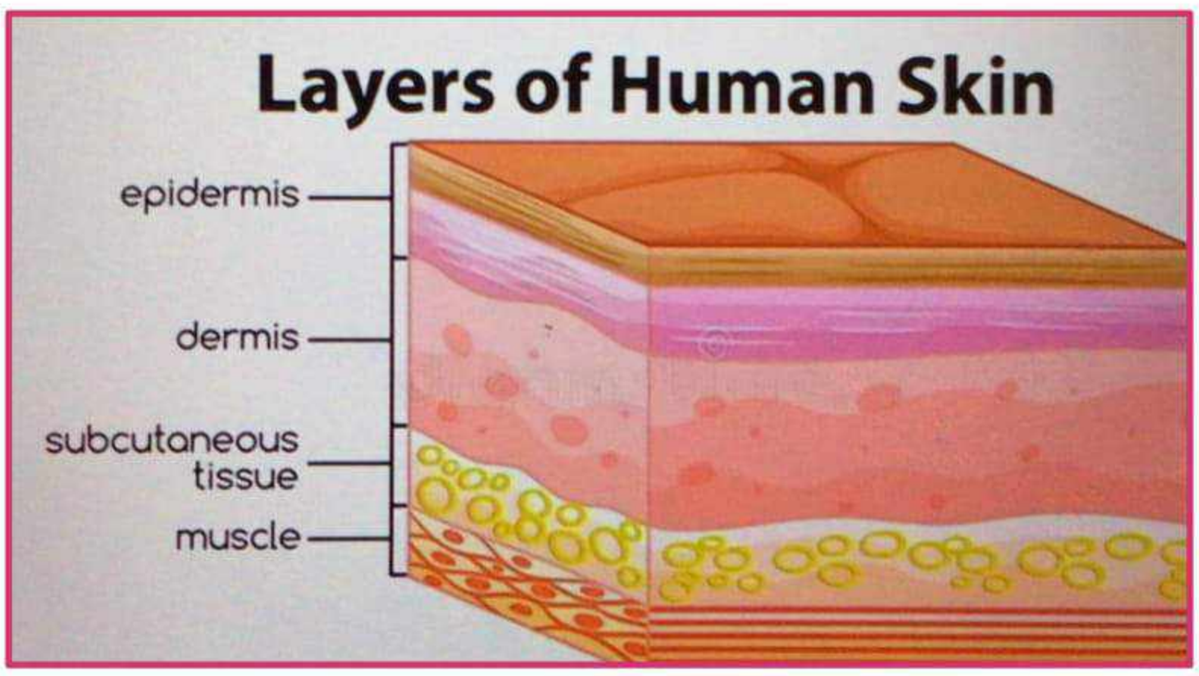
eGurukul

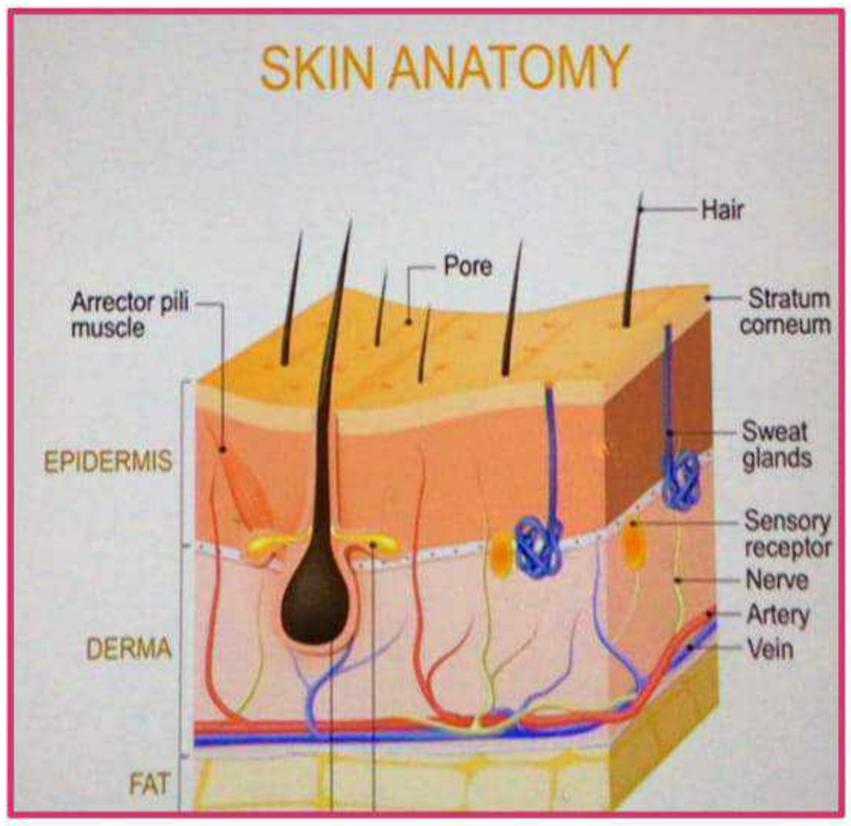
In collaboration with

Notespaedia

Skin Structure

- **Integumentary System :** Skin and its appendages
- Largest organ of the body
- 4-5 Kg
- Surface Area : 1.4 - 2 m^2
- **Glabrous skin :** non-hairy skin ⟶ palms and soles.

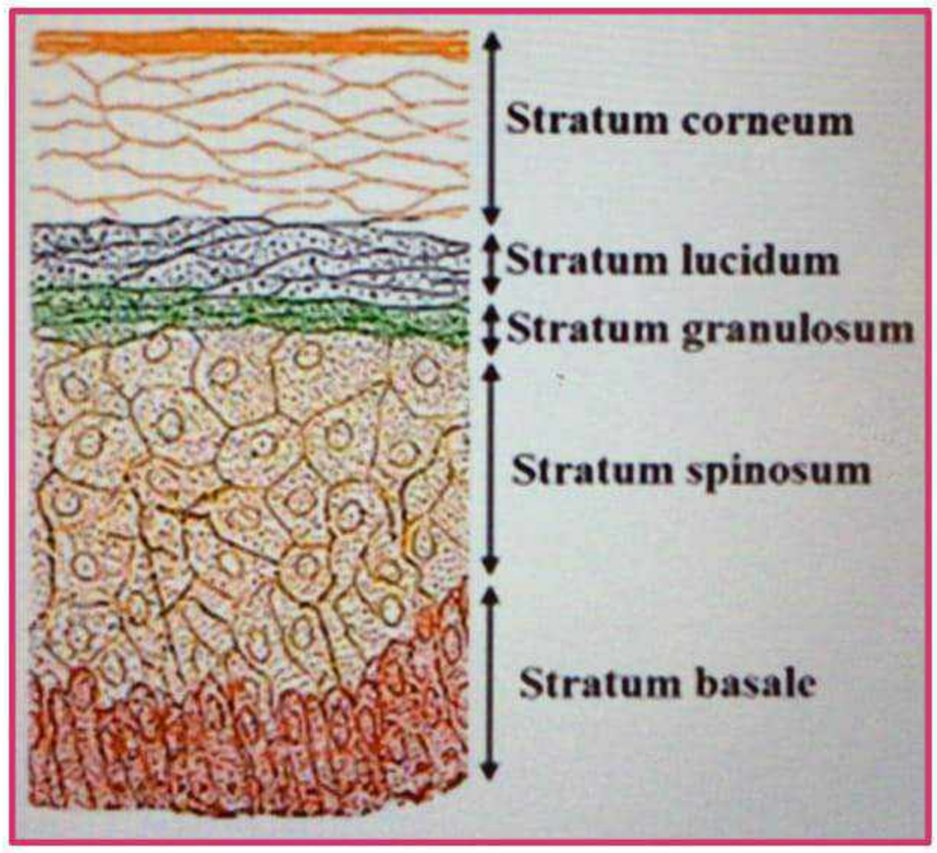




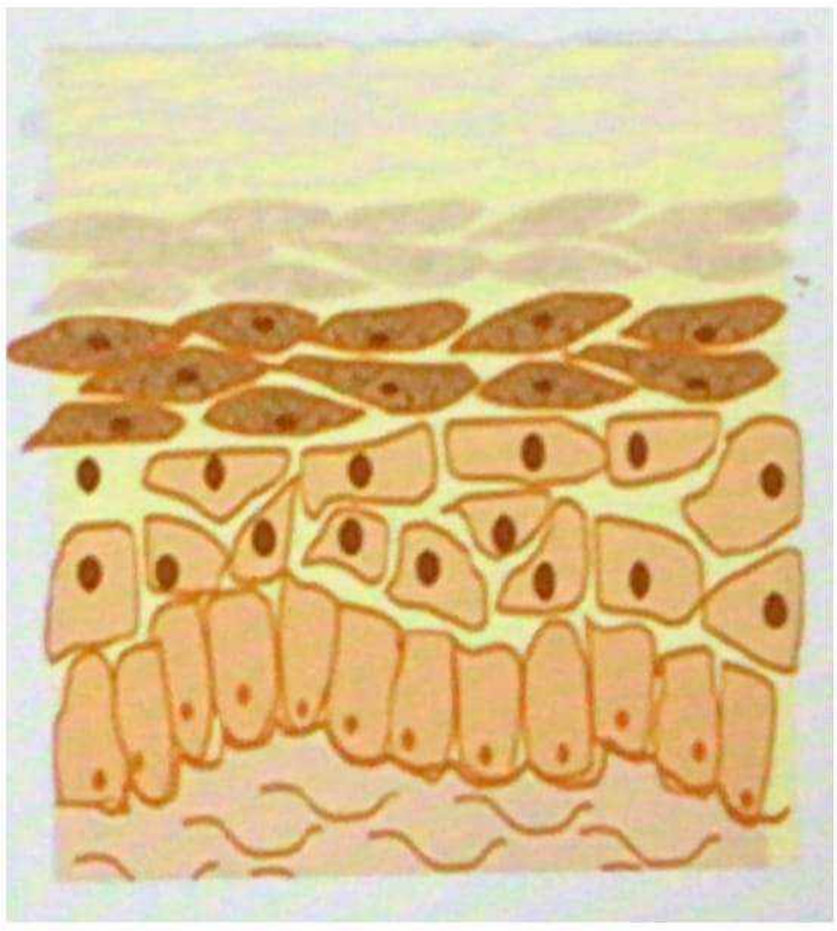
EPIDERMIS

03:38

- 0.1 mm - 1 mm in diameter
  - ↓ eyelids
  - ↓ palms and soles.
- Stratum lucidum - presents in palms and soles.



Cell differentiation



- As cells move from Stratum basale to Stratum corneum
- flat
- Loss of nuclei
- Loss of mitosis
- ↑ in size
- Dehydrated.

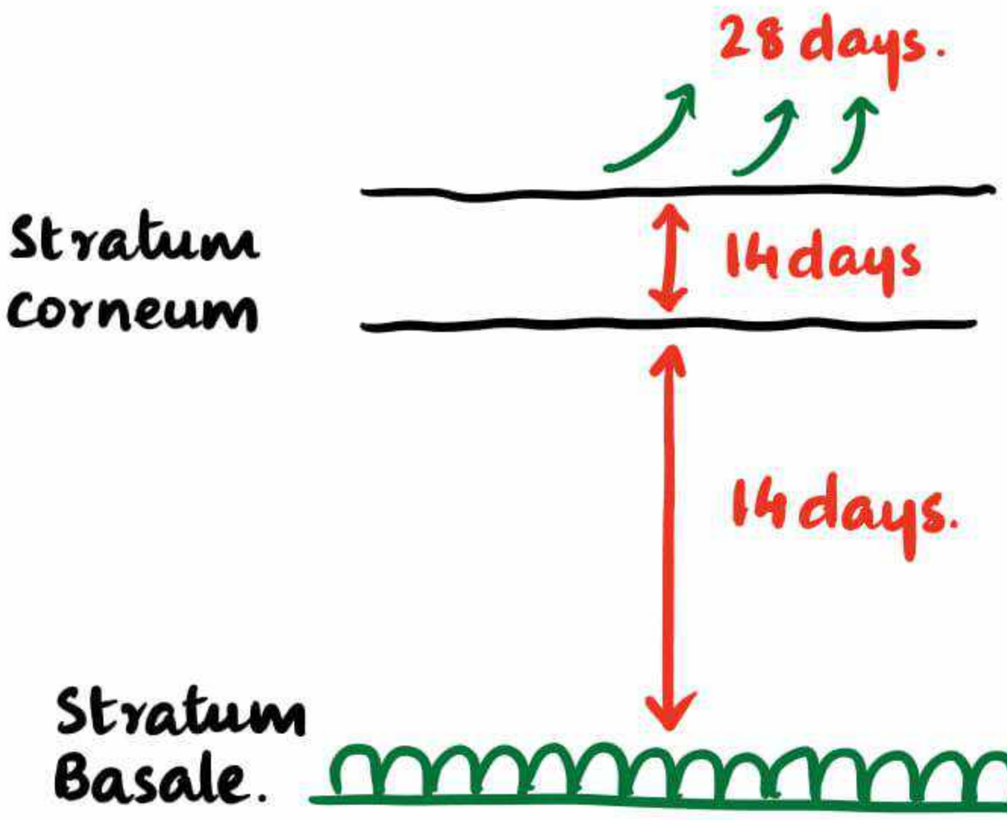
- In Preterm baby : Stratum corneum not formed.

CELL KINETICS

09:00

- Cell cycle of Keratinocyte is 300 hrs.

active space



- Epidermal Turnover time : 56 days (52-75 days)
- In Psoriasis → Cell cycle → 36hrs
  → Epidermal Turnover Time → 4 days.

STRATUM CORNEUM 14:00

- Topmost layer
- Dead Keratinocytes (corneocytes)
- Flat cells with no nuclei
- Barrier function

Parakeratosis :

- Retention of nuclei in Stratum corneum.
- It can be physiological or Pathological.

active space

- Physiological : Mouth and vagina
- Pathological : Psoriasis , Eczema , Seborrheic dermatitis.
  Actinic keratosis.

Hyperkeratosis

- Increased thickening of Stratum corneum.
- seen in
  i) Lichen planus
  ii) Psoriasis

STRATUM LUCIDUM 17:55

- Present in palms and soles.
- Clear cell layer
  ↳ Refractile granules of Eleidin.

STRATUM GRANULOSUM 19:03

- Granules present are
  i) Keratohyalin granules : Profillagrin → Stratum Basale
  (Filament Aggregating protein)
  ↓
  forms Fillagrin → Stratum corneum
  (defects leads to Icthyosis vulgaris)

active space

ii) Lamellar granules (Membrane coating granules / Odland bodies)

↳ also formed in lung (Type I Alveolar cell)

- form lipid ⟶ acts as barrier function

(defect leads to Asteatotic Eczema)

- 1-2 cell layer thick

Hypergranulosis:

- ↑ Thickening
- seen in lichen planus

Agranulosis

- Absent granular layer
- seen in Psoriasis, Icthyosis vulgaris.

Dyskeratosis

- Abnormal keratinization
- Premature keratinization occuring below S. granulosum
- Accumulation of keratin into keratinocytes occurs in lower layers.
- Benign: Hailey Hailey Disease, Dariers Disease.

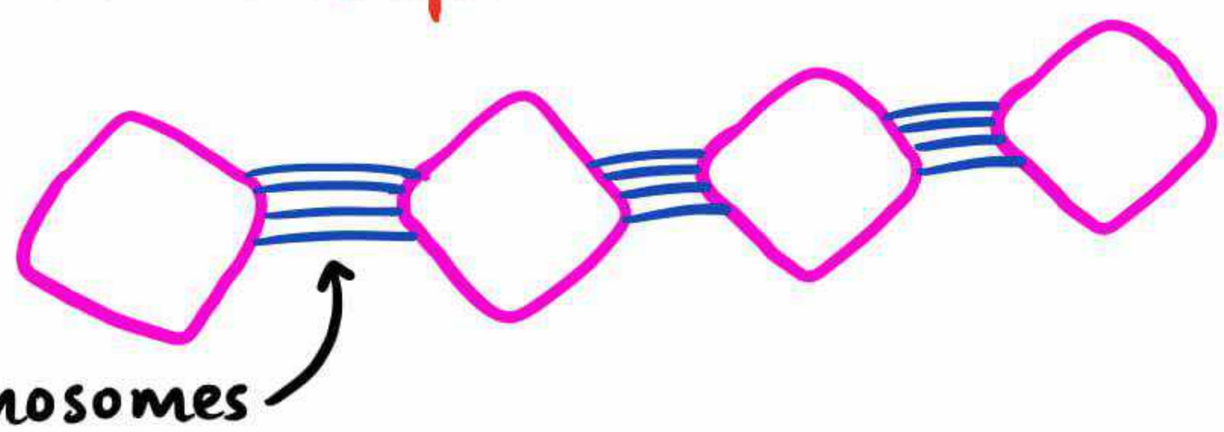
active space

- Malignant :
  - Basal cell carcinoma
  - Paget's disease
  - Squamous cell carcinoma

STRATUM SPINOSUM

25:55

- a.k.a Prickle cell layer



- Thickest layer of Epidermis

Spongiosis :

- Intercellular edema
- Acute Eczema

Ballooning :

- Intracellular edema
- Seen in Eczema

Acanthosis :

- ↑ Thickening of S. spinosum
- Seen in C/C Eczema (Lichenification)

- Malphigian layer → S. Basale + S. Spinosum

STRATUM BASALE

30:28

- Germinative cell layer
- Mitotically active
- Single Layer Thickness.

Acantholysis :

- separation of keratinocytes due to loss of desmosomal attachments.
- In Pemphigus → Acantholytic cells → oval nuclei covers 7/8 cell peripheral rim of cytoplasm.

Basal cell degeneration

- degeneration of cells of Stratum Basale
- seen in Lichen planus

active space

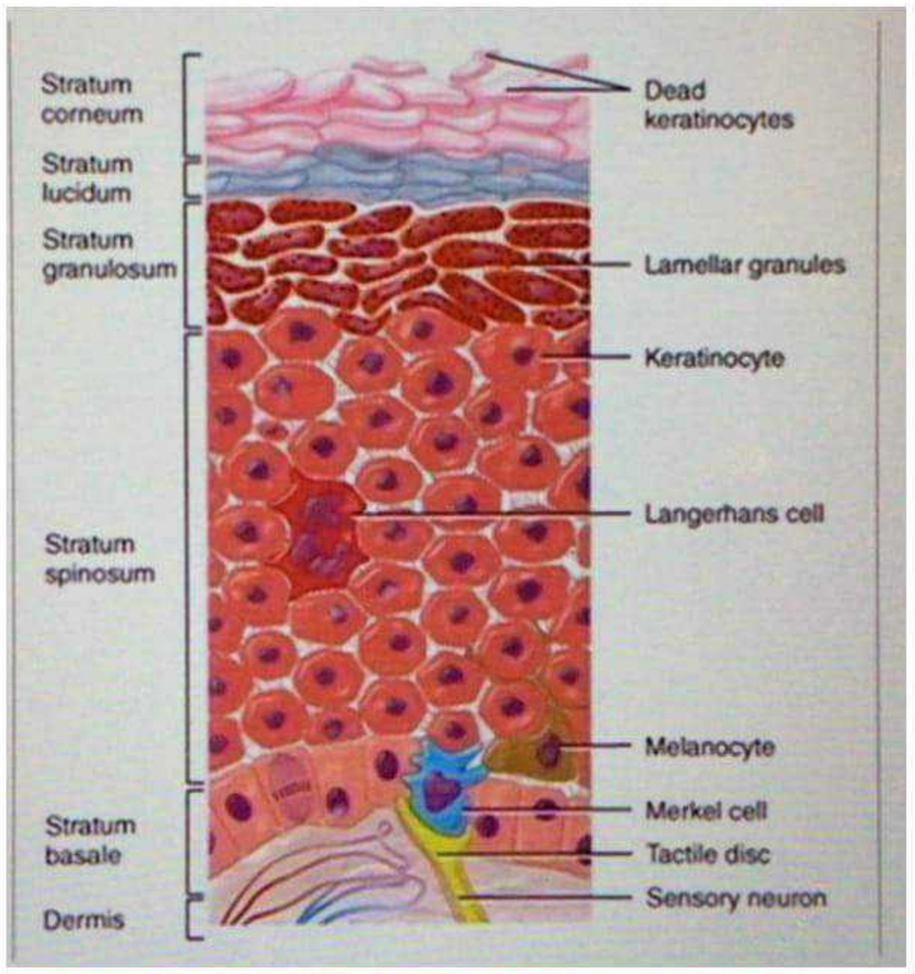
Cells in Epidermis

i) Keratinocyte

ii) Langerhans cell

iii) Melanocytes

iv) Merkel cells.



DEVELOPMENT OF EPIDERMAL CELLS 35:00

Local / Ectoderm

Keratinocytes

Migrate

Neural crest → Melanocytes

Mesenchyme → Langerhan's cell

- Merkel cells comes from Ectoderm / Neural crest

KERATINOCYTES 37:20

- 80-90% Epidermis
- Tonofilaments / Keratin intermediate Filaments

Tonofilaments / Keratin intermediate Filaments

↓

Keratin

Hard Keratin	Soft keratin
• Disulphide bonds - strong	• Disulphide bonds - weak
• seen in nails and hair	• seen in skin

• Keratinocyte present in all layers of skin

LANGERHANS CELLS 39:13

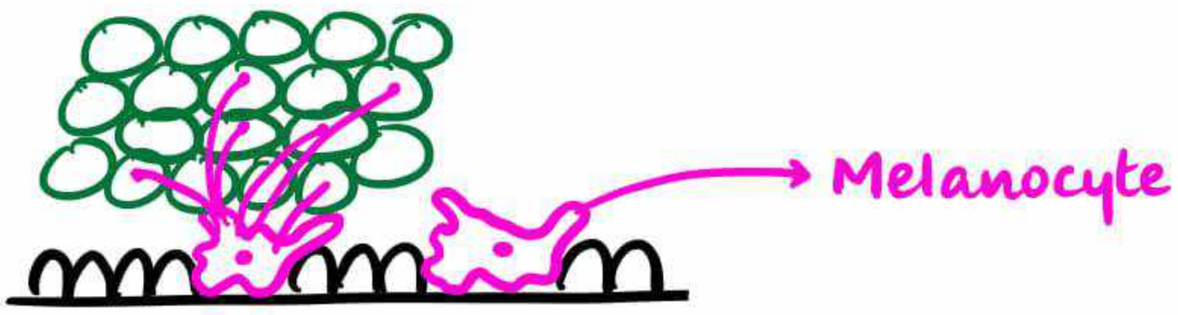
- present in Stratum spinosum
- are Antigen Presenting Cells → Immune response.
- cause Receptor mediated endocytosis
- derived from mesenchyme
- Bierbeck granules are ⊕
  ↳ (Rod / Racquet shaped)
- Markers : CD1a, CD20T, S-100.

MELANOCYTES 41:20

- pigment forming cells → ⊕ in Stratum Basale
- derived from neural crest.

active space

- are pigment forming cells
- are dendritic cells ⟶ others : Langerhans cell



Melanocyte : Keratinocyte = 1 : 10

- **Epidermal Melanin unit :**
  - Melanocytes form melanosomes (have melanin)
  - 1 : 36 (Each melanocyte transfer Melanosomes to 36 Keratinocytes)

Changes in Skin colour ⟶ due to melanosome / Melanin.

- **Melanin** ⟶ 2 Types
  - Eumelanin - Blackish brown
  - Phaeomelanin - Fake, yellowish Red

- **Melanosomes** ⟶
  - Size
  - Distribution
  - Number

active space

MERKEL CELLS

47:37

- present in Stratum Basale
- derived from Ectoderm > Neural crest
- Slow adapting Type 1 Touch Receptors

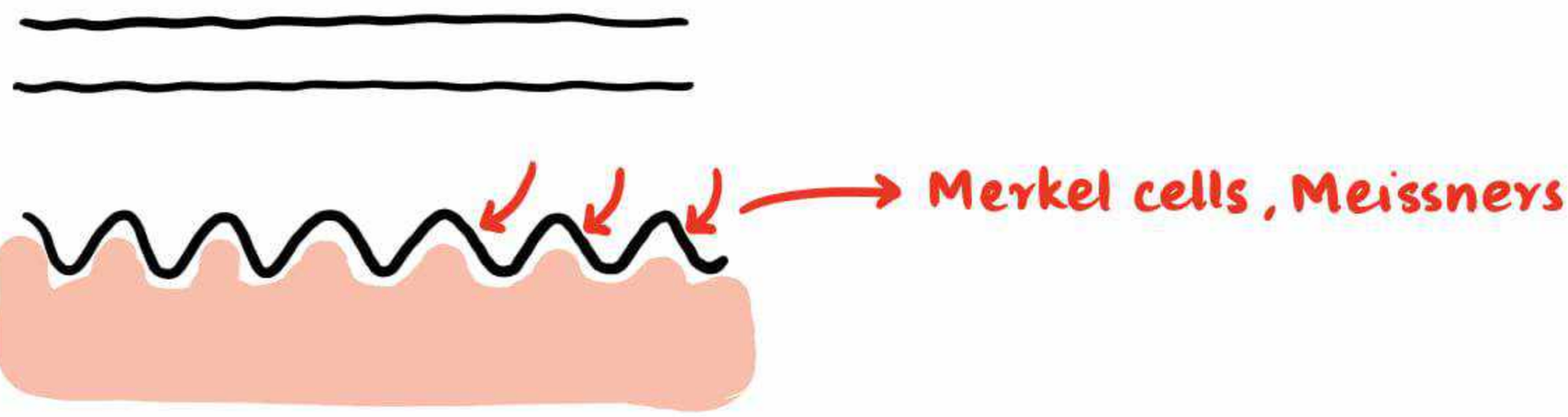
Touch Receptors

Type I Slow Adapting

- Merkel cells
- Ruffini's ending
- Free Nerve endings

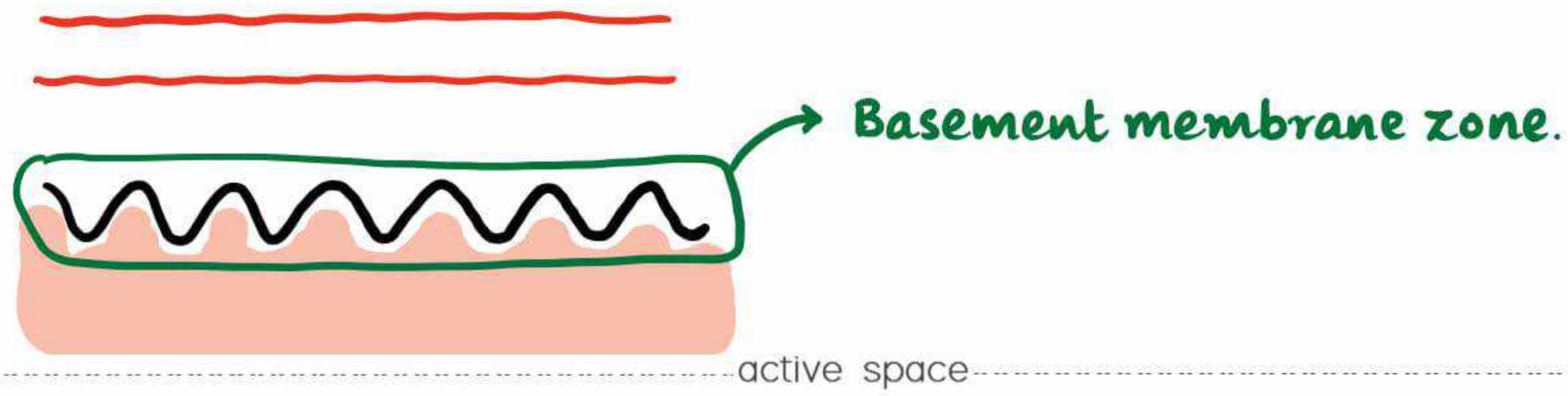
Type II Rapid Adapting.

- Meissners corpuscles
- Hair endings
- Pacinian corpuscles (Pressure and Vibration)

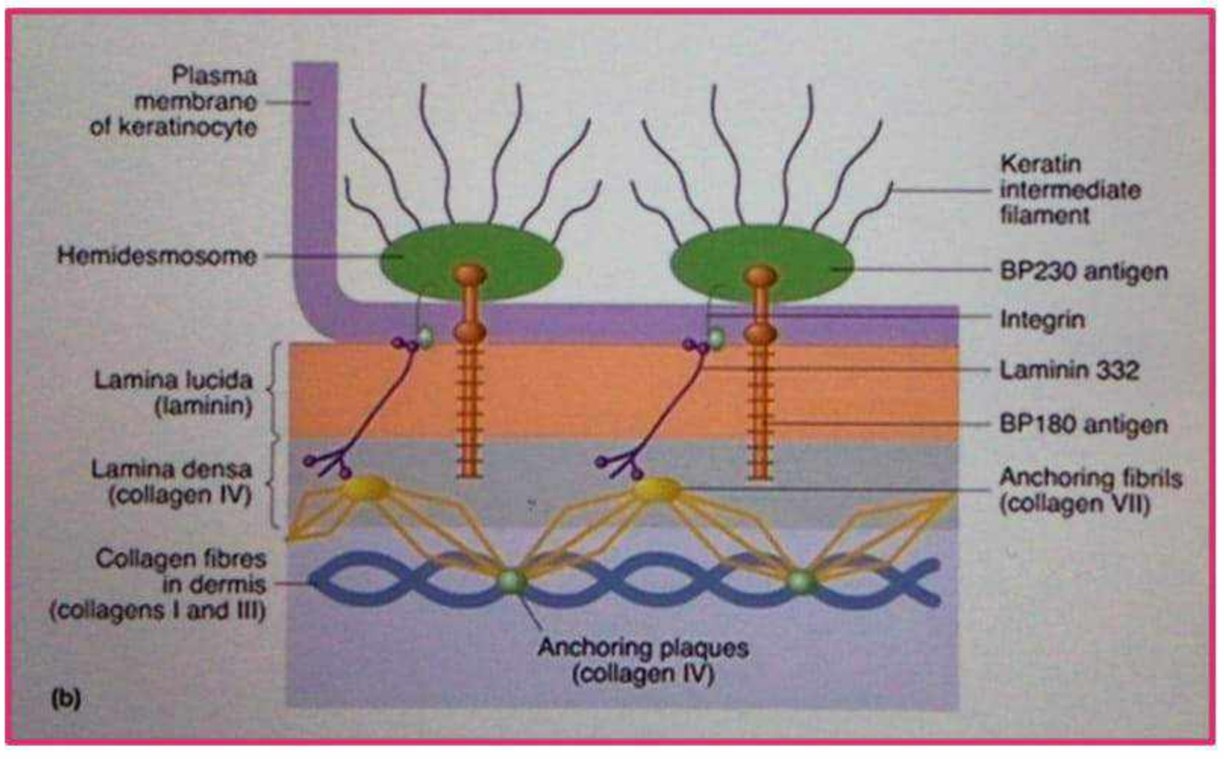


DERMO-EPIDERMAL JUNCTION

50:42

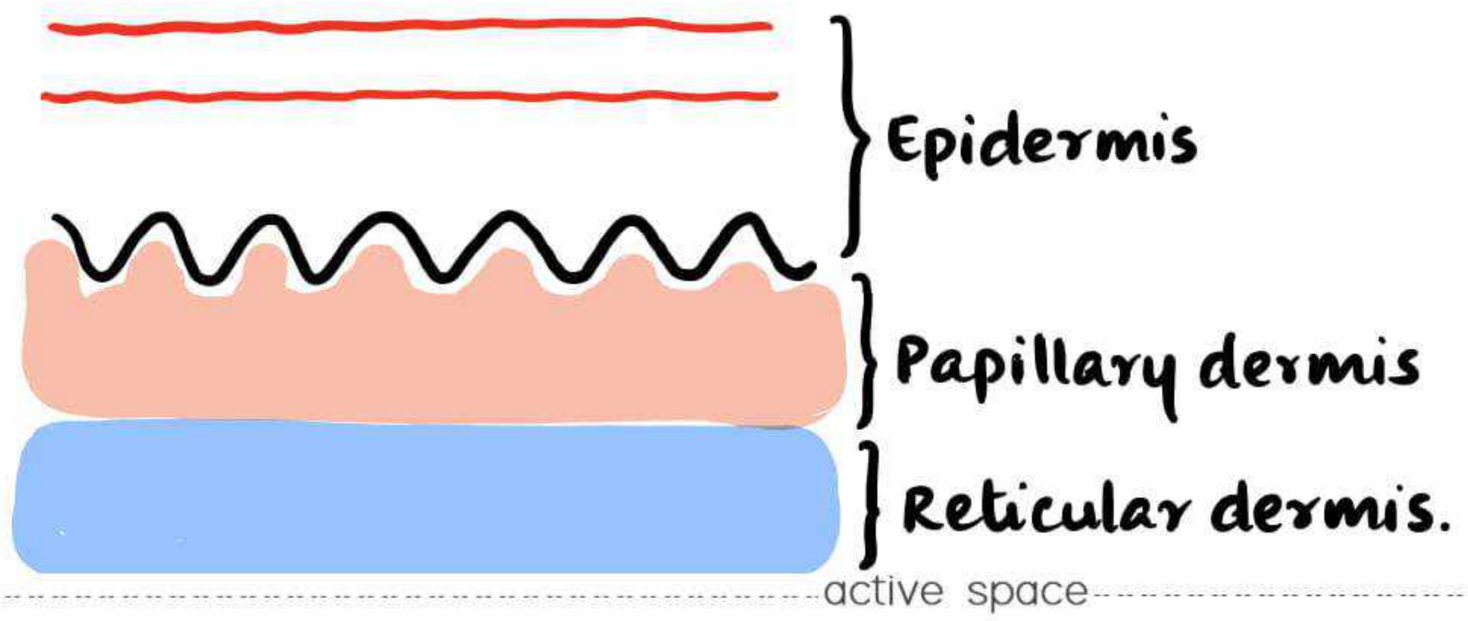


- Important Role in Adhesion and Signalling
- Major component : Type IV Collagen

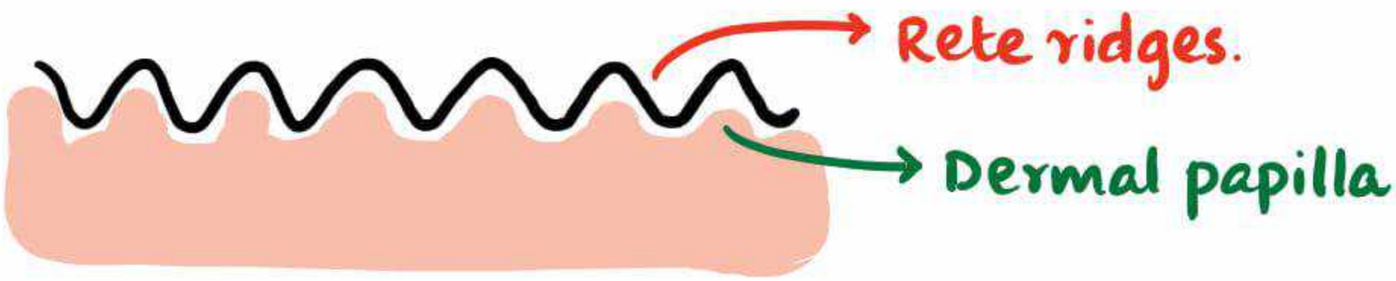


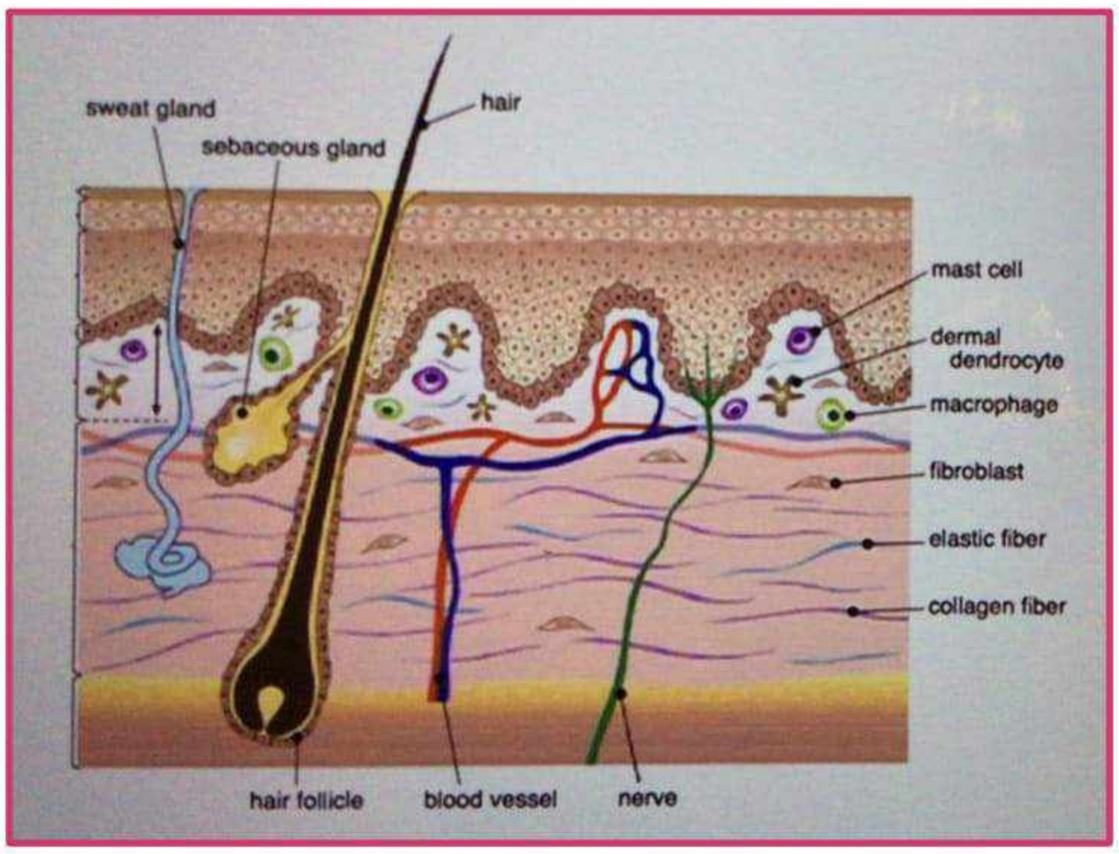
- Hemidesmosomes : contains Plectin, Integrin
- Lamina lucida : Laminin, Anchoring filaments
- Lamina Densa : Type IV Collagen.
- Sublamina densa : Anchoring fibrils
- Collagen Type I and III

DERMIS 54:19



active space





Dermis contains :

- Cells
- Fibres → Collagen, Elastin
- Ground substance → Hyaluronic acid, Chondroitin sulfate / Dermatan sulfate
- Blood vessels
- Glands
- Hair follicles } Appendages
- Accessory muscles
- Lymphatics

active space

CELLS IN DERMIS 58:40

- Fibroblast → Most important and most abundant
- Langerhan's cell
- Lymphocytes
- Phagocytes
- Mast cells

SKIN APPENDAGES 59:27

1. Glands
   - Sebaceous glands
   - Sweat glands
2. Hair
3. Nair

Sebaceous Gland

- Oil forming glands
- Holocrine glands → who cell forms secretion
- Situated on upper part of body
- Physiologically active during puberty.
- Acne → Propionobacterium acnes

active space

- Dandruff ⟶ Pityrosporum ovale
- Demodecidosis ⟶ Demodex mite
- usually associated with hair follicles
- Not associated with hair follicle :

  i) Meibomian glands ⟶ Eyelids

  ii) Fordyce's spots ⟶ Oral epithelium

  iii) Montogmery tubercles ⟶ Nipples

  iv) Tyson's gland ⟶ Penis.

Sweat glands :

- 2 Types : i) Eccrine

  ii) Apocrine

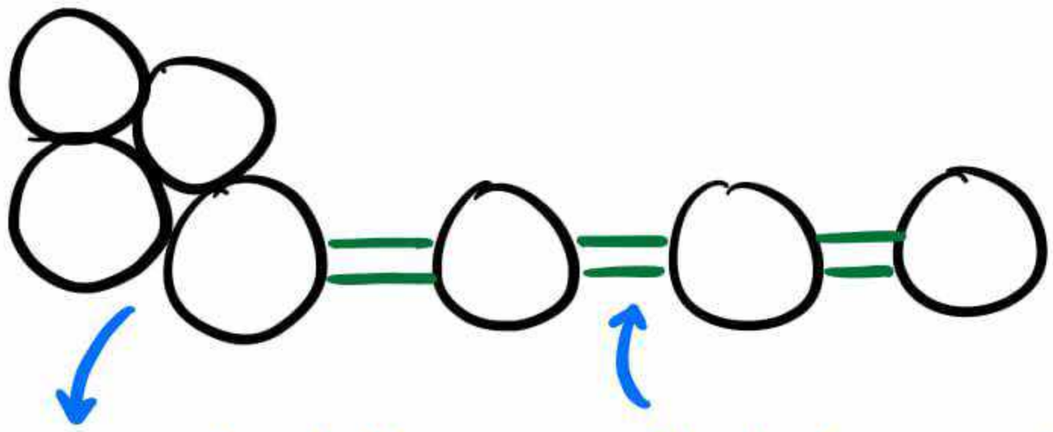
Eccrine	Apocrine
• secrete out secretions • present all over the body (max^{m} : Palms and soles) • Thermoregulation	• decapitation • always associated with hair follicles. • Present in axilla, groins, infra mammary areas • Odour

active space

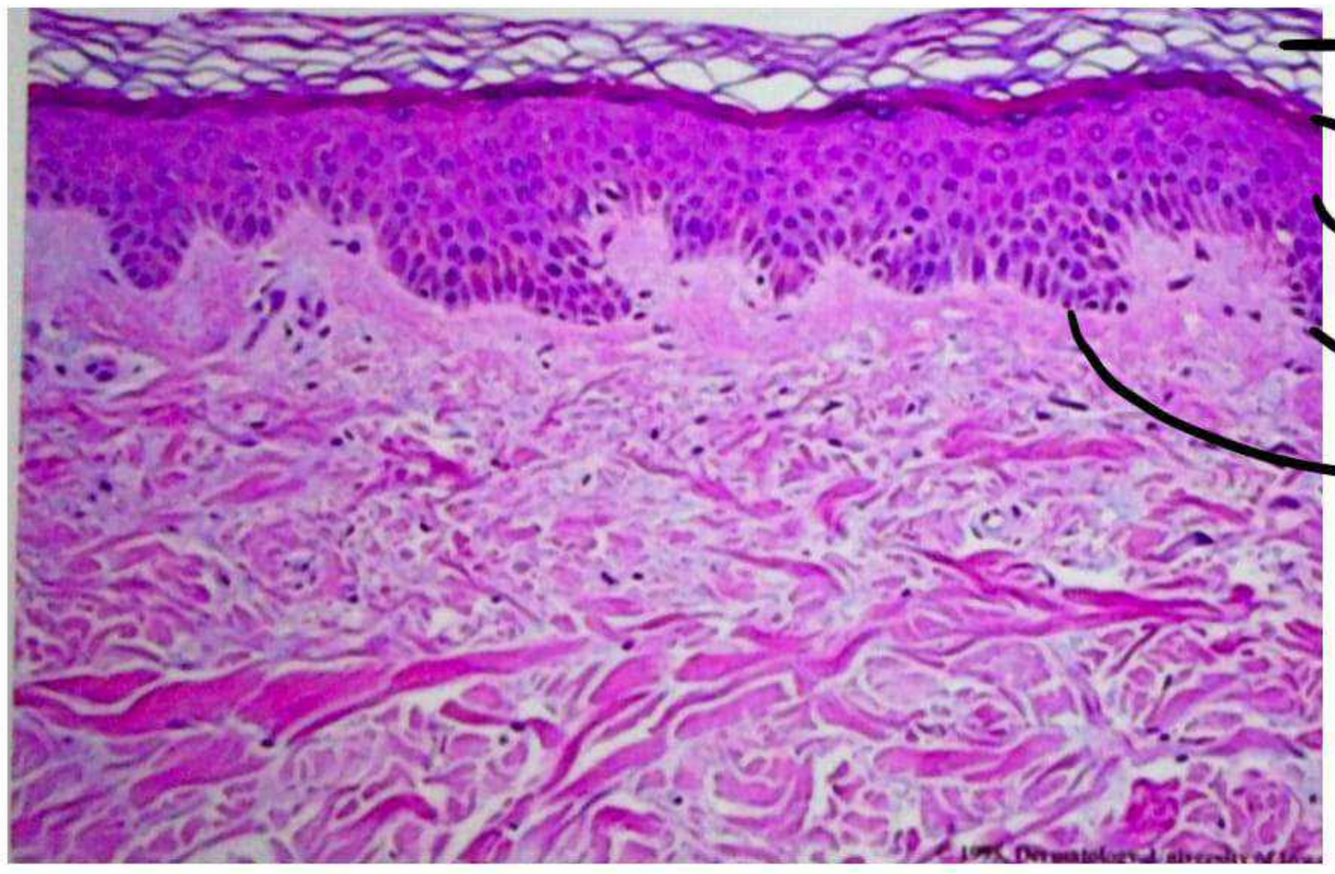
SUBCUTANEOUS FAT

01:07:35

- acts like cushion
- 80 % of Body fat present in Subcutis
- contains blood and lymphatics
- absent from eyelid and genitalia



Histology of skin



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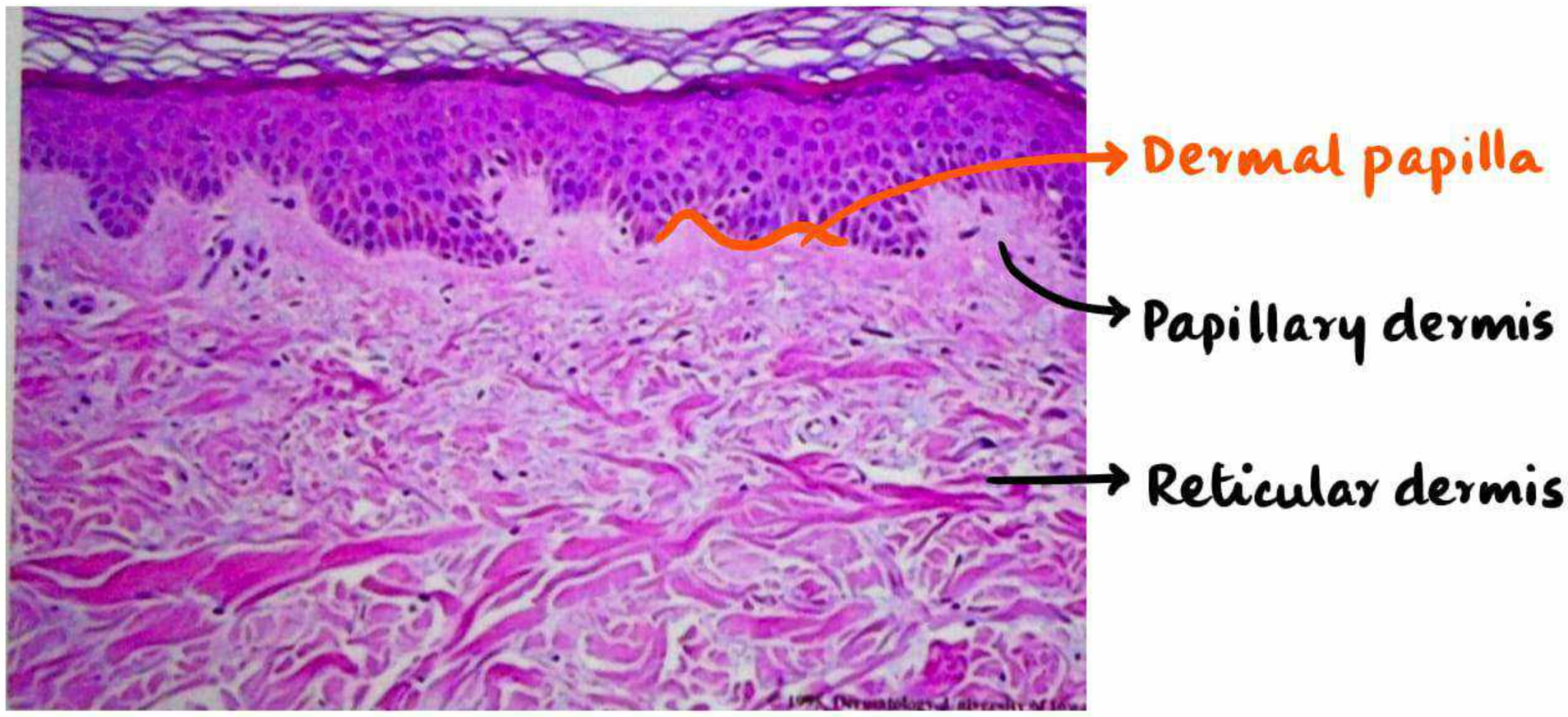


FUNCTIONS OF SKIN

01:11:03

- Barrier
- Lipids
- Sweating
- Temperature regulation
- Touch, pressure sensation
- Insulation.
- Cosmetic
- Hair, Nail
- Odour
- Vit D Synthesis

active space

Vit D Synthesis

- occurs in S.basale and S.spinosum
- Provitamin D_3 —(Sunlight)→ Pre vitamin D_3

↓

Vit. D_3

↓

25-Dihydroxy Vit.D

active space

Skin Lesions

Skin Lesions in Dermatology

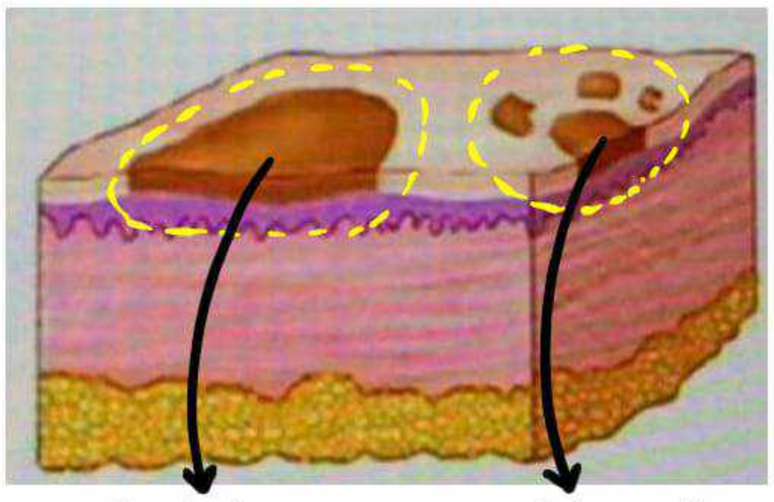
1. Primary : Appear first
2. Secondary : Changes which occur on 1°
3. Special : Characteristic of some specific dermatoses.

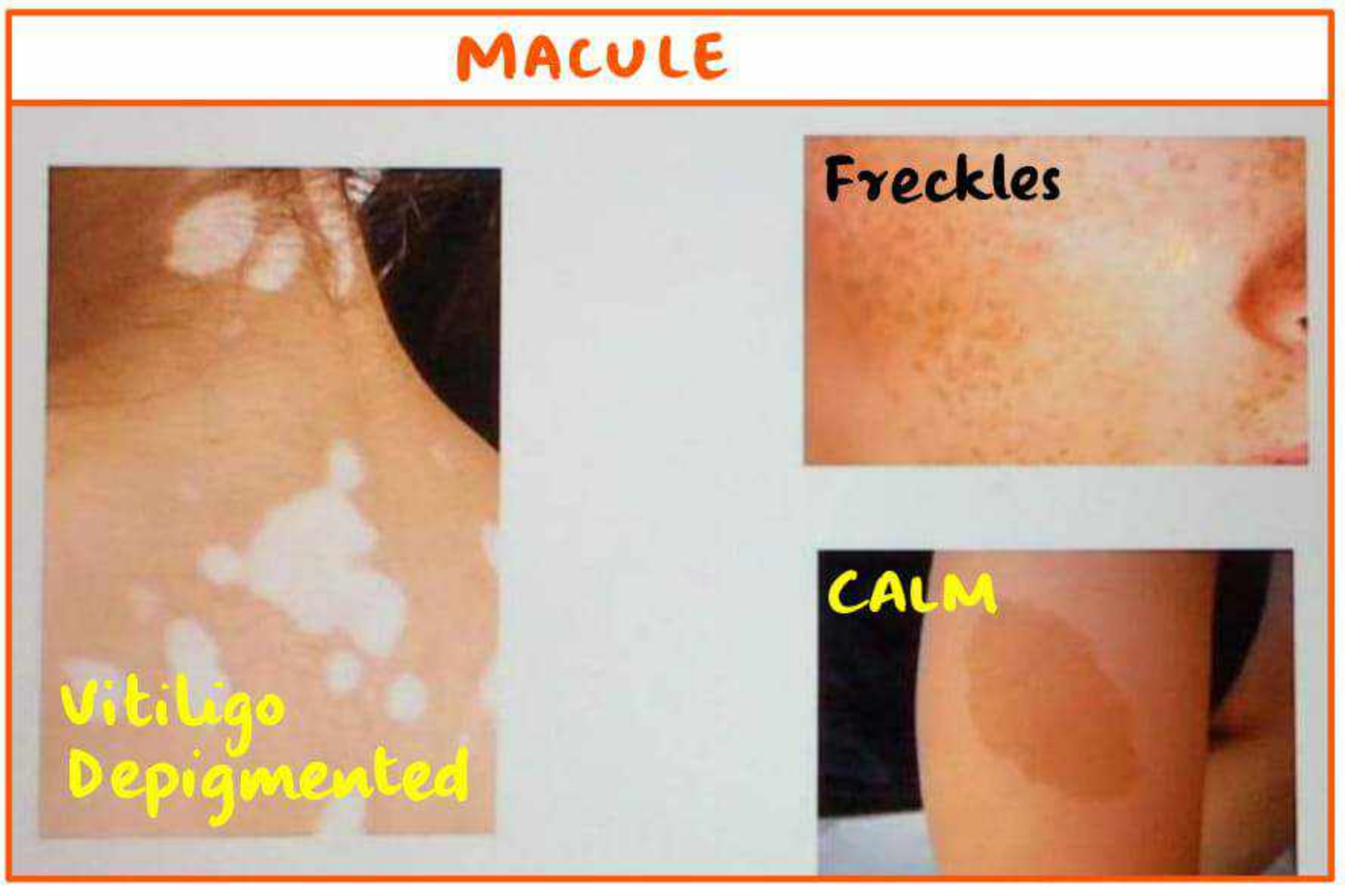
PRIMARY LESIONS

MACULE

- change in skin colour
- Neither depressed, nor raised
- < 0.5 cm in size.

PATCH : size > 0.5cm.





- **Hypopigmented :** ↓ in skin tone (Eg: CALM)
- **Hyperpigmented :** ↑ in skin colour (Eg: Freckles)
- **Depigmented :** Appears as chalky white (Eg. Vitiligo)

PAPULE

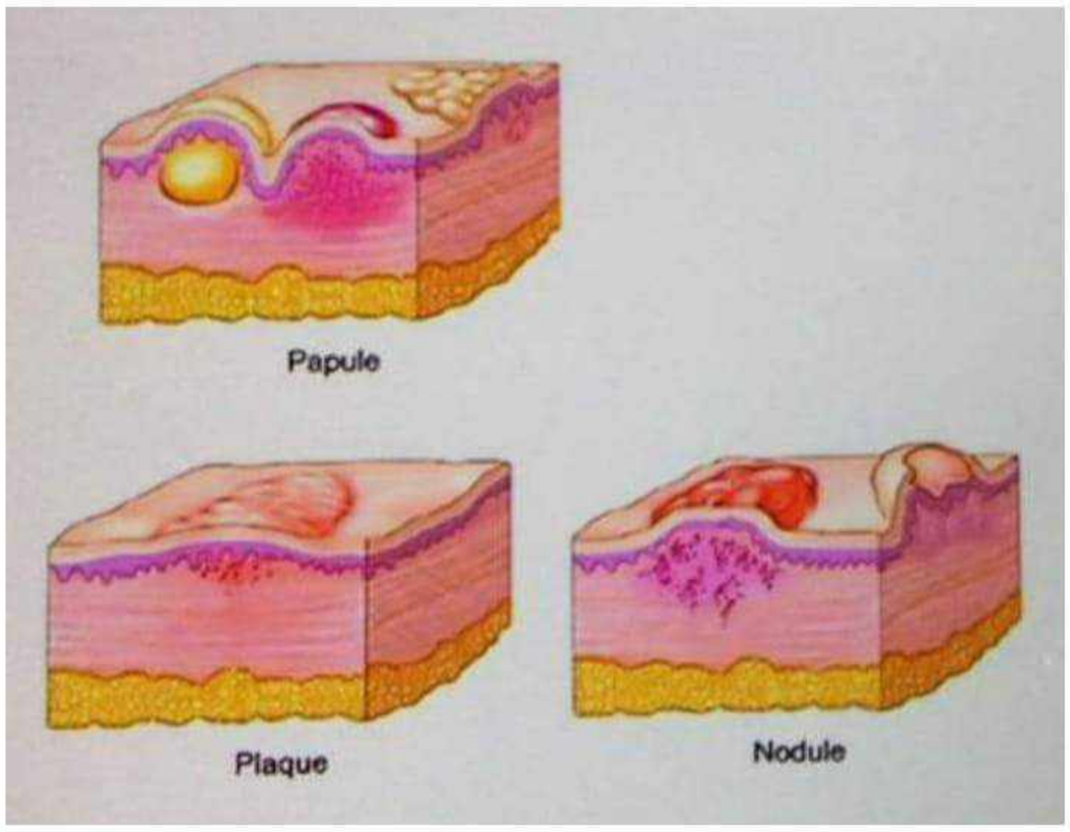
- Circumscribed solid elevated lesions, < 0.5cm

PLAQUE

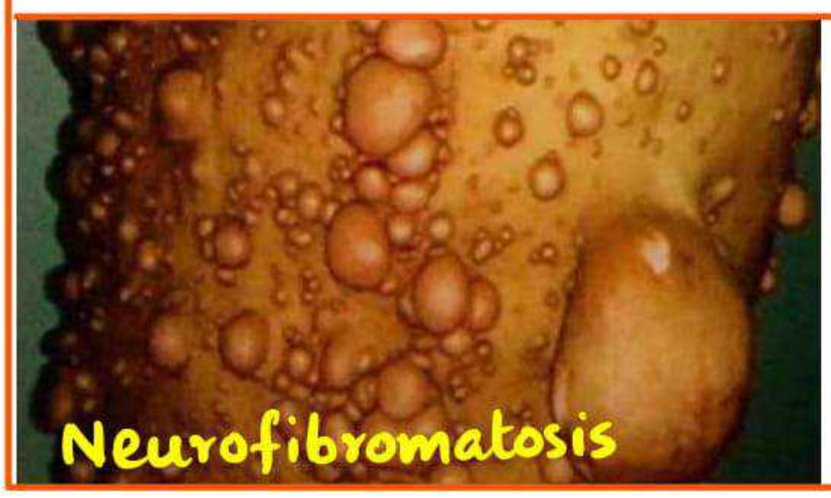
- Circumscribed solid elevated lesions, > 0.5cm
- abnormal skin texture ⊕

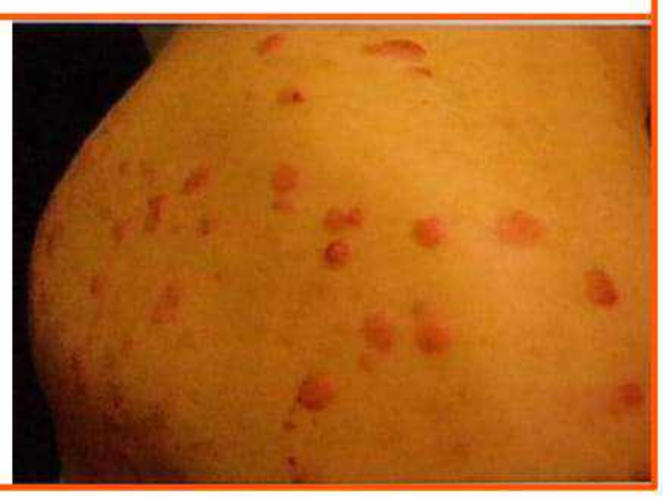
NODULE

- Circumscribed solid elevated lesions, > 0.5cm with depth.
- Better felt than seen.

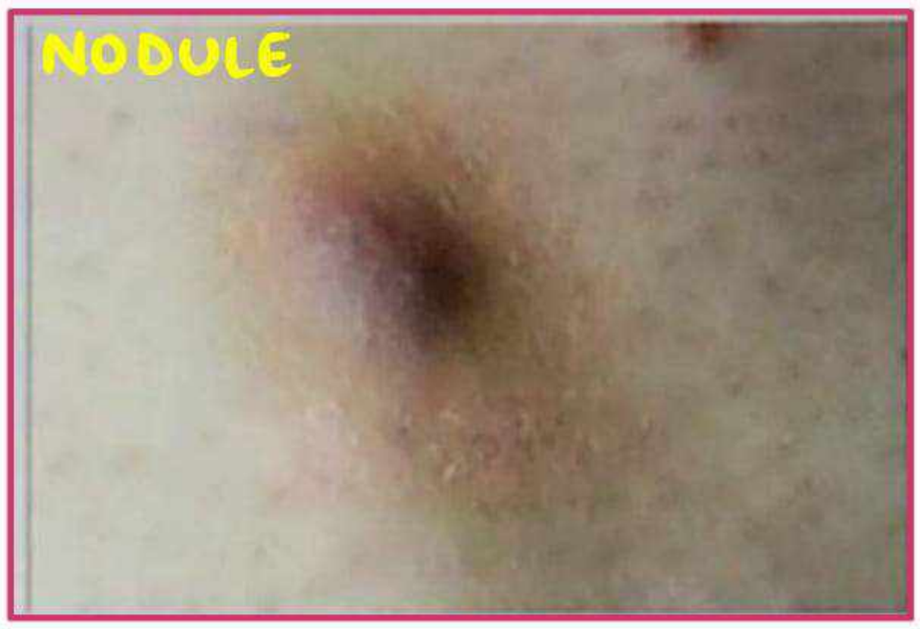


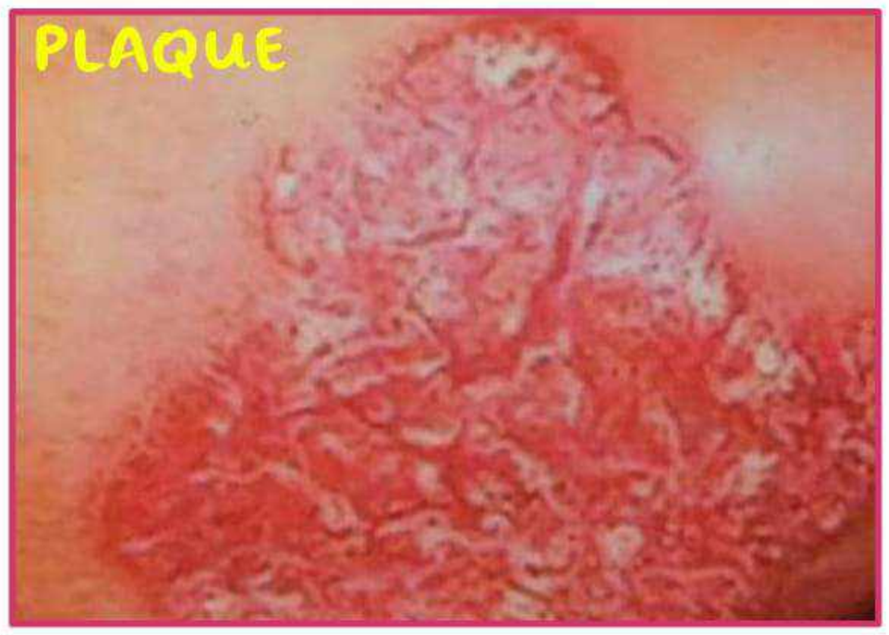
PAPULE





active space

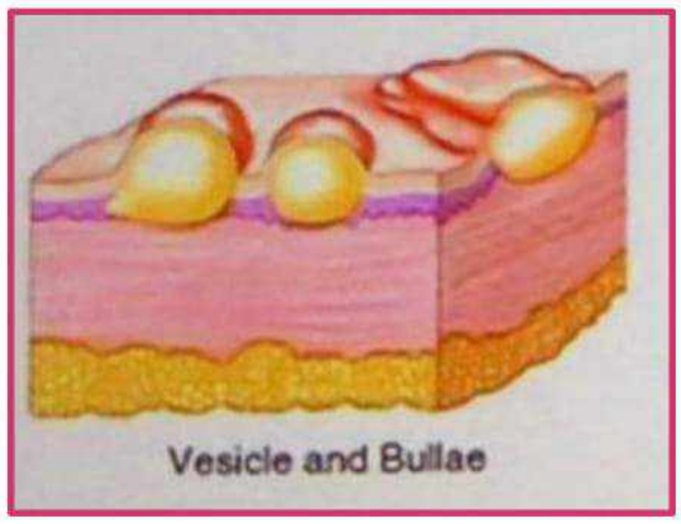


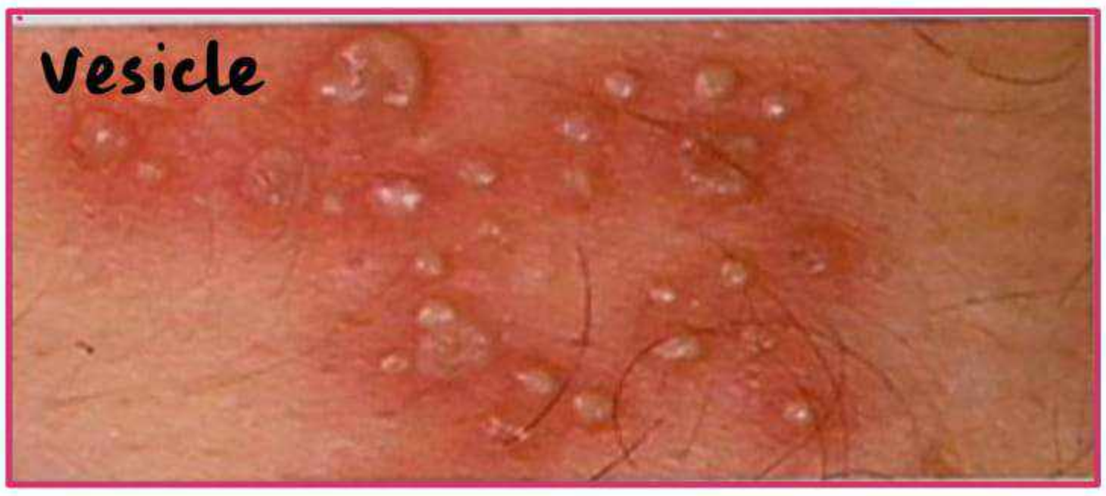


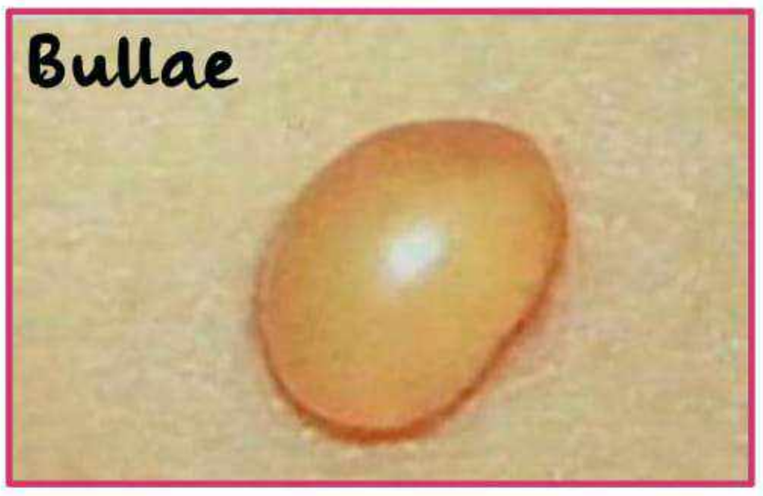
- **Vesicle :** Fluid filled lesions < 0.5 cm
- **Bullae :** Fluid filled lesions > 0.5 cm

seen in : i) Autoimmune Blistering disorders

ii) Herpetic infections


Vesicle and Bullae

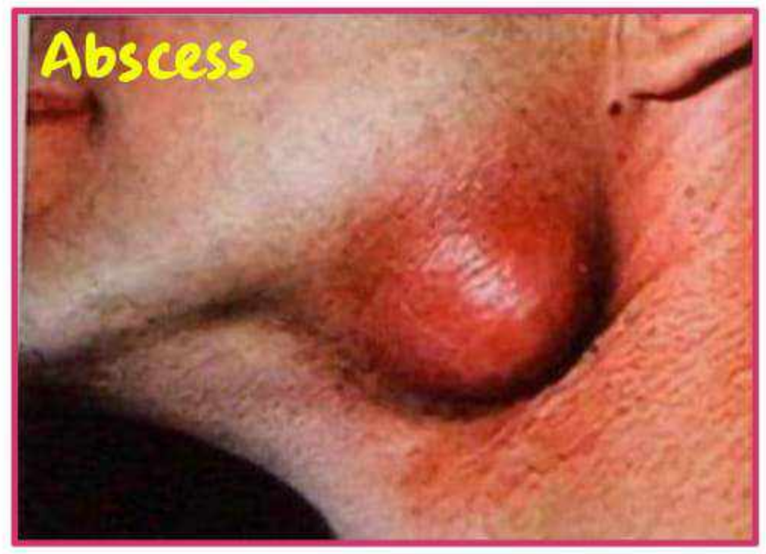


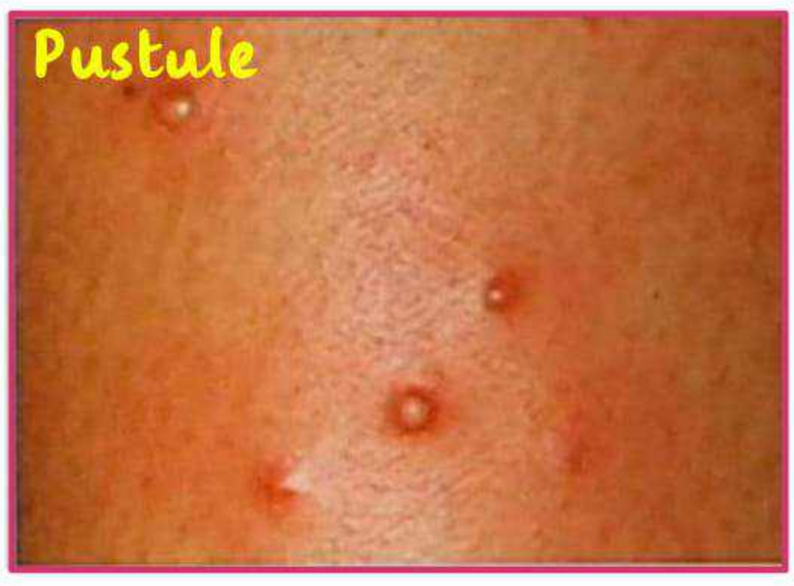


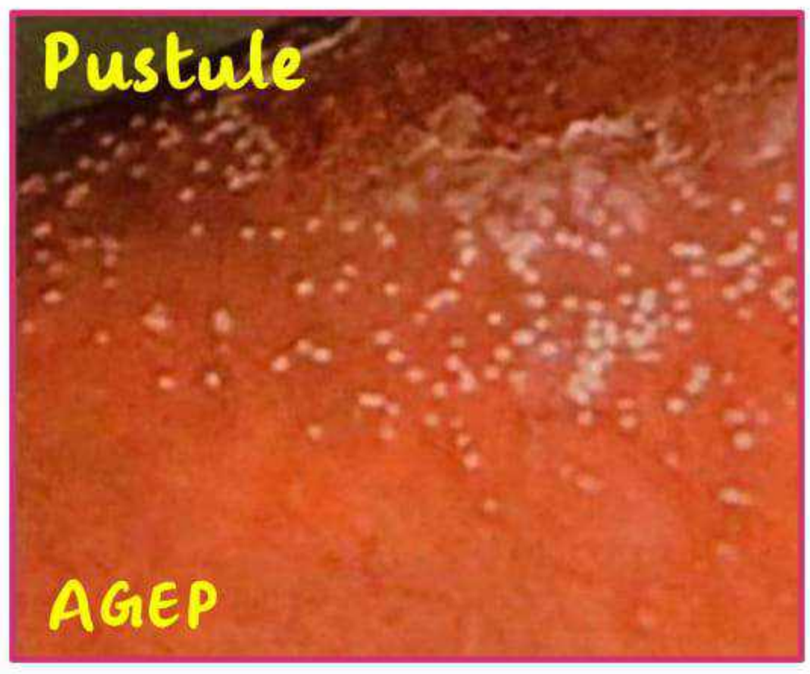
active space

Pus filled lesions

- small → **Pustule** — collection of neutrophils → can be
  - 1°
  - 2°
- Large → **Abscess**

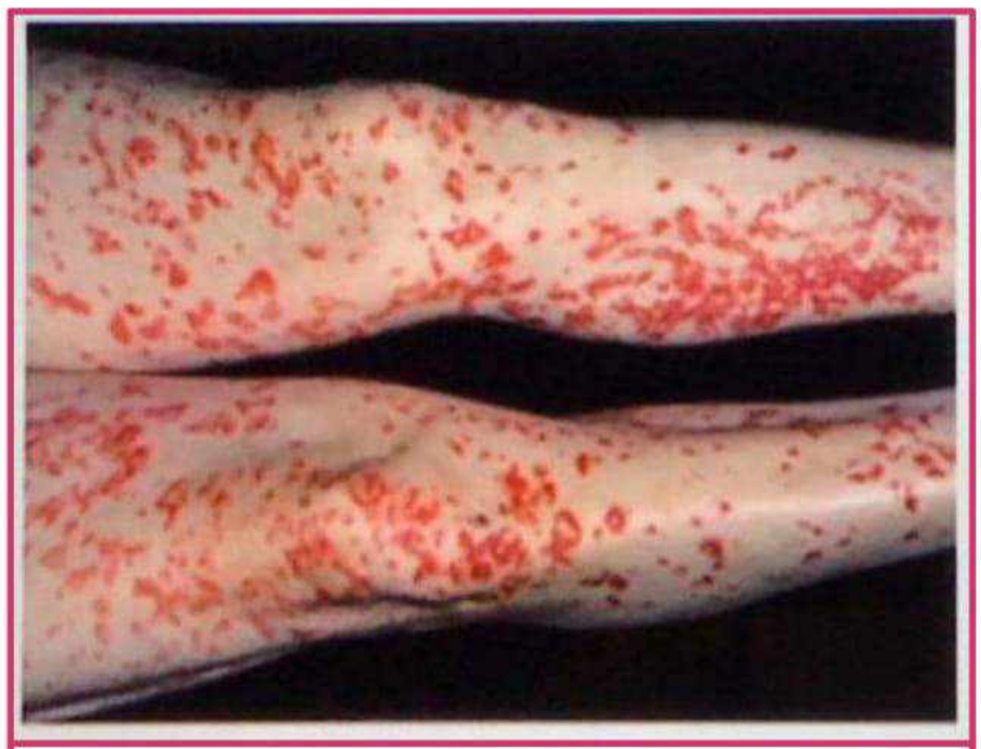




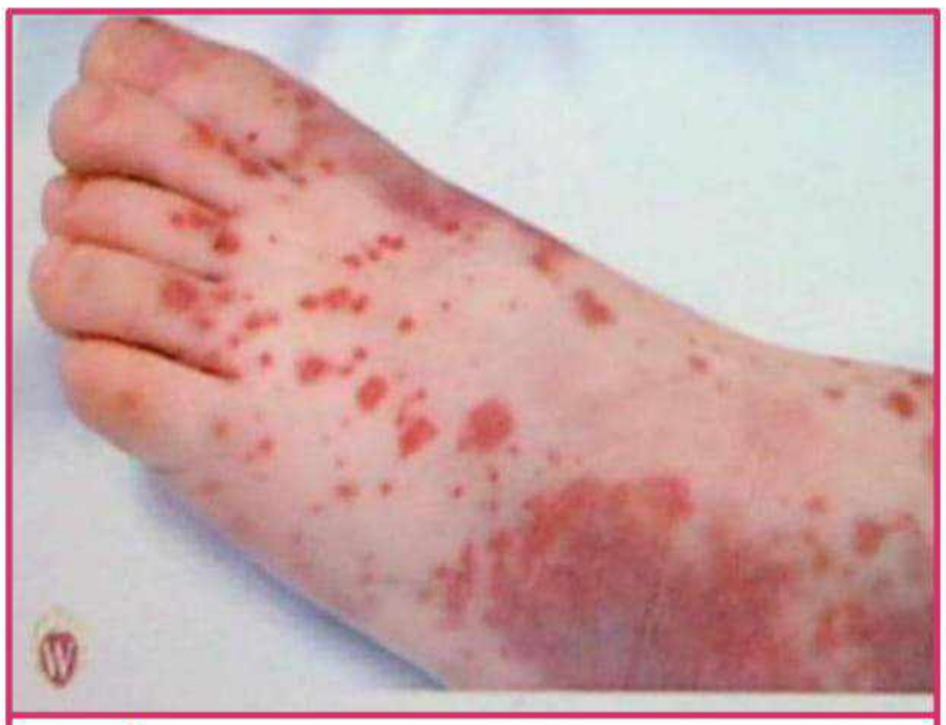


Extravasation of RBC's

Petechiae	Purpura	Ecchymosis
1-2mm	>3mm	1-2cm



Red lesions, non blanchable



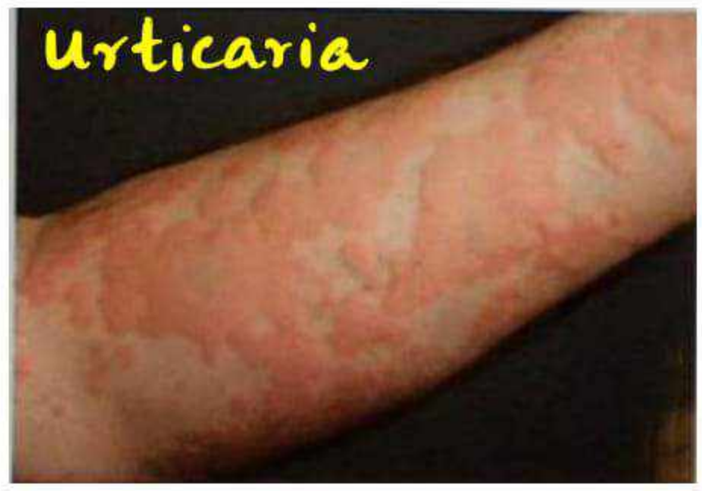
Ecchymosis

- lesions can be seen in
  - Vasculitis : palpable purpura
  - Clotting disorders : non-palpable.

WHEALS AND ERYTHEMA

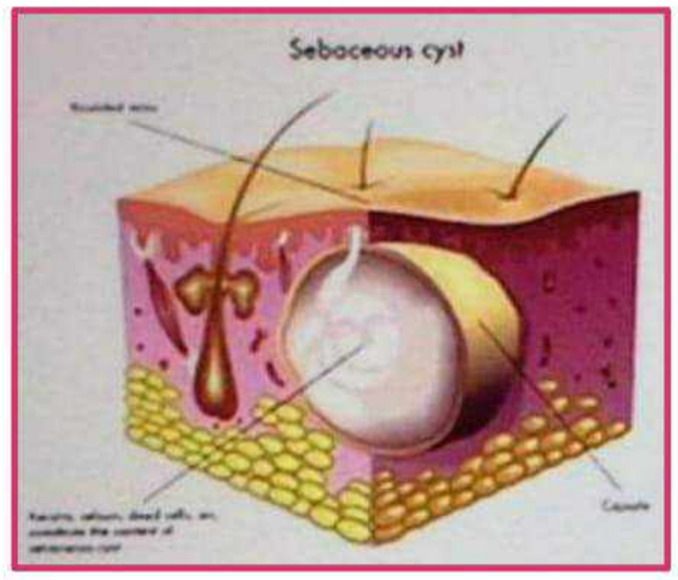
13:15

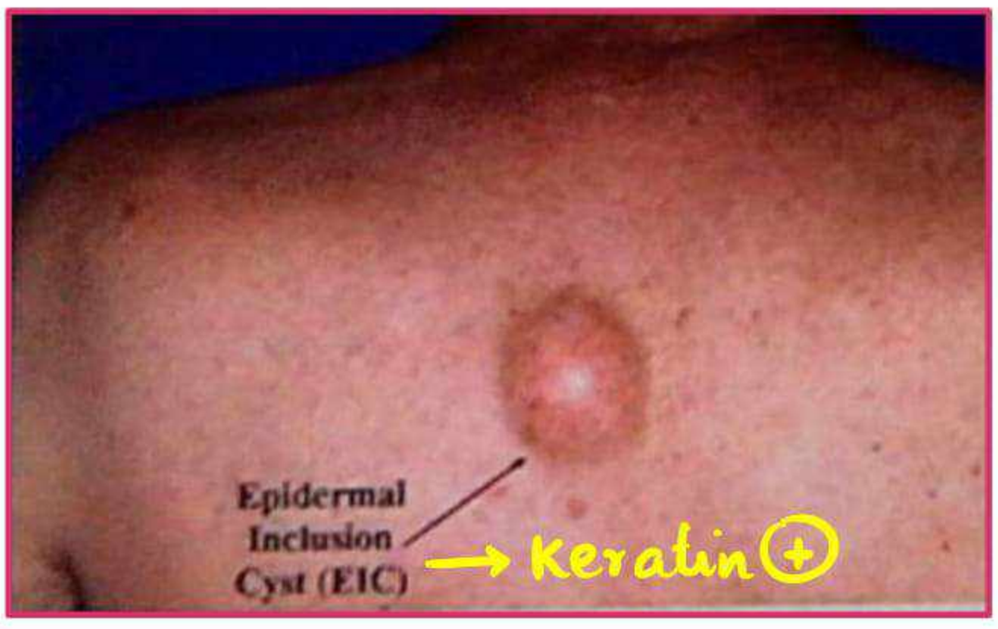
- Wheals :
  - Edematous
  - evanescent → disappears in 24-72hrs
  - erythematous → vasodilation
- Central pallor, wheals and surrounding erythema are seen in urticaria.



CYST

- are Primary lesion
- enclosed cavity lined by lining (epithelium or endothelium)
- filled with fluid or semisolid material
  - semisolid → keratin

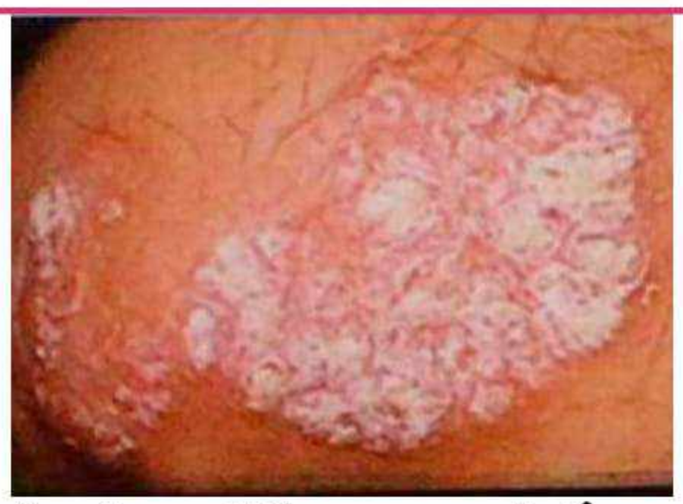




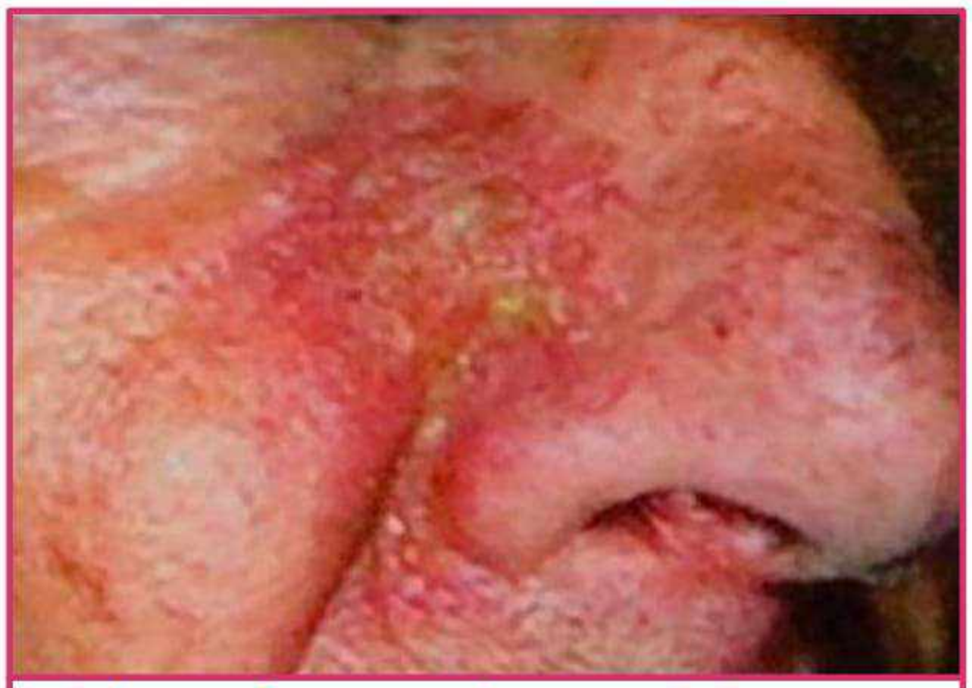
SECONDARY LESIONS

16:37

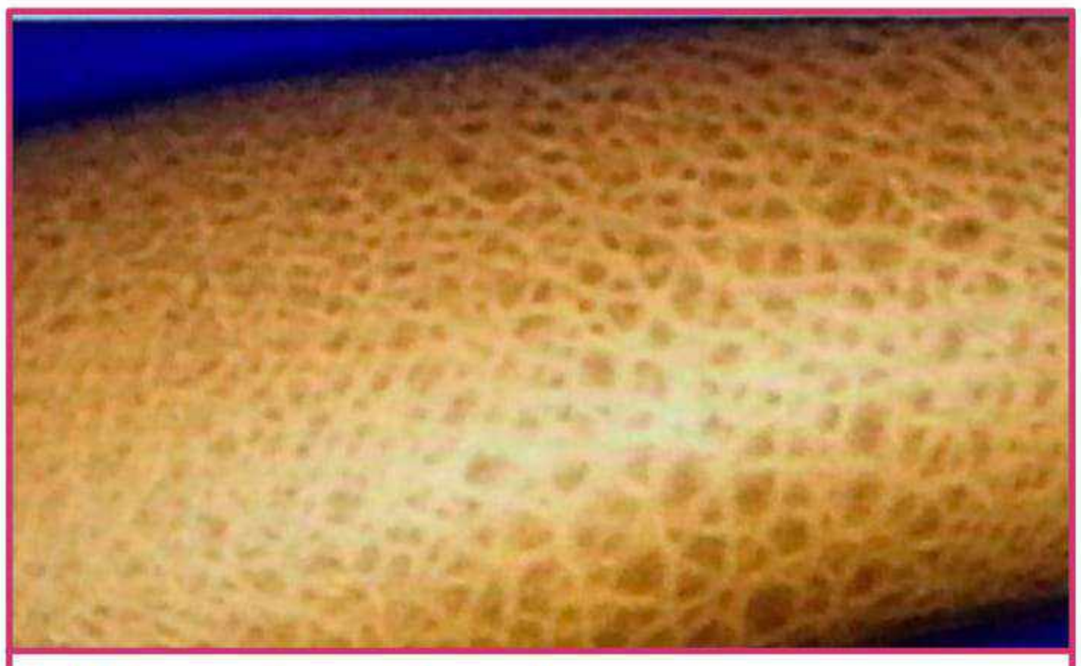
- **SCALE :** visible exfoliation of skin.
  - exfoliation → Stratum corneum.



Psoriasis : Silvery white scales

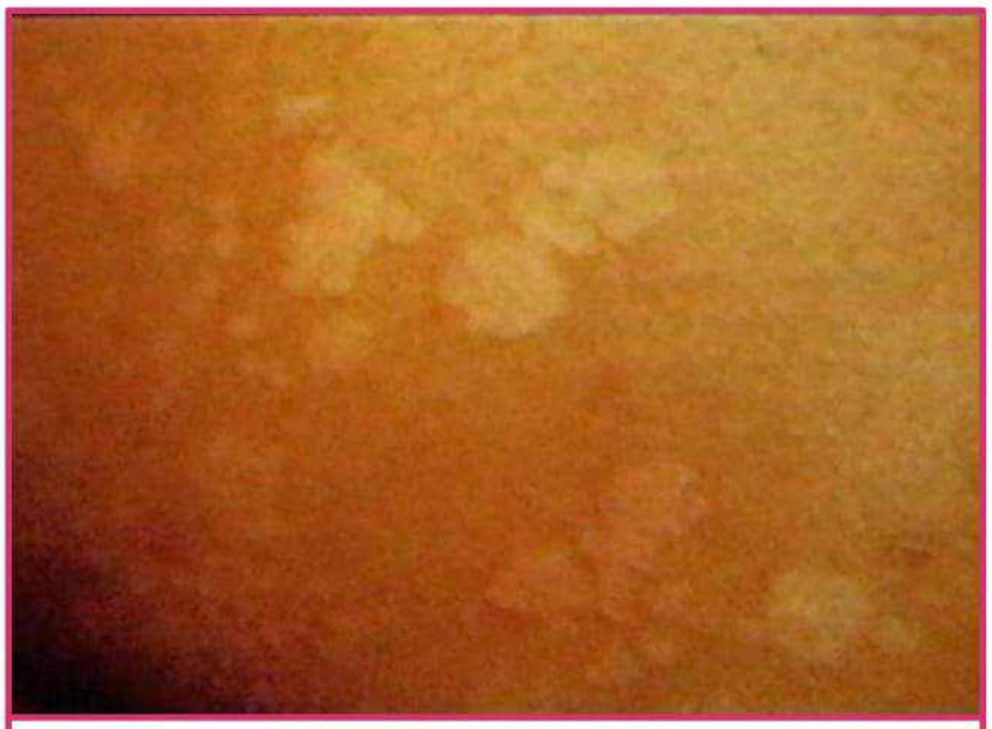


Seborrhoeic dermatitis :
Greasy scales



Icthyosis vulgaris
Fish like scales

Pityriasis rosacea
Collarette scales

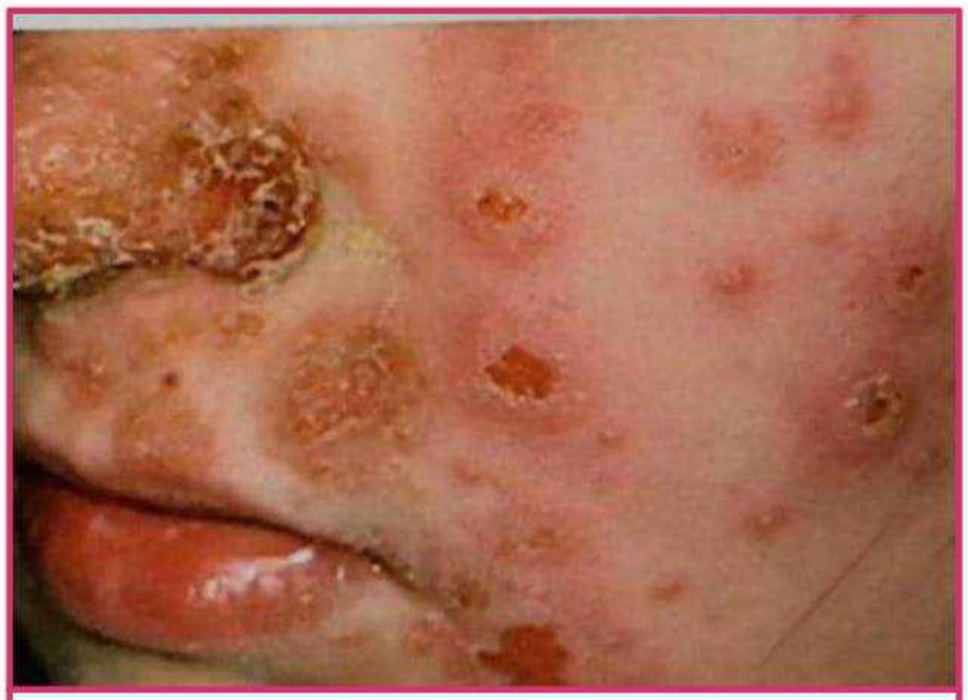


Pityriasis versicolor.
Furfuraceous brown scales

CRUST :

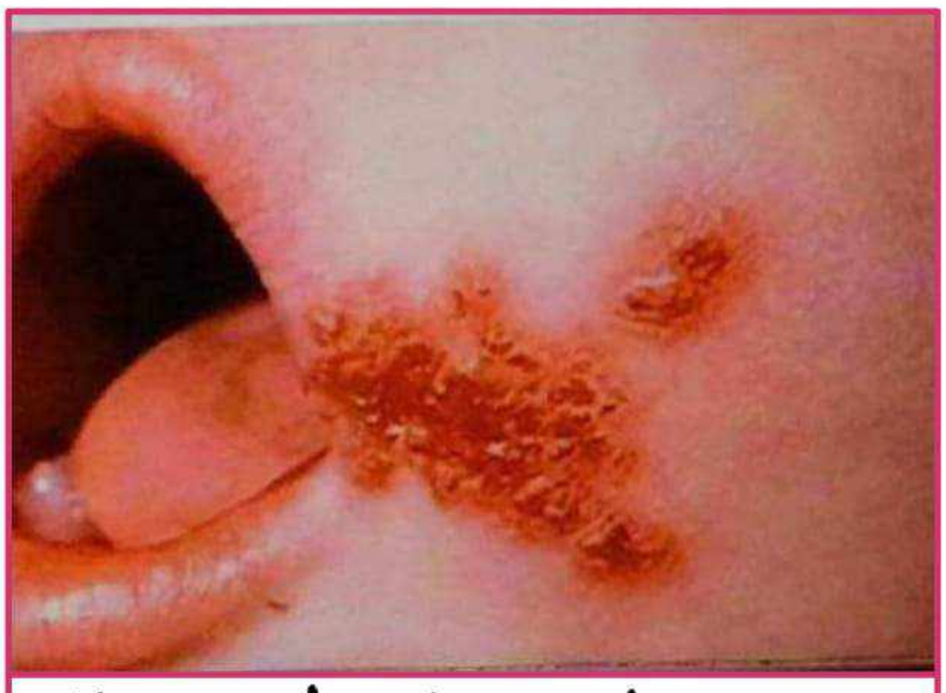
- dried up exudate
- exudate can be i) pus ii) Blood iii) serum
- Superficial.

active space



Honey coloured crusts

Non-Bullous Impetigo

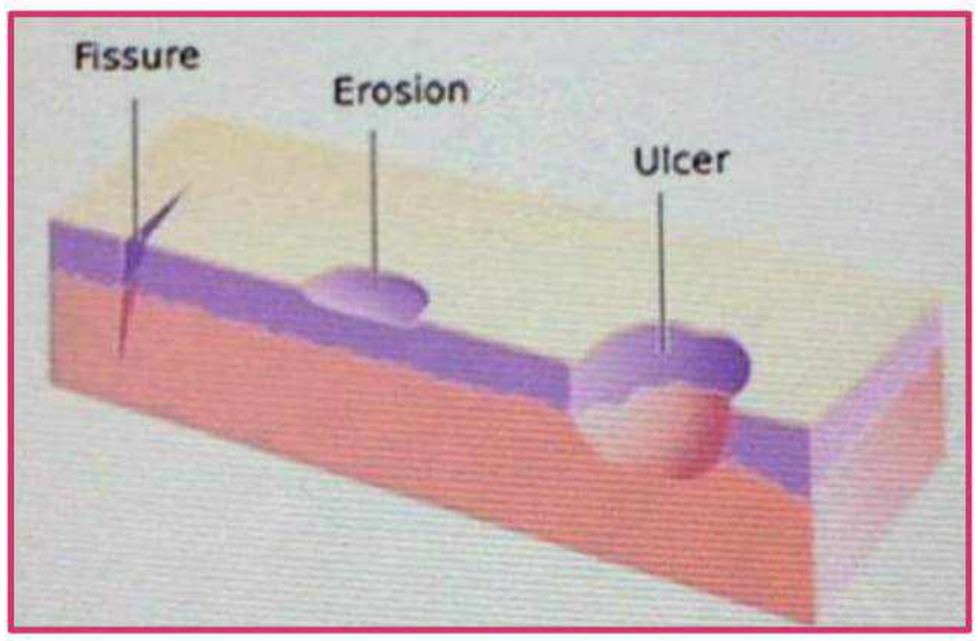


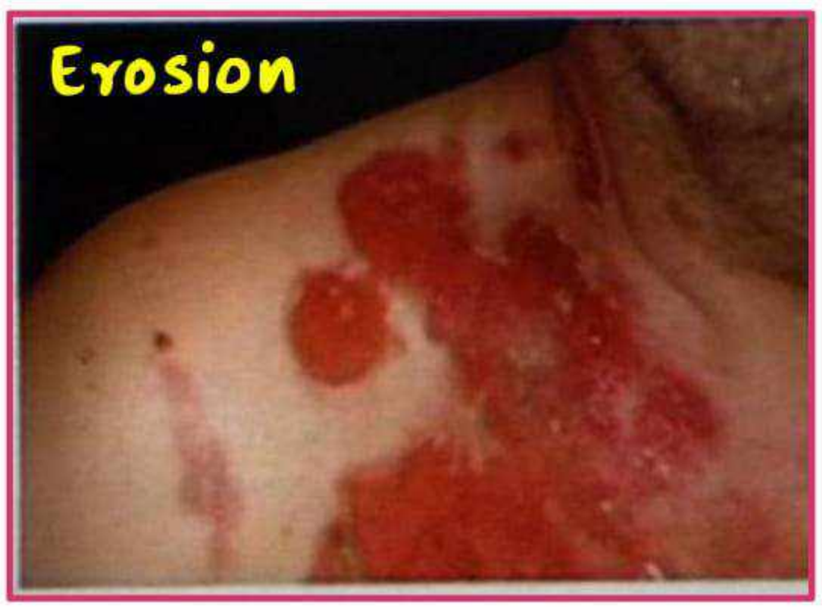
Hemorrhagic crust

Herpes

Erosion:

- Raw moist areas formed by partial or complete loss of epidermis.
- Superficial, doesnot scar. (no involvement of dermis)



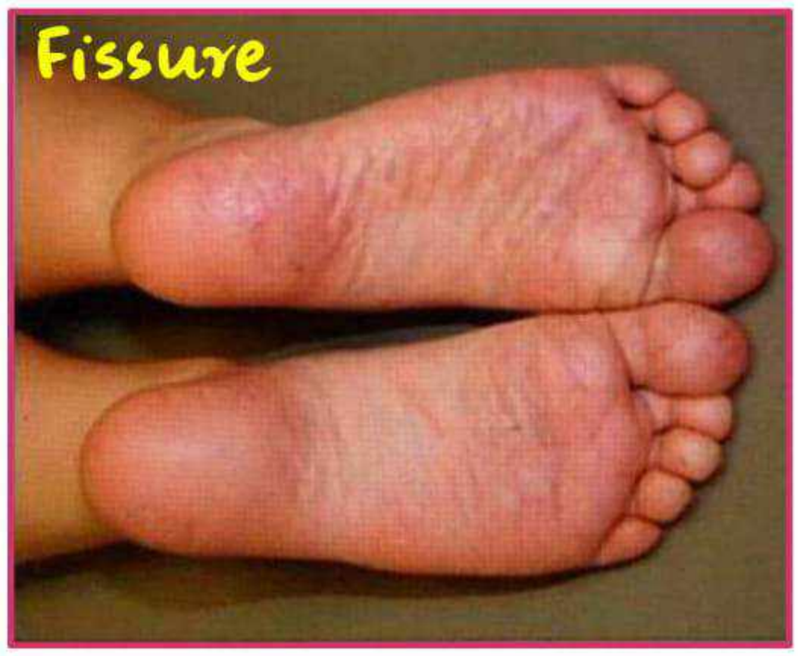


Ulcer :

- formed by loss of epidermis, dermis, subcutaneous tissue.
- heals with scarring usually.
- ulcer has margin, depth, base.

Fissure :

- linear crack or discontinuity
- it may be superficial, deep
- (MC) seen in feet of patient

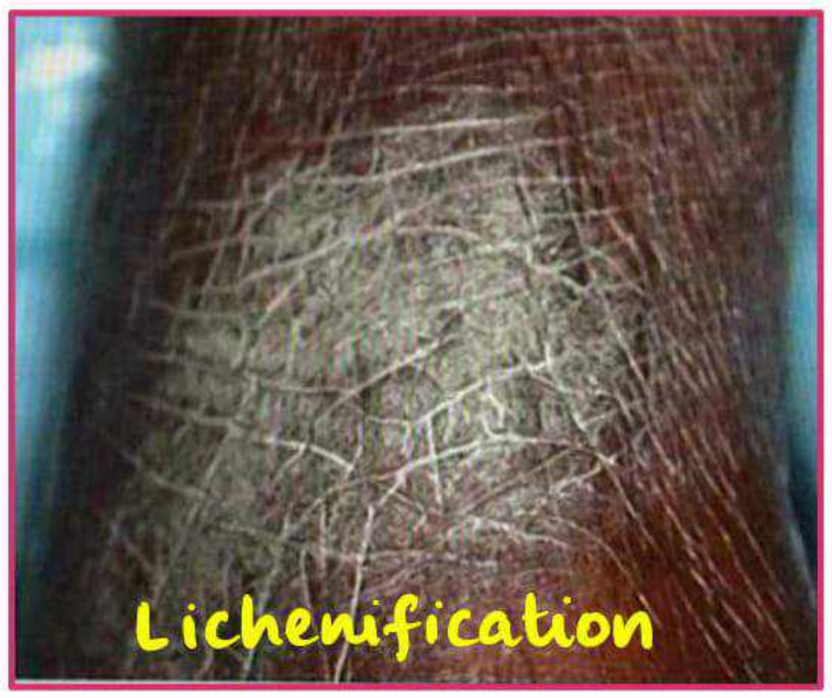


Excoriations

- Surface excavations in the stratum corneum due to scratching.

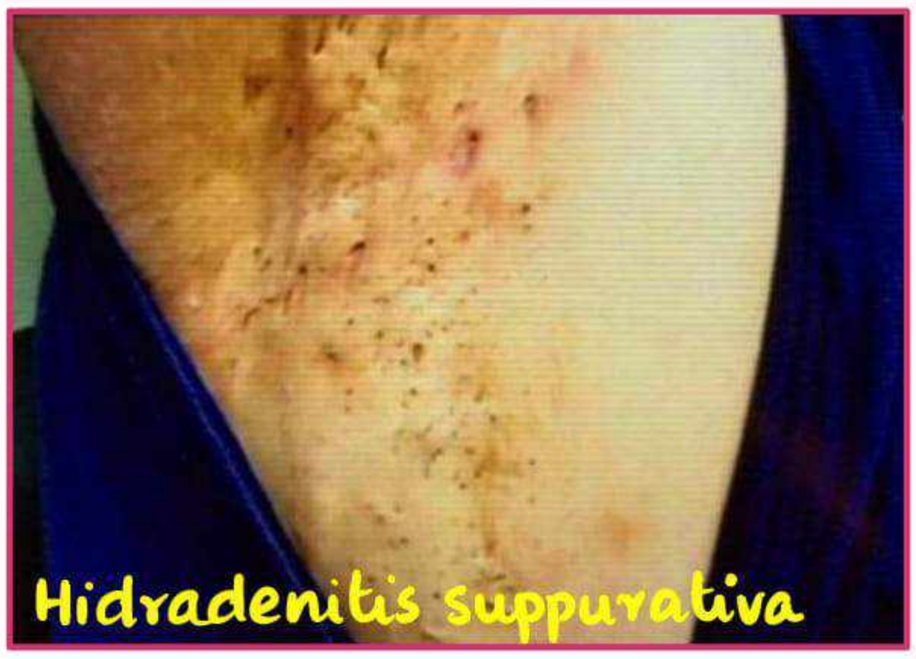
Lichenification

- due to prolonged scratching
- chronic eczema
- characterised by
  - i) Hyperpigmentation
  - ii) Increased skin markings
  - iii) Thickening of skin
- On Histopath : Acanthosis



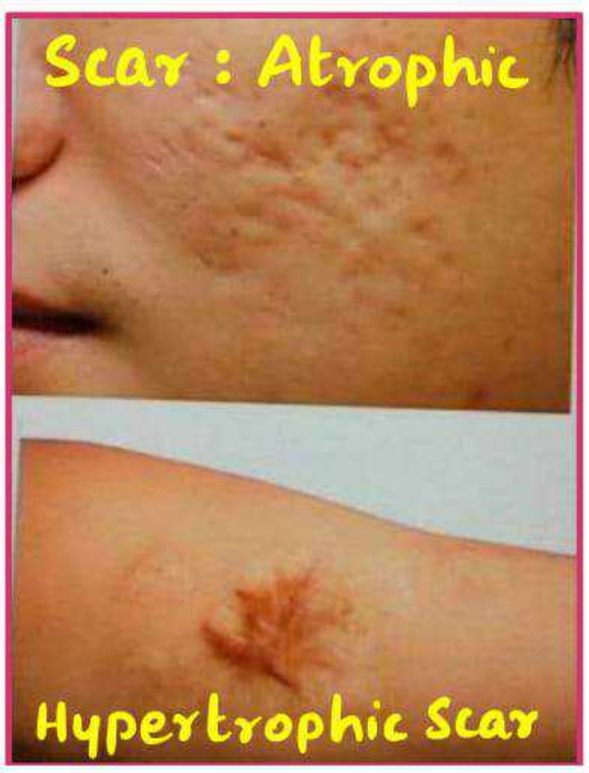
Sinus :

- Blind tract which connects skin to a deeper cavity.
- filled with pus, serum
- Exudate out in the skin

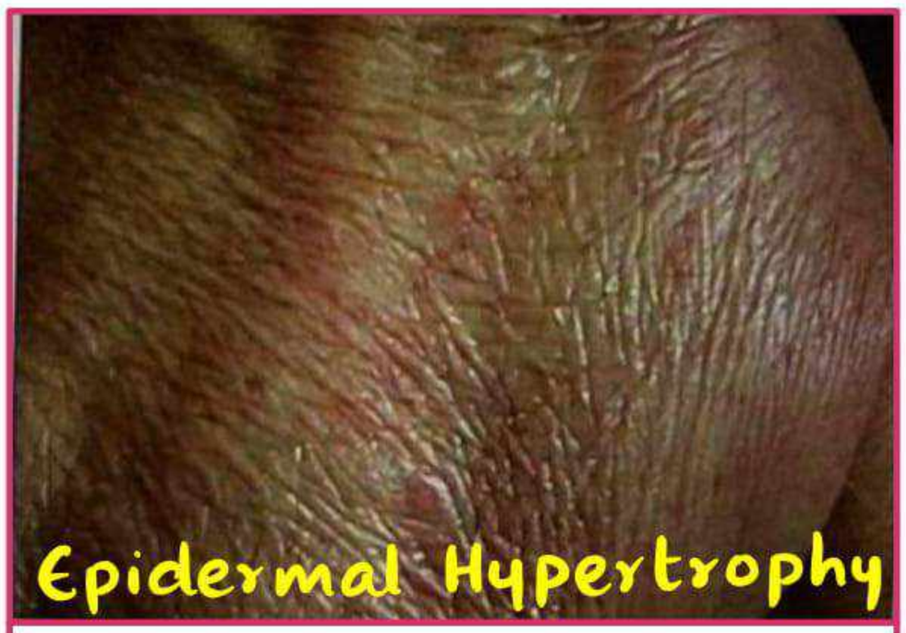


Scar :

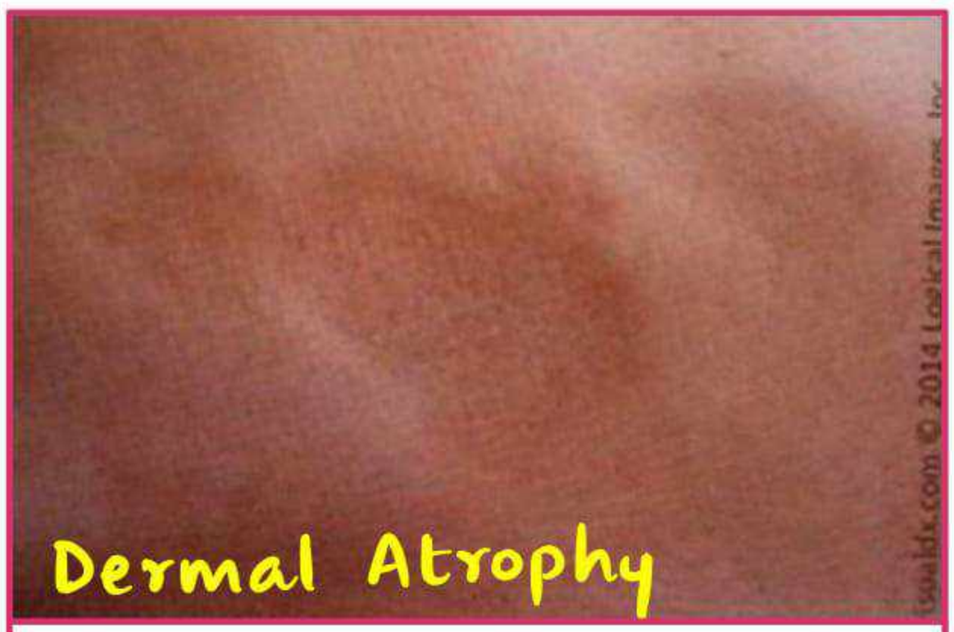
- Abnormal proliferation of fibrous tissue which replaces the normal collagen.



Atrophy: loss of any skin structure.



- glistening, wrinkled



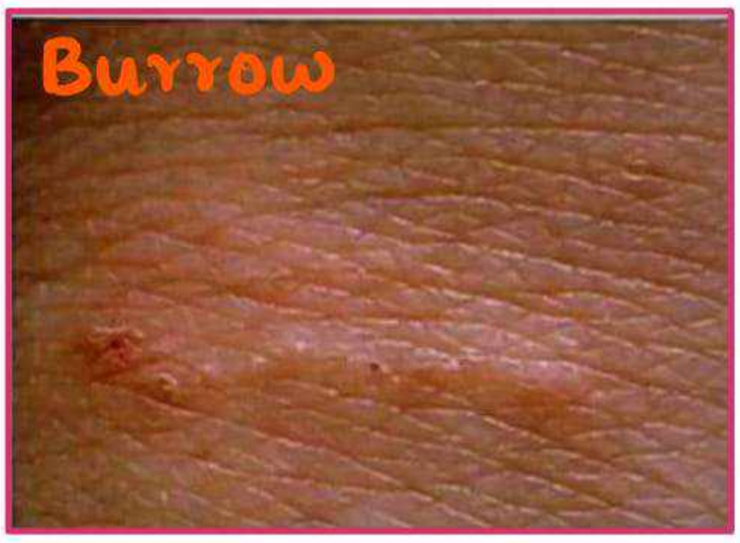
Depression with normal overlying skin.

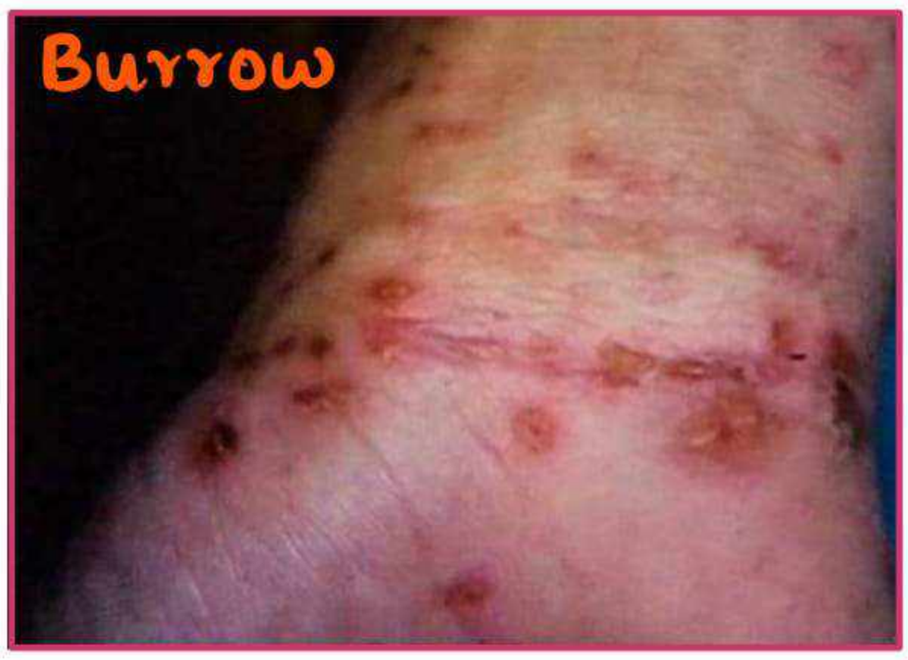
SPECIAL LESIONS

29:15

Burrow:

- Thin wavy tracts laid down by mites into the **stratum corneum**
- seen in **Scabies**
- Common sites:
  - wrist
  - flexures
  - Fingers
  - Umbilicus
- Mite lays eggs at the end of tract

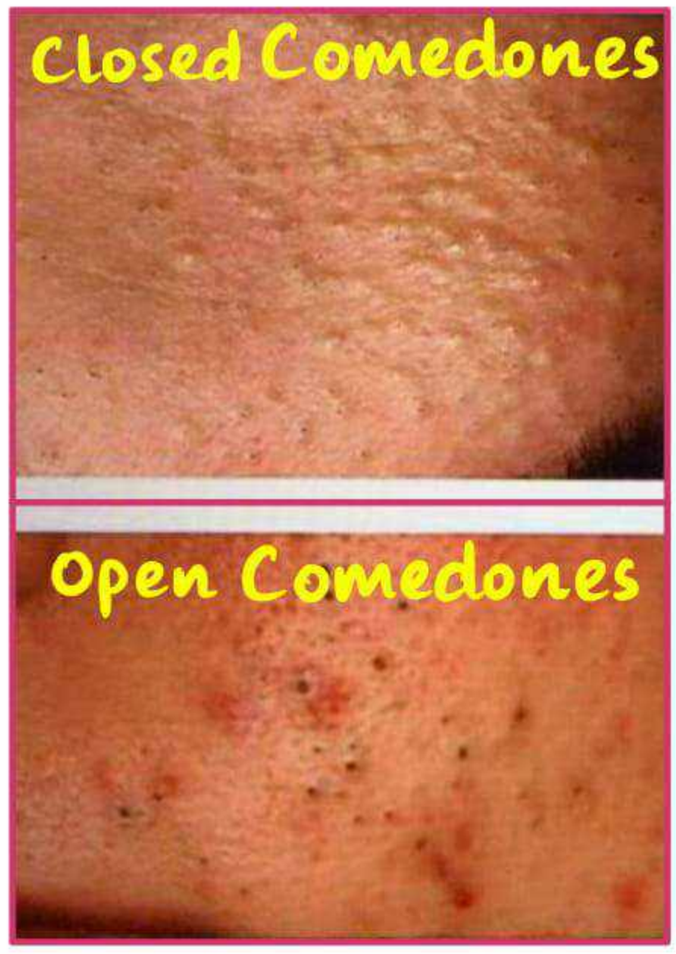




active space

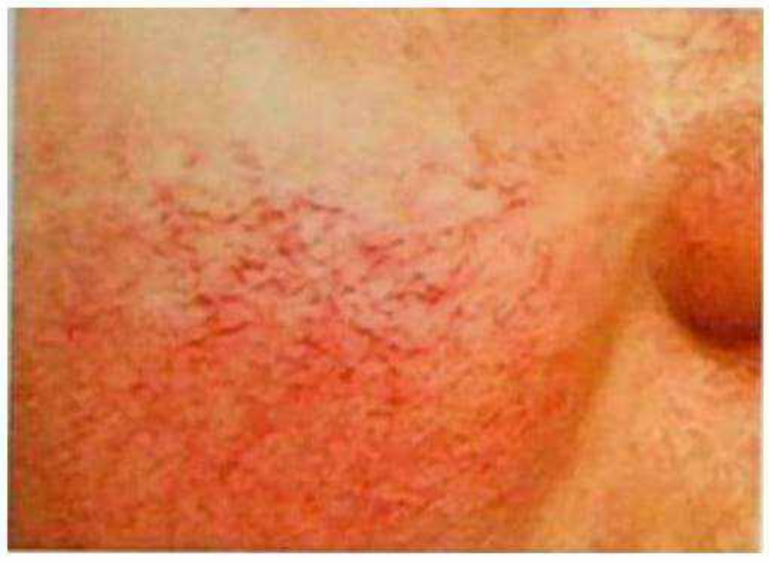
Comedones :

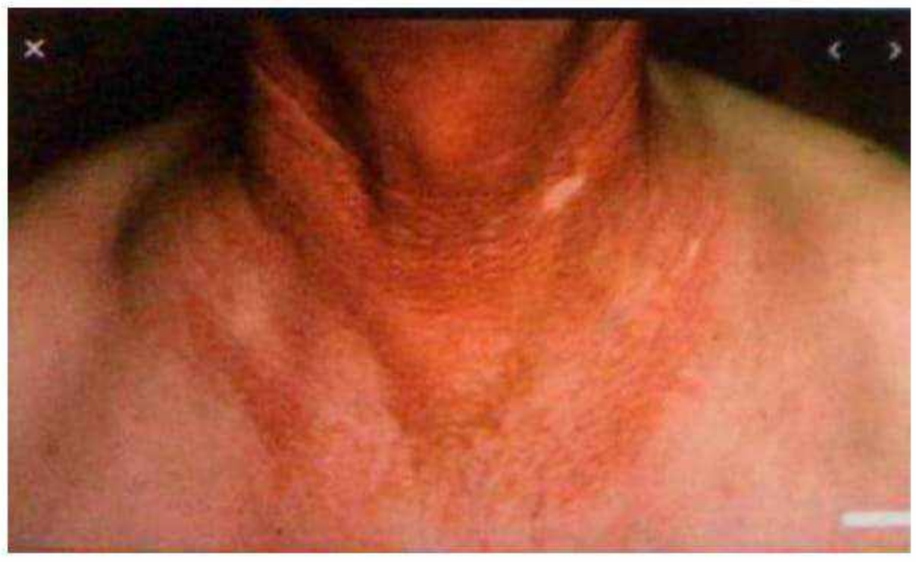
- seen in Acne
- Dilated pilosebaceous ducts filled with keratin
- can be
  - i) Open - keratin turns black due to oxidation (Black comedomes)
  - ii) Closed - appear as white.



Telangiectasias :

- visible dilatation of dermal capillaries.
- persistent and non-blanchable.





Poikiloderma :

- Telangiectasias, skin pigmentation, atrophy.

active space

Sclerosis :

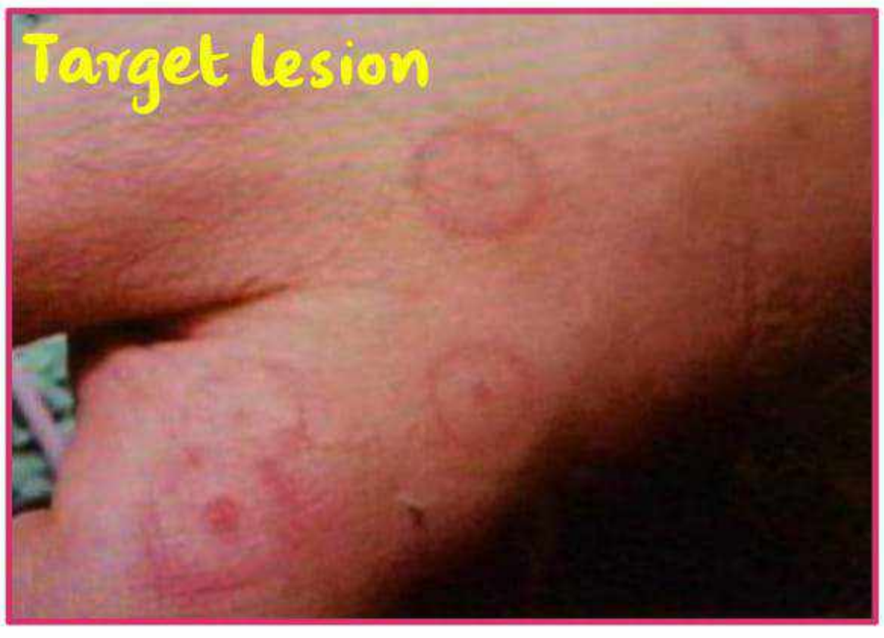
- seen in Systemic sclerosis, Scleroderma.
- Bound down appearance, indurated and stretched.

Milia :

- Cyst which has lamellated keratin
- Appears as pin-point white lesions

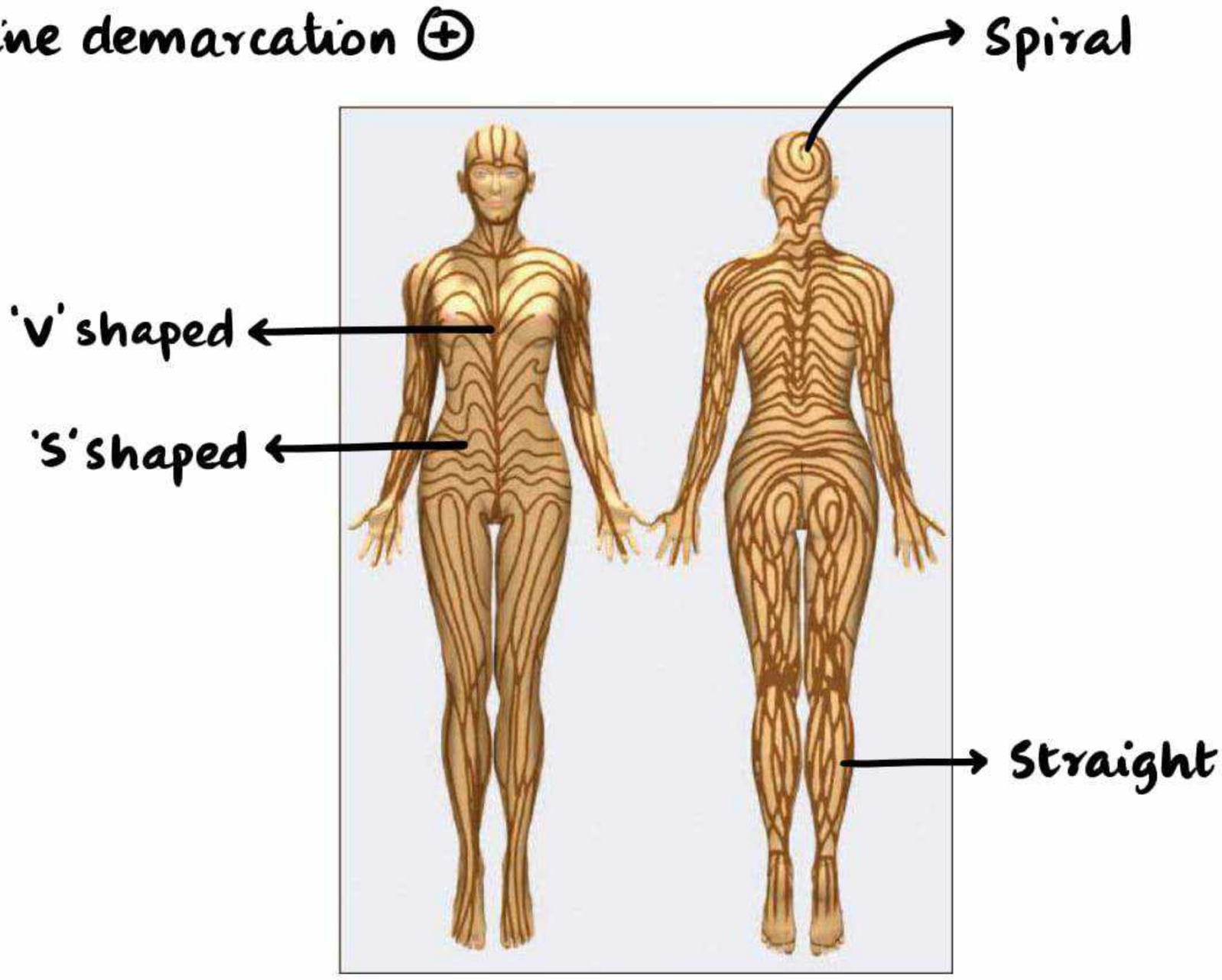
Target lesion :

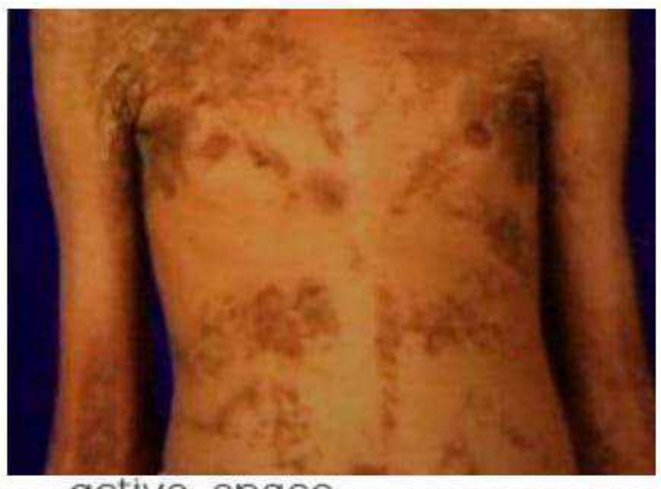
- it has 3 zones :
  - i) Central zone of purpura (Bluish)
  - ii) Zone of Edema
  - iii) Zone of erythema
- seen in Erythema multiforme.



BLASCHKO'S LINES

- Lines of embryonic development
- Keratinocyte travel to skin through these lines.
- Constant
- Midline demarcation ⊕





Dermatoses

Congenital

- Verucous Epidermal Nevus
- Hypomelanosis of Ito

Acquired

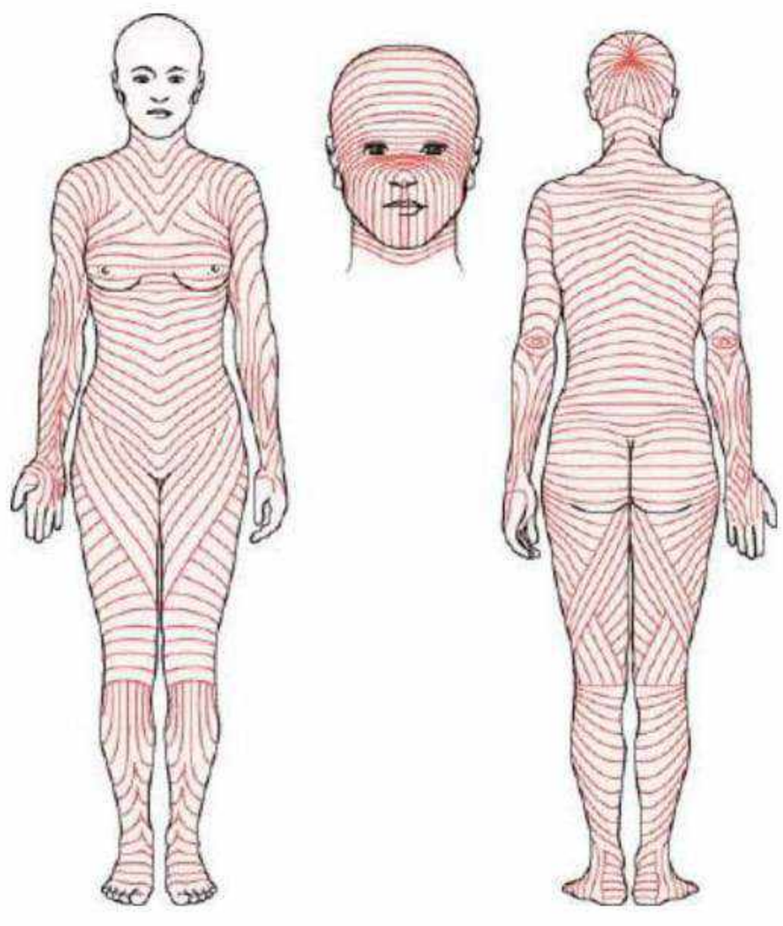
- Lichen planus
- Psoriasis

Genodermatoses.

- Incontinentia pigmenti

LANGER'S LINES

- Relaxed skin tension lines
- Orientation of collagen bundles and muscle fibres in the skin.
- Parallel to collagen bundles
- Important in surgical incisions and forensics
- Not constant

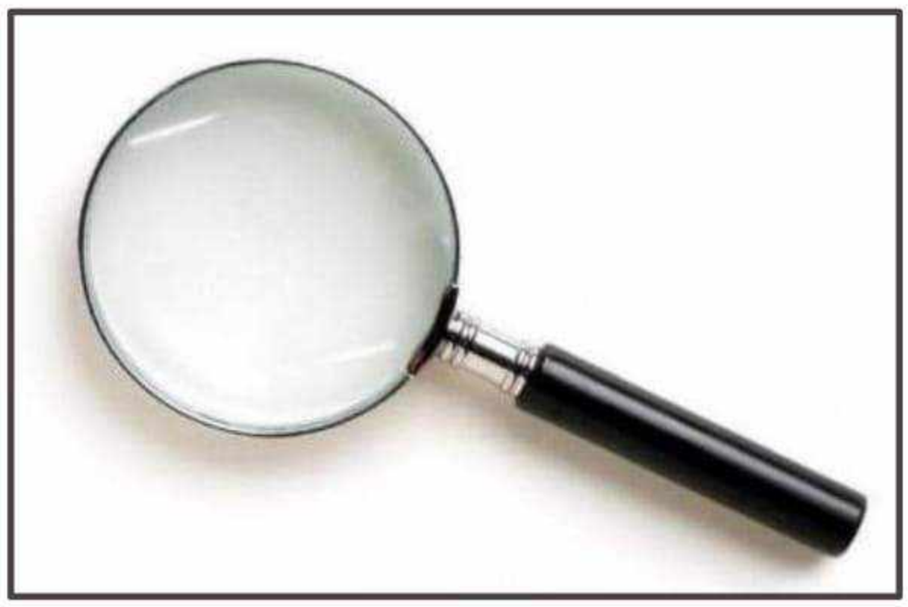


Bedside Tests

Hand Lens :

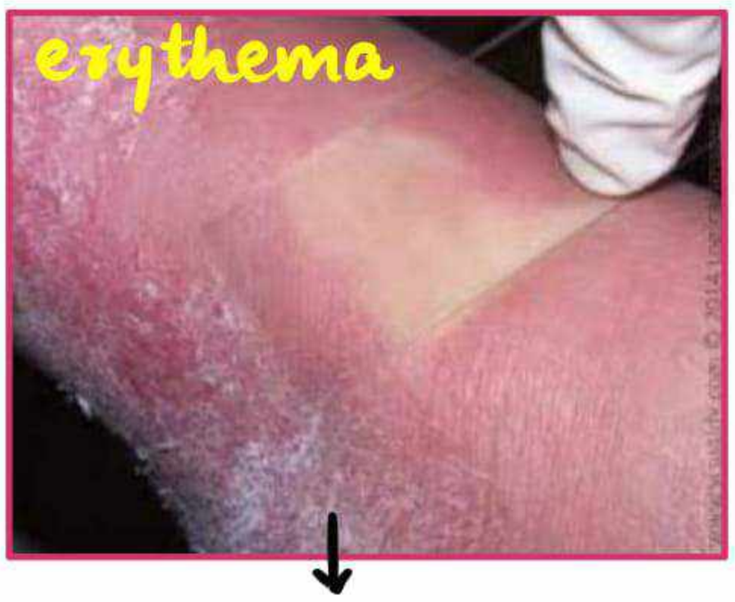
- Magnification 5-7 Times
- Convex glass.
- Helps to identify :

  Wickham's striae in Lichen planus.



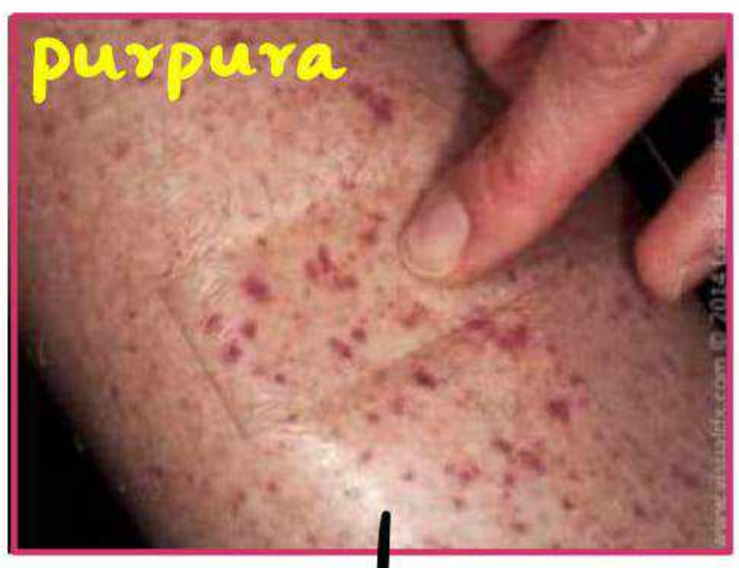
Diascopy :

- used to differentiate erythema and purpura
- Pressing glass slide over lesion → Diascopy



↓

does blanch on diascopy



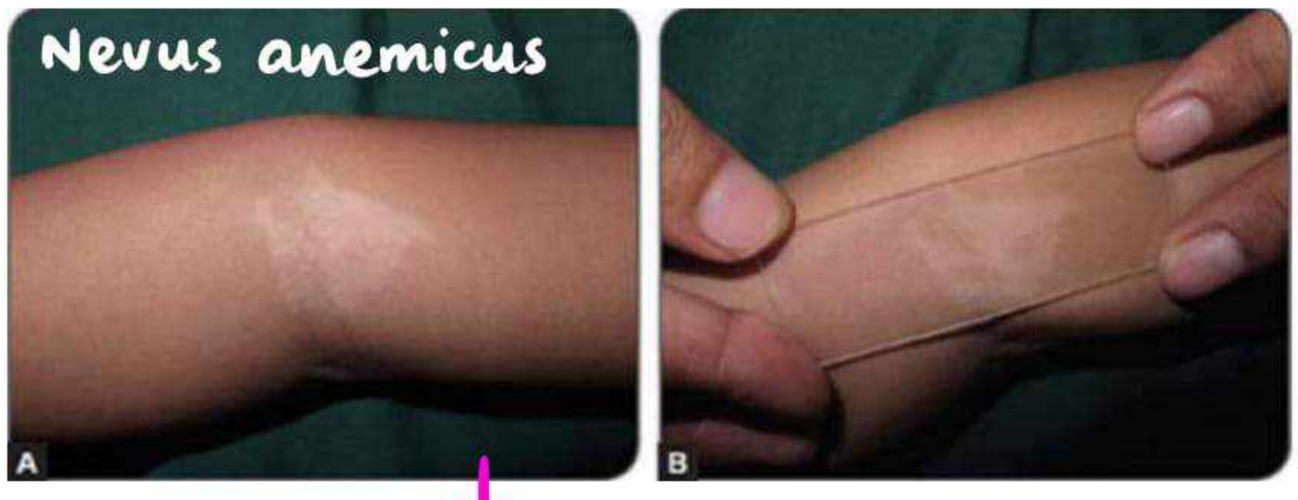
↓

doesnot blanch on Diascopy

- used to differentiate between Nevus anemicus and Depigmentosus.

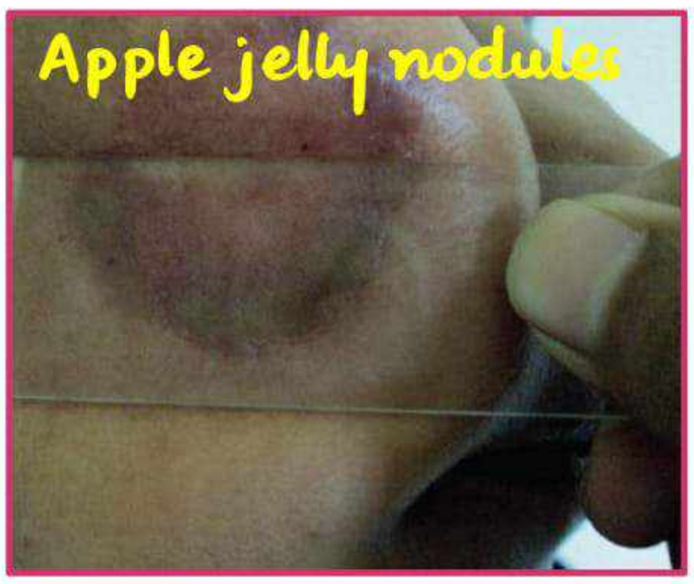
- appear as hypopigmented area.

actual absence of melanocytes



localised vasoconstriction

- used to look for Apple jelly nodules in Lupus vulgaris.



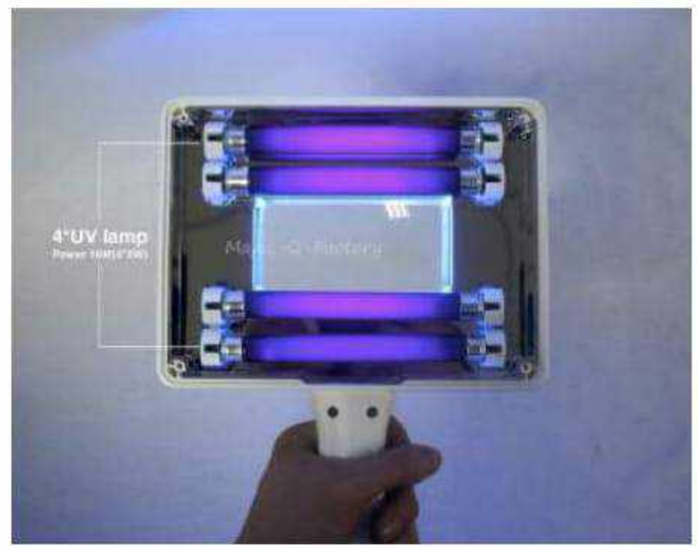
- also used in sarcoidosis.

WOOD'S LAMP

05:10

- low output mercury lamp.
- Filter → Barium silicate and Nickel oxide
- Wavelength - 320nm - 450nm

- Peak → 365nm
- Flourescence
- always done in a dark room
- switch on 1-2 min prior
- placed 4-6 inches away from lesion.

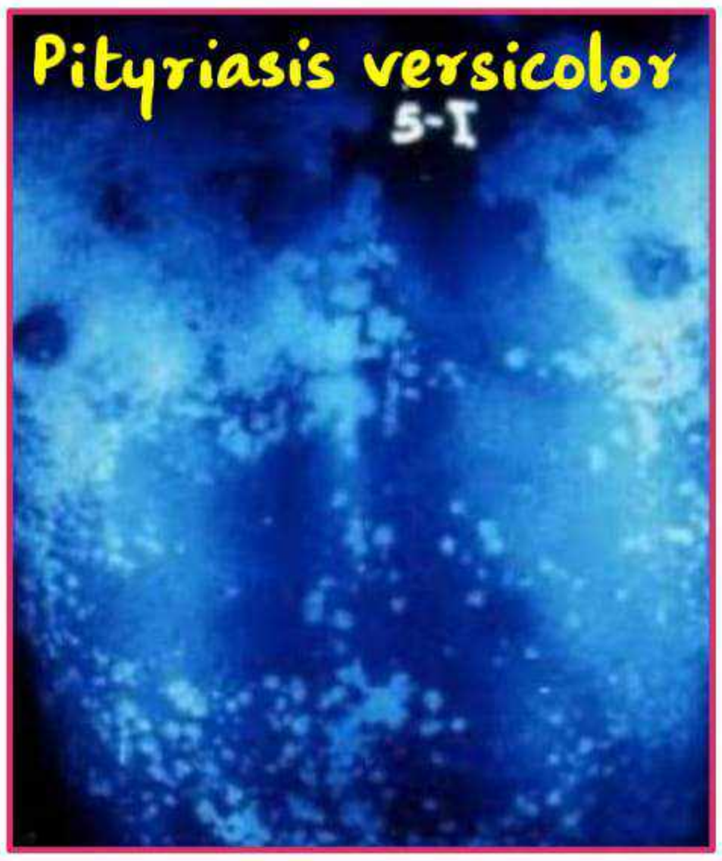


- used in :

i) Tinea capitis.

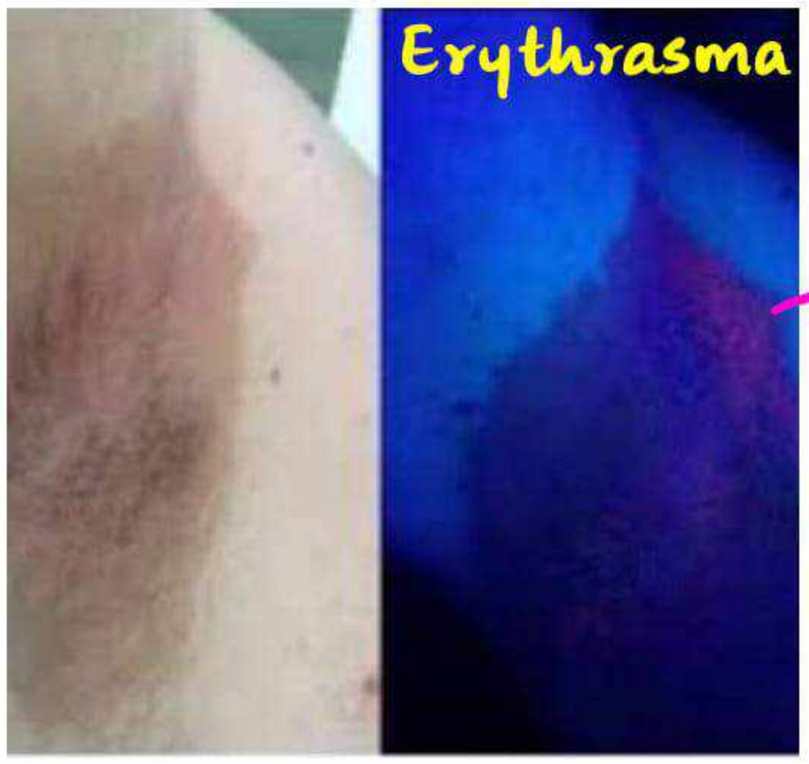
- Microsporum → Blue green Fluorescence
- Trichophyton → Dull Blue. schonlenii

ii) Pityriasis versicolor



active space

iii) Erythrasma : caused by Corynebacterium minutisimum.



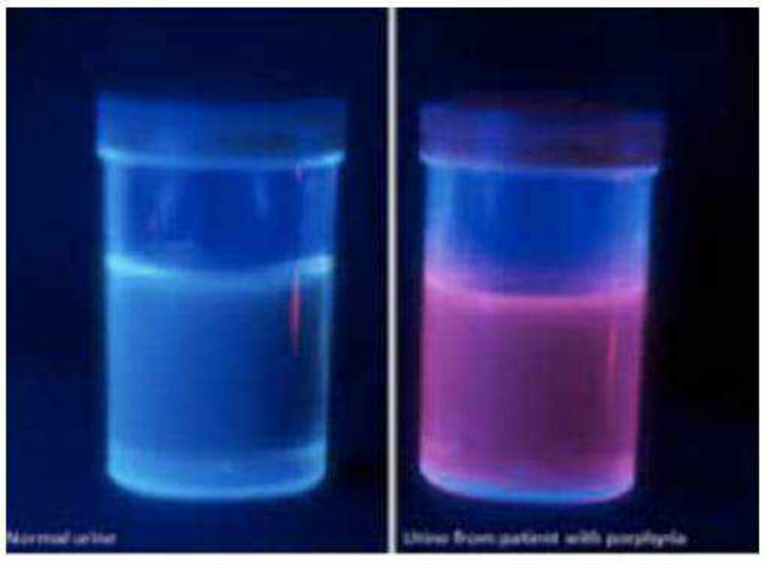
→ Coral Red fluorescence

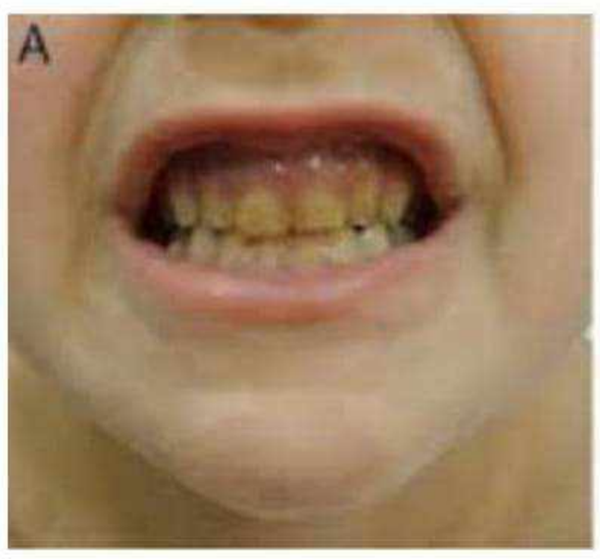
↓

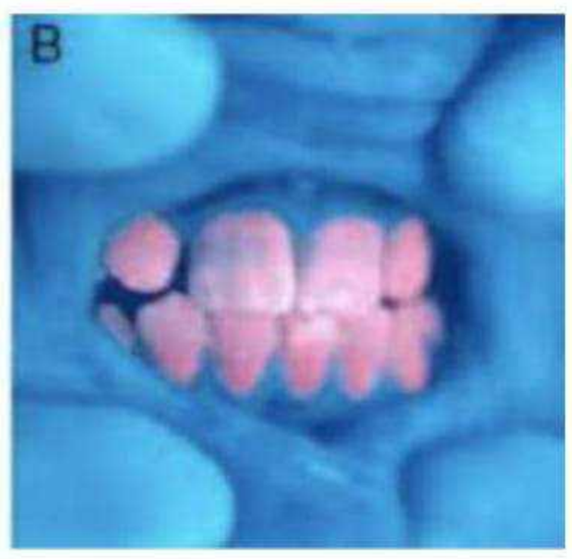
due to production of coproporphyrin

iv) Porphyria :

- urine ⟶ Red colour ⇒ seen in Porphyria cutanea tarda
- Teeth ⟶ Red colour ⇒ seen in Congenital Erythropoietic Porphyria.



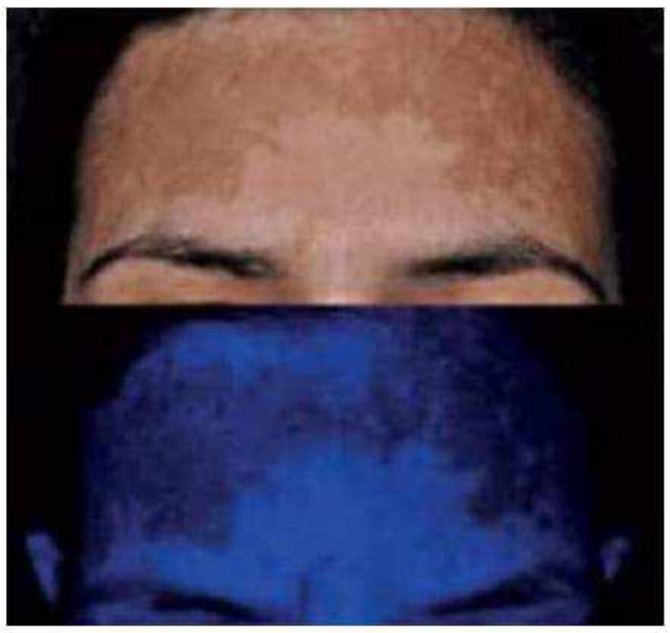




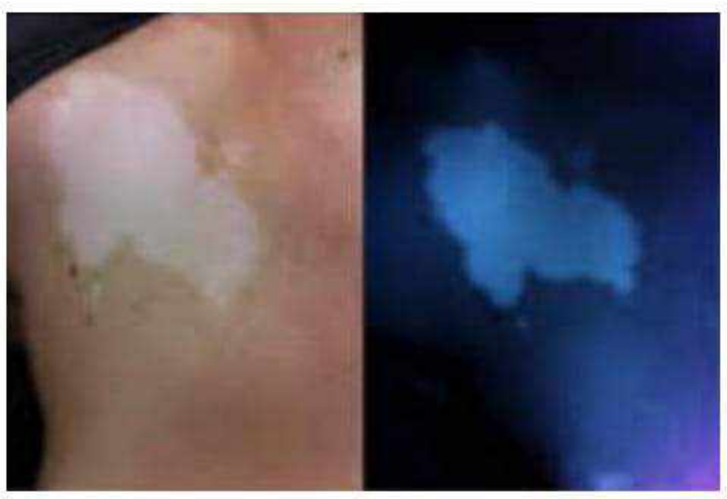
active space

v) Differentiate between epidermal and dermal pigmentation.

- **Epidermal pigmentation** : accentuate or increase.
- **Dermal pigmentation** : Bluish hue.



Melasma : accentuate ⊕



Vitiligo : accentuate ⊕

SMEARS

13:05

1. KOH smear (10% KOH)

- used in fungal infections
- KOH dissolves keratin but not fungal hyphae.
- Skin ⟶ active margin → keep for 10-15 min

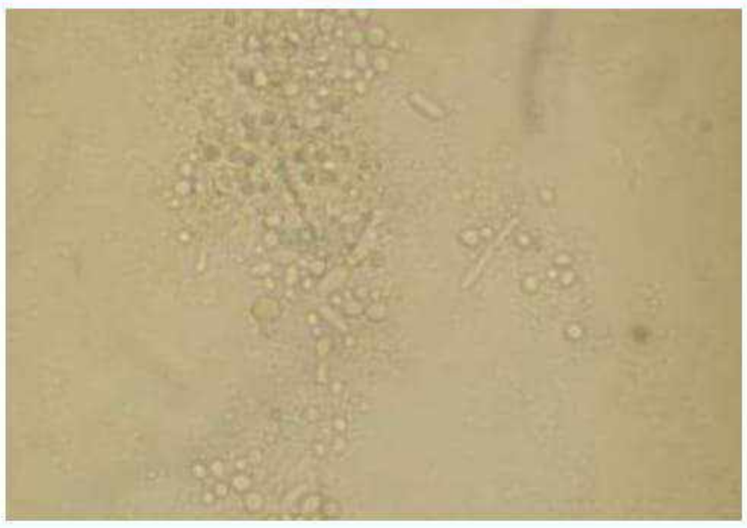
active space

- **Hair** → pluck the hair, root
- **Nails** → proximal part of nail (nail clipping)

} Dip it in **KOH solution**. for overnight

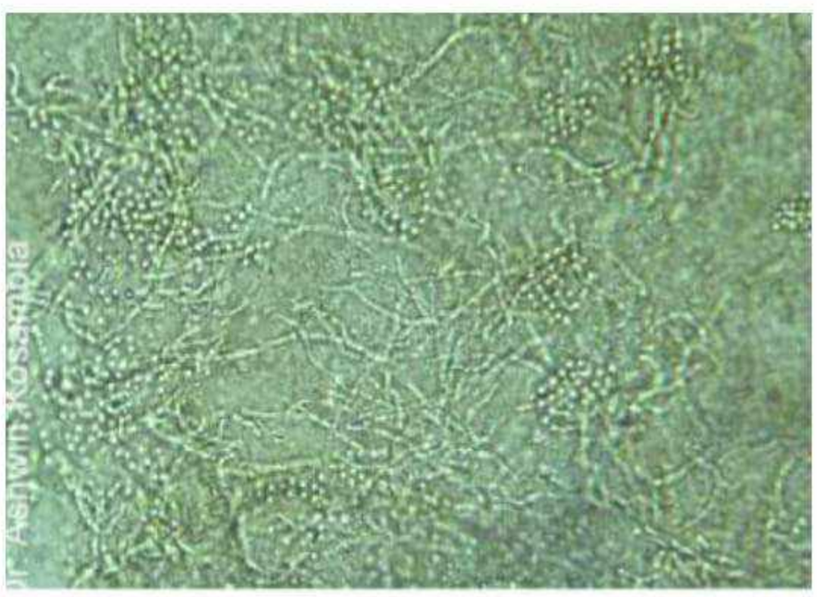
Dermatophyte

- Branched septate hyphae.



Candidal infection

- Budding yeast cells pseudohyphae.



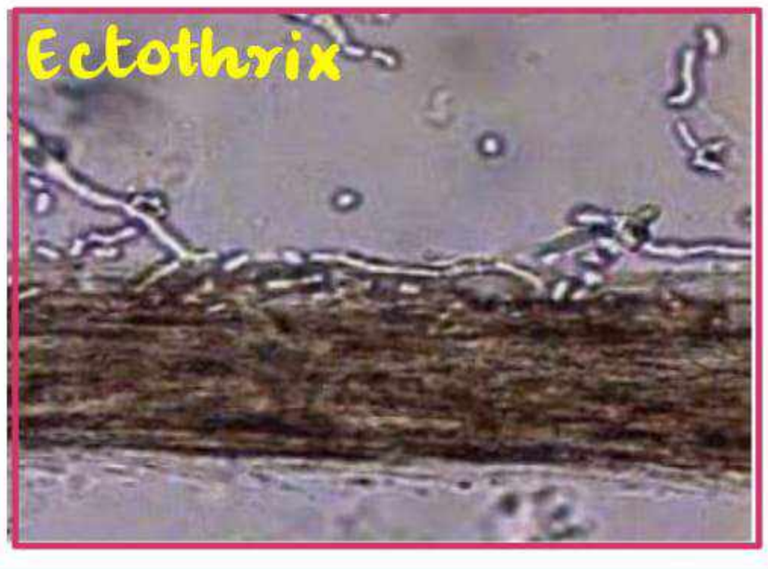
Pityriasis versicolor

- sphagetti and meatball appearance.
  - sphagetti → Hyphae, mycelium
  - meatball → Yeast
- Malassezia → Dimorphic fungus.

active space

Tinea capitis

- Ectothrix
- Endothrix ⟶ Inside

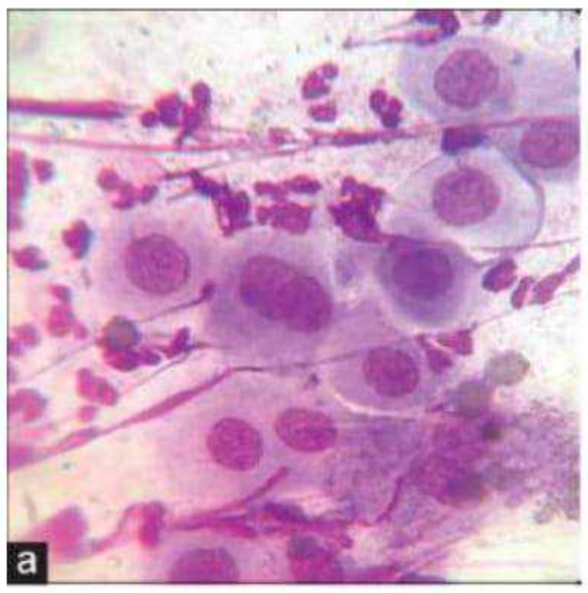


Tzanck smear :

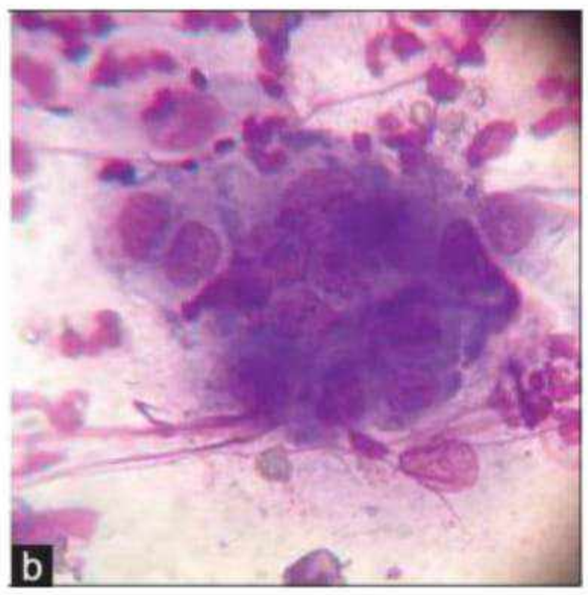
- Cytological test
- used in :

i) viral infections ⟶ Herpes, Varicella

ii) Autoimmune Blistering disorders



- Acantholytic cells
- Oval
- Nuclei occupy 7/8 of cell
- Peripheral Rim of cytoplasm.
- Autoimmune ⟶ Pemphigus



- Viral infections
- Multinucleate Giant cells + Acantholytic cells are seen.
- MNG's → fusion of histiocytes.

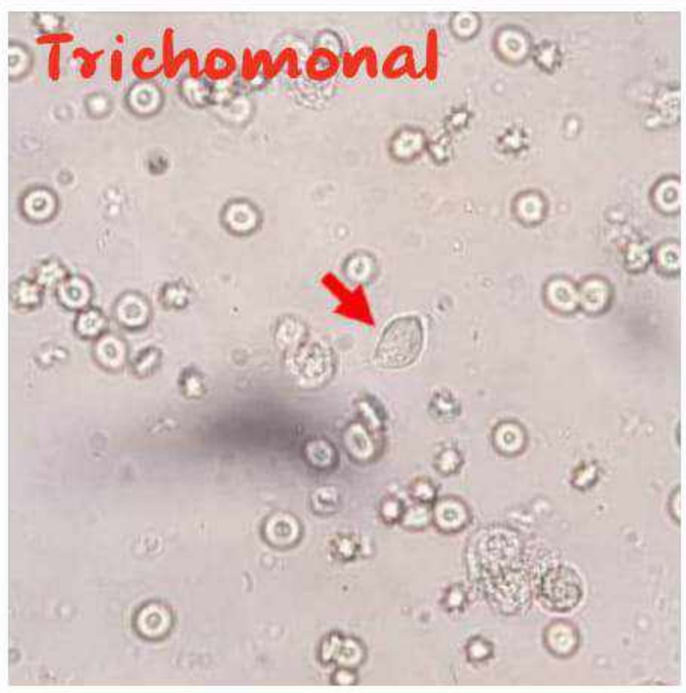
WET SMEAR

22:25

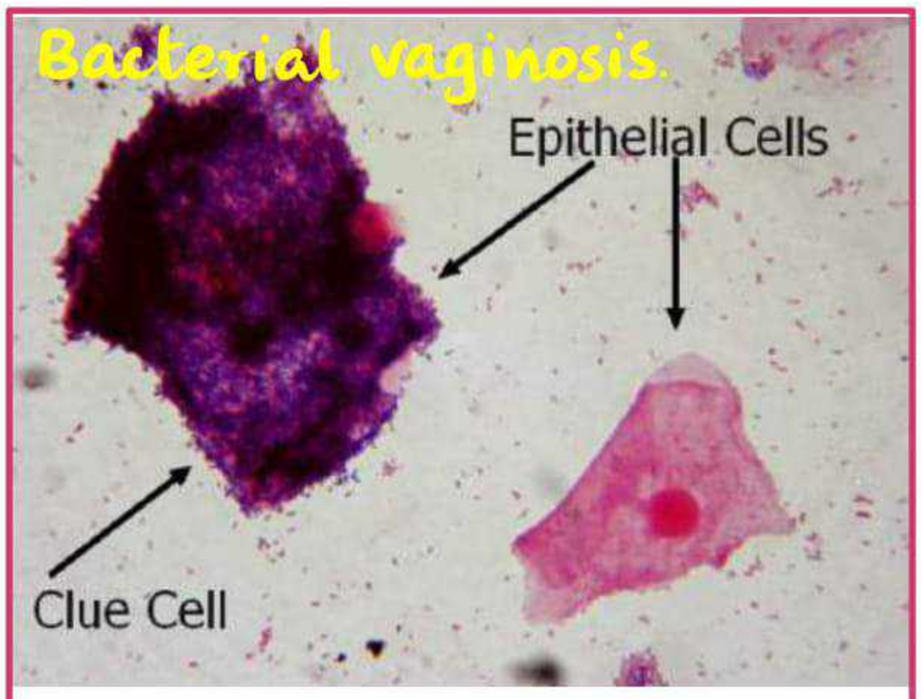
- used in

  i) Trichomonal infections

  ii) Bacterial vaginosis.



- pear shaped bodies
- jerky movement



- AMSEL'S Criteria

active space

TISSUE SMEAR

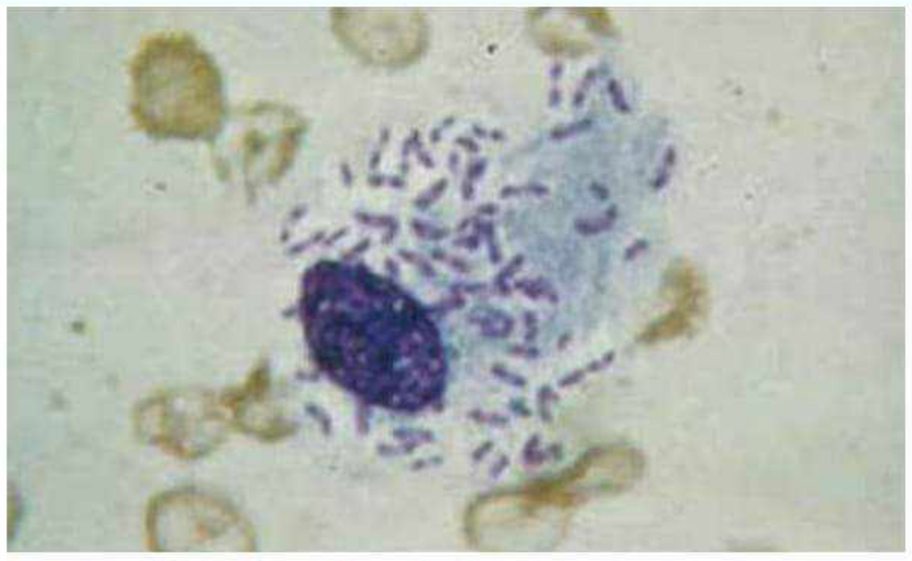
24:27

- used for identifying :

i) Donovan Bodies

ii) Molluscum bodies ⟶ seen in Molluscum contagiosum.

↳ a.k.a Henderson Patterson bodies.



Donovan Bodies

- Oval, purple

Molluscum Bodies.

- Intracytoplasmic eosinophilic inclusion bodies.

iii) Scabies Mite demonstration :

Scabies

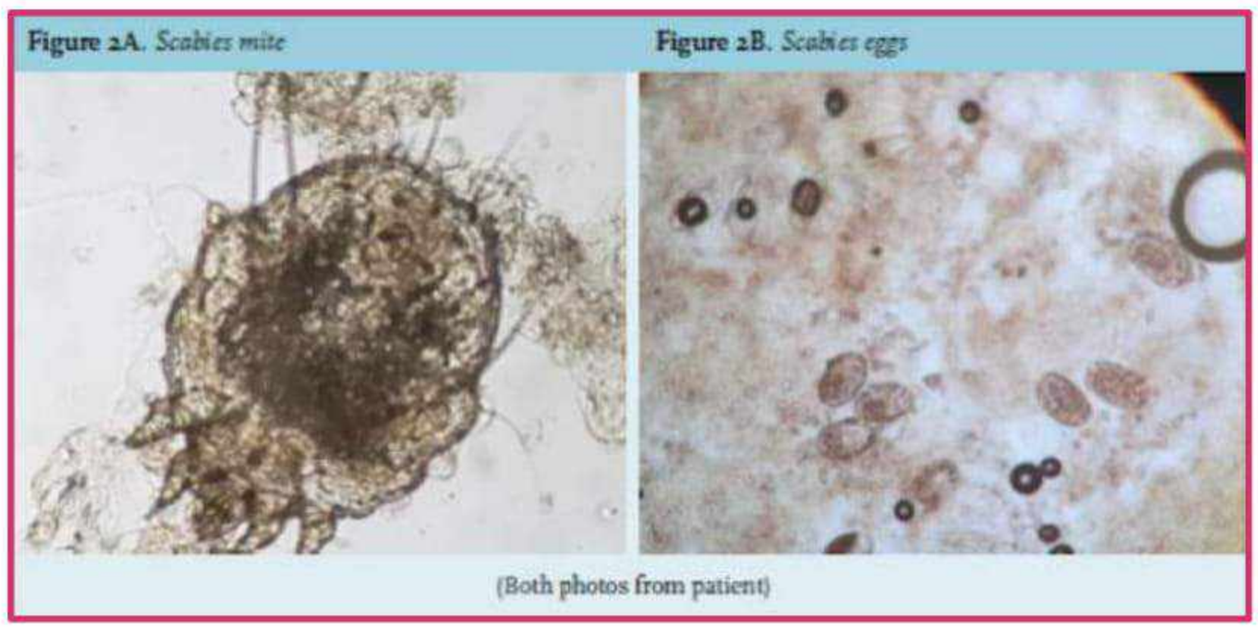


Figure 2A. *Scabies mite*

Figure 2B. *Scabies eggs*

(Both photos from patient)

active space

SLIT SKIN SMEAR

27:00

- done for

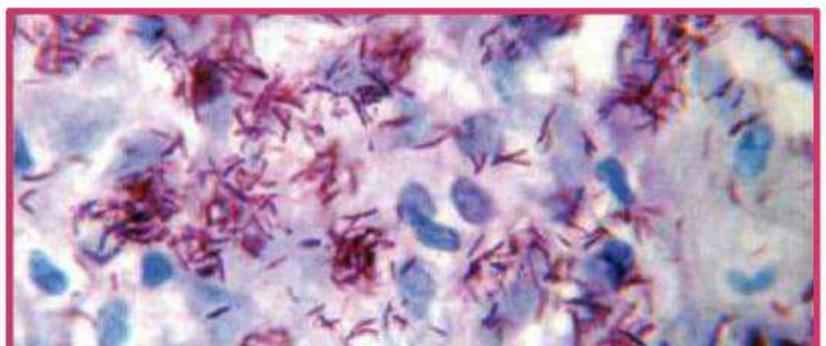
  i) Leprosy

  ii) Leishmaniasis

- No.15 Scalpel used

- Incision of 5mm length and 3mm width ⟶ on earlobe.

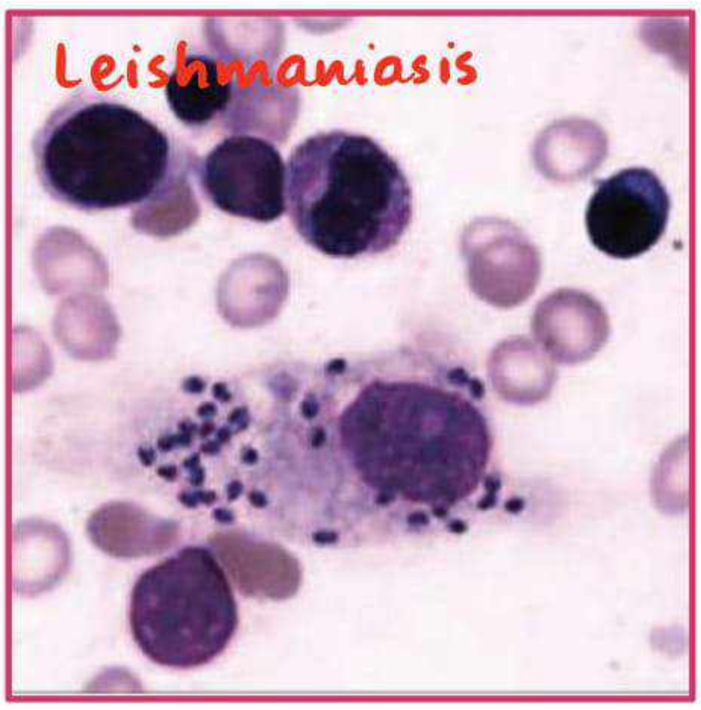
On Microscopy :



Acid Fast bacilli ⊕

- Dead bacilli ⟶ Fragmented, Granular
- Live bacilli ⟶ ▭
- Bacteriological Index
- Morphological Index.

} Role in Diagnosis and Prognosis.



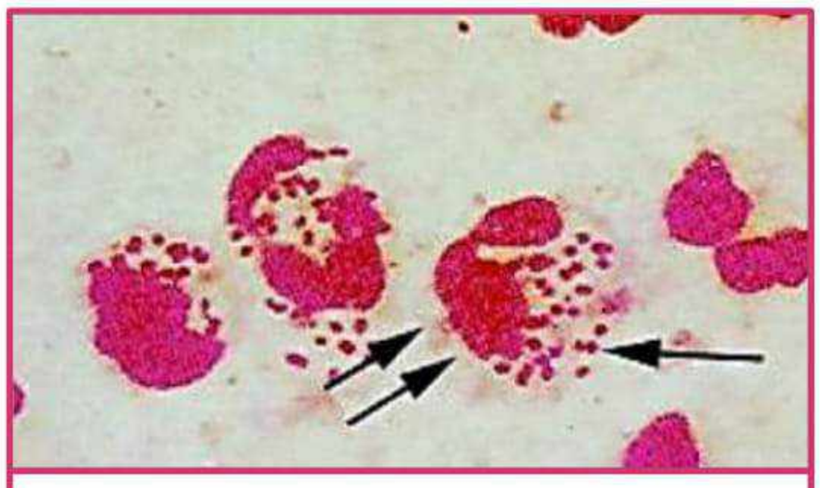
GRAM STAIN

30:33

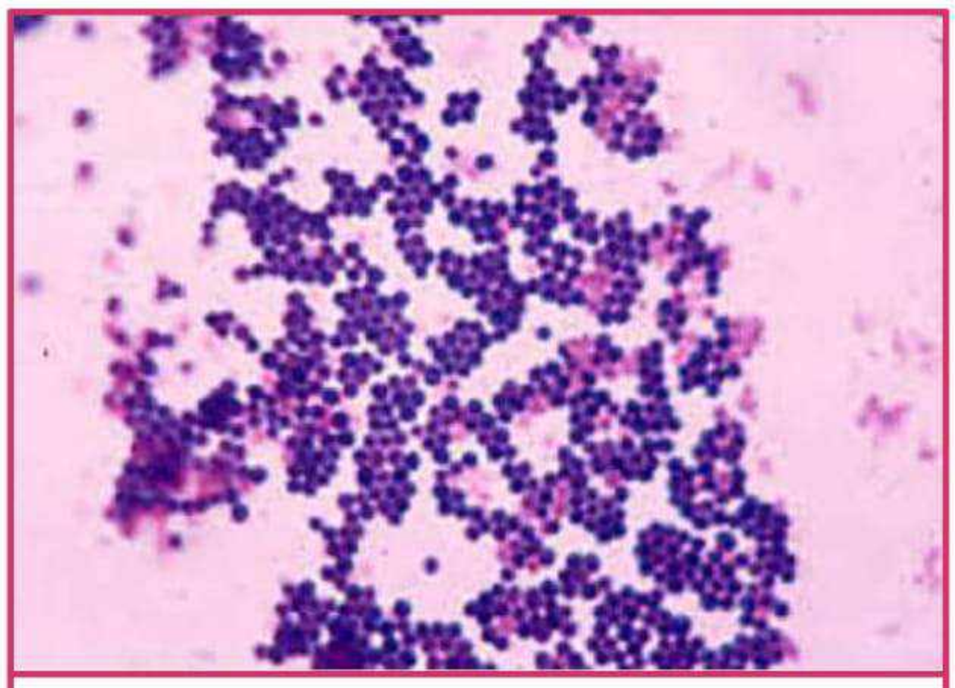
- Gram ⊕ → Retain 1° colour → Blue in colour
- Gram ⊖ → Retain 2° colour → Pink in colour

Steps

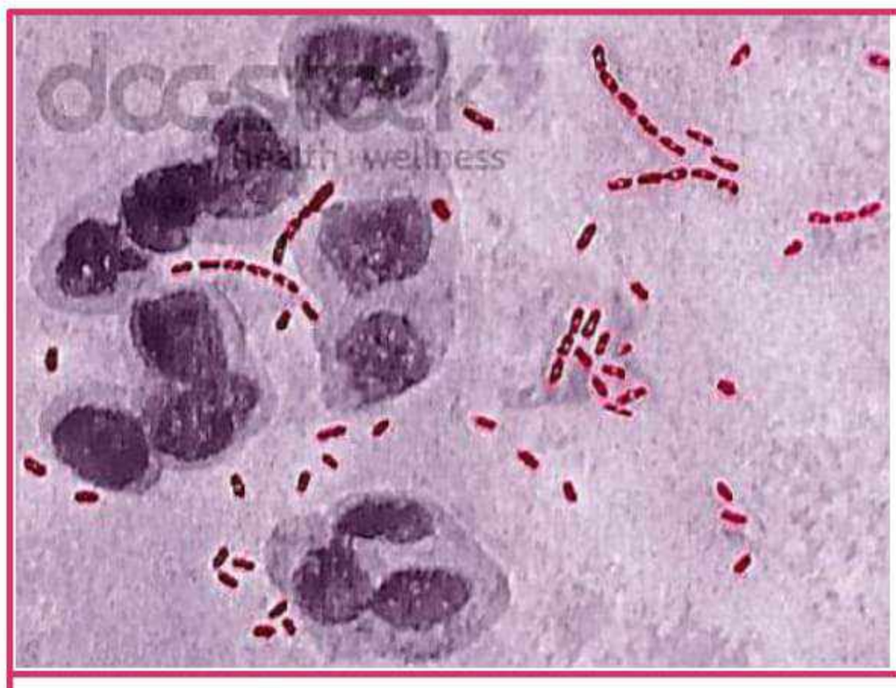
Methylene Blue
↓
Iodine
↓
Decolourise with acetone
↓
Safranin.



- Gonococcal bodies
- N. Gonorrhoea
- G ⊖



Gram ⊕ cocci Staphylococci



Gram ⊖ Rods and coccobacilli
H. ducreyi

active space

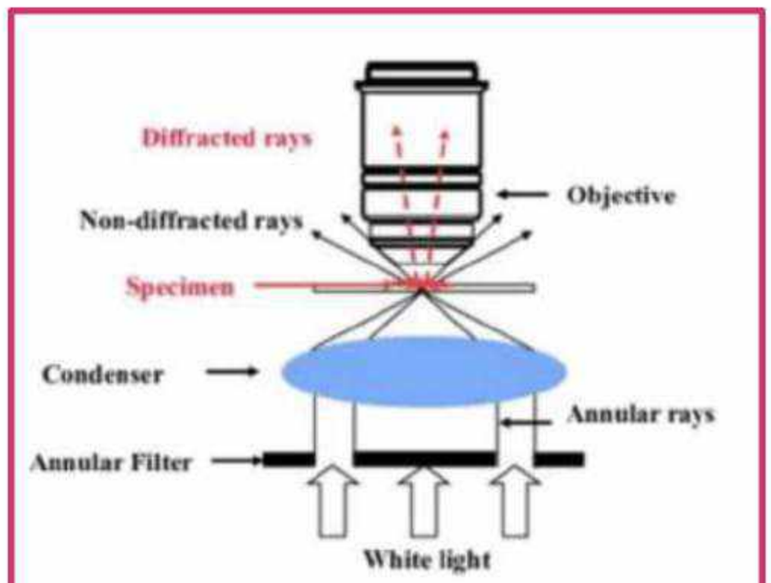
DARK GROUND MICROSCOPY

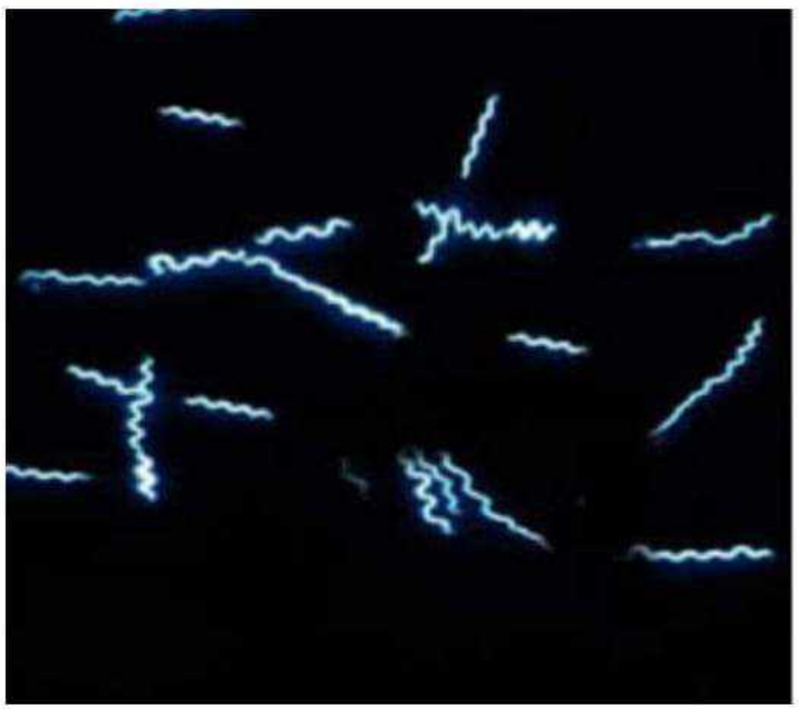
32:41

- used to diagnose

  i) 1° and 2° Syphilis
  (Treponema pallidum)

- organism appears white against dark background.





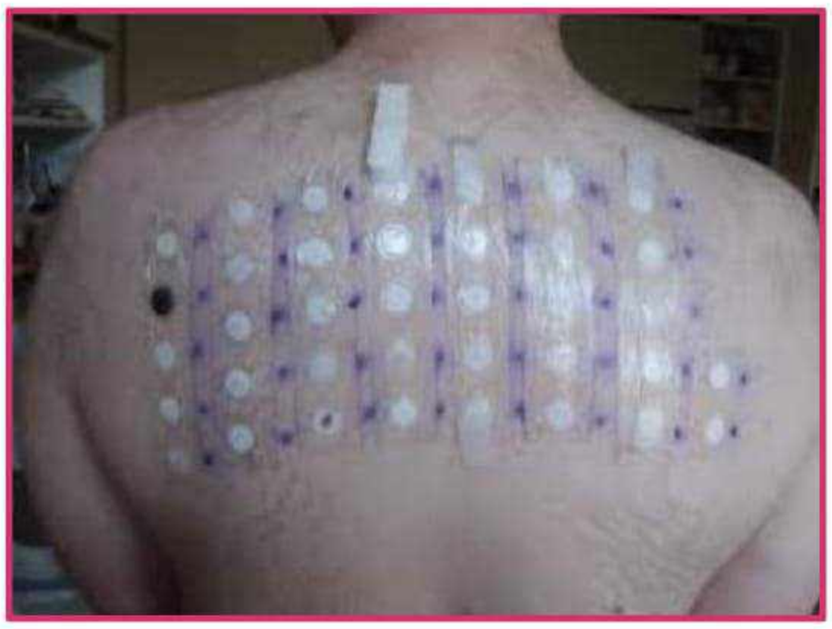
- Slender, wavy organisms which show coiling at regular intervals
- Corkscrew rotation (Flexion / Extension)
- Treponema pallidum

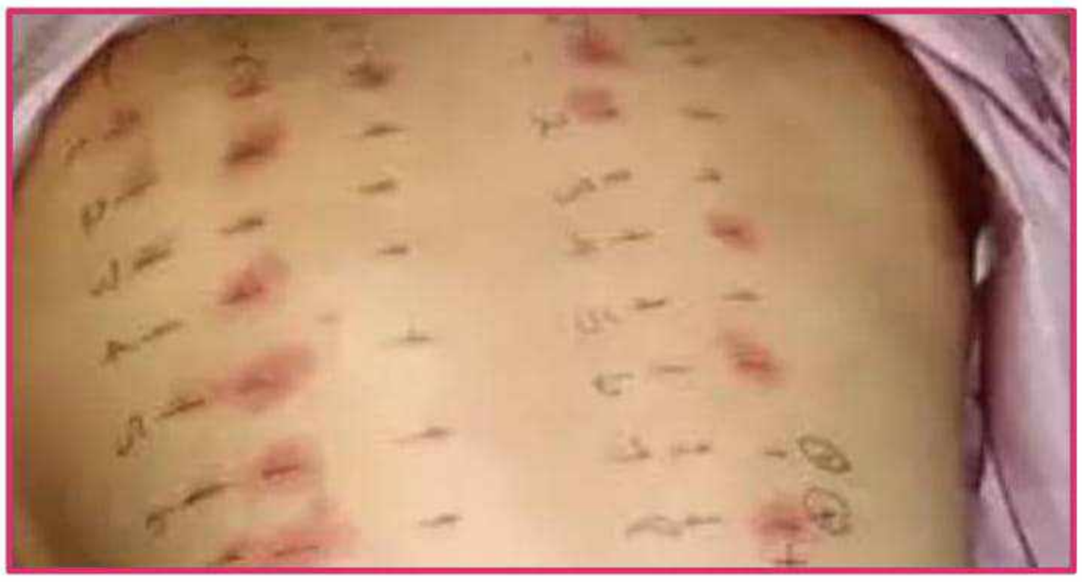
PATCH TEST

35:10

- used for identification of the causative allergin in patients of Contact Allergic Dermatitis.

- Read after **48 hrs** and **96 hrs.**





Photopatch

- Put the patch test in duplicate
- After 48hrs ⟶ irradiate one patch with UV Radiation. (UV A)

Control	Test	Result
−	−	−
+	+	?
−	+	+

- some allergen gets activated on exposure to sunlight.

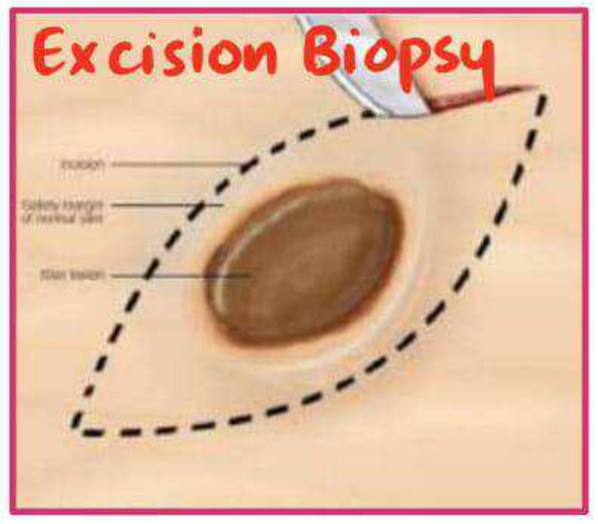
SKIN BIOPSY

40:37

- done to look for **Histopathological changes.**
- **4 Types**

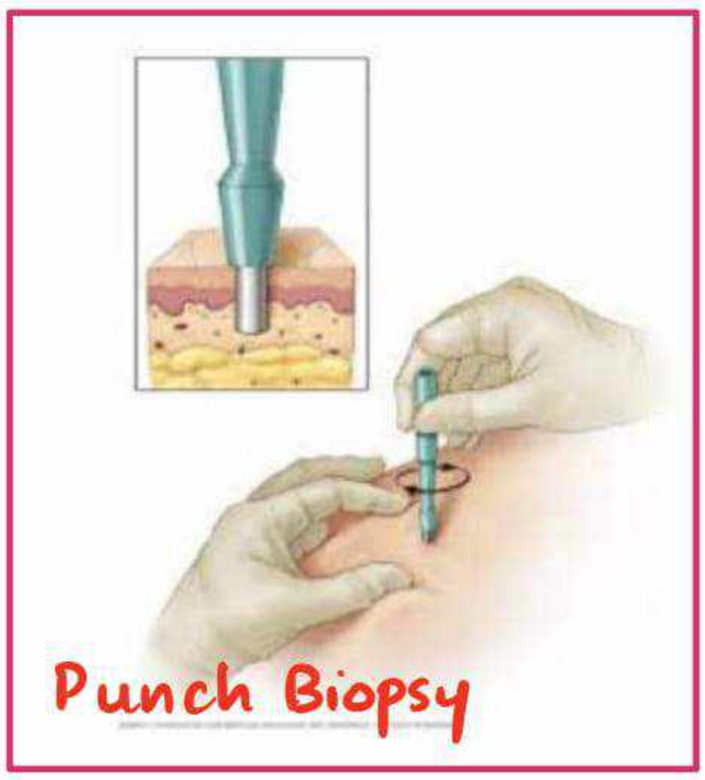
i) Excision Biopsy : Whole lesion excised

ii) Incision Biopsy : Part of the lesion is excised.



iii) Shave Biopsy

iv) Punch Biopsy → (Mc) Type done.



SIGNS IN DERMATOLOGY

43:30

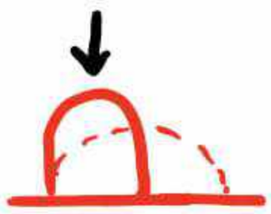
1. Nikolsky sign :

- shearing away of skin on putting tangential pressure on skin.
- seen in

  i) Pemphigus

  ii) TEN → Pseudonikolsy

2. **Bulla Spread sign** : seen in Pemphigus



3. **Grattage Test** :

- Accentuation of scaling on scraping the lesion with glass slide

4. **Auspitz sign** :

- scraping with glass slide

↓

↑sed scaling

↓

Red glazed appearance

↓

Pin point bleeding points

Bacterial Infections (Part - 1)

Flora

Resident

- present in everyones skin
- Non-pathogenic
- 1st line defence
- Staph epidermidis,
  Corynebacterium
  Propionibacterium
  Pityrosporum

Transient

- usually pathogenic
- Staph. aureus,
  β-Hemolytic streptococci
  (Strep. pyogenes)

Predisposing factors:

- Diabetes
- HIV
- Malnutrition
- Immunocompromised states

CLASSIFICATION

04:40

- Bacterial infections → Pyoderma
  - 1°
  - 2° - over underlying disorder

Primary Pyoderma:

- Follicular (Staph)
  - Follicle → Folliculitis
    - Superficial
    - Deep
  - Follicle + Perifollicular area
    - Carbuncle
    - Furuncle
- Non-Follicular (Staph + Strep)

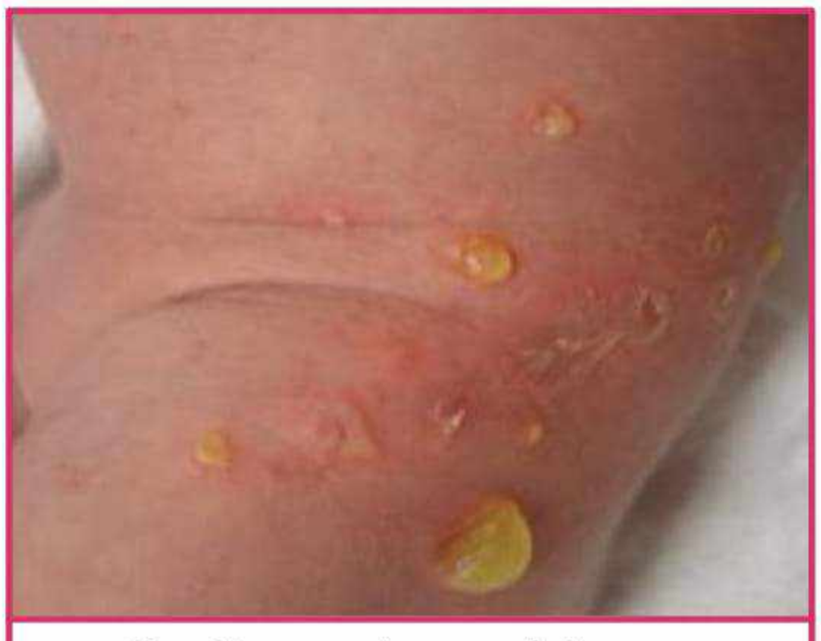
Non-Follicular (Staph + Strep)

- Localised
  - Superficial
    - Bullous impetigo (B.I)
    - Non-B.I
  - Deep
    - Ecthyma
- Spreading
  - Superficial
    - Erysipelas
  - Deep
    - Cellulitis.

IMPETIGO

10:02

- Superficial infection of skin
- (MC) Bacterial infection in children.
- (MC) involves face.



Bullous Impetigo

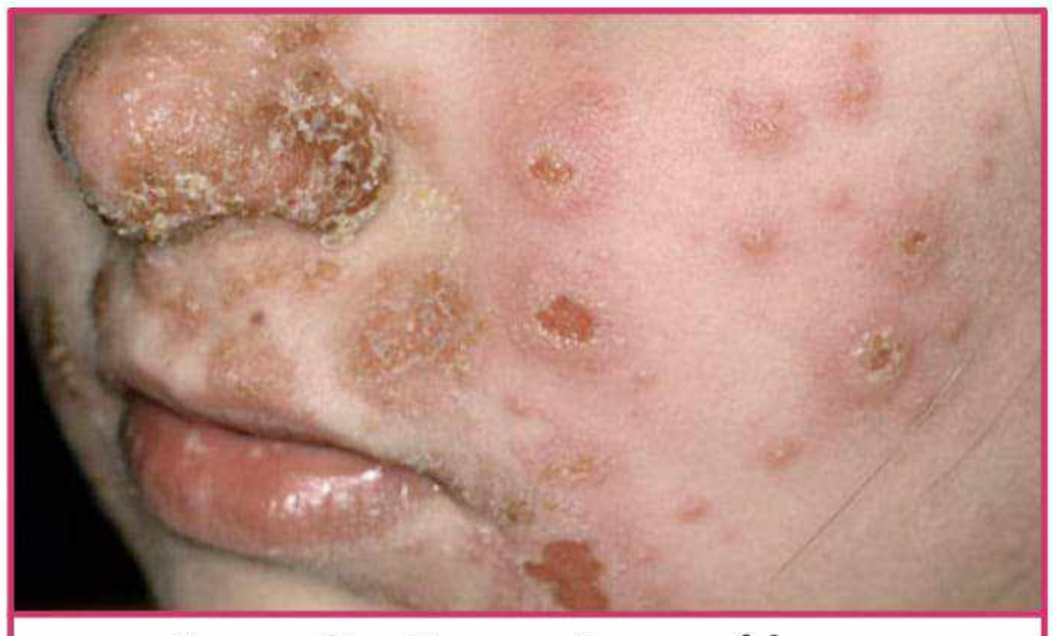
↓

- seen in newborns, children
- Caused by:

  Staph. aureus

- Bulla → Superficial
  ↓
  Rupture
  ↓

- forms Superficial erosions
- varnish like crusts
- No erythema in the surrounding area



Non-Bullous Impetigo

↓

- Preschoolers
- Caused by:

  Strep. pyogenes > Staph.

  ↓ developing nations

  ↓ developed

- Small vesicles → Rupture
  ↓

  Raw erosions with honey coloured / golden yellow crusts.

active space

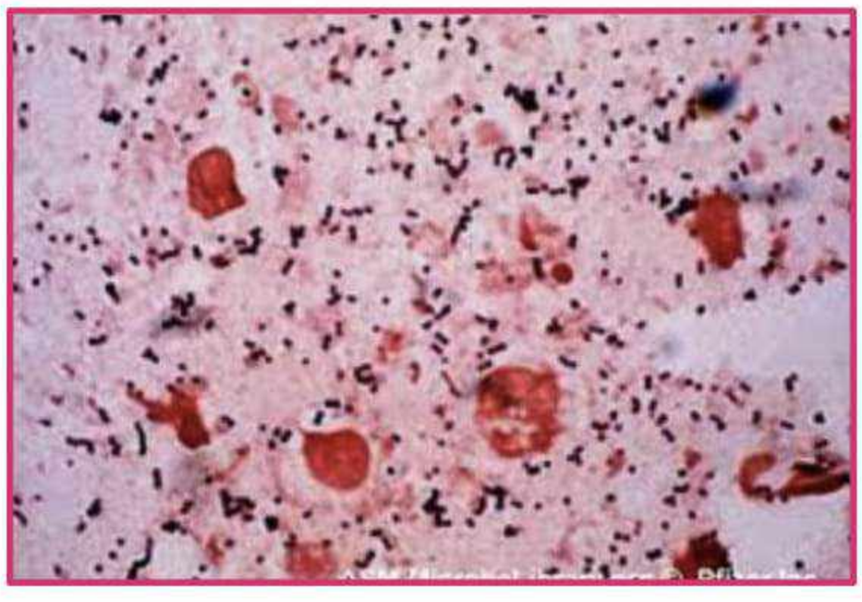
Bullous Impetigo	Non-Bullous Impetigo
• No lymphadenopathy	• lymphadenopathy ⊕ • Complication : PSGN • Polycyclic erosions ⊕

On Pus Culture :

- Intracytoplasmic, Extracytoplasmic

  Gram ⊕ ve cocci seen in clusters

- Staph → Clusters



Management

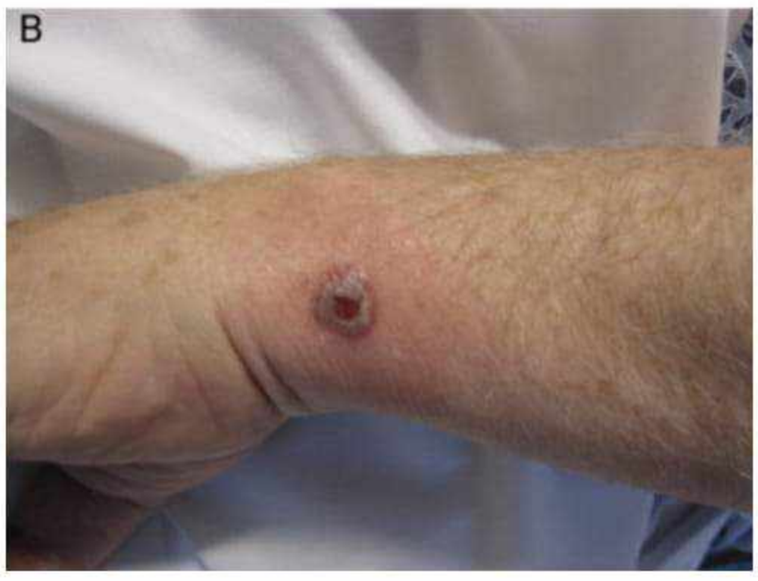
1. Topical → Fusidin, Mupirocin, Bacitracin, Ratapumalin
2. Systemic → Cloxacillin, Cephalexin, Erythromycin.

ECTHYMA

19:15

- causative organism : Streptococcus > Staph

- Small Bulla on erythematous base
  ↓
  Rupture → Heaped up crust
  Remove the crust ulcer
  Heals with scarring



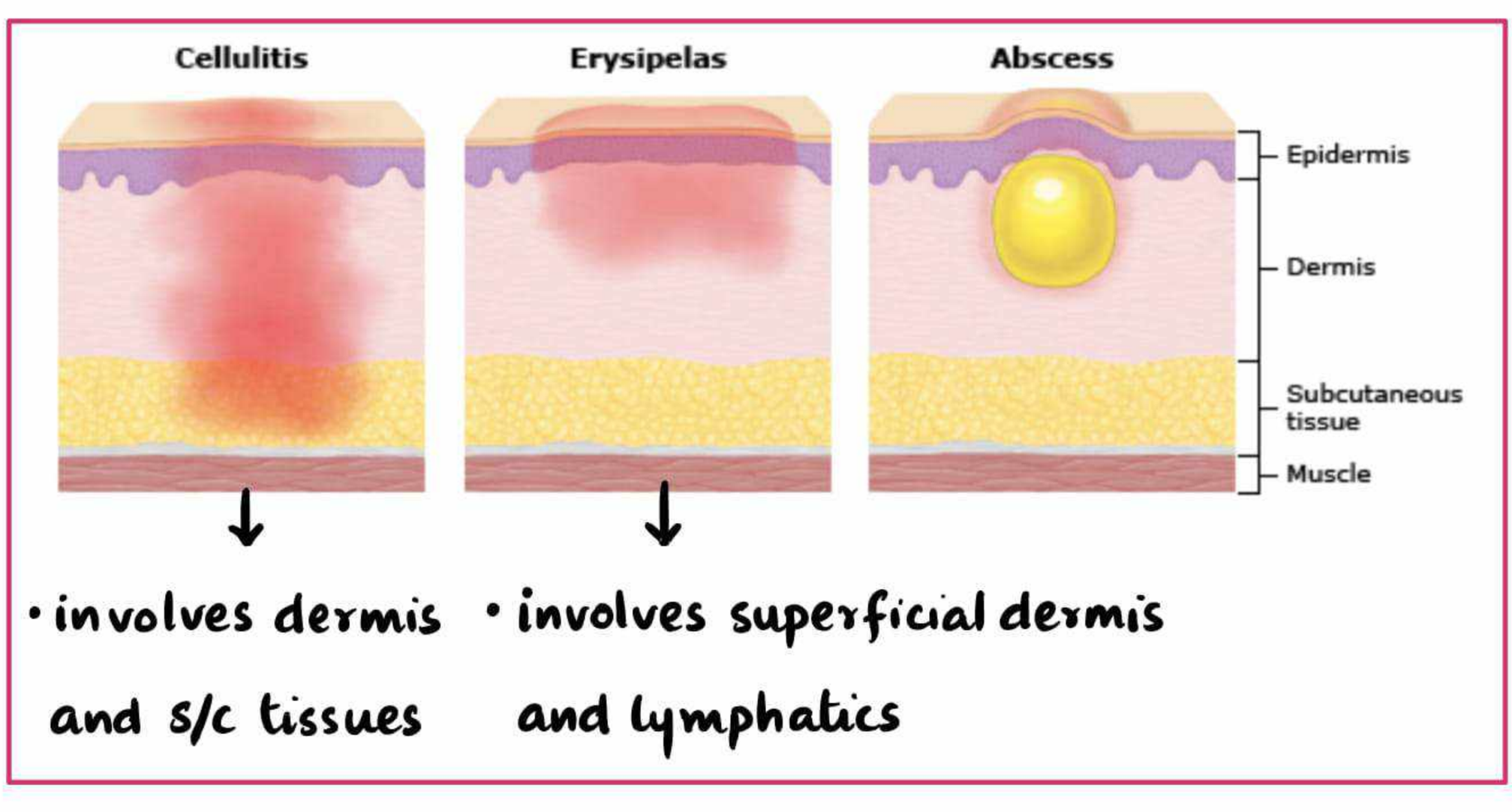
- Common sites :
  - Buttocks
  - Thigh
  - Legs.

ERYSIPELAS AND CELLULITIS

21:25

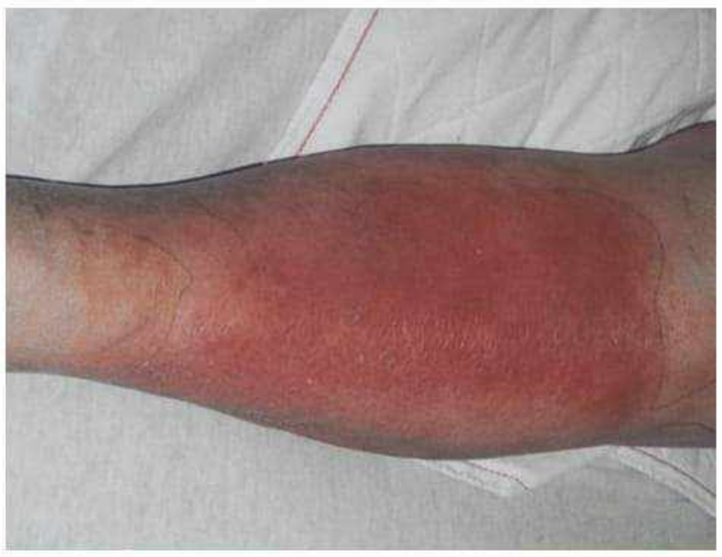
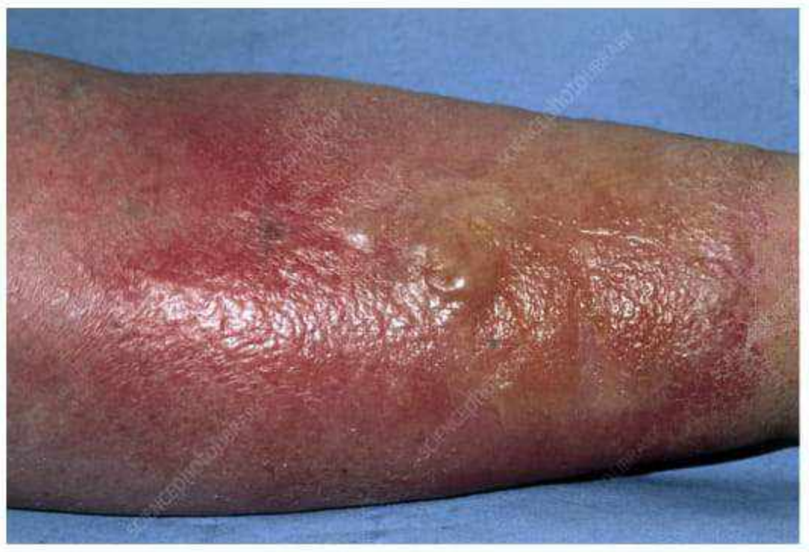
common features :

- Soft tissue infections
- Warm, tender plaques ± fever and constitutional symptoms
- sites : LL > UL > Face



- involves dermis and s/c tissues
- involves superficial dermis and lymphatics

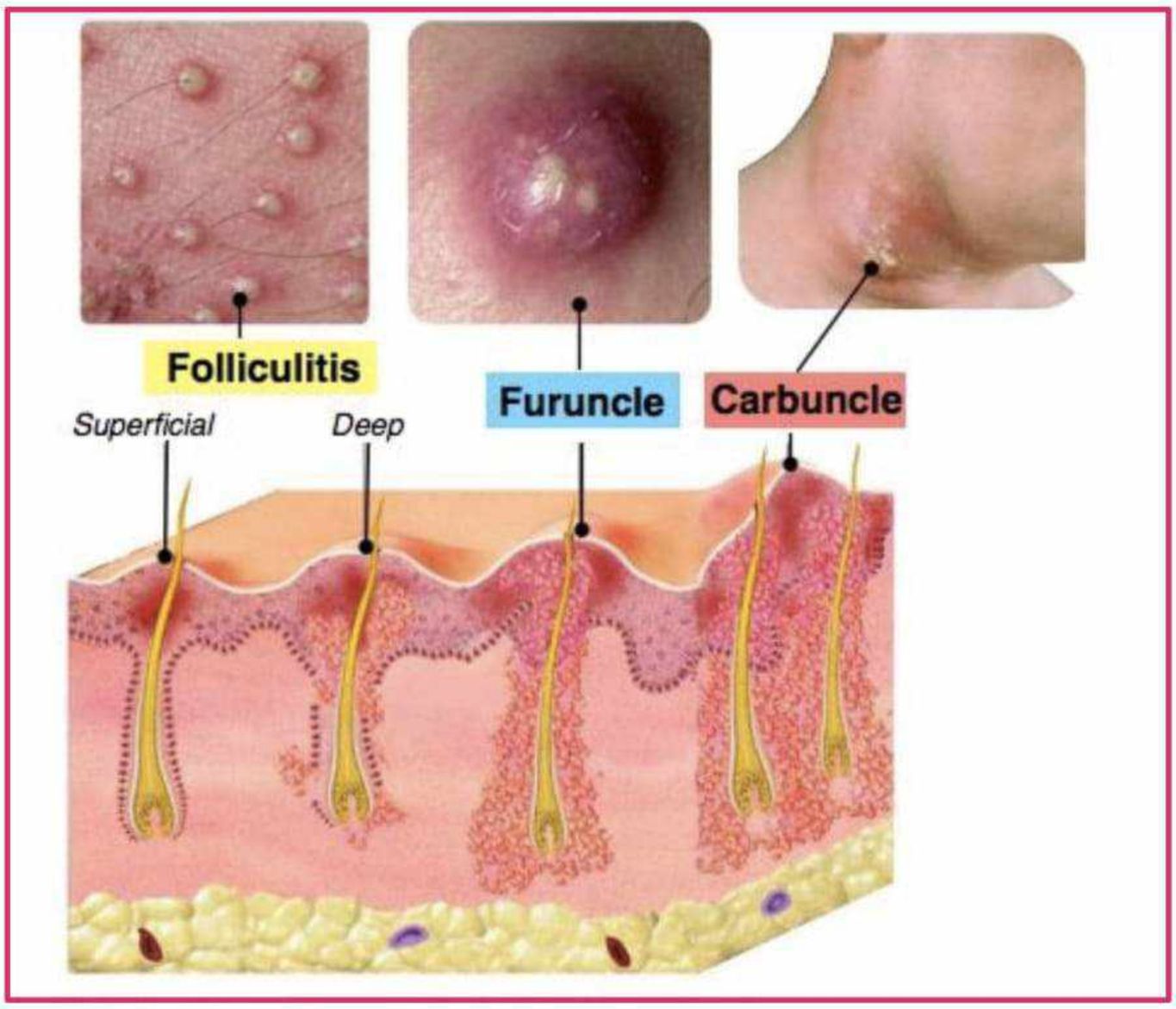
active space

Erysipelas	Cellulitis
	
• Group B Hemolytic Strep.	• Staph / Strep
• Streptococcus + lymphatics	• Deep dermis + s/c Tissue
• Well demarcated erythematous plaque	• Not well demarcated
• Superficial vesiculations ⊕	• Crusting, pustules ⊕
• Treatment : i) Systemic Antibiotics ii) NSAID's iii) Leg elevations	

active space

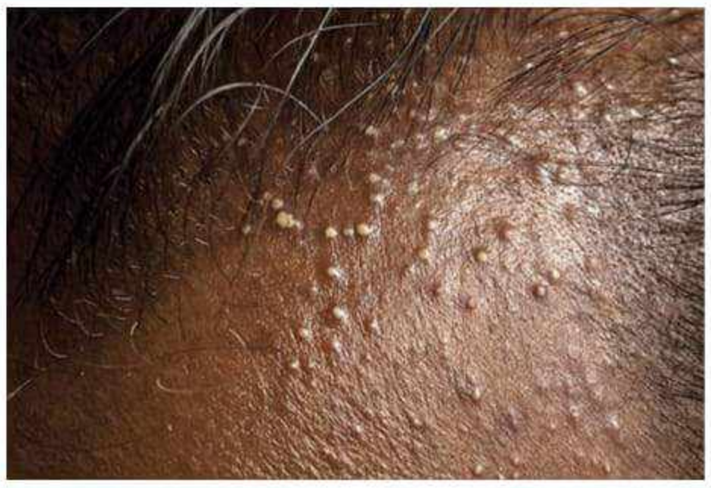
FOLLICULAR PYODERMA

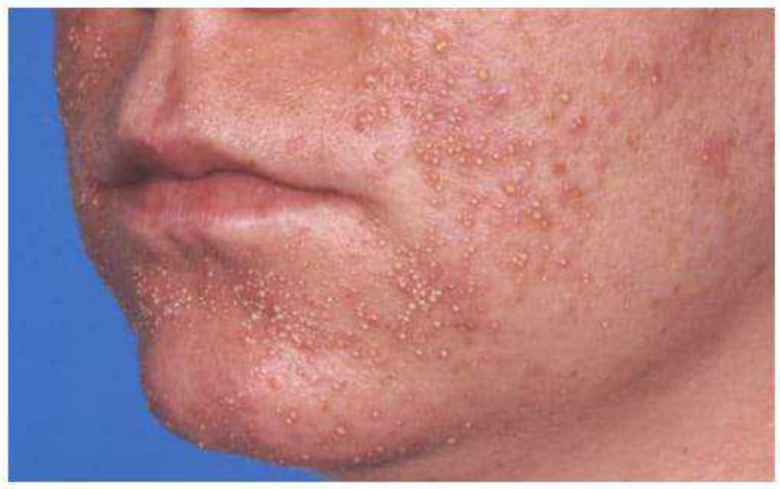
27:30



Superficial folliculitis :

- a.k.a Bockart's folliculitis
- upto infundibulum
- superficial pustules in follicular skin
- seen in scalp and face.
- 3 Types : i) Infections
  ii) Chemical → occupation
  iii) Pseudofolliculitis → Waxing





Superficial folliculitis

Deep folliculitis :

1. SYCOSIS BARBAE

- Not fungal infection
- Erythematous discrete papules and pustules which are form a plaque.

