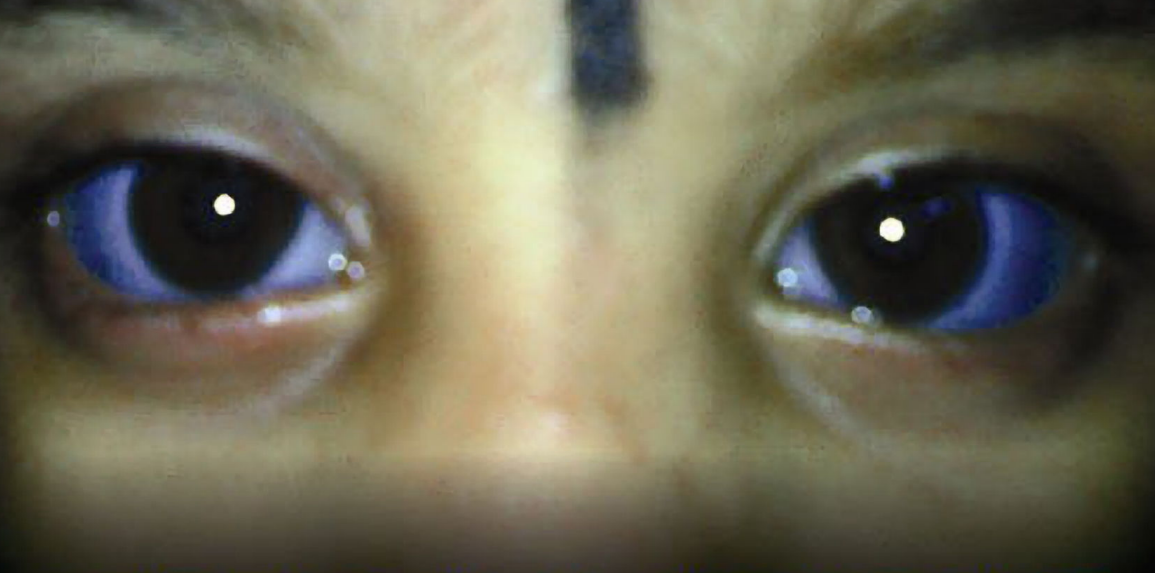
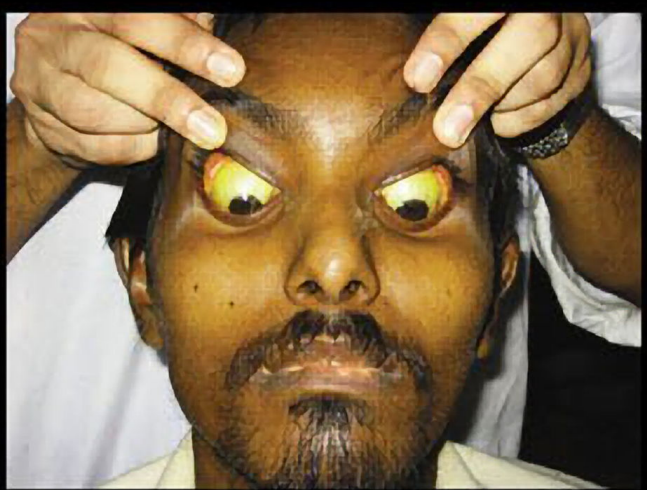
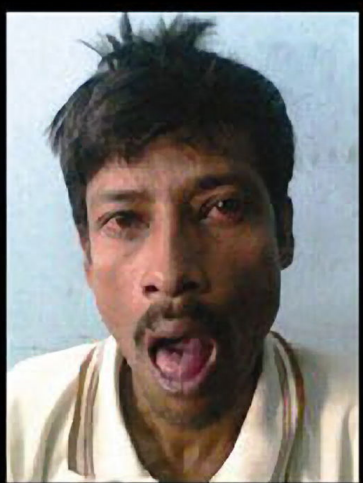


# PEARLS IN MEDICINE FOR STUDENTS

*Mysteries behind Diagnosis*







JAYPEE

Arup Kumar Kundu

# Pearls in Medicine
## *for Students*

***"Great things are not done by impulse,***
***but by a series of small things brought together"***

*—Vincent van Gogh (1853 – 1890)*

***By the Same Author:***

- *Bedside Clinics in Medicine, Part I & Part II*
- *Multiple Choice Questions in Medicine*
  *and*
- *A Section on Online Appendix of "Kumar & Clark's" Textbook, 'Clinical Medicine', 6th edition*

# Pearls in Medicine
## *for Students*

*A Treasure Island in Medicine for Undergraduate and Postgraduate Students*

**Arup Kumar Kundu** MD FICP MNAS
Associate Professor
Department of Medicine
and
In-charge, Division of Rheumatology
Nil Ratan Sircar Medical College
Kolkata, West Bengal
India

JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD

New Delhi • Ahmedabad • Bengaluru • Chennai • Hyderabad
Kochi • Kolkata • Lucknow • Mumbai • Nagpur

*Published by*

Jitendar P Vij
**Jaypee Brothers Medical Publishers (P) Ltd**
B-3 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
Rel: +91-11-32558559 Fax: +91-11-23276490 +91-11-23245683
e-mail: jaypee@jaypeebrothers.com, Visit our website: www.jaypeebrothers.com

***Branches***

- 2/B, Akruti Society, Jodhpur Gam Road Satellite, **Ahmedabad** 380 015
  Phones: +91-79-26926233, Rel: +91-79-32988717, Fax: +91-79-26927094
  e-mail: ahmedabad@jaypeebrothers.com
- 202 Batavia Chambers, 8 Kumara Krupa Road, Kumara Park East**,** **Bengaluru** 560 001
  Phones: +91-80-22285971, +91-80-22382956, +91-80-22372664, Rel: +91-80-32714073
  Fax: +91-80-22281761, e-mail: bangalore@jaypeebrothers.com
- 282 IIIrd Floor, Khaleel Shirazi Estate, Fountain Plaza, Pantheon Road, **Chennai** 600 008
  Phones: +91-44-28193265, +91-44-28194897, Rel: +91-44-32972089
  Fax: +91-44-28193231, e-mail:chennai@jaypeebrothers.com
- 4-2-1067/1-3, 1st Floor, Balaji Building, Ramkote, Cross Road, **Hyderabad** 500 095
  Phones: +91-40-66610020, +91-40-24758498, Rel: +91-40-32940929
  Fax:+91-40-24758499, e-mail: hyderabad@jaypeebrothers.com
- Kuruvi Building, 1st Floor, Plot/Door No. 41/3098, B & B1, St. Vincent Road
  **Kochi** 682 018 Kerala, Phones: +91-484-4036109, +91-484-2395739
  Fax: +91-484-2395740, e-mail: kochi@jaypeebrothers.com
- 1-A Indian Mirror Street, Wellington Square **Kolkata** 700 013
  Phones: +91-33-22651926, +91-33-22276404, +91-33-22276415, Rel: +91-33-32901926
  Fax: +91-33-22656075 e-mail: kolkata@jaypeebrothers.com
- Lekhraj Market III, B-2, Sector-4, Faizabad Road, Indira Nagar, **Lucknow** 226 016
  Phones: +91-522-3040553, +91-522-3040554, e-mail: lucknow@jaypeebrothers.com
- 106 Amit Industrial Estate, 61 Dr SS Rao Road, Near MGM Hospital, Parel
  **Mumbai** 400 012
  Phones: +91-22-24124863, +91-22-24104532, Rel: +91-22-32926896
  Fax: +91-22-24160828, e-mail: mumbai@jaypeebrothers.com
- "KAMALPUSHPA" 38, Reshimbag, Opp. Mohota Science College
  Umred Road, **Nagpur** 440 009 Phone: Rel: +91-712-3245220
  Fax: +91-712-2704275 e-mail: nagpur@jaypeebrothers.com

**Pearls in Medicine for Students**



*First Edition:* **2008**

ISBN 978-81-8448-269-0

*Typeset at* JPBMP typesetting unit

*Printed at* Ajanta Offset

*to*
*My daughter Ushasi*
*My son Abhishek*
*My beloved students*
*for their constant inspiration and moral support*
*and*
*to my sick patients who have taught me*
*the beauty of Medicine*

# Preface

I think myself a student even after learning medicine for more than two and half decades. Teaching a large number of students made me realize the need for such kind of book which deals with common medical presentations, and is lucid, handy, concise, updated as well as truly student-oriented. The aim of this book is to provide guidance for undergraduate and postgraduate students, and young physicians doing private practice or serving villages. This book is a distillation of my experience while answering questions for patients and health professionals over 25 years of practice. The manual consists of short descriptions of facts frequently encountered at the bedside, and I do believe that the cumulative symptomatology with differential diagnoses give a glimpse of real-life story of our day to day clinical practice. There are altogether 75 chapters in the book and each chapter may be regarded as a *window of medicine*; individual chapter also contains many jig-saw puzzles.

*The **contents** are arranged alphabetically while the **index** gives a wider idea about the matters or topics present in the book.* To write a book as a single-handed author is a challenging task, and I am fully aware of this. 'Pearls' help us to crystallize knowledge in our memory very easily. I hope as well as expect that the book will be used as a quick-reference ready-reckoner handbook and a learning-revision tool to increase the core knowledge during early years of medical training. The problem-solving attitude will help the students in their theoretical as well as practical examination, and also in their professional life in future. I hope the extensive and beautiful colour photographs will boost the students while confronting with the patients at the bedside.

This work would not have been possible without the constant support and encouragement from my family members, especially to speak of my wife Bijoya Kundu, my daughter Ushasi and son Abhishek, which ultimately made the book a reality. I would like to appreciate the attitude of my colleagues Dr SK Pal, Associate Professor and Dr P Chattopadhyay, Assistant Professor who helped by giving some interesting clinical

photographs for presentation in the book. I am grateful to all my patients whose photographs are printed here, and to the MSVP, N.R.S. Medical College and Hospital for permitting me to take the photographs.

I would also like to record my appreciation for Mr. Sandip Gupta, General Manager (Sales) and Mr. Sushil Shaw, Branch in-charge of Kolkata office for extending their cooperation in every step. My sincere thanks are extended to Shri JP Vij, CMD and Mr. Tarun Duneja, General Manager (Publishing), of M/S Jaypee Brothers Medical Publishers (P) Ltd, who have helped me throughout and also taken immense pain in publishing this book.

I welcome healthy suggestions and constructive criticisms from the thoughtful readers through e-mail to me (**arup_kundu@hotmail.com**) or the publishers.

**Arup Kumar Kundu**

# Contents

CHAPTER 1

# Abnormal Sweating

## PROLOGUE

The sweat glands are divided into two classes:

a. Eccrine, and
b. Apocrine gland.

The eccrine glands are the major sweat glands in the body and are generally found throughout the surface. The glands present on palms and soles do not respond to temperature but secrete at the time of emotional stress. The apocrine glands are larger sweat glands and are found in axilla, areola of the nipples, mons pubis, labia majora, ear, eyelid and mammary gland.

The eccrine glands are supplied anatomically by sympathetic fibres, yet they are functionally cholinergic (e.g. pilocarpine increases the flow of sweat and atropine abolishes sweating). The apocrine sweat glands respond to circulating adrenaline (these glands are of sexual significance and remain inactive until puberty).

The daily total amount of sweat secreted by a human is approximately 480-600 ml, which may even rise to 10 litres in extremely hot weather.

## TYPES

a. Sensible: When sweating is increased and evaporation stopped, drops of sweat appear on skin surface.
b. Insensible (approximately 500 ml/day): The loss of water from skin surface is neither visible nor perceptible; it is not due to active secretion but occurs as a result of passage of water by diffusion of tissue fluid through the epidermis.

## SWEATING MAY BE CLASSIFIED INTO (NORMAL PHYSIOLOGICAL RESPONSE)

a. Thermal sweating → due to rise of external temperature and is controlled by thermoregulatory centre at hypothalamus.
b. Emotional (mental) sweating → chiefly palms, soles and axillae are involved.
c. Sweating due to muscular exercise/exertion → factors involved are thermal sweating + emotional sweating.
d. Gustatory sweating → eating of spicy food may stimulate sweating in head and neck region.
e. Miscellaneous → as a result of sympathetic overactivity, nausea/vomiting, syncopal attack, hypoglycaemia and asphyxia.

## COMPOSITION OF HUMAN SWEAT

- A clear colourless fluid
- Specific gravity: 1.001 - 1.006; pH 3.8 to 6.5
- Contains mainly water
- Solid present in sweat are lactic acid, carbolic acid, urea, creatinine, sugar, uric acid, nitrogen and non-protein nitrogen, calcium, iodine, iron, sulphur, copper, amino acids, sodium, chloride, potassium and others
- Sodium: 24-312 mg/dl and
  Chloride: 36-468 mg/dl

## HYPERHIDROSIS (GENERALISED)

1. Exercise, anxiety, pyrexia, hot climate.
2. Thyrotoxicosis, hyperpituitarism, acromegaly, carcinoid syndrome, pheochromocytoma, menopause, pregnancy, obesity.
3. Hypoglycaemia.
4. Acute myocardial infarction, heart failure, shock.
5. Tuberculosis, other infections/pyrogens, lymphoma, malignancy, rheumatoid arthritis.
6. Alcohol intoxication, antidepressant drugs, pilocarpine, opiates.
7. Intense pain, syncope.
8. Rickets, infantile scurvy.
9. Neurological lesions of cerebral cortex, basal ganglia, spinal cord and sympathetic nervous system; familial dysautonomia.

## LOCALISED HYPERHIDROSIS

1. Organic neurological lesions–brain tumour, spinal cord injury (may help to localise site of lesion), syringomyelia.
2. Localised sweating of palms, soles and axillae–hot weather, anxiety, psychoneurosis and embarrassment.
3. Dermatological disorders–dyshidrotic eczema, vitiligo, epidermolysis bullosa, palmo-plantar keratoderma, nail-patella syndrome.
4. Pachydermoperiostitis (or primary hypertrophic osteoarthropathy with grade IV clubbing + leonine face)–affects skinfolds of forehead and extremities.
5. Granulosis rubra nasi–rare genetic disorder; sweating of tip of the nose with a diffuse erythema associated with.

## ANHIDROSIS/HYPOHIDROSIS (GENERALISED)

It is less common than hyperhidrosis.

1. Heat stroke
2. Ectodermal dysplasia
3. Scleroderma
4. Organic brain damage, especially of the hypothalamus
5. Ichthyosis
6. Anderson-Fabry's disease
7. Miscellaneous: myxoedema, atopic eczema, psoriasis, lichen planus.

## ANHIDROSIS/HYPOHIDROSIS (LOCALISED)

1. Horner's syndrome (involves half of the face, neck, front and back of upper chest, arm)
2. Diabetic or leprosy neuropathy

* Autonomic neuropathy may lead to anhidrosis and/or gustatory sweating.

## COLD AND CLAMMY SKIN

A classical physical finding in shock, and is due to sweating associated with cutaneous vasoconstriction; commonly found in:

- Hypoglycaemia
- Acute myocardial infarction
- Shock and syncopal states
- Alcohol withdrawal
- Dumping syndrome

* First two conditions give rise to drenching perspiration.

## 'NIGHT SWEATS' IN CLINICAL MEDICINE

1. Tuberculosis
2. Lymphoma
3. Chronic myeloid leukaemia
4. Brucellosis
5. Giant cell arteritis
6. AIDS
7. Nocturnal (sleeping) hypoglycaemia
8. Rheumatoid arthritis (rare).

## OSMIDROSIS (FOUL SMELLING SWEAT)

The personal body odour is basically determined by apocrine gland secretion. Eccrine sweat is usually odourless.

1. Substances excreted in the sweat, e.g. garlic, drugs like dimethyl sulphoxide, arsenic, urea in renal failure (urhidrosis).
2. Hyperhidrosis of sole, complicated by bacterial overgrowth may give rise to foul odour in some persons.
3. Imaginary foul odour is perceived in paranoid delusion.
4. Others (as a result of bacterial overgrowth after sweat excretion): acute rheumatic fever, scurvy, gout, diabetes mellitus, pneumonia, enteric fever.

## CHROMHIDROSIS (COLOURED SWEAT)

1. Pigment produced by chromogenic bacteria.
2. 10% of normal people may have coloured apocrine sweat (yellow/green/blue)–due to the pigment 'lipofuscins'.
3. Drugs excreted through sweat, e.g. rifampicin.

## MILIARIA

These are vesicles (sudmina)/papules (prickly heat) resulting from blockage and rupture of sweat ducts. These are commonly seen in tropical conditions of heat and high humidity. The clear vesicles contain sweat and are often found on the trunk during febrile illness (especially, when the body is covered by blanket during pyrexia), and is known as 'sudaminal rash'.

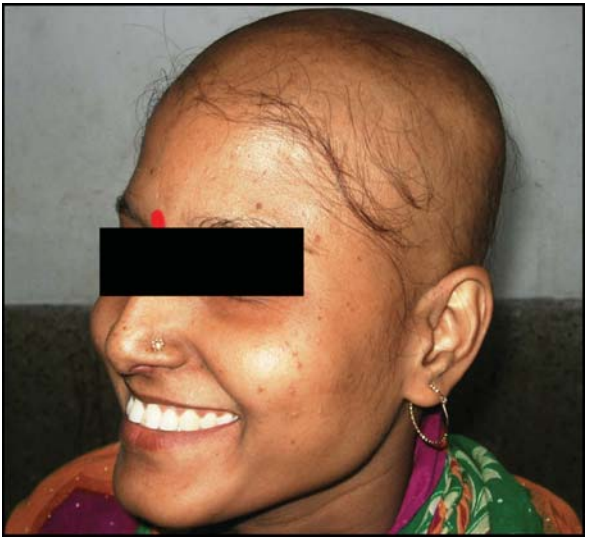
## SWEAT TEST

Pilocarpine iontophoresis test–done to diagnose cystic fibrosis by giving inj. pilocarpine to the patient with measuring the chloride concentration of the sweat → which is very high ( > 60 mEq/L) in cystic fibrosis.

—— o ——

CHAPTER 2

# Alopecia



**FIGUER 2.1:** Chemotherapy-induced non-scarring alopecia (temporary relief of symptoms at the cost of hairs!)

## SYNONYM

Loss of hair

## TYPES

Two

a. Scarring or cicatrical—permanent loss of hair follicle with replacement by scar tissue.
b. Non-scarring—temporary loss of hair follicle and the scalp skin looks normal.

## BASICS

'Hair growth' is influenced by → genetic, racial, nutritional and hormonal factors.

'Hair loss' may result from → changes in hair follicle due to follicular destruction/dysfunction or fracture of fibres.

## CAUSES

### *Scarring or Cicatrical*

- Discoid lupus erythematosus (DLE)
- Tinea capitis with inflammation (kerion)
- Lichen planus
- Imflammatory: bacterial folliculitis
- Idiopathic: 'pseudopelade'
- Folliculitis decalvans
- Scleroderma
- X-irradiation
- Trauma or chemical damage
- Neoplasm
- Congenital (aplasia cutis).

### *Non-scarring*

- Androgenetic alopecia (male-pattern baldness)
- Alopecia areata
- Telogen effluvium
- Traction alopecia
- Tinea capitis
- Trichotilomania (self-induced hair-pulling)
- Lymphoma, leukaemias
- Cosmetic treatment
- Bullous pemphigoid
- Metabolic: iron deficiency anaemia, hypothyroidism, diabetes mellitus
- Drugs: anticancer chemotherapy, oral contraceptive pills, heparin, isotretinoin.

## TRACTION ALOPECIA

'Mechanical damage' type of hair loss; from repeated tugging and pulling of the hair back into a bun or tight plating, and is usually caused by hair dressing styles or special headgear (e.g. turban wearing sikhs).

### PSEUDOPELADE

No cause known. Atrophic, adherent, depressed scarred areas or irregular plaques are seen; stray hair present without any sign of inflammation → may gradually enlarge leaving a shiny scalp without any visible follicular orifice.

### TRICHOTILOMANIA (HAIR PULLING TICS)

Unconscious twisting and pulling of scalp hair; partially alopecic area is formed with irregularly broken, twisted hair growing in different directions.

### TELOGEN EFFLUVIUM

Diffuse hair loss occurring 3 months after pregnancy or a severe illness (e.g. septicaemia, typhoid meningism) → due to 'stress', putting all the hairs into the telogen phase of hair shedding at the same time. Full recovery with normal hair growth within a few months.

### ALOPECIA AREATA

Localised or patchy hair loss which is sharply defined; immune-mediated → commonly seen in children or young adults with patches of baldness → broken 'exclamation mark' hair (i.e. narrow at the scalp, and wider and more pigmented at the tip) seen at the periphery of bald area is diagnostic → rare progression to total scalp (alopecia totalis) or the entire body (alopecia universalis). Regrowth may occur which is initially replaced by white hairs and may take months to cover the patchy area of baldness in alopecia areata.

### MALE-PATTERN BALDNESS

Hair loss is seen primarily at vertex and bi-temporal region of scalp. The thinning of the hair is asymptomatic and gradual. This type of baldness is seen in females after menopause.

### MANAGEMENT

1. Reassurance as alopecia puts the person under social and psychological stress.
2. Wig, camouflage, creative hair style or hair prosthesis, autologous hair transplantation or hair grafting may be of help.

3. Topical immunotherapy: allergic contact sensitization with dinitrochlorbenzene (DNCB), diphencyprone or PUVA may be beneficial.
4. Steroids: topical steroids or intra-lesional injection of steroids (e.g. triamcinolone in alopecia areata) may be given.
5. Topical 2% minoxidil application or systemic finasteride may be of some help in androgenetic alopecia.

—— o ——

CHAPTER 3

# Alteration of Facial Contour

**FIGURE 3.1:** Facial hemiatrophy (right): A patient of Parry-Romberg syndrome

## ASYMMETRY OF FACE

1. Hemiatrophy (e.g. linear scleroderma–en-coup de sabre)
2. Hemihypertrophy
3. Residual Bell's palsy
4. Lipodystrophy
5. Unilateral facial oedema (angioneurotic oedema, postural)
6. Paget's disease (osteitis deformans)–face seems to be an inverted triangle

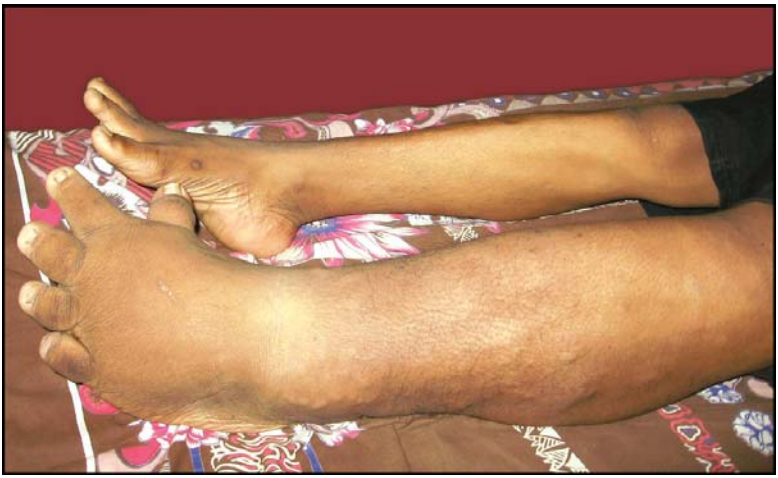


**FIGURE 3.2:** Hypertrophied, deformed and asymmetrical left lower extremity (congenital) – unilateral macrosomia

7. Fibrous dysplasia
8. Absence of condyle of mandible (congenital)
9. Massive swelling of parotid gland (e.g. mixed parotid tumour, Mikulicz's syndrome)
10. Acromegaly
11. Micrognathia (small mandible or receding chin)–e.g. Pierre Robin's syndrome
12. Hemifacial spasm (irregular, painless, clonic contraction involving one half of face, and is commonly due to sequelae of Bell's palsy, compressive lesion of facial nerve, trauma or demyelination)
13. Plexiform neurofibromatosis
14. Absence of teeth or bad (ill-fitted) dentures.

* In Parry–Romberg syndrome, the facial hemiatrophy may be associated with atrophy of skin, tongue, gingiva, soft palate, subcutaneous fat, muscle, bone and cartilage of nose/ear; it is a variety of lipodystrophy.

## COMMON CAUSES OF BILATERAL FACIAL PALSY OF LMN TYPE (FACIAL DIPLEGIA)

1. Acute infective polyneuritis (Guillain-Barré syndrome)
2. Leprosy
3. Sarcoidosis
4. Forceps delivery
5. Leukaemia or lymphoma (deposits in parotids)

6. Bilateral Bell's palsy
7. Bilateral otitis media
8. Diphtheria

* Myasthenia gravis and myopathy develop into facial weakness; there is no involvement of facial nerve.

## PROGNATHISM (BULL-DOG JAW OR LANTERN JAW)

It is the prominence of mandible, and is diagnosed clinically by inspection and observing the lower incisors (mandible) protruding in front of the upper incisors (maxilla); it is seen in:

1. Acromegaly
2. Fragile-X syndrome
3. Nemaline myopathy
4. A particular variety of osteopetrosis.

## LIMB LENGTH INEQUALITY

1. Achondroplasia
2. Fibrous dysplasia
3. Congenital asymmetry (coxa vara, short femur/tibia)
4. Epiphyseal trauma
5. Dislocation of hip joint
6. Osteitis.

## UNILATERAL HEMIHYPERTROPHY

Commonly known as Klippel-Trenaunay syndrome which is internally associated with a spinal cord vascular malformation and externally featured by haemangioma of the trunk or upper or lower extremity + hypertrophy of haemangiomatous limb. The cord lesion may bleed and leads to spinal sensorimotor paralysis.

—— o ——

CHAPTER 4

# Angular Stomatitis

## DEFINITION

Cracking with inflammation of the skin at the angle or corner of mouth. To start with, there is redness → fissuring → crusting.

## CLINICAL ASSOCIATIONS

- Excessive use of betel-leaf, tobacco, alcohol or chewing masala
- Improperly-fitted denture
- Iron deficiency anaemia (associated with glossitis and koilonychia)
- Riboflavin, nicotinic acid, folic acid or pyridoxine deficiency
- Starvation or malnutrition
- Herpes labialis, candidiasis or streptococcal infection at the angle of mouth
- Habitual lip-licking, especially in children

* Angular stomatitis: leaves no scar on healing.

## PERLECHE

- Painful small fissures at the angle of mouth (angular cheilitis)
- Often covered with yellow crusts
- Associated with candidiasis or secondary syphilis.

## RHAGADES

- Linear scars at the angles of mouth and nose
- Commonly seen in congenital syphilis.

## SWOLLEN LIPS

- Injury
- Angioneurotic oedema
- Herpetic infection
- Urticarial rash
- Acromegaly.

—— o ——

CHAPTER 5

# Aseptic Fever

## NON-INFECTIVE CAUSES OF FEVER

1. Drug fever–virtually any drug may produce unexplained pyrexia but the main drugs in the list are rifampicin, sulphonamides, methyldopa, procainamide, different vaccines, following anticancer chemotherapy
2. Connective tissue diseases, e.g. SLE, rheumatoid arthritis, polymyalgia rheumatica, temporal arteritis, polyarteritis nodosa
3. Malignancies:
   - Carcinomas (especially of lung, liver, and kidney)
   - Lymphomas
   - Leukaemias and other haematological malignancies
4. Thyroid storm
5. Pontine heamorrhage
6. Heat stroke
7. Crush injury
8. Over-atropinisation or dhatura poisoning
9. Acute myocardial infarction
10. Gout
11. Radiation sickness
12. Malignant hyperthermia (halothane-induced) or neuroleptic malignant syndrome (haloperidol-induced).

## FEVER ASSOCIATED WITH MACULO-PAPULAR RASH

1. Measles: Usually appears on the fourth day of illness, maculo-papular in type. Rash first appears at the back of the ears, and at the junction of skin and hair on the forehead; ultimately face, neck, trunk, limbs upto palms and soles may be affected. The density of rash is greatest

on the forehead. They are discrete, pink, and blanch on pressure. Later, the rashes become confluent and give rise to characteristic blotchy appearance (morbilliform rash).

2. Infectious mononucleosis: Rash in this disease usually follows the administration of penicillin or ampicillin for a presumed pharyngitis. Associated suggestive features are generalised lymphadenopathy along with splenomegaly.
3. Meningococcaemia: Usually the rash is haemorrhagic but for the first 12-24 hours it may occasionally be erythematous and maculo-papular simulating rash of measles. Usually it is a disease of children and young adults with characteristic short prodrome (unlike measles and scarlet fever), a rapidly changing rash, pallor and toxicity.
4. Toxic shock syndrome: This is a disease of females with staphylococcal infection, commonly associated with use of tampons. The patient is acutely ill with vomiting and diarrhoea, headache, and myalgia which progresses towards shock and rapidly developing maculo-erythematous rash.
5. Scarlet fever: The rash first appears behind the ears on the second day and rapidly becomes a generalised punctate erythema which is mostly abundant in flexures of the arms and legs. The affected children usually have a flushed face due to fever; the rash does not affect face.
6. Rubella: The rash (pink macules) usually begins on the second or third day on face and neck (like measles) but progresses much more rapidly than measles, and become generalised within 24-48 hours. Postauricular and occipital lymphadenopathy are characteristic.
7. Enteric fever: Rose spots are sparse, small rose-red, blanching, slightly raised macules mainly present over upper abdomen and chest during the end of the first week of illness. It is usually visible only on fair-skinned persons. Rarely, these rashes can evolve into non-blanching small haemorrhages.
8. Dengue: Primary rashes appear on the 3rd day of illness, which are erythematous (diffuse flushing) and present over face, neck and shoulder. Secondary or true rash are measly or morbilliform which appears on the 6th day of illness and is usually present over the dosrum of hands and feet. Ultimately, the rash becomes generalised (mostly over the trunk) except the face. The rash may persist from two hours to several days and terminates by desquamation. Classically, there is associated 'saddle-back' pyrexia.

9. AIDS: A mononucleosis-like syndrome may be seen 2-6 weeks after acquisition of HIV infection. The rash (occurs in 50% patients) is macular, erythematous, and predominantly affects the trunk. The illness is associated with pyrexia, perspiration, lethargy, arthralgia, myalgia and generalised lymphadenopathy.
10. Typhus: Different varieties of typhus like epidemic and endemic typhus, Rocky Mountain spotted fever, scrub typhus and rickettsialpox are associated with pyrexia and rash.
11. Kawasaki disease: The affected children may have a polymorphous rash along with fever, conjunctivitis, red lips and red tongue, red indurated hands and cervical lymphadenopathy.

## PYREXIA OF UNKNOWN ORIGIN (PUO)

Definition (by Petersdorf and Beeson, 1961):

1. Fever > 101°F on several occasions.
2. A duration of fever > 3 weeks.
3. Failure to reach a provisional diagnosis after one week of inpatient investigations or by three outpatient visits.

Recently PUO has been classified into 4 different types (Durack and Street, 1991):

1. Classic PUO (as previous definition).
2. Nosocomial PUO (hospital-acquired).
3. Neutropenic PUO (when neutrophil count is ≤ 500/mm³).
4. HIV-associated PUO (e.g. tuberculosis, NHL, drug fever).

## CLINICAL EXAMINATION OF PUO WHICH NEEDS SPECIAL ATTENTION

1. Oral cavity: Teeth and gum for sepsis.
2. Thyroid: Enlargement and tenderness → thyroiditis.
3. Eye: Phlyctenular conjunctivitis and other manifestations of systemic disease.
4. Lymph nodes: With special attention to cervical and epitrochlear nodes.
5. Bone: Sternal tenderness, gibbus with tenderness.
6. Musculoskeletal system–Still's disease, systemic onset juvenile idiopathic arthritis.
7. Skin: Rashes and nodules.
8. Blood vessels: Evidences of vasculitis/arteritis (temporal arteritis).

9. CVS: Murmurs and pericardial rub.
10. Respiratory system: Pleural rub, basal pneumonia.
11. GI tract: Enlargement of liver, spleen or kidney; tenderness of renal angle.
12. Intercostal tenderness: Amoebic liver abscess/empyema thoracis.
13. Genitalia: Phimosis, discharge per urethra, any swelling, epididymitis (filarial/tubercular).
14. Per rectal or vaginal examination: Abscess, tumours.
15. Fundoscopy: Choroid tubercles.
16. Covered area, if any: Breast abscess, any sepsis under bandages/plasters.

## POSSIBILITIES IN COMBINATION WITH ARTHRITIS, FEVER, AND RASH

- Viral infections (rubella, parvovirus B-19)
- Gonococcaemia or meningococcaemia
- Periodic fever syndrome
- Acute rheumatic fever
- Adult Still's disease
- Sarcoidosis
- Serum sickness
- Secondary syphilis
- Familial Mediterranean fever
- Vasculitis
- Lyme diaease

—— o ——

CHAPTER 6

# Bald Tongue

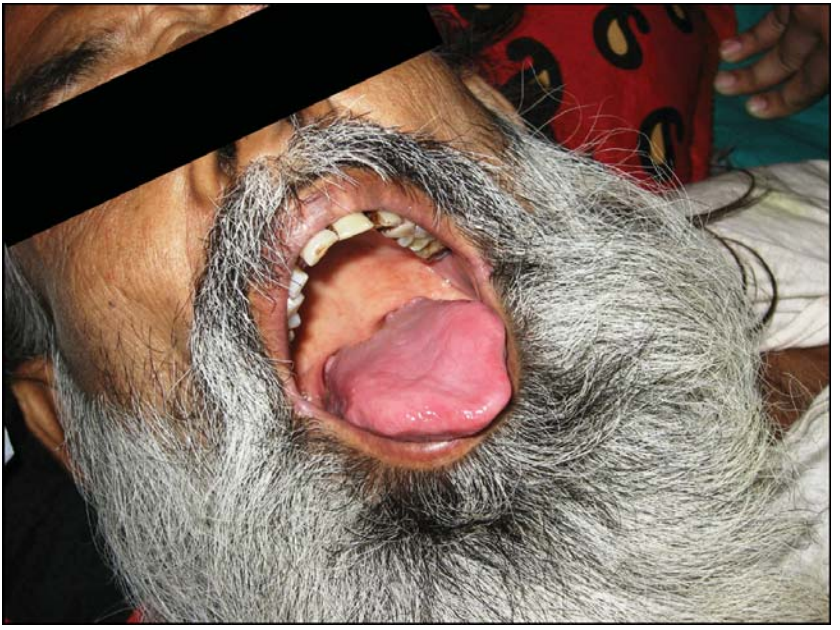


**FIGURE: 6.1:** Bald tongue developing from iron deficiency anaemia

## DEFINITION

Total loss or atrophy of the papillae resulting in a smooth or plane dorsum of the tongue.

## CLUE TO DIAGNOSIS

1. Iron deficiency anaemia (most common cause)
   ↓
   H/O bleeding piles, menorrhagia or melaena; intake of NSAIDs; dietary history is important; enquire H/O 'pica' (eating of strange substances like mud, ice etc); working bare-footed (hookworm infestation)?
   ↓
   Look for anaemia, glossitis, angular stomatitis, cheilosis, koilonychia, mild splenomegaly (rarely found)
   ↓
   Enquire for dysphagia, specially in middle-aged women (Plummer-Vinson syndrome or Paterson-Kelly syndrome)
   ↓
   Investigate: Blood for Hb%, peripheral smear, RBC morphology, colour index, serum Fe, TIBC, plus
   Stool for occult blood and hookworm ova
   ↓
   *Examine:* Per rectally or per vaginally, associated with upper and lower GI endoscopy in search of a carcinoma of large gut/fibroid of uterus or peptic ulcer.
2. Pellagra
   - Dermatitis found in nicotinic acid deficiency
   - Seen in exposed part with erythema and desquamation
   - There may be roughening and pigmentation
   - Dermatitis in the neck (Casal's collar) is pathognomonic
   - Associated cheilosis, angular stomatitis and raw-beefy tongue
3. Pernicious anaemia
   ↓
   Strict vegan or H/O gastrectomy
   ↓
   H/O glossitis, cheilitis, tingling and numbness or premature graying of hair
   ↓
   M = F, around age of 60 years
   ↓
   Investigate: Peripheral blood smear with MCV, MCHC, RBC morphology (it is the commonest cause of vit $B_{12}$↓ in temperate climate); histamine-fast achlorhydria, plus +ve Schilling's test

4. Tropical sprue
   ↓
   Residents or visitors to tropical regions
   ↓
   H/O malabsorption
   ↓
   C/F like anorexia, diarrhoea, ↓ weight, anaemia and features of different nutritional deficiency like glossitis, cheilosis
   ↓
   ↓ of Fe, folic acid and vit $B_{12}$
   ↓
   Investigate: ↑ faecal fat, and +ve d-xylose absorption test
   ↓
   +ve jejunal biopsy with shortened and thickened villi, ↑ crypt depth, infiltration of mononuclear cells in lamina propria and epithelium.
5. Syphilis
   ↓
   Young adult with H/O exposure
   ↓
   C/F like skin rash, condyloma lata, lymphadenopathy and snail track ulcers in mouth
   ↓
   Along with the bald tongue, mucous patches in lips, oral mucosa, palate, pharynx, vulva, vagina, glans penis or inner prepuce may be seen
   ↓
   Investigate: VDRL, Kahn test, FTA-ABS (fluorescent treponemal antibody-absorption) or TPI (treponema pallidum immobilisation test)

* In iron deficiency anaemia (microcytic-hypochromic anaemia) MCV, MCH and MCHC are low; serum Fe is low but TIBC is increased. Pernicious anaemia is a macrocytic anaemia with high MCV, MCH and MCHC.

## MESSAGE

Treat bald tongue mainly with Fe and vitamin B-complex

* For the causes of 'dry tongue', read dry mouth under 'Parotid swelling'.

## WHITE PATCHES IN TONGUE

1. Leukoplakia of tongue:
   - Look for sharp teeth, ill-fitting dentures, sepsis (chronic infection), H/O chewing tobacco or smoking or consuming alcohol, syphilis (rare, nowadays)
   - Pre-malignant condition
   - Superficial layer of tongue shows extensive keratinisation and cornification (causes whiteness of the patch)
   - If non-responsive to conservative treatment, biopsy should be done to exclude malignancy
2. Hairy leukoplakia (serrated white areas in margins of tongue; painless and are due to Epstein-Barr virus infection in a patient of AIDS).
3. Candidiasis of tongue.
4. Severe and chronic iron deficiency anaemia.

* Except in candidiasis, all the plaques are adherent to tongue

** 6 'S' in leukoplakia are sharp teeth, sepsis, smoking, spirits, syphilis and spices

—— o ——

CHAPTER 7

# Blackish Urine

## POSSIBILITIES

Though very rare, black discolouration of urine often puzzles the clinicians.

1. Alkaptonuria: The urine becomes black on standing, often after voiding. If collected in a test tube, it starts blackening from above downwards. The urine blackens on alkalinization too. Confirmation of the diagnosis is done by chromatographic and spectophotometric study of urine which demonstrate presence of homogentisic acid. It is due to deficiency of homogenitisic acid oxidase in human body. The other name of the disease is *ochronosis*. The brown stain of the napkin of infants makes the parents suspicious of some serious illness; the child may have grey-brown sclera. The adults may present with arthritis of big joints while the roentgenography of spine may reveal calcification of intervertebral discs.
2. Tyrosinosis: An autosomal recessive disorder of tyrosine aminotransferase deficiency associated with hepatic and renal tubular dysfunction.
3. Melanuria: In disseminated melanoma, melanogen present in urine gives the black discolouration.
4. Poisoning with phenol or cresol–Addition of ferric chloride turns the urine into blue or violet colour.
5. Drugs: Quinine, methyldopa or porphyrin therapy.

* Blackwater fever due to severe *P. falciparum* infection produces red urine

## TREATMENT

- Relieve anxiety. Reassurance
- Adequate fluid intake to prevent renal insufficiency in cases of drug-induced or poisoning-induced black urine
- Treatment of the specific cause.

## BLACK STOOL

1. Melaena (altered blood due to production of acid haematin).
2. Ingestion of iron as haematinic (produces hard stool in contrast to melaena which is semisolid in consistensy).
3. Ingestion of bismuth (used in treatment of duodenal ulcer).
4. Intake of liquorice, charcoal (used in treating poisoning) or black berries.

* No (2), (3) and (4) are non-sticky, and often known as 'pseudomelaena'.

—— o ——

CHAPTER 8

# Blue Fingers/ Toes

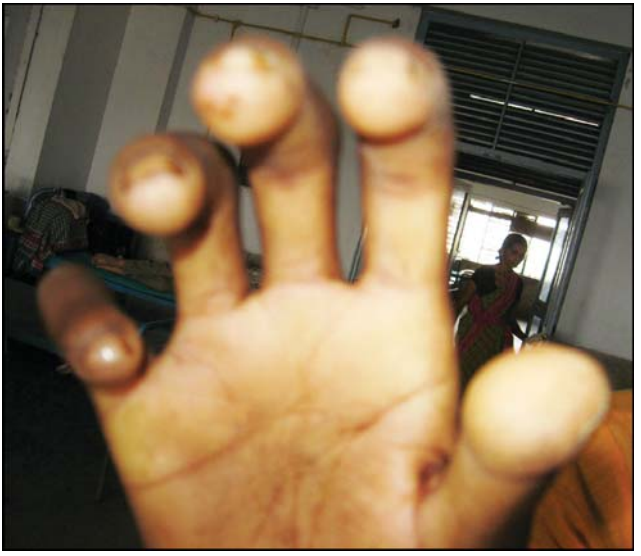


**FIGURE 8.1:** Digital infarction (cicatrical depression at fingertips) in progressive systemic sclerosis

## PROLOGUE

Blue fingers or toes are seen in vasospasm due to any condition, commonly as a result of Raynaud's phenomenon. True cyanosis (central or peripheral) is also responsible for blueness in finger/toetips and nail-beds.

## RAYNAUD'S PHENOMENON

It is a vasospastic disorder (i.e. intense vasospasm of peripheral arteries), manifested clinically by the classical 'triphasic colour response' which is

sequential development of digital blanching (pallor due to vasospasm), cyanosis (blue due to sluggish blood flow) and rubor (redness due to vasodilatation or reactive hyperaemia) of the fingers and toes following cold exposure and subsequent rewarming. Some patients may develop only pallor and cyanosis, while others may experience cyanosis only. The changes in the fingers and toes are often diagnosed by nail-fold capillography. The fingers are affected more commonly than the toes. The duration of the attack is variable but may last for hours. Numbness, burning or paraesthesia, and severe pain in the digits are common features.

## MECHANISM

1. Exaggerated reflex sympathetic vasoconstriction.
2. Enhanced digital vascular responsiveness to cold or to normal sympathetic stimuli

| *Colour* | *Mechanism* | *Clinical features* |
|---|---|---|
| Pallor (White) | Vasospasm (ischaemia) | Pain and numbness |
| Cyanosis (blue) | Subsequent venular spasm (stasis) | Pain and numbness |
| Rubor (red) | Vasodilatation (reperfusion) | Warmth, throbbing pain |

## COMMON CAUSES

A. Primary Raynaud's phenomenon: Raynaud's disease (F > M, 15-30 years, bilateral and symmetrical, no cause found, fingers > toes; +ve family history may be there; over 50% of patients with Raynaud's phenomenon have Raynaud's disease. Rarely, the earlobes and the tip of the nose may be involved. The radial, ulnar and pedal pulses remain normal as it is a disease of arterioles; it does not progress to digital ulceration or infarction. Long-acting preparation of nifedipine may be of some help.

B. Secondary Raynaud's phenomenon:
   1. Collagen vascular diseases: Scleroderma, SLE, dermatomyositis, CREST syndrome, MCTD, rheumatoid arthritis.
   2. Obliterative arterial disease: Atherosclerosis, thoracic inlet syndrome, thromboangiitis obliterans.
   3. Occupational: vibration tool injury, electric shock, exposure to cold (e.g. frost bite), piano playing, vinyl chloride, typing.
   4. Blood dyscrasias: Myeloproliferative disorders, hyperviscosity syndromes, cryoglobulinaemia.

5. Neurologic disorders: Syringomyelia, carpal tunnel syndrome, spinal cord tumours.
6. Drug-induced: Ergot derivatives, β-blockers, methysergide, vinblastin or bleomycin.
7. Miscellaneous—pulmonary hypertension, crutch pressure, carcinoid syndrome.

## OTHER VASOSPASTIC DISORDERS

1. Pernio (chilblains) → Swelling of tips of toes with ulceration, often associated with pruritus and burning sensation → associated with cold exposure.
2. Erythromelalgia → M > F, feet > hands with burning pain and erythema of the extremities → may precipitate from myeloproliferative disorders, or nifedipine/bromocriptine-induced → precipitated by warm exposure and relieved by exposing to cool air or elevation of the limb.
3. Livedo reticularis (read the section on 'Purpuric spots').
4. Frost bite.
5. Vasculitis due to any cause.
6. Acrocyanosis → persistent cyanosis of the hands, less frequently, the feet → resulting from arterial vasoconstriction and secondary dilatation of capillaries and venules → cold exposure increases the incidence. F > M and usually below 30 years of age. Asymptomatic with normal pulses and without any skin ulceration → D/D with Raynaud's phenomenon (acrocyanosis is persistent and not episodic, and blanching does not occur).

## MANAGEMENT

1. Reassurance. Avoid unnecessary exposure to cold or trauma. To wear gloves and mittens, protect the body with warm clothing and abstain from tobacco smoking.
2. *Drugs:* reserved for severe cases.
   - Nifedipine, amlodipine, diltiazem (calcium-channel blockers)
   - Reserpine (adrenergic blocking agent)
   - Prazosin, doxazocin, terazocin ($\propto_1$- adrenergic antagonist)
   - Methyldopa, guanethidine, phenoxybenzamine (sympatholytics)
   - Iloprost (prostacyclin analogue and an endogenous vasodilator)
   - Pentoxyfylline, naftidrofuryl, inositol nicotinate
   - Calcitonin gene-related peptide

- The phosphodiesterase inhibitor sildenafil and oral endothelin antagonist bosentan are currently being tried
- Surgical sympathectomy.

## ULCERS (DIGITAL) AT FINGER OR TOETIPS

1. Raynaud's phenomenon, e.g. scleroderma
2. Thromboangiitis obliterans
3. Leprosy
4. Diabetes mellitus
5. Trauma
6. Vasculitis
7. Atherothrombosis.

## ERYTHEMA OF FINGERS

1. Dermatomyositis
2. Frost bite
3. Chilblains
4. SLE
5. Urticaria.

## REGIONAL CYANOSIS (BLUENESS)

1. One limb–Arterial embolism or phlebothrombosis of a large vein with little collaterals
2. Face and upper extremity–superior vena caval syndrome
3. Differential cyanosis:
   a. Blue feet–PDA with reversal of shunt
   b. Blue hands–Coarctation of aorta with transposition of great vessels
4. Acrocyanosis (see above)
5. Erythrocyanosis (young lady → with short skirts → exposure to cold).

—— o ——

CHAPTER 9

# Blue Sclera

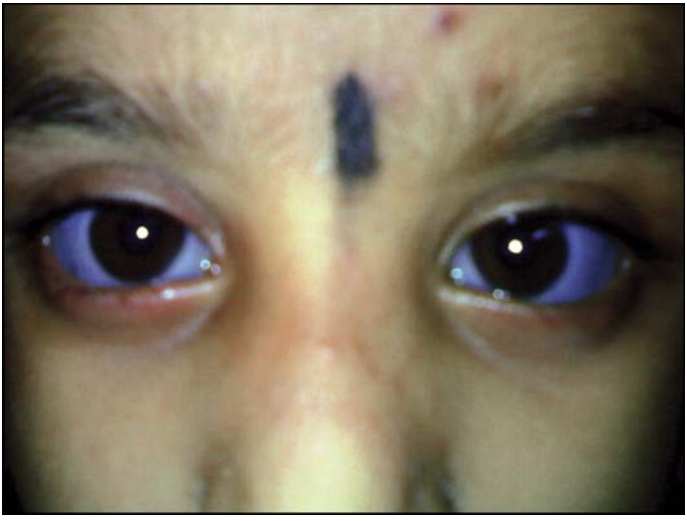


**FIGURE 9.1:** Classical blue sclera in osteogenesis imperfecta

## CLINICAL INVESTIGATION

Search for multiple fractures (generalised osteopenia makes the bone brittle), loose-jointedness and progressive deafness

↓

A case of osteogenesis imperfecta (commonest cause)

↓

Due to thinness of sclerae, the underneath choroid with its vessels gives rise to blue tinge in the eyes

↓

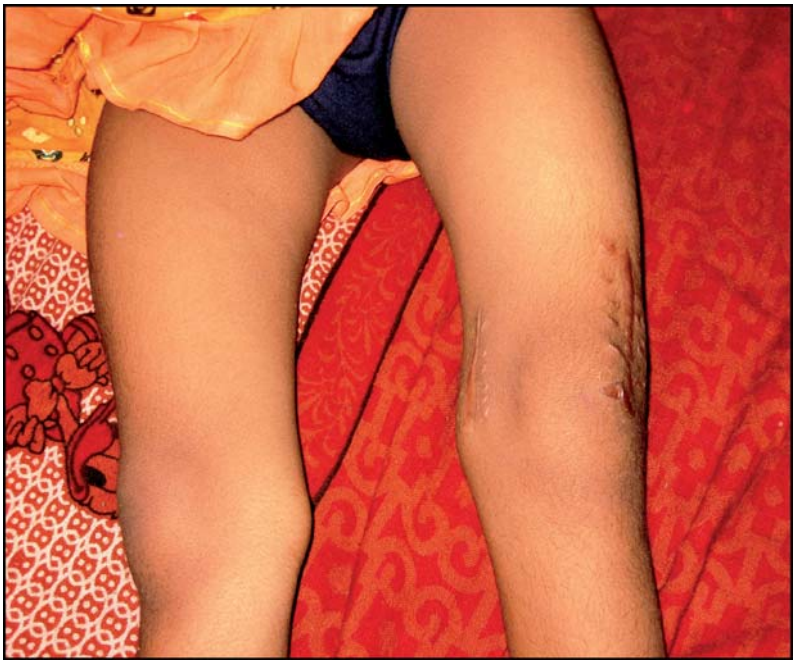


**FIGURE 9.2:** Scar marks for repair of repeated femur fracture in osteogenesis imperfecta

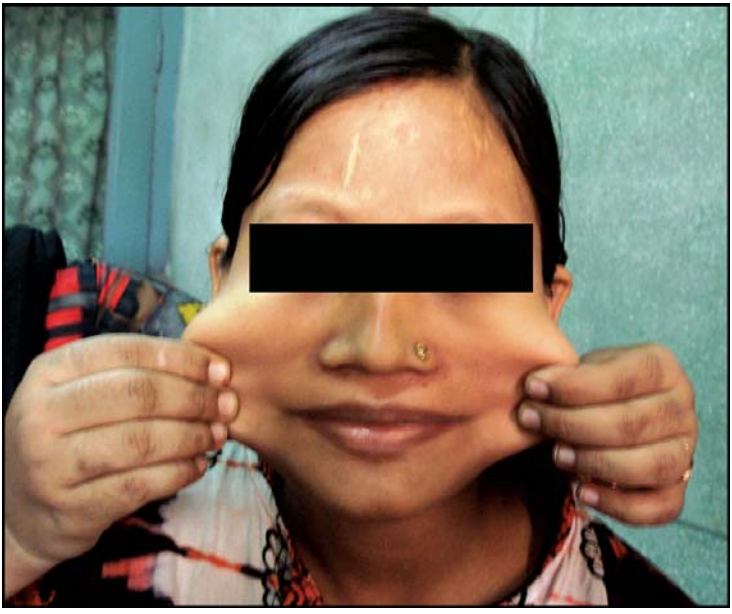


**FIGURE 9.3:** Hyperextensible skin over face in Ehlers-Danlos syndrome

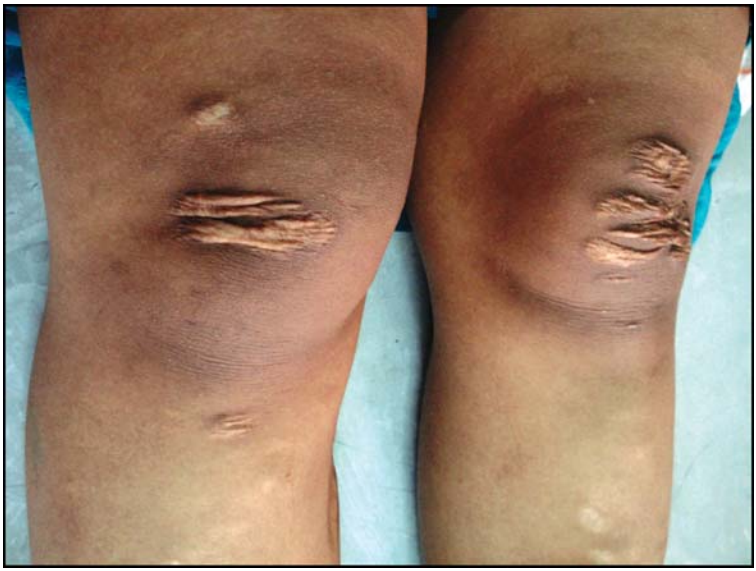


**FIGURE 9.4:** 'Cigarette-paper' scars over skin in Ehlers-Danlos syndrome

Associated features: Pseudoarthrosis, sabre tibia, pectus excavatum or carinatum, blue-yellow teeth with dental abnormalities (dentinogenesis imperfecta), blue tympanic membrane, aortic imcompetence or mitral valve prolapse

↓

Four types (type I, II, III and IV)

↓

X-ray of skull reveals 'Wormian bones' (small irregular bones related to sutures), especially in parietal bones.

