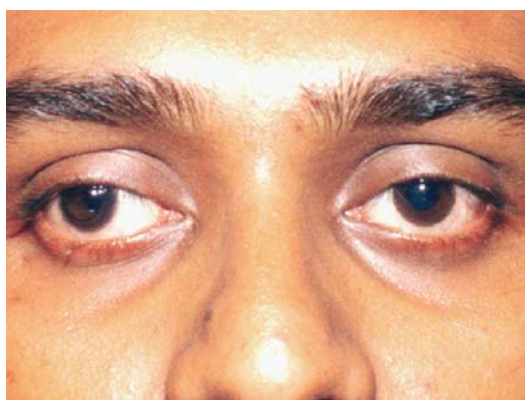


FIGURE 15.9.1: Sensory exotropia—aphakia RE

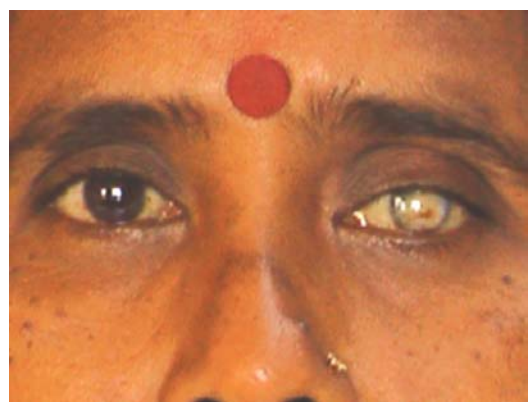


FIGURE 15.9.2: Sensory exotropia—corneal opacity LE

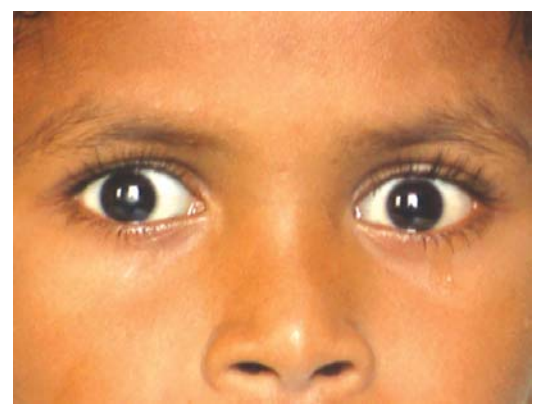


FIGURE 15.9.3: Sensory exotropia—cataract RE

Alternating Squint

- Alternating squint means when one *eye* fixes, the other *eye* deviates
- Either of the *eyes* can adopt fixation alternately and freely
- Visual acuity remains normal or near normal in each *eye*, and refractive errors are similar in both *eyes* and usually low
- Chance of amblyopia is least
- Alternating squint may be convergent **(Figs 15.10.1 and 15.10.2)** or divergent type **(Fig 15.10.3)**

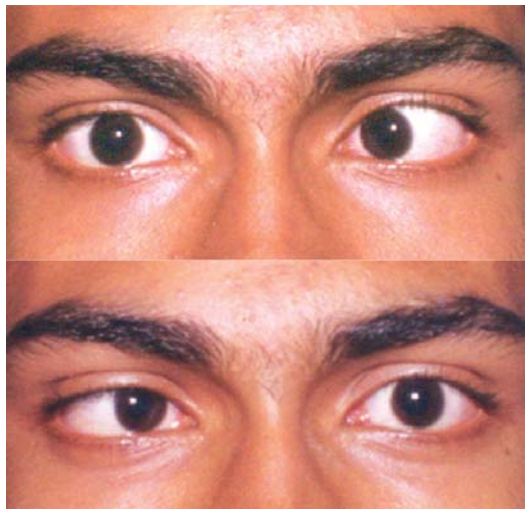


FIGURE 15.10.1: Alternate esotropia

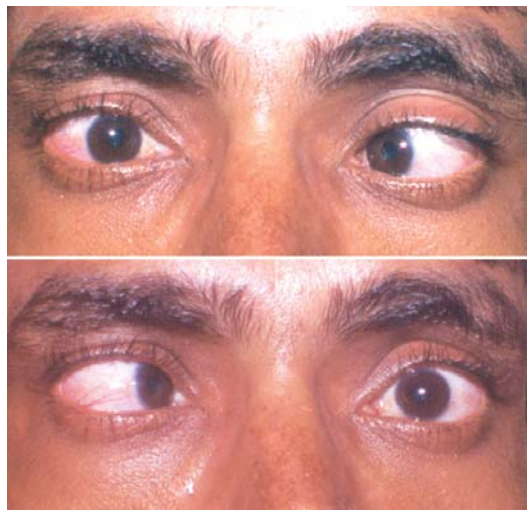


FIGURE 15.10.2: Alternate esotropia

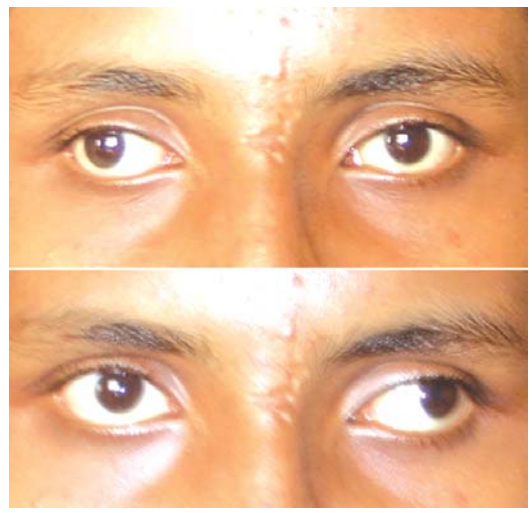


FIGURE 15.10.3: Alternate exotropia

Vertical Squint

- *Hypertropia*
  - hyperdeviation of non-fixing *eye* in primary position
  - may be primary caused by compromised ipsilateral depressors **(Fig 15.11.1)**
  - or secondary to compromised contra-lateral elevators with fixing paretic *eye* **(Fig 15.11.2)**
- *Hypotropia*
  - hypodeviation of non-fixing *eye* in primary gaze
  - may be primary or secondary, depending upon wheather non-paretic or paretic *eye* is fixing **(Figs 15.11.3 and 15.11.4)**

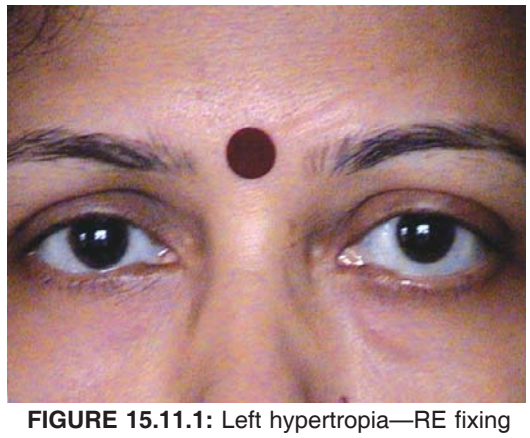


FIGURE 15.11.1: Left hypertropia—RE fixing

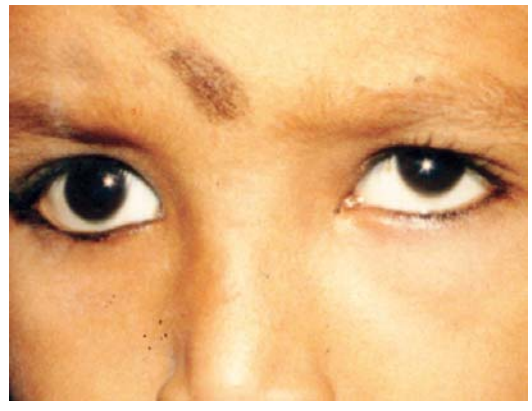


FIGURE 15.11.2: Left hypertropia—RE fixing

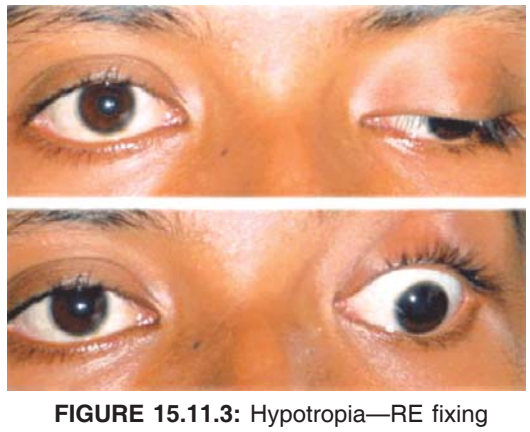


FIGURE 15.11.3: Hypotropia—RE fixing

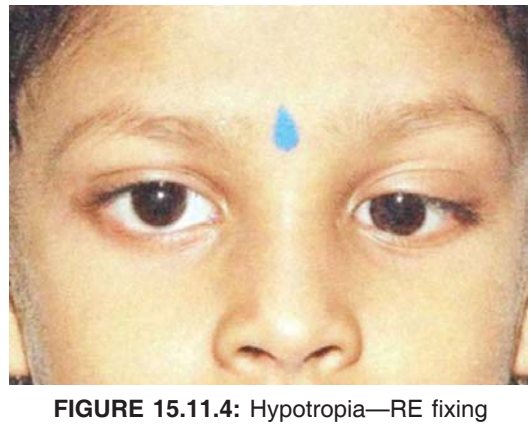


FIGURE 15.11.4: Hypotropia—RE fixing

Inferior Oblique Overaction

- Inferior oblique overaction may be unilateral or bilateral
- Primary overaction are frequently bilateral and cause upshooting in adduction **(Fig 15.12.1)**
- Associated with V-phenomenon in esodeviation (**Fig 15.12.2**)

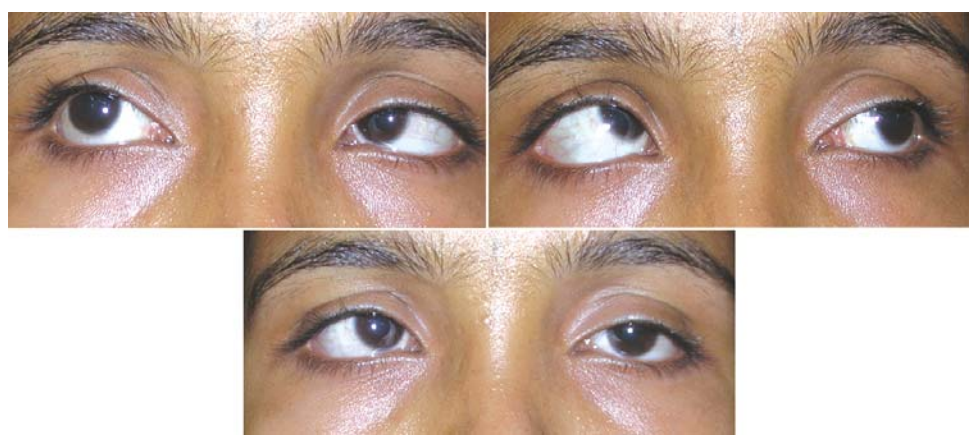


FIGURE 15.12.1: Bilateral inferior oblique overaction

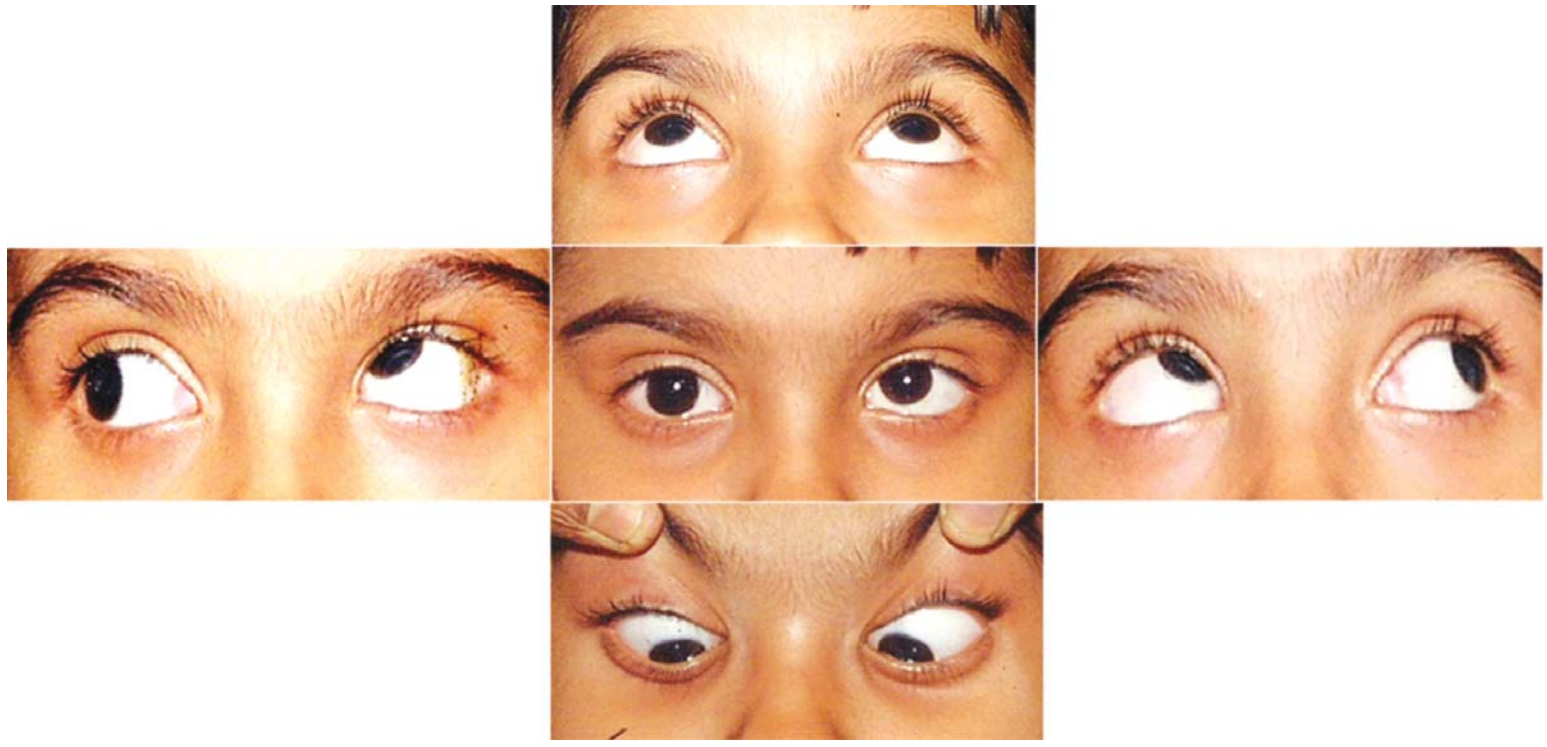


FIGURE 15.12.2: Bilateral inferior oblique overaction—V-phenomenon

PARALYTIC SQUINT

- Misalignment of the visual axes as a result of paresis, or paralysis of one or more extraocular muscles **(Fig 15.13.1)**
- Impaired movement in the field of action of muscles
- Angle of deviation varies in different direction of gaze and with fixation of eyes
- Secondary angle of deviation is more than primary deviation
- *Compensatory head posture:* in paralytic squint, to neutralize diplopia the chin may be elevated or depressed, the face is turned to right or left side, and the head may be tilted to the right or left shoulder *(ocular torticollis)* **(Fig 15.13.2)**
  - in paralysis of horizontal rectus muscle, the face is turned to field of action of the paralyzed muscle
  - in case of cyclo-vertical muscle palsy, it is more complicated and less valuable diagnostically
- Visual acuity remains normal in both eyes, without amblyopia
- *Treatment:* to give relief from diplopia, investigations to find out the cause and observation

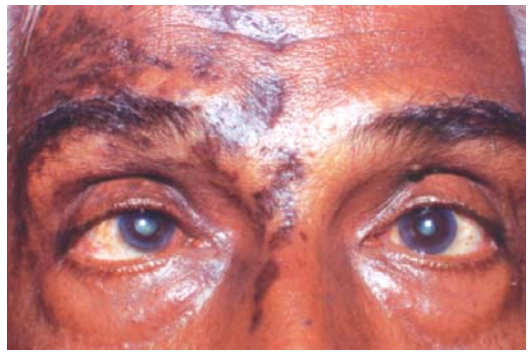


FIGURE 15.13.1: Right lateral rectus palsy—HZV ophthalmicus

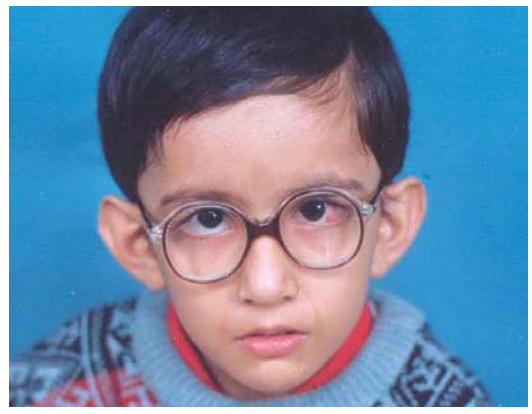


FIGURE 15.13.2: Compensatory head posture

Total Ophthalmoplegia

- Means involvement of both extrinsic and intrinsic muscles of the eyeball
- Lesion is in the cavernous sinus or in the superior orbital fissure and in bilateral cases; the lesion is widespread in the brainstem due to vascular or inflammatory cause
- *Clinical signs:* **(Fig 15.14.1)**
  - ptosis
  - eyeball is slightly proptosed and divergent
  - no movement of the eyeball
  - fixed dilated pupil
  - total loss of accommodation

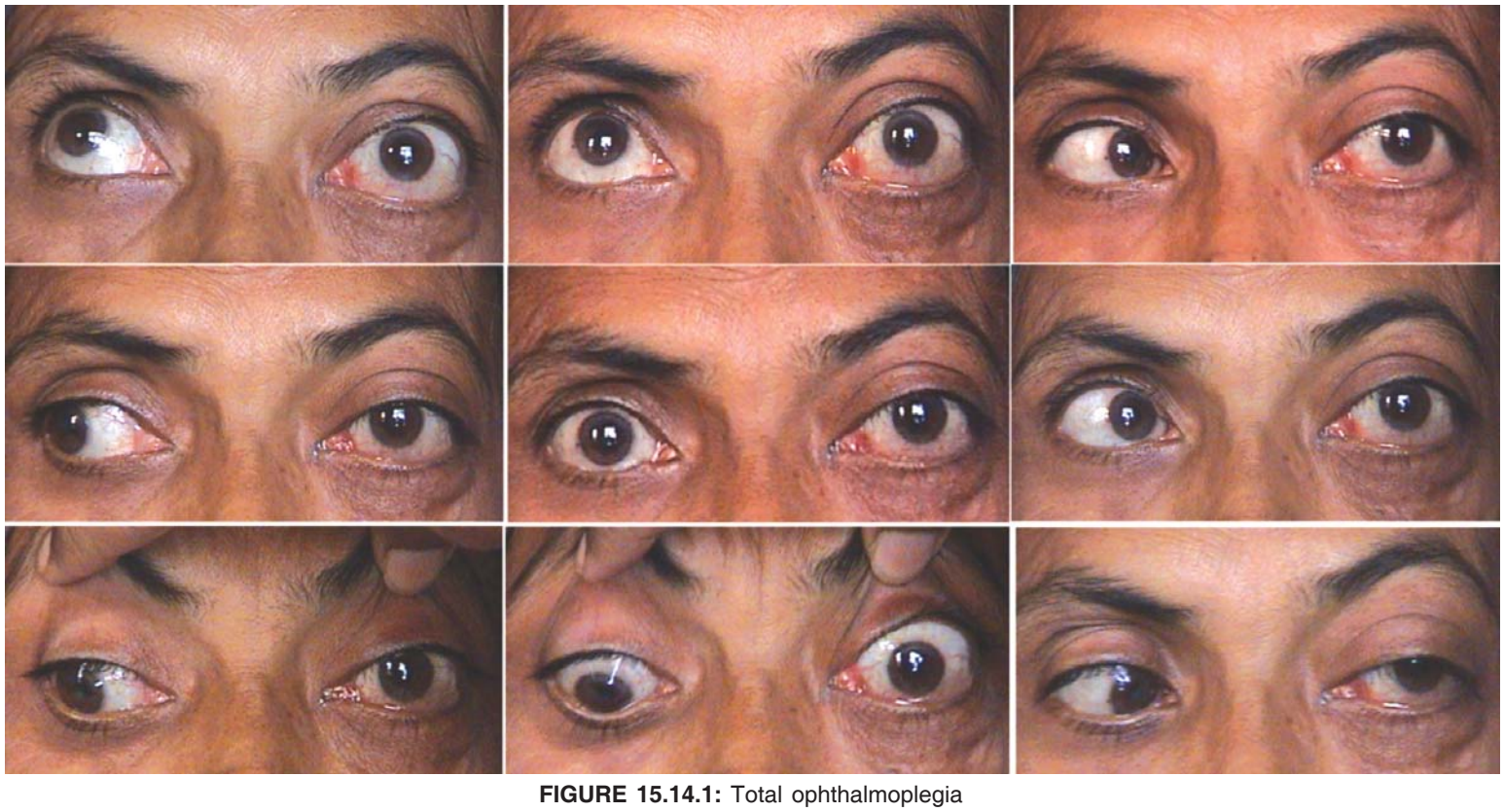


FIGURE 15.14.1: Total ophthalmoplegia

External Ophthalmoplegia

- Extrinsic muscles along with levator palpebrae superioris
- Lesion without affecting the Edinger-Westphal nucleus which supplies the intrinsic muscles **(Figs 15.15.1 and 15.15.2)**
- *Other causes:* chronic progressive external ophthalmoplegia (CPEO) **(Fig 15.15.3)**, myasthenia gravis **(Figs 15.15.4 and 15.15.5)**
- Pupillary reactions and accommodation are normal

