Essentials in Dermatology, Venereology & Leprology

Foreword
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Essentials in Dermatology, Venereology & Leprology

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Foreword
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The Health Sciences Publisher
New Delhi | London | Philadelphia | Panama
How this Book is Useful?

Features

• This book provides all essentials for learning Dermatology, Venereology and Leprosy to the undergraduates and PG aspirants.
• Text is presented in a bulleted format.
• A simplified concept has been given with more than 600 clinical photographs, over 100 illustrations and histological representations of various skin diseases and their presentation.
• Footnotes that elaborate certain words of text is the special feature of this book.
• Many flow charts and tables have been added for a quick grasp of the related topic.
• Differential diagnosis of important diseases has been given to aid the budding doctors in proper patient evaluation.
• Every disease condition is well explained with its Epidemiology, Etiology, Clinical Features, Differential Diagnosis, Laboratory Abnormalities, Investigations, Management and Treatment.
• Brief description of all dermatologic lesions has been highlighted in colored boxes for easy learning.
• The book consists of 25 chapters distributed into 5 sections, viz. Dermatology, Venereology, Leprosy, Cosmetology and Acquired Immunodeficiency Syndrome.
• Chapters on Cosmetic Dermatology and Dermatosurgery, Lasers in Dermatology, psychosexual Dysfunctions, HIV Infection and AIDS have been included keeping in mind the recent developments.
• Essential preparation guide for those appearing in various PG entrance examinations.
It gives me a sense of privilege and immense pleasure in writing this foreword for the book by Dr Ramesh Bansal. He has always been a sincere, bright and hard working student throughout his career, and this book speaks about his talent.

This book is aptly entitled *Essentials in Dermatology, Venereology and Leprology* as it provides all essentials for learning dermatology, and venereology to the undergraduates and is also sufficient for Postgraduate Medical Entrance Examination. In fact, this book is helpful for all those who have interest in dermatology irrespective of students, consultants and general practitioners.

Digital photographs of all commonly seen dermatological clinical conditions have been nicely given in various chapters. Footnotes which elaborate certain words of text is a special feature of the this book. Separate chapters have been included on glucocorticoids, cosmetology, lasers in dermatology and HIV infection.

I would recommend this book for all undergraduate students and for those who are appearing in various PG entrance examinations.

I congratulate Dr Ramesh Bansal for this well written book and wish him all good luck in this venture.

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Preface

Seeing the demand of today, my academic interest generated the idea of writing a book on dermatology, which can provide essential knowledge to undergraduate and postgraduate students. Every effort has been made to provide latest information about all aspects. A simplified concept has been given with numerous clinical photographs, illustrations and tables. In fact, this book is meant for everyone who has interest in dermatology. Special attention is given for undergraduates, who have to appear for their postgraduate entrance examinations. Seeing the recent developments, chapters on cosmetic dermatology and dermotosurgery, lasers in dermatology, psychosexual dysfunctions, HIV infection and AIDS have also been included.

It was not possible to give multiple choice questions (MCQs) at the end of the chapters because a huge number of MCQs can be made from rich text. One or two MCQs can be made from each line of the text. It requires to prepare a separate book. Attempt has been made to include answers to all possible questions in the text.

Continuous concern, cooperation and encouragement by all my family members helped me in completing this project.

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    - Endogenous Source
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      - Tinea Manuum
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      - Tinea Incognito (Steroid Modified Tinea)
    - Candidiasis
    - Pityriasis Versicolor
    - Deep Fungal Infections
      - Sporotrichosis
      - Mycetoma
    - Chromoblastomycosis (Chromomycosis)
Bacterial infections are very commonly seen in clinical practice. These can present as:
• Primary bacterial skin infection.
• Secondary bacterial infection of a primary skin disease, e.g., infected scabies.
• Reactive skin lesions to bacterial infection elsewhere, e.g., id eruption, erythema nodosum because of *Streptococcal pharyngitis*, etc.

**NORMAL FLORA OF SKIN**

**Resident Flora**

It comprises many types of bacteria which inhabit normal skin permanently and have following features:
• Live harmlessly as commensals on cutaneous surface or in hair follicles.
• They can multiply.
• Usually nonpathogenic
• Occasionally, their outgrowth may cause mild disease of skin or its appendages.

**Location**

- Moist skin sites with partial occlusion (axillae, perineum, and toe webs) harbor more microorganisms (gram-negative bacilli) than less occluded dry areas (legs, arms, and trunk).
- Most numerous in areas rich in hair follicles and sebaceous glands, e.g., scalp and face.
- Most microorganisms live in superficial layers of stratum corneum and upper parts of hair follicles.
- Some bacteria reside in deeper parts of hair follicles and are beyond the reach of ordinary disinfection procedures.

**Type**

The normal flora of skin consists of following organisms (Table 4.1).

**Function**

- Defense against bacterial infection through bacterial interference and by competing with pathogenic organisms for nutrients.
- Inhibits the growth of other organisms—*Propionibacterium acne* and gram-positive cocci hydrolyze lipids of sebum and produce free fatty acids. Acidic medium inhibits growth.

**Pathogenesis**

Involves four major factors:

<table>
<thead>
<tr>
<th>Table 4.1: Resident flora at various sites</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stratum corneum</strong></td>
</tr>
<tr>
<td><strong>Gram-positive cocci</strong></td>
</tr>
<tr>
<td>- <em>Staphylococcus epidermidis</em> (90% of aerobic flora)</td>
</tr>
<tr>
<td>- <em>Micrococcus luteus</em></td>
</tr>
<tr>
<td>- Aerobic diphtheroids** or coryneforms (causes erythrasma, pitted keratolysis and trichomycosis axillaris)</td>
</tr>
<tr>
<td>- <em>Staphylococcus aureus</em>:</td>
</tr>
<tr>
<td>- Should not be considered as resident of healthy skin</td>
</tr>
<tr>
<td>- Found in nose (especially in new born), vulval skin and skin of atopic dermatitis patient</td>
</tr>
<tr>
<td>- Streptococci are notably absent</td>
</tr>
<tr>
<td><strong>Gram negative bacilli</strong></td>
</tr>
<tr>
<td>- Acinetobacter is the only significant gram-negative flora. They make-up a small proportion of skin flora and are seen in moist intertriginous areas, such as toe webs and axillae, and not on dry skin. Desiccation prevents their multiplication on intact skin</td>
</tr>
</tbody>
</table>

| **Hair follicles**                        |
| - *Propionibacterium acne* (*P. acne* is main anaerobic diphtheroid resident) is regularly found in deeper part of adult skin follicles |
| - Aerobic cocci                           |
| - *Malassezia* species of yeast           |
| - *Demodex folliculorum*:                 |
| - It is a normal inhabitant mite which lives around hair follicles or in the secretory ducts of sebaceous glands |
| - The preferred sites are facial skin, forehead, cheeks, eyelashes, and external ear channels |

| **Sweat glands and ducts**: No bacterial inhabitants |

*Temporary flora*: It refers to flora which may sometimes temporarily colonize and establish for prolonged periods. It gets contaminated from exogenous source and is often pathogenic.

**Diphtheroid**: The term diphtheroid denotes a wide range of bacteria belonging to genus Corynebacterium
Section 1: Dermatology

Table 4.2: Various disorders caused bacterial infections

<table>
<thead>
<tr>
<th>Nonfolicular infections</th>
<th>Follicular infections</th>
<th>Diseases caused by resident flora</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial infections</td>
<td>Superficial folliculitis</td>
<td>Erythrasma</td>
</tr>
<tr>
<td>Nonbullous impetigo</td>
<td>Noninfecious folliculitis</td>
<td>Pitted keratolysis</td>
</tr>
<tr>
<td>Bullous impetigo</td>
<td>Deep folliculitis</td>
<td>Trichomycosis axillaris</td>
</tr>
<tr>
<td>Ecchyma</td>
<td>Sycosis barbae</td>
<td></td>
</tr>
<tr>
<td>Toxin-mediated disorders</td>
<td>Furuncle</td>
<td></td>
</tr>
<tr>
<td>Staphylococcal scalded skin syndrome</td>
<td>Carbuncle</td>
<td></td>
</tr>
<tr>
<td>Toxic shock syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scarlet fever</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep infections</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erysipelas and cellulitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anthrax</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Portal of entry:
   - The normal intact and functioning skin resists bacterial invasion.
   - The dry, continuously desquamating epidermis constantly removes the contaminating organisms.
   - Maceration and occlusion increase bacterial flora.
   - The premature infants do not have an effective epidermal barrier.

2. Host defense and inflammatory response.
3. Interactions between normal flora and contaminating organisms.
4. The virulence of the organism

   The bacterial infections of skin are conveniently considered in Table 4.2.

Superficial Infections

Bacterial infections in the epidermis just below the stratum corneum or in hair follicles are called superficial infections or pyodermas.

Impetigo

It refers to contagious superficial bacterial infection of skin. Two clinical forms have been recognized.