## D/D OF BLUE SCLERA

1. Marfan's syndrome
2. Hypophosphatasia
3. Pseudohypoparathyroidism
4. Brittle corneal syndrome
5. Corneal encroachment of sclera
6. Staphyloma
7. Ehlers-Danlos syndrome
8. Pseudoxanthoma elasticum
9. Newborn baby (underlying uveal tissue is visible due to thinness and immaturity of scleral collagen fibres).

## D/D OF BLUISH DISCOLOURATION OF BODY

1. Cyanosis (cyanosed skin blanches on pressure).
2. Carbon monoxide poisoning: cherry-red flush due to formation of carboxyhaemoglobin.
3. Argyria: Deposition of silver salts in the skin due to silver poisoning. The skin does not blanch on pressure.
4. Amiodarone toxicity: The skin may take a bluish hue.

—— o ——

CHAPTER 10

# Bull-neck

## PROLOGUE

The space between the mandibles and the clavicles bulges with enlarged lymph nodes and oedema; commonly from malignant diphtheria.

## CERVICAL LYMPHADENOPATHY

1. Infection, malignancy or ulcer within the oral cavity
2. Infection of ear, eye and nose
3. Scalp infection by louse or dandruff
4. Miliary tuberculosis
5. Lymphoma
6. Lymphatic leukaemia (acute and chronic)
7. Metastasis in lymph nodes
8. Infectious mononucleosis
9. Sarcoidosis
10. Rubella infection.

## LYMPHADENOPATHY WITH SINUS FORMATION

1. Tuberculosis
2. Malignancy
3. Actinomycosis.

## D/D OF BULL-NECK

1. Surgical emphysema (press the stethoscope over the swelling and listen the crepitus).
2. Enlarged submandibular glands (Mikulicz's syndrome).
3. Ludwig's angina.

## AXILLARY LYMPHADENOPATHY

1. Infection of the upper extremity
2. Breast carcinoma
3. Lymphoma
4. Miliary tuberculosis
5. Brucellosis
6. Cat-scratch disease.

## EPITROCHLEAR LYMPHADENOPATHY

1. Hand infections
2. Lymphoma, especially NHL
3. Sarcoidosis
4. Secondary syphilis
5. Tularaemia
6. Infectious mononucleosis.

## INGUINAL LYMPHADENOPATHY

1. Infection or cellulitis of the lower limb
2. Filarisis
3. Syphilis
4. Metastasis from genital malignancy, pelvic carcinoma
5. Chancroid
6. Lymphoma
7. Miliary tuberculosis
8. Lymphatic leukaemia (acute and chronic)

* The importance of epitrochlear lymphadenopathy is highest to a clinician in comparison to inguinal lymphadenopathy, which rates lowest.

## MATTING OF LYMPH NODES

- Signifies periadenitis
- Most commonly in tuberculosis; may be seen in chronic lymphadenitis, NHL

## CONSISTENCY OF LYMPH NODE SWELLING

Stony hard → malignancy
Elastic and rubbery → Hodgkin's disease
Variegated → NHL

Firm and shotty → secondary syphilis
Soft and fluctuating → cold abscess, inflammatory abscess.

## PATHOLOGICAL SIGNIFICANCE OF ENLARGED NODES

- Nodes are pathologically significant, if:
  - > 1cm in diameter
  - Firm in consistency
  - Tender on palpation
  - Matted
- Following factors should always be kept in mind in assessing the pathological nature of nodes:
  - Age of the patient
  - The physical characteristics (described above)
  - Anatomical sites involved
  - The total clinical setting.

## VIRCHOW'S NODE

- Also known as Ewald's node or sentinel node
- Position:
  Medial group of left sided supraclavicular nodes which lie in between two heads of left sternomastoid muscle; always palpated from back of the patient.
- Receive lymphatics from:
  - Upper limb (left)
  - Breast (left)
  - Lung (left)
  - Stomach (Troisier's sign)
  - Testes
- Bronchogenic carcinoma commonly produces 'scalene node' enlargement.

—— o ——

CHAPTER 11

# Bruxism (Teeth Grinding)

## WHAT IS BRUXISM ?

It is an involuntary and forceful teeth grinding during sleep. The classical age of onset is 17-20 years though this may appear in childhood, and may affect 10-20% of the population.

The person is usually unaware of the problem; M:F = 1:1, and the problem usually remits by the age 40. Dental examination may give a clue to diagnosis where the damage is minor. Night time bed partner primarily notices the situation.

Usually no treatment is necessary but in severe cases, a rubber tooth guard is required to prevent disfiguring injury of teeth. Stress management, psychotherapy or benzodiazepines may be of some help. Contrary to common belief, bruxism has no relation with intestinal infestation with helminths.

'Teeth chattering' is usually associated with fever with chill and rigor, or with shivering due to exposure to very cold weather.

## SLEEP DISORDERS

1. Insomnia (most common complaint in general population and mental disorders).
2. Sleep-walking (somnambulism).
3. Sleep terrors (occurs during first several hours after onset of sleep).
4. Sleep-related epileptic seizures.
5. Bruxism.
6. Clusture headache (a variety of migraine).
7. Abnormal swallowing, coughing, chocking and aspiration of saliva.

8. Sleep-related gastroesophageal reflux disease (GERD)–often associated with hiatal hernia.
9. Nocturnal angina.
10. Sleep enuresis (bedwetting).
11. Sleep-talking.
12. Nocturnal leg cramps.
13. Nocturnal paroxysmal dystonia.
14. Paroxysmal nocturnal haemoglobinuria (sleep related acidic reaction of the blood produces haemolysis, making the morning urine brownish red).
15. Sleep apnoea (respiratory dysfunction during sleep disturbing nocturnal sleep with excessive daytime somnolence).
16. Restless leg syndrome (or Ekbom's syndrome; causes are idiopathic, peripheral neuropathy, iron deficiency, uraemia).
17. Periodic limb movement disorder (previously known as nocturnal myoclonus).
18. Sleep paralysis (totally unable to perform a voluntary movement despite remaining alert and aware).
19. Narcolepsy (periods of irresistible sleep) may coexist with cataplexy (sudden loss of lower limbs tone and falling with full awareness)–often accompanied by hypnagogic hallucinations (on falling asleep).

## EXCESSIVE DAYTIME SLEEPINESS (HYPERSOMNIA)

1. Chronic sleep disruption at night (insomnia). Inadequate night-time sleep due to fatigue/excessive consumption of caffeine or alcohol are very common causes of daytime sleepiness.
2. Narcolepsy (recurrent bouts of irresistible sleep).
3. Obstructive sleep apnoea syndrome.
4. Sleeping sickness (African trypanosomiasis by T. brucei complex).
5. Depressive illness.
6. Following infections, e.g. infectious mononucleosis.
7. Symptomatic hypersomnia–organic brain diseases, e.g. encephalitis, toxic or metabolic encephalopathies; tumour, vascular or traumatic brain damage.
8. Neurotic hypersomnia–in hysterics or neuroasthenic individuals .
9. Drug-induced–sedatives, antiepileptics.
10. Kleine-Levin syndrome–episodic hypersomnolence and hyperphagia in adolescent boys; rare.
11. Idiopathic hypersomnia–hereditary with excessive night-time sleep; also known as 'sleep drunkenness'.

## INSOMNIA

It is the 'difficulty in sleeping' and in general 1/3rd of adults suffer from this ailment. It may be categorized by:

1. Early insomnia, i.e. difficulty in getting off to sleep (commonly due to anxiety, depression, mania and substance abuse).
2. Middle insomnia, i.e. waking up from sleep at mid-night (mostly due to sleep apnoea, polyuria or nocturia, and benign hypertrophy of prostate).
3. Late insomnia, i.e. early morning waking (commonly due to depression and malnutrition).

* Hypocretin (orexin) neuropeptides are involved in sleep-waking cycle (present between cerebral cortex and reticular formation).

** Electrophysiologic parameters of sleep are recorded by 'polysomnography' i.e. electroencephalogram (EEG) + electrooculogram (EOG → records eye-movements activity) + electromyogram (EMG → measured on chin and neck muscles).

—— o ——

CHAPTER 12

# Claw Foot

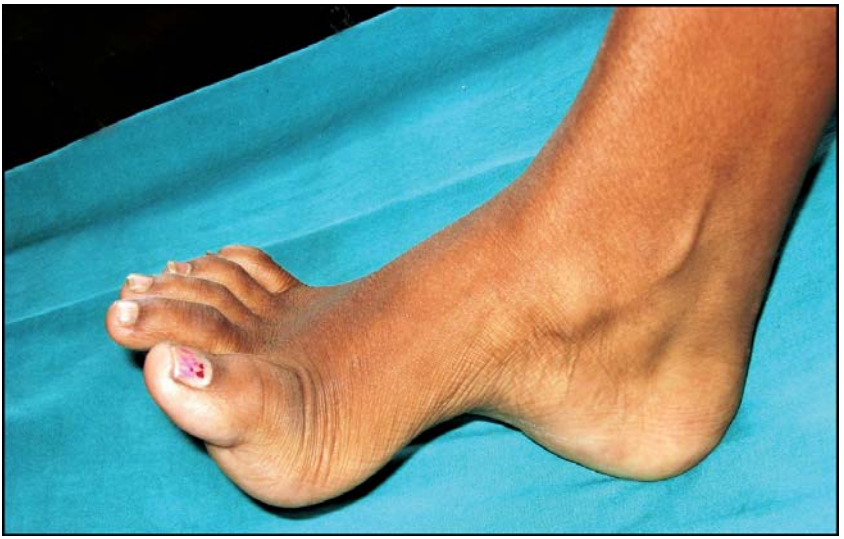


**FIGURE 12.1:** Pes cavus (claw foot or high-arched foot) due to wasting of intrinsic muscles of foot in peroneal muscular atrophy

## SYNONYM

Pes cavus

## DEFINITION

A fixed deformity of foot where both feet are more or less symmetrically high-arched, i.e. there is gross exaggerbation of the medial longitudinal arch of the feet. It is just opposite to flat foot (pes planus) deformity.

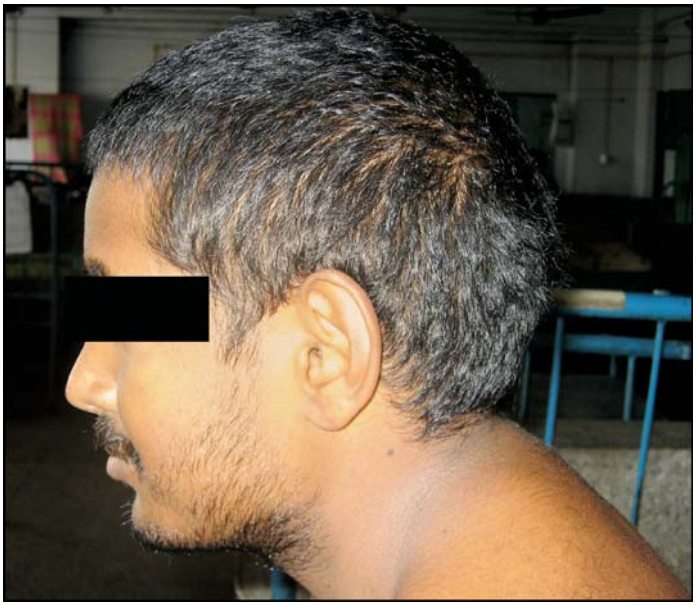


**FIGURE 12.2:** Short neck and low hairline in cranio-vertebral anomaly (patient had quadruplegia)

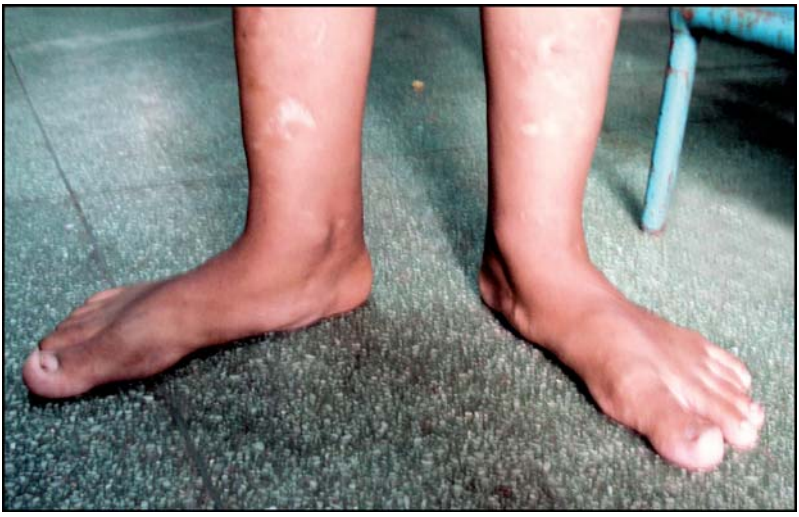


**FIGURE 12.3:** Pes planus (flat foot)

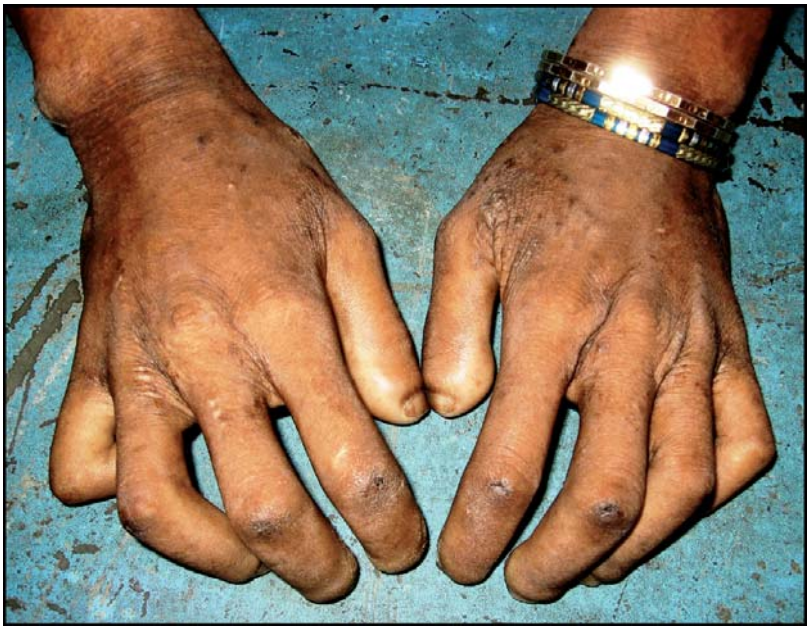


**FIGURE 12.4:** Mutilated hands with pulp atrophy, sclerodactyly, limitation of full extension of fingers with dry, coarse skin in scleroderma

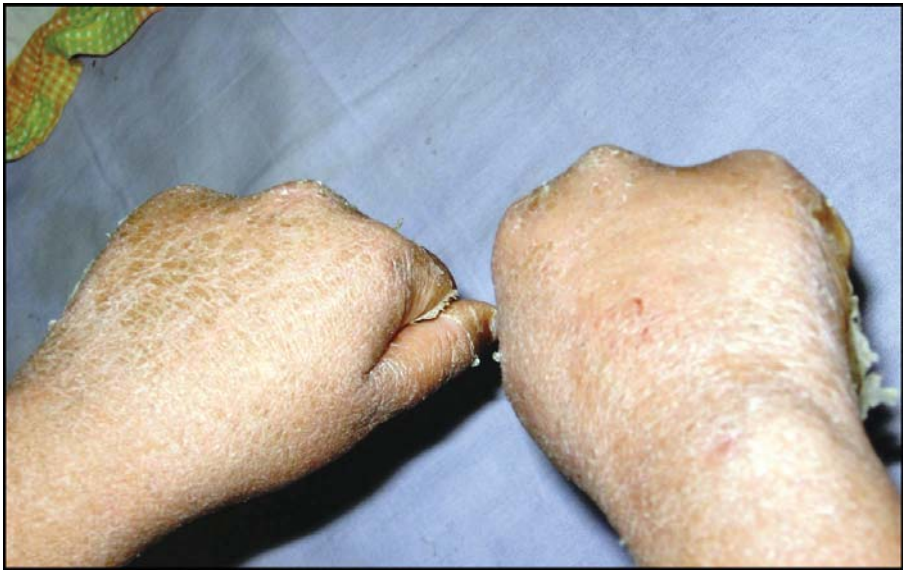


**FIGURE 12.5:** Short 4th metacarpals in pseudohypoparathyroidism–'knuckle-knuckle dimple-dimple syndrome' (the patient had peeling of skin of hands due to some drug reaction

## CONDITIONS ASSOCIATED

- Familial
- Friedreich's ataxia
- Peroneal muscular atrophy
- Spina bifida occulta
- Poliomyelitis
- Syringomyelia
- Cerebral palsy
- Refsum's disease
- Idiopathic

## METHOD OF DEMONSTRATION

Take a foot-print on a white paper after painting the feet by lac-dye, or after immersing the feet in water, ask the patient to walk barefooted in the floor → observe the foot-prints.

## GENU VARUM DEFORMITY

'Bow legs' are seen in:

- Rickets or osteomalacia
- Achondroplasia
- Paget's disease

## GENU VALGUM DEFORMITY

'Knock knees' are seen in:

- Rickets
- Congenital deformity

## SKELETAL DEFORMITIES PRESENT WITH NEUROLOGICAL DISORDERS

1. Kyphoscoliosis
2. Pes cavus
3. High-arched palate
4. Short neck (e.g. cranio-vertebral anomaly)
5. Various skull deformities, e.g. craniostenosis.

## HEEL PAD THICKNESS

A lateral view of patient's foot is taken by X-ray. The distance between the lower most point of calcaneum and the lower most point of the soft tissue shadow of heel is measured → 'heel pad thickness'.

## ↑ HEEL PAD THICKNESS

- > 18 mm in women and > 21 mm in men
- Conditions associated:
  1. Acromegaly (commonest cause)
  2. Obesity
  3. Anasarca

## DEFORMED OR MUTILATED FINGERS/TOES

- Leprosy
- Scleroderma
- Buerger's disease
- Congenital defects
- Diabetic foot
- Frost bite
- Syringomyelia
- Vasculitis
- Atherothrombosis
- Arthritis mutilans (psoriasis)
- Trauma
- Porphyria
- Amyloid neuropathy
- Lesch-Nyhan syndrome

## SHORT FOURTH METACARPALS

- Pseudohypoparathyroidism
- Down's syndrome
- Myositis ossificans congenita

* As the knuckles and dimples in closed fist of both hands are not symmetrical, presence of short fourth metacarpals is also known as knuckle-knuckle dimple-dimple syndrome (especially in pseudohypoparathyroidism).

—— o ——

CHAPTER 13

# Coprolalia

## DEFINITION

Offensive utterance of obscene words.

## ASSOCIATIONS

1. Poisoning (e.g. organophosphorous)
2. Atropinisation or overdose of atropine
3. Hepatic pre-coma
4. Subdural haematoma
5. Uraemia
6. Tourette syndrome
7. Encephalitis or encephalopathy
8. Habitual (personality disorder).

## GILLES DE LA TOURETTE SYNDROME

Inherited neuropsychiatric disorder characterised by multiple motor (e.g. blinking, grimacing, head jerking) and vocal (e.g. clearing the throat or coprolalia) tics. Compulsive utterances of coprolalia is one of the most recognizable and distressing symptom, which appears a few years after disease onset. The disease starts in childhood or adolescence; hyperactive, non-specific ECG abnormalities are seen in 50%; the cause is probably an inherited disorder of synaptic transmission.

Treatment is done by haloperidol (drug of choice), clonidine, clonazepam, pimozide or tetrabenazine.

## SPEECH DISORDER

It is the symbolic expression of thought process in spoken or written words.

- Aphasia or dysphasia–unable to speak due to defect in higher centre (e.g. Broca's or Wernicke's area in brain) with difficulty in language function
- Dysarthria–defect in articulation, commonly due to neuromuscular or muscular disorders resulting in impaired coordination of facio-lingual muscles, e.g. slurring, stammering or mumbling
- Dysphonia–disorder of phonation where the defect lies in the vocal cord, e.g. hoarse voice, voice loss
- Echolalia–repetition of examiner's word
- Palilalia–repetition of terminal words of own speech
- Jargon aphasia–neologisms (new words) making no sense at all
- Perseveration–repeated use of particular words or phrases

* Last 4 are part of speech disorder due to defect in higher centres (e.g. cerebrovascular accidents).

—— o ——

CHAPTER 14

# Cough

## DEFINITION

It is the reflex or voluntary expulsion of the inspired air by forced expiratory effort against a transitorily closed glottis. Cough is known to be one of the unique defensive reflex of human body designed to clear the tracheobronchial tree of secretion and foreign body. Though previously recognised as the 'watch-dog' (i.e. protective reflex) of the respiratory system, it may be regarded as a manifestation of diseases affecting many other systems including a major symptom of pulmonary diseases.

## MECHANISM

It requires an intricate network of neurosensory-muscular coordination:

a. Afferent pathway: it originates from the sensory receptors present in epithelium of the airways (larynx, trachea and major bronchus), and the afferent nerves involved are trigeminal, glossopharyngeal, superior laryngeal and vagus.
b. Centre: cough centre is present in medulla oblongata.
c. Efferent pathway: The efferent impulses reach the diaphragm, intercostal muscles, abdominal muscles, and to larynx through vagus, phrenic, recurrent laryngeal and spinal motor nerves which result in cough, while the laryngeal air velocities produced as a result of violent action of respiratory muscles make a sound.

Cough is usually associated with mucus secretion, bronchoconstriction and transient rise in systemic BP.

## THREE PHASES OF COUGH

1. Appropriate stimulus, i.e. mechanical (dust, mucus, foreign body), chemical (toxic gases, fumes, cigarette smoke), inflammatory (oedema and hyperaemia of respiratory mucous membrane) and thermal stimuli (inhalation of either very cold or hot) initiate deep inspiration.
2. Glottic closure + contraction of muscles of expiration including accessory muscles + relaxation of diaphragm → resulting in maximum intrathoracic pressure → narrowing of trachea.
3. Opening of glottis → high flow rate generated as a result of pressure diffenence between the airways and the atmosphere associated with tracheal narrowing→ propels excessive mucus and foreign body outside.

## AETIOLOGY

A. Respiratory tract–Acute and chronic infection/neoplasm of larynx or pharynx, post-nasal drip, acute tracheobronchitis, cigarette smoking, pulmonary tuberculosis, bronchial asthma, chronic bronchitis, bronchiectasis, emphysema, interstitial pulmonary fibrosis, pneumonia, lung abscess, bronchogenic carcinoma, sarcoidosis, tropical eosinophilia, pneumoconiosis, cystic fibrosis, cough variant asthma, pleurisy, pleural effusion, mediastinal mass.
B. System of ear, nose and throat–Inhalation of toxic gases, fumes, cooking fuels, dust or foreign body; otitis media, wax impacted in external ear, laryngitis, tracheitis, allergic rhinitis, sinusitis (maxillary) and post-nasal drip.
C. Cardiovascular system–Pulmonary oedema, pericardial effusion, aortic aneurysm, enlarged left atrium (from mitral stenosis commonly), left ventricular failure.
D. GI tract–Gastroesophageal reflux disease (GERD), hiatus hernia, achalasia cardia, oesophageal diverticulum.
E. Reflex–Happens to be due to irritation of vagus nerve and is commonly from wax impacted in external ear, otitis media, subdiaphragmatic abscess and acute distension of stomach.
F. Drug-induced–ACE-inhibitors (due to accumulation of bradykinin, substance P and prostaglandin $E_2$), β-blockers (indirect effect of bronchoconstriction).
G. Psychogenic–Found in adolescents and self-conscious adults; usually smasmodic and explosive in nature; often it is barking (or 'honking') and loud in character → may be seen as a part of obsessional neurosis or coordinated tics.
H. Idiopathic

## CLASSIFICATION

1. Acute (< 3 weeks ) or chronic (> 3 weeks).
2. Dry (upper respiratory tract infection, smoker's cough, early stage of pulmonary tuberculosis) or wet (e.g. bronchiectasis).
3. Paroxysmal cough (usually lasts for 1-2 minutes): Whooping cough, tracheal obstruction, bronchial asthma, pulmonary oedema, foreign body inhalation.

## TYPES

1. Dry or non-productive–Acute laryngotracheobronchitis; acute dry pleurisy, smoker's cough, early stage of pulmonary tuberculosis.
2. Wet, productive or moist–Sputum production may be due to lung abscess, bronchiectasis, resolution stage of pneumonia.
3. Bovine (laryngeal cough)–The explosive nature of cough is lost in recurrent laryngeal nerve palsy (commonly due to bronchogenic carcinoma).
4. Brassy (or metallic)—Dry cough with a metallic sound may be heard in carcinoma of larynx.
5. Whooping–There is rapid succession of dry cough which gradually gather speed and end in a deep inspiration when the characteristic 'whoop' is audible; found in pertussis.
6. Spluttering cough–In tracheo-oesophageal fistula (cough during swallowing).
7. Hacking (pharyngeal cough)–Dry and irritable cough in heavy smokers.
8. Barking–Harsh and loud cough; found in epiglottitis and hysteria.
9. Nocturnal–chronic bronchitis, LVF, tropical eosinophilia, post-nasal drip, aspiration (e.g. from GERD).
10. Nagging cough–Commonly after use of ACE-inhibitors.
11. 'Croupy' cough–laryngitis, especially in children.
12. Foetid cough–In bronchiectasis and lung abscess, there is cough with foul smelling expectoration.
13. Suppressed cough–Short spell of suppressed cough is found in pleurisy to avoid pain during coughing.

## FACTORS INFLUENCING COUGH

1. Cough started acutely–Foreign body aspiration, pulmonary thromboembolism (PTE), pulmonary oedema, acute exacerbation of bronchial asthma, inhalation of fumes.

2. Cough with wheeze–Bronchial asthma, chronic bronchitis, pulmonary thromboembolism (PTE).
3. Related to meals–Hiatus hernia, oesopheageal diverticulum, tracheo-oesophageal fistula, neurogenic dysphagia (e.g. bulbar palsy).
4. Related to exertion–Early left ventricular decompensation, mitral stenosis, bronchial asthma.
5. Related to posture–Bronchiectasis and lung abscess (cough evoked after change of posture in bed), GERD (cough evoked while lying horizontally).
6. Related to seasonal variation–Bronchial asthma and chronic bronchitis become worse in winter.
7. Related to working hours–Byssinosis (triggered by cotton dust).
8. Induced after inhalation of cold air–bronchial asthma.
9. Predominantly nocturnal cough–tropical eosinophilia, bronchial asthma, LVF, oesophageal disorders.
10. Predominantly early morning cough–Post-nasal drip, chronic bronchitis, sinusitis.
11. Recurrent cough since childhood–Cystic fibrosis, childhood asthma, congenital heart disease, cystic disease of lung, hypogammaglobulinaemia.

## HOW SPUTUM OR EXPECTORATION IS ANALYSED?

1. Amount (profuse or not).
2. Character (serous, mucoid, purulent, mucopurulent).
3. Colour (yellow, green, black, pinkish, rusty).
4. Odour or taste (offensive or not).
5. Mixed with blood (haemoptysis) or not.
6. Sputum production influenced by change of posture (bronchiectasis, lung abscess) or not.

* Profuse sputum means approximately a teacupful (> 100 ml) of sputum production per day.

## PROFUSE AND FOETID (FOUL-SMELLING) SPUTUM

- Bronchiectasis
- Lung abscess
- Infection with anaerobic organism
- Infected cavity or neoplasm
- Empyaema thoracis ruptured into bronchus

## DIFFERENT COLOURS OF SPUTUM

1. Red–Haemoptysis.
2. Black–Carbon particles from atmosphere, cough in coal-miners (benign in nature).
3. Green–Respiratory tract infection (verdoperoxidase from dead neutrophil turns yellow sputum to green), bronchial asthma (due to large number of eosinophils), pseudomonas infection.
4. Pink and frothy in acute pulmonary oedema.
5. Rusty or golden yellow–Pneumonia (pneumococcal).
6. Yellow–Respiratory tract infection (creamy).
7. Yellow (with sulphur granules)–Actinomycosis of lung.
8. Brown to red + tenacious–Klebsiella pneumoniae infection.
9. Anchovy-sauce like–Amoebic liver abscess ruptured into lung.
10. Mucoid–Chronic bronchitis, COPD, chronic bronchial asthma.
11. Frothy–Pulmonary oedema, bronchoalveolar carcinoma (serous and frothy).

## HOW COUGH IS ANALYSED AT THE BEDSIDE?

1. Duration (days/months/years); acute < 3 weeks, chronic > 3 weeks.
2. Variability (daytime/nocturnal/morning).
3. Precipitating factors (dust/fumes/pollen/cold air/lying down).
4. Seasonal variation? wheeze?
5. Types (dry/wet/bovine).
6. Haemoptysis, present or not.
7. Change of character of cough in a chronic heavy smoker (e.g. development of COPD or bronchogenic carcinoma).
8. Associated symptoms (port-nasal drip, GERD, occult asthma, fever, dyspnoea).
9. Chest pain (pleurisy) or breathlessness (COPD, pneumonia).
10 H/O drug intake–ACE-inhibitors or β-blockers.

## EVALUATION OF CHRONIC COUGH

Common associations are:

1. Viral infection
2. GERD
3. Post-nasal drip
4. Cough variant asthma (present with cough in the absence of wheezing or breathlessness)
5. ACE-inhibitor induced.

## POSSIBLE INVESTIGATIONS IN CHRONIC COUGH

1. Chest X-ray (PA and oblique view).
2. ENT check-up.
3. PNS X-ray and CT scan of sinus for post-nasal drip.
4. Lung function tests and histamine bronchial provocation testing for cough variant asthma.
5. CT scan of thorax (to exclude interstitial lung disease).
6. Radionuclide ventilation/perfusion ($V_A/Q$) scan for recurrent PTE.
7. Fibreoptic bronchoscopy–to rule out inhaled foreign body or carcinoma of the bronchus.
8. Ambulatory oesophageal pH monitoring for GERD.
9. ECG, echocardiography (to exclude valvular lesion or LV dysfunction).

## COMPLICATIONS

1. Chest and abdominal wall soreness form harassing cough, exhaustion, loss of sleep.
2. Severe vomiting, rib fracture (especially in elderly with osteoporosis, multiple myeloma or osteolytic metastases), cough syncope (momentary unconsciousness due to raised intrathoracic pressure during coughing which impedes venous return to the heart and reduces cardiac output), spontaneous pneumothorax, subconjunctival haemorrhage, frenal ulcer (in tongue), urinary or faecal incontinence, prolapse of rectum/uterus/ hernia.
3. Postoperative wound dehiscence, rupture of rectus abdominis (rare), heart block (rare).

## MANAGEMENT

### *Non-specific*

1. Antitussive agents:
   a. Antihistaminics–chlorpheniramine, clemastine, cetrizine.
   b. Sympathomimetic decongestants–pseudoephedrine, oxymetazoline, phenylephrine.
   c. Demulcents–menthol, tea with honey.
   d. Others–narcotic and non-narcotic analgesics, bronchodilators in asthma.
2. Protussive agents (or cough enhancing agent)
   a. Expectorants–guaiphenesin.

b. Mucolytics–bromhexine, carbocysteine, acetyl cysteine, dornase alpha, ambroxol (a metabolite of bromhexine).
c. Hypertonic saline aerosol (for chronic bronchitis).

### *Specific*

Cessation of smoking, application of antibiotics, stoppage of ACE-inhibitors, treatment of LVF.

—— o ——

CHAPTER 15

# Cramp

## DEFINITION

It is the painful, involuntary contractions of a single or a group of muscles. Cramps are seen in normal healthy persons or may be precipitated by voluntary movements. Cramps in the calf muscles are so common as to be regarded normal, but a more generalised cramps may be a sign of chronic disease of motor neurone.

## AETIOPATHOGENESIS

Not truely known but may be due to:

- Overactivity of muscle/nerve membranes
- Electrolyte imbalance

Pain associated with cramp is possibly due to:

- Focal ischaemia
- Accumulation of metabolites within the muscles

* Pathological cramps may be due to an abnormality anywhere in the pathway including anterior horn cells, peripheral nerve, neuromuscular junction and muscle membrane.

## POSSIBLE CAUSES

1. Normally occurs in healthy individual at night.
2. Electrolyte imbalance (↓Ca, ↓Mg, ↓Na, ↓K, ↓Po4).
3. Overexertion (producing dehydration by profuse sweating, heat cramps, miner's cramp, swimmer's cramps, leg cramps in long distance bus drivers).
4. Motor neurone disease, e.g. amyotrophic lateral sclerosis.

5. Metabolic: chronic renal failure, hypothyroidism, McArdle's disease, phosphofructokinase deficiency, haemodialysis.
6. Peripheral neuropathy: Diabetes mellitus, alcohol or vincristine-induced.
7. Cramps in professionals: writers, tailors, typists.
8. Muscular dystrophies: X-linked variety, myotonia.
9. Others: chronic wasting disease (e.g. tuberculosis), pregnancy, dehydration due to any cause, tetany, peripheral arterial disease, deep vein thrombosis.

* Muscle 'spasm' may occur in tetanus, tetany, epilepsy, myoclonus, etc.

## NOCTURNAL LEG CRAMPS

Majority of healthy persons at sometime or other has had experienced muscle cramp. Usually it occurs at night, especially when the feet are cold, after a day of unusually strenous activity. Muscles of calf and foot are commonly affected. The onset is sudden and may awaken the person. The muscle is visibly and palpably taut, as well as painful. Massage and vigorous stretch of the cramped muscle may give relief. Visible fasciculations may precede and follow muscle cramp. This type of nocturnal cramps are nortorious for recurrence.

All causes mentioned above are responsible for nocturnal cramps with a special reference to:

- Idiopathic
- Electrolyte abnormalities
- Deep vein thrombosis
- Hypoglycaemia
- Diabetic neuropathy
- Peripheral vascular insufficiency
- Alcohol use

## DIFFERENTIAL DIAGNOSIS (CRAMP)

The single most important condition to be differentiated is intermittent claudication.

## POST-EXERCISE LEG PAIN

Think of —

- Atherosclerosis obliterans
- Buerger's disease
- Deep vein thrombosis

- Lumbar canal stenosis (i.e. neurogenic)
- Venous claudication
- Popliteal cyst
- McArdle's disease (muscle phosphorylase deficiency)
- Popliteal artery entrapment syndrome
- Tetany (↓Ca, ↓Mg or alkalosis)

## CALF PAIN (COMMON CAUSES)

- Intermittent claudication (needs meticulous clinical examination)
- Deep vein thrombosis (do Doppler studies)
- Rupture of Baker's cyst (perform arthrography of knee joint)
- Referred pain from lumbar spine (MRI scan of lumbar cord to demonstrate compression)
- Spastic calf muscle (diagnosed by clinical examination)

## CALF SWELLING (UNILATERAL)

This may be due to local inflammation, or obstruction/damage to a vein or lymph channel. The possibilities are:

1. Deep vein thrombosis (↓ flow on Doppler ultrasound scan, filling defect in venogram).
2. Cellulitis (↑ leucocytes, response to antibiotics).
3. Ruptured Baker's cyst (usually suffering from rheumatoid arthritis. Arthrogram reveals leakage of contrast from joint capsule).
4. Traumatic (may have tender haematoma formation).
5. Filariasis or abnormal lymphatic drainage (e.g. trypanosomiasis in tropics, or pelvic malignancy)–obstruction revealed in lymphangiogram.

## TETANY

It is increased neuromuscular irritability due to decrease in the concentation of free calcium ion in the plasma. This special variety of paroxysmal cramps occur mainly in the extremities. Normal serum calcium level in 9-11 mg/dl and the ionic fraction is 4.5-5.6 mg/dl.

The common symptoms are:

- Irritability
- Muscle cramp (carpopedal spasm)
- Peripheral paraesthesia
- Triad of symptoms in children, i.e. carpopedal spasm, laryngismus stridulus (stridor, respiratory distress, cyanosis), and convulsions
- Dysphagia, dyspnoea, dysuria, abdominal colic

## PHYSICAL SIGNS IN TETANY

1. Trousseau's sign–When the pressure is raised above the systolic BP for 2-3 minutes, typical carpal spasm occurs in hands within 3 minutes. There is flexion of MCP joints with extension of interphalangeal joints, and the flexed thumb takes its position in between the index and the middle finger (opposition of thumb). This is known as "main d' accoucheur" or obstetrician's hand. Pedal spasm is less frequently demonstrated. Latent tetany is best demonstrated by this sign.
2. Chvostek's sign–Tapping of the facial nerve (by finger or hammer) in front of the ear will produce twitching of facial muscles.
3. Erb's sign–Muscular contractions can be produced by application of subthreshold electrical stimulation (0.5-2.0 milli-amps).
4. Peroneal sign–Tapping of peroneal nerve at the neck of fibula will produce pedal spasm, i.e. plantiflexion and adduction of the foot, while the knee is extended.
5. [ECG–Prolonged Q-T interval].

* All are features of ↑ neuromuscular irritability. Tetany is due to 'unstable depolarisation' of the distal segments of the motor nerves. Carpopedal spasm may uncommonly spread over face, neck and trunk muscles (except the eye muscles). Psychoneurotic patients may have tetany during hyperventilation. Hyperventilation and ischaemia of muscles ↑ the tendency of carpopedal spasm.

## TREATMENT OF CRAMPS

1. Massaging, and passive and vigorous stretching of the affected muscles.
2. Tetany: Inj. calcium gluconate (10% solution) 10 ml by IV route, slowly (inj. calcium chloride may be given). If tetany is not controlled by calcium, administration of magnesium may be necessary.
3. Tab quinine sulphate–300-600 mg three times daily.
4. Tab procainamide–250-500 mg three times daily.
5. Tab diphenhydramine hydrochloride–50 mg at bedtime.
6. Carbamazepine, clonazepam, muscles relaxants (e.g. tizanidine) may be of some help.
7. Idiopathic nocturnal cramp may be alleviated by tocopherol, 400 mg twice daily; carnitine may be of some help.

—— o ——

CHAPTER 16

# Depressed Bridge of the Nose

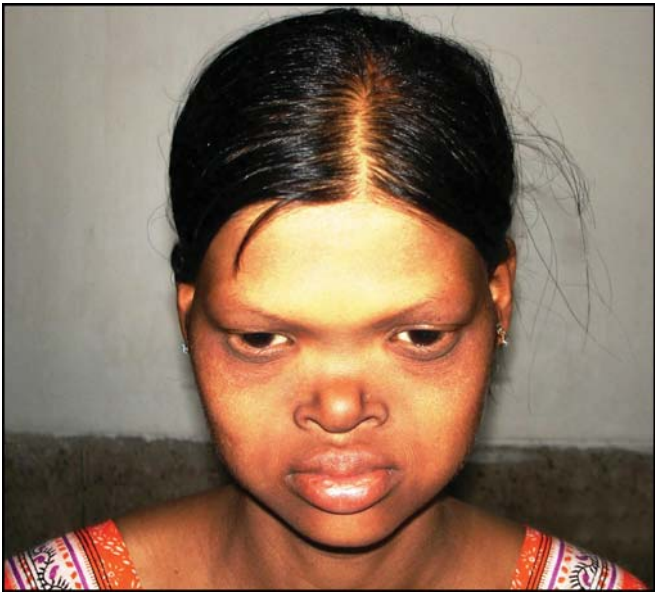


**FIGURE 16.1:** Typical saddle-nose deformity in a patient with ectodermal dysplasia

## POSSIBILITIES

1. Lepromatous leprosy (thick and coarse skin of face, especially the infiltrated earlobes, madarosis, occasional perforated nasal septum, deeper lines of face, bronzed hyperpigmentation, leonine face).
2. Thalassaemia major (frontal bossing, hypertelorism, mongoloid slant of eyes, malar prominence, dental malocclusion, icterus and pallor → the 'chipmunk' facies).

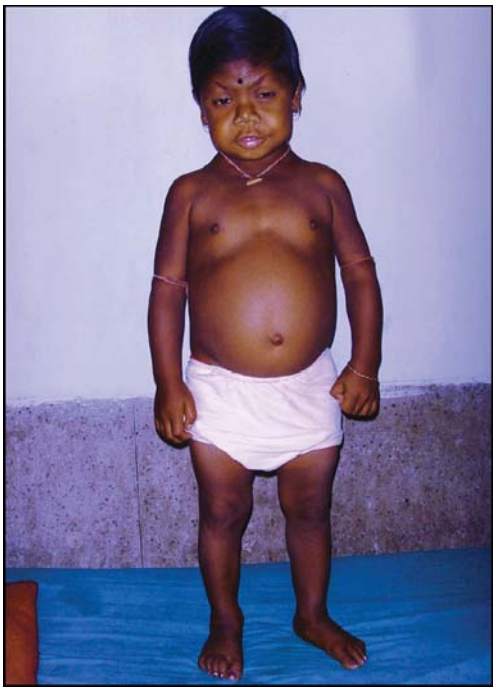


**FIGURE 16.2:** Short stature (cretinism)

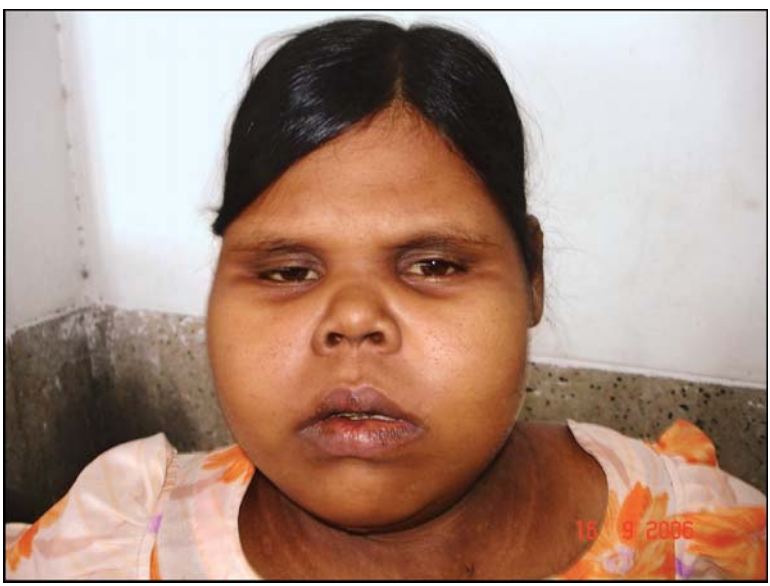



**FIGURE 16.3:** A rare patient of congenital syphilis having saddle nose with depressed bridge of the nose and peg-shaped upper central incisors (part of Hutchinson's teeth)

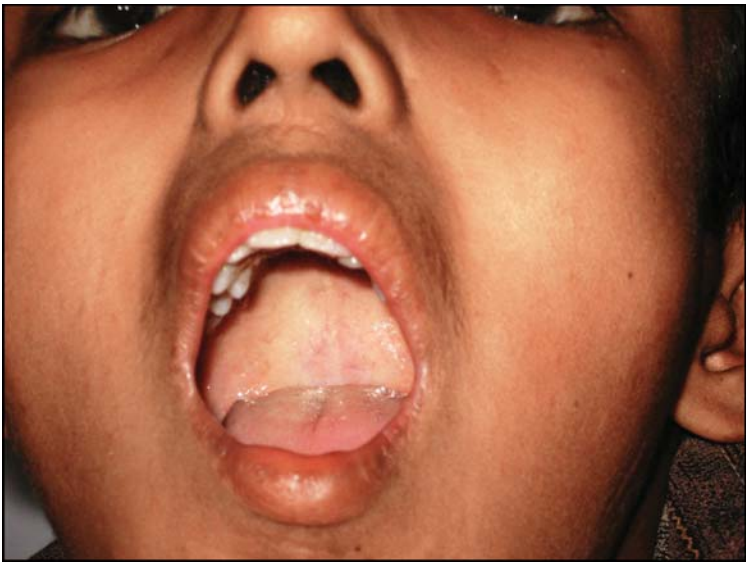


**FIGURE 16.4:** High arched palate in a suspected case of Marfan's syndrome

3. Down's syndrome (microcephaly, upwards slanting of eyes with epicanthic folds, low set ears, hyperlelorism, high arched palate, large and fissured longue with an idiotic look → mongol facies).
4. Cretinism (broad-flat nose with big nostrils, hypertelorism, thick-everted lower lip with macroglossia, sparse and dry skin, dull and idiotic look).
5. Congenital syphilis (frontal bossing, small and broad nose, poorly developed maxilla, ground-glass cornea, Hutchinson's teeth, 'Mulberry' molars [sixth-years molars have multiple poorly developed cusps instead of four] and rhagades at the angle of mouth).
6. Ectodermal dysplasia (frontal bossing, small and broad nose, fine wrinkling in skin of face, sparse and dry hair, poor dentition with conical and pointed tip of teeth, poorly developed maxilla and often there is absence of sweating).
7. Wegener's granulomatosis (purulent or bloody nasal discharge, small and broad nose may result from nasal septal perforation).
8. Midline granuloma (small and broad nose, nasal discharge with septal perforation, perforation of soft and hard palate, conjunctival inflammation or ulceration, and loosening of teeth).
9. Sarcoidosis (lupus pernio in the skin of face [deeper nodules and plaques], conjunctival nodules, uveitis, dry eye with destruction of nasal bone may be present).

10. Others: Blastomycosis, yaws, carcinoma of the nose or nasopharynx, vasculitis.

## MECHANISMS RESPONSIBLE

- Due to hyperplasia of lesser wing of sphenoid bone in thalassaemia
- In others, it is due to chronic granulomatous destruction of anterior nasal septum.

## SADDLE-NOSE DEFORMITY

1. Small and broad nose, plus.
2. Big nostrils, plus.
3. Depressed bridge of the nose.

* Classically found in 1, 5, 6, 7, 8 of above mentioned possibilities; also seen after overzealous septal excision operation or in polychondritis

* 'Destruction of nasal structures' are seen in 1, 7, 8 and lupus vulgaris.

## FRONTAL BOSSING OF SKULL (PROMINENT FOREHEAD)

- Thalassaemia major
- Rickets
- Hydrocephalus
- Congenital syphilis
- Ectodermal dysplasia
- Acromegaly
- Achondroplasia

## HIGH ARCHED PALATE

*(Arbitrarily diagnosed when the roof of the palate is not visible if the examiner's eyes are kept at the level of the patient's upper incisor teeth)*

- Down's syndrome
- Marfan's syndrome
- Thalassaemia
- Congenital cyanotic heart diseases
- Turner's syndrome

## SABRE TIBIA (ANTERIOR TIBIAL BOWING)

- Rickets
- Congenital syphilis
- Paget's disease

- Osteogenesis imperfecta

* 'Sabre' literally means cavalry sword with curved blade. In sabre tibia, the anterior border of tibia mimics the sword.

## HUTCHINSON'S TRIAD

1. Hutchinson's teeth (centrally notched, widely spaced, peg-shaped upper central incisor).
2. Interstitial keratitis.
3. Nerve deafness.

* Hutchinson's triad is a stigma of congenital syphilis

—— o ——

CHAPTER 17

# Diffuse Aches and Pains

## POSSIBLE AETIOLOGY

- Soft-tissue rheumatism
- Fibromyalgia
- Post-viral myalgia
- Myxoedema
- Polymyalgia rheumatica
- Hypermobility
- Chronic fatigue syndrome
- Hypophosphataemia
- Polymyositis/dermatomyositis
- Metabolic bone disease

* One should never forget osteomalacia and multiple myeloma as causes of diffuse pain in the body.

## NECK AND ARM PAIN

- Cervical spondylosis
- Musculoskeletal strain
- Trauma
- Rotator cuff syndrome
- Bicipital tendonitis
- Arthritis: glenohumeral, acromioclavicular
- Pancoast tumour
- Thoracic inlet syndrome
- Ischaemic heart disease

## CHRONIC FATIGUE SYNDROME (CFS)

Controversial topic; previously known as neurasthenia or myalgic encephalomyelitis. Commonly seen in females in between ages 20-50 years. The chief complaint is chronic fatigue which is made worse by minimal exertion. It is a combined physical and mental (e.g. poor concentration, irritability) fatigability. Aetiological factors are post-viral (e.g. viral hepatitis, infectious mononucleosis), physical inactivity and sleep difficulties. Though the role of stress is uncertain, there is presence of mood disorder in many patients. The onset may be sudden (fibromyalgia is insidious in onset) and tender trigger points are not associated with CFS. Psychotherepy is essential; antidepressants work better in the presence of mood disorder or insomnia.

## FIBROMYALGIA (FIBROSITIS SYNDROME)

It is a chronic multisystem illness characterised by widespread pain and associated with neuropsychological symptoms including fatigue, anxiety, depression, and many medically unexplained symptoms in other systems. Objective signs of inflammation are absent with normal laboratory studies including ESR. It is very often a diagnosis of exclusion; the cardinal feature is specific tender trigger points (note: in anxiety, tender points are present 'all over' the body). Women are the worst sufferer (F:M=10:1) who struggles much in family/day to day life. H/O familial disharmony is present in some patients; many have sleep disturbances who awake unrefreshed with poor mental concentration. There is widespread, unremitting pain. The patient may also suffer from irritable bowel syndrome, CFS, tension headache or premenstrual syndrome. There is associated subjective feeling of muscle swelling. Available evidence implicates the central nervous system as key in maintaining pain and other core symptoms of fibromyalgia. Reassurance, sympathetic attitude, aerobic exercise, NSAIDs, antidepressants (amitriptyline, dothiepin), trigger point injections (local anaesthetics/corticosteroid) or acupuncture may be of some help. Many patients do not respond to therapy.

## SOFT-TISSUE RHEUMATISM

This refers to aches and pains which arise from structures surrounding the joint such as tendons, muscles, bursae, and ligaments.
D/D with arthritis is done by,

- Tenderness away from the joint line margin

- Pain elicited with active movements (never on passive movements)
- Swelling usually away from the joint
- Dramatic relief with local injections of corticosteroid

## THE ANATOMICAL BASIS OF PAIN ARISING IN MUSCULOSKELETAL SYSTEM

*Joint:*

- Synovium–synovitis
- Joint capsule–capsulitis

*Periarticular (soft tissue):*

- Bursa–bursitis
- Tendon–tendonitis
- Tendon sheath–tenosynovitis
- Insertion of tendon, ligaments–enthesitis

soft-tissue rheumatism

*Bone*

## TINGLING AND NUMBNESS

This is the commonest paraesthesia in clinical practice → may occur in health due to sitting or sleeping in abnormal awkward position (e.g. sitting in the front rod of a bicycle) for some time (as a result of neuropraxia). Usually these are features of lesion in nerves (peripheral neuropathy), spinal roots (radiculopathy) or spinal tracts. Tingling sensation usually leads to numbness when there is total loss of function of touch as well as pain fibres. Common causes encountered in clinical practice are:

1. Peripheral neuropathy.
2. Carpal tunnel syndrome.
3. Cervical spondylitis.
4. Prolapsed intervertebral disc.
5. Lumbar canal stenosis.
6. Radiculopathy (e.g. from diabetes mellitus).
7. Hyperventilation (hysterical, salicylate overdose).

—— o ——

CHAPTER 18

# Diplopia

## DEFINITION

It is the perception of 'double vision' of a single object.

## MECHANISM OF PRODUCTION

In health, coordination of ocular muscles gives rise to conjugate movements of the eyes, and as a result binocular vision is achieved. Thus, the visual stimulus falls exactly on the similar parts of two retinae (macula), and an object is perceived as a single unit. Conjugate movements of the eyes are maintained by cortex, brainstem, and via the IIIrd, IVth and VIth cranial nerves. Defective movement of one eye may result in images (from the two eyes) arising from different points on the two retinae → as a result binocular fusion cannot occur in visual cortex → two separate or overlapping images are formed. In paralytic squint, the image from the healthy eye (true image) is clear and distinct, but the image produced on the affected eye is indistinct and blurred (false image; image derived from the retina outside macula) → false image is perceived in the true direction of action of the weak extraocular muscle.

Many a time, the head may be involuntarily turned in the dirction of action of paralysed or weak muscles (head tilt). True diplopia becomes single, if one eye is covered by hollow of the palm. Sudden appearance of diplopia usually points towards a neurological lesion.

Diplopia results form loss of parallel visual axes, and is commonly due to IIIrd, IVth and VIth nerve palsy, myasthenia gravis and myopathy (e.g. thyrotoxicosis). Diplopia should be clinically assessed in nine cardinal direction of gaze (i.e. the test of voluntary movements of extraocular muscles).

Cover one eye with a red glass, and ask the patient whether the red or the normally coloured image is the true imge → this 'red glass test' often indicates the affected eye.

## WHY THERE IS NO DIPLOPIA IN CONCOMITANT (NON-PARALYTIC) SQUINT?

Squint is of two types: paralytic and concomitant (non-paralytic).

In case of paralytic squint, the image on the paralysed side does not fall on the macula and thus diplopia occurs. Whereas in concomitant squint, the image formed by the defective eye is either rejected or suppressed by the occipital (visual) cortex and thus, there is no diplopia. Diplopia is maximum if the eye is moved in the direction of weak muscle.

## TYPES

Binocular: Diplopia perceived when both eyes remain open
Uniocular: Diplopia perceived when one eye remains open.