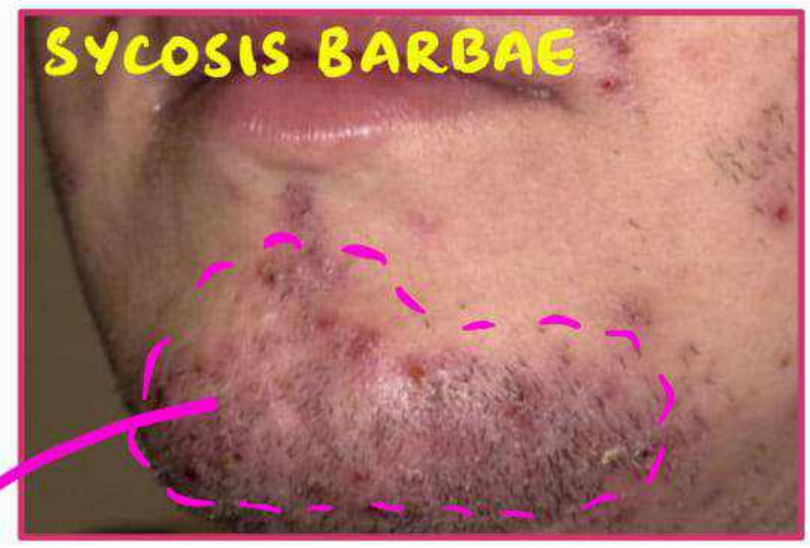
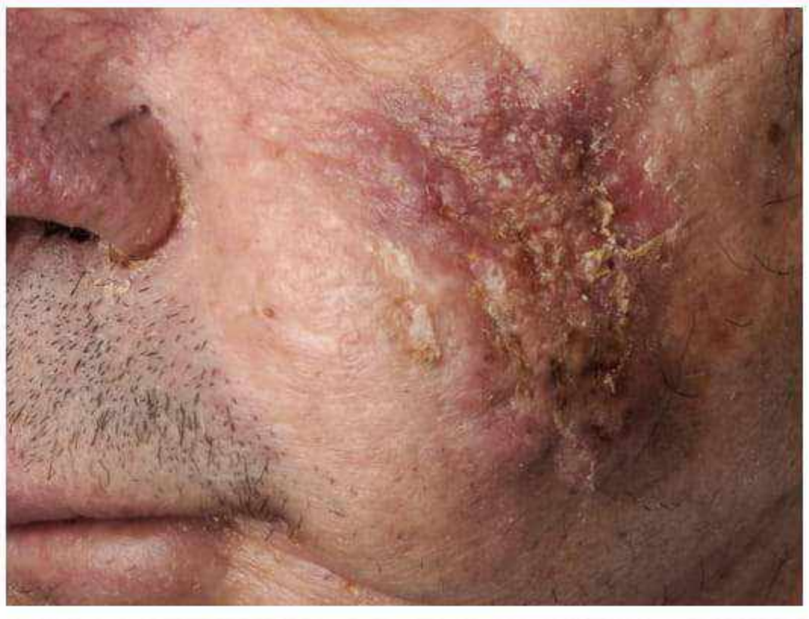


Fig like appearance

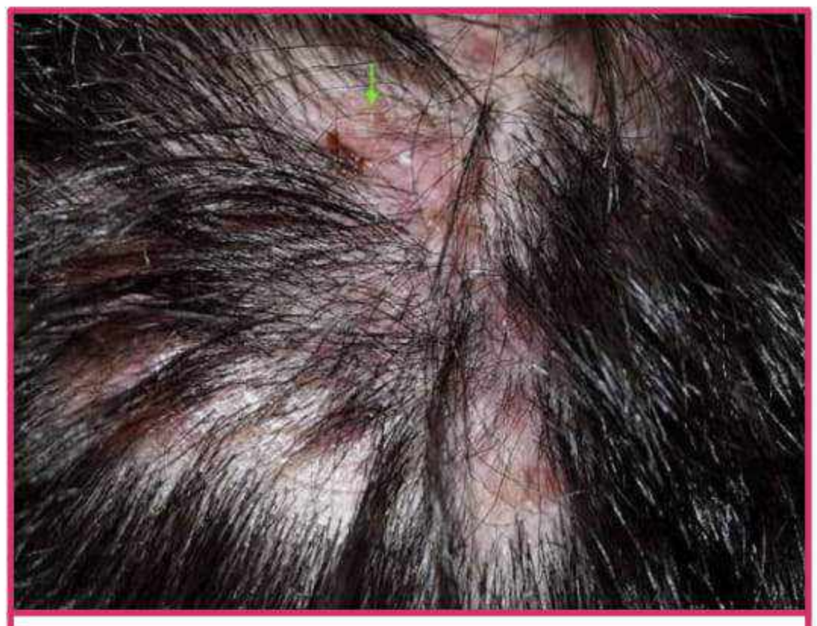
- more common in perioral, perianal

Tinea barbae : itchy , involves mandible



Lupoid Sycosis :

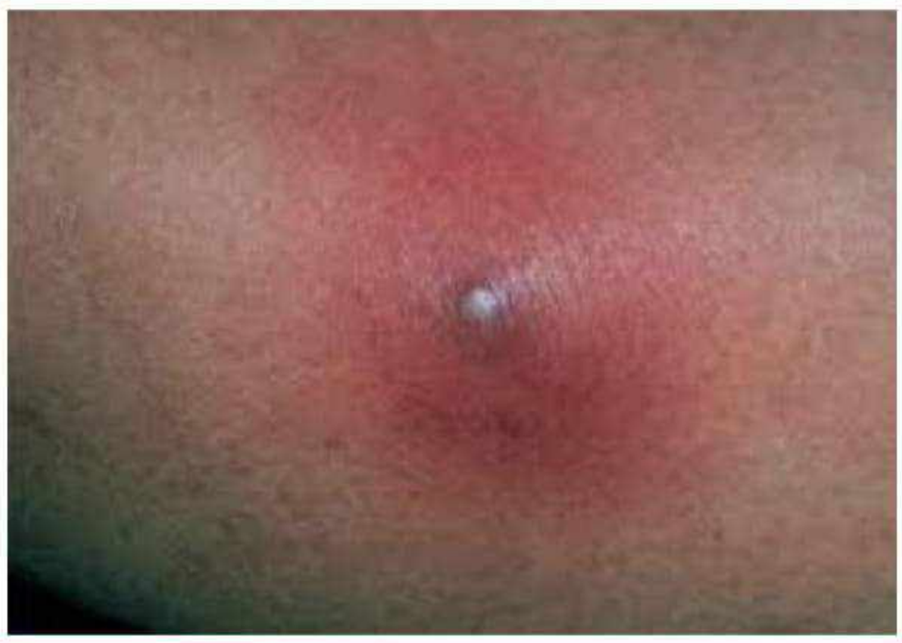
- sycosis barbae heals with scarring



Folliculitis decalvans

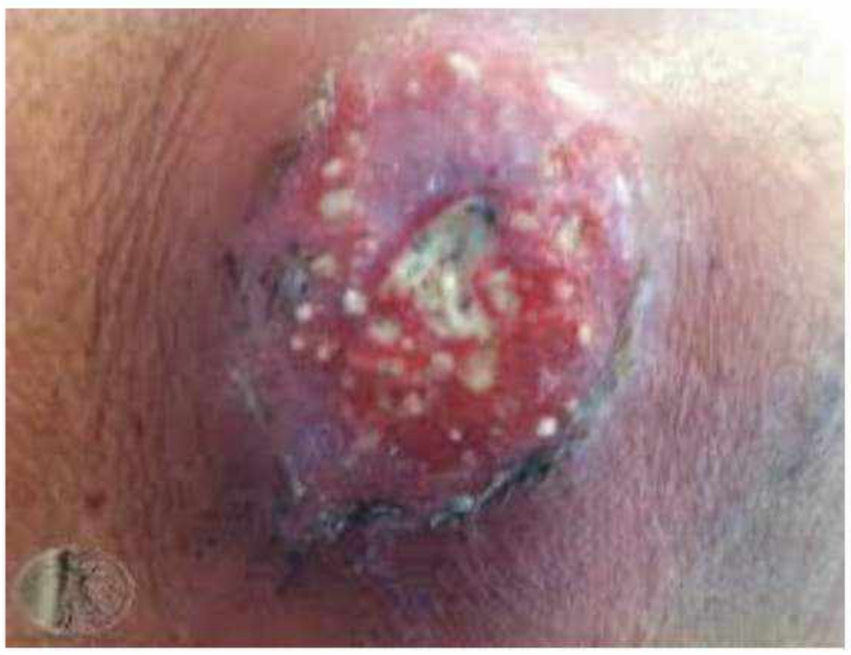
PERIFOLLICULITIS

35:00



Furuncle/Boil

- Warm, tender
- Painful swelling along with pustules seen in hair bearing areas of body.



Carbuncle

- Multiple contiguous hair follicles involved.
- commonly seen in Diabetics.
- Site : seen in Back (MC)

active space

BACTERIAL TOXIN MEDIATED

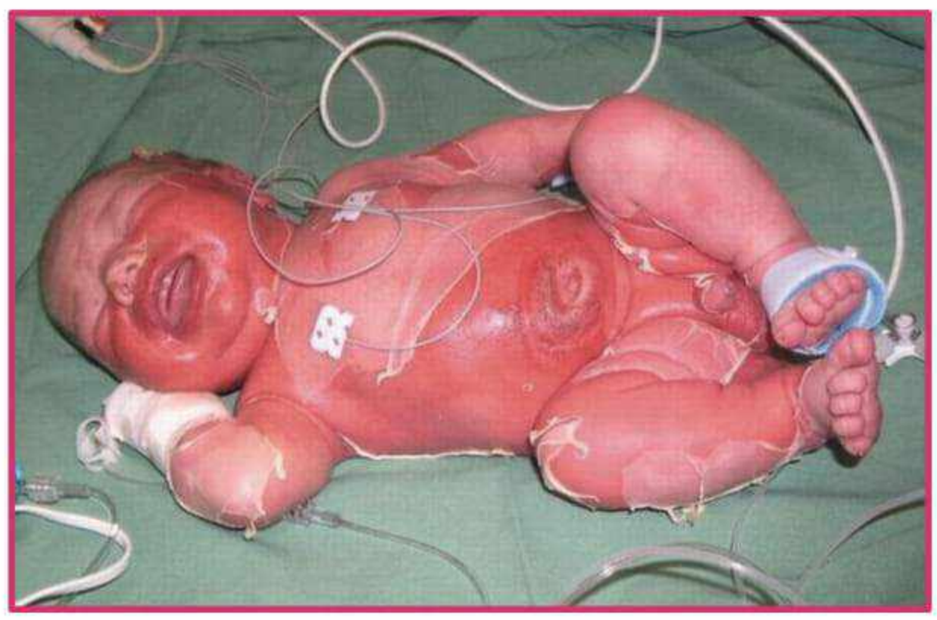
36:50

- mediated by Bacterial toxins
- 1° focus of infection ⊕
- Hematogenous spread of toxin
- Pus C/s - Negative
- Constitutional symptoms ⊕
- organism
  - Staph → SSSS, TSS
  - Strep → TSS, Scarlet fever.

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

39:40

- a.k.a SSSS
- Exfoliating dermatoses.
- seen in Newborns and children
- Distant site bacterial infection ⊕ (Impetigo, G.I Infection, Throat infection)



↓

Releases Exotoxin (Exfoliative Toxin)

↓

Spreads hematogenously, goes to skin

↓

active space

↓

Target Dsg 1

↓

separation of keratinocyte

↓

Intraepidermal
(just beneath granular layer)

↓

peeling of skin (Nikolsky Sign ⊕)

Treatment

1. Systemic I.v Antibiotics : Cloxacillin / Clindamycin.

SSSS	TEN (Toxic Epidermal Necrolysis)
• No cell necrosis • Split in the granular layer • Nikolsky sign ⊕ Tzanck smear • Acantholytic cells ⊕ but no inflammatory cells	• Drug reaction • Cell necrosis ⊕ • Pseudo nikolsky ⊕ • Many inflammatory cells ⊕

active space

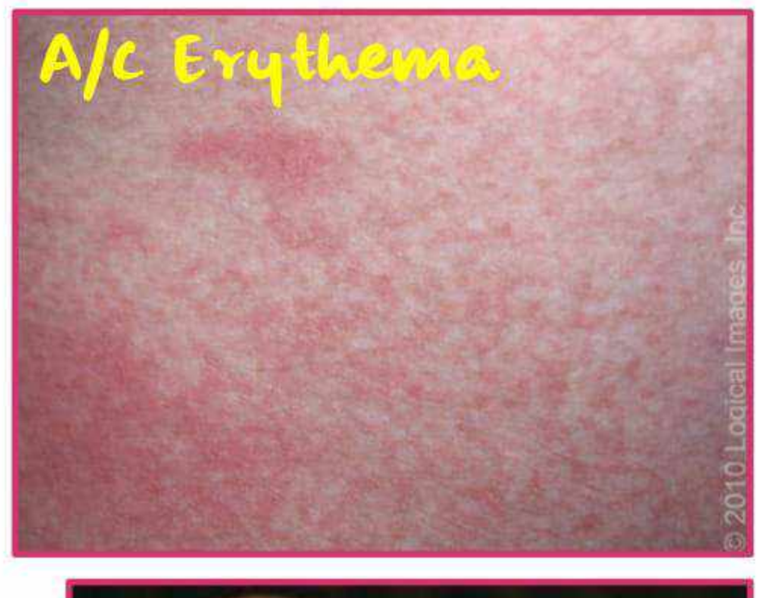
TOXIC SHOCK SYNDROME

46:50

- caused by Staphylococcus and Streptococcus.

Features :

- Fever
- Circulatory shock
- A/C Erythema f/b Desquamation
- Multiorgan involvement.



active space

Bacterial Infections (Part - 2)

CORYNEBACTERIUM INFECTIONS

00:20

- Gram ⊕ Rods / Diphtheroids
- Diseases caused are :
  - i) Erythrasma
  - ii) Pitted keratolysis
  - iii) Trichomycosis axillaris.

ERYTHRASMA

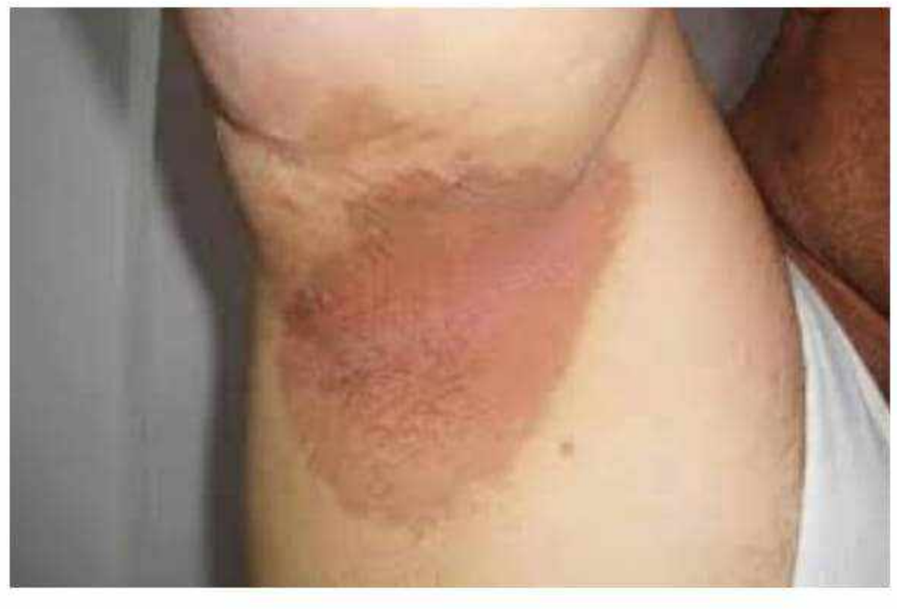
01:20

- Superficial infection caused by corynebacterium minutissimum
- seen in adults / children
- Sites : Axilla, groins, inflammatory area, toe clefts

Features :

- Well demarcated erythema to Brownish plaques, fine scales, usually asymptomatic.

  vs. fungal infections → itchy



active space

On Wood's lamp :

- Coral Red fluorescence due to Coproporphyrin III.

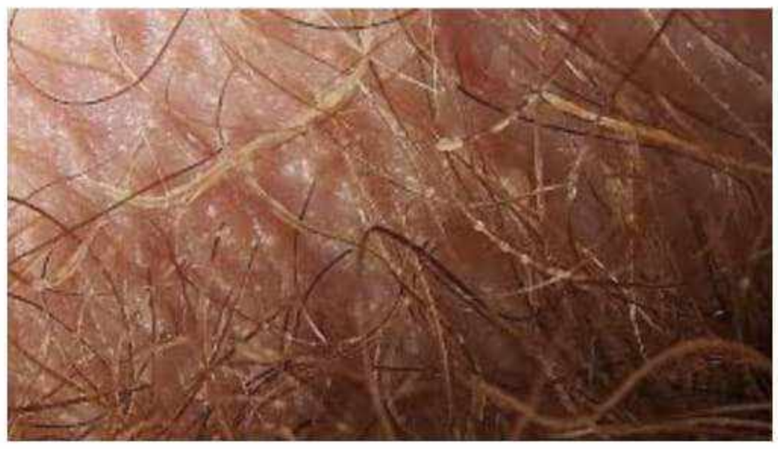
Treatment :

- Topical azoles
- Clindamycin
- Oral Erythromycin.

TRICHOMYCOSIS AXILLARIS

04:50

- Not fungal infections
- Adherent granules on hair shaft axilla, groins
- Variously coloured.
- Staining of clothes ⊕



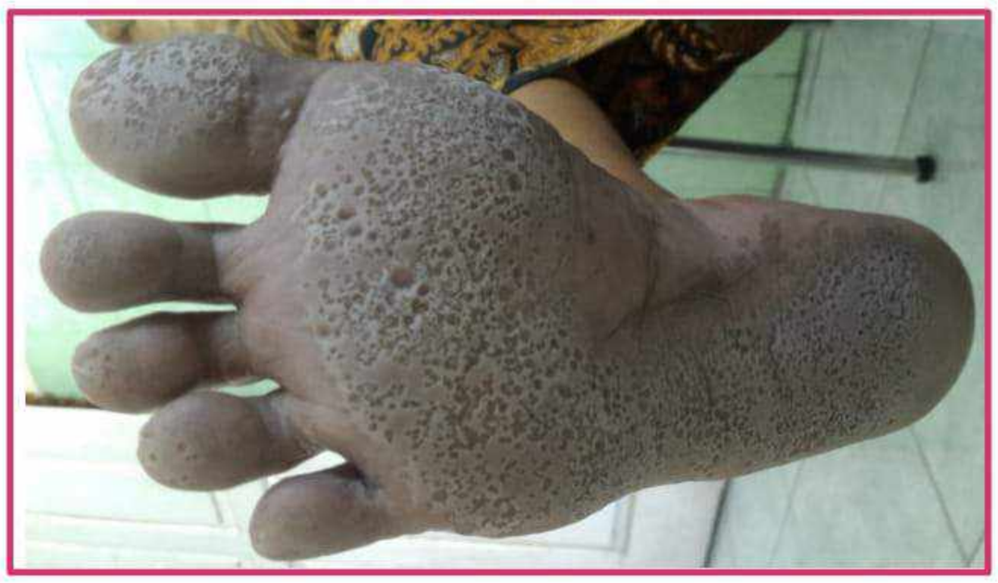
PITTED KERATOLYSIS

05:50

- also caused by Micrococci sedentarius
- superficial pits on the foot

active space

- common in individuals with hyperhidrosis
  ↘ excessive sweating



ANTHRAX 07:40

- caused by Bacillus anthracis (zoonotic infection)
- seen in persons who handles livestock

Clinical feature:

- Malignant pustule ⊕
  ↳ not painful
- Sites : Hands, neck, face,
  small pustule on
  erythematous base
  ↓
  Ruptures → Crust
  ↓
  Surrounding erythema and edema → superficial vesicles.

Treatment:

- Oral ciprofloxacin
- Doxycycline
- Amoxicillin

PSEUDOMONAS SKIN INFECTIONS

10:20

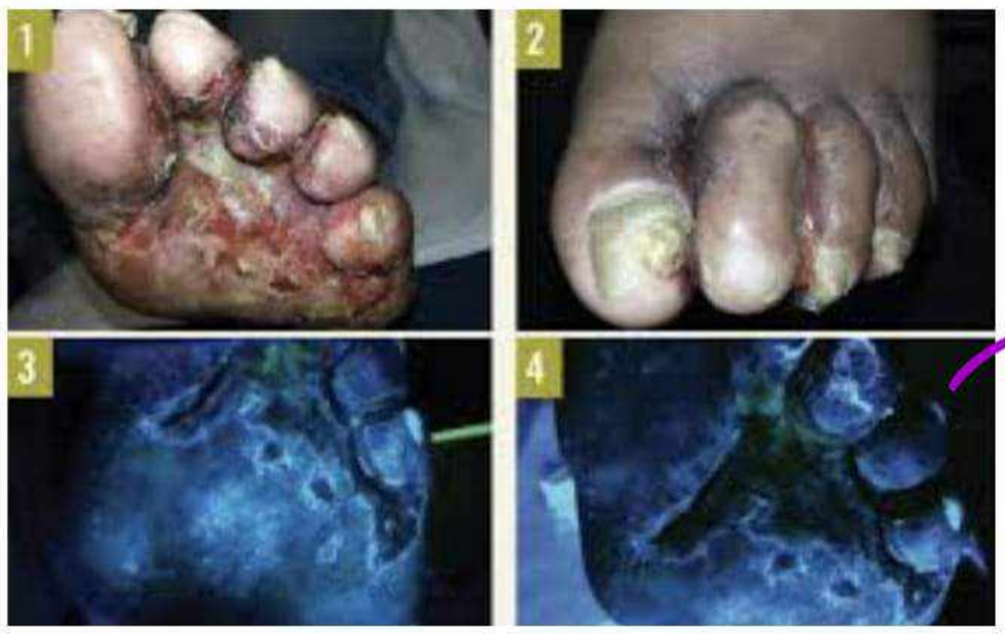
- G⊖ Bacilli
- 2 Kinds of secretions
  - Pyoverdin → Fluorescence (yellowish green) → Pseudomonas
  - Pyocyanin → Non-fluorescent → Pseudomonas aeruginosa.

Green Nail Syndrome

- Discolouration
- Onycholysis
- Inflammation → Paronychia.

active space

Interweb infections

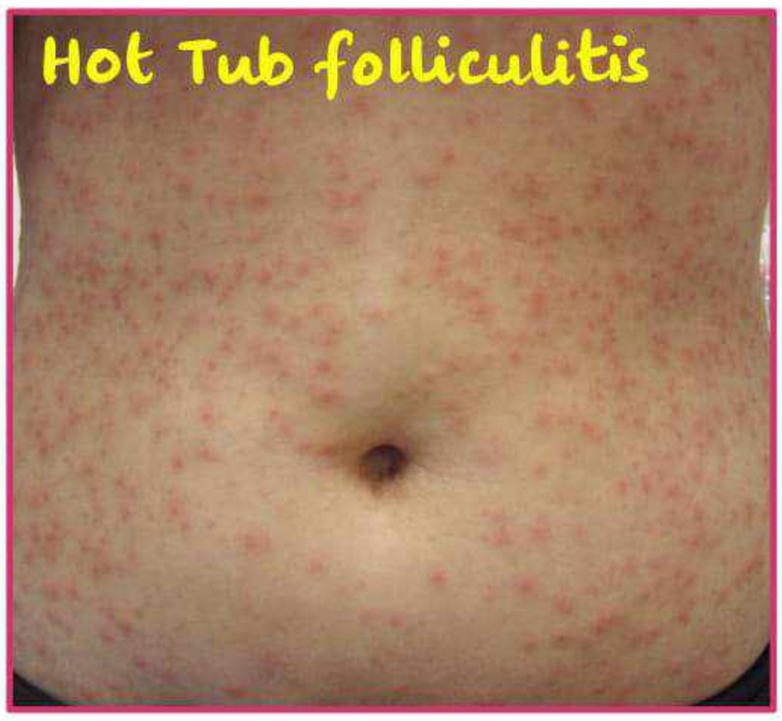


→ On Wood's lamp:

- greenish yellowish fluorescence
  ↓
  due to pyoverdin

Hot Tub folliculitis

- Multiple monomorphic erythematous papules which appears on trunk 24 hrs after exposure to contaminated water
- Jakuzzi's Swimming pool

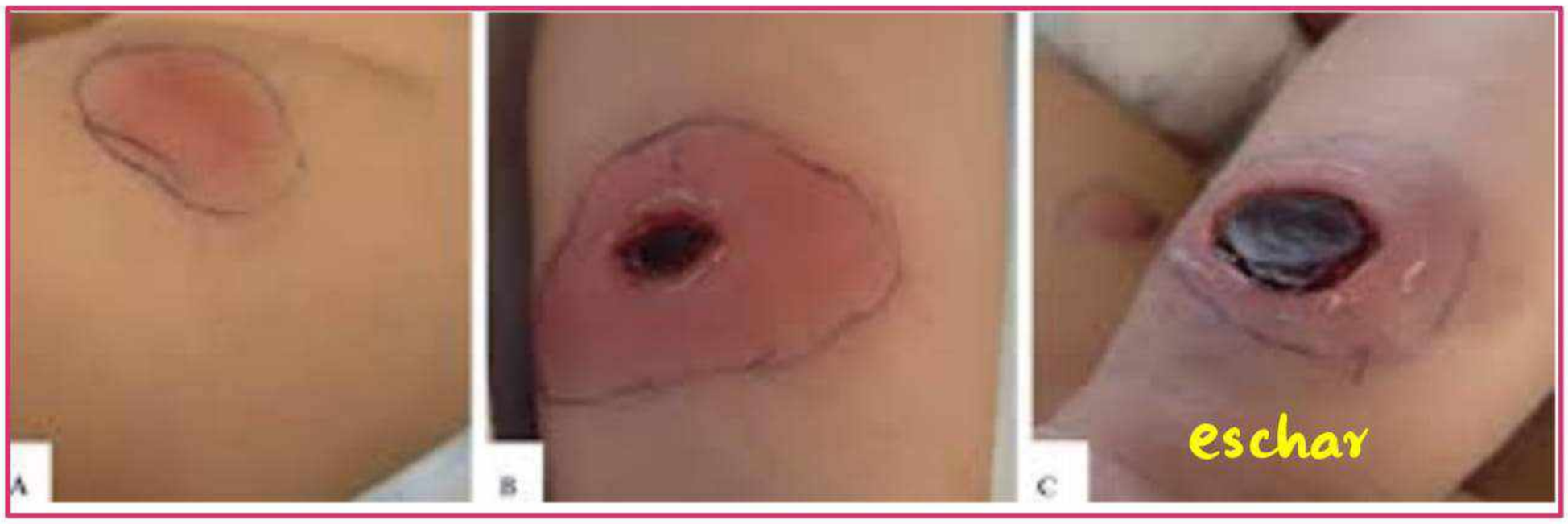


ECTHYMA GANGRENOSUM

14:35

- Ecthyma
  - contagiosum → caused by Streptococcus
  - gangrenosum → caused by Pseudomonas
  - infectiosum → caused by Orf virus.

active space



- Necrotic skin infection occuring in individuals with decreased immunity
- Pseudomonas bacteremia ⟶ severe infections

Clinical features :

- Greyish macule with surrounding erythema halo
  ↓
  ulcerates, necrosis, eschar - Necrotic crust.

Treatment :

- Aminoglycosides
- β Lactam Antibiotics.

Pseudomonas cause :

i) Necrotising fascitis
ii) Gangrenous cellulitis
iii) Infections of ear

MANAGEMENT

19:20

Topical

- Fusidic acidic
- Mupirocin
- Bacitracin
- Ratapumulin
- Condy's compresses/ $KMnO_4$

Systemic

- **Indications :**
  - i) Severe and widespread infections
  - ii) Fever
  - iii) Lymphadenopathy
  - iv) No response to topicals
  - v) Cellulitis / fascitis
  - vi) Immunocompromised / DM

Systemic Treatment

- Cloxacillins → Staph
- Cephalosporins
- Semi-synthetic penicillins
- Aminoglycosides
- Tetracyclines
- Levofloxacin, Ciprofloxacin.
- For Gram ⊖ → Fluoroquinolones, AG's

- For Corynebacterium
  ↓
  Erythromycin

active space

RECURRENT INFECTIONS

22:35

- Treatment ⟶

i) Intranasal mupirocin ⟶ for 1 wk BD / month

ii) Clindamycin. ⟶ 300mg/day x 3 months

iii) Rifampicin ⟶ 600mg OD x 10 days.

active space

Cutaneous Fungal Infections (Part - 1)

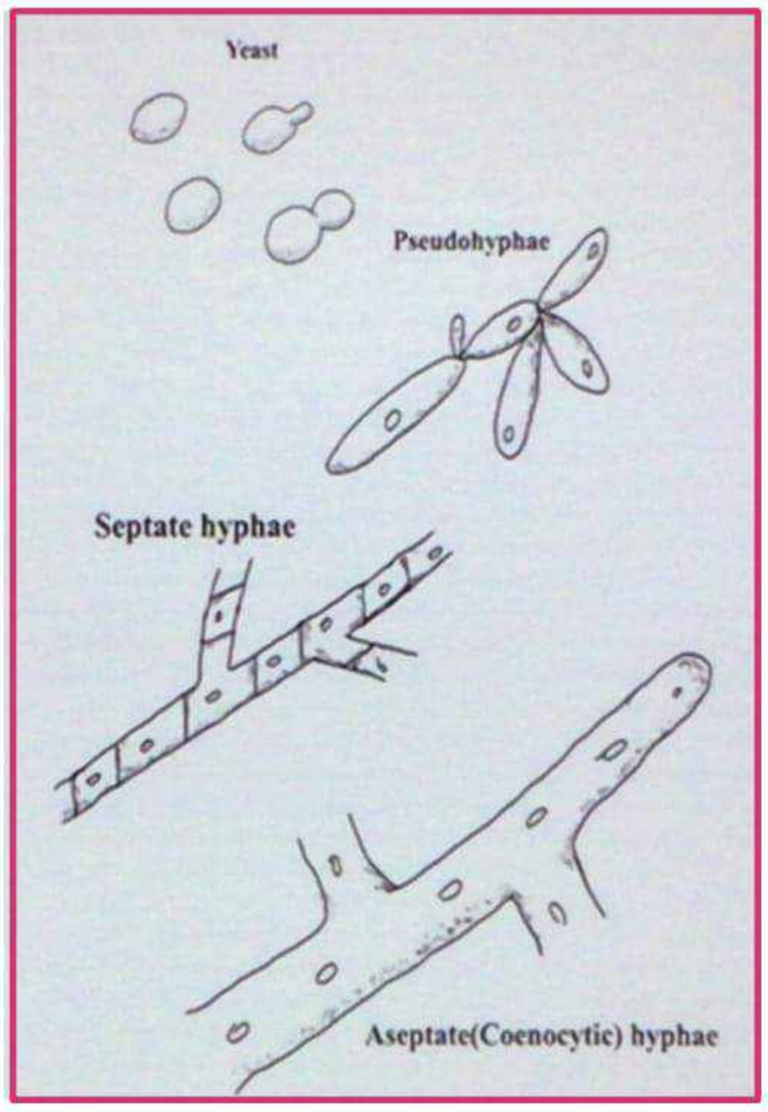
Fungus classified into

Moulds

- Nucleated long filaments a.k.a hyphae
- collection of hyphae → Mycelium.
- Dermatophytes

Yeast

- unicellular oval or globose shaped cells which divide by budding
- form pseudohyphae
- Malassezia, Candida.



Dimorphic fungus :

- Exist both as moulds and yeast.
- Eg : Sporotrichosis.

CLASSIFICATION OF CUTANEOUS FUNGAL INFECTIONS

04:30

1. Superficial
2. Subcutaneous
3. Systemic
4. Oppurtunistic

Superficial	Subcutaneous	Systemic	Oppurtunistic
• which affect superficial layers of skin. • person to person contact • fomites • fallen hairs	• Dermis and Subcutaneous layers are involved • Implantation following injury / abrasion	• involvement of internal organs Pulmonary Route → Inhalation.	• Patients with immunosuppression

SUPERFICIAL FUNGAL INFECTIONS

07:42

- limited to outermost layers of the skin, hair, nail and mucous membrane

Superficial	Cutaneous
• Stratum corneum	• Inflammation ⊕
• Inflammation - Minimal	• Eg :
• Eg : Pityriasis versicolor Tinea nigra Piedra	Dermatophytes Candida.

active space

P. versicolor

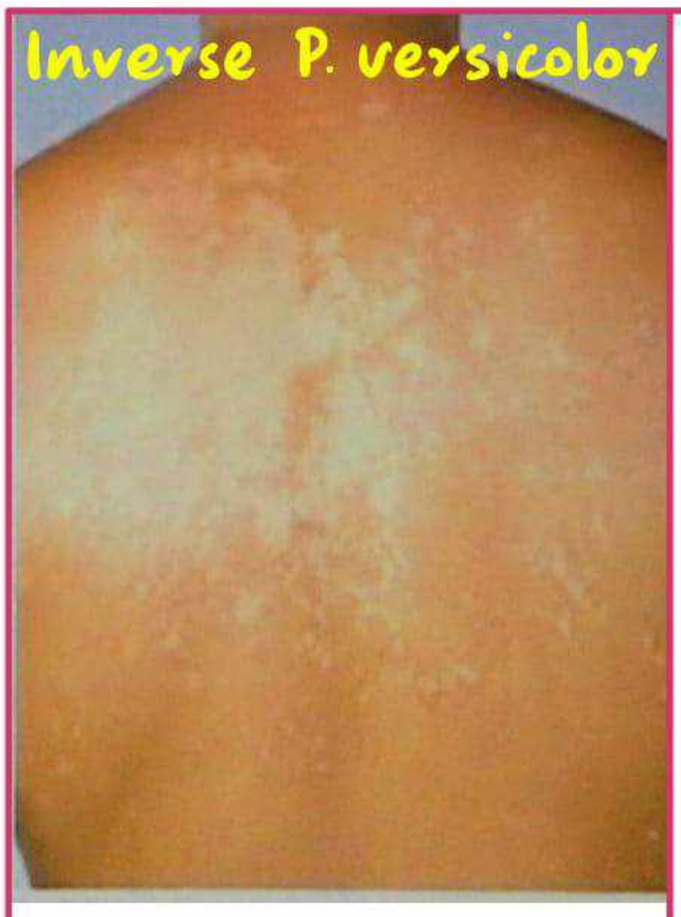
- Superficial fungal infection
- Affects stratum corneum →
  - Minimal inflammation
  - usually asymptomatic
- Organism : Malassezia globosa > furfur > sympodiasis.

(Yeast ⇌ Mycelium)

- Site : i) Upper trunk
  ii) Chest

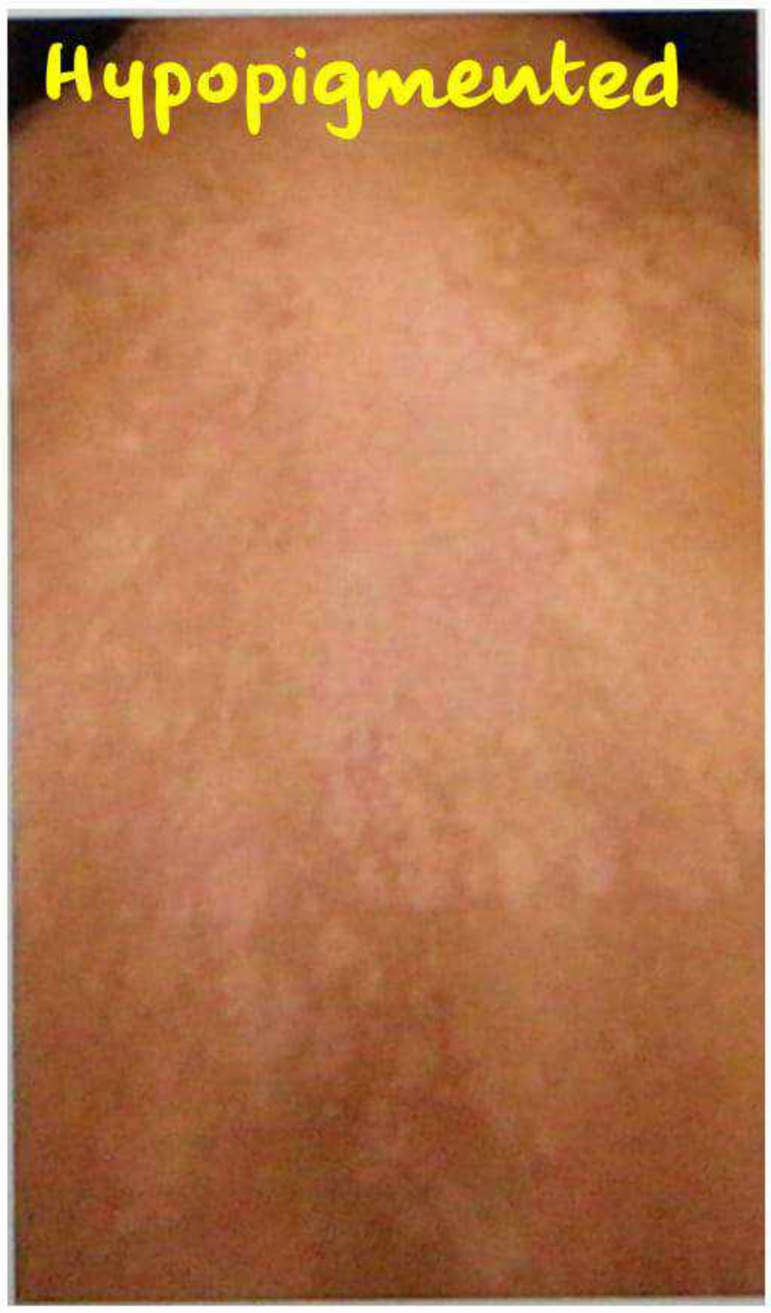
} increased density of sebaceous glands ↓ adolescents of young adults.

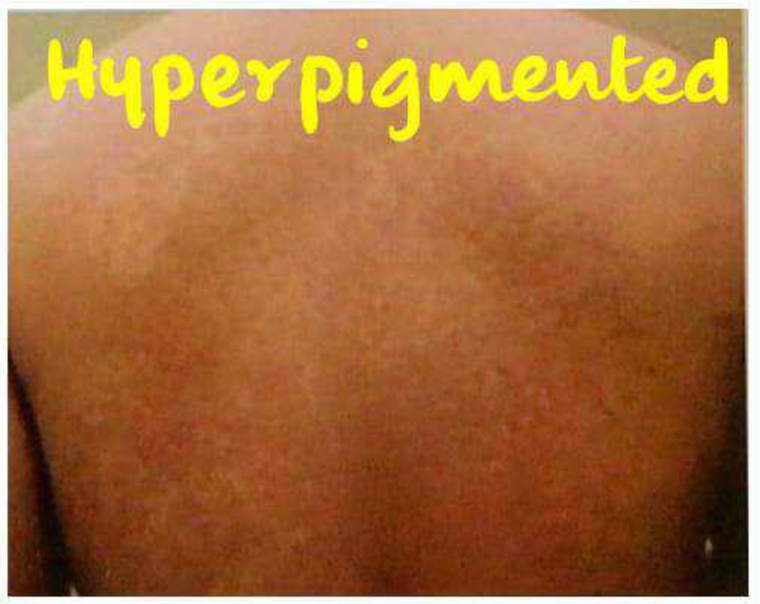
Inverse P. versicolor ⟶ seen in extremities.

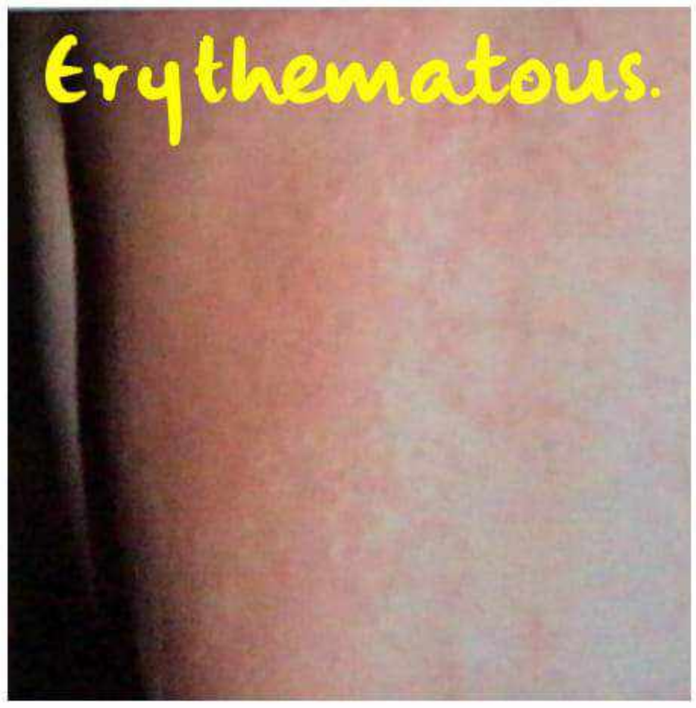


- Discrete macules, which coalesce together on upper trunk and chest
- Hypopigmented, Hyperpigmented, Erythematous. ⟶ versicolor
- Pityriasis ⟶ Scaling (Fine branny scales)
- Nail Scratch Sign / Besnier's Sign.

P. versicolor → various colour





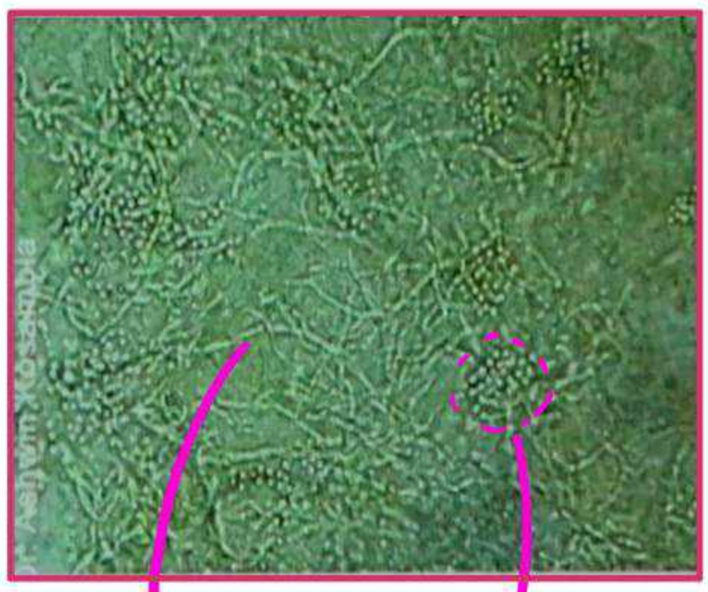


- P. versicolor → Asymptomatic.
- Hot humid climate → ↑ Malassezia
  → Recurrence.
- Hypopigmentation → due to production of carboxylic acid/ Azelaic acid
- Hyperpigmentation → Giant melanosomes.

active space

Diagnosis :

i) KOH Test :



Mycelium → Spaghetti

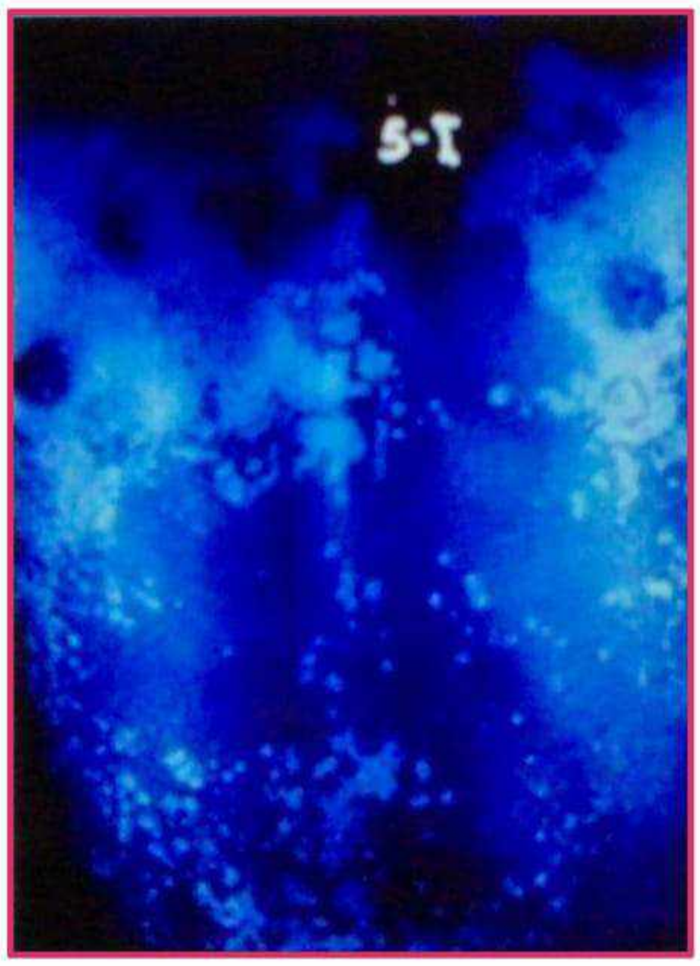
Yeast → Meatballs

- Spaghetti and meat ball appearance
- Banana and Grapes appearance.

→ Mycelium form - Pathogenic.

ii) Woodslamp :

- Yellowish fluorescence.



active space

Treatment :

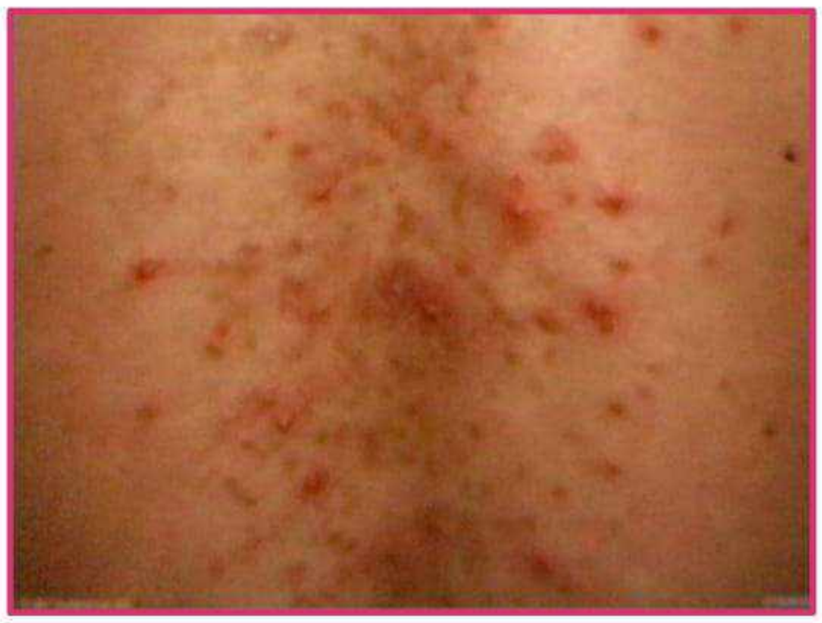
- Counsel the patient → Recurrent
- Hypopigmentation does not improve soon
- **Azoles**

i) Topical → • Clotrimazole
• Luliconazole
• Sertoconazole
• Zinc pyrithrone, Selenium sulphide → Shampoos / lotions

ii) Oral → Fluconazole
Itraconazole
Ketoconazole
} Recurrence, Extensive disease.

i) Malazzesia Folliculitis :

- Monomorphic follicular papules and pustules on trunk of young adults / adolescents.
- Itching ⊕
- No pain, No Dandruff



active space

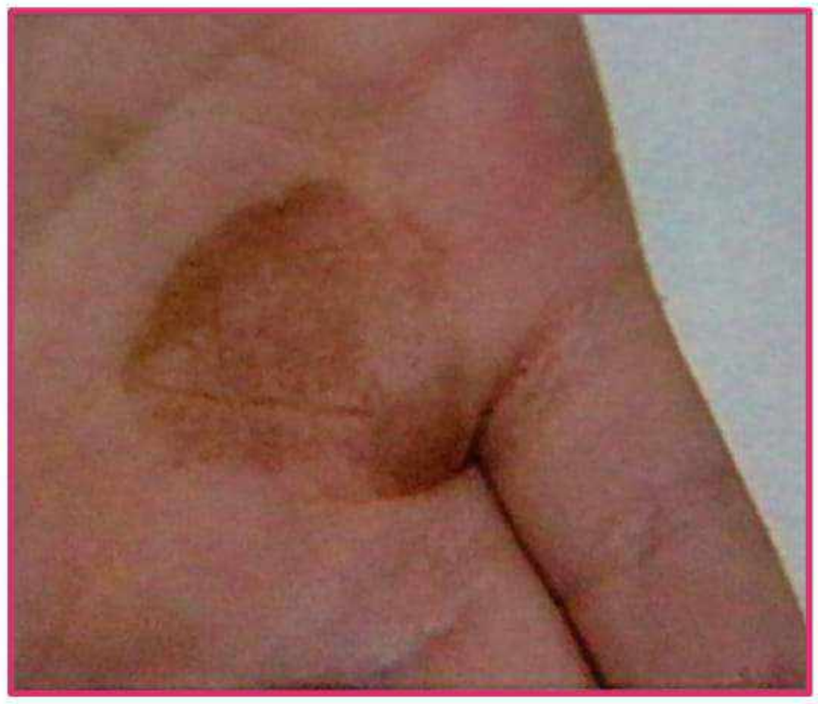
ii) Seborrheic dermatitis

iii) Confluent Reticulate Papillomatosis. (CRP)

TINEA NIGRA

26:40

- caused by Hortae werneckii / Exophilia werneckii
- Asymptomatic hyperpigmented lesion on palmar aspect of hand



Treatment

- Salicylic acid
- Topical Antifungals

PIEDRA

28:15

Black Piedra

White Piedra

active space

Black Piedra	White Piedra
• Hard gritty nodules which are adherent to hair shaft • Mainly on scalp • Organism : Piedra Hortae	• Nodules are soft and are white to grey in colour • Other hair bearing areas, moustache, beard, axillary, scalp • Trichosporosis nodosa.

DERMATOPHYTOSIS

30:45

- a.k.a Tinea
- caused Dermatophytes (Keratinophilic)
- involves skin (stratum corneum), hair and nails.
- Anthrophilic : Human to Human → Less inflammatory
  Geophilic : Soil to Human
  Zoophilic : Animal to Human → More inflammatory.

Pathogenesis :

- Contact → Fungus adheres to cell → Penetration → Invasion → disease.

active space

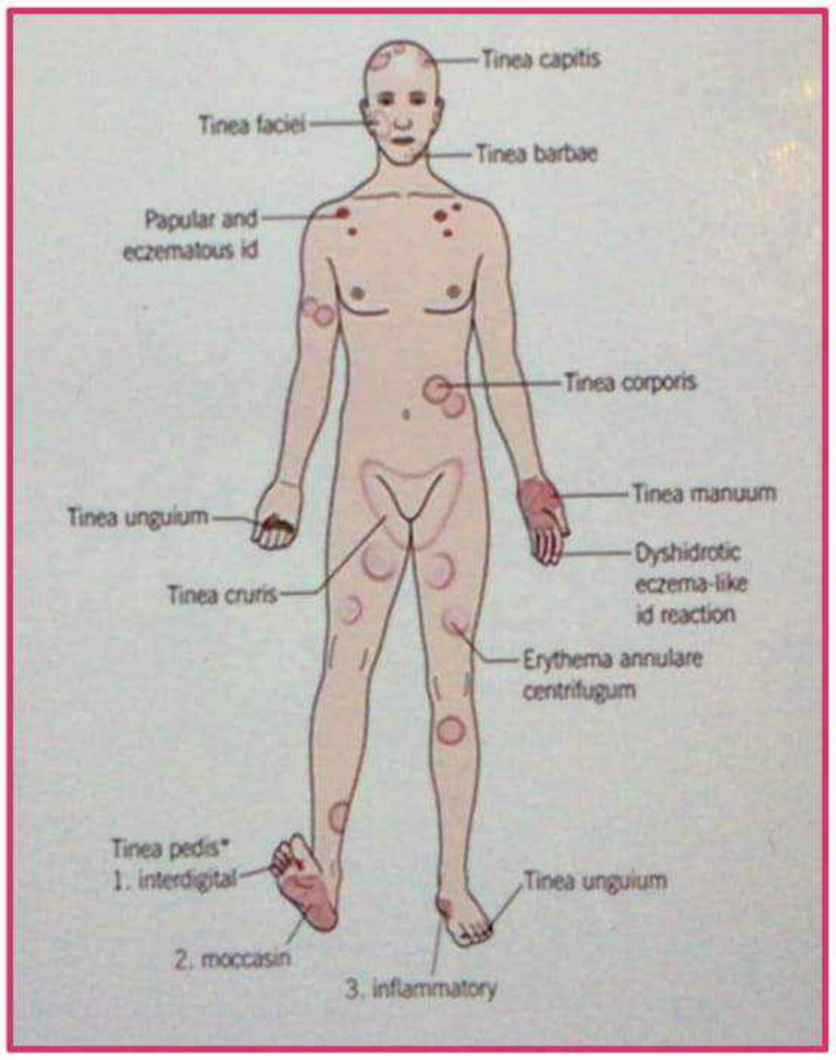
Transmission :

- Person to person
- Fomites
- Fallen Hairs

Species :

	Skin	Hair	Nails
i) Trichophyton	+	+	+
ii) Epidermophyton	+	×	+
iii) Microsporum.	+	+	×

Tinea Classification by Site :



1. Tinea capitis
2. Tinea facei
3. Tinea barbae
4. T. corporis
5. T. manuum
6. T. unguium
7. T. cruris
8. T. pedis.
9. Id Reactions.

active space

Cutaneous Fungal Infections Part 2

DERMATOPHYTOSIS / TINEA

00: 25

- caused by Dermatophytes (Keratinophilic)
- involves skin (Stratum corneum), hair and nails.
- 3 specifies :

  i) Anthrophilic : Human to Human - Less inflammatory

  ii) Geophilic : Soil to Human

  iii) Zoophilic : Animal to Human - More inflammatory

Pathogenesis

- contact ⟶ Adherence ⟶ Penetration ⟶ Invasion ⟶ Disease

Transmission

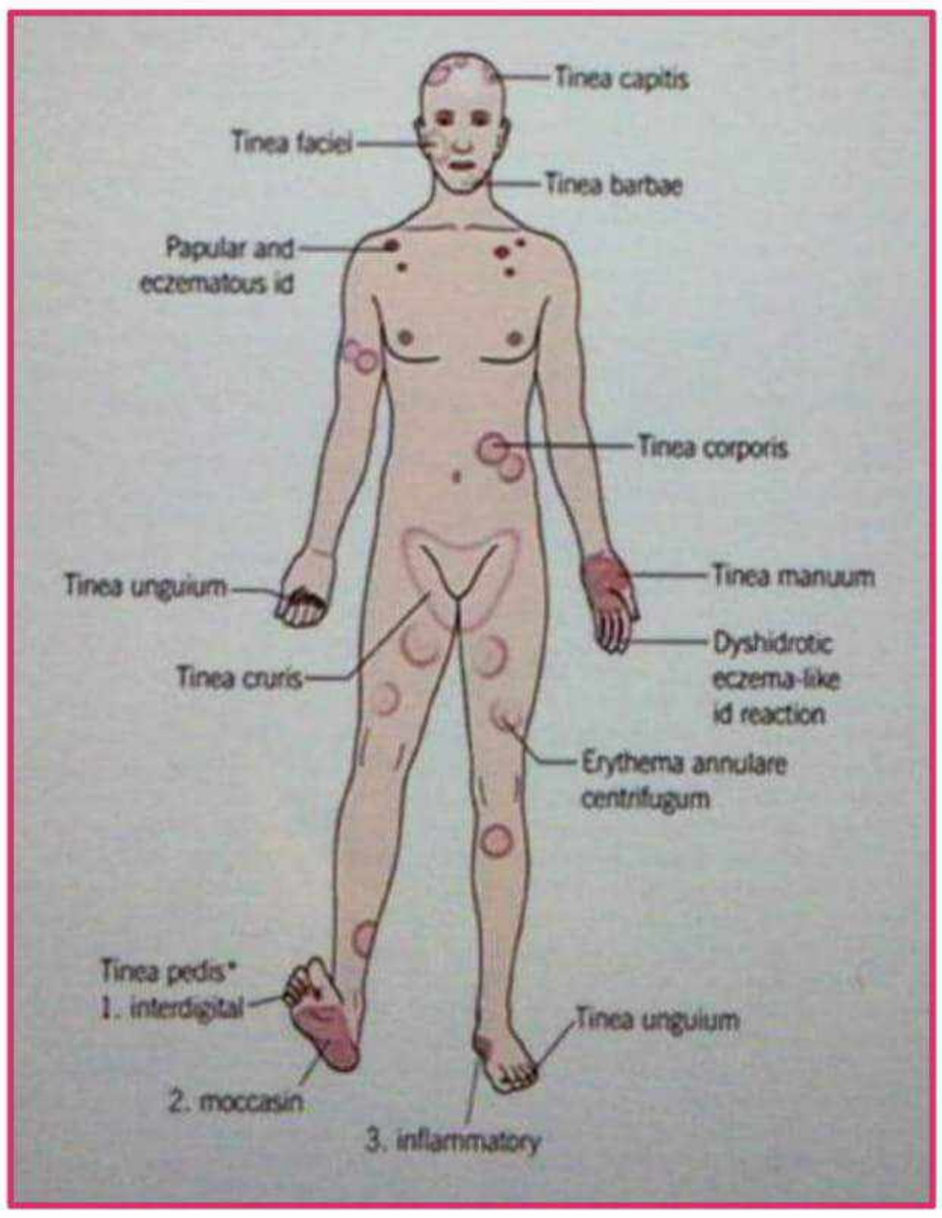
- Person to person
- Fomites
- Fallen Hairs (Tinea capitis)

active space

Species

	Skin	Hair	Nails
1. Trichophyton	+	+	+
2. Epidermophyton	+	×	+
3. Microsporum	+	+	×

Classificalion by Site :



1. Tinea capitis
2. Tinea facei
3. Tinea barbae
4. Tinea corporis
5. Tinea manuum
6. Tinea unguim
7. Tinea cruris
8. Tinea pedis
9. Id reactions.

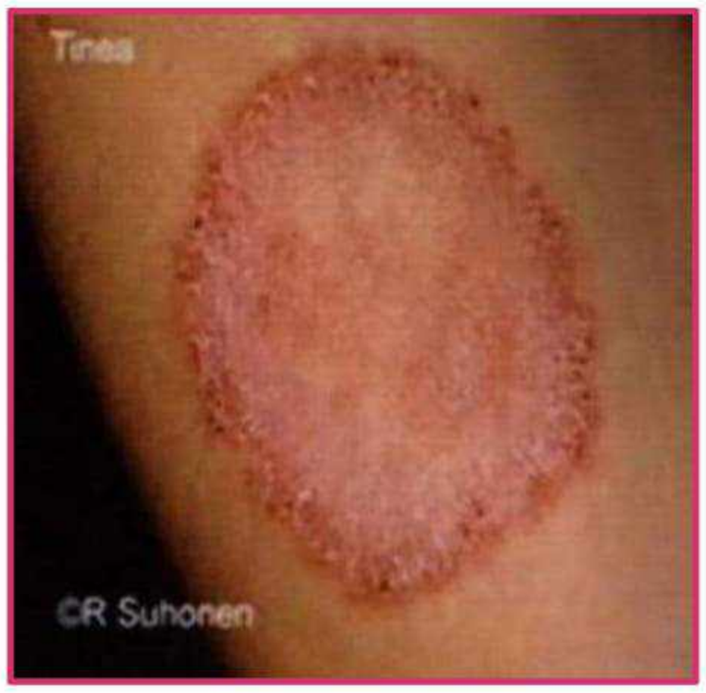
active space

CLASSICAL LESION OF TINEA

08:25

- Annular
- Central clearing
- Peripheral activity -

  Scales, vesicles, pustules, crusting
- Extends peripherally / centrifugally



When there is :

i) Central scarring : Lupus vulgaris

ii) Central clearing : Tinea

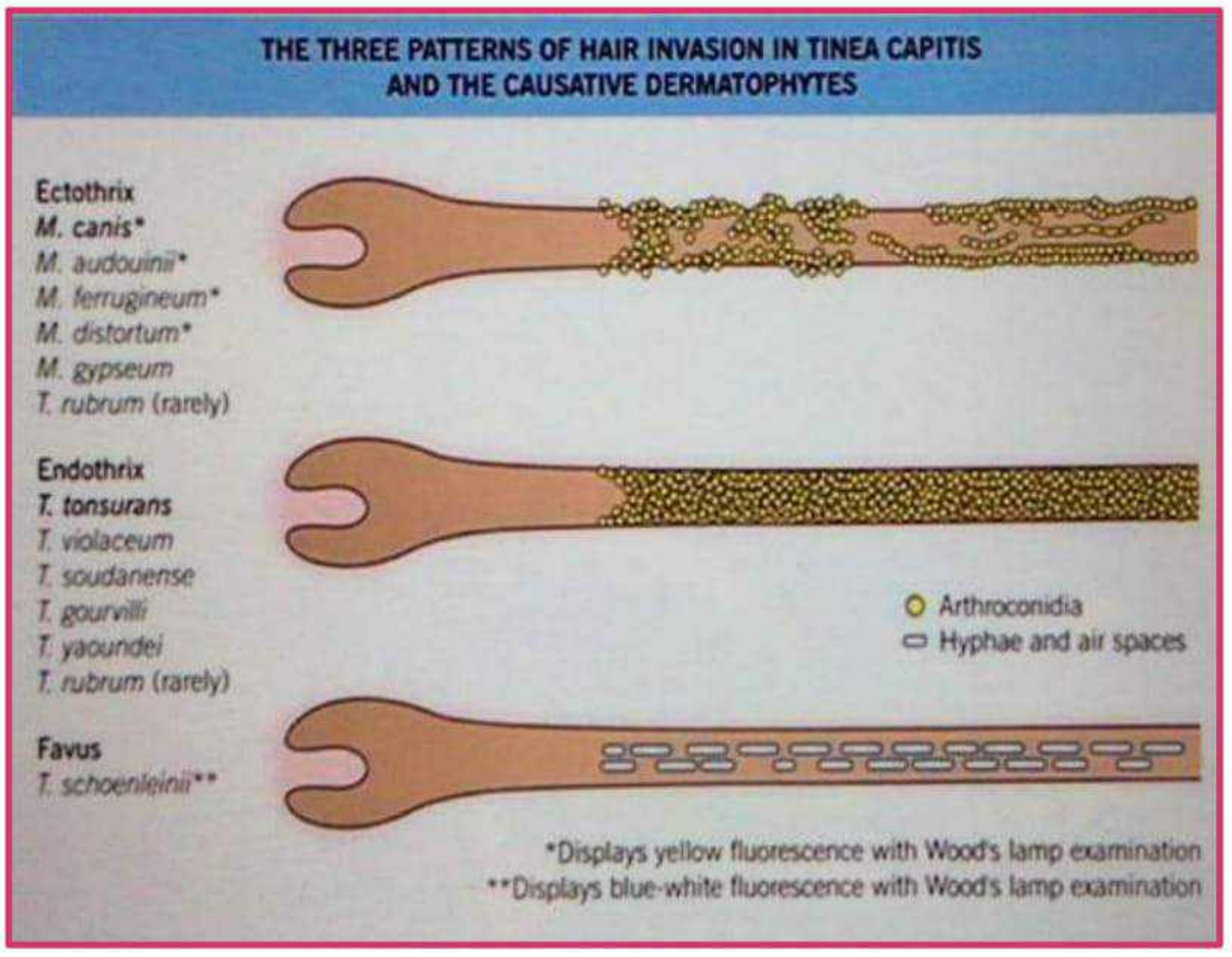
iii) Central crusting : Leishmaniasis

TINEA CAPITIS

11:00

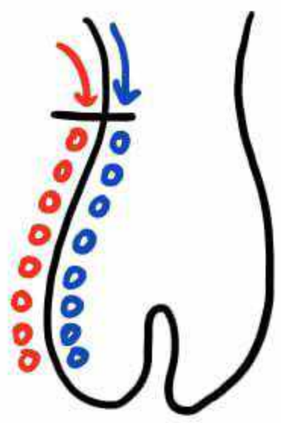
- seen in Scalp and Hair
- more in children (MC mycoses in children)
- Species : Microsporum , Trichophyton
- MC Species : i) T. Tonsurans - in Developed nation

  ii) M. canis - in Developing nation



i) Ectothrix :

- Hair is affected from outside the hair shaft
- caused by :
  - i) M.canis
  - ii) M.audonnei
- Fluorescence ⊕



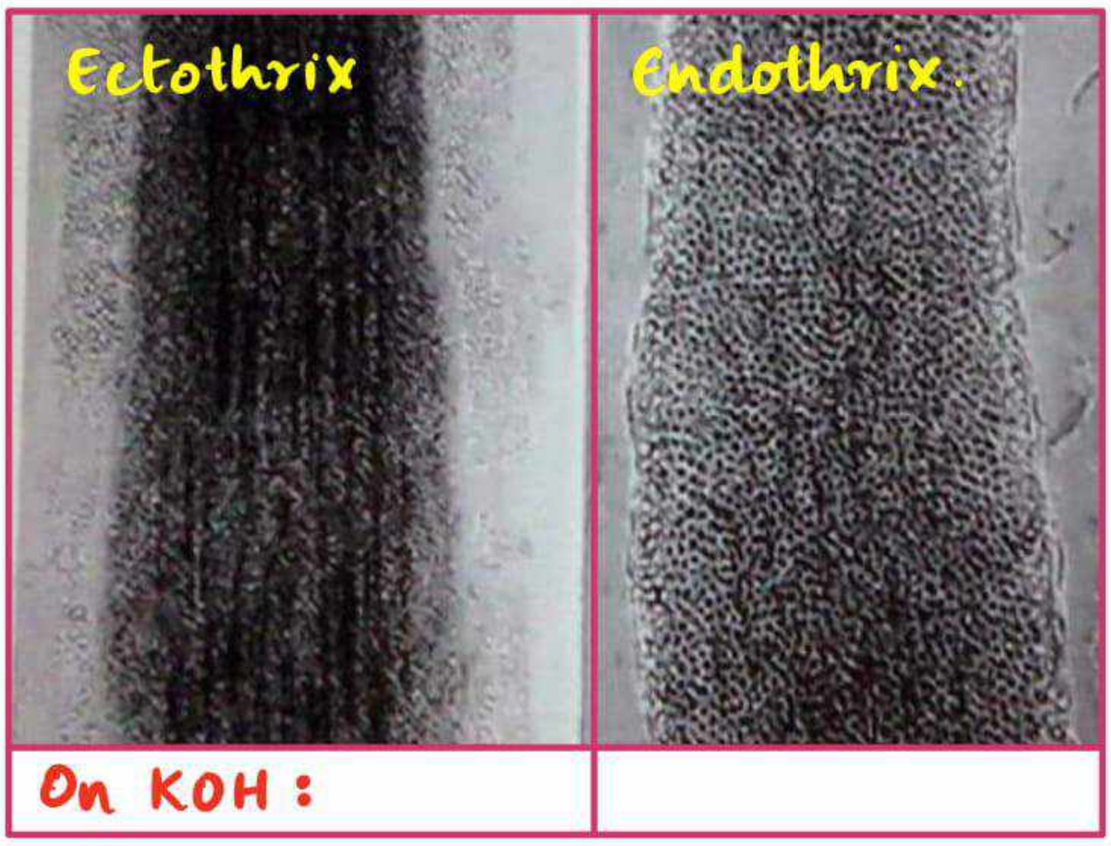
ii) Endothrix :

- Hair is affected from inside the shaft.
- caused by Trichophyton species
- Donot fluorescence.

active space

iii) Favus :

- air spaces along with regularly septate hyphae
- caused by Trichophyton schoenlenii
- Fluorescence ⊕



Tinea Capitis : Classification

Clinical Classification

Non-Inflammatory

- Grey patch
- Black dot

Inflammatory

- Kerion
- Favus.

Grey Patch

- circular patch
- partial alopecia
- Broken off hairs
- Gray fine scales

↳ Athroconia / spores

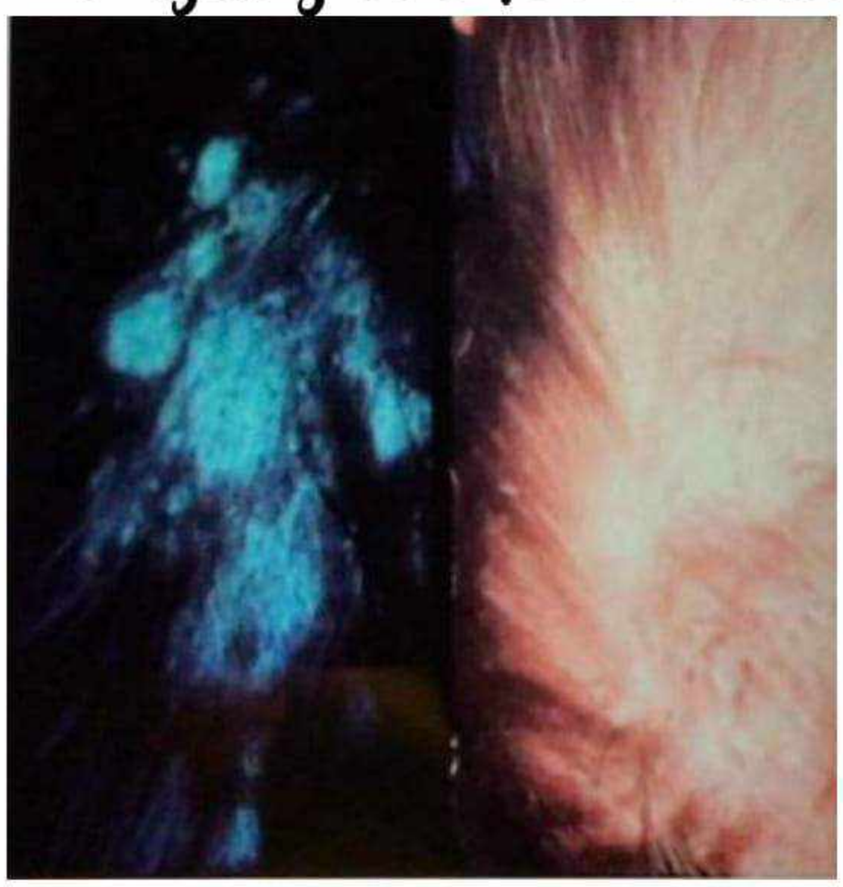
- Ectothrix
- M. ferruginum -

  donot fluorescence.
- Responsible for outbreaks in

  school children

Wood's Lamp:

- Bright green fluorescence.



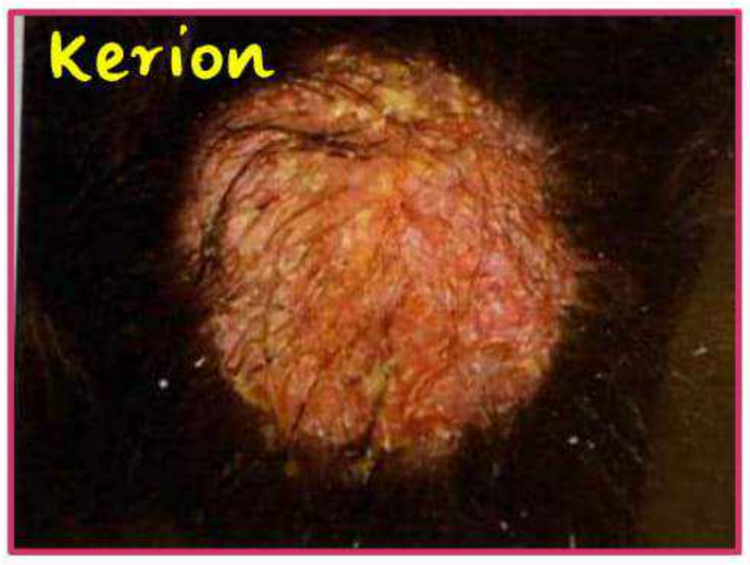
Black Dot

- Angular patches / finger like projections
- Black dots

- Black dots → Swollen hair shafts
- Hair breaks at surface of scalp
- Endothrix. (caused by Trichophyton species)
  - Tonsurans (MC)
  - violaceum
  - Sudanese
- Not fluoresce under wood's lamp

Kerion :

- Inflammed, indurated boggy swelling studded with pustules, crusting and broken off hairs.
- Inflammatory : caused by
  - i) T. verrucosum
  - ii) T. ferugineum
- On KOH, C/S - Negative
- 2° features : i) Lymphadenopathy - cervical or occipital
  - ii) 2° Bacterial infection
  - iii) Crusting, matting of hair



Favus :

- caused by T. schoenlenii
- Air spaces ⊕
- Yellowish cup shaped crusts : SCUTULA

  (crusts are aggregation of athroconidia + Epithelial debris)
- Inflammatory Types : Scarring
- Non-Inflammatory Type : No scarring.
- Shows fluorescence - Dull green.

MANAGEMENT OF TINEA CAPITIS

31:09

i) Topical management :

- No Role in treating
- Only helps in stopping community infection (community Rx)

ii) Systemic management :

- Griesofulvin : TOC ⇒ 15-20mg/kg
- Terbinafine → ⊗ Microsporum, ✓ Trichophyton.
- Itraconazole
- Fluconazole ⊗

active space

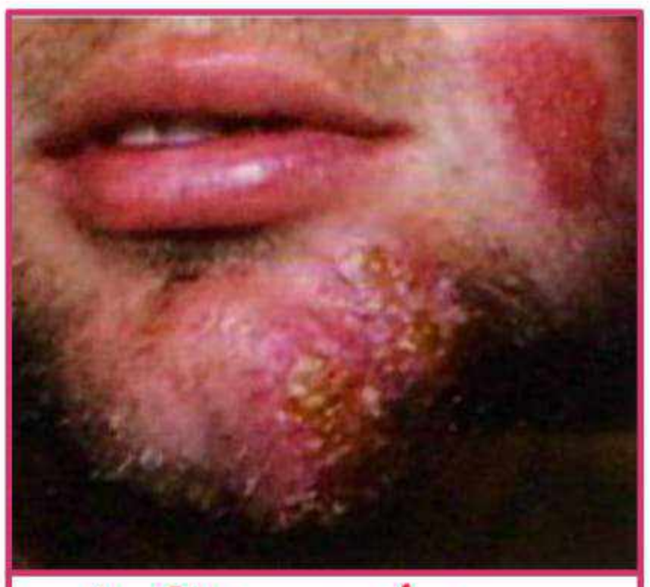
TINEA BARBAE

34:06

- involves beard and moustache area of adult males.
- Inflammation of Terminal hair

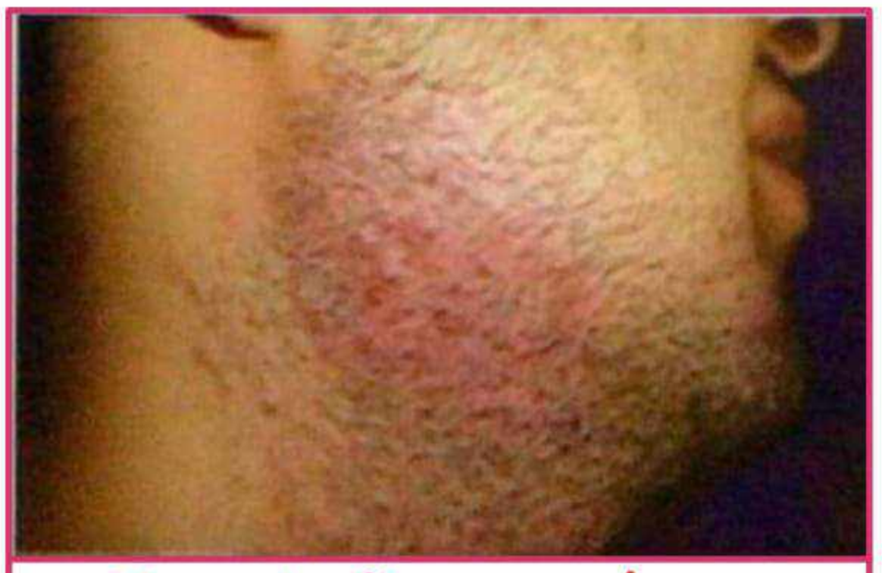
In Sycosis barbae :

- No pain
- Itching
- Loose hair.



Inflammatory

- vesicles
- crusting
- Scarring



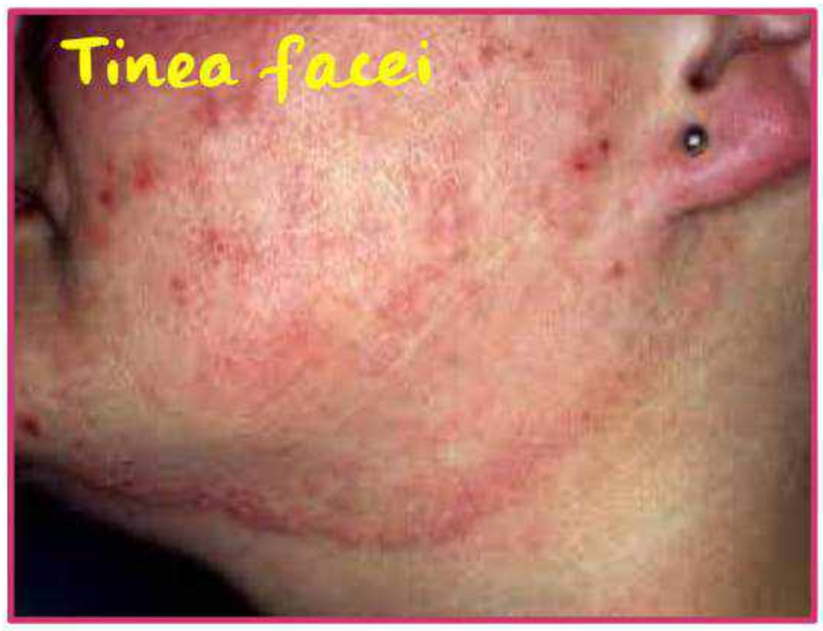
Non-Inflammatory

- Red scalp
- Patches.

TINEA FACEI

36:26

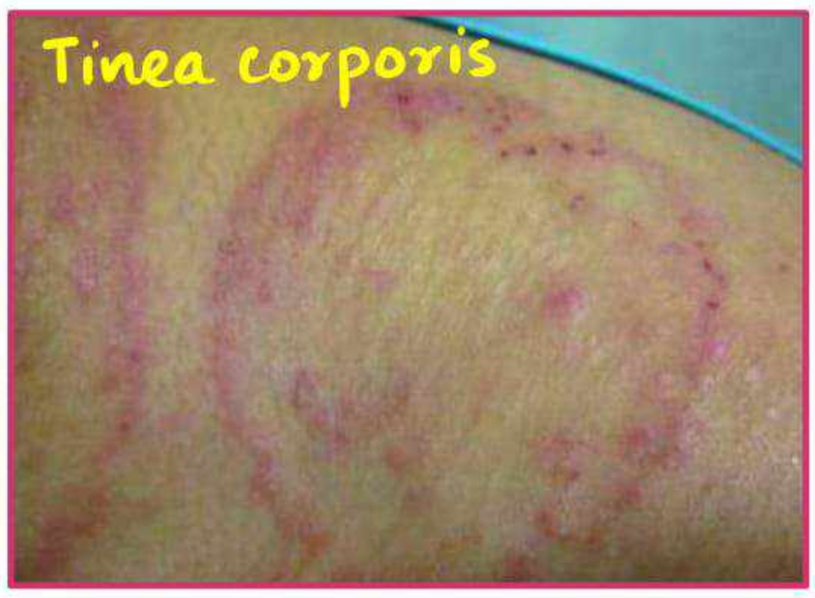
- affects face, upper limb, chin of females
- usually associated with tinea of other body parts.
- Immunocompromised states ⟶ sole feature.



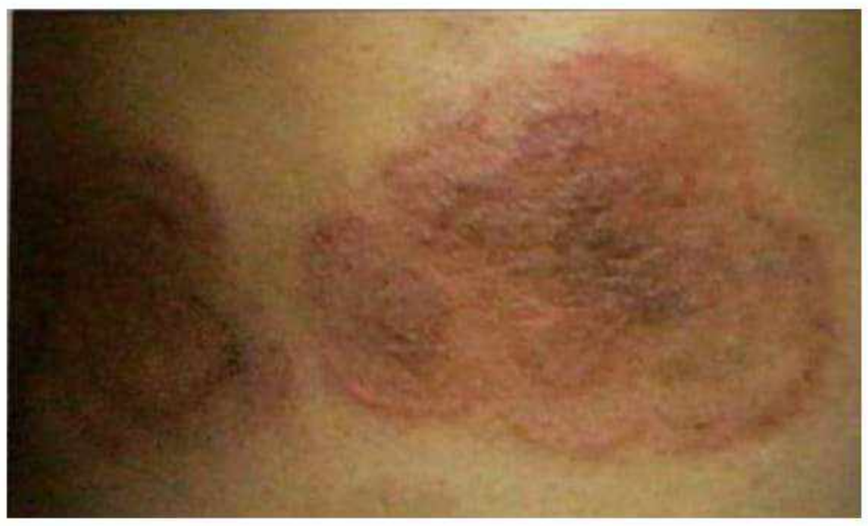
TINEA CORPORIS

37:33

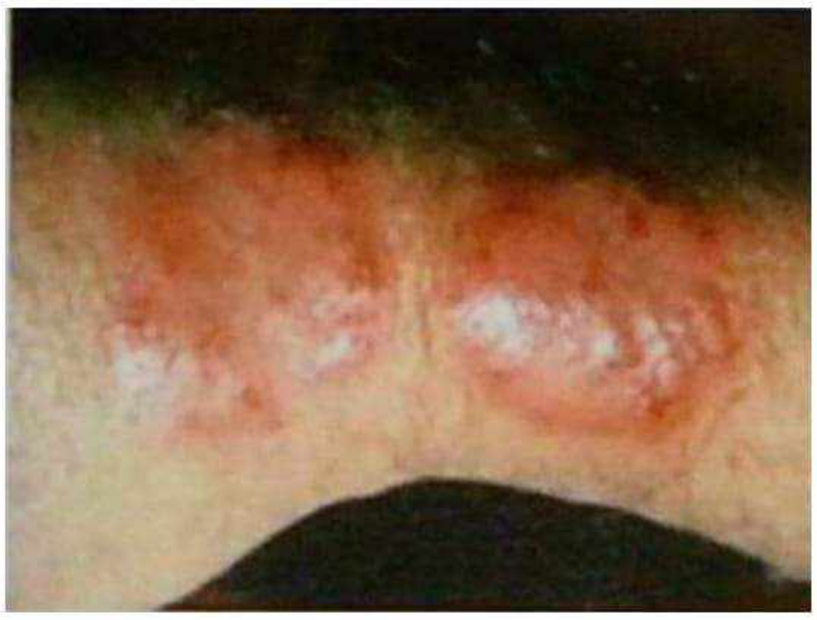
- Tinea of body excluding the special sites.
- Caused by all species
- (MC) T. mentagrophytes
- Earlier (MC) → T. rubrum.



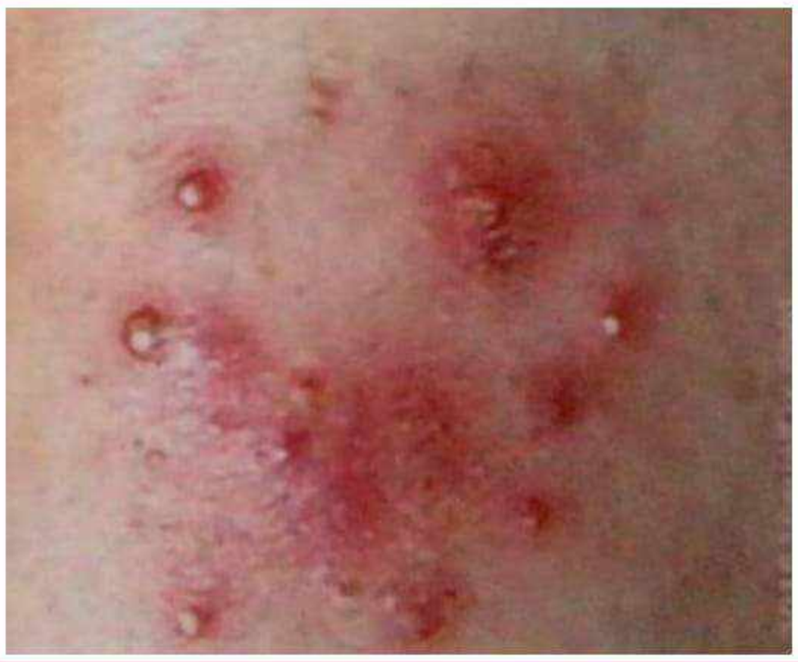
- I.P : 1-3wks.
- Special Types :



- concentric Rings
- T. circinata / imbricata.
- T. concentricum



- caused by Tinea profunda
- Inflammation is going to dermis, subcutis, nodules.



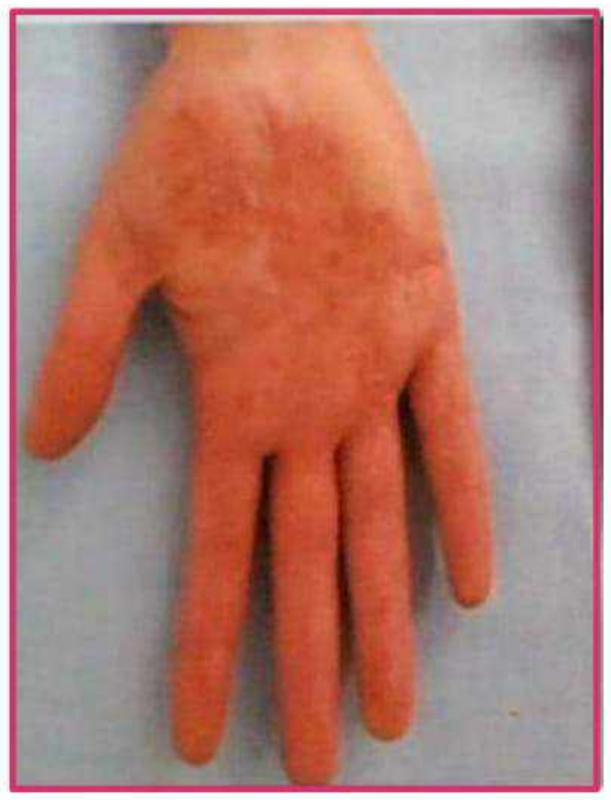
Majochi's granuloma

- Granulomatous reaction around follicle areas.

TINEA MANUUM

42:00

- Affects palmar aspect of hands.
- No sebaceous glands.
- It can :
  - i) Inflammatory : vesicular type / dyshidrotic
  - ii) Non-inflammatory : dry scaly patches.



active space

TINEA PEDIS

43:30

- Tinea of feet
- seen in people wearing tight footwear.
- Hyperhidrosis
- Atheletes foot
- Types :

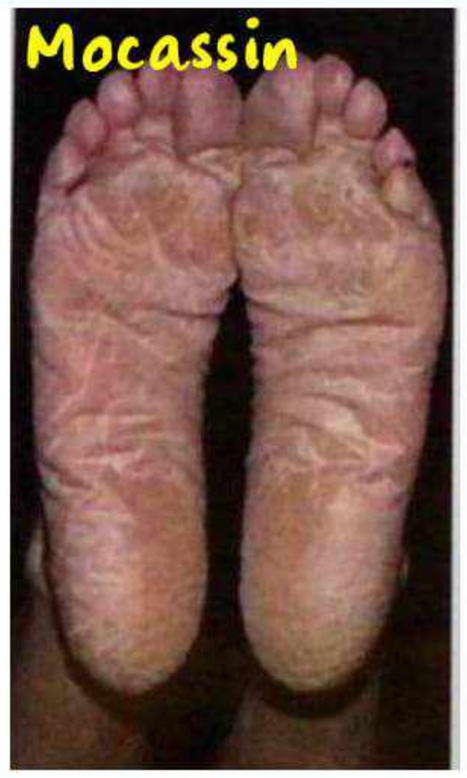
  i) Interdigital :

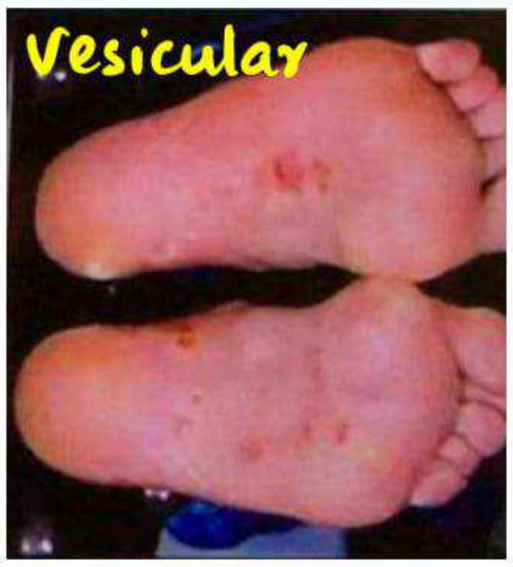
  - (MC) Type of T. pedis
  - 4th web space

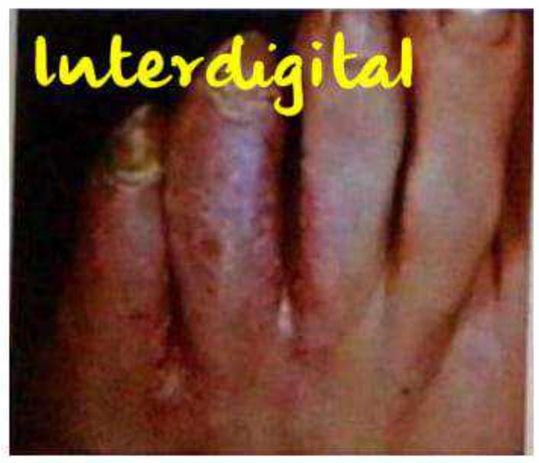
  ii) Hyperkeratotic / Mocassin

  iii) Vesicular / Inflammatory

  iv) Ulcerative.

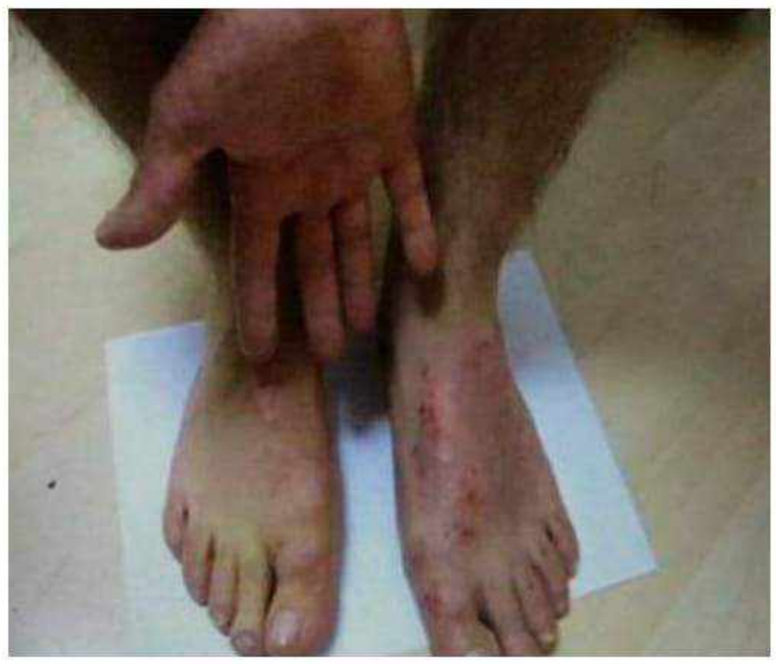






Two feet One hand Syndrome :

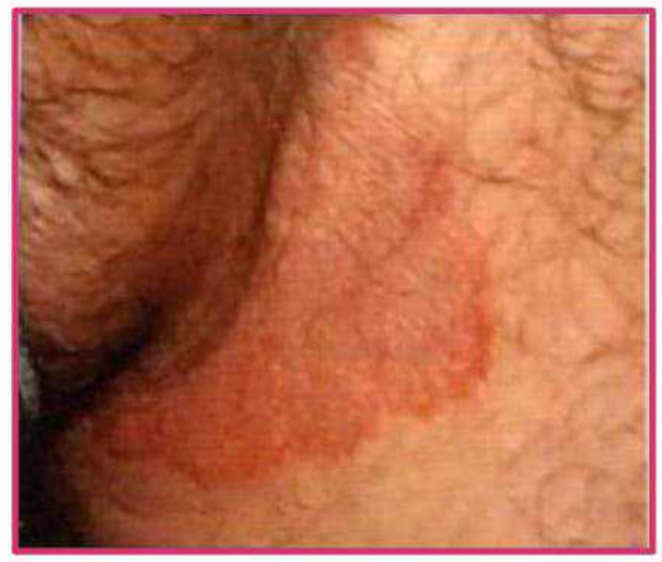
- caused by Tinea Rubrum.
- one hand is spared.



TINEA CRURIS

47:30

- affects groins, genital area
- a.k.a Dhobi itch / Jocker's itch.
- Less scaling



TINEA INCOGNITO

49:20

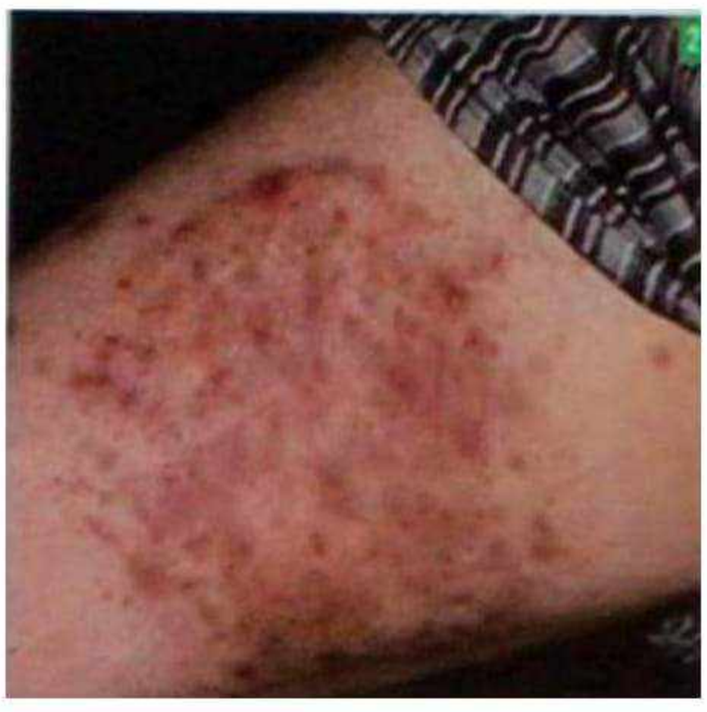
- a.k.a Steroid Modified Tinea
- patient applies OTC products containing steroids

↓

↓ Inflammation

↓

Relapse on stopping the cream.

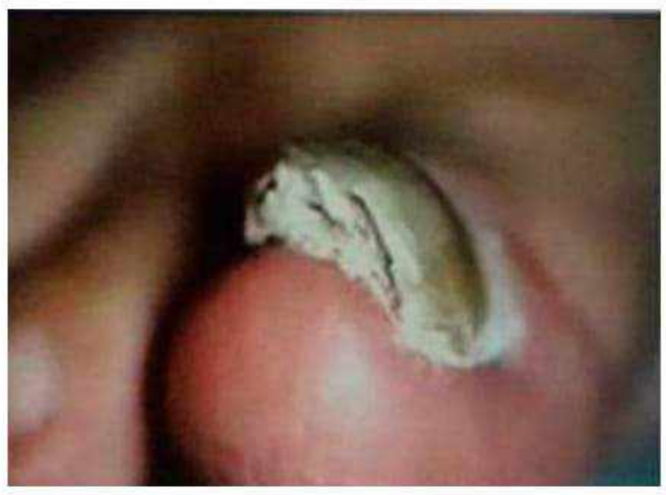


- ↓ Scaling
- ↓ Inflammation
- Bluish to brownish.

TINEA UNGUIUM

51:44

- infection of toe nails > fingernails.
- Onychomycosis : nail infection because of fungus -



i) Dermatophyte : T. unguium

ii) Candida

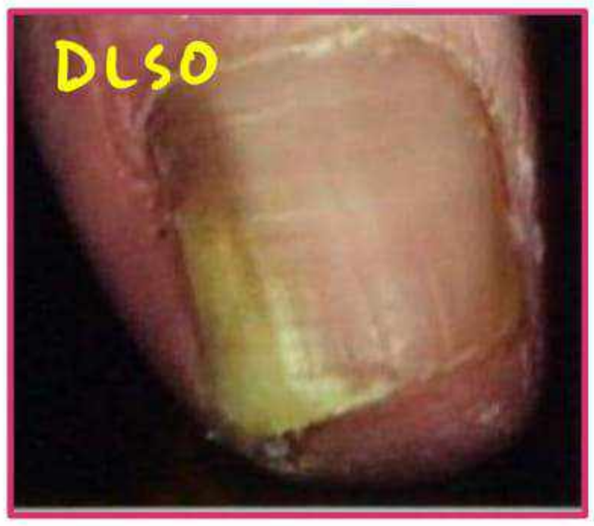
iii) Other fungus

Clinical features :

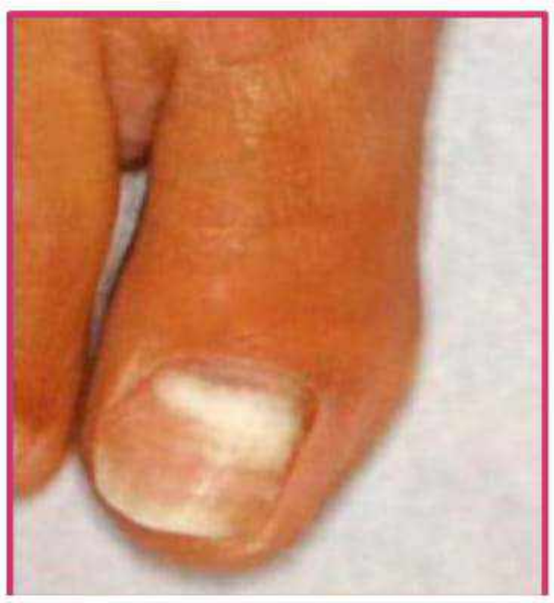
- Discoloured nail
- Subungual hyperkeratosis
- Separation of nail plate from nail bed

Clinical Classification

i) Distal Lateral Subungual Onychomycosis : (MC)

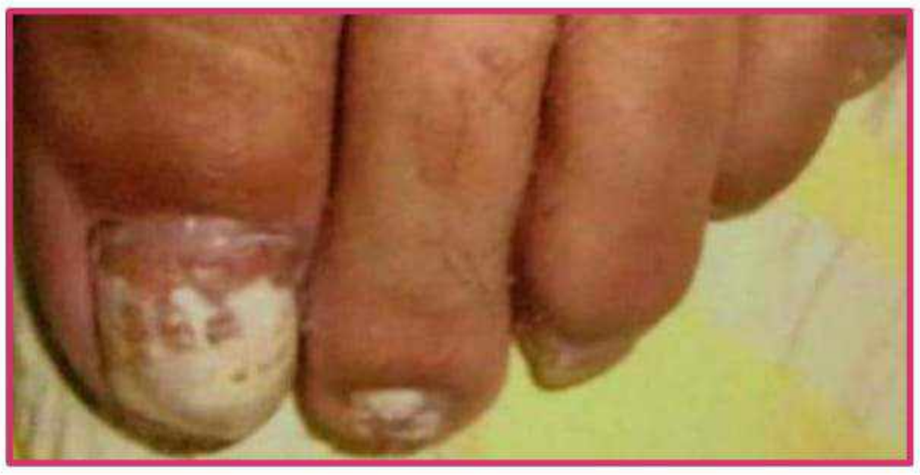


ii) Proximal Subungual Onychomycosis :



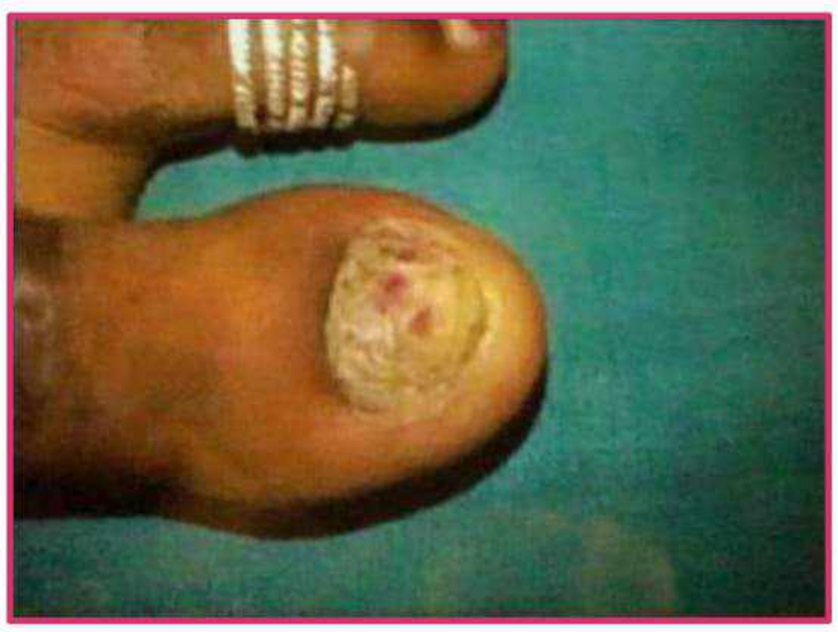
- More common in HIV patients.

iii) Superficial White Onychomycosis :



- White crumbling material which can be scraped off.

iv) Total Dystrophic Onychomycosis :



- a.k.a Endonyx.

vs Psoriatic nail : • No Pitting
• Oil drop sign is ⊖ } in Onychomycosis

Treatment of T.unguium

i) Topical Rx :

- Nail laquers : i) Amrolfine
ii) Cyclopirox olamine

ii) Systemic Rx :

	Dose	Duration: Fingernails	Duration: Toenails
• Fluconazole →	150-200mg/wk	6months	12 months
• Terbinafine →	250mg OD	6wks	12wks
• Itraconazole →	100-200mg/day	2-3months	3-4 months

Pulse Therapy : High dose of drug for short period.

↳ 400mg OD (200mg BD) of Itraconazole for 1wk/month for

i) finger nails ⟶ 2-3 cycles

ii) Toenails ⟶ 4-5 cycles.

DERMATOPHYTID

1:04:30

- Hypersensitivity Reaction of Body (Type IV HSR)
- **Criteria :**
  - Proven focus of Tinea elswhere which becomes inflamed.
    ⟶ seen with kerion, T.pedis
  - Distant eruption away from focus ⟶ Follicular papules, vesicles
  - Spontaneous resolution with resolution of 1° focus.

active space

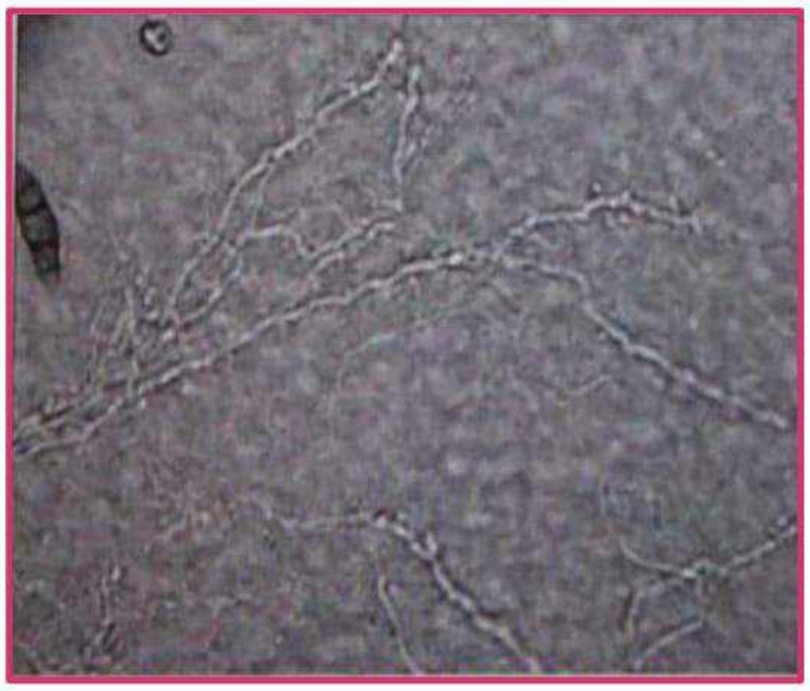
DIAGNOSIS OF DERMATOPHYTOSIS

01:07:20

i) KOH : 10% KOH used.

ii) Fungal culture.

KOH :



- Branching septate hyphae

TREATMENT OF DERMATOPHYTOSIS

01:08:30

1. Topical Rx :

- Azoles → i) Clotrimazole
  ii) Sertaconazole
  iii) Luliconazole
  iv) Eberconazole.
- Allylamines : Terbinafine
- Nail Laquers : i) Amrolfine
  ii) Selenium sulphide.

- Shampoos ⟶ i) Ketoconazole
  ii) Selenium sulphide.
  iii) Ciclopiroxolamine
- Whitfields ointment : Benzoic acid
- Castellani paint

ii) Systemic Treatment :

- Azoles : Itraconazole, Fluconazole
- Terbinafine
- Griesofulvin ⟶ T. capitis.

Condition	Duration
Tinea corporis	4-6 weeks
Tinea cruris	2-4 weeks
Tinea manuum	1-4 weeks
Tinea faciei	4-6 weeks
Tinea pedis	4-8 weeks
Tinea capitis	6-8 weeks
Tinea unguium Finger nails : Toe nails :	 12-24 weeks 24- 36 weeks

- Medications are given 2 wks more than clinical cure
  ↓ Mycological cure

Constituents of Castellani's paint :

- Basic fuschin
- Ethyl alcohol
- Boric acid
- Acetone
- Phenol
- Resocrinol
- Water

Modified Castellani's paint :

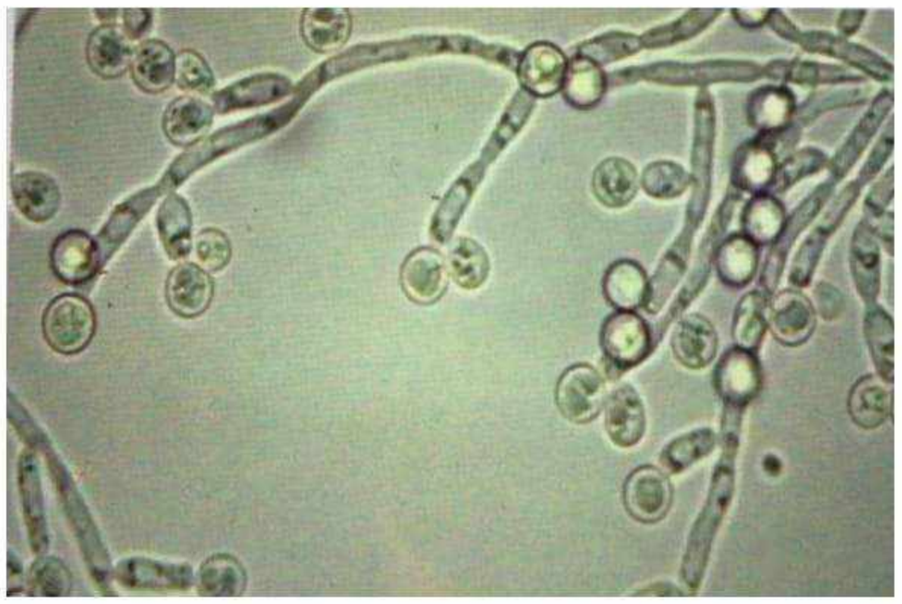
- Colourless
- Less irritating
- Less effective
- doesnot have Basic fuschin.

Cutaneous Fungal Infections Part - 3

CANDIASIS

00:10

- Candida → Yeast
- Candida albicans (MC)
  - Budding yeast,
  - Pseudohyphae.



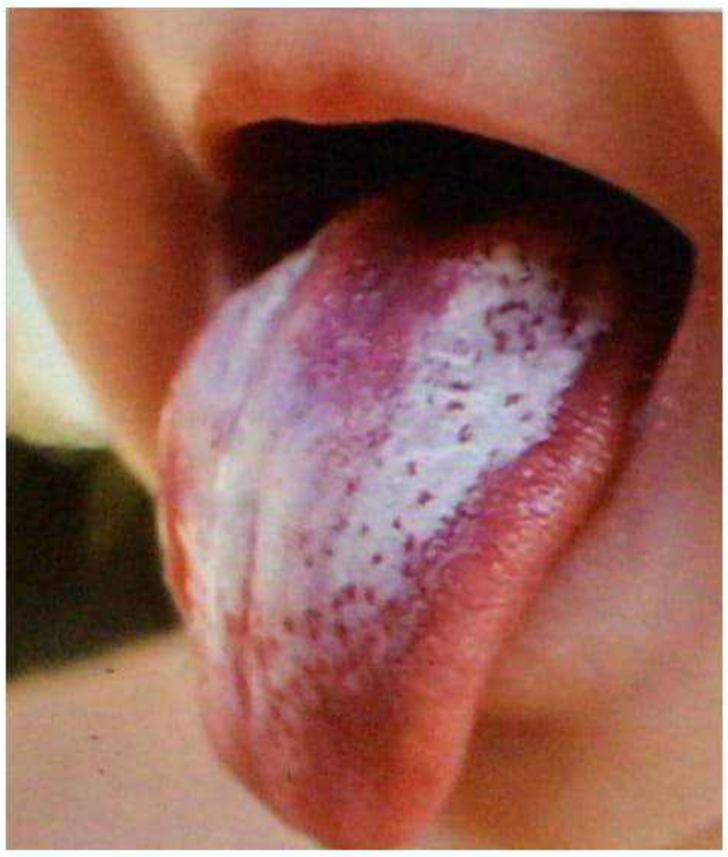
- It affects
  - i) Mucosa → Oral / Genital
  - ii) Nail → Paronychia, Onychomycosis
  - iii) Skin → Intertrigo, Napkin Rash.

ORAL CANDIDIASIS

02:00

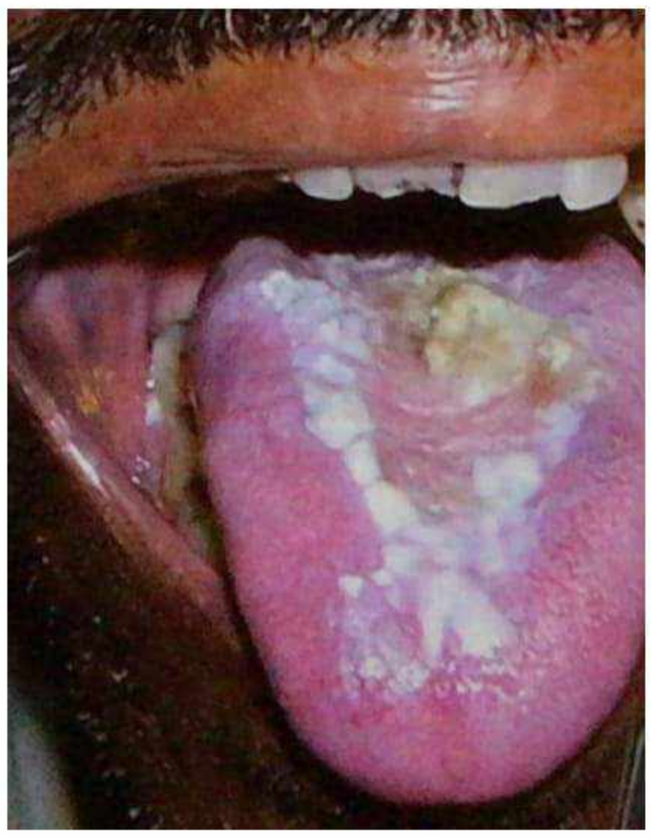
- affects oral mucosa
- affects
  - i) Immunocompetent host : Cheeks, gums and palate
  - ii) If compromised : Tongue, Pharynx, Esophagus, Ulcerative.

Acute Pseudomembranous Candidiasis / Oral Thrush



- white curdy crumbly plaque / patch
- On removal of plaque, erythematous base
- pseudomembrane

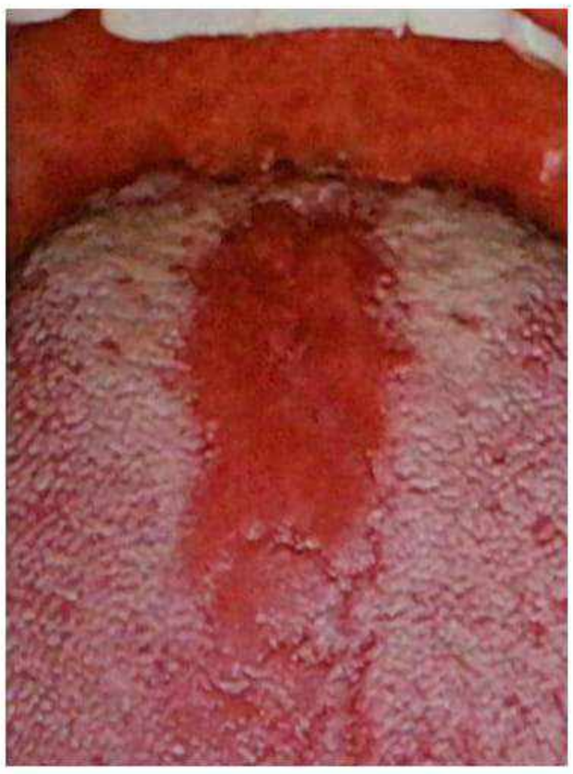
Chronic Plaque Type / Candidal Leukoplakia.



- persisting for long time
  - ↳ Thicker.
- White deposit not scraped away
- Pre-malignant condition.

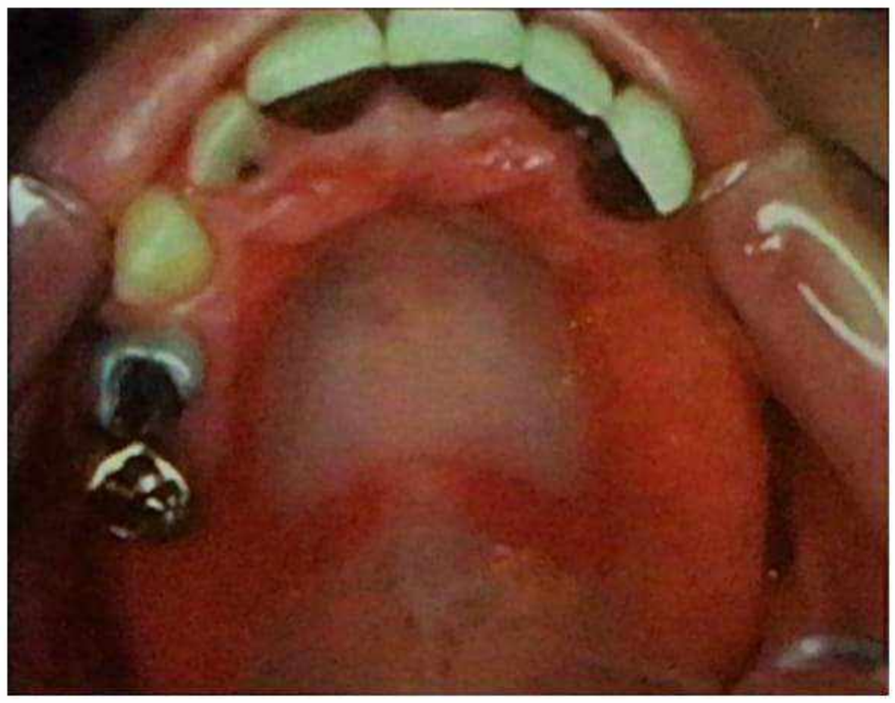
active space

Acute Erythematous Candidosis / Antibiotic Sore Mouth



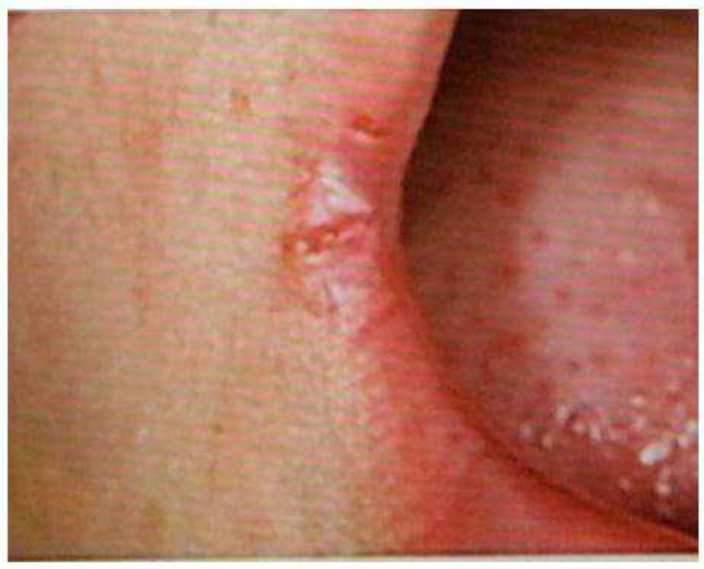
- denuded atrophic erythematous patches dorsa of tongue due to long term Antibiotic use.

Chronic Erythematous Candidosis or Denture stomatitis



- seen in denture bearing areas

Angular cheilitis / Perleche.

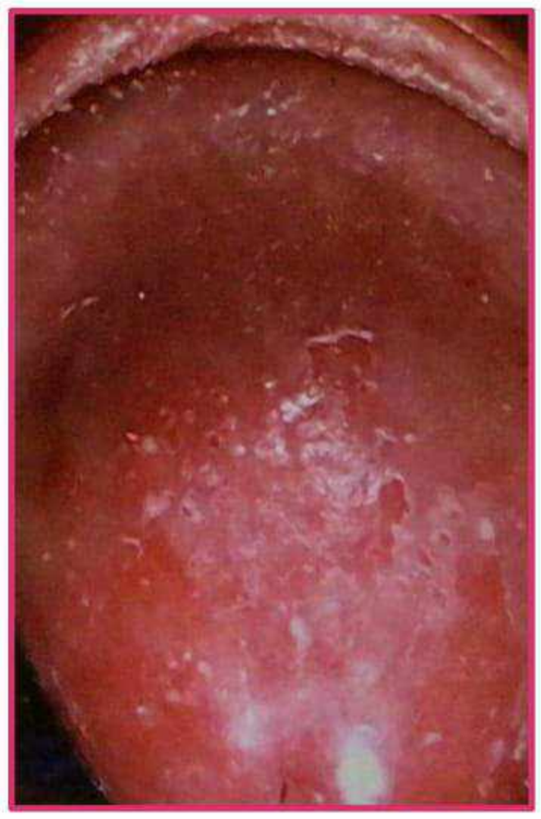


- Angles of mouth → Painful fissures.
- Candida : (MC) Infectious cause

active space

Candidal Balanoposthitis :

- More commonly seen in uncircumscribed individuals.
- commonly involved in Diabetic patient.
- There are small superficial vesicles / papules / pustules
  ↓
  on rupture
  ↓
  Frayed peeling edge.



- No pain.
- Irritation ⊕, Radial fissure
- H/O VVC in female partner.

VULVOVAGINAL CANDIDIASIS

10:45

- In females ⟶ ↑ Pre-menstrual discharge.

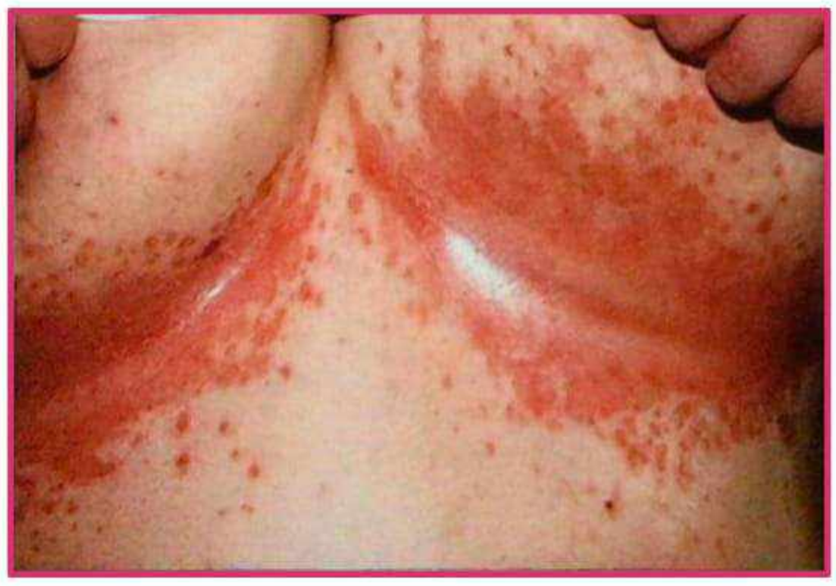
Clinical feature :

- Thick curdy white discharge
- Itching
- Redness
- Soreness.

active space

INTERTRIGO / FLEXURAL CANDIDIASIS

12:00



- Concave borders on opposing surface
- Satellite lesions ⊕
- Vesicles rupture to give frayed peeling edge ⟶ Subcorneal
- Associated Itching and soreness

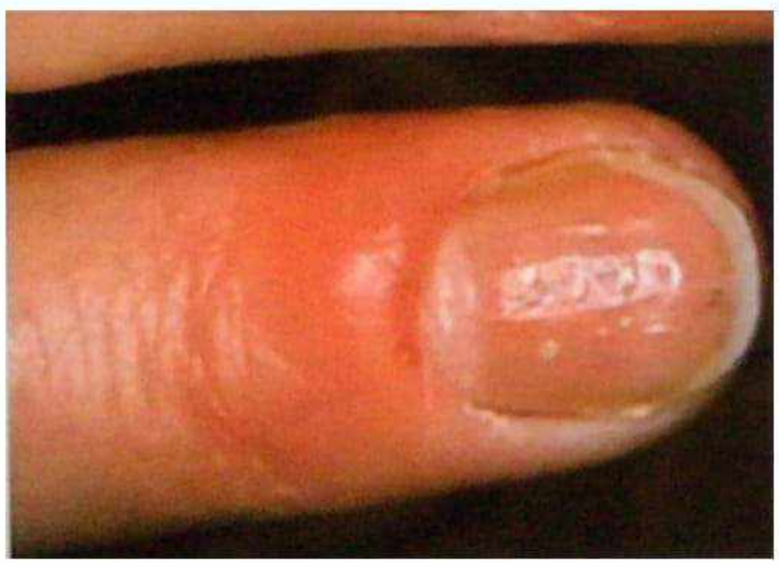
DIAPER CANDIASIS

14:20

- seen in infants wearing diapers for long period of time.
- Granuloma Gluteal Infantum: development of nodules because of candida.

CANDIDAL PARONYCHIA

15:20



- Paronychia: Red swollen proximal nail fold.
- loss of cuticle
- Nail plate separated from fold

active space

- More commonly seen in Housewife, Maids.
- cause Onychomycosis in patients with:
  - i) Raynaud's phenomenon
  - ii) Cushing's disease

Chronic Mucocutaneous Candidiasis

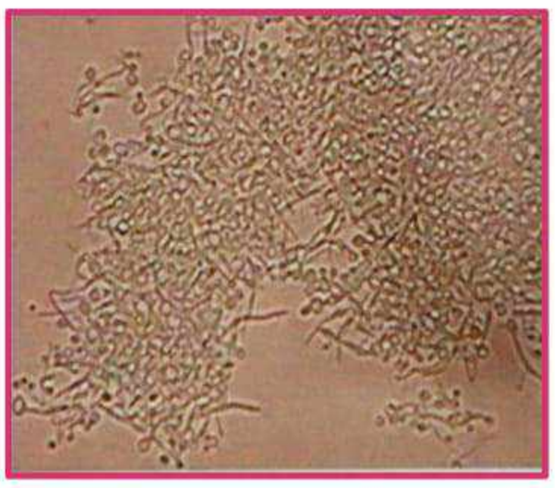
- Persistent candidal infection of skin, nails and mucosa which is refractory to topical therapy.
- Associated with Endocrinopathies (Thyroid, Adrenal)
- Associated Systemic Infection

INVESTIGATIONS

19:06

1. On KOH :

- Budding yeast cells
- Pseudohyphae

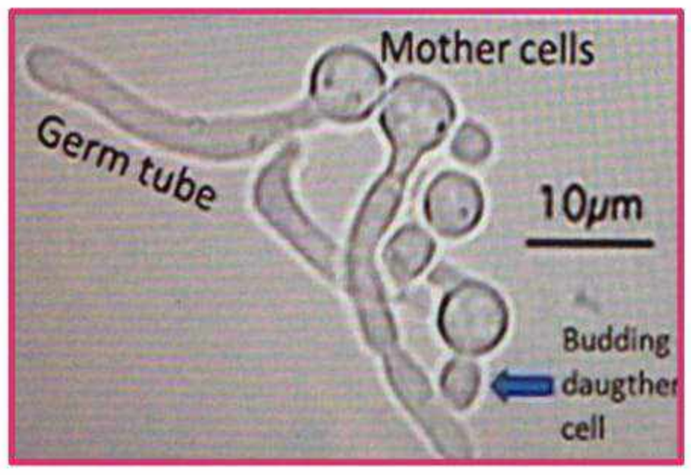


2. On Culture :

- Creamy white colonies

3. Germ Tube Test :

- Specific for Candida Albicans.
- Germ tubes are seen at 37°C.



TREATMENT - CANDIDA

20:31

1. Azoles

Topical	Oral
• Clotrimazole	• Itraconazole
• Other azoles	• Fluconazole

SUBCUTANEOUS FUNGAL INFECTIONS

21:50

- Affects dermis and subcutis.
- occurs after implantation following injury
  - Soil
  - vegetable matter

1. Sporotrichosis
2. Mycetoma
3. Chromoblastomycosis.

active space

SPOROTRICHOSIS

23:00

- a.k.a Rose Gardner's disease
- Occur on exposed parts of body
- caused by Sporothrix Schenkii ⟶ Dimorphic fungus

Single papule at the site of injury

↓

Erosion, ulceration, purulent drainage
(generally not painful)

↓

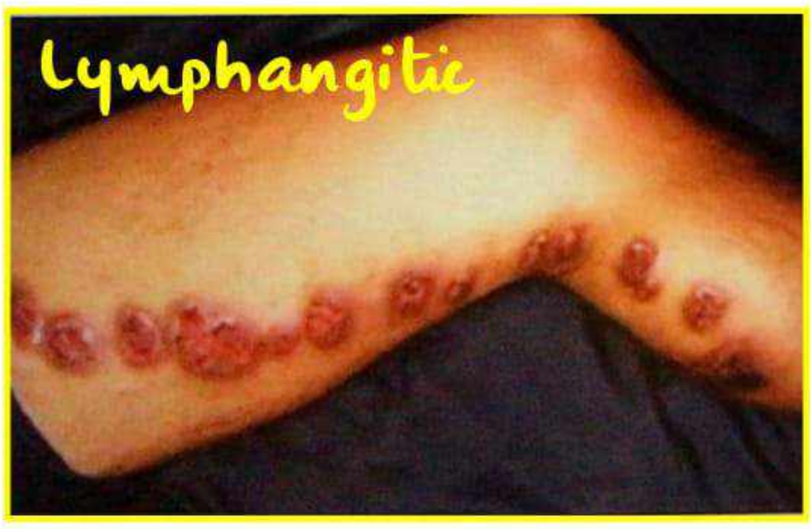
Dermal and subcutaneous nodules along path of lymphatic drainage

Clinical feature :

i) Lymphangitic : Sporotrichoid. (MC)

- lesions along lymphatic drainage.

ii) Fixed : Acneiform, ulcerated, nodular

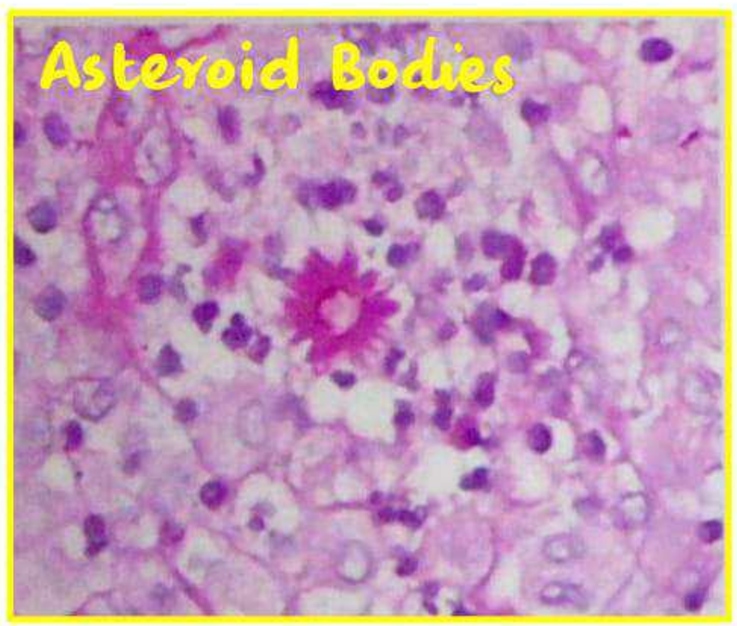


On Biopsy :

- Asteroid Bodies : Yeast surrounded by eosinophilic material.

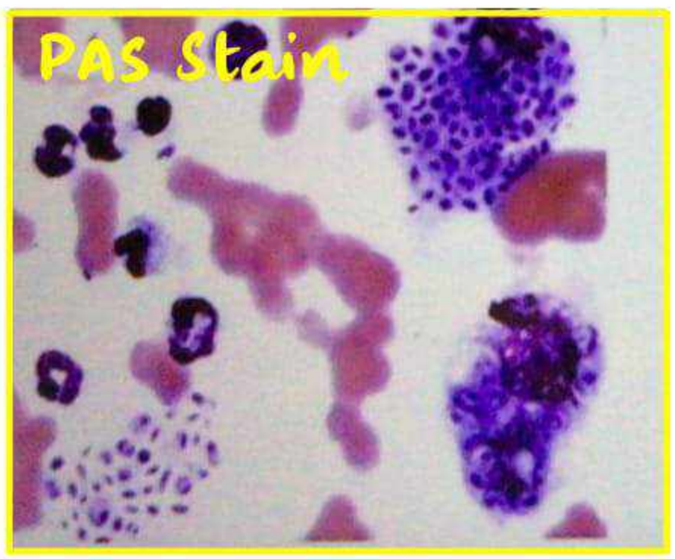
↓

Splendor Hoeplei phenomenon.



On PAS Stain :

- Cigar shaped yeast cells.



Treatment

- Itraconazole. : given for 3-4 months till the lesions resolve.
- Terbinafine
- Potassium Iodide

MYCETOMA

29:00

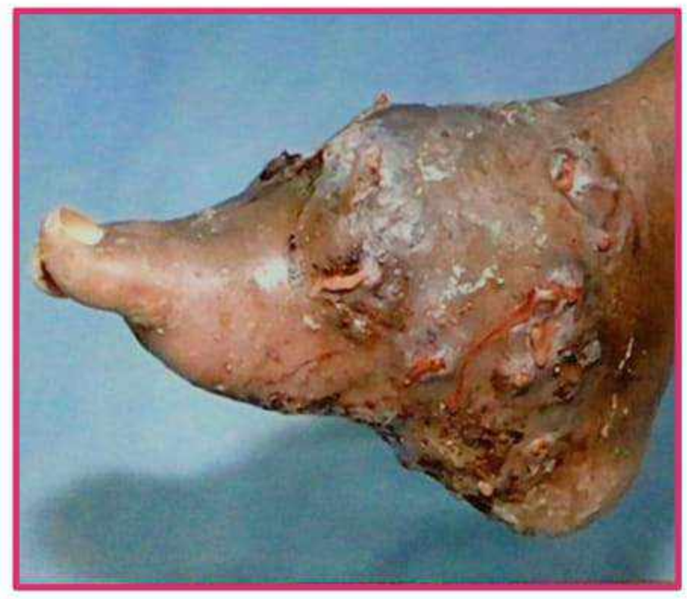
- a.k.a Madura foot / Maduramycosis
- caused by

i) Eumycetoma → Fungus

- Mycetoma mycetomatis (MC)

ii) Actinomycetoma → Bacteria

Clinical feature :



- Triad :

  i) Tumefaction/Induration of swelling

  ii) Grains ⟶ Cell clusters

  ↳ Important to differentiate between Actinomycetoma (Red grains) and Eumycetoma (Black grains)

  iii) Sinuses

- starts as firm painless nodule/papule, more commonly on foot, farmers.

  ↓

  Breakdown to form draining sinuses

  ↓

  Area becomes hard and swollen

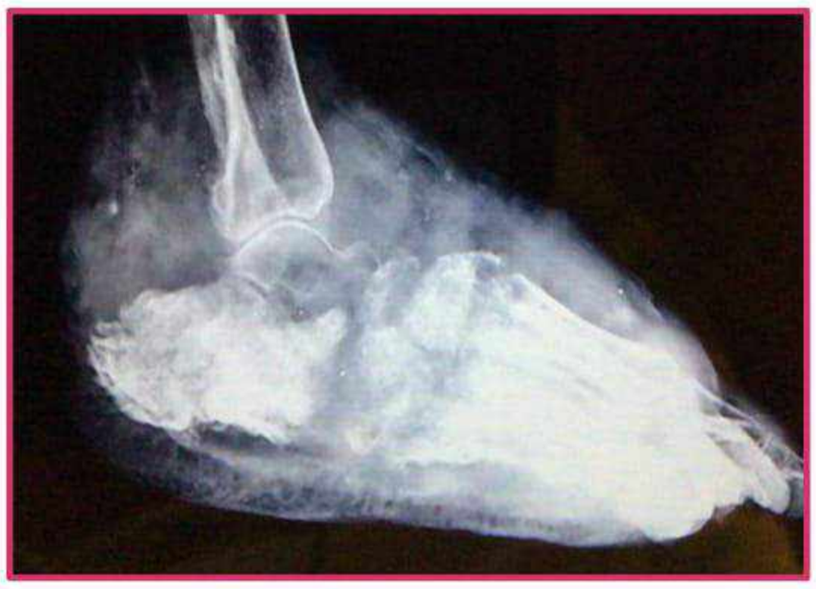
  ↓

  Bones ⟶ Deformity

- painless

ACTINOMYCETOMA	EUMYCETOMA
• Slowly invasive	• Rapidly invasive
• Late presentation, as it is relatively asymptomatic	• Early presentation
• No pus	• Pus present (Red grains)
• Black Brown granules	• Granules yellowish white
• More deformity	• Less deformity
• Granules 4-5 micron, in clusters	• Granules < 1 micron lie singly
• Gram ⊖ve GMS, PAS ⊕	• Gram ⊕ GMS, PAS ⊖
• Responds to Antifungals (Itraconazole, Amphotericin B)	• Responds to antibiotics (Sulphonamides, Doxycyclines)
On KOH :	On KOH :
• Thinner 0.5 - 1 µm in diameter	• Branched hyphae 2-6µm
On Biopsy :	
• Splender Hoeplii Phenomenon ⊕	

X-Ray Bone :



Treatment :

i) Eumycotic

- Itraconazole
- Other Azoles.

↓

- Surgical Treatment needed

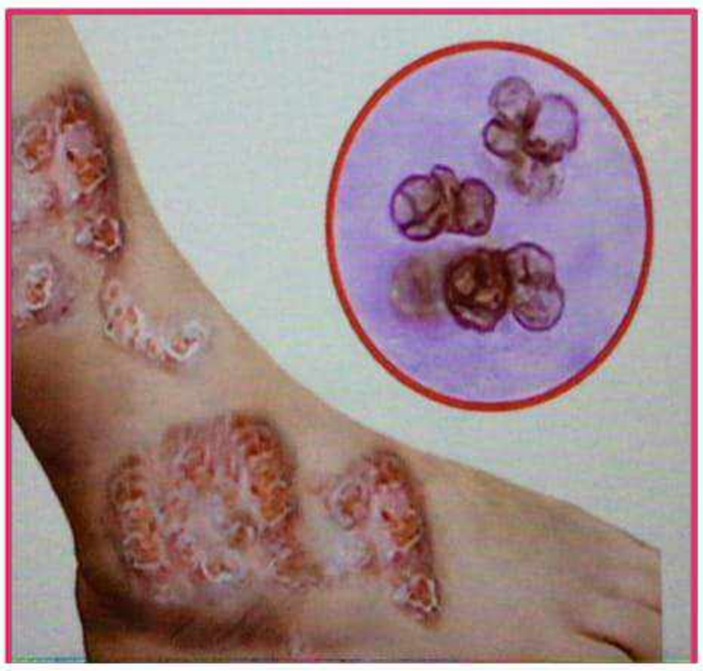
ii) Actinomycotic

- Dapsone + Streptomycin
- Cotrimoxazole + Streptomycin
- 2nd Line → Rifampicin.

CHROMOBLASTOMYCOSIS

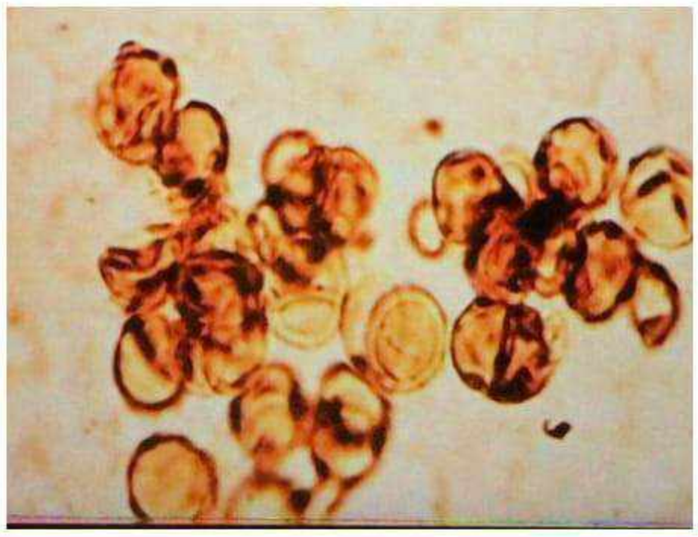
38:45

- caused by Phialophora verrucosa. (pigmented fungi)
- Presents with verrucous exophytic plaques on feet and legs.

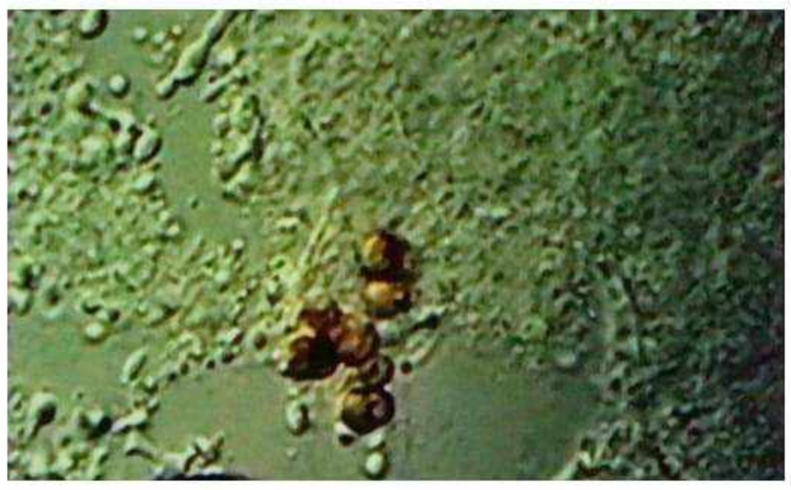


Sclerotic / Muriform / Copper Penny Bodies

- Pigmented thick walled uni/ multicell clusters.



On KOH : pigmented hyphae.



Treatment

- Azoles
- Potassium Iodide
- Heat application.

active space

Viral Infections (Part-1)

ETIOPATHOGENESIS

00:50

- Cell lysis → Herpes → vesicles
- Cell proliferation → Pox, HPV → Growths
- Carcinogenesis → Cervical CA, Hepatoma
- Exanthemata → Maculopapular rash associated with fever.

  Viraemia, Type 3 HSR (Arthus Reaction), virus lodged in capillaries and replicate in epidermis.
- Persistent infection : Periods of latency and reactivation (HSV, VZV)

MOLLUSCUM CONTAGIOSUM

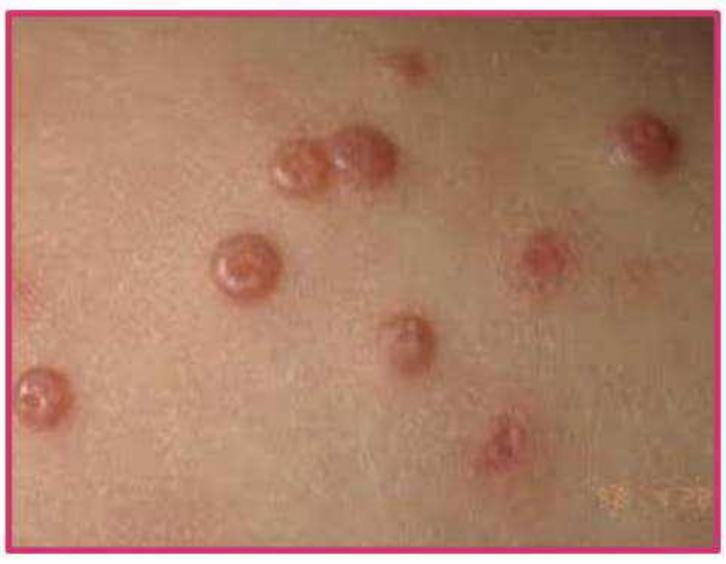
03:03

- caused by Molluscum Contagiosum Virus (Molluscipox virus) (Family → Poxviridae)
- MCV → 4 Types
  - Type 1 : in children
  - Type 2 : in adults (sexually transmitted)

- Large DNA viruses ⟶ Largest Virus known
- I.P : 2 weeks to 6 months

Clinical features :

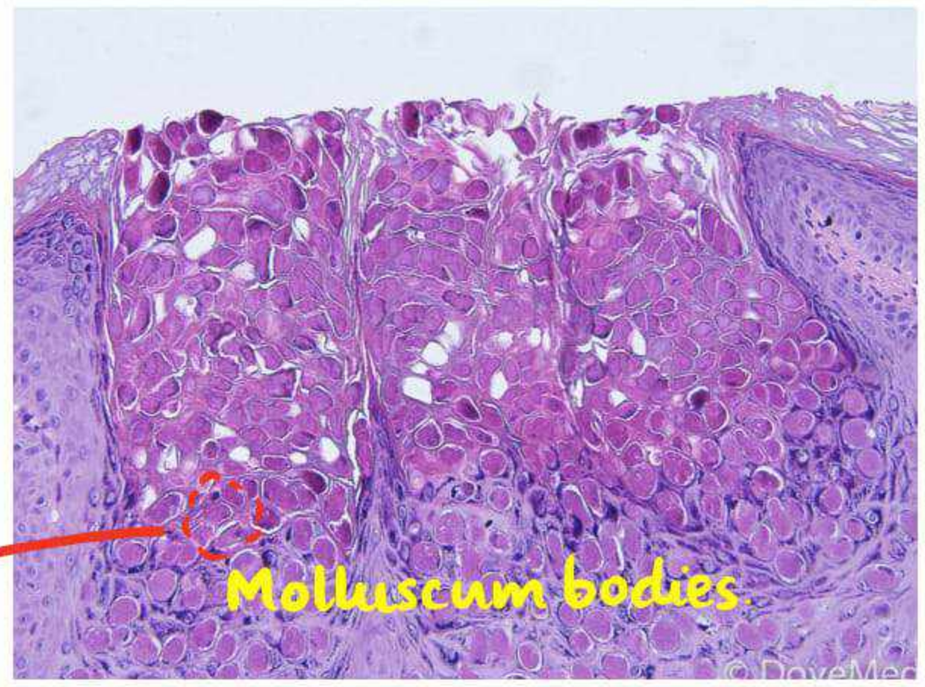
- Presents as pearly white umbilicated papule.
- (MC) occurs in children, can occur in adults.
- (MC) occurs in face.



- Pseudokoebnerization ⊕
  - ↳ due to autoinoculation of virus.
    - seen in i) Warts
      ii) Molluscum contagiosum.
- when present in genitals only ⟶ STD's
- There may be family h/o similar lesions.

On HPE :

- Cup like indentation of epidermis into dermis
- Invaginated Rete Ridges
- Acanthosis ⊕
- Henderson Patterson bodies



- **Henderson Patterson bodies :**

  - Intracytoplasmic eosinophilic inclusion bodies.

Treatment :

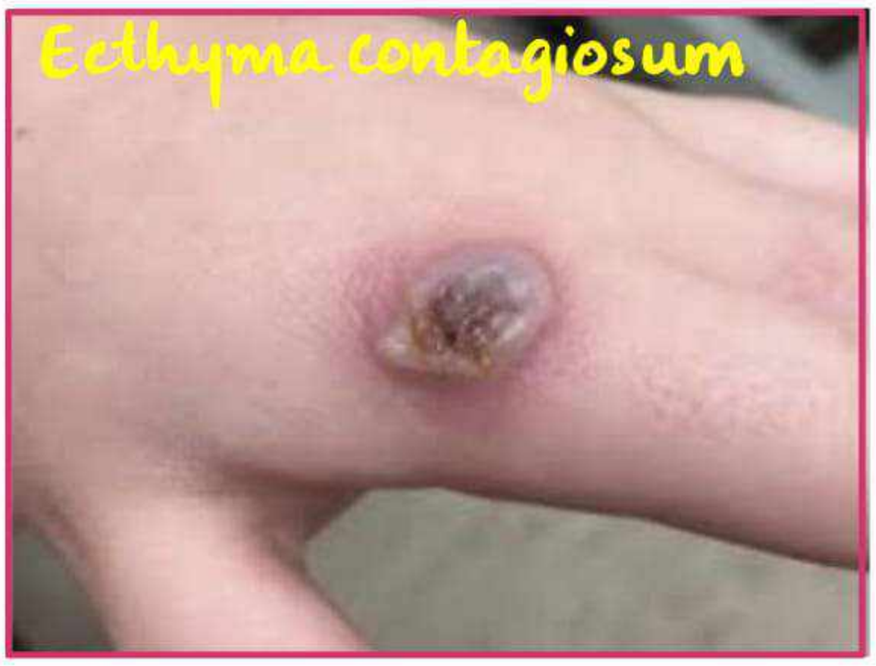
- Self limiting → **Self Resolute**
- **Chemical Treatment :**
  - i) TCA (Trichloroacetic acid)
  - ii) 10% KOH
- **Physical Treatment :**
  - i) Enucleate (MC)
  - ii) Curettage
  - iii) Radiofrequency ablation
  - iv) Cryotherapy
  - v) LASER.
- **Systemic Treatment :**
  - i) levamisole
  - ii) Cematidine.

active space

OTHER POX VIRUS INFECTIONS

18:32

1. Small Pox (variola)
2. Orf virus / Parapoxvirus → cause : Ecthyma contagiosum
3. Pseud cow pox → Milker's nodule



- Both Ecthyma contagiosum and Milker's nodule are zoonotic

Ecthyma contagiosum → Sheep

Milker's nodule → Cow

Both Presents : small firm blue papule at site of contact

↓

Pustule

↓

Crust

↓

Hemorrhagic Base surrounding erythema

HUMAN PAPILLOMA VIRUS

23:00

- small DNA viruses ⟶ causes Warts.
- Has approximately 200 subtypes.
- Infection depends on :

  i) Anatomical site

  ii) HPV Type

- Tropism for squamous epithelial cells
- Infect the basal layer ⟶ Replicate in Stratum Spinosum, Stratum granulosum.
- Transmission :

  i) Direct

  ii) Autoinoculation

  iii) Sexual transmission

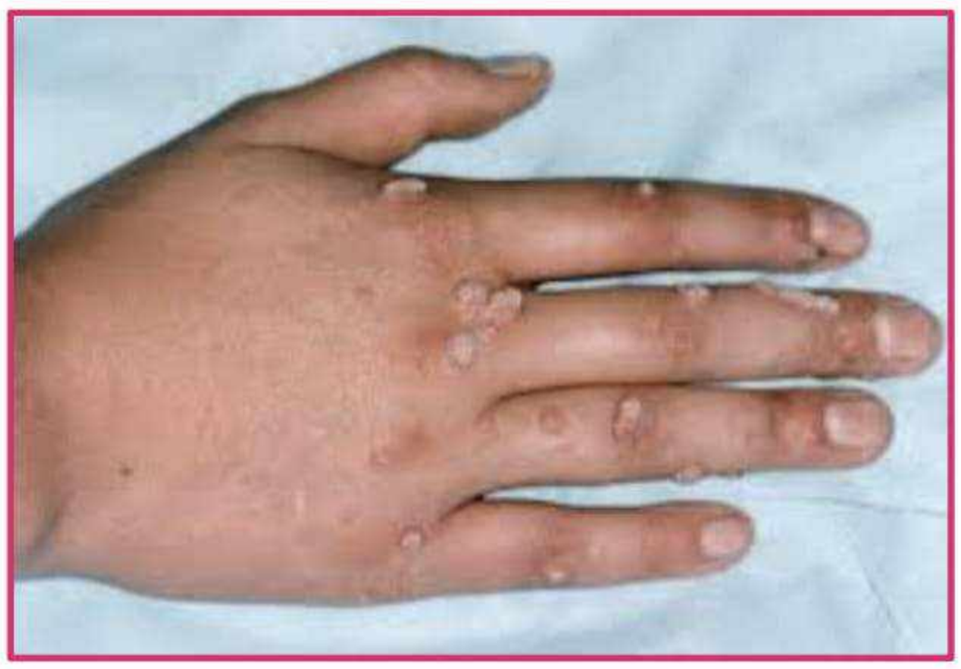
HPV Types

- Type 1 ⟶ Deep Plantar Warts
- Type 2 ⟶ Superficial Plantar Warts
- Type 3 ⟶ Plane warts ⟶ also caused by 10
- Type 4 ⟶ Common warts ⟶ also caused by 2, 27

active space

- Type 5 → EDV → also caused by Type 8
- Type 6 → Anogenital warts → also caused by Type 6 and 11 (Low Risk)
- Type 7 → Butcher warts → also caused by Type 16, 18, 31, 33, 45 (High Risk)

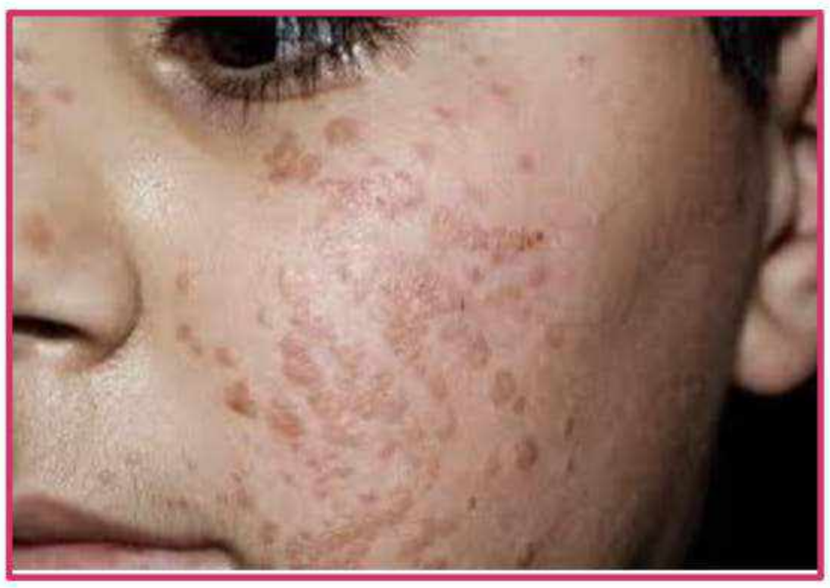
Common Warts : caused by 4, 2, 27



- a.k.a Verruca vulgaris
- Presents as :
  - verrucous papules and plaques (Rough hyperkeratotic) at any body site.
- Pseudokoebnerization ⊕

Plane Warts

- caused by HPV Type 3, 10
- a.k.a Flat warts / Verruca plana.
- Flat topped lesions. (smooth)
- (MC) seen in Face



Plantar Warts

- 2 Types :

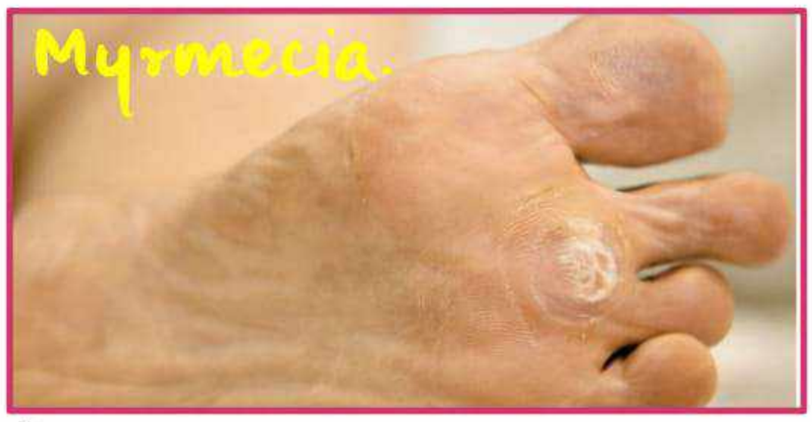
  i) Deep - HPV 1

  ↳ a.k.a Mosaic warts

  - Plaque formed of closely placed papules.

  ii) Superficial - HPV 2

  ↳ a.k.a Myrmecia.



- **Painful** (Pain on lateral pressure)

CORN	WARTS
• Hyperkeratosis of skin because of ↑ pressure. • ▽ → Pain on perpendicular pressure. • paring does not reveal bleeding points • Skin markings retained.	• Pain on lateral pressure • Paring reveals bleeding points. • loss of superficial marking.

active space

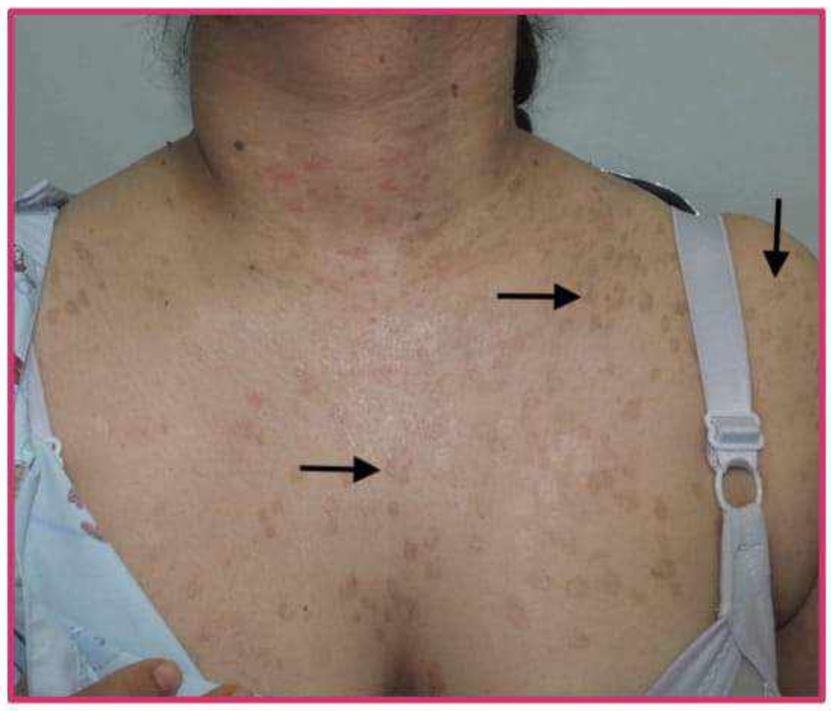
Epidermodysplasia Verruciformis (EDV)

- caused by HPV - 5,8
- Autosomal Recessive Condition
- Genetic tendency to widespread HPV infection
- Increased Risk of SCC.
- Presents as 3 kinds of lesions :

  i) Plane warts

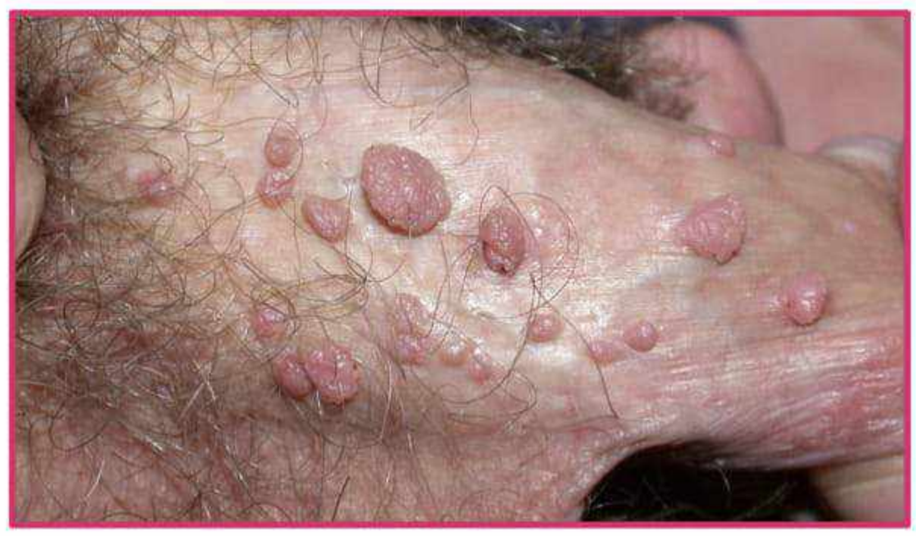
  ii) P. versicolor like lesions on trunk

  iii) Reddish plaques.



Anogenital warts :

- caused by HPV Types :
  - 6, 11 ⟶ Low Risk
  - 16, 18, 31, 33, 45 ⟶ High Risk ⟶ Development of Cancer.
- Sexually Transmitted.
- Condyloma acuminata. ↘ flat pointed
- Softer
- Flat base.



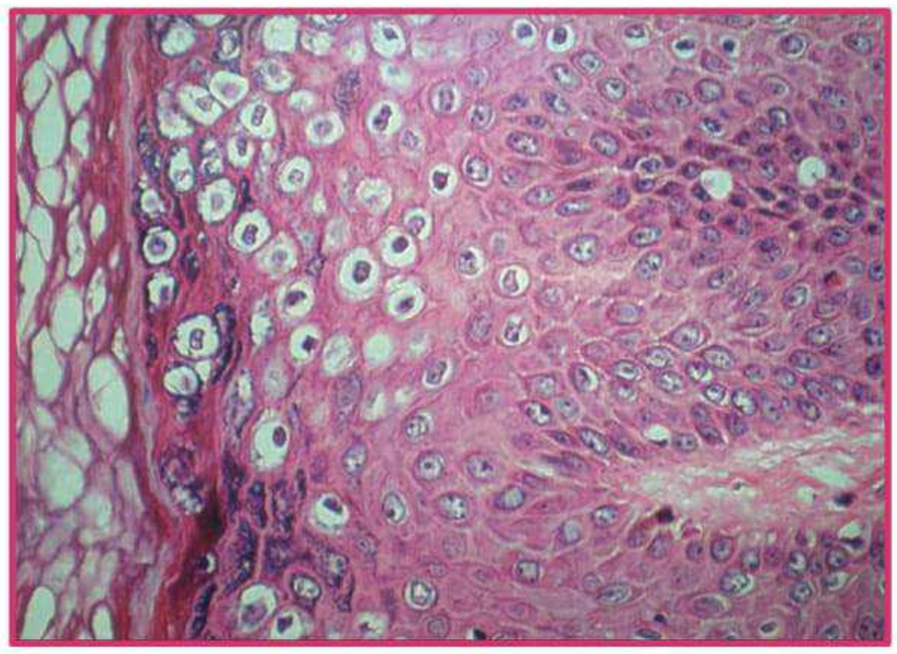
active space

Butchers Wart

- caused by HPV 7
- on hands of Butcher.

On HPE :

- Hyperkeratosis
- Acanthosis
- Papillomatosis
- Koilocytes ⊕ → Nuclear condensation at centre and peripheral condensation of cytoplasm.



 → Perinuclear pallor / Halo.

Treatment: Self limiting

Topical or Chemical
- i) Imiquimod
- ii) Salicylic acid
- iii) TCA
- iv) Podophyllotoxin (genital)

Physical
- i) Radiofrequency ablation
- ii) Electrocautery
- iii) LASERS
- iv) Cryotherapy.

Oral
- i) Zinc
- ii) Levamisole

Viral Infections (Part-2)

HUMAN HERPES VIRUS

00:10

- DNA viruses
- Replicate inside nucleus
- Tendency to be latent: 1° infections → Dorsal Root ganglion (latent)
  - → Recurrent
  - → Subclinical → Asymptomatic shedding of virus.

Subfamily		Genus	Virus	Abbreviation
Alphaherpesvirinae	α	*Simplex virus*	Human simplex virus, type 1	HSV-1
	α		Human simplex virus, type 2	HSV-2
	α	*Varicellovirus*	Varicella-zoster virus 3	VZV
Betaherpesvirinae	β	*Cytomegalovirus*	Cytomegalovirus 5	CMV
	β	*Roseolovirus*	Human herpesvirus type 6	HHV-6
	β		Human herpesvirus type 7	HHV-7
Gammaherpesvirinae	γ	*Lymphocryptovirus*	Epstein–Barr virus 4	EBV
	γ	*Rhadinovirus*	Human herpesvirus type 8	HHV-8

HUMAN HERPES SIMPLEX VIRUS (HSV)

03:26

- HSV 1 and 2
- Herpes Simplex infection → 1° → Recurrent → Milder.
  - → Latent

- HSV 1 → above waist (usually) → 50 % of Recurrent infections
- HSV 2 → below waist → Genital herpes (sexually transmitted)

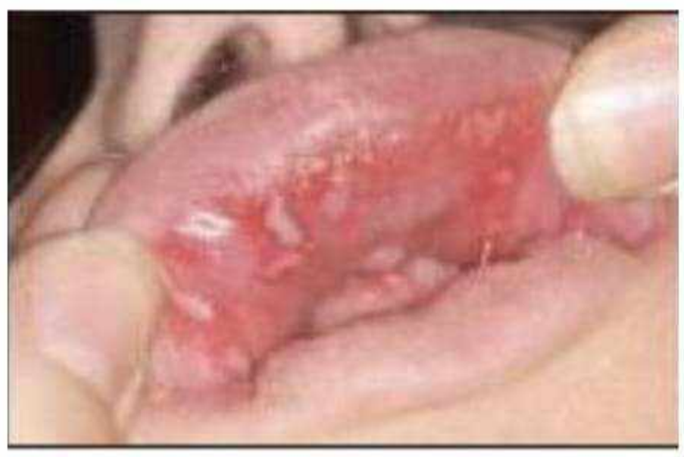
  ↘ Responsible for 90 % Recurrent infections

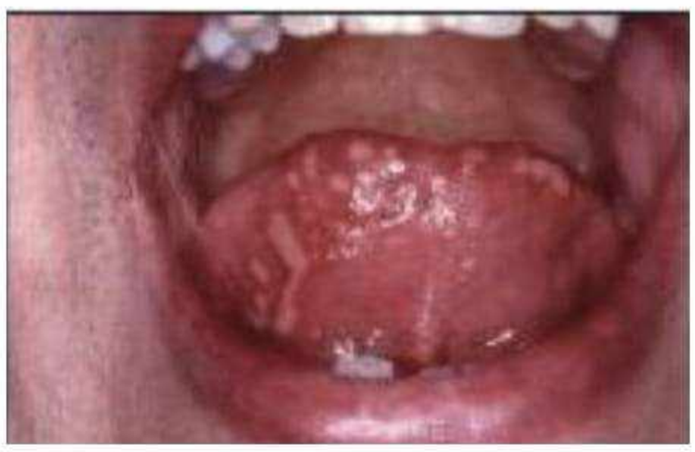
Primary Infection

- more severe, more number of lesions
- more constitutional symptoms ⊕
- lymphadenopathy ⊕

Herpetic Gingivostomatosis :

- Primary infection
- (MC) cause of gingivostomatitis in < 5yrs.
- Involves → Buccal mucosa, Palate, lips
- Presents with :
  - i) Painful vesicles which rupture to form erosions (Polycyclic erosions)
  - ii) Gingiva
  - iii) Buccal mucosa
  - iv) Palate, Tongue
  - v) Lips

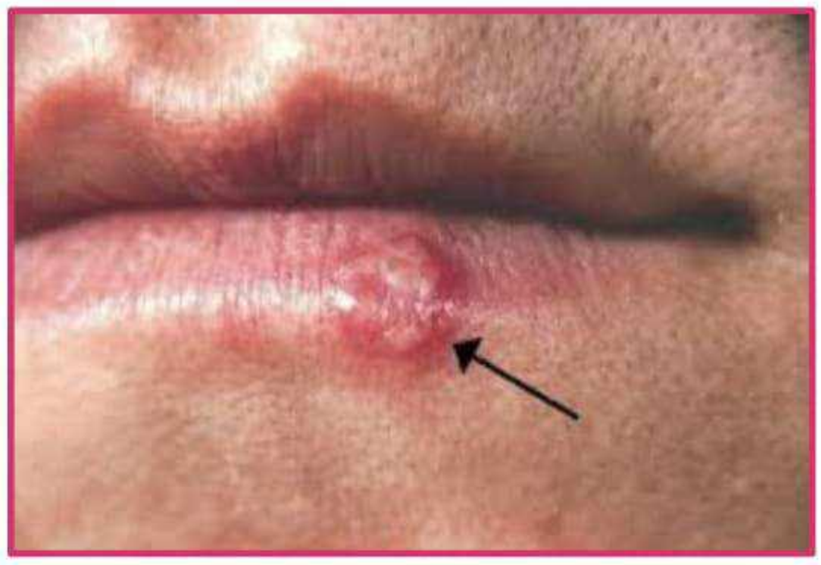




active space

- associated with constitutional symptoms (fever, malaise, Lymphadenopathy, difficulty in eating.
- overlying grey membrane (+)

Herpes Labialis



- a.k.a Cold sore
- Recurrent infection.
- Trigeminal ganglion → site of latency
- usually starts with mild prodrome
  ↓
  Burning sensation / tingling
  ↓
  vesicles at vermillion border lips
  ↓
  Ruptures to form crust
  ↓
  Heals in 3-5 days without scarring.

Aggravating factors

- Stress
- Illness (febrile)
- Premenstrually

} Reactivation at vermillion border lips

- Tendency to recur.

active space

GENITAL HERPES

14:20

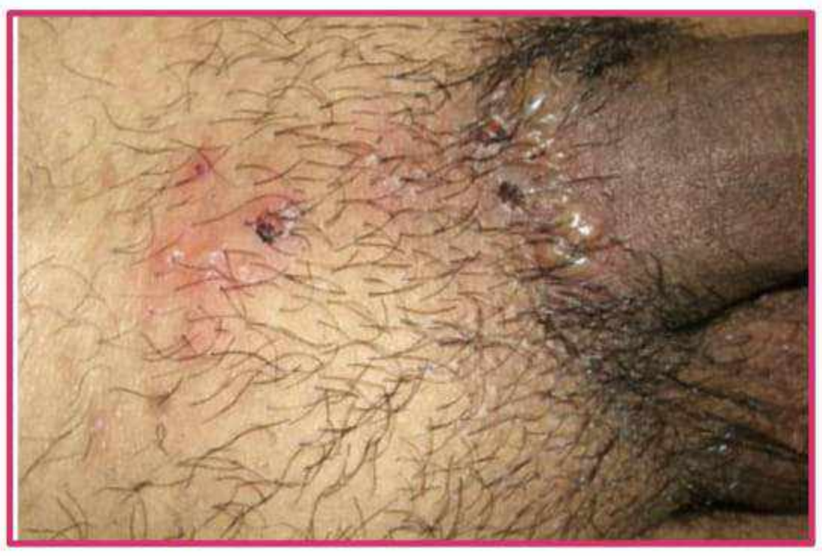
- They can be :

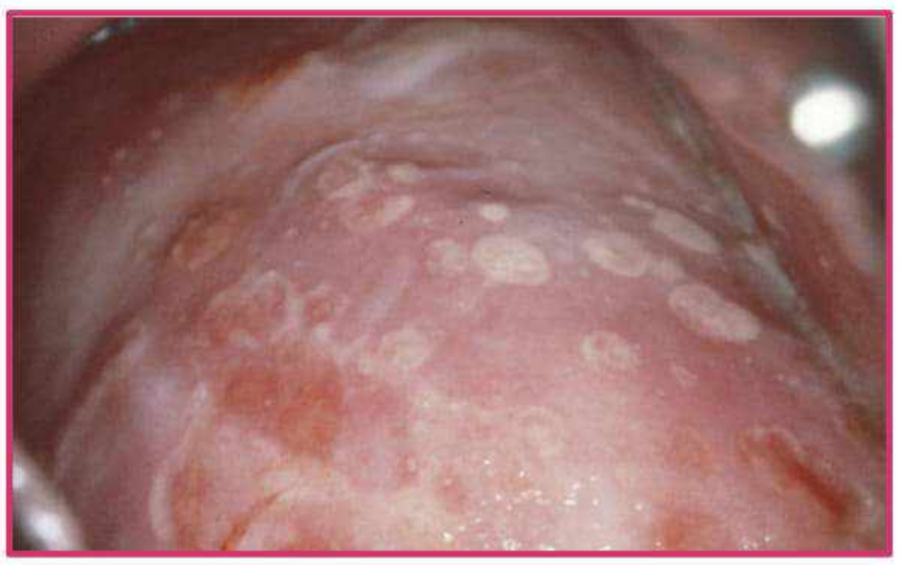
  i) Primary

  ii) Secondary

  iii) Subclinical shedding : virus can be transmitted to partner.

- Sexual transmission ⊕ ⟶ HSV-2





Presentation:

- vesicles ⟶ rupture to form polycyclic erosions on erythematous base (Painful)

  ↓

  Heals in 7-10 days without scarring

- Patient gives h/o Recurrence.
- Lymphadenopathy
- Constitutional symptoms

} seen in 1° infection

active space

Recurrent Herpes Genitalis

- Less severe
- Decreased number of lesions
- >6 Recurrences in an year ⟶ Suppressive Treatment
  ↓
  Reduces frequency and severity

Neonatal Herpes

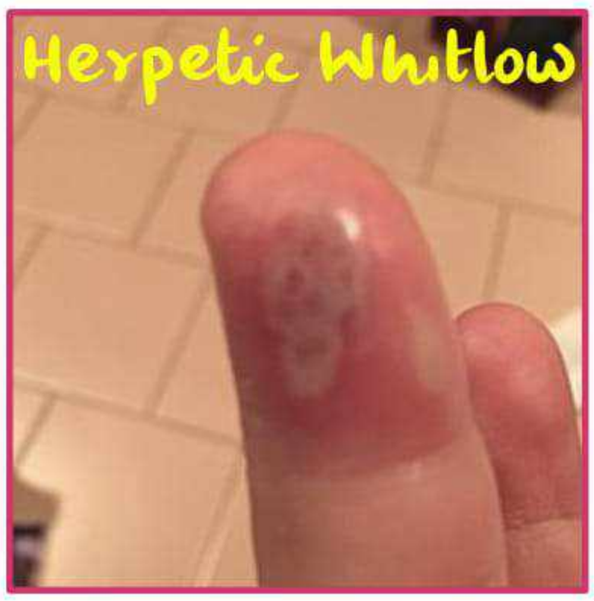
- occurs within 28 days after delivery

Presents as :

- Subtle blistering
- Widespread vesicular lesions
- Pregnant lady with herpes at the time of delivery ⟶ Do C.S
- Treatment : i) i.v Acyclovir
  ii) Immunoglobulin.

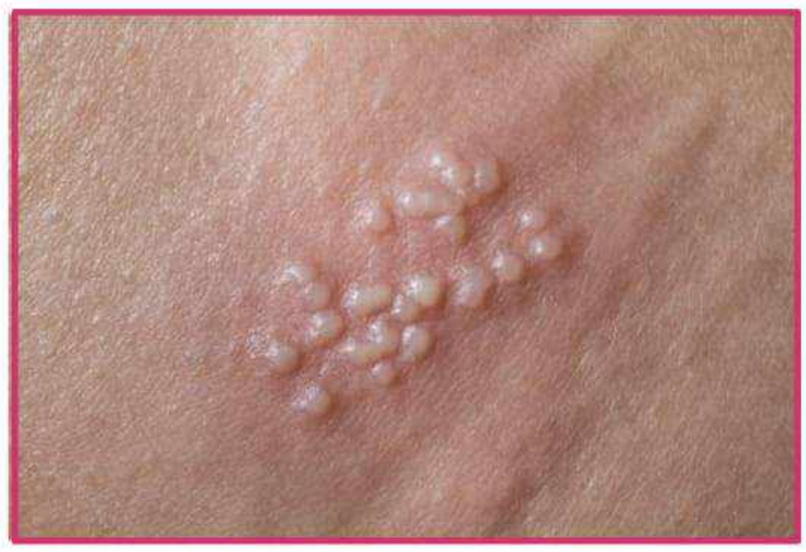
Herpetic Whitlow

- seen in Finger tips of Health care professionals (Dentist, Pathologist)



- painful vesicles on fingertips which coalesce

Herpetic Gladiatorum



- seen in wrestlers
- sites : Trunks, sides of neck.

Herpetic Keratoconjunctivitis

- can be 1° / Recurrent

Can also cause :

- Meningitis
- Keratitis.

Complications associated with HSV

i) Erythema multiforme

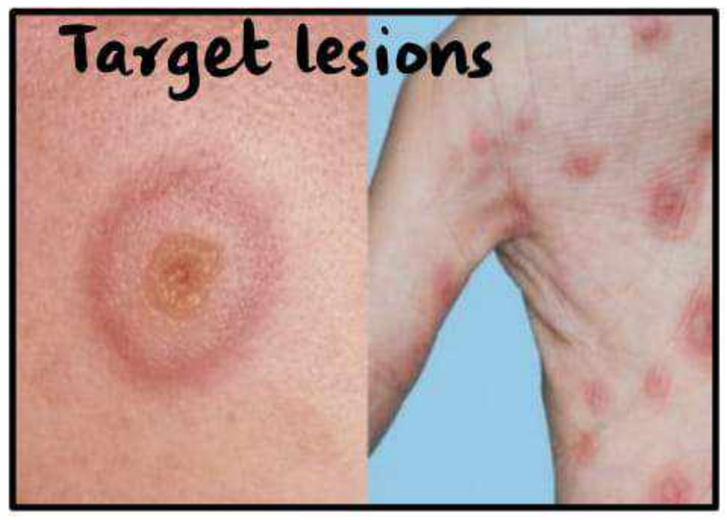
ii) Bell's palsy : facial nerve palsy → Reactivation of virus.

iii) Hepatitis, Encephalitis

iv) Eczema herpeticum

Erythema multiforme

- (Mc) Infectious cause → HSV 1
- develop Target lesions



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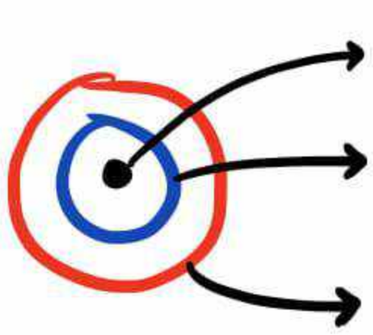
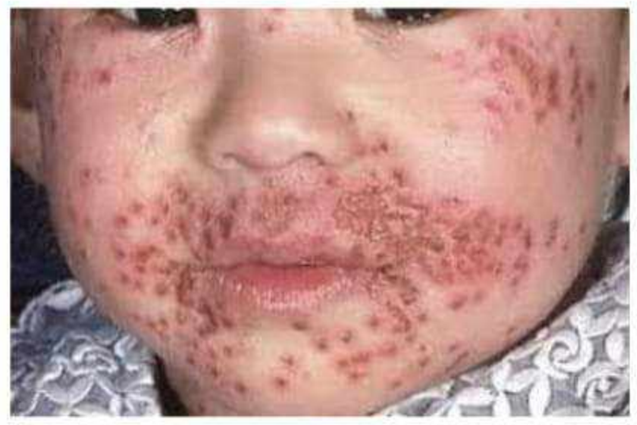


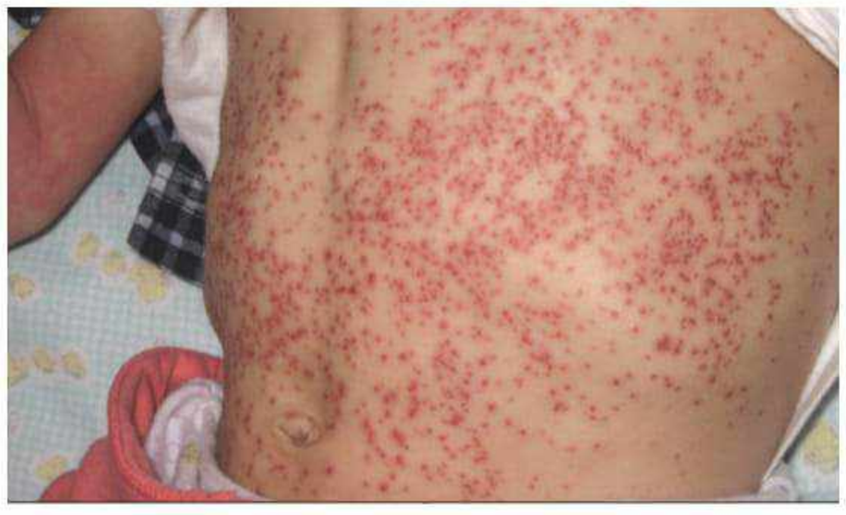
Eczema herpeticum

- a.k.a Kaposis varicelliform eruption
- not an eczema
- seen in individuals who have damaged skin.



→ pre-existing dermatoses

→ predisposed to widespread HSV 1 infections.
- Atopic dermatitis – (MC)
- Pemphigus
- Darrier's
- Sezary syndrome.



→ crops of vesicles which rupture to form crust

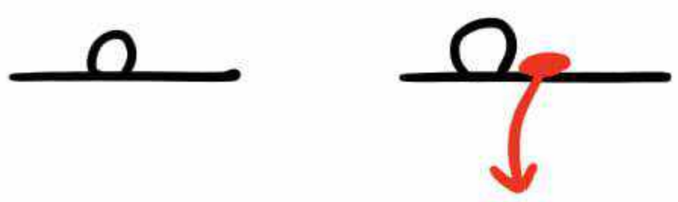
active space

Treatment :

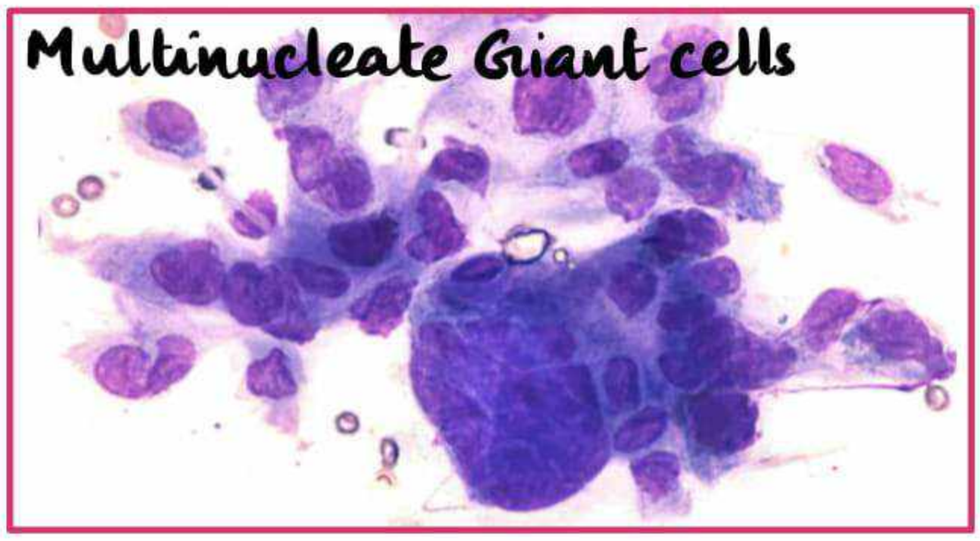
- Acyclovir
- No steroids.

Diagnosis of HSV 1

1. Tzanck smear :



Stain with Giemsa



- Acantholytic cells are seen

On Serology :

- 1° cases ⟶ Seroconversion.
- Recurrent cases ⟶ ✗

Treatment of Hsv

- Suppressive treatment usually given in >6 recurrences/year.
  - ↳ Acyclovir 400mg BD
    - ↳ It decreases frequency and severity of the episodes.

      Given minimum for 1yr (given upto 10yrs)

active space

Episodic

Antiviral	Primary (7-10 days)	Recurrence (5 days)	Suppressive 6 months - 1yr
Acyclovir	200 mg 5 times / day	400 mg tid	400 mg bd
Valaciclovir	1 gm bd	500 mg bd	500-1000 mg bd
Famciclovir	250 mg tid	125 mg bd	250 mg bd

- **Acyclovir Resistant Cases :**
  - Drugs used are **Foscarnet and Cidofovir.**

active space

Viral Infections (Part-3)

VARICELLA ZOSTER VIRUS

00:10

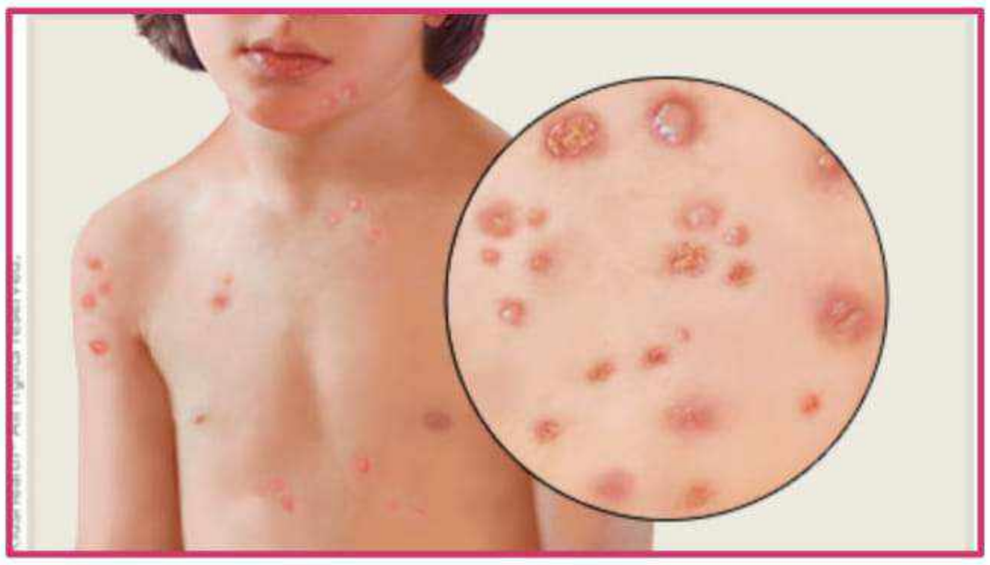
- caused by HHV-3

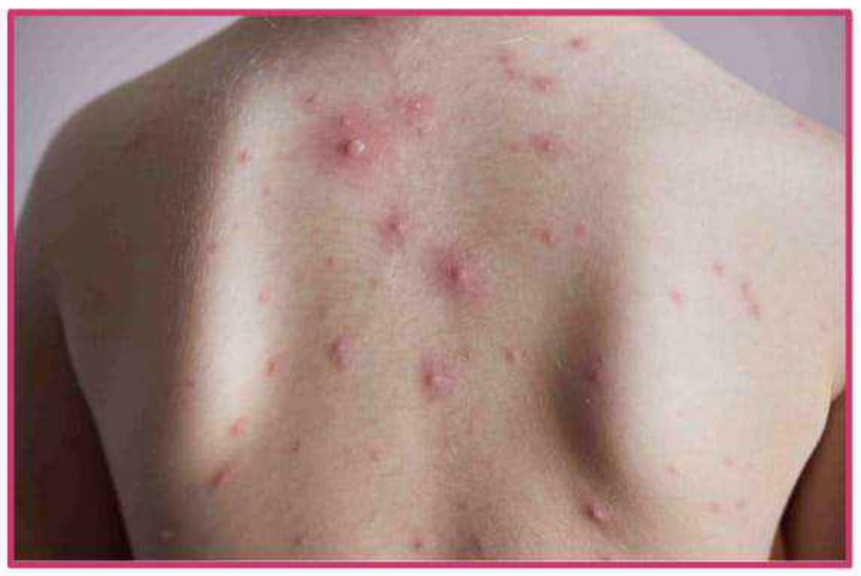
VZV
- chickenpox { Varicella → children
- Herpes zoster → Adults

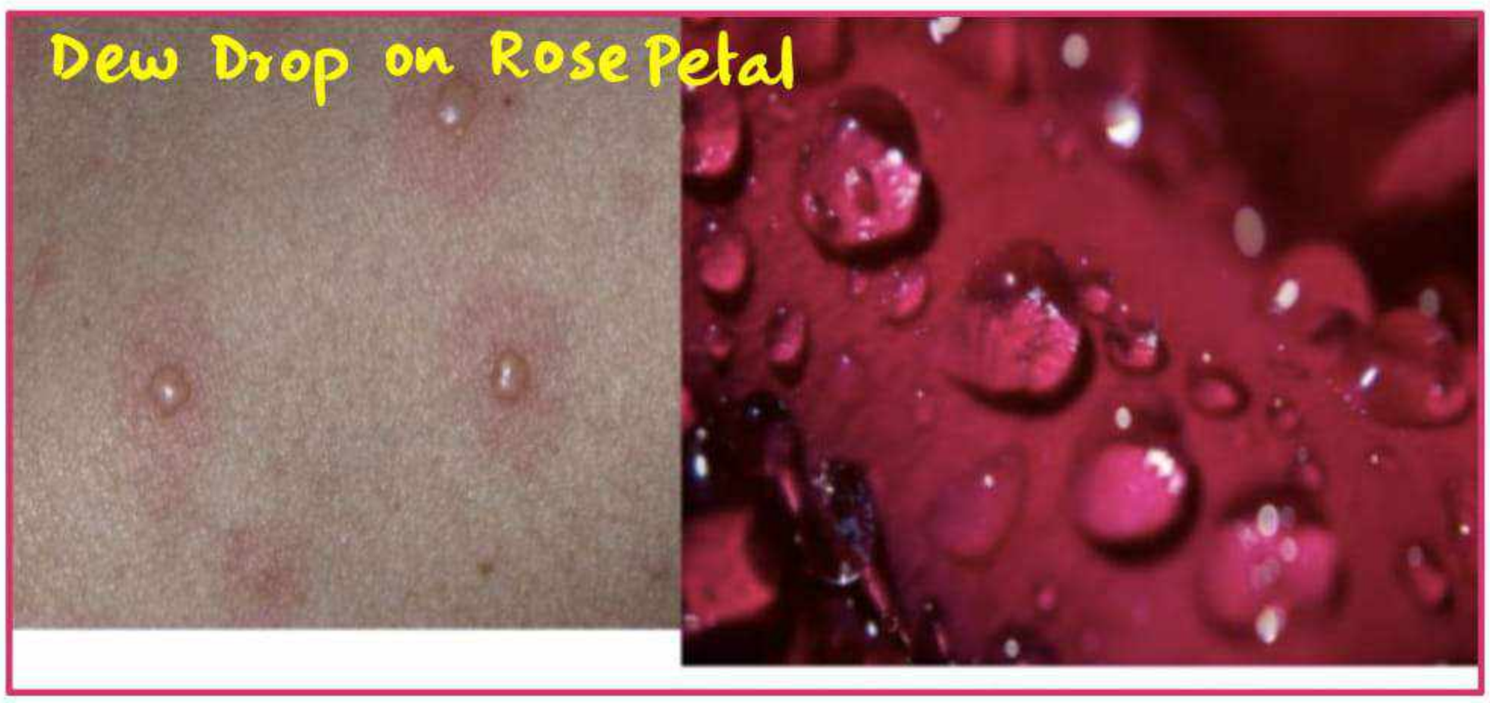
- I.P : 14-17 days
- Affects children > Adults

Clinical features:

- Polymorphic morbiliform centripetal rash
  - Polymorphic → Different lesions
  - centripetal → central part of body
- Macules → vesicles → pustules → crust
- Site : Trunk.







Associated features :

- fever
- cough
- sore throat
- very itchy lesions.
- Malaise

active space

Complications :

- Encephalitis
- Pneumonitis
- Hepatitis.

Maternal varicella :

During 2nd TM

- Varicella syndrome
- Scarring
- Hypoplasias of limb

During delivery

- 30% Mortality
- Severe infection
- Rx : i.v Acyclovir
  Immunoglobulin.

Treatment

1. Pre-exposure prophylaxis :

- Live attenuated vaccine
- Oka strain
- 2 doses are given 3 months apart

2. Post-exposure prophylaxis : given within 10 days of contact.

- Vz Ig
- i.v Acyclovir

3. Treatment :

- Supportive Rx → i) Paracetamol.

  ii) Antiallergic

- Treat according to symptoms

- Patient of chicken pox becomes non-infective once crusting occurs in all lesions.

- Acyclovir given when:

  i) Adults

  ii) Systemic complications

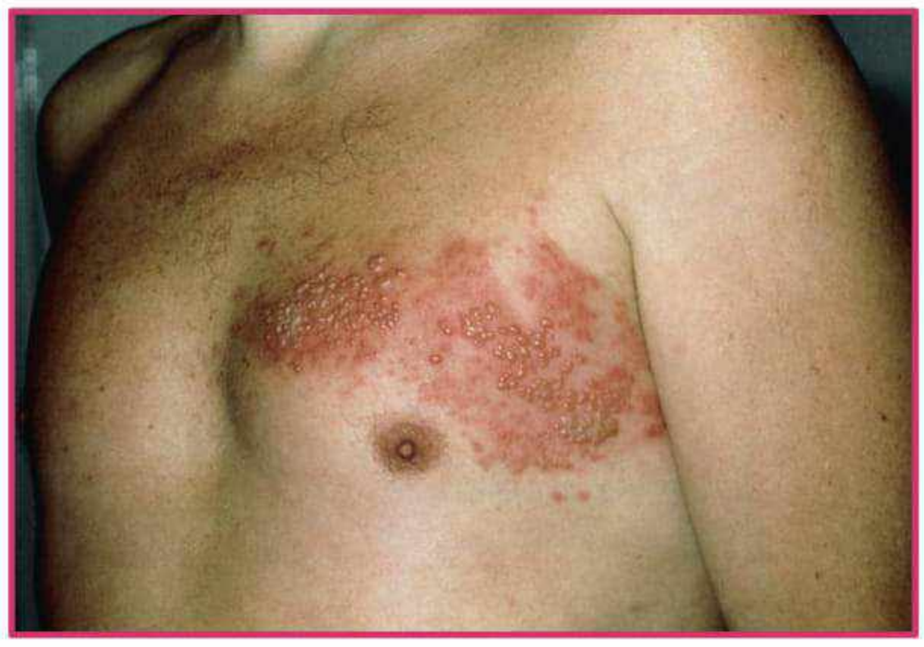
  iii) Immunocompromised.

- (Mc) seen in adults
- virus becomes latent in Dorsal Root ganglion

Predisposing factors :

- Infection
- Old age.
- Chemotherapy
- Immunocompromised
- Stress

Clinical features :

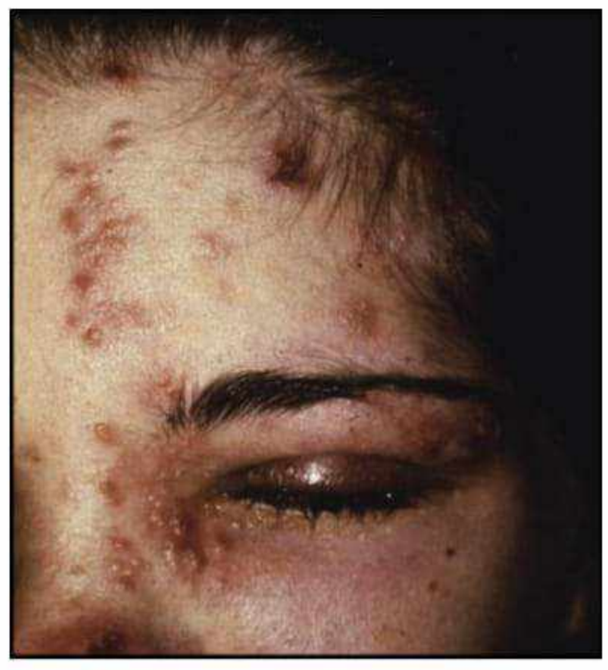


- vesicles in a dermatomal distribution with midline cut off.
- Pain ⊕
- vesicles ⟶ pustule ⟶ crust
- (MC) Dermatome involved :

  Thoracic > Trigeminal > cervical > lumbosacral.

Other associations :

1. Herpes Zoster Ophthalmicus :



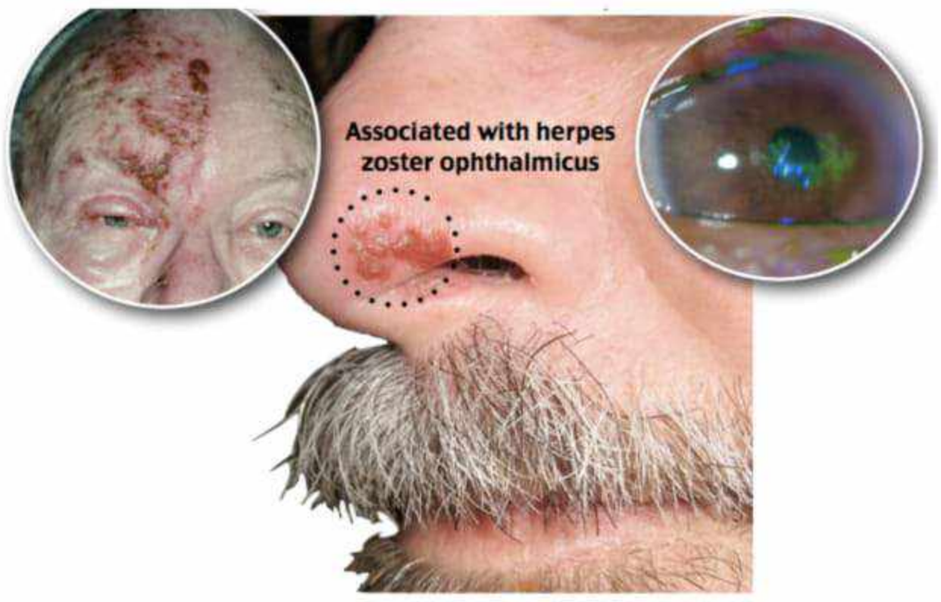
- when ophthalmic branch of Trigeminal nerve is involved.
- involvement of eye :

  i) Conjunctivitis

  ii) Keratitis

  iii) uveitis ⟶ severe uveitis indication for oral steroids.

active space

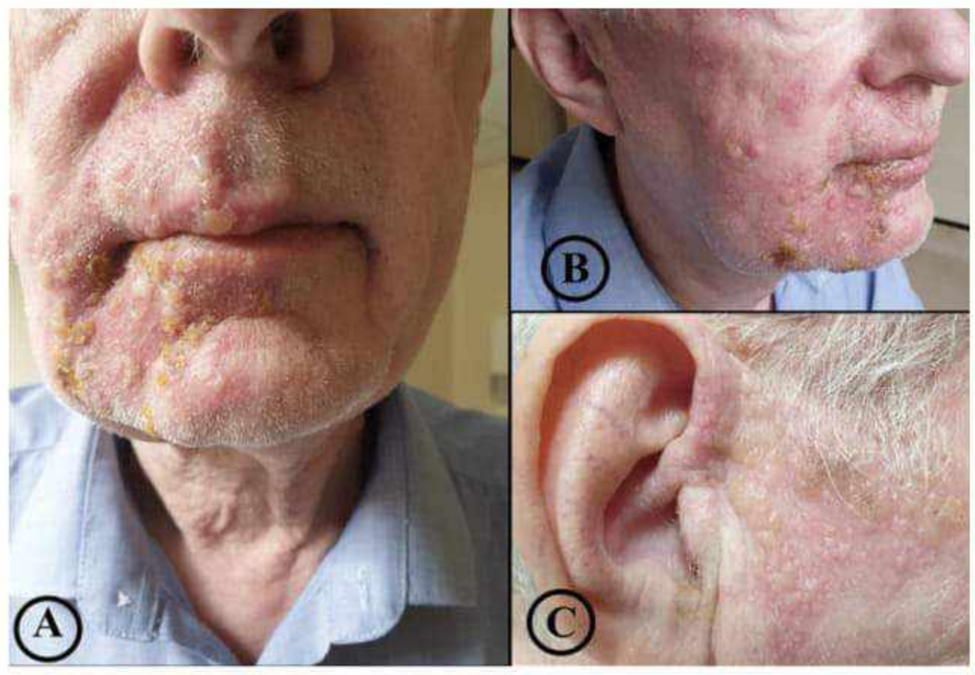


HUTCHINSON SIGN :

- due to involvement of nasociliary branch of Ophthalmic nerve (Trigeminal nerve)
- vesicles on side of nose ⟶ HZO

2. Herpes Zoster Oticus :

- Inflammation of geniculate ganglion, involves both facial nerve and Trigeminal nerve



Clinical features:

- Ear pain
- Tinnitus
- Vesicles on the tympanic membrane / pinna
- Facial nerve palsy

"RAMSAY HUNT SYNDROME"

3. Post Herpetic Neuralgia :

- sequelae to Herpes Zoster
- pain / neurological symptoms persists for > 3 months (> 1 month) after onset of Herpes zoster.
- Symptoms : Parasthesias, Dysthesias.
- Seen in : Elderly, Immunocompromised, chemotherapy.

Treatment :

- Acyclovir 800mg at 5 times a day for 7-10 days. (start within 48-72hrs of infection)
- Valacyclovir 1g TID
- Famciclovir 500mg TDS
- Symptomatic :

  i) Painkillers

  ii) Anti allergies

  iii) Manage 2° infections

Treatment for PHN :

i) TCA

ii) Gabapentin

active space

iii) Pregabalin

iv) Tramadol

v) Lignocaine patches

EPSTEIN BAR VIRUS

23:40

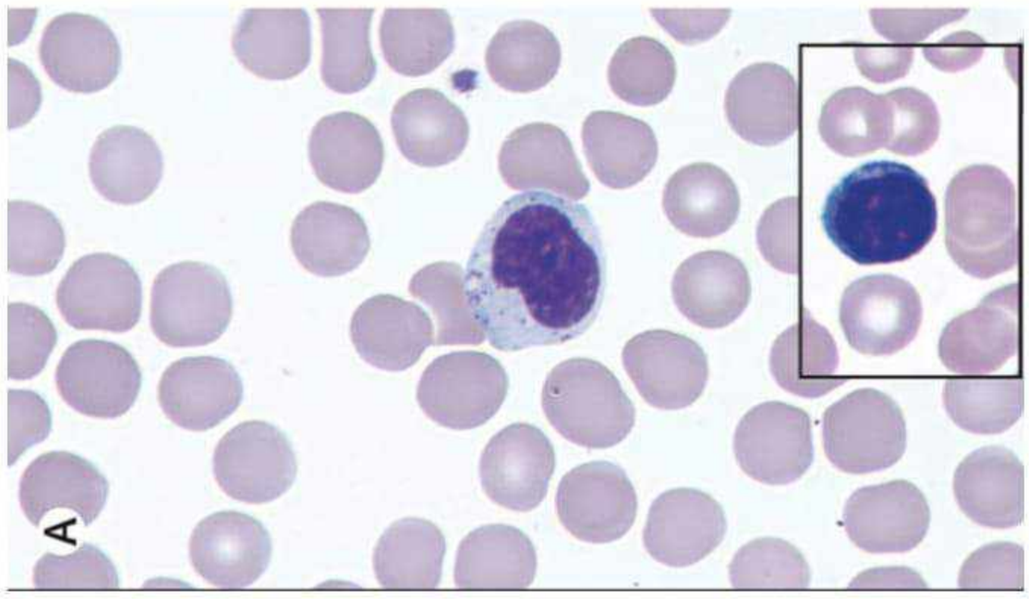
- cause Infectious mononucleosis / Glandular fever / Kissing disease
- HHV-4
- Infects B lymphocytes > T lymphocytes.
- Lymphadenopathy ⊕
- spreads by saliva.

