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DERMA CLINICAL NOTES SDNOTES

S. No	Topic
0	**BASIC TERMINOLOGIES**
1	Acne Vulgaris
2	Vitiligo
3	Eczema
4	Psoriasis
5	Leprosy
6	Syphilis
7	Non – syphilis
8	Vesiculobullous
9	Cutaneous Manifestations of HIV
10	EOP – ORAL VIVA EXAM

BASIC DERMATOLOGICAL TERMINOLOGIES

1. MACULE

- A **flat**, **circumscribed** area of skin discoloration.
- **No elevation or depression** felt on palpation.
- Size usually **<1 cm**.
- Caused by change in **melanin**, **hemoglobin**, or **pigment deposition**.
- **Examples:** Freckles, vitiligo patch, lentigo.

2. PATCH

- Similar to macule but **larger than 1 cm**.
- Flat lesion with color change but **no surface change**.
- **Examples:** Vitiligo, café-au-lait spots.

3. PAPULE

- **Small**, **solid**, **raised** lesion.
- Usually **<1 cm** in diameter.
- May arise from **epidermis**, **dermis**, or both.
- **Examples:** Lichen planus, acne papule, wart.

4. NODULE

- **Solid**, **palpable**, **deep-seated** lesion extending into the dermis or subcutis.
- Usually **>1 cm**.
- Has **depth and volume** compared to papule.
- **Examples:** Nodular acne, dermatofibroma.

5. TUBERCLE

- **Firm**, circumscribed, palpable lesion in dermis.
- Size between **0.5 – 1 cm**.
- Often leads to **scarring** on healing.
- **Example:** Lupus vulgaris (tuberculous skin lesion).

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6. PLAQUE

- **Flat-topped**, **elevated** lesion.
- Formed by **coalescence of papules**.
- Diameter usually **>1 cm**.
- **Examples:** Psoriasis, lichen planus (large lesions).

---

7. PUSTULE

- **Superficial**, **raised**, **pus-filled** lesion.
- Indicates **infection or inflammation**.
- May arise from hair follicle (follicular pustule).
- **Examples:** Acne pustule, impetigo.

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8. VESICLE

- **Small**, **fluid-filled**, **translucent** blister.
- Contains **clear serous fluid**.
- Size **<0.5 cm**.
- **Examples:** Chickenpox, herpes simplex.

---

9. BULLA (BLISTER)

- **Large vesicle** with diameter **>0.5 cm**.
- May contain **serous or hemorrhagic fluid**.
- Roof may be thin and rupture easily.
- **Examples:** Pemphigus vulgaris, bullous pemphigoid.

---

10. COMEDONE

- **Primary lesion in acne** due to follicular blockage by keratin and sebum.
- **Open comedone:** "Blackhead" – dark due to oxidized keratin.
- **Closed comedone:** "Whitehead" – covered by thin epithelium.
- Non-inflammatory lesion.

---

11. CYST

- **Closed sac-like structure** with **epithelial lining**.
- Contains **fluid, semi-fluid, or keratinous material**.
- Located in dermis or subcutaneous tissue.
- **Examples:** Epidermoid cyst, sebaceous cyst.

---

12. WHEAL

- **Transient**, **edematous**, **raised** lesion.
- Caused by **dermal edema** due to histamine release.
- Lesions are **itchy**, **evanescent**, and **migrate**.
- **Example:** Urticaria.

---

13. SCALE

- **Flakes of keratin** that separate from the surface.
- Indicates **abnormal keratinization or desquamation**.
- **Examples:** Psoriasis (silvery scales), pityriasis rosea.

---

14. CRUST

- **Dried exudate** (serum, pus, or blood) on the surface.
- May form after vesicle or pustule ruptures.
- **Examples:** Impetigo (honey-colored crusts).

---

15. EROSION

- **Superficial loss of epidermis** only.
- Heals **without scarring**.
- Occurs after rupture of vesicle or pustule.
- **Example:** Herpes simplex after vesicle rupture.

---

16. ULCER

- **Loss of epidermis and part of dermis**.
- **Heals with scar formation**.
- May have discharge or slough.
- **Examples:** Syphilitic chancre, venous ulcer.

---

17. FISSURE

- **Linear crack** in skin extending into dermis.
- Caused by dryness or thickened skin.
- **Examples:** Angular cheilitis, heel fissures.

---

18. SCAR (CICATRIX)

- **Fibrous tissue** replacing damaged dermis after healing.
- **Types:**
  - **Atrophic:** Depressed (e.g., acne scar)
  - **Hypertrophic:** Raised but within boundary
  - **Keloid:** Extends beyond wound margin

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19. LICHENIFICATION

- **Thickened skin** with **accentuated skin markings**.
- Due to **chronic scratching or rubbing**.
- **Example:** Lichen simplex chronicus.

---

20. EXCORIATION

- **Linear abrasion** caused by scratching.
- May lead to **secondary infection**.
- **Examples:** Pruritic dermatoses.

---

21. ATROPHY

- **Thinning** of epidermis, dermis, or subcutaneous tissue.
- Skin appears **thin, wrinkled, shiny**.
- **Examples:** Senile atrophy, long-term steroid use.

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22. HYPERKERATOSIS

- **Thickening of stratum corneum** (outermost epidermal layer).
- Seen in **chronic friction or keratinization disorders**.
- **Examples:** Callus, psoriasis.

---

23. TELANGIECTASIA

- **Visible dilated superficial blood vessels**.
- Seen in **rosacea, chronic sun exposure, corticosteroid use**.

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24. SINUS

- **Abnormal tract** from deeper tissue to skin surface.
- Lined by granulation tissue, may discharge pus.
- **Example:** Acne conglobata, pilonidal sinus.

---

25. FISTULA

- **Abnormal communication** between two epithelial-lined surfaces.
- **Example:** Orocutaneous fistula, anal fistula.

ACNE VULGARIS

Definition

Acne vulgaris is a **chronic inflammatory disease of the pilosebaceous unit** (hair follicle and sebaceous gland) characterized by comedones, papules, pustules, nodules, and sometimes cysts.

Epidemiology

- Common in **adolescents** and young adults.
- Affects both sexes; males often have more severe disease.
- Peak incidence: **13–18 years**.
- Can persist into adulthood (especially in females).

Etiopathogenesis

Factor	Description
1. Increased Sebum Production	Due to androgen stimulation during puberty.
2. Follicular Hyperkeratinization	Increased keratinocyte proliferation and abnormal desquamation lead to comedone formation.
3. Cutibacterium acnes (formerly Propionibacterium acnes)	Bacterial colonization in blocked follicles causes inflammation.
4. Inflammation	Triggered by bacterial lipases, cytokines, and immune response.

Types of Acne Lesions

Type	Description	Example
Non-inflammatory	Comedones	Open (blackhead), Closed (whitehead)
Inflammatory	Papules, pustules, nodules, cysts	Red, tender lesions
Severe/Complicated	Acne conglobata, acne fulminans	Nodulocystic or ulcerative forms

Grading of Acne

Grade	Lesions	Description
I	Comedonal	Blackheads and whiteheads only
II	Papulopustular	Inflammatory papules and pustules
III	Nodulocystic	Deep, painful nodules and cysts
IV	Confluent/Nodular	Severe scarring and sinus formation

Risk Factors

Category	Examples
Hormonal	Puberty, menstrual irregularities, PCOS, androgens
Genetic	Family history of acne
Lifestyle	High-glycemic diet, dairy intake, stress, cosmetics ("pomade acne")
Drugs	Steroids, lithium, isoniazid, phenytoin
Occupational	Exposure to oils, tar, chlorinated hydrocarbons

Clinical Features

- **Sites:** Face, chest, shoulders, back.
- **Lesions:** Comedones (open/closed), papules, pustules, nodules, cysts.
- **Post-acne sequelae:** Pigmentation, scarring (atrophic, hypertrophic, keloidal).

Investigations

Usually not required.
If indicated:

- **Hormonal profile:** For adult female acne or resistant cases.
- **Culture:** For recurrent/infective lesions.

Management

1. General Measures

- Gentle cleansing with mild soap or pH-balanced face wash.
- Avoid picking/squeezing lesions.
- Non-comedogenic moisturizers and sunscreens.
- Balanced diet (avoid high sugar/dairy).

---

2. Topical Therapy

Drug/Class	Examples	Mechanism / Use
Retinoids	Tretinoin, adapalene, tazarotene	Normalize keratinization, comedolytic
Antibiotics	Clindamycin, erythromycin	Reduce bacterial load and inflammation
Benzoyl Peroxide	—	Antibacterial, prevents resistance
Azelaic Acid	—	Comedolytic and anti-inflammatory

Combination therapy (e.g., retinoid + antibiotic or benzoyl peroxide) is preferred.

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3. Systemic Therapy

Drug/Class	Indication	Notes
Antibiotics	Moderate–severe inflammatory acne	Doxycycline, minocycline (limit to 3–6 months)
Hormonal Therapy	Females with hormonal acne	OCPs (cyproterone acetate), spironolactone
Isotretinoin	Severe nodulocystic acne	Teratogenic, monitor LFTs & lipids
Corticosteroids	Acne fulminans	Short course only

---

4. Procedural Treatments

- **Comedone extraction**
- **Chemical peels (salicylic/glycolic acid)**
- **Laser or light therapy**
- **Scar management:** Microneedling, subcision, laser resurfacing

---

Complications

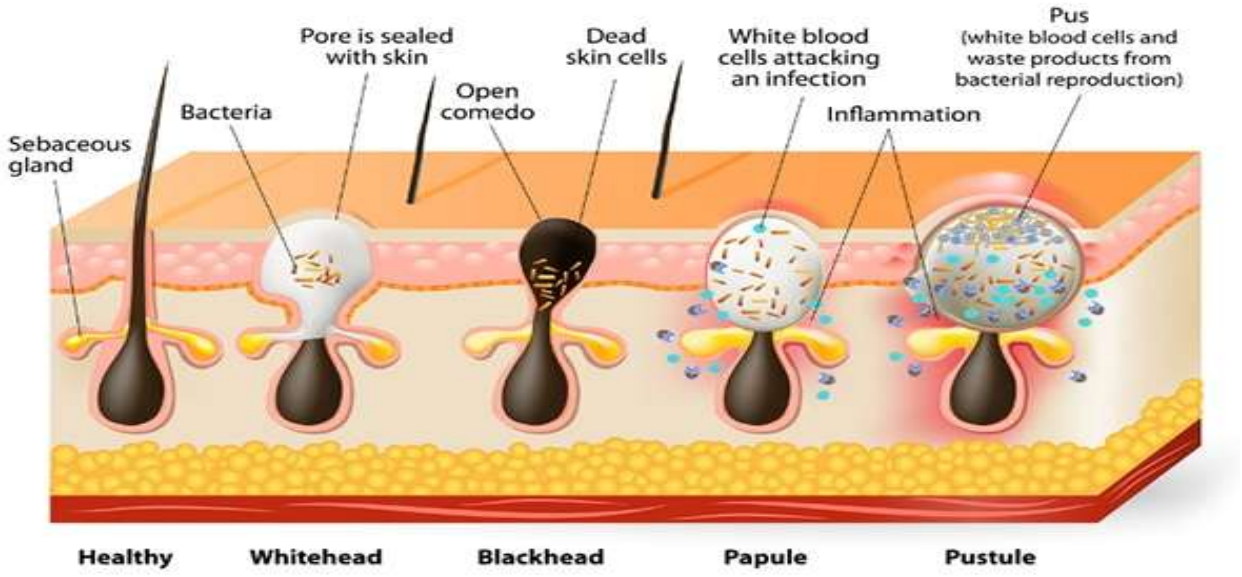
- Post-inflammatory hyperpigmentation
- Scarring (atrophic, hypertrophic, keloidal)
- Psychosocial distress

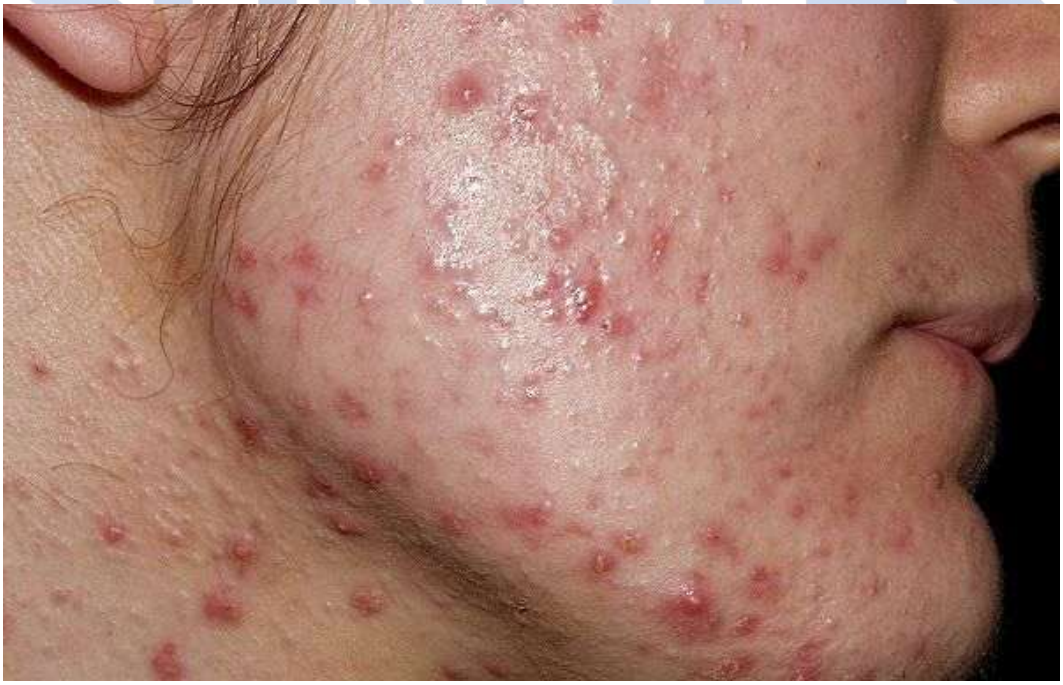
Prognosis

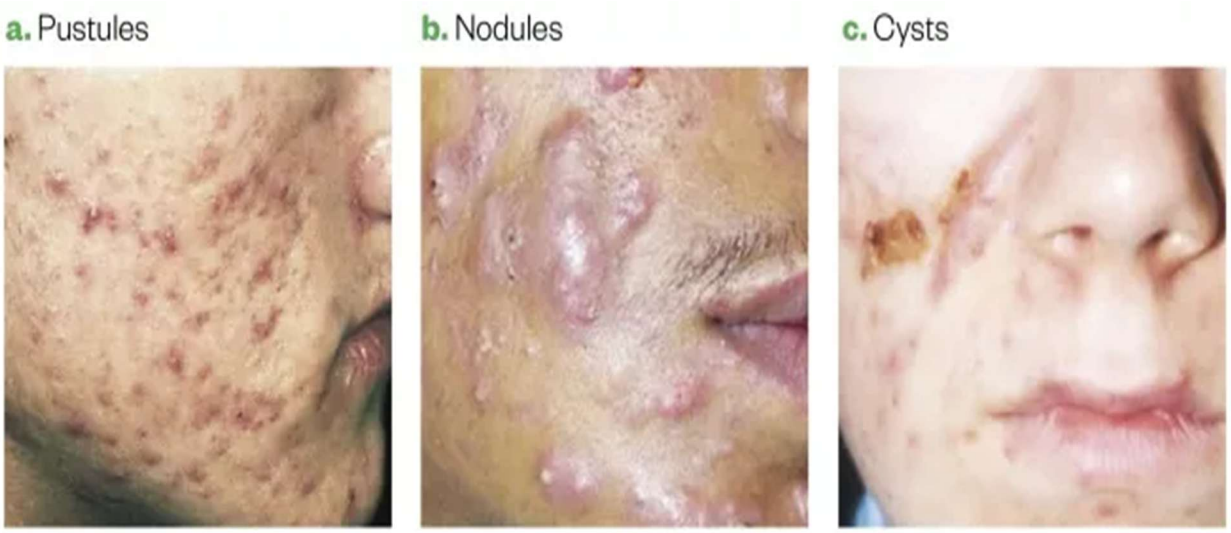
- Generally good with early treatment.
- May relapse; maintenance therapy needed.