## POSSIBLE ASSOCIATIONS OF BINOCULAR DIPLOPIA

a. Loss of parallel visual axes: Displacement of eyeball by intraocular SOL, orbital pseudotumour.
b. IIIrd, IVth, VIth cranial nerve palsy: Intracranial SOL, demyelinating disease, cavernous sinus thrombosis, raised intracranial tension, meningitis, diabetes mellitus, vasculitis, vertebrobasilar insufficiency.
c. Muscle disorders: Congenital weakness of extraocular muscles, myasthemia gravis, muscular dystrophy, severe exophthalmos from thyrotoxicosis.
d. Miscellaneous: Ophthalmoplegic migraine, temporal arteritis, syphilis, restriction of eye movement (e.g. in pterygium, symblepharon).

## MONOCULAR (UNIOCULAR) DIPLOPIA

Rare and due to eye disorders like:

- Incipient stage of cataract (water molecule within lens)
- Subluxation of the lens
- Peripheral iridectomy (large)
- Astigmatism
- Keratoconus
- Iridolysis as a result of injury
- Hysteria.

## THE PEARLS

- IIIrd nerve palsy does not produce diplopia because of the presence of complete ptosis. Diplopia is complained by the patient as soon as the affected drooped upper eyelid is elevated by fingers
- In IIIrd, IVth and VIth nerve palsy, there is paralytic squint over and above diplopia
- In myasthenia gravis, diplopia is associated with diurnal variation of ptosis
- In concomitant squint, diplopia is absent
- In IVth nerve palsy, double vision is present for reading and walking downstairs. In IIIrd nerve palsy, diplopia and squint are maximum on looking up and in, while in VIth nerve palsy diplopia occurs in looking in the direction of affected muscle. In myaesthenia gravis and Graves' disease, diplopia occurs in all directions of gaze
- Treatment is aimed at correction of the primary defect. In incapacitating diplopia, shielding of the affected eye is advised.

—— o ——

CHAPTER 19

# Discoloured Teeth

## POSSIBILITIES OF STAINED TEETH

1. Chronic betel-leaf chewer, tobacco staining by smokers.
2. Tetracycline therapy (if given during second half of pregnancy, in infancy or in childhood < 8 years of age → permanent discolouration of teeth as well as enamel hypoplasia)–irregularly stained appearance.
3. Minocycline therapy (affects middle part of teeth with a greyish pigmentation; involves permanent teeth in comparison to tetracycline which affects temporary teeth. The drug is used in leprosy and nocardiosis).
4. Fluorosis (produces chalky-which patches, yellowish-brown discolouration).
5. Congenital erythropoietic prophyria (teeth are brownish-pink).
   ↓
   erythrodontia, as a result of high porphyrin content → orange-red fluorescence under Wood's lamp.
6. Unhealthy oral hygiene (the yellowish-brown discolouration may be eradicated after repeated mouth wash/brushing).

## GRADES OF DENTAL FLUOROSIS

Three grades like —

1. White chalky opacities of patches on enamel with or without faint yellow lines.
2. Distinct brownish discolouration.
3. Pitting of enamel surface, sometimes with chipping of edges.

Dental fluorosis which is classically known as 'mottled enamel' is essentially a dental hypoplasia with areas of ↓ calcification and ↓ mineralisation.

In Punjab, fluorosis manifests in one of the severest forms leading to advanced invalidism. In fluorosis, considerable disability is associated with spinal rigidity, restricted movements of the joints, and flexion deformity of the hips and knees. There is ↑ bone density; tendons, ligaments and even muscles may be mineralised. Ultimately, compressive myelopathy leads to progressive neurological disability.

## OSTEOSCLEROSIS (BONE TURNS ABSOLUTELY WHITISH IN X-RAY)

- Fluorosis
- Metastatic deposits from carcinoma of prostate (commonly), breast, intestine or bronchus
- Hodgkin's disease (ivory vertebra)
- Marble bone disease (osteopetrosis)
- Osteopoikilosis
- Vitamin A or D toxicity
- Jaw in Paget's disease
- Diffuse idiopathic skeletal hyperostosis (DISH).

—— o ——

CHAPTER 20

# Drop Attacks

## DEFINITION

A fall occurring without warning, giddiness, tripping, apparent paralysis or loss of consciousness.

## DESCRIBE THE FALL

The victims are usually middle-aged or elderly women. They suddenly drop (i.e. fall) to the floor while standing or walking. She/he feels her/himself falling (intense leg weakness), then crashes the ground (knees buckle, striking the knees/nose upon the ground) and picks her/himself up almost immediately. Some patients describe sudden and momentary loss of power in the legs and they are unable to prevent the fall by raising their hands. Drop attacks are presumably of brainstem origin, rather than thromboembolism, and consciousness is preserved throughout. No post-incidence confusion is there but considerable embarrassment persists.

## COMMON ASSOCIATIONS OF DROP ATTACKS

1. No obvious cause
2. Vertebrobasilar insufficiency (may complain diplopia)–one of the very common cause of drop attack
3. Epilepsy
4. Hydrocephalus, third ventricular tumour (both the patients are young)
5. Parkinson's disease
6. Quadriceps weakness (they fall simply due to knees flexing; may be able to protect the face by raising the arms)
7. Causes of 'falls in the elderly' (see below).

## INVESTIGATIONS FOR DROP ATTACKS

1. Blood for sugar (F)
2. X-ray of neck to rule out cervical spondylosis (as a cause of vertebrobasilar insufficiency)
3. Electroencephalogram (EEG)
4. CT or MRI scan to rule out any organic lesion in brain
5. Doppler studies of carotid/vertebral arteries.

## CAUSES OF FALLS WITH DISTURBED CONSCIOUSNESS

1. Epilepsy
2. Syncope (simple faints, cough/micturition/carotid sinus)
3. Transient ischaemic attack (TIA)
4. Cardiac dysrhythmias
5. Pseudo-seizures (non-epileptic attacks)
6. Panic attacks
7. Vertigo
8. Hyperventilation
9. Breath-holding (children)
10. Pheochromocytoma, carcinoid syndrome, drug reactions
11. Hypoglycaemia, hypocalcaemia

* One should do ECG and Holter monitoring (24-hours ambulatory ECG), if necessary.

## MAJOR CAUSES OF 'FALLS IN THE ELDERLY'

1. Visual impairment (e.g. cataract)
2. Reduced hearing
3. Muscle weakness
4. Postural hypotension (e.g. use of antihypertensives or diuretics)
5. Lack of concentration
6. Musculoskeletal disorder (e.g. rheumatoid arthritis)
7. Use of medications (sedatives, antidepressants, diuretics)
8. Foot disorders (deformities/oedema)
9. Postural instability/vestibular dysfunction
10. Gait or balance abnormalities (e.g. acute labyrinthitis)
11. Depression
12. Cognitive impairment
13. Environmental: insufficient light, high-stepping stairs, waxed slippery mozaic floor, uneven carpet edge, raised toilet seat in bathroom, uneven/high heel shoes.

## D/D OF 'FIT' IN CLINICAL PRACTICE

1. Idiopathic epilepsy–H/O previous fits; perform EEG (abnormal) and CT scan (normal).
2. Febrile convulsions–Age ranges 6 months to 5 years, often with a +ve family history; normal EEG and CT scan. Always associated with febrile illness.
3. Intracranial space occupying lesion (ICSOL)–Any age, headache, papilloedema; confirmed by CT or MRI scan.
4. Convulsions due to meningitis–Pyrexia, +ve neck rigidity; confirmed by lumbar puncture and CT scan.
5. Hypoglycaemia–Perspiration, ↑ hunger, a diabetic on insulin or oral hypoglycaemic agents; confirmed by blood sugar level during the attack (hypoglycaemic symptoms when blood sugar is <50 mg/dl and convulsions occur when it is <36 mg/dl).
6. Alcohol withdrawal–History from relatives is diagnostic and is often suggested by recent heavy intake of alcohol; confirmed by recurrent episodes in similar circumstances.
7. Severe dyselectrolytaemia (↓Na, ↑Na, ↓Ca, ↓Mg)–Confirmed by serum biochemistry.
8. Severe hypotension (sudden)–Pulse not palpable, BP not recordable, central + peripheral cyanosis; ECG shows asystole or electro-mechanical dissociation.
9. Pseudo-seizures (functional)–Always happens to occur in front of people/relatives + eyes closed during seizure; normal EEG and CT scan.

—— o ——

CHAPTER 21

# Erectile Dysfunction (Impotence)

## DEFINITION

Inability of the male to achieve an erection is impotence (a better terminology is 'erectile dysfunction' or ED). Male erection is a neurovascular reflex, which depends on a healthy anatomy of penis with an ideal hormonal environment. Impotence is of two types: primary (ED from the beginning) and secondary (initiates after a period of normal penile erection).

## BASICS

Three basic mechanisms needed to develop ED —

1. Failure to initiate (psychogenic, endrocrinologic, or neurogenic), or
2. Failure to fill (arteriogenic), or
3. Failure to store (venoocclusive dysfunction) sufficient volume of blood within the lacunar network of penis.

Multiple factors contribute to ED in many patients. Diabetes mellitus, atherosclerosis, and drug-related aetiologies are responsible for major causes of ED in older people.

## POSSIBLE ASSOCIATIONS

### *Psychological (i.e. situational)*

Variety of psychogenic inputs like anxiety, depression, sexual inhibition, sexual abuse in childhood, fear of pregnancy/STD, sense of guilt, ignorance of sex act, conflicted parent-child relationship or religious orthodoxy.

### *Organic (i.e. constitutional)*

a. Endocrine–Kallman's syndrome, hypopituitarism, hyperprolactinaemia, pituitary tumour, Klinefelter's syndrome, testicular tumour/trauma/orchitis, alcoholic liver disease induced testicular atrophy, hypogonadism, andropause, Addison's disease, hypo- or hyperthyroidism.
b. Diabetes mellitus (DM-associated neurologic as well as vascular complications are responsible for ED in 35-75% patients).
c. Vascular–atherosclerosis (e.g. Leriche's syndrome) or traumatic arterial disease.
d. Neurogenic–autonomic neuropathy (e.g. from DM), cauda equina lesion, multiple sclerosis, peripheral neuropathy (e.g. alcoholism), following pelvic surgery, spinal cord injury, alcohol excess.
e. Drug-induced–antihypertensives, antidepressants, tranquilizers, psychotropics, anticholinergics, cytotoxic drugs, hormones (e.g. oestrogens), β-blockers.
f. Miscellaneous — recreational drugs or addictions, alcohol, cocaine, marijuana), chronic debilitating diseases (chronic renal failure, motor neurone disease), pelvic fracture, mechanical interference from morbid obesity, prostatectomy, Peyronie's desease.

## CLINICAL EVALUATION

A close-door sympathetic interview with the patient is the first task of the physician. Differentiate organic from psychological cause; ED with only one partner, of sudden onset, intermittent (i.e. not permanent), with ability to masturbate, having nocturnal and early morning erections, and with normal nocturnal penile tumescence test (a plethysmograph placed around the penis overnight to determine the neurovascular action sufficient to produce erection during sleep) suggest psychogenic ED. Other aspect of history should focus on duration and persistence of ED, symptoms suggestive of endocrine disorders, neuropathy, vascular disease or diabetes.

Normal level of testosterone, gonadotrophin and prolactin with history of nocturnal emissions and frequent satisfactory morning erections make endocrime disorders unlikely. A careful history of stress, alcohol abuse, drugs (mentioned above) should be taken.

Details physical examination of BP, thyroid, liver, CVS, renal system should be sought for. Size of testicles and penis (e.g. priapism), secondary sexual characters, and testing of peripheral neuropathy must be done.

In selected patients, specialized testing may give clue to diagnosis:

a. Studies of 'nocturnal penile tumescence (NPT)' and rigidity.
b. Vascular testing (penile doppler USG, penile angiography, dynamic infusion covernosometry).
c. Neurologic testings (vibratory perception; so called somato-sensory evoked potentials).
d. Psychological diagnostic tests.

It is important to remember that, psychogenic ED is frequently a diagnosis of exclusion.

* Reduced libido: hypogonadism and depression
Intact libido: others including psychological problems

** NPT test is normal in psychogenic ED.

## MANAGEMENT

1. Patients education is started with gynaecological examination of the female partner to rule out any obstructive pathology in female genital tract. Psychiatric examination of both the partners is mandatory. It is important to discuss the matter frankly with the patient.
2. Drugs–Phosphodiesterase type-5 inhibitors (sildenafil, tadalafil, vardenafil) ↑ penile blood flow and remain the first line of drug therapy in ED; apomorphine, intraurethral or intracavernosal self-injections of alprostadil, papavarine or phentolamine.
3. Androgen therapy–Androgen replacement treatment is used in primary and secondary causes of hypogonadism. Loss of libido is corrected by androgen therapy.
4. Devices–Vacuum constriction devices, insertion of inflatable penile prosthesis or revascularization surgery may be done in selected cases.
5. Sex therapy–It addresses specific interpersonal factors; and consists of in-session discussion and at-home exercises specific to the person and the relationship. Both the partners should be involved in sex therapy to have a favourable outcome.

## PEARLS

First try to rule out diabetes, ↑ lipids, ↑ prolactin, ↓ androgen, hypo- or hyperthyroidism. In neurological examination, penile and perianal sensation are carefully examined.

## PREMATURE EJACULATION

It is the discharge of the semen before the orgasm is attained, i.e. it is an early orgasmic response. If it is persistently or recurrently experienced, one seeks advice of a doctor. The main causes in clinical practice are –

- Psychological (no sexual experience, anxiety)
- Injury to genitourinary tract, genital anomalies or urinary infection (e.g. burning micturition)
- Diabetic autonomic neuropathy (affects parasympathetic control)
- Spinal cord injury.

While treating the patient, the physician should consider duration of excitement phase, age of the patient, frequency and duration of coitus; in the day to day practice, it is seen in young patients with lack of sex knowledge. Usually, it requires no treatment but psychiatric counselling with clearing of myths/misconceptions, or application of anti-anxiety drugs (sertraline, fluoxetine) may be of some help.

## CARDINAL ELEMENTS OF NORMAL SEXUAL FUNCTION

Libido (desire)
↓
Erection (lubrication in female)
↓
Intromission
↓
Ejaculation and orgasm (only orgasm in female).

## PRIAPISM

It is the unwanted, painful and persistent erection of penis, and is commonly due to:

- Sickle cell anaemia
- Hypercoagulable states
- Chronic myeloid leukaemia
- Spinal cord injury
- Injection of vasodilators (e.g. papavarine) into penis
- Pelvic vascular thrombosis
- Megapenis.

It is a very embarrassing situation for the patient. To treat the first 3 causes, analgesics, hydration and α-adrenergic blockers are used. In others, treatment of the aetiology is solicited.

## RETROGRADE EJACULATION

The process of ejaculation starts by → stimulation of sympathetic nervous system → contraction of vas deferens + seminal vesicles + prostate → seminal fluid entering into urethra, associated with rhythmic contractions of bulbocavernosus and ischiocavernosus muscles → ejaculation follows. When the internal urethral sphincter remains open during the process of ejaculation, semen enters into urinary bladder, and is known as retrograde ejaculation. It is commonly seen in diabetic patients with autonomic neuropathy or after surgery involving the bladder neck.

—— o ——

CHAPTER 22

# Eyes: a Clue to Diagnosis

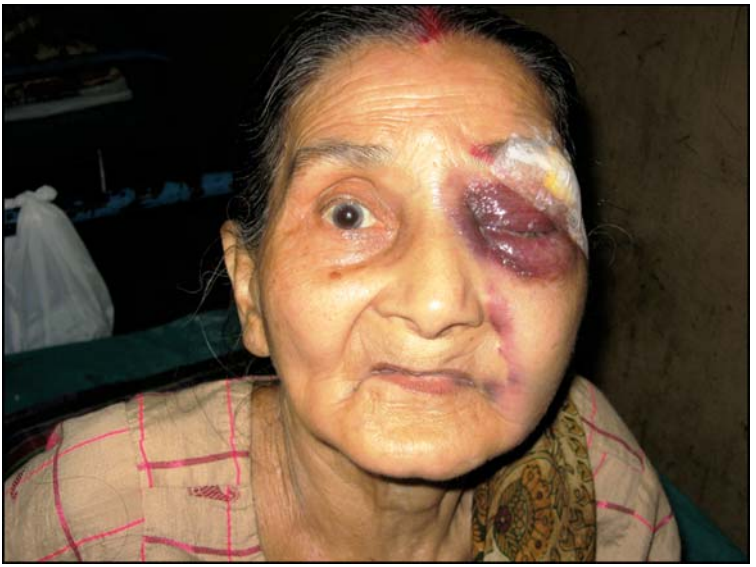


**FIGURE 22.1:** Periorbital haematoma from head injury

Very often the face, especially the eyes speak for a diagnosis of medical illness as many diseases may have manifestations in and around the eyes:

1. Ptosis (pseudoptosis too).
2. Exophthalmos, e.g. thyrotoxicosis.
3. Retraction of upper eyelids → hyperthyroidism.
4. Photophobia → meningitis, painful diseases of the eye (or 'red eyes'), migraine, tetanus, albinism.
5. Voluntary closure of eyes with occasional blinking → hysteria.

**FIGURE 22.2:** Bilateral subconjunctival haemorrhage in the second day of viperidae snake bite

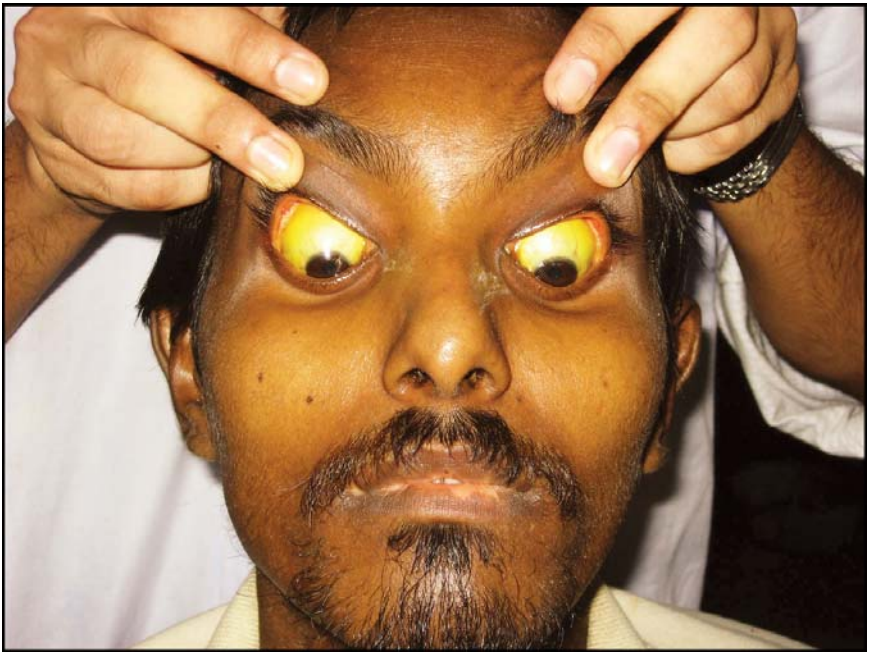


**FIGURE 22.3:** Jaundice demonstrated in yellowish upper bulbar conjunctiva

6. Periorbital oedema → angio-oedema, drug hypersensitivity, acute glomerulonephritis, hypothyroidism, infection with Trichinella spiralis (trichinosis), congestive cardiac failure, dermatomyositis.
7. Xanthelasma around the eyes (often indicates hypercholesterolaemia).
8. Pallor → the lower palpebral conjunctiva.
9. Jaundice → the upper bulbar conjunctiva.
10. Cyanosis → the lower palpebral conjunctiva.
11. Chemosis (oedema) of conjunctiva → type II respiratory failure (hypercapnoea), SVC syndrome, severe exophthalmos, alcoholism, Weil's disease, hypoalbuminaemia.
12. Subconjunctival haemorrhage → severe cough (e.g. whooping cough), bleeding disorder, fracture of skull, snake bite, anticoagulant therapy or without any obvious cause.
13. Blue sclera → osteogenesis imperfecta (read the chapter on 'Blue sclera').
14. Scleromalacia perforans (choroidal pigments are seen as small brown patches on either sides of the iris) → rheumatoid arthritis.
15. Corneal damage or opacities → trauma, infection, malignant exophthalmos, xerophthalmia from Sjögren's syndrome, Bell's palsy, Vth cranial nerve palsy.
16. Archus senilis→ indirect clue to atherosclerosis.
17. Kayser-Fleischer (K-F) ring → Wilson's disease, prolonged cholestasis.
18. Phlyctenular conjunctivitis→allergic reaction to primary tuberculosis.
19. Iridocyclitis → tuberculosis, sarcoidosis, sero-negative arthritis.
20. Cataract → hypoparathyroidism, diabetes mellitus, galactosaemia, prolonged steroid therapy, Down's syndrome, Wilson's disease, myotonia dystrophica, atopic dermatitis.
21. Enlarged lacrimal glands → Sjögren's syndrome, sarcoidosis.
22. Ectopia lenis → look for iridodonesis
    Upward subluxation: Marfan's syndrome
    Downward subluxation: Homocystinuria.
23. Bitot's spot → vitamin A deficiency (whitish, foamy, raised and triangular spot with its base towards limbus; present on the bulbar conjunctiva, a little away from limbus).
24. Brushfield's spot → Down's syndrome (radially arranged small whitish inclusions having the appearance of grains of salt, present in between middle and other third of iris).
25. Squint (concomitant or paralytic)–in paralytic squint, it indicates external ophthalmoplegia (IIIrd, IVth, or VIth cranial nerve palsy).

26. Epicanthic folds at inner angle of eyes → Down's syndrome, Treacher-Collins syndrome.
27. Hypertelorism (widely set eyes) → Down's syndrome, associated with congenital supravalvular aortic stenosis (elfin facies) or pulmonary stenosis, mental retardation, thalassaemia, craniofacial dysostosis and carpenter syndrome.
28. Pupil → miosis, midriasis or anisocoria (unequal pupils).
29. Nystagmus → Pendular nystagmus when the patient looks forward; jerky nystagmus on fixation of gaze laterally.
30. Myokymia → benign phenomenon in fatigued or anxious person (persistent twitching and occasionally rhythmical movement, especially of peri-orbital muscles).
31. Corneal opacities → 'band keratopathy' due to hypercalcaemia; cholesterol deposition (arcus senilis) due to hypercholesterolaemia; chloroquine crystals as a result of treatment in DLE; copper deposition (K-F ring) in Wilson's disease; cystine crystals in cystinosis; deposition of amiodarone used in the treatment of cardiac dysrhythmias; keratitis with corneal opacities in herpes zoster or simplex infection, congenital syphilis, Reiter's syndrome.
32. Grey-brown sclera → alkaptonuria.
33. Madarosis (loss of lateral 1/3rd of eyebrows) → myxoedema, lepromatous leprosy, amyloidosis and neurodermatitis.
34. Palpebral fissure:
    Wide → exophthalmos
    Narrow → partial ptosis, photophobia and blepharospasm
    Oblique → Down's syndrome
    Absent → Evisceration or enucleation of eyeball.
35. Red eyes → trauma, conjunctivitis, keratitis, scleritis, episcleritis, uveitis, subconjunctival haemorrhage, acute congestive glaucoma.
36. 'Bags under the eyes' → may not have any clinical value but often associated with insomnia, chronic alcoholism and myxoedema.
37. Blepharospasm → involuntary spasmodic closure of eyelids (a form of dystonia).
38. Enophthalmos → senility, phthisis bulbi, microphthalmos, Horner's syndrome, severe dehydration, as a consequence of resolved orbital cellulitis.
39. Lagophthalmos → physiological, extreme degree of proptosis, Bell's palsy, eyelid scarring.

## FIVE COMMON CAUSES OF PAINFUL EYE

1. Conjunctivitis
2. Iritis
3. Corneal ulcer
4. Acute glaucoma
5. Retrobulbar neuritis

* 'Aching' pain indicates internal cause while 'gritty' pain points towards external cause. ↑ sensitivity to light is always associated with inflammation of eyes.

## COMMON CAUSES OF 'TUBULAR VISION'

1. Hysteria (tunnel vision)
2. Glaucoma (terminal)
3. Retinitis pigmentosa (advanced)
4. Papilloedema
5. Migraine
6. Posterior cerebral artery occlusion

* Tubular vision is the result of peripheral constriction of visual field.

—— o ——

CHAPTER 23

# Face Reading

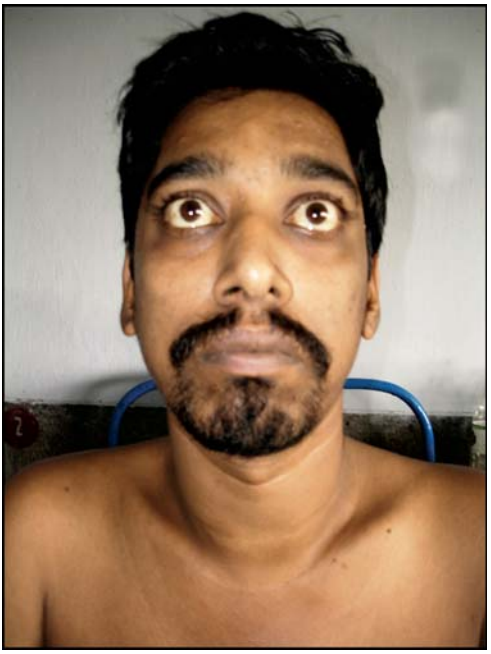


**FIGURE 23.1:** Exophthalmos in a patient with thyrotoxicosis

## PROLOGUE

Reading the expression of the face of the patients or 'facies' often suggests an instant or 'spot' diagnosis. Experience teaches a doctor to read the face vividly. One may prove to be wrong in face reading but every doctor should learn how to read the emotional play, response to questening and intellectual shadows in the patient's face. The vacant stare of a mentally

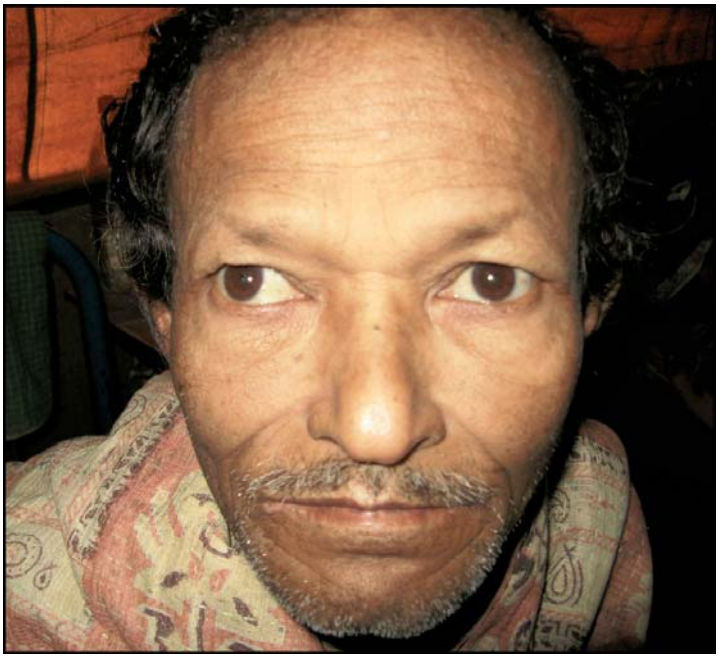


**FIGURE 23.2:** Madarosis (loss of hair in lateral third of eyebrows) with concomitant squint in a male hypothyroid

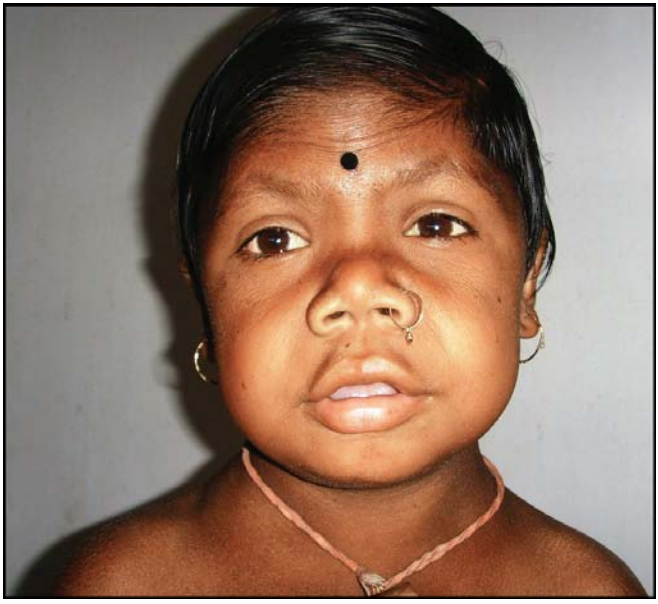


**FIGURE 23.3:** Classical facies of cretinism: a dull and idiotic look associated with depressed bridge of the nose, wrinkled eyebrows, broad flat nose with big nostrils, narrow palpebral fissures, thick and everted lips

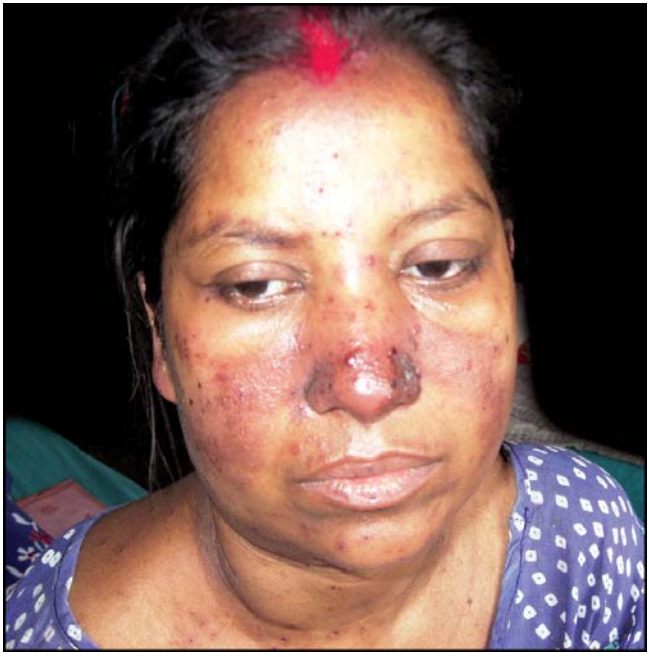


**FIGURE 23.4:** Classical photosensitive butterfly rash over face with erythema, oedema and telangiectasia in a patient of SLE

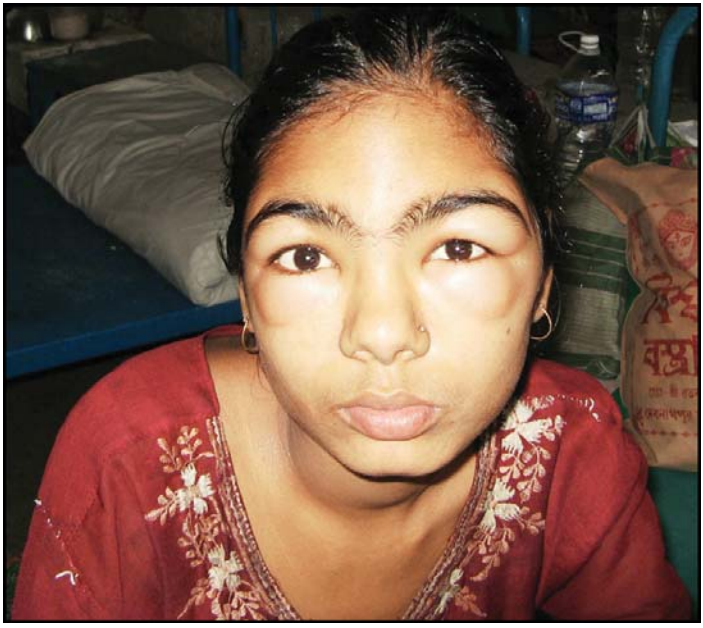


**FIGURE 23.5:** Periorbital oedema in nephrotic syndrome

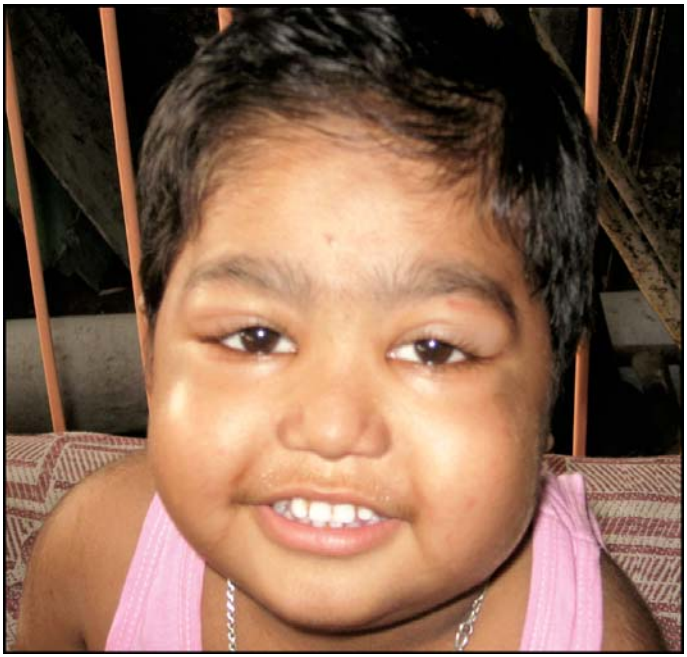


**FIGURE 23.6:** Cushingoid facies in a child suffering from steroid-dependent minimal lesion nephropathy (nephrotic syndrome)

retarded child, sad appearance of depression or anxious facies of thyrotoxicosis can never be overlooked. Though the 'facies' may be deceptive, one should meticulously examine for complexion, eyes, eyebrows, skin of cheeks, mouth, nose, nasolabial folds, head, hairs or any sign of wasting present or not.

Few 'facies' are described below where the patient carry their diagnosis in the face.

## DIFFERENT 'FACIES' IN CLINICAL MEDICINE

### ACROMEGALY

a. Prognathism (lantern or bull-dog jaw) where the lower incisors (mandible) protrude in front of the upper jaw (maxilla), and widened spaces between upper and lower teeth; heavy chin.
b. Facial enlargement (large wide face) with prominent supraorbital ridges as a result of enlargement of maxillary, frontal and ethmoid sinuses. Frontal bossing may be associated with.
c. Macroglossia with thick lips, and large ears and nose; wide spacing of teeth may be present.

d. Thick skin with hypertrichosis (e.g. bushy eyebrows), hyperhidrosis and increased sebum production (skin becomes greasy); exaggerated nasolabial folds.
e. Deep, husky and resonant voice due to increased thickness of vocal cord.

## THYROTOXICOSIS

a. Staring as well as anxious look
b. Exophthalmos
c. Lid retraction reading to an anxious startled look
d. Infrequent blinking
e. Lack of harmonious movement between the eyeball and the eyelid
f. Moist skin of face with a malar flush.

## BELL'S PALSY

a. Facial asymmetry
b. Asymmetry of blinking and eye closure
c. Lack of spontaneous movements of face
d. Wide palpebral fissure with epiphora
e. Bell's phenomenon (the eyeball rolls upwards and inwards during attempted forced eye closure)
f. Loss of nasolabial fold
g. Angle of the mouth is drawn to the healthy side when asked to show the upper teeth
h. Inability to blow or whistle properly

* b) to f) are noticed on the affected side of facial paralysis.

## THALASSAEMIA (MONGOLOID)

a. Frontal bossing (prominent forehead as a result of marrow hyperplasia)
b. Depressed bridge of the nose (due to hyperplasia of lesser wing of sphenoid)
c. Hypertelorism (widely set eyes)
d. Apparent mongoloid slant of the eyes
e. Malar prominence (due to marrow hyperplasia)–'Chipmunk facies'
f. Dental malocclusion with prominent upper incisors
g. Associated with mild icteric tinge of the conjunctiva, and pallor.

### DOWN'S SYNDROME (MONGOL)

a. Small, round and flat face, brachycephaly; downy forehead; short fleshy neck
b. Upwards-slanting eyes (oblique orbital fissure) with epicanthic folds at inner angles
c. Low set ears; ears are small and dysplastic
d. Small nose with depressed bridge of the nose
e. Hypertelorism
f. High arched palate with small teeth
g. Open mouth with protruded and furrowed tongue (macroglossia), with an idiotic look (often called as cheerful idiot).

### CRETINISM

a. Dull, expressionless and idiotic look; large head with scanty scalp hair
b. Depressed bridge of the nose, blood flat nose with big nostrils
c. Hypertelorism with wrinkling of eyebrows; narrow palpebral fissures
d. Sparse, coarse and brittle hair with dry skin
e. Thick and everted lips with big, fissured, protruded tongue (macroglossia)
f. Delayed dentition with hoarse voice.

### TABETIC

This is the classical facies of tabes dorsalis and is rarely seen now-a-days.
a. Bilateral partial ptosis
b. Compensatory wrinkling in the forehead
c. Elevated eyebrows
d. Very little subcutaneous fat with loss of emotional reflexes
e. Accompanied by Argyll Robertson pupil.

### DEHYDRATION (FACIES HIPPOCRATICUS)

a. Face is drawn
b. Shrunken eyes; the eyeballs are soft as a result of lowering of intraocular tension
c. Pinched-up nose
d. Parched lips
e. Hollowed temporal fossa; depressed anterior fontanelle (infants)

f. Tongue is dry and coated
g. Skin is dry and wrinkled.

### HEPATIC

a. Shrunken eyes
b. Hollowed temporal fossa
c. Pinched-up nose associated with malar prominence
d. Parched lips
e. Muddy complexion of skin (blending of pallor, jaundice and melanosis)
f. Shallow and dry face
g. Icteric tinge of conjunctiva.

* Hepatic facies is characteristic of chronic liver disease.

### MOON FACE

The face looks bloated and rounded (like the full moon). Moon face is commonly associated with:

- Cushing's syndrome (facial plethora + hirsutism)
- Nephrotic syndrome (periorbital oedema)
- Acute glomerulonephritis (periorbital oedema)
- Myxoedema (baggy lower eyelids)
- Prolonged corticosteroid therapy (facial plethora + hirsutism)
- Superior mediastinal syndrome (prominent veins in forehead and temple)
- Angioneurotic oedema of face (patchy involvement; lips are swollen ++)
- Subcutaneous emphysema of face, extending from chest (asymmetrical).

### NEPHRITIC (ACUTE GLOMERULONEPHRITIS OR AGN)

a. The face is pale and puffy
b. Pronounced periorbital oedema with narrowing of the parpebral fissure.

### NEPHROTIC

The facies looks like nephritic one but the features of moon face is more pronounced in nephrotic syndrome.

* Periorbital oedema is characteristically seen in nephrotic syndrome, AGN, myxoedema, angioneurotic oedema, dermatomyositis, orbital cellulitis and thyroid eye disease.

## SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

a. Photosensitive 'butterfly' rash over the malar areas and bridge of the nose (does not affect the nasolabial folds)
b. The lesion will have erythema + oedema during acute phase, and atrophy + telangiectasia in chronic phase
c. Patchy alopecia with 'lupus hairs' (short, broken hairs) seen above the forehead
d. Ulcer in the oral cavity (especially, palatal ulcer).

## SCLERODERMA

a. Mask-like facies (lacking facial expression)
b. Absence of normal wrinkling of skin ('ironed' out skin folds)
c. Pinched-up nose or beaking of the nose
d. Inability to open the mouth, i.e. microstomia (small mouth) with radial furrows on closing (tobacco-pouch or fish-mouth appearance)
e. Skin over the face seems taut and shiny
f. Pigmentation + depigmentation + telangiectasia.

## MASKED FACIES

a. Lack of facial expression (i.e. a blank look)
b. Infrequent blinking with staring look (spontaneous ocular movements are lacking)
c. Widened palpebral fissure

***Poverty of expression (i.e. masked facies) is commonly seen in:***

- Parkinsonism
- Scleroderma
- Bilateral UMN type facial nerve palsy
- Myxoedema
- Depression
- Myasthenia gravis and facial myopathies (rare)
- Dementia
- Sometimes in pseudobulbar palsy.

## MALAR FLUSH

Read the chapter on 'Flushing of face'.

## PLETHORIC FACE

↑ redness of face is confronted in clinical situations like:

- Chronic alcoholism
- Cushing's syndrome
- Polycythemia
- SVC syndrome
- Chronic cor pulmonale
- Carcinoid syndrome.

## MITRAL FACIES

It is rarely seen in India. This is the pinkish purple patches on cheeks → it is due to:

- Low cardiac output in mitral stenosis → vsoconstriction → peripheral cyanosis in lips, tip of the nose and cheeks
- Vasodilatation (vascular stasis) in malar area leads to malar flush.

## PARKINSONISM

It is a classical masked facies with all features present. Speech is monotonous + bradylalia + hypophonia. The eye signs in parkinsonism are:

- Infrequent blinking with a staring look (akinetic face)
- Reptelian gaze
- Impaired pursuit movement of eyeball
- Hypometric saccades
- Oculogyric crisis (involuntary upward conjugate deviation of eyes)–in post-encephalitic variety
- Blepharoclonus (fluttering of eyelids on closing the eyes).
- Reversed Argyll Robertson pupil (post-encephlitic variety).

## POLYCYTHEMIA (RUDDY CYANOSIS)

Dusky-red (plethora + cyanosis) discolouration of nose, lips, malar prominence, ears and palpebral conjunctiva (i.e. suffused conjunctiva)

## MYXOEDEMA

a. Dull, espressionless, puffy face (periorbital puffiness with baggy lower eyelids)
b. Coarse hair, patchy alopecia, thin and sparse eyebrows (loss of lateral 1/3rd of eyebrows–madarosis)

c. Dry, rough skin with swollen lips
d. Expressionless face, facial pallor (may have rose-purple malar flush) with macroglossia
e. Deep and hoarse voice + bradylalia.

### MYOTONIA (DYSTROPHICA)

a. Ptosis with ophthalmoplegia
b. Long facial structure ('hatchet face' due to atrophy of temporalis + 'swan neck' due to atrophy of sternomastoid muscle)
c. Frontal baldness
d. Cataract
e. Mental retardation (mild)

### MYOPATHIC FACE (E.G. FACIOSCAPULOHUMERAL DYSTROPHY)

a. Loose pout of lips at rest due to facial weakness
b. A transverse smile (rire en travers)
c. Ptosis (+ external ophthalmoplegia in 'ocular' variety).

### MYASTHENIC FACIES

a. Uni- or bilateral (usual) ptosis; fluctuating ptosis → sustained upward gaze for two minutes leads to increase ptosis, and injection of edrophonium corrects ptosis dramatically (Tensilon test)
b. A peculiar smile ('myasthenic snarl') where the lips elevate but do not retract
c. Pupils are never affected
d. Rarely, there may be complete paralysis of ocular movements.

### LEONINE FACE (LEPROMATOUS LEPROSY)

a. Forehead lines become deeper; madarosis with thick eyebrows
b. Depressed bridge of the nose, broad nose with nasal collapse (saddle-nose)
c. Thickened skin of face + forehead, especially of infiltrated earlobes
d. There may be perforated nasal septum
e. Loosen upper central incisor teeth + hoarse voice.

### ELFIN FACIES (IN SUPRAVALVULAR AORTIC STENOSIS)

a. Broad forehead with pointed chin
b. Cupid's bow-like upper lip with upturned nose
c. Hypertelorism with low set ears.

## CUSHING'S SYNDROME (I.E. CLASSICAL MOON FACE)

a. Moon face
b. Dusky and plethoric face
c. Hirsutism.

N.B: Other than facial expression and different physical changes, one has to be very careful about alteration of facial contour, voluntary and involuntary movements of face, different eye signs and stigmata of acute illness printed in the face (i.e. herpes labialis). Very often the clue to diagnosis remains in examination of tongue, pseudoptosis, mild exophthalmos, malar flush, masked appearance, small goitre in neck and asking the patient to show the upper teeth.

—— o ——

CHAPTER 24

# Facial Pain

## PREFACE

Different pain-sensitive parts of face are teeth, gums, sinuses, temporomandibular joints, jaw and eyes; facial pain may be evoked by specific neurological conditions.

## CLUE TO DIAGNOSIS

1. Facial infection, cellulitis, abscess
2. Chronic subclinical sinus infection
3. Trigeminal neuralgia (tic douloureux)
   ↓
   - Never extends outside the Vth nerve territory
   - Mouth-ear zone and naso-orbit zone are commonly affected; ophthalmic division is involved rarely (5%)
   - F:M = 3:1; middle-aged or elderly
   - Strictly unilateral
   - Sudden, severe, stabbing/shooting/lancinating pain, for seconds which may be repetitive. Precipitated by touching the 'trigger zones' in face or by eating; usually no neurological sign present
   - Remit and relapse
   - May be related to disseminated sclerosis or posterior fossa tumour
4. Post-herpetic neuralgia
   ↓
   - Very elderly females > males, over 70 years
   - Over ophthalmic division of Vth nerve (forehead) → most severe over eyebrows and may mimic headache of temporal arteritis

- Very severe; often non-stopping type
- Post-herpetic scars are anaesthetic; normal skin between scars are tender
- High suicidal tendency

5. Trauma/post-traumatic neuralgia
6. Facial migraine syndrome

↓

- Males > females; any age
- Deep eye pain; often a feeling as if the mastoid is swollen
- Throbbing type with lacrymation and conjunctival injection
- Alcohol aggravates the pain
- Probably a variety of 'clusture headache'; previously called ciliary neuralgia, vidian neuralgia, petrosal neuralgia, spheno-palatine neuralgia or geniculate neuralgia are probably variants of facial migraine
- 'Sluder's lower-half headache' (rare)
  - Bursting; near base of the nose, near mastoid, behind the eye with nasal congestion

7. Atypical facial pain

↓

- F > M; 30-50 years
- Continual, unbearable, deep bruning type pain; over either or both maxillary region; the patient clutches his face in pain
- May be associated with delusional overtones

8. Temporo-mandibular osteoarthritis (Costen's syndrome)

↓

- Usually in elderly females
- The site of pain is over the joint or just anterior to it
- May be mistaken with temoporal arteritis
- Aggravates on chewing or yawning
- Severe aching type; only present on eating

9. Carotidynia

↓

- Episodic throbbing type pain in neck
- Associated with swelling and tenderness of the carotid artery
- A little presure over carotid → pain ↑
- A firm presure over carotid → pain ↓

10. Temporal arteritis
    ↓
    - Intermittent claudication of jaw muscles and tongue; headache
    - Elderly; F > M = 4:1
    - Associated with scalp tenderness, fever, malaise, visual problems (from diplopia to permanent visual loss) or ptosis
    - Thickened and tender temporal arteries
11. Anginal pain
    ↓
    - Along with central, stabbing chest pain with radiation to arms (left > right), there may be radiation of pain to neck and lower jaw
    - Sublingual isosorbide dinitrate relieves the pain
12. Somatisation syndrome/anxiety
13. Glossopharyngeal neuralgia
14. Miscellaneous
    - Paget's disease
    - Cerotid artery aneurysm
    - Cerebello-pontine angle tumour
    - Nasopharyngeal carcinoma
    - Tolosa-Hunt syndrome

* To diagnose the facial pain variants, one should totally depend clinically on:
- History
- Age
- Sex
- Site of pain
- Character of pain
- Aggravating and relieving factors

** There is no such special investigation which may pin-point the diagnosis.

## PAIN IN THE EYES

- Trauma
- Conjunctivitis, blepharitis
- Iritis, iridocyclitis, uveitis
- Foreign body
- Glaucoma
- Clusture headache
- Tic douloureux
- Periorbital cellulitis
- Xerophthalmia
- Entropion
- Retrobulbar neuritis
- Cerebral tumour/aneurysm
- Irritation from eye drops
- Ultraviolet light

## LUMP IN THE FACE

1. Parotid swelling (mumps, mixed parotid tumour)
2. Preauricular lymphadenititis (tender lymph nodes in front of ear)
3. Subcutaneous abscess (a tender and fluctuant swelling)
4. Dental abscess (tenderness of underlying tooth on gentle tapping)
5. Preauricular lymphoma (non-tender lymph nodes in front of ear)
6. Melanoma (painless swelling with pigmented lesion), sebaceous cyst (swelling with punctum)
7. Basal cell carcinoma (painless ulcer)
8. Nasopharyngeal carcinoma (swelling at root of nose, ophthalmoplegia)
9. Angioneurotic oedema (solid asymmetrical oedema involving lips, eyelids)
10. Facial haematoma (from trauma)
11. Neurofibroma (single or multiple painless rubbery cutaneous tumour).

—— o ——

CHAPTER 25

# Fatigue

## DEFINITION

It is the excessive tiredness on exertion, and occurs in organic or functional ill-health. The severe form of fatigue is known as 'exhaustion'. Fatigue literally means that the patient is 'tired all the time'. It is one of the most distressing symptom to the patient.

## COMMON CAUSES

1. Physiological–overwork, insomnia, boredom.
2. Pathological–
   a. Nutritional deficiency, dyselectrolytaemia
   b. Congestive cardiac failure (CCF), hepatic failure, uraemia, malignancy, immunocompromised states (e.g. AIDS), sleep-apnoea syndrome
   c. Myxoedema, diabetes mellitus, Addison's disease, thyrotoxicosis
   d. Tuberculosis, brucellosis, post-viral infectious states (e.g. influenza or infectious mononucleosis), collagen vascular diseases (e.g. SLE)
   e. Anaemia, lymphoma and leukaemias, terminally-ill patients
   f. Multiple sclerosis, myasthenia gravis, myopathies, poliomyelitis
   g. Functional–anxiety, sleep disorders, depression, chronic fatigue syndrome (CFS)
   h. Drugs—beta-blockers, sedatives, corticosteroids, α-methyldopa, antihistaminics, anti-epileptic drugs
   i. Terminally ill patients—disseminated carcinomatosis.

* At least in 50% cases the cause is functional, i.e. depression, anxiety or somatoform disorders

## UNDERLYING MECHANISM

1. Accumulation of lactic acid in muscles and circulation

2. Deficiency of oxygen
3. Creatinine depletion form muscles
4. Tumour necrosis factor and cytokines.

## FATIGUE IN CCF

1. Inadequate systemic perfusion
2. Sleep disturbance (due to paroxysmal nocturnal dyspnoea, orthopnoea, decubitus angina, nocturia)
3. Side-effects of beta-blockers
4. Dyselectrolytaemia due to diuretic therapy
5. Systemic manifestations of SBE.

## CLUE TO DIAGNOSIS

1. Functional–more at rest, disappears on activity, clinically WNL, investigations NAD (WNL = within normal limit, NAD = no abnormality detected).
2. Cachexia with loss of weight–tuberculosis, haematological or systemic malignancy.
3. Tremor + muscle cramps → dyselectrolytaemia.
4. Females in their 20-50 years of age → fatigue after minimal exertion–chronic fatigue syndrome (vide chapter on 'Diffuse aches and pains').
5. Hypotension + hyperpigmentation–Addison's disease.
6. ↑ Appetite + weight loss → thyrotoxicosis.
7. Polyuria + polyphagia + polydipsia → diabetes mellitus (fatigue may be presenting complaint).
8. Frequent awakening at night + snoring + pauses in breathing during sleep + day-time sleepiness → sleep-apnoea syndrome.
9. Recent H/O viral illness + tiredness goes away after few weeks or month → post-viral.
10. Early morning wake-up + tiredness maximum in the morning which persists all along the day + anorexia → depression.

## ASSESSMENT

Fatigue is a subjective symptom, and even objective changes like loss of body weight may be absent. So, clinically assessment should rely on self-reporting by the patient. Scales which measure fatigue, for example, Edmonton Functional Assessment tool, the Fatigue Self-Report scales are useful in research rather than clinical purposes. The 'Karnofsky

Performance Status' is a simple performance assessment clinical scale with questions like–"how much of the day does the patient spend in bed?" such a scale having grading, and allows assessment over time by third parties.

## TREATMENT

1. Graded exercise programme
2. Treatment of underlying aetiology
3. Psychotherapy (e.g. CFS)
4. Antidepressants (e.g. SSRI), tranquilizers, anti-anxiety drugs (e.g. clonazepam, buspirone), vitamins and electrolytes powder as and when necessary.

—— o ——

CHAPTER 26

# Flushing of Face

**FIGURE 26.1:** A patient of Fallot's tetralogy complicated by cyanosis, polycythemia (suffused conjunctiva) and respiratory distress

## DEFINITION

It is a slowly spreading erythema of the skin of face (often accompanied by neck and upper anterior chest) due to temporary capillary dilatation. Sometimes, it may be associated with light-headedness, tinnitus, tremulousness, nausea and a sense of suffocation. Flushing is common in females in comparison to males.

## CLUE TO DIAGNOSIS

Take H/O alcohol intake, sun-exposure, H/O treatment with disulfirum, intake of metronidazole while taking alcohol, menopause, emotional outburst (blushing), diabetes mellitus.

## DIFFERENTIAL DIAGNOSIS

1. Alcohol abuse
2. Menopausal syndrome: 'hot flushes' + perspiration
3. H/O intake of alcohol, especially while on treatment with chlorpropamide, metronidazole or disulfiram
4. Carcinoid syndrome: classic triad is flushing, diarrhoea and valvular heart disease; wheeze +, telangiectasia +
5. Autonomic nervous system dysfunction: with special reference to diabetes mellitus
6. Hormone-secreting tumours like pheochromocytoma (H/O episodic hypertension or fainting), medullary carcinoma of thyroid, VIPomas or Zollinger-Ellison syndrome
7. Systemic mastocytosis: with pruritus, recurrent headache, lower abdominal crampy pain and palpitations
8. Hypoglycaemia
9 Rosacea: flushing after taking tea, coffee (usually postprandial).