Nonbullous Impetigo (Impetigo Contagiosa)

Epidemiology
- **Prevalence**: Common
- **Age**: Preschool and young school age children
- **Sex**: No sex predilection

Etiology
- *Staphylococcus aureus* is the most common cause in developed countries.
- *Streptococcus pyogenes* remain a common cause in developing countries.
- Or both

Clinical features

Morphology
- Initially, there is a thin-walled vesicle, which ruptures rapidly and is seldom seen as such. Surrounding erythema may be present.
- The exudates get dried to form yellowish, thick and dirty brown (honey colored) crusts (Fig. 4.1).
- Multiple lesions are usually present, which may coalesce. Eventually crusts dry up and separate without scarring.

Sites
- The common sites of involvement are skin around the nose, mouth, and limbs.
- Extremities and scalp are less frequent sites.
- Regional lymph node enlargement and fever is present in severe cases.

Fig. 4.1: Impetigo contagiosa showing thick dirty brown crusts
Complications
- Serious complications are unusual in the absence of malnutrition or any systemic diseases.
- Eczematization is frequent.
- Nephritis due to nephritogenic strains of streptococci types—2, 49, 52, 55, 57 and 60. The latent period required for the development of nephritis following streptococcal pyoderma and throat infections is in the range of 18–21 days and 10 days, respectively.
- Scarlet fever, urticaria and erythema multiforme are few other complications,* which may be seen after streptococcal impetigo.

Investigations
- Gram staining of exudates can reveal streptococci and staphylococci as gram-positive cocci in chains and clusters, respectively.
- **Culture:** Culture and sensitivity to etiological agent can also be performed.

Clinical diagnosis
Clinical diagnosis is made on the basis of following characteristics:
- Honey colored multiple crusted lesions with surrounding erythema
- Involvement of face in a child

Differential diagnosis
Sometimes it needs to be differentiated from bullous impetigo and herpes labialis (Table 4.3 and 4.4)

**Bullous Impetigo**

**Epidemiology**
- **Prevalence:** Sporadic
- **Age:** Newborn and infants
- **Sex:** No predilection

**Etiology**
It is caused by *S. aureus* group II phage, especially strains** 77 and 55 by producing extracellular exfoliative exotoxins types A and B.

**Pathogenesis**
- It forms large blister (1–2 cm) by splitting the epidermis just below the stratum granulosum.
- This splitting is due to exfoliative toxin A that acts on desmoglein-1 in the similar way as caused by autoantibodies in pemphigus foliaceus.

**Table 4.3: Difference between bullous impetigo and impetigo contagiosa**

<table>
<thead>
<tr>
<th>Bullous impetigo</th>
<th>Impetigo contagiosa</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Caused by:</strong> Exotoxins produced by <em>Staphylococcus aureus</em> group II phage</td>
<td><em>S. aureus</em> or <em>Staphylococcus pyogenes</em> or both</td>
</tr>
<tr>
<td><strong>Prevalence:</strong> Sporadic</td>
<td>Very common</td>
</tr>
<tr>
<td><strong>Age:</strong> Common in new born and infants</td>
<td>Preschool and young school children</td>
</tr>
<tr>
<td><strong>Epidermal split:</strong> Just below the stratum granulosum</td>
<td>Above, within, or below stratum granulosum</td>
</tr>
<tr>
<td><strong>Morphology:</strong> Bullae are thick wall, persistent and usually of small size</td>
<td>Thin-walled, transient and vesicles</td>
</tr>
<tr>
<td><strong>Surrounding erythema:</strong> Not present</td>
<td>May be present</td>
</tr>
<tr>
<td><strong>Crusts:</strong> Thin and dark yellow (varnish like)</td>
<td>Thick and yellow or dirty brown (honey colored)</td>
</tr>
<tr>
<td><strong>Site:</strong> Face and other body parts</td>
<td>Mainly seen on face</td>
</tr>
<tr>
<td><strong>Lymphadenopathy:</strong> Not a feature</td>
<td>Present in severe cases</td>
</tr>
</tbody>
</table>

**Table 4.4: Difference between herpes labialis and impetigo contagiosa**

<table>
<thead>
<tr>
<th>Herpes labialis</th>
<th>Impetigo contagiosa</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Recurrence:</strong> It is an important feature</td>
<td>Don't recur</td>
</tr>
<tr>
<td><strong>Morphology:</strong> It may present as small, grouped vesicles/crusted lesions/polycyclic erosions</td>
<td>Single or multiple (common) crusted lesions which may or may not coalesce</td>
</tr>
<tr>
<td><strong>Site:</strong> Usually limited around mouth</td>
<td>Not limited around mouth</td>
</tr>
</tbody>
</table>

**Clinical features**

**Morphology**
- Thick walled bullae which rupture less rapidly and may persist for 2–3 days (Fig. 4.2).
- Bullae arise on normal skin without an erythematous halo and Nikolsky’s sign is negative.
- Initially contents are clear, later it becomes turbid.
- After rupture, thin dark yellow crusts (varnish like) are formed.
- Lesions are seldom multiple. Extensive bullous impetigo was previously called as *pemphigus neonatorum*.
- Lymphadenopathy is not a feature.

**Sites**
Besides face, involvement of other sites is also common.

*Rheumatic fever* is not seen after streptococcal impetigo.

**Exotoxins produced by *S. aureus* cause three types of cutaneous eruptions, i.e., bullous impetigo, staphylococcal scalded skin syndrome (SSSS) and staphylococcal scarlatiniform eruption.
Chromogenic Ecthyma: Thicker adherent crusts and deeper ulcer.

Ecthyma
Ecthyma is a deeper cutaneous pyogenic infection.

Etiology
- **Etiological bacteria:** Similar to that of impetigo.
- **Predisposing factors:** Poor hygiene, neglect and malnutrition.
- Untreated impetigo can also extend more deeply resulting into shallow and crusted ulcer.

Clinical Features

Morphology
- Characterized by thick adherent crusts (Fig. 4.3) with underlying shallow and punched out ulcers.
- The margins of ulcer are indurated and raised. Base is also indurated. A red, edematous surrounding areola is often seen. The crust can be removed with difficulty revealing an irregular punched out ulcer.
- Healing occurs with scarring in few weeks.

Sites
- Buttocks, thighs and legs

Treatment
- **Local treatment:**
  - It is sufficient in mild to moderate patients.
  - Infected crust should be removed with potassium permanganate (KMnO₄) compresses.
  - Mupirocin ointment

- **Systemic treatment:**
  - It should be given when involvement is extensive or lymphadenopathy or constitutional symptoms are there.
  - For streptococcal impetigo, penicillin is the drug of choice. Benzathine penicillin (0.3–0.6 million for children and 1.2 million for adults) can be given as a single injection.
  - For staphylococcal impetigo in adults responds nicely to dicloxacillin.
  - Combination of amoxicillin and clavulanic acid (25 mg/kg/day), erythromycin and cephalaxin (50 mg/kg/day) are good alternative drugs.

Staphylococcal Scalded Skin Syndrome

Epidemiology
- **Age:** Infants or children, rare in adults
**Predisposing factors:** Adults having renal insufficiency, immunosuppression, malignancy and alcohol abuse.

**Etiopathogenesis**
- Initially, patient usually has localized staphylococcal infection anywhere in the body, e.g., conjunctiva, ear, nasopharynx, umbilicus or occasionally skin.
- Exfoliative toxin is produced at this distant site which disseminates hematogenously.
- Exfoliative toxin binds to desmoglein-1 and results in loss of cell-to-cell adhesion and epidermolysis below the stratum granulosum.

**Clinical Features**
- A few days later of pyogenic focus, there is a development of fever, irritability, and cutaneous tenderness.
- It is followed by widespread erythema, which progresses into blister formation (Fig. 4.4A). Nikolsky’s sign is positive (Fig. 4.4B). The blister contents are usually sterile, which is consistent with toxin-mediated pathogenesis.
- The epidermal surface is peeled off in widespread manner leaving underlying moist red base. Periorificial and flexural skin show characteristic wrinkling of skin.
- The patients do not appear toxic unless complicated by septicemia or pneumonia.
- **Course:** It resolves spontaneously or faster with antibiotics within 7–10 days.

**Investigation**
Gram staining and culture of blister fluid does not reveal any microorganism.

**Clinical Diagnosis**
Important features for diagnosis:
- Patient is usually infant or a child
- Thin-walled vesicles or bullae filled with sterile clear fluid
- Superficial peeling of skin
- Clue of focus of staphylococcal infection
- Absence of mucosal involvement

**Differential Diagnosis**
It needs to be differentiated from toxic epidermal necrolysis (TEN) (Table 4.5).

**Treatment**
- Oral or intravenous suitable antibiotics against *S. aureus* should be administered early.
- The nonadherent petroleum-impregnated dressings on widespread raw areas are helpful.
- Fluid and electrolyte imbalance should also be taken care of.

**Prognosis**
It is good in children with very low mortality (2%). Mortality is higher in adults (10%).

**Toxic Shock Syndrome**

**Etiology**
- Due to toxic shock syndrome (TSS) toxin-1 (TSST-1) produced by *S. aureus*.
- *Streptococcus pyogenes* which shows positive blood cultures in 50% of patients in contrast to only 10% in staphylococcal TSS.