Clinical features :

- fever
- Lymphadenopathy
- Pharyngotonsilitis. ⟶ sore throat
- Exanthem ⊕
- Forscheimer spots - Petechial lesions at junction of uvula and palate.

active space

On microscopy : Blood film.



- Lymphocytosis
- 10 % → Atypical lymphocytes.

⇓

Downey's cell

Treatment : Symptomatic

- If Ampicillin / Amoxicillin is given

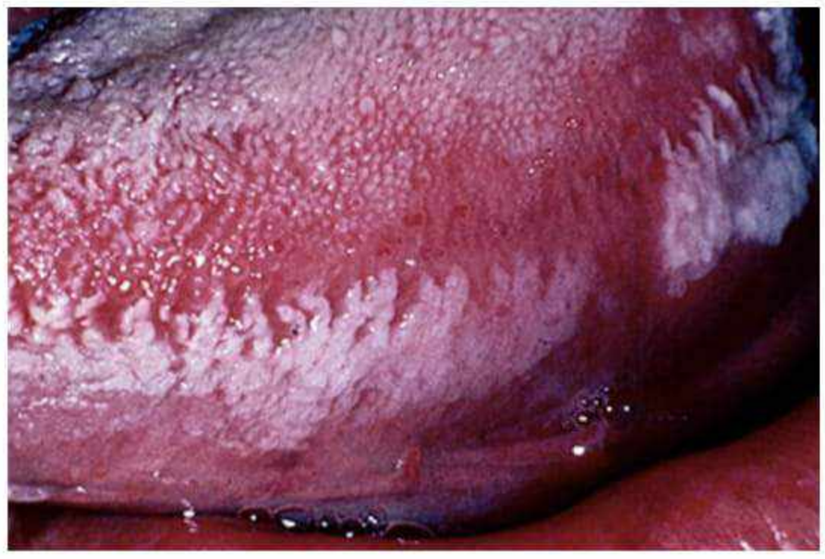
↓

extensive Maculopapular Rash which occurs after 7-10 days.

⇒'AMPI RASH'

2. Oral Hairy Leukoplakia

- white rippled feathery plaques on tongue (lateral border)
- seen in chronic EBV shedding / HIV
- would not come out on scraping
- Pre-malignant condition.



Treatment : HAART Regimen.

active space

Other EBV associated conditions :

i) Burkitt's lymphoma

ii) Hodgkin's lymphoma

iii) Nasopharyngeal carcinoma

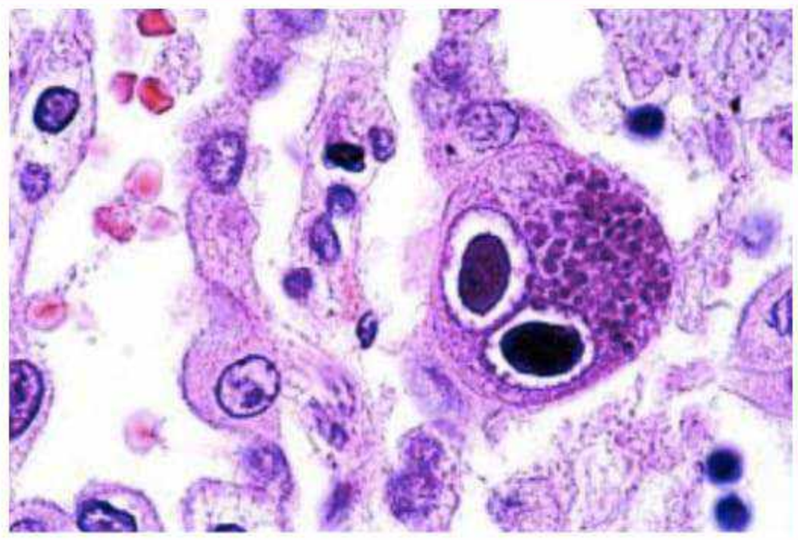
CYTOMEGALOVIRUS

28:42

- HHV-5
- presents with Flu like symptoms

Owl's eye nucleus :

- Intranuclear basophilic inclusion bodies present in enlarged cells with peripheral halo.



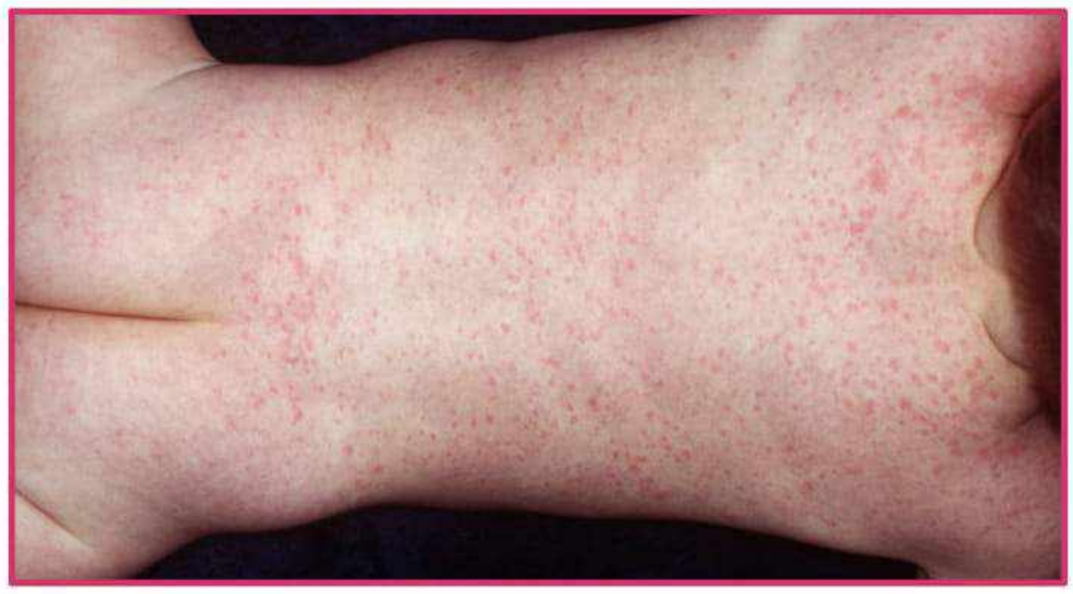
HHV 6 and 7

30:00

- cause P. rosea
- Trigger factor for Lichen planus
- cause Roseola infantum / Fifth disease.

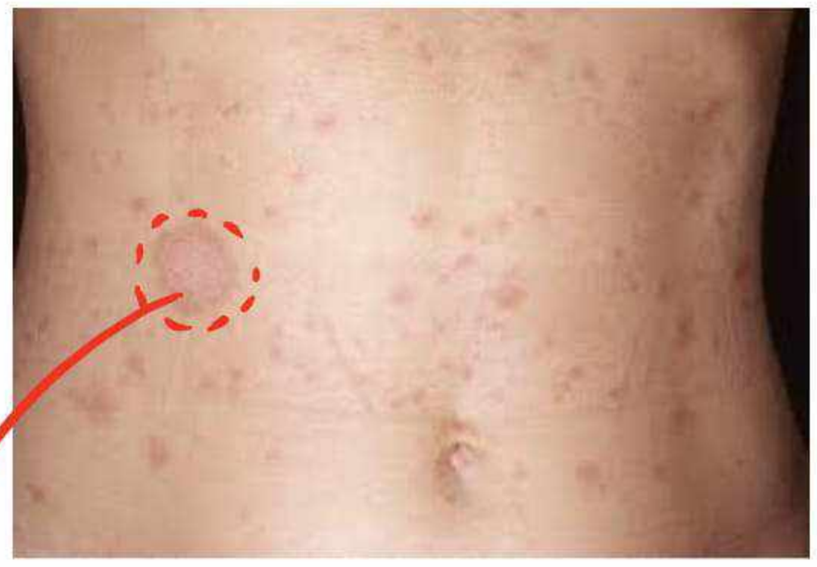
active space

Roseola Infantum



- Exanthem subitum → sudden.
- (MC) exanthematic fever in children < 2yrs.
- I.P → 10-15 days
- Abrupt onset of fever x 3-5days
- Sudden onset of Red rose pink rash over the body.
- Lymphadenopathy ⊕
- Nagayama spots ⊕ → Red papules on palate

Pityriasis Rosea



- caused by HHV 6 and 7 has been postulated.
- Sorethroat or G.I infect 2-4 wks prior to rash. → May be

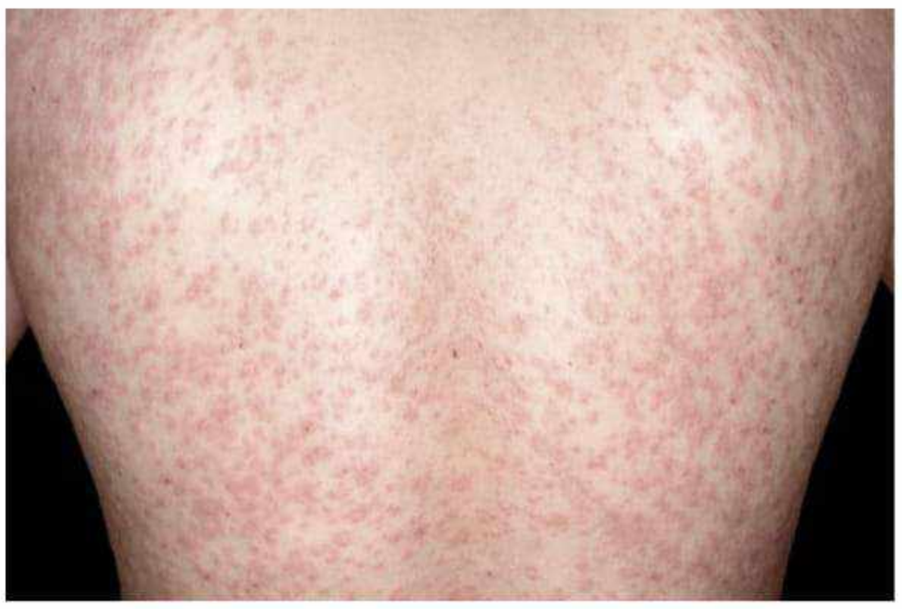
Mother patch / Herald patch

active space

Herald patch: large erythematous plaque with scaling present on trunk ⟶ 1st lesion

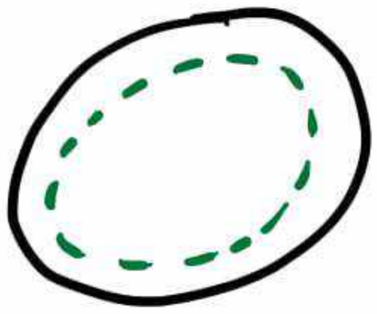
↓

- Multiple smaller (daughter patches) plaques all over the trunk
- Back ⟶ lines of cleavage.



} Christmas Tree appearance

- Collarette of scales ⟶



Treatment: Symptomatic

HHV - 8

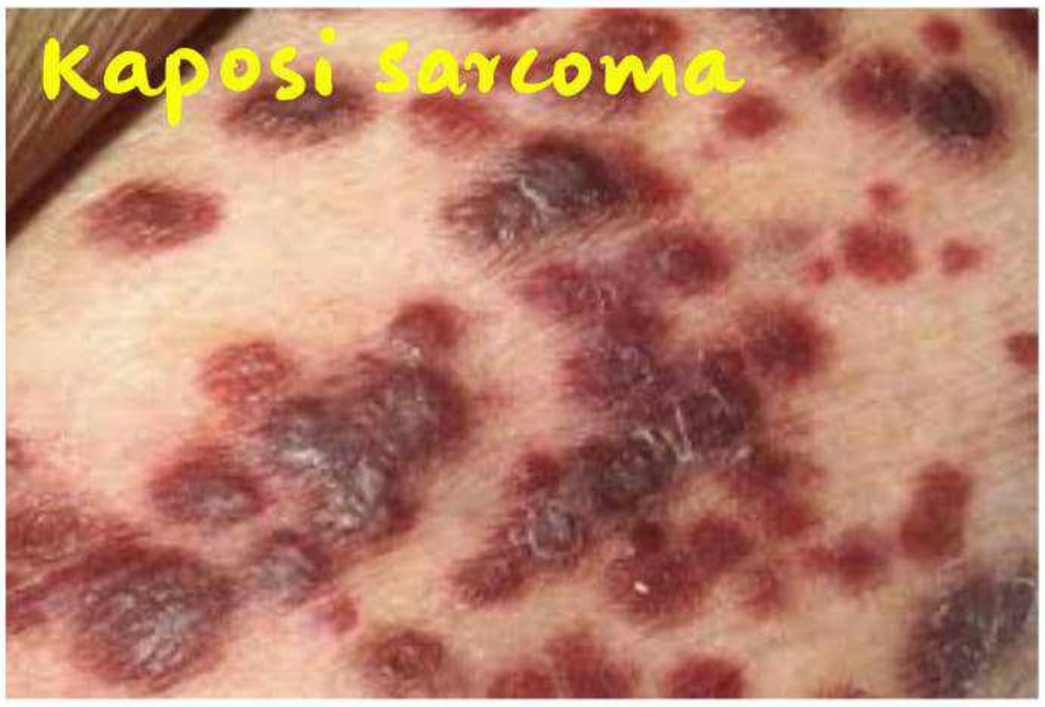
33:45

- Cause Kaposi sarcoma, Primary Effusion lymphoma and Castleman's disease.

active space

Kaposi sarcoma

- vascular tumor endothelial cells of HIV⊕ patient
- Types :
  - i) Endemic - African
  - ii) Sporadic
  - iii) Epidemic
  - iv) Iatrogenic



- Deep dusky red papules/nodules present on legs, mucosa, viscera

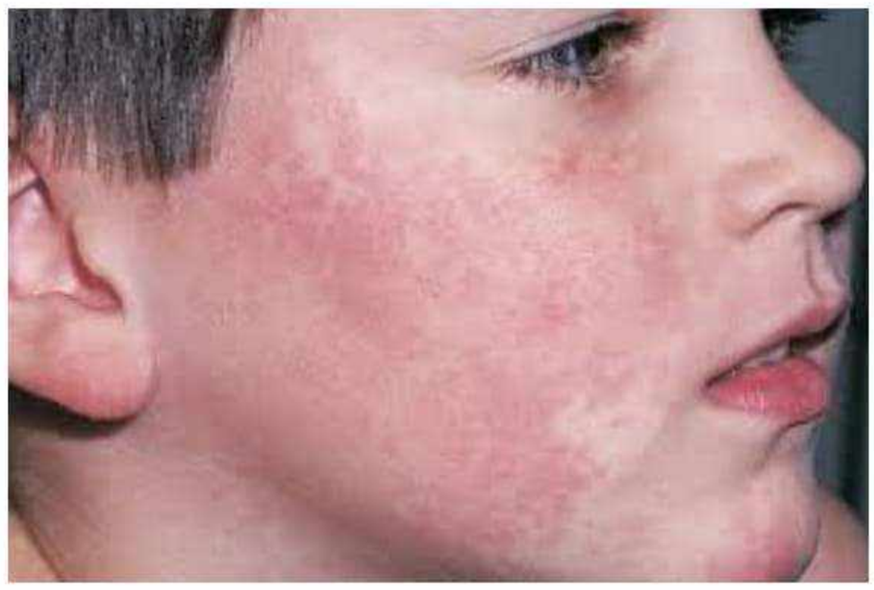
PARVOVIRUS B-19

38:00

- cause

Erythema infectiosum.

- seen in children
- No prodrome
- Slapped cheek appearance :



Rose Red discrete papules on cheek
↓
coalesce to form hot turgid erythema.

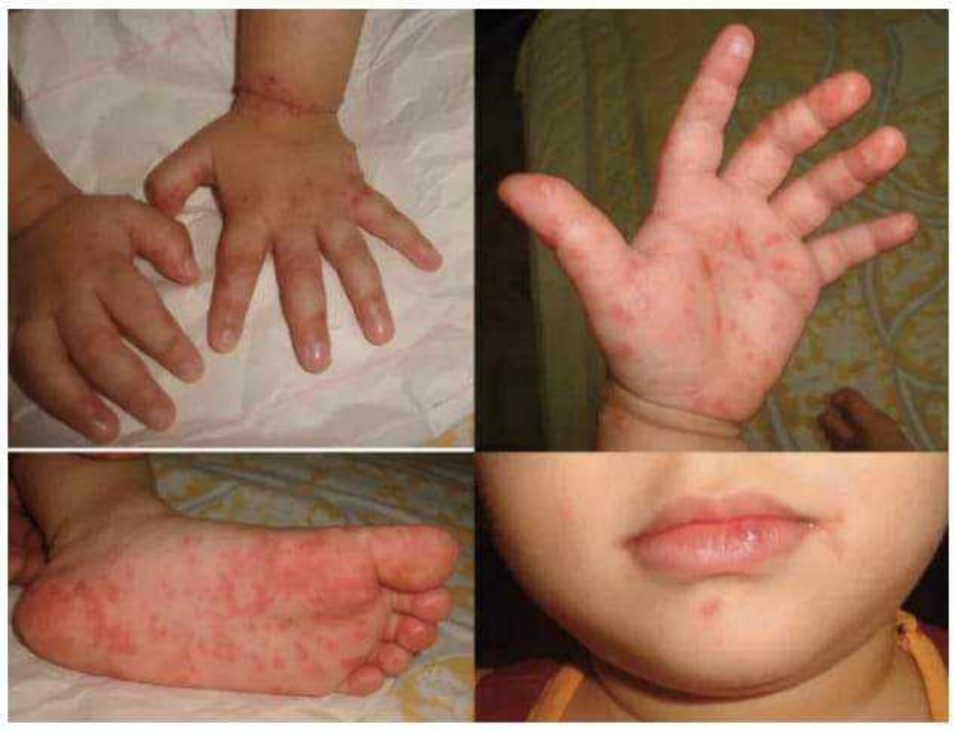
- Perioral pallor

ENTERO VIRAL DISEASE

39:30

1. HFMD (Hand Foot Mouth Disease)

- caused by Coxsackie A16 and Enterovirus 71.



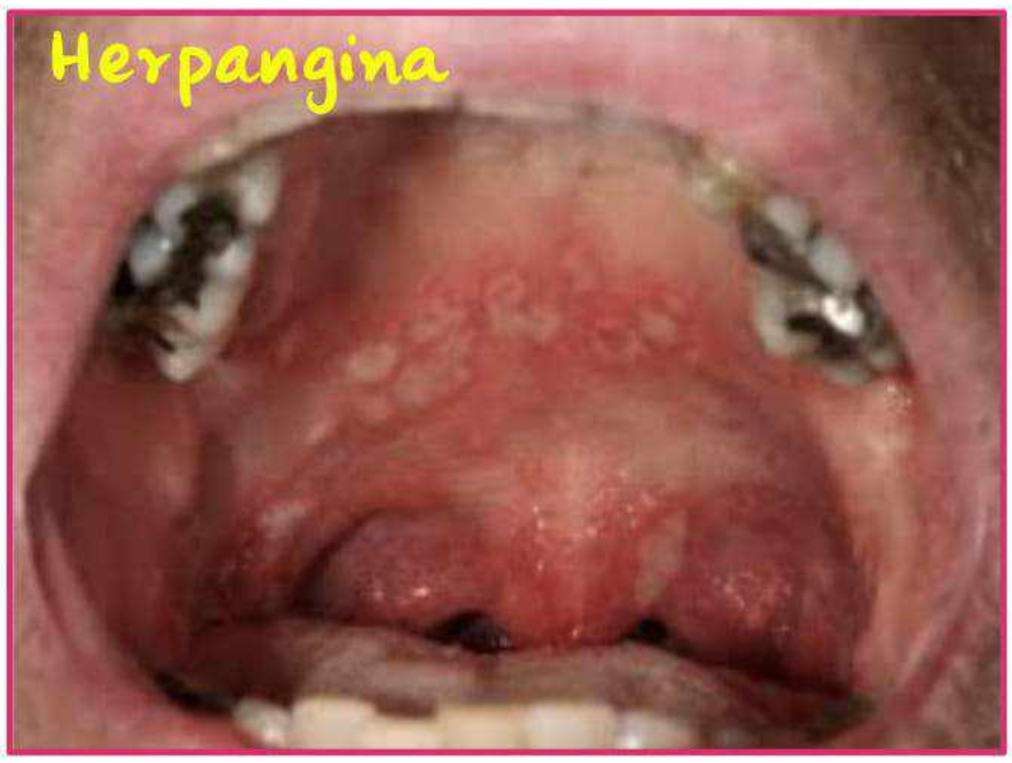
Clinical feature:

- vesicles on hand, foot and around mouth in children.

Treatment: symptomatic (supportive)

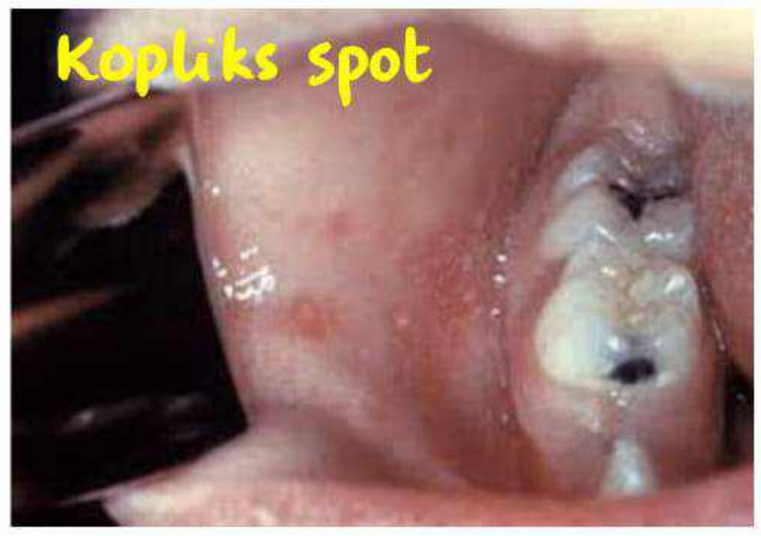
2. Herpangina

- caused by Enterovirus 71 and Cox virus 2,4,5
- presents with superficial ulcers on the palate.
- painful ulcers on palate.

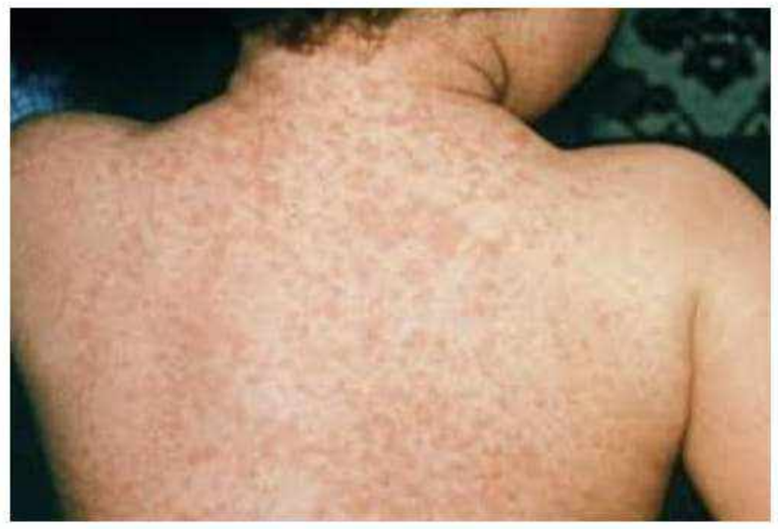


MEASLES

41:50



- not commonly seen
- highly infectious
- caused by Morbillivirus - Myxovirus.
- Presents as :
  - Prodrome → cough, coryza, conjunctivitis, Kopliks spot.



Kopliks spot :

- Bluish white macules seen by an erythematous halo → seen opposite to pre-molars.

↓ followed by

4th day → Rash → Exanthem neck and behind ears

↓

spreads to trunk and limbs within 24 hrs.

RUBELLA

44:35

- a.k.a German measles.
- caused by Rubella virus (Togaviridae)
- IP : 14 days.

active space

- No prodrome
- Forscheimer's spots : Red petechial like lesions on soft palate

↓

Lymphadenopathy

↓

Rash

- Congenital Rubella Syndrome

Gianotti Crosti Syndrome :

- ? viral etiology
- a.k.a Papular acrodermatitis of childhood

Clinical feature:

- Papules - discrete

  seen on buttocks, thighs, legs, arms and face.
- Self limiting condition.

- First disease - Measles
- Second disease - Scarlet fever (Streptococcus)
- Third disease - German measles / Rubella
- Fourth disease - SSSS / Duke's disease.

active space

- **Fifth disease -** Erythema infectiosum (Parvovirus)
- **Sixth disease -** Roseola infantum (HHV 6,7)

active space

Parasitic Infestations

i) Scabies

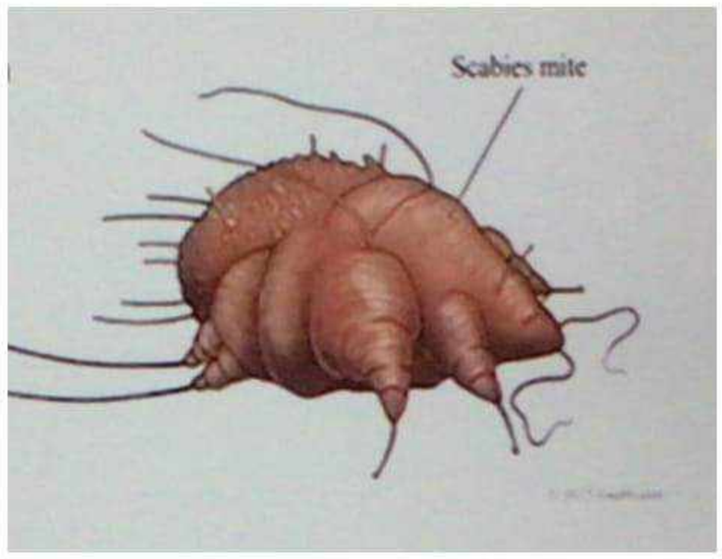
ii) Pediculosis

SCABIES

01:05

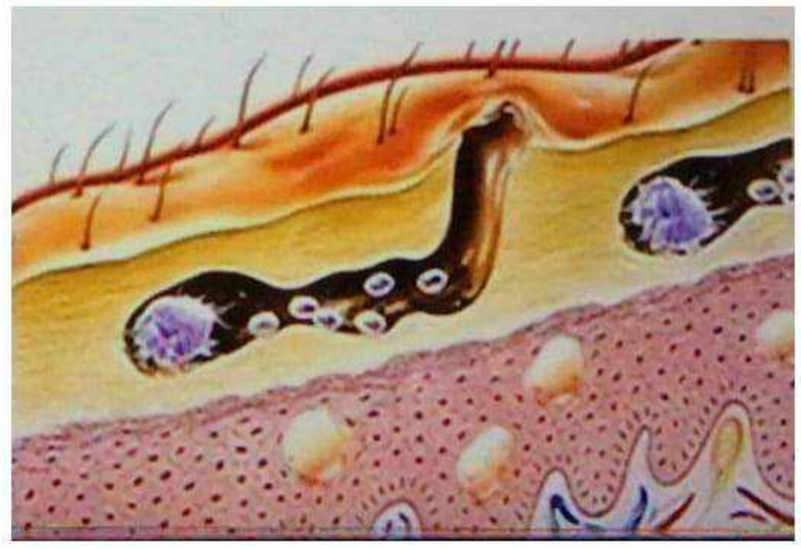
- organism : Mite ⟶ Sarcoptes scabei (variety humanus)
- Transmission :
  - close personal contact
  - overcrowding
  - Lack of sunlight
  - clothes not wash.
  - ⊕ Family h/o
- Predisposing factors :
- Sexually transmitted (genital lesions)
- a.k.a Water Washed disease.
- Immunity → Immediate
  → Delayed → Itching may persist for a month even after treatment

- a.k.a 7 year Itch.



Mite :

- Female → 0.4 mm ; Responsible for transmission
- Male → 0.2 mm.
- Identified by 4 pairs of legs.
- Female mite copulates and lays eggs at end of burrow
- Lays 40 - 50 eggs in a life span of 4 - 6 wks



- Doesnot like areas with high sebaceous gland activity.
  face is not affected

Life cycle

- Egg → Larva → Nymph —(3 moults)→ Adult
- Number of mites in Normal Scabies : 10 - 12 mites
  - in Crusted scabies : Millions.

active space

Clinical features :

- I.P :
  - 4 to 6 wks in Primary infection
  - 1 to 2 wks in Recurrent infection.

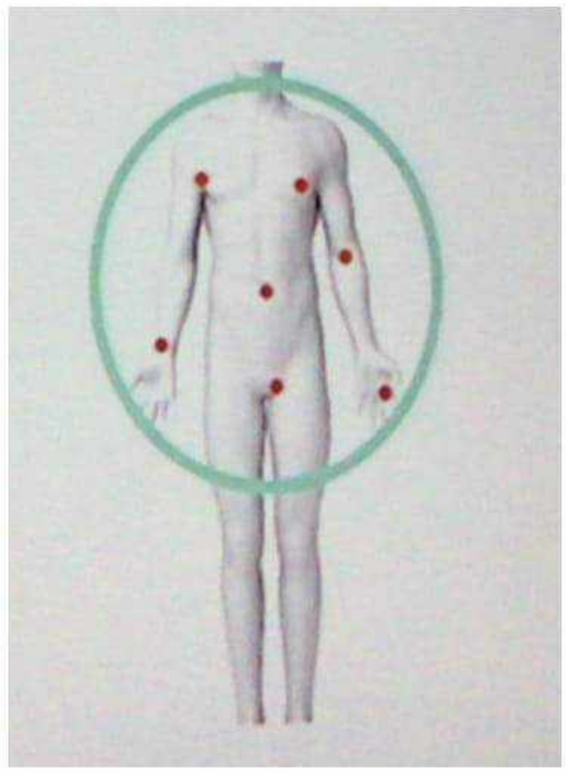
Symptoms :

- Itching (Nocturnal)

Site :

- doesnot affect areas with ↑sed Pilosebaceous activity.
- Affects :
  - i) Inter web spaces
  - ii) wrists
  - iii) Anterior axillary fold
  - iv) Cubital fossa
  - v) Mammary area
  - vi) Umbilicus
  - vii) Groins.

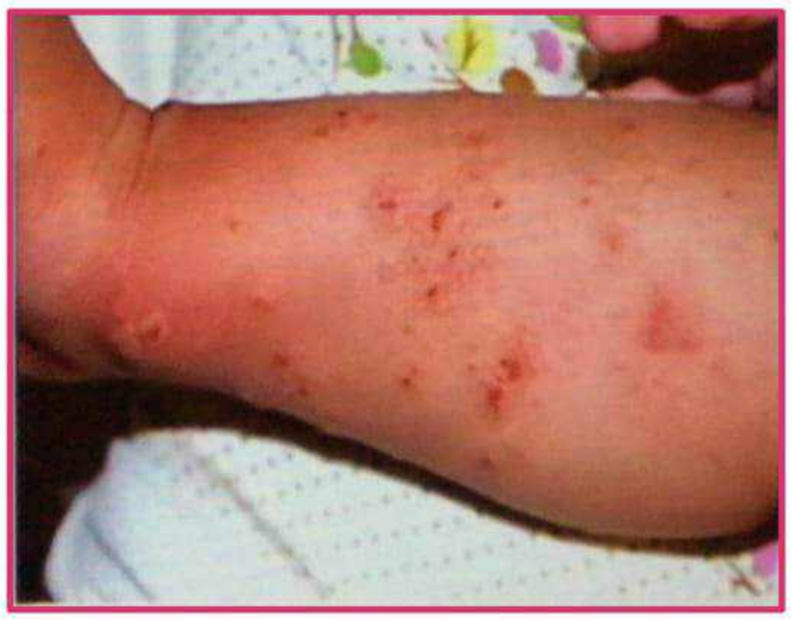
} Circle of Hebra



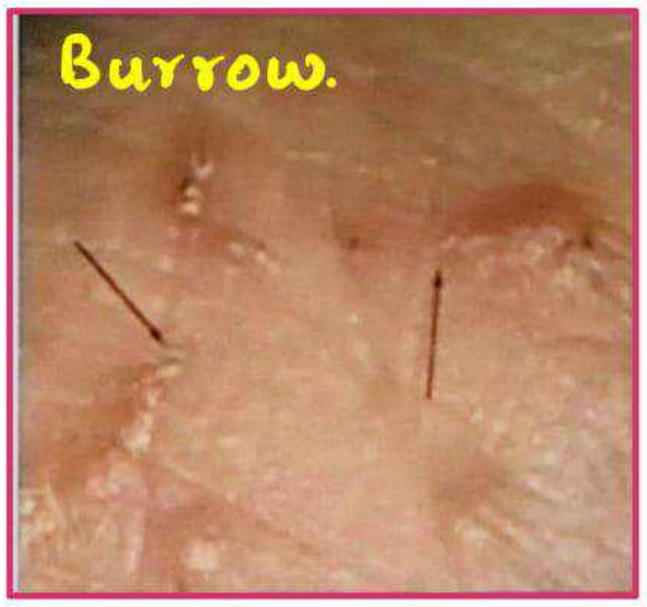
- doesnot affect face, palms and soles.

presents with :

- Itchy papules
- vesicles
- excoriation marks
- crusting



- Burrow.
  ↓
  special skin lesion
  ↓
  Pathognomic scabies lesions
  ↓
  linear wavy or



serpentine lesion which represent the track laid down by female mite.

- Burrow can extend upto Stratum malphigi

- (MC) site of Burrow → Stratum corneum.
  ↳ (MC) seen in Wrist

- Nodules (+) on genital area

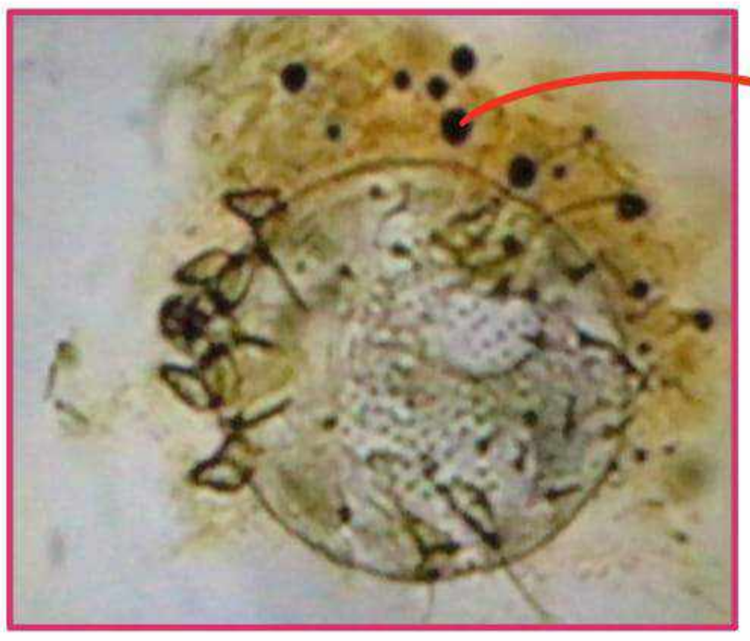
active space

Secondary changes :

- 2° infection → Bacterial
- Crusting
- Lymphadenopathy
- Nodules → ↑ Hypersensitivity response → Groins, axillary folds.

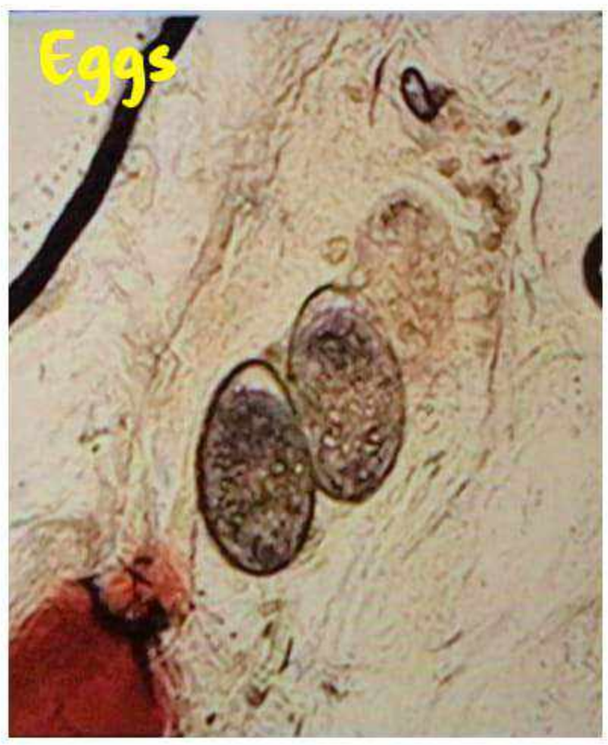
Diagnosis :

- mainly clinical →
  - H/o Itching ↑ at night
  - Typical sites
  - H/o Family members
  - Classical lesions.



On examination under microscope by putting mineral oil :

- Mite
- Eggs
- Faecal matter. → Scybala



active space

On Dermatoscopy :

- Mite in its burrow
- Jet in contrail appearance.

Treatment :

1.General measures :

- usually needs to be repeated after 7-10days.
- Treatment of family members /contacts.
- Wash all linen and clothing
- Pruritus may persist for 4wks - Extended antihistamines
- Treat 2° infections → Antibiotics
- If only Genital scabies → consider possibility of STI
- Application of creams.
  - ↳ it has to applied :
    - i) in adults - below neck → 30g in one time
    - ii) in infants - face and scalp
      - ↓
      - 15g - < 5yrs

active space

Drugs used :

Topical

- 5% Permethrin - for > 2months age.
- Precipitated Sulphur - for < 2months age.

} TOC

- Permethrin blocks Na^+ gated channels → Paralyse the mite
- Repeat application after a week.
- Others :

i) 1% GBHC (Lindane) → Neurological S/E
↳ Not used in infants, pregnant females, individuals with neurological issue.

ii) Malathion

iii) Crotamiton

iv) Benzyl Benzoate 25%

v) Monosulfiram

Systemic :

- Oral Ivermectin (200µg/kg)
↳ also used in • Onchocerciasis
• Strongyloides.

active space

- Ivermectin MOA : ⊖ Gamabutyric acid induced transmission
  ↓
  Paralysis of mite

Supportive Treatment :

- Antihistaminics
- Antibacterials

Crusted Scabies :

- a.k.a Norwegian Scabies
  ↳ 1st reported outbreaks in lepers of Norway.
- Patients with sensory impairment
- DOWN'S Syndrome
- Immunocompromised individual
- Mental Retardation
- Physical disability

} doesnot Itch.

- Millions of Mites ⊕
  ↓
  Masses of Horny debris
  ↓
  accumulate → crust (heaped up)
  ↓
  Teaming with mites ⟶ Highly contagious

- Responsible for Outbreaks of Scabies.

Treatment :

- Topical + Oral / Systemic Treatment
- Isolated and Hospitalised
- Salicylic acid ⟶ Keratolytic
- Cut short nails.

Atypical Scabies :

- Infantile scabies :
  - Scalps, Palms and Soles involved
  - Vesicular lesions on palms and soles
  - Extensive eczematization

Nodular Scabies :

- Formation of nodules on genitalia
- ↑ Hypersensitivity
- Persistent ⟶ use Topical steroids

Genital Scabies :

- STI
- Nodular

active space

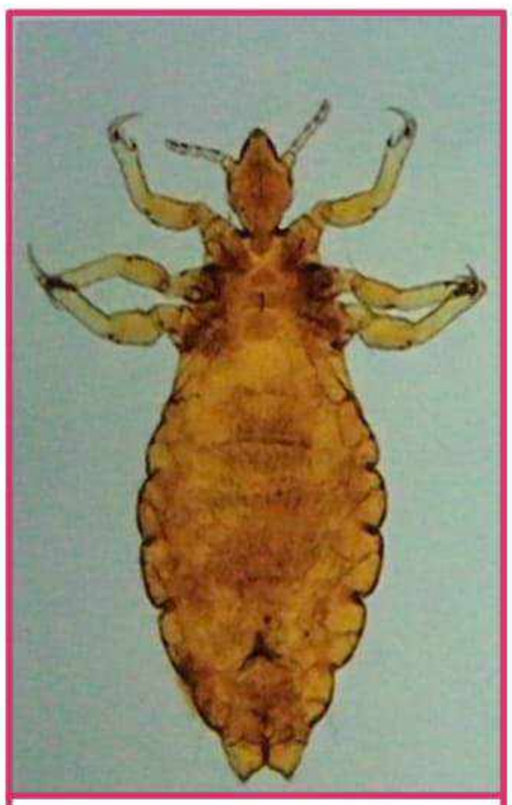
Animal Scabies: No burrows.

PEDICULOSIS

39:06

- caused by a lice → Pediculus

Pediculus
- P. humanus → P. humanus humanus → **Causes:**
  - Pediculosis corporis
  - Body lice
- Pthirus pubis → P. pubis.
  - P. capitis



- Pediculus
- 3 pairs of legs

- Phthirus Pubis
- 3 pairs of legs.
- Broader, Stouter, Pincer like claws.

Pediculosis capitis

- seen in children (school children females > Males)
- Age group : 3 - 11yrs
- Lice or Nits can be seen
- Nits → Empty egg cases
- Active infection
  ↓
  i) Presence of Lice
  ii) Nits within 1cm of scalp

Presents with :

- Itching / Pruritus of scalp
  ↓
  2° infection
  ↓
  Crusting, Lymphadenopathy, Matting of hair → Plica polonica.
- H/o similar complaints → Family members
  → Schoolmate.

Pediculosis Corporis :

- a.k.a Vagabonds disease.
- seen in destitutes who don't change clothes / don't wash them

active space

- present in seams of cloths
- lice is in cloth

↓

at night come to skin

↓

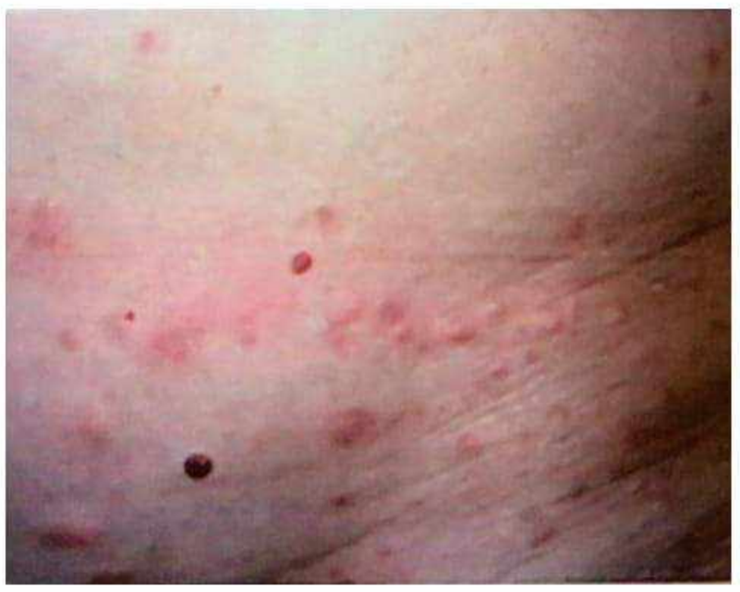
Take feed and go back

Presents as :

- Itching
- Excoriation marks on body

↓

No lice isolated from body.



- Morbus eronum → Post inflammatory phenomenon, brownish lesion.

Pediculosis pubis

- caused by Pthirus pubis
- Lice sitting in pubic hair (can be seen in other hair bearing areas also).
- Macula cerulae - Brownish macules

↓

extravasation of blood on skin.

active space

Treatment :

- Repeat after 7-10 days
- Treat contacts → P. capitis.

i) Topical :

- 1% Permethrin
- In cases of P. capitis → Treat the 2° infection
  ↓
  Streptococcus
  ↓
  PSGN
- In cases of P. corporis → Treat the clothes
- In cases of P. pubis → Rule out STI
- Cotrimoxazole → used in P. capitis
  ↳ used systemic → destroys symbiotic bacteria in the gut of lice.
- Recurrent folliculitis in young girl → (MC) cause Pediculosis

ii) Systemic :

- Ivermectin 200µg/Kg at 0,7 days

active space

Vector Borne Diseases

LEISHMANIASIS

00:25

- Protozoal disease → Leishmania
- vector → Sandfly
- Classification :

i) Cutaneous → Present with Chronic ulcer → Old World / New World.

ii) Mucocutaneous

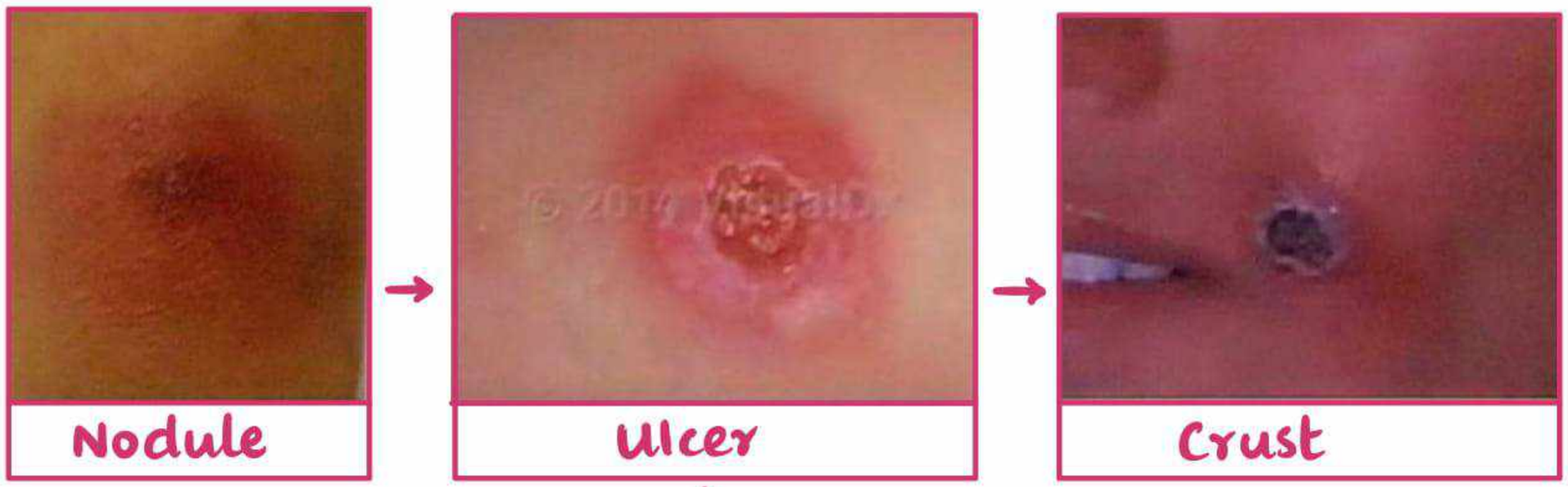
iii) Visceral → Kala azar → Patient presents with
- fever
- Hepatosplenomegaly
- Leucopenia

→ sequelae → Post Kala Azar Dermal Leishmaniasis.

Old World Cutaneous Leishmaniasis :

- a.k.a Oriental Sore, Delhi Boil, Baghdad boil.
- Species causing are → L.major, L.tropica, L.aethiopica and L.infantum.

- Endemic areas in India → Rajasthan
- Site : Face, Exposed parts of limbs.

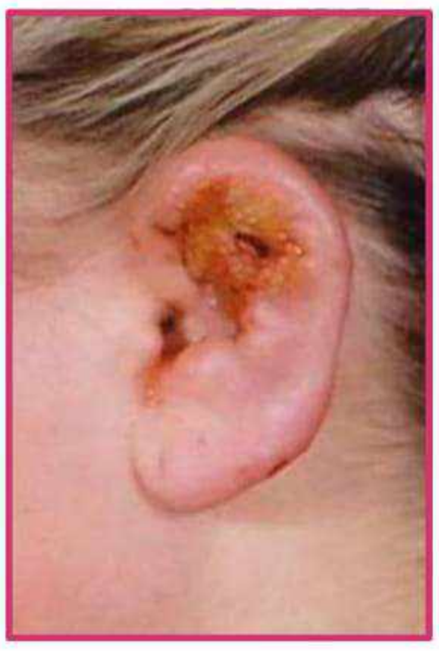


↓

Heals

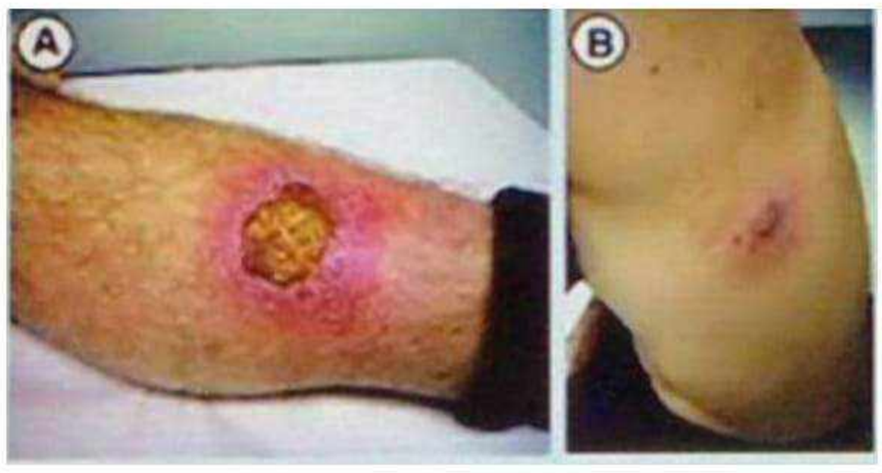
- Annular lesion with central crusting → Leishmaniasis

New World Cutaneous Leishmaniasis



- caused by L.mexicana
- cutaneous ulcerations involves ear cartilage
- Chiclero's ulcer.

Mucocutaneous Leishmaniasis



- caused by L. braziliensis
- 1° lesion → painless nodular lesion
  ↓
  ulcerates
  ↓
  present on lower limbs
  ↓

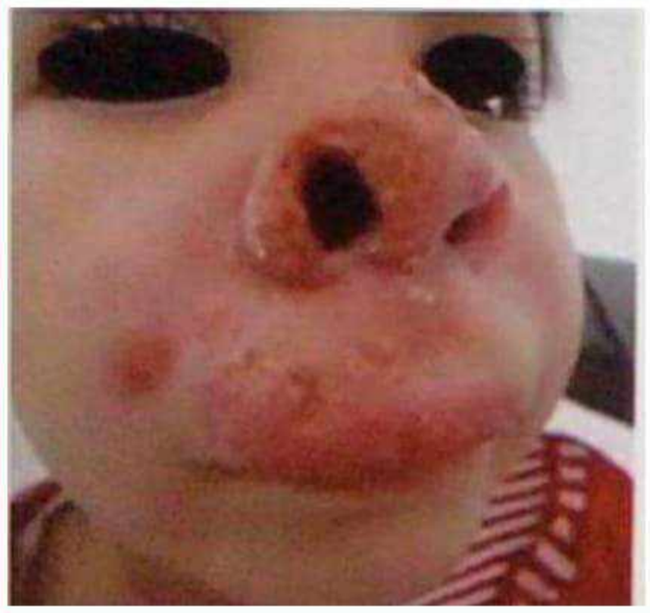
↓

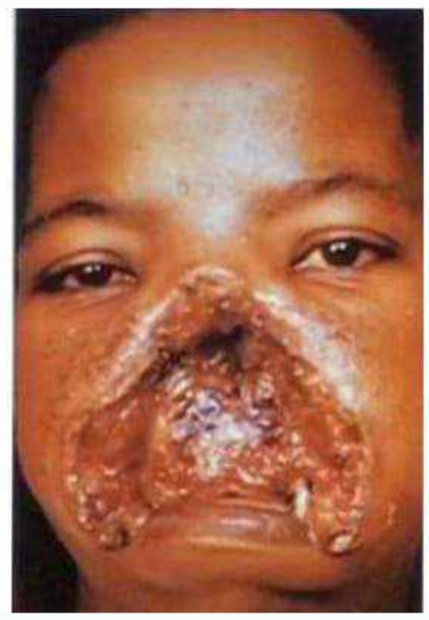
Hematogenous spread to mucosa

↓

Mucosal lesions (ESPUNDIA / SPONGE)

↓





- may involve :
  - Nose
  - Larynx
  - Lips
  - Palate
  - Pharynx

- Nodules on nose → Crusts → Ulcerates → Perforation of nasal cartilage.
- 'Tapir Nose' Sign.

Post Kala azar Dermal Leishmanias

- develops years after visceral leishmaniasis (Kala azar)
- H/o prolonged fever, Hepatosplenomegaly,
- species causing : L.infantum, L.chagasi and L.donovani (In India)
- Endemic areas : North Eastern areas, Bihar, Assam, West Bengal.

Presentation

ill defined hypopigmented macules

↓

Starts over face

↓

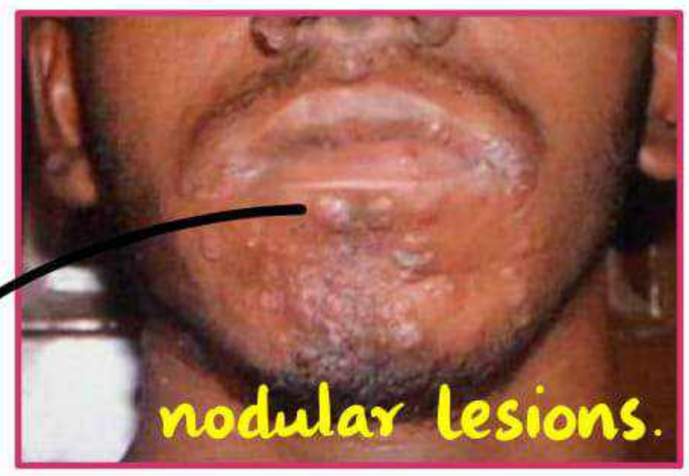
spreads to trunk and extremities.

↓

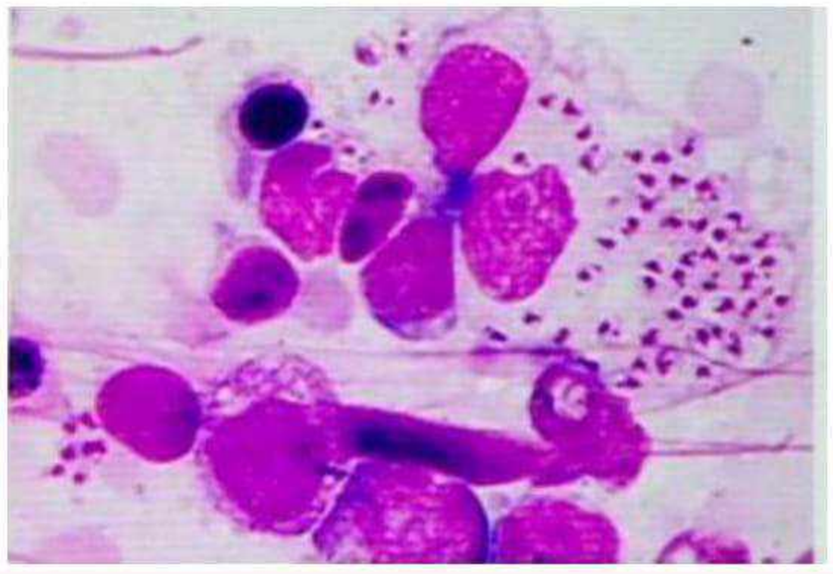
Becomes nodular lesions.

(seen on muzzle area of face, butterfly rash may also be seen)

soft skin coloured nodular lesions



On Slit Skin Smear : Giemsa Stain



LD Bodies (Leishman Donovan Bodies)

↓

These are amastigote form of Protozoa (non-motile and seen intracellularly)

Treatment : Miltefosine

TRYPANOSOMIASIS

12:15

- cause by Trypanosoma
- Classified into 2 forms :

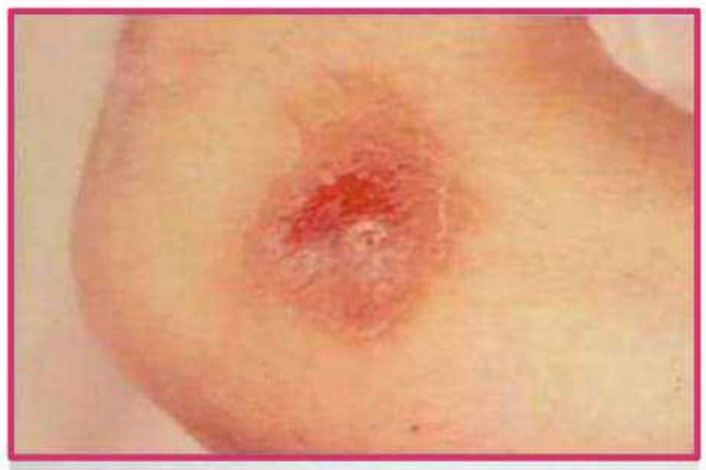
  i) African → a.k.a Sleeping Sickness

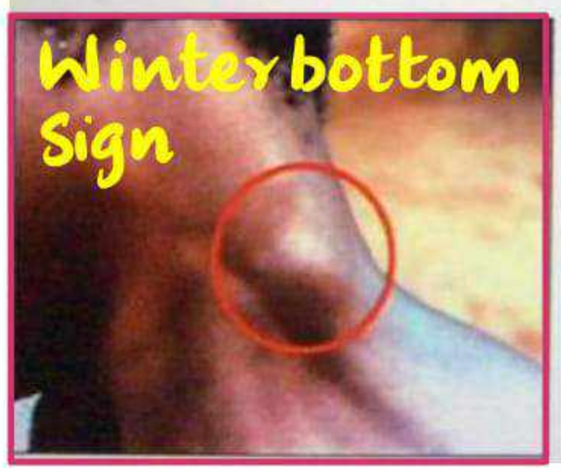
  ii) American → a.k.a Chagas disease

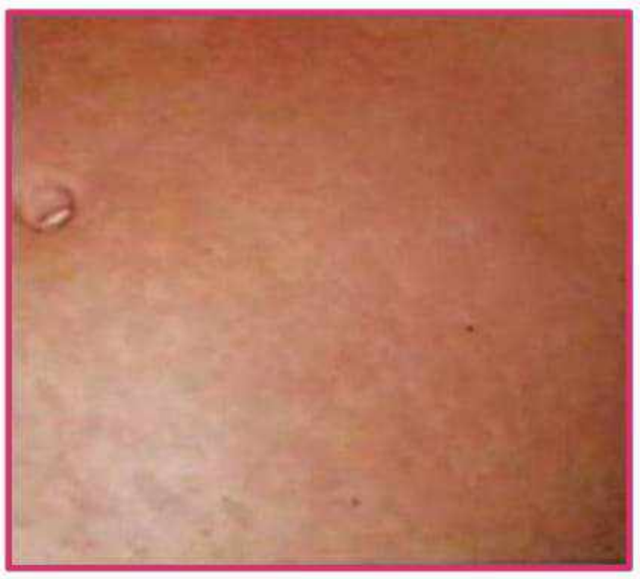
- African form :
  - a.k.a Sleeping Sickness
  - Species : i) T. brucei gambiense → cause in West Africa
    ii) T. brucei rhodisiense → more virulent in East Africa
  - Vector : Tsetse fly

Presentation

- Chancre on lower limbs
  ↓
  Painfull well defined ulcer
  ↓
  lymphadenopathy involves cervical and posterior occipital LN
  (Winterbottom Sign)







→ Generalised circinate Rash

Treatment :

1. Suramin
2. Pentamidine

American form

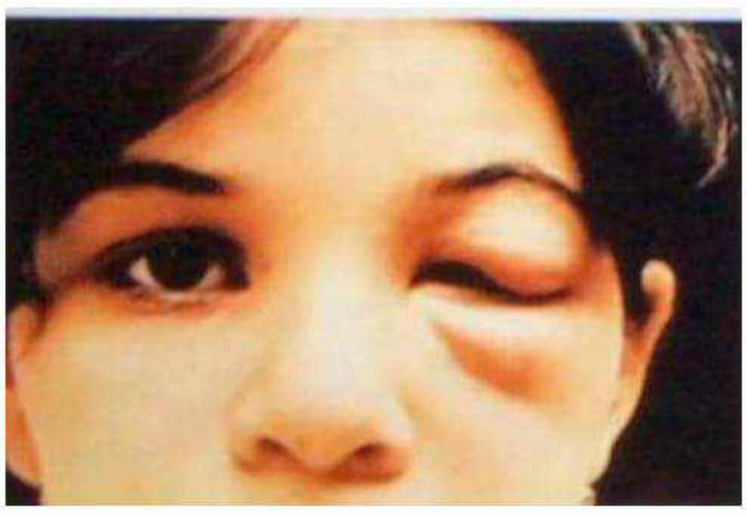
- a.k.a Chagas disease
- Species : T. cruzi
- vector : Reduvid bugs (Kissing Bugs)

Clinical feature

- u/l edema of eyelids and lacrimal gland

↓

ROMANA's Sign



Treatment : Nifurtimox (Lampit) or Benznidazole (Rochagan)

active space

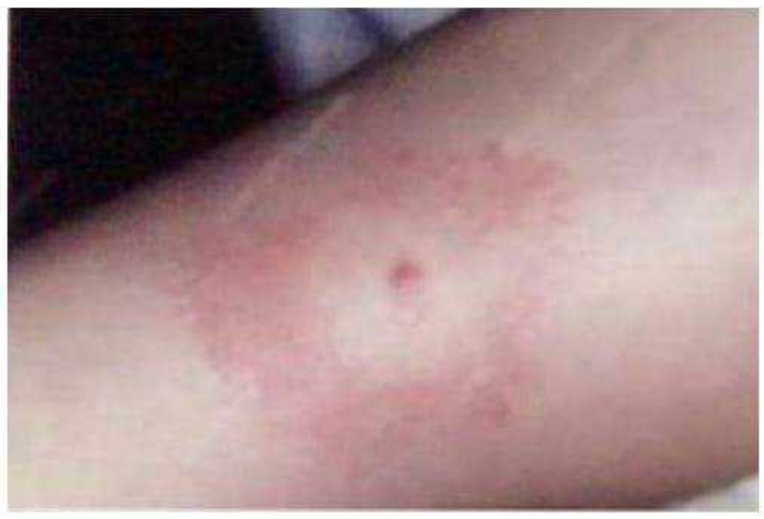
ERYTHEMA CHRONICUM MIGRANS

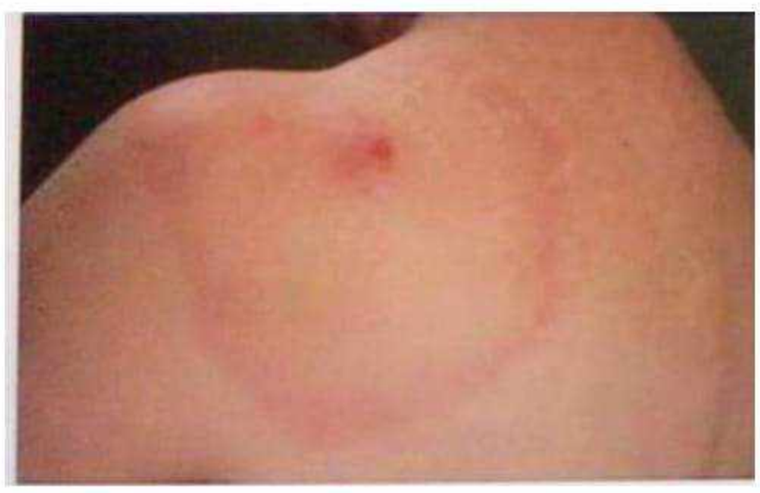
17:05

- Cutaneous manifestation of lyme disease
- Chronic migrating Red rash.
- caused by → Borrelia burgdorferi (spirochete)
- vector : Ixodid Tics.

3 phases of Lymes disease :

- Phase 1 : ECM
- Phase 2 : Cardiac and neurologic disease
- Phase 3 : Arthritis and a chronic neurologic syndrome





- Central bite → Erythematous papule.
- Migrating annular Ring surrounding the central punctum.
- No scaling , No vesiculation

↓

central areas of clearing.

active space

Cutaneous Tuberculosis

- Caused by M.tuberculosis
- Comprise 1-2% cases of extra-pulmonary TB.
- Cutaneous Tuberculosis will depend on :
  - i) Virulence of organism
  - ii) Infective dose
  - iii) Immune status
  - iv) Route of Infection.

  } Most important

CLASSIFICATION OF CUTANEOUS TUBERCULOSIS 01:45

- 3 Types:
  - i) Exogenous
  - ii) Endogenous
  - iii) Tuberculid.

Exogenous

- Source is outside
- Direct inoculation of bacilli from an infected individual through a breach in skin.

Naive

- never been exposed to TB bacilli

↓

TB Chancre

Immune

- prior exposure is present

↓

Tuberculous Verrucosa Cutis (TBVC)

Endogenous

- Source is inside the body
- Source of infection / focus of TB already present

↓

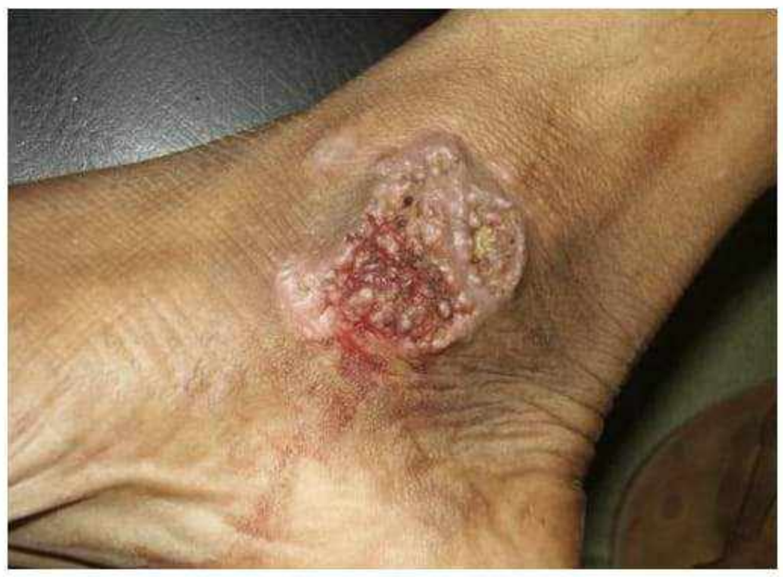
- Contiguous spread
- Autoinoculation
- Hematogenous

- Contiguous spread → i) Scrofuloderma
  ii) Lupus vulgaris (sometimes)
- Autoinoculation → Orificial TB
- Hematogenous → i) Lupus vulgaris
  ii) Acute Miliary TB
  iii) TB Gumma.

active space

TB CHANCRE

- Exogenous infection ; occurs after breach.
- seen in Naive individuals ⟶ low immunity (Multibacillary)
- Common sites : Face and Limbs
- Primary inoculation TB
- Autologous to Ghon's focus in lungs.



Presents as :

Starts as Brownish to Reddish papule

↓

Breaks down to form an ulcer with undermined edges with overlying hemorrhagic crust , bluish margins.

Undermined Edges also seen in :

i) TB Chancre

ii) Chancroid

iii) Pyoderma gangrenosum

- Inoculation $\xrightarrow{2\text{-}4\ wks}$ TB Chancre
- Regional lymphadenopathy after 4-8wks ⟶ forms Cold abscess.

active space

TBVC

13:10

- Exogenous infection in Immune individuals
- High to moderate degree of immunity
- Paucibacillary
- Post 1° cutaneous TB / Anatomist's wart.

- **Population affected:**

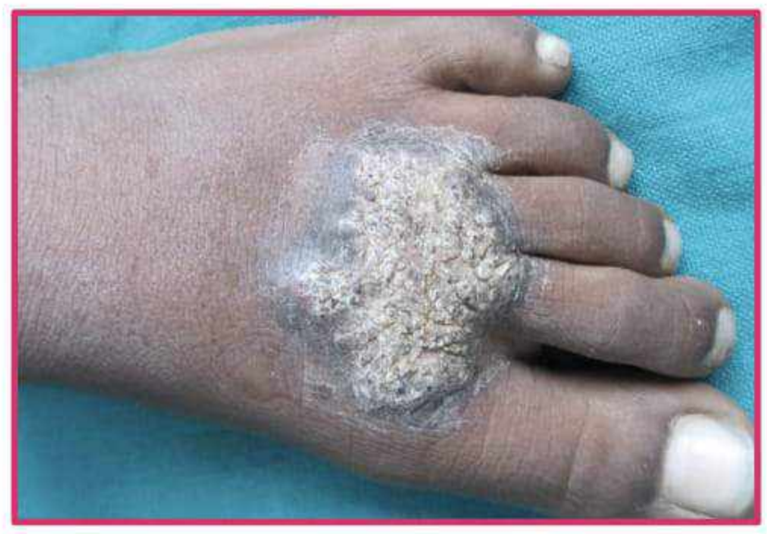
  → Health care workers ⟶ Pathologists, Anatomists

  → Farmers - infected sputum

  → Children - squat in oper fields ⟶ seen in Buttocks.

Presents as:

- Rough hyperkeratotic verrucous plaque which has induration + discharge/crust.
- seen in limbs, buttocks.



Viral warts ⟶ usually multiple, no induration, no discharge.

- heals with scarring

active space

LUPUS VULGARIS

18:00

- Endogenous , good immunity
- Spread
  - Hematogenous (MC)
  - Lymphatic
  - Inoculation.
  - Contiguous
- (MC) form of TB in adults in India
- Plaque Type of TB
- There will be usually presence of underlying focus of TB

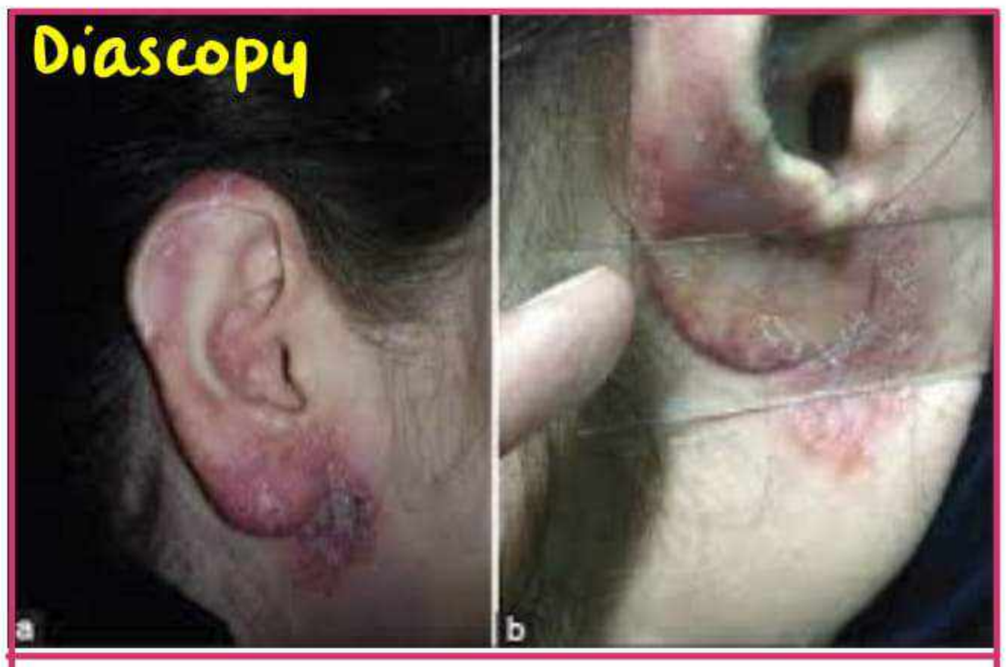
  ↓

  Lymph node, Bones , joints
- Common sites :
  - 80% → Head and Neck
  - Arms
  - Legs
  - Buttocks

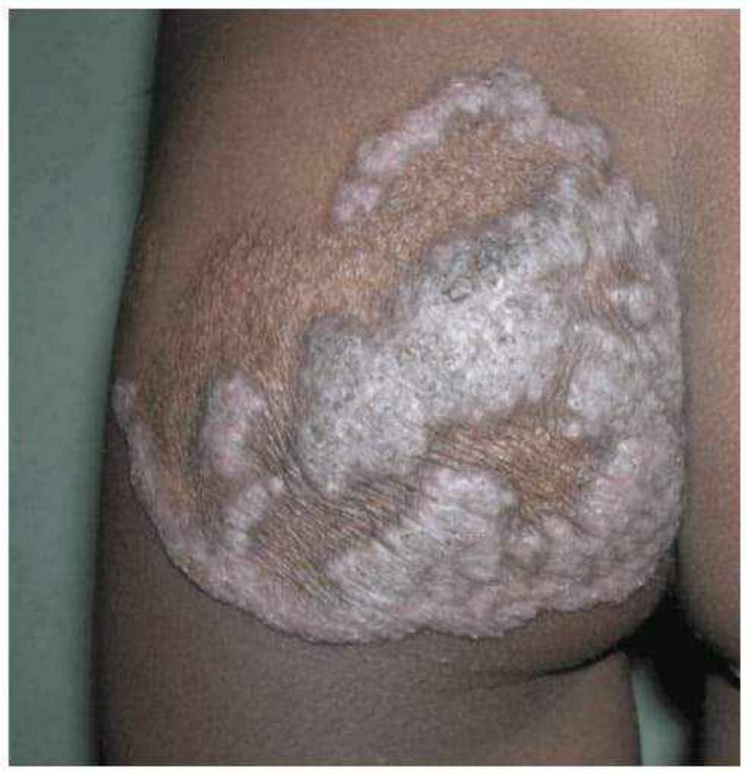
Clinical feature:

- Erythematous plaques which on Diascopy shows Apple Jelly nodules. (represents Granuloma)

active space



Apple Jelly nodules.



- Peripheral extension with healing atrophy and scarring
- Squamous cell CA
- Basal cell CA.

SCROFULODERMA

24:10

- Endogenous
- Contagious spread
- Low immunity, multibacillary

active space

- Contiguous foci :
  - Lymph node ⟶ Cervical > Axillary > Pre and Post auricular
  - Bone
  - Joint
  - Lacrimal glands

Presents as :

- Starts subcutaneous asymptomatic

Bluish Red swelling overlying a gland.

↓

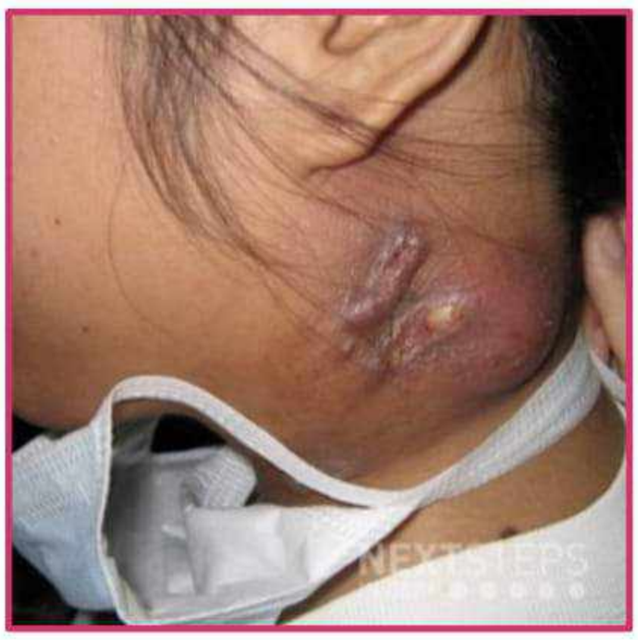
Breaks down to form ulcer

↓

Fistula and Sinuses

↓

Scarring / Fluctuant disease



ORIFICIAL TUBERCULOSIS 27:30

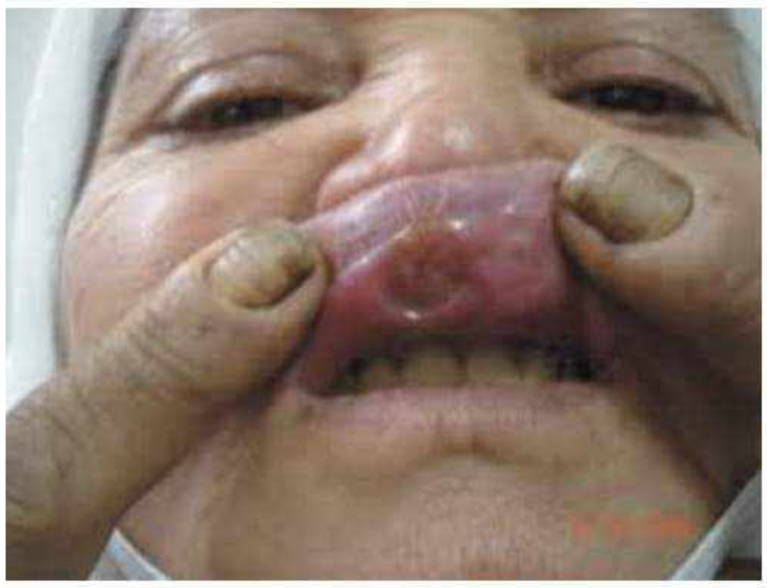
- caused by Autoinoculation
- seen in immunocompromised
- Tuberculosis underlying are:

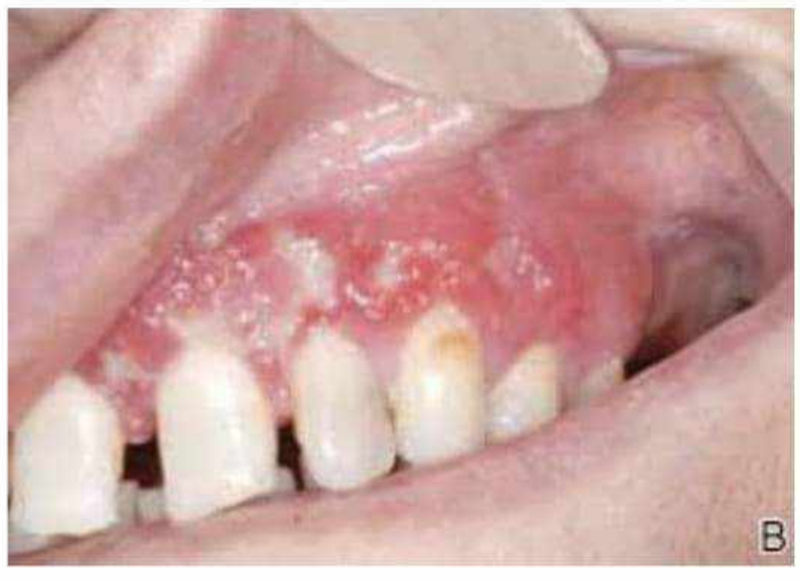
i) Pulmonary iii) Genitourinary.

ii) Intestinal

active space

- (MC) site : Oral mucosa , also seen in perianal area.





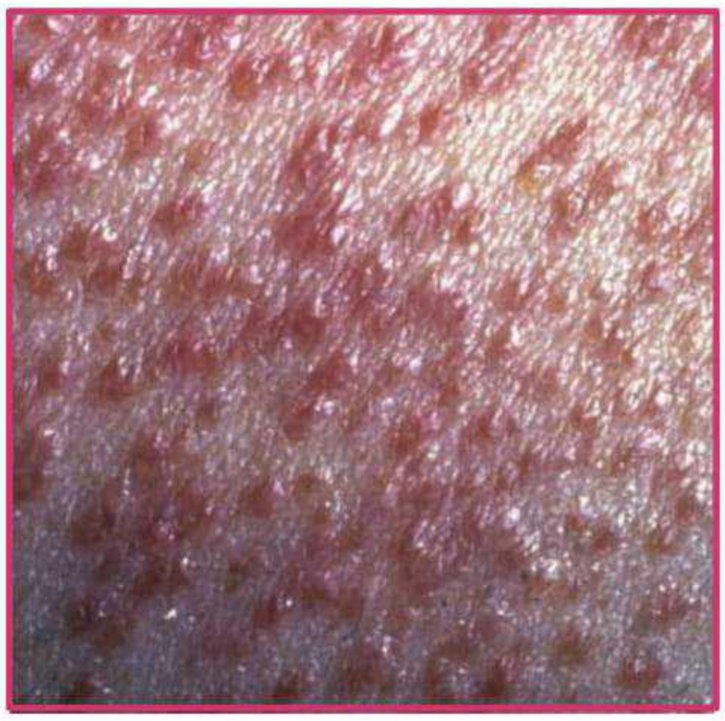
Ulcers with undermined edges bluish margins

- Painful.

ACUTE CUTANEOUS MILIARY TB

29:40

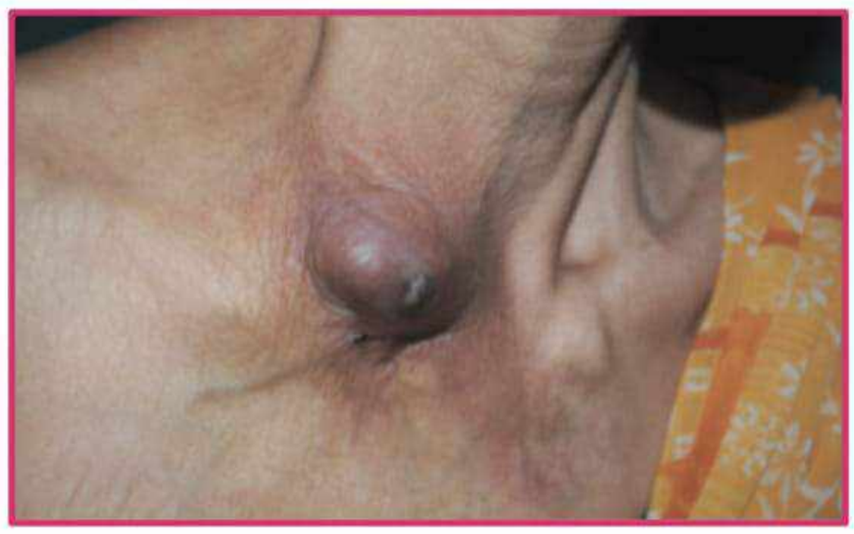
- caused due to Hematogenous spread.
- Patient is sick with low immunity.
- Papules, vesicles are over all body



TB GUMMA

30:30

- Hematogenous dissemination



active space

TUBERCULIDS

31:00

- Hypersensitivity reaction (Type IV) to hematogenous dissemination of bacilli / toxin in patient with good immunity

Criteria to Diagnose Tuberculids:

- Primary focus of TB elsewhere
- Mantoux test positive
- Culture of TB bacilli ⟶ Negative
- AFB smears ⟶ Negative
- Tuberculoid Histology
- Response to ATT ⊕

Tuberculids

- 2 Types :

i) True

↓

Only M.TB is the cause

- Micropapular ↓ Lichen scrofulosorum
- Papular ↓ Papulonecrotic Tuberculid.

ii) Facultative

↓

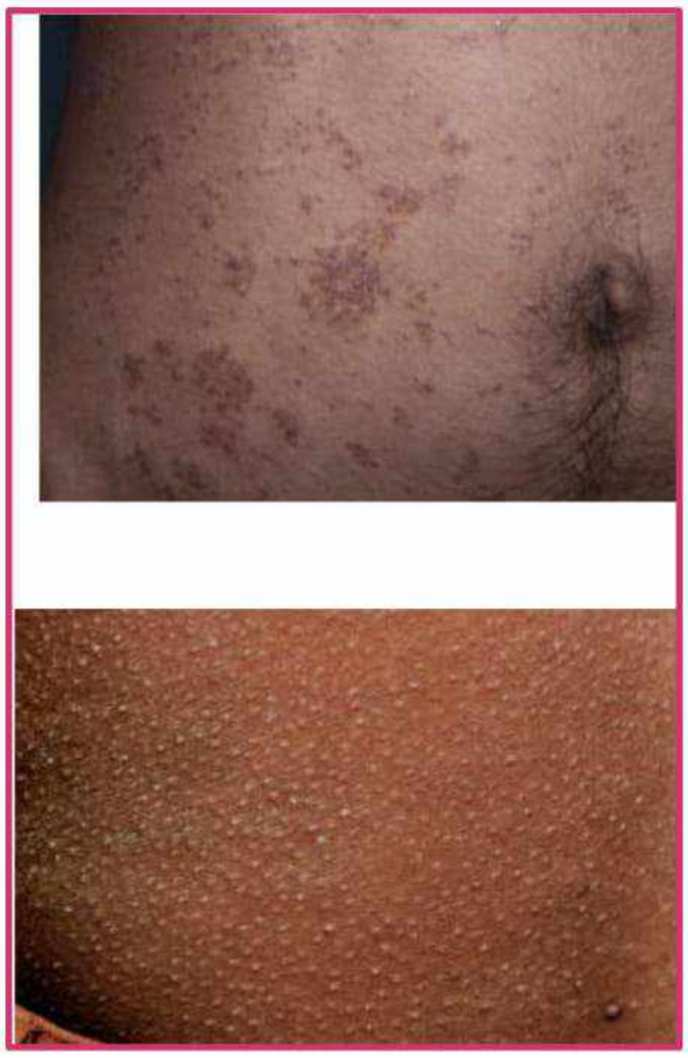
M.TB is one of the causes.

↓

Erythema Induratum of Bazin.

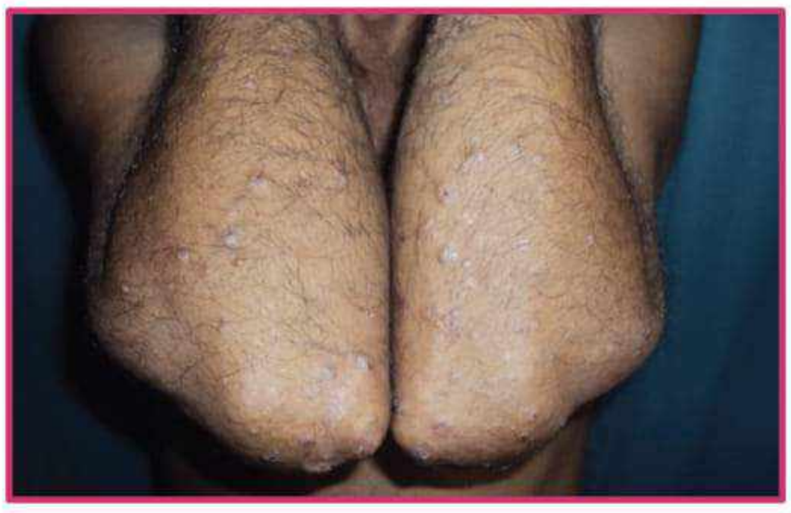
Lichen Scrofulosorum

- Micropapular
- seen on Trunk
- Closely grouped lichenoid papules
- Perifollicular
- Histology : Perifollicular superficial dermal granulomas.
- No scarring



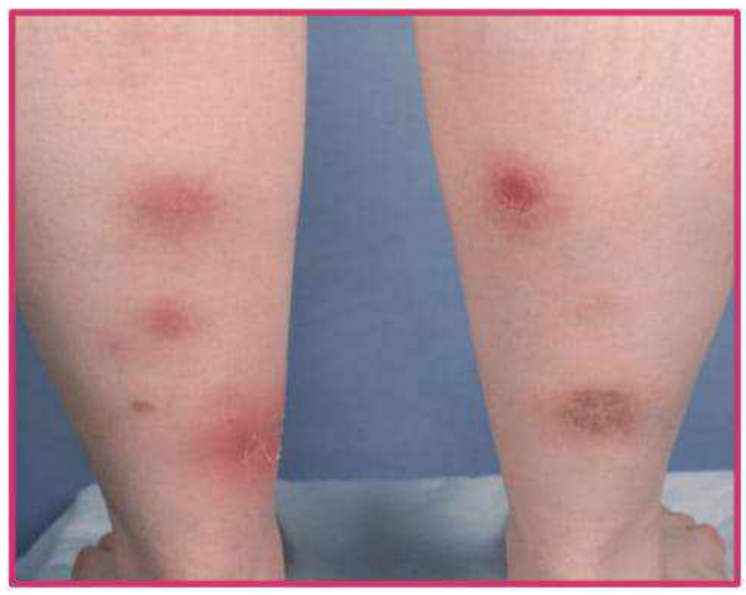
Papulonecrotic Tuberculid

- papular
- present on limbs / extremities
- symmetrical papules which undergo central necrosis and crusting.
- Heal with Scarring.
- On HPE : Obliterative vasculitis and Thrombosis



active space

Erythema Induratum of Bazin



- Nodular Tuberculid.
- Nodular vasculitis ↘

  Not caused by M.TB.
- Presents as :
  - in Middle age females
  - site : Calves / posterior aspect of legs
  - Nodules ⟶ Breakdown.
  - Ulcers ⟶ Scarring.
- On HPE : vasculitis and panniculitis.

Other Nodular Tuberculids :

1. Erythema nodosum :

- site : Shins (Anterior aspect of legs)
- Never ulcerates
- Never Scars.

2. Nodular vasculitis

3. Lupus Miliaris Disseminata Facei.

MANAGEMENT

44:00

- **ATT**

2 months of Intensive phase

- 2 (HRZE)

4 months of Continuous

- 4 (HRE)

Dose	
• Isoniazid (H)	- 5
• Rifampicin (R)	- 10
• Ethambutol (E)	- 15
• Streptomycin (S)	- 15
• Pyrazinamide (Z)	- 25

Atypical Mycobacterial Infections

1. Swimming Pool granuloma :

- Caused by **M.marinum**
- seen in individuals handling **fish tanks**, **contaminated water**
- Nodule ⟶ Ulcerates.

Treatment : Clarithromycin + Ethambutol.

2. Buruli ulcer :

- caused by **M.ulcerans**

Treatment : Rifampicin + Streptomycin.

active space

Leprosy (Part - 1)

- chronic granulomatous disease caused by M.leprae

Cardinal Signs :

1. A skin lesion associated with loss of sensation / an area of loss of sensation which is supplied by peripheral nerve affected by leprosy.
2. Thickening / deficit in a peripheral nerve affected by leprosy
3. Demonstration of bacilli

History

- Bacilli was identified by Dr. Hansen
- a.k.a Hansen's disease / Kushth Rog.

Transmission

- Routes : i) Droplet → most important
  - ii) Skin to skin contact
  - iii) Transplacental
  - iv) Conjugal
  - v) G.I

- I.P - 3 - 5yrs (upto 10yrs)
- Reservoir : Man (only known reservoir)

Bacteriology

- caused by Mycobacterium leprae.
- Obligate intracellular bacilli → Acid fast

- Appear as Solid rods with curved ends
- Acid fast : due to presence of mycolic acid.
- when compared with MTB → less acid fast ($5\%\ H_2SO_4$)
  ($20\%\ H_2SO_4$)
- Tropism for Schwann cells (Nerves)
- Generation Time/ Doubling Time : 11 - 13 days
- Culture : Not cultivable
  → on Mouse foot pad , Nine banded armadillo.
- New Species : M. lepromatosis.

DETECTION OF BACTERIA 10:10

- detected by

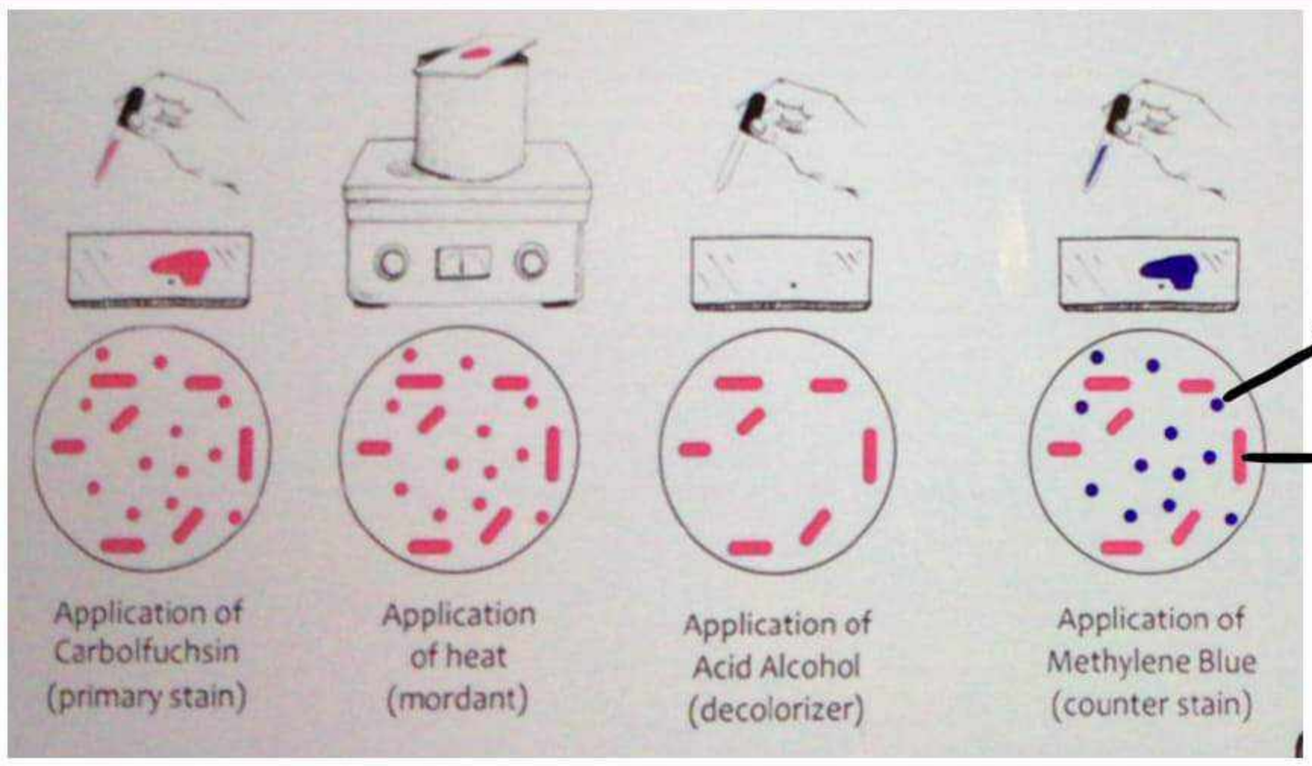
i) Slit Skin Smear ii) Culture iii) Serology

active space

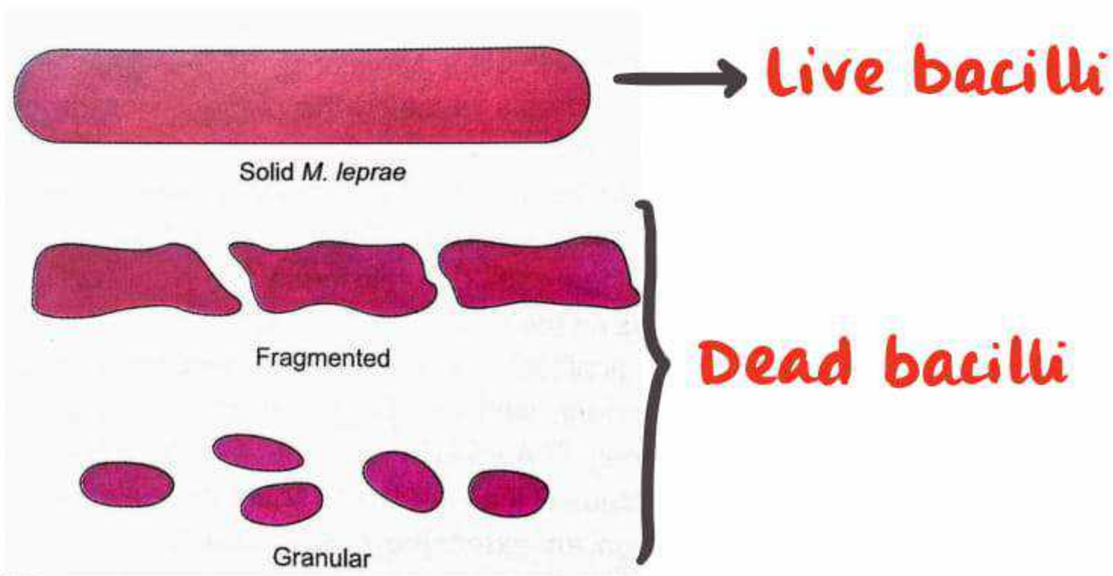
Slit Skin Smear :

- Procedure :

- We make slit from blanched area of skin after pinching skin between thumb and index finger.
- 15 No. Blade
- 5mm long
- 3mm deep



Morphology of Bacilli



- **Site :** Taken from 2 sites.

  Preferred site :

  i) Ear lobe

  ii) Active lesion → from periphery of lesion

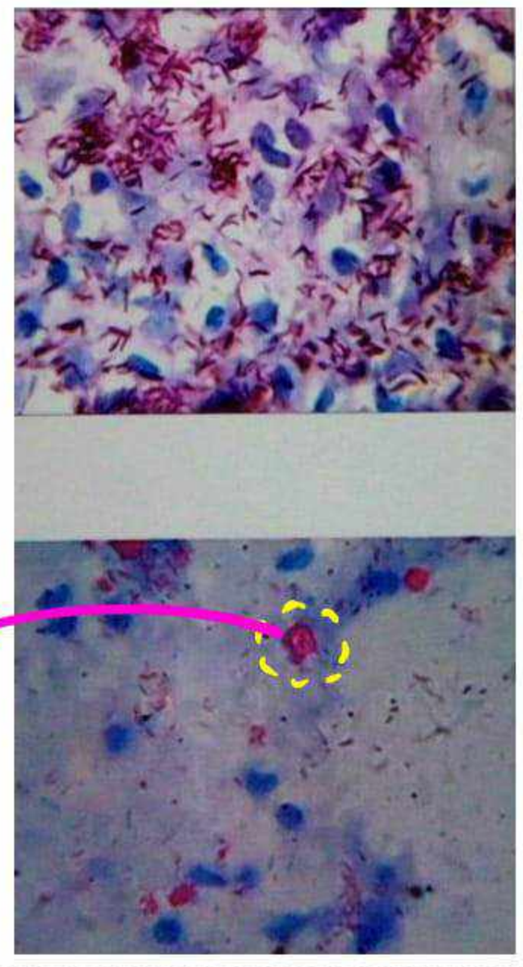
- **Density of bacilli in tissue :** 10^4 bacilli/gm of tissue

Bacteriological Index :

- density of bacilli / HPF
- no. of dead and living bacilli / HPF

6 Grading

1. → 1-10 Bacilli/100 HPF
2. → 1-10 Bacilli/10 HPF
3. → 1-10 Bacilli/HPF



4. → 10-100 Bacilli/HPF

5. → 100-1000 Bacilli/HPF

6. → >1000 Bacilli/HPF, globi ⊕

Morphological Index :

- No. of solid staining bacilli (**live bacilli**) counted after examining **200 Red stained elements** lying singly
- Helps as guide in Treatment
- Globi → clumps of bacteria.

Serology :

i) Phosphoglycolipid - I → Virulence factor

Tuberculoid ⟶ Lepromatous pole
Increase

Lepromin Test : measures CMI

- Obselete (Not for Diagnosis anymore)
- used in past for Diagnosis
- Immunological Response to M.leprae
  - ↳ Inject I/D into forearm
    - Early Reaction of Fernandez
    - Late Reaction of Mitsuda.

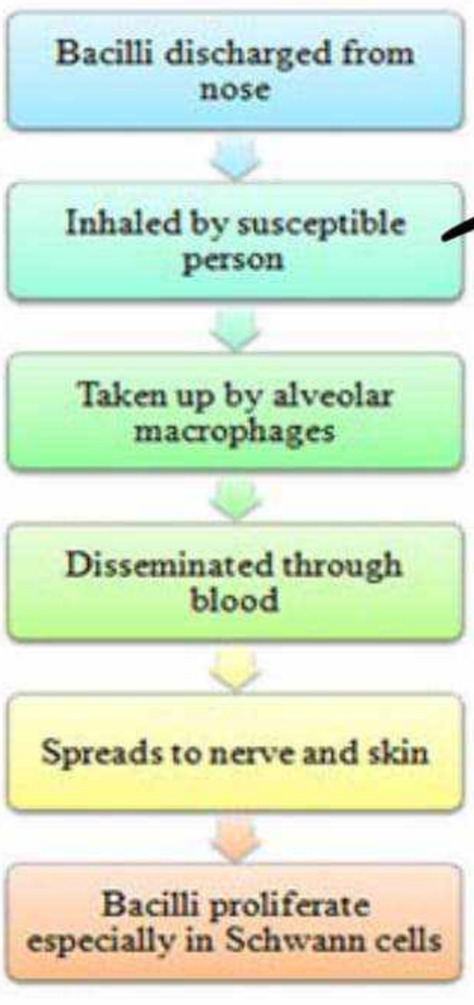
active space

- **Early Reaction of Fernandez :**
  - occurs in 24-48 hrs
  - stays for 2-3 days.
- **Late Reaction of Mitsuda :**
  - occurs in 1-2 wks
  - stays for 4 wks

Lepromin Test

- Positive → Tuberculoid
- Negative → Lepromatous.

Immunopathogenesis :



active space

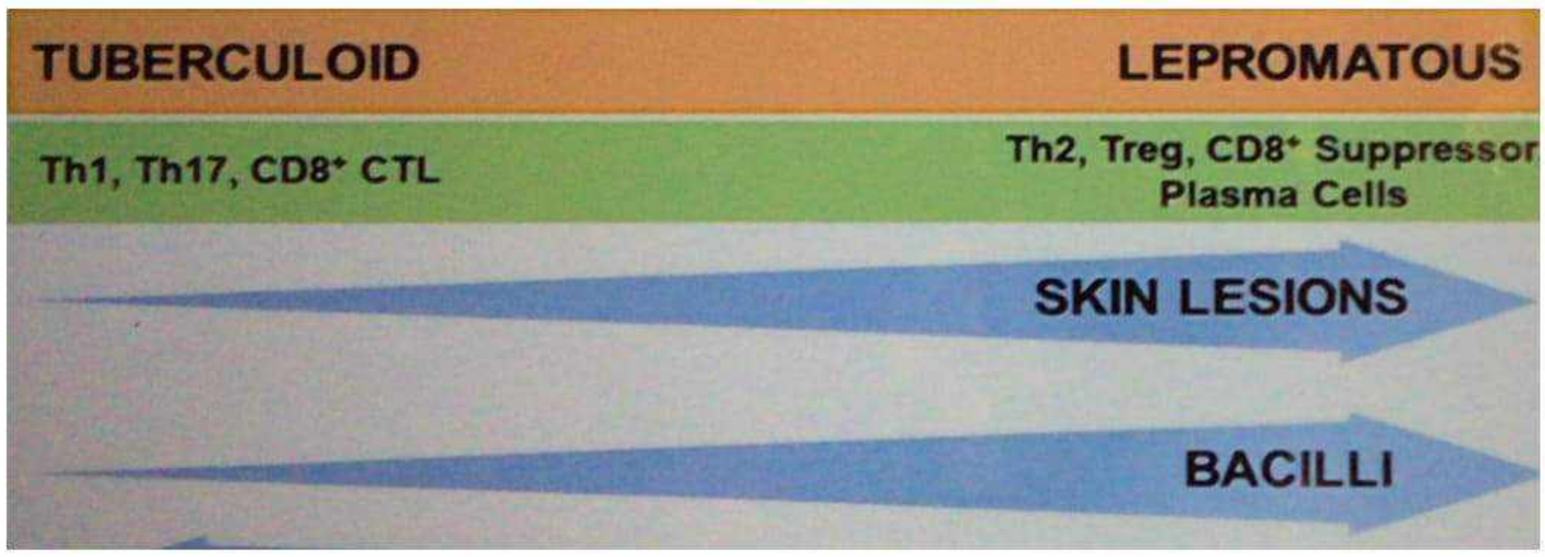
Immunology :

- Cell Mediated Immunity (CMI)

T_H1 Response → IFN γ, TNF-α → protective for Body → Tuberculoid pole

T_H2 Response → IL-4, IL-10 → protective for bacilli → Lepromatous pole.

- Innate immunity



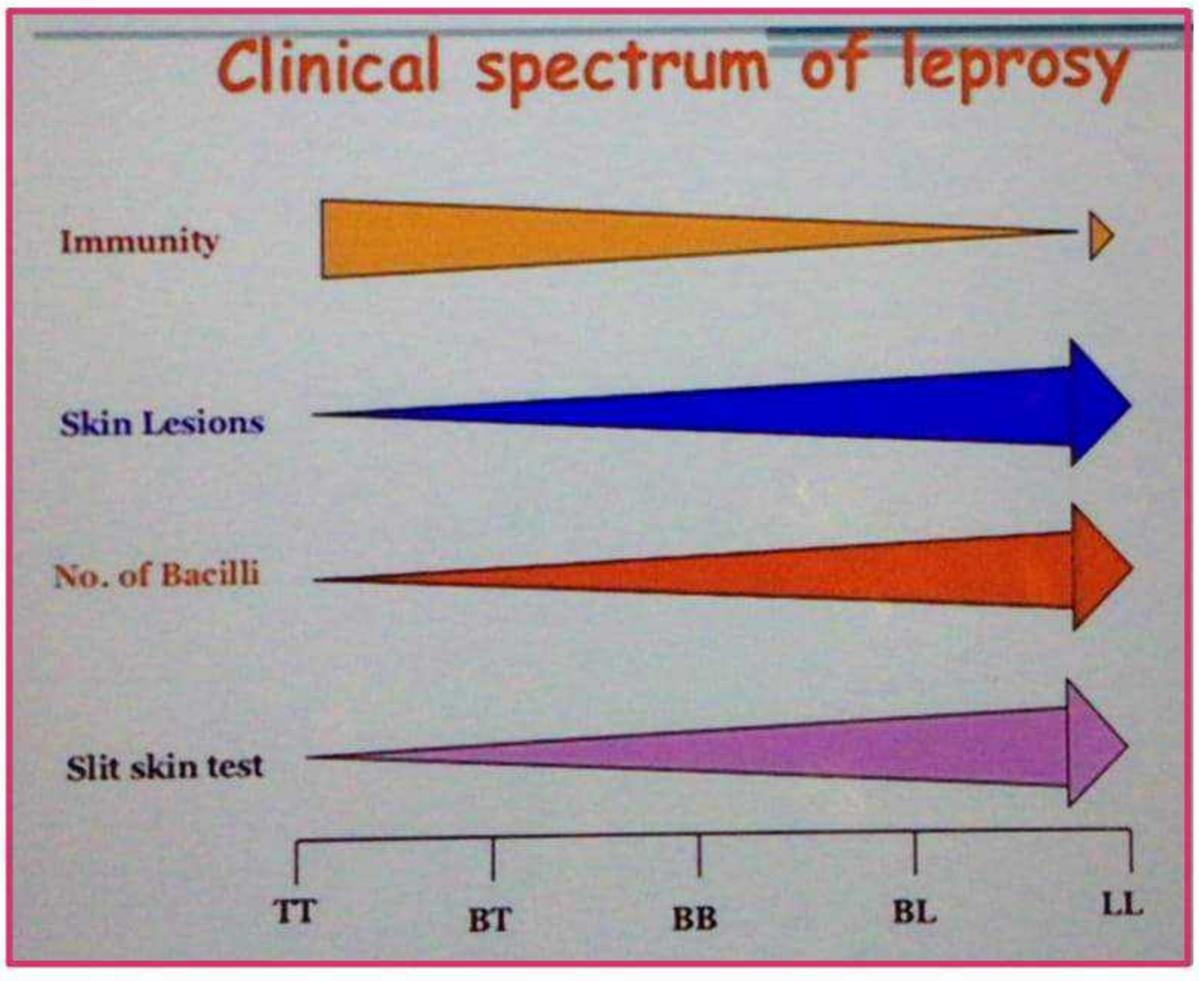
Cell Mediated Immunity ←

Antibodies →

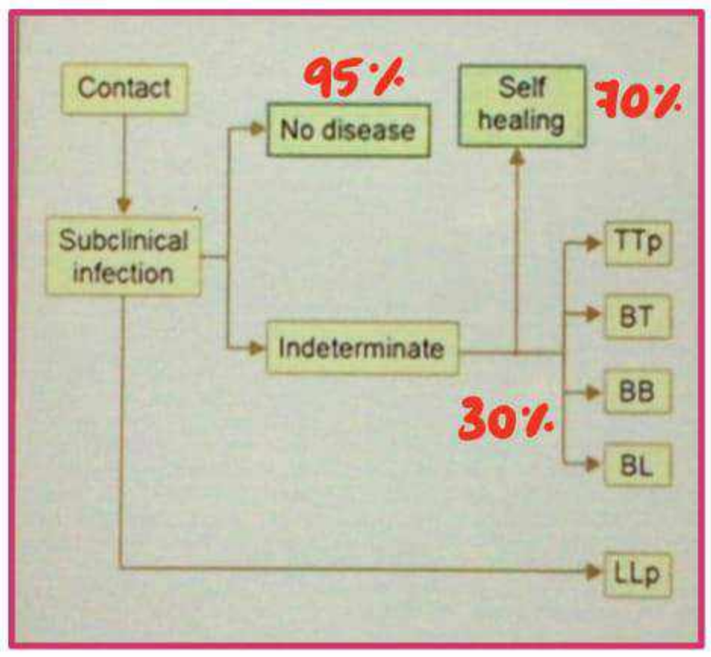
Nerve Damage. ↔

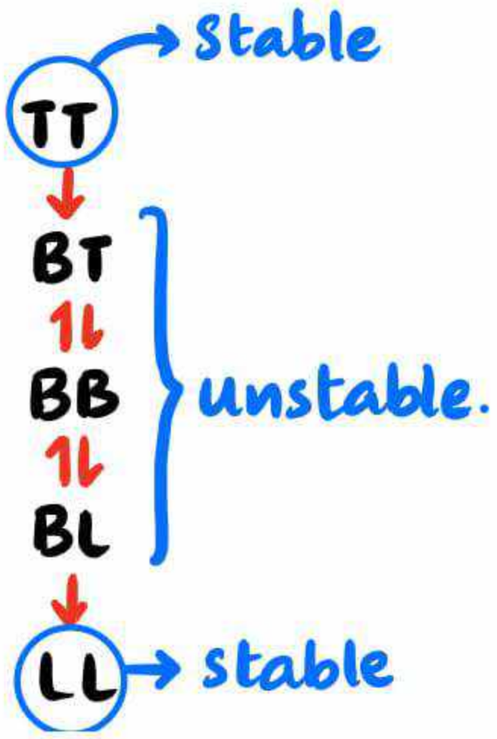
↑CMI → perineural thickening

due to ↑ Bacillary load → active space



- TT → Tuberculoid → very good immune response
- BT → Borderline Tuberculoid → fair immune response
- BB → Borderline Borderline → Borderline immune response
- BL → Borderline Lepromatous → Low immune response
- LL → Lepromatous → very low immune response





active space

Leprosy (Part - 2)

CLASSIFICATION OF LEPROSY

00:50

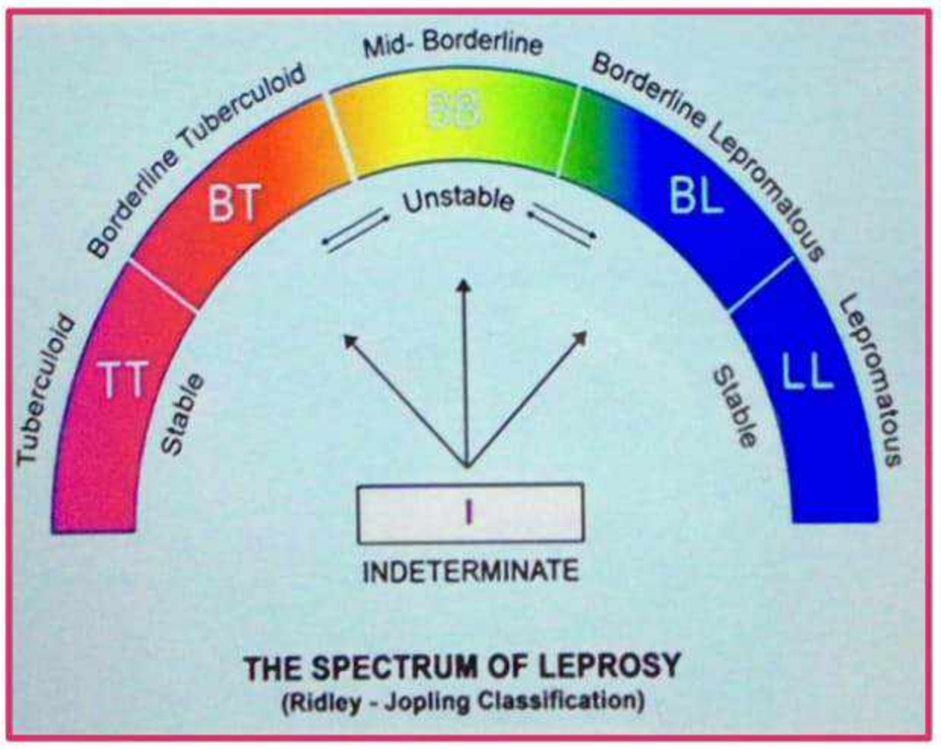
Indian Classification

→ 5 Classes :

- Tuberculoid
- Borderline
- Lepromatous
- Indeterminate
- Pure neuritic

Ridley Jopling Classification

- Based on :
  - i) Clinical features
  - ii) Histopathology
  - iii) Bacteriological
  - iv) Immunological
- Most scientific classification
- doesnot include Indeterminate or Pure neuritic Hansen



	Tuberculoid	Lepromatous
Clinical features:		
Skin lesions : Number	→	↑
Margins	Well defined	Ill defined
Glands	Dry ↑ Anesthesia on lesion	Shiny Glove and stocking anesthesia
Nerve involvement :	Less in No. but more severe damage	More in No. but Late destruction
Bacterial load	→	↑
Lepromin Test	⊕	⊖

WHO Classification

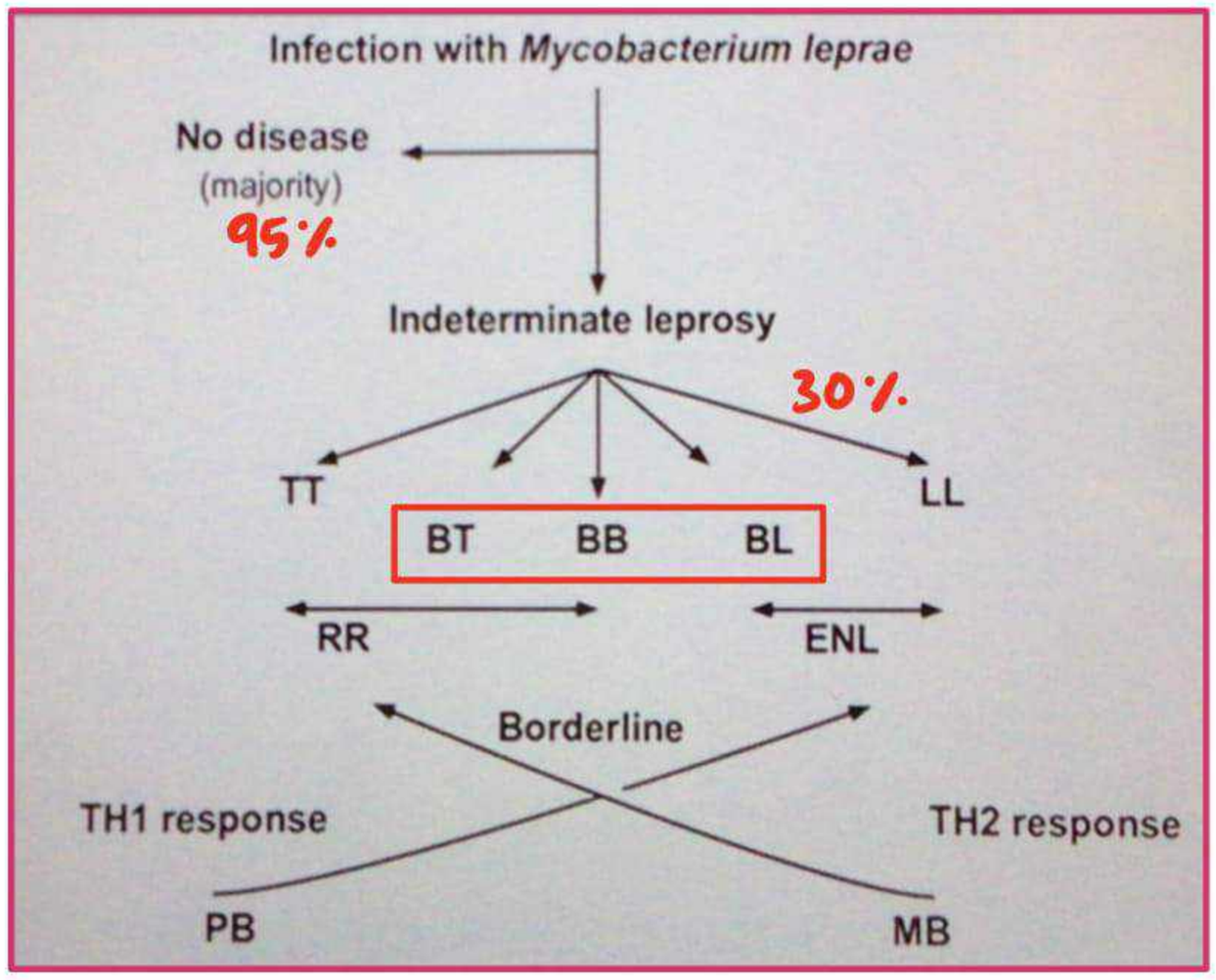
- Practical for Treatment

	MB(Multibacillary)	PB (Paucibacillary)
No. of skin lesions	>5	1-5
Nerve involvement	≥1	0
Presence of AFB	+	-

active space

NLEP CLASSIFICATION

	MB (Multibacillary)	PB (Paucibacillary)
No. of skin lesions	>5	1-5
Nerve involvement	>1	1
Presence of AFB	+	-



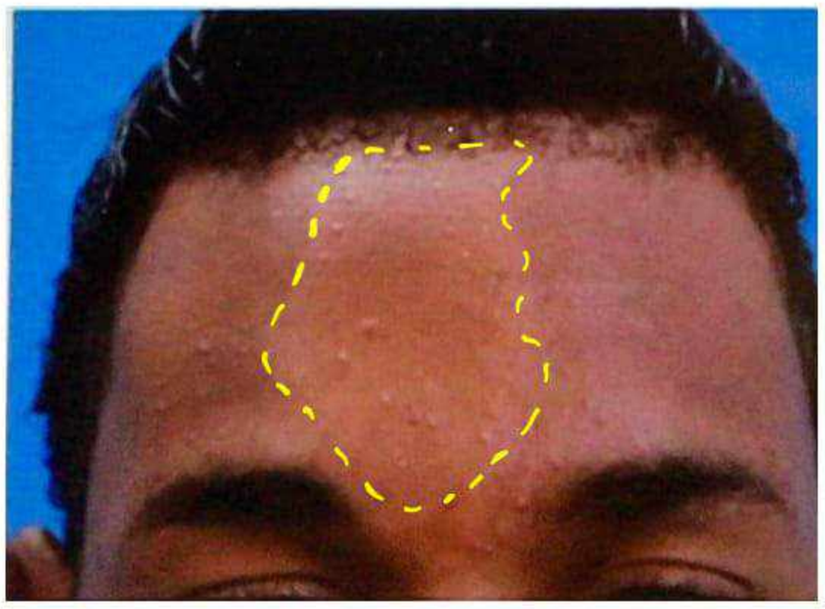
INDETERMINATE LEPROSY

14:30

- (MC) form of leprosy in India
- Endemic to India (Jharkhand, Bihar, UP)
- Site : Exposed - Limbs, buttocks (Africans - Face)

active space

- Single macule
  - → with ill defined borders
  - → no loss of sensation
  - → no infiltration
- Nerve involvement : ⊖



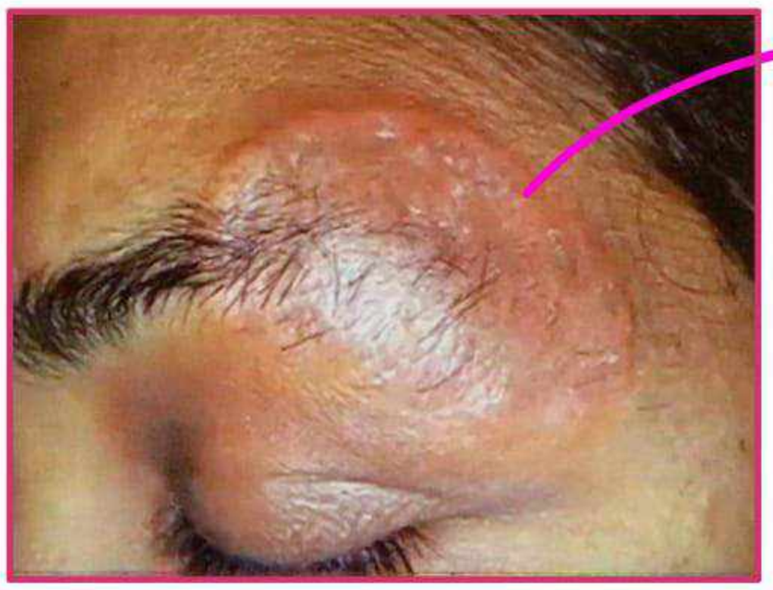
- Lepromin test : Variable
- Slit Skin Smear : ⊖
- Histopathology : Periappendageal / Perineural infiltrate
- Course : → 30% → Determinate form
  → 70% → Heal
- D/D : i) Pityriasis alba - Hypopigmented lesion of face of child.
  - scaling, multiple
  - On HPE → Spongiosis

TT LEPROSY 20:10

- High/good immunity, stable.
- Site : Any site
- Number : few ; 1-10
- Size : < 10cm

active space

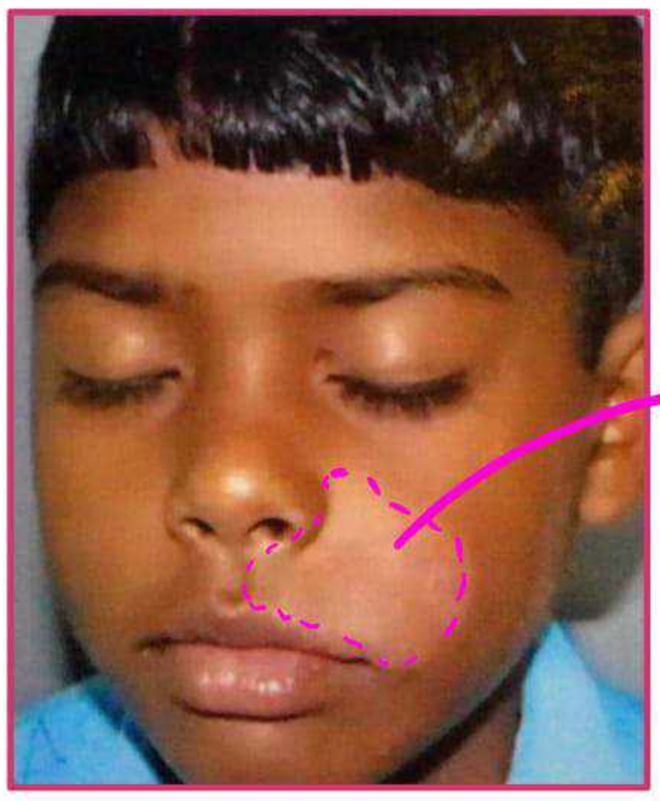
- **Morphology :**
  - Well defined hypopigmented / erythematous plaques.
  - Completely anesthetic
  - Dry, scaly
  - Saucer, Right way up



Raised well defined margins

Central clearing

complete loss of hair.



Well defined dry scaly lesions.

- **Sensation complete loss**
- **Hair lost**

active space

- Autonomic damage - ⊕, loss of sweating
- Nerve - Nerve to patch
- Slit-Skin smear - Negative
- Lepromin Test - Positive

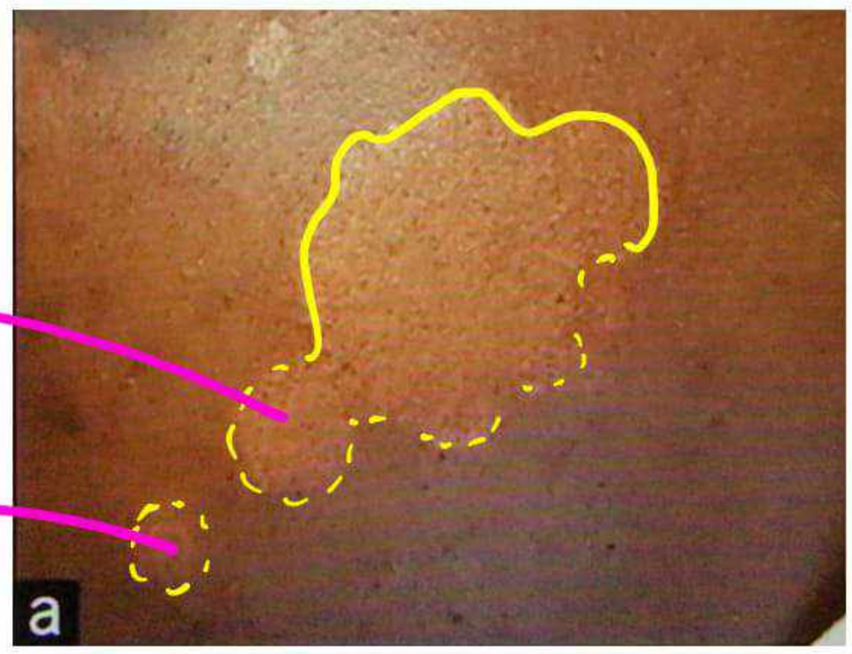
BORDERLINE TUBERCULOID

26:00

- No. of lesions : 10-20
- Size : 10-20cm
- Lesion : well to ill defined margins

plaques which are hypopigmented or erythema

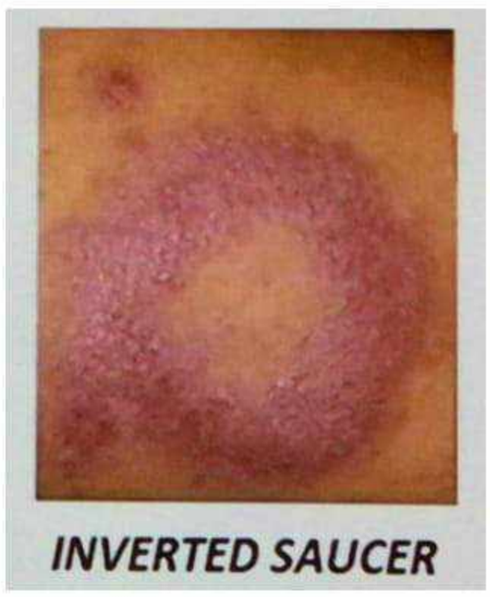
- Nerve involvement : + + + ⟶ Tenderness, Thickening
- Anesthesia would not be complete - Hypoesthesia.
- partial loss of hair
- partial loss of sweating

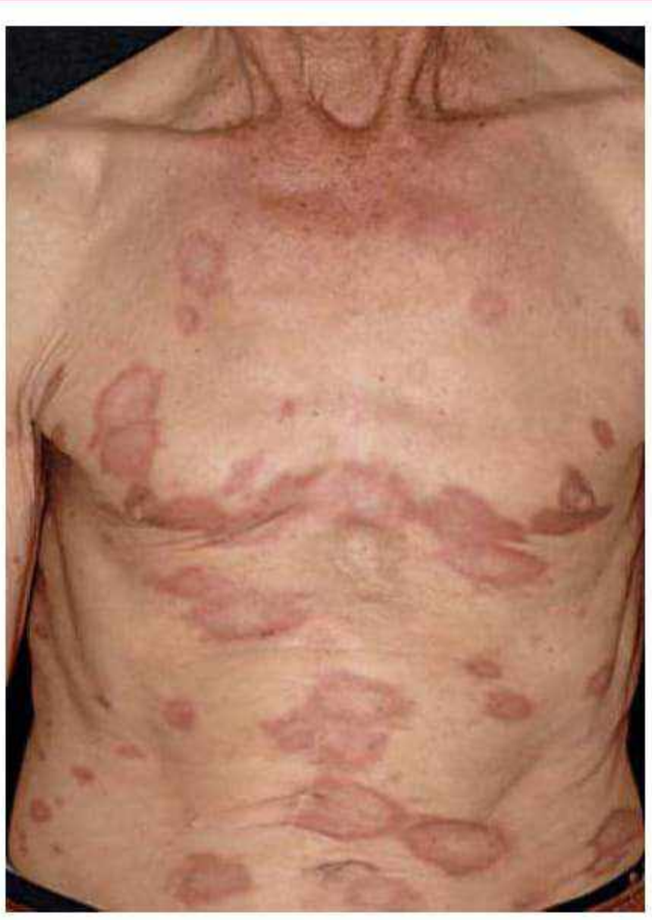


BORDERLINE LEPROMATOUS

30:15

- Numerous lesions → not entirely symmetrical, small lesions
- Hypopigmented / erythematous macules, papules or nodules.
- Ill defined.
- More widespread nerve involvement → but not symmetrical.
- Glove and Stocking anesthesia. → Hypothesia occuring on extremities (M.leprae → cooler area of body)





active space

- **Other lesions**

  i) Annular lesions

  ii) Punched out / Swiss cheese appearance. } More characteristic of BB

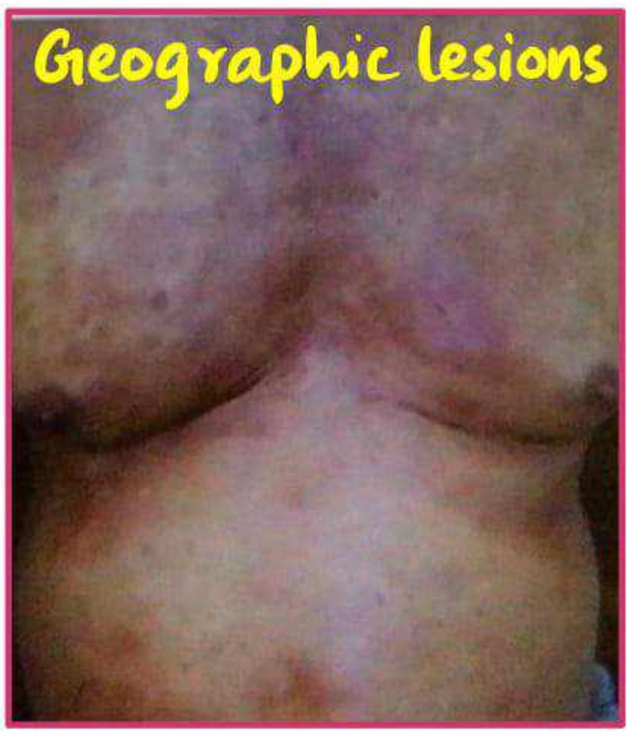
BORDERLINE BORDERLINE 37:15

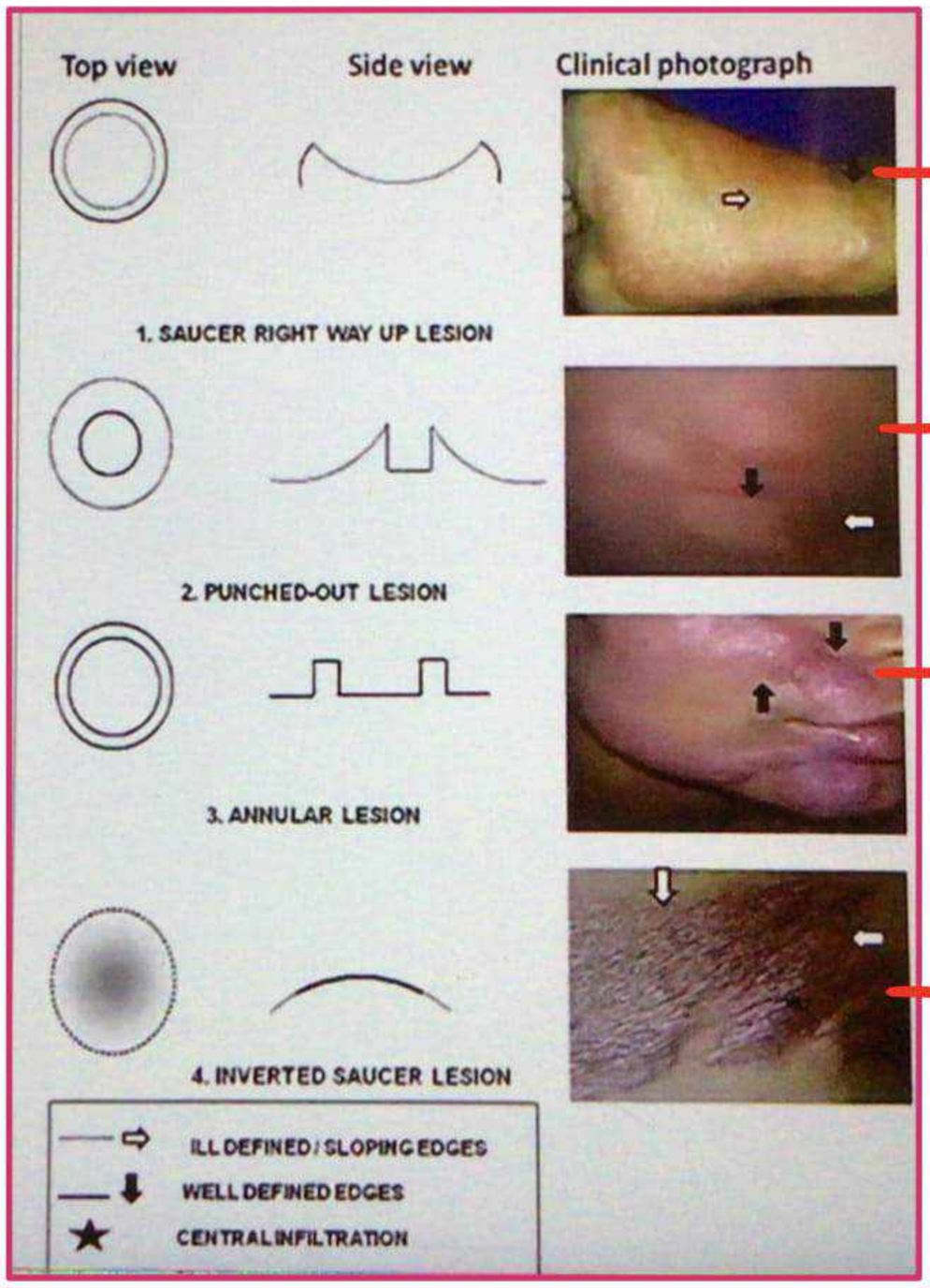
- most unstable form.
- Rarest
- BT → (MC) Determinate Type of Hansen.
- **Lesions :**

  i) Punched out lesions

  ii) Geographic lesions

  iii) Annular lesions





→ TT

→ BB > BL

→ Borderline BL, BB

→ BL

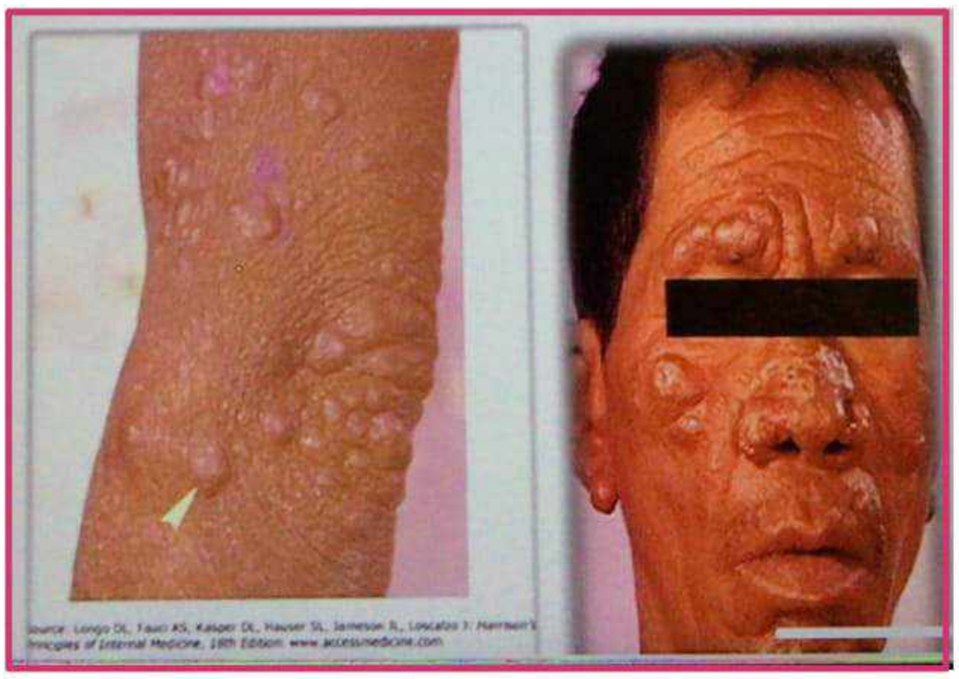
COURSE OF BORDERLINE 41:00

- BT → High chances of Type I Reaction
- BB → Unstable, Rarest
- BL → Maximum damage.

LEPROMATOUS LEPROSY 41:45

- Earliest symptoms : Nasal symptoms, pedal edema
  ↓
  Stuffiness, crusting, discharge

- **Site :** Any areas, even the warmer areas
- **Number :** Numerous
- **Size :** small
- **Symmetry :** Symmetrical (symmetrical nerve involvement also)
- **Morphology :**
  - macules ⟶ Body
  - Nodules ⟶ face
  - Plaques
  - Glove and Stocking anesthesia
  - lesions are shiny and ill defined
- Infiltrated skin ⊕
- can involve other organs as well
- **Surface :** Shiny
- **On Slit Skin Smear -** ⊕
- **Lepromin Test -** ⊖



Facial deformities seen are :

1. **Madarosis :** loss of eyelashes / eyebrows
2. **Sagging face :** destruction of collagen

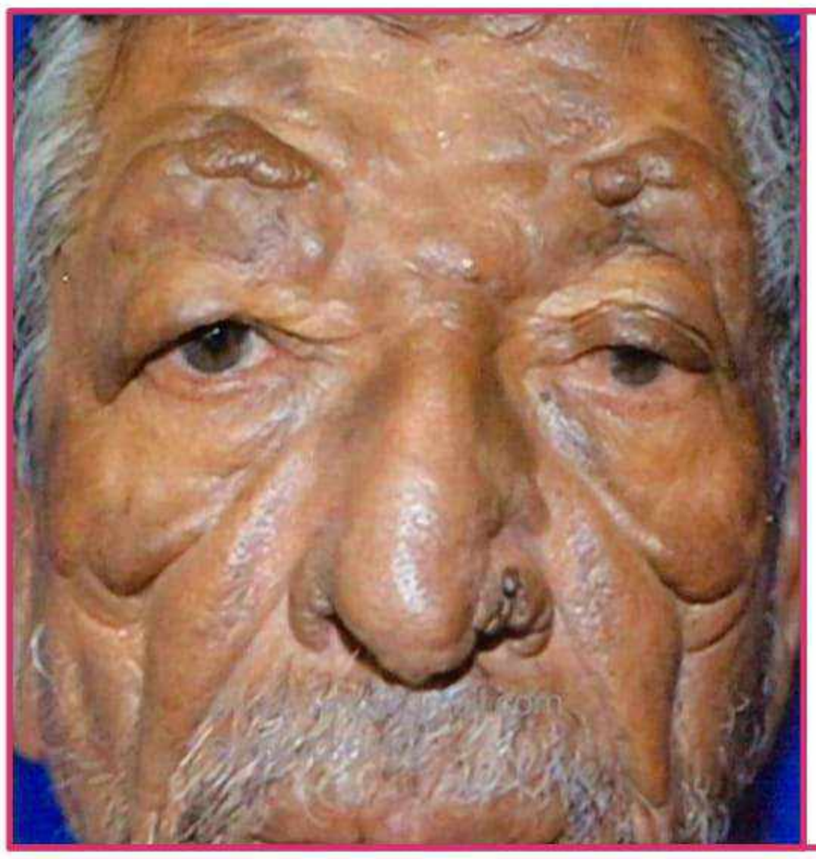
active space

3. Buddha ears : Sagging of ears

4. Rat Bitten ears } Chondritis
5. Collapsed pinna }

6. Saddle nose deformities : Anterior Nasal spine collapses
nasal septal perforation.

7. Loss of upper incisor teeth



LEONINE FACIES / LION LIKE FACIES

- Seen in
  - i) LL leprosy
  - ii) Lichen myxedematous
  - iii) CTCL
  - iv) CAD

Ocular Involvement :

i) Lagophthalmos → of lower eyelid. (Facial Nr involvement)

ii) Corneal hypoanesthesia → Trigeminal Nr

iii) Superficial punctate keratitis  → Torch, grains of chalk
↓
group of lepra bacilli (Milaria lepromata)

active space

iv) Corneal ulceration

v) Iritis, scleritis

vi) Iris pearls ⟶ On Slit lamp : Collection of lepra bacilli in Swollen macrophages

- (MC) cause of Blindness in Leprosy cataract (Age-related, steroids, chronic uveitis)

Other Systems involved : LL

- Nails
- Nose
- Bone
- Mouth, Pharynx and Larynx
- Eyes
- Testes
- Muscle
- Lymph nodes
- Kidney
- RES, Adrenals, Hematology

Sanctuary / Safe Sites : Lepra bacilli doesnot involve -

- Warmer areas ⟶ Axilla, groins, Scalp (Hair bearing areas)
- Leprous alopecia
- CNS
- Posterior chamber of eye
- Lower Respiratory tract
- Female Reproductive Tract

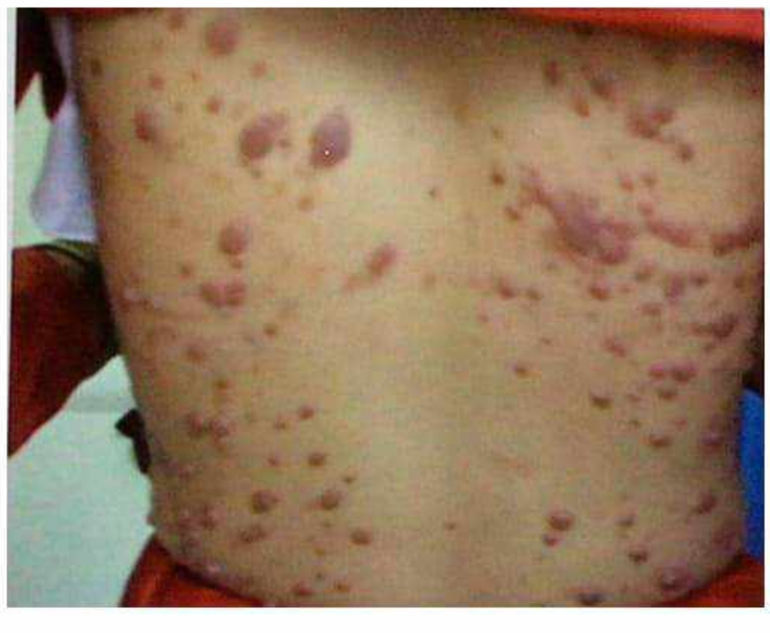
active space

Leprosy (Part - 3)

HISTOID LEPROSY

00:20

- unusual presentation of LL
- Identified by Wade
- Well defined papules and nodules on clinically normal skin.
- Focal loss of immunity
- Dapsone monotherapy
- On Slit Skin Smear :
  - High BI/MI
  - on lesions rods are longer than usual.
- On HPE : Spindle shaped cells



Lucio leprosy :

- a.k.a Lepra Bonita (Beautiful leprosy)
- diffuse infiltration with sensory involvement
- No skin lesions
- No motor involvement
- No eye involvement

active space

Lazarine leprosy

- undernourished
- ulceration over leprosy lesion ⊕

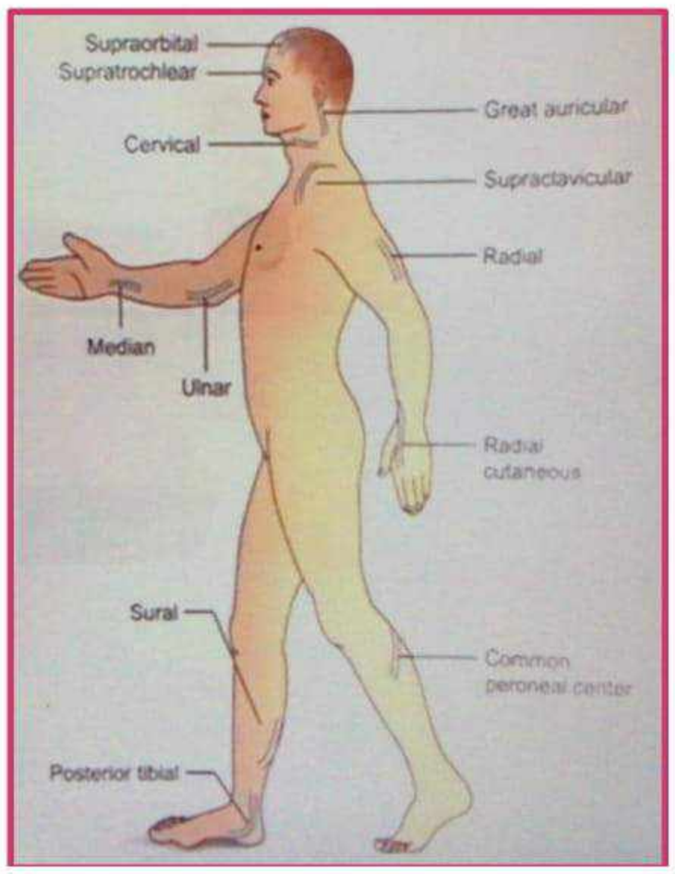
PURE NEURITIC HANSEN 04:45

- No skin lesions
- Area of loss of sensation in an area of skin typically supplied by peripheral nerve which is typically involved in leprosy
- Nerve tenderness / Neural deficit
- Slit Skin Smear - Negative
- Absence of any other causes of nerve involvement.
- Endemic to India
- Nerve Biopsy :
  - from purely sensory nerve
  - preferred nerves are :
    i) Radial cutaneous Nerve
    ii) Sural Nerve.

Nerve Involvement in leprosy:

- universal
- 3 Types :

SPECIFIC	PARALYTIC	ANESTHETIC
• due to infiltration of leprosy	• Motor deficit ⊕	• Sensory deficit ⊕ • (MC) sensory impairment ↓ loss of Temperature. ↓ f/b fine touch ↓ f/b pain. • Deep touch, vibration and proprioception not affected.



Better seen than palpated

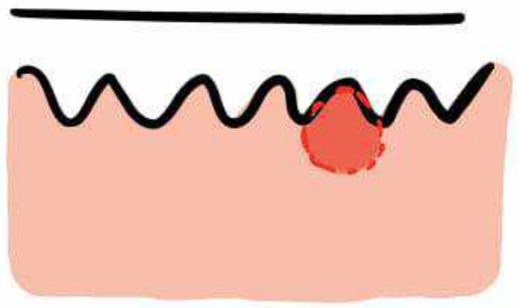
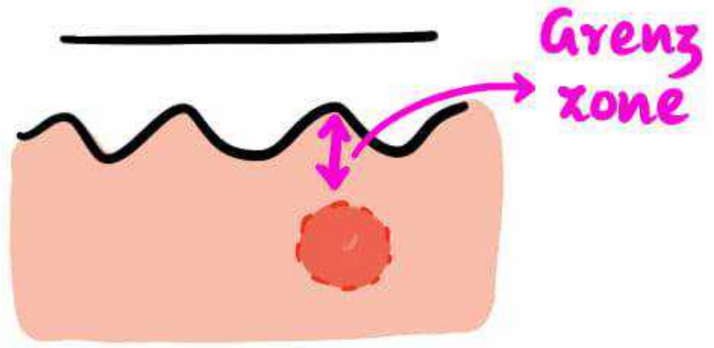
↓

Greater auricular nerve

- (MC) Cranial nerve involved : Facial > Trigeminal Nr.
- (MC) peripheral nerve involved : Ulnar Nr.
- Type of Palsy - Ulnar (High Ulnar), Median (Low median)

On Histopathology :

- Biopsy is taken from : most active, new lesion, smaller lesion.
- Fite Farco stain to demonstrate AFB in tissue specimen
- Interpretation :

	Tuberculoid	Lepromatous
Granuloma formation	• well formed compact • ↑ Epitheloid • ↓ Macrophage • Erodes epidermis 	• Grenz zone/ Clear zone seen 
Nerves		Perineural thickening Onion skin appearance 

active space

	Granuloma	T-Lympho	Epitheloid cells	Giant cells
TT	compact Epitheloid	↑↑ (CD_4)	↑	Langerhan Giant cell
BT	less compact Epitheloid + Macrophages	↑	↑	Foreign Body Giant cell
BB	Mixed	↑	↓	-
BL	Macrophage Granuloma	↑↑(CD_8)	↓	-
LL	Macrophage.	↓	-	-

	Macrophages	Grenz Zone	Perineural lamination
TT	-	-	-
BT	-	-	-
BB	+/-	+	-
BL	+	+	+ +
LL	+	+ +	+ +

active space

REACTIONS

19:35

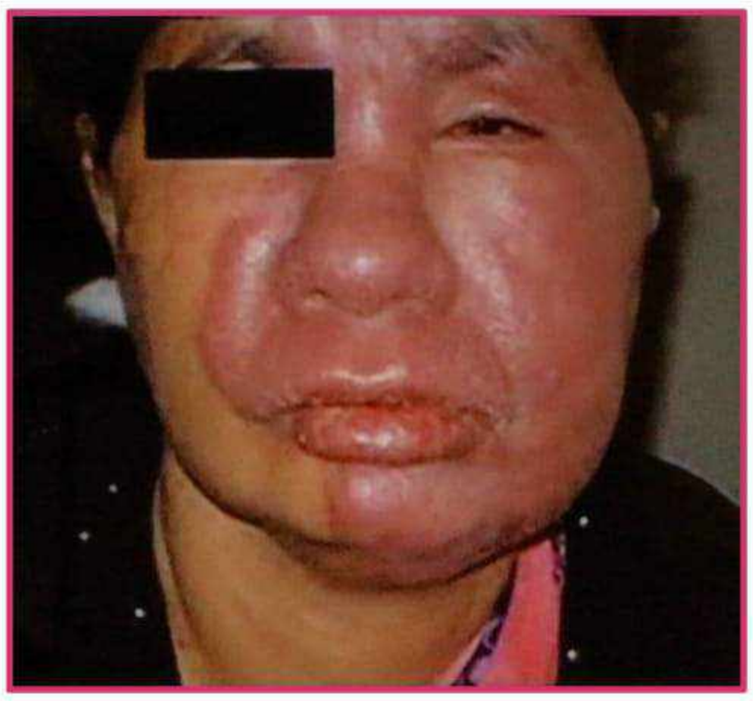
- Type I Reaction
  - Upgrading or Reversal Reaction
  - Downgrading
- Type II Reaction (ENL)

BT → BB → BL

- TT ← BT

Type I Reaction :

- usually seen in upper part of Spectrum.
- Type IV HSR
- usually occur after treatment (2wks - 6 months of starting MDT)
- Existing lesions become erythematous, edematous, tender
- New lesions present.
- Nerve involvement ⊕
- Nerve tenderness ⊕
- New nerve involvement in the form of deficit.
- Nerve abscess ⊕



active space

Treatment :

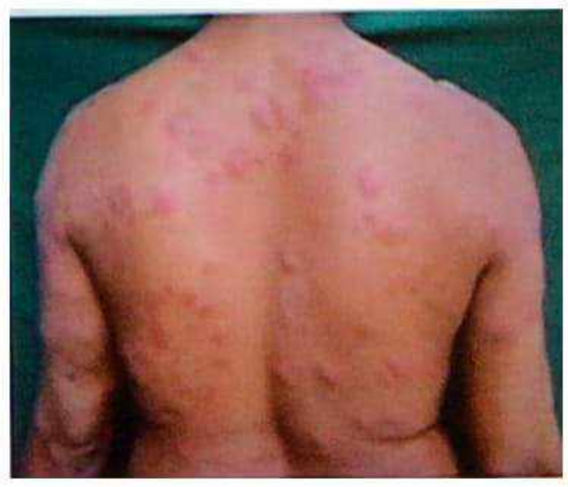
- Continue MDT
- In Mild Reaction → NSAID's, Aspirin
- In Moderate to Severe (Neuritis) → Steroids ⇒ DOC
  ↓
  Oral Prednisolone 40mg/day
- Nerve abscess → 1st → Steroids
  → 2nd → Drain
- Splints

Other drugs used :

- Azathioprine
- Cyclosporin
- Methotrexate.

Type II Reaction

- Erythema Nodosum Leprosum
- Type III HSR
- seen in Lower end of spectrum (Lepromatous end)
- Not Related to MDT



active space

- Other Trigger Factors :

  i) Pregnancy

  ii) Infection

  iii) ↑ B.I load

  iv) Immunocompromised State.

- No change in existing lesion
- New Crops of lesions → disappear in 24-48 hrs.
- Crops of Tender, evanescent papules and nodules all of body
- Associated with evening rise of temperature
- Lots of constitutional symptoms → Myalgia, Malaise → More sick
- Systemic involvement ⊕

leaves behind PIH/ Bluish marks.

Management :

- Mild → NSAIDS
- Moderate to Severe → Steroids (DOC)
- Others : Thalidomide, Azathioprine, Methotrexate, Cyclosporin, Clofazamine, Minocycline.

active space

Systemic involvement in Type 2 Reaction

Organs involved	Signs and Symptoms
Joints	Polyarthritis or polyarthralgia
Lymph nodes	Tender generalized lymphadenopathy (especially femoral)
Eyes	Uveitis (iritis and iridocyclitis), glaucoma, Blindness
Organomegaly	Hepatosplenomegaly - may be tender
Genitalia	Orchitis and Epididymitis
Kidneys	Glomerulonephritis, Acute Tubulointerstitial nephritis and amyloidosis which cause chronic kidney disease
Bone	Dactylitis, Periosteitis
Muscles	Myalgia, Myositis
Nerves	Neuritis.

	TYPE 1 Reaction	TYPE 2 Reaction
Spectrum	Tuberculoid	Lepromatous
Type of Reaction	IV	II
Morphology	existing lesions	Crops of new lesions
Nerves	+++	Less common
Constitutional Symptoms	Mild	↑↑↑
Other organ involvement	-	+

Thalidomide

- used for Type 2 Reaction
- S/E to steroids, intolerant to steroids
- Dose : 100-400mg/day (start with 400mg ⟶ Taper to 100mg)
- Effect can be seen in 2-3 days.
- MOA : Anti-TNF α
- S/E : i) Teratogenic
  - ii) Constipation
  - iii) Sedation
  - iv) Neuropathy ⟶ Monitored using SNAP.

active space

LUCIO PHENOMENON 35:25

- There is vasculitis and Thrombus formation
- Bacilli invades endothelium

Clinical feature:

Redness in the centre of existing lesion
↓
Bluish - purplish hue.
↓
Necrotic
↓
Ulcerate
↓
Eschar

- ulceration with eschar formation on existing lesions

TREATMENT OF LEPROSY 36:50

- MDT - Multi Drug Therapy

i) Rifampicin
ii) Clofazamine
iii) Dapsone

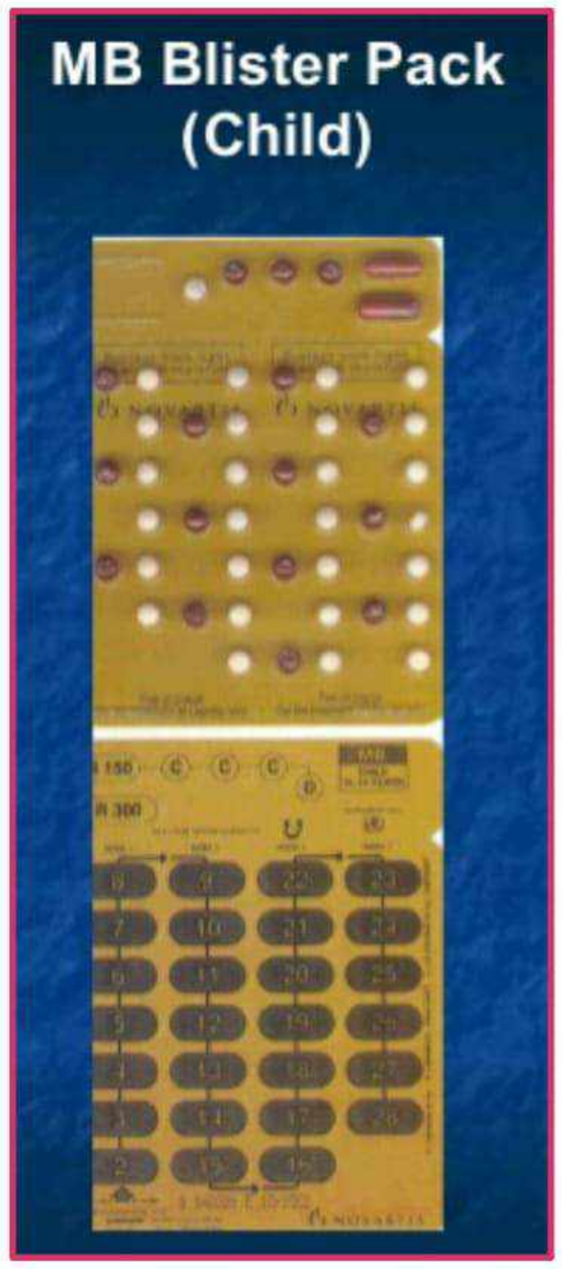
} given as MDT to prevent resistance.

- Rifampicin - kills 99% of viable bacilli are killed with 1st dose

active space

- In MB Leprosy → MDT given for 12 months
- In PB Leprosy → MDT given for 6 months

For Adult (Blister pack)



- Rifampicin → 600mg once a month single supervised dose
  450mg
  (child)
- Clofazamine → 300mg once a month and 50mg daily.
  150mg
  50mg on alternate days
- Dapsone → 100mg daily.
  50mg

active space

Rifampicin

- most potent
- Inhibits RNA Polymerase
- S/E : i) Flu like symptoms
  ii) Hepatotoxicity
  iii) Red urine.

Clofazamine

- acts on membrane → Affects Respiratory chain of bacilli
  ↓
  cell death.
- used also for Reactions / Treatment
- S/E : i) Reddish discolouration of skin → Reversible
  ii) Icthyosis
  iii) Nausea, Vomiting, Abdominal pain.
- Isolated Reports of Resistance are seen

Dapsone

- Inhibits Folic acid synthesis
- Maximum Resistance reported with it.

active space

- **S/E :** i) Hemolytic anemia,

  ii) DDS ⟶ Rash, Hepatomegaly, LAN, Hepatitis

  iii) Neuropathies ⟶ Sensory ⟶ Reversible

  iv) Methhemoglobinemia

NEWER DRUGS

47:50

- for increasing compliance
- for Resistance cases

i) Clarithromycin

ii) Ofloxacin

iii) Minocycline

iv) Moxifloxacin

v) Rifabutin

- In Pregnancy ⟶ same MDT is given. (category C)
- In Concomitant TB ⟶ Stop monthly Rifampicin dose, Rest same as MDT and continue ATT

DEFORMITIES OF LEPROSY

50:10

- Deformity : visual effects of the impairment

i) Specific → Lepromatous infiltration → seen in face

ii) Paralytic → Motor nerves → seen in hands

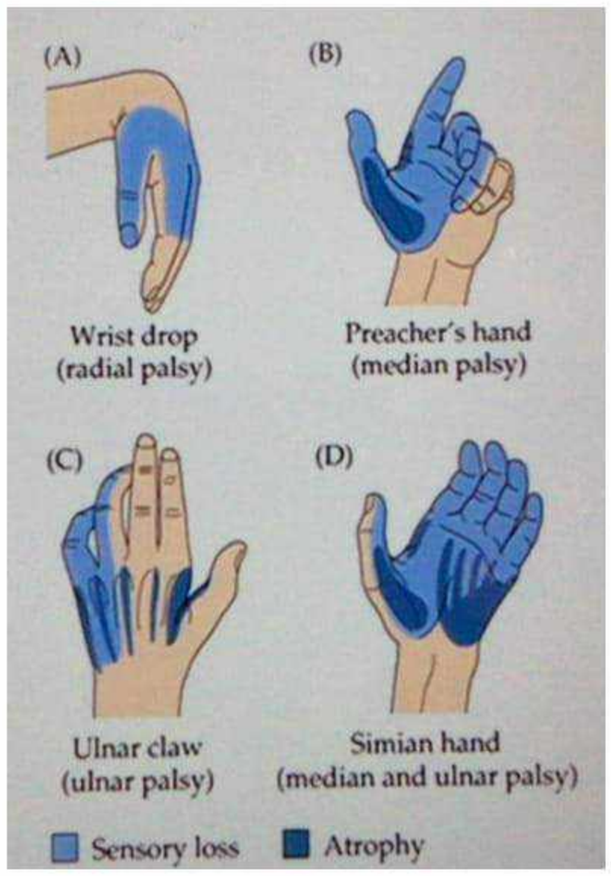
iii) Anesthetic → Sensory nerves. → feet

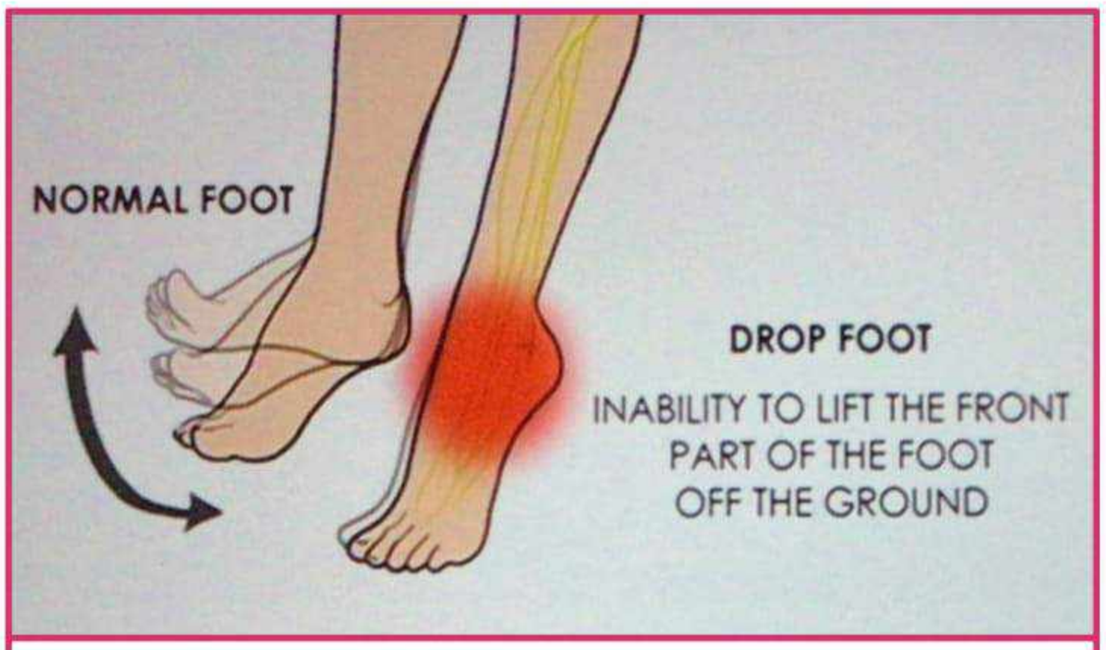
WHO Grading of Deformities

	Hands and feet	Eye
0	No anesthesia No visible deformity	No loss of vision
1	Anesthesia ⊕ No visible deformity	< 6/60 , Finger counting ⊕
2	visible deformity	No finger counting, Lag ophthalmos ⊕

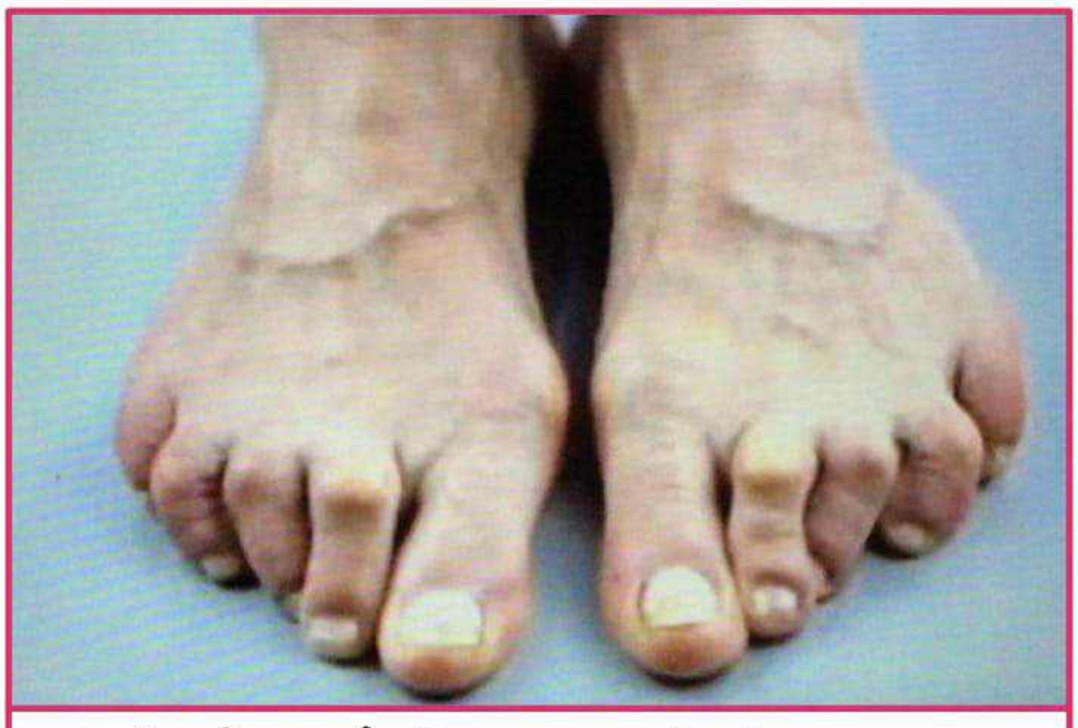
Motor Nerve defects :

- Ulnar nerve → Partial claw Hand
- Median and Ulnar → Total claw hand nerve palsy
- Radial nerve → Wrist drop.



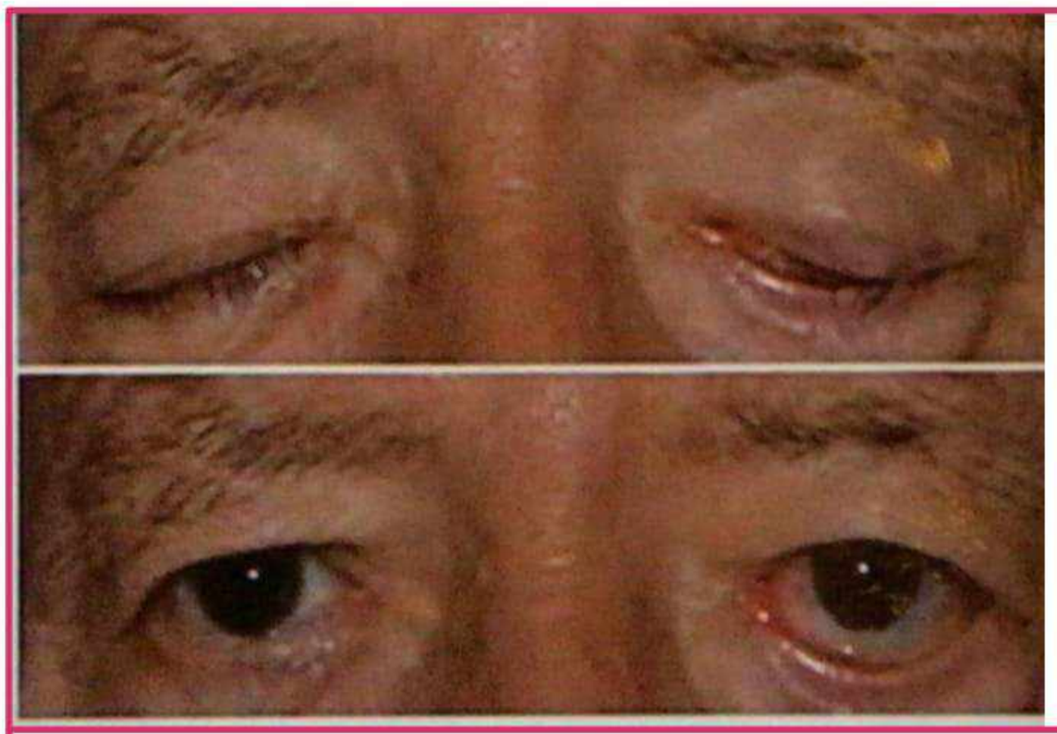


Common Peroneal Nerve affected.



Lateral Peroneal Nerve affected → Claw foot

active space



Facial Nerve affected
Lagophthalmos → Lower eyelid

active space

Sexually Transmitted Diseases (Part - 1)

Sexually Transmitted Infections :

1. Genital ulcer disease
2. Discharges → Urethral / cervical
   → Vaginal
3. Warts

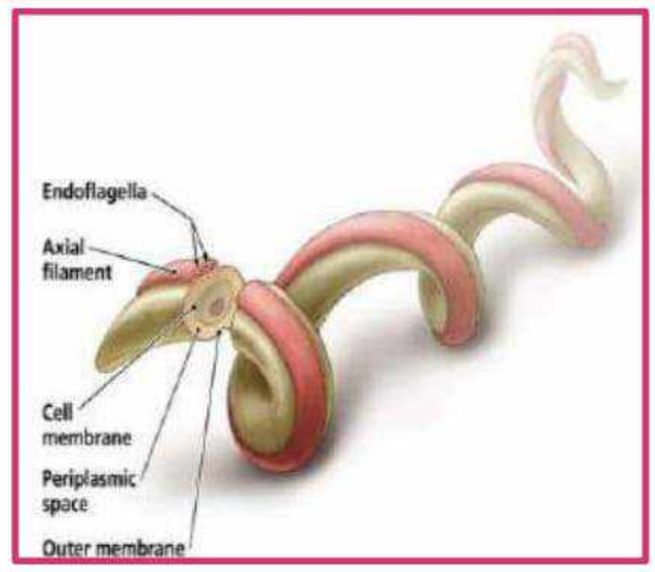
GENITAL ULCER DISEASE 02:35

Caused by : STD causes of GUD.

1. Syphilis
2. Chancroid
3. Herpes genitalis
4. Donovanosis
5. LGV → Lympho Granuloma venerum.

SYPHILIS 04:15

- chronic
- involves all systems of body
- a.k.a Great Imitator



- caused by Treponema pallidum

Treponema pallidum

- family → Spirochetacea
- length → 6-15 µm
- Width → 0.25 µm
- Coils → 8-24 coils
- Non-cultivable.
- Motility
  - Locomotion
    - cork screw rotation
    - Propulsion
  - change of shape
    - A - Angulation
    - B - Buckling
    - C - Coiling
    - D - Undulation
    - E - Expansion.

Classification of Syphilis

- Acquired
- Congenital
  - Mother to fetus (in-utero)
    - < 2yrs
    - > 2yrs

active space

Acquired : transmitted by

- sexual route
- Blood transfusion
- Primary
- Secondary → Early Latent } Infective.
  → Late Latent }
- Tertiary

Natural course :

Exposure to Treponema pallidum ⅓ infected

↓ (10-90 days)

Primary (chancre)

↓ (2-12 wks)

Secondary (mucocutaneous lesions/organ involvement)

↓ (2-12 wks) ↗

Early Latent ‡ Relapsing (in 25%) (1-2yr from contact)

75% ↓

Late Latent (more than 2yrs)

Remission ⅔

Tertiary ⅓

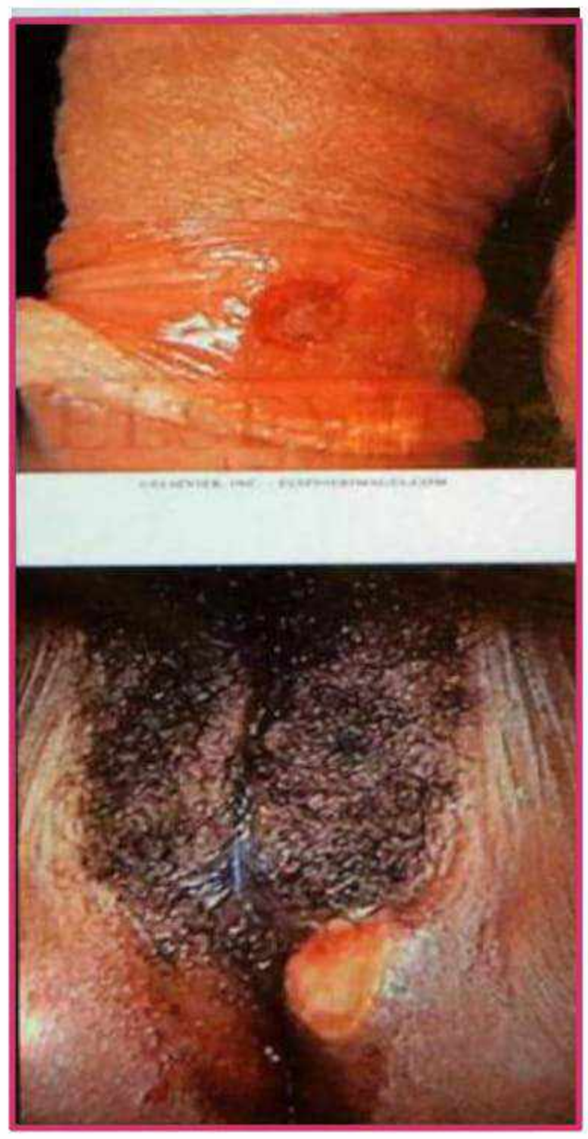
- Late Benign (16%)
- Cardiovascular (10.4%)
- Neurosyphillis (6.4%)

active space

Primary Syphilis

- I.P → 10-90 days (size of inoculum)
- Lesion → Hard chancre / Hunterian chancre / sore
- Number → One
- Site :
  - i) Male : Glans, Sulcus, Foreskin
  - ii) Female : Labia, Fourchette.
- Symptom : None.

Starts as Dull Red macule
↳ Papule
↳ Ulcer

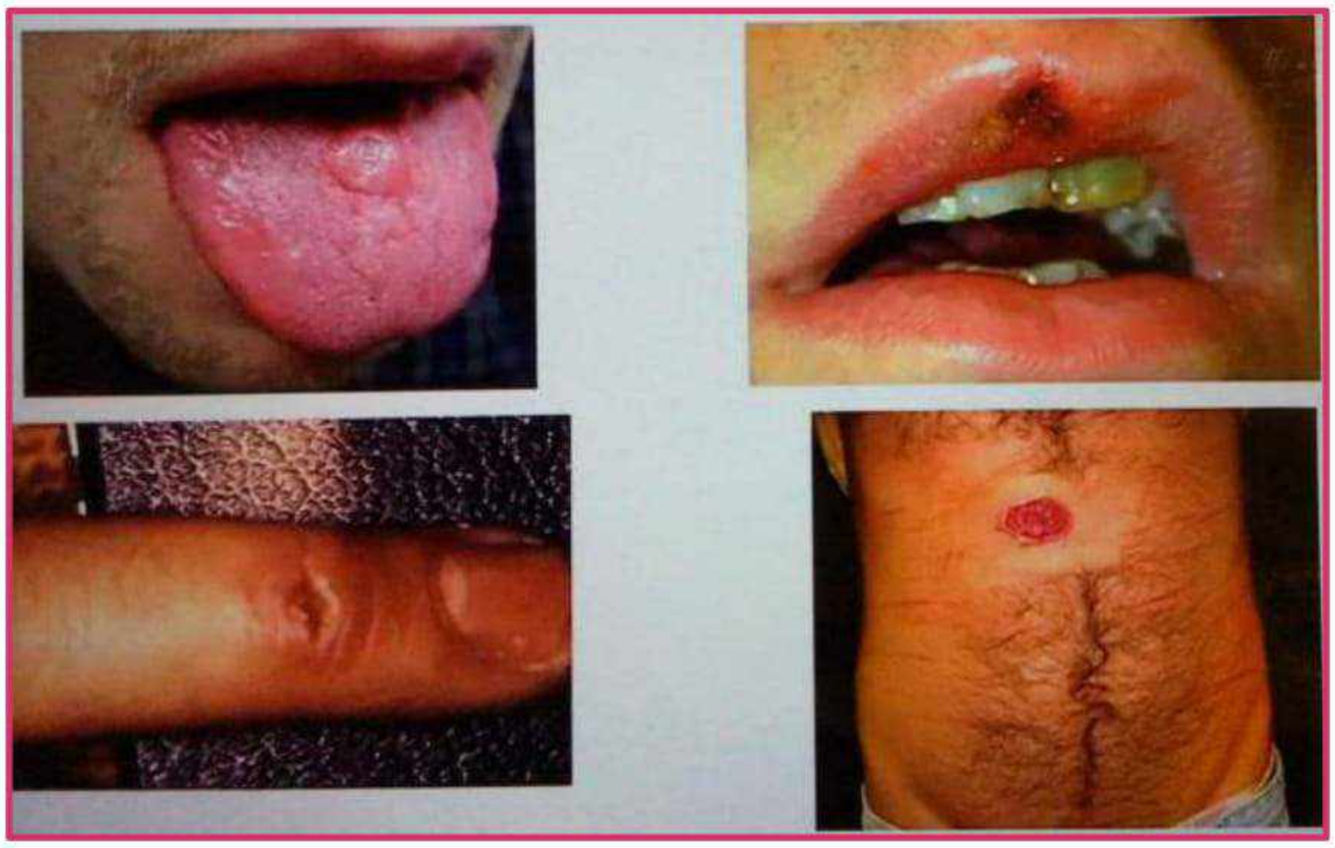


Features of Ulcer :

- Painless
- Single
- Raised Round Rubbery Margins
- Base → Ham coloured clean base
  ↓
  covered by a greyish slough.

- Indurated
- Round
- Well demarcated ulcer
- Button sign : because of induration of ulcer
- Lymph nodes :
  - Bilateral
  - Discrete
  - Painless
  - Rubbery in consistency
  - Shotty

Extra Genital lesions :



active space

Secondary Syphilis :

Primary chancre —(2-12 wks)→ 2° Syphilis

Primary syphilis

↓

Spirochaetemia

↓

Treponemes disseminate to all the tissues via Blood stream

↓

Multiply in the tissues

↓

Secondary syphilis.

Clinical features :

- Constitutional symptoms → Flu like syndrome

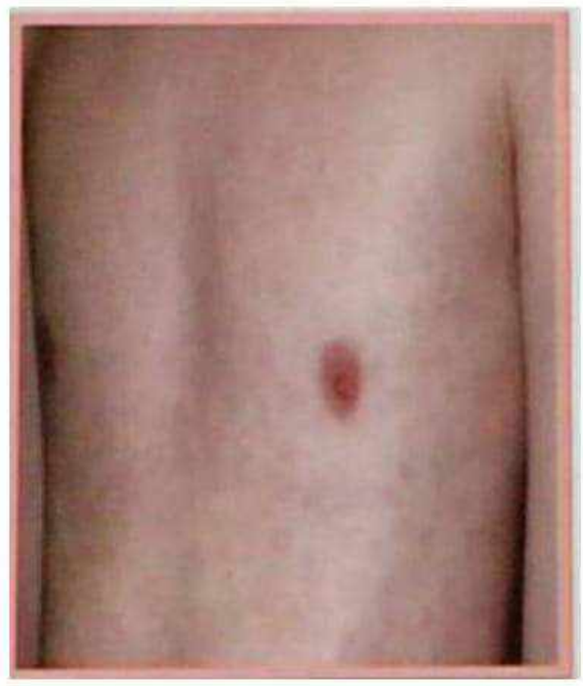
  ⇩

  fever, myalgias, headache

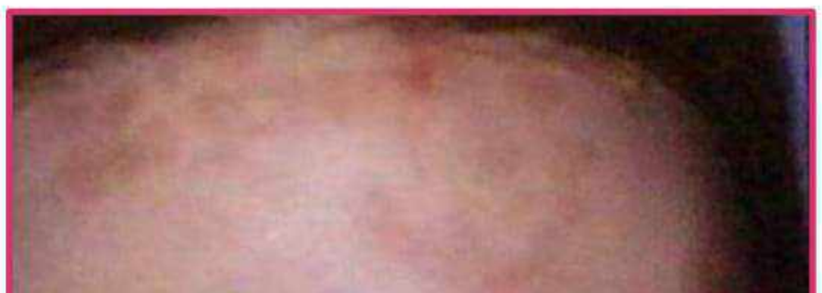
- The Great Imitator :
  - Skin
  - Lymph nodes
  - Mucous membranes.
  - Involves different systems.

active space

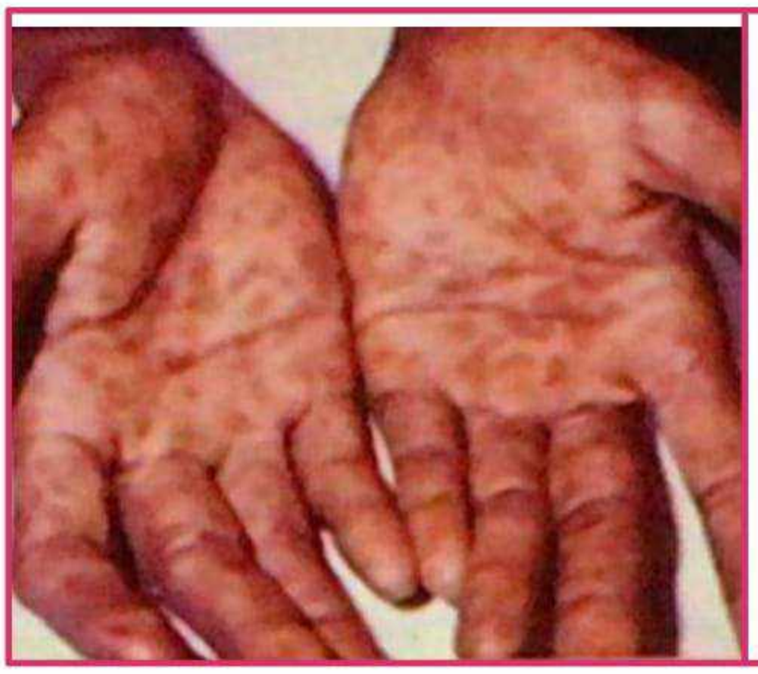
- **Skin lesions :**
  - Widespread
  - B/L Symmetrical
  - Painless
  - Polymorphic → No vesicles and no bulla
    - Ham coloured / Dusky Red coloured.
- **Macular / Roseolar Syphilide**
  - Rose like
  - 1st presentation
  - might be missed
  - Discrete dull red macules all over body
  - Asymptomatic.



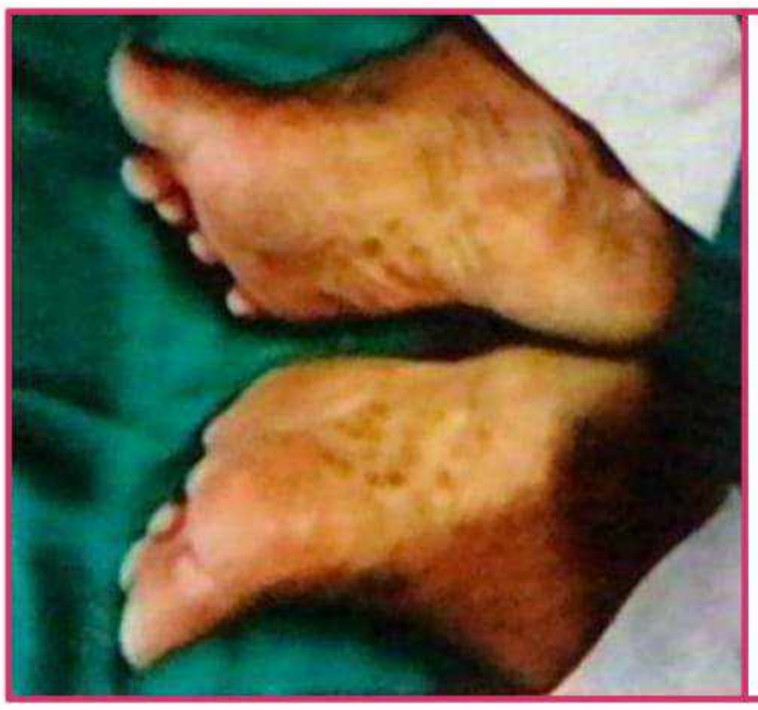
- **Papular Syphilide :**
  - most characteristic 2° syphilitic Rash



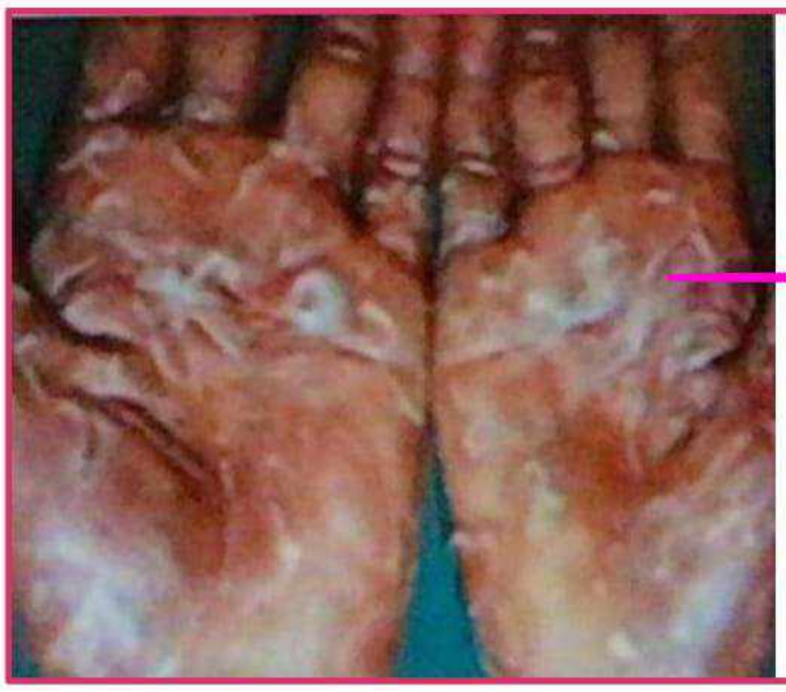
- discrete dull red papules all over the body.
- Diascopy → Blanch.



- Dull red macules / papules on palms and soles



- Hyperkeratotic
  - ↳ Syphilitic corn

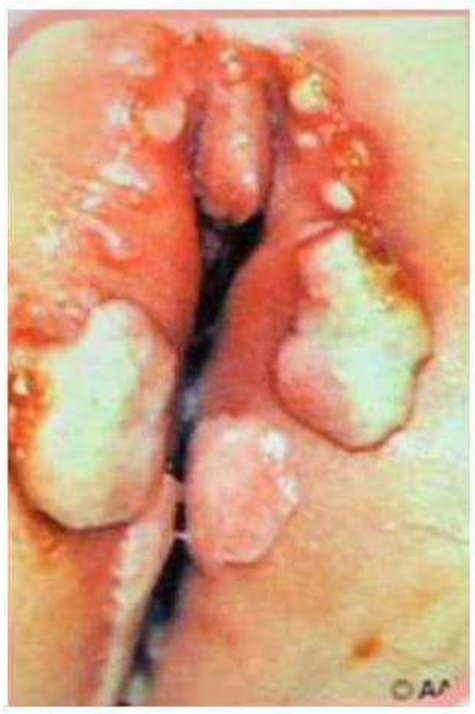


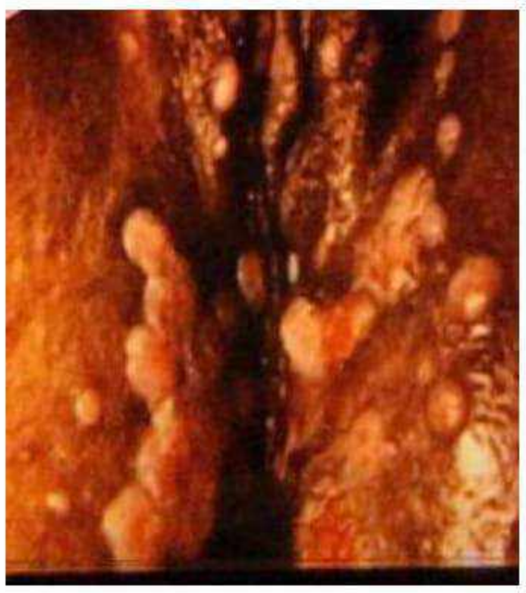
Clavi syphilitica - calluses

→ Scaling

active space

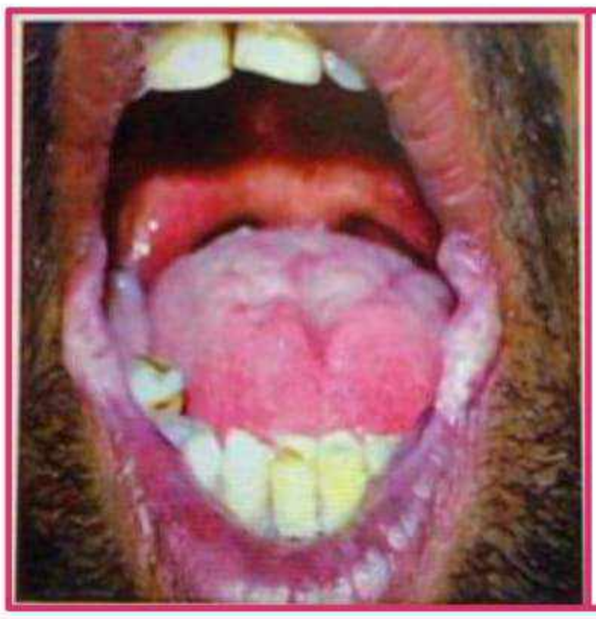
- **Buschke Ollendorf Sign :**
  - Tenderness elicited by giving a vertical pressure with a blunt end of pin/ pen.
    (Endarteritis Obliterans)



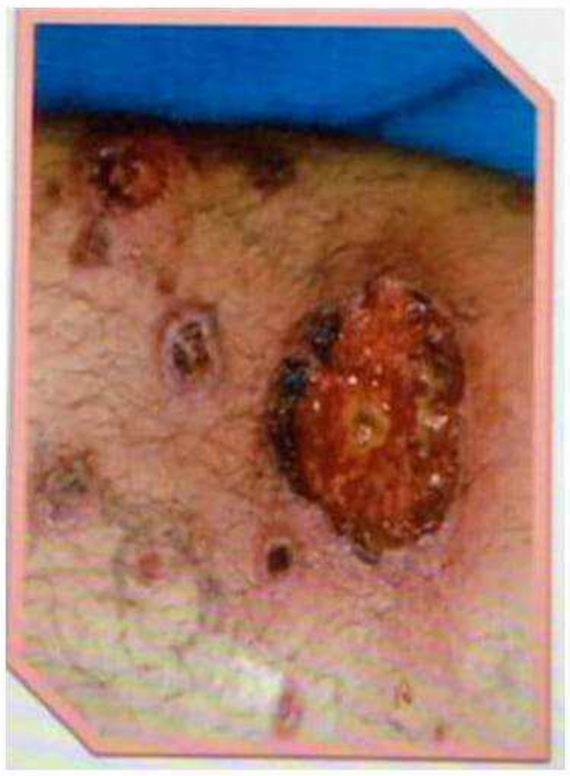


Condyloma lata :

- Broad based flat well defined round lesions seen over genital/ perianal area / axilla.
- covered with greyish slough → if removed
  ↓
  oozy surface.
  ↓
  teaming with spirochaetes



- Papules at angles of mouth.
  ↓
  Split papules.



Pustular lesions

↓

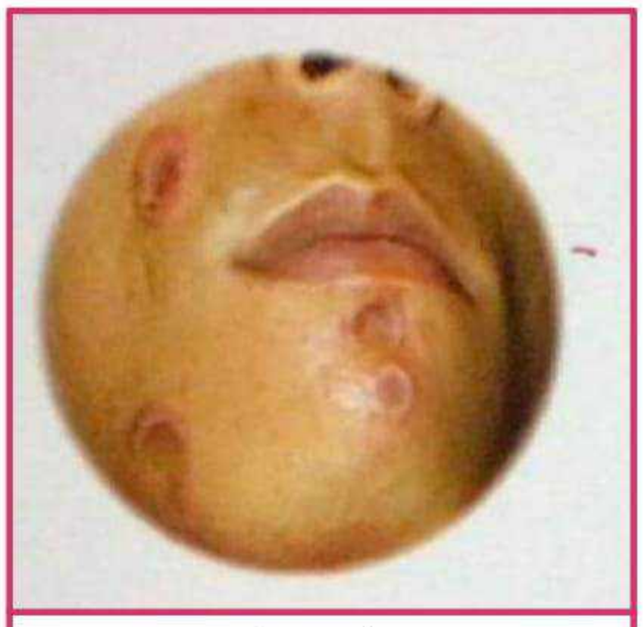
Necrose

↓

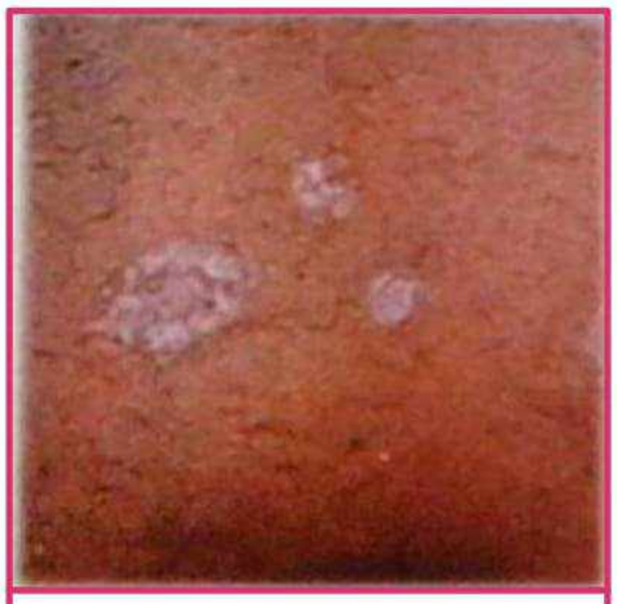
Necrotic ulcer + +

constitutional symptoms.

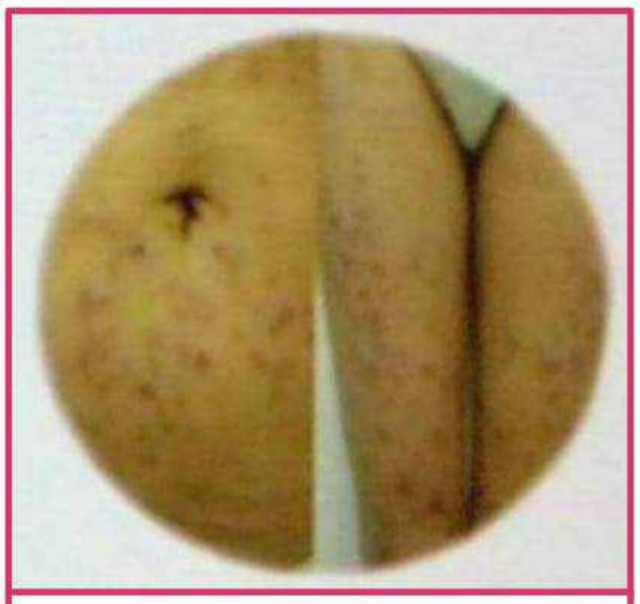
→ LUES MALIGNA



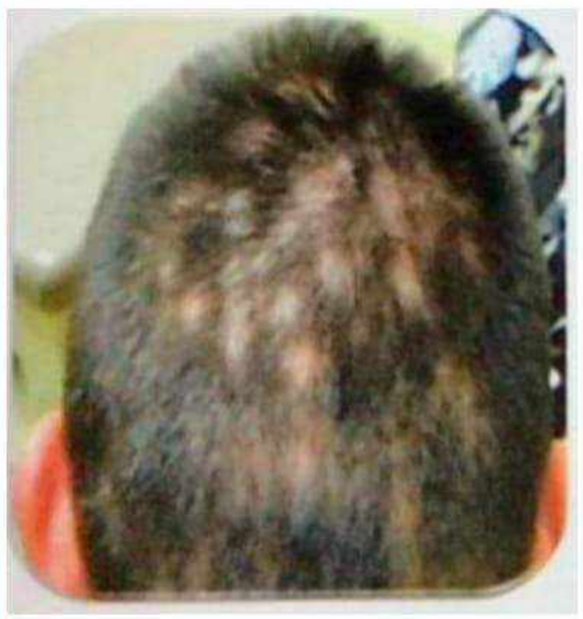
Annular lesions



Papulosquamous lesions



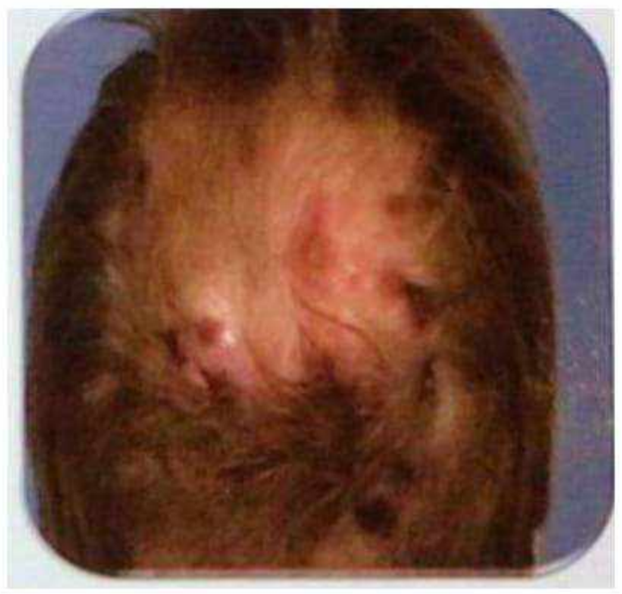
Lichenoid lesions.



Follicular

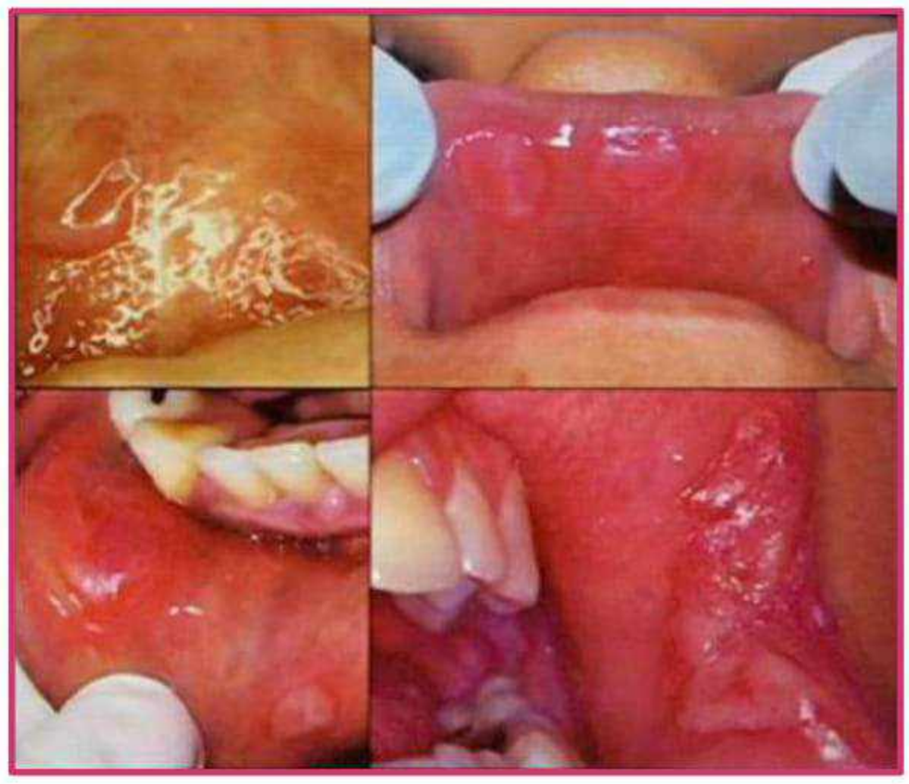
Moth Eaten Alopecia -

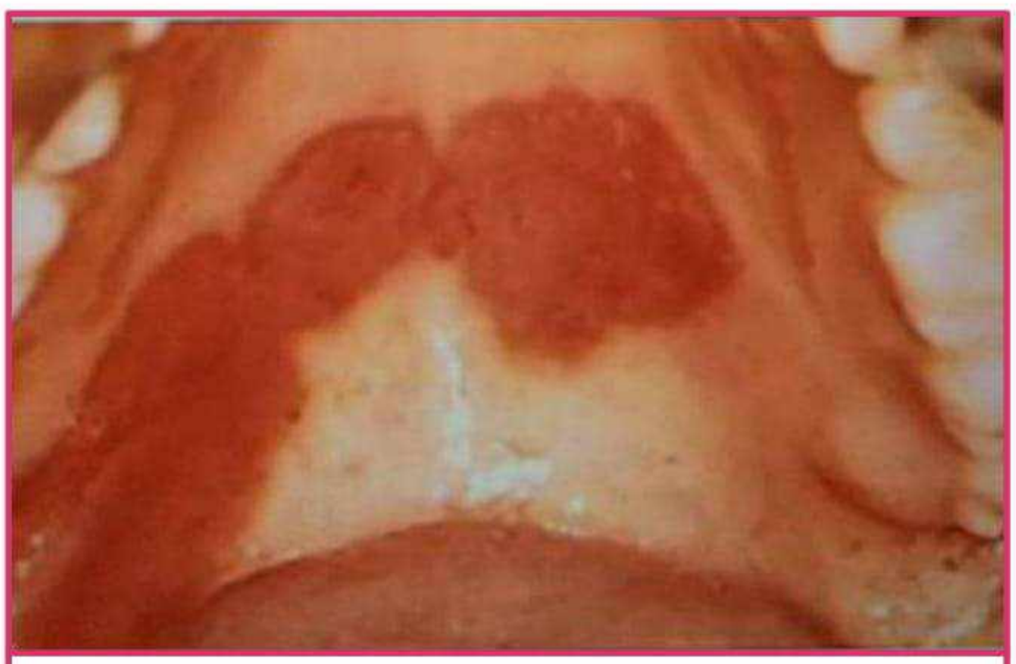
- Multiple patches of non-cicatricial alopecia



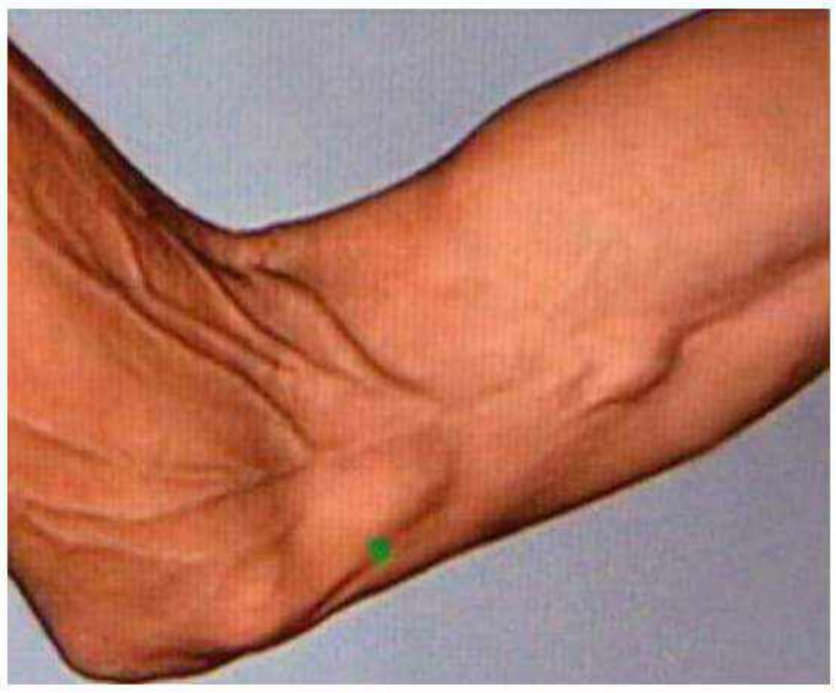
active space

- **Mucosal Lesions :** Mucous patches.
  - Well defined round patches covered with a greyish slough surrounded by dull red areola.
  - Asymptomatic.