## FLUSHED FACE APPEARANCE

- Menopause, embarrassment, sunburn, healthy normal persons
- High altitude
- Cushing's syndrome
- Myxoedema
- Mitral senosis
- Chronic alcoholism
- Polycythemia vera
- Thyrotoxicosis
- Carcinoid syndrome
- Systemic mastocytosis
- Systemic hypertension
- Use of corticosteroid.

## ROLE OF URINE EXAMINATION

1. Sugar–diabetes mellitus
2. Alcohol–chronic alcohol abuse

3. 5-HIAA (hydroxyindoleacetic acid)–carcinoid syndrome
4. VMA (vanillylmandelic acid)–pheochromocytoma.

## DRUG-INDUCED/FOOD-INDUCED FLUSHING

### *Drug-induced*

- Nicotinic acid
- Nifedipine
- Bromocriptine
- Amyl nitrite
- Diltiazem
- Levodopa
- Monosulfiram (tetmosol → anti-scabetic)–after percutaneous absorption when used as soap.

### *Food-induced*

Chinese restaurant syndrome after ingestion of monosodium glutamate (present in azina moto)

### *Alcohol-induced*

Alcohol consumed with or without chlorpropamide, metronidazole or disulfiram.

—— o ——

CHAPTER 27

# Foul Breath

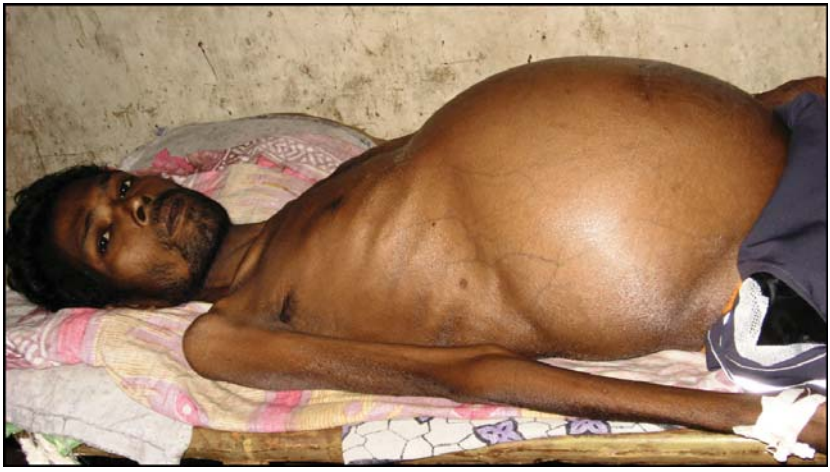


**FIGURE 27.1:** A sallow and dehydrated patient of cirrhosis of liver with profound muscle wasting; the thin limb with protuberant abdomen (with venous prominence) mimics a 'spider man'

## SYNONYM

- Halitosis
- Malodorous breath.

## COMMON CAUSES

Normal breath should be devoid of any kind of odour. The causes of halitosis are –

- Smoking, alcoholism
- Decomposed food debris collected in between teeth

- Consumption of onion, garlic, meats
- Stomatitis, gingivitis, septic tonsillitis, caries tooth, pyorrhoea, diphtheria
- Vincent's angina
- Sinusitis
- Atrophic rhinitis
- Carcinoma of the tongue
- Achalasia cardia, stasis in pharyngeal pouch, pyloric stenosis
- Bronchiectasis, lung abscess, respiratory tract infections, pulmonary tuberculosis
- Gastro-colic fistula, intestinal obstruction
- Hepatic failure
- Diabetic ketoacidosis, starvation ketosis
- Uraemia
- Septicaemia, peritonitis
- Anxiety or depression (functional).

## SPECIAL CHARACTERISTICS OF MALODOROUS BREATH (SMELL AS A PHYSICAL SIGN)

1. Diabetic ketoacidosis: sickly-sweet, fruity odour
2. Uraemia: Ammoniacal, fishy or urinany odour
3. Hepatic failure: Sweetish-faecal small → 'fetor hepaticus' (smell of a dead mouse)
4. Gastro-colic fistula or intestinal obstruction: Faeculent-foul smell
5. Bronchiectasis/lung abscess/sinusitis/atrophic rhinitis: Putrid smell
6. Gingivostomatitis/tonsillitis: Foul smelling offensive breath
7. Pulmonary tuberculosis: Cinnamon-like breath
8. Arsenic/thallium/phosphorous poisoning: Garlic-like (from body)
9. Acute alcoholism: Ether like smell from breath
10. Maple-syrup urine disease: Burnt sugar odour of urine (i.e. from body).

## CLUE TO DIAGNOSIS

- History (smoking, diabetes mellitus, odynophagia, profuse expectoration, vomiting)
- Examination
  - Oral cavity (e.g. gingivostomatitis, caries, tartar, malignancy)
  - Nose (e.g. atrophic rhinitis, sinusitis)
  - Dehydration (e.g. diabetic ketoacidosis)

- Cervical lymph nodes (e.g. carcinoma of the tongue)
- Pallor (e.g. uraemia, cirrhosis of liver)
- Jaundice (e.g. hepatic failure)
- Tremor (e.g. flapping → hepatic failure or uraemia)
- Oedema (e.g. uraemia, hepatic failure or diabetes mellitus)
- Auscultation of respiratory system (e.g. bronchiectasis or lung abscess)
- Auscultation of CVS (e.g. pericardial rub from uraemia)
- Hepatosplenomegaly (e.g. cirrhosis of liver with hepato-cellular failure)
- Ankle jerks (peripheral neuropathy from uraemia, diabetes)

## INVESTIGATIONS

- Blood for TC, DC, ESR, atypical cells (e.g. leukaemia with bad oral hygiene), sugar (e.g. diabetes mellitus), urea and creatinine (e.g. uraemia), LFT (e.g. hepatic failure)
- X-ray of sinuses, lung (e.g. bronchiectasis or lung abscess)
- Straight X-ray of abdomen (e.g. intestinal obstruction)
- Urine for ketone bodies and sugar (e.g. diabetic ketoacidosis), routine examination (e.g. proteinuria and casts reflect intrinsic renal disease).

## MANAGEMENT

- Often very difficult to treat; recalcitrant
- ℞ of aetiology
- Frequent gargling with some antiseptic solutions
- Keeping clove or elaichi within the mouth
- Dryness of the mouth can be alleviated by peppermints.

## PEARL

In some cases, the cause of halitosis remains undiscovered even after meticulous search → the clue may be hidden in 'poor oral hygiene' or the patient is suffering from functional disorder.

—— o ——

CHAPTER 28

# Genital Ulcer

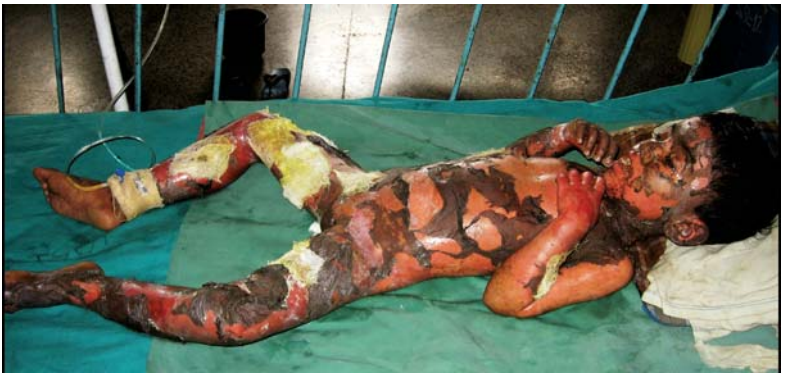


**FIGURE 28.1:** Toxic epidermal necrolysis – childhood type (widespread subepidermal blistering and sloughing of most of the skin)

## PREFACE

Genital ulcer may be infective or non-infective as well as ulcerative or non-ulcerative.

## POSSIBILITIES

### INFECTIVE

- Herpes simplex (genital herpes) → primary, recurrent
- Syphilis → primary chancre, secondary mucous patches, tertiary gumma
- Chancroid (soft sore)
- Lymphogranuloma venereum

- Granuloma inguinale
- Herpes zoster
- Condyloma acuminatum
- Scabies
- Others: balanitis (by Vincent's organism), fungal infection, tuberculosis.

### NON-INFECTIVE

- Behcet's disease
- Malignancy
- Trauma
- Toxic epidermal necrolysis
- Stevens-Johnson syndrome
- Others: pemphigus vulgaris, lichen planus, psoriasis, functional (psychosis).

### EVALUATION

Fever, pain, pruritus, malodour, genital swelling, joint pain, skin rash and eye symptoms should be enquired into. Ask for dysuria, haematuria and joint pain. Drug history and H/O allergy should be asked for. A detailed sexual history (number and types of sexual contacts) with partner's sex, regular/casual partner, use of condom/contraception, history of travel abroad, hepatitis B vaccination status should be explored. O/E → oral cavity, throat, skin, lymph nodes, inguinal, genital and perianal areas should be meticulously examined. Penile foreskin should be retracted, urethral meatus should be looked for any dischage and scrotal contents should be palpated for consistency of the testes. If history of anal intercourse is obtained, a rectal examination or proctoscopy should be performed.

### BREAK-UP

1. Syphilis: The 'primary' chancre is caused by Treponema pallidum with an incubation period of 9 to 90 days. Usually, one ulcer present over the coronal sulcus or glans penis (or any part of penis) → sharply demarcated, oval, regular with dull, non-purulent, relatively non-vascular base. Induration +, may bleed on palpation. Inguinal nodes are firm and non-tender (bilateral affection). The 'secondary' syphilitic lesion appears 6-8 weeks after the primary one → dull red, painless,

indurated and is the most infectious form. 'Gumma' occurs 3-10 years after the primary lesion →non-tender, oval, punched-out ulcers often associated with a wash leather slough.

2. Chancroid: Caused by Haemophilus ducreyi with an incubation period of 1-14 days. Single or multiple (usually) ulcers, few millimeter to 2 centemeter, tender and painful with shaggy undermined edge which bleeds easily; prepuce, frenum and external meatus are commonly involved. Inguinal lymph nodes (unilateral affection usually) are tender and may suppurate.
3. Lymphogranuloma venereum: Caused by *Chlamydia trachomatis* with an incubation period of 3 days to 6 weeks. Usually single oval, non-indurated ulcer of 2-10 mm size is present on coronal sulcus, glans penis, prepuce or shaft of the penis. Chains of enlarged inguinal lymph nodes (buboes) above and below the inguinal ligament ("the sign of the groove") may be seen.
4. Granuloma inguinale (donovanosis): Caused by Calymmatobacterium granulomatis with an incubation period of 1-4 weeks. Single or multiple ulcers are seen which are preceded by papule and vesicle. Ulcers are non-tender and of variable size with irregular edge, elevated and velvety; they bleed readily on touch. The lesion may spread to other areas by autoinoculation ('kissing' lesion). Inguinal lymphadenopathy is uncommon though 'pseudobubo' may be seen ('bubo' is seen in plague, tularaemia and lymphogranuloma venereum).
5. Herpes genitalis: Caused by herpes simplex virus type II and having an incubation period of 2-7 days. It commences as an oval vesicle with surrounding erythema over glans and shaft of the penis → ultimately the vesicle breaks down to develop into multiple, superficial erosions. The base of the erosions are serous, erythematous and non-vascular. Induration is absent; very often bilateral, firm, tender lymphadenopathy is present in the inguinal region.

## POSSIBLE CAUSES OF GENITAL DISCHARGE (FEMALE)

- Psychological: cervical mucus (i.e. excessive normal secretion), vaginal transudation
- Pregnancy
- Sexual response
- Infection *(Candida trichomonas)*
- Cervical erosion

- Foreign body, e.g. tampon, cervical cap, ring pessary
- Malignancy of cervix (or cervical polyp)
- IUCD
- Fistula (Crohn's disease, rectovaginal fistula)
- Chemical irritation (e.g. spermicide).

## URETHRAL DISCHARGE

- Urethritis (gonococcal, chlamydial, trichomonal, ureaplasma urealyticum)
- Cystitis, prostatitis, vaginitis (e.g. candidiasis)
- Trauma (e.g. masturbation, foreign body)
- Urethral stricture
- Meatal chancre (Treponema pallidum)
- Idiopathic.

## D/D OF INGUINAL SWELLING (I.E. LUMP IN THE GROIN)

- Hernia (inguinal, femoral) → impulse on coughing, reducible
- Inguinal lymphadenopathy (bubo, pseudobubo, lymphoma)
- Femoral artery aneurysm → expansile pulsation.

—— o ——

CHAPTER 29

# Gingival Bleeding

## PATHOPHYSIOLOGY

The connective tissue of gum is highly supplied by capillaries. Bleeding from gingiva may be due to physical injuries (e.g. brushing, trauma), thermal injuries (e.g. consumption of hot tea) or may be precipitated by various infections (e.g. gingivitis). 'Platelet plug' formation at the bleeding site is the most important haemostatic mechanism which comes to halt bleeding. Coagulation disorders are usually associated with delayed haemorrhage; in clinical practice, severe degree of clotting factor deficiencies and platelet disorders result in gum bleeding. Connective tissue disorders hampering the supporting tissues of gingival capillaries may result in gum bleeding in day to day practice.

## PROBABLE AETIOLOGY

1. Thrombocytopenias–idiopathic thrombocytopenic purpura (ITP), aplastic anaemia, acute leukaemias.
2. Platelet functional defects –
   a. Adhesion defect, e.g. von-Willebrand's disease, Bernard-Soulier syndrome
   b. Aggregation defect, e.g. thrombasthania (Glanzmann's syndrome)
3. Coagulation disorders, e.g. haemophilia, Christmas disease, afibrinogenaemia, vitamin K deficiency, anticoagulation therapy.
4. Vessel wall disorders–scurvy, Cushing's syndrome, Henoch-schonlein purpura, dysproteinaemias (e.g. multiple myeloma), Ehlers-Danlos syndrome, pseudoxanthoma elasticum.
5. Gingival bleeding due to gum inflammation, e.g. gingivitis/periodontitis, pregnancy, Vincent's angina, poor oral hygiene.

* In clinical practice, thrombocytopenia, the first cause (e.g. ITP) of gum bleeding comes in the mind of clinicians.

## GUM BLEEDING + HYPERTROPHY

- Scurvy
- Acute monocytic leukaemia
- Poor oral hygiene
- Pregnancy.

## CLINICAL CLUE

- Bleeding from other sites: platelet disorder, coagulation disorder
- Profuse and induced bleeding: platelet disorder
- Persistent bleeding: coagulation disorder
- Associated gum hypertrophy: probable causes mentioned above
- Splenomegaly: AML, ALL, blast crisis of CML and CLL, SLE, hypersplenism, lymphoma, myelofibrosis
- Absence of splenomegaly: aplastic anaemia, ITP, coagulation disorder
- Sternal tenderness: acute leukaemias, CML
- Associated haemarthrosis or muscle haematoma: coagulation disorder

## CLUE TO DIAGNOSIS BY INVESTIGATIONS

1. ↑ Bleeding time: Thrombocytopenia → low platelet count
   Platelet functional defects → platelet count WNL
2. ↑ Clotting time → coagulation factor disorders
3. Bone marrow exmination
   a. Hypocellular–aplastic or hypoplastic anaemia
   b. > 30% blast cells–acute leukaemias
   c. Normal or ↑ megakaryocytes–ITP
   d. Normal or hypercellular–hypersplenism
4. Vitamin C level estimation in WBC–scurvy (low level).

## TREATMENT

- ℞ of aetiology, e.g. antibiotics for gingivitis, platelet transfusion for platelet number and function disorders, specific clotting factors to be given in coagulation disorders
- Vitamin C 100 mg, tds orally in scurvy
- Maintenance of good oral hygiene by frequent gargling/application of boro-glycerine as and when necessary. Avoid brushing the teeth in active bleeding.

## MESSAGE

- Don't carry out 'occult blood test in stool' in the presence of gum bleeding
- Though, initially looks harmless, it may come out to be a serious illness.

—— o ——

CHAPTER 30

# Gum Hypertrophy

## PROLOGUE

True hypertrophy of gum is rare. Gingival hypertrophy may be due to infiltration by a) fibrous tissue and b) cellular elements. The most common cause of gum hypertrophy is infection associated with the dental structures.

## CLUE TO DIAGNOSIS

- With gum bleeding:
  1. Scurvy (soft, red, spongy and hypertrophied)
  2. Acute monocytic leukaemia
  3. Poor oral hygiene (e.g. caries tooth)
  4. Pregnancy
  5. Congenital cyanotic heart disease (spongy and haemorrhagic).
- Without gum bleeding:
  1. Phenytoin therapy in epileptics (firm and hypertrophied)
  2. Nifedipine or amlodipine therapy in systemic hypertension
  3. Cyclosporine therapy
  4. Idiopathic familial fibromatosis
  5. Infiltration of gum by haemangioma
  6. Mouth breathers since childhood
  7. Ill-fitted dentures.

## GUM BLEEDING (COMMONLY SEEN IN DAY TO DAY PRACTICE)

1. Gingivitis, periodontitis, injury, tartar, pyorrhoea
2. Idiopathic thrombocytopenic purpura (ITP)
3. Acute leukaemias

4. Aplastic anaemia
5. Haemophilia
6. Vincent's angina (painful ulcero-membranous gingivitis due to spirochaete and fusiform bacillus infection)
7. Anticoagulant therapy
8. Hyperviscosity syndrome.

## GREY LINE IN THE GUM

- Tartar on teeth
- Bismuth therapy.

## DARK LINE IN THE GUM

- Mercury poisoning.

## BLUE LINE IN THE GUM

- Chronic lead poisoning.

* If a piece of white paper is inserted between the gum and the teeth, the stippled line due to lead poisoning will be more pronounced whereas colour change due to tartar on the teeth will disappear.

## GUM PIGMENTATION

1. People with dark complexion
2. Addison's disease
3. Peutz-Jeghers syndrome
4. Nelson's syndrome
5. Chronic betel-leaf chewer.

## PYORRHOEA ALVEOLARIS

Gingivitis → bleeding → swollen interdental papillae → as time progresses food debris, bacteria and pus collect between the teeth and the gum margin → 'pyorrhoea alveolaris'→ tender, swollen gum with halitosis → pus may be aspirated and lead to pneumonia → teeth loosen and may be aspirated and obstructed within bronchus (non-resolving pneumonia) → transient bacteraemia from pyorrhoea alveolaris may lead to infective endocarditis in a patient with valvular heart disease.

* Healthy gums are pink, adhere to teeth and have a sharp border. In gingivitis, gums are retracted and sometimes pus can be squeezed on pressing the gum (pyorrhoea alveolaris).

—— o ——

CHAPTER 31

# Hardness (Thickening) of Skin

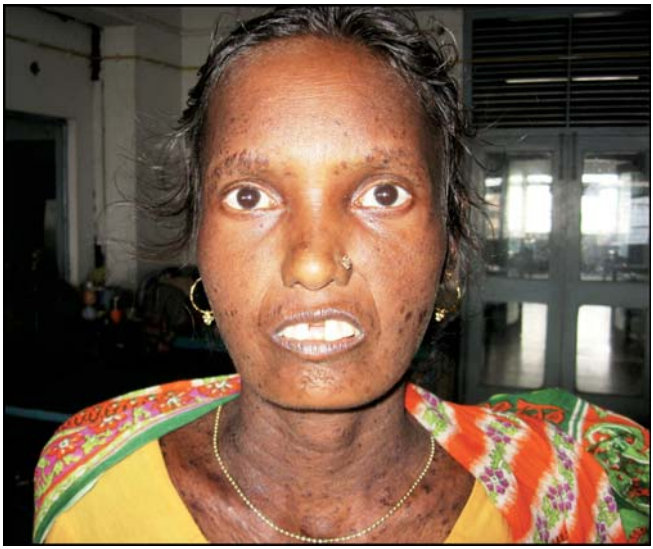


**FIGURE 31.1:** Mask-like face, absence of normal wrinkling of skin, microstomia, loss of eyebrows and pigmentation – facies of scleroderma

## COMMON POSSIBILITIES

1. Scleroderma and morphea (localised form of progressive systemic sclerosis)
2. Lymphoedema (may be in the background of carcinomatous or lymphomatous metastasis into skin; peau d'orange-like skin)
3. Vascular insufficiencies (chronic)
4. Recurrent cellulitis associated with venous stasis
5. Lipoid proteinosis (hyaline deposition into skin and mucous membrane, firm tongue which is difficult to protrude, hoarseness

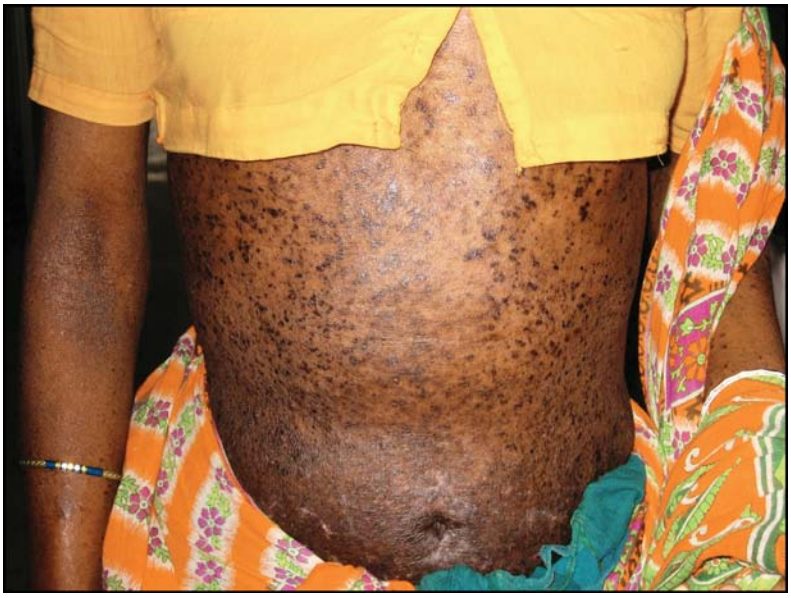


**FIGURE 31.2:** Calcinosis cutis in progressive systemic sclerosis

of voice; face/dorsum of hand and feet, and eyelid margins are commonly involved)
6. Carcinoid syndrome (in carcinoid tumour of lung or gut, there may be violaceous cyanosis-like discolouration of skin)
7. Porphyria cutanea tarda (presence of fragility of sun-exposed skin)
8. Chronic Lyme disease
9. Chronic graft-versus host disease
10. Eosinophilic-myalgia syndrome (as a result of tryptophan therapy)
11. Bleomycin or polyvinyl chloride-induced
12. Pseudo-scleroderma (amyloidosis, scleredema, scleromyxoedema)
13. Keloid–localised hardening.

## 'STIFF AND FIRM TONGUE' ON PALPATION

1. Primary amyloidosis
2. Mucopolysaccharidosis, e.g. Hurler syndrome
3. von Gierke's disease (glycogen storage disease)
4. Cretinism, acromegaly and myxoedema (when associated with macroglossia)
5. Carcinoma of the tongue
6. Lipoid proteinosis.

## DIFFERENTIAL DIAGNOSIS OF SCLERODERMA (THICKENING OF SKIN)

A. Disorders associated with skin thickening in fingers and hands:
- Digital sclerosis of diabetes mellitus
- Vinyl chloride-induced
- Bleomycin-induced
- Chronic reflex sympathetic dystrophy
- Mycosis fungoides
- Adult celiac disease
- Vibration disease
- Amyloidosis.

B. Disorders associated with generalised skin thickening but sparing fingers and hands:
- Scleredema adultorum of Buschke
- Scleromyxoedema
- Eosinophilic fascitis
- Eosinophilic-myalgia syndrome
- Pentazocine-induced
- Chronic graft-versus host disease
- Porphyria cutanea tarda
- Amyloidosis.

C. Disorders associated with asymmetric skin change:
- Linear scleroderma
- Morphea
- Coup de sabre (linear scleroderma affecting face in children).

## WHAT IS SCLEREDEMA?

Scleredema adultorum of Buschke is painless oedematous induration of face, neck, trunk and proximal extremities (sparing hands and feet), which occurs commonly in children. This may be associated with previous streptococcal infection in some cases. Sparing of hands/feet and absence of Raynaud's phenomenon differentiate this condition from scleroderma. Histopathology shows minimal epidermal changes with markedly thickened dermis as well as accumulation of proteoglycan, hyaluronic acid and collagen.

—— o ——

CHAPTER 32

# Head-nodding

## DEFINITION

To and fro movement of head is known as head-nodding.

## POSSIBLE ASSOCIATIONS

1. May be a part of tics (habbit spasm). Tics are brief, sudden, rapid, intermittent and stereotyped movements (motortics) or sounds (vocal tics)
2. Physiologic–mannerisms, gestures
3. Mental retardation, autism
4. Creutzfeldt-Jacob disease, encephalitis
5. Titubation–head-nodding in antero-posterior direction, and is found in midline cerebellar disorder
6. de-Musset's sign–to and fro head-nodding synchronous with carotid pulsation, and is seen in aortic regurgitation (named after a French poet)
7. Parkinsonism
8. Drug-induced–anticonvulsants, levodopa, cocaine, dopamine-receptor blocking drugs
9. Benign or familial disorder of tremor.

## COMMON MOTORTICS

1. Blinking
2. Facial grimace
3. Shoulder movement
4. Nose twitching

5. Sniffing
6. Head shaking/jerking
7. Torticollis.

## HEAD TILT

1. Extension of head (head tilt) done along with lifting of chin (chin lift) in maintenance of airways in Basic Life Support (BLS) of cardiopulmonary resuscitation (CPR).
2. Head is tilted (head tilt) towards the direction of action of weak extraocular muscle (e.g. in IVth cranial nerve or superior oblique palsy) to overcome diplopia.
3. In cerebellar hemispheric lesion, the patient may have head tilt towards the side of lesion.

## HEAD RETRACTION

1. Severe meningitis and meningism
2. Subarachnoid haemorrhage
3. Asphyxia especially in children with bronchiolitis, bronchopneumonia or foreign body in larynx
4. Intermittent retraction:
   - Tetanus
   - Rabies
   - Strychnine poisoning
   - Spasmodic torticollis (wry neck) and torsion spasm
5. Cerebellar pressure cone syndrome (resulting from ICSOL, haematoma, cerebral abscess, cerebral oedema or hydrocephalus)– lumbar puncture may result in death
6. Spinal disorder involving upper part of spine

* All of the above produce stiffness of neck.

—— o ——

CHAPTER 33

# Heel Pain

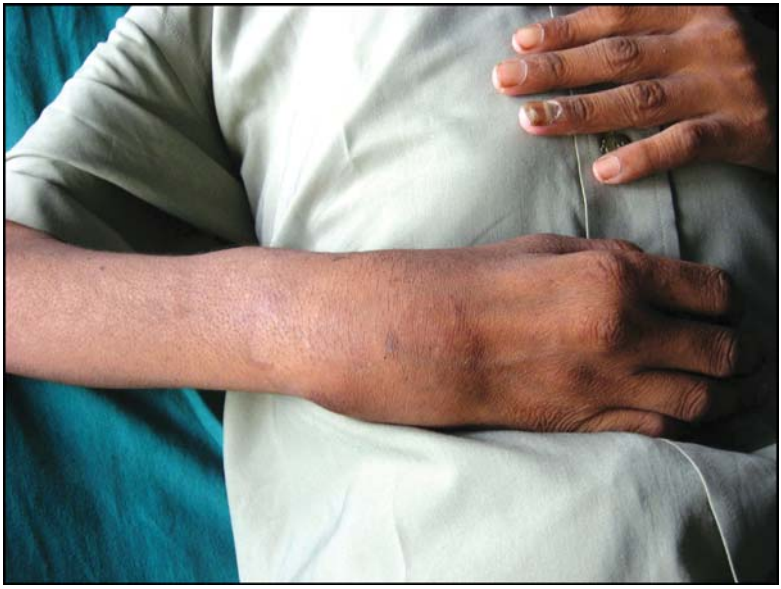


**FIGURE 33.1:** Synovitis of right wrist joint in seronegative spondyloarthropathy

## AETIOLOGY

- Dermatological:
  - ulcer
  - fissure
  - keratosis
  - verruca
- Connective tissue:
  - panniculitis

plantar fascitis
bursitis
enthesopathy
stress fracture
osteopenia
malignant bone tumour
Paget's disease
calcanean spur
plantar nerve entrapment
painful peripheral neuropathy
tarsal tunnel syndrome
gout

- Miscellaneous:
  acute osteomyelitis
  plantar bursitis
  plantar abscess
  foreign body
  psychogenic
  idiopathic.

## COMMON CAUSES OF HEEL PAIN

1. Plantar fascitis—an enthesitis at the tendon insertion into calcaneum. Pain and tenderness occur in the midline during standing or walking. It may occur as an isolated entity or associated with seronegative spondyloarthropathy (SpA). Radiology may show soft tissue clacification. Treatment modalities are sensible restriction of activity, gentle stretching exercise, NSAIDs, local steroid or lignocaine injection, lifting of heel, night brace and daytime cast brace. Usually self-limiting disease.
2. Calcanean spur—traction lesions at the insertion of plantar fascia; painful after trauma.
3. Calcanean bursitis—it is a pressure-induced bursa, and compression of the heel pad from sides become painful (ref: midline pain in plantar fascitis).

## PAIN BEHIND THE HEEL

1. Achilles tendonitis–it is an enthesitis at tendon insertion into calcaneum. Heel raising reduces pain; may be associated with seronegative SpA. Steroid injection may be beneficial.

2. Achilles tendonosis–a painful and tender swelling may present few centemeter above the tendon's insertion. Ultrasonography is helpful and local steroid injection may result in rupture of tendon. Advice to avoid jumping and walking barefooted.
3. Achilles' bursitis–lies anterior to the tendon; steroid injection is beneficial and can be done safely.
4. Sever's disease–a traction apophysitis of Achilles tendon; affects young people.

## PAIN IN THE BALL OF THE FIRST TOE

1. Osteoarthritis of 1st metatarsophalangeal (MTP) joint
2. Gout
3. Hallux rigidus (a stiff, dorsiflexed great toe)
4. Hallux valgus
5. Metatarsalgia (as a result of high heel, trauma or rheumatoid arthritis)
6. Pressure-induced bursa, corn or callosities
7. Septic arthritis of 1st MTP joint.

## PAIN IN THE SHIN BONE

1. Referred pain from above (e.g. osteoarthritis of knee joint or sciatica)
2. Periostitis due to any cause, trauma, osteomyelitis
3. Hypertrophic pulmonary osteoarthropathy
4. Tabes dorsalis (lightning pain, stabbing in nature)–rare
5. Associated with erythema nodosum (panniculitis).

—— o ——

CHAPTER 34

# Herpes Labialis

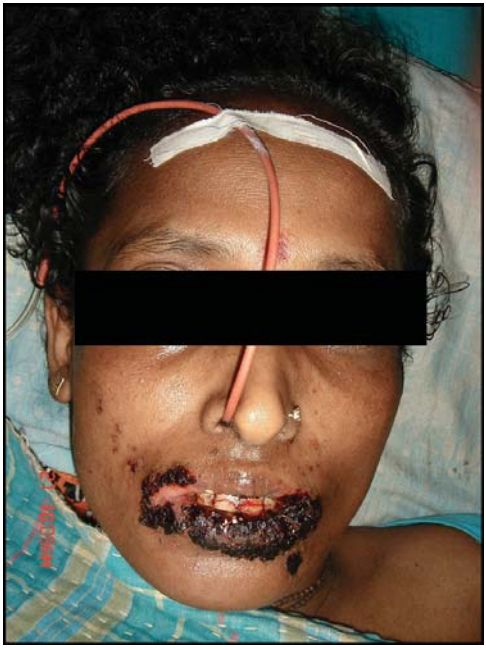


**FIGURE 34.1:** Extensive herpes labialis with crusting in a moribund patient with jaundice

## DEFINITION

Small, grouped, closely-set vesicles on an erythematous base found on the skin of the face innervated by cutaneous branches of the maxillary and mandibular divisions of the Vth cranial nerve, particularly around the lips and is due to herpes simplex virus-type 1.

## CHARACTERISTICS

- Often recurrent
- Usually no constitutional symptoms
- Starts with prodromal symptoms like pain, burning, tingling or itching
- Usually last for 6-48 hours
- Rapid vesiculation, pustulation and crusting
- Complete healing takes about 10 days
- Heals without residue
- Caused by herpes simplex virus - type 1
- Tongue, gum and palate may be involved
- Trigger factors:
  - Fever
  - Sunlight
  - Stress
  - Menstruation
  - Local trauma.

## CLINICAL ASSOCIATIONS

1. Influenza
2. Acute lobar pneumonia (often gives clue to the side affected, e.g. left side of the lip is affected in left lung involvement)
3. Meningococcal meningitis
4. Malaria
5. Weil's disease
6. Mycoplasma pneumoniae infection
7. AIDS.

## SYNONYMS

Fever blisters, cold sore.

## DIFFERENTIAL DIAGNOSIS

- Herpes zoster infection (does not recur in the same site as herpes labialis)
- Hand, foot and mouth disease caused by Coxsackievirus (vesicular eruptions in hands and feet as well)
- Impetigo (may be confused with herpes labialis infected by staphylococcus).

## TREATMENT

Apply solutions of 5-iodo-2-deoxy-uridine (IDU).

## CLINICAL FEATURES OF HERPESVIRUS HOMINIS (HERPES SIMPLEX)

### *HSV-1*

- Acute gingivostomatitis
- Rhinitis
- Keratoconjunctivitis
- Meningoencephalitis
- Eczema herpeticum (Kaposi's varicelliform eruption)
- Traumatic herpes (includes herpetic whitlow, generalised cutaneous herpes simplex and herpes gladiatorum)
- Oesophageal ulceration or interstitial pneumonia in immuno-suppressed patients.

### *HSV-2*

- Vulvovaginitis (HSV-2 infection is the most common cause of genital vesicles/ulcer in women)
  - Associated with increased risk of carcinoma of the cervix
  - Tzanck smears are positive
  - D/D with CMV, varicella-zoster virus, variola virus, drugs or contact dermatitis or Behcet's disease-induced genital vesicles/ulcers
  - Treated with acyclovir
- Aseptic meningitis
- Mild hepatitis.

## CHEILOSIS OR CHEILITIS (CONDITIONS TO BE LOOKED FOR)

1. Iron deficiency anaemia
2. Riboflavin, nicotinic acid, folic acid, vitamin $B_{12}$ or pyridoxine deficiency
3. Solar or actinic cheilitis:
   - Uncommon
   - Cracked lower lip
   - Yellow-white thickenings
   - Scaling and crusting
   - May be a manifestation of light eruption, xeroderma pigmentosum or secondary to use of lipsticks
   - Long standing cheilitis → warty lesions → may become malignant
4. May be a part of stomatitis.

## SORE MOUTH

1. Aphthous ulcers
2. Infections–candidiasis, Vincent's angina, dental sepsis, HSV-1, herpangina, Coxsackievirus
3. Traumatic ulcers due to ill-fitted dentures
4. Angular stomatitis
5. Sore tongue from vitamin B-complex or iron deficiency, malignancy, glossitis
6. Miscellaneous–Drug allergy (sulphonamides, penicillins, cytotoxics), recurrent gingivitis from blood dyscrasia, persistent ulceration from agranulocytosis, Behcet's disease, inflammatory bowel disease and dermatological conditions like pemphigoid, erythema multiforme, pemphigus vulgaris and lichen planus.

## SORE THROAT

A very common presentation in clinical practice and most of the cases are viral in origin (pharyngitis), self-limiting and need no specific treatment. The clinician should be careful so that any serious underlying life-threatening condition is not overlooked. The common causes are:

1. Viral–adenovirus, herpes simples, Epstein-Barr virus
2. Tonsillitis (streptococcal commonly)
3. Infectious mononucleosis
4. Candidiasis (thrush)
5. Vincent's angina (spirochaetes and fusiform bacilli)
6. Diphtheria
7. Agranulocytosis, acute leukaemias, aplastic anaemia.

* Sore throat is associated with odynophagia (painful swallowing).

## PIGMENTATION OF ORAL CAVITY

1. Racial
2. Addison's disease
3. Peutz-Jeghers syndrome
4. Melanotic macule
5. Chloasma
6. Drug reaction: chlorpromazine, busulphan, quinacrine
7. Miscellaneous: Lead line, amalgam tattoo, neurofibromatosis.

—— o ——

CHAPTER 35

# Hiccough

## SYNONYMS

Hiccup, singultus.

## DEFINITION

Abrupt, involuntary, synchronous contraction of the diaphragm and the inspiratory intercostal muscles, followed by immediate closure of the glottis → the glottic closure is responsible for characteristic inspiratory sound and associated discomfort. Though hiccough is a normal, benign and transient physiological phenomenon, many a time persistent hiccough pose problem in management.

## POSSIBLE ASSOCIATIONS

A. Transient: Ingestion of chilli/irritant spicy food in a quick surcession, gastric distension after a rapid meal, sudden excitement, ingestion of alcohol, oesophageal obstruction.
B. Persistent:
   a. CNS disorders–CVA, lateral medullary syndrome, encephalitis, posterior fossa SOL or lower brainstem lesion.
   b. Thoracic disorders–Basal pneumonia (diaphragmatic pleurisy), AMI (mainly in inferior wall involvement), pleurisy (e.g. diaphragmatic), pericarditis, aneurysm of the aorta, diaphragmatic herniation or irritation (e.g. subphrenic abscess), mediastinal tumour.
   c. Metabolic disorders–Uraemia, hyperventilation.
   d. Abdominal disorders–Acute gastritis (e.g. NSAID-induced), gastric ulcer/carcinoma, acute hepatitis, amoebic liver abscess, intestinal obstruction, acute pancreatitis, acute peritonitis.

e. Miscellaneous–Psychogenic, idiopathic, surgery, general anaesthesia, irritation of external auditory canal, drug-induced (benzodiazepines, barbiturates), epidemic hiccough (viral infection related to influenza and encephalitis, occurring in epidemics), high pyrexia, septicaemia.

## COMMON CAUSE OF HICCOUGH IN CLINICAL PRACTICE

- Overdistension of stomach
- Acute gastritis
- Chronic renal failure (uraemia)
- Diaphragmatic pleurisy
- CVA
- Idiopathic.

## MANAGEMENT

Recurrent hiccough is very distressing to the patient and difficult to manage.

1. Reassurance: The patient along with the relatives should be reassured.
2. Simple household remedies: Divert patient's attention (e.g. by conversation, sudden slapping), intake of ice-cold water, series of deep breath holding, Valsalva menoeuvre, lifting uvula with cold spoon, breathing in and out in a platic bag for 5 minutes, induction of vomiting by pharyngeal stimulation, spray of ethyl chloride under the costal margins, swallowing rapidly one teaspoonful of 'dry' granulated sugar or dry bread, drinking water without taking any breath; coughing, sneezing.
3. Local measures: Intake of local anaesthetic viscus, e.g. lignocaine, nasogastric suction followed by ice-cold stomach wash or alkaline stomach wash through a Ryle's tube.
4. Antacids/$H_2$-RA/PPI: Any liquid antacid (preferably containing oxethazaine) is given 2-4 tsf, 6-8 hourly daily, orally, or ranitidine 150 mg BDAC, or omeprazole 20 mg ODAC, orally. It is often advised to take the tablets with little or no water and irritation of the pharynx may end about of hiccough in a stubborn case.
5. Drugs or pharmacotherapy: Chlorpromazine (probably best tried first, as an 1V bolus; 25 mg, orally tds or 25 mg IM stat), baclofen (10 mg, orally tds), haloperidol (5 mg, IM stat or 0.25 mg, orally, tds), clonazepam (2 mg, orally, tds), amitriptyline, amantadine, quinidine (200 mg, orally tds), ondansetron (4 mg, IV, tds), anticonvulsants like

phenytoin sodium, carbamazepine, valproic acid are worth trying in resistant cases. Baclofen (beta-agonist) is an effective drug in the treatment of intractable hiccough.

6. Surgery: In a recalcitrant case, phrenic nerve block by bupivacaine or nerve section may be effective.
7. ℞ of the underlying cause.

## PROBABLE CAUSES OF BELCHING (ERUCTATION)

It is the forceful regurgitation (expulsion) of air from the stomach or oesophagus.

1. Consumption of carbonated beverages, hurried eating habit, gum chewing, ill-fitted dentures.
2. Addictions like smoking, chewing betel nut/pan; mouth breather.
3. Abdominal disorders: acute gastritis or duodenitis, hiatal hernia, chronic cholecystitis, irritable bowel syndrome, intestinal obstruction.
4. Psychogenic conditions like anxiety, emotional disturbances, depression.

* Avoidance of addiction and faulty habits as well as intake of conventional antacid containing methylpolysiloxane (MPS) many a time relieves the person.

—— o ——

CHAPTER 36

# Hirsutism

**FIGURE 36.1:** Moon face (Cushingoid features – facial plethora, hirsutism) after prolonged corticosteroid therapy in nephrotic syndrome

## HIRSUTISM

Hirsutism is the growth of hair in women in a pattern characteristic of men. In females, excess hair growth is seen in sides of the face, moustache and beard area (upper lip and chin), in between the breast, peri-areolar region, over the abdomen and in the extemities. It usually affects 10% women in the community.

## VIRILISATION

The characteristic features are:

- Frontal baldness
- ↑ in size of shoulder girdle muscles
- Coarsening of voice
- Acne and seborrhoea
- Hirsutism
- Clitoromegaly
- ↑ libido

* Congenital adrenal hyperplasia is the commonest cause of virilisation (associated with ↑ androgens with ↓ or normal glucocorticoids and mineralocorticoids).

## DEFEMINISATION (DIMINISHING FEMALE CHARACTERISTICS)

- ↓ in breast size
- Loss of female body contour
- Amenorrhoea.

## HYPERTRICHOSIS

Excessive hair growth in both sexes and is commonly due to anorexia nervosa, malnutrition, hypothyroidism, dermatomyositis, porphyria cutanea tarda, drugs (e.g. minoxidil, cyclosporin, phenytoin, diazoxide), underlying malignancy and Cushing's syndrome.

## SOME BASICS

Certain races (e.g. Mediterranean and Asian) have more male pattern hair growth and is not due to androgen excess but happens to be due to as a result of genetically determined altered sensitivity to androgens. Fetal hair is known as 'lanugo' hair. Hair can be categorized into 2 types: 'vellus' (fine, soft and non-pigmented) and 'terminal' (long, coarse and pigmented). In the lifetime, the number of hair follicle does not increase but the follicle size and type of hair changes in response to multiple factors, e.g. androgens (it can transform vellus hair into a terminal hair).

Three phases in the cycle of hair growth have been observed:

- Anagen (growth phase) = has 6 stages like pro-anagen (1 to 5), met-anagen (6) stages.
  Met-anagen to catagen phase = 2-10 years

- Catagen (involution or transition phase) = 6 weeks
- Telogen (rest phase) = 3 months.

## HORMONAL INFLUENCES ON GROWTH OF HAIR

A. Eyebrows, eyelashes, and vellus hair → androgen-sensitive.
B. Axillary and pubic hair → sensitive to low levels of androgens.
C. Hairs on face, chest, upper abdomen, and back → needs high level of androgens and thus seen in males.

N.B:

Nonsexual or neutral hairs are under the control of growth hormone, e.g. hairs on the scalp, eyelashes, forehead and lower part of the body in both sexes; whereas ambisexual hair, in both sexes, are under the influence of testosterone. The axillary and pubic hair in females are under the control of adrenal androgen and thus hair loss in those regions are seen in Addison's disease, whereas this does not occur in males because testosterone alone maintains the growth of hair in axilla and pubic region.

Remember: "androgen excess underlies most of the cases of hirsutism". Androgen excess in females lead to increased growth of hair in most of the androgen-sensitive sites (C above) except in the scalp region. In females, androgens are derived from ovaries and adrenal glands, as well as from peripheral conversion → hence, aetiology lies either in adrenal or in ovary.

## POSSIBLE ASSOCIATIONS

1. Familial and racial
2. Possible ↑ sensitivity to androgens (idiopathic or simple hirsutism)
3. Menopause
4. Polycystic ovarian syndrome (Stein-Leventhal syndrome)
5. Cushing's syndrome, hyperprolactinaemia, virilising ovarian tumour (e.g. arrhenoblastoma or hilus cell tumour), acromegaly, congenital adrenal hyperplasia, adrenal androgen secreting tumour (adenoma, carcinoma), true hermaphroditism
6. Obesity (in the adipose tissue, oestrogens are converted into androgens)
7. Drug-induced: phenytoin, oral contraceptive pills, androgens, diazoxide, cyclosporin, minoxidil, psoralens, anabolic steroids, corticosteroids.

## CLINICAL PRESENTATION

A. Hirsutism with virilisation:
- Severe polycystic ovarian syndrome (USG of ovary, ↑ LH, ↑ androgens)
- Congenital adrenal hyperplasia (↑ 17-hydroxyprogesterone)
- Ovarian tumour (USG/laparoscopy).

B. Hirsutism without virilisation:
- Idiopathic or simple (with normal menstruation, normal ovaries/adrenals)
- Familial (+ve family history of hirsutism in mother or grandmother)
- Mild polycystic ovarian syndrome (same as above)
- Drugs (hypertrichosis mainly, H/O specific drug intake)
- Congenital adrenal hyperplasia (late onset)–↑ 17-hydroxyprogesterone, after ACTH injection
- Adrenal tumours (CT scan of adrenals).

## OUTLINE OF INVESTIGATIONS IN HIRSUTISM

1. Serum androgens (very high in adrenal neoplasm or congenital adrenal hyperplasia).
2. Other hormones: TSH, ACTH, LH, FSH; a short ACTH stimulation test is helpful in congenital adrenal hyperplasia, and LH:FSH ratio > 3:1 very often suggests polycystic ovarian disease.
3. Serum insulin level and glucose tolerance test in polycystic ovarian disease → diagnose insulin resistance.
4. USG of ovaries (e.g. polycystic ovarian disease); CT scan of adrenal glands.
5. Laparoscopy (for diagnosis of ovarian pathology).
6. Biopsy of the ovary (e.g. ovarian neoplasm).

### *TREATMENT*

A. Non-pharmacological:
- Bleaching
- Shaving
- Chemical treatment (depilatory creams)
- Epilatory treatment (plucking, waxes, electrolysis and laser therapy)

* Electrolysis usually removes hairs permanently, especially in the hand of a skilled electrologist but the process is lengthy as well as expensive.

** At firsthand, stop the offending drug, if drug-induced.

B. Pharmacological:
- Oral contraceptive pills to suppress androgen production
- Spironolactone (100-200 mg daily) is a weak anti-androgen; may be used in combination with oral contraceptives; bromocryptine and cimetidine have no value
- Cyproterone acetate (50-100 mg/day) on days 1 to 15, and ethinyl estradiol (50 μg/day) is given on days 5 to 26 of menstrual cycle; cyproterone is a prototypic anti-androgen
- Eflornithine cream is novel treatment for removal of facial hair in women
- Flutamide (non-steroidal anti-androgen) may be used; finasteride (5 mg/day), a 5 α-reductase inhibitor has also been shown to be effective
- Low dose dexamethasone, prednisolone (to suppress ACTH), ketoconazole are also tried.

## MESSAGE

Most of the 'hairy women' have no underlying disease and only faces cosmetic problems.

—— o ——

CHAPTER 37

# Hoarsenes of Voice

## DEFINITION

It is the rough and harsh voice due to abnormality in vocal cords, and may range from slight huskiness to complete aphonia.

## AETIOLOGY

### *Congenital*

Laryngeal web, dermoid cyst, cystic hygroma, lipoma, fibroma or haemangioma of larynx.

### *Acquired*

a. Traumatic:
   - Overuse of voice–singer's nodule, hawker's voice
   - Anaesthetic intubation injury, strangulation or post-cough injury
   - Tobacco, irritrant gases

b. Inflammatory:
   - Specific–diphtheria, tuberculosis, leprosy, lupus, syphilis of the larynx
   - Non-specific–simple infective laryngitis, acute laryngo-tracheo-bronchitis, oedema of the larynx, chronic simple laryngitis, tracheitis, croup, epiglottitis

c. Neoplastic:
   Papilloma, squamous cell carcinoma, polyp, lipoma, angioma, fibroma, leiomyoma of the larynx

d. Paralytic (as a result of recurrent laryngeal or vagus nerve palsy):
   Bronchogenic carcinoma, cardiac surgery, post-thyroidectomy/parathyroidectomy, chest surgery, mediastinoscopy, pulmonary hypertension

e. Neurological:
   - Cerebrovascular accidents (CVA), parkinsonism, GB syndrome, disseminated sclerosis, amyotrophic lateral sclerosis
   - Hysteria (often with H/O recurrence under stress; coughs normally)

f. Systemic:
   - Myxoedema (very common medical cause in clinical practice)
   - Leprosy
   - Rheumatoid arthritis (crico-arytenoid joint affection)
   - Poliomyelitis (bulbar)
   - Angioneurotic oedema
   - Wegener's granulomatosis
   - SLE
   - Mitral stenosis (Ortner's syndrome)
   - Puberty/menopause
   - Lipoid proteinosis
   - Acromegaly
   - Sicca syndrome.

### OLLIVER'S SIGN (TRACHEAL TUG) + HOARSENESS

- Aneurysm of aorta

### BOVINE COUGH + STRIDOR + HOARSENESS

- Recurrent laryngeal nerve palsy

### HORNER'S SYNDROME + HOARSENESS

- Mediastinal mass

### MOON FACE + PLETHORIC FACE + HOARSENESS

- Superior mediastinal syndrome

* Right sided recurrent laryngeal nerve remains high up. Left sided recurrent laryngeal nerve is usually compressed by a mediastinal mass (e.g. hilar adenopathy from bronchogenic carcinoma, lymphoma etc.) → 'indirect laryngoscopy' shows the position of the left vocal cord in paramedian or cadaveric position during phonation → a method of quick diagnosis of mediastinal mass-induced hoarseness in clinical practice.

** In health, during phonation the vocal cords become adducted and during respiration the cords are abducted.

## COMMON CAUSES OF HOARSENESS IN CLINICAL PRACTICE

1. Chronic simple laryngitis (H/O recurrent acute laryngitis, inflamed vocal cord at laryngoscopy, benign in nature).
2. Carcinoma of the larynx (progressive hoarseness, smoker, confirmed by laryngoscopy with biopsy).
3. Paralytic (having H/O surgery or stigmata of SVC syndrome, confirmed by laryngoscopy with cadaveric position of vocal cord).
4. Myxoedema (gradual hoarseness over months, tiredness, obesity, constipation, menorrhagia, swollen vocal cords at laryngoscopy).
5. Singer's nodule (+ve occupational history like singer, orator, hawker or teacher as a result of voice abuse; vocal cord shows nodules at laryngoscopy).
6. Granuloma, i.e. tuberculosis, sarcoidosis, Wegener's granulomatosis (onset over months, confirmed by laryngoscopic biopsy).

## MECHANISM

Normal voice is produced as a result of vibration as well as movement of vocal cord. If the vocal cord is affected by oedema, inflammation, infiltrative diseases, tumour or compression over recurrent laryngeal nerve → hoarseness of voice results.

## CLUE TO DIAGNOSIS OF 'MEDICAL' CAUSES OF HOARSENESS

Look for:

Dull expressionless puffy face + madarosis + delayed relaxation of ankle jerk → Myxoedema
↓
Thickened ulnar nerve + leonine facies → Leprosy
↓
H/O sudden chocking sensation + presence of giant hives → Angioneurotic oedema
↓
Butterfly rash in face of a female, patchy alopecia, arthralgia + H/O Raynaud's phenomenon → SLE
↓
Swan-neck and button-hole deformity along with spindle-shaped fingers, and restricted + painful joint movement → Rheumatoid arthritis
↓

Destruction of nose and nasal septum, H/O Raynaud's phenomenon + features of vasculitis like digital infarction, digital ulceration, nail-fold thrombi, splinter haemorrhage → Wegener's granulomatosis

↓

Dry mouth and eyes over months + arthritis + deglutition difficulty → sicca syndrome

## MESSAGE

Persistent hoarseness of few weeks' or months' duration needs urgent intervention.

—— o ——

CHAPTER 38

# Hyperkeratosis of Palms

## CLUE TO DIAGNOSIS

1. Manual labourer.
2. Phrenoderma (vitamin A or essential fatty acid deficiency).
3. Arsenic poisoning (dry, grey, irregular, hyperkeratotic papules).
4. Psoriasis (usually bilaterally symmetrical).
5. Tylosis palmaris et plantaris (congenital hyperkeratosis, and pitting of palms and soles believed to be associated with carcinoma of the oesophagus).
6. Secondary to rash of secondary syphilis or keratoderma blenorrhagica (Reiter's syndrome).
7. Drug-induced, e.g. β-blockers.
8. Eczematous dermatitis (e.g. hand of housewives).
9. Ichthyosis (especially from Hodgkin's disease).

## HYPERKERATOSIS

It is the thickening of horny layer of skin usually resulting from retention and increased adhesion of epidermal cells.

