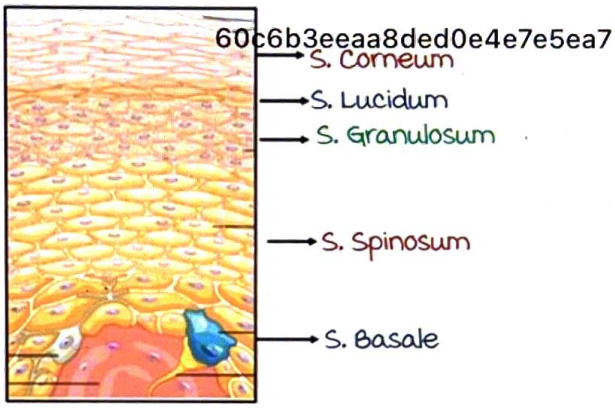
BASICS OF DERMATOLOGY : PART - 1

Skin : introduction

00:00:36

Weight of skin : 4 to 5 kg (largest organ of body).
Total area covered by skin is 1.7 square meters.
Layers of the skin (from above downwards) : Epidermis, dermis and hypodermis.
Epidermis is composed of Stratified squamous epithelium.
Layers of epidermis : (Above to below)

1. Stratum Corneum :
   - Outermost layer.
   - Flat cells.
   - Fully Keratinized layer.
   - Anucleate (no nucleus : Dead layer).



2. Stratum Lucidum :
   - Lucidum refers to Translucency.
   - Extra layer located over palms and soles
   - Eleidin granules can be seen.
3. Stratum Granulosum :
   - Prominent granules.
     i. Keratohyalin granules :
        - Basophilic.
        - Profilaggrin protein → Cleaved → Filaggrin (active form) → Filament aggregating protein.
        - Gene for Filaggrin mutated in :
          1. Atopic dermatitis.
          2. Ichthyosis vulgaris (Fish like scales) : most common form.

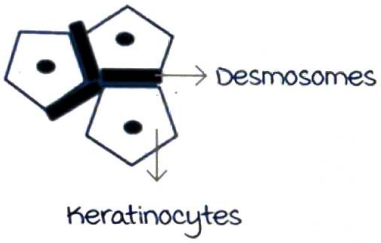
Active space

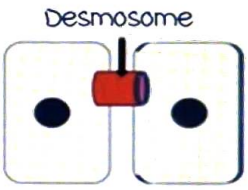
ii. Lamellar granules :

- Odland bodies / membrane coating granules / Cementosomes.
- Responsible for producing the lipid content of epidermis which contributes to the barrier function of skin.

4. Stratum Spinosum :

- Prickle cell layer.
- This layer is composed of large polygonal Keratinocytes connected by Desmosomes.
- Desmosomes on light microscopy have a spiny appearance.
- Desmosomes : Intraepidermal intercellular connections between Keratinocytes.
  Antibodies against Desmosomes : Pemphigus group of diseases.



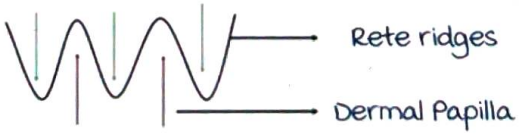


5. Stratum Basale :

- Stratum Germinativum.
- Single layer of cells.
- Columnar cells with central nucleus.

Rete ridges vs dermal papilla 00:12:08

- Rete ridge is projection of epidermis into dermis.
- Dermal papilla is projection of dermis to epidermis.

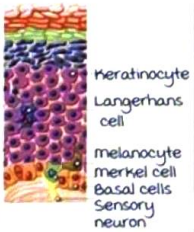
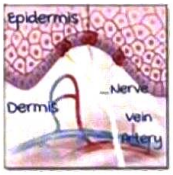


Different transitional changes (cells travelling up) :

1. Stratum Corneum, 2. Shape of cells flattened, 3. Nucleus is lost, 4. Keratin content increases, 5. Dehydration.

6. Epidermal transit time : Time taken by cells to move from Stratum basale to stratum corneum (28 days.)

Cells of epidermis :

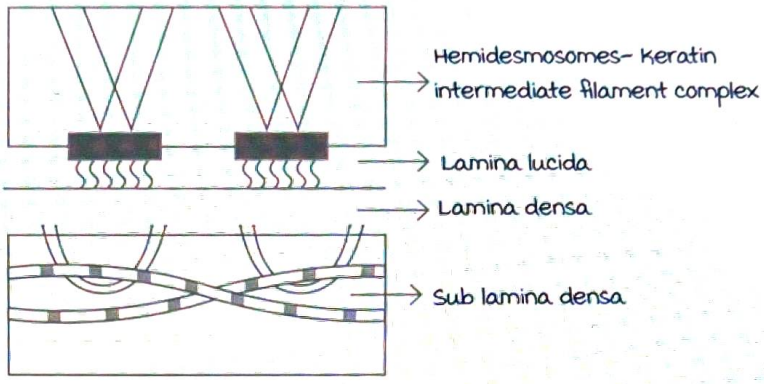
	Keratinocytes	Non-Keratinocytes		
	Keratinocytes	Langerhans cells.	Melanocytes.	Merkel cells.
Nature of cell.	Most abundant cells in epidermis (90%).	Dendritic cell.	Dendritic cell.	Receptor cells.
Location.		Stratum Spinosum.	Stratum Basale.	Stratum Basale.
Origin.	Ectoderm.	Bone marrow (macrophages).	Neural crest.	Ectoderm.
Components	KIF (Keratin Intermediate Filaments).	Birbeck granules. Tennis Racquet appearance on electron microscopy.	Melanosomes.	Neuro-secretory granules.
Function.		Antigen Presenting Cell (APC).	Melanin pigment producing cell.	Mechanoreceptors (Touch) : Slow adapting, low threshold receptors.
Example of a Disease.		Langerhans cell histiocytosis	Melanoma.	Merkel cell carcinoma.
Markers.		S100, CD1a, [illegible] (Langerin) : most specific marker.	S100, HMB45, [illegible]	Cytokeratin-20
		  Birbeck granules	 Melanosomes	

Active space

Epidermal melanin unit : 1 Melanocyte transfer its melanosomes to 36 Keratinocytes. It is responsible for unifrom skin colour.

Dermal-epidermal junction

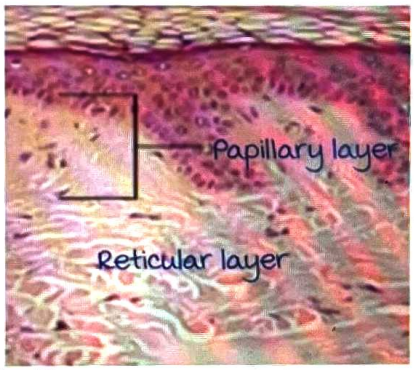
00:25:58



- Also known as Basement membrane zone.
- Specialized junction connecting the epidermis and dermis.
- Parts :
  1. Hemidesmosomes – Keratin intermediate filament complex (K5, K14).
  2. Lamina lucida.
  3. Lamina densa.
  4. Sub Lamina densa.

Dermis :

- Papillary dermis is 1/10th of dermis : Loose connective tissue.
- Reticular dermis is 9/10th of dermis : Dense, irregular connective tissue.



60c6b3eeaa8ded0e4e7e5ea7

Content of Dermis :

- Connective tissue.
- Cells :
  a. Fibroblasts : produce connective tissue.
  b. Macrophages.
  c. Lymphocytes.

Active space

d. Ground substance (GAG : Glycosaminoglycans).
e. Blood vessels.
f. Nerve receptors.

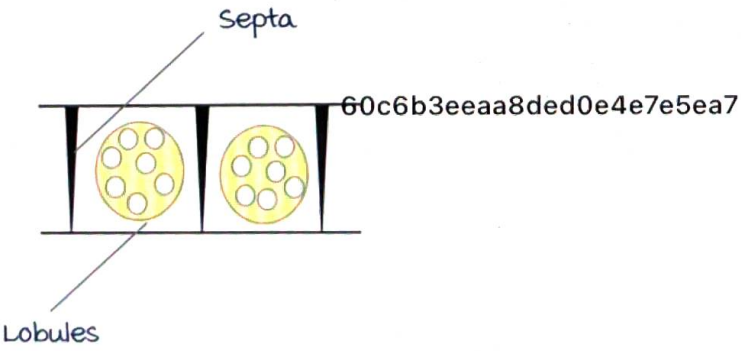
- Appendages of skin :
  i. Hair follicle.
  ii. Sweat glands.
  iii. Sebaceous glands.
  iv. Nail.

Connective tissue :

1. Collagen fibers :
   - 70% dermis.
   - Responsible for tensile strength.
   - MC type of collagen in skin : Type 1.
   - 2nd MC type of collagen in skin : Type 3.
   - MC type of collagen in cartilage : Type 2.
   - Type I collagen : Osteogenesis imperfecta.
2. Elastic fibers :
   - Elastin.
   - Elastin associated microfibrils (Fibrillin/Fibulin).
     Fibrillin gene involved in marfan's syndrome.

Hypodermis 00:34:25

- Subcutaneous tissue.
- Function : Insulation (Subcutaneous fat).
  Inflammation of Subcutaneous fat (panniculus) :
  Panniculitis.
- 2 important components of SC fat :
  1. Septa made of Fibrous tissue.
  2. Lobules made of Adipocytes.



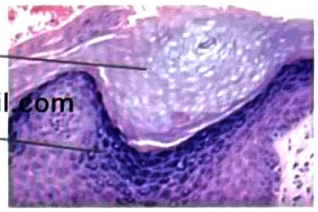
Active space

Dermato pathological terms

00:36:16

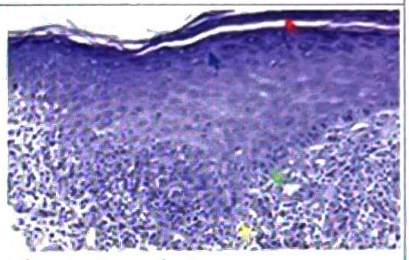
Stratum Corneum :

- Hyperkeratosis : Pathological thickening.
- Parakeratosis : Retention of nucleus



Stratum Granulosum :

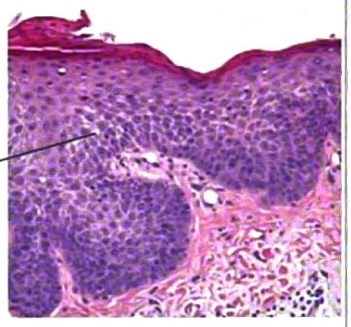
- Wedge shaped hyper granulosis : Lichen Planus.
- Absent/decreased granular layer : Psoriasis vulgaris, Ichthyosis vulgaris.



Hyperkeratosis
Thickened granular layer
Jagged outline of epidermis
Lymphocytes obscuring the dermal : epidermal infiltrate

Stratum Spinosum :

- Balloon degeneration (intracellular edema) : HSV infections.
- Spongiosis (intercellular edema between cells) : Acute Eczema.
- Acanthosis → Thickening of Stratum Spinosum.
- Acantholysis → Loss of attachment between Keratinocytes.



- Acantholysis

Dyskeratosis :

- Abnormal premature Keratinization of individual Keratinocytes : Darier disease, Bowen's disease, Squamous cell Carcinoma.

Spine = Acantha.

- Acanthosis → Thickening of Stratum Spinosum.
- Acantholysis → Loss of attachment between Keratinocytes → Desmosomes rupture → Cells transform to circular cells : Acantholytic cells/Tzanck cells with central hypochromatic nucleus (Perinuclear halo).

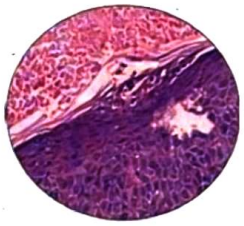
Active space

- Causes :
  1. Autoimmune : Pemphigus group.
  2. Infection : Bacterial/Viral.
     60c6b3eeaa8ded0e4e7e5ea7
     Bacterial : Bullous impetigo, Staphylococcus Scalded Skin Syndrome (SSSS).
     Viral : HSV infection.
  3. Genetic : Hailey Hailey disease, Darier disease.

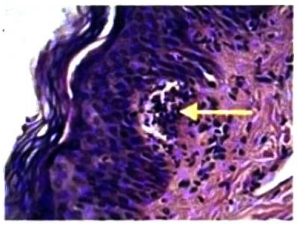
Micro abscess

00:46:18

- Microscopic small collection of cells.
- Classified Based on which type of cell accumulates :
  1. Neutrophils :
     Stratum corneum : Munro's micro abscess (Psoriasis).
     Stratum spinosum : Spongiform Pustule of Kogoj (Psoriasis).
     Papillary tip : Dermatitis herpetiformis.

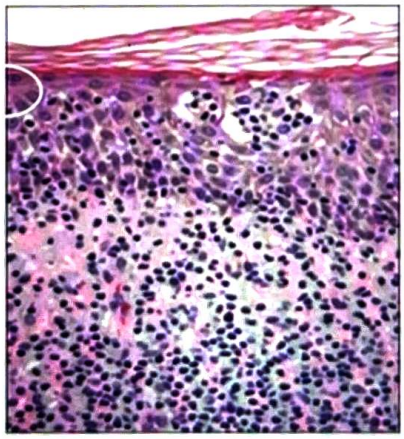


Munro's microabscess



Dermatitis herpetiformis

  2. Eosinophils : Pemphigus vegetans.
  3. Atypical T cells : Pautrier's microabscess → CTCL (Cutaneous T-Cell Lymphoma/Mycosis Fungoides).



Pautrier Microabscess

Active space

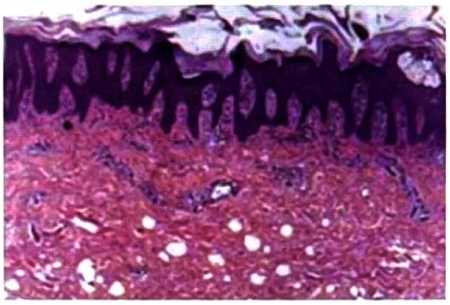
4. Rete ridges :

Projections of epidermis into dermis.

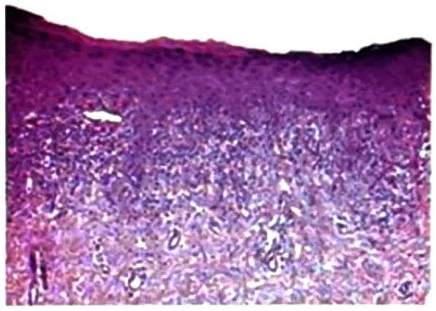
Seen in :

1. Psoriasis → Regular elongation of rete ridges.
2. Lichen Planus → Saw toothing of rete ridges.

Psoriasis



Epidermis shows psoriasiform hyperplasia with markedly elongated rete ridges.



Saw toothed Rete Ridges

60c6b3eeaa8ded0e4e7e5ea7

Active space

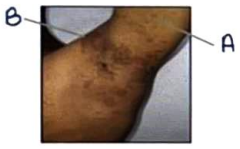
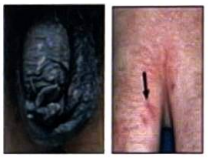
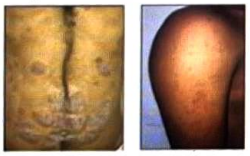
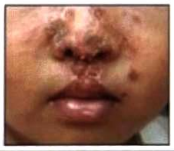
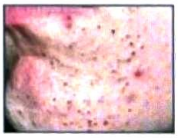
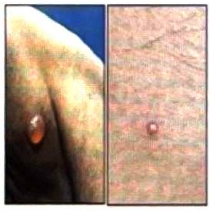
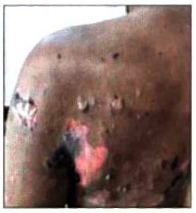
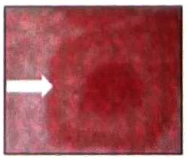
BASICS OF DERMATOLOGY : PART - 2

60c6b3eeaa8ded0e4e7e5ea7

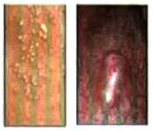
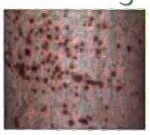
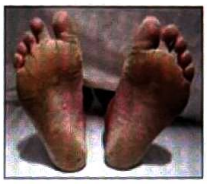
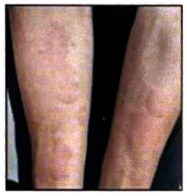
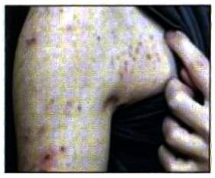
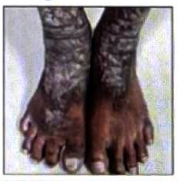
Clinical diagnosis of a disease

00:00:42

1. Morphology of the lesion.
2. Configuration of the lesion.
3. Distribution of the lesion.

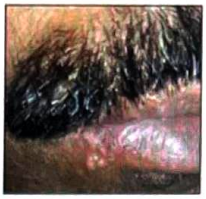
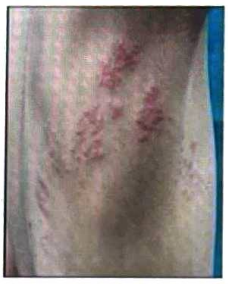
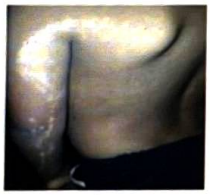
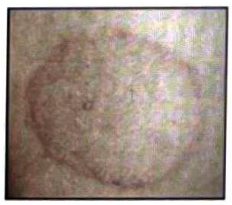
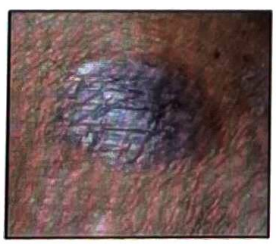
Lesion	Primary	Secondary skin lesions	Special
Description	Initial skin lesions to appear.	Modified skin lesions due to : i. Itching. ii. Treatment.	Specific to a particular disease.
Types of lesions	1. Flat non palpable skin lesions. • < 1 cm : Macule (A) • > 1 cm : Patch (B) 	1. Scale : Visible exfoliation of Stratum corneum. • Seborrheic dermatitis / dandruff • Scalp psoriasis. 	1. Burrow : Wavy, grayish white tunnel in the skin (stratum corneum) • Scabies. 
	2. Circumscribed solid raised lesions. • < 1 cm : Papule. • > 1 cm : Plaque. • > 1 cm + Depth : Nodule. 	2. Crust : Hard exudate formed due to drying of serum pus/blood on the skin surface. Eg. Non bullous impetigo : Honey colored crust 	2. Comedone : Follicular oriented lesions plugged by serum + Keratin. Open (black) or Closed (white) comedones. Eg. Acne Vulgaris. 
	3. Clear fluid filled lesion • < 1 cm : Vesicle. • > 1 cm : Bulla. 	3. Erosion : Focal/Total loss of Epidermis. Eg: Pemphigus vulgaris. 	3. Target lesion : 3 zones : i. Central zone : Dusky hue. ii. Intermediate zone : Edema. iii. Periphery : Erythema. Erythema multiforme (Palms and soles). 

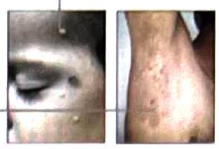
Active space

	4. Pus filled lesion. • Pin-point Pustule. • Larger collection : Abscess. 	4. Ulcer : Loss of epidermis + partial/ total loss of dermis + molecular death. 	
	5. RBC extravasation (moving out of the vessel) • 1-2 mm : Petechia. • >/= 3 mm : Purpura. • > 1 cm : Ecchymoses. 	5. Fissure : Linear deep cleft in the skin (Cracks). • Plantar Eczema. 	
	6. Wheal : Pruritic, transient plaque + Central pallor + Peripheral erythema + Itching (seen in Urticaria) 	6. Excoriation : Scratch marks. Linear or punctate abrasion of the skin due to scratching. 	
60c6b3eeaa8ded0e4e7e5ea7		7. Lichenification : Due to continuous chronic scratching → Hyperpigmentation + Thick skin + Exaggerated skin markings. 	
		8. Atrophy : Decreased/Loss of structural components of the skin. Clinically : Wrinkled appearance of skin. Topical steroid usage. 	

Active space

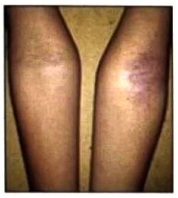
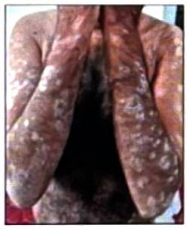
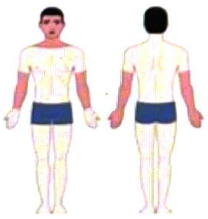
Configuration/pattern of skin lesions 00:20:26

Configuration.	Arrangement of lesion.	Seen in.
Grouped configuration. 	Vesicles arranged in a cluster.	Herpes labialis.
Dermatomal pattern. 	Vesicles arranged in a dermatomal pattern (area supplied by single nerve).	Herpes Zoster
Linear configuration. 	Resembles a line.	
Blaschkoid configuration. 	Whorled/wavy. Along the Blaschko lines.	
Annular lesions. 	Ring shaped lesions. • Centre : Inactive/clear. • Periphery : Active.	Tinea corporis (Dermatophyte).
Discoid lesion/Nummular lesion. 	Disc like lesion. • Centre + Periphery : Active.	Discoid eczema.

Discrete pattern.	Lesions individually present.	Discrete
Confluent pattern.	Lesions join each other. confluent	

Distribution

00:26:04

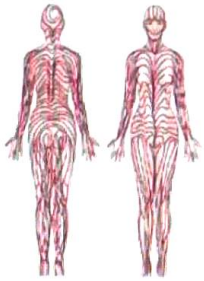
Acral distribution 	Extremities	
Flexural distribution. 	Flexor surfaces.	Atopic Dermatitis
Extensor distribution. 	Extensor surfaces.	Psoriasis.
Photodistributed lesions. 	Sun exposed areas.	Pellagra

Lines in dermatology :

1. Langers lines :
   - Also called lines of skin tension.
   - Correspond to orientation of collagen fibers in the dermis.
   - Applied : Incision put along or parallel to these lines → Healing better without scarring.

II. Blaschko lines :

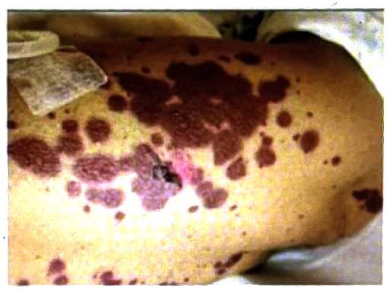
- Pathways of epidermal cell migration during embryonic development.
- Do not correspond to arteries/veins/nerves.
  a. V shaped on upper spine.
  b. S shaped on abdomen.
  c. Spiralled on scalp.
  d. Linear on lower extremities.
- Eg., Incontinentia pigmenti (X linked dominant inheritance).



Dermatological diagnosis 00:33:00

Nikolsky sign :

- Tangential pressure applied over skin → upper layers of epidermis separate from the lower layers → Skin peels off → Nikolsky's sign.

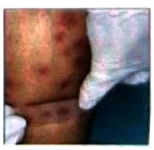


Types	True Nikolsky's sign	Pseudo Nikolsky's sign
mechanism	Acantholysis	Necrosis of keratinocytes
Examples	Pemphigus foliaceous Pemphigus vulgaris Staphylococcal scalded skin syndrome	Usually, drug induced. TEN (Toxic Epidermo Necrolysis)

Diascopy :

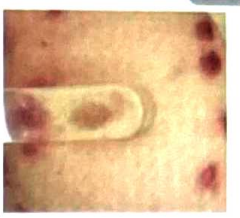
- Also called Vitro pression test.
- Glass slide→ Press the lesion.
- Uses :

I. Distinguish between erythema and purpura.

Erythema	Purpura
Redness disappears on pressure. Blanching response.	Extravasation of RBC's due to rupture & degradation & staining of blood vessel wall. Redness persists : Non blanching response. 

Active space

2. Lupus Vulgaris : Type of cutaneous TB.
Press a glass slide : Erythema disappears, Granulomas become prominent : Apple jelly nodules.



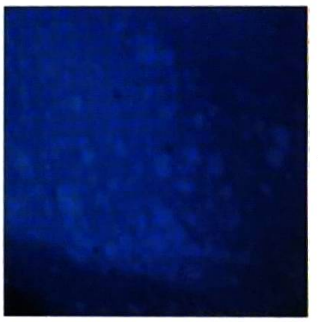
Dermatological investigations

00:39:45

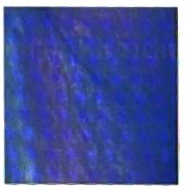
Woods lamp examination :

- Wavelength : 360 to 364 nm.
- Made of filter : 9% Nickel oxide + Barium silicate.
- Infections : Fungal (Tinea capitis).
  - i. Microsporum species : Blue green fluorescence.
  - ii. Trichophyton schoenleinii (Favus) : Dull blue color.
    Most other Trichophyton species are negative.
- Pityriasis Versicolor (Malassezia species) : Yellow fluorescence.
- Erythrasma : Corynebacterium minutissimum.
  Coral red fluorescence.
  Liberation of Coproporphyrin 3.
- Pigmentary disorders :
  - i. Ash leaf macule : Tuberous sclerosis complex.
  - ii. Vitiligo.
  - iii. Melasma.
- Metabolic disorders :
  - i. Porphyria cutanea tarda
    Enzyme involved : Uroporphyrinogen decarboxylase.
    Pink color fluorescence (Urine).
  - ii. Congenital erythropoietic porphyria / Gunther's disease : Autosomal recessive.
    Enzyme : Uroporphyrinogen 3 Co-synthase.
    Red teeth (Erythrodontia) is a characteristic sign.

wood's lamp



Erythrasma



Porphyria Cutanea Tarda

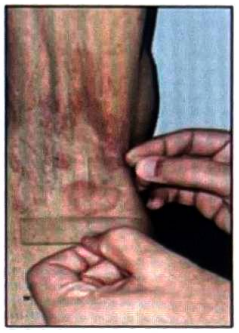
Congenital Erythropoietic Porphyria

Laboratory investigation

00:46:05

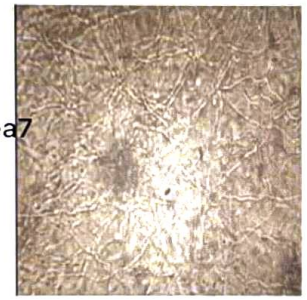
1. KOH (Potassium hydroxide) mount:
   Scraping of skin/nail/hair → KOH mount → Keratin dissolved.

Potassium Hydroxide/KOH mount

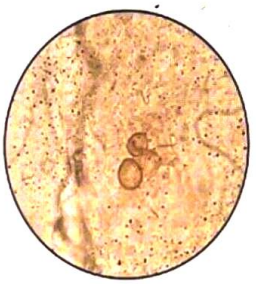


- Dermatophytosis (Tinea infections) → Branching hyphae.
- Pityriasis versicolor → Short hyphae + round spores (Spaghetti + meat ball appearance).
- Chromoblastomycosis : Subcutaneous mycosis → Copper penny bodies (Round, brown thick walled bodies).

Pityriasis versicolor



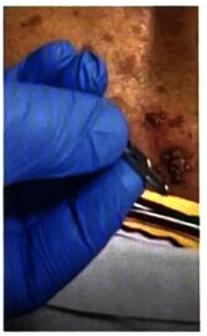
Dermatophytosis



Chromoblastomycosis

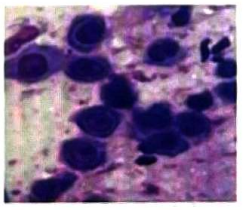
2. Tzanck smear (Cyto-diagnostic test) :
   Fluid filled lesion (Vesicle/Bulla) → Deroof the lesion → Scraping from floor and roof → Giemsa stain.

Tzanck smear



- Acantholytic cells : Pemphigus group.

Pemphigus group



- Multinucleated giant cells : HSV 1, 2 and Varicella infections.

Active space

Dermatological therapy

00:51:56

1. Cryotherapy :
   - Agent : Liquid Nitrogen (- 196 °C).
   - Mechanism : Freeze tissue ⟶ Osmolarity changes ⟶ Rupture of cells.
   - Indication : Management of Warts & Keloids.

Cryotherapy

2. Phototherapy.
   - Agent : UV Radiation.
   - NB-UVB (Narrow band UV B radiation) Wavelength (311 ± 2 nm).
   - PUVA : Psoralen (P) (Photosensitizer) + UVA therapy. Wavelength (320 - 400 nm). Psoralen is given prior to UVA for better penetration of skin.
   - Indications :
     Psoriasis.
     Vitiligo.
     Atopic dermatitis.

Whole body phototherapy unit

PAPULOSQUAMOUS DISEASES : PART - 1

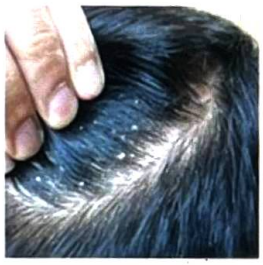
Psoriasis

00:00:20

Psoriasis : Primary skin lesions (solid circumscribed raised lesions).

- Papules <1 cm
- Plaques >1 cm

Secondary lesion : Visible exfoliation of stratum corneum : Scales.



Scales

Definition : Chronic, T-cell mediated inflammatory disease that involves skin and other systems.

Etiopathogenesis

00:01:59

Factors involved :

- Cellular components : Th_1, Th_{17}, Th_{22} (subsets of T cells).
- Signalling molecules : Cytokines.
- Environmental factors.
- Genetic factors.

Signalling molecules : Cytokines

- Th_1 subset : Produces IL - 2, TNF - α, IFN - γ.
- Th_{17} subset : produces IL - 17.
- Th_{22} subset : produces IL - 22.

There is an increase in proinflammatory cytokines.

Genetic factors :

HLA Cw6 : Associated with early onset psoriasis.

HLA B27 : Associated with psoriatic spondylitis.

PSORS 1 gene (Psoriasis susceptibility gene) :

- Expressed in chromosome 6.
- 50% risk of developing psoriasis.

Environmental factors : Drugs, infections, seasons, substance abuse.

Drugs that worsen psoriasis :

60c6b3eeaa8ded0e4e7e5ea7

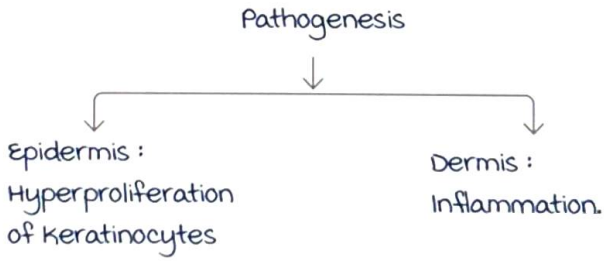
- Lithium.
- Antimalarials.
- β-blockers (anti-hypertensive drug).
- NSAIDS (common painkillers).
- Systemic steroids.

Infections :

- Group A β-hemolytic streptococci can trigger guttate psoriasis.
- HIV worsen psoriasis.

Season : Exacerbations during winter are common.

Substance abuse : Alcohol consumption and cigarette smoking can also trigger psoriasis.



Epidermal transit time : Time taken for a cell to go from stratum basale to stratum corneum normally is 28 days.
In psoriasis, because of epidermal hyperproliferation, complete process occurs in 3 to 5 days.

Clinical types of psoriasis 00:08:10

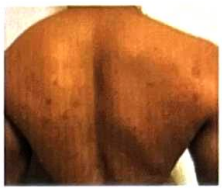
1. Chronic plaque psoriasis/psoriasis vulgaris.
   most common type of psoriasis.
2. Guttate psoriasis :
   Seen in Children with history of Sore throat (pharyngitis) caused by Group A β hemolytic streptococci.

Lesions : Rain drop like lesions.

"Gutta" refers to raindrop.

Site : Trunk.

Drug of choice : Macrolides.



Rain drop like lesions

3. Pustular psoriasis :

Characterised by pustules/tiny pus filled lesions.

Trigger : Sudden withdrawal of systemic steroids.

Presentation : Sheets or lakes of pus.

Pustular psoriasis

Classification based on extent of disease

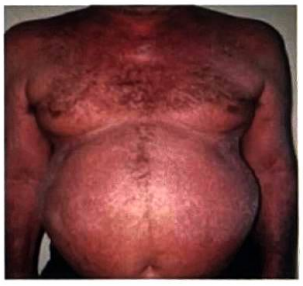
- Generalised pustular
  - Acute form : Von Zumbusch type.
  - Pregnancy : Pustular Psoriasis of Pregnancy (PPP) or Impetigo herpetiformis (misnomer).
    impetigo = bacterial, herpes = viral infection
- Localised pustular

4. Erythrodermic psoriasis :

Characterised by erythroderma/ exfoliative dermatitis.

Criteria for diagnosing erythroderma :

- more than 90% of body surface area (BSA) is involved with erythema ± scaling.

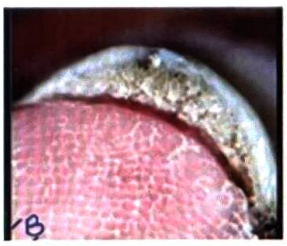


Erythroderma

There are multiple causes of erythroderma e.g. psoriasis.

Nail psoriasis :

A



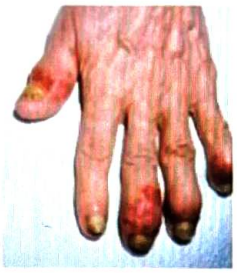
B

5. Psoriatic arthritis : Seronegative (RF -ve) erosive arthritis.

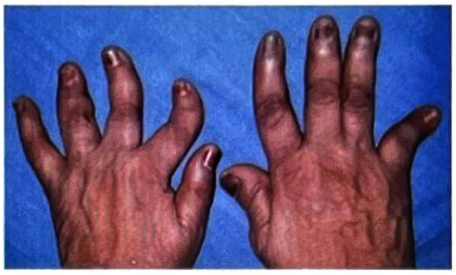
Destroys joint spaces.

Classical joint involved : Distal interphalangeal joint.

Worst form of psoriatic arthropathy : Arthritis mutilans.



Psoriatic arthropathy



Arthitis mutilans

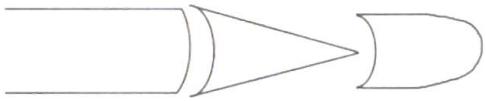
Digits : Inflammation of digits is known as dactylitis.

"Dactyly" means digit.

Sausage digit : Whole finger is swollen.

X-ray of psoriatic arthritis : Proximal bone tapers into the distal bone.

Pencil in cup appearance.



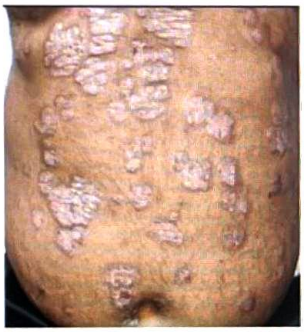
Chronic plaque psoriasis

00:15:50

Lesion : Erythematous, scaly papules and plaques.

Scales : Micaceous silvery white scales.

Site : "Extensor surfaces" : Elbows/ Knees.



Named features :

- Auspitz sign.
- Koebner's phenomenon.
- Woronoff's ring.

Auspitz sign :

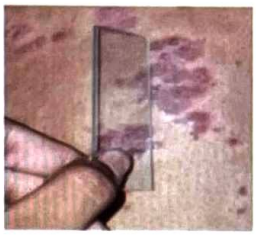
Method : Grattage test

Glass slide → Scrape psoriasis lesion.

Small papule, large plaque

Active space

Accentuation of silvery white scales.
↓ Continue scraping
Bulkeley membrane.
↓ Continue scraping
Pinpoint bleeding spots.
↓
Auspitz sign



Koebner's Phenomenon :
Synonym : Isomorphic response.
"Iso" : Similar; "Morphic" : Morphology.

Koebner's Phenomenon

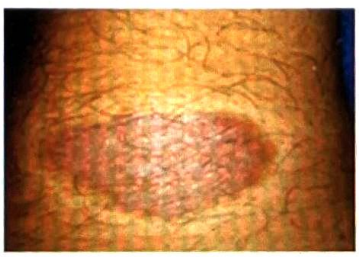
Definition : Appearance of morphologically similar lesions along line of trauma over normal skin.
Implies an active disease.

Types of Koebner's phenomenon (KP) :

True KP	Pseudo KP	Rare causes
Immunologic Eg., Psoriasis. Lichen planus. Vitiligo.	Auto inoculation Occurs due to viral infection. Lesions multiply in a line following trauma (scratching). Eg., Molluscum contagiosum. Viral warts.	Kaposi sarcoma Darier disease (genetic cause of acantholysis) Lichen nitidus.

Woronoff's ring :

Hypopigmented rim around the lesions of psoriasis.



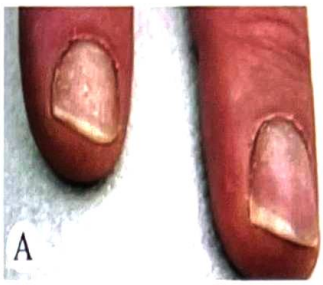
Woronoff's ring

Active space

Nail changes in psoriasis

00:24:20

Nail changes :



Nail pitting

Pitting of the nail :

Lesions : Depressions in nail plate.

Importance : Most common nail change in psoriasis.

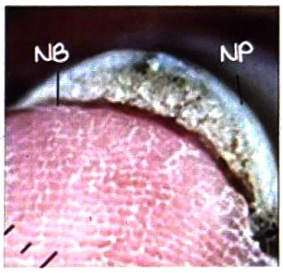
Types : Coarse, irregular and deep pits.

Defect : Proximal nail matrix is involved in pitting.

Sub ungual hyperkeratosis :

Sub ungual Accumulation of hyperkeratotic material.

Distal onycholysis : Separation of nail plate from underlying nail bed due to sub ungual hyperkeratosis.

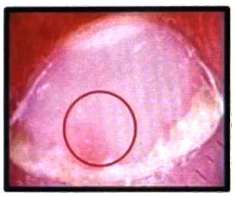


Sub ungual hyperkeratosis

Oil drop sign/salmon patch :

Importance : Pathognomonic of nail psoriasis.

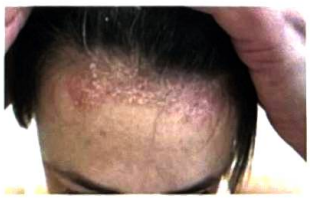
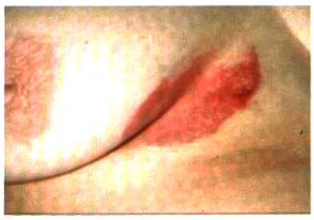
Presentation : Reddish yellow circular discoloration involving the nail bed.



Oil drop sign

Regional variation of psoriasis :

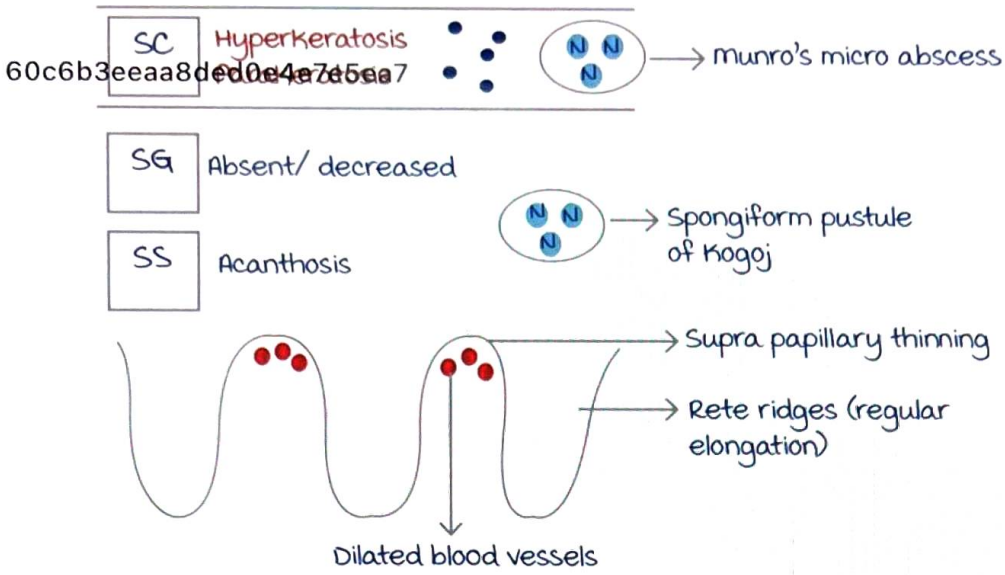
kumarankitindia1@gmail.com

Scalp psoriasis		Only scalp lesions
Flexural psoriasis		Also known as inverse psoriasis. Affects only the flexural areas.

Active space

Histopathology of psoriasis

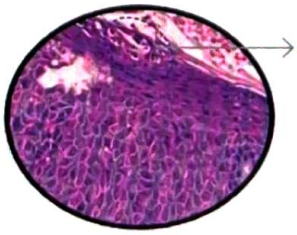
00:29:28



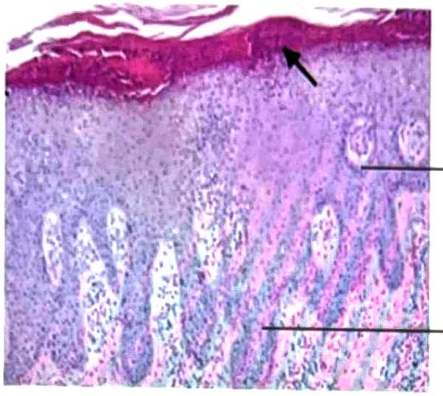
Hyperkeratosis : Thickening of stratum corneum.
Parakeratosis : Retention of nucleus in stratum corneum due to altered epidermal differentiation, impaired Keratinisation.
Normally, nucleus is absent in stratum corneum.
Munro's micro abscess : Collection of neutrophils at the level of stratum corneum.

Stratum granulosum is absent in psoriasis.
As epidermal transit time is reduced, there is no time for granular layer to form.

Acanthosis : Thickening of stratum spinosum.
Spongiform pustule of Kogoj : Collection of neutrophils at the level of stratum spinosum.
Rete ridges : Projection of epidermis into dermis.
In psoriasis, there is regular elongation of rete ridges.
Presence of dilated blood vessels in dermal papilla.
Pinpoint bleeding spots in Auspitz sign is due to rupture of these dilated blood vessels.
Supra papillary thinning : Area just above dermal papillae will be thinned out.



Active space



Treatment options in psoriasis

00:33:28

Topical : < 10 % BSA (Body surface area) involved.

Phototherapy
Systemic
} > 10 % BSA involved.

Topical therapy :

Aim of therapy	Drug
Reduce scaling	Emollients/mosturisers
Anti inflammatory	Topical steroids
DNA synthesis blocker to reduce epidermal hyperproliferation.	Coal tar, Dithranol
Normalise Keratinisation.	Vitamin A analogues : Retinoids. Tazarotene (Topical retinoid). Vitamin D analogues : Calcipotriol.

Photo therapy :

Mechanism :

Psoriasis : Increased pro inflammatory cytokines.

Phototherapy increases anti inflammatory cytokines (IL - 10 and IL - 13) to neutralize pro - inflammatory cytokines.

NB UVB (narrow band UV-B therapy) : 311 ± 2 nm.

PUVA : Psoralen + UV-A (320 to 400 nm)

Psoralen is a photosensitiser (topical or systemic) which allows UV-A to penetrate better.

Topical psoralen is given 30 minutes before UVA radiation.

Active space

Phototherapy regimens :

Goeckerman Regimen : Coal tar + NB UV-B.

Ingrams Regimen : Coal tar + NB UV-B + Dithranol.

Systemic therapy :

Drug	mechanism of action	Indication	Side effects
Methotrexate (Once weekly regimen). Given with folic acid usually.	Acts at S phase of cell cycle. Inhibits Dihydrofolate reductase (inhibits conversion of DHFA to THFA). which is required for DNA synthesis.	• Chronic Plaque psoriasis. • Pustular psoriasis. • Erythrodermic psoriasis.	• Bone marrow suppression (severe if given daily). • Teratogenicity. • Hepatotoxicity.
Cyclosporine	Calcineurin inhibitor : Decreases IL 2.	• Severe erythrodermic psoriasis (rapid action). • Pustular psoriasis.	• Hypertension (check BP) • Nephrotoxicity.(Do RFT) • Gum hyperplasia.
Acitretin (Oral retinoid)	Modulates Keratinocyte differentiation.	Drug of choice : Pustular psoriasis.	• Hyperlipidemia. • Hepatotoxicity (do LFT). • Teratogenicity (all vit A analogues are category X drugs).
Apremilast	Inhibitor of phosphodiesterase 4 required to convert cAMP → AMP, cAMP increases. ↓ Pro-inflammatory cytokines decreases.	moderate to severe chronic plaque psoriasis.	• Headache. • Diarrhoea (self limiting). • Persistent secretory diarrhoea : Stop the drug.

Systemic steroids : Systemic steroids are contraindicated in psoriasis.

Only Indication : Pustular psoriasis of pregnancy (Impetigo herpetiformis).

2nd drug of choice : Cyclosporine (expensive drug).

Active space

Biologics (Targeted therapy) :

Cytokine targeted	Drug
IL - 12 IL - 23 } Common P 40 subunit	Ustekinumab
IL - 23	Guselkumab Tildrakizumab Risankizumab
TNF - α	Infliximab Etanercept Adalimumab
IL - 17	Secukinumab Ixekizumab
IL - 17 receptor	Brodalumab

Advantage of biologics : No organ specific side effects.

Systemic association of psoriasis : metabolic syndrome.

PAPULOSQUAMOUS DISEASES : PART - 2

60c6b3eeaa8ded0e4e7e5ea7

Lichen planus

00:00:27

Definition : Chronic, immune mediated inflammatory skin disease.

Triggers :

- Most important : Dental amalgam (presence of mercury).
- Hepatitis C and Hepatitis B.
- Drugs : Anti malarials.

C/F :

Symptoms : Presents with skin lesions & pruritus.

Oral lesions : Asymptomatic/burning sensation on spicy food intake.

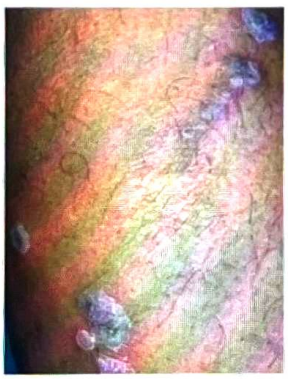
Morphology 6P's :

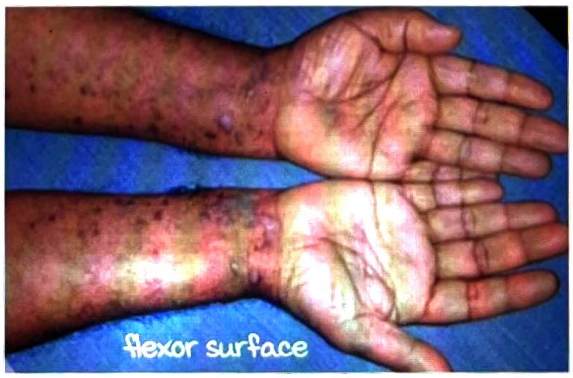
- Purple and pruritic.
- Polygonal and plain topped.
- Papules and plaques (image-violeceous purplish)

Surface of LP seen with magnifying lens with oil on top of lesion : Whitish linear streaks → Wickhams striae.

Distribution : Predominantly distributed over flexor surfaces.

True Koebner phenomenon/isomorphic response : Seen in Psoriasis, Vitiligo & Lichen planus along lines of trauma.

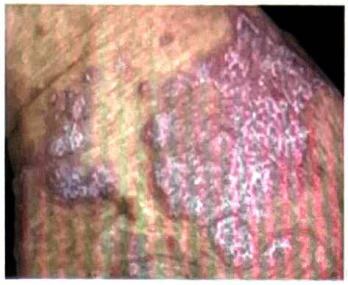




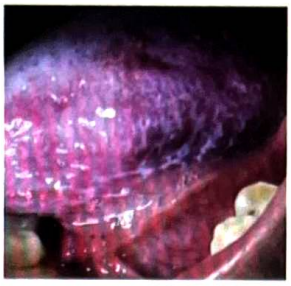
Oral lesions : Reticulate lacy white pattern (Net like pattern).

Erosive LP : Pre malignant lesion (chance of developing malignancy).

Active space



Wickham's striae



Reticulate lacy white pattern

True Koebner phenomenon

Nail involvement :

Most common finding : Thinning of nail plate.

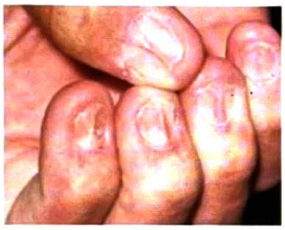
Most characteristic nail finding :

Pterygium of the nail → wing shaped/ triangular shaped fold → extends from

proximal nail fold → destroys nail plate → reaches nail bed.

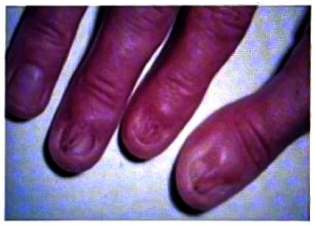
Anonychia : Absence of nail plate due to destruction.

Pup tent sign : Tenting of nail plate.



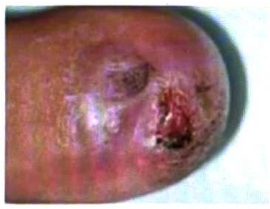
Thinning of Nail plate

Pterygium



Pup tent sign

60c6b3eeaa8ded0e4e7e5ea7



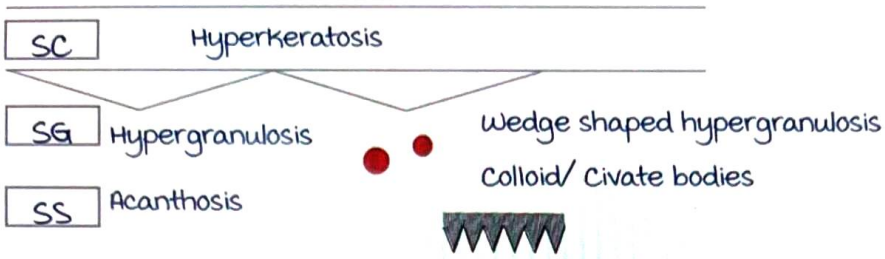
Anonychia

Lichen plano pilaris/ hair manifestation of LP 00:10:48

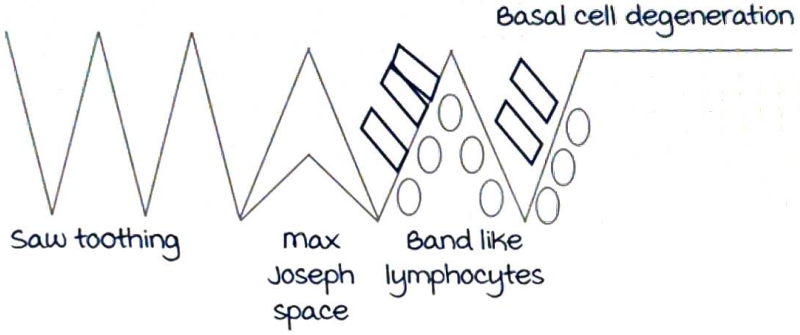
Patient presents with scarring alopecia (hair destroyed) showing perifollicular blue grey hue.

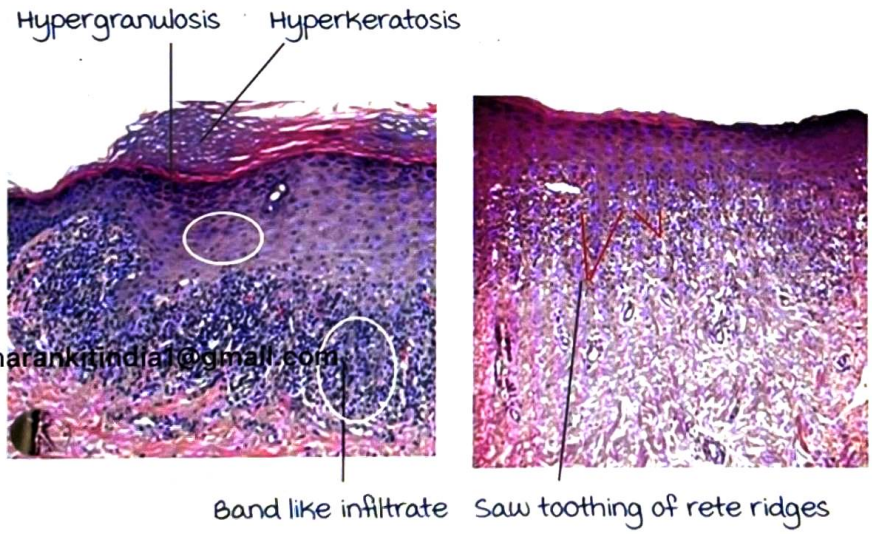
Histopathology of LP :

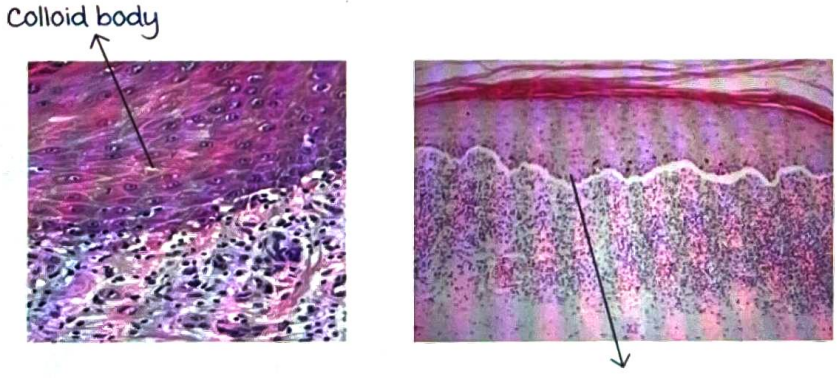
- Hyperkeratosis in stratum corneum (SC).
- Wedge shaped hypergranulosis in stratum granulosum (SG).
- Thickening of stratum spinosum (SS) : Acanthosis.
- Necrotic keratinocytes (colloid bodies/civatte bodies).



- Saw toothing of rete ridges.
- Artefactual cleft at Dermo Epidermal Junction (DEJ) : Max Joseph space.
- Band like lymphocytic infiltrate at DEJ.
- Basal cell degeneration (moving up)







Treatment of Lichen planus :
Localized : Topical steroids.
Extensive : Systemic steroids.

Pityriasis rosea

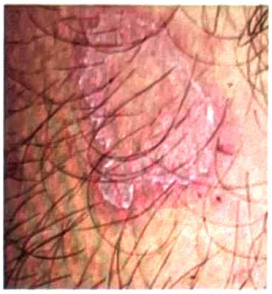
00:17:28

Pityriasis : Scaling ; Rosea : Red colour eruption.

Definition : Acute, self limiting, papulo squamous eruption.

Aetiology : m/c viral trigger : HHV 7.

Mostly asymptomatic/mild pruritis may be present.



Herald Patch

Morphology/course :

1st lesion : Herald patch/mother patch.

↓

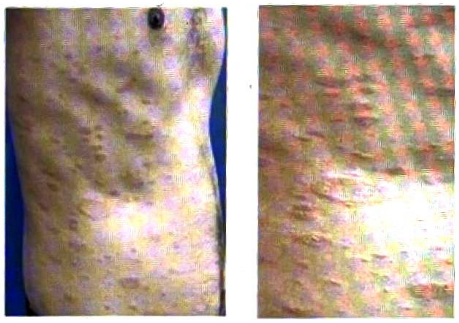
Secondary lesion : Scaly papules → oval annular plaques.

↓

mild pigmentary changes.

↓

Fades slowly in 4 to 6 weeks (total duration).



Secondary lesions

Herald patch :

- Lesion : Annular lesion.
- Scales : Collarette.
- Outer edge : Within the active margin of lesion (attached).
- Inner edge : Free.

Clinical sign : Hanging curtain sign.

(Stretch skin along long axis of herald patch → Scales fold along line of stretch).

Distribution of lesions

00:23:28

60c6b3eeaa8ded0e4e7e5ea7

Lesions are distributed in a particular christmas tree pattern.

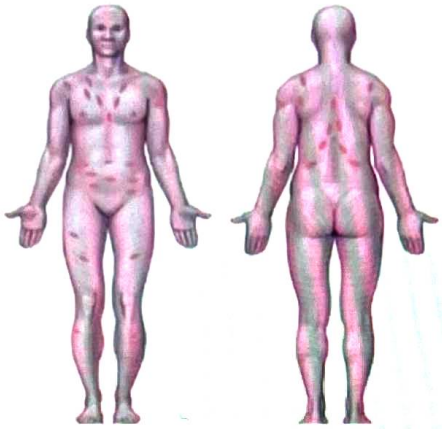
Long axis of lesion.

↓

Downwards and outwards from the spine.

→ Parallel to the ribs, along the lines of Langer (lines of skin tension).

Active space



Christmas tree distribution

Treatment :

60c6b3eeaa8ded0e4e7e5ea7

Course : Counsel patient (self limiting : 4 -6 weeks)

Itchy lesions : Mild to potent steroids Anti histamines.

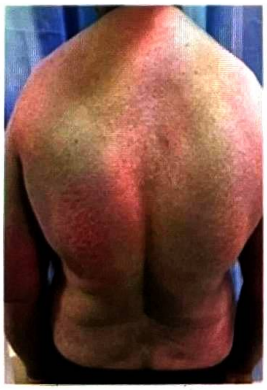
Recalcitrant : Narrow band UV-B phototherapy (311 ± 2nm)

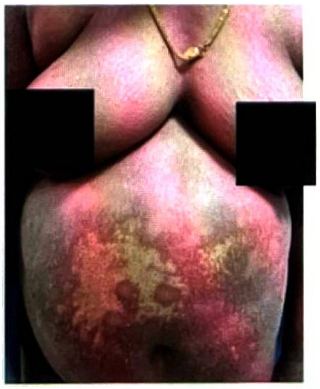
Pityriasis rubra pilaris

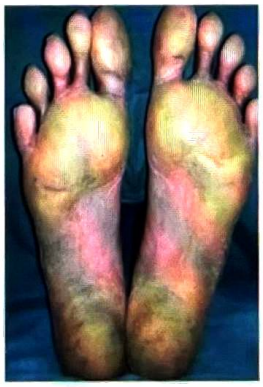
00:26:19

Pityriasis refers to scaling , rubra (red), pilaris (hair follicle).
Definition : Chronic inflammatory papulo squamous disease of unknown aetiology.

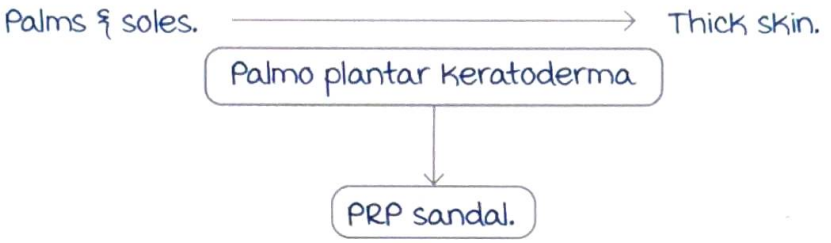
morphology : Follicular papules around hair follicles.
Progresses to form orange red erythema in cephalo caudal direction.
>90% of body surface area gets involved (erythroderma).
Special feature : Erythroderma + islands of sparing (some patches of the body are spared).