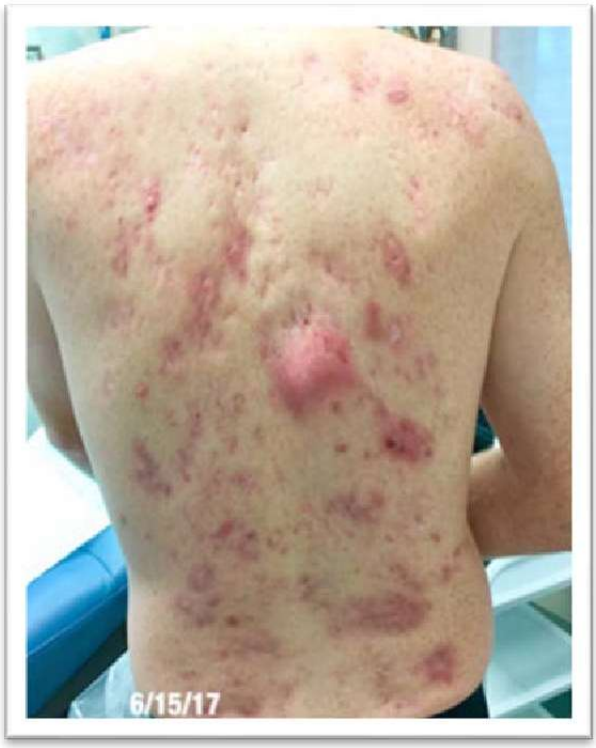
Key Points for Clinics

- Identify lesion types and grades.
- Assess for scarring and pigmentation.
- Choose appropriate therapy according to grade.
- Educate patient on adherence and skincare.


Pore is sealed with skin
Dead skin cells
White blood cells attacking an infection
Pus
(white blood cells and waste products from bacterial reproduction)
Bacteria
Open comedo
Inflammation
Sebaceous gland
Healthy
Whitehead
Blackhead
Papule
Pustule




a. Pustules
b. Nodules
c. Cysts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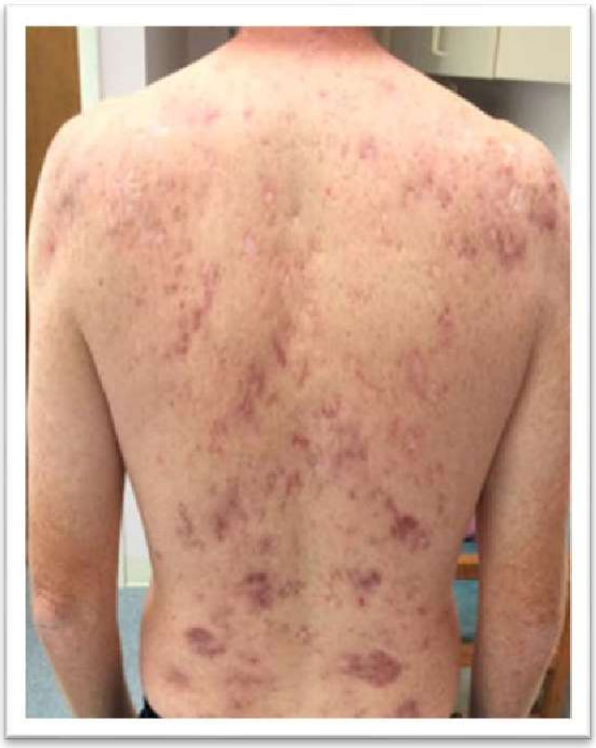

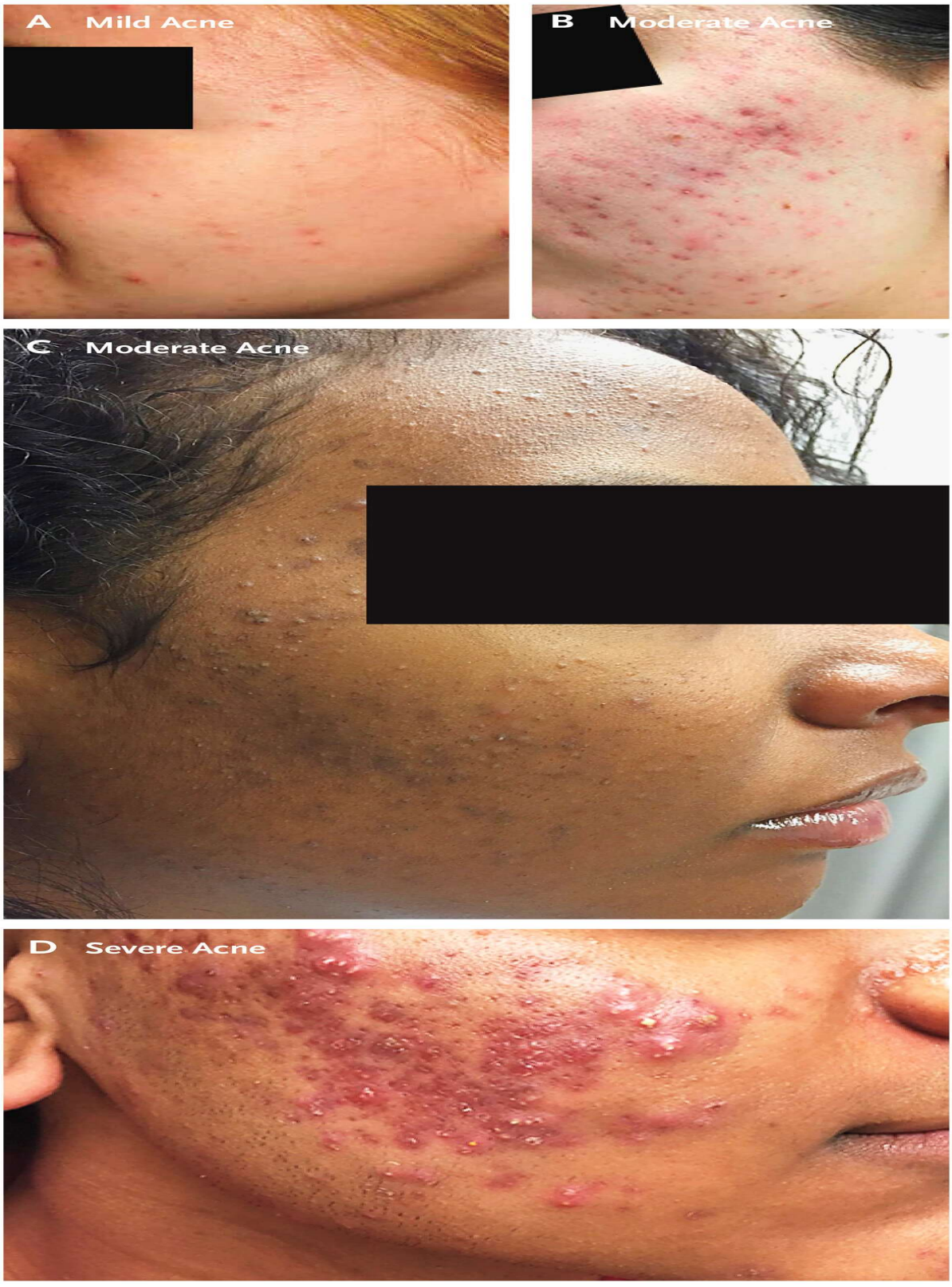
A Mild Acne
B Moderate Acne
C Moderate Acne
D Severe Acne

FOR NOTES -

VITILIGO

Definition

Vitiligo is an **acquired, idiopathic depigmentary disorder** of the skin and mucous membranes, characterized by **well-defined milky white macules and patches** due to loss of functional melanocytes.

Epidemiology

- Affects about **0.5–2%** of the population worldwide.
- **No sex predilection.**
- Commonly starts **before 20 years of age.**
- Often associated with **autoimmune diseases.**

Etiopathogenesis

Vitiligo results from **destruction or functional loss of melanocytes** in the epidermis.
Multiple theories explain its cause:

Theory	Description
Autoimmune theory	Most accepted; autoantibodies and cytotoxic T-cells destroy melanocytes.
Neurogenic theory	Nerve endings release toxic substances affecting melanocytes.
Self-destructive theory	Defective melanin synthesis leads to accumulation of toxic intermediates that destroy melanocytes.
Genetic factors	20–30% cases show family history; polygenic inheritance pattern.

Risk Factors / Associations

Category	Examples
Genetic	Family history of vitiligo
Autoimmune diseases	Thyroid disorders, diabetes mellitus type 1, alopecia areata, pernicious anemia, Addison's disease
Environmental / External	Sunburn, emotional stress, trauma (Koebner phenomenon), chemicals (phenols, catechols)
Others	Oxidative stress, infections, nutritional deficiencies (B12, folate, copper, zinc)

Clinical Features

- **Depigmented macules and patches**: well-defined, chalky-white, often symmetrical.
- **Sites commonly affected**: face, hands, feet, elbows, knees, around body orifices, and genitals.
- **Hair** over lesions may turn **white (leukotrichia).**
- **Koebner phenomenon**: new lesions appear over sites of trauma.
- **Course**: Progressive, stable, or rarely spontaneous repigmentation.

Classification / Types

Type	Description / Distribution	Examples
Non-segmental (generalized)	Bilateral symmetrical distribution; most common type	Vulgaris, Acrofacial, Universal
Segmental	Unilateral, follows dermatomal pattern; early onset; stable course	One side of face or limb
Mixed	Both segmental and non-segmental lesions present	Combination type
Localized	Few patches in one area	Focal or mucosal vitiligo

Investigations

- **Clinical diagnosis** (based on appearance).
- **Wood's lamp examination**: lesions show bright blue-white fluorescence.
- **Thyroid function tests**: to rule out autoimmune thyroiditis.
- **Autoantibody screening**: ANA, anti-thyroid antibodies if indicated.
- **Skin biopsy (rare)**: shows absence of melanocytes.

Differential Diagnosis

Condition	Differentiating Features
Pityriasis alba	Hypopigmented (not depigmented), fine scaling
Nevus depigmentosus	Present from birth, non-progressive
Tinea versicolor	Fine scaling, positive for fungal elements on KOH
Chemical leukoderma	History of exposure to chemicals

Management

1. General Measures

- Patient counseling and reassurance (chronic but benign condition).
- Avoid trauma, sunburn, and harsh chemicals.
- Use **sunscreens** to prevent tanning of surrounding skin.
- Cosmetic camouflage if desired.

---

2. Medical Therapy

Drug / Treatment	Mechanism / Use
Topical corticosteroids	First-line for localized disease; anti-inflammatory; stimulate repigmentation
Topical calcineurin inhibitors (Tacrolimus / Pimecrolimus)	Especially for face and intertriginous areas
Phototherapy (NB-UVB)	Most effective for generalized vitiligo; induces melanocyte proliferation
PUVA therapy (Psoralen + UVA)	For widespread lesions; more side effects
Systemic corticosteroids	Used in rapidly progressive vitiligo (short course)
Antioxidants (Vit C, E, alpha-lipoic acid)	Adjunctive therapy

3. Surgical Therapy

Indicated for **stable vitiligo (>1 year stability)** not responding to medical therapy.

Procedure	Description
Punch grafting	Small grafts transplanted to depigmented area
Split-thickness skin grafting	Thin skin grafts placed over lesions
Suction blister grafting	Blisters created by suction used for grafting
Melanocyte-keratinocyte transplantation	Advanced technique for stable cases

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4. Depigmentation Therapy

- For **extensive vitiligo (>50% body surface)**.
- **Monobenzyl ether of hydroquinone** used to depigment remaining normal skin for uniformity.