## DERMATOLOGICAL CHANGES OFTEN ASSOCIATED WITH INTERNAL MALIGNANCY

- Dermatomyositis (in ovarian/breast/colon carcinoma, lymphoma)
- Acanthosis nigricans (in gastric carcinoma, lymphoma)
- Ichthyosis (adult-onset)
- Alopecia mucinosa (in mycosis fungoides)
- Pachydermoperiostosis (acquired; in bronchogenic carcinoma)

- Erythema gyratum repens (concentric, arcuate lesions look like the grain of a soft wood)
- Intractable pruritus without any skin lesion
- Hypertrichosis lanugosa (acquired in malignancy of breast, lung, colon)
- Migrating thrombophlebitis (related to carcinoma of the pancreas)
- Multiple irritable seborrhoeic warts (sign of Leser-Trelat).

## POMPHOLYX

- An endogenous eczema
- Bilaterally symmetrical, recurrent vesicular eruptions in palms (cheiropompholyx) and soles
- Sago-grain like, excruciatingly itchy; may be painful
- Affects young adults; in summer; multifactorial; provoked by heat or stress
- Hyperhydrosis
- Spontaneous resolution with scaling
- Treatment by topical corticosteroids.

## PALMAR XANTHOMA

- Yellowish discolourations of the palmar digital creases
- Seen in familial dysbetalipoproteinaemia (accumulation of remnant-like particles in plasma)
- Associated with fulminant atherosclerosis and premature coronary artery disease
- Often associated with obesity, diabetes mellitus or hypothyroidism
- Histology includes xanthoma cells (i.e. lipid-laden foam cells), Tuton giant cells with admixture of inflammatory cells.

## PALMAR ERYTHEMA (BRIGHT RED PALM)

The thenar and hypothenar eminences, base and pulp of the fingers turn red in:

- Cirrhosis of liver ('liver palms')
- Alcoholics
- Pregnancy
- Rheumatoid arthritis (long-continued)
- Thyrotoxicosis
- Hyperdynamic circulation, e.g. pyrexia, pregnancy

- Polycythaemia
- Rarely in normal persons (familial).

## BLACK PALMAR CREASES

Addison's disease.

## CHEIROARTHROPATHY

It is the limited joint mobility of hands in diabetes mellitus. The patient cannot extend finger/fingers at metacarpophalangeal or interphalangeal joints → resulting in painless stiffness in hands → the classical 'prayer sign'.

## SWELLING/NODULES IN HAND

- Osler's node (tender papule in pulp → subacute bacterial endocarditis)
- Heberden's node (bony nodules at dorsal aspect of DIP joint → osteoarthritis)
- Bouchard's node (bony nodules at dorsal aspect of PIP joint → osteoarthritis)
- Gouty lophi (over dorsal aspect of PIP and DIP joints)
- Rheumatoid nodule or nodule formation in leprosy
- Calcinosis (in scleroderma)
- Ganglion, neurofibroma, lipoma (of surgical interest).

## THREE 'P' OF UNDERLYING MALIGNANCY

- Pallor
- Pigmentation
- Pruritus.

—— o ——

CHAPTER 39

# Hypertelorism

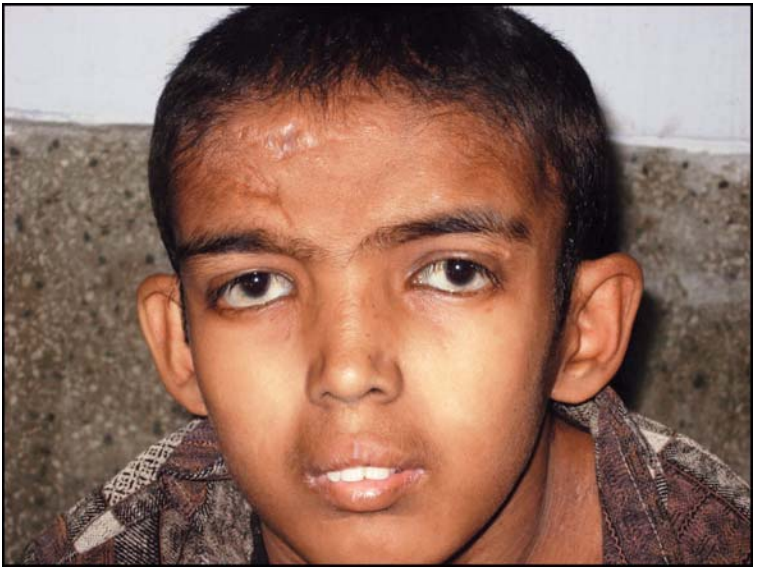


**FIGURE 39.1:** Hypertelorism (widely set eyes) in Ehlers-Danlos syndrome with mild blue sclera

## DEFINITION

This is synonymous with widely set (spaced) eyes. When the distance between inner canthus of two eyes is greater than half of the interpupillary distance, hypertolorism is said to be present. Quantitatively, it is defined as the ratio between the inner canthal distance over the outer canthal distance and if it is > 0.38, it is known as hypertelorism.

Hence, the root of the nose appears broad and the distance between the eyes seems to be increased.

## POSSIBLE CAUSES

1. Mild hypertelorism may be seen in normal children
2. Racial
3. Down's syndrome
4. Thalassaemia major
5. Cretinism
6. Craniostenosis, e.g. Crouzon syndrome
7. Turner's syndrome
8. Elfin facies seen in congenital supravalvular aortic stenosis
9. Associated with congenital pulmonary stenosis
10. Associated with mental retardation
11. Carpenter syndrome
12. LEOPARD syndrome

* Probable pathology–due to hyperplasia of lesser wing of sphenoid bone.

## HYPOTELORISM

Eyes being located too close to each other with decreased interpupillary distance.

## WHY HYPOTELORISM?

When the brain has not divided into two hemispheres and there exists a single ventricle (holoprosencephaly), hypotelorism is said to exist. Common examples are trisomy 13 (Patau syndrome), ethmocephalus, cebocephalia; may be familial.

## LOW SET EARS

In health, if an arbitrary horizontal line is drawn from the outer canthus of the eye to the ipsilateral pinna, about 1/3rd of the pinna is seen above the line. In low set ears, < 1/3rd of total length of pinna lies above the line. It is seen in disorders like:

- Down's syndrome
- Trisomy 18 (Edward's syndrome.
- Trisomy 13 (Patau syndrome)
- Mental retardation
- Elfin facies.

## DIAGONAL CREASE IN EAR LOBULE

Very often a prominent horizontal crease is seen over the lobule of the ear → designated as a marker for ischaemic heart disease (IHD).

—— o ——

CHAPTER 40

# Indigestion (Dyspepsia)

## DEFINITION

A vague and non-specific term → meaning changes from patient to patient. It may mean abdominal fullness, gaseous distension, nausea, heartburn, epigastric discomfort, belching, bloating, heartburn, flatulence, port-prandial fullness or loose motion.

## POSSIBLE ASSOCIATIONS

1. Gastroesophageal reflux disease.
2. Gastritis.
3. Functional dyspepsia (anxiety/stress).
   - Reflux-like → acid reflux+heartburn
   - Ulcer-like → epigastric pain+nocturnal pain → relieved by vomiting/food/antacid
   - Dysmotility-like → nausea, eructation, bloating and early satiety
4. Drugs–NSAIDs, corticosteroid, nitroimidazoles (e.g. metronidazole).
5. Lactose intolerance.
6. Cholecystitis.
7. Chronic pancreatic insufficiency.
8. Peptic ulcer disease or non-ulcer dyspepsia.
9. Dietary–consumption of fat, spicy food, cabbage, radish, onion, legumes.
10. Miscellaneous–aerophagia, magenblase, gas entrapment syndrome, gastroparesis (e.g. autonomic neuropathy from diabetes), constipation, malignancy or lymphoma of stomach, irritable bowel syndrome, psychological disorders, chronic renal failure, congestive cardiac failure, cirrhosis of liver, mouth breathing, betel nut chewing.

## AEROPHAGIA (AIR SWALLOWING)

Approximately 20-60% of intraluminal gas (seen in fluoroscopy) is swallowed air. Aerophagia can also be called as eructation or belching. It may happen to occur with/without the patient's awareness → some people attempts to do it in a false hope to relieve abdominal pain/distension/discomfort. It is seen under certain circumstances, eg.

- Chronic anxiety
- Poor eating habit (e.g. rapid ingestion of food)
- Consumption of food in supine position
- Gum chewing, ill-filled dentures
- Comsumption of cabbage, onion, peppers or carbonated beverages
- Cholecystitis, hiatal hernia, non-ulcer dyspepsia
- Any organic intestinal disease (aerophagia may increase in magnitude day by day).

## NON-ULCER DYSPEPSIA

- Symptoms suggestive of dyspepsia and peptic ulcer but no evidence of ulcer on endoscopy
- Probably a combination of mucosal + motility + psychiatric disorders
- Young (< 40 years); F:M = 2:1
- C/O nausea, bloating, incomplete rectal evacuation, irritable bowel syndrome-like symptoms
- Many have *H. pylori* infection and often associated with prolonged gastric emptying time
- Endoscopy is necessary to exclude mucosal disease. USG should be performed to detect gallstones, if any
- Management is done by reassurance, prokinetics and anti-*H. pylori* regimen.

## MAGENBLASE

Synonym → 'gastric bubble syndrome'. It is the acute gastric distension due to aerophagia, resulting in sharp precordial pain mimicking angina pectoris. It is a perplexing situation in older patients with coronary artery disease, who may experience true postprandial angina from time to time.

## SPLENIC FLEXURE SYNDROME (GAS ENTRAPMENT SYNDROME)

Swallowed air may be trapped in the splenic flexure of colon, if not eructed out orally. The person may complain of left upper quadrant fullness and

pressure with radiation to the left side of the chest → relief may be obtained with defaecation or expulsion of flatus. It can be diagnosed by ↑ tympanicity on percussion over the extreme left lateral portion of the upper abdomen.

## FLATULENCE (WIND)

Flatulence or excess gas in the abdomen is one of the most common complaint encountered in clinical practice. The patient may present in three ways like (i) excessive belching, (ii) intestinal distension, bloating or meteorism, and (iii) the passage of excessive flatus.

The meaning of flatulence varies from patient to patient like repeated belching → abdominal fullness → offensive rectal flatus → borborygmi (i.e. audible intestinal peristaltic sounds). Intestinal gas (have three sources) may be derived from swallowed air + colonic bacterial fermentation of poorly absorbed carbohydrates + a very small quantity from diffusion from the blood into the gut lumen; the normal volume of flatus varies from person to person, i.e. 200-2000 ml/day. The composition of intestinal gas is basically by $N_2$, $O_2$, $H_2$ and $CO_2$ (99%) though a small quantity of methane may be present. Increased flatus formation may be due to lactase deficiency, malabsorption or small bowel dysmotility. Obstipation or absolute constipation (absence of stool+flatus per rectally) is seen in acute intestinal obstruction. Excessive wind formation may result in social embarrassment.

## ABDOMINAL DISTENSION

A. Acute or sudden onset: Intestinal perforation, intestinal obstruction, paralytic ileus, post-pneumoperitoneum (e.g. after peritoneoscopy).
B. Gradual: Ascites (cirrhosis, CCF, hypoproteinaemia with anaemia, nephrotic syndrome, constrictive pericarditis, pericardial effusion, tuberculous or malignant peritonitis), cystic swelling (pseudopancreatic, hepatic, splenic, mesenteric, ovarian), pregnancy or any visceromegaly.

N. B: Never forget 7 F's, i.e. fat (obesity), faeces (gut obstruction), fetus (pregnancy), flatus (gaseous distension), fluid (ascites or any cystic swelling), full bladder (urinary) and fibroid (or any huge mass) as causes of abdominal distension.

## FEW TERMINOLOGY

Nausea → A subjective feeling of vomiting.

Vomiting → Expulsion of upper GI contents orally.

Regurgitation → Effortless passage of gastric contents into the mouth.

Water brash → Salivary hypersecretion, i.e. filling of the mouth suddenly with a clear but slightly salty fluid, secreted by the salivary glands. It may be a transient episode in normal individuals.

Heartburn (pyrosis) → Substernal sensation of warmth or burning and usually due to reflux of gastroesophageal contents.

Rumination → Repeated regurgitation of contents of stomach which are often rechewed and reswallowed.

Eructation (belching) → Forceful regurgitation of air from the stomach or oesophagus.

—— o ——

CHAPTER 41

# Intermittent Claudication

## DEFINITION

It is a cramp-like pain associated with tightness, numbness and extreme fatigue in muscles, and occurs most commonly in calf muscles on walking. The pain is relieved by rest and reappears when the person starts walking. The pain during walking may be so intense that the patient is bound to halt immediately.

The pain is due to muscle ischaemia which is felt on walking. The actual distance a patient can walk before the onset of intermittent claudication is known as 'claudication distance', which is a good index of severity of arterial occlusion. Later, the pain becomes constant and aching in nature, and persists even on rest, i.e. 'rest pain', which is due to ischaemic changes in the somatic nerves (so called, cry of the dying nerves).

## POSSIBLE CAUSES

A. With vascular insufficiency (i.e. narrowed arteries):
   1. Atheroma or embolism of lower limb arteries.
   2. Buerger's disease.
   3. Arteritis (e.g. syphilitic, aorto-arteritis).
   4. Coarctation of aorta
   5. Leriche's syndrome (embolism at branching of common iliac arteries, i.e. claudication of thigh, and impotence).
   6. Aortoiliac occlusion.
   7. Diabetes mellitus.

B. Without vascular insufficiency (i.e. normal arteries):
   1. Over-exertion (e.g. marathon runner).

2. Severe anaemia.
3. McArdle's disease (muscle phosphorylase ↓).
4. Lumbar canal stenosis (i.e. neurogenic claudication).

## NEUROGENIC CLAUDICATION

It is also known as claudication of cauda equina or lumbar canal stenosis. This entity is the end result of combination of disc lesion and a congenital narrowing of lumbar theca. The disease is made worse at middle age due to degenerative changes, especially between $L_4$ and $L_5$ vertebra. Walking or prolonged standing interferes with the blood supply to the cauda equina which leads to root pain, weakness of legs, paraesthesia and even foot drop (pulse remains normal). Ankle jerk may be diminished or absent; rest pain never occurs in contradiction to vascular claudication. Patient usually relieves by rest or stooping forward position.

## ON EXAMINATION

- Colour–Pale to pink on leg raising at 45°. Trophic changes+
- Palpation–determination of arterial occlusion by careful palpation of pulses; cold extremities (lower). Calf muscle may be tender on palpation
- Auscultation–for bruit in distal aorta, femoral or iliac arteries; auscultate heart for source of embolism
- Examination of motor function–At rest, motor functions (nutrition, tone, power, coordination) are normal. After exercise, pulses (arteria dorsalis pedis) may be diminished or absent, which is an important diagnostic clue in obstructive arterial disease
  Ankle jerk may be diminished in neurogenic claudication; pre-gangrenous conditions may develop (i.e. with vascular decompromise) after exercise.
- Ophthalmocopy–for search of atherosclerotic retinal vessels, haemorrhage etc.

## INVESTIGATIONS

1. Peripheral colour Doppler studies of both legs.
2. Arteriography.
3. Impedence plethysmography.
4. USG/CT scan of abdomen to detect cause of vascular occlusion/ aneurysm etc.

5. Biopsy of artery (to diagnose Buerger's disease, arteritis).
6. X-ray of lumbar spine, MRI scan of lower spinal cord to diagnose lumbar canal stenosis.

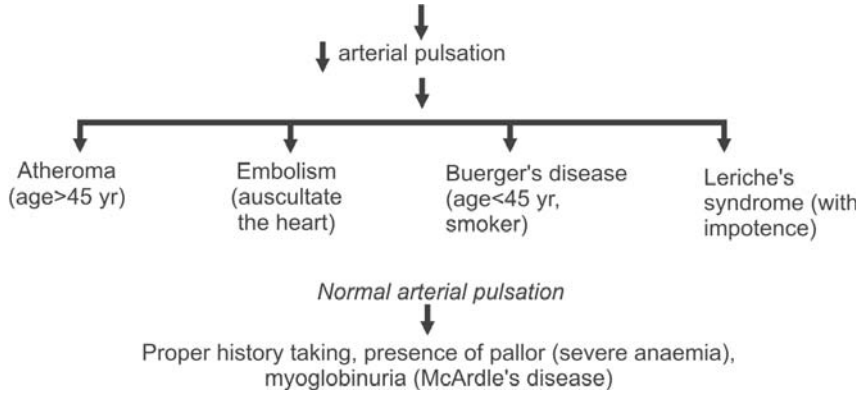
* At the bedside, palpation of peripheral pulses and measurement of lower extremity BP after exercise often clinch the diagnosis.

## TREATMENT

1. Abstinence form smoking or consumption of tobacco in any form.
2. ℞ of diabetes, hypertension and hyperlipidaemia, if present.
3. Vasodilators (e.g. nifedipine 5 mg OD/BDS) or haemorrheology modifier (e.g. pentoxifylline 400 mg BDS/TDS) may be used.
4. Analgesics or vitamin E (400 mg BDS) may be helpful.
5. ℞ of specific diseases (e.g. amputation for Buerger's disease, transluminal balloon angioplasty for severe atherosclerosis).
6. Amitriptyline 25-50 mg OD/HS may be used in rest pain/nocturnal pain.

## MESSAGE

Palpate peripheral leg arteries (popliteal, posterior tibial or arteria dorsalis pedis)




## ACROPARAESTHESIA

Feeling of tingling and numbness (described by the patient as 'pins and needles') or often burning sensation in the tip of the fingers and toes. The common aetiologies considered in clinical practice are:

- Cervical (in fingers) or lumbar spondylosis (in toes)

- Cervical rib
- Lesion in brachial plexus
- Carpar tunnel syndrome
- Peripheral neuropathy (leprosy, alcohol, diabetes mellitus)
- Physiological: Prolonged sitting over the front rod of a bycycle (foot and toes), arms compressed by body due to malposition while sleeping (hand and fingers)
- Functional: Often complained by middle-aged female patients as a manifestation of weakness.

## LEG PAIN: ↑ ON STANDING AND ↓ BY LYING

The two common diseases in clinical practice are:

- Varicose veins (or peripheral venous disease)
- Prolapsed intervertebral disc.

## MESSAGE

Vascular claudication: cold leg pallor, colour change, trophic changes, feeble/absent pulse

Neurogenic claudication: paraesthesia, limb weakness, ↓ ankle jerk, normal pulse

* A patient of arterial disease in legs (e.g. severe atheroembolism) sleeps with legs hanging down, i.e. over edge of the bed or in a chair.

—— o ——

CHAPTER 42

# Joint Pain

**FIGURE 42.1:** Swelling of wrist and metacarpophalangeal joints, radial deviation of wrist with ulnar deviation of digits in rheumatoid arthritis

## MONOARTHRITIS

- Septic arthritis (*S. aureus, N. gonorrhoea,* meningococci, *S. pneumoniae,* gram-negative infections)–extremely tender
- Crystal-induced arthritis (gout, pseudogout, calcium oxalate or hydroxyapatite crystals)
- Traumatic joint injury
- Osteoarthritis
- Tuberculosis of the joint (e.g. knee)
- Haemarthrosis (traumatic)

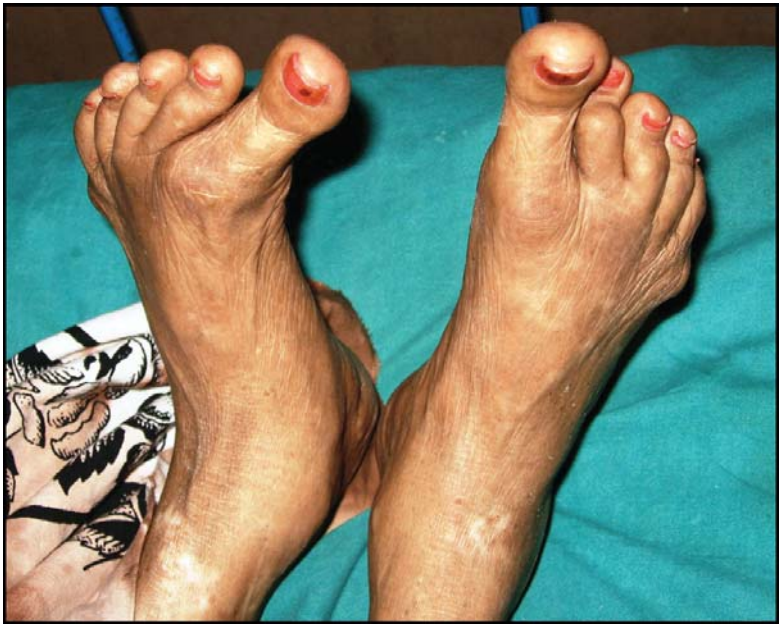


**FIGURE 42.2:** Halux varus deformity, widening of the forefeet with cock-up great toes in rheumatoid arthritis

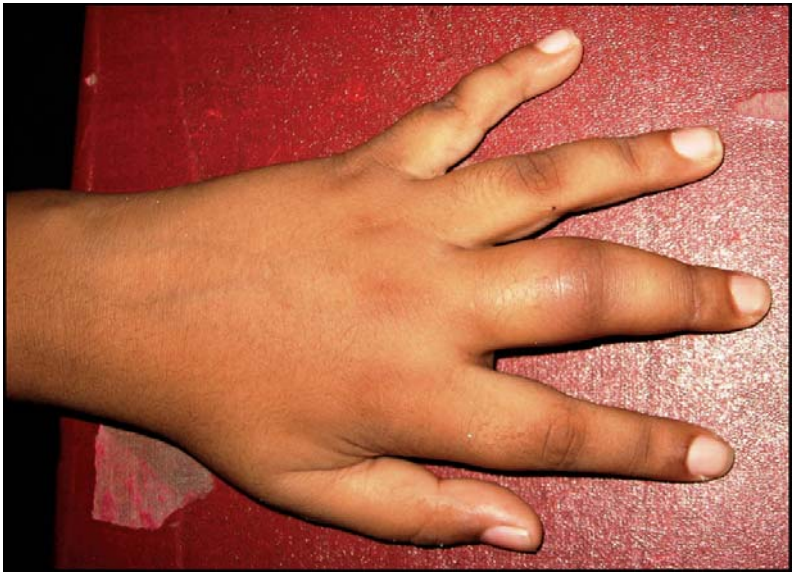


**FIGURE 42.3:** Fusiform swelling of entire left middle finger (dactylitis or sausage digit) – often a clinical clue in reactive arthritis

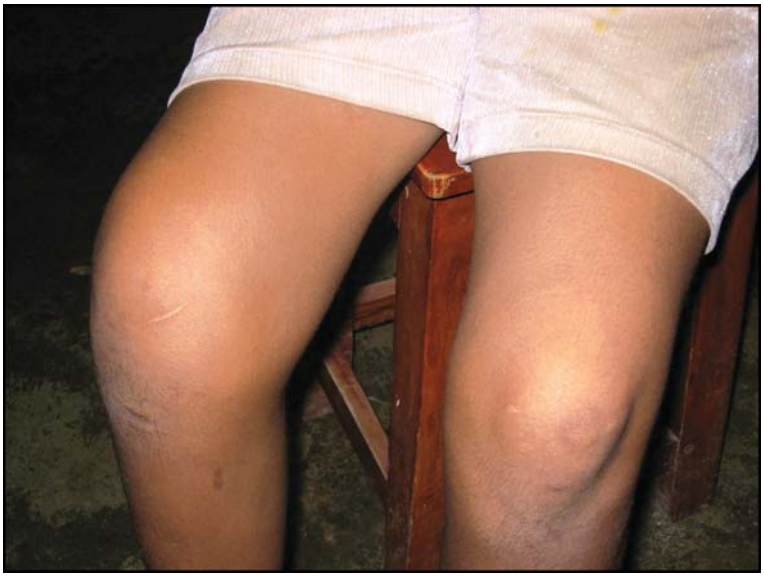


**FIGURE 42.4:** Painful and swollen knee (monoarthritis) in a patient of haemophilia

- Monoarticular flare of polyarticular rheumatic diseases (e.g. rheumatoid arthritis, SLE, psoriasis, reactive arthritis)
- Charcot joint (neuropathic joint from diabetes, leprosy, syringomyelia or tabes dorsalis)
- Villonodular synovitis
- Haemophiliac joint (e.g. knee), acute leukaemias, Henoch-Schönlein purpura

  Note: As any delay in the treatment of septic arthritis would lead to joint destruction, it is prudent to start antibiotic therapy empirically before laboratory reports give a definitive diagnosis. Urgent synovial fluid examination is mandatory in acute monoarthritis for:
- Crystals (under polarised light microscopy)
- Pathogens (Gram staining and microbial culture)
- WBC (> 2000/$mm^3$ is diagnostic of inflammatory joint disease)

* Charcot's joint and villonodular sinovitis give rise to chronic monoarthritis.

## POLYARTHRITIS

- Rheumatoid arthritis (RA), juvenile idiopathic arthritis (JIA)
- SLE and other connective tissue diseases

- Psoriatic arthritis
- Ankylosing spondylitis
- Palindromic rheumatism
- Rheumatic fever, SBE, Lyme arthritis
- Reactive arthritis
- Crystal-induced arthritis (e.g. gout)
- Drug hypersensitivity (e.g. surum sickness)
- HIV, hepatitis B, Parvovirus B19, chikungunya or rubella infection
- Miscellaneous–generalised osteoarthritis, lymphoma, leukaemia, sarcoidosis, Behcet's disease, spondyloarthropathies, Whipple's disease, Henoch-Schonlein purpura, neuropathic joint, HPOA (hypertrophic pulmonary osteoarthropathy), relapsing polychondritis, malignancy, post-streptococcal reactive arthritis, amyloidosis.

## FEW TERMINOLOGY

- Arthralgia–only pain in the joints
- Arthritis–pain + swelling in the joints
- Monoarthritis–affection of single joint
- Oligo– or pauciarticular arthritis–affection of 2-4 joints
- Polyarthritis–affection of 5 or more joints

*The anatomical basis of pain in musculoskeletal system could be:*
Joint:

- Synovium–synovitis
- Joint capsule–capsulitis

Periarticular soft tissue:

- Bursa–bursitis
- Tendon sheath–tenosynovitis
- Tendon–tendonitis
- Insertion of tendon, ligament–enthesitis

Bone

## DURATION OF JOINT PAIN

- Acute (< 6 weeks)
- Chronic (> 6 weeks).

## PATTERN OF INVOLVEMENT

- Axial (spine, sacroiliac, anterior chest wall, shoulder and hip joint)

- Appendicular (peripheral joints)

* Shoulder and hip joints are known as root joints.

## CLINICAL PARAMETERS OF 'INFLAMMATORY' JOINT DISEASES

- Significant early morning stiffness (usually > 30 minutes)
- Symptomatic improvement on gentle use of joint
- Spontaneously up-and-down course (i.e. 'spontaneous flare')
- Constitutional symptoms (e.g. fatigue, ↓ appetite, ↓ body weight, low-grade fever, night sweats).

## COMMON LABORATORY INVESTIGATIONS PERFORMED IN JOINT DISEASES

1. Acute phase reactants (confirm inflammatory nature of the disease).
   - ESR
   - Platelet count
   - Albumin-globulin ratio
   - C-reactive protein (CRP)
   - Alkaline phosphatase.
2. Rheumatoid factor (RF).
3. Anti-nuclear antibody (ANA) and its subsets, e.g. anti-ds DNA, anti-RNP etc.
4. Complement $C_3$ and $C_4$.
5. Antibodies to cyclic citrullinated peptide (anti-CCP) to diagnose early rheumatoid arthritis.
6. Anti-streptolysin 'O' antibody (ASO) titre.
7. Anti-neutrophil cytoplasmic antibody (ANCA, i.e. c- or p-ANCA).
8. Synovial fluid analysis.
9. Serum uric acid level.
10. Others: HLA-B 27 screening, synovial biopsy.

## PATIENTS COMPLAINING OF STIFF/PAINFUL MUSCLES

1. Strenuous exercise (H/O unaccustomed exercise 24-48 hr before).
2. Ankylosing spondylitis (young, low backache, progressive loss of spinal movement).
3. Polymyositis/dermatomyositis (proximal muscle weakness+).
4. Polymyalgia rheumatica (elderly, fatigue, painful proximal muscle).
5. Fibromyalgia (females with specific tender points all over the body, anxiety+, depression+).

6. Rheumatoid arthritis (middle aged female, morning stiffness in active disease, MCP and PIP joints involved in hands).
7. Myxoedema (middle aged female, obese, hoarse voice, cold intolerance, muscle weakness+).

## DRUGS PRODUCING ARTHRALGIA/ARTHRITIS

- Sulphonamides
- Penicillin
- Hydralazine
- Procainamide
- Phenytoin
- Iodides.

## ARTHRITIS ASSOCIATED WITH

A. Murmurs in the heart—
   - Acute rheumatic fever
   - Ankylosing spondylitis
   - SBE
   - Rheumatoid arthritis
   - SLE (Libman-Sacks endocarditis)
   - Atrial myxoma
   - Relapsing polychondritis

B. Subcutaneous nodules—
   - Rheumatoid arthritis
   - Gout
   - Acute rheumatic fever
   - Sarcoidosis
   - Amyloidosis
   - Reticulohistiocytosis (multicentric)
   - Whipple's disease

C. Rash—
   - Vasculitis
   - SLE
   - Dermatomyositis
   - Psoriasis
   - Chronic urticaria
   - Sarcoidosis
   - Leprosy

D. Enthesopathy –
- Ankylosing spondylitis
- Psoriatic arthritis
- Reactive arthritis
- Viraemia or bacteraemia
- Drugs (e.g. ciprofloxacin)
- Disseminated idiopathic sketelal hyperostosis (DISH).

## COMMON CAUSES OF POLYARTHRITIS IN HANDS

1. Rheumatoid arthritis (MCP, PIP).
2. Nodal osteoarthritis (DIP but spares MCP).
3. Psoriatic arthritis (commonly DIP).
4. Chronic tophaceous gout (MCP, IPs).
5. SLE (Jaccoud's arthritis; MCP joints commonly).
6. Viral arthritides (all joints).

## D/D OF ACUTE MONOARTHRITIS PRESENTING AS 'RED HOT JOINT'

A. Infections (septic arthritis): bacterial (non-gonococcal/gonococcal), viral.
B. Crystal-induced: gout, pseudogout.
C. Acute exacerbation of rheumatoid arthritis, reactive arthritis, psoriatic arthritis and palindromic rheumatism (monoarticular RA lasting 24-48 hours).
D. Haemophilia.
E. Traumatic.

## DISEASE COURSE OF POLYARTHRITIS

A.
- → Intermittent: gout, palindromic RA
- → Progressive: classical RA

B.
- → Migratory (as the inflammation of one joint is subsided, other tend to become affected, i.e. usually one joint is affected at a time for about 3 days): rheumatic arthritis, SLE, drug reaction/serum sickness, arthritis following gonoccocal or meningococcal septicaemia, viral arthritis (Lyme arthritis, chikungunya), following inflammatory bowel disease/Whipple's disease, 'seroconversion' in AIDS, septicaemia, sarcoidosis, following intestinal by-pass surgery
- → Additive: RA, ankylosing spondylitis, reactive arthritis.

## AGE AND SEX RELATED ARTHRITIS

A. Age:

- Children : Rheumatic arthritis, JIA, haemophilia, trauma
- Adolescents : Rheumatic arthritis, spondyloarthropathy, trauma, JIA, post-streptococcal reactive arthritis
- Young : Trauma, gonococcal
- Adults : Spondyloarthropathy, reactive arthritis, psoriasis, SLE, gout
- Middle age : RA, gout, osteoarthritis, scleroderma

B. Sex:

Arthritis predominant in males are:

- Gout
- Ankylosing spondylitis
- Reiter's syndrome (i.e. reactive arthritis)
- Polyarteritis nodosa

* Other arthritides are dominant in females.

## ARTHRITIS AFFECTING DISTAL INTERPHALANGEAL (DIP) JOINTS

- Osteoarthritis
- Psoriatic arthritis
- Scleroderma
- Sarcoidosis
- Gout
- Septic arthritis.

## JACCOUD'S ARTHRITIS

Ulnar deviation of MCP joints due to subluxation may develop from,

- Rheumatic arthritis
- SLE
- Sjögren's syndrome.

—— o ——

CHAPTER 43

# Leg Ulceration

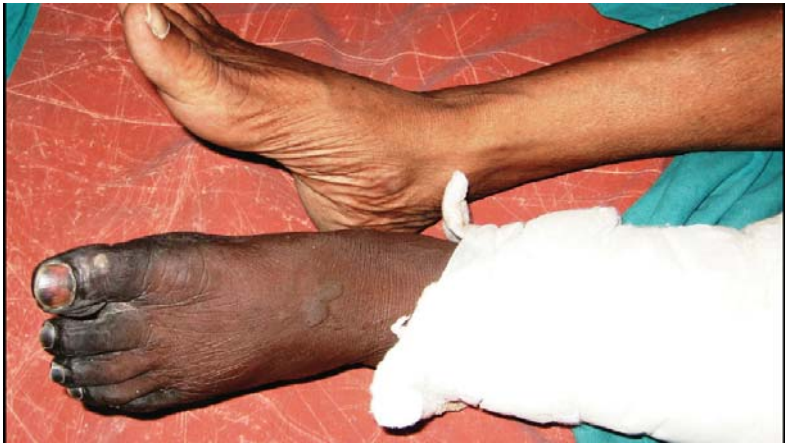


**FIGURE 43.1:** Diabetic foot with dry gangrene

## AETIOLOGY

1. Venous diseases: Varicose ulcer, DVT, deep venous obstruction from pelvic growth, incompetent valves.
2. Arterial insufficiency: Atherosclerosis, Buerger's disease, vasculitis.
3. Small vessel diseases: Diabetes mellitus, vasculitis.
4. Neuropathy: Diabetes mellitus, leprosy, tabes dorsalis, syringomyelia.
5. Haemorrhagelogical: Sickle cell disease, hereditary spherocytosis, thalassaemia major, cryoglobulinaemia, immune complex diseases, cold agglutinin disease, macroglobulinaemia.
6. Tumour: Squamous cell carcinoma, Kaposi's sarcoma, malignant melanoma, basal cell carcinoma, mycosis fungoides, metastasis

7. Traumatic ulcer: Burns, cold injury, factitial
8. Tropical ulcer (a chronic form of callous ulcer with its edge raised and undermined, and very often refuses to heal).
9. Trophic ulcer (affecting sole of foot, especially over the heel or ball of the great toe → diabetes mellitus, leprosy, syringomyelia and tabes dorsalis) → painless ulcer.
10. Chronic atopic eczema
11. Miscellaneous–Pyoderma gangrenosum (ulcerative colitis, rheumatoid arthritis, immunodeficiency), gout, necrobiosis lipoidica diabeticorum (diabetes), tuberculous ulcer, actinomycosis, panniculitis, malingering, Bazin's disease, bullous pemphigoid, filariasis.

## CLUE TO DIAGNOSIS

1. Surface temperature
   Normal → Venous disease or neuropathy, and others
   Cold → Arterial insufficiency
2. Site of ulcer:
   Venous → lower leg ankle
   Arterial → shin, foot
   Vasculitis → shin, upper leg (painful)
   Neuropathy → heel, ball of the great toe (painless)
3. Peripheral arterial pulsation, e.g. feel the pulsation of arteria dorsalis pedis/posterior tibial artery
   ↓
   ↓ pulsation in arterial insufficiency, vasculitis
4. Homan's sign and Moses' sign present: DVT
5. Ankle jerk: ↓ in neuropathy
6. Vibration sense: ↓ in neuropathy with special reference to diabetes mellitus
7. Oedema: Commonly in venous diseases.
8. Thickened peripheral nerves: Present in leprosy (with peripheral neuropathy).
9. Anaesthetic patches (e.g. leprosy).
10. Trendelenburg test: Positive in varicose veins.
11. Blood pressure: Hypertensive in atherosclerosis, diabetes mellitus.
12. Local cyanotic hue: Especially in arterial insufficiency.

## BASIC INVESTIGATIONS PERFORMED

- Blood: For peripheral smear examination, anaemia, blood dyscrasias; VDRL, sugar and cholesterol
- Urine: For sugar
- Bacterial swab: For detection of pathogens
- Doppler ultrasound: For documentation of arterial insufficiency
- Venography: For venous diseases
- Nerve conduction study: For peripheral neuropathy.

## BAZIN'S DISEASE

- Erythema induratum
- Uncommon, bilateral, painful and tender duskey-red nodules usually over calves
- Females > males
- Recurrent; irregular edges; heals with scar
- May need prolonged anti-tuberculosis treatment.

—— o ——

CHAPTER 44

# Lock Jaw

## SYNONYM

Trismus (inability to open the mouth).

## MECHANISM

Develops as a result of sustained spasm of masseter muscle leading to closure of the jaws so that the mouth cannot be opened.

## POSSIBILITIES

1. Tetanus (a cause par excellence of trismus with positive spatula test).
2. Strychnine toxicity (often a late manifestation).
3. Tetany.
4. Drug-induced dyskinesia (e.g. metoclopramide, phenothiazines).
5. Temporo-mandibular joint osteoarthritis or ankylosis.
6. Impacted wisdom teeth.
7. Peritonsillar abscess (quinsy), dental abscess, Ludwig's angina, dislocation of jaw, cyanide poisoning.
8. Acute follicular tonsillitis.
9. Parotitis, mumps.
10. Hydrophidae group of snake bite.
11. Hysterical or malingering (during sleep, muscles relaxes completely).
12. Stiff-man syndrome (progressive fluctuating muscular rigidity).
13. Rabies (rare)

* In temporo-mandibular joint dislocation, the patient cannot close the 'opened mouth'.

## SPATULA TEST

In health, if the posterior pharyngeal wall is touched by a spatula, it produces reflex opening of mouth. In tetanus, the mouth closes paradoxically in such a way that the spatula cannot be taken out easily. Spatula test is positive in tetanus and negative in others.

## RISUS SARDONICUS

When more and more muscles are involved in tetanus, rigidity becomes generalised, and sustained contractions of facial muscles give rise to a characteristic expression → a fixed sardonic smile, i.e. smile of 'Satan' or devil (where the smile does not reach the eyes) → risus sardonicus.

* In tetanus, trismus is an early sign (convulsions lately)
In strychnine poisoning, trismus is a late sign (convulsions early)

** In scleroderma and submucosal fibrosis of oral cavity, the patient may find difficulty in opening the mouth; but this is not true trismus (pseudotrismus).

## MANAGEMENT

- Reassurance
- Treatment of the aetiology
- Maintenance of nutrition by feeding through side of the mouth/Ryle's tube/IV fluid
- In severe cases, air entry may be maintained by tracheostomy
- Analgesics and anti-inflammatory drugs with muscle relaxants
- Role of injectable corticosteroids at the site/surgery should be considered.

—— o ——

CHAPTER 45

# Lump in Right Iliac Fossa

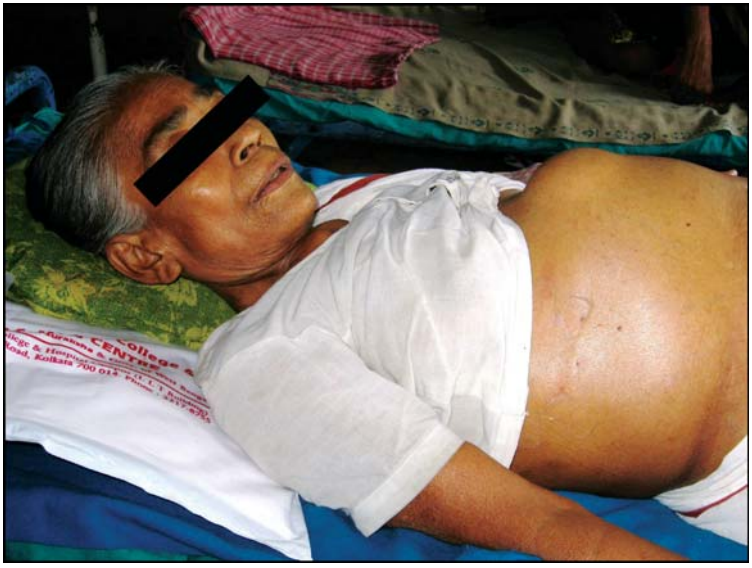


**FIGURE 45.1:** Visible lump in epigastrium: a case of hepato-cellular carcinoma (hepatoma) – imprints of leucoplast straps after liver biopsy is seen here

## ASSOCIATIONS

1. Ileocaecal tuberculosis
2. Amoebic typhlitis (inflammation of caecum)
3. Appendicular lump
4. Carcinoma of caecum or ascending colon
5. Tubo-ovarian mass
6. Crohn's disease (granuloma)
7. Intussusception
8. Impaction of round worms

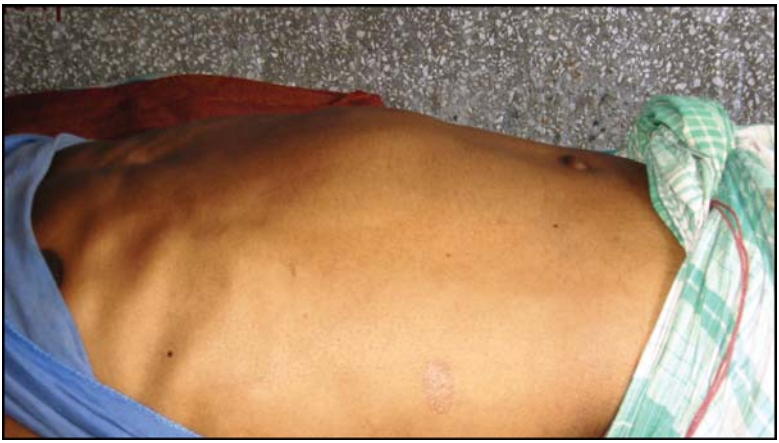


**FIGURE 45.2:** Visible fullness in epigastrium and left hypochondrium: splenic mass in a patient of chronic myeloid leukaemia

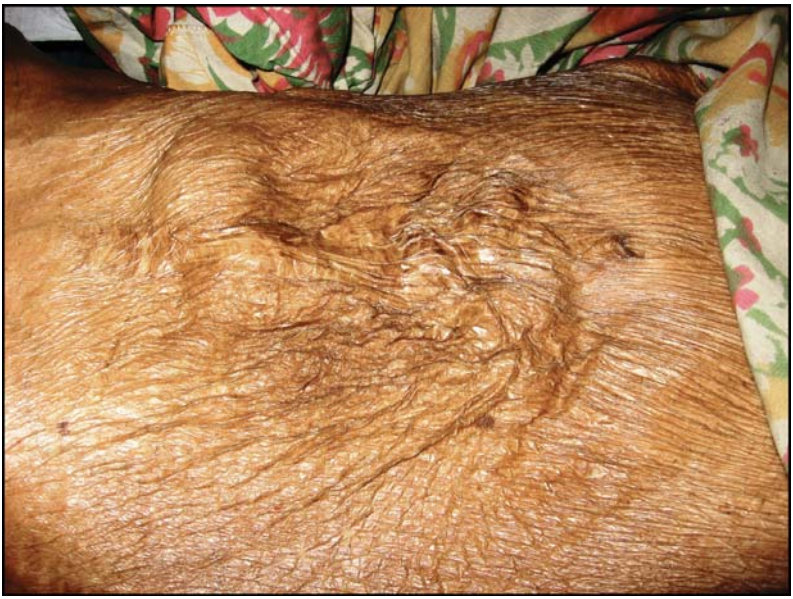


**FIGURE 45.3:** Intestinal coils with visible peristalsis (seen through a post-operative scar) present in a woman of 76 years

9. Dropped or unascended right kidney
10. Lymphoma
11. Carcinoid syndrome
12. Iliac aneurysm, psoas abscess
13. Malignant undescended testicle
14. Transplanted kidney.

## RIGHT LOWER QUADRANT ABDOMINAL PAIN

1. Acute appendicitis
2. Crohn's disease
3. Meckel's diverticulitis
4. Incarcerated hernia
5. Ectopic pregnancy
6. Salpingitis
7. Tubo-ovarian abscess
8. Endometriosis
9. Torsion of ovarian cyst
10. Perforated ulcer of caecum
11. Intestinal obstruction
12. Renal or ureteral calculi
13. Psoas abscess/haematoma
14. Leaking aortic aneurysm
15. Mittelschmerz
16. Caecal diverticulitis
17. Trauma

* So, a proper history taking may solve the mystery of Pandora's box.

## 'PERIUMBILICAL' ABDOMINAL PAIN

1. Small bowel obstruction
2. Mesenteric thrombosis (abdominal angina)
3. Intestinal amoebiasis
4. Roundworm infestations
5. Dissecting aneurysm of aorta
6. Acute pancreatitis
7. Early phase of acute appendicitis
8. Miscellaneous: diabetic ketoacidosis, uraemia, trauma.

## POSSIBLE CAUSES OF 'ACUTE SCROTUM'

1. Torsion of testis
2. Epididymitis (filarial, tuberculous)
3. Testicular malignancy
4. Orchitis (e.g. mumps).

## DISCHARGING 'SINUSES' OVER THE ABDOMEN

1. Faecal fistula
2. Tuberculosis of the intestine

3. Intra-abdominal malignancy
4. Crohn's disease
5. Actinomycosis.

## 'LYMPH NODE LUMPS' IN THE ABDOMEN

1. Mesenteric lymph node tuberculosis
2. Lymphoma (commonly pre- and paraaortic nodes)
3. Metastasis from neighbouring carcinoma
4. Metastasis from carcinoma of testis
5. Filariasis (retroperitoneal lymphadenitis; rare).

## PAIN ABDOMEN REFERRED FROM EXTRA-ABDOMINAL SOURCES

| | |
|---|---|
| Thorax: | Pneumonic consolidation, basal pleurisy, acute myocardial infarction (inferior wall) ↓ referred to upper abdomen |
| Spine: | Arthritis, radiculitis ↓ referred to upper, mid or lower abdomen according to level of lesion in spine |
| Genitalia: | Torsion of testis or ovary ↓ referred to lower abdomen |

## METABOLIC CAUSES OF PAIN ABDOMEN

- Diabetic ketoacidosis (polyuria, dehydration, Kussmaul's breathing)
- Acute intermittent porphyria (family history, ↑ BP, peripheral neuropathy)
- Uraemia (anorexia, pallor, oliguria)
- Lead poisoning (anorexia, headache, metallic taste)
- Hypercalcaemia (e.g. hyperparathyroidism — lethargy, confusion, polyuria).

## DIARRHOEA ALTERNATING WITH CONSTIPATION

- Intestinal tuberculosis
- Irritable bowel syndrome
- Carcinoma of colon
- Diverticulosis of colon

## MEDICAL CAUSES OF PAIN ABDOMEN

1. Basal pneumonia (i.e. basal pleurisy)
2. Acute myocardial infarction (commonly inferior wall infarction)
3. Diabetic ketoacidosis (may be due to acute gastric dilatation)
4. Intercostal herpes zoster (radicular pain)
5. Henoch-Schönlein purpura (vasculitis)
6. Sickle cell anaemia (vaso-occlusive crisis)
7. Acute intermittent porphyria (autonomic neuropathy)
8. Caries spine/cord compression/collapse of vertebra (radicular pain)
9. Lead poisoning (colic)
10. Torsion of testis
11. Polyarteritis nodosa (vasculitis)
12. Tabetic crisis (probably due to autonomic neuropathy)
13. Allergic pain ($C_1$ esterase inhibitor deficiency)
14. Functional
15. Miscellaneous–peptic ulcer, acute pancreatitis, acute hepatitis, hyperlipidaemia.

## ABSOLUTE 'WATERY' DIARRHOEA

Dehydration commonly develops in this setting from,

1. Enterotoxigenic *E. coli* (incubation period 12-72 hours)
2. Traveller's diarrhoea (H/O recent travel)
3. Cholera (fever, vomiting, severe watery diarrhoea)
4. Rota virus (in children mainly)
5. Norwalk virus (in older children and adults).

—— o ——

CHAPTER 46

# Macroglossia

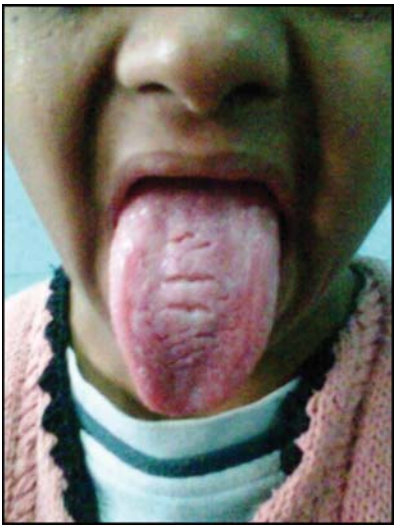


**FIGURE 46.1:** Macroglossia with grossly fissured/furrowed tongue in mongolism

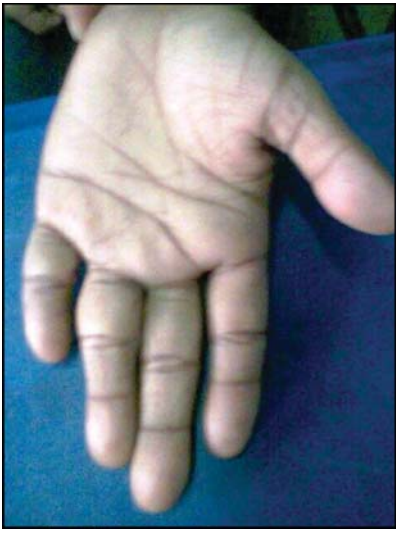


**FIGURE 46.2:** Clinidactyly – absent (or hypoplastic) middle phalanx as well as one crease in the little finger (produces incurved little finger) in Down's syndrome

## CLUE TO DIAGNOSIS

Ask the patient to protrude the tongue

↓

Enlarged tongue with indentation of teeth at the margins

↓

Macrosomia with prognathism: acromegaly

↓

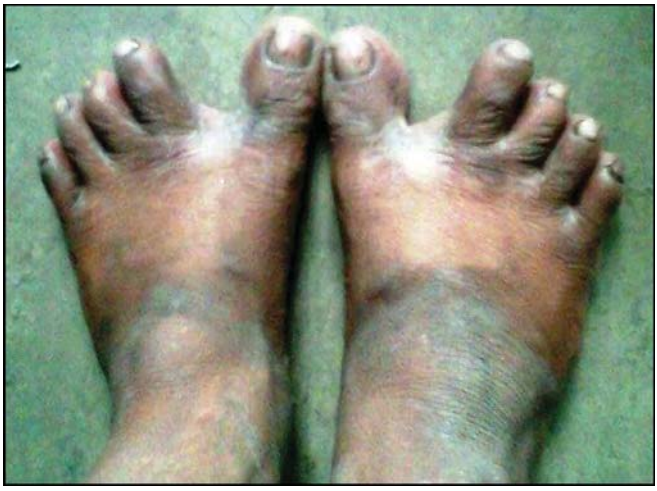


**FIGURE 46.3:** Sandal gap, i.e. increased gap between great toe and second toe in Down's syndrome

Mental retardation: cretinism, Down's syndrome (furrowed tongue), Hurler syndrome (mucopolysaccharidosis)

↓

Expressionless puffy face, baggy lower eyelids along with madarosis (loss of hair in lateral 1/3rd of eyebrow): myxoedema

↓

Tongue feels stiff and firm to palpation with hepatosplenomegaly and lymphadenopathy: primary amyloidosis

↓

'Doll-like' facies with retarded physical growth in a child: von Gierke's disease

↓

Facial palsy, oedema of lips, plication as well as deeply furrowed tongue: Melkersson's syndrome

↓

Surgical conditions like lymphangioma or haemangioma of tongue: colour changes, very large tongue, may have localised swelling

## MICROGLOSSIA

1. Motor neurone disease (MND)
2. Bulbar (flaccid) and pseudobulbar palsy (spastic)
3. Wasting of the tongue (LMN lesion of XIIth cranial nerve)
4. Cerebral diplegia.

## DIFFERENT MOVEMENTS OF TONGUE

- Tremor (fine; in anxiety neurosis, thyrotoxicosis, chronic alcoholism)
- Fasciculation (MND or bulbar palsy)
- Chewing tongue (athetosis)
- Lizard tongue–tongue is protruded momentarily and taken back within the oral cavity instantaneously (chorea)
- Jack-in-the-box tongue (Sydenham's chorea)
- Trombone tongue (general paresis of insane → GPI)
- Irregular and continual rotatory movement (dyskinesia, mainly drug-induced or extrapyramidal syndromes)
- Rolling movements (Down's syndrome, cretinism)

* *Method of examination:* For tremor–ask to protrude the tongue, for fasciculation–ask to keep the tongue in floor of the mouth, other movements–can be examined by opening the mouth.

## SIALORRHOEA (PTYALISM OR EXCESSIVE SALIVATION )

1. Carcinoma of the tongue or mouth (with cervical lymphadenopathy).
2. Caries tooth, stomatitis.
3. Wilson's disease (drooling of saliva with K-F ring in cornea).
4. Post-encephalitic parkinsonism (past H/O encephalitis, mental retardation, rigidity > tremor, plantar response may be extensor; seborrhoea).
5. Bulbar palsy (history is important; dysphagia, dyarthria, dysphonia with nasal regurgitation; small, wasted tongue).
6. Pregnancy.
7. Hydrophobia (H/O dog bite).
8. Schizophrenia, mania.
9. Arsenic, mercury or lead poisoning (area of residence, occupation or H/O poisoning).
10. Smell and sight of spicy food.

* Read 'Xerostomia' from the chapter on 'Parotid swelling'.

