DERMA CLINICAL POSTING NOTES -

FINAL YEAR MBBS – SDNOTES - EDITION - 02

DR SD CLINICALS

Topics –

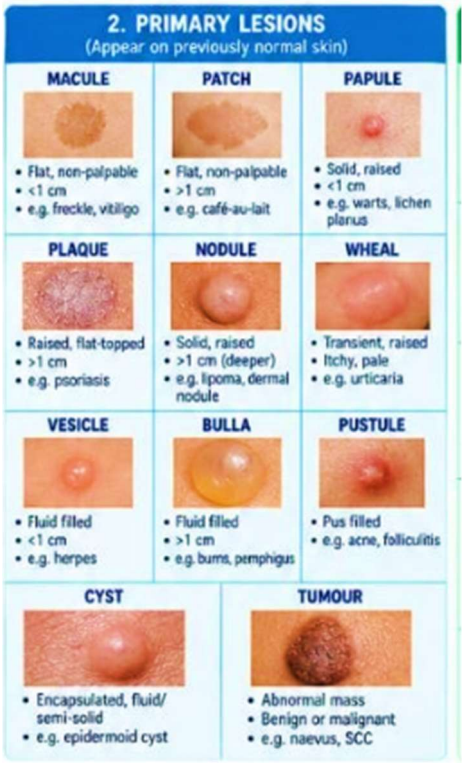
S. No.	Date	Topic
1	01-09-2026	Fungal Infections
2	02-09-2026	Viral Infections
3	03-09-2026	Bacterial Infections
4	04-09-2026	Scabies and Pediculosis
5	05-09-2026	Acne Vulgaris
6	07-09-2026	Leprosy
7	08-09-2026	Vitiligo
8	09-09-2026	Psoriasis
9	10-09-2026	Syphilis
10	11-09-2026	Non-Syphilis STD
11	12-09-2026	Vesiculobullous Disorders
12	15-09-2026	End of Posting

September 2026

LESIONS OF SKIN

A skin lesion is an abnormal change in the skin in colour, contour, texture or structure.

1. CLASSIFICATION OF SKIN LESIONS



A. Primary Lesions

Occur on previously normal skin.

1. Macule
2. Patch
3. Papule
4. Plaque
5. Nodule
6. Wheal
7. Vesicle
8. Bulla
9. Pustule
10. Cyst
11. Tumour

B. Secondary Lesions

Result from the evolution or alteration of primary lesions.

1. Scale
2. Crust
3. Erosion
4. Ulcer
5. Fissure
6. Excoriation
7. Lichenification
8. Atrophy
9. Scarring
10. Keloid
11. Eschar



2. PRIMARY SKIN LESIONS

1. Macule

- Flat, non-palpable lesion.
- Size: less than 1 cm.
- Examples:
    - Freckle
    - Vitiligo
    - Drug eruption

2. Patch

- Flat, non-palpable lesion.
- Size: greater than 1 cm.
- Examples:
    - Café-au-lait macule
    - Vitiligo

3. Papule

- Solid, raised lesion.
- Size: less than 1 cm.
- Examples:
    - Warts
    - Lichen planus
    - Insect bite

4. Plaque

- Raised, flat-topped lesion.
- Size: greater than 1 cm.
- Examples:
    - Psoriasis
    - Chronic eczema

5. Nodule

- Solid, raised lesion.
- Usually deeper than a papule.
- Examples:
    - Lipoma
    - Dermal nodule

6. Wheal

- Transient, raised lesion.
- Itchy and pale.
- Example:
    - Urticaria

7. Vesicle

- Small, fluid-filled lesion.
- Size: less than 1 cm.
- Example:
    - Herpes

8. Bulla

- Large, fluid-filled lesion.
- Size: 1 cm or greater.
- Examples:
    - Burns
    - Pemphigus vulgaris

9. Pustule

- Contains pus.
- Examples:
    - Acne
    - Folliculitis

10. Cyst

- Encapsulated, fluid-filled or semi-solid lesion.
- Example:
    - Epidermoid cyst

11. Tumour

- Abnormal mass.
- May be benign or malignant.
- Examples:
    - Naevus
    - Squamous cell carcinoma (SCC)

3. SECONDARY SKIN LESIONS

1. Scale

- Lamellar, flaky fragments of the stratum corneum.
- Example: Psoriasis.

2. Crust

- Dried serum, pus or blood.
- Example: Impetigo.

3. Erosion

- Loss of epidermis only.
- Usually heals without scarring.

4. Ulcer

- Loss of epidermis and dermis.
- Heals with scarring.

5. Fissure

- Linear crack in the skin.
- Examples:
    - Eczema
    - Fissured heel

6. Excoriation

- Linear skin damage caused by scratching.
- Example: Pruritic dermatoses.

7. Lichenification

- Thickened skin with accentuated skin markings.
- Example: Chronic eczema.

8. Atrophy

- Thinning of the skin.
- Example: Steroid-induced skin atrophy.

9. Scar

- Replacement of damaged tissue by fibrous tissue.
- Example: Acne scar.

10. Keloid

- Excessive collagen deposition.
- Extends beyond the original wound.

11. Eschar

- Dark, dry, necrotic tissue.

4. HOW TO DESCRIBE A SKIN LESION

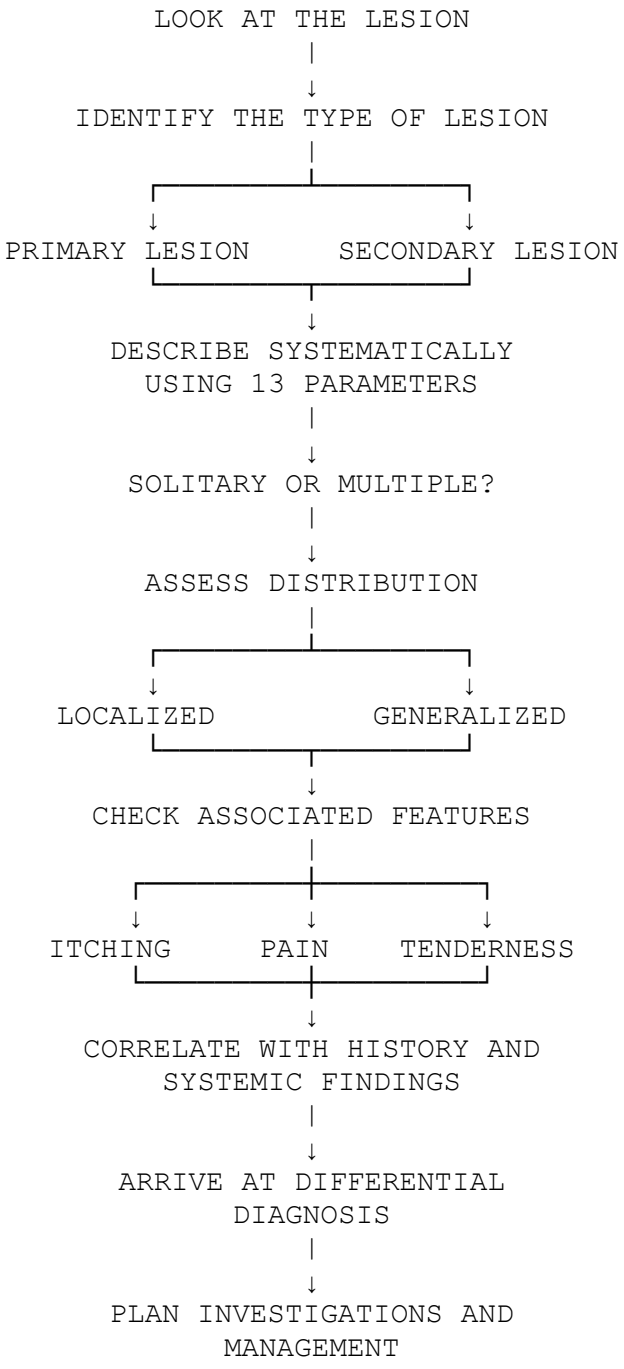
Use the following 13 parameters during clinical examination.

1. Site: Exact location and distribution.
2. Number: Solitary or multiple.
3. Size: Measure in cm or mm.
4. Shape: Round, oval, irregular or linear.
5. Colour: Erythematous, hypopigmented, pale, etc.
6. Surface: Smooth, scaly, crusted or verrucous.
7. Margin: Well-defined or ill-defined.
8. Elevation: Flat, raised or depressed.
9. Consistency: Soft, firm or cystic.
10. Tenderness: Present or absent.
11. Temperature: Hot, normal or cold.
12. Surrounding skin: Normal, inflamed or oedematous.
13. Associated features: Itching, pain, discharge or bleeding.

ABCDE of lesion description

- A: Arrange systematically.
- B: Be specific.
- C: Complete details.
- D: Do not miss secondary changes.
- E: Examine all areas.

5. APPROACH TO A SKIN LESION -



6. IMPORTANT CLINICAL POINTS

- Always examine the skin in good light.
- Use proper anatomical terminology.
- Do not miss secondary changes.
- Look for distribution patterns:
  - Acral
  - Flexural
  - Extensor
- Examine hair, nails and mucosa.
- Compare lesions with normal skin.
- Take relevant history:
  - Duration
  - Progression
  - Itching
  - Triggers
- Consider systemic associations.

7. COMMON EXAMPLES OF SKIN LESIONS

Lesion	Common conditions
Macule	Freckle, vitiligo, drug eruption
Patch	Café-au-lait, vitiligo
Papule	Warts, lichen planus, insect bite
Plaque	Psoriasis, chronic eczema
Nodule	Dermatofibroma, lipoma
Wheal	Urticaria
Vesicle	Chickenpox, herpes
Bulla	Burns, pemphigus vulgaris
Pustule	Acne, folliculitis
Cyst	Epidermoid cyst
Tumour	Naevus, basal cell carcinoma
Scale	Psoriasis, seborrhoeic dermatitis
Crust	Impetigo
Erosion	Herpes, ruptured vesicle
Ulcer	Diabetic ulcer, tuberculosis
Fissure	Chronic eczema, fissured heel
Lichenification	Atopic dermatitis
Scar	Post-chickenpox, post-surgery
Keloid	Keloid tendency

8. IN LIST -

Primary Lesions

- Macule
- Patch
- Papule
- Plaque
- Nodule
- Wheal
- Vesicle
- Bulla
- Pustule
- Cyst
- Tumour

Secondary Lesions

- Scale
- Crust
- Erosion
- Ulcer
- Fissure
- Excoriation
- Lichenification
- Atrophy
- Scarring
- Keloid
- Eschar

9. PRIMARY VS SECONDARY LESIONS

Feature	Primary lesion	Secondary lesion
Origin	Appears on normal skin	Results from alteration or evolution of a lesion
Examples	Macule, papule, vesicle	Scale, crust, ulcer
Significance	Helps identify the initial disease pattern	Shows evolution, duration or complications
Management	Treat the underlying cause	Treat the cause and prevent complications

10. REVISE -

- Macule: Less than 1 cm, flat.
- Patch: Greater than 1 cm, flat.
- Papule: Less than 1 cm, raised.
- Plaque: Greater than 1 cm, raised and flat-topped.
- Vesicle: Less than 1 cm, fluid-filled.
- Bulla: 1 cm or greater, fluid-filled.
- Pustule: Pus-filled.
- Nodule: Solid, deep lesion.
- Erosion: Loss of epidermis, usually no scar.
- Ulcer: Loss of epidermis and dermis, usually heals with a scar.
- Scale: Flaky keratin.
- Crust: Dried exudate.
- Fissure: Linear crack.
- Excoriation: Scratch mark.
- Lichenification: Thickened skin with prominent markings.
- Keloid: Excessive scar tissue extending beyond the original wound.

Remember - See the lesion → Describe the lesion → Correlate clinically → Reach the diagnosis.