Active space



Treatment :

Oral retinoids. Acitretin is the preferred drug.

60c6b3eeaa8ded0e4e7e5ea7

Active space

APPENDAGES AND DISORDERS : PART - 1

Hair

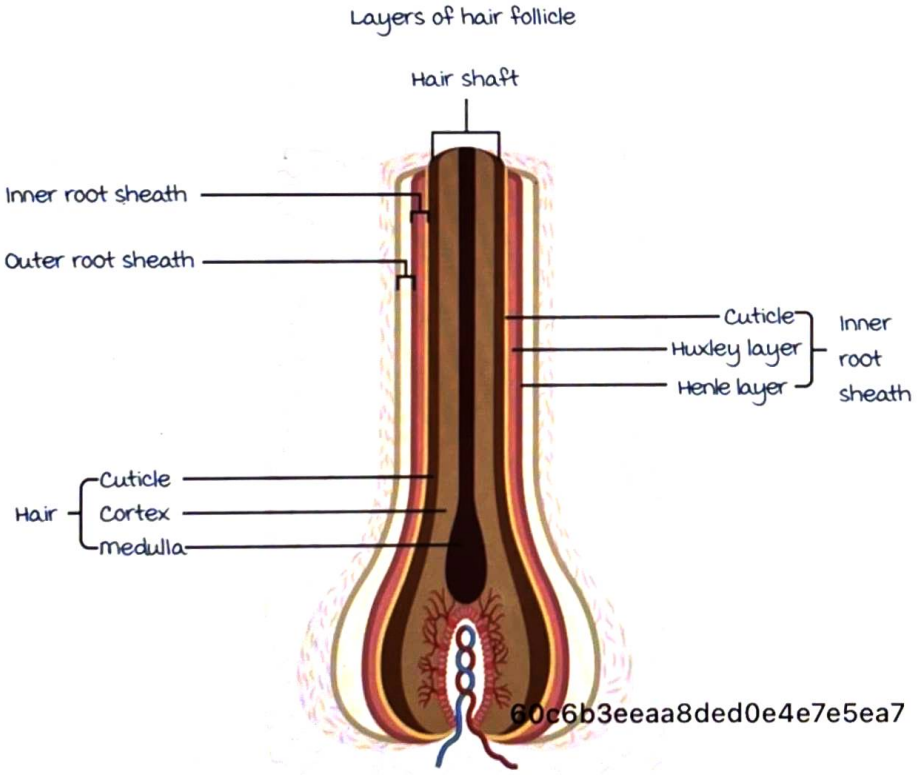
00:00:34

Structure : Ectodermal appendage.

Importance : Defining characteristic of a mammal.

Layers of hair (from within outwards) :

1. Hair shaft.
2. Inner root sheath.
3. Outer root sheath.
4. Glassy membrane.
5. Connective tissue sheath.



Layers of hair shaft (within outwards) :

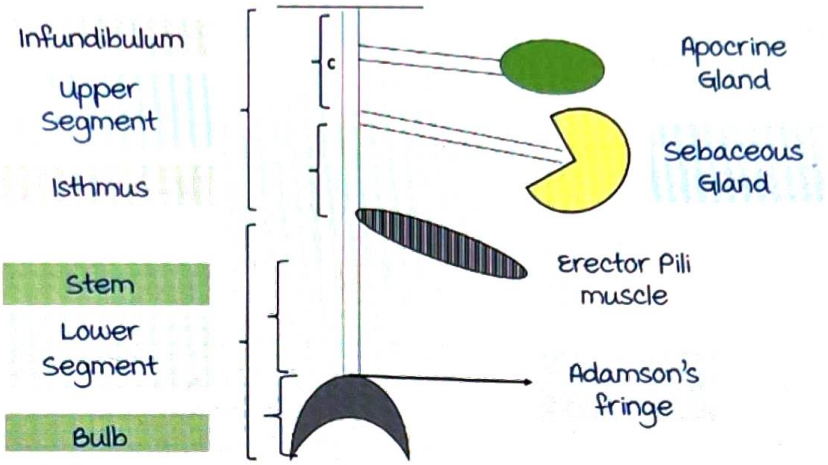
1. Medulla.
2. Cortex.
3. Cuticle.

Layers of inner root sheath (within outwards):

1. Cuticle.
2. Huxley layer.
3. Henley layer.

Active space

Longitudinal section of hair :



Divided into upper segment and lower segment, attached with the help of erector pili muscle.

Upper segment is further divided into upper infundibulum and lower isthmus by sebaceous gland.

Apocrine gland opens just above the opening of sebaceous gland.

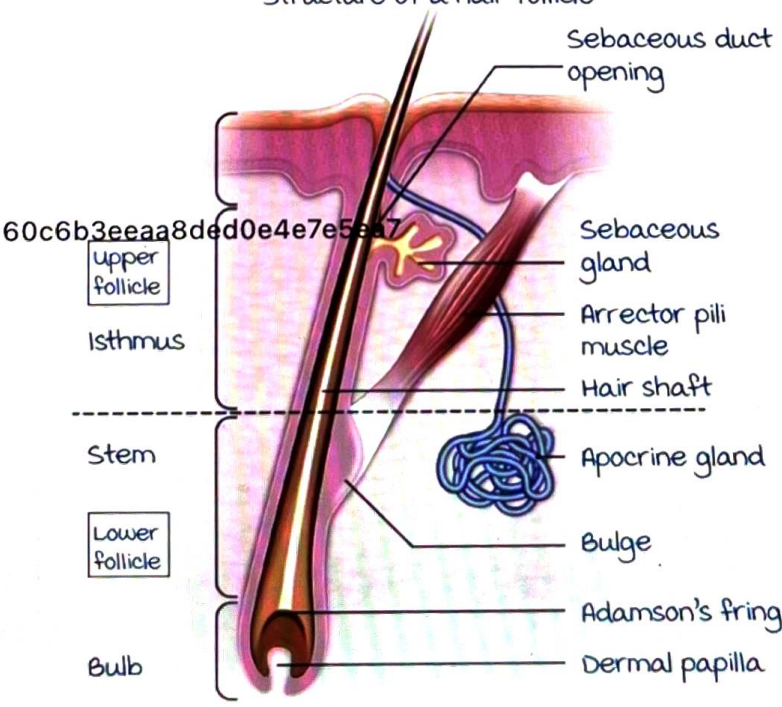
Lower segment is further divided into upper stem and lower bulb by Adamson's fringe.

Hair follicle stem cells :

Type : multipotent cells.

Location : At bulge area, which corresponds to the attachment of erector pili muscle.

Structure of a hair follicle



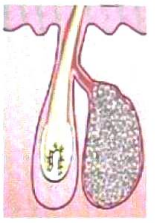
Damage to hair follicle stem cells leads to scarring alopecia.

Types of hair

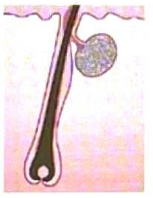
00:06:00

1. Lanugo :
   Seen in the fetus.
   Soft, fine hair.
   Usually shed in utero.

2. Vellus :
   Fine, unmedullated, soft hair.
   The bulb is at the level of the reticular dermis.



3. Terminal :
   Thick, medullated, coarse hair.
   The bulb is at the level of subcutaneous tissue.



In hirsutism :
Vellus hair gets converted to terminal hair.

In androgenetic alopecia :
Terminal hair gets converted to vellus hair.

Hair cycle:
A process in which hair goes through different stages.

Stage	% of Hair	Duration
Anagen (growth phase)	86% (max in number)	3 years
Catagen (involution/destruction phase)	1% (least in number)	3 weeks
Telogen (resting phase)	13%	3 months
Exogen (shedding phase)	-	-

Hair growth rate : 1 cm per month.

Active space

Alopecia

00:11:30

Defined as loss of hair.

Types :

1. Non scarring (non-cicatricial).
2. Scarring (cicatricial).

Cicatrix means scar.

Features	Non scarring (non-cicatricial)	Scarring (cicatricial)
Hair follicles	Preserved	Destroyed
Visible signs of inflammation	Absent	Pustules/scaling present
Course	Generally reversible	Irreversible/permanent

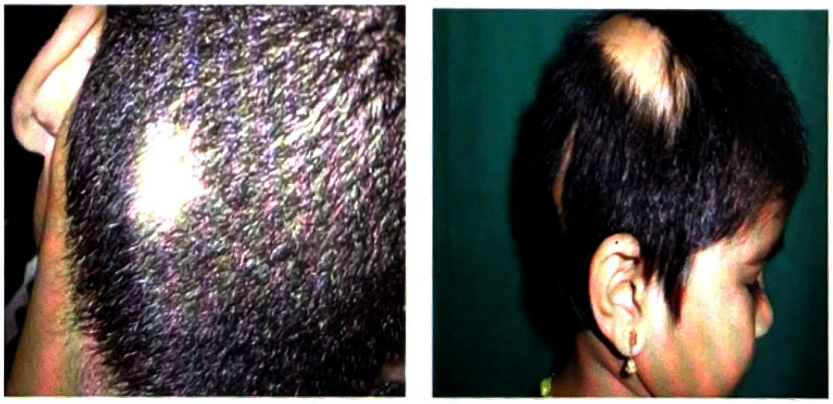
Non-cicatricial alopecia classification :

Patchy	Diffuse	Patterned	Systemic
Alopecia areata. Trichotillomania. Tinea capitis (non inflammatory). Secondary syphilis.	Effluvium (anagen effluvium and telogen effluvium).	Androgenetic alopecia.	SLE. Thyroid dysfunction.

Alopecia areata:

- Autoimmune disease.
- Targets anagen hair bulb.
- Areata : Patchy/spotty areas of alopecia.

60c6b3eeaa8ded0e4e7e5ea7



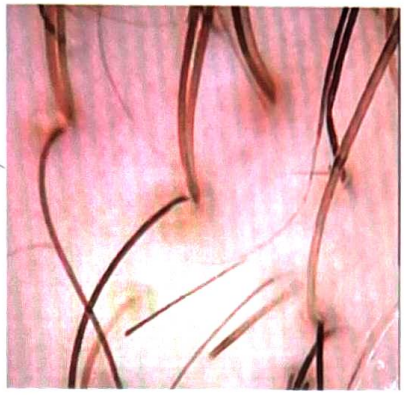
Circular areas of complete hair loss

Margin of lesions :

Exclamation mark hair.

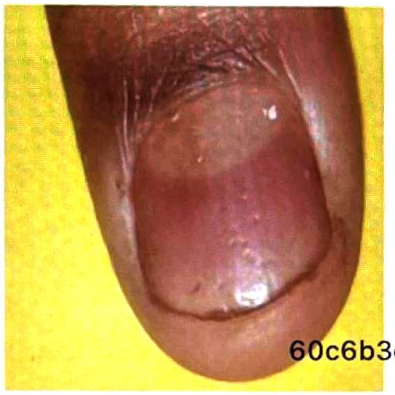
Distal end : Broad

Proximal end : Tapering.



Alopecia areata nail findings :

Geometrical pitting : Fine, superficial, regular pits.



Histopathology of alopecia areata :

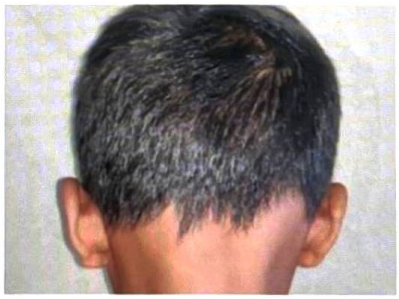
Peribulbar lymphocytic infiltrates :

Swarm of bees appearance.

Lymphocytes are compared to bees, anagen bulb is compared to beehive.

Bad prognostic factors for alopecia areata 00:19:30

1. Early onset (children).
2. Patterns like :
   - Ophiasis (occipital hair loss).
   - Alopecia totalis (involvement of total scalp hair).
   - Alopecia universalis (involvement of body hair).
3. Association with :
   - Atopy (localized type I hypersensitivity reaction).
   - Autoimmune diseases.



Ophiasis (occipital hair loss)

Alopecia totalis

Treatment for alopecia areata :

Localized diseases :

Intralesional steroids (Triamcinolone acetonide).

Active space

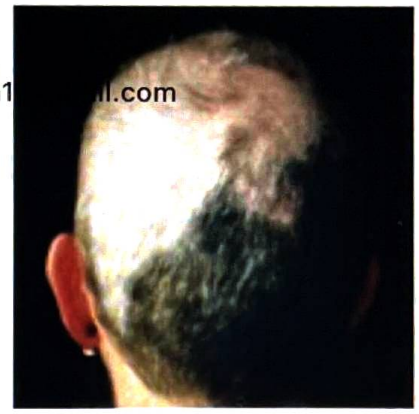
Extensive disease :

- Systemic steroids.
- Azathioprine.
- Sulfasalazine.
- Tofacitinib : Potent JAK-1/JAK-3 inhibitors.

Trichotillomania

00:23:58

An obsessive compulsive disorder, characterized by pulling out one's own hair.



- Incomplete loss of hair within a patch.
- Hairs of varying length.
- Perifollicular haemorrhage.

Infections :

Tinea capitis (non-inflammatory) : Invasion of hair shaft.

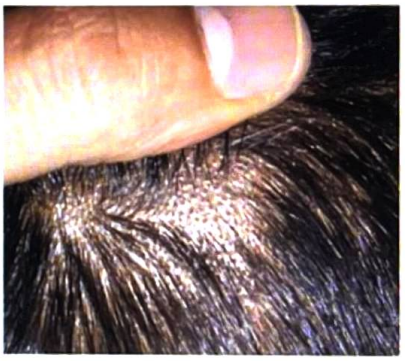
Types :

- Black dot type.
- Grey patch type.

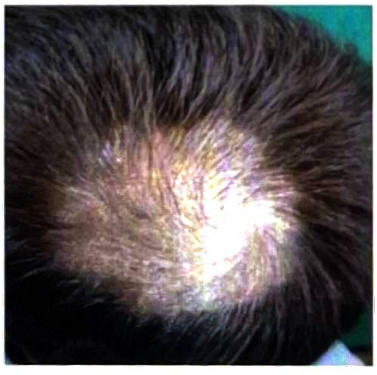
Black dot type : Endothrix infection that goes into the hair.

Causative organism :

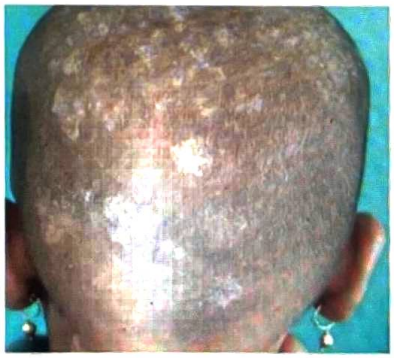
- Trichophyton tonsurans.
- Trichophyton violaceum.



Black dot type



Grey patch type : Lustreless hair



Grey patch type : Patchy alopecia + scaling

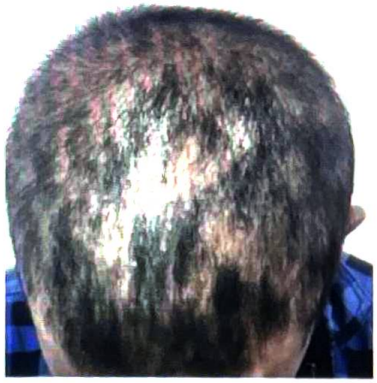
Active space

Grey patch type : Ectothrix infection that covers the hair.
Causative organism : Microsporum canis

Secondary Syphilis :

Hematogenous dissemination of Treponema pallidum.

Characteristic alopecia : Moth eaten alopecia.



Non-cicatricial alopecia : diffuse type 00:28:08

Effluvium : Loss of hair

- Anagen effluvium.
- Telogen effluvium.

Anagen effluvium :

Chemotherapy /radiotherapy (3-4 weeks)

↓

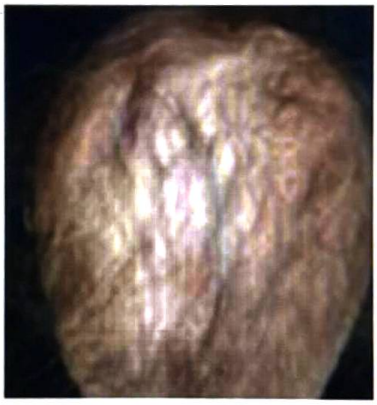
The arrest of hair growth in anagen phase

↓

Dystrophic anagen

↓

Hair loss



Telogen effluvium :

Major stress/post pregnancy/ infections (malaria, typhoid, COVID-19)/surgical procedures

60c6b3eeaa8ded0e4e7e5ea7

↓

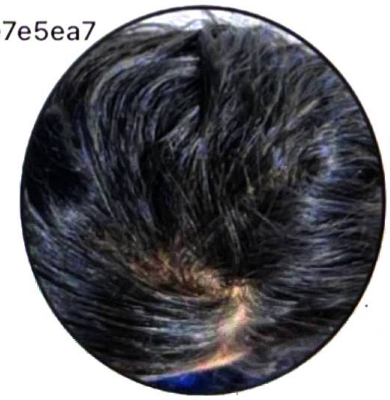
Premature entry of hair into the telogen phase

↓

3 months later

↓

Hair loss



Active space

Patterned type/ non-cicatricial alopecia

00:31:48

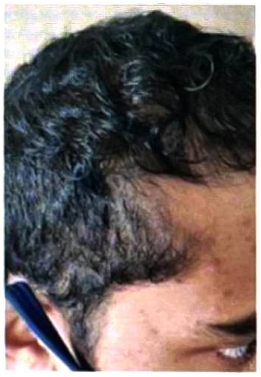
Synonym : Androgenetic alopecia.

Factors : Genetic factors + androgens.

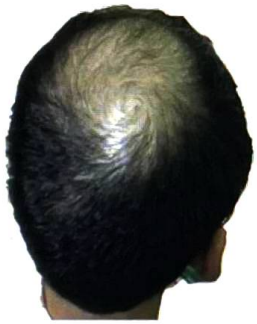
Testosterone is converted to dihydrotestosterone, which is responsible for thinning of hair & alopecia.

- Leads to the shortening of the anagen phase.
- Miniaturization of the hair follicle.

Men : Androgenetic alopecia.

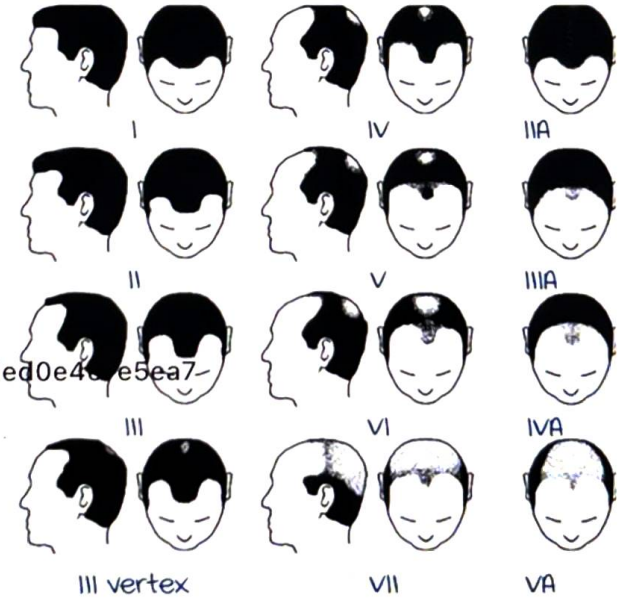


Frontotemporal recession

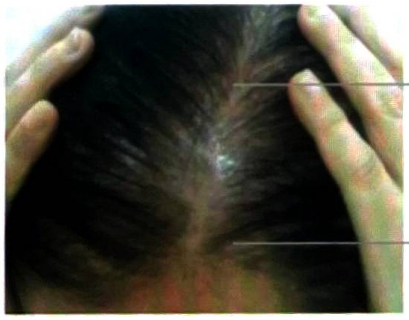


Balding of vertex

Hamilton Norwood grading :



Women : Androgenetic Alopecia

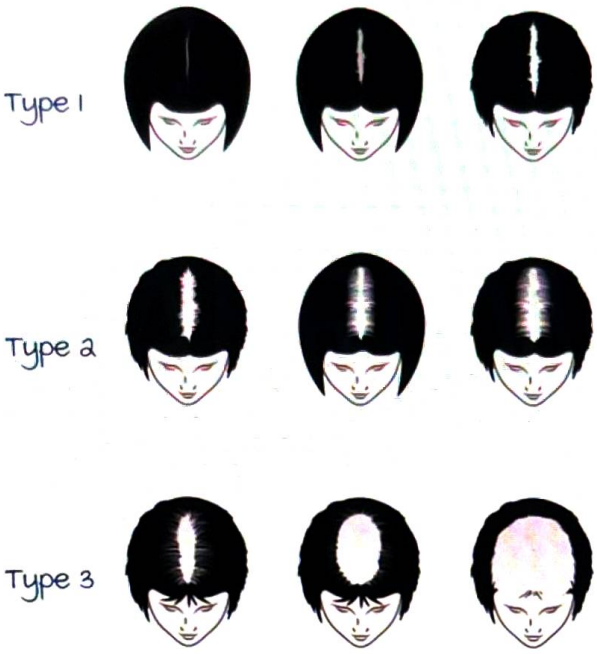


Loss of hair over the central portion of the scalp.

Frontal hair line maintained.

Ludwig's grading for female androgenetic alopecia :

Ludwig staging for female.
Pattern hair loss



Treatment for androgenetic alopecia :

- Minoxidil

  Increases anagen phase.

  Increases the size of the miniaturized hair follicle.

  Dosage :

  In female : 2% minoxidil BD (1ml - 0 - 1ml).

  In males : 5% minoxidil BD (1ml - 0 - 1ml).

- Finasteride

  Inhibits 5 α reductase which converts testosterone to dihydrotestosterone.

  Decreases dihydrotestosterone (responsible for hair thinning).

  Dosage : 1 mg in Androgenic alopecia.

  Other indication : Benign Prostatic Hyperplasia.

- Platelet-rich plasma therapy.
- Hair transplantation (in advanced stage).

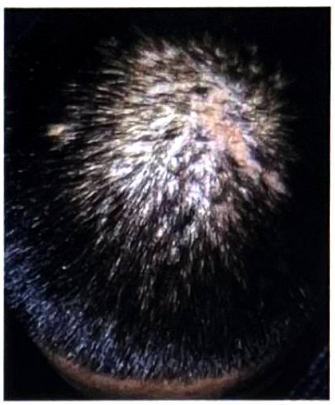
Systemic non cicatricial alopecia :

- SLE.
- Thyroid dysfunctions.

Scarring alopecia

00:37:54

1. Papulosquamous disease :
   - Caused by lichen planus/ lichen plano pilaris.
   - Diagnosed by : Perifollicular blue-grey hue.

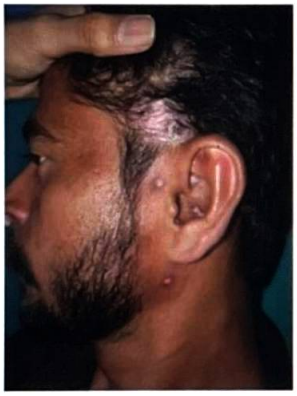


Papulosquamous disease

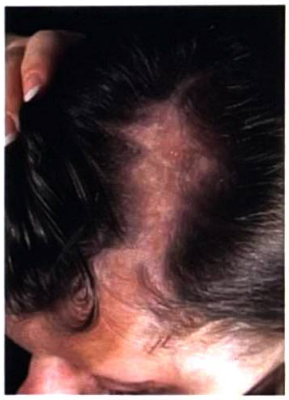
2. Granulomatous disorders :

Sarcoidosis

3. Connective tissue diseases :
   - Discoid lupus erythematosus/DLE
   - Discoid lesions of SLE.
   - Linear morphea : Thickening restricted to skin only.
     Affecting scalp is known as en coup de sabre (sword).



Discoid lupus erythematosus



Linear morphea

4. Infections : Tinea capitis (inflammatory) :
   - Favus.
   - Kerion.

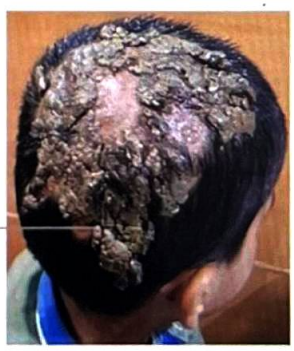
Favus :

Causative organism is T. schoenleinii.

Endemic in Kashmir.

Lesion : Yellow cup shaped crusts ←

Known as scutula.



Kerion :

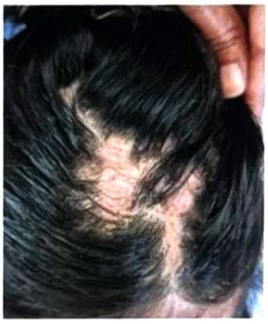
Causative organisms are zoophilic fungi :
   - T. mentagrophytes
   - T. verrucosum.

History : Contact with pet animals.
Commonly seen in children.
Lesion : Boggy indurated scalp swelling, easily pluckable hair, associated with regional lymphadenopathy.

Investigation : 10% KOH mount.

Treatment :
Overall drug of choice for T. Capitis : Griseofulvin.
For T. Capitis with trichophyton species : Terbinafine.

5. Inflammatory diseases :
- Folliculitis decalvans (balding of scalp).
- Dissecting cellulitis of scalp.

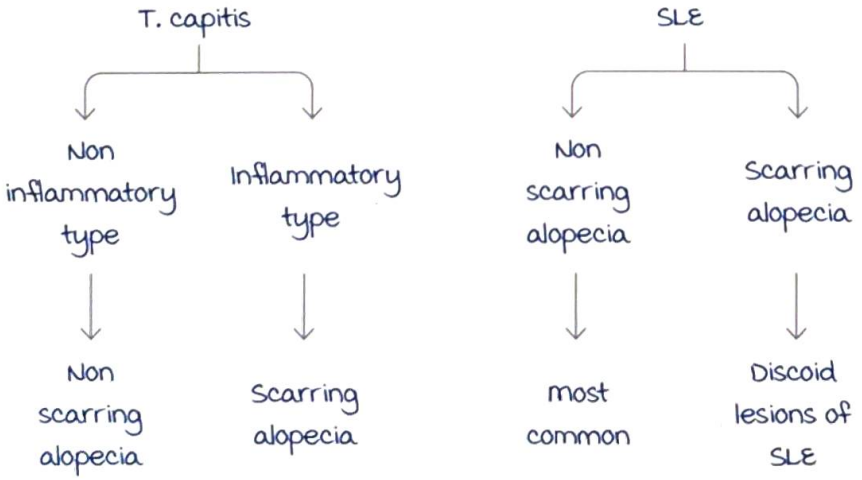


Pseudopelade of Brocq

6. Idiopathic condition :
Pseudopelade of Brocq.
Footprints in snow appearance.
Histopathology : Perifollicular concentric fibrosis.

Non-scarring/scarring alopecia

00:45:54



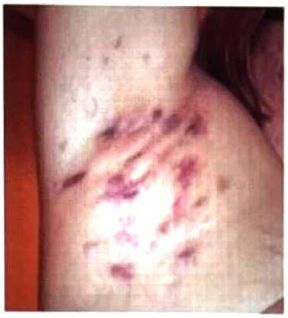
Hidradenitis suppurativa :
Hair follicle occlusion disease.
Defined as chronic recurrent inflammatory skin disease involving intertriginous areas (axilla/groin/perineum).

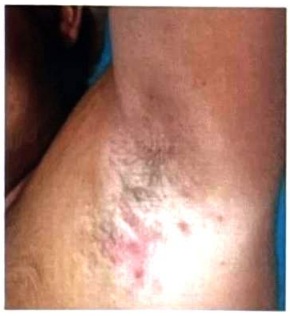
- Deep seated red nodules, abscesses.
- Discharging sinuses (later).
- Fibrotic scars : Patient unable to raise hands.

60c6b3eeaa8ded0e4e7e5ea7

Tretment :

1. Antibiotics (Clindamycin/Doxycycline).
2. Isotretinoin.
3. Adalimumab (TNF alpha inhibitor).



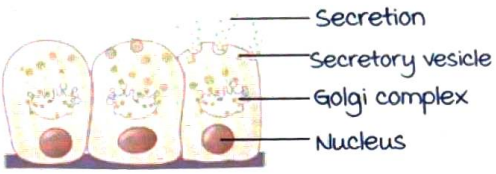


APPENDAGES AND DISORDERS : PART - 2

Classification of sweat glands

00:00:22

- Eccrine glands.
- Apocrine glands.



Eccrine glands :

Function : Sweat production.

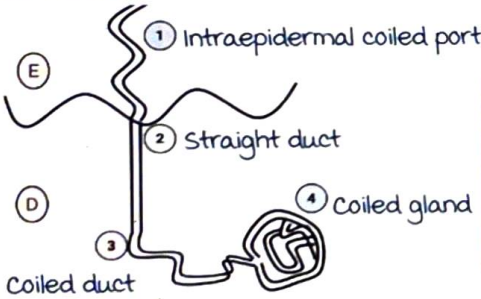
Secretion : Cell border is intact during the secretion (merocrine secretion).

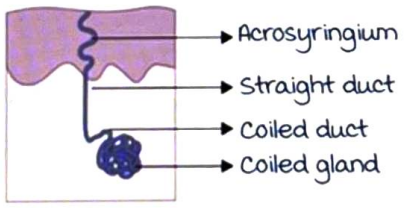
Sites : Almost all over the body (high concentration in palms and soles).

Opening : Duct directly opens into the skin surface.

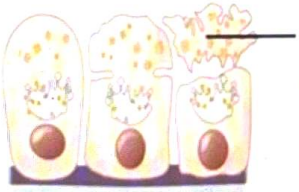
Nerve supply : Sympathetic cholinergic fibers.

Structure :





- Acrosyringium : Intraepidermal coiled portion.
- Straight duct.
- Coiled duct.
- Coiled gland.



Apocrine glands :

Function : Produce body odor.

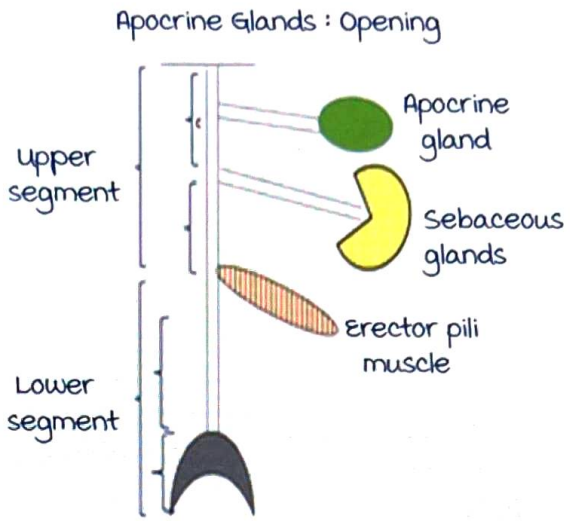
Secretion : Apex of the cell is pinched off (apocrine secretion).

Sites : Axilla, perineum.

Opening : Opens into hair follicle above the sebaceous duct.

Nerve supply : Adrenergic nerve supply.

Pilosebaceous unit :



Modified apocrine glands :

- Mammary gland.
- Glands of Moll in the eyelid.
- Ceruminous glands of EAC.

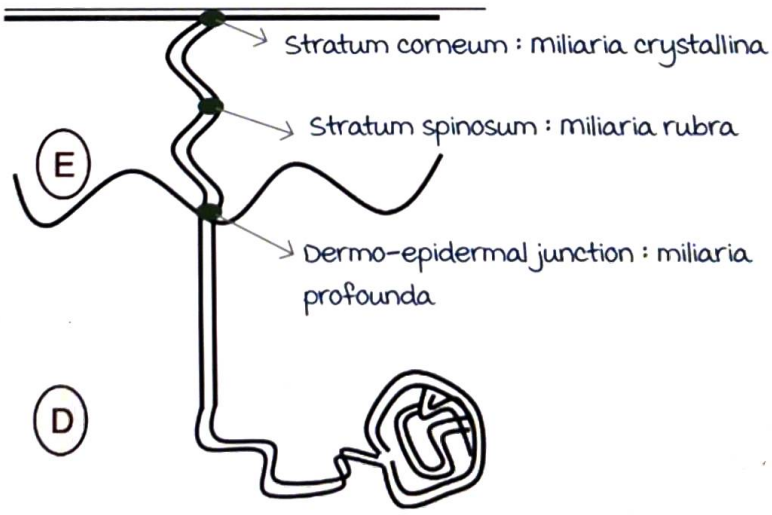
Disorders of eccrine and apocrine glands 00:05:16

Miliaria :

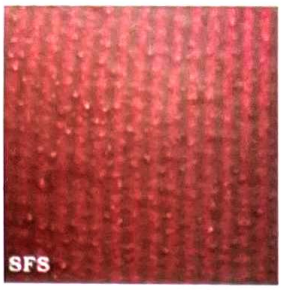
Definition : Obstruction to the eccrine sweat duct.

Predisposing factors : Hot humid climate.

Classification (based on the level of obstruction) :



- Stratum corneum : Miliaria crystallina.
  Tiny superficial vesicles which easily rupture.
  more common in neonates.
- Stratum spinosum : Miliaria rubra/prickly heat.
  Multiple erythematous papules.
- Dermo-epidermal junction (DEJ) : Miliaria profunda.

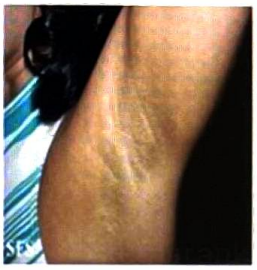


Miliaria crystallina

Miliaria rubra

Bromhidrosis : Disorder which is characterized by offensive body odor.

Chromhidrosis : Colored sweat.



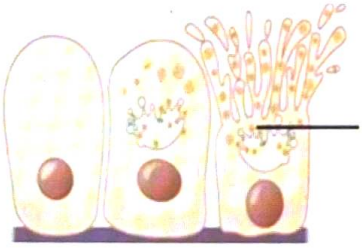
Fox-Fordyce disease

Fox-Fordyce disease :
Synonym : Apocrine miliaria.
Due to obstruction of the apocrine duct.
Morphology : Multiple skin colored pruritic papules.
Sites : Axilla, areola of the nipple.
Treatment : Topical corticosteroids/topical retinoids.

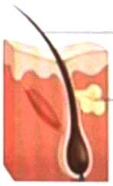
Sebaceous gland

00:10:05

Function : Sebum production.
Secretion : Holocrine secretion (the whole cell disintegrates during secretion).



Mature cell dies and becomes secretory product

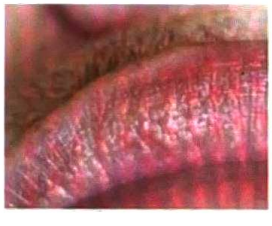


Pilosebaceous unit

Classification (based on the association with hair follicle) :

- Pilosebaceous unit (pilo : hair).
- Ectopic sebaceous glands (no association with hair follicle) :

1. Upper lip, buccal mucosa :
   Fordyce spots.
   Presents as yellow micro papules.
   Treatment : Reassurement.
2. Breast : Montgomery tubercles.



Fordyce spots

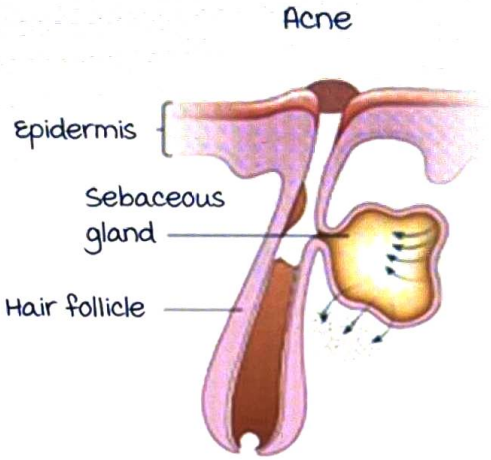
3. Eyelid : Meibomian glands and Glands of Zeis.
4. Prepuce : Tyson's gland.

Acne vulgaris

00:13:19

Definition : Chronic inflammatory disease of the pilosebaceous unit characterized by polymorphic cutaneous lesions.

Pathogenesis :



- Androgens activate the sebaceous gland leading to increased sebum production.
- Hyperkeratinisation of the infundibulum due to block in the passage of sebum.
- Presence of Cutibacterium acnes (old name : Propionibacterium).
- Inflammation.

Factors modifying acne :

- Climate : Hot and humid climate.
- Diet : Avoid high glycemic index foods, milk and milk products as they induce IGF -1 that can block the pilosebaceous ducts.
- Emotional stress and psychological factors.
- Facial cosmetics
- Genetic predisposition.
- Hormonal factors :

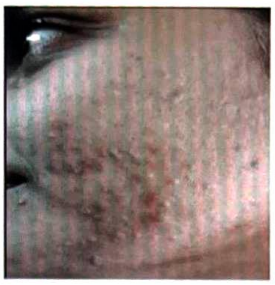
  In girls :

  Premenstrual flare (70% cases),

  Polycystic ovarian syndrome (PCOS).

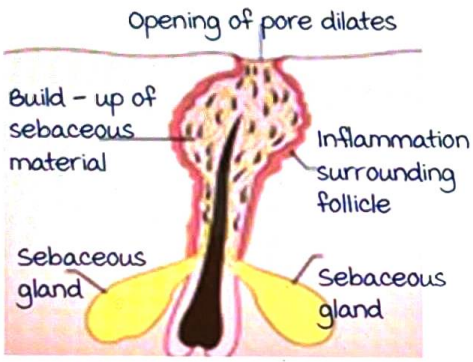
Grading of acne : Based on the predominant lesion 00:19:01

Grade 1/Comedones : Follicullo-centric lesions plugged by sebum and keratin.
Open comedones : Black (because of oxidation of the sebum by atmospheric air).
Closed comedones : white

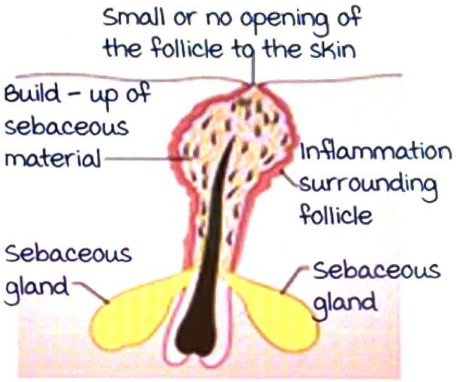


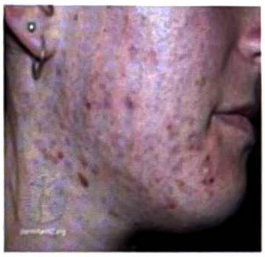
Grade 1

Open comedone : Blackhead

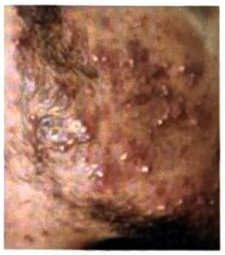


Closed comedone : Whitehead

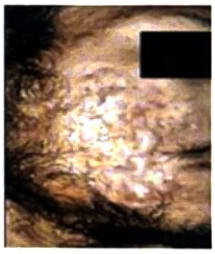




Grade 2



Grade 3



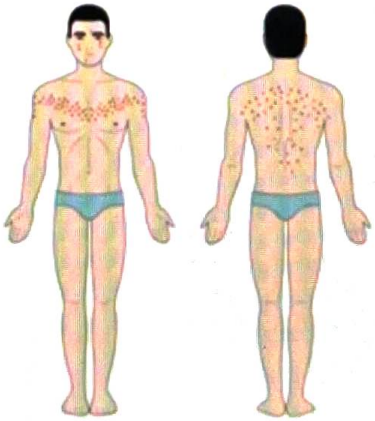
Grade 4

Grade 2 : Predominant papular lesions.
Grade 3 : Predominant pustular lesions.
Grade 4 : Nodulocystic acne with scars.

60c6b3eeaa8ded0e4e7e5ea7

Distribution :
Face.
Trunk (truncal acne due to high concentrations of pilosebaceous units even in the back).
Chest.

Distribution of lesions



Active space

Acneiform eruption/drug induced acne :

Variant of acne.

Onset : Sudden in onset.

Drugs : Systemic steroids,
ATT (Isoniazid).

Lesions : monomorphic lesions.

Lesions look similar to each other.

Comedones are absent.

Differential diagnosis of Acne vulgaris :

	Acne	Rosacea
Morphology	Comedones present. Papules, nodules, pustules and scars.	Comedones absent. Papules, nodules and pustules present.
Associations (Background)	Skin greasy	Erythema, flushing and telangiectasia prominent.
Distribution	Face, upper part of trunk, chest and shoulders.	Convexities of face (centrofacial lesion) (cheeks, nose & forehead); spares periorificial area.

Treatment of acne :

General measures :

- Cleansers to remove excess sebum secretion.
- Water based cosmetics.
- Avoid foods which are high in glycemic index.

Specific measures :

- Grade 1 (comedones) : Topical retinoids : Vitamin A analogue. (Tretinoin, Adapalene).
- Grade 2 : Topical retinoids + topical antibiotics (Clindamycin, Nadifloxacin)
- Grade 3 : Topical retinoids + oral antibiotics (Azithromycin, Doxycycline, Minocycline) (+/-) Benzoyl peroxide.
- Grade 4 : Oral retinoids.

Role of oral retinoids in acne

00:26:48

DOC : Isotretinoin.

Indication : Nodulocystic acne, resistant acne.

Most common side effect : Cheilitis (due to destruction of sebaceous glands).

Lab monitoring : Fasting lipid profile, LFT (as oral retinoids are associated with increased triglycerides and total cholesterol).

Pregnancy category : X (teratogenic).

Period of drug stoppage prior conception (women) : 28 days.

Rosacea

00:28:58

Definition : Chronic inflammatory facial dermatoses.

Sites : Centrofacial area (convexities of the face).

Pathogenesis/triggers :

- Blood vessels : Dysregulation of facial blood flow that manifests as abnormal vascular reactivity.
- Microbes : Demodex mite.
- Triggers : Sunlight, spicy food, alcohol, stress.

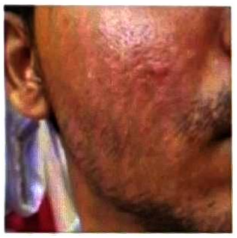
Morphological types :

- Erythemato telangiectatic type.
- Papulo pustular.
- Phymatous.
- Ocular.

Erythemato telangiectatic type :

Symptoms :

- Photosensitivity.
- Flushing (feeling of warmth with redness).



Centrofacial erythema

Lesions :

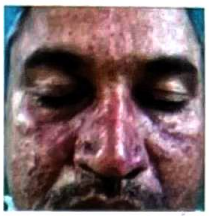
- Centrofacial erythema (transient /permanent).
- Telangiectasia (multiple dilated blood vessels).

Telangiectasia

Active space

Papulo-pustular stage :

Papules and pustules present.



Papulopustular type

Phymatous type :

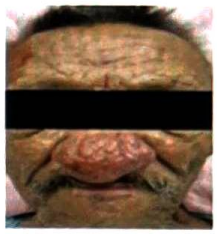
Synonym : Potato nose.

Lesion :

Skin over the nose : Thick skin.

Surface of nose : Prominent pores, surface nodularity.

Pathology : Characterized by hypertrophy and fibrosis of the sebaceous gland.



Phymatous type

Ocular rosacea :

Symptoms : Photophobia.

Eyelid margin telangiectasia.

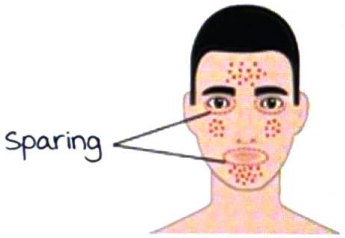
Ocular rosacea

Distribution of lesions :

Centrofacial disease.

Perioral and periocular sparing.

Distribution of lesions

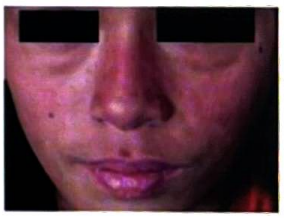


Differential diagnosis :

- Acne vulgaris.
- Malar rash of SLE : Acute cutaneous lupus erythematosus, with systemic symptoms like fever, arthralgia, myalgia etc.

  Papules and pustules are absent.

60c6b3eeaa8ded0e4e7e5ea7



Malar rash : SLE

Treatment of rosacea :

General measures :

- Sunscreen (for photoprotection).
- Avoid the triggers like spicy food, alcohol, stress.
- Topical steroids worsens rosacea.

Specific measures :

- Alpha agonist : Brimonidine.
- Antimicrobials :

  Topical : metronidazole.

  Systemic : Doxycycline or Ivermectin (systemic).

- For Rhinophyma :
  Early stages : Isotretinoin.
  Late stages : CO_2 laser excision followed by resurfacing (treatment of choice).

APPENDAGES AND DISORDERS : PART - 3

Structure of the nail

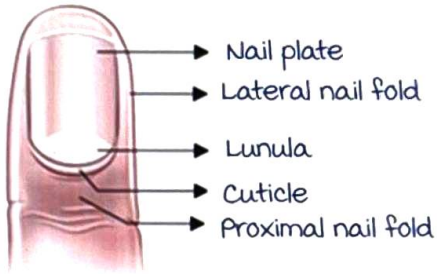
00:00:27

Structure : Ectodermal appendage.

Site : Dorsal aspect of the terminal phalanges.

Function : Protects the tips of the digits.

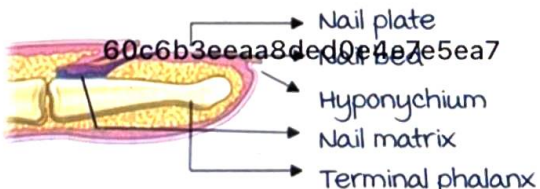
Dorsal view of nail :



Dorsal view of nail

- Nail plate : Dorsal rectangular structure of the nail.
- Lateral nail fold : Present laterally around nail plate.
- Lunula : Semilunar structure which represents the distal portion of the nail matrix.
- Cuticle : Seals the nail apparatus and protects the nail from exogenous agents.
- Proximal nail fold.

Lateral view of nail :



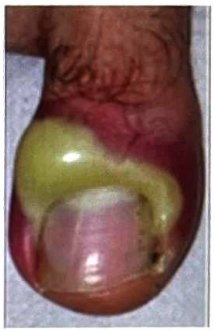
Lateral view of nail

- Nail plate : Superficial part.
- Nail bed : Structure which is below nail plate.
- Hyponychium : Area below the nail plate distally.
- Nail matrix : Responsible for the production of nail plate.
- Terminal phalanx.

Nail growth rate (mm/month) :
Finger nail growth : 3 mm/month.
Toe nail growth : 1 mm/month.
Nail disorders require longer duration of treatment due to slow growth rate.

Paronychia 00:04:06

Definition : Infection of the nail fold.


Acute paronychia

Acute paronychia :

- Cause : Staphylococcus aureus.
- Nail fold shows tender erythema and purulent collection.
- Treatment :
  If no abscess formation :
  Topical/systemic antibiotics.
  If abscess is formed : Surgical drainage.

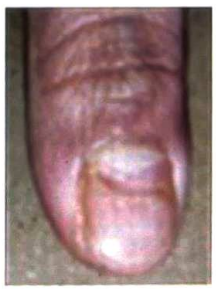
Chronic paronychia :


Chronic paronychia

- Cause : Candida albicans.
- Irritant contact dermatitis due to continuous contact with water with superadded candida albicans infection.
- Loss of cuticle and nail fold swelling is seen.
- Treatment : Antifungals + steroids (for inflammation/eczema component).

Onychomycosis 00:06:57

Definition : Fungal infection of the nail unit.

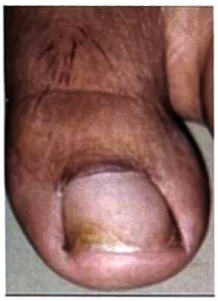


Etiologic classification :

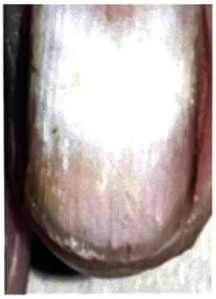
- Dermatophytes (Trichophyton species) : Tinea unguium.
- Non dermatophytes : Fusarium.
- Candida.

Clinical types :

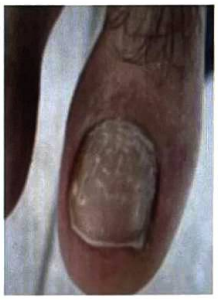
- DLSO (Distal Lateral Subungual Onychomycosis) : Most common type.
- Proximal subungual onychomycosis : Associated with HIV infection.
- White superficial onychomycosis : Due to superficial localization of the fungus over the dorsal aspect of the nail plate.
- Total dystrophic onychomycosis : Occurs as an end result of multiple nail traumas.
  There is nail plate crumbling and dystrophy.



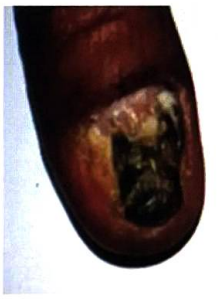
DLSO



Proximal subungual onychomycosis



White superficial onychomycosis



Total dystrophic onychomycosis

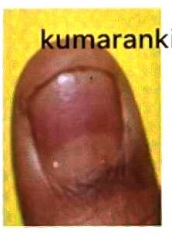
Treatment :

- Terbinafine (allyl amine group), 250mg OD.
  If fingernails : For 6 weeks.
  If toe nails : 12 weeks.
- Itraconazole (azole) : Given as pulse therapy.
  1 pulse : 200mg BD for 1 week per month.
  Fingernails : 2 pulses.
  Toe nails : 3 pulses.

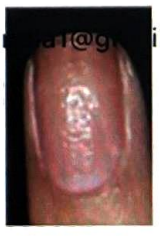
Pitting

00:11:02

Definition : Depressions over the nail plate.
Defect : Proximal nail matrix.



Alopecia areata



Psoriasis

Causes :

- Alopecia areata : Geometric pitting (superficial, regular pitting).

- Psoriasis : Coarse irregular deep pits.
- Eczema.

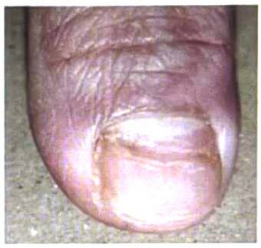
Pterygium 00:12:40

Definition : Wing shaped fold connecting the proximal nail fold to the nail bed after encroaching and destruction of the nail plate.
Dorsal pterygium is associated with Lichen planus.


Pterygium

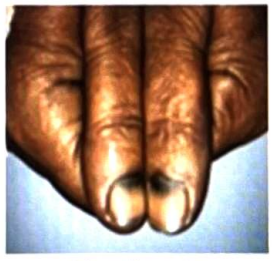
Beaus lines 00:13:59

Lesion : Horizontal depressions over the nail plate.
Pathology : Due to temporary arrest of growth in the nail matrix.
Etiology : Chemotherapy, severe illnesses.


Beaus lines

Chromonychia/colored nails 00:14:56

Blue lunula/Azure lunula :
Associated with Wilson's disease and silver intoxication/Argyria.

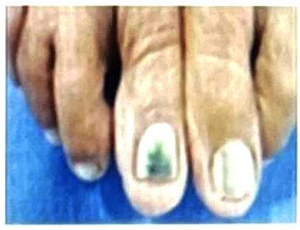

Blue lunula

Yellow nail syndrome : Triad of
1) Lymphatic system : Primary lymphedema.
2) Pulmonary system :
Chronic respiratory illness in the form of pleural effusion or bronchiectasis.
3) Yellow nails :

- Loss of cuticle.
- Thickening of the nail plate.
- Slow growing nails.


Yellow nails

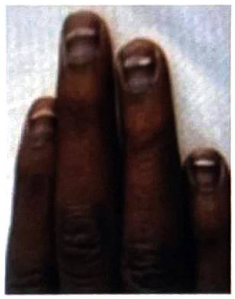
Green nail syndrome :
Seen in association with Pseudomonas infection.
It is due to pyocyanin pigment liberated from the Pseudomonas.



Green nail syndrome

Leukonychia/white nails :

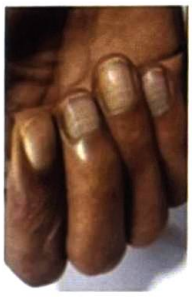
1) True leuckonychia : Problem is in the nail matrix.
E.g., mees lines : Horizontal, transverse whitebands on the nail plate.
Seen in chronic Arsenic poisoning.



mees lines

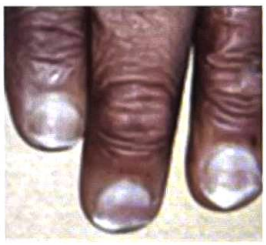
2) Apparent leukonychia : Problem is in the nail bed.

- muehrcke's bands.
  Lesion : Paired transverse white bands.
  Seen in hypoalbuminemia.



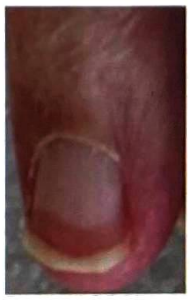
muehrcke's bands

- Half and half nail/Lindsay nail.
  Proximal (50%) : White.
  Distal (50%) : Brown to pink.
  Association : Chronic renal failure.



Lindsay's nail

- Terry's nails
  Proximal (90%) : White.
  Distal (10%) : Pink to brown.
  Association : Chronic liver disease.



Terry's nail

Active space

BULLOUS DISORDERS : PART - 1

Lesions of bullous diseases

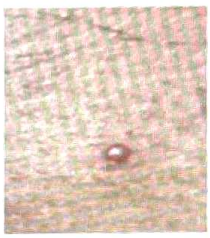
00:00:24

Primary lesions :

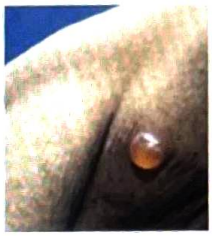
Clear fluid filled lesions.

Vesicles : < 1cm.

Bulla : > 1cm.



Vesicle

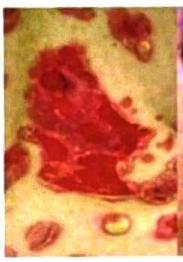


Bulla

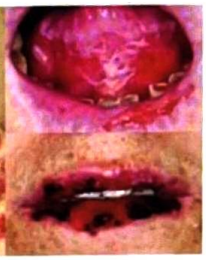
Secondary lesions :

Erosion : Focal or total loss of epidermis.

Crust : Hard exudate due to drying of serum, pus or blood.



Erosion



Crust

Classification of bullous disorders :

1. Immunobullous disorders.
2. Mechanobullous disorders.
3. Inherited acantholytic disorders.

Immuno bullous disorders (immuno = autoimmune) :

Bullous disorders which are mediated by autoantibodies.

Classification : Based on the level of the split in the skin :

- Intra epidermal blistering disorder (superficial)
  - Pemphigus group of disorders
- Subepidermal blistering disorders (deeper)
  - Pemphigoid group
  - Dermatitis herpetiformis

Pemphigus group of disorders

00:04:14

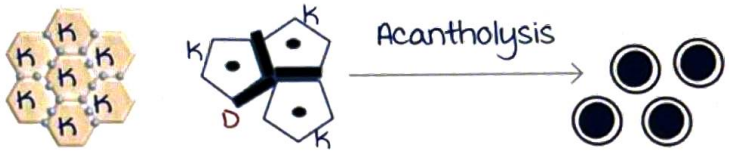
Immunobullous group of disease.

Level : Intraepidermal.

Target protiens : Desmosomes (connects Keratinocytes to each other).

Acantholysis is the process by which desmosomes get ruptured.
Keratinocytes are polyhedral shaped cells of epidermis.
Desmosomes : Protein connecting Keratinocytes to each other.

Acantholysis (acantha=spine, lysis=loss) : Loss of attachment between the Keratinocytes. Prominent in Stratum spinosum.
Antibodies attack desmosomes → Acantholysis → circular Keratinocytes (prominent hyperchromatic nucleus with perinuclear halo) → acantholytic cells/Tzanck cells on Tzanck smear.



Polyhederal Keratinocytes

Circular Tzanck cells/ acantholytic cells.

Classification of pemphigus group of disorders :

1. Pemphigus foliaceus and its variant pemphigus erythematosus.
2. Pemphigus vulgaris and its variant pemphigus vegetans.
3. IgA pemphigus.
4. Drug induced pemphigus.
5. Paraneoplastic pemphigus.

Target antigen in the desmosomes in different pemphigus disorders :

- Desmoglein : P. foliaceus, P. vulgaris.
- Desmocollin : IgA pemphigus.
- Plakins (desmoplakin, periplakin, envoplakin) : Paraneoplastic pemphigus.

Pemphigus foliaceous and pemphigus vulgaris 00:10:21

Common features :
Disease : Immunobullous disorders.
Level : Intraepidermal split.

Target protien : Demosomes.

Target antigen : Desmoglein.

- Pemphigus foliaceus : Desmoglein-1.
- Pemphigus vulgaris : Demoglein-3 > Demoglein-1.

Expression of desmoglein 1 and 3 (skin/mucosa) :

	Desmoglein-1	Desmoglein-3
Skin	Upper epidermis	Lower epidermis
Oral mucosa	Low concentration	High concentration

↓ ↓

	P. Foliaceus	P. Vulgaris
Skin split	Subcorneal split	Suprabasal split
Mucosa	No oral lesions	++ in 90-95% cases

Pemphigus vulgaris

00:15:12

Epidemiology : Most common type of pemphigus.

It is an immunobullous disease.

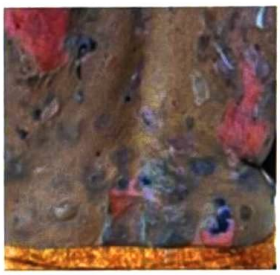
IgG autoantibody mediated disease.

Target protein : Desmosomes.

Target antigen : Desmoglein-3 > 1.

Pathological process : Acantholysis.

Age group : 40-60 years.



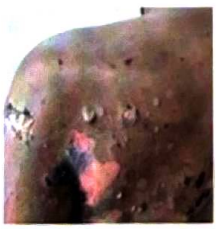
Symptoms : Burning sensations over the raw areas (erosions).

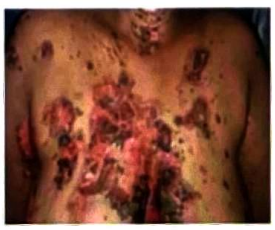
Morphology :

Primary lesions : Flaccid bullae over the normal skin (flaccid because split is superficial).

↓ ruptures because of thin roof.

Secondary lesions : Erosions (tend to extend because of acantholysis/no tendency to heal).





Sites : Scalp, oral mucosa, chest, trunk, flexures.

