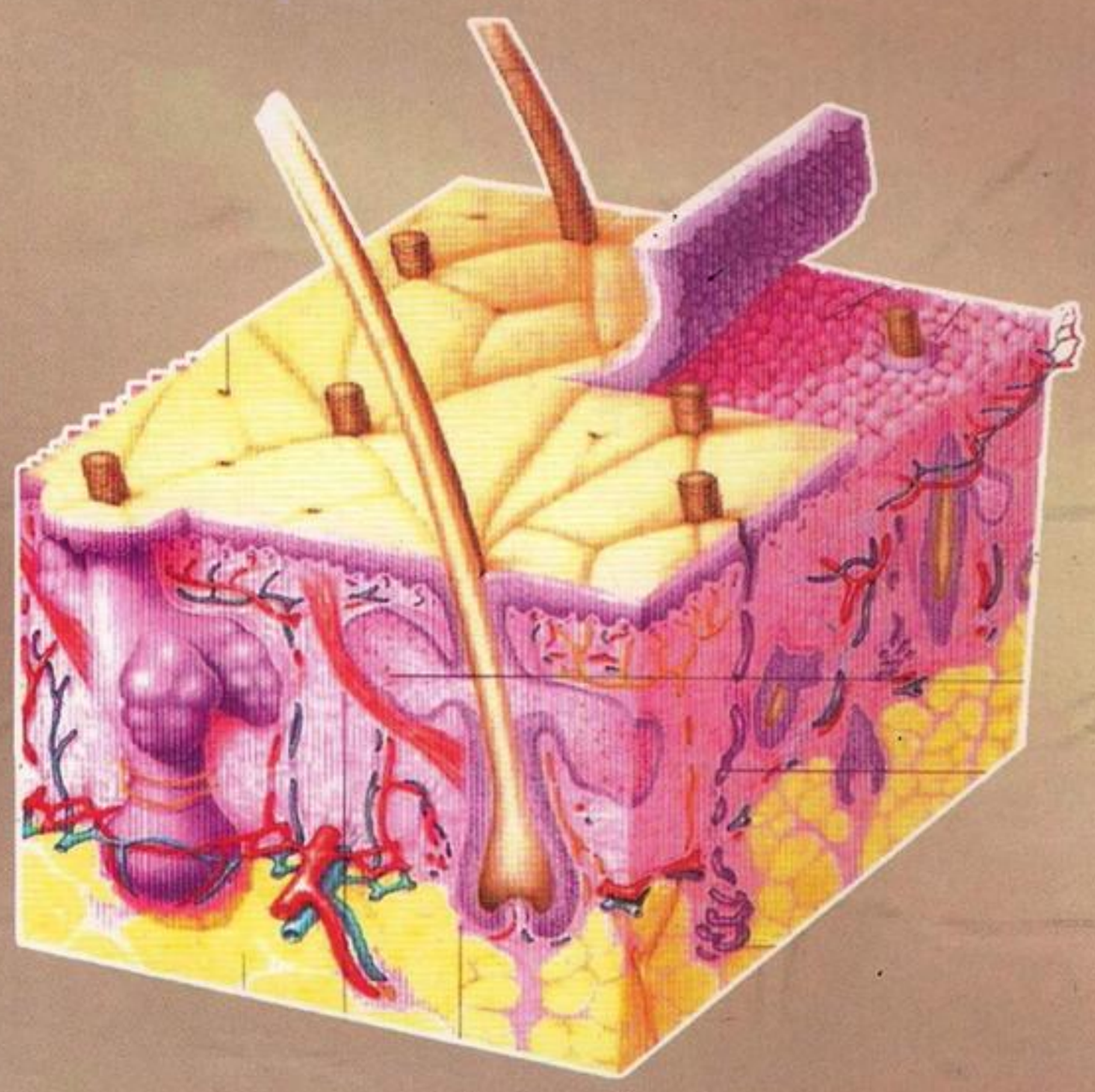
Department of Dermatology & Venereology

BASICS OF DERMATOLOGY, SEXUALLY TRANSMITTED DISEASES AND ANDROLOGY FOR UNDERGRADUATE STUDENTS



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Section 1

DERMATOLOGY

BASIC PRINCIPALS

SKIN ANATOMY

The skin is divided into three layers: epidermis, dermis, and subcutaneous tissue. In addition, there are skin appendages: nails, hair follicles, sebaceous glands, eccrine and apocrine sweat glands (Fig. 1).

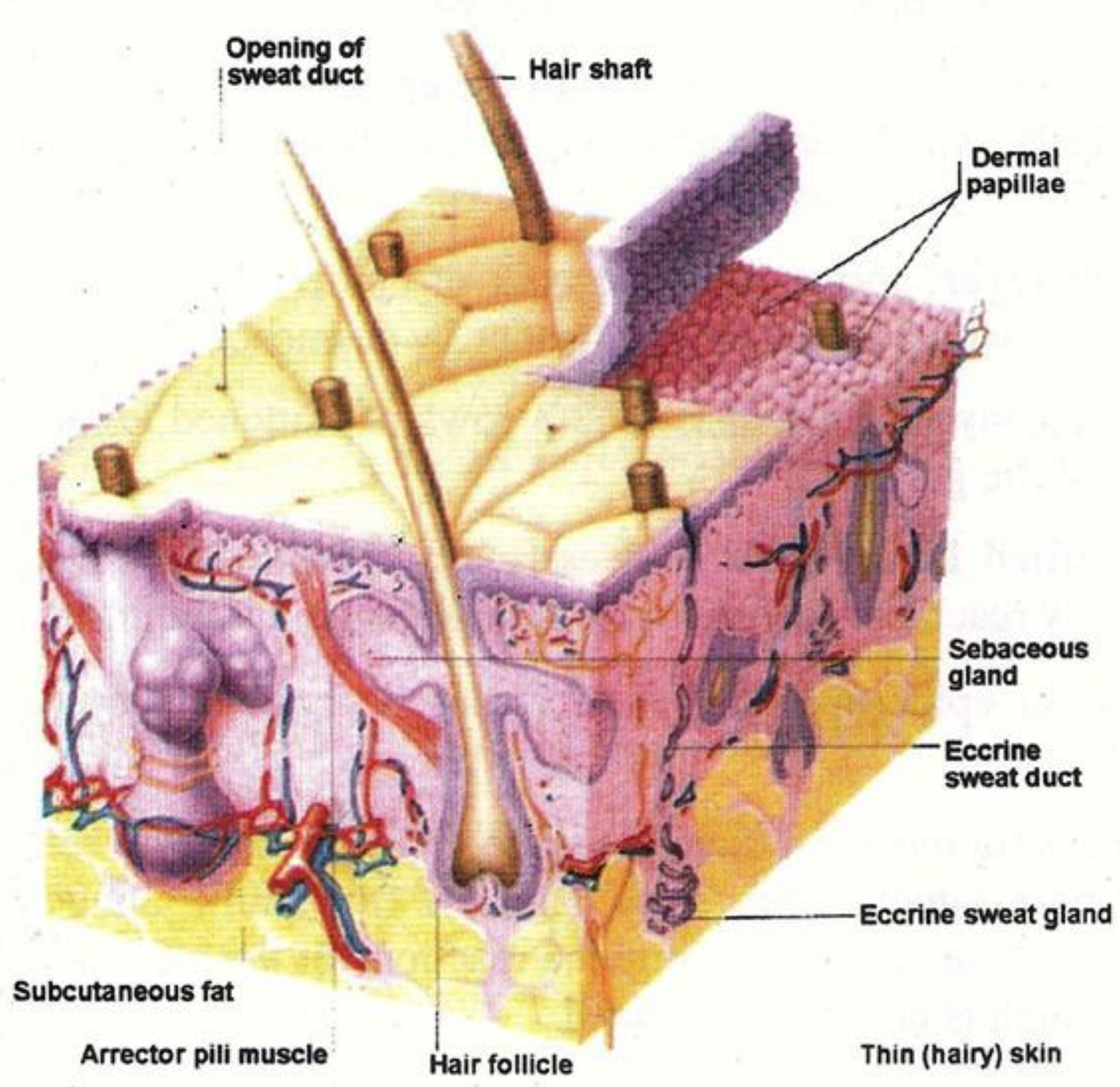


Fig. 1: Schematic illustration of the skin layers and structure.

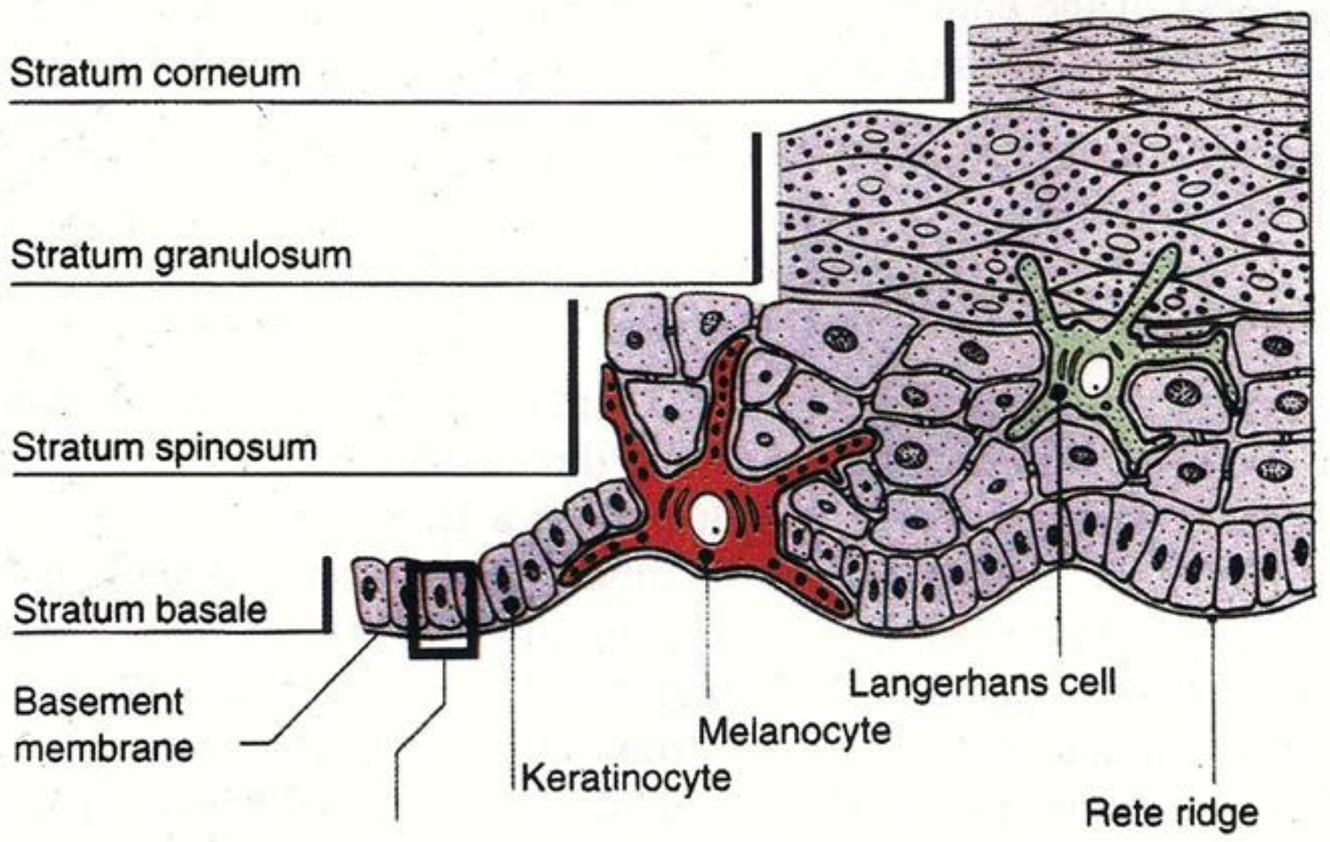


Fig. 2: Different layers of the skin.

The Epidermis

The epidermis is the outermost part of the skin. It is stratified squamous epithelium. It is thin on the eyelids and thick on the palms and soles. Its prime function is to act as a protective barrier. The main cell of the epidermis is the **keratinocyte** which produces the protein keratin. The epidermis is avascular structure and its main blood supply is through the dermis. The four layers of the epidermis (Fig.2) represent the stages of maturation of keratin by keratinocytes.

1. **Basal cell layer:** The innermost layer of the epidermis. It consists of a single row of columnar cells which are anchored to the basement membrane by adhesion junctions called hemidesmosomes.
2. **Spinous cell layer**: forms the great bulk of epidermis. It consists of 5-7 rows of polygonal cells which are attached together by desmosomes.
3. **Granular cell layer:** it consists of 2-3 rows of flattened keratinocytes filled with deeply basophilic granules.
4. **Horny cornified layer:** keratinocytes finally die (loose their organelles including nuclei) as they reach the surface to form the stratum corneum.

- Regeneration of epidermis and hair follicles depends on population of epidermal stem cells.
- In addition to keratinocytes, there are three types of branched cells in the epidermis: the melanocyte, which synthesizes pigment (melanin); Langerhans' cell, which serves as a frontline element in immune reactions of the skin; and Merkel's cell, the function of which is not clearly defined.

Proliferation and shedding

- The thickness of the epidermis is regulated by the balance between proliferation of basal keratinocytes and shedding of cells at the surface (desquamation).
- It takes about 40 days for a keratinocyte to move upwards from the basal layer to the horny layer.

The Dermis

- The dermis varies in thickness from 0.3 mm on the eyelid to 3.0 mm on the back; it is composed of cellular constituents and three types of connective tissue: collagen, elastic tissue, and reticular fibers. It acts as a frame work and support for blood vessels, lymphatic vessels and nerve supply of the skin and skin appendages. The dermis is divided into two layers: the thin upper layer, called the papillary layer, is composed of thin, haphazardly arranged collagen fibers; the thicker lower layer, called the reticular layer, extends from the base of the papillary layer to the subcutaneous tissue and is composed of thick collagen fibers that are arranged parallel to the surface of the skin.
- The junction of the epidermis and dermis is formed by the basement membrane zone and is called the dermo-epidermal junction.

Skin appendages:

1- Eccrine sweat glands:

- They are present on all body sites except very few areas as the labia minora and the glans penis.
- The gland consists of coiled secretory part deep in the dermis and a duct which opens separately on the skin surface.
- The secretion of eccrine sweat gland is a clear watery fluid which plays an important role in the process of heat regulation and has antimicrobial properties as well.

2- Apocrine sweat glands:

- They are present in few areas predominantly in the axilla, anogenital area, female breast, eyelids and external auditory canal.
- The gland consists of coiled secretory part in the deep dermis and duct which opens into the upper portion of the hair follicle.
- The glands produce on oily secretion of protein, carbohydrate, ammonia, and lipid.
- Apocrine glands become active at puberty.

3- Sebaceous glands:

- They are found all over the skin surface except palms and soles.
- It consists of secretory portion and a duct which opens into upper part of a hair follicle, the hair follicle and associated gland is known as a pilosebaceous unit.
- Sebocytes secrete lipid-rich sebum with emollient as well as antimicrobial properties.
- Both apocrine sweat glands and sebaceous glands are under control of testosterone and therefore, become active at puberty.

4- Hair follicle:

- Hairs are present all over the body except palms, soles, dorsal aspects of the distal phalanges, labia minora and glans penis.
- The hair follicle comprises pocket of epithelium which is continuous with the superficial epidermis and a hair shaft which is formed of medulla, cortex and cuticle.
- There are three types of hair: lanugo, vellus and terminal hair.
- And three types of hair follicles: vellus, terminal and sebaceous hair follicles.

5- Nails

They are solid translucent plates composed of modified horny cells.

Dermal Nerves and Vasculature:

- The sensations of touch and pressure are received by Meissner's and the Vater-Pacini corpuscles. The sensations of pain, itch and temperature are received by unmyelinated nerve endings in the papillary dermis. A low intensity of stimulation created by inflammation causes itching, whereas a high intensity of stimulation created by inflammation causes pain. Therefore, scratching converts the intolerable sensation of itching to the more tolerable sensation of pain and eliminates pruritus.
- Autonomic nerves supply the blood vessels, sweat glands and arrector pili muscles.

Melanocytes :

See p.78

FUNCTIONS OF THE SKIN

- The skin is the largest immunologically active organ in the body, and its relative accessibility for basic science research has recently allowed much progress in our understanding of its multiplicity of functions.
- When the skin is unable to play its usual role in the maintenance of fluid balance and temperature regulation, this clinical situation is referred to as "skin failure" and it happens in cases of severe burns and some other skin diseases that simulate severe burns.
- Skin functions can be summarized in the following:

1. **Prevention of water loss.**
2. **Immune defence**:
   - Structural integrity of stratum corneum.
   - Acid Ph of sweat.
   - Fungistatic activity of sebaceous secretions.
3. **Protection against UV damage:**
   - Melanin synthesized by melanocytes helps to protect keratinocyte nuclei from the harmful effects of UV radiation.
4. **Temperature regulation**:
   - Vasoconstriction and vasodilatation control the flow of blood and transfer of heat to the body surface.
   - Evaporation of sweat cools the body.
5. **Synthesis of vitamin D**
6. **Sensation:**
   - Free nerve endings detect potentially harmful stimuli (heat, pain).
   - Specialized end organs detect pressure, vibration, and touch.
   - Autonomic nerves supply blood vessels, sweat glands, and arrector pili muscles.
7. **Aesthetic:** the skin has an important role in social interaction.

DERMATOLOGIC DIAGNOSIS

History taking:

Personal history: age, sex, occupation, special habits …

Present history:

- Onset, course, and duration of the disease.
- The first site affected.
- Is the condition recurrent or it has any particular occurrence or seasonal variation?
- Does it itch or burn & if so is it worse at any particular time?
- Does the skin ooze clear fluid, pus or blood?

- What makes the rash better or worse? Does sunlight makes a difference?

Family history: of similar disorders.

Drug history:

- The response of the condition to previous treatment.
- Drug intake to exclude drug reaction.
- History of any topical agents that have been applied to the skin for medical or cosmetic purposes.

Examination:

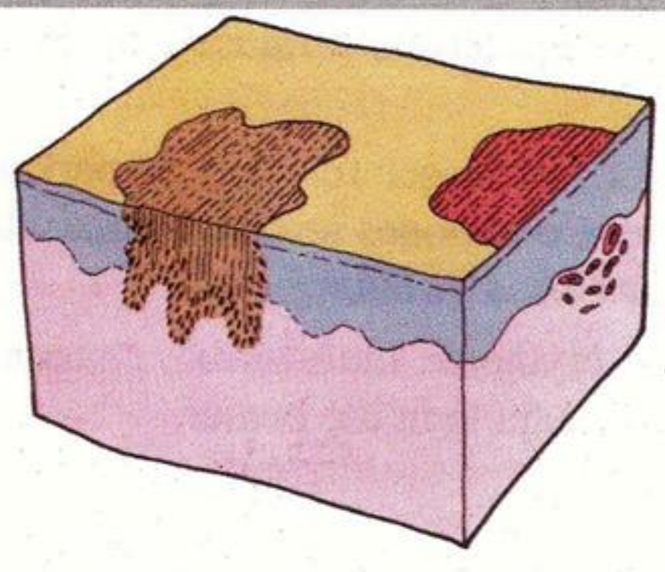
- The patient must be examined under good light closer to natural sun light.
- A magnifying lens is of value in examining small lesions.
- Sometimes the patient should be undressed & examined to determine the overall distribution of the disease.
- Identify the primary lesions & determine the secondary lesions.
- **Diagnostic details of the lesions:**
  - The shape, size, configuration, consistency, content, color, edge & nature of the surface (if it is dry or wet).
  - Presence or absence of scales.
  - Examination of the scalp, hair, nails & mucous membranes.
  - Absence or presence & character of any enlarged lymph nodes must be assessed.

PRIMARY LESIONS

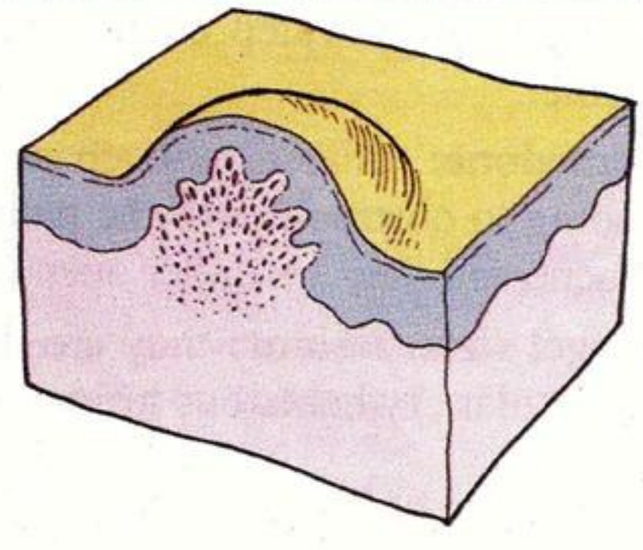
Most skin diseases begin with a basic lesion that is referred to as a primary lesion. Identification of the primary lesion is the key to accurate interpretation and description of cutaneous disease. Its presence provides the initial orientation and allows the formulation of a differential diagnosis. The following are the definitions of the primary lesions (Fig. 3):

1. **Macule:** circumscribed, flat discoloration that may be brown, blue, red or hypopigmented. There is no elevation, depression or change of consistency of the skin. It is less than 1 cm in diameter, confluent macules give rise to a patch.
2. **Papule:** An elevated solid lesion up to 0.5 cm in diameter. Their surface may be flat, conical, rounded or umbilicated. They may be skin colored, red, violet or brown. Papules may become confluent and form plaques.
3. **Nodules:** a circumscribed, elevated, solid lesion more than 1 cm in diameter. They are more deeply seated in the skin. They project both upwards and downwards. A large nodule is referred to as a tumour.
4. **Pustule:** a circumscribed collection of pus (leukocytes and free fluid). Primary pustule contains pus from the start. Secondary pustule is a vesicle that was secondarily infected.
5. **Vesicle:** a circumscribed collection of free fluid up to 0.5 cm in diameter.
6. **Bulla:** a circumscribed collection of free fluid more than 0.5 cm in diameter.
7. **Wheal (hive):** a firm edematous plaque resulting from infiltration of the dermis with fluid. Wheals are itchy evanescent lesions that may last only a few hours, it is the characteristic primary lesion of urticaria.

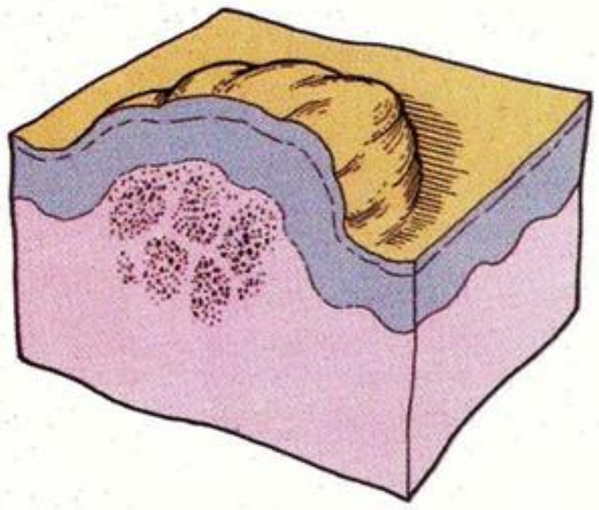
8. **Burrow:** an elevated channel in the superficial epidermis produced by Sarcoptes Scabiei.
9. **Comedone:** dilated pilocebaceous duct filled with sebum and hyperkeratosis leading to obstruction of the pathway of sebum. Comedones are the primary lesions of acne and they are either open (blackheads) or closed (whiteheads).
10. **Target (iris) lesions:** they are the hallmark of erythema multiforme, characterized by circular erythematous lesions with a vesicle or papule in the center.



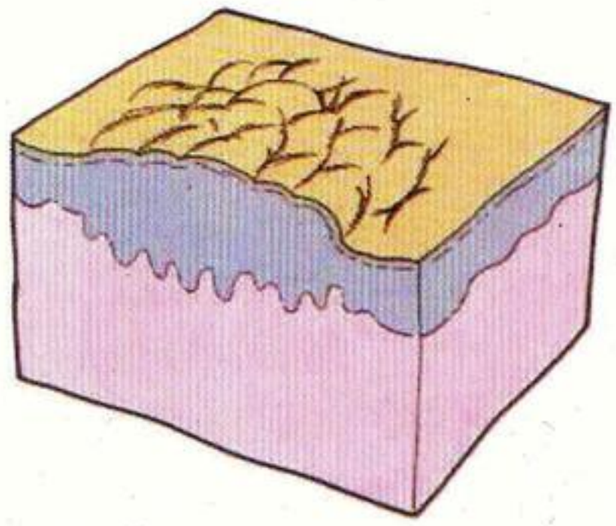
Macule



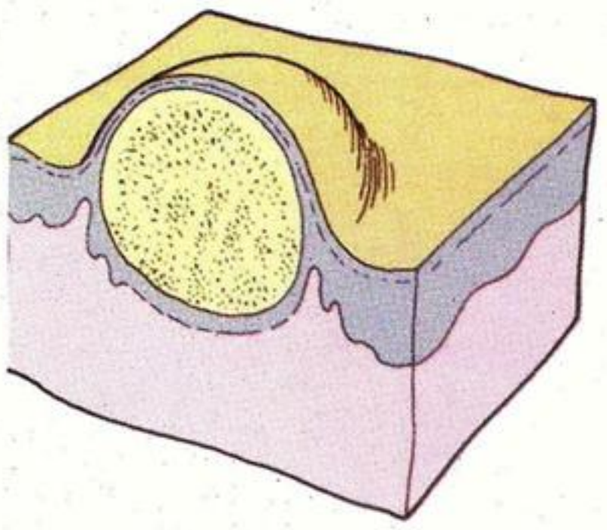
Papule



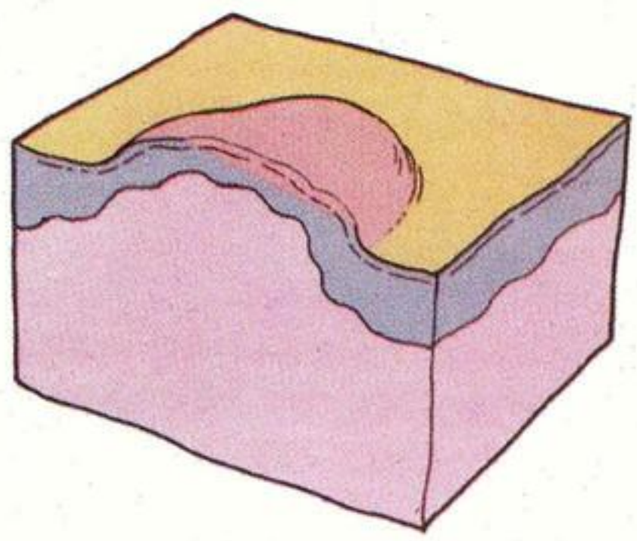
Nodule



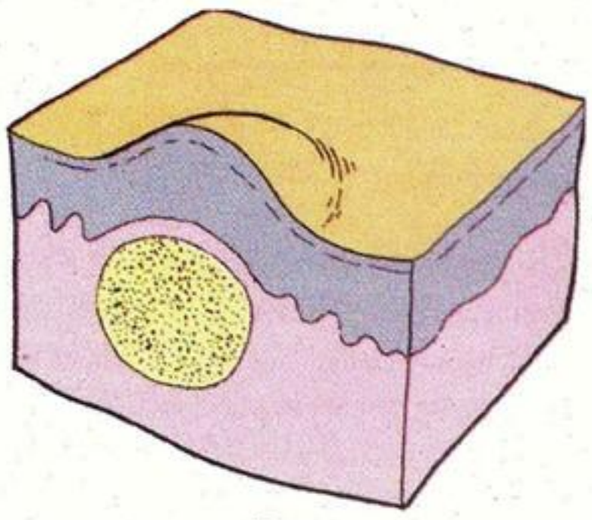
Plaque



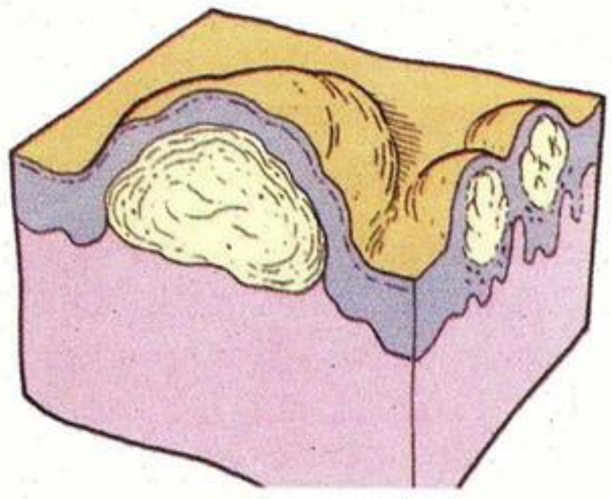
Pustule



Wheal



Cyst



Bulla

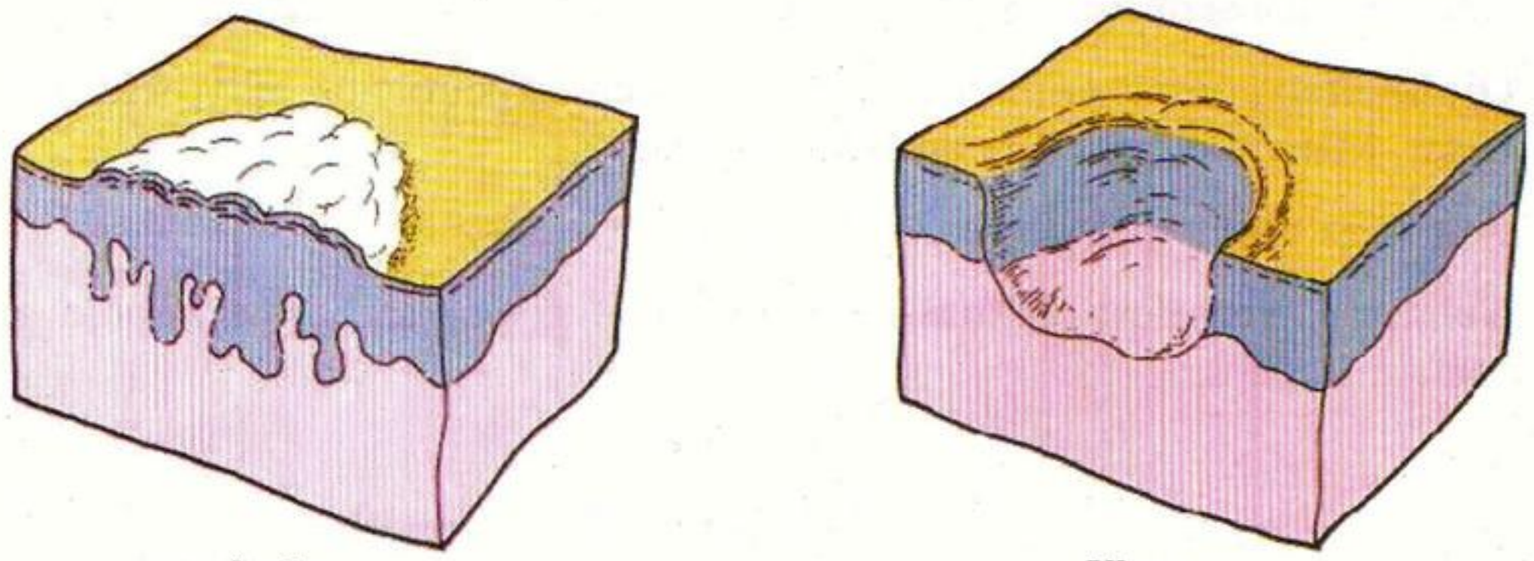
Vesicle

Fig. 3: Primary lesions of the skin.