NOTES -

FUNGAL INFECTIONS IN DERMATOLOGY

THINK FUNGUS Think KOH **-**Think ANTIFUNGAL - Think TARGETED TREATMENT

1. CLASSIFICATION OF FUNGAL INFECTIONS

Fungal Infections

→ Superficial
(stay in stratum corneum)

- Pityriasis versicolor
- Tinea nigra

→ Cutaneous
(keratinized tissue: skin, hair, nails)

- Dermatophytoses (Tinea)
- Candidiasis
- Others

→ Subcutaneous
(deeper tissue)

- Sporotrichosis
- Chromoblastomycosis
- Mycetoma
- Phaeohyphomycosis

2. COMMON FUNGI & FEATURES

Type	Organism	Key Features
Dermatophytes	Trichophyton, Microsporum, Epidermophyton	Keratinophilic; cause tinea of skin, hair and nails
Yeasts	Candida spp.	Moist areas; satellite lesions; pseudohyphae
Malassezia	M. furfur	Pityriasis versicolor; superficial; diagnosed with KOH
Dimorphic fungi	Sporothrix schenckii	Sporotrichosis; "rose gardener's disease"
Dematiaceous fungi	Fonsecaea, Phialophora	Chromoblastomycosis
Actinomycetes-like fungi	Madurella etc.	Mycetoma

3. MODES OF TRANSMISSION

- **Direct contact** → Human/animal
- **Zoonotic** → Cats, dogs, e.g. M. canis
- **From soil** → Geophilic fungi
- **Fomite** → Towels, clothing, combs, footwear

- **Autoinoculation** → Scratching
- **Predisposing factors**
  - Heat
  - Humidity
  - Occlusion
  - Diabetes
  - Immunosuppression

4. DIAGNOSIS: STEPWISE APPROACH

Clinical suspicion
↓
KOH mount
↓
Culture, if needed
↓
Histopathology, when indicated

Important investigations-

- **KOH examination**
- **Fungal culture**
- **Histopathology**
- Clinical examination

5. DERMATOPHYTOSIS / TINEA

Clinical Types -

Type	Site	Important Features
Tinea corporis	Body	Annular, erythematous, scaly patch with active edge and central clearing
Tinea cruris	Groin	Pruritic, erythematous plaques; scrotum usually spared; "Jock itch"
Tinea pedis	Foot	Interdigital maceration/scaling; vesicles; moccasin type
Tinea manuum	Hand	Unilateral often; associated with tinea pedis; "two feet-one hand syndrome"
Tinea capitis	Scalp	Black dot/gray patch; kerion; boggy swelling; common in children
Tinea barbae	Beard	Inflammatory plaques/nodules; common in men
Tinea unguium	Nails	Thickened, discoloured brittle nail; subungual debris; difficult to treat

Clinical images looks like -

- Tinea corporis: annular erythematous lesion with central clearing
- Tinea cruris: groin erythematous plaque
- Tinea pedis: interdigital/moccasin lesions
- Tinea manuum: scaling of hand
- Tinea capitis: scalp involvement

- Tinea barbae: inflammatory beard lesion
- Tinea unguium: thickened/discoloured nail



6. ORGANISMS - REMEMBER

Organism	Typical Site / Feature
Trichophyton	Skin + hair + nails
Microsporum	Skin + hair; fluorescence under Wood's lamp
Epidermophyton	Skin + nails
Candida	Moist areas, mucosa, nails
Malassezia	Pityriasis versicolor, superficial
Sporothrix	Subcutaneous, rose gardener

7. CANDIDIASIS

1. Intertrigo

- Beefy red, moist
- Satellite pustules
- Skin folds involved:
    - Groin
    - Breasts
    - Groin/axillary folds

2. Oral Thrush

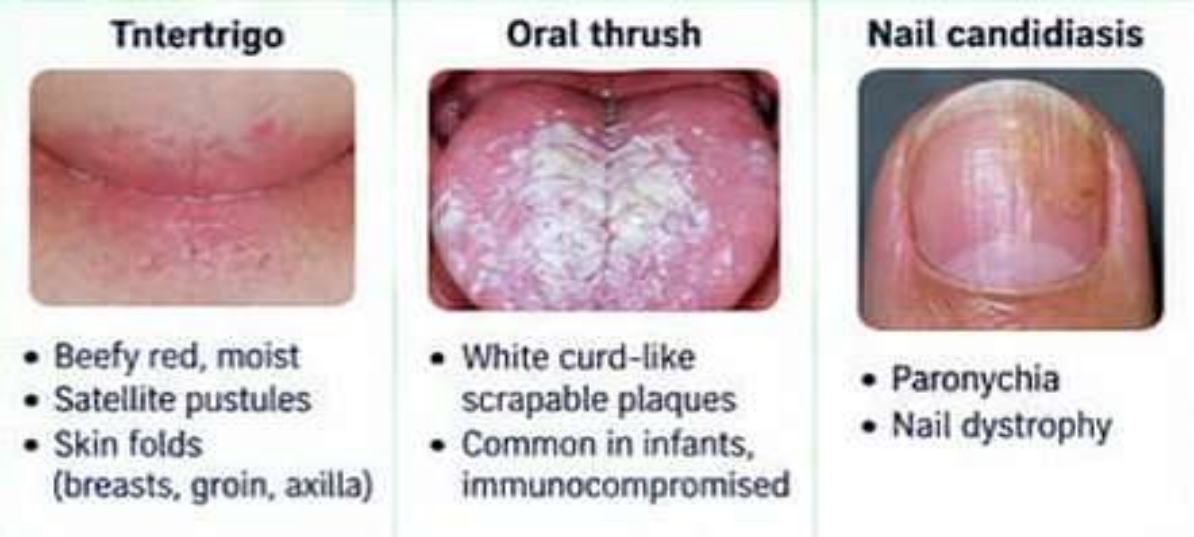
- White curd-like scrapable plaques
- Common in immunocompromised patients

3. Nail Candidiasis

- Paronychia
- Nail dystrophy

Typical image appearances -

- Beefy-red intertriginous rash with satellite pustules
- White oral plaques
- Inflamed nail fold with nail dystrophy



8. PITYRIASIS VERSICOLOR

Causative organism: Malassezia furfur

Features

- Caused by **Malassezia**
- Multiple hypo/hyperpigmented macules
- Fine bran-like scale
- Common on:
  - Trunk
  - Neck
  - Proximal extremities
- **KOH:** "Spaghetti & meatballs" appearance

Image Looks like -

Multiple hypopigmented/light-brown scaly macules over the trunk.



9. SUBCUTANEOUS MYCOSES

A. Sporotrichosis

- Traumatic inoculation
- Nodular lymphangitis
- "Sporotrichoid spread"
- Associated with plants
- **"Rose gardener's disease"**

B. Chromoblastomycosis

- Warty plaques
- Common on lower limbs
- Caused by dematiaceous fungi such as:
    - Fonsecaea
    - Phialophora

C. Mycetoma

- Triad:
    - **Triad swelling**
    - **Sinus formation**
    - **Discharge with grains**
- Caused by fungi (**eumycetoma**) or actinomycetes
- Common on tropical areas

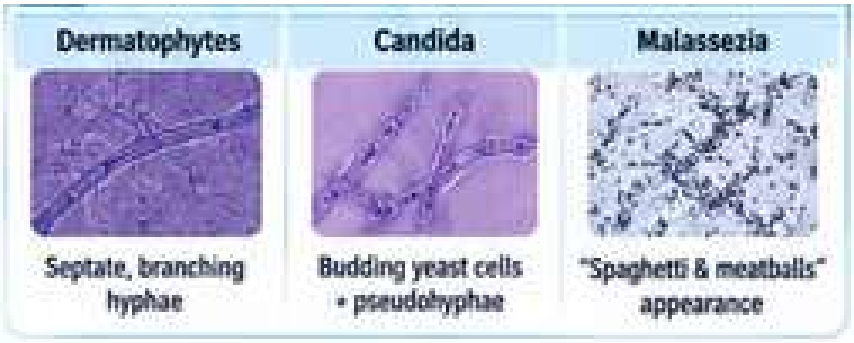
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10. KOH FINDINGS: HIGH-YIELD

Organism	KOH Finding
Dermatophytes	Septate, branching hyphae
Candida	Budding yeast cells + pseudohyphae
Malassezia	"Spaghetti & meatballs" appearance

Dermatophytes → Septate hyphae
Candida → Budding yeast + pseudohyphae
Malassezia → Spaghetti & meatballs



11. TREATMENT -

- **Topical antifungals** for limited superficial infections
- **Systemic antifungals** for extensive, recurrent, hair/nail/subcutaneous infections
- Treat **adequately for the appropriate duration**
- Treat affected family members/pets when indicated
- Keep area **dry**
- Avoid occlusive clothing
- Avoid topical steroids alone because of risk of **tinea incognito**

Common antifungal drugs

- Clotrimazole
- Ketoconazole
- Terbinafine
- Fluconazole
- Itraconazole
- Voriconazole
- Amphotericin B

12. DIFFERENTIAL DIAGNOSIS OF ANNULAR LESIONS

Condition	Clue
Tinea corporis	Active scaly edge + central clearing
Nummular eczema	More uniform, no active edge
Psoriasis	Well-demarcated, silvery scale
Granuloma annulare	Non-scaly, firm papules
Erythema multiforme	Target-like, migratory
Secondary syphilis	Generalized rash, serology positive

Identifications -

1. **Identify the type:** superficial / cutaneous / subcutaneous.
2. **KOH is simple and very useful.**
3. **Treat adequately and prevent recurrence.**
4. Think of fungal infection when there is an **annular scaly lesion with an active border**.
5. **KOH patterns are high-yield for exams and clinical practice.**

"When in doubt, do a KOH!"

Spaghetti + meatballs → Malassezia
Budding yeast + pseudohyphae → Candida
Septate branching hyphae → Dermatophytes

FUNGUS FACTS TO REMEMBER

- **Tinea = Dermatophyte**
- **Satellite lesions = Candida**
- **Spaghetti & meatballs = Malassezia**
- **Sporotrichoid spread = Tinea?** No, **Sporotrichosis**
- **Griseofulvin → Tinea capitis**
- **Nystatin → Candida**
- **Steroid misuse → Tinea incognito**
- **Keep it dry, keep it clean!**

Notes –

VIRAL INFECTIONS OF SKIN

- Common, contagious and self-limiting in many cases
- Diagnosis mainly clinical
- Confirm with **Tzanck smear / PCR** where needed
- Treatment is mostly symptomatic
- Antiviral therapy is used in selected cases
- Prevention by vaccination, hygiene and avoiding contact

2. CLASSIFICATION BY VIRUS FAMILY

Virus Family	Important Viruses / Diseases
Herpesviridae	HSV-1, HSV-2, Varicella zoster virus (VZV), EBV, CMV
Poxviridae	Molluscum contagiosum, Smallpox, Mpox
Papillomaviridae	HPV → Warts
Picornaviridae	Hand, Foot and Mouth Disease
Others	Measles virus, Rubella virus, Parvovirus B19

3. COMMON MODES OF TRANSMISSION

1. **Direct contact** → skin-to-skin
2. **Droplet spread** → respiratory
3. **Fomite** → towels, clothes, razors
4. **Autoinoculation** → scratching
5. **Vector transmission** → e.g. mosquito for some arboviruses
6. **Vertical transmission** → mother to child