Snail Track ulcers



Lymphadenopathy :

- Widespread, Rubbery consistency
- B/L Symmetrical, Painless
- Axillary, Cervical, Inguinal.
- Epitrochlear lymphadenopathy

active space

Systemic Manifestations :

- CVS
- CNS
- Hematological
- Hepatosplenomegaly.

Syphilis d'emblee :

- patient does not have 1° stage
- starts with 2° stage
- transmission through Blood transfusion.
- Non-venereal.

LATENT SYPHILIS 42:00

- **Latent :**
  - No signs and symptoms
  - Serological evidence ⊕
  - CSF ⊖
  - Look for tell tale signs
  - Classified into → Early → < 2yrs - CDC < 1yr → Infective
    → Non-infective.
    Late → >2yrs - CDC > 1yr

RELAPSING SYPHILIS 44:50

- Patient in latent phase → 25 % of them → Relapse
- usually it is manifested as 2° syphilis
- Clinical / serological
- **Monorecedive or Chancre Redux :**
  - Appearance of lesion resembling 1° chancre which appears at the same site.
  - Type of Relapsing syphilis

TERTIARY SYPHILIS 46:40

- Late Latent → $^{2}/_{3}^{rd}$ → Remission
  → $^{1}/_{3}^{rd}$ → 3° syphilis
- It is a non-infective phase
- **Hypersensitivity Reaction to few remaining Treponemes / Components.**
- It is a Type 3 HSR.
- IP : 10-40yrs.
- 3° Syphilis → 3 Types → CVS (Late Benign)
  → Neurosyphilis

active space

- **Late Benign :**
  - 3° Syphilis affecting any part of body except CNS and CVS.
  - (MC) involves skin, bones, mucous membranes.

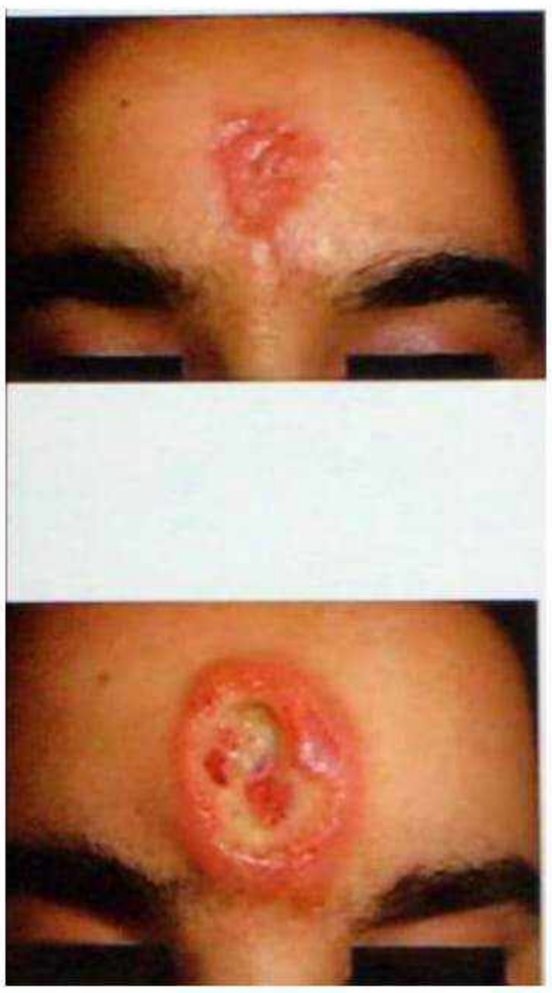
Skin lesions in 3° Syphilis

- can be :

i) Granulomatous plaques

ii) Psoriasiform plaques

iii) Gummas. → present on any body organ/part



- Deep papules/nodules asymptomatic
- Dusky Red in colour

↓

Gummy consistency

↓

Breaks down to form ulcer

Well defined margin

Greyish necrotic slough in centre.

Heal with scarring

Non-infective.

active space

Mucous membrane lesions :

- destruction of nasal cartilage
- perforation of nose and palate
- **Pseudo chancre redux :**
  - Gumma develops at the site of 1° chancre.

Skeletal changes :

- Osteitis, periosteitis
- Nocturnal pain, swelling, tenderness.

CVS Syphilis :

- Aortitis ⟶ (MC) Ascending Aorta
- Aortic aneurysm
- Aortic wall incompetence

Neurosyphilis :

- Asymptomatic.
- Meningeal
- Parenchymatous
- Investigation : CSF examination needed.

General Paresis of Insane :

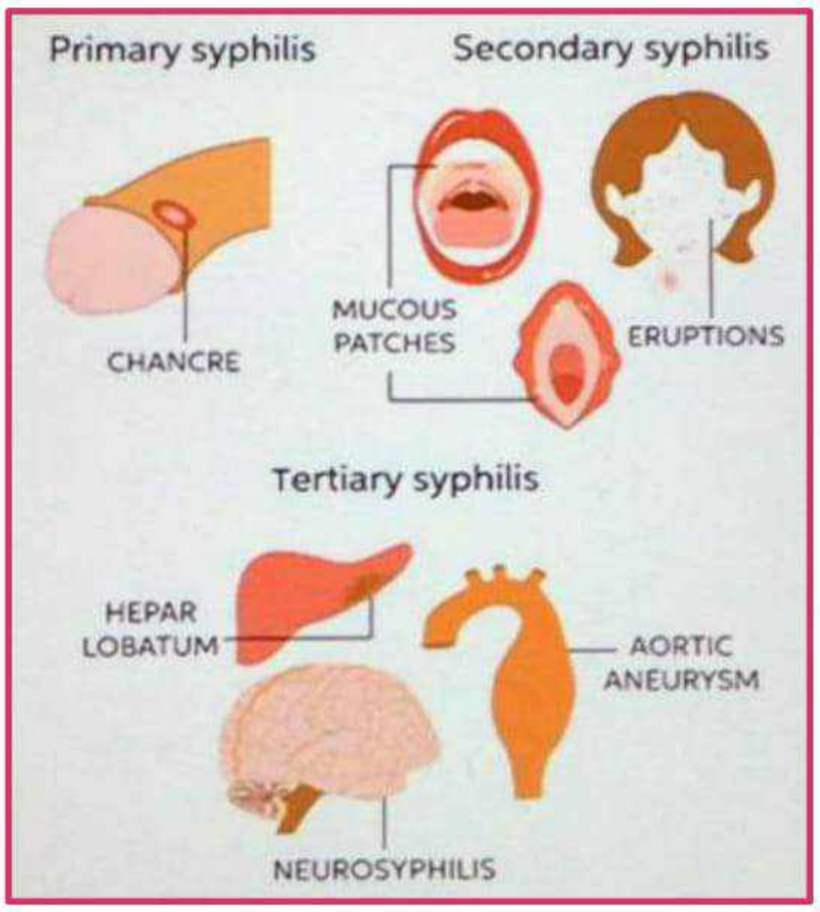
- P → Personality / Psychiatric changes
- A → Affect disorder
- R → Reflexes
- E → Emotional disturbances
- S → Sensorineural defect
- I → Intelligent affected
- S

Tabes dorsalis

- Posterior column affected
- Gait abnormalities ⊕
- Slapped foot gait

Charcot joints

- Non-inflammatory, non-painful swelling of joints



Congenital Syphilis :

- Transmission mother to fetus in utero
- affected after 20wks of Gestation

"A little wrinkled pot bellied old man with a cold in his head"

Early

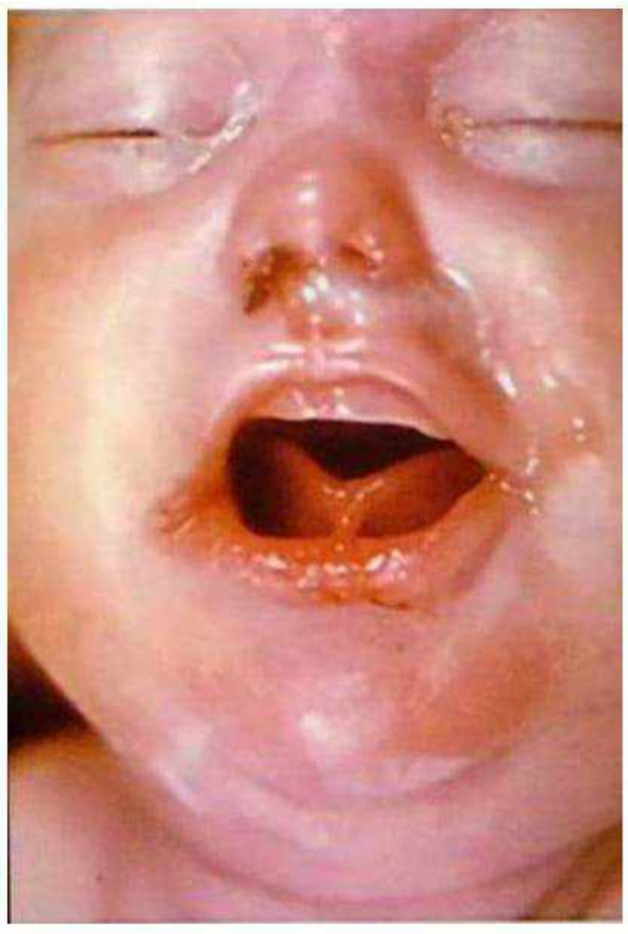
- < 2yrs
- ∽ 2° syphilis
- Infective

Late

- > 2yrs
- ∽ 3° syphilis (stigmata)
- Non-infective.

Clinical features :

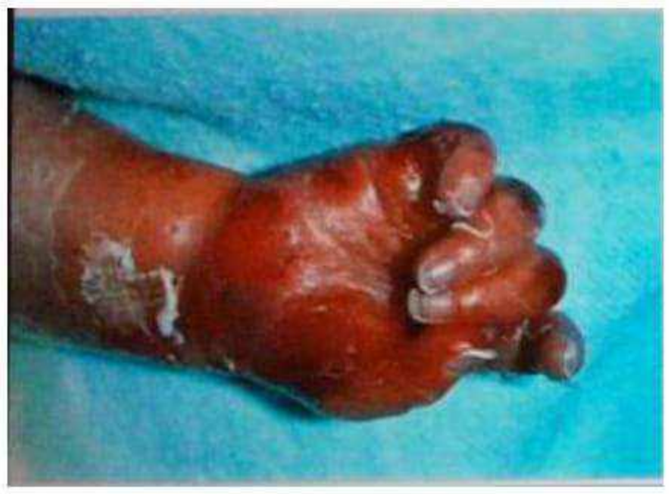
- Low Birth Weight
- Anemia
- Hepatosplenomegaly



Snuffles → Rhinitis.

- discharge teaming with spirochaetes.
- very infective

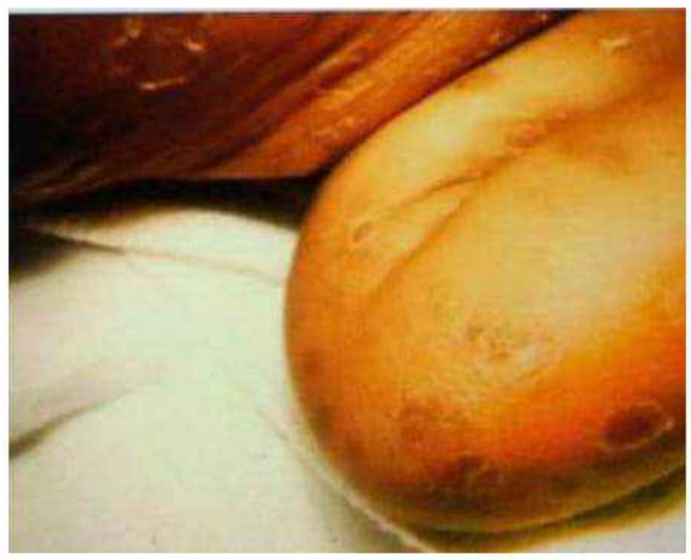
Fleur de lis nose : Saddle nose, destruction of cartilages.

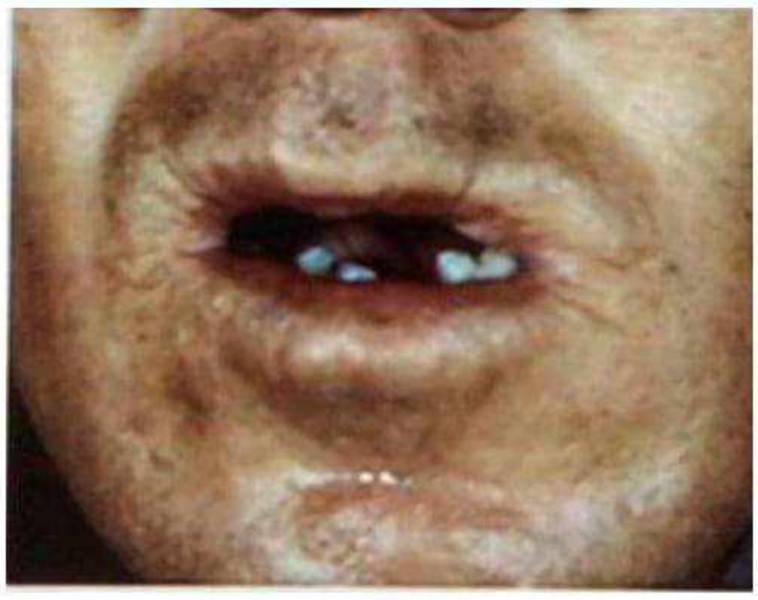


Bulla and vesicles may be present.

↓

Syphilitic pemphigus.



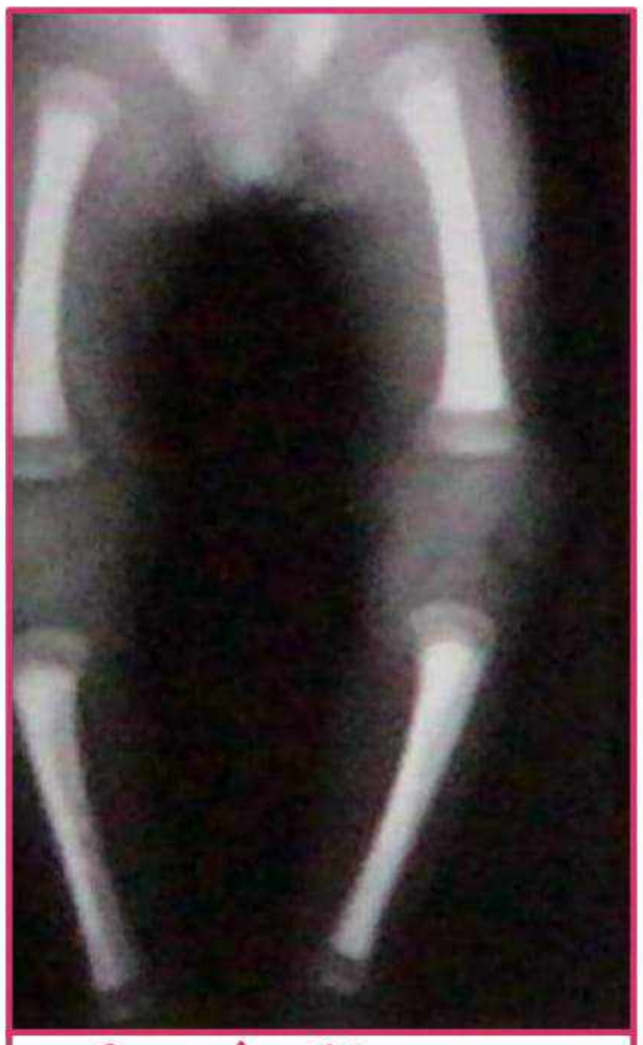


Rhagades / Parrots beak appearance

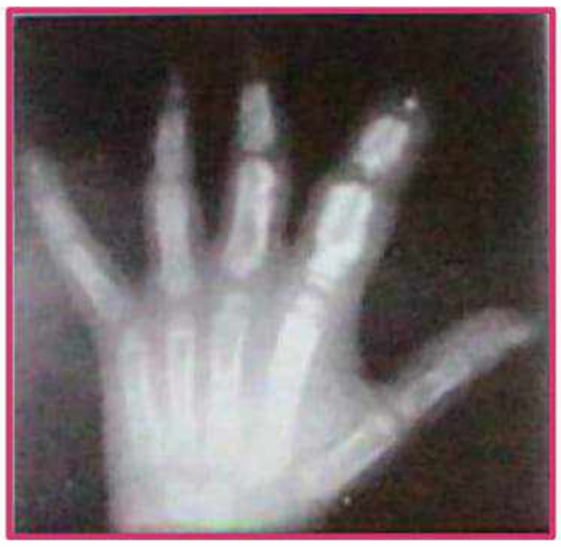
- linear scar at angles of mouth.

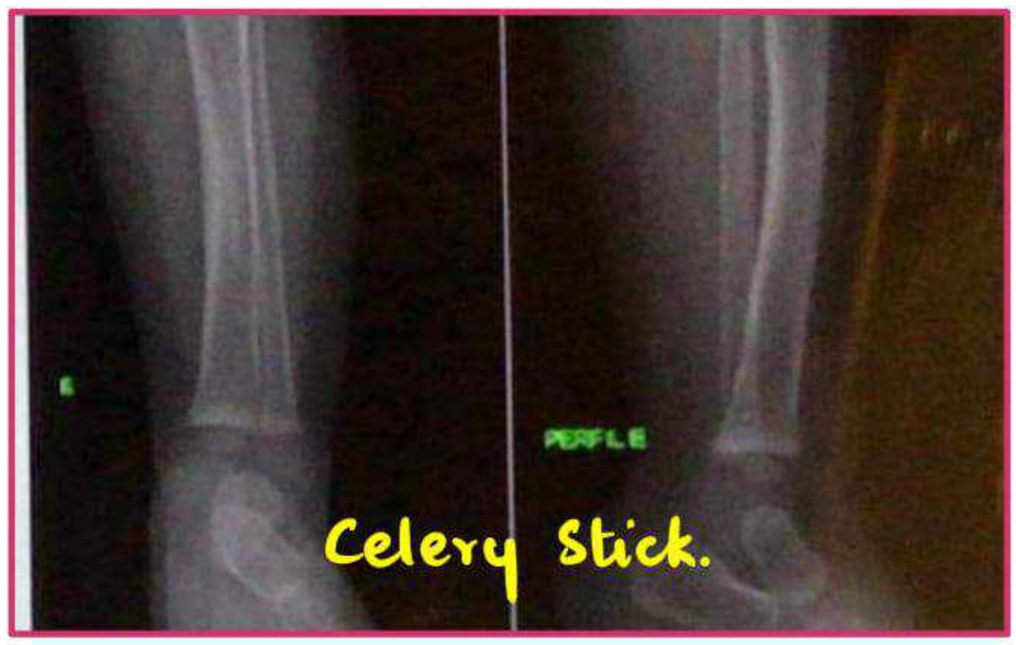
Bone

- Osteochondritis - (MC)
- Pseudoparalysis of Parrot ⟶ child not able to move the limbs because of pain
- Wimberger or Cat Bite sign : Osteochondritis of metaphysis - proximal end of tibia.
- Celeray Sticks : Lines of rarefaction on long bones
- Syphilitic dactylitis : Fusiform swellings of digits.



Saw tooth
Wimberger or
Cat bite sign.



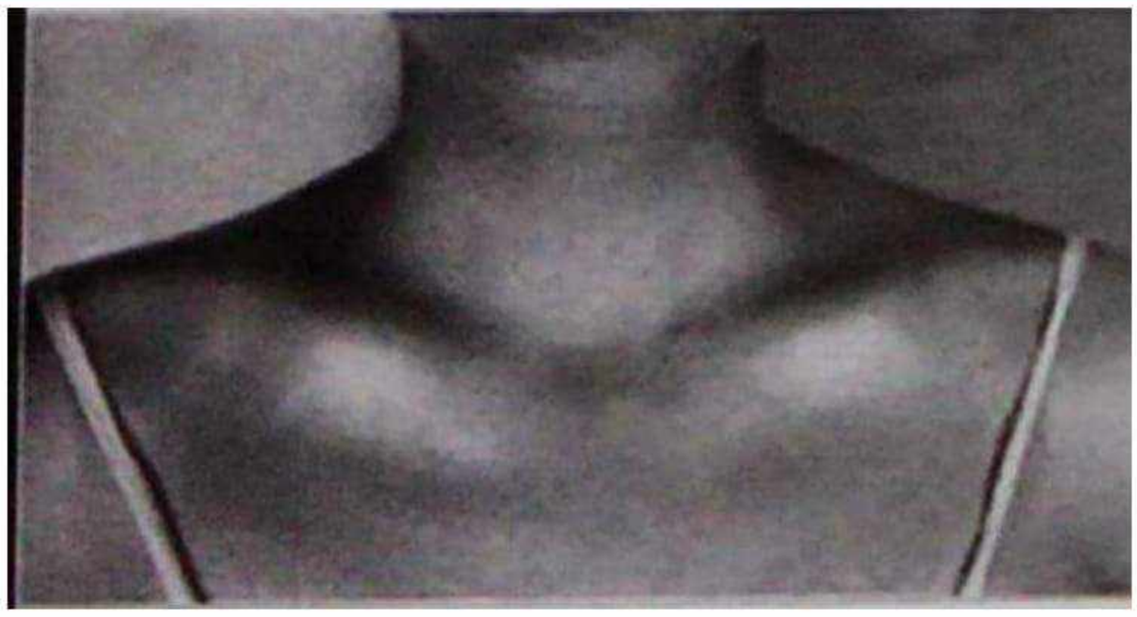


Late Congenital Syphilis

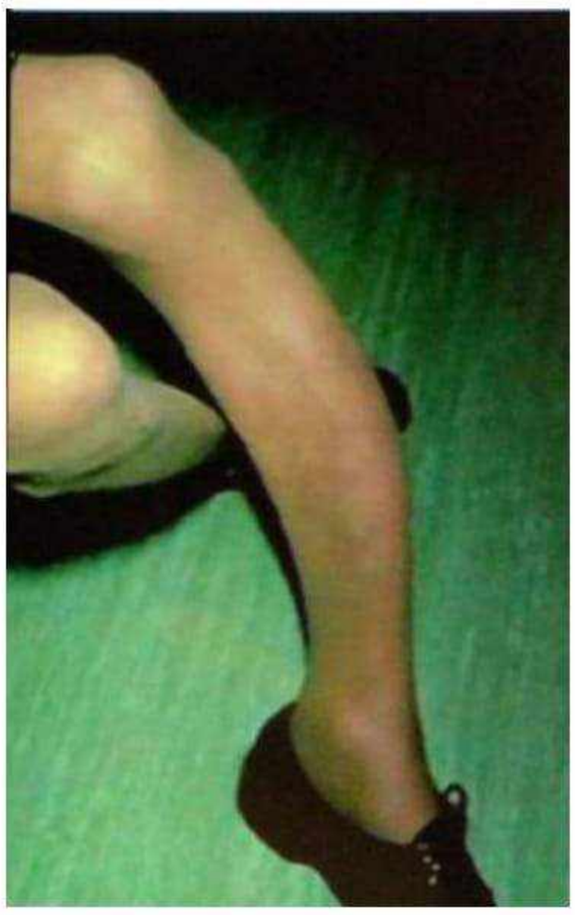
- > 2yrs
- ∽ Tertiary syphilis
- Non-infective
- Patient presents with:
  - Frontal bossing

    Hot cross bun appearance

    OC/Osteitis of frontal bone.



Prominence of medial end of clavicle → Higoumenaki's sign.



Sabre shin / Sabre Tibia

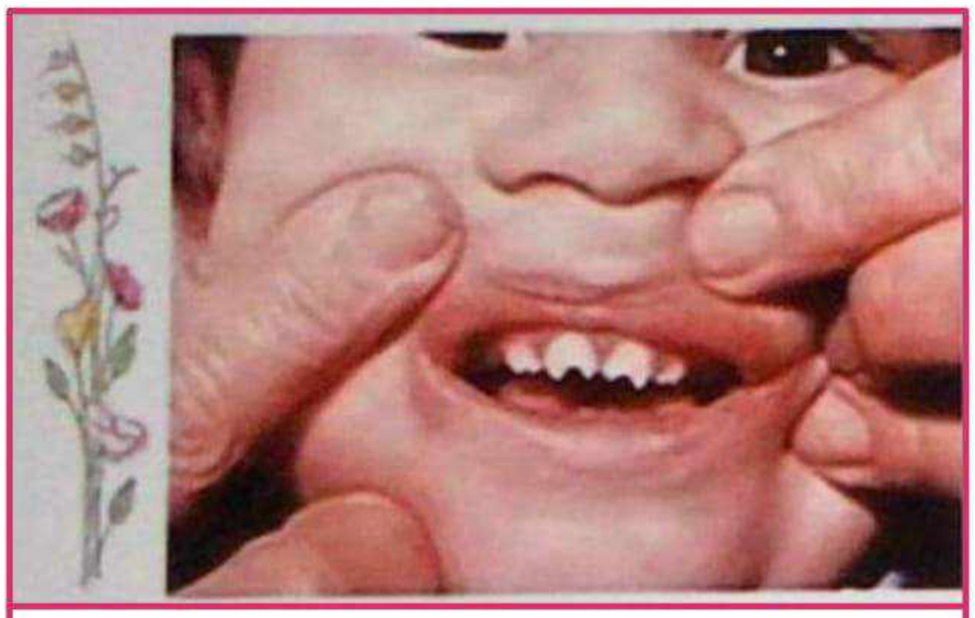
- Anterior border of tibia shows bowing.

Bone changes:

- Olympian brow, hot cross bun
- Short maxilla
- Higoumenaki sign

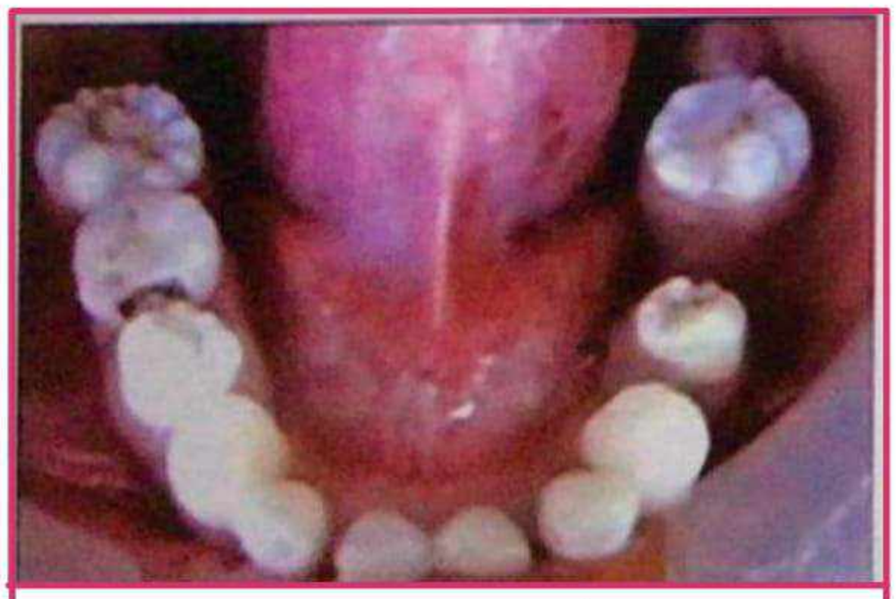
active space

- Saber shin
- Scaphoid scapula
- Bull dog jaw → Prominence of mandible
- Clutton's joint. → Non- infectious, non-painful swelling of knee joint 2° to trauma.



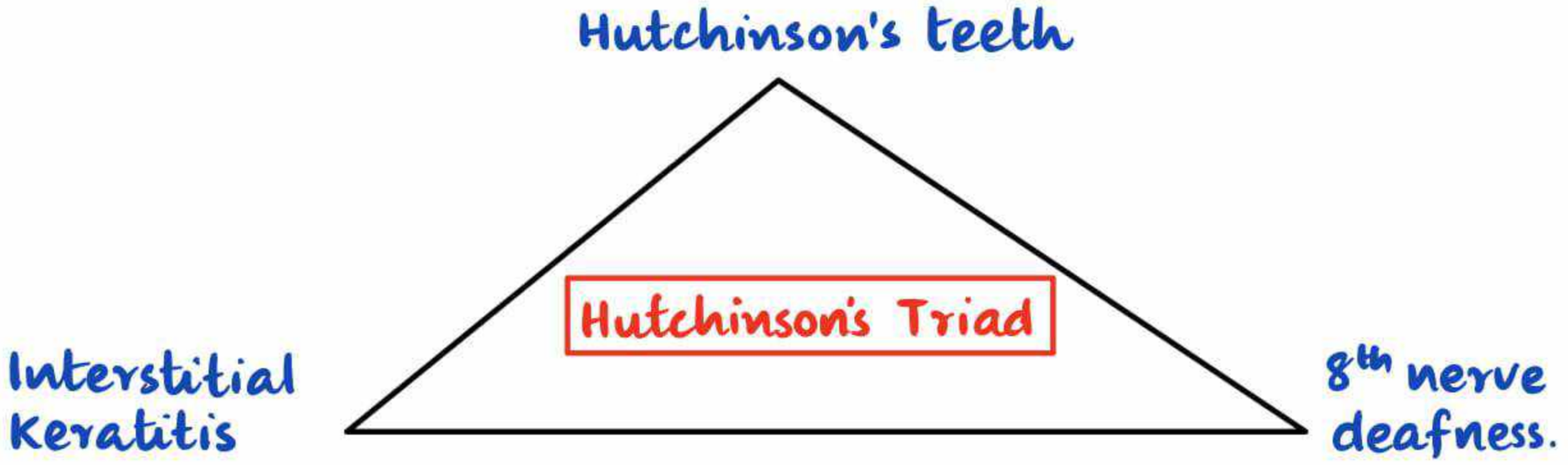
Peg shaped permanent central incisors
↓
central notches
Hutchinson's Teeth.

Palatal perforation



Mulberry molars

- 2 characteristic / specific features of late congenital syphilis -
  - i) Mulberry molars
  - ii) Interstitial keratitis.

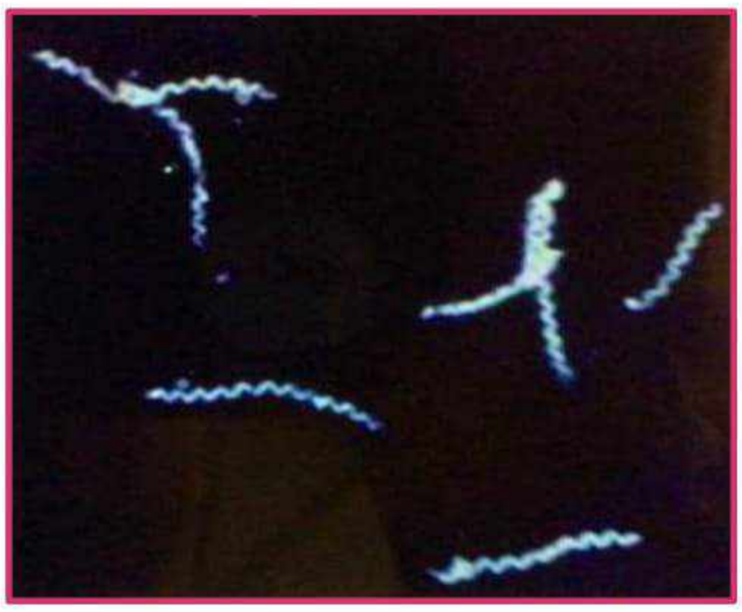


INVESTIGATIONS

1:16:40

- Identification of organism.
- Serology
- CSF
- Radiological

1. Identification of organism: Dark ground microscopy.



- organism seen as slender, thin, coiled, wavy.
- Sample from: Condyloma lata, mucous patches, chancre, lymph node.

active space

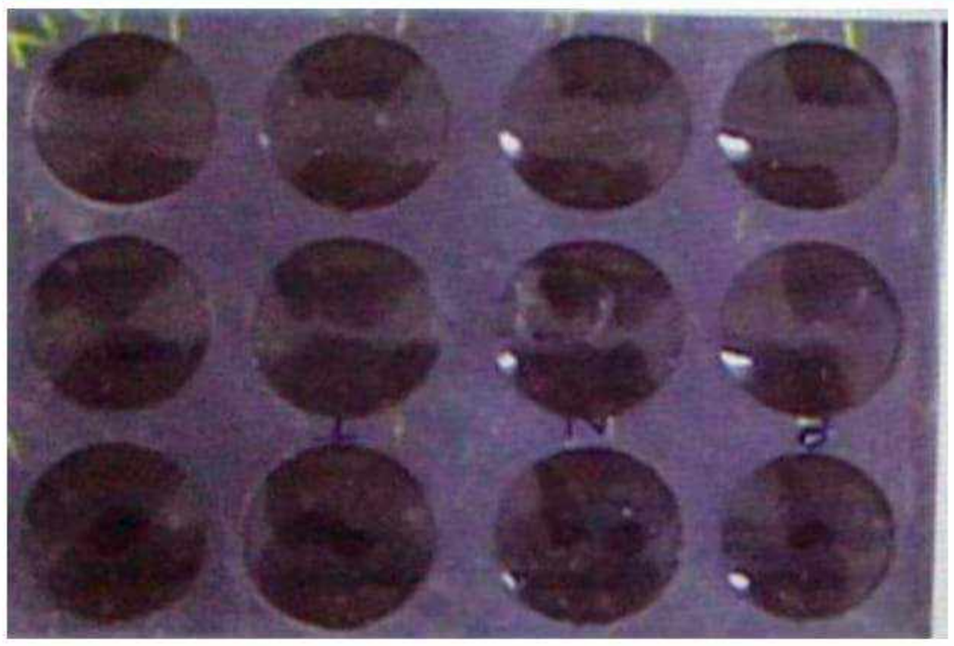
2. Serological tests

Treponemal / Specific Tests

- TPHA, FTA-ABS
- TPI, TPPA
- EIA
- Latex agglutination

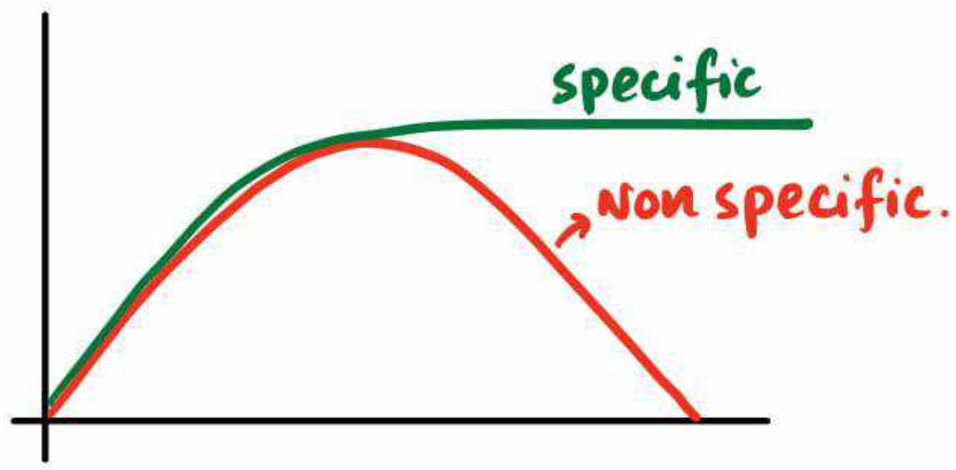
Non-Treponemal / Reagin Tests

- VDRL, RPR
- WASSERMAN
- TRUST
- RST, UST

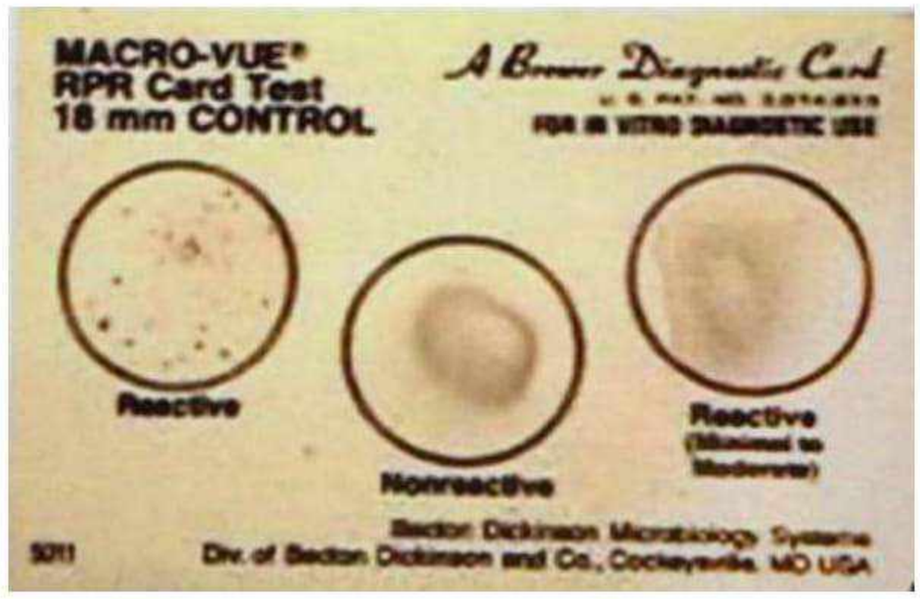


VDRL Slide flocculation Test :

- Read in dilutions
- > 1 : 8 ⟶ Significant
- Screening test
- Congenital Syphilis
- Monitoring response to Treatment
- Reinfection.



RPR - Rapid Plasma Reagin Test



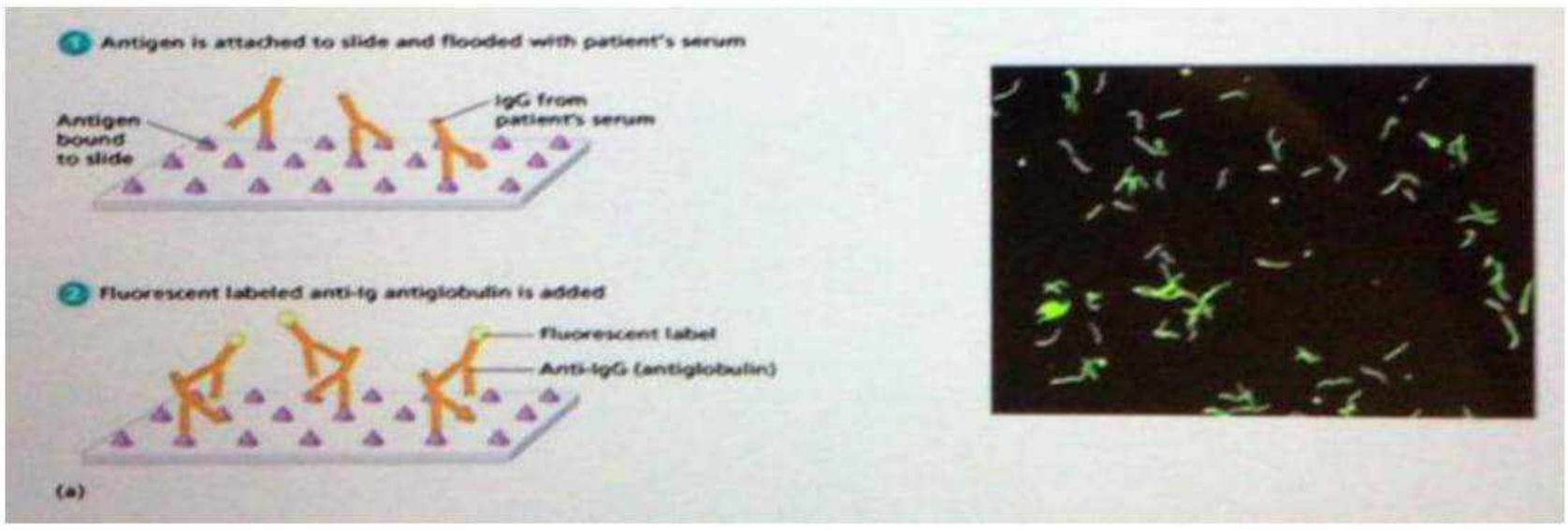
- Modified VDRL antigen is coated on carbon particles
- used in field screening.

TPHA : Treponema Pallidum Hemagglutination test.

- Antigen - suspension of tanned sheep red cell coated with an extract of Nichol's strain.
- High specificity and sensitivity

FTA-Abs (Fluorescent Treponemal Antibody Test)

- Antigen - drop of suspension of dead treponema pallidum (Nichol's virulent strain)



- Earliest test to become ⊕ ve (IgM captia (EIA) → 1st to become positive)

active space

PCR

- Gold Standard test for early syphilis.
- Multiplex PCR → i) H. ducreyi
  ii) HSV
  iii) Syphilis

IgM EIA (captia)

- very early 1° infection
- Assessment of re-infection
- Congenital Syphilis
- End of 2nd wk of infection.

Investigations in Congenital Syphilis

- Increased VDRL dilution in child serum vis a vis mother serum
- FTA-Abs
- IgM captia-EIA

Neurosyphilis → CSF examination

Summary

- 1st test to become ⊕ve - IgM Captia > FTA Abs
- Screening test - VDRL
- Screening test in fields - RPR
- Specific test : FTA-Abs, TPHA
- Congenital Syphilis : IgM Captia > FTA-Abs.

TREATMENT OF SYPHILIS 01:29:00

i) Penicillin

- Inj. Benzathine Penicillin - 2·4 Million units divided into 2 equal dose of 1.2 Million units each i.m
  - In 1°, 2°, Early Latent → Single dose
  - In Late latent, CVS and 3° Benign Syphilis
    ↓
    3 doses at weekly intervals
  - In children : 50,000 U/Kg upto a maximum of 2·4 MU
- In Neurosyphilis and Congenital Syphilis :
  - Aqueous Crystalline Penicillin i.v
  - Alternative : Procaine penicillin + Probenicid.

active space

- In Pregnancy:
  - dose, duration, Treatment ⟶ same
  - If allergic to Penicillin ⟶ Desensitisation.
- If allergic to Penicillin ⟶
  - Doxycycline 100mg BD → 14 days - Early / 28 days - Late
  - Erythromycin
  - Ceftriaxone
  - Tetracycline

Jarisch Herxheimer Reaction

- after giving penicillin
- (MC) in 2° stage
- Rapid Killing of treponemes
- Within few hours of giving penicillin
- severe constitutional symptoms
  - fever, headache, nausea, vomiting, skin rash, hypotension
  - Symptomatic management: Anti-pyretics

active space

Sexually Transmitted Diseases (Part - 2)

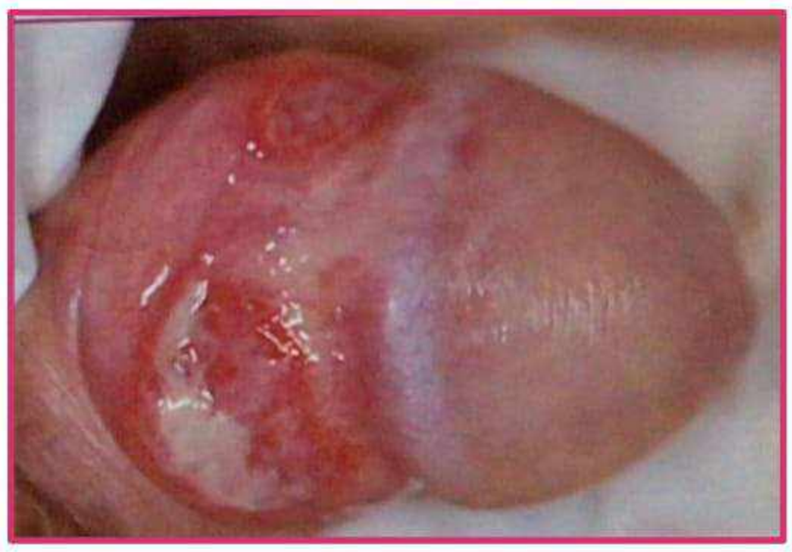
Genital Ulcer Disease

1. Syphilis
2. Chancroid
3. Herpes Genitalis
4. Donovanosis
5. LGV

CHANCROID

00:50

- a.k.a Soft chancre
- caused by Hemophilus ducreyi
- I.P ⟶ 1 - 14 days



Features of Chancroid ulcer :

- Painful
- Multiple
- Well circumscribed
- Ragged undermined edge
- Not indurated
- covered with greyish slough ⟶ Bleeds on touch.

- Unilateral painful lymphadenopathy

↓

Suppuration

↓

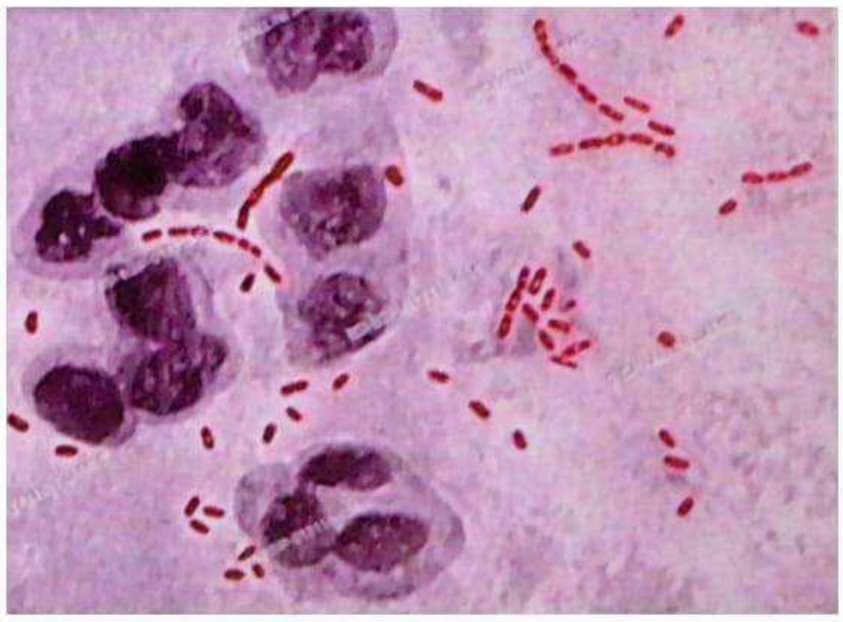
Unilocular abscess → "BUBO"

- Presence of ulcer

Investigations :

1. Gram Stain :

- Gram ⊖ Bacilli arranged in groups of 2-4 → Extracellular
- School of Fish / Rail Track Appearance.



2. Culture : Specific → on Muller Hinton Agar (MHA) enriched with Chocolate Horse Blood.

3. PCR

4. IIF

Treatment :

- Azithromycin 1g stat or Ceftriaxone 250mg i.m stat

active space

HERPES GENITALIS

08:40

- Organism : HSV 2 > HSV 1
- I.P : 2 - 7 days
- Stages : 1°, 2°, Recurrent

Primary → more severe

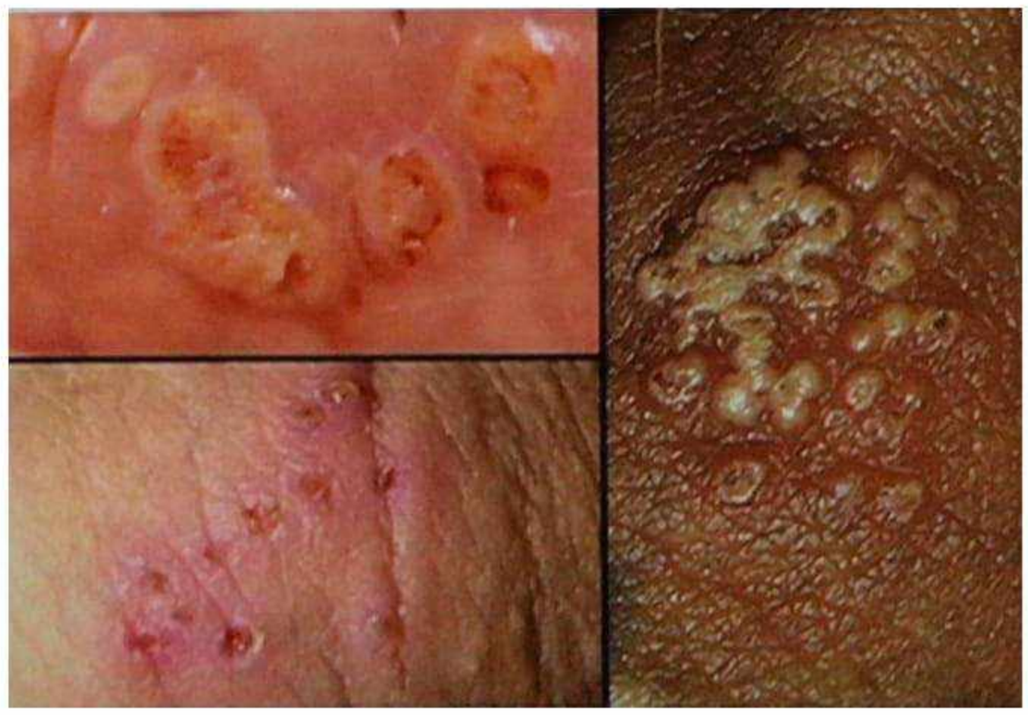
- more constitutional features
- more no. of lesions
- more size
- more pain
- more lymphadenopathy

Recurrent : > 6 episodes / year.

- Latent : Dorsal Root of Sacral ganglion.

Clinical feature :

- Multiple painful vesicles rupture to form polycyclic erosions.
- Sometimes → ulcers.



active space

- B/L painful lymphadenopathy

Investigation :

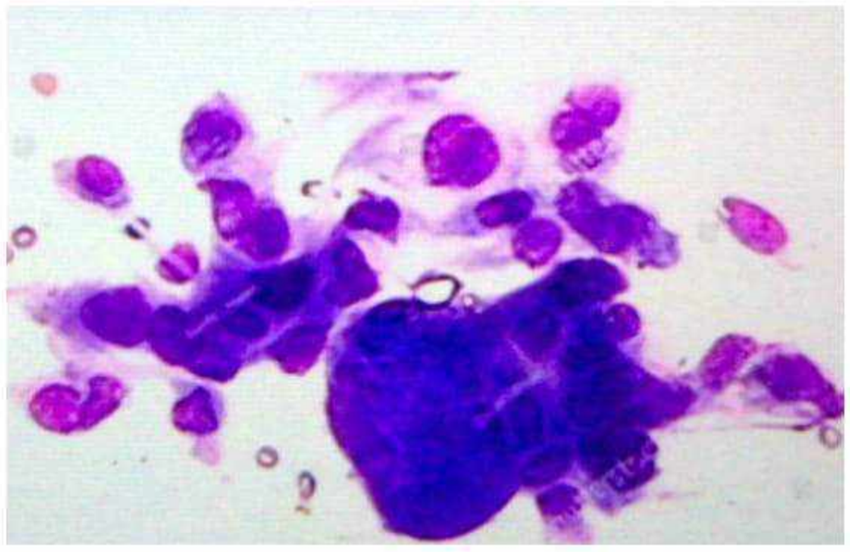
1. Tzanck smear →

   - Multinucleate Giant cells +
   - Acantholytic cells.

   ↳ because of Ballooning degeneration.

   +

   Desmosomal separation



2. Serology : ELISA → Ag- HSV Glycoprotein.

Treatment

i) Acyclovir →

- in 1° → 400mg TDS / 200mg PID x 7-10days
- in 2° → 400mg TDS / 200mg PID x 2-5days
- Recurrent → 400mg BD

ii) Valacyclovir

iii) Famcyclovir

active space

DONOVANOSIS

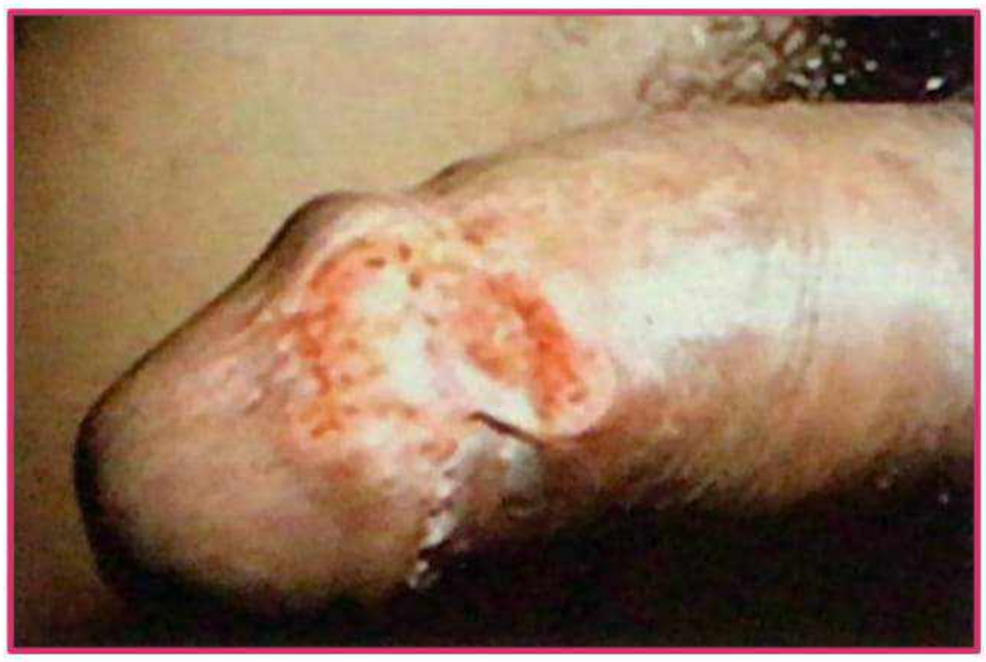
14:30

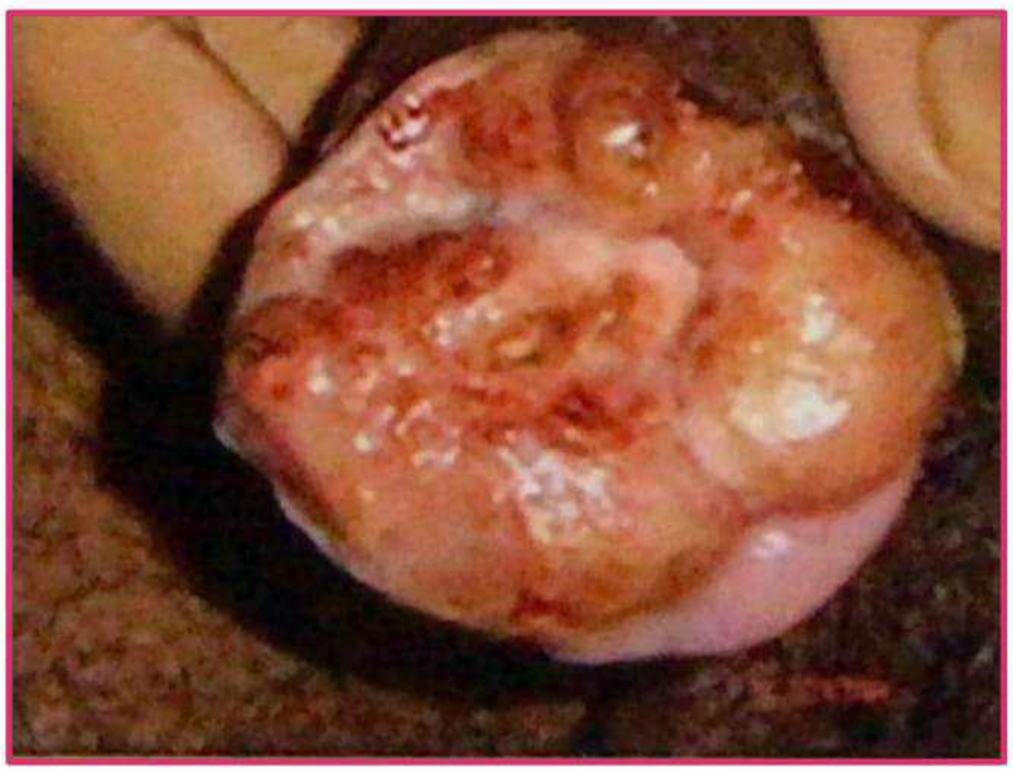
- a.k.a Granuloma inguinale / Granuloma venereum.
- Chronic Granulomatous disease
- organism : Klebsiella granulomatis/
  Calymmatobacterium granulomatis
- I.P : 3days - 3months

Clinical feature :

- Painful ulcer
- U/L or B/L
- Granulomatous ulcer
- Beefy Red colour
- Bleeds touch

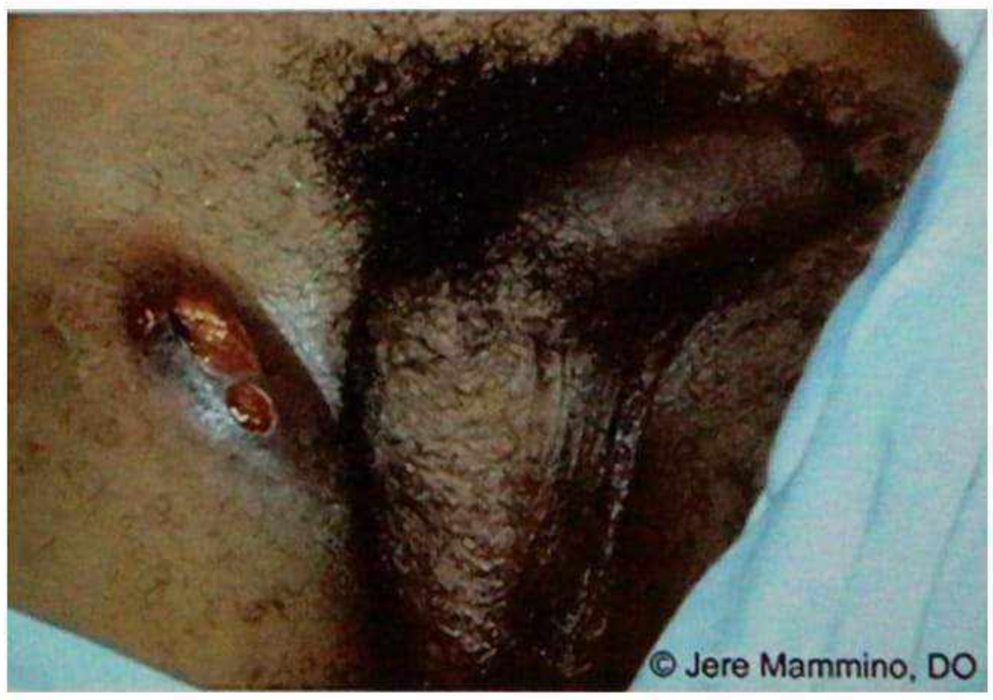
Exuberant Granulomatous disease.





active space

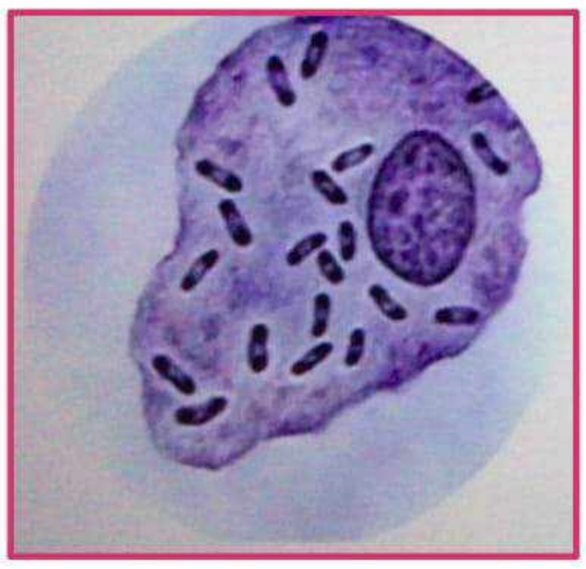
- No lymph node involvement
- Pseudobubo
  ↳ ulcer of Donovanosis over lymph node.



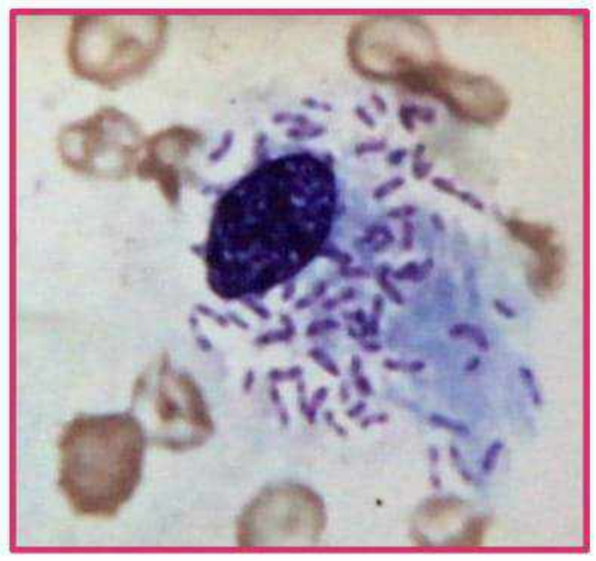
Complications :

- Stenosis of urethra
- Stricture
- Pseudoelephantiasis.
- Destructive ulcer

Investigation :



- G⊖ Intracellular organism peripheral condensation of cytoplasm.
- Safety pin appearance.



active space

- NAAT → IOC
- CFT

Treatment

- Azithromycin 1g/wk, 500mg day → upto 3wks or till lesions resolve
  or
- Doxycycline 100mg BD for upto 3wks or till lesions resolve

LYMPHOGRANULOMA VENERUM

23:00

- a.k.a Climatic Bubo / lymphogranuloma inguinale
- organism : Chlamydia trachomatis (L_1, L_2, L_3)
- I.P : 10-30 days.

Clinical features :

Primary Stage	Secondary Stage	Tertiary Stage
inconspicuous vesicles/erosion/ ulcer.	• Inguinal syndrome	

- unilateral / Bilateral
- Painful lymph node enlargement

+

constitutional symptoms

↓

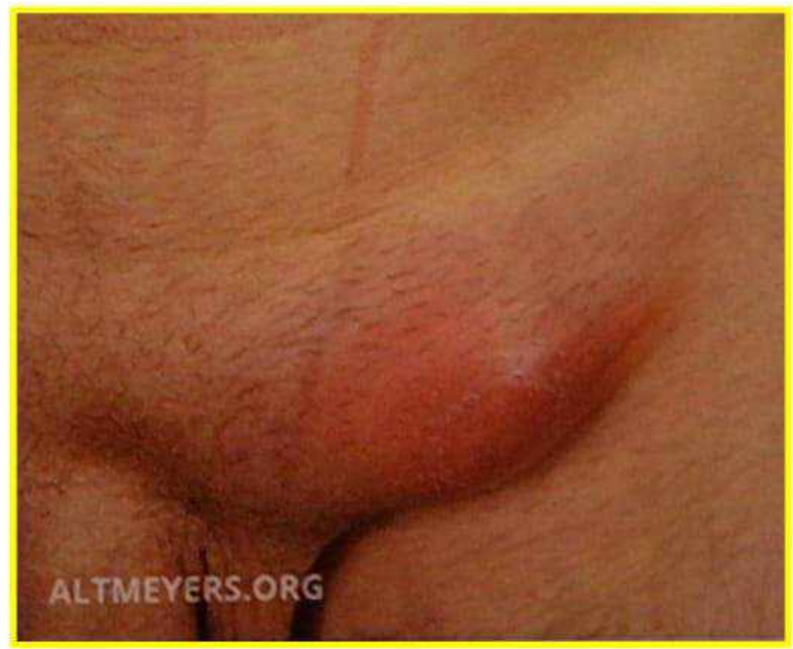
Suppuration

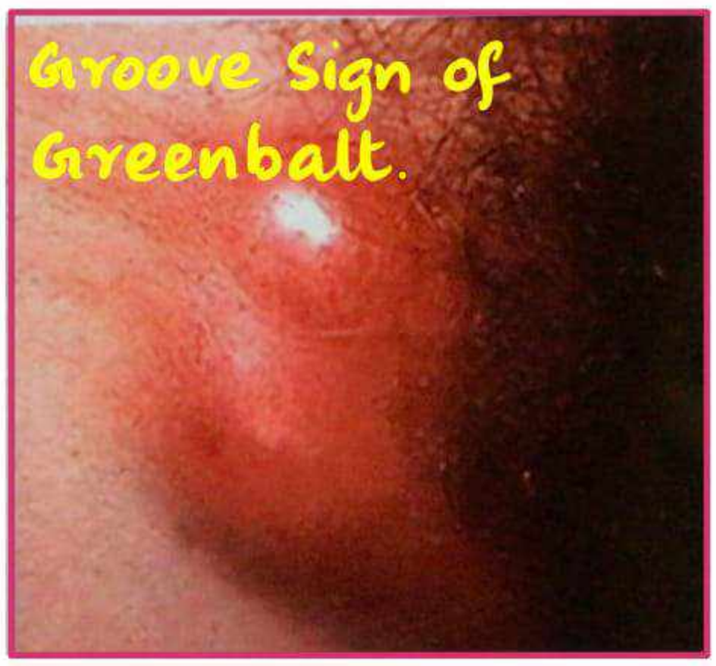
+

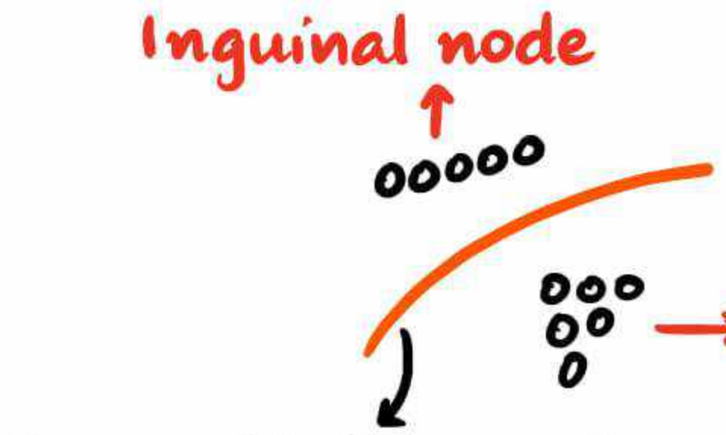
Multilocular abscess

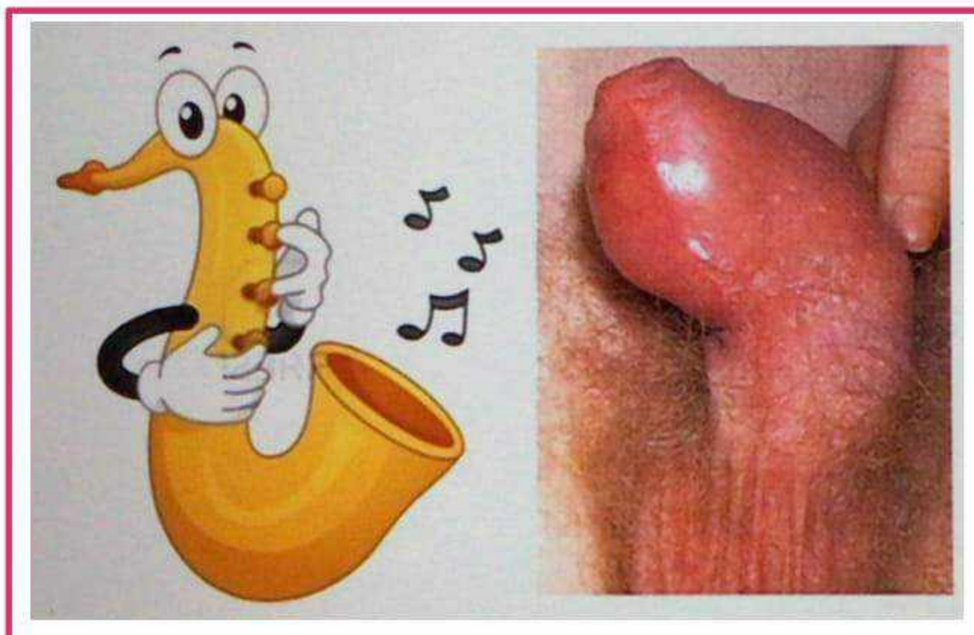
+

Breaks down to form multiple sinus









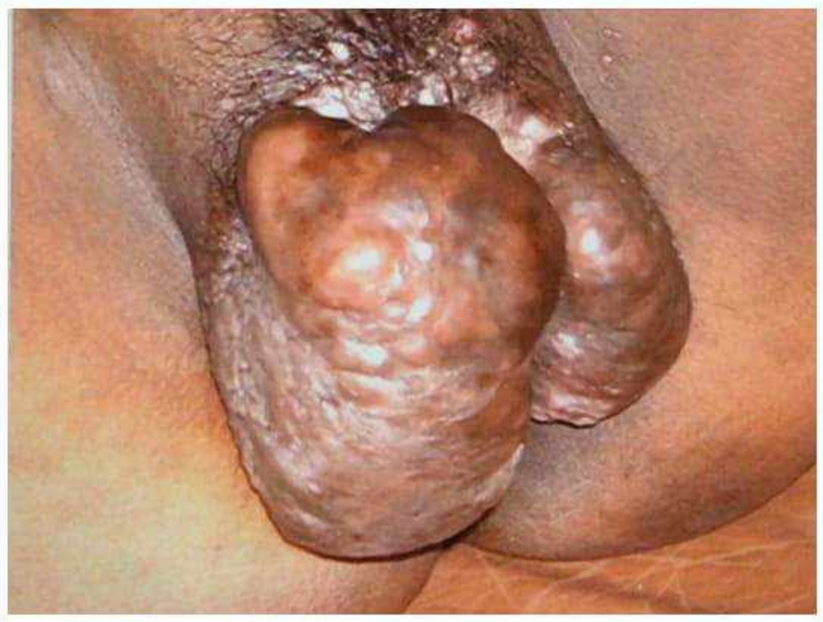
3° Stage

- lymphatic obstruction

↓

Elephantiasis

- Ramhorn penis / Saxophone penis.



Esthiomene

- Elephantiasis of vulvae.

LGV vs CHANCROID

- Both have painful bubo
- Chancroid ⟶ • Unilateral
  - Unilocular
  - Ulcer.
- LGV ⟶ • Multilocular
  - Multiple sinuses.

Investigations:

- Giemsa stain: Inclusion bodies.
- NAAT ⟶ IOC.
- CFT

Treatment: Doxycycline 100mg BD x 3wks.

active space

- pain ⟶ painless : Syphilis , Donovanosis
- Lymph nodes
  - Painful ⟶ Chancroid → u/L , LGV
  - Herpes → B/L
  - Painless → Syphilis

- Syphilis → painless , non-indurated , shotty lymphadenopathy
- Chancroid ⟶ painfull, multiple , indurated bubo
- HSV ⟶ Polycyclic erosions + B/L painful LN
- Donovanosis ⟶ painless granulomatous ulcer , No LN
- LGV ⟶ Buboes → Painful , multilocular , multiple sinuses.

Sexually Transmitted Diseases (Part - 3)

Discharge :

1. Urethral discharge / Cervical discharge
2. Vagical discharge

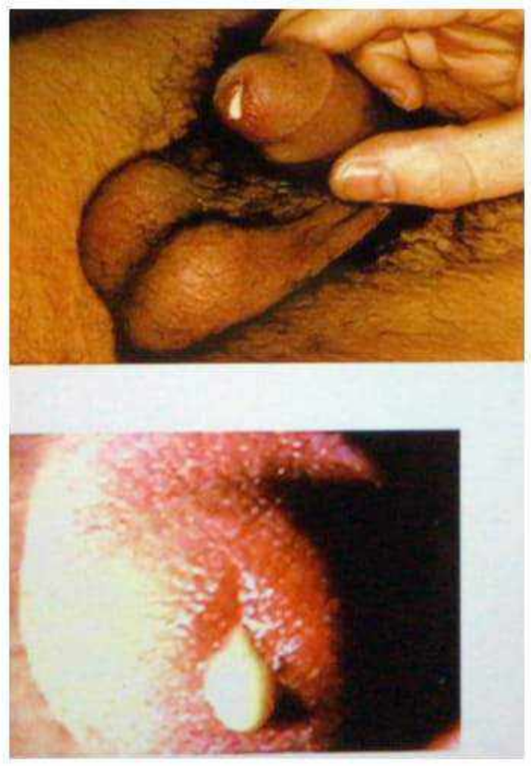
Causes of Discharge :

1. Urethral discharge / Cervical discharge
   - STD Causes
     - Gonococcal
     - Non-Gonococcal
       - Chlamydia
       - Non-Chlamydia
   - Non-STD Causes

GONOCOCCAL URETHRITIS / CERVICITIS

02:20

- Gonorrhoea → flow of seed
- Organism : Neisseria gonorrhoea
- I.P : 2-5 days (1-14 days)



Symptoms :

- Constitutional symptoms
- Burning micturition

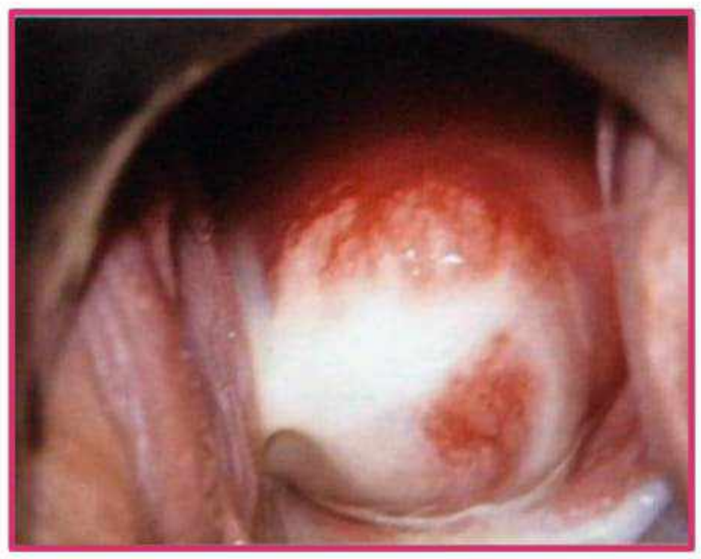
- Increased frequency
- Increased urgency.

Clinical features :

- Thick purulent discharge
- Copious
- Perimeatal erythema.

Gonococcal cervicitis :

- constitutional symptoms ⊕
- urinary symptoms ⊕
- Discharge scantay, purulent, thick
- On P/s :
  - Redness / erythema ⊕ of cervix.
  - Bleeds to touch
  - friable cervix.



Complications

- Males → i) Balanoposthitis
  ii) Posterior urethritis
  iii) Inflammation of gland
  iv) Vesiculitis

- Females → i) Salpingitis

  ii) PID → Infertility

  iii) Bartholin gland abscess.

Disseminated Gonococcal Infection : (Systemic complication)

- Acute Arthritis Dermatitis Syndrome

  involves :

  - wrist
  - MCP
  - Knees
  - Ankles

  - Extremities → Pustules ⊕

Investigation :

1. Gram's Stain :

- Gram ⊖ Diplococci
- Kidney shaped
- Intracytoplasmic, Extracytoplasmic
  ↓
  within PMN's
- urethritis → Diagnosed if >5 PMN's /HPF

active space

2. Culture : Thayer Martin Medium

- Most definitive investigation.

3. NAAT

Treatment :

- Ceftriaxone 250mg i.m stat + Azithromycin 1g orally stat
- **Alternative :**
  - Oral Cefixime 400mg + Azithromycin 1g } Stat single dose.
- **Treatment of DGI :**
  - Ceftriaxone 1gm i.m i.v every 24 hrs x 7days

    +

    Azithromycin 1gm.

NON-GONOCOCCAL

14:30

Etiology :

- Chlamydia trachomatis (D-K) ⇒ (MC)
- Mycoplasma
- Ureaplasma

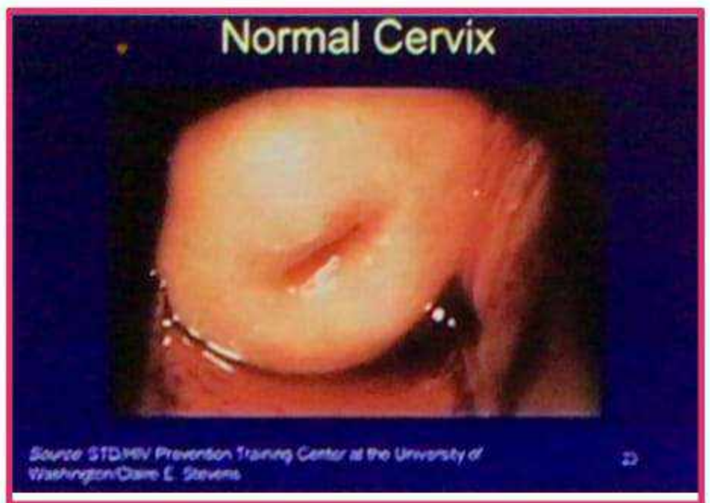
active space

- Adenovirus
- HSV
- Trichomonas vaginalis.

- I.P → 3-5 days.
- Discharge :
  - Scanty, watery / mucoid, clear discharge, serous
  - Constitutional symptoms ⟶ Absent
  - may be asymptomatic.

In females

- Discharge : Scanty, mucoid
- On P/S : Redness of cervix + Friable cervix



Complications

- PID → (MC) cause of 2° Infertility in females.
  - Chlamydia
- Conjunctivitis
- Ophthalmia neonatorum

active space

- Arthritis
- Fitz Hugh Curtis Syndrome : Perihepatitis

Investigations :

1. Gram's Stain : >5 PMN's/HPF (No diplococci)
2. Giemsa : Inclusion bodies
3. NAAT
4. CFT

Treatment :

- Azithromycin 1g stat
- Alternative : Doxycycline

VAGINAL DISCHARGE

20:30

- (MC) Physiological
- Pathological causes :
  - i) Candidiasis
  - ii) Trichomonal vaginitis
  - iii) Bacterial vaginosis. → (MC)

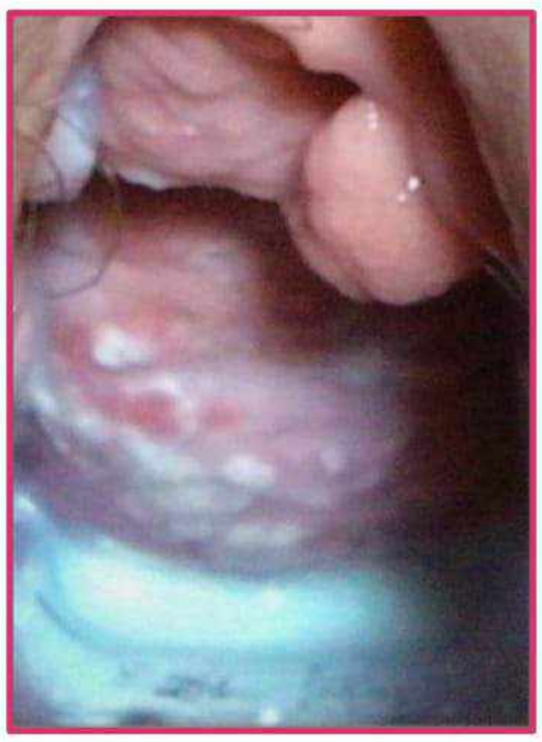
active space

Candidal Vaginitis :

- caused by
  - Candida albicans (MC)
  - Candida krussei
  - Candida glabrata.
- Predisposing factors :
  - i) DM
  - ii) Pregnancy
  - iii) Immunocompromised.

Presentation :

- Severe pruritus
- White cheesy creamy discharge
- Copious discharge
- Pre-menstrual flare
- Adherent to vaginal walls.



Investigation

1. KOH → Budding yeasts, Hyphae (+)

Treatment :

- Azoles → DOC

Topical

- Creams
- Vaginal
- Pessary

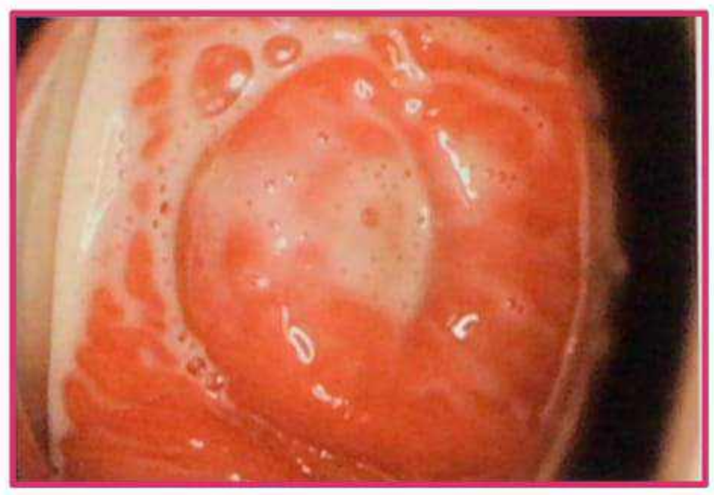
Systemic

- Fluconazole 150mg stat
- For Recurrent: Fluconazole 150mg on Day 1, 3, 5

TRICHOMONAL VAGINITIS

26:00

- caused by Trichomonas vaginalis (Flagellate Protozoa)
- pH > 4.5
- Complications in Pregnancy :
  - i) Premature labor
  - ii) LBW



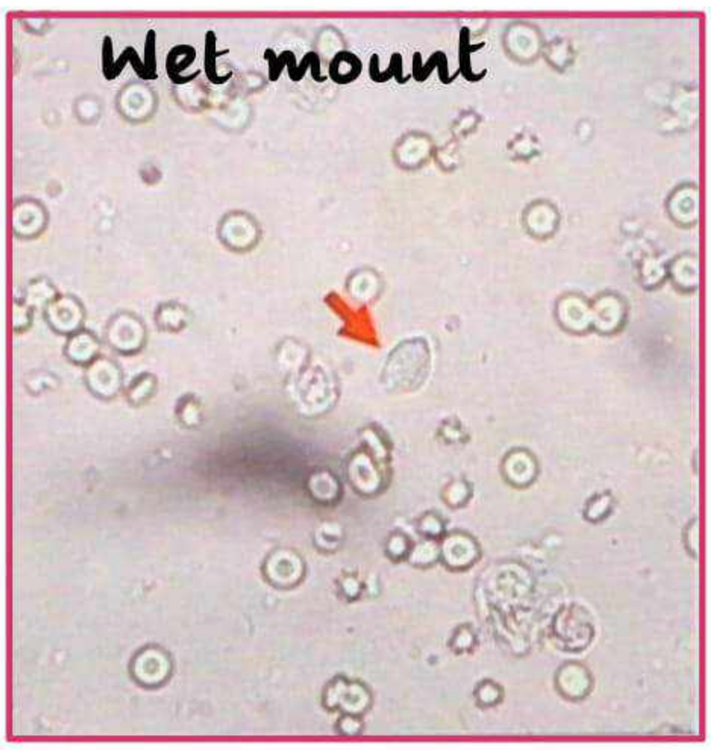
Clinical feature :

- Greenish frothy / foamy discharge
- Severe pruritus
- Strawberry cervix.

Investigations

1. Wet mount : Motile flagellates

2. NAAT



Treatment :

- Secnidazole 2g stat
- Tinidazole 2g stat

BACTERIAL VAGINOSIS

30:00

- (MC) cause of Pathological vaginal discharge
- Altered flora
- ↑ Anaerobes > Lactobacilli
  - i) Gardenella vaginalis
  - ii) Mycoplasma hominis
  - iii) Ureaplasma.
- No partner treatment required

AMSEL'S CRITERIA

1. Discharge : Greyish odoriferous, scanty discharge, watery, fishy odour, homogenous
2. pH > 4.5

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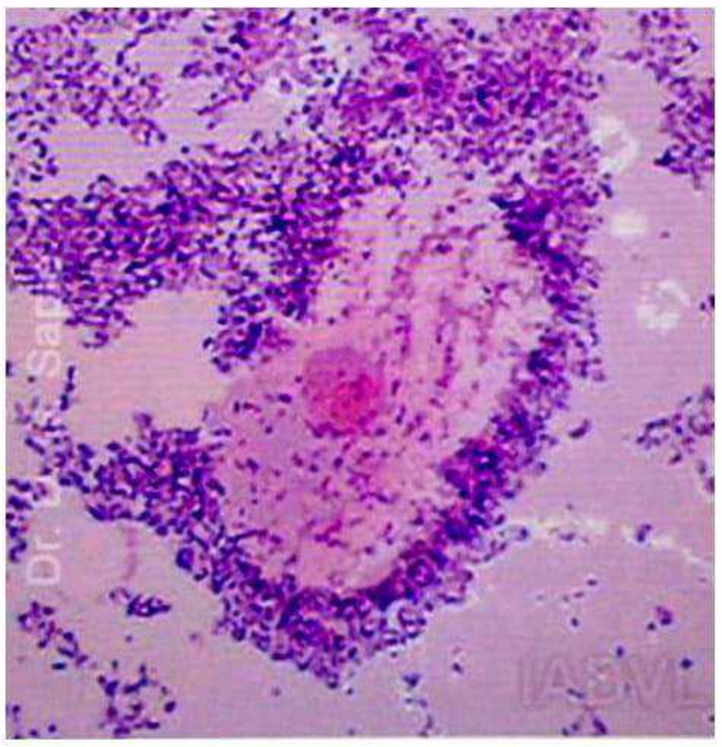
3. 10% KOH on the discharge: Fishy odour
"WHIFF TEST"
↳ due to production of amines

4. Clue cells : > 20%



Clue cell :

- Vaginal squamous cell which is studded with anaerobe at the periphery.
- Stippled appearance.
- Clue cells > 20% in number.

Treatment :

- Metronidazole 500mg TDS x 7days.

ANOGENITAL WART

37:05

- caused by HPV.
- 2 Types : i) Low Risk → 6, 11
  ii) High Risk → 16, 18
- Sites : Sites of Trauma
  - In Males → Frenulum sulcus
  - In Females → Posterior fourchette.

active space

Presentation :

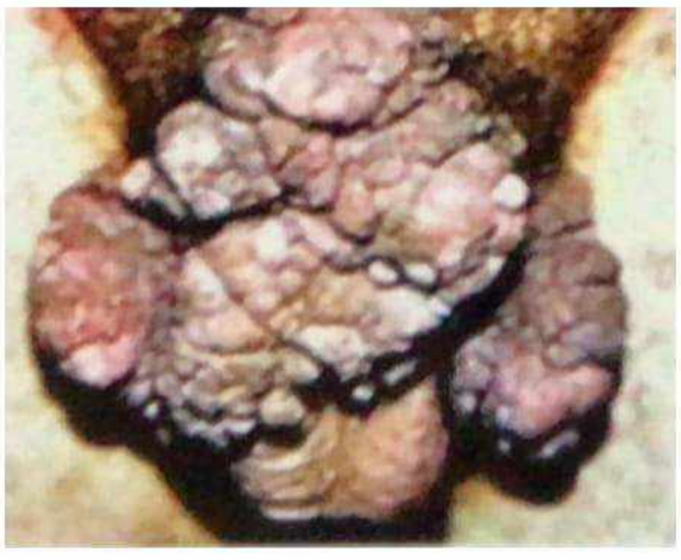
- Condyloma acuminata

  (flat based → pointed)

- Buschke lowenstein tumor.

  ↳ Giant condyloma acuminata.

  carcinomatous changes ⊕



Treatment :

i) Patient →
- Podofilox
- Imiquimod
- Sinectachinins.

ii) Provider administered :

- Chemical → i) TCA
  ii) Podophyllotoxin
- Physical → i) Cryotherapy
  ii) R/F
  iii) Electrocautery.

- In Pregnant female / mucosal involvement / Intraurethral lesions ↘
  Cryotherapy, TCA ↙

Other STD's :

i) Genital Scabies

ii) Molluscum contagiosum.

SYNDROMIC MANAGEMENT

43:00

- Treating it as

i) Syndrome

ii) Genital ulcer

iii) Discharge → urethral
                → vaginal

Clinical Condition	Kit to be prescribed	Drugs included	Image
Urethral or Anorectal or Cervical discharge	KIT 1: Gray	Tab Azithromycin 1 g (1 tab) Tab Cefixime 400 mg (1 tab)	
Vaginal Discharge (Vaginitis)	KIT 2: Green	Tab Secnidazole 2 g (1 tab) Tab Fluconazole 150 mg (1 tab)	
Genital Ulcer Disease (Non Herpitic)	KIT 3: White	Inj. Benzathine Penicillin 2.4 MU (1 vial) + Tab Azithromycin 1 g (Kit also contains 10 ml disposable syringe+ 21 gauge needle + 1 vial of 10 ml sterile water	
Genital ulcer disease (nonherpetic) in patient allergic to penicillin	KIT 4: Blue	Tab Doxycycline 100 mg (1 tab BD for 14 days) Tab Azithromycin 1 g x 1 tab	
Genital ulcer disease (Herpetic)	KIT 5: Red	Tab Acyclovir 400 mg x 1 tab TDS x 7 days	
Lower abdominal pain (Pelvic Inflammatory Disease)	KIT 6: Yellow	Tab Cefixime 400 mg x 1 tab Tab Metronidazole 400 mg x (1 BD 14 days) Tab Doxycycline 1 g (1 BD 14 days)	
Inguinal Bubo	KIT 6: Black	Tab Doxycycline 100 mg (1 BD x 21 days) Tab Azithromycin 1 g x 1 tab	

Great Girls won't Buy

Red and Yellow Bags

active space

Kits for :

1. Gray Kit :

- used for

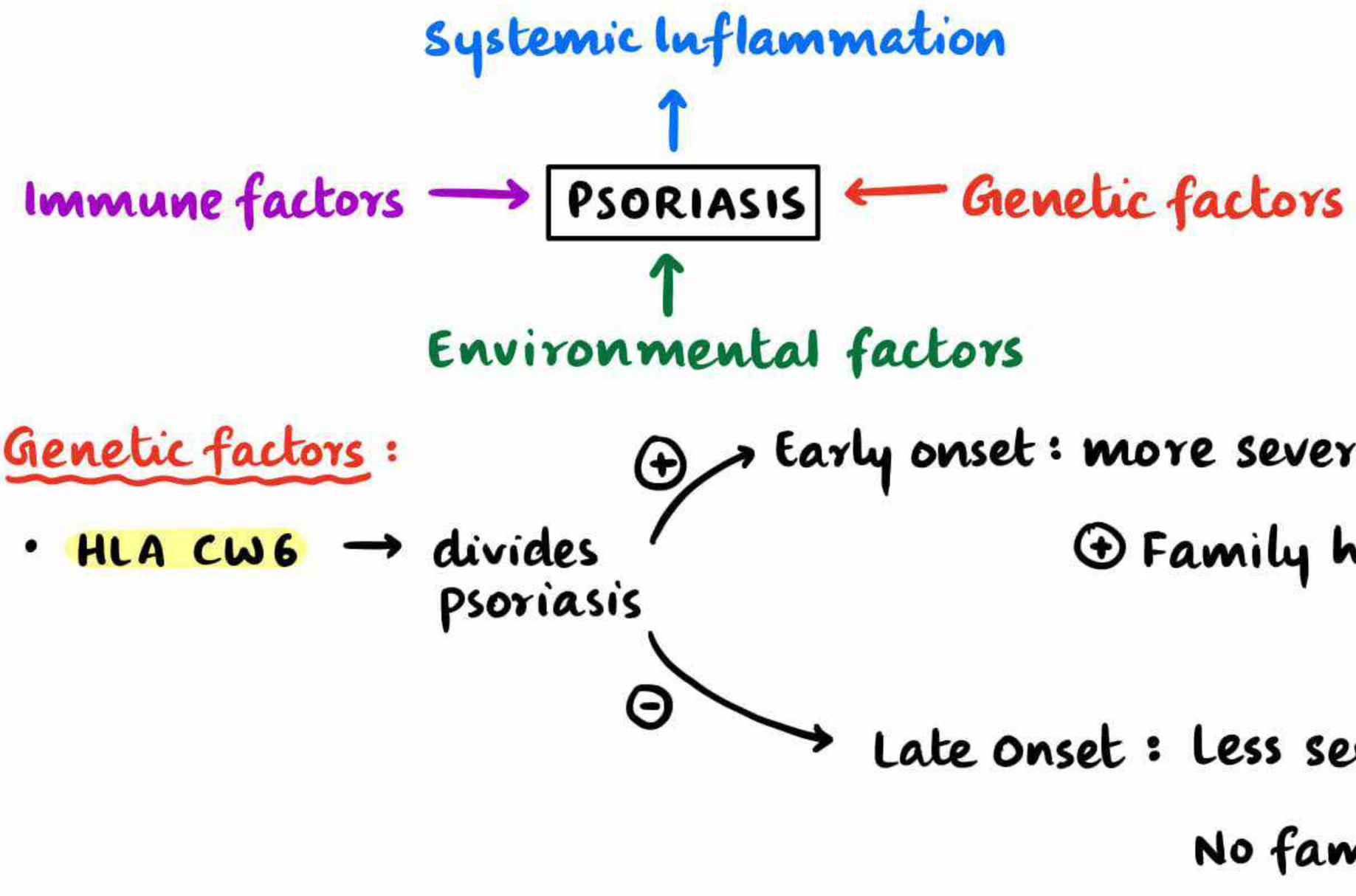
  - Gonorrhoea ⟶ urethral discharge
  - Trachomatis

2. Green Kit : used for vaginal discharge
3. White Kit : used for chancre / chancroid
4. Blue Kit : used for Genital ulcer allergic to penicillin
5. Red Kit : for Herpes
6. Yellow Kit : for lower abdominal pain , PID , CMD. (for females)
7. Black Kit : (for males) ⟶ used for Bubo.

active space

Psoriasis (Part - 1)

- It is a papulosquamous disorder (papule/plaque ⊕)
- Complex
- Multifactorial
- Chronic
- Recurrent
- Predilection for winter



- HLA B27 ⟶ ↑ Risk of Psoriatic arthritis
- PSORS - 1.

Environmental factors : Aggravating factors

- Infection
  - Streptococcal ⟶
    - leads to Guttate Psoriasis.
    - ASLO titre , Throat swab ⊕
    - Antibiotics R_x
  - HIV ⟶ More severe.

Medications known to aggravate psoriasis : (BAT NAILS)

- β Blockers
- ACE inhibitors
- TNF α Inhibitors
- NSAID's
- Anti-malarials
- INF - γ
- Lithium
- Steroids.

active space

Other aggravating factors :

- Alcohol
- Cigarette
- Stress
- Sunlight → usually beneficial
- Trauma. → Koebner's phenomenon.

PATHOGENESIS

08:40

1. Hyperproliferation of Keratinocytes -

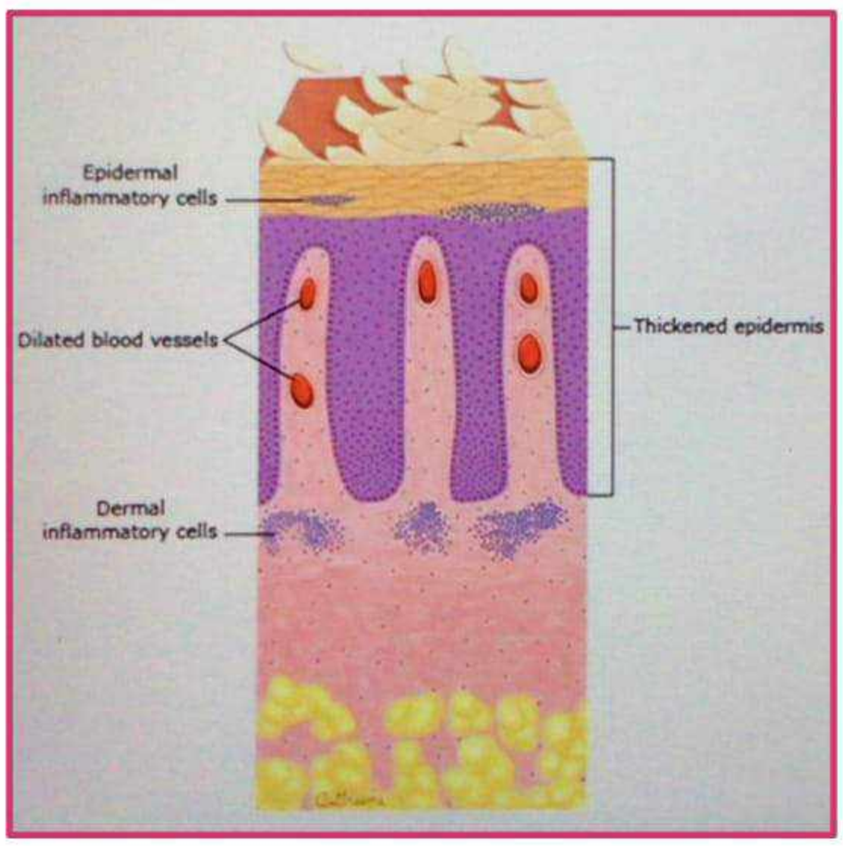
- Epidermal Turn Over Time

↓

Reduced from 30days to 14days

⇓

Scaling



2. Increased Angiogenesis.

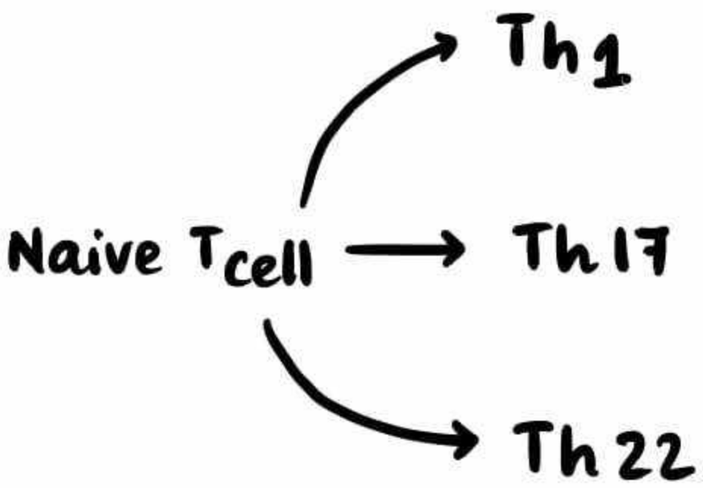
- Increased Blood supply and vessel formation (Dilated)

⇓

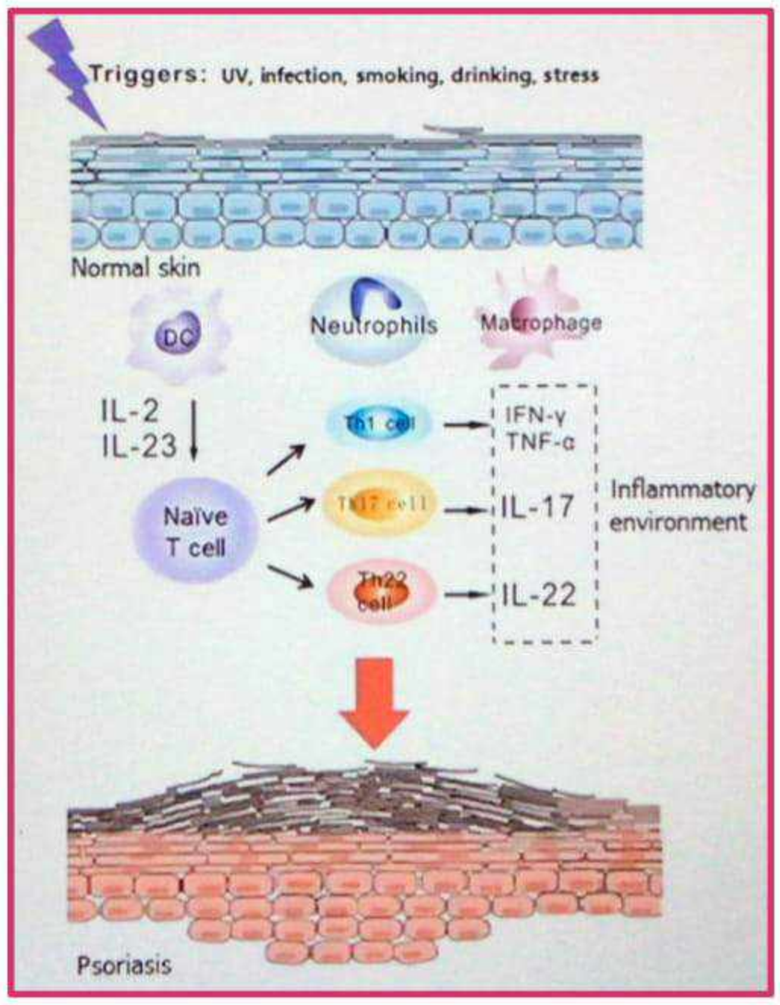
Erythema

3. Inflammatory mediators

- changes start in dermis



No Th2 Response

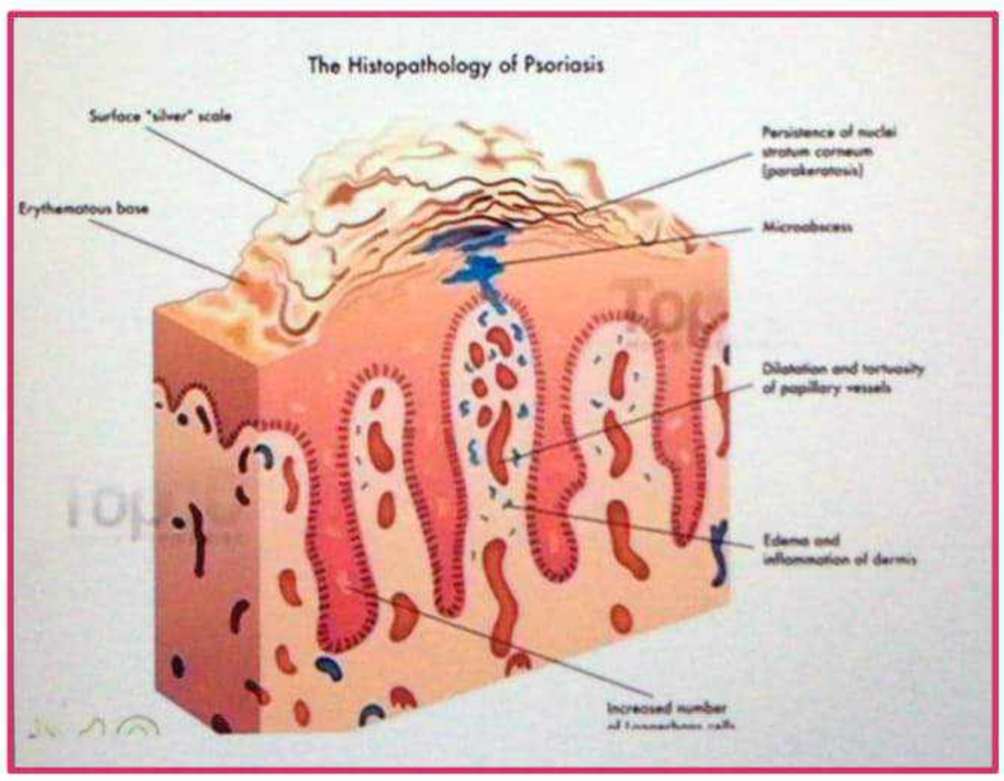


On Histopathology :

- dermal changes precede epidermal changes

1. Stratum corneum :

- Hyperkeratosis ⊕
- Parakeratosis ⊕
- Munro microabscesses
  - ↳ Collection of neutrophils



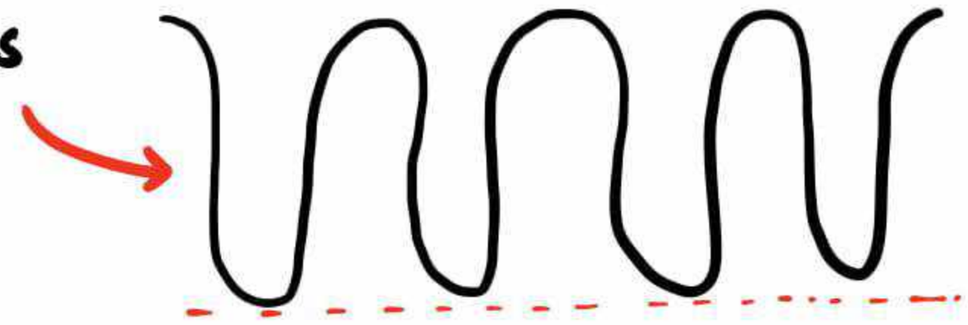
2. Stratum granulosum :

- Absent
- Hypogranulosis / Agranulosis.

active space

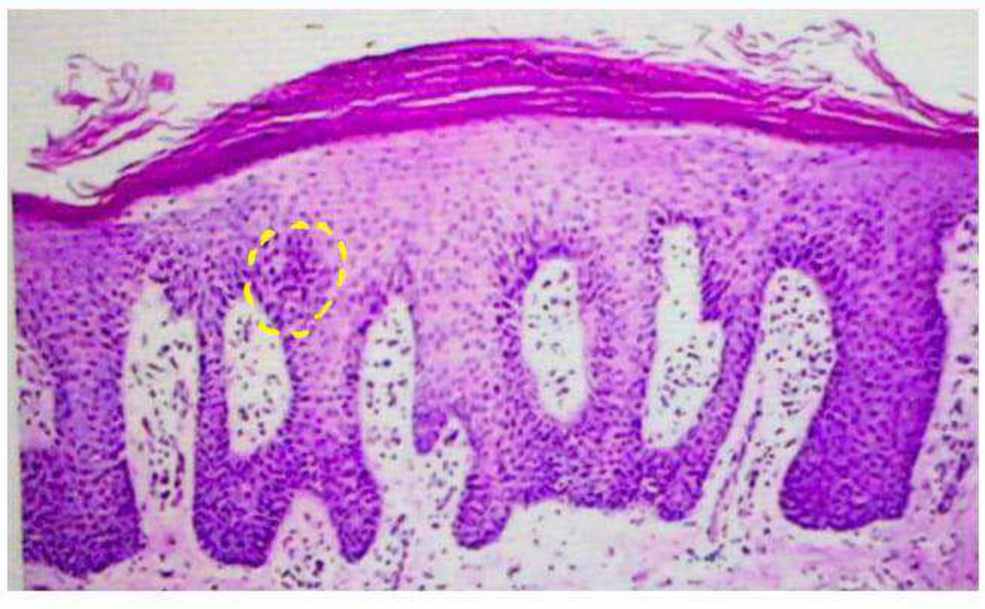
3. In Malphigian layer :

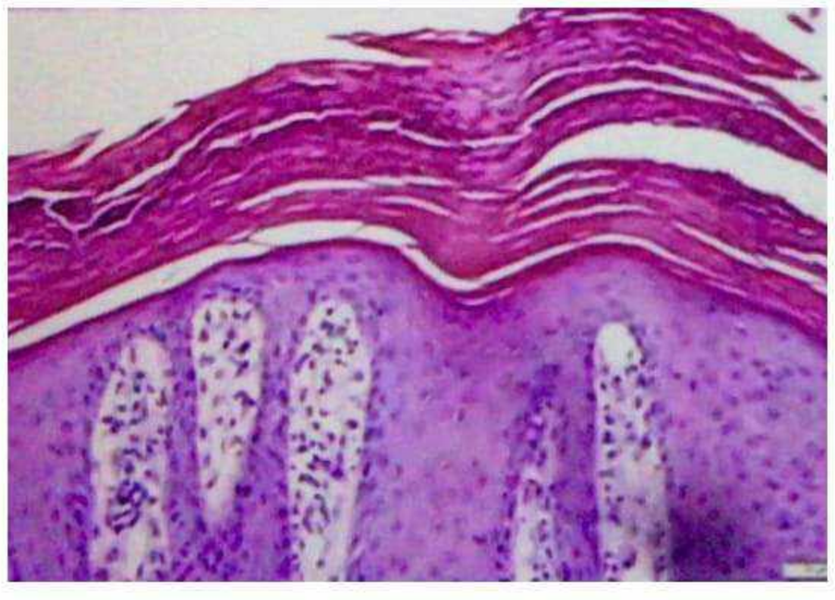
- Acanthosis (Thickening of SS)
- Spongiform pustules of Kogoj (collection of neutrophils)
  - ↳ seen in Stratum Spinosum.
- Regular elongation of Rete ridges
  (Club shaped Rete Ridges)



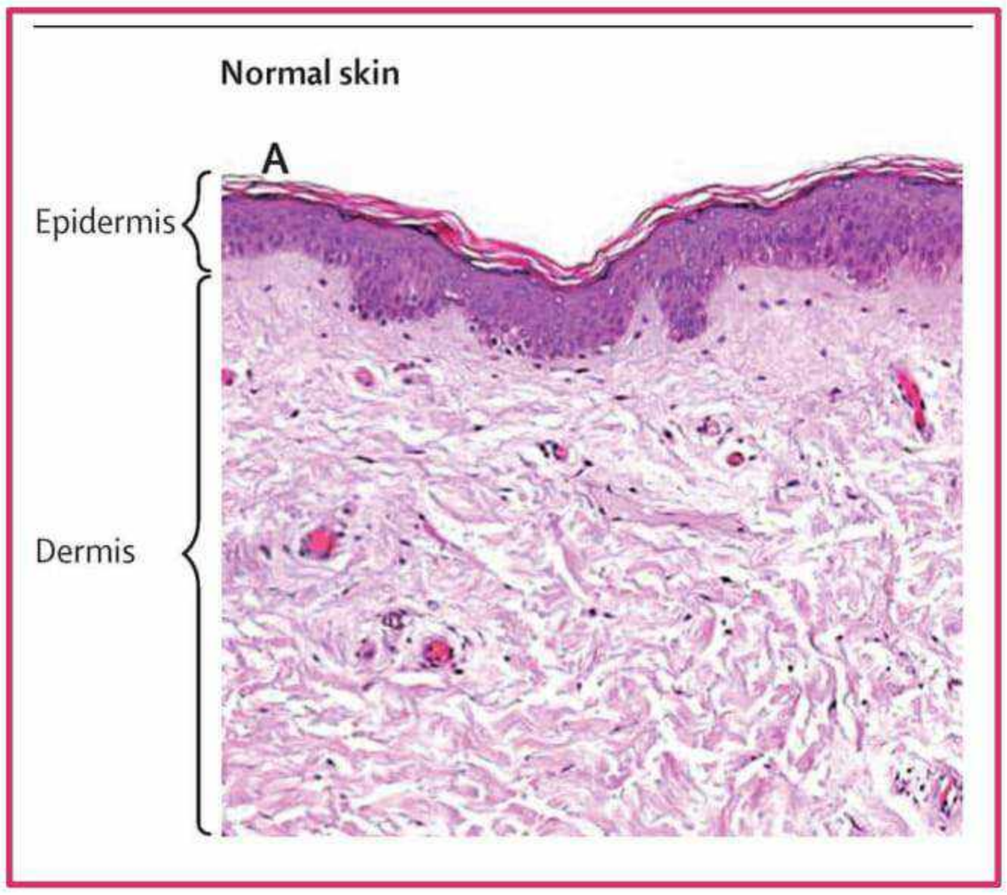
4. In Dermis :

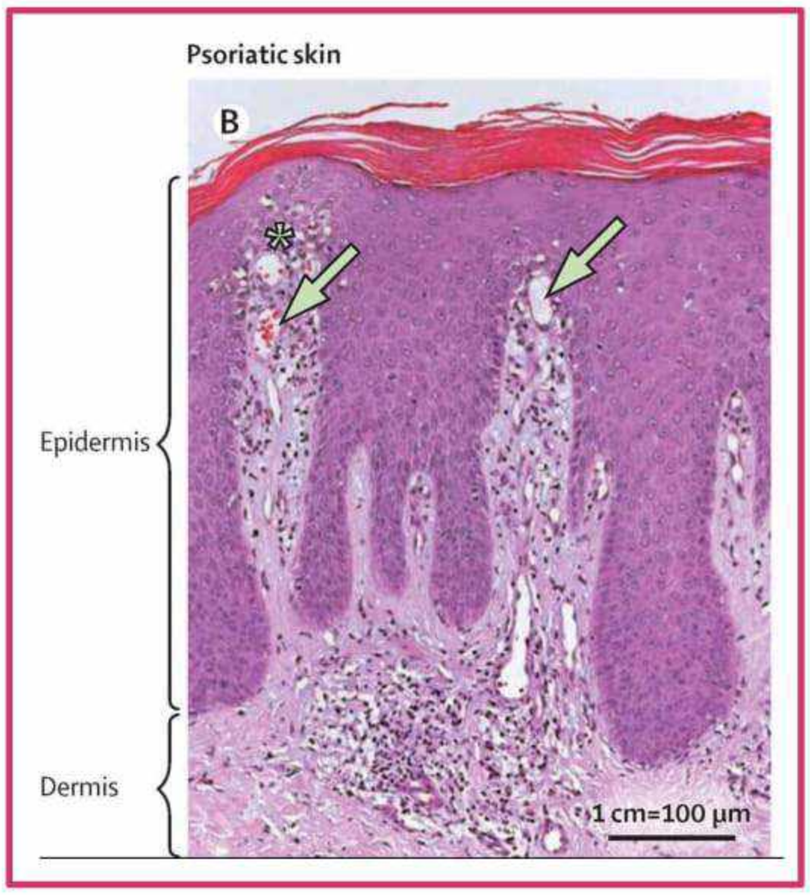
- Dilation of Blood vessels
- Inflammatory cells
- Suprapapillary thickening.





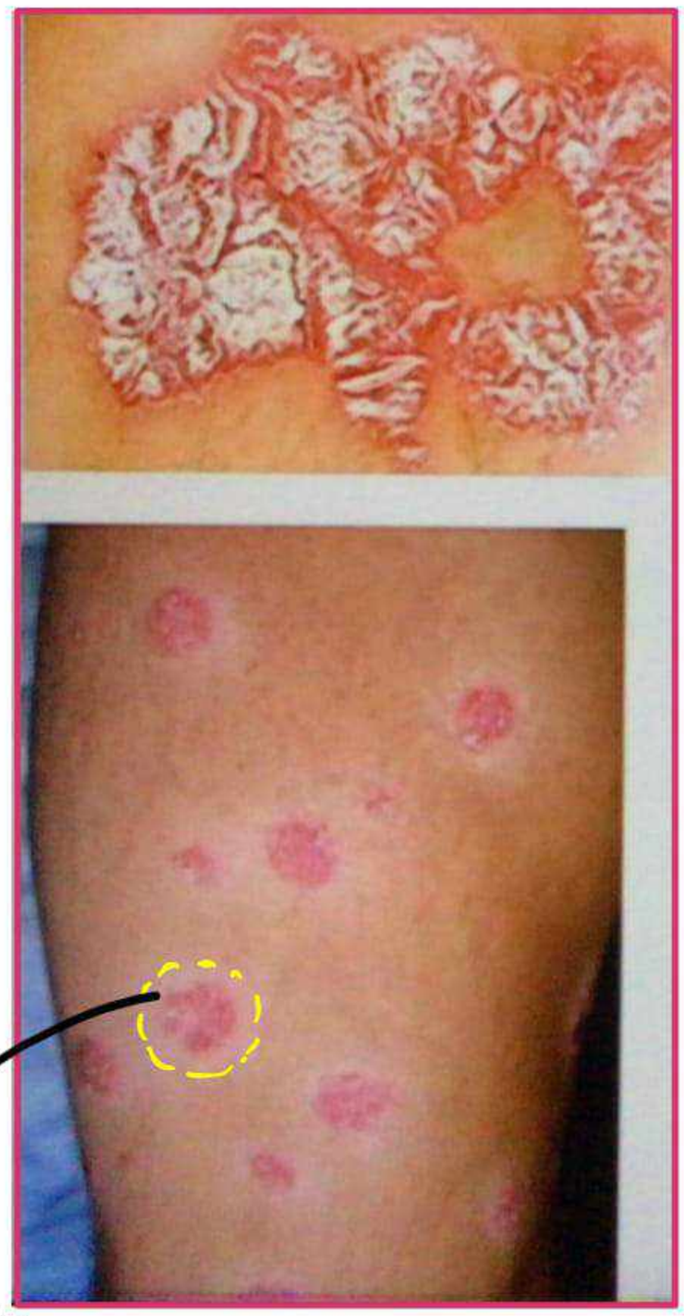
active space





Clinical features :

- No itching (usually)
- Winter excerbation
- Sites : It can involve any area of body (Predominantly Extensors)
- Symmetrical lesions
- Colour : Salmon Pink
- Erythematous plaque → Well defined
- Scaling → Silvery white
- Induration
- Ring of Wornoff : pallor around a psoriatic lesion.



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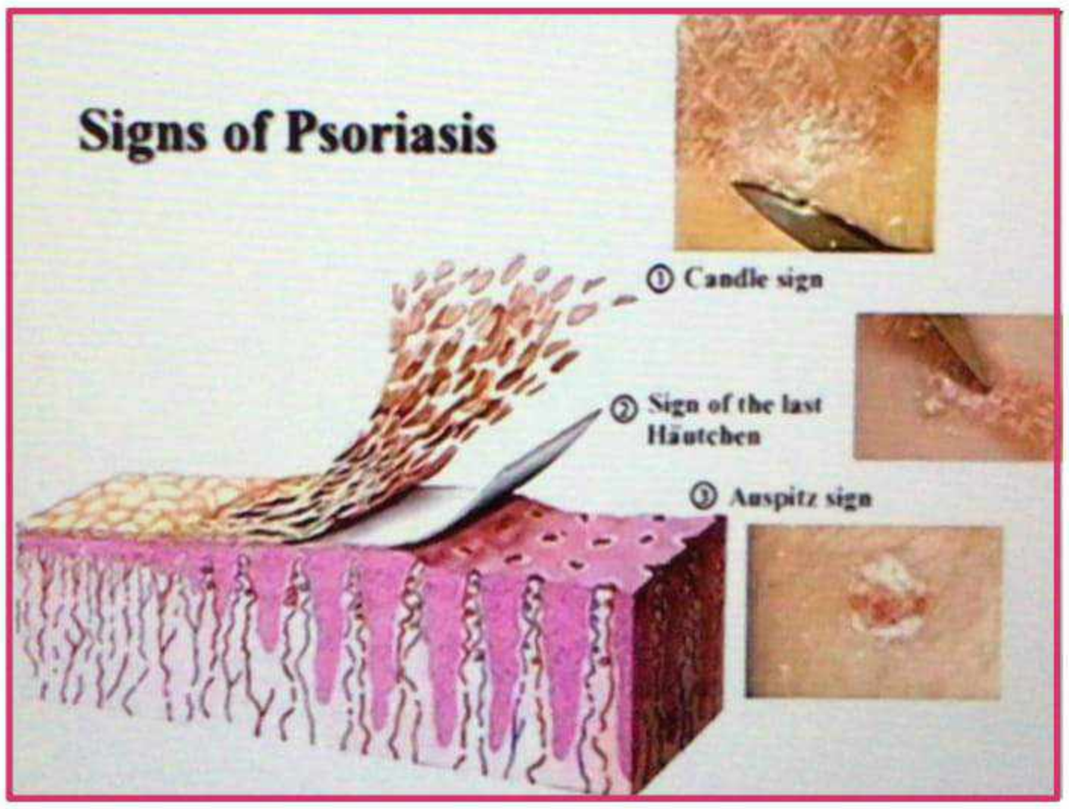
- It is due to inhibition of Prostaglandin synthesis (PGE_2)



Signs of Psoriasis :

- On scraping a lesion of Psoriasis with glass slide

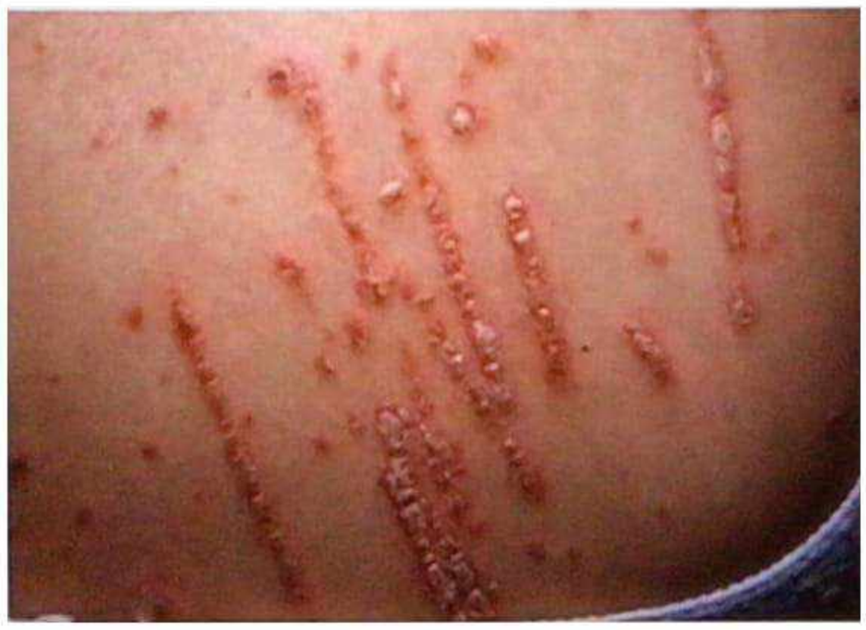
1. Accentuation of scaling (Grattage Test, Candle sign)
2. Shiny membrane (Berkeley's membrane)
3. Pinpoint bleeding points → Dermal vessel vasodilation

} AUSPITZ SIGN.

→ suprapapillary thinning.

active space

Koebner's phenomenon ⊕



- appearance of lesion along the lines of trauma
- a.k.a **Isomorphic phenomenon.**
- usually seen in 10-14 days of trauma.
- All or none phenomenon.

- **3 Types :**

i) True :
- Psoriasis
- Vitiligo
- Lichen planus

ii) Pseudo -
- Warts
- Molluscum contagiosum

iii) Rare -
- Darrier's
- Erythema multiforme.

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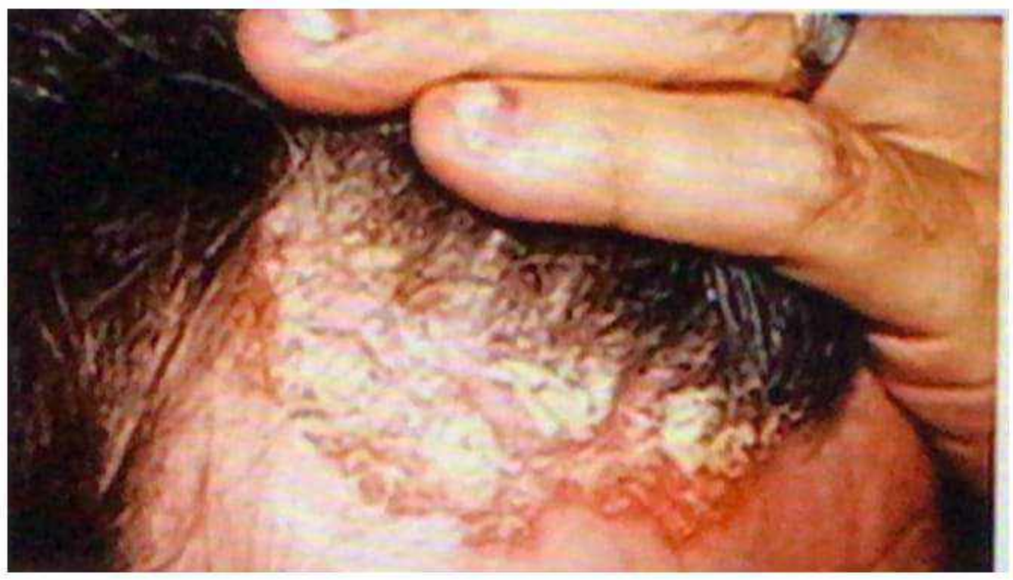
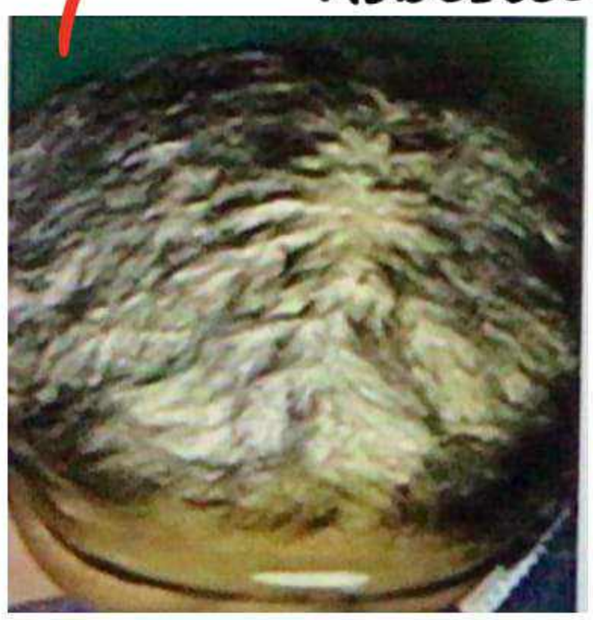
Variants of Psoriasis

1. Scalp Psoriasis :





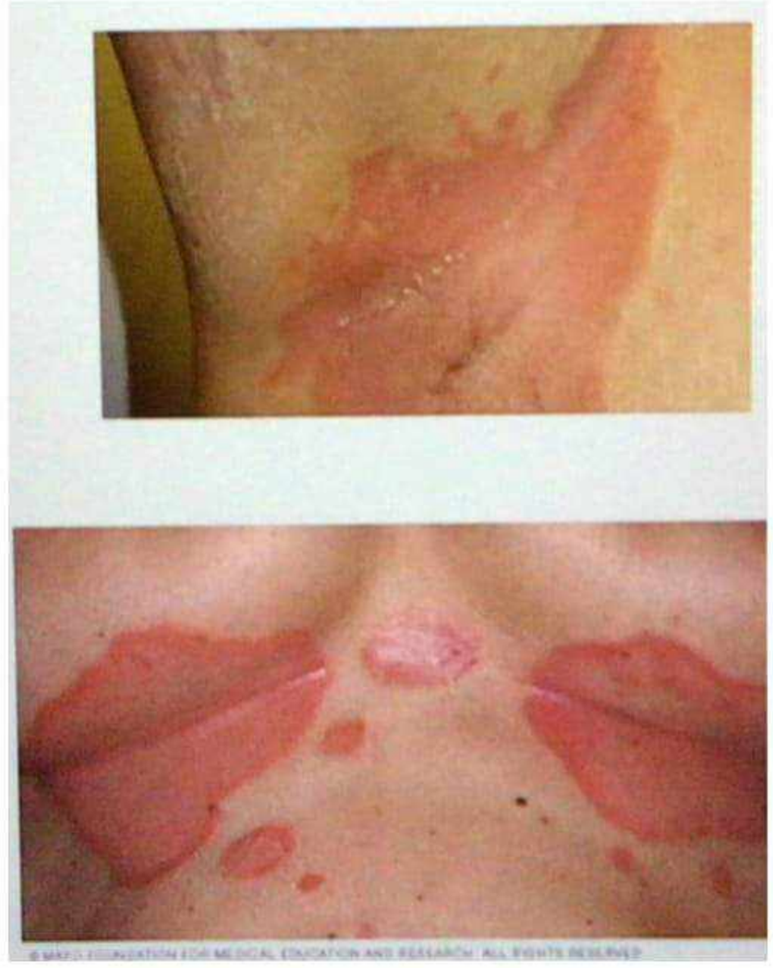
→ Pityriasis amiantacea

- Asbestos like scaling.

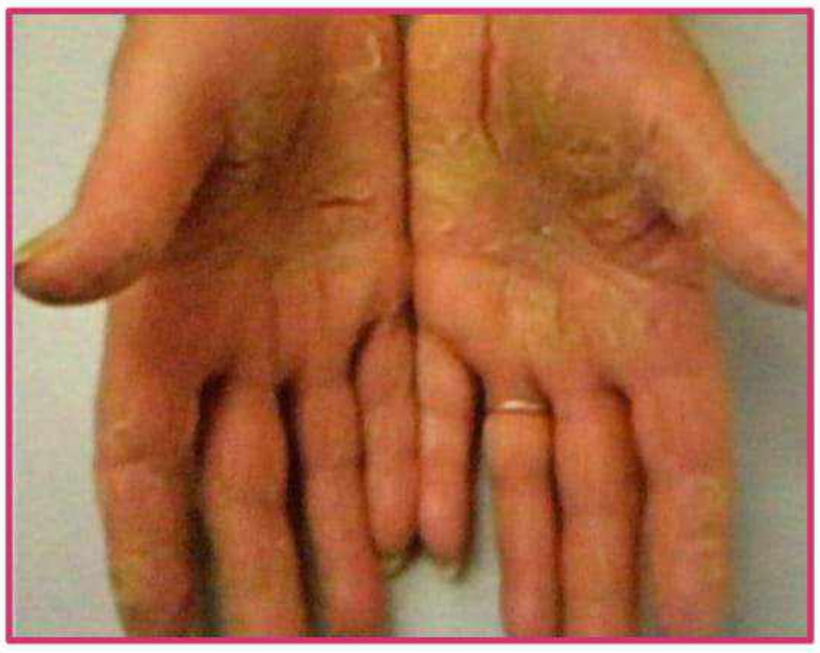
- crosses the hairline
- Isolated / with other lesions of psoriasis in body.
- usually non-scarring (does not cause alopecia)
- vs. Seborrheic dermatitis (Itchy)

2. Flexural Psoriasis.

- seen in axilla, groins, umbilicus, inframammary, gluteal cleft
- a.k.a Inverse Psoriasis.
- No scaling
- No/Mild induration
- vs. Intertrigo → • Itchy
  - B/L symmetrical.



3. Palmoplantar psoriasis

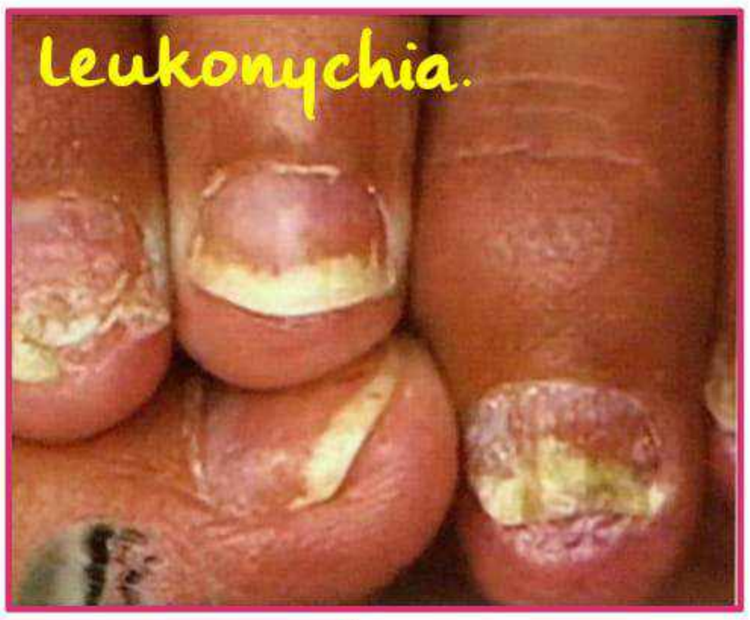


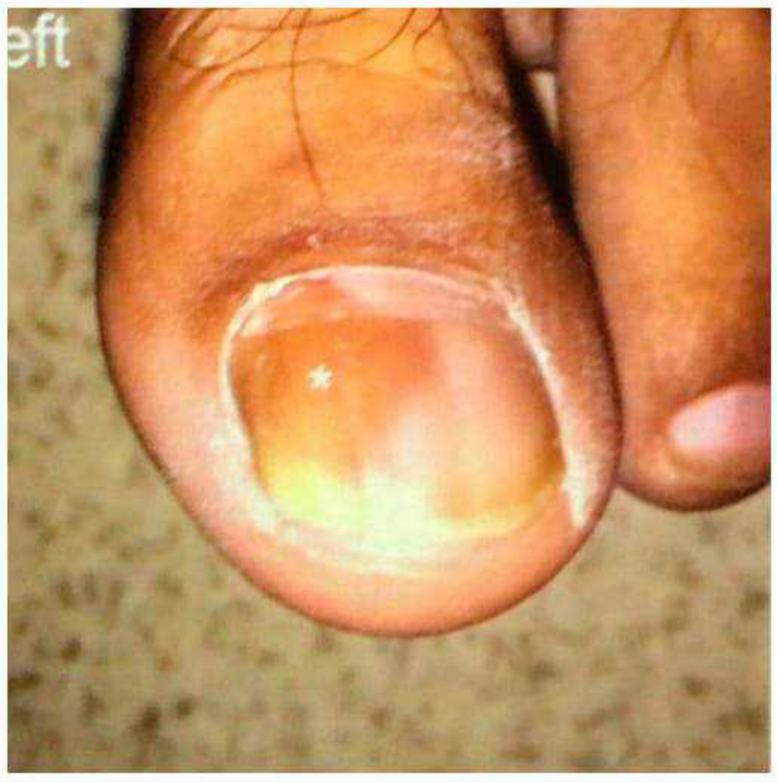
- usually extends beyond the margins
- Psoriatic lesions on knuckle
- Not itchy.
- More of fissuring
- Instep involvement
- Nail changes ⊕

4. Nail Psoriasis :

- Association
  - ↑ severity
  - ⊕ Family h/o
  - ↑ Arthritis
- Involvement of Nail plate : i) Pitting → (MC)
  ii) Ridging
  iii) Leukonychia.
- Involvement of nail bed : i) Oil drop sign
  ii) Subungual Hyperkeratosis
  iii) Dilated vessels.
  iv) Onycholysis (Distal)

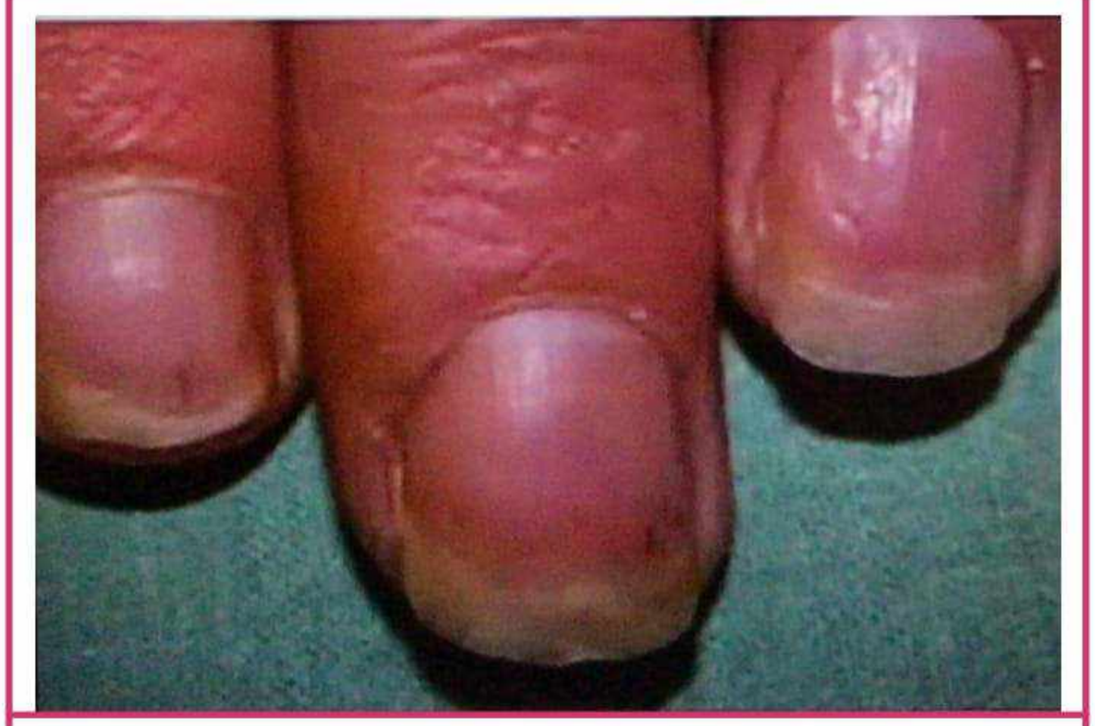
- **Pitting :**
  - depression of nail plate
  - Irregularly placed uniform in size.





Oil drop sign / Salmon Patch :

- Most specific sign
- Parakeratosis in nail bed.



Dilated capillaries.

active space

Onychomycosis	Psoriasis
more toenails	more fingernails
usually single nail	Multiple nails
⊗	Pitting ⊕
⊗	Oil drop sign ⊕
KOH ⊕	⊗

PSORIATIC ARTHRITIS 51:50

- Seronegative
- associated with HLA B-27
- associated with nail psoriasis (severe disease)

Symptoms:

- Edema / joint swelling
- Morning stiffness.

Moll and Wright Classification:

1. Asymmetric poly-oligoarthritis
   - DIP ⇒ (MC) joint involved.
2. Symmetric polyarthritis (RA like)
3. Arthritis mutilans

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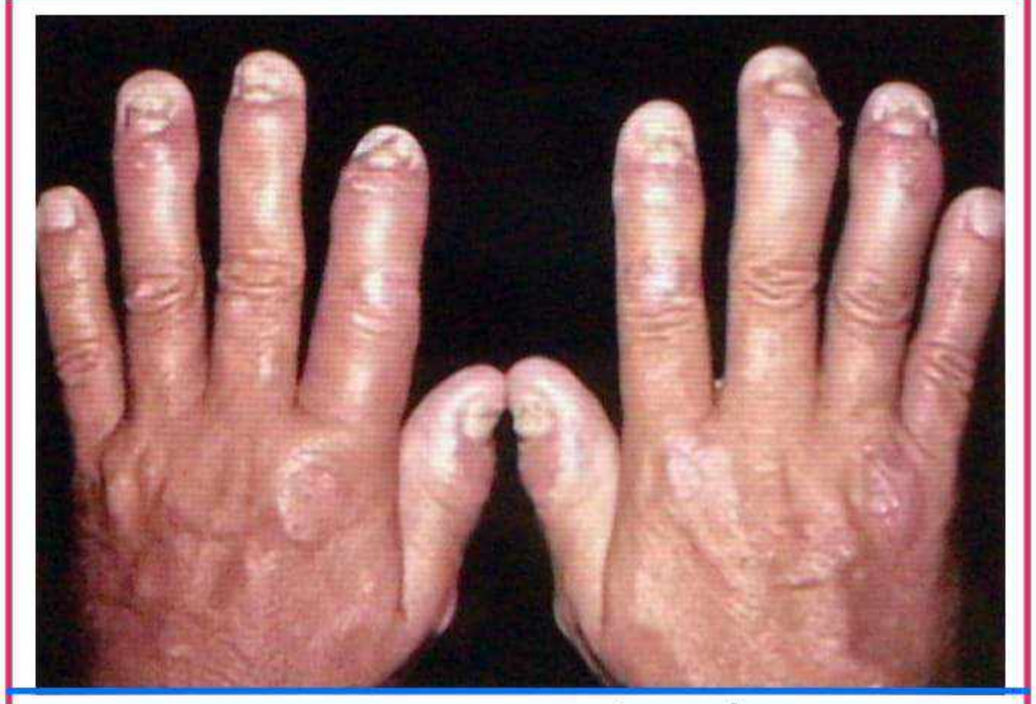
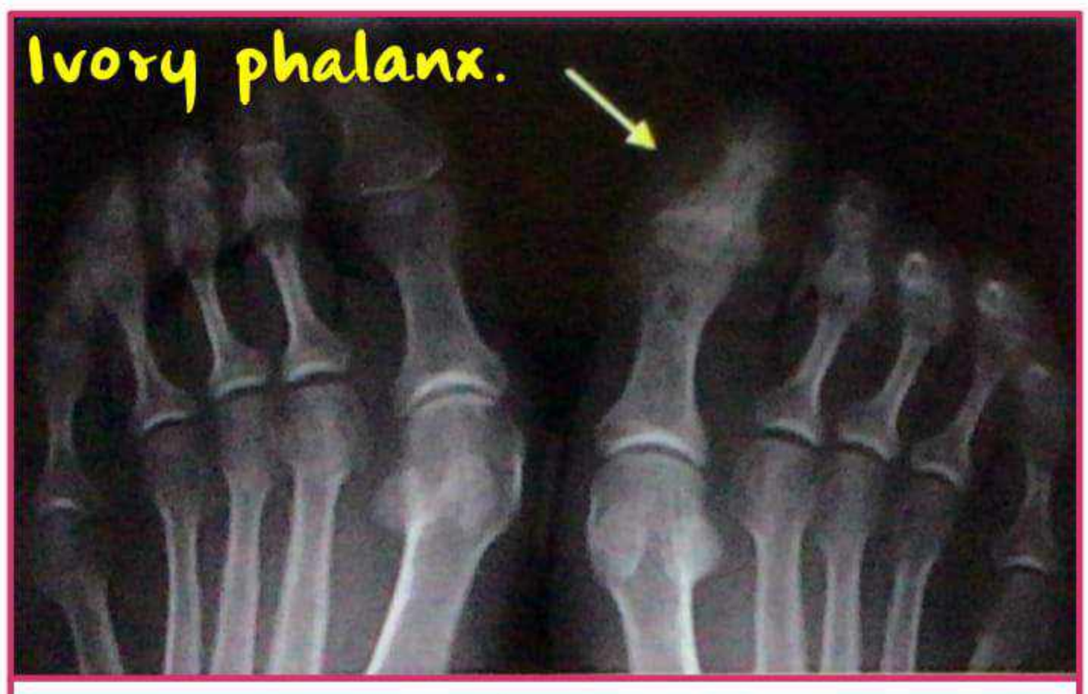
4. Axial involvement : Spondylosis, Sacro iliac involvement

5. Isolated DIP

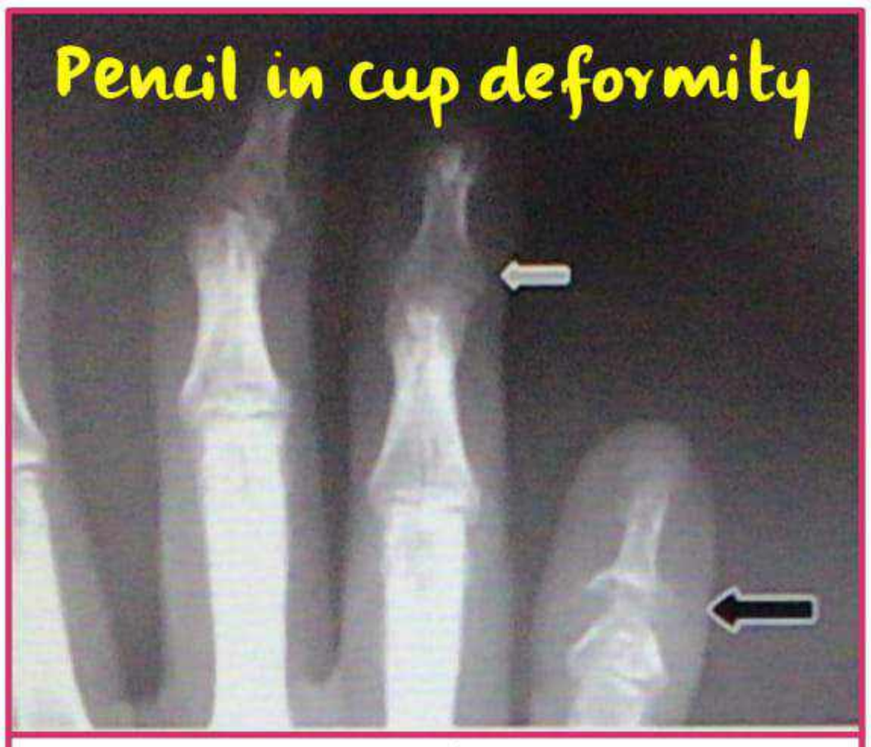


Sausage digits : due to dactylitis.

On X-Ray :



↑ Bone formation



Erosion osteolysis.

- Mucosa is not involved
- Geographic tongue ⊕
- Ocular involvement ⊕ → uveitis → also associated psoriatic arthritis.

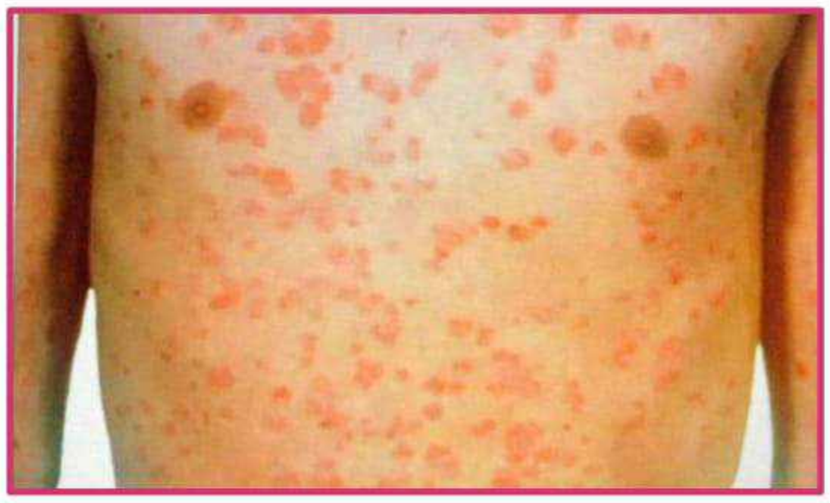
active space

Psoriasis (Part - 2)

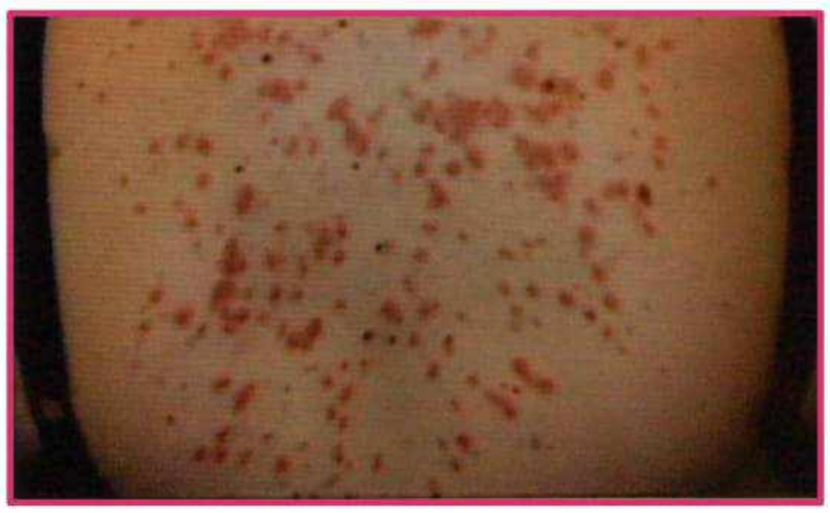
GUTTATE PSORIASIS

00:35

- Sudden crops of psoriatic lesion over body predominantly on trunk.



- seen in
  - children
  - Preceeding h/o Streptococcal infection
  - Trunk.
  - Rain drop appearance.



- Smaller and less scaly.

Investigation :

- ASLO
- Throat swab.

Treatment :

- **Antibiotics :** Erythromycin, Amoxicillin.
- **In Recurrent cases :** Tonsillectomy can be done.

UNSTABLE PSORIASIS

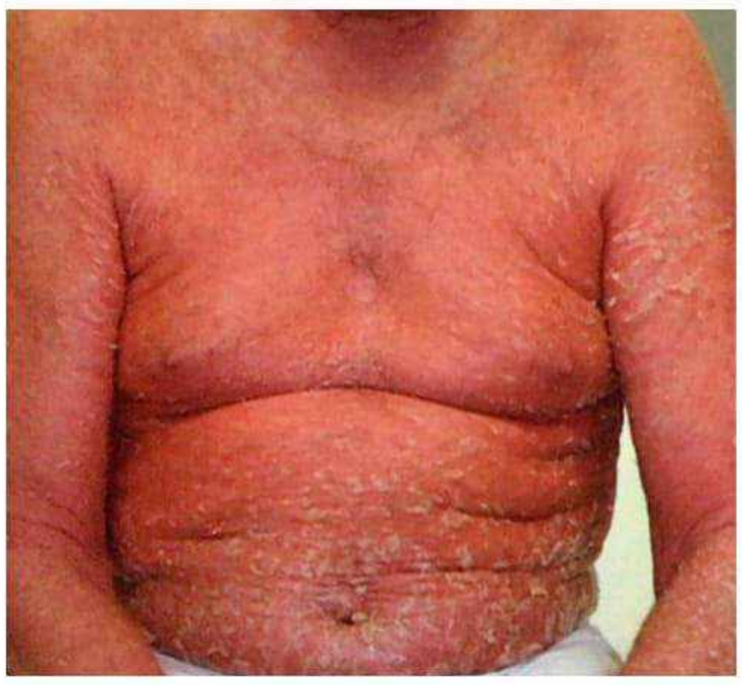
03:45

- Increased redness over existing lesion

Clinical features:

- Pain and tenderness ⊕
- Constitutional symptoms → Fever and Malaise ⊕
- → Chronic Plaque Psoriasis → Unstable psoriasis
  → de novo → Erythrodermic Pustular psoriasis.

Erythrodermic Psoriasis



- Erythro → Erythema and Scaling
  > 90% of BSA.
- Classical Psoriatic lesions
  ↳ less sick ,
  less constitutional symptom.
- It can be Denovo.
- Prone to thermoregulatory failure → circulatory shock.

Management:

- Fluid management
- Temperature monitoring

active space

- Prevent 2° Bacterial infection
- Manage Hypoalbuminemia , hypocalcemia.

PUSTULAR PSORIASIS

08:40

- Presence of visible pustules on skin lesions

Generalised

- Acute Generalised Pustular Psoriasis (von Zumbusch)
- Pustular psoriasis of pregnancy
- Infantile psoriasis
- Juvenile psoriasis
- Circinate psoriasis

Localised

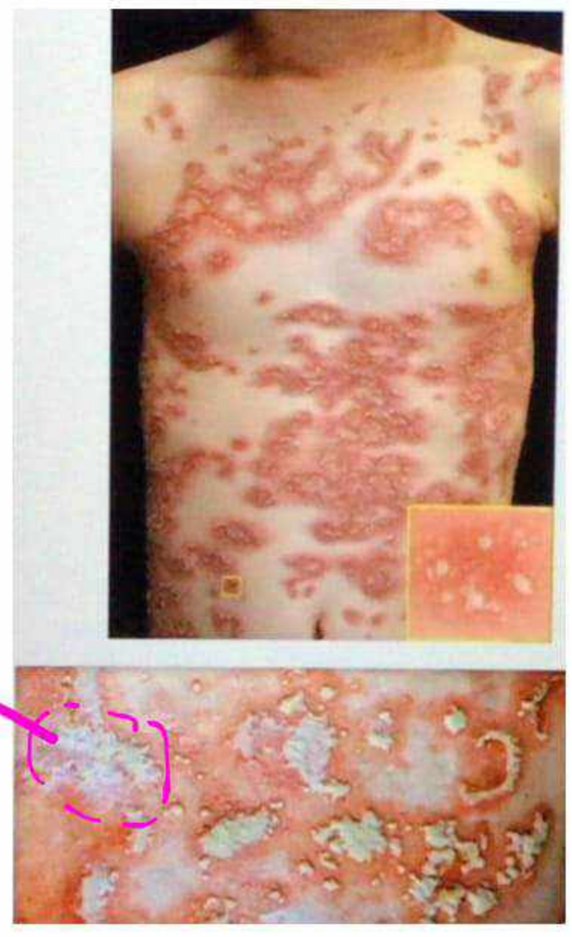
- Palmoplantar pustulosis
- Acrodermatitis of Hallopeau

Aggravating factors :

- Withdrawal of Steroids
- Coal tar application
- Hypocalcemia
- Infections
- Stress.
- Pregnancy

Acute Generalised Pustular Psoriasis

- a.k.a **Von Zumbusch**
- Multiple pustules over an erythematous background.
- Lake of Pus → ⊕
- Sterile pustules.
  - non-infective
  - Neutrophils.



- Patient is usually sick
- Constitutional symptoms ⊕ → Fever, Malaise

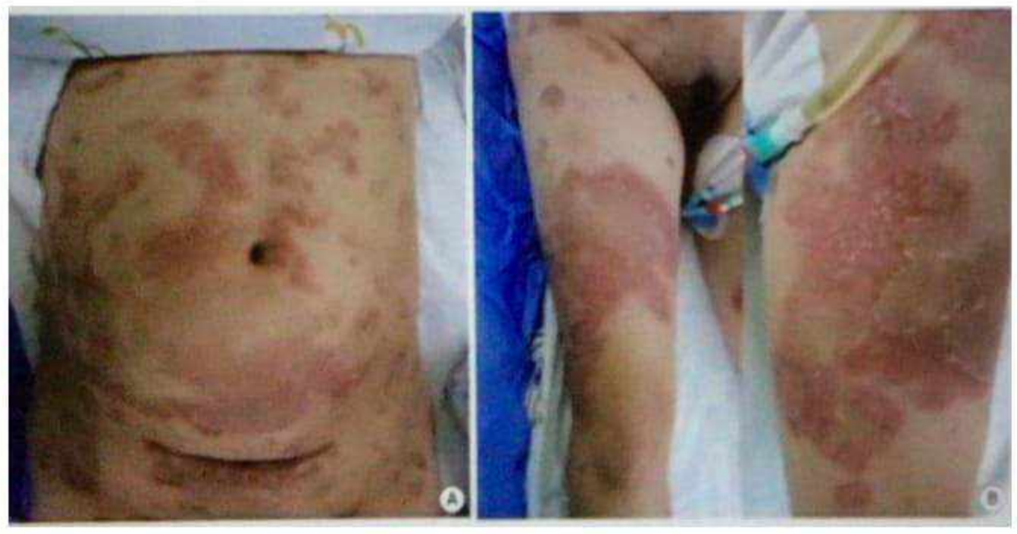
Management :

- Admit the patient
- Look into aggravating factors
- Treat any underlying infections
- Fluid management
- Temperature Regulation
- DOC → **Acitretin**
- **Methotrexate.**

active space

Pustular Psoriasis of Pregnancy:

- a.k.a **Impetigo herpetiformis**
- occurs in last TM of pregnancy
- Recurrence in Multiple pregnancy



Presentation:

- pustules which start in flexures, umbilicus.
- Rupture of pustules ⟶ **Infringing scales**
- Affects the pregnancy outcome :
  - i) LBW
  - ii) Fetal mortality

Treatment: Steroids

Other Atypical forms :

1. Linear, Segmental
2. Napkin Rash in Infants
3. Rupoid → cone like scales
4. Elephantine ⟶ Thick scales
5. Ostraceous ⟶ Oyster shell like scales.

active space

ASSOCIATIONS WITH PSORIASIS

19:40

- Metabolic syndrome
  - ↳ Psoriatic March: systemic Inflammation
    - ↑ Obesity
    - ↑ HTN
    - ↑ Hyperlipidemia
    - Hyperinsulinemia
    - ↑ Risk of CAD.

} Metabolic Syndrome.

- Other Autoimmune disorders such as
  - i) Vitiligo
  - ii) Alopecia.
- Non-Melanoma Skin Cancers.

Treatment: depends on severity of Psoriasis.

1. Topical:
   - used for localised disease (<10% BSA involvement)
   - PASI < 10
2. Systemic
3. Phototherapy.

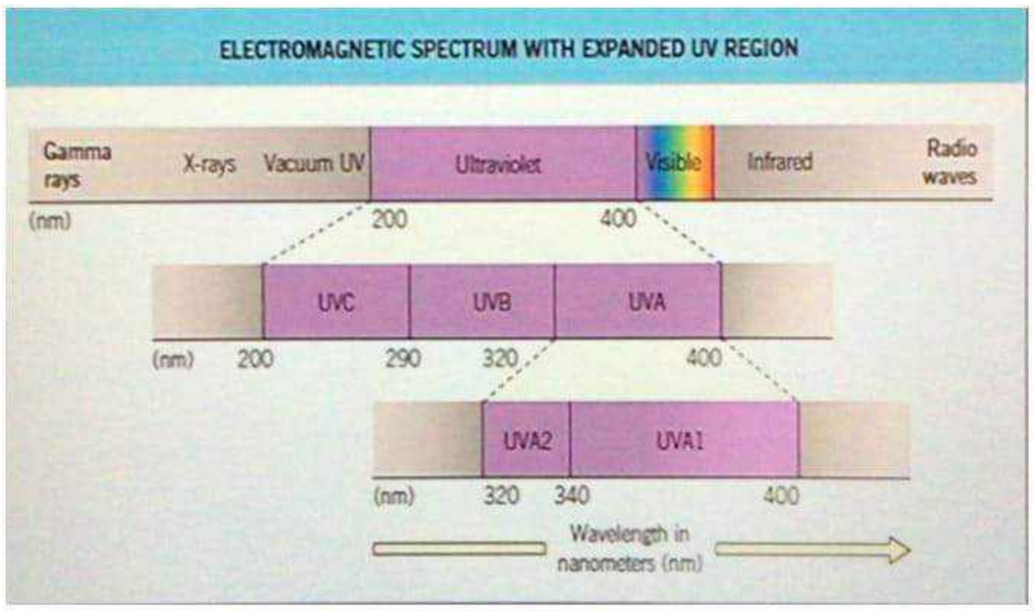
active space

Topical agents :

1. Emollients (systemic Corticosteroids
2. Topical corticosteroids → TOC are not used)
3. Topical Retinoids → Decrease proliferation and improves keratinization process
   - TAZAROTENE
4. Topical Vit D Analogues
   - Decrease inflammation
   - Decrease hyperproliferation.
   - Calcipotriol
4. Anthralin/ Dithranol (Ingram regimen previously used)
   - causes irritation (not used in flexural psoriasis)
   - Brown staining of Clothes ⊕
6. Coal Tar → used in Goeckerman's Regimen
7. Salicyclic acid
8. Calcineurin inhibitors →
   - Tacrolimus, Pimecrolimus
   - used in psoriasis involves face and flexures.

active space

Phototherapy



1. NB UVB (Narrow Band UV-B)

- 311 - 313 nm (Preferred)
- given 3 times a week
- Look for erythema at 48 - 72hrs.
- combined with other therapies

2. PUVA

- Psoralen + UV-A.
  - 8- Methoxy Psoralen (MOP)
  - 5 - MOP
  - Tri - MOP
- given topically / orally.

Psoralen binds with DNA
↓ ← UV A
DNA damage.

- Increased Risk of Skin cancers (BCC, SCC)
- Eye protectors must

active space

- given 2 times a week
- Not given in unstable patient, non-compliant, neurological diseases.

Targeted Phototherapy

- Phototherapy given only to target areas
- 308 nm Xenon chloride excimer LASER
- Specific devices needed.

Systemic Treatment:

- Methotrexate
- Acitretin
- Cyclosporin.
- Given in i) >10% BSA
  ii) Disease is hampering quality of life.
  iii) PASI > 10

Methotrexate:

- Inhibitor of Folate synthesis
- Inhibits DHFR

active space

- TOC for
    - i) Chronic Plaque Psoriasis.
    - ii) Erythrodermic Psoriasis
    - iii) Psoriatic arthritis
- Given as weekly dose 7·5mg - 30mg/wk.
- can cause :
    - i) ↓ Platelet count → CBC
    - ii) G.I Side effects → Nausea, Vomiting → change to s/c Mtx.
    - iii) Liver toxicity → So, it can lead to fibrosis

Cyclosporin :

- Inhibit IL-2
- Crisis Drug
- Not used for long term because it causes
    - i) Hypertension
    - ii) Hyperlipidemia
    - iii) Derangement of RFT
    - iv) Malignancies

active space

Acitretin

- derivative of Etretinate
- Retinoids used are
  - i) Isotretinoin → Acne
  - ii) Acitretin → Psoriasis.
- Improved Keratinization
- Decreased Inflammation
- DOC : i) Pustular psoriasis
  - ii) HIV / Immunodeficient patient
- Monitor CBC, LFT, TG's

Other Oral Agents used :

i) Fumaric acid esters

ii) Hydroxyurea

iii) Apremilast → PDE_4 inhibitor, Oral, G.I S/E

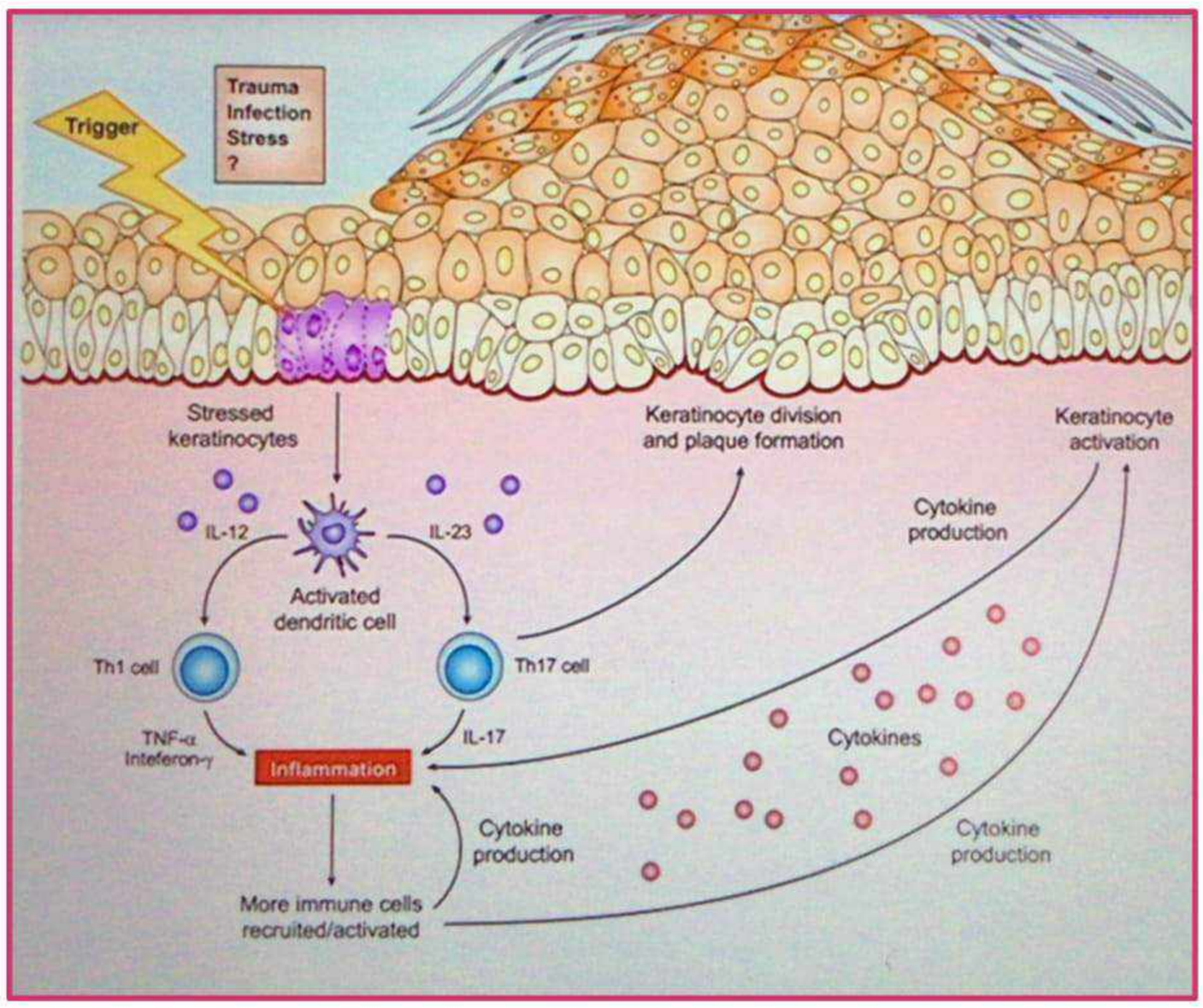
iv) Tofacitinib → JAK Kinase inhibitor

Biologicals

- used in i) >10% BSA
  - ii) Patient not responding to conventional Rx.

active space

- Anti TNF α inhibitor
  - Etarnacept
  - Infliximab ⟶ given as i.v
  - Adalimumab
- IL - 17 inhibitor ⟶ Sekukinumab.
- LFA - 1 inhibitor ⟶ Alefacept
- IL 12/23 inhibitor ⟶ Ustekinumab.



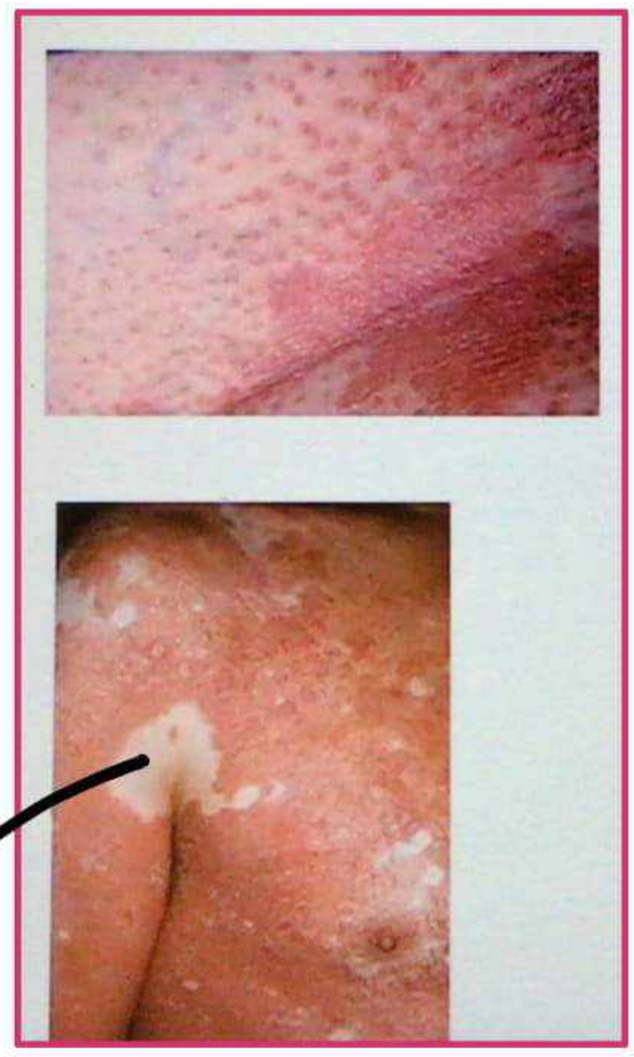
PITYRIASIS RUBRA PILARIS

52:18

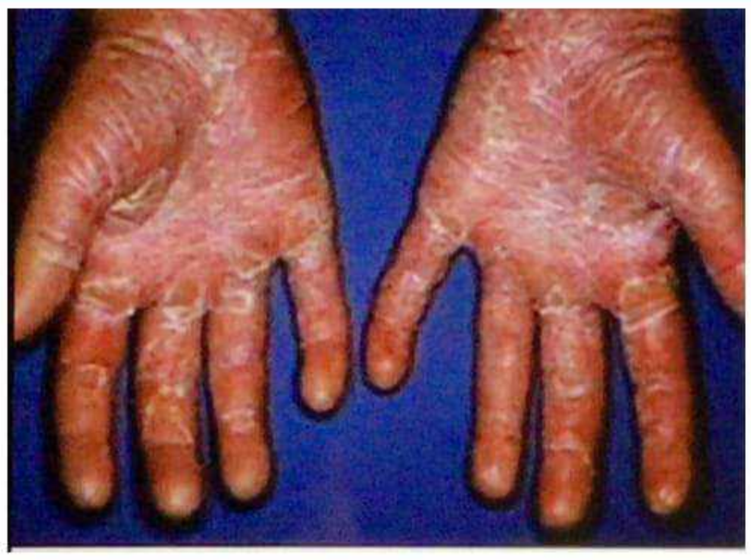
- Papulosquamous dermatosis of unknown cause.
- Associations : i) Streptococcal
  ii) HIV
- On HPE : • Checkerboard pattern.
  • Alternating vertical and horizontal ortho/ Hyperkeratosis and Parakeratosis.

Clinical feature :

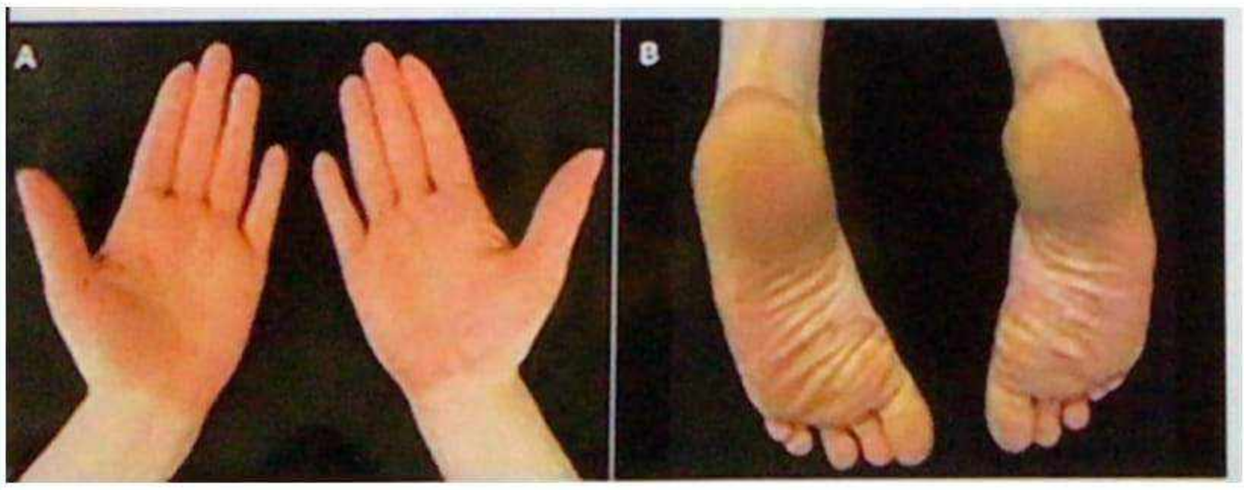
- Follicular erythematous papules
  ↓
  These are surrounded by interfollicular erythema. (Salmon coloured)
  ↓
  Merge → form sheets of dry scaly salmon coloured plaques.
  ↓
  May progress to Erythroderma

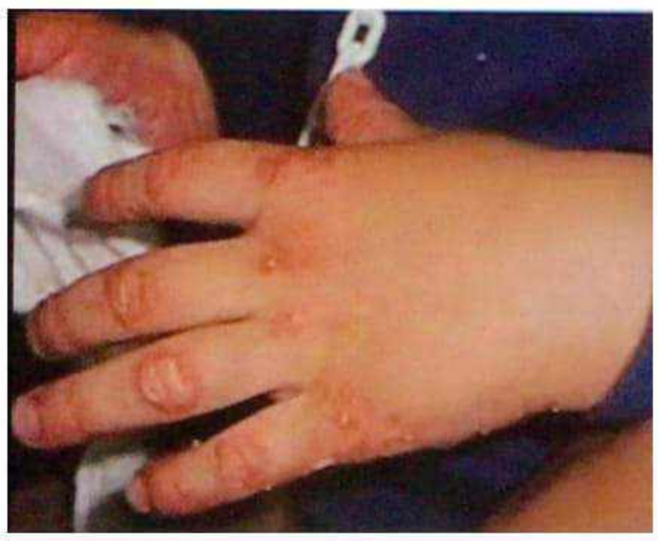


active space



- Diffuse Hyperkeratosis and Salmon discolouration of Hands and feet
- a.k.a **PRP Sandal**





- **Nutmeg Grater papules**
- Erythematous follicular papules
- seen in → Elbows, knees, IP joints, MCP.

- **Types :**
  1. Classical Adult Onset PRP (Type I) → (MC)
  2. Atypical Adult Onset PRP (Type II)
  3. Classical Juvenile Onset PRP (Type III) → Similar to type I Age 5-10yrs.
  4. Circumscribed juvenile PRP (Type IV) → on knees and elbows.

active space

5. Atypical Juvenile PRP (Type v)

6. HIV Related PRP (Type vi)

Treatment

- Topical Steroids
- Topical Keratolytics
- Acitretin.
- Methotrexate

Lichen Planus

- It is an autoimmune T-cell mediated disorder
- Chronic inflammatory
- Affects Skin + Nails + Mucosa and Hair

Pathogenesis :

- Antigen → unknown
  ↓
  may be

Self peptide acting as Ag.

External agent

- Infections → HCV, HBV
- Drugs → i) Antibacterials : Ethambutol, Streptomycin
  - ii) Antihypertension :
    - ACE ⊖ (captopril) Beta Blockers
  - iii) Anti-malarials
  - iv) Anti-convulsants
  - v) Heavy metals.
- Contact Allergen.

Contact Allergen:

- Dental Amalgam (Mercury)
- Gold
- T cell response to this Antigen

↓

Basement membrane

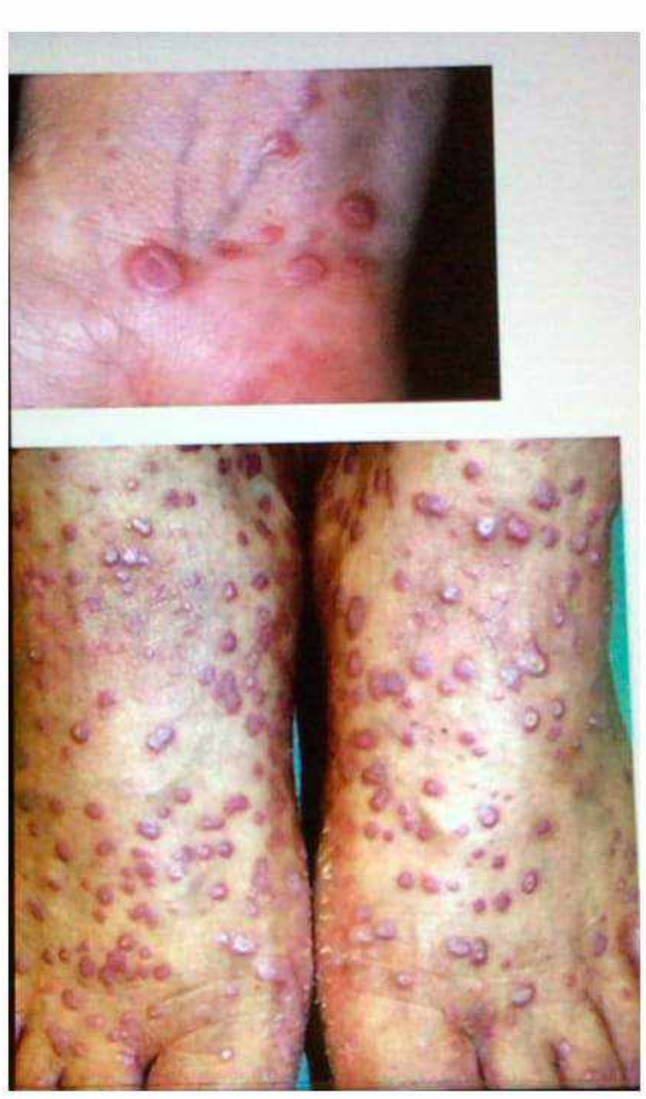
↓

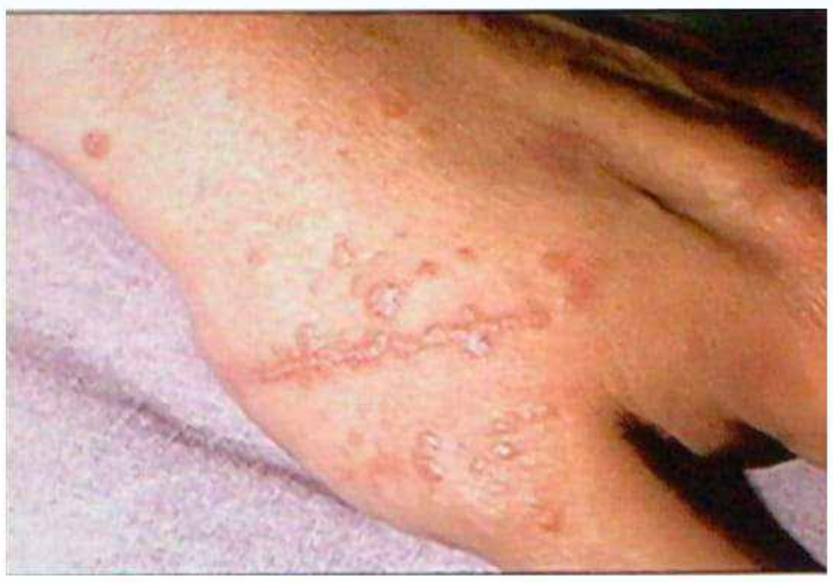
Band like infiltrate along the basement membrane

Clinical features:

In Skin:

- Itching (pruritic) → doesnot scratch, but Rubs.
- Sites involved:
  - Wrists
  - Elbows → Arms, trunk.
  - Flexures
- 5 P's
  - i) Pruritic
  - ii) Purple / Violaceous.
  - iii) Polygonal
  - iv) Plane topped → Smooth
  - v) Regular and Pleasur



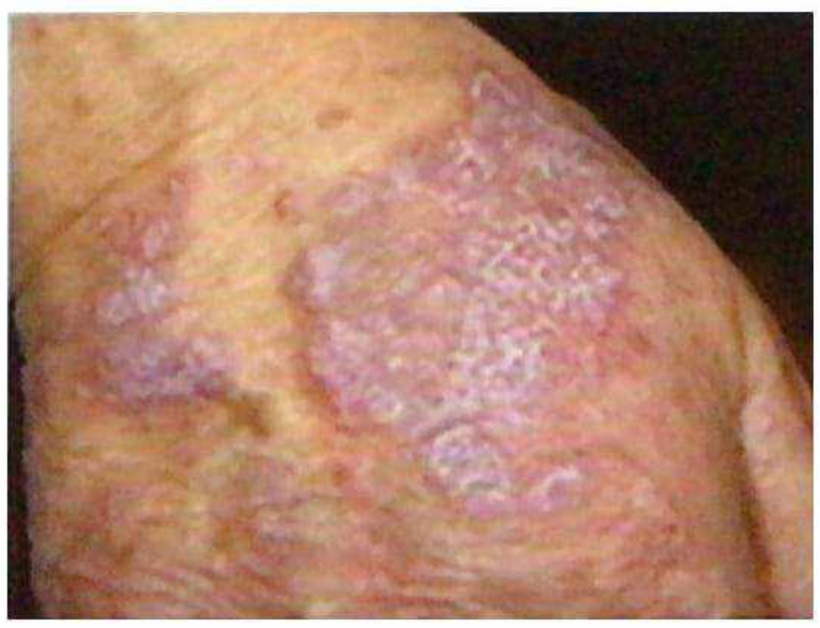


- Koebners phenomenon is seen.

  ↓

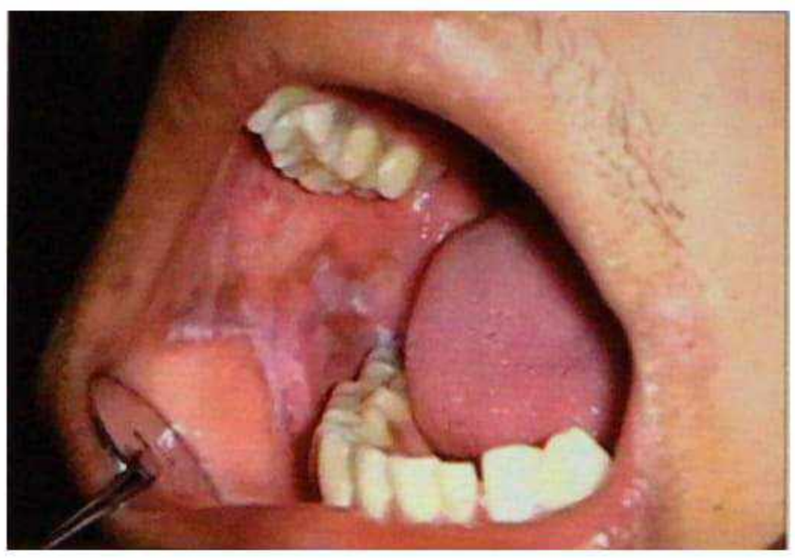
  Lesions along the line of trauma.

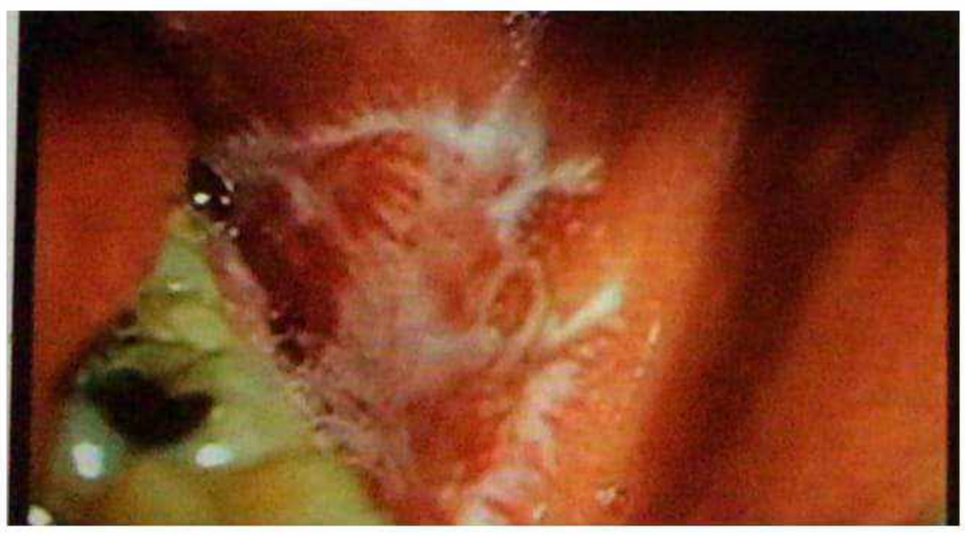
- also seen in Psoriasis, Molluscum contagiosum, Warts.



Wickham's Striae.

- White lacy net like pattern on LP lesions
- Representative of hypergranulosis on Biopsy.





- 70% patients → oral mucosa involvement

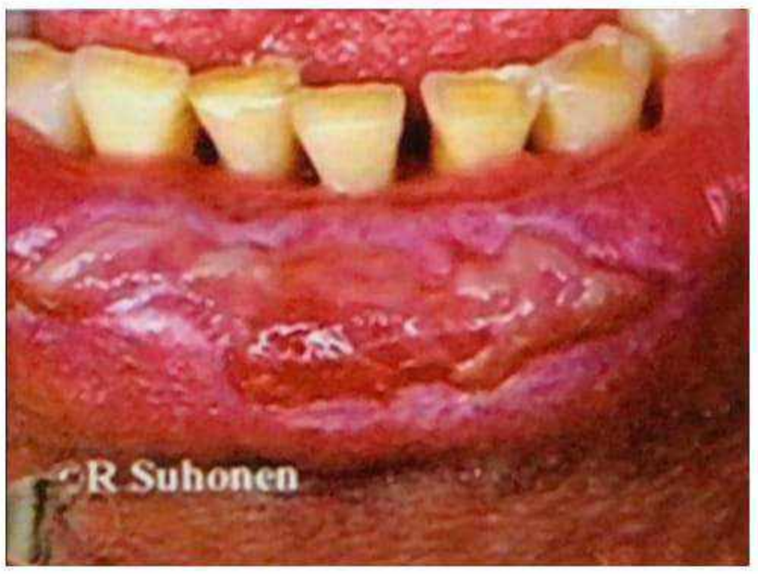
active space

- Symptoms :
  - Asymptomatic
  - Burning sensation on hot and spicy food
  - Reticular LP → White lacy pattern usually on buccal mucosa, tongue, gingival mucosa.

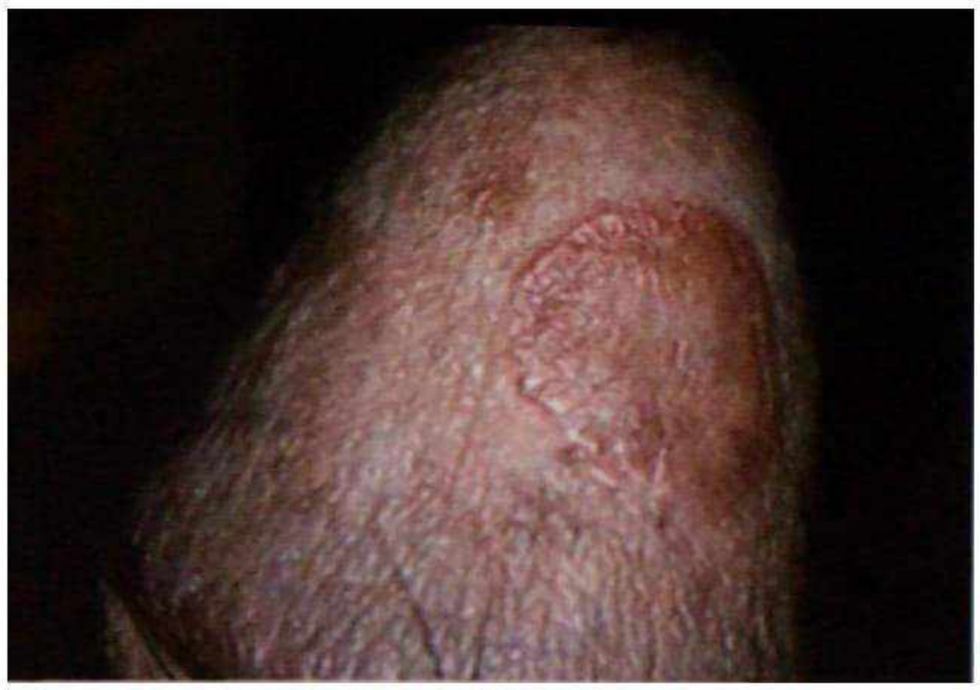
Variants

1. Erosive
2. Atrophic
3. Bullous
4. Ulcerative

- sometimes it may be pre-malignant.

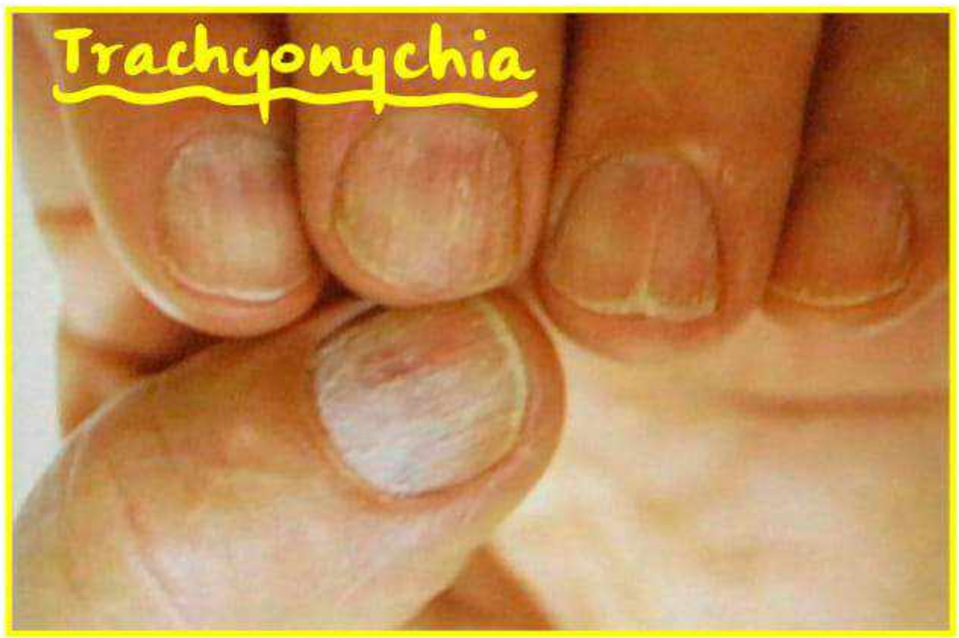


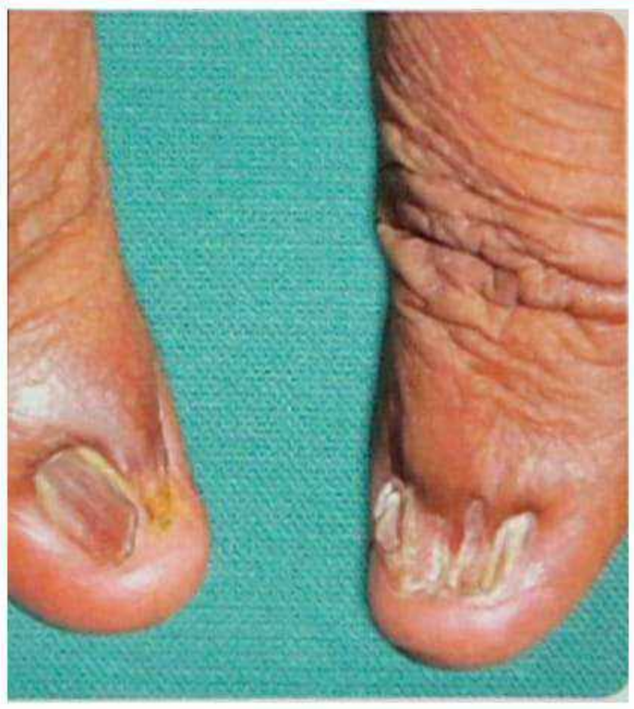
Genital mucosa : Annular LP



active space

- **Nail changes**
  - Roughening
  - Longitudinal Ridging
    → (MC) finding
  - Thinning

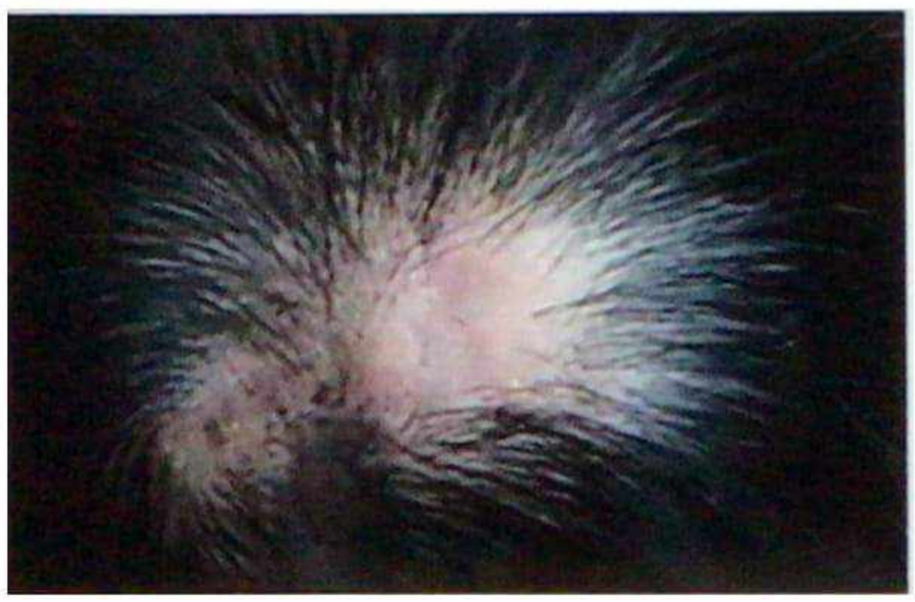


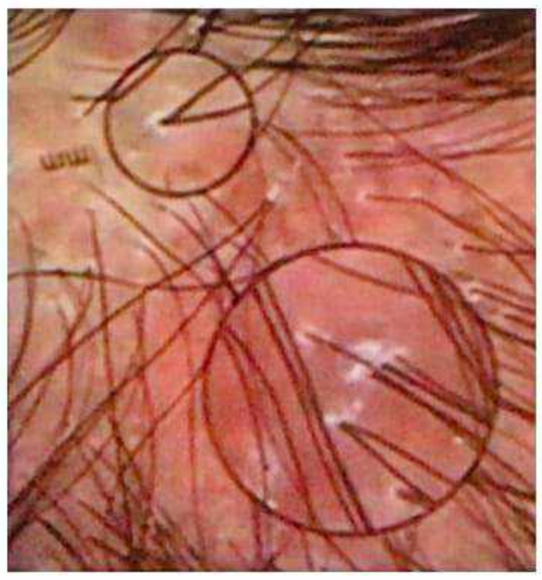


Pterygium

- Most specific finding of LP
- Wing shaped extension of Proximal Nail Fold on the nail bed.

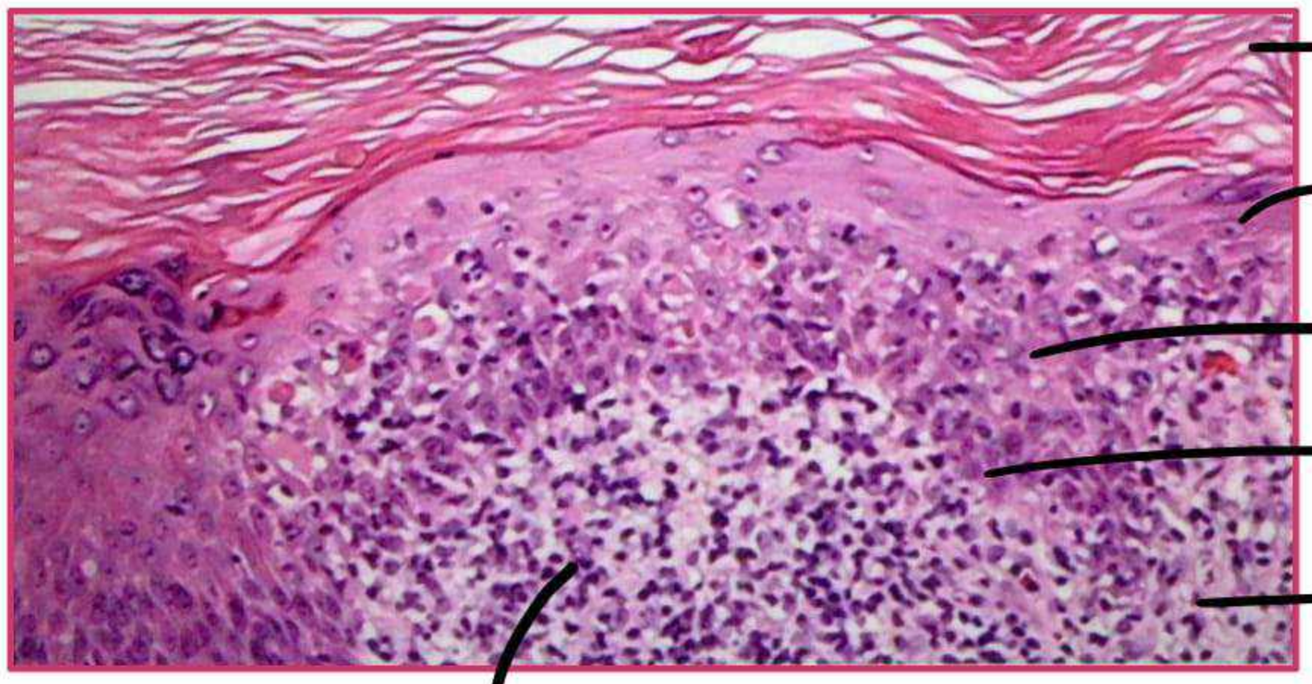
In Hair :
- Cicatricial / Scarring Alopecia.
- Destruction of follicles ⟶ Permanent.





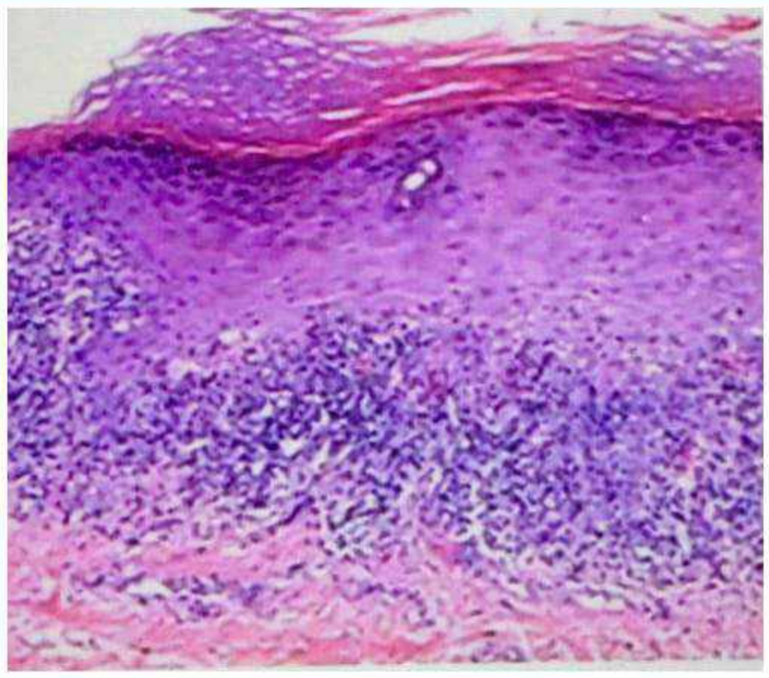
active space

On Histopathology:



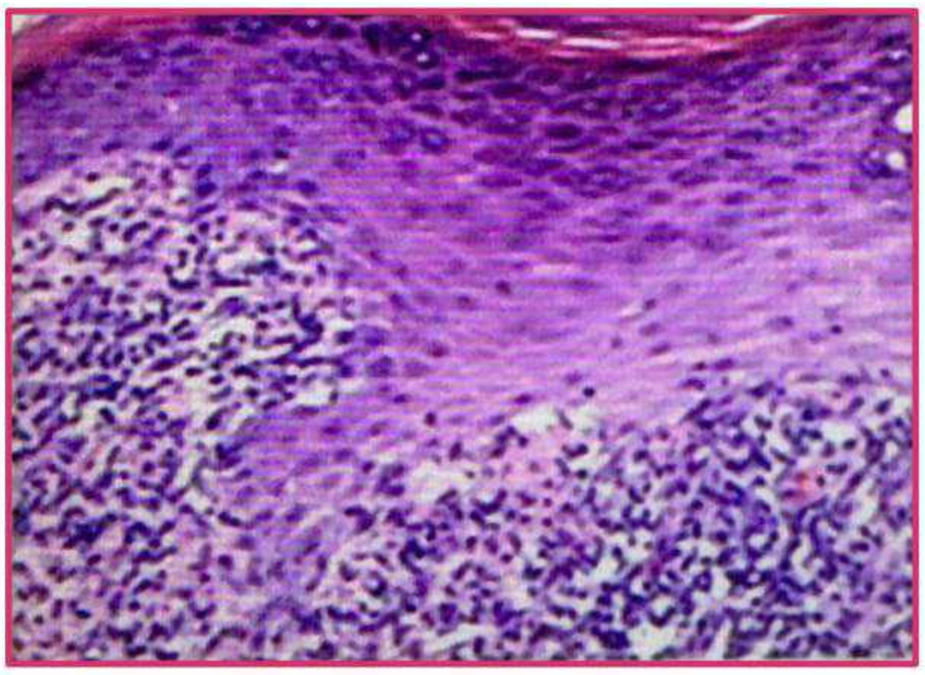
Colloid Bodies / Civatte Bodies

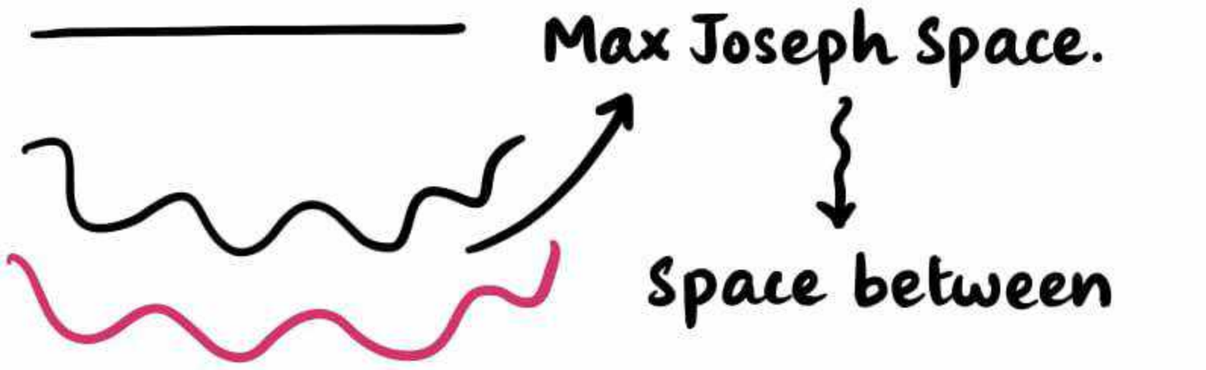
- Hyperkeratosis (Stratum corneum)
- Hypergranulosis (focal Wedge shaped)
- Acanthosis (Stratum spinosum)
- Basal cell degeneration
- Colloid Bodies / Civatte Bodies → Apoptotic keratinocyte.
- Band like infiltrate in dermis.

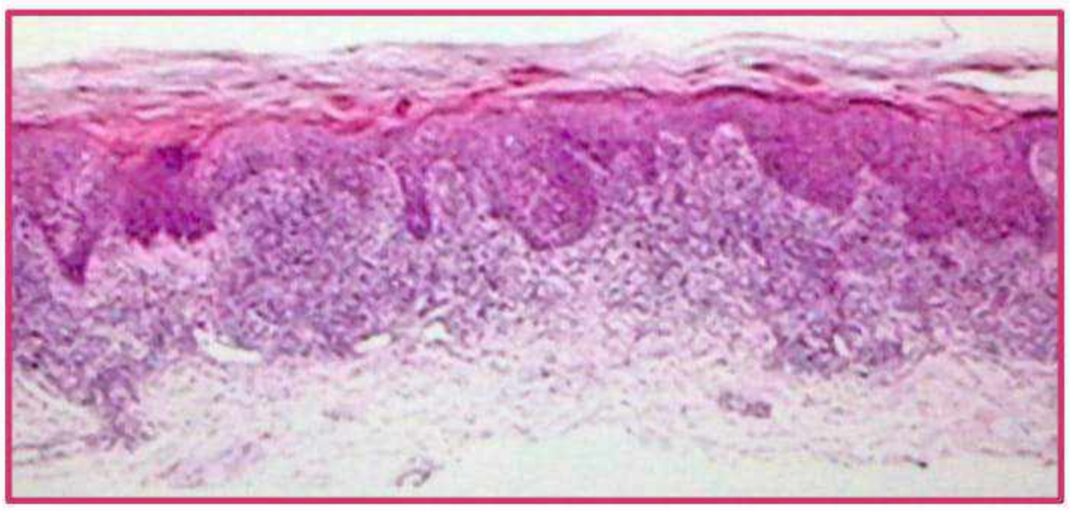


- Pigmentary incontinence

active space







TREATMENT

31:45

Topical | Systemic | Phototherapy

1. Topical

- Corticosteroids
  - Topical
  - I/L → Hypertrophic LP
    → Nail LP

active space

- Calcineurin inhibitors inhibitors : Tacrolimus, Pimecrolimus.
  ↳ used when face, genitalia are involved.
- ⟶ Topical Anesthetic agents → Oral mucosa.