## DROOLING OF SALIVA (DRIBBLING OF SALIVA)

Common examples in clinical practice are:

1. Mental retardation
2. Stomatitis
3. Facial palsy
4. Wilson's disease

5. Epilepsy
6. During sleep (often embarrassing).

## SCROTAL TONGUE

- Tongue shows deep horizontal fissures where debris may collect
- Of no clinical importance.

## D/D OF ACUTE SWELLING OF TONGUE

1. Angioneurotic oedema or giant urticaria in tongue
2. Haemophilia or bleeding disorder (e.g. scurvy)
3. Pemphigus (a fatal 'blistering' disease)
4. Tongue bite (accidental or during convulsions) or sting
5. Infection (e.g. associated with stomatitis)
6. Corrosives or acute irritant applications
7. Effect of drugs: mercury, aspirin (rare).

—— o ——

CHAPTER 47

# Nerve Thickening

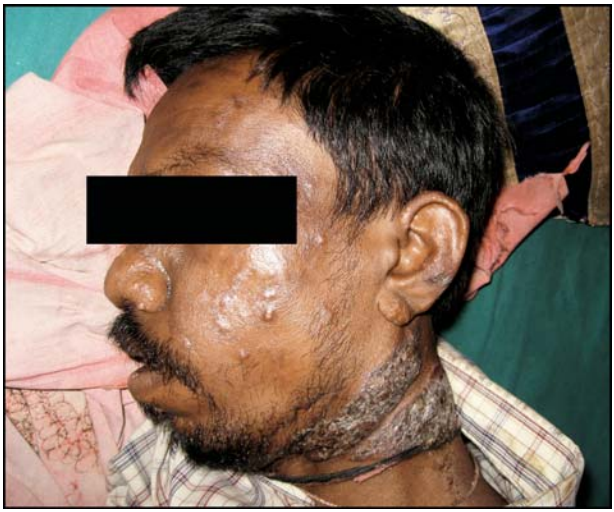


**FIGURE 47.1:** Nodular infiltrative lesions in leprosy with scrofuloderma in neck (simultaneous *M. leprae* and *M. tuberculosis* infections in a single patient)

## PERIPHERAL NERVES COMMONLY THICKENED (ESPECIALLY IN LEPROSY)

1. Great auricular nerve in the neck across the sternomastoid muscle: the nerve stands out by turning the head to opposite side.
2. Ulnar nerve at elbow.
3. Common peroneal nerve at the neck of fibula.

## NERVE THICKENING COMMONLY ASSOCIATED WITH

- Leprosy
- Neurofibromatosis

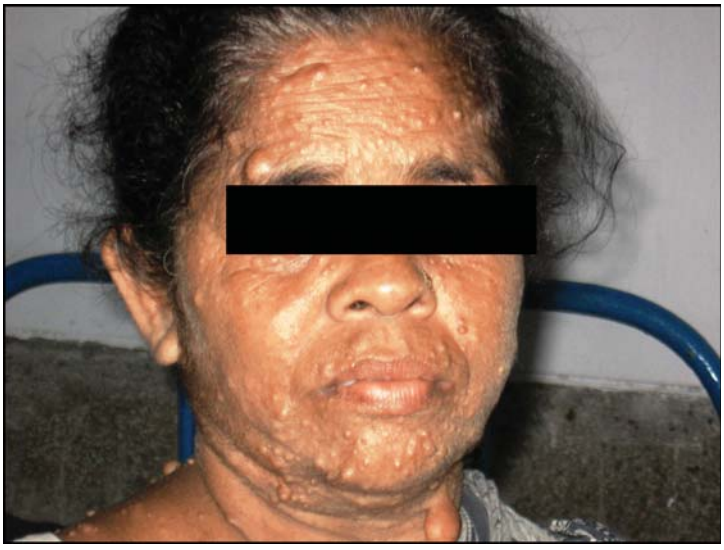


**FIGURE 47.2:** Multiple neurofibromatosis

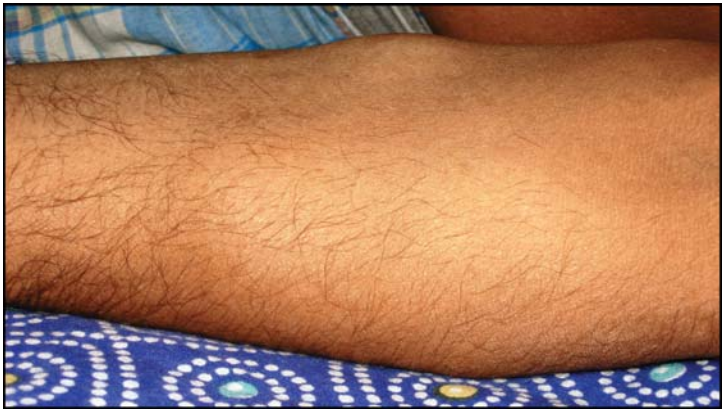


**FIGURE 47.3:** Subcutaneous swelling due to multiple neurofibromatosis

- Acromegaly
- Charcot-Marie-Tooth disease (juvenile)
- Amyloidosis
- Chronic Guillain-Barre syndrome or chronic inflammatory demyelinating polyneuropathy (CIDP)
- Sarcoidosis
- Dejerine-Sottas type neuropathy or Refsum's disease
- Idiopathic hypertrophic neuropathy
- Roussy-Levy syndrome.

## CLUE TO DIAGNOSIS

- Anaesthetic patch → leprosy
- Cafe-au-lait spots+kyphoscoliosis → neurofibromatosis
- Hepatosplenomegaly → amyloidosis, leprosy
- Lymphadenopathy → sarcoidosis, amyloidosis
- Parotid swelling, uveitis → sarcoidosis
- Ichthyosis, deafness, retinitis pigmentosa → Refsum's disease
- Facial palsy → chronic Guillain-Barre syndrome, sarcoidosis
- Prognathism, facial enlargement → acromegaly
- Macroglossia → acromegaly, amyloidosis
- Nerve biopsy: onion bulb formation → Dejerine-Sotta's or Refsum's disease.

—— o ——

CHAPTER 48

# Night Blindness

## SYNONYM

Nyctalopia

## ASSOCIATIONS

1. Vitamin A deficiency
2. Retinitis pigmentosa
3. Peripheral chorioretinitis
4. Chronic simple glaucoma associated with marked contraction of visual field
5. Myopic degeneration in the periphery of retina (i.e. congenital high myopia)
6. Detachment of the retina
7. Zinc deficiency
8. Malingering.

## DETECTION

Done by history (e.g. poor vision at night especially in feeble illumination), clinical examination (signs of vitamin A deficiency, e.g. Bitot's spot or xerophthalmia; ophthalmoscopy), and clinical detection by an instrument called 'adaptometer'. Nyctalopia results from either to the damage of rods or due to deficient regeneration of visual purple.

## DAY BLINDNESS (HEMERALOPIA)

Here, the vision is poor in bright light but better in dim light. Pupil dilates in dim light, and the peripheral retina is used for vision. The common causes are:

1. Pathological changes in macula
2. Central opacity of cornea or lens
3. Congenital, when associated with colour blindness.

## AMBLYOPIA

It means partial loss of vision, e.g. in suppression of macular function, and often associated with squinting eye in the child.

## AMAUROSIS FUGAX

It means sudden but 'temporary' complete loss of vision, e.g. as on rising suddenly from sitting position, migraine (from spasm of retinal arteries), etc.

## AMAUROSIS

It is the complete loss of vision. 'Acute' loss of vision is found in:

A. Painful:
- Acute angle-closure glaucoma
- Uveitis
- Corneal ulcer
- Endophthalmitis
- Trauma
- Factitious.

B. Painless:
- Central retinal vein thrombosis
- Retinal artery occlusion
- Optic neuritis
- Detachment of the retina
- Ischaemic optic neuropathy (e.g. as a complication of temporal arteritis)
- Vitreous haemorrhage (massive)
- Exudative macular degeneration
- Cerebrovascular accidents
- Factitious

* *'Chronic' progressive vision loss* is found in cataract, refractive error, macular degeneration, primary optic atrophy, chorioretinitis and open-angle glaucoma.

## COLOUR BLINDNESS

Three primary colours are red, green and blue. Congenital red or green colour blindness is X-linked and almost exclusively found in male children. Green-blindness is the commonest in clinical practice. Ideally colour vision is tested by Ishihara's chart (pseudo-isochromatic plates). The colour vision assesses the function of the retinal cones and optic nerve. The causes are:

A. Congenital (hereditary):
   a. Partial–cannot recognise primary colours
   b. Total–cannot recognise any colour and sees everything grey.

B. Acquired:
   a. Age-related macular disease
   b. Optic nerve diseases
   c. Toxic amblyopias, e.g. ethambutol or chloroquine-induced.

## TUBULAR VISION

Classical tubular vision is found in hysteria or psychogenic field defect (tunnel vision); may also be seen in advanced retinitis pigmentosa and terminal glaucoma (see chapter-22).

## PERIPHERAL DEFECT IN VISUAL FIELD

1. Retinitis pigmentosa (funnel vision)
2. Chorioretinitis (blurring or gradual loss of vision)
3. Psychogenic (tunnel vision; all other parameters–WNL)
4. Lesion in optic chiasma lesion (bitemporal hemianopia)
5. Lesion in optic tract (homonymous hemianopia)
6. Lesion in visual cortex (homonymous hemianopia or quadrantopia).

—— o ——

CHAPTER 49

# Nocturnal Enuresis

## SYNONYM

Bed-wetting, sleep enuresis, self-wetting

## DEFINITION

Nocturral enuresis is the involuntary voiding of micturition during sleep in the young. It is one of the parasomnias like sleep-walking or sleep terrors. Bed-wetting is considered a normal feature of development before the age of 5 years; usually improves at puberty and is rare in adults. Incidence rate is 1-3% in late adolescence and affection of males > females.

## CLINICAL PRESENTATION OF ENURESIS

- Nocturnal only–occurs in first third of sleep, frequently during REM sleep
- Diurnal only–more in girls, voiding in early afternoon on school days mainly
- Combined nocturnal and diurnal enuresis.

## CLINICAL ASSOCIATIONS

1. Lack of toilet training, anxiety, fear, ↑ intake of water
2. Epilepsy, mental subnormality, meningomyelocele, cauda equina lesion
3. Urinary tract infections (urethritis, cystitis), epispadias, phimosis, meatal stenosis
4. Threadworm infestation, neurovesical dysfunction.

## MANAGEMENT

1. Bladder training and behavioural therapy
2. Phimosis should be operated, if present
3. Secondary enuresis is commonly due to emotional disturbances. UTI, urinary tract malformations, cauda equina lesions, sleep apnoea or epilepsy → the cause should be corrected
4. Pharmacotherapy: desmopressin (0.2 mg at bedtime), oxybutynin (5-10 mg at bedtime), or imipramine (10-50 mg at bedtime).

## INCREASED FREQUENCY OF MICTURITION

Normally an adult with an average fluid intake evacuates his/her bladder approximately 4-5 times during daytime and once during night. If the micturition is more than this, it is known as increased frequency of micturition, which may be associated with increased or normal urine output.

*The common associations noticed are:*

A. Urinary tract infection (UTI) as a whole
B. Urinary bladder: cystitis, small bladder (e.g. thimble bladder), bladder stone/tumour, cystocele in females
C. Bladder neck: benign hypertrophy of prostate (BHP), uterine prolapse, lax internal urethral sphincter
D. Urethra: urethritis, stricture of urethra, phimosis, pinhole meatus
E. Psychogenic
F. As a part of polyuria
G. Pregnancy, and intra-abdominal tumour pressing over the urinary bladder

* 4 common causes in clinical practice are UTI, BHP, calculus in urinary bladder and uterine prolapse.

## IMPROPER URINARY STREAM

Improper urinary stream occurs when there is impairment in the smooth and free flow of urine.

For example, BHP is associated with nocturia, reduced size and force of urinary stream, straining to urinate and dribbling at the end; often these are associated with increased frequency, precipitancy and urgency.

*The common causes of improper urinary stream are:*

A. Children: Phimosis, meatal stenosis, posterior urethral valve
B. Adults: BHP, stricture of urethra (fork urine), UTI, bladder neck obstruction, vesical calculus, phimosis or bladder dysfunction due to neurological diseases (neurogenic bladder).

## HESITANCY AND PRECIPITANCY

One has to remember that parasympathetic nerves ($S_{2,3,4}$) are nerve of evacuation and sympathetic nerves ($L_{1,2,3}$) are nerve of filling of the urinary bladder.

Hesitancy–difficulty in initiating micturition in spite of presence of urge to do so.

Precipitancy–inability to stop voiding of urine when desire for micturition occurs.

*The common causes in clinical practice are:*

A. Uninhibited bladder–found in frontal lobe tumours, parasagittal meningioma, dementia. There is urgency at low bladder volume (like a child) with sudden uncontrolled evacuation of urine. It is also known as 'mental incontinence' (i.e. loss of social control of micturition).
B. Spinal bladder–incomplete lesion in spinal cord results in:
   a. Hesitancy–due to involvement of facilitatory fibres, e.g. incomplete cord compression
   b. Precipitancy–due to involvement of inhibitory fibres, e.g. multiple sclerosis
   c. Disorders of urinary tract–BHP, bladder neck obstruction.

## TREATMENT

1. Hesitancy–Reassurance and not to exert undue stain
2. Precipitancy–a condom catheter is applied in male patients. Surgical correction of the urinary tract, if needed.

## NOCTURIA

Increased frequency of micturition at night and passing of more than 1/3rd of the total daily output by night is nocturia. The common causes are:

1. All polyuric states
2. Oedema-forming states (i.e. cirrhosis of liver, congestive cardiac failure)
3. Diabetes mellitus

4. Benign hypertrophy of prostate
5. Salt-losing nephropathy
6. Cystitis
7. Vesico-ureteric reflux
8. Low bladder capacity (e.g. infection, tumour or stone).

## URINARY INCONTINENCE

It is the inability to hold urine in the bladder.

## TYPES OF URINARY INCONTINENCE

1. Physiological–upto 3 years of age.
2. Urge incontinence–the person cannot hold the urge and soils his/her cloth if toilet facility is not available nearby. It is due to overactivity of detrusor muscle and commonly results from UTI or vesical calculus.
3. Stress incontinence–urinary incontinence occurs after stress, e.g. coughing, sneezing or laughing (laughing-induced incontinence in young girls are known as giggle incontinence), and are commonly seen in multiparous women (due to obstetrical trauma). It is also noticed in males after prostatic surgery.
4. Overflow incontinence–commonly seen in acute transverse myelitis when the neural shock stage is over (retention of urine with overflow incontinence); may be seen in stricture of urethra or secondary to bladder neck obstruction.
5. True incontinence–the urine leakage is constant in vesico-vaginal fistula.
6. Psychogenic incontinence–found in children in an attempt to draw attention.

* Incontinence of urine is commonly seen in BHP (hesitancy, dribbling, ↑ frequency), UTI (pyrexia, dysuria, ↑ frequency), uterine prolapse (characteristically low volume bladder frequency), and weakness of pelvic floor muscles (stress incontinence).

## ANURIA

No urine formation for 12 hours (some nephrologists define anuria as < 100 ml urine output per day). Oliguria is < 400 ml of urine output per day.

## CAUSE OF LOW URINE OUTPUT

A. Oliguria–acute gastroenteritis, high fever, glomerulonephritis, congestive cardiac failure (decompensated), renal failure (acute and chronic), hypovolaemia and shock, 'third space' loss in acute peritonitis and acute pancreatitis.

B. Anuria –
   a. Pre-renal:
      1. Shock, sepsis (septicaemia), haemorrhage (massive)
      2. Dehydration due to any cause (e.g. acute gastroenteritis)
      3. Crush syndrome
      4. Burn (extensive)
      5. Intravascular haemolysis, mismatched blood transfusion
      6. Congestive cardiac failure
      7. Acute pancreatitis.
   b. Renal:
      1. Acute glomerulonephritis, rapidly progressive glomerulonephritis (RPGN)
      2. Acute renal failure (ARF)
      3. Acute papillary necrosis (diabetes, sickle cell disease, phenacetin-induced)
      4. Diffuse cortical necrosis
      5. Complete renal arterial and venous obstruction
      6. Chronic renal failure (produces anuria terminally).
   c. Post-renal:
      1. Reflex anuria (calculus in one ureter may produce reflex obstruction of the other ureter)
      2. Ligation of the ureters (accidental) or bilateral ureteric obstruction by clots, stone or crystals
      3. Ureteric obstruction due to retroperitoneal fibrosis or malignant infiltration around the ureters.

C. Complete anuria (i.e. no urine by catheterisation):
   1. Bilateral ureteric obstruction
   2. RPGN
   3. Diffuse cortical necrosis
   4. Bilateral renal artery stenosis.

## POLYURIA

Urine output persistently above 3 litres per day. The normal urine output for a healthy adult is approximately 1.5 litre (400 ml-3 litre) per day.

*The common causes of polyuria are:*

1. Chronic renal failure
2. Diabetes mellitus
3. Diabetes insipidus
4. Psychogenic polydipsia (compulsive water drinking)
5. Use of diuretics in patients with oedema
6. Diuretic phase of ARF
7. Hypercalcaemic nephropathy
8. Hypokalaemic nephropathy
9. Solute diuresis–glycosuria (diabetes mellitus), high protein tube feeding, infusion of mannitol
10. Transient polyuria–after epileptic seizure, paroxysmal atrial tachycardia, migraine or asthmatic attack; pregnancy.

—— o ——

CHAPTER 50

# Pallor

## DEFINITION

It is the waxy appearance of skin and mucous membrane. When the skin and mucous membrane lack the normal colour/complexion and look pale, it is known as pallor.

## PATHOGENESIS

In health, blood flows through capillaries and make the skin, mucous membrane and nails pink. Pallor depends on thickness and quality of skin, and quality and amount of blood in the capillaries. Thus in the presence of normal haemoglobin concentration, a person looks pale if he/she has thicker skin and nail than an average individual. A person, who is having ↓ RBC or ↓ haemoglobin, always looks pale. Pallor and anaemia are not interchangeable terms; there are many causes of pallor and anaemia is commonest of them.

***Pallor may be:***

a. Temporary–intense emotion, fright, shock (due to vasoconstriction, associated with cold and clammy skin)
b. Permanent–anaemia due to any cause or peripheral vasoconstriction (e.g. severe atopy, oedematous part or myxoedema)

Anaemia denotes the colour of the blood and itself a 'pathological' condition while pallor is a 'clinical' entity. A person without losing a drop of blood may become deadly pale (e.g. shock and collapse), and similarly a person looking severely pale may not be grossly anaemic (e.g. Sheehan's syndrome). It is difficult to detect pallor in deeply pigmented individuals.

The observation of increasing pallor by friends or relatives may point towards early manifestation of progressive anaemia or any other disease menifested by pallor (see below).

## SITES TO BE LOOKED FOR ANAEMIA

1. Lower palpebral conjunctiva
2. Tongue, especially the tip and the dorsum
3. Soft palate
4. Nail-beds (these are the windows of capillary network)
5. Palms, soles and general skin surfaces.

Pallor due to anaemia are best manifested in mucous membranes (e.g. mouth), conjunctiva and palmar creases. In a thick skinned person, mucous membranes are not pale. 'False pallor' may be due to anasarca (e.g. nephrotic syndrome), myxoedema or thick skin, and are mainly menifested in skin.

## CAUSES OF 'PALLOR WITHOUT ANAEMIA'

1. Peripheral circulatory failure (e.g. low cardiac output in acute LVF) or shock (ashen-grey pallor) or vasoconstriction due to any cause
2. Acute myocardial infarction
3. Very tight aortic stenosis or mitral stenosis
4. Myxoedema (pallor > anaemia)–pallor is due to vasoconstriction plus lemon-yellow tint due to carotenaemia plus anaemia
5. Nephrotic syndrome
6. Sheehan's syndrome or panhypopituitarism
7. Night workers
8. Thick skin (e.g. scleroderma)
9. Vasovagal attack, fear, exposure to cold, intense emotion, syncope due to any cause

* Patients of subacute bacterial endocarditis have a pallor which is known as cafe-au-lait pallor (i.e. colour of white coffee)

** A limb or part of a limb may look pale due to arterial spasm (e.g. Raynaud's phenomenon), exposure to cold, deprived of blood supply (e.g. ligature applied in snake bite) or having oedematous swelling

*** So, pallor may be of two types:

a. Pallor associated with anaemia
b. Pallor without anaemia.

—— o ——

CHAPTER 51

# Parotid Swelling

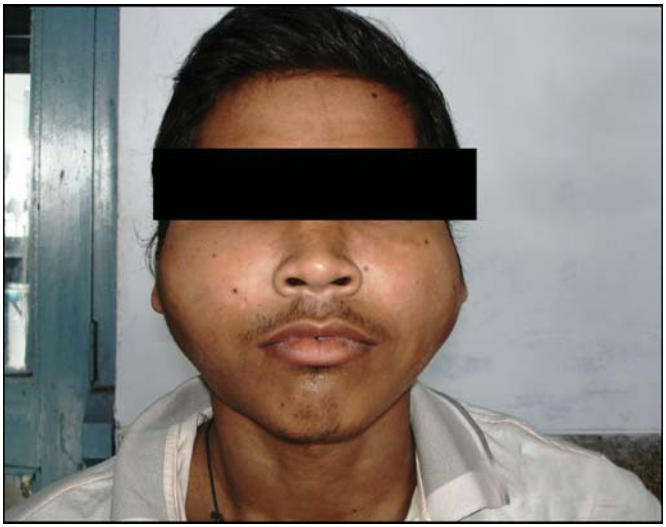


**FIGURE 51.1:** Bilateral parotid swelling in mumps

## STEPS TO DIAGNOSIS

A. Unilateral: think of tumour (look for LMN type VIIth nerve palsy), parotitis (painful and tender on palpation, H/O pyrexia)
B. Bilateral:
   - Mumps (commonly children with pyrexia and tender parotids)
   - Bacterial parotitis (with H/O poor oral hygiene, pyrexia ± hot and tender fluctuating swelling)
   - Chronic alcoholism (H/O alcohol intake with flushed face)
   - Cirrhosis of liver (especially, alcoholic cirrhosis + look for other stigma like gynaecomastia, Dupuytren's contracture, loss of body hair and features of portal hypertension)

- Sjögren's syndrome (xerostomia + keratoconjunctivitis sicca + arthritis)
- Sarcoidosis (uveoparotid fever or Heerfordt's syndrome → with facial palsy, may have generalised adenopathy, swelling of lacrimal glands, uveitis)
- Drug-induced ('iodine mumps' after IVP, phenylbutazone, guanethidine or bretylium → tender parotids)
- Amyloidosis (macroglossia with hepatosplenomegaly, malabsorption, nephrotic syndrome or lymphadenopathy)
- Metabolic: cirrhosis of liver, diabetes mellitus, chronic pancreatitis, hyperlipoproteinaemias
- Endocrine: acromegaly, gonadal hypofunction
- Leukaemic or lymphomatous deposits.

## MESSAGE

In a patient of parotid swelling, always examine:
- Other salivary glands, lacrimal glands
- Lymph nodes all over the body, especially the glands in neck
- Look for LMN type VIIth cranial nerve palsy.

## D/D OF PAROTID SWELLING

1. Hypertrophy of masseter muscle
2. Jaw tumours
3. Lymphadenopathy (preauricular nodes mainly)
4. Temporo-mandibular joint swelling (e.g. from rheumatoid arthritis)
5. Dental abscess
6. Infantile cortical hyperostosis (of mandible).

## DRY EYES (XEROPHTHALMIA)

1. Sjögren's syndrome (keratoconjunctivitis sicca)
2. Stevens-Johnson syndrome
3. Pemphigoid
4. Impaired lacrimal gland function
5. Hypovitaminosis A (advanced stage)
6. Anaesthetic cornea
7. Trachoma stage IV (due to severe scarring).

## DRY MOUTH (XEROSTOMIA)

1. Fear, anxiety, tension, high fever, dehydration, mouth breathing–transient.

2. Psychogenic
3. Drug-induced: diuretics, antihypertensives, parasympatholytics, psychotherapeutics, levodopa
4. Sjögren's syndrome, sarcoidosis, amyloidosis, dehydration
5. Irradiation for head and neck cancers
6. Diabetes mellitus, alcohol-intake–as a result of polyuria
7. Old age (especially females).

## MIKULICZ'S SYNDROME

Long-standing, painless parotid swelling + lacrimal and other salivary adenitis + fever + uveitis may occur in tuberculosis, Hodgkin's disease, leukaemia or SLE, and is known as Mikulicz's syndrome.

## DRUG-INDUCED SALIVARY GLAND SWELLING

1. Guanethidine
2. Bretylium
3. Iodides
4. Bethanidine
5. Phenylbutazone
6. Clonidine
7. Lead, mercury
8. Thiouracil.

—— o ——

CHAPTER 52

# Patch Tonsil

## PATCH OR MEMBRANES SEEN OVER TONSILS IN SITUATIONS LIKE

- Acute follicular tonsillitis
- Faucal diphtheria
- Thrush or candidial infection
- Agranulocytosis
- Acute lymphoblastic leukaemia (ALL)
- Vincent's angina (spirochaetes and fusiform bacilli)
- Infectious mononucleosis
- Milk card (in neonates and infants).

## CLUE TO DIAGNOSIS

- H/O sore throat, odynophagia, fever, weakness, intake of drugs like carbimazole should be enquired into
- Cervical lymphadenopathy (diphtheria, tonsillitis, infectious mononucleosis, ALL)
- Petechial rashes at the junction of hard and soft palate (infectious mononucleosis)
- Splenomegaly (ALL, infectious mononucleosis)
- Pyrexia (tonsillitis, diphtheria, ALL, infectious mononucleosis)
- Trismus (may be present in tonsillitis)
- Easily Detachable Membrane (all except diphtheria where the membrane cannot be separated easily and it leaves a bleeding, raw surface after separation)
- Blood for TC, DC (leucocytosis/agranulocytosis), abnormal cells (leukaemia), monospot and Paul-Bunell test (infectious mononucleosis)

## PREDISPOSING CONDITIONS FOR ORAL CANDIDIASIS

1. AIDS
2. Diabetes mellitus
3. Haematological malignancy
4. Hypoparathyroidism
5. Disseminated malignancy
6. Immunosuppressive therapy with corticosteroids, anti-cancer chemotherapy
7. Antibiotic therapy, e.g. penicillins
8. Neutropenia due to any cause (neutrophils are resposible for major host defence against candida)

* Candidiasis of mucous membranes is known as 'thrush'.

—— o ——

CHAPTER 53

# Photosensitivity

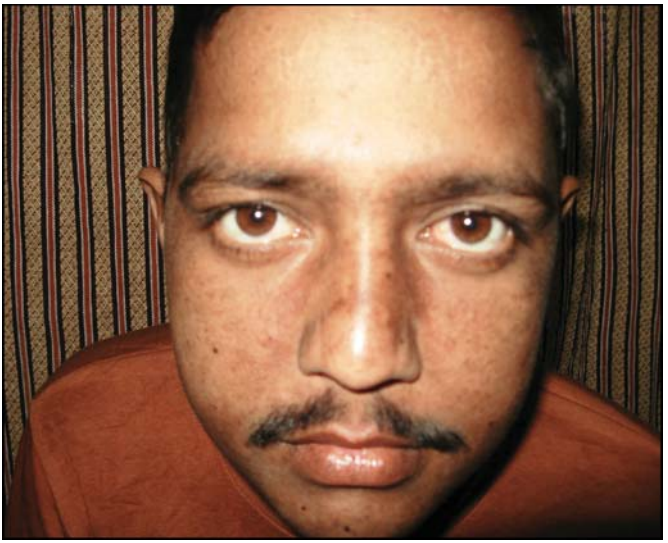


**FIGURE 53.1:** A male patient of SLE with butterfly rash in face

## PHOTOSENSITIVE DISEASES

- Solar urticaria
- Photoallergic reaction
- Erythropoietic porphyria
- Porphyria cutanea tarda
- Pellagra
- Kwashiorkor
- Carcinoid syndrome
- Hartnup disease
- Xeroderma pigmentosum
- Phenylketonuria
- Drug-induced
- Photoaging
- Polymorphous light eruption
- Basal cell carcinoma
- Actinic keratoses
- Actinic reticuloid
- Melanoma
- Phototoxic reaction

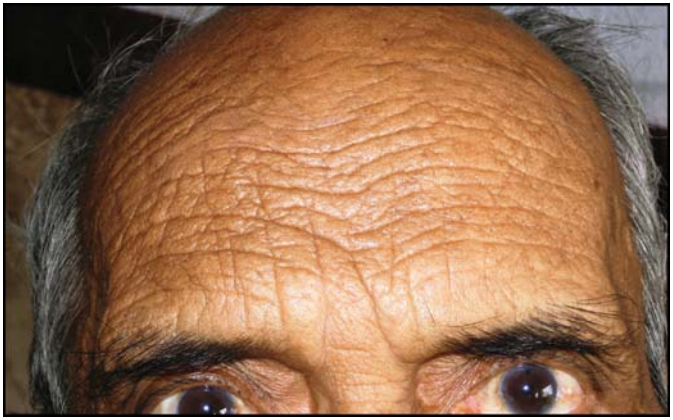


**FIGURE 53.2:** Chronic photodamage (photoaging): wrinkling and changes in photo-exposed forehead skin are due to chronic damage from solar UV radiation

## PHOTOSENSITIVE REACTIONS

- Phototoxic (nonimmunologic) reaction
  - Exaggerated sunburn reactions
  - Precipitated by tars, psoralens, plants (phytophotodermatitis) or phenothiazines
  - Erythema → peals off, oedema, vesicles, bullae
- Photoallergic (immunologic) reaction
  - Mimics contact dermatitis
  - Restricted to light-exposed areas (i.e. face, anterior 'V' of the chest, ears and back of the hands)
  - Common allergens are bithionol, buclosamide, plants like parthenium hysterophorus
  - Intensely pruritic eczematous dermatitis → lichenified

* Drug hypersensitivity, SLE and porphyria should be excluded in all photosensitive patients.

## PHOTOAGING

- A chronic effect of sun exposure
- Photodamaged skin consists of wrinkling, blotchiness, telangiectasia and a rough, irregular 'weather-beaten' appearance
- Also known as 'dermatoheliosis'.

## PHOTOTOXIC DRUGS

- Amiodarone
- Tetracyclines
- 5-Fluorouracil
- Frusemide

- Sulphonamides
- Sulphonylureas
- Phenothiazines
- Psoralens
- Vinblastine
- Retinoids
- Thiazides
- Dyes (methylene blue) or Rose Bengal
- Coal tar derivatives

## POLYMORPHOUS LIGHT ERUPTION

This is the commonest type of photosensitivity after sunburn → usually transient and ignored by the person → gradually 'hardening' of skin occurs → ultimately pruritic erythematous papules form and coalesce into plaques in sun-exposed areas of forearms and trunk → affection of face is mild → the diagnosis is done by skin biopsy and the treatment is aimed at use of sunscreens along with administration of artificial UV-A and UV-B for 2-3 weeks when maximum seasonal skin reaction occurs.

## BUTTERFLY-LIKE ERYTHEMATOUS LESION IN FACE

1. Systemic lupus erythematosus (SLE)
2. Rosacea
3. Malar flush
4. Lupus vulgaris (commonest form of skin tuberculosis)
5. Leprosy
6. Dermatomyositis
7. Post kala-azar dermal leishmaniasis
8. Pemphigus erythematosus
9. Chloasma or melasma ('mask of pregnancy')
10. Photosensitive reaction (after intake of tetracycline or phenothiazines)
11. Polymorphous light eruption
12. Sarcoidosis (rare).

* Butterfly rash in SLE is due to apoptosis of keratinized layer of skin exposed to sunlight/UV light.

## SPECTRUM OF ULTRAVIOLET RAYS

Three major segments (wavelengths between 10-400 nm)

- UV-C: Wavelengths 10-290 nm
  (does not reach the earth)
- UV-B: Wavelengths 290-320 nm
  (produces redness and erythema: the 'sunburn spectrum'.
- UV-A: Wavelengths 320-400 nm
  (very very less efficient than UV-B in producing skin hyperaemia)

Visible wavelengths 400-700 nm → the white light we see which breaks into VIBGYOR while passing through a prism

- Wavelengths > 700 nm → infrared rays (primarily evoke heat)

* The ultraviolet radiation can cause skin diseases (e.g. these are mutagenic and carcinogenic) and may also be used to treat certain dermatological ailments in necessity.

## PHOTOCHEMOTHERAPY

A. PUVA

Psoralens + UV-A therapy are used in:

- Psoriasis
- Vitiligo
- Cutaneous T-cell lymphoma.

B. PUVB

Psoralens + UV-B therapy are used for remission in psoriasis

—— o ——

CHAPTER 54

# Polycythemia

## DEFINITION

Polycythemia (often used interchangeably with erythrocytosis) is an increase in Hb, PCV and red cell count.

Polycythemia may be divided into absolute (true increase in red cell mass) or relative (↓ in plasma volume with normal red cell mass) erythrocytosis. Absolute erythrocytosis is due to primary polycythemia (i.e. polycythemia vera) and secondary polycythemia (i.e. anoxia, neoplasms).

## CLINICAL FEATURES

1. Facial plethora
2. Suffused conjunctiva
3. ↑ redness of lower palpebral conjunctiva
4. Oral mucous membrane turns dusky-red
5. Palmar erythema.

## SYNONYMS

Dusky cyanosis or ruddy cyanosis.

## SITES TO BE LOOKED FOR:

- Lower palpebral conjunctiva
- Tongue
- Soft palate
- Nail-beds
- Palms, soles and general skin surfaces
- Face

## D/D OF PLETHORIC FACE

- Chronic alcoholism
- Cushing's syndrome
- Chronic cor pulmonale
- SVC syndrome
- Carcinoid syndrome

## FEATURES OF POLYCYTHEMIA VERA

1. ↑ Red cell mass
2. Arterial $O_2$ saturation ≥ 92%
3. Splenomegaly (75% cases)
4. ↑ Platelets (> 4 lacs/$mm^3$)
5. ↑ WBC (> 12000/$mm^3$)
6. ↑ LAP (leucocyte alkaline phosphatase) score
7. ↑ Serum vitamin $B_{12}$ level (> 900 pg/ml).

## ERYTHROPOIETIN STATUS

- Polycythemia vera: ↓ EP
- Secondary erthrocytosis, e.g. hypoxia, neoplasms: ↑ EP

## NEOPLASMS ASSOCIATED WITH POLYCYTHEMIA

1. Renal cell carcinoma.
2. Hepatocellular carcinoma.
3. Uterine leiomyoma.
4. Cerebellar haemangioblastoma.
5. Ovarian carcinoma.
6. Pheochromocytoma.

## GAISBOCK'S SYNDROME

- Also known as 'relative', 'apparent' or 'spurious' polycythemia
- Originally thought to be stress-induced → decreased plasma volume with normal red cell volume
- However, spurious polycythemia is more common than polycythemia vera and seen in middle-aged men who are obese, smokers and hypertensive (probably diuretic therapy reduces the plasma volume)
- May present with AMI or cerebral infarction
- Treated by venesection; smoking stopped

## KEY FINDING FOR D/D OF POLYCYTHEMIA VERA FROM OTHERS

Splenomegaly

## COMMONEST CAUSE OF POLYCYTHEMIA IN A CITY HOSPITAL

Chronic obstructive pulmonary disease.

## HYPERVISCOSITY SYNDROME

### *Causes*

- Multiple myeloma
- Polycythemia vera
- Chronic myeloid leukaemia (CML)
- Essential thrombocythemia
- Myeloid metaplasia
- Waldenström's macroglobulinaemia
- Malignancy-induced (e.g. untreated acute leukaemia).

### *Features*

- Visual disturbances (mild vision loss to abrupt blindness)
- Gum bleeding
- Thrombotic episodes (CVA, AMI, perepheral vascular disease, mesenteric/hepatic vein thrombosis)
- Pruritus (especially, after a hot bath in polycythemia vera)
- Raynaud's phenomenon
- Neurological features, e.g. fatigue, malaise, dizziness, headache, fluctuating consciousness, transient paresis
- Peripheral cyanosis.

## OPHTHALMOSCOPY (HYPERVISCOSITY SYNDROME)

- Engorgement of retinal veins
- Vessel tortuosity
- Constriction at A-V crossings
- Areas of beading (vascular segmentation) and dilatation of small venules ('string of sausages' appearance)
- Haemorrhages and exudates.

## TREATMENT

- ℞ of basic disease

- Plasmapheresis
- Leucopheresis.

## TREATMENT OF POLYCYTHEMIA VERA

1. Phlebotomy (venesection).
2. Hydroxyurea; busulphan.
3. Radioactive $32_P$.
4. Baby aspirin (100 mg/day) to prevent thrombotic complications.
5. Allupurinol.

—— o ——

CHAPTER 55

# Pruritus

## BASIC PATHOPHYSIOLOGY

Pruritus is the unpleasant and irritating cutaneous sensation with intense desire to itch or scratch.

There are no end organs in the body specified for itching. The itch-receptors lie in the papillary layer of the dermis. The sensation of poorly-localised itching is principally carried by unmyelinated, slowly-conducting 'C' group of fibres to the central pool present in the spinal cord → posterior roots of the spinal nerves → thalamus → sensory area of gyrus postcentralis of the cortex. The well-localised itch is conducted by myelinated, rapidly conducting delta 'A' fibres.

Itching receptors are stimulated by several exogenous and endogenous stimuli, e.g. histamine, bradykinin, prostaglandins, eicosanoids, opioid peptides, proteases, bile salt, platelet activating factors etc. Inadequate lipid production (leads to lack of moisturing effect of skin) resulting in xerosis, and certain psychological factors (e.g. extreme emotional stress) may provoke itching.

## PRINCIPAL CAUSES

A. Local (i.e. itchy lesion in skin)
1. Scabies
2. Dermatitis herpeteformis
3. Lichen planus
4. Atopic dermatitis
5. Psoriasis
6. Pediculosis pubis
7. Xerosis (dry skin)

8. Seborrhoeic dermatitis
9. Urticaria
10. Contact dermatitis
11. Fungal infection (e.g. ringworm)
12. Pityriasis rosea
13. Lichen simplex chronicus
14. Drug eruption
15. Insect bite.

B. Systemic (i.e. internal causes)

1. Obstructive liver diseases, hepatitis B or C infection
2. Diabetes mellitus
3. Chronic renal failure
4. Lymphoma (especially, Hodgkin's disease), leukaemias
5. Multiple myeloma
6. Polycythemia vera
7. Iron deficiency anaemia
8. Systemic mastocytosis
9. Carcinoid syndrome
10. Multiple sclerosis
11. Intestinal parasitic infestations
12. Pregnancy (last trimester)
13. Abdominal or CNS tumour
14. Thyrotoxicosis or myxoedema
15. Neuropsychiatric disorders—delusions of parasitosis
16. Senile pruritus
17. AIDS
18. Drug-induced: aspirin, opiates, quinidine, phenothiazines, ultraviolet-A radiation.

* In Hodgkin's disease and primary biliary cirrhosis, pruritus may be the first symptom.

## PRURITUS DISTURBING SLEEP

Severe, persistent, irresistably severe pruritus is often paroxysmal in character and awakens the patient from sleep as soon as pain is induced by scratching (also called paroxysmal pruritus).

Severe itching in lichen simplex chronicus, atopic dermatitis, dermatitis herpeteformis, pediculosis corporis, uraemic pruritus, subacute prurigo, prurigo nodularis may interfere with patient's sleep. Usually, neither psychogenic nor senile pruritus leads to a loss of sleep.

## AQUAGENIC PRURITUS

Sometimes, itching is evoked by contact with water of any temperature. The common situations are:

- Polycythemia vera (specially after a warm bath)
- Hypereosinophilic syndrome
- Myelodysplastic syndromes
- Juvenile xanthogranuloma
- Fungal infections.

* Often +ve family history is obtained from the patient. There is ↑ degeneration of mast cells, and ↑ concentrations of histamine and acetylcholine which are liberated after contact with water. In polycythemia-induced pruritus aspirin, PUVA and therapy with interferon alfa-2b are tried.

## PRURITUS ANI

Very often pruritus is concentrated in perianal region (chiefly nocturnal) with little or no pruritus elsewhere. It is a social embarrassment for the patient. Possible aetiology could be:

1. Pinworm (Enterobius vermicularis) infestation, especially in children; other intestinal parasites; scabies.
2. Allergic contact dermatitis (from local anaesthetic used in suppositories for haemorrhoids, cathartics; failure to cleanse the area adequately after defaecation (poor anal hygiene); spicy foods/citrous foods or colchicine.
3. Anal tags, fissure, fistula.
4. Anal warts, condylomata lata (syphilis), anal granuloma; HSV or human papillomavirus.
5. Perianal candidiasis (diabetes mellitus).
6. Seborrhoeic dermatitis.
7. Lichen planus.
8. Psoriasis.
9. Fungal pruritus ani (e.g. erythrasma of groin and perianal region).
10. Anal neurodermatitis (characterised by paroxysm of violent itching leading to tearing and bleeding).
11. As a part of pruritus due to systemic causes.

* Proper cleansing of local area and treatment of specific aetiology are done. Topical pramoxine hydrochloride (non-steroidal topical anaesthetic) with hydrocortisone is tried.

## RECTAL PAIN (OR PERIANAL PAIN)

1. Anal fissure
2. Thrombosed haemorrhoids
3. Anorectal abscess
4. Rectal carcinoma
5. Pelvic inflammatory disease
6. Prostatitis
7. Compression/inflammation of sacral nerves
8. Impaction of faeces/foreign bodies
9. Proctitis (radiation, ulcerative, sexually-transmitted)
10. Miscellaneous: proctalgia fugax, coccygodynia (fleeting pain in rectum or coccyx, often related to sitting, but not with defaecation).

## PRURITUS VULVAE

Think of:

1. Vaginitis *(Candida albicans, Trichomonas vaginalis)*
2. Chronic cervicitis, carcinoma of cervix
3. Contact dermatitis (nylon, detergent, scent)
4. Lichen sclerosis of vulva, lichen planus
5. Scabies
6. As a part of generalised pruritus
7. Physiological: lack of cleanliness, menstruation, pregnancy, menopause, senility.

N.B: Always try to exclude diabetes mellitus, lymphoma, leukaemia and anxiety states in a case of pruritus vulvae

## SUGGESTED WORK-UP FOR CHRONIC GENERALISED PRURITUS

1. Thorough history with psychoanalysis
2. Meticulous clinical examination
3. Blood for TC, DC, ESR; sugar (F)
4. Chest rontgenography
5. Thyroid, renal and liver function tests
6. Hepatitis B and C serology
7. Screening for internal malignancy (CT/MRI scan, tumour markers).

## TREATMENT MODALITIES

℞ of primary cause (e.g. cholestyramine in cholestasis), PUVA therapy in uraemia/mastocytosis/chronic liver diseases, antihistaminics,

$H_2$-receptor antagonists (e.g. in polycythemia, mastocytosis), benzodiazepines, corticosteroids, topical emollient cream (e.g. calamine lotion), aspirin; acupuncture, mechanical vibratory stimulation, transcutaneous electrical nerve stimulation may be beneficial in some patients.

Nowadays anti-*H. pylori* ℞, terbutaline or montelukast are tried in nagging cases.

—— o ——

CHAPTER 56

# Ptosis

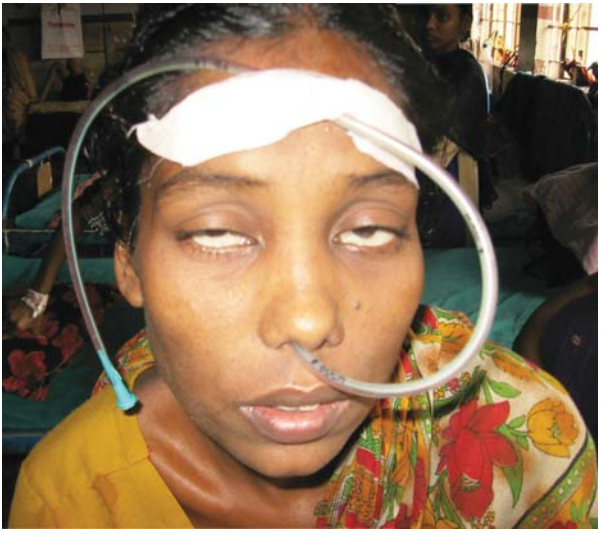


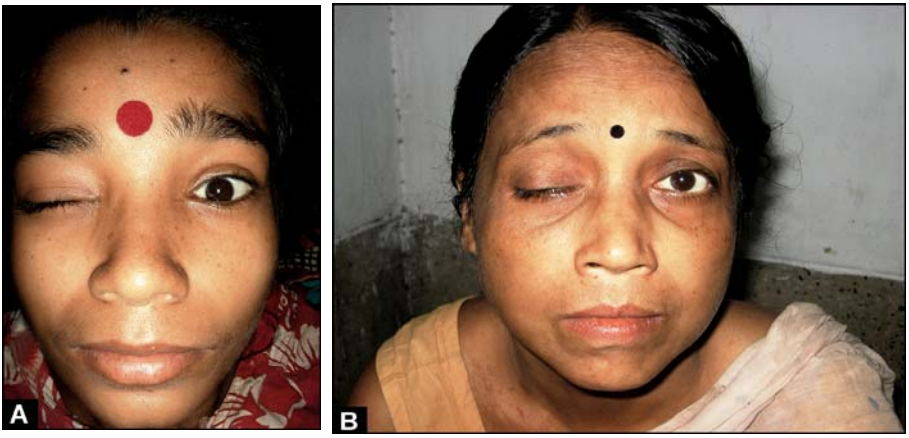
**FIGURE 56.1:** Bilateral partial ptosis in myasthenia gravis (Ryle's tube introduced to combat nasal regurgitation)

## DEFINITION

Drooping of the upper eyelid.

## MECHANISM

The upper eyelid muscles are levator palpebrae superioris (LPS, supplied by IIIrd cranial nerve) and Muller's muscle (supplied by sympathetic trunk). Paralysis of either of the nerves gives rise to ptosis. In LPS palsy, there is complete ptosis whereas in Muller's muscle palsy, there is




**FIGURES 56.2A and B:** Complete ptosis in right eye – patients with right sided IIIrd cranial nerve palsy (A – from intracranial SOL; B – from type 2 diabetes mellitus)

development of partial ptosis. In LPS weakness, on attempted elevation of upper eyelid there is compensatory, contraction of frontal belly of occipitofrontalis muscle, and thus increased furrowing in the forehead may be seen in any long-standing ptosis. In orbicularis oculi paralysis (VIIth cranial nerve palsy), there may be apparent diminusion of palpebral fissure but ptosis is never present.

## TYPES

1. Blepharoptosis
2. Mechanical
3. Aponeurotic
4. Myogenic
5. Neurogenic

1. Congenital
2. Acquired

1. Complete
2. Partial

## DESCRIPTION

I. Blepharoptosis–unilateral or bilateral, may be congenital dysgenesis of the LPS or from abnormal insertion of aponeurosis of LPS into the eyelid. A history of eye surgery, old trauma, contact lens, or a family history of ptosis should be sought for. Inspection of old photographs may help to diagnose an acquired ptosis.

II. Mechanical–excessive weight [e.g. oedema of the upper eyelid, tumours or dermatochalasis (redundancy of eyelid skin and subcutaneous fat, commonly seen in elderly)] and conjunctival scarring are two chief causes.

III. Aponeurotic–as a result of restricted transmission of force from LPS muscle to the upper eyelid, and is due to acquired dehiscence or stretching of the aponeurotic tendon, which connects the LPS muscle to the tarsal plate. This is commonly due to cataract surgery, blunt orbital trauma or sequela of eyelid swelling from infection.
IV. Myogenic–the causes are myasthenia gravis, and different ocular and oculopharyngeal myopathies; myotonic dystrophy also produces ptosis.
V. Neurogenic–developed as a result of IIIrd cranial nerve (complete ptosis) or sympathetic palsy (partial ptosis).

## DIFFERENT CAUSES

1. IIIrd cranial nerve (oculomotor) paralysis
2. Sympathetic paralysis (Horner's syndrome)
3. Tabes dorsalis
4. Myasthenia gravis
5. Myotonic dystrophy or ocular myopathy
6. Congenital
7. Snake bite (Elapidae group)
8. Botulism
9. Periodic paralysis
10. Hysterical
11. Oedema or tumour of upper eyelid, enucleation of the eyeball
12. Temporal arteritis

* Congenital ptosis may be unilateral or bilateral. Unilateral: 1, 2, 6, 10, 11, 12; bilateral: 3, 4, 5, 6, 7, 8, 9, 10; unilateral ptosis is a recognised finding in clusture headache (a variety of migraine), syringobulbia and cavernous sinus thrombosis.

## HOW TO RECOGNISE PARTIAL PTOSIS ?

It is clinically evaluated by the narrowing of the palpebral fissure. By standing in front of the patient, the width of the palpebral fissure is measured in primary gaze. Partial ptosis is characteristic of Horner's syndrome, recovering IIIrd nerve palsy, myasthenia gravis, myotonic dystrophy and snake bite.

## SOME FACTS

- Congenital ptosis–the horizontal wrinkling in the upper eyelid is absent (i.e. there is smoothness of upper eyelid)

- Myasthenia gravis–'fluctuating' ptosis and most prominent towards the end of the day (i.e. evening onwards); no alteration in pupillary reflex or size
- Horner's syndrome–this syndrome produces partial ptosis which is also known as pseudoptosis, i.e. the ptosis is corrected on looking upwards voluntarily
- Hysterical–blepharospasm and hysterical ptosis (both are spastic ptosis) are associated with wrinkling of the eyelid and angle of the eye, and absent contraction of frontalis muscle
- Marcus-Gunn jaw-winking phenomenon–ptosis occurs on opening of the jaw as a result of anomalous communication between IIIrd and Vth cranial nerves
- Senile ptosis–senile atrophy of skin and subcutaneous tissue are commonly seen after cataract, glaucoma and retinal detachment surgeries
- Ptosis and pupil:

i. Ptosis + dilated pupil → IIIrd nerve palsy.
ii Ptosis + constricted pupil → Horner's syndrome.
iii Ptosis + normal-size pupil → myasthemia gravis, myotonic dystrophy, botulism (in majority), snake bite, congenital variety, hysterical and rarely in infarction of IIIrd nerve (i.e. vasculitis or diabetes mellitus).

## COMPONENTS OF THIRD NERVE PALSY

- Complete ptosis
- Lateral squint
- Fixed and dilated pupil.

## COMPONENTS OF HORNER'S SYNDROME

- Pseudoptosis
- Miosis (constriction of the pupil)
- Anhidrosis (of ipsilateral half of face and neck, front and back of upper chest, arm)
- Enophthalmos
- Loss of ciliospinal reflex

N.B: Exophthalmos in one eye may mislead the clinician by putting a diagnosis of ptosis (partial) in the other eye; the pupillary size in the other eye helps in that situation to determine whether the ptosis is true or false (i.e. pupillary size remains normal).