4. IMPORTANT VIRAL INFECTIONS

Disease	Causative Virus	Clinical Features	Characteristic Features	Treatment
Herpes Simplex (HSV-1/HSV-2)	Herpes simplex virus	Grouped vesicles on erythematous base; painful; recurrent; oral or genital	Recurrent episodes; burning/tingling prodrome	Acyclovir oral/topical; symptomatic
Varicella (Chickenpox)	Varicella zoster virus	Generalized itchy vesicles in different stages	Crops of lesions; more common in children	Supportive; acyclovir in severe/immunocompromised
Herpes Zoster (Shingles)	Varicella zoster virus	Painful unilateral grouped vesicles in dermatomal distribution	Dermatomal; does not cross midline; post-herpetic neuralgia	Acyclovir/Valacyclovir; pain control
Molluscum Contagiosum	Molluscum contagiosum virus (Poxvirus)	Multiple small, dome-shaped umbilicated papules	Central umbilication; common in children/immunocompromised	Curettage, cryotherapy, topical agents e.g. KOH, TCA
Viral Warts (Verruca)	Human papilloma virus (HPV)	Hyperkeratotic papule/nodule; rough surface	Common, plantar, flat, filiform, digit types	Salicylic acid, cryotherapy, immunotherapy
Hand, Foot and Mouth Disease	Coxsackie A virus (Picornaviridae)	Vesicles on hands, feet, oral ulcers, fever	Enanthem in mouth; self-limiting	Supportive: analgesics, hydration
Measles (Rubeola)	Measles virus	Fever, cough, coryza, maculopapular rash	Koplik spots on buccal mucosa; highly contagious	Supportive; Vitamin A
Rubella (German measles)	Rubella virus	Mild fever, lymphadenopathy, maculopapular rash	Post-auricular lymphadenopathy; teratogenic risk	Supportive
Erythema Infectiosum (Fifth disease)	Parvovirus B19	Slapped-cheek appearance; lacy rash on body	Self-limiting; common in children	Supportive
Mpox (Monkeypox)	Mpox virus (Poxviridae)	Fever, lymphadenopathy, vesiculopustular lesions	Deep-seated pustules; umbilication; lymphadenopathy	Supportive; Tecovirimat in severe cases

5. DIAGNOSIS

Mainly clinical

Based on:

- History
- Morphology

Investigations -

Tzanck smear

- Multinucleated giant cells in herpes infection

PCR

- Confirmatory test where required

Serology

- IgM/IgG in selected cases

Viral culture

- Rarely used

Biopsy

- Unusual cases

Diagnostic Flow

History + Morphology
↓
Clinical diagnosis
↓
Tzanck smear / PCR when required
↓
Serology / culture / biopsy in selected cases

6. MANAGEMENT APPROACH

1. Determine the specific virus

Based on:

- Clinical features
- Symptom pattern
- Lesion morphology

2. Symptomatic treatment

- Analgesics
- Antipyretics
- Antihistamines

3. Antiviral drugs

Examples:

- **Acyclovir**
- **Valacyclovir**

4. Local treatment

According to lesion type.

5. Prevent complications

Example:

- Post-herpetic neuralgia

6. Counselling

- Transmission
- Recurrence

7. PREVENTION

- Vaccination:
  - Measles
  - Rubella
  - Varicella
  - Herpes zoster
- Maintain hygiene
- Avoid sharing personal items
- Avoid close contact with active lesions
- Hand hygiene
- Isolation in contagious cases
- Safe sexual practices for HSV and HPV

Treatment	Image
Acyclovir (oral/topical), symptomatic	
Supportive, Acyclovir (in severe/immunocompromised)	
Acyclovir/Valacyclovir, pain control	
Curettage, cryotherapy, topical agents (e.g. KOH, TCA)	
Salicylic acid, cryotherapy, immunotherapy	
Supportive (analgesics, hydration)	
Supportive, Vitamin A	
Supportive	
Supportive	
Supportive, Tecovirimat (in severe cases)	

8. IMPORTANT IDENTIFICATIONS

- **HSV** → recurrent, painful lesions; tingling prodrome
- **VZV** → chickenpox, primary infection
- **Herpes zoster** → shingles; reactivation of VZV
- **Warts** → HPV; different forms including plantar, flat and filiform
- **Molluscum** → central umbilication
- **Measles** → Koplik spots
- **Rubella** → post-auricular lymphadenopathy + pregnancy risk
- **Parvovirus B19** → slapped-cheek appearance + lacy rash
- **Mpox** → lymphadenopathy + deep-seated pustules
- **Most are self-limiting**, but early targeted treatment can prevent complications.

NEET PG PREPARATION -

Disease	One-Liner
HSV	Painful recurrent grouped vesicles
Chickenpox	Generalized lesions in different stages
Herpes zoster	Unilateral dermatomal vesicles
Molluscum	Umbilicated papules
Warts	HPV-induced hyperkeratotic lesions
HFMD	Hand + foot vesicles + oral lesions
Measles	Koplik spots
Rubella	Post-auricular nodes
Fifth disease	Slapped-cheek rash
Mpox	Deep pustules + lymphadenopathy

NOTES

BACTERIAL INFECTIONS

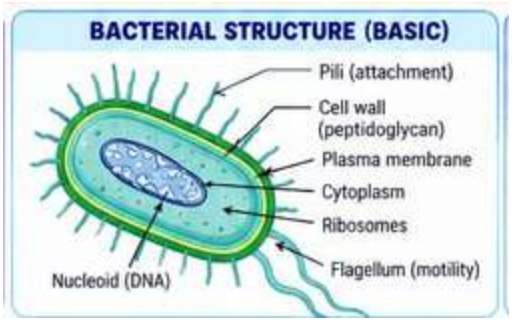
Bacteria - single-celled microorganisms that can cause a wide range of infections.

1. Definition

- Prokaryotic, single-celled organisms
- Can be beneficial, commensal or pathogenic
- Cause infections by:
  - Invasion
  - Toxin production
  - Immune-mediated damage
- Commonly treated with antibiotics
- Rising antimicrobial resistance is a major threat

2. BACTERIAL STRUCTURE

Main components



- **Pili** → attachment
- **Cell wall** → peptidoglycan
- **Plasma membrane**
- **Cytoplasm**
- **Ribosomes**
- **Nucleoid** → DNA
- **Flagellum** → motility

3. CLASSIFICATION BY GRAM STAIN -

Gram Positive (+)	Gram Negative (-)
Thick peptidoglycan cell wall	Thin peptidoglycan + outer membrane
Stains purple	Stains pink
Examples: *Staphylococcus, Streptococcus, Enterococcus, Clostridium*	Examples: *E. coli, Klebsiella, Pseudomonas, Neisseria, Salmonella*
Gram-positive cocci commonly occur in clusters/chains	Includes gram-negative bacilli and diplococci



4.BACTERIAL INFECTIONS - SYSTEM-WISE

System	Common Bacteria	Diseases / Infections
Respiratory	Streptococcus pneumoniae, Haemophilus influenzae, Mycobacterium tuberculosis	Pneumonia, otitis media, sinusitis, tuberculosis
ENT	Streptococcus pyogenes, Staphylococcus aureus	Tonsillitis, pharyngitis, otitis externa/media
Skin & Soft Tissue	Staphylococcus aureus, Streptococcus pyogenes	Cellulitis, impetigo, abscess, necrotizing fasciitis
Gastrointestinal	E. coli, Salmonella, Shigella, Vibrio cholerae, Campylobacter, Helicobacter pylori	Diarrhoea, typhoid fever, cholera, gastritis, peptic ulcer
Urinary Tract	E. coli, Klebsiella, Proteus, Enterococcus	UTI, pyelonephritis
CNS	N. meningitidis, S. pneumoniae, Listeria monocytogenes	Meningitis, encephalitis, brain abscess
Genital / STD	Neisseria gonorrhoeae, Chlamydia trachomatis, Treponema pallidum	Gonorrhoea, chlamydia, syphilis
Systemic	Staphylococcus aureus, E. coli, Streptococcus	Bacteraemia, sepsis, typhoid fever

5. COMMON BACTERIA -

Bacterium	Gram	Key Feature	Common Infection
Staphylococcus aureus	+	Clusters, catalase +, coagulase +	Skin infections, pneumonia, food poisoning, sepsis
Streptococcus pyogenes	+	Chains, β-haemolytic	Pharyngitis, cellulitis, scarlet fever, rheumatic fever
Streptococcus pneumoniae	+	Lancet-shaped, α-haemolytic	Pneumonia, meningitis, otitis media
Enterococcus	+	Bile resistant	UTI, endocarditis
Escherichia coli	−	Lactose fermenter	UTI, diarrhoea, sepsis
Klebsiella pneumoniae	−	Capsulated, non-motile	Pneumonia, UTI
Pseudomonas aeruginosa	−	Non-lactose fermenter, oxidase +	Hospital infections, burn wounds
Salmonella typhi	−	Non-lactose fermenter, motile	Typhoid fever
Neisseria meningitidis	−	Diplococci, oxidase +	Meningitis, meningococcaemia
Clostridium tetani	+	Spore-forming, anaerobe	Tetanus
Mycobacterium tuberculosis	Acid-fast	Acid-fast, slow-growing	Tuberculosis

6. CLINICAL APPROACH -

Flowchart

Patient with suspected bacterial infection
↓
Identify site of infection
(e.g. respiratory, urinary, skin, CNS)
↓
Send appropriate samples for investigation
(Gram stain, culture & sensitivity, others)
↓
Start empirical antibiotics
(based on likely organism + local guidelines)
↓
Modify treatment
(according to culture report & clinical response)
↓
Monitor and prevent complications

7. DIAGNOSIS -

Test	Purpose
Gram stain	Rapid identification of Gram-positive / Gram-negative bacteria
Culture & sensitivity	Definitive diagnosis + antibiotic selection
CBC	Look for leucocytosis
CRP / ESR	Inflammatory markers
Procalcitonin	Useful in systemic bacterial infections
Blood culture	Bacteraemia / sepsis
Urine culture	UTI
Sputum culture	Respiratory infections
Stool culture	Enteric infections
Imaging: X-ray / USG / CT	Identify complications such as abscess or pneumonia

8. TREATMENT PRINCIPLES - ANTIBIOTICS

1. Start empirical therapy early in **severe infections**
2. Choose antibiotic based on:
    - Likely organism
    - Site of infection
3. **De-escalate** according to culture report
4. Use correct:
    - Dose
    - Route
    - Duration
5. Follow local antibiotic guidelines
6. Watch for adverse effects
7. Prevent and manage complications
8. Avoid unnecessary antibiotics to reduce resistance

9. PREVENTION -

- Vaccination:
    - Pneumococcal
    - Hib
    - Typhoid
    - Tetanus
- Hand hygiene
- Safe food and water
- Screening and early treatment
- Rational use of antibiotics
- Awareness and public-health measures

10. COMMON COMPLICATIONS

- Sepsis and septic shock
- Abscess formation
- Chronic infection
- Toxic shock syndrome
- Post-infectious immune complications
    - e.g. rheumatic fever
    - glomerulonephritis
- Antibiotic resistance

11. IMP POINTS -

- Think bacterial infection when there are **fever with focal signs**.
- Always send **samples before antibiotics**, whenever feasible.
- Do not use antibiotics for every viral infection.
- Use antibiotics judiciously to prevent resistance.
- Know the **common organism for each system**.
- Reassess if there is no improvement within **48–72 hours**.
- **Right diagnosis → Right drug → Better outcome**

NEET PG PREAPARATION -

Gram +
→ Purple
→ Thick peptidoglycan
→ Staphylococcus, Streptococcus, Enterococcus, Clostridium

Gram −
→ Pink
→ Thin peptidoglycan + outer membrane
→ E. coli, Klebsiella, Pseudomonas, Neisseria, Salmonella

Best diagnostic principle -
Clinical suspicion → Sample → Gram stain → Culture & sensitivity → Targeted treatment

Golden rule - USE ANTIBIOTICS WISELY → FIGHT INFECTIONS → STOP RESISTANCE

NOTES -

SCABIES

1. FEATURES

Feature	Details
Causative agent	Sarcoptes scabiei var. hominis (female mite)
Type	Ectoparasitic infestation
Size	0.3–0.5 mm
Habitat	Stratum corneum, burrows
Survival	2–3 days off human body
Transmission	Close skin contact, fomites (clothes, bedding)
Incubation period	2–6 weeks; 1–4 days in re-infestation
Prevalence	Worldwide; common in crowded, low socioeconomic settings

2. LIFE CYCLE

Adult female mite burrows in stratum corneum
↓
Lays 2–3 eggs/day
Total: 40–50 eggs
↓
Egg hatches in 3–4 days → Larva
↓
Larva → Nymph
in 7–10 days
↓
Nymph → Adult
Total cycle: 10–14 days

3. MORPHOLOGY



- Oval, dorsoventrally flattened female mite
- Short, stubby legs
- Ventral suckers on **1st and 2nd pairs of legs**
- Dorsal spines and setae
- Smaller male does not burrow
- **Only female mite is responsible for burrows and infestation**