Intraepidermal : Flaccid bulla with wrinkles (less tension).

Subepidermal group : Tense bulla.

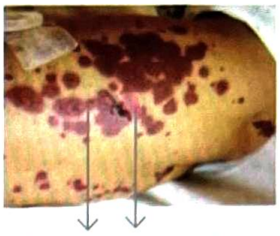
Active space

Clinical signs :

1. Nikolsky sign :

   Procedure : Tangential pressure is applied over the skin.

   Observation : Upper layers separate from lower layers.



   True Nikolsky sign : Acantholysis.

   Pseudo Nikolsky sign : Necrosis of keratinocytes.

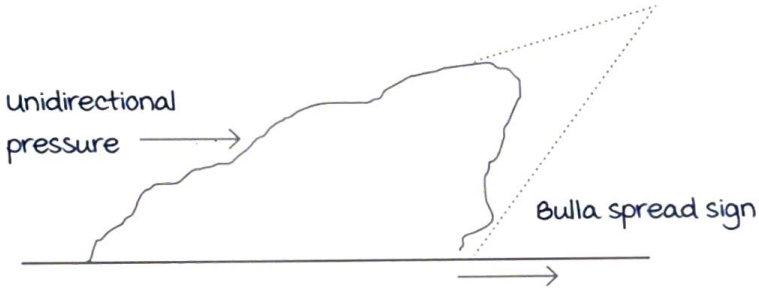
   In pemphigus vulgaris : True Nikolsky sign is positive.

2. Bulla spread sign :

   Procedure : Unidirectional pressure is applied over the bulla.

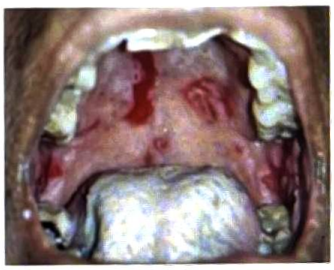
   Observation : Extension of margin of the bulla with an irregular angulated border.

   In bullous pemphigoid round regular border is seen.



3. Oral mucosa examination : Painful erosions.

   Seen in 90–95 % of patients with pemphigus vulgaris.



Investigations :

1. Tzanck smear of the vesicle.
2. Histopathology of bullae by skin biopsy.
3. Direct immunoflouroscence.

Active space

Tzanck smear

00:25:18

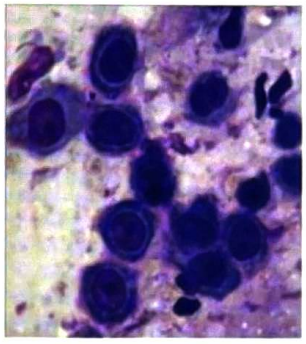
Vesicle is deroofed with a blade and the material from the bulla is taken.

It is a cytodiagnostic test (based on cell morphology).

Round acantholytic cells/Tzanck cells are seen.

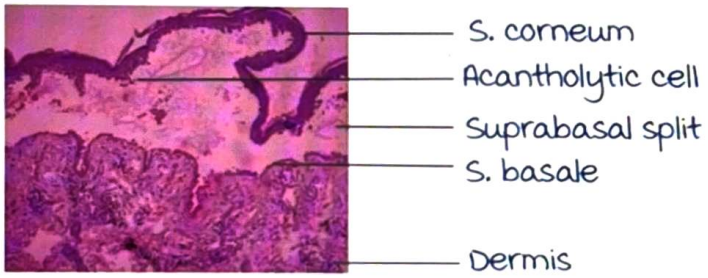
Histopathology of skin :

- Suprabasal split.
- Few rounded cells in the blister cavity : Acantholytic cells.
- In stratum basale, keratinocytes remain separated from each other but attached to the underlying dermis (row of tombstone appearance).



Tzanck smear with round acantholytic cell

Cells are tightly attached to the dermal layer as hemidesmosomes are intact.



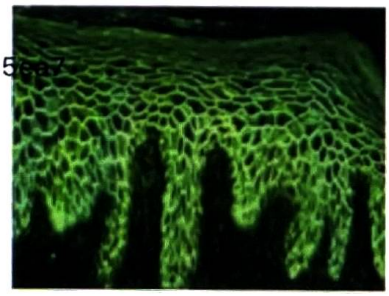
Row of Tombstone appearance

Investigation of choice :

Direct immunofluorescence (DIF)

To detect intraepidermal and intercellular deposition of IgG and C3 (pathogenic autoantibodies).

Shows Fishnet/chicken-wire pattern.



Fishnet pattern on DIF

Active space

Treatment of pemphigus vulgaris :

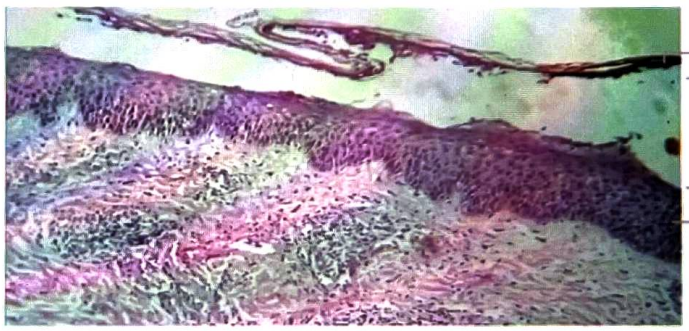
- Systemic steroids are the main treatment modality.
- Cyclophosphamide : Monitor urine routine.
  (side effect : Hemorrhagic cystitis by acrolein metabolite. Treat with MESNA/mercapto Ethyl Sulfonate Sodium).
- Rituximab (FDA approved in 2018)
  For moderate to severe pemphigus vulgaris.
  Anti-CD20 (CD20 on B cells).

Pemphigus foliaceus

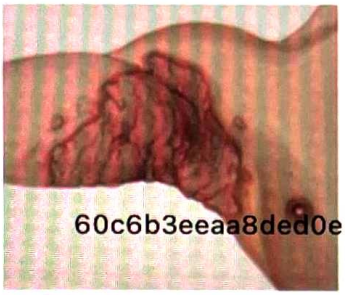
00:33:45

Target antigen : Desmoglein-1.
Skin lesions : Transient superficial bulla (lasts for very short interval and rupture rapidly, resulting in scaling and crusting).
Distribution : Initially involves only the Seborrhoeic areas (rich in sebaeceous glands) like chest, presternal area, scalp.
Later, evolves to form erythroderma/ exfoliative dermatitis
(> 90% of BSA should be involved).
Mucosa : Normal.
Histopathology : Subcorneal split with small acantholytic cells.

Erythroderma



Pemphigus vegetans 00:37:18



Rarest form of pemphigus.
Variant of pemphigus vulgaris.
Characterized by vegetative plaques.
Usually seen over the flexures (axilla, groin).
Split : Suprabasal.
Eosinophilic micro abscess on histopathology.

Drug induced pemphigus 00:38:29

Drugs which can induce pemphigus :

- D-penicillamine
- Captopril.
- Rifampicin.

Paraneoplastic pemphigus 00:38:58

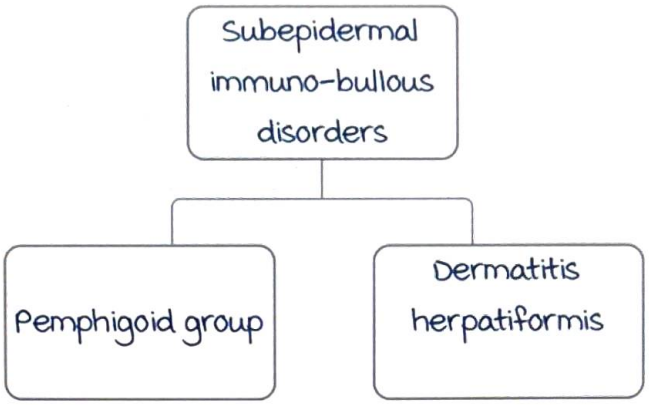
Antigens are the plakins (desmoplakin/periplakin/envoplakins).
Malignancies which can trigger pemphigus :

- Non-Hodgkin's lymphoma (most common).
- CLL (Chronic Lymphocytic Leukemia).
- Castleman's disease.
- Thymoma.

Active space

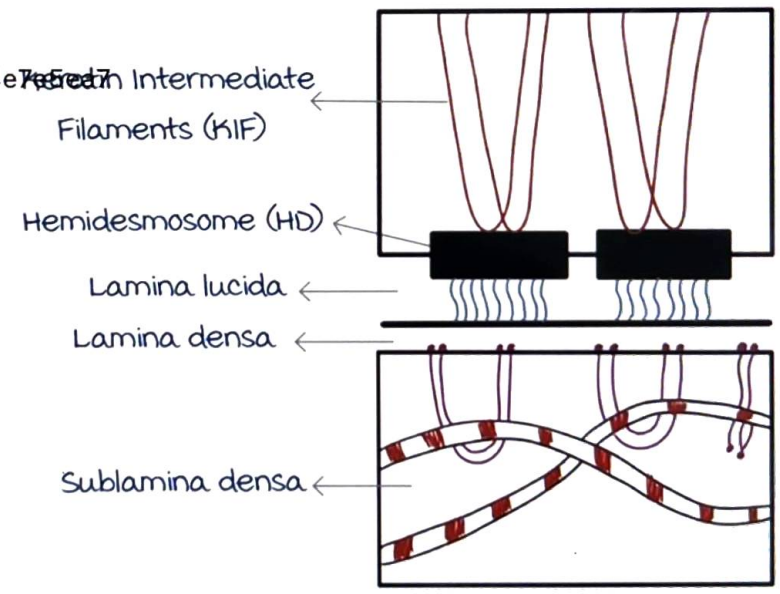
BULLOUS DISORDERS : PART - 2

Subepidermal immuno-bullous disorders 00:00:29



Pemphigoid group : Defect in the basement membrane zone proteins.

Basement membrane zones :



Basement membrane zone components :

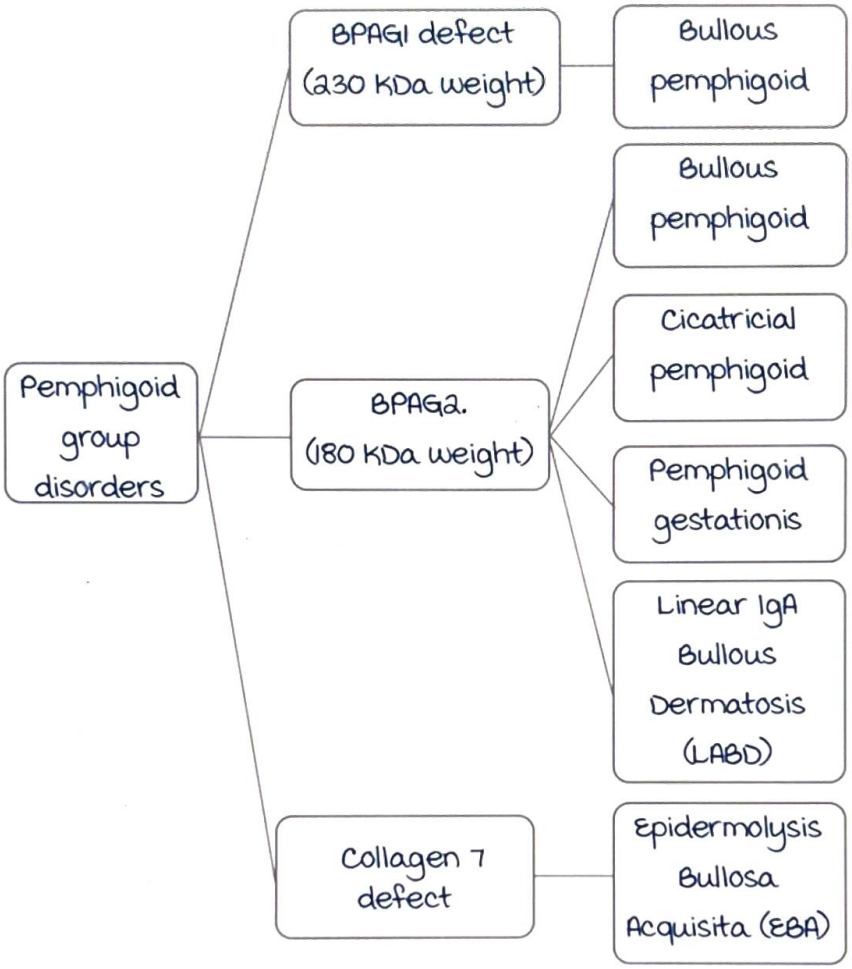
Zones	Components
Keratin intermediate filaments – Hemidesmosomes complex	Keratin 5/14, BPAG 1 & 2.
Lamina lucida	BPAG 2 (transmembrane molecule), laminin.

Active space

Lamina densa	Laminin, collagen 4
Sub lamina densa	Collagen 7 (anchoring fibrils)

BPAG 2 : Transmembranous as it is present in zones 1 & 2.

Classification based on defect :



Bullous pemphigoid

00:06:15

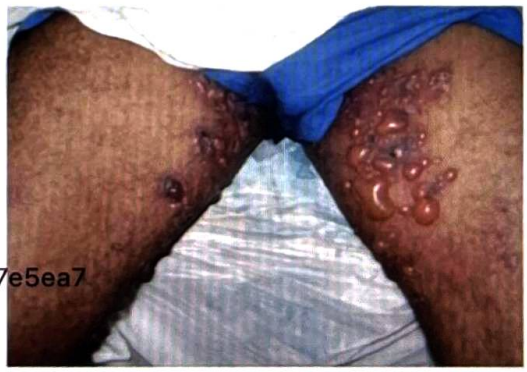
Immuno-bullous disease.

Characterized by subepidermal split.

IgG is the autoantibody deposited.

Target protein : Hemidesmosomes

Target antigens : BPAG2 and BPAG1.



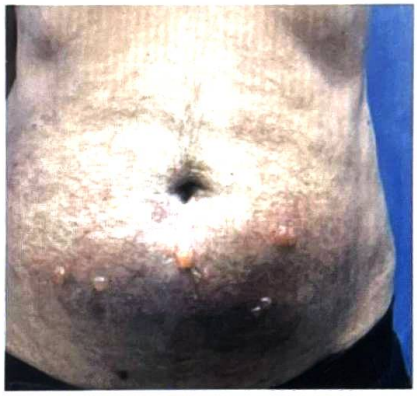
Active space

No acantholysis. (Acantholysis occurs in pemphigus group of diseases).

Disease profile : 60 to 80 years of age (males = females)

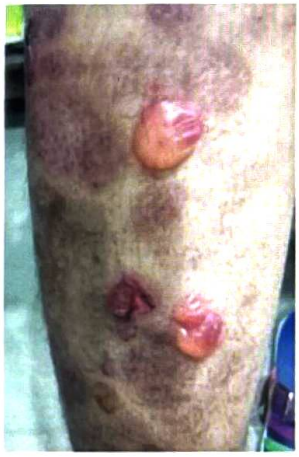
Morphology :

- Primary lesions :
  Tense itchy bullae on red erythematous skin or normal skin.
- Secondary lesions :
  Erosions, following the rupture of bullae due to scratching.



Tend to heal.

No tendency to extend.

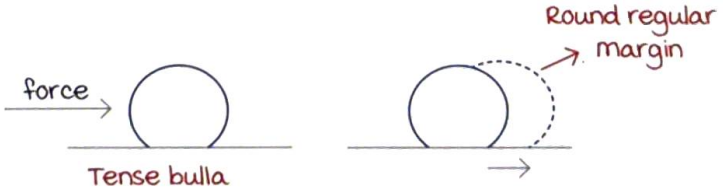


Sites : Trunk, abdomen, flexural aspects of extremities.

10–30% of patients have oral erosions.

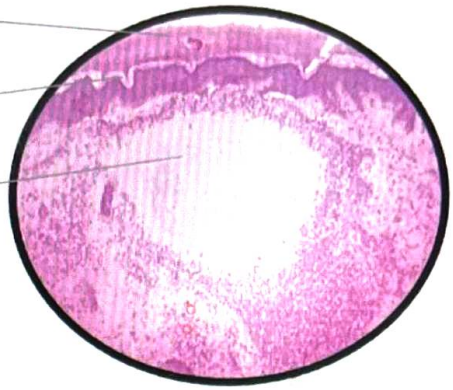
Signs in bullous diseases :

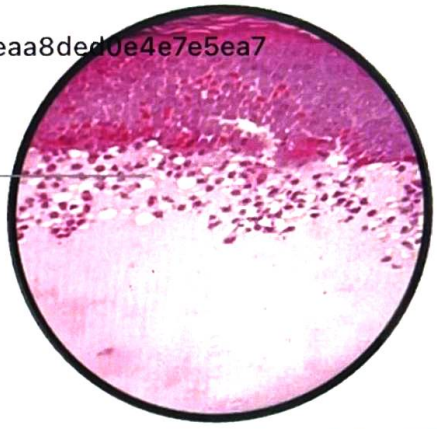
- Nikolsky sign : Apply tangential pressure over the skin.
  Negative here.
  Positive in pemphigus vulgaris due to acantholysis.
- Bulla spread sign : Margin of the bulla extends to adjacent skin, when force applied from the side.
- Asboe Hansen sign : Pressure applied from the top.

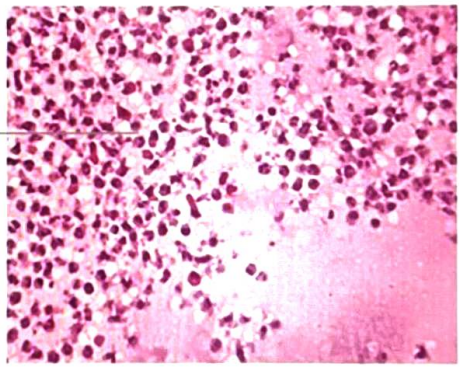


Investigations : Tzanck smear :
Presence of eosinophils.
Absence of acantholytic cells.

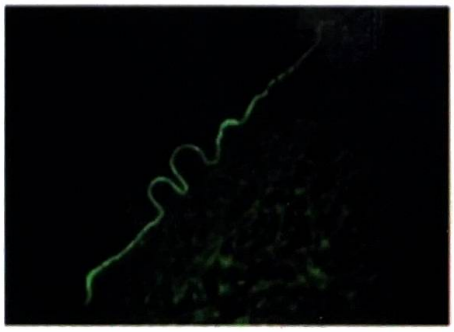
Histopathology :







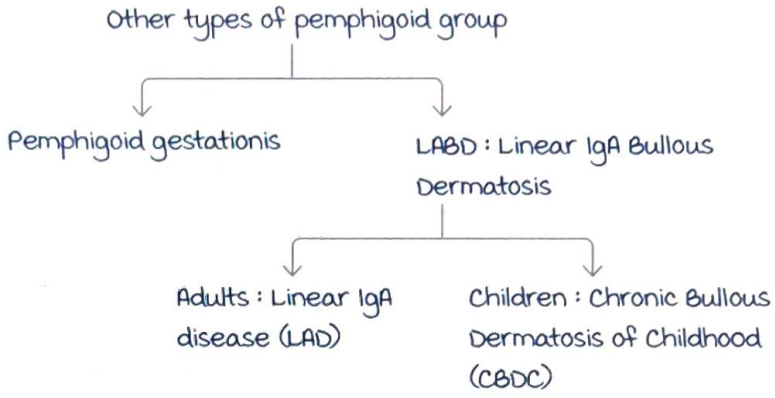
Direct Immunofluorescence :
Detects the pathogenic autoantibody, using immunofluorescence technique.
Autoantibody : IgG, C3.
Site : Basement membrane zone.
Pattern : Linear.



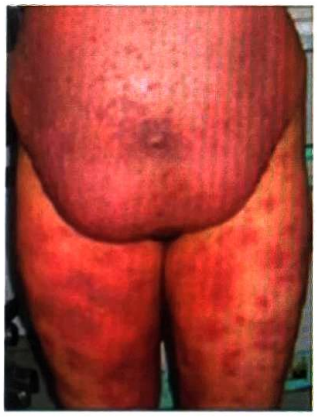
Treatment : Low dose oral steroids.

Other types of pemphigoid group

00:15:58



Pemphigoid gestationis :



Also called Herpes gestationis (Milsmmer).

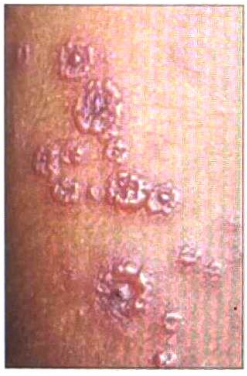
Bullous pemphigoid in pregnancy.

Target antigen : BPAG2.

Morphology of lesions :

- Urticarial plaques & tense bullae in Periumbilical region.
- Extends to thighs and abdomen.

Treatment : Systemic steroids.



Chronic Bullous Disease of Childhood (CBDC) :

Autoantibody involved is IgA.

Presents in children.

Morphology : Annular arrangement of vesicles.

Cluster of jewels/string of pearls appearance.

Distribution : Perioral, perineum.

Treatment : Dapsone (DOC), inhibits neutrophil chemotaxis.

Dermatitis herpetiformis

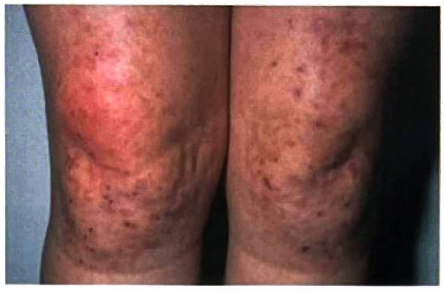
00:21:43

Chronic, relapsing, pruritic subepidermal immuno-bullous disease. Also known as Duhrings disease.

Autoantibody : IgA.

Target antigen : Epidermal transglutaminase 3.
The antigen is at the epidermis. It moves into the subepidermal level when IgA antibody comes in contact with it and presents with the bullae in the dermis.
HLA associations : HLA-DQ2, HLA-DQ8.
Systemic associations : Gluten sensitive enteropathy.
Usually those with the enteropathy is asymptomatic.
Other symptoms : malabsorption, diarrhea, abdominal pain.

Course :
Chronic disease.
20-40 years of age.
M : F ratio is 2 : 1.

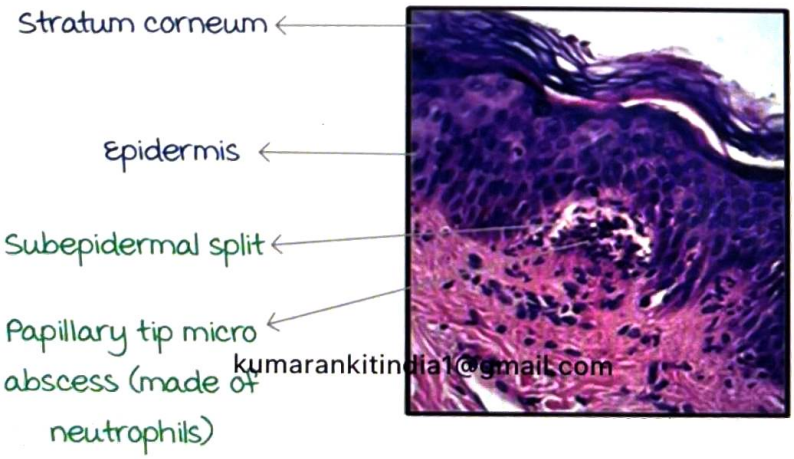


Morphology :
Primary lesions : Intensely pruritic papulo-vesicles.
Secondary lesions : Grouped excoriations.

Sites : Extensor & pressure points over the trunk and extremities.

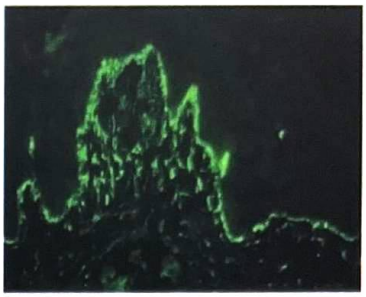
Investigations : Tzanck smear. Neutrophils are seen (Neutrophil chemotactic factor, IL 8 is elevated.)

Histopathology :



Active space

Direct immunofluorescence :
IgA is deposited at the basement membrane zone & dermal papilla.
Granular pattern.

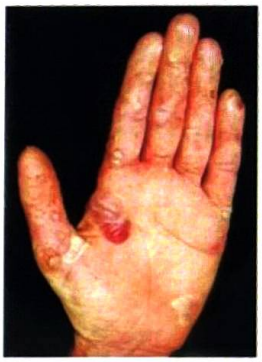


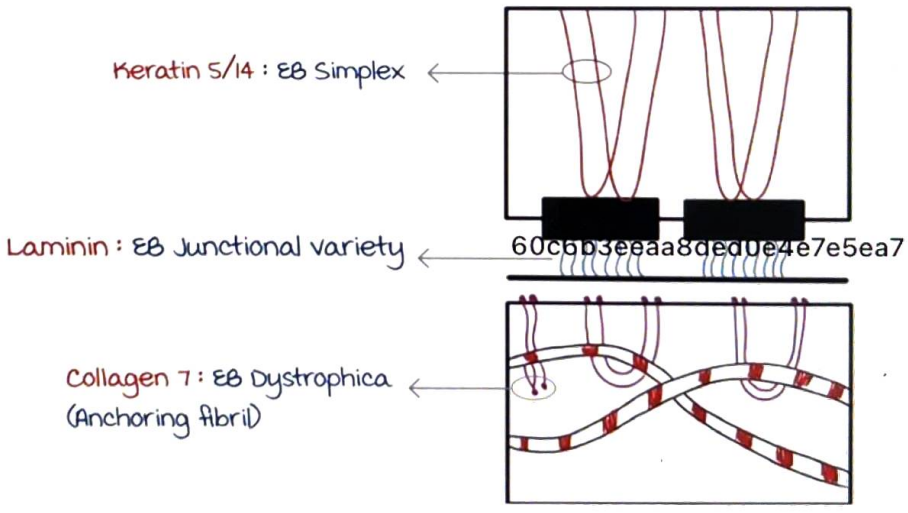
Treatment :
Gluten free diet.
Barley, rye, oats and wheat (BROW) should not be consumed.
DOC : Dapsone (inhibits neutrophil chemotaxis).

Mechano bullous disorders

00:31:09

Also known as Epidermolysis bullosa (EB).
Trivial mechanical trauma produces blisters/ bullae.
Usually genetic, except EBA.





How to identify mechano bullous disorder in MCQ ?
Genetic : Child born of consanguineous marriage (common).
Mechanical trauma induces blisters : Mother on handling the baby notices blisters.
Treatment : Gene therapy (definitive) and supportive management.

Active space

Inherited acantholytic disorders 00:36:13

- Hailey Hailey Disease (HHD).
- Darier disease.

Both are autosomal dominant.

Defect in calcium ATPase, within the desmosomes.

Process :

Acantholysis (loss of attachment between the keratinocytes) predominates in HHD.

Dyskeratosis (abnormal premature keratinisation) predominates in Darier disease.

Genetics :

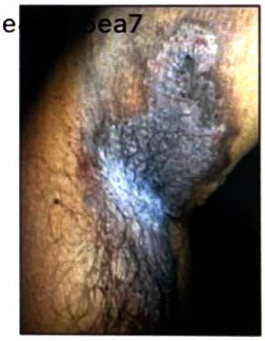
	Chromosome	Gene
HHD	3	ATP2C1
Darier disease	12	ATP2A2

Hailey Hailey disease :

Also known as Familial benign chronic pemphigus.

Primary lesions : Flaccid vesicles.

Secondary lesions : macerated plaques (soft and wet to touch) due to rupture of vesicles.



Site : Flexures of axilla, infra mammary areas, inguinal folds.

Nails : Longitudinal leukonychia (white longitudinal bands on the nails).

Darier disease :

Also known as Keratosis follicularis (misnomer).

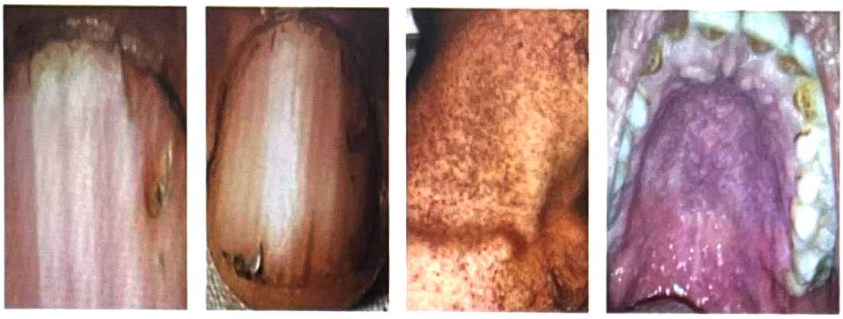
Lesions : Dirty warty greasy papules.

Sites : Seborrheic areas (sides of face and neck, pre-sternal area, upper back).

Nails : Red (erythronychia) and white bands (leukonychia) alternating longitudinally.

Free edges of the nail have a V shaped nick.

Active space



Oral cavity : Whitish papule seen generally over the hard palate, which coalesce to form Cobble stone appearance.

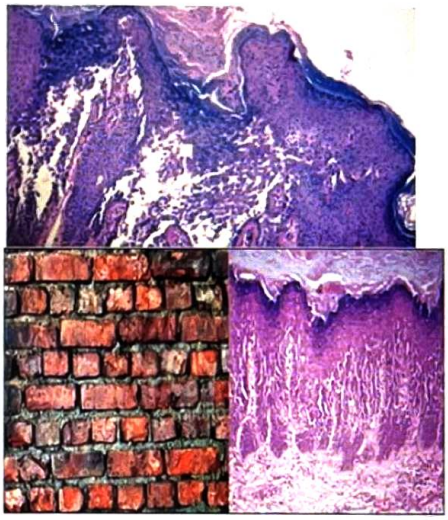
Palms : Pits in palms.

Histopathology :

Hailey Hailey disease :

Supra-basal split with partial acantholysis.

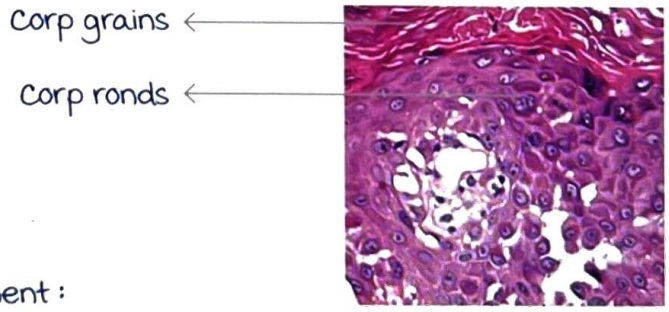
Dilapidated brick wall appearance.



Darier disease : Dyskeratotic cells seen.

Corp grains : Grain like, seen in upper epidermis.

Corp ronds : Rounded cells, seen in lower epidermis.



Treatment :

Hailey Hailey disease :

- Treatment of secondary bacterial and fungal infections.
- Topical steroids.

Darier disease :

Oral retinoids.

BACTERIAL INFECTIONS OF SKIN

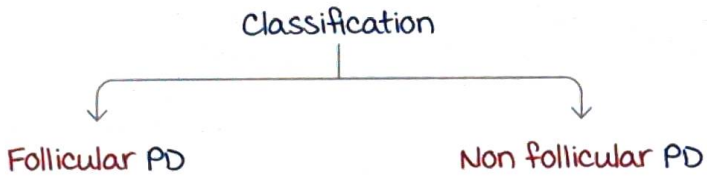
Pyodermas (PD)

00:00:40

Definition : Purulent bacterial skin infections (pyo = pus).

Classification :

Based on the involvement of hair follicles :



Follicular PD :

Hair follicles are involved.

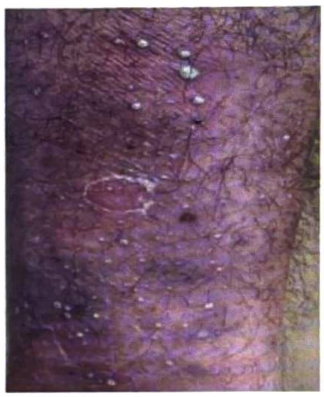
Etiology : Staphylococcus aureus.

Diseases :

1. Folliculitis :

   Infection of the hair follicle.

   Lesion : Folliculocentric pustule



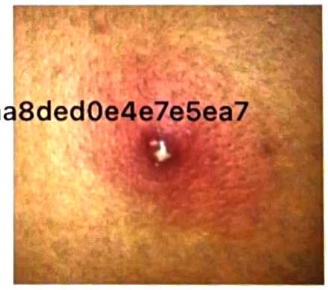
Folliculitis

2. Furuncle/Boil :

   Infection of the hair follicle + Peri follicular area.

   Lesion : Erythematous, indurated, tender nodule with a pus point.

60c6b3eeaa8ded0e4e7e5ea7



Furuncle/Boil with pus point.

3. Carbuncle :

   Infection of group of multiple hair follicles + contiguous (adjacent) area.

Lesion : Red, indurated, tender plaque with multiple discharging pus points.

Carbuncle

Active space

Most common site : Nape of the neck and upper back.

Most common association : Uncontrolled diabetes mellitus (RBS > 400-500 mg/dl).

Treatment :

Folliculitis and furuncle : Topical antibacterials.

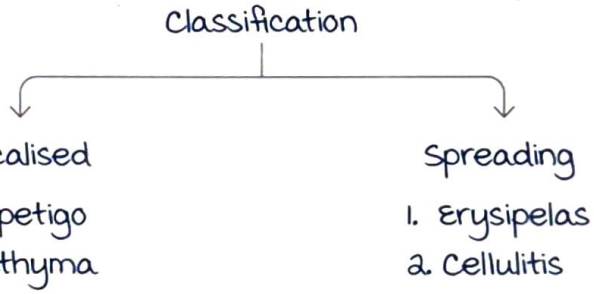
Carbuncle :

- Treat diabetes with insulin.
- Parenteral antibiotics.
- Incision and Drainage + Surgical debridement of affected area.

Non follicular PD

00:08:35

Hair follicles are not involved.



Localised :

Impetigo : It is the bacterial infection of the epidermis.

Classification :

1. Non-bullous impetigo.
2. Bullous impetigo.

Non-bullous impetigo/Impetigo contagiosa :

more contagious than bullous impetigo.

Etiology : Group A streptococci and staphylococcus aureus.

Seen in Children.

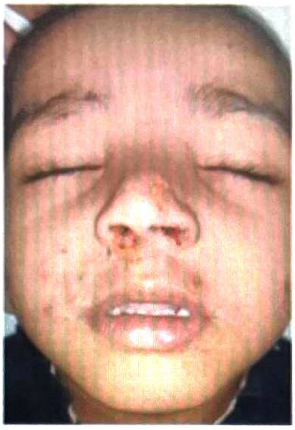
Site : Face (Predilection towards perioral, perinasal areas).

Lesion : Golden brown or honey coloured crust.

Remote complication : Post-streptococcal glomerulonephritis.

Seen 1 to 2 weeks post-streptococcal impetigo.

The child presents with fever, hypertension, edema and hematuria. It is a serious complication. Treat without delay.

Bullous impetigo :

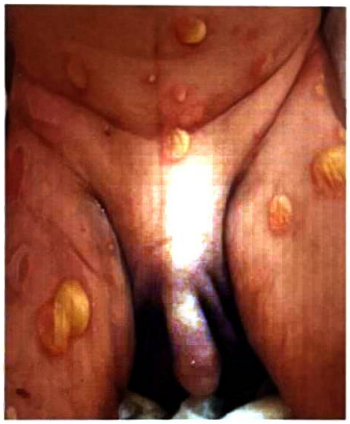
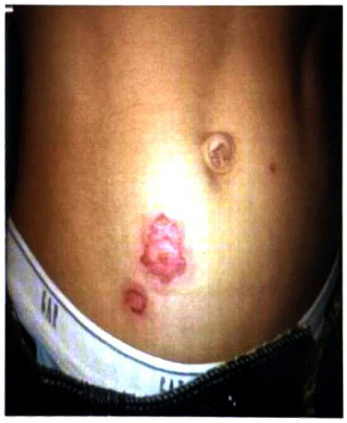
Etiology : Always Staphylococcus aureus.

Toxin : Exfoliative toxin A.

Lesion : Superficial flaccid bullae ruptures to give varnish coloured thin crust.

Hypopyon sign : Pus settles in the lower half of the bulla due to gravity.

Site : Trunk.

Flaccid bullae : Hypopyon sign Varnish crust on trunk

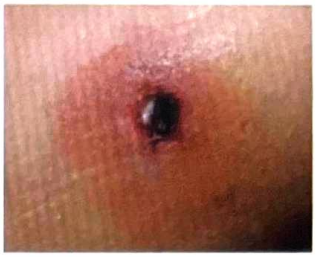
Management :

Topical antibacterials :

1. Fusidic acid.
2. Mupirocin : Inhibits methicillin-resistant Staphylococcus aureus (MRSA), therefore used to eradicate nasal carriers of MRSA (Staphylococcus aureus colonize nasal cavity).
3. Retapamulin.
4. Ozenoxacin : Quinolone drug, inhibits MRSA. More effectivethan mupirocin. Currently available in India.

Active space

Ecthyma :

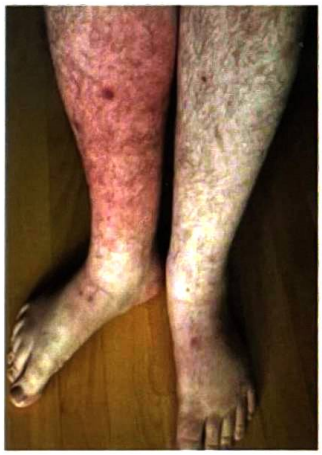


Definition : Deeper/ulcerative variant of impetigo.
Etiology : Group A streptococci.
Most common site : Lower extremities.
Lesion : Hard adherent chocolate brown crust.
Upon removing the crust : Purulent irregular ulcer is seen.

Spreading infections

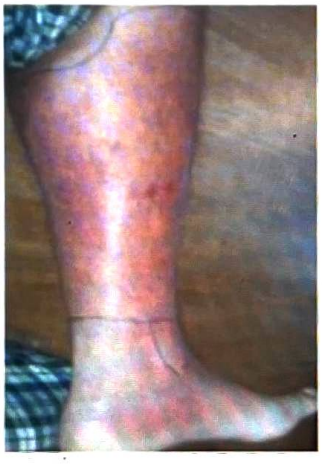
00:19:35

Erysipelas :
Level : Upper half of the dermis + lymphatics (superficial).
Etiology : Group A streptococci.
Sites : Lower extremities, face and progresses to pinna of ear (milian's ear sign).
Lesions : Erythematous indurated tender plaque.
Margin : Well defined, raised margin with clear demarcation between involved & normal skin



Erysipelas

Cellulitis :
Level : Lower half of the dermis + subcutaneous tissue.
Etiology : Group A streptococci, Staphylococcus aureus (rare).
Sites : Lower extremities.
Lesions : Red indurated tender plaque.
Margin : Ill-defined, diffuse (as subcutaneous fat is involved & is a loose compartment causing lateral spread).



Cellulitis

In spreading infections, streptococcus is commonly involved.

Treatment of spreading infections : Parenteral antibiotics.

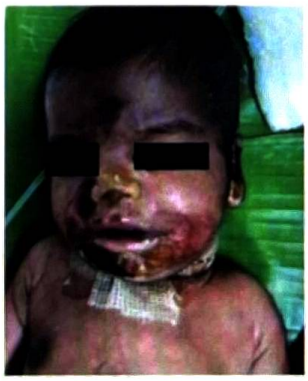
Staphylococcal scalded skin syndrome (SSSS) 00:25:20

Definition : Acute skin exfoliation secondary to toxin producing strains of Staphylococcus aureus (scald = burn).
Etiology : Staphylococcus aureus.

Disease process : Acantholysis (sub corneal split).
Source of Infection : Distant staphylococcal infection in ear or conjunctiva.

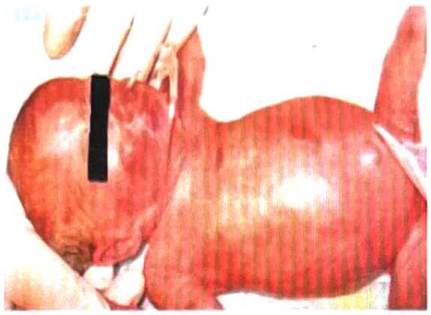
Epidermolytic toxin (exotoxin) produced from the foci spreads hematogenously & target the skin.
Target protein : Desmosomes in the epidermis.
Target antigen : Desmoglein-1 (DSG-1) in upper epidermis.

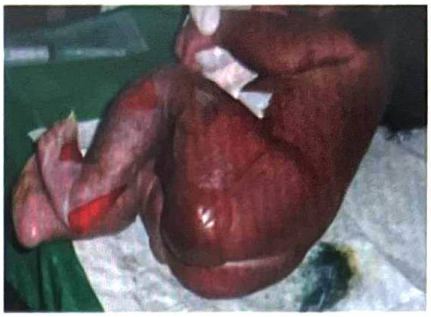
Distant source of infection
Conjunctiva/Ear
↓ Staph aureus
Epidermolytic toxin (exotoxin)
↓ Blood
Targets skin
↓
Target Protein : Desmosomes
↓
Target Antigen : Desmoglein-1 (upper epidermis)
↓
[illegible] split)



History of distant infection like conjunctivitis/otitis media.
Constitutional symptom like fever.
Lesions : Initially, periorificial erythema, skin tenderness (due to acantholysis).
If untreated, progresses to epidermolysis (sheet like epidermal peeling).

Active space





True Nikolsky's sign (acantholysis) :
Apply tangential pressure over the skin, upper layers of epidermis separates from lower layers.

Mucosa : Normal (because DSG-1 is expressed in low concentration in the oral cavity).

Differential diagnosis :

Sheet like epidermal peeling is seen in

- SSSS → Normal mucosa
- Toxic epidermal necrolysis → Mucosal involvement is prominent with hemorrhagic crusts.

Treatment of SSSS :
Penicillinase resistant penicillins :

- IV Nafcillin.
- IV Oxacillin.

Alternatives:

- IV Cefazolin.
- IV Vancomycin.

Acute paronychia, Botryomycosis, Erysipeloid

00:34:19

Acute paronychia :
It is an acute infection of the nail folds.
Etiology : Staphylococcus aureus.

Predisposing factors : Thumb sucking/nail biting.

Lesion : Initially erythema around nail folds.

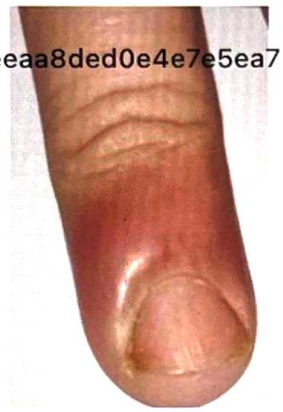
Later, pus collection/purulent discharge at nail folds.

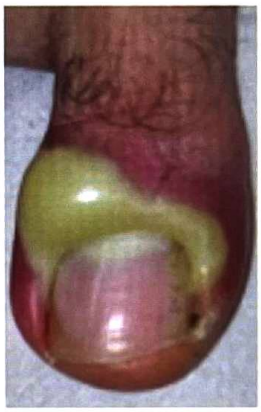
Treatment :

Without abscess : Topical antibacterials.

Abscess formation : Surgical incision & drainage.

60c6b3eeaa8ded0e4e7e5ea7

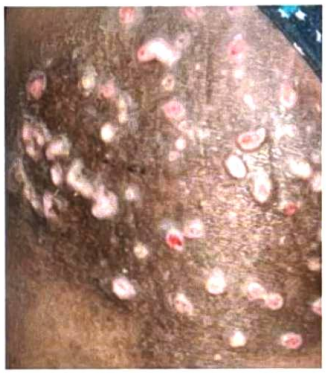




Botryomycosis :

Misnomer : It is not a fungal infection.

It is a chronic suppurative granulomatous response secondary to a bacterial pathogen.



most common etiology :

Staphylococcus aureus.

Other organisms : Proteus, E.coli, Klebsiella.

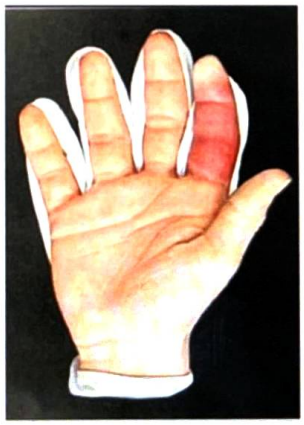
Erysipeloid :

Etiology : Erysipelothrix rhusiopathiae.

Occupational dermatoses : meat/fish handlers.

Lesion : Purplish erythema with painful edema.

Treatment : Penicillin.



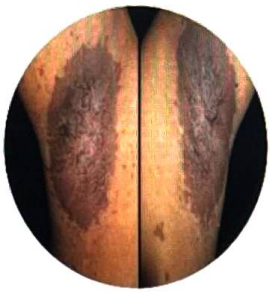
Cutaneous corynebacterial infections 00:39:21

- Erythrasma
- Trichomycosis axillaris
- Pitted Keratolysis

Active space

Erythrasma :

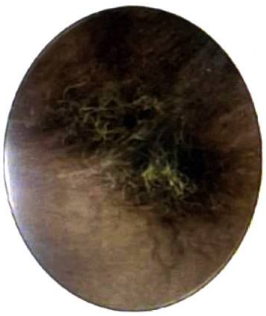
Etiology : Corynebacterium minutissimum.
Lesion : Asymptomatic hyperpigmented macules.
Sites : Axilla, inguinal region, toe-web spaces (predilection to folds).
Woods lamp : Coral red fluorescence (due to coproporphyrin-III).



Treatment :

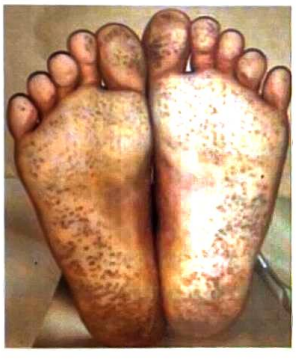
- Fusidic acid.
- Erythromycin (macrolide).

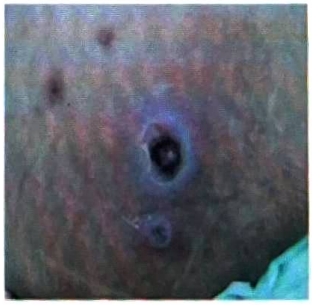
Trichomycosis axillaris :



Misnomer : Not a fungal infection.
Etiology : Corynebacterium tenuis.
C/F : Yellow concretions/deposits over hair shafts.
Site : Axilla.
Treatment : Clipping/removing the affected hair.

Pitted Keratolysis :
Etiology : Micrococcus/Kytococcus sedentarius.
C/F : Pits or depressions in Stratum corneum (due to keratolysis).
Sites : Soles & palms (in those who sweat a lot).
Treatment : Whitfield's ointment (3% salicylic acid + 6% benzoic acid).





Pseudomonas infections

00:44:32

Ecthyma gangrenosum :

It is due to the haemorrhagic necrosis of the skin. Seen in patients with Pseudomonas septicemia.

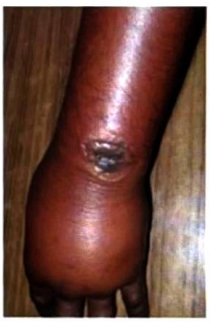
Necrotic crusted ulcers are seen.

Hot tub folliculitis by pseudomonas :

Bathing/shower in an ill-maintained spa.
Sites : Bathing area/trunk.

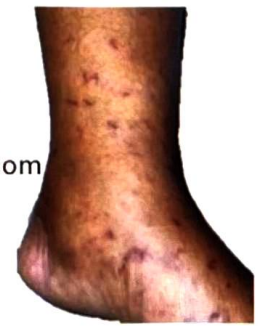
Green nail syndrome :
Discoloration of nail is due to pyocyanin pigment produced by pseudomonas.

Cutaneous anthrax :
Etiology : Bacillus anthracis.
Lesion : Malignant pustule (misnomer).
Starts as painless papule, progresses to an eschar surrounded by rim of vesicles.



Meningococcemia :
Etiology : Neisseria meningitidis.
Presents with fever + signs of meningitis like neck rigidity.
Lesion :

- Petechial rash/purpuric lesion (does not blanch when pressed with slide).
- Angular cutaneous infarcts.



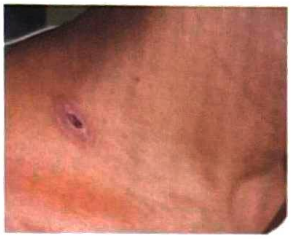
Scrub typhus

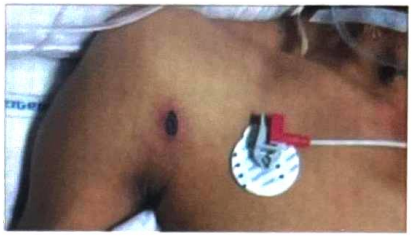
00:49:51

Definition : Acute febrile mite borne infectious disease.
Etiology : Orientia tsutsugamushi (tsutsuga : small but dangerous).
vector : Trombiculid mite.
Infective form of mite : Chiggers (Larval form).
Presents to emergency with fever, CNS symptoms.

Lesion : Eschar (black scab surrounded by erythema).

DOC : Doxycycline.





Only

~60 % of patients with scrub typhus will have an eschar.

But if present, it is diagnostic.

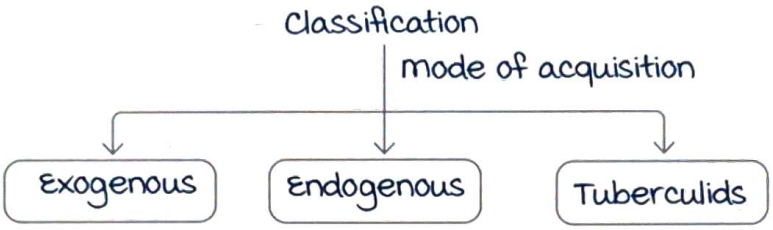
Differential diagnosis for Eschar :

1. Cutaneous anthrax.
2. Brown recluse spider bite (poisonous spider).

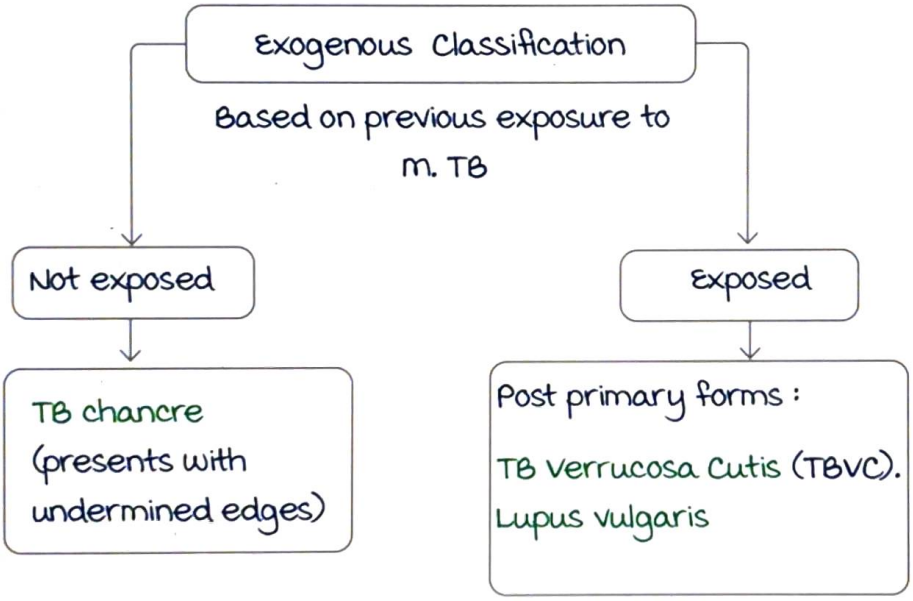
MYCOBACTERIAL SKIN INFECTIONS

Cutaneous tuberculosis 00:00:25

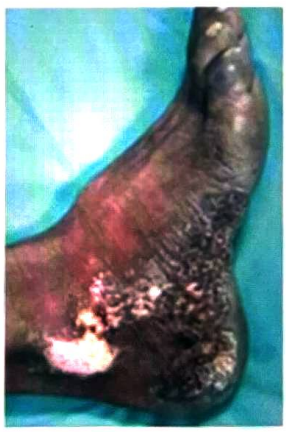
Caused by mycobacterium tuberculosis.



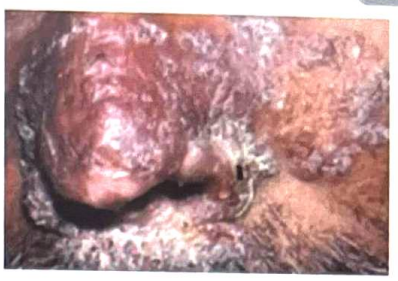
Exogenous Classification :



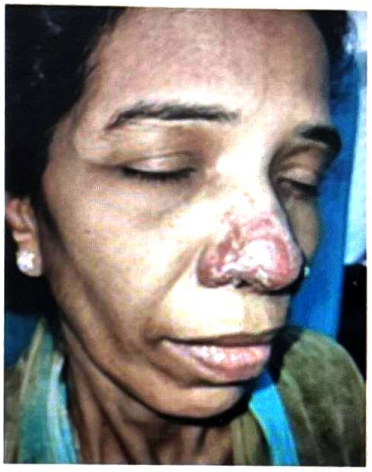
1. Tuberculosis Verrucosa Cutis (TBVC)/Warty TB/ Anatomist's Wart :
   Verrucose means rough or uneven surface.
   Seen in anatomists, due to handling of dead bodies, M.Tb gets inoculated on skin.
   Lesion : Indurated, verrucous plaque.
   Site : On distal or acral extremities.



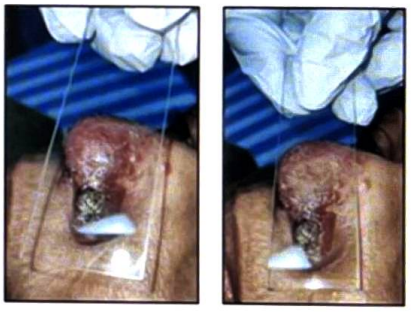
2. Lupus Vulgaris :
Most common form of cutaneous TB in adults.
Route of spread : Exogenous or endogenous.



This is paucibacillary form, since patient's cell mediated immunity is high.
Lesion : Annular infiltrated plaque (active margins).
Centre : Atrophy and scarring.
Periphery shows extension.
Sites : Face, buttocks and extremities.



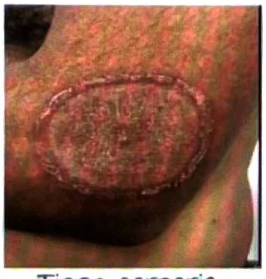
Diascopy :
Test where a glass slide is pressed over the lesion.
Lesion color changes to yellow brown color which are called apple jelly nodules.



Apple jelly nodules are a feature of granulomatous disease,

Differential diagnosis for annular plaque :

1. Centre is atrophied or scarred : Lupus vulgaris.
2. Central clearing, peripherally raised scaly margins with itch : Tinea corporis.
3. Central crusting lesion on face, patient from Jaipur : Cutaneous leishmaniasis.

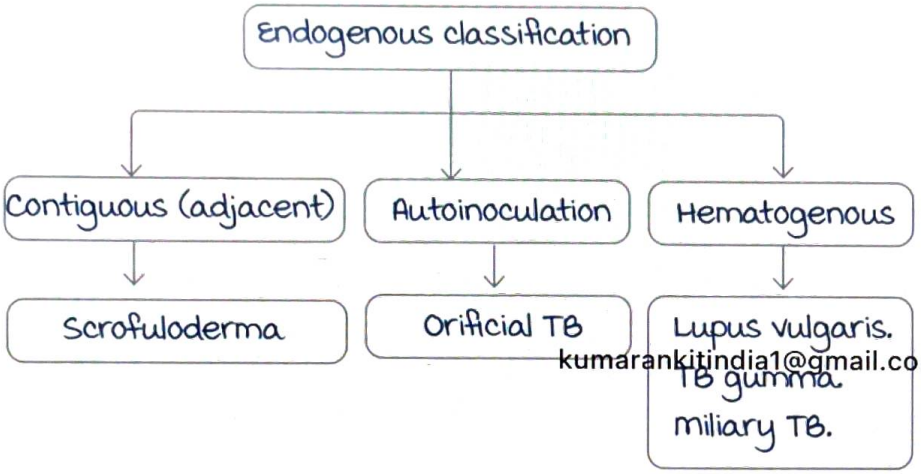


Tinea corporis

Cutaneous leishmaniasis

Endogenous classification of TB

00:10:35



1. Scrofuloderma :

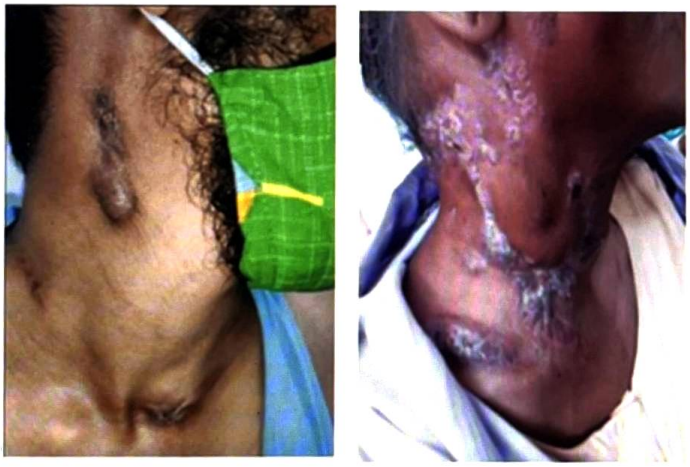
   most common form of cutaneous TB in children.

   multibacillary status.

   mode of spread : Direct extension of infection to the skin from an underlying TB focus (lymph node, bone, joint).

   most common focus of infection : Cervical lymph nodes.

   most common site : Neck and axilla.

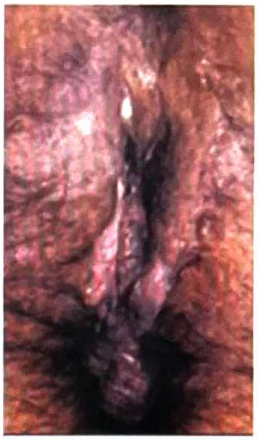


Lesion presents as :

Painless subcutaneous swellings → Ruptures into the overlying skin → Discharging sinuses/ undermined ulcers/ fibrotic scars

Active space

Orificial TB : multibacillary status.