**Clinical Features**
- It begins with acute onset of fever (> 102°F).
- **Rash:** Commonly a diffuse macular erythema.
Fever
2. Rash
3. Desquamation
4. Hypotension

• Three or more of following multisystems must show involvement:
  - Gastrointestinal tract (GIT)
  - Muscles—myalgia or raised creatine kinase levels
  - Mucosal hyperemia
  - Kidney—raised creatinine levels
  - Liver—raised bilirubin or enzyme levels
  - Blood—platelets less than 100,000/mm³
  - Central nervous system (CNS) involvement

Differential Diagnosis
It should be differentiated from Kawasaki disease, which is characterized by prolonged fever, involvement of heart, enlargement of lymph nodes and absence of shock.

Treatment
• Intensive general supportive measures.
• Systemic antibiotics: A combination of vancomycin and clindamycin is recommended for serious staphylococcal infections.
• Intravenous immunoglobulin: Through neutralizing antibodies against toxins.

Scarlet Fever
It is another clinical syndrome caused by toxin producing gram-positive cocci. It is characterized by exudative pharyngitis, fever, and rash.

Epidemiology
• Age: Children
• Season: Winter season

Etiology
• Microbes: Streptococcus species
• Portal of entry: Upper respiratory tract
• Incubation period: Within 5 days

Clinical Features
Onset
• Abrupt onset of fever, pharyngitis, headache and pain in abdomen.
• Skin rash:
  - Appears 1–2 days after onset of fever
  - Finely punctate erythema (scarlatiniform) rash having coarse texture like a fine sand paper
Cutaneous Infections  CHAPTER 4

- Spares palms and soles
- Transverse red lines may be seen in skin folds due to capillary fragility (Pastia’s sign).

**Mucosal lesions:**
- The oral mucosa is bright red
- Tonsils become red, edematous and covered with exudate
- The soft palate and uvula show punctuate macules (Forschheimer’s spots).
- Initially, tongue is swollen and coated white (white strawberry tongue). Later on, desquamation results into red tongue (red strawberry tongue).
- Anterior cervical lymph nodes become enlarged and tender.
- Similar picture may be produced by *Staphylococcus* also. Pharyngitis is not a feature of staphylococcal-mediated disease.

**Course**
- Fever usually subsides in 7–10 days
- Usually runs a benign course.

**Complication**
Glomerulonephritis can occur in 10–15% of patients.

**Treatment**
As soon as the diagnosis is suspected, patient should be treated by penicillin or erythromycin in full doses for 10 days.

**Kawasaki Syndrome (Mucocutaneous Lymph Node Syndrome)**

**Epidemiology**
- **Age:** Most cases (younger than 2 years) and 80% cases (younger than 5 years)
- **Prevalence:** Rare

**Etiology**
- **Agent:** It is considered as immune activation in response to an infectious agent. Previously agent was thought to be human coronavirus. But, recently evidences are in favor of bacterial toxin that acts as superantigen.
- **Transmission:** Unlikely to be transmitted from person to person.

**Clinical Features**
- It is a multisystem vasculitis.
- Prolonged (1–2 weeks) high grade fever (104°F)

- **Exanthem:**
  - Develops in initial days of fever
  - Favors trunk and proximal part of extremities
  - Variable, can be macules, papules, morbilliform or urticarial. Perineal blanching erythema is characteristic.
  - Within 2 days, erythematous areas desquamate.
- **Bilateral**, **painless**, **nonexudative**, and **bulbar conjunctival redness**
- **Mucosal changes:** Red, dry, cracked lips, strawberry tongue, and oral mucosa shows diffused erythema
- Edema and redness of hands and feet
- Characteristic desquamation of fingers and toes
- **Lymphadenopathy:** Single, nonsuppurative lymph node in anterior cervical triangle is common.

**Complications**
Cardiac complications are common. It is a leading cause of acquired heart disease in children.

**Laboratory Findings**
- Leukocytosis
- Thrombocytosis in third week is a characteristic feature.
- Elevation of erythrocyte sedimentation rate (ESR), liver enzymes, bilirubin, and alkaline phosphatase.
- Sterile pyuria
- Electrocardiography (ECG) abnormalities

**Criteria for Diagnosis**
Unexplained fever of 5 or more days with four of the five following criteria:
1. Bilateral, painless, nonexudative and bulbar conjunctival redness
2. **One of the following oropharyngeal features:** Fissured lips, strawberry tongue or injected pharynx.
3. **One of the changes on limbs:** Palmar or plantar erythema, edema of hands or feet, periungual desquamation.
4. Polymorphous exanthem
5. Nonsuppurative cervical lymphadenopathy

**Treatment**
- **Aim:** Reduction of myocarditis and prevention of coronary thrombosis.
- **Intravenous immunoglobulin (IVIG) and acetyl salicylic acid (ASA):** 1 gm/kg IVIG in combination with ASA (80–100 mg/kg/day in four divided doses) is recommended for acute stage of illness.
DEEP INFECTIONS

These are acute, painful, edematous, pyogenic inflammation of deeper soft tissues, i.e., dermis, subcutaneous tissue or muscular tissues.

Also known as soft tissue infections and are usually associated with local pain and fever.

Predisposing factors: Diminished immune status, underlying malignancy, diabetes, renal failure, neutropenia, human immunodeficiency virus (HIV) infection, glucocorticoid therapy, etc.

Erysipelas and Cellulitis

Erysipelas is a superficial bacterial infection with marked lymphatic vessel involvement in dermis and upper subcutaneous tissue.

The term “cellulitis” is mainly applied to acute, subacute or chronic inflammation of subcutaneous tissues, but may extend superficially to involve dermis also.

Etiology

- Usually caused by group A β-hemolytic streptococci and rarely by S. aureus.
- Bacteria enter through any break in the skin (any sharp injury or around surgical wound).
- Predisposing factors: Venous stasis, intertrigo and obesity are important factors for recurrent infection.

Clinical Features

Symptoms

Fever, severe pain, swelling and redness of the affected part of the body.

Morphology

Affected part is red, warm, indurated (firm to hard). Cellulitis is differentiated from erysipelas by indistinct margins between affected and unaffected skin (Fig. 4.5). Regional lymphadenopathy is frequent. Erysipelas is seen as superficial, painful edematous red plaque with sharp well-defined raised edge to normal tissue (Fig. 4.6).

Site

Lower limbs, less frequently face and upper limbs

Course

- Infection may localize in the dermis or subcutaneous tissue with formation of abscess.
- The overlying epidermis may undergo bulla formation or necrosis to form extensive areas of epidermal sloughing.
- In more severe cases, it can lead to fasciitis or myositis.

Treatment

Supportive measures

- Bed rest
- Elevation of affected part
- Moist heat: It will help in localization of abscess in patient with cellulitis.
- Nonsteroidal anti-inflammatory drugs (NSAIDs)
Specific measures

- Mild erysipelas can be treated as an outpatient with intramuscular (IM) procaine penicillin in the dose of 600,000 units BD.
- Cellulitis should be treated with dicloxacillin (500 mg QID) or oral cephalosporins. Macrolides and clindamycin are alternative drugs for penicillin allergic patients.
- Extensive infections especially with underlying medical problems:
  - Need hospitalization
  - Treated with 1–2 million units of aqueous penicillin every 4–6 hours intravenous (IV) for 10 days.
- Necrosis and slough formation: Require early and complete necrotic tissue debridement.

Anthrax

Anthrax is a toxigenic zoonotic* infectious disease from herbivorous animals.

The infection is most often seen after occupational exposure, e.g., caretaker of livestock, handling of infected soil by farmers and exposure of workers during handling of hides, wool, hair, and meat.

Clinical Forms

There are three clinical forms:

1. Cutaneous anthrax
2. Inhalational anthrax (Woolsorter’s disease): It is due to inhalation of spores. Mild primary illness of 2–3 days is followed by respiratory distress.
3. Gastrointestinal anthrax: It is due to intake of infected meat or milk.

Cutaneous Anthrax (Most Common Type in Man)

Etiology

- It is caused by Bacillus anthracis, a large gram-positive, aerobic and spore forming rod.
- Acquired through percutaneous inoculation of anthrax spores through minor cuts in skin on exposed parts of hands, legs, and face.

Clinical features

- After 1–2 days of inoculation, an irritable, but painless papule develops which progresses through vesicular, pustular and escharotic phases surrounded by edema. The edema is brawny, nonpitting and is named as malignant edema. The individual lesions appear like a pustule which do not suppurate and is named as malignant pustule.
- In suitable environment of human skin, spores revert to rods which produce toxins. It may be accompanied by malaise, fever, and lymphadenopathy.

Course

- In later stages, lesions shows hemorrhagic central necrosis giving umbilicated appearance and become painless. The central black eschar** drops and falls off in 1–2 weeks.
- Uncommonly, in severe untreated cases high fever, toxemia, and prostration may lead to delirium, collapse, and death.
- Prognosis: Severe edema and toxemia are associated with poor prognosis.

Diagnosis

Gram staining of fluid from cutaneous lesion may show the bacilli.