SECONDARY LESIONS

Secondary lesions develop during the evolutionary process of skin disease or are created by scratching or infection. They may be the only type of lesion present, in which case the primary disease process must be inferred. The differential diagnoses of secondary lesions are as follows (Fig.4):

1. **Scales:** Excess dead epidermal cells (horny layer) that are produced by abnormal keratinization and shedding. They may be dry or greasy, large or small.
2. **Crust:** a collection of dried secretions.
3. **Erosion:** a focal loss of epidermis. Erosions do not penetrate below the dermoepidermal junction and therefore heal without scarring.
4. **Ulcer:** a focal loss of epidermis and dermis. Ulcers heal with scarring.
5. **Fissure:** a linear loss of epidermis and dermis with sharply defined nearly vertical walls.
6. **Atrophy:** a depression in the skin resulting from thinning of the epidermis or dermis.
7. **Scar:** an abnormal formation of connective tissue implying dermal damage, after injury or surgery. Scars are initially thick and pink but with time become white and atrophic. It is the fibrous tissue replacement of the skin substance destroyed by injury or disease.
8. **Lichenification:** it is a change in the skin which results from prolonged and repeated irritation. The skin is thickened with accentuation of skin markings and is usually hyperpigmented.



Scale **Ulcer**

Fig 4. Secondary lesions of the skin.

Special skin lesions:

Telangiectasia, petichae, purpura.

HISTOPATHOLOGICAL TERMS

- **Hyperkeratosis:** Increase thickness of the cornified layer
- **Orthokeratosis:** Normal cornification (cells have cornified completely without retained nuclei).
- **Parakeratosis:** Pyknotic nuclei are retained in cells of horny layer with reduction in granular cell layer.
- **Acanthosis:** Increase in the thickness of stratum malpighii.
- **Papillomatosis:** Accentuation of dermal papillae.

BASIC PRINCIPLES OF TOPICAL THERAPY

- Topical therapy plays an important role in dermatologic treatment. Topical preparations are composed of a vehicle which is an inert carrier of the active ingredient
- The vehicles are present in different forms:
  - **Liquids:** They may be in the form of:
    - *Solutions:* aqueous solutions of pharmacologically active substances e.g. potassium permanganate 1/8000, lead subacetate 0.5%.
    - *Lotions:* suspension of powder in water and require shaking before application e.g. calamine lotion.
    - *Tincture:* alcoholic solution e.g. tincture iodine.
  - **Creams**: are semisolid emulsions of oil in water less occlusive than ointment.
  - **Ointments**: consist primarily of greases such as petroleum jelly, with little or no water. Many ointments are preservative-free.
  - **Powders:** promote drying e.g. talc powder.
  - **Gels**: are greaseless mixtures of propylene glycol and water; some gels also contain alcohol.

Questions

Match the following with the appropriate phrases:

1. Stratum corneum.
2. Stratum basale.
3. Spinous cell layer.
4. Granular cell layer.
5. Epidermis.
6. Dermis.

1 Basic Principles

a. Stratified squamous epithelium.

b. Cells loose all characteristics of life.

c. Cells are flattened.

d. Contains elastic and collagen bundles.

e. Intercellular bridges between cells are evident with light microscopy.

f. Single row of cells.

BACTERIAL INFECTIONS OF THE SKIN

Normal flora of the skin:

The normal human skin is colonized by huge numbers of bacteria which live harmlessly as commensals on its surface and within its follicles: e.g. Staphylococci (S. epidermidis, S. hominis, ..), aerobic coryneform bacteria, anaerobic propionibacteria (P. acne, ...), gram negative bacilli, in addition to yeasts (Pityrosporum ovale and P. orbiculare).

Natural Resistance of the skin:

The normal skin of healthy individuals is highly resistant to invasion by bacteria to which it is constantly exposed. The factors involved in this local resistance are:

1. The relative dryness of the skin surface limits the multiplication of bacteria.
2. The constant shedding of the cells of the horny layer.
3. The low pH of the skin environment (5.5).
4. The natural antibacterial substance in the sebaceous secretion.
5. The normal cutaneous flora (bacterial interference).

Classification of pyogenic diseases of the skin:-

Pyogenic diseases of the skin are mainly caused by Staphylococci (Staph. aureus) and Streptococci (Strept. pyogenes). They may be the primary cause of certain skin diseases (Table 2.1) or may cause secondary infection of other skin diseases, such as eczema, ulcers, itchy skin conditions etc.

Table 2.1 Diseases caused by direct infection of the skin and skin appendages by Staph. aureus and Strept. pyogenes

Staph. aureus	**Strept. pyogenes**
Impetigo	Impetigo
Ecthyma	Ecthyma
Cellulitis (occasionally)	Cellulitis
Folliculitis	Erysipelas
Furunculosis	Bacterial intertrigo
Carbuncle	Angular stomatitis

IMPETIGO

Impetigo is an acute contagious superficial pyogenic infection of the skin. It is the most common bacterial infection of the skin. It is more common in the first 5-10 years of life, particularly during the summer months.

Causative organism: Staph aureus or Strept pyogenes or both.

Predisposing Factors:

1. Unhygienic conditions and overcrowding.
2. Malnutrition.
3. Presence of a septic focus or systemic disease.
4. Itchy skin conditions as pediculosis or scabies.

Clinical varieties:

1. Ordinary impetigo contagiosum:

This is the commonest form (70%). It is usually caused by Staphylococcus aureus and/or Group A β-hemolytic Streptococci.

Distribution:

- The usual location of ordinary impetigo is the face especially around the mouth & nose and the extremities.
- It occurs frequently on the scalp, complicating pediculosis.

Clinical Picture:

- The initial lesion is a thin-walled vesicle on an erythematous base. Then, it ruptures so rapidly that it is rarely seen as such.
- The exuding serum dries to form yellowish – brown, honey-colored, stuck – on crusts (hallmark of the disease).
- The crusts dry & separate (within 2 weeks if untreated), leading to erythema that fades without scarring (may leave temporary hypo- or hyperpigmentation).
- Regional lymphadenopathy is common.
- **Circinate impetigo:** Results when impetigo lesions extend peripherally, with central healing forming annular, circinate or gyrate patterns. Also circinate impetigo might result from multiple lesions arranged in a circular fashion.

2. Bullous impetigo:

Causative organism is staphylococci (phage II type 71), which secrete toxins that lead to acantholysis at the epidermal granular layer.

It mainly affects neonates & infants, whose kidneys cannot excrete this toxin.

Distribution:

- The trunk is the most common affected site. It may spread & become generalized.

Clinical Picture:

- The initial lesion is a small flaccid bulla with a faint rim of surrounding erythema containing clear yellow turbid fluid. The roof of the bulla tends to collapse rather than rupture, retaining the fluid content that finally desiccates & a thin flat crust is formed.
- Central healing and peripheral extension may form a circinate lesion.
- Regional lymph nodes are usually not enlarged.

Diagnosis of Impetigo:

1. Based primarily on clinical presentation
2. Gram stain and appropriate cultures are needed for isolating the specific bacterial pathogen. Sensitivity testing is done to use appropriate therapy.

Complications of Impetigo:

They are unusual. The post–streptococcus glomerulonephritis (most serious) occurs 2-3 weeks after acute infection. It showed a decrease in its incidence in recent years mainly due to shift in the infecting strains.

Treatment of Impetigo:

1) Treatment of predisposing factors (Pediculosis and scabies if present should be treated as soon as there is satisfactory response to treatment of impetigo).
2) Local treatment:
   i) Gentle removal of the crust using olive oil (hot foments).
   ii) Antiseptic lotions as potassium permanganate 1/5000-1/10000.
   iii) Topical antibiotic creams: fusidic acid or bacitracin cream in localized lesions.
3) Systemic broad spectrum antibiotics in widespread lesions (for 5-7 days):
   i) β-lactamase resistant antibiotics e.g. amoxicillin/clavulonate, ampicillin/cloxacillin, cephalexin.
   ii) Erythromycin, azithromycin.

ECTHYMA

Ecthyma is a deeper form of pyogenic infection (also known as ulcerative impetigo). It heals by scar formation.

Causative organism: It is mainly caused by streptococci (less by streptococci & staphylococci).

Predisposing Factors:

1. Minor trauma e.g. itchy skin conditions.
2. Poor hygiene, malnutrition.

Distribution: The most common involved site is on the lower extremities (shin of tibia).

Clinical picture:

- It mainly affects children, neglected elderly, lymphedematous patients and immunocompromised patients.
- The initial lesion is a vesicle with an erythematous base & surrounding halo that enlarges to 0.5 –3 cm in size and then ruptures leaving a dark adherent crust surrounded by a rim of erythematous indurated area.
- Removal of the crust shows a saucer (dish) shaped ulcer with raised, indurated, violaceous margin. The base extends deeply in the dermis. It heals with scar formation.
- Local lymphadenopathy may be present.

Complications:

- Cellulitis and osteomyelitis occur infrequently.
- Rarely systemic symptoms and bacteremia are seen.

Treatment:

1- Treatment of predisposing factors.

2- Appropriate antibacterial therapy (for 10-14 days).

FOLLICULITIS

It is infection of the pilosebaceous apparatus usually by staphylococci.

Predisposing Factors:

1. Occlusion, maceration, hyperhydration.
2. Diabetes mellitus.
3. Shaving, plucking hair.
4. Hot humid weather.
5. Atopic dermatitis (due to increased colonization by staph).

Classification:

1. Acute folliculitis:

i. Acute superficial folliculitis (Bockhart's impetigo):

Distribution: Sites involved are the scalp & extremities. It can be seen also on the face.

Clinical Picture:

- It occurs at any age (more frequent at school age).
- The initial lesion is thin walled yellowish–white domed pustule at the follicular orifices, which is pierced at its center by a hair. It heals without scarring.

Treatment:

- Topical antiseptics.
- Topical antibiotics in localized lesions and systemic antibiotics in disseminated lesions.

ii. Acute deep folliculitis (Furunculosis or boils):

Distribution: Face & neck, buttocks & perineum, thighs & arms, wrist & fingers.

Clinical Picture:

- It occurs at any age.
- It affects the deeper parts of the hair follicles that ends in central suppuration and formation of the core (characteristic of the boil).
- The initial lesions are single or multiple hard, tender nodules that enlarge and become painful, then pustular and necrotic.
- Healing after discharge of the necrotic core may be complicated by scarring & post–inflammatory hyperpigmentation.

Treatment:

- Warm compresses may enhance maturation, drainage and resolution.
- Incision and drainage of fluctuant lesions.
- Systemic antibiotics for large and recurrent lesions, lesions around nose, in nares or external auditory canal, lesions with surrounding cellulitis, or lesions not responsive to local care.

iii. Carbuncle

Distribution:

The most commonly involved sites are the back of the neck, shoulders, buttocks and thighs.

Clinical Picture:

- Adults, mainly diabetics & immunocompromised individuals are the susceptible group.
- The initial lesion is in the form of indurated, tender circumscribed area with deep staphylococcal infection of a group of adjacent follicles with subsequent perforation of the hair follicles.
- Local spread of infection leads to mass necrosis of the infected tissue and expulsion of the necrotic material to the outside through multiple openings.

Treatment:

Surgical drainage and appropriate antibiotics.

2. Subacute & Chronic folliculitis:

i. Sycosis barbae:

Distribution: Beard area.

Clinical Picture:

- Adult males are susceptible.
- The initial lesion is in the form of recurrent discrete inflammatory papules or pustules in the beard area pierced by hairs. If neighboring follicles are affected, a plaque studded with pustules may result.
- Rarely in some chronic forms a foreign body reaction results which finally ends in scar formation.

Differential Diagnosis:

- **Pseudofolliculits:** It is a non-infectious, inflammatory condition (foreign body reaction to hair) caused by ingrowing hairs in people who shave. It mostly affects men with curled hair. Papules and pustules commonly occur on the anterolateral aspect of the neck or on the angle of the jaw in men.

Note that pseudofolliculitis could also affect women who shave especially in the groin area.

- **Tinea barbae:** Inflammatory papules and pustules on beard area with crusting and loosened hair. KOH and culture are positive for fungus.

Treatment:

- Topical antibiotics.
- Systemic antibiotics for resistant cases.
- Chronic forms may require steroid/antibiotic combination.

ii. Acne keloidalis (folliculitis keloidalis):

Distribution:

It most commonly involves the occipital area of the scalp.

Clinical Picture:

- It occurs mostly in adult males.
- The initial lesion is in the form of small pustules that are followed by firm, dome-shaped follicular papules. These slowly enlarge and coalesce and may form keloid-like plaques.

Treatment:

- Local or intralesional corticosteroids that decrease scar formation.
- Topical or systemic antibiotics for pustules.

ERYSIPELAS

This is an infection of the dermis with significant lymphatic involvement caused by haemolytic streptococci. The organism reaches the dermis through a wound or a small abrasion.

Distribution:

The Commonest sites are face and legs.

Clinical picture:

- It mainly affects very young children, elderly, debilitated, lymphoedematous people and those with chronic cutaneous ulcers.
- After an incubation period of 2-5 days, the disease begins with sudden onset of fever, rigors & malaise. This is followed few hours or up to 1 day later by the eruption. The skin shows redness, hotness, swelling and pain.
- The lesion is in the form of a well-demarcated red area. The lesion has an advancing edge and the surface may show vesicles, pustules or bullae.

Complications:

1. Recurrent erysipelas may lead to lymphoedema.
2. Nephritis.
3. Subcutaneous abscess.
4. Septicaemia.

Differential diagnosis:

1. Cellulitis.
2. Angio-oedema.
3. Acute eczema.

Treatment:

1. Rest in bed and antipyretics.
2. Penicillin is the drug of choice (given for 10-14 days). Other antibiotics (e.g.macrolides) may be used if the patient is sensitive to penicillin or the condition does not respond.

CELLULITIS

Cellulitis is an acute or subacute inflammation of deep dermis & subcutaneous tissue. It is deeper than erysipelas. It is caused by strept or staph. It occurs most commonly as a complication of a wound or skin lesion.

Distribution: The leg is the commonest site.

Clinical picture:

- Ill-defined, indurated, red, tender hot area of skin appears at the affected site.
- Systemic symptoms (fever, malaise).

Treatment:

1. Proper antibiotics.
2. Hospitalization of severely ill patients.

INTERTRIGO

The term intertrigo denotes inflammation of skin areas in between folds, e.g. behind the ears, under the breasts, axillae, groins and skin folds in obese persons.

Causes of intertrigo:

1. Streptococcal intertrigo: Longitudinal painful fissure occurs at the angle of the skin folds. The skin around is red, moist & may be crusted.
2. Simple intertrigo due to friction.
3. Candidal intertrigo.
4. Flexural psoriasis.
5. Seborrheic dermatitis.
6. Tinea cruris.
7. Erythrasma.
8. Contact Dermatitis.

Treatment of bacterial intertrigo:

1. Frequent washing with antiseptic lotion as potassium permanganate lotion 1/8000.
2. Antibiotic creams.
3. Systemic antibiotics.

PARONYCHIA

Paronychia is inflammation of the nail fold. It may be acute and chronic.

Acute Paronychia:

- Bacterial paronychia is common, especially in children and follows minor trauma to the nail.
- It is mainly caused by staphylococci and streptococci.
- **Clinical picture**:

  The affected digit is red, swollen and painful. Compression of the nail fold may drain pus.

- **Treatment:**

  1. Drainage of pus.
  2. Systemic antibiotics

Chronic Paronychia:

- Chronic Paronychia involves the nail folds of adult women.
- Occupational paronychia is common in food handlers.

Etiology:

The condition represents a contact reaction to irritants or allergens. Secondary infections by candida or bacteria are common.

- **Clinical Picture:**

One or several fingernails are affected. There is inflammation of the proximal nail fold with erythema, edema and absence of the cuticle. Damage to the nail matrix results in nail plate changes.

- **Treatment:**

1. Avoidance of water and chemical exposure.
2. Combined topical corticosteroids, antifungals and antibiotics.

ANGULAR CHEILITIS

Angular cheilitis (perleche) is inflammation of the angle of the mouth.

Predisposing factors:

1. Excessive salivation.
2. Ill-fitting dentures.
3. Debilitating diseases and old age.

Causes of angular cheilitis:

1. Chronic streptococcal infection.
2. Infection with candida.
3. Riboflavin deficiency.

Treatment:

1. Treatment of the predisposing factors.
2. Vitamin B-complex.
3. Topical antifungal &/or topical antibiotic.

Erythrasma

A mild chronic, localized, superficial infection of the intertriginous areas.

- **Etiology:**

Corynebacterium minitissimum.

- **C/P:**

  Well defined reddish brown patches with no active edge, covered with fine scales, that affect the intertriginous areas. Asymptomatic or associated with mild itching.

- **Diagnostic methods:**

  Wood's light examination gives coral red fluorescence.

- **Treatment :**

  Topical antifungal and systemic erythromycin (if needed).

❖ Atlas Fig. (20-26).

Questions

- Impetigo contagiosum skin lesions usually heal by:

  a. Scarring.

  b. Pigmentation.

  c. Normal skin.

  d. Thinned out skin.

- Impetigo contagiosum primary skin lesion is:

  a. A pustule.

  b. A papule.

  c. A vesicle.

  d. A comedone.

- Corynaebacterium minutissimum is the cause of:

  a. Diphtheria.

  b. Erythrasma.

  c. Folliculitis.

  d. Carbuncle.

MYCOBACTERIAL INFECTIONS

Mycobacteria is a large group of bacteria that are thin, aerobic, slender, slightly curved to straight, non-motile and non spore-forming with a waxy coating that makes them resistant to most stains. They are acid-fast which means they are not readily decolorized by acid after staining. The most important mycobacterial infections are Tuberculosis (T.B.) and Leprosy.

CUTANEOUS TUBERCULOSIS

Etiology: It is caused by Mycobacterium tuberculosis (M. tuberculosis) and Mycobacterium bovis (M. bovis).

Epidemiology:

It has a world-wide distribution but more prevalent in cold humid climates and in tropics. It is transmitted by inhalation, ingestion or inoculation.

Clinical picture:

The various clinical presentations depend on:

- Virulence and number of organisms.
- Resistance and immune status of the host.
- Route of skin involvement.

Classification of cutaneous tuberculosis (clinical types):

Lesions caused by direct inoculation of the skin	Lesions due to endogenous infections	Cutaneous immune reactions to M. tuberculosis
Tuberculous chancre Tuberculosis verrucosa cutis Lupus vulgaris (occasionally)	Scrofuloderma Acute miliary T.B. Orificial T.B. Lupus vulgaris	Papulonecrotic tuberculids Erythema induratum of Bazin

Tuberculous chancre:

- **Incubation Period=** 2-4 weeks after inoculation of M. tuberculosis into the skin of persons without immunity to this organism (not previously exposed to it). It is analogous to Ghon's complex of the lung.
- It is firm, painless, reddish-brown papulonodule which enlarge and eventually ulcerates. It usually heals spontaneously in 3-12 months leaving atrophic scar and rarely progress to lupus vulgaris or TB verrucosa cutis. Frequently it spread to draining lymphatics and lymph nodes. It occurs at the site of inoculation.

3 Mycobacterial Infections

Tuberculosis verrucosa cutis (warty T.B.):

- It is due to direct inoculation in previously infected persons with high degree of immunity.
- There is asymptomatic, indurated wart-like plaque with surrounding dusky erythema. The center may become fluctuant and the fissured surface express pus and keratinous debris on pressure.
- It appears more commonly on fingers, dorsum of hands and sole of feet. It is common in butchers, doctors, pathologist…etc.
- **Differential Diagnosis:** Viral warts. Warty T.B. has erythema and signs of inflammation.

Lupus vulgaris:

- It is the commonest type. It occurs in previously sensitized persons with moderate to high immunity.
- The primary lesion is a deep-seated yellowish-red to red-brown papulonodule called "apple jelly nodule" which enlarges gradually and coalesce forming plaque. The plaque extends slowly and a scar forms centrally which is thick, contractile and unhealthy (i.e. contains active nodules). Pressing on the lesion by a glass slide leads to fading of the erythema and the nodules appear as yellow-brown spots (**Diascopy test**).
- It occurs more on the face, cheeks and nose, but can also occur on distal extremities.
- **Complications:**
    1. Destruction of underlying structures specially cartilages.
    2. Ectropion, microstomia.
    3. Squamous cell carcinoma may develop in chronic long-standing scars.

  All these complications occur over a period of years.

Scrofuloderma:

- This occurs in persons with low immunity. It is skin lesions extending directly from T.B. of underlying structures mostly lymph nodes or bones but may be joints or epididymis.
- Red indurated nodule which becomes fluctuant and opens by sinuses discharging caseous material. Healing occurs by scars and keloids.
- It is seem commonly on the axillae, neck, ankles and back.

Acute miliary T.B:

- It occurs in persons with low immunity as a result of bacteremia and the primary focus is often in the lungs.
- Lesions are non-specific pin-head-sized bluish-red papules capped with minute vesicles which may evolve into crusts.

T.B. orificialis:

- It results from autoinoculation of M. tuberculosis, although occasionally it is due to exogenous origin. It affects persons with severely impaired Cell Mediated Immunity and advanced internal T.B.
- Usually there are edematous red papules that ulcerate and develop undermined edge.
- It occurs within or adjacent to natural orifice: lips, tongue, nose (pulmonary T.B.); around the anus (intestinal T.B.); around the urethra (genitourinary T.B.).

Diagnosis of cutaneous T.B.:

- History and clinical picture. This includes the presence of T.B. elsewhere in the body.
- Smear from the lesion stained by Ziehl-Neelsen stain to show the organism.
- Biopsy and histopathological examination that may show caseating granuloma.
- Tuberculin test: to show the immune status of the individual. It can be negative in:
  a) Immunocompromised patients.
  b) Persons never exposed to M. tuberculosis.
- Culture on Lowenstein-Jensen medium.
- Polymerase chain reaction (PCR): to detect the DNA of the mycobacteria in tissues.

Treatment of cutaneous T.B.:

It is the same as for systemic T.B.

A- General measures: to improve health and nutrition.

B- Antituberculous drugs:

1. Isoniazid (INH): 300 mg/day. It is cheap, effective, of low toxicity but may cause peripheral neuritis.
2. Rifampicin: 450 mg/day in patients <50 kg. 600 mg/day in patients >50 kg. it is effective but hepatotoxic and causes red discoloration of urine.
3. Pyrazinamide: 2 gm/day in patients from 50-75 kg. It may cause hepatitis and arthritis.
4. Ethambutol: 15 mg/kg/day. It may cause visual disturbance and retrobulbar neuritis in children.

5. Streptomycin: 0.75-1 gm intramuscularly in adults. It may cause ototoxicity.

Treatment must start with 3 drugs for 2 months e.g. INH, rifampicin and pyrazinamide then continued with 2 drugs e.g. INH and rifampicin. All drugs are taken on empty stomach. Small lesions can be exised.

LEPROSY (HANSEN'S DISEASE)

It is a chronic infectious disease affecting mainly nervous tissue and the skin caused by the bacillus Mycobacterium leprae.

Etiology: M. leprae is an obligate intracellular microorganism i.e. a parasite that lives within macrophages and Schwann cells. It can not be cultivated in vitro but can be cultivated in vivo using the nine-banded armadillo which has a body temperature of 25 °C. As it prefers lower temperatures, M. leprae affects cooler regions of the body such as nose, testicles, ear lobes as well as regions where peripheral nerves are close to the skin.

Epidemiology: The disease is endemic in the Indian subcontinent, Amazon (Brazil) and central Africa. It is of low infectivity and requires long close contact to be infectious.

The mode of transmission is a matter of debate. Suggested modes are:

1- Droplet infection.

2- Contamination with infected blood or blood transfusion.

3- Inoculation through nasal mucosa and less commonly breaks in the skin.

Pathogenesis:

The bacilli when admitted to the body search for a cell for their incubation (Schwann cell) where they lodge and multiply until the capacity of the Schwann cells exceeds their sizes leading to their rupture and the bacilli either go to the skin (dermis) or the blood. Most of the exposed individuals do not develop disease and the susceptibility/resistance depends upon genetic and environmental factors.

Clinical picture:

Incubation Period varies widely from months to 30 years (usually 4-10 years). There is a wide **spectrum** of clinical findings in leprosy (Fig. 5). Cell mediated immunity (CMI) is the most important factor determining the outcome of the disease. M. leprae first may cause a condition called **Indeterminate leprosy** which mostly fades without further sequelae in 75% of cases. In those who will develop further disease, CMI determines whether the patient will develop **Tuberculoid leprosy (TT)** if CMI is fair, will develop **Lepromatous leprosy (LL)** if CMI is weak or will develop one of the **Borderline leprosy** conditions between these polar types.

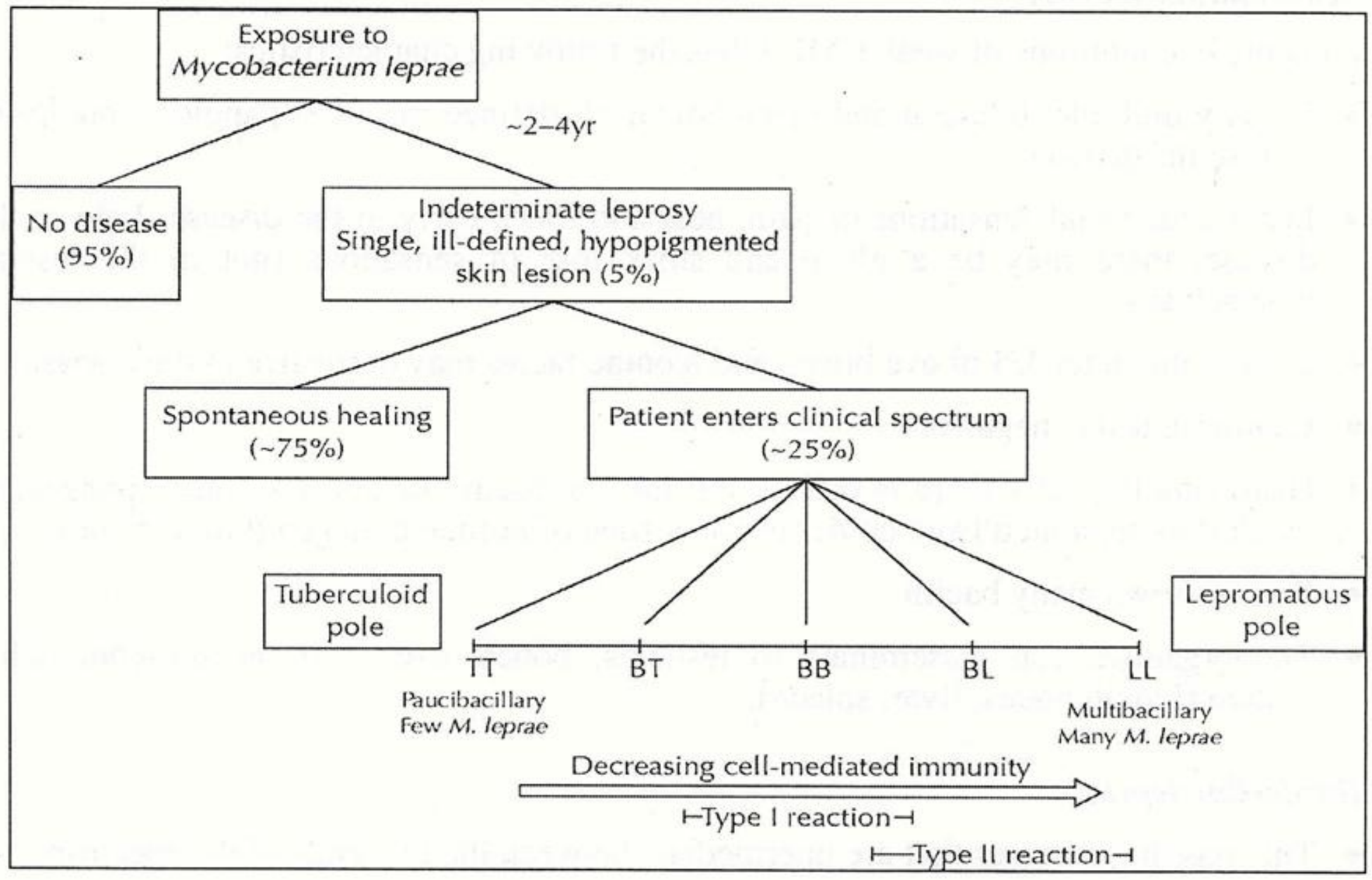


Fig. 5: Clinical spectrum of leprosy.

Indeterminate leprosy

- This is a stage in the evolutional story of leprosy and not actually a leprosy type. It is named "indeterminate" because its clinical, pathological and immunological background can not be determined.
- It may occur as single or rarely multiple ill-defined macules with preserved sensations of heat, touch and pain. Sometimes it may occur in the form of hypopigmented macule and rarely in the form of erythematous non-itchy wheal.

Tuberculoid leprosy

It occurs in conditions of fairly good CMI. It has the following characteristics:

- It is usually single. If multiple, it does not exceed 4-5 lesions.
- It is usually unilateral. If bilateral, it is asymmetrical.
- The lesion is usually well-defined.
- There is usually early impairment of the superficial sensations of pain, heat and touch.
- Lesions are usually hypopigmented, hypohidrotic (reduced sweat secretion) macules and loss of hair in hairy areas may be observed.
- Usually lepromin test is positive.
- Histopathologically there may be linear granuloma following a nerve.
- Smear shows no bacilli.

Lepromatous leprosy

It occurs in conditions of weak CMI. It has the following characteristics:

- Usually multiple, bilateral and symmetrical, ill-defined macules, papules, nodules or diffuse infiltration.
- Intact superficial sensations of pain, heat and touch early in the disease. Late in the disease, there may be a glove and stock loss of sensations (not in the lesions themselves).
- Loss of the outer 1/3 of eye brows and leonine facies may occur late in the disease.
- Lepromin test is negative.
- Histopathologically there is diffuse infiltrate of foamy histiocytes (macrophages) in the dermis separated from epidermis by a zone of normal collagen (Grenz zone).
- Smear shows many bacilli.
- The organism can disseminate to testicles, bones, eyes and reticulo-endothelial system (lymph nodes, liver, spleen).

Borderline leprosy

- This type has features that are intermediate between the two ends of the spectrum.
- Skin lesions are usually asymmetrical.
- If lesions are more towards the TT, it is called **Borderline tuberculoid leprosy (BT).**
- If lesions are more towards the LL, it is called **Borderline lepromatous leprosy (BL).**

Nerve involvement in leprosy

- It appears more in certain superficial nerves like the ulnar, great auricular, posterior tibial and superficial peroneal nerves.
- The ulnar nerve is a classically involved nerve in leprosy due to the following:

  a) Covered by a thick Schwann sheath.

  b) Vulnerable to trauma as it lies behind a bony structure.

  c) Located superficially where the temperature is low; a condition that favors the growth and multiplication of M. leprae.

- The following facts should also be noted about nervous affection in leprosy:

a) TT affects the skin after the nervous tissue.

b) Muscle atrophy occurs in TT due to affection of the nerve supply.

c) The affected cranial nerves in leprosy are: olfactory, trigeminal and facial.

Lepra reactions

They are characterized by acute inflammation that occurs either spontaneously, with drug administration, infections, pregnancy or even mental stress.

- *Type 1 reaction (reversal):* either upgrading or downgrading immune state. It is often associated with neuritis. Constitutional symptoms are less common. It occurs mostly in borderline leprosy.
- *Type 2 reaction (Erythema nodosum leprosum [ENL]):* It is immune-complex reaction in patients with LL. There are tender nodules and constitutional symptoms (fever, arthritis).

Diagnosis of Leprosy:

- History and clinical picture.
- Smears (nasal and skin slit) examined by modified Ziehl-Neelsen (Fite) stain for bacteriological examination.
- Biopsy for histopathological examination.
- Lepromin test: to show the immune status of the patient.
- Histamine test and Pilorcarpine test: to detect dermal nerve damage in leprosy.