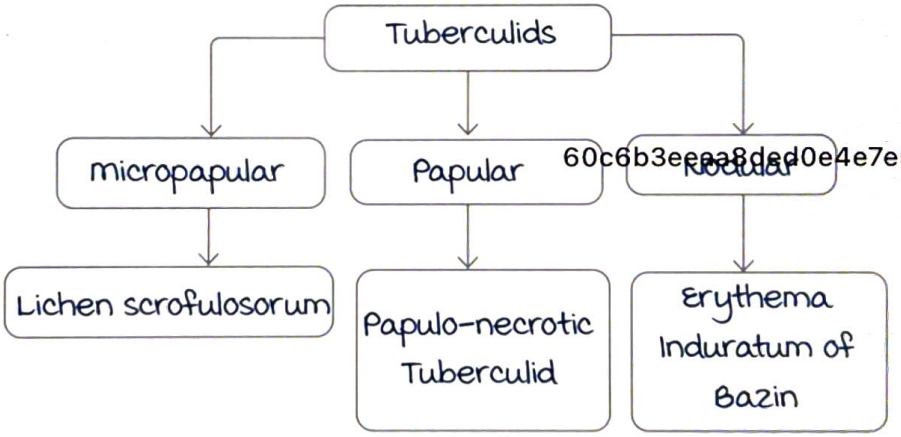
Defective cell mediated immunity
and have advanced forms of internal TB.
Internal disease :
Genitourinary, GIT, Pulmonary TB.
Auto-inoculation to mucocutaneous
tissues near body orifices.

Present as peri-orificial non healing ulcers.

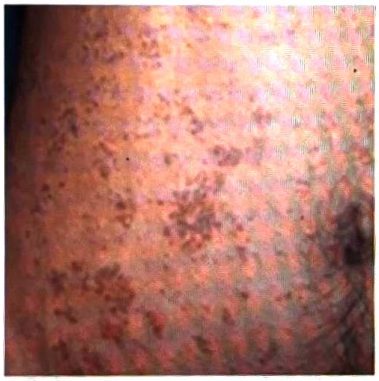
Tuberculids

00:17:12

Cutaneous hypersensitivity response to mycobacterium.
Cell mediated immunity is high.
Mantoux test turns positive.
Classification :



Lichen scrofulosorum :
Seen in children.
Asymptomatic lichenoid grouped
papules.
MC Site : Trunk.
Histopathology : Perifollicular
non-caseating granuloma.

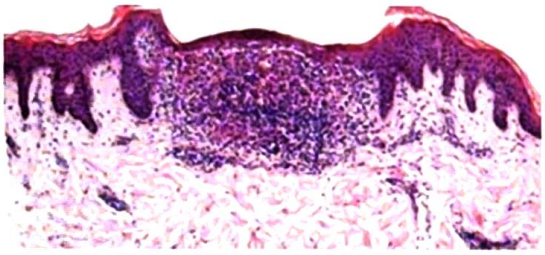


Differential diagnosis :
Lichen nitidus :
Asymptomatic flat topped, shiny papules.

Seen in children.

Site : Face, dorsum aspect of forearm, shaft of penis.

It has a characteristic histopathological pattern : Rete ridges (claw) & chronic inflammatory cells in the centre (ball) resembling "Claw clutching the ball" appearance.



Q. All the following are types of Lichen planus except?

60c6b3eeaa8ded0e4e7e5ea7

A. Hypertrophic Lichen Planus.

B. Lichen planus pigmentosus.

C. Atrophic Lichen Planus.

D. Lichen Scrofulosorum.

Answer : D

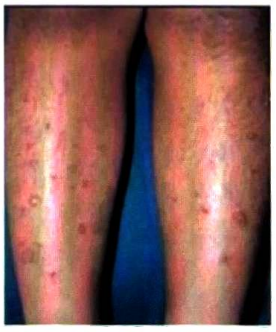
Lichen scrofulosorum is a type of Cutaneous tuberculid.

Papulonecrotic tuberculid :

Seen in young adults.

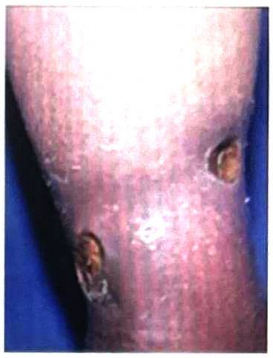
Lesions : Asymptomatic papules with central necrosis.

Site : Extensor of extremities, usually on lower extremity.



Active space

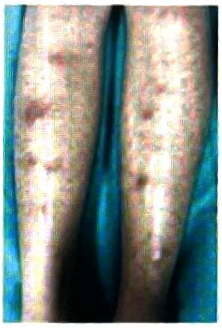
Erythema Induratum of Bazin :



Form of lobular panniculitis.
Lesion : Red, tender nodules.
Site : Posterior aspect of the leg.
Fate : Ulcers heal with scarring.

Differential diagnosis :

Erythema nodosum :

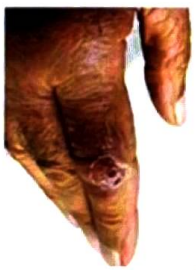


It is form of septal panniculitis.
Lesions : Red, tender nodules
Site : Anterior aspect of the leg.
Fate : Ulcers heal without scarring.

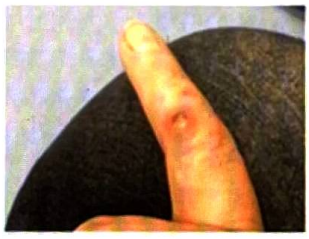
Treatment of cutaneous TB :
Antitubercular therapy.

Atypical mycobacterial skin infections 00:26:18

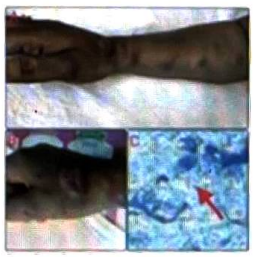
Swimming pool granuloma/Fish tank granuloma :
Acquired through occupational exposure to contaminated fresh water.
Causative organism : M. marinum.
Lesion : Nodulo-ulcerative lesion/nodular lesion with proximal extension to form a linear pattern of lesions along lymphatics of the extremities (linear pattern also seen in sporothrix).



An erythematous, scaly nodule over the right index finger.



Nodule on the index finger.

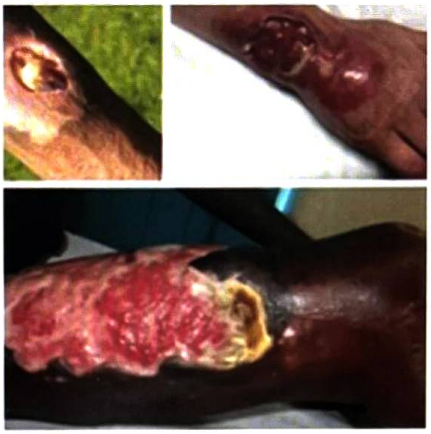


A. Sporotrichoid pattern of nodular rashes ;
B. large nodule which had ulcerated ;
C. Modified Ziehl- Neelsen staining showing acid-fast bacilli (red arrow).

Differential diagnosis for Sporotrichoid lesions :

1. Sporotrichosis.
2. Nocardiosis.
3. Cutaneous leishmaniasis.
4. Tularemia.

Buruli ulcer



Caused by : Mycobacterium ulcerans.
Toxin mediated ulcer.
Toxin : Myolactone, a cytotoxin which causes necrosis of the tissue.

Lesion : Painless ulcer with undermined edges.

Treatment :
Swimming pool granuloma : Minocycline
Buruli ulcer : Rifampicin + Clarithromycin for 8 weeks.
Inj. Streptomycin can be used in case of Clarithromycin intolerance.

VIRAL INFECTIONS OF SKIN

60c6b3eeaa8ded0e4e7e5ea7

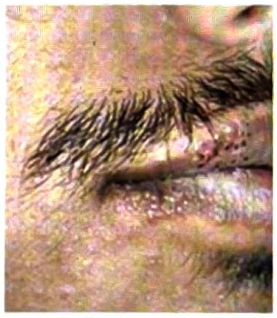
Herpes virus infections 00:00:36

Herpes : DNA virus.
It multiplies within the nucleus giving intranuclear inclusion bodies.
The most important property is latency : Lifelong persistent infection.

Herpes simplex virus-1 (HSV-1) 00:01:15

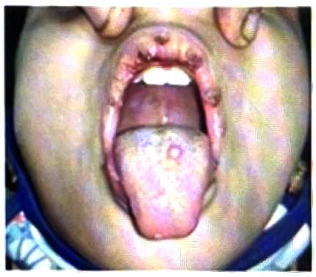
HSV I virus may cause the following infections :

- Orolabial herpes/cold sores/fever blister :
  Lesions : Multiple vesicles arranged in a grouped configuration at the lip margin.
  Treatment of choice : Tab Acyclovir 400mg TDS x 5 days.



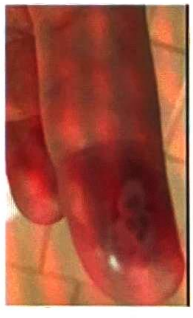
Orolabial herpes.

- Herpetic gingivostomatitis :
  Generally more common among children.
  Children present with high grade fever and rapidly spreading vesicular lesions that may rupture and cause erosions covered by a membrane. It has a propensity to involve the gingival mucosa and hence called as gingivostomatitis.



Herpetic gingivostomatitis.

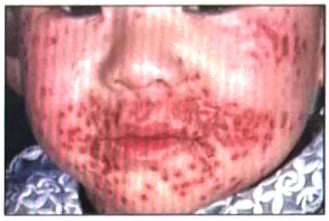
- Herpetic whitlow :
  Infection of distal pulp of finger.
  More common among dentists.



Herpetic whitlow

Active space

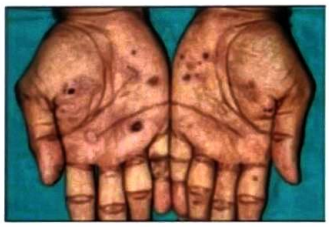
- Eczema herpeticum (Kaposi varicelliform eruption) :
  An acute disseminated cutaneous HSV I infection.
  It's usually due to a defective skin barrier e.g., in patients with atopic eczema, Darier disease or Sezary syndrome (leukemic form of cutaneous T-cell lymphoma).


Eczema herpeticum

  Patient presents with umbilicated vesiculo-pustules which may rupture to form punched out erosions covered by hemorrhagic crusts.
  Treatment of choice : Acyclovir,

Mild	Severe
Oral Acyclovir	IV Acyclovir

- Erythema multiforme :
  Example for cytotoxic dermatitis.
  Multiple viruses can produce this condition, but most common triggering factor is HSV I.
  Characteristic target lesions with 3 zones found over palms and soles.


Erythema multiforme

- Mollaret meningitis is the characteristic meningitis where HSV I is implicated.

Herpes simplex virus 2(HSV-2)

00:08:14

60c6b3eeaa8ded0e4e7e5ea7

HSV 2 virus causes genital herpes, a sexually transmitted infection.
There are multiple vesicles that rupture to form erosions that are grouped.
With time, the margins of these erosions begin to fuse with each other to form characteristic polycyclic margins.

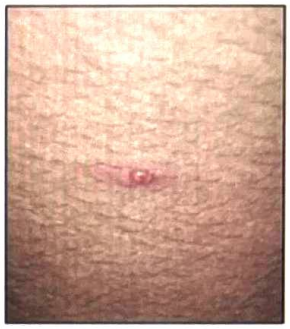

Genital herpes

Active space

Varicella Zoster virus (VZV)

00:09:08

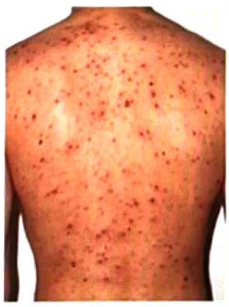
The primary infection is Varicella/ chicken pox.
On reactivation it can cause Herpes Zoster.



Vesicles on an erythematous base.

Varicella/Chicken pox :
Incubation period : 14 – 17 days.
Infectious period : 2 days before the rash develops till all the lesions have crusted.

Lesions : Vesicles that appear on an erythematous base (dew drops on rose petal appearance) is characteristic of a chicken pox rash.
Distribution of lesions : Predominantly over the trunk.
Pleomorphic rash : Characteristic of varicella.
Different stages of rash coexist together simultaneously, this is because the lesions occur as crops. The different stages of rashes in chicken pox are as follows :
Macules → Papules → Vesicles → Pustules → Scabbing.



Rash on trunk

Pleomorphic rash

Congenital varicella :

Timeline : Maternal infection at < 20 weeks of gestation.

Skin lesions :

- Cutaneous dermatomal scars.
- Limb atrophy.

Neonatal varicella :

Timeline : Maternal infection 5 days

before the delivery to 2 days after the delivery.

Skin lesions : Disseminated vesicular rash.

Herpes zoster (shingles) :

Acute cutaneous segmental eruption secondary to reactivation of VZV.

Pathogenesis of shingles :

Primary infection with VZV causes varicella, after which the virus lies latent in the dorsal root ganglion (sensory).

Factors that can reactivate this infection are :

- Old age.
- HIV
- Lymphoreticular malignancies.

These risk factors can reactivate and cause segmental eruptions called as herpes zoster.

Clinical features can be explained as phases :

1. Pre-eruptive phase :

Characterized by fever, malaise, excruciating burning pain within the dermatome before the rash appears.

2. Eruptive phase :

- Unilateral.
- Dermatomal (m/c dermatome is the thoracic dermatome).
- Painful grouped vesicles on erythematous base.

Complications : 60c6b3eeaa8ded0e4e7e5ea7

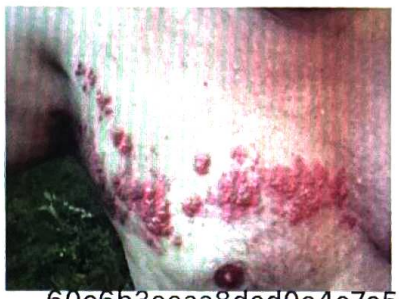
Post herpetic neuralgia is most disabling complication, patient presents with allodynia (non-painful stimulus like touch perceived as painful, due to involvement of nerve fibres).

Treatment of Herpes Zoster/Varicella : Acyclovir or Valacyclovir.

Acyclovir	Valacyclovir
Tab Acyclovir 800mg 1-1-1-1-1 x 7 days	Tab Valacyclovir 1g 1-1-1 x 7 days

Active space

Treatment of Post herpetic neuralgia : Any of the 3 drugs Amitriptyline, Gabapentin and Pregabalin can be given for long term treatment, usually from 6 months up to 1 year.



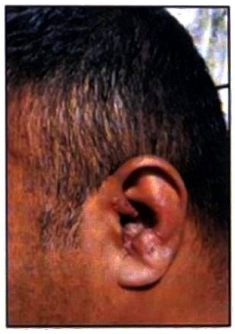
60c6b3eeaa8ded0e4e7e5ea7
Shingles/Herpes zoster

Variants of herpes zoster

00:21:25

Herpes Zoster Ophthalmicus	Herpes Zoster Oticus/Ramsay Hunt syndrome
Ophthalmic division of trigeminal nerve (V), usually the frontal branch.	It involves the geniculate ganglion.
Hutchinson sign of the nose : If there is a vesicle over the tip or side of the nose, it means there is an increased chance of corneal involvement. This is because both the top of nose/side of nose and cornea are supplied by the nasociliary nerve.	3 important features of Ramsay Hunt syndrome are : • Ipsilateral 7th nerve palsy. • Otalgia. • Vesicles over the external ear.

Herpes Zoster Ophthalmicus

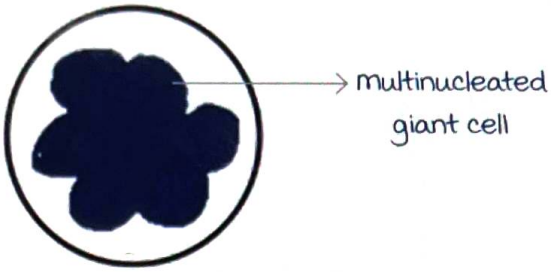


Herpes Zoster Oticus

Investigations for HSV-1, HSV-2, VZV :
As the most prevalent lesions are fluid filled i.e., vesicles, investigation of choice is Tzank smear.

Active space

Here, tzank smear shows multinucleated giant cells diagnostic of herpes infections.



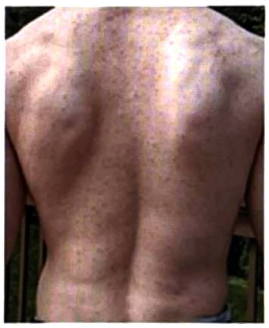
Infectious mononucleosis/glandular fever/ kissing disease

00:25:01

Triad of infectious mononucleosis :

- Fever.
- Post cervical lymphadenopathy.
- Pharyngitis.

On administering Ampicillin in these patients to treat pharyngitis they develop a rash called Ampicillin induced maculo-papular rash.


Ampicillin induced maculo-papular rash

Malignancies triggered by Epstein-Barr Virus (EBV) :

- Burkitt's lymphoma.
- Hodgkin's lymphoma.
- Nasopharyngeal carcinoma.

Site for nasopharyngeal carcinoma : Fossa of Rosenmuller.

Oral hairy leucoplakia (OHL) :
In HIV infected patient (with EBV), white corrugated/ridge-like plaque over lateral border of tongue that cannot be rubbed off is called as OHL.

HHV-5 : Cytomegalovirus (CMV) :
It produces infectious mononucleosis like illness.
'Owl eye' inclusion bodies.

HHV-6, HHV-7, HHV-8

00:28:45

HHV-6 :

Causes the 6th disease.

Also known as roseola infantum (roseola : Rose like, infantum : Seen in infants) or exanthem subitum (exanthem : Rash on the body, subitum : Sudden/abrupt).

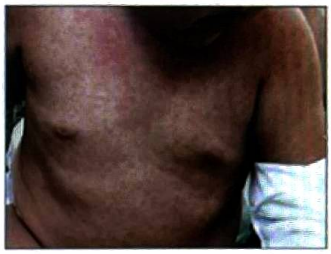
Patients present with abrupt onset of high grade fever.

Maculopapular rashes appear after the fever goes down.

Rash resembles measles.

Red papules in oral cavity, usually over the palate called Nagayama spots.

Roseola infantum



HHV-7 :

Produce pityriasis rosea (pityriasis : Scaling, rosea : Rose like rash).

First lesion is called the herald patch.

Characteristic scaling called the collarette of scale.

Distributed along the lines of langer : Christmas tree pattern.

HHV-8 diseases :

- Kaposi sarcoma.
- Multicentric Castleman disease.
- Primary effusion lymphoma.

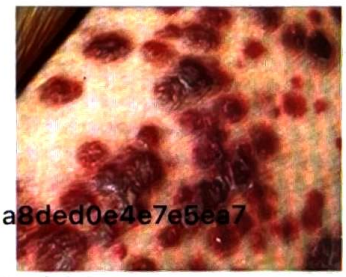
} Lymphoproliferative diseases

Kaposi Sarcoma :

Multifocal vascular tumor.

Patient presents with purplish nodules and plaques.

It is classified as an AIDS defining illness.



Kaposi Sarcoma

Molluscum contagiosum

00:35:05

Benign self limiting viral infection of epidermal keratinocytes.

Etiology :

Family : Pox virus.

Virus : Molluscum Contagiosum Virus (MCV).

The strains of MCV are as follows :

Virus	Age group	Sites (common)
MCV-1	Children	Face
MCV-2	Adults	Genitals

Transmission : Skin to skin contact.

Incubation period : 2 weeks to 6 months.

Disease patterns : (3 important patterns)

1. Children :

Pearly white, dome shaped umbilicated papule, generally over the face.

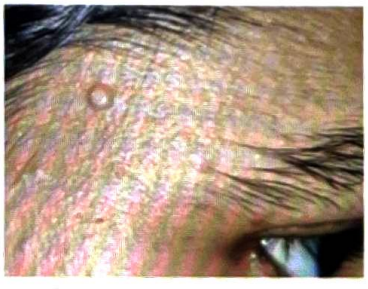
Pseudo Koebner's phenomenon, defined as the appearance of morphologically similar lesions along line of trauma (scratching) due to autoinoculation, It is also seen in viral warts.

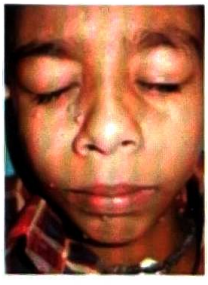
2. Sexually active adults :

Morphologically similar to the above mentioned pattern. However, the site of appearance is anogenital region.

3. Immunosuppressed individuals :

Such as in HIV infected individuals, MCV presents as giant molluscum > 1 cm as opposed to their normal size of 2-5 mm. The lesions are very extensive, atypical and recalcitrant (not responding to therapy).





Pattern in children

Pseudo Koebner's phenomenon

Pattern in sexually active adults

Complications :

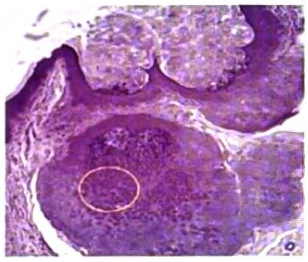
- Secondary infections due to scratching of lesions.
- In cases of eyelid molluscum, it may also cause conjunctivitis or superficial punctate keratitis.

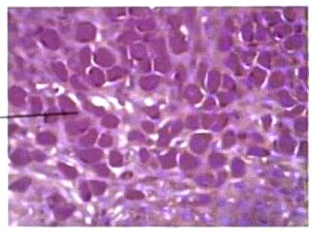
Investigations :

Generally, no investigations required.

In giant molluscum, HPE will show :

Eosinophilic intra cytoplasmic inclusion bodies/Henderson Paterson bodies within the cells.





Eosinophilic intra cytoplasmic bodies.

Treatment :

Usually, it is self-limiting and will disappear by itself. Thus, there is no need for treatment.

Physical methods :

- Curettage : MC must be completely removed, as its remnants may lead to growth of new lesions.
- Cryotherapy : Liquid nitrogen is used at a temperature of -196°C, where the lesion is frozen.

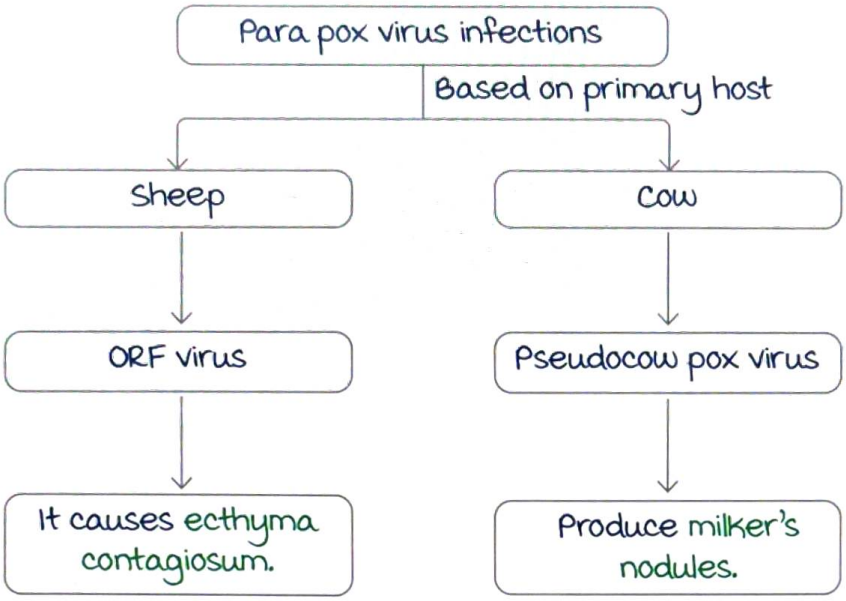
Topicals :

- Trichloroacetic acid.
- Potassium hydroxide (KOH).
- Imiquimod : Immune response modifier. It acts by activating Toll like receptor-7 (TLR-7 agonist).

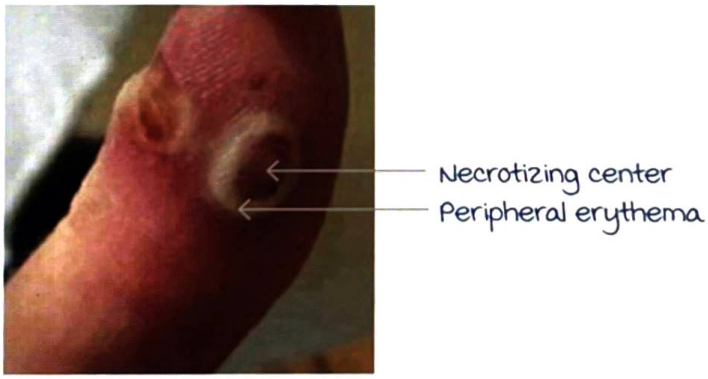
Parapox virus infections

00:44:54

Classification of para pox virus is as follows:

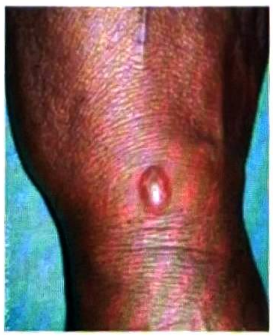


Ecthyma contagiosum presents like a target lesion with central necrosis and peripheral erythema.



Target lesion in ecthyma contagiosum

Milkers nodules present as nodular lesions over the body.

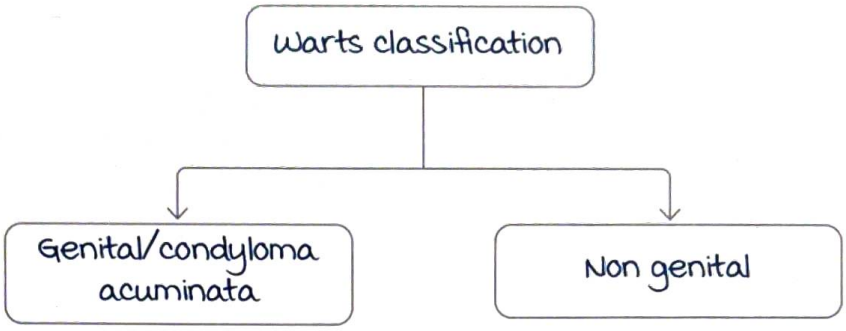


Milker's nodule

Active space

Human Papilloma Virus (HPV) infections 00:46:42

HPV induces benign proliferation of the skin called as warts.
HPV is a DNA virus.
It has affinity to squamous epithelium and produces projection/ papilloma or warts.



Non genital warts :

1. Verruca vulgaris :

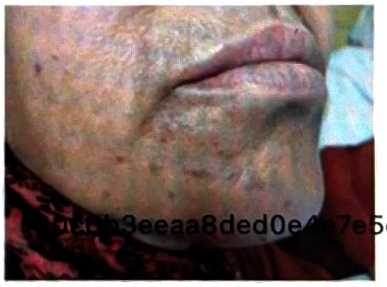
It is the most common non-genital warts.
(Verruca : Rough or uneven surface, vulgaris : most common).


Verruca vulgaris

Etiology :
Cutaneous warts : HPV 2, 4, 27.
For genital warts : HPV 6, 11.
Transmission : Skin to skin contact.

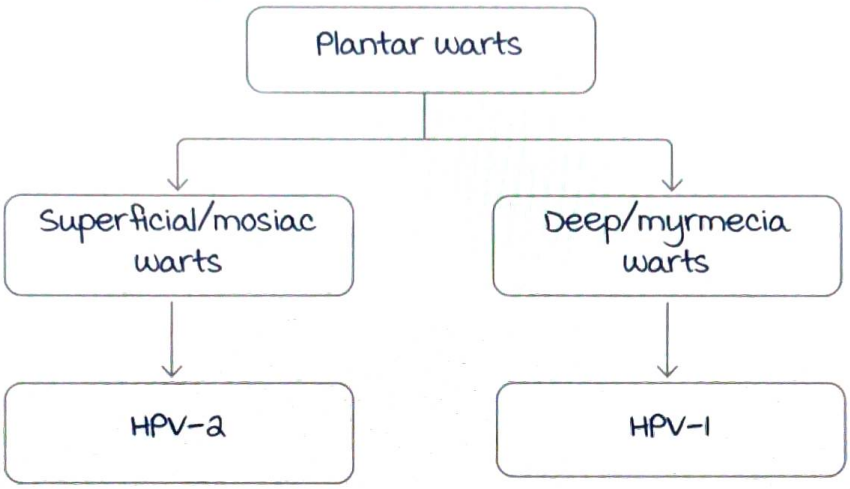
Lesions :
They are asymptomatic, skin colored papules and plaques with a rough or verrucous surface.
They spread by autoinoculation.

2. Planar warts :
Warts with flat surface.
Etiology : HPV 3, 10.


Planar warts

3. Plantar warts :

They are broadly classified as follows :



Differential diagnosis of plantar warts :

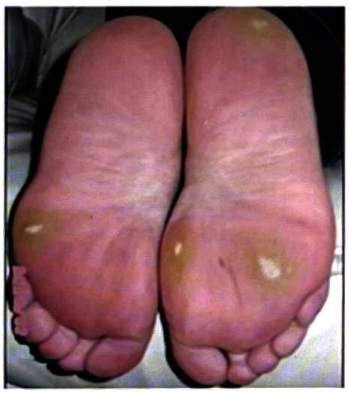
Corn : Can be clinically differentiated by applying pressure.

Plantar warts ⟶ Tenderness on applying lateral pressure.

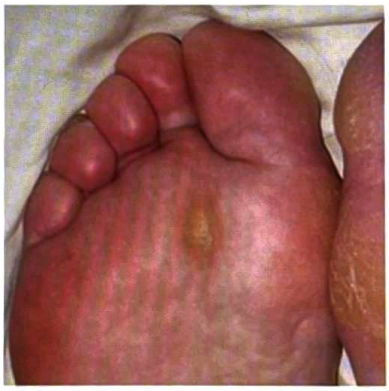
Corn ⟶ Tenderness on applying vertical pressure.

Corn is formed due to wearing ill-formed footwear.

Definitive assessment can be done using a scalpel blade to remove the upper part of stratum corneum (paring). In a wart, paring causes multiple bleeding points to occur.



Plantar warts



Corn

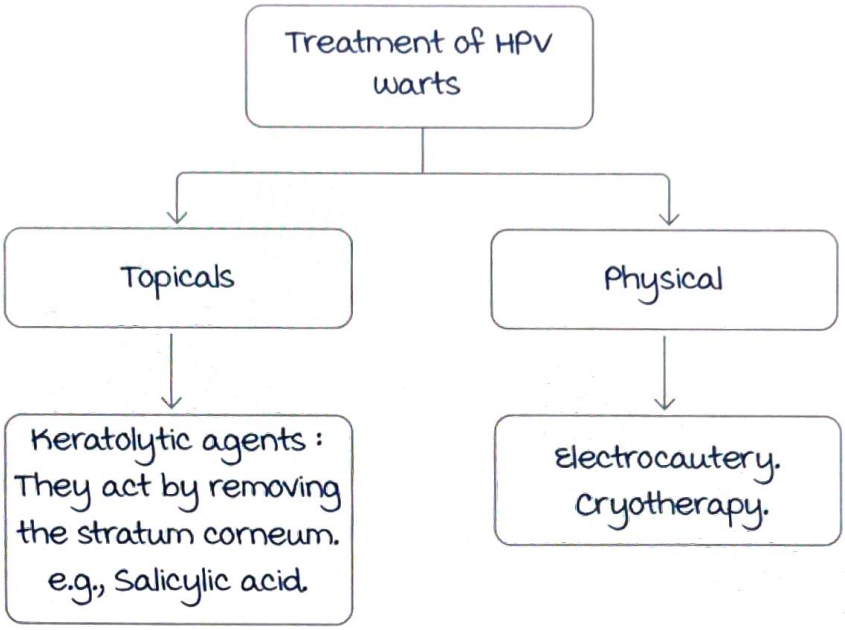
4. Filiform warts : Finger like projection.

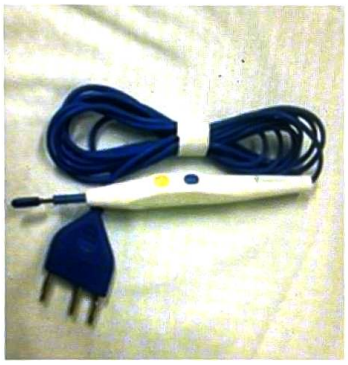
5. Periungual warts : Warts associated with the nail plate. This is difficult to treat.

Filiform warts

Periungual warts

Treatment of HPV warts :





Electrocautery

Cryogun

Epidermodysplasia verruciformis 00:54:09

It is an autosomal recessive inherited disorder.

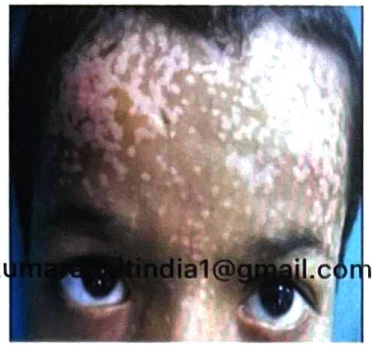
Etiology : HPV 5, 8.

Lesions :

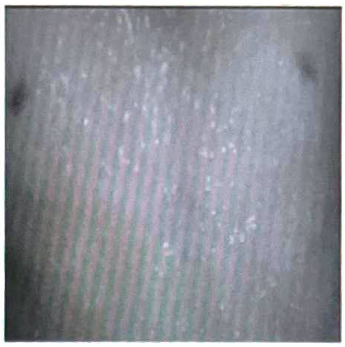
- Plane warts over face.
- Pityriasis versicolor like lesions over the trunk.

Complication :

Squamous cell carcinoma is one of the most common complications of this condition.



Epidermodysplasia verruciformis – facial warts

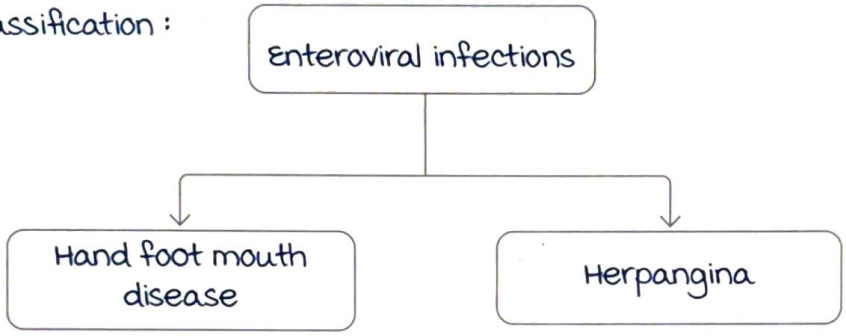


Epidermodysplasia verruciformis– truncal lesions

Enteroviral infections

00:55:51

Classification :

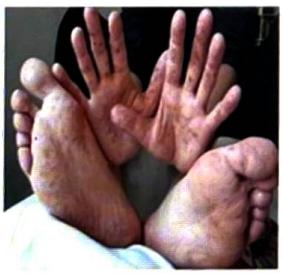


Hand foot mouth disease :

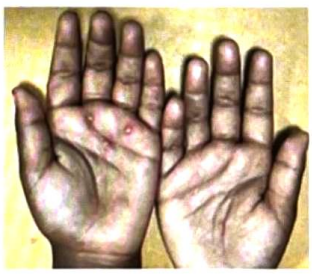
Etiology : Coxsackie A16, Enterovirus 71.

Lesions : Papulovesicular lesions.

Sites : Present on palms, soles, oral cavity and the buttocks.



Hand foot mouth disease



Hand foot mouth disease

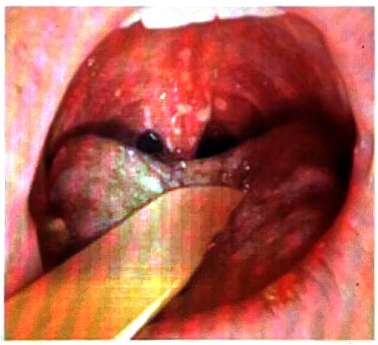
Herpangina :

Etiology : Coxsackie A1 – A6, A8, A10 and Enterovirus 71.

Symptoms : High grade fever.

Lesions : Painful papulo-vesiculo-ulcerative enanthem (rash within the oral cavity).

Sites : Present on posterior oropharynx.

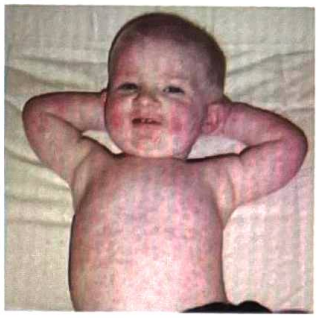


Herpangina

Active space

Parvovirus B19 infections

00:58:53



Parvovirus B19 is a DNA virus.
It causes 5th disease or erythema infectiosum.

Skin lesions :
The rash presents over the face as a classic malar erythematous rash with relative sparing of the mouth or circumoral sparing. There is a typical slapped cheek appearance.
Unlike malar rash of SLE, the bridge of the nose is not involved here.
It first presents as a maculopapular rash following which it becomes a reticulate lacy exanthema characterised by net like rash on the trunk.

Active space

60c6b3eeaa8ded0e4e7e5ea7

FUNGAL INFECTIONS

Superficial mycoses 00:00:14

It is defined as fungal infection which involve the superficial skin layers i.e. non-living tissues.

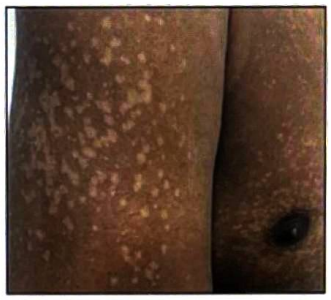
Pityriasis versicolor :

60c6b3eeaa8ded0e4e7e5ea7

Etiology :

Malassezia globosa > M. furfur.

It targets the hair follicle and produces perifollicular scaly macules (characteristic lesion in pityriasis versicolor).



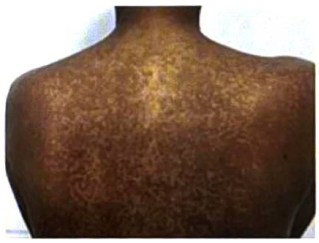
Pityriasis versicolor scales

Nomenclature :

Pityriasis means scaling i.e fine/branny/furfuraceous scales.

Versicolor means variety of colors.

Both hypopigmented (more common) as well as hyperpigmented macules are seen.

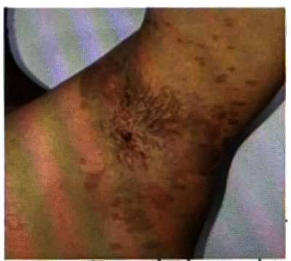


Hypopigmented macules

Hypopigmented macules are seen due to production of azelaic acid by the fungus.

Azelaic acid inhibits Tyrosinase enzyme resulting in hypopigmentation.

Hyperpigmented lesions are due to induction of large melanosomes.



Hyperpigmented macules

Site :

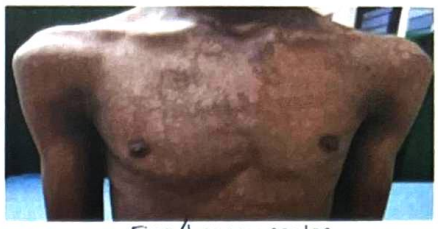
Trunk is the most common site.

Scaling is not visible → scratch with nail results → Scales become prominent.

Active space

This is known as Besnier sign/ Coup de ongle sign.

60c6b3eeaa8ded0e4e7e5ea7



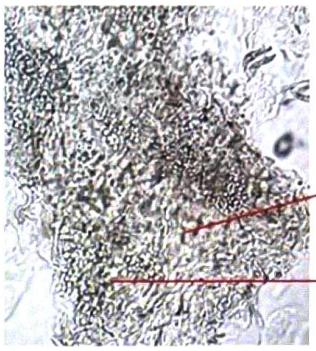
Fine/branny scales

Investigations :

10% KOH mount is required to dissolve the keratin and help visualize the fungal filaments better.

KOH mount in Pityriasis versicolor :

short hyphae + round spores, giving a spaghetti & meatball appearance.



Spaghetti & meatball appearance

Wood's lamp examination done at wavelength of 360–364nm shows yellow florescence.

Treatment of pityriasis versicolor :

- Azoles like Ketoconazole, Clotrimazole are the mainstay.
- Selenium sulphide available as a shampoo has both antifungal and cytostatic properties.

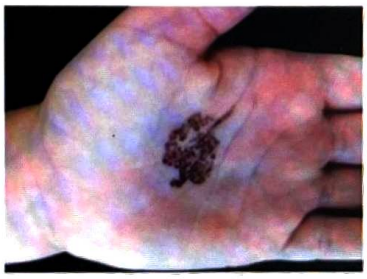
Oral Terbinafine and Griseofulvin are two drugs that do not work in Pityriasis versicolor.

Tinea nigra

00:08:03

Etiology :

Produced by Hortaea werneckii.



Tinea nigra

Clinical features :

Asymptomatic hyperpigmented macules over the palms.

Treatment :

Topical azoles.

Active space

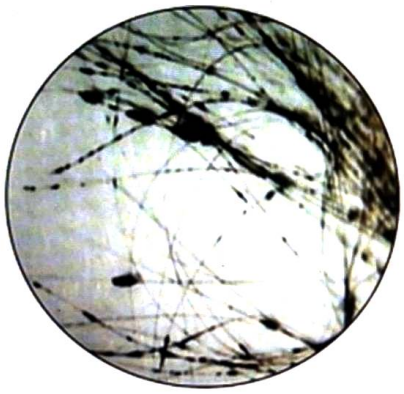
Piedra 00:08:56

Also known as trichomycoses nodularis.

It is defined as superficial fungal infection of hair shaft.

It is classified into black piedra and white piedra.

Feature	Black Piedra	White Piedra
Etiology	Piedraia hortae	Trichosporon beigelii
Nodules : 1) Color	Black	White
2) Consistency	Hard	Soft
3) Attachment to hair shaft	Tightly attached	Loosely attached



Black piedra

White piedra

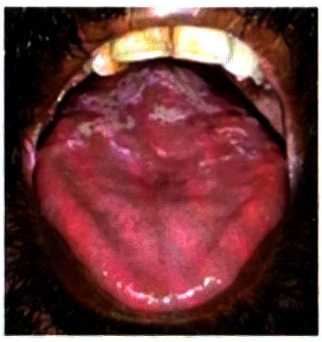
Treatment :

- Clipping of the hair.
- Topical antifungals.

Candidiasis 00:11:18

Etiology : Candida albicans (most important organism).

1. Mucocutaneous candidiasis :
   - Oral candidiasis (white plaques), there are two types of candidiasis :
     a. Pseudomembranous candidiasis (oral thrush) : Lesions can be rubbed off using a spatula.
     b. Chronic hyperplastic type.



Oral thrush

- Red plaques/atrophic candidiasis

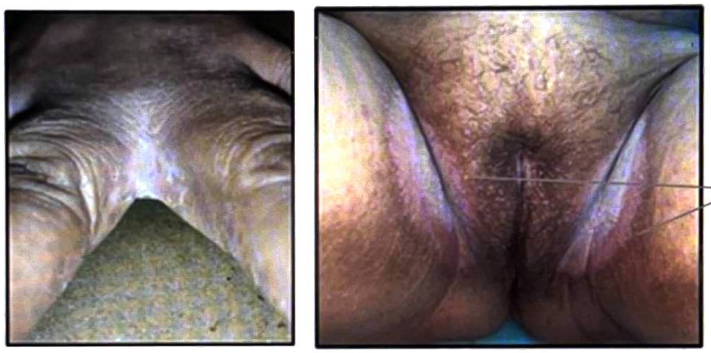
Atrophic candidiasis maybe acute or chronic.

Acute	Chronic
Occurs after taking antibiotics hence known as antibiotic sore tongue or sore mouth (burning sensation)	Denture mouth.

2. Candidial intertrigo :

Candidiasis that involves the Intertriginous areas. Skin fold facilitates more sweating, more friction and more candidial proliferation.

Lesions are red, macerated (soft and wet to touch) plaques and in the vicinity of the plaques there are satellite pustules.



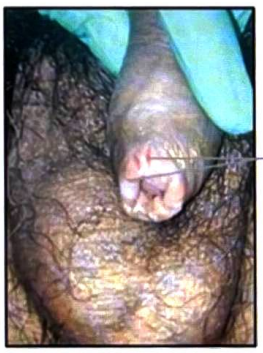
Candidial intertrigo

3. Candidial balanoposthitis :

Inflammation of glans penis (balano) & prepuce (posthitis).

Clinical features :

- Swelling of glans penis with burning sensation.
- Prepuce shows multiple radial fissures.



Candidial balanoposthitis

Associated with diabetes mellitus.

Candidial paronychia :

Characterized by swelling of nail fold and loss of cuticle.

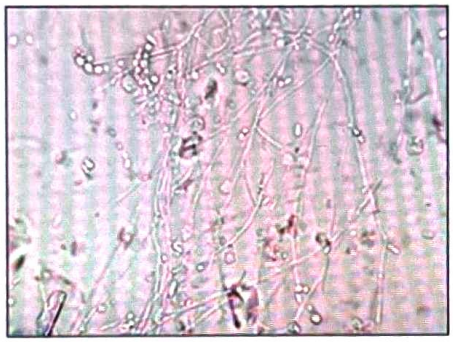
Investigations :

In KOH mount budding yeast cells along with elongated hyphae called the pseudo hyphae are seen.

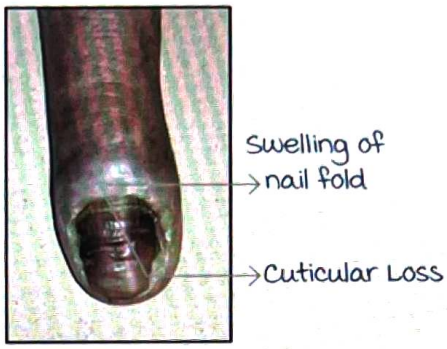
In sabouraud dextrose agar, candida are seen as creamy

white colonies.

Treatment of candidiasis : Fluconazole.



KOH mount : Budding yeast cells and pseudo hyphae



Candidial paronychia

Dermatophytosis

00:18:04

It is defined as the superficial fungal infection of the keratinized tissue produced by dermatophytes.

Dermatophytes are keratinophilic fungi.

Hence they affect the skin, hair and nail.

Genera : 3 important genera as follows.

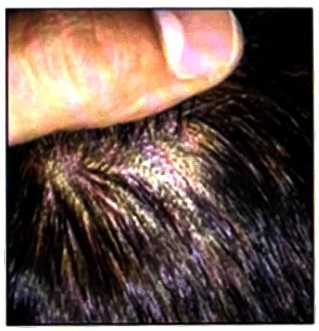
Microsporum	Epidermophyton	Trichophyton
Skin and hair	Skin and nail	Involves all 3 : Skin, hair and nails

Pathogenesis :

Commonly referred to as tinea infections.

3 steps of pathogenesis are as follows :

- Adhesion to superficial skin tissues.
- Invasion and growth.
- Immune response (determines kind of lesion that may develop).



Tinea capitis

Dermatophytosis region wise :

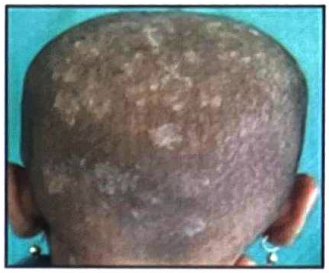
Tinea capitis : Scalp hair involvement.

- Black dot type : Non inflammatory tinea capitis. The fungus goes into the hair, referred to as endothrix & hair shaft breaks

Active space

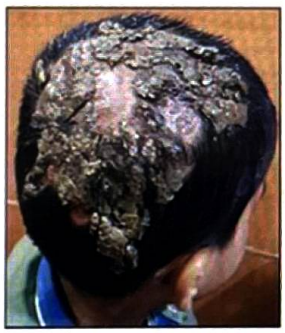
Two important species involved are : Trichophyton tonsurans, T. violaceum.

- Grey patch : Non inflammatory tinea capitis. The fungus is covering the hair (Ectothrix). Lustreless grey hair with patchy alopecia.
  Organism causing this is microspora canis.



Grey patch

- Favus : Inflammatory type produced by Trichophyton schoenleinii.
  Endemic to Kashmir. Lesions are yellow cup shaped crust called scutula adherent to the hair follicles.



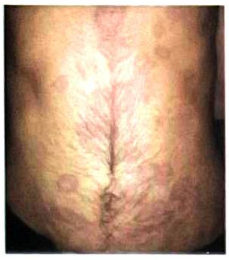
Favus

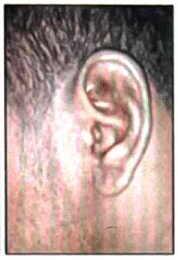
- Kerion : Inflammatory type produced by zoophilic fungi.
  H/O contact with pet animals must be always be elicited.
  Two important species :
  T. mentagrophytes, T. verrucosum.
  Usually seen in children. Presents with boggy scalp swelling, easily pluckable hair. Human body will react profusely causing intense inflammation and regional lymphadenopathy as it a zoophilic fungi and humans are accidental hosts.

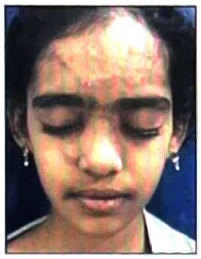
Kerion

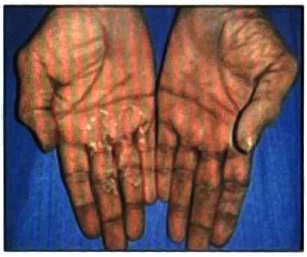
- Tinea faciei : Face.
- Tinea barbae : Beard.
- Tinea manuum : Involves palmar aspect of the hands.
- Tinea corporis : Involves glabrous/non-hairy (thick hair) skin except palms, groin and soles.
- Tinea cruris : Involves groin/Jock itch/dhobi itch.
  Characterized by annular plaque with raised border seen in the groin.

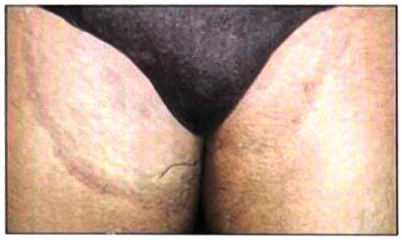
- Tinea pedis : Also referred to as Athlete's foot as it involves the feet. Most common site is 4th toe webspace.

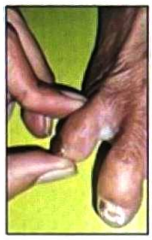

Tinea corporis


Tinea faciei


Tinea faciei


Tinea manuum


Tinea cruris

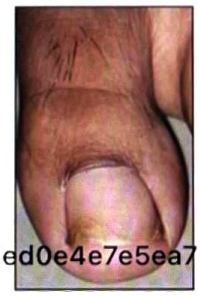

Tinea pedis

Onychomycoses

00:27:33

Fungal infection of the nail unit.
It can be caused by dermatophytes, non-dermatophytes or candida.
If it is caused by dermatophytes, then it is called as Tinea unguium.
Can be identified by yellow discoloration of the nail plate and presence of subungual hyperkeratosis.
And many times, there may be the presence of Subungual hyperkeratotic tunneling.


Onychomycoses


DLSO

Differential diagnosis :
Nail psoriasis presents with yellowing of nails & subungual hyperkeratosis.
Psoriatic nail classically has pits which are absent in Onychomycosis.

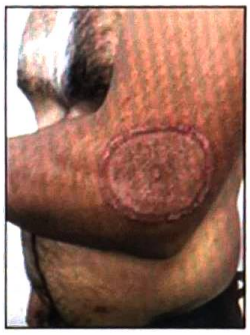
Active space

Distal Lateral Subungual Onychomycoses (DLSO) is the most common type.

Morphology of Tinea corporis/cruris 00:29:48

Etiology :
T.mentagrophytes, also has anthropophilic strains.
T.rubrum.

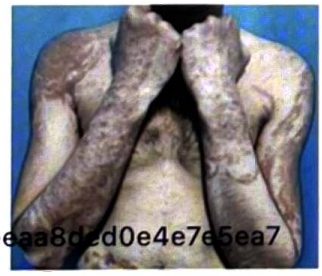
Lesion :
Annular plaque with central clearing and peripheral raised scaly margins.
Associated with itching.



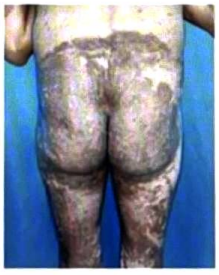
T.corporis

T.incognito 00:31:27

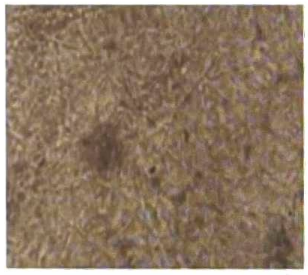
Incognito means unrecognizable.
Steroid modified Tinea.
Anti-inflammatory property of steroids decreases the itching but the active margin of lesion is lost and striae is formed.
Leads to extensive & atypical disease with diffuse scaling.



Tinea incognito



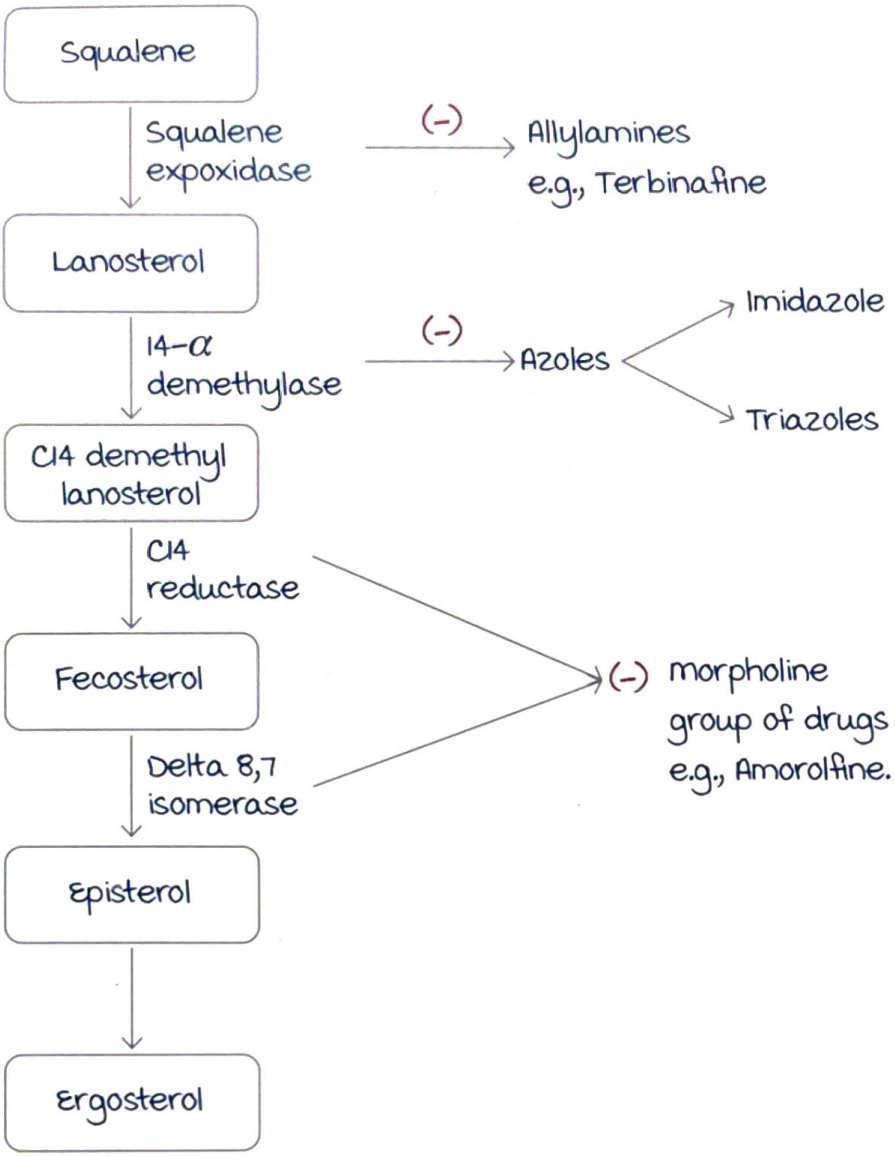
Diffuse lesions seen



Refractile branching hyphae as seen in 10% KOH.

Investigations of dermatophytosis :
10% KOH mount : Presence of refractile branching hyphae s/o dermatophyte hyphae.

Treatment of dermatophytosis :
Basic structure of fungal cell wall and drugs acting at each step are as follows :



Treatment of dermatophytosis (topical) :

1. Azoles : They inhibit 14-α demethylase.
   Imidazoles : Ketoconazole, Luliconazole and Sertaconazole.
2. Allylamines like Terbinafine.

Treatment of dermatophytosis (systemic) :

1. Itraconazole 100mg BD.
2. Terbinafine 250mg OD

Duration : 4-6 weeks.

Treatment of tinea capitis :

Overall drug of choice : Griseofulvin.

T.capitis secondary to Trichophyton species : Terbinafine.

Active space

Treatment of onychomycosis :

Special formulations called nail lacquers :

- Amorolfine 5%.
- Ciclopirox-olamine 8%.

Systemic treatment of dermatophytic onychomycosis :

Terbinafine : 250mg OD.
Finger nail : 6 weeks.
Toe nails : 12 weeks.

Itraconazole : 200mg BD/week, this is 1 pulse and patient should be treated with 1 pulse/month = pulse therapy
Fingernail : 2 pulses.
Toe nails : 3 pulses.

Subcutaneous mycoses

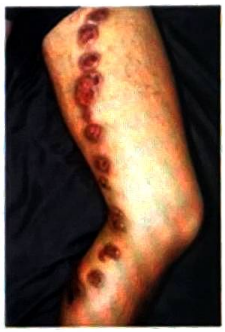
00:39:28

Also referred to as implantation mycoses as it is brought about by trauma induced inoculation.
There are 3 important subcutaneous mycoses :

1. Sporotrichosis.
2. Chromoblastomycosis.
3. Mycetoma.

Sporotrichosis :

Etiology :
Sporothrix schenckii.
Dimorphic fungus i.e. mold at 25°C and yeast at 37°C.
Also referred to as Rose gardener's disease.
Target : Extremity lymphatics.
Frequent H/O thorn prick (organism gets inoculated into the skin).
most common type : Lymphocutaneous.
Nodulo-ulcerative lesion seen at site of inoculation → Fungus shows 'proximal extension' i.e. lesion spreads upward along the lymphatics → Forms linear pattern



Sporotrichosis

along the lymphatics of the extremity.
Proximal extension along the lymphatics of the extremity is characteristic of sporotrichosis.

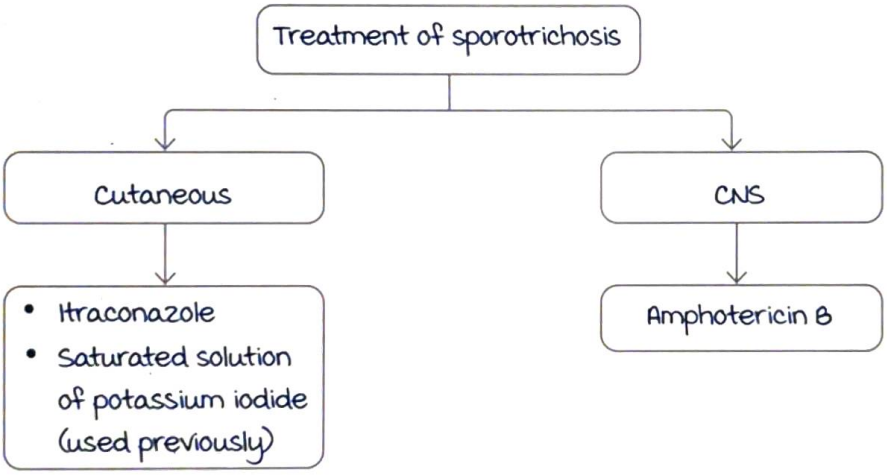
Differential diagnosis of sporotrichoid lesions :

1. Nocardiosis.
2. Atypical mycobacterium : Swimming pool granuloma, produced by mycobacterium marinum.
3. Cutaneous leishmaniasis.
4. Tularemia.