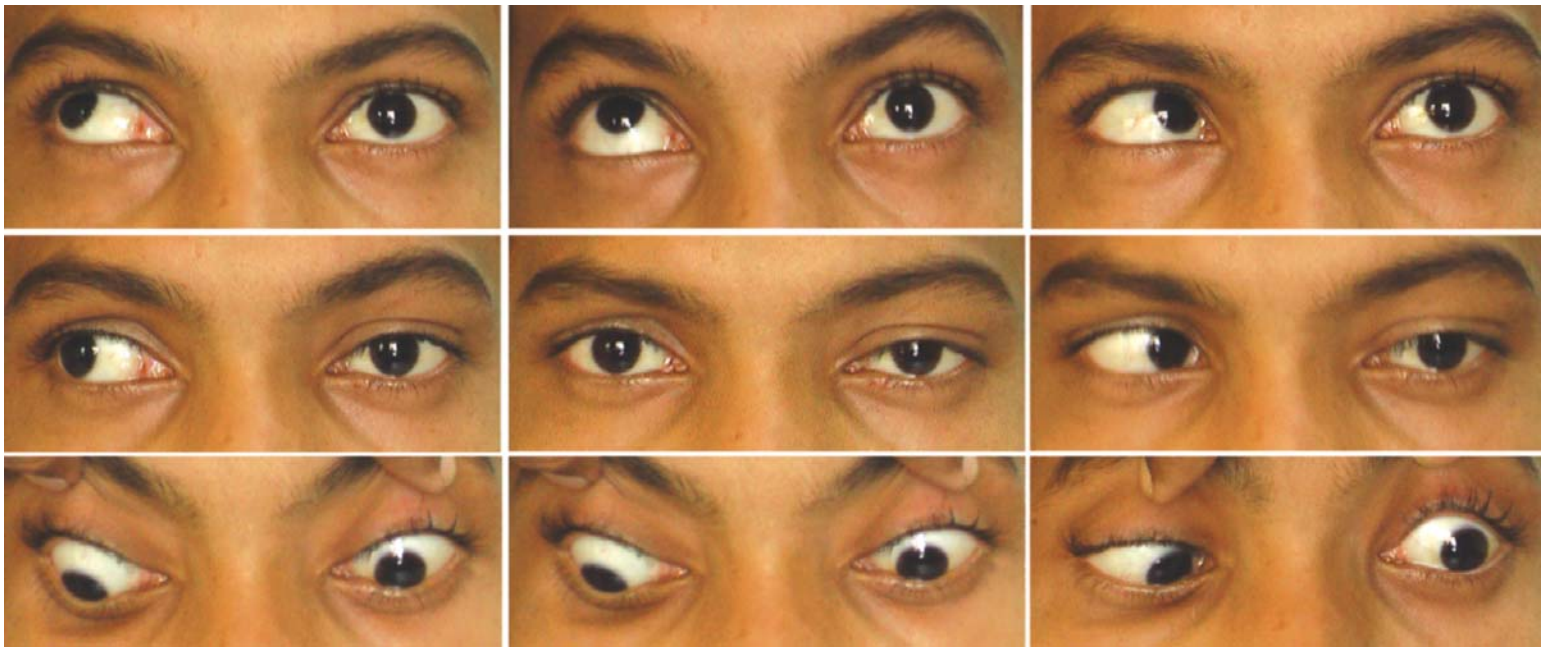


FIGURE 15.15.1: External ophthalmoplegia

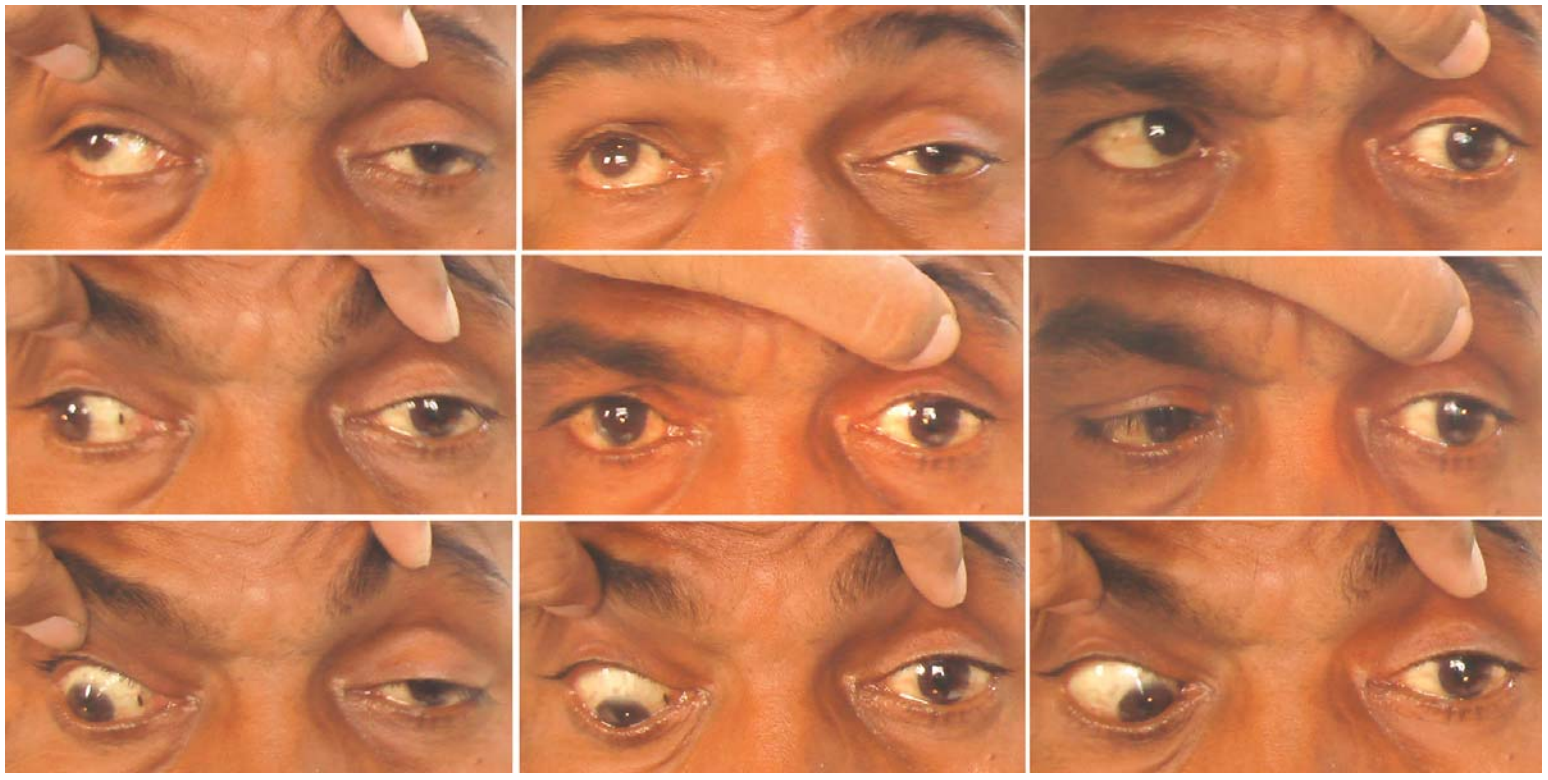


FIGURE 15.15.2: External ophthalmoplegia

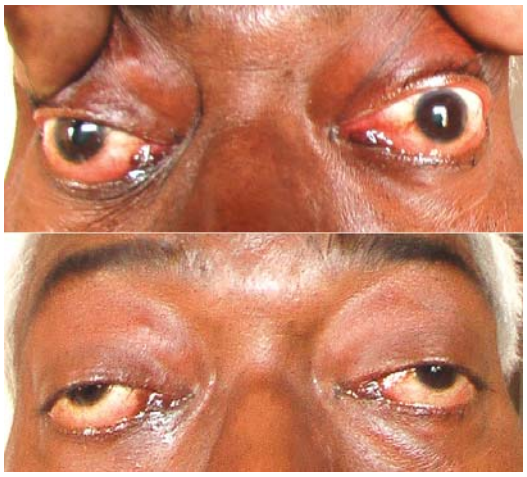


FIGURE 15.15.3: External ophthalmoplegia—CPEO

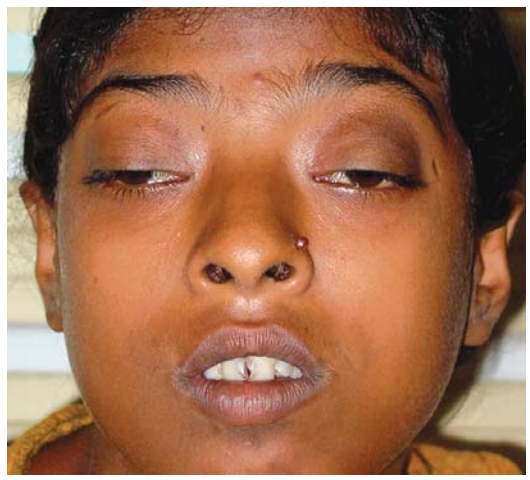


FIGURE 15.15.4: External ophthalmoplegia—myasthenia gravis

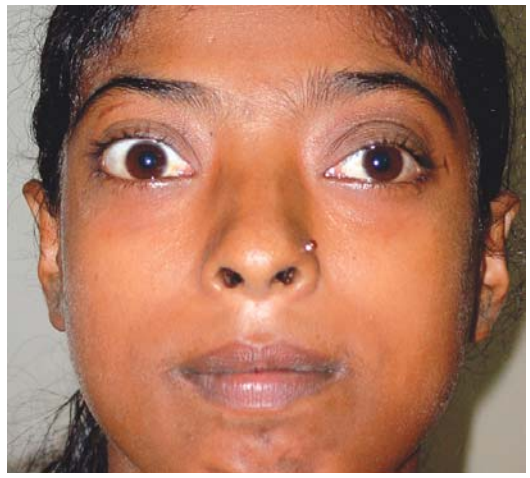


FIGURE 15.15.5: Same patient after Tensilon test

Third (Oculomotor) Nerve Palsy

- Ptosis
- Eyeball rotates outwards (divergent) and slightly downwards **(Fig 15.16.1)**
- Intorsion of the eyeball on attempted downgaze
- Ocular movements are restricted in all gaze except lalerally
- Pupil is dilated and fixed

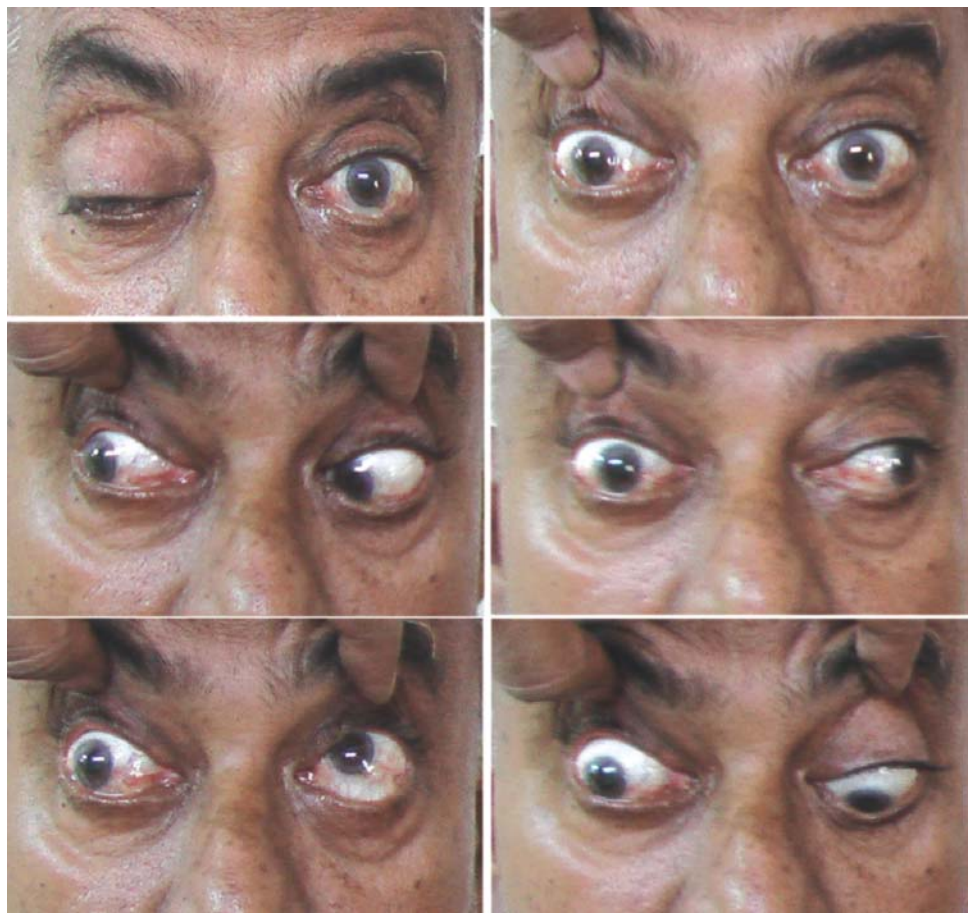


FIGURE 15.16.1: Right third nerve palsy

Fourth (Trochlear) Nerve Palsy

- Trauma is the most common cause of isolated fourth nerve palsy
- Abnormal head posture
- Eyeball deviated upwards and inwards (ipsilateral hypertropia)
- Extorsion of eyeball
- Restriction of the ocular movements on downwards and inwards
- Bielschowsky's head tilt test is useful to diagnose a fourth nerve palsy **(Figs 15.17.1 and 15.17.2)**

FIGURE 15.17.1: Left superior oblique palsy

FIGURE 15.17.2: Left superior oblique palsy

Sixth (Abducens) Nerve Palsy

- This is the most common type, and commonly occurs in raised intracranial tension
- Sixth nerve palsy may be responsible for false localizing sign
- The eyeball is rotated inwards (convergent squint) **(Figs 15.18.1 and 15.18.2)**
- Defective abduction of the eye
- Face turns towards the field of action of paralyzed muscle

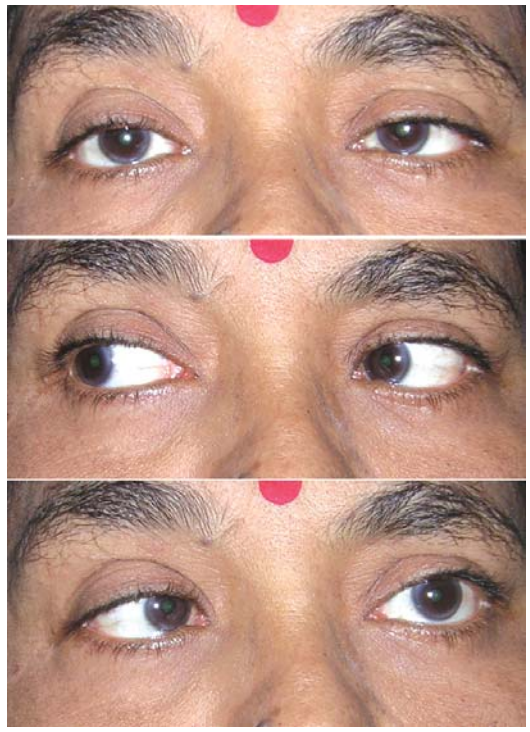


FIGURE 15.18.1: Left lateral rectus palsy

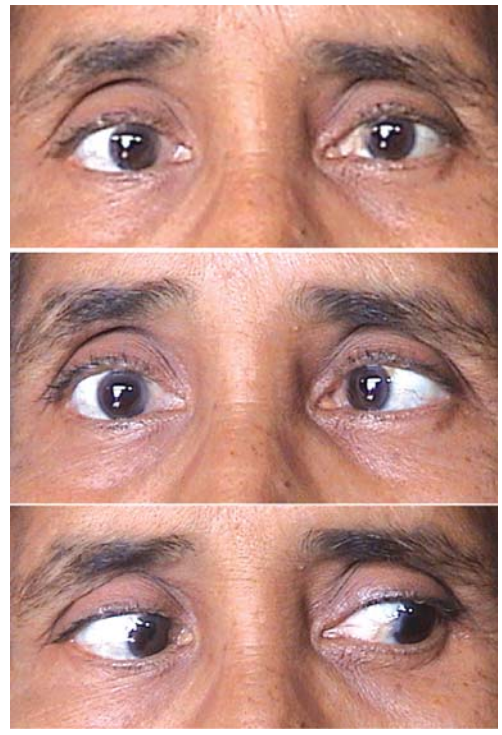


FIGURE 15.18.2: Right lateral rectus palsy

MISCELLANEOUS OCULAR MOVEMENT DISORDERS

Duane's Retraction Syndrome

- This type of non-comitant squint, occurs because of aberrant innervation which causes contraction of lateral rectus muscle instead of relaxation
- Marked restriction
- Absence of abduction
- Slight restriction of the adduction
- Retraction of the globe on attempted adduction
- Palpebral aperture of adduction, and widening on attempted abduction
- 'Up-shoot' or 'down-shoot' of the eyeball on adduction
- This is Type I and most common type **(Fig 15.19.1)**
- *The other types:*
  - *Type II:* Limitation of abduction with relatively normal abduction
  - *Type III:* Limitation of the both abduction and adduction

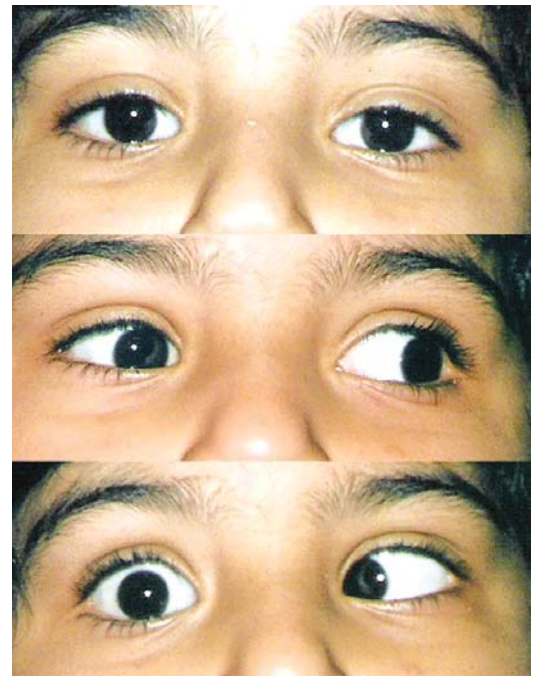


FIGURE 15.19.1: Duane's retraction syndrome

Superior Oblique Tendon-sheath Syndrome (Brown's syndrome)

- Usually congenital, but acquired form may occur following trauma or tenosynovitis of superior oblique tendon-trochlea apparatus
- Eyeballs are straight in primary position
- Limitation of elevation of the eye in adduction, simulating inferior oblique muscle palsy **(Fig 15.20.1)**
- Normal elevation on abduction
- No overaction of superior oblique muscle
- Positive forced duction test