Treatment of leprosy:

It is based on the multidrug therapy strategy.

- First-line drugs: rifampicin, clofazimine, dapsone.
- Other drugs: ofloxacin, minocyline.
- For Paucibacillary leprosy (TT, BT): for 6 months
  a) Dapsone (bacteriostatic) 100 mg daily unsupervised.
  b) Rifampicin (bactericidal) 600 mg monthly supervised.

- For Multibacillary leprosy (BB, BL, LL): for 2 years
  a) Dapsone 100 mg daily unsupervised.
  b) Clofazimine 50 mg daily unsupervised.
  c) Rifampicin 600 mg monthly supervised.
  d) Clofazimine 300 mg monthly supervised.

❖ Atlas Fig. (27&28)

Questions

- Scrofuloderma can develop from the following:
  a. Lungs and lymph nodes.
  b. Bones and lymph nodes.
  c. Brain and lymph nodes.
  d. Gastro intestinal tract and lymph nodes.

- Hansen's disease is mainly a chronic inflammatory disease of the:
  a. Nervous tissue and the skin.
  b. Muscular tissue and the skin.
  c. connective tissue an d the skin.
  d. All of the above.
  e. None of the above.

- Lepromatous leprosy (LL) is usually associated with:
  a.Low level of cell mediated immunity.
  b. High level of cell mediated immunity.
  c. Cell mediated immunity has nothing to do with the development of LL.
  d. Only genetic back ground can determine the clinical form of leprosy.

- A 24 years old male presented with single well defined hypopigmented non itchy macule of one month duration over the fore arm. Sensations of heat, pain and touch were impaired. You have to suspect:
  a. Pityriasis versicolor.
  b. Vitiligo.
  c. Tuberculoid leprosy.
  d. Pityriasis alba.

VIRAL INFECTIONS OF THE SKIN

HERPES SIMPLEX

- It is a very common, acute, self-limiting vesicular eruption due to infection with Herpes virus hominis. Herpes simplex virus is highly contagious and is spread by direct contact with infected individuals.
- There are two types of herpes simplex virus:

a-Type I disease is usually facial.

b-Type II lesions are commonly genital.

Although this distinction is not absolute. Herpes simplex virus type I can be considered a causative agent for genital herpes due to oral sex. In addition, herpes simplex virus type II can cause herpetic gingivostomatitis.

- Recurrence is a hallmark of infection and occurs at a similar site each time.

Type I Herpes simplex:

- Incubation period: 2-5 days.
- Pathogenesis: After the first attack the virus remains latent in the root ganglion and upon reactivation by predisposing factors, the virus travels through the cutaneous nerves to the skin and manifests its clinical picture.

Clinical picture:

A- Primary attack:

i. Usually occurs in children. In a great majority, lesions are subclinical but when clinical attacks occur, vesicles are painful, often accompanied with fever, malaise and local lymphadenopathy. They do not show a tendency to grouping as in recurrent herpes simplex. They affect certain sites:

- Herpetic gingivostomatitis.
- Herpetic keratoconjunctivitis.
- Herpetic whitlow (fingers affection as in dentists)

B- Recurrent Herpes simplex:

i. Predisposing factors:

- Fever.
- Gastrointestinal tract disturbance.
- Psychological disturbance (e.g. in anxiety).
- Flu and upper respiratory tract infection.
- Exposure to sun.

- Mechanical trauma.
- Menstruation.

ii. C.P:

-Appears after reactivation due to exposure to precipitating factors

-It is presented by **group of vesicular eruption** on an erythematous base usually around the mouth. Burning sensation may precede or accompany the eruption. The vesicles are replaced by crusts in few days. Healing occurs in 5-7 days with normal skin.

iii. **Differential Diagnosis:**

- Impetigo.
- Herpes zoster.

Type II (Herpes progenitalis)

- It differs from type I by the following:
  i. Mode of transmission: sexually transmitted.
  ii. Site of affection: vulva, vagina and cervix in females. Glans and shaft of the penis, scrotum in males.

Clinical picture:

- As in Type I, it can occur as:
  A. Primary attack.
  B. Recurrent herpes progenitalis.
- Sexual partner should be examined.
- Special consideration should be taken for herpes progenitalis in pregnancy as primary attack when occurs few days before delivery can be dangerous to the fetus and indicates cesarean section but secondary attack in pregnancy usually doesn't affect the fetus.

Treatment:

Topical:

- Gentian violet 2 % paint.
- Acyclovir cream.

Systemic:

Antiviral drugs as Acyclovir 200mg 5 times/day/7-10 days.

VARICELLA (CHICKENPOX)

- It is the primary infection with varicella-zoster virus.
- Incubation period: 2-3 weeks.
- Mode of transmission:
  i. Droplet infection.
  ii. Direct contact.

The patient is infectious 4 days before the eruption until lesions are crusted.

Clinical picture:

- Prodromal stage of approximately 1-2 days shows fever, malaise, headache… etc.
- The eruption is itchy shows macules, papules, vesicles, pustules and crusts. The eruption is **polymorphic** (shows lesions at different evolutional stages) and **centripetal** in distribution (affecting mainly the trunk, face, scalp and neck) together with mucous membrane affection (eye, mouth, gingival). The extremities are less involved..
- Healing occurs within 10 days without sequelae, although it sometimes leaves postinflammatory pigmentation and scarring especially in adults.

Differential Diagnosis:

- Papular urticaria.
- Scabies.

Treatment:

- Topical:
  - Gentian violet 2 % paint.
  - Calamine lotion
- Systemic:
  - Antihistamines
  - Antiviral drugs.

HERPES ZOSTER

It is an acute, self-limiting, vesicular eruption, occurring in a dermatomal distribution. It is caused by reactivation of latent varicella-zoster virus present in dorsal root or cranial nerve ganglion that travels to the skin through cutaneous nerve.

Clinical picture:

1- Pain varies from mild to severe. It may accompany or usually it precedes the attack.

2- It is strictly unilateral, if bilateral or generalized it indicates that the patient is immunocompromized (e.g. internal malignancy, lymphoma, systemic corticosteroids, HIV infection).

3- The regional lymph nodes are usually enlarged and tender.

4- A group of vesicular eruption on an erythematous base appearing along the pathway of cutaneous nerve (dermatome). The vesicles contents dry up in 1-2 weeks forming crusts and healing usually by scarring.

- Common sites:
  - Thoracic affection.
  - Cervical affection (usually c 2, 3, 4)
  - Trigeminal affection (including ophthalmic division)
  - Lumbosacral affection

Complications of Herpes zoster:

- Post-herpetic neuralgia (persistence of pain after healing of cutaneous lesions).
- Secondary infection.
- Gangrene of the skin.
- Affection of the eye (herpes zoster ophthalmicus).

Differential Diagnosis: Herpes simplex

Treatment:

- **Topical**
  - Gentian violet 2 % paint.
  - Antiviral cream
- **Systemic:**
  - Antiviral drugs: Acyclovir 800mg/5times/7-10 days.
  - Pain killers are indicated whenever there is pain.

VIRAL WARTS (VERRUCAE)

Etiology:

Human papilloma viruses.

- **Verruca vulgaris (Ordinary warts):**

  Incubation period: 1-8 months.

  Mode of transmission: Direct contact or autoinoculation.

Clinical picture:

They appear as asymptomatic grayish papules which are few mm in diameters with verrucous (irregular) rough surface.

4 Viral Infections of the Skin

- **Plane warts:**

  - Usually affect children.
  - Multiple skin colored small papules with flat smooth surface.
  - Affect the face or extensor surface of hands.

- **Plantar warts:**

  - Affect the soles or in between the toes, usually against bony prominence, pain is variable

- ***Differential Diagnosis:*** callus which is hyperkeratotic area present in weight bearing area. Callus is crossed by skin creases

- **Genital warts:**

  - Appear as pedunculated grayish cauliflower mass called condyloma accuminata. or in the form of grayish small papules.
  - Affect the vulva, vagina and cervix in female and glans and penile shaft in men.
  - Sexual partner of genital warts patients should be examined and follow up of two years is required.

- ***Differential Diagnosis:*** Condyloma lata of syphilis should be differentiated.

- ***Complications:***

  - Condyloma accuminata can affect the cervix where it may be too huge and obliterate normal delivery necessitating Cesarean section.
  - Cervical intraepithelial neoplasm may develop which is a premalignant condition.

N.B.: Other morphological types of warts include digitiform and filiform warts.

Treatment of warts:

Warts may resolve spontaneously without trace and consequently treatment modalities that carry the risk of scarring should be avoided or used with care

- Chemical cautery:

  a- glacial acetic acid.

  b-salicylic acid 10%-60% in plantar warts.

  c-salicylic acid + lactic acid in flexible collodion.

- Electric cautery
- Cryotherapy with liquid nitrogen (-196°C)
- CO2 laser
- Topical keratolytics e.g. retinoic acid preparation in plane warts.
- Genital warts are treated by 25% podophyllin in tincture benzoin (contraindicated in pregnancy).

MOLLUSCUM CONTAGIOSUM (POX VIRUS)

Clinical picture:
Incubation period:-6-9 months

- Highly contagious lesions.
- The primary lesion is solitary, painless, shiny, pearly white papule, usually umblicated in the center.

Sites: any areas can be involved but more on the face, genitalias & trunk.

- Multiple lesions are usually present.

Treatment:

- Mechanical methods: Expressing the content of the papule by squeezing it with forceps or curettage followed by cauterizing the base.
- Concentrated phenol paint.
- Electric cautery.
- Cryotherapy.
- CO2 laser.
    - ❖ Atlas Fig. (29-34).

Questions

- Herpetic whitlow is a primary herpes simplex infection of:
    a. The finger nail fold.
    b. The crural area.
    c. The toe web space.
    d. The perinasal area.

- Chickenpox is:
    a. The primary infection with herpes simplex virus.
    b. The primary infection of varicella zoster virus.
    c. The secondary infection with herpes simplex virus.
    d. The secondary infection of varicella zoster virus.

PARASITIC INFESTATIONS

SCABIES

avoid pilosebaceous glands (scalp)

Etiology: Sarcoptes scabiei hominus.

Life cycle:

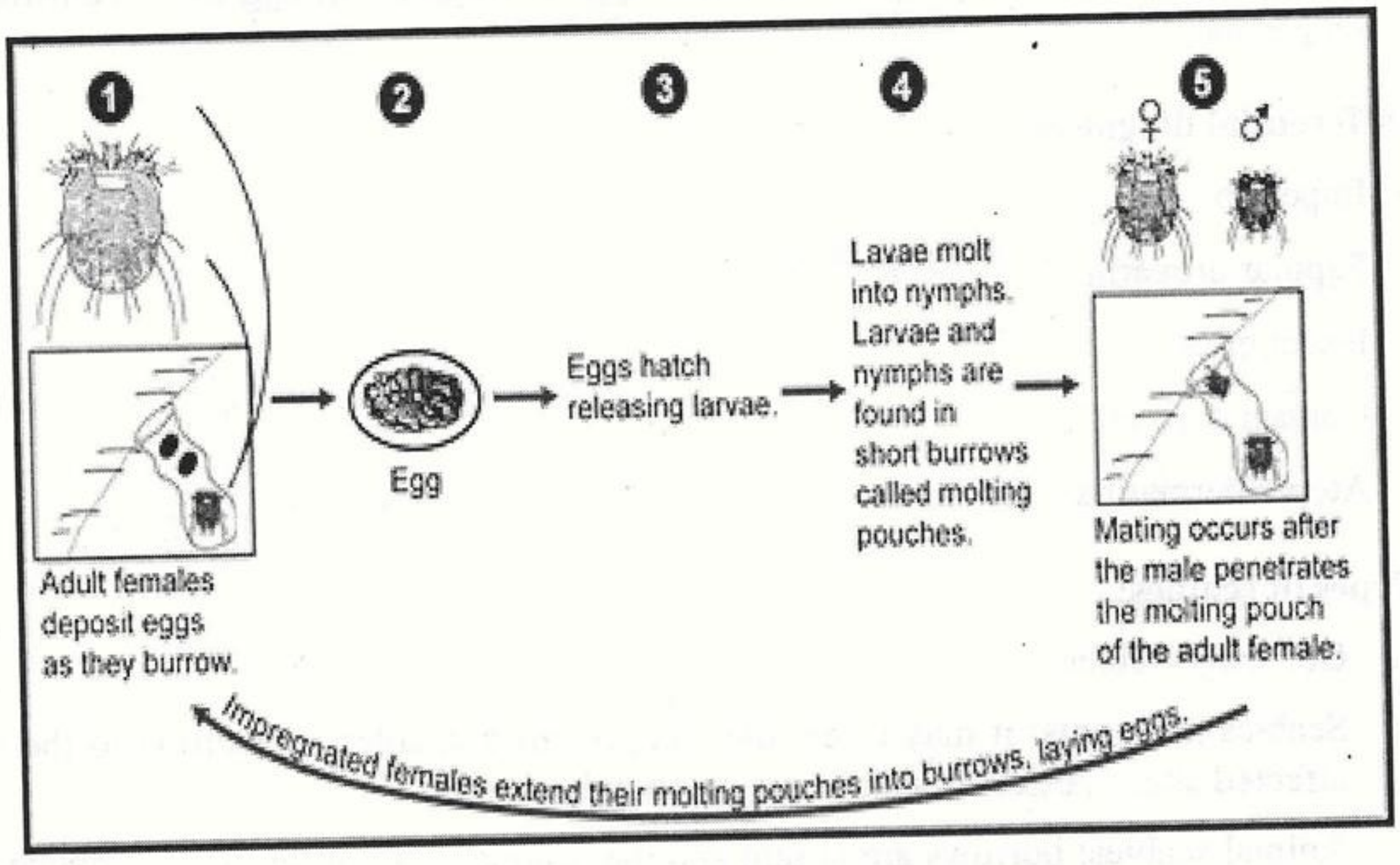


Fig. 6: Life cycle of Sarcoptes scabiei varity hominus

Predisposing Factors:

- Overcrowding and poor hygiene
- Wars, natural disasters

Mode of transmission:

- From human by close personal contact
- From animals (cats, dogs, rabbits, birds).

Clinical picture:

- Incubation period: 3-4 weeks
- Distribution: Hands , including interdigital areas, flexor aspects of wrists, extensor surfaces of elbows, anterior axillary folds, genitals, particularly the penis and scrotum, buttocks, abdomen specifically periumbilical area, extensor areas of knees, ankles and toes. In infants, all skin surfaces are susceptible including the scalp and face.
- Types of lesion: Pleomorphic mainly, burrows (characteristic lesion), papules, scales, vesicles, bullae, crusts, pustules, nodules and excoriation.

elevated tortous brownish lesion

Diagnosis:

- Scabies should always be suspected in the presence of any intensely nocturnal itching eruption that spares the face, head and interscapular area in the adult with typical clinical picture.
- Diagnosis is confirmed by gently scraping a burrow with a blunt scalpel, and the material placed in a drop of 10% potassium hydroxide or mineral oil on a microscopic slide. The presence of mites, eggs or fragment of egg shells confirms the diagnosis.

Differential diagnosis:

- Impetigo.
- Papular urticaria. (child) hypersensitivity to insect bite
- Insect bites (Adult)
- Contact dermatitis
- Atopic dermatitis

Types of scabies:

1. Ordinary scabies.
2. Scabies in infants: it may affect the face, palms and soles in addition to the usual affected sites .Vesicular lesions are commonly present.
3. Animal scabies: burrows are absent and the lesions occur at the site of contact with the animal. The condition is self limiting.
4. Scabies incognito: reduced signs and symptoms due to systemic administration or topical application of steroids.
5. Scabies in clean: burrows are hardly seen due to frequent bathing.
6. Nodular scabies: persistently pruritic nodules that remain for months after adequate scabies therapy. They are most commonly seen in genitalia, crural area and axillae. The nodules represent a continued hypersensitivity reaction and may respond to topical or intralesional corticosteroids.
7. Norwegian scabies (severe scabies, keratotic scabies): A highly contagious type that occurs in immune-compromised, physically disabled and mentally deficient individuals. It is characterized by heavily crusted skin lesions with thousands to millions of mites.

Complications:

- Post scabies pruritus: due to failure or inadequate treatment, reinfection, irritation from topical treatment or acarophobia.
- Secondary pyogenic infection.

Management:

-All members of the family should be treated.

-Clothing and bedding should be boiled. or iron them

-If secondary infection is present, it should be treated at first.

A-Topical application: All except sulfur can be used in Pediculosis

1-Sulfur : 5% for children and 10% for adults. 2.5% infants (in yellow soft paraffin)

neuralgia ↓ avoid after bath

2-Gamma benzene hexachloride 1% C.I in pregnant in child — for 4 successive nights after hot bath and hard brush

3-Permethrin 2.5% for children and 5% for adults.

4- Malathione 0.5%.

5-Crotamiton 10 %

6-Benzyl benzoate emulsion 25-33 %:

N.B: The only topical treatments used in pregnant females are sulfur and permethrin preparations.

B-Systemic therapy:

1. *Ivermectin.*
2. *Oral antihistamines.*

PEDICULOSIS (LICE INFESTATION)

- **Etiology:** Humans are parasitized by two species: Pediculus humanus and phthirus Pubis

I. **Pediculus Humanus:** Two subspecies are present which are pediculus humanus capitis and pediculus humanus corporis.

A. Pediculus Humanus capitis (head louse): start in occipital & parietal area of scalp

It is commonest among school children and spread by head to head contact or indirectly through combs and brushes. Spread of lice is encouraged by poor hygiene and overcrowding.

Clinical Picture:

- Scalp pruritus is the characteristic manifestation of head louse infection.
- The nits (lice eggs) are often easier to see than the lice. hatch after 8 days
- Secondary bacterial infection may occur (scalp impetigo) with enlarged draining cervical lymph nodes.

so off is. repeated after 8 days)

Treatment:

1. Treatment of pyogenic infection if present with systemic antibiotics at first.
2. Anti-pediculous agents:
   - Permethrin : 2.5% for children and 5% for adults.
   - Malathion : 0.5%.
   - Benzyl benzoate emulsion 25%.
   - Gamma Benzene hexachloride 1%.
3. Removal of remaining eggs with fine toothed comb +/- vinegar.

B. Pediculus humanus corporis (body or clothing louse):

- The natural habitat is the clothing of the host, and it only visits the skin to feed.

Clinical picture:

- Itching is the main complaint. It results from sensitization to louse salivary antigens. Asymptomatic cases occur in non-sensitized persons or in those with acquired tolerance to the bites.
- Lesions: mainly scratch marks, excoriations and hemorrhagic spots.
- Secondary bacterial infection may occur.

Treatment of pediculosis corporis:

- Ironing of the clothes is the most effective means of killing both lice and eggs.
- Antipediculosis lotions applied to the patient.

II. Phthirus pubis (Pubic or crab louse):

- The pubic or crab louse is quite distinct in appearance. It uses her huge claws to grip adjacent hairs close to the skin surface.
- It may colonize axillary hair, eyebrows, and eyelashes.
- Crab lice are transmitted by close physical contact, usually sexual.

Clinical picture:

- Severe itching in the pubic area is the principle symptom.
- Red spots in the under wears (parasite excreta).
- Close inspection of the affected areas will reveal lice grasping hairs close to the skin surface, and louse eggs attached to the hair shafts.

Treatment of phthirus pubis:

- Shaving the pubic area is the first step in the treatment.
- Malathion is effective against crab lice.

❖ Atlas Fig. (35-36).

Questions

- An adult with nocturnal itchy eruption that spares the interscapular area and the face should primarily give a suspicion of:
    a. Scabies.
    b. Papular urticaria.
    c. Atopic dermatitis.
    d. Body louse infestation.

- Persistent itching after adequate treatment of scabies may be due to :
    a. Post scabietic nodule.
    b. Re-infection.
    c. Hypersensitivity to anti scabietic locals.
    d. All of the above.

- Body louse habitat is the:
    a. Skin.
    b. Clothing fibers.
    c. Body hairs.
    d. All of the above.

FUNGAL INFECTIONS OF THE SKIN

Fungal infections of the skin are usually confined to the stratum corneum, but deep mycoses invade other tissues.

Fungal infection in human is common and mainly due to two main groups of fungi:

A. Dermatophytes: multicellular filaments (hyphae), reproduce by spores formation.

B. Yeasts: unicellular forms that replicate with budding.

Clinical presentation:

Tinea denotes a fungal skin infection which is often annular. The exact feature depends on the site:

1. Affection of the scalp (Tinea capitis).
2. Affection of the beard (Tinea barbae).
3. Affection of the glabrous skin (Tinea corporis).
4. Affection of the genitocrural area (Tinea cruris).
5. Macerated web space [Tinea pedis (feet) and manum (hand)].
6. Affection of nails (Onychomycosis).

TINEA CAPITIS (RINGWORM OF THE SCALP):

Sources of infection: humans, animals such as (dogs, cats, sheep) and rarely soil.

Tinea capitis commonly affect children more than adults (except favus which may also affect adults), this could be explained by the fungistatic effect of sebum produced by the androgen dependant sebaceous glands by the age of puberty.

Tinea capitis has **four clinical types**:

A. ***Scaly ring worm*. Trichophyton violaceum and Microsporum canis** are the most common organisms responsible for this condition. It presents as single or multiple scaly patches with the hairs cut short. Healing is complete and occurs without scarring.

B. ***Black dot ring worm*. Trichophyton violaceum** is the causative fungus. This condition is similar to scaly ring worm, however the hair is broken off shortly after it erupts from the scalp and so appears as black stumps (hence the name). Scaling is mild and healing occurs also without scarring.

C. ***Kerion.*** This represents a severe inflammatory reaction to the invading fungi (mostly of animal origin i.e. zoophilic as **Trichophyton verrucosum** and **Trichophyton mentagrophytes**). The condition presents as a single sometimes multiple painless swellings of the scalp. The hair is easily detached and the surface of the swelling is studded with few pustules. Regional lymph nodes are not enlarged with no affection of the general condition of the patient. The condition should be differentiated from acute pyogenic abscess and incision is contraindicated. It heals with scarring alopecia if neglected or with normal regrowth of hair if treated early.

D. ***Favus***.. Due to infection by **Trichophyton schoenleinii**. There is a diffuse involvement of the scalp hair. Affected hair is surrounded by thick yellow adherent crust that is characteristically cup shaped (sulfur cups or scutula). Slow extension of the lesion may occur to involve the entire scalp. Unlike other fungal infections of the scalp, healing occurs by scar formation and permanent alopecia with scattered "coconut hairs" in it.

Differential diagnosis:

1. Other causes of scaly scalp as: Seborrheic dermatitis, impetigo, psoriasis, are the most common differential diagnoses.
2. Causes of localized non cicatricial alopecia: alopecia areata, trichotillomania.

TINEA BARBAE:

The lesion is similar to kerion but it affects beard hair. The angle of the jaw is the commonest site affected.

TINEA CORPORIS (trunk and limbs)**:**

It presents as a severely itchy circinate lesion i.e., healing center and active raised edge showing minute papules, pustules and crusts. The edge is the recommended site for scraping or biopsy.

Differential diagnosis

1. Circinate impetigo (vesicles are larger, gummy crusts are present, non itchy and negative direct microscopic examination).
2. Pityriasis rosea (the edge is not raised, the scales are forming a collarette and negative direct microscopic examination).

TINEA CRURIS (groin):

It is caused by **Epidermophyton floccosum** and **Trichophyton rubrum**. It is more common in adult males. It is a well defined erythematous patch with a raised active edge affecting the upper medial side of the thigh but may extend to involve the buttocks and pubic area.

Differential diagnosis

1. Erythrasma
   a. Caused by corynebacterium minutissimum.
   b. The lesion is well defined brownish and scaly, with no active edge.
   c. Treatment is with systemic erythromycin and topical broad-spectrum antifungal agents, e.g., clotrimazol.
   d. Wood's light examination: coral red fluorescence.
2. Candidiasis of the groin.
3. Intertrigo.
4. Psoriasis (flexural).

5. Seborrheic dermatitis.

FUNGAL INFECTION OF THE NAIL PLATE (ONYCHOMYCOSIS):

This could be due to infection by dermatophytes, candida or moulds. The infection starts laterally or at the free margin of the nail and is facilitated by trauma, diabetes and moisture. Affected nails appear lusterless, brittle and friable with evidence of subungual hyperkeratosis.

Differential diagnosis

Psoriasis of the nail and paronychia due to candida and bacteria are the common differential diagnoses.

N.B. **Tinea incognito**: Fungal infection modified in appearance and spread (less erythematous and the active edge becomes less prominent) by the anti-inflammatory effect of topical or systemic steroids.

TINEA PEDIS (Athletes foot): it is common in adults especially young men and is often predisposed to by communal washing, swimming baths, occlusive footwear and hot weather.

Clinical variants

- Chronic interdigital (macerated toe web) which is the most common presentation . Itchy interdigital maceration usually of the 4^{th} / 5^{th} toe-web space.
- Squamous hyperkeratotic (chronic scaly).
- Acute vesiculobullous. The latter 2 conditions are less common presentations.

Etiology

Macerated web space can be a manifestation of one or more of the following:

- Dermatophytes.
- Candida.
- Gram negative bacteria.

PITYRIASIS VERSICOLOR (PITYROSPORUM OVALE INFECTION):

- Very common mild chronic infection of the skin affecting adults and rarely children.
- Etiology: Malassezia yeasts which are normal commensals of human skin.
- In most cases, the condition represents a shift in the relationship between the human and his resident yeast flora (Pityrosporum ovale).
- Factors contributing to this change are mainly excessive sweating and humidity.
- Well-defined hyper- and hypo-pigmented macules and patches covered by cigarette paper-like scales.
- It affects mainly the trunk and neck; less commonly the face and scalp.
- Wood's light examination gives a characteristic golden yellow fluorescence.
- Recurrence rate after treatment is very high.

CUTANEOUS CANDIDAL INFECTIONS:

It is a common condition caused by the yeast candida albicans (is a ubiquitous commensal of the mouth and gastrointestinal tract and can produce opportunistic infection). Predisposing factors diabetes, obesity, moisture, immunocompromised individuals, prolonged intake of broad spectrum antibiotics and old individuals. It may present as

A. ***Oral candidiasis (oral thrush):*** Clinically, whitish curd-like patches (pseudomembrane) on tongue and buccal mucosa characterize it. The patches can be scrubbed and leave erythematous base. Angular stomatitis (perleche) is commonly associated.

B. ***Genital candidiasis:*** It may affect the vagina in females (candidal vaginitis) and the glans penis in males (candidal balanitis). It is also one of the sexually transmitted diseases.

C. ***Napkin dermatitis (Diaper rash):*** This is a common condition that affects young infants and presents as ill defined erythematous lesions showing superficial pustules on the edge and satellite lesions.

D. ***Candidal intertrigo:*** It affects the groin, axillae and submammary regions. The lesion is well defined, moist and erythematous. The edge is festooned and small satellite lesions (papules & pustules) are seen outside the edge. Also the interdigital clefts are involved in wetworkers who do not dry their hands properly.

E. ***Chronic mucocutaneous candidiasis:*** It is a rare condition and is due to a state of immune deficiency against candidal organisms. It is characterized by chronic paronychia, oral candidiasis, hyperkeratotic lesions on hands and feet and subungual hyperkeratosis.

F. ***Candidal paronychia:*** see page (19).

Diagnosis of fungal infection:

1. ***Direct microscopy:*** Scrapings from the active edge of the lesion is collected on a glass slide and a drop of KOH (10-20%) is added. Gentle warming (avoid excessive heat and boiling) will facilitate examination. The specimen is then examined by light microscope, which shows, in positive cases, hyphae and spores.
2. ***Culture:*** This step not only confirms the presence of fungi but also specifies the causative fungus (by morphology of the colonies and microscopic appearance). Dermatophytes are cultured on Sabouraud's agar. They need 2-4 weeks to grow.
3. ***Wood's light examination:*** it is used for tinea capitis especially for screening during outbreaks (such as school children and army barracks). Hair infected with Microsporum canis and Microsporum audounii show green fluorescence but Trichophyton violaceum does not fluoresce.

Treatment of superficial fungal infections:

Avoid predisposing factors: humid and sweaty conditions including occlusive footwear should be minimized.

Minor fungal infections respond to topical treatment but widespread involvement or affection of the nails or scalp requires systemic therapy.

- Topical therapy:

a. Tincture iodine 2-5%
b. Whitfield ointment (Salicylic acid 3, Benzoic acid 6, Lanolin 10 and Vaseline 100).
c. Broad spectrum topical antifungals e.g., Imidazol group of drugs, such as Clotrimazole, Econazole, Ketoconazole, fluconazole etc.
d. Topical aqueous solution of sodium hyposulphite (20-25%) for pityriasis versicolor.

- Systemic therapy:

- **Griseofulvin** is an oral fungistatic agent that is readily absorbed from the gastrointestinal tract, particularly if it is taken after a fatty meal. It is deposited in the keratin and hair and prevents further hair invasion.

  Dose: 12.5 mg/kg/day. Each tablet contains 125 mg and the maximum allowed dose is six tablets per day (i.e. a 30 kg patient would receive 3 tabs/day, whereas a 90 kg patient would receive only 6 tabs/day).

  Duration of treatment: is 6-8 weeks in tinea capitis and it is usually extended to 8-10 weeks in favus. In tinea corporis and tinea circinata, the duration of treatment is 2-4 weeks while it is 6 months in onychomycosis (finger nail) and 12 month (toe nail).

  Side effects: gastrointestinal disturbances, headache, photosensitivity, drug induced SLE.

Contraindications: in pregnancy and in patients with hepatic failure or photosensitivity.

The drug is not effective in cases of candidiasis and pityriasis versicolor.

Systemic broad spectrum antifungals e.g. ketoconazole, itraconazole may be used in severe and extensive cases.

Treatment of candidal infections:

- Topical antifungal agents e.g. imidazoles.
- Systemic treatment in severe, extensive and resistant cases. It is in the form of broad spectrum antifungal agents e.g. itraconazole (Griseofulvin is not effective).

❖ Atlas Fig. (37-42).

Questions

- Match the following diagnoses with the appropriate region affected:

1. Tinea cruris.	a. Genital folds.
2. Tinea capitis.	b. Nails.
3. Tinea corporis.	c. Glabrous skin.
4. Onychomycosis.	d. Beard.
5. Tinea barbae.	e. Foot.
6. Tinea pedis.	f. Hand.
7. Tinea manum.	g. Scalp.

ALLERGIC SKIN DISEASES

ECZEMA (DERMATITIS)

Eczema is a non-infective inflammatory condition of the skin characterized clinically by ill defined erythematous patches, papulo-vesicular eruption with exudation of serum in its acute stage, erythema and scaling in its subacute stage and dryness and lichenification in the chronic stage.

Classification:

Eczema represents a reaction pattern to a variety of stimuli.

Endogenous eczema

- Atopic dermatitis
- Discoid eczema
- Pityriasis alba
- Stasis dermatitis.
- Seborrhoeic dermatitis.

Exogenous eczema

- Contact dermatitis.

Contact Dermatitis

Dermatitis precipitated by an exogenous agent, often a chemical, is known as contact dermatitis.

Irritant contact dermatitis:

- Irritants either physical or chemical cause dermatitis by a direct noxious effect on the skin barrier function.
- The most important irritants are acids, alkalis [cement, organic solvents (detergents)].
- Irritants cause more cases of contact dermatitis than do allergens, although the clinical appearance are often similar.

Allergic contact dermatitis (Sensitization dermatitis):

Allergic contact dermatitis is an example of type IV hypersensitivity reaction and is diagnosed by patch test.

Common allergens *(usually incomplete antigens i.e. haptens)*:

- Chemicals e.g. Dyes, nickel, cement, detergents etc.
- Cosmetics: Lipsticks, nail polish, hair dyes, eyeliners, depilators, etc.

- Locally applied drugs e.g. neomycin and sulfonamides.
- Textiles: Nylon, wool, rubber, etc.