4. CLINICAL FEATURES

Main Symptom

- **Intense itching**
- Usually **worse at night**
- Called **nocturnal pruritus**

Skin Lesions

- Burrows
- Papules
- Vesicles
- Excoriations
- Eczematous lesions

Common Sites

- Finger webs
- Flexor aspect of wrists
- Axillae
- Areola
- Umbilicus
- Genitalia
- Buttocks
- Inner thighs



Spares

- Usually **face and neck in adults**

Infants

- May involve:
    - Scalp
    - Face
    - Palms
    - Soles

Secondary Infection

- Common
- Usually due to **pyoderma**

5. CLASSICAL SIGNS

Sign	Description
Burrow	Thin, grey, wavy raised line, usually 2–10 mm, with a vesicle at one end
Delta-wing jet sign	Dermoscopy shows a triangular mite with lines representing the burrow
Ink test	Ink fills the burrow and helps demonstrate it
Nocturnal pruritus	Very characteristic
Symmetrical distribution	Common
Family members affected	Important diagnostic clue

6. TYPES OF SCABIES

Type	Features
Classic scabies	Common type; pruritic papules and burrows
Crusted scabies / Norwegian scabies	Massive infestation, thick crusts; highly contagious; commonly seen in immunocompromised patients
Nodular scabies	Persistent itchy nodules, especially genitalia and axillae
Scabies in infants	Involves face, scalp, palms and soles
Incognito scabies	Atypical presentation due to steroid use

7. DIFFERENTIAL DIAGNOSIS

Condition	Key Differentiating Features
Atopic dermatitis	Chronic, flexural distribution, history of atopy
Contact dermatitis	History of contact; localized
Insect bites	Grouped, exposed areas
Diabetic pruritus	No burrows; generalized
Prurigo nodularis	Thick nodules
Pediculosis	Lice or nits
Papular urticaria	Acute, history of insect bite
Tinea	Annular lesions; KOH positive

8. INVESTIGATIONS

Usually a clinical diagnosis

Skin scraping

- Demonstrates:
  - Mite
  - Eggs
  - Scybala
- **KOH mount**

Dermoscopy

- Demonstrates **delta-wing jet sign**

Adhesive tape test

- Detects mites/eggs

Ink test

- Helps demonstrate burrows

Investigations are rarely needed in typical cases.

9. MANAGEMENT

Drug / Measure	Dose / Application	Important Point
Permethrin 5% cream	Apply from neck down; leave for 8–12 h; repeat after 7 days	First-line; safe in children and pregnancy
Benzyl benzoate 25%	Apply once daily for 3 days	Irritant; not for infants <2 months
Oral ivermectin 200 µg/kg	Single dose; repeat after 7–14 days	Useful in crusted scabies/out-breaks
Lindane 1%	Avoid	Neurotoxicity; not recom-mended
Crotamiton 10%	Apply for 2 consecutive days	Less effective
Sulfur 5–10%	Apply for 3 days	Safe in neonates
Antihistamines	Symptomatic treatment	Reduces itching
Treat all close con-tacts	Simultaneous treatment	Prevents re-infestation

10. PREVENTION & CONTROL

1. Treat **all household and close contacts**.
2. Wash clothes, bed sheets and towels in **hot water (>60°C)**.
3. Sun-dry or iron non-washable items.
4. Avoid close skin-to-skin contact with infected persons until treated.
5. Maintain personal hygiene.

6. Identify and treat cases in institutions such as hostels and old-age homes.

IMP POINTS -

1. **Intense nocturnal itching** is the hallmark.
2. Look for **burrows**, especially at characteristic sites.
3. **Finger webs** are commonly involved.
4. **Permethrin 5% cream** is the drug of choice in the sheet.
5. **Treat the patient and all close contacts simultaneously.**
6. Prevention of **re-infestation** is essential.

VIVA PURPOSE -

SCABIES = Severe nocturnal itch + Characteristic burrows + All close contacts treated + Bedding/clothes decontaminated

NOTES –

PEDICULOSIS

Definition: Infestation of human skin, hair or clothes by lice (*Pediculus, Pthirus* or *Phthirus*).

Common • Contagious • Treatable

1. TYPES OF PEDICULOSIS

Type	Causative louse	Site	Key feature
Head louse infestation	Pediculus humanus capitis	Scalp hair	Common in children
Body louse infestation	Pediculus humanus corporis	Body, clothes, seams	Associated with poor hygiene, overcrowding
Pubic louse infestation (Phthiriasis)	Pthirus pubis	Pubic hair, also axilla, beard, chest	Crab-like louse, sexually transmitted

2. THE LOUSE AND NIT

Head louse

- *P. h. capitis*
- Size: **2–3 mm**

Body louse

- *P. h. corporis*
- Size: **2.5–3.5 mm**

Pubic louse

- *P. pubis*
- Size: **1–2 mm**
- Crab-like appearance

Nit (Egg)

- Oval
- Approximately **0.8 mm**
- Firmly attached to hair shaft

3. LIFE CYCLE

Egg (Nit)

↓

Hatches in 7–10 days

↓

Nymph

- 3 nymphal stages
- **7–10 days**

↓

Adult louse

- Lives approximately **30–40 days**
- Feeds on human blood several times a day

Total life cycle

Approximately 3–4 weeks

4. MODE OF TRANSMISSION

- Direct **head-to-head contact** is common.
- Sharing:
  - Combs
  - Towels
  - Hats
  - Clothes
  - Bedding
- Overcrowding:
  - Schools
  - Hostels
- Poor personal hygiene, especially in **body louse infestation**
- Sexual contact in **pubic louse infestation**

Key concept

Close contact → spread of lice

5. RISK FACTORS

- Children → especially for head lice
- Overcrowding
- Poor hygiene
- Low socioeconomic status
- Sharing personal items
- Sexually active individuals → pubic lice
- Homelessness / refugee camps → body lice

6. CLINICAL FEATURES

Type	Main symptoms	Signs	Common sites
Head louse	Intense scalp itching, irritability, disturbed sleep	Nits attached to hair shafts, excoriations, papules	Scalp, occipital region and behind ears
Body louse	Itching, trench fever may occur rarely	Lice on clothes, excoriations, eczematous changes	Clothes/seams, body, neck, trunk
Pubic louse	Severe itching; may be asymptomatic	Crab-like lice, nits, bluish macules (**maculae caeruleae**)	Pubic hair, axilla, beard, chest, eyelashes

7. DIAGNOSIS

Clinical examination

- Visualization of **live lice or nits**
- Nits firmly attached to hair shaft, unlike dandruff
- Magnifying lens / dermoscope
- Look for characteristic sites

Important point

Nits do not move; dandruff does.

8. DIFFERENTIAL DIAGNOSIS

Condition	How to differentiate
Dandruff	Loose scales, not attached; no live lice
Seborrhoeic dermatitis	Greasy scales and erythema
Scabies	Burrows and nocturnal itching
Folliculitis	Pustules around hair follicles
Allergic dermatitis	No nits or lice

9. MANAGEMENT

1. Topical Pediculicides — First Line

- **Permethrin 1% lotion**
  - Leave for approximately **10 minutes**
  - Repeat after **7–10 days**
- **Malathion 0.5% lotion**
  - Used in resistant cases
- **Lindane 2%**
  - Second-line
  - Avoid in children **<2 years** and pregnancy

2. Mechanical Removal

- Wet combing with a **fine-toothed nit comb**
- Remove nits manually

3. Treat Contacts

- Examine and treat **family members / close contacts**

4. Environmental Measures

- Wash clothes, bed linen and towels in **hot water ≥60°C**
- Avoid sharing personal items

- **Vacuum furniture** for pubic/body louse infestation

10. PREVENTION



- Do not share:
  - Combs
  - Hats
  - Clothes
  - Towels
- Regular washing of clothes and bed linen in hot water
- Health education in schools
- Avoid close contact with infected persons

11. COMPLICATIONS

- Secondary bacterial infection (**impetigo**)
- Eczematization
- Post-inflammatory hyperpigmentation
- Lymphadenopathy, rarely
- Trench fever in body louse infestation due to **Bartonella quintana**

12. IMP POINTS

1. Pediculosis is a **common, contagious but easily treatable infestation**.
2. Look for **live lice and nits**.
3. **Head lice** are most common in children.
4. **Pubic lice** are sexually transmitted.
5. **Permethrin is the drug of choice** according to the revision sheet.
6. Treat **close contacts** and perform environmental decontamination.
7. Prevention depends on personal hygiene and avoiding sharing personal items.

VIVA PURPOSE -

Identify → Treat → Prevent → Break the cycle

Early recognition → Proper treatment → Break the chain

NOTES –

ACNE VULGARIS

- **Chronic inflammatory disorder** of the pilosebaceous unit.
- Common in **adolescents**, especially around **16–18 years**.
- Affects:
  - Face
  - Chest
  - Back
- **Male > Female** in mild disease.
- **Female > Male** in severe disease.
- **Multifactorial** disorder.
- Usually self-limiting, but may cause:
  - Acne scarring
  - Psychological impact

2. PATHOGENESIS

Main factors

↑ Androgens

↓

↑ Sebum production

+

Follicular hyperkeratinization

+

C. acnes colonization

↓

Microcomedone formation

↓

Obstruction of pilosebaceous unit

↓

Inflammation

(IL-1, TNF-α, etc.) Viva purpose

↓

ACNE LESIONS

Comedones → Inflammatory lesions → Scarring

3.CLINICAL FEATURES

Primary lesions

1. **Open comedone**
    - Blackhead
2. **Closed comedone**
    - Whitehead
3. **Papule**
    - Red, inflammatory lesion
4. **Pustule**
    - Pus-filled inflammatory lesion
5. **Nodule**
    - Deep, firm lesion
6. **Cyst**
    - Deep, painful lesion

4. RISK FACTORS / AGGRAVATING FACTORS

- Hormonal changes:
    - Puberty
    - PCOS
- Genetic predisposition
- Oily skin / high sebum production
- High glycaemic diet
- Drugs:
    - Steroids
    - Lithium
    - Phenytoin
    - Isoniazid
- Stress
- Heat and humidity
- Occlusive cosmetics
- Occupational exposure:
    - Oils
    - Chemicals

5. TYPES OF LESIONS

Type	Features
Comedones	Open = blackhead; Closed = whitehead
Inflammatory	Papules = red, tender; Pustules = pus-filled
Severe	Nodules = deep, firm; Cysts = painful, may scar

6. DISTRIBUTION

Common sites

Face

- Forehead
- Cheeks
- Nose
- Chin

Chest

Back

- Particularly upper back

7. GRADING

Grade	Lesions
Mild	Comedones, few papules/pustules
Moderate	Multiple papules/pustules + nodules
Severe	Nodules, cysts, extensive involvement

8. DIFFERENTIAL DIAGNOSIS

- Rosacea
- Folliculitis
- Steroid-induced eruption
- Acneiform drug eruption
- Hidradenitis suppurativa

9. HISTORY TAKING

Point	What to ask
Onset & duration	Age, course, progression
Site & type of lesions	Comedones, papules, pustules, nodules
Symptoms	Pain, itching
Aggravating factors	Menstruation, stress, diet, cosmetics, drugs
Past treatment	Response and duration
Menstrual history	Irregular cycles, hirsutism, PCOS
Drug history	Steroids, antiepileptics etc.
Family history	Acne, seborrhoea
Psychological impact	Self-esteem, social withdrawal

10. MANAGEMENT

Step 1: Assess severity

Mild → Moderate → Severe

Treatment approach

Mild

→ Topical therapy ± oral therapy

Moderate

→ Combined topical + oral therapy

Severe

→ Oral isotretinoin ± hormonal therapy

Important

Always give advice on skincare, diet and follow-up.