Treatment

- Cutaneous anthrax: Oral ciprofloxacin, doxycycline, and amoxicillin for 7–10 days
- Systemic anthrax: IV penicillin G
- Severely toxic patient: Add IV fluids and corticosteroids

FOLLICULAR INFECTIONS

Follicular infection or folliculitis is a pyoderma of the hair follicles and is classified into superficial and deep according to the depth of hair invasion.

Superficial Folliculitis

- Very common condition
- Involvement is confined to the osteum (Fig. 4.7A) or may extend only slightly below it.
- Heals without scarring

Causes of superficial folliculitis can be:

- Infection:
  - Bockhart’s impetigo: It is a superficial folliculitis by S. aureus.
  - Chronic superficial folliculitis: It is a profuse eruption of follicular pustules on lower extremities

**Zoonosis: Disease of animals transmissible to man.
**Eschar (Greek anthrakos, coal): It is a slough produced by a thermal burn, corrosive application or by gangrene.
Deep Folliculitis

It includes three clinical entities:

* **Sycosis* Barbae**
  - It is a subacute or chronic bacterial infection of whole depth of hair follicles commonly in the beard region or upper lip (Fig. 4.9).

**Pseudofolliculitis Barbae**
- Etiology: It results due to mechanical reason. It is seen in males who have curly hairs. Penetration of sharp tips of shaved hairs to the neighboring skin is responsible for the disorder.

---

* Greek sukosis means fig
• It presents as red edematous papule or pustule with a hair in the center. Many neighboring such papules may coalesce to form raised plaques studded with pustules giving an appearance of a ripen fig, which has given the name sycosis.
• Often hairs are retained with no evident scarring.
• In most cases, it begins between third and fourth decade. Sometimes, follicles are destroyed with evident scarring. It is termed as lupoid sycosis.
• It should be differentiated from dermatophytic folliculitis in which there are nodules rather than pustules. Hairs are loose and easily pluckable.

**Furuncle (Boil)**

Furuncle comprises deep folliculitis and parafolliculitis*.

**Etiology**

- It is usually caused by *S. aureus* and often evolves into an abscess.
- **Predisposing factors:** Mechanical damage, perspiration, diabetes, systemic steroids, cytotoxic agents, blood dyscrasias and obesity.

**Clinical features**

- It presents as acute, necrotic deep-seated inflammatory nodule (Fig. 4.10A) around a hair follicle.
- Initially, it presents as hard, tender, follicular and red inflammatory nodule in the hair bearing area of skin.
- Face, neck, buttocks, anogenital region, arms, and thighs are the most common sites.
- Later, it enlarges and becomes fluctuant due to abscess formation (Fig. 4.10B). Rupture occurs with discharge of pus and necrotic core. Pain, redness and edema subside in a week time, leaving ultimately a permanent scar.

**Treatment**

- It is treated by systemic flucloxacillin or other penicillinase resistant antibiotic.
- Application of moist heat is helpful.
- When becomes soft, express pus manually or do incision and drainage.

**Carbuncle**

A carbuncle is a large, more serious deep staphylococcal infection involving a group of contiguous hair follicles characterized by intense inflammation of surrounding and underlying connective tissues.

- **Predisposing factors:** Middle or old age, underlying diabetes, steroid therapy, malnutrition, severe dermatitis, and cardiac failure.
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• **Incision and drainage:** Of large fluctuant and painful lesions.

• **Management of recurrent furunculosis:**
  - In addition to systemic causes, the local predisposing factors like exposure to chemical, poor hygiene, tight belts, clothing and obesity should be taken care of.
  - Patient should use loose clothing.
  - In addition to use of soap and water for care of hands and body, use of 4% chlorhexidine solution may help in decreasing cutaneous colonization by *Staphylococcus*.
  - Efforts can also be made for elimination of *S. aureus* from nose. Intranasal application of 2% mupirocin ointment for 5 days in a month for 1 year is found to be effective.
  - The use of 600 mg of rifampicin per day for 10 days is also helpful.

**NONPYOGENIC CUTANEOUS INFECTIONS**

- **Under normal circumstances, normal flora of skin does not cause any disease.**
- **In the presence of certain favorable predisposing factors like moisture, they overgrow and results in superficial bacterial infections.**
- **These disorders are not characterized by pus formation.**
- **They can also be called as nonpyogenic cutaneous infections.**

**Erythrasma**

*Etiology*

- *Corynebacterium minutissimum* (short, gram-positive aerobic rod)

• **Predisposing factors:** Hot and humid climate

**Clinical Features**

- Asymptomatic, irregularly shaped, sharply marginated red or brown patches in axillae, groins and submammary and intergluteal regions.
- The patches show fine scaling and wrinkling, and unlike tinea infections there is no central clearing, but have relatively uniform appearance.
- Involvement of web spaces of feet is often asymptomatic where it presents as scaling, fissuring and maceration especially between fourth and fifth toes.

**Investigations**

- **Wood’s lamp:** Demonstrates coral-red fluorescence due to coproporphyrin III.
- **Scrapings:** From lesions may reveal Gram-positive rod-like bacteria on staining. Potassium hydroxide (KOH) smear may show fine filaments.

**Treatment**

- **For localized lesions:** Topical application of azole antifungal agents (miconazole or clotrimazole), 2% clindamycin solution and benzoyl peroxide is quite effective for localized lesions.
- **For widespread lesions:** 1 gm clarithromycin is most effective. Alternatively, topical fucidin and oral tetracycline can be used.
- **For relapsing lesions:** Long-term use of antiseptic soaps and drying agents (powders).

**Pitted Keratolysis**

*Etiology*

- *Corynebacterium*

• **Predisposing factors:** Plantar hyperhidrosis and wearing of shoes for long time.

**Pathogenesis**

Digestion of keratin by keratinophilic corynebacteria.

**Clinical Features**

- Circular shaped erosions on the soles (Fig. 4.12).
- The lesions have a punched out appearance, and may coalesce to form large irregular erosions on pressure bearing areas.
- Plantar hyperhidrosis and malodorous feet
Treatment

- Hyperhidrosis should be controlled with formalin soaks and feet should be kept as dry as possible.
- Azoles, sodium fusidate, benzoyl peroxide (5%) and erythromycin may be applied topically.

**Trichomycosis Axillaris**

- It is a double misnomer.
- Firstly, it is not caused by fungus. It is caused by commensal bacteria coryneform.
- Secondly, it involves both axillary and pubic hair and not the axillary hair alone.

**Clinical Features**

- Usually asymptomatic.
- Patient may complain of colored sweating, yellow brown staining of undergarments and malodor.
- On examination, axillary hair are beaded due to yellow, black or red thickenings on hair shaft composed of colonies of gram-positive bacilli. The underlying skin is essentially normal.

**Treatment**

- Patient is usually concerned about lesions and malodor.
- Effective measures are:
  - Benzoyl peroxide
  - Regular shaving of involved hair

**TUBERCULAR INFECTIONS**

Tuberculosis has the highest morbidity and mortality rates among all infectious diseases and is a commonly seen mycobacterial infection* in developing countries like India.

- It can virtually affect any organ of the body, e.g., lung, bone, skin, meninges, lymph node, visceral organs, etc.
- Incidence of tuberculosis in acquired immunodeficiency syndrome (AIDS) patients is about 500 times more than that of general population.
- Sharp increase in pulmonary infection has not reflected as an equal increase in mycobacterial skin disease.
- *Mycobacterium tuberculosis* and *M. leprae* are most important obligate** human pathogens. They are neither very virulent nor very infectious.

**ETIOLOGY**

Tuberculosis of skin is caused by:

- *M. tuberculosis*
- *M. bovis*
- Under certain conditions by Bacille Calmette-Guérin (BCG, an attenuated strain of *M. bovis*)

Mycobacteria are slender, nonmotile, noncapsulated, nonsporing and aerobic rods (0.5 × 3 µm) having waxy coating which makes them resistant to most stains. They do not stain easily, but once stained, resist decolorization with dilute mineral acids and hence are known as acid fast bacilli (AFB). ***

**PATHOGENESIS**

- Inhalation of airborne droplets containing *M. tuberculosis* complex results in pulmonary infection.
- In persons having intact cell mediated immunity (CMI), spread of infection is limited by formation of granuloma (Ghon focus) by activated T-cells and macrophages at the site of pulmonary infection. The latter, together with enlarged hilar lymph nodes is known as primary complex. On the other hand, granuloma due to primary cutaneous infection results in tuberculous chancre and if the involved lymph nodes break down, it results into scrofuloderma.

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*Robert Koch*: He discovered tubercle bacillus in 1882.

**Obligate**: An organism which cannot survive outside the host.

***Acid fastness**: It is mainly due to organism’s high content of mycolic acids and long chain fatty acids.
• Following *M. tuberculosis* infection, only 5–10% of individuals develop progressive disease. It can result into a wide range of cutaneous manifestations depending on the route of infection and immunological status of the host.

**CLASSIFICATION**

Similar routes of infection can produce different clinical manifestations due to different host’s CMI, while different routes of infection (Fig. 4.13) can produce similar clinical manifestations. Consequently, it makes the task of classifying tuberculous skin diseases difficult.