Atopic Dermatitis

A chronic pruritic inflammation of the skin often associated with a personal or family history of allergy e.g. urticaria, asthma, allergic rhinitis, or conjunctivitis. Uncontrollable scratching is prominent and the course is remittent.

Pathogenesis:

- Genetic factors.
- Environmental factors e.g. pollution, microbes.
- Immune dysregulation:
  - **TH2** cells predominance in acute phase.
  - **TH1** cells predominance in chronic phase.

Clinical picture:

Itching is the constant symptom. The distribution and morphology of the lesions vary with age.

Infantile phase:

- The lesion s most frequently start on the face.
- They consist of erythematous discrete or confluent edematous papules. The papules are intensely itchy and may become exudative and crusted. Secondary infection and lymphadenopathy are common.

Childhood phase:

The sites mostly involved are the elbows and knee flexures, sides of the neck, wrists and ankles and the hands. Lesions consist of erythematous, edematous papules which tend to be replaced by lichenification, vesiculation may occur, often in discoid patches.

Adulthood phase (neurodermatitis):

- Lesions consist of lichenified plaques.
- Sites most commonly affected are outer arms, upper back, and shin of the tibia.

Discoid Eczema

- It is characterized by circular or oval coin shaped lesions with a demarcated edge.
- It is more common in adults on the extensor aspects of limbs; usually the lesions are bilateral and symmetrical.

Varicose Eczema (Stasis Dermatitis)

- There is increase venous pressure in the lower limbs e.g. varicose veins.
- Lesion: Present on the lower part of legs as an erythematous scaly oozing area surrounded by small slate blue macules resulting from hemosiderin deposits.
- Ulceration occurring around one of malleoli usually complicates the condition.

Seborrhoeic dermatitis

- It is a scaly erythematous dermatitis associated with seborrhea, abnormal sebum composition and the lipophilic yeast Malassezia.
- **Clinical picture**

  Infantile SD:

  - From 2 weeks to 10 months.
  - Greasy yellowish scales on erythematous area usually on the scalp, diaper area and intertriginous folds.

  Adult SD:

  - Diffuse or localized scaly erythematous patches involving hairy areas and body flexures.

Infective Eczematous Dermatitis

- It affects areas of skin around a discharging focus e.g. discharging ear or nose.
- Lesion is characterized by area of erythematous patches with exudation and crustation.

Dyshydrotic Eczema (Pompholyx)

- It is an acute or subacute vesicular eruption starting usually on the sides of the fingers and toes and extending to soles by deep-seated vesicles. The vesicles tend to dry up in two weeks with desquamation of the skin. Itching is very severe.
- It occurs more commonly in heavily perspiring individuals and it may develop as an allergic reaction to a distant focus of infection e.g. tinea pedis.

Pityriasis Alba

- It is a chronic eczema of unknown origin, but mostly related to environmental factors. Pityriasis alba usually occurs in children. It is sometimes a manifestation of atopic dermatitis.
- The lesion appears as rounded, oval patch, or erythematous plaques with fine lamellar or branny whitish scaling. Later it subsides to leave areas of scaling and

hypopigmentation. The most common site is the face, the course of pityriasis alba is very variable. Recurrent crops of new lesions may develop at intervals.

Treatment of Eczema

1- Systemic:

i) Antihistaminic to alleviate itching.

ii) Antibiotics if there is secondary infection.

iii) Corticosteroids are indicated in severe generalized forms.

2- Local:

Local treatment of eczema depends on the clinical stage of the lesion whether acute, subacute or chronic almost irrespective of the type of eczema.

i. Acute eczema: Compresses as
   a. potassium permanganate solution 1/8000
   b. 1/10.000 of lead subacetate solution 0.5% for oozing cases in addition to a corticosteroid cream.
ii. In subacute eczema: Corticosteroid creams or zinc oxide paste.
iii. In chronic eczema: Topical steroid preparation of variable strengths.
iv. As a whole the best line of treatment of eczema is to try to find a cause and avoid it.
v. In cases of atopic dermatitis, long term use of emollients is indicated.

URTICARIA AND ANGIOEDEMA

Urticaria is a vascular reaction of the skin in which transient (evanescent) erythematous swellings of the dermis or subcutaneous tissues (wheals) occur. It is accompanied by severe itching. It may be acute or chronic if it is more than 6 weeks duration.

Aetiopathogenesis:

- Wheals result from mast cell degranulation that leads to release of proinflammatory mediators and vasodilator substances e.g. **histamine**, **TNF, interleukins (IL) 3, 4, 5, 6, 8 & 13,** causing vasodilatation and increased permeability of the blood vessels of upper dermis. Vasodilatation results in erythema while increased permeability leads to localized dermal edema.
- Urticaria is mediated through immune (allergic) or non immune (non allergic) mechanism
  - *I. **Immunologic (Allergic cause):*** classic type I hypersensitivity reaction which involves binding of receptor bound specific IgE to allergen releasing vasodilating substance or substances. The antigen may be: Inhalant, ingestant, injectant, infectant or infestant.

II. Non immunologic (Non-allergic cause): Release of the vasodilator substances does not result from antigen antibody reaction. The exact mechanism is not yet fully understood. Non allergic urticaria may be caused by:

1. Drugs as aspirin or morphine, chemicals as food additives.
2. Physical agents as heat, cold, sun rays.
3. Psychogenic factors as anxiety and emotional upset.

Clinical picture:

- Itching is severe.
- Onset is sudden; wheals of various sizes appear and may involve any part of the skin.
- The lesion usually subsides within a few hours and new lesions may appear.

Clinical presentation:

1. Chronic idiopathic urticaria, the condition resolves spontaneously within few months.
2. Acute urticaria.
3. Physical and cholinergic urticaria:
   a. Due to mechanical factor e.g. dermographism.
   b. Due to temperature changes e.g. cold urticaria.
   c. Solar uricaria.
   d. Aquagenic urticaria.
4. Urticarial vasculitis due to type III hypersensitivity reaction.
5. Allergic contact urticaria: hypersensitivity IV response diagnosed by patch testing.

   Associations :

   Chronic urticaria has been associated with autoimmune thyroid disease, vitiligo and insulin dependant diabetes. An association between Helicobacter pylori gastritis and chronic urticaria has also been proposed.

Angioedema:

Recurrent angioedema without wheals is a special type of urticaria of sudden onset involving the subcutaneous tissue leading to edema of the eyelids, lips, hands and genitalia. It may affect the laryngeal mucosa and is then life-threatening. It will be then associated with respiratory distress and hypotension with laryngeal edema, and shock secondary to peripheral vasodilatation. Death may occur within minutes.

Treatment of urticaria:

Causative or provoking factors should be eliminated.

- Adrenaline 1/1000 1cc subcutaneously (contraindicated in hypertension and heart disease).
- Antihistaminics:

- Histamine type 1 receptor blockers (H1 blockers): reduces the wheal size and itching. Side effect is drowsiness.
- 2nd and 3rd generation non sedating antihistaminics.
- Addition of H2 antagonists to conventional H1 antihistaminics may be helpful.

- Calcium gluconate 10%, 10 cc slow intravenous injection.
- Systemic steroids: indicated in sever acute urticaria and urticarial vasculitis.
- Treatment is only complete when the cause is found and avoided.

PAPULAR URTICARIA

It is a chronic and recurrent eruption of irritable edematous papules affecting predominantly infants and children. It is due to hypersensitivity to insect bites.

Clinical picture:

- Age: Infants and children.
- Distribution: Extensor surfaces of the limbs and trunk.
- Lesions: Initially, an extremely itchy edematous papule develops at the site of the insect bite and this is followed by a firm pruritic papule which usually persists for several days. Lesions are often grouped in clusters and develop in crops at irregular intervals and may vesiculate. Lesions are accompanied by scratch marks and may be complicated by secondary infection.

Treatment:

- Avoid insect bites and use insect repellant.
- Antihistaminics.
- Short term oral steroids for extensive and spreading lesions.
- Antibiotics in secondary infection.
- Soothing lotion as calamine lotion or other topical antipruritic preparations.

PRURIGO OF HEBRA

It is a chronic itchy skin condition that usually commences in childhood that is characterized by the presence of itchy papules. Lichenification and scratch marks are usually associated with superficial lymphadenopathy.

Etiology:

- Some cases represent papular urticaria modified in an atopic subject as an allergy to insect bites.
- Environmental factors as many cases are improved by changing the environment. The role of poor nutritional and hygienic condition should be emphasized.

Clinical picture:

- The condition usually affects young adolescents (9-14 years). It may be preceded by papular urticaria.
- Distribution: The extremities are affected more than the trunk, the extensor surface more than the flexors and the distal parts more than the proximal. Cubital and popliteal fossae are usually spared.
- Lesion: Consists of a combination of:
  - Itchy papules which are better felt than seen. Itching may be severe and papules may be topped by minute vesicles.
  - Lichenification.
  - .Scratch marks.
- Superficial lymph nodes: As supratrochlear and inguinal groups are usually enlarged. The enlargement is not due to secondary infection.

Treatment:

- Change of the environment e.g. hospitalization usually ameliorates the condition even without treatment.
- Systemic antihistaminics.
- Locally anti-pruritic remedies e.g. zinc oxide paste.
- Topical steroids.
- Nourishing diet and vitamins.
- Antibiotics for secondary infection.

ERYTHEMA MULTIFORME (EM)

It is an acute self-limited inflammatory skin condition characterized by sudden onset of symmetrical multiform lesions including erythematous macules, papules vesicles and sometimes bullae. Some of the lesions evolve into *target* or *iris lesion* which is considered to be the hallmark of the eruption.

Etiology:

Erythema multiform is a reactive pattern to many different stimuli. It is an immune-mediated disorder: EM therefore represents a hypersensitivity reaction to:

1. Infection usually viral e.g., herpes simplex (most common cause), Orf virus and rarely Epstein Barr virus. Mycoplasma pneumoniae may be associated with EM.
2. Drugs, e.g., penicillin and sulpha.
3. Other factors, e.g., vaccination, malignant disease, exposure to radiation or chemicals.

4. Idiopathic.

Clinical picture:

- Erythematous, slightly edematous macules, papules, plaques and sometimes vesiculobullous or purpuric eruptions. The lesions appear in successive crops at intervals for few days and fade in 1-2 weeks. The eruption is usually bilateral and symmetrical with tendency to recurrences.
- Sites: dorsum of the hands, palms, wrists, forearms, feet, elbows and knees are affected. Less commonly the face, neck and trunk could be affected. The characteristic lesion is the *target or iris lesion* which is a circular erythematous lesion with a vesicle or papule in its center. The center is darker in color thus resembling the iris of the eye.

Treatment:

- Symptomatic treatment is sufficient for the usual attack.
- Oral antihistaminics for few days reduce the stinging and burning of the skin.
- Short term oral steroids is helpful in moderate to severe cases.
- Oral acyclovir at 10 mg/kg/d for 6-12 weeks may be considered as a prophylactic treatment for HSV associated with frequent recurrences.

Steven's Johnson Syndrome

- SJS is a rare serious disorder in which the skin and mucous membranes react severely to a medication or infection. Often SJS begins with flu like symptoms followed by a painful red or purplish rash that spreads and blisters eventually causing shedding of the top layers of the skin.

Etiology:

The exact cause of SJS cannot always be identified. Usually the condition is an allergic reaction in response to medication, infection or illness.

- There is marked constitutional symptoms of high fever, malaise, and arthralgia.
- The onset is sudden with extensive typical maculopapular rash as well as generalized bullous eruption with affection of oral mucus membrane. This is followed by eruption of new crops of lesions develop over a period of 3-4 weeks. The eyes show a severe catarrhal or purulent conjunctivitis.

Complications:

- Secondary skin infection (cellulitis).
- Sepsis.
- Eye problems eg: inflammation and irritation of the eyes leading to extensive tissue damage and scarring that ultimately may lead to blindness.
- Damage to internal organs eg: pneumonitis, myocarditis, nephritis and hepatitis.

- Death may occur in 5-15% of untreated cases.

Treatment

- Admission to an intensive care unit results in a significant reduction in morbidity and mortality.
- Treatment of the cause and discontinuation of the offending drugs.
- Systemic steroids may help, intravenous immunoglobulins are tried with beneficial effect.

ERYTHEMA NODOSUM (EN)

It is a type of hypersensitivity reaction to a wide variety of provoking agents.

Etiology:

- Streptococcal infection e.g. throat infection.
- Tuberculosis.
- Sarcoidosis and Chlamydia.
- Viruses e.g. hepatitis B viruses and infectious mononucleosis.
- Deep fungal infection.
- Drugs: Sulphonamides and oral contraceptives.
- Enteropathies e.g. ulcerative colitis.
- Malignant diseases.
- Leprosy e.g. erythema nodosum leprosum representing acute panniculitis of leprosy.
- Idiopathic

Clinical features:

- Most patients have fever, general aching and malaise.
- There are multiple bilateral, tender, warm, erythematous nodules (may be ecchymotic) mainly on the anterior aspects of the lower limbs.

Treatment:

- Rest in bed, analgesics and antibiotics.
- Acetyl salicylic acid.
- Systemic steroids in some cases.
- Treatment of the actual cause.

DRUG ERUPTIONS

They are unwanted cutaneous or mucocutaneous reactions to administration of drugs. Drug reactions may simulate any skin disorder. Cutaneous reactions to topically applied drugs are considered examples of contact dermatitis rather than drug eruptions.

Diagnosis:

- History of drug intake.
- Drug eruptions may occur after the first dosage, during the course of therapy or few days after stoppage of the drug.
- The eruption usually occurs suddenly, is symmetrical and generalized. It may be associated with systemic symptoms as fever. Drug eruptions improve when the suspected drugs are stopped and recur on re-administration.

Fixed Drug Eruption (FDE):

It is a specific entity of skin reaction to drugs where the eruption recurs on the same site or sites each time the drug is administered.

Clinical picture:

- The eruption is sharply marginated rounded or oval erythematous patches which becomes dusky violaceous in color.
- Sometimes bullae develop on top and when rupture they may leave superficial erosions. On healing FDE leaves permanent slate blue discoloration. The commonest sites to be involved are the penis, lips and dorsum of the hands; however any site can be affected including the mucous membrane of the mouth. Examples of drugs which usually produce FDE are **sulfonamides and analgesics.**

Diagnosis of FDE:

The probable points that can diagnose FDE are:

- History of drug intake.
- Recurrence of the lesion in the same site each time the drug is administered.
- The violaceous erythema during the active change.
- The slate blue pigmentation.

Treatment:

- Stop the drug.
- Emollient and topical steroids.
    - ❖ Atlas Fig (43-51).

Questions

- Acute eczema is characterized by:
  a. Crust .
  b. Oozing.
  c. Ill defined borders.
  d. All of the above.
  e. None of the above.

- Chronic eczema is characterized by:
  a. Dryness and lichenification
  b. Oozing.
  c. Erythema and scaleness.
  d. All of the above.
  e. None of the above.

- Wheals are :
  a. Evanescent skin lesions.
  b. Dermal swellings.
  c. Related to urticaria.
  d. All of the above.
  e. None of the above.

- Lymph nodes in prurigo of hebra are:
  a. Enlarged when 2ry infection occurs.
  b. Enlarged regardless 2ry infection.
  c. Usually not affected.
  d. Only deep lymph nodes are affected.

DISEASES OF SWEAT AND SEBACEOUS GLANDS

SWEAT GLANDS

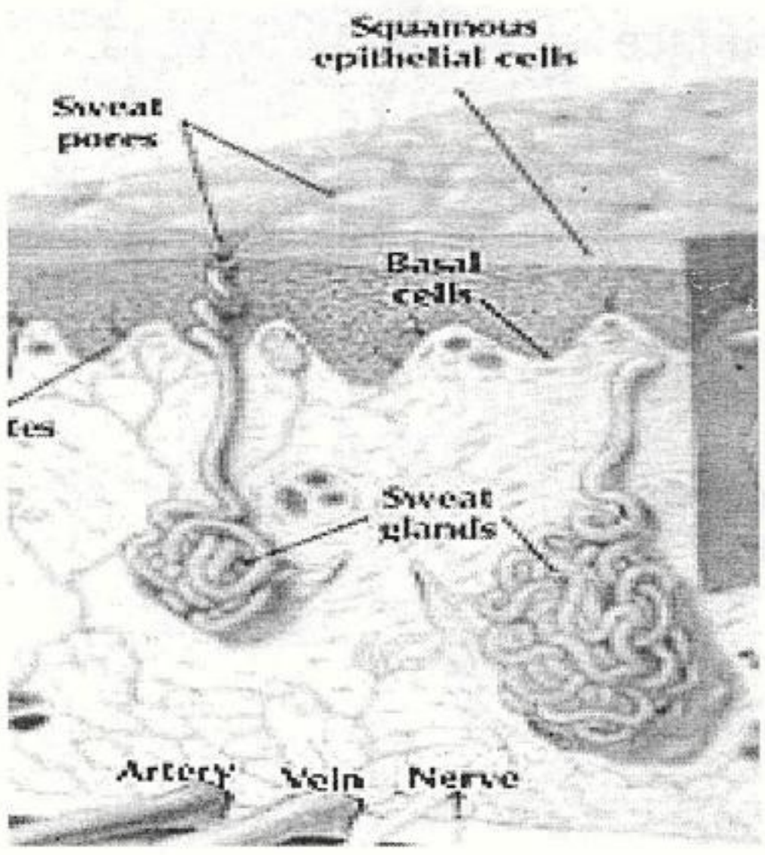


Fig. 7: The sweat glands.

- Eccrine sweat glands: see p.3 (Fig.7).

MILIARIA (SWEAT RASH)

- It occurs in warm climates, while warming in an incubator, during an attack of fever, or from wearing occlusive dressings or warm clothing.
- Eccrine sweat-duct occlusion is the initial event. The duct ruptures, leaks sweat into the surrounding tissues, and induces an inflammatory response. According to the level of occlusion, distinct form of miliaria develops:
  1. **Miliaria crystallina:** occlusion of the eccrine duct at the skin surface resulting in accumulation of sweat under the stratum corneum. The lesion appears as a clear drop or vesicles with little or no erythema.
  2. **Miliaria rubra:** results from occlusion of the intraepidermal part of the eccrine sweat duct. Papules and vesicles surrounded by a red halo or diffuse erythema develop as the inflammatory response occurs.
  3. **Miliaria profunda:** results from occlusion of the eccrine sweat duct at the dermoepidermal junction.

Unless secondarily infected **(miliaria pustulosa)**, cold water compresses and proper ventilation are all that is necessary to treat this self-limited process.

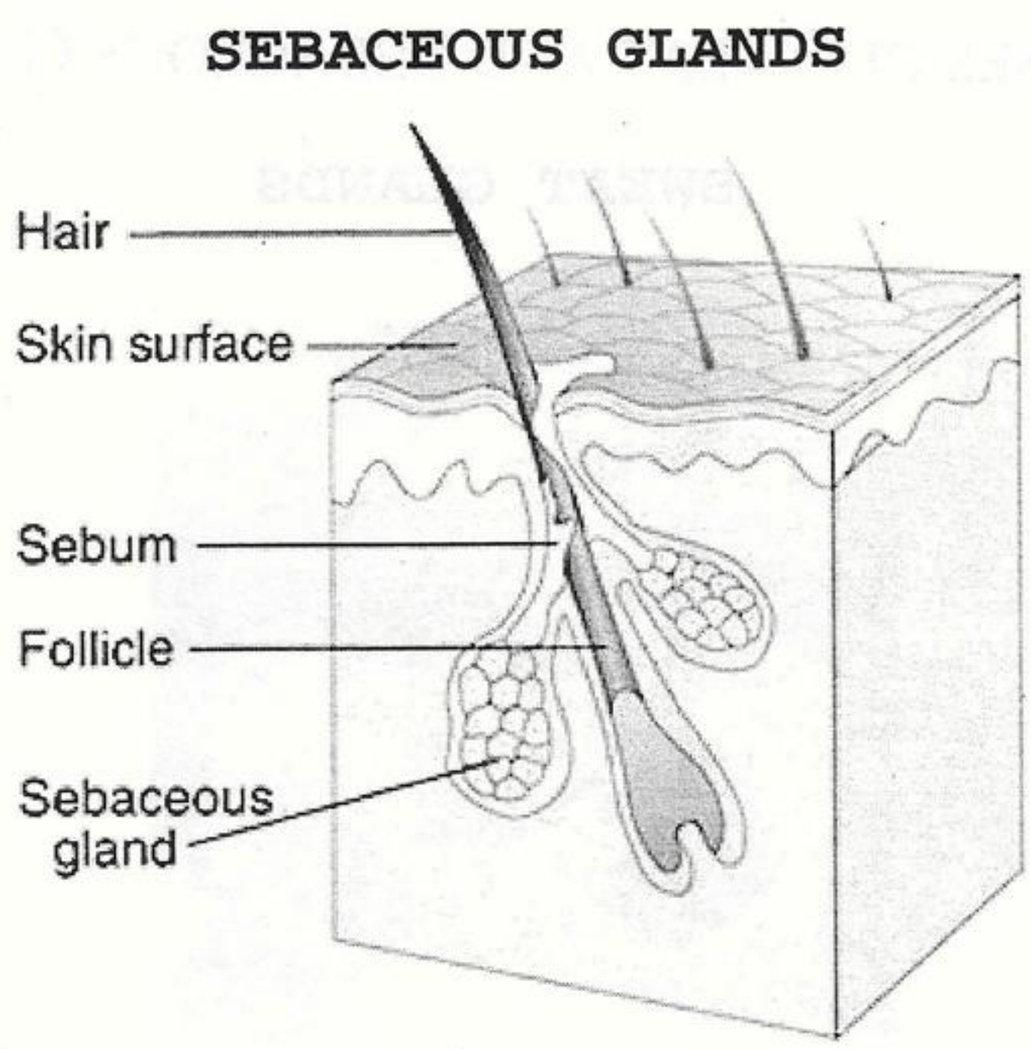


Fig. 8: The sebaceous glands.

- Sebaceous glands are **holocrine glands**; secretion is formed by complete disintegration of glandular cells.
- They are associated with hair follicles **(pilosebaceous units).** Sebaceous glands consist of a series of lobes, each with a duct lined by a keratinizing squamous epithelium. The ducts then converge towards the main sebaceous duct, which normally opens into the pilary canal, whose epithelium is continuous with the surface epidermis (Fig.8).
- In children, the glands are small but become large and active at puberty, being sensitive to androgens.
- In the face, chest and back; the pilosebaceous units are called "**sebaceous hair follicle**"; characterized by voluminous hyperactive sebaceous glands and weak vellus hair.
- The gland secretes sebum. Sebum has both bacteriostatic and fungistatic action.

ACNE VULGARIS

Acne is a chronic inflammation of the sebaceous hair follicles.

Pathogenesis:

- Acne results from:
  1. Increased sebum production.
  2. Pilosebaceous duct cornification.
  3. Colonization of the duct by Propionibacterium acnes (P.acnes).
  4. Release of inflammatory mediators.
- In acne, the androgen-sensitive sebaceous hair follicles show a hyper-responsiveness that result in increased sebum excretion. Sebum is deficient in leinoleic acid (a fatty acid important for the normal cornification of cells of the pilosebaceous follicle). Accordingly, hyperkeratosis of the pilosebaceous duct occurs with obstruction of the pathway of sebum. These result in the occurrence of the primary lesions of acne, "**comedones**". They are either open (blackheads) or closed (whiteheads).
- P.acnes are responsible for the inflammatory process of acne vulgaris (papules-nodules-pustules). They are anaerobic organisms that increase abruptly at puberty. They secrete many chemical mediators, the most important of which is lipase.
- Lipase is responsible for:
  1. Degradation of sebum triglycerides to free fatty acids and glycerol. Free fatty acids are irritant, adding more & more to the problems of hyperkeratosis during their pathway along the hair shaft.
  2. Chemoattraction for inflammatory cells.

Clinical picture:

- Age: starts at puberty.
- Sex: both sexes are equally affected.
- Sites: face, chest and back.
- Lesions are pleomorphic: comedones, papules, pustules, nodules and cysts. Post acne scars may also occur.

Treatment

Local treatment is adequate for mild acne and is used with systemic drugs for more severe cases.

Local therapy:

1) Peeling agents as 2% sulfur ppt. in calamine lotion, 3% salicylic acid lotion or 0.05% retinoic acid.
2) Antibiotics: erythromycin, clindamycin.
3) Benzoyl peroxide which changes the medium around the organism (P. acnes) from anaerobic to aerobic by production of hydrogen peroxide.
4) Topical azaleic acid: It can control the cornification of the ducts and has an anti-inflammatory effect.

Systemic treatment:

1) **Antibiotics**:
   a. The first-line systemic antibiotic drug is tetracycline, 500mg twice daily, given for a minimum of 4 months. It is contraindicated in children and pregnancy. Doxycycline and minocycline are alternative tetracyclines that are better absorbed.
   b. Antibiotics when given in therapeutic dose act as bacteriostatic but in sub-therapeutic dose they exert an anti-lipase action.
2) **Antiandrogens:** cyproterone acetate, may be used in females with moderate to severe acne which is resistant to conventional therapy.
3) **Systemic retinoids (isotretinoin):**
   - Used in severe nodulo-cystic acne or not responding to the usual treatment.
   - Dose: 0.5-1mg/kg/day.
   - Side effects:

   Cracked lips, dry skin, nose bleeding, hair loss and muscle aches. Transient increase in serum triglycerides and liver enzymes may also occur. Isotretinoin is teratogenic. Women given the drug must not be pregnant and need to take oral contraceptive throughout treatment and for the month before and after.
4) Post acne scars can be treated with dermabrasion, chemical peeling or LASER resurfacing.

❖ Atlas Fig. (52&53).

Questions

- Eccrine sweat glands are not evident on:

  a.Palms and soles.

  b.Oral mucosa.

  c.Below nails.

  d.Ear pinnae.

- Occlusion of the eccrine duct at the skin surface is known as:

  a. Milia.

  b. Miliaria profunda.

  c. Miliaria rubra.

  d. Miliaria crystalina.

PAPULOSQUAMOUS ERUPTIONS

Papulosqamous eruptions are raised, scaly, well defined lesions. They include psoriasis, lichen planus and pityriasis rosea.

PSORIASIS

A chronic inflammatory, non infectious skin disease characterized by well defined erythematous papules and plaques covered by silvery laminated scales.

Epidemiology:

Psoriasis affects 1-3% of the population of Europe and North America. It is usually a disease of adults with 2 exceptions: napkin psoriasis and guttate psoriasis. The sex incidence is equal.

Etiopathogenesis:

- The exact pathogenesis of psoriasis is unknown. It is believed to be an immune reaction in which activated T lymphocytes trigger increase in epidermal cell proliferation up to 20 foids and vascular proliferation.
- More than 30% of patients show a positive family history. Environmental factors are thought to precipitate the disease in genetically predisposed individuals.

Precipitating factors:

1. Trauma: Development of psoriatic lesions commonly occurs at the site of trauma such as scratching, mechanical friction, incisions 1 to 2 weeks after trauma [Koebner phenomenon].
2. Infection: Typically, streptococcal sore throat may precipitate guttate psoriasis.
3. Endocrine factors: The peak of incidence may occur at puberty or menopause. Pregnancy may improve psoriasis, but occasionally provokes generalized pustular psoriasis.
4. Climate: High temperature, humidity and sunlight are generally beneficial.
5. Metabolic Factors: Hypocalcaemia.
6. Drugs: Antimalarials, B-Blockers and lithium may precipitate or exacerbate psoriasis vulgaris , while topical and systemic steroids may predispose for erythrodermic or pustular psoriasis .
7. Psychogenic factors: Emotional stress is an important triggering factor.

Clinical Picture:

Primary lesion: Erythematous papule covered with laminated silvery scales which coalesce to form sharply defined plaques.

Clinical types:

1. **Chronic plaque Psoriasis**: the commonest. The lesions are usually asymptomatic, bilateral and symmetrical. Sites of predilection are the extensor surface of the limbs, elbows, knees, sacral region and scalp. According to shape of the lesions, it may be coin shaped "discoid or nummular psoriasis", "annular psoriasis" produced by peripheral extension of lesion with central involution, "linear psoriasis" as a presentation of Koebner phenomenon or "geographic psoriasis" with curved patterns, occurring mostly on a large area e.g. back.
2. **Guttate psoriasis:** affects children and young adults, mainly on the trunk in the form of rounded small lesions "drop-like". It is usually preceded by acute streptococcal infection.
3. **Flexural psoriasis:** affects intertrigenous areas [groin, axilla, retroauricular and submammary areas], characterized by absent or decreased scales due to moisture and continuous friction. There is usually considerable itching.
4. **Localized forms of psoriasis:**

   a-Psoriasis of the palms and soles.

   b-Scalp psoriasis: either diffuse scales or solitary plaques. It is usually not associated with hair loss.

   c-Psoriasis of the nails: in 30-50% of cases, in the form of nail pitting, transverse striations, subungual hyperkeratosis or onycholysis.

   d-Napkin psoriasis: affection of napkin area in infants as a presentation of Köebner phenomenon.

5. **Severe forms:**

- Erythrodermic psoriasis: Extensive disease involving all the body with generalized erythema and scaliness. It can be precipitated by infection, hypocalcaemia or drugs.
- Arthropathic psoriasis: This is either oligoarticular or polyarticular, typically affecting distal interphalangeal joints.
- Pustular psoriasis: localized to palms and soles or generalized; the most severe form of psoriasis. It can occur in last trimester of pregnancy and may be fatal.

Diagnosis

- Clinical examination.
- Grattage test: Scraping of psoriatic lesions with the edge of a glass slide results in removal of the scales in layers until a thin membrane is finally left. On removal of this membrane, pin point hemorrhages appear [Auspitz sign].
- Skin biopsy.

Differential Diagnosis of psoriasis

Variant of psoriasis	Differential diagnosis
Plaque psoriasis	Hyperkeratotic lichen planus
Guttate psoriais	Pityriasis rosea
Flexural psoriasis	Flexural candidiasis
Scalp psoriasis	Seborrheic dermatitis
Nail psoriasis	Fungal infection of the nails

Treatment:

Topical treatment:

1. Emollients to minimize scaling.
2. Keratolytics to remove the scales e.g salicylic acid 3-5%.
3. Tar preparation e.g crude coal tar 2-5%. It appears to have anti-proliferative actions. Its main disadvantages are its unpleasant odour and staining of cloths. It could be combined with UVB as Goekerman technique.
4. Anthralin [Dithranol]: it has an antimitotic effect. It is highly effective but irritant and stains skin also. The patient can be given tar bath, followed by UVB, then dithranol paste 0.1% [Ingram technique].Tar and anthralin are contraindicated in sensitive areas as face, genitalia and flexures and also in pustular and erythrodermic psoriasis.
5. Topical steroids: can be used with or without occlusion .Their benefits should be balanced against the risk of side effects. Potent steroids should be avoided on face, flexures and in young patients. Intralesional steroid can be used in small resistant localized lesions and in the nails.
6. Vitamin D3 analogues [e.g. calcipotriol]. They inhibit cell proliferation and stimulate keratinocyte differentiation. They are effective but expensive. Side effects include irritation and hypercalcaemia if the maximum dose is exceeded.
7. Topical retinoids as Tazarotene gel.

Phototherapy: UVB [narrow band or broad band].

Photochemotherapy:

PUVA (psoralen + UVA): popular but with long-term risk of skin cancer.

Systemic therapy:

Indications:

I. Generalized pustular, erythrodermic or arthropathic psoriasis.
II. Extensive plaque psoriasis resistant to topical therapy.
III. Psychological distress because of the disease.

1. Methotrexate: Folic acid antagonist. Indicated in erythrodermic, generalized pustular, arthropathic and severe extensive psoriasis. The dose is 0.2-0.4 mg/Kg in a weekly dose.

   Main side effects are hepatotoxicity and bone marrow suppression. It is contraindicated in pregnancy and liver disease.