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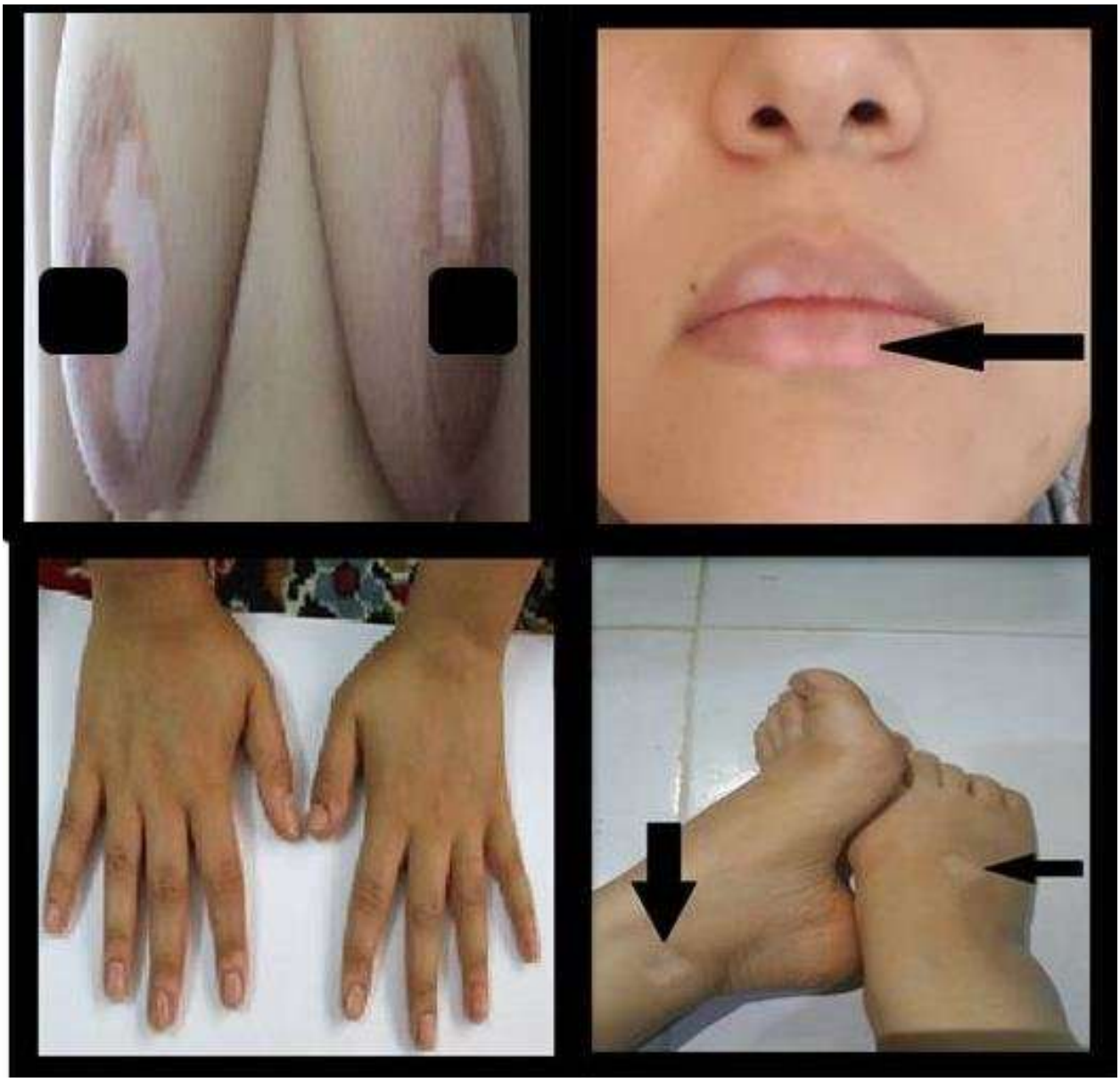
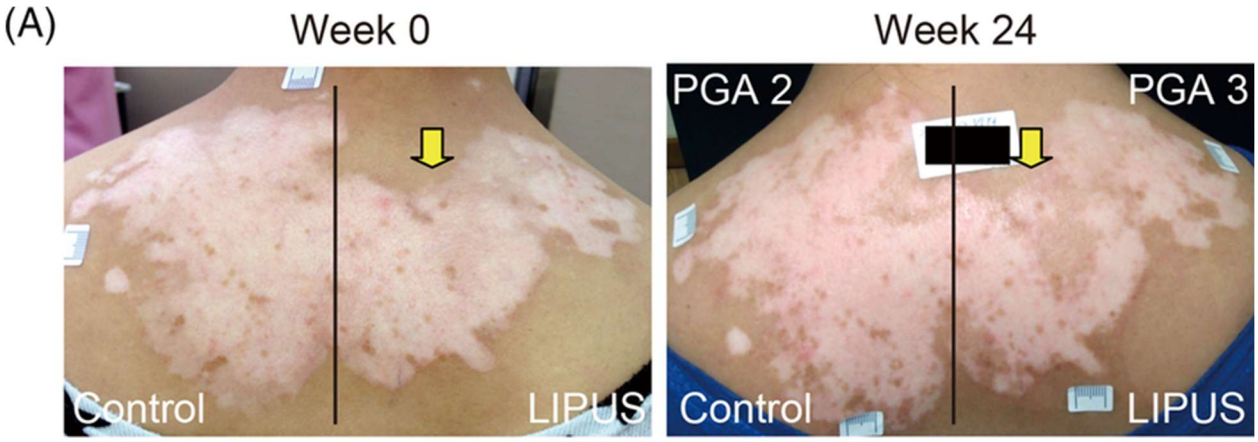
5. Psychological Support

- Address anxiety, depression, and social stigma.
- Support groups and counseling can help coping.

Prognosis

- Chronic course; variable response to treatment.
- **Better prognosis** in: recent onset, face/trunk lesions, dark skin.
- **Poor prognosis** in: acral areas, mucosal involvement, leukotrichia.

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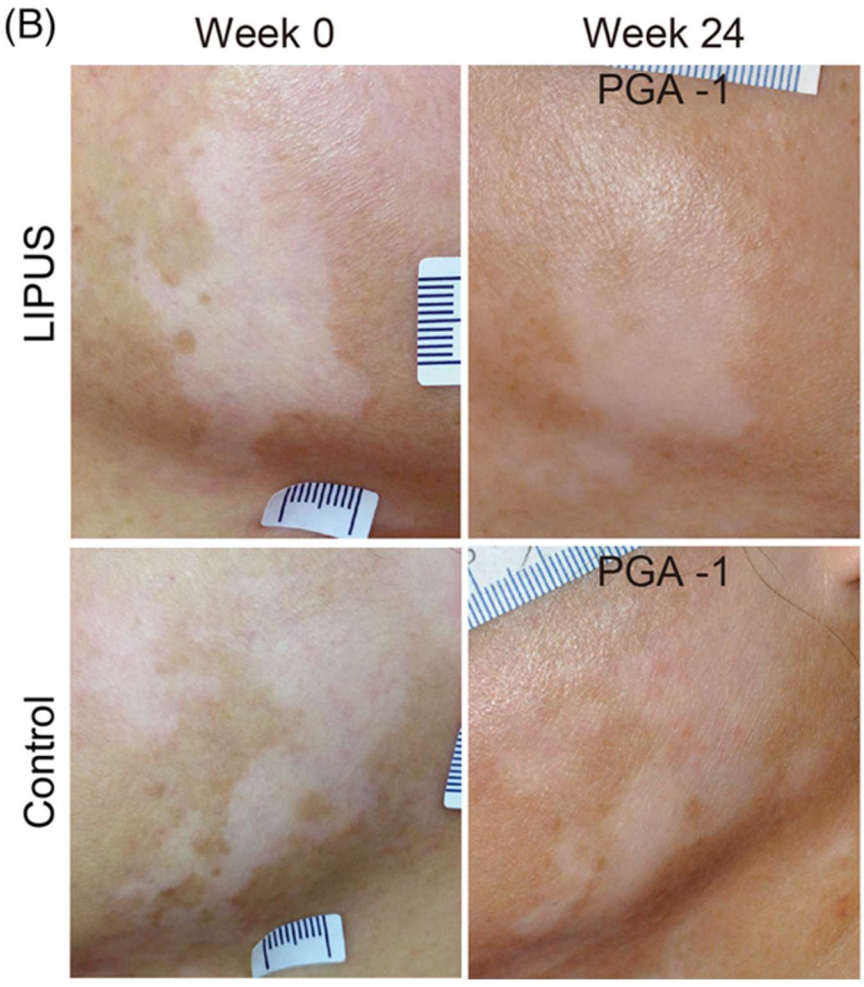
Week 24
PGA 2
PGA 3
Control
LIPUS
Control
LIPUS


(B)
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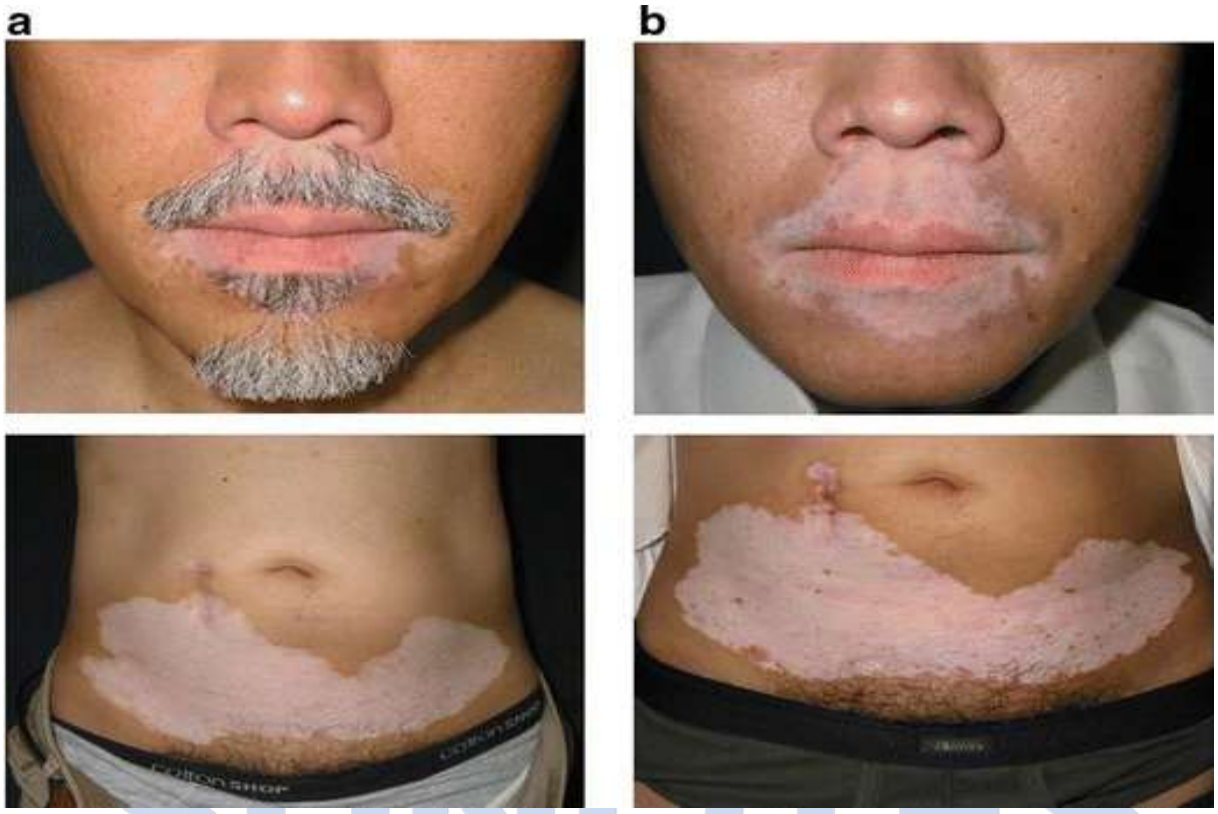
PGA -1
LIPUS
PGA -1
Control


a
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FOR NOTES -

Eczema (Dermatitis)

Definition

Eczema is a **chronic, relapsing, inflammatory skin disease** characterized by **itching, redness, vesiculation, and scaling**.
It represents a pattern of skin inflammation caused by a variety of external and internal factors.

Classification / Types

Type	Description / Key Features
1. Atopic dermatitis	Common in children; associated with personal/family history of atopy (asthma, allergic rhinitis). Common sites: flexures, face, neck.
2. Contact dermatitis	Due to contact with irritants or allergens. • *Irritant contact dermatitis* – chemical, detergents • *Allergic contact dermatitis* – nickel, cosmetics, plants.
3. Seborrheic dermatitis	In sebaceous areas (scalp, face, chest). Greasy scales and erythema. Linked to *Malassezia*.
4. Nummular eczema	Coin-shaped lesions, usually on limbs, very itchy.
5. Dyshidrotic (Pompholyx)	Deep-seated vesicles on palms, soles, sides of fingers; aggravated by stress, sweating.
6. Stasis dermatitis	Seen in lower legs with venous insufficiency. Pigmentation, scaling, ulceration.
7. Asteatotic eczema	Due to excessive dryness; common in elderly, cold weather. Cracked skin ("eczema craquelé").

Etiopathogenesis

- **Genetic factors:** Filaggrin gene mutation → impaired skin barrier.
- **Immune dysregulation:** ↑ Th2 cytokines → inflammation.
- **Environmental triggers:** soaps, detergents, allergens, microbes, climate.
- **Psychological stress:** exacerbates itching and flare-ups.

Risk Factors

Intrinsic	Extrinsic
Family history of atopy	Contact with irritants/allergens
Dry skin (xerosis)	Climate – cold, low humidity
Genetic predisposition	Stress, infection, sweating
Immune dysfunction	Excessive washing, harsh soaps

Clinical Features

- **Pruritus** – hallmark symptom
- **Erythema**, **papules**, **vesicles**, **oozing**, **crusting**, and **scaling**
- **Lichenification** in chronic cases
- **Distribution:**
  - Infants – face, scalp, extensor surfaces
  - Children – flexural areas
  - Adults – hands, eyelids, neck, flexures

Investigations (if needed)

- Mostly **clinical diagnosis**
- Patch test → to identify allergens (contact dermatitis)
- Skin biopsy → rarely needed to rule out other dermatoses
- Serum IgE → often elevated in atopic dermatitis

Management

General Measures

- Avoid triggering factors (irritants, allergens, stress).
- Use **lukewarm water**, mild **fragrance-free soaps**.
- Apply **emollients** frequently (restores barrier).
- Avoid scratching – keep nails short.
- Wear cotton clothing.

Topical Therapy

Medication	Use / Notes
Topical corticosteroids	Mainstay of treatment; reduces inflammation. Use mild to moderate potency depending on site.
Topical calcineurin inhibitors (Tacrolimus, Pimecrolimus)	For sensitive areas (face, eyelids, flexures).
Emollients / moisturizers	Regular use to prevent dryness.
Antibiotic creams	If secondary infection present.

Systemic Therapy

Drug	Indication
Antihistamines	Relieve itching.
Oral corticosteroids	For acute severe flares (short course).
Immunosuppressants (Cyclosporine, Methotrexate, Azathioprine)	For refractory or chronic cases.
Antibiotics	If secondary bacterial infection.
Phototherapy (NB-UVB)	For chronic, extensive eczema.

Complications

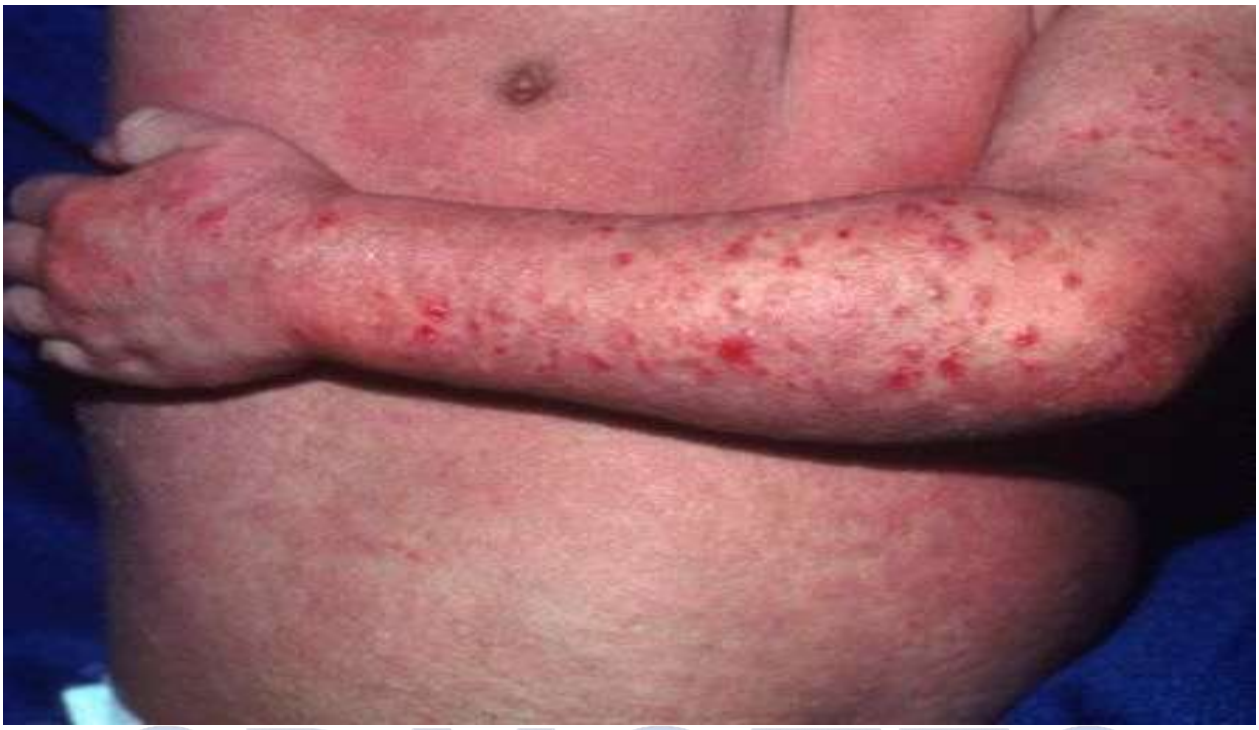
- Secondary infection (Staphylococcus aureus)
- Lichenification (thickened skin)
- Sleep disturbance due to itching
- Psychological stress, low self-esteem