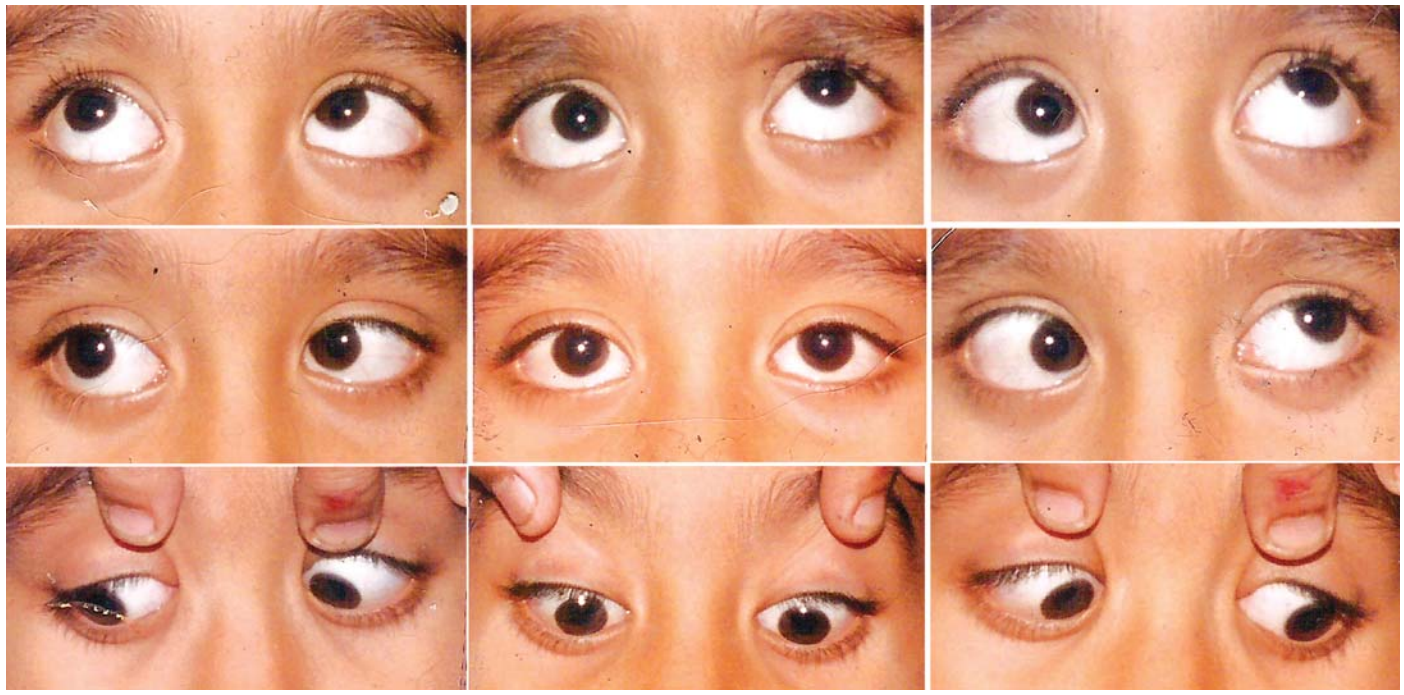


FIGURE 15.20.1: Brown's syndrome

Double Elevator Palsy

- May be congenital **(Fig 15.21.1)** or acquired **(Fig 15.21.2)**
- Eyeballs are straight in primary position
- Mild degree of ptosis may be present in some cases
- Limitation of elevation in all position of upgaze
- Normal adduction and abduction
- Normal depression in all downgaze position

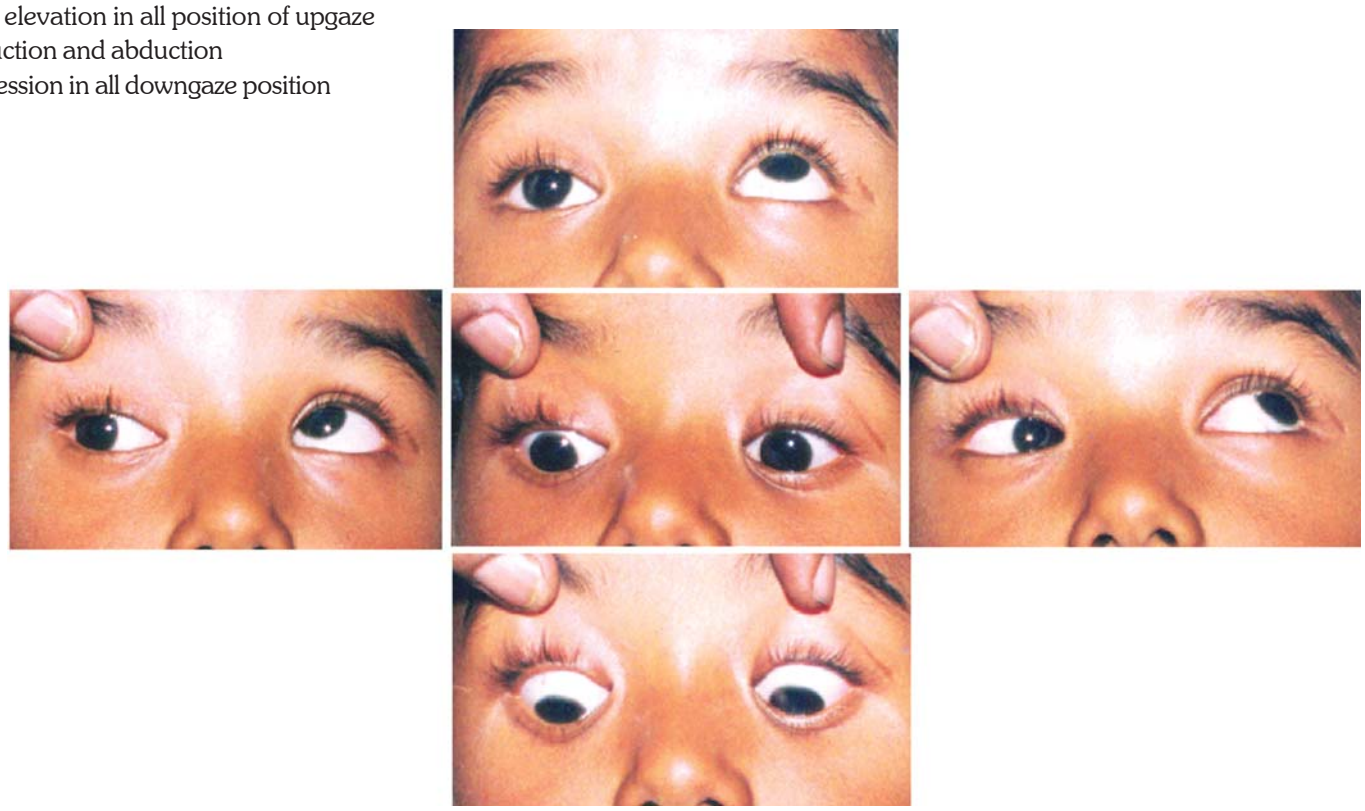


FIGURE 15.21.1: Double elevator palsy—congenital

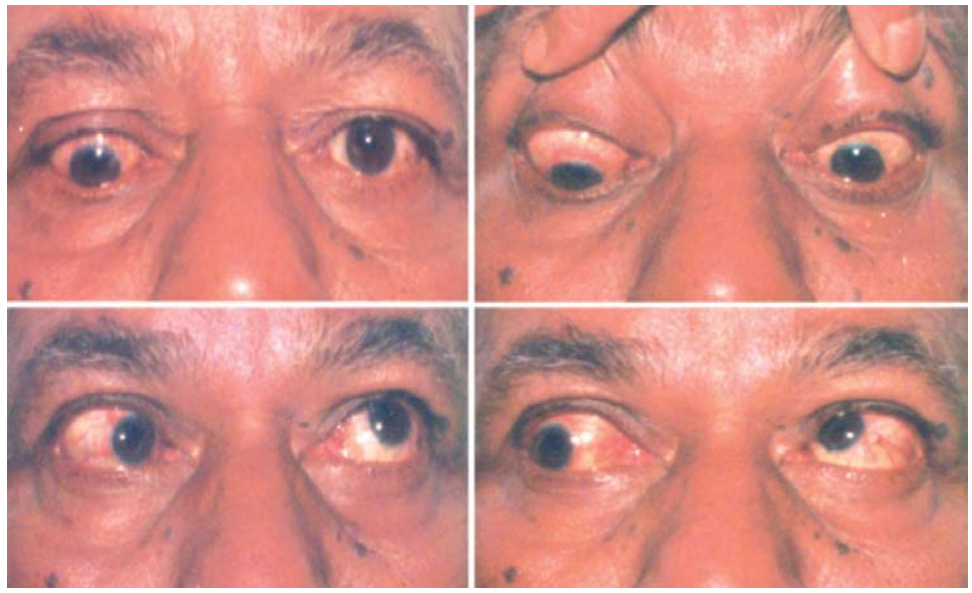


FIGURE 15.21.2: Double elevator palsy—acquired

Strabismus Fixus

- Very rare unilateral or bilateral squinting condition
- Affected eye is fixed in either convergent or divergent position due to fibrous contracture of medial or lateral rectus muscle **(Fig 15.22.1)**
- Forced duction test (FDT) is positive

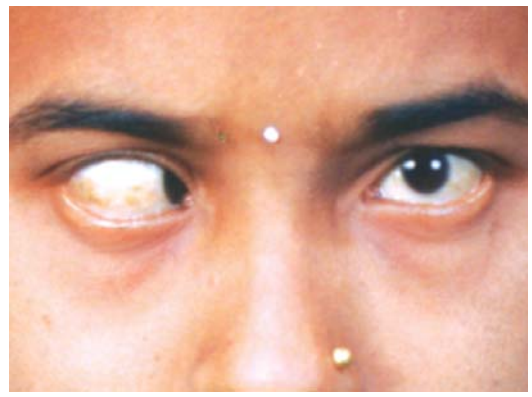


FIGURE 15.22.1: Strabismus fixus

16

Ocular Injuries

CONTUSIONS (BLUNT INJURIES)

- Eyelids
- Conjunctiva
- Cornea
- Sclera
- Anterior chamber
- Iris
- Ciliary body
- Crystalline lens
- Vitreous
- Choroid
- Retina
- Optic nerve
- Orbit

PENETRATING (PERFORATING) INJURIES

- Immediate effect of trauma
- Signs of globe perforation
- Introduction of infection
- Sympathetic ophthalmia

FOREIGN BODIES IN THE EYE

- Extraocular foreign bodies
- Corneal foreign bodies
- Conjunctival foreign body
- Intraocular foreign body (IOFB)
- Siderosis bulbi
- Chalcosis bulbi
- Miscellaneous organic materials

CHEMICAL INJURIES (BURNS)

THERMAL BURNS

MISCELLANEOUS INJURIES

- Blast injuries
- Radiational injuries

CONTUSIONS (BLUNT INJURIES)

- Ocular injuries by various blunt objects vary in severity depending upon the nature of impact
- It may be from simple subconjunctival hemorrhage to rupture of the globe
- Moreover some effects are progressive or may be delayed
- So during treatment, a guarded prognosis should be given to such injuries

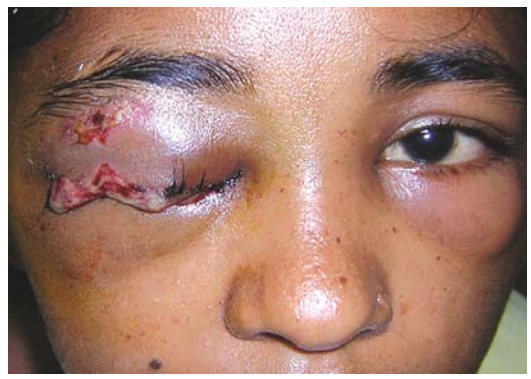


FIGURE 16.1.1: Lid laceration

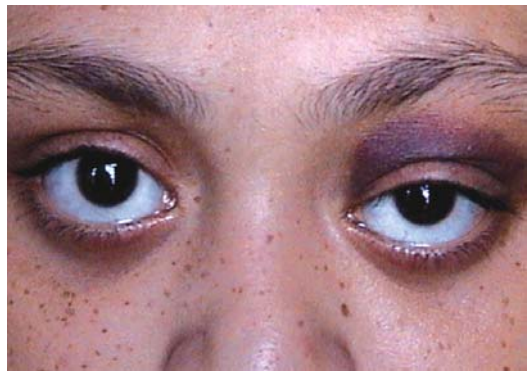


FIGURE 16.1.2: Ecchymosis of eyelids

Various effects resulting from contusions:

Eyelids

- Lid lacerations **(Fig 16.1.1)**
- Swelling and ecchymosis (black eye) of the lids **(Fig 16.1.2)** also responsible for *'Panda bear'* sign **(Fig 16.1.3)**
- Emphysema of the eyelids **(Fig 16.1.4)**

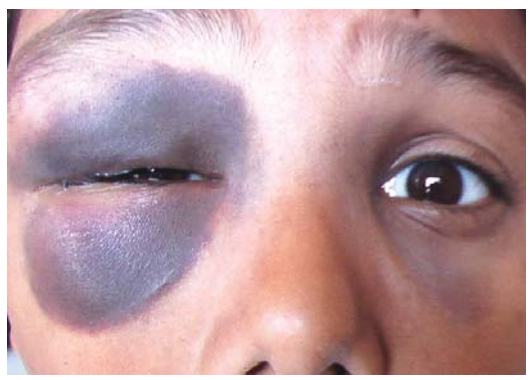


FIGURE 16.1.3: Black eye—Panda bear sign

FIGURE 16.1.4: Ecchymosis with emphysema lid

Conjunctiva

- Conjunctival lacerations and chemosis **(Fig 16.2.1)**
- Sub-conjunctival hemorrhage **(Figs 16.2.2 and 16.2.3)**

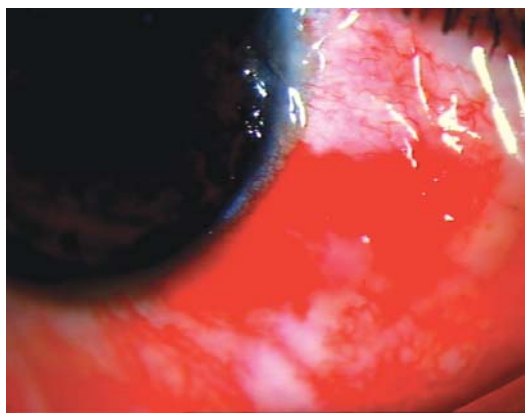


FIGURE 16.2.1: Conjunctival laceration

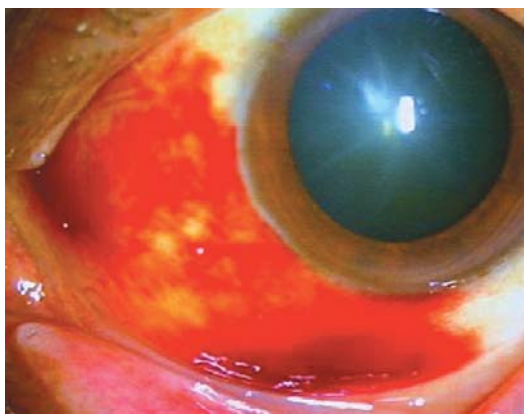


FIGURE 16.2.2: Subconjunctival hemorrhage

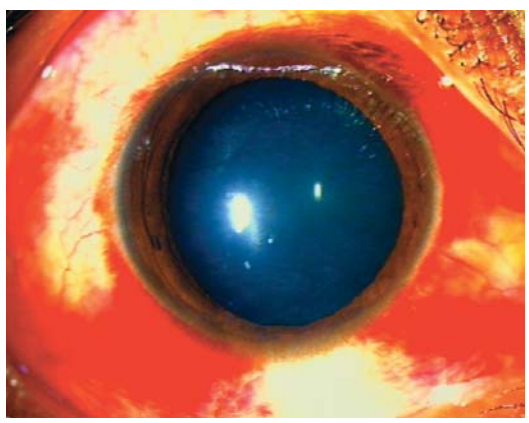


FIGURE 16.2.3: Subconjunctival hemorrhage

Cornea

- Simple abrasions **(Fig 16.3.1)**
- Rupture of the Descemet's membrane with folds and corneal edema **(Fig 16.3.2)**
- Blood staining of the cornea
  - traumatic hyphema with raised IOP
  - endothelial damage and subsequently whole cornea is reddish-brown or greenish stained **(Figs 16.3.3 and 16.3.4)**
  - cornea gradually and slowly clears from the periphery **(Fig 16.3.5)**
  - urgent paracentesis is required to prevent blood staining

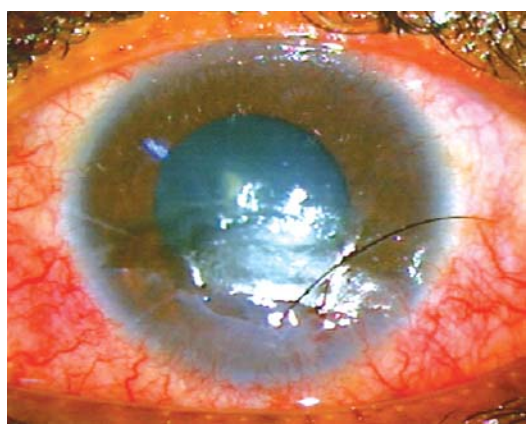


FIGURE 16.3.1: Corneal abrasion

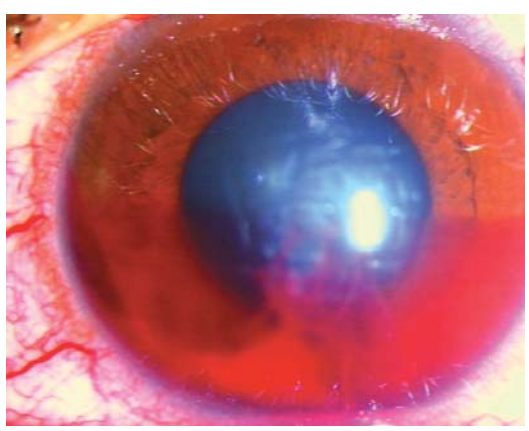


FIGURE 16.3.2: Descemet's folds and corneal edema with hyphema

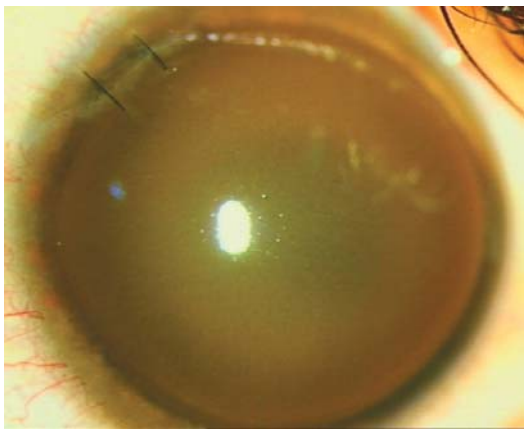


FIGURE 16.3.3: Blood staining of cornea-early

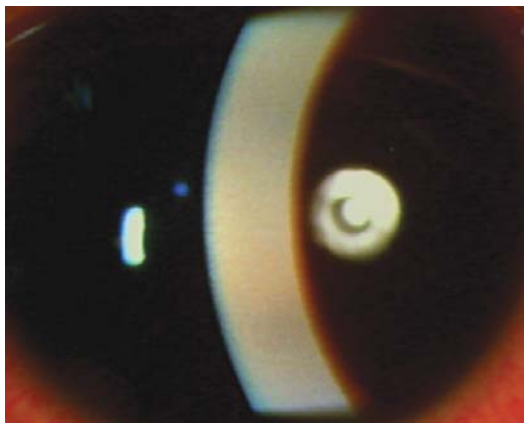


FIGURE 16.3.4: Blood staining of cornea-early

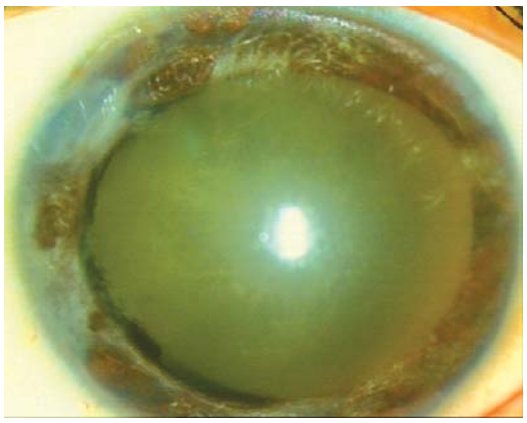


FIGURE 16.3.5: Blood staining of cornea-late

Sclera

- Scleral rupture (rupture of the globe)
  - usually near canal of Schlemm (weakest point) and runs concentrically with the limbus
  - conjunctiva is often intact
  - associated uveal prolapse **(Fig 16.4.1)** or subconjunctival dislocation of lens **(Fig 16.4.2)**
  - eyeball usually collapses with total loss of vision
  - prognosis is often very poor