Without considering host’s CMI, a simple classification on the basis of routes of infection can be made as follows:

- **Exogenous source:**
  - Primary inoculation tuberculosis
  - Tuberculosis verrucosa cutis
  - Lupus vulgaris (some patients)

- **Endogenous source:**
  - Scrofuloderma (contiguous spread)
  - Orificial tuberculosis (autoinoculation)
  - Lupus vulgaris (contiguous or hematogenous source)
  - Miliary skin tuberculosis
  - Metastatic tubercular abscess (tuberculous gumma)

**EXOGENOUS SOURCE**

**Primary Inoculation Tuberculosis** *(Tuberculous chancre)*

- **Prevalence:** Very uncommon form
- **Age:** Common in children

**Immunity**

- Patient has no previous tuberculous infection
- Does not harbor natural or acquired immunity to this organism.

**Pathogenesis**

- Bacillus enters the skin through a minor cut or abrasion.
- Early changes consist of acute neutrophilic infiltration, necrosis and presence of numerous bacilli (multibacillary).
- But as the immunity develops in 3–6 weeks, appearance of caseating granuloma coincides with disappearance of bacilli.

---

**Fig. 4.13:** Pathogenesis of paucibacillary (dark green color) and multibacillary (light green color) cutaneous tuberculosis
**Clinical Features**
- Painless brownish papule or nodule or a typical ragged tuberculous ulcer (tuberculous chancre) having reddish blue undermined edge and hemorrhagic granular base.
- Face and limbs are common sites of involvement.
- It may be accompanied by regional lymphadenopathy. Together with tuberculous chancre, it constitutes the **tuberculous primary complex** in skin.
- **Course:** Ulcer can heal spontaneously with scar formation.

**Diagnosis**
- Any nonhealing ulcer with unilateral regional lymphadenopathy in a child should arouse suspicion.
- Diagnosis can be confirmed by microscopy and culture.

**Tuberculosis Verrucosa Cutis**
It is also known as **warty tuberculosis**. It is a paucibacillary type of skin tuberculosis. It is common in men.

**Etiology**
It is caused by exogenous reinfection in a previously infected patient.

**Immunity**
High degree of immunity and tuberculin sensitivity is highly positive.

**Pathogenesis**
- Bacilli enter the skin through minor wounds or abrasions.
- Physicians, pathologists, medical students or laboratory attendants are more liable.
- Autoinoculation with sputum in an active tuberculosis patient is another mode of infection.

**Clinical Features**
- It may present as a small, purplish red or brown, hyperkeratotic, asymptomatic, indurated, papule or plaque surrounded by erythema. Margins show finger like projections or serpiginous outline (Fig. 14A). Sometimes, the lesion may be psoriasiform or keloidal.
- Lesion may show white atrophic scar, clefts, and fissures with pus discharge.

**Differential Diagnosis**
- Lichen planus hypertrophicus which consist of multiple and itchy lesions.
- Lupus vulgaris lesions are usually not hyperkeratotic and apple jelly nodules can be demonstrated in them.
- Verruca vulgaris lesions are usually multiple with no induration, scarring or discharge.
- Blastomycosis and chromomycosis can be excluded histopathologically.

**Histology**
- Epidermis shows striking pseudoepitheliomatous hyperplasia.
- Dermis shows mixed infiltrate with sparse tuberculosis foci.

*Anatomist’s wart, verruca necrogenica, prosector’s wart and lupus verrucosus are other names of this condition.*
SECTION 1  Dermatology

ENDOGENOUS SOURCE

Scrofuloderma (Tuberculosis Colliquativa Cutis)

It results from contiguous spread of infection from underlying tuberculous focus.

Sites

- **Lymph nodes**: Cervical (most common), axillary and inguinal lymph nodes.
- **Infected bone and joint (Fig. 4.15 A and B)**: Tubercular osteomyelitis.
- **Infected gland**: May at times be responsible.

Clinical Features

- Typically, bluish red nodule overlying a suspected tuberculous focus breaks down to form an ulcer.
- Ulcer has watery discharge, bluish margins and granulating base.
- After healing, peculiar puckered scarring (Figs 4.16A and B) is left at the site of infection.

Differential Diagnosis

- **Furuncle**: It is an acute painful short lasting swelling.
- **Actinomycosis**: It is characterized by dull red nodules with multiple discharging sinuses. Discharge may contain characteristic sulfur granules.

Figs 4.15A and B: Scrofuloderma. A. Due to involvement of cervical lymph node; B. Involvement of shoulder joint

Figs 4.16A and B: Untreated scrofuloderma. A. Involvement of multiple cervical lymph nodes; B. Puckered scarring
• **Hidradenitis suppurativa**: It is a chronic suppurrative disorder of apocrine gland bearing areas (axillae, perianal area and buttocks). It manifests as bilateral symmetrical, red, tender nodules which later on rupture to form sinus tracks and scars.

**Orificial Tuberculosis**
• It is a tuberculous infection of mucosa or adjoining skin from advanced internal tuberculosis.
• Patient is usually severely ill, adult having impaired CMI.
• **Morphology**: It may present with small red nodules which may breakdown to form typical tuberculous ulcers.
• **Sites**: Tip and margins of tongue (most frequent)

**Miliary Tuberculosis**
It is a rare type which results from hematogenous dissemination of mycobacteria into the skin.

**Etiology**
Usually seen in infants and children or immunosuppressed patients having HIV infection or measles.

**Morphology**
Profuse crops of papules, vesicles or pustules in a severely ill patient. At times, underlying disease may not be obvious.

**Diagnosis**
• An unusual exanthematous rash in a patient having internal tuberculosis should suggest the diagnosis.
• Diagnosis can only be confirmed by biopsy of skin in which AFB can be demonstrated.
• A search should be made for internal tuberculosis.
• The tuberculin test is negative.

**Treatment**
• Antitubercular therapy (ATT) should be started without delay.
• Prognosis is poor.

**Lupus* Vulgaris**

**Prevalence**
Most common type

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*The word “lupus” means wolf and lesion often gives an appearance as if it is eaten by a wolf.
**The word “vulgaris” is used for a common type.
Fig. 4.18: Lupus vulgaris on buttock. Annular gradually progressive plaque on buttock with crusting, induration at periphery and scarring in the central region.

Table 4.6: Differentiating features of lupus vulgaris from cutaneous leishmaniasis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Lupus Vulgaris</th>
<th>Cutaneous Leishmaniasis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Morphology:</strong></td>
<td>Reddish brown soft gelatinous plaque with advancing active margin and a trailing scar at centre</td>
<td>Follows sequence of nodule, ulceration, crusting and scar formation</td>
</tr>
<tr>
<td><strong>Diascopy:</strong></td>
<td>May show apple jelly nodules</td>
<td>Don't form plaque</td>
</tr>
<tr>
<td><strong>Ulcerative Type:</strong></td>
<td>It is progressive and may involve deep tissues and cartilage</td>
<td>Ulcer is crateriform and do not involve under line tissues</td>
</tr>
<tr>
<td><strong>Sites:</strong></td>
<td>Head, neck and buttocks</td>
<td>Bite prone sites</td>
</tr>
<tr>
<td><strong>Course:</strong></td>
<td>Tends to be chronic</td>
<td>Have a self healing tendency</td>
</tr>
</tbody>
</table>

**Tuberculous Gumma**

It results from hematogenous dissemination from a primary focus.

- **Predisposing factors:** Lowered resistance due to malnutrition, immunosuppression or underlying lymphoma.
- **Morphology:** Initially it may present as firm subcutaneous nodule or fluctuant abscess usually on extremities. Overlying skin may break down to form multiple undermined ulcer and sinuses (Fig. 4.19).
ERUPTIVE TUBERCULIDS

These are characterized as:
- Recurrent
- Disseminated and systemic reactions to tubercle bacilli toxins
- Tendency for spontaneous involution

Lichen Scrofulosorum

It consists of asymptomatic, firm, small, and follicular or parafollicular papular lesions usually confined to trunk. Lichenoid grouping is pronounced and may form plaques which tend to coalesce.

Histopathologically superficial dermal granulomas are seen in the vicinity of hair follicles or sweat ducts.

Papulonecrotic Tuberculids

It consists of eruption of symmetric crops of necrotizing papules usually on extensor aspects of extremities, buttocks, and sacral region. Pitted scars are left behind after spontaneous involution.

Erythema Induratum of Bazin

- This condition is described by Bazin in patients having etiological relationship with tuberculosis.
- Others showing no evidence of tuberculosis are referred as nodular vasculitis. It should be distinguished from erythema nodosum (Table 15.4).
- Morphology: Indolent eruption of painful erythematous subcutaneous nodules on posterior aspects of legs. Calves are often heavy column-like with associated erythrocyanosis. Nodules may become adherent to overlying skin, ulcerate and heal with scarring. Upper limbs, trunk, and buttocks may also be involved.
- Predisposing factors: Obesity, venous insufficiency, winter season and female sex.
- Histopathology: Characterized by lobular panniculitis with additional features of vasculitis.