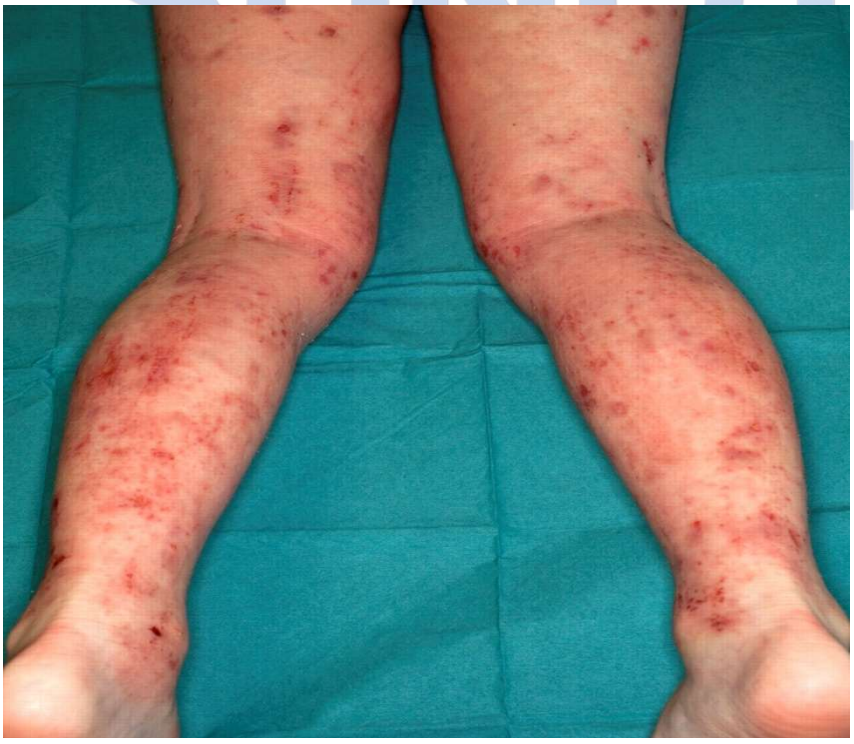
Prognosis

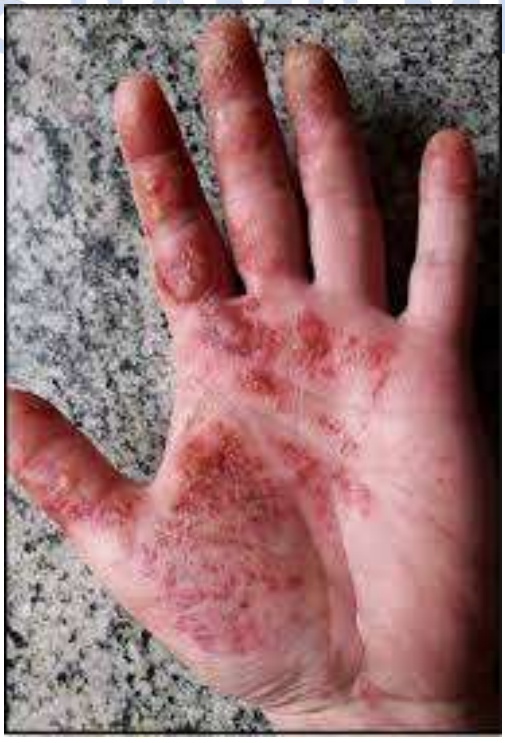
- Chronic, relapsing course.
- Many childhood cases improve with age.
- Proper skin care and trigger avoidance → good control.

Summary -

Aspect	Key Points
Main symptom	Itching
Common sites	Flexures, hands, face
Key finding	Erythema, scaling, vesicles
Treatment	Emollients + topical steroids
Prevention	Avoid irritants, maintain hydration







FOR NOTES –

Psoriasis

Definition

Psoriasis is a **chronic, immune-mediated inflammatory skin disorder** characterized by **well-defined, erythematous plaques** with **silvery-white scales**.
It shows **remission and relapse** and involves **genetic, immunologic, and environmental** factors.

Epidemiology

- Common worldwide (affects ~2–3% of population)
- **Bimodal onset:**
  - Type I: Early onset (<40 years), familial, severe
  - Type II: Late onset (>40 years), sporadic, mild
- No sex predilection

Etiopathogenesis

Factor	Description
Genetic	Strong family history (HLA-Cw6, HLA-B13, HLA-B17 association)
Immunologic	T-cell mediated autoimmune response → cytokine release (TNF-α, IL-17, IL-23) → keratinocyte proliferation
Environmental triggers	Trauma (Koebner phenomenon), infections (Streptococcal), stress, drugs, cold climate, alcohol, smoking

Types of Psoriasis

Type	Features
1. Chronic Plaque Psoriasis (Psoriasis vulgaris)	Most common; symmetrical plaques with silvery scales on extensor surfaces (elbows, knees, scalp, lumbosacral area)
2. Guttate Psoriasis	Small, drop-like lesions; often after streptococcal throat infection; seen in children/young adults
3. Erythrodermic Psoriasis	Generalized erythema and scaling involving >90% BSA; may lead to systemic symptoms
4. Pustular Psoriasis	Sterile pustules over erythematous skin; generalized (Von Zumbusch type) or localized (palmo-plantar)
5. Inverse (Flexural) Psoriasis	Smooth, shiny plaques in flexures (axilla, groin, inframammary); less scaling
6. Nail Psoriasis	Pitting, onycholysis, subungual hyperkeratosis, oil drop sign
7. Psoriatic Arthritis	Joint involvement; can resemble rheumatoid arthritis; DIP joints often affected

Clinical Features

- **Lesions:** Well-demarcated erythematous plaques with silvery-white scales
- **Distribution:** Extensor surfaces, scalp, lumbosacral area
- **Auspitz sign:** Pinpoint bleeding spots on removing scales
- **Koebner phenomenon:** New lesions on sites of trauma
- **Systemic features:** Arthropathy, nail changes, metabolic syndrome association

Risk Factors

Category	Examples
Genetic	Family history of psoriasis
Immunologic	Autoimmune T-cell activation
Environmental	Cold climate, dry weather
Infectious	Streptococcal throat infections (especially guttate type)
Drugs	β-blockers, lithium, antimalarials, NSAIDs, ACE inhibitors
Lifestyle	Smoking, alcohol intake, obesity
Stress	Psychological stress can precipitate/exacerbate disease

Complications

- Psoriatic arthritis
- Secondary infection
- Erythroderma
- Metabolic syndrome
- Psychological impact (depression, anxiety)

Management

1. General Measures

- Avoid triggers (trauma, stress, alcohol, smoking)
- Moisturizers to reduce dryness
- Gentle skin care
- Patient education and reassurance (chronic but controllable disease)

---

2. Topical Therapy

Agent	Examples	Indication
Emollients	Petroleum jelly, paraffin	All cases (base therapy)
Keratolytics	Salicylic acid	Remove scales
Topical corticosteroids	Betamethasone, clobetasol	Anti-inflammatory
Vitamin D analogues	Calcipotriol	Regulates keratinocyte proliferation
Coal tar / Dithranol	Traditional agents	Chronic stable plaques
Topical calcineurin inhibitors	Tacrolimus, pimecrolimus	Sensitive areas (face, flexures)

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3. Phototherapy

- **UVB narrowband** or **PUVA (Psoralen + UVA)**
- Indicated for extensive or refractory cases
- Avoid in photosensitive patients

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4. Systemic Therapy

Drug	Use / Notes
Methotrexate	Severe, extensive psoriasis; monitor liver and blood counts
Cyclosporine	Rapid control; nephrotoxicity risk
Acitretin	Pustular / erythrodermic types; teratogenic
Apremilast	PDE4 inhibitor; moderate disease
Biologics	Anti-TNF (Etanercept, Infliximab), IL-17/23 inhibitors (Secukinumab, Ustekinumab)

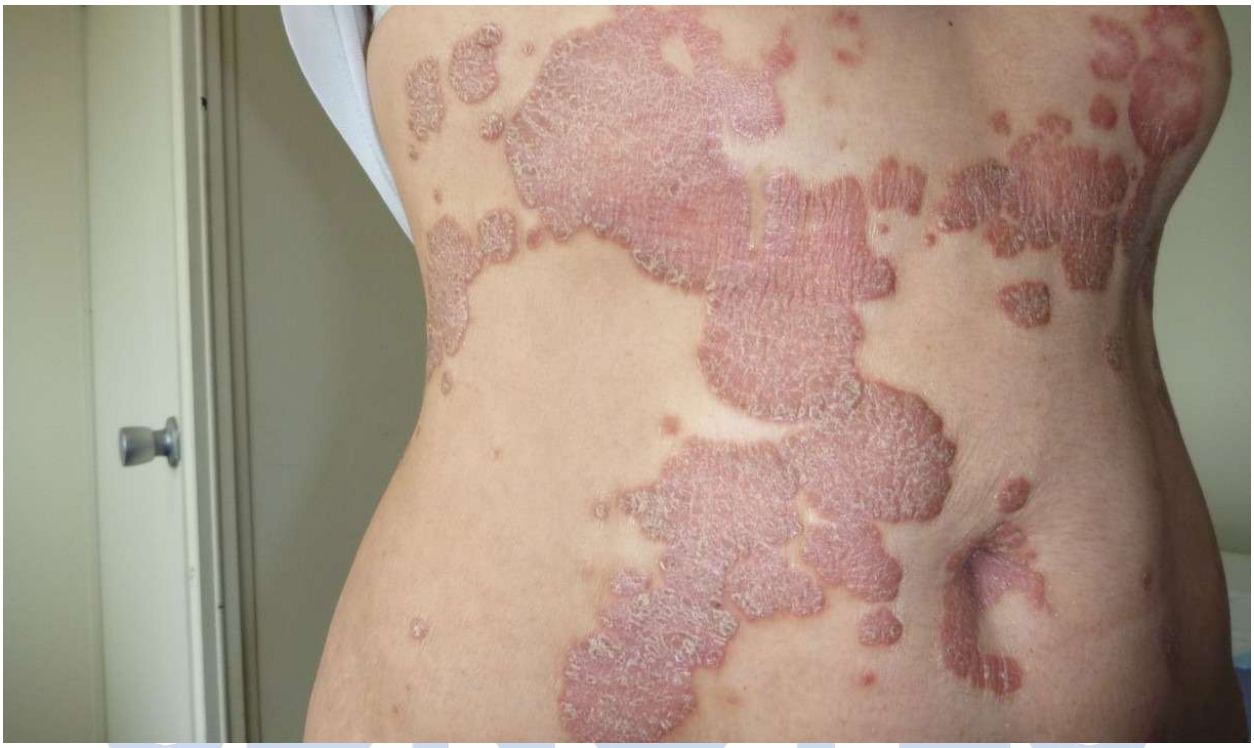
5. Management of Psoriatic Arthritis

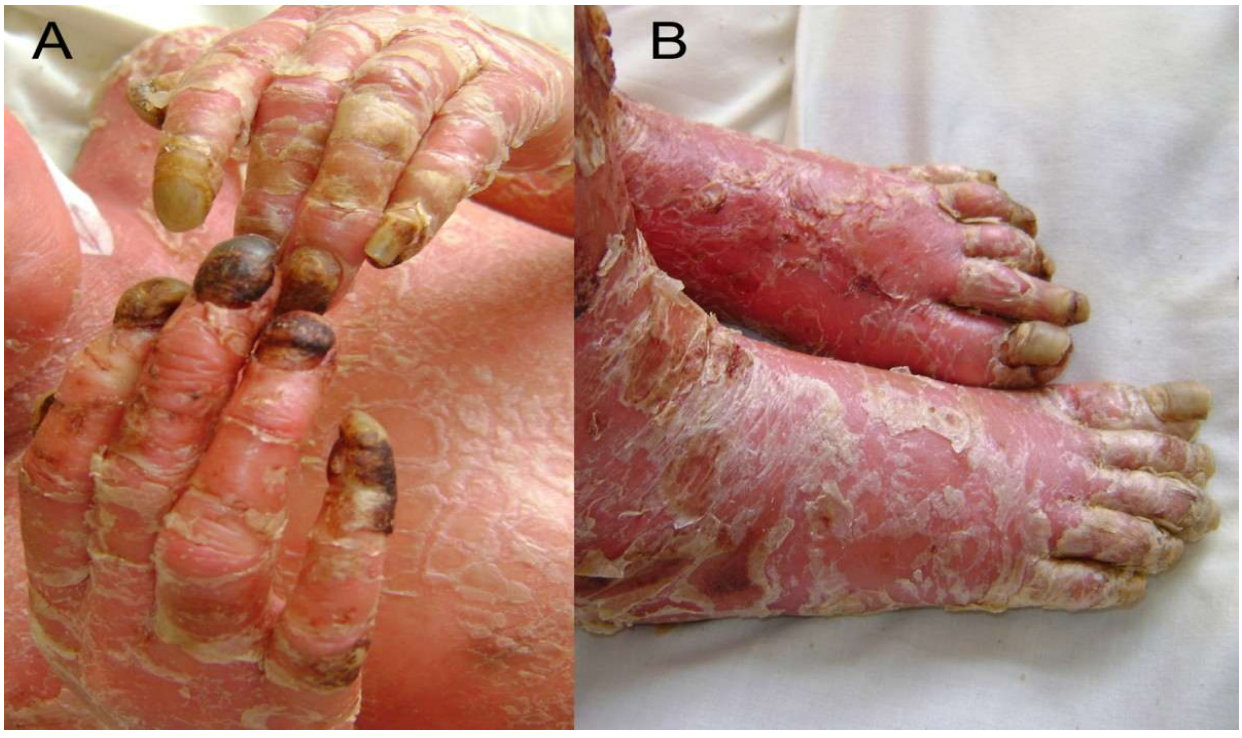
- NSAIDs for pain relief
- DMARDs (Methotrexate, Leflunomide)
- Biologic agents if refractory