2. Acitretin: systemic derivative of vitamin A. It is indicated in generalized pustular and erythrodermic psoriasis. The dose is 0.5- 1mg/kg/day. Main side effects are teratogenecity and increase in serum cholesterol. Contraindicated in children and pregnancy.
3. Cyclosporin A: selective immunosuppressive. Indicated in severe psoriasis. The dose is 2.5-5 mg/Kg/day. Main side effects are hypertension and nephrotoxicity.
4. Combined therapy: e.g. Re-PUVA [Acitretin and PUVA]
5. Biological therapy: as tumor necrosis factor inhibitors.

PITYRIASIS ROSEA

Pityriasis rosea is an acute self-limited papulosquamous eruption chararacterised by scaly oval papules and plaques which mainly occur on the trunk. The prefix "pityriasis" means bran-like scales.

Etiology:

- The etiology of Pityriasis rosea is unknown, but viral etiology is suggested.
- The disease is more common in spring and autumn.

Clinical picture:

- The disease is more common in young adults. It is usually asymptomatic but patients may present with variable degrees of itching
- Lesions usually disappear within 6-8 weeks. In severe and atypical forms, lesions may persist for a longer time. Recurrence is uncommon.

Herald patch:

The first clinical lesion in pityriasis rosea is the herald patch which is a solitary papule that enlarges rapidly to form an oval or an annular patch with a fine collarete of scales. The centre of the lesion tends to be clear and is light brown in color. The trunk, mainly the anterior chest is the most common site, but it can occur on the neck or extremities.

Secondary rash:

- The secondary eruption is more generalized and symmetrical. It occurs 1-2 weeks after the appearance of the herald patch as macules or maculopapules, erythematous and covered by fine scales. They enlarge gradually, becoming oval in shape and acquiring the same appearance of the herald patch.

- The eruption most commonly appears on the trunk, adjacent parts of the neck and upper arms. It usually follows the lines of cleavage of the skin in a distribution that resembles a Christmas tree pattern particularly when it affects the back.

Clinical varieties:

- Abortive type: Herald patch appears only and is not followed by secondary rash.
- Inverted type: Face and limbs are affected sparing the trunk.

Differential Diagnosis:

- Tinea circinata [herald patch].
- Guttate psoriasis
- Secondary syphilitic rash.
- Pityriasiform drug eruption.

Treatment:

It is mainly asymptomatic and the condition is self–limited. Treatment does not hasten clearance.

- Reassurance of the patient.
- Avoidance of irritants
- Soothing lotion [Calamine lotion].
- Moderate-potent topical steroid can help relieve pruritus.

LICHEN PLANUS

Lichen planus is an acute or chronic inflammatory skin disease characterized by itchy flat topped violaceous papules with affection of the mucous membrane, nails and hair.

Etiology:

- The etiology of lichen planus is unknown.
- Viral theory is suggested and it may be associated with hepatitis C virus infection.
- Immune pathogenesis is also suggested and it may be associated with autoimmune diseases.
- Lichen planus may be precipitated by trauma or exposure to sun, the so called Köebner phenomenon.

Clinical picture:

- Two-thirds of cases occur in 30-60 year old age group. It is uncommon at the extremes of age.
- Itching is a marked symptom of the disease.
- The primary lesion is an itchy flat topped polygonal papule. It is violet or purple in color and shows whitish striations called **Wickham's striae** which can be seen by a magnifying lens.
- Lesions heal with residual hyperpigmentation.
- The sites commonly affected are the front of wrists, forearms, legs and genitalia.

Variants of lichen planus:

1. Acute (eruptive) lichen planus.
2. Atrophic lichen planus.
3. Annular lichen planus.
4. Linear lichen planus.
5. Bullous lichen planus.
6. Lichen planopilaris: the lesions affect the hair follicles. On the scalp it causes cicatricial alopecia. **Graham Little syndrome**, it comprises the triad of : multifocal scalp cicatricial alopecia, non scarring alopecia of axillae and/or groin and keratotic lichenoid follicular papules.
7. Hypertrophic lichen planus: commonly seen along the shin of the tibia in the form of purplish thick pruritic plaques.
8. Lichen planus actinicus: occurs on sun- exposed areas especially the face .The lesions are non itchy macules and occur as a Koebner phenomenon to the sun rays.

9. Lichen planus of the mucous membrane: It commonly affects the buccal mucosa and lips in the form of white lines, streaks or patches. Ulcers may occur and can turn malignant in 1-2% of the cases [squamous cell carcinoma]. Glans penis, vulvovaginal region and anus are less commonly affected.
10. Lichen planus of the nails: Longitudinal ridging, pitting or even complete shedding of the nail occurs in cases of lichen planus.

Differential diagnosis:

Type of lichen planus	Differential diagnosis
Generalized lichen planus	• Lichenoid drug eruption • Guttate psoriasis • Pityriasis rosea
Genital lichen planus	• Psoriasis • Scabies

Lichenoid drug eruption: Gold, antimalarials, paraamino salicylic acid, and streptomycin may produce an eruption identical to lichen planus. It has been also reported in color film processors.

Treatment:

Local treatment:

- Topical steroids.
- Intralesional steroids [hypertrophic lichen planus].
- Sun screens [actinic lichen planus].

Systemic treatment: indicated for extensive involvement, ulcerative mucous membrane lesions or scarring nail dystrophy

- Antihistamines for itching.
- Systemic steroids in acute and extensive cases.
- Antimalarials [actinic lichen planus].

❖ Atlas Fig. (54&56)

CONNECTIVE TISSUE DISEASES

The inflammatory disorders of connective tissue often affect several organs, as in systemic lupus erythromatosus (SLE), but they may also involve the skin alone, as in discoid lupus erythromatosus (DLE). Autoantibodies and cell mediated immunity against normal cellular components (eg nuclei) are a feature of these diseases, which can thus be regarded as "autoimmune".

Etiology:

- Genetic predisposition.
- Precipitating factors:
  1. Sun exposure
  2. Physical trauma
  3. Mental stress
  4. Infections
  5. Drugs
  6. Pregnancy.

LUPUS ERYTHEMATOSUS (LE)

Acute Cutaneous LE (ACLE) = SLE:

Serious multisystem disorder that involves vascular and connective tissues.

Subacute Cutaneous LE (SCLE):

A subset between ACLE and DLE.

Chronic Cutaneous LE (CCLE) = DLE:

- Confined to skin.
- **Age:** 3rd and 4th decades
- **Sex**: female : Male =2:1
- Clinically, lesions typically present as sharply demarcated, coin-shaped (discoid) erythematous plaques covered by a prominent, adherent scale that extends into the orifices of dilated hair follicles (horny plugs or follicular plugging).

- **Distribution:**

1. **Localized DLE**: affects the scalp, butterfly area of the face, ears, V-shaped area of the neck and dorsa of the hands.
2. **Disseminated DLE:** widespread lesions above and below the neck.

- Healing occurs with thin atrophic non contractile, healthy stippled **scars.**
- Remission occurs in 40%. Internal involvement is not a feature, and only 5% develop SLE.
- **Differential diagnosis:**

  1. Lupus vulgaris
  2. Psoriasis.

- **Management of DLE:**

  1. ***Local therapy:***
     - Sun protective measures and broad spectrum sun screens
     - Potent topical steroids
  2. ***Systemic therapy:***
     - Oral antimalarial therapy for widespread disease.

❖ Atlas Fig. (57).

DISEASES OF THE HAIR

Anatomic characteristics of the hair:

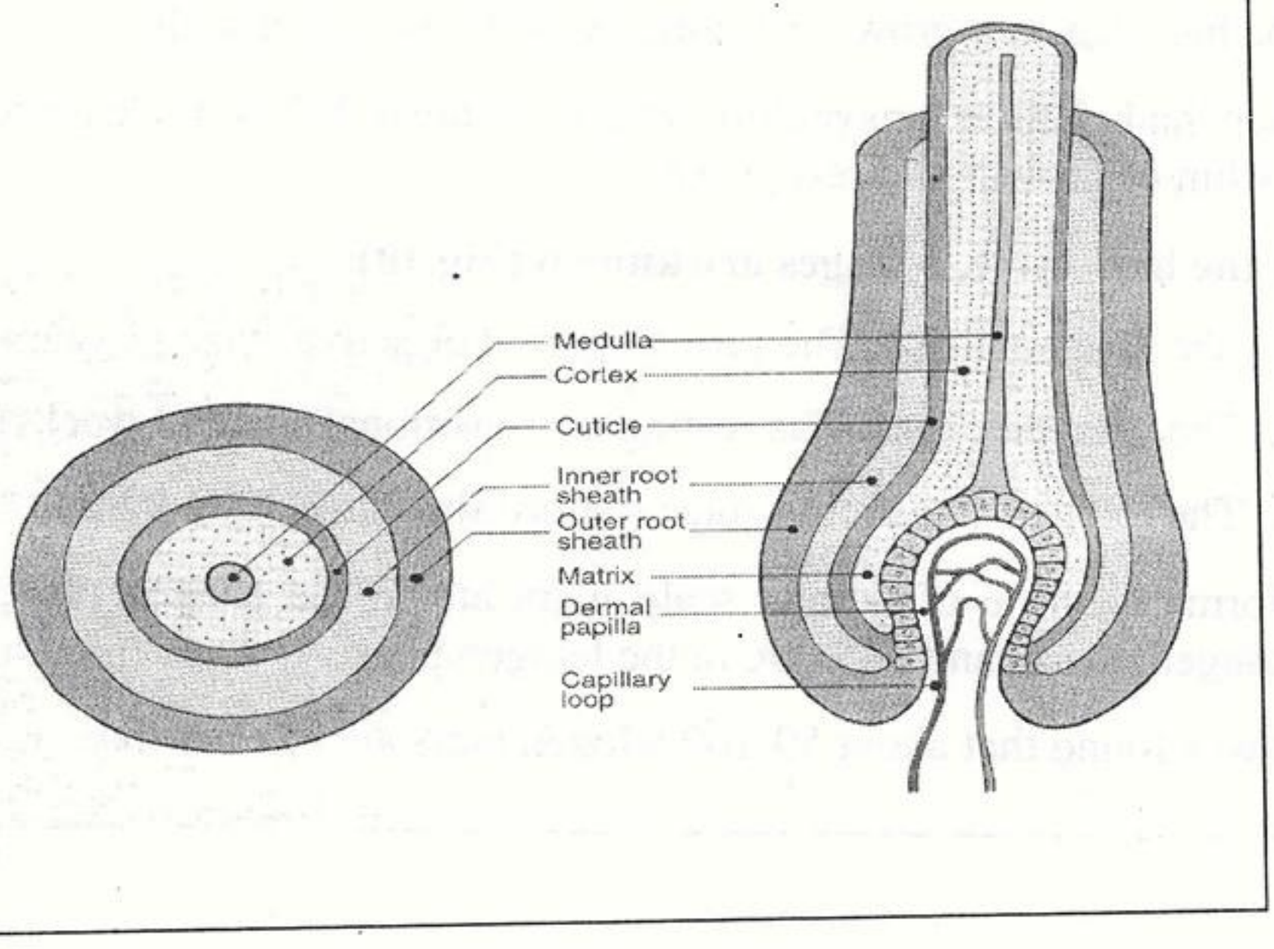


Fig. 9: The structure of hair.

Hair is present all over the body except the palms, soles, dorsal aspect of terminal phalanges of digits, glans penis, labia minora, lips, eye lids, areola and mucocutaneous junctions.

Embryologically: The hair is epithelial in origin. As early as 9th week of embryonic life, the hair follicle begins as an epithelial bud projecting downwards from fetal epidermal basal cells. (N.B. There is no neogenesis of hair follicles after birth).

Structure: (Fig 9). A hair follicle is mainly subdivided into upper and lower segment.

The hair follicle is composed of:

1. Outer root sheath.
2. Inner root sheath.
3. Hair shaft: composed of outer cuticle which encloses a cortex of packed keratinocytes with (in terminal hair) an inner medulla.

There are three types of hair:

- Lanugo hair: long, fine, soft, unmedullated usually unpigmented, covering the fetus prenatally, sheded in utero one month before term. Second coat of shorter lanugo hairs is present at birth in full term infants.

- Vellus hair: short, fine, soft, unmedullated hair covering most of body of youngsters.
- Terminal hair: Long, thick, dark, pigmented, medullated hair which is present in adults on the scalp, eye brows and eyelashes. Also, on pubic, axilla and beard areas, they originate as vellus hair, differentiation is stimulated at puberty by androgen.

The hair growth cycle:

- The hair does not grow continuously as do the finger nails.
- Each hair follicle unsynchronized with other hair follicles undergoes a cyclic rhythm of growth and rest phases.
- **In the hair cycle, 3 stages are known (Fig.10):**
  1. The anagen phase: The growth period or active stage (3 years).
  2. The catagen phase: The resting or transitional stage (3 weeks).
  3. The telogen stage: club stage (before shedding) (3 months).
- Normally, 84% of human scalp hairs are in the anagen phase, 2% in the catagen phase, and 14% are in the telogen phase.
- It was found that about 50-100 telogen hairs are lost per day.

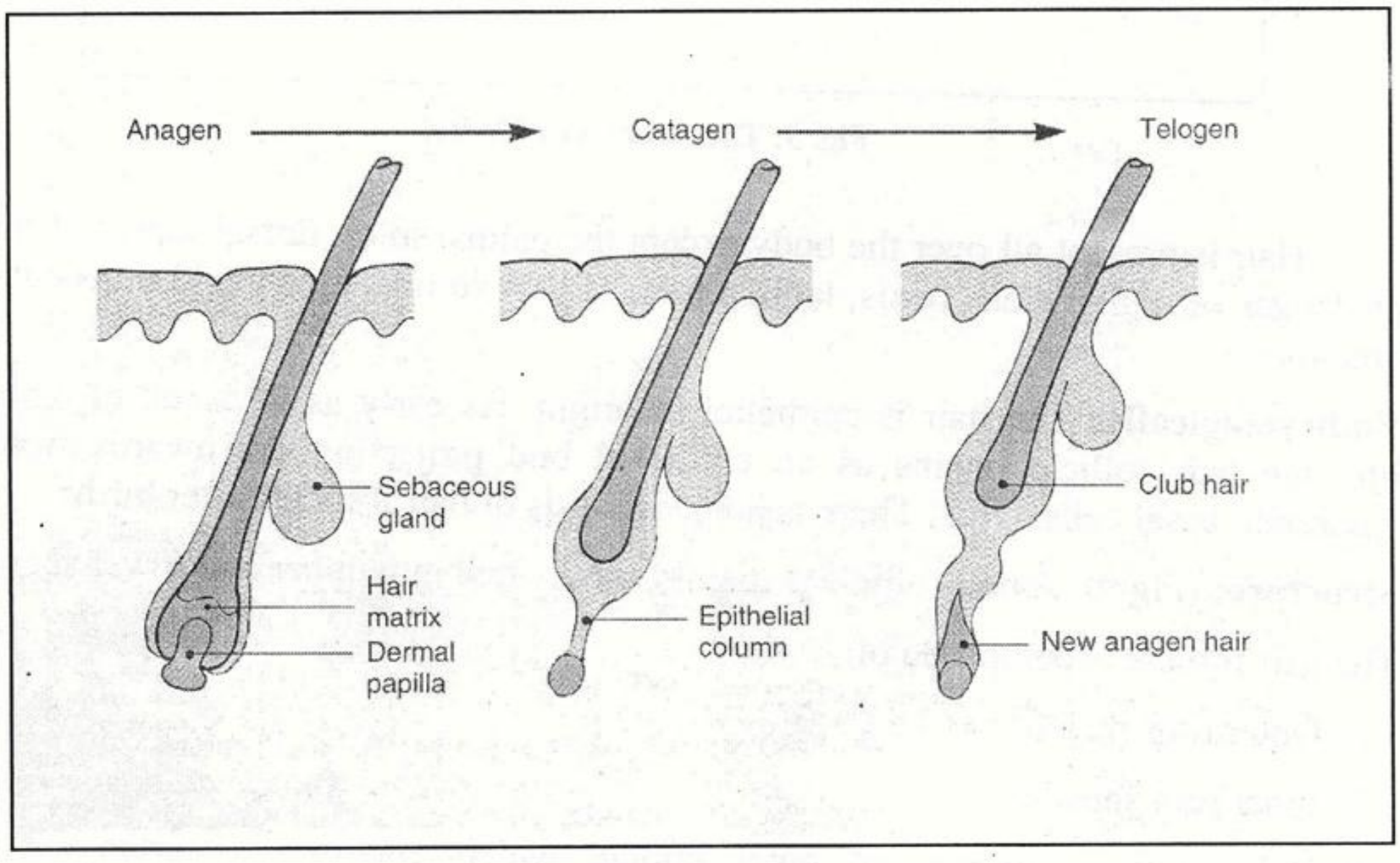


Fig. 10: The hair cycle.

ALOPECIAS

Definition: Alopecia means loss of hair which may be congenital or acquired.

A. ***Cicatricial alopecia:***

- Caused by destruction of the hair follicles by scar tissue formed in the scalp leading to permanent alopecia.
- The essential clinical picture is the presence of scarring and absence of hair follicles.
- Evidence of the disease or condition which caused scarring may be found.
- Loss of hair is permanent in cicatricial alopecia and re-growth of hairs in the affected area is not expected.
- Cicatricial alopecia may be due to:
  i. Trauma: physical (burns, radiodermatitis or mechanical) or chemical.
  ii. Infections: such as fungal infections (favus and neglected kerion), bacterial infections (staphylococcal infections, lupus vulgaris, syphilitic gumma) and viral infections (herpes zoster).
  iii. Collagen diseases: as DLE and scleroderma.
  iv. Neoplasms: as basal cell carcinoma.
  v. Lichen planus (lichen planopilaris).
  vi. Idiopathic.

B. ***Non-cicatricial alopecia:*** may be localized (circumscribed patches of hair loss) or diffuse.

- Localized non-cicatricial alopecia may be caused by:
  i. Alopecia areata.
  ii. Male baldness.
  iii. Trichotillomania.
  iv. Syphilis (moth-eaten alopecia in 2ry stage).
  v. Tinea capitis (scaly ring worm, black dot ring worm).

- Diffuse non-cicatricial alopecia may be caused by:
  i. Stress conditions: telogen effluvium.
  ii. Following prolonged fevers: e.g. typhoid.
  iii. Endocrinal: hypo- and hyper-thyroidism, hypopituitarism.
  iv. Drugs: as cytotoxic drugs and oral contraceptives.
  v. Anemia and malnutrition.
  vi. Debilitating diseases: e.g. malignancies.
  vii. Psychogenic causes.

ALOPECIA AREATA

Definition: It is a common skin disorder characterized by sudden loss of hair in a circumscribed area (or areas) usually on the scalp, but may involve other areas as the beard, moustache and eyebrows.

Etiology: is not settled. Many factors are implicated:

- Genetic factors: 10-20% of cases give positive family history.
- Immunological factors (most accepted): in favor of autoimmune mechanism.
- Psychological and emotional factors.
- Other factors: the condition may follow reflex irritation of ocular or dental origin and focal sepsis.

Clinical picture:

- It can affect any hairy part of the skin.
- It presents as asymptomatic sudden onset of hair loss.
- The affected areas are variable in size.
- Single lesion mostly occurs but there may be multiple lesions as well.
- Characteristically, the scalp skin is completely normal with no crusts or scales.
- Loss of hair is complete with no hair roots or stumps.
- The presence of **exclamation mark hairs** (hairs which are thin proximally and thick distally) indicates progression of the disease.
- The appearance of thin, fine and almost colorless hairs within the patch of alopecia areata indicates starting re-growth.
- ***Clinical types:***

  1. *Patchy type:* the commonest.
  2. *Alopecia totalis:* the hair loss involves the whole scalp.
  3. *Alopecia universalis:* the hair loss involves the whole body surface.

Prepubertal onset, extensive involvement (especially on the posterior scalp) and atopy are signals of poor prognosis.

Differential Diagnosis: from other causes of localized non-cicatricial alopecia.

Treatment (depends on the extent):

1. Topical treatment:
   a. Primary local irritants such as tincture iodine, tincture capsicum and tincture cantharidis.

b. Contact allergens such as diphenylcyclopropenone "DPCP".

c. Corticosteroids: topical and intralesional.

2. Systemic treatment: with corticosteroids in extensive resistant cases.
3. Phototherapy: UVB or topical PUVA.
4. Reassurance and correction of any precipitating factors as error of refraction and septic foci.

ANDROGENETIC ALOPECIA

- Inherited (exact mode is unclear) and is androgen dependant.
- Over several cycles the androgen – sensitive follicles convert from terminal to vellus.
- Males are affected from the second decade, bitemporal recession followed by a bald crown is the usual pattern.
- Only specific sites of the scalp hair are affected (i.e. androgen sensitive hairs) namely frontal region, parietal region & crown of scalp; the occipital and temporal parts are never affected.
- Androgenetic alopecia also occurs in females, the majority of whom are hormonally normal.

Treatment (most patients require no treatment):

- Local minoxidil 2-5% (produces some response)
- Systemic anti-androgens in females.

Traumatic (traction) alopecia

- **Trichotillomania:**

  It is the compulsive habit that induces the individual to pluck his or her own hairs and occurs in neurotic individuals.

- Traction from tight rollers or pulling causes alopecia at the scalp margin.

TELOGEN EFFLUVIUM

- Following stress conditions e.g. labour, operations, shock or fever; many hair follicles may prematurely enter the telogen phase resulting in hair loss.
- Characteristics of hair loss in telogen effluvium:
  - Occurs 3-4 months after exposure to stress.
  - Diffuse but not total.
  - All sheded hairs are in the telogen phase.

- Has a good prognosis and hair re-growth occurs within 3-4 months.

❖ Atlas Fig. (58).

PIGMENTARY DISORDERS OF THE SKIN

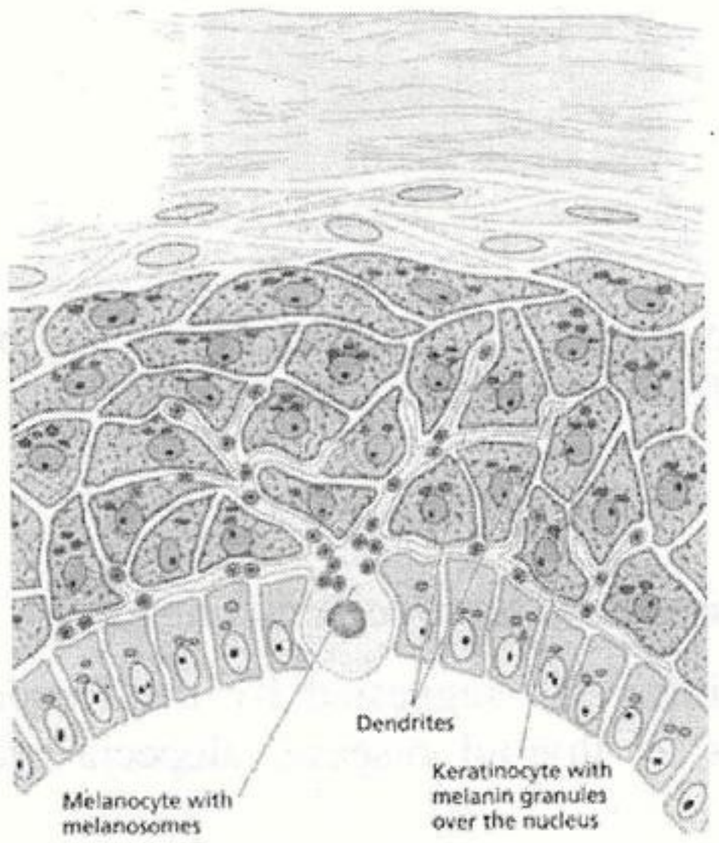


Fig. 11: A dendritic melanocyte among the basal cell layer.

- Melanocytes are found in the epidermis, hair follicles, eyes, leptomeninges and inner ear.
- Melanocytes (dendritic cells located in the basal layer) produce the pigment melanin in elongated membrane-bound organells known as melanosomes (Fig 11).
- Melanin pigmentation of human skin involves two processes:
  1. Melanogensis: biosynthesis of melanin in melanosomes within the melanocytes where the amino acid tyrosine is catalyzed by tyrosinase enzyme through multiple steps into melanin.
  2. Distribution and transfer of pigment (melanin) granules to the surrounding keratinocytes.

HYPOPIGMENTATION

Pigment loss may be generalized or localized (patchy).

Causes:

A. Localized hypopigmentation:
   1. Postinflammatory e.g. psoriasis vulgaris, pityriasis rosea.
   2. Vitiligo (***depigmented***).
   3. Pityriasis versicolor.
   4. Tuberculoid leprosy.
   5. Pityriasis alba.

B. Generalized hypopigmentation:

1. Albinism.
2. Vitiligo.
3. Hypopituitirism.

VITILIGO

Is an acquired idiopathic disorder characterized clinically by white non- scaly macules. Melanocytes are absent from lesions in histopathology.

Etiopathogenesis:

1- About 30 – 40% of cases have positive family history.

2- An auto immune etiology is suggested by the frequent associations with other autoimmune diseases as thyroid disease, alopecia areata, diabetes mellitus and pernicious anemia.

3- Neurogenic and Psychogenic factors: based on the assumption that a compound is released at peripheral nerves that have a toxic effect on melanocytes.

4- Melanocyte self-destruction theory.

Clinical features:

- Vitiligo affects 1% of the population. It is seen in all races with equal sex incidence.
- Any age could be affected, about 50% of cases begin between the age 10 and 30 years
- Skin lesions are in the form of "milky or chalk white" well-defined macules or patches of variable sizes that might range from 1 mm to the affection of large areas of the body. Individual lesions are often oval or forming geographic pattern.
- The hands, wrist, knees, neck and areas around orifices (e.g the mouth) are frequently affected.
- The disease has a gradually progressive course.
- Linear pattern represent Koebner's phenomenon.
- Pigmented or white hairs may be present in a vilitigo macule.

Differential Diagnosis:

- Pityrasis alba: hypopigmented, ill-defined macules with slight scaling.
- Tuberculoid leprosy: in endemic areas, anesthetic macule.
- Post-inflammatory hypopigmentation: usually a history of psoriasis or eczema in the same macular area and not sharply defined.

- Pityriasis versicolor: fine scales with golden yellow fluorescence under wood's light and positive KOH examination.
- **Albinism:** It's an autosomal recessive disease characterized by the absence of melanin in skin, hair, and eyes. Melanocytes are present but not functioning due to absence of tyrosinase enzyme.
- **Nevus anemicus:** It's a congenital anomaly of the skin, characterized by macular areas of pallor inspite of having a normal melanin pigmentation. It is due to reduced blood flow.

Management:

Treatment is unsatisfactory

1-Topical:

- Potent topical steroids occasionally induce repigmentation.
- Topical PUVA (psoralens followed by ultraviolet radiation).

2-Systemic:

- Systemic steroids and immunosuppressive drugs.

3-Phototherapy e.g.

- PUVA therapy [UVA (320-400 nm) + 8-methoxy psoralen].
- Narrow band -UVB (311-313 nm).

4-Surgical treatment: Micropigmentation (tattooing) or the use of autografting method.

HYPERPIGMENTATION

Hyperpigmentation is mostly hypermelanosis but sometimes other pigments colour the skin.

Causes:

A. Localized hyperpigmentation:
   - Post inflammatory e.g. lichen planus, eczema:
   - Chloasma.
   - Naevi.
   - Drugs: e.g.
     - Estrogen.
     - Oral contraceptives.
     - Tetracyclins, minocycline.
     - Chloroquines.

B. Diffuse hyperpigmentation:
   - Addison's Disease:
     - Generalized.

- Buccal mucosa, palmar creases, flexures, scars.

- Cushing's syndrome: on the trunk, upper arms.

CHLOASMA (MELASMA)

It's an acquired hyperpigmentation disorder that affects usually the sun exposed areas specially the face. It may be associated with pregnancy, ovarian carcinoma, and oral contraceptives but sometimes idiopathic. It is more common in female but it does occur in males.

Clinical features:

- Well-demarcated hyperpigmented macules.
- Occurs on the cheeks, forehead, nose upper lip and chin.

Differential Diagnosis:

- Postinflammatory hyperpigmentation.
- Actinic lichen planus.

Treatment:

Chloasma improves spontaneously. Sunscreens and camouflage cosmetics can help

- Discontinuation of oral contraceptives.
- Use of topical remedies that minimizes the melanocytic activity as hydroquinone, azaleic acid.
- Chemical peeling using trichloroacetic acid can be used for resistant cases.

❖ Atlas Fig. (59&60).

Section 2

SEXUALLY-TRANSMITTED DISEASES and ANDROLOGY

URETHRITIS

Anatomy of the female urethra: 4 cm canal lined with transitional epithelium in its proximal part and stratified epithelium distally. Skene's glands open into urethral meatus.

Anatomy of the male urethra: 20 cm from bladder neck to external urinary meatus. It is formed of:

A- Posterior urethra: This can be divided into:

1. **Prostatic urethra**: It is three cm, and lined with transitional epithelium. The ducts of prostatic acini and the common ejaculatory duct open in it.
2. **Membranous urethra**: It is 1.2 cm in length, and lined with transitional epithelium. It extends from apex of prostate to bulb of penis, between the two layers of the triangular ligament. Cowper's glands lie on either side of its posterior surface.

B- Anterior Urethra (spongy-penile): It is 15 cm in length. It is lined with columnar epithelium except its terminal part (fossa navicularis), which is lined with stratified squamous epithelium. It is formed of two parts:

1. **Bulbous urethra**: from anterior layer of triangular ligament to penile angle. Ducts of Cowper's glands open in this part of the urethra.
2. **Distal or pendulous urethra**: from penile angle to fossa navicularis. Littre's glands are present in the roof & sides of penile urethra and their ducts open in this part of the urethra. Their function is mucus secretion. Paraurethral ducts open near the external meatus.

Etiology of urethritis

Urethritis	
Gonococcal	**Non gonococcal**
Due to Neisseria gonorrhoea	• Sexually transmitted - Chlamydia trachomatis (main cause) - Mycoplasma and Ureaplasma urealyticum. - Trichomonas vaginalis • Non- sexually transmitted - Urinary tract infection • *Secondary to:* urethral instrumentation, foreign bodies, renal stones or crystalluria.

GONORRHEA

Causative organism:

- Neisseria gonorrhoea.
- Gram negative, kidney-shaped, intracellular diplococci, (i.e. inside the polymorphonuclear leucocytes).
- Pili or fimbriae are present on surface of virulent gonococci for attachment to host cells.
- Rapidly destroyed outside the body by: dryness, heat, and antiseptics.
- Attaches to columnar & transitional epithelium [stratified squamous epithelium resistant to infection].

Mode of infection:

- Sexual intercourse.
- Accidental (e.g. from towels or lavatory seats: rare).
- Newborn infection: passage during birth in infected females leads to ophthalmia neonatorum.

Clinical Picture:

- *Incubation period:* 2 - 5 days.
- ***Sites of involvement :***
  - Male: urethra, Littre's glands, Cowper's gland, prostate, seminal vesicles, epididymis, rectum, and oropharynx.
  - Female: Urethra, Skene's glands, Bartholin's glands, cervix, fallopian tubes, rectum, oropharynx.
- The clinical outcome of the infection depends on:
  - The amount & type of inoculum.
  - Degree of sex excitement.
  - Local condition of the mucous membrane.
  - Local congenital anomaly.
  - Local congestion.
  - Personal susceptibility.
  - Immunity (local and general).
- ***Male:*** - Purulent anterior urethritis with profuse yellowish discharge (if neglected for 2 weeks posterior urethritis results with associated dysuria, frequency and urgency).

- Gonococcal proctitis with anal pruritus, pain, tenesmus and discharge (rectum is lined with columnar epithelium).
- Oropharnyngeal gonorrhoea: sore throat & tonsillitis.
- Gonococcal conjunctivitis: direct contamination of eyes with discharge.