Treatment

- Antitubercular therapy for patients who show strong positive Mantoux test or show evidence of *M. tuberculosis* deoxyribonucleic acid (DNA) by polymerase chain reaction (PCR) techniques.
- Potassium iodide is also an effective treatment.
- Elastic stockings and bed rest.

DIAGNOSIS OF SKIN TUBERCULOSIS

Diagnosis may be made under the following guidelines:

**Absolute Criteria**

Definitive diagnosis is dependent on the isolation and identification of *M. tuberculosis* from the lesion.
- Most species of mycobacteria are slow growing, 4–8 weeks may be required before growth is detected.
- Nowadays, the use of liquid media with radiometric growth detection (e.g. BACTEC-460) has replaced the traditional methods of isolation on solid media. It has decreased the time required for isolation to 2–3 weeks.
- Identification of mycobacterial DNA by PCR technique in skin specimens.

**Supporting Features**

- Characteristic clinical features
- Proved active tuberculosis elsewhere in the body.
- Demonstration of AFB in the lesion: AFBs can be seen in infection due to other mycobacteria also.
- Supportive histopathological findings: Hallmark of tuberculous infections is finding of tubercle in the dermis. Tubercle consists of:
  - Accumulation of epithelioid histiocytes and few Langhans-type giant cells.
  - Caseation necrosis in the center and a surrounding rim of lymphocytes and monocytes.
  - Highly characteristic of tuberculosis but it is not pathognomonic.
  - Identical tubercles can be produced by syphilis, leprosy, deep fungal infections, and leishmaniasis.
- A positive tuberculin sensitivity test.
- Response to ATT

Fig. 4.19: Tuberculous gumma on lateral aspect of thigh
Tuberculin Skin Test

Antigen
- Purified protein derivative (PPD) is used as antigen known as tuberculin.
- It should be refrigerated (not frozen) and kept in dark place.

Test
Three main tuberculin tests are currently in use. These include—single puncture test (Mantoux test), the multiple puncture test (Heaf test) and Tine test.
- **Mantoux test**: In India, tuberculin test is performed by using 5 TU.
- It takes 2–10 weeks for sensitivity to develop after the onset of infection with *M. tuberculosis*, and is usually life-long.
- 5 TU, i.e., 0.1 mL of purified protein derivative (PPD) is injected intradermally on volar aspect of forearm about 4 inches below the elbow joint using a 26-gauge needle by keeping the bevel upward just below the surface of skin. It will raise a wheal of 6–10 mm.
- The test is observed at 48–72 hours and widest diameter of the area of induration (not erythema) in millimeters is recorded. It is interpreted as under.
  - 0–5 mm negative
  - 6–10 mm doubtful
  - 10–20 mm positive
  - > 20 mm strong reactors

Interpretation
To have correct assessment, Mantoux test should be administered, read and interpreted with caution.
- Tuberculin test assesses delayed-type hypersensitivity (DTH) to mycobacterial antigens and is mediated by lymphocytes. DTH is associated with protective immunity.

Positive tuberculin test
It can be due to clinical or sub clinical infection.
- Positive tuberculin reaction (Fig. 4.20) in a patient having clinical tuberculosis suggests a favorable prognosis.
- PPD-positive patients are less susceptible to new *M. tuberculosis* infection than PPD-negative patients, but does not imply immunity to tuberculosis.
- A large necrotic response or blister formation (Fig. 4.21) suggests either susceptibility or presence of active disease. While, a small necrotic response indicates protection.
- A positive reaction is significant in younger age group. If a nonvaccinated child below 2 years is found to be tuberculin positive, it is an indirect evidence of an active tuberculous lesion in the body even if it has not manifested.

Negative tuberculin test
Tuberculin reaction may be altered by immunosuppressive drugs, corticosteroids and calciferol.
- Expected anergy** in a patient should not preclude Mantoux test. In such patients especially if they are

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*The test is named after Charles Mantoux, a French physician who developed it.
*Anergy: It is the inability to react to skin tests because of a weakened immune system.
nonvaccinated, a positive tuberculin test is highly suggestive of tuberculosis.

- **False negative reactions (anergy)** are seen in:
  - Infections (HIV, measles, mumps, miliary tuberculosis, typhoid, etc.)
  - Chronic renal failure, chronic liver disease
  - Neonates (below 12 weeks), elderly
  - Sarcoidosis
  - Malnutrition
  - Malignancies especially lymphoma and lymphocytic leukemia—wrong technique, e.g., subcutaneous injection
  - Improper antigen or dilution.

- **False positive reactions** can be due to:
  - BCG vaccination
  - Exposure to environmental mycobacteria
  - Over dosage or contaminated tuberculin
  - Wrong technique

### Heaf Test

Heaf Test is meant for mass screening because it does not require skill for administration as for Mantoux test. It can be administered by tuberculin coated prongs even by unskilled persons.

**Note:** The use of currently available commercial blood (serological) tests to diagnose active tuberculosis (TB) often leads to misdiagnosis, mistreatment. World Health Organization (WHO) is urging countries to ban these inaccurate and unapproved blood test.

### Treatment

#### General Measures

- Search for a tuberculous infection
  - X-ray examination of chest, bone and joints
  - Fine needle aspiration cytology (FNAC) of enlarged lymph nodes
- Investigate for HIV infection

#### Chemotherapy

- Five drugs, i.e., isoniazid, rifampicin, pyrazinamide, ethambutol and streptomycin are first line drugs.
- The first four drugs are given orally and are well-absorbed.
- Standard drug regimen consists of giving ATT for at least 6 months.
- This regimen (Table 4.7) is highly effective.

### Table 4.7: Treatment regimen for cutaneous tuberculosis

<table>
<thead>
<tr>
<th>Phases</th>
<th>Duration</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial or bactericidal or intensive phase</td>
<td>2 months</td>
<td>Isoniazid, Rifampicin, Ethambutol, Pyrazinamide</td>
</tr>
<tr>
<td>Continuation or sterilization or maintenance phase</td>
<td>4 months</td>
<td>Isoniazid, Rifampicin</td>
</tr>
</tbody>
</table>

#### Isoniazid

Due to its effectiveness (tuberculocidal), low cost and low toxicity, it finds place in all regimens.

- Peripheral neuropathy (most common) can be countered by giving 10 mg pyridoxine per day.
- Hepatitis is another common side effect in alcoholics and adults over 35 years of age.

**Dose**

5 mg/kg max 300 mg daily for 6 months

#### Rifampicin

Like isoniazid, it is a very effective tuberculocidal drug. It is also given for 6 months and is an essential component of all antitubercular regimens.

- Elevation of serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) is common, but therapy can be continued.
- Prior information regarding red discoloration of urine and other body secretions should be given to the patient.
- Effectiveness of oral contraceptives may be decreased.

**Dose**

10 mg/kg max 450 mg/day or 600 mg/day in patients weighing less than 50 kg or more than 50 kg respectively. Drug should be taken empty stomach.

#### Pyrazinamide

It is a weak tuberculocidal drug. But, it is highly effective during first 2 months of therapy when inflammatory changes are present.

- It can cause cutaneous hypersensitivity and arthritis.
- It inhibits the uric acid excretion in kidney and can precipitate gout.
- It is contraindicated in patients with liver disease.

**Dose**

15–30 mg/kg/day
Ethambutol

It is tuberculostatic and is given for first 2 months.
- Patient acceptability is very good due to fewer side effects.
- Loss of visual acuity or color vision due to optic neuritis is most important. Young children may not report their early visual impairment. So it should not be used below 6 years of age. Visual toxicity is reversible if it is recognized early and drug is discontinued.

Dose
15–20 mg/kg/day

Streptomycin

It is less tuberculocidal than isonicotinic acid hydrazide (INH) or rifampicin.
- Popularity of streptomycin in the treatment of tuberculosis has declined due to its lower margin of safety.
- It can cause ototoxicity and nephrotoxicity especially in elderly and in those having impaired renal function.
- It is used in the dose of 1 gm IM for 2 months. In patients over 50 years of age, it should be used in the dose of 0.75 g/day.

BCG Vaccination

Calmette and Guerin of the Pasteur Institute, Paris succeeded in attenuating a bovine strain of M. tuberculosis in 1906 with a view to develop a safe vaccine. They were able to evolve a strain known as Bacillus Calmette-Guerin which was harmless and capable of conferring a state of immunity when administered by vaccination.
- The vaccine (0.1 mL) is administered intradermally using a tuberculin syringe on the middle of deltoid region (below the shoulder) of the left arm. A satisfactory injection will raise a wheal of about 5 mm diameter.
- After BCG vaccination, a small papule occurs after 2–6 weeks.
- It may enlarge and leave a shallow ulcer after discharging purulent material.
- Regional lymph nodes may be enlarged but do not breakdown.

Efficacy
- Tuberculin conversion takes place within 5–12 weeks after vaccination.
- BCG affords 80% protection for 15 years.
- It protects the host by blocking the secondary hematogenous spread of pathogen and limits the primary infection to subclinical levels.