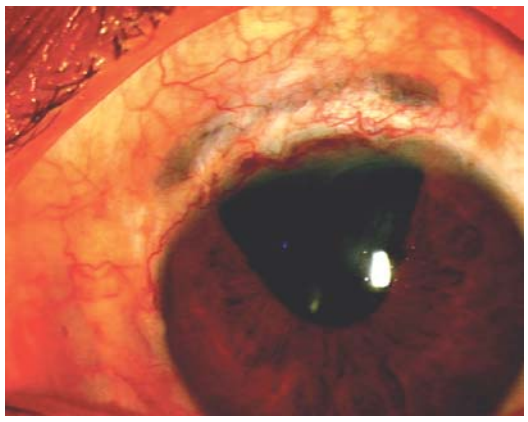


FIGURE 16.4.1: Scleral rupture-uveal prolapse

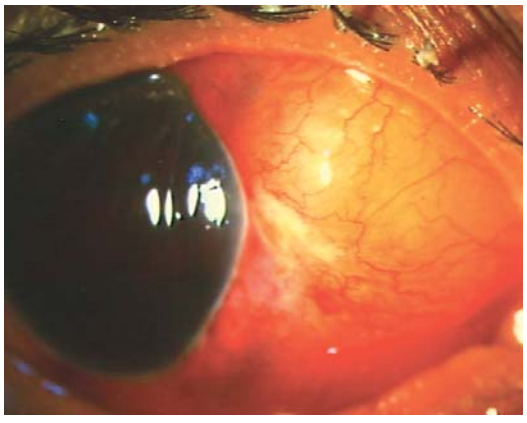


FIGURE 16.4.2: Scleral rupture-subconjunctival dislocation of crystalline lens

Anterior Chamber

- *Hyphema* (blood in the anterior chamber)
  - blood usually does not get clotted **(Figs 16.5.1 and 16.5.2)**
  - recurrent and more severe bleeding may occur within 24-72 hours
  - when the blood gets clotted, the hyphema appears as small black ball, called "8 ball hyphema" (like No.'8' ball in billiards game) **(Figs 16.5.3 and 16.5.4)**
  - associated secondary glaucoma
  - *treatment:* conservative with systemic and topical steroids, and antiglaucoma medication; followed by paracentesis if necessary

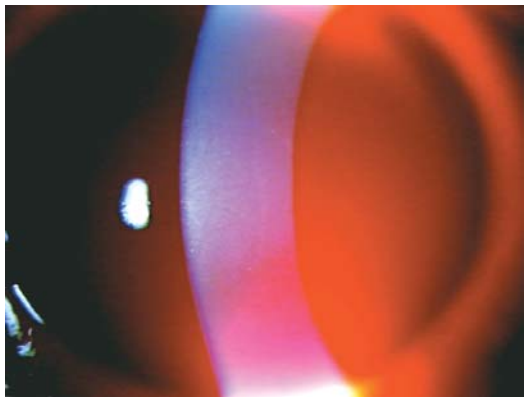


FIGURE 16.5.1: Traumatic hyphema

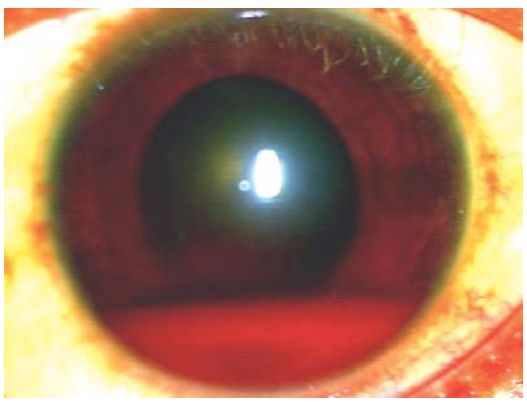


FIGURE 16.5.2: Traumatic hyphema-blood level

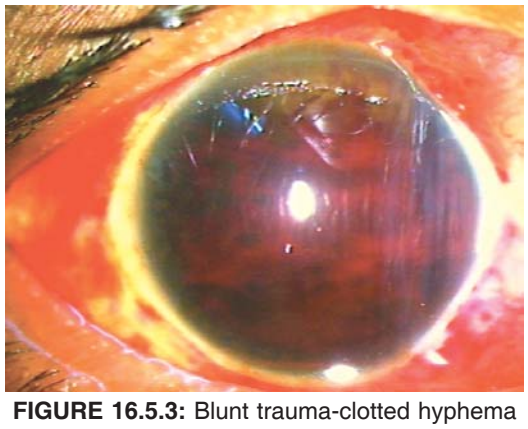


FIGURE 16.5.3: Blunt trauma-clotted hyphema

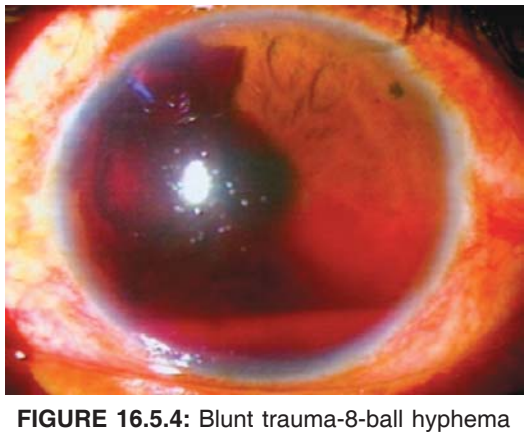


FIGURE 16.5.4: Blunt trauma-8-ball hyphema

Iris

- *Iridodialysis:*
  - iris is partially torn away from its ciliary attachment
  - biconvex area is seen at the periphery, causing a D-shaped pupil **(Fig 16.6.1)**
  - a fundal glow is obtained through the peripheral gap
- *Traumatic aniridia:* partial **(Fig 16.6.2)** or total **(Fig 16.6.3)**
- Anteflexion and retroflexion of the iris
- Traumatic iridocyclitis—usually mild
- Traumatic mydriasis **(Fig 16.6.4)**
- Post-traumatic iris atropy **(Fig 16.6.5)**

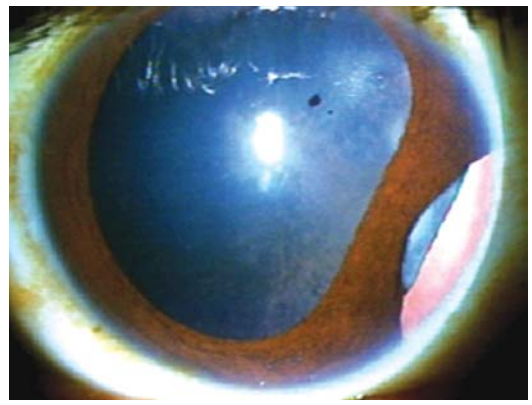


FIGURE 16.6.1: Iridodialysis-D-shaped pupil

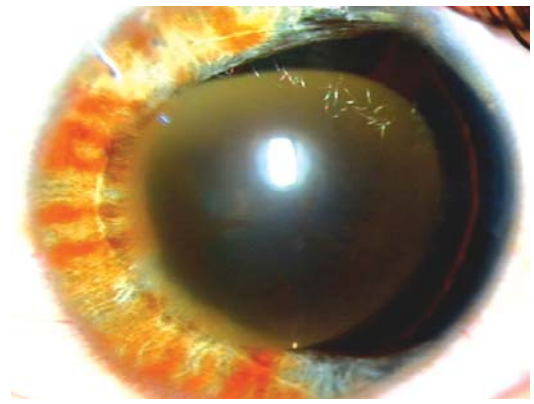


FIGURE 16.6.2: Traumatic aniridia-partial

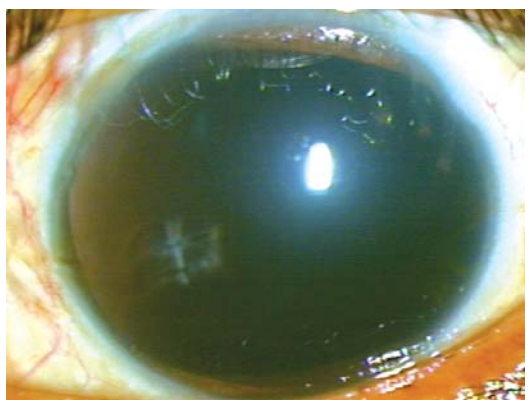


FIGURE 16.6.3: Traumatic aniridia-total

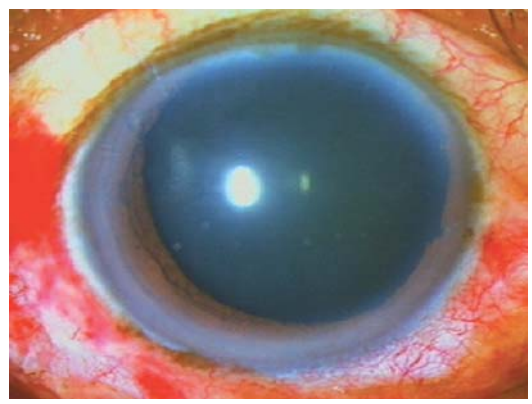


FIGURE 16.6.4: Traumatic mydriasis with iritis

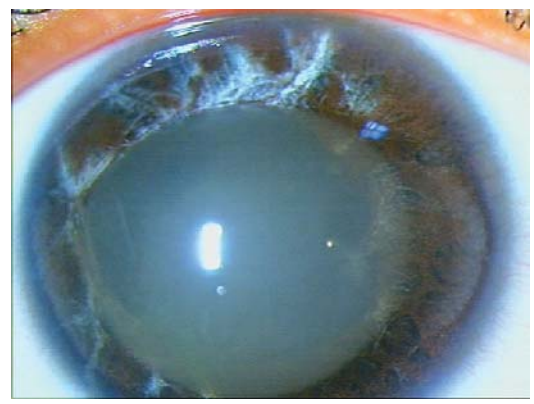


FIGURE 16.6.5: Post-traumatic iris atrophy

Ciliary Body

- *Angle recession:*
  - longitudinal tear in the face of the ciliary body
  - splitting of the circular fibers from the longitudinal fibers
  - deepening of the anterior chamber **(Fig 16.7.1)**
  - widening of the ciliary band on gonioscopy **(Fig 16.7.2)**

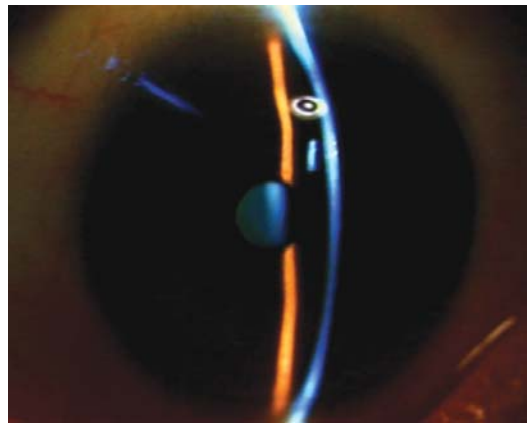


FIGURE 16.7.1: Angle recession—deep AC in upper part

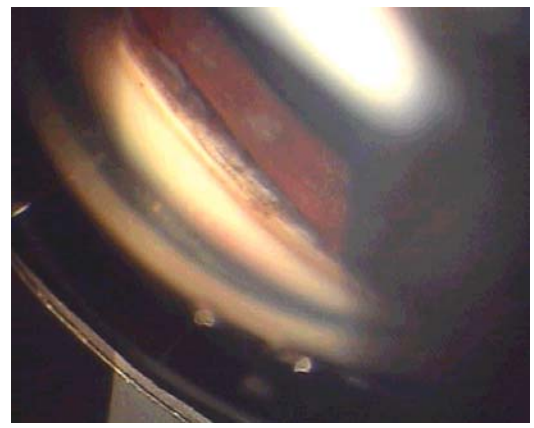


FIGURE 16.7.2: Angle recession

Crystalline Lens

- *Vossius's ring:* a circular pigmented imprint of the moised pupil on the anterior lens surface **(Fig 16.8.1)**
- *Concussion cataract:* imbibitions of aqueous through the damaged capsule, and partly due to direct mechanical effect on the lens fibers.
  - *localized cataract* often behind the iris **(Fig 16.8.2)**
  - *total cataract* in case of capsular tear **(Fig 16.8.3)**
- *Early rosette cataract:* Star-shaped cataract, usually in the posterior cortex
  - the leaves of the feathery opacities are formed by the suture acting as a vein from which the opacities radiate **(Fig 16.8.4)**

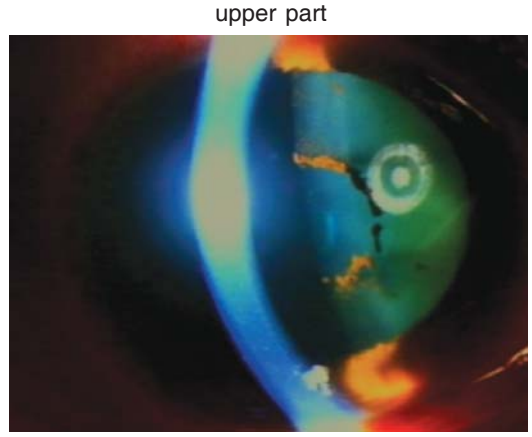


FIGURE 16.8.1: Vossius's ring

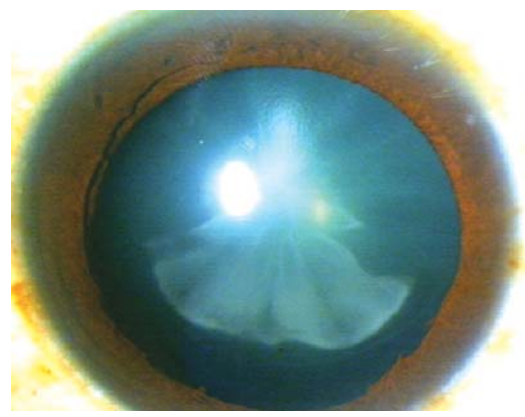


FIGURE 16.8.2: Traumatic partial cataract

- remains stationary, and sometimes, it may progress into a total cataract
- *Late rosette cataract*
  - usually develops 1-2 years after a concussion
  - smaller and more compact with short sutural extension
  - feathery opacities lie in the angle between two adjacent sutures **(Fig 16.8.5)**
- *Subluxation* of the crystalline lens or IOL **(Figs 16.8.6 and 16.8.7)**
- *Dislocation* of the lens or IOL: in the anterior chamber **(Fig 16.8.8)** or in the vitreous cavity **(Figs 16.8.9 and 16.8.10)**

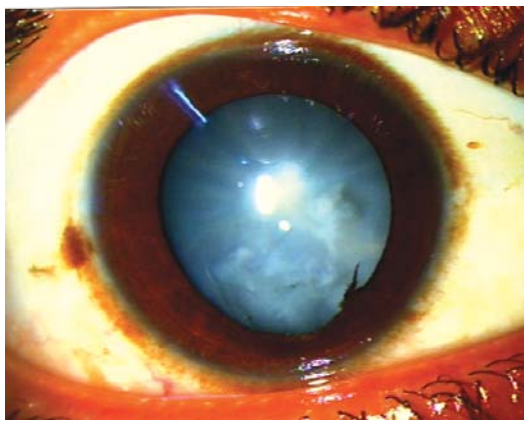


FIGURE 16.8.3: Blunt trauma-capsular rupture

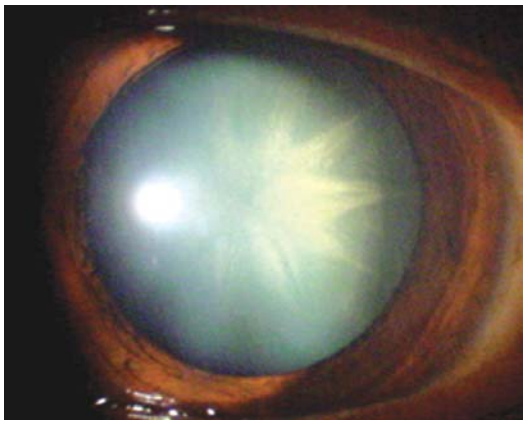


FIGURE 16.8.4: Early rosette cataract

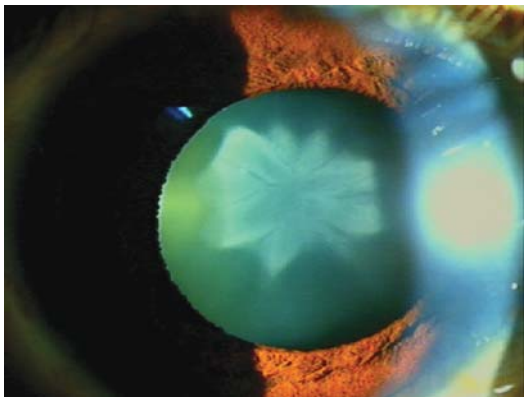


FIGURE 16.8.5: Late rosette cataract

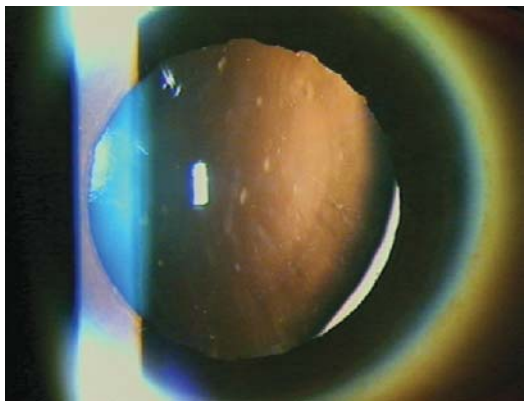


FIGURE 16.8.6: Subluxation of the lens

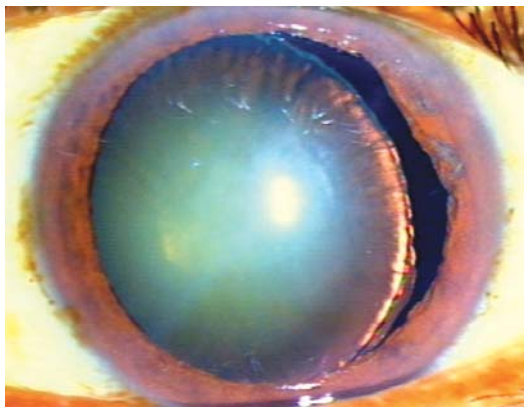


FIGURE 16.8.7: Subluxation of the lens

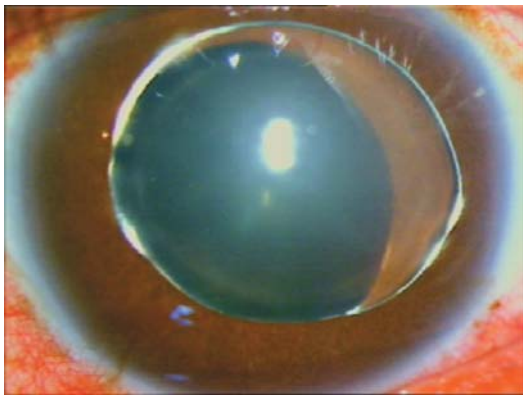


FIGURE 16.8.8: Dislocation of lens in AC

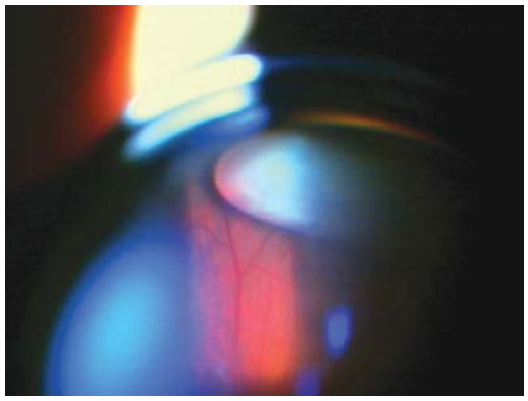


FIGURE 16.8.9: Dislocation of lens-vitreous

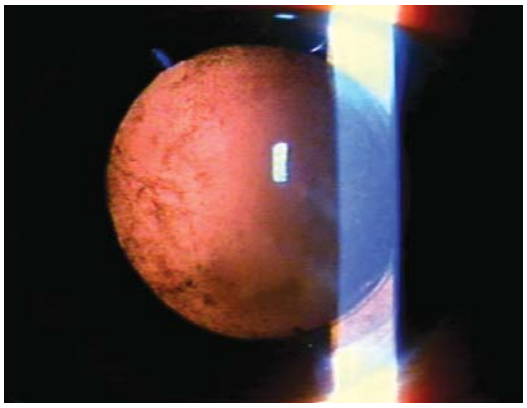


FIGURE 16.8.10: Dislocation of lens-total

Vitreous *(See Chapter 10)*

- Liquefaction of the vitreous
- Posterior vitreous detachment
- Vitreous hemorrhage with its sequelae

Choroid

- *Choroidal rupture:*
  - rupture is seen as one or two curved white lines **(Fig 16.9.1)**
  - concentric with the disc margin and on its temporal side
  - edges of this line are often pigmented **(Fig 16.9.2)**