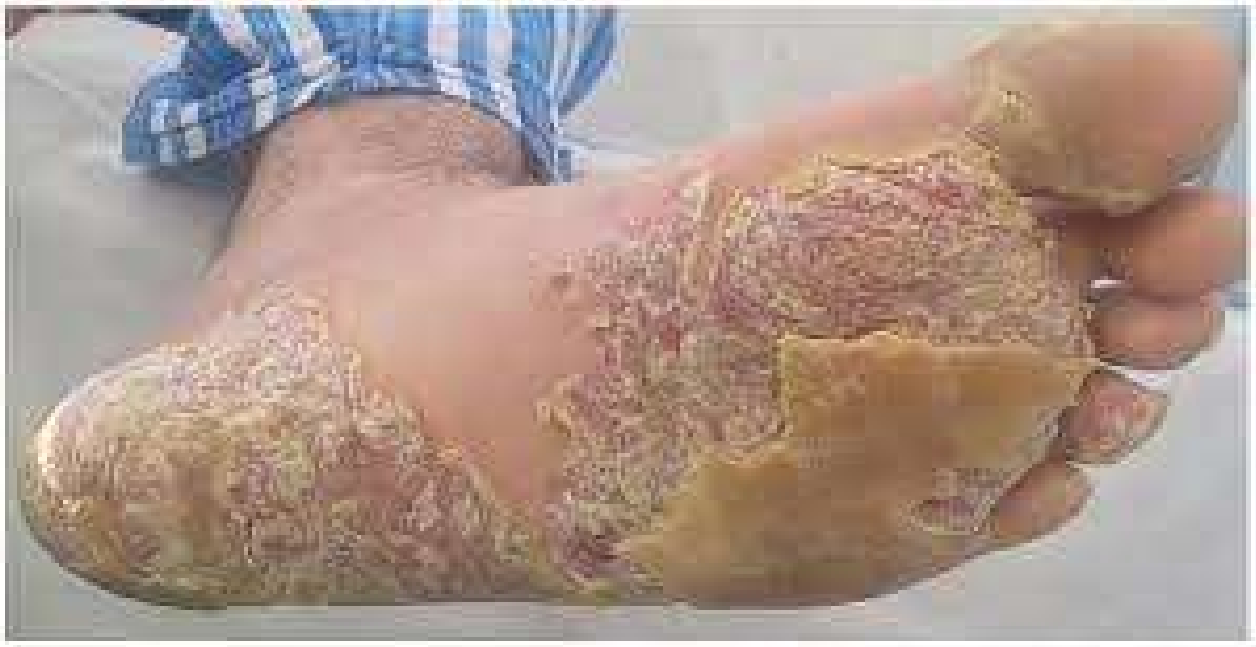
Prognosis

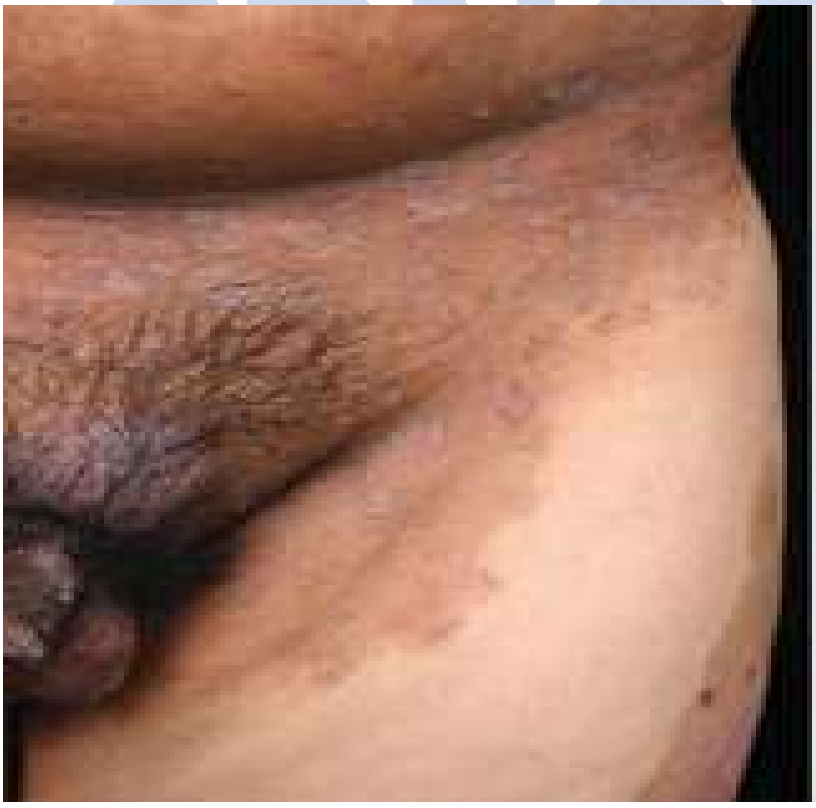
- Chronic course with remissions and exacerbations
- May improve in summer, worsen in winter
- Good prognosis with adherence to treatment and lifestyle modification

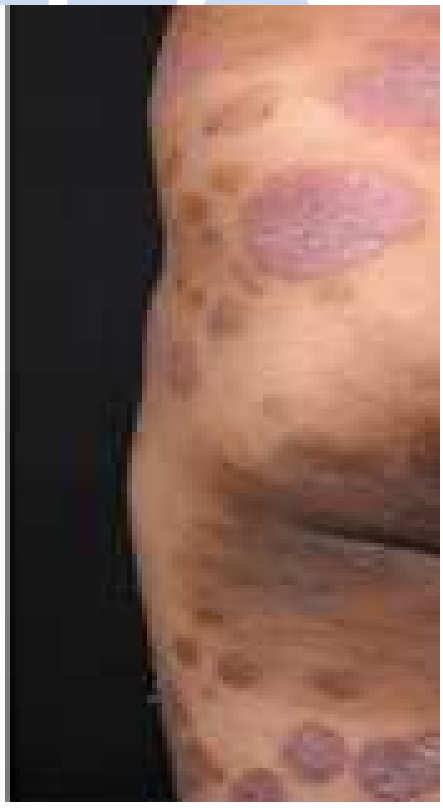
Chronic Plaque Psoriasis











FOR NOTES -

Leprosy (Hansen's Disease)

Definition

Leprosy is a **chronic infectious disease** caused by *Mycobacterium leprae*, an **acid-fast bacillus** that primarily affects the **skin, peripheral nerves, mucosa of the upper respiratory tract, and eyes**.

Causative Agent

- *Mycobacterium leprae*
- Acid-fast, rod-shaped, obligate intracellular parasite
- Grows best at cooler temperatures (27–33°C) → hence affects peripheral areas (e.g., face, hands, feet)

Mode of Transmission

Route	Description
Droplet infection	Prolonged close contact with untreated lepromatous cases through nasal droplets
Skin contact	Rare; only if there are breaks in the skin
Vertical transmission	Very rare
Incubation period	Long – usually **3–10 years** (average 5 years)

Risk Factors

Category	Examples
Host factors	Genetic susceptibility (HLA associations), poor immunity
Environmental	Overcrowding, poor sanitation, endemic areas
Socioeconomic	Poverty, malnutrition, limited access to healthcare
Contact factors	Close contact with untreated multibacillary cases

Pathogenesis

1. *M. leprae* enters the body → multiplies slowly in macrophages and Schwann cells.
2. Immune response determines the **clinical spectrum**:
    - **Strong cell-mediated immunity (CMI)** → tuberculoid form
    - **Poor CMI** → lepromatous form
3. Nerve involvement leads to anesthesia, deformities, and disability.

Classification (Ridley–Jopling Spectrum)

Type	Immunity	Skin Lesions	Nerve Involvement	Lepromin Test	Bacilli (AFB)
TT (Tuberculoid)	High CMI	Few, well-defined, hypopigmented patches	Marked, asymmetrical	Positive	Negative
BT (Borderline Tuberculoid)	Moderate	Few to several lesions	Asymmetrical	Weakly positive	Few
BB (Borderline Borderline)	Intermediate	Many lesions, irregular	Symmetrical or asymmetrical	Negative	Moderate
BL (Borderline Lepromatous)	Poor	Numerous, ill-defined	Symmetrical	Negative	Many
LL (Lepromatous)	Absent	Diffuse infiltration, shiny skin, nodules	Symmetrical	Negative	Numerous

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Clinical Features

A. Skin Lesions

- Hypopigmented or erythematous macules/plaques/nodules
- Loss of sensation (touch, pain, temperature) over lesions
- Dryness, loss of sweating, and hair loss over affected area

B. Nerve Involvement

- Peripheral nerve thickening (e.g., ulnar, common peroneal, posterior auricular)
- Sensory loss → burns and ulcers
- Motor weakness → claw hand, foot drop

C. Systemic Features (in lepromatous type)

- Nasal stuffiness, epistaxis
- Loss of eyebrows (madarosis)
- Gynecomastia, infertility (testicular atrophy)
- Ocular involvement → lagophthalmos, keratitis

Investigations

Test	Findings
Slit-skin smear	Demonstrates acid-fast bacilli (Ziehl-Neelsen or Fite stain)
Skin biopsy	Histopathological confirmation
Lepromin test	Assesses host immunity; positive in TT, negative in LL
Nerve biopsy (if needed)	In indeterminate cases

Reactions

Type	Mechanism	Clinical Features	Management
Type 1 (Reversal Reaction)	Type IV hypersensitivity	Acute inflammation of existing lesions, neuritis	Prednisolone
Type 2 (Erythema Nodosum Leprosum – ENL)	Immune complex mediated	Tender nodules, fever, malaise, neuritis	Thalidomide (if available) or corticosteroids

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Management

Multidrug Therapy (MDT) – WHO Regimen

Type	Drugs	Duration
Paucibacillary (PB) (≤5 lesions, smear negative)	Rifampicin 600 mg monthly (supervised) + Dapsone 100 mg daily (self-administered)	6 months
Multibacillary (MB) (>5 lesions or smear positive)	Rifampicin 600 mg monthly + Clofazimine 300 mg monthly + Dapsone 100 mg daily + Clofazimine 50 mg daily	12 months

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Supportive Management

- **Physiotherapy**: Prevent deformities
- **Protective footwear**
- **Reconstructive surgery** for deformities
- **Health education**: Avoid stigma, early diagnosis, adherence to MDT

Complications

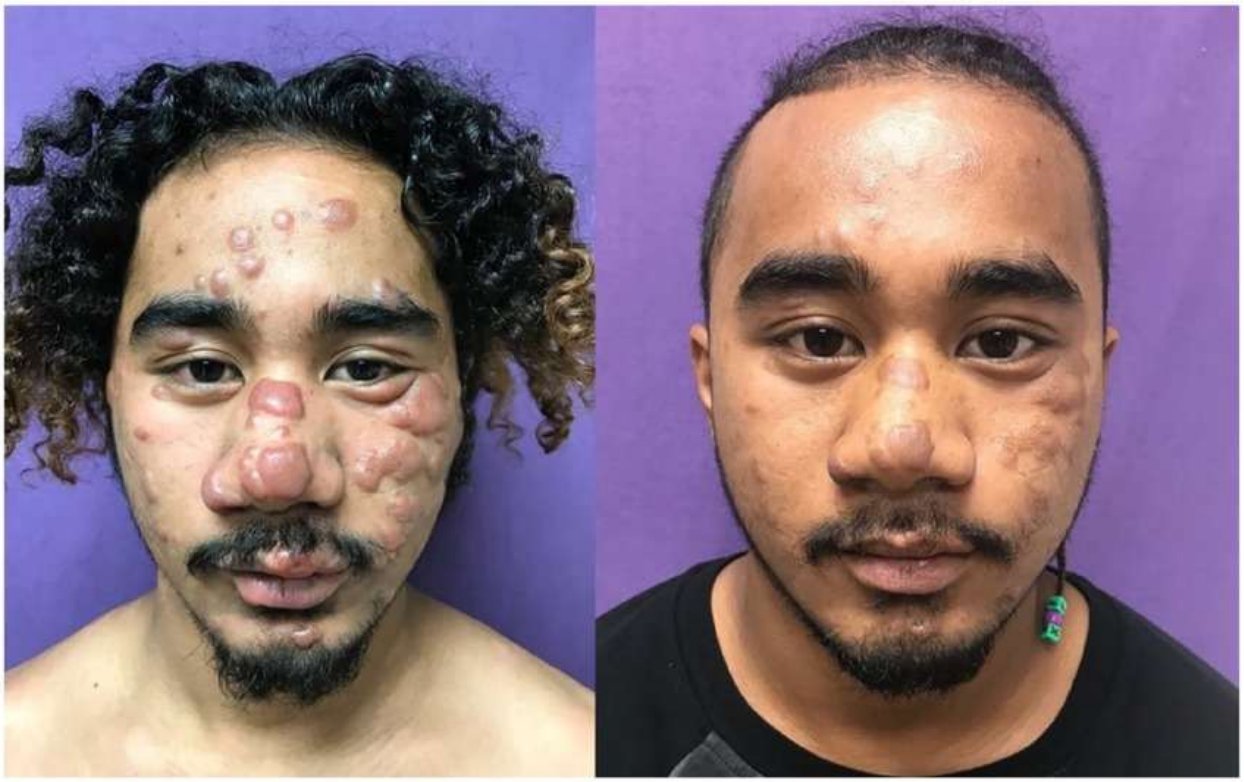
- Neuritis and deformities (claw hand, foot drop)
- Chronic ulcers
- Blindness (ocular involvement)
- Secondary infections