Complications
- Complications are rare. There may be nonspecific reactions like urticaria and erythema multiforme.
- Rarely, lupus vulgaris may develop at the site of vaccination.

Uses
- Tuberculin negative health workers and staff exposed to AIDS patients should be offered BCG vaccination.
- Immunotherapy of transitional cell carcinoma of bladder. Vaccine is instilled into bladder at monthly intervals.
- To increase CMI in lepromatous leprosy patients twice weekly injections of BCG can be given in combination with antileprotic treatment.

VIRAL INFECTIONS

Viruses are not considered as unicellular microorganisms due to following reasons:
- They do not possess a cellular organization. Even the simplest microorganism must have a cell wall containing both nucleic acids, i.e., DNA and ribonucleic acid (RNA). But viruses consist of only one type of nucleic acid, either DNA or RNA.
- They are obligate intracellular parasites. They do not possess functional ribosome. They require synthetic machinery of host cells for their replication.
- They multiply by a complex process and not by binary fission.
- They are unaffected by antibacterial antibiotics.

In spite of these facts, viruses are generally considered as microorganisms. They are not given cellular status.

LIFE CYCLE

The genetic information of a virus is stored inside an element known as viral genome which may be either DNA or RNA. The life cycle of a virus consists of two parts.
1. In one part, the viral genome gets surrounded by a protective protein coat (capsid) to constitute an extracellular particle known as virion. Virion enables the transmission of viral genome to a susceptible host. The capsid is arranged in one of the two patterns in most viruses—helical or icosahedral. Capsid of poxviruses is more complex and is classified as complex.
2. The second part of viral life cycle refers to the presence of viral genome inside the cell in a nonparticulate form. - In certain viral families (herpes viruses and retroviruses) capsid is surrounded by virion
envelope which is required for infectivity. This viral envelope is sensitive to drying. So, infectivity of these viruses is lost on drying.
- Poxvirus virion is also enveloped, but the envelope is not required for this virus to be infectious. Hence, poxviruses remain infective even after drying. The virions of other viruses (e.g. papilloma viruses) do not have envelopes. Their naked capsids are stable in dry environments and remain infectious for long periods after drying.

CLASSIFICATION
DNA viruses include:
- Herpes viruses
- Parovirus
- Papovaviruses (human papovaviruses)
- Poxviruses (molluscum contagiosum virus)
- Parapoxviruses (Milker’s nodule virus)
RNA viruses include:
- Paramyxovirus (measles virus)
- Rubivirus (rubella virus)
- Picornaviruses (Caxsackievirus A16 and enterovirus 71)
- Retroviruses (HIV)

HERPES VIRUSES
Nomenclature
The herpes family of viruses include eight different viruses that affect human beings. The formal names according to the International Committee on Taxonomy of Viruses (ITCV) are known by numerical designations (Table 4.8).

Table 4.8: Formal names of herpes viruses

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Formal names</th>
<th>Informal names</th>
</tr>
</thead>
<tbody>
<tr>
<td>α</td>
<td>Human herpes virus 1 (HHV1)</td>
<td>Herpes simplex virus type 1</td>
</tr>
<tr>
<td></td>
<td>Human herpes virus 2 (HHV2)</td>
<td>Herpes simplex virus type 2</td>
</tr>
<tr>
<td></td>
<td>Human herpes virus 3 (HHV3)</td>
<td>Varicella zoster virus</td>
</tr>
<tr>
<td>β</td>
<td>Human herpes virus 5 (HHV5)</td>
<td>Cytomegalovirus</td>
</tr>
<tr>
<td></td>
<td>Human herpes virus 6 (HHV6)</td>
<td>HHV6A is neurovirulent and is found in multiple sclerosis.</td>
</tr>
<tr>
<td></td>
<td>Human herpes virus 7 (HHV7)</td>
<td>HHV6B and HHV7 together often referred as roseola infantum virus</td>
</tr>
<tr>
<td>γ</td>
<td>Human herpes virus 4 (HHV4)</td>
<td>Epstein Barr virus</td>
</tr>
<tr>
<td></td>
<td>Human herpes virus 8 (HHV8)</td>
<td>Kaposi’s sarcoma-associated herpes virus (KSHV)</td>
</tr>
</tbody>
</table>

Features
- They replicate inside the nucleus and produce intranuclear inclusion bodies.
- After clinical recovery, virus is not eliminated from the body, but persists throughout life as latent infection in certain specific nerve cells for that strain.
- The latent infection becomes reactivated under certain conditions.

HERPES SIMPLEX
Etiology
It is caused by herpes simplex virus (HSV) (herpesvirus hominis). It comprises two antigenic types.
1. HSV-1 is usually associated with facial infections and occurs mainly in infants and young children.
2. HSV-2 is classically found in genital infections and occurs mainly after puberty and often transmitted sexually.

The sites infected by the two HSV types are not mutually exclusive and show considerable overlap in the sites of involvement.

Route of Transmission
May be through skin or mucosa by:
- By direct contact or droplets of infection: It is usually seen in children.
- By sexual contact: It is usually seen in adults.

Herpes simplex virus (HSV) infections are best described to have three stages.
1. Primary infection: After transmission, virus replicates at the inoculation site and results into primary lesions. The virus then infects sensory nerve terminals and travels by retrograde axonal transport to the nuclei of neurons in regional sensory ganglion.
2. Latent infection: After reaching sensory ganglion, HSV gene expression is severely restricted and produces no viral proteins. Therefore, remains undetected by host defense mechanisms.
3. Recurrent infection: In this last stage, due to certain risk factors like old age and cellular immune dysfunction, the latent virus starts replicating and via antegrade axonal transport reaches peripheral site where it causes recurrent disease.

Histopathology (Fig. 4.22)
Profound degeneration of keratinocytes is an important feature which results in acantholysis. Degeneration occurs in two forms:
1. In ballooning degeneration, the cytoplasm of infected epithelial cells becomes edematous. These cells are...
called as balloon cells which lose their intercellular bridges and get separated from one another. These are mainly seen at bases of vesicles.

- In addition, there is formation of intranuclear inclusion bodies inside the nuclei of balloon cells. The nucleus exhibits margination of chromatin and inclusion bodies are of classical Cowdry Type A*.
- Nonspecific reticular degeneration is seen in the upper portions and at the periphery of vesicles. It results due to cell wall rupture of distended or edematous epidermal cells and lead to formation of multilocular vesicle (2–15).
- Multinucleate (2–15) giant cells are invariably observed in cutaneous epithelium.
- The dermis is infiltrated by neutrophils.

**Clinical Features**

**Primary Infection**

It is often subclinical. It comprises following clinical entities.

- **Orofacial infections:**
  - Herpetic gingivostomatitis is the most common manifestation of primary HSV-1 infection. It may also be caused by HSV-2 infection.
  - It is mostly seen in children (1–5 years).
  - Incubation period: About 5 days.
  - Manifests as stomatitis, fever, restlessness, dribbling and painful eating and drinking.
  - On examination, gums are swollen and bleed easily. The vesicles are seen as white plaques on the tongue, pharynx, palate and buccal mucosa. Plaques develop into ulcers covered with yellowish pseudomembrane. Neighboring facial areas may be involved.
  - Lymph nodes may be enlarged and tender.
  - Fever subsides in 3–5 days and patient recovers in about 2 weeks.
- **Herpes genitalis:** (Table 21.6)

- **Keratoconjunctivitis:** Primary infection may cause purulent conjunctivitis, corneal ulceration.
- **Herpetic whitlow:**
  - It is an inoculation herpes simplex by direct inoculation from mucosa during primary infection.
  - Seen in children who suck their fingers or in medical personnel as an occupational hazard.
  - Painful deep vesicles at the fingertip give a honeycombed appearance.
  - Surgical drainage is potentially harmful. Antiviral therapy speeds up healing.
- **Herpes gladiatorum:** **It** is an infection seen in wrestlers acquired secondary to direct contact with opponents.

**Complications of primary HSV-1 infections**

- **Eczema herpeticum (Kaposi’s varicelliform eruption):**
  - Etiology: It results from widespread infection of damaged skin due to HSV-1 in atopic child or patients on immunosuppressive therapy, etc.
  - Morphology: Several days after exposure to the virus, patient presents with large number of vesicles on recently healed areas of atopic dermatitis. The vesicles may become pustular and umbilicated.
  - High fever and adenopathy usually accompany the rash.
  - Involvement of internal organs due to viremia can be fatal (mortality 10%).
  - So, it should be considered in child with infected eczema who is more ill and toxic than expectation.
  - It is a medical emergency and early IV therapy with acyclovir may be proved life-saving.
- **Systemic or disseminated infection:** May occur in immunodeficient and neonates (not protected by maternally acquired antibodies).
- **Encephalitis:** Headache and meningism may be seen in patients having primary genital herpes simplex.
- **Radiculoneuropathy:**
  - Seen in women or homosexual men having primary anogenital infection.
  - It manifests as urinary retention, sacral paresthesia and impotence.
  - It recovers in a few weeks.
- **Keratitis:** Dendritic corneal ulcers may cause opacities and blindness.
- **Erythema multiforme (EM).**

**Recurrent Infection**

Recurrence after a period of latency is a troublesome and common feature of HSV infection.