- ***Female:*** -Scanty urethral or genital discharge, or asymptomatic.
  -Gonococcal proctitis.
  -Oropharyngeal gonorrhoea.
  - Gonococcal conjunctivitis.

Complications of gonococcal urethritis:

Local complications:	
Male ▪ Balanitis or balanoposthitis, tysonitis. ▪ Littritis, periurethral abscess, cowperitis. ▪ Prostatitis (acute, chronic, abscess). ▪ Chronic prostatitis>10 pus cells / high power field, with clumps. ▪ Seminal vesiculitis (painful erections and haemospermia). ▪ Epididymitis. ▪ Urethral stricture.	Female ▪ Skenitis. ▪ Bartholinitis. ▪ Pelvic inflammatory disease (PID). ▪ Perihepatitis in some cases with salpingitis.

Gonorrhea in children:

- Gonococcal vulvovaginitis: It occurs in female children as vulvovaginal epithelium is immature stratified squamous epithelium with poor glycogen and alkaline pH, which are predisposing factors. It might be accidental or as a result of child sex abuse with the result of vaginal discharge, itching and dysuria.
- Gonococcal ophthalmia: from infected pregnant mother during birth and it might lead to blindness.

Distant and systemic complications:

Disseminated gonorrhoea: It occurs in less than 1% of cases and is more in females. It usually presents with fever, joint affection, and skin rash. Blood cultures are usually positive.

Diagnosis:

1. *Smears:* from discharge (in acute cases) from morning drop (in complicated chronic cases) from over/night urine (in contacts) - Gram stain.
2. *Culture* : is more reliable than smear especially in females, oral & rectal infections, and in disseminated gonococcal infection. The gonococci are cultured under aerobic conditions. The growth medium: is the chocolate agar, while the selective medium is known as Thayer Martin medium. The organism is identified from the growing colonies as being oxidase test positive and by being glucose fermenter with acid production (using phenol red as an indicator, it turns yellow upon positivity).
3. *Direct immunofluorescence.*
4. *Radio immunoassay:* It is the most sensitive test as it detects antibodies to gonococci fimbriae.
5. *Monoclonal antibodies technique:* uses types of major outer membrane proteins.
6. *Tests for antibiotic sensitivity:* disc method (most strains are still sensitive to penicillin).

Treatment of gonococcal urethritis:

I. Systemic antibiotics:

1. Penicillins:

- Penicillin interferes with the cell wall synthesis.
- Procaine penicillin 4.8 million units IM at 2 sites.
- Ampicillin 3 gm single dose.
- Amoxycillin 3 gm single dose.

2. Spectinomycin: 2 gm IM single dose.

3. Ceftriaxone: 250 mg IM single dose.

4. Quinolones: as Ofloxacin 400 mg single dose.

II. Abstain from alcohol and sexual intercourse.

Follow up

At the 3rd day of treatment there will be no more discharge and this means a successful treatment.

NON GONOCOCCAL URETHRITIS

I. CHLAMYDIA TRACHOMATIS

Biology of Chlamydia trachomatis:

- It is an obligatory intracellular organism, with two forms:
  - Elementary body: infectious form (adapted to extracellular survival).
  - Initial (inclusion body): non infectious form (intracellular replicating form).
- It contains both DNA and RNA.
- It can not grow on artificial media, but on yolk sac and chick embryo.

Clinical picture:

- *Incubation period:* 2 - 3 weeks.
- Urethritis (milder than gonococcal urethritis) with mucoid or mucopurulent discharge. Carriers and partners may be asymptomatic.
- Postgonococcal urethritis.
- Other features:

Male	Female
Prostatitis, epididymitis, urethral stricture, Proctitis.	Bartholinitis, cervicitis (mainly).
Infertility.	PID.
Reiter's syndrome (urethritis-conjunctivitis - arthritis).	Abortion, premature labour, stillbirth.

- Neonatal Chlamydial infection: from infected cervix (conjunctivitis - pneumonia - otitis media).

Laboratory diagnosis:

- Direct examination of smears using anti-Chlamydial antibodies by immunoflourescence.
- Isolation on living cells.
- Anti-chlamydial antibody detection (serodiagnosis) by microimmunofluorescence.
- Chlamydial DNA detection by polymerase chain reaction (PCR).

II. MYCOPLASMA AND UREAPLASMA UREALYTICUM

Biology of Mycoplasma: between rickettsia and bacteria. It contains both DNA and RNA. It grows on artificial media.

Clinical picture:

- ***Male:*** urethritis, epididymitis, prostatitis, infertility and Reiter's disease.
- ***Female:*** Urethritis, PID, abortion, still birth and premature labour.

Laboratory diagnosis:

- Culture.
- Serology: immunoflourescence.

Treatment of Non gonococcal urethritis;

- Tetracycline 500 mg 1×4×14.
- Doxycycline 100 mg 1×2×14.
- Azithromycin 1 gm single dose.
- During pregnancy: erythromycin 500 mg 1×4×14.

III. TRICHOMONIASIS

Etiology:

Trichomonas vaginalis is an anaerobic protozoan with oval shape. It is motile with an irregular jerky movement. It can survive outside the body for up to 24 hours in moist environment.

Epidemiology:

- Females are more affected than males since the organism needs the glycogen available in the vagina.
- It may be associated with other types of urethritis.
- The asymptomatic infected patients are important reservoirs for infection.
- Males are transient carriers due to the antitrichomonal effect of the prostatic secretion.
- ***Mode of infection:***
  - Sexual intercourse.
  - Non sexual transmission through fomites.

13 Urethritis

Clinical Picture:

- *Incubation period:* 3days - 3weeks.
- ***Females*** may show minor symptoms or vaginitis and profuse offensive discharge, urethritis & dysuria. Upon examination red vaginal wall and red spots on cervix (strawberry cervix).
- ***Males*** might be asymptomatic or may show urethritis with a tickling sensation in the urethra.

Diagnosis:

1. Direct microscopic examination (wet mount preparation): secretions from posterior vaginal fornix or urethral scrapings in men are taken and a drop of saline is added. A cover slip is applied & examination is done under dark field.
2. Stained smear (less reliable): Papanicolaou Stain.
3. Culture.

Treatment:

- Metronidazole 250 mg 1×3×7.
- Tinidazole 2 gm single dose.
- Treatment of the sexual partner.

Questions

- Match each of the following diseases (A) with its appropriate treatment (B):

1-Herpes progenitalis	a. podophyline.
2-Chlamydial urethritis	b. acyclovir.
3-Condyloma accuminatum	c. tetracycline.
4-Trichomonas vaginalis	d. benzathin penicillin.
	e. tinidazole.

- Ureaplasma urealyticum can be treated by all the following except:
  a. Doxycycline.
  b. Cloxacycline.
  c. Azithromycine.
  d. Erythromycine.

SYPHILIS

It is a chronic, infectious sexually transmitted disease caused by the spirochaete; Treponema pallidum (T. pallidum).

T. pallidum:

- A very small (6-15 μm in length, 0.15 μm in width), spiral spirochaete.
- Seen by dark ground microscopy and not by light one (because it is below the level of resolution).
- Has a characteristic corkscrew rotation motility that helps to differentiate it from other treponemas.
- Doesn't grow on any artificial media.
- Can't be stained easily.
- Heat, dryness and washing hands with soap and water are enough to kill the organism.

Transmission:

1- Acquired:

- **Sexual transmission:** through contact with infectious lesion during sexual activity (most common).
- **Accidental transmission:** through direct contact by an examining doctor, indirect contact with contaminated glasses or toilet seats (very rare)
- **Fresh blood transfusion:** if the donor is infectious.

2- Congenital:

Vertical transmission: from an infected mother to her baby.

Incubation period: 9-90 days of infection.

Stages and natural history of the disease:

It may be either acquired or congenital.

1- Acquired:

Untreated syphilis may pass through three stages, beginning with the infectious cutaneous primary and secondary stages, which may terminate without further sequelae or may evolve into a latent stage that lasts for months or years before the now-rare tertiary stage, marked by the appearance of cardiovascular, neurologic, and deep cutaneous complications.

14 Syphilis

Primary stage: It occurs due to local tissue reaction at the site of entry of treponemas in the form of a papule that breaks into syphilitic ulcer (chancre) which is characterized by the following:

- Usually single.
- Painless (except if secondarily infected).
- Indurated base.
- The edge is sloping (raised and sharply defined at the periphery and sloping towards the center).
- The floor is clean and fleshy red which does not bleed easily due to endarteritis obliterans.
- The lymph nodes are bilaterally enlarged, painless, discrete, non suppurative and firm rubbery in consistency.
- Usually on the genitals (90%) but in 10% it may occur on the lips, tongue, nipples or fingers (extragenital).
- Spontaneous healing occurs in 2-6 weeks with thin atrophic unnoticed scar.
- ***Differential diagnosis:***
  - Chancroid.
  - Lymphogranuloma venereum.
  - Granuloma inguinale.
  - Herpes progenitalis (recurrent).
  - Behcet's disease (recurrent oral and genital).
  - Traumatic ulcer.
  - Fixed drug eruption (recurrent with the offending drug).

Secondary syphilis:

- It starts in 4-6 weeks from the appearance of the primary stage (chancre may be present in 1/3 of patients). T. pallidum gain access to blood stream and cause mucocutaneous lesions, musculoskeletal lesions, generalized lymphadenopathy, in addition to hepatic and CNS involvement. Spontaneous clearance occurs in few weeks.
- **Clinical picture:**

  -Fever and constitutional symptoms.

  -Skin rash.

  -Condyloma lata.

  -Genaralized lymphadenopathy (as with chancre, they are painless, discrete, mobile, firm, rubbery and never suppurate).

- Mucous membrane lesions (mucous patches and snail track ulcers).
- Hepatitis, nephritis or nephritic manifestations.
- Arthralgia and arthritis.
- Epididymitis.
- Iritis.
- C.S.F. changes in 1/3 of patients.

- **Skin Rash:**

  The cutaneous lesions vary greatly and may resemble any skin eruption **(great imitator)** with the following features:

  - Bilateral.
  - Symmetrical.
  - Symptomless (non-itchy).
  - Never vesiculates.
  - Widespread, affects the whole integument even palms and soles (characteristic).
  - Polymorphic in the form of: macules, papules and pustules.
  - Patchy, irregular, non-cicatricial alopecia (moth-eaten alopecia).
  - **Genital condyloma lata:**

    These are one of the characteristic lesions of 2ry syphilis. They are greyish white, moist, sessile, flat lesions with smooth surface and indurated base. Similar lesions may occur in other mucocutaneous junctions (perioral and periorbital).

    D.D.: Viral condyloma accuminata

Differences between the syphilitic condyloma lata and the viral condyloma accuminata

	Condyloma lata	**Condyloma accuminata**
Surface	Flat moist	Rough verrucous
Shape	Sessile	Pedunculated
Base	Indurated	Soft
Bleeding	Does not bleed easily	Bleeds easily
Dark ground examination	Positive	Negative
Serology for syphilis	Positive	Negative

Latent syphilis:

It follows secondary stage and is characterized by absence of symptoms and signs but positive serological tests. It is divided into: early latent: during the 1st two years during which relapse can occur e.g. relapse of skin rash, mucous patches or condyloma (infectious) and late latent during the period after the 1st two years (no relapses occur).

Fate of untreated latent Syphilis:

1-Spontaneous cure: Serological tests become negative and the disease is described as **"burnt out"** (30% of cases).

2-Persistent latent Syphilis: Serological tests remain positive and no further clinical manifestations develop (30%).

3-Benign tertiary Syphilis: characterized by development of **gumma** in skin, mucous membranes, bones or internal organs (15%).

4-Chronic syphilitic inflammation of vital organs: manifestations of cardiovascular and neurosyphilis (25%).

Benign Tertiary stage "Gumma":

- Appears within 3-7 years of chancre.
- Represents an allergic response to T.P.
- May occur anywhere in any organ as well as in the skin and subcutaneous tissue.
- Gumma of the skin and subcutaneous tissues:

  - Nodule or grouped nodules that ulcerate giving punched out polycyclic ulcer with the floor covered with yellow necrotic material (wash leather slough). This necrotic material is of gummy consistency, hence the name"Gumma".
  - Painless.
  - Indurated base.
  - Non-infectious (no organisms).
  - No enlargement of draining lymph nodes (no organisms).
  - Heals spontaneously with atrophic non-contractile scar.

Congenital Syphilis (no primary stage and no chancre)

- *T. pallidum* can be transmitted by an infected mother to the fetus in utero. The clinical appearance depends upon the time of acquisition of the disease by the mother. When a syphilitic woman becomes pregnant, any of the following may happen:

  1- The child might escape infection.

2- The fetus might be affected early in pregnancy and miscarriage occurs after 12-16th week (following the formation of the placenta). Miscarriages before the third month are not especially indicative of syphilis.

3- The child might die in utero late in pregnancy resulting in stillborn macerated baby.

4- The child might be born showing signs of syphilis.

5- The child might be born apparently well, but develops signs of **early congenital syphilis** during the first weeks or months of extra-uterine life.

6- The child may show no early signs, but **late congenital syphilis** may become manifest in later years.

- The Treponema pallidum can cross the placenta at any time during pregnancy. **Syphlitic placenta** is usually bulky, heavy, pale and greasy, infarcts are common (this represents the primary stage in congenital syphilis).
- The fetus is at greatest risk when maternal syphilis is of less than 2 years duration. The ability of the mother to infect the fetus diminishes but never disappears in late latent stages.

Early congenital syphilis:

- Early congenital syphilis is defined as syphilis acquired in utero that becomes symptomatic during the first 2 years of life.
- The manifestations are those of the acquired secondary stage syphilis in addition to the following characteristic lesions:

-The skin rash:

- Vesiculobullous eruptions mainly on the palms and soles (vesicles and bullae never occur in acquired syphilis).
- Perinasal, perioral and perianal skin lesions become fissured and heal by radiating scars called **rhagades.**
- Purpuric eruption may occur due to thrombocytopenia.

-Bone and joint symptoms are common.Rhinitis with highly infectious nasal discharge, non tender generalized lymphadenopathy, alopecia, iritis, and failure to thrive occur less frequently.

Late congenital syphilis:

- Symptoms and signs of late congenital syphilis become evident after age 5 years. The average age at first diagnosis is 30 years. Its manifestations are those of acquired tertiary stage in addition to the following characteristic lesions:
  - Frontal bossae (bony prominences of the forehead).
  - Saddle nose.
  - Short maxilla.
  - High arched palate.

- Sabre tibia: due to periostitis, new bone deposition occurs on the anterior aspect of the tibia giving it the shape of the sabre.
- Clutton's joint: due to synoivitis of the joint, painless, self-limited effusion of the joint occur with preservation of normal mobility.
- Paroxysmal cold hemoglobinuria: attacks of chills, body pains and dark urine on exposure to cold.
- Hutchinson's teeth (peg-shaped, serrated and widely spaced upper central incisors of the permenant dentition that appear after the age of 6 years).
- Rhagades (linear scars radiating from the angle of the eyes, nose, mouth, and anus).
- Hutchinson's triad (Hutchinson's teeth, interstitial keratitis, and eighth nerve deafness) is considered pathognomonic of late congenital syphilis.

Stigmata of Congenital Syphilis:

These are permenant marks that occur and remain for life after healing of congenital (early or late) syphilitic lesions as:

1- Corneal opacities after interstitial keratitis.

2- Sabre tibia.

3- Hutchinson's teeth.

4- Syphilitic facies: frontal bossing, saddle-shaped nose, prominent mandible and short maxilla.

5- Rhagades: radiating scars perinasal, perioral and perianal.

Diagnosis of Syphilis:

I. Dark field microscopy (primary & secondary stages)

II. Syphilis Serology

Two classes of IgM and IgG antibodies are produced in response to infection with *T. pallidum*; these are:

1. **Non specific** antibodies (the reagin). They are known as the Standard Tests of Syphilis (STS). They include the following flocculation tests: Venereal Disease Research Laboratory (VDRL) test and rapid plasma reagin (RPR) test. They give quantitative as well as qualitative results and can be used to monitor response to therapy. These tests are used for screening purposes and have a **high degree of sensitivity** (positive in most patients with syphilis) but **relatively low specificity** (positive in patients without syphilis, i.e. biological false positive results e.g. pregnancy, malignancy, collagen diseases and viral diseases). A rising titer indicates active disease; the titer falls in response to treatment.
2. **Specific** antibodies measured by the fluorescent treponemal antibody absorption (FTA-ABS IgM) test, treponema pallidum immobilization (TPI) and treponema pallidum haemagglutination test (TPHA).

Fluorescent Treponemal Antibody absorption (FTA-ABS IgM) test

- Its main use is to rule out biological false-positive reagin test reactions and to detect late syphilis in which the reagin test result may be non-reactive.
- It is the first specific test to become positive during the course of infection. It is the most sensitive test among them (i.e. does not miss low concentrations of antibodies). It utilizes another strain than treponema pallidum (Nichol's strain).
- It is the only test that can diagnose neonatal syphilis as IgM can not cross placenta.

Treponema Pallidum Immobilization (TPI) test

- It is an expensive complicated test, but it is the most specific among the treponemal test (i.e. has almost no percentage of false positivity).

Treponema Pallidum haemagglutination (TPHA) test

- It has midway specificity and sensitivity between the above two tests.

N.B: A patient who has a reactive treponemal (specific) test usually will have a reactive test for a lifetime, regardless of treatment or disease activity (**Serological scar**). However, 15% to 25% of patients treated during the primary stage may revert to serologically non-reactive after 2 to 3 years.

Treponemal test antibody titers correlate poorly with disease activity and should not be used to assess response to treatment.

III. Examination of C.S.F. to confirm the diagnosis of neurosyphilis.

Treatment of Syphilis

- Patients to be treated for syphilis should have a baseline serum VDRL or RPR determination. The best indication of successful therapy is a falling titer.
- The drug of choice in the treatment of syphilis is benzathine penicillin G. Patients sensitive to penicillin should be treated with alternative drugs .

Penicillins:

- Long acting benzathine penicillin or procaine penicillin in oil with 2% aluminium monoesterate (PAM) in a total dose of 12 million units, 1.200.000 units IM twice/week is given until total dose is reached.
- **Side effects:**
  - Herxheimer reaction (therapeutic shock): it occurs after the 1st penicillin injection due to the sudden release of treponemal antigens as a result of massive killing of the organism especially in 2ry stage. Unlike anaphylactic drug reaction, penicillin is continued under an umbrella of systemic steroids.

- Therapeutic paradox: symptoms become worse due to rapid fibrosis that occurs with therapy.
- Penicillin allergy

Other antibiotics:

- Tetracyclines 2 gms daily for 15 days.
- Erythromycin 2gms daily for 15 days

Treatment regimens:

Early Syphilis:

- Benzathine penicillin G 2.4 million units single dose intramuscularly.
- In penicillin allergy:
  - Erythromycin 500 mg / 4 times daily / 15 days.
  - Or Tetracycline 500 mg / 4 times daily / 15 Days.

Late Syphilis:

- Benzathine penicillin G: 2.4 million units intramuscularly 1 week apart for three doses or until total dose is reached.
- In penicillin allergy:
  - Erythromycin 500 mg / 4 times daily / 30 days.
  - Or Tetracycline 500 mg / 4 times daily / 30 Days.

Congenital Syphilis:

A-Prophylactic:

The prevention or cure of congenital syphilis in utero is by treating the mother during pregnancy.

B-Curative:

- Early Congenital Syphilis: Penicillin total dose is 400,000 units per Kg in divided doses over 10-15 days either of aqueous procaine penicillin or PAM.
- Late congenital Syphilis: like late acquired syphilis.

Questions

- While treating syphilis, Herxheimer reaction may occur due to:
  a. Sudden release of treponemal antigens.
  b. Rapid fibrosis after therapy.
  c. Delayed hypersensitivity reaction.
  d. All of the above.

- All of the following is true about FTA-ABS IgM test except:
  a. Shows the highest specificity among specific tests.
  b. The first specific test to become positive.
  c. The most sensitive among specific tests of syphilis.
  d. The antibody titre correlates poorly with the disease activity.

LYMPHOGRANULOMA VENEREUM, CHANCROID AND GRANULOMA INGUINALE

LYMPHOGRANULOMA VENEREUM

Caused by **Chlamydia trachomatis;** serotypes L-1, L-2, and L-3. These are different from D-K serotypes that cause non-specific urethritis and cervicitis.

Clinical picture:

- The primary lesion is a painless non-indurated penile vesicle or papule which develops 1-4 weeks after infection which passes often unnoticed, since it heals quickly.
- Regional adenitis, with stretched and discolored overlying skin, and constitutional symptoms follow 1-2 weeks later. The lymph nodes become matted and suppurate, with multiple fused abscesses (**buboes**). Buboes may rupture through the overlying skin leading to multiple sinus formation.
- In males, the inguinal glands are most commonly involved but in females and homosexual males, the peri-rectal glands suppurate and may be associated with painful proctitis and bloody anal discharge .
- Unless treated, the inflammation becomes chronic leading to fibrosis and lymphatic obstruction. Rectal strictures, sometimes with the formation of peri-rectal abscesses and fistulas, and elephantiasis of the genitalia may follow.

Diagnosis:

- Serology: complement fixation and microimmunofluorescence.
- Tissue culture from aspirates of suppurative lymph nodes or abscesses.
- PCR.
- Biopsy.

Treatment:

Tetracyclines or Sulphonamides during the acute phase will prevent the subsequent chronic changes, but once these have developed antimicrobial agents have little effect.

CHANCROID (SOFT SORE)

Caused by **Haemophilus ducreyi,** gram-negative bacilli.

Clinical picture:

- The incubation period is usually 4-10 days.
- Lesion begins as a soft papule surrounded by erythema. It then becomes pustular, eroded, and ulcerated. Vesicles are not seen.
- The chancroid ulcers are usually tender and quite painful. The edges of the ulcers are often ragged and undermined and its base is composed of granulation tissue that bleeds readily on manipulation.
- The lesions are usually accompanied by unilateral, painful, tender and matted lymphadenopathy.

	Chancre	Chancroid
Incubation period	9-90 days	4-10 days
Number of lesions	single	multiple
Pain	painless	painful
Edge	sloping	ragged undermined
Base	firm indurated	soft
Bleeding	does not bleed easily	bleeds easily
Lymph nodes	painless, discrete, firm, never suppurate	tender, matted, suppurate and form sinuses

Diagnosis:

- Gram-stained smear of material from a genital lesion.
- Culture of lymph node aspirate on enriched media.
- PCR
- Biopsy

Treatment:

- Azithromycin: single oral dose 1gm.
- Ceftriaxone: single 250 mg intramuscular dose
- Ciprofloxacin: 500 mg orally twice a day for 3 days.
- Erythromycin: 500 mg orally 4 times a day for 7 days.

GRANULOMA INGUINALE

Caused by **Calymmatobacterium granulomatis;** a non-motile, gram-negative coccobacillus, often showing bipolar staining.

Clinical picture:

- The incubation period ranges from 2 weeks to 3 months.
- The primary lesion may be button-like papule, subcutaneous nodule, or ulcer.
- Papules or nodules are quickly denuded and ulcerate within several days. Regional lymphadenopathy does not usually occur unless there is secondary infection. However, the enlarging granulomatous process may mimic lymph nodes **(pseudo-buboes).**
- Sequelae include stenosis of the urethral, vaginal or anal orifices, and massive elephantoid oedema.

Diagnosis:

- Stained smear with Wright's or Geimsa stain: Donovan bodies in mononuclear cells appear as safety pin-like due to bipolar distribution of chromatin.
- Biopsy.

Treatment:

Tetracycline or Cotrimoxazole given until healing has occurred (usually 2-3 weeks).

ACQUIRED IMMUNODEFICIENCY SYNDROME

Definition:

Acquired immunodeficiency syndrome (AIDS) is the end stage of human immunodeficiency virus (HIV) infection. It is a pandemic and is considered the most common cause of death among infections worldwide.

Virology:

- HIV1 and HIV2 with their several serotypes are responsible for the disease. HIV2 (prevalent in West Africa) is genetically different and less transmissible than HIV1. Infection with HIV2 provides protective role against superinfection with HIV1.
- Both HIV1 and HIV2 are retroviruses that carry two strands of RNA and use a DNA polymerase enzyme called *reverse transcriptase* to convert viral RNA to DNA "in a retrodirection". This reverses the usual process of transcription whereby DNA is converted to RNA, thus the term retrovirus.
- The viral genome is surrounded with a core (that contains p24 and p17 core proteins) and an envelope. The envelope contains two glycopropteins that play an important role in the attachment of the virus to its receptor, the surface gp120 and the transmembrane gp41.
- HIV gp120 attaches to a protein receptor site (CD4) on the surface of CD4+ lymphocytes (the primary target of HIV). The virus is uncoated as it penetrates the cell, and exposes its RNA core; then reverse transcriptase converts viral RNA to DNA, which becomes part of the host genome. New viral particles are then produced during normal cellular division, and CD4+ lymphocytes are destroyed, releasing new virions to infect more cells.

Transmission:

- HIV is present in all body fluids including saliva and tears. However, the highest concentrations are found in blood and genital (semen and vaginal) secretions.
- Transmission can occur by the following:
  - *Sexual:* Hetero sexual (most common), homosexual and bisexual.
  - *Mother to child:* intrauterine through placenta, during delivery through vaginal secretions (most common) or through breast milk.
  - *Contaminated instruments* as sharing common needles by IV drug abusers.
  - *Accidental* needle stick by medical personnel.
  - *Blood and blood products* transfusion.
- Casual life contact with HIV patients and mosquito bites are not considered methods of disease transmission.

Pathogenesis:

- HIV selectively attacks helper/inducer CD4+ T lymphocytes, decreases their number, and interferes with their function. CD4+ T lymphocytes are responsible for modulation of essentially the entire immune response. Thus, progressive immune dysfunction is the hallmark of HIV infection.
- In an undetermined number of patients, symptoms emerge and the disease progresses to AIDS. It causes a profound defect in cell-mediated immunity, which causes complicating opportunistic infections and neoplastic processes. Internal infections are the major cause of death.

Clinical Picture:

The initial HIV infection:

The incubation period is unknown but has been estimated to be 3 to 6 weeks, after which an acute mononucleosis-like syndrome develops in 50% to 70% of patients. There is fever, myalgia, arthralgias, pharyngitis, and a diffuse red eruption consisting of macules 0.5 to 2.0 cm in diameter. The symptoms resolve spontaneously within 8 to 12 days. Seroconversion (development of anti-virus antibodies) may take place within 1 week to 3 months. Initially, there is a transient rise in viral load and a short-lived fall in CD4+ T lymphocytes, and then there is a dramatic decline in viremia. The T-helper cells remain at a normal level of more than 500/microlitre.

Asymptomatic HIV infection:

After primary infection, viral dissemination occurs, and HIV-specific antibodies continue to be detectable in blood. CD4+ T lymphocytes continue to decrease but still >350/mL. Most patients have a period of "clinical latency" that lasts for years.

Symptomatic HIV infection:

- The clinical presentation of patients with HIV infections ranges from: chronic generalized lymphadenopathy, to subclinical and clinical T-cell deficiency.
- There is a strong association between the development of life-threatening opportunistic illnesses and the absolute number (per microliter of blood) or percentage of CD4+ T lymphocytes. As the number of CD4+ T lymphocytes decreases, the risk and severity of opportunistic illnesses increase. Infections normally controlled by cellular immunity occur with markedly increased frequency. They are severe and commonly disseminated. Most result from reactivation of previously acquired organisms. Concurrent or consecutive infections with a different organism are common. Infections can be controlled but are rarely curable.
- The revised Centers for Disease Control classification system for HIV infection categorizes persons on the basis of clinical conditions associated with HIV infection and CD4+ T-lymphocyte counts.

Progression to AIDS:

- Patients have a number of symptoms during disease progression. They exhibit prolonged constitutional symptoms such as chronic fatigue, night sweats, fever, weight loss, and diarrhea. They frequently have clinical manifestations of T-cell dysfunction such as mucous membrane disease (oral candidiasis, oral hairy leukoplakia) and dermatologic diseases (herpes simplex/zoster, fungal infections).
- AIDS is the end stage of HIV infection. It is characterized by a variety of unusual tumors (Kaposi's sarcoma and lymphomas) as well as opportunistic infections. CD4 count falls <200/mL. There appears to be a spectrum of disease, and long-term survival may be possible. The probability of survival is 49% at 1 year and 15% at 5 years.

Diagnosis:

I. Detection of viral antibodies in the serum:

- ELISA (sensitive).
- Western blot (confirmatory).

II. Detection of viral antigens in blood:

- P24

III. Detection of viral RNA in blood:

- Reverse transcriptase-PCR

The levels correlate with viremia (viral load) which is high early in the acute disease and late in AIDS stage (when the number of CD4+ cells is lowered).

IV.Measures of CD4+ T lymphocytes are used to guide clinical progression and therapeutic response.

Management:

- To date, there is no curative treatment for HIV that eventually will end in death of the infected person.
- Prevention is of utmost importance through prohibiting promiscuous sexual relations, avoiding sharing common needles, and ensuring effective sterilization of surgical instruments.
- Encourage casual social life contact with known HIV–infected persons, in view of taking enough precautions, to prevent antisocial behavior by these persons.
- Antimicrobial prophylaxis and antiretroviral therapies have been shown to be most effective within certain levels of immune dysfunction (monitored by CD4 count).

- Antiretroviral therapy should be considered for all persons with CD4+ T-lymphocyte counts of less than 500/mL.

- Prophylaxis against Pneumocystis carinii pneumonia (PCP), the most common serious opportunistic infection in AIDS, is recommended for all persons with CD4+ T-lymphocyte counts of less than 200/mL and for persons who have had prior episodes of PCP.

Questions

The most serious opportunistic infection in acquired immune deficiency syndrome (AIDS) is:

a. Chronic mucocutaneous candidiasis.

b. Pneumocystitis carnii.

c. Bilateral extensive herpes zoster.

d. Herpes simplex type II.

INTRODUCTION TO MALE INFERTILITY

Anatomy and structure of male reproductive system:

- The testes are the sites of sperm production. They are accommodated in the scrotum which is formed of thin skin devoid of fat, this to keep the testicular temperature some what lower than the internal body temperature which is necessary in sperm production. Testicular temperature is adjusted by the contraction or relaxation of cremasteric muscles and the dartos muscle.
- Each testis is divided into about 300 tiny lobes, each containing one to three coiled tubes in the walls of which the sperms develop. The total length of these seminiferous tubules is about 500 meter, and they join up into a single, highly coiled tube that forms a slug-shape mass in the outside of the testis, the epididymis. This is several meters long and it takes a number of days for sperms to traverse it. This time is necessary for their maturation. Sperms do not swim up these tubes; they are pushed up as a dense sludge, by the pressure of fluid from below.
- **The testes histologically are formed of**
  i. Seminiferous tubules.
  ii. Interstitial tissue.