HPE :

Asteroid body with central basophilic yeast with radiating eosinophilic material is seen.

Treatment :



Chromoblastomycosis :

Chromo means Color.

Named so, as the organism producing chromoblastomycosis is pigmented/dematiaceous fungi.
(meaning : fungus which has got color in its structure).
It is also known as verrucous dermatitis, as the surface of the lesion is rough.

Etiology :

- Fonsecaea pedrosoi.
- Fonsecaea compactum.
- Phialophora verrucosum.

Active space

Occupation :

Agricultural workers with history of trauma from vegetative material.

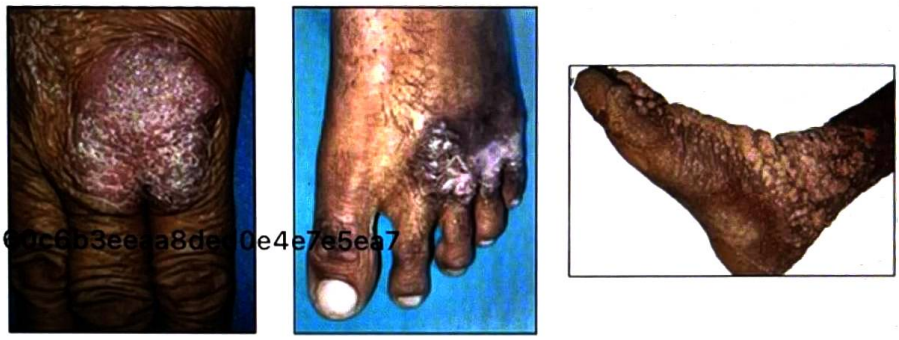
Skin lesions :

Morphologically, presents with :

- A slow growing verrucous plaque.
- Cauliflower like nodules/masses.
- Black dots on the surface of the lesion.

Site :

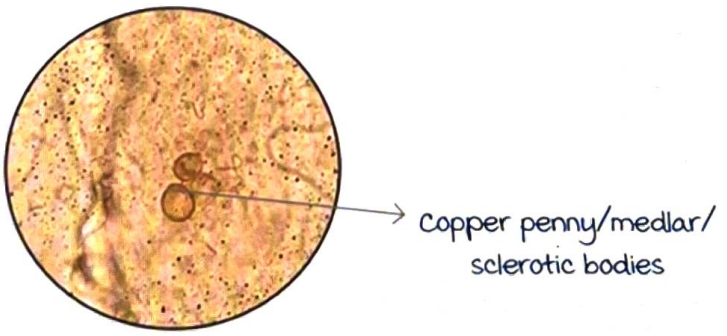
Usually on the extremities; trauma prone sites.



Chromoblastomycosis

Investigations :

10% KOH mount : Colour is visualized by brown, round, thick walled bodies called as Copper penny/medlar/sclerotic bodies , diagnostic of chromoblastomycosis.



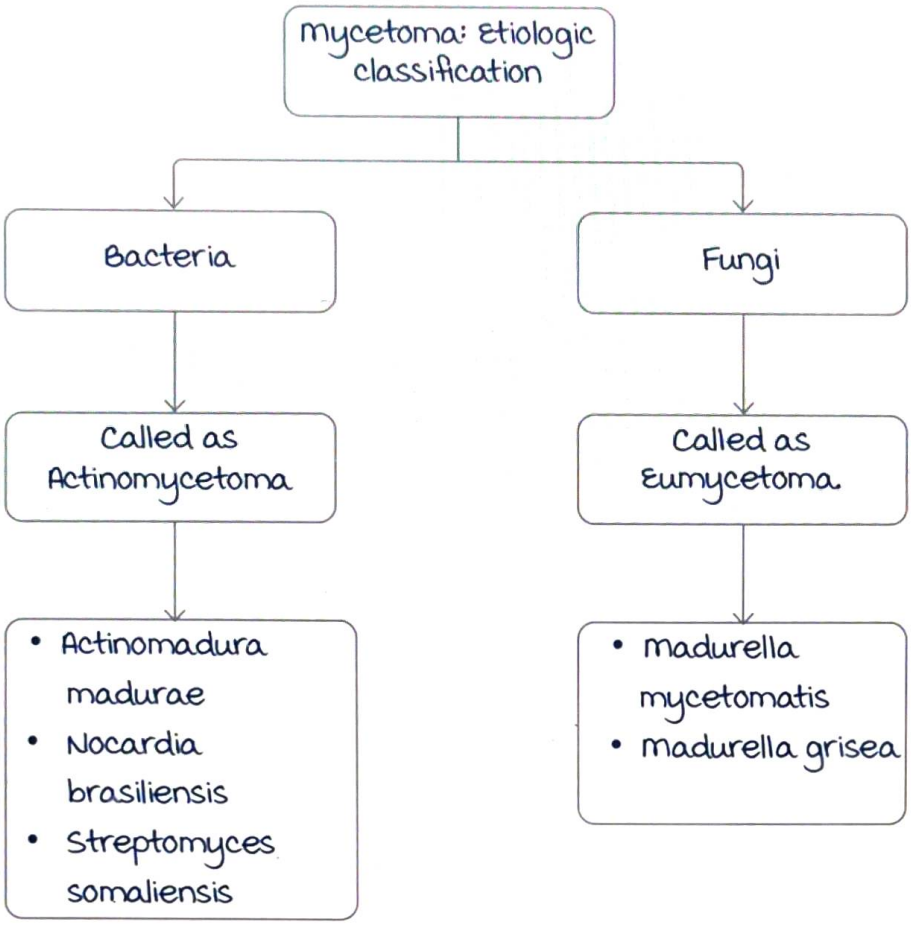
KOH mount of Chromoblastomycosis.

Treatment : Itraconazole is the drug of choice.

Mycetoma :

Chronic suppurative granulomatous disease which could be either fungal or bacterial in origin.

Etiologic classification :



Clinical features :

Sex predilection : males > females.

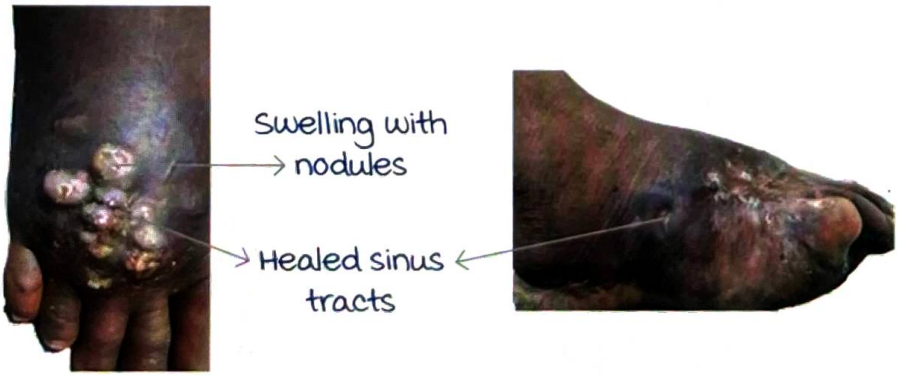
Patients present with history of trivial penetrating trauma.

Site :

Usually seen over the extremities.

Triad of mycetoma :

1. Tumefaction (tumor like swelling).
2. Multiple draining sinus tracts.
3. Discharging granules that represent colonies of the microbe.

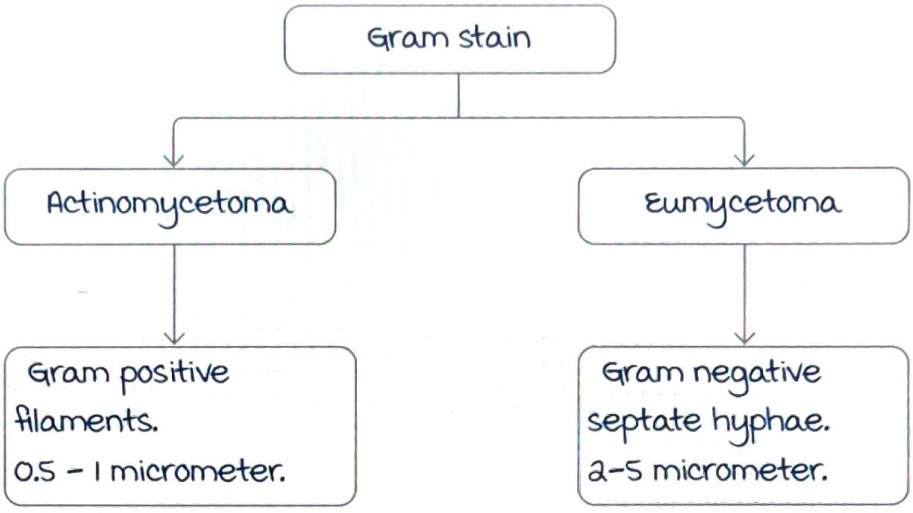


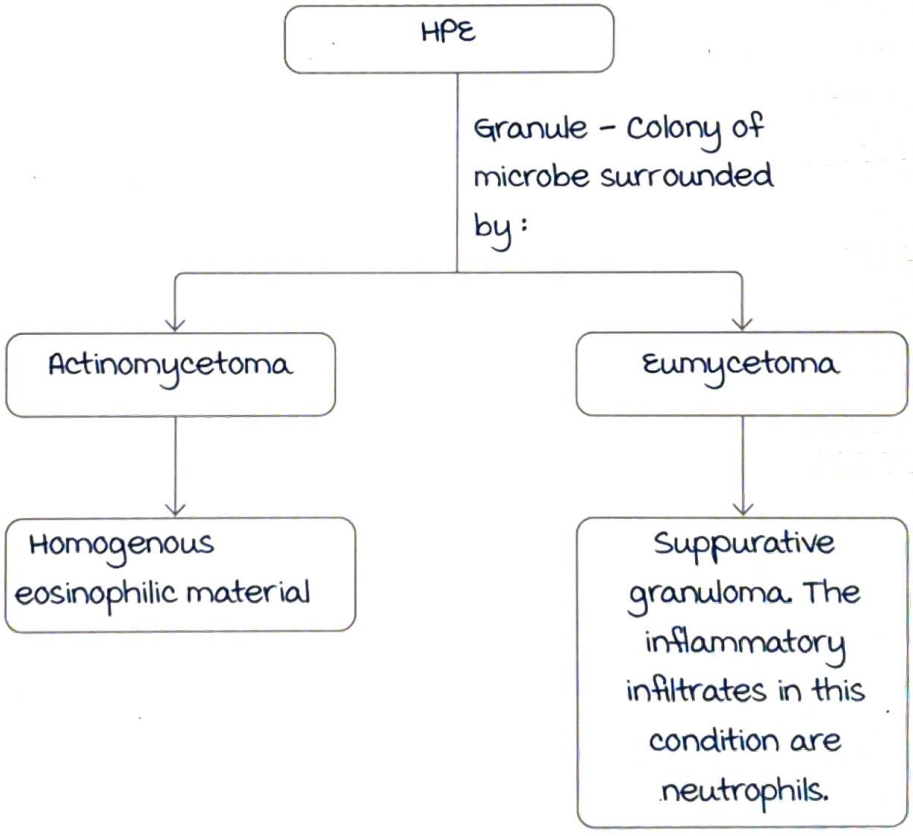
mycetoma

mycetoma

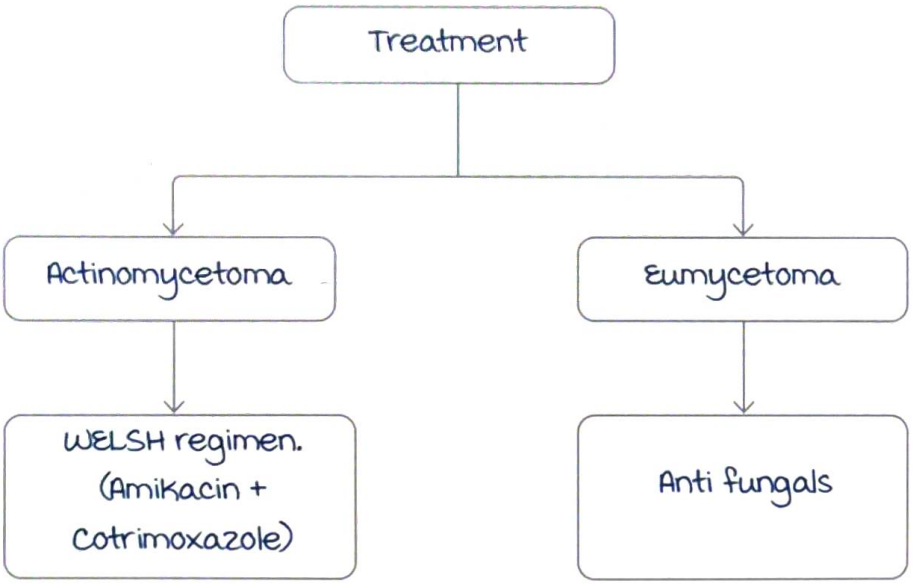
Active space

Investigations :

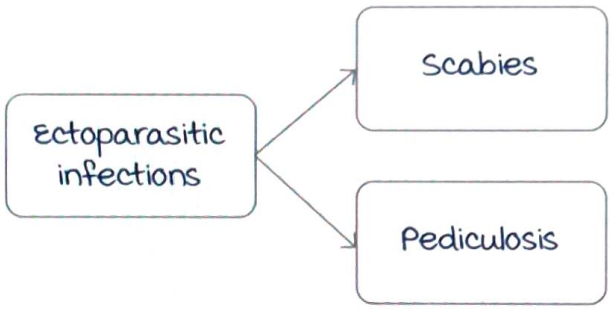




Treatment of mycetoma :



PARASITIC SKIN INFECTIONS



Scabies

00:00:32

Itchy contagious ectoparasitic infestation.

Etiology :

Itch mite → Sarcoptes scabiei var hominis.

Number of mites in a patient : 10-12 mites per patient, in a person with normal immunity.

Transmission :

Close personal contact : Skin to skin contact.

Also transmitted via fomites like clothes, bedding.

60c6b3eeaa8ded0e4e7e5ea7

Classified as a water washed disease.

- Seen with decreased personal hygiene.

Incubation period :

1st time : 4 weeks.

Re-infestation : 1-2 days.

Clinical features :

History of nocturnal pruritus.

- Mite becomes active at night.
- Pruritus happens due to delayed hypersensitivity response to the mite.

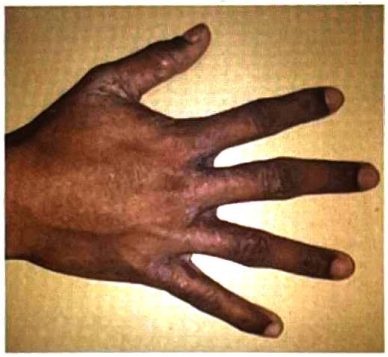
History of similar complaints among the family members/ close contacts are to be assessed.

Examination findings :

Excoriated papules seen predominantly over the web spaces.

On the external genitalia : Burrows are seen.

- Specific to scabies.
- Burrows are wavy greyish white tunnels in the stratum corneum.
- Absent in animal scabies.



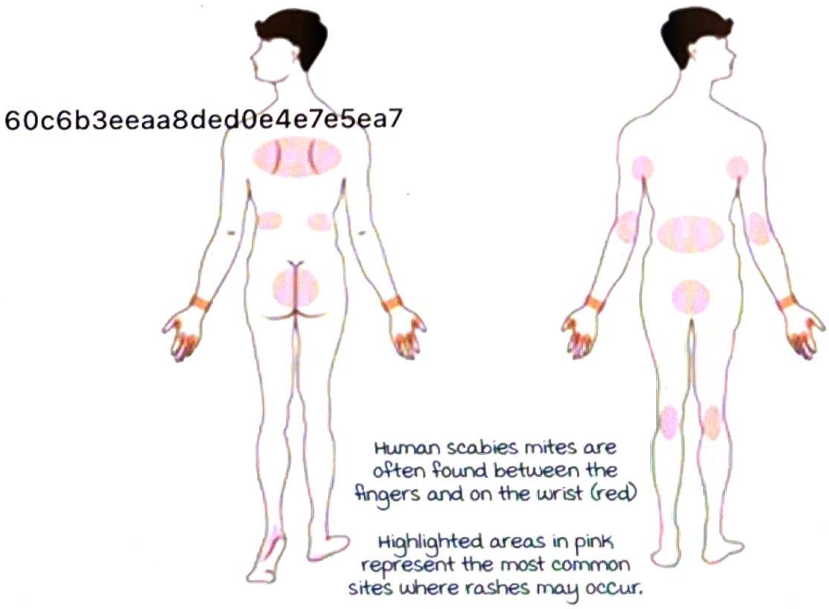
Excoriated papules

Scrotal burrows

Distribution of scabies : Circle of Hebra.

1. Web spaces of the finger.
2. Medial aspect of the wrist.
3. Medial aspect of the forearm and arm.
4. Axilla.
5. Nipple.
6. Umbilicus.
7. Genitalia and groin.

Sites of infection of Scabies



Spared in adults : Face (due to increased sebum activity that repels mites).

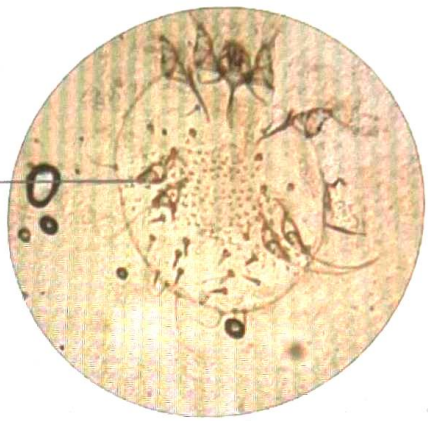
Active space

Investigations :

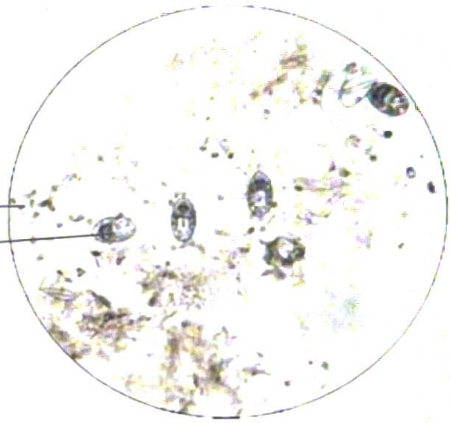
KOH mount of skin scrapping.

The mite is identified by :

- Two anterior pairs of legs.
- Two posterior pairs of legs.



Scabies mite with two anterior and two posterior pairs of legs



Fecal pellets : Scybala

Eggs of the scabies mite

Treatment :

General management :

- Treatment of pruritus : Antihistamines.
- Clothes should be washed.
- Treat family members/close contacts.

Specific management :

Topical drugs :

- 5% Permethrin : Best drug.
  Inhibits the sodium channels in the parasite → Induces paralysis.
  Below neck application, kept for 12 hours.
  Use the entire tube in one application.
  Repeated after one week.
  To be applied everywhere, not on lesions alone.
- Benzyl benzoate emulsion.
- Gamma benzene hexachloride.
- Crotamiton : Has anti pruritic properties.
- Precipitated sulfur → Safe in pregnancy.

Systemic drugs :

Ivermectin :

Oral dose 200µg/kg.

MOA : Binds to the glutamate gated chloride channels → Induces chloride influx → Hyperpolarization → mite paralysis.

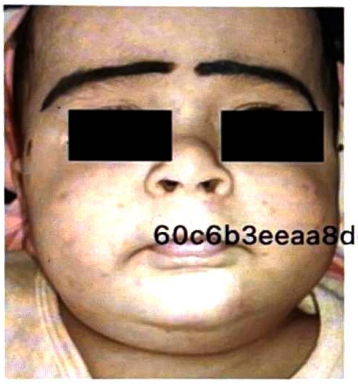
Special forms of scabies

00:13:10

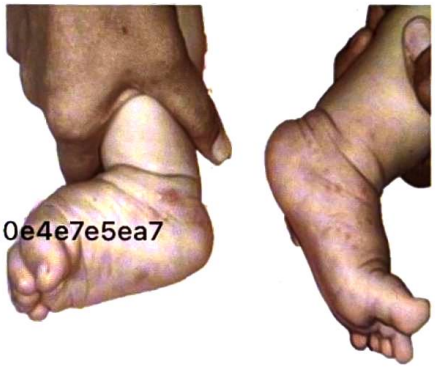
1. Infantile scabies.
2. Norwegian scabies.
3. Nodular scabies.

Infantile scabies :

- Face is involved as sebaceous glands are not active in an infant.
- Vesicles in palms and soles.



Lesions on the face



Vesicles seen on the soles

Norweignan scabies :

Also known as crusted scabies.

most severe form.

Seen among immunosuppressed individuals (HIV, lepromatous leprosy, malignancy).

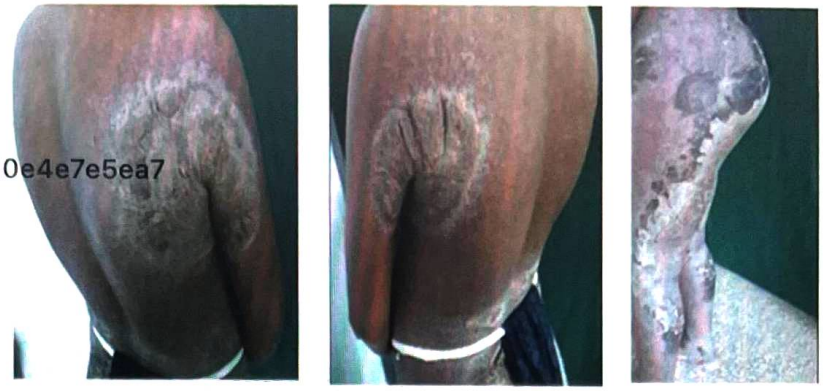
Pruritus is minimal as the immunity is low.

Millions of mites may be seen on the patient.

Skin lesions :

- Hyperkeratotic plaques.
- Erythroderma (> 90% of body surface has erythema ± scaling).

Active space



Norwegian scabies

Treatment :

- Ivermectin.
- 5% permethrin.
- Keratolytic agent to remove the crust : salicylic acid.
  Helps in better drug penetration of permethrin.

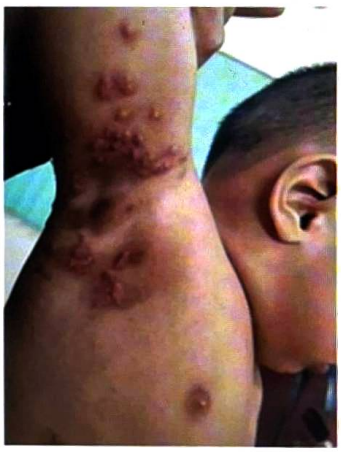
Nodular scabies :

Hypersensitivity response to the scabies mite.

Presents with pruritic nodules.

Sites : Axilla and scrotum.

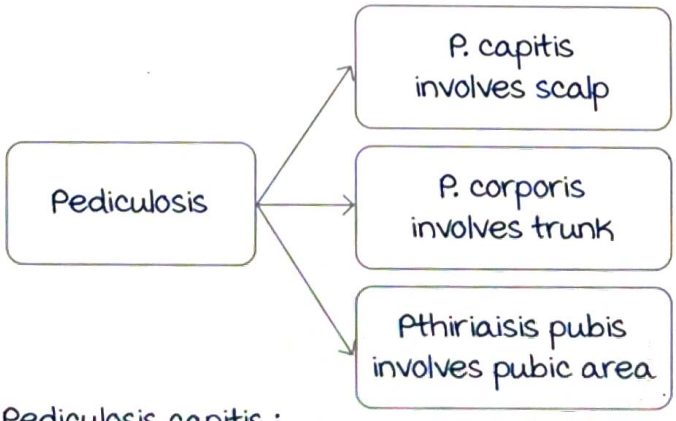
Treatment : Anti-scabetic treatment + intralesional steroid to reduce the hypersensitivity response (only type of scabies where steroids are given).



Nodular scabies

Pediculosis

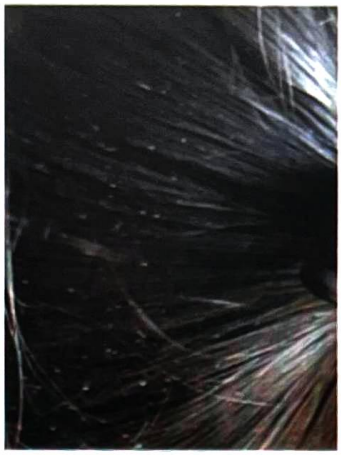
00:19:11



Pediculosis capitis :

Caused by head louse (Pediculus humanus capitis).

Transmitted by close personal contact.
Nits : Eggs of the louse.
Presentation :
more common in girls.
Presents scalp pruritus.


Nits on hair

O/E : Nits and louse seen.
May also present with enlarged regional lymph nodes.

Pediculosis corporis :
Caused by body louse/clothing louse.
Also called vagabond's disease.
Seen among wanderers, in those with decreased personal hygiene.
Symptom : Pruritus.
Clothing louse : Seen in the seams of the clothing.
The louse comes to the human skin to take a blood meal and then goes back to clothing.
Excoriations seen are called morbus errorum.

Pediculosis pubis :
Also called pthiriaisis pubis.
Caused by pubic louse or crab louse.
Transmission by sexual contact.
2nd and 3rd pair of legs end as pincer like claws.


Nit (egg)


Adult pubic louse

Symptom : Pruritus.
Lesion : Macula cerulea → blue coloured macule at the lower abdomen.
Anticoagulant property in louse saliva.

Treatment :
Pediculosis capitis and pediculosis pubis : 1% Permethrin.
Pediculosis corporis : Disinfection of clothes.

Active space

Migratory infective skin lesions

00:26:11

1. Cutaneous larva migrans.
2. Larva currens.
3. Calabar swellings.

Cutaneous larva migrans :

Skin penetration by larva of animal hookworm.

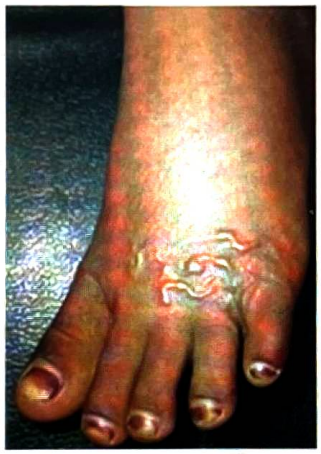
- Ancylostoma braziliensis.
- Ancylostoma caninum.

History of walking barefoot (beach) → Soil containing active forms of the larva in animal excreta → Enters skin.

Lesion : Pruritic, elevated erythematous serpiginous tract.

Speed : 1 – 2cm/day.

Treatment : Albendazole, Ivermectin.



Cutaneous larva migrans

Larva currens :

60c6b3eeaa8ded0e4e7e5ea7

Produced by Strongyloides stercoralis (racing larva).

Speed : 5–15cm/hour.

Lesions : Urticaria like lesions.

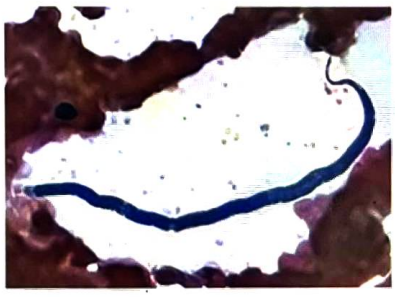
DOC : Ivermectin.

Calabar swellings :

Caused by Loa loa (African eye worm).

Lesions : Painless, non pruritic transient swellings on face.

DOC : Diethyl carbamazine (DEC).



Loa loa

Trypanosomiasis

00:30:56

Two types :

- African trypanosomiasis (sleeping sickness).
- American trypanosomiasis (Chaga's disease).

African trypanosomiasis :

Caused by Trypanosoma brucei.

Vector is tse tse fly.

- West African :

  Two stages : Early and late.

  Early : Shows winter bottom sign → Posterior cervical lymphadenopathy.
- East African.

American trypanosomiasis :

Caused by Trypanosoma cruzi.

Vector is reduviid bug.

- Acute stage.

  Romana sign : Sensitization response to bug bite.

  Unilateral painless, palpebral edema.
- Chronic stage.

Romana sign

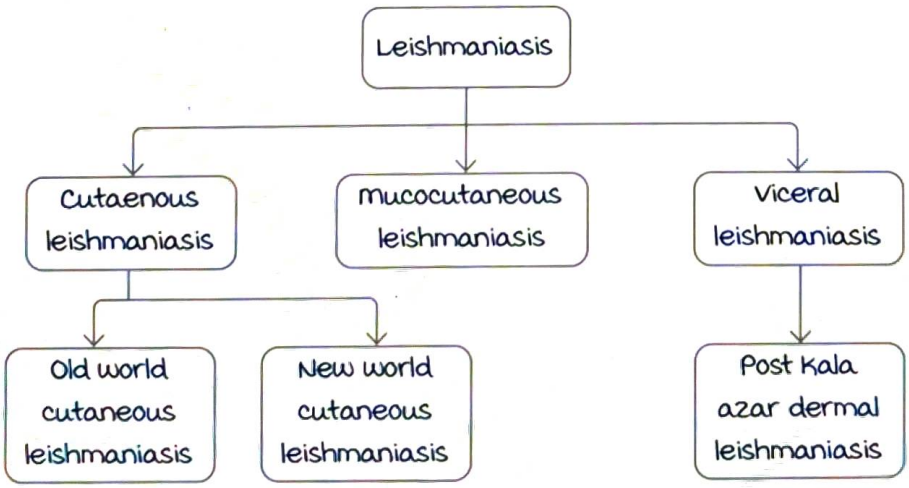
Leishmaniasis

00:34:05

Transmitted by sandfly.

Classified into 4 types.

60c6b3eeaa8ded0e4e7e5ea7



Old world cutaneous leishmaniasis :

Also called Delhi boil/oriental sore.

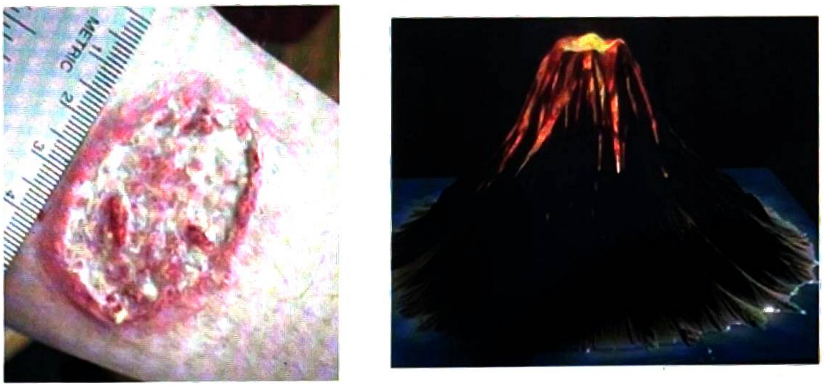
Caused by L. major and L. tropica.

Nodulo ulcerative lesion with crusting in the center.

Ulcer margins are raised : Volcano sign.

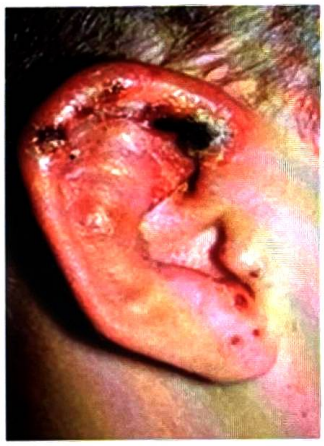
Active space

Nodulo-ulcerative lesion with crusting in the centre



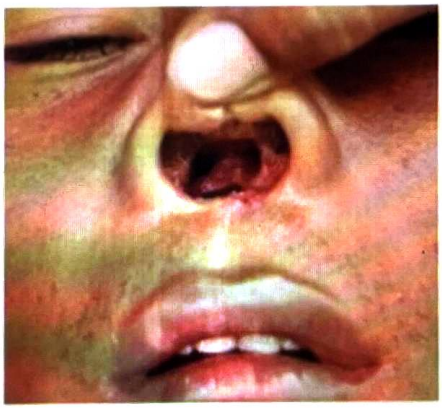
Volcano sign in the ulcer

New world cutaneous leishmaniasis :
Also called gum tree harvesters ulcer/ Chiclero ulcer.
Caused by L. mexicana.
Lesion : Ulcer affecting the cartilage of the ear.



Cutaneous leishmaniasis (New world)

Mucocutaneous leishmaniasis :
Caused by L. braziliensis.
Invasion and destruction of nasopharynx.
Lesions : Mutilating lesions of face, nose → Espundia.



Espundia

Visceral leishmaniasis :

Also called Kala-azar.

Caused by L. donovani.

Presents with fever and hepatosplenomegaly (massive splenomegaly).

Post Kala-azar Dermal Leishmaniasis (PKDL) :

Cutaneous sequalae secondary to Kala azar.

Seen when Kala azar is partially or inadequately treated.

Common in Bihar.

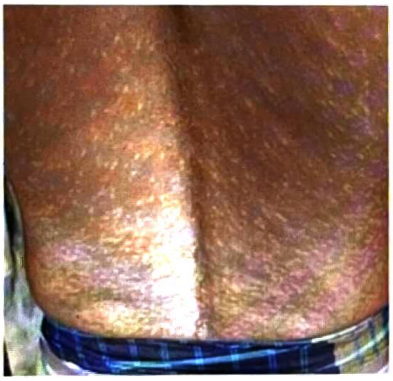
History of prolonged fever may indicate history of visceral leishmaniasis.

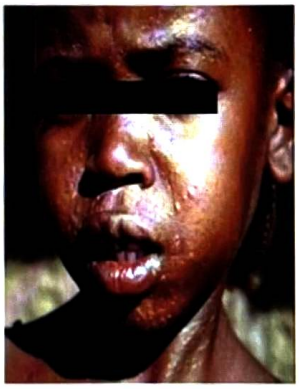
Lesions :

- Trunk : Hypopigmented macules.
- Face : Infiltrated nodules.

Differential diagnosis → Lepromatous leprosy :

- Nerve thickening.
- Slit skin smear +ve for acid fast bacilli.





Lab diagnosis :

Leishman Donovan (LD) bodies in Giemsa stain.

Can also be detected in HPE.

Culture media for leishmania : NNN media (Novy MacNeal Nicolle).

Treatment :

For Kala-azar : Liposomal amphotericin B.

For PKDL : Miltefosine.

Active space

HANSEN'S DISEASE : PART - 1

Leprosy/ Hansen's disease :
Leprosy is a chronic infectious granulomatous disease predominantly affecting the skin and nerves.

Etiology :
- Mycobacterium leprae.
- Mycobacterium lepromatosis (diffuse forms of leprosy in Mexico).

Incubation period : 2 – 5 years.

Transmission : Respiratory portal/ inhalation (major) & skin to skin contact.

Staining : Modified Ziehl – Neelsen stain used (5% H_2SO_4).

Generation time : 11 – 13 days.

Pathogenesis : Phenolic glycolipid-1 (PGL-1 of M. leprae).

Culture :
- No artificial culture media available for M. leprae.
- Animal models are used.
  1. Foot pad of mouse.
  2. Armadillo.

M. leprae targets :
- Skin : Macrophages.
- Nerves : Schwann cells.

Nerves :
- First modality to be lost : Sensory.
- First sensory modality to be lost : Temperature (hot/cold indiscrimination).

Course of leprosy :
- Exposure to M. leprae.
- 95% of cases are : Immune to this disease.
  5% of cases : Enter indeterminate leprosy state (cell

mediated response is not yet determined).

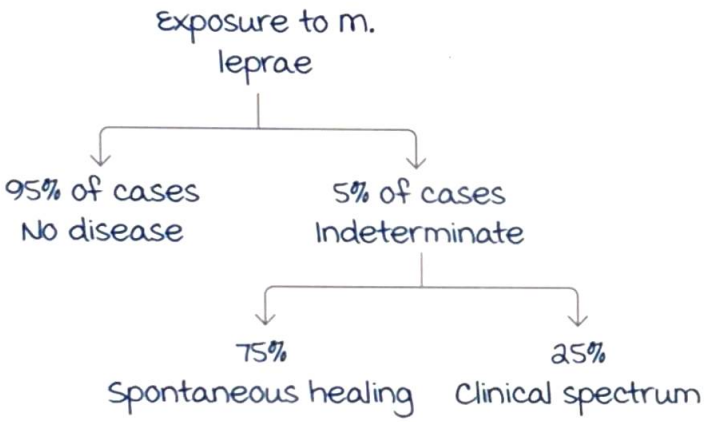
- Indeterminate leprosy :
  75% of cases : Spontaneous healing occurs.
  25% of cases : Enter clinical spectrum of leprosy.

Clinical spectrum of leprosy

00:07:05

Ridley Jopling's classification :

- Based on Cell mediated Immunity (CMI) of the host.
- Tuberculoid pole of classification : Good CMI and limited disease.
- Borderline spectrum.
- Lepromatous pole of classification : Poor CMI and disseminated disease.



5 types of classification in Ridley Jopling's classification :

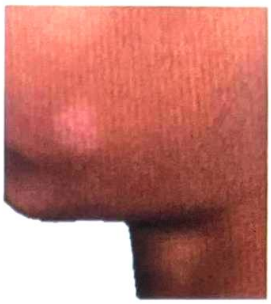
- TT : Tuberculoid type.
- BT : Borderline tuberculoid.
- BB : Borderline borderline.
- BL : Borderline leprosy.
- LL : Lepromatous leprosy.

Decreasing CMI

Indeterminate leprosy :

- CMI : Not yet determined.
- Age : more commonly seen in children.
- Region : Seen in endemic areas like, Bihar, Tamil Nadu, U.P.
- Lesion : Presents with ill-defined hypopigmented macules on the face.
- Biopsy : Perineural, peri appendageal lymphocytosis around the nerves and skin.

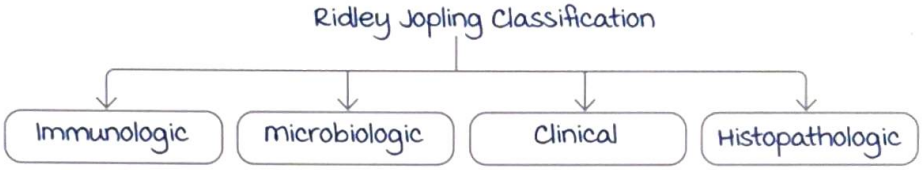
An important differential diagnosis for the same lesion is pityriasis alba (endogenous eczema). But,
Scaling present : Pityriasis alba.
Scaling absent : Indeterminate leprosy.



Determinate type of leprosy

00:12:24

Classified by Ridley Jopling :
Based on 4 important factors.



Immunological :
1. CMI.
2. Lepromin test.

Micorbiological :
1. Acid fast bacilli.
2. Slit skin smear.

Clinical parameters :
1. Skin lesions.
2. Nerve involvement.

Histopathological :
1. Granuloma.
2. Status of macrophages.

Immunological :
As we move from TT → LL, the CMI decreases from good (in TT) to poor (in LL).
Lepromin test : Intradermal test that checks for the individual's immunity.
CMI & lepromin test.
Lepromin Test : Positive in higher CMI & negative in lower CMI.
Positive lepromin test in TT.
Negative lepromin test in LL.

Microbiological :

Acid Fast Bacilli :

TT : Few M. leprae seen.

LL : Numerous M. leprae seen.

Slit Skin Smear (SSS) requires a substantial number of bacteria to turn positive, therefore, even though TT stage has M. leprae, the SSS cannot detect it.

SSS :

TT : Negative.

LL : Positive.

Clinical :

	TT	LL
Skin lesions	1 – 3	Innumerable
Symmetry	Asymmetric	Symmetrical
Margins	Well defined	Ill defined
Nerve	1 nerve thickened	Multiple symmetric nerve involvement

Histopathological :

TT : Tuberculoid granuloma seen.

LL : Deficient immunity (therefore, no granuloma seen).
Foamy macrophages with acid fast bacteria seen.

Clinical features and types of leprosy 00:17:55

Tuberculoid Leprosy (TT) :

1. No of skin lesions : 1 – 3.
2. Morphology of lesion : Annular plaque.
3. Center of lesion : Clearing.
4. Periphery : Well defined raised margin.
5. Associations : Granuloma formed in response to the bacteria, damages the nerves, sweat glands and hair.
   Loss of nerve : Anaesthesia.

Active space

Loss of hair : Alopecia.

Loss of sweat glands : Anhidrosis.

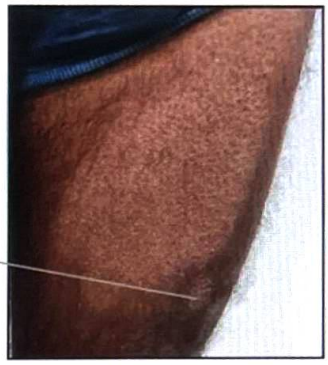
6. Side profile of TT lesion :

Raised well defined margins with sloping inwards towards center appearing like a saucer right way up appearance.

Borderline Tuberculoid (BT) :

1. No. of skin lesions : 3 – 10.
2. Margins : Each lesion has well defined and ill defined (some places) margins present.

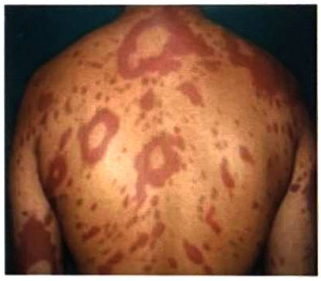
Satellite lesions are also seen.



60c6b3eeaa8ded0e4e7e5ea7

Mid Borderline Leprosy (BB) :

1. No of skin lesions : 10 – 30.
2. Most unstable form of Hansen's disease. There is immunological instability in borderline type of leprosy, therefore, lesions of different morphology are seen.



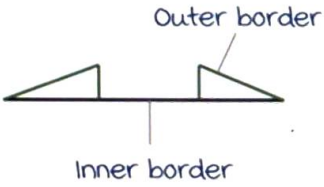
3. Morphology of lesions : Polymorphic skin lesions (varying sizes and shapes) appearing like geographic map like appearance.
4. Margin of lesion : Dimorphous skin lesions seen, hence known as dimorphous leprosy.

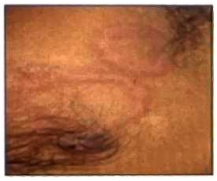
Inner border : Punched out.

Swiss cheese appearance.

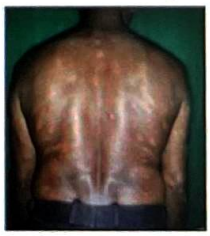
Outer border : Sloping outwards.

Inverted saucer appearance (seen in both BB and BL).





BB leprosy



Borderline Lepromatous (BL) :

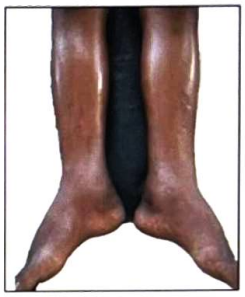
1. Skin lesions : Numerous, bilateral and almost symmetrical.

Active space

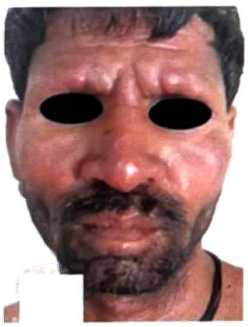
2. Nerve involvement : Bilateral asymmetric nerve thickening.

Lepromatous Leprosy (LL) :

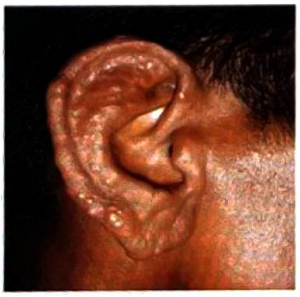
1. Disease : Systemic disease with extensive cutaneous, nerve and internal organs involvement.
2. Skin lesions : Symmetrical bilateral skin lesions consisting of ill-defined macules, papulonodules, diffuse infiltration and thickening of skin.
3. Nerve : Symmetrical bilateral nerve thickenings.
4. Earliest feature of LL :
   a. Epistaxis, nasal crusting and nasal mucosa involvement due to respiratory mode of entry.
   b. Pedal edema in lepromatous leprosy is due to autonomic failure.
5. Late features of LL :
   - Leonine facies/lion like facies.
   - Occurs due to diffuse infiltration of the face.
   - There is loss of eyebrows called madrosis.
   - Glove and stocking peripheral neuropathy.

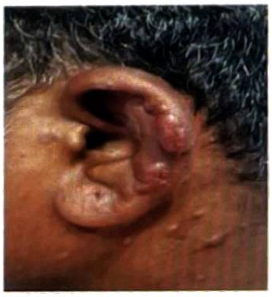


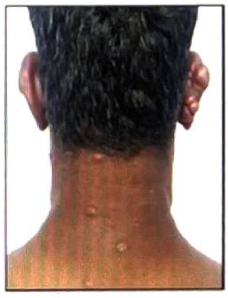
Pedal edema



Leonine Facies





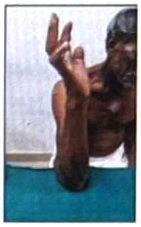


Infiltrative papules

Deformities in leprosy

00:34:07

Deformity : Visible alteration of form, shape or appearance of body part.

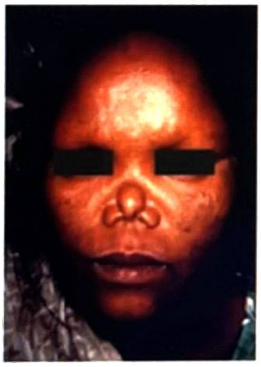


- Lateral Madarosis : Absence of eyebrows.
- Saddle Nose deformity.
- Total claw hand (both ulnar and medial nerve are involved).
- Partial claw hand (if only ulnar nerve is involved).
- The patient can have [illegible] deformities due to sensory impairment.
- Foot drop due to lateral popliteal nerve involvement.
- Eye deformities :

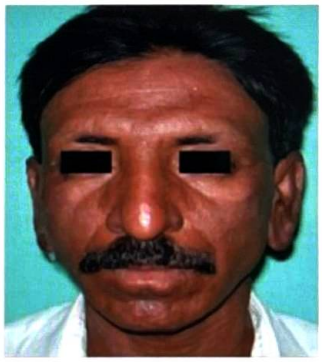
  Lagophthalmos : If orbicularis oculi is involved (facial nerve), the patient won't be able to close their eyes. Predisposes patient to have exposure Keratitis, corneal ulcers which may heal with corneal opacities.
- Trophic ulcers :

  Chronic ulceration in an anaesthetic foot.

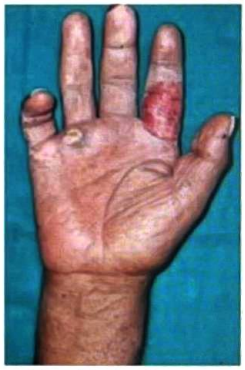
  more commonly found at the soles.



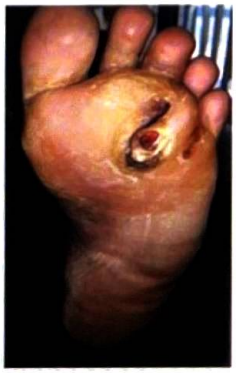
Saddle nose Deformity



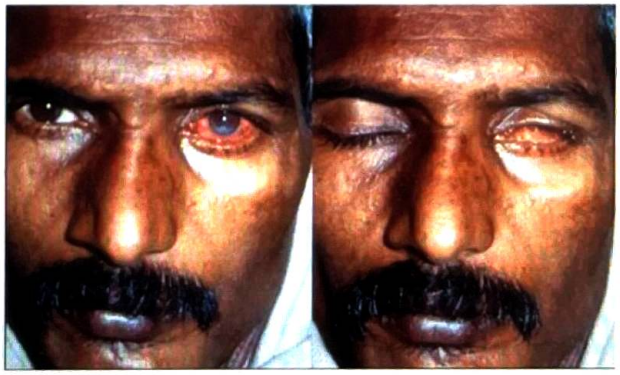
Lateral madarosis



Partial claw Hand



Trophic ulcers

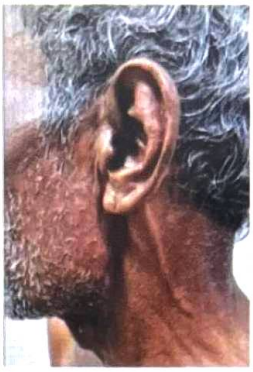


Lagophthalmos

Q. Identify the thickened nerve? (NEET 2018)

Ans. Greater auricular nerve.

This nerve is better seen than felt.



60c6b3eeaa8ded0e4e7e5ea7

Active space

HANSEN'S DISEASE : PART - 2

60c6b3eeaa8ded0e4e7e5ea7

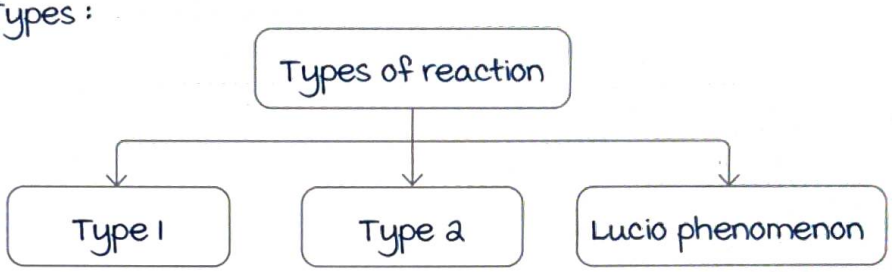
Leprosy is a chronic, infectious and granulomatous disease.

Lepra reaction

00:00:23

Immune mediated episodes of acute inflammation during the chronic course of Hansen's disease.

Types :



Type I lepra reaction :

Spectrum of disease seen in borderline spectrum (BT/BB/BL).

Type of hypersensitivity reaction : Type 4 reaction.

Pathogenesis :

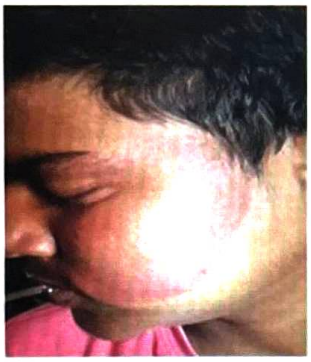
Altered host cell mediated immunity (CMI).

- Upgrading reaction :
  Improvement of CMI (patient's immunity moves towards tuberculoid end).
  Usually occurs on treatment of Hansen's disease.
- Downgrading reaction :
  Cell mediated immunity is low (moves towards lepromatous end).
  Seen in pregnancy.

Clinical features :

- Constitutional symptoms :
  Usually, absent.
  No fever.
- Skin lesions :
  Pre-existing skin lesions suddenly become red, tender and edematous.

- Nerve involvement :
  Severe neuritis.



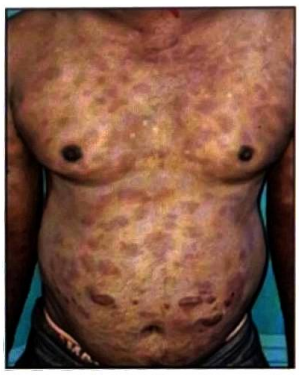
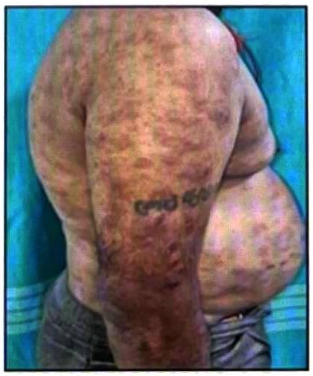

Skin : Type 1 Lepra Reaction

Type 2 lepra reaction :

Aka Erythema Nodosum Leprosum (ENL).
Spectrum of disease : Seen in lepromatous spectrum (BL/LL).
Type of hypersensitivity reaction : Type 3 (immune complex deposition can occur all over body ⟶ systemic features).

Clinical features

- Systemic features :
  Presence of fever.
- Skin lesions :
  New nodules → ENL (red, tender, evanescent nodules).
- Nerve involvement :
  may or may not be involved.

Skin : Type 2 Lepra Reaction

Lucio phenomenon :

Type of reaction seen in Lucio leprosy. 60c6b3eeaa8ded0e4e7e5ea7
(Lucio leprosy is a diffuse, non-nodular leprosy seen in mexico).

Active space

Pathogenesis :

Blood vessels show vasculitis and thrombosis.

Clinical features :

- No fever.
- Present with necrotic ulcer with jagged edges.

Cardinal features in Hansen's disease 00:09:59

For diagnosis :

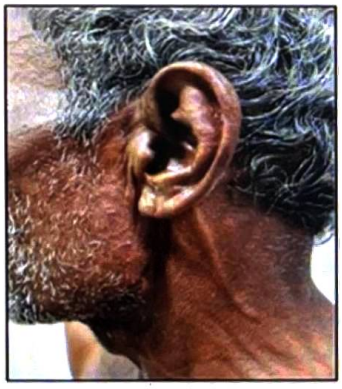
One out of 3 important features should be present.

- Skin lesion :

  Hypo pigmented/erythematous skin lesion with definite loss of sensation.

- Nerve involvement :

  Thickening of peripheral nerves with/without tenderness.



- Slit skin smear – Demonstration of acid-fast bacilli.

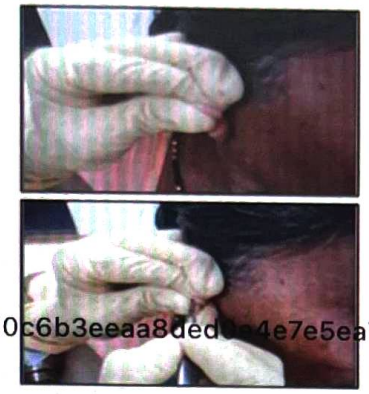
Investigations in Hansen's disease :

1. Slit skin smear :

Procedure by which sample is obtained to demonstrate acid fast bacilli.

Sites :

- Right earlobes.
- Chin.
- Forehead.
- Left buttocks (men).
- Left upper thigh (women).



60c6b3eeaa8ded[illegible]4e7e5ea7

Slit skin smear (staining patterns) :

Modified ZN staining → 5% sulphuric acid → Red colored bacilli. 3 staining patterns observed :

Solid pattern	Fragmented pattern	Granular pattern
		
Live bacilli.	Dead bacilli.	

Bacteriologic index

00:15:05

Density of bacterial load on slit skin smear (for both live and dead bacilli).

Always read on oil immersion field (OIF).

Graded from 1+ to 6+.

6+ : >1000 bacilli/OIF.

5+ : 100–1000 bacilli/ OIF.

4+ : 10–100 bacilli/ OIF.

3 + : 1–10 bacilli/ OIF.

2+ : 1–10 bacilli/10 OIF.

1+ : 1–10 bacilli/100 OIF.

Morphologic index (%) :

Solid staining bacilli expressed in percentage.

Calculation of live bacilli.

Utility : Treatment efficacy and prognosis.

2. Skin biopsy : Histopathology.

- Lepromatous leprosy : Atrophy of epidermis.

Grenz zone :

Sub epidermal free zone (No cellular infiltrate).

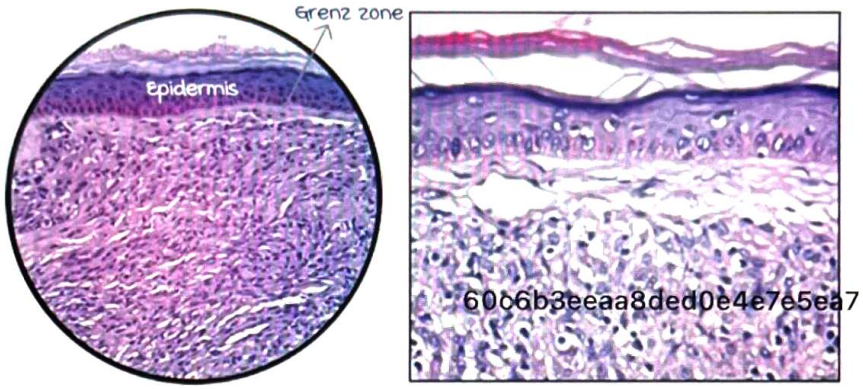
Thick zone.

Active space

Foamy macrophages :

Because of AFB.

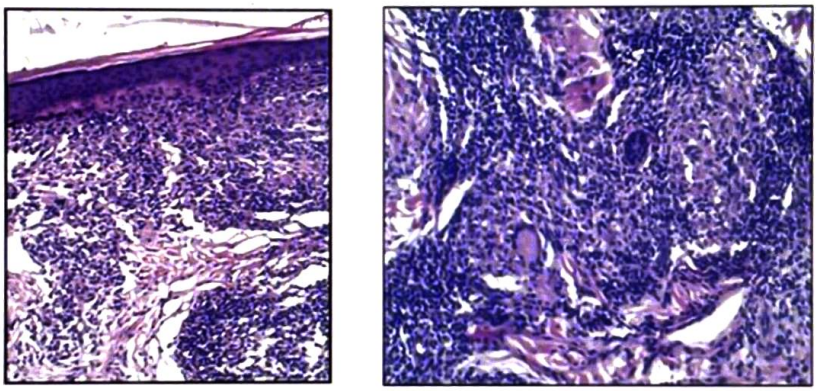
Also known as Virchow's cell/ lepra cell.



- Tuberculoid leprosy :

  Presence of Tuberculoid granuloma.

  Absence of Grenz zone (body tries to eliminate the bacteria and hence the granulomas move up).



Pure neuritic Hansen's disease 00:21:47

Skin lesions are absent, only nerve involvement.

Investigation of choice : Nerve biopsy.

- Only sensory nerves are biopsied.
- Ulnar nerve is never used since it's a motor nerve.
- Upper limb : Radial cutaneous nerve.
- Lower limb : Sural nerve.

1. Lepromin test :

   Intra dermal test.

   Example of type 4 hypersensitivity reaction.

Biphasic reaction :

- Early reaction : Fernandez reaction.

  Reading taken at 48–72 hours.

- Late reaction : Mitsuda reaction.
  Reading taken at 3 weeks (21 days).

Uses of lepromin test :

- Prognosis : Positive → Good prognosis.
- Classification : Ridley Jopling classification.
- Resistance : Positive → High resistance to M. leprae.

Treatment of Hansen's disease.

Classification :

Features	Paucibacillary	multi bacillary
Skin lesions.	1 – 5	≥6
Nerve involvement.	≤ 1 nerve involved.	> 1 nerve irrespective of skin lesion.
Slit skin smear.	Negative	Positive

Treatment of Hansen's disease (WHO regimen).