2. Systemic

- used when there is extensive lesion
- Oral corticosteroids ⟶ Acute Eruptive LP
- Dapsone
- Acitretin
- Anti-malarials ⟶ HCQ's
- Methotrexate
- Thalidomide
- Azathioprine

→ ⊕ Antihistamines.

3. Phototherapy

- Narrowband UV-B (not useful in Generalised Acute LP)
- PUVA - uncommon.
- In Hair LP ⟶ Counsel

Prognosis

active space

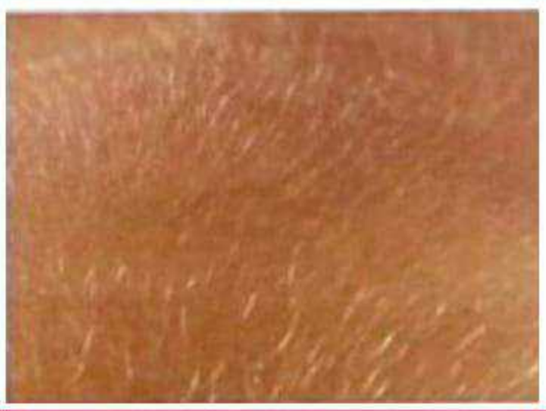
- spontaneous remissions and Relapses

Hair Disorders (Part - 1)

- 3 Types of Hairs :

Lanugo hairs

- in-utero
- develop 8-9th intrauterine month
- Soft, non-pigmented.
- Pre-mature babies



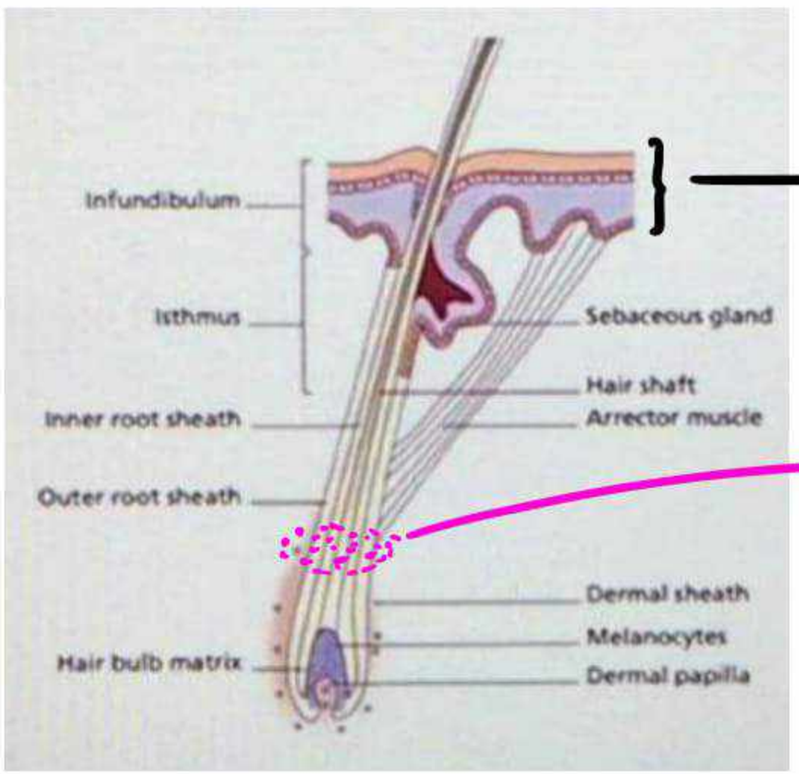
Vellous hairs

- Soft
- unmedullated
- usually non-pigmented
- short < 2cm.

Terminal hairs

- Hard
- Pigmented
- Medullated
- long
- Androgen dependent

Hair Structure :



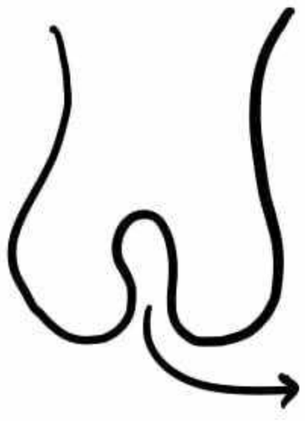
} → Epidermis.

Bulge : location of stem cells.

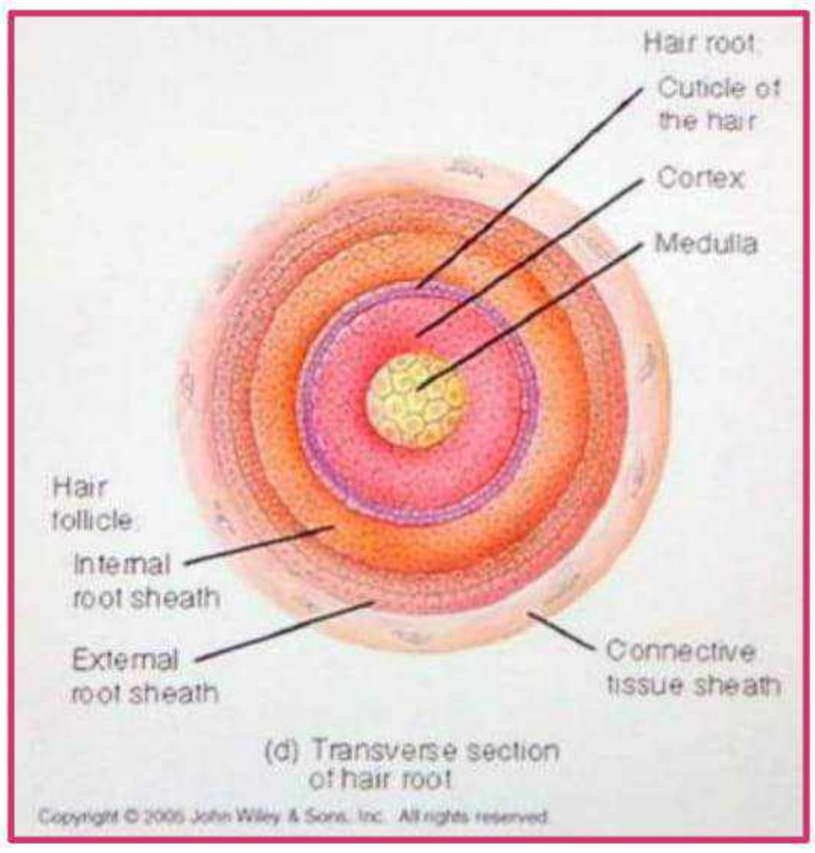
↑ if affected → cause scarring alopecia

- upper part → Infundibulum
  - upper part → Isthmus
  - } Relatively constant
- Lower part → Suprabulbar
  - Lower part → Bulbar
  - } undergoes changes.

- **Infundibulum :** part from epidermis to sebaceous gland.
- **Isthmus :** part from sebaceous gland to arrector pili muscle.



Hair, Transverse Section of Hair Root



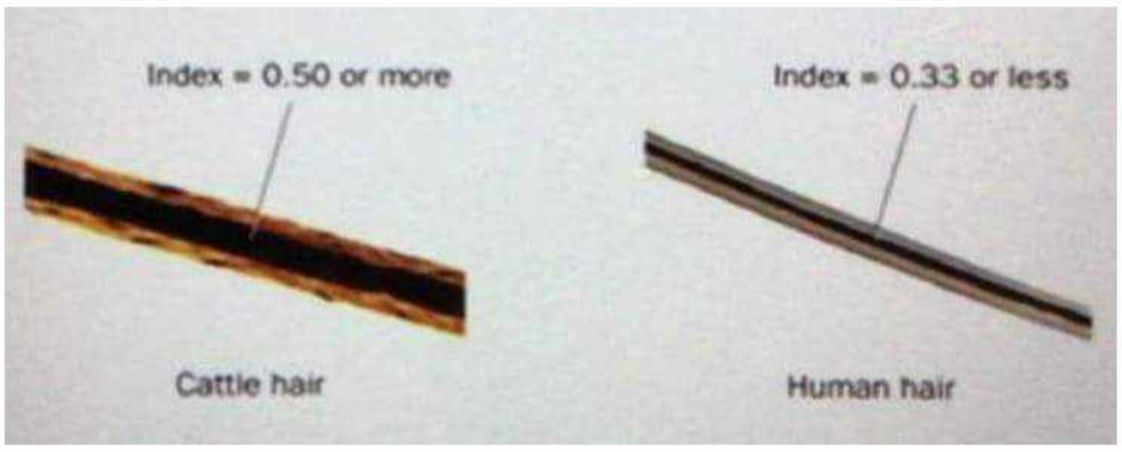
- Cortex → Thickest
- Inner Root Sheath made of 3 layers
  - i) IRS cuticle
  - ii) Huxley layer
  - iii) Henley layer

active space

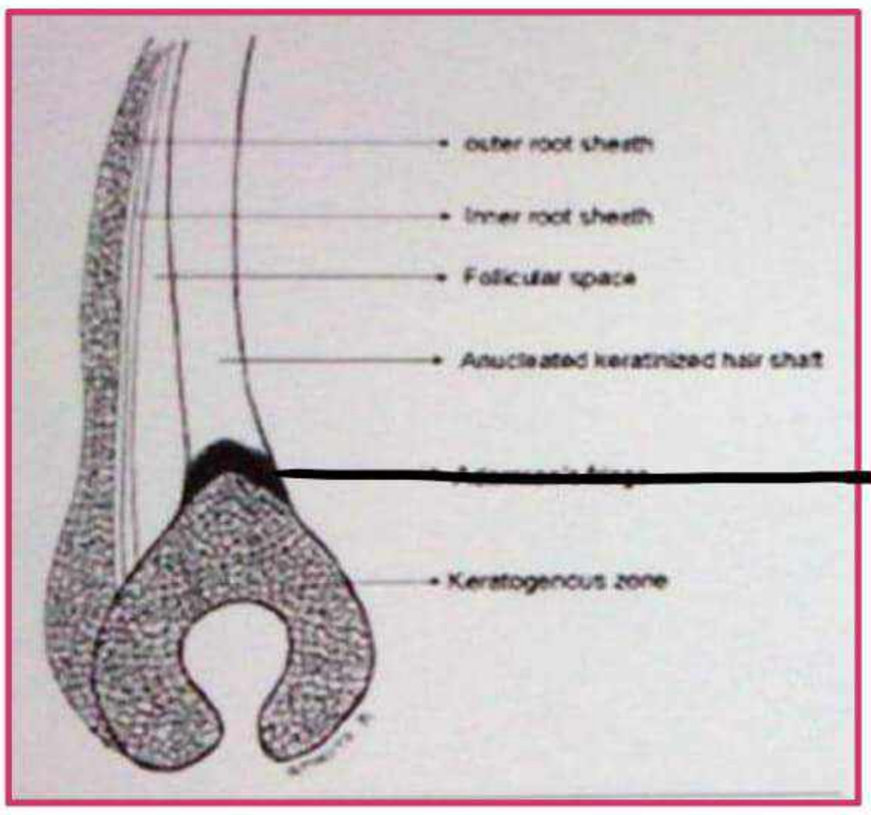
MEDULLARY INDEX

13:30

- Measure of the diameter of the medulla relative to the diameter of the hair shaft.



- usually expressed as fraction
- Humans : M.I ⟶ < 1/3 ⟶ < 0.33
- Animals : M.I ⟶ > 1/2 ⟶ > 0.5



Adamson's fringe : junction between keratinized and non-keratinized part of hair.

- Tinea capitis ⟶ Restricted

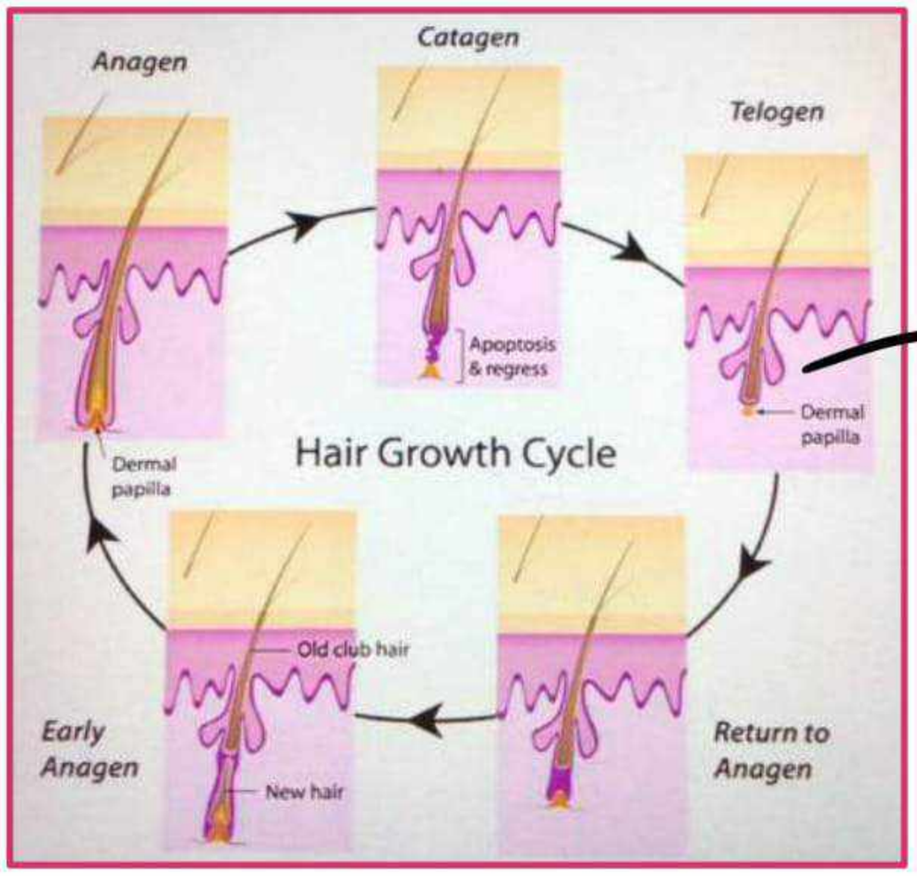
active space

HAIR CYCLE

16:35

- Repeated cycle ↘

Growing phase (Anagen) → Transition phase (catagen) → Falling phase (Telogen)



→ Club shaped hair root

ANAGEN	CATAGEN	TELOGEN
• Phase of hair growth • determines the length of the hair • 3 years • 85-90% of hair	• Resting / Transition phase • 3 weeks • 1% of hair	• Phase of hair fall • 3 months. • 10-15% • 50-100 hair shed/ day → Normal.

active space

Anagen to Telogen = 9:1 ⇒ Normal

- Hair growth Rate : 0.35mm/day (1cm/month)

Functions :

- protects our scalp
- cosmetic.

HAIR DISORDERS

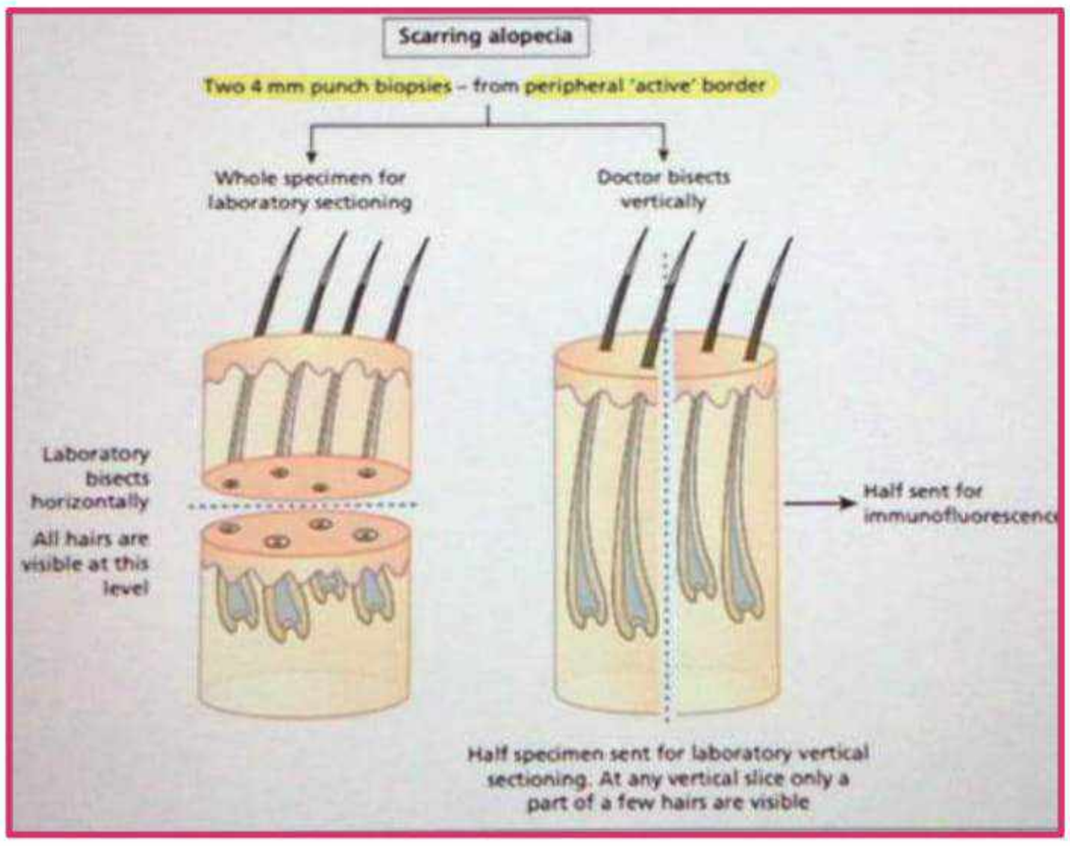
24:20

- Hair fall (Alopecia)
  - Non-scarring / Non-cicatricial → Hair follicle not getting destroyed
  - Scarring / Cicatricial → Hair follicle getting destroyed
- Excessive Hair Growth

Tests for Hair

1. Hair Pull Test
2. Trichogram
3. Trichoscan
4. Scalp Biopsy

active space



Sectioning

i) Non-scarring → Horizontally

ii) Scarring → Vertically.

NON-CICATRICIAL ALOPECIA

30:40

- a.k.a **Non-scarring alopecia**
- Hair loss is not permanent, follicles are retained.
- **3 Types :**

i) **Patterned** → Androgenitic Alopecia → Male Pattern Hair loss / Female Pattern Hair loss

ii) **Diffuse** → Effluvium → Anagen / Telogen

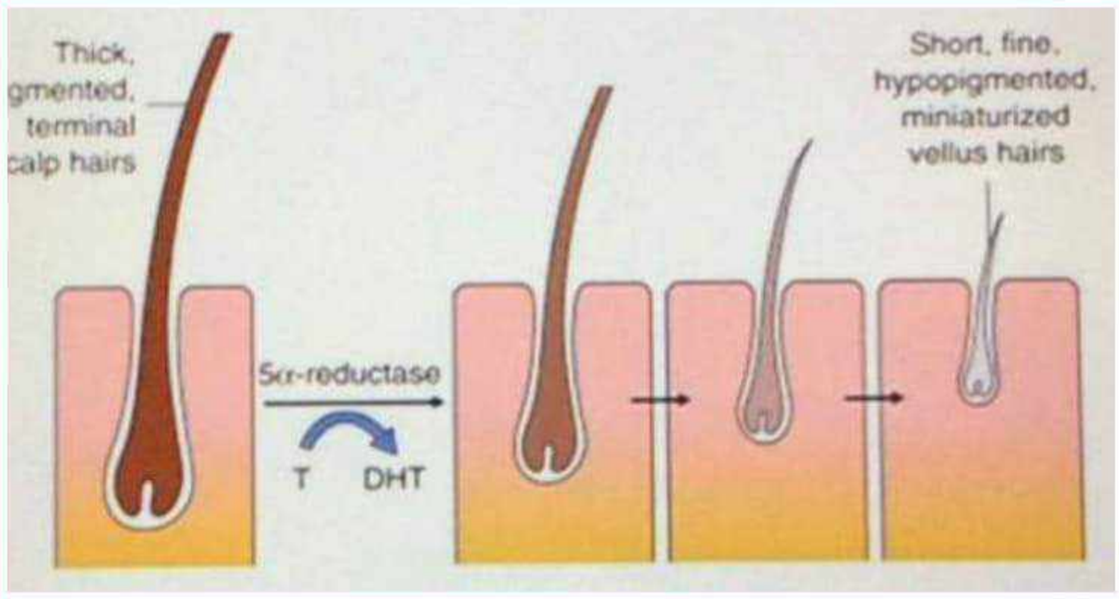
iii) **Patchy** →
- Alopecia areata
- Tinea capitis
- Trichotillomania.
- Syphilis.

active space

ANDROGENETIC ALOPECIA

32:50

- a.k.a Patterned Hair loss
- Types :
  - i) Male Patterned Hair loss
  - ii) Female Patterned Hair loss
- Associations :
  - vertex balding → ↑ Risk of Prostate cancer
  - Metabolic syndrome → ↑ Risk of CAD.



T-Testosterone

DHT - Dihydrotestosterone.

Androgen mediated

Testosterone —(5α Reductase)→ Dihydrotestosterone

- Hair → 5α Reductase (2) is present.
- DHT → causes miniaturization of hair follicles
- conversion from Terminal hair → Vellus hair

active space

- Anagen is shortened
- It is seen Androgen dependent areas : (occiput hair is not androgen dependent)

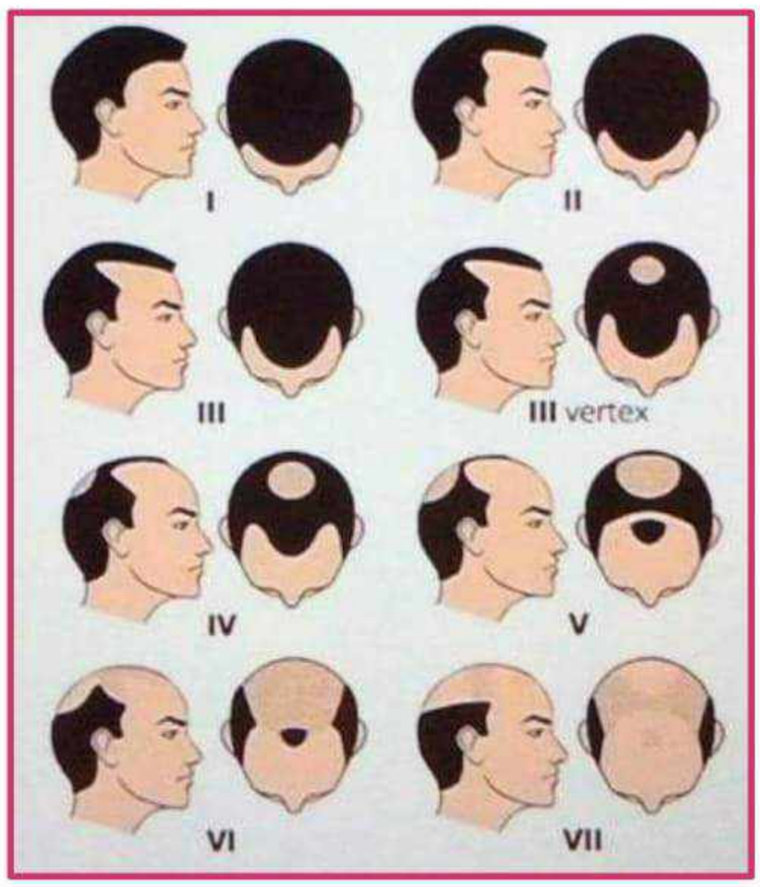
↓

Basis of using occipital hair for Hair Transplant.

- Androgens :
  - ↑ Hair on beard and moustache
  - Reverse action on scalp.
- Arao Perkins Bodies : remnant of old follicle
- T : V Ratio → 8:1 → 4:1
- A : T Ratio → 5:1

} leads to Balding

- Genetic susceptibility → ⊕ Family h/o (Polygenic inheritance)

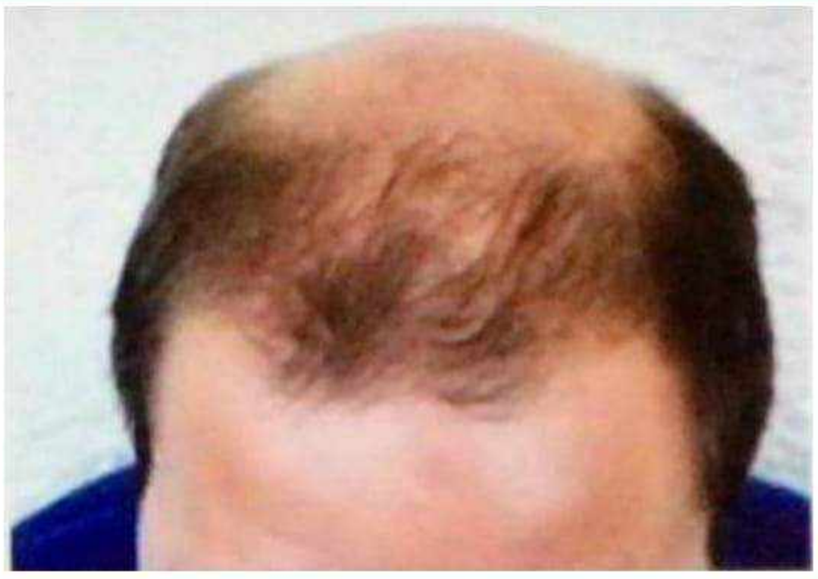


- In males → fronto temporal recession
- Norwood Hamilton Grading

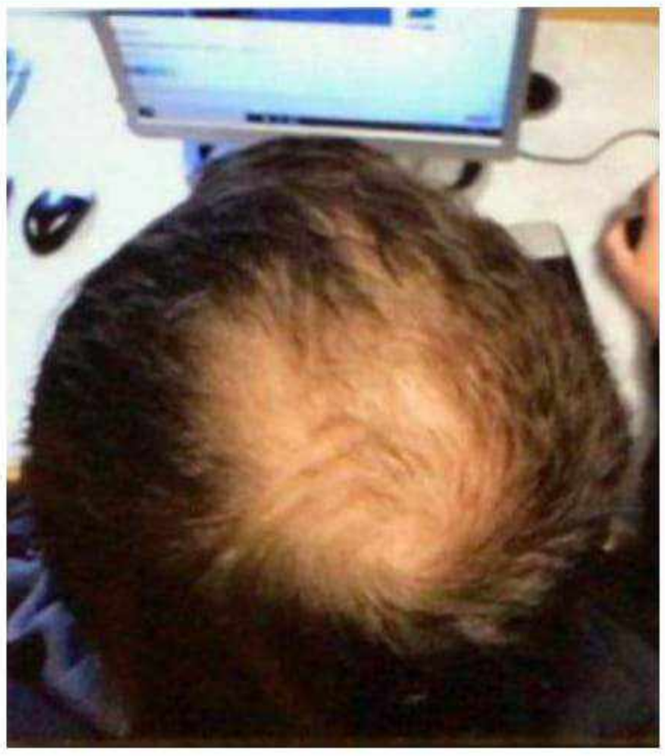
↓

Grade I - VII

- Asymptomatic
- Decreased Hair density

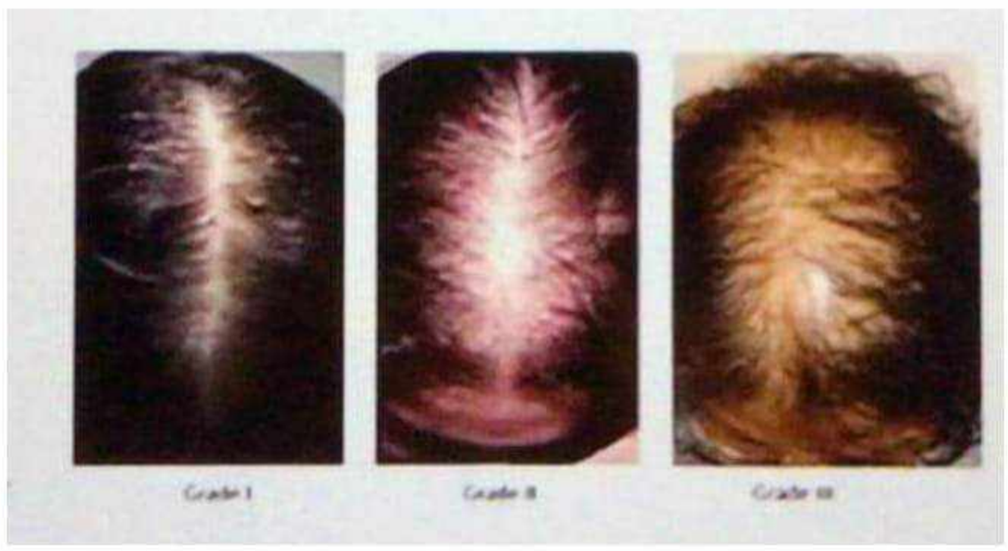


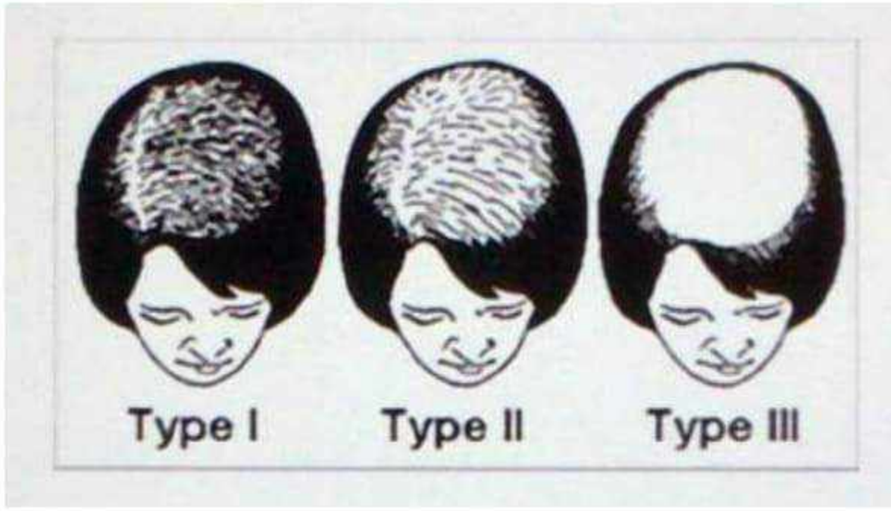
Fronto-temporal recession



Vertex involved

- In females :





- Widening of central parting
- Ludwing Scale → Female Patterned Hair loss
  - 3 Types → Grade I to III
  - Grade III → Christmas tree / Fir tree pattern.

Investigation :

1. Hair Pull Test → ⊖

2. Biopsy

vs CTE → • T:V Ratio → Not altered

• T:A Ratio → Altered.

3. Trichoscan : Hair diameter diversity

Management :

- Counselling : Lifelong treatment

i) Topical :

- Minoxidil → K^+ channel opener → Arteriodilator

↳ 5% or 10% → Males } FDA Approved.

2% → in Females }

- S/E i) Hypertrichosis

ii) Headache

iii) Contact Irritant Reaction.

2) Systemic :

- 5 α Reductase inhibitor

- Finasteride → 5α Reductase 2 inhibitor → 1 mg/day.

- Dutasteride → 5α Reductase 1 and 2 inhibitor

- S/E of Finasteride : i) loss of libido

  ii) Impotency in < 1% of population.

- In females → Anti-Androgens like } can be used.
  - Spironolactone
  - CPA (Cyproterone Acetate)
  - Flutamide.

Surgical Treatment :

- Hair transplantation (Donor → Occipital)
- PRP (Platelet Rich Plasma)
- Cosmetic camouflage → Wigs.

EFFLUVIUM

56:09

- disturbances in hair cycle
  ↓
  2 Types
  ↙ ↘
  Anagen Effluvium | Telogen Effluvium

- arrest in Anagen phase
- Global / diffuse alopecia
- Chemotherapy

Telogen Effluvium

- Premature conversion of anagen into telogen

Acute Telogen Effluvium

Stress → Anagen —converts→ Telogen phase

- Surgery
- Illness ⟶ TB, Malaria, Dengue etc
- Pregnancy ⟶ Telogen gravidarum.
- CAD

- Stress ⟶ 3 months later ⟶ Hairfall
- Hair Pull Test ⟶ ⊕
- Treatment : Counselling

Chronic Telogen Effluvium

- Insult is repititive
- Hairfall > 6 months
- Causes :
  - Iron deficiency Anemia. (MC) cause
  - Thyroid disorder ⟶ Hypo / Hyper.

active space

- Malnutrition
- Crash diet

- ↓ ponytail thickness
- Biopsy :
  - Altered A : T Ratio , T : V Ratio preserved.

Treatment : Correct underlying cause.

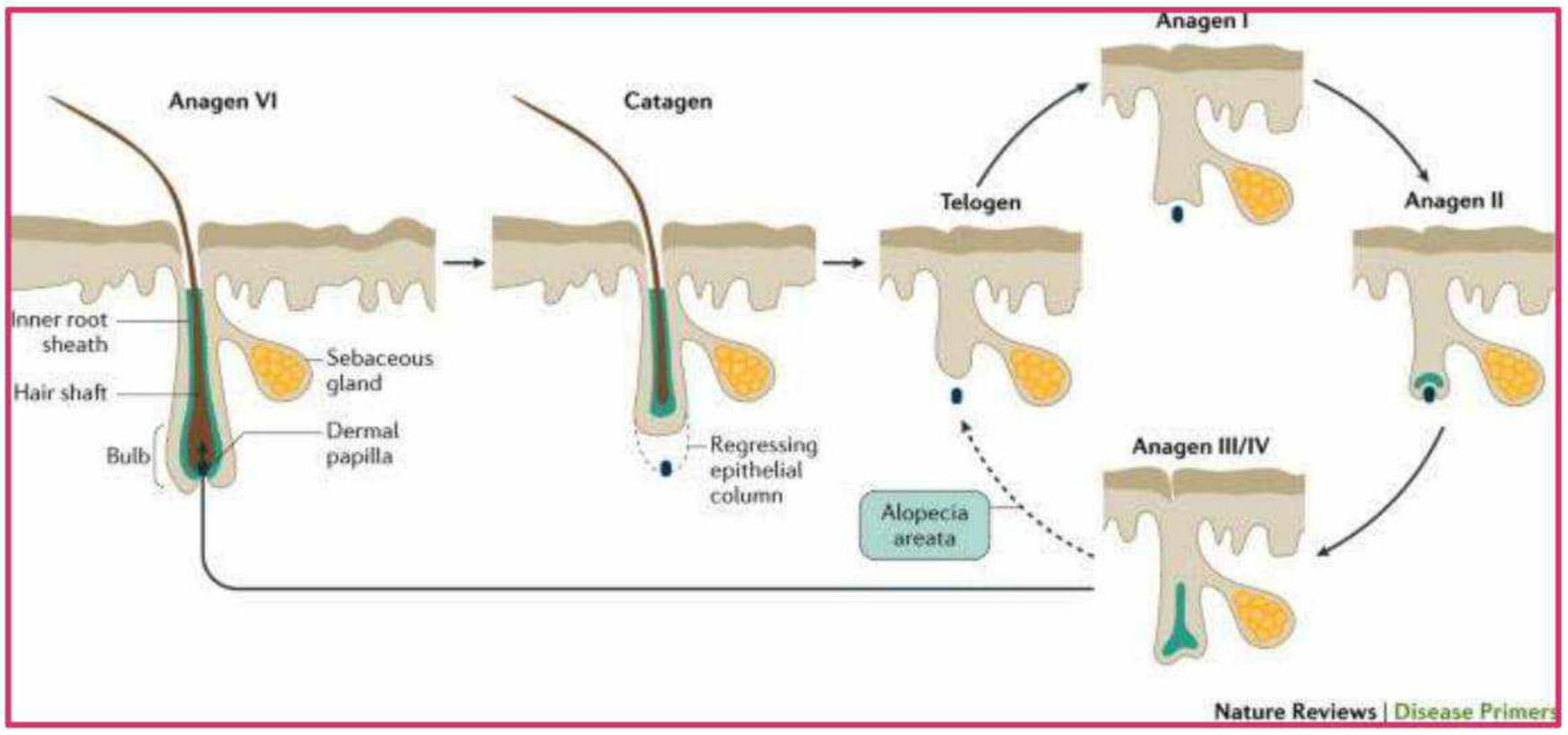
ALOPECIA AREATA

01:05:00

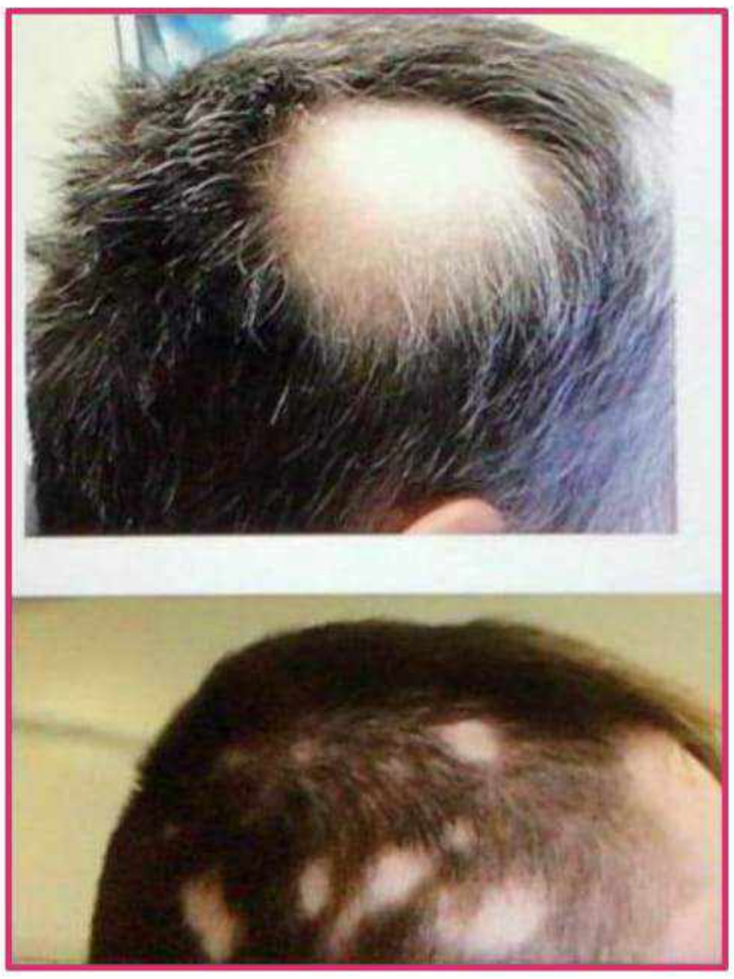
- chronic T-cell mediated disease
  ↓
  patchy non-scarring hair loss ⊕
- Autoimmune
- Associations :
  - Other autoimmune disorders → Thyroid, Pernicious anemia, Type 1 DM.
  - Downs syndrome
- Genetic → HLA association ⊕

active space

Pathogenesis :



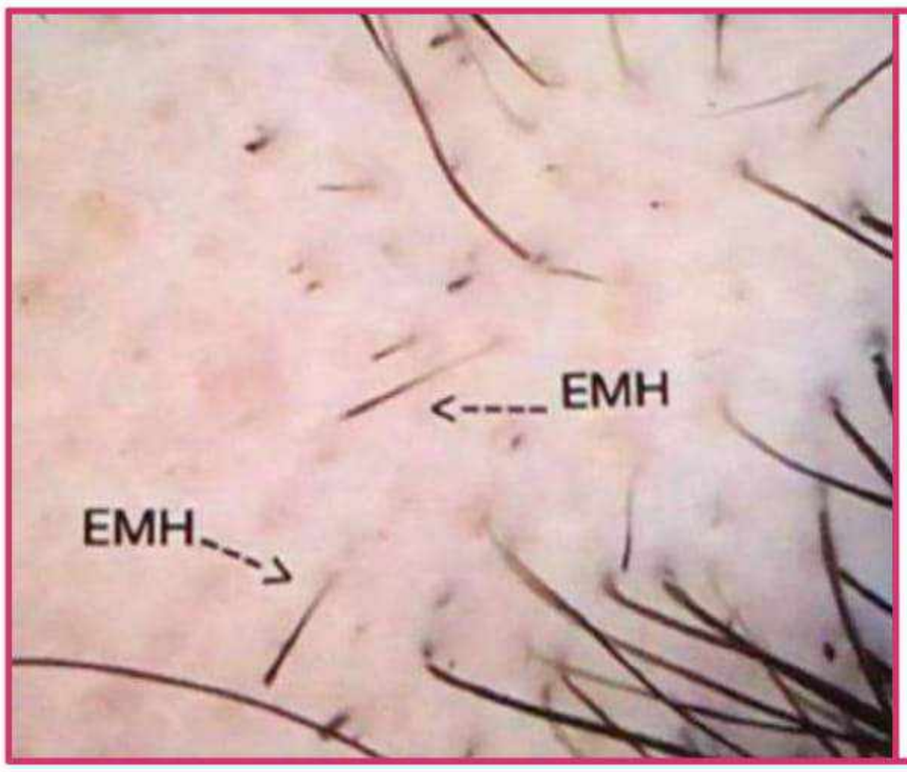
- T cell mediated disease
  ↓
  Anagen hair follicle
  ↓
  Converts Prematurely into telogen



Clinical feature:

- Patchy loss of hair → patch → smooth non-scarring, asymptomatic, no scaling, no redness.
- Easy breakability from periphery
- Single / Multiple.

active space

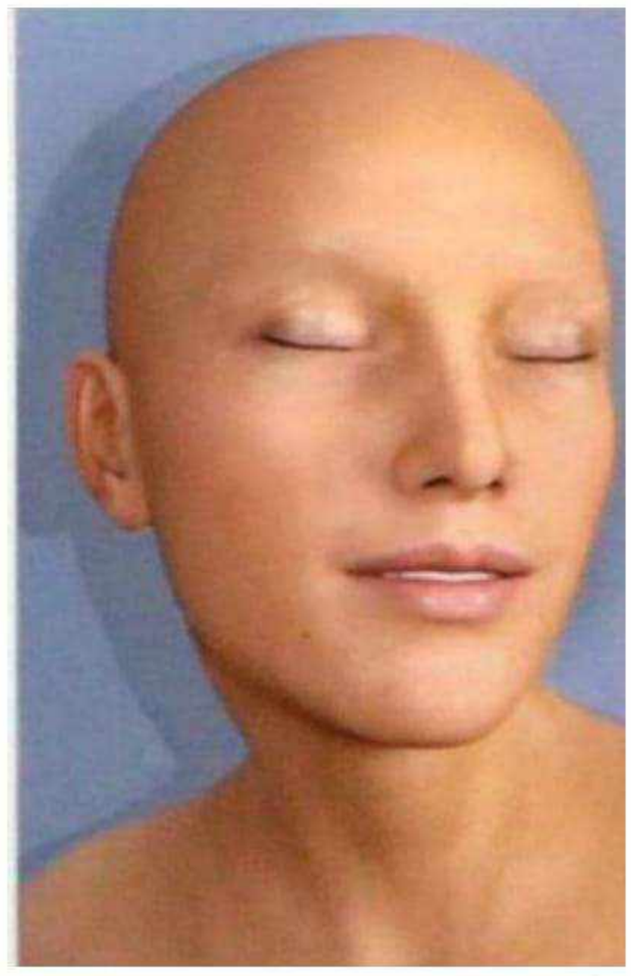


Esclammation mark

- Seen in Alopecia Areata periphery of AA patches

Types of Alopecia areata

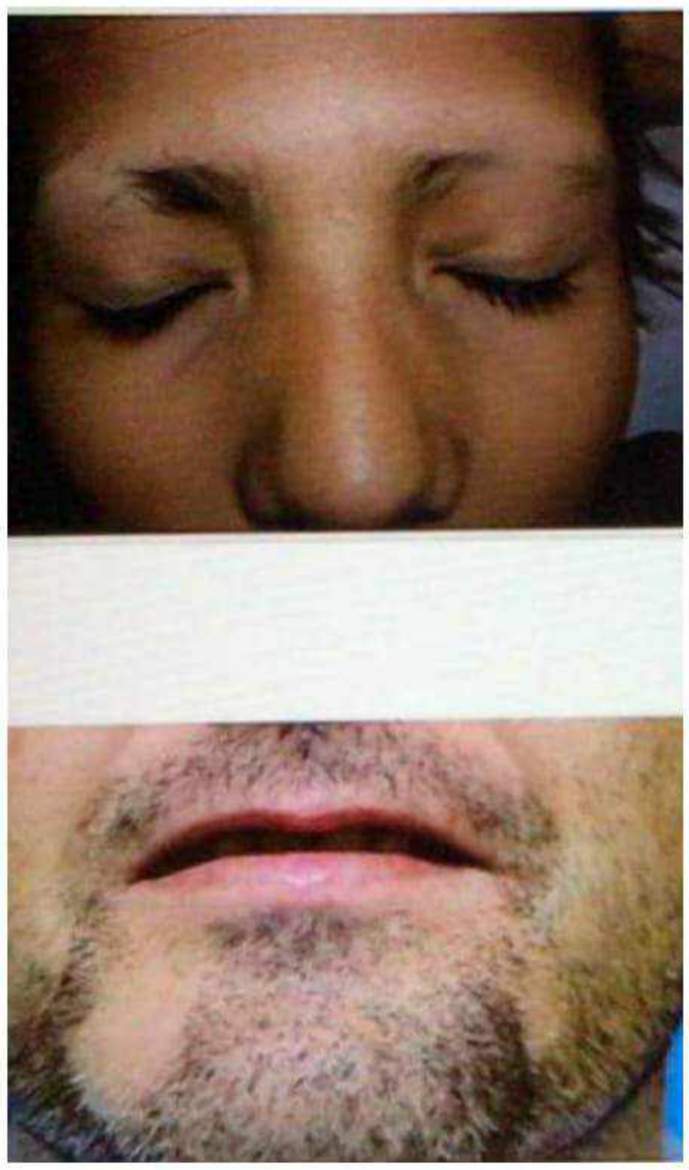
Ophiasis - loss of occipital band of hair.



Alopecia universalis : loss of hair from other body parts also.

- **Saphio :** occipital hair are retained.
- **Alopecia totalis :** Complete loss of scalp hair

- **Alopecia universalis :** loss of hair from other body parts also.



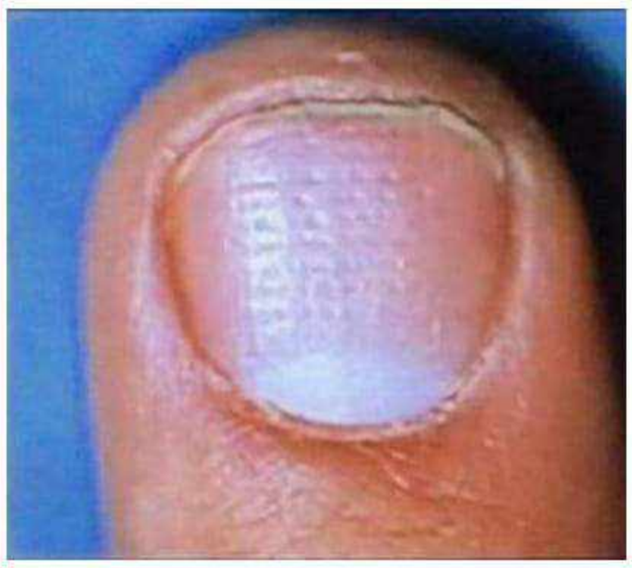
Alopecia areata

- loss of hair from
  - i) scalp – (MC) site.
  - ii) Eyebrows
  - iii) Eyelashes
  - iv) Beard

Reticular pattern Alopecia areata.

- Alopecia areata → Types → Diffuse
  - → Reticular

Nail changes :



i) Pits

- Smaller, superficial regularly arranged
- Fine stippling
- Geometric pattern.

active space

2) Roughening of nail plate :

- a.k.a Trachyonychia
- Sandpaper appearance.

- Course of Alopecia areata: 80% resolve on its own (Spontaneous Resolution)
- D/D's

  vs. Tinea capitis → scaling, itchy, broken hairs, on KOH staining (Fungus ⊕)

  vs. Trichotillomania

  vs. Syphilis → • Moth eaten alopecia
  • Other signs on body
  • VDRL ⊕

Prognostic factors :

- Extensive involvement
- Reticular
- ⊕ Family h/o

active space

- Atopic
- Down's syndrome
- Other autoimmune
- Duration more.

Treatment :

- Spontaneous resolution is a possibility

i) Topical :

- Corticosteroids → Topical
  → Intralesional steroids - TOC.

- Intradermal infection into the patch of Alopecia areata.
- Triamcinolone acetonide : 2.5mg - 5mg/ml every 3 wks.

ii) Contact immunotherapy

- SADBE
- DPCP
- DNCB

iii) Topical :

- Minoxidil
- Phenol
- Anthralin

active space

iv) Systemic :

- Levamisole
- Oral Mini Pulse Steroids
- Cyclosporin

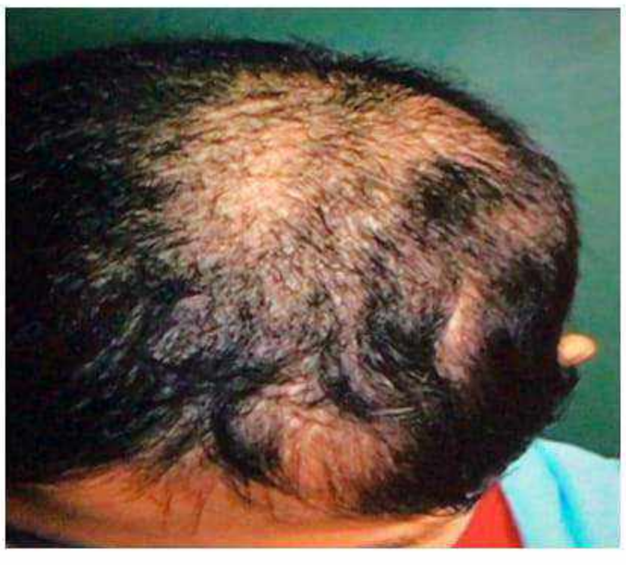
active space

Hair Disorders (Part - 2)

TRICHOTILLOMANIA

00:10

- Type of disease : OCD of pulling hair.(DSM - IV)
- History : Negative



Clinical features :

- Patchy areas of hair loss
- Site :- Frontoparietal area
  - Hair will break.
- Broken hairs of variable length

Other associated features :

i) Trichophagia

ii) Trichobezoar ⟶ can lead to obstruction

- Perifollicular hemorrhages ⊕

On Skin Biopsy :

- Empty casts
- Perifollicular hemosiderin deposition

Treatment:

- Cognitive Behavioural Therapy (CBT)
- SSRI's
- Paroxetine
- NAC ⟶ N-Acetyl Cysteine.

CICATRICIAL ALOPECIA

06:10

- a.k.a Scarring Alopecia
- Damage is occuring at bulb (at lower end of isthmus)
  - bulb → Stem cells → if affected → leads to permanent damage to hair follicles

Symptoms:

- Itching
- Pain
- Burning sensation

Diagnosis:

1. Scalp Biopsy for definite diagnosis

active space

Course: permanent damage ⊕ → prolonged.

Management:

- Irreversible
- Treat the activity and prevent further progression.

- **Primary:** direct involvement of hair follicles
- **Secondary:**
  - Sclerosing disorder
  - Trauma: damage from outside.

Classification of Primary Cicatricial Alopecia

Lymphocytic primary cicatricial alopecia
• Chronic cutaneous lupus erythematosus (discoid lupus erythematosus)
• Lichen planopilaris
- Classic lichen planopilaris
- Frontal fibrosing alopecia
- Graham Little syndrome
• Classic pseudopelade of Brocq
• Central centrifugal cicatricial alopecia
• Alopecia mucinosa
• Keratosis follicularis spinulosa decalvans
Neutrophilic primary cicatricial alopecia
• Folliculitis decalvans
• Dissecting cellulites/folliculitis (perifolliculitis abscedens et suffodiens)
Mixed cicatricial alopecia
• Folliculitis (acne) keloidalis
• Folliculitis (acne) necrotica
• Erosive pustular dermatosis

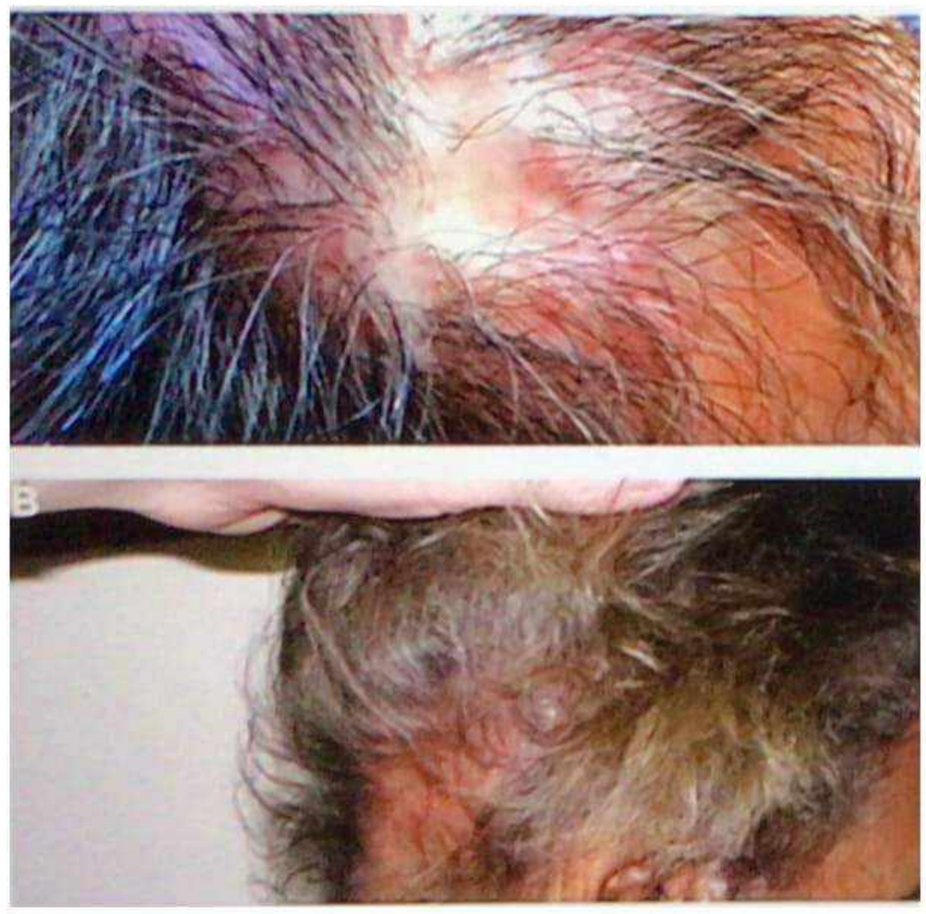
FOLLICULAR LP — 12:00

- Lichen Plano Pilaris
- Frontal Fibrosing Alopecia
- Graham Little Picardi Syndrome.

active space

Lichen Plano Pilaris (LPP)

- violaceous follicular papules with perifollicular scaling.
- Central scarring ⊕
- Itching, ± burning sensation.



On HPE :

- changes of LP around the follicles.

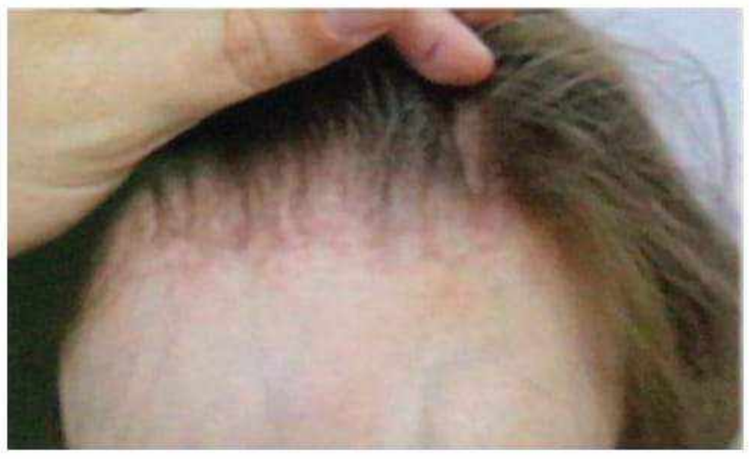
Treatment :

- Steroids ⟶ Topical / Oral / Intralesional
- HCQ's (Antimalarial)
- Acitretin

Frontal Fibrosing Alopecia

- Postmenopausal females
- Recession of hair line →

  Straight line x Bitemporal
- Follicular keratosis
- Cicatricial alopecia
- Loss of lateral eyebrows



active space

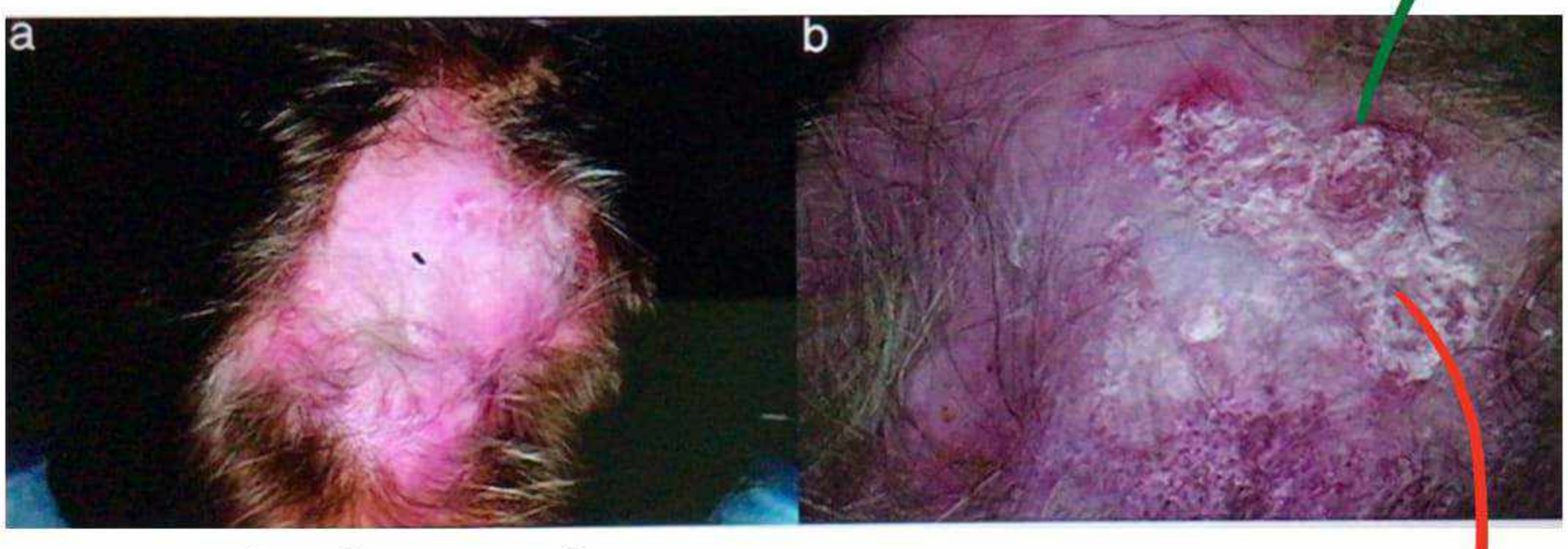
Graham Little Syndrome :

- a.k.a Graham Little Picardi Syndrome.
- Progressive cicatricial alopecia of scalp
- Non cicatricial loss of pubic and axillary hair.
- Extensive keratosis pilaris

Lupus Erythematosus :

- DLE → cause cicatricial alopecia
- SCLE
- Acute LE

} Non-cicatricial alopecia.



- Central Scarring
- Scaling, follicular plugging, telangiectasias.

DLE

active space

- Carpet tack / Tin Tack sign.

- Look for evidence of DLE in other body parts

↓

Concha of the ear → Schustner sign.

Scalp Biopsy

- Interface dermatitis ,
- Follicular plugging

DIF : ⊕ in DLE , ⊖ in LP

Treatment :

- Corticosteroids
- Antimalarials
- Sunscreens.

PSEUDOPELADE OF BROCQ

25:30

- Chronic disorder
- Idiopathic
- Cicatricial alopecia
- No inflammation
- Atrophy ⊕

active space

Box 89.2 Diagnostic criteria for pseudopelade of Brocq

Clinical criteria
- Irregularly defined and confluent patches of alopecia
- Moderate atrophy (late stage)
- Mild perifollicular erythema (early stage)
- Female : male ratio 3 : 1
- Long course (more than 2 years)
- Slow progression with spontaneous termination possible

Histological criteria
- Absence of marked inflammation
- Absence of widespread scarring (best seen with elastin stain)
- Absence of significant follicular plugging
- Absence, or at least a decrease, of sebaceous glands
- Presence of normal epidermis (only occasional atrophy)
- Fibrotic streams into the dermis

Direct immunofluorescence
- Negative (or only weak IgM on sun-exposed skin)

From Braun-Falco *et al.* 1986 [3].

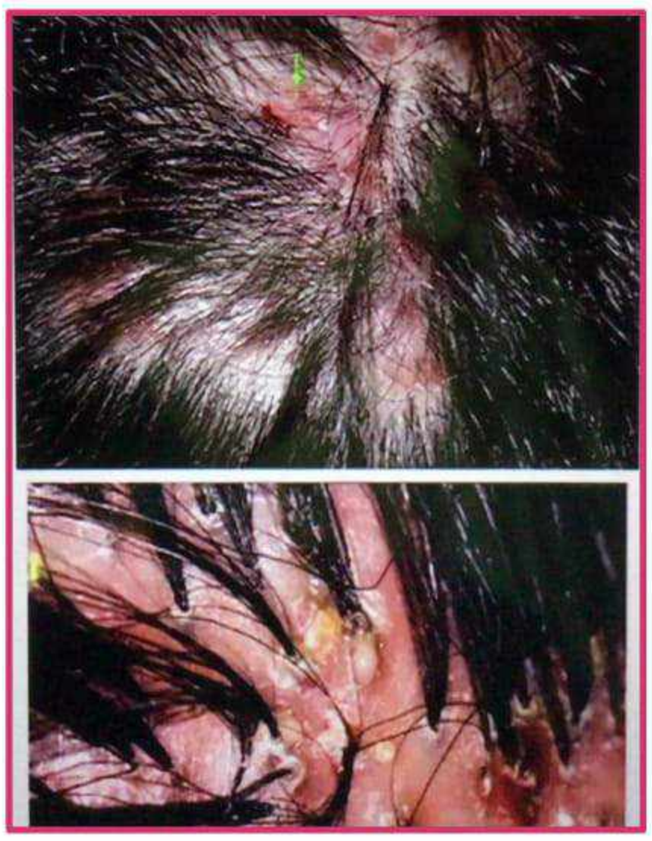
FOLLICULITIS DECALVANS / TUFTED FOLLICULITIS

28:45

- Inflammation of follicles 2° to a bacterial infection
- Mostly due to Staph.
- Follicular pustules and crusting (deep folliculitis)

↓

Boggy scarring



Treatment :

- Oral antibiotics

active space

- Cephalosporins
- Amoxicillins
- Clindamycin
- Rifampicin

Tufted folliculitis :

- 30-40 hairs coming out of a single hair follicle.

DISSECTING CELLULITIS

32:30

- It is a part of Triad :

  i) Dissecting cellulitis of scalp

  ii) Acne conglobata

  iii) Hidradenitis suppurativa.

  ⇓

  Follicular Occlusion Triad.

Traction alopecia

- due to traction (Hair styles)
- Site : Bitemporal
- Treatment : Counsel the patient

active space

EXCESSIVE HAIR GROWTH

35:10

Hypertrichosis

- Not androgen dependent
- It can be :
  - i) Congenital
  - ii) Acquired : Drugs,
    Systemic illness

Hirsuitism

- Androgen dependent

Hirsuitism

- Increased hair growth in androgen dependent areas which is socially unacceptable
- seen in females

Pathophysiology :

- Conversion of vellus hair to terminal hair
- Androgen dependent

Causes :

- Androgen production from → Adrenals
  → Ovaries.
  - i) PCOS

ii) Congenital Adrenal Hyperplasia

iii) Virilizing tumors

iv) SAHA → Seborrhoea Acne Hirsuitism and Androgenetic Alopecia.

v) Idiopathic

- Scoring: Ferriman Galleway Scoring

Investigations:

- Rule out
  - i) PCOS
  - ii) LH
  - iii) Prolactin
- USG lower abdomen.
- Adrenal causes → i) DHEAS
  - ii) 17-OH P
- Total testosterone
- TSH

Management: counsel

i) Topical - Eflornithine

active space

ii) Oral →

- Anti-androgens → i) Cyproterone Acetate
  ii) Spironolactone
  iii) OCP's
  iv) 5α Reductase ⊖

iii) Physical modalities :

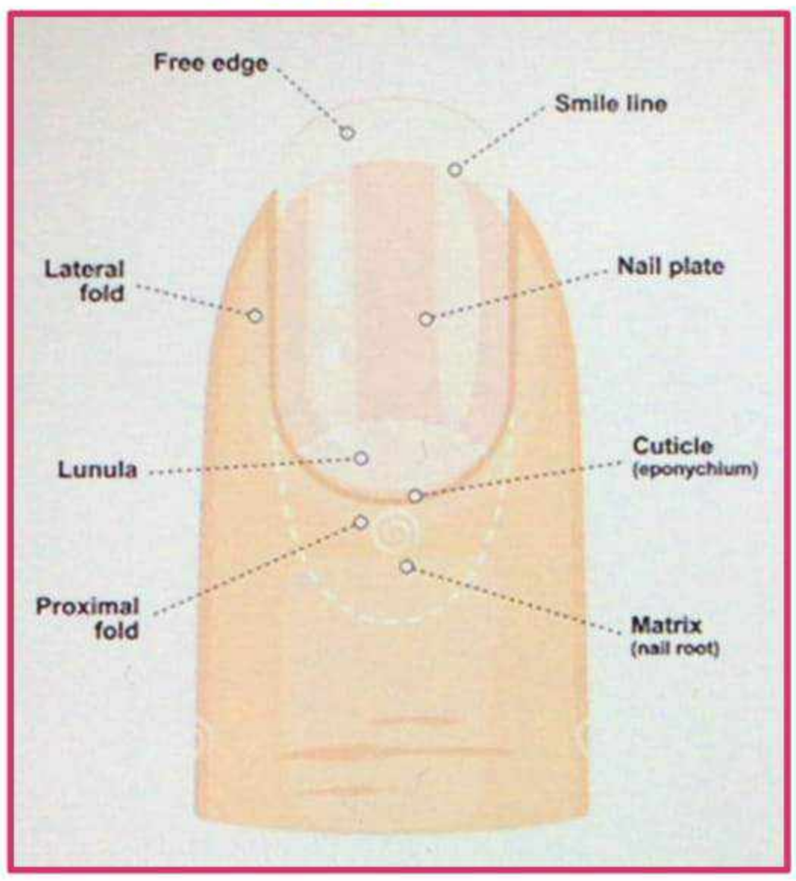
- LASER
  - diode
  - long pulsed Nd Yag
- Electropilation.

active space

Nail Disorders

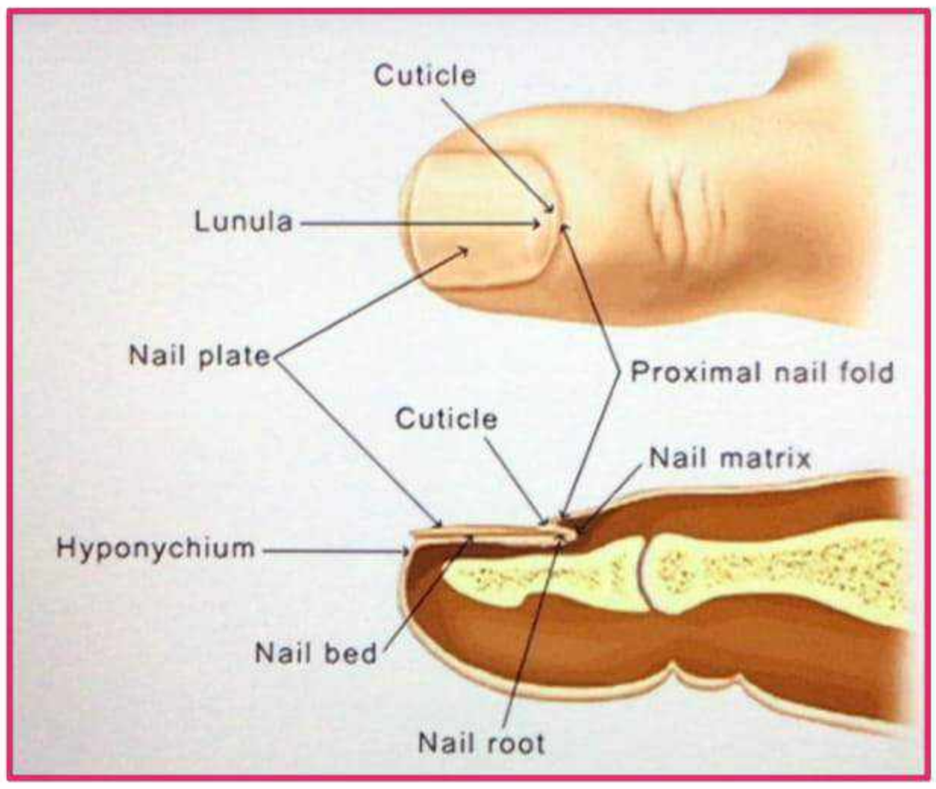
- Nail derived from Ectoderm.
- composed of Hard Keratin.
- Functions :
  - cosmetic purpose
  - protecting nail bed
  - Barrier function.
- Growth Rate :
  - 3mm / month ⟶ Fingernail
  - 1mm / month ⟶ Toenail

Parts of Nail



- Linula → semi-circular reddish area
- Cuticle → protects nail matrix

- **Nail bed:** skin below nail bed

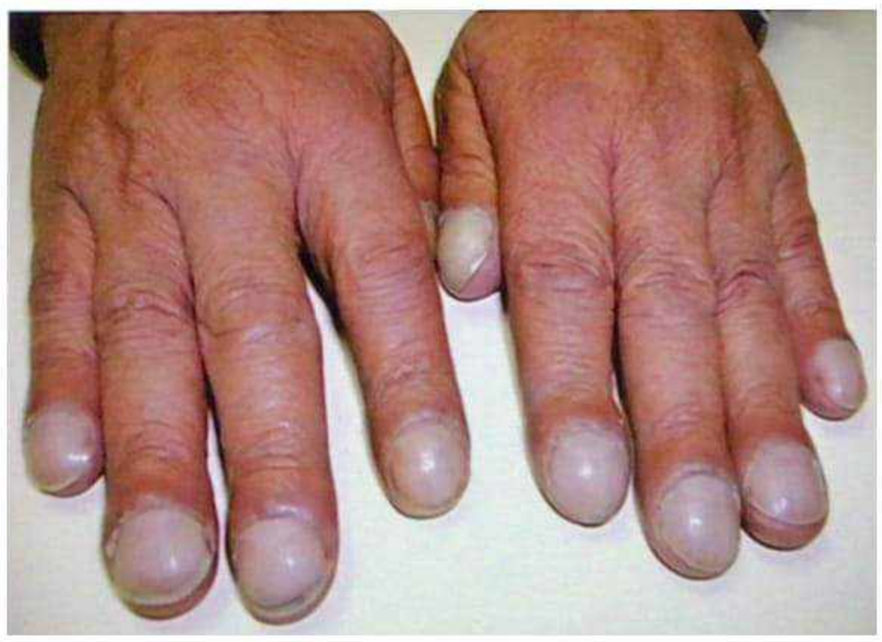


NAIL CHANGES IN SYSTEMIC DISEASES

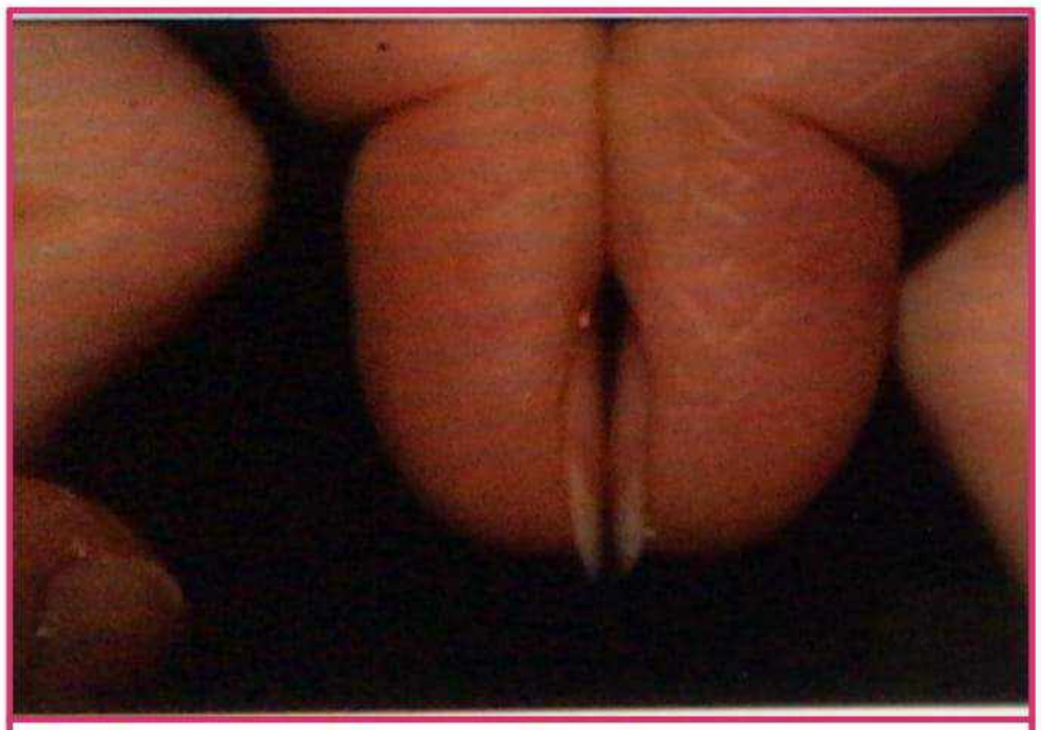
05:05

Clubbing :

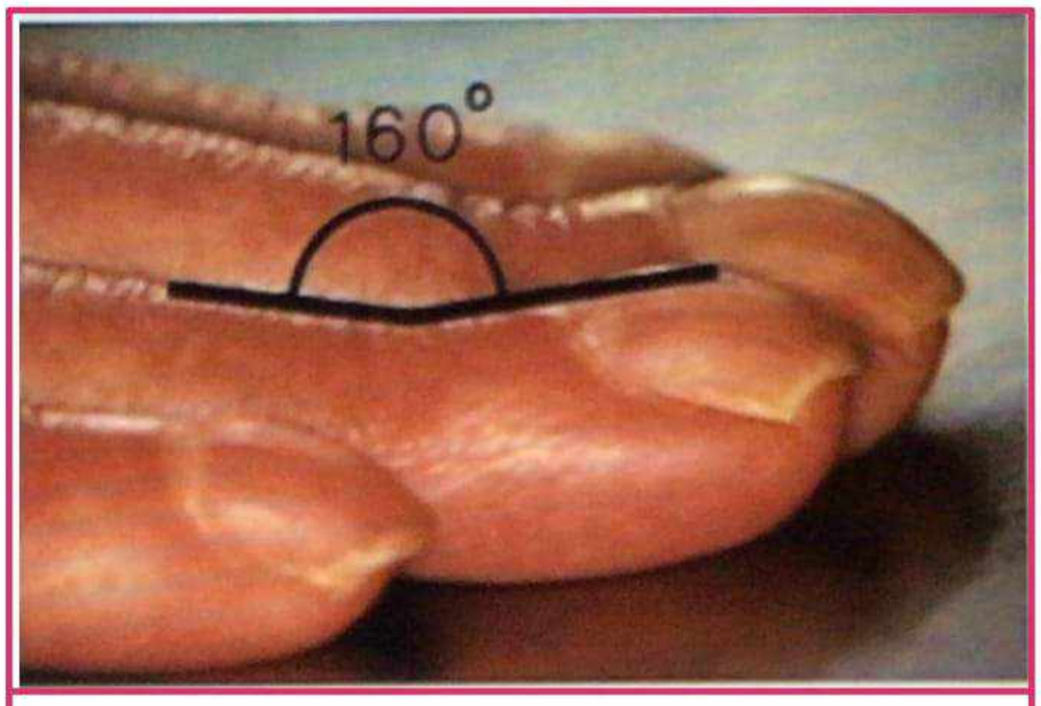
- Increased transverse and horizontal curvature of nail plate along with soft tissue hypertrophy.



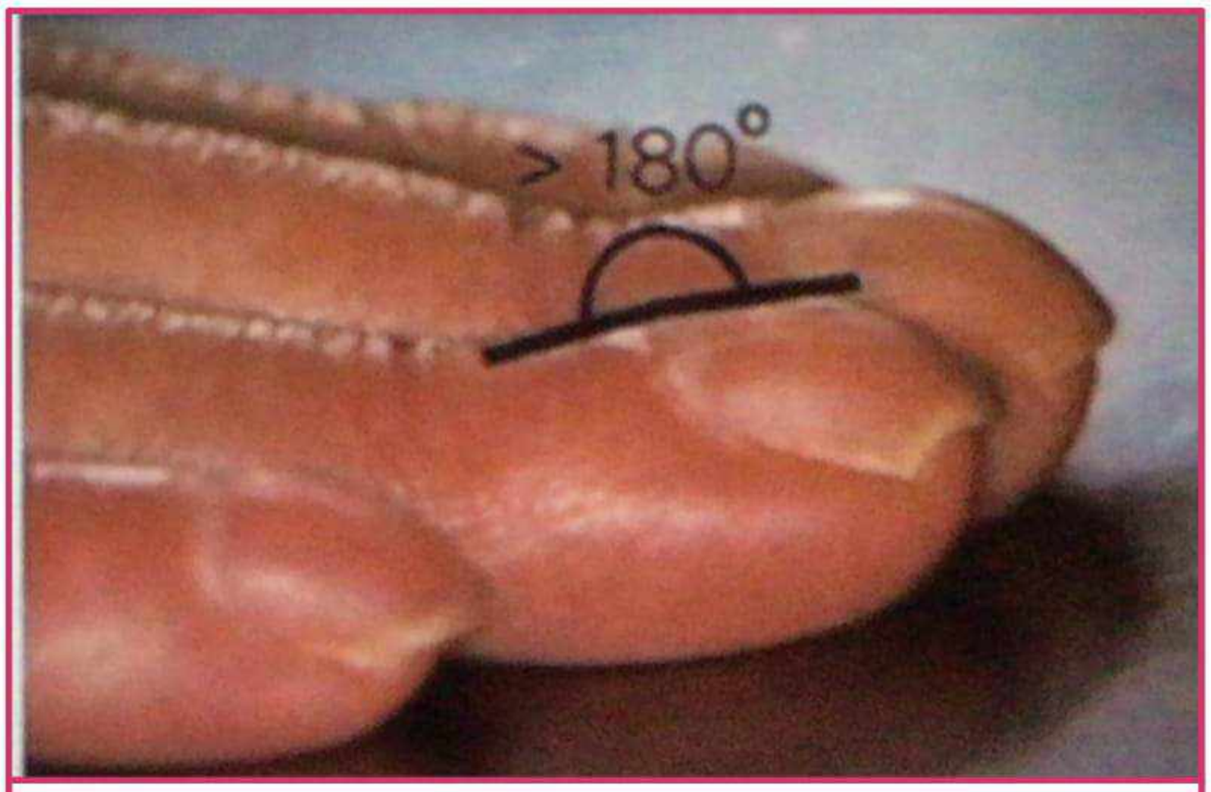
- Cause :
  - Hereditary
  - Acquired → i) Lung disorders (Respiratory ailments)
    ii) Cardiac disorders.



Schamroth's window is seen clearly in this image of normal nails.



Clubbing : Curth's modified profile sign.



Clubbing : Lovibond's profile sign

Angle is normally < 160° but exceeds 180° in clubbing.

Koilonychia :

- spoon shaped nails

Causes :

- Idiopathic
- Iron deficiency Anemia (MC)
- Haemochromatosis

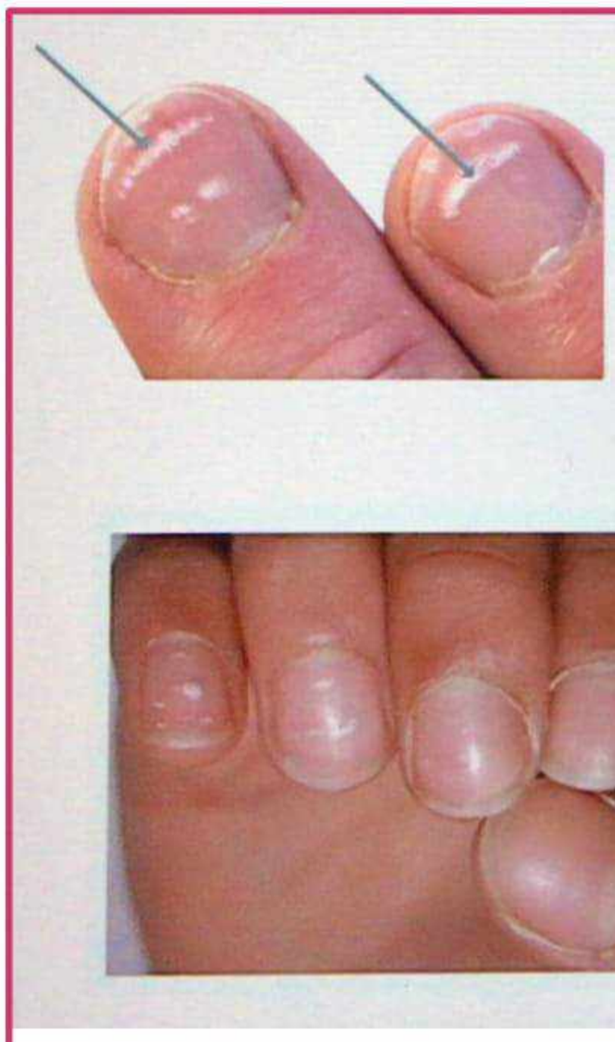
Beau's Lines :

- Transverse grooves on nail plate
- They signify arrest in nail growth.
- Causes :
  - M.I, Major Surgery, Prolonged Illness.
- Width of Beau's line → duration of insult

Leukonychia

- White lines on the nails
- True → defect in nail matrix
- False → defect in nail bed.

active space



True leukonychia :

i) Hereditary

ii) Transverse → Mee's Lines

- similar to Beau's lines
- seen in - Chemotherapy
  - Chronic Arsenic Poisoning

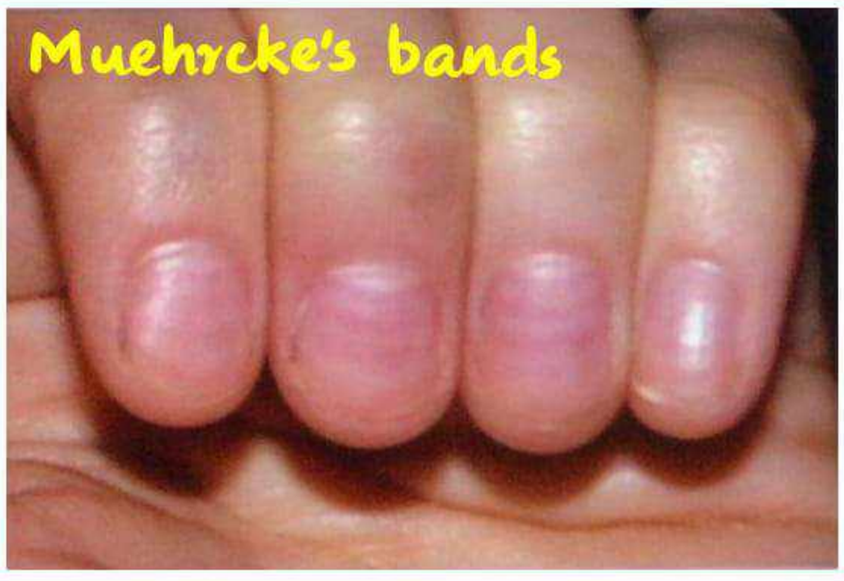
iii) Punctate :

- Alopecia areata
- Trauma.

False Leukonychia

Muehrcke's bands :

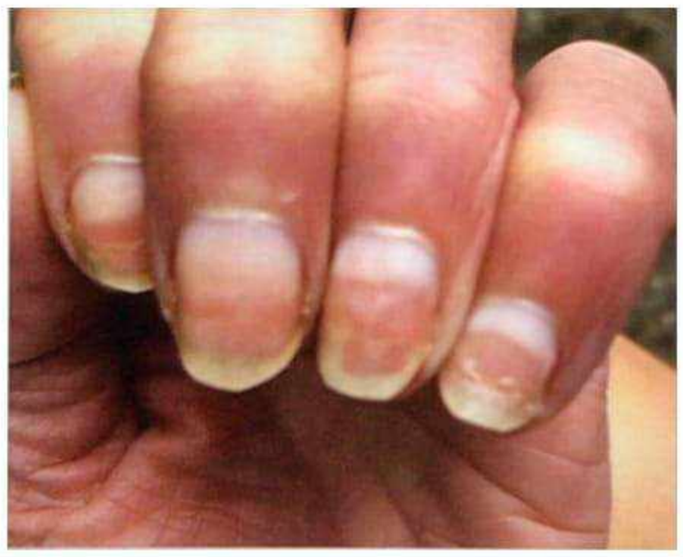
- paired white bands.
- seen in Hypoalbuminemia



active space

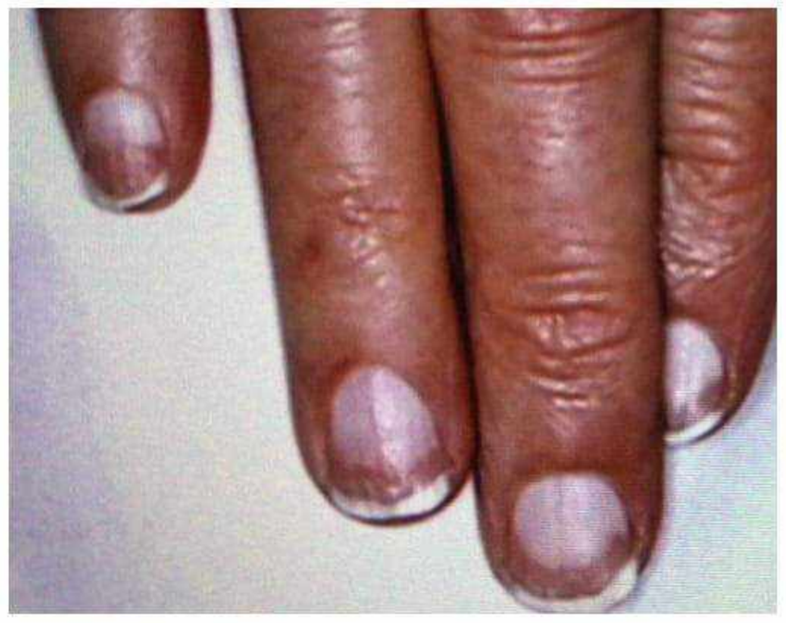
Terry's Lines

- Proximal nail whitening and distal part of nail normal.
- seen in - Cirrhosis , CHF



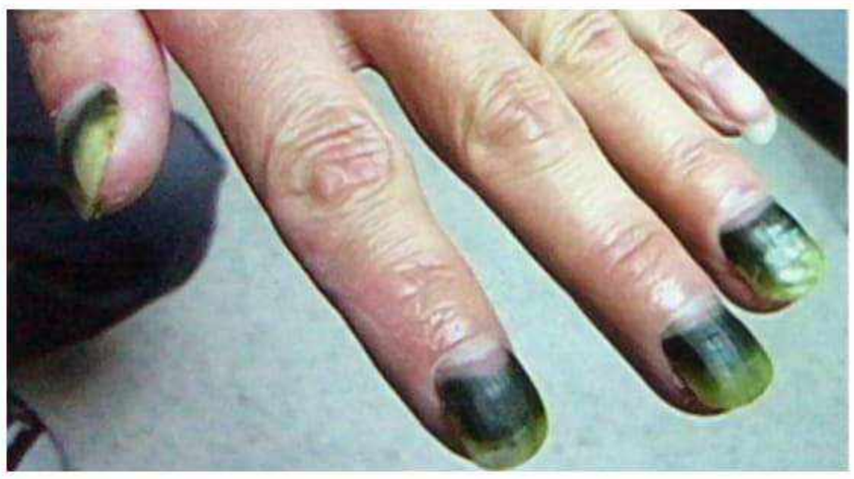
Half and Half Nails / Lindsay Nails.

- seen in Chronic Renal Failure.

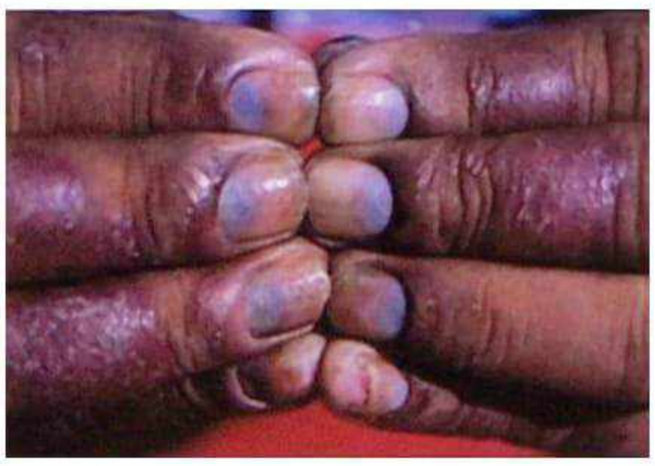


Chromonychia:

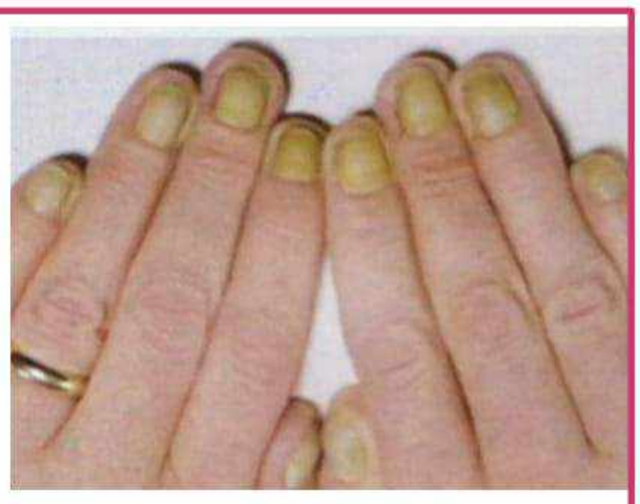
- Pigment / colour in Nail.



Green Nail - Pseudomonas



Blue lunula - Wilson's disease



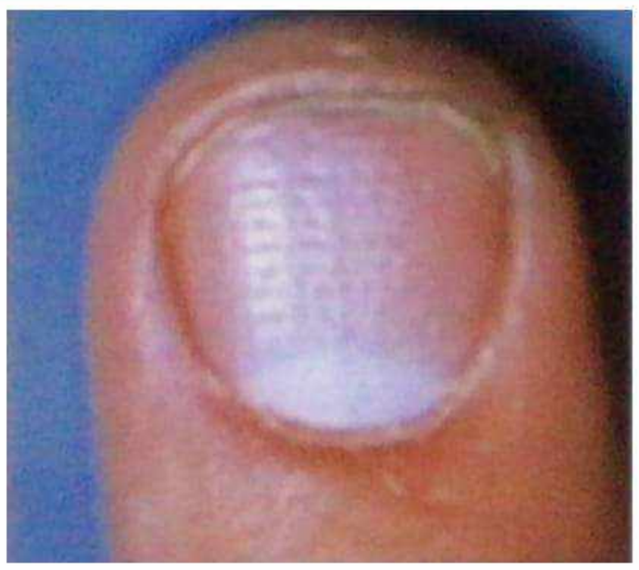
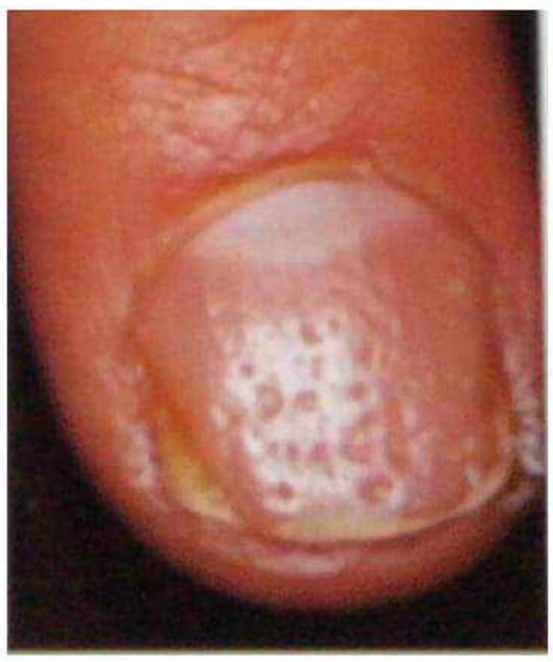
Yellow nail - Pulmonary edema or effusions.

Red lunula → SLE

active space

Pitting :

- Parakeratosis in the nail matrix
- seen in Alopecia areata.

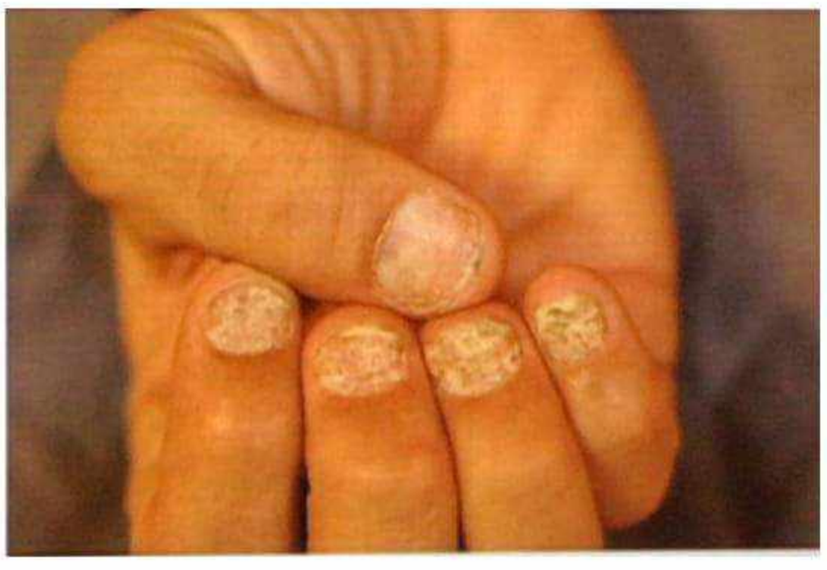
Alopecia areata.	Psoriasis
	
• Regular smooth shallow pits • Geometric appearance	• Coarse, deep, irregular pits.

Trachyonychia :

- 20 nail dystrophy
- Nails are Roughened surface (sandblasted nail)
- seen in :

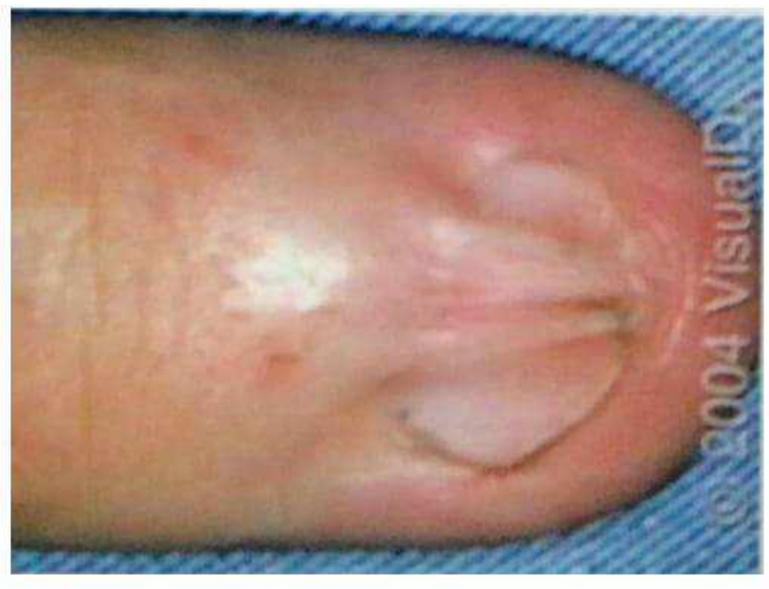
  i) Isolated

  ii) Lichen planus, Alopecia areata. (MC)



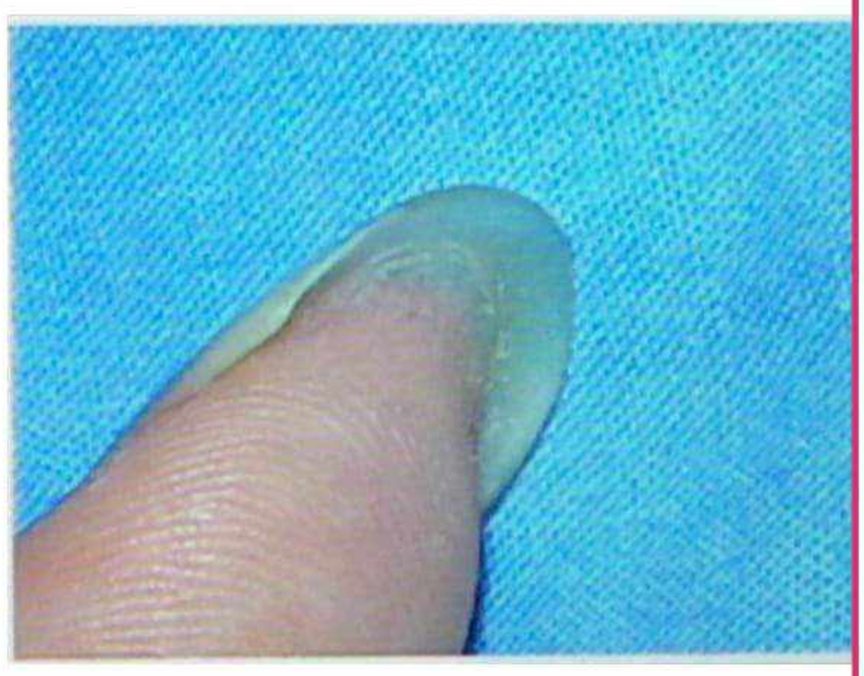
active space

Pterygium :



- Wing shaped appearance
- Extension of Proximal Nail Fold to Nail plate
- causes permanent dystrophy of nail.
- Lichen planus - (Mc) cause.

Inverse Pterygium :



- Nail bed grows and obscures distal groove
- seen in
  - Connective Tissue Disorders.
    - Systemic Sclerosis
    - Raynaud's phenomenon
    - SLE

active space

Paronychia

- Inflammation of nail folds
- It can i) Acute
  ii) Chronic.

→ Acute Paronychia :

- Predominant infection with Staph
- Presents as : swelling, tenderness and pain of nail folds, pus points
- Treatment :
  i) Oral Antibiotics
  ii) Drain pus

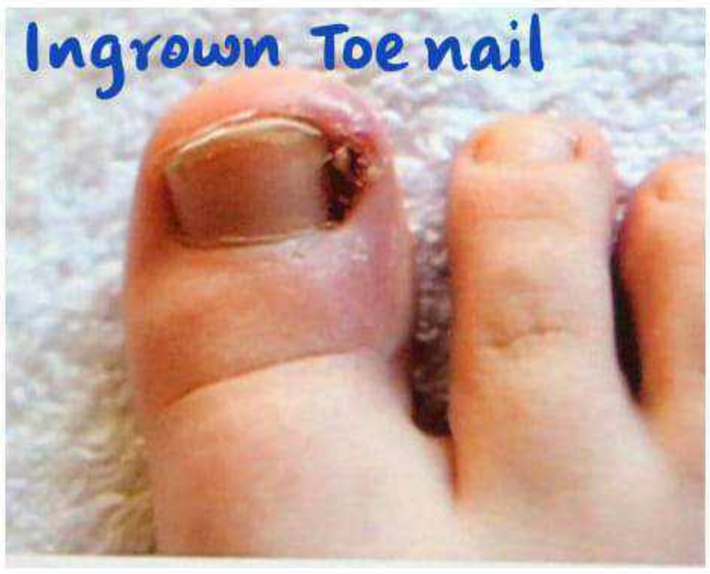
→ Chronic Paronychia :

- loss of cuticle
- chronic inflammatory dermatoses of nail folds
- seen in Housewifes / maids.
- Treatment :
  i) wear gloves
  ii) Moisturisers

iii) Topical steroids + Antibiotics + Antifungals.

- Counselling

Ingrown Toe nail



- improper cutting of nail
- improper footwear
- **Treatment :**

  i) Oral antibiotics

  ii) Anti-inflammatory

  iii) Surgery → Remove the nail - cauterize the nail bed.

Other Nail Disorders :

- Onychomycosis
- Nail psoriasis
- Nail LP.

active space

Eczema (Part- 1)

- derived from "To boil"
- **Dermatosis :** any skin condition
- **Dermatitis :** any inflammation
- **Eczema :** Type of dermatitis.

"All eczemas are dermatitis, but not all dermatitis are eczema"

Classification of Eczema

Exogenous

- cause is outside the body
- Types :
  - i) Irritant contact dermatitis
  - ii) Allergic contact dermatitis
  - iii) Phytodermatitis
  - iv) Photodermatitis

Endogenous

- cause is inside the body.
- Types :
  - i) Atopic dermatitis
  - ii) Seborrheic dermatitis
  - iii) Pityriasis alba
  - iv) Discoid eczema
  - v) Hand eczemas
  - vi) Asteatotic dermatitis
  - v) Venous eczemas
  - vi) Lichen Simplex chronicus
  - vii) Prurigo nodularis

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Clinical Stages

- Stages of Skin Inflammation.

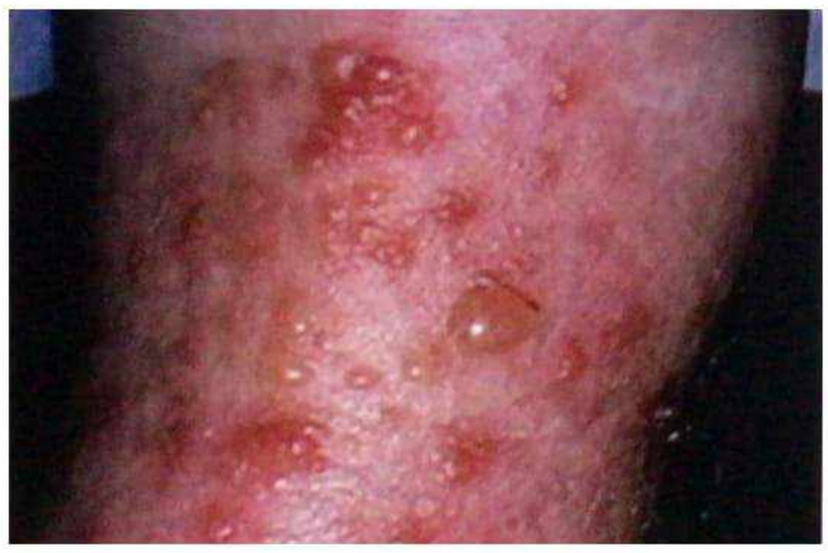
  i) Acute

  ii) Subacute

  iii) Chronic

Acute Eczema

- Intense itching
- Clinical features :
  - Erythema
  - Edema
  - Oozing.



On HPE :

- Ballooning (intracellular edema) } accumulation of fluid.
- Spongiosis (Intercellular edema) }

Subacute Eczema

- Mild itching
- Irritation ⊕

active space

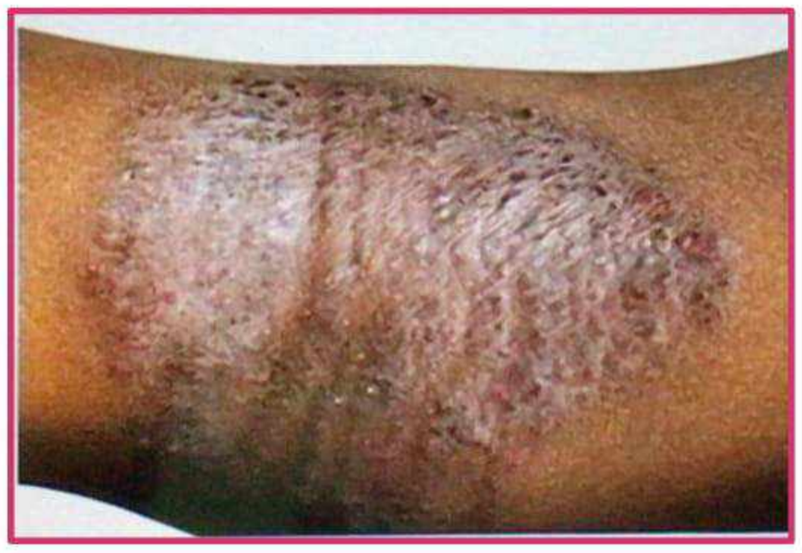
Clinical features:

- less erythema
- Scaling
- Crusting

Chronic Eczema

- due to constant scratching
- Hyperpigmentation
- Increased skin thickenings
- Thickening / Induration of skin

} Lichenification.



- On HPE: Acanthosis ⊕ → Thickening of Stratum spinosum.

Exogenous Eczemas

Contact dermatitis → Irritant
Contact dermatitis → Allergic.

IRRITANT CONTACT DERMATITIS

11:00

- Its non-immunological
- contact with a substance cause dermatitis.
- Happens within few minutes to hours of contact.

active space

- occurs with first exposure.
- Types : (Irritant Reactions)

  i) Symptomatic Responses : Chloroform, Methanol

  ii) Burns : Acid, Alkali

  iii) ICD : Acute/Chronic.

Acute Irritant Dermatitis :

- Acid, alkalis, solvents
- Depend upon :
  - Nature of substance
  - Amount of substance
- c/f :
  - Erythema
  - vesiculation
  - Edema
  - Tenderness
  - Pruritus
- usually occupational

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Chronic Irritant Dermatitis :

- Depends on :

  i) Nature of substance + patient characteristics

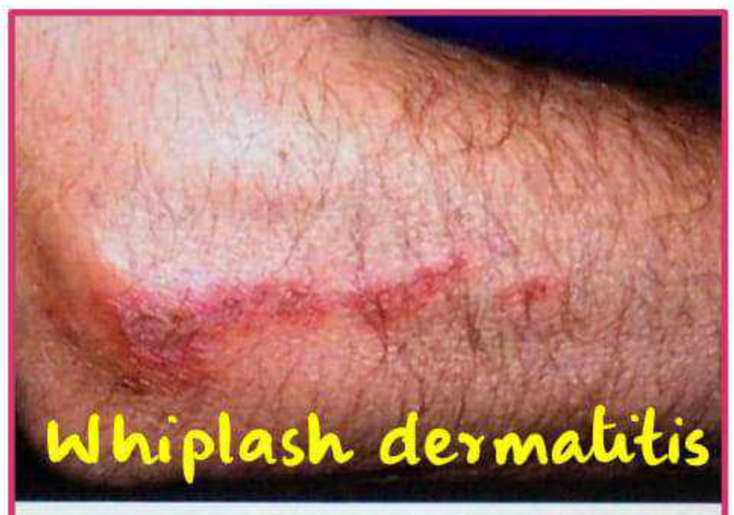
  - dry skin
  - Atopic
  - Wet work

  → Detergents → Housewifes → Housewife dermatitis.

  → Cutting oils → Oil machinitis.

Paedrous Dermatitis :

- a.k.a Blister Beetle Dermatitis
- due to bite of Blister Beetle
- Irritant : Paedrin
- C/F :
  - Lesion develops overnight
  - More common during monsoon season.
  - Erythematous vesicular rash
  - Whiplash dermatitis
  - Kissing lesions.


Whiplash dermatitis

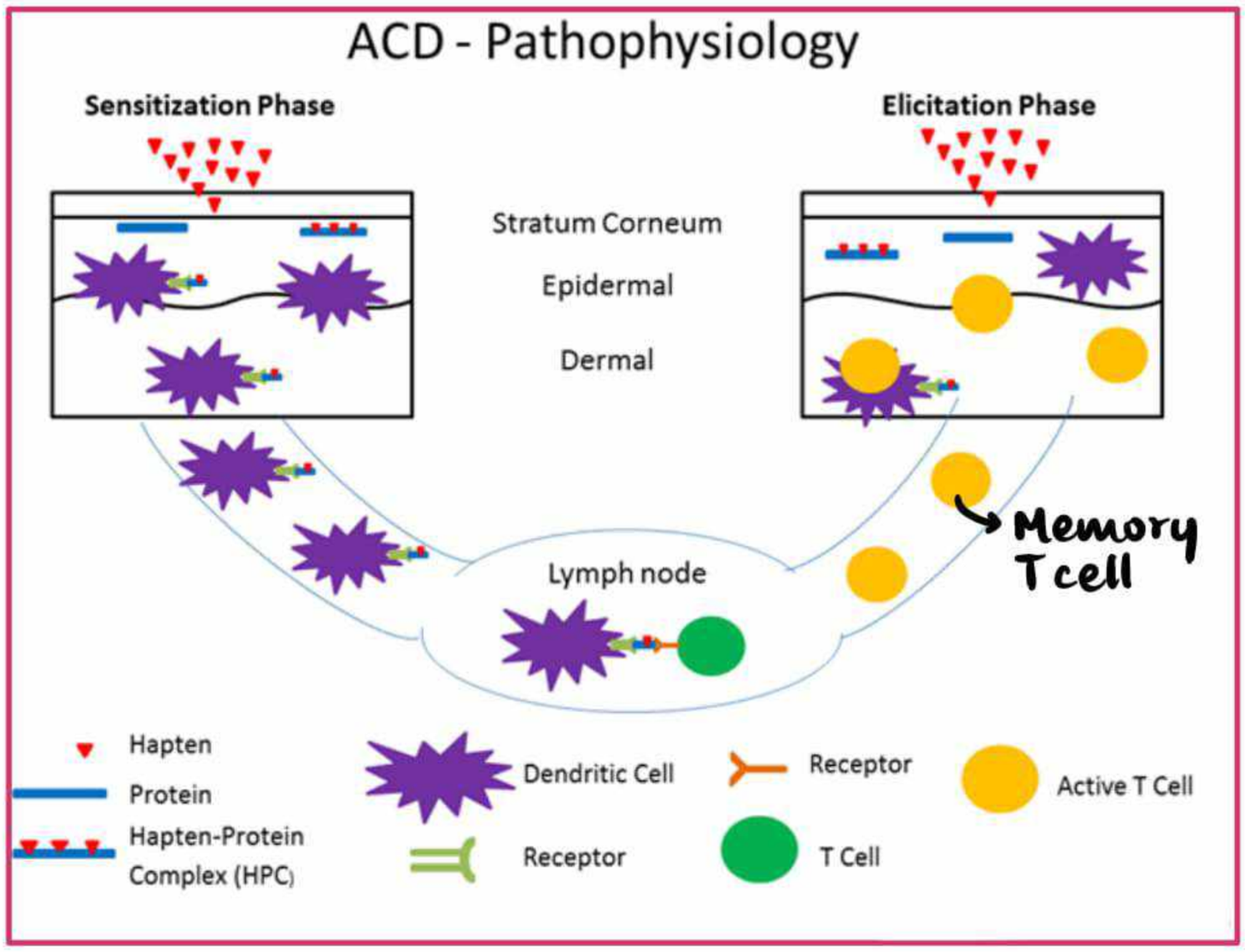

Kissing lesions.

Treatment :

- Topical steroids
- Topical antibiotics
- Antihistamines.

Allergic Contact Dermatitis

- Immunological in nature
- Type IV HSR
- Sensitisation ⟶ Elicitation
- occurs on 2nd / subsequent exposure (c/f)



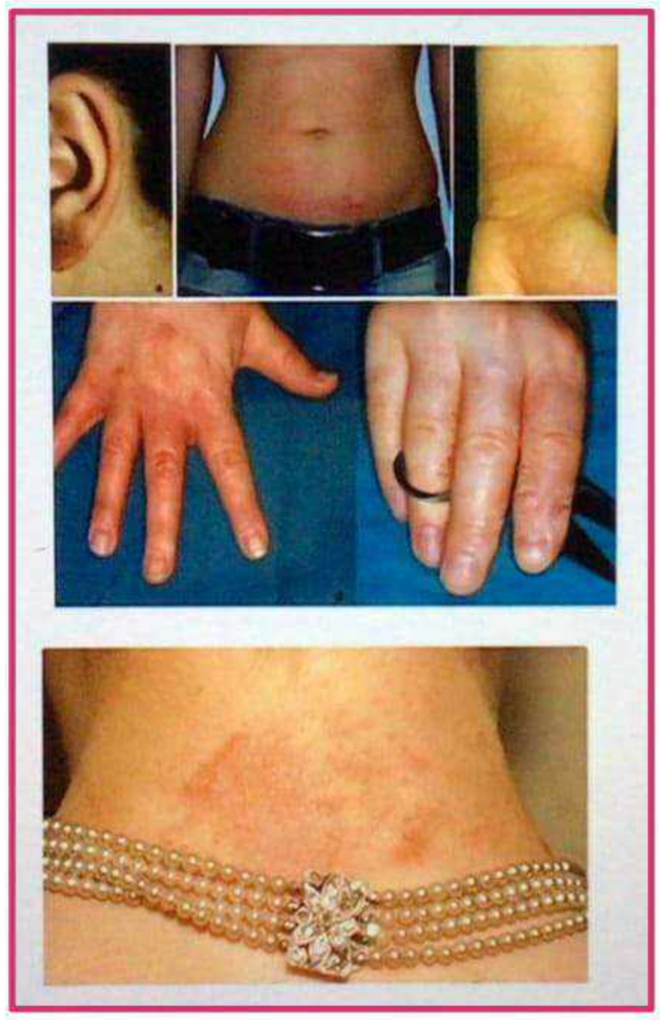
active space

Clinical features :

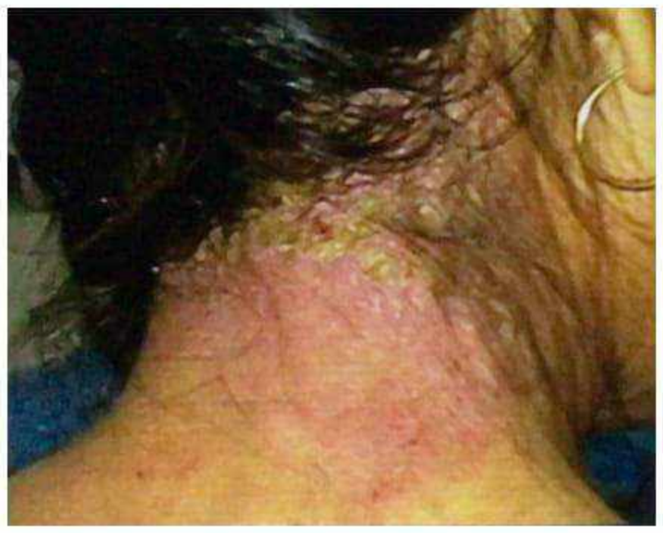
- can present as acute or as chronic

Allergens :

- Metals → Nickel (MC cause of ACD in India)
- Cosmetics
- Wearing allergens
- Medicines / drugs.
- Chromates → cement, shaving cream,
  or
  Potassium dichromate — detergents, shoes.
- Rubber → Gloves, Shoes, Belts.
  or
  Latex
- Cosmetics →
  - Fragnances and preservatives
    (MC cause of ACD due to cosmetics)
  - Lipstick, nail polish
  - Hair gels.



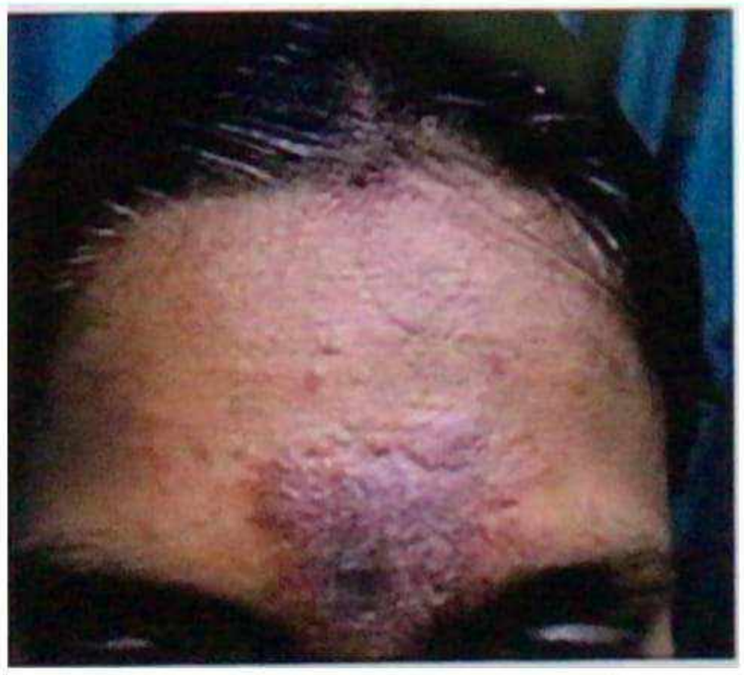
active space



- due to Hair dye

  i) Natural → Henna.
  ii) Semi synthetic
  iii) Synthetic.

→ PPD (Paraphenyldiamine)



Bindi dermatitis

- It is due to

  PTBP → ParaTertiary Butyl Phenol.

- (MC) metal allergen : Nickel
- (MC) allergen in India : Nickel
- (MC) Topical medicine causing ACD : Neomycin

Difference b/w ACD and ICD :

ICD	ACD
• dependent on concentration of substance • Non-immunological	• Not dependent on concentration (little amount) • Type IV HSR

active space

ICD	ACD
• happens with mins to hrs	• within 24 to 48 hrs (sensitization → Elicitation)
• occurs with first exposure	• occurs with 2nd or subsequent exposure
• Localised to the area of contact	• Disseminate from area of contact.

PHYTODERMATITIS

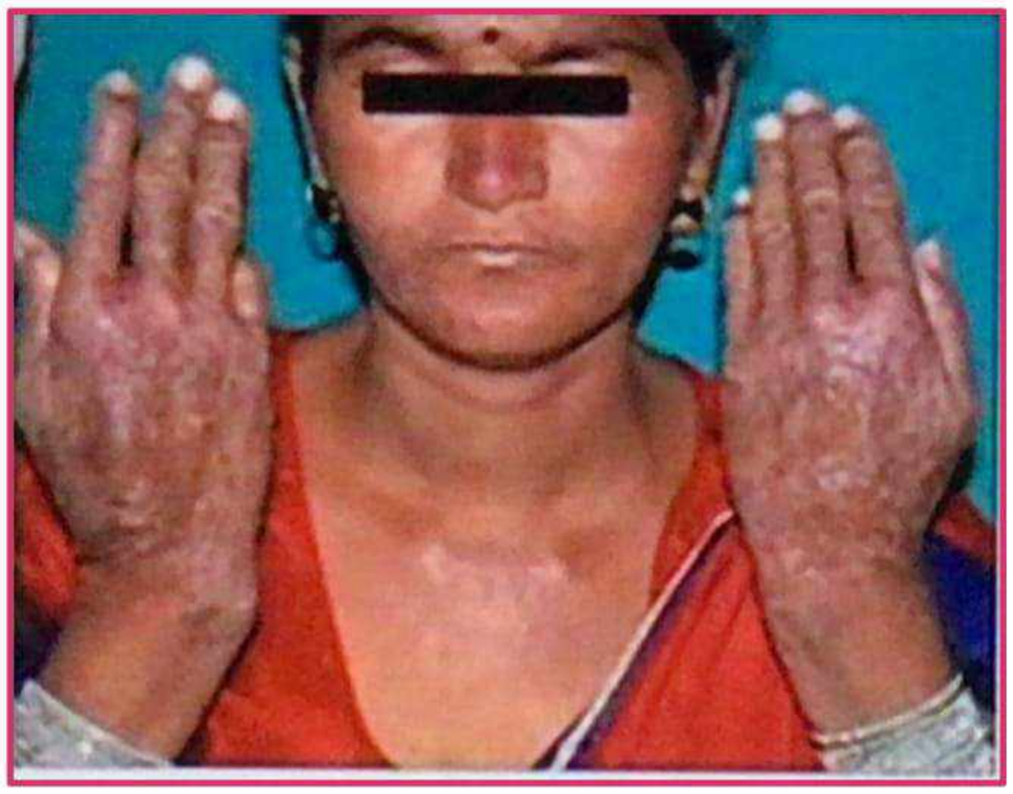
31:32

- occurs due to plants
- (MC) cause in India: Parthenium hysterophorus or Congress grass
- Allergen : Sesquiterpene lactones
- Population affected → Rural farmer.

C/F :

- Subacute eczema on exposed parts of body.
- Type of Air borne Contact dermatitis
- 1st site → Periorbital area (upper eyelids)
- Nose tip sign

active space



Treatment :

- Occupation change / change of place
- Reduce exposure to allergen
- Topical steroids
- Oral steroids.
- Azathioprine
- Course → c/c Relapsing course (seen more in summers, monsoons)

Systemic Contact Dermatitis

- Topical contact with an allergen → Sensitization

↓

Oral / Systemic contact with same allergen → Oral, Injection → Elicitation

↓

active space

↓

Dermatitis → at the site of previous attack

→ Generalised rash.

Common Allergens:

i) Penicillin

ii) Streptomycin

iii) Neomycin

iv) Nickel

PHOTODERMATITIS

44:10

- Eczema due to sun exposure
- can be i) Phototoxic reaction
  ii) Photoallergic reaction
  iii) PMLE → Polymorphous Light Eruptions.

	Phototoxic	Photoallergic
Incidence	Common	Less common
Mechanism	Non-Immunological	Type IV HSR
Onset on UV exposure	Minutes to days	24-48 hrs
Morphology of the lesion.	Sunburn	Eczematous
Diagnosis	Clinically diagnosed	Photopatch testing.

active space

Agents commonly producing phototoxic and photoallergic reactions.

Phototoxic agents	Photoallergic agents
Topical agents	**Topical agents**
Psoralens	Sunscreens
Tar	Fragrances
	Antibacterials → Sparfloxacin, Quinolones.
Systemic agents	Plants of Compositae family
Psoralens	
Antimicrobials: Nalidixic acid, doxycycline, quinolones	**Systemic agents**
Anticancer drugs: Fluorouracil, vinblastine	Antifungals: Griseofulvin
Antidepressants: Amitryptiline	Antimicrobials: Quinolones, sulfonamides
NSAIDs	NSAIDs: Ketoprofen,
Others: Phenothiazines, amiodarone, frusemide	Others: Phenothiazines, thiazides

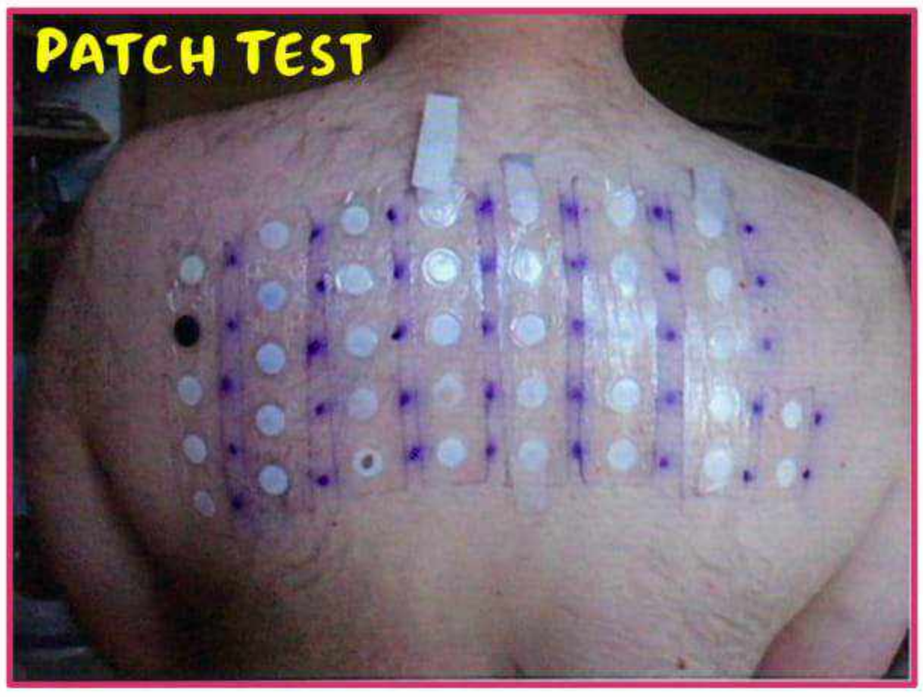
PATCH TEST

- Principle : simulate ACD
- Type IV Hypersensity Reaction
- Indications :

  i) To identify the allergen.

  ii) To diagnose ACD

  iii) To differentiate between ICD and ACD.



active space

In Finn chambers

↓

Put different allergens on them

↓

upper back

↓

Read after 48 hrs

↓

2nd Reading done after 96hrs ideally. (4-7days)

PHOTOPATCH TEST

- photoallergic dermatitis
- put the patches in duplicate.

↓

Shield them

↓

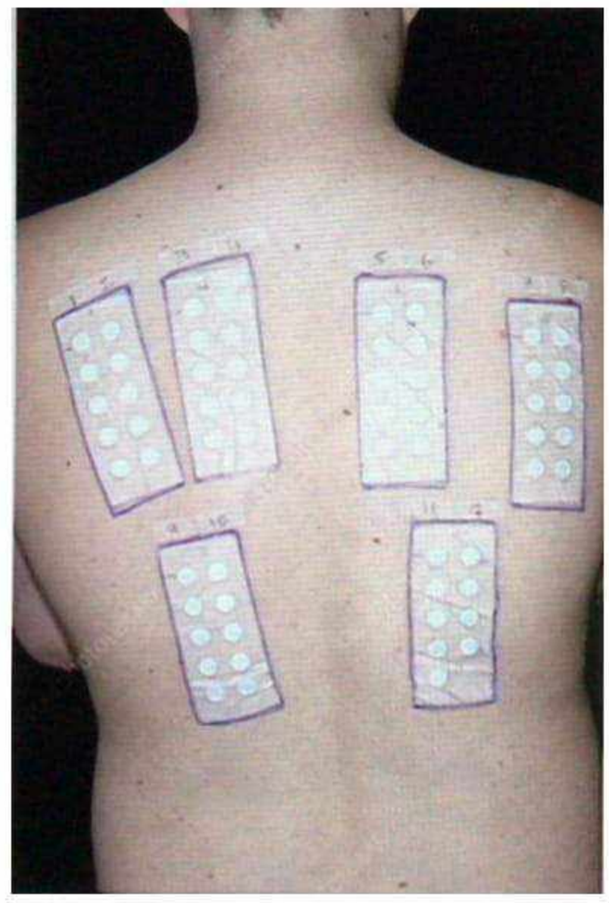
Read at 48hrs

↓

Shield one side, on other side give UV A Radiation

↓ ↓ ↓

ACD Read at 96 hrs Photoallergens.



Management

- Remove the allergen
- Antihistamines
- Steroids → i) Topical
  ii) Systemic
- Adjuvants :
  - Cyclosporine
  - Azathioprine

active space

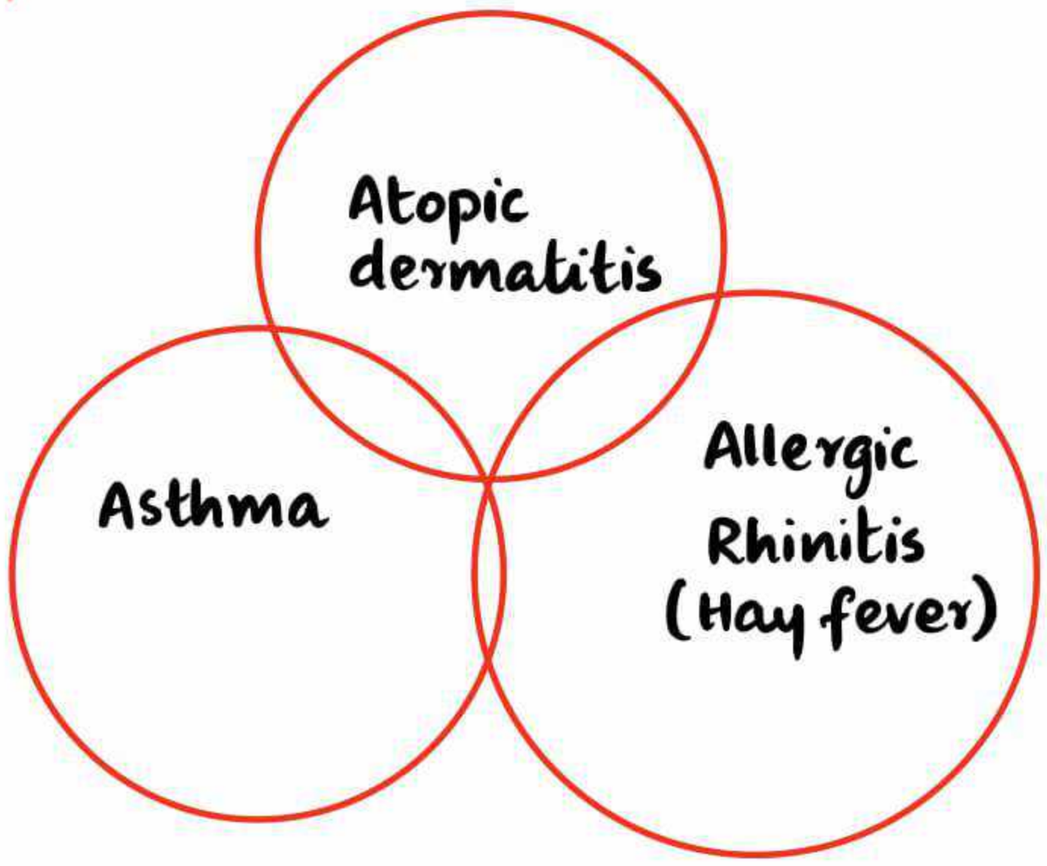
Eczema (Part- 2)

Endogenous Eczema :

ATOPIC DERMATITIS

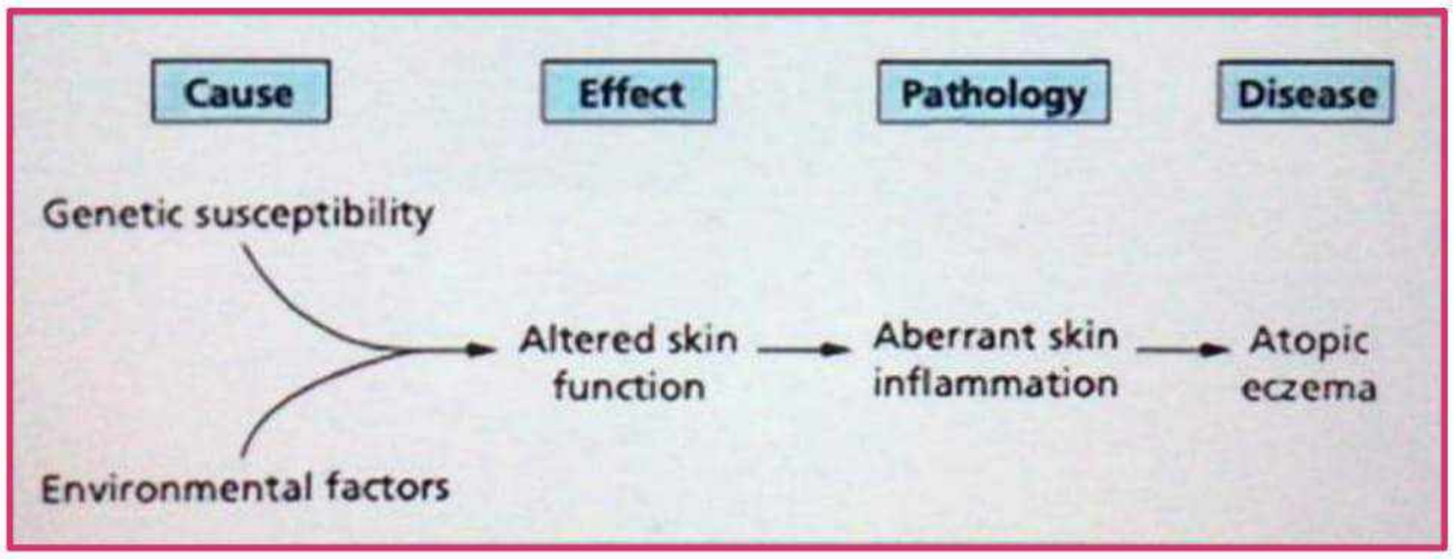
00:15

- Itchy condition
- Remission and Relapses ⊕
- Dermatitis seen in atopic individuals
- Mediated by Genetic , Immunological factors
- Itch that rashes
- Atopic Triad



- Personal history / Family h/o may be ⊕.

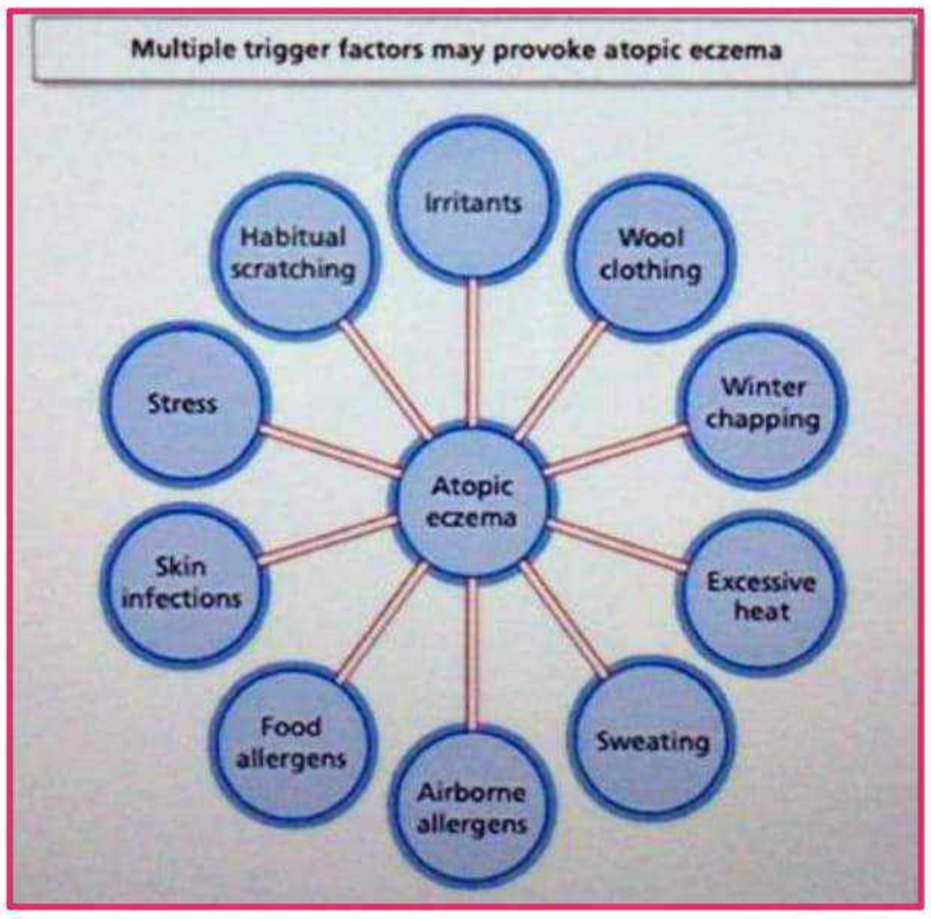
Pathogenesis

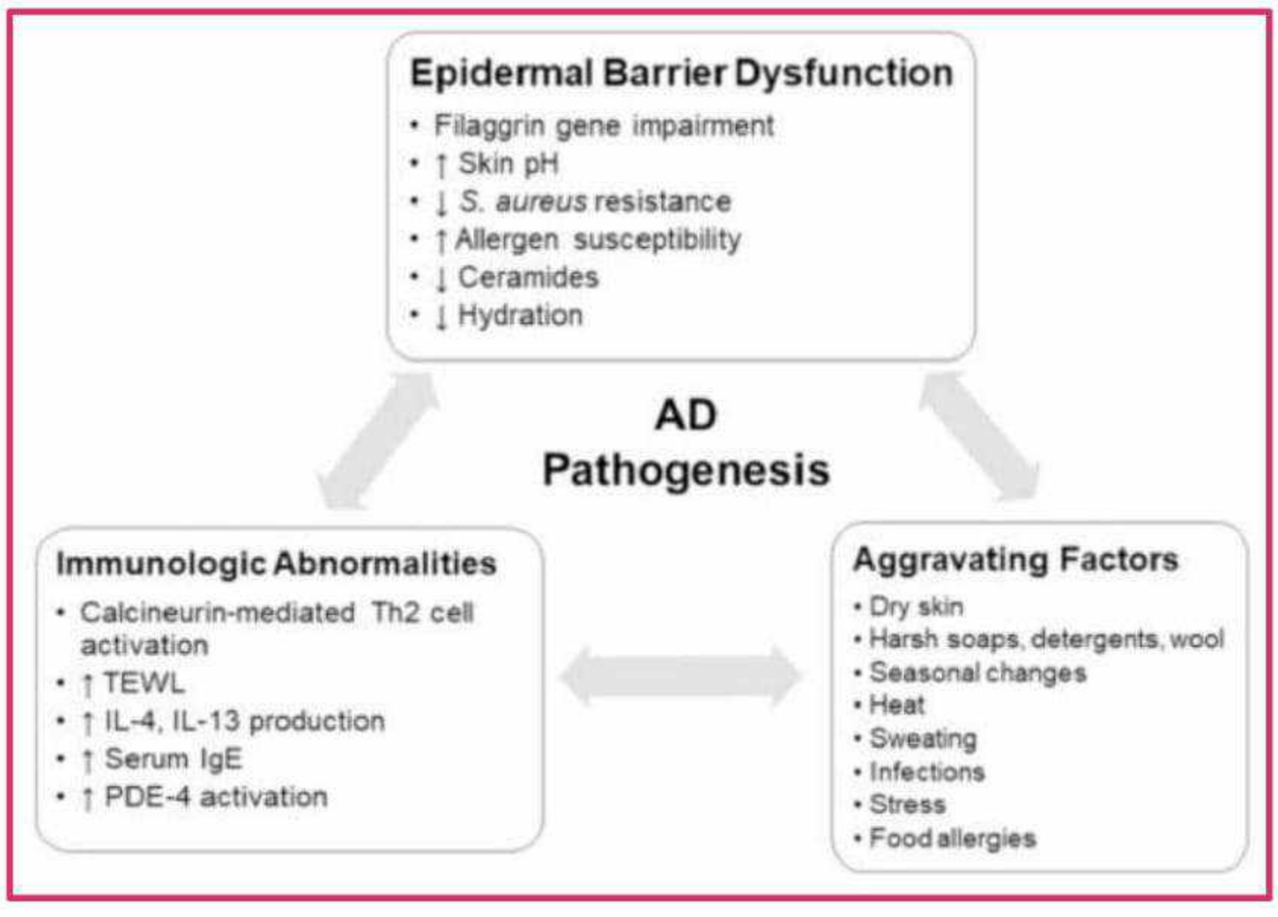


- **Defective Barrier:**
  - Defect in Fillagrin / Locirin
  - Decreased lipids / ceramides
  - Increased Transepidermal water loss (TEWL)
- **Genetic factors**
  - FLG gene defect → codes for Fillagrin → Aggregating Protein
  - Immune genes → Increased production of IL-4, IL-13 ↓ ↑ IgE and Eosinophil
- **Immunological factors:**
  - Shift to TH_2 pathway.
  - Increased IL-4, IL-13 → ↑ IgE → ↑ Eosinophils.

active space

- **Environmental factors:**

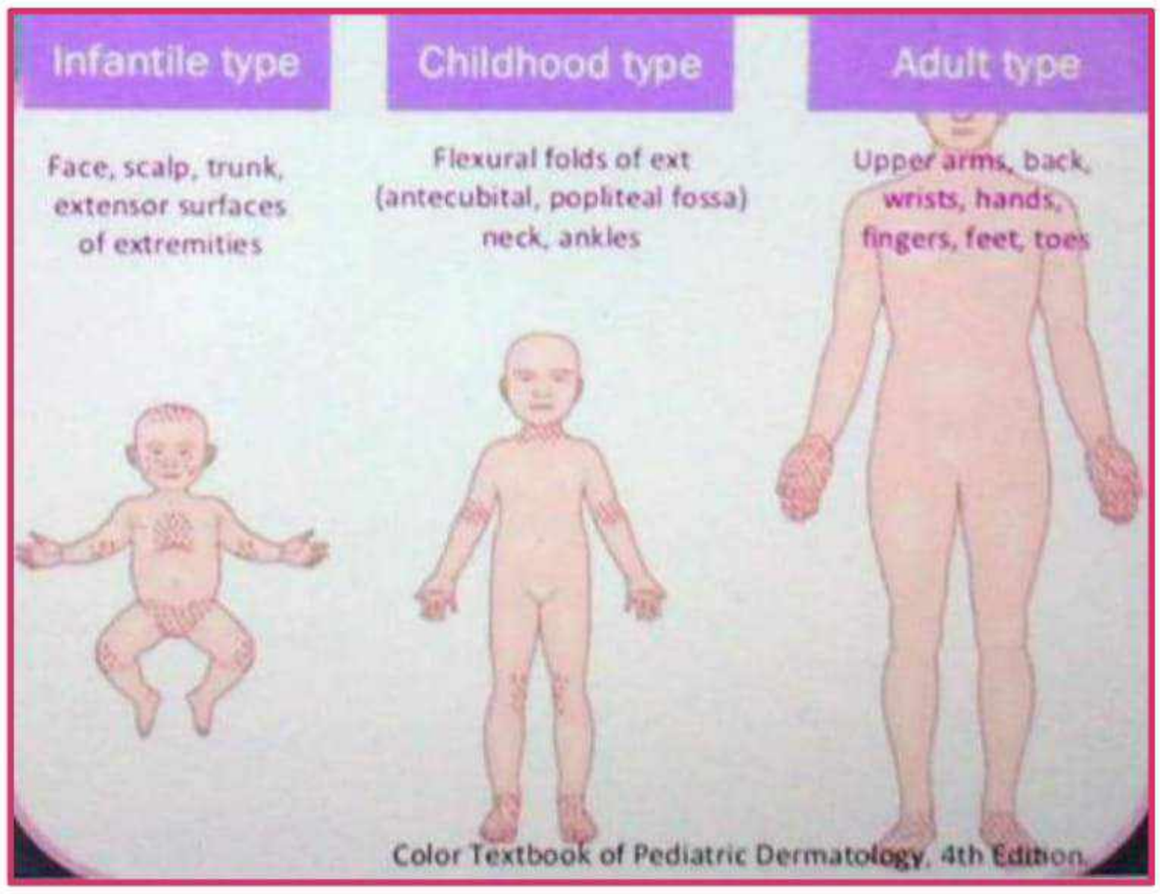




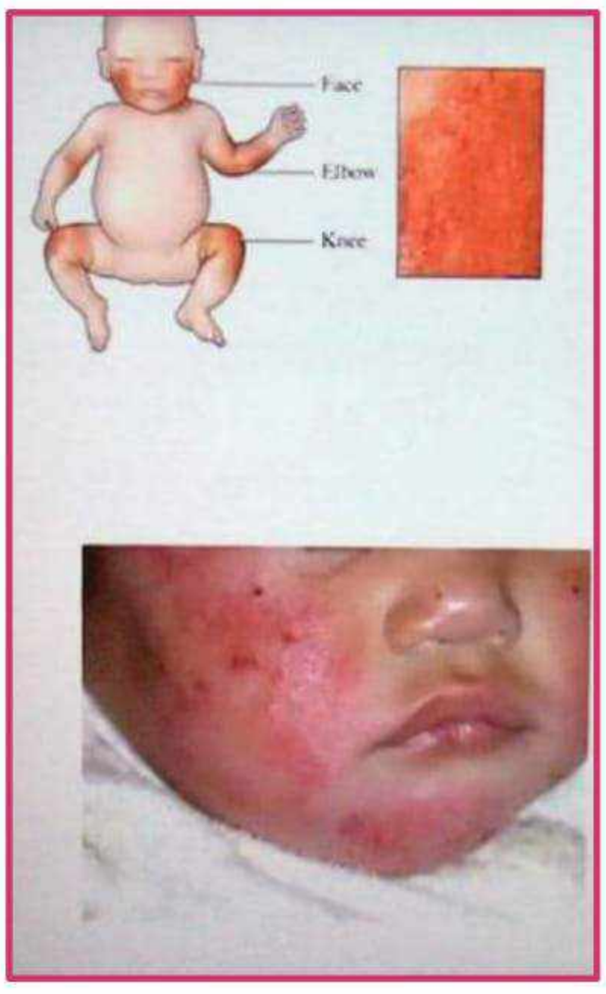
Hygiene Hypothesis: more hygienic → more chances of getting atopic dermatitis

active space

Clinical features :



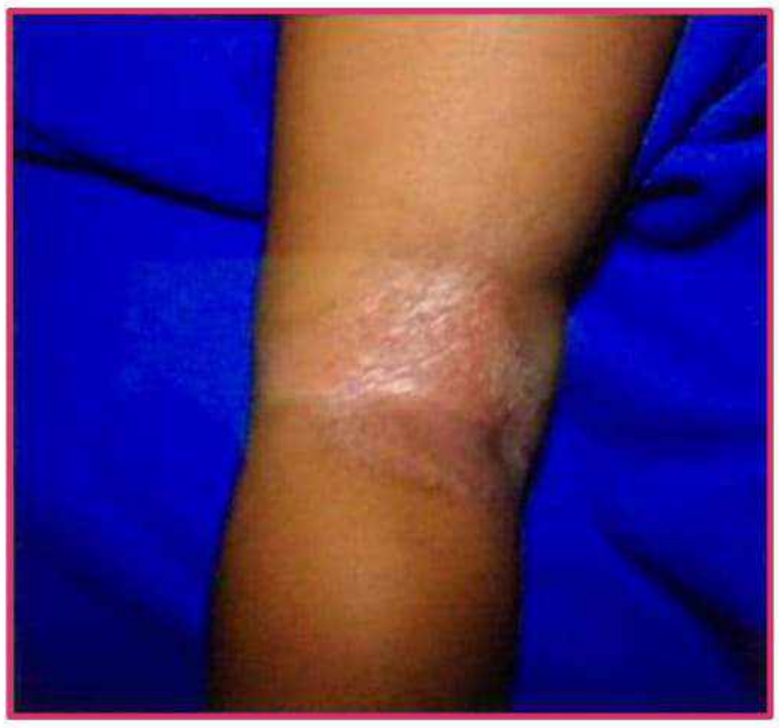
i) Infantile Type :



- Common sites → cheeks, hair, skin.
- Erythematous vesicular lesion
- usually aggravated by URTI.
- Extensors are usally involved
- Features of A/C Eczema ⊕.

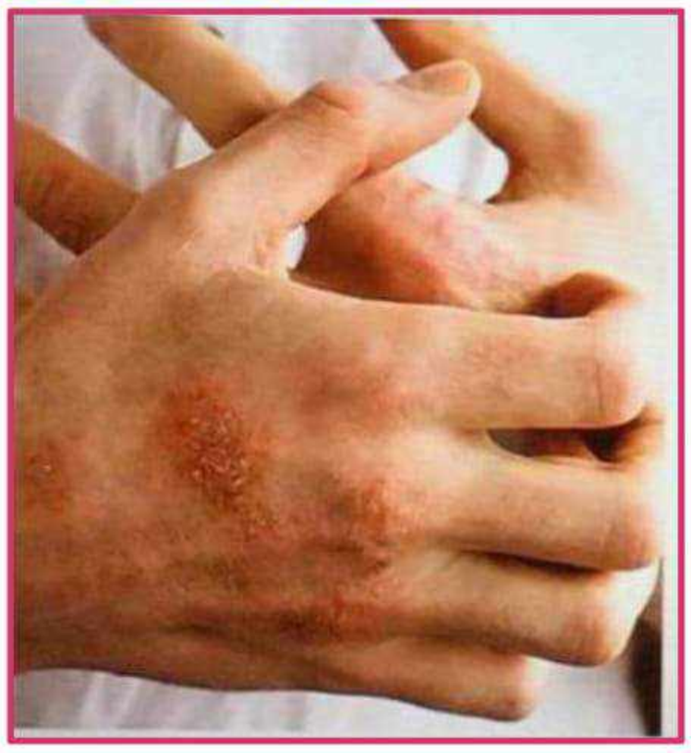
active space

ii) Childhood phase :



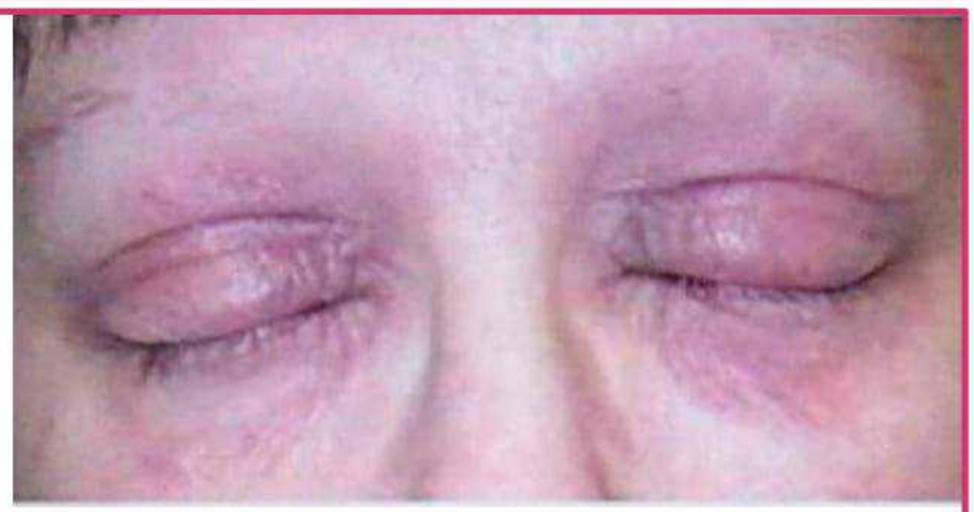
- Subacute eczema
- Flexors are involved
- Universal pruritus present

Adult Phase:



- More of hand dermatitis
- Flexors are involved.
- More of chronic eczema.

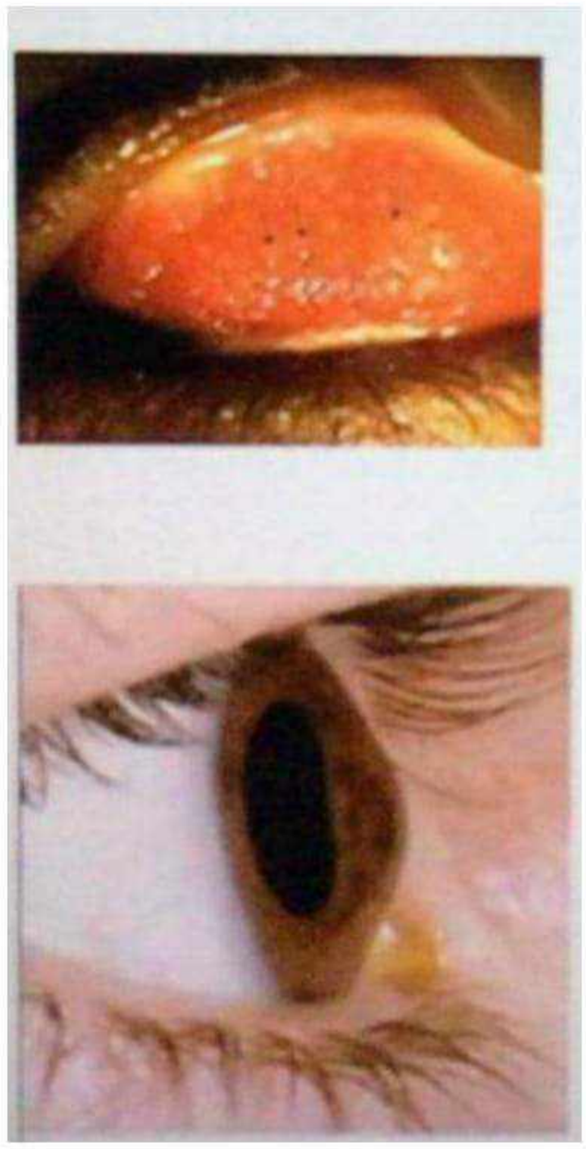
Minor features:



Periorbital dermatitis / Blepharitis

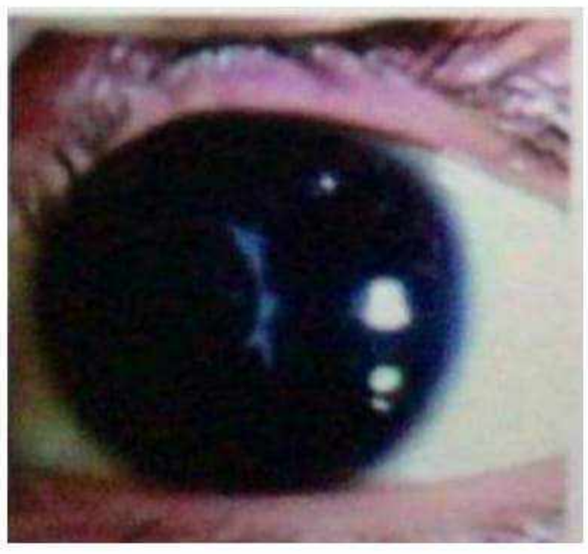
Extrafold of skin under the eye. → Dennie Morgan Fold

Darkening under the eye.
Allergic shiners.

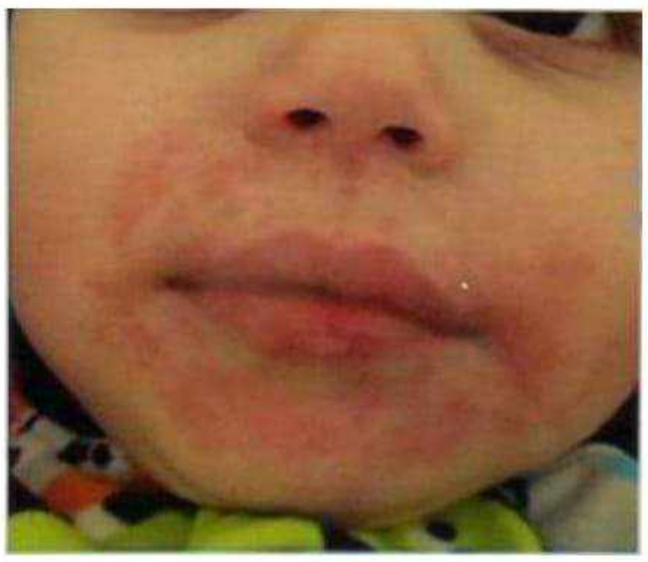


→ Papillary conjunctivitis

→ Keratoconus



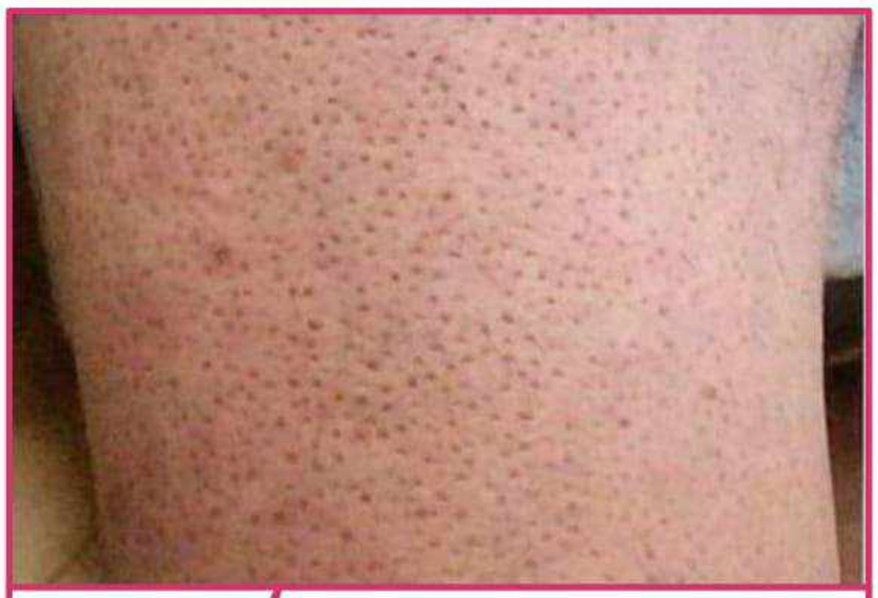
→ Acute Subcapsular cataract.



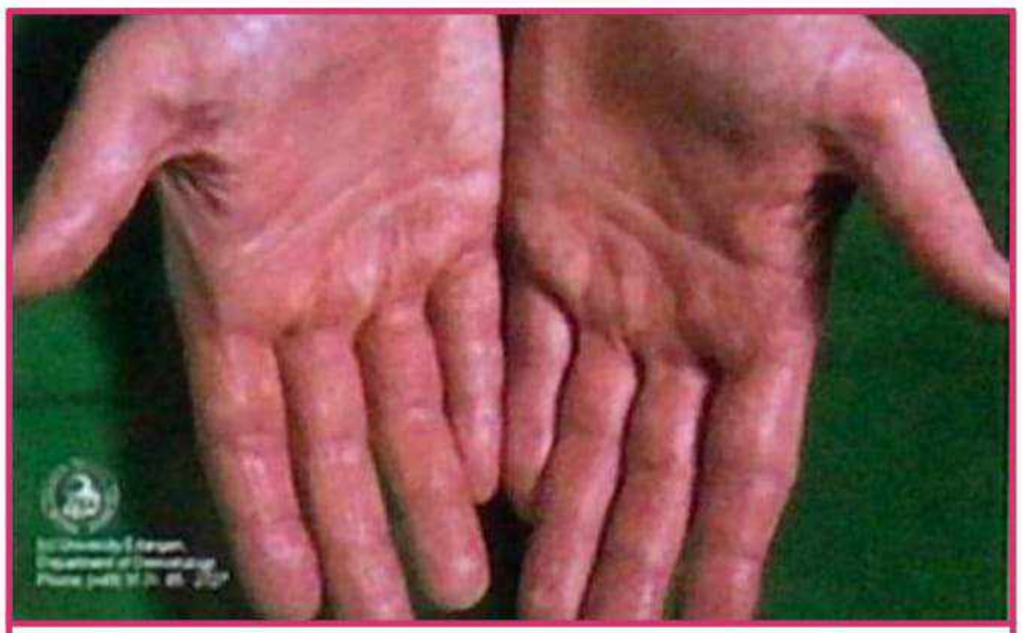
→ Cheilitis + Perioral pallor (Head light sign)

Liplick dermatitis

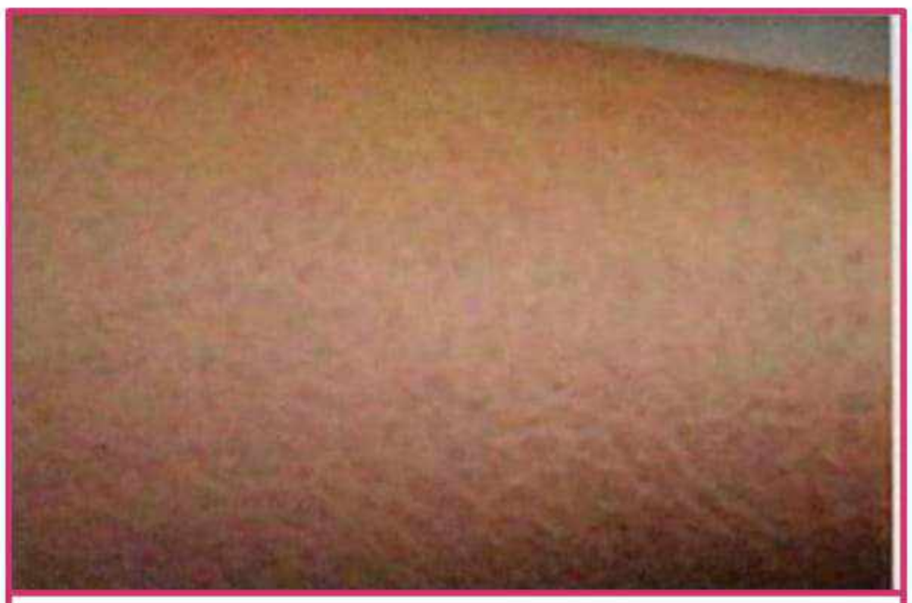
active space



Xerosis / Icthyosis vulgaris

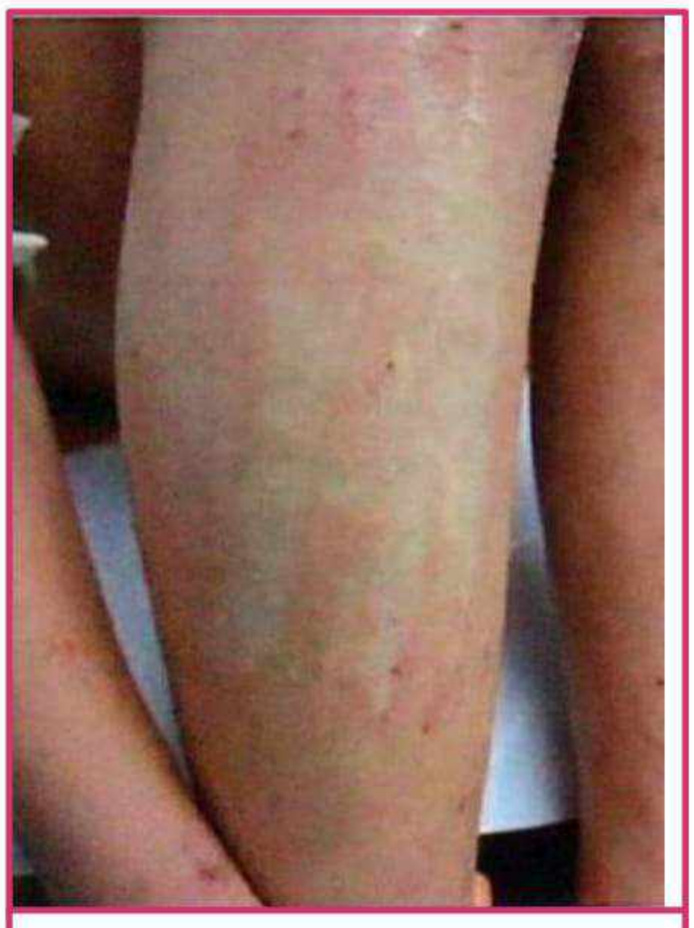


Hyperlinear palms and soles



Keratosis pilaris

- Coiled up hairs - inflammation
- Rough papules on the arm and back.

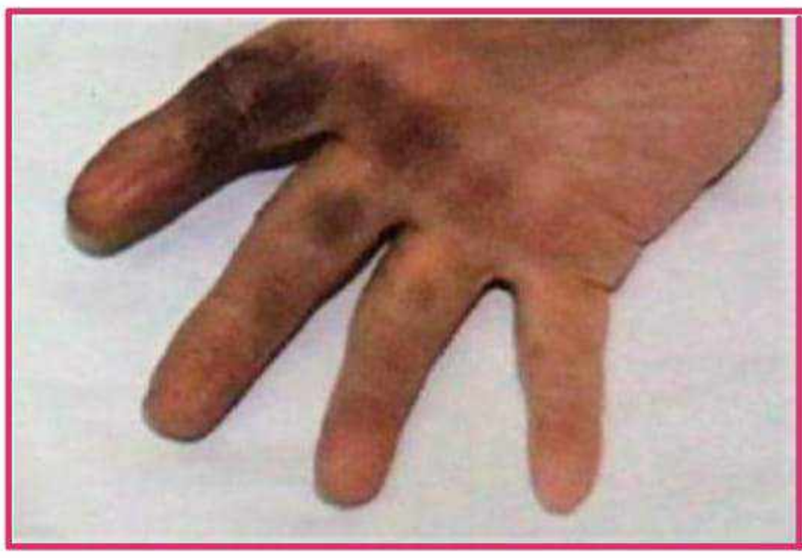


White dermographism

vasoconstriction

Red dermographism → due to urticaria.

active space



Black Dermographism

- seen with metals
- Metals causing are : Nickel and Gold

Diagnosis : Clinical

- High IgE

Criteria : Hanifin and Rajka Criteria

- Major : Any itchy skin condition
- Minor : i) Age of onset < 2yrs

ii) Flexural dermatitis

iii) Skin crease involvement

iv) Personal or Family H/o atopy.

Management

- Remove triggers
- Hydration
- Pharmacological treatment.

active space

- First line:
  - Control of flare / factors
  - Topicals → • Emolients, Moisturisers, Steroids.
    • Calcineurin inhibitors → • Tacrolimus
    • Pimecrolimus.
    • Icthyamol.
  - Systemic → • Corticosteroids
    • Cyclosporine
  - Anti-histamines
  - Anti-biotics.

2nd line
- Allergy management
- Wet compresses
- Phototherapy

3rd Line - Adjuvants: Cyclosporine, Azathioprine, Methotrexate, MMF.

Newer agents i) Crisabole → PDE 4 ⊖ → Topical
ii) Dupilimumab → IL-4 Receptor ∝ Antagonist → Oral

active space

PITYRIASIS ALBA

27:50

- ill defined white hypopigmented lesions associated with mild scaling
- seen more commonly in atopics.
- seen more commonly in children.
- D/D -

i) Vitiligo → depigmented, no scaling

ii) Indeterminate Hansen → single, not scaly and patient is from endemic area.

iii) P. versicolor → Do Wood's lamp, no dryness of skin.

Treatment:

- Emollients
- Mild topical corticosteroids
- Calcineurin inhibitors.

SEBORRHOEIC DERMATITIS

30:45

- Papulosquamous disorder which affects seborrhoeic sites.
- **Pathogenesis:**

- ↑ production of sebum

active space

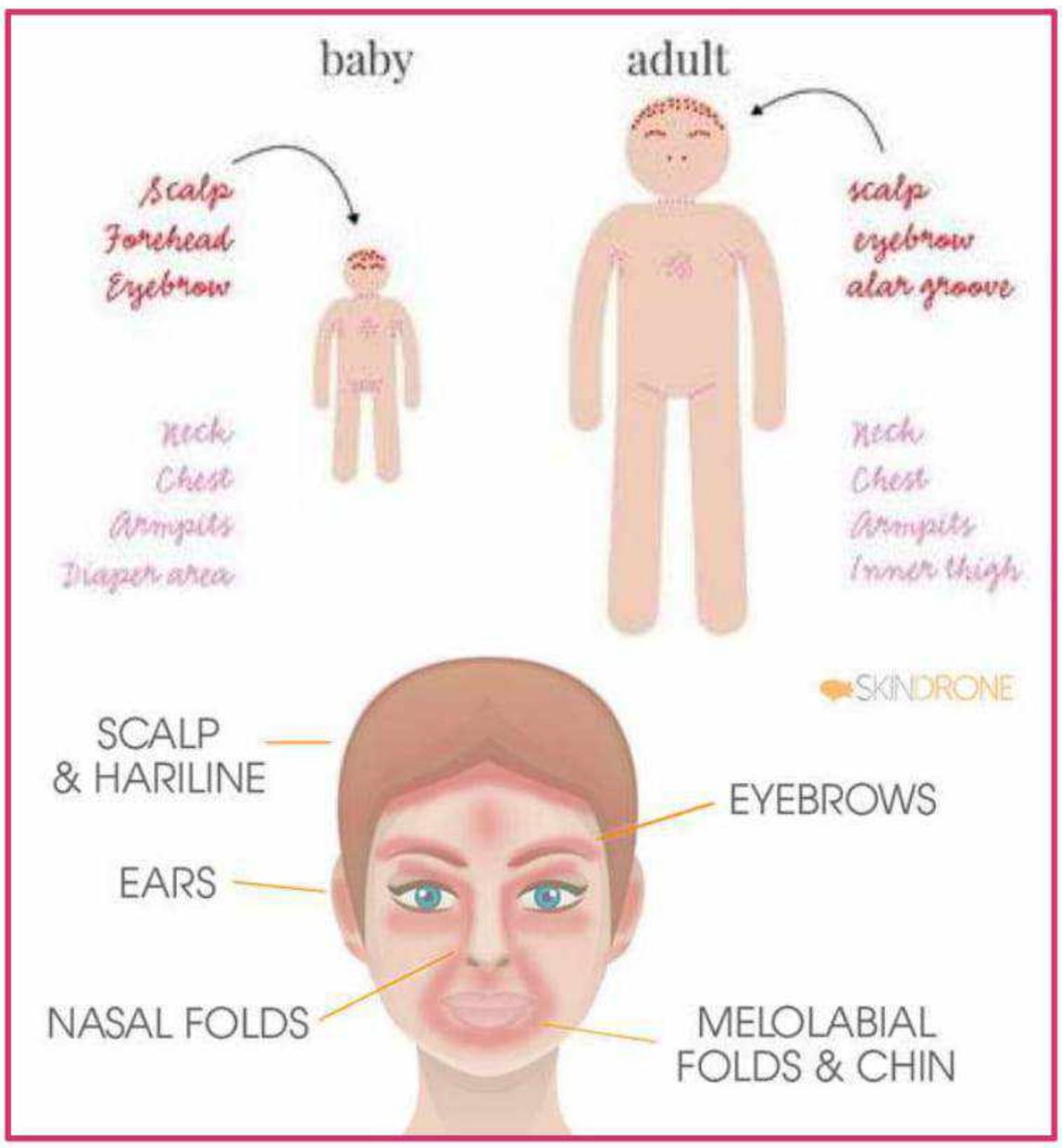
- ↑ Sebaceous gland activity
- organism involved → Malassezia / P. ovale → Increased population.

- Age group affected → Infants, adolescents, adults
- Other associated disorders :

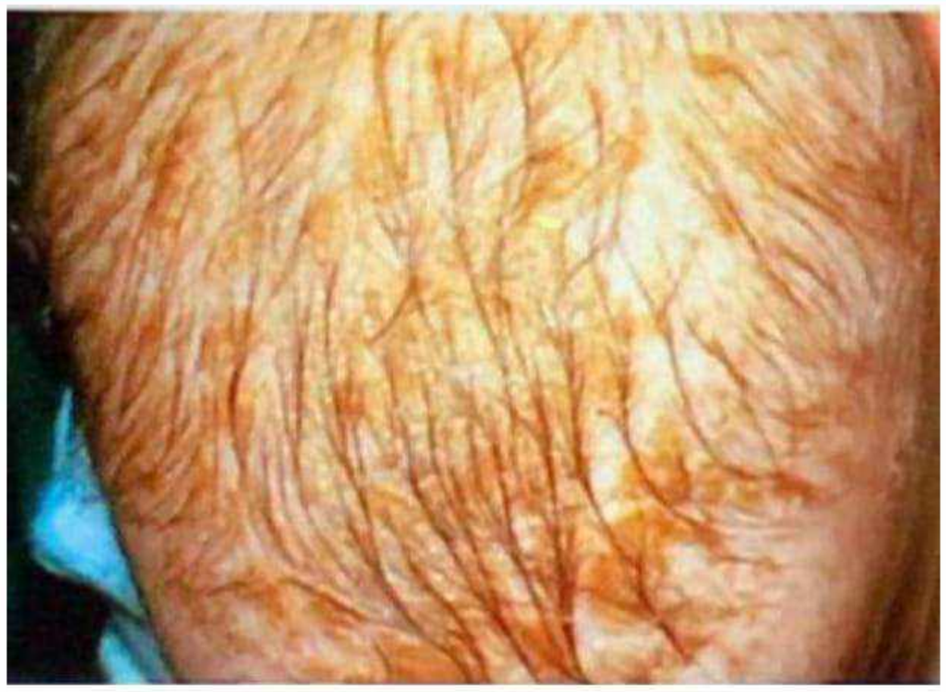
  i) Parkinsons disease

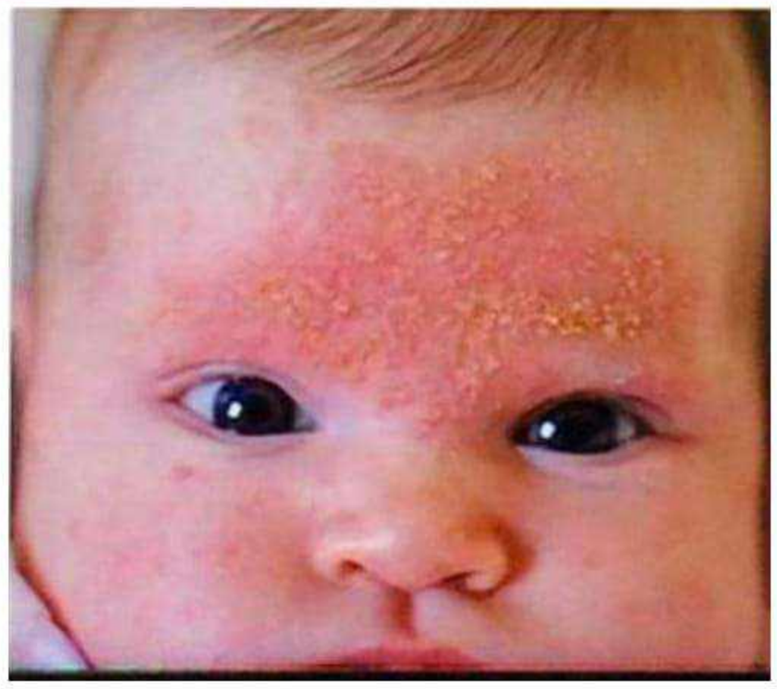
  ii) HIV

- Sites :

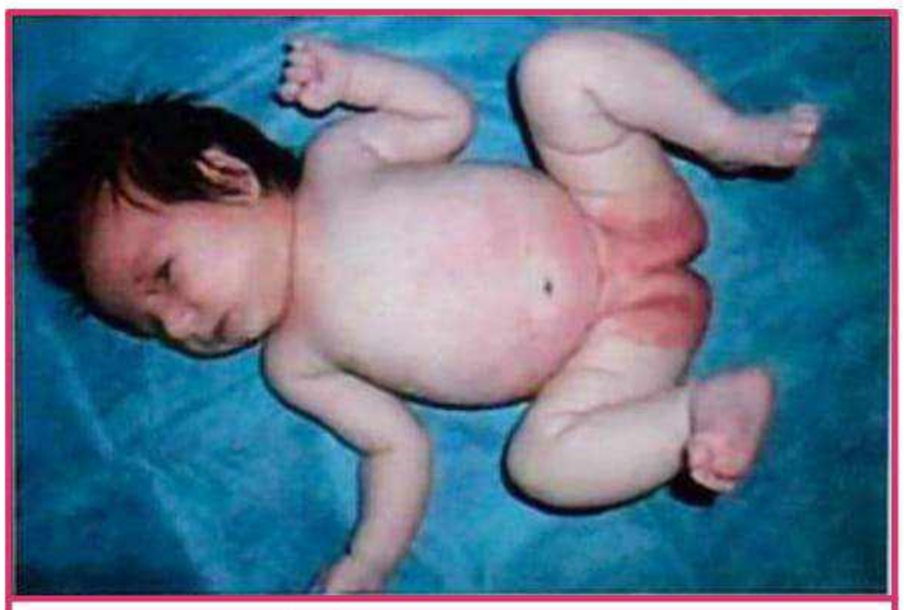


active space





	Seborrhoeic dermatitis	Atopic dermatitis
Age group :	• < 3 months • greasy looking yellowish scales on frontal and vertex area of scalp → CRADLE CAP. • due to maternal Androgens.	• Infantile : 3m - 2yrs • Adolescent : 2yrs - 12yrs • Adults : > 12yrs



Diaper area

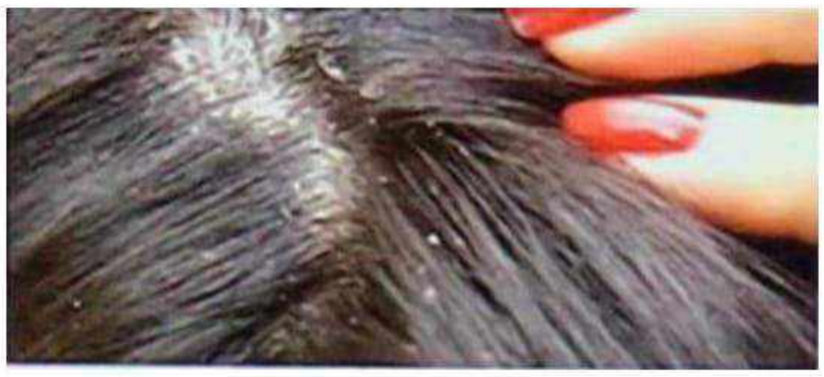
active space

- **In adults :**

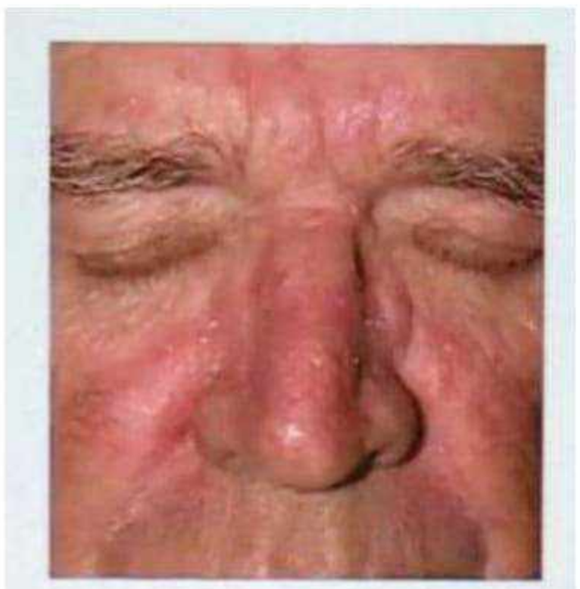
start with dandruff - scalp

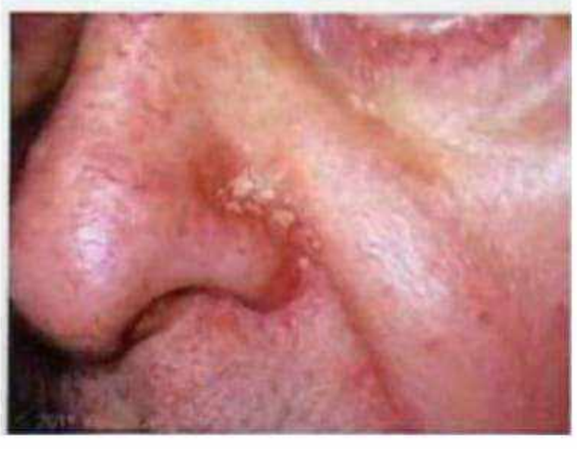
↓

Erythematous scaly lesions covered with greasy yellow scales.



- **In face :** Seborrhoeic areas





→ Eyebrows , forehead , nose

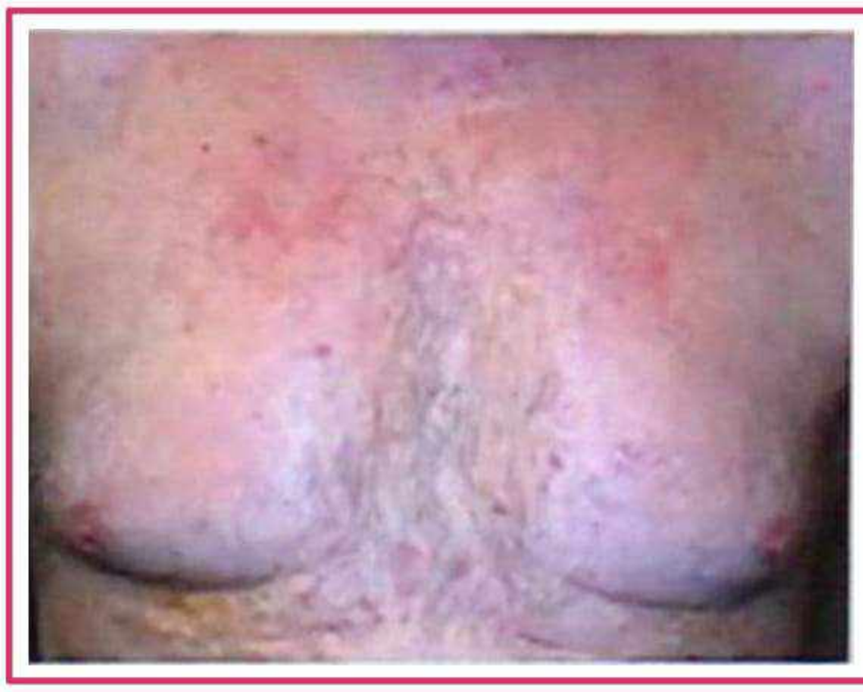
Malar area , Perioral area

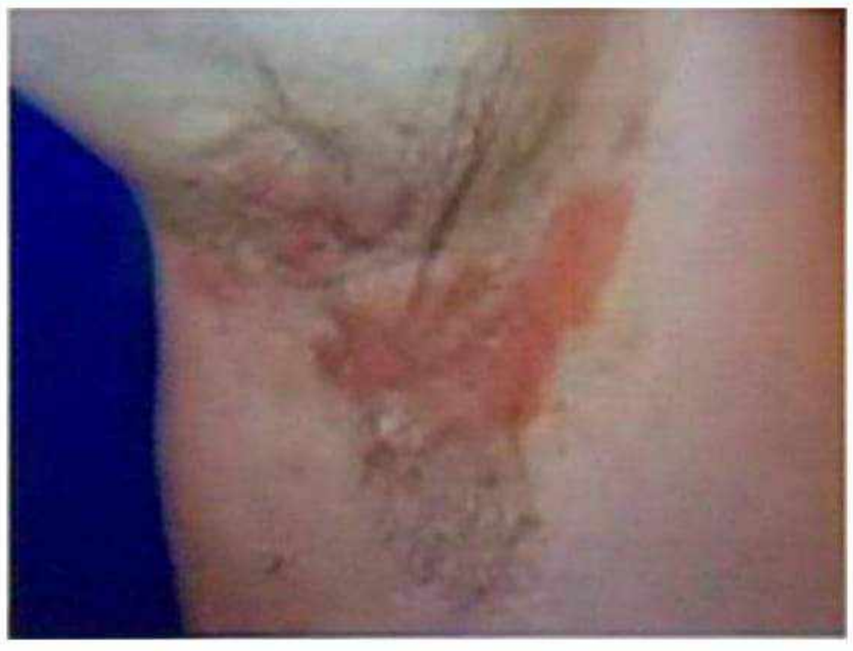
→ Nasolabial folds.

active space

Clinical features:

- Itching
- Dandruff
- Burning sensation on sun exposure.
- In trunks → seen in upper chest and flexures



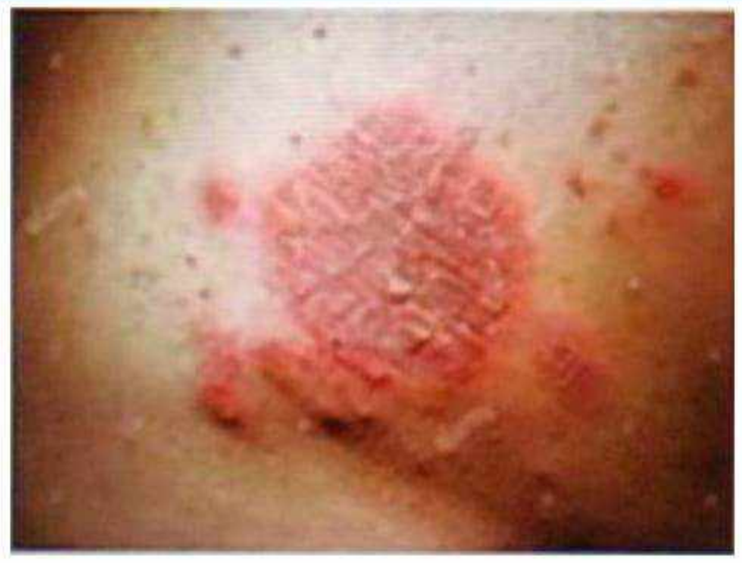


Management :

- shampoos →
  - Ketoconazole
  - Selenium sulfide
  - Cyclopirox oleamine shampoos
  - Tar shampoos
- Topical →
  - Antifungals
  - Steroids
- Oral →
  - Antihistamines
  - Antifungals → Azoles

DISCOID ECZEMA

42:15

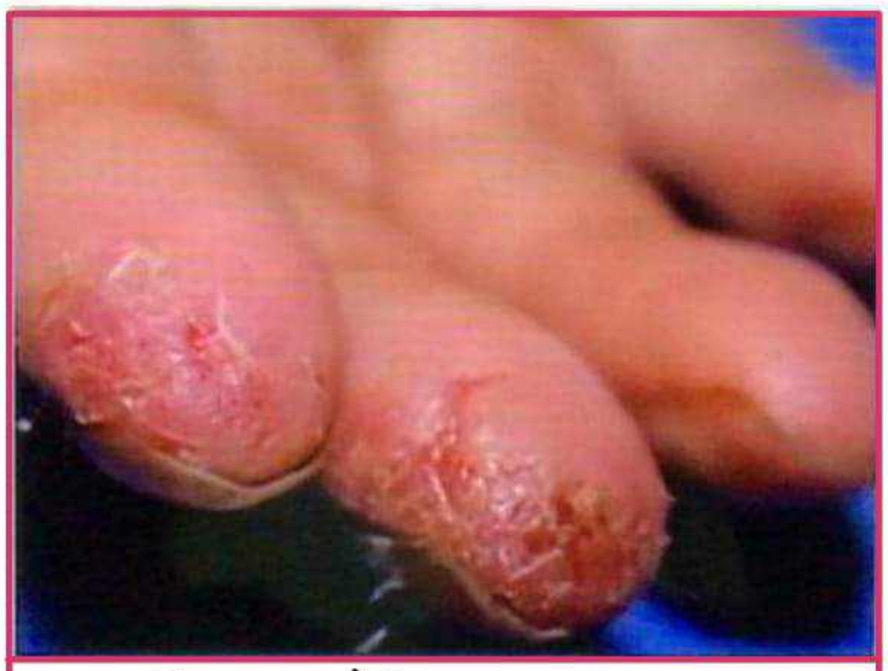


- (MC) pathological pattern
- coin shaped / round lesions
- Nummular eczemas
- seen in legs
- Treatment – • Emollients
  - Topical steroids

HAND ECZEMA

- Can be endogenous or exogenous.
- Patterns seen are :

i) ACD
ii) ICD
iii) Focal peeling
iv) Hyperkeratotic Eczema
v) Pompholyx

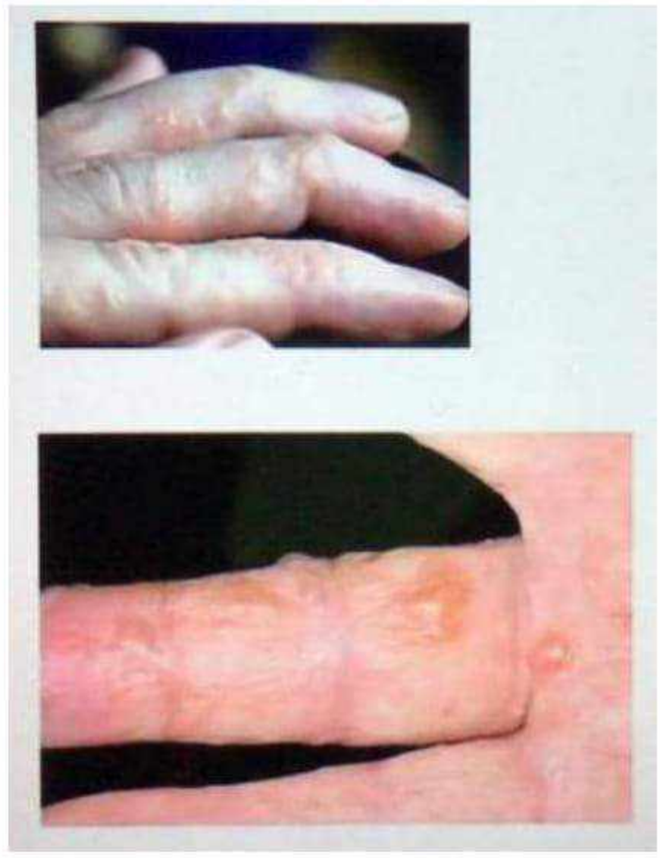


Finger tip eczema.

active space

Pompholyx

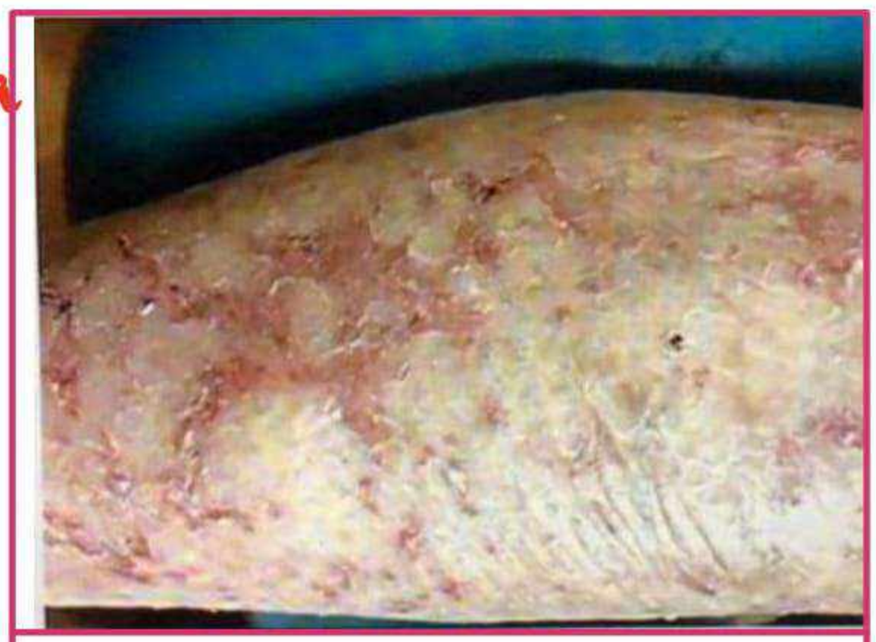
- Endogenous eczema
- B/L symmetrical deep seated vesicles on fingers and palms.
- Relapsing Remitting course.
- Pruritic
- a.k.a Sago grain like lesions.



ASTEATOTIC ECZEMA

46:20

- a.k.a Eczema craquele / winter eczema
- Decreased lipids
- Dry skin → seen in atopics
- seen in elderly
- seen in lower legs



- Cracked appearance
- Xerosis
- Crazy pavement /crackled porcelain pattern

Treatment :

- Emollients
- Topical steroids
- Antihistamines.

active space

Venous Eczema

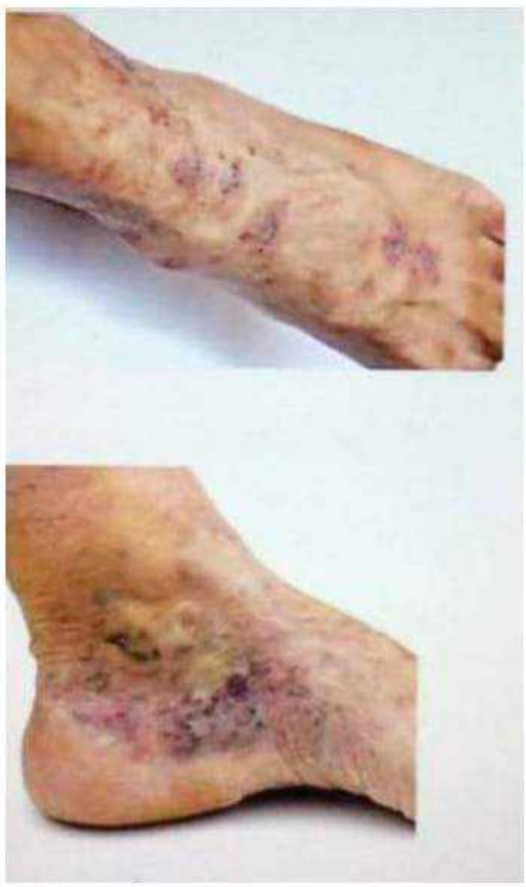
- patient would have underlying venous insufficiency
- a.k.a Gravitational / Stasis eczema.
- Dilated varicose veins on medial aspect of lower legs

↓

Microangiopathy and chronic damage to skin.

Signs

- Varicose veins
- Atrophic blanches
- Hemosiderin deposition.



Treatment :

- Treat underlying disorder
- Stockings
- leg elevation.
- Antihistamines
- Topical steroids.

NEURODERMATITIS

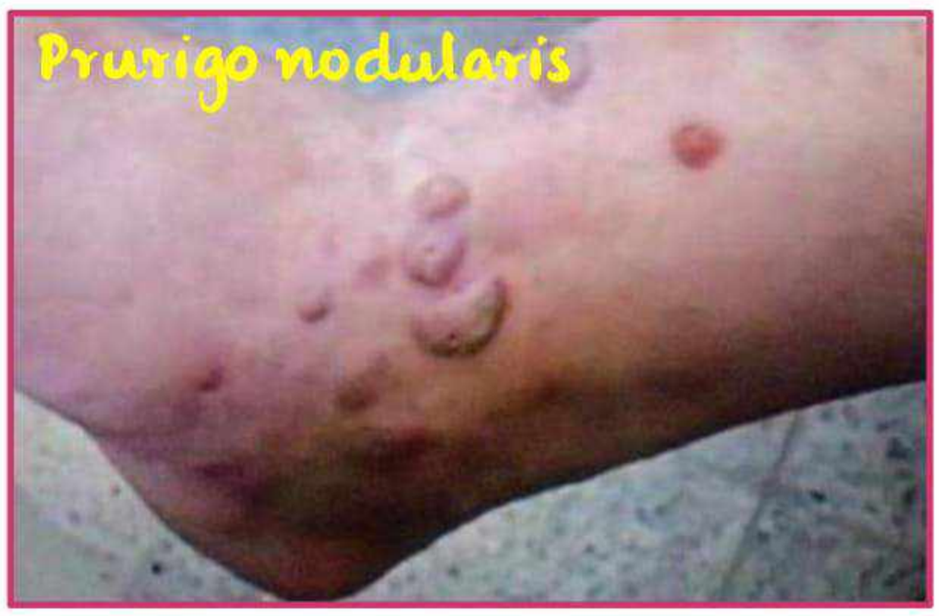
51:30

- On Itching → Patient Scratches (1st Itching → lesions)

Itch Scratch cycle

active space

- accessible parts of body are involved.



Treatment: Treat the itch.

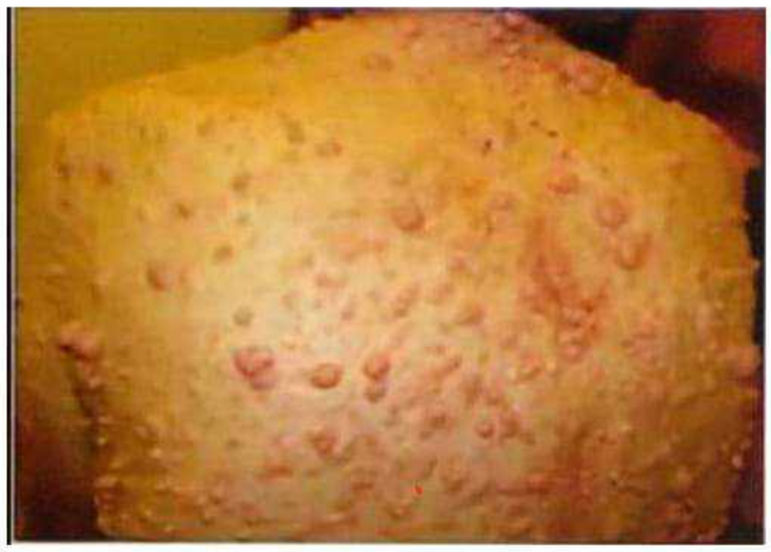
active space

Genodermatoses

- Genetic disorders of skin

NEUROFIBROMATOSIS 1

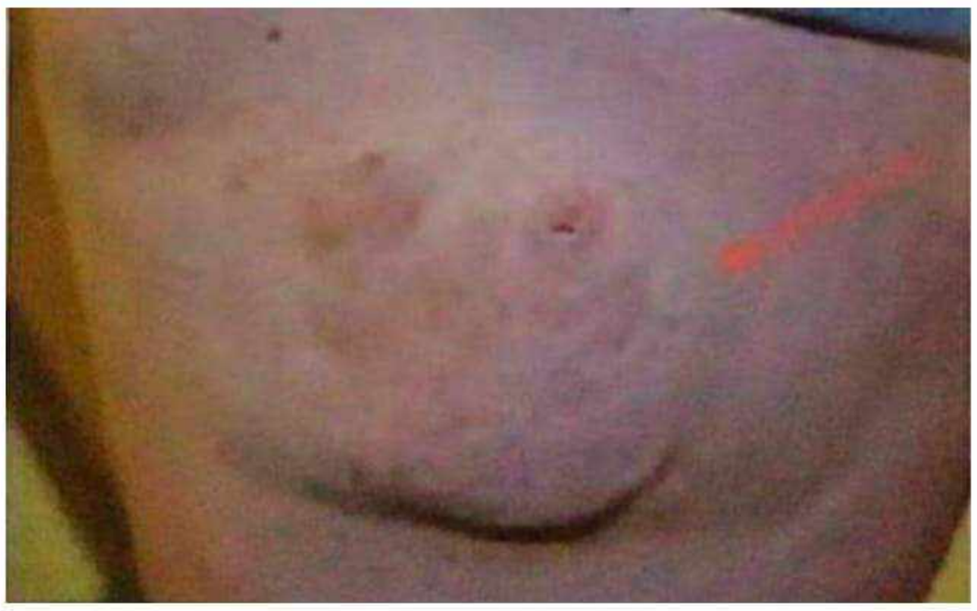
00:35



- a.k.a Von Recklinghausen's Disease.
- Autosomal dominant
- Gene → NF-1 on Chr. 17
- Encodes protein → Neurofibromin.

Clinical features:

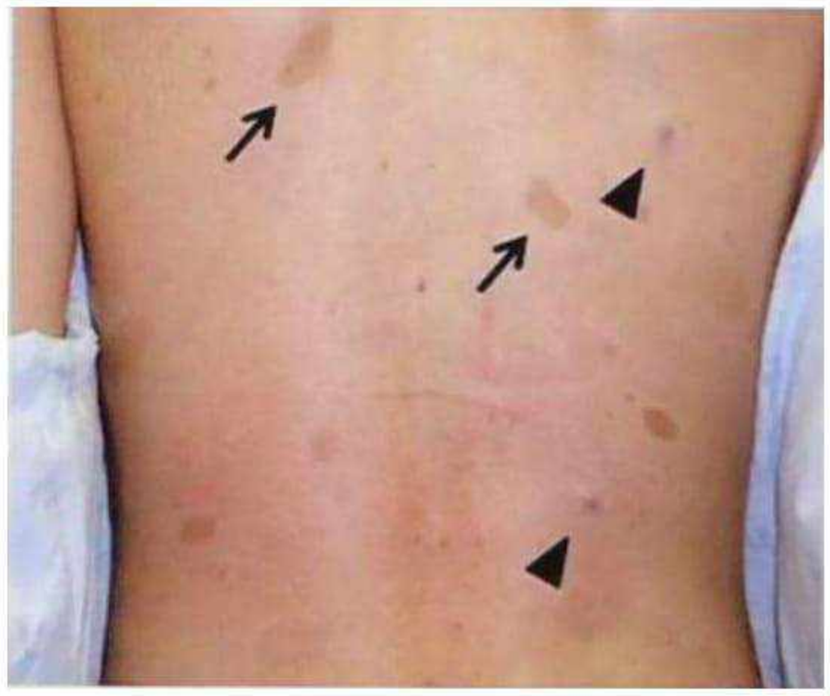
- Soft swellings - sessile/pedunculated.
- Button Hole Sign
- Present all over the body



- Bag of worms

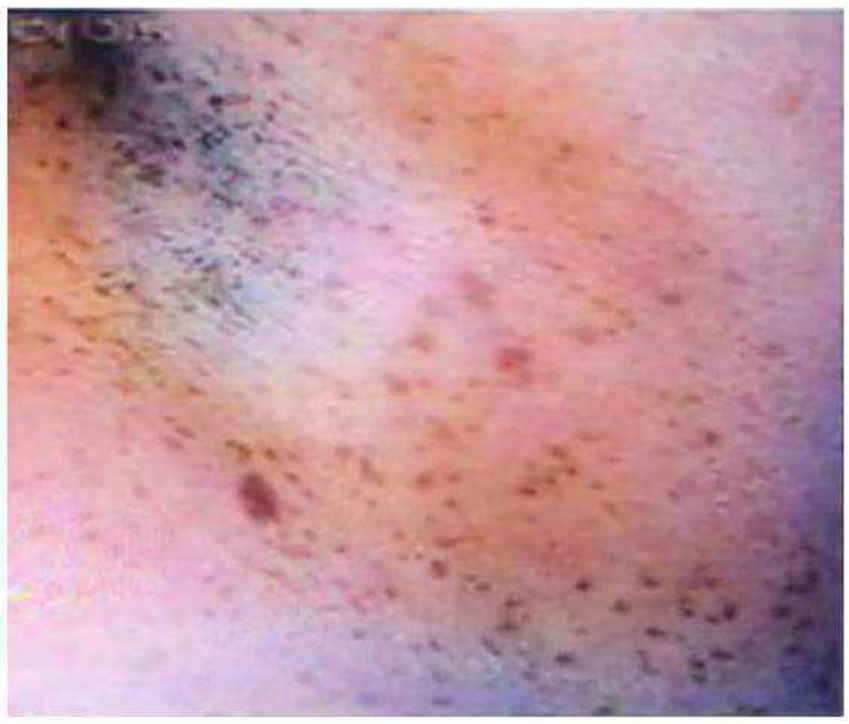
Plexiform neurofibroma

- Elongated, along a nerve
- commonly involved nerve
  - ↳ Trigeminal and cervical nerves.



Cafe au lait macules

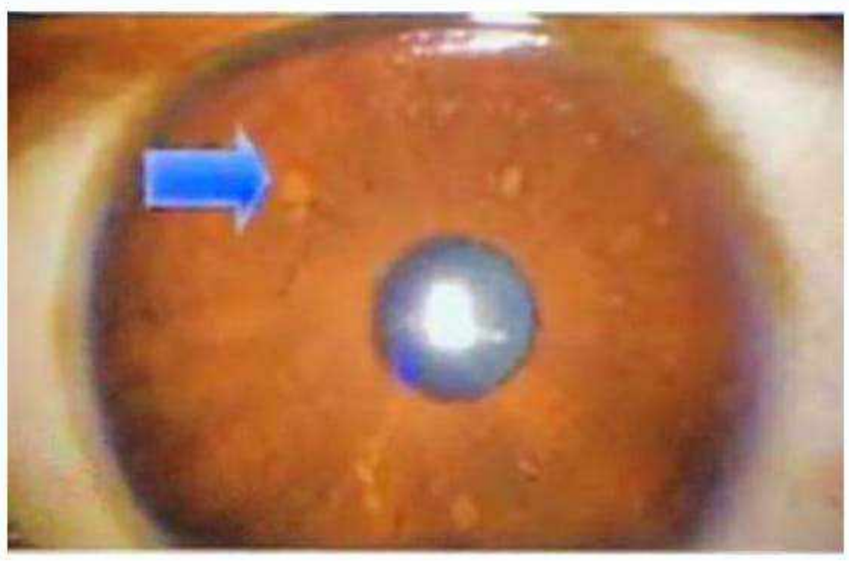
- Hyperpigmented / Coffee coloured macules on the body.
- ≥ 6 CALM
  - >0.5cm in prepubertal
  - >1.5cm in adults



Freckling

- seen in axilla and groin.