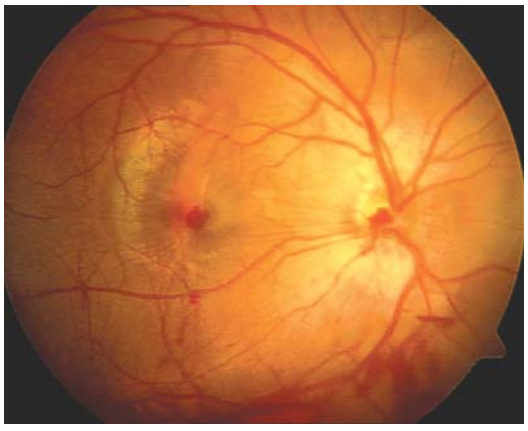


FIGURE 16.9.1: Choroidal rupture-early

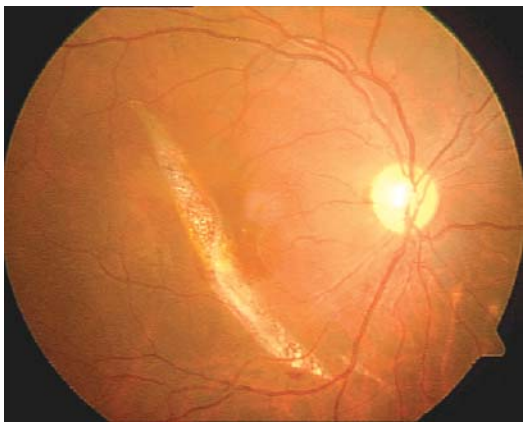


FIGURE 16.9.2: Choroidal rupture-late

Retina

- *Commotio retinae* (Berlin's edema):
  - milky white cloudiness at the macular area due to edema
  - often with a cherry red spot **(Fig 16.10.1)**
  - later there may be pigmentary deposits at the macula
- Traumatic macular hemorrhage **(Fig 16.10.2)** with the formation of a macular cyst and subsequently, a macular hole **(Fig 16.10.3)**
- Retinal or preretinal hemorrhage **(Fig 16.10.4)**
- Retinal tears at periphery, especially in myopic subjects
- Rhegmatogenous or tractional detachment **(Fig 16.10.5)** retinal detachment
- Traumatic proliferative vitreoretinopathy usually secondary to vitreous hemorrhage **(Fig 16.10.6)**

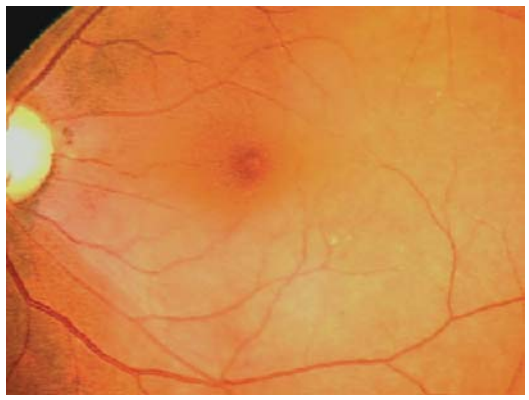


FIGURE 16.10.1: Barlin's edema-cherry red spot

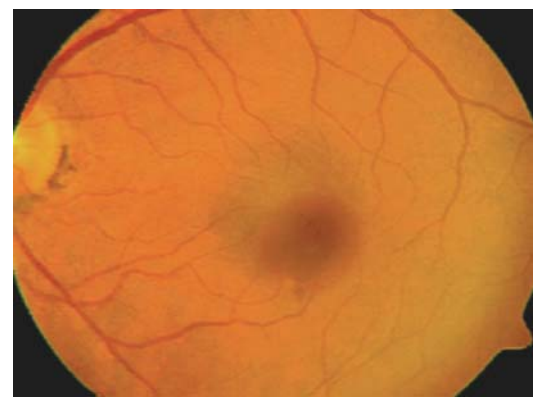


FIGURE 16.10.2: Traumatic macular hemorrhage

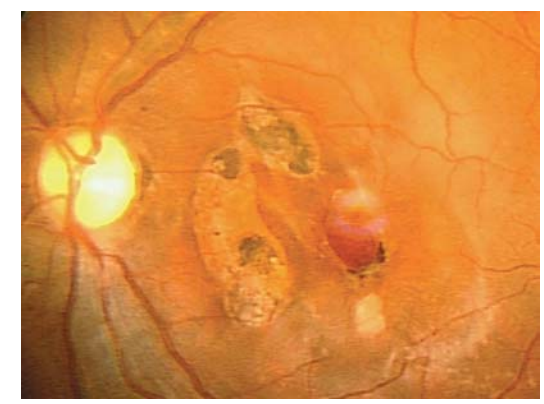


FIGURE 16.10.3: Traumatic macular hole

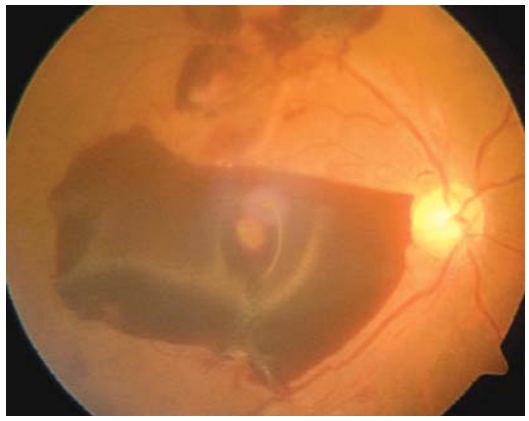


FIGURE 16.10.4: Traumatic preretinal hemorrhage

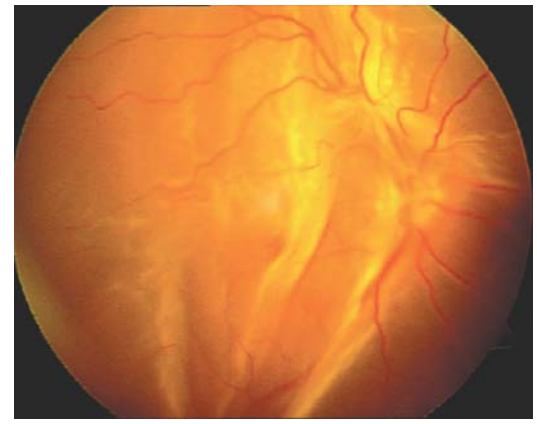


FIGURE 16.10.5: Traumatic retinal detachment

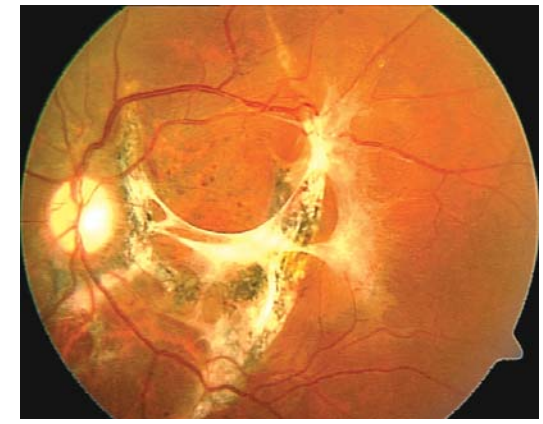


FIGURE 16.10.6: Traumatic proliferative vitreoretinopathy

Optic Nerve

- Optic nerve sheath hemorrhage **(Fig 16.11.1)**
- Peripapillary hemorrhage **(Fig 16.11.2)**
- Avulsion of the optic nerve leading to optic atrophy, which may be partial or total **(Fig 16.11.3)**

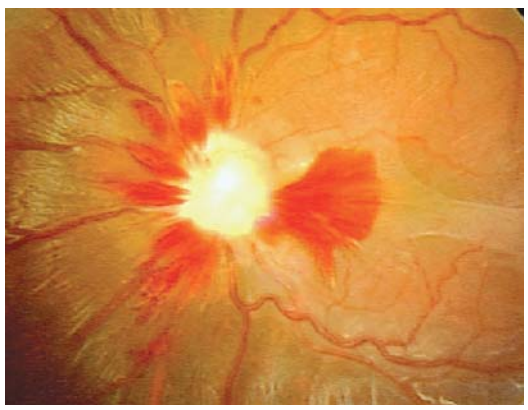


FIGURE 16.11.1: Optic nerve sheath hemorrhage

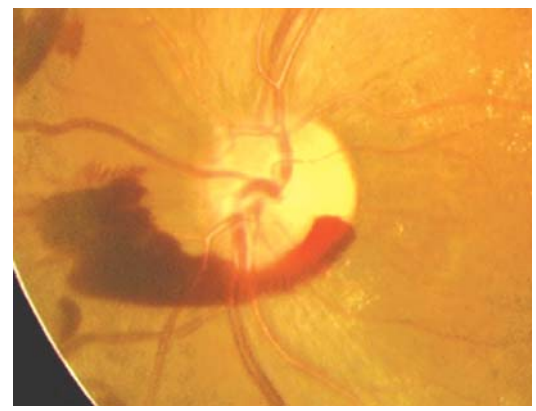


FIGURE 16.11.2: Peripapillary hemorrhage

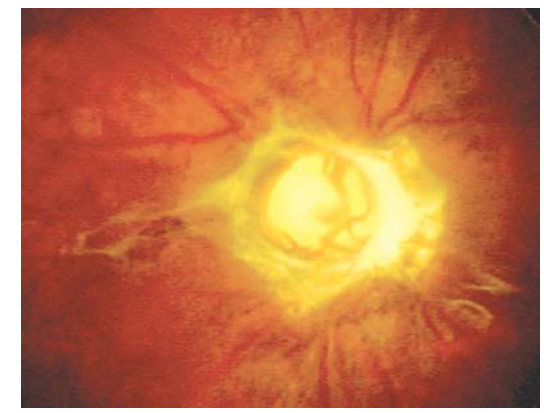


FIGURE 16.11.3: Avulsion of optic nerve with atrophy

Orbit

- *Blow-out fracture:* Pure blow-out fracture is typically caused by a sudden rise in intraorbital pressure by a striking object which is greater than 5 cm in diameter, like tennis ball or blow by a fist
  - periocular ecchymosis and edema
  - enophthalmos with pseudo-ptosis usually appears after 10-14 days **(Fig 16.12.1)**
  - infraorbital nerve anesthesia
  - nasal bleeding and subconjunctival hemorrhage
- Fractures of the orbital bones
- Retrobulbar hemorrhage **(Fig 16.12.2)**
- Orbital emphysema and proptosis

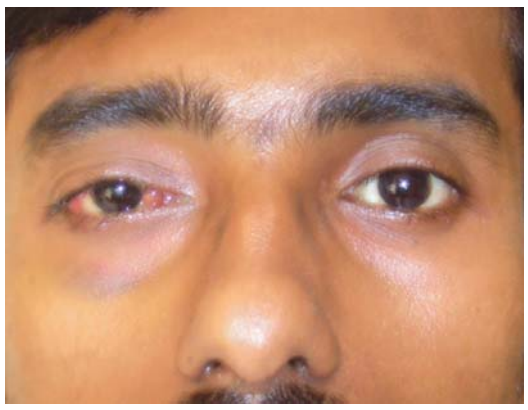


FIGURE 16.12.1: Blow out fracture—enophthalmos right eye

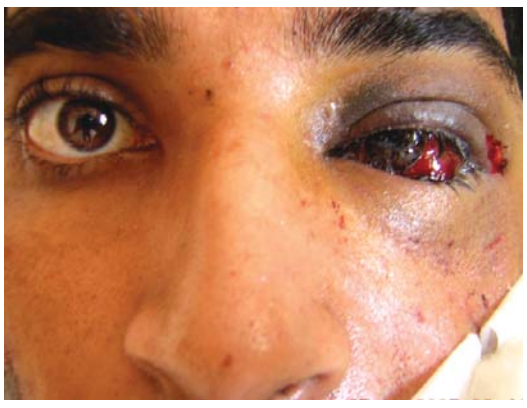


FIGURE 16.12.2: Retrobulbar hemorrhage

PENETRATING (PERFORATING) INJURIES

- They are caused by sharp objects or projectile foreign bodies
- All perforating injuries are potentially serious
- So, the patients should be urgently admitted and treated promptly
- *Seriousness arises from*:
  - immediate effect of trauma
  - introduction of infection
  - chance of sympathetic ophthalmia

Immediate Effect of Trauma

- Wounds of the lid **(Figs 16.13.1 and 16.13.2)** and conjunctiva **(Fig 16.13.3)**
- *Wounds of the cornea*
  - may be linear **(Fig 16.13.4)**, curved, triradiate **(Fig 16.13.5)** and lacerated **(Fig 16.13.6)**

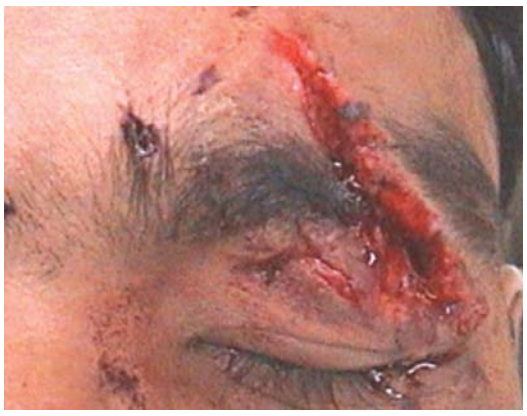


FIGURE 16.13.1: Cut injury of lid

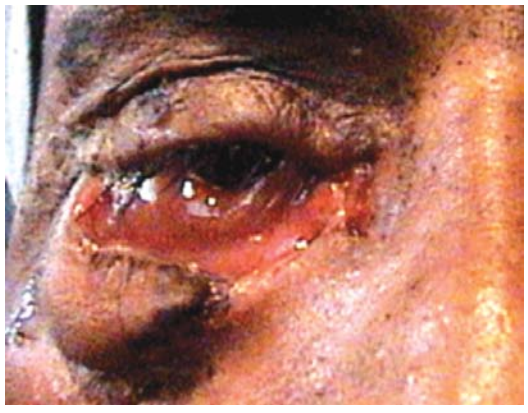


FIGURE 16.13.2: Lid injury-lower canaliculus

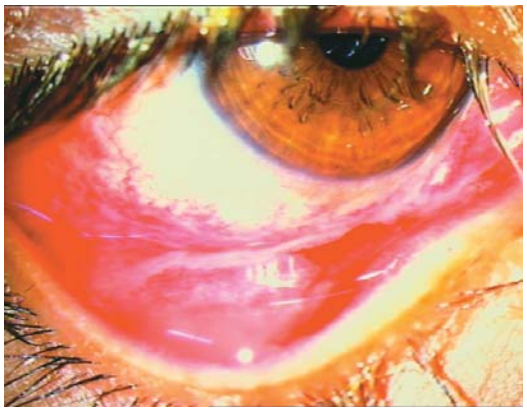


FIGURE 16.13.3: Conjunctival laceration

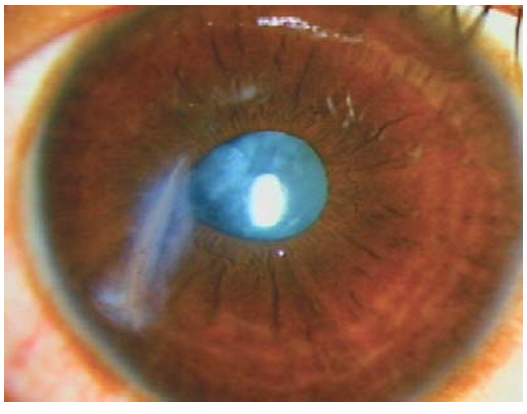


FIGURE 16.13.4: Corneal rupture

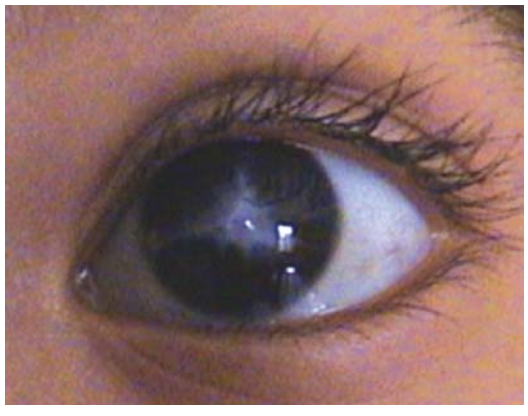


FIGURE 16.13.5: Corneal rupture-triradiate

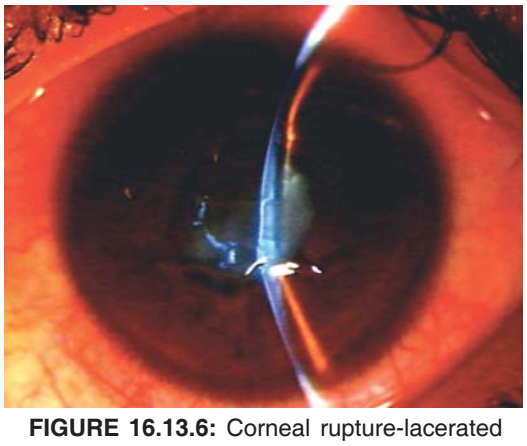


FIGURE 16.13.6: Corneal rupture-lacerated

- may be small, large or corneoscleral **(Fig 16.13.7)**
- may be at the limbus **(Fig 16.13.8)**
- may be associated with iris incarceration or frank iris prolapse
- may be lamellar **(Fig 16.13.9)**
- margins soon swell-up after the injury, and become cloudy

- *Wounds of the sclera*
  - is recognized by the uveal prolapse **(Fig 16.13.10)** and
  - may be associated with vitreous prolapse
- *Wounds of the lens and its capsule*
  - localized lens opacity
  - total lens opacity **(Fig 16.13.11)**
  - may be with early rosette cataract **(Fig 16.13.12)**
  - in case of large capsular wound, the flocculent white cortical matters protrude through the capsular opening **(Fig 16.13.13)**
  - anterior chamber is full of white flocculi **(Fig 16.13.14)**
- Injuries in other parts of the eye in cases of severe impact
- *Iatrogenic globe perforation by needle:* may happen during peribulbar or retrobulbar block **(Fig 16.13.15)**