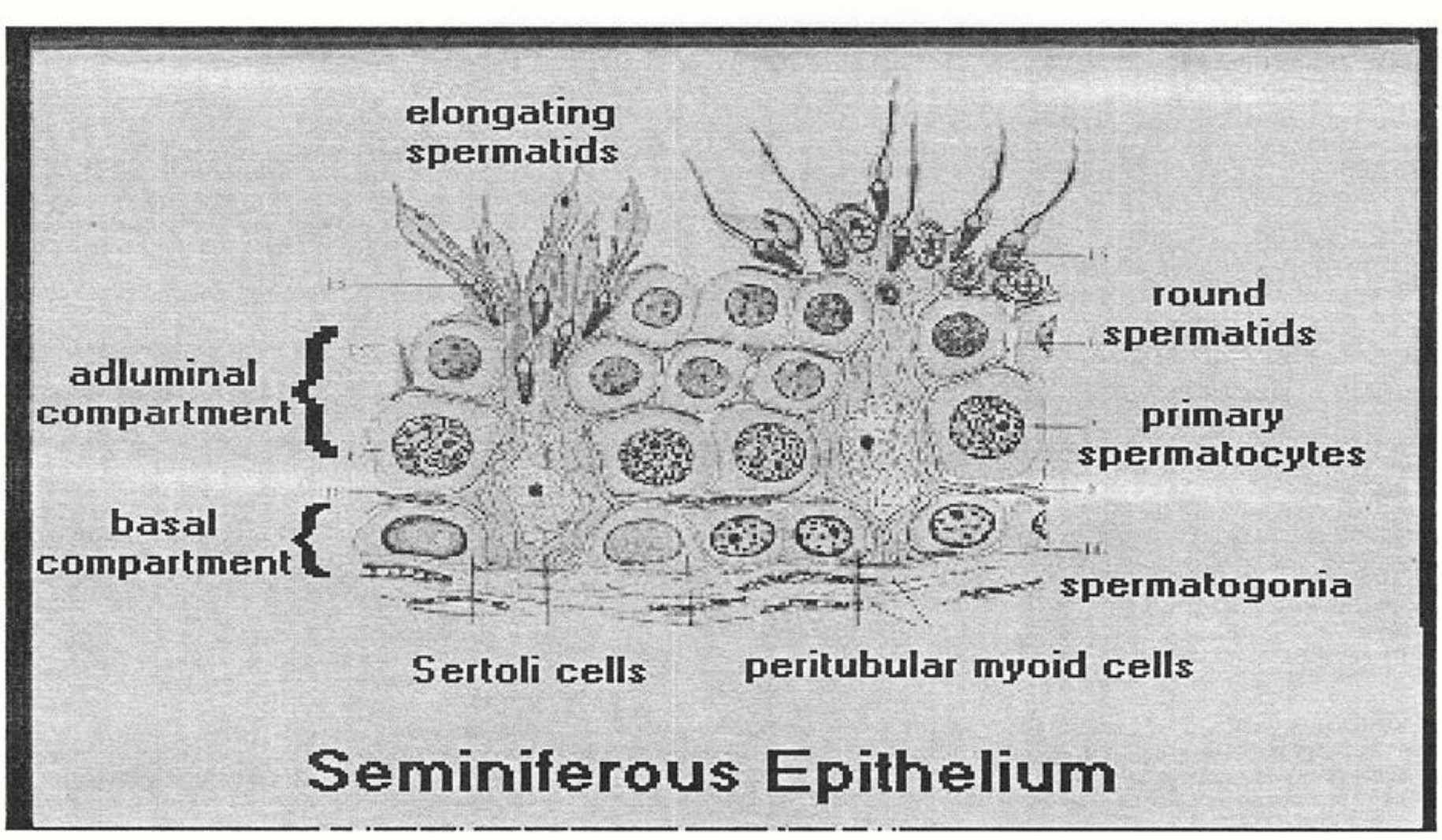


Fig. 12: The lining of seminiferous tubules includes germ cells and Sertoli cells.

I- The seminiferous tubules contain (Fig.12)

- The germ cells (spermatogonia, primary spermatocytes, secondary spermatocytes, spermatids and sperms).
- The sertoli cells.

Function of Sertoli cells

1. Formation of blood-testicular barrier: Sertoli cells are joined to each other by tight junctions constituting the blood-testicular barrier which prevents the passage of toxic substances into the lumen .
2. Phagocytosis of degenerated germ cells.
3. Mechanical support of germinal epithelium.
4. Provision of nutrients to the germ cells.
5. Secretory function: Under the effect of follicle stimulating hormone (FSH) of the anterior pituitary, Sertoli cells produce the following:

   - *Androgen-binding protein (ABP):* it binds with testosterone in the interstitial tissue, preventing its rapid metabolism to estradiol.
   - *Inhibin:* it is responsible for the negative feedback regulation of FSH.
   - *Müllerian Inhibitory Factor (MIF):* Secreted by fetal Sertoli cells during fetal development.
   - *Trasnferrin:* a significant marker of Sertoli cell function.
   - *Growth factors.*

II- The interstitial tissue

It lies between the seminiferous tubules. It contains the interstitial cells of Leydig which secrete testosterone, areolar tissue, collagen fibres, elastic fibres, blood vessels, and lymphatic vessels

Each epididymis ends in a vas deferens, the tube that carries the sperms up to the ejaculatory duct. Seminal vesicles are small, elongated sacs lying on either side of the prostate gland, just under the urinary bladder.(Fig.14) The prostate secretes about one third of the fluid content of the seminal ejaculate, the ducts of the seminal vesicles join the vas deference to form the ejaculatory ducts that open in the prostatic urethra. Most of the fluid content of the ejaculate (about two thirds) comes from the seminal vesicles. The urethra runs along the inside of the penis.

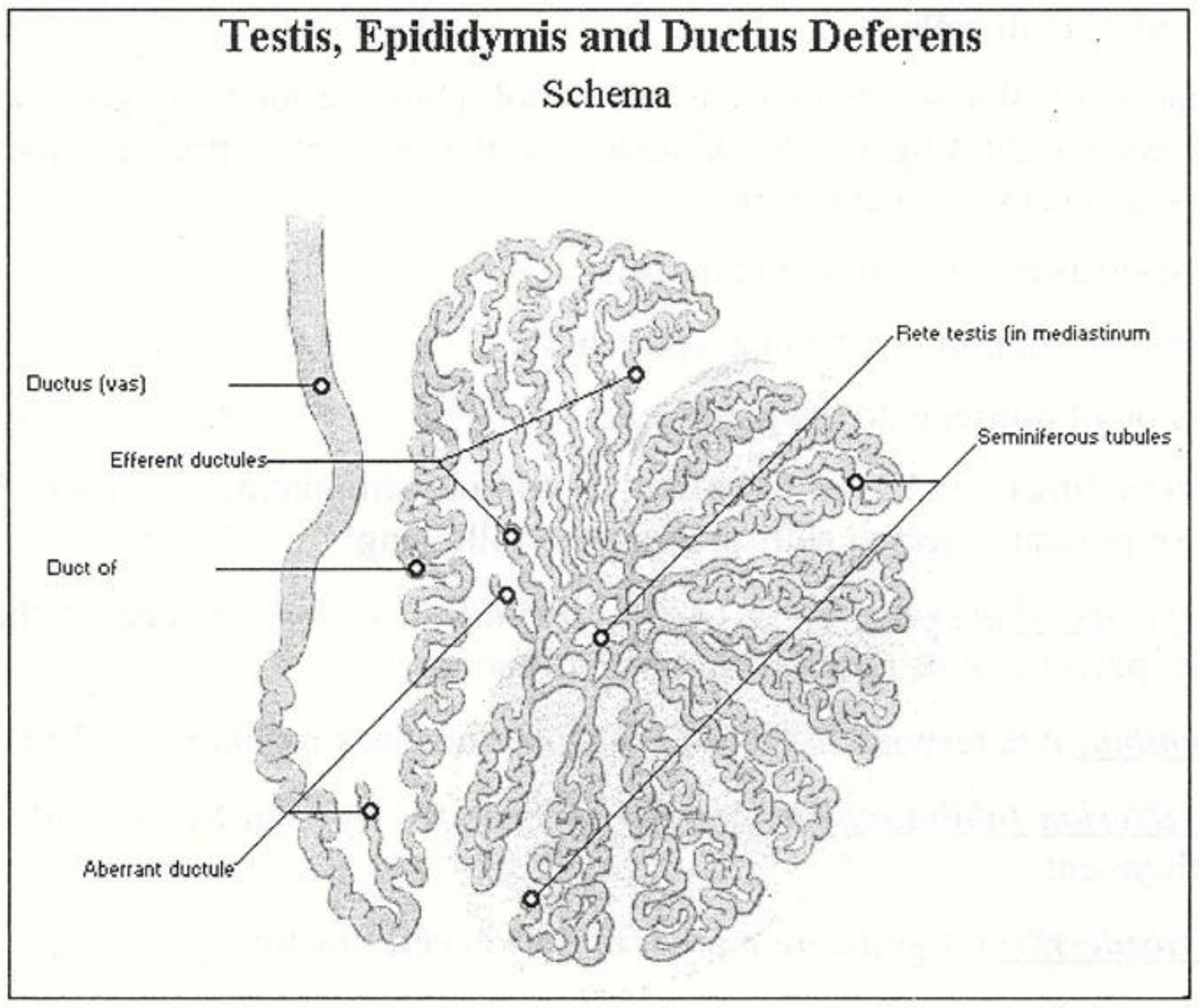


Fig. 13: Schematic illustration of testis, epididymis and vas deferens.

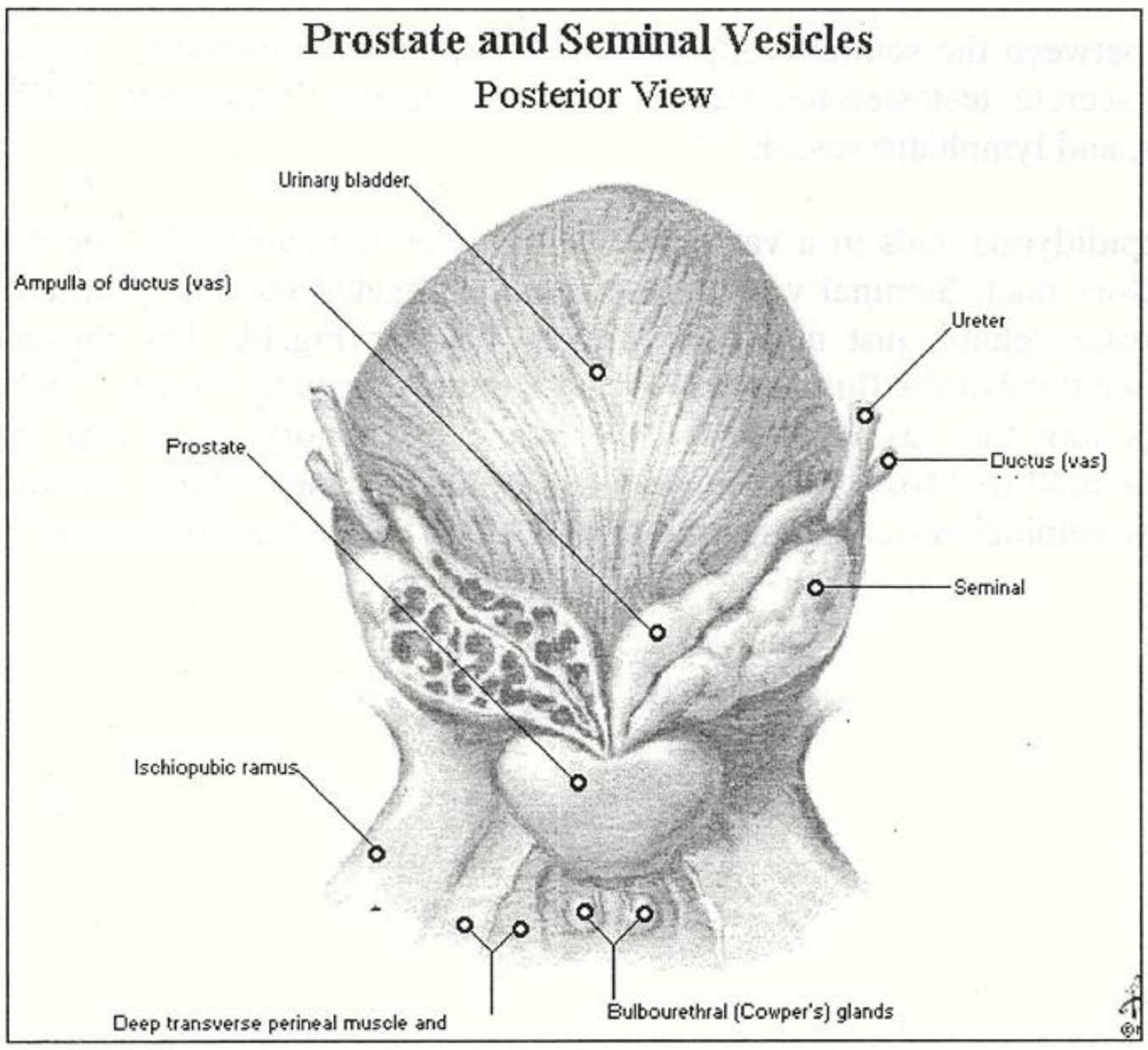


Fig. 14: Posterior view of the prostate and seminal vesicles.

The testis serves two main functions:

1. Endocrinal (secretion of androgens).
2. Spermatogenesis

Androgens (mainly testosterone) are secreted by the interstitial cells of leydig then enter the nearby semineferous tubules with the help of androgen binding protein (ABP) secreted by the Sertoli cells to help spermatogenesis.

* Testosterone is converted peripherally into dihydrotestosterone (DHT) by the enzyme 5-α reductase.

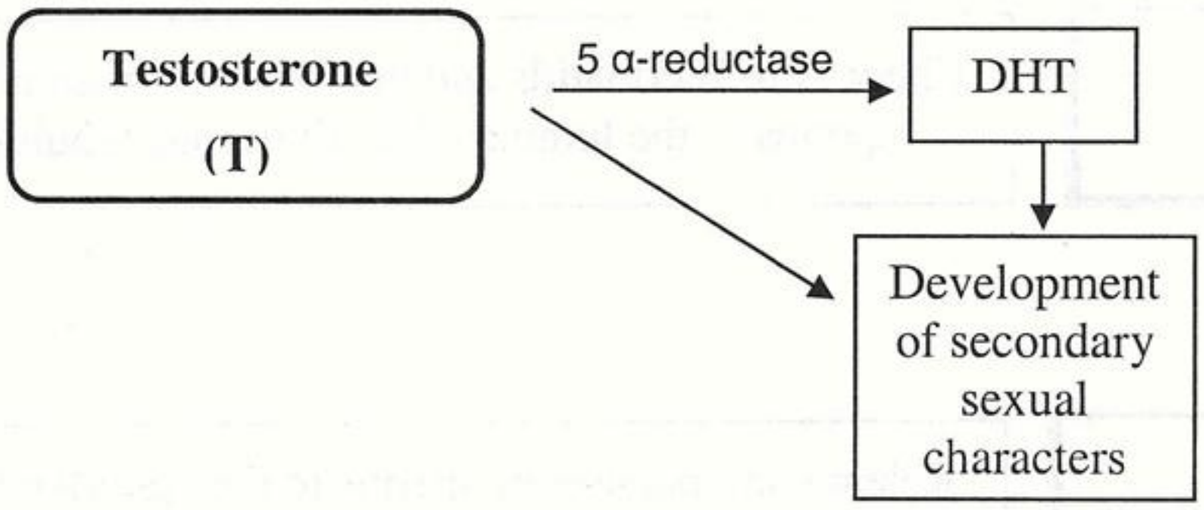


Fig. 15: Testosterone action.

Endocrinal control of spermatogenesis:

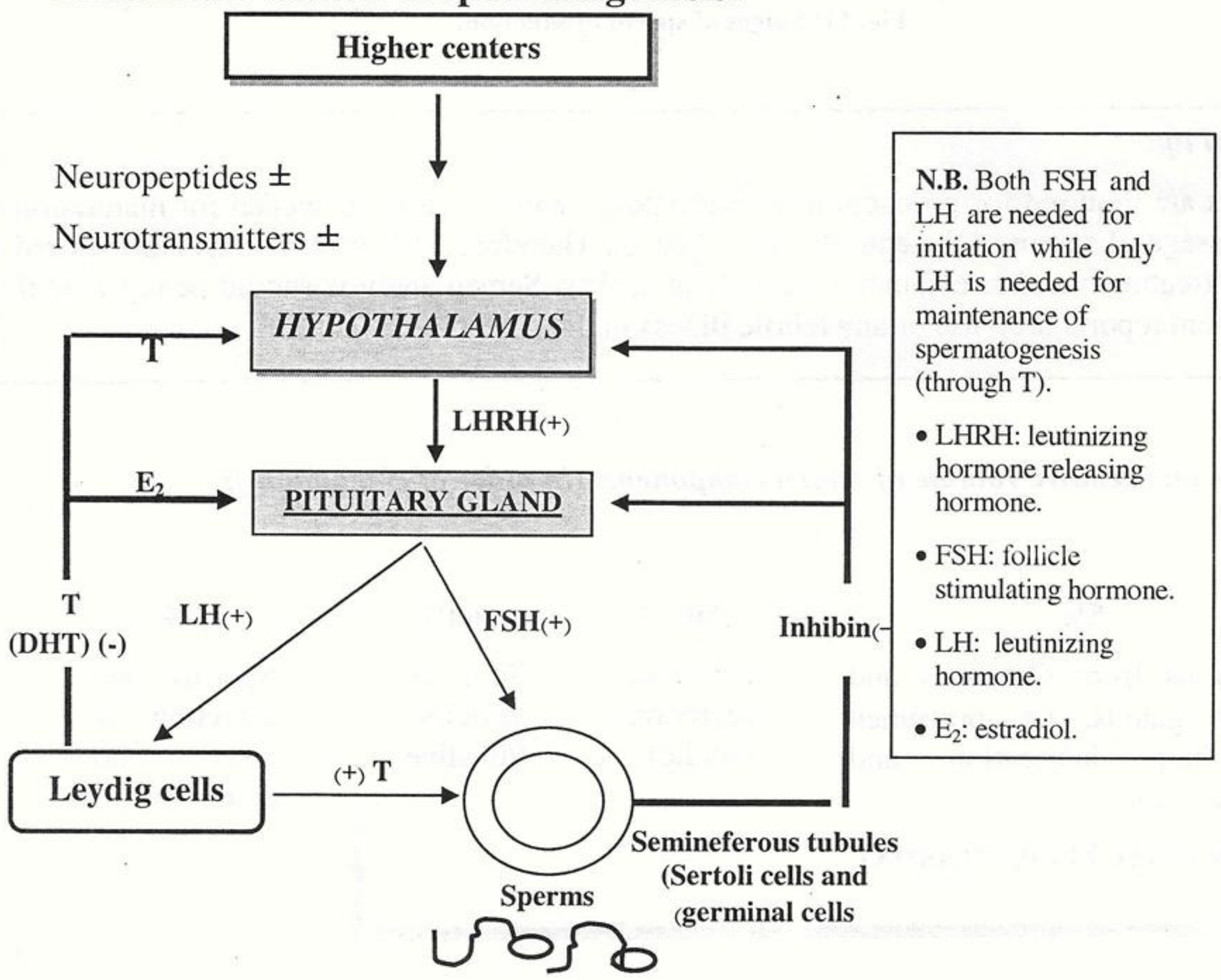


Fig.16: Hormonal control of spermatogenesis.

Sperm production passes into three stages:

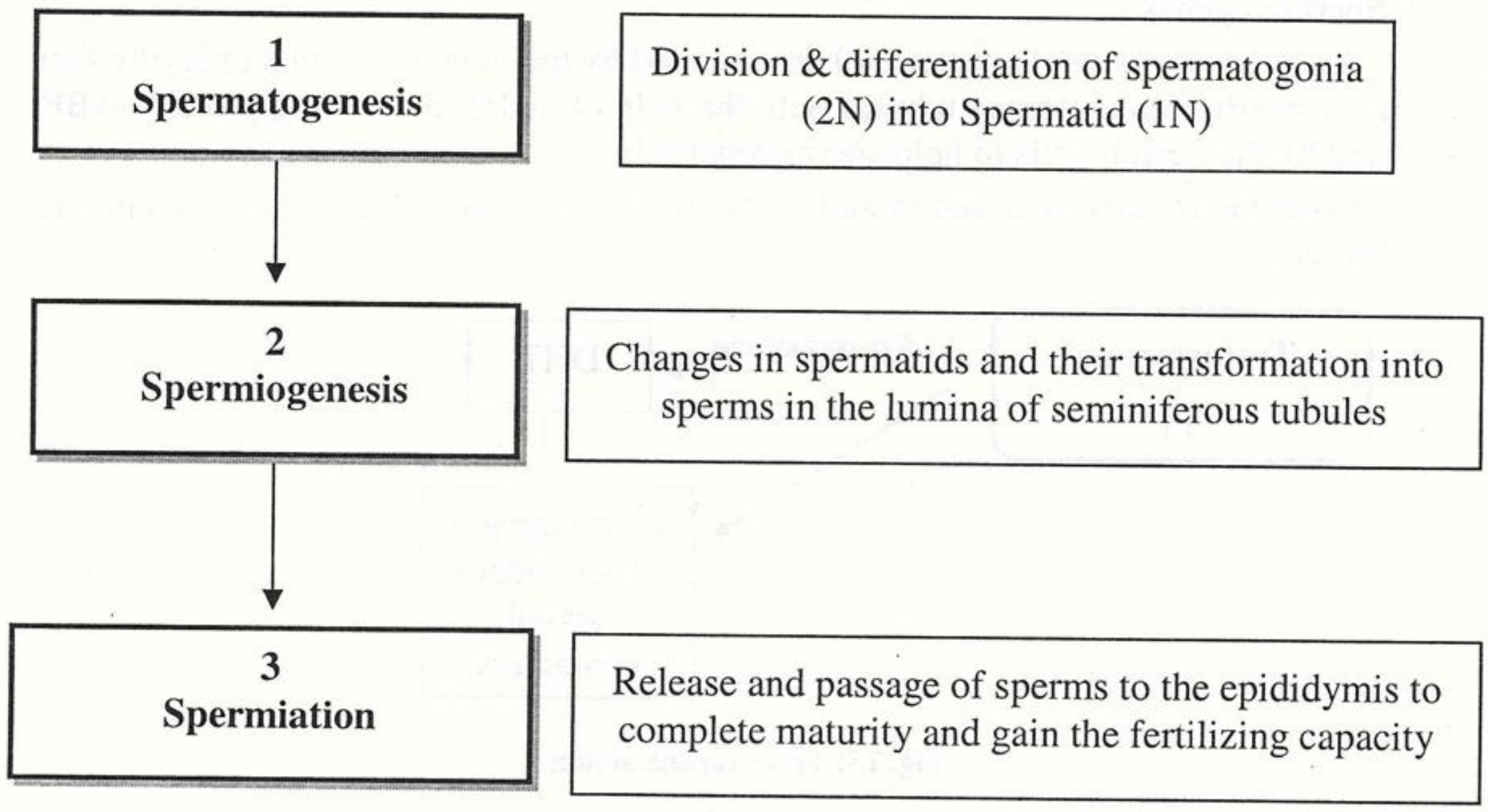


Fig. 17: Stages of sperm production.

Clinical tip:

75 days are required for production of spermatozoa and 15 days are needed for maturation and passage of sperm in the epididymis (90 days). Therefore, at least 3 months are required for the treatment and re-evaluation of male infertility. Semen analysis should be repeated if the patient reports drug use or any febrile illness in the preceding 3 months.

Source and relative volume of semen components (in order of ejaculation):

5%	+	**30%**	+	**60%**	+	**5%**
Secretions from Cowper's and Littre's glands, i.e. prosemen that help lubrication and neutralization		Prostatic secretion (acidic)		Seminal vesicles (alkaline)		Sperms and secretion of testes and epididymis

THE SEMINAL FLUID (PLASMA)

Evaluation of male infertility:

A. History:

1. History of infertility:

- Duration of infertility.
- Previous evaluation and / or treatment(s)
- Previous pregnancies, whether with another partner or with the present partner.
- Evaluation of the partner (female age whether above or below 35 is important, as well as history of infertility in a previous marriage / relationship).
- History of gonadotoxins, smoking, alcohol, marijuana, and cocaine.

2. Sexual history:

- Erectile dysfunction (ED).
- Use of spermicidal lubricants during intercourse.
- Timing and frequency of intercourse. The most effective frequency is every other day around ovulation because sperm survive for about 2 days in normal cervix.
- Assessment of libido which crudely reflects serum testosterone levels.

3. Ejaculate history:

- History of premature ejaculation.
- Normal orgasm with low or absent semen volume suggests retrograde ejaculation, and requires examination of post-ejaculatory urine specimen for the presence of sperm.

4. Medical history:

- Approximate age of onset of puberty.
- History of cryptorchidism (hidden testis).
- History of undescended testicle.
- History of mumps orchitis.
- History of genital tract infections e.g. STDs, T.B., or bilharziasis.

5. Drug history:

- Cancer chemotherapy has a dose-dependent and potentially devastating effect on the testicular germinal epithelium.
- History of any systemic illness:
- Loss of libido associated with headaches, visual abnormalities and galactorrhea may suggest pituitary tumor.
- History of diabetes. Diabetic neuropathy may result in either retrograde ejaculation or erectile dysfunction (ED).

6. **Surgical history:**
   - History of bladder neck operation (may cause retrograde ejaculation).
   - History of retroperitoneal lymph node dissection for testicular cancer (may cause retrograde ejaculation or absent emission).
   - History of varicocelectomy.
7. **Family history:** Chromosomal abnormalities.

B. Clinical examination:

- Clinical examination should be performed in a warm room by an examiner with warm-gloved hands. Contraction of the dartos muscle induced by a cold examining hand makes examination of the scrotum and its contents difficult.
- The patient should be completely undressed, in standing position with his arms outstretched.

I. General examination:

- Gynecomastia.
- Body hair distribution.
- Affection of muscular development and mass.
- Skeletal proportion.

II. Local examination:

a. Penis:

Size, position of urethral meatus (exclude hypo or epispadias), penile curvature, any nodules or masses (exclude peyronie`s disease). The urethra should be milked for any discharge.

b. Examination of testes:

Size, consistency, and testicular sensation should be assessed. Transillumination of the scrotum in a darkened room helps differentiate solid from cystic masses i.e. excludes hydrocele.

c. Examination of the epididymis:

The normal epididymis, posterolateral to the testis, is soft and barely palpable. Induration, tenderness, nodularity, or irregularities are suggestive of epididymal pathology.

d. Examination of the spermatic cord:

- Vas deferens: It is felt as a rolling firm cord inside the spermatic cord on both sides between the index and thumb. The vas deference should be palpated bilaterally.
- Detection of varicocele: Large varicosities are readily seen through the relaxed scrotal skin in a warm room with the patient standing. Small varicoceles may be appreciated as a distinct impulse and palpable dilation of the internal spermatic veins during the Valsulva's maneuver, confirmation is achieved by Doppler examination

e. Examination of accessory genital organs (through P/R):

- Prostate is examined per rectum for evaluation of its size, consistency, and any tenderness. Prostatic smear is examined under microscope for detection of prostatitis (> 20 pus cells/ HPF= chronic prostatitis).
- Seminal vesicles are normally not felt. When indurated, they can be felt on both sides on the upper pole of the prostate.

C. Laboratory investigations:

I. Semen analysis:

- Semen can be collected by masturbation (best), coitus interruptus, electro-ejaculation and condom (it should be dry with no spermicides).
- Semen sample should be collected after a 3-5 days of sexual abstinence.

WHO recommendations for semen analysis

Adequate semen parameters	Abnormal semen parameters
• Volume: 2-6 ml after 3-5 days of sexual abstinence. • PH: 7.2-8 (alkaline) • Consistency: viscous. • Liquefaction: within 10-20 minutes • Sperm count: >20 million/ml (normozoospermia). • Percent of motile sperms: >60% in the first hour. • Forward progressive motility • Abnormal morphology < 30% • Percent of viable sperms: >50% • Fructose content: 1200 □g/ml • No sperm agglutination • Pus cells 3-5/High power field	**Spermia: refers to semen volume** • Aspermia: No semen, e.g. retrograde ejaculation. • Hypo- or oligospermia: low semen volume< 1.8 ml.(eg: bilateral cong. absent vas, ejaculatory duct obstruction) • Hyper- or polyspermia: >6 ml • Pyospermia: > 6 pus cells/high power field **Zoospermia: refers to spermatozoa** • Azoospermia: No spermatozoa in semen (obstructive or functional) • Oligozoospermia: sperms <20 million/ml • Polyzoospermia: > 250 million/ml • Asthenozoospermia: decreased motility of sperms. • Teratozoospermia: >35% abnormal spermatozoa • Globozoospermia: round heads of sperms (no acrosomes) • Necrozoospermia: All sperms are dead.
N.B. To differentiate immotile from dead sperms, supravital stain with Eosin-Negrosin will stain heads of dead sperms red.	

Causes of Azoospermia:

Obstructive: obstruction in the excretory ducts

- Epididymal obstruction (post inflammatory).
- Vasal obstruction (congenital absence, surgical ligation, inflammatory obstruction)
- Ejaculatory ducts obstruction.

Functional:

- Congenital: cryptorchidism (undescended testes)
- Chromosomal: Kleinfelter syndrome 47 XXY, Down syndrome
- Traumatic: bilateral trauma, orchiectomy, torsion.
- Hormonal: hypogonadotrophic hypogonadism (hypopituitarism).
- Inflammatory: orchitis(mumps orchitis...).
- Neoplastic: testicular tumors (teratoma, seminoma).
- Exposure to radiation and chemotherapy

II. Hormonal evaluation:

FSH, LH, testosterone, Prolactin and thyroid hormonal assessment.

D. Testicular biopsy:

Its diagnostic role is to differentiate functional azoospermia (Sertoli cell only syndrome, spermatogenic arrest), from obstructive azoospermia, where normal spermatogenesis is noted.

Treatment of male infertility:

1. Medical, e.g. hormonal.
2. Surgery for correction of obstruction, varicocele ligation
3. Assisted reproductive techniques (ART) e.g. Artificial insemination husband (AIH), in vitro fertilization (IVF), intracytoplasmic sperm injection (ICSI).

Questions

Functions of sertoli cells include all of the following except:

a. Phagocytosis of degenerated germ cells.

b. Mechanical support to germ cells.

c. Sharing in the formation of blood testicular barrier.

d. Secretion of testosterone.

INTRODUCTION TO SEXOLOGY

Anatomy of the penis (Fig. 18):

The penis has a fixed root and a body that hangs free.

a. The body of penis:

It consists of 3 corporeal bodies, two corpora cavernosa dorsally and a single corpus spongiosum ventrally which contains the urethra and expands distally to form the glans penis.

b. The root of penis consists of:

1. **Two crura**, the proximal extension of the 2 corpora cavernosa, which are attached to the ipsilateral ischiopubic ramus and covered by the ischiocavernosus muscles.
2. **The bulb**, the proximal extension of the corpus spongiosum. It lies between the 2 crura and is covered by the bulbocavernosus muscle.

The erectile tissue of the corporeal bodies is composed of a network of interconnected sinusoidal spaces separated by trabeculae consisting of connective tissue and smooth muscles.