11. TREATMENT OPTIONS

Severity	Topical	Oral	Other / Notes
Mild	Benzoyl peroxide, topical retinoids, topical antibiotics	Usually not required	Add BPO if resistant
Moderate	Topical retinoid + BPO ± topical antibiotic	Oral antibiotics such as doxycycline, minocycline	Consider hormonal therapy in females
Severe	Adjunct topical therapy	**Isotretinoin**	Consider hormonal therapy such as OCPs/anti-androgens

12. GENERAL MEASURES

- Use a **gentle cleanser**.
- Use **non-comedogenic products**.
- Avoid picking or squeezing lesions.
- Use oil-free, non-comedogenic cosmetics.
- Maintain a balanced diet with a **low glycaemic load**.
- Manage stress.
- Regular follow-up.
- Counsel about realistic expectations.

13. SPECIAL SITUATIONS

Situation	Management / Consideration
Females with hormonal acne	OCP, anti-androgens such as spironolactone
PCOS	Endocrine evaluation + hormonal management
Pregnancy	Avoid oral retinoids and tetracyclines; use topical BPO, azelaic acid
Scarring	Early control; procedural therapy such as chemical peels/lasers
Psychological impact	Counselling; psychiatric support may be required

14. COMPLICATIONS

- **Post-acne scarring**
  - Atrophic scars
  - Hypertrophic scars
- **Post-inflammatory hyperpigmentation**
- **Psychological distress**
- Rare:
  - **Acne fulminans**

15. VIVA PURPOSE

1. Acne vulgaris is a **multifactorial disease of the pilosebaceous unit**.
2. **Androgens** are an important trigger.
3. Major pathogenic factors:
    - Increased sebum
    - Follicular hyperkeratinization
    - *C. acnes* colonization
    - Inflammation
4. Lesions include **comedones, papules, pustules, nodules and cysts**.
5. Assess **severity and psychological impact**.
6. Individualized treatment is important.
7. **Early treatment helps prevent scarring**.
8. Patient education and follow-up are essential.

16. REMEMBER – 5A

A – Acne: Common disorder
A – Androgens: Major trigger
A – All types: Comedones → inflammatory lesions → severe lesions
A – Assess: Severity and impact
A – Adequate treatment: Follow-up is essential

Quick sequence -

Androgens → Sebum ↑ → Hyperkeratinization → Microcomedone → Obstruction → Inflammation → Acne → Scarring

NOTES –

LEPROSY (HANSEN'S DISEASE)

- **Causative organism:** Mycobacterium leprae (acid-fast bacillus)
- **Incubation period:** 2–5 years
  - Range: 6 months–20 years
- **Mode of spread:** Prolonged close contact, mainly through respiratory droplets
- **Mainly affects:**
  - Skin
  - Peripheral nerves
  - Upper respiratory tract
  - Eyes
  - Testes
- **Treatment:** Multidrug therapy (MDT), free under NLEP
- **Important:** Early diagnosis prevents disability.

2. RISK FACTORS

- Close contact with untreated patient
- Overcrowding
- Poor socioeconomic status
- Malnutrition
- Genetic susceptibility
- Low immunity, e.g. HIV, steroids

3. PATHOGENESIS

Inhalation of M. leprae

↓

Multiplication in macrophages

- Skin
- Nerves
- Nasal mucosa

↓

Cell-mediated immunity (CMI)
determines the clinical type

↓

Immune response causes nerve damage and tissue injury

↓

Deformities if untreated

4. CLINICAL SPECTRUM

Ridley–Jopling Classification

TT → BT → BB → BL → LL

Type	CMI	Skin lesions	Nerve involvement	Smear
TT Tuberculoid	Strong	Few, well-defined, hypopigmented anesthetic lesions	Asymmetric, few nerves	Negative
BT Borderline tuberculoid	Good	More lesions, less defined	Asymmetric, more nerves	Usually negative
BB Mid-borderline	Moderate	Multiple, ill-defined	Symmetric	Variable
BL Borderline lepromatous	Poor	Numerous, symmetric	Symmetric, multiple nerves	Positive
LL Lepromatous	Very poor	Diffuse, numerous, non-anaesthetic nodules/infiltration	Symmetric, many nerves	Strongly positive

- **TT:** Dry, scaly, early nerve damage
- **BT:** Between TT and BB
- **BB:** Unstable type
- **BL:** Between BB and LL
- **LL:** Facies leontina, madarosis, high infectivity

5. CLINICAL FEATURES

TT

- Hypopigmented patch
- Loss of sensation
- Few lesions
- Early nerve involvement

BT / BB

- More numerous lesions
- Less well-defined lesions
- Variable nerve involvement

BL

- Numerous symmetric lesions
- Multiple nerve involvement
- Positive smear

LL

- Diffuse infiltration
- Numerous nodules
- Symmetrical nerve involvement
- **Facies leontina**
- **Madarosis**

6. IMPORTANT SIGNS

Skin

- Hypopigmented or reddish skin lesions
- **Loss of sensation** is an important clue

Nerves

- Thickened peripheral nerves

Sensory loss

- Loss of:
  - Touch
  - Pain
  - Temperature

Motor involvement

- Weakness
- Deformities
- Claw hand
- Foot drop

Facial changes

- Loss of eyebrows (**madarosis**)

7. NERVE PALPATION SITES

Important peripheral nerves to examine:

1. **Greater auricular nerve**
2. **Ulnar nerve**
3. **Radial cutaneous nerve**
4. **Common peroneal nerve**
5. **Posterior tibial nerve**

Palpate for:

- Thickening
- Tenderness
- Pain

8. CLASSICAL CLINICAL FEATURES

Skin lesions

- Hypopigmented patch with sensory loss
- Erythematous plaques
- Multiple nodules
- Infiltration of ear lobes

Nerve manifestations

- Thickened nerves
- Sensory loss
- Motor weakness
- Claw hand
- Foot drop

Other manifestations

- Trophic ulcers
- Eye involvement
- Testicular involvement

9. DIAGNOSIS

Clinical features

- Characteristic skin lesions
- Sensory loss
- Thickened peripheral nerves

Investigations

- **Skin smear for AFB**
- **Skin biopsy**, when needed
- **Nerve function assessment**
    - Sensory
    - Motor
- Classification as **PB or MB**

10. WHO CLASSIFICATION

Paucibacillary (PB)

- **1–5 skin lesions**
- Smear negative

Multibacillary (MB)

- **>5 skin lesions**
- Smear positive

11. DIFFERENTIAL DIAGNOSIS

- Vitiligo
- Pityriasis versicolor
- Tinea corporis
- Post-inflammatory hyperpigmentation
- Psoriasis
- Lichen planus
- Secondary syphilis

12. LEPROSY REACTIONS

Type 1 Reaction — Reversal Reaction

- Inflammation in existing lesions
- Nerve pain
- Tender nerves
- Fever
- Tender nodules
- Arthritis
- Neuritis
- Seen particularly in **BT/BL**

Treatment

- Requires prompt treatment
- **Steroids** may be used for neuritis.

13. TREATMENT

NLEP / WHO MDT

Category	Regimen in adults	Duration
Paucibacillary (PB)	Rifampicin 600 mg supervised monthly + Dapsone 100 mg daily	**6 months**
Multibacillary (MB)	Rifampicin 600 mg monthly + Clofazimine 300 mg monthly + Clofazimine 50 mg daily + Dapsone 100 mg daily	**12 months**

- Free of cost under **National Leprosy Elimination Programme (NLEP), India**
- Ensure adherence and regular follow-up.
- Treat reactions appropriately.
- Physiotherapy, self-care and reconstructive surgery may be required.

14. MANAGEMENT OF DISABILITY

- Early diagnosis and treatment
- Regular nerve function assessment
- Protective footwear and self-care
- Physiotherapy and occupational therapy
- Reconstructive surgery when required
- Counselling and social rehabilitation

15. PREVENTION & CONTROL

- Early detection and complete treatment
- Contact examination and prophylaxis
- **Single-dose rifampicin** for eligible contacts
- **BCG vaccination** for partial protection
- Health education and removal of stigma
- Improve living conditions
- Community-based surveillance

16. VIVA IMP POINTS

1. **Leprosy is caused by Mycobacterium leprae.**
2. Think of leprosy when a skin lesion has **loss of sensation**.
3. **Thickened peripheral nerves** are an important diagnostic clue.
4. Always assess **sensory and motor nerve function**.
5. **TT → BT → BB → BL → LL** represents the Ridley–Jopling spectrum.
6. **PB:** 1–5 lesions.
7. **MB:** >5 lesions.
8. Early diagnosis and MDT prevent deformities.
9. Leprosy reactions require **prompt recognition and treatment**.
10. **Treat the patient, not the stigma.**

LEPROSY = Lesion + Loss of sensation + Enlarged nerves + Peripheral nerve damage + Ophthalmic/systemic involvement + Skin smear

VITILIGO

1. DEFINITION

Vitiligo is an acquired, chronic depigmenting disorder characterized by well-demarcated, milky-white macules and patches due to loss or dysfunction of functional melanocytes.

Features -

- Acquired, chronic condition
- Loss of functional melanocytes
- Well-demarcated depigmented macules/patches
- No primary scale or atrophy
- Sensation is normal
- May have **leukotrichia** (white hair)
- Important cosmetic and psychological impact

2. PATHOGENESIS

Genetic predisposition
+
Environmental triggers
↓
Immune-mediated destruction of melanocytes
(autoreactive T cells)
↓
Loss of functional melanocytes in epidermis
↓
Depigmented macules and patches

3. CLINICAL APPEARANCE

Common patterns

- **Acral vitiligo**
  - Hands and feet
- **Non-segmental vitiligo**
  - Trunk and other areas
- **Facial vitiligo**



Typical lesion features

Colour

- Milky-white / chalk-white

Border

- Well-demarcated

Surface

- Normal
- No scale

Atrophy

- Absent

Sensation

- Normal

Hair

- May become white
- **Leukotrichia**

Symptoms

- Usually asymptomatic

4. CASE TAKING – SYSTEMATIC APPROACH

I – Identification

Ask about:

- Age
- Sex
- Occupation
- Residence

C – Chief complaints

- White patches
- Duration
- Progression

H – History of present illness

Ask about:

- Onset
- First site
- Number of lesions
- Spread
- Stable or progressive disease

T – Triggers

- Trauma
- Stress
- Sunburn
- Chemical exposure
- Koebner phenomenon

P – Past history

- Previous episodes
- Previous treatment

D – Drug history

- New medications
- Topical therapy
- Systemic therapy

F – Family history

- Vitiligo
- Autoimmune diseases

S – Personal history

- Comorbid autoimmune disease
- Lifestyle
- Cosmetics

O – Others

- Psychological impact
- Quality of life

5. TYPES / CLINICAL PATTERNS

A. NON-SEGMENTAL VITILIGO

Most common type

Features:

- Usually bilateral
- Symmetrical
- Generalized
- Acral involvement
- Focal involvement
- Mucosal involvement
- Universal type is rare

B. SEGMENTAL VITILIGO

Features:

- Usually unilateral
- Dermatomal pattern
- Early onset
- Often becomes stable
- Less responsive to treatment

comparison

Non-segmental > Segmental

6. DISTRIBUTION – COMMON SITES

Common sites include -

- Face
- Especially periorificial areas
- Hands
- Fingers
- Knuckles
- Feet
- Toes
- Elbows
- Knees
- Genital area



- Groin
- Flexural areas
- Lips and oral mucosa
- Scalp
- Can produce leukotrichia

Often symmetrical, except in segmental vitiligo.

7. ASSOCIATED CONDITIONS

Vitiligo is associated with autoimmune disorders.

Common associations

- **Thyroid disease**
  - Most common association
- Type 1 diabetes mellitus
- Pernicious anaemia
- Alopecia areata
- Addison disease

Clinical approach

Think AUTOIMMUNE → Ask and screen when indicated.