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*In this type, sharply demarcated eosinophilic inclusions are separated from the deeply basophilic ring of marginated chromatin by clear halo.

**Eponyms are wrestler’s herpes or mat pox (after wrestling) and herpes rugbiorum or scrum pox (after rugby or football).
It occurs due to reactivation of HSV-1 in trigeminal ganglion and HSV-2 in sacral ganglion.

**Triggering factors:** Minor trauma, emotional stress, premenstrual period, etc.

**Sites of predilection:** Outer third of lower lip and perioral areas (herpes labialis).

**Morphology:** Small, closely grouped vesicles in herpes labialis (Fig. 4.23) on erythematous base, commonly preceded by pain or burning sensation.

**Course:** Crust forms in few days and heals completely in 7–14 days.

**Complication:** Recurrent precipitating factor for recurrent EM.

**Diagnosis**

Many a times, following clinical findings are sufficient to make a diagnosis of herpes labialis.

- Presence of grouped tiny vesicles or polycyclic erosions near the angle of mouth.
- History of recurrence.

**Differential Diagnosis**

Sometimes, it needs to be differentiated from herpes zoster (Table 4.9).

**Investigations**

- **Tzanck smear (Fig 4.24):**
  - Prepare from the base of freshly ruptured vesicle.
  - Stained with Giemsa and Wright stain.

### Table 4.9: Difference between herpes zoster and herpes labialis

<table>
<thead>
<tr>
<th></th>
<th>Herpes zoster</th>
<th>Herpes labialis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prodrome:</strong></td>
<td>Severe dermatomal pain</td>
<td>Mild burning or stinging sensation</td>
</tr>
<tr>
<td><strong>Morphology:</strong></td>
<td>Group vesicles on erythematous base. Such groups are multiple in a dermatomal pattern</td>
<td>Groups of tiny vesicles on an erythematous base or erosions with polycyclic margins</td>
</tr>
<tr>
<td><strong>Recurrence:</strong></td>
<td>Do not occur. But can occur in HIV infected patients</td>
<td>Very common at the same site in an otherwise healthy man</td>
</tr>
</tbody>
</table>

*Abbreviation: HIV, human immunodeficiency virus.*

- Search is made for multinucleated giant cells [diagnostic for both HSV and varicella zoster virus (VZV) infections].
- **Cell culture:** Not available at all centers.
- Polymerase chain reaction technique is more sensitive than viral isolation. Both PCR and viral culture enable the typing of isolate as HSV-1 or HSV-2. This information will help in predicting recurrence rate. Recurrence develops in 95% of individuals having genital herpes due to HSV-2 in comparison with 50% of those due to HSV-1 type.
- The detection of HSV DNA in cerebrospinal fluid PCR is the diagnostic method of choice for herpes encephalitis.
- **Direct fluorescent antibody staining technique:** It will detect antigen by staining the lesional scrapings.

- Serological detection of antibodies to HSV is helpful in differentiating a primary episode from a recurrent infection.
- **Skin biopsy**

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![Fig 4.23: Herpes labialis showing small grouped vesicles on erythematous base](image)

![Figs 4.24: Tzanck smear showing multinucleate giant cells and intranuclear inclusion bodies](image)
Management of HSV Infections

Refrain from intercourse during outbreak of recurrence and for one to two days after. Condoms should be used between outbreaks.

- **Mild uncomplicated eruptions:**
  - Need no systemic antiviral treatment
  - Keep the lesions clean and dry is all that is required
  - Topical antibacterial agents may be used to treat secondary bacterial infection.
- For severe primary infection, antiviral therapy should be started.

**Antiviral Therapy**

**Topical antiviral agents**

- **Herpes simplex labialis:**
  - Penciclovir 1% cream (denavir) every 2 hours.
  - Docosanol 10% cream (abrev) five times a day.
  - **Acyclovir cream and ointments** are not beneficial.
- **Primary herpes genitalis:** Acyclovir cream and ointments (applied 4 to 5 times) are beneficial.
- **Recurrent herpes genitalis:**
  - Topical imiquimod has some beneficial effect.
  - Topical acyclovir in not beneficial.

**Systemic antiviral agents (Table 4.10)**

- **Aciclovir** gets converted to aciclovir triphosphate by cellular kinases. Aciclovir triphosphate competes with natural substrate and gets incorporated into viral DNA. This incorporation prevents chain elongation and terminates viral DNA synthesis.
- **Valaciclovir** is an oral prodrug of aciclovir. With oral valaciclovir, bioavailability of aciclovir is 3–5 times greater than that achieved after oral aciclovir. Its dosage regimens are also convenient.

**Note:**

- Valaciclovir and famciclovir are not approved for use in children. Use of valaciclovir is contraindicated in immuno-compromised patients. It can cause thrombocytopenic purpura and hemolytic uremic syndrome in them.
- For aciclovir resistant HSV infections, foscarnet* and cidofovir are two antiviral agents which can be used.

**VARICELLA AND HERPES ZOSTER**

Varicella and herpes zoster are two distinct clinical entities which are caused by the same virus, referred as VZV.

**Varicella (Chickenpox)**

**Epidemiology**

- **Prevalence:** Very common
- **Age:** Often during childhood
- **Season:** Common in cooler months (January to April), while it drops off in summer months

**Etiology**

- **Causative agent:** VZV
- **Route of transmission:** Respiratory tract through the droplets shed from the patients
- **Incubation period:** It is about 2 weeks (10–23 days)
- **Infectivity:** Varicella is highly infectious. Infective period is between 1 day and 2 days before the eruption and 4–5 days thereafter, i.e., until last crop of vesicles has crusted. The varicella crusts (Fig. 4.25C) are not infectious. Zoster is less (one-third) infectious than varicella.

**Pathogenesis**

**Primary viremia**

Initially virus multiplies at portal of entry. Then, disseminates via blood and lymphatics and results into primary viremia.

The virus is then cleared by reticuloendothelial system which is the major site of viral replication during incubation period.

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**Table 4.10: Dosage regimen of antiviral therapy for orofacial herpes**

<table>
<thead>
<tr>
<th></th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary infection</strong> (7–10 days)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aciclovir</td>
<td>200 mg 5 times/day</td>
<td>15 mg/kg/day orally</td>
</tr>
<tr>
<td></td>
<td>or 400 mg TDS 5 times a day</td>
<td></td>
</tr>
<tr>
<td>Valaciclovir</td>
<td>1,000 mg BD</td>
<td></td>
</tr>
<tr>
<td>Famciclovir</td>
<td>250 mg TDS</td>
<td></td>
</tr>
<tr>
<td><strong>Recurrent infection</strong> (1 day)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aciclovir</td>
<td>400 mg 5 times/day</td>
<td></td>
</tr>
<tr>
<td>Valaciclovir</td>
<td>500 mg BD</td>
<td></td>
</tr>
<tr>
<td>Famciclovir</td>
<td>500 mg two to three times/day</td>
<td></td>
</tr>
<tr>
<td><strong>Prophylaxis and suppressive therapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aciclovir</td>
<td>400 mg BD</td>
<td></td>
</tr>
</tbody>
</table>

Eczema herpeticum, disseminated infection and encephalitis may preferably be treated with intravenous aciclovir in the dose of 25mg/kg/day (divided in to five equal doses) for 5–10 days

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*Foscarnet: Antiviral drug which can cause penile ulcers.

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Secondary viremia
The virus eventually overwhelms the developing immune responses and results into much larger secondary viremia with the appearance of symptoms of varicella.
- Successive crops of vesicles appear which are terminated in 3–4 days by immune system.
- Cell-mediated immunity is more important. Organ transplant patients may have severe and fatal varicella.

Transportation
During varicella, VZV travels from skin and is transported centripetally up along the sensory fibers to the sensory ganglia.

The virus establishes there as a latent infection and persists for life.

Recurrence
Under the influence of certain risk factors (older age, immunosuppression, local trauma, irradiation of spinal column, tumor involvements, etc.) gets reactivated and multiplies in the ganglia.

Virus then spreads down and is released around the sensory nerve endings where it produces vesicles in cluster. Such clusters in a dermatome manifest as herpes zoster.

Histopathology
Histopathological changes in lesions of varicella and herpes zoster are indistinguishable and similar to those seen in herpes simplex.

Clinical Features
It is an acute, highly contagious exanthem.

Prodromal symptoms
- In children: Mild and inconspicuous.
- In older children and adults: Fever, malaise, headache and anorexia often precedes the rash by 2–3 days.

Rash
- Begins on the face and spreads down to trunk.
- Lesions are most profuse on trunk (Figs 4.25A and B) and least on the peripheral parts of limbs (centripetal distribution).
- Lesions appear in crops and progress rapidly from macules to papules, vesicles, pustules, and crusts.
- The vesicles are smaller in size and are surrounded by irregular area of erythema giving a dew drop on rose petal appearance.

Figs 4.25A to C: Varicella. A. Centripetal distribution; B. Magnified view showing a “dew drop on rose petal” appearance; C. Crusts and shallow depressions.