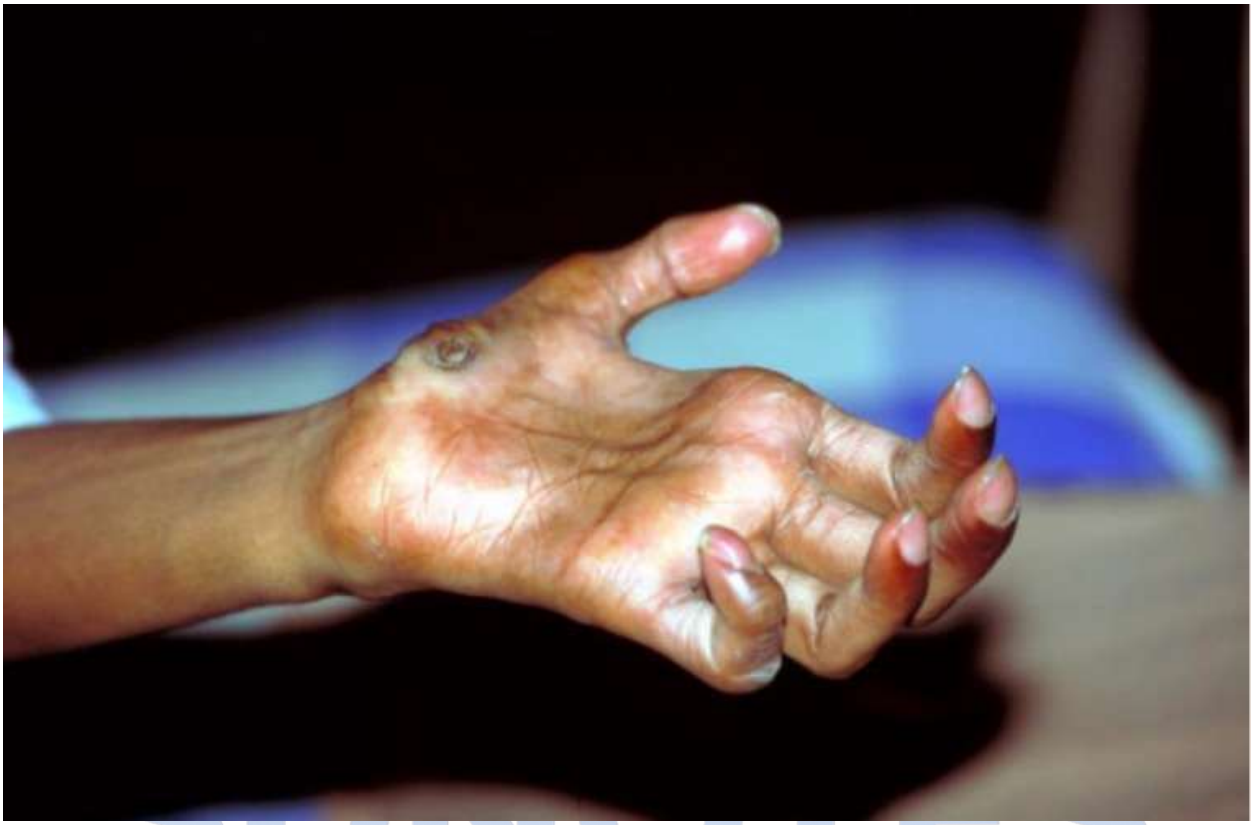
Prevention and Control

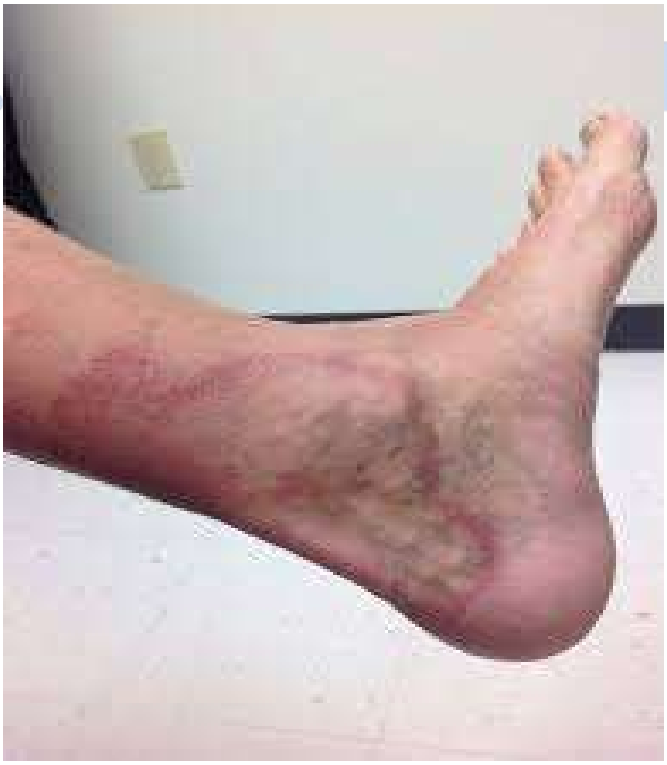
- Early detection and MDT of all cases
- Contact tracing and surveillance
- BCG vaccination offers partial protection
- Health education and anti-stigma campaigns

Prognosis

- **Good** with early diagnosis and complete MDT
- **Poor** if untreated — leads to irreversible nerve damage and deformities







FOR NOTES -

SYPHILIS

Definition

Syphilis is a **chronic, systemic, sexually transmitted infection (STI)** caused by the **spirochete *Treponema pallidum***.
It can affect **skin, mucous membranes, cardiovascular, neurological, and other organs** if untreated.

Causative Organism

- *Treponema pallidum* (subspecies *pallidum*)
- A **thin, motile, spiral-shaped spirochete** visible by **dark-field microscopy**.

Mode of Transmission

1. **Sexual contact** (most common)
2. **Vertical transmission** (mother to fetus → congenital syphilis)
3. **Blood transfusion** (rare, due to screening)
4. **Direct inoculation** (accidental exposure in healthcare settings)

Incubation Period

- **10 to 90 days** (average **21 days**)

Classification / Stages

Stage	Clinical Features	Infectivity	Comments
Primary Syphilis	Single painless ulcer (**chancre**) at site of inoculation, firm base, clean edge, non-tender inguinal lymphadenopathy	Highly infectious	Heals spontaneously in 3–6 weeks
Secondary Syphilis	Systemic spread → **maculopapular rash** (esp. palms and soles), **mucous patches**, **condyloma lata**, generalized lymphadenopathy	Highly infectious	Occurs 6–8 weeks after chancre
Latent Syphilis	No symptoms, positive serology	Early latent: potentially infectious; Late latent: non-infectious	May last for years
Tertiary Syphilis	**Gummatous lesions**, **cardiovascular (aortitis, aneurysm)**, **neurosyphilis (tabes dorsalis, general paresis)**	Non-infectious	Occurs after years of latency

Congenital Syphilis

Type	Features
Early (≤2 yrs)	Rhinitis ("snuffles"), rash, hepatosplenomegaly, bone changes
Late (>2 yrs)	Hutchinson's triad – **interstitial keratitis, notched incisors, 8th nerve deafness**

Risk Factors

Category	Examples
Behavioral	Multiple sexual partners, unprotected intercourse, MSM (men who have sex with men)
Medical	Presence of other STIs (esp. HIV)
Socioeconomic	Low education, poor access to healthcare
Perinatal	Infected mother → congenital syphilis

Investigations

Test Type	Examples	Remarks
Direct Detection	Dark-field microscopy, Direct fluorescent antibody test	For early lesions
Non-treponemal tests	**VDRL**, **RPR**	Used for screening and monitoring treatment response
Treponemal tests	**FTA-ABS**, **TPHA**	Confirmatory; remains positive for life

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Management

Stage	Drug of Choice	Regimen
Primary, Secondary, Early latent	**Benzathine Penicillin G**	2.4 million units IM single dose
Late latent or Tertiary (without neurosyphilis)	**Benzathine Penicillin G**	2.4 million units IM weekly × 3 weeks
Neurosyphilis	**Aqueous crystalline Penicillin G**	18–24 million units/day IV × 10–14 days
Penicillin allergy (non-pregnant)	**Doxycycline 100 mg** orally twice daily × 14 days	Alternative regimen

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Follow-up

- Repeat **VDRL titers** at **3, 6, 12 months** after treatment.
- Fourfold fall in titer = adequate response.
- HIV testing recommended for all patients.

Complications

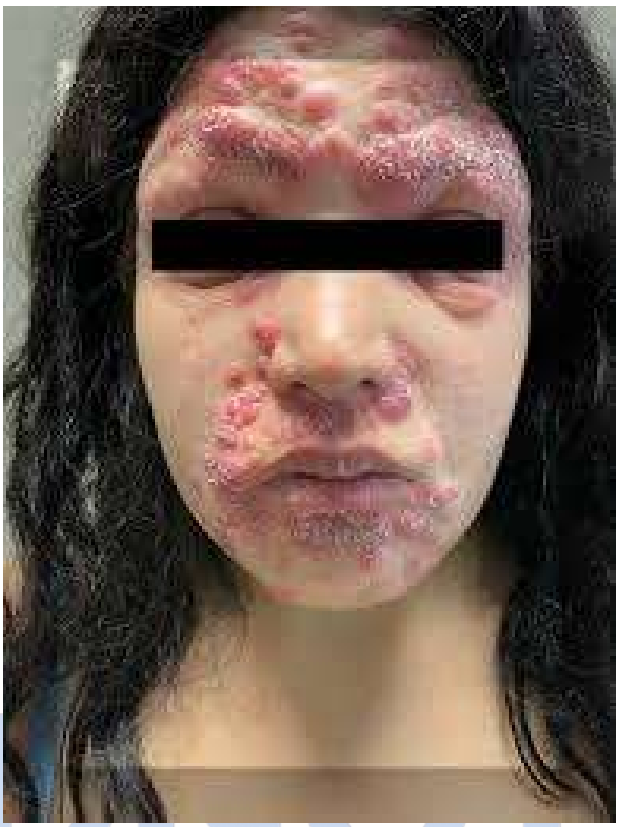
1. Cardiovascular syphilis – aortitis, aneurysm
2. Neurosyphilis – meningitis, tabes dorsalis, general paresis
3. Gummatous lesions (skin, bone, liver)
4. Congenital syphilis in neonates

Prevention

- Safe sexual practices (condom use)
- Routine antenatal screening
- Early detection and treatment of contacts
- Health education programs

Summary

Stage	Lesion	Infectivity	Treatment
Primary	Chancre	High	Benzathine Penicillin G 2.4 MU IM single dose
Secondary	Rash, mucous patches	High	Same regimen
Latent	No lesion	Variable	2.4 MU weekly × 3
Tertiary	Gummas, neuro, cardio	None	Based on involvement



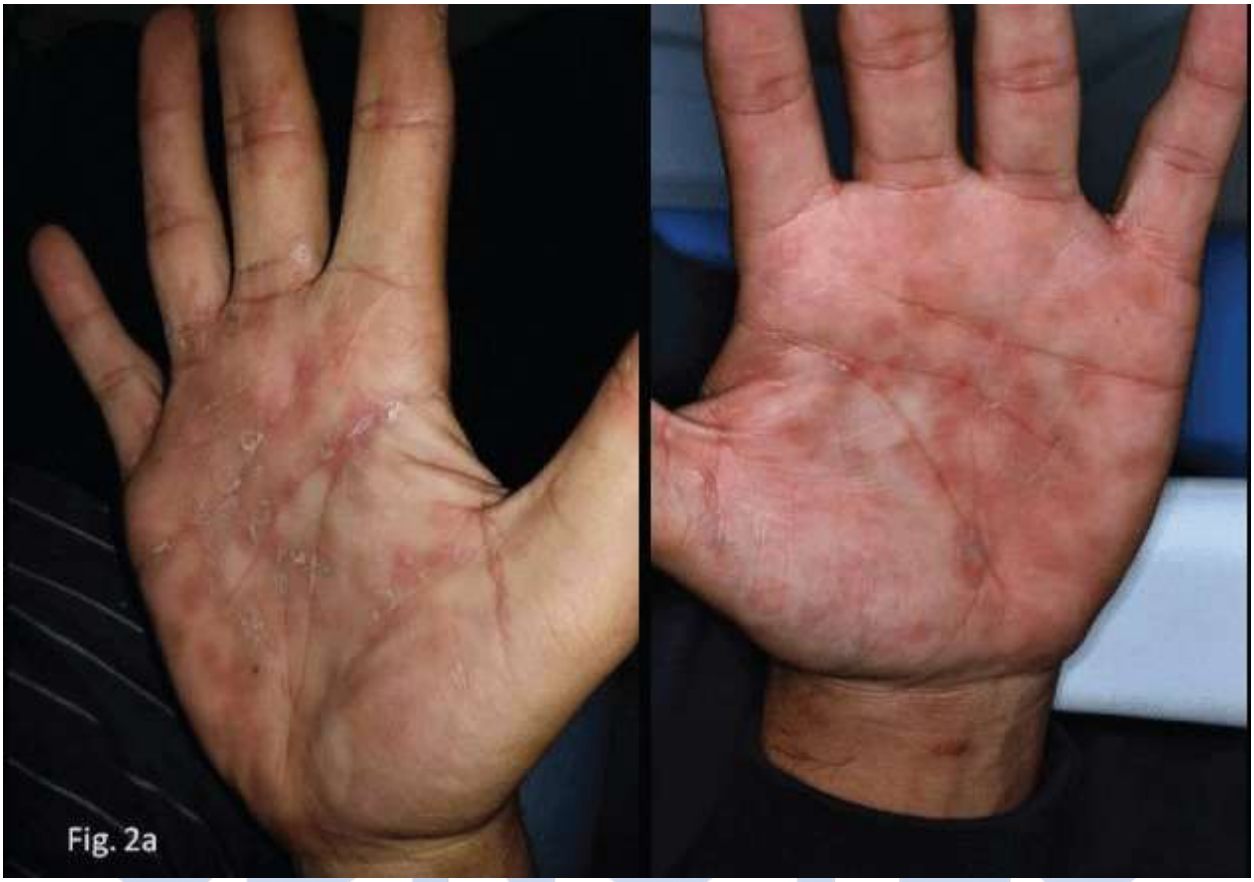


Fig. 2a

FOR NOTES -

Vesiculobullous Disorders

Definition

Vesiculobullous disorders are a group of skin diseases characterized by **vesicles (≤0.5 cm)** and **bullae (>0.5 cm)** — fluid-filled blisters resulting from separation within or beneath the epidermis.

Classification

Level of Blister Formation	Examples
Intraepidermal (within epidermis)	Pemphigus vulgaris, Pemphigus foliaceus, Hailey-Hailey disease
Subepidermal (below epidermis)	Bullous pemphigoid, Dermatitis herpetiformis, Epidermolysis bullosa, Linear IgA disease

Important Types

1. Pemphigus Vulgaris

- **Autoimmune** blistering disease with **IgG autoantibodies** against **desmoglein-3 (and sometimes desmoglein-1)**.
- **Blisters form intraepidermally**, leading to easy rupture.

Clinical features:

- Flaccid blisters that rupture easily → **erosions and crusts**
- Common sites: **oral mucosa**, trunk, scalp
- **Nikolsky's sign**: Positive (skin shears on gentle pressure)
- **Asboe-Hansen sign**: Positive (extension of blister on pressure)

Investigations:

- **Tzanck smear** → Acantholytic cells
- **Direct Immunofluorescence (DIF)** → IgG and C3 in a "fish-net" pattern

Treatment:

Stage	Drugs
Initial control	Systemic corticosteroids (prednisolone)
Maintenance	Immunosuppressants – Azathioprine, Mycophenolate mofetil
Refractory cases	Rituximab, IVIG, Plasmapheresis

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2. Bullous Pemphigoid

- **Autoimmune**, with antibodies against **BP180 and BP230** (hemidesmosomal proteins).
- **Subepidermal** blister → **tense, non-rupturing bullae**.

Clinical features:

- Elderly patients
- Tense bullae on normal or erythematous base
- Oral lesions less common
- **Nikolsky's sign negative**

Investigations:

- **DIF:** Linear deposition of IgG and C3 along basement membrane

Treatment:

- Systemic or topical steroids
- Tetracycline + Nicotinamide (for mild cases)
- Immunosuppressants (Azathioprine, Methotrexate)

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3. Dermatitis Herpetiformis

- **Autoimmune**, associated with **gluten-sensitive enteropathy (celiac disease)**
- **IgA** deposits at dermal papillae

Clinical features:

- Grouped vesicles and papules with intense **itching**
- Sites: Elbows, knees, buttocks, scalp
- Excoriations due to scratching

Investigations:

- **DIF:** Granular IgA at dermal papillae

Treatment:

- **Dapsone** – drug of choice
- Gluten-free diet

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4. Linear IgA Bullous Dermatosis

- Autoantibodies of **IgA** type against basement membrane zone antigens
- **Subepidermal** blister

Clinical features:

- "String of pearls" arrangement of vesicles
- Can affect both children (chronic bullous disease of childhood) and adults

Treatment:

- Dapsone
- Sulfapyridine
- Mild cases – topical steroids

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5. Epidermolysis Bullosa (EB)

- **Genetic** blistering disorder due to mutations in keratin, laminin, or collagen genes.