8. DIFFERENTIAL DIAGNOSIS

Pityriasis alba

- Hypopigmented
- Ill-defined
- Usually in children

Tinea versicolor

- Fine scale
- Trunk involvement
- KOH positive

Nevus depigmentosus

- Stable
- Congenital or early onset

Post-inflammatory hypopigmentation

- History of preceding inflammation

Chemical leukoderma

- History of chemical exposure

Idiopathic guttate hypomelanosis

- Multiple small macules
- Usually sun-exposed areas

Leprosy

- Hypopigmented patch
- **Loss of sensation**
- May have nerve involvement

9. WOOD'S LAMP



Under Wood's lamp:

- Depigmented areas appear **bright blue-white**
- Helps detect subtle lesions
- Helps determine extent of lesions
- Useful for assessing activity

10. INVESTIGATIONS

Wood's lamp

- Demonstrates depigmentation

Thyroid function tests

- If clinically indicated

Blood glucose

- If indicated

Other autoimmune tests

- Based on history and examination

Skin biopsy

- Rarely required
- Consider when diagnosis is uncertain

Diagnosis is mainly clinical…..

11. ACTIVITY & EXTENT ASSESSMENT

Assess -

- Number of lesions
- Body surface area (BSA)
- New lesions or recent spread
- Koebner phenomenon
- Leukotrichia
- **Vitiligo Area Scoring Index (VASI)**

Remember

VASI = Extent + Activity assessment

12. MANAGEMENT

Education & Counselling

- Explain chronic nature
- Provide cosmetic support
- Address psychological impact

Sun Protection

- Avoid sunburn
- Use sunscreen

Topical Therapy

- Corticosteroids
- Calcineurin inhibitors

Phototherapy

- **Narrowband UVB**
- Especially useful in non-segmental vitiligo

Systemic Therapy

- Selected cases
- JAK inhibitors may be considered where appropriate and based on current evidence

Surgical Options

Consider in:

- Stable disease
- Refractory disease
- Selected cases

Examples:

- Melanocyte transplantation

13. CASE TAKING / PRESENTATION FLOW

History
↓
Examination
↓
Investigations
↓
Diagnosis
↓
Assess extent & activity
↓
Management plan

REVISION POINTS -

VITILIGO -

Acquired + Chronic + Depigmented patches + Normal sensation + Autoimmune association

Important examination points

1. **Milky-white, well-demarcated patches**
2. **No scale or atrophy**
3. **Sensation remains normal**
4. **Leukotrichia may occur**
5. Examine common sites and mucosa
6. Look for **Koebner phenomenon**
7. Check for autoimmune associations when indicated
8. Wood's lamp helps identify subtle lesions
9. Assess extent using **BSA/VASI**
10. Treatment depends on **type, activity, extent and stability**.

NOTES –

PSORIASIS

Psoriasis is a -

- Chronic
- Immune-mediated
- Inflammatory
- Hyperproliferative disorder

It primarily affects the:

- Skin
- Nails
- Joints

Psoriasis is non-contagious.

2. EPIDEMIOLOGY

- Prevalence: approximately **1–3%**
- Common age groups:
  - **20–30 years**
  - **50–60 years**
- **Male > Female**
- Family history present in approximately **20–30%**

3. ETIOLOGY & PATHOGENESIS

Major factors

1. **Genetic predisposition**
   - Especially **HLA-Cw6**
2. **Immune dysregulation**
   - Mainly Th1 and Th17 cytokines
3. **Keratinocyte hyperproliferation**
4. **Environmental triggers**
   - Infections
   - Stress
   - Trauma
   - Other triggers in susceptible individuals

Histopathological changes

- Parakeratosis
- Acanthosis
- Elongated rete ridges
- Dilated capillaries

4. CLINICAL FEATURES OF CLASSIC PSORIASIS

- **Well-defined erythematous plaques**
- Covered with **silvery-white scales**
- Sharply demarcated margins
- Symmetrical distribution
- Extensor surface predominance
- Chronic relapsing course
- Itching may be variable

Classic appearance

Erythematous plaque + Silvery scale + Well-defined margin = Psoriasis

5. SPECIAL SIGNS



Auspitz Sign

- **Pinpoint bleeding** occurs after removal of scale.

Koebner Phenomenon

- New psoriatic lesions develop at sites of **trauma**.

Candle-Grease Sign

- Scraping the lesion produces a **candle-wax appearance**.

6. TYPES OF PSORIASIS

1. Plaque Psoriasis

- Most common type
- Approximately **80–90%**
- Well-defined plaques

2. Guttate Psoriasis

- Drop-like lesions
- Often follows **streptococcal throat infection**

3. Inverse Psoriasis

- Smooth, shiny lesions
- Occur in skin folds

4. Pustular Psoriasis

- Pus-filled lesions
- May be localized or generalized

5. Erythrodermic Psoriasis

- Generalized erythema and scaling
- Involves **>90% body surface area**

6. Palmoplantar Psoriasis

- Hyperkeratotic plaques
- Palms and soles

7. Scalp Psoriasis

- Well-defined scaly plaques
- May resemble dandruff

8. Nail Psoriasis

- Pitting
- Onycholysis
- Subungual hyperkeratosis

7. COMMON SITES



Important sites include:

- Scalp
- Elbows
- Umbilicus
- Gluteal cleft
- Lumbosacral region
- Extensor surfaces
- Palms
- Soles
- Nails

Typical distribution

Scalp + Elbows + Knees + Lumbosacral region + Gluteal cleft

8. NAIL CHANGES



Common nail manifestations:

- **Pitting**
- **Onycholysis**
- **Subungual hyperkeratosis**
- **Oil-drop / salmon-patch sign**

9. JOINT INVOLVEMENT

Psoriatic Arthritis

Features include:

- Asymmetric oligoarthritis
- DIP joint involvement
- **Dactylitis**
  - Sausage digits
- Enthesitis
- Spondylitis

Important

Psoriasis + joint symptoms → assess for psoriatic arthritis.

10. TRIGGER FACTORS

- Infections
  - Especially streptococcal throat infection
- Stress
- Trauma
- Injury
- Surgery
  - Koebner phenomenon
- Drugs:
  - Lithium
  - Beta-blockers
  - Antimalarials
  - NSAIDs
  - ACE inhibitors
  - Steroids withdrawal
- Smoking
- Alcohol
- Climate
  - Cold
  - Dry weather

11. PATHOGENESIS

Genetic susceptibility

- HLA-Cw6

↓

Environmental triggers

- Infection
- Stress
- Trauma

↓

Immune activation

- Th1
- Th17 cells

↓

Cytokine release

- TNF-α
- IL-17
- IL-23

↓

Keratinocyte hyperproliferation + inflammation

↓

Psoriatic lesions

12. DIFFERENTIAL DIAGNOSIS

Seborrhoeic dermatitis

- Greasy scale
- Ill-defined
- Scalp/face commonly involved

Tinea corporis

- Annular lesion
- Active advancing edge

Lichen planus

- Violaceous, flat-topped papules
- Wickham striae

Pityriasis rosea

- Herald patch
- Christmas-tree distribution

Secondary syphilis

- Generalized rash
- Palms and soles involvement
- Other systemic signs

Atopic dermatitis

- Flexural distribution
- Intense itching
- Dry skin

13. INVESTIGATIONS

Psoriasis is primarily a **clinical diagnosis**.

Investigations may be performed when required:

- CBC
- Liver function tests
- Renal function tests
- FBS / HbA1c

- Lipid profile
- ESR / CRP
- Hepatitis screening before systemic/biologic therapy
- Skin biopsy if diagnosis is doubtful

14. SEVERITY ASSESSMENT

PASI

Psoriasis Area and Severity Index

- Standard severity assessment tool

BSA

Body Surface Area

- Assesses percentage of body involvement

DLQI

Dermatology Life Quality Index

- Measures quality-of-life impact

Joint assessment

- Assess number and severity of joint symptoms/comorbidities.

15. MANAGEMENT OVERVIEW

A. General Measures

- Reassurance
- Explain chronic relapsing nature
- Avoid known triggers
- Regular emollient use
- Treat associated conditions:
    - Obesity
    - Diabetes mellitus
    - Hypertension
    - Dyslipidaemia
    - Depression

16. TOPICAL THERAPY

Emollients

- Basic supportive treatment

Topical corticosteroids

- Reduce inflammation

Vitamin D analogues

- Example: **Calcipotriol**

Tazarotene

- Topical retinoid

Calcineurin inhibitors

- Useful particularly at sensitive sites

Keratolytics

- Salicylic acid
- Urea

17. PHOTOTHERAPY

Narrow-band UVB

- **311–313 nm**

PUVA therapy

- Psoralen + UVA

18. SYSTEMIC THERAPY

Conventional systemic drugs

- **Methotrexate**
- **Cyclosporine**
- **Acitretin**
- **Apremilast**

Biological agents

Used particularly in **moderate-to-severe psoriasis**.

Examples:

Anti-TNF agents

- Etanercept
- Adalimumab

Anti-IL-17 agents

- Secukinumab

Anti-IL-23 agents

- Ustekinumab

19. PROGNOSIS

- Chronic relapsing condition
- Symptoms can be controlled but may recur
- Early treatment improves quality of life
- Poor control can cause permanent damage, particularly with joint involvement

20. COMPLICATIONS

Cutaneous

- Secondary infection
- Fissuring
- Erythroderma

Associated comorbidities

- Psoriatic arthritis
- Metabolic syndrome
- Diabetes mellitus
- Hypertension
- Dyslipidaemia
- Cardiovascular disease
- Depression and anxiety
- Non-alcoholic fatty liver disease

21. IMP - POINTS

1. **Silvery scale + well-defined plaque = psoriasis**
2. **Auspitz sign** = pinpoint bleeding after scale removal.
3. **Koebner phenomenon** = lesions at sites of trauma.
4. Always examine:
    - Scalp
    - Nails
    - Joints
5. Assess severity using:
    - **PASI**
    - **BSA**
    - **DLQI**
6. Treat according to severity and disease impact.
7. Look for associated metabolic and cardiovascular comorbidities.
8. Psoriasis is **non-contagious**.

MNEMONIC – PSORIASIS -

P – Plaques
S – Silvery scale
O – Olecranon/extensor sites
R – Recurrent & relapsing
I – Itching variable
A – Auspitz sign
S – Scalp & nails
I – Inflammatory arthritis
S – Severity assessment

FLOWCHART FOR THEORY -

Genetic predisposition
↓
Environmental trigger
↓
Th1 / Th17 activation
↓
TNF-α + IL-17 + IL-23
↓
Keratinocyte hyperproliferation
↓
Erythematous plaque + silvery scale
↓
Assess PASI / BSA / DLQI + joints
↓
Topical therapy / Phototherapy / Systemic therapy

NOTES -

SYPHILIS

- Causative agent: Treponema pallidum (subspecies pallidum)
- Type: Spirochaete (thin, delicate)
- Transmission:
    - Sexual
    - Transplacental
    - Blood transfusion (rare)
- Incubation period: Approximately 3 weeks (10–90 days)
- Chancre: Painless, indurated ulcer
- Important: Can mimic many diseases. Hence, syphilis is called "The Great Imitator."

2. MODES OF TRANSMISSION

1. Sexual contact
    - Vaginal, anal or oral
    - Most common route
2. Transplacental transmission
    - Congenital syphilis
3. Blood transfusion
    - Rare
4. Direct inoculation
    - Accidental laboratory exposure

3. RISK FACTORS

- Multiple sexual partners
- Unprotected sex
- History of other sexually transmitted infections (HIV, HSV)
- Men who have sex with men (MSM)
- Commercial sex work
- Substance abuse
- Low socioeconomic status

4. CLINICAL STAGES AND FEATURES

A. Primary Syphilis

Occurs approximately 3–6 weeks after infection.

- Chancre: Single, painless, indurated ulcer with a clean base.
- Usually genital:
  - Penis
  - Vulva
  - Cervix
  - Anal or oral region
- Non-tender regional lymphadenopathy.
- Heals spontaneously in approximately 3–6 weeks, even without treatment.

B. Secondary Syphilis

Occurs approximately 6 weeks to 6 months after infection.

- Generalized, non-itchy maculopapular rash, including palms and soles.
- Mucous patches in the oral cavity.
- Condyloma lata: Moist, greyish-white lesions in moist areas.
- Generalized lymphadenopathy.
- Constitutional symptoms:
  - Fever
  - Malaise
  - Sore throat
- Alopecia: Moth-eaten pattern.