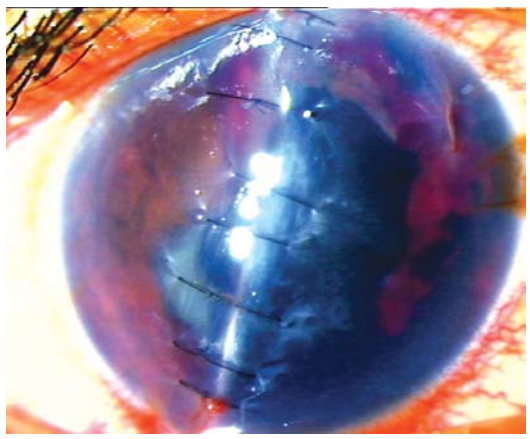


FIGURE 16.13.7: Sclerocorneal rupture

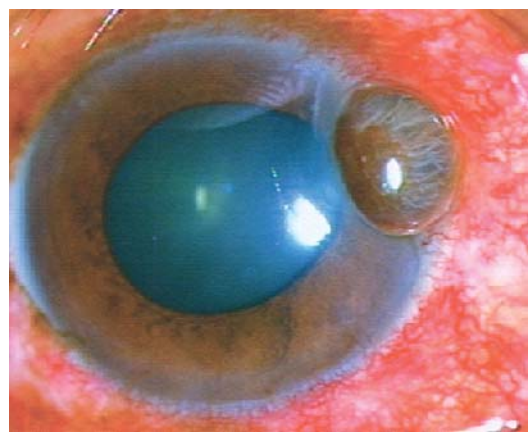


FIGURE 16.13.8: Limbal rupture with iris prolapse

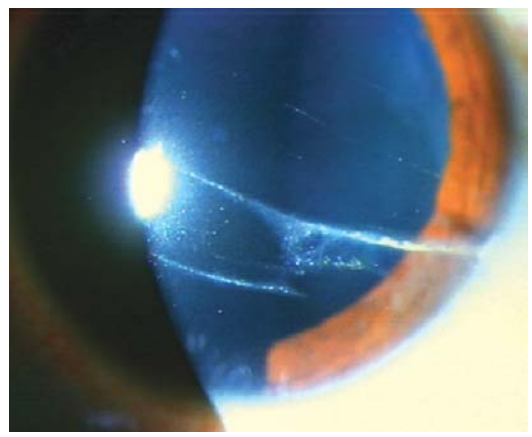


FIGURE 16.13.9: Lamellar injury of cornea

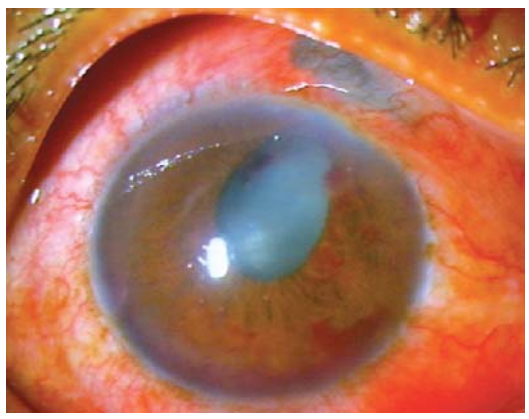


FIGURE 16.13.10: Scleral rupture-uveal prolapse

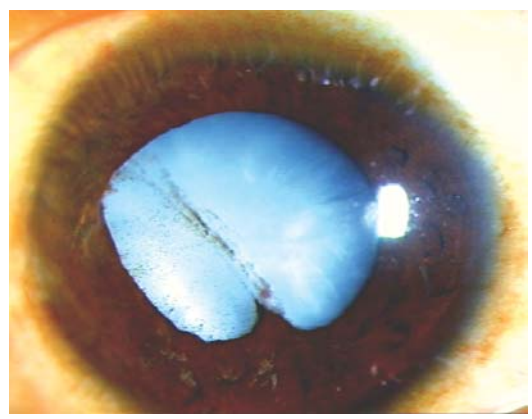


FIGURE 16.13.11: Total cataract-capsular tear

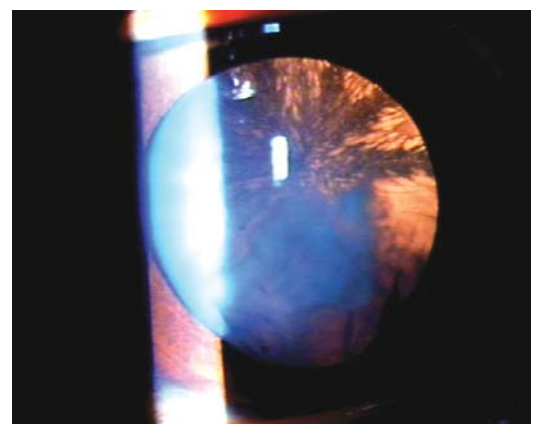


FIGURE 16.13.12: Early rosette cataract

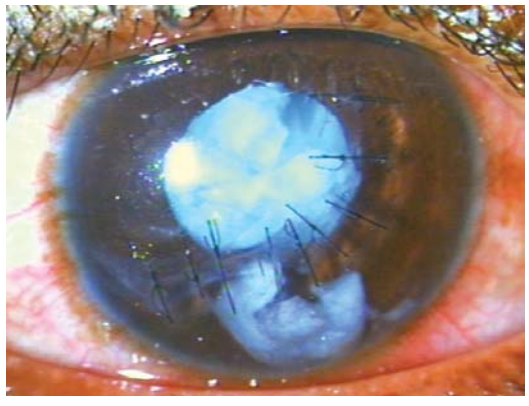


FIGURE 16.13.13: Lens matter in anterior chamber

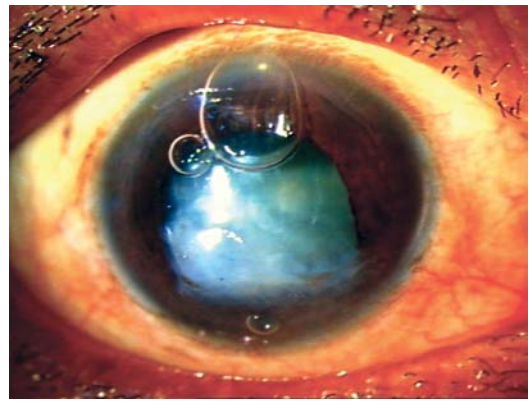


FIGURE 16.13.14: Total cataract-cortical mater in anterior chamber

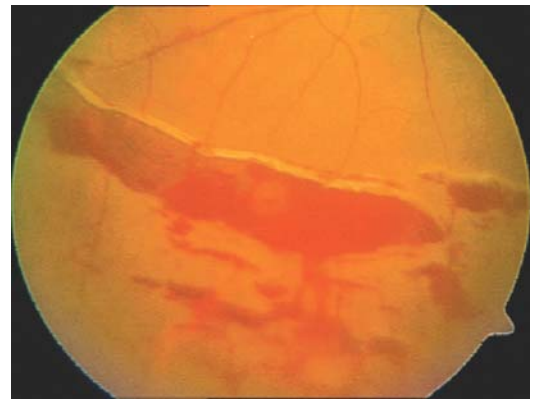


FIGURE 16.13.15: Globe puncture during peribulbar injection

Signs of Globe Perforation

- Conjunctival chemosis
- Shallow or flat anterior chamber
- Alteration of papillary size, shape and location
- Focal iris tear or hole
- Injury tract in the cornea, sclera, lens or vitreous
- Wound leak (a positive Seidel's test)
- Hypotony

Introduction of Infection

- Purulent keratitis
- Purulent iridocyclitis with hypopyon **(Fig 16.14.1)**
- Endophthalmitis **(Fig 16.14.2)**
- Panophthalmitis in extreme situation
- *Lental abscess*
  - after a sharp penetrating injury with direct inoculation of organism inside the lens
  - yellowish exudate visible within the lens **(Fig 16.14.3)**

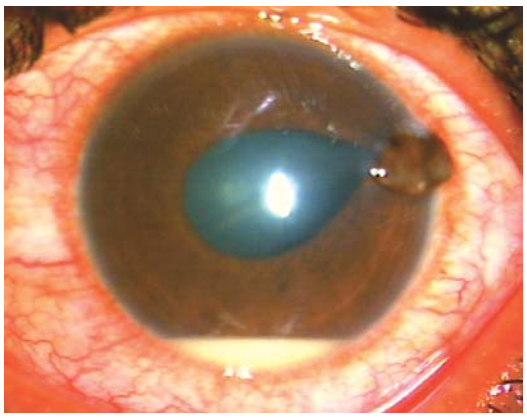


FIGURE 16.14.1: Perforating injury-purulent iritis with hypopyon

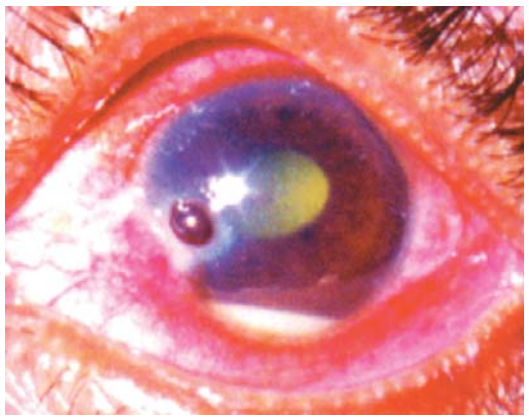


FIGURE 16.14.2: Perforating injury-endophthalmitis

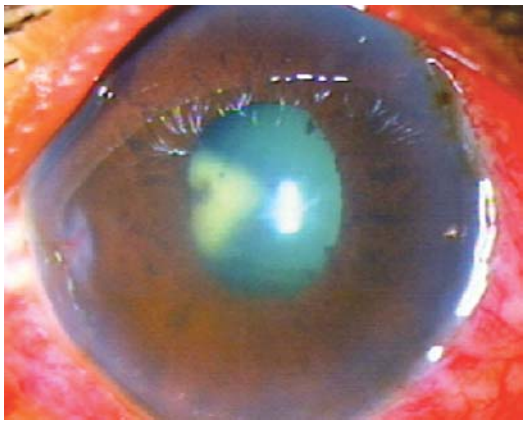


FIGURE 16.14.3: Perforating injury-lental abscess

Sympathetic Ophthalmia

Most dreadful complication of a penetrating injury—discussed in **Chapter 6**

FOREIGN BODIES IN THE EYE

Extraocular Foreign Bodies

Small foreign bodies—*e.g.* coal, dust, sand, iron particles, eyelash, wood-piece, husks of seeds, wings of insects, etc may pitch upon the conjunctiva, cornea or the limbus

Conjunctival Foreign Body

- *Commonest site:* at the middle of the upper subtarsal sulcus **(Fig 16.15.1)**
- May be embedded in bulbar conjunctiva, fornix **(Fig 16.15.2, Fig 16.15.3)** or at the limbus **(Fig 16.15.4)**
- Lid eversion is a must for all cases if there is corneal abrasion or scratch marks
- *Treatment:* easy removal with a sterile cotton bud

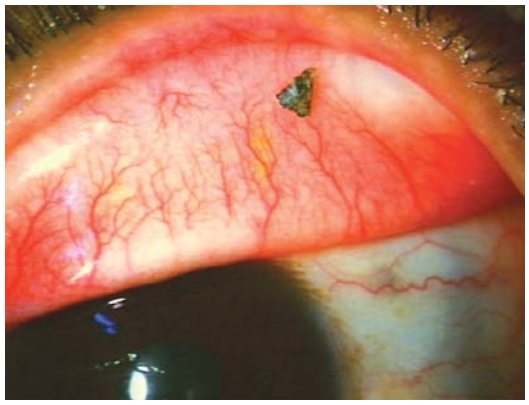


FIGURE 16.15.1: Foreign body-supratarsal sulcus

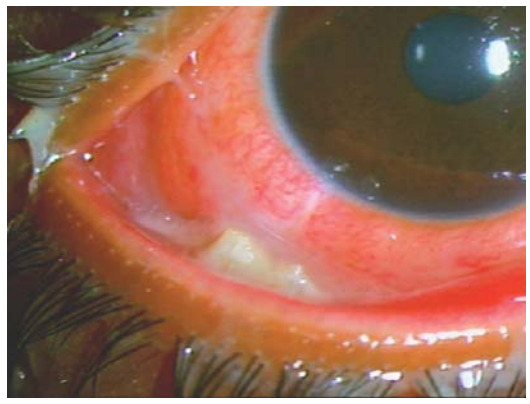


FIGURE 16.15.2: Foreign body in the fornix

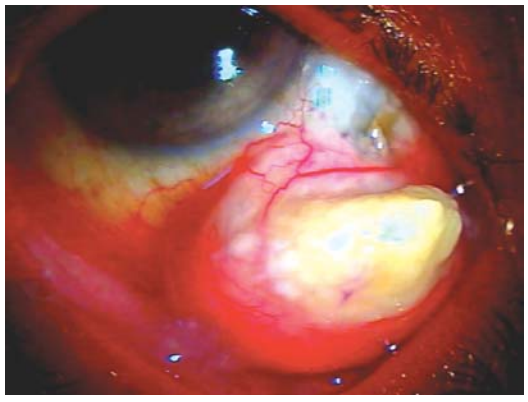


FIGURE 16.15.3: Large foreign body at fornix

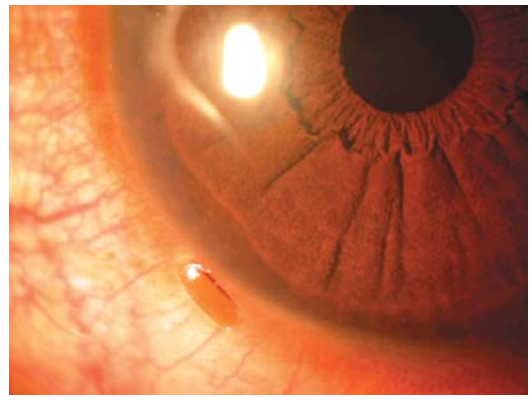


FIGURE 16.15.4: Limbal foreign body-insect wing

Corneal Foreign Body

- Foreign body sensation
- Marked photophobia and redness
- Ciliary congestion
- Particle may be superficial **(Fig 16.16.1)**, anterior stromal, deep stromal **(Figs 16.16.2 and 16.16.3)**, or partly in the anterior chamber **(Figs 16.16.4 and 16.16.5)**
- In old cases of iron foreign body, there may be associated rust ring **(Fig 16.16.6)**
- In addition, there may be surrounding infiltration **(Fig 16.16.7)** or frank corneal ulcer
- Check for tarsal conjunctiva for foreign body and chronic dacryocystitis in all cases of corneal foreign bodies
- *Treatment:* removal of foreign body by a disposable needle under strict aseptic condition and broad spectrum antibiotic drops and ointment for few days

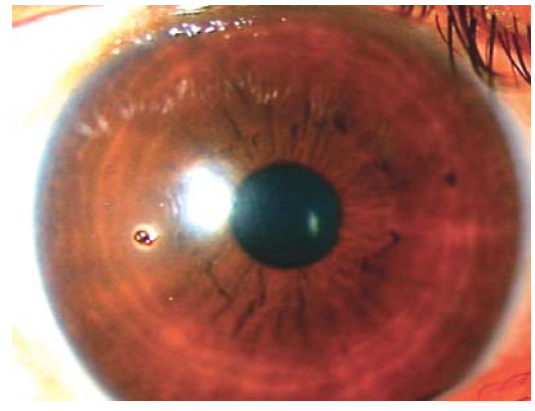


FIGURE 16.16.1: Foreign body cornea-superficial

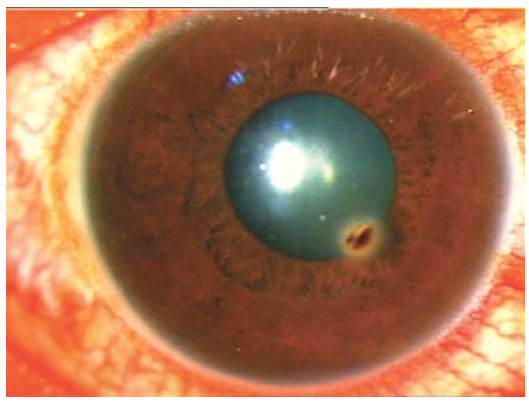


FIGURE 16.16.2: Foreign body cornea-deep

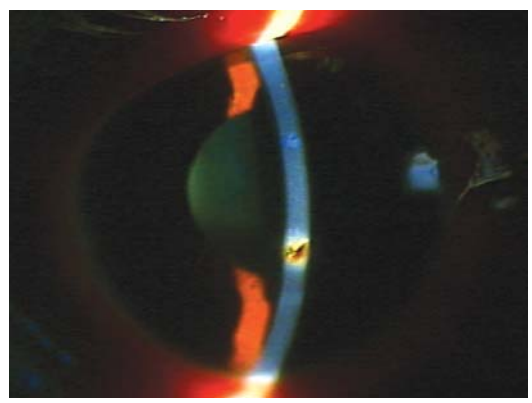


FIGURE 16.16.3: Foreign body cornea-deep

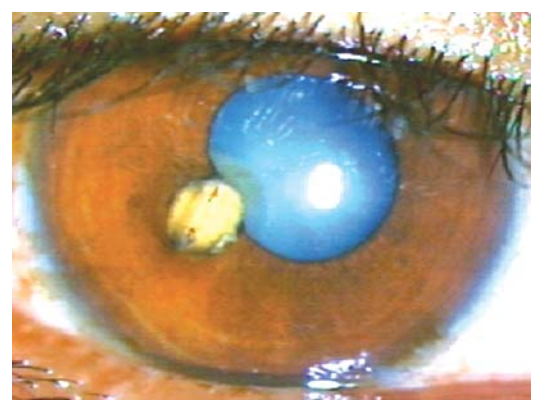


FIGURE 16.16.4: Foreign body partly in anterior chamber

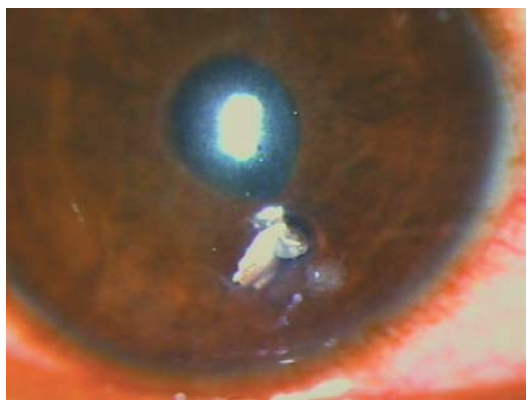


FIGURE 16.16.5: Foreign body partly in anterior chamber

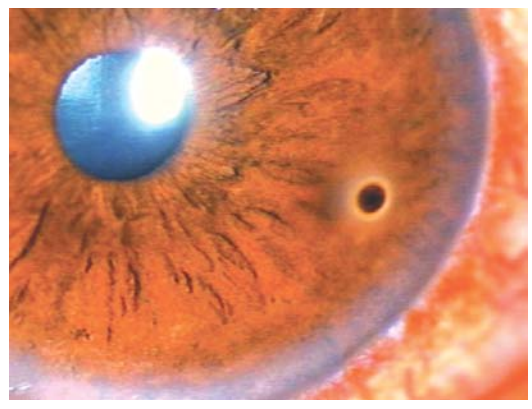


FIGURE 16.16.6: Foreign body cornea-rust ring

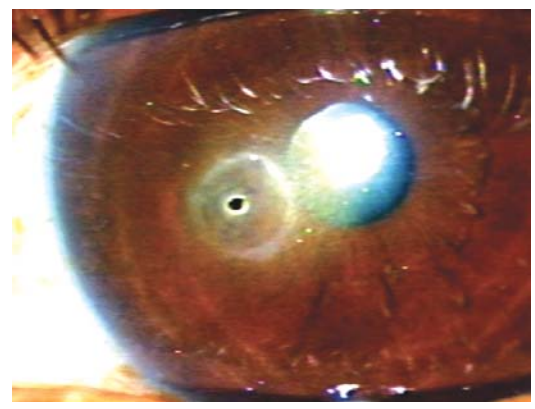


FIGURE 16.16.7: Foreign body cornea with ring infiltration

Intraocular Foreign Body (IOFB)

- While chipping the stone, with an iron-chisel and a hammer, it is a chip of the chisel (from cutting edge, or from its mushroomed head) which enters the eyes, but not the chip of the stone
- May pass directly through the conjunctiva, cornea **(Fig 16.17.1)** or sclera, or sometimes through the lid then through the sclera and may finally lodge onto the retina **(Figs 16.17.2 to 16.17.4)**
- IOFB may retain in the iris **(Figs 16.17.5 and 16.17.6)** or anterior chamber angle **(Figs 16.17.7 to 16.17.9)**
- May lodge in the crystalline lens **(Fig 16.17.10)**, causing cataract or siderotic cataract **(Fig 16.17.11)**
- Pass through the lens, either by way of the iris **(Figs 16.17.12 and 16.17.13)** or pupil, and causing a traumatic cataract
- May retain in the vitreous **(Fig 16.17.14)**
- May rest on to the retina **(Figs 16.17.4 and 16.17.15)**
- Rarely pierce the opposite wall and rest within the orbit (double perforation) **(Fig 16.17.16)**
- May be associated with retinal hole, degeneration, or detachment