Paucibacillary : 3 drugs for 6 months.

Multibacillary : 3 drugs for 12 months.

Treatment in adults :

Rifampicin : 600 mg once a month (before food).

Dapsone : 100 mg once a day (preferably at night).

Clofazimine : 300 mg once a month.
50 mg daily on other days.

Treatment in children (10 – 14 years) :

Rifampicin : 450 mg once a month.

Dapsone : 50 mg once daily.

Clofazimine : 150 mg once a month.
Other days : 50 mg on alternate days.

Treatment of Hansen's disease (NLEP regimen).

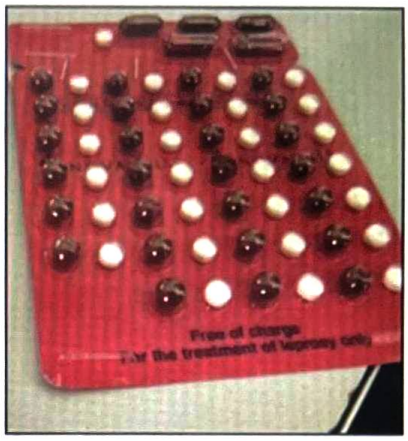
Paucibacillary : 2 drugs for 6 months (Rifampicin+ Dapsone).

Multibacillary : 3 drugs for 12 months.

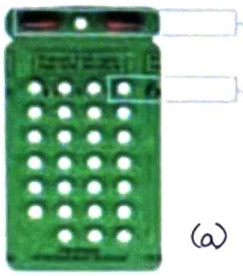
Treatment of Hansen's disease (NLEP regimen) : MB-MDT (maroon kit).

Active space

Day 1 : Rifampicin 300 mg x 2.
Dapsone 100mg.
Clofazimine 100 mg x 3.
Day 2 - 28 :
Clofazimine 50 mg.
Dapsone 100 mg.



Treatment of Hansen's disease (NLEP regimen) : PB-MDT (green kit)



(a)

PB adult treatment :
Once a month : Day 1
2 capsules of Rifampicin (300mg x 2) +
1 tablet of Dapsone (100mg)
Once a day : Day 2-28
1 tablet of Dapsone (100mg)
Full course : 6 blister packs

PB adult blister pack

Side effect profile of MDT

00:31:33

Rifampicin :

- Reddish orange discoloration of secretions (urine).
- Hepatitis.

Dapsone :

- Hemolytic anemia.
- Agranulocytosis.
- Methemoglobinemia.
- Hepatitis.

Clofazimine :

- Pigmentation.
- Ichthyosis.
- GI disturbances.

Newer drugs in leprosy :

- Macrolides : Clarithromycin.
- Ansamycins :
  1. Rifabutin.
  2. Rifapentine

- Tetracyclines : Minocycline.
- Fluoroquinolones : Ofloxacin.

Treatment of lepra reactions :

General management : Continue Multi Drug Therapy.

Type 2 reaction :

Drug of choice : Systemic steroids.

Other options :

- Chloroquine.
- Clofazimine (higher doses).
- Thalidomide (TNF – alpha inhibitor) : Not used much (teratogenic, causes peripheral neuropathy).

Nerve abscess : Incision and drainage + steroids.

National Leprosy Eradication Program 00:36:37

Launched in year 1983.

Elimination : Prevalence of <1/10,000 population.

In India, leprosy is eliminated as a public health problem on December 31st 2005.

NLEP emblem :

Represents : Positive action and hope.

Lotus :

- Beauty and Purity.

Partially affected thumb :

- Leprosy can be cured.
- Leprosy patient can be useful member of the society.

Rising sun :

- Hope and optimism.

Vaccine in leprosy :

MW vaccine.

Strain :

- MIP strain → mycobacterium indicus pranii.
- Designated from India.
- Dr. Pran : Founder of National Institute of Immunology.

Active space

Utility :

- Administered to the contacts of leprosy patient.
- Given along with MDT (immunotherapy) → Faster clearance of bacilli.

Single Dose Rifampicin (SDR) :

Purpose : Chemoprophylaxis for contacts of leprosy patients.

Indications :

- Adults.
- Children >2 years.

Dosage :

600mg (adults).

SEXUALLY TRANSMITTED DISEASES : PART - 1

Anogenital warts

00:00:32

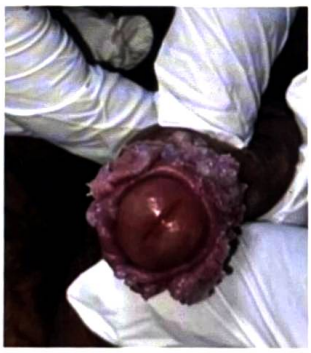
HPV induced skin proliferations involving the anogenital region.
Synonym : Condyloma acuminata (acuminata : Pointed).
Etiology : HPV-6, 11 (low risk HPVs) (HPV 6 > 11).

Clinical features :

- Asymptomatic pointed, fleshy, pink papules and plaques.
- Cauliflower like hypertrophic masses.

Sites :

- Men : Coronal sulcus, frenulum.
- Women : Posterior fourchette

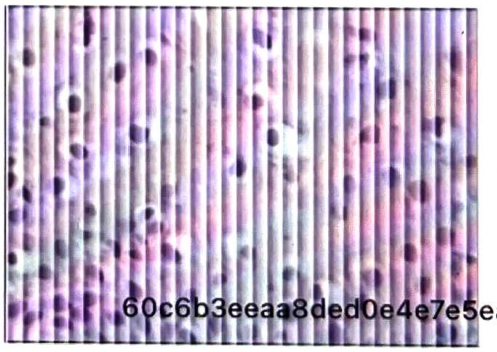


Anogenital warts

Giant condyloma acuminata :
Buschke Lowensteins tumor.

Investigations :

1. Histopathology :
   Biopsy from an HPV induced tissue : Koilocyte cells (squamous epithelial cell with central hyperchromatic nucleus and a perinuclear halo).



Complications :

1) Large vulvar warts → Obstruction of labour during pregnancy.

2) During labour (mother to child) → Laryngeal papillomatoses (if child passes through the infected birth canal of mother).

Treatment :

Non pregnant :

1) Podophyllin resin : Binds to mitotic spindle → Metaphase arrest → Necrosis of warts.
2) Imiquimod : Immune response modifier.
   Toll like receptor 7 agonist → Induces release of cytokines → Clearance of warts.
   Available as 5% cream (thrice weekly for 16 weeks).

Pregnant :

1) Cryotherapy (treatment of choice) : Liquid nitrogen (-196 degree celsius) → Freezes wart.
2) Trichloroacetic acid (TCA) 70-80% → Coagulation of proteins (chemical cauterization).
   TCA is also used for melasma and other pigmented disorder in lower concentration.

Giant condyloma acuminata :

- Surgical excision.

Genital discharge disease

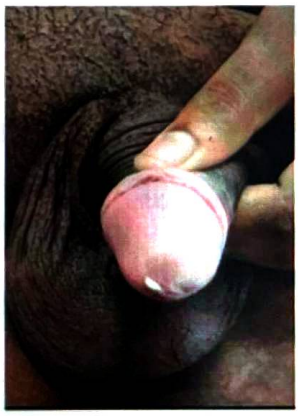
00:11:20

Two types of discharges :

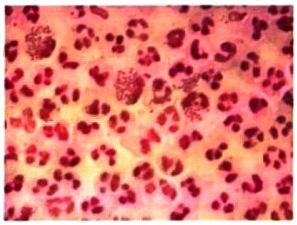
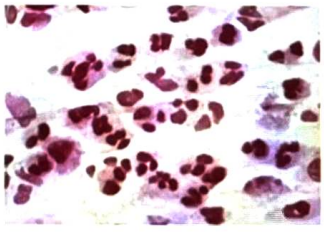
1) Urethral discharge.
2) Vaginal discharge.

Urethral discharge disease :

- Urethritis : Inflammation of urethra characterized by urethral discharge and dysuria.
- Types :
  1) Gonococcal urethritis.
  2) Non gonococcal urethritis.



Urethral discharge

	Gonococcal urethritis	Non-gonococcal urethritis
Etiology	Neisseria gonorrhea.	• Chlamydia trachomatis (D-K). • Ureaplasma ureapyticum. • mycoplasma genitalium. • Trichomonas vaginalis.
Incubation period	2 to 5 days.	7 to 14 days.
Constitutional symptoms	Very severe.	Absent.
Dysuria	Severe (presents early)	Variable.
Discharge	Profuse purulent urethral discharge Gonorrhea (gono : seed, rhea : flow).	Scanty, mucoid urethral discharge.
Investigations : Objective evidence/ diagnostic criteria	1. Visible abnormal discharge : mucoid/purulent/ mucopurulent. 2. Gram stain of urethral swab : ≥ 2 WBCs per oil emulsion field. 3. First void urine : ≥ 10 WBCs/HP field.	
Gram stain of urethral smear	Gram negative intra cellular diplococci within PMNL (polymorpho nuclear lymphocytes). 	Only PMNLs in the absence of organisms. 
Culture media	Modified Thayer Martin medium (Selective medium : Allows only Gonococci to grow & suppress others).	
NAAT (Nucleic Acid Amplification Test)	Done on first void urine. Gonococcal vs chlamydia can be made out through this test.	

Treatment	CDC 2021 criteria : Inj. Ceftriaxone IM stat < 150 Kg : 500 mg. ≥ 150 Kg : 1 g. + Doxycycline 100 mg BD x 7 day(if chlamydia is not excluded).	Doxycycline 100 mg BD x 7 days or T. Azithromycin 1g stat

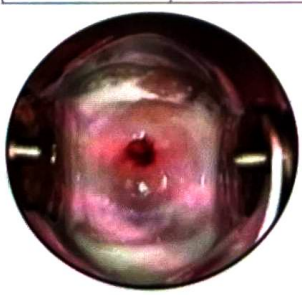
Modified Thayer Martin medium :

- Vancomycin inhibits gram positive organisms.
- Colistin inhibits gram negative organisms.
- Nystatin inhibits fungal organisms.
- Trimethoprim inhibits proteus.

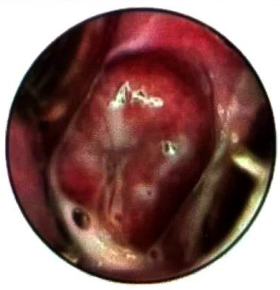
Vaginal discharge syndrome

00:23:45

	Candidiasis	Trichomoniasis	Bacterial vaginosis
Etiology	Candida albicans	Trichomonas vaginalis	Altered vaginal microflora
Discharge	Curdy white vaginal discharge	Greenish yellow frothy vaginal discharge	Homogenous white adherent discharge
Notes		Multiple punctate hemorrhages seen on cervix → Strawberry cervix (colpitis macularis)	Amsels criteria (given below the table)
Wet mount			Clue cells > 20 %
Treatment	Fluconazole		metronidazole



Candidiasis

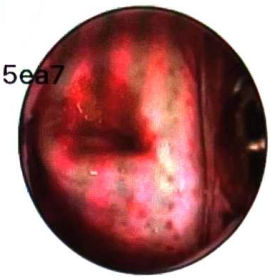


Trichomoniasis

Extra points :

Trichomonas vaginalis (flagellate protozoa) can cause :

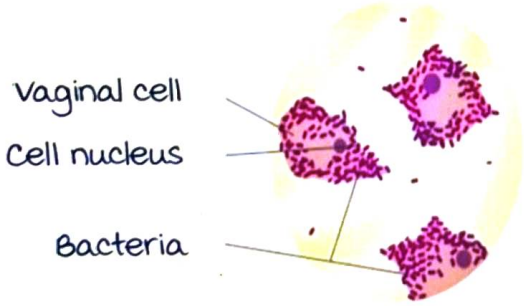
1) Trichomoniasis.
2) NGU (non-gonococcal urethritis).



Cervix (trichomoniasis)

Bacterial vaginosis :

- Amsels criteria :
  1) Vaginal discharge : Homogenous white adherent discharge.
  2) Vaginal pH > 4.5 (Altered vaginal microflora → Less lactobacilli → Less lactic acid → Increased vaginal pH >4.5).
  3) Whiff test/amine test : Vaginal discharge + KOH → Fishy odor.
- Wet mount shows clue cells : Vaginal epithelial cells on surface with stippled border (covered by bacteria).



Bacterial vaginosis

Syndromic management of STDs 00:30:22

Syndrome : Collection of symptoms + signs pertaining to STDs.

Syndromic management : Use of clinical algorithms in treatment of STDs.

Indications :

- Resource poor settings (primary health care).

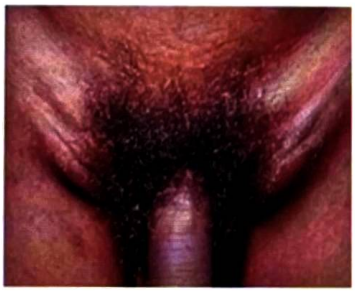
- Lab facilities are not available.

Principle : Treat most important organisms responsible for a syndrome.

NACO : Colour coded kits :

- 7 Kits.
- Remember Number/colour/drugs/indication.

	Color	Indications	Treatment
Kit 1	Grey	Urethral discharge / cervicitis/ anorectal discharge	1) Tab. Cefixime 400 mg/stat (Gonococci). 2) Tab. Azithromycin 1g/stat.
Kit 2	Green	Vaginal discharge	1) Tab. Fluconazole 150 mg 1 stat (for Candidiasis). 2) Tab. Secnidazole 1g 2 stat (for Bacterial vaginosis, Trichomoniasis).
Kit 3	White	Genital ulcer disease : Non-herpetic (Syphilis/ chancroid)	1) Injection Benzathine penicillin 2.4 MU deep/IM stat single dose (for Syphilis). 2) Tab. Azithromycin 1g stat (Chancroid).
Kit 4	Blue	Genital ulcer disease : Non-herpetic (allergic to penicillin)	1) Tab. Doxycycline 100 mg Bd for 15 days (Syphilis). 2) Tab. Azithromycin 1g stat (Chancroid).
Kit 5	Red	Genital ulcer disease : Herpetic	Tab. Acyclovir 400 mg TID for 7 days.
Kit 6	Yellow	Lower abdominal pain (pelvic inflammatory disease)	1) Tab. Cefixime 400 mg 1 stat (Gonococci). 2) Tab. Doxycycline 100 mg Bd for 14 days (Chlamydia trachomatis). 3) Tab. Metronidazole 400 mg BD for 14 days (anaerobes).
Kit 7	Black	Inguinal bubo (enlarged + pus filled lymph node)	1) Tab. Azithromycin 1 g stat (chancroid). 2) Tab Doxycycline 100 mg BD for 3 weeks (Lymphogranuloma venereum).



60c6b3eeaa8ded0e4e7e5ea7

Inguinal bubo

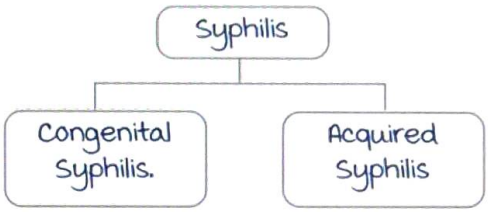
Active space

SEXUALLY TRANSMITTED DISEASES : PART - 2

Syphilis

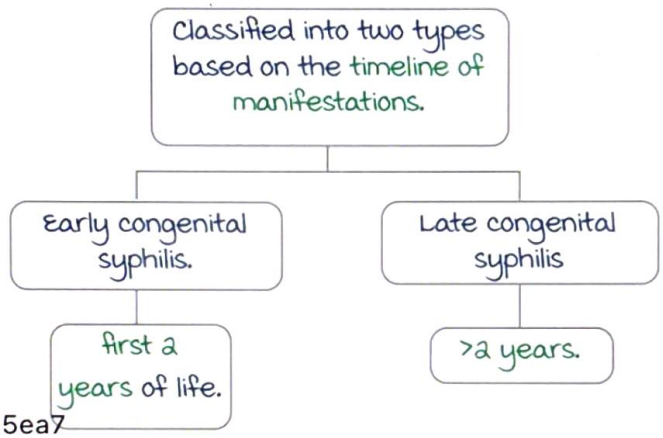
00:00:18

Etiology : Treponema pallidum.



Congenital syphilis.

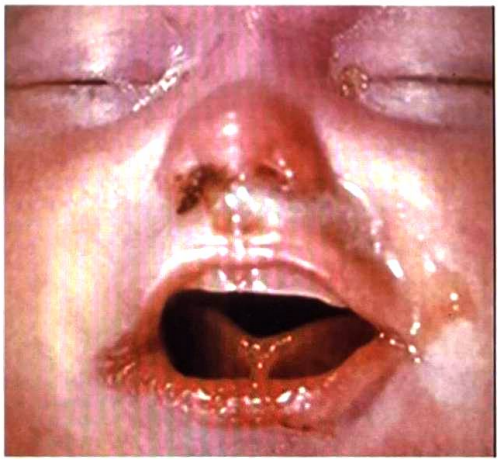
- Mode of acquisition : Mother to fetus in utero.



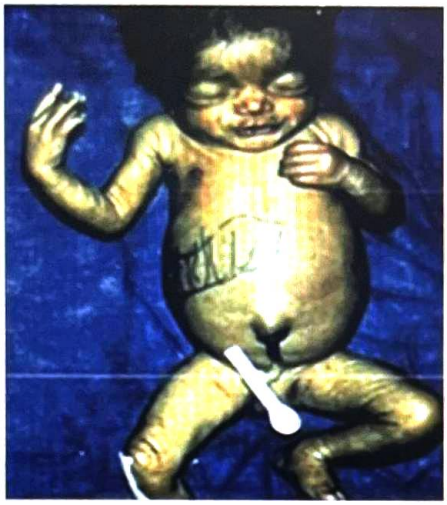
Early congenital syphilis :

Triad :

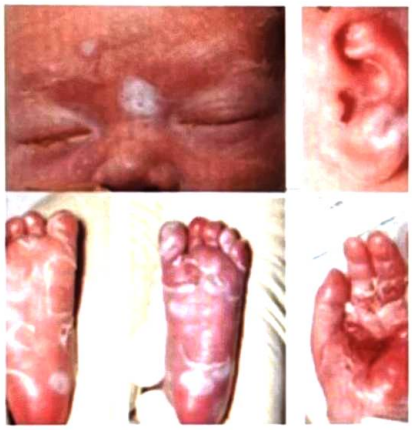
- Snuffles : Persistent rhinitis.



- Hepato-splenomegaly.

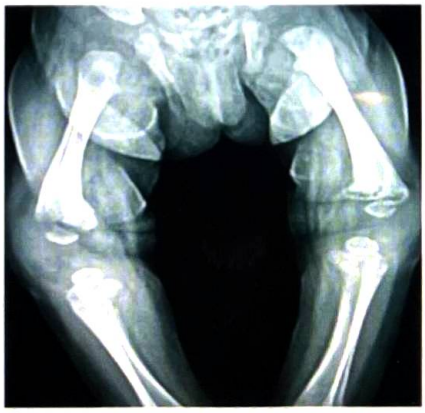


- Syphilitic pemphigus :
  Dermatological manifestation of early congenital syphilis.
  Characterized by Vesiculo-bullous lesions.
  Especially over the palms and soles.



Radiological findings :
Wimberger sign : Bilateral erosion of medial aspect of upper end of tibia. It is a specific sign for early congenital syphilis.

Wimberger sign



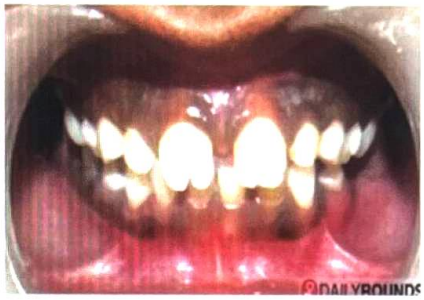
60c6b3eeaa8ded0e4e7e5ea7

Active space

Late congenital syphilis

Triad : Hutchinson's triad.

- Hutchinson's teeth :

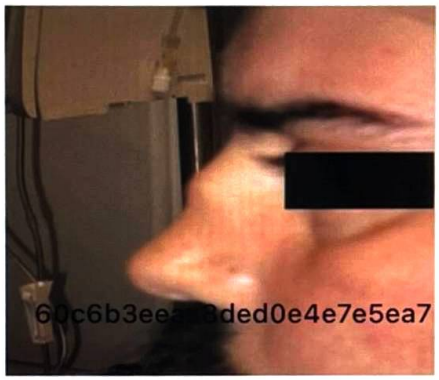


Notching of upper central incisors.

- Interstitial keratitis.
- Sensorineural hearing loss (SNHL).

Other manifestations include :

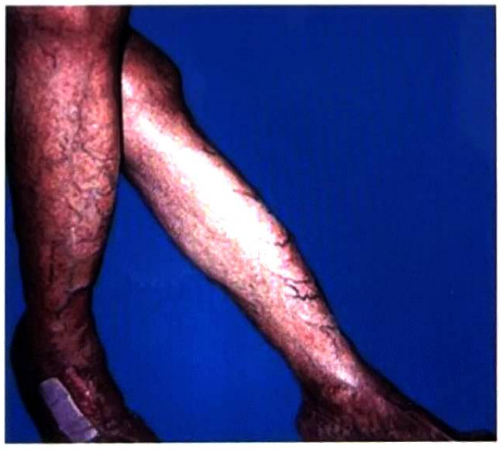
Saddle nose :



Saddle nose

Periostitis :

- Involving frontal bone : Olympian brow.
- Involving unilateral sternal end of clavicle : Higoumenaki's sign.
- Involving anterior aspect of tibia : Sabre tibia.



Sabre tibia

Investigation of Choice : Congenital syphilis.

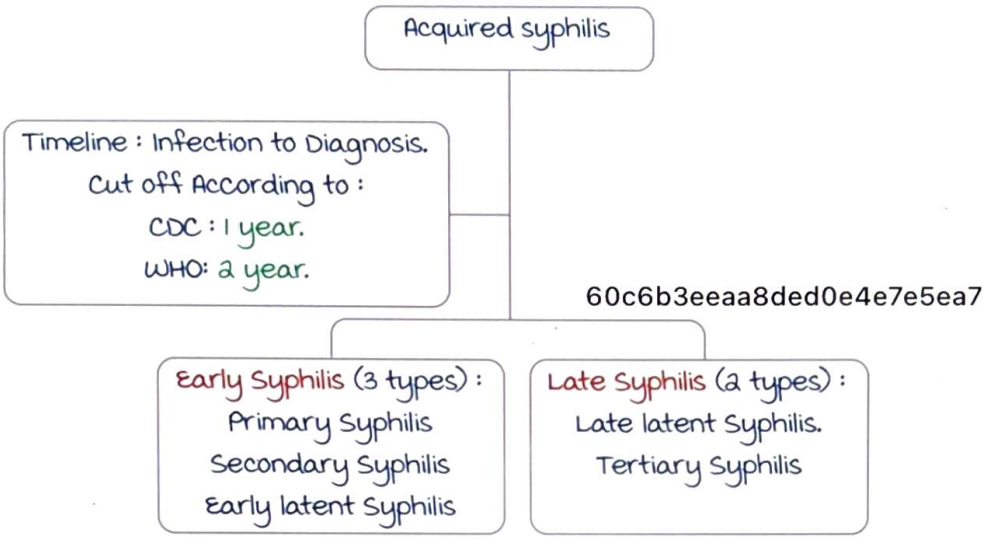
VDRL Titres of Infant should be four times more than VDRL titres of mother.

Acquired Syphilis

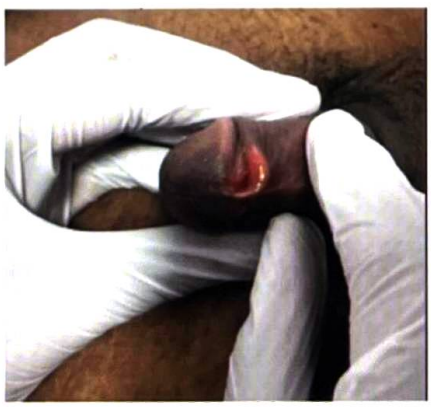
00:07:20

mode of acquisition :

- Sexual transmission.
- Blood transfusion.



1. Primary syphilis :
   - Etiology : Treponema pallidum.
     Trepo : Twist.
     Nema : Thread.
   - Incubation period : 9 - 90 days.
   - Primary lesion : Papule.
   - Secondary lesion : Genital ulcer/ Hard chancre.

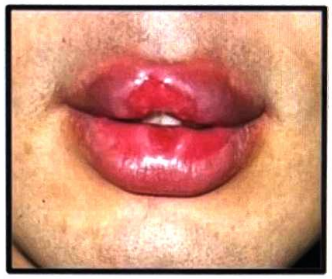


Active space

Description of hard chancre : Single.
Clean based.
Indurated (hard).
Non tender.
Doesn't bleed on touch.

- Inguinal lymph nodes :
  Bilaterally enlarged.
  Non tender.
  Rubbery/ Shotty consistency.

Most common site of extra-genital chancre : Lips.

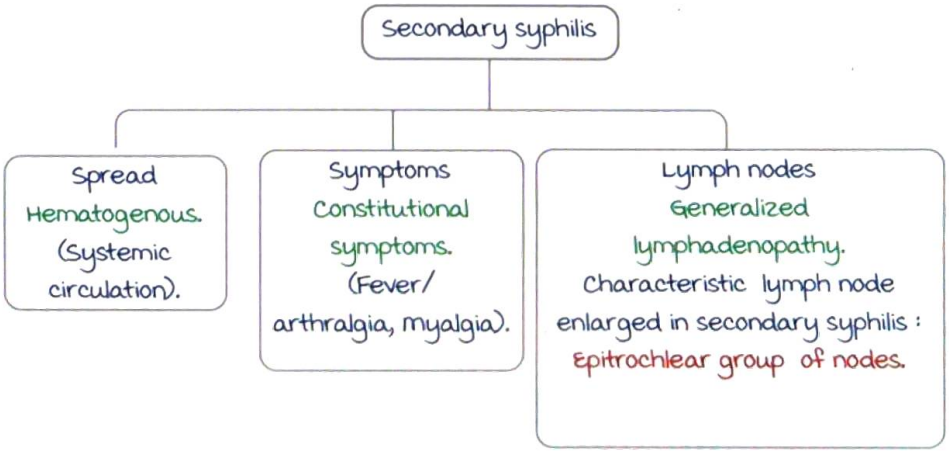


Investigation of choice :
Dark ground microscopy of ulcer exudate : corkscrew motility

ku il.com

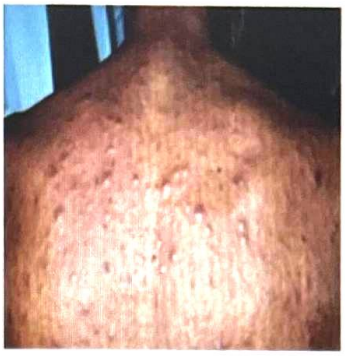
Corkscrew motility

2. Secondary syphilis

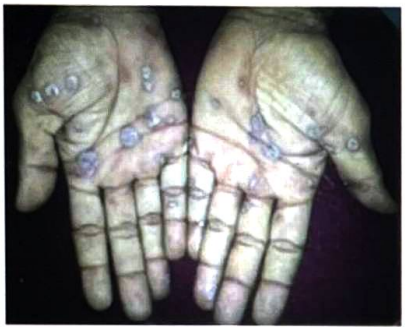


Rash

- Morphology :
  Symmetric.
  Polymorphous.
  Non pruritic (generally).



- All types of morphology of rash seen (except Vesicles and bullae).
- Characteristic of secondary syphilis : Examination of palms & Soles.



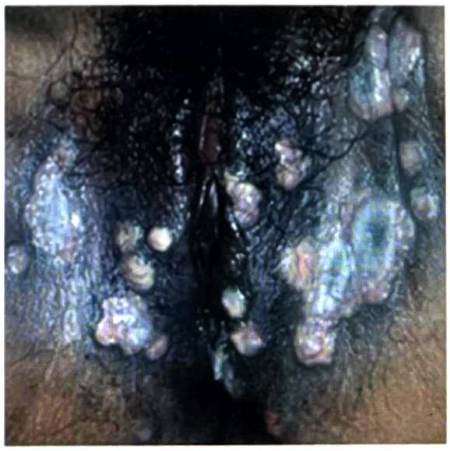
Buschke Olendorrf Sign 00:17:00

Pressing the lesion of secondary syphilis with the blunt end of Pin will lead to deep dermal tenderness.

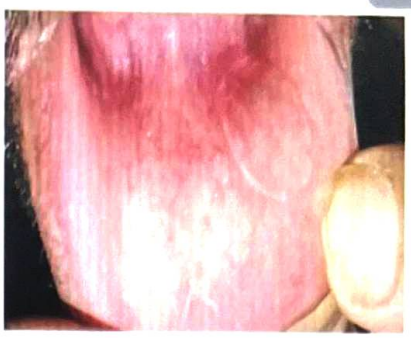
Mucosal lesions

- Condylomata Lata

Lata : Means flat.
Lesions are flat topped, moist papules and plaques.
Accuminata means pointed.



- Mucous patches
  Result due to breakdown of mucosa secondary to secondary syphilis.

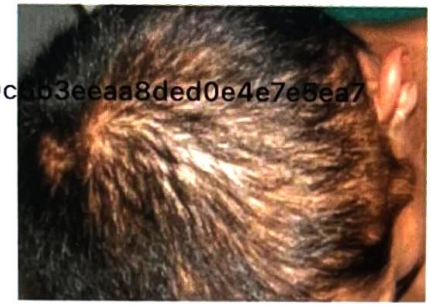


- Snail track ulcers.

Alopecia

Moth eaten alopecia.

Non scarring type of alopecia.



Syphilis d'emblee

00:20:22

- Route : Blood transfusion.
- Special feature : No chancre.
  Directly presents with secondary syphilis.
  d'emblee : means straightaway.
- Investigation of choice in secondary syphilis :
  Venereal Disease Research Laboratory test/VDRL
  Rapid Plasma Reagent test/RPR

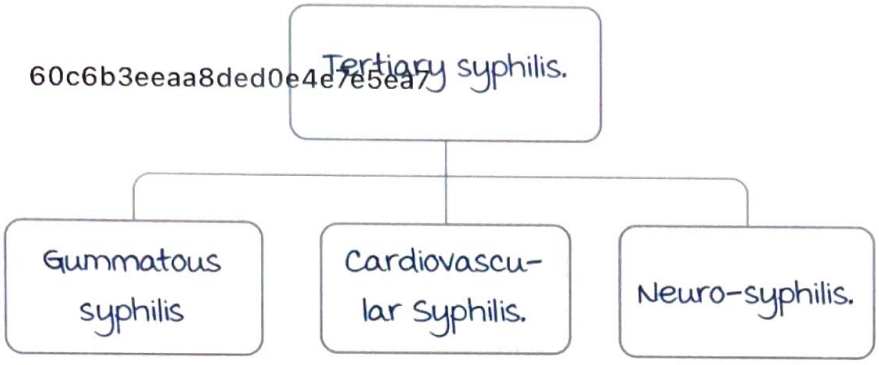
3. Latent Syphilis
   - Early latent syphilis
     Patient is infectious.
   - Late latent syphilis
     Patient is non infectious.

NO symptoms/ signs.

Diagnosis : Serological test.

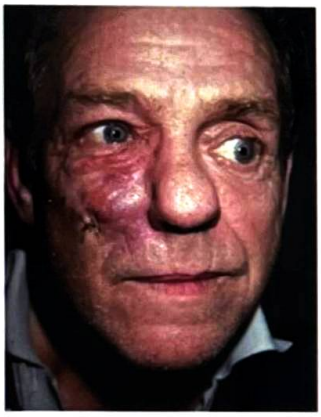
IOC : Enzyme Immuno Assay.

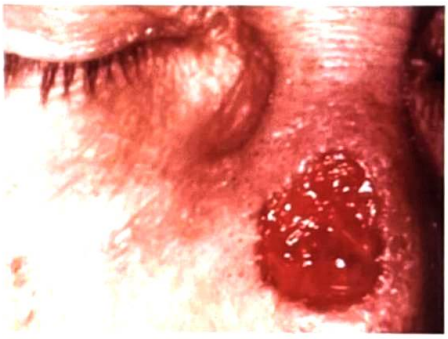
4. Tertiary syphilis



Gummatous syphilis

- Gumma : Granulomatous rubbery nodule.
- Gumma breakout to form punched out edge ulcer.
- Characteristic slough of ulcer is called wash leather slough.





Cardiovascular syphilis :

Two Important manifestations :

1. Aortic aneurysm :
   Most common site : Ascending aorta.
2. Aortic regurgitation :
   Most common valvular heart disease.

Neurosyphilis :

Two manifestations

- General Paresis of Insane :
  Rapidly progressive dementia.
  Personality changes.
  Delusions.
  Hallucinations.

Active space

- Tabes dorsalis :
  Slow progressive degeneration of the sensory neurons of dorsal column of the spinal cord.

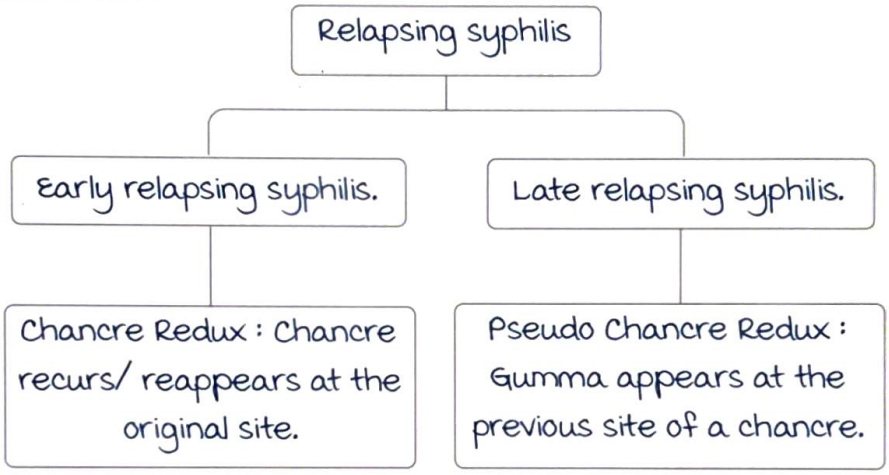
1. Gait abnormalities : Locomotor/ sensory ataxia.
2. Lancinating Pains : Episodes of severe stabbing kind of pain in face & extremities.
3. Argyl Robertson pupil : Bilateral small pupils.

Investigation of Choice of Neurosyphilis : CSF-VDRL.

Relapsing Syphilis :

Term : Redux means 'To comeback'

Classification :



Treatment of Syphilis

00:30:38

Drug : Injection Benzathine penicillin.
Dosage : 2.4 million units.
Route : Deep Intramuscular.
Duration :

Early Syphilis : Single dose.
Late Syphilis (except neurosyphilis) : 3 doses (once weekly).

Treatment of Neurosyphilis
Drug : Injection Aqueous crystalline penicillin.
Dosage : 18–24 million units per day.
Route : Intravenous.
Duration : 10–14 days.

Treatment : Allergic to Penicillin (Non-Pregnant) :

Early Syphilis : Doxycycline 100 mg BD for 2 weeks.

Late Syphilis : Doxycycline 100mg BD for 4 weeks.

Treatment : Allergic to Penicillin (Pregnant) :

Desensitization followed by Penicillin.

- Repeated administration of sub-threshold doses of drug (Penicillin).
- Antigenic determinants bind IgE on surface of basophils and mast cells without cross-linking.
- Rendering these cells unresponsive to higher doses of the drug.

Complications of Penicillin Therapy

Hypersensitivity Reaction :

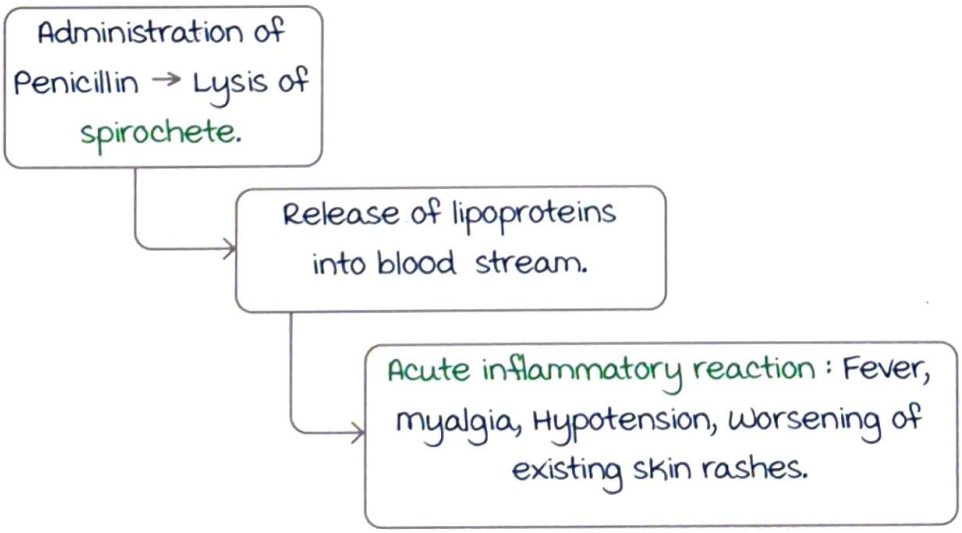
- Urticaria.
- Angioedema.
- Anaphylaxis.

Jarisch Herxheimer Reaction

Definition : Acute febrile self limiting reaction seen within 24 hours of administering penicillin in a patient.

60c6b3eeaa8ded0e4e7e5ea7

Pathogenesis



Treatment

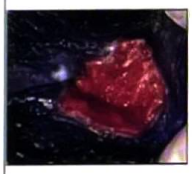
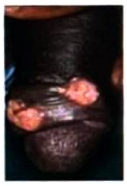
- Self resolution.
- Symptomatic Treatment : NSAIDS.

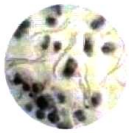
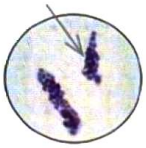
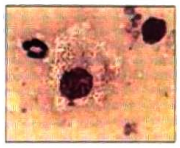
Active space

SEXUALLY TRANSMITTED DISEASES : PART - 3

Non syphilitic genital ulcer diseases

00:00:19

	Chancroid	Herpes genitalis	Lymphogranuloma Venereum	Donovanosis
Etiological factor	Hemophilus ducreyi	HSV 2 (90%) or HSV 1 (10%)	Chlamydia Trachomatis (L1, L2, L3)	Klebsiella granulomatis (Calymmatobacter granulomatis)
Incubation period	2-5 days	3-12 days	3-30 days	8-80 days
Clinical presentation Primary lesion	Pustule	Vesicle (clear fluid filled lesion < 1 cm) 	Primary stage: Genital ulcer.	Genital ulcer : Beefy red (increased vascularity). Painless. Exuberant granulation tissue. 
Secondary lesion	Ulcer 	Vesicles rupture → Genital ulcers 		
Ulcer	Soft chancre/ soft sore : multiple, Necrotic based, Non indurated/ soft, Bleeds on touch, Tender, Undermined edges.	multiple tender ulcers. Grouped. Polycyclic margins.	Painless. Herpetiform. Transient ulcer.	
Inguinal region	Bubo : Enlarged, pus filled, tender, inguinal lymph node, Unilateral. 	B/L enlarged tender lymph nodes. No bubo formation.	Secondary/inguinal stage : Bubo - Enlarged, Tender lymph nodes. Unilateral (2/3rd)/ Bilateral (1/3rd). Groove sign of green Blatt : Enlarged lymph nodes above and below the inguinal ligament causing groove. 	Lymph nodes : Not involved. Normal. Also called granuloma inguinale. Granuloma : Subcutaneous nodule. Inguinale : Groin. Subcutaneous nodule in groin = Pseudo bubo (not a lymph node).

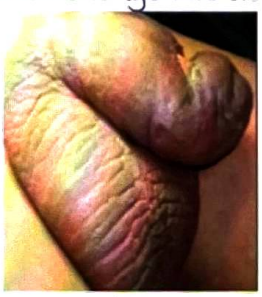
	Concomittant genital ulcer is present		Tertiary stage : Deformities. Genital Elephantiasis. (See below the table)	
Investigation	Gram stain of exudate → gram negative coccobacilli. Arranged in long parallel strands. Railroad track appearance. School of fish appearance. 	Tzanck Smear (cyto diagnostic test) Multinucleated giant cells 	Nucleic Acid Amplification test	Specimen → Crush it between a glass slides (crushed tissue smear) 
	Mueller Hinton agar + 5% chocolatized horse blood agar.	Vesicle → Scrape the floor and undersurface of the roof → material smeared with Giemsa stain → Cells with multiple nuclei (Multinucleated Giant cells).		Stain with Giemsa stain → Mononuclear cell (Pund cell). Inside the cell → Intracellular Donovan bodies (closed Safety pin appearance : Due to bipolar condensation of chromatin)
Treatment	Tablet Azithromycin (macrolides) 1 g stat.	Tablet Acyclovir 400 mg thrice daily → 7 days. Syndromic management : Kit no 5 (Red).	Doxycycline 100 mg BD → 3 weeks.	Tablet Azithromycin 1 gm per week. (Or) CDC 2021 guidelines : Tablet Azithromycin 500 mg daily → 3 weeks till ulcer heals.

60c6b3eeaa8ded0e4e7e5ea7

Genital elephantiasis :

Men : Saxophone penis.

Women : Esthiomene (genital elephantiasis + chronic ulceration).

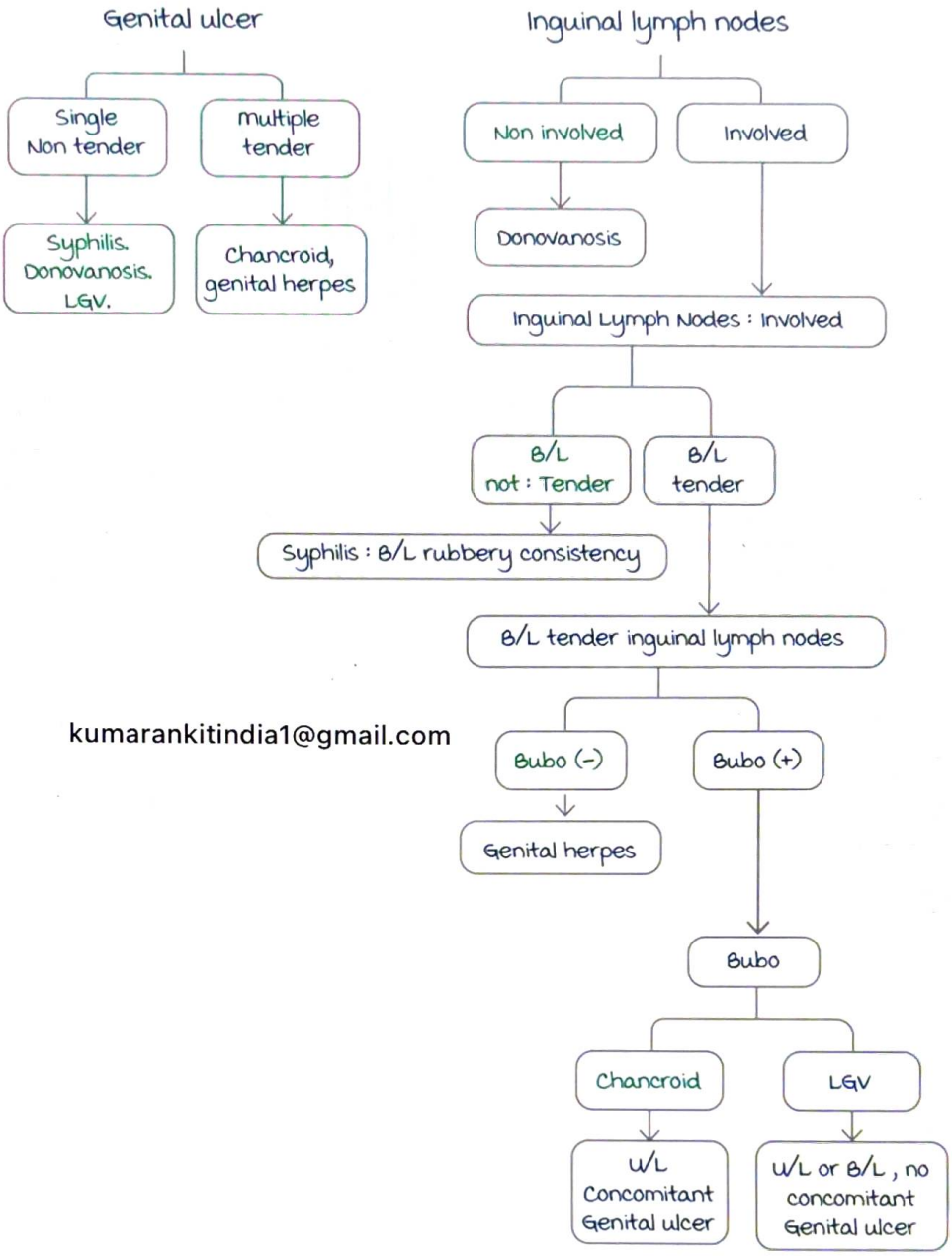


Men : Saxophone penis

Women : Esthiomene

Active space

Approach to a genital ulcer



GENODERMATOSES

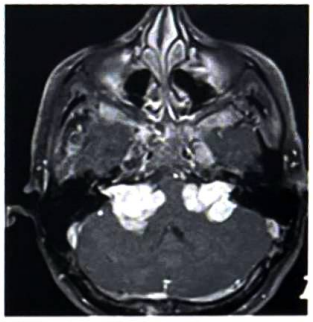
Definition : Genetic diseases with skin manifestations.

Neurofibromatosis :

Autosomal dominant.

Types → Type 1 NF : Also referred as Von Reckling Hausen disease.

→ Type 2 NF : Also referred as MISME.



MISME : Multiple Inherited Schwannomas Meningiomas & Ependymomas.

Characterised by bilateral acoustic schwannoma.

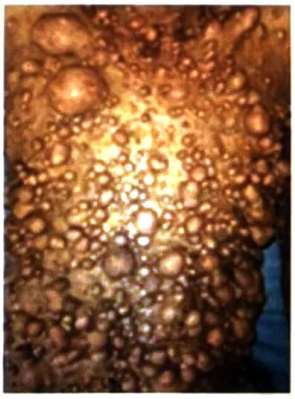
	Type 1	Type 2
Gene	NF 1	NF 2
Chromosome	17	22
Protein	Neurofibromin	merlin or Schwannomin

NF -1

00:03:35

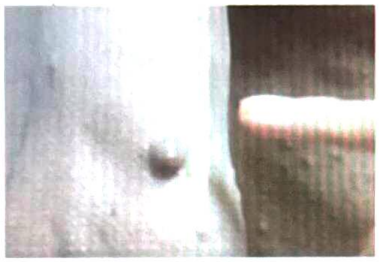
Lesions In NF 1 :

1. Neurofibromas (Nf) :
   Defined as the benign tumor of connective tissue of peripheral nerve sheath.
   Onset : Adolescence.
   Lesions : Soft, skin coloured, non-tender, papules or nodules.



Button hole sign : Clinically, sign elicited on pressing the NF.

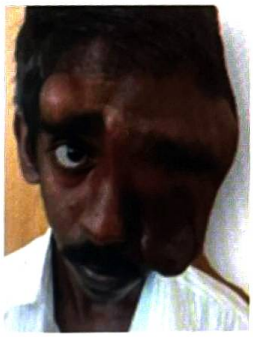
It gets invaginated by finger pressure (On applying pressure, the lesion goes back in the skin and comes out on removing the pressure).



Applying pressure

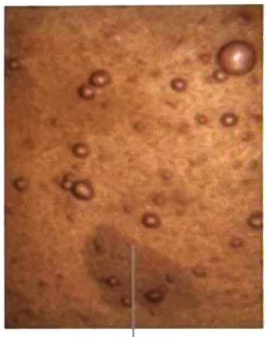
Invaginated by finger pressure

2. Plexiform NF :
   Origin : multiple nerve fascicles.
   Onset : Congenital
   Lesions : Diffuse expanding plaque. Bag of worm feel on palpatation.



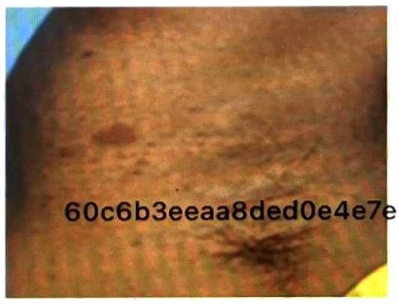
Plexiform Nf

3. Cafe-Au-Lait macules :
   Hyperpigmented macules.
   1 to 2 lesions are normal.
   If > or = 6 macules : Diagnostic.
   Size :
   Prepubertal > 5 mm.
   Post pubertal > 15 mm.



Cafe-au-lait macules

4. Skin fold freckling
   Lesion : Hyperpigmented macules.
   Seen in : Axilla which is called as Crowes Sign and it is pathognomic for NF-1.
   Also seen in groin.

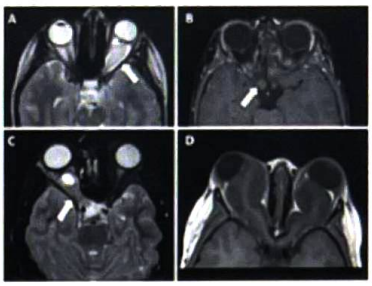


Ocular manifestation in NF-1 :

- Lisch iris nodules :
  Pigmented iris hamartomas.
  Best visualized in slit lamp examination.
  No effect on visual acuity.

LISCH Iris nodules

- Optic pathway glioma :
  Clinically present with proptosis.
  Also called as optic glioma.



Optic Pathway Gliomas

Tuberous sclerosis complex

00:11:17

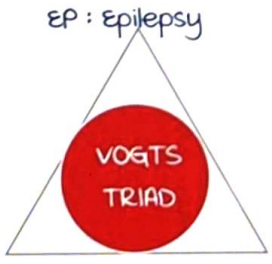
Autosomal dominant.

Also called Bournvilles disease.

Genetics	TSC 1	TSC 2
Chromosome	9	16
Protein	Hamartin	Tuberin

Triad is known as EPILOIA/Vogts triad.

EP: Epilepsy LOI : LOw Intelligence A : Adenoma sebaceum



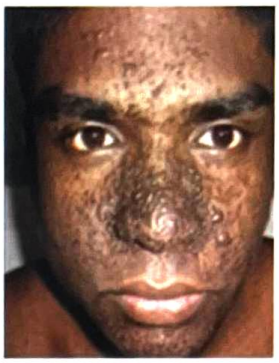
Major skin features :

1. Ash leaf macules :
   Earliest skin lesion.
   Hypopigmented lesion.
   Lance ovate shape : Oval on one side & pointed on another side.

Active space

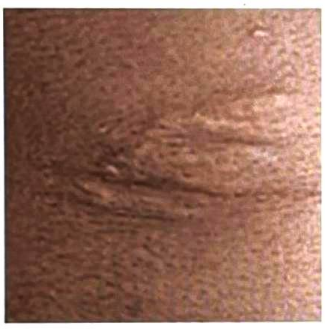
O/E : In an infant with epilepsy (infantile spasm) look for ash leaf macules by woods lamp examination.

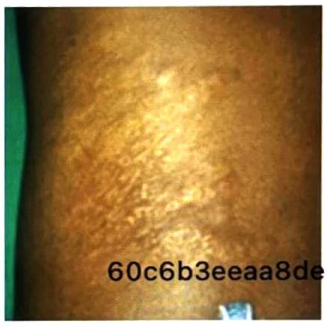
2. Adenoma sebaceum :



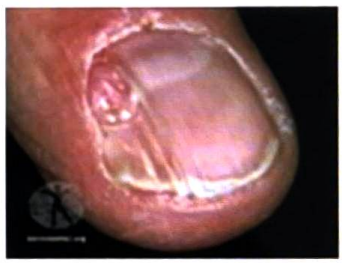
Also known as facial angiofibroma.
Proliferation of blood vessels and fibrous tissue.
Lesions are symmetrical reddish brown papules.
Seen in malar area, nasolabial fold, nose.
It is characteristic of tuberous sclerosis complex.

3. Shagreen patch :
Leathery plaque.
Located in lumbosacral region.
Composition : Collagenoma (made of collagen).



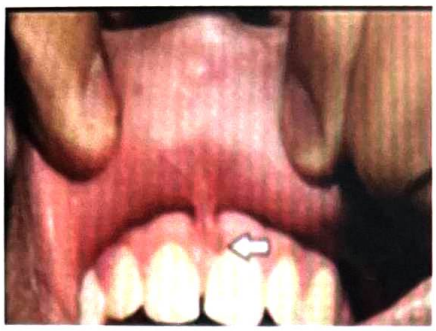


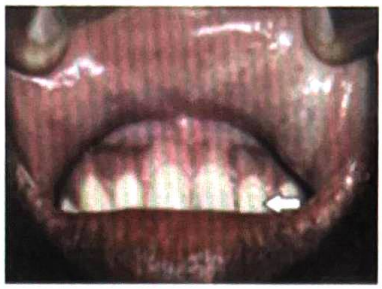
4. Koenens tumors (KT)
Seen at puberty.
Periungual fibroma is characteristic of KT.



Minor skin features of Tuberous sclerosis

- Skin : Confetti macules. Hypopigmented macules.
- Dental enamel shows pit.
- Fibroma in gingival mucosa.





Systemic features of TS

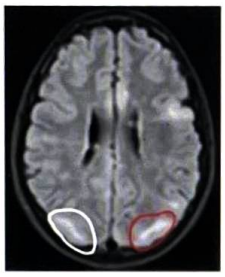
- Kidney : Renal angioma, myolipoma (benign renal neoplasia)
- Lung : Pulmonary lymphangioleiomyomatosis
- Heart : Cardiac rhabdomyoma
- Retina : Hamartoma

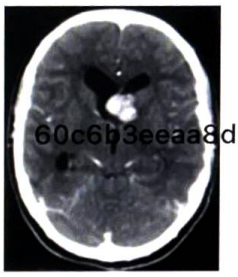
CNS :

Cortical tubers present.

Subependymal nodule.

Subependymal giant cell Astrocytoma.



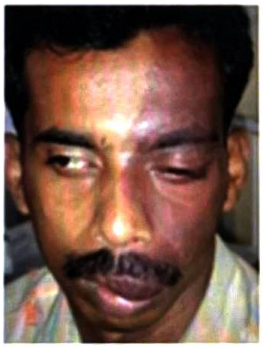


Sturge weber /Encephalo trigeminal angiomatosis

00:22:28

Inheritance is sporadic .

Manifestation :



- CNS : Epilepsy (due to leptomeningeal angioma).
- Eye : Glaucoma .
- Skin lesion : Facial port wine stain.
- Pathology : Low flow capillary vascular malformation.
- Onset : At birth.
- Port wine stain is permanent.
- Lesion is unilateral.
- Colour is pink-red overtime turning to purplish.
- Site : face.
- Treatment : Pulse Dye Laser (PDL).

Active space

Ichthyosis classification :

- Congenital :
  - Ichthyosis vulgaris.
  - X Linked recessive Ichthyosis.
  - Lamellar Ichthyosis.
- Acquired
  - Hypothyroidism .
  - Leprosy.
  - Hodgkins Lymphoma.
  - HIV .
- drugs :
  - Clofazamine.
  - Nicotinic acid.
  - Statins.

Ichthyosis vulgaris

00:27:10

Most common form.

Onset : 3 month to 12 months of age.

Autosomal dominant inheritance.

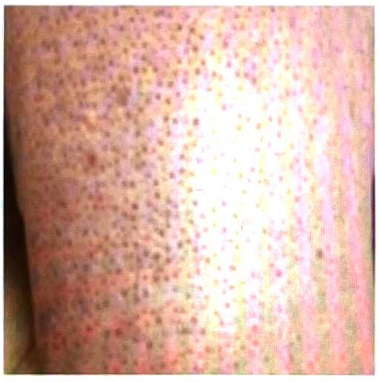
Defect in filament aggregating protein (filaggrin).

Scales : Fine white scales.

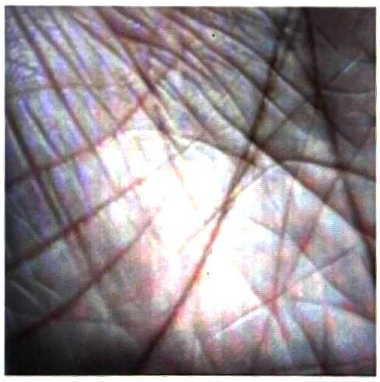
Spared areas : Faces and flexures.

Associations :

1. Keratosis pilaris :
   Hair follicle keratinisation defect.
   Usually in outer aspect of arm.
   Multiple follicular papules.
   Cosmetic concern.
2. Hyperlinear palms : Exaggerated lines on palms.



Keratosis pilaris



Hyperlinear palm

X-linked ichthyosis :

Also called as Ichthyosis nigra (brown black skin).

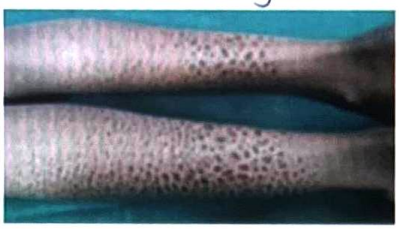
Onset < 3 months of age.

X-linked recessive defect.

Defect in steroid sulphatase.

Responsible for conversion of cholesterol sulphate to cholesterol.

X linked Ichthyosis



Defect causes increase in cholesterol sulphate → Dry skin.

Spared area is palm and soles.

Association :

- Corneal Opacities.
- Cryptorchidism (undescended testis).

Lamellar ichthyosis

00:32:13

Onset is at birth.

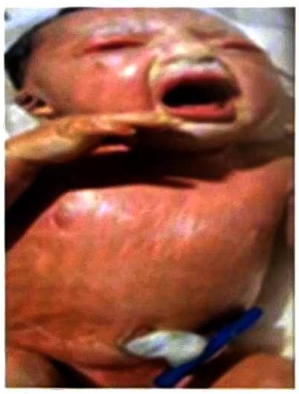
Autosomal recessive.

Defect is in epidermal transglutaminase I.

At birth, the child's body is covered with parchment like translucent membrane called collodion membrane.

Gradually, this membrane turns to dark plate like adherent scales.

L. ichthyosis, onset

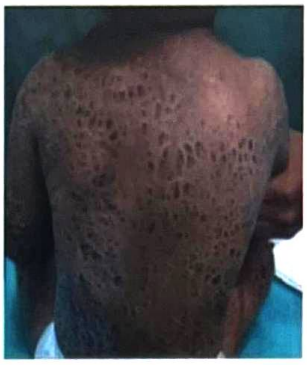


Translucent, parchment like membrane covers the neonate.

↓

After 4 weeks, dark plate like adherent scales are formed.