CROWNE's sign → Pathognomic.



LISCH NODULES

- Iris hamartomas
- Better visualised on slit lamp.
- Donot affect vision

active space

Skeletal features :

- Kyphoscoliosis
- Sphenoid wing dysplasias
- Pseudoarthrosis of long bones.

CNS features.

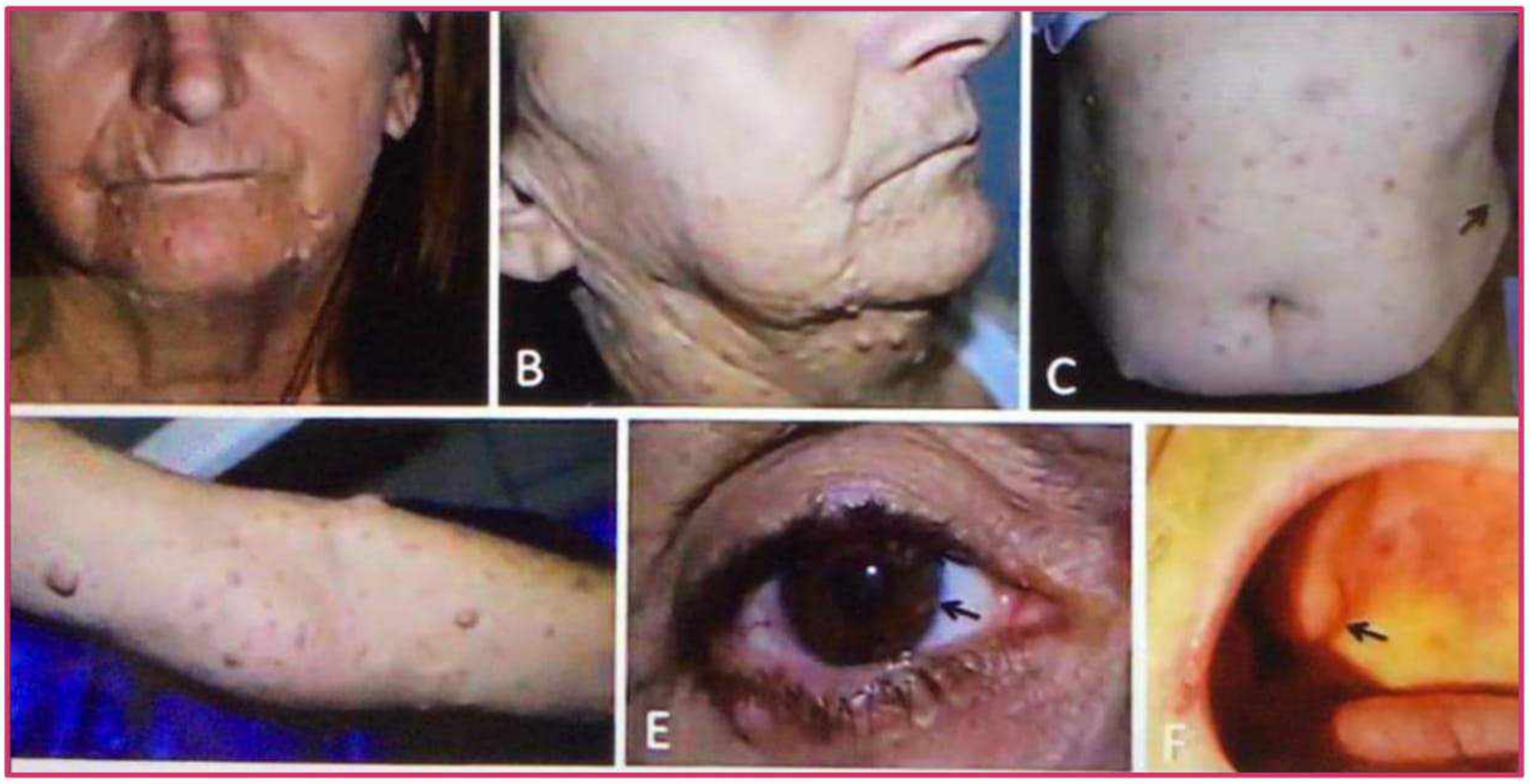
- Epilepsy
- Low intelligence

Malignancies

- Gliomas
- Astrocytomas

A diagnosis of NF1 can be made in an individual with 2 or more of the following:
≥6 café au lait macules (>5 mm in greatest dimension for prepubertal persons and >15 mm in greatest dimension for postpubertal persons)
Axillary or inguinal freckles
≥2 neurofibromas (any type) or 1 plexiform neurofibroma
Optic glioma
≥2 Lisch nodules
Sphenoid dysplasia, tibial pseudoarthrosis, or other distinctive bone lesion
First-degree relative with a diagnosis of NF1

Abbreviation: NF1, Neurofibromatosis type 1.



Treatment

- No cure
- Pirfenidone → Farnesyl inhibitor has been used.

NEUROFIBROMATOSIS 2

12:20

- a.k.a Bilateral Acoustic Schwannoma
- A.D Inheritance
- Gene: NF-2 on Chr. 22
- Protein: Schwanomine

Clinical features:

1. Cutaneous: few neurofibromas / CALM.

active space

2. Systemic - Schwanomas, Meningioma, Gliomas.

↳ Tinnitus ⊕

TUBEROUS SCLEROSIS COMPLEX

13:50

- a.k.a **Bourneville's Disease, Epiloia**

E
P
I } Epilepsy

L
O
I } Low intelligence

A - Adenoma sebaceum.

- Inheritance : AD
- Chromosome: Chr 9 and 16
- Gene : TSC 1 and TSC 2
- Protein : Hamartin and Tuberin.

Feature :

Adenoma sebaceum

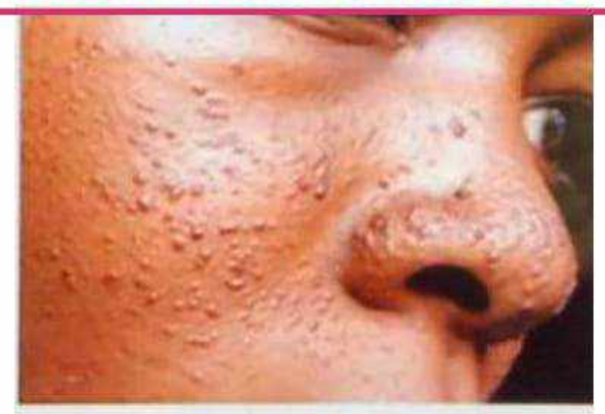
- These are Angiofibromas

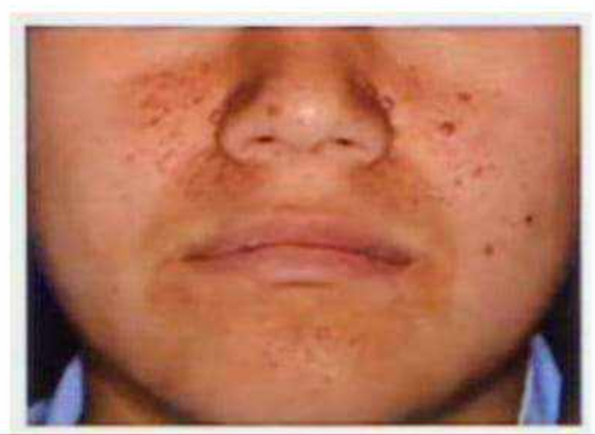
↓

Soft, reddish, papules seen on nose, Nasolabial folds, cheeks. (Butterfly)

- Increased during puberty.

Adenoma sebaceum



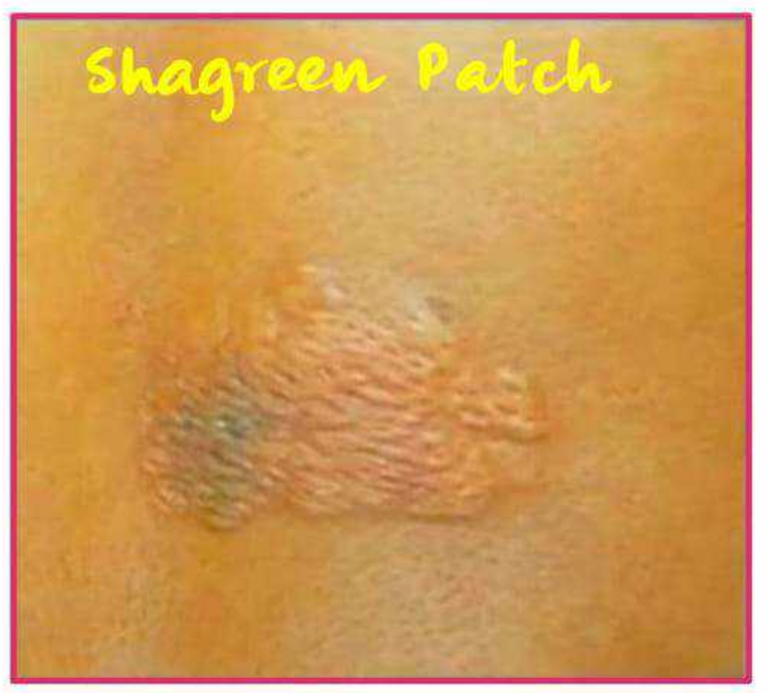


Ash Leaf Macules

- Lanceolate shaped
- Hypopigmented macule enhance on Wood's Lamp.
- (MC) situated on trunk.

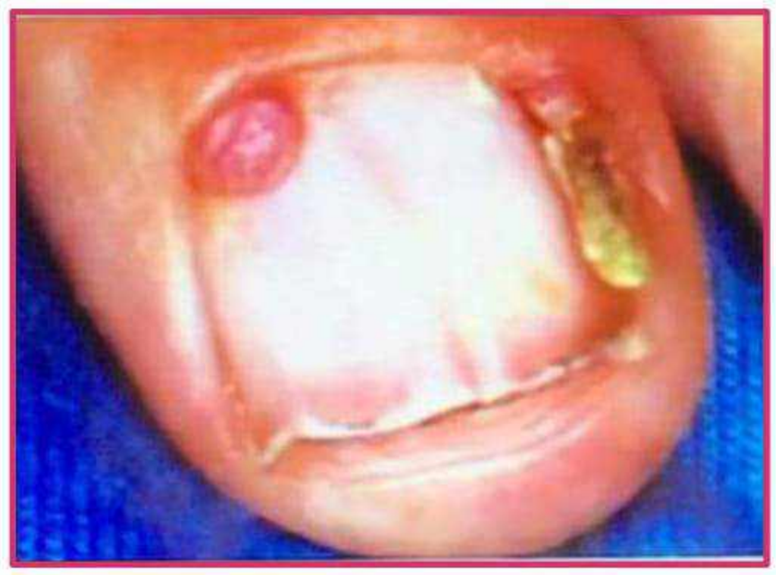
Shagreen Patch

- Collagenomas
- Soft well defined plaques with rugosities and dimpling
- (MC) site : Lumbosacral.

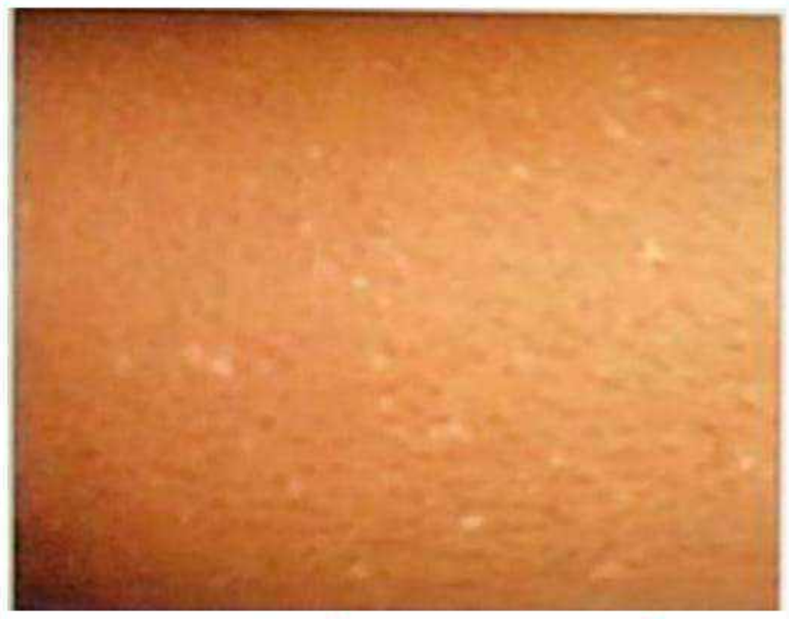


Koenon's Tumor

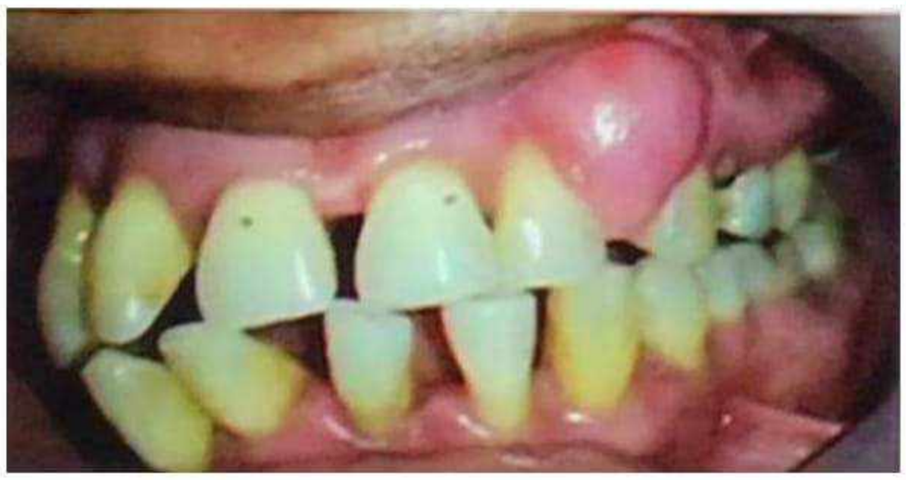
- a.k.a Periungual fibromas
- Soft growth originates from nail bed.



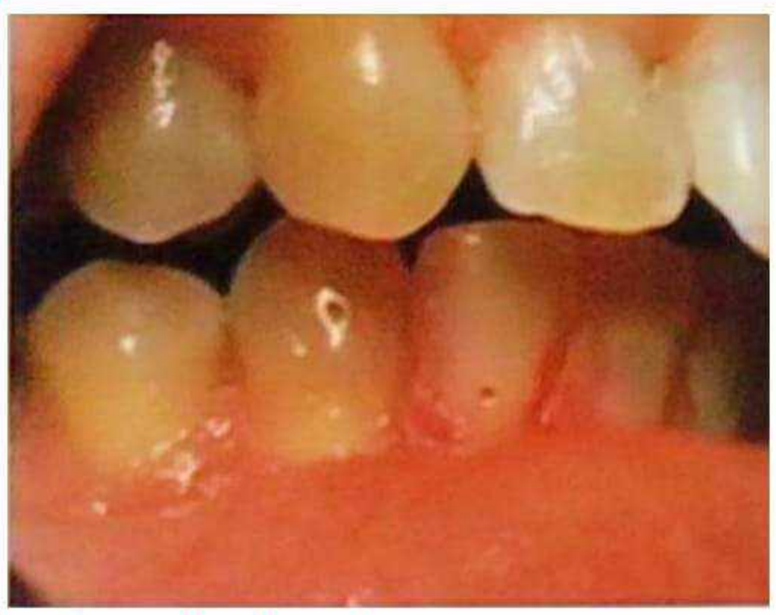
active space



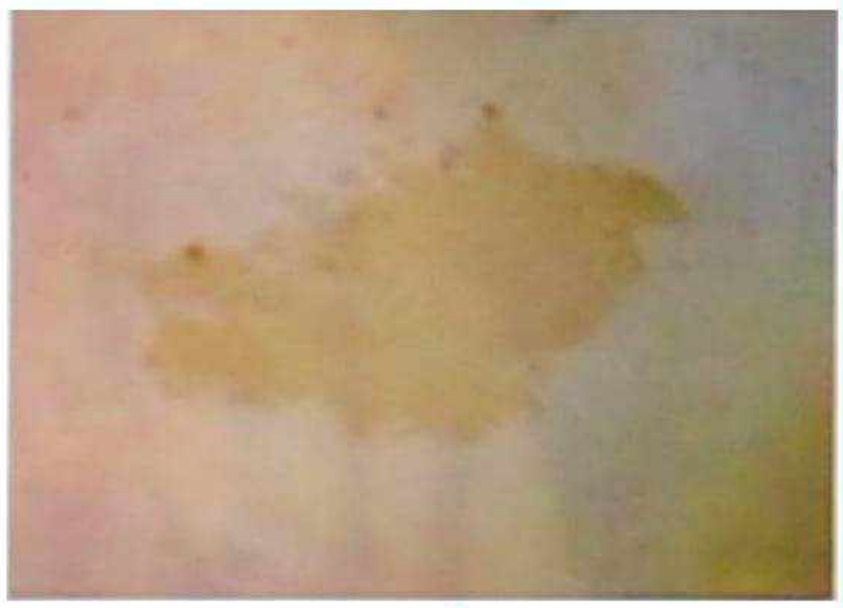
Confetti like Macules



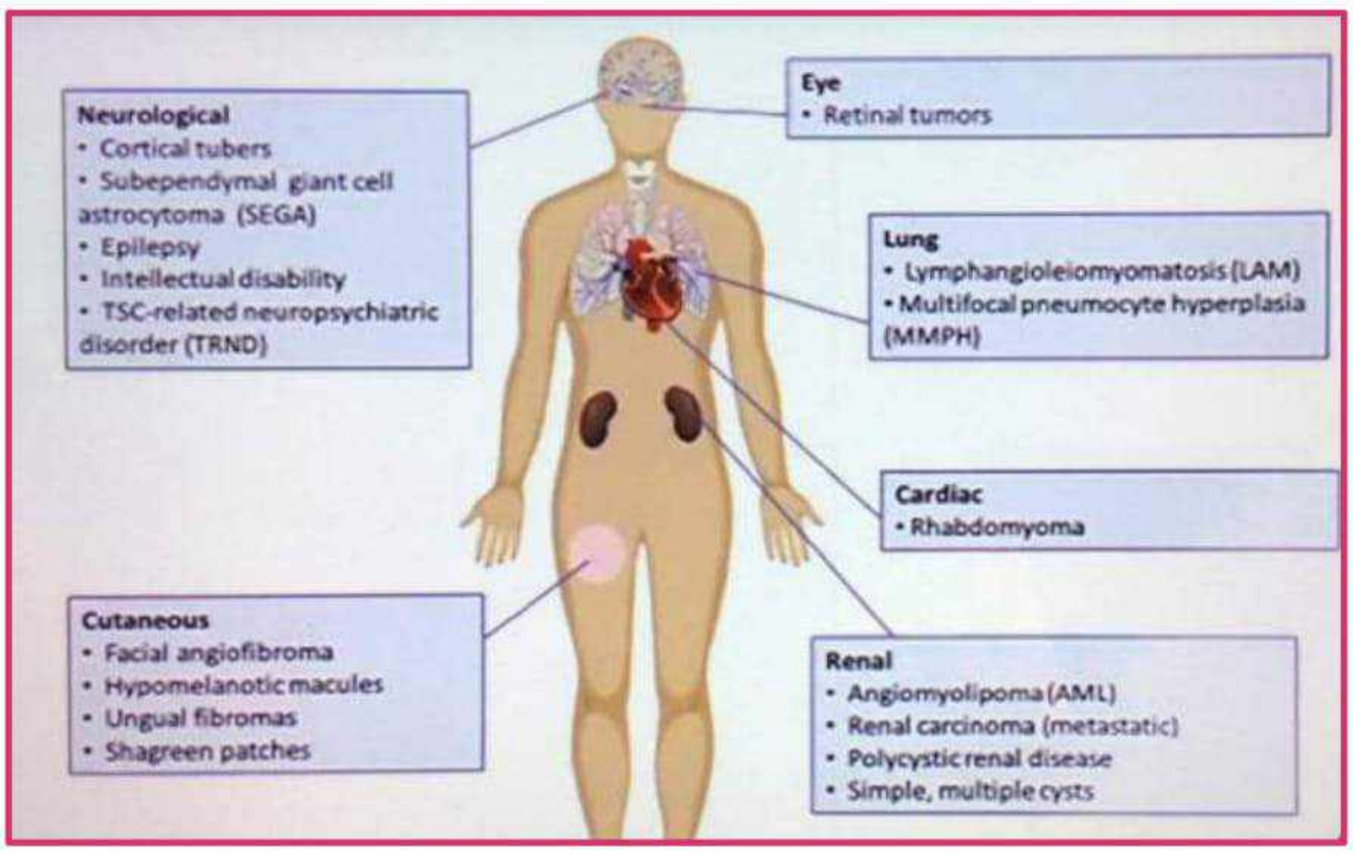
Gingival fibromas.



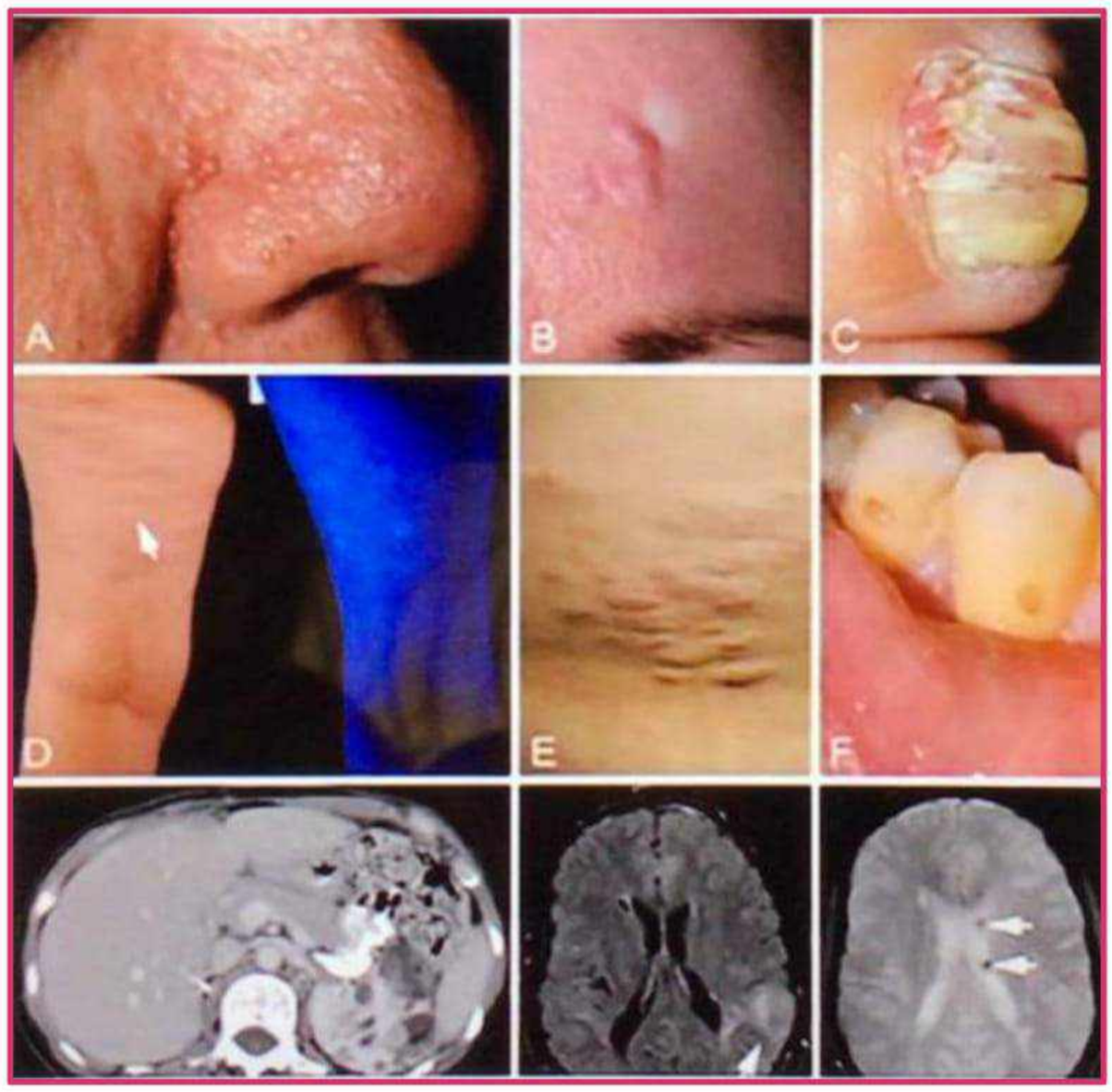
Dental pits



CALM's



active space



MAJOR FEATURES		MINOR FEATURES	
1	Hypomelanotic macules (>3, at least 5-mm diameter)	1	"Confetti" skin lesions
2	Angiofibromas (>3) or fibrous cephalic plaque	2	Dental enamel pits (>3)
3	Ungual fibromas (>2)	3	Intraoral fibromas (>2)
4	Shagreen patch	4	Retinal achromatic patch
5	Multiple retinal hamartomas	5	Multiple renal cysts
6	Cortical dysplasias*	6	Nonrenal hamartomas
7	Subependymal nodules		
8	Subependymal giant cell astrocytoma		
9	Cardiac rhabdomyoma		
10	Lymphangioleiomyomatosis (LAM)†		
11	Angiomyolipomas (>2)†		

Definite diagnosis: Two major features or one major feature with >2 minor features.

Possible diagnosis: Either one major feature or >2 minor features.

*Includes tubers and cerebral white matter radial migration lines.

†A combination of the two major clinical features (LAM and angiomyolipomas) without other features does not meet criteria for a definite diagnosis.

- Course : Progressive

Treatment

- No treatment
- Rapamycin has been tried.

GI POLYPOSIS SYNDROME

24:30

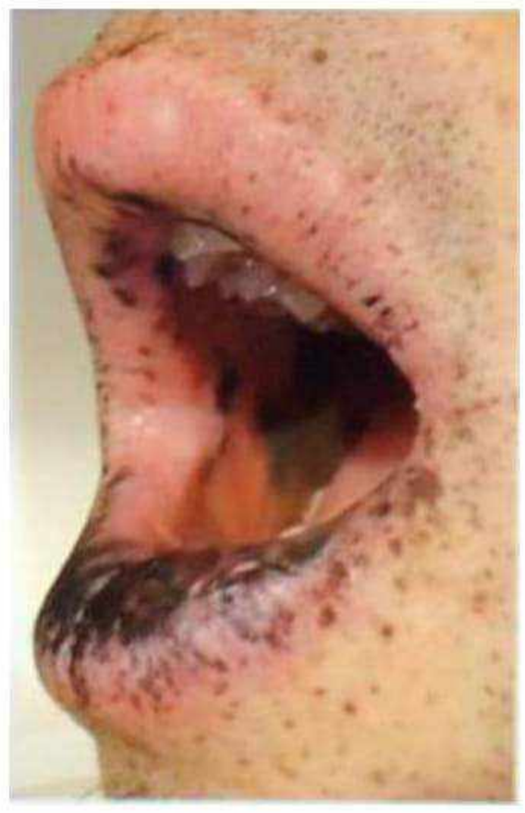
1. Peutz Jegher's } Hamartomatous G.I polyposis
2. Cowden's
   ↳ PTEN mutation
3. Gardner - Adenomatous polyposis
   ↳ APC mutation

Peutz Jegher's

- Periorificial lentigenosis

Presents with :

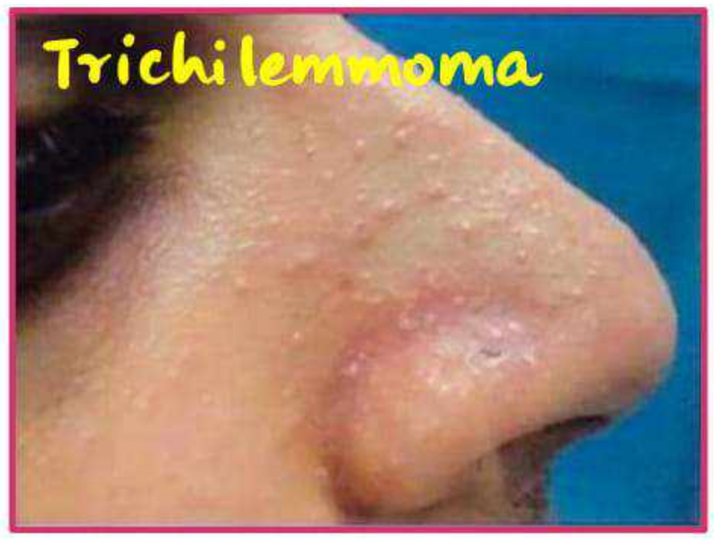
- lentigenes in and around oral mucosa
- G.I polyposis in around 10-30yrs of age.



active space

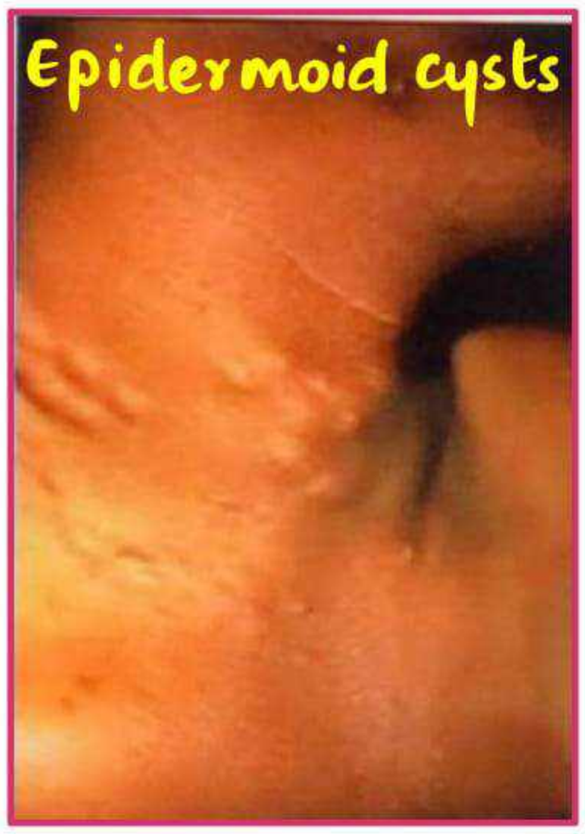
Cowden Syndrome

- Trichilemmoma
- Lichenoid skin coloured papules around nose and face.
- Gene involved PTEN.



Gardner Syndrome

- APC gene involved
- Epidermoid cysts are present.



DNA REPAIR DEFECTS

28:30

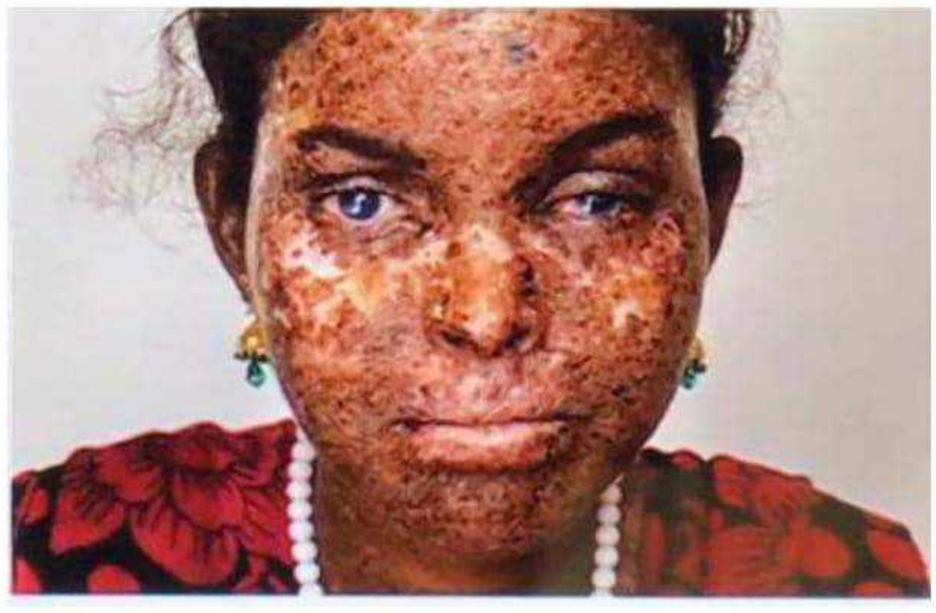
Xeroderma Pigmentosa

- characterised by photosensitivity, oculocutaneous pigmentation and early neoplasia
- A.R Inheritance
- Nucleotide Excision Repair defect

active space

- Multisystem disorder : Skin, Eyes and Nervous System.

Cutaneous manifestation :



- Child normal at birth
- Freckle like lesions on sunexposed lesions
- White interspersed atrophic lesions
- Telangiectasias ⊕
- Erythema ⊕
- Sunburns.
- Xerotic / Dry looking skin.

Malignancy associated :

i) **Benign :** Actinic keratosis, keratoacanthoma

ii) **Malignant :** SCC, BCC, Malignant melanoma.

Ocular manifestations : Photophobia, conjunctivitis, Lentigenes

Neurological :

- Mental Retardation.
- Low Intelligence
- Altered reflexes

Systemic Malignancies:

- Medulloblastoma
- Astrocytoma.

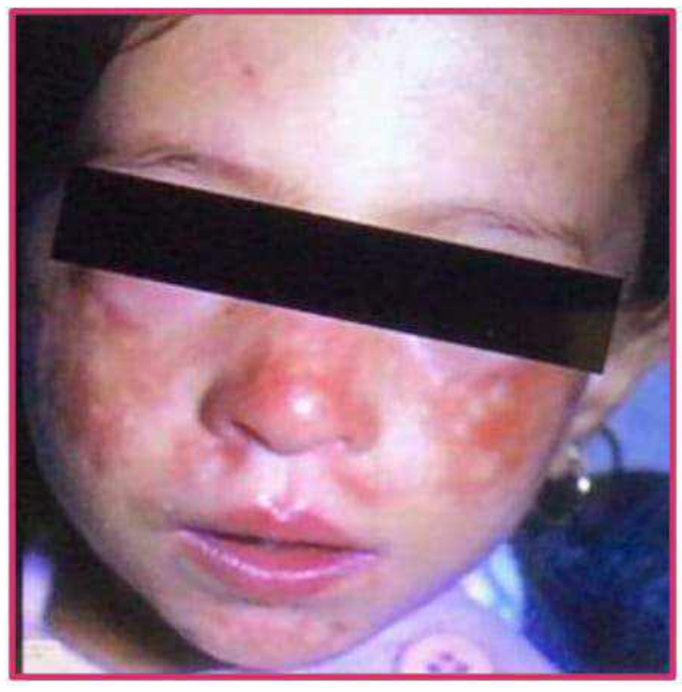
Treatment

- No cure
- Complete sunprotection
- Genetic counselling
- Screening
- Liposomal preparation of Endonuclease V.

BLOOM SYNDROME

36:30

- a.k.a Congenital Telangiectatic Erythema



Findings

- Erythema and Telangiectasias over face
  - Photosensitivity
- Stunted growth
- Early Neoplasias.
- Autosomal Recessive.

active space

EPIDERMOLYSIS BULLOSA

38:10

- a.k.a **Mechanobullous disorders**
- formation of bulla at the site of mechanical trauma

Types :

1. **Congenital :** EBS, EBJ, EBD
2. **Acquired :** EBA

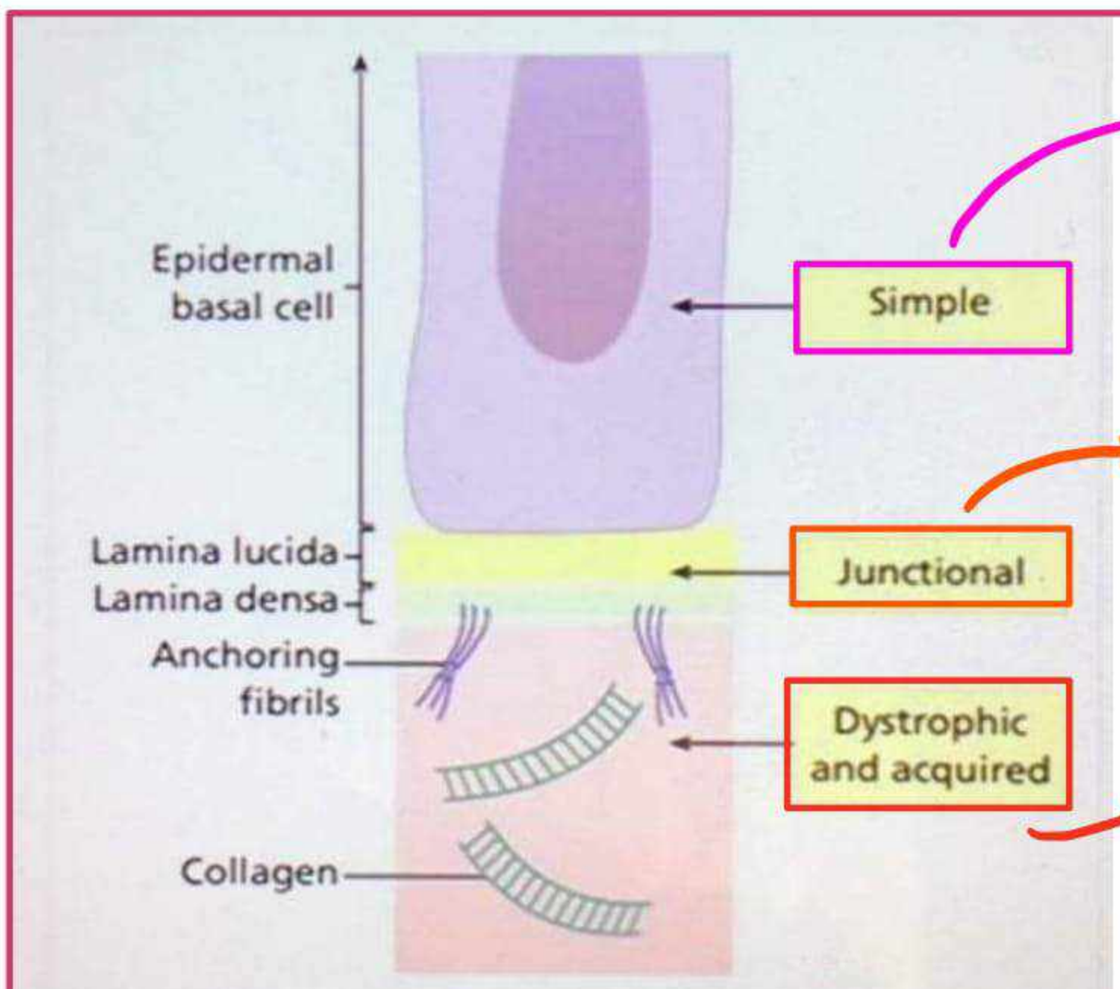


Fig. 9.12 Levels of blister formation in epidermolysis bullosa at the dermo-epidermal junction.

Defect: Keratin 5 and 14

Split : Intra-epidermal

Defect: Laminin 5

Split : BMZ

Defect: Collagen VII

Split : Subepidermal.

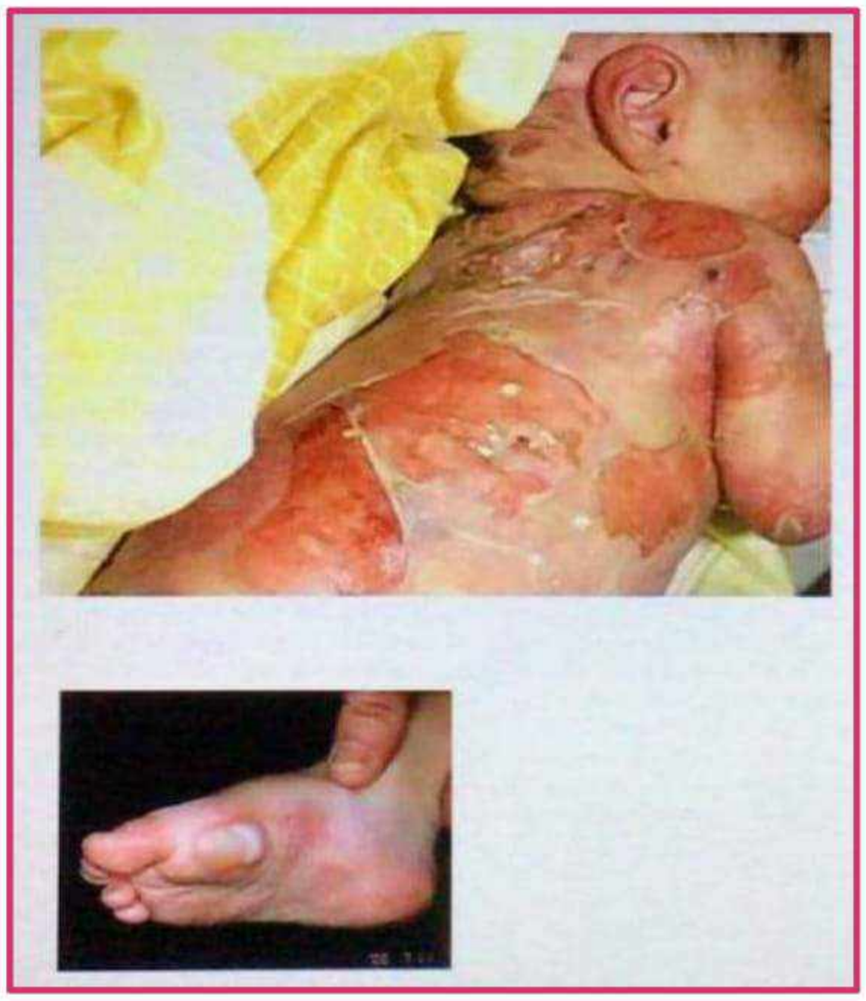
active space

	EBS	EBJ	EBD
Inheritence	AD	AR	AD/AR
Types	• Cockayne - Weber • Dowling Meara	• Herlitz • Non-Herlitz	• AD → Cockayne Tauraine • AR → Hallapeu Simens.
Defect	KRT 5, 14 (Keratin 5, 14)	Laminin 5	Collagen VII
Split	Intraepidermal	Basement Membrane Zone	Subepidermal.

- Age : At birth
- History : Bulla forms at trauma prone site.
- Site : Trauma prone
- Mucosal involvement : +/-

Clinical feature :

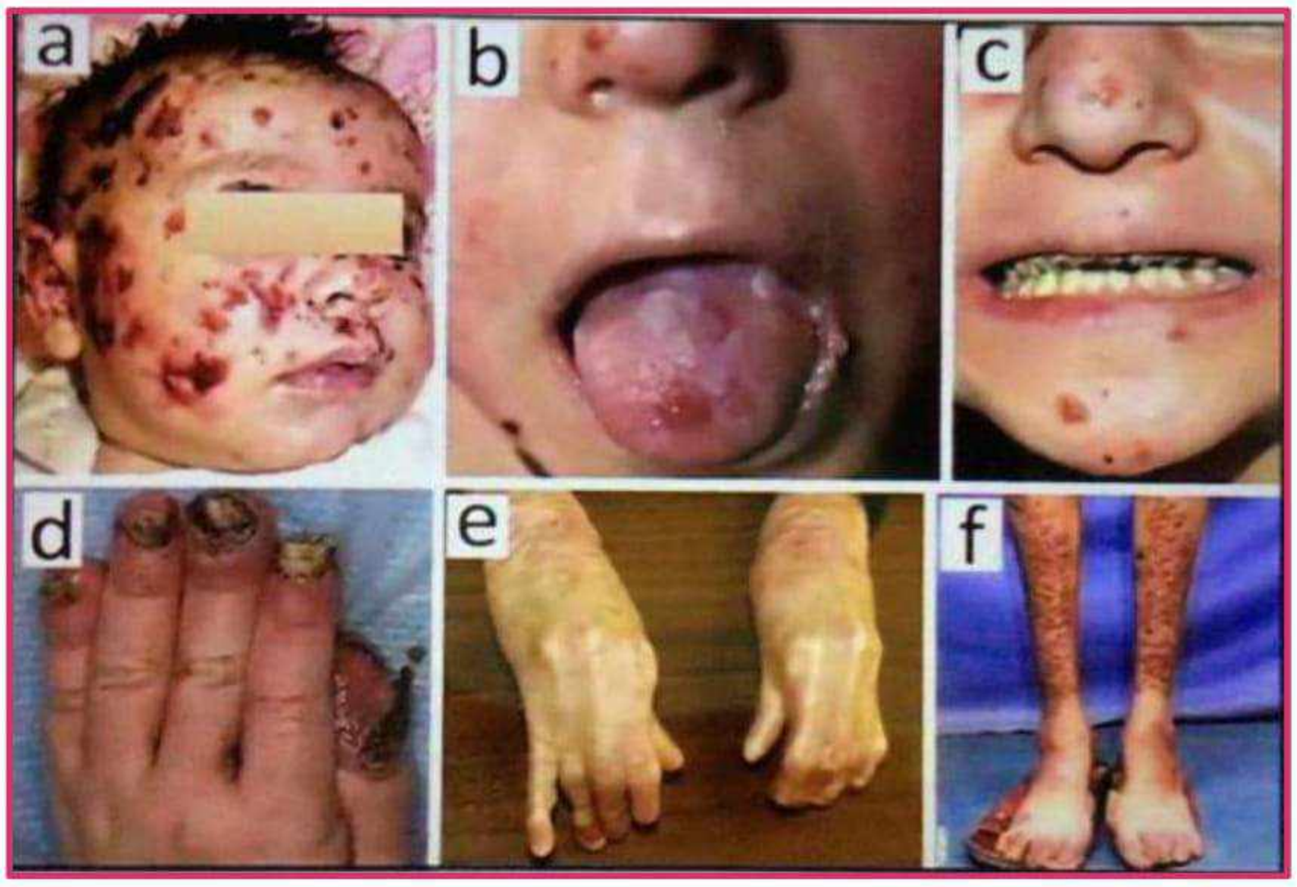
- Bulla and crusted erosions at site of trauma.



active space

Other associated features :

- Mitten hands and feet



Diagnosis

- Skin Biopsy → Split
- Electron microscopy → Diagnostic.
- DIF

Treatment :

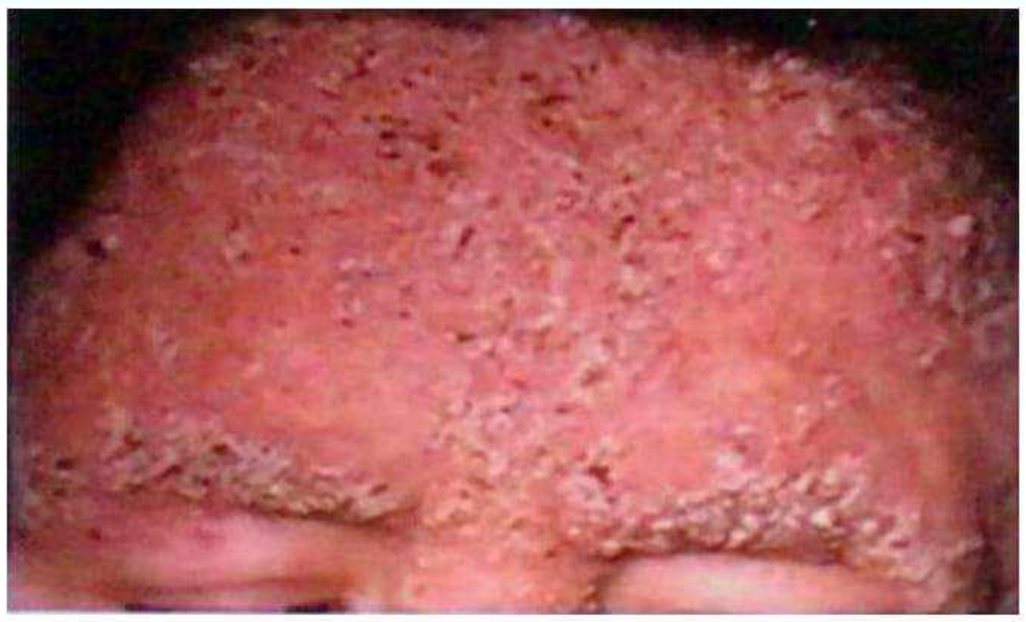
- No cure
- Symptomatic Treatment
- Gene therapy

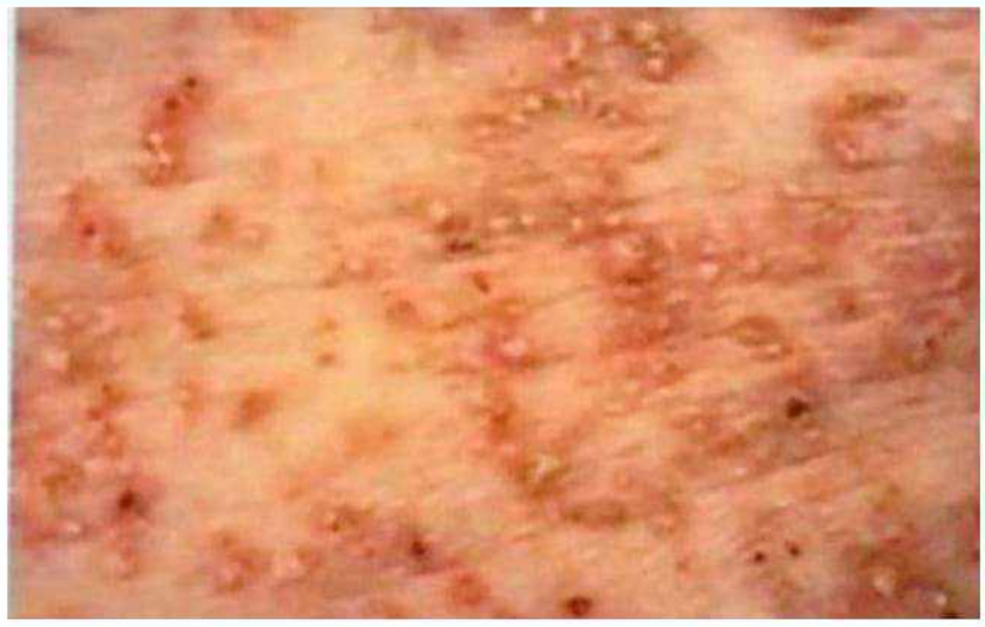
INHERITED ACANTHOLYTIC DISORDERS 48:25

Dariers Disease

- Inheritance : AD

- Defect : ATP2A$_2$ gene ⟶ regulates SERCA 2 pump
- Onset : usually childhood, Increases in Puberty
- Season : Worsens in summer season and on sun exposure.





Warty dirty looking papule on seborrheic areas

↓

covered with dirty looking crusts

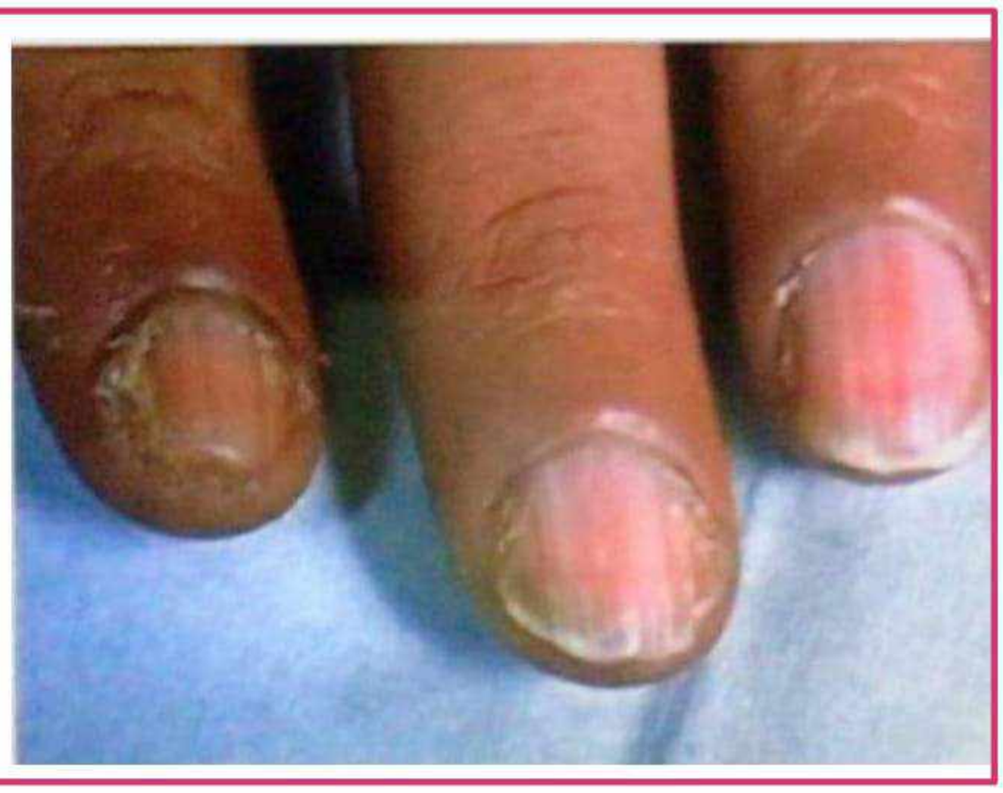
↓

Dimple at the summit of papule.

Nail changes :

- Longitudinal erythroleuconychia
- Triangular notching at free end of nail plate

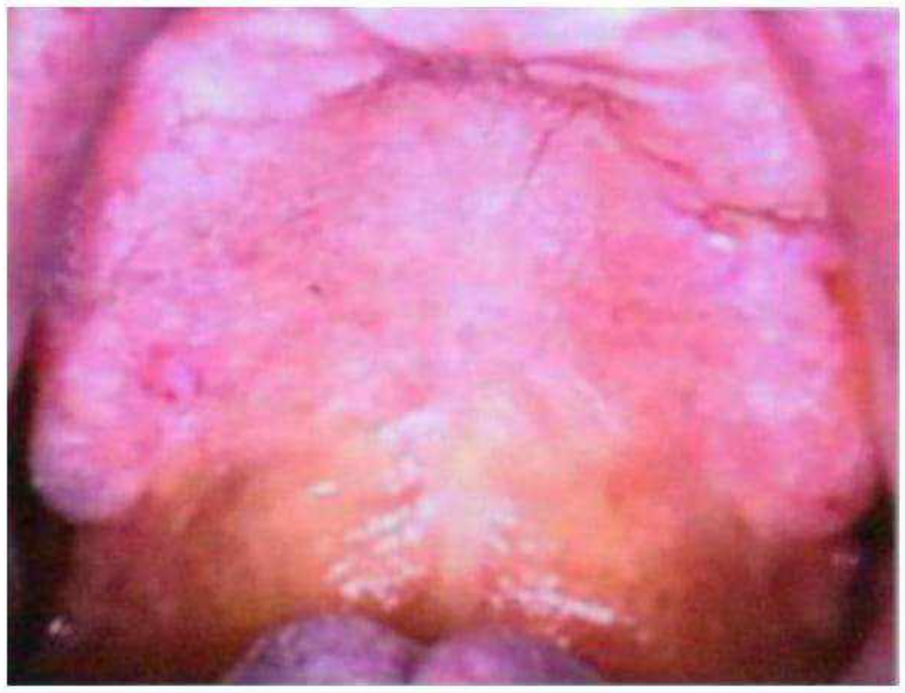
↳ 'v' sign.



active space

Oral mucosa:

- Cobble stoning of the palate.



On Histopathology:

- Suprabasal acantholysis
- Dyskeratosis - faulty or premature keratinizations
  - → Corps and Rods.

Causes of Dyskeratosis:

- Benign → Darrier's, Hailey Hailey, Grovers
- Malignancy → BCC, SCC, Actinic keratosis,
  Leukoplakia - Dyskeratotic congenita.

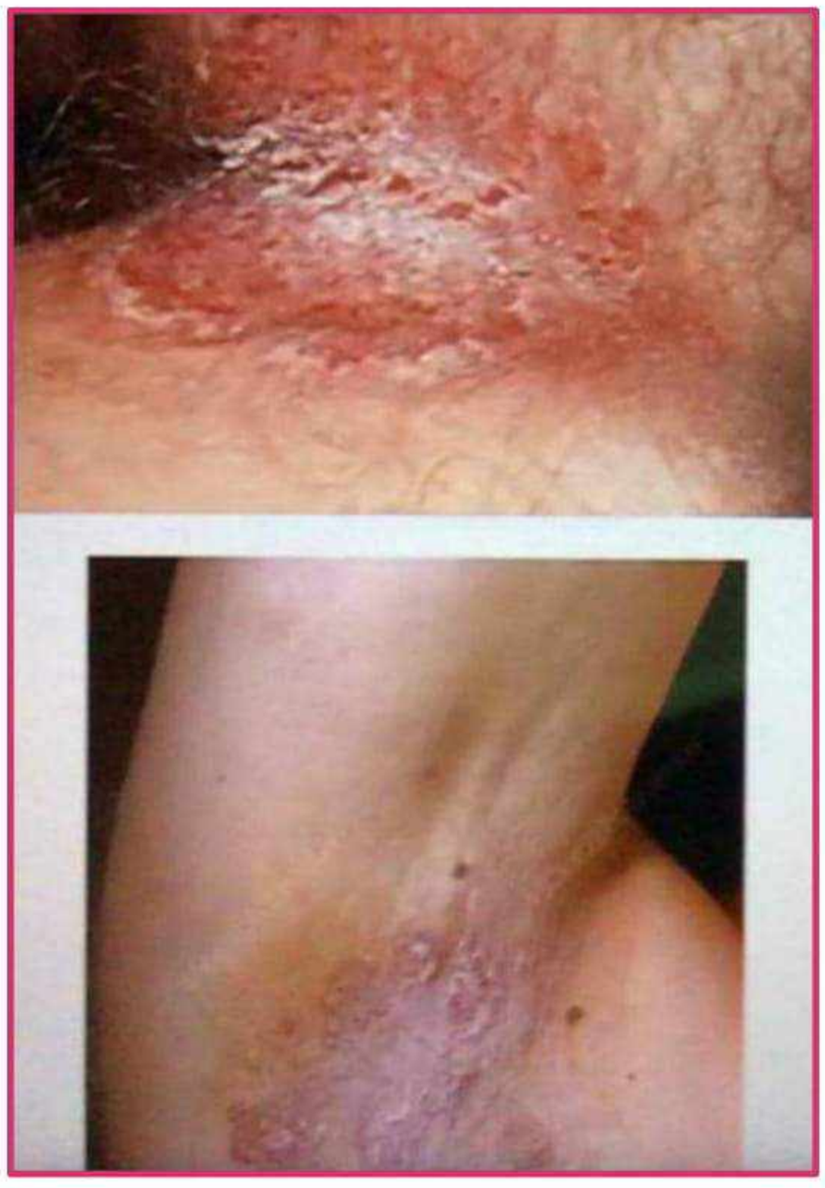
Treatment:

- Sunprotection
- Retinoids → Topical
  - → Oral

active space

Hailey Hailey Disease :

- a.k.a Benign Familial Pemphigus
- Inheritance : AD
- Defect : ATP2C1



Clinical feature :

- Vesicular eruptions on flexors
- Chronic course
- Maceration greyish scales over erythematous plaques.
- No mucosal involvement

On Biopsy :

- Suprabasal acantholysis
- Dipalidated Brick wall appearance.

active space

D/D : vs

i) Dariers → Dyskeratosis, Warty papules

ii) Pemphigus vegetans → No Family h/o, Mucosal involvement ⊕
Biopsy and DIF

iii) Tinea → KOH ⊕

Treatment :

- Topical steroids
- Antibiotics
- Dapsone.

ICTHYOSIS

01 : 01 : 25

- Disorder of keratinisation
- Dry scaly skin.
- Scaling - persistant.

Classification :

i) Congenital →
- Icthyosis vulgaris
- XLR - Icthyosis.
- Lamellar Icthyosis.

ii) Acquired

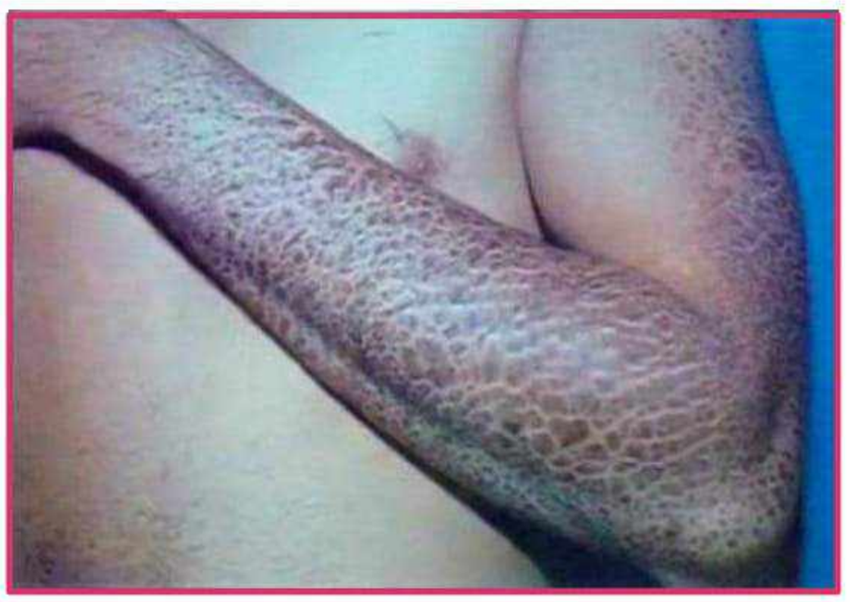
active space

Icthyosis Vulgaris :

- Inheritance : AD
- Defect : Fillagrin
- Onset : After 3 months

Clinical feature :

- Distribution : Extensors, flexures are spared
- Symptoms : Asymptomatic, dry itchy skin
- Season : Worsening in Winters
- Scale : Fine grey scales adherent at centre and upturned at edges
- Course : Persistent , improve with age
- Association : i) atopy
  ii) Palmoplantar hyperlinearity
  iii) Keratosis pilaris



Treatment :

- Moisturizers
- Keratolytics → Salicylic acid, Lactic acid.

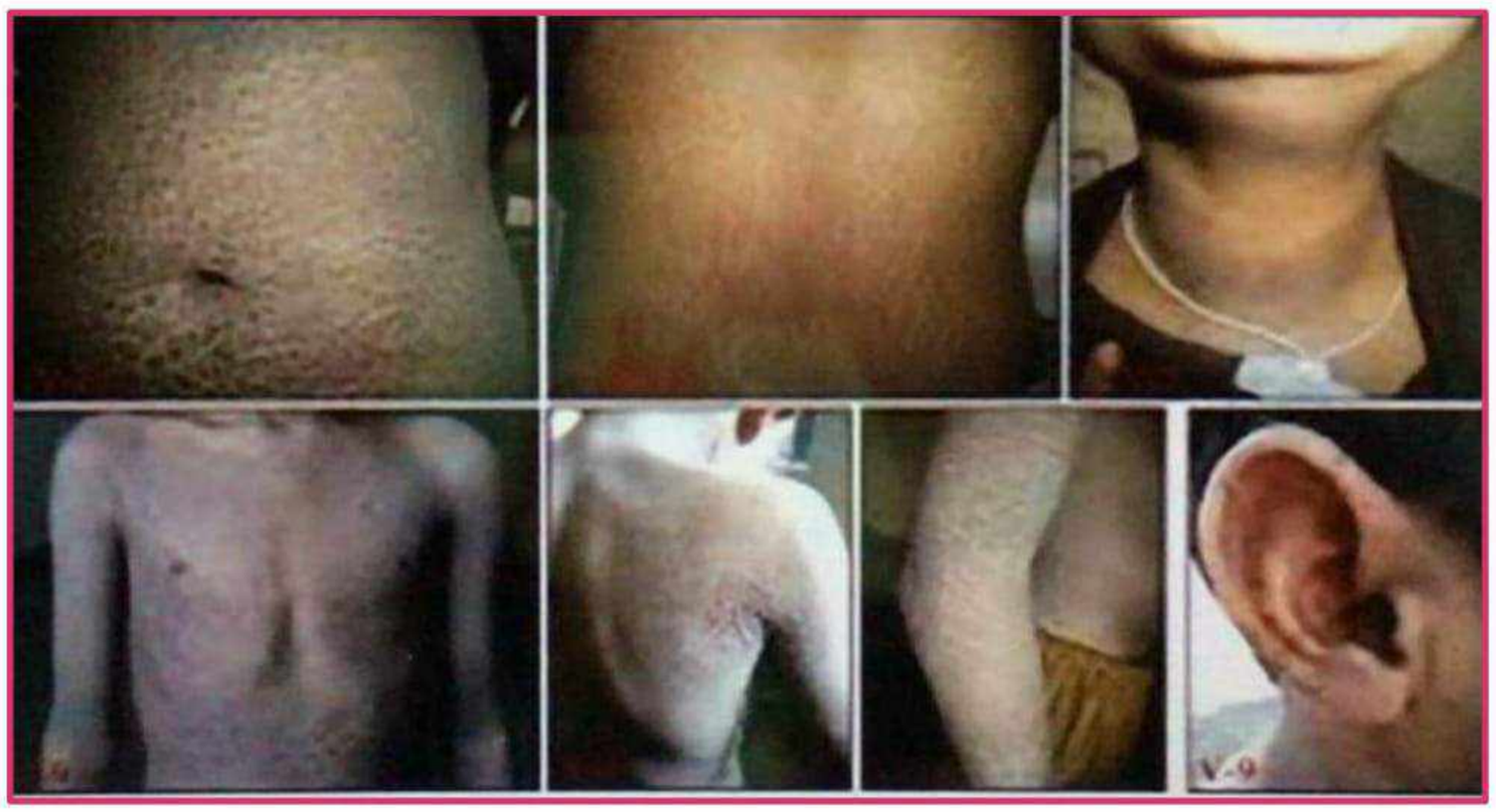
active space

X-Linked Recessive Icthyosis

- Inheritance: XLR (females - carrier, Males - Manifest)
- Defect : Steroid Sulphatase gene (STS gene)

Clinical features:

- Onset : After 3 months.
- Scale : Dirty Looking Scales
- Distribution : all body parts, palms and soles are spared.
  Flexural and Preauricular accentuation.



- Associations :
  - Comma shaped corneal opacities
  - Cryptorchidism
  - Prolonged labour

active space

Treatment: (similar to IV)

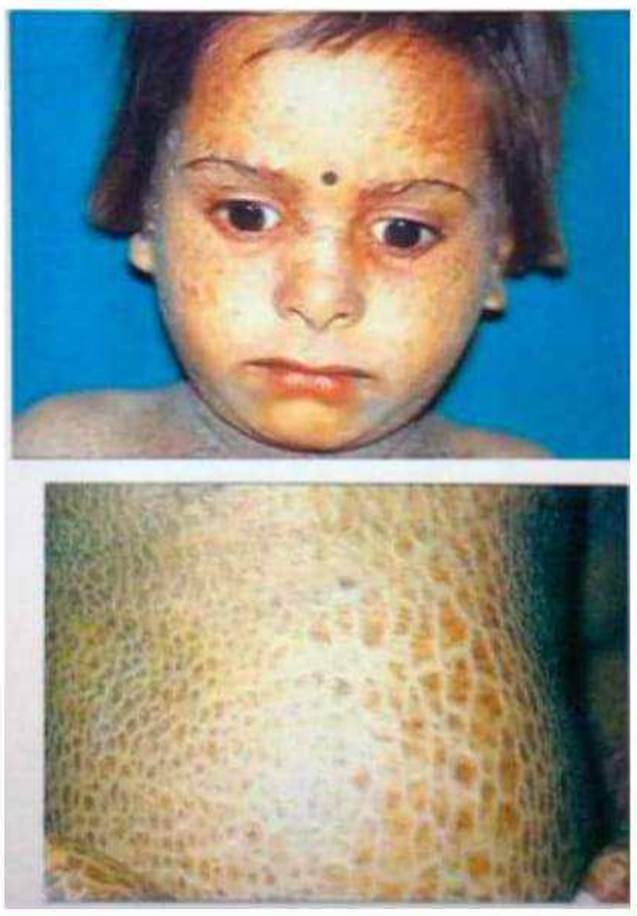
- Retinoids
- Oral Liarozole

Lamellar Icthyosis:

- Inheritance: AR
- Defect: Transglutaminase gene
- Onset: at birth → COLLODION BABY
  - Translucent parchment membrane covering over the body.

↓

Membrane ruptures in few weeks → Ectropion
→ Eclabium.



- Pasted adherent brown scales all over the body with flexural accentuation.

↓

FISH LIKE SCALES
(lower limb)

active space

Treatment :

- Retinoids can be given.

HARLEQUIN ICTHYOSIS

01:14:40

- Armoured appearance present at birth
- High mortality.

Causes of Acquired Icthyosis:

- Malignancy → Lymphomas
- Thyroid disorders
- Drugs → Clofazamine.
- Infections → Leprosy
- HIV

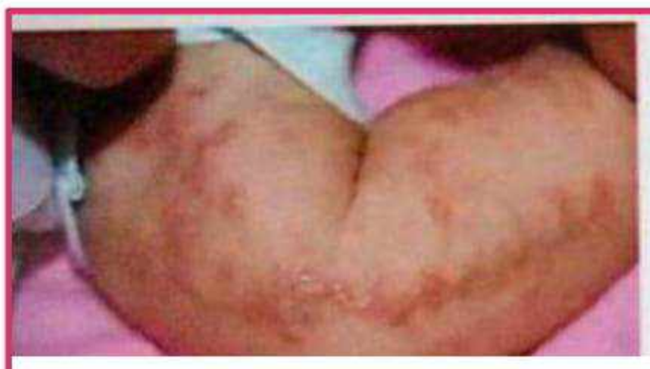
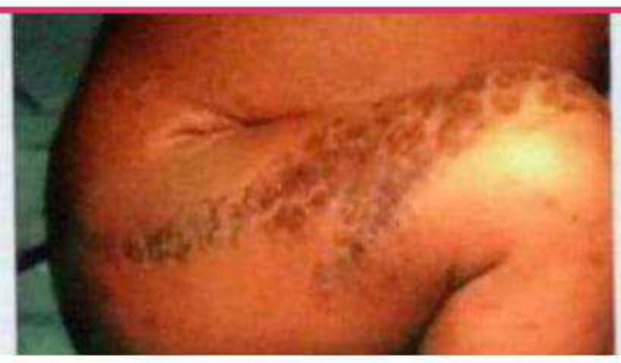
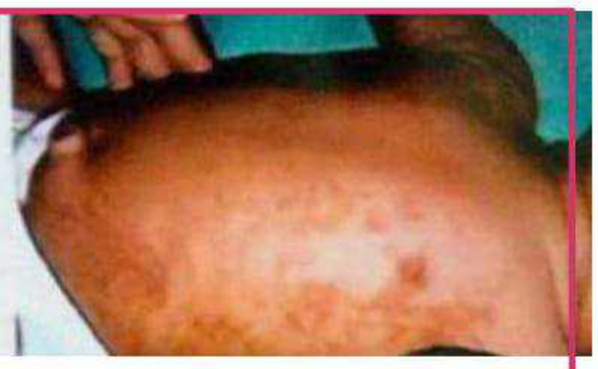
INCONTINENTI PIGMENTI

01:16:14

- a.k.a Bloch - Sulzberger Syndrome
- Inheritance: XLD (Male die in-utero)
- Gene involved : NEMO gene.

active space

- **3 Stages of Disease :**

		
Vesicular • 2-3wks (2 months)	**Verrucous** • 2wks to 6wks	**Pigmentation.** • 12 wks - 26wks. • (MC) Presentation

Extracutaneous features :

- **Dental** → Cone shaped crown
- **Ocular** → Blindness, Strabismus, Blue sclera
- **CNS** → MR, Epilepsy
- **Skeletal defects** → Dwarfism.


Cone shaped crown

Treatment :

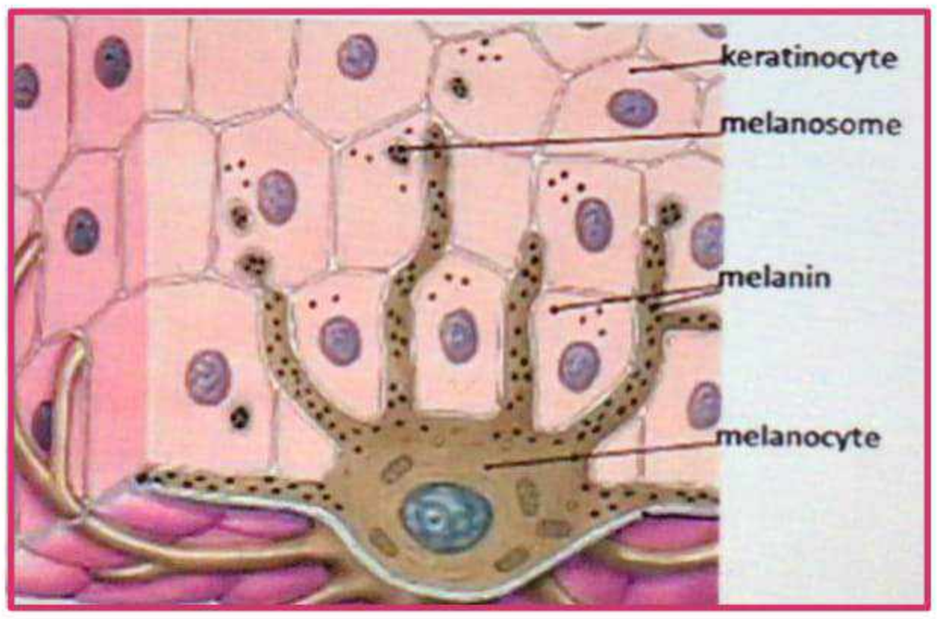
- No treatment
- Genetic counselling.

active space

Hyperpigmentary Disorders

Skin colour :

- determined by : i) Melanin
  ii) Hemoglobin
  iii) Carotenoids.
- 2 Types → Constitutive → genetically determined
  → Facultative → sun exposure and hormones.



- Cell → Melanocyte → present in Basal layer, derived from neural crest.

Melanosomes - organelles containing pigment melanin.

↓

Keratinocytes.

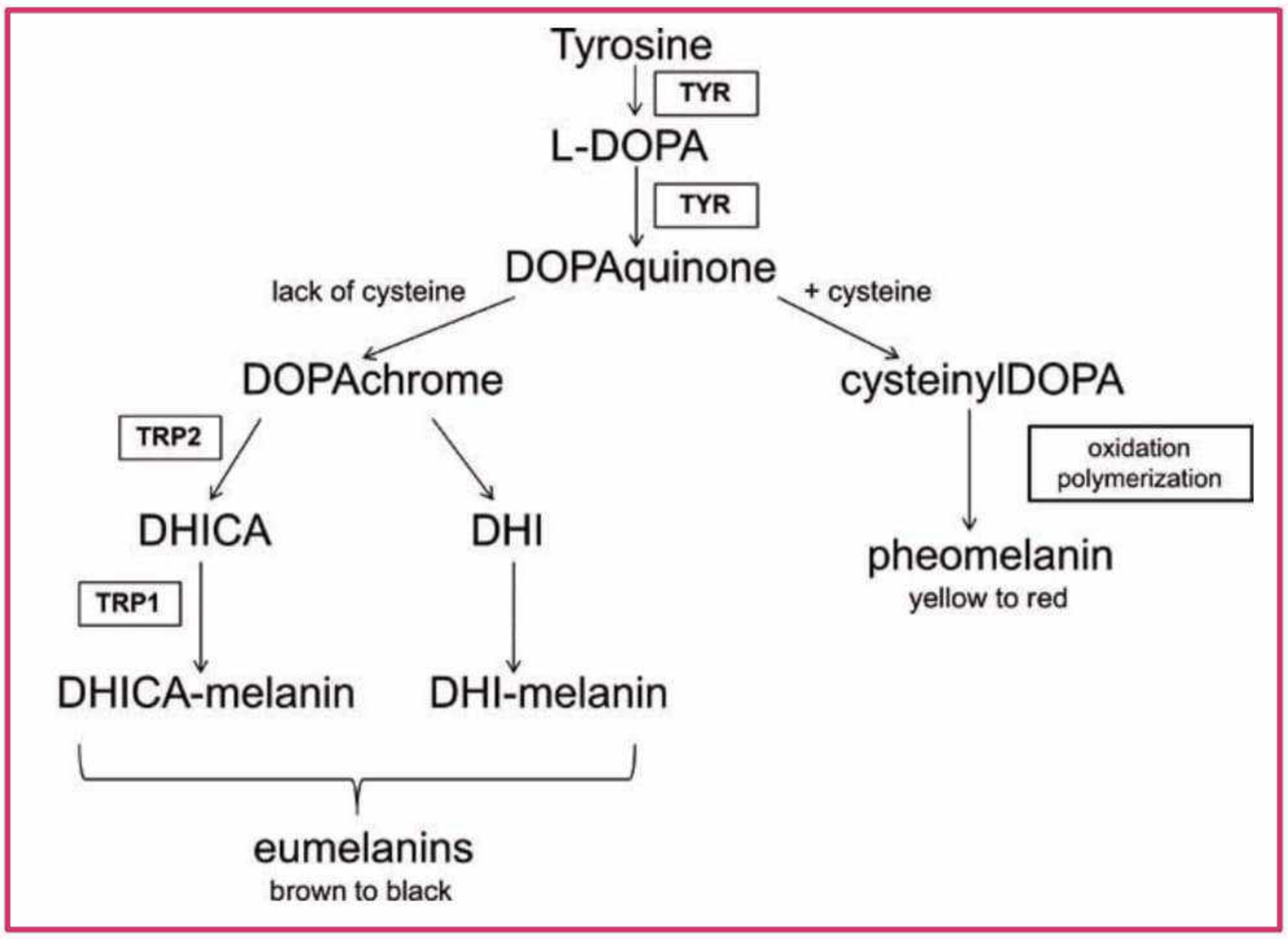
[Epidermal Melanin Unit → 1 : 36]

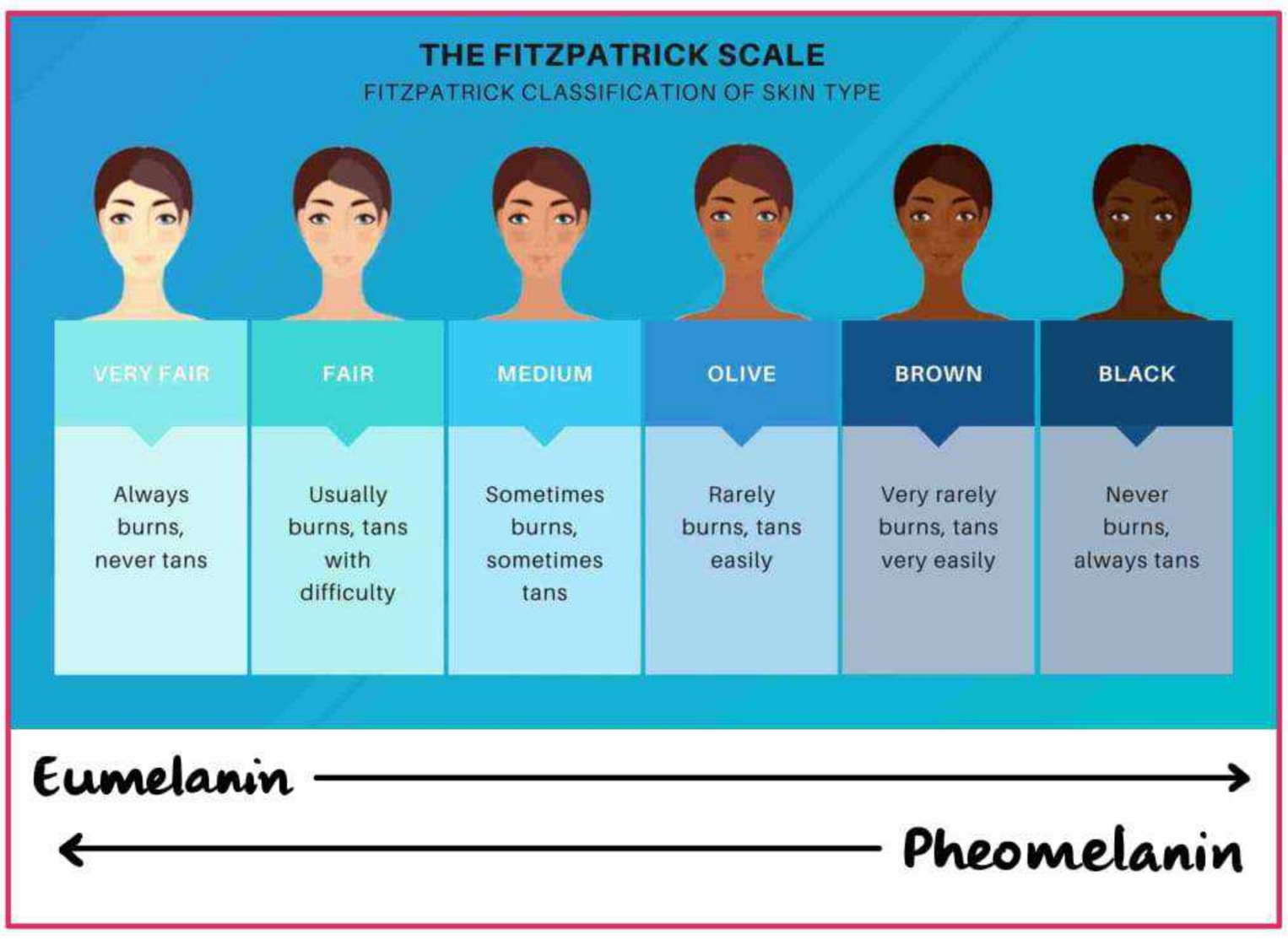
- Skin colour is determined by :

  i) Number and distribution of melanosomes

  ii) Type of melanin.

- Eumelanin → Black to Brown colour
- Pheomelanin → Reddish yellow colour.





Eumelanin →

← Pheomelanin

active space

- Indian skin type → III to IV

Classification of Pigmentary disorders

Increased pigmentation → **Hyperpigmentary**

Decreased pigmentation. → **Hypopigmentary**

- Depigmentation → No colour
- Hypopigmentation → Decreased pigmentation.

Disorders of Hyperpigmentation

- **Hyperchromia:** Increased pigmentation
  - → Melanotic
  - → Non-melanotic drugs and metals.

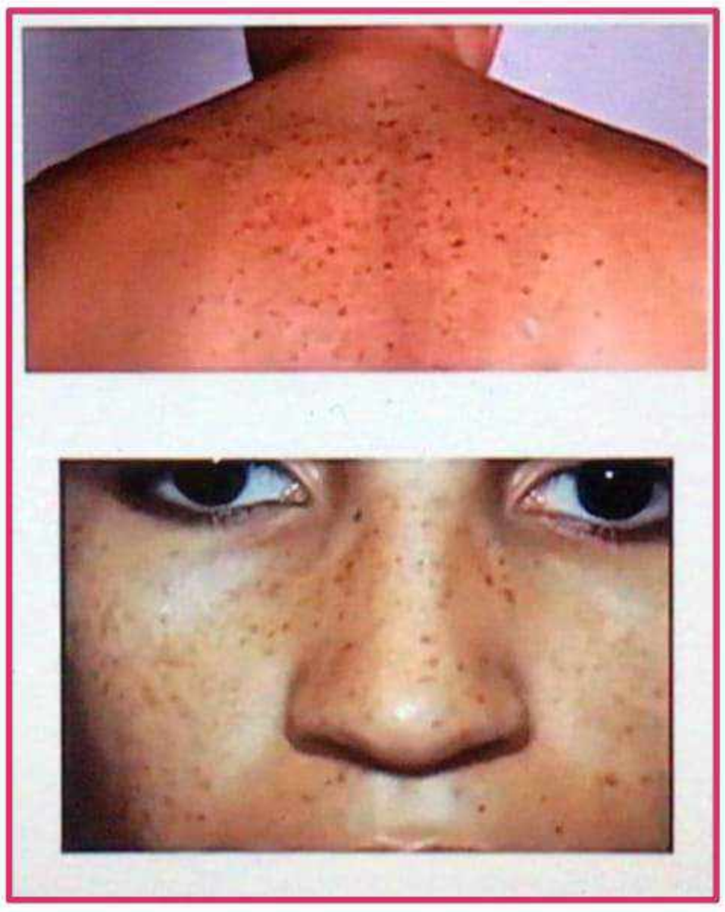
- **Hyperpigmentation** → Melanotic
  - → ↑ Melanin
  - → ↑ Melanocytes.

- **Melanotic hyperpigmentation:**
  - Classification
    - → Epidermal pigmentation → Brownish
    - → Dermal pigmentation. → Bluish, resistant to treatment.
      - → Tyndall Effect.

active space

LENTIGO

15:00



- a.k.a **Lentigines** (if > 1 are present)
- **Lentil shaped**.

Clinical feature:

- **Brown circumscribed macules** present anywhere on body.

Pathology: **Increased number** of melanocytes

Associated with:

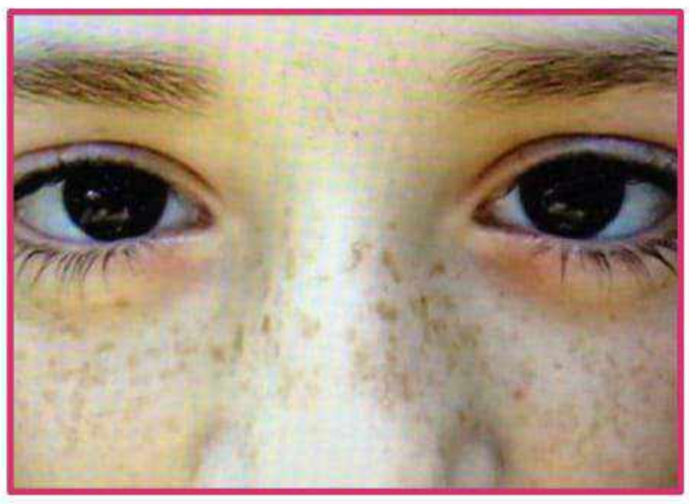
i) Leopard

ii) Peutz jegher

iii) Conckite canda syndrome.

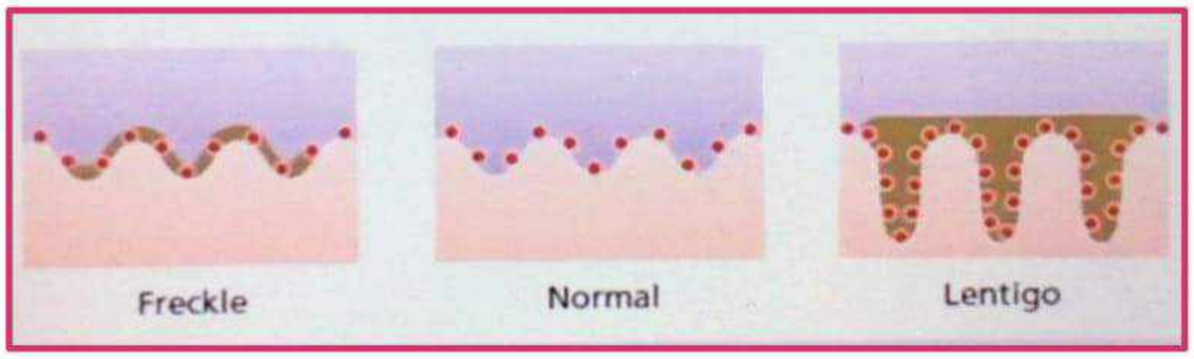
FRECKLES

17:05

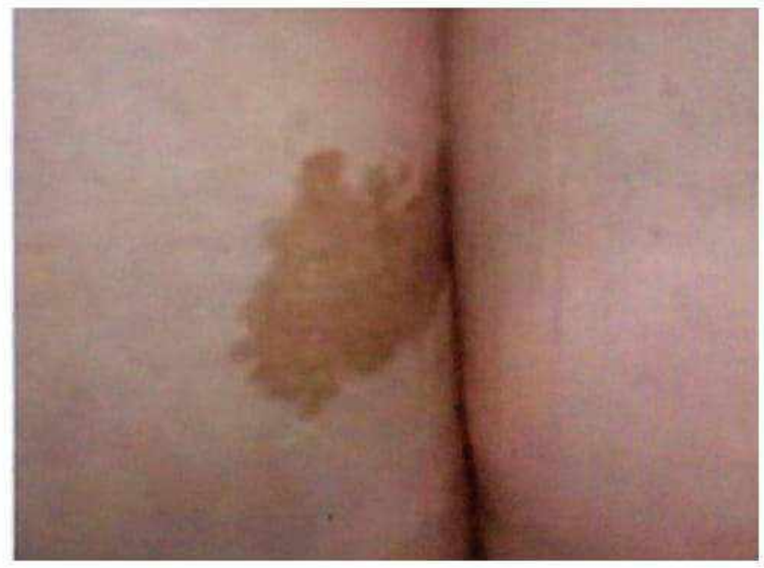


- present on **sun exposed areas**.
- **Light brown** macules
- **Increased melanin production** by melanocytes in response to **UV radiation**.
- It increases in summer season.

active space



- Cafe au lait macules
- Coffee coloured macules



Clinical feature:

- Tan brown macule with irregular / serrated borders
- Age : present at birth

Pathology: ↑ Melanosome production.

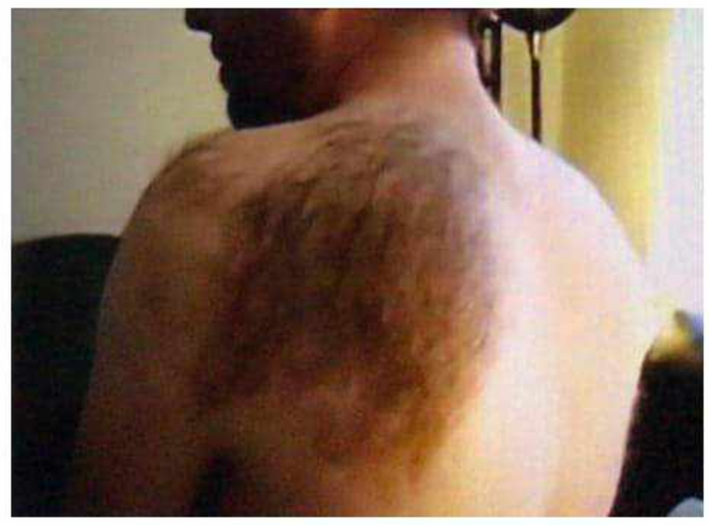
- They can be of 2 Types:

i) Isolated → • < 3 in number
• 0.5 to 1.5 cm in size
} seen in 10-20% cases

ii) Multiple: • > 3 in number
• > 0.5 to 1.5 cm in size

Associations: i) Neurofibromatosis iii) Albright syndrome
ii) Tuberous sclerosis

Becker's Nevi



- Epidermal naevi
- present in adolescent age group
- presents as patch of hyperpigmented multiple coalescing macules

↓

Hypertrichosis

- Site : shoulder, upper back , chest.

MELANOCYTIC NEVI 25:07

- a.k.a **Moles**
- proliferating nevus cells derived from neural crest.
- Classification :

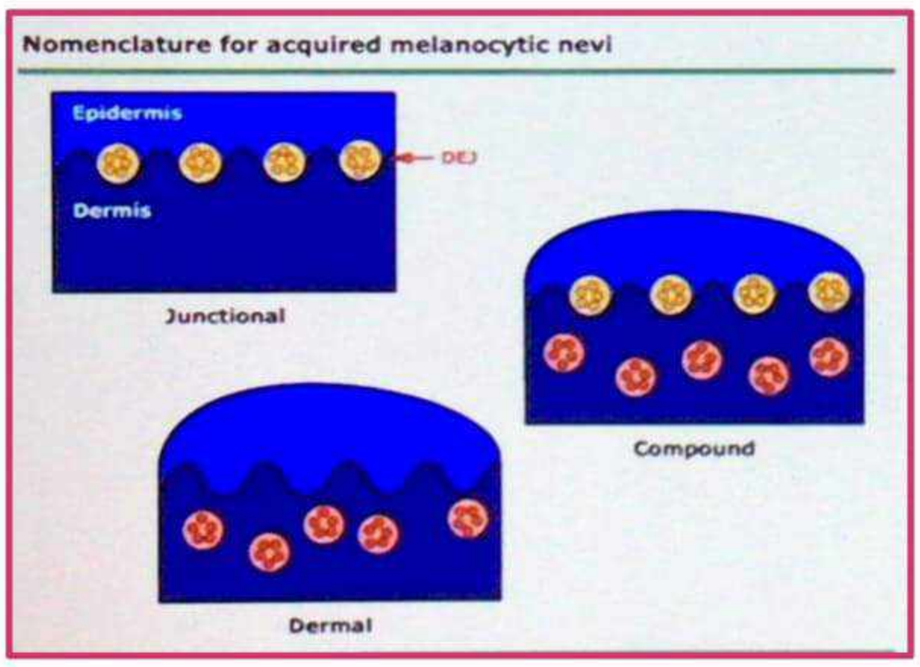
i) Epidermal → • Congenital
• Acquired

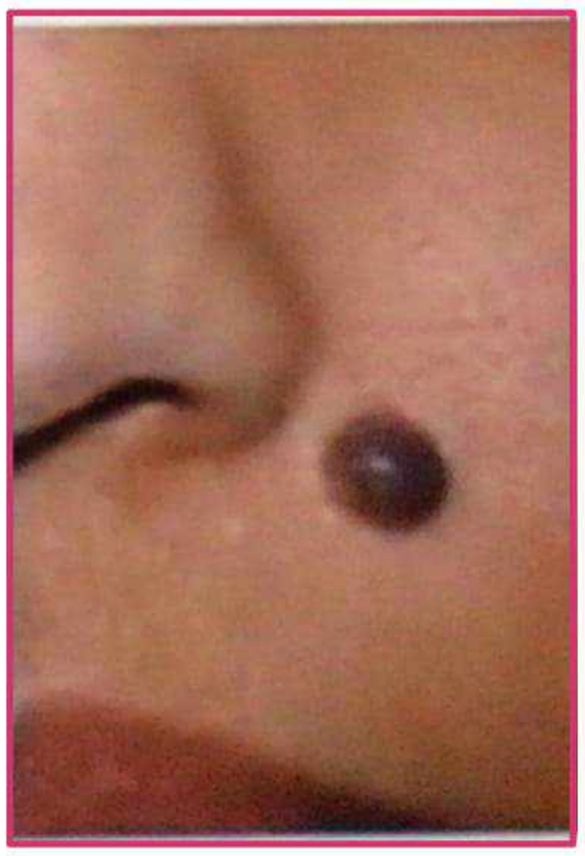
ii) Dermal → i) Nevus of Ota
ii) Nevus of Ito
iii) Mongolian spots.

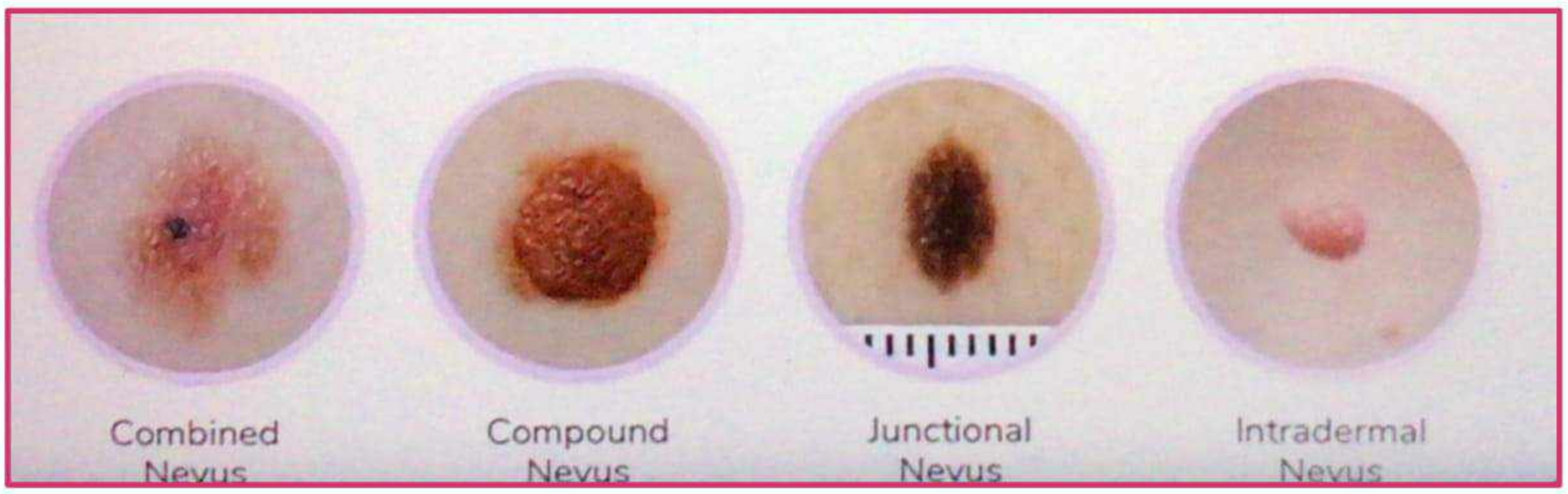
active space

Acquired Melanocytic Nevi

- a.k.a Mole
- Defect :







↓ Light Brown papule/ nodule

↓ Brownish Black macule.

↓ Skin coloured hemispherical nodule.

- Nests of nevi cells at DEJ ⟶ Junctional nevi
- Nests at DEJ + dermis ⟶ Compound nevi
- Nests in dermis ⟶ Dermal nevi

active space

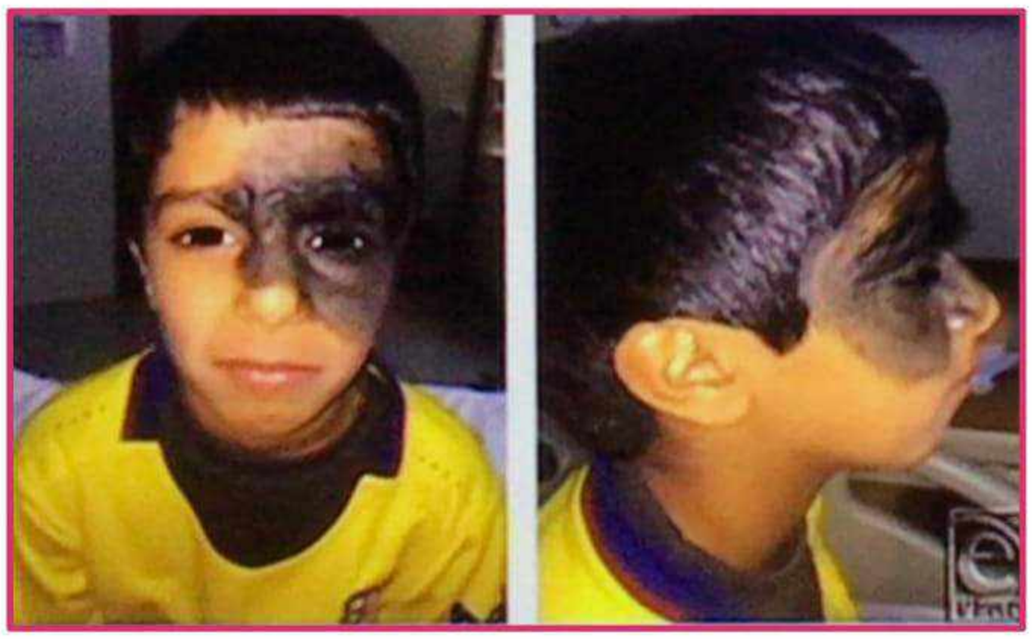
- Malignancy potential → low

Suspect malignancy when: i) size > 7cm

ii) Irregular border or pigment

iii) Bleeding, oozing, crusting, redness.

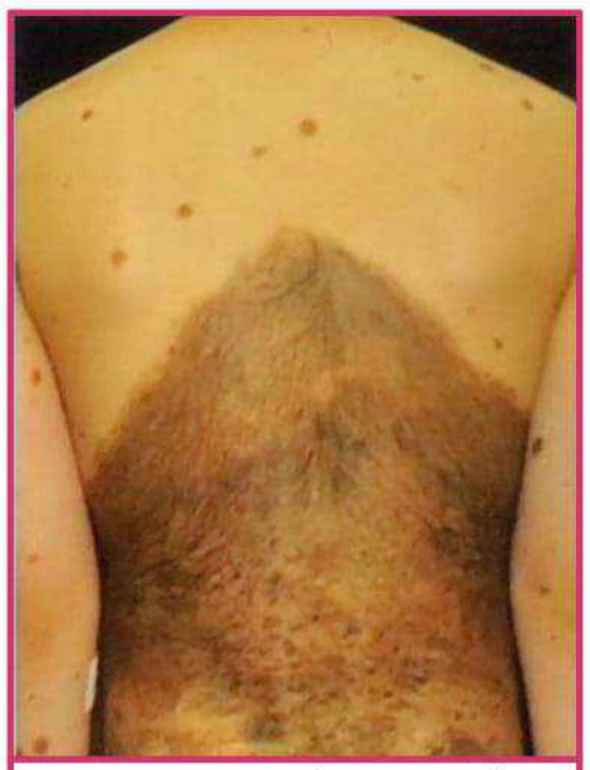
Congenital Melanocytic Nevi :



- present at birth
- solitary lesion
- Hyperpigmented
- Hypertrichosis
- Rugositis

} are seen.

- Grows in proportion with body.



Giant Melanocytic Nevi.
Bathing Trunk / Garment Nevi

↓

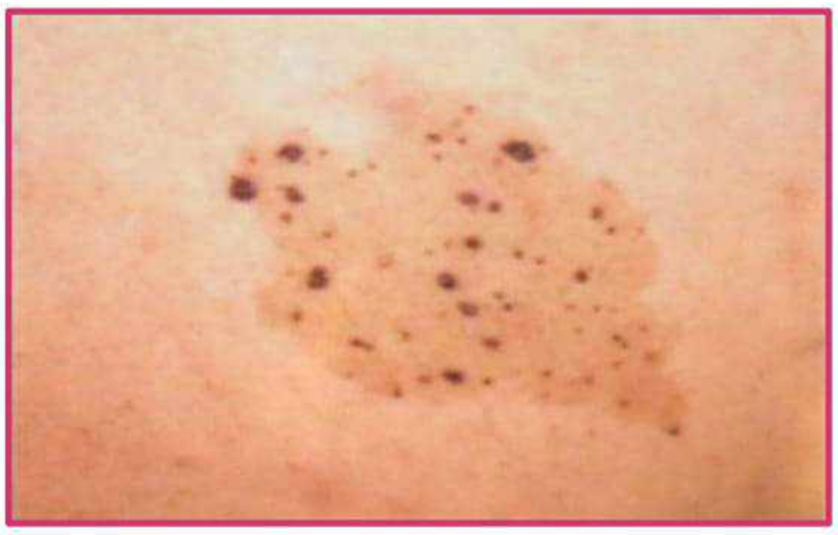
High Malignant Potential.

Nevus spilus

- speckled lentiginous nevus
- Tan / light brown macule

↓

Interspersed dark brown macules / papules



Halo Nevi

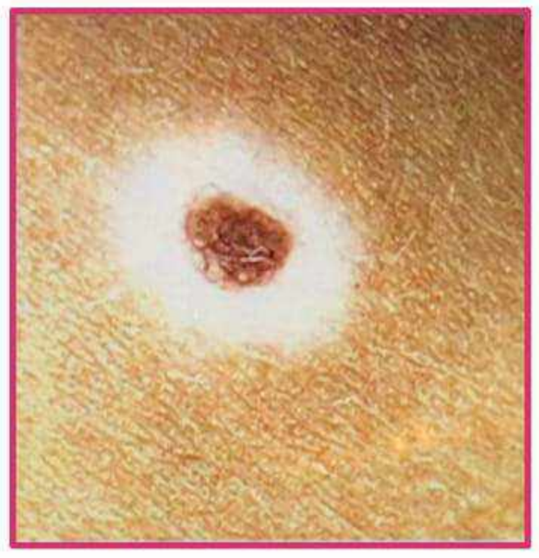
Acquired Melanocytic Nevi

↓

Surrounding melanocytes get destroyed

↓

Halo / depigmentation around nevi.



Treatment of Epidermal Melanocytic Nevi :

- Rx done due to

  i) Cosmetic reasons

  ii) Suspecting malignancy.

- Do excision → Send for Biopsy.

DERMAL MELANOCYTIC NEVI

38:40

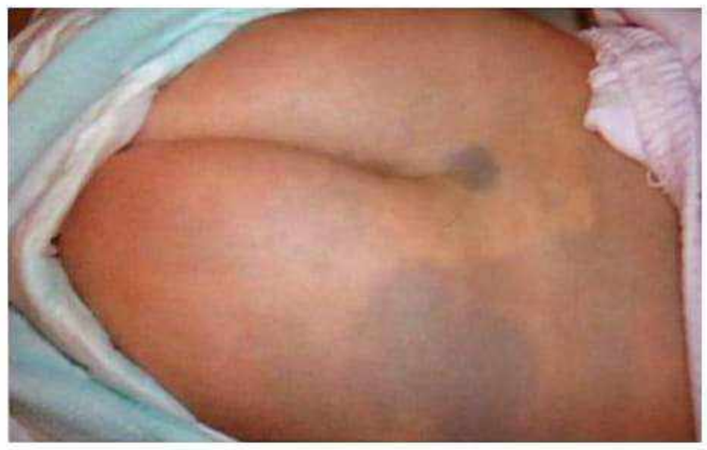
- Nests of nerve cells in Dermis → Tyndall Effect.

↓

Bluish colour.

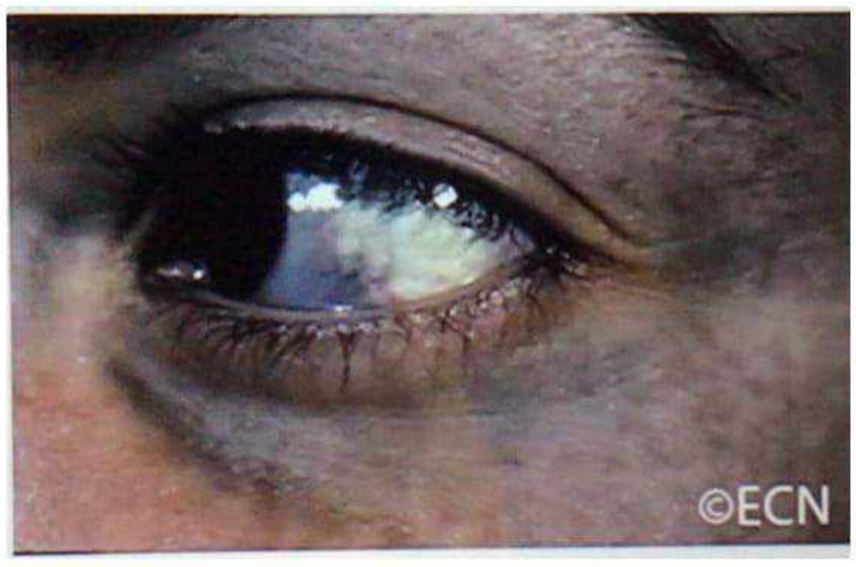
active space

Mongolian Spots



- present at birth
- Site : Sacral areas
- Bluish macules with well to ill defined borders.
- Spontaneously Resolve.

Nevus of Ota



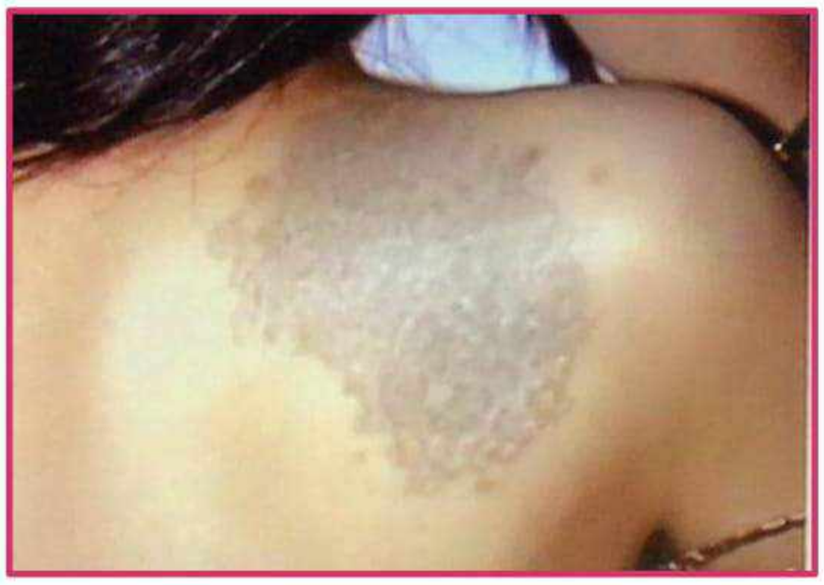
- a.k.a Nevus Fuscoceruleus Ophthalmomaxillaris.
- More commonly seen in Japan.
- Presents as
  - U/L speckled lesion
    ↓
    Brownish or Bluish macules
- seen along ophthalmic and maxillary division of Trigeminal nerve
- Involves →
  - Periorbital
  - Forehead
  - Temples
  - Malar
  - Nose.

- Bluish sclera
- Brown spots on conjunctiva

} other findings.

Nevus of Ito

- a.k.a Nevus Fuscoceruleus Acromioclavicularis.
- involves :
  - i) Lateral Brachial Nerve
  - ii) Posterior Suprascapular Nerve



- seen over scapula, deltoid, supraclavicular area.

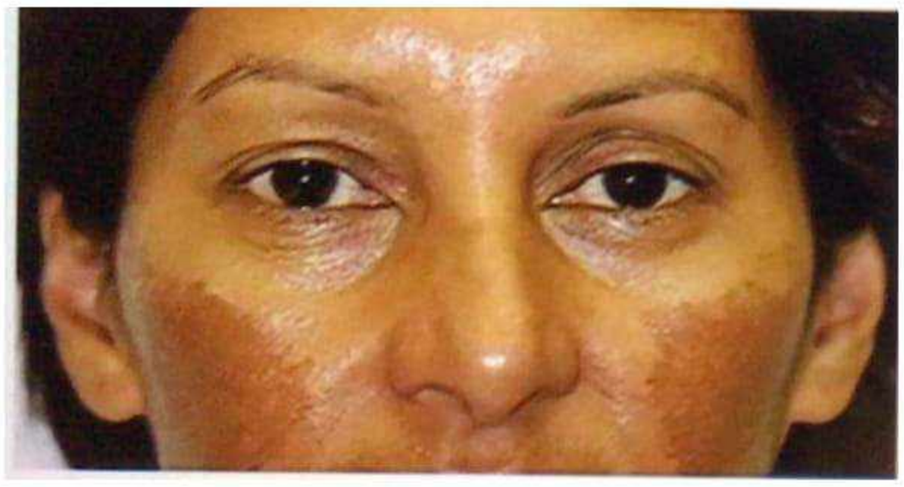
Treatment

- Cosmetic camouflage
- LASERS used → i) Ruby
  - ii) Alexandrite
  - iii) Nd : YAG

MELASMA

44:35

- (MC) Facial melanosis.
- seen in F > M



active space

- **Predisposing factors :**

  i) Sun

  ii) Pregnancy (Chloasma)

  iii) Drugs → OCP's

  iv) Thyroid disorder

Clinical feature : B/L symmetrical lesions brownish macules.

- Defect : Melanocytes
  ↓
  Hyperactive
  ↓
  Larger and more dendritic
  ↓
  secrete more melanin.

- site : i) Centrofacial (MC)

  ii) Malar

  iii) Mandibular

- Types :

  i) Epidermal

  ii) Dermal

  iii) Mixed

active space

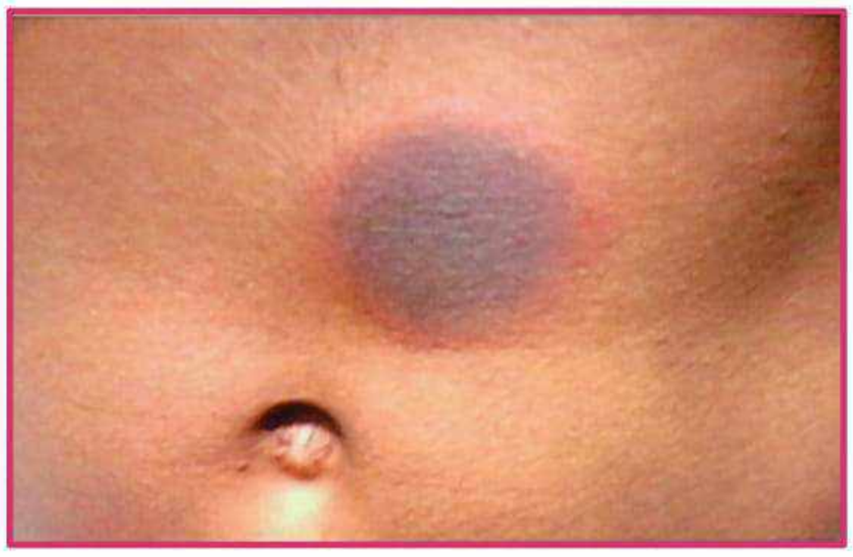
Epidermal		Dermal
Brown		Bluish hue
Accentuation	On Wood's lamp	No accentuation. Decreased Response to Rx.

Treatment:

- Sun protection → - Sunscreens
  - Barriers.
- Topical agents
  - Hydroquinone.
  - Glycolic acids
  - Arbutinin
  - Lactic acid.
  - Azelaic acid.
- Oral → Tranexamic acid.
- Chemical peels
- LASER Therapy
- KLIGMAN's Regimen → Topical
  - ↳ Triple combination : Hydroquinone + Tretinoin + Mild Steroid.

active space

Fixed Drug Eruptions



- occurs at the same site whenever patient take the drug NSAID's, Antibiotics.
- Appear as hyperpigmented violaceous lesions.

Diffuse Hyperpigmentation

- **Cause** :

  i) Addison's disease.

  ii) Hemochromatosis

  iii) Chronic Renal Failure

  iv) HIV, AIDS

  v) Drugs → Anti-malarials, Minocycline.

Treatment

Agents used.

Tyrosinase inhibitors	Melanocyte Cytotoxic	Others
• Hydroquinone • Arbutin • Kojic acid • Licorice extract • Vit.E	• Azelaic acid	• AHA • Resocrinol • Vit.C • Tretinoin.

active space

- Tranexamic acid
- Chemical peels
- LASERS.

active space

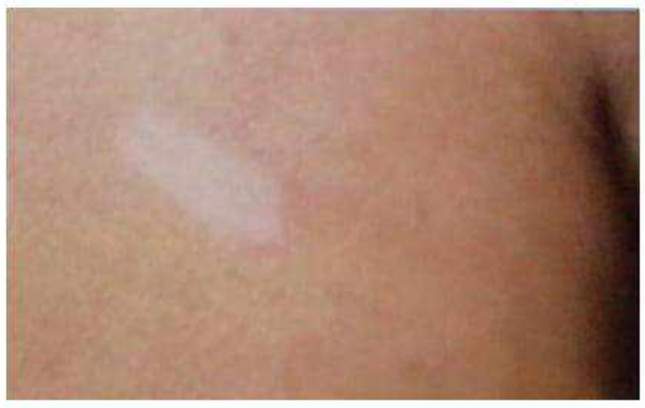
Hypopigmentary Disorders

HYPOPIGMENTARY DISORDERS

00:10

- **Hypopigmentation:** Decreased pigment
- **Depigmentation:** Complete loss of pigmentation.
- **Developmental:** Nevus depigmentosus, Nevus Anemicus
- **Genetic:** OCA, Piebaldism
- **Miscellaneous:** Vitiligo, Hypomelanosis of Ito
- **Post inflammatory:** T. versicolor, PKDL, Leprosy
- **Nutritional:** Kwarshiorkar
- **Chemical**

Nevus Depigmentosus



- Congenital condition
- Stable leukoderma
- Melanosome aggregation in melanocytes.

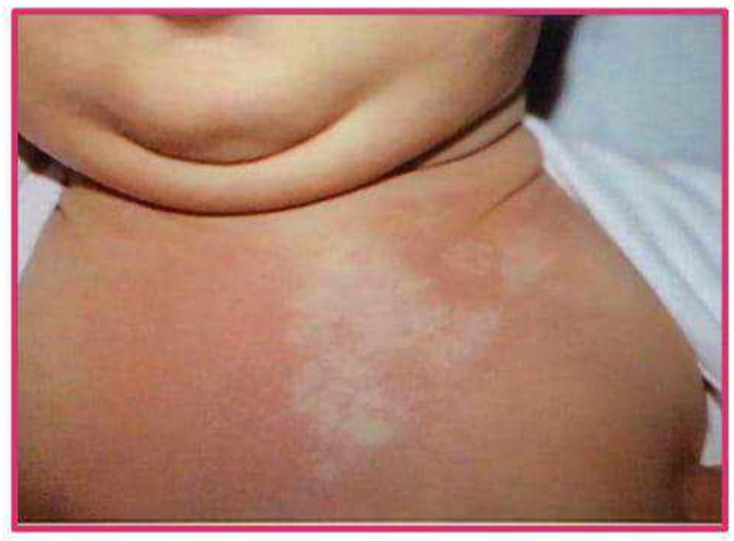
  ↓

  But not transferred to keratinocyte.
- Presents at birth as solitary de/hypopigmented macule has a serrated margin.

active space

- Doesnot cross midline.
- vs. Vitiligo -
  - present at birth
  - Stable
  - grows in proportion to body

Nevus anemicus



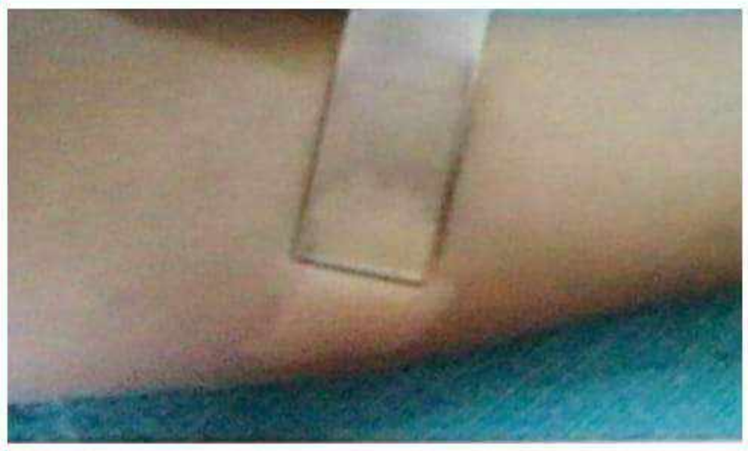
- it is a pharmacological nevus
- developmental anomaly
- capillaries are oversensitive to catecholamines

↓

Vasoconstriction

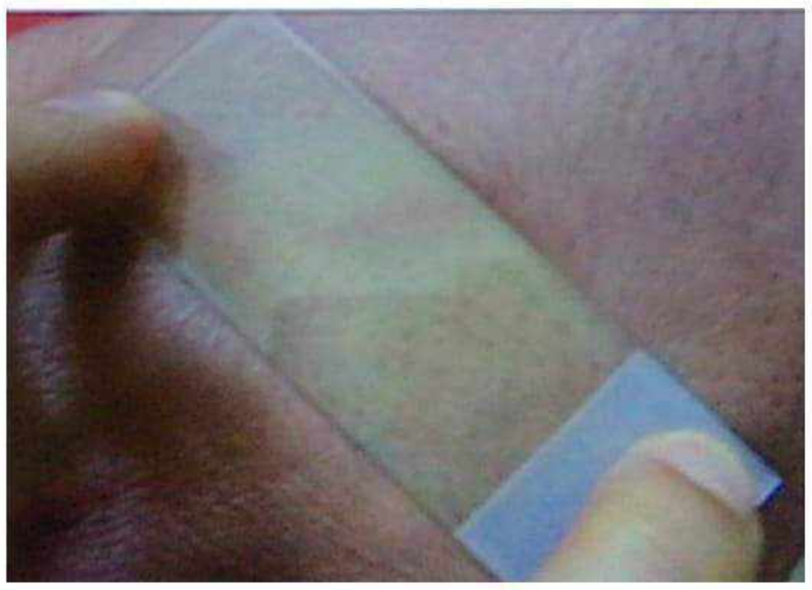
- Present as hypopigmented lesion.
- Present since birth

On Diascopy



Nevus depigmentosus

- No change in the margins.



Nevus anemicus

- Margins will be lost and merges with normal skin.

ALBINISM

09:03

- Defect : Congenital disorder → absence of Eumelanin from skin, hair and eyes.
- Pathogenesis : defect in production of Tyrosinase
- Types :

i) Ocular albinism :

- Only eye involvement
- XLRI

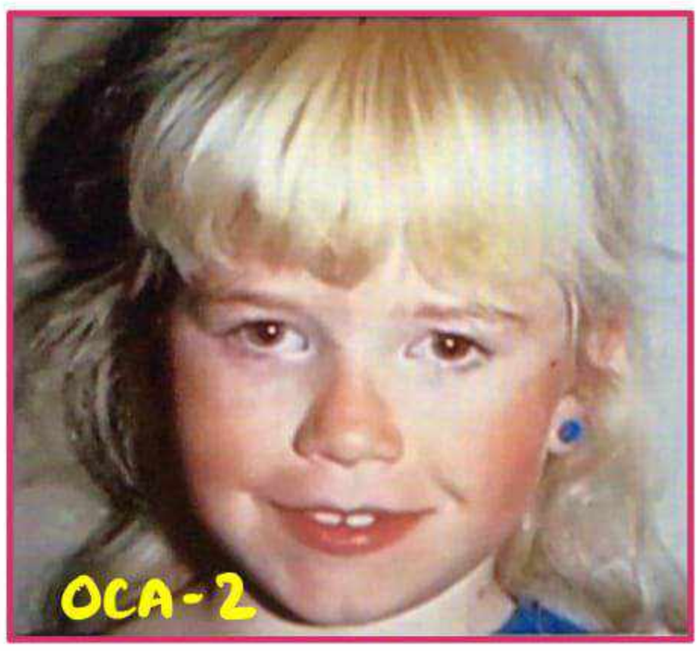
ii) Oculocutaneous albinism (OCA) → Albino kid

- There is involvement of skin, hair, eyes.
- AR
- Types : i) OCA 1 - Tyrosinase Negative OCA

ii) OCA 2 - Tyrosinase Positive OCA.

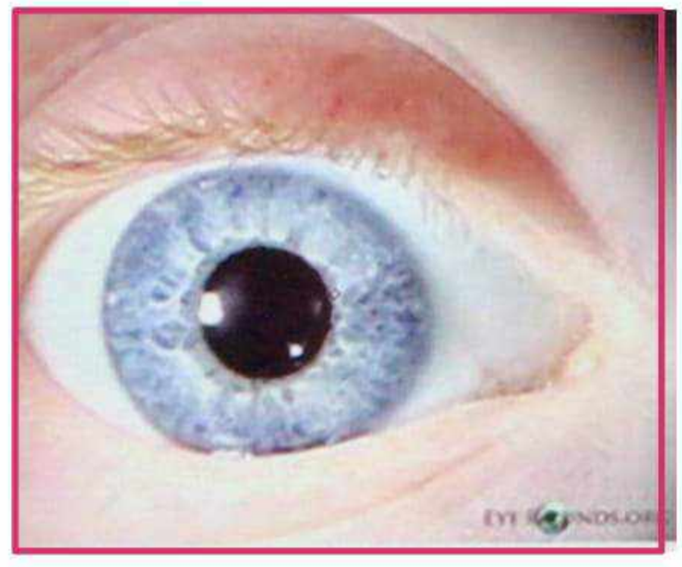
active space

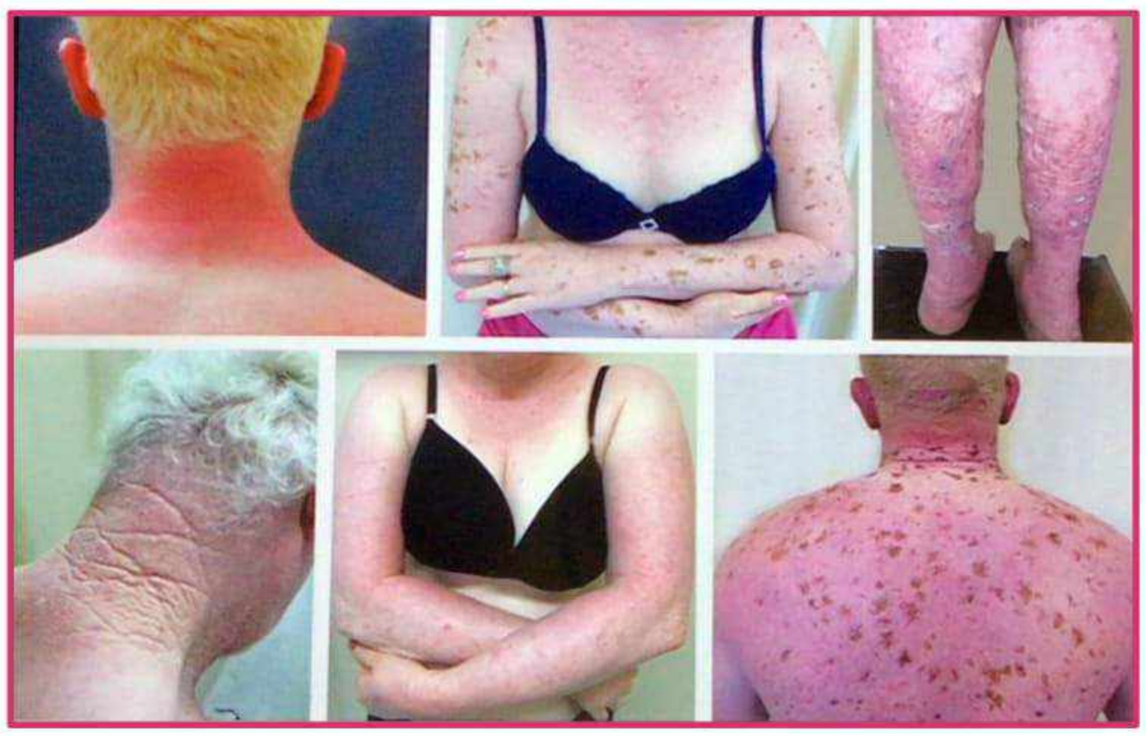
- OCA 2 → some pigmentation on hair and also on skin.



Clinical feature :

- Skin → depigmented white skin
- Hair → White hair.
- Eye → Blue Iris.
- Photophobia
- Nystagmus
- Impaired visual activity.





Complications

- Sunburns
- Pigmentary changes.
- Prone to Malignancies
- Photosensitivity.
- Actinic keratosis.

Treatment

- No definite cure
- Strict sun protection
- Genetic counselling
- Screen for Malignancies every year.

PIEBALDISM

17:50

- AD
- Defect: KIT gene

C/f

- White triangular or rhomboid patch of depigmentation
- Present in frontal, median.

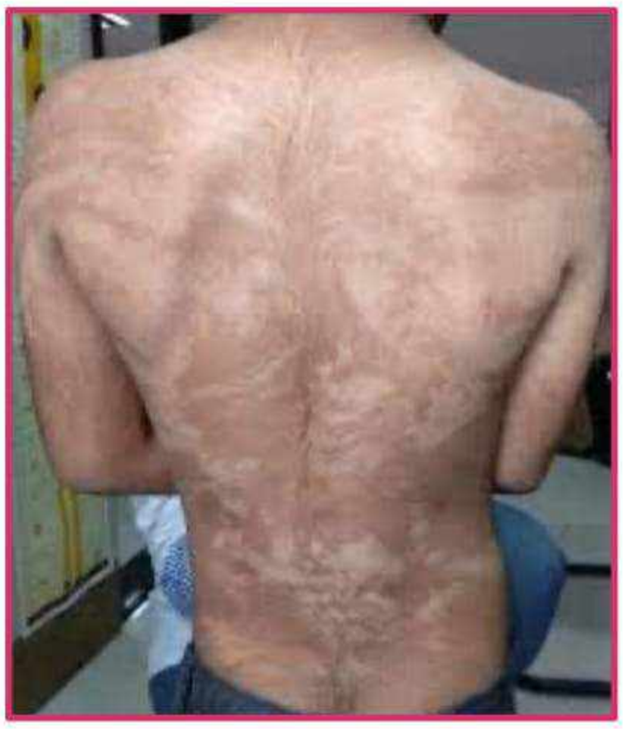
active space

- White forelock of hair.
- Islets of hyperpigmentation on depigmented macule.
- Stable, present since birth

HYPOMELANOSIS OF ITO

20:40

- Etiology: Mosaicism
- Lesions are present at birth



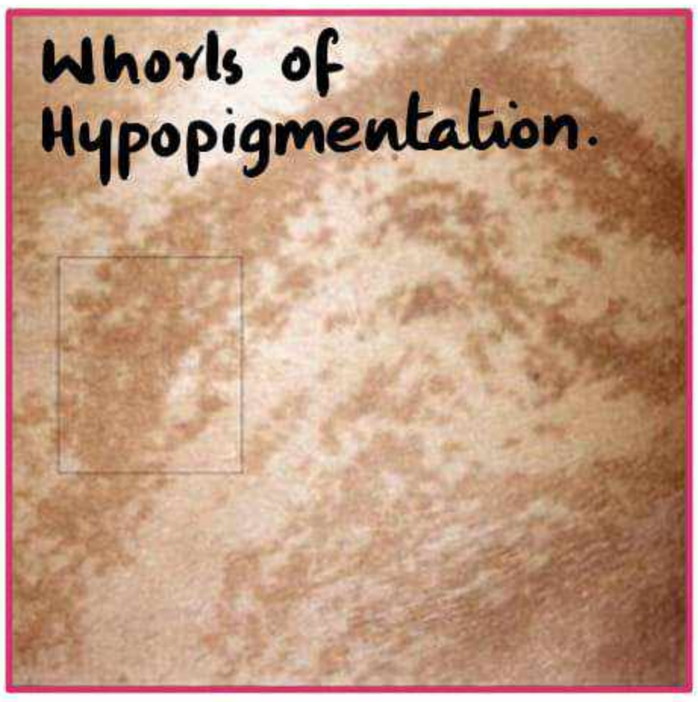
Clinical features

- Whorls of hypopigmented macules along the lines of Blaschko

Associated with:

i) Mental Retardation

ii) Epilepsy



VITILIGO

22:50

- Type: Autoimmune, Acquired, Progressive
- Defect: Melanocytopenia.

active space

- Family h/o : positive in 20-30%
- Association : other autoimmune disorders
  - Hashimoto's thyroiditis (MC)
  - Pernicious anemia.
  - DM
  - Grave's disease
  - Hypothyroidism disease.

Pathogenesis :

1. Genetic : defect in Catalase genes
2. Immune hypothesis : Immune cells which destroy melanocyte
3. Neural hypothesis : Neurotransmitters destroy melanocyte.
4. Autotoxic Self Destructive or Free Radical Hypothesis :
   - Intermediates in melanin synthesis → destroy melanocytes

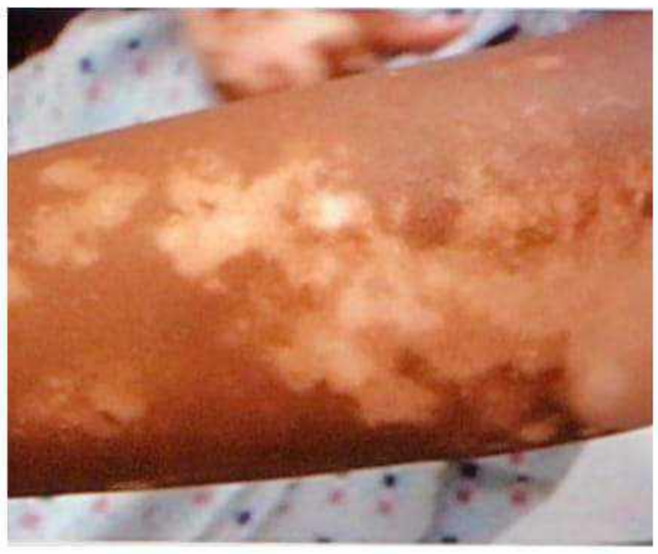
Clinical features :

- depigmented macules over any body part.
- chalky white
- Present over any bodypart.
- Concave or scalloped borders.

active space

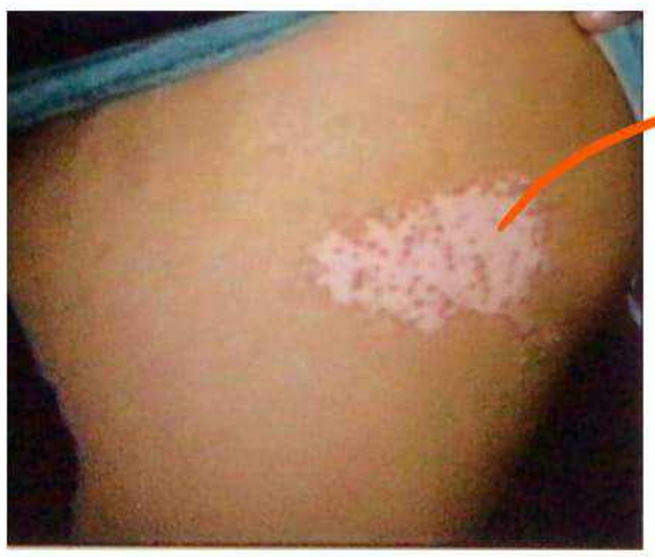
- Leukotrichia over patches
  - poorer prognosis.

- Types :

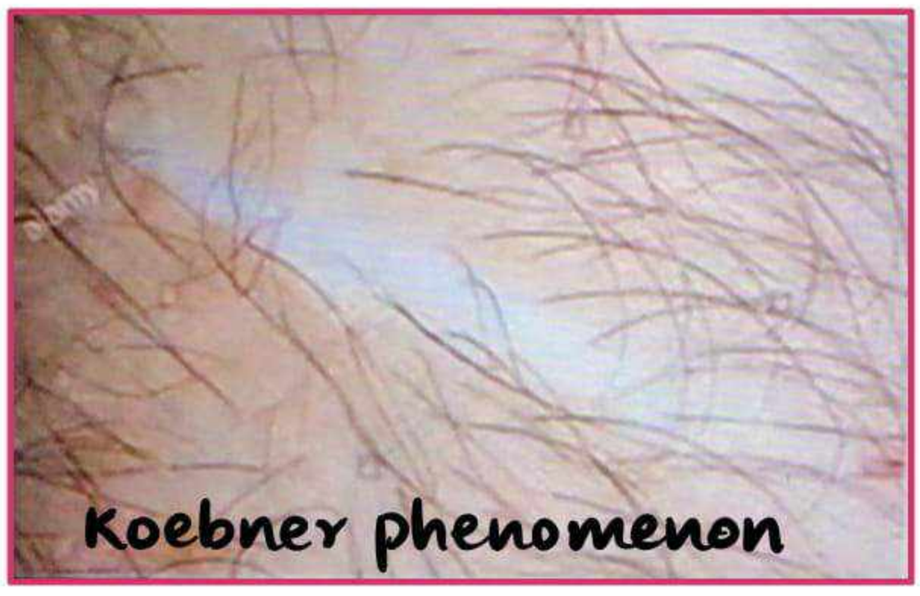


Trichrome vitiligo

- Depigmented zone
- Hypopigmented zone
- Depigmented zone



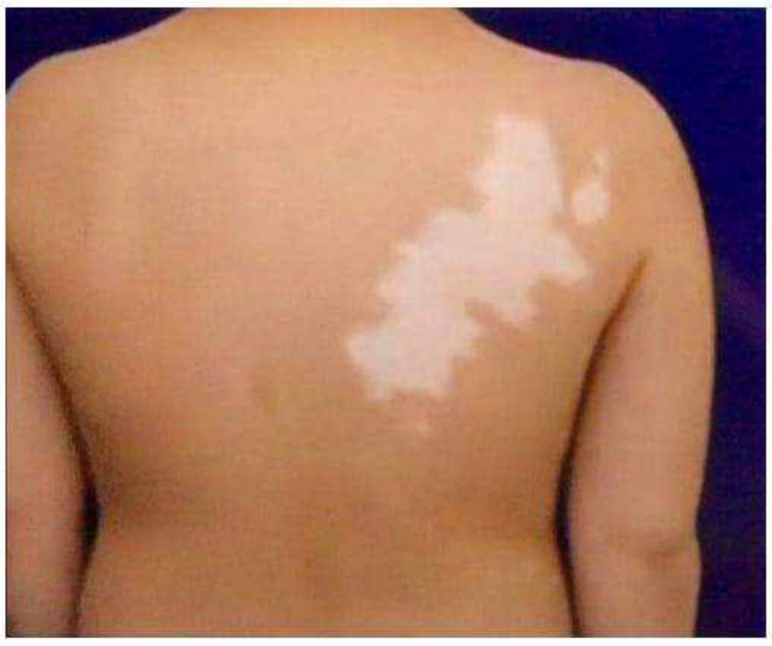
Perifollicular pigmentation
⇓
QUADRICHROME VITILIGO.



active space

Classification of Vitiligo

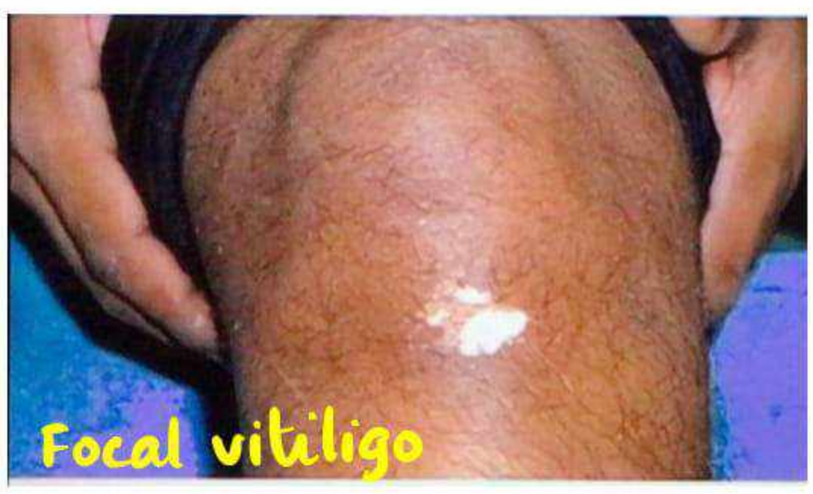
- Segmental
- Non-segmental
  - Localised
    - Focal
    - Mucosal
  - Generalised.
    - Vitiligo vulgaris
    - Acrofacial
    - Vililigo universalis

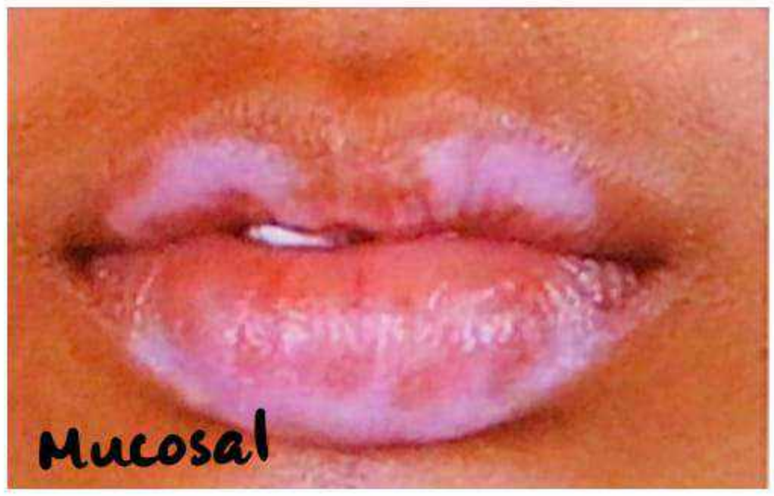


Segmental Vitiligo

- present at birth
- along a segment.
- Stable.
- Not respond to medical treatment.

Non-Segmental Vitiligo



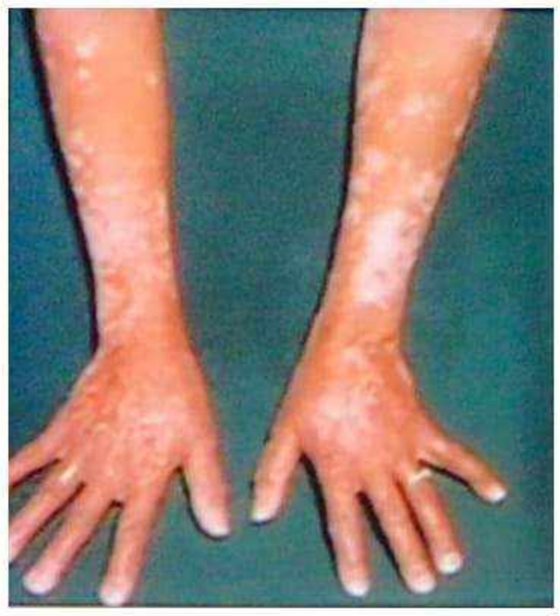


active space

Generalised Vitiligo

i) Vitiligo vulgaris. (MC)

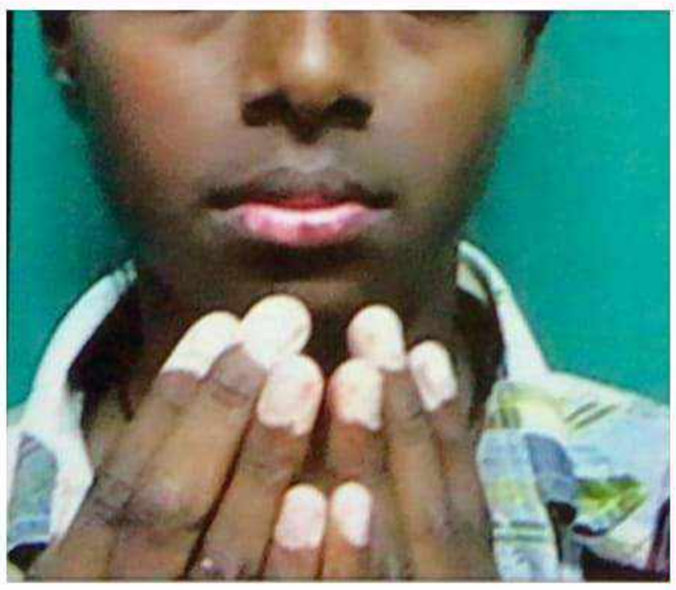
- B/L symmetrical lesions.



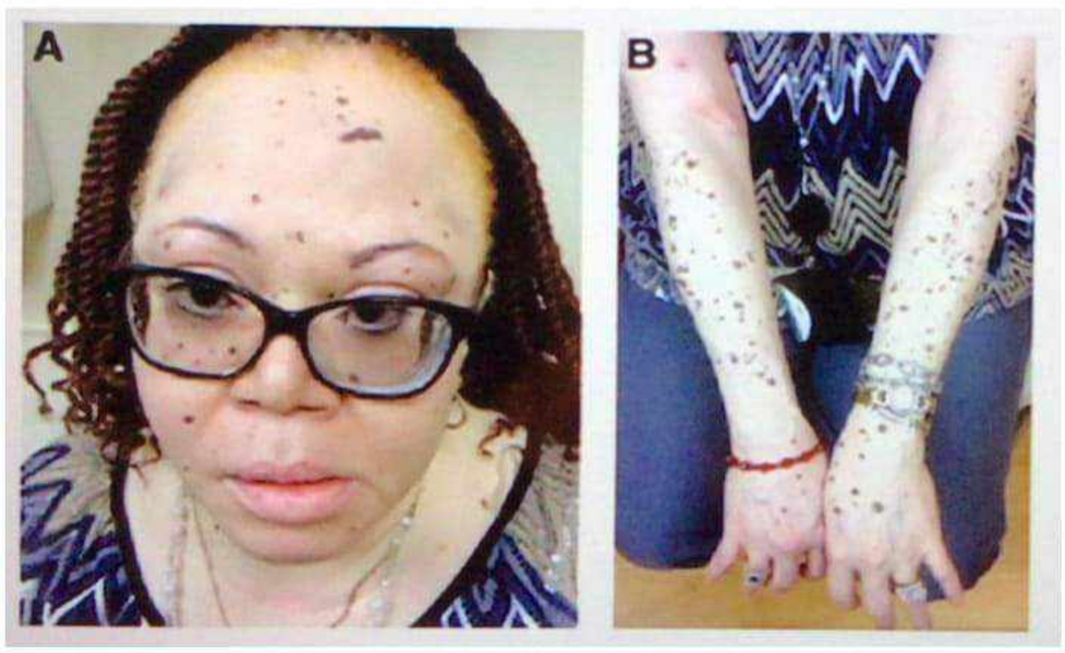
ii) Acrofacial vitiligo

↓

- involves extremities and face
- Poor prognostic marker.
- a.k.a Lip-Tip vitiligo



iii) Vitiligo universalis.



- when >80% of BSA is involved (depigmented)
- MBEH (Mono Benzyl Ether of Hydroquinone)

active space

- **Prognostic factors :**
  - Family h/o
  - Other autoimmune disorders
  - Leukotrichia
  - Acrofacial
  - Progressive disease
  - Generalised disease.

} Poor Prognostic Factors.

Management

- **General Aspects -**
  - avoid trauma
  - screen for other autoimmune disease.
- **Medical :**

Topical

- < 20 % BSA involved
- Topical Calcineurin inhibitors
  - Tacrolimus
  - used when face/genitalia is affected.
- Topical Psoralens

Systemic

- > 20 % BSA, Progressive
- Corticosteroids → Minipulse or Daily.
- Levamisole
- Azathioprine
- Cyclophosphamide
- Methotrexate.

active space

Phototherapy :

- NB UVB (Narrow Band UV B)
- PUVA
- Targeted Phototherapy
- Excimer LASER (308nm Xenon)

Psoralens

- can be used :

  i) Topically → PUVAsol (Kept for 10-15min → upto ½ hr)

  ii) Orally → Oral PUVA (Taken 2hrs before)

- Agents used :

  i) 5 Methoxy Psoralen
  ii) 8 Methoxy Psoralen
  iii) Tri Methoxy Psoralen

  → Phototherapy ↓ → Affect DNA → Melanising agents

Surgical Treatment •

- done only if vitiligo is stable → lesions are not progressing for 2yrs / 1yr.
- Refractory to medical treatment

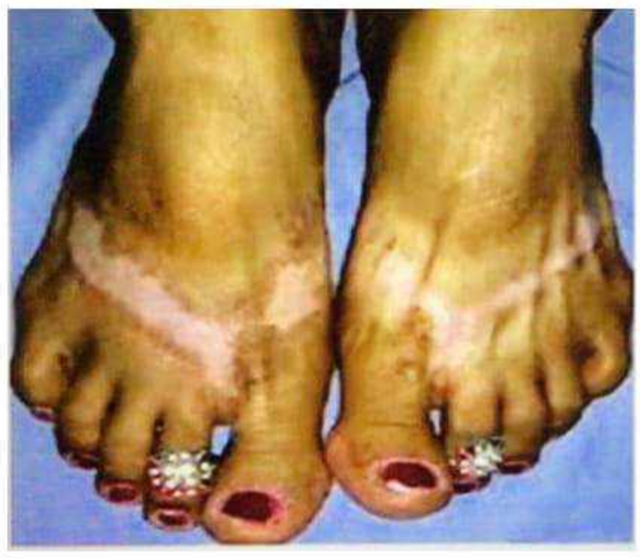
active space

- Camouflage
- Bleaching → done for universalis (MBEH)

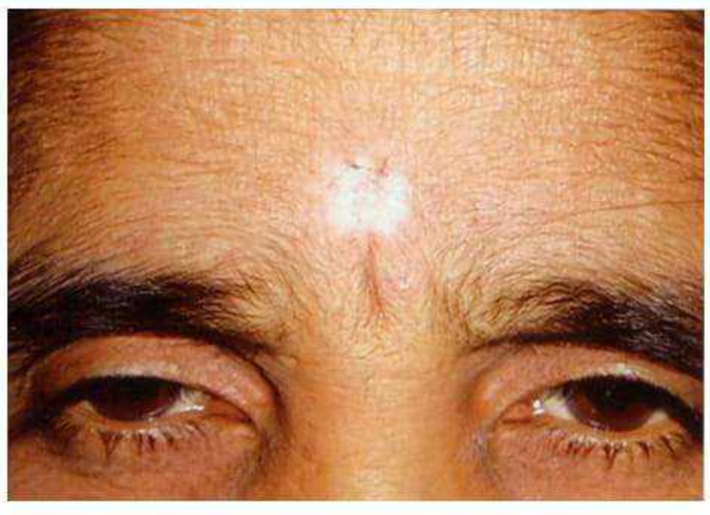
CHEMICAL LEUKODERMA

49:00

- a.k.a Contact leukoderma
- chemical is melanocytic



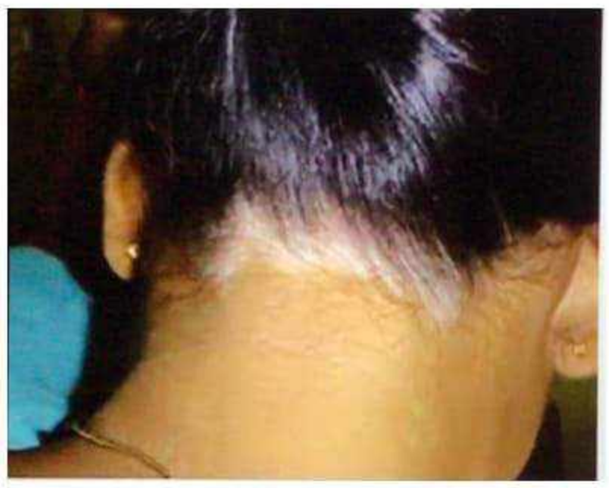
- Because of Rubber footwear
- There is MBEH.



Bindi dermatitis

- Agent : PTBP
- can cause
  - i) Bindi dermatitis
  - ii) leukoderma.

active space



- Use of Hair dye
- Agent :

  i) PPD

  ii) PTBC (PTB Catechol)

active space

Vesiculobullous Disorders (Part - 1)

- Patient presents as vesicles or bullae.
- Fluid filled lesions
- Vesicles → < 0.5cm size
- Bulla → > 0.5 cm size
- Crust → 2° lesions

Immunobullous disorders

- Bulla are formed due to immunological process.

↓

Antigen - Antibody complexes are formed

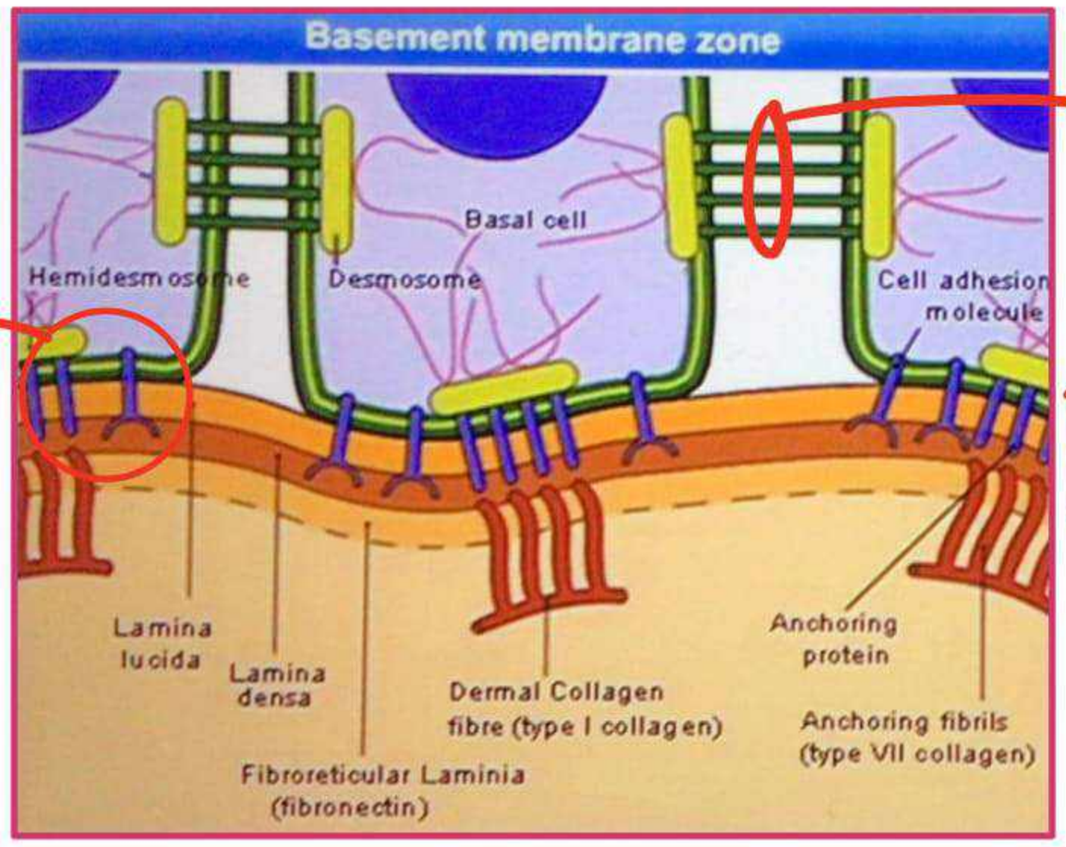
↓

Type III HSR

Immunobullous disorders Classification

- Intraepidermal : Pemphigous group
- Subepidermal : Pemphigoid group + Dermatitis herpetiformis

Subepidermal



Intraepidermal

BMZ or DEJ

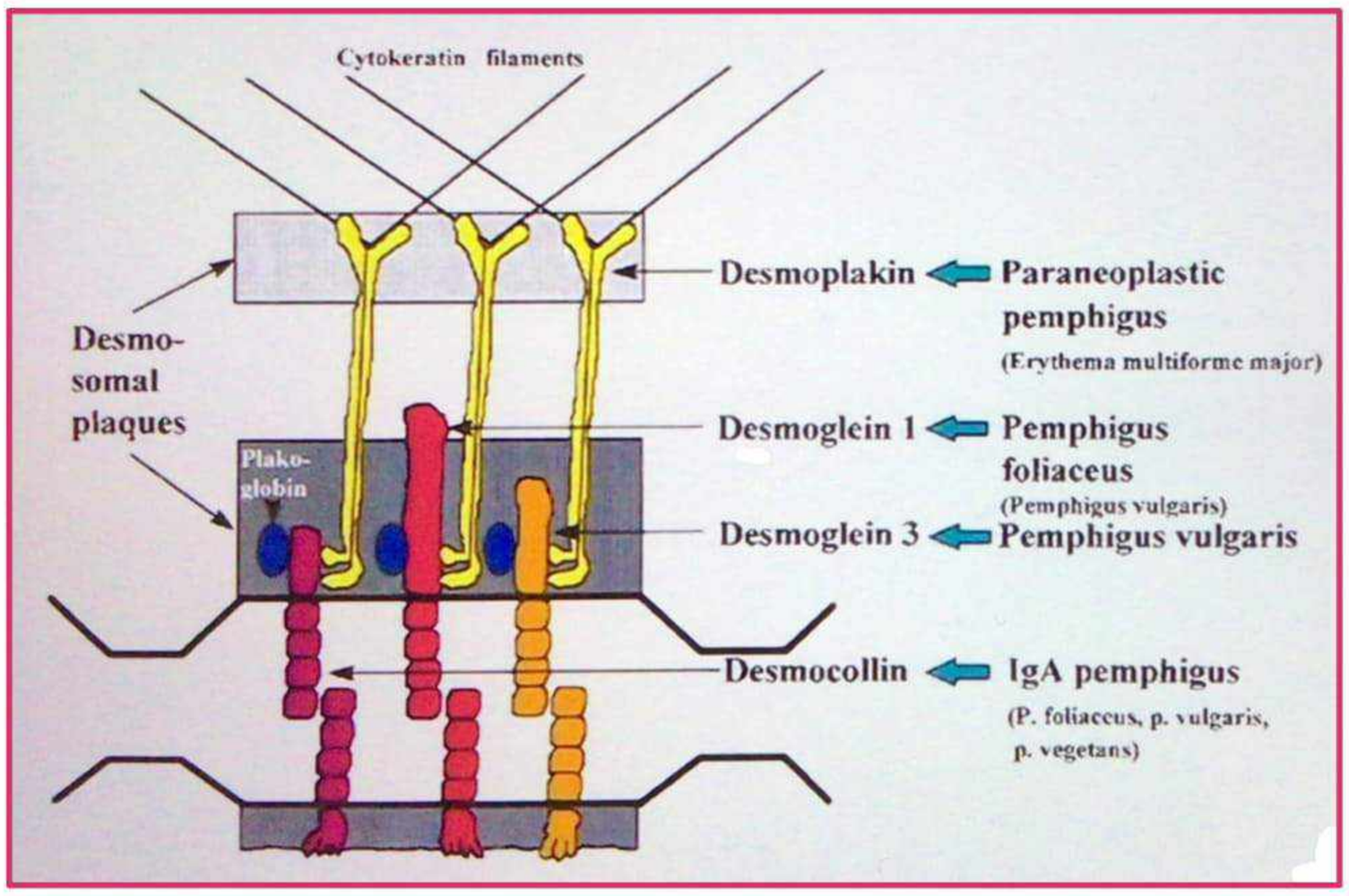
Intraepidermal group of disorders

- a.k.a Pemphigus group
- Target protein ⟶ Desmosomes
- Target Antigen ⟶ Desmogleins, Desmocolins, Desmoplakins
- Intraepidermal split.

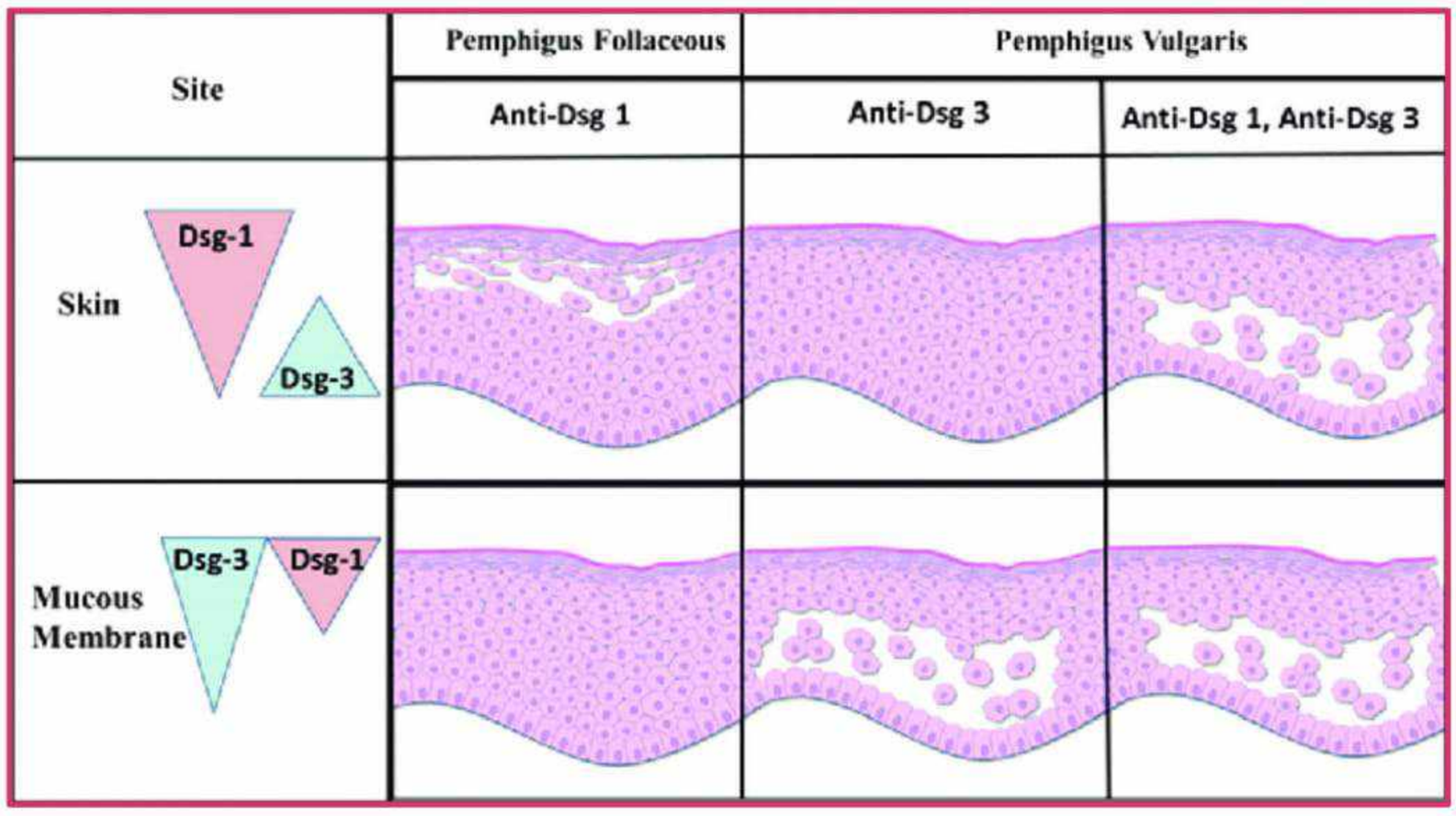
Pathogenesis :

Ab binds with Ag
↓
Ag - Ab complexes
↓
Weakens desmosomes
↓
Cell separation / Acantholytic

active space



Desmoglein 1 and 3



- Anti Dsg 1 Ab → No mucosal involvement
  Only upper layers of skin } Pemphigous foliaceous

active space

- Anti Dsg 3 → No skin involvement

  Mucosal involvement ⊕

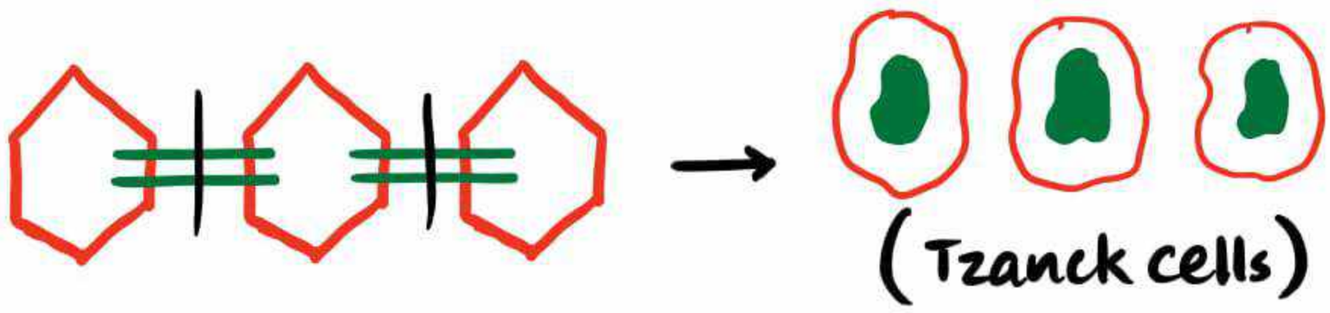
- Desmoglein Compensation theory.

Antibodies :

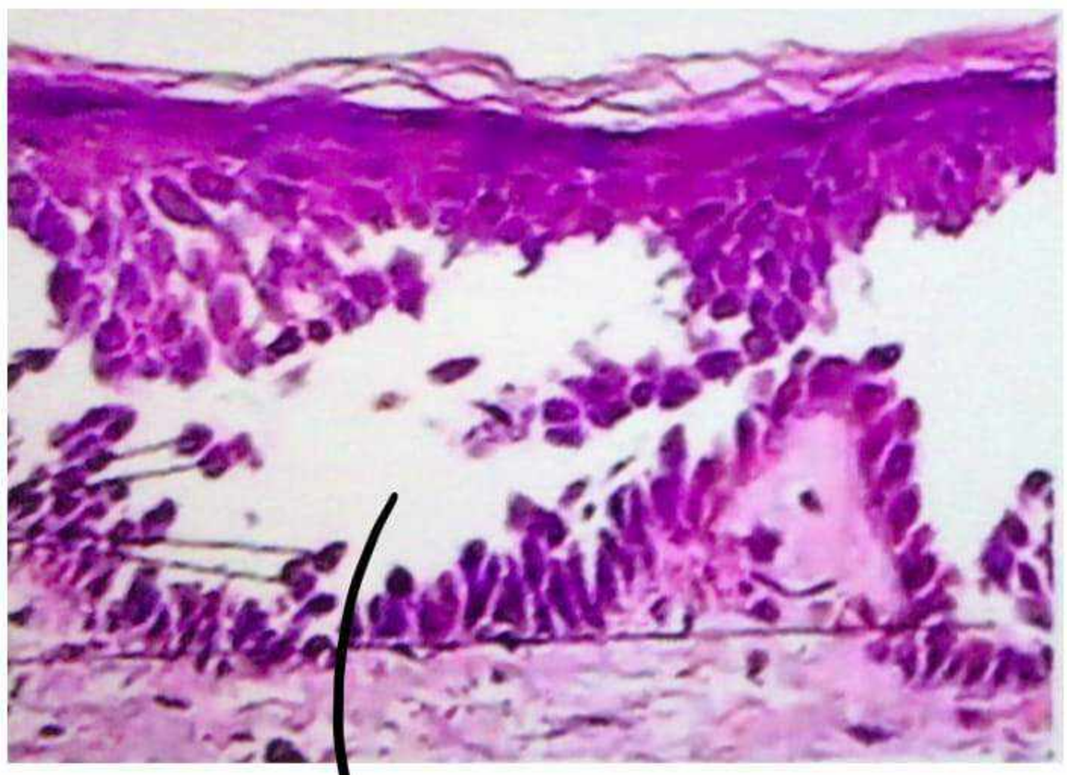
- IgG → IgG1 and IgG4
- Rarely : IgA, IgM, IgE

Acantholysis :

- separation of Keratinocytes

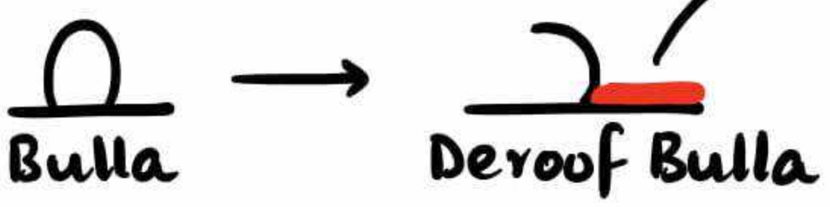


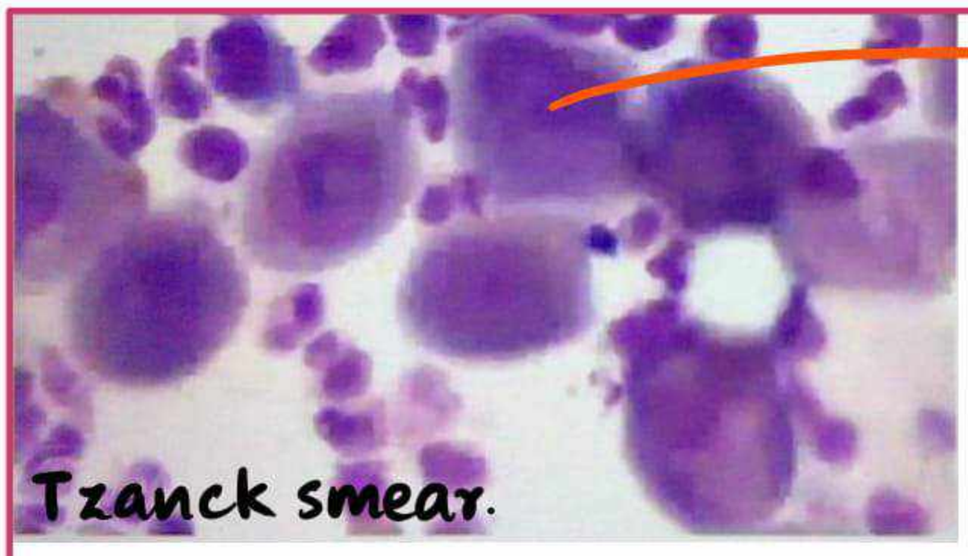
On HPE :



active space

Tzanck smear.




Tzanck smear.

→ **Tzanck cells / Acantholytic cells**

- round / oval in shape.
- nuclei occupying 7/8th of cell
- peripheral condensation of cytoplasm.

CLASSIFICATION OF PEMPHIGUS

26:00

1. Intra-epidermal / Pemphigus :

   i) P. vulgaris → P. vegetans. → Target Ag : Dsg 3 > Dsg 1

   ii) P. foliaceous
   - P. erythematosus
   - P. herpetiformis
   - Endemic pemphigus.

   } Target Ag : Dsg 1

   iii) Drug Induced Pemphigus

   iv) IgA Pemphigus → Target Ag : Desmocollin

active space

v) Paraneoplastic pemphigus. → Target Ag :

- Desmoplakin.
- Enviplakin.
- Periplakin
- Dsg

- Genetic associations : HLA association present
- Environmental : Smoking helps

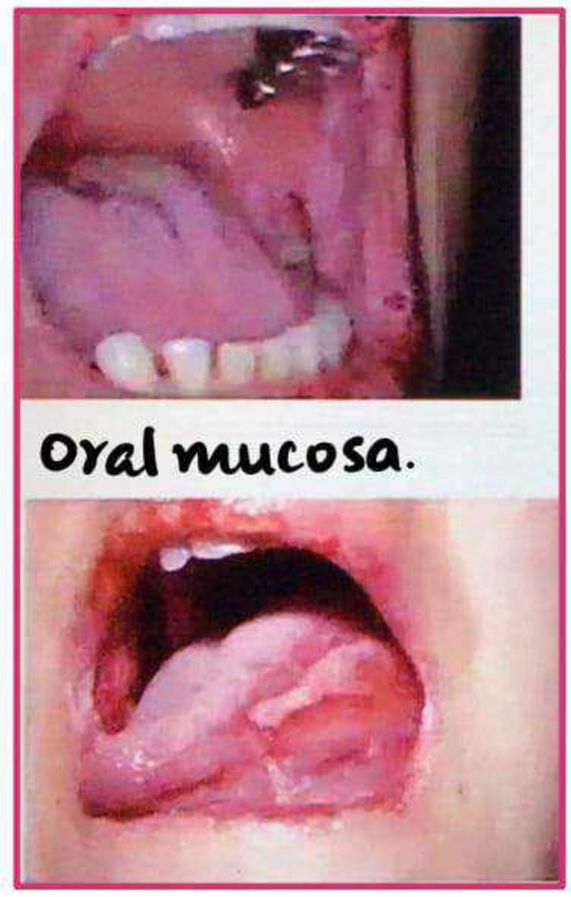
PEMPHIGUS VULGARIS 30:00

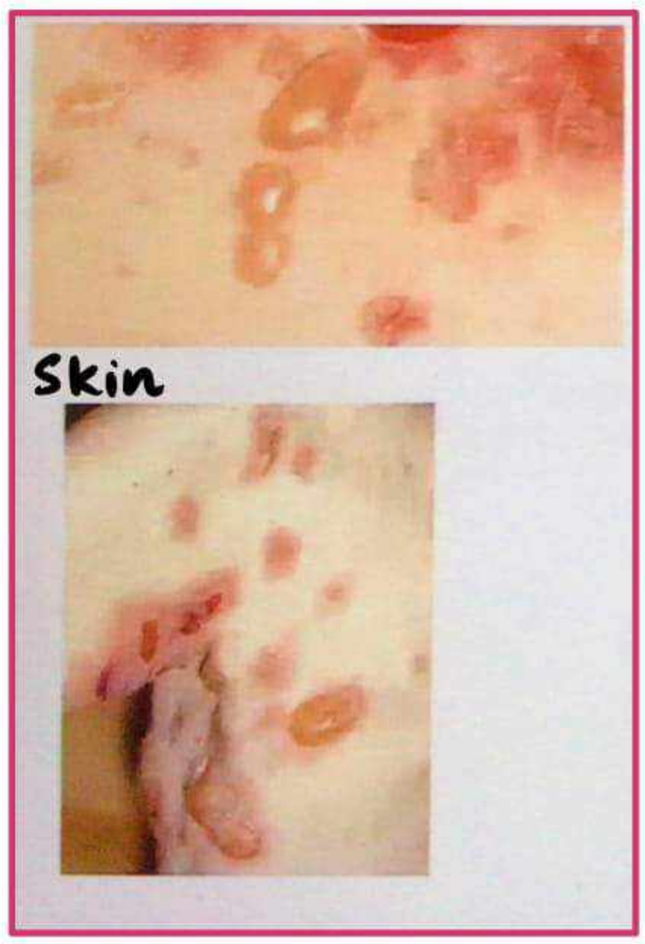
- Commonest
- Age : Middle Age (In India → Earlier age group)
- Ethinicity :
  - Ashkenazi Jews.
  - Mediterranean
  - Iranian.
- Associated with other autoimmune disorders.
- Target Structure : Desmosomes
- Target Antigen : Dsg 3 > Dsg 1
- Split : Lower layer → Suprabasal split (stratum spinosum)

active space

Clinical feature :

- involves mucosa and skin
- Mucosa involved → Oral, Perianal, Genital, Oesophagus.
- Skin involved → Head, Neck, Upper chest → all over body.
- 60 - 70 % → present with oral involvement
- Oral mucosa is involved in 100 % of Pemphigus vulgaris patients
- usually no intact bullae seen.
- Erosions →
  - Erythematous
  - Spreading periphery
  - No tendency to heal
  - Whitish exudate ⊕
  - Painful erosions
- In skin → Flaccid bulla and vesicles are seen.
  - Flaccid → wrinkles old grape


Oral mucosa.


Skin

active space

vesicle

↓

Bulla

↓

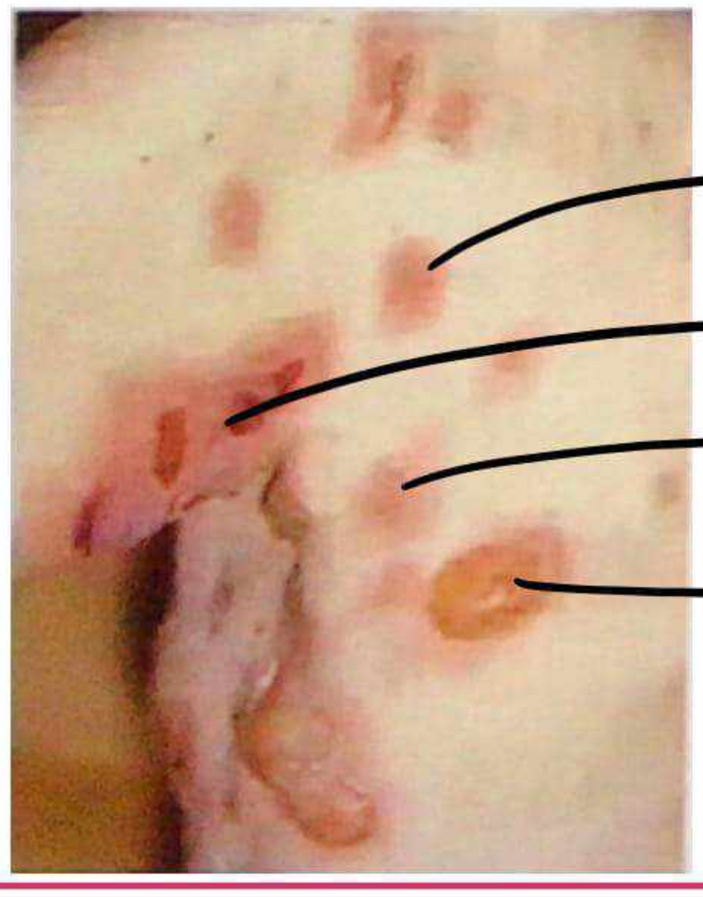
Ruptures in 2-3days

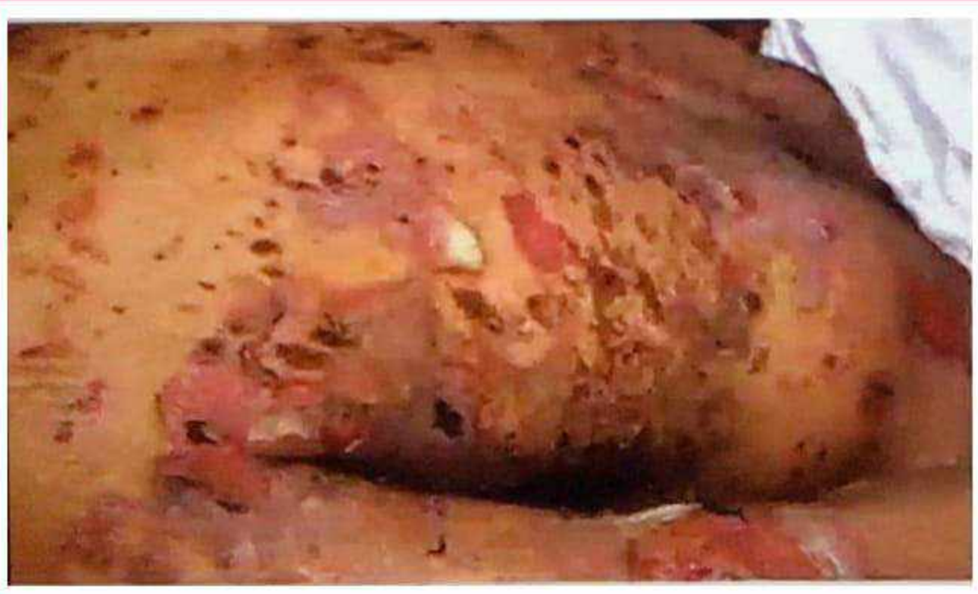
↓

Erythematous erosions which extends peripherally

↓

Healing without scarring





active space

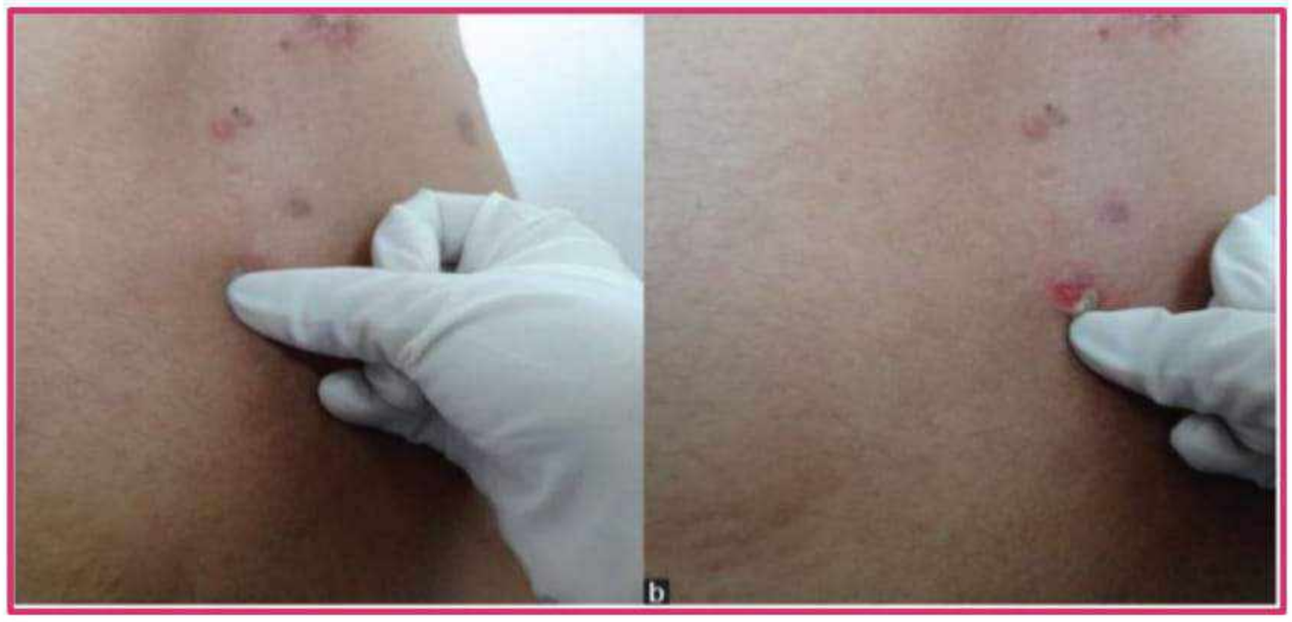
Signs Elicited :

1. Nikolsky sign :

- Tangential pressure on normal skin

↓

Peeling of skin / Bulla formation



- Tells about :

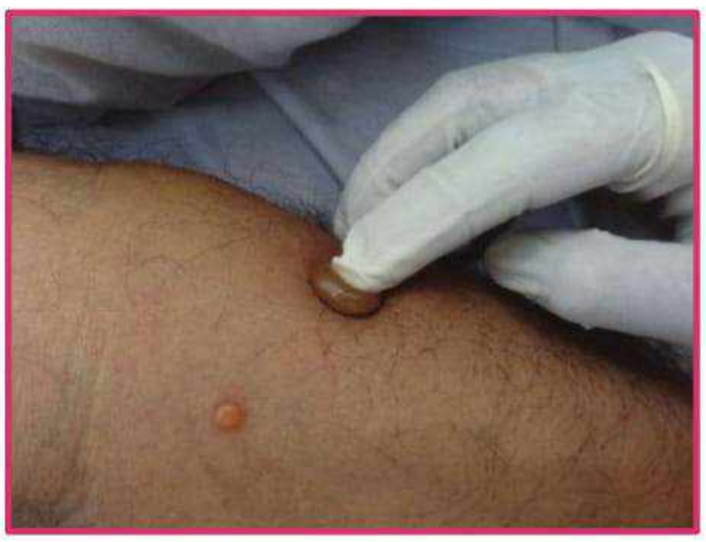
  i) Activity of disease

  ii) Severity disease.

  - Positive in Intraepidermal disorders.
  - Negative in Subepidermal conditions.

2. Bulla spread sign :

- Putting lateral pressure on the bulla
- Angulated spread
- In subepidermal disorders (Round)



active space

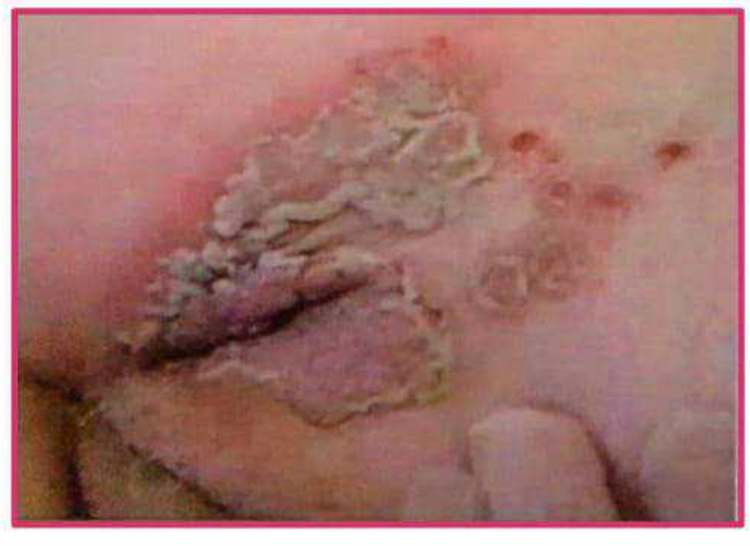
3. Asboe Hansen Sign

- done in small lesions
- vertical pressure is applied.

PEMPHIGUS VEGETANS

50:00

- very rare form.
- vegetating plaques over flexures
- cerebriform appearance of tongue.



- 2 Types :

  i) Hallopeau → mild

  ii) Neumann Type. → Severe.

PEMPHIGUS FOLIACEOUS

51 : 13

- Antigen : Dsg 1
- Subcorneal split (Stratum granulosum affected)
- Site : Seborrhoeic areas → upper chest, neck, face, scalp.
- Less severe than PV
- Mucosa : Not involved

active space

Clinical feature :

- vesicle and Bulla are formed.

↓

But they rupture in day

↓

Crusted erosions are seen

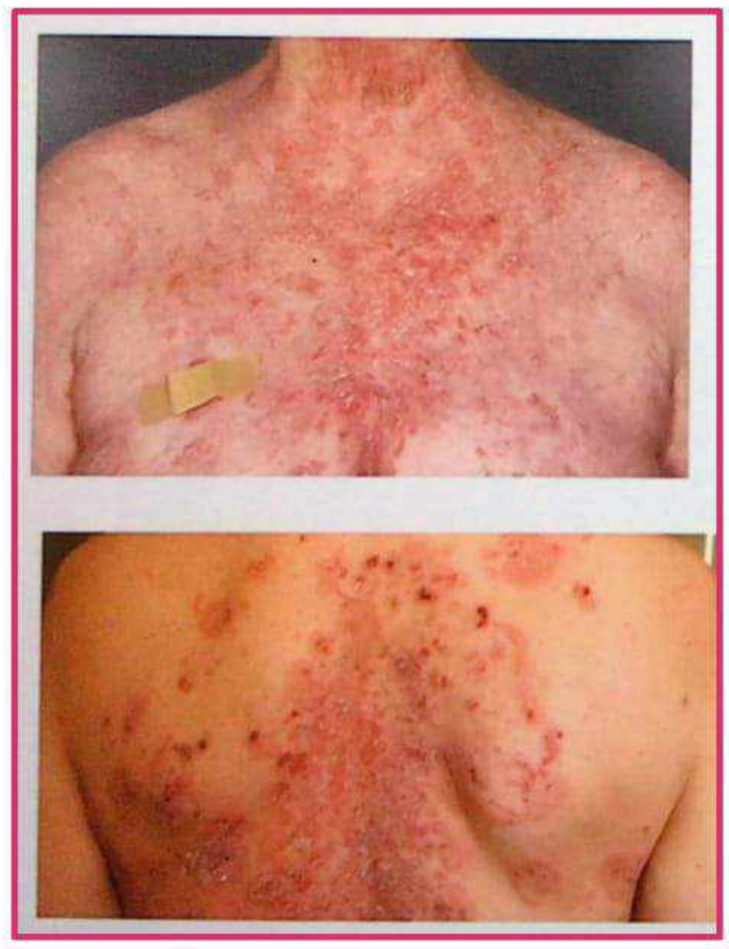
(crusting ↑↑ - Pv)

- Erosions have a peripheral scale.

↓

Heal without scarring

- Hardly any bulla is seen.



	Pemphigus Vulgaris	Pemphigus Foliaceous
Antigen :	Dsg 3 > Dsg 1	Dsg 1
Split :	Suprabasal	Subcorneal
Mucosa :	Mucosa involved	Mucosa not involved.
Skin :	Flaccid Blisters erosions	Crusted erosions ⊕

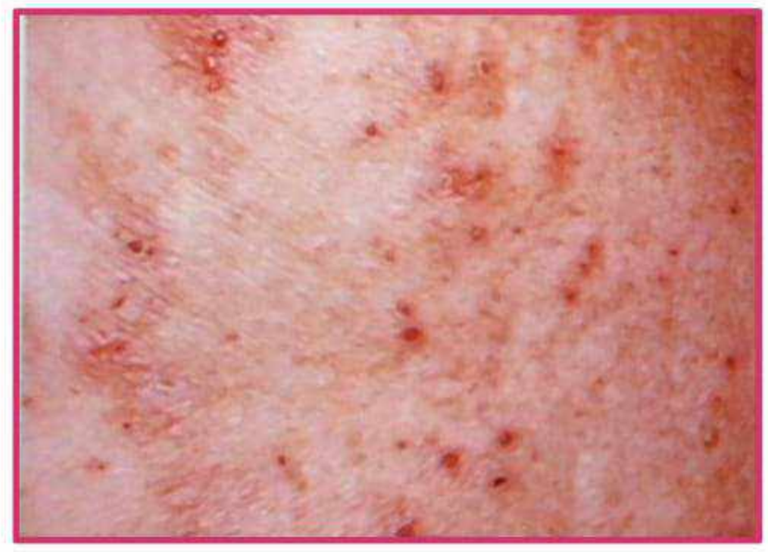
active space

Variants of Pemphigous foliaceous :

1. Pemphigus herpetiformis (PH)

(PF + DH ⟶ PH)

- itchy excoriated papules and vesicles all over the body.



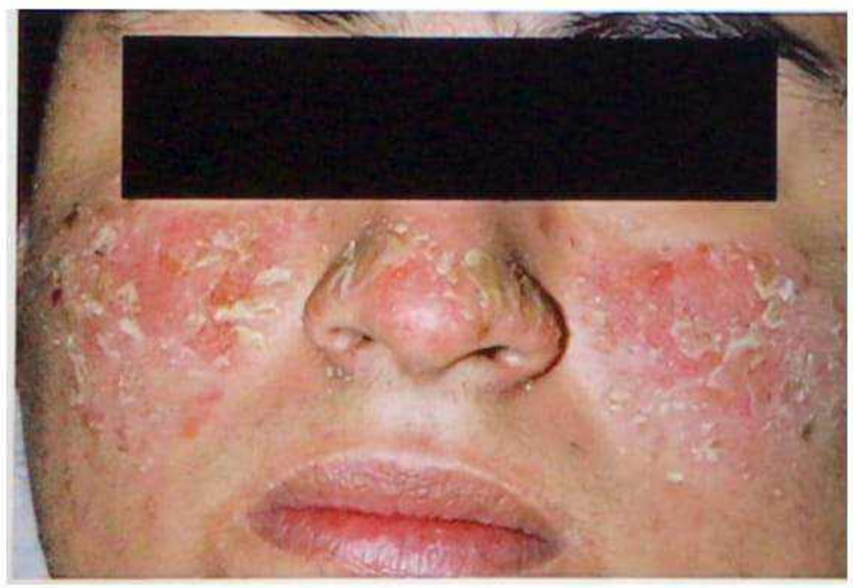
On Biopsy : Features of DH

On DIF : Intercellular deposits like PF

2. Pemphigus erythematosus :

(PF + LE ⟶ PE)

- lesions : Butterfly distribution
  ↓
  involves Malar area of face.
- photosensitivity
- ANA ⊕



On DIF : PF + LE

3. Endemic pemphigus

- Endemic to rural areas of Brazil and Tunisia.
- More common in children

- Site : Head and Neck
- Wild fire / Fogo selvegm : Burning sensation.
- Involvement of Black fly (Simmidae)
- Its a variant of PF

DRUG INDUCED PEMPHIGUS

01:05:48

- more of PF than PV
- less mucosal involvement
- Drugs caused :
  - i) Thiol group → Penicillamine
  - ii) Non-Thiol → ACE inhibitors (captopril)
  - iii) Phenol.

PARANEOPLASTIC PEMPHIGUS

01:07:07

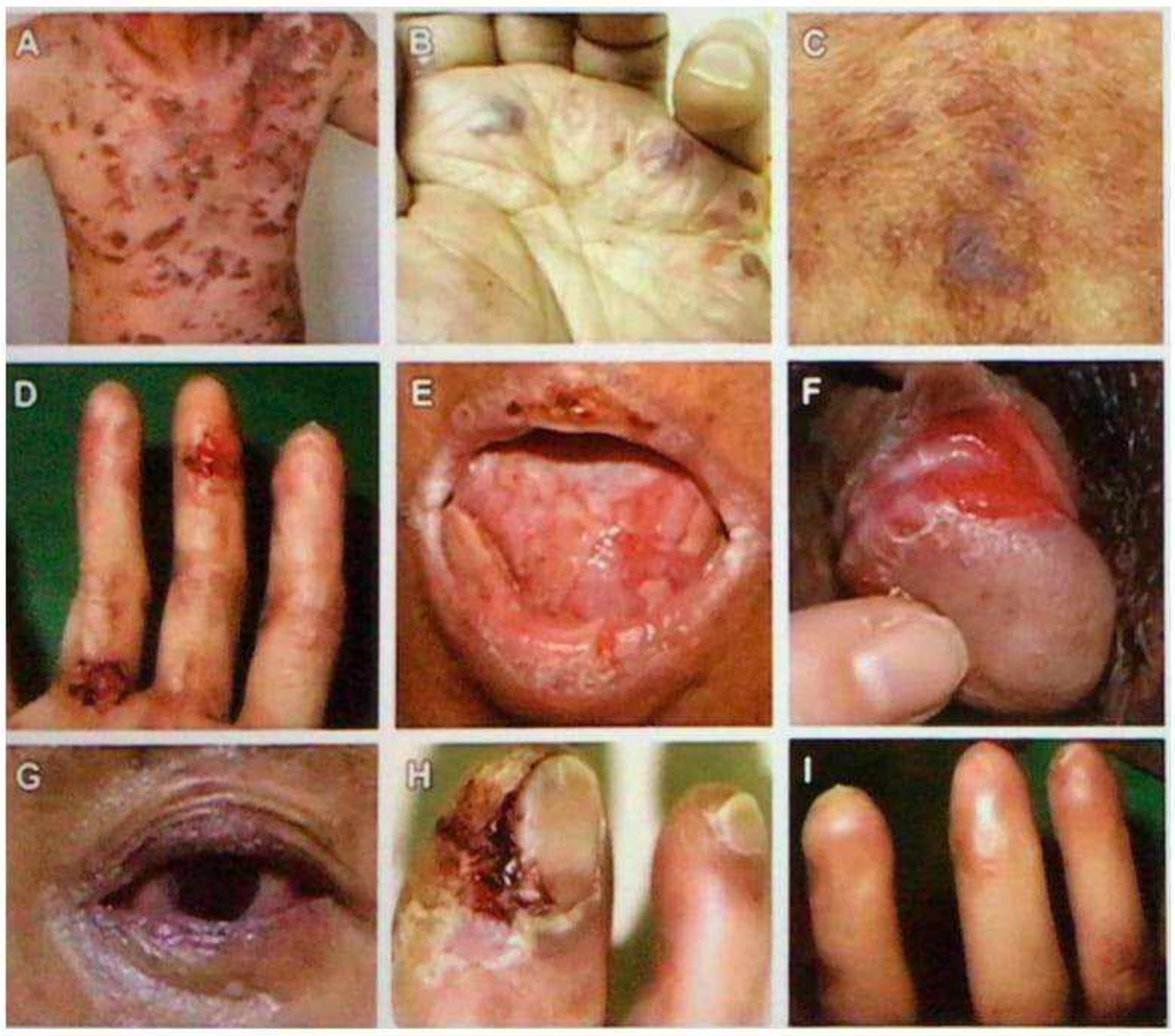
- Antigen : Desmoplakin (others - Dsg 1,3)
- Association :
  - i) Non-Hodgkins lymphoma.
  - ii) Thymoma
  - iii) Castleman's disease
  - iv) Waldenstorms Aglobinemia.

active space

- Criteria : Anhalt's criteria

Clinical features :

- Polymorphic
- Features of severe pemphigus + Lichenoids.



↓

- severe mucosal involvement → stomatitis, conjunctival involvement, genital mucosal involvement, perianal involvement, Oesophageal involvement

↓

Severe scarring seen.

- Skin → Polymorphia (Blisters, erosions, lichenoid lesions)
- On Biopsy : Pemphigus

  Basement membrane degeneration.
- On DIF : Fluorescence ⊕

IgA PEMPHIGUS

01:12:50

- Antigen : Desmocollin
- Antibody : IgA

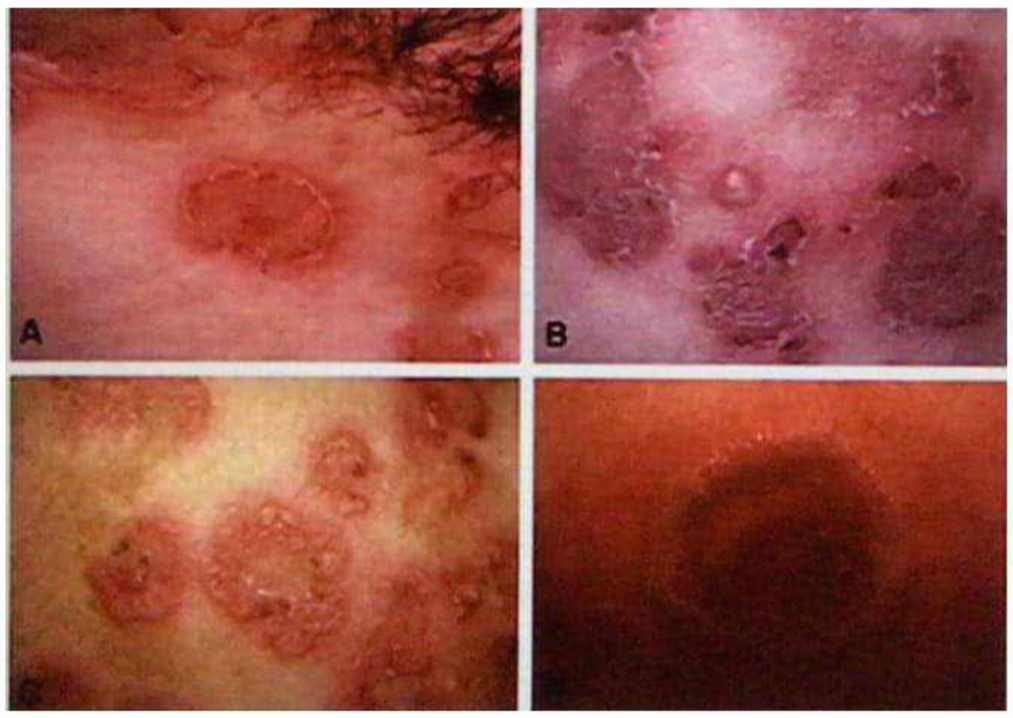
active space

- Types : i) Intraepidermal type

ii) Neutrophilic type.

Clinical features :

- Crusted erosions which are
  - Circinate
  - Annular
  - Itchy



Diagnosis of Pemphigus :

1. On Tzanck smear : Acantholytic cells / Tzanck cells.

2. On HPE :

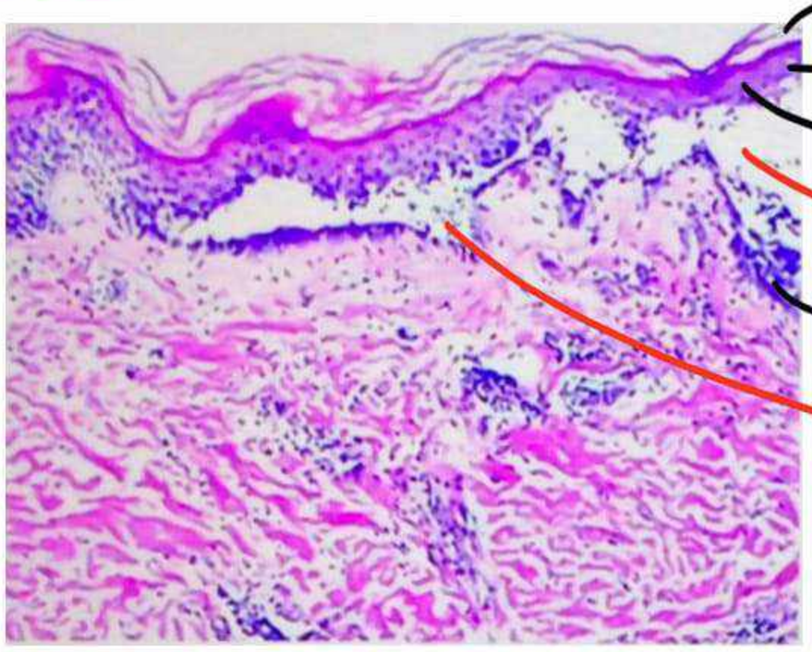
- 2 specimens are taken
  - → Lesional → for Hand E
  - → Perilesional → DIF (Direct Immunofluorescence)

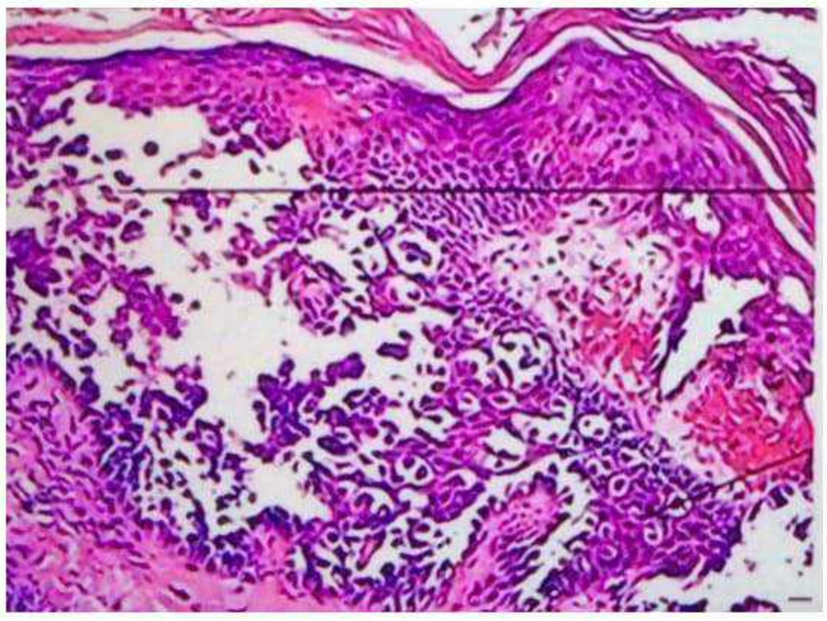
↓

used to demonstrate Ag-Ab complexes.