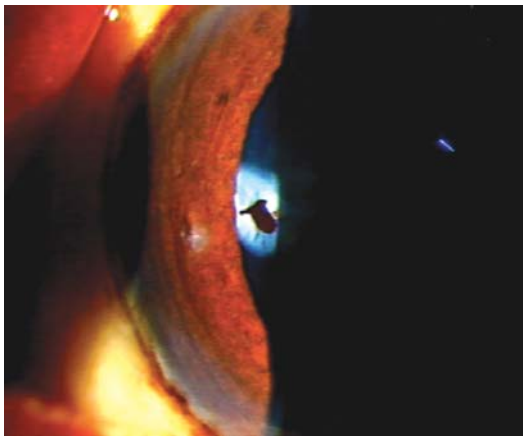


FIGURE 16.17.1: Retained IOFB-wound of entry-cornea

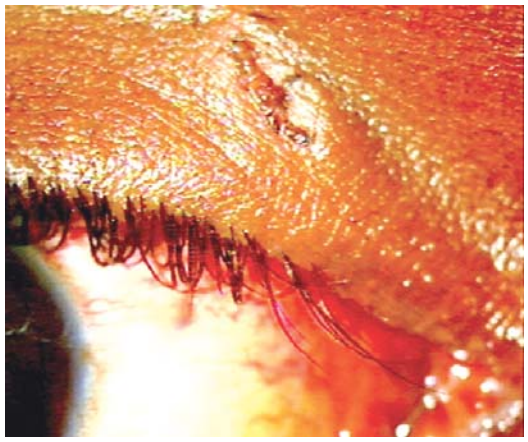


FIGURE 16.17.2: Retained IOFB-wound of entry-lid

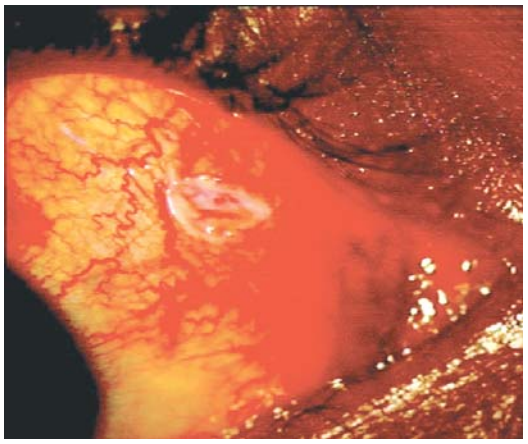


FIGURE 16.17.3: Retained IOFB-wound of entry-sclera

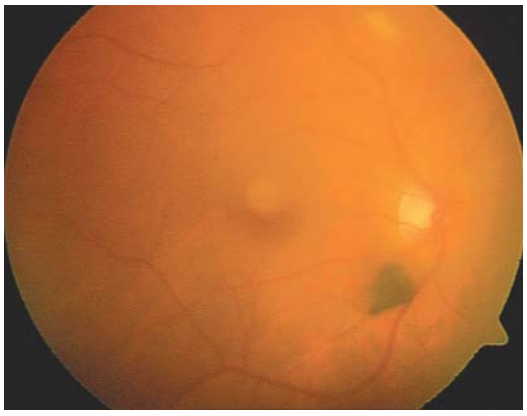


FIGURE 16.17.4: Retained IOFB-on retina

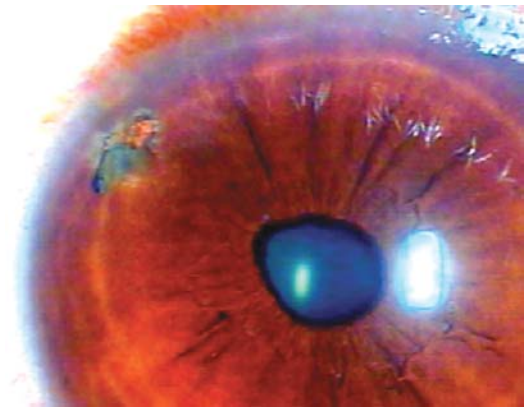


FIGURE 16.17.5: Retained IOFB-through the iris

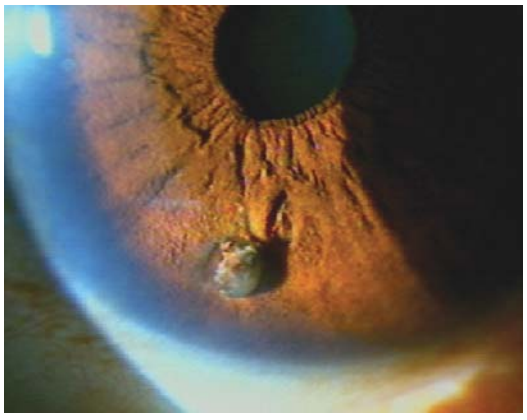


FIGURE 16.17.6: Retained IOFB-on the iris surface

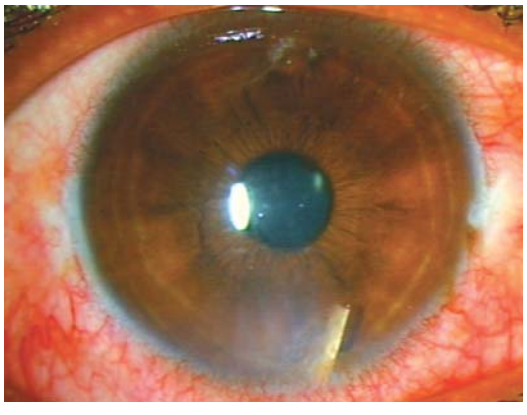


FIGURE 16.17.7: Retained IOFB-angle of anterior chamber

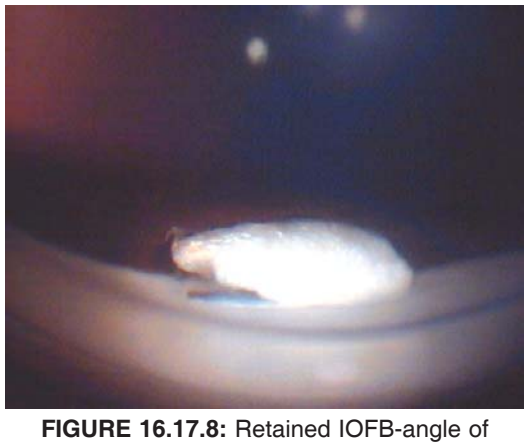


FIGURE 16.17.8: Retained IOFB-angle of anterior chamber

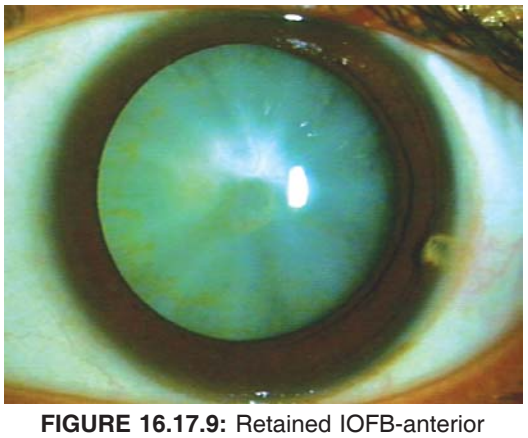


FIGURE 16.17.9: Retained IOFB-anterior chamber-siderotic cataract

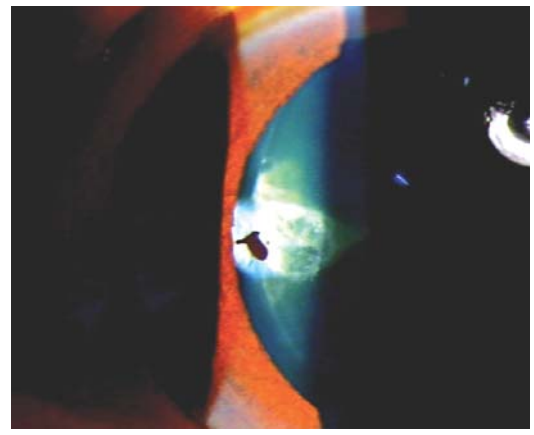


FIGURE 16.17.10: Retained intralenticular IOFB

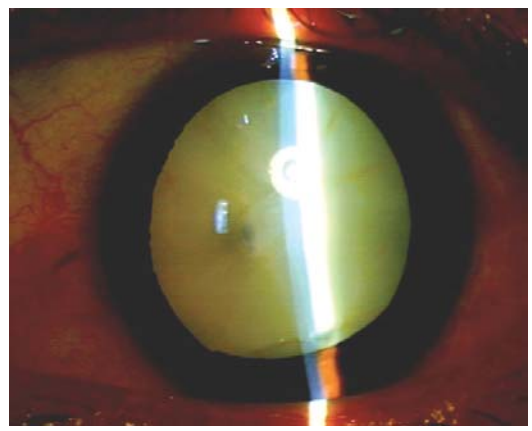


FIGURE 16.17.11: Retained intralenticular IOFB with siderosis

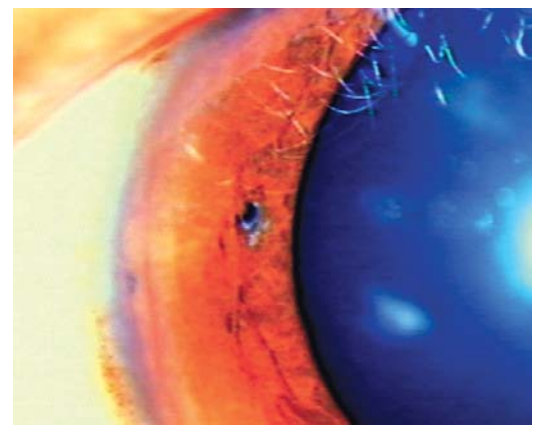


FIGURE 16.17.12: Retained IOFB-iris hole

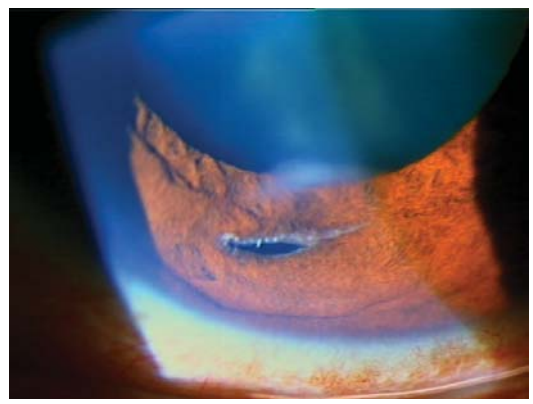


FIGURE 16.17.13: Retained IOFB-iris hole

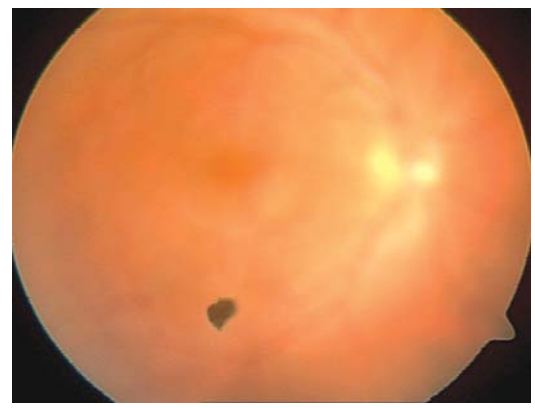


FIGURE 16.17.14: Retained IOFB-in the vitreous

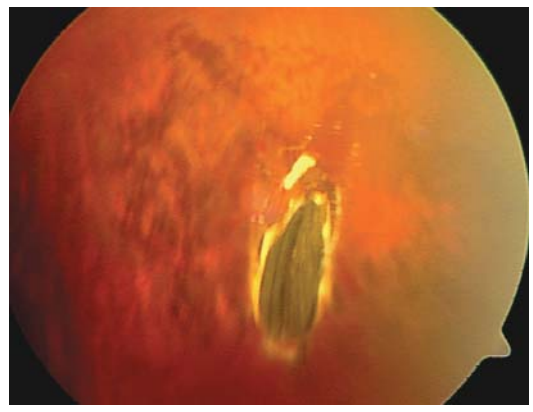


FIGURE 16.17.15: Retained IOFB-on retina

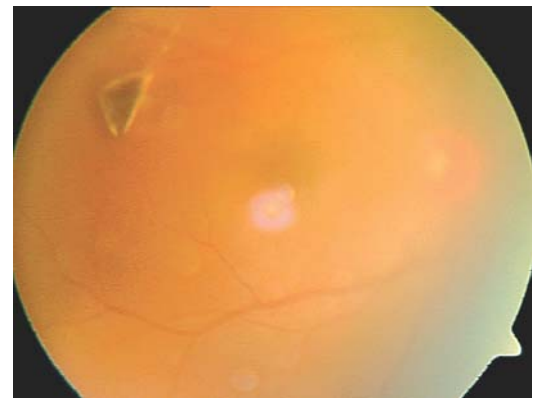


FIGURE 16.17.16: Double perforation

Siderosis Bulbi

- Chronic irreversible degenerative changes of the ocular tissues, caused by retained intraocular iron (and also steel, in proportion of its ferrous content) foreign body
- Rusty deposition on the anterior lens surface (earliest clinical sign) **(Fig 16.18.1)**
- Frank siderotic cataract of variable degree **(Figs 16.18.2 to 16.18.4)**
- Heterochromia of the iris **(Fig 16.18.5)** and rusty discoloration of the cornea **(Fig 16.18.6)**
- Retinal pigmentary changes with attenuation of blood vessels
- Prognosis is always poor

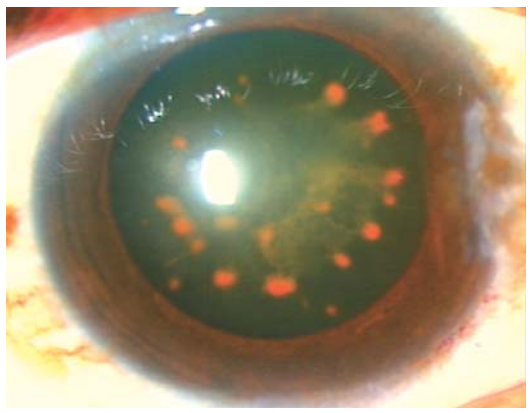


FIGURE 16.18.1: Siderosis bulbi-rusty deposits

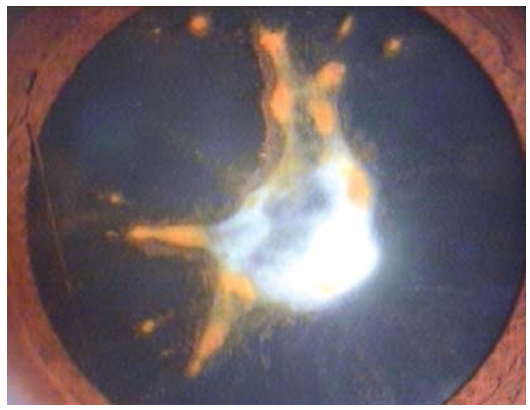


FIGURE 16.18.2: Siderosis bulbi-cataract

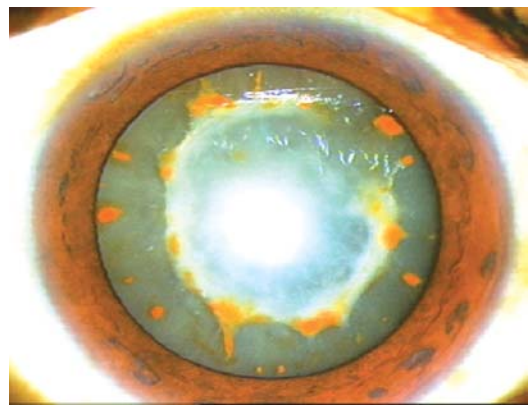


FIGURE 16.18.3: Siderosis bulbi-cataract

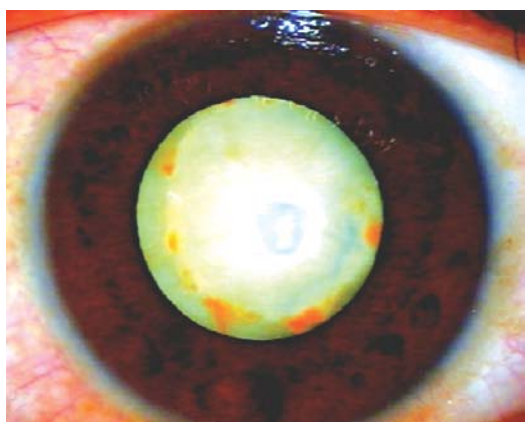


FIGURE 16.18.4: Siderosis bulbi-total cataract

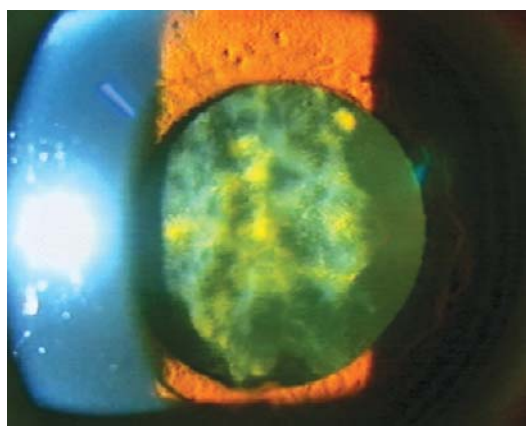


FIGURE 16.18.5: Siderosis bulbi-iris discoloration

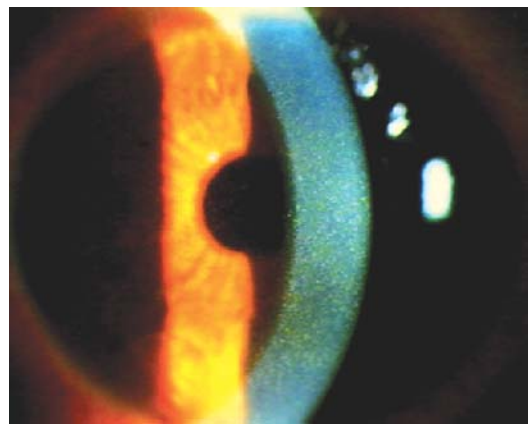


FIGURE 16.18.6: Siderosis bulbi-corneal deposits

Chalcosis Bulbi

- Kayser-Fleischer's (K-F) ring: a golden-brown ring at the level of Descemet's membrane of the cornea **(Fig 16.19.1)**
- *Sun-flower cataract*: a brilliant golden-green sheen in the form of petals of sun-flower **(Fig 16.19.2)**
- Associated retinal changes with golden plaques
- Prognosis is always good

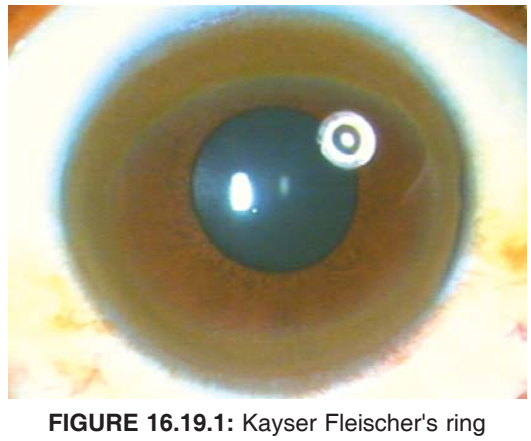


FIGURE 16.19.1: Kayser Fleischer's ring

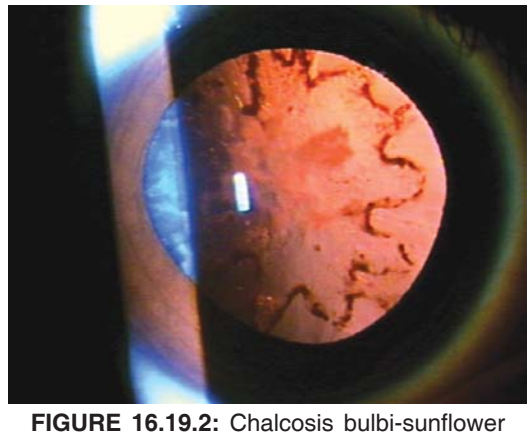


FIGURE 16.19.2: Chalcosis bulbi-sunflower cataract

Miscellaneous Organic Materials

- *Intraocular eyelashes:* cause proliferation of the hair root epithelium leading to the formation of intraocular cysts **(Fig 16.20.1)**
- *Caterpillar hairs*—excite a severe iridocyclitis **(Fig 16.20.2)** with granulomatous nodules called *ophthalmia nodosa* **(Fig 16.20.3)**
- Wood, stone or vegetable materials: produce severe proliferative granulomatous reactions

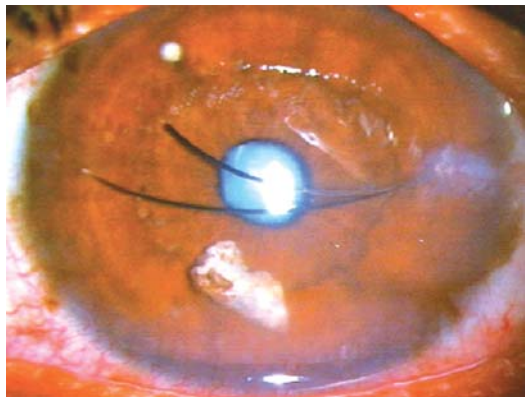


FIGURE 16.20.1: Intraocular eye lashes

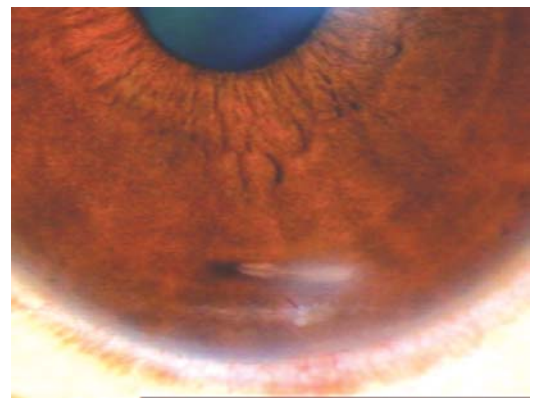


FIGURE 16.20.2: Caterpillar hair-intracorneal

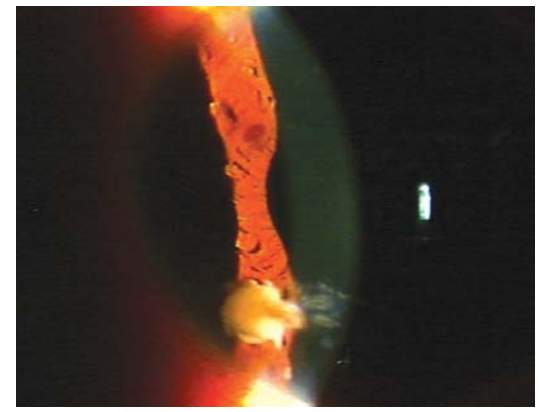


FIGURE 16.20.3: Caterpillar hair-ophthalmia nodosa

CHEMICAL INJURIES (BURNS)