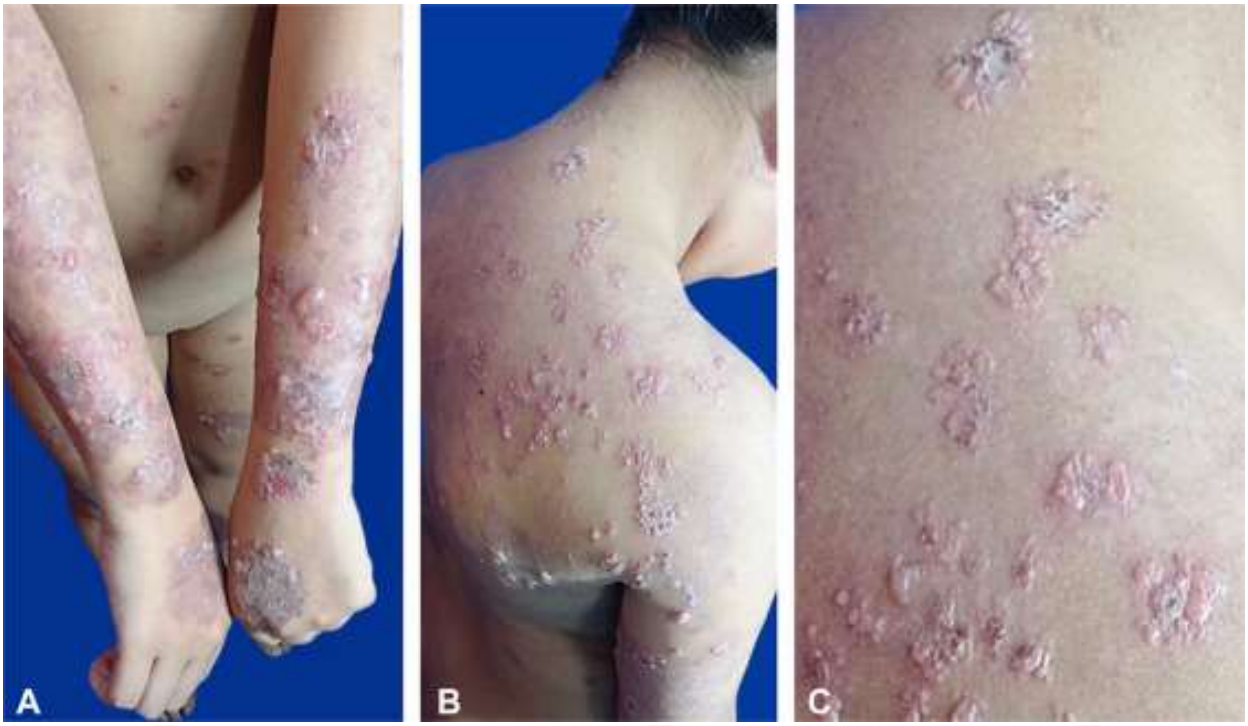
Type	Level of split	Notes
EB Simplex	Intraepidermal	Mild, blisters on trauma
Junctional EB	Within lamina lucida	Severe, may be fatal in infancy
Dystrophic EB	Below lamina densa	Scarring, milia formation

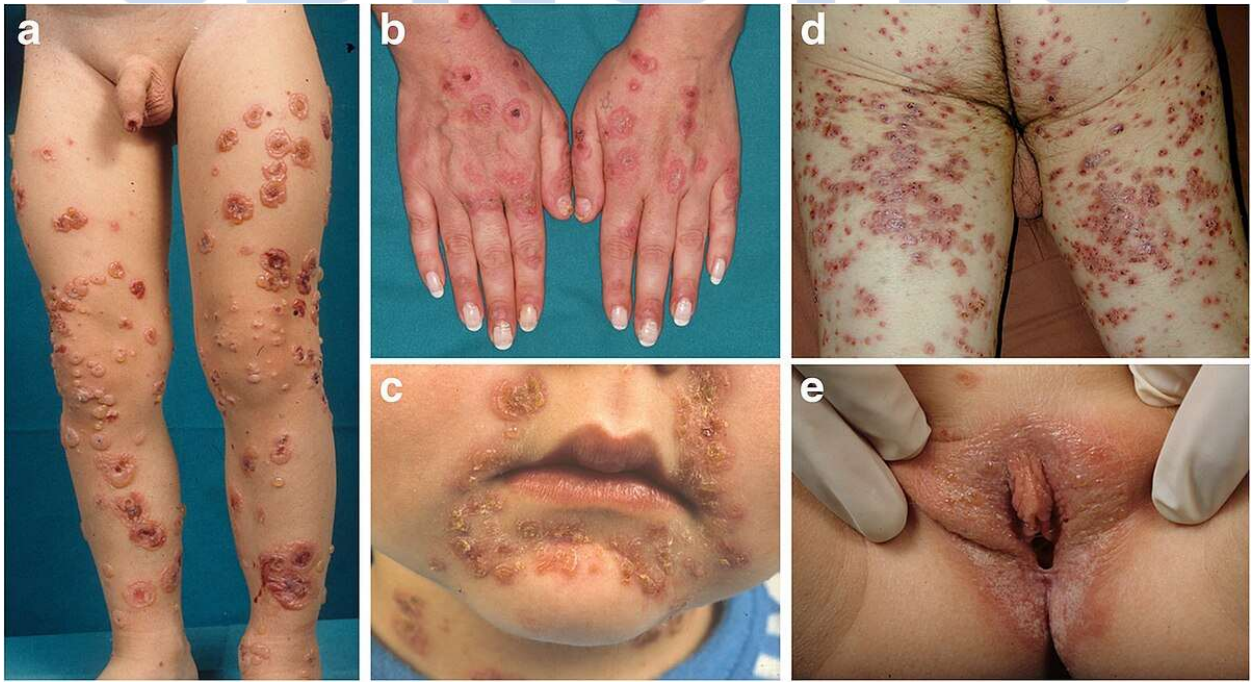
Management:

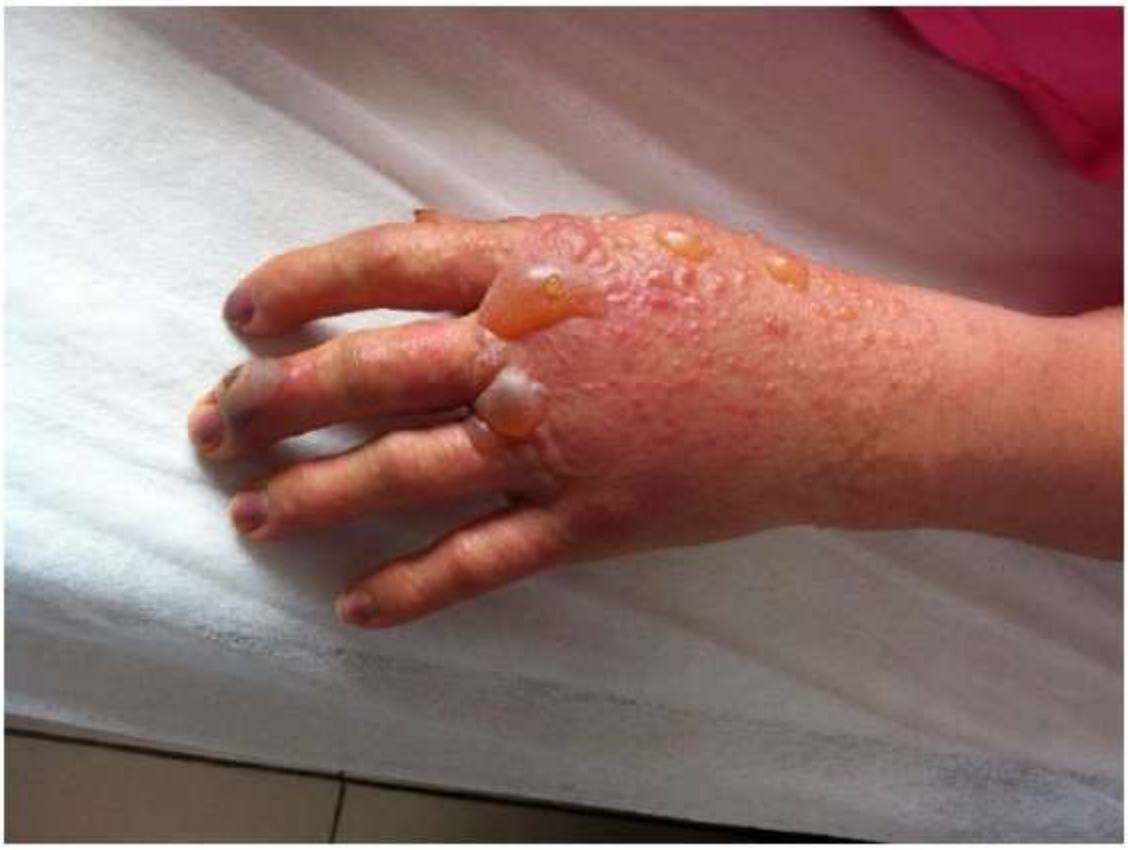
- Supportive: wound care, infection prevention
- Genetic counseling
- Avoid trauma

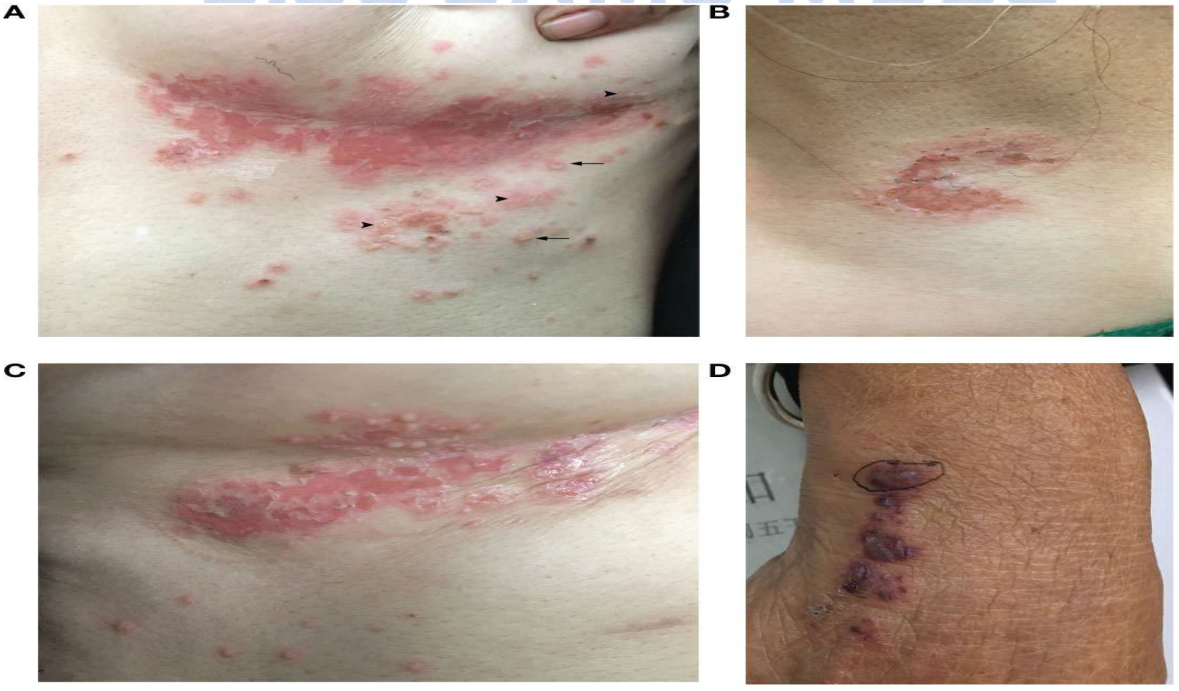
ALL OVER -

Disease	Level of Blister	Antibody Target	Nikolsky Sign	Main Treatment
Pemphigus vulgaris	Intraepidermal	Desmoglein-3 (±1)	Positive	Steroids + Immunosuppressants
Bullous pemphigoid	Subepidermal	BP180, BP230	Negative	Steroids ± Immunosuppressants
Dermatitis herpetiformis	Subepidermal	IgA at dermal papillae	Negative	Dapsone + Gluten-free diet
Linear IgA disease	Subepidermal	Basement membrane IgA	Negative	Dapsone
Epidermolysis bullosa	Genetic (varies)	Structural proteins	—	Supportive









CUTANEOUS MANIFESTATIONS OF HIV

Definition

Cutaneous manifestations are skin, hair, nail, or mucous membrane changes occurring due to the direct effect of HIV infection, secondary infections, or drug reactions.
They often provide **early clues** to HIV infection and correlate with the **degree of immunosuppression (CD4 count)**.

Types / Classification

Category	Examples	Comments
1. Infectious Dermatoses	*Bacterial*: Impetigo, folliculitis, abscesses (often by Staph.) *Viral*: Herpes simplex, Herpes zoster, Molluscum contagiosum, HPV warts *Fungal*: Oral candidiasis, Tinea infections, Seborrheic dermatitis, Cryptococcosis	Common in all stages, severity increases as CD4 falls
2. Non-infectious (Inflammatory) Dermatoses	Seborrheic dermatitis, Psoriasis, Xerosis, Atopic dermatitis, Pruritic papular eruption (PPE)	PPE is very common; associated with CD4 <200
3. Neoplastic Lesions	Kaposi's sarcoma, Non-Hodgkin lymphoma, Squamous cell carcinoma	Opportunistic tumors due to immunosuppression
4. Drug Reactions	Maculopapular rash, Stevens-Johnson syndrome, Toxic epidermal necrolysis (TEN), Fixed drug eruption	Due to ART drugs, cotrimoxazole, anti-TB drugs
5. Other Manifestations	Nail changes, Oral hairy leukoplakia, Hair loss (diffuse or patchy), Pigmentation	May reflect nutritional or metabolic effects of HIV

Common Lesions and Their Features

Lesion	Typical Presentation	CD4 Count Correlation	Remarks
Seborrheic dermatitis	Greasy scales on scalp, face, chest	Any stage (more severe with low CD4)	Common early sign
Pruritic papular eruption (PPE)	Intensely itchy papules on trunk and limbs	<200 cells/mm³	Most frequent HIV-related dermatosis
Oral candidiasis	White curd-like plaques on tongue, buccal mucosa	<400 cells/mm³	Marker of progression
Herpes zoster	Painful vesicular eruption along dermatome	Any stage	Often early manifestation
Kaposi's sarcoma	Violaceous macules, papules, nodules	<200 cells/mm³	AIDS-defining illness
Molluscum contagiosum	Multiple, large, atypical lesions	<100 cells/mm³	Recalcitrant lesions common
Drug eruptions	Maculopapular rash or severe reactions	Any stage (esp. early ART)	Often due to NNRTIs, sulfa drugs

Risk Factors

- Low **CD4 count (<200/mm³)**
- Poor **ART adherence**
- **Malnutrition** and poor hygiene
- Co-infection with **tuberculosis** or **hepatitis viruses**
- Use of **multiple drugs** (polypharmacy)
- **Unprotected sexual exposure** and repeated infections

Diagnosis

- **Clinical examination** — type, distribution, morphology of lesions
- **CD4 count** estimation
- **Microbiological tests**: KOH mount, Gram stain, culture
- **Histopathology** for neoplastic lesions (e.g., Kaposi's sarcoma)
- **Drug history** to rule out adverse drug reactions

Management

Aspect	Approach
General	Start or optimize **ART** (most skin lesions improve with immune reconstitution)
Infectious lesions	Treat specific infection: antibiotics, antifungals, antivirals as per etiology
Inflammatory conditions	Topical corticosteroids, emollients, antihistamines for itch
Neoplastic lesions	Chemotherapy, radiotherapy, or HAART-induced regression (Kaposi's sarcoma)
Drug reactions	Stop offending drug, give systemic steroids if severe, supportive care
Counseling & Prevention	Patient education on hygiene, early reporting, ART adherence