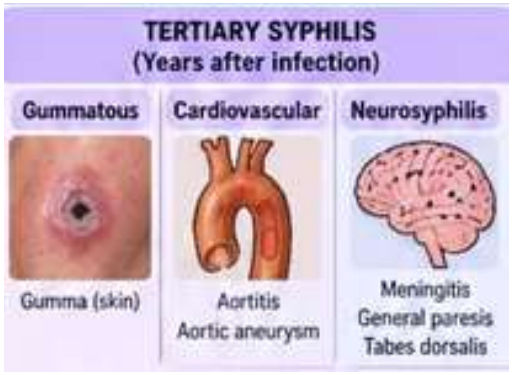


C. Latent Syphilis

Asymptomatic stage.

- No clinical signs or symptoms.
- Detected only by serology.
- Early latent: Less than 1 year.
- Late latent: 1 year or more.
- May last for years.
- Still infectious in the early latent stage.

D. Tertiary Syphilis



Occurs years after infection.

1. Gummatous syphilis

- Gummas involving:

- Skin
- Bone
- Liver

2. Cardiovascular syphilis

- Aortitis
- Aortic aneurysm
- Aortic regurgitation (AR)

3. Neurosyphilis

- Meningitis
- General paresis
- Tabes dorsalis

5. CONGENITAL SYPHILIS

Transmission: Transplacental, at any stage of maternal infection.

Early congenital syphilis

- Snuffles
- Rash
- Hepatosplenomegaly
- Osteochondritis
- Pseudoparalysis

Late congenital syphilis

- Hutchinson's triad:
    1. Interstitial keratitis
    2. Sensorineural deafness
    3. Hutchinson teeth
- Frontal bossing
- Saber shins

6. LABORATORY DIAGNOSIS

A. Direct Detection

- Dark-field microscopy
- DFA-TP (direct fluorescent antibody test)
- PCR (NAAT), where available

B. Serological Tests

1. Non-treponemal tests

Used for screening and monitoring treatment response.

- VDRL
- RPR

Uses:

- Screening
- Monitoring treatment response
- Detecting possible reinfection

2. Treponemal tests

Used to confirm infection.

- TPHA / TPPA
- FTA-ABS
- EIA
- CIA

Important point

Use both non-treponemal and treponemal tests for diagnosis.

A negative non-treponemal test does not completely exclude very early or late syphilis.

7. DIFFERENTIAL DIAGNOSIS

Primary syphilis

- Chancroid
- Herpes genitalis
- Lymphogranuloma venereum (LGV)
- Carcinoma

Secondary syphilis

- Drug rash
- Viral exanthems
- Pityriasis rosea
- Psoriasis

Latent syphilis

- Other asymptomatic infections

Tertiary syphilis

- TB
- Leprosy
- Fungal infections
- Sarcoidosis
- Vasculitis

8. TREATMENT

Stage	Treatment of choice shown in the sheet
Primary, secondary and early latent	Benzathine penicillin G 2.4 million units IM, single dose
Late latent	Benzathine penicillin G 2.4 million units IM, weekly for 3 doses
Tertiary without neurosyphilis	Benzathine penicillin G 2.4 million units IM, weekly for 3 doses
Neurosyphilis	Aqueous crystalline penicillin G, 18–24 million units/day IV for 10–14 days
Pregnancy	Penicillin according to stage; penicillin is essential in pregnancy
Penicillin allergy (non-pregnant)	Doxycycline 100 mg orally twice daily for 14–28 days, according to stage

Important: Pregnant patients with syphilis should receive penicillin. If allergic, desensitization followed by penicillin treatment is generally required. Neurosyphilis requires a specific regimen and specialist assessment.

9. FOLLOW-UP

- Assess clinical improvement.
- Repeat RPR/VDRL titres.
- Follow-up intervals shown in the sheet:
    - Primary and secondary: 4–6, 12 months
    - Monitor at 3, 6 and 12 months for late latent disease, as appropriate
- Persistent high titres may indicate:
    - Treatment failure
    - Reinfection

10. PARTNER MANAGEMENT

- Screen and treat sexual partners.
- Treat partners when indicated, even if asymptomatic.
- Advise safer sexual practices.
- Test for other sexually transmitted infections, including HIV.

11. PREVENTION

- Use condoms to reduce transmission risk.
- Limit the number of sexual partners.
- Screen high-risk groups.
- Routine antenatal screening (VDRL/RPR).
- Screen blood donors.
- Provide health education.
- Ensure early diagnosis and complete treatment.

12. COMPLICATIONS

- Neurosyphilis
- Cardiovascular syphilis
- Gummatous disease
- Ocular syphilis:
    - Uveitis
    - Retinitis
- Otosyphilis:
    - Hearing loss
- Congenital syphilis
- Jarisch–Herxheimer reaction after treatment

13. APPROACH TO A SUSPECTED CASE



14. MNEMONICS

Syphilis stages: P-S-L-T

- P: Primary
- S: Secondary
- L: Latent
- T: Tertiary

Hutchinson's triad

- I: Interstitial keratitis
- D: Deafness
- D: Dental abnormalities (Hutchinson teeth)

Modes of transmission: S-T-B-D

- S: Sexual
- T: Transplacental
- B: Blood
- D: Direct inoculation

RPR use

R = Response monitoring

RPR titres help monitor treatment response.

Great imitator

“Many faces, one disease.”

1. Syphilis is a systemic infection with varied clinical presentations.
2. Primary chancre is classically painless and heals spontaneously.
3. Secondary syphilis commonly involves the palms and soles.
4. Use both non-treponemal and treponemal tests for diagnosis.
5. Penicillin is the treatment of choice, including during pregnancy.
6. Screen and manage sexual partners.
7. Early diagnosis and treatment prevent serious complications.

NOTES -

NON-SYPHILIS

Non-syphilitic Sexually Transmitted Infections and Syphilis Mimickers

Ulcer, Discharge, Warts, Inguinal Lymphadenopathy, No Treponema cause.

- Non-syphilitic STIs include sexually transmitted infections and other conditions causing genital ulcers, urethral/vaginal discharge, or genital warts that are not due to Treponema pallidum.
- Important to differentiate them from syphilis.
- Common agents:
    - HSV
    - Haemophilus ducreyi (chancroid)
    - Klebsiella granulomatis (donovanosis)
    - Non-gonococcal urethritis (Chlamydia, Mycoplasma)
    - Gonorrhoea
    - Trichomoniasis
    - Genital warts (HPV)
- Management includes specific antimicrobial therapy, partner treatment and prevention.

2. CLASSIFICATION OF NON-SYPHILIS CONDITIONS

A. Genital ulcer diseases

- HSV
- Chancroid
- LGV
- Donovanosis

B. Urethral discharge (male)

- Gonorrhoea
- Chlamydia
- Mycoplasma
- Ureaplasma
- Trichomoniasis

C. Cervical/vaginal discharge (female)

- Chlamydia
- Gonorrhoea
- Trichomoniasis
- Bacterial vaginosis
- Candidiasis

D. Genital warts

- Human papillomavirus (HPV)

E. Other syphilis mimickers

- Fixed drug eruption
- Behçet disease
- Lipschütz ulcer

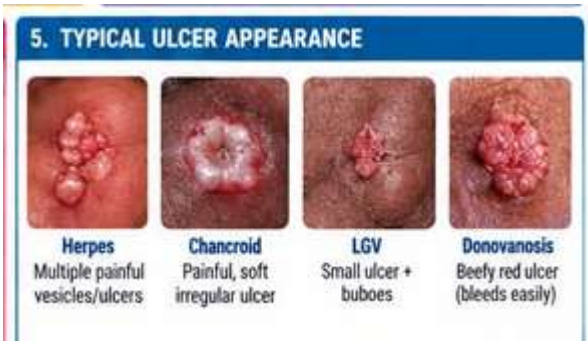
3. IMPORTANT DIFFERENCES: SYPHILIS VS NON-SYPHILIS

Feature	Syphilis	Non-syphilis
Causative agent	Treponema pallidum	Various bacteria, viruses and protozoa
Ulcer	Painless, indurated, clean base	Painful (usually), soft, irregular
Lymph nodes	Non-tender, rubbery	Tender (usually)
Serology	VDRL/RPR, TPHA positive	Treponemal tests negative

4. GENITAL ULCER DISEASES (NON-SYPHILIS)

Condition	Causative agent	Clinical features	Lymph nodes	Diagnosis	Treatment shown in sheet
Genital herpes (HSV)	Herpes simplex virus (HSV-2 > HSV-1)	Multiple, painful vesicles → ulcers; recurrent	Clinical, PCR	Clinical, PCR	Acyclovir
Chancroid	Haemophilus ducreyi	Painful, soft, irregular ulcer with foul-smelling discharge	Tender, suppurative	Clinical, culture	Azithromycin or ceftriaxone
LGV	Chlamydia trachomatis (L1, L2, L3)	Small transient ulcer → painful inguinal nodes (buboes)	Tender, buboes	NAAT	Doxycycline (21 days)
Donovanosis (granuloma inguinale)	Klebsiella granulomatis	Painless, beefy-red ulcers; bleed easily	Usually non-tender	Tissue smear (Donovan bodies)	Azithromycin or doxycycline (prolonged)

5. TYPICAL ULCER APPEARANCE



Genital herpes

- Multiple painful vesicles/ulcers

Chancroid

- Painful, soft, irregular ulcer

LGV

- Small ulcer with painful inguinal lymphadenopathy

Donovanosis

- Beefy-red ulcer
- Bleeds easily

6. URETHRAL DISCHARGE (MALE)

Condition	Causative agent	Features	Diagnosis	Treatment shown in sheet
Gonorrhoea	Neisseria gonorrhoeae	Purulent, profuse discharge; dysuria	Gram stain (Gram-negative diplococci), NAAT	Ceftriaxone ± azithromycin
Non-gonococcal urethritis (NGU)	Chlamydia trachomatis	Mucopurulent, mild discharge	NAAT	Doxycycline
Mycoplasma genitalium	M. genitalium	Persistent/recurrent symptoms	NAAT	Doxycycline followed by moxifloxacin, as indicated
Trichomoniasis	Trichomonas vaginalis	Scanty discharge, pruritus	Wet mount / NAAT	Metronidazole

Note: Treatment should follow current STI guidelines and local antimicrobial resistance recommendations.

7. VAGINAL DISCHARGE (FEMALE)

Condition	Key features	Diagnosis	Treatment shown in sheet
Chlamydia	Mucopurulent discharge, cervicitis	NAAT	Doxycycline
Gonorrhoea	Purulent discharge, cervicitis	NAAT	Ceftriaxone
Trichomoniasis	Frothy, yellow-green, fishy-smelling discharge	Wet mount / NAAT	Metronidazole
Bacterial vaginosis	Thin, grey-white, fishy odour	Clue cells, pH >4.5	Metronidazole
Candidiasis	Thick, white, curdy discharge; itching	KOH mount (budding yeast)	Fluconazole

8. GENITAL WARTS (HPV)

- Causative agent: Human papillomavirus (HPV)
- Types 6 and 11: Responsible for most genital warts.
- Clinical features: Multiple, soft, cauliflower-like growths.
- Diagnosis: Usually clinical.
- Treatment options:
  - Topical podophyllin, where appropriate
  - Imiquimod
  - Cryotherapy
  - Surgical removal
- Important: Counsel about HPV vaccination.