- Alkali burns are more dangerous than acid burns
- Severity depends on type of chemical, its quantity, pH of the solution, concentration of chemical agents, duration of exposure and the time of presentation **(Figs 16.21.1 and 16.21.2)**
- *Acute phase* (upto 1 week)
  - chemosis of conjunctival and eyelids
  - congestion and discharge
  - perilimbal ischemia of variable degree **(Fig 16.21.3)**
  - corneal epithelial defects and stromal clouding **(Fig 16.21.4)**
  - increased IOP
- *Early repairative phase* (1-3 weeks)
  - cornea and conjunctival regeneration
  - persistent corneal epithelial defects **(Fig 16.21.5)**
  - corneal opacity of variable degree **(Fig 16.21.6)**
  - iridocyclitis

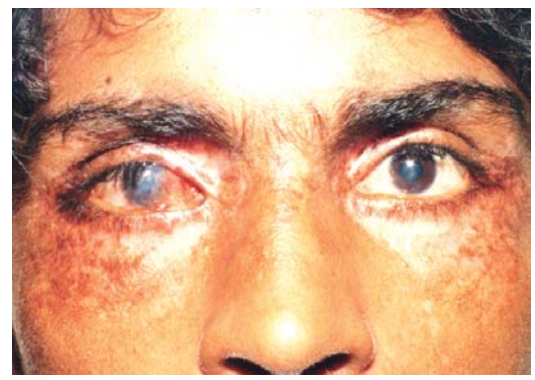


FIGURE 16.21.1: Acid burn

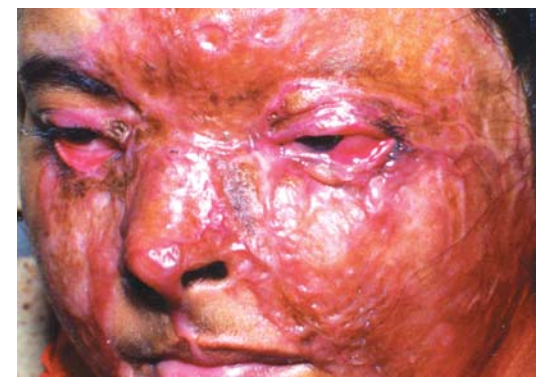


FIGURE 16.21.2: Severe acid burn

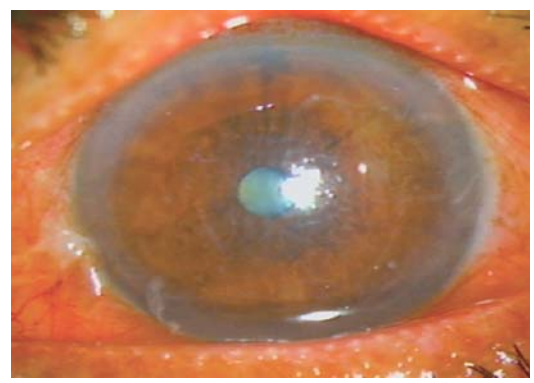


FIGURE 16.21.3: Alkali burn burn-acute phase

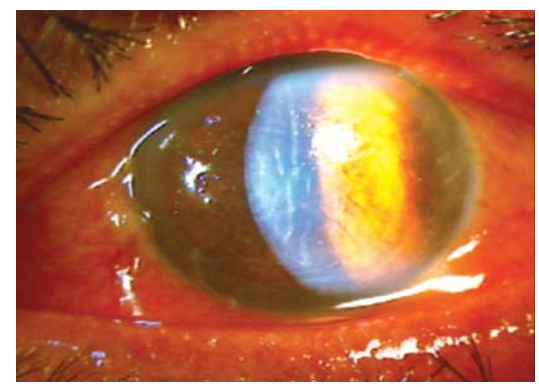


FIGURE 16.21.4: Alkali burn burn-acute phase

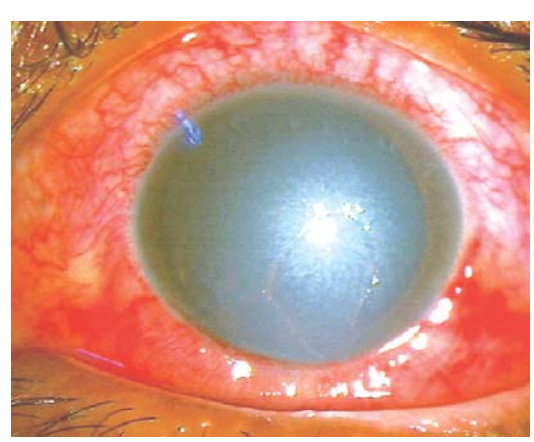


FIGURE 16.21.5: Alkali burn burn-intermediate

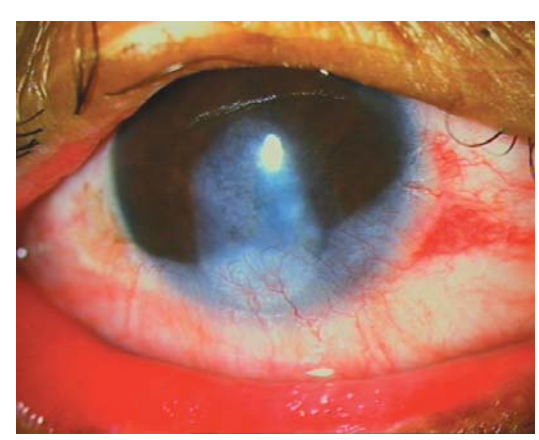


FIGURE 16.21.6: Chemical burn corneal opacity with partial limbal cell deficiency

- *Late repairative phase* (3 weeks to several months)
  - irregular scarring of the cornea
  - corneal thinning and Descemetocele formation
  - dry eye, due to scarring of the ducts of lacrimal glands and goblet cells **(Fig 16.21.7)**
  - hypotony due to ciliary shock
  - development of symblepharon, entropion and trichiasis **(Fig 16.21.8)**

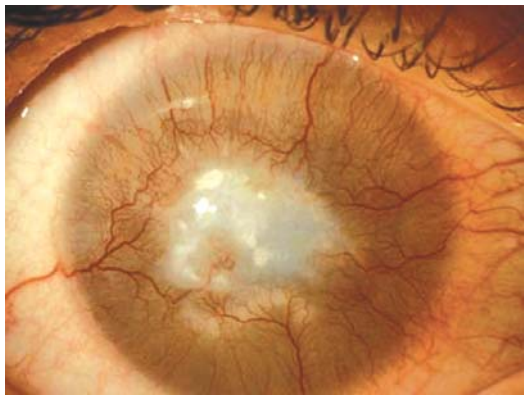


FIGURE 16.21.7: Chemical burn-severe LCDS

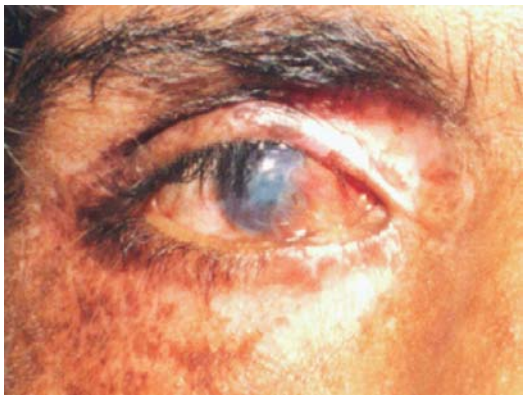


FIGURE 16.21.8: Chemical burn-late stage

THERMAL BURNS

- Usually do not involve the eyeball proper
- May be due to direct burn **(Fig 16.22.1)**, by boiled cooking oil **(Fig 16.22.2)** or by molten metal **(Fig 16.22.3)**
- Thermal burns of the eyelids require prompt care to prevent ectropion formation
- Early skin grafting may be required in some cases
- Associated other features may be due to asphyxia and carbon monoxide poisoning

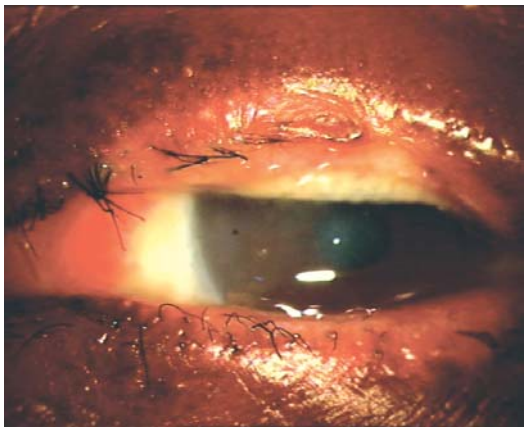


FIGURE 16.22.1: Thermal burn-lid

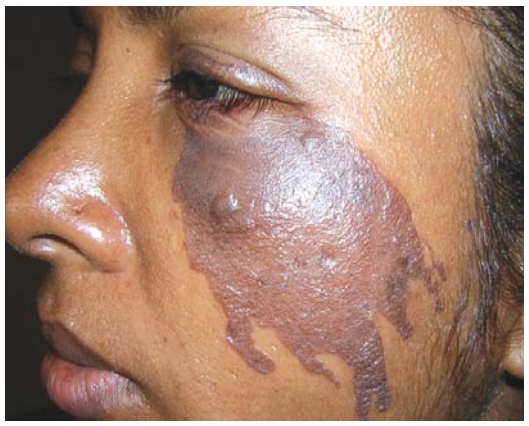


FIGURE 16.22.2: Thermal burn

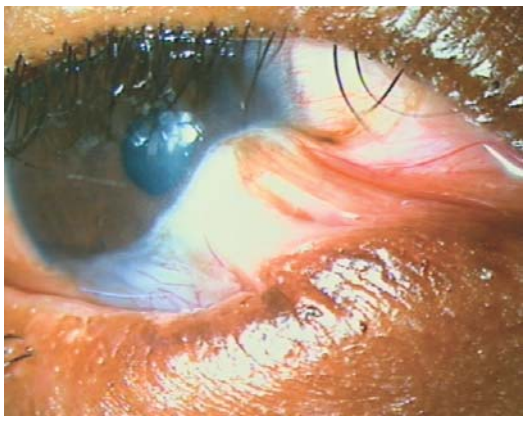


FIGURE 16.22.3: Thermal burn-molten metal

MISCELLANEOUS INJURIES

Blast Injuries

- More common nowadays by explosives, bombs or fire crackers **(Fig 16.23.1)**
- May also be accidental, due to gas cylinder burst, car-tyre burst, etc. **(Fig 16.23.2)**
- Usually associated with facial burn or injuries **(Fig 16.23.3)**

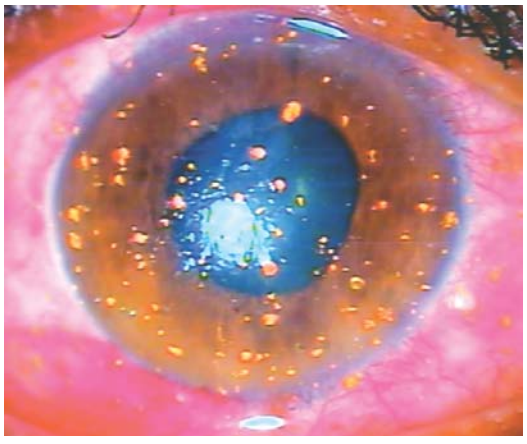


FIGURE 16.23.1: Bomb blast injury-sulphur particles

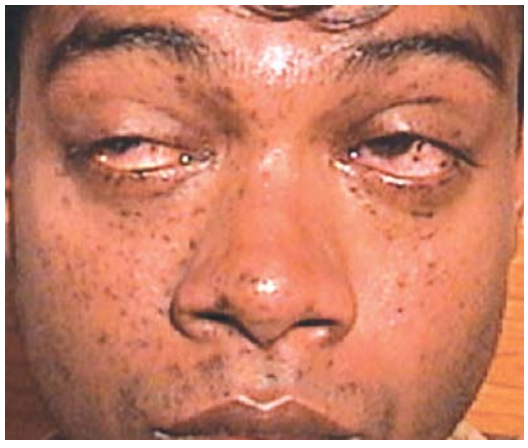


FIGURE 16.23.2: Blast injury-gas cylinder

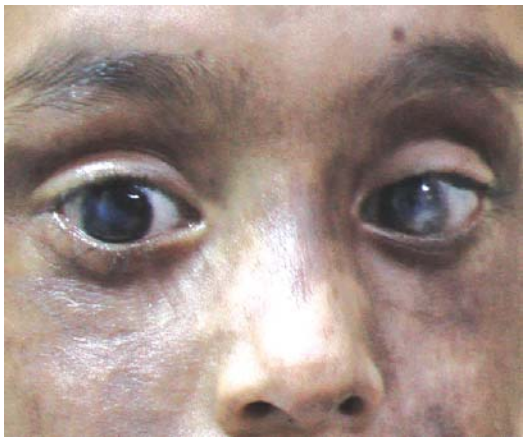


FIGURE 16.23.3: Blast injury-associated facial burn

- Multiple sulphur **(Fig 16.23.4)** or other dusty particles **(Fig 16.23.5)** are scattered all over the anterior surface of the eye, some may be intracameral **(Figs 16.23.6 and 16.23.7)**
- *Treatment:* difficult, initially scraping and BCL, and later a lamellar keratoplasty may be helpful in some cases

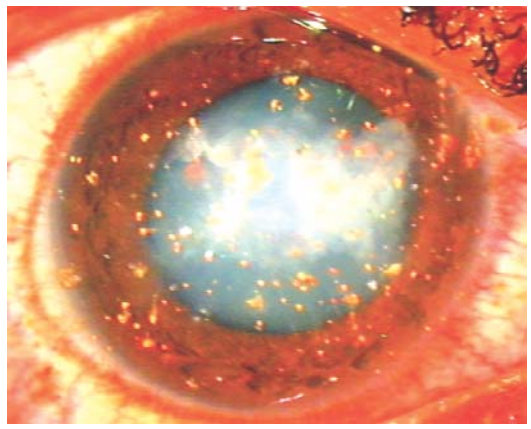


FIGURE 16.23.4: Bomb injury-intracameral sulphur particles with cataract

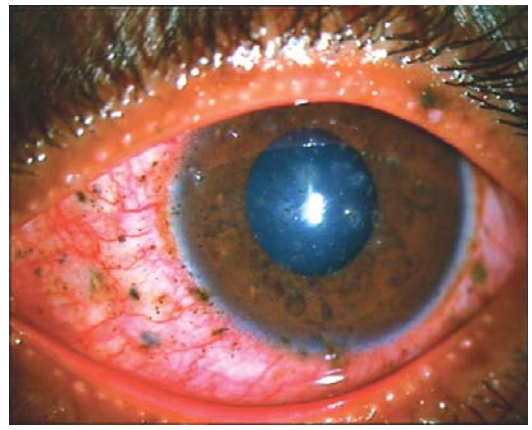


FIGURE 16.23.5: Blast injury-gas cylinder

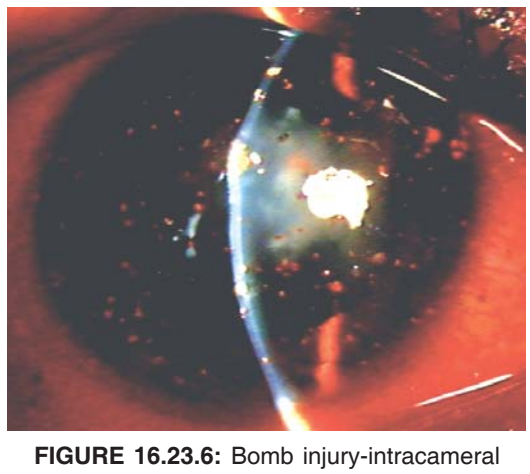


FIGURE 16.23.6: Bomb injury-intracameral sulphur particles with cataract

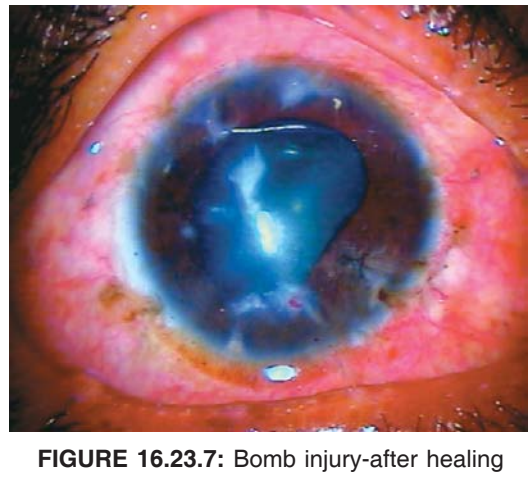


FIGURE 16.23.7: Bomb injury-after healing

Radiational Injuries

- *Ultraviolet (U-V rays):* This results photo-keratitis (UV-keratitis) or snow blindness, or photophthalmia.
- *Infra-red rays (above 700 mm):* They are absorbed by the iris, and the resultant heat is transmitted to the lens, which becomes cataractous *(glass-blower's cataract)* **(Fig 16.24.1)**.
  - solar eclipse with the naked eye, causes solar retinopathy
  - causing a focal macular burn (eclipse burn or eclipse blindness) **(Fig 16.24.2)**
- *Electromagnetic energy* of short wave lengths (X-rays or gamma rays)
  - any part of the eye is affected, e.g. blepharoconjunctivitis (Steven Johnson syndrome-like picture) **(Fig 16.24.3)**, keratitis, radiation cataract and radiation retinopathy **(Fig 16.24.4)**
- *Electric injury or injury after eletrocution*
  - may cause electric cataract in some cases
  - other organ may also be affected
  - retinal damage in various extent may be associated with this injuries

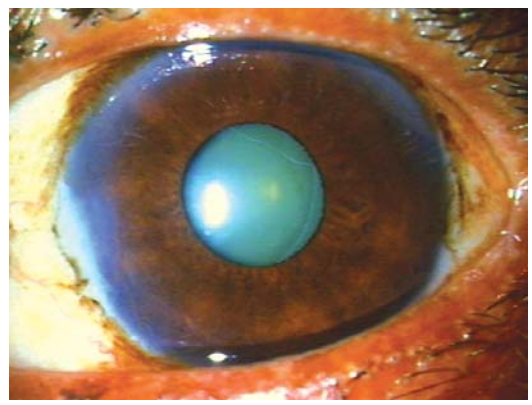


FIGURE 16.24.1: Glass Blower's cataract

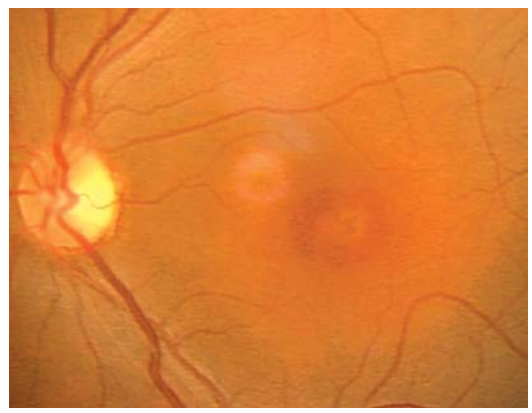


FIGURE 16.24.2: Solar retinopathy

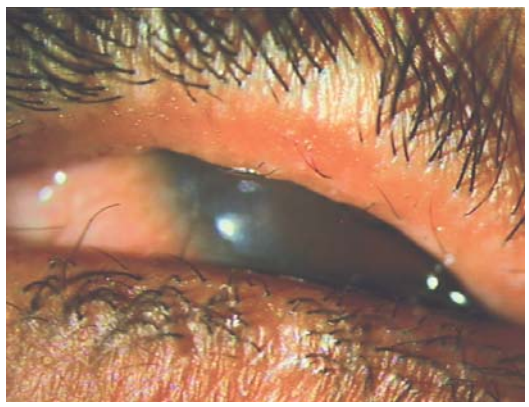


FIGURE 16.24.1: Radiation keratopathy-dry eye

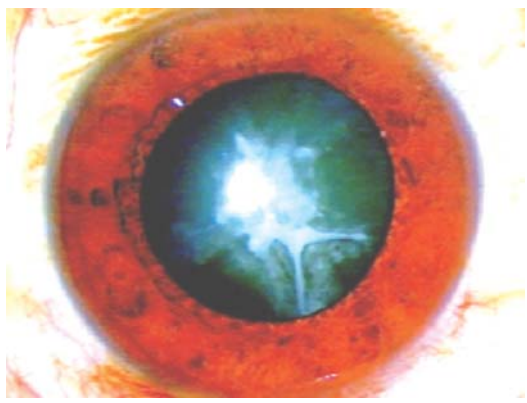


FIGURE 16.24.2: Radiation cataract

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