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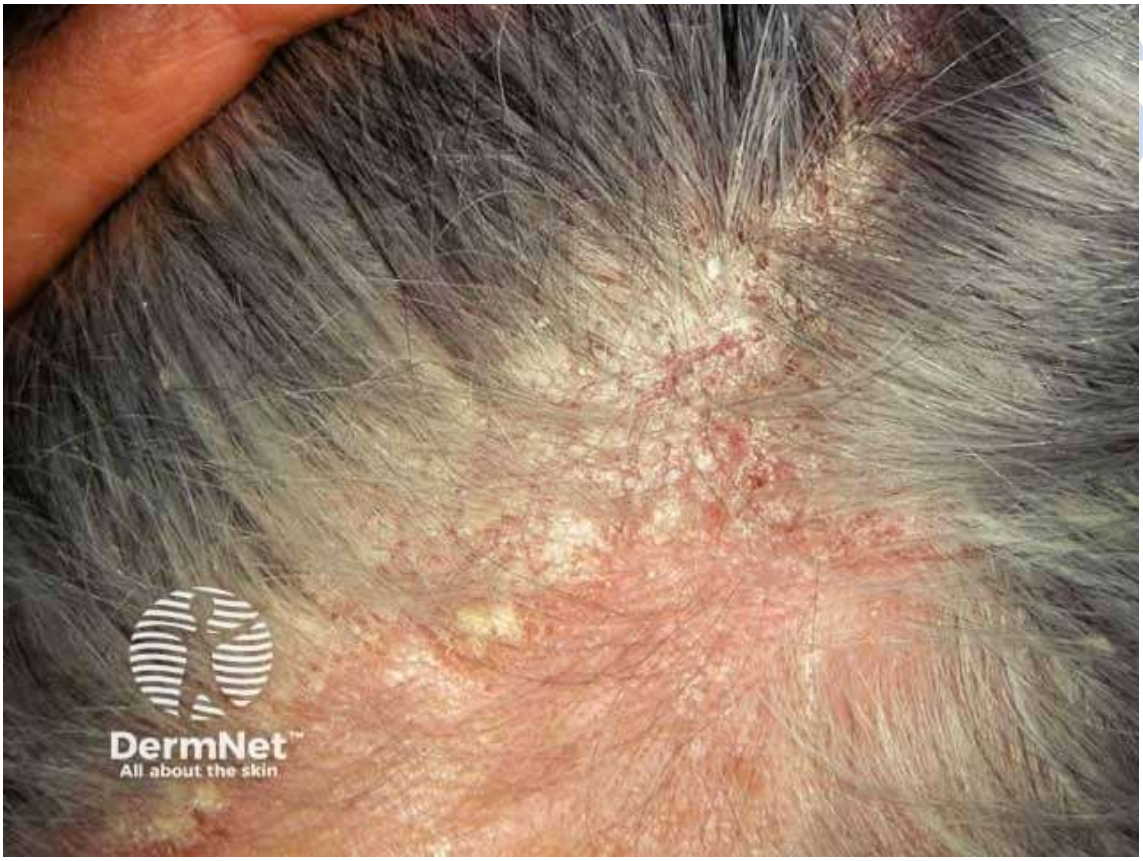
Prognostic Importance

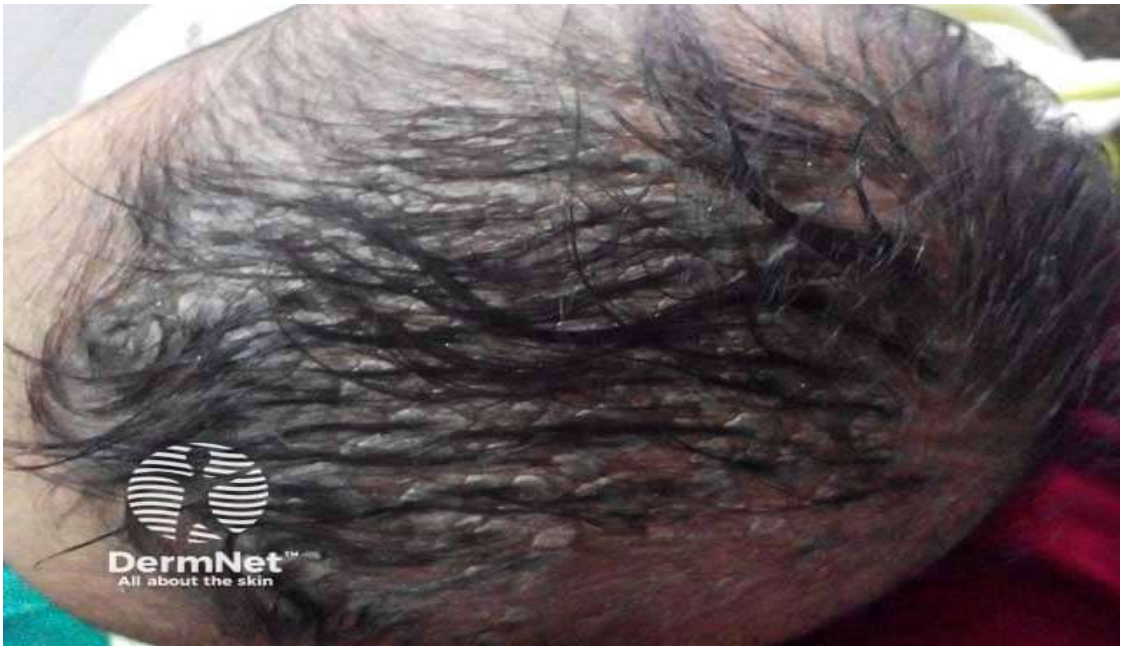
- Skin lesions often **reflect immune status**
- **Reappearance or persistence** may suggest **ART failure or non-adherence**
- Resolution indicates **immune reconstitution**

Summary Points

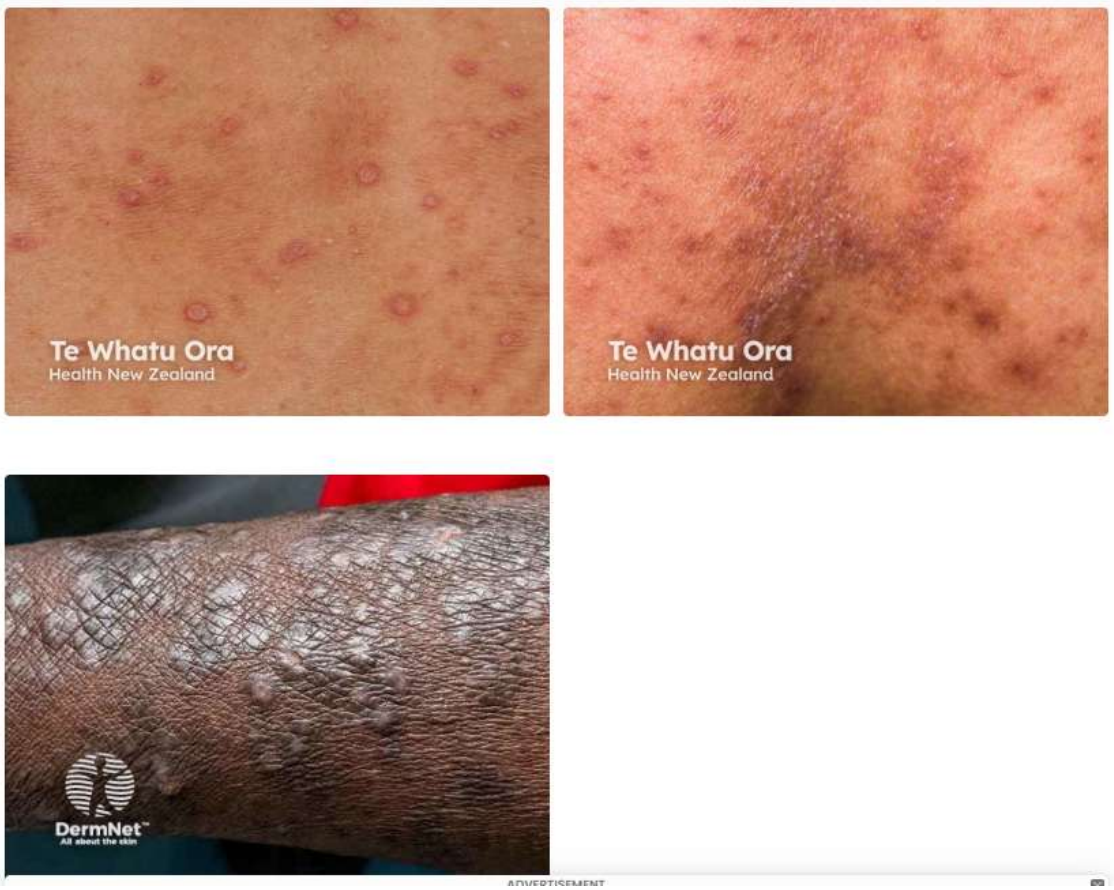
- Cutaneous manifestations are **early and common** in HIV infection.
- Most common: **Seborrheic dermatitis, PPE, oral candidiasis**.
- Severity correlates with **CD4 count**.
- **ART initiation and adherence** are crucial for control.

Seborrheic dermatitis



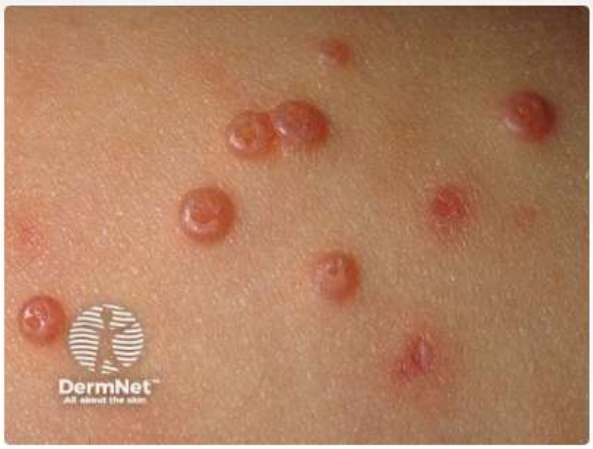


Pruritic papular eruption (PPE)

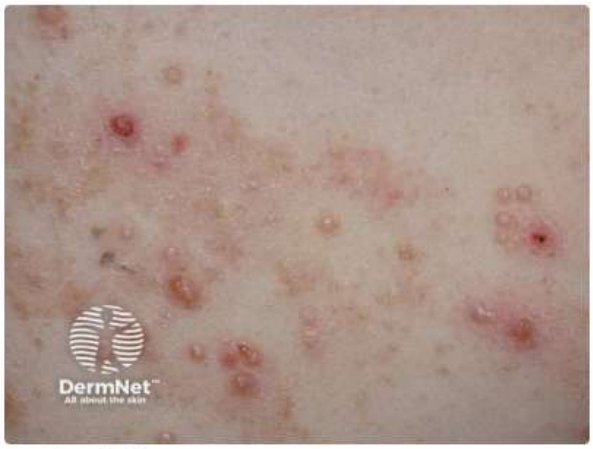


Molluscum contagiosum

Shiny umbilicated papules



Typical umbilicated papules



Associated with eczema

DERMA CLINICAL POSTING VIVA – SHORT NOTES

1. Acne Vulgaris

1. **Cause:** Increased sebum + P. acnes infection
2. **Lesions:** Comedones, papules, pustules
3. **Common site:** Face, chest, back
4. **Aggravating factor:** Hormonal, oily cosmetics
5. **Treatment:** Topical retinoids, benzoyl peroxide
6. **Complication:** Scarring, post-inflammatory pigmentation
7. **Grading:** Mild / Moderate / Severe

2. Vitiligo

1. **Definition:** Acquired depigmentary disorder
2. **Cause:** Autoimmune destruction of melanocytes
3. **Lesions:** Milky white macules
4. **Sites:** Face, hands, genitalia
5. **Type:** Focal, segmental, generalized
6. **Test:** Wood's lamp – chalky white fluorescence
7. **Treatment:** Topical steroids, phototherapy (NB-UVB)

3. Eczema (Dermatitis)

1. **Definition:** Inflammatory skin reaction with itching
2. **Types:** Acute, subacute, chronic
3. **Lesions:** Erythema, vesicles, oozing, crusting
4. **Example:** Atopic dermatitis, contact dermatitis
5. **Itch:** Hallmark feature
6. **Treatment:** Emollients, topical steroids, antihistamines
7. **Complication:** Secondary infection

4. Psoriasis

1. **Definition:** Chronic inflammatory papulosquamous disorder
2. **Lesions:** Silvery scales on erythematous plaques
3. **Sites:** Elbows, knees, scalp, lumbosacral area
4. **Auspitz sign:** Pinpoint bleeding on scale removal
5. **Koebner phenomenon:** Lesions at trauma site
6. **Treatment:** Topical steroids, calcipotriol, phototherapy
7. **Type:** Plaque, guttate, pustular, erythrodermic

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5. Leprosy

1. **Cause:** *Mycobacterium leprae*
2. **Transmission:** Prolonged close contact
3. **Lesions:** Hypopigmented patch with loss of sensation
4. **Nerve involved:** Ulnar, posterior auricular, peroneal
5. **Type:** Tuberculoid to Lepromatous spectrum
6. **Test:** Slit-skin smear (AFB +)
7. **Treatment:** MDT (Rifampicin, Clofazimine, Dapsone)

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6. Syphilis

1. **Causative agent:** *Treponema pallidum*
2. **Primary lesion:** Painless indurated chancre
3. **Secondary:** Rash on palms & soles
4. **Tertiary:** Gummatous, cardiovascular, neuro
5. **Test:** VDRL / RPR (screening)
6. **Confirmatory:** TPHA / FTA-ABS
7. **Treatment:** Benzathine Penicillin G

---

7. Non-Syphilitic STDs

1. **Examples:** Gonorrhea, Chancroid, LGV, Granuloma inguinale, Genital warts, Herpes
2. **Gonorrhea:** Purulent urethral discharge
3. **Chancroid:** Painful ulcer with ragged edges
4. **LGV:** Painless ulcer → buboes
5. **Genital warts:** HPV 6, 11 – cauliflower growth
6. **Treatment:** Antibiotics or antivirals per etiology
7. **Prevention:** Safe sex, partner treatment

8. Vesiculobullous Disorders

1. **Definition:** Group of blistering skin diseases
2. **Examples:** Pemphigus vulgaris, Bullous pemphigoid
3. **Pemphigus vulgaris:** Flaccid bullae, oral erosions
4. **Tzanck smear:** Acantholytic cells
5. **Nikolsky sign:** Positive in pemphigus
6. **Bullous pemphigoid:** Tense bullae, elderly, subepidermal
7. **Treatment:** Systemic steroids, immunosuppressants

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9. Cutaneous Manifestations of HIV

1. **Cause:** Immunosuppression due to HIV
2. **Infectious lesions:** Candidiasis, herpes, tinea
3. **Non-infectious:** Seborrheic dermatitis, PPE, psoriasis
4. **Neoplastic:** Kaposi's sarcoma
5. **Drug reaction:** Common due to ART
6. **Indicator:** Correlates with CD4 count
7. **Treatment:** ART + specific management

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