L. ichthyosis scales



Associations :

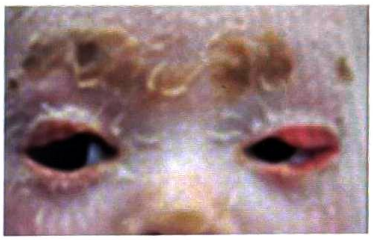
Eye : Ectropion (outward turning of eyelid margin).

Lips : Eclabium (outward turning of lip).

Treatment

Topical : Emollients.

Systemic : Retinoids (Acitretin).



Ectropion

Incontinentia pigmentii

00:35:17

60c6b3eeaa8ded0e4e7e5ea7

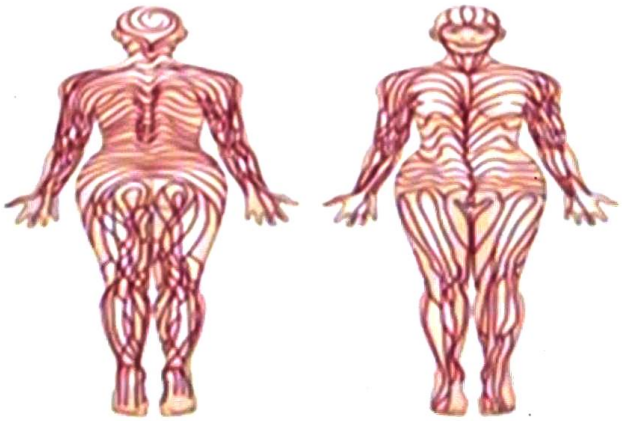
Genetic ectodermal dysplasia involving skin, CNS, eyes, teeth.

X linked dominant.

Features :

Follows lines of epidermal cell migration during embryological development called as Blaschko Lines (BL).

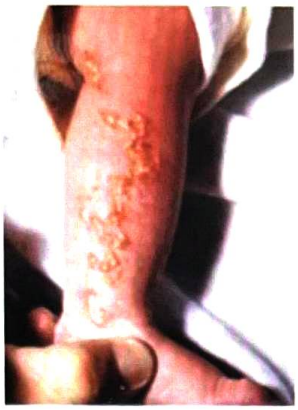
The lines are seen to be spiral over scalp and linear over lower extremities.



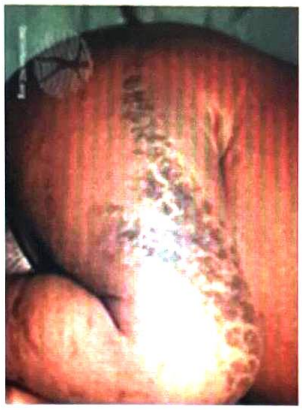
Clinical phases :

1. Vesicular phase : Vesicles along blaschko lines.
2. Verrucous phase : Rough surface called verrucous.
3. Hyperpigmented phase.

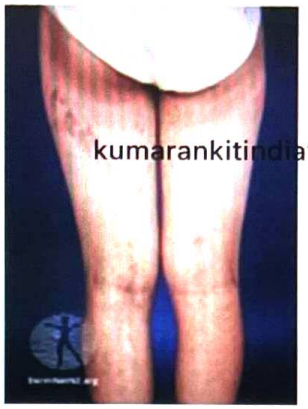
4. Hypo pigmented phase.



Vesicular



Verrucous



Hyperpigmentation

Ocular manifestations :

fundus fluorescenceangiography : Avascular peripheral retina seen.

Xeroderma pigmentosum(XP) 00:38:22

Inherited disorder of DNA repair.

Characterized by cellular hypersensitivity to UV radiation.

Autosomal recessive inheritance.

Defect in Nucleotide Excision Repair (NER).

Function of NER :

1. Normal : Excise damaged/abnormal DNA that is UV radiation induced.
2. Defect →

Accumulation of abnormal damaged DNA.

↓

Genomic instability .

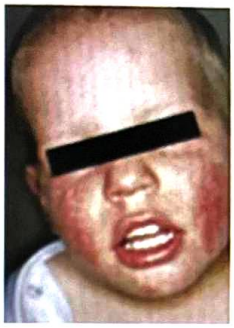
↓

Carcinogenesis.

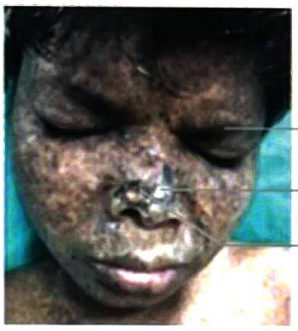
Lesions :

- Sunburn : Radiation burn on UV exposure.
  Characterised by erythema.
  In normal person, sunburn develops only on prolonged exposure to sun.

In XP patient, minimal exposure to sun itself causes sunburn.



- Multiple freckles :
  Hyperpigmented macules
- Skin malignancy :
  Basal cell carcinoma.
  Squamous cell carcinoma.



Continuous exposure to sunlight.

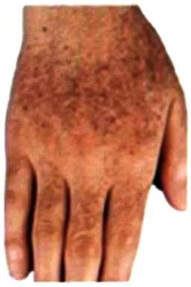
↓

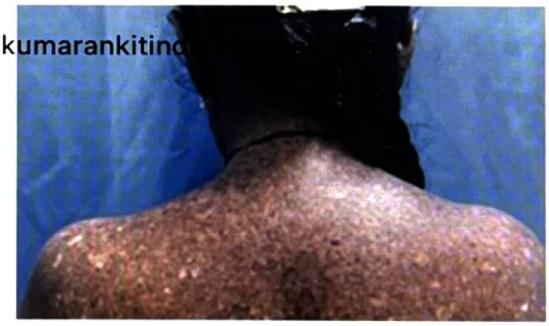
Photoageing.

↓

Skin becomes dry and it is called as Xeroderma.

Xerodermas





Systemic features :

- CNS :
  In 25% mental retardation.
  Areflexia.
  Ataxia.
  Sensorineural hearing loss.

- Systemic : Haematological malignancies like :
  - myelodysplastic syndrome.
  - AML.
  - ALL.

Treatment :

Retinoids : Isotretinoin/acitretin.

↓

modulate keratinocyte differentiation.

60c6b3eeaa8ded0e4e7e5ea7

↓

Prevent neoplasm in XP.

ECZEMA

Definition

00:00:40

Reaction pattern of the skin secondary to the endogenous (internal) or exogenous (external) stimuli.

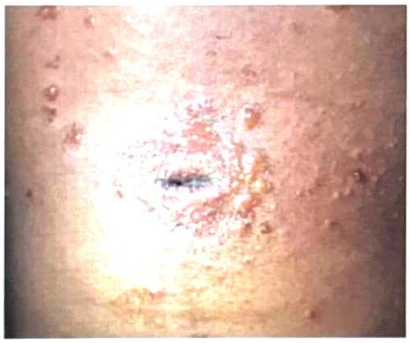
Morphological classification :

1. Acute.
2. Subacute.
3. Chronic.

Acute eczema :

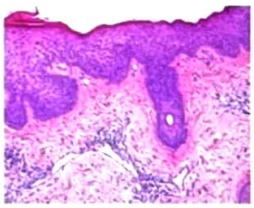
Characterized by all signs of acute inflammation of the skin :

- Erythema.
- Edema.
- Vesicles.
- Oozing.
- Crusting.



Histopathology :

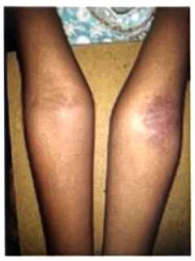
Spongiosis : Intraepidermal intercellular edema.



Subacute eczema :

Characterised by

- Erythema
- Scaling

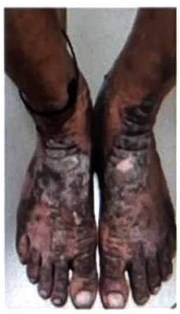


Chronic eczema :

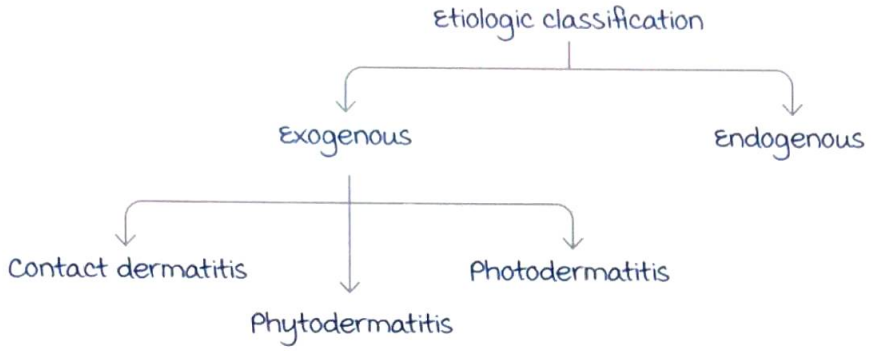
It happens as a response to chronic itching.

Lichenification (as a result of continuous itching) :

- Hyperpigmentation.
- Thick skin.
- Exaggerated skin markings.



Etiologic Classification



Phytodermatitis : Plant induced.

Photodermatitis : Sun light induced.

Contact dermatitis

00:05:10

Inflammation of the skin secondary to contact between skin & substance.

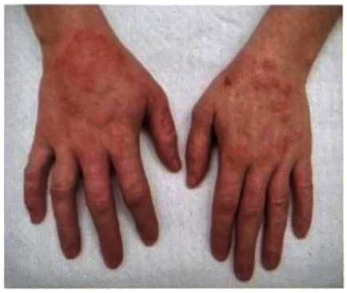
Two types :

1. Irritant contact dermatitis.
2. Allergic contact dermatitis (CD).

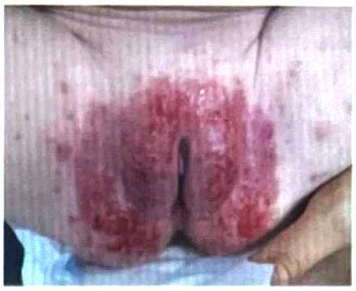
	Irritant CD	Allergic CD
Predisposition	Affects all who are exposed	Affects genetically predisposed
Mechanism	Non immunologic (direct tissue damage)	Immunologic (type IV hypersensitivity reaction)
Distribution	Restricted to the site of contact	Tends to disseminate beyond the site of contact
Symptoms	more burning	more itching

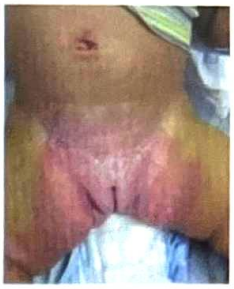
Examples of irritant CD :

Detergents Acids/alkalis

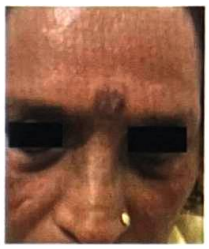


Diaper dermatitis :
Involves convex areas sparing the folds.



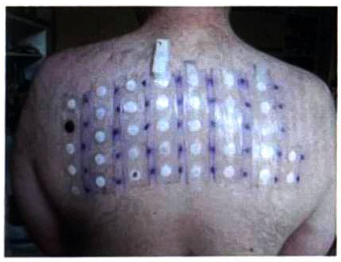


Examples of Allergic CD :
Hair dye : PPD (para phenylene diamine).
Metal : Nickel (most common).
Topical antibiotic : Neomycin.
Cement : Potassium dichromate.
Bindi : PTBP (para tertiary butyl phenol).



Patch test : Diagnosis of allergic CD.

Type of hypersensitivity reaction : Type 4.



Method : Allergens are applied over the patient's back & wait for type 4/ delayed hypersensitivity reaction.

Read at : 48 hours/2 days.
Best read at : 96 hours/4 days.

Airborne contact dermatitis

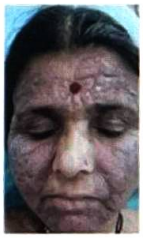
00:12:38

Disease : Type of contact dermatitis caused by release of substance into air which settles onto the exposed parts of the skin.

Plant : Parthenium hysterophorus (congress grass/communist plant).

Allergen : Sesquiterpene lactone (SQL).

Site : Neck, upper eyelids, face, V areas of chest.



Diagnosis : Photo patch test (patch test + UV radiation).

Disease : Phytophotodermatitis.
The plant chemicals on exposure to sunlight causes dermatitis.

Treatment :

Acute : Systemic steroids.

Long term : Azathioprine.

Prevention :

Try to avoid contact with the parthenium plant.

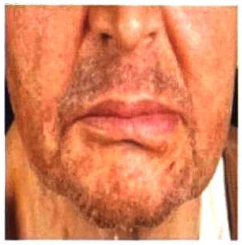
Endogenous eczema

00:16:26

Seborrheic dermatitis/dandruff.

Feature : Inflammatory response of seborrheic areas to Malassezia yeast.

Sites : Scalp, face, nasolabial folds, retro-auricular region.



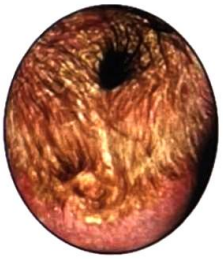
Greasy yellow scales

Scales : Greasy yellow.

Severe disease : Seen in Parkinson's disease, HIV infection.

Diiferential diagnosis with scalp psoriasis : Silvery white scales.

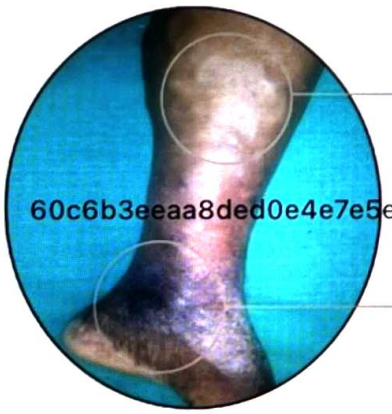
Infantile seborrheic dermatitis : Cradle cap.



Stasis eczema :

Synonym : Gravitational/varicose eczema.

Associated with chronic venous insufficiency.

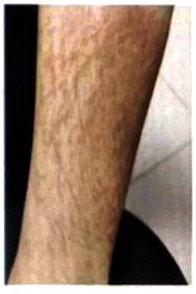


Asteatotic eczema :

Synonym : Eczema craquelé.

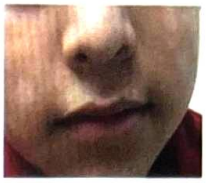
Age group : Older (decreased skin surface lipids).

Lesions : Itchy, cracks, dryness.



Active space

Pityriasis alba :



Pityriasis : Scaling, alba : White.

Profile : Children.

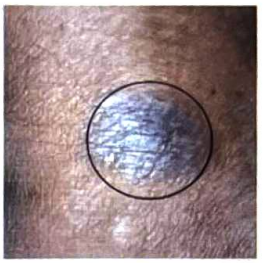
Lesions : Hypopigmented scaly macules (face).

Not a nutritional deficiency.

Differential diagnosis :

Indeterminate leprosy : Scaling is absent.

Nummular eczema :

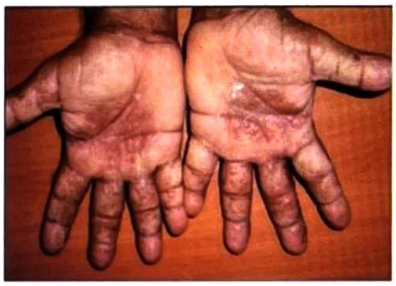


Synonym : Discoid eczema.

Lesions : Coin shaped lesions with well demarcated edges.

Differential diagnosis : Tinea.

Pompholyx :



Synonym : Dyshidrotic eczema.

Lesions : Intensely pruritic deep seated vesicles.

Appearance : Sago grain like.

Sites : Sides of digits.

Atopic dermatitis

00:24:38

Chronic, relapsing, inflammatory skin disease.

It is classified as an endogenous eczema triggered by exogenous factors.

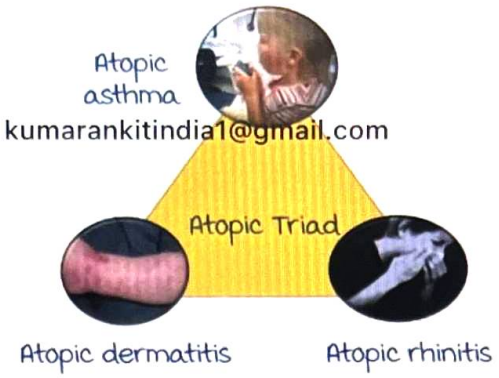
Atopy : Localized form of type I hypersensitivity reaction.

Atopic triad :

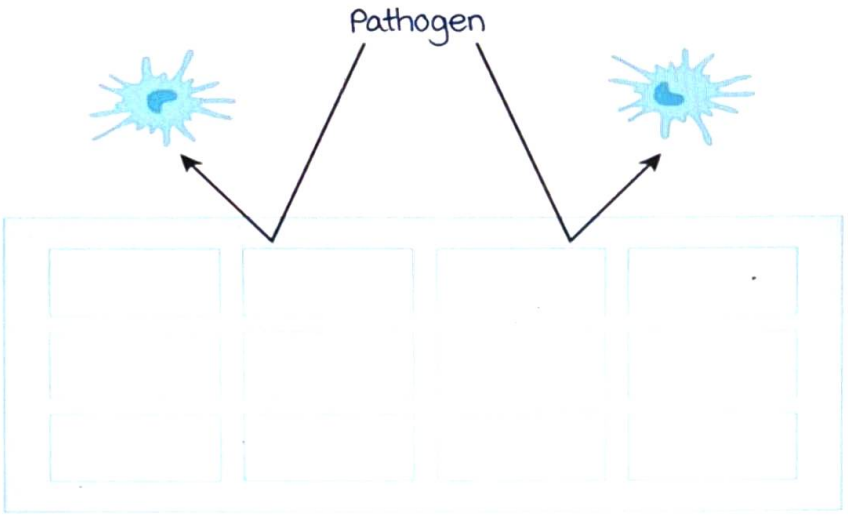
- Recurrent allergic rhinitis (upper respiratory tract).
- Atopic asthma/bronchial asthma (lower respiratory tract).
- Atopic dermatitis.

Pathogenesis :

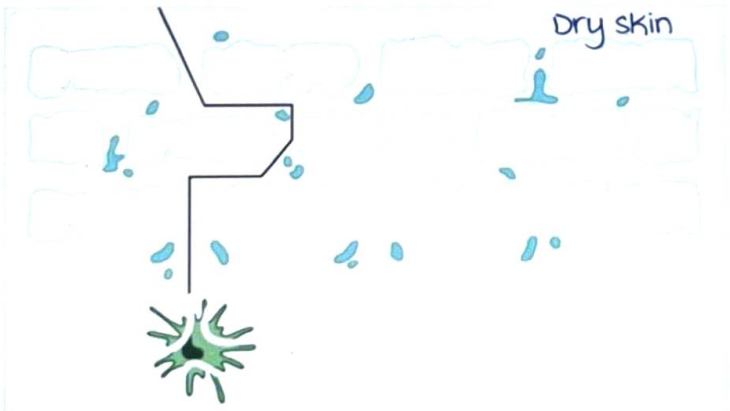
1. Defective barrier function of the skin (dry skin).
2. Filaggrin mutations (filament aggregating protein).
3. Increased IgE response to an allergens : Due to high levels of IL-4, IL-10, IL-13.



Atopic dermatitis – Pathogenesis



Atopic dermatitis – Pathogenesis



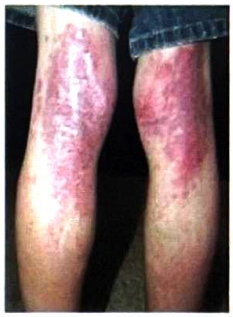
Atopic dermatitis : clinical phases 00:29:32

1. Infantile.
2. Childhood.
3. Adult.

Active space

Infantile phase:
Age group up to 2 years.
Lesions : Papulovesicular, exudative.
Sites : Face, scalp & extensor of extremities

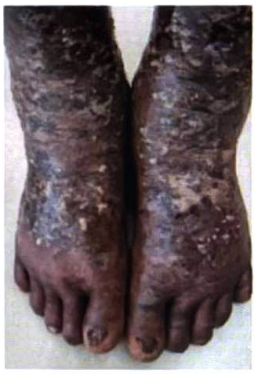
Childhood phase :
Age group : 2 years to puberty.
Lesions : Red, scaly excoriated papules, plaques, lichenification.
Sites : Flexor



Adult phase :
Age group : Beyond puberty.
Lesions : Lichenification.
Sites : Flexors.

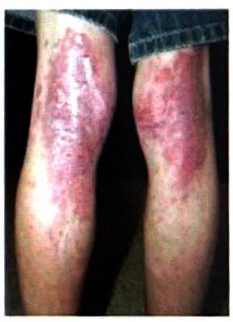
Complications :
1. Bacterial infection : Staphylococcus aureus.
Viral infection : Eczema herpeticum, disseminated cutaneous HSV I infection.

2. Erythroderma/exfoliative dermatitis : Involves > 90% BSA /body surface with erythema with or without scaling.

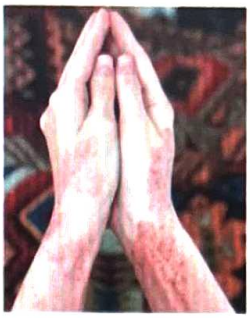


Secondary bacterial infection

Diagnosis of atopic dermatitis :
Key diagnostic criteria of atopic dermatitis.
Proposed By Hanifin and Rajka.
1. Pruritus : Hallmark (intermittent & intense, termed as itch that rashes).
2. Typical morphology & distribution with 3 phases.



3. Chronic or chronically relapsing dermatitis.
4. Personal/family history of atopy.

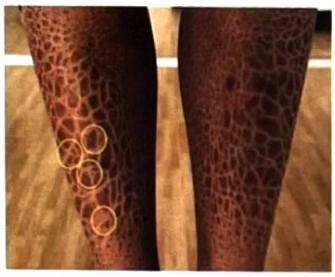


IOC : Clinical examination.

Other skin features :

- Xerosis : Dry skin.
- Ichthyosis : Fish like scales.
- White dermographism : On stroking the skin, white line appears due to vasoconstriction.
  Normal skin : On stroking, Wheal flare response.

Xerosis



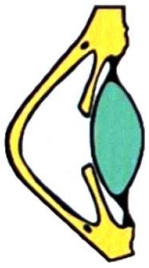
Ichthyosis

White dermographism

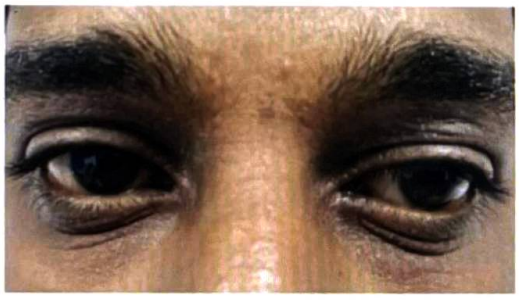
- Headlight sign : Facial eczema shows sparing of periorbital, perioral & perinasal area.

Ophthalmic manifestations :

- Conical cornea/Keratoconus.
- Shield cataract/anterior subcapsular cataract.
- Infraorbital fold of skin : Dennie-Morgan's fold.
- Lateral madarosis : Hertoghe's sign due to continuous rubbing.



Keratoconus



Dennie-morgan's fold.

Active space

Treatment of atopic dermatitis

00:42:06

Topical :

- Emollients (decreases dry skin).
- Topical steroids (anti-inflammatory).
  Ointment (if lesions are lichenified : thick plaques).
  Cream (for scaly & oozy acute & subacute eczema).

Systemic :

- Systemic steroids.
- Cyclosporine (calcineurin inhibitor).
  Side effect : Hypertension, nephrotoxicity.

New drugs :

- Crisaborole :
  Route : Topical.
  Mechanism of action : Phosphodiesterase 4 inhibitor.
  Indication : Mild to moderate disease.
- Dupilumab :
  Route : Subcutaneous.
  Mechanism of action : Binds to the α subunit of IL-4 receptor.
  Blocks signal transduction of IL-4, IL-13.
  Indication : Moderate to severe disease.

HISTAMINE RELATED DISEASES AND ANGIOEDEMA

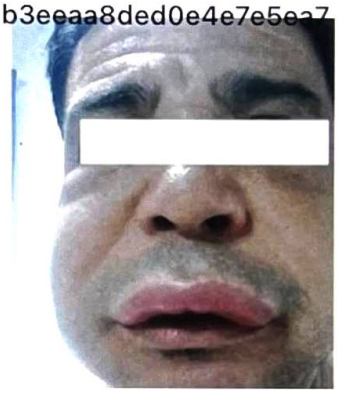
Histamine mediated emergencies 00:00:46

It is Type I hypersensitivity reactions where IgE mediated degranulation of mast cell occurs to release histamine.

It results in vascular reaction pattern that is manifested as :

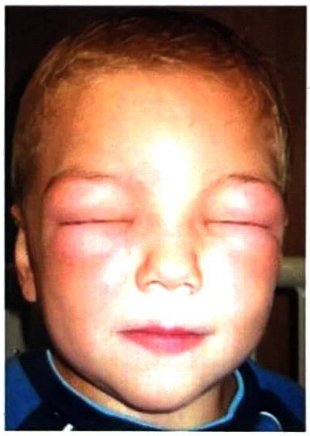
1. Urticaria : Dermal oedema with itchy wheals.

2. Angioedema : Subcutaneous/ submucosal oedema with non pitting oedema.
   Most common sites :
   Lips, eyelid (water accumulation in the loose connective tissue).



3. Anaphylaxis : It is acute with life threatening multisystem involvement.

- Skin : Urticaria, angioedema.
- Respiratory system : Wheeze due to bronchoconstriction.
- Cardiovascular system : Hypotension.
- Gastrointestinal system : Abdominal pain.



Urticaria

00:04:53

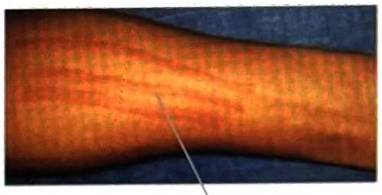
Itchy wheals (pruritic, transient plaque).

Characterised by central pallor and peripheral erythema.

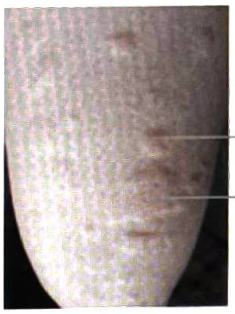
Shows dermographism :

Dermo : skin, graphy : to write.

In patients with urticaria, stroking the normal skin with blunt object results in an exaggerated wheal flare response.



Exaggerated wheal response



Peripheral erythema

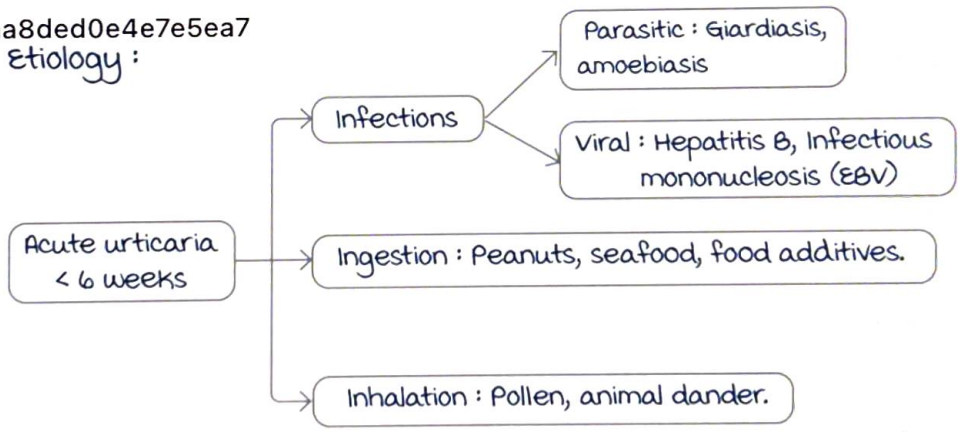
Central pallor

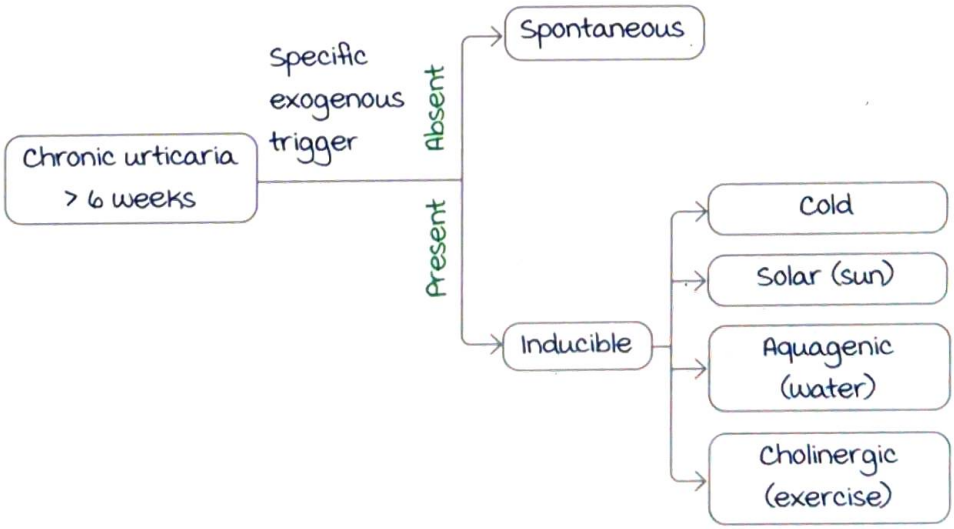
Classification (based on duration) :

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

60c6b3eeaa8ded0e4e7e5ea7

Etiology :





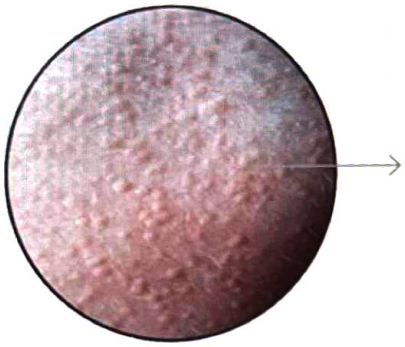
Active space

Cholinergic urticaria

00:11:18

Related to increased core body temperature.
Stimuli : Exercise /any form of physical exertion.

Exercise causes stimulation of eccrine sweat glands, supplied by the sympathetic cholinergic fibres. Stimulation of eccrine sweat glands leads to acetylcholine release causing pin point itchy wheals.



Chronic spontaneous urticaria

00:13:18

Duration > 6 weeks.
Managed by :

- 2nd generation antihistamines : Loratidine, Desloratidine, Cetrizine, Levocetrizine, Fexofenadine.
  Dose can be increased upto 4 times a day.
- Omalizumab : Monoclonal antibody against IgE.
- Cyclosporine : Calcineurin inhibitor.

Hereditary angioedema

00:15:26

- Autosomal dominant.
- Defective C 1 Esterase inhibitor (component of complement system).
- Features :
  1. Recurrent episodes of angioedema involving skin, oropharynx, respiratory tract, GIT on minor trauma.
  2. Not mediated by histamines, so there is absence of wheals.
  3. No response to antihistamines/steroids.

Active space

C I Esterase inhibitor inhibits :

a. Complement system.

b. Kallikrein system.

c. Factor 12.

Deficient and defective CI Esterase inhibitor causes activation of :

1. Complement system : C4 will be used up, so C4 level is reduced and is used as screening test for hereditary angioedema.
2. Kallikrein system : High molecular weight Kininogen is converted to bradykinin which acts on $\beta 2$ receptors causing increased vascular permeability.

Pharmacotherapy :

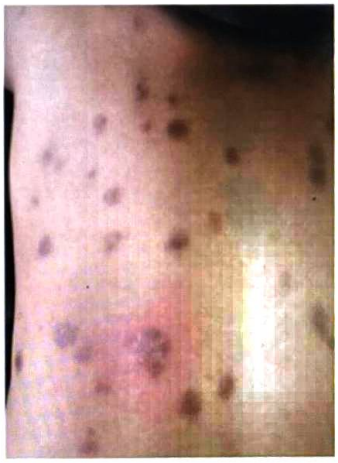
1. Increase C I Esterase inhibitor :
   - Recombinant C I Esterase inhibitor.
   - Danazol.
   - Fresh frozen plasma.
2. Specific target on :
   - Kallikrein inhibition : Ecallantide, Lanadelumab (newly accepted).
   - $\beta 2$ receptor inhibition : Icatibant.

Cutaneous mastocytosis

00:21:28

Also called urticaria pigmentosa.

- Characterized by multiple skin lesion with mast cells proliferation.
- Seen in children.
- Seen as multiple hyperpigmented macules distributed over the trunk.



Erythema + urticarial halo

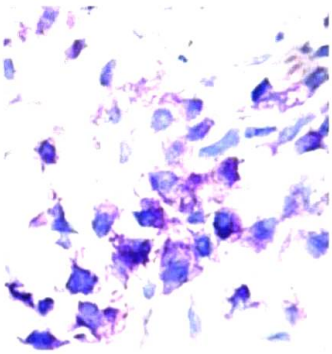
Dariers sign : Stroking the lesion with blunt object, mast cell degranulation occurs causing histamine release, forming erythema with urticarial halo.

Pseudo Darier sign :

- Differential diagnosis of cutaneous mastocytosis.
- Seen in congenital smooth muscle hamartoma : Characterised by hamartomatous proliferation of dermal smooth muscles.
  On stroking the lesion the muscle will contract causing transient induration and piloerection.

Histopathology :

- Toluidine blue stain shows metachromatic granules (purplish red coloured).
- metachromasia : The property by which cells take up another colour than the primary colour or same colour of stain.
- CD marker : CD 117



60c6b3eeaa8ded0e4e7e5ea7

Treatment :

- Antihistamines.
- Mast cell stabilizers : Ketotifen.

PIGMENTARY DISEASES

Melanocytes :

Located in stratum basale.

Embryologically derived from neural crest.

Made up of melanosomes.

Melanin pigment is produced (colour to skin).

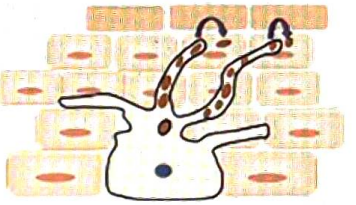
Markers : S 100, Melan-A, HMB 45.

Epidermal Melanin Unit (EMU)

00:01:32

The melanocytes at the stratum basale has dendritic processes.

The dendritic processes transfer melanosomes to keratinocytes.



Melanocyte

One melanocyte transfers melanosomes to 36 keratinocytes → Epidermal melanin unit.

1 melanocyte : 36 keratinocytes.

Function : Imparts uniform skin colour.

	Fair skin	Dark skin
Number of melanocytes	Same number	
Melanosomes	Smaller	Larger
Melanin	Pheomelanin (yellowish red)	Eumelanin (brown to black)

Disorders of hyperpigmentation

00:04:06

Melasma :

Acquired hyperpigmentary disease.

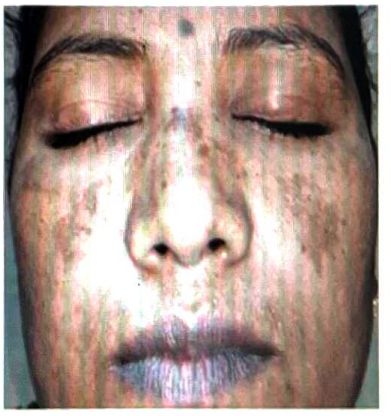
Etiology :

Increased sunlight exposure.

Thyroid dysfunction.

Drugs like OCPs, Phenytoin.

Pregnancy → Chloasma (mask of pregnancy).



Melasma

Active space

Lesion : Symmetric hyperpigmented brownish macules.

Sites : Nose, malar area and mandibular area.

Differential diagnosis : SLE.

Malar rash (of SLE) is red in colour.

Treatment :

Photoprotection → use of sunscreen.

Skin lightening agents.

Chemical peels.

Skin lightening agents : Inhibits tyrosinase.

Agents are : 60c6b3eeaa8ded0e4e7e5ea7

- Hydroquinone.
- Azelaic acid.
- Kojic acid.

Also used in the treatment of post inflammatory hyperpigmentation of acne.

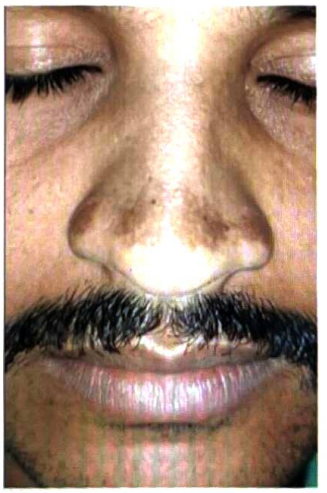
Chemical peels :

Controlled chemical exfoliation of the skin.

Agents used in melasma.

- Glycolic acid.
- Trichloroacetic acid 15–25%.

Chikungunya fever shows melasma like hyperpigmentation over the nose → Chik sign.

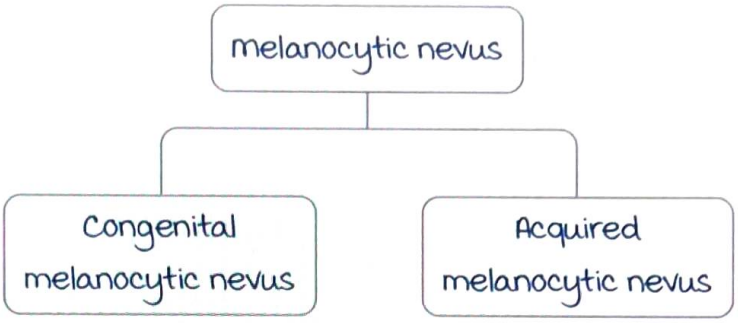


Chik sign

Melanocytic nevus :

Benign proliferation of a type of melanocyte, known as nevus cell. It is of two types :

Active space



Congenital melanocytic nevus :

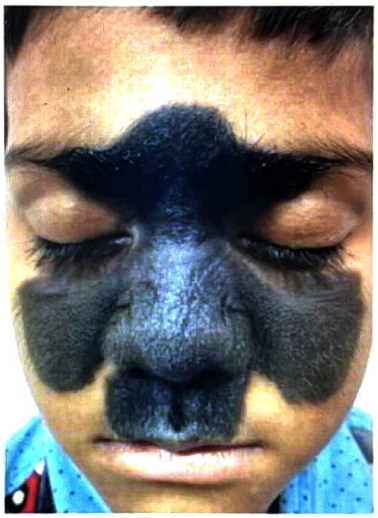
Onset : At birth.

Black coloured with well demarcated borders.

Surface is irregular, hypertrichotic.

>20 cm → Giant congenital melanocytic nevus.

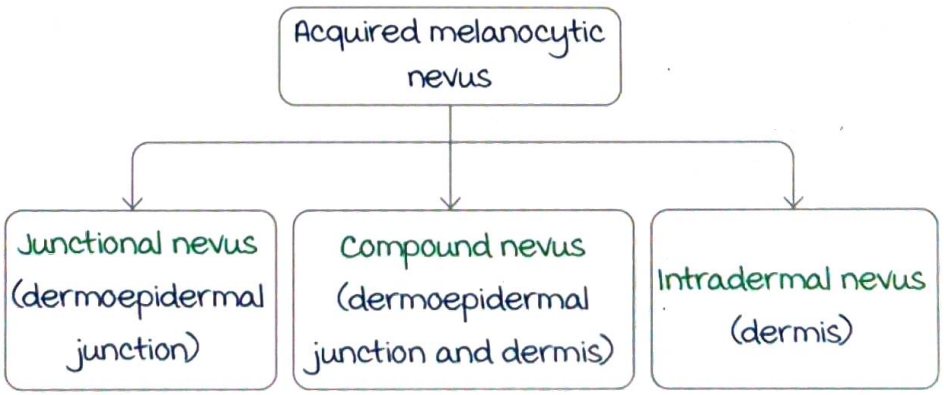
Risk of melanoma is 2- 5%.



Congenital melanocytic melanoma

Acquired melanocytic nevus :

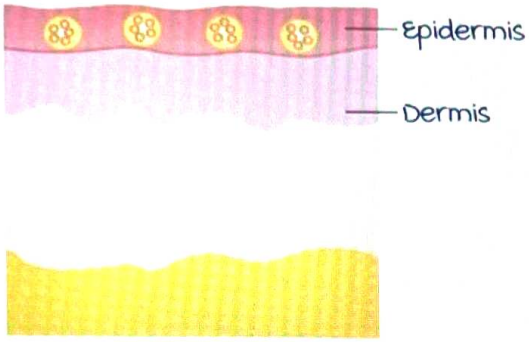
It is classified based on the position of the nevus cell cluster.



Junctional nevus :

Hyperpigmented macule.

Flat lesion.

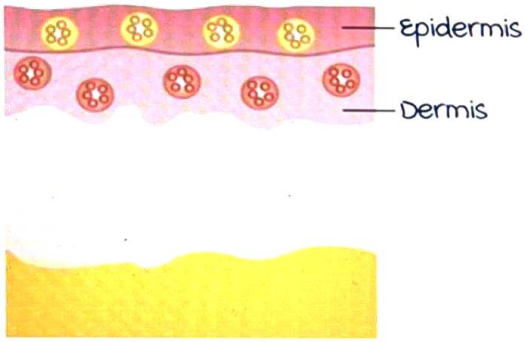


Junctional nevus

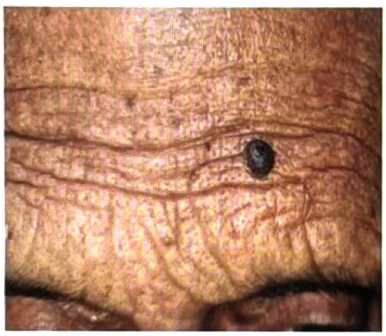
Compound nevus :

Hyperpigmented dome shaped nodule.

Dermal and dermoepidermal junction involvement.

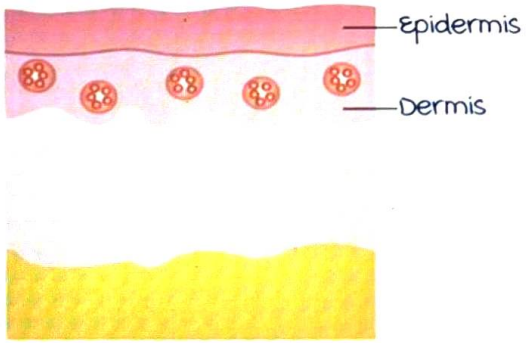


Compound nevus

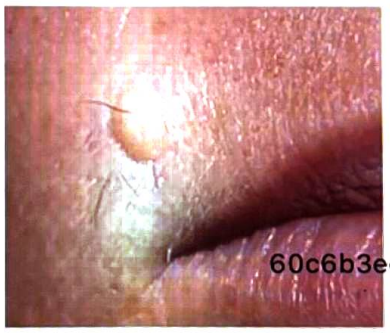


Intradermal nevus :

Dome shaped skin coloured nodule.



Acquired melanocytic nevus



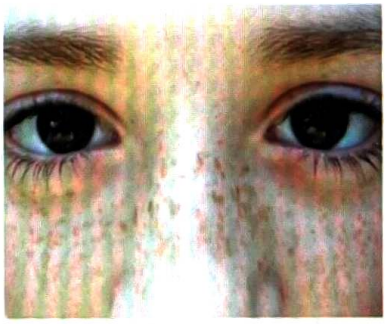
60c6b3eeaa8ded0e4e7e5ea7

Freckles :

Hyperpigmented macules seen in sun exposed areas.

Normal number of melanocytes.

Activity is increased.



Active space

Associated with xeroderma pigmentosum.

Also seen in fair skinned individuals when exposed to excessive sunlight.

Lentigines :

Hyperpigmented macules.

Melanocyte numbers are increased.

Activity of the melanocytes are normal.

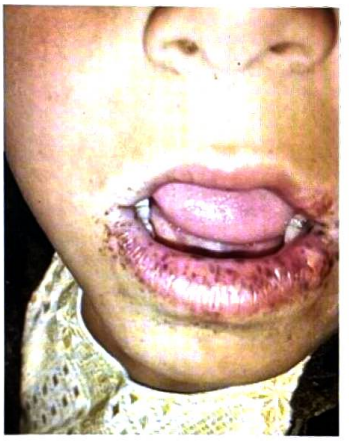
Peutz Jeghers syndrome :

Autosomal dominant disorder.

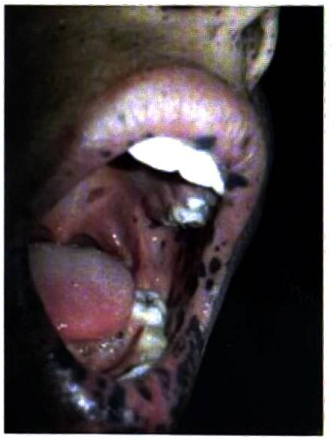
Characterised by mucosal lentigines and GI polyps.

Most common site for polyp is jejunum.

Most common type of polyp is hamartomatous.



Lentigines



Peutz Jeghers syndrome

Café au lait macules :

Hyperpigmented macule.

For the diagnosis of neurofibromatosis type 1 :

Number ≥ 6.

Size in prepubertal individuals > 5 mm.

Size in post-pubertal individuals > 15 mm.

Café au lait macules

In Mc.Cune Albright syndrome :

- café au lait macule.
- Fibrous dysplasia : Polyostotic type.
- Endocrine abnormality → Precocious puberty.

Active space

Becker's nevus :

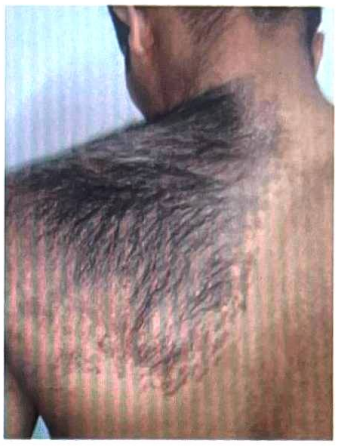
Acquired nevus, seen during puberty.

Unilateral.

Hyperpigmented.

Hypertrichotic.

Site : Chest and upper shoulder.



Becker's nevus

Dermal melanocytosis

00:18:32

Proliferation of melanocytes in the dermis → Blue colour.

Ceruloderma : Cerulo = blue, derma = skin.

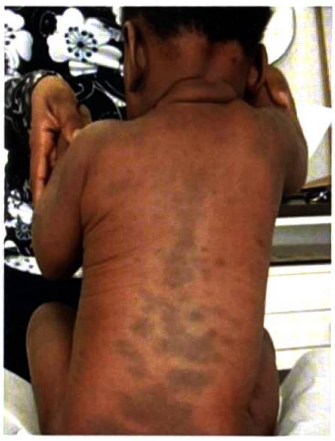
Blue to slate grey in colour (due to Tyndall effect).

Mongolian spot :

Usually seen in infants.

Colour is blue to slate grey.

Common site is lumbo-sacral region.



Mongolian spot

Nevus of Ota :

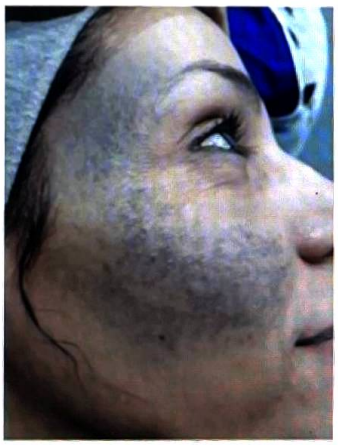
Unilateral.

Blue to slate grey colour.

Follows the 1st and 2nd division of trigeminal nerve (ophthalmic and maxillary divisions).

Site : Face.

2/3rd of patients have scleral involvement.



Nevus of Ota

Active space

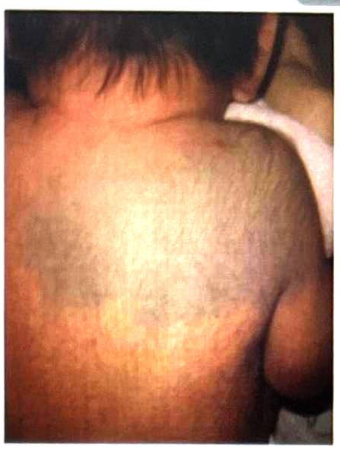
Nevus of Ito :

Unilateral condition.

Blue to slate grey in colour.

Follows the posterior supraclavicular and lateral brachial cutaneous nerves.

Site : Upper shoulder and back.



Nevus of Ito

Treatment of nevus of Ota/Ito :

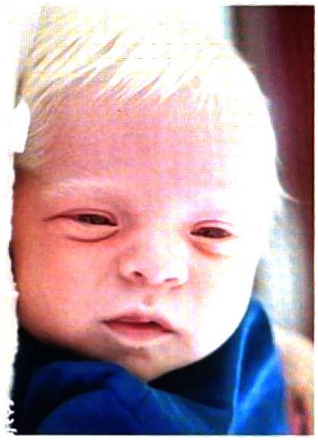
Q-switched NdYAG laser.

Disorders of hypo/depigmentation

00:23:01

Can be congenital or acquired.

Congenital disorders of hypo/depigmentation.



Oculocutaneous albinism

Albinism :

Defect in the enzyme tyrosinase.

60c6b3eeaa8ded0e4e7e5ea7

Two types :

- Ocular albinism : Involves only the eyes.
- Oculo- cutaneous albinism : Involves eyes, skin and hair.

Chediak Higashi syndrome :

Autosomal recessive condition.

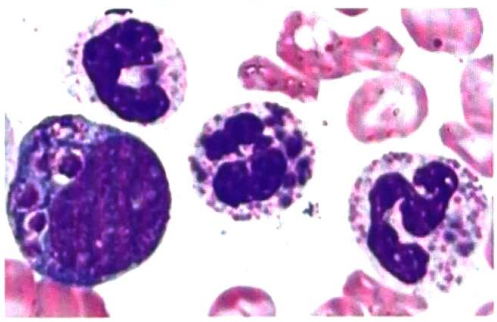
Defective phagolysosome fusion.

Presents with :

Oculocutaneous albinism.

Recurrent infections.

Peripheral smear shows giant granules in the neutrophil.



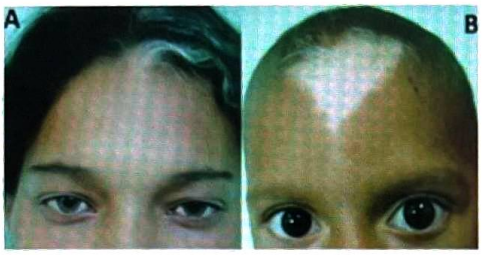
Giant granules in neutrophils

Piebaldism :

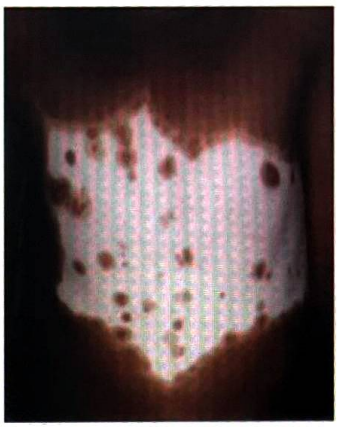
Autosomal disorder.

Defect is neural crest dysfunction.

White forelock → Characteristic lesion.
White coloured bunch of hair over the forehead.



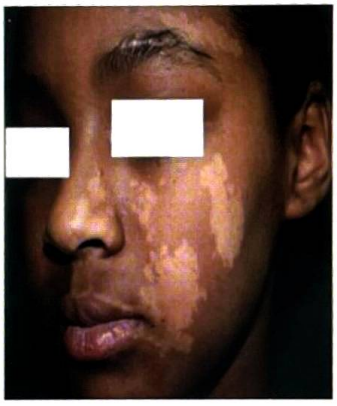
Piebaldism



Islands of normal skin in the depigmented area

In the trunk, within the white patch there are islands of normal skin.

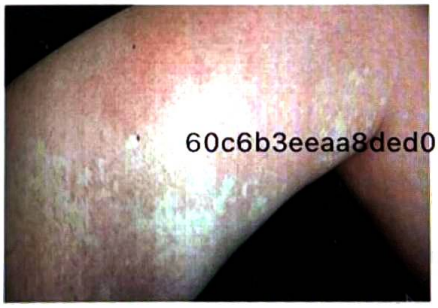
Nevus depigmentosus :
Defect in the transfer of melanosomes to the keratinocytes.
Onset is at birth.
Presents as depigmented macule with feathery margins.



Nevus depigmentosus

Nevus anemicus :
Congenital white patch of vasoconstriction.

Pale looking → Decreased blood supply.

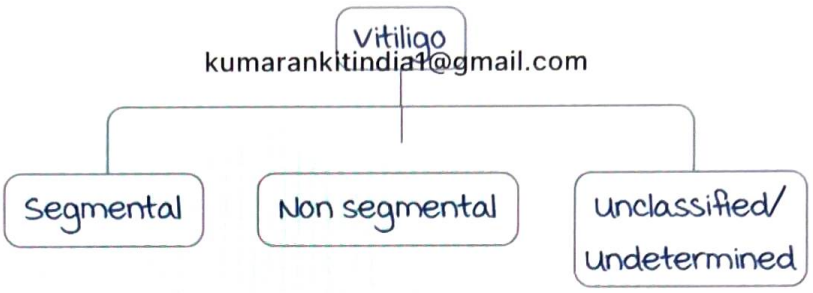


Nevus anemicus

Acquired disorders of hypo/depigmentation 00:29:43

Vitiligo :
Acquired chronic disease of depigmentation.
Most common theory explaining the loss of melanocyte :
Autoimmune theory : Auto-antibodies destroy the melanocytes → Melanocyte absent from the lesion.

Association : Thyroid dysfunction.



Segmental vitiligo :

Starts at early childhood.

Course : Initially rapid, stabilizes later on.

Skin lesions :

- Unilateral.
- Segmental.
- Does not cross the midline.

Leukotrichia → White hair.

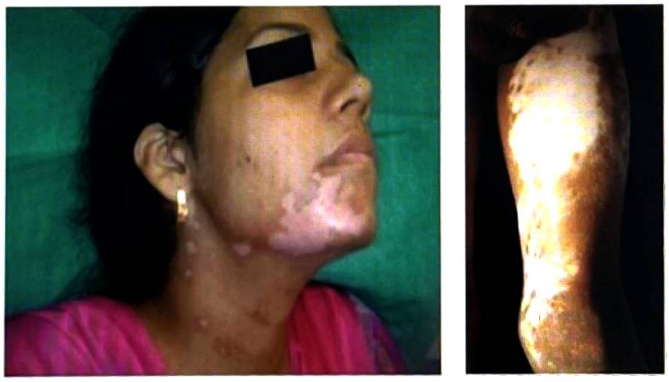
Involvement of melanocytes of hair.

Common feature in segmental vitiligo.

Seen early in the disease.

Treatment :

Surgical intervention : melanocyte transplanting, skin grafting.



Segmental vitiligo

Non-segmental vitiligo :

Acrofacial vitiligo → Face, head and distal extremities.

Mucosal vitiligo → Involves > 1 site.

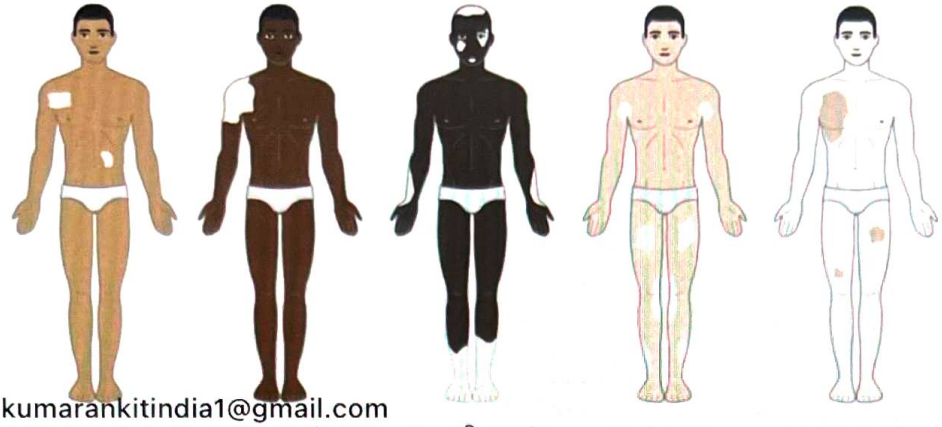
Vitiligo vulgaris (most common type) : Symmetrically distributed depigmented macular lesions.

Universal vitiligo → >80- 90% body surface area depigmented.

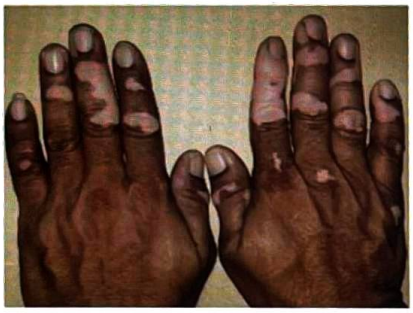
Unclassified/ undetermined vitiligo :

Focal vitiligo → Small, isolated depigmented lesions.

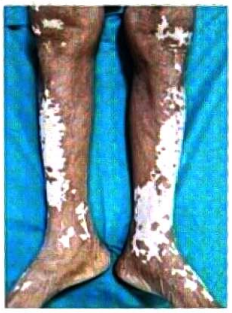
Types of vitiligo



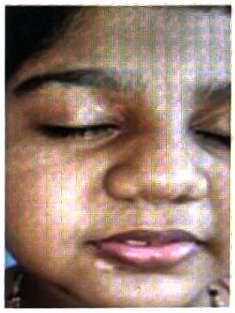
Focal Segmental Acrofacial Vulgaris Universalis



Acral vitiligo



Vitiligo vulgaris



Focal vitiligo

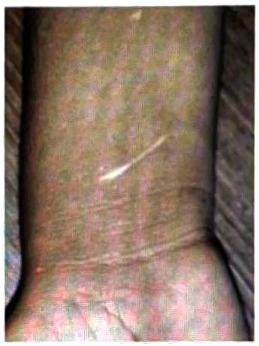
Clinical features :

Depigmented chalky white macules → melanocytes are absent.

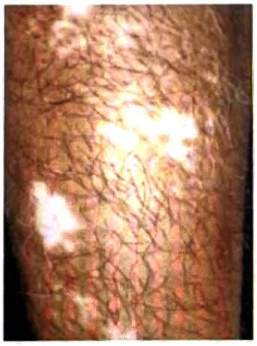
Koebner's phenomenon :

True Koebner's phenomenon : New lesions appear at the site of trauma.

Hair follicle melanocytes are involved → Leukotrichia



Koebner's phenomenon



Leukotrichia

Treatment :

Based on the body surface area (BSA) involved.

<20% BSA involved :

Topical steroids.

Active space

Topical tacrolimus → Calcineurin inhibitor.

>20% BSA involved :

Systemic steroids (halt the progress of disease).

Azathioprine.

NB- UVB (narrow band UV- B radiation) at 311 ± 2 nm wavelength.