9. SYPHILIS MIMICKERS (IMPORTANT DIFFERENTIALS)

Fixed drug eruption

- Well-defined solitary/recurrent ulcer
- Same site on re-exposure to a drug
- History of drug intake

Behçet disease

- Recurrent oral and genital ulcers
- Systemic features

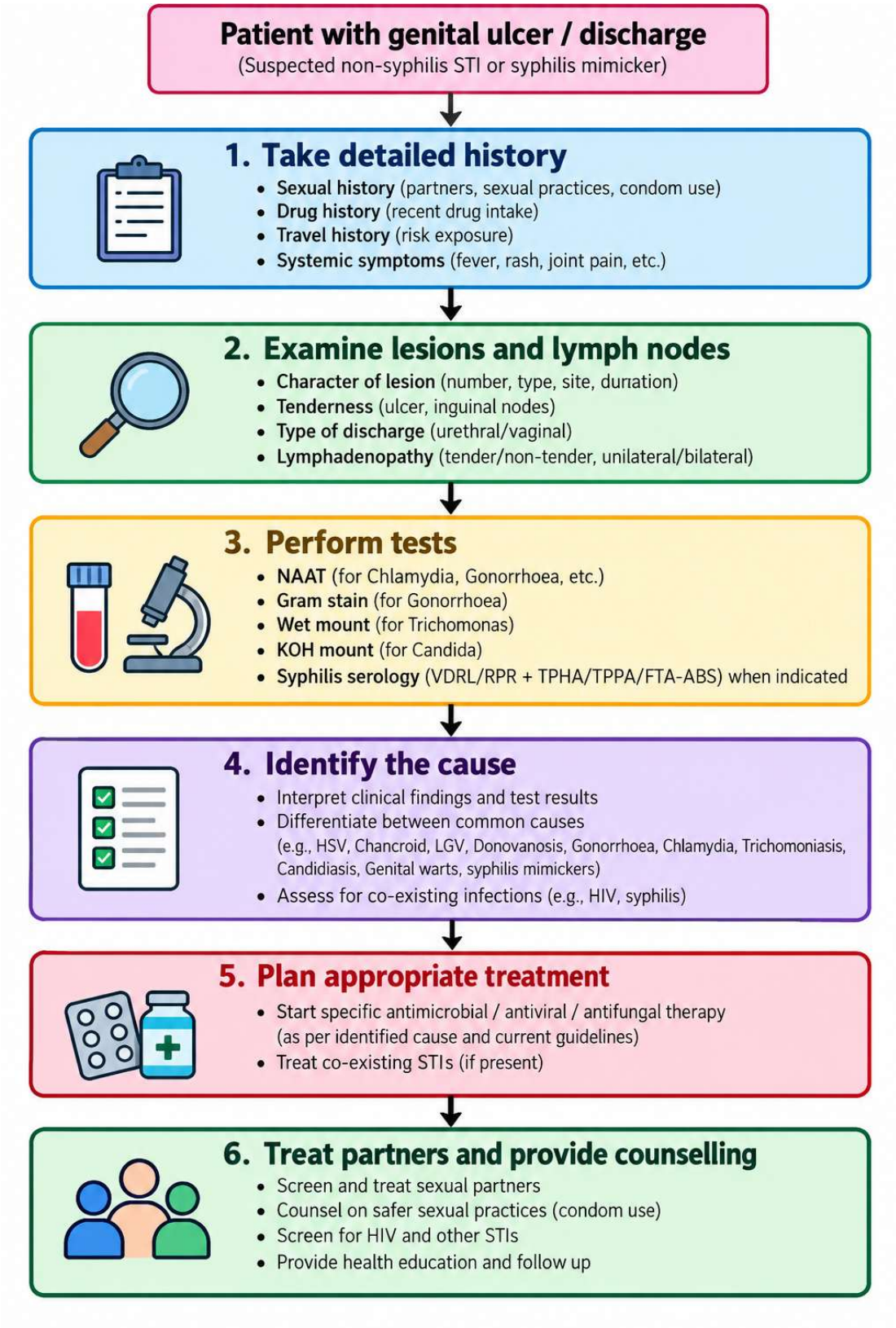
Lipschütz ulcer

- Sudden-onset painful genital ulcers
- Usually in young females
- Often no sexual history
- Self-limiting

Trauma / irritant dermatitis

- History of local trauma or irritant exposure
- Local cause identified

9. DIAGNOSTIC APPROACH



11. MANAGEMENT PRINCIPLES

1. Specific antimicrobial or antiviral therapy.
2. Treat sexual partners when indicated.
3. Screen for other STIs, including HIV and syphilis.
4. Counsel on condom use and safer sexual practices.
5. Offer HPV vaccination when eligible.
6. Follow up for cure, recurrence or persistent symptoms.

- HSV: Painful vesicles and ulcers.
- Chancroid: Painful, soft ulcer with tender lymph nodes.
- LGV: Small initial ulcer followed by painful inguinal lymphadenopathy.
- Donovanosis: Painless, beefy-red ulcer that bleeds easily.
- Gonorrhoea: Profuse purulent discharge.
- Chlamydia: Common cause of non-gonococcal urethritis and cervicitis.
- Trichomoniasis: Frothy, yellow-green discharge.
- Candidiasis: Thick, white, curdy discharge with itching.
- Genital warts: Usually caused by HPV types 6 and 11.
- Always differentiate genital ulcer disease from syphilis and test for coexisting STIs when indicated.

Remember: STIs = Think, Test, Treat, Prevent.

NOTES -

VESICULOBULLOUS DISORDERS

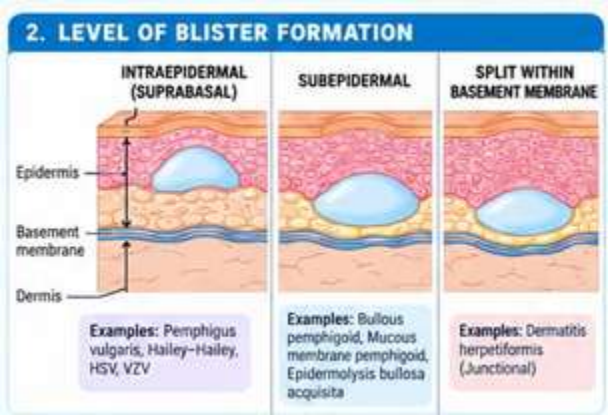
- Vesicle: Small, fluid-filled lesion less than 1 cm.
- Bulla: Large, fluid-filled lesion 1 cm or more.
- Pustule: Contains pus (neutrophils).
- Erosion: Loss of epidermis after rupture.
- Ulcer: Loss of epidermis and dermis.
- Crust: Dried serum, pus or blood.
- Nikolsky sign: Shearing of the epidermis with lateral pressure.
- Tzanck smear: Demonstrates intraepidermal acantholytic cells in pemphigus and HSV/VZV infections.

2. LEVEL OF BLISTER FORMATION

A. Intraepidermal (Suprabasal)

Examples:

- Pemphigus vulgaris
- Pemphigus foliaceus
- Herpes simplex virus (HSV)
- Varicella-zoster virus (VZV)



B. Subepidermal

Examples:

- Bullous pemphigoid
- Mucous membrane pemphigoid
- Epidermolysis bullosa acquisita

C. Split Within the Basement Membrane Zone

Examples:

- Dermatitis herpetiformis
- Linear IgA disease
- Junctional epidermolysis bullosa

3. APPROACH TO A PATIENT

History

- Onset and progression
- Symptoms
- Drug history
- Autoimmune history
- Family history
- Triggers

Examination

- Type of lesion
- Distribution
- Mucosal involvement
- General signs

Classification

- Intraepidermal
- Subepidermal
- Junctional

Relevant Investigations

- Tzanck smear
- Skin biopsy
- Direct immunofluorescence (DIF)
- Serology

Flow

History → Examination → Classification → Investigations → Diagnosis → Management

Always look for

- Mucosal involvement
- Nikolsky sign
- Drug history
- Systemic symptoms
- Family history
- Distribution pattern

4. IMPORTANT VESICULOBULLOUS DISORDERS

Disorder	Level	Key clinical features	Mucosa	Nikolsky sign
Pemphigus vulgaris	Intraepidermal, suprabasal	Flaccid bullae, erosions, usually ruptured	Common	Positive
Pemphigus foliaceus	Superficial epidermal	Superficial crusted erosions, flakes	Absent	Positive
Bullous pemphigoid	Subepidermal	Tense bullae on erythematous base, elderly	Uncommon	Negative
Mucous membrane pemphigoid	Subepidermal	Scarring, chronic mucosal disease	Common	Negative
Dermatitis herpetiformis	Subepidermal	Intensely itchy grouped vesicles, extensor surfaces	Usually absent	Negative
Linear IgA disease	Subepidermal	Annular arrangement of blisters, children	May be involved	Negative
Epidermolysis bullosa (hereditary)	Variable	Trauma-induced blisters, family history	May be involved	Negative
Herpes simplex	Intraepidermal	Grouped painful vesicles, recurrent	Present in some cases	Negative
Varicella-zoster	Intraepidermal	Crops of vesicles, dermatomal distribution in zoster	May be involved	Negative
Impetigo (bullous)	Subepidermal	Flaccid bullae, honey-coloured crust	Absent	Negative
Stevens–Johnson syndrome / TEN	Full-thickness epidermal necrosis	Widespread erosions, target lesions, systemic illness	Common	Positive

5. CLINICAL PEARLS

- Pemphigus vulgaris: Flaccid bullae and erosions.
- Bullous pemphigoid: Tense bullae, usually in elderly patients.
- Dermatitis herpetiformis: Grouped vesicles with intense itching, commonly on extensor surfaces.
- Herpes zoster: Dermatomal vesicles.
- Pemphigus foliaceus: Crusted erosions.
- Linear IgA disease: Annular bullae.
- Impetigo: Honey-coloured crust.
- SJS/TEN: Widespread erosions with mucosal involvement.



6. INVESTIGATIONS

Tzanck smear

- Acantholytic cells: pemphigus.
- Multinucleated giant cells: HSV/VZV.

Skin biopsy (H&E)

- Identifies the level of blister formation and cellular changes.

Direct immunofluorescence (DIF)

- Gold-standard investigation for immunobullous disorders.
- Identifies the pattern of immune deposits.

Indirect immunofluorescence (IIF)

- Detects circulating antibodies.

Serology (ELISA)

- Anti-desmoglein 1 and 3: pemphigus.
- BP180/BP230: bullous pemphigoid.
- Anti-tTG: dermatitis herpetiformis.

Routine tests

- CBC
- Renal function tests

- Liver function tests
- ESR

7. MANAGEMENT PRINCIPLES

- Treat the underlying cause.
- Topical or systemic corticosteroids.
- Immunosuppressants:
  - Azathioprine
  - Mycophenolate mofetil
  - Cyclophosphamide
- Dapsone:
  - Dermatitis herpetiformis
  - Linear IgA disease
- Antivirals:
  - Acyclovir for HSV/VZV, when indicated.
- Supportive care:
  - Wound care
  - Fluid and nutritional support
- Treat secondary infections.
- Regular follow-up.

8. APPROACH TO A BLISTER

Vesicle / Bulla ↓ History + Examination ↓ Assess the blister

- It is flaccid or tense?
- Is mucosal involvement present?
- Is Nikolsky sign positive?

↓

Flaccid blister → Consider intraepidermal disorders → Pemphigus / HSV / VZV → Tzanck smear and skin biopsy as indicated

Tense blister → Consider subepidermal disorders → Bullous pemphigoid / Dermatitis herpetiformis → Skin biopsy + DIF

↓

Confirm diagnosis ↓ Start appropriate treatment

9. IMP - POINTS

1. Vesicle is less than 1 cm; bulla is 1 cm or more.
2. The level of blister formation helps in diagnosis.
3. Nikolsky sign is commonly positive in pemphigus and SJS/TEN.
4. Tense bullae suggest bullous pemphigoid.
5. Direct immunofluorescence is essential in diagnosing many immunobullous disorders.
6. Dermatitis herpetiformis is associated with coeliac disease.
7. Early recognition and supportive care reduce morbidity and mortality.

10. MNEMONIC - BLISTER

- B: Base level (intraepidermal / subepidermal / junctional)
- L: Look for mucosal involvement
- I: Investigations (biopsy, DIF)
- S: Systemic symptoms
- T: Triggers (drugs, infections)
- E: Examine distribution
- R: Right management

EXAM POINTS -

- Flaccid bullae + mucosal involvement → Pemphigus vulgaris
- Tense bullae in elderly → Bullous pemphigoid
- Grouped pruritic vesicles on extensor surfaces → Dermatitis herpetiformis
- Dermatomal vesicles → Herpes zoster
- Widespread erosions + mucosal involvement → SJS/TEN
- Annular bullae → Linear IgA disease

Remember - Identify the level, look for mucosa, and treat early.