—— o ——

CHAPTER 57

# Purpuric Spots

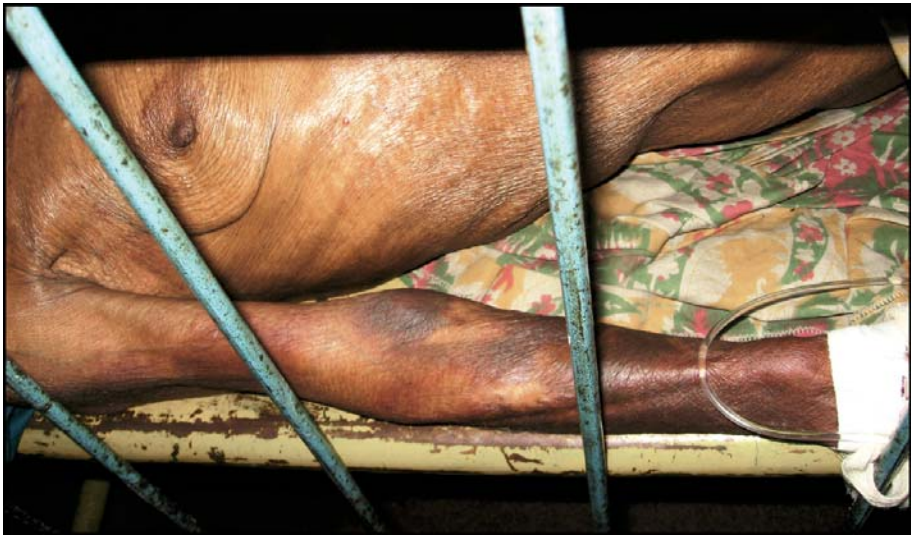


**FIGURE 57.1:** Subcutaneous haemorrhages (echymoses) in right upper limb in an aged unconscious patient

## CLASSIFY HAEMORRHAGIC SPOTS

- Petechiae–1-2 mm in diameter (pin-head size)
- Purpura–2-5 mm in diameter
- Ecchymosis/bruise–large purpura
- Suggillation–>20 mm in diameter
- Haematoma–large haemorrhages in the skin with surface elevation

* Haemorrhagic spots never blanch on compression as they contain extravasated blood

** Haemorrhagic spots are collectively known as 'purpuric spots'.

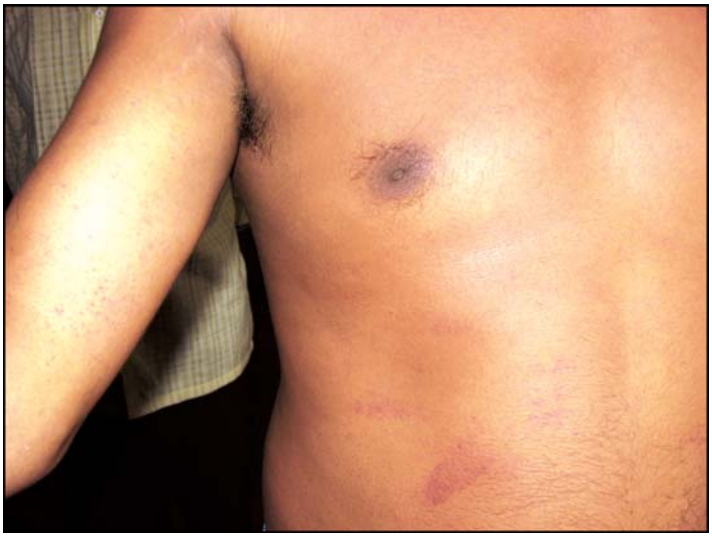
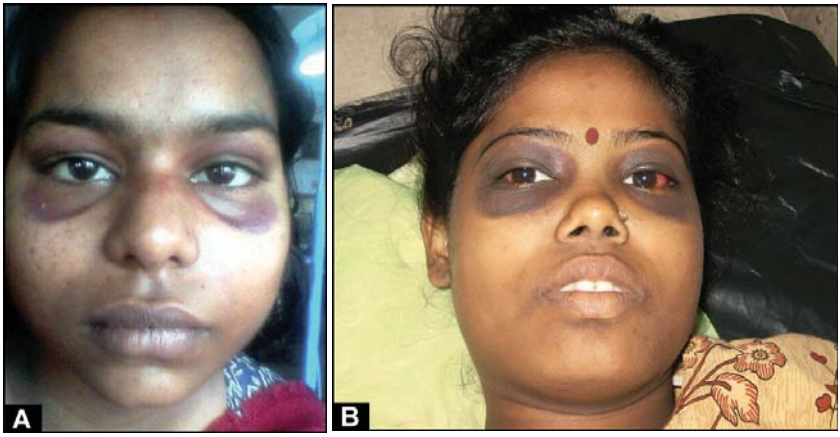


**FIGURE 57.2:** Subcutaneous haemorrhages—petechiae/purpura in inner aspect of right arm and ecchymoses in the trunk, seen in a patient of idiopathic thrombocytopenic purpura (ITP)




**FIGURES 57.3A and B** 'Panda sign' in patients with bleeding manifestations from (A) idiopathic thrombocytopenic purpura, and (B) acute leukaemia

## MECHANISMS

Purpuric spots are produced due to thrombocytopenia and/or vessel wall abnormalities and/or platelet function defect

- Petechiae/purpura → bleeding from small capillaries into skin, mucous membrane or retina
- Ecchymoses/bruises → bleeding from vessels which are larger than capillaries; suggillations, haematoma commonly associated with bleeding disorders or coagulopathy.

## COMMON SITES

Commonly legs are involved. Rashes may be seen over buttocks too.

## CLINICAL EXAMINATIONS

- Anaemia
- Examine the oral cavity and whole of the body surface (skin)
- Lymph nodes, all over the body
- Sternal tenderness
- Liver and spleen
- Ophthalmoscopy.

## PURPURA WITH SPLENOMEGALY

1. Acute leukaemias
2. Lymphoma
3. Systemic lupus erythematosus (SLE)
4. Idiopathic thrombocytopenic purpura (ITP) → spleen is palable in 10% cases only
5. Blast crisis of CML and rarely CLL
6. Subacute bacterial endocarditis (SBE)
7. Myelofibrosis.

## CRITICAL PLATELET COUNT

10000/$mm^3$. Below this level, spontaneous haemorrhage in any vital organ may endanger the life of the patient.

## HISTORY

1. Bleeding gum, epistaxis, menorrhagia or haematuria
2. Joint pain and/or swelling
3. Drug intake (sulphonamide, penicillins, aspirin, quinine, etc. may have side effects as thrombocytopenia).

## QUALITY OF PURPURIC RASH

Thrombocytopenia/thrombasthenia → legs commonly; flat purpura.
Vessel wall abnormalities/vasculitis → may involve buttocks; commonly urticarial (i.e. raised and itchy).

## HOW TO SUSPECT INTERNAL BLEEDING?

- Shock and peripheral circulatory failure
- Pallor

- Tachycardia and hypotension
- Internal organs
  - Chest (haemothorax)
  - CVS (cardiac tamponade)
  - CNS (neck rigidity, subhyaloid haemorrhage)
  - Retroperitoneum (Cullen's sign and/or Grey Turner's sign)
  - GI tract (distended and tender abdomen; rebound tenderness+)
  - Intramuscular (swelling of thigh in fracture femur).

## CAUSES OF PURPURA

- Thrombocytopenia or platelet functional defects
- Meningococcaemia, echo or coxsackie-infection, septicaemia
- Henoch-Schönlein purpura
- Hereditary haemorrhagic telangiectasis
- Purpura simplex (devil's pinches)
- Harmolytic-uraemic syndrome
- Paraproteinaemias
- Scurvy
- Rocky Mountain spotted fever
- Disseminated intravascular coagulation (DIC)
- Cryoglobulinaemia
- Vasculitis
- Hyperglobulinaemic purpura
- Cushing's syndrome
- Uraemia.

## MESSAGE

- Purpuric spots should be clinically differentiated from telangiectasia (small, dilated blood vessels visible on skin surface), mosquito bite marks, spider naevi, cherry angioma and drug rashes
- Bleeding from mucous membrane (gum), nose (epistaxis), skin (petechiae, purpura), per vaginum → platelet disorders
- Bleeding into joints, muscles → haemophilia
- Bleeding from multiple sites → DIC
- Bleeding out of proportion to anaemia → think of acute leukaemias, aplastic anaemia, coagulation disorders, anticoagulant therapy and SLE instead of simple thrombocytopenia where anaemia is usually proportionate to bleeding

* Telangiectasia, spiders and fresh mosquito bite marks blanch on pressure.

## LIVEDO RETICULARIS

It is the cyanotic mottlings of skin with a fishnet appearance, and is usually seen in:

- Antiphospholipid syndrome
- Atheroembolism (left atrial myxoma, cholesterol emboli, SBE)
- Leucocytoclastic vasculitis (hypersensitivity vasculitis)
- Cryoglobulinaemia
- Thrombocythemia or polycythemia
- Pancreatitis, SLE, dermatomyositis
- Polyarteritis nodosa (chronic cases).

—— o ——

CHAPTER 58

# Purse-lip Respiration

## CONDITIONS ASSOCIATED

Chronic obstructive pulmonary diseases (COPD), i.e. where there is chronic obstruction to airflow due to chronic bronchitis and/or emphysema, or chronic bronchial asthma (some cases).

## WHY THERE IS PURSE-LIP RESPIRATION?

Pursing of lips during expiration helps the patient to maintain high intrabronchial pressure above that within the surrounding alveoli (alveoli are overinflated or air-trapped in COPD patients) and thus helps to prevent the collapse of bronchial walls by overinflated alveoli.

## BEDSIDE FINDINGS IN ADVANCED AIRFLOW OBSTRUCTION

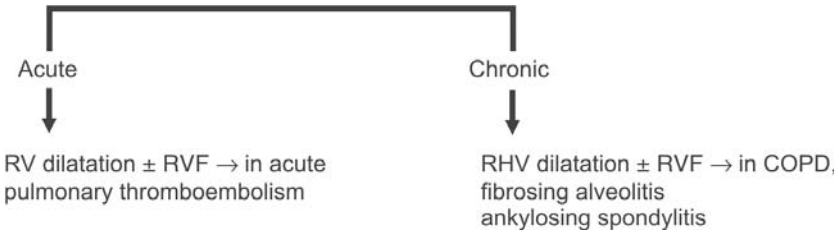
1. Dyspnoea and even orthopnoea
2. Hyperactive accessory muscles of respiration
3. ↓ in the length of trachea palpable above the suprasternal notch
4. Tracheal tug, i.e. tracheal descent during inspiration
5. Inspiratory excavation of the suprasternal notch and supraclavicular fossa
6. Expiratory filling of neck veins
7. Central cyanosis
8. Intercostal suction
9. Ralative ↑ in antero-posterior diameter of the chest
10. Wheeze.

## AIR HUNGER

Deep sighing, rapid breathing at a regular rate and with a hissing sound (i.e. hyperpnoea) is known as Kussmaul's breathing or air hunger. It is commonly seen in metabolic acidosis and may be called as 'acidotic breathing'. It is seen in:

- Diabetic ketoacidosis
- Uraemia
- Cerebral tumour
- Sometimes in hepatic coma

## WHAT IS COR PULMONALE?




## SIGNS OF DETERIORATING PULMONARY FUNCTION (E.G. ACUTE SEVERE ASTHMA)

1. Tachypnoea, severe dyspnoea
2. Rising pulse rate
3. Central cyanosis
4. Inability to speak
5. Progressive exhaustion
6. Altered sensorium
7. Profuse perspiration
8. Pulsus paradoxus
9. Silent chest.

## MOUTH BREATHING

- Nasal obstruction due to any cause, e.g. adenoids, nasopharyngeal fibroma, deviated nasal septum, antrochoanal polyp, mental subnormality etc.
- If starts from childhood, and there are presence of:
  - High arched palate
  - Crowding of teeth

- Underslung lower jaw and protrusion of upper jaw
- Absence of nasolabial furrows.

## HYPERVENTILATION

- ↑ depth of respiration (↑ rate + ↑ depth is hyperpnoea; ↑ rate is tachypnoea)
- Persistent hyperventilation is seen in:
  - Hysteria (young females mainly, if associated with 'hyperventilation syndrome', patient may complain of giddiness, tingling of extremities, black-out, weakness, palpitation; "main d' accoucheur" hand)
  - Anxiety neurosis, phobia, pain, panic reaction
  - Salicylate overdose
  - Pain anywhere in the body
  - Metabolic acidosis (diabetes mellitus, uraemia)
  - Pyrexia
  - Fibrosing alveolitis, pneumothorax, bronchial asthma, pulmonary thromboembolism
  - Midbrain or pontine lesions.

## SIGHING RESPIRATION

- Commonly seen in young females and children; functional in nature
- Deep inspiration → gap → forceful and deep expiration
- Occur in the presence of relatives or parents; never occurs during sleep or when left alone
- May have H/O 'hyperventilation syndrome' → giddiness, black-out, tingling of extremities, circumoral numbness, palpitation, chest tightness with features of tetany (carpopedal spasm).

## PERSISTENT HYPERCAPNOEA

It is commonly seen in:

- COPD
- Hypercapnoea with normal lung–metabolic alkalosis, primary alveolar hypoventilation, encephalitis
- Severe kyphoscoliosis, ankylosing spondylitis
- Myasthenia gravis, GB syndrome, poliomyelitis, motor neurone disease

## NOISY BREATHING

- URT infection, peritonsillar/retropharyngeal abscess, epiglottitis, tracheitis, bronchitis, bronchiolitis
- Bronchial asthma, angioedema, rhinitis
- Laryngeal webs, laryngo-tracheomalacia, cystic fibrosis, bronchiectasis
- Nasal polyp, hypertrophied adenoids/tonsils, vocal cord palsy.

## RESPIRATORY PATTERNS IN COMA

1. Post-hyperventilation apnoea (periods of apnoea coming after 5-10 deep breaths)–indicates bifrontal disease.
2. Cheyne–Stokes respiration (periods of apnoea rhythmically alternates with periods of hyperpnoea, i.e. a variety of periodic breathing)–indicates supratentorial lesion, i.e. massive cerebral lesions.
3. Central neurogenic hyperpnoea (regular breathing with increased rate and depth)–indicates midbrain-upper pontine lesion.
4. Apneustic breathing (a pause of 2-3 seconds after full inspiration).
   or
   Biot's breathing (i.e. 3-4 respirations without waxing or waning, followed by a pause)–indicates lower pontine lesion.
5. Ataxic breathing (with chaotic rate, depth and rhythm)–indicates medullary lesion.

—— o ——

CHAPTER 59

# Rectal Bleeding

## DEFINITION

It is the passage of bright red (i.e. fresh) blood rectally during defecation. Blood in stool may be present in three different forms like,

1. Frank blood or haematochezia
2. Altered blood or melaena
3. Invisible blood or occult blood (detected chemically at laboratory).

Visible blood per anum signifies bleeding from a source which is usually distal to ligament of Treitz, in comparison to melaena where the bleeding site is proximal to ligament of Treitz.

* Frank blood per anum with or without stool is haematochezia.

** Massive upper GI bleed may give rise to bright or dark red (maroon) coloured stool, if there is hurried peristalsis.

## POSSIBLE ASSOCIATIONS

A. Anal:
Fissure, fistula, trauma, foreign body, carcinoma, prolapse of mucosa.

B. Rectal:
Haemorrhoids, polyp, carcinoma, foreign body, proctitis (ulcerative/radiation), villous adeaoma, solitary rectal ulcer.

C. Colon:
Carcinoma (especially left colon), diverticular disease, angiodysplasia, dysentery (amoebic/bacillary), colitis (ischaemic/radiation/uraemic/infectious), inflammatory bowel disease, irritant drug-induced.

D. Small intestine:
Ischaemic bowel disease (mesenteric embolism/thrombosis, vasculitis), Peutz-Jeghers syndrome, tuberculous enteritis, Crohn's

disease, volvulus, intussusception, Henoch-Schönlein purpura, lymphoma/Kaposi's sarcoma, Meckel's diverticulitis, hereditary haemorrhagic telangiectasis, aorto-enteric fistula.

E. Miscellaneous:
Blood dyscrasias, anticoagulant therapy, uraemia, haemangioma, endometriosis of rectum (rare), massive upper G.I bleeding.

## HOW TO MANAGE THIS PATIENT AT THE INITIAL PART?

- IV infusion + colloids
- Check Hb, urine (R/E)
- CVP line
- Blood transfusion
- Thereafter investigations like rectal examination, protoscopy and sigmoidoscopy should be done.

## BLACK STOOL IN CLINICAL PRACTICE

- Melaena
- Ingestion of iron (as a haematinic)–usually associated with hard stool
- Ingestion of bismuth (in anti-H. pylori treatment)
- Intake of licorice, charcoal (in the treatment of poisoning) or black berries.

* Other than melaena, all are non-sticky ('pseudomelaena').

## CHARACTERISTICS OF MELAENA STOOL

Melaena is 'altered blood in stool' with features of:

- Black tarry stool (due to acid haematin); sticky too
- Offensive (acid haematin is altered by bacteria)
- Semisolid in consistensy
- Red-coloured fluid comes out from the stool after addition of water
- Usually associated with vertigo, dizziness or syncopal attack during defecation.

## SEVEN COMMON CAUSES OF BRIGHT RED BLEEDING PER RECTUM

- Haemorrhoids
- Polyps
- Diverticular disease
- Colitis

- Angiodysplasia of colon
- Carcinoma
- Ischaemic bowel disease.

Note: In the presence of severe diverticular disease, a polyp or carcinoma should always be excluded by colonoscopy as they can be the cause of bleeding. Haemorrhoids are so common that they should not be granted as the cause and effect of rectal bleeding in a particular patient → always search above.

## FROM BENCH TO BEDSIDE

- Anal fissure: bright red; clearly seperated from the faeces → often seen only in toilet paper; associated with anal pain 'during and after defecation'
- Haemorrhoids: profuse → splash the toilet pan and/or continue 'following defecation'
- Colitis (e.g. ulcerative): associated with urgency of defecation → passage of liquid or semisolid bloody stool mixed with mucus and pus.

## BLEEDING PER RECTUM WITH ACUTE ABDOMEN

- Mesenteric ischaemia
- Ischaemic colitis
- Necrotising enterocolitis
- Intussusception.

## 'TENESMUS' IN CLINICAL PRACTICE

It is the feeling of incomplete evacuation with a constant desire for defecation, and is commonly seen in:

- Infective colitis (e.g. amoebic and bacillary dysentery)
- Rectal carcinoma
- Rectal prolapse
- Tumour of descending colon
- Irritable bowel syndrome
- Pelvic inflammatory disease.

—— o ——

CHAPTER 60

# Recurrent Oral Ulcers

## DEFINITION

Recurrent oral ulcers (ROUs) consist of repeated bouts of one/more, small/large, painless/painful ulcers occurring at intervals of days to a few months.

## POSSIBLE ASSOCIATIONS

1. Unknown: aphthous ulcer, periodic fever, tumour necrosis factor receptor-associated periodic syndrome.
2. Infections: HSV (type 1 mainly), HIV, herpes zoster (in acute infective phase), CMV in immunocompromized host, coxsackie A (hand, foot and mouth disease).
3. Rheumatic diseases: Behcet's disease, SLE and DLE, reactive arthritis (Reiter's syndrome), Sweet's syndrome.
4. Dermatological diseases: erythema multiforme, toxic epidermal necrolysis, pemphigus vulgaris, lichen planus, bullous pemphigoid, dermatitis herpeteformis, 'epidermolysis bullosa'.
5. Haematological diseases: cyclic neutropenia, leukaemia, immunodeficiency disorders.
6. Gastrointestinal diseases: coeliac disease, inflammatory bowel diseases (IBD), i.e. ulcerative colitis and Crohn's disease.
7. Traumatic: laceration by sharp teeth, tooth brushing, ill-filted dentures.
8. Drugs: NSAIDs, β-blockers, nicorandil, alendronate, gold salts, antimalarials, sodium lauryl sulfate (a component of toothpaste).

## NOTABLE CAUSES OF ROU

- Aphthous ulcer
- Coeliac disease
- IBD
- Behcet's disease
- SLE
- Erythema multiforme
- Neutropenia (cyclic)
- Oral trauma
- Idiopathic.

## RECURRENT APHTHOUS STOMATITIS (RAS)

These are also known as aphthae or canker sores. RAS is prevalent in women, < 40 years of age, whites, non-smokers, and people of high socio-economic status. Probably, it is the commonest cause of ROU in a community. These are recurring painful ulcers of the mouth that are round or ovoid, shallow with a grey/white centre, and have inflammatory halos. The ulcers typically appear first in childhood (may have a positive family H/O RAS) and tend to abate around the third decade. The term 'RAS' should be reserved for recurrent ulcers confined to the mouth (labial or buccal mucosae, floor of the mouth, ventral surface or sides of the tongue), and seen in the absence of any systemic disease. There is associated lymphadenopathy. However, ulcers that resemble RAS, can be found in systemic disorders like Behcet's syndrome, coeliae disease or IBD, and immunodeficiency syndromes such as HIV infection or cyclic neutropenia.

### *Types*

Minor and major type. *Minor* (80%) aphthous ulcers are < 10 mm in diameter, affect non-keratinised mucosae, and heal spontaneously within 10-14 days without scarring. Much less common are *major* aphthous ulcers–larger ulcers–often 1 cm or more in diameter, persists for weeks or months, and heal with scarring. Various factors have been suggested to precipitate outbreaks of RAS in predisposed persons, including oral trauma, cessation of smoking, anxiety or stress, sensitivities to food preservatives, infections and hormonal changes ralated to the menstrual cycle.

## EVALUATION OF ROU

A. History: first attack/recurrent, oral trauma, stress, promiscuous sex, drug history.
B. Clinical examination: Fever, joint pain/swelling, diarrhoea, skin rash (butterfly-rash in face), urethritis, site of ulcer (palate - SLE, under-surface of tongue–probably aphthous ulcer).

## INVESTIGATIONS

1. Complete blood count
2. Measurement of red-cell folate, serum vitamin $B_{12}$, serrum ferritin (especially, when findings suggest nutritional deficiency or a haematologic disorder)
3. Biopsy should be considered, if lasts for > 3 weeks
4. Immunostaining is mandatory if a mucocutaneous disorder is suspected
5. Serologic tests for rheumatologic diseases, e.g. RF, ANA, ds-DNA
6. Cultures or other specific tests for infectious agents (e.g. HSV, HIV, CMV)
7. Evaluation of GI diseases (e.g. studies of malabsorption, intestinal biopsy).

## MANAGEMENT

Aphthous ulcer may affect as much as 20% of the population and often recalcitrant to treatment. Amount of pain is a matter of concern to the patient. Avoid oral trauma (e.g. hard toothbrush or foods such as toast) and acidic foods/drinks. Topical anaesthetics (e.g. 0.15% benzydamine or 5% lidocaine gel) or protective bioadhesives (e.g. carmellose or cyanoacrylate) may be used QDS for 2 weeks. Corticosteroids (1% triamcinolone paste, 2.5 mg hydrocortisone pellets or 0.05% fluocinonide in Orabase) may be used locally. Chlorhexidine gluconate (0.12-2%), triclosan or listerine mouthwash may be of some help; even tetracycline mouthrinse (contents of 100 mg doxycycline capsule dissolved in 10 ml of water) may also benefit patients. Amlexanox paste (5%) is a topical anti-inflammatory agent and early therapy with this drug may reduce size, pain and duration of aphthous ulcer.

Major ulcers may be treated with systemic corticosteroid (30-60 mg prednisolone daily for 7 days, and tapered over the second week) and thalidomide (50-200 mg daily).

Other drugs like levamisole, colchicine, dapsone, azathioprine and pentoxyfylline have also been tried in more refractory cases with variable effects.

## MESSAGE

Always exclude oral carcinoma by repeated systemic examination (e.g. ipsilateral cervical adenopathy in an oral ulcer with induration/leukoplakia may indicate malignancy). Aphthous ulcer is benign per se but ulcers associated with vasculitis (e.g. Behcet's syndrome) are stubborn to treatment.

## POSSIBLE CAUSES OF INFLAMED (RED) PHARYNX ± TONSILS

1. Viral pharyngitis (sore throat, odynophagia, pyrexia, inflammed fauces, cervical nodes +).
2. Acute follicular tonsillitis (sore throat ++, odynophagia, pyrexia, swollen and 'patch tonsil', cervical nodes +).
3. Agranulocytosis (sore throat, H/O intake of offending drugs).
4. Infectious mononucleosis (sore throat ++, petechial haemorrhages at the junction of soft and hard palate, pyrexia, cervical/generalised lymphadenopathy, palpable spleen).
5. Meningitis [meningococcal] (headache, vomiting, nuchal headache, sore throat, inflamed fauces ± purulent patches, neck rigidity).

—— o ——

CHAPTER 61

# Red Urine

## POSSIBILITIES

1. Haematuria (commonest cause)
2. Haemoglobinuria
3. Myoglobinuria
4. Acute intermittent porphyria
5. Drugs and vegetables, and others:
   - Phenolphthalein (in alkaline medium)
   - Rifampicin
   - Phenazopyridine
   - Clofazimine
   - Pigment of malignant melanoma
   - Beet roots and vegetable dyes (e.g. food dye like acanthocyanins)
   - Phenol, thymol, senna.

* Dipstick-positive red urine are: haematuria, haemoglobinuria and myoglobinuria.

## DIAGNOSIS

- Haematuria–often turbid red urine. M/E of urine demonstrates plenty of RBCs (normal urine contains not more than 3 RBCs/HPF of uncentrifuged urine)
- Haemoglobinuria/myoglobinuria–clear red or coca-cola coloured urine. Serum is also turned red or pinkish. Special investigation like absorption spectroscopy confirms the constituents of urine sample as haemoglobin or myoglobin. When urine sediment gives a positive occult blood test, but no RBC are microscopically seen, myoglobinuria or haemoglobinuria is suspected. If the blood from these patients are

kept in a test tube for few minutes, the supernatent serum will be reddish in patients with myoglobinuria but the serum will be clear in patients suffering from haemoglobinuria

- Porphyria–Freshly voided urine may be of normal colour but typical port-wine colour develops on standing because porphobilinogen polymerizes spontaneously to uroporphyrin and porphobilin. The confirmation of these products in urine is done by Watson-Schwartz test
- Drugs and others–Abstinence from intake of drugs will make the urine clear. Few drops of sodium hydroxide make the urine yellow, if it is beet root-induced redness of urine.

* Serratia marcescens produces red urine and is known as 'red diaper syndrome'.

** Collection of urine during menstruation and patients on anticoagulant therapy may falsely record RBC in urine.

## COMMON CAUSES OF HAEMOGLOBINURIA (I.E. INTRAVASCULAR HAEMOLYSIS)

A. Acquired–Incompatible transfusion, malaria (P. falciparum), claustridial infection, snake bite, SLE or lymphoma-induced immune haemolytic anaemia, paroxysmal nocturnal haemoglobinuria, paroxysmal cold haemoglobinuria, methyl dopa or penicillin-induced, septicaemia, or extensive burn.

B. Hereditary–Sickle cell disease, haemolytic crises, $G_6PD$ deficiency, hereditary spherocytosis/elliptocytosis.

* In haemoglobinuria, haemoglobin passes into urine (but no intact RBC is found).

## LABORATORY FEATURES OF HAEMOLYSIS

1. ↑ Serum bilirubin (unconjugated > conjugated)
2. ↑ Urinary urobilinogen
3. ↓ Plasma haptoglobin
4. ↓ Plasma haemopexin
5. ↑ Methaemalbumin in blood (Schumm test)
6. ↑ Urinary haemosiderin
7. Fragmented RBCs in peripheral blood
8. Reticulocytosis
9. Hyperplastic bone marrow.

## HAEMATURIA

- Renal–Acute glomerulonephritis, papillary necrosis
- Ureter–Calculus, neoplasm
- Bladder–Cystitis, papilloma
- Urethra–Urethritis, trauma
- Prostate–Benign/malignant hypertrophy
- Miscellaneous–SLE, SBE, leukaemia
- Drugs–Aspirin, anticoagulants, hexamine.

## HARD DATA IN HAEMATURIA

A. Renal disease–associated with significant proteinuria and RBC casts.
B. Infection–presence of pyuria.
C. Painless or painful–infection, calculus, trauma are associated with pain. Painless haematuria is classically seen in hypernephroma, tuberculosis of the kidney, sometimes in papilloma of urinary bladder, polycystic kidney, SLE, acute glomerulonephritis and acute cortical necrosis.
D. Timing of haematuria:
   - Initial part–lesion in urethra
   - Terminal part–lesion in bladder
   - Uniformly distributed and mixed–usually renal or ureteric lesion but may occur in bladder lesion.
E. Microscopic haematuria–when the urine colour is normal and presence of RBCs are detectable only under light microscopic examination, it is designated as microscopic haematuria. The common associations are:
   - Any cause of haematuria
   - Urinary tract infection (UTI)
   - Subacute bacterial endocarditis (SBE)
   - Drug-induced
   - Calculus in the urinary tract.

## COMMON MEDICAL CAUSES OF HAEMATURIA

1. Acute glomerulonephritis
2. SBE (usually microscopic haematuria)
3. Malignant hypertension
4. Snake bite (viperidae group)
5. Henoch-Schönlein purpura or other coagulation disorders
6. Weil's disease

7. Anticoagulant therapy
8. Renal tuberculosis
9. Papillary necrosis (diabetes mellitus, sickle cell disease, analgesic-induced)
10. SLE (e.g. vasculitis).

## MYOGLOBINURIA

- Severe muscle trauma (crush injury)
- Hyperthermia
- Polymyositis/dermatomyositis
- Carbon monoxide poisoning
- Drugs toxicity, e.g. nicotinic acid or amphetamine
- Acute myocardial infarction
- Hypothyroidism
- Muscle ischaemia
- Rhabdomyolysis (heat stroke, crush injury, severe exercise, seizures, alcohol-induced toxicity, influenza-induced, arterial insufficiency, $\downarrow$ K, $\downarrow$ $PO_4$ or $G_6PD$ $\downarrow$ )
- Phosphorylase deficiency (hereditary myoglobinuria).

## TREATMENT (HAEMATURIA)

1. Reassurance. The patient may require tranquilizer
2. Plenty of fluid to be taken to flush the crystals and clots
3. Treatment of specific aetiology
4. Renal colic may be alleviated by antispasmodic-analgesic
5. Blood transfusion in profound haematuria.

—— o ——

CHAPTER 62

# Rings around Cornea

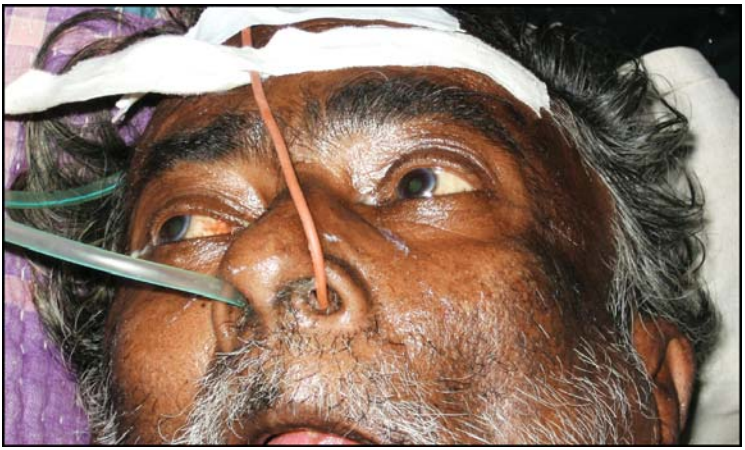


**FIGURE 62.1:** Coma with conjugate deviation to the right in a case of right-sided cerebral haemorrhage: conjugate deviation towards site of lesion and away from the site of paralysis (cataract and arcus corneae are seen)

## DIFFERENTIAL DIAGNOSIS OF RINGS OR PIGMENTATION AT LIMBUS

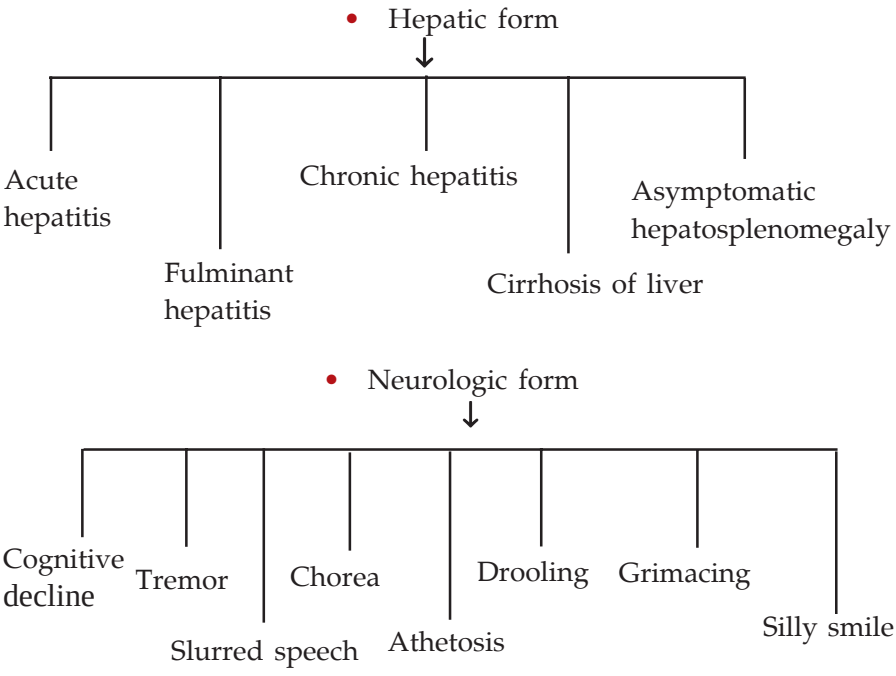
1. Kayser-Fleischer ring (K-F ring)
2. Arcus senilis or arcus juvenilis
3. Band keratopathy
4. Argyrosis (dark-brown discolouration at limbus or of conjunctiva due to prolonged use of silver nitrate preparation as eye drop or as a result of working with silver dust)
5. Cystinosis (deposition of cystine crystals)

* Blood-staining ring in severe hyphaema, and deposition of melanin pigment in high myopia are not uncommon.

## K-F RING

- Golden-brown pigment of copper deposited in the Descemet's membrane layer of cornea in a patient suffering from Wilson's disease
- Seen at limbus; broaded superiorly and inferiorly than laterally and medially; the superior pole is affected first
- May be seen in naked-eye examination but confirmation with slit-lamp examination by an ophthalmologist is a must
- Clinical hallmark of Wilson's disease
- Eyes in Wilson's disease: look for
  - Jaundice
  - K-F ring
  - Sunflower cataract
- Never hampers vision
- K-F like ring is seen in:
  - Cryptogenic cirrhosis
  - Prolonged cholestasis, e.g. primary biliary cirrhosis
  - Chronic biliary cirrhosis

## WILSON'S DISEASE



## DIAGNOSIS

1. Ceruloplasmin in serum (< 20 mg/dl)
2. K-F ring
3. Copper in liver biopsy > 200 µg/g of dry weight of liver
   Confirmation: 1 + 2, or 1 + 3.

## DRUGS USED IN WILSON'S DISEASE

1. D-penicillamine (1 g/day) with pyridoxine supplementation (25 mg/day)
2. Elemental zinc as acetate (50 mg thrice daity)
3. Trientine (1000 mg/day)
4. Potassium iodide (20 mg, QDS)
5. Cobalt chloride
6. Tetrathiomolybdate

* D-penicillamine is the mainstay of treatment.

## ARCUS SENILIS (ARCUS CORNEAE)

- In aged persons (usually > 50 yrs); starts in lower pole
- Greyish-white circular ring, may be partial or complete
- Just within limbus, so a clear area of cornea remains within the arcus and the limbus
- Represents deposition of cholesterol and phospholipid mainly in the substantia propria layer of cornea; search for xanthelasma around the eyes
- Virtually no significance in elderly people (as like gray hair in scalp) but if present < 50 yrs (arcus juvenilis), may be associated with atherosclerosis, systemic hypertension, IHD, diabetes mellitus or stroke.

## BAND KERATOPATHY

- Corneal calcification, usually at the lateral and medial margins of cornea
- Seen in long-standing hypercalcaemia, e.g. hyperparathyroidism.

—— o ——

CHAPTER 63

# Shake Hands with the Patient

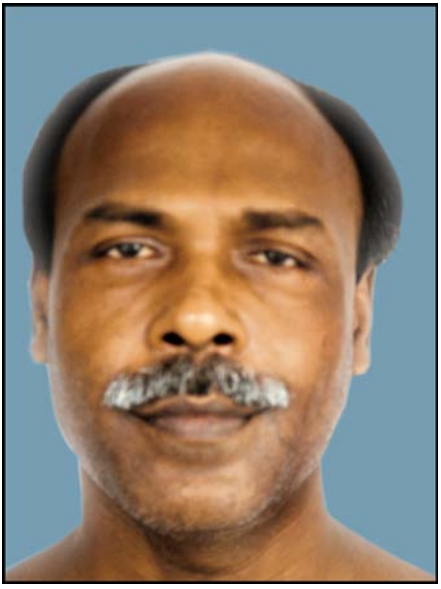


**FIGURE 63.1:** Frontal baldness and bilateral partial ptosis in myotonic dystrophy

## CLUE TO DIAGNOSIS

- The first physical contact with the doctor diagnoses the dominant hand, i.e. the handedness of the patient
- A determined grip or soft, careless handshake often reflects the personality of the patient
- Weak grip-strength: weakness of small muscles of the hand, weakness of long flexor muscles or diseases of joints

**FIGURE 63.2:** Thyrotoxicosis with exophthalmos

- Warm and moist hand: thyrotoxicosis, type II respiratory failure; pyrexia, palmar erythema and Paget's disease may give rise to warm hands
- Cold and moist hand: anxiety neurosis; hypothermia and Raynaud's phenomenon produce only coldness of hands
- Cold and dry hand: myxoedema
- Moist and doghy handshake: acromegaly
- Shake hand producing pain: rheumatoid arthritis
- Rough and thick palm: phrenoderma (vitamin A or essential fatty acid deficiency), tylosis (hyperkeratosis of palm often associated with carcinoma of the oesophagus), arsenic poisoning and psoriasis (palmar)
- Unable to relax the grip: myotonia
- Digital throbbing: may point towards aortic incompetence
- Tremor: specially coarse tremor like flapping tremor
- Warmth, sincerety and personality: shake hand with both the hands

* Face → mirror of the mind
Hand → directed by the mind.

## HANDS IN THYROTOXICOSIS

1. Moist hand
2. Warm palm

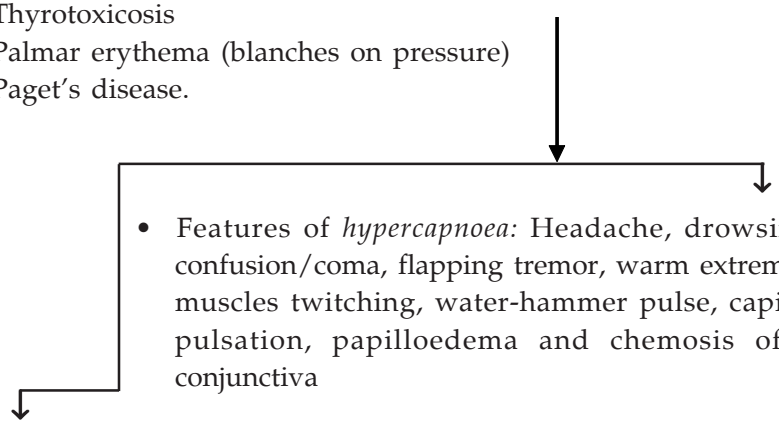
3. Tremor (fine)
4. Tachycardia
5. Palmar erythema
6. Plummer's nail (separation of distal margin of the nail from nail-bed)
7. Clubbing (thyroid acropachy)
8. Hyperpigmentation and vitiligo, rarely
9. Spider naevi, rarely.

## HANDS IN CIRRHOSIS OF LIVER

1. Palmar erythema
2. Leuconychia
3. Clubbing, especially in biliary cirrhosis
4. Jaundice
5. Spider neavi
6. Bleeding manifestations, e.g. petechiae, purpura, ecchymosis
7. Flapping tremor in hepatic pre-coma
8. Dupuytren's contracture in alcoholic cirrhosis
9. Diffuse pigmentation in dorsum.

## WARM PALMS

- Pyrexia
- Type II respiratory failure, especially from COPD
- Thyrotoxicosis
- Palmar erythema (blanches on pressure)
- Paget's disease.




* Commonest cause of respiratory failure is chronic bronchitis.

## FACIES OF MYOTONIA DYSTROPHICA

1. 'Hatchet face' with 'swan-neck' '(hatchet face' and 'swan-neck' are due to atrophy of temporalis and sternomastoid muscles respectively) — i.e. a long facial structure
2. Frontal baldness
3. Cataracts
4. Ptosis with ophthalmoplegia associated with
5. Mental retardation (mild).

—— o ——

CHAPTER 64

# Sneezing, Yawning and Snoring

## SNEEZING

### THE ACT

Deep inspiration → violent and forceful expulsion of air through nose and mouth → give rise to a characteristic sound (sneeze). Sneezing is controlled by a reflex through trigeminal and vagus nerves.

### MECHANISM

Stimuli (dust, dirt, other irritant foreign body)
↓
Irritates the nasal endings of the trigeminal nerve present in nasal mucosa
↓
Sensory impulses are transmitted
↓
Sneezing.

### COMMON CAUSES

A. Local–acute coryza (common cold), nasal allergy (allergic rhintis), hay fever, foreign body in nose, irritant gases like tear gas/chlorine/phosgene/fumes from cooking oil, nasal polyp
B. Systemic–prodromal stage of measles/chickenpox/influenza/whooping cough
C. Miscellaneous–neurosis, hysteria, aspirin/iodide-induced, voluntary induction, change of environmental temperature, withdrawal state in opium addict.

## THE PEARLS

- Essentially a protective reflex similar to cough and a helpful compensatory mechanism to clean the nasal cavity of foreign bodies or irritant gases
- Sneezing may be voluntary or involuntary; voluntary sneeze can be induced with a wisp of cloth/cotton/paper by touching the nasal mucosa or septum to find pleasure out of it
- Change of temperature may induce sneezing in some persons having hyperalgesia of nasal mucosa
- Bouts of sneezing, especially on waking in the morning, may be an indicator of hay fever
- Strong sunlight may stimulate trigeminal nerve endings resulting in sneezing
- In some of the places in india, sneezing is regarded as a social taboo where it is an indicator of a bad sign if the characteristic sound is heard before a journey or initiation of an act
- Antiallergics often gives magic relief
- Sneezing is also known as sternutation.

# YAWNING

## THE ACT

Tonic contraction of facial and pharyngeal muscles → a deep inspiration follows → dilatation of pharynx associated with depression of tongue with mandible → air goes inside lungs → often seen along with stretching of arms as when awaking (pandiculation).

## MECHANISM

Stimuli (boredom, feeling sleepy, seeing other person to yawn)
↓
Centre in brain is probably at basal ganglia
↓
Yawning.

## CAUSES IN CLINICAL PRACTICE

Very little is known about its aetiology. Yawning is basically physiological but few pathological causes may give rise to yawning like,

1. Following attacks of encephalitis
2. Posterior fossa tumour

3. Lower brainstem lesion (yawning, vomiting and hiccough)
4. Seizure disorders (e.g. epilepsy)
5. Opium addict (when effect of opium cases).

### THE PEARLS

- Treatment is not required
- Increases venous return to the heart; closed pulmonary alveoli are also opened up
- Sense of smell is very high during the act of yawning, probably because of the entry of large amount of air within the nasopharynx
- Regarded as a negative 'body language' if happens to occure in an interview table or in the classroom
- Paralysed arm in 'pandiculation' may be stretched when no voluntary movement is possible
- Yawning may be provoked on sight or sound of someone else's yawn. No valid explanation is known for the contagious nature of yawning though it is well known.

## SNORING

### THE ACT

It is a disorder of sleep which is due to vibration of soft tissue above larynx. Three potential areas may be responsible for obstruction, e.g. a. Nose, b. Palate and/or, c. hypopharynx.

### THE PEARLS

- Approximately 40% of middle-aged men and 20% of middle-aged female snore
- Common in obese people
- Snoring have association with obstructive sleep apnoea syndrome. Simple snorers can develop sleep apnoea after ingestion of alcohol
- Habitual snorers have a higher incidene of pulmonary hypertension
- 'Epworth questionnaire' (e.g. how likely are you to doze off or fall asleep ....) differentiate sleep apnoea from simple snoring
- Habitual, non-positional and heroic snorers are easily identified in the family, and are avoided by others during sleeping hours.

### MANAGEMENT

- Thorough ENT check-up

- Weight reduction
- Repair of deviated nasal septum or nasal polyps, if present
- Sleep nasendoscopy to identify the source of vibration
- Surgery–uvulo-palato-pharyngoplasty, softening or shortening of soft palate via laser surgery
- Dental prosthesis at night (to hold the mandible forward) for hypopharyngeal snorers
- Continuous positive airway pressure (CPAP) via a mask in selected cases.

—— o ——

CHAPTER 65

# Splinter Haemorrhage

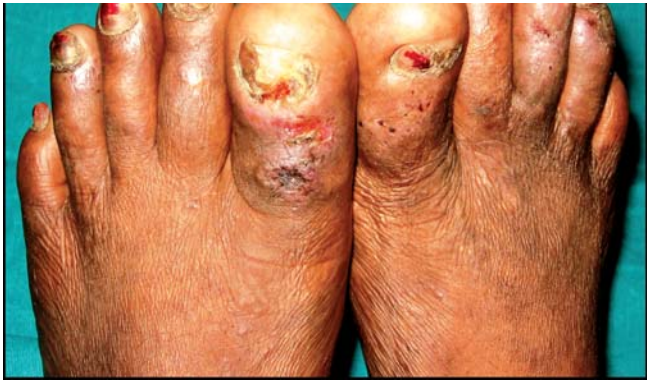


**FIGURE 65.1:** Periungual erythema, nail dystrophy, swelling of the toes and digital ulceration in vasculitis

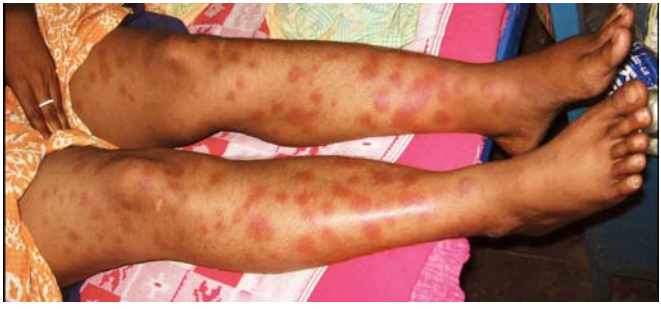


**FIGURE 65.2:** Panniculitis (multiple erythema nodosum)

## POSSIBLE CAUSES

These are linear longitudinal haemorrhages under the nail and are also known as Horder's line. They are found in,

1. Trauma (most common)
2. Acute leukaemias

3. Scurvy
4. Subacute bacterial endocarditis (SBE)
5. Systemic vasculitis
6. Rheumatoid arthritis
7. Psoriasis
8. Trichinosis.

## WHAT IS VASCULITIS ?

It is the heterogeneous group of conditions with necrotising inflammation of the blood vessels. Endothelial oedema and proliferation with haemorrhage contribute to the occlusion of vascular lumen, and subsequent ischaemic changes along with organ damage.

## CLASSIFICATION FOLLOWED GLOBALLY

A. Large-vessel vasculitis: Giant cell arteritis (temporal arteritis) and Takayasu's arteritis (aortic arch syndrome or pulseless disease)
B. Medium-sized vessel vasculitis: Polyarteritis nodosa and Kawasaki disease (mucocutaneous lymph node syndrome)
C. Small vessel vasculitis of arterioles, capillaries and venules: Wegener's granulomatosis, microscopic polyangiitis, Churg-Strauss syndrome, leucocytoclastic vasculitis (hypersensitivity vasculitis), Henöch-Schönlein purpura and essential mixed cryoglobulinaemia.

* This is the most accepted classification (Chapel Hill consensus Conference Classification) though 'overlap' is not uncommon.

** SLE, Behcet's disease, scleroderma, polymyositis or dermatomyositis, SBE and paraneoplastic syndromes are also associated with vasculitis.

## DIFFERENT DERMATOLOGICAL MANIFESTATIONS OF VASCULITIS

A. Large arteries–Digital gangrene
B. Small arteries –
   a. Palpable purpura
   b. Urticarial rashes (tender)
   c. Morbilliform eruptions
   d. Maculopapular eruptions
   e. Subcutaneous nodules
C. Arterioles and very small arteries –
   a. Digital infarction

b. Digital ulceration
c. Nail-fold thrombi
d. Splinter haemorrhage
e. Vesicular/bullous lesion
f. Raynaud's phenomenon
g. Pulp atrophy
h. Necrotic ulcer.

## DRUGS COMMONLY RESPONSIBLE FOR VASCULITIS

- Allopurinol
- Propylthiouracil
- Carbimazole, methimazole
- Penicillins, minocycline, azithromycin
- Montelukast, pranlukast
- Cocaine, morphine
- Hydralazine.

## CUTANEOUS VERSUS SYSTEMIC VASCULITIS

A. Predominantly cutaneous vasculitis –
Usual presentation as palpable purpura, tender urticaria, bullous ulcers or splinter haemorrhages, commonly distributed over legs; buttocks and arms may be affected. The salient features are:
   - Absence of systemic involvement
   - Small vessel leucocytoclastic vasculitis (numerous disrupted polymorphs at the site of damaged vessel)
   - Negative serology
   - Course: variable, may be chronic and recurrent
   - A better prognosis than systemic variety

B. Systemic vasculitis –
   - Organ-specific clinical features (e.g. kidney: hypertension and renal failure; nerves: mononeuritis multiplex; G. I. tract: bowel infarction, etc.)
   - Obviously, the prognosis is poor.

## WHEN DO YOU SUSPECT VASCULITIS IN A PATIENT?

Presence of one or more findings described below should make the clinician suspicious. They are:

- Pyrexia of unknown origin, loss of weight
- CVA or CHD events in the young

- Acute onset of mononeuropathy, e.g. wrist drop or foot drop
- Unexplained retinal vascular changes of acute onset without having diabetes mellitus or hypertension
- Occlusive arterial diseases or systemic hypertension in the young
- Unexplained proteinuria with or without casts
- Palpable purpura, splinter haemorrhage, necrotic ulcers or Raynaud's phenomenon
- Claudication of jaw
- Sudden monocular blindness in elderly with persistent headache
- Unexplained chest X-ray with pulmonary nodular or cavitary lesions.

## VASCULITIS MIMICKERS (PSEUDOVASCULITIS)

Often the pin-point diagnosis is very difficult in situations mentioned below–

- Septicaemia
- Antiphospholipid antibody syndrome
- Bacterial endocarditis
- Thrombotic thrombocytopenic purpura
- Lymphomas
- Ergot overdose/poisoning
- Viral infections, e.g. HBV, HCV, HIV, CMV
- Cholesterol emboli syndrome
- Atrial myxoma
- DIC
- Hypertensive arteriopathy
- Sarcoidosis

* Infections, thrombosis and neoplasia commonly mimic vasculitis.

## DIAGNOSTIC WORK-UP FOR VASCULITIS

1. History and clinical examination with special reference to peripheral pulses, discrepancies in BP, small nodules in the course of an artery, arterial bruit, different dermatological manifestations mentioned above, breath sound with adventitious sounds etc.
2. Laboratory investigations (done to confirm the diagnosis and to assess the extent of organ damage) –
   a. Complete haemogram–majority have normocytic-normochromic anaemia, leucocytosis (eosinophilia in Churg-Strauss syndrome), ↑ ESR, ↑ CRP

b. Urine analysis–commonly having proteinuria, haematuria and red blood cell casts. Large-vessel vasculitis does not usually affect kidney
c. Serum proteins –
   ↑ gamma-globulin of IgG type in majority (in Henoch-Schonlein purpura and Wegener's granulomatosis, the gamma-globulin is IgA type)
   ↑ Ig E level in Churg-Strauss syndrome
   ↓ complement level in cryoglobulinaemia, polyarteritis nodosa
d. Antineutrophil cytoplasmic antibodies (ANCA)
   i. c-ANCA ('c' stands for cytoplasmic)–Antibodies are directed against proteinase-3 (PR-3) and are commonly found in Wegener's granulomatosis.
   ii. p-ANCA ('p' stands for perinuclear)–Antibodies are directed against myeloperoxidase enzyme (MPO). They are commonly found in microscopic polyangiitis, crescentic glomerulonephritis, inflammatory bowel disease, primary sclerosing cholangitis, classic polyarteritis nodosa, autoimmune chronic active hepatitis, primary biliary cirrhosis, Kawasaki disease, etc.
   iii. 'A' or x-ANCA ('A' stands for atypical)–It is a mixed pattern of fluorescence (i.e. cytoplasmic plus perinuclear). It is found in drug-induced vasculitis where the auto-antibodies are directed against lactoferrin and elastase.
e. Antinuclear antibody (ANA) test with anti-dsDNA (SLE) or anti-topoisomerase 1 assay (scleroderma)
f. Selective organ biopsy–It remains the gold standard for diagnosis of vasculitis. Full thickness biopsy should be taken from the involved site to diagnose cutaneous vasculitis. Usually, the histology shows vessel wall inflammation with perivascular involvement with or without leucocytoclasis. Renal biopsy is commonly performed
g. Angiography–It is helpful in diagnosing large and medium-vessel vasculitis. Polyarteritis nodosa may reveal multiple aneurysmal lesions
h. Miscellaneous —
   i. Liver function panel
   ii. Renal function panel
   iii. Chest X-ray

iv. CT and MRI scan of thorax
v. Echocardiography (to rule out left ventricular dysfunction)
vi. Serology for HBV, HCV, HIV, CMV, etc.
vii. Pulmonary function tests which are useful in detecting stenosis of the airways (e.g. Wegener's granulomatosis).

* ANCA-positive vasculitis are: Wegener's granulomatosis, microscopic polyangiitis and Churg-Strauss syndrome.

## SUBEPIDERMAL BLISTERS

- Bullous drug reaction
- Bullous pemphigoid
- Toxic epidermal necrolysis
- Dermatitis herpeteformis
- Epidermolysis bullosa
- Linear IgA disease
- Leucocytoclastic vasculitis
- Burns
- Porphyria cutanea tarda
- Acute graft-versus host reaction
- Pressure necrosis
- Insect bite reaction
- Amyloidosis.

—— o ——

CHAPTER 66

# Spoon-shaped Nails

## KOILONYCHIA (SPOON-SHAPED NAILS)

- Koilos means hollow
- Onych means nail

## METHOD OF EXAMINATION

Keep the patient's fingers at your eye level and look tangentially as done in clinical examination of clubbing. Observe as well as palpate the nails for spooning.

## CLUE TO DIAGNOSIS

1. Iron deficiency anaemia (commonest cause; search for anaemia, glossitis, angular stomatitis, cheilosis; enquire for dysphagia especially in middle aged women → Plummer-Vinson syndrome or Paterson-Kelly syndrome → endoscopic finding of 'post-cricoid web' which is a premalignant condition) → meticulous history taking regarding chronic blood loss like menorrhagia, bleeding piles, melaena, NSAID therapy; dietary history.
2. Idiopathic or familial (examine the other members of the family) → autosomal dominant.
3. Onycholysis (enquire for occupation like washer-woman; a person living in a high altitude may have onycholysis → koilonychia).
4. Overuse of solvent (e.g. nail varnish remover) or detergents for a long period.
5. Very rarely in thyrotoxicosis (look for exophthalmos, tremor and tachycardia).