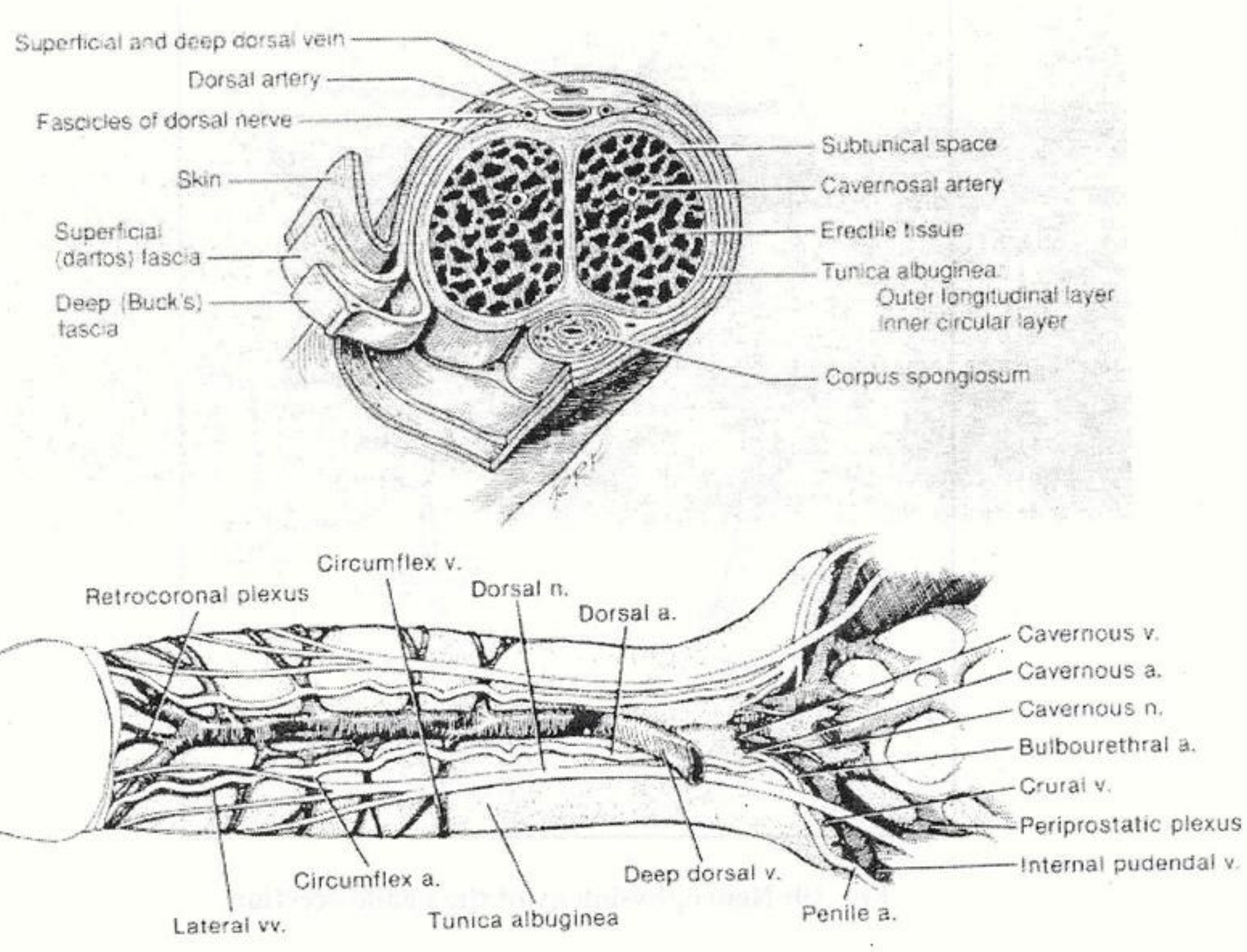


Fig. 18: Anatomy of the penis.

Neurophysiology and neuroanatomy (Fig.19):

The penis is supplied by autonomic and somatic nerve fibers:

1. ***Autonomic fibers reach the penis through cavernous nerves carrying fibers from:***

   Thoracolumbar sympathetic pathway, arising from T11 to L2 segments of the spinal cord. The neurotransmitter is mainly norepinephrine that leads to vasoconstriction, i.e. anti-erectile or flaccid state.

   Sacral parasympathetic (S2 to S4). The neurotransmitter is mainly acetyl choline which acts through nitric oxide production and consequent transformation of the guanosine triphosphate (GTP) into cyclic guanosine monophosphate (cGMP) with ultimate vasodilatation (erectile state)

   However, nonadrenergic-noncholinergic neurotransmitters are also released from autonomic nerve ending .e.g. VIP, NP-Y and substance P.

2. ***Somatic fibers (peudendal nerve):***

   - Sensory from the glans penis & penile shaft reach the sacral segments (S2-S4).
   - Motor fibers to ischiocavernosus and bulbocavernosus muscles.

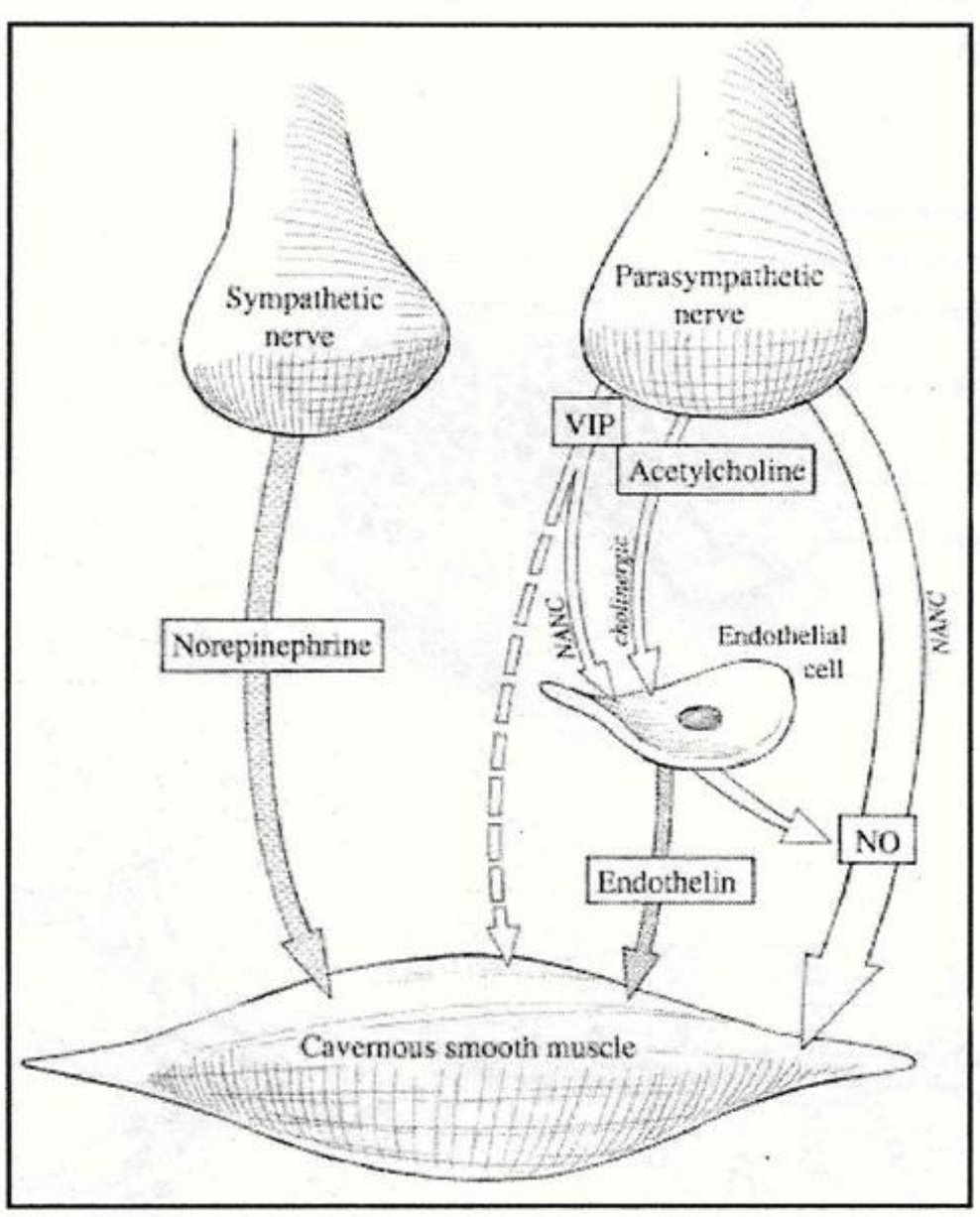


Fig. 19: Neurophysiology of the penile erection.

Mechanism of penile erection:

- For normal erection to occur, interplay between intact neurogenic pathways and adequate haemodynamics of the penis is essential; in presence of normal endocrinological status and normal psychological status.
- Following orgasm, vasoconstriction will occur & cGMP is degraded under the influence of phosphodiestrase type 5 (PDE 5). This leads to decreased blood flow and consequent decreased compression & venous resistance. Ultimately, the blood flows from corpora cavernosa and detumescence occurs.

N.B. During sleep, erection occurs in Rapid eye movement (REM) phase followed by detumescence in non REM phase and so forth. This is called "nocturnal penile tumescence" (NPT)

ERECTILE DYSFUNCTION (ED)

Definition:

It is the inability to achieve or maintain erection sufficient for vaginal penetration.

Etiology:

I. Organic:

- Vasculogenic (arterial insufficiency and/or venous leakage).
- Neurogenic.
- Endocrinal.
- Mixed.

II. Psychogenic:

Intra- or interpersonal factors (normal morning erection and NPT)

Evaluation of erectile dysfunction:

(A) History:

I. Personal history:

Age, marital status (whether this is the first marriage or there is a previous one), occupation (stability of work, nature of work i.e. manual or mental, and personal satisfaction with the kind of work and its environment), special habits (smoking, alcohol, recreational drugs), and sleep pattern or sleep disorders.

II. Sexual history:

1. Sexual development (negative attitude to sex, onset of puberty, masturbation, first sexual experience).
2. Sex education.
3. Depression, anxiety, or phobias.

III. History of ED:

- Onset, course, duration.
- Desire: normal, increased, diminished or absent.
- Sexual arousal: response to erotic situations/material.
- Erection:
  - In the morning, during masturbation.
  - Degree of penile rigidity: easy intromission or with manual help.
- Ejaculation: normal, premature, or retarded.
- Orgasm: present, absent, or pleasureless

IV. Medical and surgical history

A history of peripheral vascular disease, coronary artery disease, hypertension, diabetes, hepatic or renal disease, chronic debilitating disease, neurological diseases e.g. stroke, cancer or its treatment i.e. chemotherapy or radiation, prostatitis, penile or pelvic trauma, and pelvic operations e.g. prostatectomy.

V. History about the wife/partner:

Complete sexual history of the wife/partner including sexual attitude and orientation.

(B) Clinical examination:

1) General examinations:

- Secondary sex characteristics to detect signs of hypogonadism.
- Signs of systemic disease that may lead to ED.
- Examination of the breast for swelling or enlargement or any discharge which may indicate a hormonal or a drug causes of the ED.
- Femoral and lower extremity pulses should be examined. Temperature or any trophic changes should be checked.
- Careful neurological examination for all patients with diabetes and neurodegenerative disorders to elicit evidence of peripheral neuropathy.

2) Genital examination:

- Penis: size e.g. micropenis, Peyronies' plaque or cordee, examination of dorsal penile pulsation, detection of balanitis or urethritis (urethra should be milked for any discharge).
- Testes: size, consistency, varicocele, and testicular sensation.
- Digital rectal examination (P/R) for assessment of prostate size, or presence of any tenderness and / or congestion and for assessment of anal sphincter tone.
- Testing the genital and perineal sensations.

(C) Rigiscan:

It is a computerized system that measures circumferential changes & rigidity of the penis during sleep. Normal rigiscan results are suggestive of psychogenic erectile dysfunction (ED).

(D) Evaluation of penile arteries:

These include Doppler, penile duplex U/S, injection of vasoactive substances (ICI), e.g. papaverine, PGE1, trimix, to induce artificial erection.

(E) Evaluation of penile venous system:

Cavernosometry and cavernosography.

Treatment:

- **Medical:**
  - Hormones (if endocrinal deficiency is documented).
  - Intracorporeal injection (ICI) of vasoactive substances.
  - PDE5 inhibitors as Sildenafil citrate (® Viagra).
  - Others: Yohimbine, bromocriptine (in hyperprolactinemia)
- **Vaccum device**
- **Surgical:**
  - Penile implants (penile prosthesis).

PREMATURE EJACULATION

Definition:

- The inability of the male to control ejaculation long enough to satisfy his partner in at least 50% of their sexual encounters, when there is no female orgasmic dysfunction.
- The notion of premature ejaculation is very subjective. Ejaculation occurring within 2 minutes of penetration may be considered by some couples as perfectly satisfying, while by others as totally unacceptable.

Etiology:

Premature ejaculation is mainly **psychogenic** in origin.

1) **Mal-conditioning:** If a man is unable to control his ejaculation, he simply has not learned to do so. Therefore, the man becomes conditioned to fast ejaculation. Examples:
   - Making love under conditions of fear of being discovered, e.g. in back seat of a car or in the parent's house.
   - Encouragement for rapid ejaculation from a prostitute.
   - Masturbation, with lack of the motive to prolong sexual activity due to the absence of a sexual partner.

2) **Anxiety:** related to performance, or satisfaction of the wife/partner.

3) **Organic causes (rare):** Chronic prostatitis, and multiple sclerosis.

Treatment:

- Reassurance of the couple and explanation of the problem with basic information about the physiology of ejaculation to correct any sexual false ideas.
- **Drugs:** several drugs are claimed to be beneficial e.g. Thioridazine (Meleril®), clomipramine (Anafranil®) and selective serotonin reuptake inhibitors (SSRI).
- **Sex therapy:**

  It aims at increasing the pre-ejaculatory period by training the male to control the ejaculatory reflex through several exercises and techniques, e.g. squeeze technique.
- Local anesthetics or condoms (decrease peripheral sensation).

Questions

- The flaccid state of the penis is due to the constant release of:
  a. Acetyl choline .
  b. Nitric oxide.
  c. Norepimephrine.
  d. Prostaglandine E.
- Rigiscan is used mainly for:
  a. Evaluation of penile venous system.
  b. Scanning of cavernous tissue.
  c. Recording of penile rigidity.
  d. Registering of penile nerve conduction.

Section 3

ATLAS OF DERMATOLOGY

Pyogenic Infections of the Skin



Fig. (20a): Impetigo contagiosum.



Fig. (20b): Circinate impetigo.



Fig. (21): Ecthyma.



Fig. (22): Boil.



Fig. (23): Cellulitis.



Fig. (24): Intertrigo.



Fig. (25): Paronychia.



Fig. (26): Erythrasma

Mycobacterial Infections



Fig. (27): Lupus vulgaris.



Fig. (28): Lepromatous leprosy.

Viral Infections of the Skin



Fig. (29): Herpes simplex.



Fig. (30): Chickenpox.



Fig. (31): Herpes zoster.

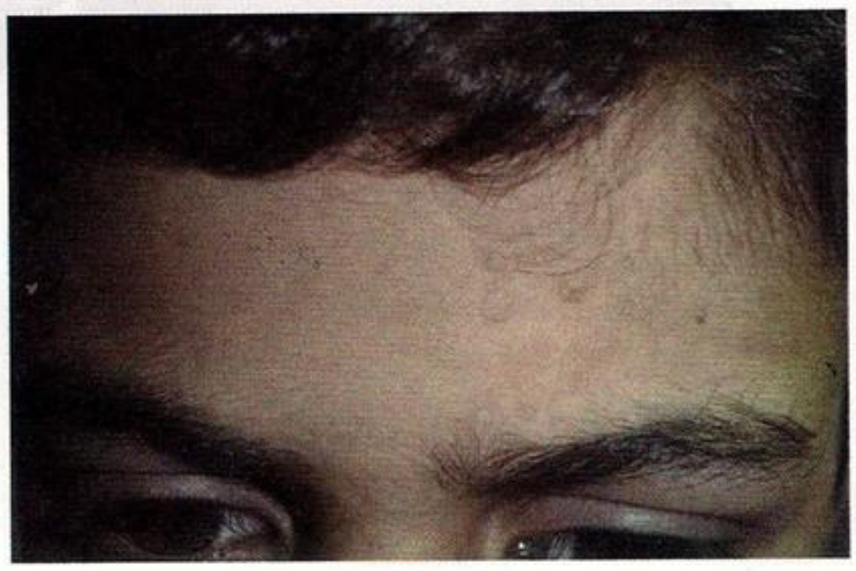


Fig. (32): Plane warts.



Fig. (33): Verruca Vulgaris.

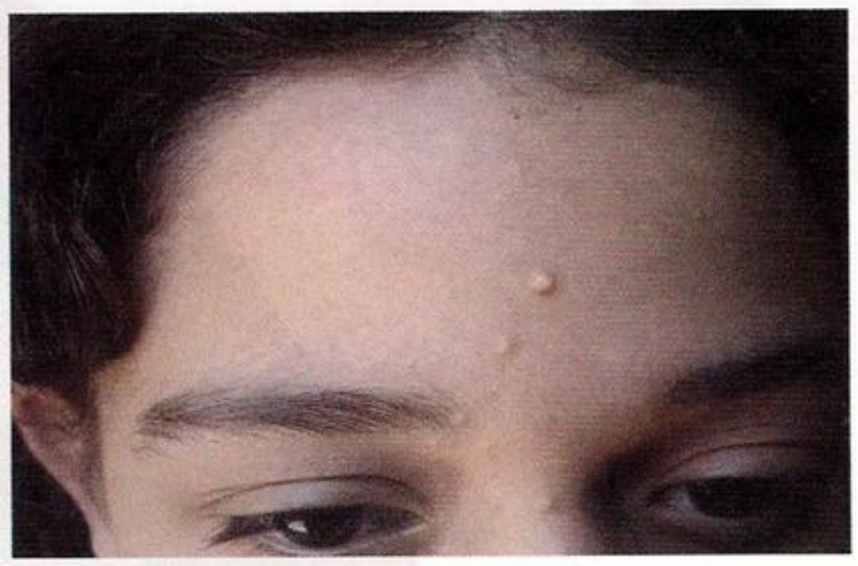


Fig. (34a): Molluscum contagiosum.



Fig.(34b): Molluscum contagiosum (genital).

Parasitic Infestations



Fig. (35a): Scabies.



Fig. (35b): Scabies.



Fig. (36): Pediculosis of the Scalp.

Fungal Infections of the Skin



Fig. (37): Scaly ring worm.



Fig. (38): Kerion.



Fig. (39): Tinea corporis.



Fig. (40): Onychomycosis.

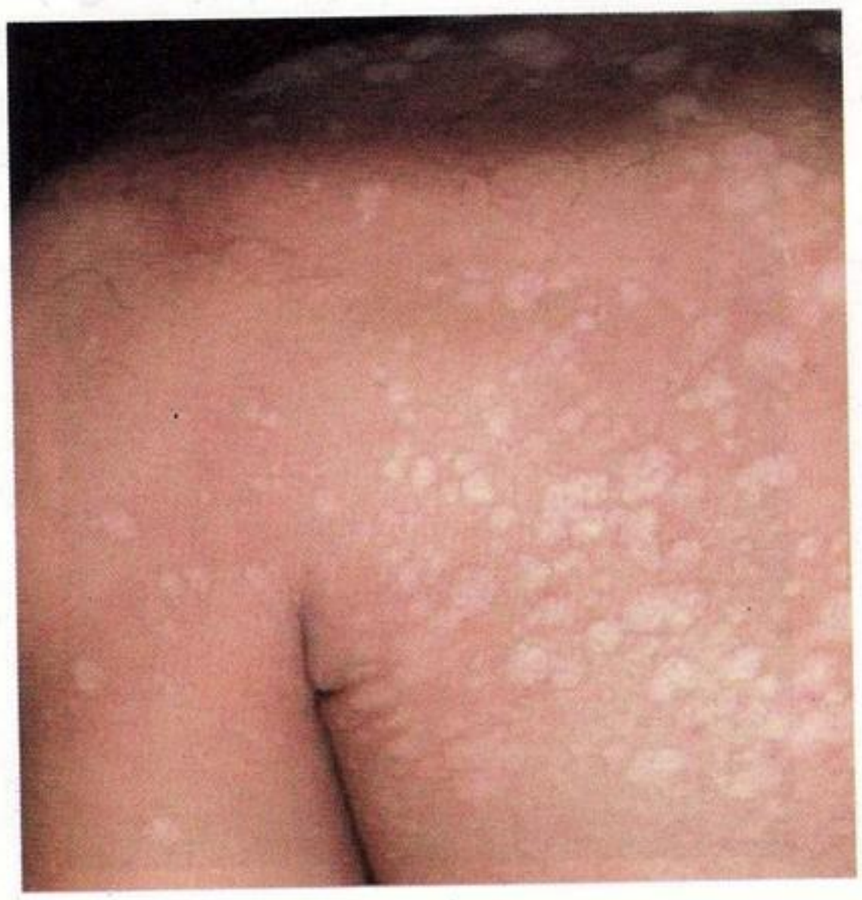


Fig. (41): Pityriasis Versicolor.



Fig. (42): Macerated toe web.

Allergic Skin Diseases



Fig.(43a): Contact dermatitis (acute).



Fig. (43b): Contact dermatitis (subacute).



Fig.(44): Napkin dermatitis.



Fig.(45): Atopic dermatitis (infantile stage).



Fig.(46): Atopic dermatitis (childhood stage).

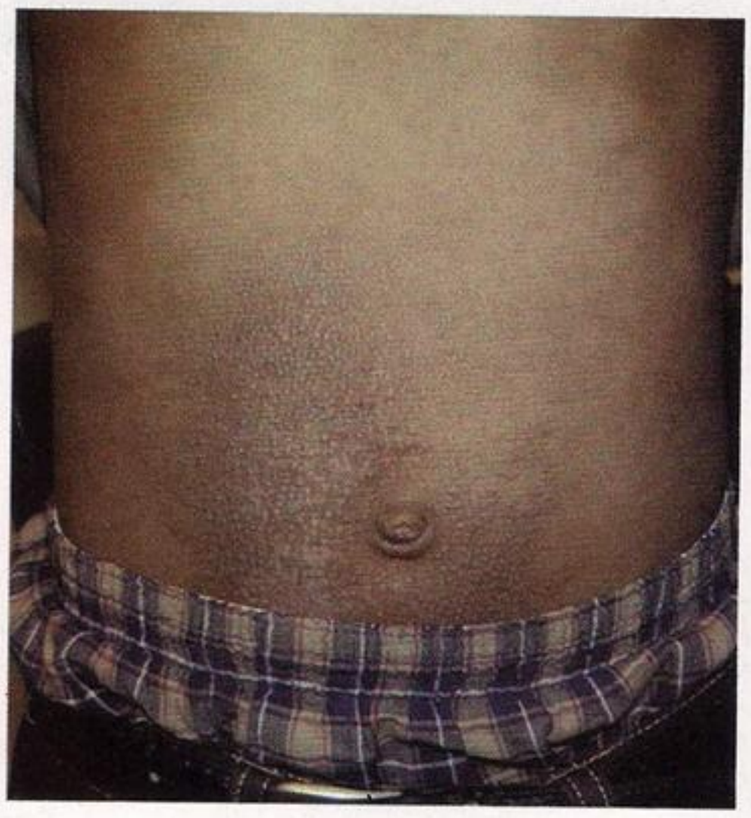


Fig.(47): Atopic dermatitis (adult stage).



Fig.(48): Stasis dermatitis.



Fig.(49): Pityriasis alba.

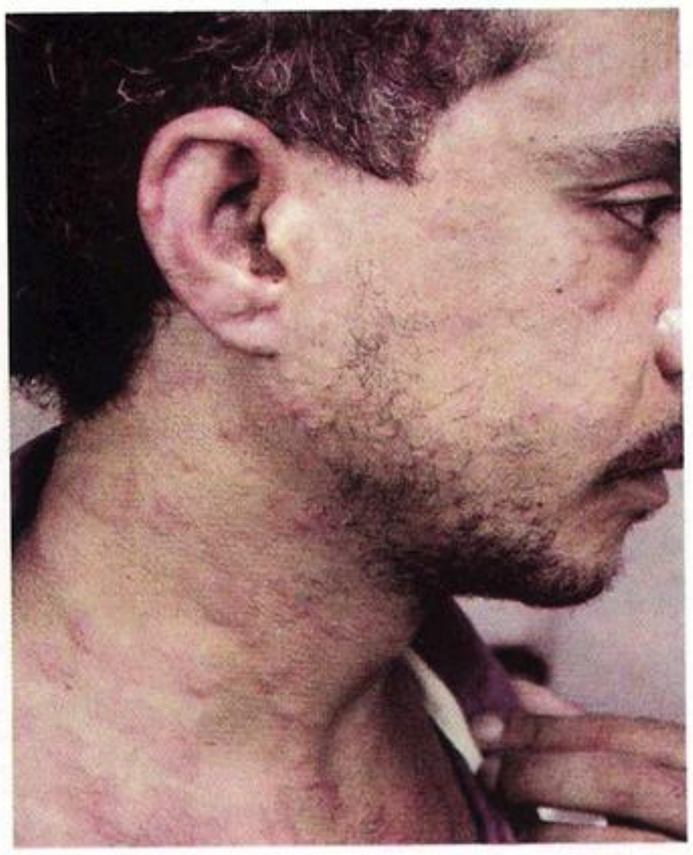


Fig. (50a): Urticaria.



Fig. (50b): Angioedema.



Fig. (51): Erythema Multiforme (Iris lesion).

Diseases of sweat and sebaceous glands



Fig. (52): Miliaria.



Fig. (53a): Acne vulgaris.



Fig. (53b): Acne vulgaris.

Papulosquamous Eruptions



Fig. (54): Plaque psoriasis.



Fig. (55): Pityriasis rosea.



Fig. (56a): Lichen planus.



Fig. (56b): Lichen planus.

Connective Tissue Diseases

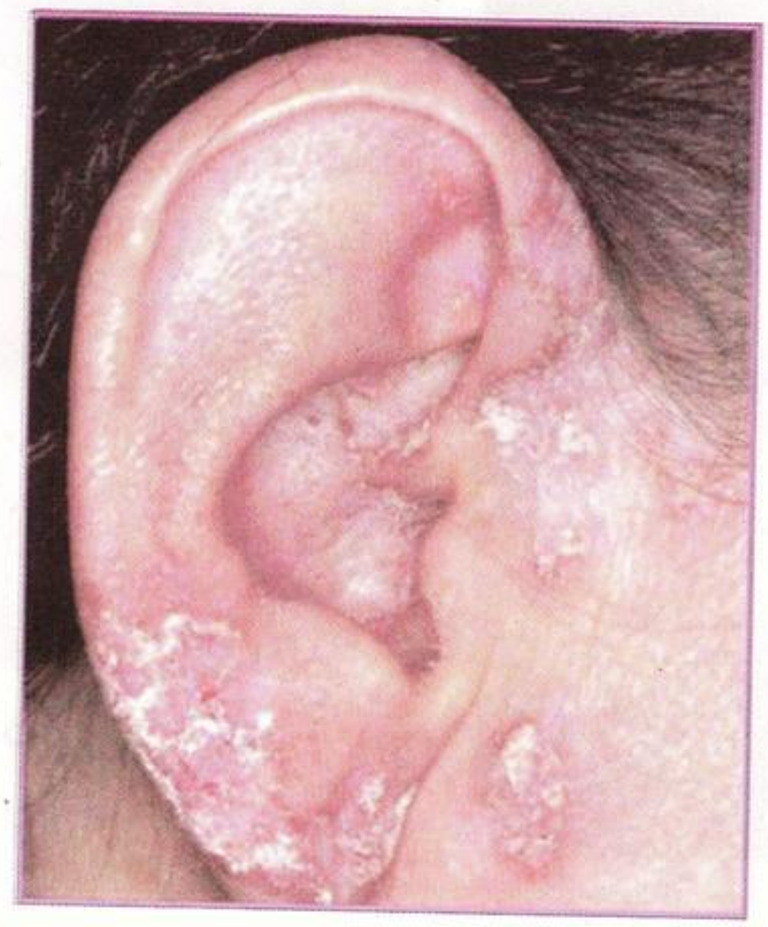


Fig. (57a): Discoid lupus erythematosus.



Fig. (57b): Discoid lupus erythematosus – Cicatricial alopecia.

Diseases of the Hair



Fig.(58a): Alopecia areata (scalp).

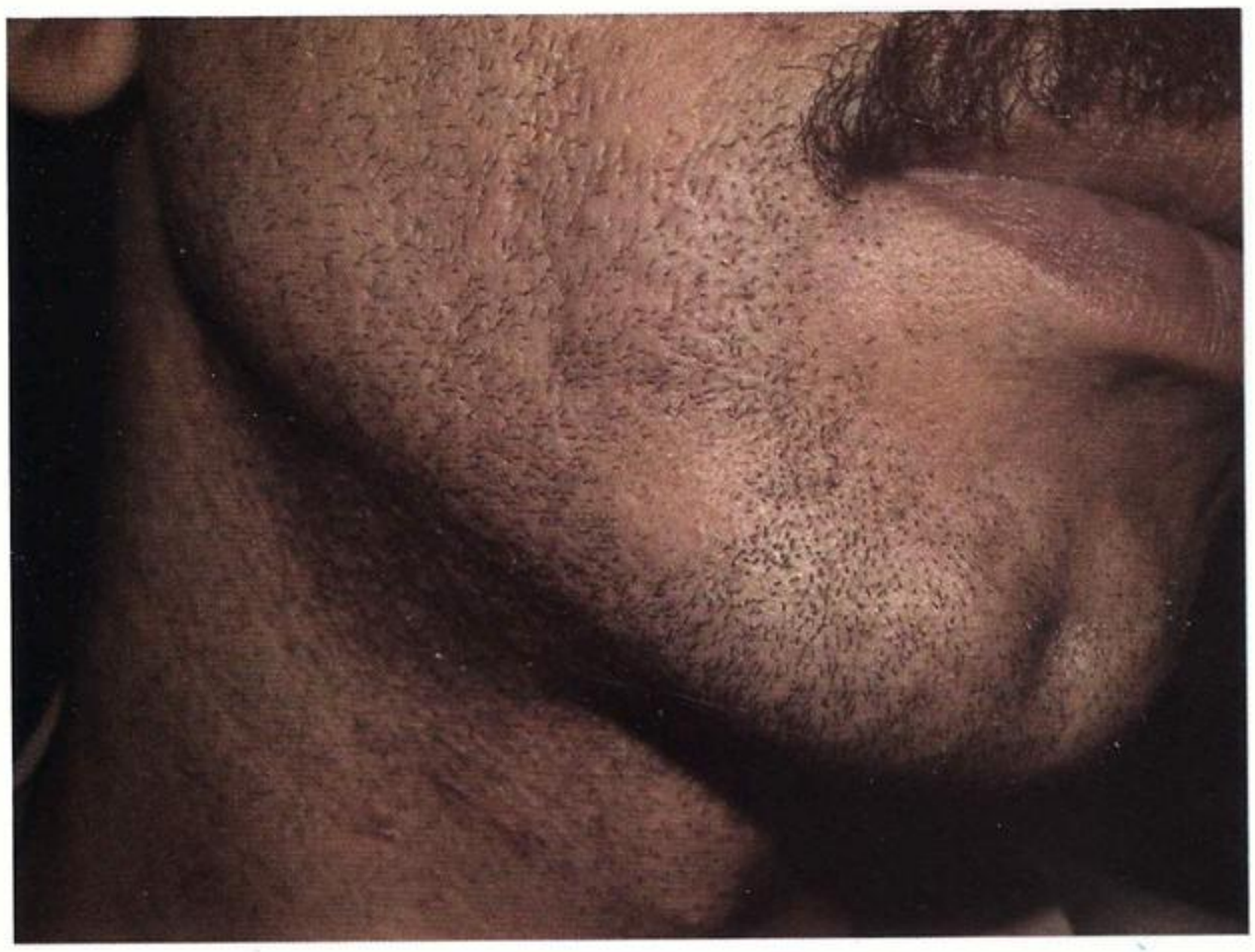


Fig.(58b): Alopecia Areata (beard).

Pigmentary Disorders of the Skin



Fig.(59): Vitiligo.



Fig. (60): Chloasma.

Translation to Arabic Terms

Acne vulgaris	حب الشباب
Alopecia	ثعلبه
Boil.....	خراج
Cellulitis.....	التهاب خلوى
Chickenpox	جديرى مائى
Chloasma.....	كلف
Carbuncle	جمرة
Erysipelas.....	حمرة
Gonorrhea	سيلان
Herpes zoster.....	حزام نارى
Impetigo	حصف
Leprosy	جذام
Lichen planus	حزاز
Lupus erythematosus	ذئبة حمراء
Miliaria.....	حمو النيل
Pediculosis	قمل
Pityriasis rosea	نخالة وردية
Pityriasis versicolor.....	نخالة مبرقشة
Psoriasis	صدفية
Scabies	جرب
Scleroderma	تصلب الجلد
Tuberculosis.....	سل
Vitiligo	بهاق
Warts.....	سنط