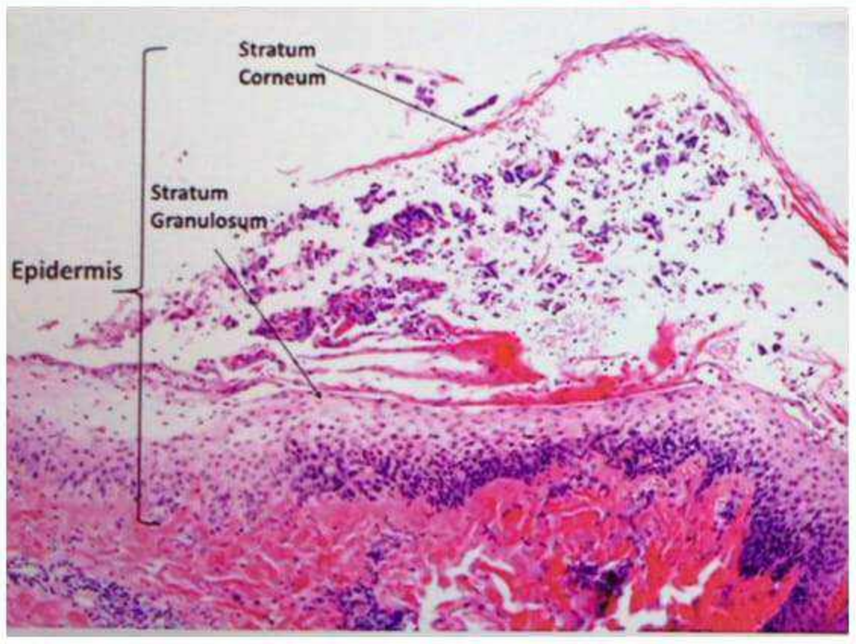
active space

On HPE :





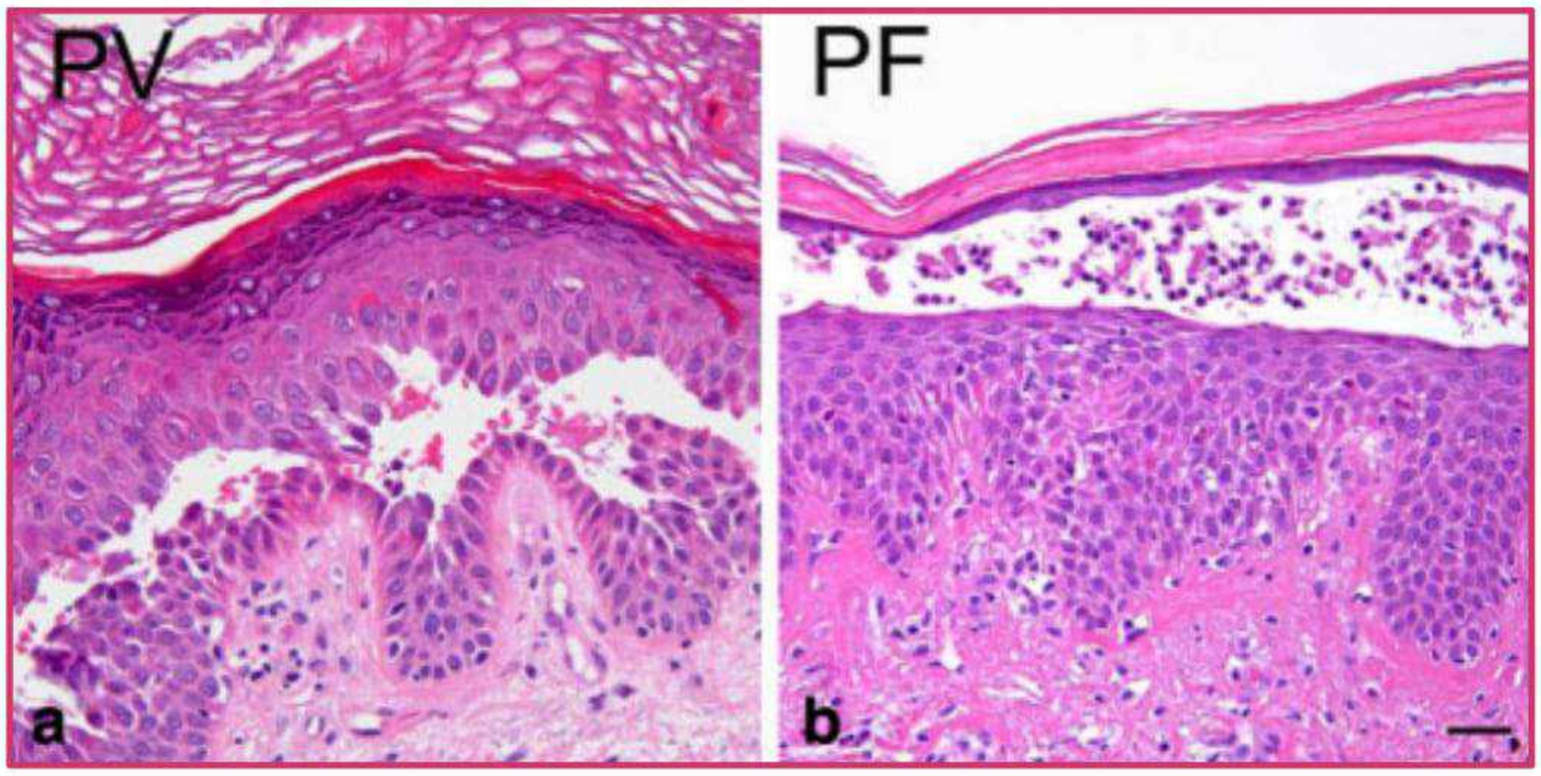
→ Pemphigus vulgaris.



Pemphigus foliaceous

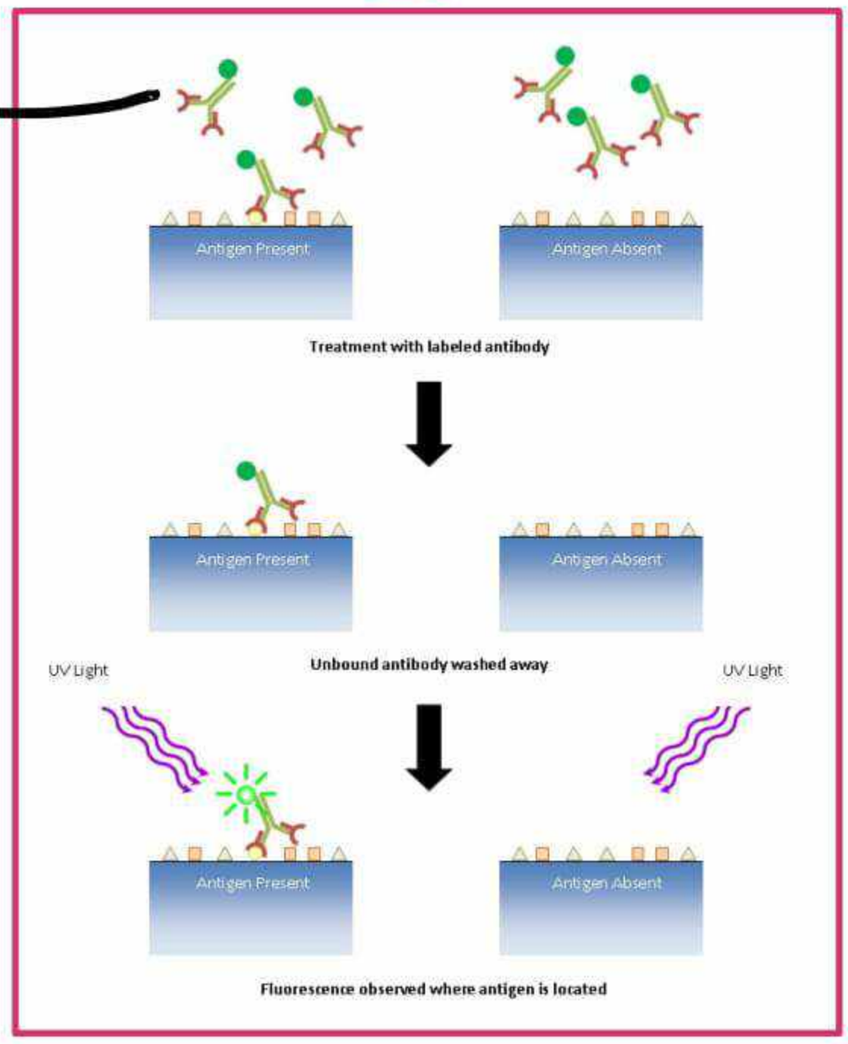
- split → Subcorneal

active space



Direct Immunofluorescence: → (IOC) most diagnostic test for pemphigus.

Fluorescent labelled Antibody.



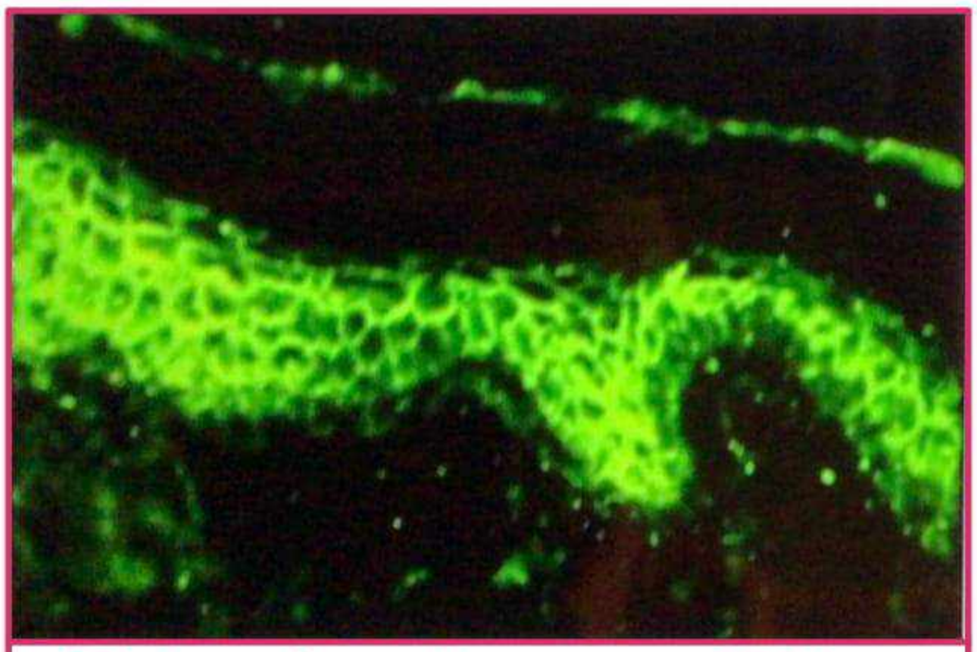
- can be used to

  i) Identify type of Antibody.

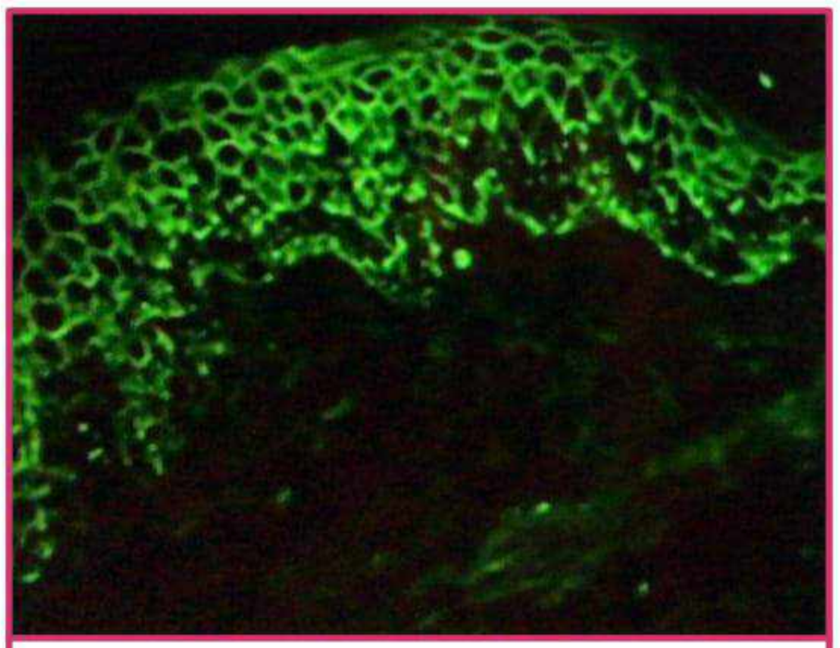
  ii) Extent of disease

active space

iii) Marker of Disease activity.



Fish net pattern on DIF
Pemphigus vulgaris



Pemphigus foliaceous

- Indirect Immunofluorescence
- ELISA

active space

Vesiculobullous Disorders (Part - 2)

Treatment of Pemphigus

i) Topical

- used in case of localised diseased.
- Topical corticosteroids

ii) Systemic

- Steroids
- Immunosuppressants
  - i) Azathioprine
  - ii) Mycophenolate mofetil
  - iii) Cyclophosphamide.
- Biologicals

iii) Adjunctive :

- Prevent 2° infection
- Crusting
- Fluid management.

- TOC → Steroids + Adjuvants.

Corticosteroids : TOC

- Topical
- Systemic : • 2-3mg/Kg/Day ⟶ Pemphigus vulgaris
  • 1-2 mg/Kg/Day ⟶ Pemphigus foliaceous
  ↓
  Taper gradually
- Pulse : High doses of drug for limited time
  i) Methyl prednisolone
  ii) DCP → Dexamethasone, Cyclophosphamide
  (High dose of C.S for 3 consecutive days in a month)

Adjuvants used :

	Azathioprine	Mycophenolate Mofetil	Cyclophosphamide.
MOA :	inhibit denovo synthesis	inhibit IMP dehydrogenase	Alkylating agents crosslinking of DNA.
Dose	1-3 mg/day	2-3g/day	1-2 mg/kg/day.
Side Effects	BM suppression Liver toxicity	BM suppression Nausea, Vomiting	Gonadal failure Hemorrhagic cystitis

active space

	Azathioprine	Mycophenolate Mofetil	Cyclophosphamide.
Side Effects	Monitor TPMT levels		Bladder cancer (Acrolein) ↓ prevented by : i) fluids ii) MESNA

- Other drugs :

  i) Gold

  ii) Dapsone

  iii) Methotrexate.

Cyclosporine not used

Rituximab

- Biological.
- Anti CD-20 Monoclonal Antibody.
- Patients refractory to conventional treatment - Steroids and adjuvants.
- Given in 2 protocols → Lymphoma
  → RA Protocol.

active space

Other Therapies:

i) i.v Ig

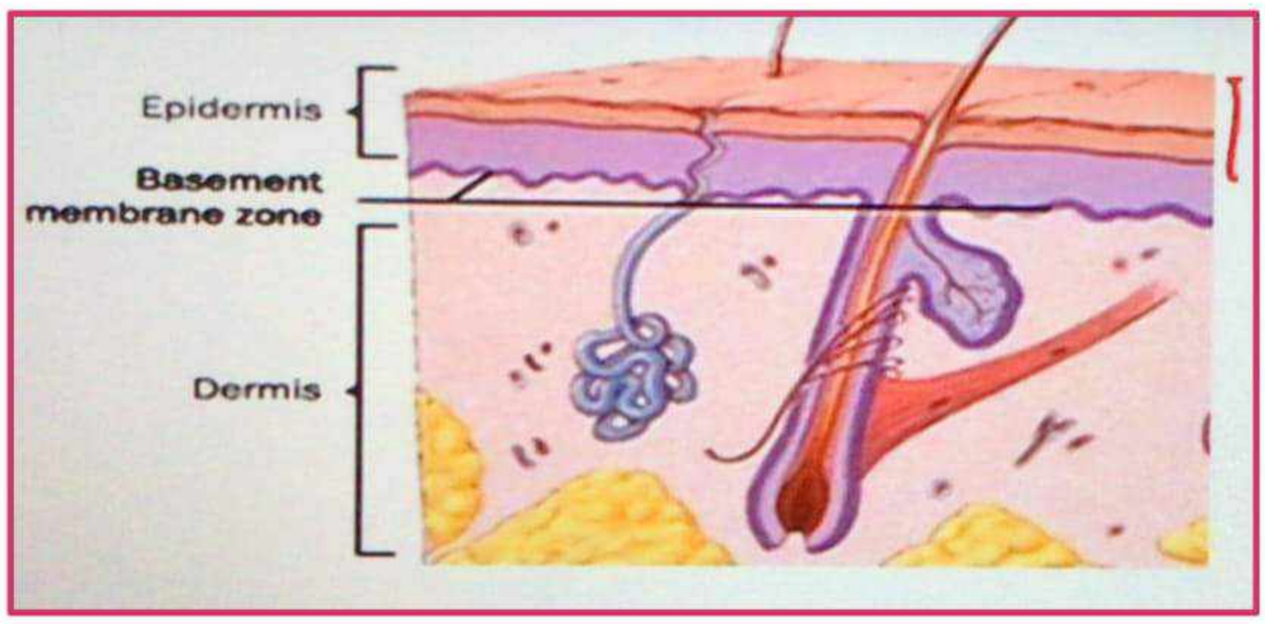
ii) Immunoadsorption

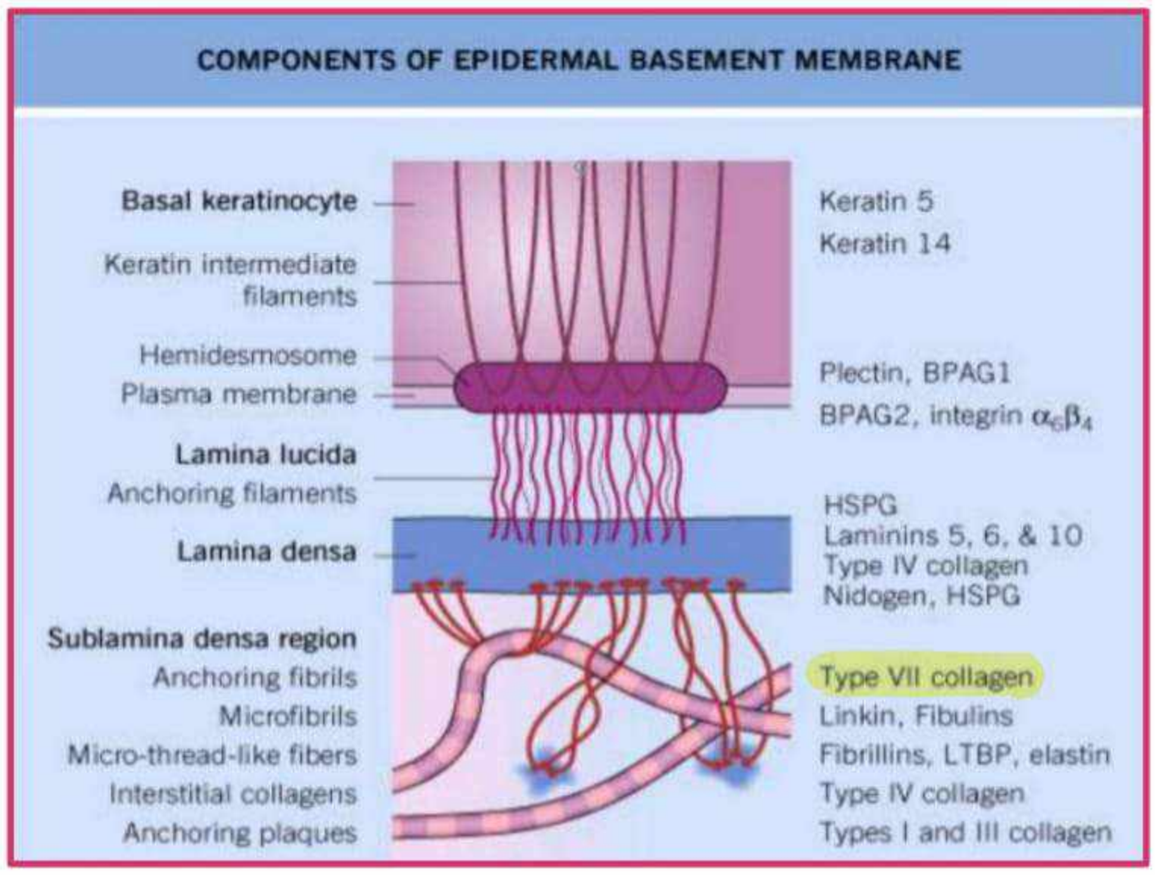
iii) Plasmapharesis

SUBEPIDERMAL DISORDERS

13:20

- split in the Basement Membrane Zone.
- a.k.a Pemphigoid + Dermatitis Herpetiformis.





active space

- In Hemidesmosomes:
  - $BPAG_1 \rightarrow$ 230 kDa
  - $BPAG_2 \rightarrow$ 180 kDa.
- Lamina lucida:
- Lamina densa:
- Sublamina densa
- Anchoring fibrils
- Collagen plaque.

Pathogenesis:

Ag - Ab complex forms
↓
Complement activation
↓
Recruitment of Inflammatory mediators.
↓
Lysis and separation

Classification and Antigen

- Bullous pemphigoid. - BP 180 (BP Ag 2), BP 230 (BP Ag 1)
- Mucous membrane pemphigoid - BP 180 > BP 230
- Cicatricial pemphigoid. - BP 180 > BP 230

active space

- Linear IgA disease - LAD_1
- Herpes gestationis - BP 180
- Dermatitis herpetiformis - Transglutaminase
- Bullous SLE
- Epidermolysis Bullosa Acquisita. } Collagen VII

BULLOUS PEMPHIGOID

23:20

- Antigen: BP180, BP230
- Antigen: IgG → IgG1 and 4

Pathogenesis:

Circulating Anti-BP 180 and Anti-BP 230 Ab bind to target Antigens within the DEJ.

↓

Activate the complement pathway and release chemoattractants.

↓

Recruitment of Eosinophils and Neutrophils

↓

Release destructive proteolytic enzymes

↓

BMZ separates

↓

Sub-Epidermal blister is formed

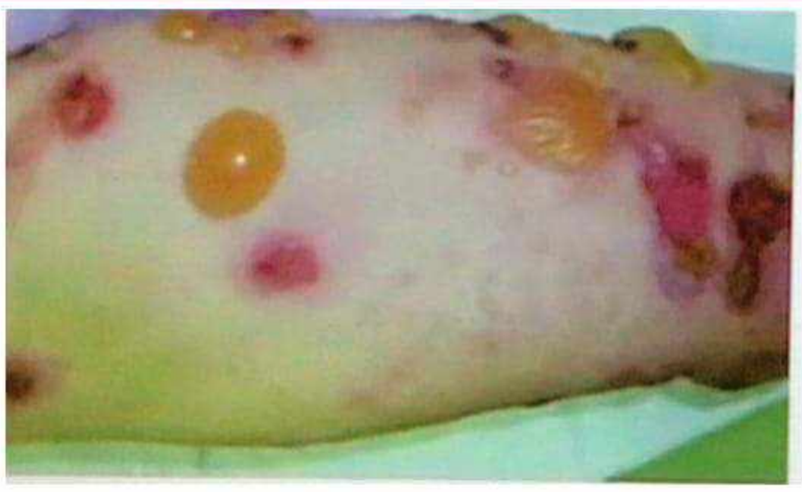
active space

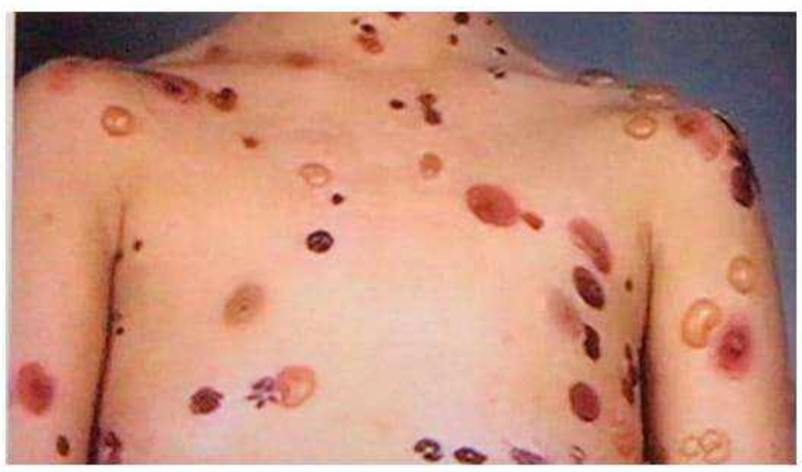
- **Age :** Elderly (60-80 yrs)
- **Association :**
  - i) Neurological disorders
  - ii) Autoimmune disorders
- **Triggers :**
  - i) Trauma
  - ii) Friction
  - iii) Drugs.
- **Site :**
  - Flexural areas (Trunk)
  - Mucosal involvement rare

Clinical feature :

- 2 Stages → Pre-Bullous (Prodromal)
  - → Bullous
    - ↓
    - Tense bulla

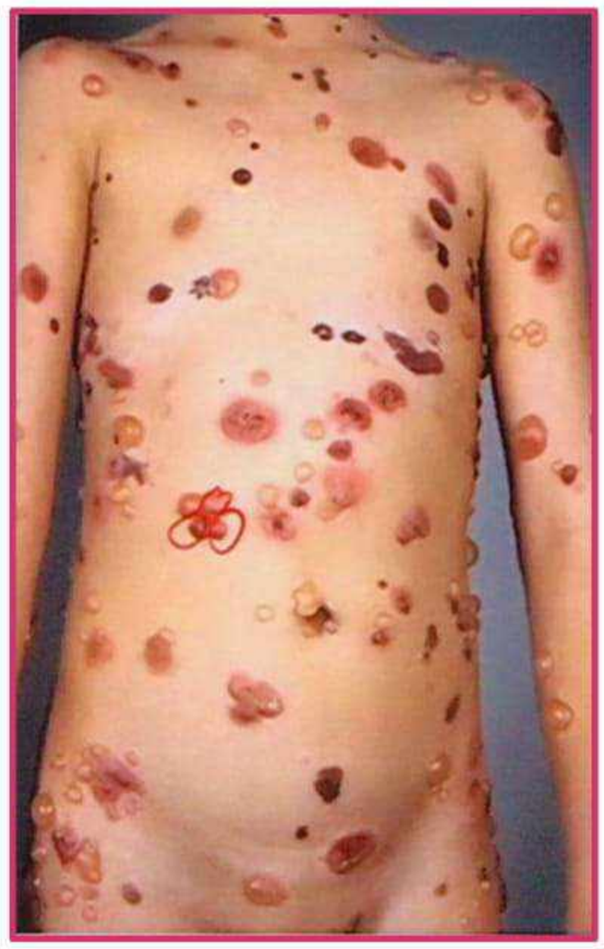
- ↓
- Itching
- Urticarial lesions
- Excoriations
- Eczematous.





active space

- Patient presents with tense bulla which donot rupture easily.
  Surrounding erythematous base / urticaria



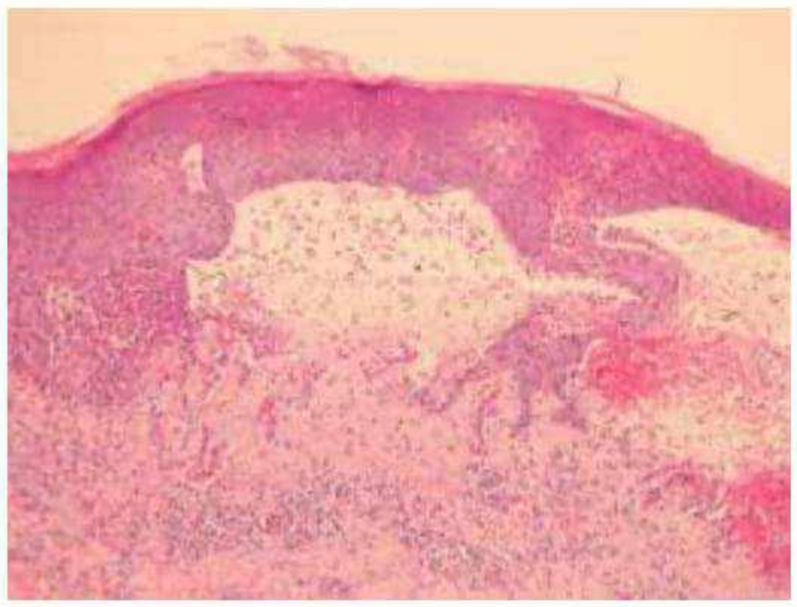
- Itching ⊕
- Hemorrhagic bulla may be present.
- Erosions covered with hemorrhagic crusts.

↓

Heal without scarring.

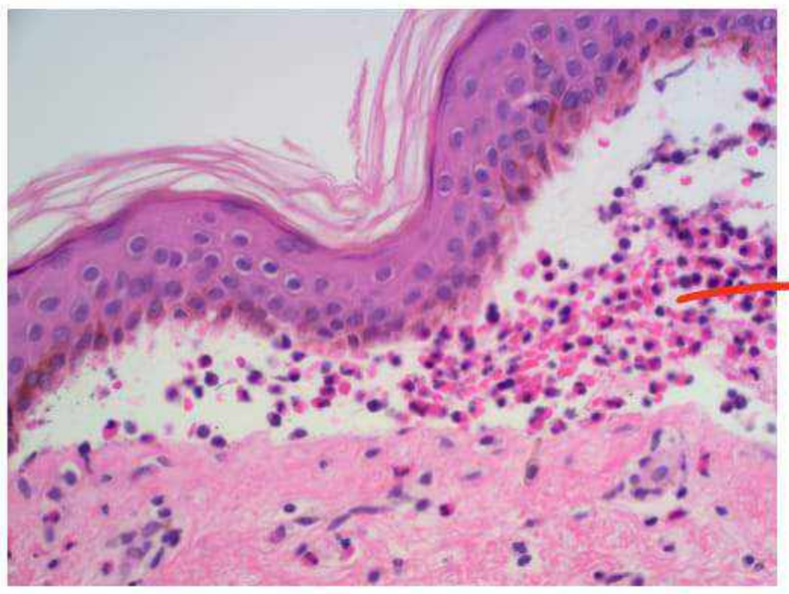
- Mucosa: not involved
- Nikolsky sign : Negative
- Bulla Spread sign : Positive, Bulla spreads uniformly.
  (Rounded spread)
- Tzanck smear : - Acantholytic cells not seen
  - Inflammatory mediators are seen.
    ↳ Eosinophils, Neutrophils.

On HPE :



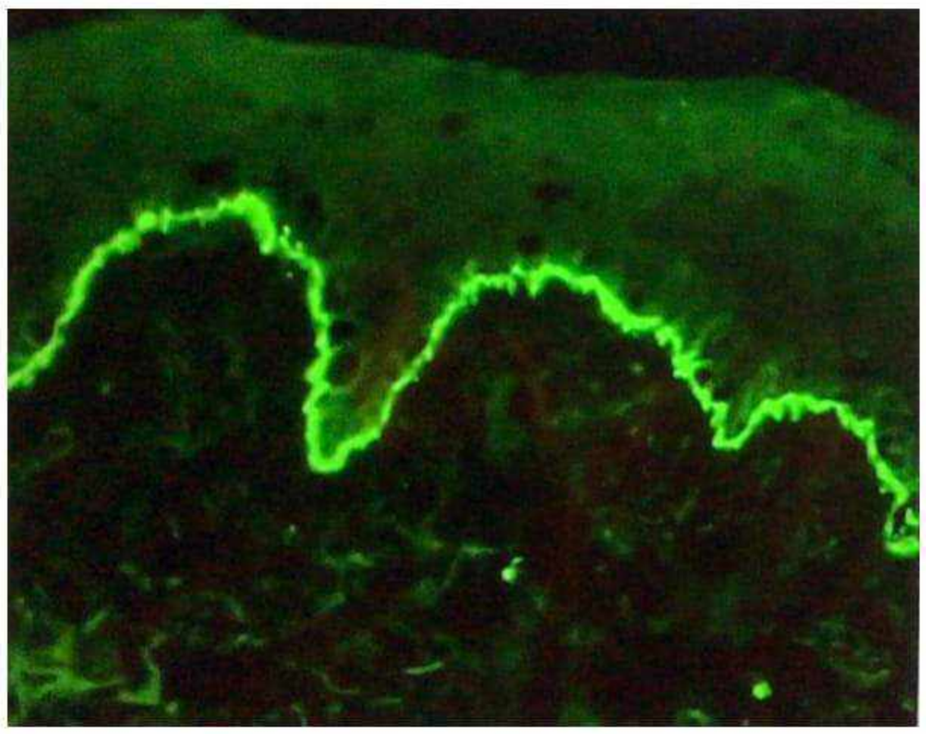
- Subepidermal split.

active space



→ Eosinophils (↑) + Neutrophils

On DIF :



- Antibody → IgG and C_3.
- Linear pattern

Treatment

- Localised → Topical Corticosteroids
- Severe disease →
    - Oral corticosteroids.
    - Dapsone
    - Tetracycline Antibiotics
    - Nicotinamide

active space

- Immunosuppressants →

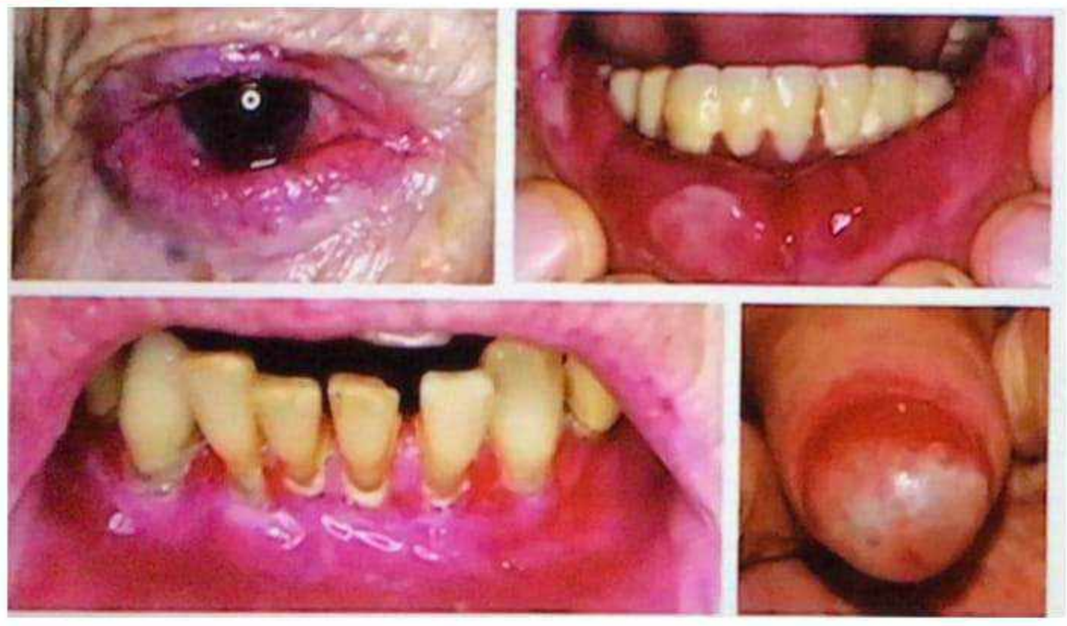
  i) Azathioprine

  ii) Cyclophosphamide

  iii) MMF

MUCUOUS MEMBRANE PEMPHIGOID 38:08

- Severe mucosal involvement
  ↓
  Scarring



CICATRICIAL PEMPHIGOID 38:46

- Skin lesions are present
- Extensive scarring present over lesion.
- Variant : Burnsting Perring Syndrome
  - There is involvement of Head and neck
  - No mucosal involvement

Pemphigoid Gestationis

- **Population affected :**
  - Pregnant females 3rd TM and after delivery.
  - also associated with Trophoblastic tumors
- **Antigen :** Herpes Gestationis factor (BP 180)
- **Antibody :** IgG → C_3 deposition

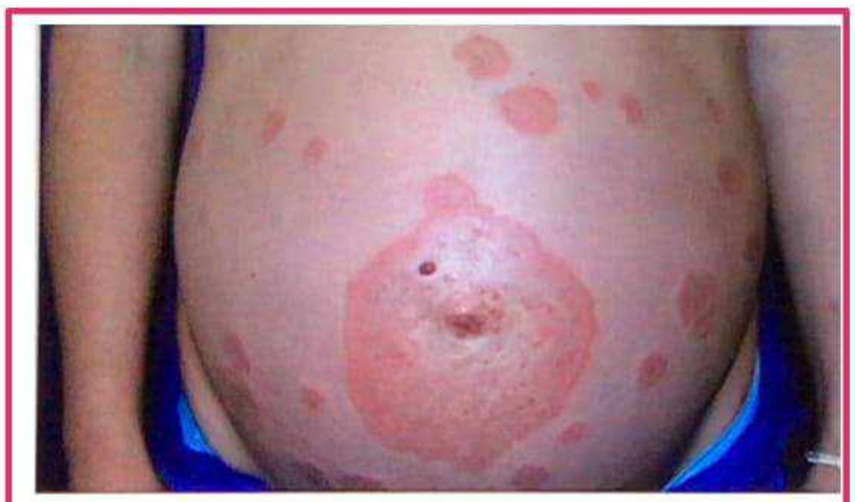
Clinical features :

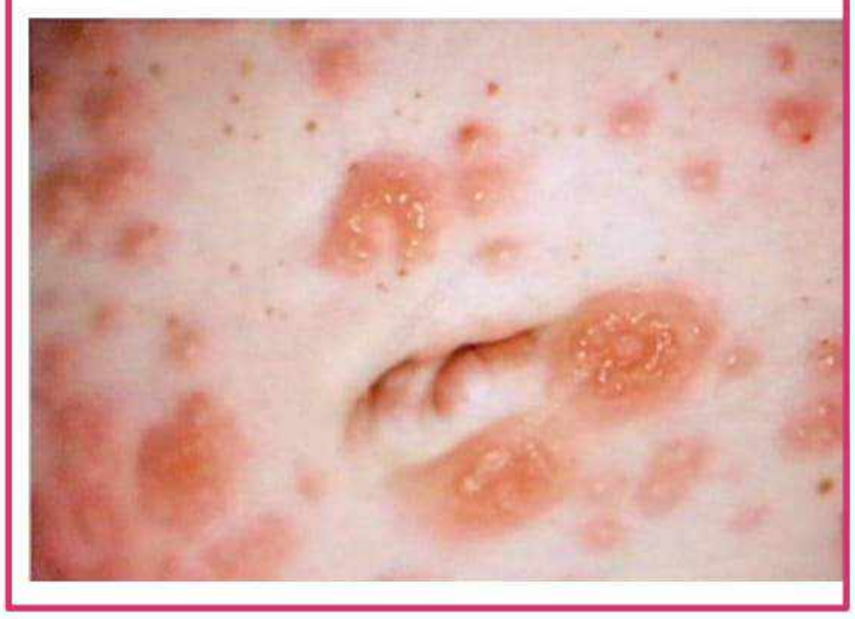
- Starts around the umbilicus as urticarial lesions
- very itchy

↓

Patient develops bulla and vesicles over the lesions

- **Course :** may recur in subsequent pregnancy





On Histopath : Subepidermal split

On DIF : C_3 deposition in 100% of cases. (Linear C_3 deposit IgG)

Treatment

- Supportive → Anti-histamines + Topical steroids.
- Oral corticosteroids.

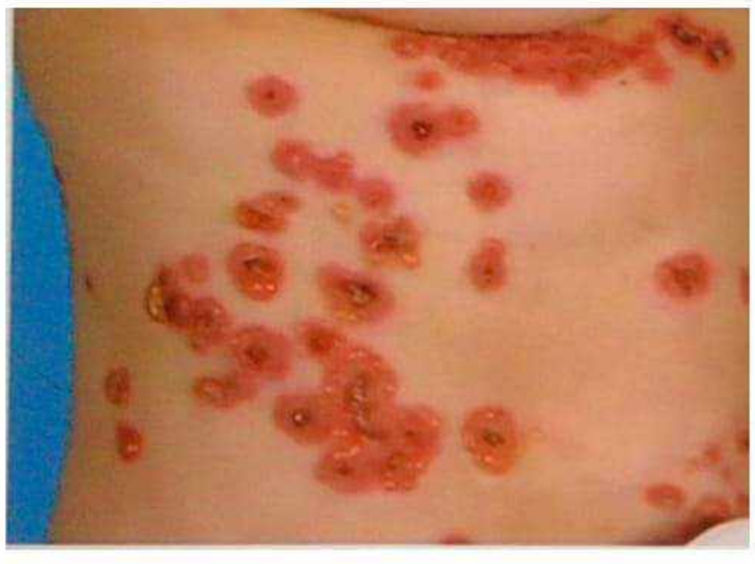
LINEAR IgA DISEASE

45:00

- Antigen : LAD_1
- Antibody : IgA
- Age group : "Bimodal"
  - Children → Chronic Bullous Disease of Childhood. (CBDC)
  - Adults → LAD
- Sites :
  - In children → starts in perioral area

Clinical features :

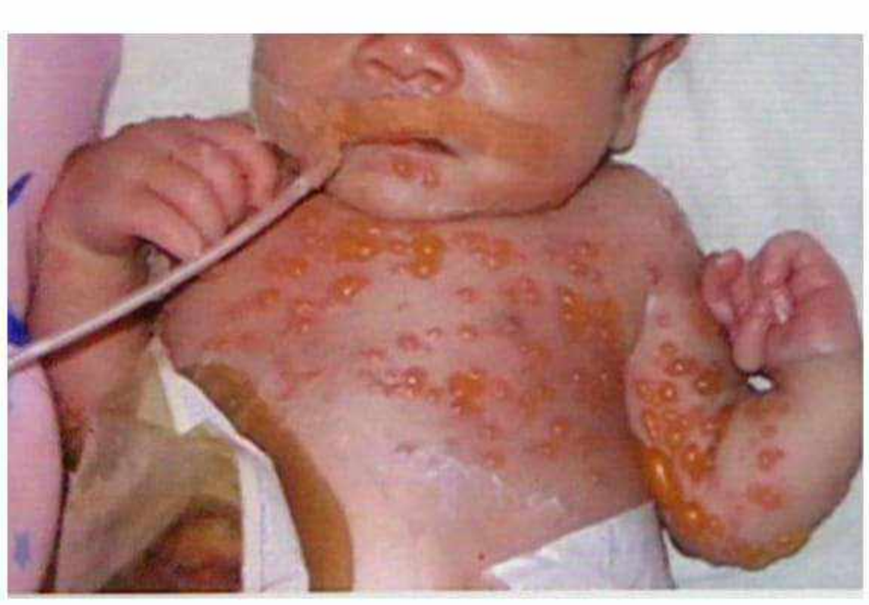
- Tense Bullae on erythematous Background.
- Crown of Jewels / Cluster of Jewels
- Itchy
- Crusting ⊕
- Excoriations

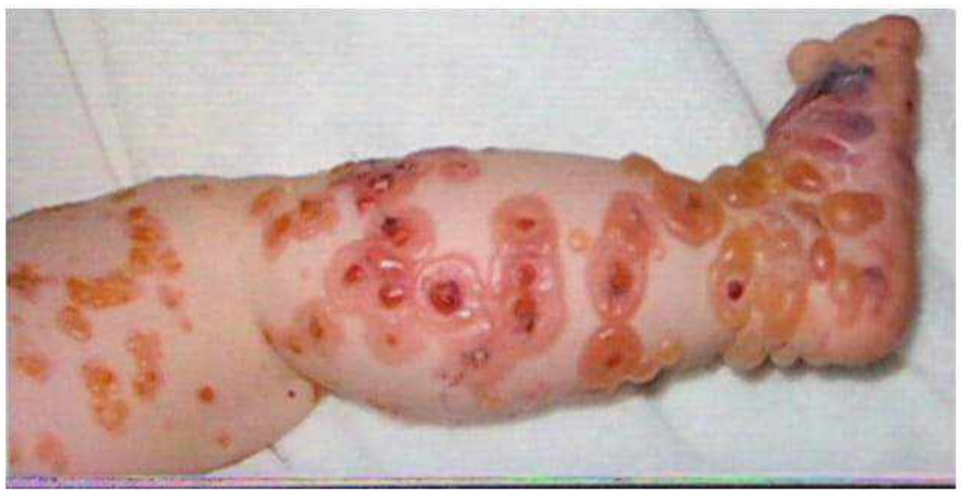


active space

CBDC

- lesions starts periorally
- Tense Bullae arranged in annular pattern.
- Crown of Jewels appearance





On Histopathology : Supepidermal split

On DIF : Linear deposit of IgA

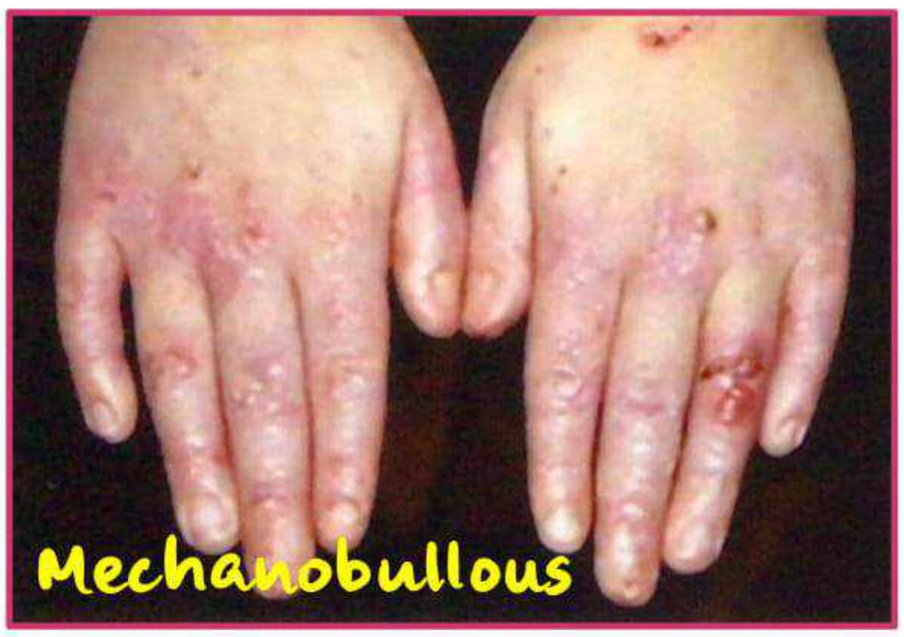
Treatment

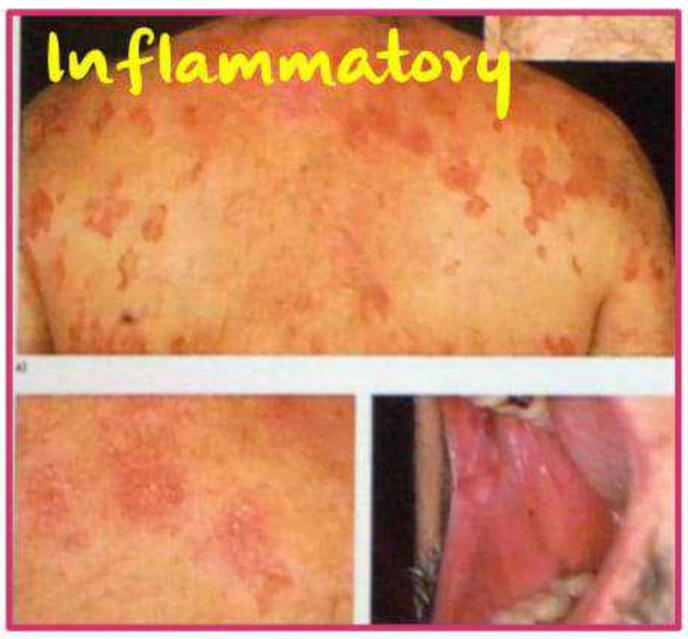
- Dapsone - TOC

EPIDERMOLYSIS BULLOSA ACQUISITA 50:30

- Antigen: Collagen VII
- Types : i) Mechanobullous → happens at site of trauma, scarring ⊕

  ii) Inflammatory





- How to differentiate b/w EBA and BP ?

- Take specimen → Put in 1M Nacl
↓
Split BMZ at Lamina lucida
↓
Add fluorescent Ab

} Salt-Split Technique

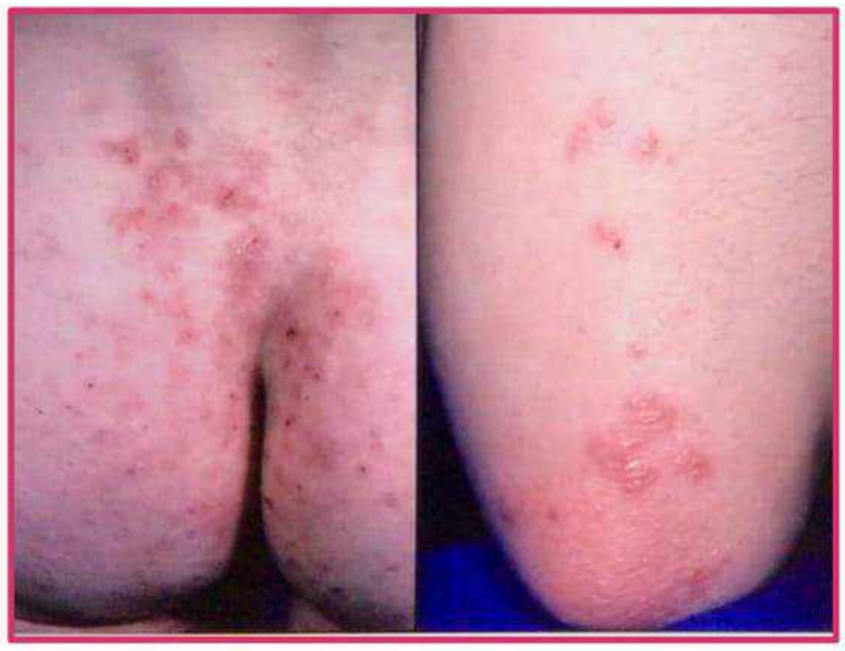
- In BP → Ab settle above lamina lucida
- In EBA → Ab settle below lamina lucida

DERMATITIS HERPETIFORMIS

55:15

- Association : Gluten Sensitive Enteropathy
- HLA association : DQ2, B8
- Iodide provocates
- Antigen : Transglutaminase (Epidermal)
- Antibodies ⊕

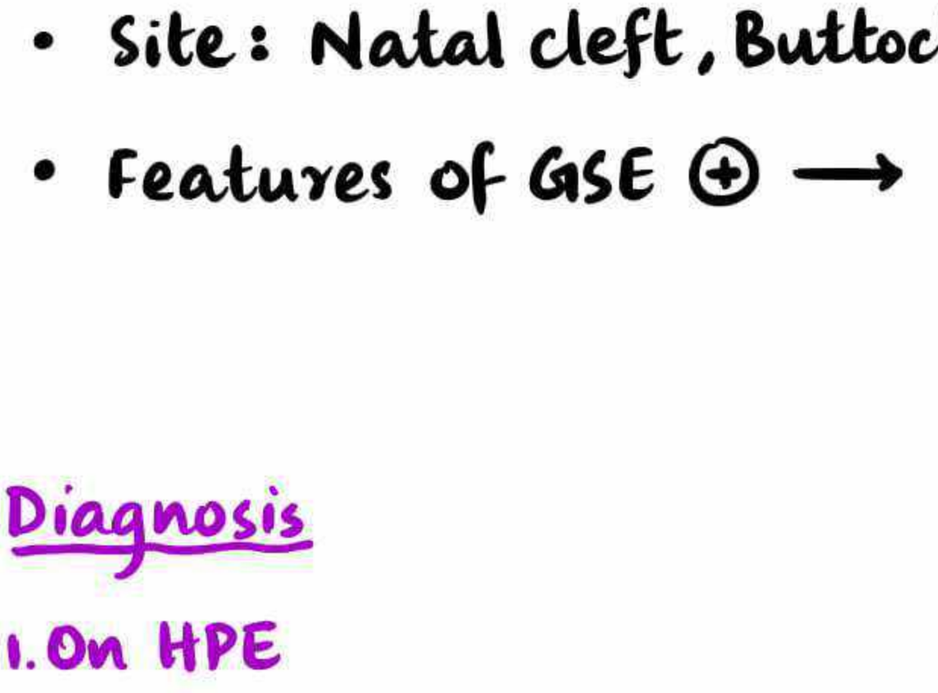
Clinical feature :



- No tense blisters
- Itchy excoriated papulovesicular lesions
- present more on extensors.
- Site : Natal cleft, Buttocks.
- Features of GSE ⊕ ⟶ Malabsorption, Bloating, Diarrhoea.

Diagnosis

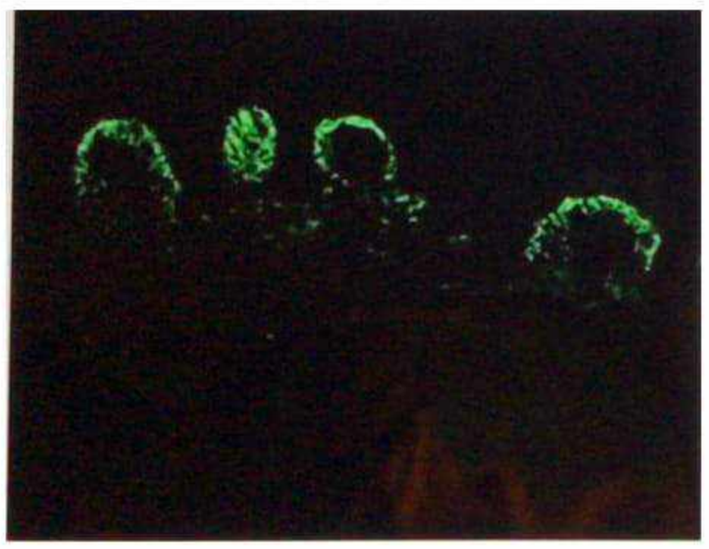
1. On HPE



- Cleft (subepidermal) ±
- Papillary microabscess ⊕ (Neutrophils) present at papillary tip

active space

On DIF :



- Immunofluorescence at papillary tip

Treatment

- Avoid Gluten (Barley, Rye, Oats, Wheat)
- Patient can have maize and Rice.
- Dapsone and other sulphonamides may be used.
  ↳ used as therapeutic test

BULLOUS SLE

- Antigen : Collagen VII
- seen usually in SLE patient
- Bulla may not always be of LE lesions
- LE lesions + Tense Bulla.

On DIF : Granular and Linear deposit of IgG and C_3

active space

Disorders of Glands

- 2 Types of Glands present are :

  i) Sweat glands

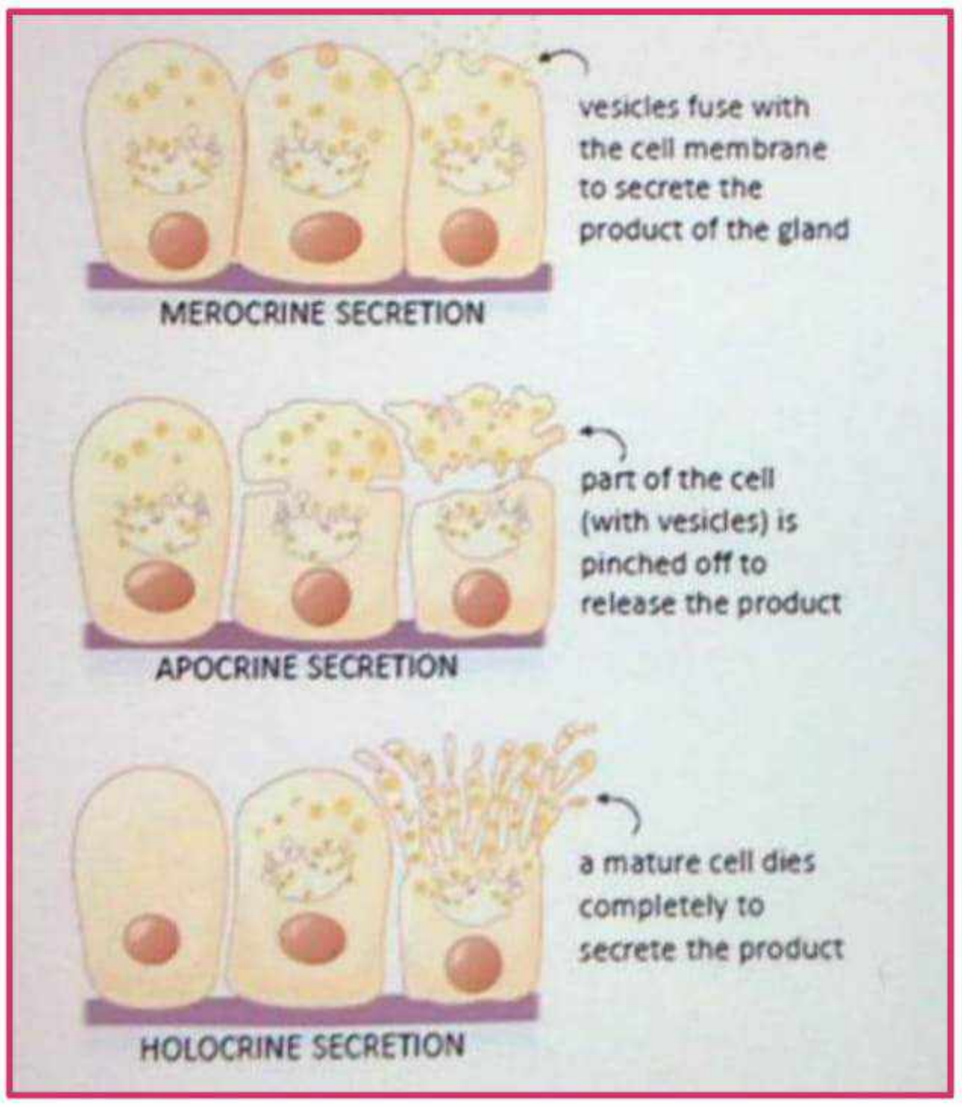
    - Eccrine glands
    - Apocrine glands

  ii) Sebaceous glands.

TYPE OF SECRETIONS

01:13

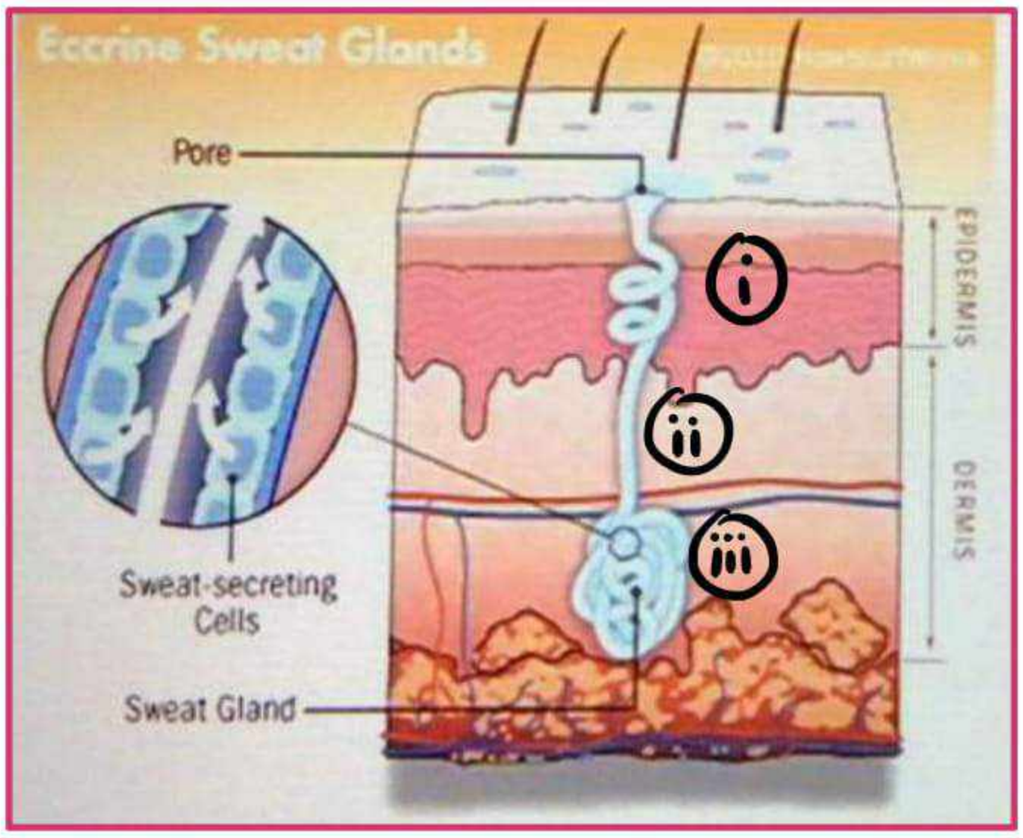
- Merocrine secretion seen in Eccrine glands
- Holocrine secretions seen in Sebaceous glands.



ECCRINE SWEAT GLANDS

03:03

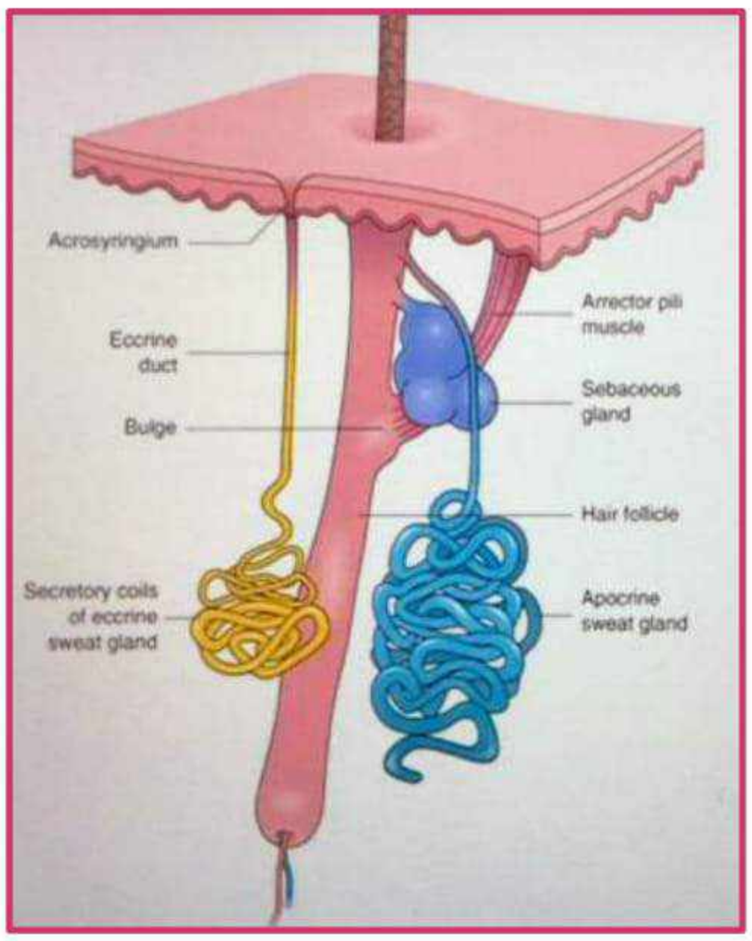
- has 3 parts :
  - i) coiled intraepidermal part (Acrosyringium)
  - ii) Straight tubular part
  - iii) Secretory coil.
- open directly into skin



APOCRINE SWEAT GLAND

04:45

- always open into hair follicle.
- has 2 parts :
  - i) Straight tubular part
  - ii) Coiled secretory part.



active space

	ECCRINE	APOCRINE
Location	• all over the body • predominant on palms and soles.	• present in Groins, Axilla, around nipples.
Type of secretion	• Merocrine	• Apocrine
Opening	• Directly into skin	• Opens into hair follicle
Innervation	Cholinergic	• Adrenergic
Secretion	Watery secretion (Thermoregulation)	• Viscous secretion – in response to stimuli • Pheromones → Odour.
		• donot have role in thermoregulation
onset of activity	• From beginning	• From puberty.

active space

ECCRINE GLAND DISORDERS

10:45

HYPERHIDROSIS

- Increased eccrine secretion more than that required for thermoregulation.
- It can be :

i) Generalised -

- Physiological → • Hot Humid environment
  - Stress
- Pathological → • Acute febrile illness
  - M.I

ii) Localised -

- Palmoplantar
- Axillary
- Gustatory

- Palmoplantar hyperhidrosis patients are more prone to :

i) Dyshidrotic eczema / Pompholyx

ii) Dermatophytosis.

iii) Pitted keratolysis.

active space

- secretion can be due to stress, emotional stimuli.

On Minor's Starch Iodine Test :

Axillary Hyperhidrosis :

- more commonly seen in post-pubertal individuals.
- seen in age group : 13-15yrs.

Treatment of Hyperhidrosis :

i) Topicals → • 20% Aluminium hexachloride.
- Formaldehyde
- Glutaraldehyde

ii) Oral anticholinergics :

- Glutaraldehyde.
- Oxybutynin

iii) Botulinum toxin

iv) Iontophoresis.

v) Surgical therapy

ANHIDROSIS / HYPERHIDROSIS

18:50

- production of less sweat even in the presence of appropriate stimuli.

MILIARIA

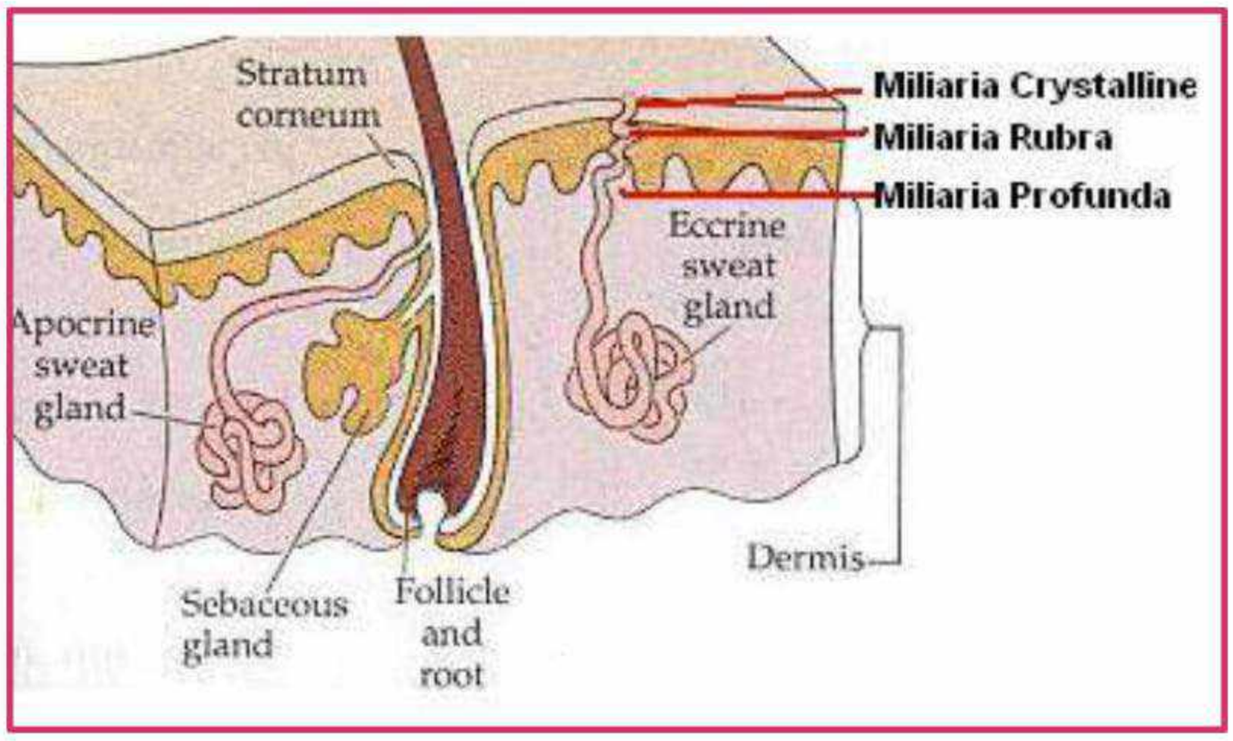
- commenest sweat gland disorder
- Defect : No eggress of sweat because of blockage in intraepidermal part of sweat duct.
- Types :

i) Miliaria Crystallina → Blockage at Stratum corneum.

ii) Miliaria rubra → defect in intraepidermal part.

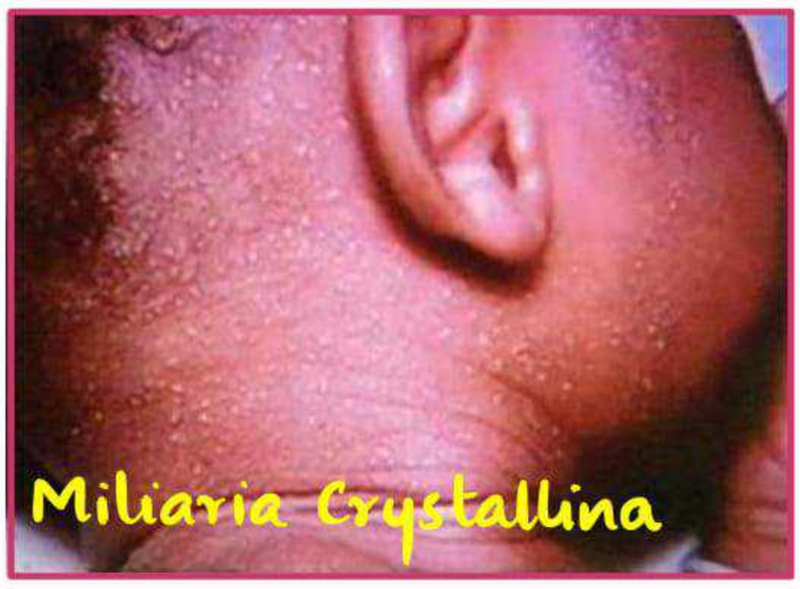
iii) Miliaria profunda → Blockage is at or below the Dermo epidermal junction.

active space



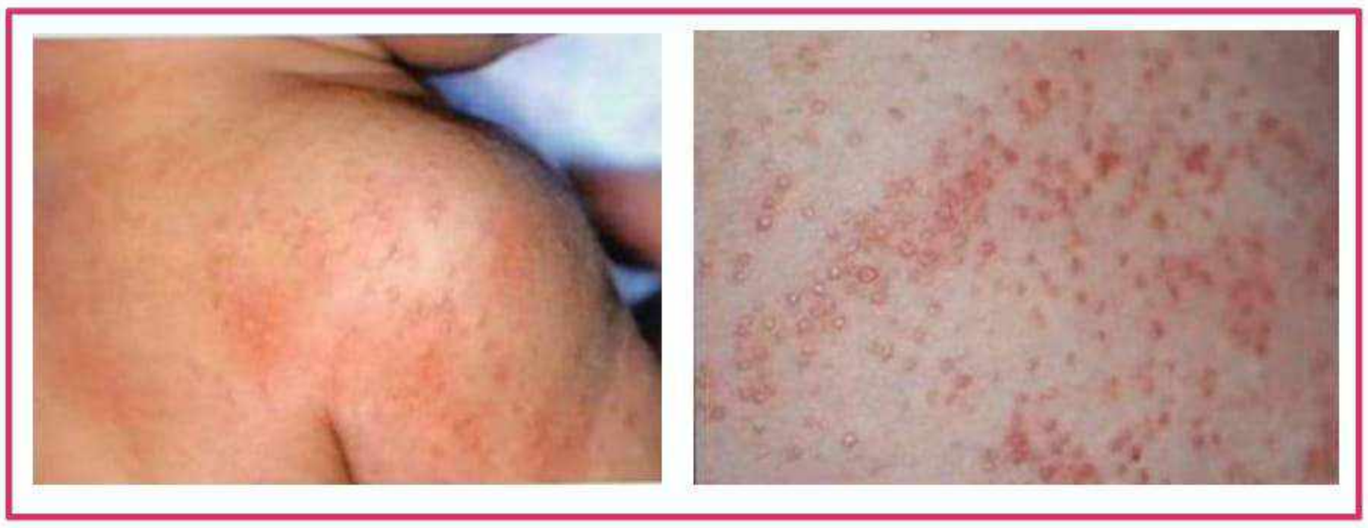
1. MILIARIA CRYSTALLINA

- superficial thin walled vesicles filled with clear fluid.
- Asymptomatic.
- seen commonly in infants.



2. MILIARIA RUBRA

- most common type
- a.k.a Prickly Heat



Symptoms

- Itchy
- Burning sensation.
- small erythematous red papular lesions on trunk, face etc.

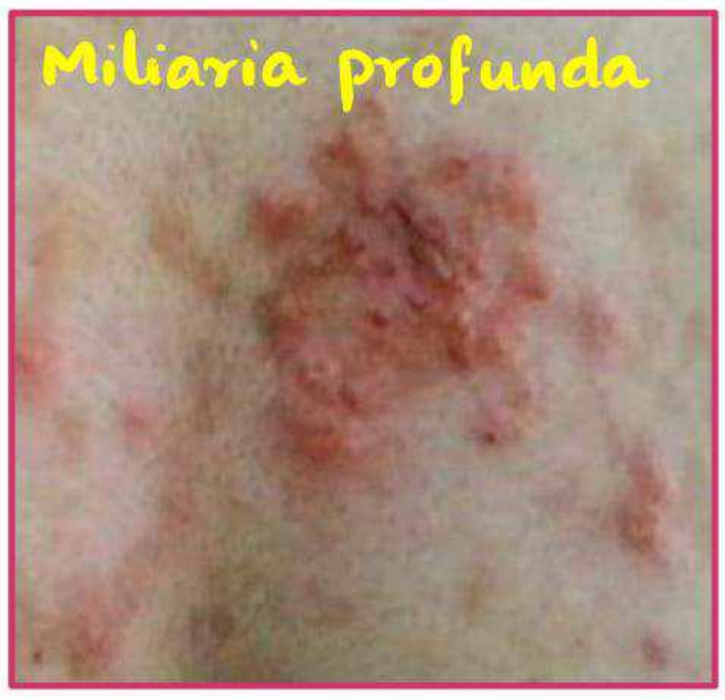
active space

Miliaria pustulosa :

- when Miliaria rubra gets infected with staphylococcus pustules are formed.

3. MILIARIA PROFUNDA

- usually seen in Tropics
- Rare
- Asymptomatic
- Presents with deep papules and nodules.



Treatment

- Stay in cool environment.
- cold compresses
- Menthol , Calamine lotions
- Antihistamines
- Topical steroids.
- In case of pustulosa → Antibiotics may be used.

active space

NEUTROPHILIC ECCRINE HIDRADENITIS 28:35

- occurs in patients on chemotherapy
- There is inflammation and necrosis of eccrine epithelium and inflammation is neutrophilic in nature.
- Presentation : Painful papules and plaques over upper trunk.

DISORDERS OF APOCRINE GLANDS 30:00

1. BROMHIDROSIS

- Bad odour → because of Bacterial overgrowth on apocrine sweat gland.
- seen in pubertal or post-pubertal age group.

Treatment

- Decrease Garlic in diet
- use deodarants.

2. CHROMHIDROSIS

- colour in apocrine sweat
- colour is due oxidation of lipofuschins
- Colour can be → Blue, Green, Yellow

active space

3. FOX FORDYCE DISEASE

- a.k.a Apocrine miliaria.
- Obstruction in opening of the apocrine gland.
- seen in post-pubertal (13-18yrs)
- Affected : Females > Males.

Clinical feature :

- Pruritic skin coloured / brownish papules on axilla, groins, around nipples.

4. HIDRADENITIS SUPPURATIVA

- chronic recurrent inflammatory disease affecting apocrine gland.
- a.k.a Acne inversa
- Site : Axilla, Groins, Mammary area, Buttocks, Around umbilicus
- Age : Post-pubertal individuals
- Course : chronic remitting relapsing course.

active space

Criteria

1. Typical site
2. Chronic Recurrent Nature
3. Typical lesions → Nodules, Abscesses, Bridging scars, Sinuses, Comedomes.

Predisposing factors

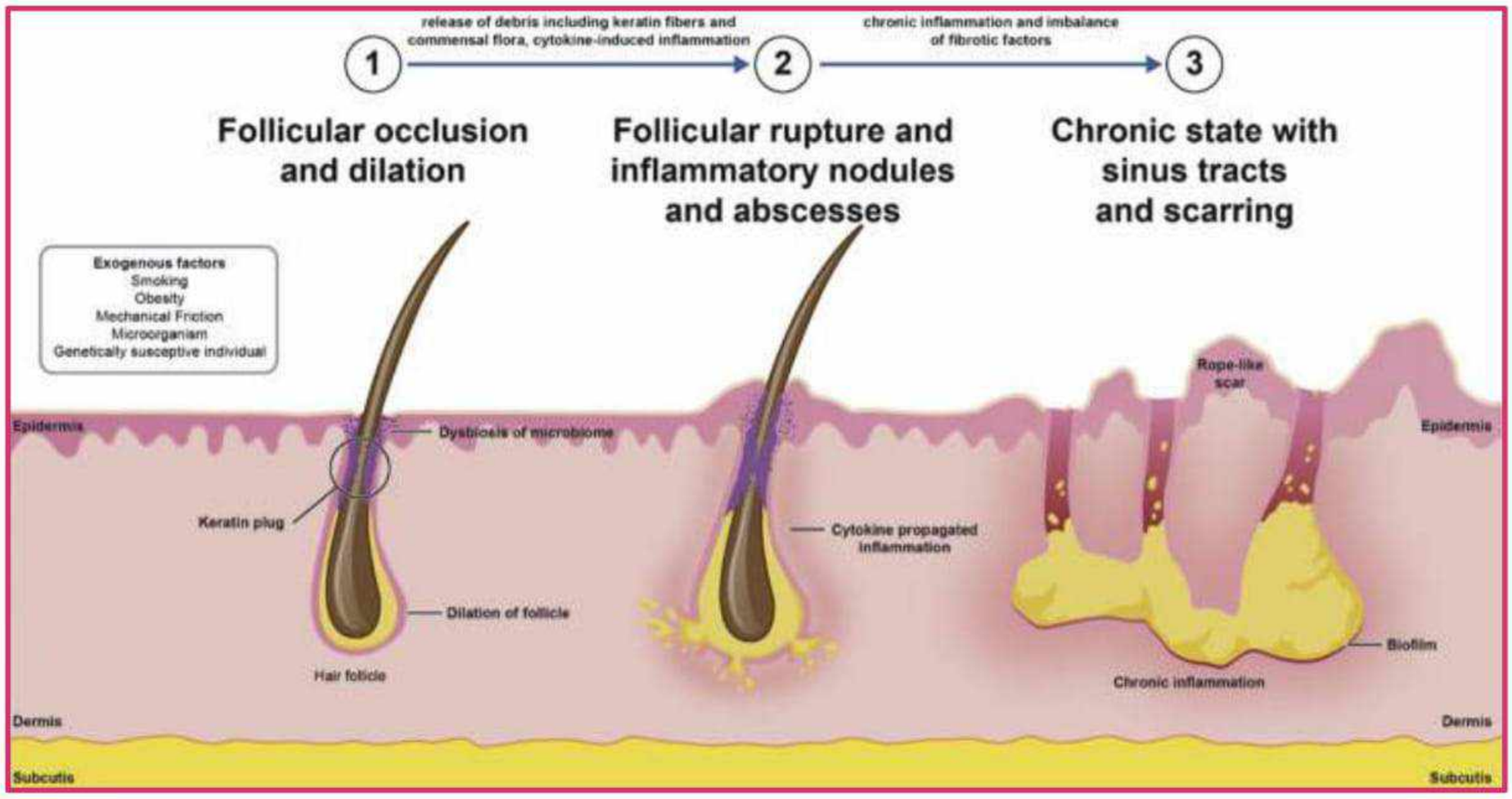
- Obesity
- Smoking
- Premenstrual flare

Association

- Follicular Occlusion Triad :
  - i) HS
  - ii) Acne conglobata
  - iii) Dissecting cellulitis of Scalp.
- Follicular Occlusion Tetrad :
  - Triad + iv) Pilonidal sinus.
- Crohn's disease.
- Severity Staging → HURLEY Staging.

active space

Pathogenesis :

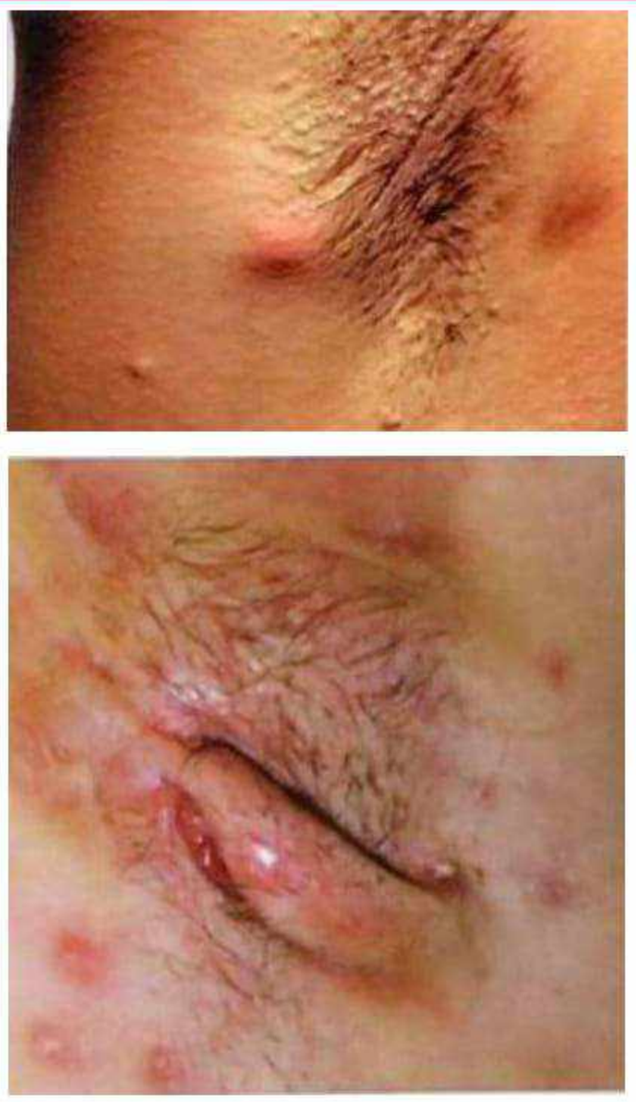


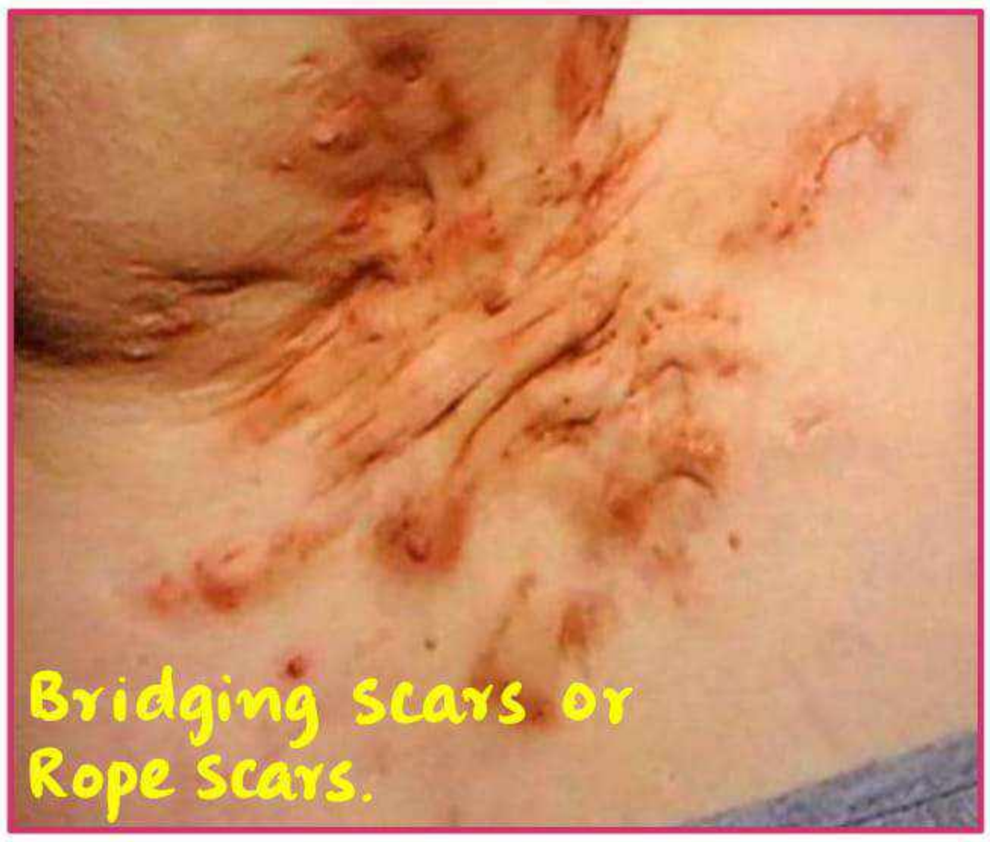
Patient presents with :

- Comedomes → Polyporous
- Deep s/c nodules and few papules → painful s/c nodules and abscess

↓

forms multiple sinuses.





Treatment :

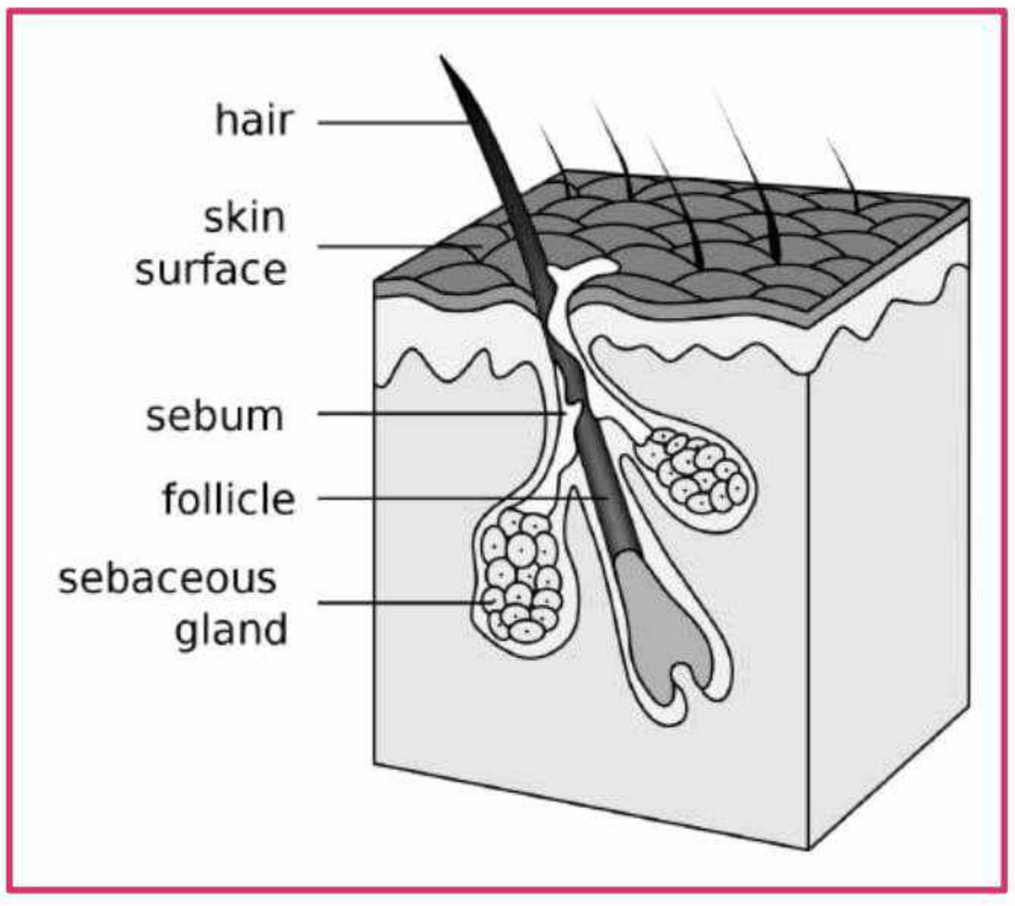
- General advice →
  - Decrease weight
  - Quit smoking
- Analgesics → NSAID's
- Antibiotics → Tetracyclines
  - Doxycycline, Clindamycin, Rifampicin
- Anti-inflammatory → Dapsone, Intralesional Steroids
- Retinoids → Acitretin
- Biological → Adalimumab (Anti TNFα inhibitor)
- Surgical therapy.

active space

DISORDERS OF SEBACEOUS GLANDS

48:35

- Location:
  - present on all over the body except on palms and soles.
  - more concentrated on seborrhoeic areas → face, scalp, upper back and chest.
- opens into hair follicle
- Development: Increased during puberty.
- Function: Lubricant, Anti-microbial.
- Type of secretion: Holocrine secretion.



active space

Modifications : open directly into skin

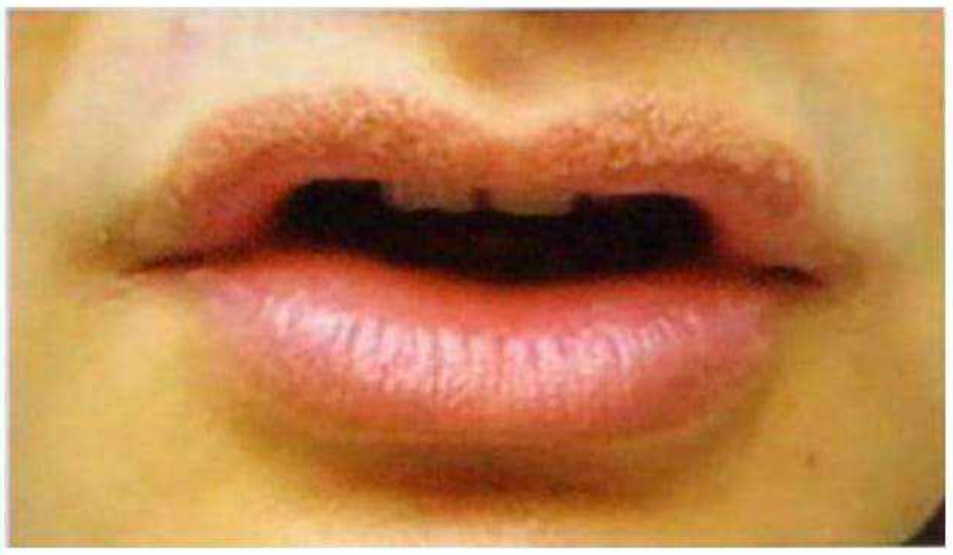
i) Eyelids → Meibomian glands

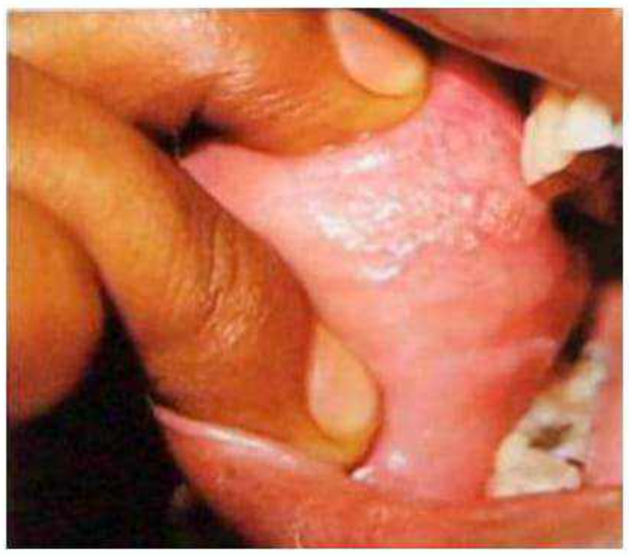
ii) Mammary → Montgomery tubercles

iii) Oral mucosa → Fordyce spots

iv) Penis → Tysons glands.

Fordyce spots :





ACNE VULGARIS

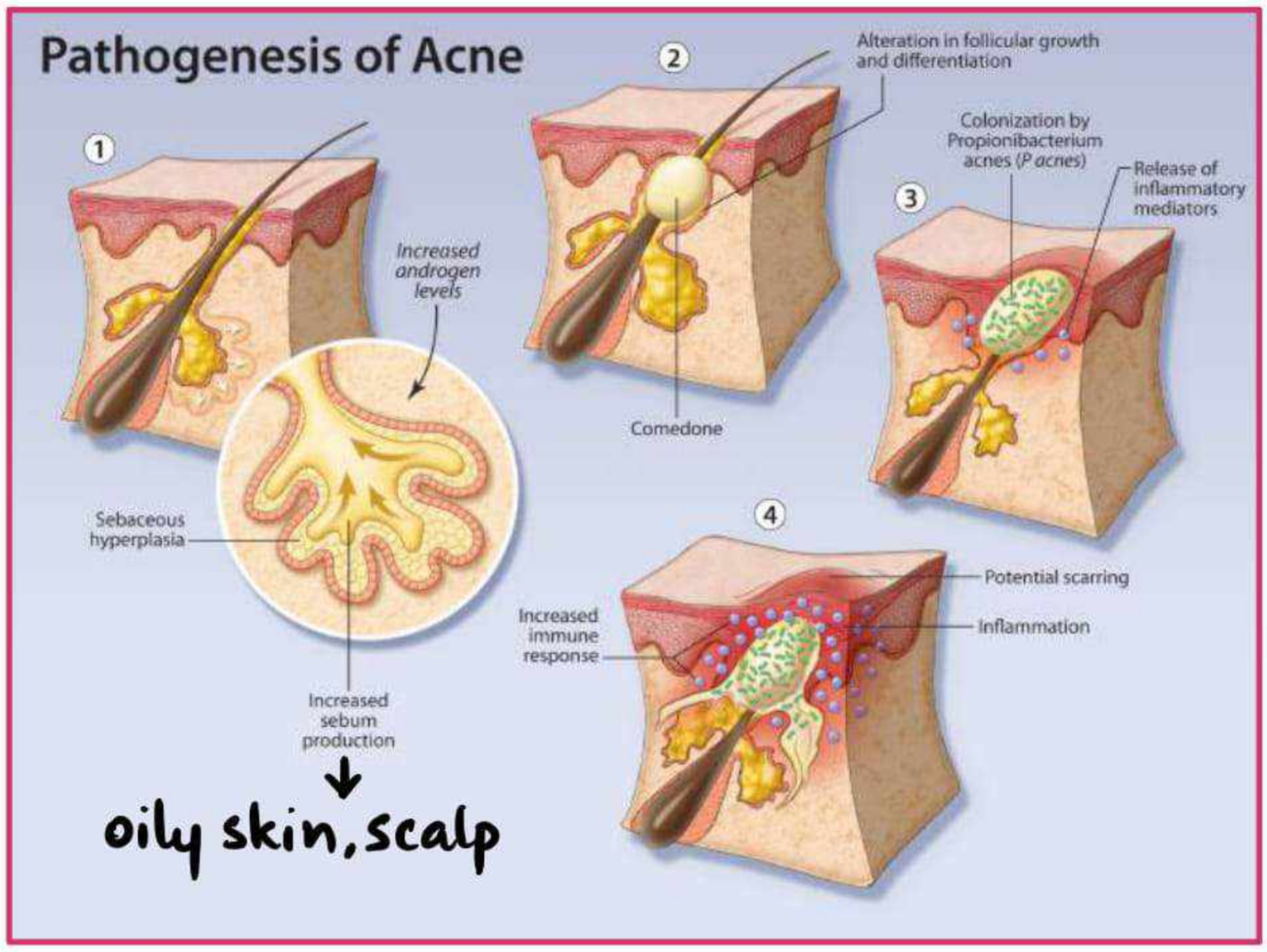
53:15

- chronic inflammatory disorder of sebaceous gland.
- usually seen post-puberty
- Polymorphic lesions
- seen on Face, Trunk, Chest, upper arms.

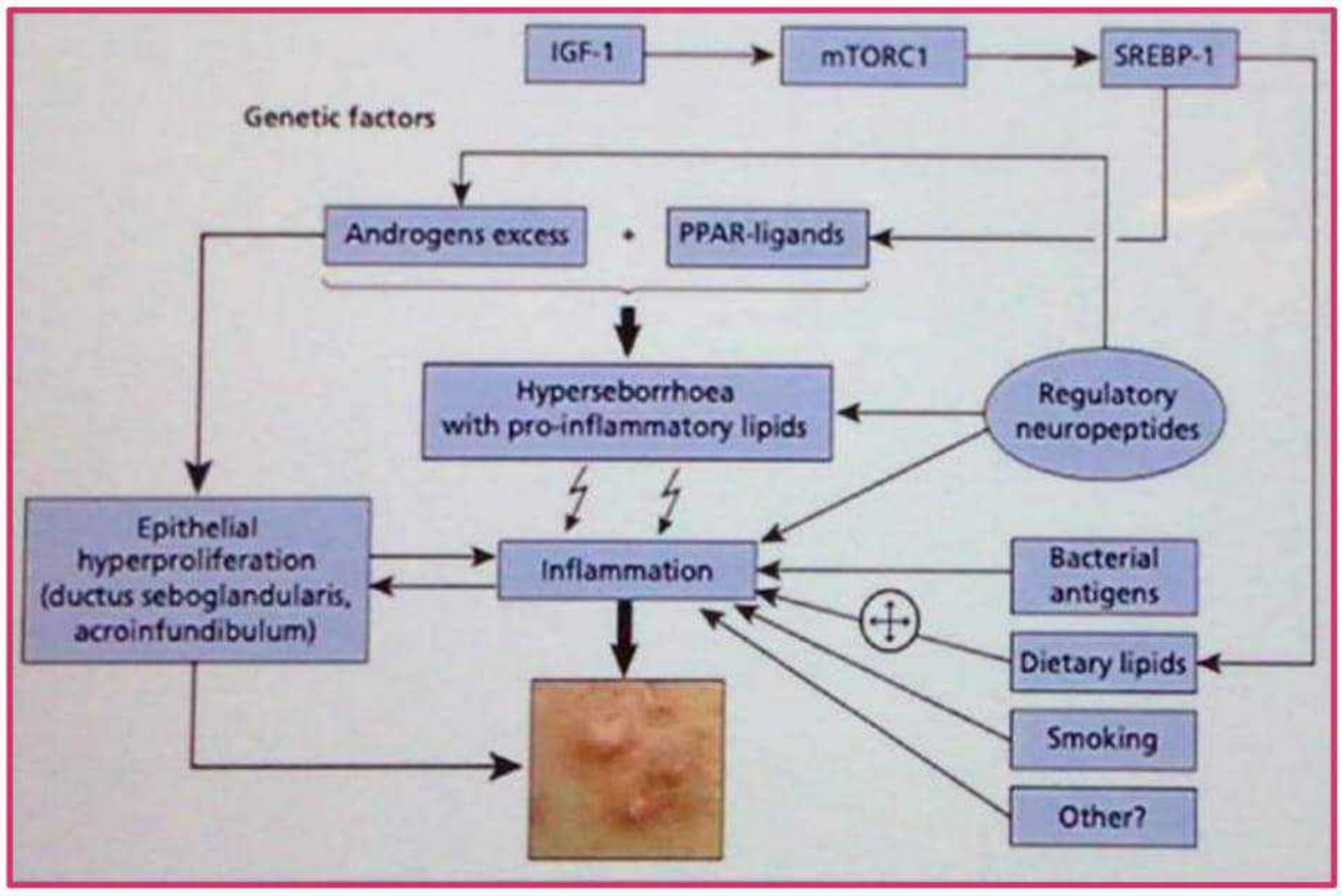
active space

Pathogenesis of Acne :

1. Seborrhoea
2. Colonization
3. Hyperkeratinization
4. Inflammation.



- Androgens :
  - Seborrhoea,
  - Follicular keratinization.
- Resident flora
  - P.ovale
  - P.acnes
  - Staph.epidermidis.



- Release lipases → converts TG's to FFA → Blockage Inflammation.

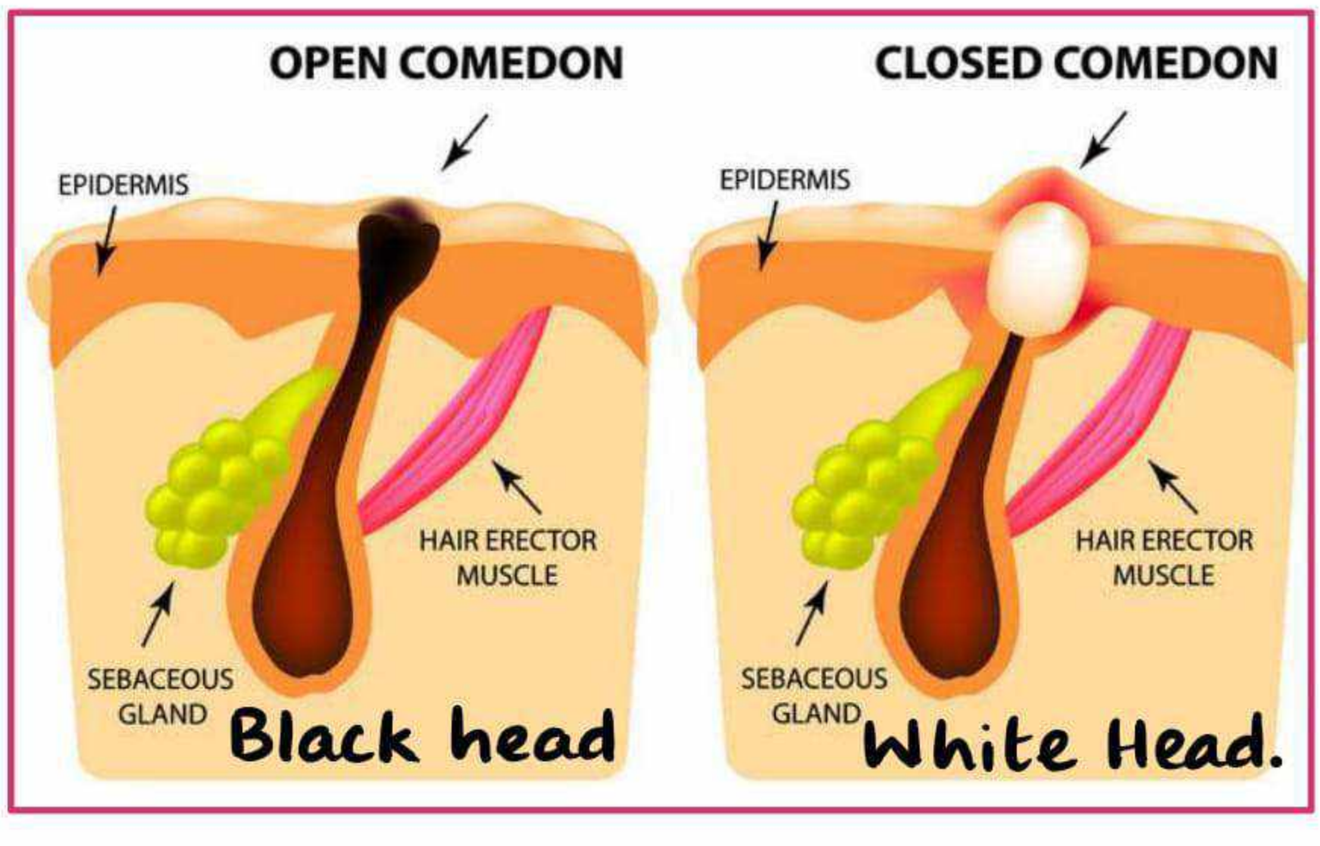
active space

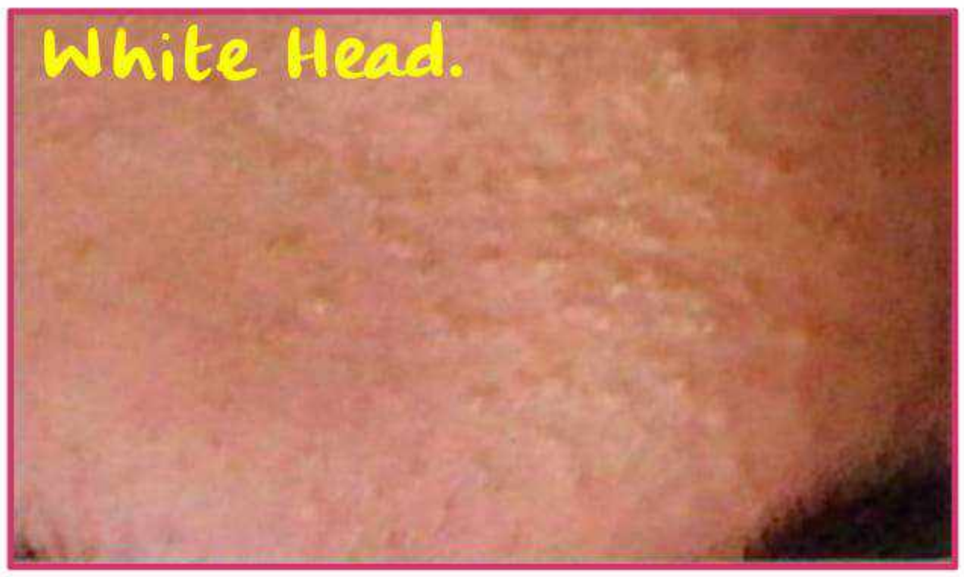
- **Inflammatory markers :** IL-1 , TNF-α , IL-2, IL-8
- **Environmental factors :**
  - Hot Humid Climate
- **Diet :**
  - Foods with high glycemic index ⟶ ↑ IGF-1
    ↓
  - are dairy products, milk
  - sugars

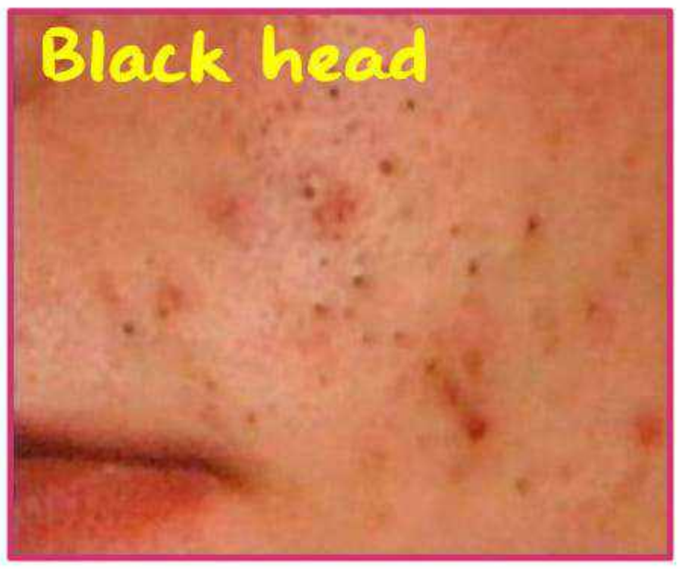
Clinical features :

- Location : Seborrhoeic distribution
- Sequence :

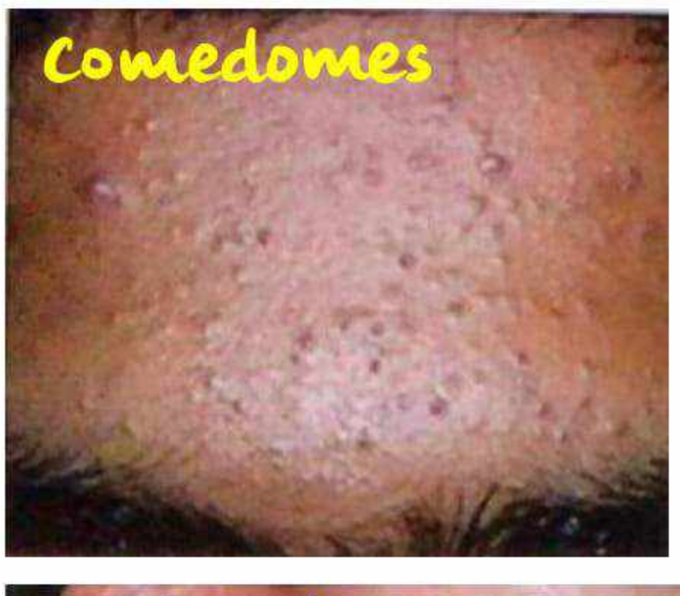
comedomes → papules → pustules → nodules/ cysts → Scar.

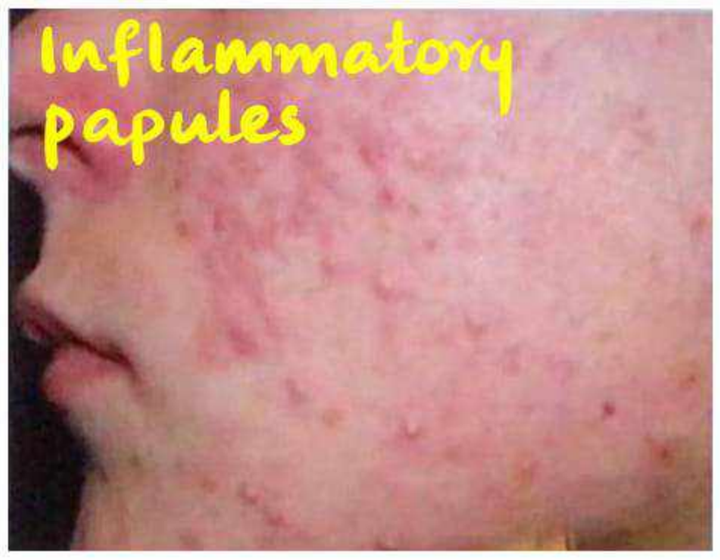


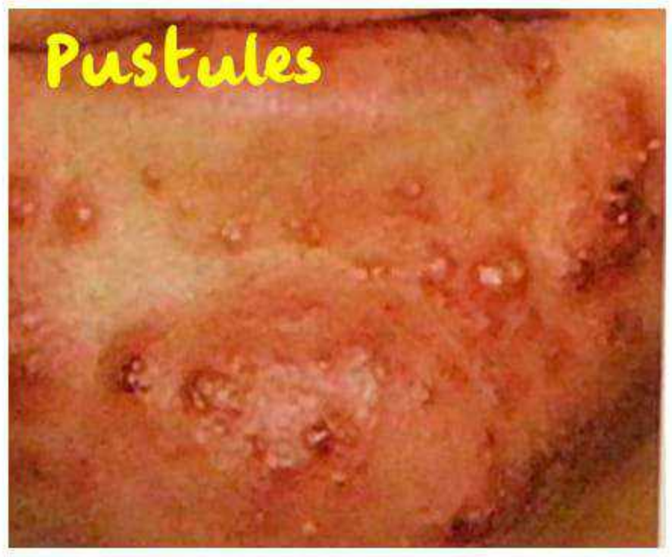


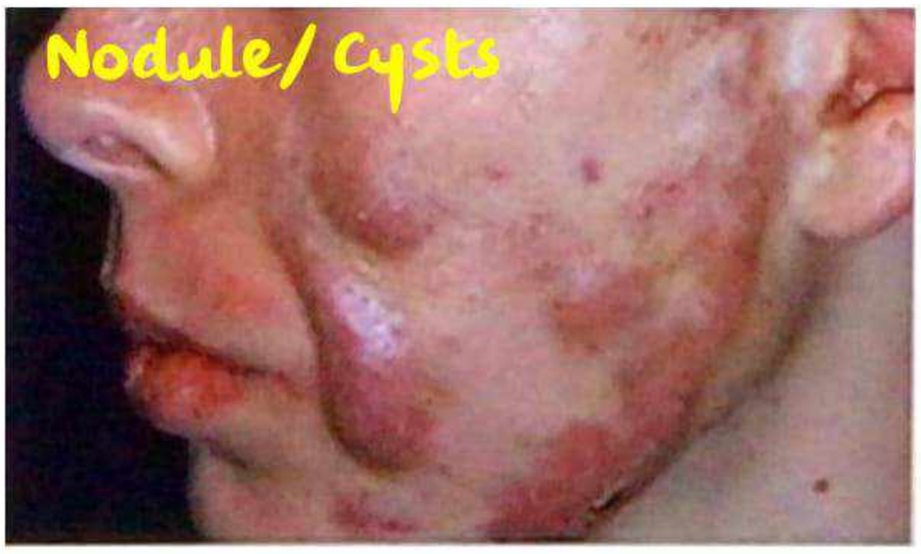


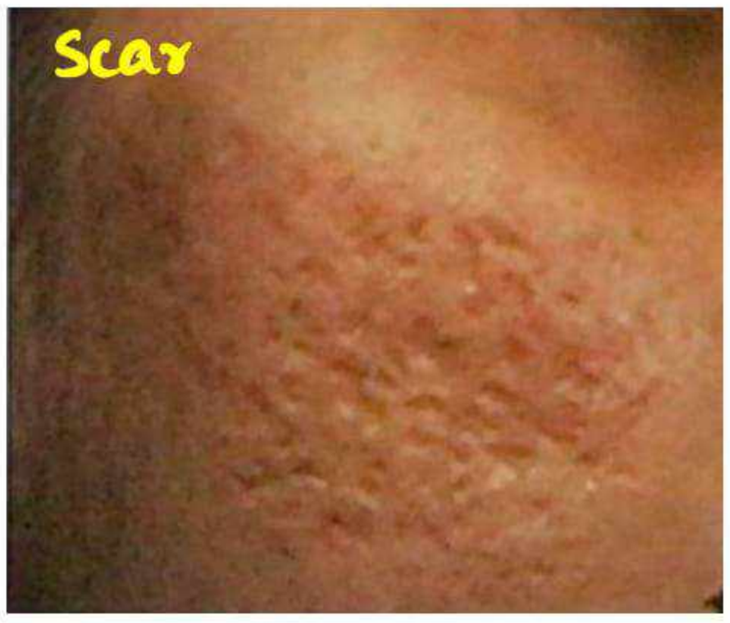
Black head: due to oxidation of melanin



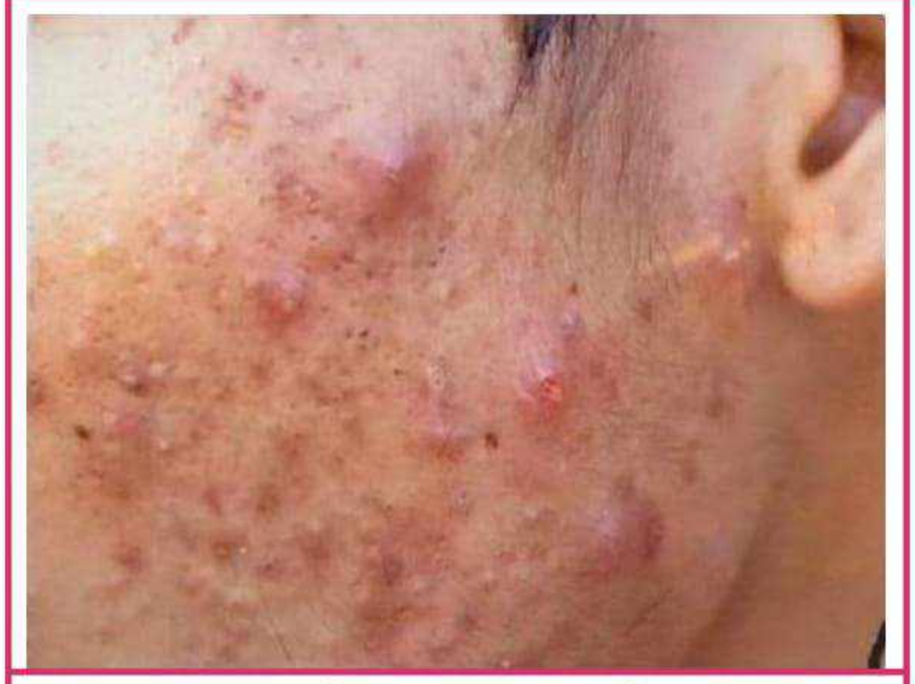








active space



Acne → Polymorphic lesions

ACNE VARIANTS

01:08:35

1. HORMONAL ACNE

- location : lower face
- cause : Androgens

Produced from

Adrenals	Ovaries
• CAH	• PCOS
• Adrenal tumors	• Ovarian tumors.

Variants of PCOS :

1. SAHA Syndrome : Seborrhoea Acne Hirsuitism AGA.
   - Signs of Hyperandrogenism

active space

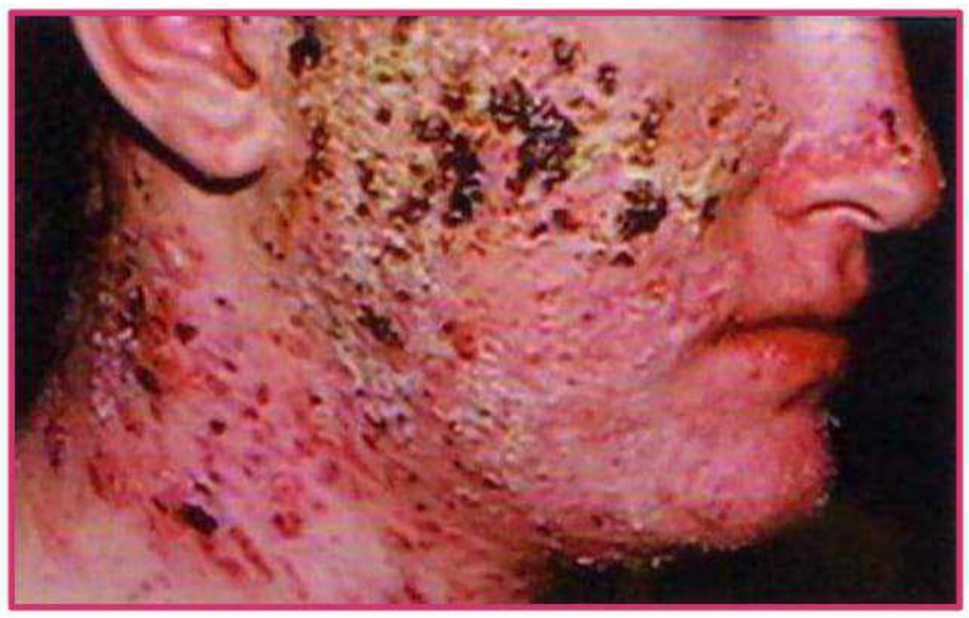
- Signs of Hyperandrogenism
  - Hirsuitism
  - Increased Seborrhoea
  - Early Female Pattern Hair loss
  - Lower face acne.

2. HAIR-AN Syndrome :

- Hyperandrogenism
- Insulin Resistance
- Acanthosis nigricans

2. ACNE FULMINANS

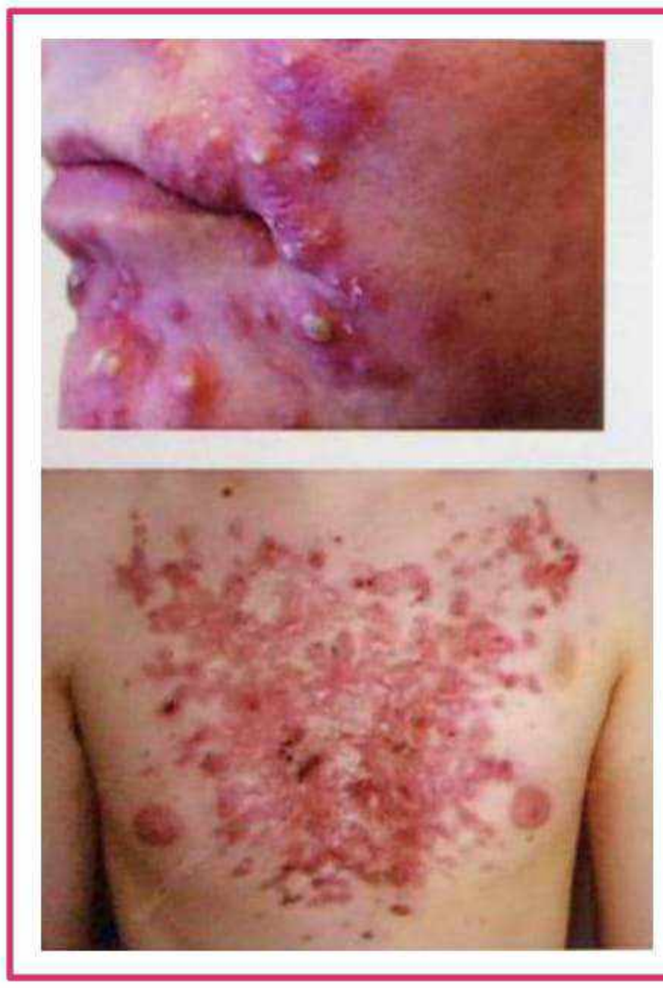
- ulcerative form
- Systemic symptoms :
  - i) fever
  - ii) Muscle pain
  - iii) Osteomyelitis of Bone.
- Acute condition



Treatment

- Antibiotics + Oral Steroids
- Oral Retinoids

3. ACNE CONGLOBATA



- Inflammatory form of acne
- Nodules, abscesses, cysts, interconnecting sinuses are seen.
- seen more on trunk.
- Males > Females

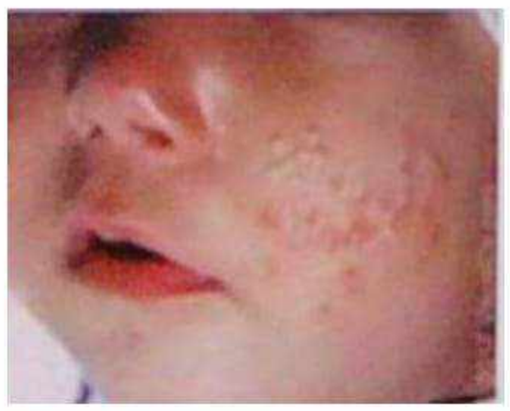
Treatment

- Oral Retinoids.
- Short course of oral steroids.

4. NEONATAL ACNE

- seen in < 3 months old
- Mild form.
- Occurs because of transfer of maternal androgens.
- No treatment.

5. INFANTILE ACNE



- seen in < 1yr old
- seen over cheeks
- No treatment

6. MID-CHILDHOOD ACNE

- seen in 1-7yrs of age.
- may be indicative of precocious puberty

7. PREPUBERTAL ACNE

- Adrenarche precedes menarche by 2-3yrs
- seen in 7-8yrs of age.

8. DRUG INDUCED ACNE

- a.k.a **Acneiform eruptions**
- Monomorphic papules
- Seen on trunk
- No comedomes are seen.

- Drugs that cause are :
  - i) ATT
  - ii) Anticonvulsants.
  - iii) Steroids.
  - iv) Antidepressants.

active space

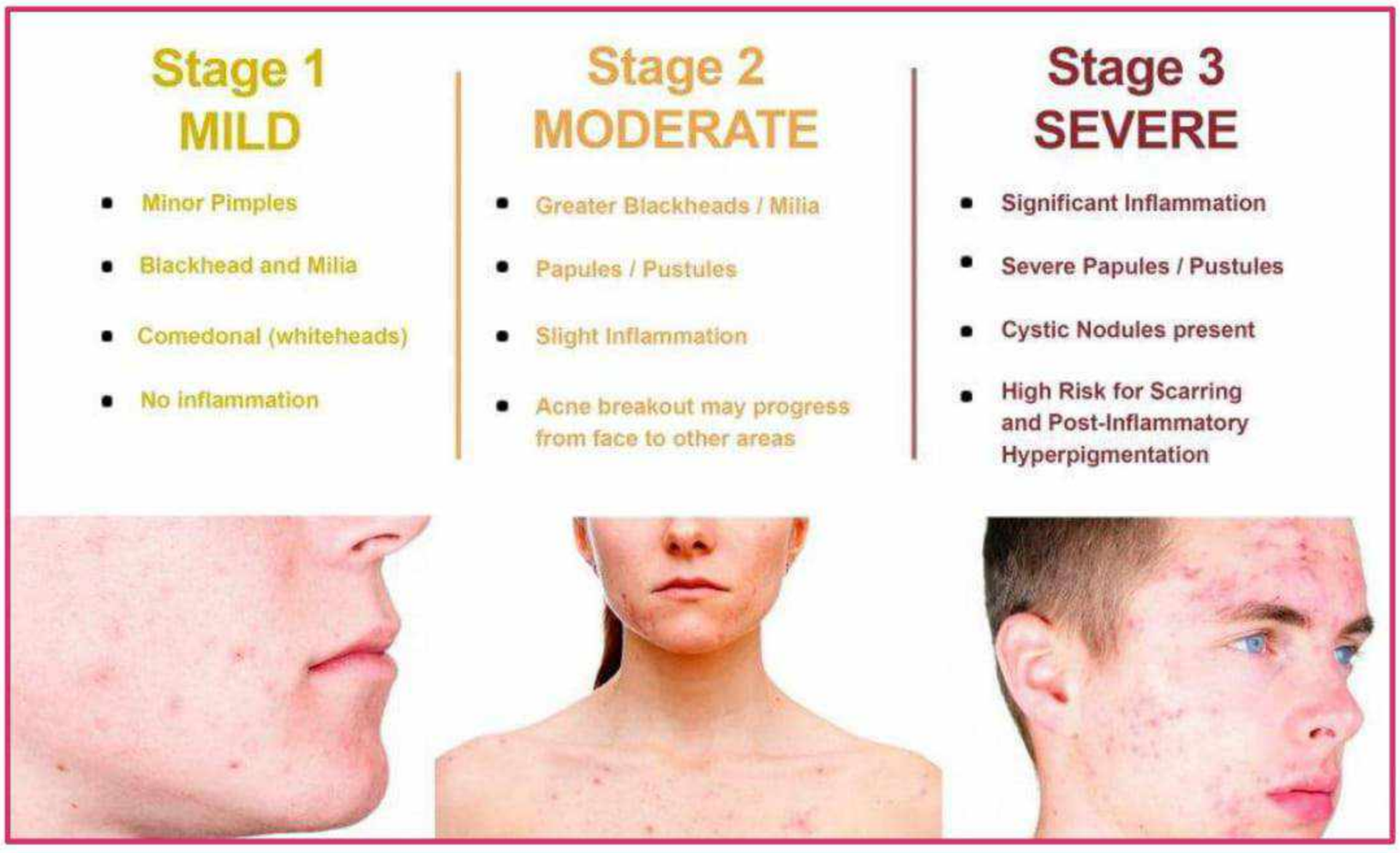
9. ACNE EXCORIEE

- Tendency to excoriate lesion.
- Extensive scarring seen
- Psychiatric disorder (OCD)

Treatment :
- Counselling
- Triflurezamine
- Primodine

10. COSMETIC ACNE

- Because of use of cosmetics.



active space

Treatment of Acne :

- if predominantly comedonal → Topical Retinoid
- if predominantly papular → Topical Retinoid + Topical Antibiotic
- if pustular : Topical Retinoid + Topical Antibiotic + Systemic Antibiotics ± Benzoyl Peroxide.
- if nodular/cystic → Oral Retinoids

Topical Retinoids

- Tretinoin
- Adaplene
- Tazarotene
- Modulate follicular keratinization
- S/E - i) Irritation

Benzoyl Peroxide

- used as 2.5%, 5% concentration.
- Both Anti-inflammatory and Antimicrobial.
- S/E : Irritation, Bleaching effect.

Topical Antibiotics

- Clindamycin
- Erythromycin.
- Lymecycline

Fixed Combination Regimens

- Topical Antibiotic + Benzoyl peroxide. (BPO)
- BPO + Adaplene.

Other Topicals

- Nicotinamide
- Azelaic acid
- Topical Dapsone
- Salicylic acid.

Chemical peels

- Salicylic acid
- Glycolic acid.

Intralesional Steroids (ILs)

- given in nodules, cysts.

active space

Systemic Antibiotics

- Doxycycline
- Minocycline
- Lymecycline
- Erythromycin (in pregnancy)

Hormonal Therapy

- OCP's
- Anti-Androgens → Spironolactone, Cyproterone Acetate.
- In Insulin Resistance patient → Metformin.

Oral Retinoids

- Isotretinoin
- works on all pathogenic factors.
- Dose : 0.5 mg/Kg to 1 mg/Kg/day.
- Maximum cummulative dose : 120 - 150 mg
- S/E :

  i) Teratogenic (wash off period is 1 month)

  ii) Dryness → presents as cheilitis, xerosis, dryness in eyes.

  iii) 2° Infections → Impetigination

active space

iv) Headache.

v) Flare up of Acne in 1st month

Surgical treatment

- Scar Revision surgeries
- LASERS.

ROSACEA

01:38:00

- Inflammatory disorder + vascular reactivity + Hyperemia.
- seen in **Adults**
- site : Convexities of face, sparing of nasolabial fold.
- **Associations :**

  i) Hot spicy foods

  ii) H.pylori infection

- **Pathogenesis**

  - Inflammation + vascular reactivity.

    ↓

    vasodilation + Hyperemia

    ↓

    Erythema and Flushing

- Pathophysiology

  i) Innate immunity

  ii) UV light

  iii) Organisms : Demodex, Staph epidermidis, Bacillus oreneous

  H.pylori

  iv) Stress

  v) Diet : Hot spicy foods.

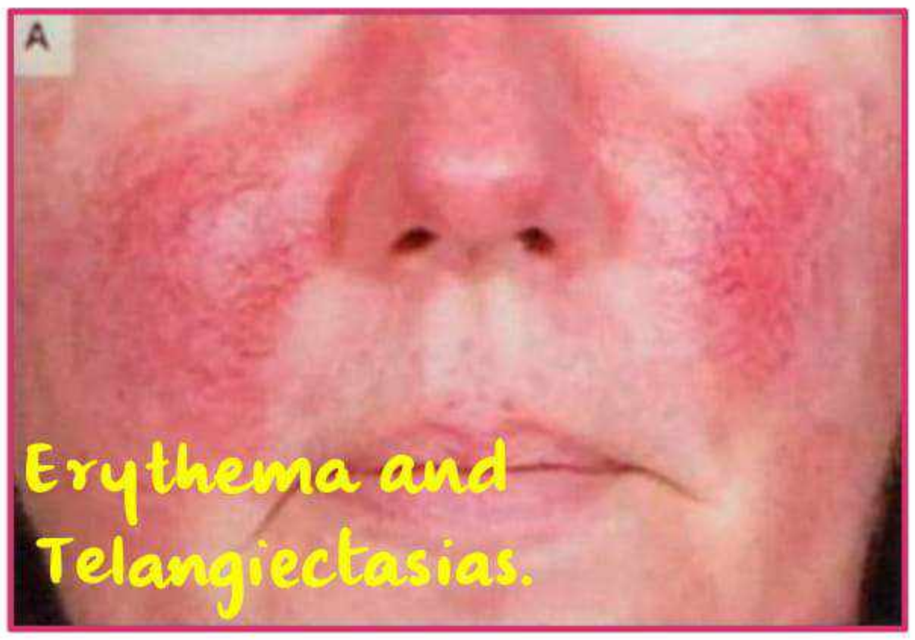
  vi) Phymatous - Increased Matrix Metallo Proteinases.

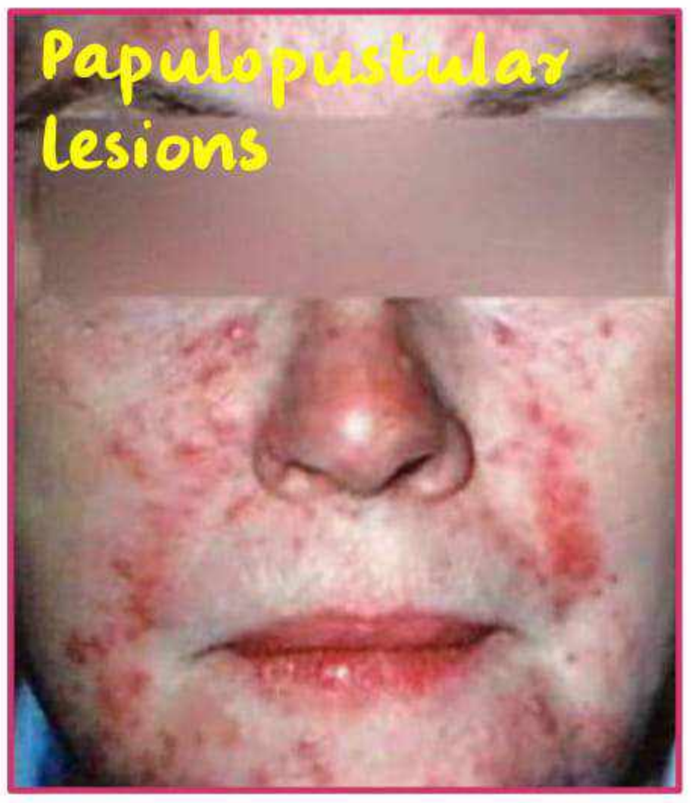
Types of Rosacea

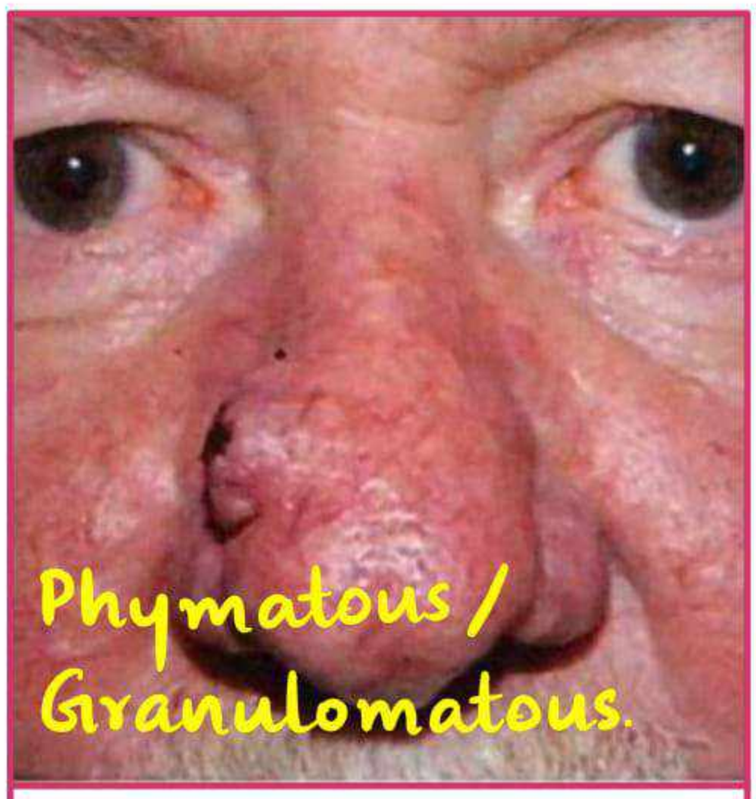
1. Erythemotelangiectatic
2. Papulopustular
3. Phymatous
4. Ocular

Clinical features :

- H/o Flushing → Red on exposure to provocative factors.
- Sensitive face
- Burning sensation.
- H/o Ocular features → watering from eyes.







Potato nose.

vs Acne

- No comedomes
- Flushing, Telangiectasias
- Sparing of nasolabial fold.

Treatment :

- in Erythemotelangiectatic face →
  - Brimonidine
  - Oral Propanolol.
  - LASERS

active space

- In Papulopustular →
  - Topical Metronidazole
  - Topical Ivermectin
  - Oral Antibiotics → i) Doxycycline
    ii) Erythromycin.
    - Low Dose Isotretinoin.
- Avoid Sun exposure.
- Avoid Hot Spicy foods
- In phymatous →
  - Active → Oral Retinoids.
  - CO_2 LASER + Resurfacing.

active space

Adverse Cutaneous Drug Reactions

- Drug reactions that cause skin lesions.

IMMUNOLOGICAL DRUG REACTIONS

00:45

Coombs and Gell Classification

1. Type I : IgE dependent → Urticaria / Angioedema
2. Type II : Cytotoxic reaction → Purpura
3. Type III : Immune Complex Reaction → Vasculitis
4. Type IV : Cell mediated Hypersensitivity
    - Maculopapular rash
    - Lichenoids
    - DRESS
    - SJS / TEN
    - Dermatitis
    - Erythroderma.
    - FDE.

active space

CLINICAL PATTERNS OF ADR

01:45

- Exanthematous reactions
- Urticaria, Angioedema
- Anaphylaxis
- Fixed Drug Eruption
- Lichenoid eruption
- Photosensitivity reactions
- Acneiform eruptions
- Erythema multiforme
- Psoriasiform rash
- Pigmentation
- Hair loss
- (MC) Type :

  i) Exanthematous ⟶ Maculopapular rash.

  ↓

  - Itchy
  - H/o Drug
  - Truncal and Acral involvement

  ii) Urticarial.

Severe Cutaneous Adverse Drug Reaction (SCAR)

- Steven Johnson Syndrome
- Toxic Epidermal Necrolysis (TEN)
- Drug Hypersensitivity Reaction (DHS/DRESs)
- Acute Generalised Exanthem Pustulosis (AGEP)
- Exfoliative dermatitis
- Serum Sickness like Rash.

active space

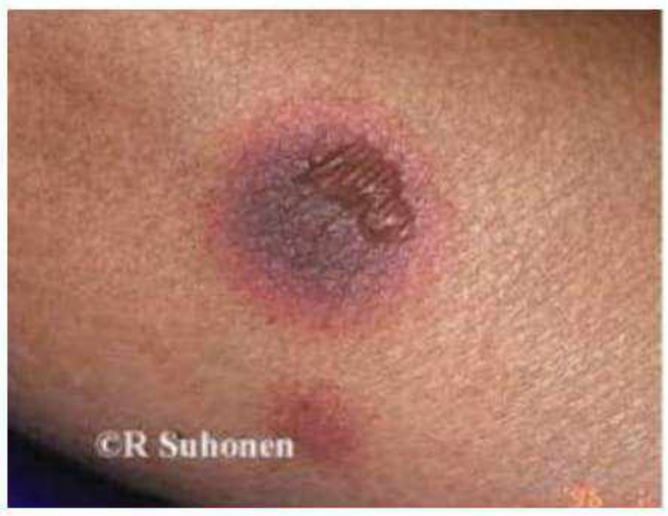
FIXED DRUG ERUPTIONS

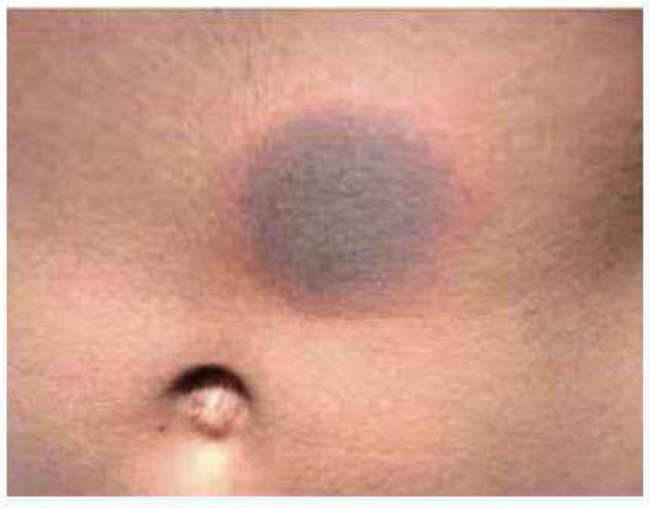
03:33

- Type IV HS
- Onset : 1st time → 7-10 days, on subsequent exposure within 30 min to few hours.
- Characteristic : Recurs at same site, Same morphology.
- Sites : Hands, feet, trunk, genitalia.
- Pathogenesis : Memory T cells ⊕
- Drugs that cause are :

  i) Cotrimoxazole
  ii) NSAID's
  iii) Tetracyclines.

  } (MC)

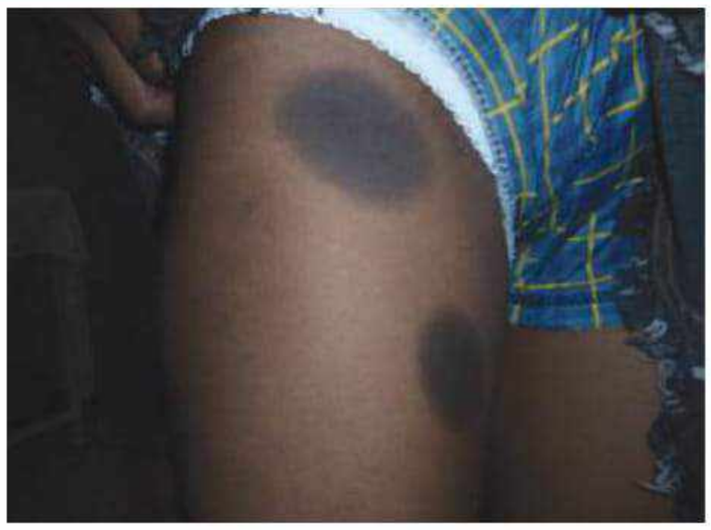


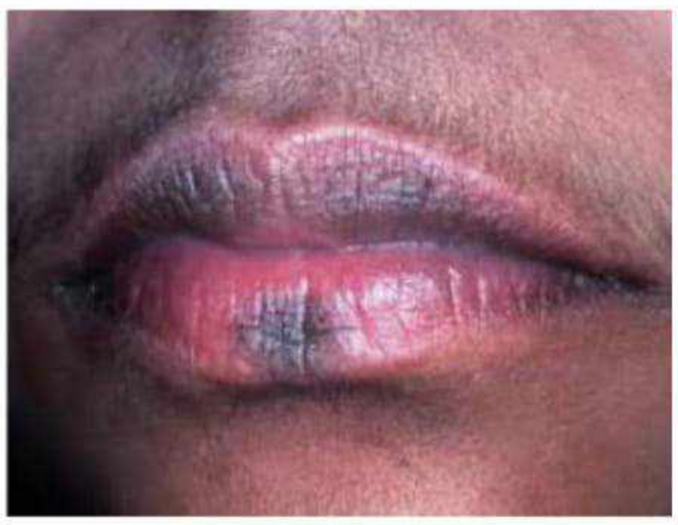


- well defined, circular lesion (plaque)

Erosions

active space





Lesions heal with hyperpigmentation.

Treatment :

- Avoid drugs
- Topical steroids.

ERYTHEMA MULTIFORME

08:18

Etiology

- 90% → HSV
- Mycoplasma
- Drugs : i) Sulphonamides
  ii) Tetracyclines
  iii) NSAID's
  iv) Anticonvulsants.
- Onset : Starts within 72hrs, heals in 1-2 wks

- Distribution : Acral (Hands and feet, Palmar > Dorsal aspect)

Mucosal involvement ⊕

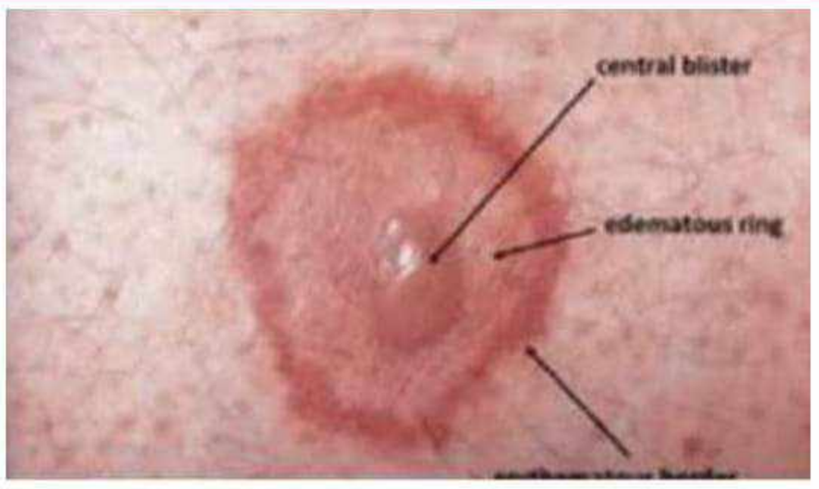
Clinical features

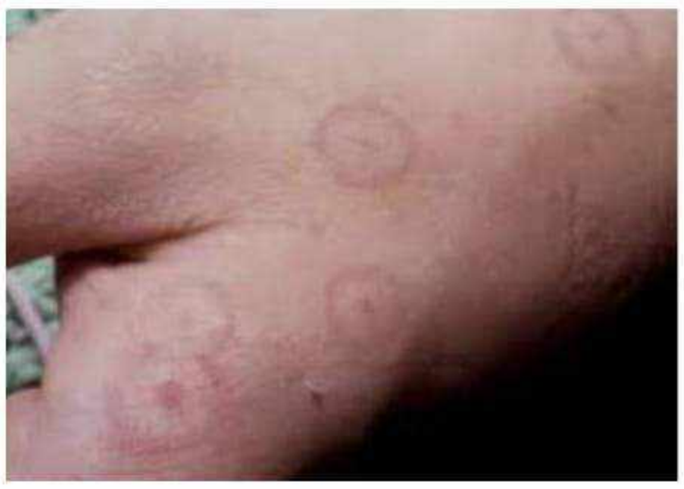
- Mild prodrome following which patient develops classical skin lesion.
- Target lesion : 3 zones ⊕
  - Central - purpura/necrosis
  - Middle - Edema
  - Peripheral - Erythema.

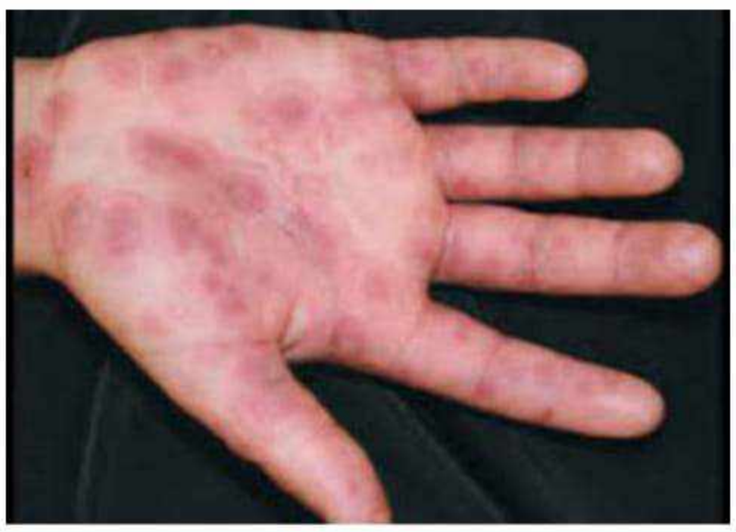
(Targetoid lesions when 2/3 zones are present).

- Oral mucosal lesions :

Hemorrhagic crusts are seen.







Treatment

- Stop the offending drug
- Topical corticosteroids. (cs)
- Oral cs
- Oral Acyclovir → in Recurrent EM → used as Prophylaxis.

STEVEN JOHNSON SYNDROME AND TEN 14:00

- TEN → Toxic Epidermal Necrolysis
- SJS/TEN are Severe Cutaneous Adverse Reactions, which involves skin, mucosa and systemic complications.
- Body surface Area involved :
  - < 10% → SJS
  - 10-30% → SJS-TEN Overlap
  - > 30% → TEN.

Triggers

- Drugs (85% cases) - Idiosyncratic, not dose dependent.

  i) Short term : Sulfonamides, Penicillins, Quinolones, Cephalosporins, Acetaminophen, NSAID's.

active space

ii) Longer periods:

- Carbamazepine, phenobarbital, phenytoin, Valproic acid, Oxicam group of NSAIDs, Antitubercular drugs, Anti-retroviral agents and allopurinol

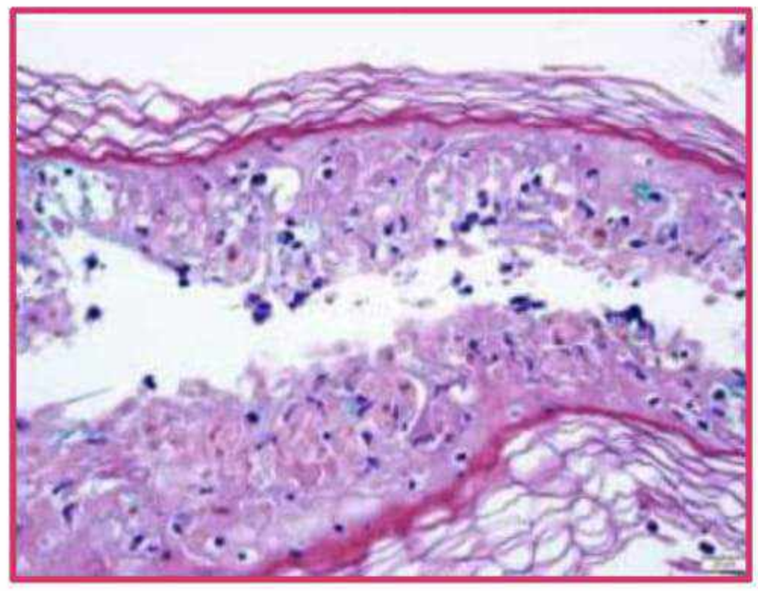
- Mycoplasma pneumonia
- Food additives, infections, Malignancies
- Genetics:
  - HLA-B* 1502 → associated with carbamazepine
  - HLA-B* 5801 → associated with allopurinol

Pathophysiology

- Keratinocyte apoptosis and necrosis
- Cytotoxic T cell lymphocytes CTCL's
- Fas ligands
- Perforins
- Granulysin
- Granzyme

active space

On Histopathology:

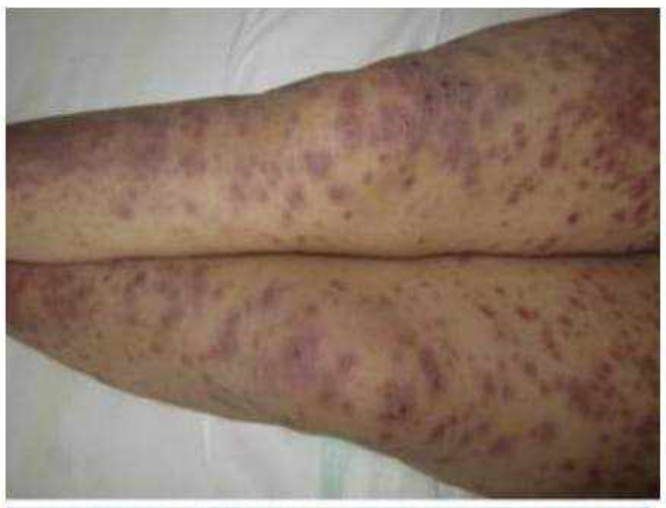


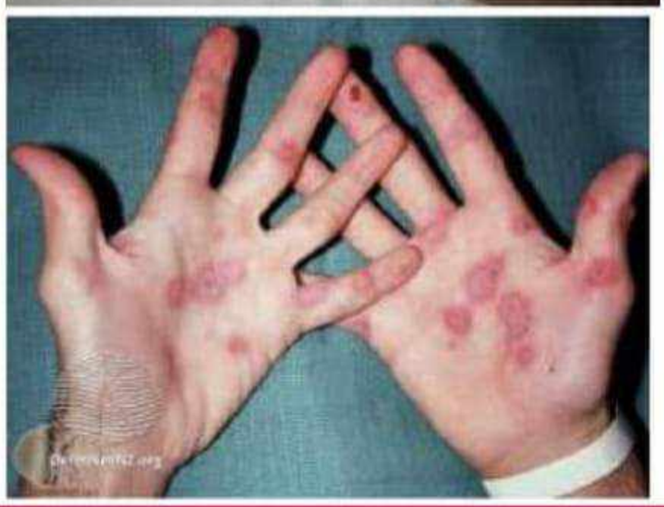
- Keratinocyte necrosis and apoptosis
- Interface dermatitis

- Latent period : 7-10 days (5-28 days)

Clinical features:

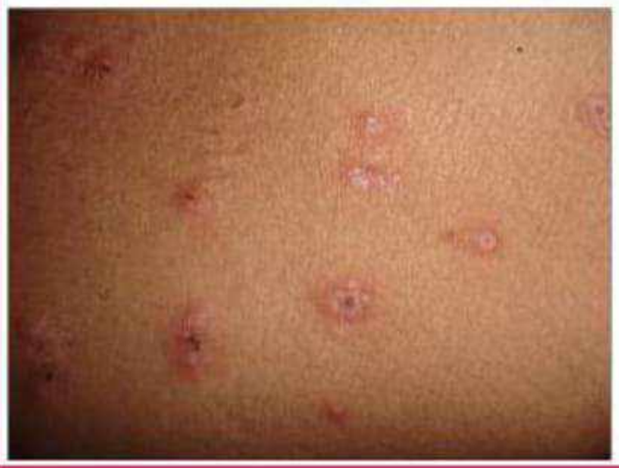
- Prodrome ⊕
  - Fever, Malaise, Constitutional symptoms.
- Pain, Pruritus
- Site : face and upper trunk ⟶ Progress to involve whole body.



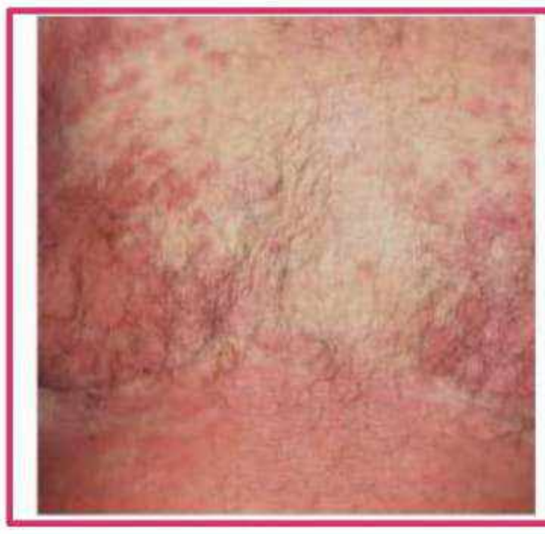


- Purpuric and targetoid lesions
  ↓
  gradually spreads and involves hands and feet as well.

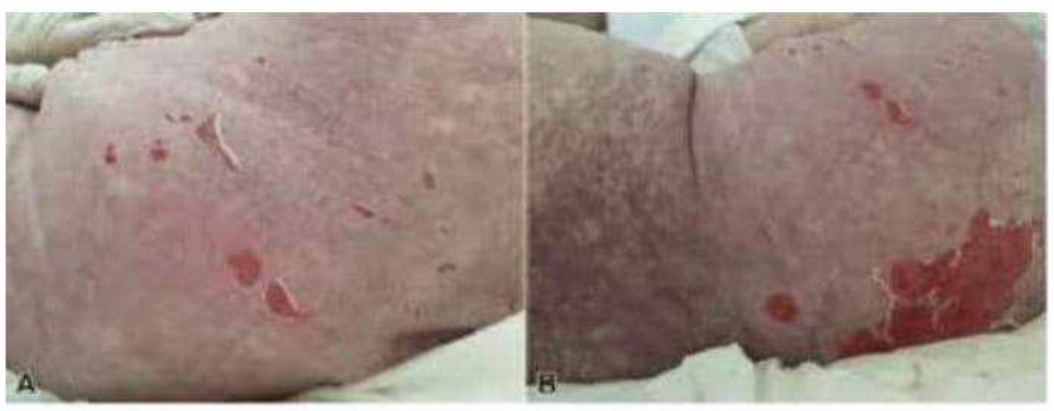
active space



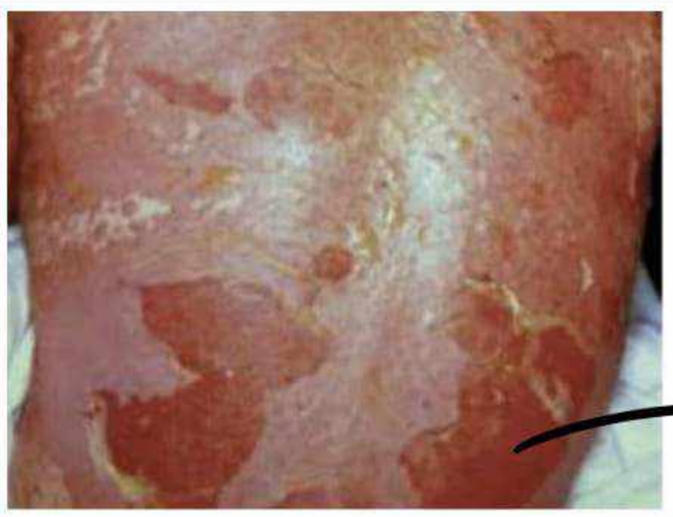
- develop vesicles / bullae on the centre of lesion



- Confluent erythema



- skin peeling at sites of friction.



- peeling extends
- sheets of Peeling

→ Raw areas on body.

active space

- **Mucosal involvement ⊕ :** ≥ 2 mucosa involved.

↓

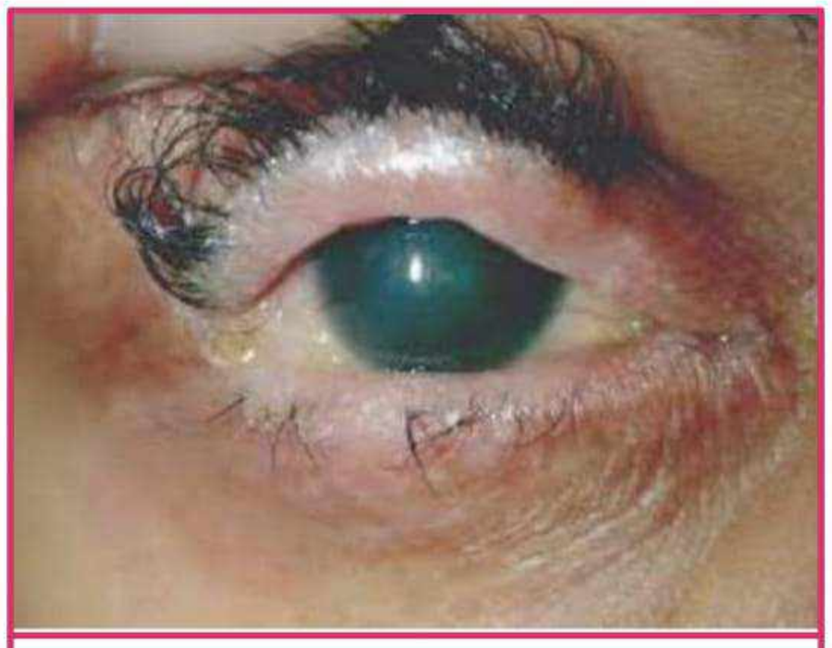
Mucosa involved are :

i) Oral

ii) Conjunctival

iii) Genital

iv) Perianal.



- Discharge
- Membrane formation
- Conjunctivitis

Complications

- Hypothermia
- Fluid imbalance
- Hypoalbuminemia

- Anemia , Leucopenia
- Bronchial erosions and airway obstruction
- Septicemia → Major cause of mortality
- Multiorgan failure

SJS - 5%
TEN - 30%
} Mortality

SCORTEN parameters	Score
Age > 40 years	1
Malignancy	1
Tachycardia (>120/min)	1
Initial surface of epidermal detachment >10%	1
Serum urea >10 mmol/l	1
Serum glucose >14 mmol/l	1
Bicarbonate >20 mmol/l	1
SCORTEN Score	**Predicted mortality (%)**
0-1	3.2
2	12.1
3	35.8
4	58.3
≥5	90

SCORTEN → predicts mortality

Treatment

- Supportive treatment
- Stop drug

active space

- Supportive treatment includes :
  - Fluids
  - Temperature
  - Dressings.
  - Proteins
- Specific Therapies :
  - iv IgG
  - Cyclosporin
  - Steroids (in severe cases)

DRESS

28:05

- Drug rash Eosinophilia Systemic Symptoms
- a.k.a Anti convulsant Hypersensitivity Syndrome (AHS) / Drug Hypersensitivity Syndrome.
- Drug Rash : Maculopapular Rash to even SJS/TEN
- Eosinophilia : Abnormal lymphocytes
- Systemic Symptoms : Lymphadenopathy, Facial swelling. Hepatitis.
- (MC) cause → Anticonvulsants.

active space

ANAPHYLAXIS

30:00

- Its an acute emergency
- IgE mediated disorder.

Clinical features

- skin → Urticaria, Angioedema
- Bronchospasm
- Abdominal cramps

Causes :

- Sulphonamides
- Penicillins
- Radiocontrast dyes.

ACUTE GENERALISED EXANTHEMATOUS PUSTULOSIS

31:20

- a.k.a AGEP
- non-follicular eruption of pinpoint pustules over trunk
- Drugs involved are :
  - Tetracyclines
  - NSAIDs
  - Antibiotics.

active space

ERYTHRODERMA 32:30

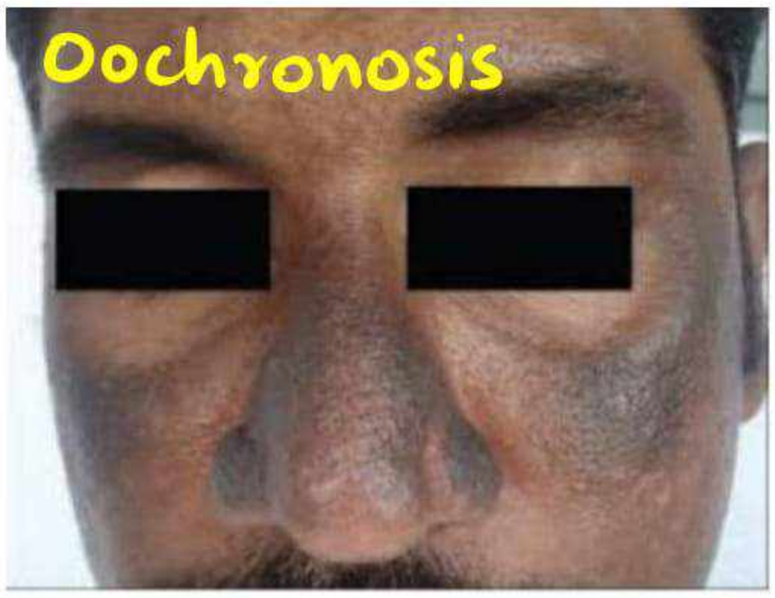
- Erythema and scaling >90% BSA
- **Drugs which cause :** HCQ's, Anticonvulsants.

DRUG INDUCED PIGMENTATION 33:20

1. Minocycline

- Black Blue pigmentation on the scars of acne.
- Deposition of Iron chelates of Minocycline.
- also cause Lupus like features
- Drug Reaction ⊕

2. Oochronosis :



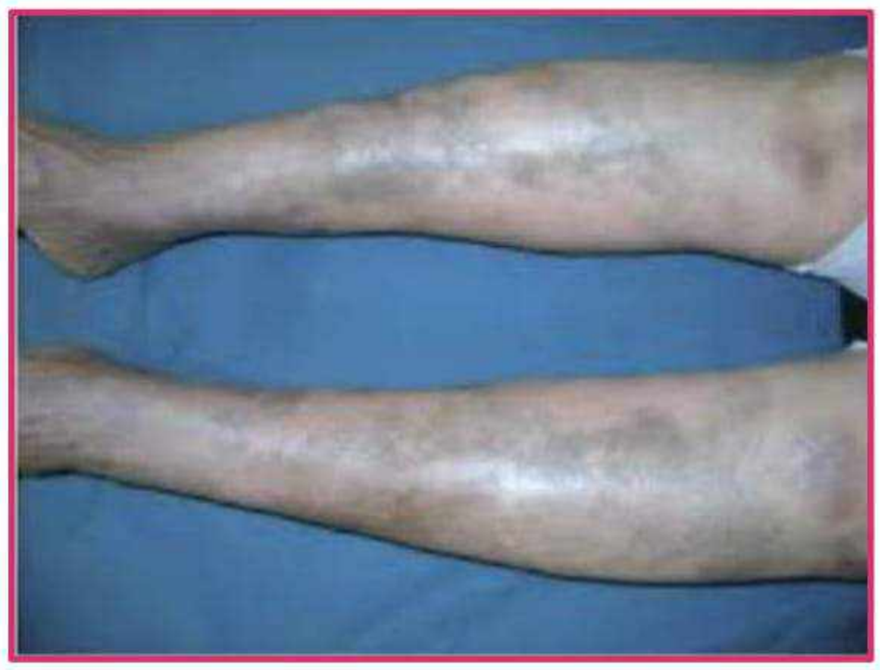
- speckles dark brown bluish pigmentation + atrophic depigmented macules.
- **Causes :**
  - i) **Endogenous :** Alkaptonuria
  - ii) **Exogenous :** Hydroquinone.

active space

- In Alkaptonuria ⟶ Inhibition of Homogentisic oxidase
  ↓
  Accumulation of Homogentisic acid.
- Ochre shaped / Banana shaped bodies are seen on Biopsy.

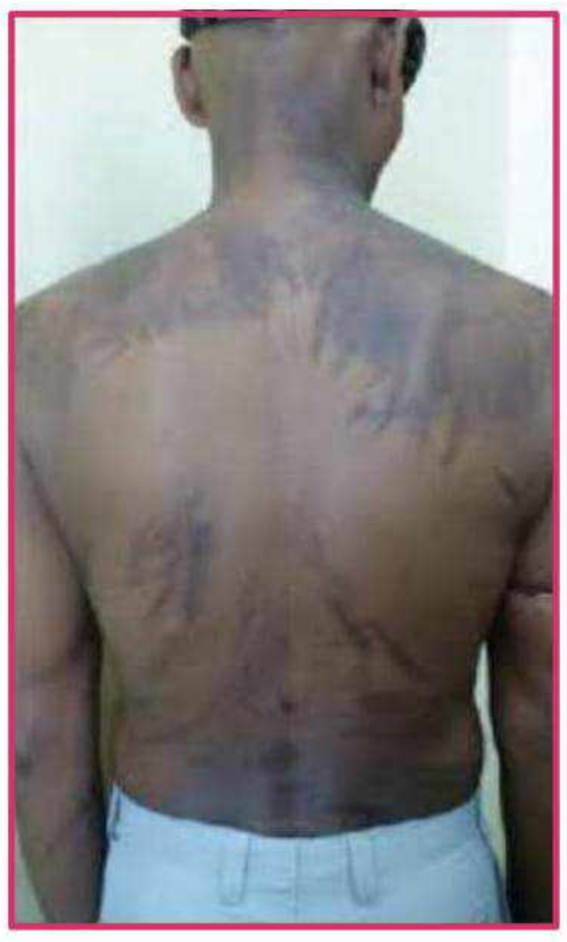
3. Hydroxychloroquine

- Blue Black pigmentation on shin of tibia and other body parts.



4. Bleomycin

- Cause flagellate pigmentation.
- also gives rise to flagellate erythema.



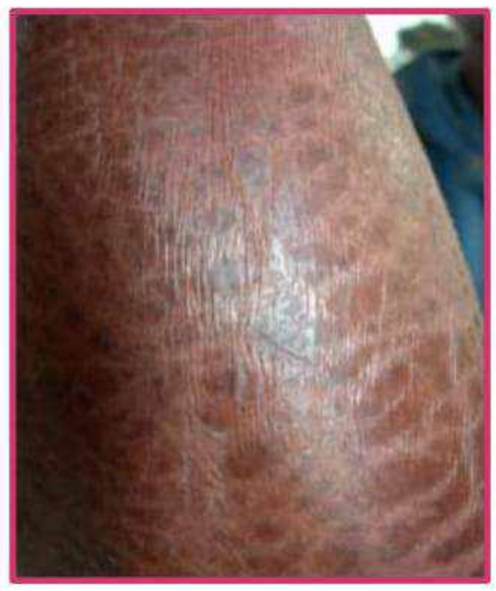
active space

5. Zidovudine

- cause pigmentation of hands and body
- Melanonychia → pigmentation of nails.

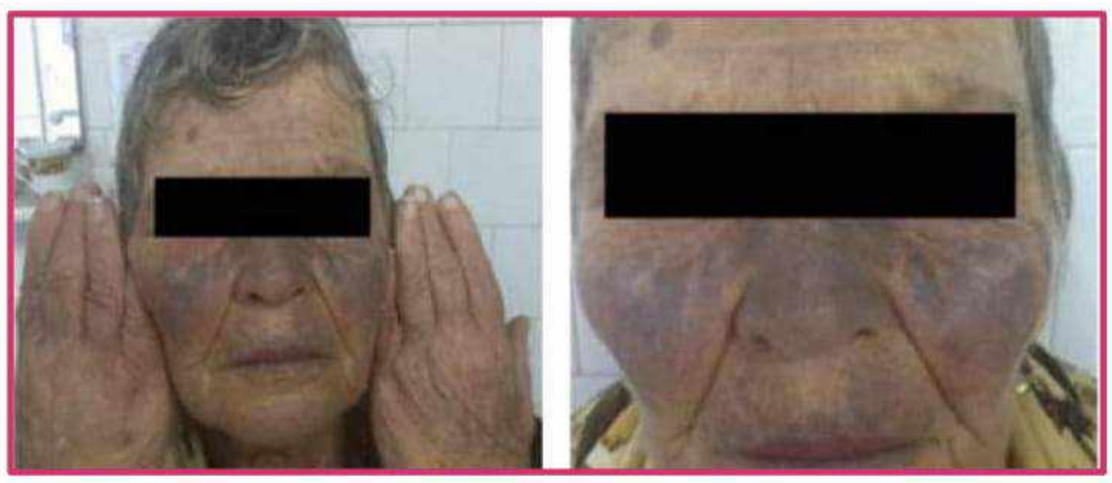
6. Clofazamine

- orangish to reddish discolouration of skin + Icthyosis



7. Amiodarone

- Photosensitivity ⊕
- Pigmentation on photoexposed areas.



active space

Skin Tumours (Part - 1)

Skin Tumors :

- It can be i) Benign
  ii) Premalignant
  iii) Malignant

Tumors Arising from Epidermis

Benign	Pre-Malignant	Malignant
• Seborrheic keratosis	• Actinic keratoses	• Squamous cell Carcinoma
• Sebaceous cyst	• Leukoplakia	• Basal cell Carcinoma
• Steatocystoma complex	• Cutaneous horn	
	• Bowen's disease	• Paget's disease.

Tumors of Epidermal Appendages

Benign	Malignant
• Trichoepithelioma	• Basal cell carcinoma.
• Syringoma	

Tumors arising from Melanocyte : Malignant Melanoma.

Tumors of Mesodermal Origin

Connective Tissue

- Dermatofibroma
- Acrochordon (skin tag)
- Keloid

Vascular Tumors

- Hemangioma
- Angiokeratoma
- Glomus tumor.

Other Cutaneous tumors

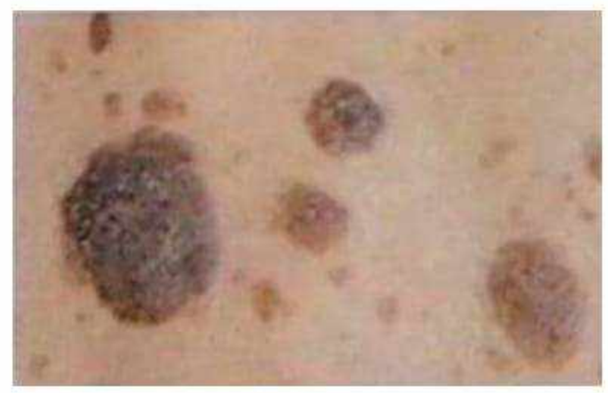
i) Lymphomas and leukemias :

- Cutaneous T-cell Lymphomas : Mycosis fungoides, Sezary Syndrome.

BENIGN KERATOLYTIC PROLIFERATIONS

02:30

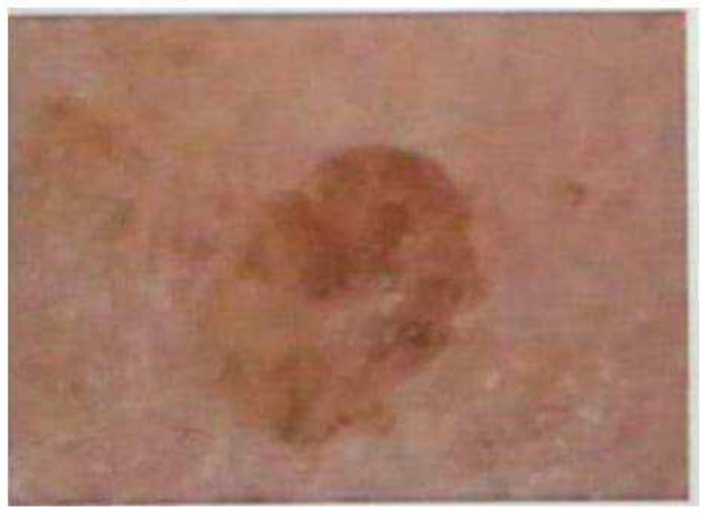
1. Seborrhoeic Keratosis



- Benign proliferation of keratocytes
- usually seen on photo exposed parts.

active space

- (MC) seen on face, dorsa of hands
- More commonly seen in elderly



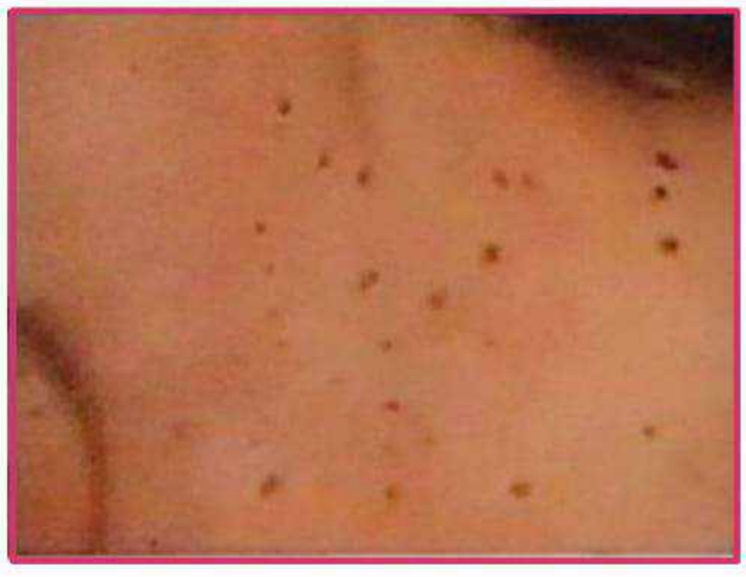
Clinical feature

- Mildly to deeply pigmented plaques with flat surface showing **Stuck on Appearance.** (Follicular plugs)
- **Sign of Leser-Trélat :**
  - Sudden appearance of numerous SK's on body.
  - might be associated with underlying malignancy.
    - ↓
    - Gastric cancer, Colon cancer

DERMATOSIS PAPULOSA NIGRA

06:40

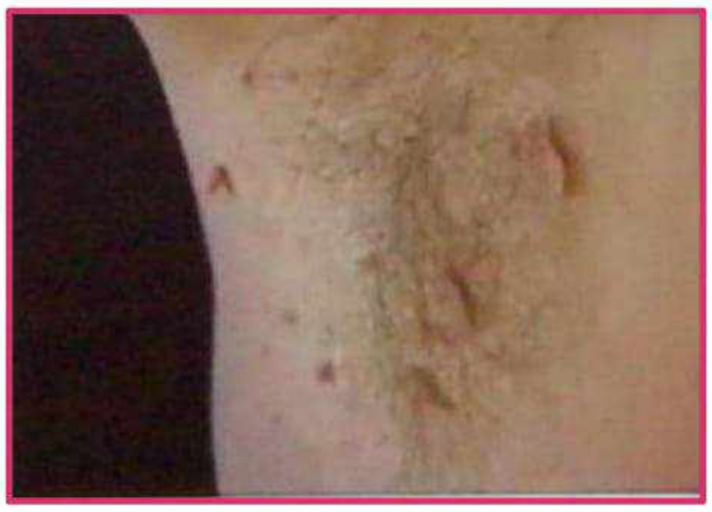
- variant of SK
- usually seen in Dark skinned people
- Pigmented papular lesions
- seen on face and neck.

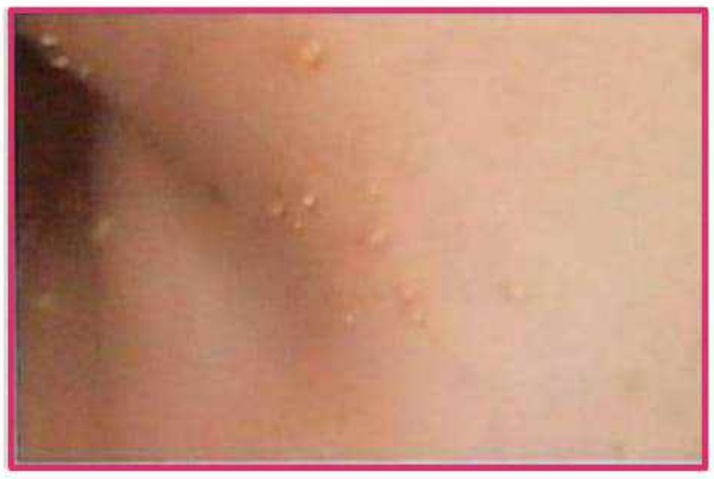


active space

SKIN TAGS

07:25





- a.k.a **Acrochordons**
- Benign Proliferation of loose fibrous tissue.
- Pedunculated
- Skin coloured to lightly coloured lesions.
- (MC) located on → Flexures
  - Neck
  - Axilla
  - Groins.
- **Associations :** Obesity, DM, Acanthosis nigrican.

CUTANEOUS CYSTS

09:30

- Divided into :

i) Sebaceous → True sebaceous globules are present

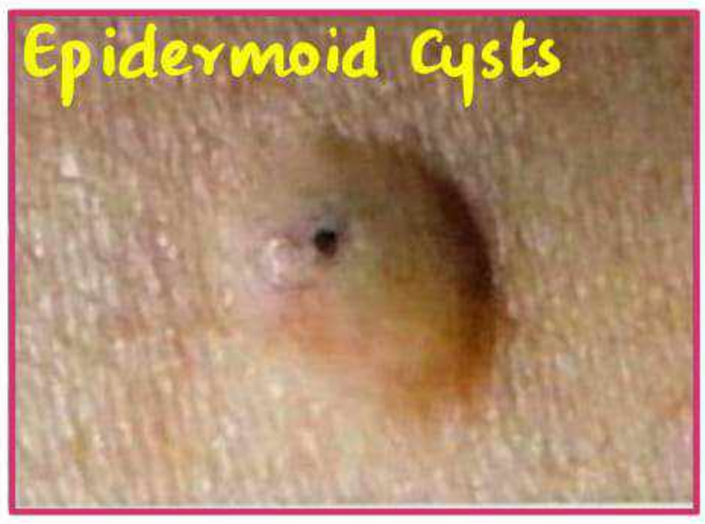
ii) Epidermoid → Epidermoid

iii) Trichilemmal. } Keratinous origin

Seen on Scalp. (lining External Root Sheath)

active space

Epidermoid Cysts



- a.k.a Epidermal Inclusion Cysts
- Not sebaceous cysts.
- (MC) common cysts
- Cysts which contain keratinous material
- Association : Gardner's syndrome, Nevoid form Basal Cell Carcinoma.
- Pathogenesis : Inflammation around pilosebaceous follicles.
  - seen in Acne vulgaris.
- Location : upper trunk and face

Clinical feature

- protuberant soft swellings with adherent epidermis.
- Dome shaped swellings
- Central Keratin plug.
- Suppurate and Rupture → Releasing foul smelling keratinous content
- May get 2° infected

active space

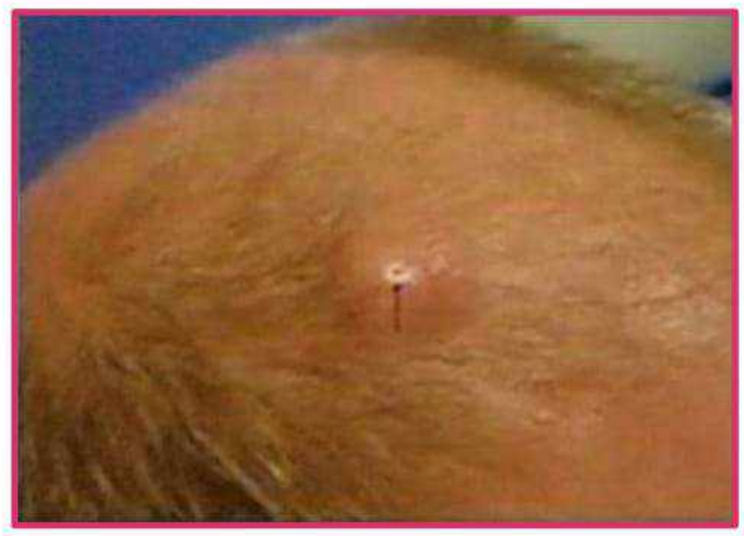
Treatment

- if Asymptomatic → No Rx
- if 2° Infection ⊕ → Give Antibiotics.
- Cyst excision.

TRICHILEMMAL CYSTS

15:30

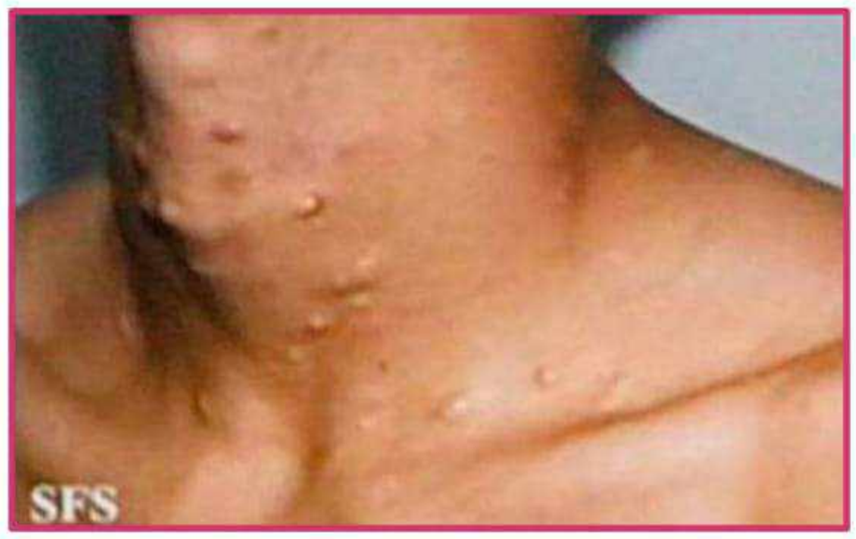
- No punctum
- seen on hair bearing areas
- (MC) seen on Scalp.
- lined by External Root Sheath.



STEATOCYSTOMA MULTIPLEX

16:20

- a.k.a True Sebaceous cyst
- Autosomal Dominant Condition
- Site : upper trunk, proximal aspect of arms
- Pathology : Sebaceous globules and sebum.



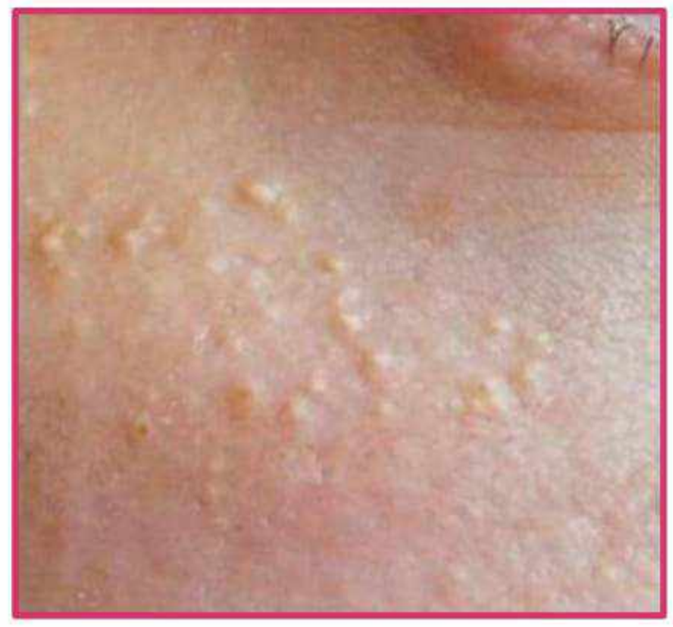
Clinical feature

- Multiple dermal swelling with no punctum.
- Yellowish hue ⊕

MILIUM / MILIA

17:45

- Benign keratinous cyst
- seen on face : areas of vellus hair ↘
  cheeks, periorbital
- White small papules with no umbilication.
- can occur 2° to burns, Bullous disorders. Radiation.



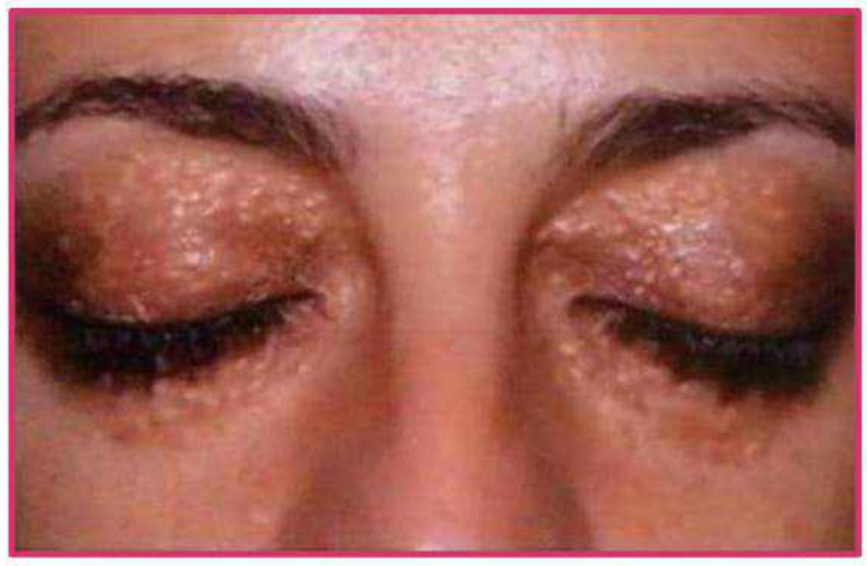
SYRINGOMA

19:25

- Benign tumor of Eccrine sweat gland (Appendages)
- origin → Acrosyringium of eccrine sweat gland.
- On HPE : Tadpole / Comma shaped appearance.
- Site : (MC) seen over Periorbital areas.

Clinical feature

- Translucent papules grouped
- Bilateral
- seen in periorbital areas.
- Angular margins.
- On Rupture → Watery secretions ⊕

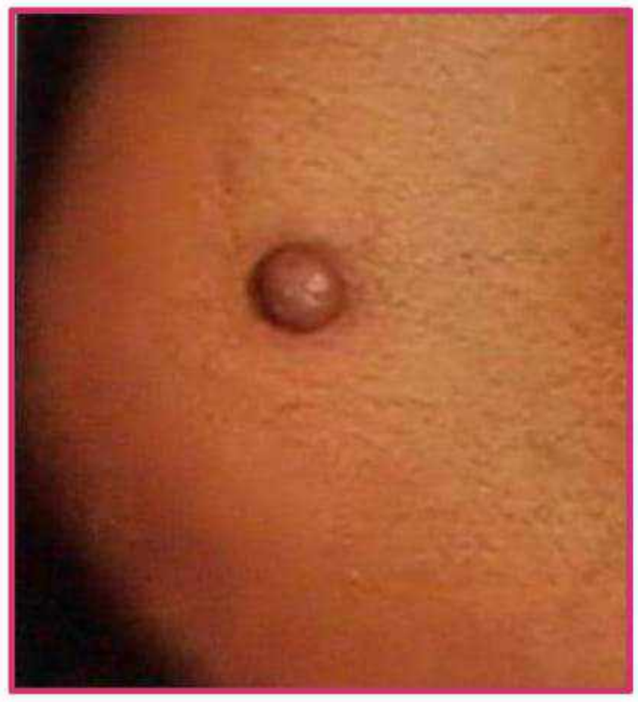


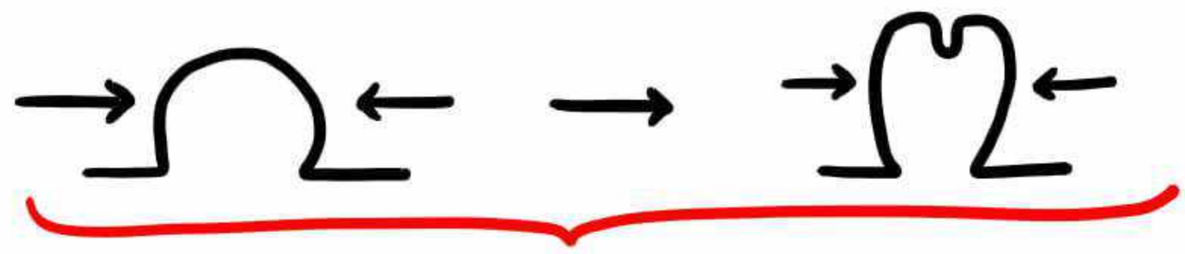
active space

DERMATOFIBROMA

21:45

- Benign fibrous Histiocytoma
- Round to oval pedunculated soft swellings
- more commonly seen on lower legs
- Mobile on the base
- On lateral pressure → Central Dimple can be seen.



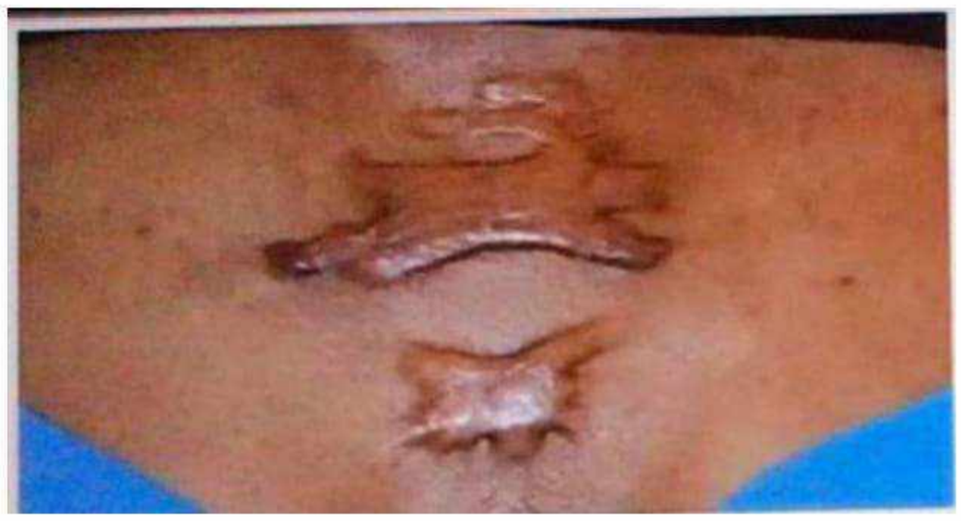


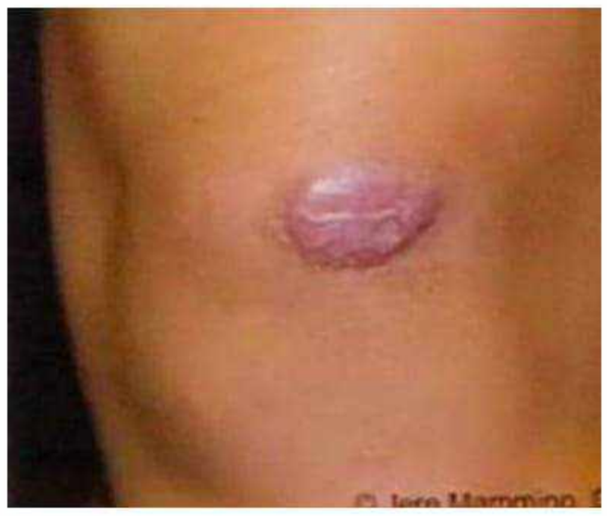
" DIMPLE SIGN "

KELOID

23:17

- chele = Crab's claw, and -oid = like.
- area of overgrowth of fibrous tissue that usually develops after healing of a skin injury and extends beyond the original defect.
- HLA Association
- Autosomal Dominant inheritance
- Site : Pre-sternal areas, ears, upper backs.
  ↳ Dumb bell configuration.





Keloid

- Keloid will extent beyond the margin of original pathology
- Keloid doesnot resolve spontaneously
- Surgery → ↑ Keloid.

vs Hypertrophic scar

- limited to original margin
- it usually resolves in 1-2 yrs
- Surgical excision can be done.

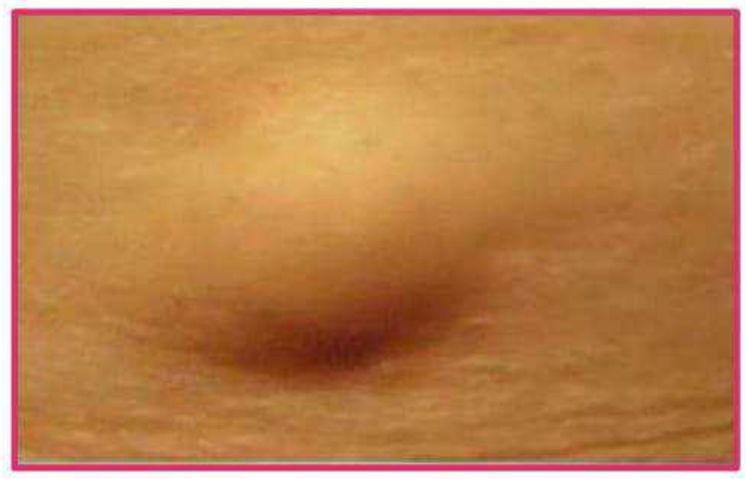
Treatment

- Intra lesional steroids
- Intra lesional Fluorouracil.
- Silicon compression
- Bleomycin

active space

LIPOMA

26:50



- Benign tumor of mature adipose tissue
- S/c freely mobile soft swelling, compressible
- seen commonly in obese individuals

NON MELANOMA SKIN CANCER

27:50

Skin cancers → Melanomas

→ Non-Melanomas (NMSC)

↓

- BCC
- SCC.

}
- Both are keratinous in origin.
- Less tendency for invasion and metastasis.

Squamous Cell Carcinoma and its precursors

1. Actinic keratoses

- (MC) premalignant lesion
- Predisposing factors: i) Elderly
  ii) White population
  iii) Exposed to UV Radiation.

active space

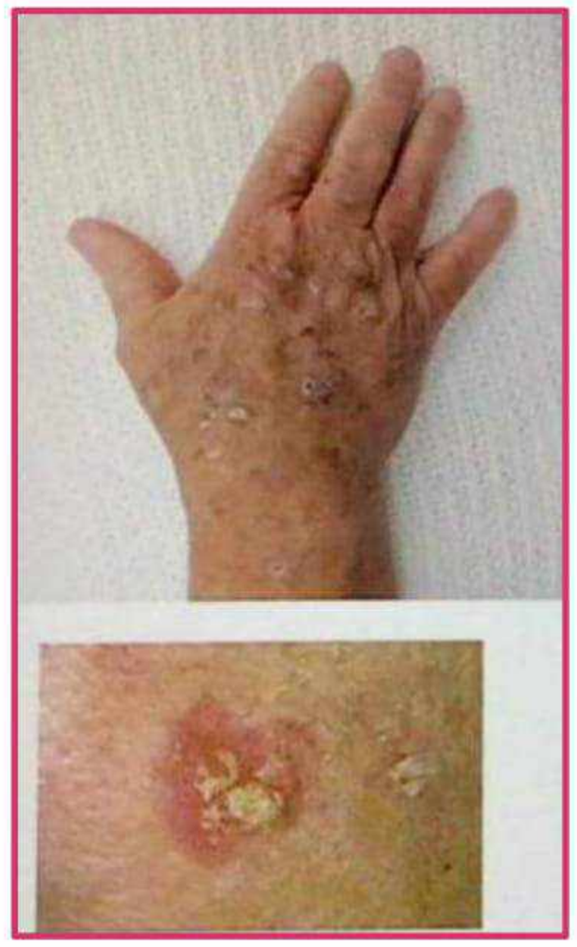
- **Pathology :**
  - Cytological atypia
  - disordered differentiation
  - Parakeratosis, Hyperkeratosis, Hypogranulosis

Clinical feature

- usually seen on exposed parts of body - face, dorsa of hands.
- slightly raised asymptomatic plaques with whitish to yellowish adherent crust.

↓

Pinpoint Bleeding points on Removal of crusts.



Treatment

- Photodynamic therapy
- 5-FU
- Imiquimod
- Cryotherapy
- Curettage

active space

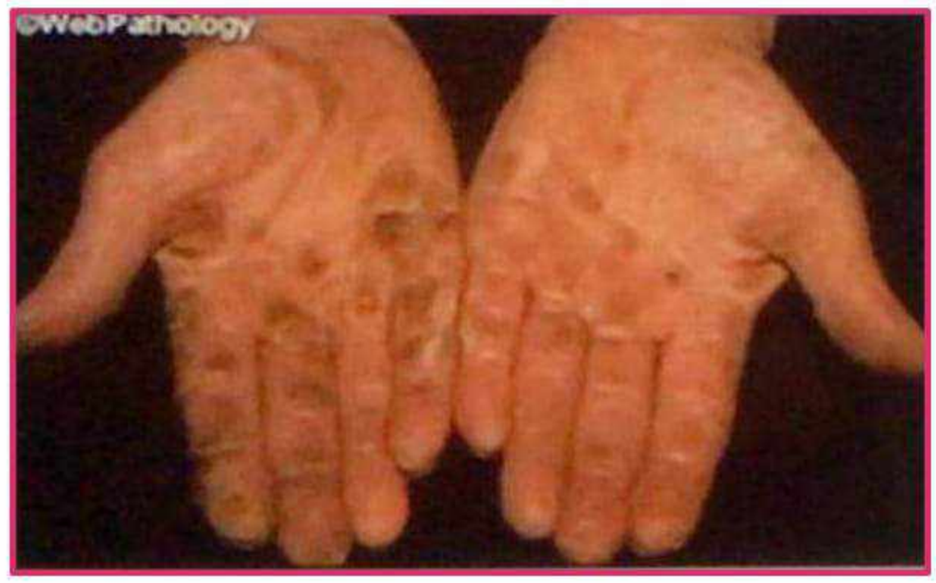
ARSENICAL KERATOSES

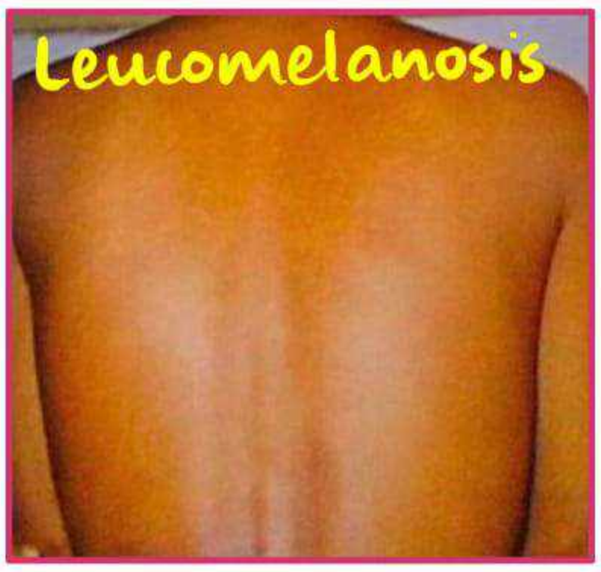
33:53

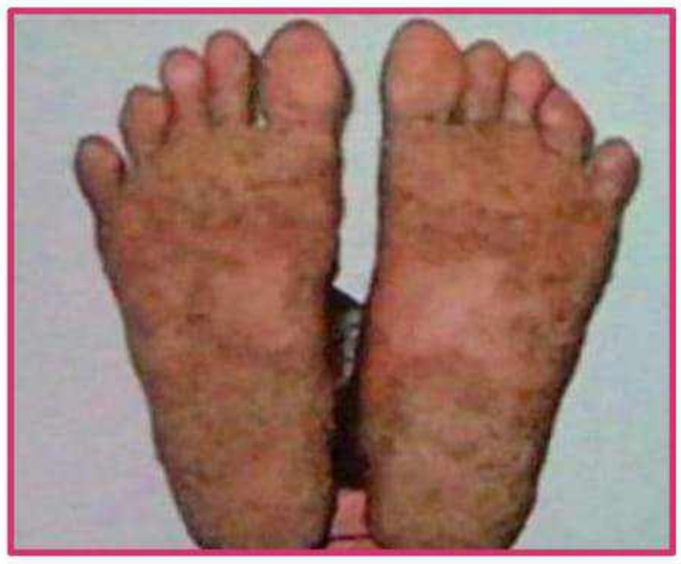
- it is caused by Arsenic (present in contaminated drinking water)

Clinical feature

- corn like punctate keratoses on palms and soles.







Treatment

- Photodynamic therapy
- 5-FU
- Imiquimod

active space

CUTANEOUS HORN

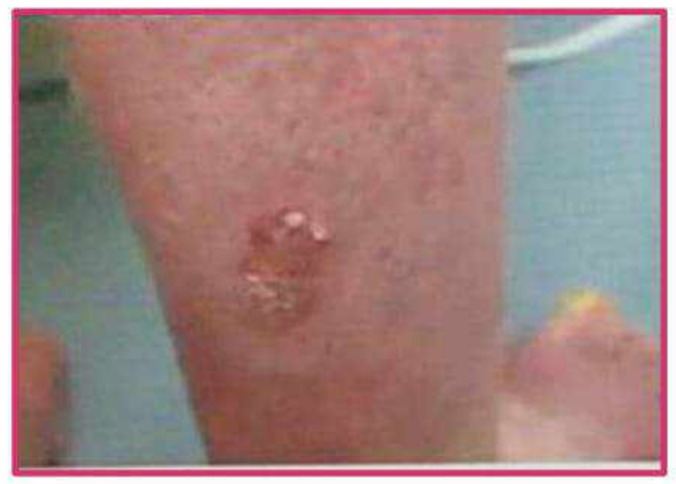
36:15

- Hard conical projection usually present on photodamaged skin.
- pre-malignant

BOWEN'S DISEASE

36:50

- Intra-epidermal (in-situ) SCC
- small potential for invasive malignancy
- Population affected : White elderly, sun exposed, More common in females.
- Predisposing factors : • Arsenic exposure
  • Immunosuppressant.
- On HPE :
  - full thickness epidermal dysplasia and disordered differentiation, keratinocytes show variable pleomorphism.
- Sites :
  - seen on lower legs of elderly females.

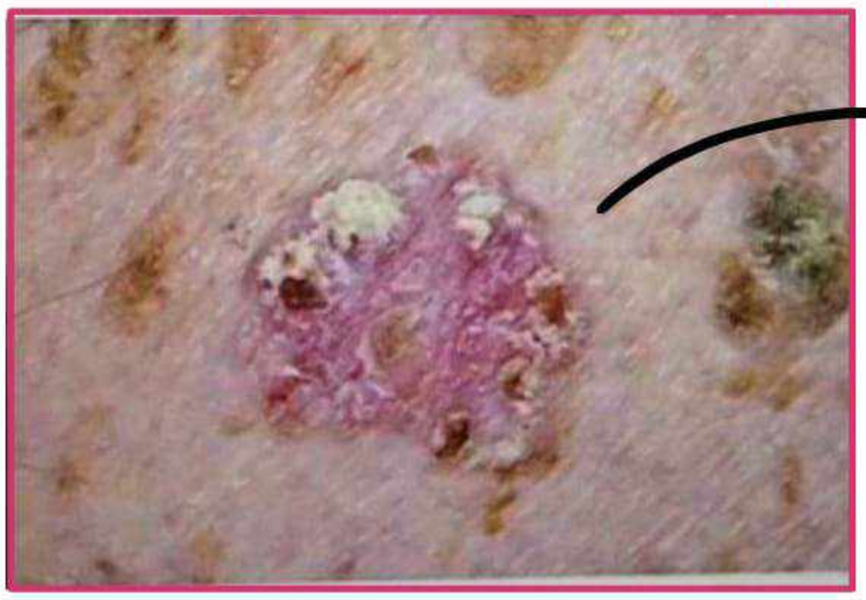


Clinical feature

- Begins with erythematous flat plaque

with irregular borders which are covered with scales or yellowish crusts.

- D/D → Inflammatory eczemas.



→ Adherent crust
↳ doesnot leave bleeding points on removal.

Management

- Biopsy should be done.
- Photodynamic therapy
- 5-FU
- Imiquimod
- Cryotherapy.

SQUAMOUS CELL CARCINOMA

41:08

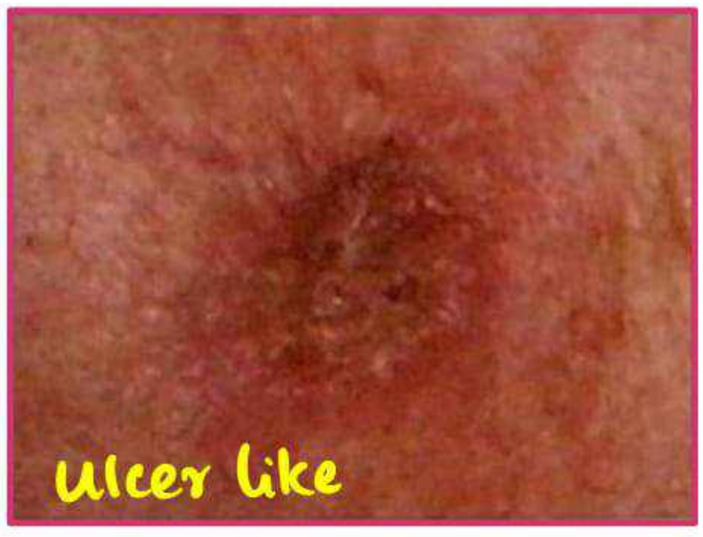
- Malignant tumor of keratinocytes or its appendages.
- Low potential for invasiveness / metastasis.
  ↳ if it happens → lymphatic spread.

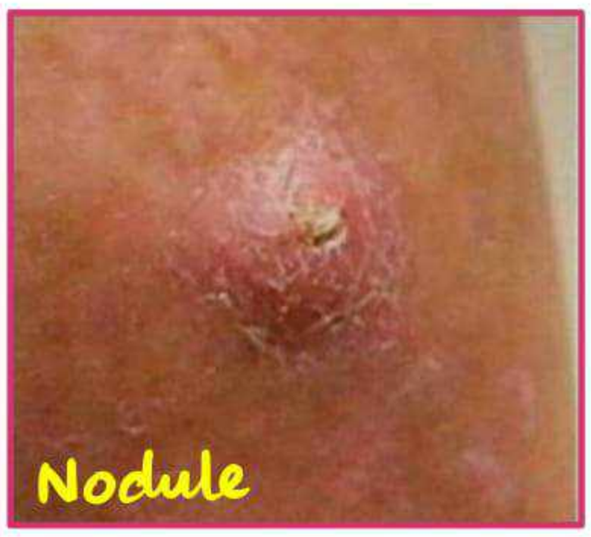
active space

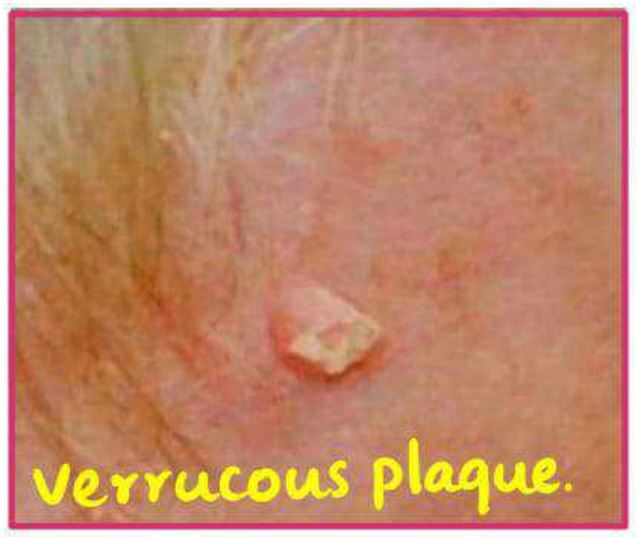
- SCC is 2nd most common skin cancer
- Predisposing factors :
  - i) UV Exposure
  - ii) Immunosuppression. → (MC) Skin cancer.
  - iii) Precursor lesions
  - iv) Albinism, Xeroderma pigmentosa.
  - v) Chronic DLE
  - vi) Burns.