## STAGES OF KOILONYCHIA

- First stage: 'stage of brittleness' → nails are brittle and rough
- Second stage: 'stage of flattening (platynychia)' → nails are flat and thin, and there is absence of longitudinal ridges
- Third stage: 'stage of spooning' → nails are concave

* There is loss of normal convexity of nails

** The diagnosis of iron deficiency anaemia is incomplete without an indication of its cause.

## INVESTIGATIONS IN IRON DEFICIENCY ANAEMIA

- Blood: Peripheral blood smear examination shows → ↓Hb%, anisocytosis, poikilocytosis, occasionally target cells, microcytic-hypochromic anaemia (central pallor of RBC is considerably increased)
  ↓ MCH, ↓MCHC, ↓MCV
  ↓ Serum iron (normal value: 70-140 µg/dl)
  ↑ TIBC (normal value: 270-335 µg/dl)
- Stool: Occult blood +
  Hookworm ova + (chance finding)
- Per rectal and per vaginal examination: Carcinoma, piles or fibroid
- Upper and lower GI endoscopy: Peptic ulcer, carcinoma of stomach or colon
- Bone marrow examination: Usually not necessary; ↑ erythroblasts in later stages of maturation with deficient Hb formation; staining reveals ↓ of storage iron (haemosiderin)

## PITTING NAILS

1. Psoriasis ('thimble pitting')
2. Eczema
3. Alopecia areata

## ONYCHOLYSIS

1. Candidiasis
2. Ringworm infections
3. Psoriasis
4. Trauma
5. Lichen planus
6. Thyrotoxicosis

### PLATYNYCHIA (FLAT NAIL)

1. Hereditary
2. Iron deficiency anaemia

### BITTEN NAILS

Persons with anxious personality.

### ONYCHODYSTROPHY

1. Fungal infections
2. Repeated trauma
3. Reactive arthritis (Reiter's syndrome)

—— o ——

CHAPTER 67

# Sternal (Bone) Tenderness

## BONES EXAMINED FOR DETECTION OF TENDERNESS

- Sternum
- Vertebrae
- Skull (forehead)
- Shin bone
- Ribs
- Wrists, ankles, pelvic bones

## STERNAL TENDERNESS

Press the upper part of the body of sternum with ball of the right thumb for 2-3 seconds. In the presence of sternal tenderness, the patient winces with pain (makes facial grimace) or complains of pain verbally.

- *Clue to diagnosis in the presence of sternal tenderness*
  1. Acute leukaemias (AML and ALL)
  2. Chronic myeloid leukaemia (CML)
  3. Severe anaemia due to any cause, e.g. thalassaemia, kala-azar
  4. Multiple myeloma
  5. After sternal puncture
     → So, look for haemorrhagic spots, splenomegaly, pallor, benzene seal over sternum.

## MECHANISMS RESPONSIBLE

In leukaemias –

1. Proliferation as well as hypercellularity of marrow
2. Thinning/flattening of inner and outer layer of sternum

## PRE-STERNAL OEDEMA

It is a rare clinical entity and is found in:

- A part and parcel of anasarca
- Mumps
- Caries teeth with root abscess

## TENDERNESS IN VERTEBRAE

- Press each spine of vertebral column one after another and enquire about tenderness
- Look for any kyphosis (smooth bending with convexity backwards): Osteoporosis
  Gibbus (acute angulation in the spine): Caries spine (tuberculosis)
- If multiple vertebrae are involved, think of secondary deposits

## TENDERNESS IN RIBS

- Trauma
- Multiple myeloma
- Cough fracture
- Secondaries with pathological fracture

## TENDERNESS IN SKULL

- Trauma
- Multiple myeloma
- Secondary deposits
- Chronic haemolytic anaemia

## MESSAGE

A definite bony tenderness (without H/O trauma) always indicates some underlying pathology, which needs immediate care.

—— o ——

CHAPTER 68

# Sudden Cardiac Death (SCD)

## DEFINITION

Non-traumatic and unexplained death in a previously well person due to cardiac causes, which occurs within 6 hours of the onset of symptoms.

### *Facts*

1. The major causes (80%) are due to coronary artery diseases (CAD), 10-15% are due to cardiomyopathies and 5% as a result of valvular heart disease.
2. SCD is a direct consequence of cardiac arrest, which is potentially reversible if treated promptly. Cardiac arrest is due to ventricular fibrillation, pulseless ventricular tachycardia, cardiac asystole or electromechanical dissociation.
3. Sudden death can be due to cardiac or non-cardiac causes (see below).
4. Commonest cause implemented is ventricular fibrillation in the setting of CAD.
5. Ventricular fibrillation–a chaotic ventricular rhythm, is almost always the terminal rhythm in a case of SCD. As both organised electrical activity and ventricular depolarisation do not occur, the patients have no pulse or recordable BP.
6. Patients with cardiac arrest require immediate cardiopulmonary resuscitation (CPR).
7. Patients surviving cardiac arrest, an automatic implantable cardioverter-defibrillator (AICD) is often required to prevent further cardiac arrest in future. Antiarrhythmic drugs like amiodarone may be used but are less effective than AICD.

## CAUSES OF SCD

A. Coronary artery disease–AMI, following coronary artery by-pass surgery, congenital anomaly of coronary arteries, coronary arteritis, embolism of coronary artery or chronic IHD
B. Non-coronary artery disease–hypertrophic cardiomyopathy, dilated cardiomyopathy, myocarditis, congenital long QT syndrome, aortic stenosis, mitral valve prolapse, ball valve thrombus obstructing mitral valve, Fallot's tetralogy or transposition of great vessels, VSD/PDA.

## NON-CARDIAC CAUSES OF SUDDEN DEATH

1. Pulmonary thromboembolism
2. Dissection of the aorta
3. Cerebrovascular accidents (e.g. cerebral haemorrhage)
4. Anaphylaxis
5. Acute adrenal failure
6. Malignant hyperpyrexia (during or after anaesthesia)
7. Poisoning (e.g. cyanide)
8. Foreign body in trachea
9. IV injections of a drug in hypersensitive subject.

## FOUR MODES (4C'S) OF DEATH

- Coronary artery disease
- Cerebrovascular accidents
- Casualty
- Carcinoma

—— o ——

CHAPTER 69

# Swollen Legs

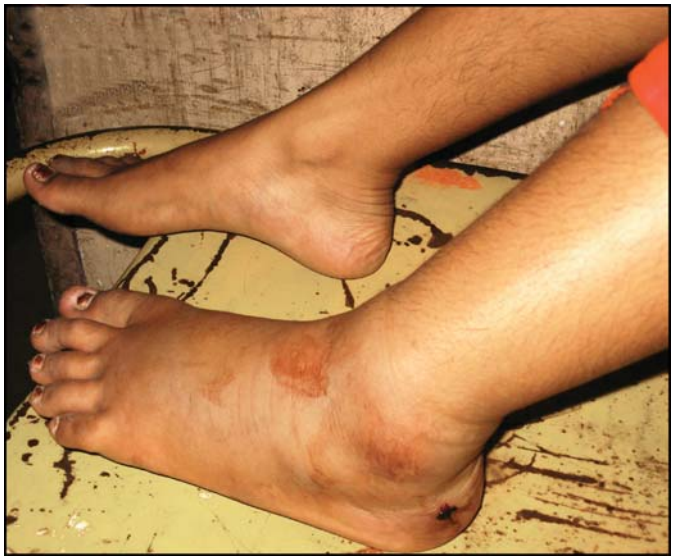


**FIGURE 69.1:** Unilateral pedal oedema as a result of snake bite; bite mark is seen in the back of left foot

## CLUE TO DIAGNOSIS

A. Pedal oedema:

- Congestive cardiac failure or CCF (initially noticed at the end of days work)
- Nutritional oedema (e.g. malnutrition or protein-losing enteropathy) → look for leuconychia and poor muscle mass
- Varicose veins (examine for varicosities in standing position; enquire the occupation, which requires prolonged erect posture)

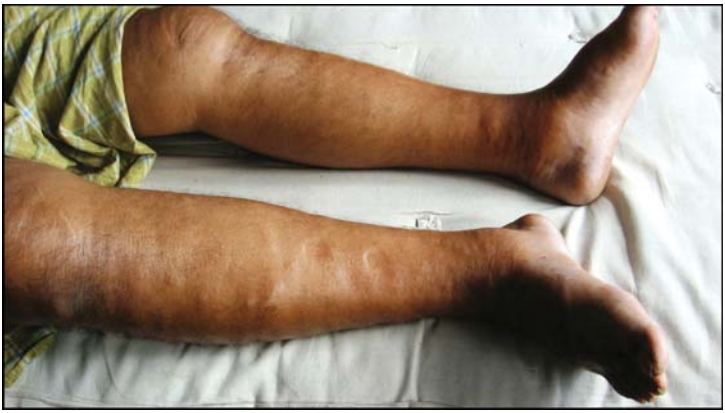
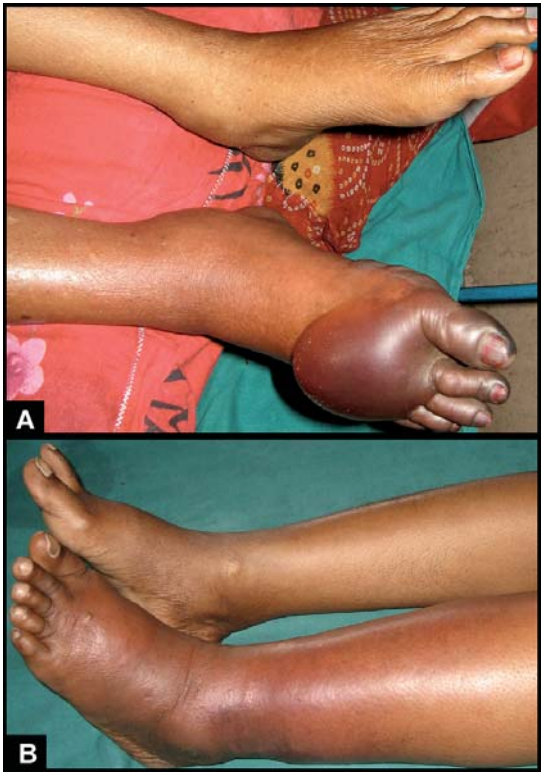


**FIGURE 69.2:** Bilateral pedal pitting oedema in membranous nephropathy (nephrotic syndrome)




**FIGURES 69.3A and B:** Deep vein thrombosis (A – cellulitis, erythema, ecchymosis, blister and gangrene formation in right leg secondary to iliofemoral venous thrombosis; B – erythematous solid oedema in left leg)

- Deep vein thrombosis or DVT (pain + tenderness in thigh or calf, +ve Homan's sign; H/O prolonged immobilisation or in a post-operative patient → look for recent onset varicosities)
- Abdominal mass → tumour, cyst or pregnancy
- Cirrhosis of liver (ascites, palmar erythema, scanty pubic hairs, spider naevi, splenomegaly, superficial abdominal veins)
- Nephrotic syndrome (moon face, white striae, scrotal oedema, peri-orbital puffiness, leuconychia)
- Cyclical oedema (female > male; temporal relationship with menstrual cycles)
- Drug-induced (H/O fludrocortisone, amlodipine/nifedipine, oestrogens, NSAID or corticosteroid ingestion)
- Miscellaneous: Pericardial effusion/constrictive pericarditis (examine the neck veins), irradiation, myxodema, lymphoedema, pregnancy, retroperitoneal fibrosis, inferior vena caval (IVC) obstruction

B. Calf haematoma (H/O trauma, haemophilia)
C. Cellulitis (H/O diabetes mellitus, trauma, infection or any bite) → Fever + pain + tenderness.

## BILATERAL INVOLVEMENT

CCF, nutritional oedema, cirrhosis of liver, nephrotic syndrome, drug-induced, cyclical oedema, IVC obstruction.

## UNILATERAL INVOLVEMENT

DVT, cellulitis, popliteal cyst/gastrocnemius rupture, venous insufficiency, lymphoedema (carcinoma, lymphoma, filariasis, retroperitoneal fibrosis), post-phlebitis syndrome.

## PAINFUL LEGS

1. Deep vein thrombosis (DVT)/thrombophlebitis.
2. Lymphangitis.
3. Cellulitis (i.e. infection).
4. Trauma → fracture.
5. Arthritis of knee, ankle, tarsal joints.
6. Lymphatic obstruction, e.g. malignancy of pelvic organs, filariasis.
7. Vasculitis (i.e. ischaemia).
8. Crops of erythema nodosum.

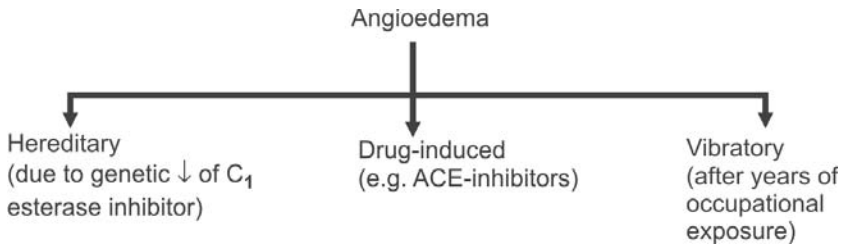
9. Buerger's disease.
10. Phlegmasia alba dolens (swollen, white leg especially in relation to DVT/pregnancy).

## FACIAL PUFFINESS (SWELLING)

1. Familial
2. Myxoedema
3. Cushing's syndrome (primary or secondary to prolonged corticosteroid therapy)
4. Angioneurotic oedema (chiefly the lips and eyelids)
5. Hypoproteinaemia → nephrotic syndrome, acute nephritis, severe anaemia, protein-energy malnutrition)
6. CCF, pericardial effusion, constrictive pericarditis
7. Superior mediastinal syndrome
8. Trichinosis (due to infected pork ingestion)–oedema of the eyelids are common
9. Facial cellulitis
10. Subcutaneous emphysema of face, extending from chest
11. Infection by Marburg and Ebola viruses

* Facial swelling may also be due to anasarca, cellulitis, dental abscess, sinusitis (maxillary), preauricular lymphadenitis or bilateral parotid swelling.

## RECURRENT SWELLING OF FACE AND LIMBS




* Patients are usually non-responsive to treatment in hereditary form but cyproheptadine, danazol, hydroxyzine, epsilon-amino caproic acid or fresh frozen plasma may be beneficial. The patients may complain of recurrent abdominal pain too.

## PRETHROMBOTIC DISORDERS IN RECURRENT DVT (PROCOAGULANT STATES)

- Inherited:
  a. Antithrombin III ↓
  b. Protein C or S ↓
  c. Homocystinuria
  d. Dysfibrinogenaemia
  e. ↑ release of plasminogen activator inhibitor (PAI-1)
  f. Defective release of plasminogen activator
  g. Activated protein C resistance
  h. Factor V Leiden
  i. Prothrombin mutations
- Acquired:
  a. Metastatic tumour
  b. Extensive trauma/major surgery
  c. Myeloproliferative disorders
  d. Chronic congestive cardiac failure
  e. Behcet's syndrome
  f. OC pills or L-asparaginase-induced
  g. Hyperviscosity states
  h. Antiphospholipid syndrome
  i. Pregnancy and puerperium

## DEEP VEIN THROMBOSIS

The patient presents with pain or tight feeling in the calf with swelling, redness and non-pitting oedema of ankle or of legs. The local part feels warm, and Homan's sign (induction of calf pain on dorsiflexion of foot) is often present → non-specific sign, and may be positive in all inflammatory lesions in the calf.

Predisposing factors (procoagulant states) are described above; moreover, post-AMI, CCF, varicose veins, prostatectomy patients, and CVA patients are prone to develop DVT. Obesity, long air travel or prolonged immobility (bed rest > 4 days) are important provocating factors.

*Phlegmasia cerulae dolens:* Deoxygenated haemoglobin in stagnant veins gives a cyanotic hue, and

*Phlegmasia alba dolens:* Markedly oedematons leg in DVT leads to ↑ in interstitial pressure exceeding capillary perfusion presure → gives rise to pallor (i.e. alba or whiteness) of the limb.

Clinical diagnosis of DVT is often unreliable; D-dimer level assay ↑ the sensitivity of diagnosis. B-mode venous compression, USG or Doppler ultrasound, impedence plethysmography and venography are diagnostic. The principal aim of diagnosis and treatment is to prevent pulmonary thromboembolism.

Treatment is done by extremity elevation and bed rest. An elastic stocking may be used in the limb. Anticoagulation is started with low-molecular-weight heparin (i.e. LMWH) with a goal PTT twice normal; warfarin is started immediately and LMWH is stopped when the INR is in target range (i.e. 2.5); LMWH do not require monitoring of coagulation factors and there is less chance of bleeding when compared to unfractioned heparin. NSAID may be used for pain and swelling of the limb.

## BEDSIDE FEATURES OF INCREASED EXTRACELLULAR VOLUME

Peripheral oedema is produced by expansion of extracellular volume by at least 15% (i.e. 2 litre). The features are:

- Oedema (face in the morning; sacral oedema in bed-ridden patient)
- Pulmonary oedema
- Pleural effusion
- Pericardial effusion
- Ascites
- Raised JVP, gallop rhythm, cardiomegaly, basal crepitations and systemic hypertension (in few cases) are features of expansion of the blood volume.

—— o ——

CHAPTER 70

# Tongue: a Clue to Many Diseases

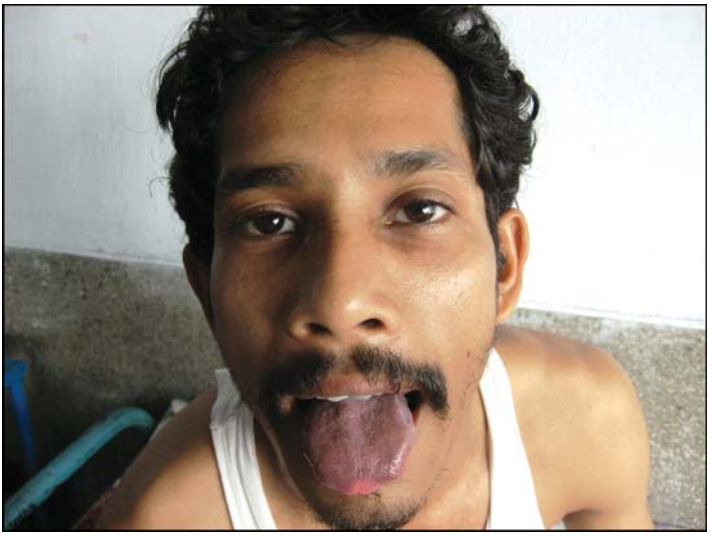


**FIGURE 70.1:** Bluish-black tongue in methaemoglobinaemia

## INTRODUCTION

Tongue is composed on three elements: epithelium, muscles and glands.

Epithelium → stratified and non-cornified; two special structures are seen over the epithelium, i.e. the papillae and the taste bud. There are four types of papillae (minute projections over mucous membrane), e.g. filiform (major papillae in tongue), conical, fungiform (flat-rounded head) and circumvallate papillae (present at the back of the tongue).

Muscles → voluntary and cross-striated.

Glands → small and scattered throughout the tongue. They are mucous glands, serous glands, and lymph nodes (glands).

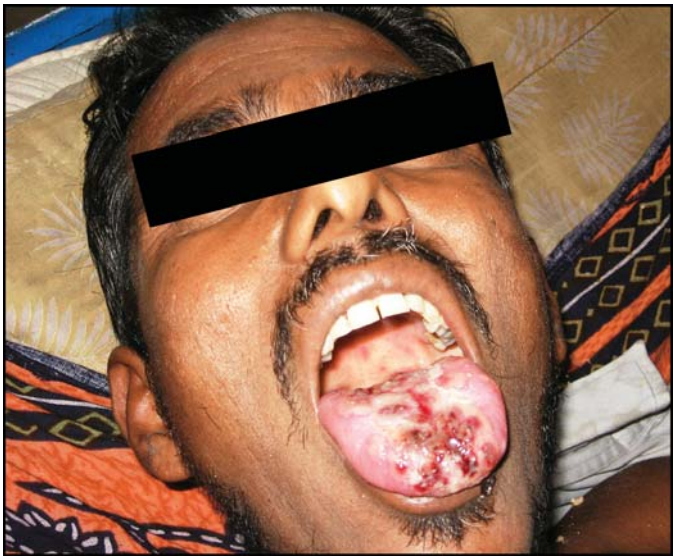


**FIGURE 70.2:** Glossitis with superadded fungal infection and haemorrhage – a case in convalescence from viperidae snake bite

Discolouration of the tongue are commonly associated with infections, deficiency disorders and metabolic diseases, and are important diagnostic clue in clinical medicine.

## COLOUR AND MORPHOLOGICAL CHANGES

1. Moist tongue–Sialorrhoea, heavy metal poisoning.
2. Dry tongue (also brown and a little furred)–Dehydration, mouth breathing, xerostomia, atropinisation.
3. Pale tongue–Anaemia (severe).
4. Macroglossia and microglossia–Read the section on 'Macroglossia'.
5. Yellow tongue–Intake of yellow coloured food/sweets, jaundice (the undersurface).
6. Blue tongue–Central cyanosis, meth- or sulphaemoglobinaemia, intake of blue coloured food/sweets.
7. Bluish-red tongue–Polycythaemia.
8. Black tongue–Addison's disease; after ingestion of liquorice, bismuth or charcoal.
9. 'Black hairy' tongue–Developed as a result of failure of keratin layer of the filiform papillae to desquamate normally. It is found in brown staining by tobacco, food, chromogenic organism, fungal infections, and after use of penicillins/tetracyclines/antiseptic mouthwashes.

10. Magenta-coloured tongue (a bit pinkish)–Riboflavin deficiency.
11. 'Furred' tongue–Heavy smokers, chronic debilitating disorders, acute tonsillitis, sore throat (i.e. painful conditions in mouth).
12. 'Geographic' tongue–Denuded red patches 'wandering' or migrating across the surface of the tongue due to rapid loss and regrowth of filiform papillae. The colour and features change from day to day creating a 'wandering rash', which is an asymptomatic inflammatory condition. Though looks odd, it has no clinical significance and can be regarded as a variant of normal; may be familial. Though painless, the patient should be reassured.
13. 'Strawberry' and 'raspberry' tongue–Hypertrophy of fungiform papillae with changes in filiform papillae, and are seen in scarlet fever.
14. Blotting paper like pallor (with black pigmentation in the margins)–Often seen in hookworm infestation.
15. 'Bald tongue'–There is total loss or atrophy of papillae (i.e. pale and smooth tongue) and is classically seen in iron deficiency anaemia, pernicious anaemia, folic acid deficiency, tropical sprue and syphilis.
16. 'Raw-beefy tongue'–The tongue is red, swollen and painful; classically found in pellagra and vitamin $B_{12}$ deficiency. This condition is associated with mucosal atrophy in the mouth and angular stomatitis.
17. 'Angry-looking tongue'–Central coating with red tip and margins; common in enteric fever (first week).
18. 'Scrotal' tongue–Deep horizontal fissures in the tongue where debris may collect; it is of no clinical significance.
19. White patches on the tongue–Due to leukoplakia (a pre-cancerous lesion), thrush (i.e. moniliasis due to immumodeficiency, diabetes mellitus, AIDS, debilitated patients, immumosuppressive/corticosteroid treatment), other fungal infections, chronic superficial glossitis, hairy leuloplakia (raised, painless, corrugated, poorly demarcated lesion mainly at the margins, which does not rub off, and is found in AIDS). The lesion of thrush looks creamy-white curd-like patches which reveals a raw bleeding area when scraped.
20. Median rhomboid glossitis–It is a lozenge-shaped denuded area in the middle of the tongue posteriorly, itself a congenital abnormality. It should be differentiated from carcinoma of the tongue as it feels nodular.

21. Bite mark in the tongue with haematoma around–Accidental bite during eating or convulsions.
22. 'flaccid tongue' with rounded tip and the tongue rests on the floor of the mouth like a mushroom (grossly wasted), often with fasciculations–Seen in bulbar palsy.
23. 'Spastic tongue' with pointed tip (without any fasciculation)–Seen in pseudobulbar palsy.
24. Growth (cauliflower-like) in the tongue is observed in squamous cell carcinoma (with halitosis).
25. Mushroom like tongue–Sore tongue with white slough and is seen in corrosive (e.g. acid or alkali) poisoning.
26. Horny tongue (crocodile tongue)–Tongue with cornification of mucosa and of no clinical value.
27. Dry, red tongue with atrophy of the papillae and fissures–Seen in Sjögren's syndrome.
28. Ulcer in the tongue–Aphthous ulcer (very painful, anywhere in tongue), frenal ulcer (in frenum), tuberculous ulcer (in dorsum), marginal ulcer (anywhere in tongue), snail-track ulcer (in dorsum and is seen in secondary syphilis), Behcet's disease (painless), ulcers at tongue margins (by ill-fitted denture) may be present.
29. Shaggy, papillomatous dorsum due to hypertrophy of filiform papillae with blackish pigmentation–Found in Addison's disease and acanthosis nigricans.
30. Miscellaneous–Lichen planus (glossy or glazed tongue), erythema multiforme (ulcers or blisters), pemphigus, pemphigoid, hereditary haemorrhagic telangiectasis (telangiectasia on the tongue) and Crohn's disease (raised, smooth, red nodules with hyperplastic ridges) may affect the tongue.

* Tongue may be called as mirror of systemic dysfunction of human body.

—— o ——

CHAPTER 71

# Weight Gain/Loss

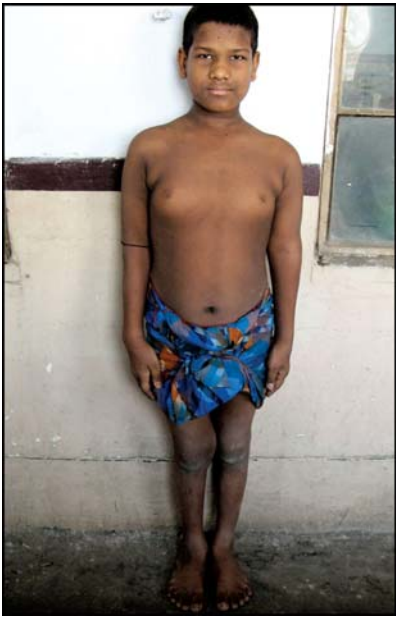
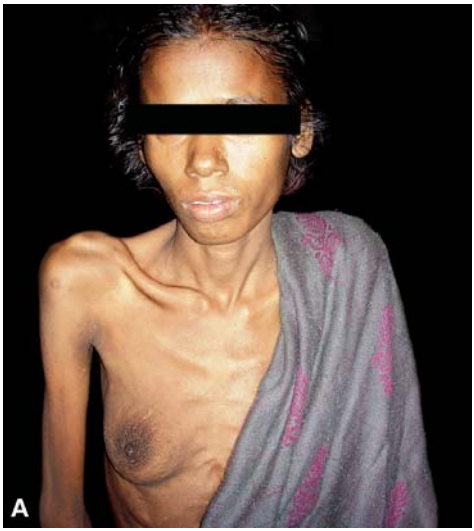


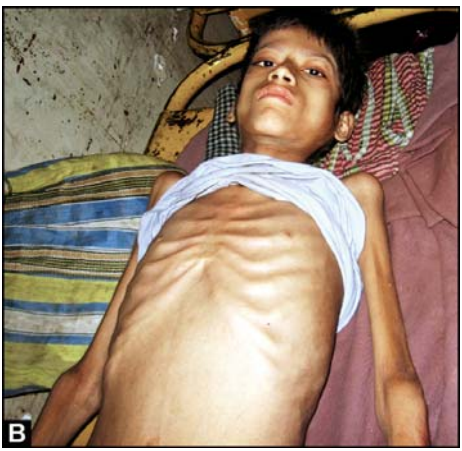
**FIGURE 71.1:** A 9-year-old boy with gigantism (hypersecretion of growth hormone before puberty) and gynaecomastia

## WEIGHT GAIN

Acceptable range of BMI is 18.5 -24.9 kg/m$^2$. When BMI $\geq$ 25 kg/m$^2$, it is defined as overweight and when $\geq$ 30 kg/m$^2$, it is designated as obesity. Abdominal or truncal obesity is measured by 'waist-hip ratio', which when > 1.0 in males and > 0.9 in females increase the morbidity and mortality risks. Weight gain is commonly due to:

1. Sedentary life style.




**FIGURES 71.2A and B:** Emaciation (all skin and bone) as a result of pulmonary tuberculosis (A), and type 1 diabetes mellitus (B)

2. Fluid overload (CCF, cirrhosis, nephrotic syndrome, hypoproteinaemia, pregnancy).
3. Obesity (Cushing's syndrome, hypothyroidism, Laurence-Moon-Biedl syndrome, Prader-Willi syndrome, hypothalamic disorders, e.g. Froehlich's syndrome).
4. Prolonged therapy with corticosteroids or oestrogen (e.g. oral contraceptive pills).
5. Anxiety disorder with compulsive eating.
6. Miscellaneous–Insulinoma, hypogonadism, acromegaly, discontinuation of smoking, depression, craniopharyngioma.

Gain in weight is commonly associated with insulin resistance, diabetes mellitus, hypertension, osteoarthritis, atherosclerosis, dyslipidaemia, cholelithiasis and sleep-apnoea syndrome.

## CLINICAL DIAGNOSIS OF OBESITY

- Body weight > 20% more than the upper limit of standard weight in relation to age and sex of the individual
- BMI $\geq$ 30 kg/m$^2$
- Skin thickness > 1 inch in inferior angle of scapula (male) or mid-triceps region (females), when measured by a special calliper (e.g. Harpenden or Schofield's calliper)
- Obesity can be quantified by anthropometry (i.e. skinfold thickness), densitometry (under water weight), CT or MRI scan (measures mesenteric fat).

## APPETITE IN INTERNAL MEDICINE

- Increase in appetite (polyphagia or hyperorexia):
  - Diabetes, thyrotoxicosis, growth hormone excess (gigantism/acromegaly)
  - Malabsorption syndrome (e.g. sprue)
  - Binge eating, bulimia nervosa
  - Worm infestations
- Diminished appetite –
  - Depression, anorexia nervosa, boredom, emotional upset
  - Disseminated malignancy, tuberculosis, acute febrile illness
  - Hepatitis (acute viral), cholecystitis.

## DRUGS USED IN TREATMENT OF OBESITY

Phentermine, fenfluramine, sibutramine, orlistat (inhibitor of intestinal lipase), fluoxetine or sertraline (appetite suppressant), rimonabant (selective cannabinoid 1 receptor blocker), bulk preparation (e.g. guar gum), recombinant leptin etc.

## WEIGHT LOSS

'Generalised undernutrition' i.e. loss of total body fat and diminution of muscle bulk is known as emaciation. Undernutrition with signs of vitamin, mineral and essential amino acid deficiency is designated as malnutrition. Cachexia is a profound state of general ill-health and a

combined manifestation of anorexia, anaemia plus emaciation. Decrease in muscle mass is known as 'sarcopenia'. Weight loss is commonly due to:

1. Chronic malnutrition (protein-energy malnutrition)
2. Tuberculosis
3. Diabetes mellitus
4. Thyrotoxicosis
5. Disseminated malignancy
6. AIDS (slim disease)
7. Anorexia nervosa
8. Malabsorption syndrome
9. Addison's disease, panhypopituitarism, pheochromocytoma, hyperparathyroidism
10. Collagen vascular diseases, e.g. SLE
11. Motor neurone disease
12. Food faddism
13. Congestive cardiac failure (cardiac cachexia)
14. Drug-induced, e.g. metformin.

## RELATION OF WEIGHT LOSS WITH APPETITE

Weight loss + ↑ or normal appetite

- Diabetes mellitus (uncontrolled)
- Thyrotoxicosis
- Chronic kala-azar
- Malabsorption syndrome
- Pheochromocytoma

Weight loss + ↓ appetite

- Malignancy (advanced)
- Tuberculosis (disseminated)
- Depression
- Addison's disease
- Anorexia nervosa

## SIGNIFICANT WEIGHT LOSS

When there is unexplained and unintentional weight loss of > 3 kg in the previous 6 months, it is known as 'significant' weight loss. It is very often associated with vitamin and nutrient deficiencies, higher surgical mortality rates, and proneness to acquire infections. Unintentional weight loss is associated with ↑ morbidity and mortality.

## SCREENING TESTS FOR EVALUATION OF WEIGHT LOSS

A loss of 5% of body weight in the preceding 6-12 months should prompt further evaluation.

- Complete blood count
- Liver and renal function tests
- Urine analysis
- Chest X-ray
- Glucose, electrotytes, calcium, TSH
- Cancer screening
- Additional tests:
  - HIV
  - Upper and lower GI endoscopy
  - Abdominal CT/MRI
  - HRCT thorax

Remember: As 7000 kcal is the equivalent of 1 kg of human adipose tissue, a calorie deficit of 1000 kcal/day will produce a loss of weight approximately 1 kg/week. A variety of cytokines, e.g. TNF-α, IL-6, IL-1 and IFN-8 can induce cachexia.

—— o ——

CHAPTER 72

# Wheeze/Stridor

## WHEEZE OR WHEEZING: CHARACTERISTICS

1. High-pitched musical sound heard from a distance.
2. Better heard in expiratory phase.
3. Indicates small airways obstruction.
4. Usually associated with rhonchi in the chest.

### *Wheezing is found in*

1. Bronchial asthma (reversible wheeze)
2. COPD (irreversible wheeze)
3. Interstitial lung disease
4. Tropical eosinophilia
5. Cardiac asthma
6. Infections: pneumonia, bronchitis, bronchiolitis, epiglottitis
7. Endobronchial disease (e.g. neoplasm)
8. Airways obstruction (goitre, aneurysm, spasm, oedema or haemorrhage)
9. Carcinoid syndrome
10. GERD (gastroesophageal reflux disease) with aspiration.

## STRIDOR: CHARACTERISTICS

1. Low-pitched crowing sound (loud) heard from a distance
2. Better heard in inspiratory phase
3. Indicates larger airways obstruction like larynx, trachea and major bronchus
4. Very common in children

5. Laryngeal stridor is a medical emergency and urgent respiratory support may be required.

### *Stridor is found in:*

1. Foreign body in larynx or trachea
2. Laryngeal oedema (anaphylaxis)
3. Diphtheria, whooping cough
4. Allergic (spasmodic) croup, i.e. acute laryngotracheobronchitis
5. Acute epiglottitis
6. Laryngeal obstruction (laryngomalacia, laryngeal webs, bilateral abductor paralysis of the vocal cord, foreign body)
7. Mediastinal mass
8. Vocal cord palsy due to recurrent laryngeal nerve paralysis (other features of recurrent laryngeal nerve palsy are bovine cough and hoarseness of voice)
9. Miscellaneous: hysterical, Cri-du-chat syndrome, Pierre Robin's syndrome, subglottic tracheal stenosis, tracheo-oesophageal fistula.

## CARDINAL SYMPTOMS OF RESPIRATORY SYSTEM

- Cough
- Expectoration
- Haemoptysis
- Chest pain
- Breathlessness
- Wheeze or stridor.

—— o ——

CHAPTER 73

# White Nails

## SYNONYM

Leuconychia, Terry nail

## IMPRESSION

Hypoalbuminaemia

## CONDITIONS ASSOCIATED

- Cirrhosis of liver
- Nephrotic syndrome
- Severe malnutrition

## BLUE LUNULA (AZURE ARCS)

- $CuSO_4$ poisoning
- Wilson's disease
- After zidovudine therapy

## RED LUNULA

Congestive cardiac failure

## BROWN-BLACK NAIL

Haematoma underneath the nail, melanoma

## HALF-AND-HALF NAIL (PROXIMAL HALF IS WHITE WHILE THE DISTAL HALF IS PINK)

Chronic renal failure

### MEES LINE (WHITE LINE OF 1-2 MM WIDTH APPEARS ABOVE LUNULA)

Arsenic poisoning.

### BEAU'S LINE (TRANSVERSE RIDGES ON NAILS)

- Recovery from any febrile illness
- Zinc deficiency

### HORDER'S LINE (SPLINTER HAEMORRHAGE)

- Trauma (most common cause; ask for occupation)
- SBE
- Trichinosis
- Leukaemias (acute)
- Vasculitis
- Psoriasis
- Scurvy

### GREEN OR BLACK-GREEN NAIL

Pseudomonas infection.

### YELLOW NAIL

Fungal infection, psoriasis.

### BLACKISH, YELLOW-GREEN NAIL

Yellow nail syndrome (lymphoedema of extremitics + sinusitis + pleural effusion).

### ONYCHOLYSIS (SEPARATION OF 'TERMINAL' NAIL PLATE FROM UNDERLYING NAIL-BED)

Candidiasis, ringworm infection, psoriasis, trauma, lichen planus, chronic paronychia.

### ONYCHOMEDESIS (SEPARATION OF 'PROXIMAL' NAIL PLATE FROM UNDERLYING NAIL-BED)

- Trauma with haematoma underneath the nail
- Any period of severe illness

### ONYCHORHEXIS

In older women, the nails may break easily and separate into 'horizontal' strata (probably from repeated hydration and drying during cooking + mechanical + chemical trauma).

### 'LONGITUDINAL' RIDGES IN NAIL

- Lichen planus
- Alopecia areata
- Darier's disease

### MUEHRCK'E LINES

Paired white transverse bands due to hypoalbuminaemia.

### BLACK NAIL

Hair dyes, Peutz-Jeghers syndrome.

### BROWN NAIL

Hydroquinone-induced.

### ABSENT OR ATROPHY OF NAIL

Nail-patella syndrome, congenital ectodermal defect.

—— o ——

CHAPTER 74

# White (Milky) Urine

## POSSIBILITIES

1. Chyluria → as a result of passage of chylomicrons in urine.
2. Phosphaturia → passing out of large amount of phosphates (amorphous or crystalline) in urine.
3. Lipiduria → presence of fat droplets in urine.
4. Pseudochyluria (milky or gold paint) → passage of large amount of protein, desquamated cells may turn the urine milky (there is mixed cellularity with cholesterol excess).
5. Pyruria → Urine is full of pus cells in urinary tract infection.

## AETIOLOGY OF CHYLURIA

A. Blockage of lymph channels or thoracic duct by –
   a. Parasites: filariasis, ascariasis, cysticercosis, taenia and H. nana infection.
   b. Non-parasitic: trauma to thoracic duct, tumours, obstruction of thoracic duct, pregnancy, tuberculosis, retroperitoneal fibrosis, congenital malformation of lymph channels or lymphangiectasia.

B. Idiopathic

### *Clue to diagnosis*

Add *acedic acid* to urine sample

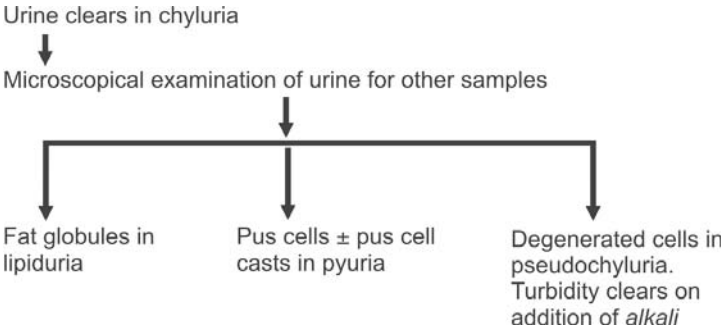
↓

Urine clears in phosphaturia

↓

Add *ether* to other samples

↓




## SPECIAL POINTS TO NOTE

1. Chyluria–Commonest cause is filariasis (e.g. *W. bancrofti*-induced), especially in India. A state of lymphatic hypertension with cavernous malformations of lymph channels at hilum of pelvis prevails which ultimately ruptures into the urinary passage (may be in the bladder or urethra too). The colour of the urine is milky or creamy and the fat globules may be demonstrated microscopically by staining with Sudan III. Reassurance and treatment of the cause are the mainstay of management.
2. Phosphaturia–Urine clears on addition of acetic acid. Oral alluminium hydroxide (antacid) may alleviate the problem.
3. Lipiduria–Fat droplets usually float on the top of the urine or may be separated as a layer on top after ultracentrifugation. Conditions associated with lipiduria are diabetes mellitus, nephrotic syndrome, eclampsia, arsenic poisoning.
4. Pseudochyluria–Usually turbid urine as a result of large number of degenerated cells or tumour cells. Turbidity clears on addition of alkali.
5. Pyuria–Large number of pus cells make the urine turbid and is commonly associated with pyelonephritis, cystitis or urethritis. Broad-spectrum antibiotics cure the ailment.

—— o ——

CHAPTER 75

# Yellowish Palms/ Soles

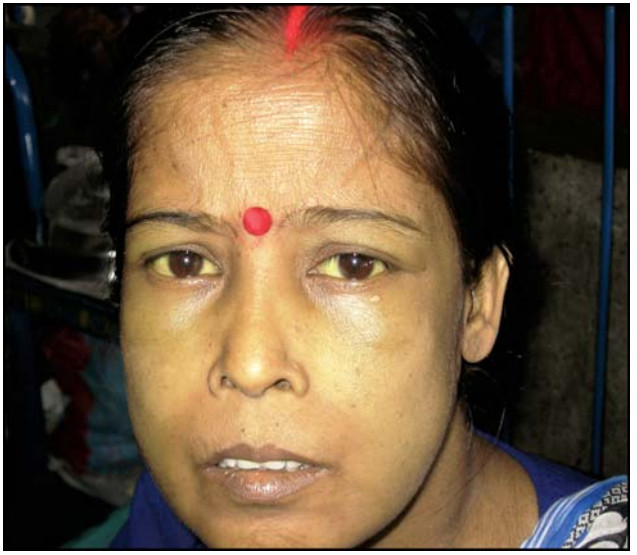


**FIGURE 75.1:** Obstructive jaundice evidenced by deep yellow conjunctiva, greenish-yellow hue in skin and xanthelasma (underneath left eye)

## DIFFERENTIAL DIAGNOSIS

- Jaundice (sclera + mucous membrane + skin) → yellow
- Carotenaemia (only the skin, mainly the palms and soles take a lemon-yellow tint)
- Diffuse xanthomatosis (skin takes a yellowish-orange colour; may have tuberous or palmar xanthoma, xanthelasma → lipid disorder)
- Quinacrine therapy (antimalarial, anti-giardia lamblia)
- Using turmeric for coocking/cosmetic purpose/taboo.

* Carotene, a lipid-soluble yellow pigment in the plasma, does not stain the conjunctiva and mucous membrane. However, the serum looks yellow in carotenaemia.

## CLUE TO DIAGNOSIS OF CAROTENAEMIA

1. Eating large quantities of carotene (vitamin A), carrots, mango, papaya, squash and other coloured fruits/vegetables.
2. Hypothyroidism → due to impaired metabolism of β-carotene in the liver.
3. Diabetes mellitus.
4. Receiving β-carotene therapy for erythropoietic porphyria.
5. Simmond's disease (panhypopituitarism).
6. Anorexia nervosa (possible defect in conversion of carotene to vitamin A).
7. Castrated male.

## IS CAROTENAEMIA INJURIOUS FOR HEALTH?

Except the cosmetic effect, excess carotene is non-injurious to health. Carotenaemia does not develop into hypervitaminosis A.

## MESSAGE

Sclera never turns yellow in carotenaemia. Hypothyroid patients are mostly susceptible to develop carotenaemia.

## XANTHOMA

1. Planous → classical example is 'xanthelasma' around the eyes (the upper eyelids more common than the lower). They are characteristically soft, yellowish and slightly raised from the surface, and are also seen in palmar creases, underneath the breast, chest or back. Xanthelasma is classically seen in:
   - Familial hypercholesterolaemia
   - Familial dysbetalipoproteinaemia
   - Diabetes mellitus
   - Myxoedema
   - Prolonged cholestasis, e.g. primary biliary cirrhosis
   - Nephrotic syndrome
2. Tuberous → commonly seen in extensor surface of elbow, knee, wrist, ankle and 'buttock'.

## TRIAD OF FAMILIAL HYPERCHOLESTEROLAEMIA

- Tendon xanthoma
- Xanthelasma
- Arcus corneae

## MESSAGE

Alway check the serum lipids, sugar level and thyroid hormones to come an aetiological diagnosis. Remember, few middle-aged or elderly people may have no lipid disorder with xanthelasma.

—— o ——

CHAPTER 73

# White Nails

## SYNONYM

Leuconychia, Terry nail

## IMPRESSION

Hypoalbuminaemia

## CONDITIONS ASSOCIATED

- Cirrhosis of liver
- Nephrotic syndrome
- Severe malnutrition

## BLUE LUNULA (AZURE ARCS)

- $CuSO_4$ poisoning
- Wilson's disease
- After zidovudine therapy

## RED LUNULA

Congestive cardiac failure

## BROWN-BLACK NAIL

Haematoma underneath the nail, melanoma

## HALF-AND-HALF NAIL (PROXIMAL HALF IS WHITE WHILE THE DISTAL HALF IS PINK)

Chronic renal failure

### MEES LINE (WHITE LINE OF 1-2 MM WIDTH APPEARS ABOVE LUNULA)

Arsenic poisoning.

### BEAU'S LINE (TRANSVERSE RIDGES ON NAILS)

- Recovery from any febrile illness
- Zinc deficiency

### HORDER'S LINE (SPLINTER HAEMORRHAGE)

- Trauma (most common cause; ask for occupation)
- SBE
- Trichinosis
- Leukaemias (acute)
- Vasculitis
- Psoriasis
- Scurvy

### GREEN OR BLACK-GREEN NAIL

Pseudomonas infection.

### YELLOW NAIL

Fungal infection, psoriasis.

### BLACKISH, YELLOW-GREEN NAIL

Yellow nail syndrome (lymphoedema of extremitics + sinusitis + pleural effusion).

### ONYCHOLYSIS (SEPARATION OF 'TERMINAL' NAIL PLATE FROM UNDERLYING NAIL-BED)

Candidiasis, ringworm infection, psoriasis, trauma, lichen planus, chronic paronychia.

### ONYCHOMEDESIS (SEPARATION OF 'PROXIMAL' NAIL PLATE FROM UNDERLYING NAIL-BED)

- Trauma with haematoma underneath the nail
- Any period of severe illness

### ONYCHORHEXIS

In older women, the nails may break easily and separate into 'horizontal' strata (probably from repeated hydration and drying during cooking + mechanical + chemical trauma).

### 'LONGITUDINAL' RIDGES IN NAIL

- Lichen planus
- Alopecia areata
- Darier's disease

### MUEHRCK'E LINES

Paired white transverse bands due to hypoalbuminaemia.

### BLACK NAIL

Hair dyes, Peutz-Jeghers syndrome.

### BROWN NAIL

Hydroquinone-induced.

### ABSENT OR ATROPHY OF NAIL

Nail-patella syndrome, congenital ectodermal defect.

—— o ——

CHAPTER 74

# White (Milky) Urine

## POSSIBILITIES

1. Chyluria → as a result of passage of chylomicrons in urine.
2. Phosphaturia → passing out of large amount of phosphates (amorphous or crystalline) in urine.
3. Lipiduria → presence of fat droplets in urine.
4. Pseudochyluria (milky or gold paint) → passage of large amount of protein, desquamated cells may turn the urine milky (there is mixed cellularity with cholesterol excess).
5. Pyruria → Urine is full of pus cells in urinary tract infection.

## AETIOLOGY OF CHYLURIA

A. Blockage of lymph channels or thoracic duct by –
   a. Parasites: filariasis, ascariasis, cysticercosis, taenia and H. nana infection.
   b. Non-parasitic: trauma to thoracic duct, tumours, obstruction of thoracic duct, pregnancy, tuberculosis, retroperitoneal fibrosis, congenital malformation of lymph channels or lymphangiectasia.

B. Idiopathic

### *Clue to diagnosis*

Add *acedic acid* to urine sample

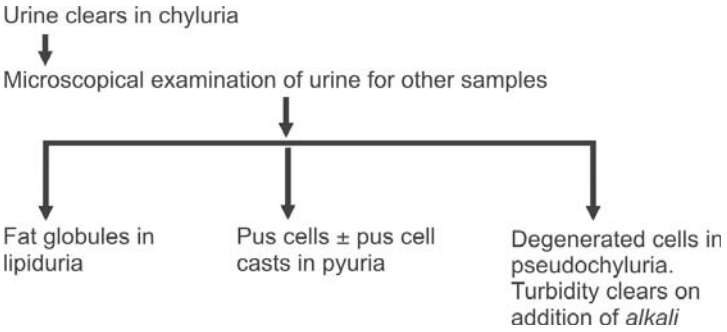
↓

Urine clears in phosphaturia

↓

Add *ether* to other samples

↓




## SPECIAL POINTS TO NOTE

1. Chyluria–Commonest cause is filariasis (e.g. *W. bancrofti*-induced), especially in India. A state of lymphatic hypertension with cavernous malformations of lymph channels at hilum of pelvis prevails which ultimately ruptures into the urinary passage (may be in the bladder or urethra too). The colour of the urine is milky or creamy and the fat globules may be demonstrated microscopically by staining with Sudan III. Reassurance and treatment of the cause are the mainstay of management.
2. Phosphaturia–Urine clears on addition of acetic acid. Oral alluminium hydroxide (antacid) may alleviate the problem.
3. Lipiduria–Fat droplets usually float on the top of the urine or may be separated as a layer on top after ultracentrifugation. Conditions associated with lipiduria are diabetes mellitus, nephrotic syndrome, eclampsia, arsenic poisoning.
4. Pseudochyluria–Usually turbid urine as a result of large number of degenerated cells or tumour cells. Turbidity clears on addition of alkali.
5. Pyuria–Large number of pus cells make the urine turbid and is commonly associated with pyelonephritis, cystitis or urethritis. Broad-spectrum antibiotics cure the ailment.

—— o ——

CHAPTER 75

# Yellowish Palms/ Soles

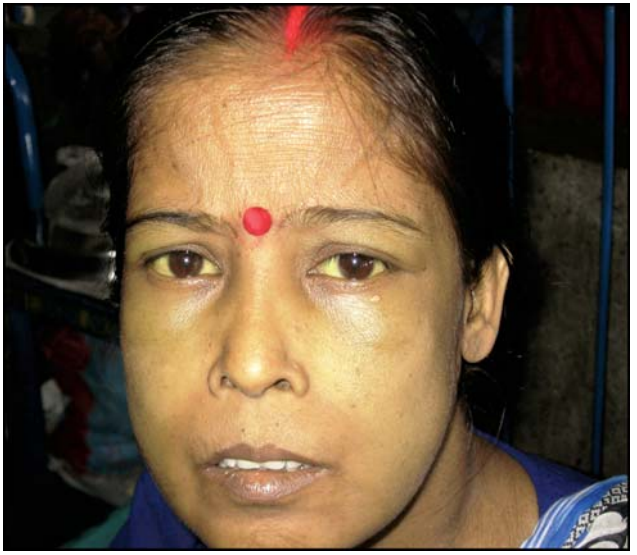


**FIGURE 75.1:** Obstructive jaundice evidenced by deep yellow conjunctiva, greenish-yellow hue in skin and xanthelasma (underneath left eye)

## DIFFERENTIAL DIAGNOSIS

- Jaundice (sclera + mucous membrane + skin) → yellow
- Carotenaemia (only the skin, mainly the palms and soles take a lemon-yellow tint)
- Diffuse xanthomatosis (skin takes a yellowish-orange colour; may have tuberous or palmar xanthoma, xanthelasma → lipid disorder)
- Quinacrine therapy (antimalarial, anti-giardia lamblia)
- Using turmeric for coocking/cosmetic purpose/taboo.

* Carotene, a lipid-soluble yellow pigment in the plasma, does not stain the conjunctiva and mucous membrane. However, the serum looks yellow in carotenaemia.

## CLUE TO DIAGNOSIS OF CAROTENAEMIA

1. Eating large quantities of carotene (vitamin A), carrots, mango, papaya, squash and other coloured fruits/vegetables.
2. Hypothyroidism → due to impaired metabolism of β-carotene in the liver.
3. Diabetes mellitus.
4. Receiving β-carotene therapy for erythropoietic porphyria.
5. Simmond's disease (panhypopituitarism).
6. Anorexia nervosa (possible defect in conversion of carotene to vitamin A).
7. Castrated male.

## IS CAROTENAEMIA INJURIOUS FOR HEALTH?

Except the cosmetic effect, excess carotene is non-injurious to health. Carotenaemia does not develop into hypervitaminosis A.

## MESSAGE

Sclera never turns yellow in carotenaemia. Hypothyroid patients are mostly susceptible to develop carotenaemia.

## XANTHOMA

1. Planous → classical example is 'xanthelasma' around the eyes (the upper eyelids more common than the lower). They are characteristically soft, yellowish and slightly raised from the surface, and are also seen in palmar creases, underneath the breast, chest or back. Xanthelasma is classically seen in:
   - Familial hypercholesterolaemia
   - Familial dysbetalipoproteinaemia
   - Diabetes mellitus
   - Myxoedema
   - Prolonged cholestasis, e.g. primary biliary cirrhosis
   - Nephrotic syndrome
2. Tuberous → commonly seen in extensor surface of elbow, knee, wrist, ankle and 'buttock'.

## TRIAD OF FAMILIAL HYPERCHOLESTEROLAEMIA

- Tendon xanthoma
- Xanthelasma
- Arcus corneae

## MESSAGE

Alway check the serum lipids, sugar level and thyroid hormones to come an aetiological diagnosis. Remember, few middle-aged or elderly people may have no lipid disorder with xanthelasma.

—— o ——

# Index

## I



## J



## K



## L



## M



## N

## T



## U



## V



## W

## X



## Y



## Z