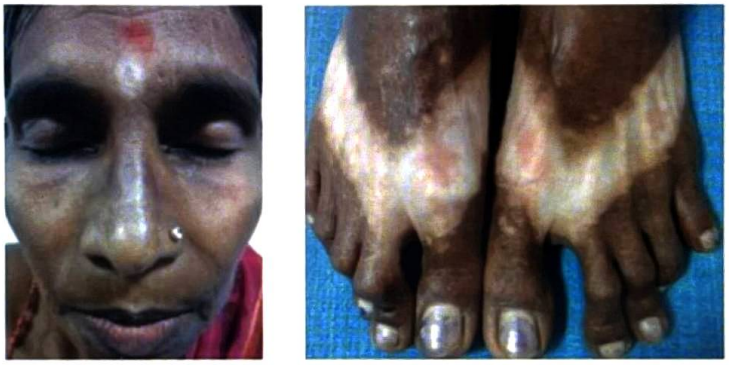
Chemical leukoderma :

Chemical induced melanocyte destruction. (Acquired disorder).

Bindi dermatitis → Para tertiary butyl phenol (PTBP) destroys the melanocytes.

Rubber footwear to be avoided in eczema prone patients.

Contains Mono Benzyl Ether of Hydroquinone (MBEH) destroys melanocytes.



Chemical leukoderma

CUTANEOUS DRUG REACTIONS

Fixed drug eruptions

Mechanism : Characterised by localised Type 4 (delayed) hypersensitivity response.

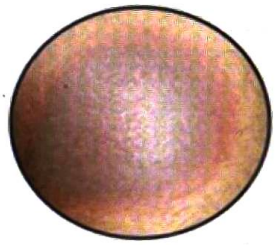
Drugs implicated :

- NSAIDs.
- Cotrimoxazole.
- Tetracyclines.

"Fixed" because it always recurs at a fixed location.

Sites : Mucocutaneous junctions (lips, glans penis), limbs.

Morphology of the lesion :



Well demarcated, circular, dusky red plaque with erythema around the margins.

Timeline :

- 1st time : 7 to 10 days after the intake of the drug.
- Re-intake : 30 minutes to 8 hours after the intake of the drug.

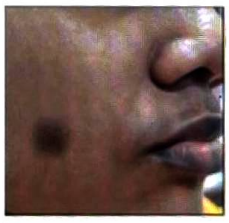
The location of the drug reactions is fixed because of the activation of the memory T cells.

1st intake of the drug → memory is formed in the lesional skin → reintake of the same drug → memory T cells reactivated at the site of lesion → lesions occur at the same site.

Special feature : Lesions always heal with hyperpigmentation

Treatment :

- Stop the offending drug.
- If extensive disease : Steroids (topical/systemic).



Fixed drug eruption

Erythema multiforme

00:04:36

Definition : Acute, immune mediated, self limiting, cytotoxic dermatitis.

Aetiology :

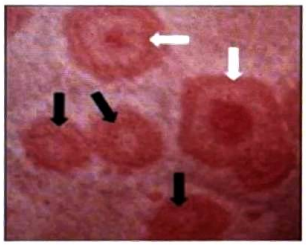
- Infections : Herpes simplex virus I (most common), Mycoplasma pneumonia.
- Drugs : Antimalarials, NSAIDs, Sulphonamides.
- Connective tissue disorder : Systemic Lupus Erythematosus (SLE).
- Neoplasia : Leukaemia, internal malignancies.

Types of Erythema multiforme (EM) :

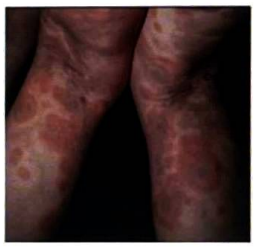
	EM major	EM minor
Illness severity	Severe	Mild
Mucosal involvement	Involved.	Not involved.

Special lesion : Target lesion with 3 zones.

1. Central zone : Dusky hue.
2. Intermediate zone : Pale oedema.
3. Peripheral zone : Erythema.



Target lesions



Distibution

Distribution : Distal extremities (like palms, soles).

Treatment :

- Treat the underlying cause
- Short course of systemic steroids if necessary.

SJS - TEN

00:09:23

SJS - TEN : Stevens Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN).

Definition : Acute, life threatening, severe, mucocutaneous adverse drug reaction.

Drugs implicated : >90 %, the aetiology for SJS-TEN is drugs.

- NSAIDs (Oxicam groups).
- Antiepileptic drugs : Phenytoin, Carbamazepine, Lamotrigine.
- Sulphonamides.

- Allopurinol.
- Anti retrovirals : Abacavir, Nevirapine.

Carbamazepine and gene testing :

Patients with HLA B 1502 positive have increased chance of developing SJS-TEN. So, HLA B 1502 gene testing is recommended before giving Carbamazepine.

Pathogenesis :

- Event : Necrosis of Keratinocytes.
- Mediators :
  1. CD8 T cells and NK cells → Granulysin (cytolytic protein) → Destruction of Keratinocytes.
  2. IL-15 → Promotion and maintenance of CD8 T cells and NK cells.
  3. Fas ligand binds to Fas receptor (death receptor) → mediates necrosis of the Keratinocytes.

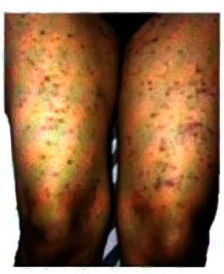
Assessment of a patient with SJS-TEN : Based on Body Surface Area (BSA).

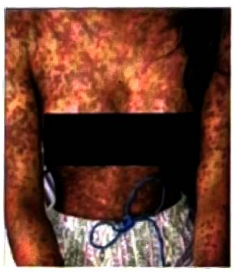
- <10% → SJS.
- 10% to 30% → SJS-TEN overlap.
- >30% → TEN.

Timeline : 1 to 4 weeks of intake of the drug.

Cutaneous examination :

- Skin lesions : Atypical target lesion (only 2 zones).
- Distribution : Proximal extremities and trunk.
- Progression : Diffuse erythema → Bullae → Sheet like epidermal peeling.


Atypical target lesion


Diffuse erythema



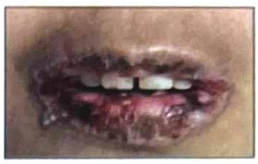
Sheet like epidermal peeling

Clinical sign : Pseudo Nikolsky sign positive (separation of upper layers of epidermis from lower layers on applying tangential pressure on the skin).

Mucosa : Painful erosions covered by haemorrhagic crusts, which bleed on peeling.

Treatment :

- Identify & stop the offending drug.
- Specific management : Cyclosporine.
- IV Immunoglobulins.
- Supportive management.
    1. Treating secondary infections.
    2. Stabilization of the patient.
    3. Adequate nutrition.



Hemorrhagic crusts

Cutaneous manifestations of chemotherapy drugs 00:21:18

Bleomycin induced pigmentation :

Pattern : Flagellate dermatitis (Whip like pattern).

Lesions : Diffuse, pruritic, flagellate lesions.

Distribution : Extremities.

Healing : Heals with persistent hyperpigmentation.

Flagellate dermatitis

Mechanism of flagellate dermatitis :

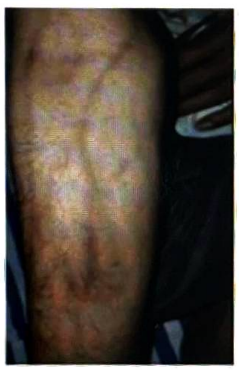
Hydrolase is the enzyme involved in the metabolism of Bleomycin.

Lack of this inactivating enzyme (Hydrolase) causes increased accumulation of Bleomycin which results in

- Skin : Flagellate dermatitis.
- Lungs : Pulmonary fibrosis.

Serpentine supravenous hyperpigmentation :

Pattern : Linear hyperpigmentation over the skin along the course of the vein.



Drugs :

- 5 Fluorouracil (5-FU).
- Cyclophosphamide.
- Docetaxel.
- Doxorubicin.

Adverse effect of IV chemotherapy agents.

Mechanism for lesion formation along the course of the vein :

- Endothelial injury.
- Subclinical thrombophlebitis.

SKIN IN IMMUNOLOGY AND RHEUMATOLOGY

Autoimmune connective tissue disorders :

1. Lupus erythematosus.
2. Dermatomyositis.
3. Scleroderma.

Vasculitis :

1. Henoch Schonlein Purpura (HSP).
2. Behcet's disease.

Skin in :

1. Reactive arthritis.
2. Rheumatoid arthritis.
3. Acute rheumatic fever.

Lupus Erythematosus (LE) 00:00:32

6006[illegible] disease characterized by non-organ specific auto antibodies.

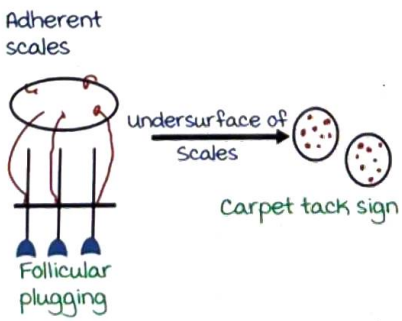
Target : Cellular nuclear antigen.

Classification :

1. Purely cutaneous form : Discoid LE.
2. mainly cutaneous, limited systemic : Subacute cutaneous LE (SCLE).
3. Widespread systemic : Systemic LE (SLE).

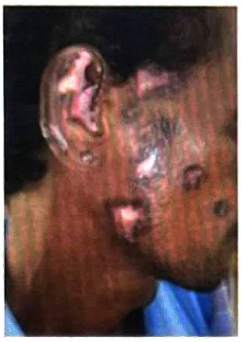
Discoid LE/CCLE :

- Chronic Cutaneous LE.
- Important sites : Face, scalp, ears.
- Morphology of the lesion :
  Discoid plaque : Coin shaped lesion.
  Central atrophy and scarring.
  Peripheral hyperpigmentation.
- Scales : Adherent scales (tightly attached to the hair

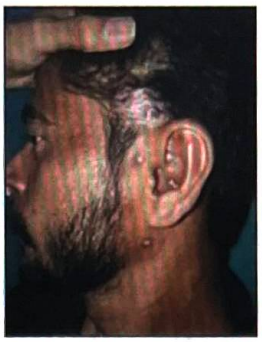


follicle → Blocks follicles → Follicular plugging).

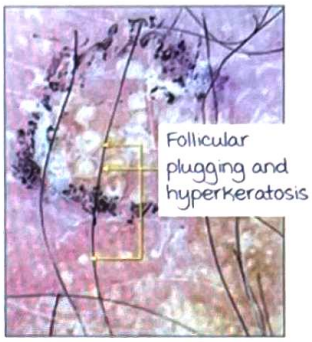
- Remove the scale → Follicular plugs visible on the undersurface → Carpet tack sign.
- Alopecia : Scarring alopecia.



Discoid lesions



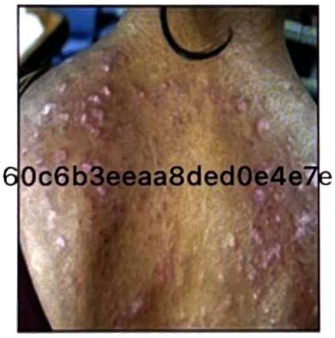
Scarring alopecia



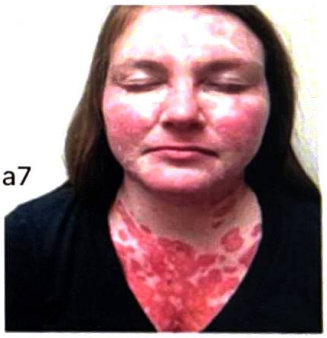
Carpet tack sign

Subacute cutaneous LE :

- Most photosensitive variant.
- Non scarring rash.
- Associated with anti-Ro and anti-La antibodies.
- Lesions :
  1. Annular and polycyclic lesions (individual rings merge together).
  2. Papulosquamous/psoriasiform lesions.



Papulosquamous lesions



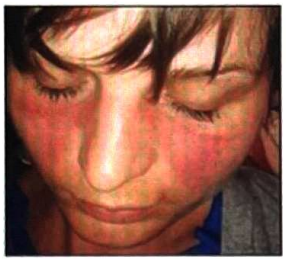
Annular and polycyclic lesions

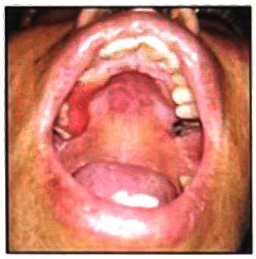
Systemic LE :

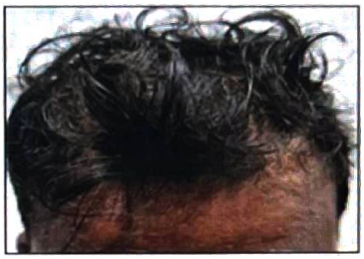
1. Malar rash :
   - Acute cutaneous LE.
   - Erythema over malar area and bridge of nose.
   - Nasolabial folds spared.
   - Photosensitive rash.
   - Differential diagnosis : Rosacea (papules + pustules seen which are not present in LE).

2. Discoid rash.
3. Oral/nasal ulcers :
   - Oral ulcers → Painless, seen over palate.
4. Alopecia : Non-scarring (lupus alopecia) or scarring type (produced by discoid rash).

   Lupus hair :
   - Site : Frontal region.
   - Texture : Unruly, fragile, short hair.
   - Associated with disease flare.


Malar rash


Oral ulcers


Lupus hair

Dermatomyositis

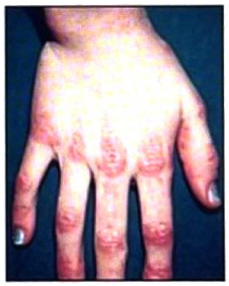
00:11:57

Autoimmune connective tissue disease.

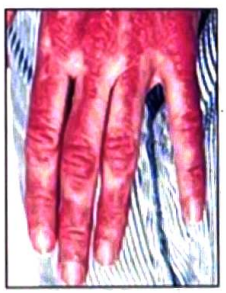
Involves skin, skeletal muscles (proximal myopathy).

Skin lesions :

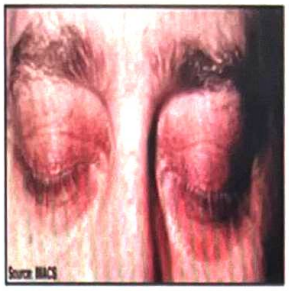
1. Gottron's papules : Violaceous flat top papules affecting metacarpophalangeal and interphalangeal joints.


Gottron's papules

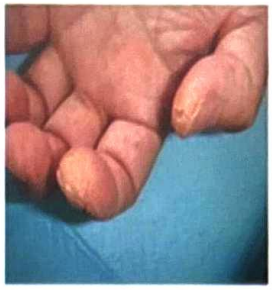
2. Gottron's sign : Violaceous macular erythema seen on metacarpophalangeal and interphalangeal joints.
3. Heliotrope rash : Violaceous macular edema/erythema seen in the periorbital region, especially affecting the upper eye lid.
   - Helios : Sun, tropism : Affinity.
   - Heliotrope flower → violet colored flower that grows facing towards sun.


Gottron's sign

4. Mechanic's hand.
   - Hyperkeratotic fissuring, involving tips and sides of digits.
   - Associated with anti-Jo-1 antibodies.



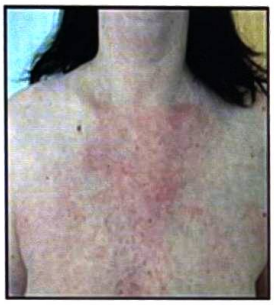
Heliotrope rash



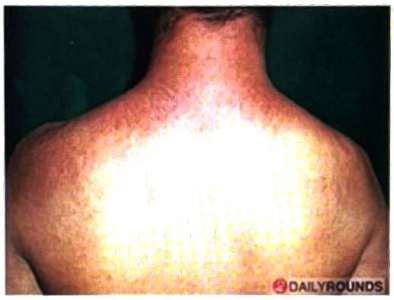
mechanic's hand

Named signs of dermatomyositis 00:17:32

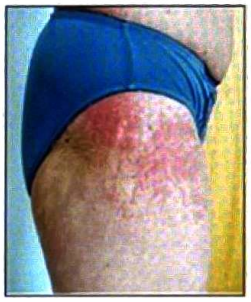
- Anterior chest : V sign.
- Upper back, shoulder : Shawl sign.
- Lateral aspect of thigh : Holster sign.



V sign



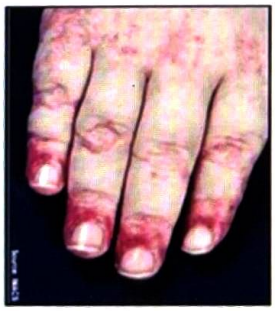
Shawl sign

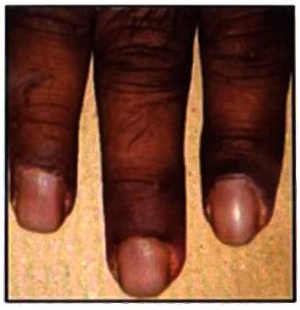


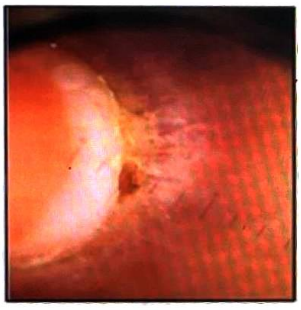
Holster sign

Nail features :

1. Periungual erythema : Redness around nails.
2. Samitz sign : Ragged cuticle.
3. Nail fold capillary telangiectasias.





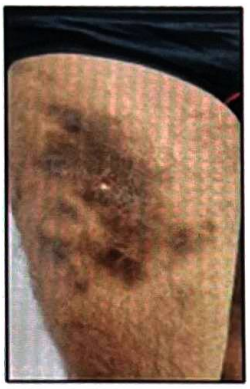


Scleroderma 00:20:00

- Sclero : Thick.
- Derma : Skin.

1. Localised cutaneous form : morphea.
   - Limited to skin.
   - Early : Violaceous indurated plaque (active stage).
     Later : Ivory white lesions (healed).

   - En coup de sabre alopecia :
     Linear pattern of morphea affecting scalp : Scarring alopecia.


Early morphea


En coup de sabre alopecia

Resembles a sabre sword.

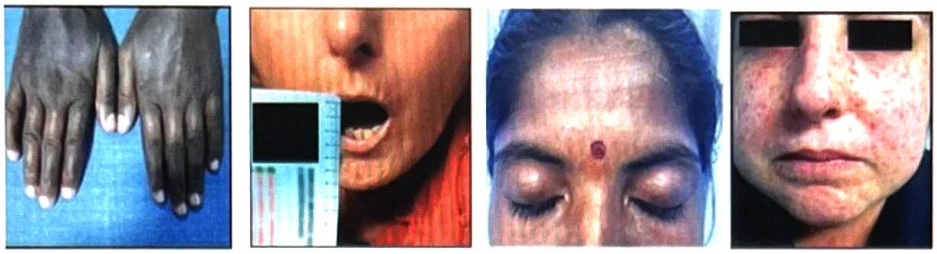
2. Systemic sclerosis :
   1. Limited cutaneous systemic sclerosis : Skin thickening restricted to distal to the elbow, knee and face.
      CREST syndrome :
      - Calcinosis cutis
      - Raynaud's phenomenon.
      - Esophageal dysmotility.
      - Sclerodactyly.
      - Telangiectasia.
   2. Diffuse cutaneous systemic sclerosis : Distal + areas proximal to elbow, knee and trunk involved.

Pathogenesis :
- Blood vessels → Vascular dysfunction.
- Immune dysregulation.
- Fibroblasts ⟶ TGF β ⟶ fibrosis.

Skin features :
1. Hide bound/taut skin : Unable to pinch skin (fibrosis).
2. Wrinkling reduces + mask like face
3. Perioral radiating furrows.

Active space

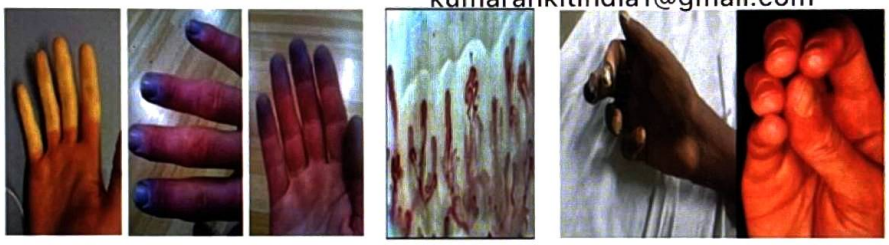
4. Microstomia.
5. Mat like telangiectasias.



Taut skin | Perioral radiating furrows + microstomia | Wrinkling reduces + mask like face | Mat like telangiectasias

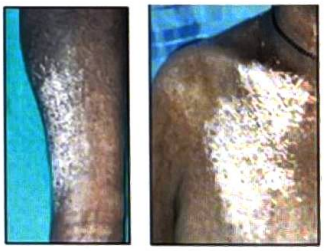
Extremities :

1. Raynaud's phenomenon :
   - Episodic vasoconstriction secondary to cold/emotional stimuli.
   - 3 phases : Pallor → Cyanosis → Rubor.
2. Nail fold capillary telangiectasias : Dilated blood vessels in nail bed.
3. Digital ulcers, gangrene → Digital scars.



Raynaud's phenomenon | Telangiectasias | Digital ulcers and pitted scars

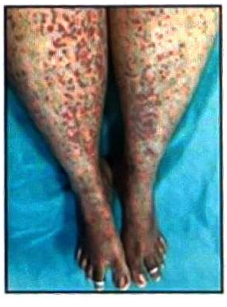
Pigmentation : Depigmentation (white in color) + Perifollicular pigment retention (brown in color) → Salt and pepper pigmentation.



Salt and pepper pigmentation

Henoch Schonlein Purpura(HSP) 00:32:00

IgA vasculitis.
Small vessel vasculitis.
Palpable purpura : Symmetrically distributed over lower extremities.
Platelet count : Normal (non thrombocytopenic purpura).



Palpable purpura

Other features :

- Arthralgia.
- Abdominal pain.
- Hematuria.

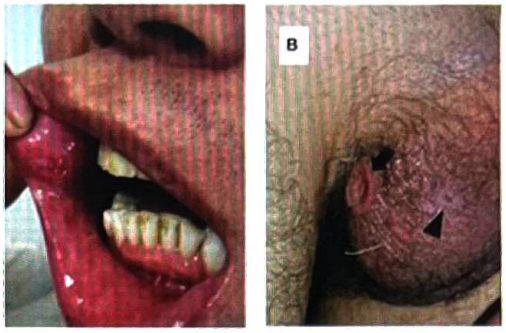
Behcet's disease :

Multisystem disease.

Variable vessel vasculitis.

Mucocutaneous lesions :

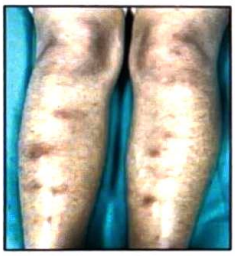
- Recurrent oral ulcers (at least 3 episodes per year & heal without scarring).
- Recurrent genital ulcers that heal with scarring.

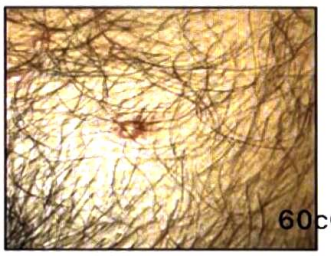


Well demarcated ulcer with necrotic base

Patterns of lesions :

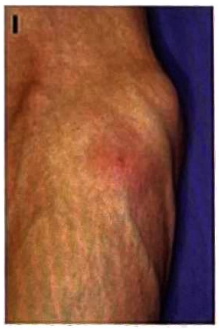
- Erythema nodosum like lesions.
- Papulopustular lesions.
- Acneiform lesions.


Erythema nodosum like lesions


Papulopustular lesions

Eye : Anterior/posterior uveitis.

Pathergy test : Intradermal saline injection (inject or prick with needle) → Wait for 48 hours → Hypersensitive reaction → Papulopustular lesions → Positive for Behcets disease.



Pathergy test

Reactive arthritis :

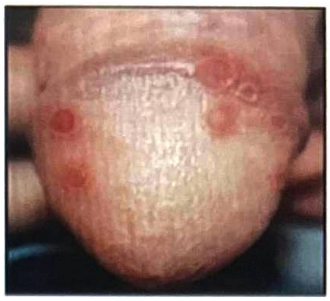
Arthritis secondary to distant focus of infection.

GI (enterally acquired) : Salmonella, Shigella, Campylobacter.

Sexually acquired (SARA) : Chlamydia trachomatis.

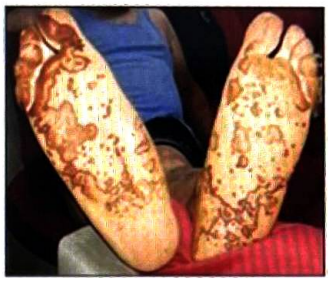
Skin lesions :

1. Circinate balanitis :
   - Well demarcated patches.
   - Painless.
   - Circular erosions.
   - Site : Glans penis.
   - Characteristic inflammation of the glans penis in reactive arthritis.



Circinate balanitis

2. Keratoderma blenorrhagicum :
   - Keratoderma : Thickening of skin.
   - Blenorrhagicum : Mucous discharge flow (Bleno : Mucus, Rhoea : Flow)
   - Hyperkeratotic thickened papules and plaques.
   - Sexual route of acquiring.
   - Site : Palms and soles.



Keratoderma blenorrhagicum

Rheumatoid arthritis

00:43:10

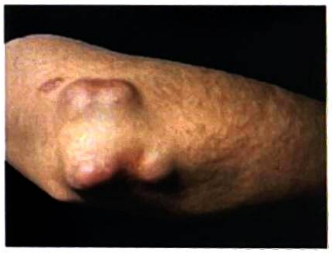
Rheumatoid nodules :

Painless, subcutaneous nodules.

Site : Pressure sites.

Indicates :

1. Disease severity.
2. Rapid progress of joint destruction. RA factor positive mostly.



Rheumatoid nodules

Acute rheumatic fever :

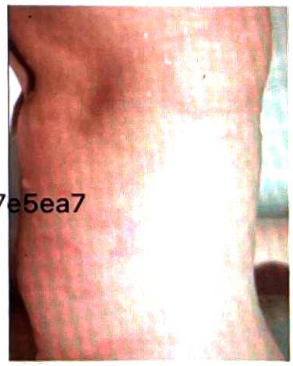
1) Subcutaneous nodules :

Painless, smaller, short-lived nodules.

Associated with carditis.

2) Erythema marginatum :

- Non-pruritic pink macular erythema.
- Central clearing.
- Periphery : Outward extension.
- Trunk involved, face is spared.



SKIN AND SYSTEMS

Acanthosis nigricans

00:00:18

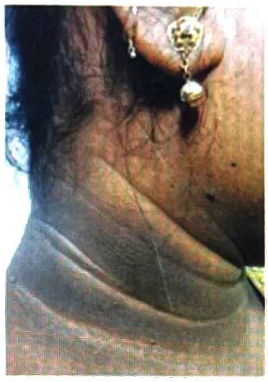
Morphology : Hyperpigmented velvety plaques.

Sites : Neck, axilla.

Most Common cause : Obesity.

Mechanism : In obesity → Increased insulin like growth factor → Stimulate epidermal Keratinocytes.

Other causes : Diabetes, drugs and GI adenocarcinoma (rare).



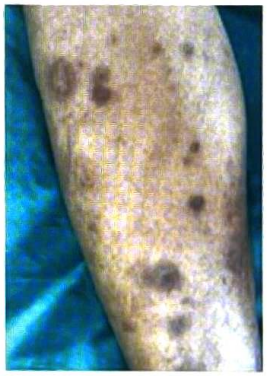
60c6b3eeaa8ded0e4e7e5ea7

Diabetes and skin

00:01:55

1. Diabetic dermopathy :
   - Lesion : Hyperpigmented atrophic macules.
   - Site : Lower extremities.

Significance : Marker for complications like diabetic nephropathy/neuropathy and retinopathy.



Diabetic dermopathy

2. Necrobiotic disorders :

Necrobiosis means degeneration of collagen.

2 disorders :

- Granuloma annulare.
- Necrobiosis lipoidica diabeticorum.

Granuloma annulare :

Lesion :

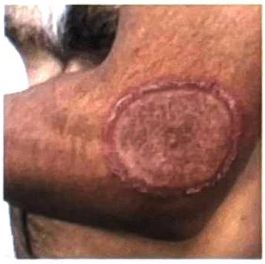
- Asymptomatic.
- Annular arrangement of papules.
- Central clearing.

Site : Dorsum of the extremities.

Differential diagnosis : Tinea corporis.

Granuloma annulare	Tinea corporis
Asymptomatic	Itchy
No scaling	Scaling present

Granuloma annulare



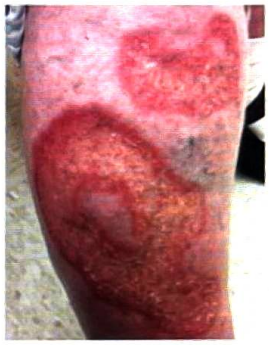
Tinea corporis

- Necrobiosis lipoidica diabeticorum :

Morphology :

- Waxy yellow.
- Atrophic plaque.
- Surface : Telangiectasia.

Site : Anterior aspect of leg.



Necrobiosis lipoidica diabeticorum

Sarcoidosis

00:06:23

Sarcoidosis :

- Acute : Lofgren's syndrome.
- Chronic : Lupus Pernio.

Lofgren syndrome :

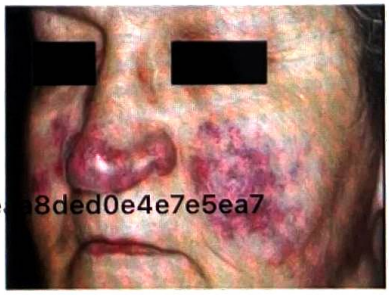
- Lung : B/L hilar lymphadenopathy.
- Skin lesion : Erythema nodosum.
- Other features : Fever, arthralgia.

Lupus pernio :

Classification : Chronic, specific lesion of sarcoidosis.

Lesion : Reddish-violaceous infiltrated plaque.

Site : Centro facial region.



Lupus pernio

Association :

- Pulmonary fibrosis.
- Bone cysts (phalanges of fingers).
- Sarcoidosis of upper respiratory tract.

Primary systemic amyloidosis :

Association : Plasma cell dyscrasias.

Deposition :

AL type of amyloid deposition

↓

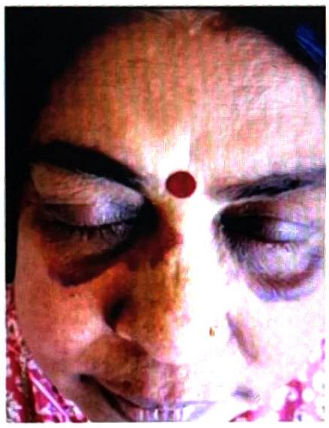
Blood vessels (Periorbital region)

↓

Trauma/pinch

↓

Pinch purpura



Primary systemic amyloidosis

Pretibial myxoedema :

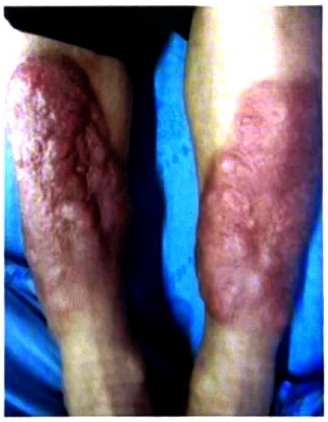
Synonym : Thyroid dermopathy.

Lesion : Non pitting edema.

Site : Anterior aspect of legs.

Association : Graves disease.

Mechanism : Deposition of glycosamino glycans (GAGs).



Pretibial myxoedema

Xanthelasma palpebrarum : Xanthos (yellow).

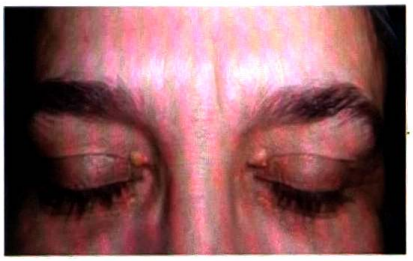
Skin Lesions : Symmetric, yellow papules and plaques.

Site : Periorbital.

Associations :

Hyperlipidemia (Type 2/4).

Cholestasis.



Xanthelasma Palpebrarum

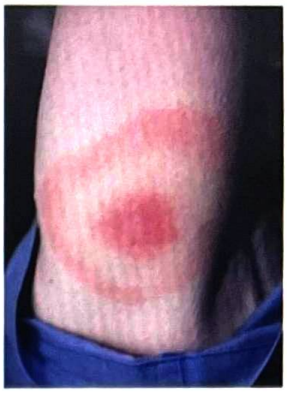
Lyme disease :

Etiology : Borrelia burgdorferi.

Vector : Ixodes tick.

Skin rash :

- Around tick bite.
- Red expanding rash.
- Erythema Chronicum Migrans (ECM).



Target appearance of ECM

Erythema multiforme	Lyme disease
multiple target lesions.	Single target lesion.
Site : Distal extremities (palm/sole).	Depends on site of tick bite.

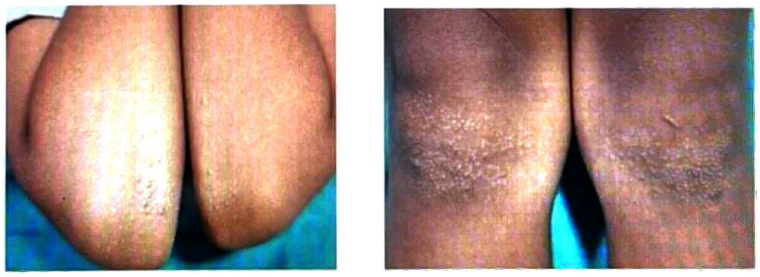
Skin and nutritional dermatoses 00:15:28

60c6b3eeaa8ded0e4e7e5ea7

Phrynoderma : Phryno : Toad.

Derma : Skin.

- Skin lesions : Hyperkeratotic follicular papules.
- Distribution : Extensor aspect of knees and elbows.
- Association : Essential fatty acid deficiency, Vit. A deficiency.



Hyperkeratotic follicular papules in phrynoderma

Pellagra :

Pelle : Skin

Agra : Rough.

Deficiency :

- Niacin (Vit B3).
- Amino acid : Tryptophan.

Diet :

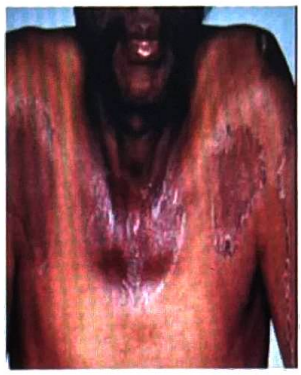
- Jowar.
- Maize.

Substance abuse : Chronic alcohol intake.

Triad :

- Dermatitis : Photodermatitis (sun exposed areas) → Casal's necklace.
- Diarrhea.
- Dementia.

Death if not treated.



Casal's necklace in pellagra

Acrodermatitis enteropathica :

Inheritance : Autosomal recessive.

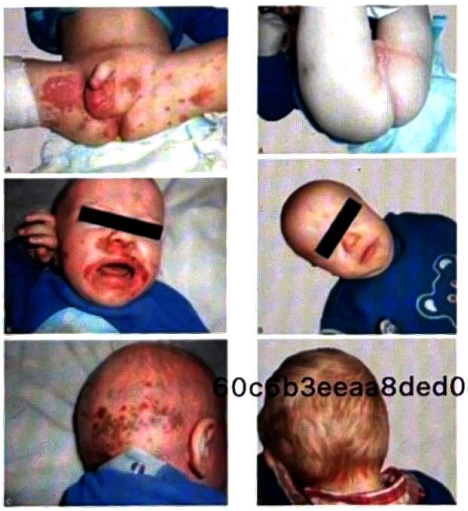
Gene : SLC39A4 gene (Solute Carrier protein) that facilitates zinc absorption.

Defective intestinal zinc absorption.

Age of presentation : At time of weaning of child from breast milk (6 months).

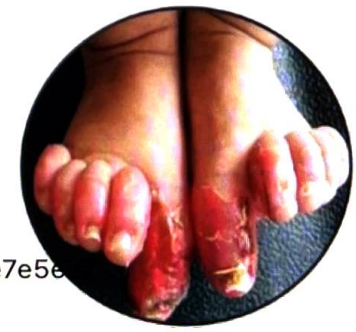
Triad :

- Dermatitis : Periorificial and acral dermatitis.
- Diarrhea.
- Alopecia.



Before treatment

After zinc treatment



Acral dermatitis

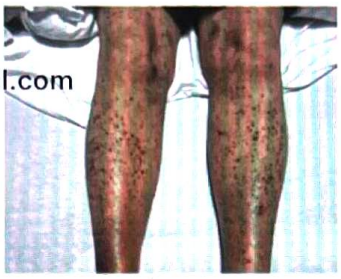
Treatment : Lifelong zinc supplementation.

Scurvy :

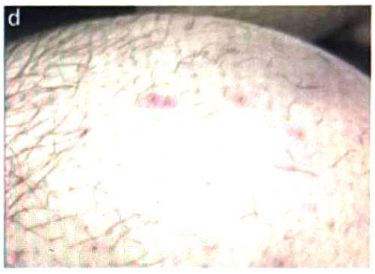
Deficiency : Vitamin C.

Skin lesions : Perifollicular purpura.

Hair feature : Cork screw hair.



Perifollicular purpura in scurvy



Corkscrew hair in scurvy

Vit B2 deficiency (riboflavin) :

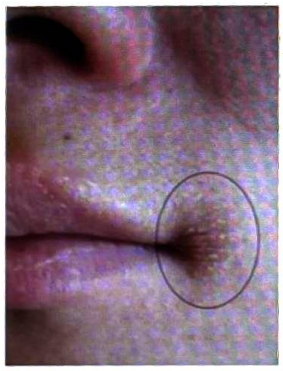
Skin :

- Nasolabial dyssebacia.
- Seborrheic dermatitis.

Oral cavity : Angular cheilitis/ stomatitis.

Tongue : Glossitis (magenta colored tongue).

Eye : Neovascularisation of cornea.



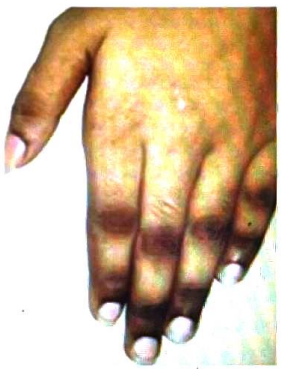
Angular cheilitis

Vitamin B12 deficiency :

Skin : Hyperpigmentation of knuckles.

Tongue : Glossitis → Beefy red tongue.

Hair : Premature greying of hair (canities).



Hyperpigmented knuckles

Kwashiorkar :

Skin lesions :

- Flaky paint dermatoses.
- Crazy pavement dermatoses.

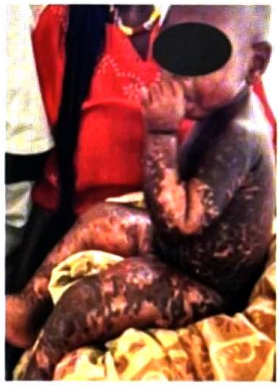
Active space

Hair :

Flag sign : Sharply demarcated areas of hypopigmentation and normal hair in an alternating pattern.

Mechanism : Intermittent malnutrition.

Flag sign



Flaky paint dermatoses

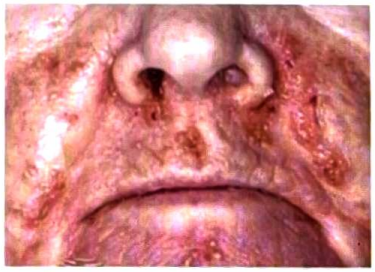
Skin And internal malignancy 00:29:012

Glucaganoma :

It is a tumour of alpha cells of pancreas.

Skin eruption : NME

(Necrolytic migratory erythema).



Glucaganoma

Necrolytic migratory erythema :

- Incidence : 70% presenting feature of glucaganoma.
- Site : Periorificial, extremities.
- Lesions : Plaques with central clearing.
  Margin of lesions will have vesicles which rupture into Crusted erosions.

Active space

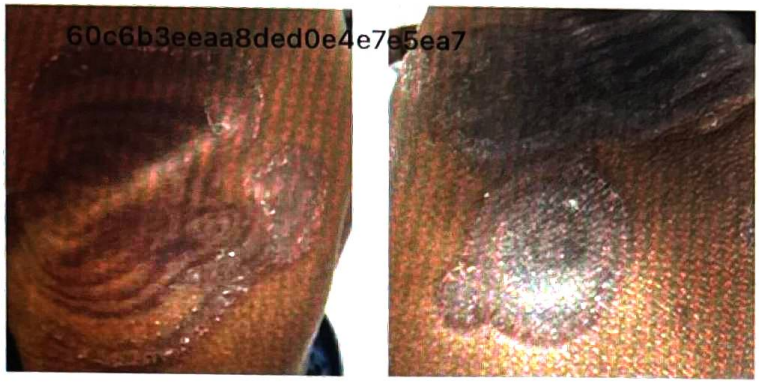
CA lung :

Rash : EGR (Erythema Gyratum Repens).

- Erythema : Red eruption.
- Gyratum : Coiled around a point.
- Repens : To crawl.

Sites : Proximal extremities and trunk.

Appearance : Wood grain pattern.



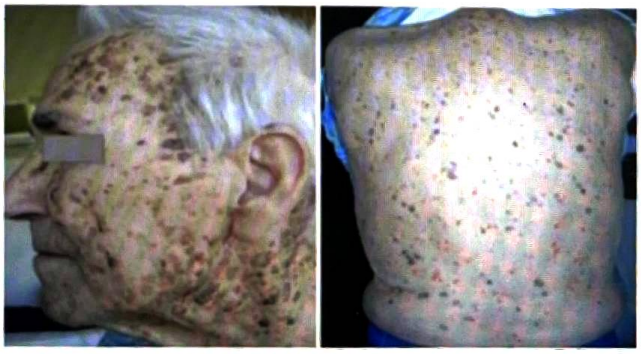
Erythema Gyratum Repens

CA pancreas :

Association : migratory thrombophlebitis (Trousseau syndrome).

GI adenocarcinoma :

- Seborrheic Keratosis (benign tumor of the Keratinocytes).
- In GI adenocarcinoma : Rapid, multiple, sudden onset seborrheic Keratosis (benign tumor of Keratinocytes) → Sign of Lesser Trelat.



Sign of Lesser Trelat

SHORT TOPICS IN DERMATOLOGY

Index :

Panniculitis

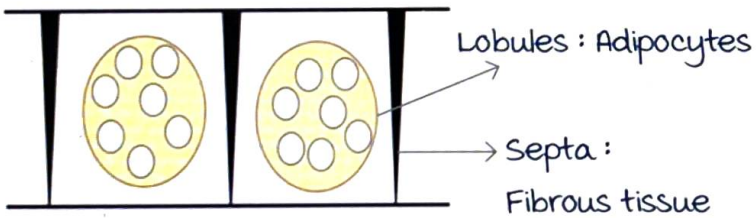
00:00:28

Term : Panniculus means fat.

Definition : Inflammation of subcutaneous fat.

Types based on structure :

- Septal panniculitis : Inflammation restricted to septae.
- Lobular panniculitis : Inflammation restricted to lobules.



Erythema nodosum :

Definition : Delayed hypersensitivity reaction in response to variable trigger antigens that clinically presents as acute nodular septal panniculitis.

Etiology :

MC cause : Idiopathic (no demonstrable cause).

Infections : Streptococci (MC), Tuberculosis.

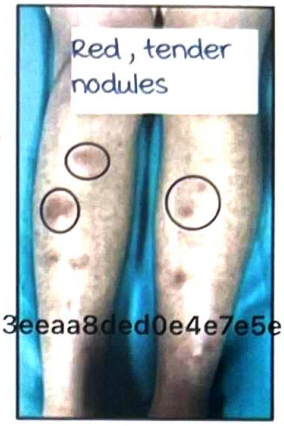
Drugs : OCPs (Oral contraceptive pills), Sulfonamides, Iodides.

IBD (Inflammatory Bowel Disease) : Crohn's Disease >>> Ulcerative Colitis (UC).

Other causes : Sarcoidosis, Behcet's disease.

Clinical features :

- Age group : Seen in children
- Lesions : Multiple, red , tender nodules.
- Site : Anterior aspect of leg.
- Heals without scarring.



Erythema nodosum

Histopathology : Septal panniculitis.

Treatment :

- Mainstay : Treatment of underlying cause.
- Pain management (for nodules) : NSAIDS (anti-inflammatory drugs).

Neutrophilic dermatoses

00:05:30

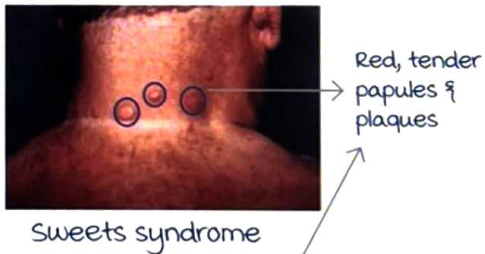
Histopathology : Dense, diffuse, dermal neutrophilic infiltrates can be seen.

Diseases :

- Sweets syndrome.
- Pyoderma gangrenosum.

Sweets syndrome :

Also known as acute febrile neutrophilic dermatoses.



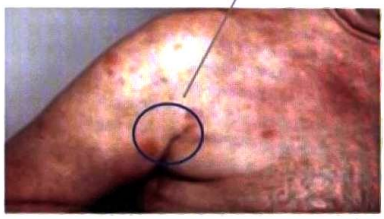
Sweets syndrome

Etiology :

- Infections.
- Pregnancy.
- Hematologic malignancies.

Clinical features :

- Fever.
- Abrupt onset of tender red papules & plaques.
- Site → Face, chest, trunk.



Sweets syndrome

Histopathology : Dermal neutrophilia.

Treatment : Systemic steroids.

Pyoderma gangrenosum :

Misnomer : Not a bacterial infection.

Pyoderma gangrenosum

Etiology :

- Rheumatoid arthritis (extra-articular manifestation).
- IBD (extra-intestinal manifestation) : UC & Crohn's disease.
- Leukemias.

Clinical features :

- Lesion : Rapidly progressive, painful, cutaneous ulcer with an undermined edge & violaceous hue.

Histopathology : Dermal neutrophilic infiltrate.

Treatment : Treat the underlying cause + systemic steroids.
Do not do grafting as it is detrimental.

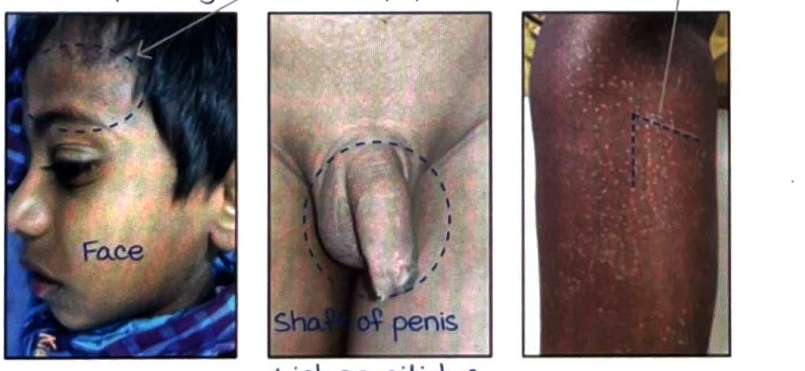
Lichen nitidus

Definition : Chronic inflammatory disease of unknown etiology.

Term : Nitidus means shiny.

Clinical features :

- Age group : Seen in children.
- Lesions : Papules (shiny, skin colored).
- Site : Face, dorsum of forearm, shaft of penis.
- Named phenomenon : Koebner's phenomenon : New lesions along the line of trauma.



Lichen nitidus

Histopathology : Multiple lymphocytes and epithelioid cells in the infiltrate.

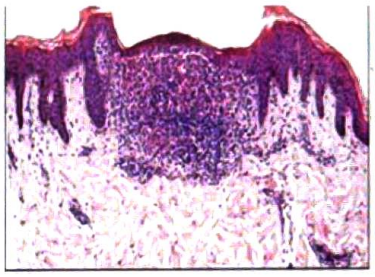
This chronic inflammatory infiltrate (ball-like) is encircled by rete ridges (claw-like). The claw clutching the ball appearance is seen.

Treatment :

- Usually self-resolving.
- If symptoms or resistant lesions present : Topical steroids.

Claw clutching ball appearance

60c6b3eeaa8ded0e4e7e5ea7



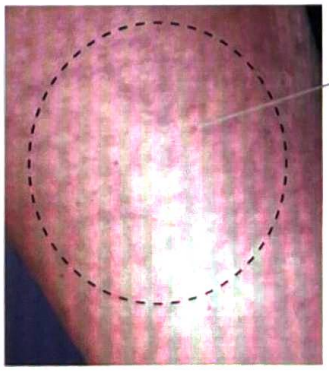
Cutis marmorata

00:16:24

Term : Cutis means skin, Marmorata means marbling.

Clinical features :

- Disease : Physiologic vascular response to cold.
- Age group : Seen in neonates.
- Lesion : Reticular (network-like) symmetric marbling.
- Site : Trunk ,extremities.
- Changes with temperature : Warming the child resolves cutis marmorata.



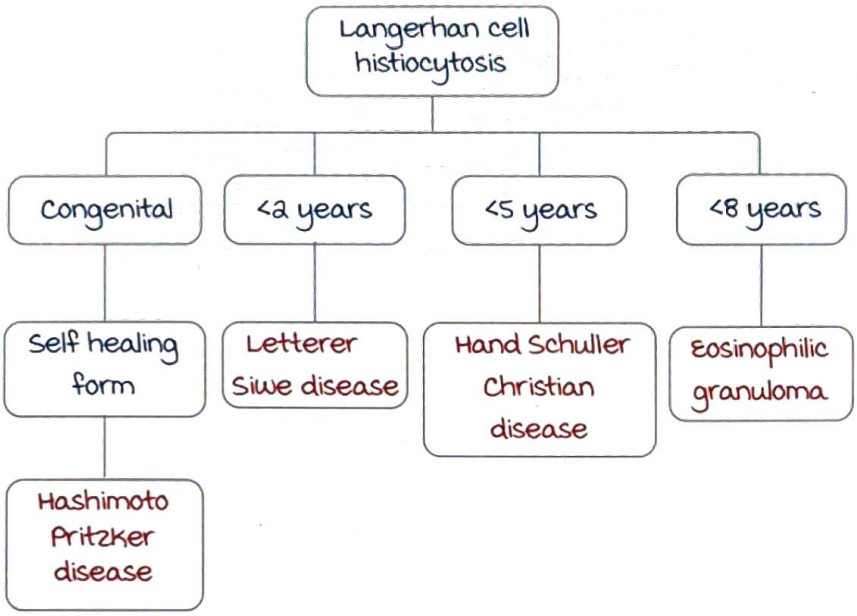
Cutis marmorata

Langerhans cell histiocytosis 00:17:47

Incidence : Rare histiocytic disorder.

Immunohistochemical markers :

- S100.
- CD 1a.
- CD 207 (most specific) : Also referred as Langerin.



Letterer Siwe disease :

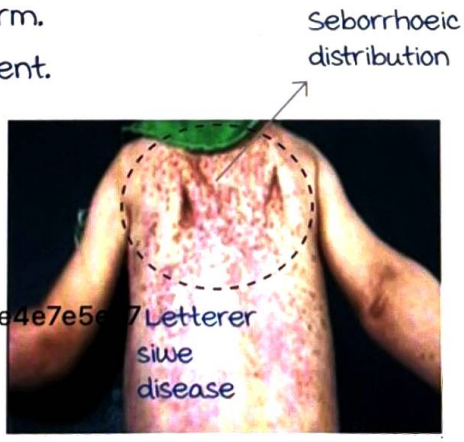
Course : Acute disseminated form.

80% of patients : Bone involvement.

30% : Hepatosplenomegaly.

50% : Skin involvement :

Seborrhoeic distribution (can be seen where density of sebaceous glands are more e.g., external auditory canal, pre sternal area , face, chest).



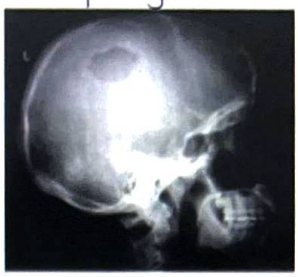
Hand Schuller Christian disease :

- Calvarial skull defects.
- Diabetes insipidus.
- Exophthalmos.

Eosinophilic granuloma :

- Lesion : Osteolytic lesion.
- Site : Temporoparietal region.

Eosinophilic granuloma



Mycosis fungoides

00:22:06

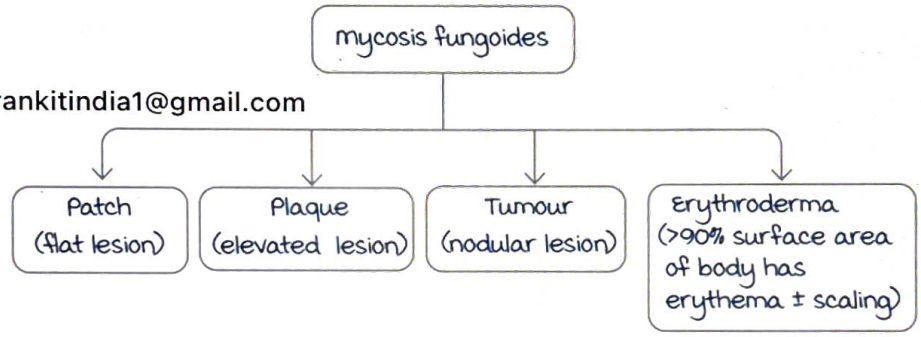
Also known as Cutaneous T-cell lymphoma (CTCL).

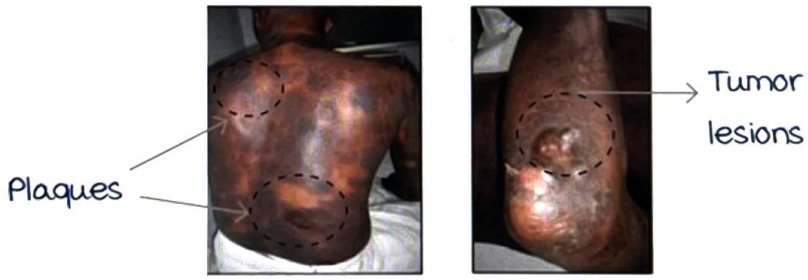
Type : Non hodgkin's lymphoma.

Cell : CD4 T cells.

Course : Indolent (slow progression).

Stages :





Histopathology :

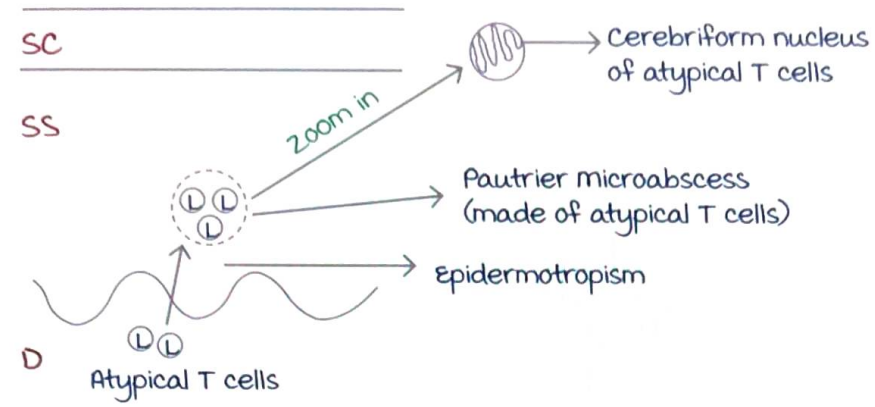
Atypical T cells (lymphocytes present in dermis) migrate to epidermis

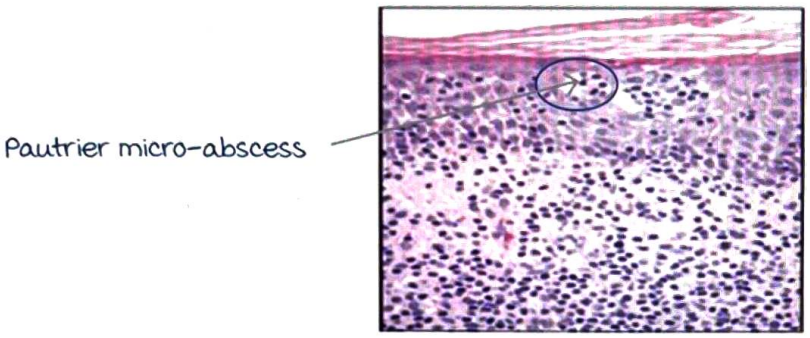
↓

The migration process is called epidermotropism

↓

These migrated T cells will form a collection called Pautrier microabscess (made of atypical cells seen in mycosis fungoides).





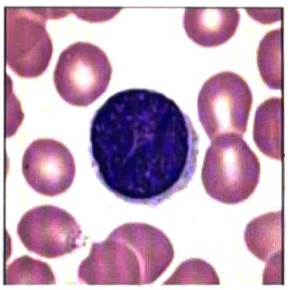
The nuclei of the migrated T cells are cerebriform in shape.

Treatment :

- Topical : Nitrogen mustards.
- Systemic chemotherapy drugs : Chlorambucil.
- Phototherapy : Thin plaques → Narrow Band Ultraviolet-B radiation (UV-B).
  Thick plaques → PUVA radiation (Psoralen + UV-A).
- Total Skin Electron Beam Therapy (TSEB).

60c6b3eeaa8ded0e4e7e5ea7

Sezary syndrome :
Definition : Erythroderma + leukemic variant of CTCL.
Course : Aggressive.
Triad :


Sezary cells

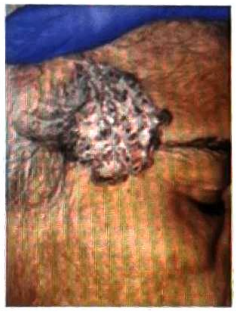
- Erythroderma (4th stage of mycosis fungoides)
- Generalized lymphadenopathy.
- Circulating Sezary cells (atypical T cells).

Active space

Basal cell carcinoma (BCC)

m/c primary malignancy of the skin.
Special features : Locally invasive tumor, metastasis is rare.
Pathway activated : Sonic Hedgehog pathway.

m/c site : Above the line joining the angle of the mouth to lobule of the ear.
m/c type : Nodule type - Translucent nodule with surface telangiectasia (prominent blood vessels on top of nodule) → undergoes central necrosis → Rodent Ulcer



Classical presentation of BCC - Rodent Ulcer

Treament :

- Surgery : Mohs micrographic surgery (sequential removal of tumor till the margin is negative (-) for tumor cells). Preferred as it has better cosmetic outcome.
- Targetted therapy (BCC) : Sonic Hedgehog pathway inhibitors → Vismodegib & Sonidegib.

Mnemonic :
Hedgehog → Vismodegib (DGE flipped to DEG).
Sonidegib.
IB → Inhibitors of pathway.