Clinical features

- Sites: sun exposed sites.
- Surrounding areas of photodamage can be seen } Telangiectasias, atrophy, SK's.

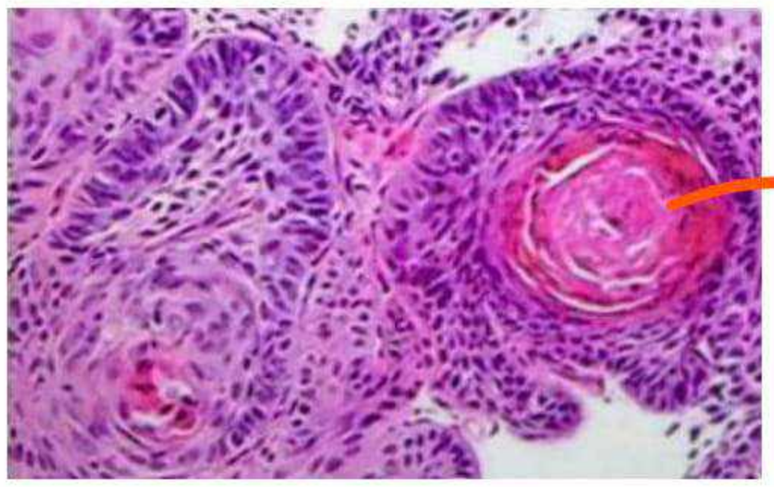

Ulcer like


Nodule


Verrucous plaque.

- presence of induration → Important sign for SCC.

- **On HPE :**
  - Invasion of dermis with keratinocytes



→ Keratin pearls.

- Prognosis : Has tendency for Regional and distant metastasis

Treatment

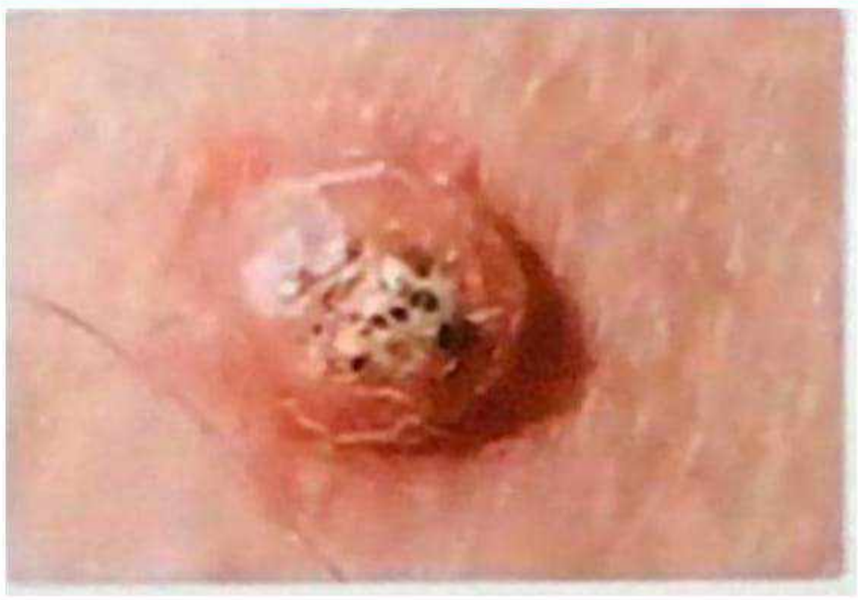
- Wide surgical excision with margins
- Moh's micrographic surgery.
- Radiation, Chemotherapy → in case of Metastsis, Invasion.

KERATOACANTHOMA

47:35

- Recently been reclassified as well differentiated SCC (Keratoacanthomatous type)
- Site : Face > Hands
- Phases : I → Growing phase
  II → Maturation phase.
  III → Resolution phase

- Can resolve spontaneously.



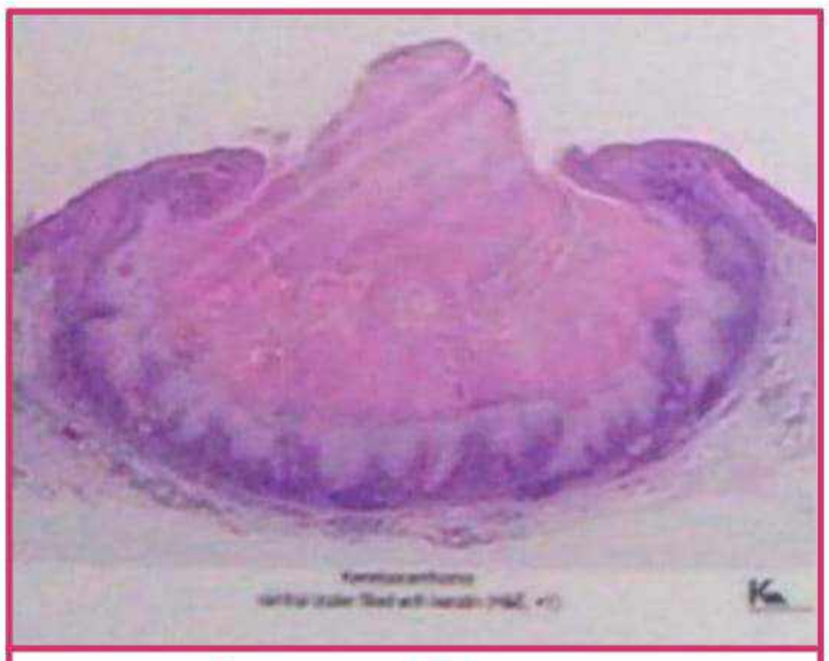
Round nodule (Dome shaped)

which has a central plug.

↓

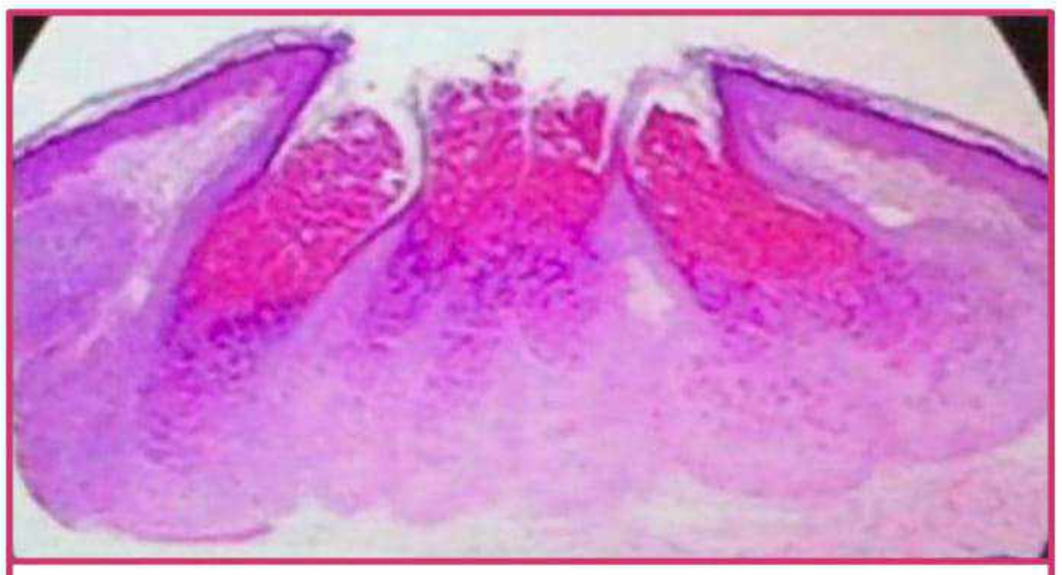
filled with keratin material.

- **On HPE :**



Keratoacanthoma

Epidermal lipping can be seen.

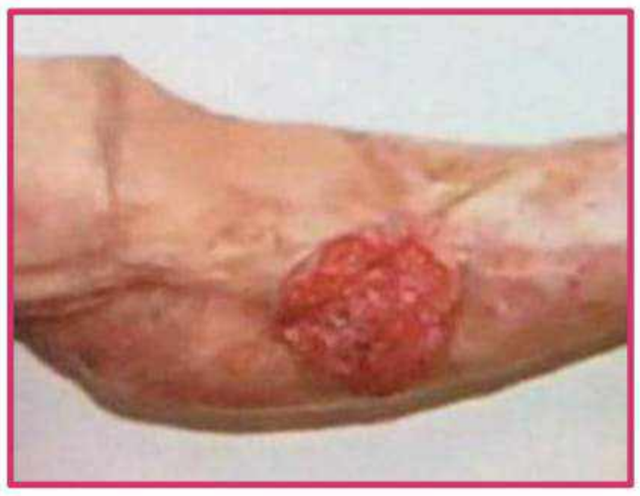


Molluscum contagiosum

MARJOLIN'S ULCER

51:15

- occurs at site of burns / scars.
- High tendency to develop into carcinoma.
  SCC > BCC
- It is Recurrent and has high invasive potential.



Skin Tumours (Part - 2)

BASAL CELL CARCINOMA

00:10

- (MC) Human cancer
- Slow growing, locally invasive tumor, rarely metastasizes.
- It arises from pluripotent cells within the basal layers of epidermis or follicular structures.
- Syn: Rodent ulcer (Basilioma)

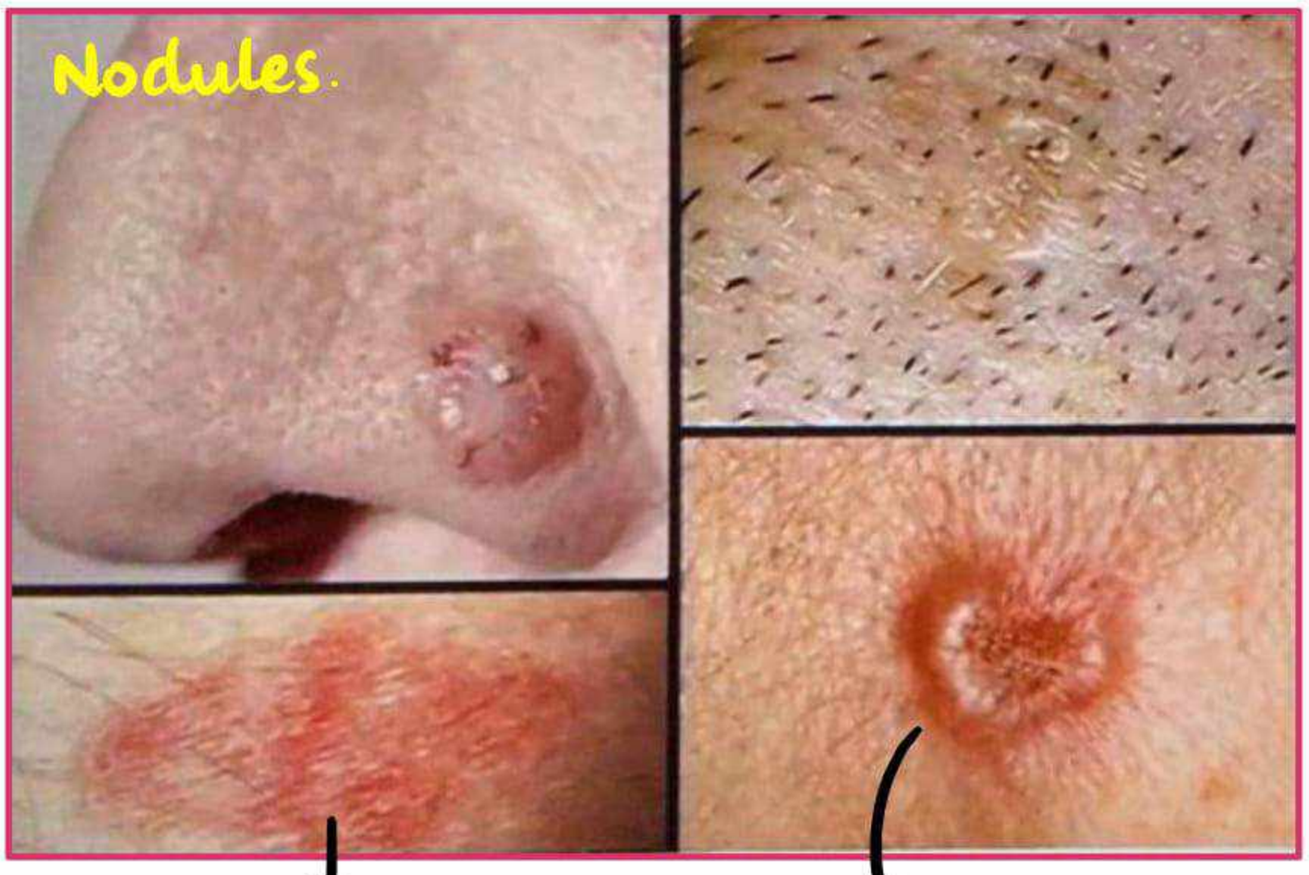
Box 141.1 Risk factors for basal cell carcinomas

Fitzpatrick skin type I and II
- Solar UV radiation → SCC > BCC
- Human papillomavirus
- Iatrogenic immunosuppression
- Acquired immunodeficiency syndrome and non-Hodgkin lymphoma
- PUVA therapy
- Photosensitizing drugs
- UVB phototherapy
- Ionizing radiation
- Occupational factors
- Arsenic exposure
- Previous history of basal cell carcinomas

Clinical features

- No recognized pre-malignant stage
- Slow progressive course of peripheral extension

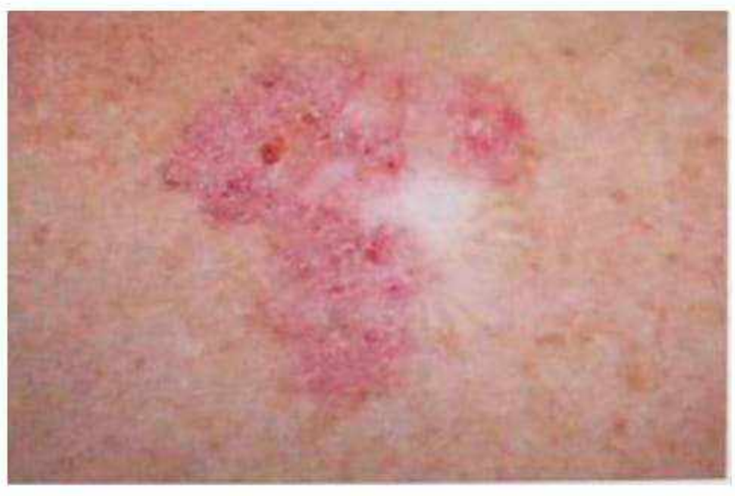
- Site : face (commonly seen around eyes, nose, upper cheeks)

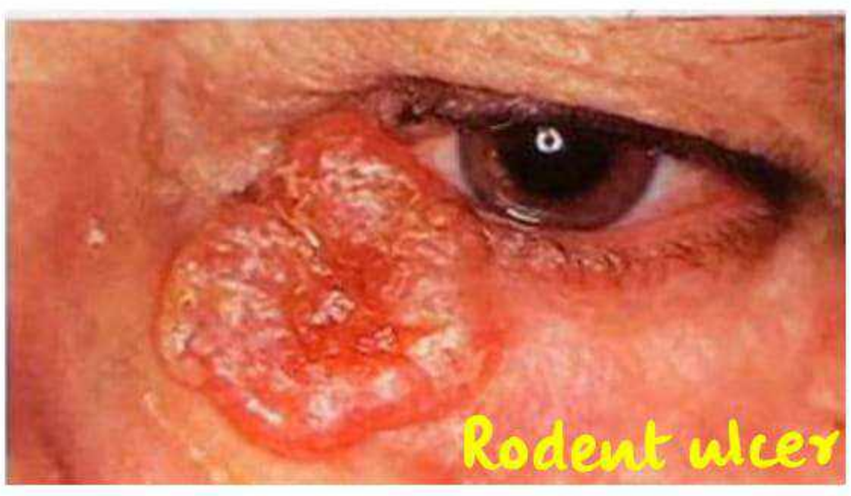


- Thready margin
- Telangiectasias

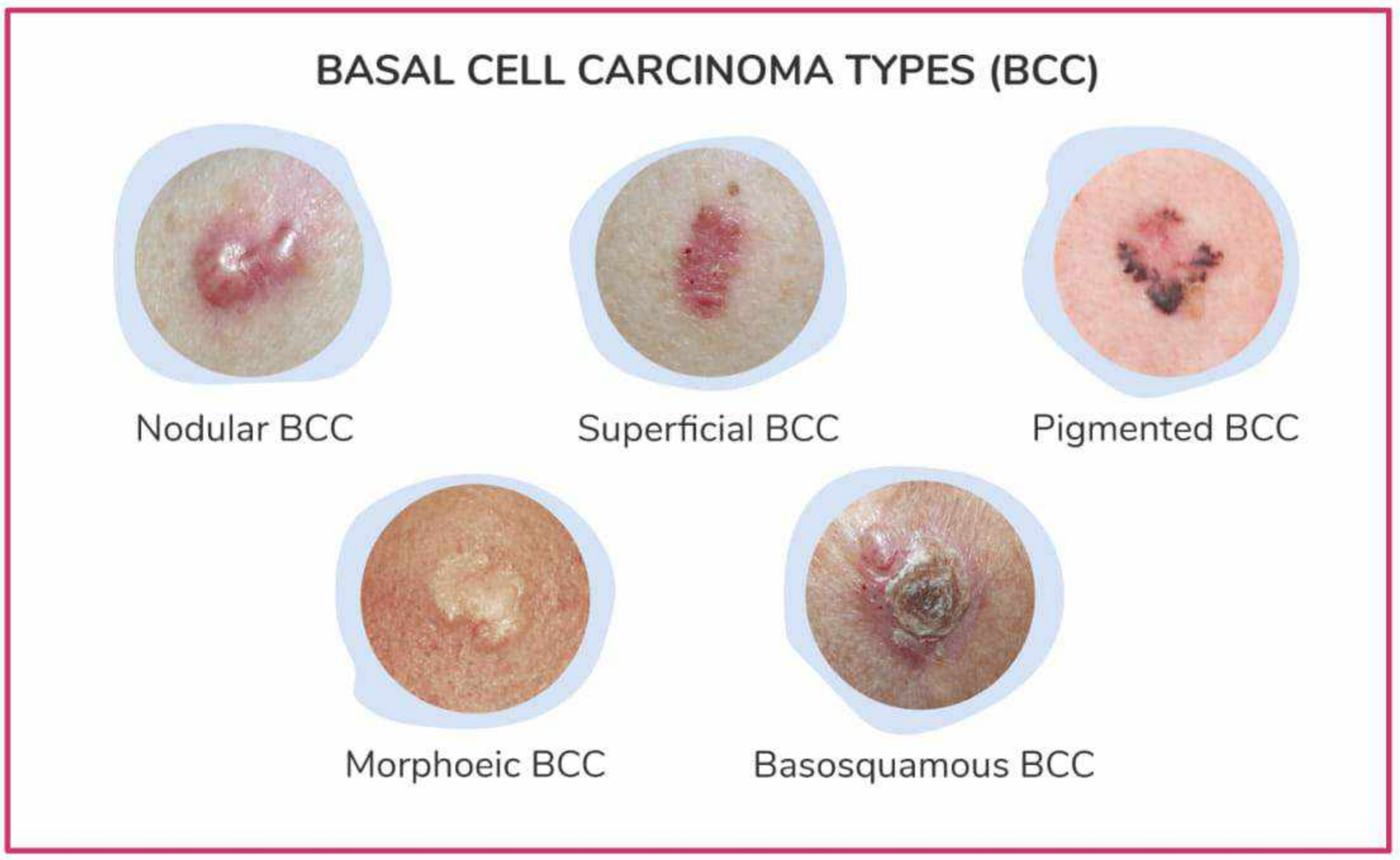
Rodent ulcer :

- Central ulceration
- Indurated raised border ± telangiectasias
- Bleeds on touch





active space



Nodular BCC

- (MC) Type
- a.k.a Noduloulcerative

Superficial BCC

- less common
- usually present on trunk

Pigmented BCC

- needs to differentiated from melanoma.

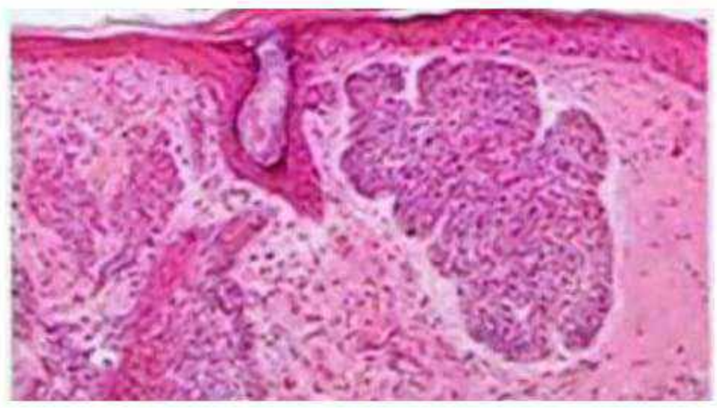
Morphoeic BCC

Basosquamous BCC : There are both features of BCC and SCC.

active space

- Associations :
  - Xeroderma pigmentosa
  - Albinism
  - Naevoid BCC Syndrome : (syn - Gorlin syndrome)
    - Autosomal dominant disorder with distinct clinical features including palmoplantar pits, odontogenic cysts. calcification of the falx cerebri, skeletal abnormalities, medulloblastomas and multiple BCC.

- On HPE :
  - All variants have certain common features
    - Presence of nests of Basaloid tumor cells with hyperchromatic nuclei and scanty cytoplasm.
    - Palisading of cells at the periphery.



Treatment

- Medical : low Risk Superficial and small nodular BCC
- Imiquimod, 5-FU
- PDT

active space

- Intralesional Interferon α-2b
- Hedgehog pathway inhibitors → used when BCC is recurrent, advanced, invasive and metastatic.
  ↓
  Vimodegib
- Surgical
  - Excision with predetermined margins : 4-5 mm surgical margin
  - Mohs migraphic surgery : Recurrent, Morphoeic or large Bcc which require a 3-15 mm margin in order to obtain a similar clearance rate
  - Cryotherapy
  - Superficial and Electron Beam Radiotherapy or Brachytherapy : 1° or surgically recurrent BCC as well as High Risk BCC patients who are unwilling or unable to tolerate surgery.

MALIGNANT MELANOMA 09:55

- A malignant tumor arising from melanocytes
- Metastasis seen

- **Predisposing factors :**

  i) Precursor lesions

  ii) UV Radiation : Controversial

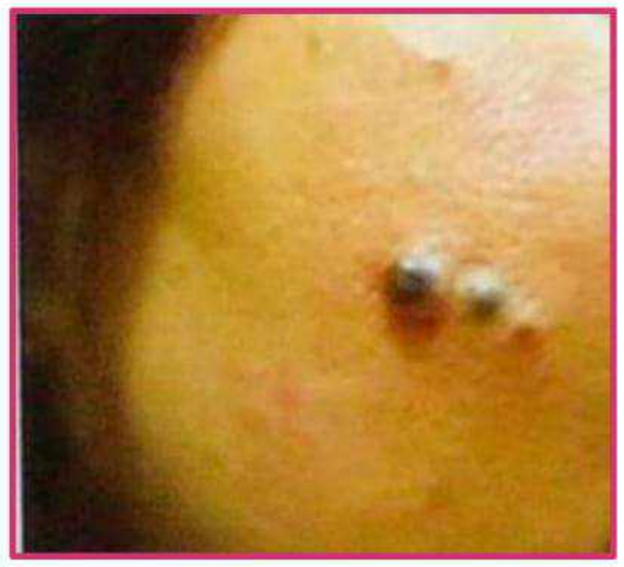
  iii) Genetics : Cyclin dependent kinase (CDK) inhibitor 2A (CDKN 2A).

Precursor lesions

- 20-50 % of melanomas are thought to develop in a cutaneous melanocytic naevus.

i) Congenital Melanocytic Naevus :

- large CMN (> 20cm), more often located on the trunk and present with satellites.

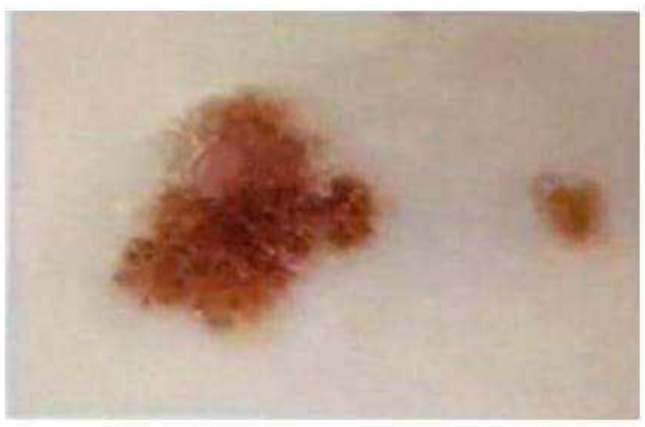


ii) Common Naevi - Risk of transformation very low, highest with junctional naevi.

iii) Dysplastic / Atypical Naevi

- **Atypical Naevi :**

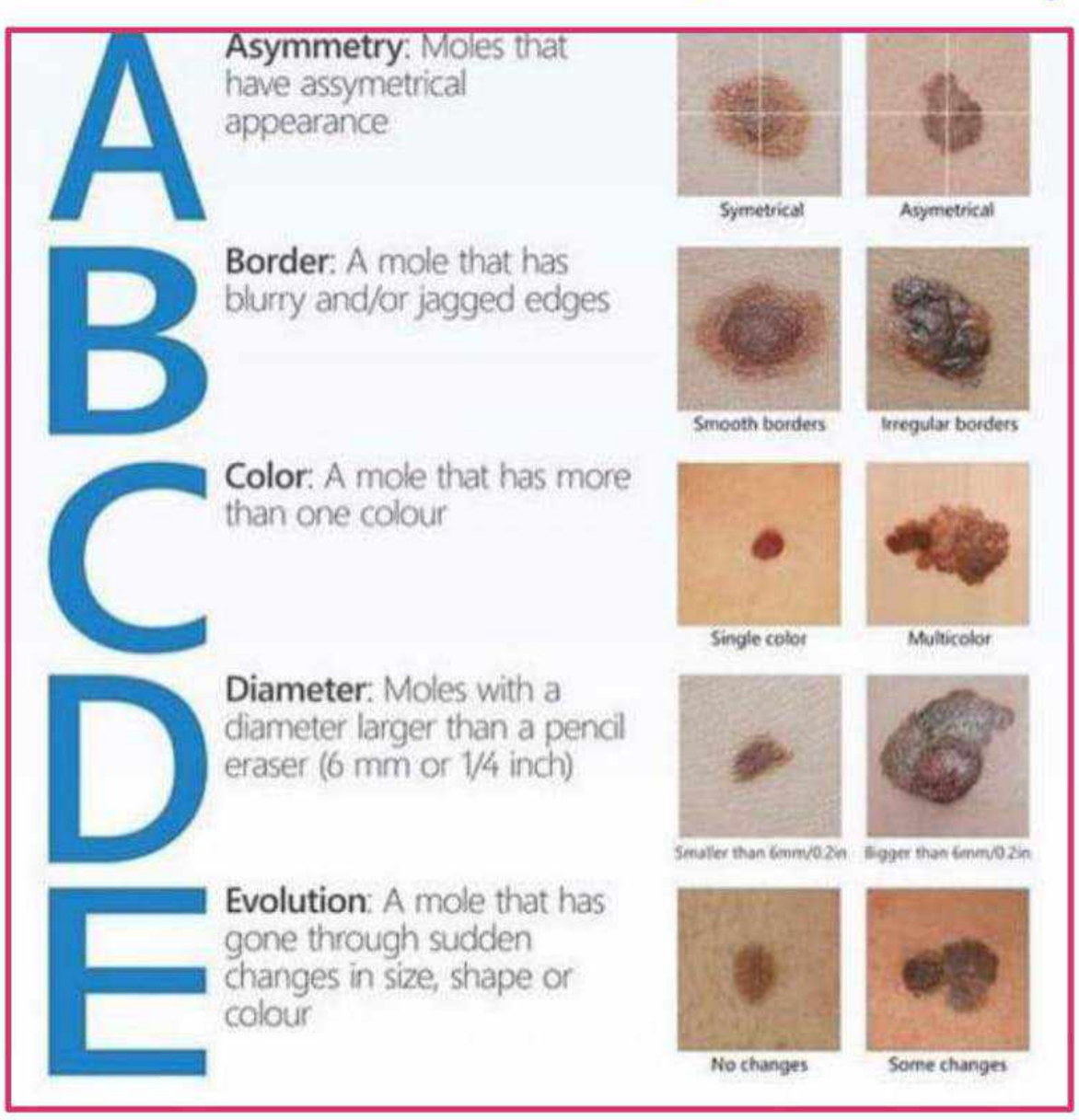
  (>5mm) with irregularly distributed colours and a tendency to emerge after young age.



active space

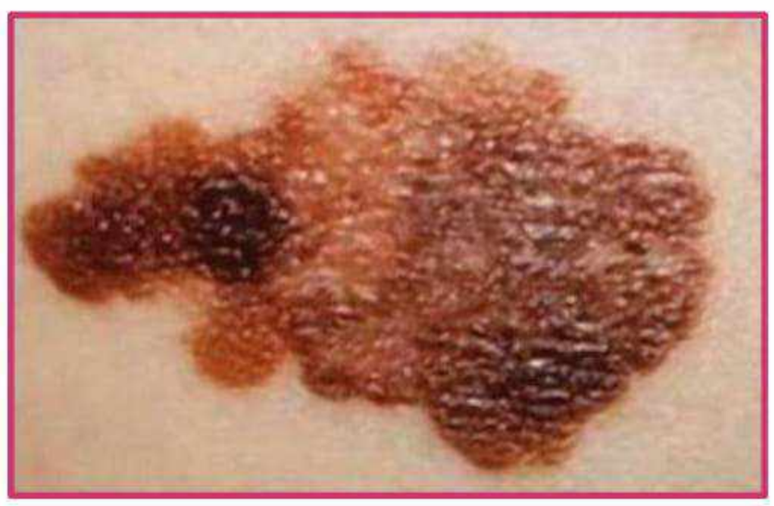
- **Dysplastic Naevi :** have some distinct patterns which are interpreted either as a pattern of naevus growth, or as a pre-malignant status.

- Acquired Melanocytic Naevi
  - → Clinical → Atypical
  - → Pathological → Dysplastic

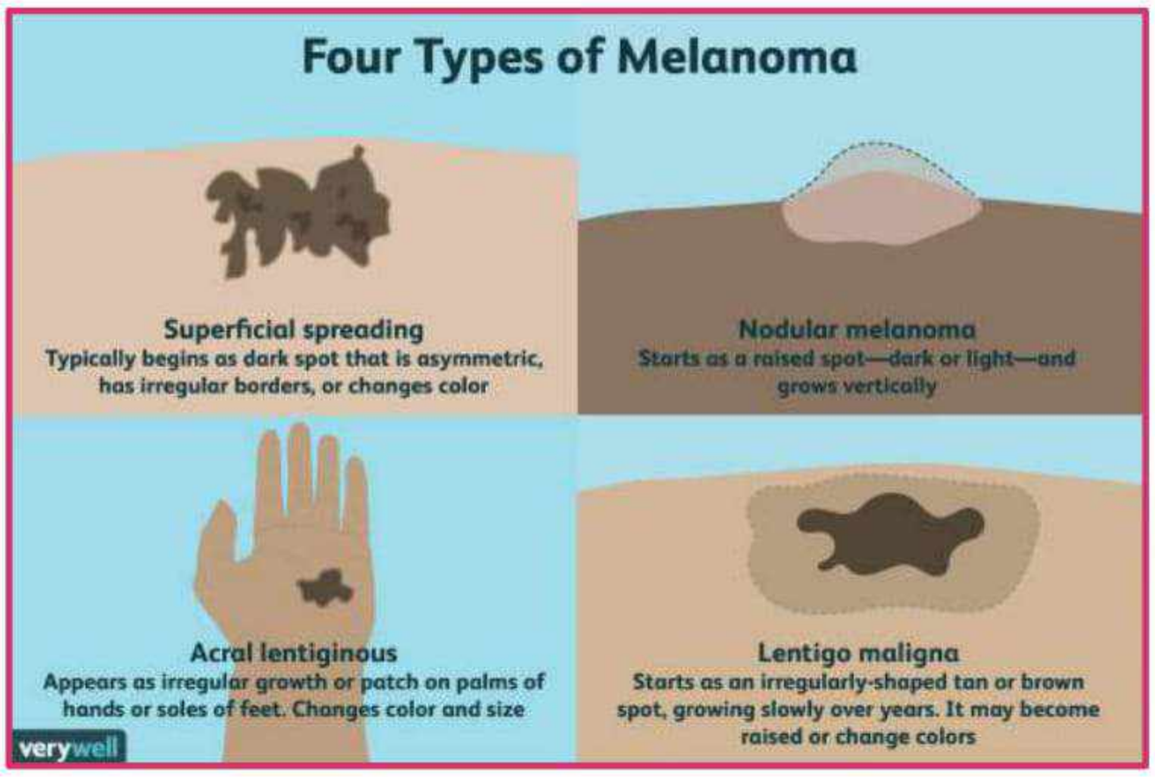


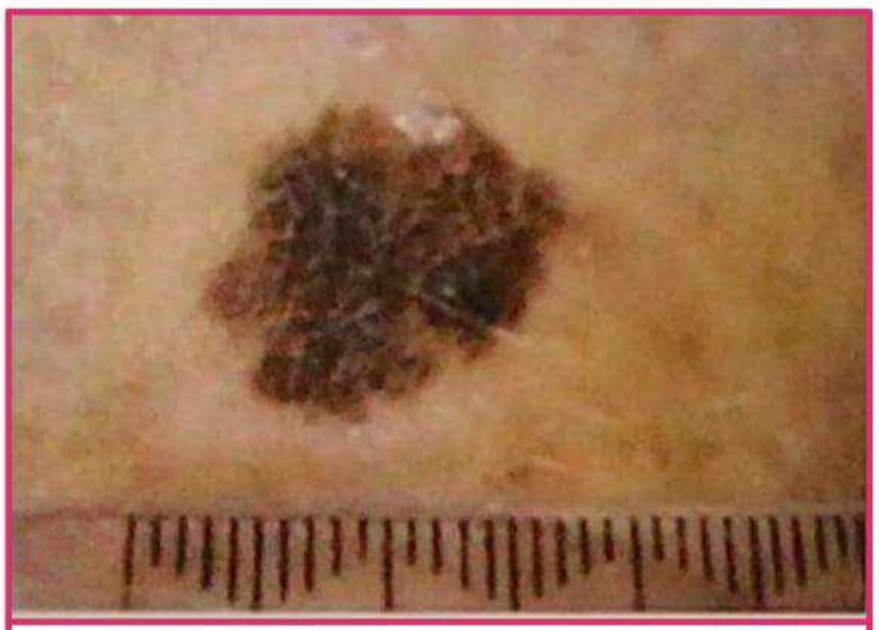
Clinical feature

- present as Pigmented lesion
- Dermoscopy is also done.

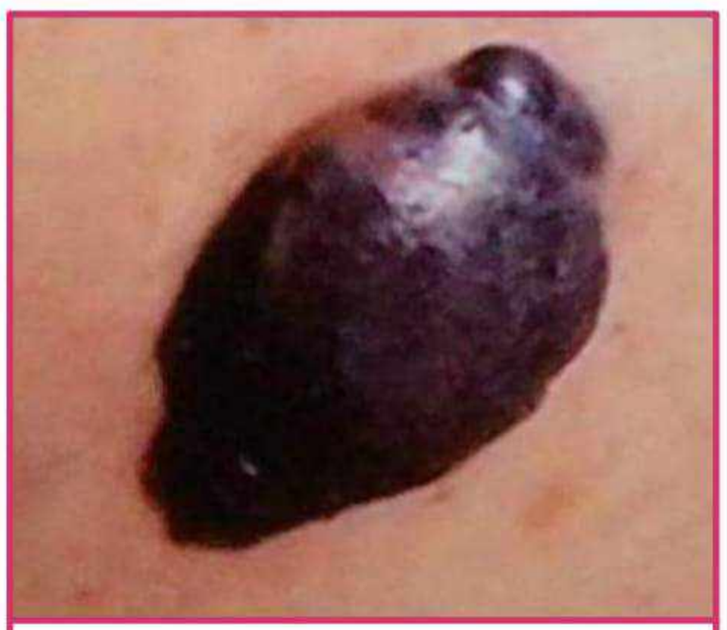


active space

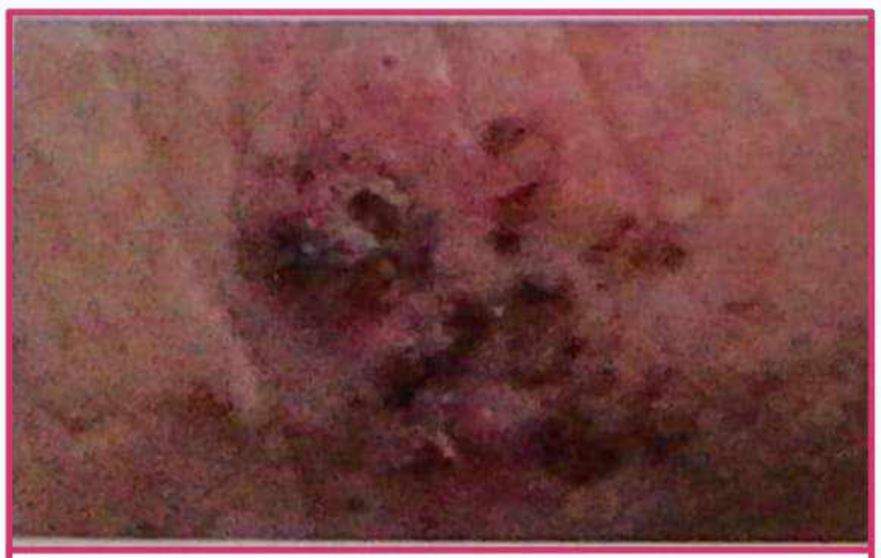




Superficial Spreading (MC)

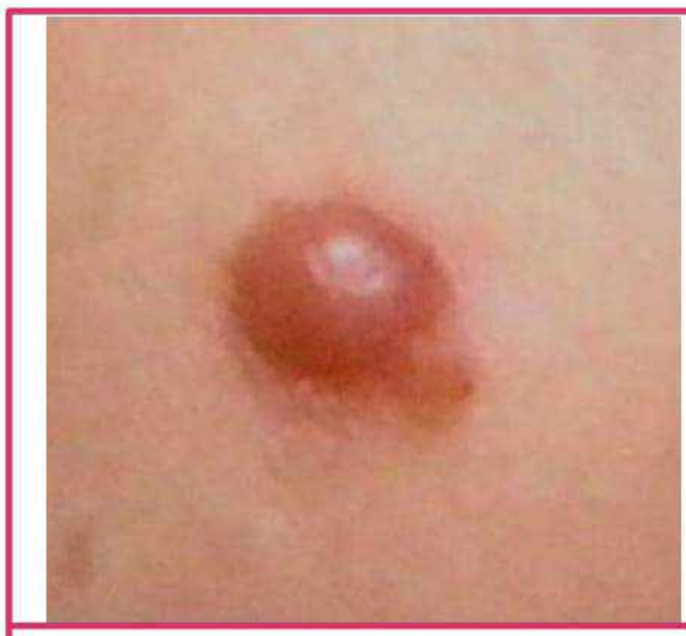


Nodular



Lentigo Maligna

→ Best Prognosis

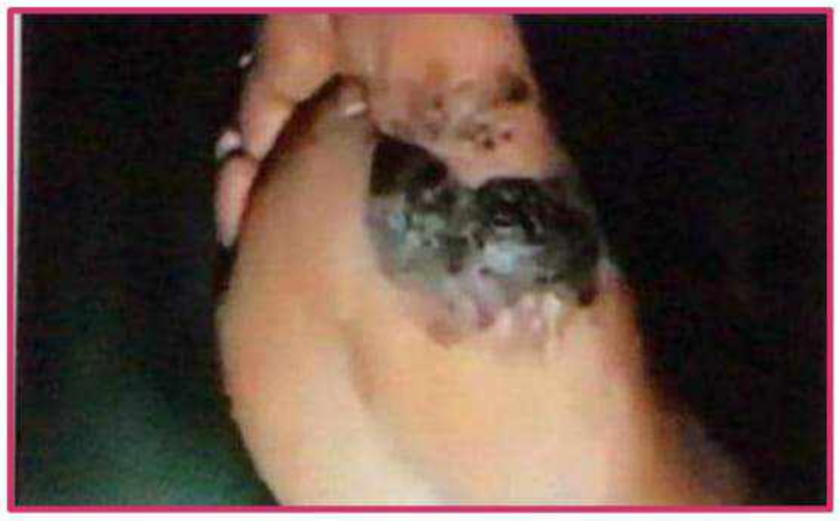


Amelanotic Melanoma

Worst Prognosis

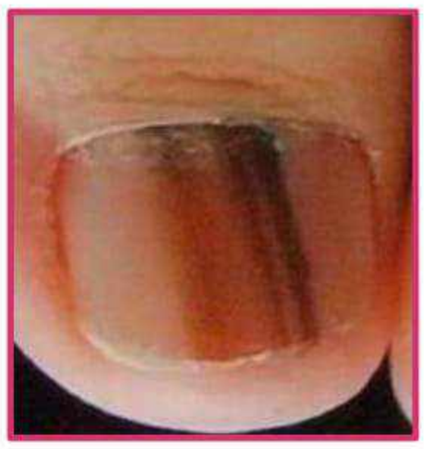
active space

Acral lentiginous melanoma



- seen on feets > Hands.
- Induration
- Ulceration.
- (MC) in South Asian people
- (MC) Type of Melanoma ⟶ India.

Subungual Melanoma

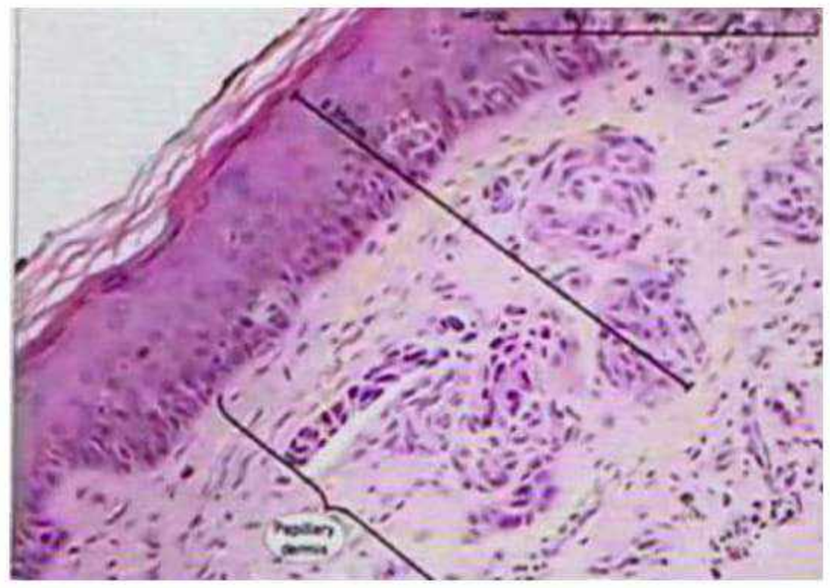


- Irregular longitudinal melanonychia.
- More common on Hands > Feet
- Hutchinson sign : pigmentation on Proximal Nail fold ⟶ Evaluate for Melanoma.

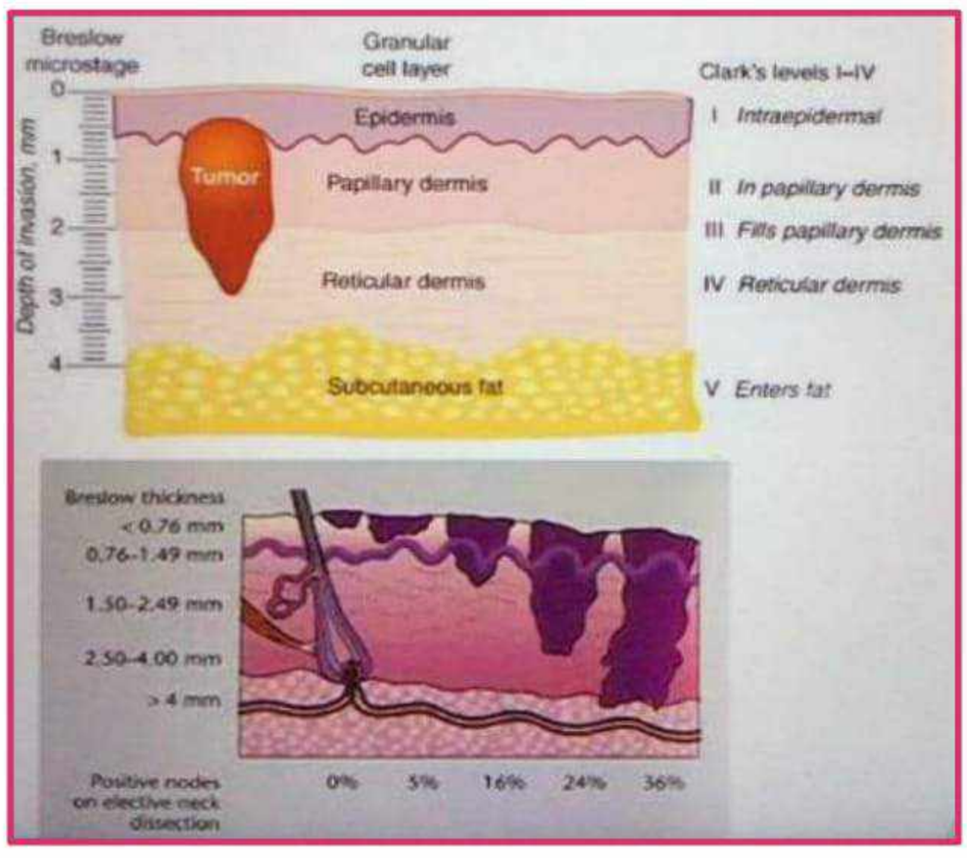
Investigations

- On HPE :
  - The ideal Biopsy is a narrow ~ 2mm margin excision of the entire clinically apparent lesion
  - Gold Standard for melanoma diagnosis
  - There is a vast spectrum of morphologies : nests of cells showing radial or vertical growth

- **Pathological Prognostic markers :**
  - **Breslow Depth :** measurement (in mm) of the distance between the overlying epidermal granular layer and the deepest level of invasion of the primary lesion.



    - **Mitotic Rate**
    - **Ulceration**
    - **sentinel node.**



- **Immunohistochemistry :** Melanocytes stain with **S-100, HMB 45, and MART-1** (a.k.a Melan A)
- **FISH.**

active space

- Staging of Melanoma : TNM Staging
  - Tumor size
  - Lymphnode involvement
  - Metastasis.
- Prognosis : can metastasize and go to different parts of body.

Treatment :

- Wide local excision of a melanoma (with margins measured before excision and determined by Breslow depth, with sentinel LN Biopsy indicated for tumors with a depth of >1mm)

Table 1. Recommendation of margins for melanoma.

Tumor thickness (Breslow)	Recommendation of margins
In situ	0.5 a 1 cm
≤ 1.0 mm	1.0 cm
1.01 a 2 mm	1 a 2 cm
2.01 a 4 mm	2 cm
> 4 mm	2 cm

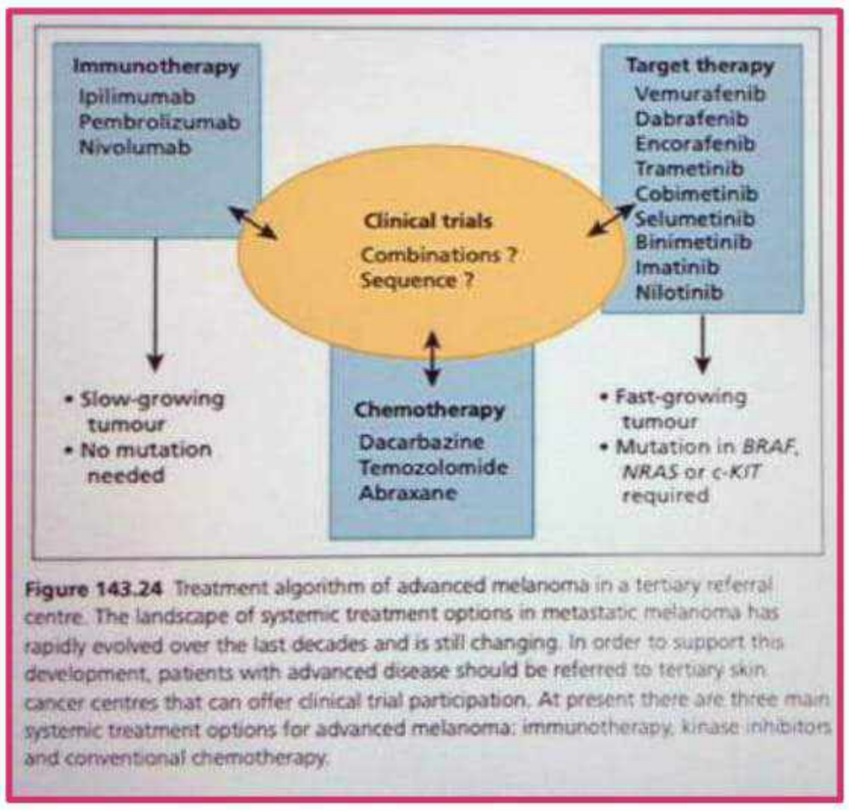


Figure 143.24 Treatment algorithm of advanced melanoma in a tertiary referral centre. The landscape of systemic treatment options in metastatic melanoma has rapidly evolved over the last decades and is still changing. In order to support this development, patients with advanced disease should be referred to tertiary skin cancer centres that can offer clinical trial participation. At present there are three main systemic treatment options for advanced melanoma: immunotherapy, kinase inhibitors and conventional chemotherapy.

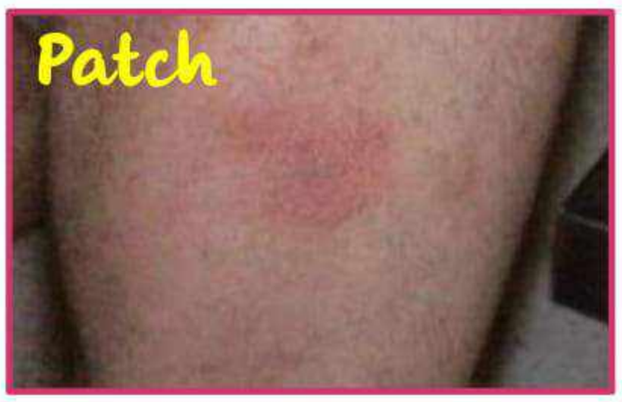
MYCOSIS FUNGOIDES

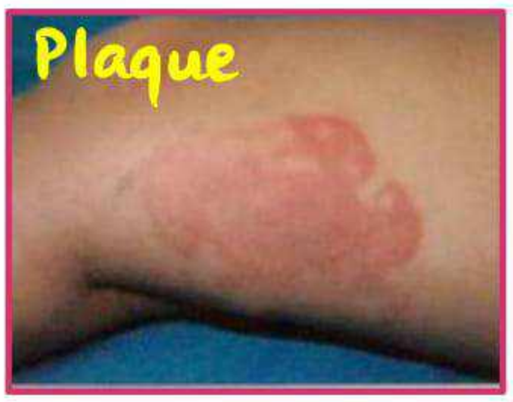
28:50

- Tumor of T cell in skin.
- (MC) variant of 1° CTCL
- characterized by monoclonal proliferation of predominantly CD4+/CD45RO+ helper T cells and the loss of mature T cell antigens in the skin and other involved organs.
- course: T lymphocytes go into epidermis (Epidermotropism)
  ↓
  Dermal invasion
  ↓
  Regional LN
  ↓
  Systemic Invasion
  - Indolent clinical course.
- Sites: Girdle → Trunk, Buttock, Nipple area.

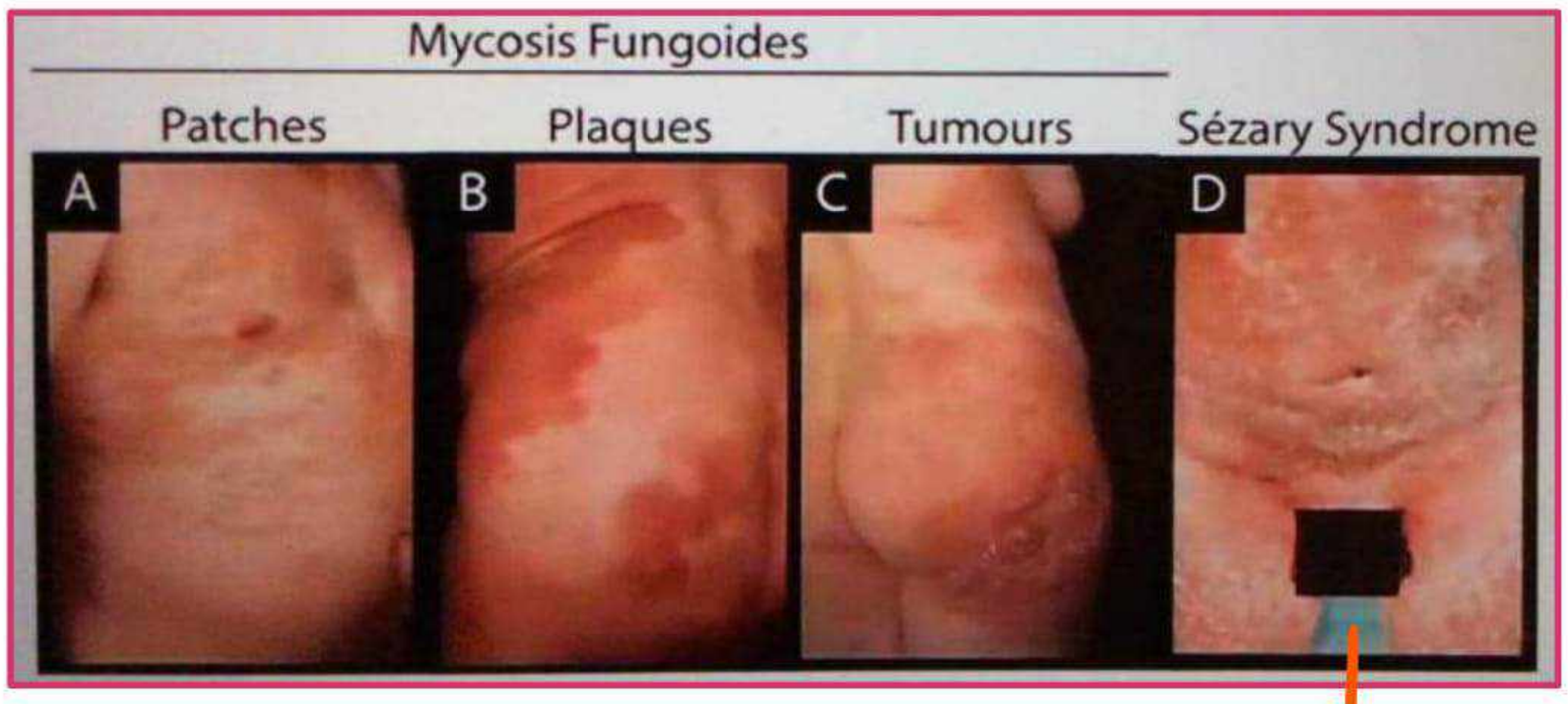
Clinical feature:

- Polymorphic
- Stages: Patch → Plaque → Tumor





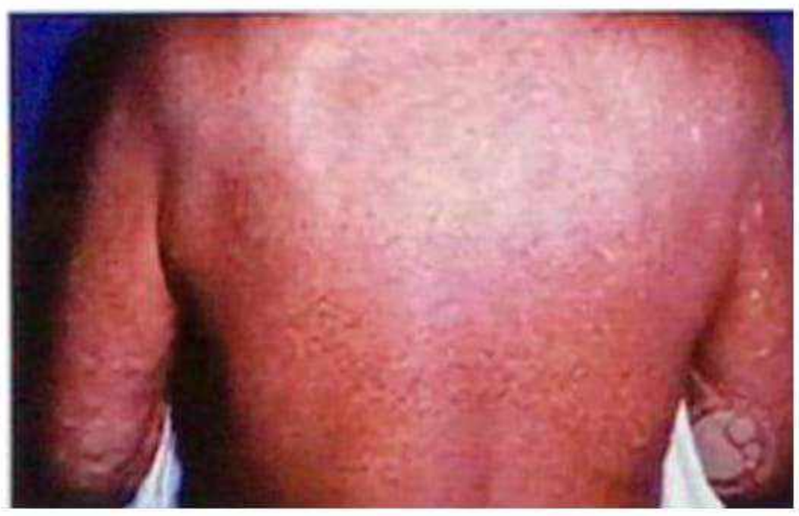
- **Patch:** Erythematous slightly scaly, slightly atrophic, wrinkled plaque.
- **Plaque:** Polymorphic erythematous raised plaque.



Erythroderma: may be due MF or Sezary syndrome.

Sezary Syndrome

- **3 features:**
  - i) Erythroderma
  - ii) Peripheral lymphadenopathy
  - iii) Sezary cells ⊕ (atypical lymphocytes)

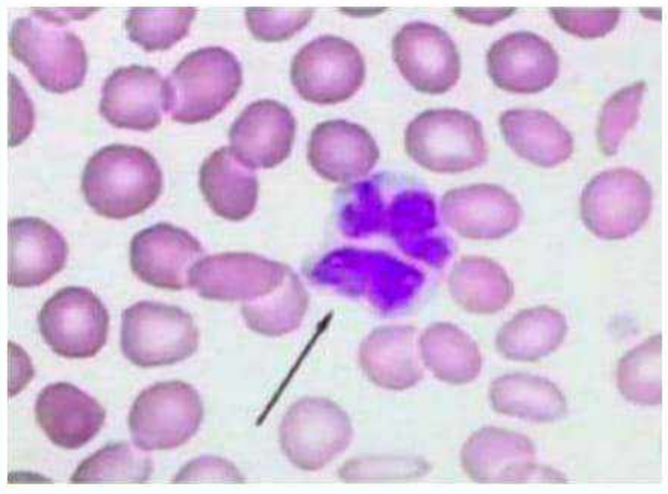


- > 20% of Total lymphocyte count
- > 1000 mm^3

} seen in peripheral Blood.

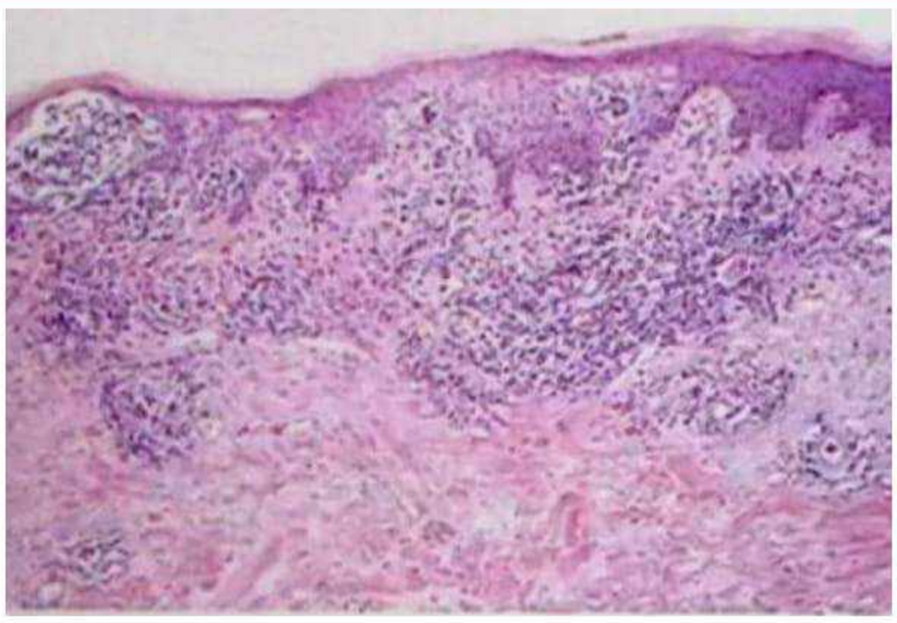
→ Helps to differentiate from Erythrodermic MF.

active space



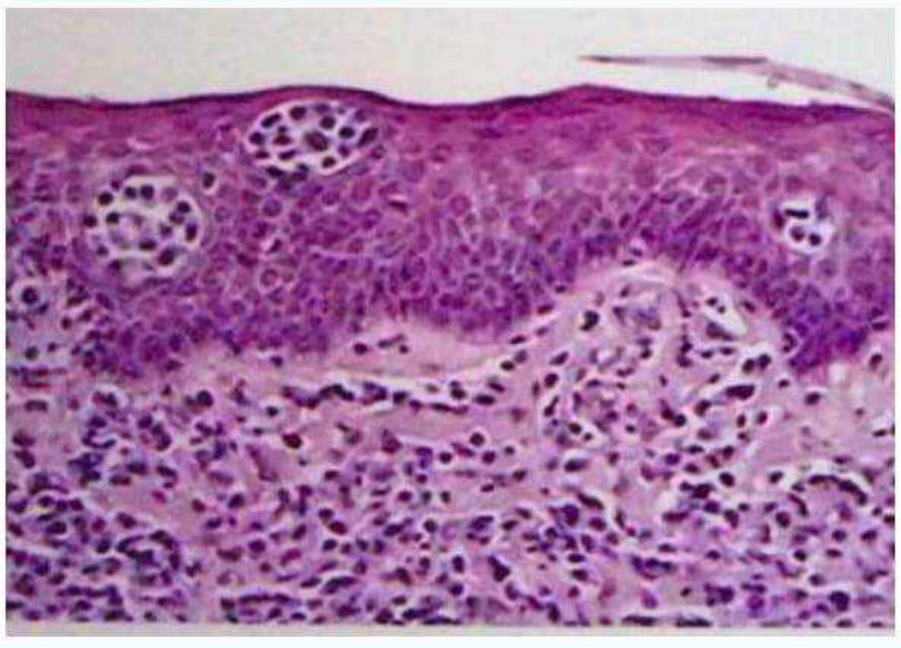
Sezary cell : atypical lymphocytes with a grooved or cerebriform nucleus seen both in tissue and blood.

- **On HPE :**



Mycosis fungoides

- Epidermotropism
- Atypical lymphocytes invading the epidermis.



Pautrier's Microabscesses

- seen in MF
- collection of atypical lymphocytes in epidermis.

active space

Treatment

i) For Patch :

- Topical steroids , Nitrogen mustard (Mechlorethamine) and carmustine (BCNU), Bexarotene gel , PUVA , UV-B

ii) For Plaque :

- Total skin electron beam therapy , Interferon α , Oral Retinoids

iii) Nodal and Visceral type :

- Aggressive chemotherapy consisting of CHOP Regimen (Cyclophosphamide, Doxorubicin, Vincristine and Prednisone)

iv) Newer :

- Denileukin diftitox , a DAB - IL-2 fusion toxin , Histone deacetylase inhibitors.

active space

Urticaria and Angioedema

PATHOGENESIS

00:40

- Vasodilatation → Erythema
- Plasma leakage. → Edema.
- These are caused by Histamine ← IgE ↙ Mast cells

IgE Dependent → Allergic / Immunologic

Mast cell dependent

Non-IgE Dependent

Mast Cell Independent

- Mast cell dependent is more common

IgE + Allergen → Mast cell → Degranulation → Release of Histamine

Allergens and Autoantibodies against IgE (Immunological) ↘

Substance P, Morphine Codeine, Complement 5a (Non-immunological) ↙

Mast cell dependent

↓

Histamines and leukotrienes

↓

active space

↓

Vasodilation and increased permeability

↓ ↑

Wheal and Angioedema. — Mast cell independent

↑

Sorbic acid, Benzoic acid, food additives and Bradykinin (HAE)

Lewis Triple Response → Erythema, Flare and Wheal

- Histamine
  - Blood vessels
    - Erythema
    - Edema
  - Nerve endings
    - Pruritus
    - Axon Reflex and Flare

Etiology

- Idipathic → 60%
- Allergic cause : i) Drugs
  ii) Food
  iii) Insect Bite
  iv) Pollens

T's

- Idiopathic
- Inhalation
- Ingestion
- Infection → UTI, URTI
- Injection → Drugs.
- Insect

active space

- **Non-Allergic :**

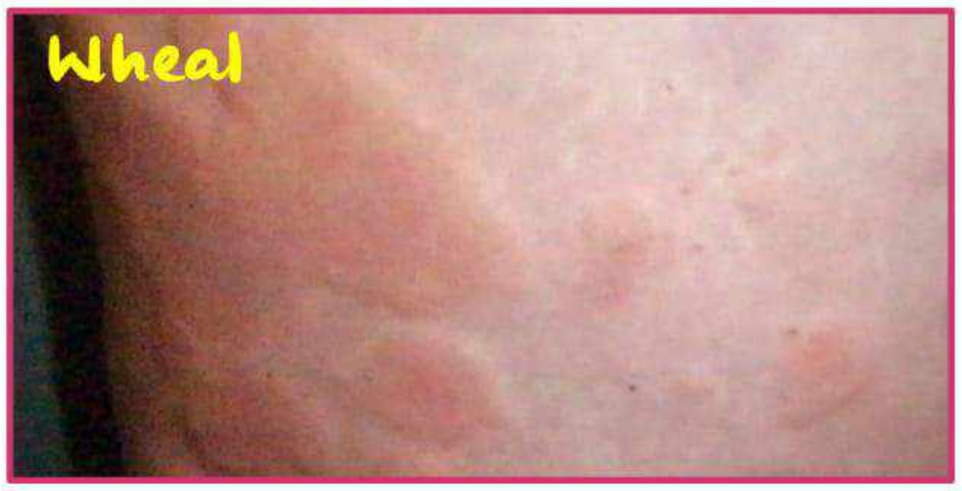
  i) Iodinated Radiocontrast dyes

  ii) Morphine, Codeine, Neuromuscular blocking agents and antibiotics such as polymxin and vancomycin.

  iii) Streptococcal throat infections, dental sepsis, sinusitis, UTI and Gall bladder infection.

Clinical features

- Wheal → • Itchy
  - Evanescent
  - Edematous
  - Erythematous.



- Evanescent → Transient in nature.
- **Acute Urticaria :** Duration of urticaria < 6wks
- **Chronic urticaria :** Duration of urticaria > 6wks

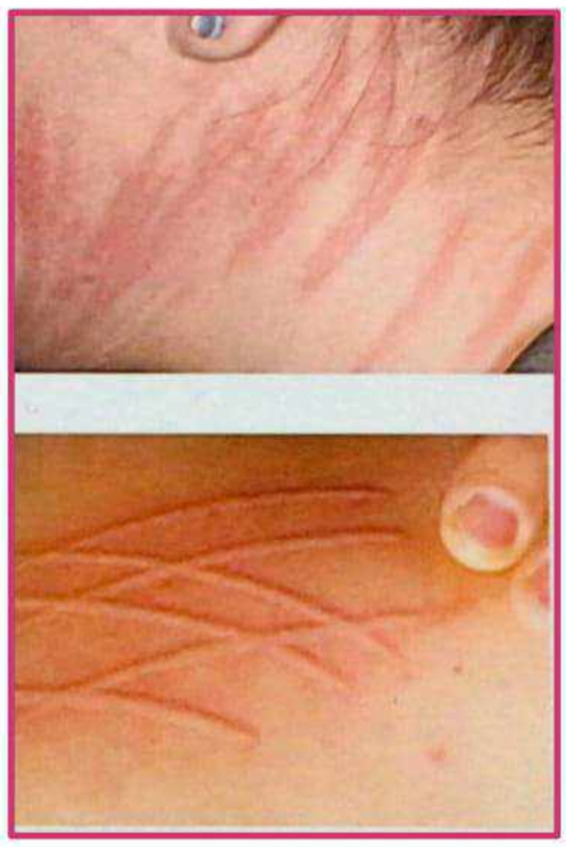
active space

- **Causes :**

ACUTE URTICARIA	CHRONIC URTICARIA
• Duration < 6wks • Infections (40%) : viral • Drugs (9%) - Penicillin, Sulfa drugs • Food items (1%) - Additives • Inhalation - Pollen. • Idiopathic (50%)	• Daily or almost daily for > 6 wks • Chronic spontaneous urticaria (autoimmune, pseudoallergic, infection and idiopathic - 65%) • Physical / Inducible urticaria (35%) • urticarial vasculitis (5%)

Physical Urticaria

Dermographism / Dermatographism.



- a.k.a Dermatographic urticaria
- linear wheals generated on gentle stroking of skin
- starts within minutes and disappears in hour
- (MC) Physical urticaria.

active space

Heat Contact Urticaria

- develops urticaria when exposed to higher temperatures (↑ 38°c)

Solar Urticaria

- urticaria develops on uv exposure.

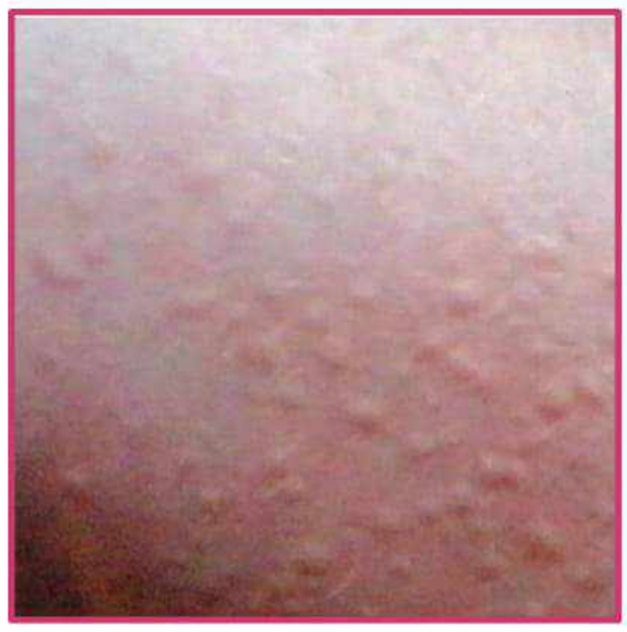
Aquagenic urticaria

- urticaria develops on contact with water

Aquagenic pruritus

- There is itching and no wheals on contact with water.
- seen in Polycythemia vera

Cholinergic urticaria



- urticaria develops whenever patient sweats.
- seen during → i) ↑ core body temperature
  ii) Emotional / Anxiety
  iii) Gustatory sweating
- due to stimulation of cholinergic post ganglionic nerve supplying sweat gland.
- Pinpoint 1-3mm papules

active space

Delayed Pressure Urticaria

- caused by mechanical pressure
- develops after 3-12hrs.

Management

- Identify the cause and avoid it.

Modern 2nd Gen. H1 Antihistamines in standard doses → 1-2 wks

Four fold increase dosages of same antihistamines → 2-4 wks.

Add Omalizumab, Cyclosporine, Monteleukast, 1st Gen Antihistamines at Bed time

↓

Dapsone, Colchicine, HCQ's, Ranitidine, Methotrexate, Azathioprine, Phototherapy, Plasmapharesis etc.

URTICARIAL VASCULITIS

22:50

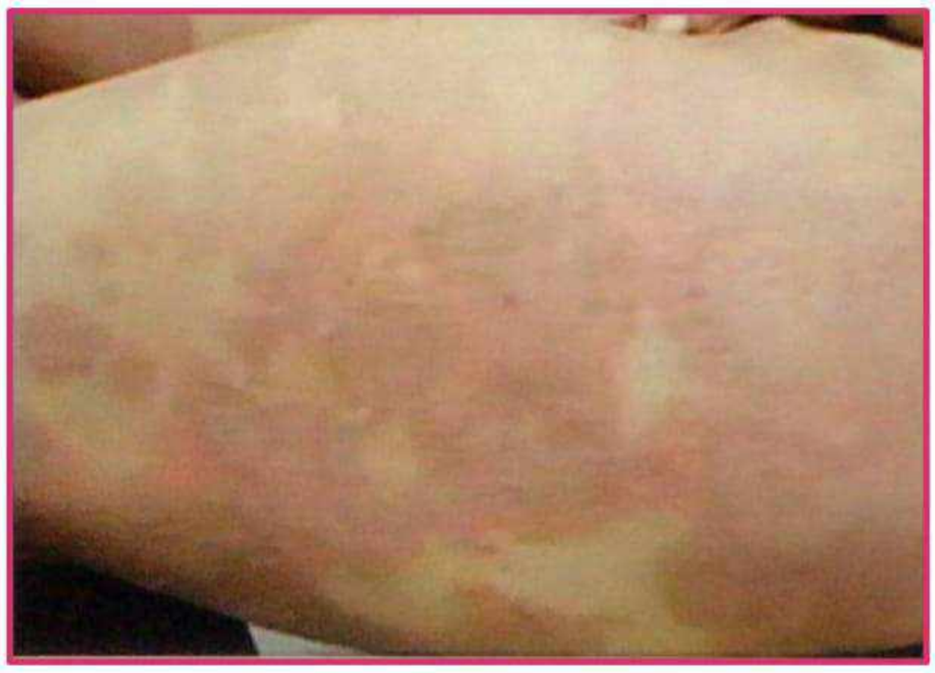
- urticarial wheals + Leukocytoclastic vasculitis on Biopsy.

Clinical features

- Persistent for 24-48 hrs
- more painful, less itchy
- heal with Post Inflammatory Hyperpigmentation

active space

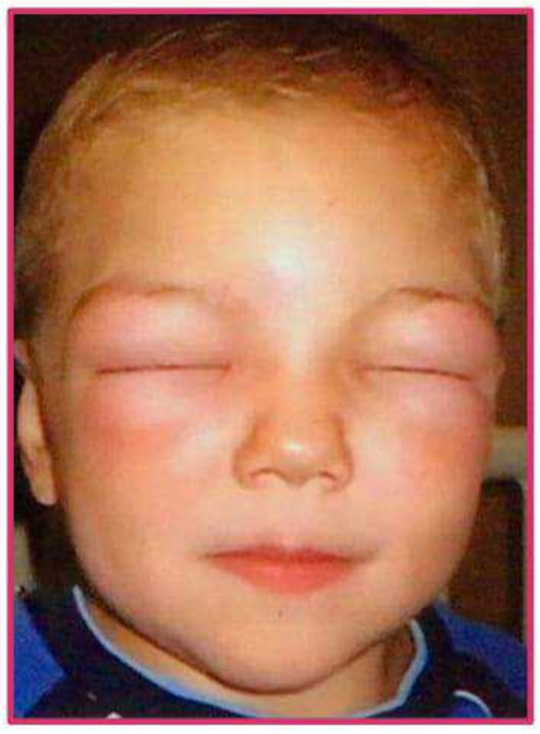
- associated with malaise, joint pains
- **Associations :** CTD, Hematological malignancies



ANGIOEDEMA

26:00

- swelling of hypodermis / mucosa
- submucosal swelling
- Deeper
- No margins
- Painful, less itchy
- Less erythematous.



Types

i) With wheals

ii) Without wheals. → Bradykinin mediated

↳ Heriditary / Acquired.

active space

- Hereditary :
  - 3 Types → Type I, Type II, Type III
  - C 1 Esterase inhibitor deficiency
  - Type I → Low C_1 INH
  - Type II → loss of function of C_1INH
  - Type III → Normal levels of C_1INH
- Acquired
  - Acquired deficiency of C_1INH
  - ACE inhibitor
  - Idiopathic.

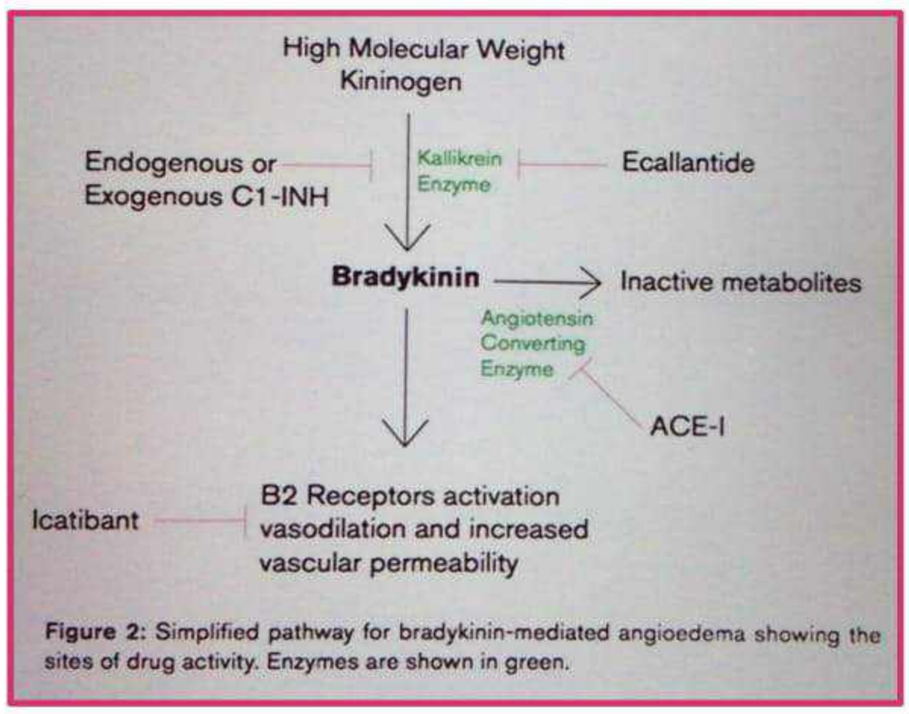


Figure 2: Simplified pathway for bradykinin-mediated angioedema showing the sites of drug activity. Enzymes are shown in green.

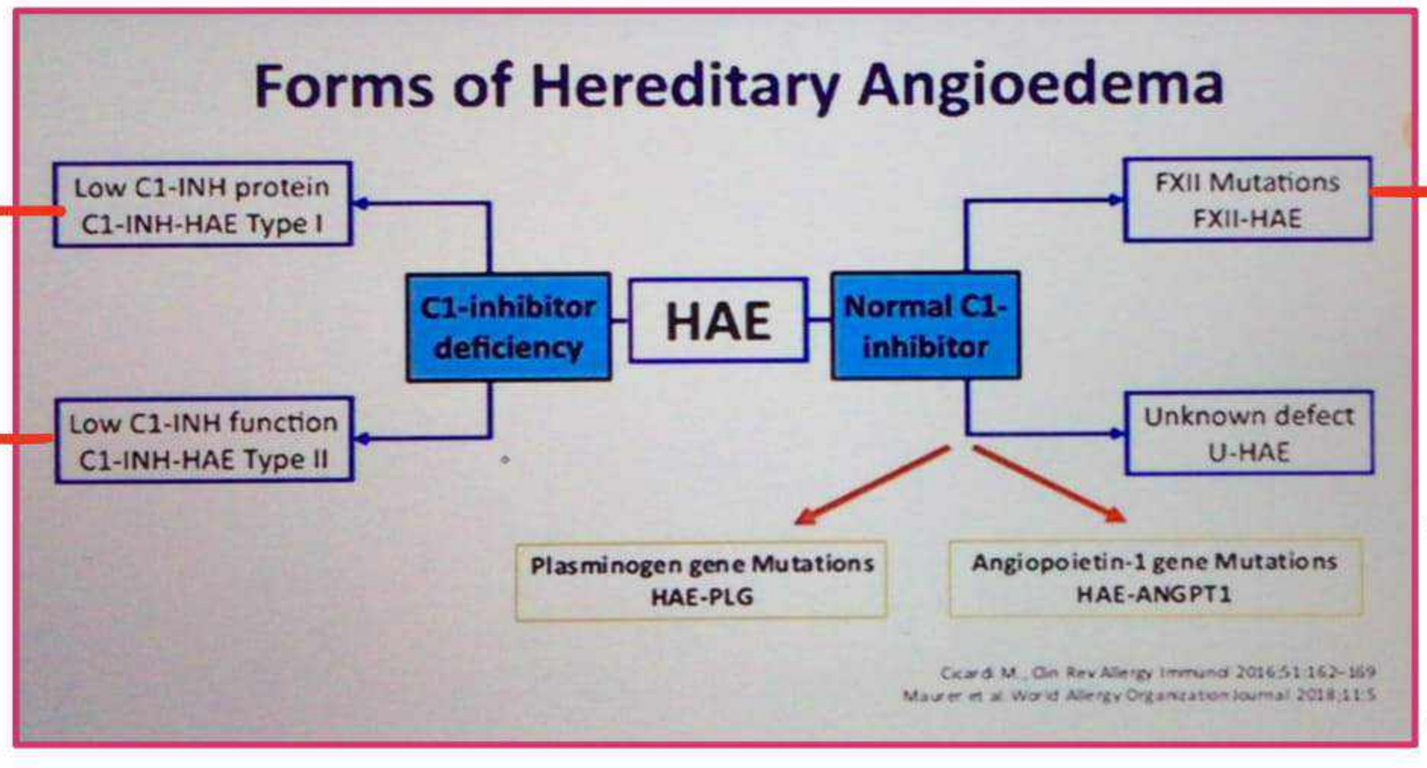
- C_4 and C_2 → low levels

active space

HEREDITARY ANGIOEDEMA

32:55

- a.k.a Quincke's disease
- Inheritance → A.D
- Bradykinin mediated



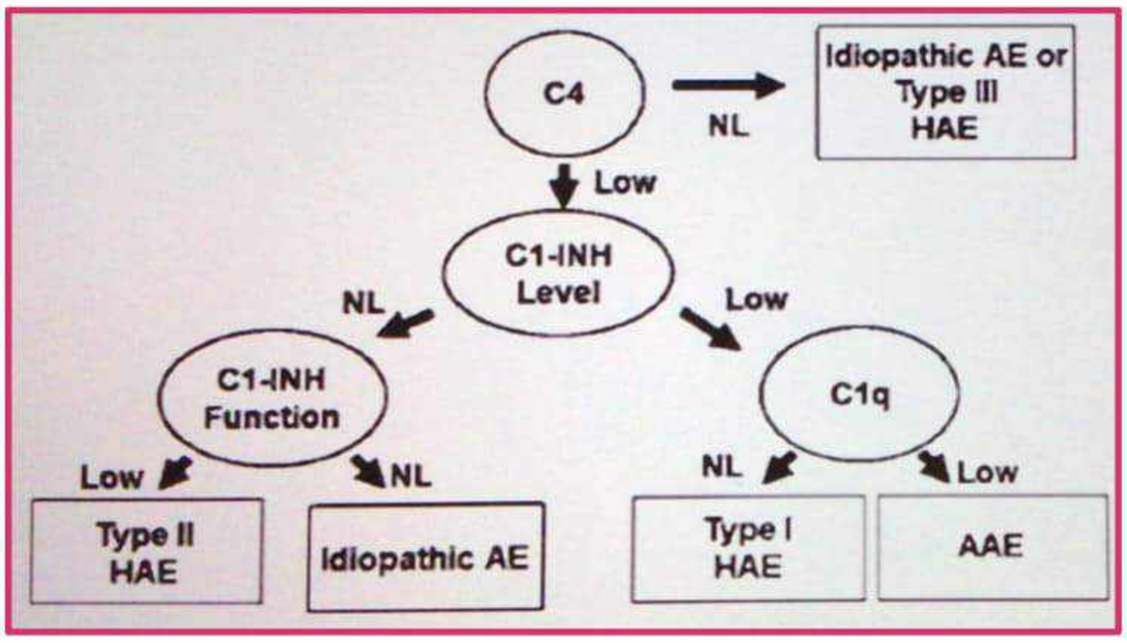
- Family H/o
- Recurrent Angioedema
- C_1INH ↘ Normal

- Positive Family History

Symptoms

- Recurrent Angioedema (without wheals)
- Abdominal Colicky pains
- Recurrent Laryngeal edema.
- Investigation :

  C4 value screening test



active space

Treatment

Acute Management

- Airway management
- Purified plasma derived C_1INH
- **Icatibant**, a bradykinin receptor 2 Antagonist
- **Ecallantide**, A Kallikrein inhibitor
- **Fresh Frozen Plasma**, containing C_1INH

For Prophylaxis

- Plasma-derived C_1INH Replacement therapy
- Tranexamic acid
- Anabolic steroids (Danazol, Stanozolol)

active space

Pediatric Dermatoses

NAEVI

00:35

- Birthmark.
- synonymous with cutaneous hamartoma tumor-like, non-neoplastic proliferation of abnormal mixtures of the normal components of a tissue
- due to genetic mosaicism
- Follow Blaschko's lines.

Table 10.1: Classification of nevi

1. **Epidermal nevi**
   a. Keratinocyte:
      Verrucous epidermal nevus
      Inflammatory epidermal nevus:
         Inflammatory linear verrucous epidermal nevus
         Other inflammatory nevi
   b. Appendageal: Sebaceous: Nevus sebaceus
      Follicular: Nevus comedonicus
      Eccrine nevi
      Apocrine nevi
   c. Becker nevus
   d. Epidermal nevus syndromes
2. **Melanocytic nevi**
   Congenital melanocytic nevi
   Acquired melanocytic nevi (junctional, compound, intradermal)
   Special variants of acquired nevi
   Clinically atypical nevi
   Dermal melanocytic nevi
3. **Dermal and subcutaneous nevi**
   a. Connective tissue: Of the reticular connective tissue (collagen, elastin, glycosaminoglycan)
      Of the adventitial connective tissue
   b. Fat: Nevus lipomatosus cutaneous superficialis
   c. Smooth and striated muscle nevi
   d. Vascular: Tumors (hemangioma, tufted hemangioma, Kaposiform hemangioendothelioma)
      Malformations (capillary, venous, mixed, complex combined)

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c. Smooth and striated muscle nevi
d. Vascular: Tumors (hemangioma, tufted hemangioma, Kaposiform hemangioendothelioma)
Malformations (capillary, venous, mixed, complex combined)

- vascular syndromes
- Angiokeratomas

active space

VERRUCOUS EPIDERMAL NEVI

03:40

- formed of Keratinocyte
- (MC) Epidermal Nevi
- Presents at birth → erythematous papules along lines of Blaschko's.

  ↓

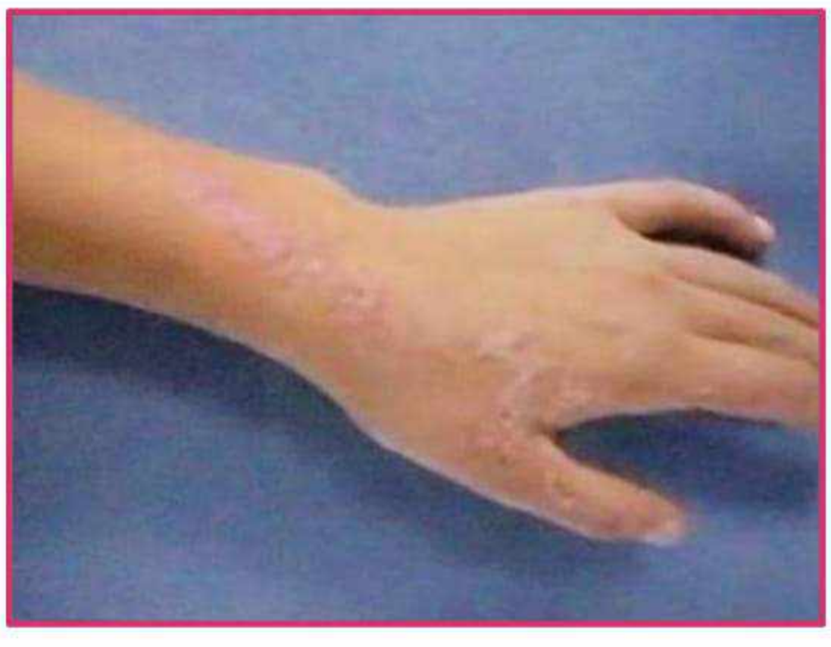
  more rough, pigmented, verrucous over time.

Treatment

- CO_2 LASER
- Electrocautery

ILVEN

05:55



- a.k.a Inflammatory Linear VEN
- appears by 6 months of age.
- Inflammatory erythematous sometimes eczematous.
- associated with pruritus.

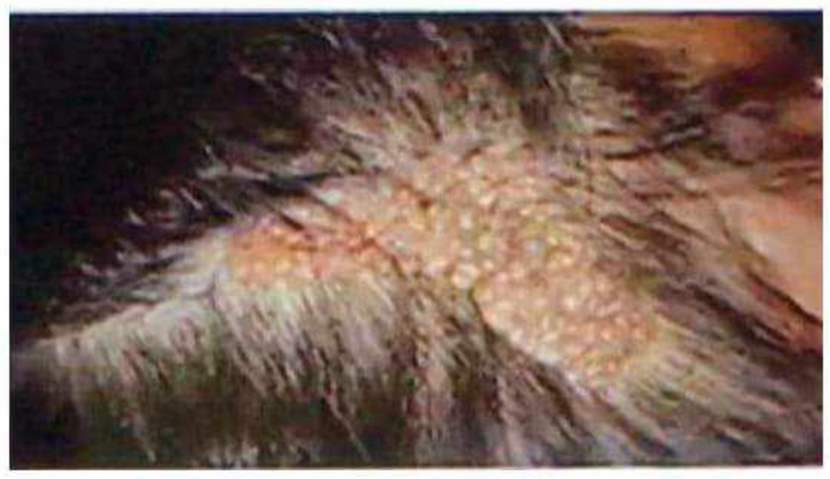
Treatment

- Resistant to Rx
- Topical steroids provide symptomatic relief.

active space

SEBACEOUS NEVI

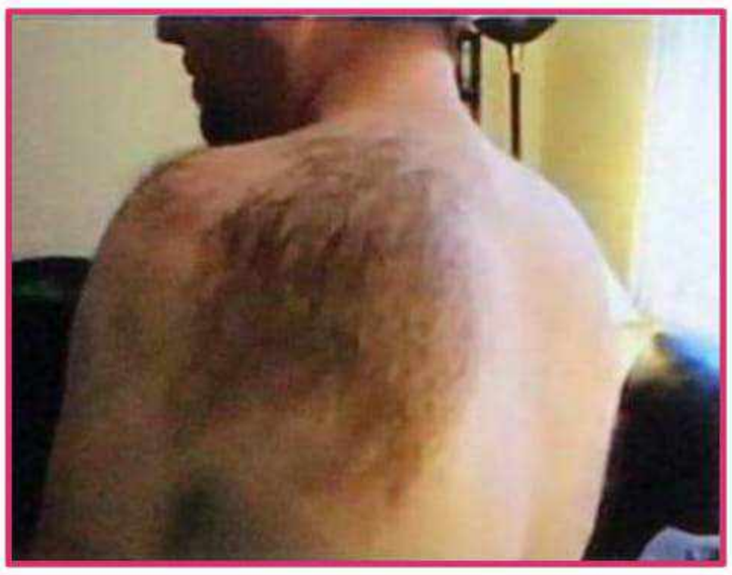
07:30



- Its an appendageal nevi
- seen on scalp and face
- sebaceous globules, abortive hair follicles.
- it increase with age.
- Appear as smooth yellow coloured papules which coalesce to form plaques.
- They increase in puberty
- There are chances for malignant transformation (5%)
- (MC) Benign tumor associated with it → Syringocystadenoma papillefera.
- (MC) skin cancer associated with it → BCC

BECKER NEVUS

10:45



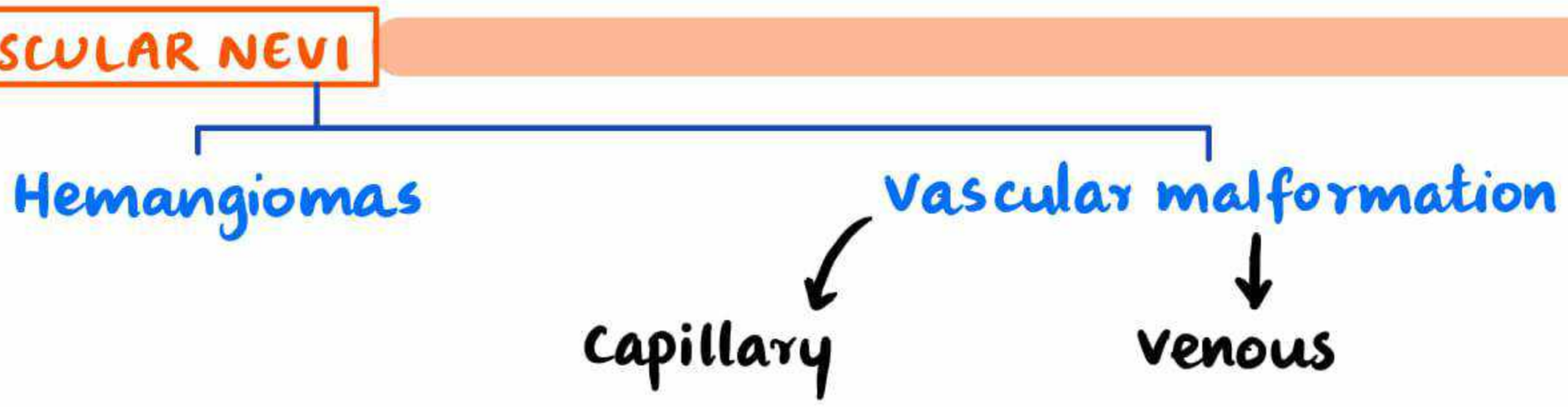
- Nevus present at adolescence
- Presents with hyperpigmented macules
- Site : Shoulder, Anterior chest, Scapula

- usually U/L, can be B/L
- As child attains puberty they become thicker, corrugated, thick pigmented hair overlying the macules.

  ± Acne
- Males > Females
- It is probably due to androgen hypersensitivity

Treatment → LASERS (not satisfactory)

VASCULAR NEVI



14:00

Hemangioma :

- appear in neonatal period, early infancy
- Progressive stage → Maturation Stage → Regression stage
- No treatment needed.

Vascular malformations

- are present at birth
- are persistent
- Needs Treatment

Infantile Hemangiomas

- (MC) Type of vascular nevi of childhood
- Age of onset: Neonatal, Early infancy, appears 1 month of age
- Types:

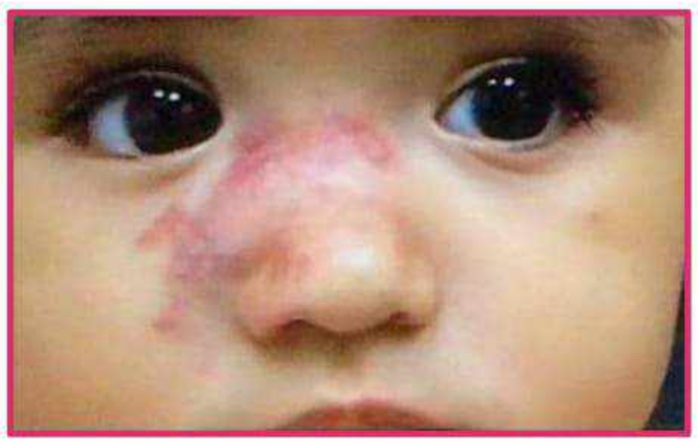
  i) Superficial → Strawberry

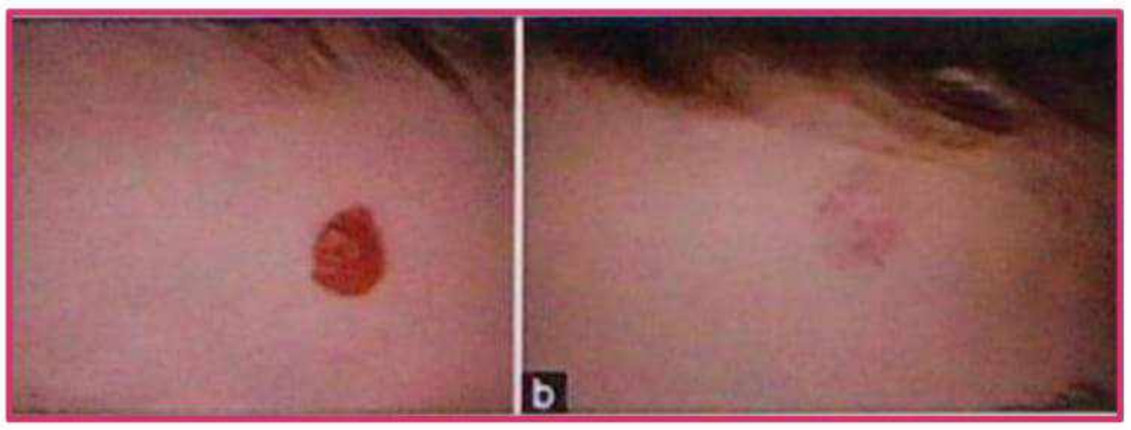
  ii) Deep → Cavernous / s.c → Deep, Bluish, Bag of worms.

  iii) Mixed.

- Course: 3 Stages of Evolution ↘

appear at 1 month
↓
↑ in size from 3-9 months (maximum size by 1yr of age)
↓
Regression starts after 10 months.





active space

Pinnochio nose

- Infantile hemangioma on nose tip

Indications for treatment

- ulceration
- Bleeding
- Recurrent infections
- Affecting a vital structure

Treatment

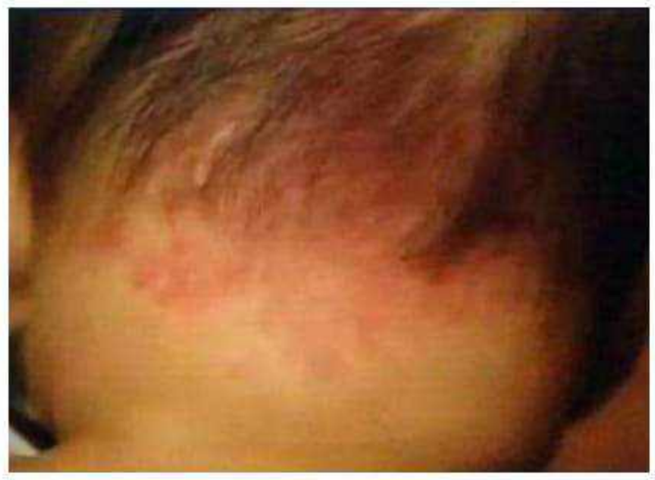
- Steroids
- Propanolol
- Kasabach Merritt Syndrome (Hemangioma Hemorrhage Syndrome)
  - occurence of thrombocytopenia in patients with hemangioma

active space

- Hemorrhage occurs due to platelet sequestration and a resultant consumptive coagulopathy with activation of the fibrinolytic system.
- mainly with tufted angiomas and kaposiform hemangioendothelioma.

SALMON PATCH

22:45



- capillary malformation
- a.k.a Erythema nuchae, Stork Bite.

Clinical feature

- present at birth
- Salmon erythematous macules (non-blanchable) flat
  ↓
  seen on nape of neck, eyelids, forehead globular in shape
- AD Inheritance
- Disappears with age (within 1yr)

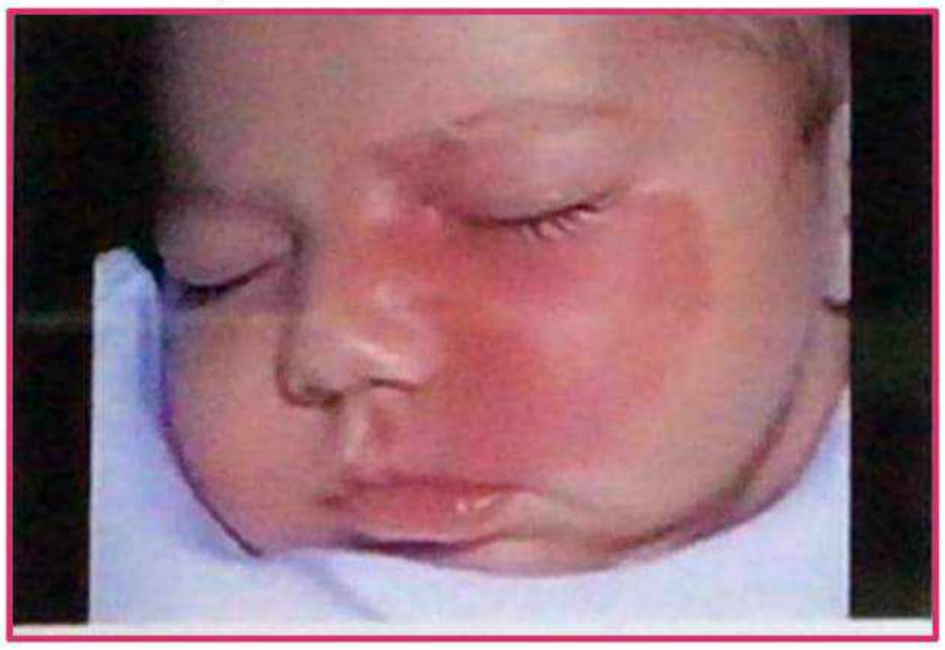
PORT WINE STAIN

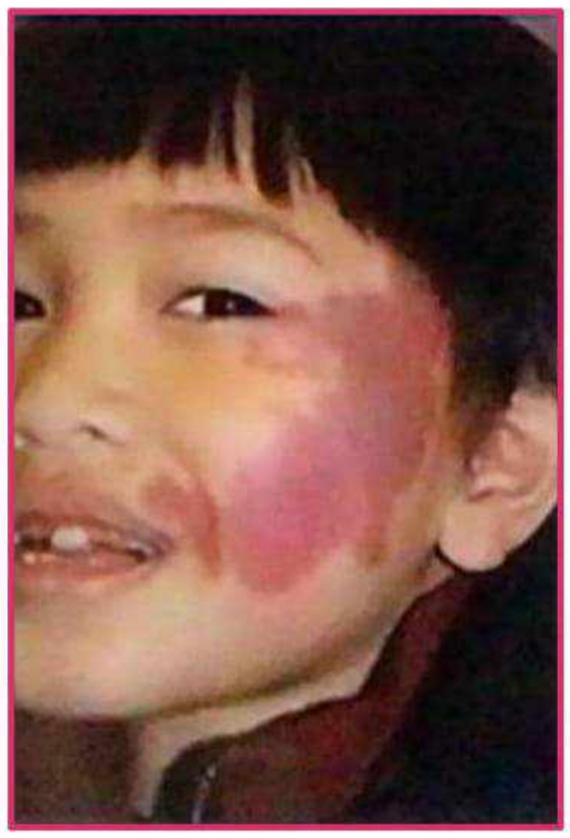
24:20

- a.k.a Nevus flamneus
- it is due to capillary malformations
- Present at birth, persistent in nature ⟶ donot disappear

active space

- Site : face
- Sharply unilateral distribution.



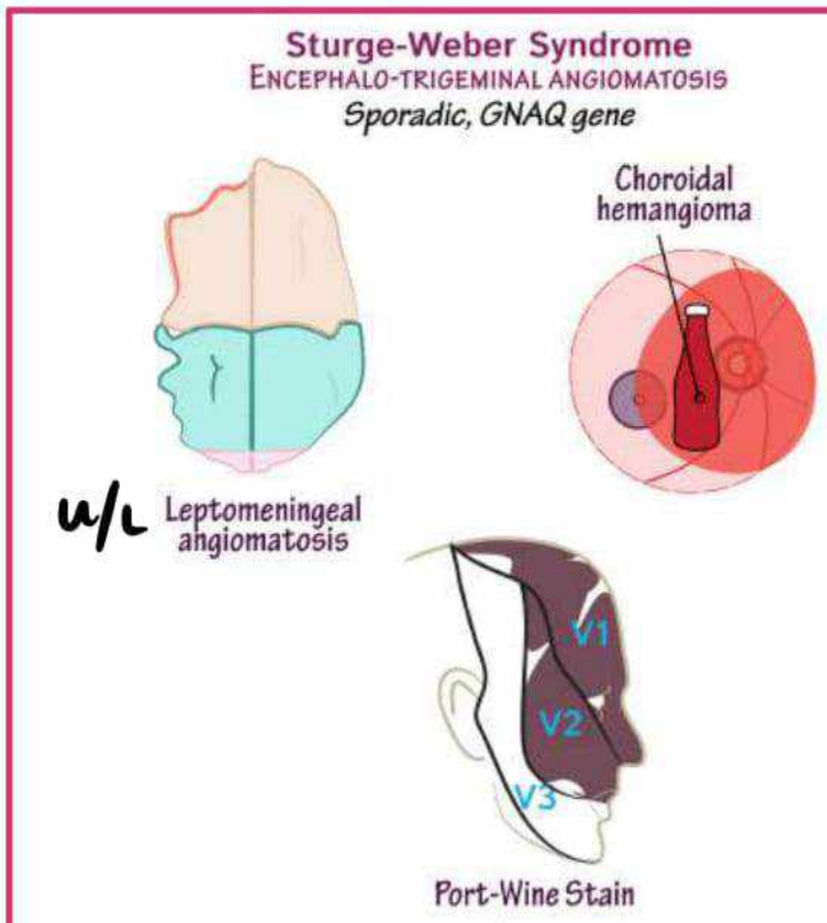


Begins as flat lesion, U/L distribution

↓

Becomes more darker, thicker, corrugated (cobblestone)

Treatment → LASER (PDL)



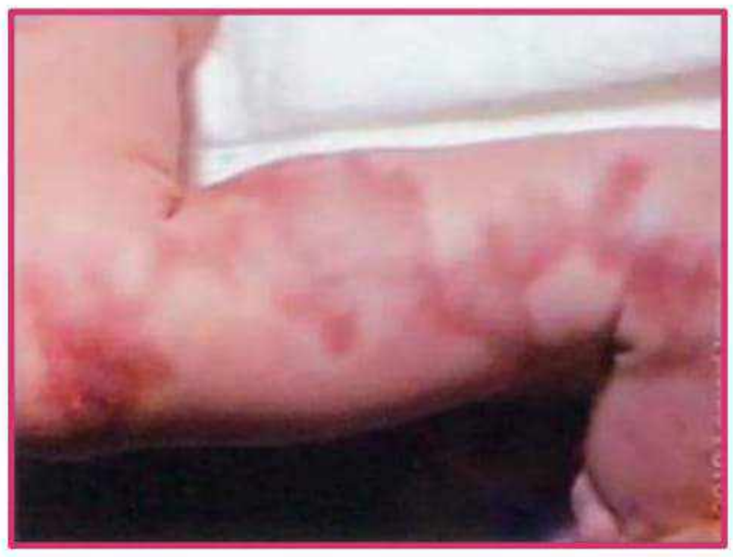
- Portwine Stain → distribution of Trigeminal nerve.
- U/L leptomeningeal angiomatosis
- Convulsions, behavioural abnormality, Sub Normal intelligence.
- Strabismus, vision loss.

Klippel - Trenaunay Syndrome (KTS) : Triad of

- Port wine stain
- venous and lymphatic malformations
- Bony and soft tissue hypertrophy of an extremity.

Cutis Marmorata Telangiectata Congenita

- a.k.a **Congenital Livedo Reticularis**
- It is **mixed capillary and venous malformation**
- extremities most commonly involved parts, followed by the trunk and face.



Clinical feature

- Reticular nevus → Reticular / Marbled
- **erythematous plaques, purplish hue.**
- overlying telangiectasias

Cutis marmorata

- physiological response to cold
- seen in children/adults
- It is transient

active space

MASTOCYTOSIS

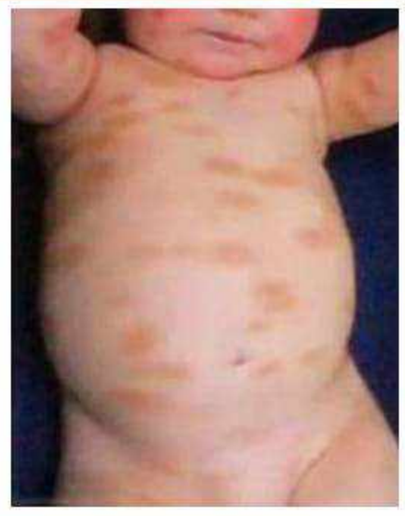
32:00

- collection of mast cells ⟶ skin, bone, other organs
- Mastocytosis limited to the skin is primarily a disease of children.
- On HPE : Collection of mast cells around dermal blood vessels
- Stains with Toluidine blue
- Cutaneous mastocytosis : Types
  - i) Urticaria pigmentosa
  - ii) Solitary mastocytoma
  - iii) Diffuse cutaneous mastocytosis
  - iv) Telangiectasia macularis eruptiva perstans.

URTICARIA PIGMENTOSA

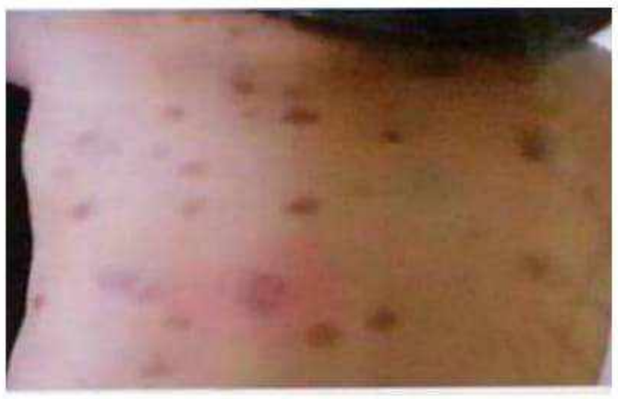
33: 33

- commonest form of mastocytosis
- usually no systemic involvement
- Onset : by age of 2yrs



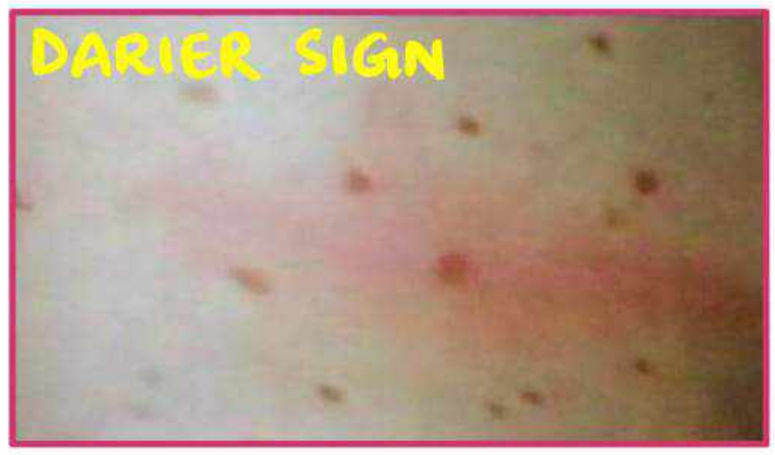
Clinical feature

- Erythematous maculopapular lesions with margins which are not sharp



active space

- seen on trunk.
- Resolve leaving hyperpigmented macules.
- " DARIER SIGN "



- On stroking → a lesion of UP appears

↓

This erythema and wheals appear

within mins

↓

Subsides in 30mins

- Happens due to degranulation of mast cells → Release Histamine

- On Biopsy: Mast cells can be seen

Treatment

- Avoid triggers → - Anxiety, emotional stress, Physical force.
  - Drugs: NSAIDs, Radiocontrast dyes.
- Antihistamines,
- Ketotifens.

HISTIOCYTOSES 39:20

- includes disorder resulting from a proliferation of cells of monocyte - macrophage lineage

active space

- It is a multisystem disease
- Class I : Langerhans cell histiocytosis → Proliferation of langerhans cells.

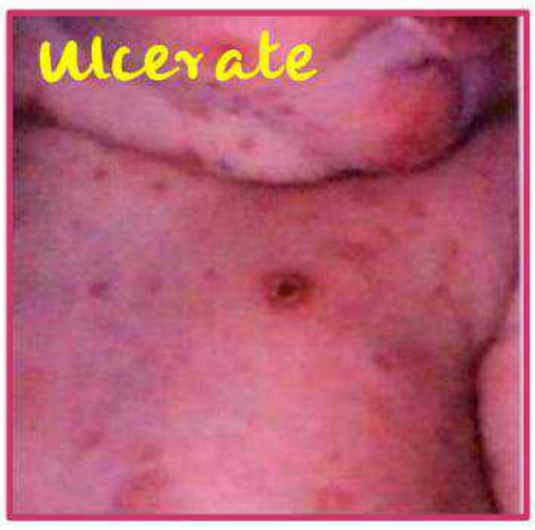
LANGERHAN CELL HISTIOCYTOSIS

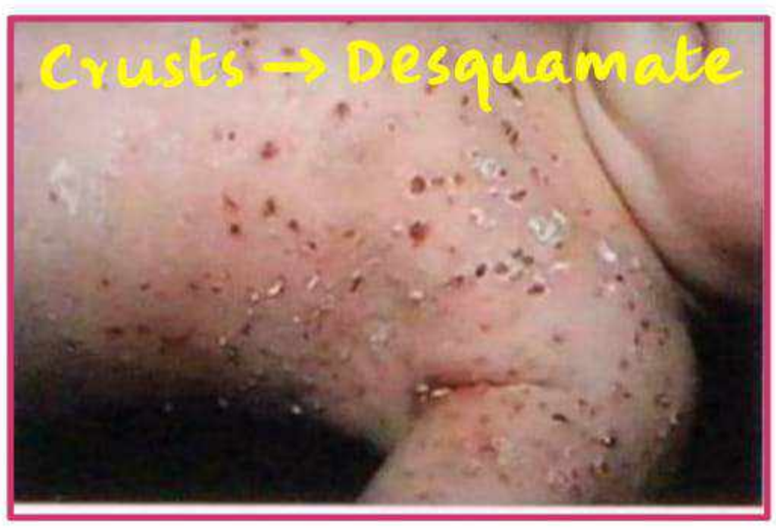
40:22

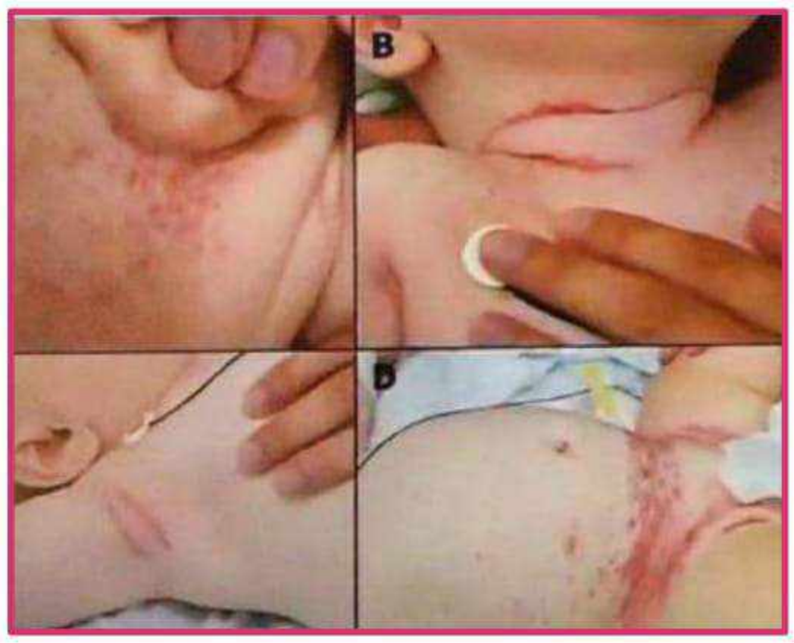
- Clonal proliferative disease of langerhans cells
- Langerhans cell characteristics : CD_{1a} , S100 +ve.
  - ↳ Birbeck granules.
- Multiorgan involvement :
  - Bone, skin , LN, Lungs, Spleen and Liver, Endocrine glands and nervous system.
- Traditional Types:
  - Eosinophillic granuloma (localized lesions confined to the bones) - older children / adults
  - Hand-Schüller - Christian Disease (classical triad of skull defects , Diabetes insipidus and exophthalmos) seen in 2-6yrs.
- Letterer - Siwe disease (visceral and skin involvement) seen in 0-2yrs.

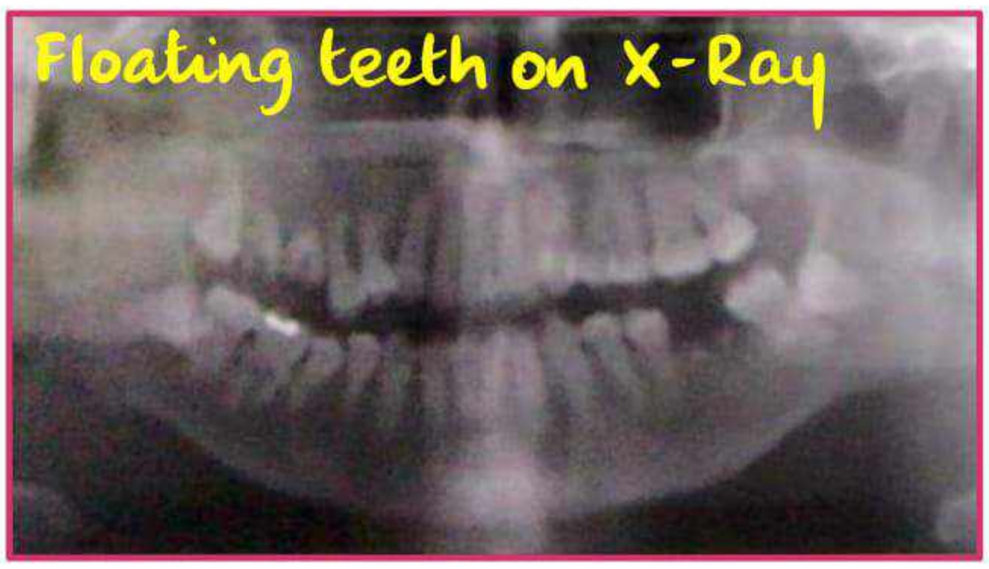
active space

- **unifying feature:** presence of rod shaped cytoplasmic structures indistinguishable from Bierbeck granules of Epidermal Langerhans cell.
- Age : 1-3yrs
- **Symptoms:** fever, failure to thrive
- **Cutaneous features :** Seborrhoeic dermatitis like.
  - papules, vesicles and pustules present in seborrhoeic distribution → Scalp, post auricular, face, flexures
  - forms crust, painful.









active space

- Visceral involvement :
  - Lymphadenopathy
  - Bone → skull, long bones.
  - Liver
  - Eye
- Three markers are expressed by LCH cells but not by normal epidermal Langerhans cells : peanut agglutinin, an epitope shared with IFN α and placental alkaline phosphatase.

Treatment

- Limited involvement of the skin is left untreated
- Extensive and symptomatic cutaneous changes : Prednisolone therapy.
- Recalcitrant ulcerated plaques : PUVA Therapy and Topical Nitrogen Beam therapy.
- Chemo and Radiation therapy for systemic disease
- Vinblastine and corticosteroids are the current standard of treatment of mutlisystem disease in children.

active space

Systemic Diseases and Skin (Part - 1)

NUTRITIONAL DISORDERS

00:55

1. PEM
2. Deficiency of vitamins
3. Deficiency of minerals

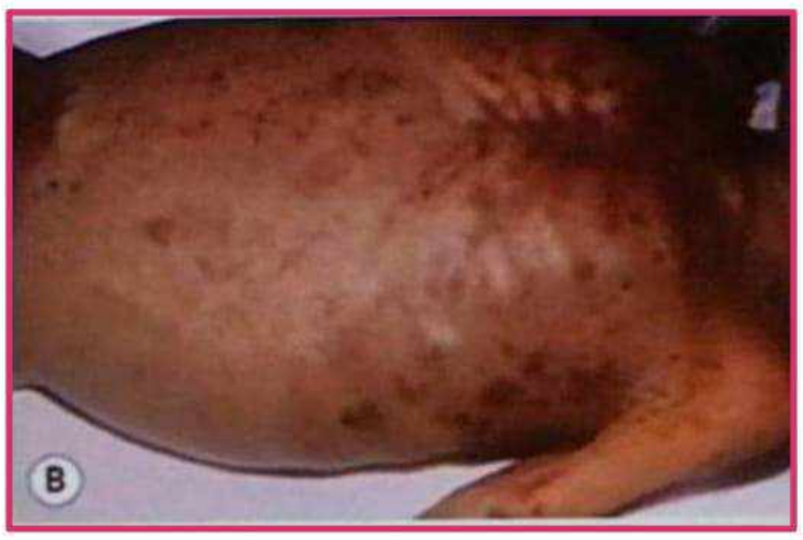
MARASMUS

- **Skin manifestation:** Dry, inelastic, lustreless.
- **Hair:** Brittle, lustreless.
- **Facies:** Monkey facies (loss of Buccal fat)

KWARSHIORKAR

02:00

- **Skin features:** Enamel paint/ Flaky paint/ Crazy pavement pattern.
- Hyperpigmented lesions on torso – Burnt appearance flaky skin.



active space

- In Mucosa : Glossitis, Cheilitis
- In Hair : Flag sign → Alternate bands of Normal and decreased pigmentation. Corresponds to periods of malnutrition.

VITAMIN A DEFICIENCY

04:55

- Earliest sign : Night Blindness
- Earliest skin manifestation : Asteatosis
- Phrynoderma (Toad Skin) - due mixed deficiency (Vit. A,B,C,D,E and fatty acids) - follicular hyperkeratotic grouped papules with horny spine on elbows, knees, postero lateral aspect of thighs and arms.

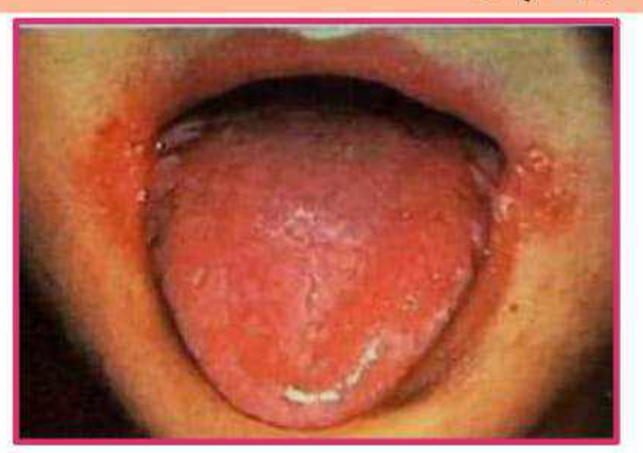
Treatment → 50,000 - 2,00,000 IU of Vit A

Hypervitaminosis : deposition of carotenoids → CAROTENEMIA
↓
Yellowish hue on skin.

VITAMIN B2 DEFICIENCY (RIBOFLAVIN)

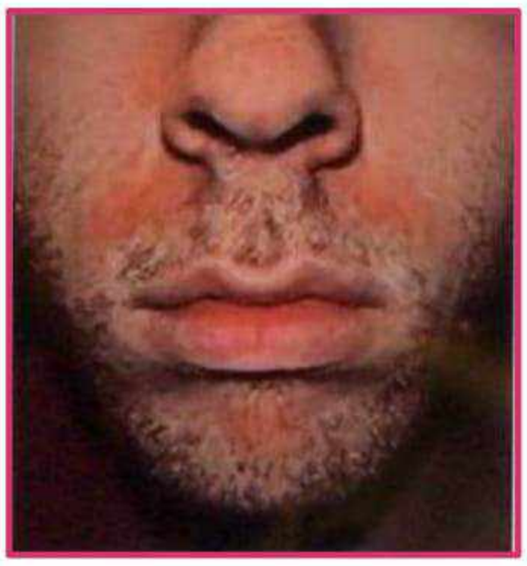
07:45

- Leads to Angular cheilitis (Perleche)
- Cheilosis
- Atrophic glossitis (Strawberry tongue).



active space

- Seborrhoeic dermatitis like rash / Dyssebacea



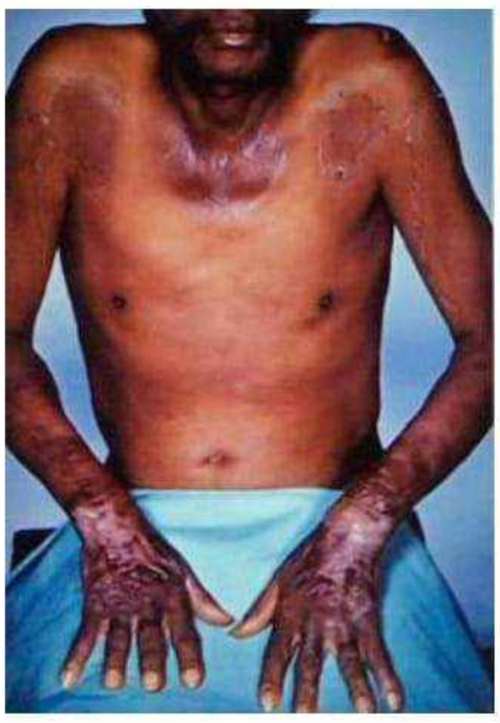
VITAMIN B3 (NICOTINIC ACID, NICOTINAMIDE, NIACIN) DEFICIENCY

09:20

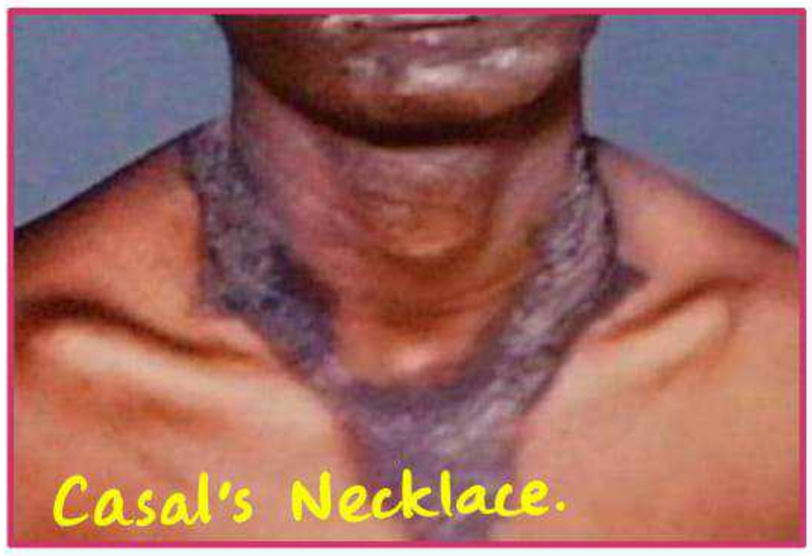
- it gives rise to Pellagra
- 4 D's →
  - Diarrhoea
  - Dermatitis
  - Dementia
  - Death.
- (Mc) seen in people on staple diet of maize and jowar, chronic alcoholic.

Symptoms:

- Pruritis
- Burning sensation → Sunburn like feeling
- Sites → Acral, Face, Neck (photoexposed site)
- In Acute phase: Erythematous eczematous lesions



- In Chronic phase : Plaque like scaly lesions.



- CARAVAT SIGN : when lesions extend beyond chest.
- PELLAGRIN'S NOSE
- CASAL'S NECKLACE : involvement of neck and anterior chest
- On mucous membranes : Glossitis, Cheilitis
- GIT : Anorexia, Nausea, Vomiting, Diarrhoea
- CNS : Psychosis, Depression

Treatment → 500mg/day of Nicotinamide.

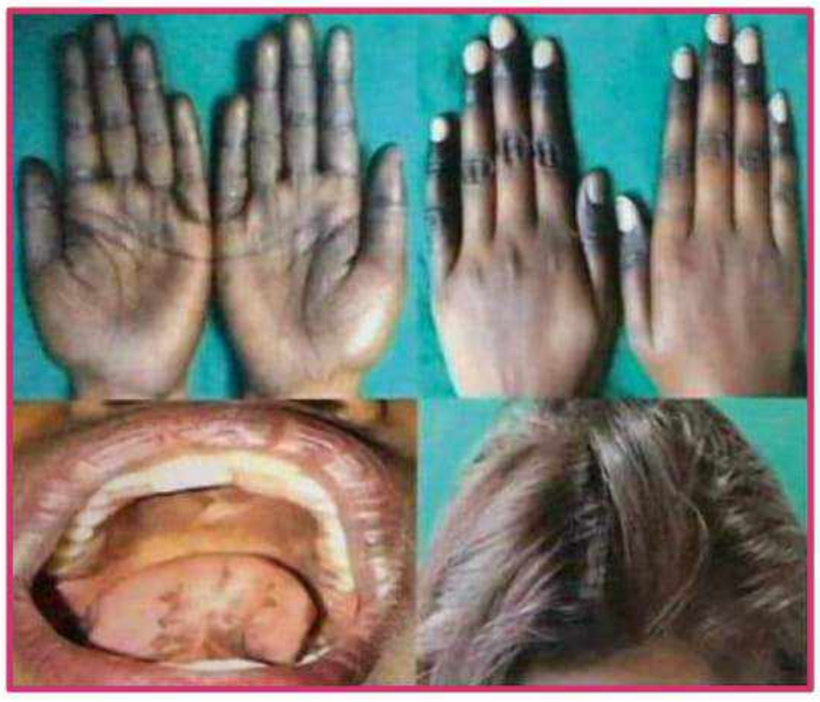
VITAMIN B12 DEFICIENCY

14:30

- leads to Addisonian pigmentation :

  Hyperpigmentation on palmar creases, nails, knuckles, mucosa, flexures.

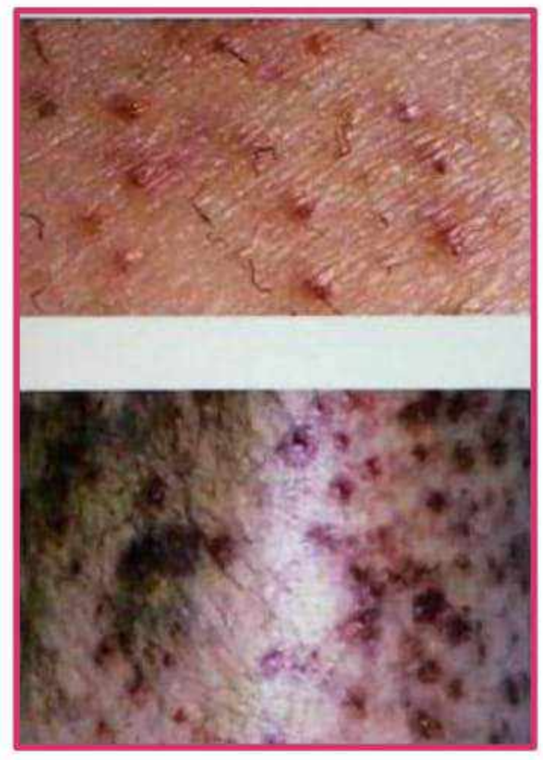
- Premature canites → Greying of hair.

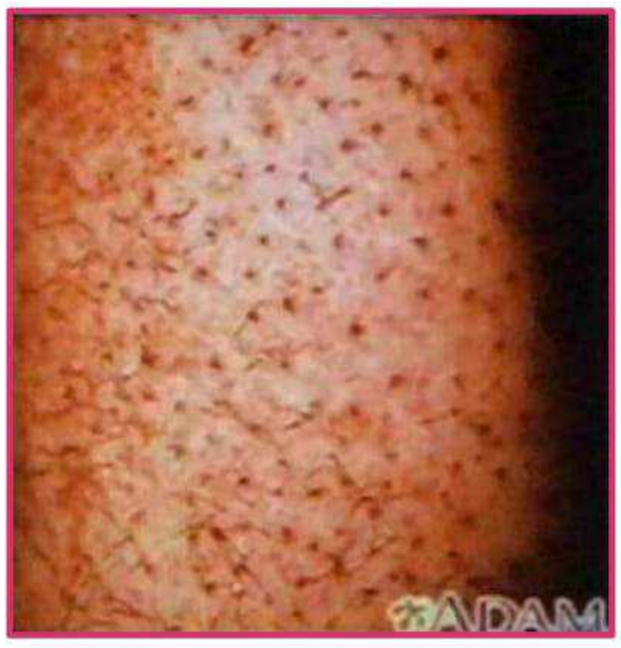


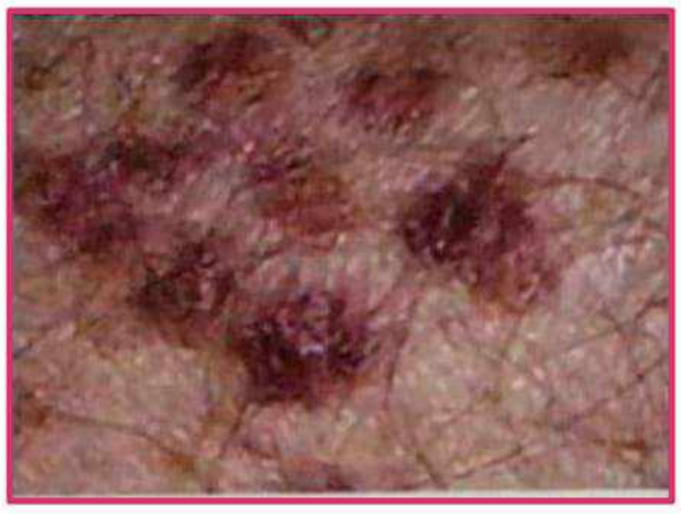
VITAMIN C DEFICIENCY

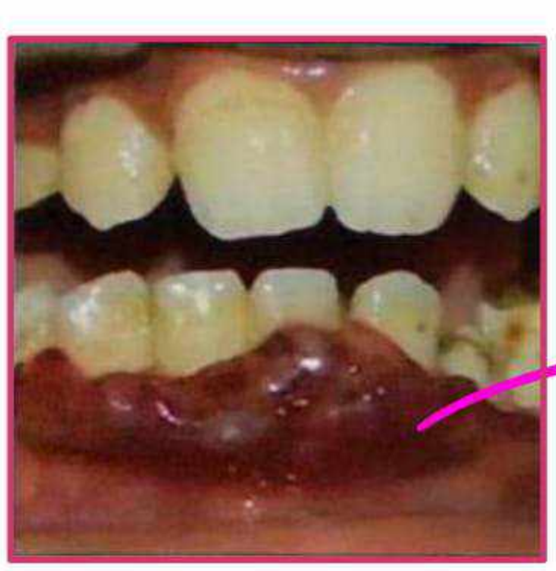
15:45

- Leads to follicular hyperkeratosis
- Corkscrew hair (Breakage of Disulphide Bonds)
- Petechiae and purpura → Bruises
- Perifollicular hemorrhages.



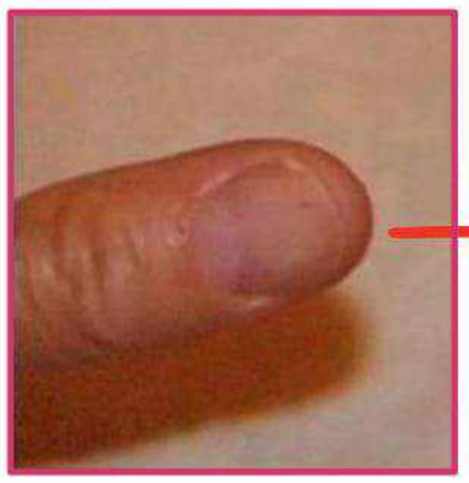






→ Hemorrhagic gingivitis → H/o Bleeding gums.

↳ a.k.a Spongy gums

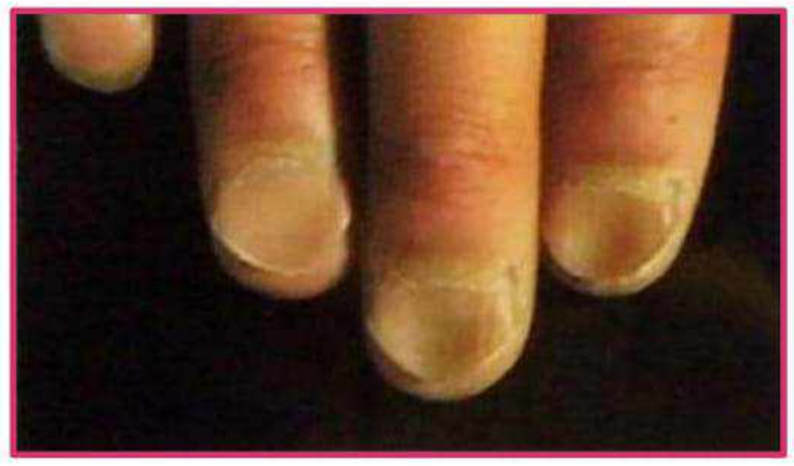


→ Splinter hemorrhages.

active space

IRON DEFICIENCY

18:13



- spoon shaped nails (KOILONYCHIA)
- Hair fall → Telogen effluvium. (chronic)
- itchy dry skin, pruritus.

ZINC DEFICIENCY

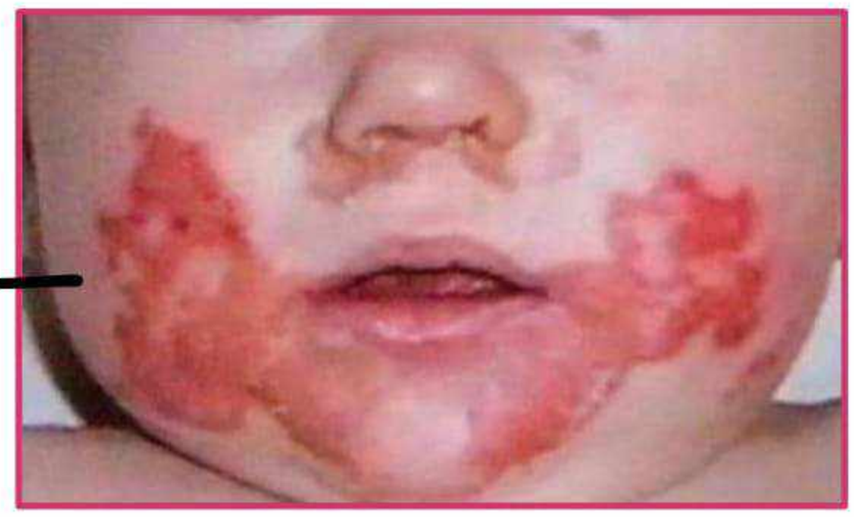
19:15

- Genetic : Acrodermatitis enteropathica → occurs at infancy
  ↓ Weaned of from mother's milk
- Acquired : occurs at any age.

Acrodermatitis Enteropathica

- Inheritance : Autosomal Recessive.
- Etiology : Defective Intestinal Zinc transporter → ZIP4.
- Onset : Infancy after 6months
- Triad : 1. Dermatitis
  2. Diarrhoea
  3. Alopecia.

Dermatitis : Acro-orificial eruption ←
↓
Sites : periorificial, perianal, acral areas.

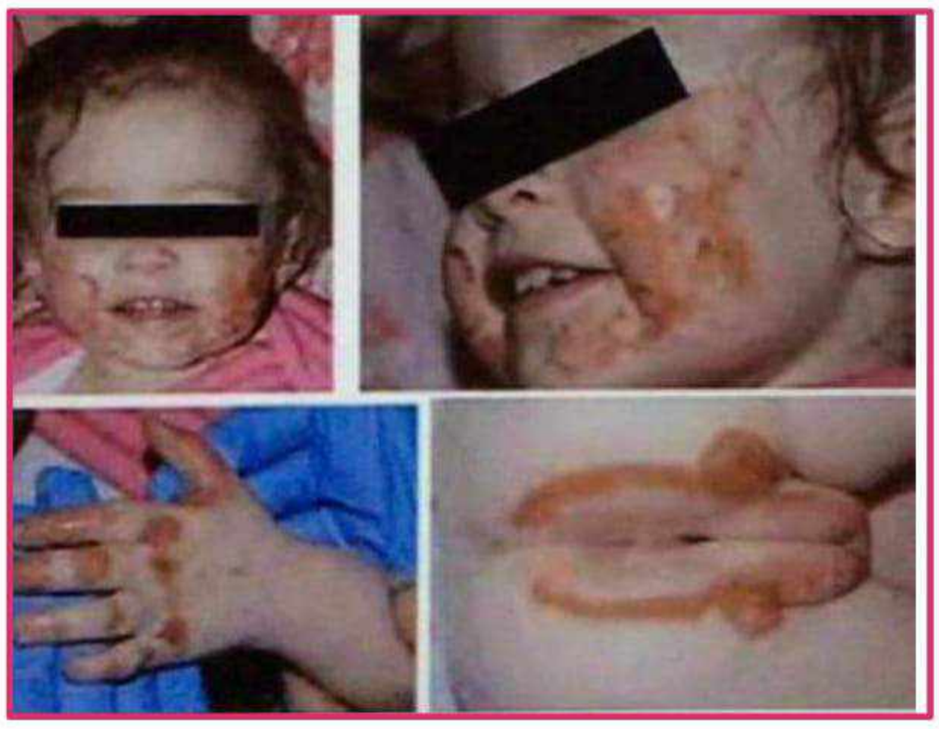


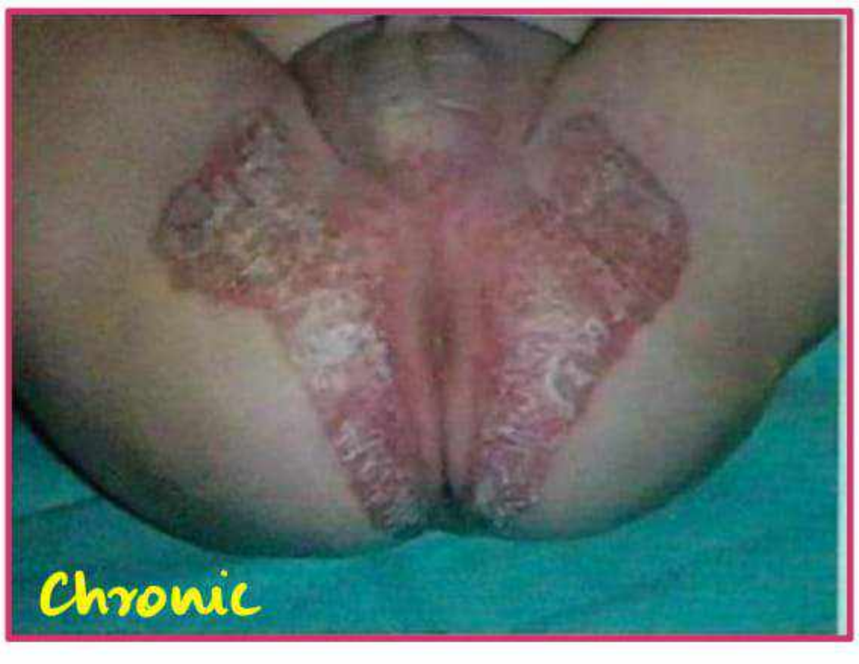
active space

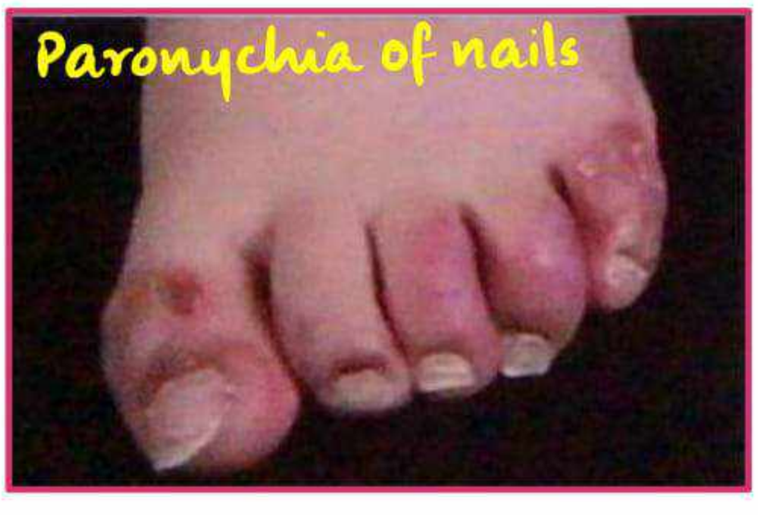
Dermatitis

Acute
- Erythematous crusted plaques

Chronic
- Psoriasiform dermatitis







- Associated features :
  - Diarrhoea
  - Alopecia

Treatment → 2mg/kg/day of Zinc

METABOLIC DISORDERS

DIABETES MELLITUS 24:00

Skin lesions seen are :
- Acanthosis nigricans
- Diabetic dermopathy

- Necrobiosis lipoidica
- Granuloma annulare
- Limited joint mobility
- Sclerederma diabeticorum
- Eruptive xanthomas
- Perforating disorders
- Bullous diabeticorum
- Bacterial and fungal infections
- Pruritus
- Diabetic bulla.

DIABETIC DERMOPATHY

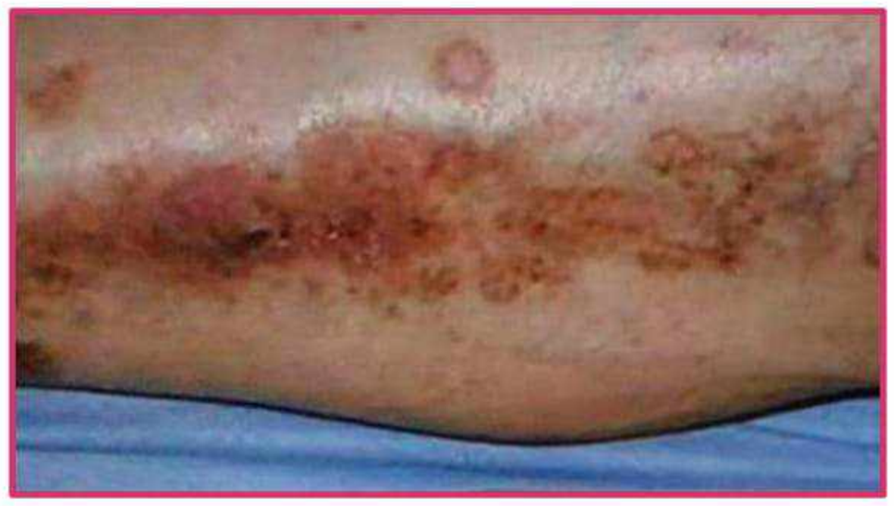
25:12

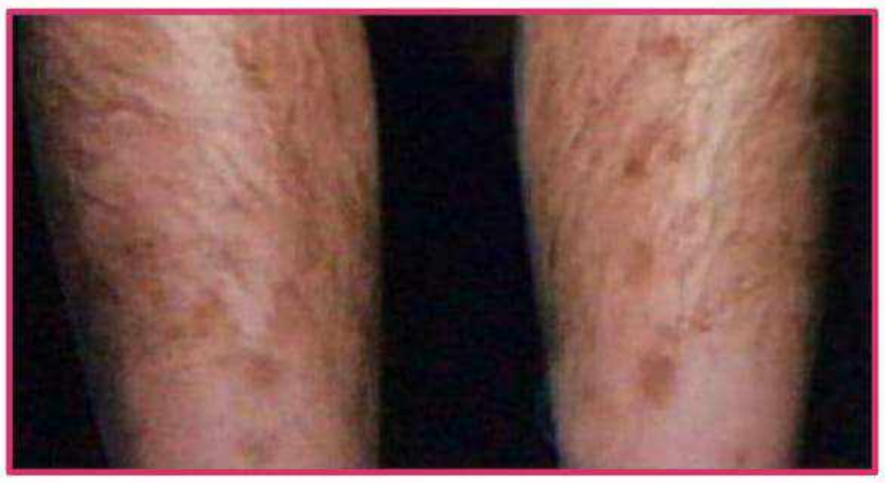
- Marker of underlying retinopathy, nephropathy.
- Brownish atrophic scaly papules and plaques present on shins Bilateral

↓

Heal with brownish atrophic scars.

usually asymptomatic.

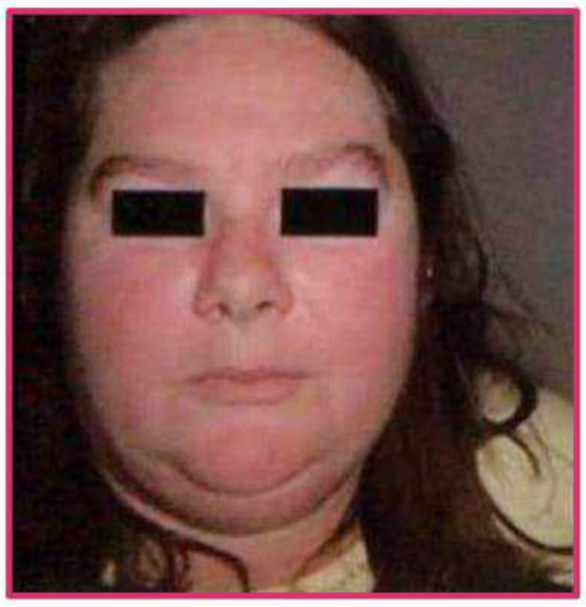




active space

Diabetic Rubeosis

- Rosy swelling on the face → Microangiopathy.

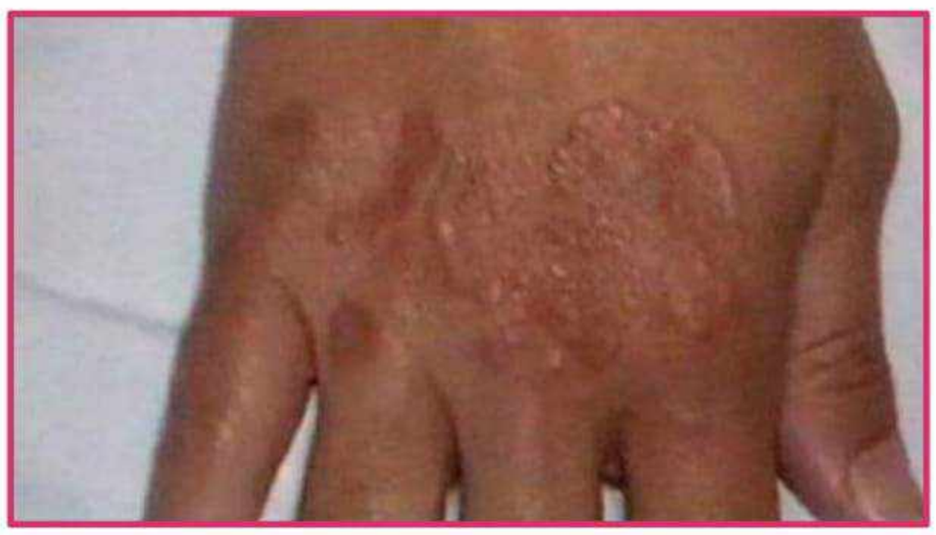


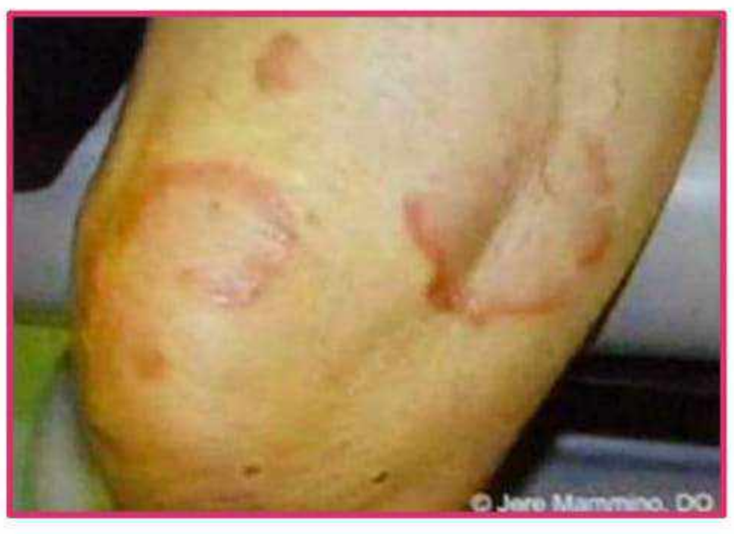
Granulomatous disorders in DM.

1. Necrobiosis lipoidica → Specific
2. Granuloma annulare. → Non-specific.

GRANULOMA ANNULARE 27:30

- Associated diseases : DM, Autoimmune thyroiditis
- On HPE : Necrobiotic granuloma → Necrosis of Collagen.
  ↓
  surrounded by lymphocytes and histiocytes





Symptoms

- Asymptomatic ± pruritus
- Sites : Dorsa of Hands and feet
- Annular papules and plaques with reddish brown granulomatous look with a normal overlying surface

central clearing is seen

- Course : Spontaneous resolution
- variants : i) Generalised → HIV
  ii) Subcutaneous
  iii) Perforating

Treatment : Topical steroids.

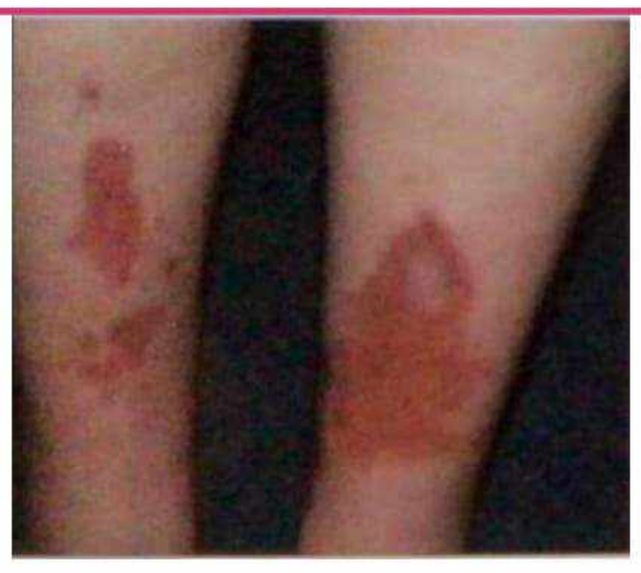
NECROBIOSIS LIPOIDICA 31:45

- altered collagen → lipid like
- marker of underlying Retinopathy, Nephropathy.

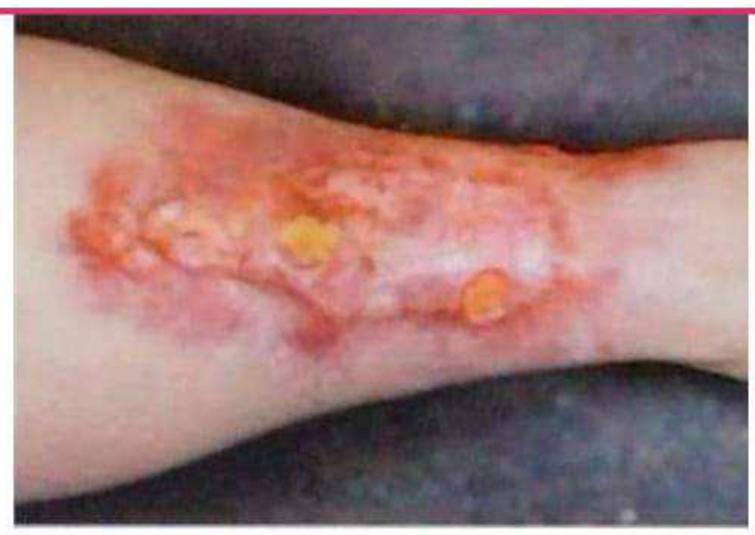
Clinical feature :

- Site : Shins, B/L symmetrical
- Asymptomatic
- if ulcerated → painful.

active space



- Erythematous papules and plaques on shins



- Atrophic and yellowish
- Associated telangiectasias. waxing looking

- **On HPE:** Necrobiotic condition, full thickness lymphohistiocytic infiltrate

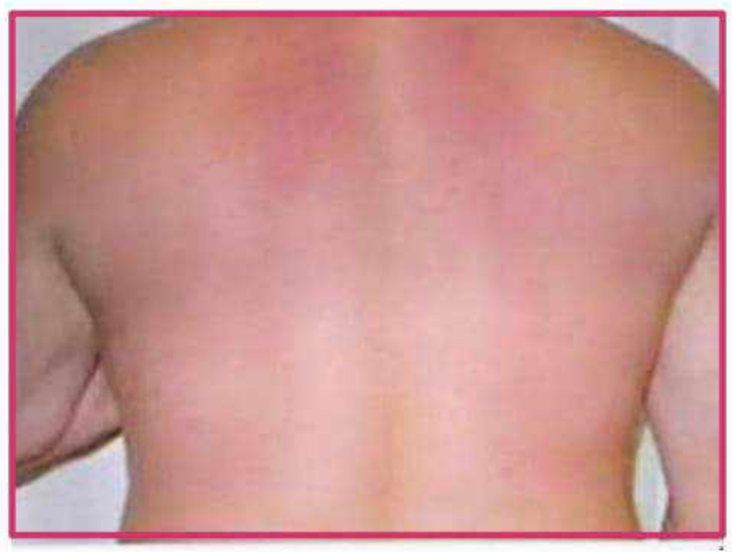
Treatment:

- unsatisfactory
- Topical steroids.

SCLEREDERMA DIABETICORUM

34:25

- Diffuse thickening and induration of upper part of body.
- Not specific
- commonly seen in Obese.



active space

DISORDERS OF THYROID GLAND

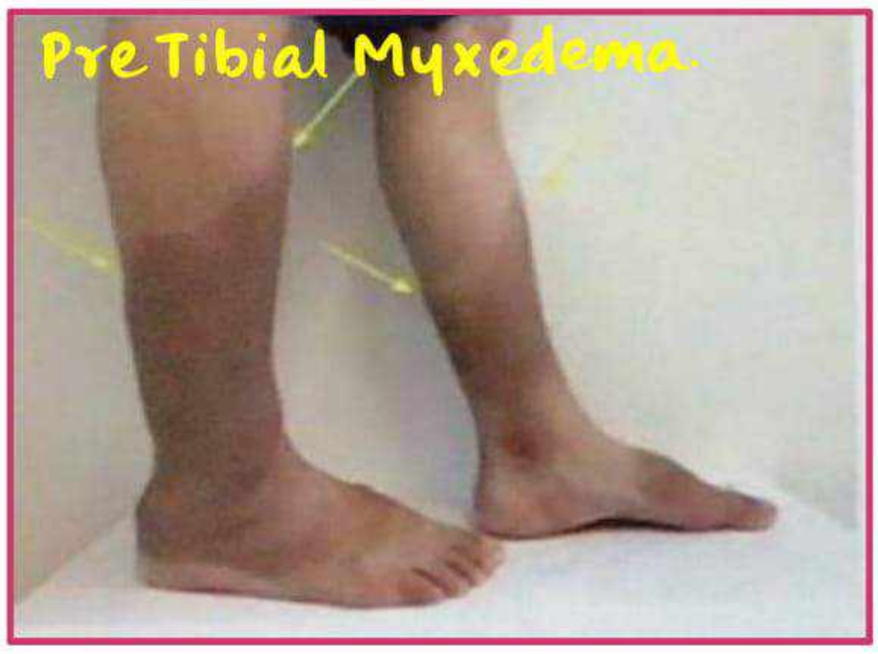
35:00

- Hyperthyroidism → Grave's disease
- Hypothyroidism → Autoimmune / Hashimoto's thyroiditis.

- Hair fall → CTE
- Pruritus
- Dryness of skin.

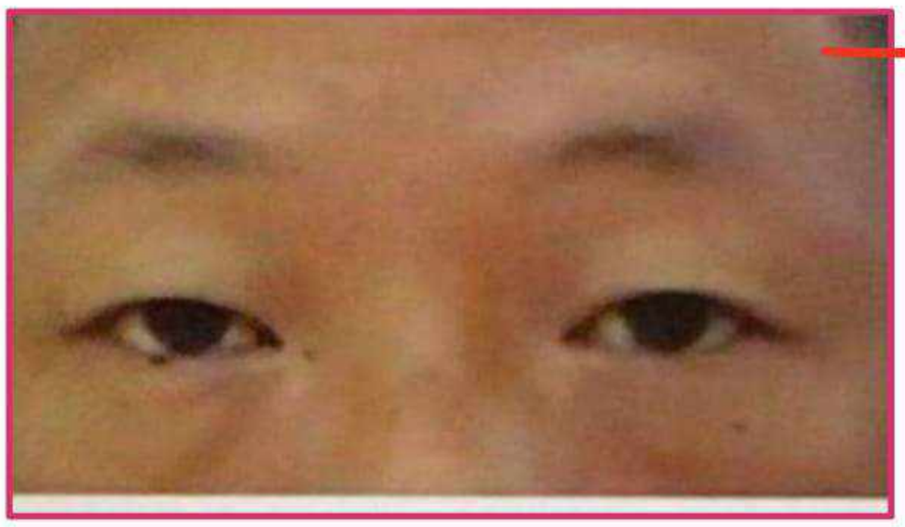
Pretibial Myxedema

- seen in Graves disease + Hashimoto's thyroiditis.
- Marker of Ophthalmopathy.
- Non-pitting edema with erythematous plaques on shins
- Bilateral.
- Deposition of Glycosaminoglycans.



HASHIMOTO'S THYROIDITIS

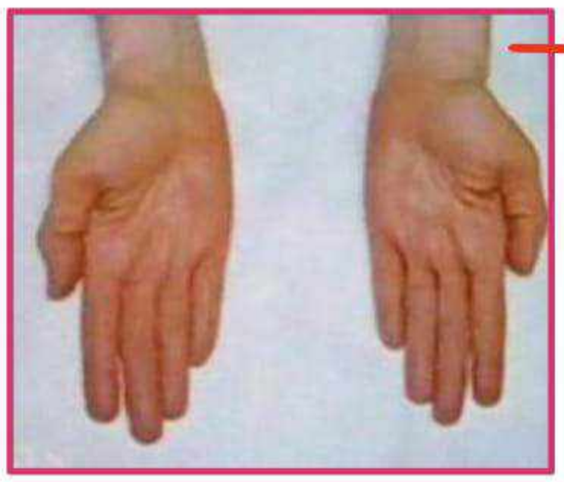
38:00



→ **HERTOGHE'S SIGN:**

- loss of lateral 1/3rd of eyebrows
- seen in i) Atopic dermatitis
  ii) Lepromatous leprosy.

active space



→ Yellowish hue on skin due to deposition of carotenoids.

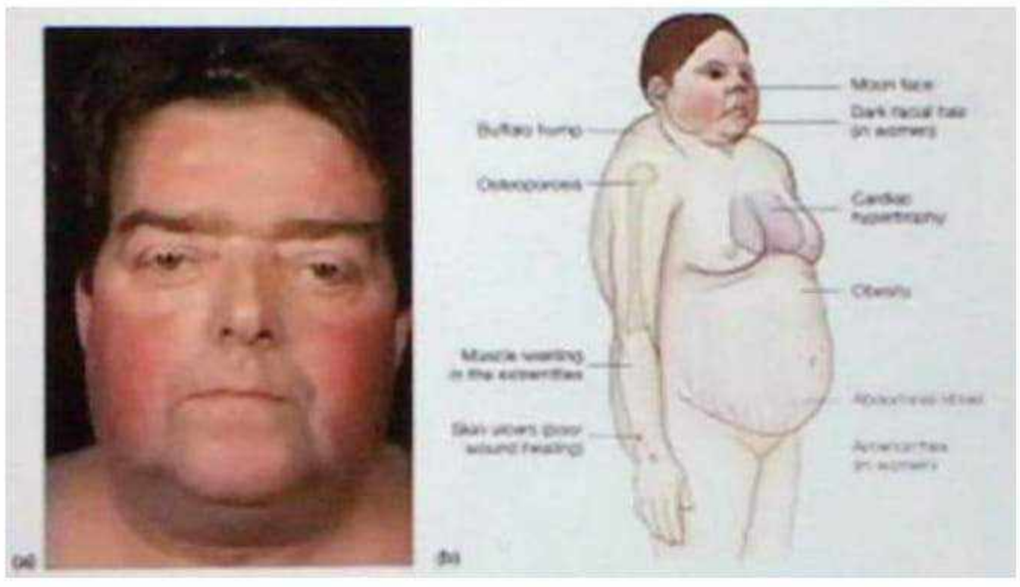
DISORDERS OF ADRENAL GLANDS

38:40

CUSHING'S SYNDROME :

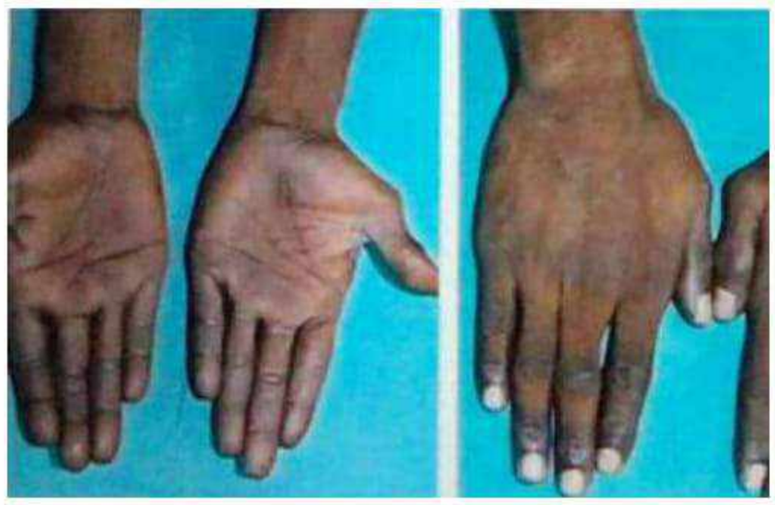
Clinical feature :

- Moon facies
- Buffalo Hump
- Truncal obesity
- Abdominal striae.
- Acanthosis nigricans



ADDISON'S DISEASE (Adrenal Insufficiency) :

- Addisonian pigmentation
- Diffuse pigmentation.



active space

Renal failure

- pruritus, dry, scaly skin.
- Tendency to develop purpura/ecchymoses on minor trauma
- Half and Half nails
- **Pale yellow skin :** associated anemia and pitting edema due to accumulation of urochrome or carotene pigments
- **Uremic frost :** deposition of urea crystals on the nose and malar area due to high urea levels
- Calcinosis cutis, pseudoporphyria cutanea tarda, nephrogenic fibrosing dermatopathy.

Reno-cutaneous disease

- SLE
- Systemic Sclerosis
- vasculitides (Henoch Schonlein, Wegner's, Polyarteritis nodosa)
- Lepromatous leprosy.

active space

SARCOIDOSIS

41:15

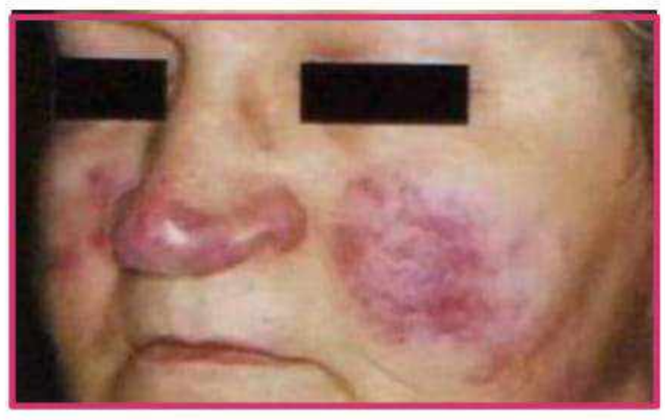
- Granulomatous disorder which affects internal organs and skin.
- **Etiology :** Antigen mediated disorder
- **Organs affected :** Pulmonary system, Peripheral LN, Skin, Eyes.

Cutaneous lesions

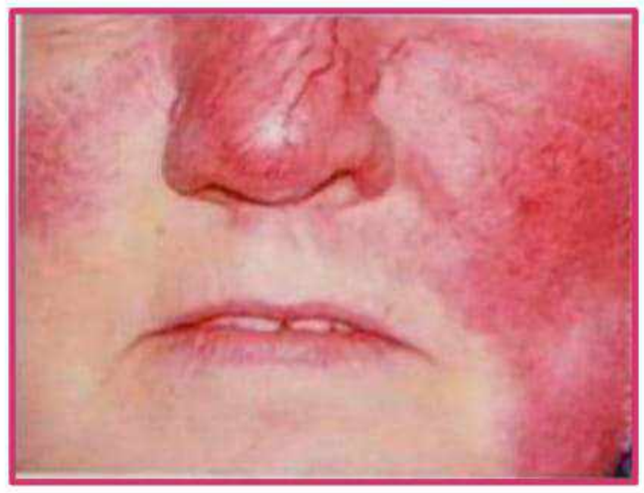
- Non-specific → Erythema nodosum
- Specific → Sarcoidal granulomas on Biopsy
  - → Red to brown papules with normal surface.
  - → On Diascopy : Apple jelly nodules.
    - ↳ (in Lupus vulgaris → More opaque)

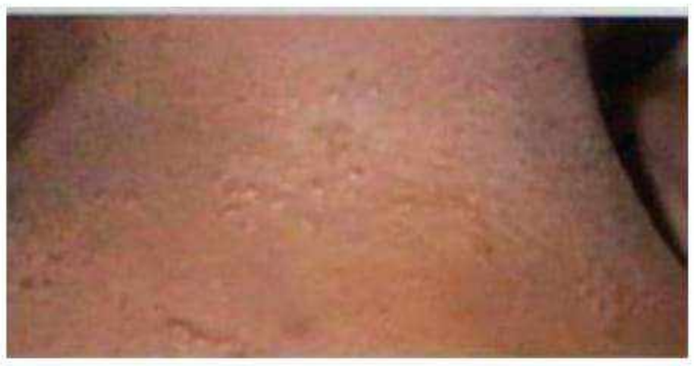
Specific lesions

1. **Lupus pernio :** Sarcoidosis seen on bridge of nose, cheeks, forehead
   - Erythematous slightly infiltrative plaques with telangiectasias.

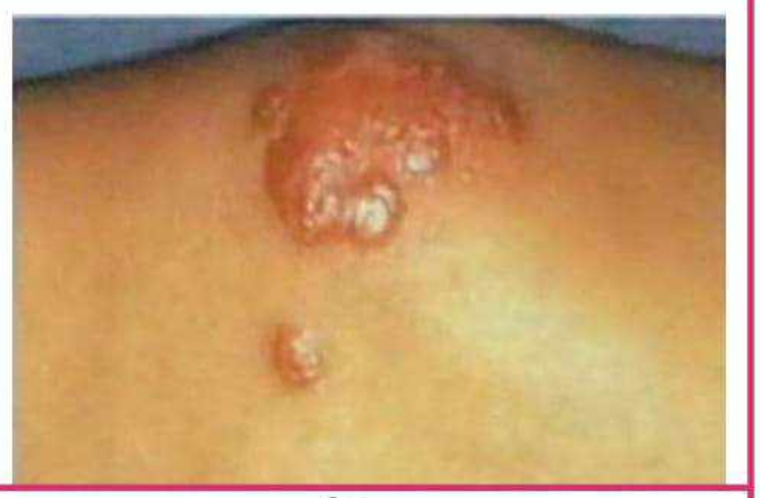


- **Angiolupoid sarcoidosis :**
  - Erythema and Telangiectasias seen on bridge of nose.

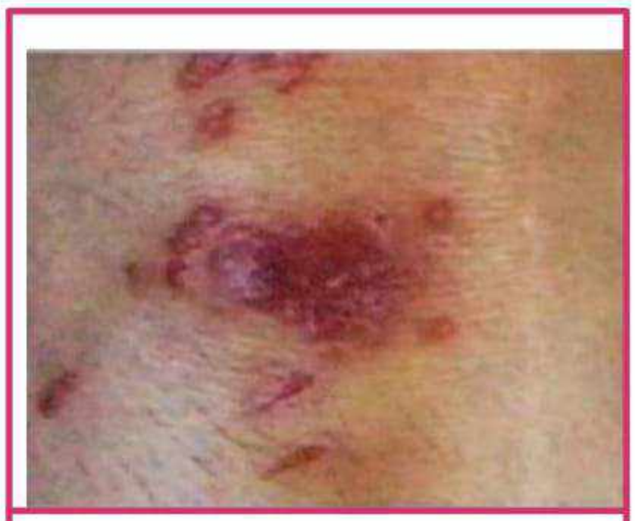




Maculopapular sarcoid



Nodular / Plaque Sarcoid.



Scar Sarcoid.

- **On HPE :** characteristically naked granulomas
  - Necrobiotic granulomas
  - very sparse lymphocytic infiltrate around them.
  - Giant cells → Schaumann bodies and Asteroid bodies

Treatment

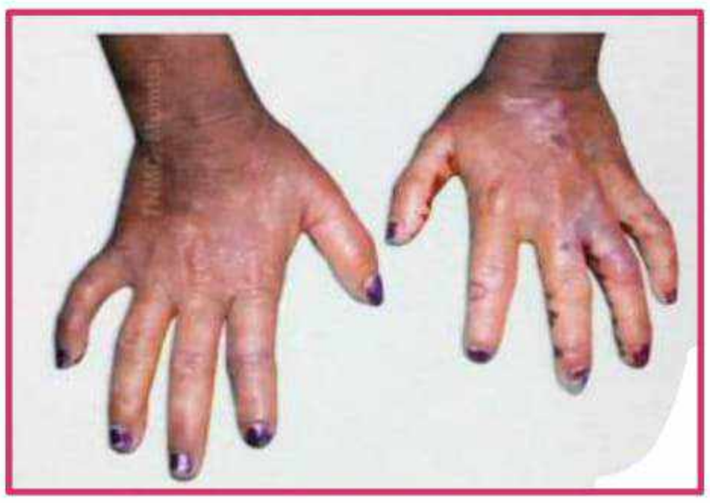
- Topical / Intralesional corticosteroids.
- Antimalarials.

PORPHYRIAS

46:20

Cutaneous disease only

- Porphyria cutanea tarda
- Congenital erythropoietic porphyria
- Erythropoietic protoporphyria.



active space

Cutaneous disease and Acute attacks

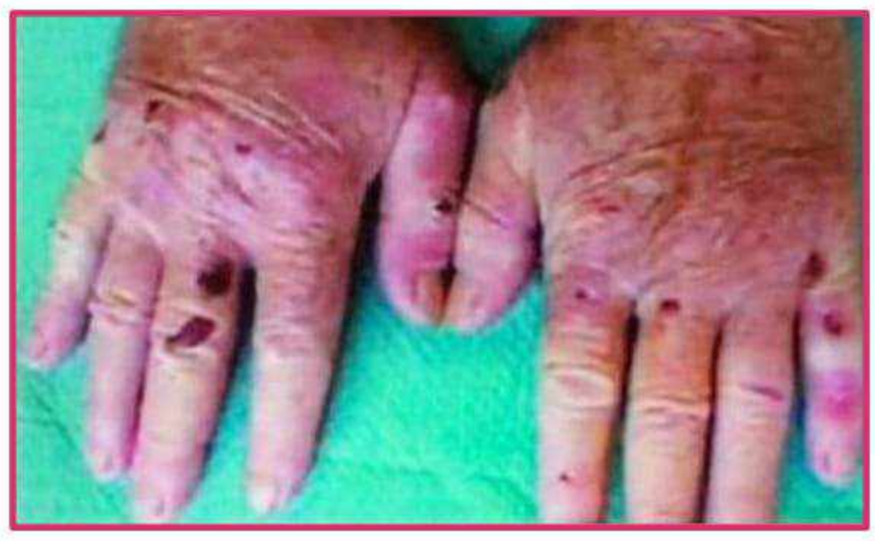
- Hereditary Coproporphyria
- Variegate porphyria

Acute attacks only

- Acute Intermittent Porphyria.

Porphyria Cutanea Tarda

- **Cause:** Uroporphyrinogen carboxylase deficiency
- Age of Onset: 3rd - 4th Decade



Clinical features

- vesicles and crusted lesions on sites of repeated trauma
- Healing ⟶ Milia formation, Sclerodermoid appearance.

Treatment → Phlebotomy, Antimalarials.

active space

Systemic Diseases and Skin (Part - 2)

PARANEOPLASTIC DERMATOSES

00:30

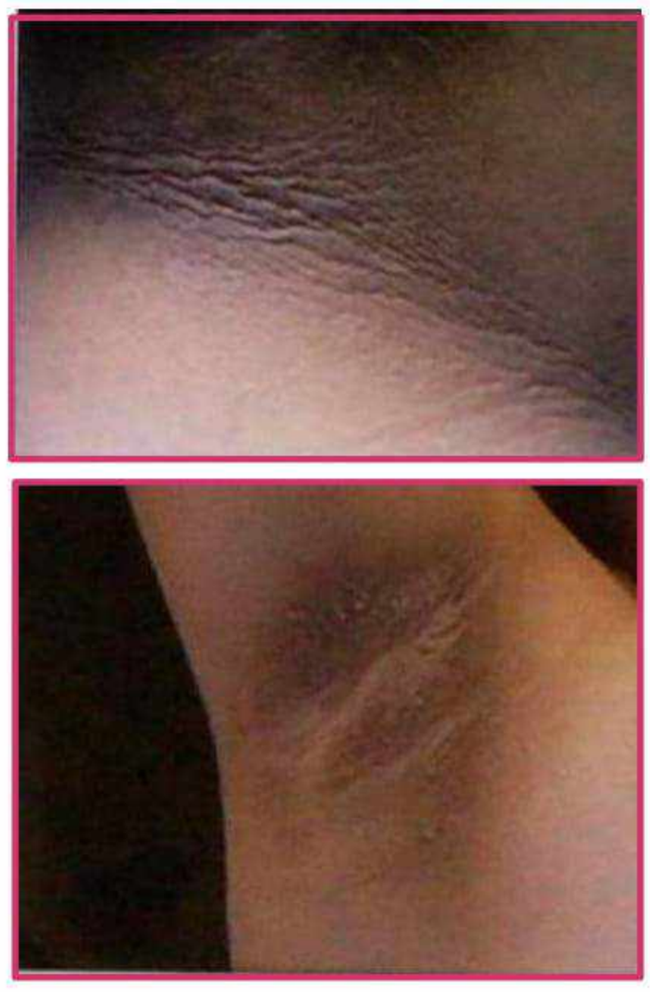
- are skin lesions associated with internal malignancies, but are not themselves malignant.

Acanthosis nigricans

- misnomer : No acanthosis on HPE

Clinical feature

- Asymptomatic hyperpigmented velvety texture over the flexures.



- Associations :
  - Obesity
  - DM
  - Metabolic syndrome → Insulin resistance.
  - Drugs → OCP's.
- Associated findings : Skin tags over Acanthosis nigricans.

active space

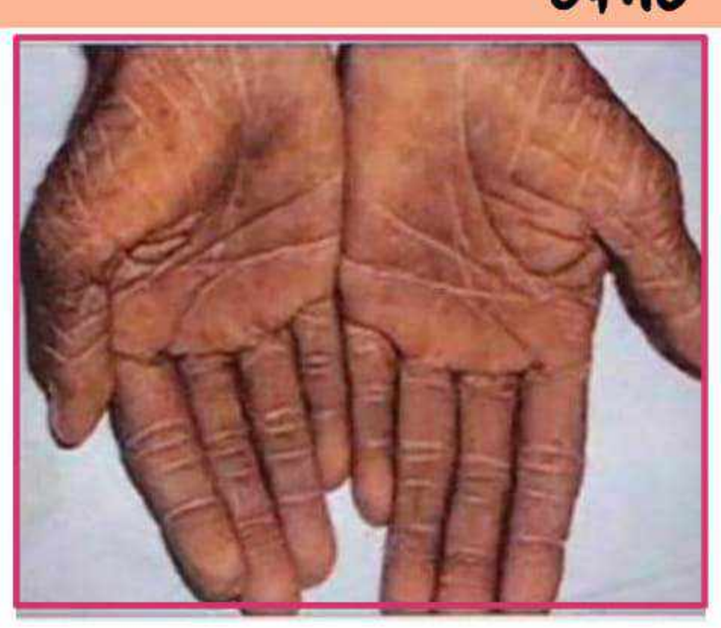
- Pathogenesis: ↑ IGF-1
- 2 Types → Benign
  → Malignant

Malignant AN

- Rapid in onset
- Generalised, B/L symmetrical
- associated with adenocarcinoma of GIT
- Associated findings:
  - Tripe palms
  - Sign of Lesev Trelat

TRIPE PALMS

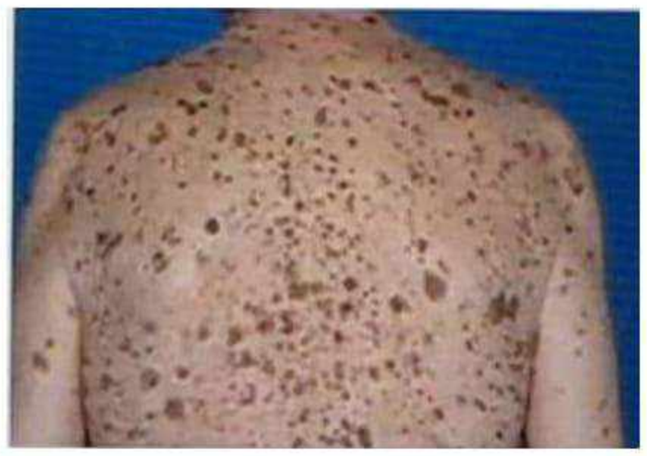
07:10



- Acanthosis over palms → Acanthosis palmaris
- Dermatoglyphics.
- when present alone with clubbing } Bronchial CA
- When present in association with AN } Adenocarcinoma of GIT

active space

Sign of Trelat



- Sudden eruption of multiple SKS over the body
- Neoplasm of GIT should be suspected.
  ↓
  Adenocarcinoma
- Acquired Icthyosis → Hodgkin's lymphoma
- Clubbing and associated hypertrophic osteoarthropathy } Bronchial CA.
- Paraneoplastic pemphigus → B cell lymphomas and Thymomas.

MIGRATORY ERYTHEMA

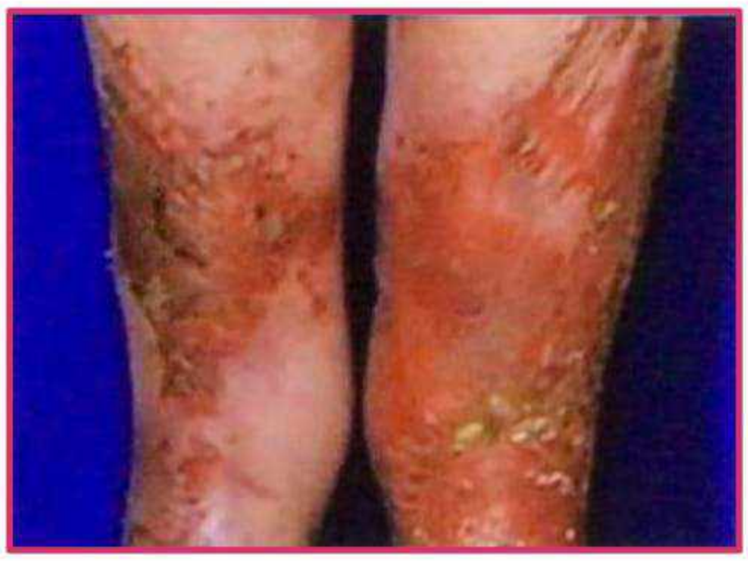
10:23

1. Erythema gyratum repens → associated with Lung CA
2. Necrolytic Migratory Erythema → associated with Glucagonoma

Erythema gyratum repens

- concentric rings of erythematous plaques which has palpable migratory margins
  ↓
  1cm/day.
  + scaling.
- Trunk predominantly involved.

Necrolytic Migratory Erythema



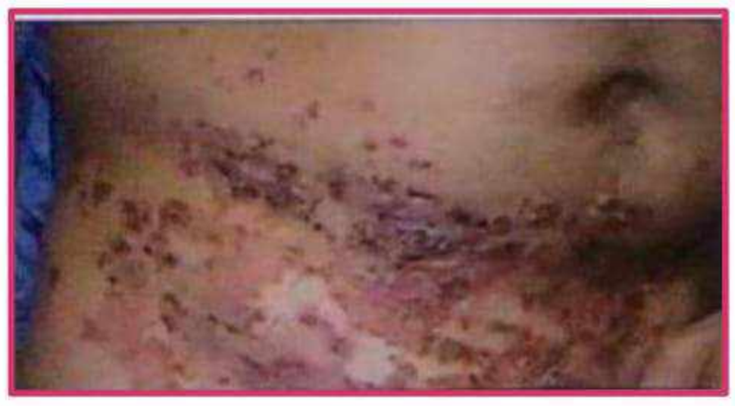
- Lesions are painful and tender
- Recurrent polycyclic eruptions acute, inflammatory, erythematous, eczematous

↓

Superficial vesiculations

↓

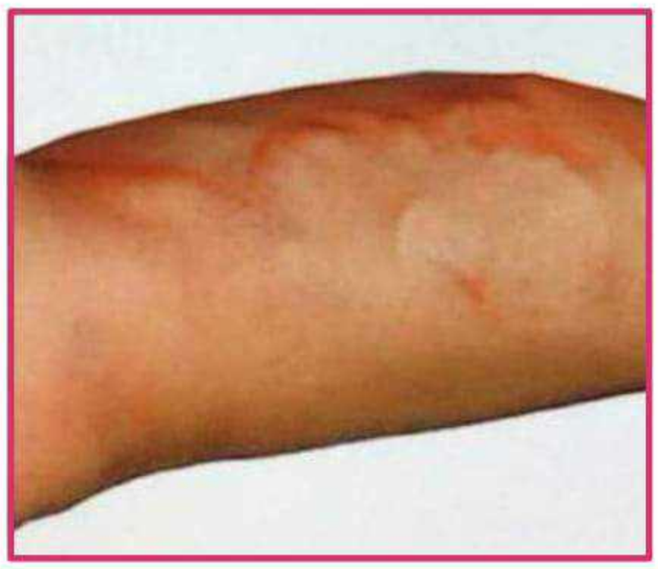
Necrolysis.



- seen on anogenital area, trunk.

MIGRATORY THROMBOPHLEBITIS

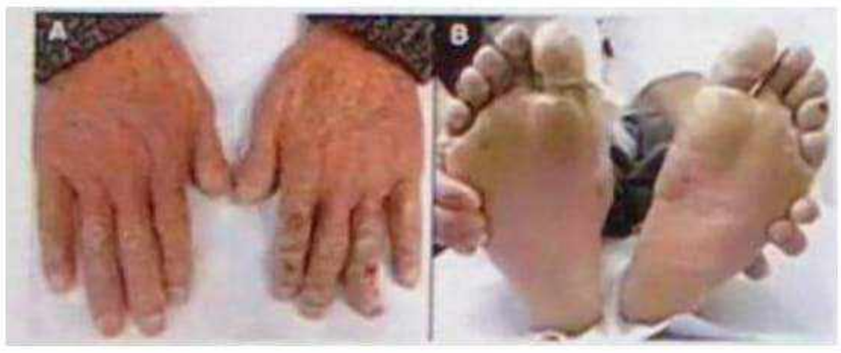
15:50



- a.k.a Trousseau's sign.
- seen in Pancreatic carcinoma.

BAZEX SYNDROME

17:10



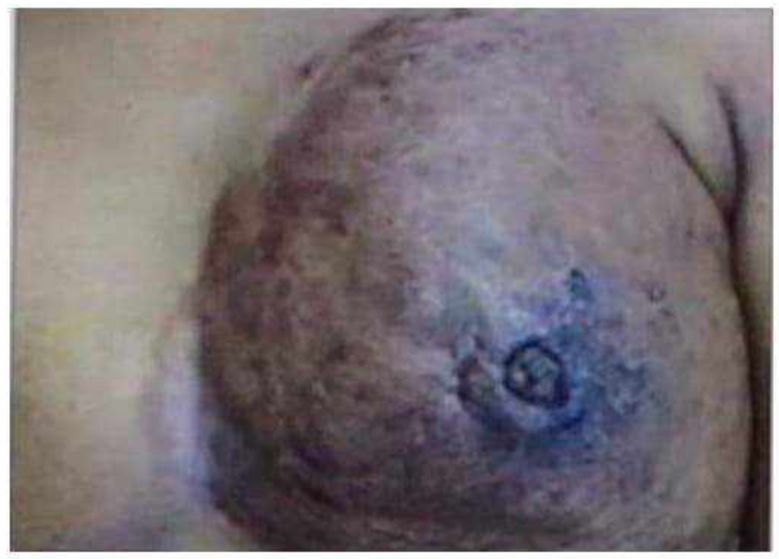
- a.k.a Acrokeratosis paraneoplastica
- SCC of Bronchial, G.I Tract
- Keratoderma of fingers and toes.

active space

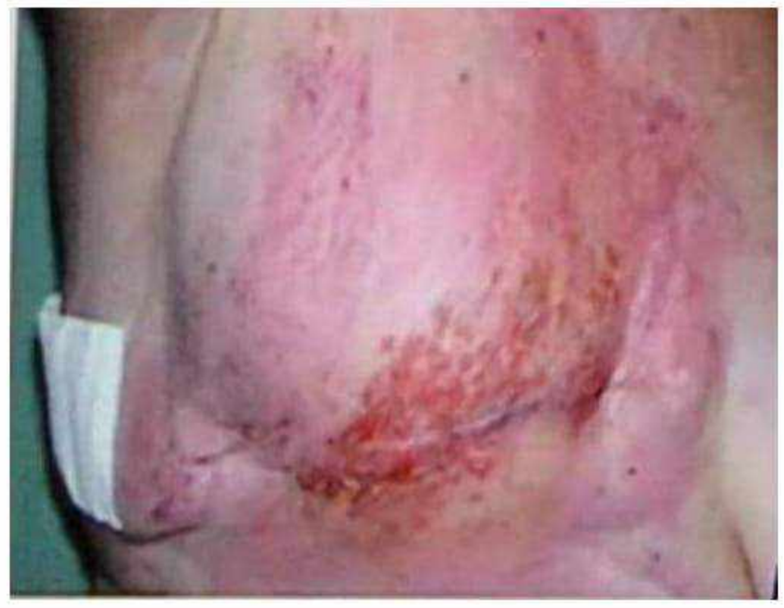
Direct Tumor Spread and Invasion

- usually associated with Breast cancer

- **Carcinoma en Cuirasse.**
  - Sclerodermoid appearance due to direct tumor spread.



- **Carcinoma erysipeloides**
  - Erysipela like appearance of overlying skin.



PAGETS DISEASE OF NIPPLE 19:20

- Origin : Intraductal carcinoma of Breast (Apocrine derived epithelial cells)
- characteristic cells → Paget cells seen in Biopsy

Symptom

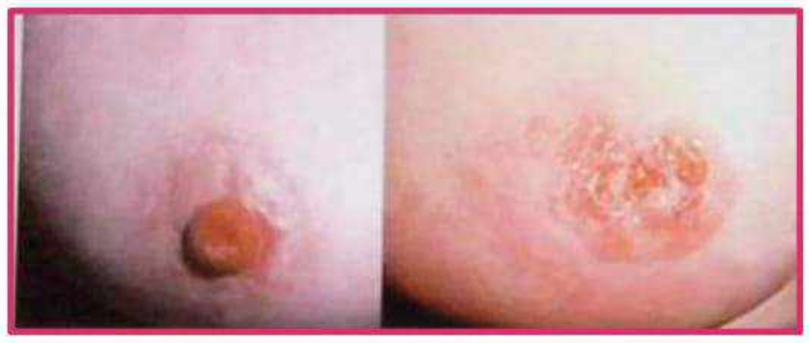
- Itching

active space

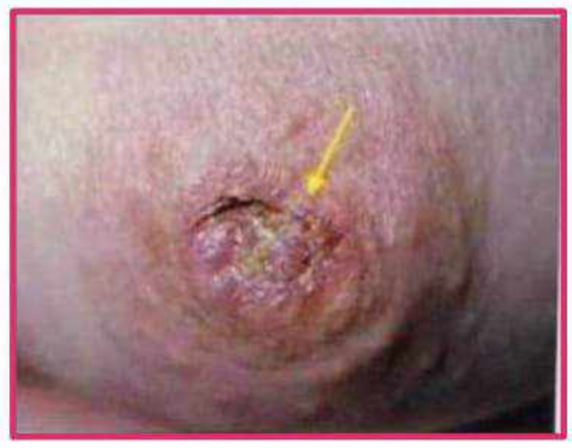
Start small erythematous papule

↓

Crusted erythematous plaque



- U/L, sharply defined margins
- Sometimes there is bloody to serous discharge.



- D/D's :
  - Eczema → B/L, well defined margins

    Response to topical steroids

Extramammary Paget's

- Pagets disease involving other apocrine duct bearing areas → groins and axilla

CUTANEOUS METASTASIS

- B → Breast
- L → Lung
- O → Oesophageal
- C → Colon
- K → Kidney

} carcinomas presents with cutaneous metastasis.

- Head and Neck → (MC) site
- Sister Mary Joseph's nodule seen around umbilicus

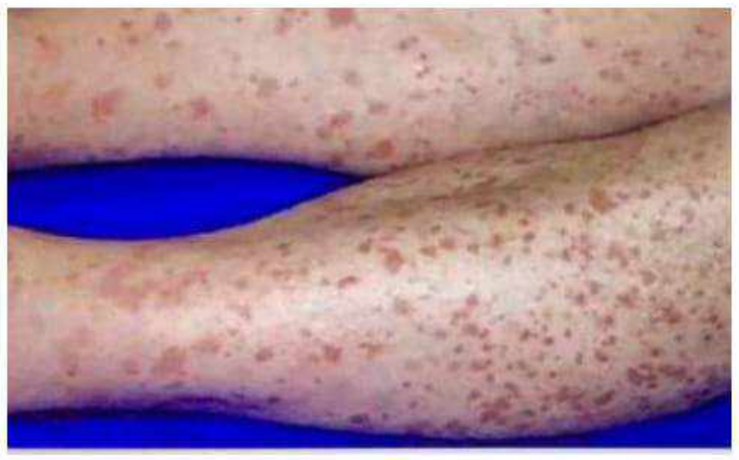
INFLAMMATORY DERMATOSES WITH SYSTEMIC ASSOCIATIONS

VASCULITIS

- Inflammation of vessels → Systemic
  → Cutaneous
- Classical lesion of cutaneous vasculitis : Palpable purpura.
- Nomenclature: Revised at the 2012 Chapel Hill Consensus conference is based upon the size of blood vessel affected.

Clinical feature

- Palpable purpura + painful
- Associated systemic feature ⊕ → fever
- Site : lower limb



Box 102.2 Areas in the history of a patient with cutaneous vasculitis that may give clues indicating systemic disease

- Weight loss, fatigue, fever
- Arthralgia, myalgia, arthritis
- Dry eyes, dry mouth
- Red eye, eye pain, vision loss
- Nasal or sinus congestion
- Ear pain
- Oral/nasal ulcers
- Chest pain/dyspnoea
- Abdominal pain, blood in faeces
- Blackouts, weakness, fits

active space

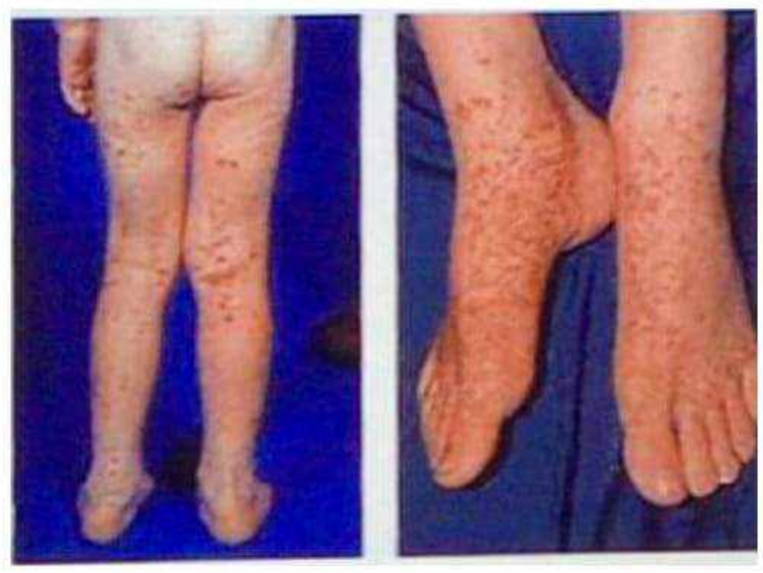
CUTANEOUS SMALL VESSEL VASCULITIS 30:45

- purely cutaneous in nature
- a.k.a leukocytoclastic vasculitis (CSVV/LCV)
- Idiopathic
- Palpable purpura → lower limb.
- Self limiting.

HENOCH SCHONLEIN PURPURA 32:00

- Systemic small vessel vasculitis
- Age : childhood
- Etiology : ℞ Streptococci, Infection, Drugs
- Features of leukocytoclastic vasculitis -
  - The disease is mediated by IgA-containing immune complexes
- Organs affected :

1. Skin : - Palpable purpura
   - painful
   - seen on lowerlimb, lower part of trunks.



active space

2. G.I Tract → Nausea, vomiting, Abdominal pain, Bloody Stools

3. Joints → Polyarthralgia

4. Kidneys → Hematuria, Proteinuria, RBC casts in urine.
   ↳ Self Limiting.

Treatment

- Steroids
- Dapsone
- Medium vessel Vasculitis → PAN, Kawasaki
- Large vessel → Giant cell, Takayasu.

Physical signs may give clue as to the predominant vessel size involved in vasculitis

Blood vessel size	Physical Signs
Small Blood vessels	Purpuric macules and papules, haemorrhagic vesicles, urticarial plaques
Large vessel vasculitis	Broken livedo (net / Reticulate pattern) Infarction, ulceration, deep nodules.

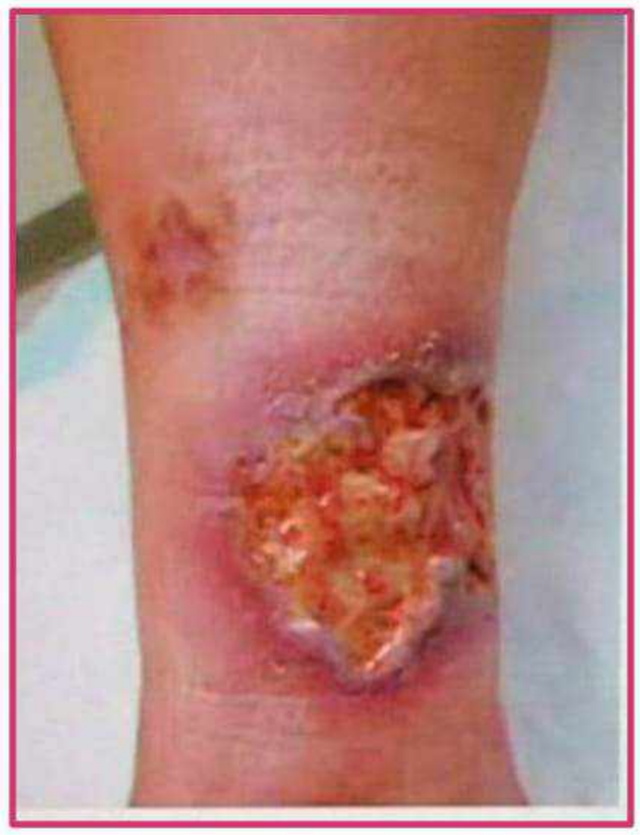
active space

Neutrophilic dermatoses

PYODERMA GANGRENOSUM

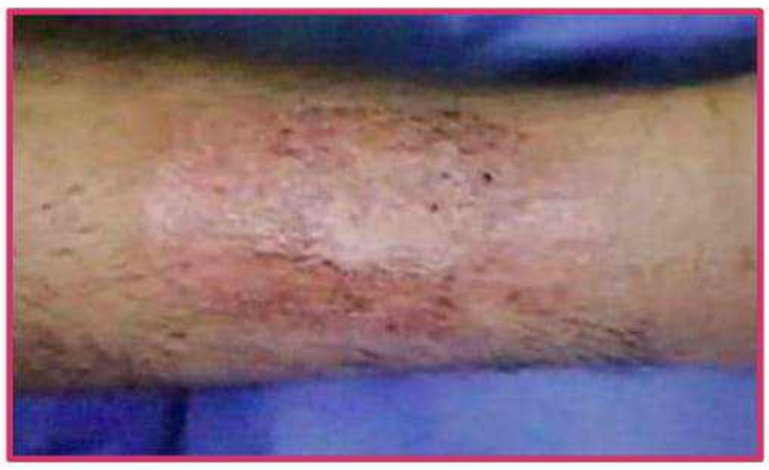
36:05

- Misnomer
- ulcerations of the skin associated with an underlying systemic disorder
- Diagnosis of exclusion
- No characteristics on HPE.
- Association :
  - i) IBD
  - ii) RA
  - iii) Malignancies (Hematological)
- Symptoms : Painful
- Pathergy Positive.
- Presentation : Small papule / pustule → Lower limbs
  ↓
  Breaks down to form an ulcer
  ↓
  covered by Granulation tissue
  ↓
  undermined violaceous margin.



active space

- Heals with atrophic cribriform scaring



- **PAPA:** Pyogenic Arthritis, Pyoderma gangrenosum and Acne.
- **PASH:** PG, Cystic Acne and Hidradenitis.

Treatment

- Steroids
- Dapsone
- Colchicine.

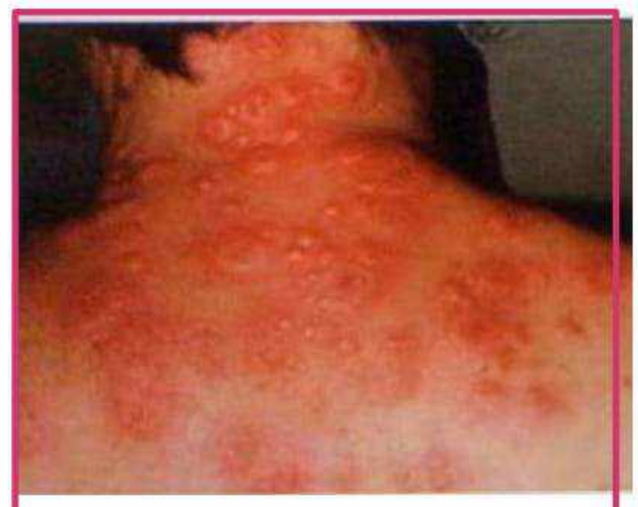
SWEET SYNDROME 41:40

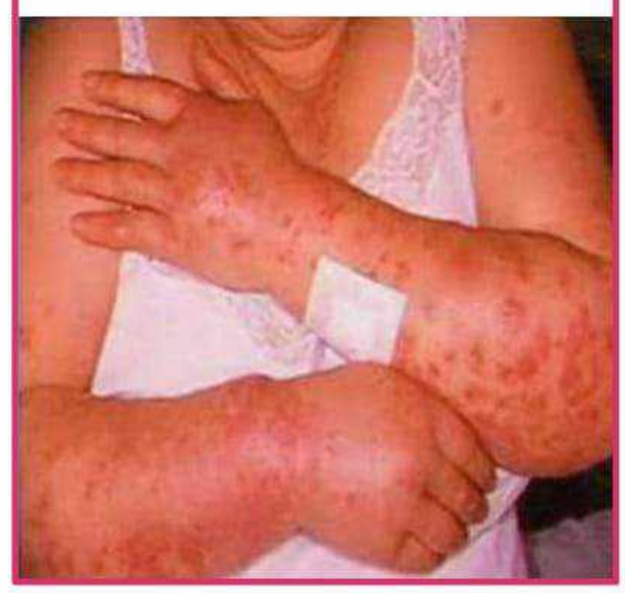
- Acute febrile neutrophilic dermatoses
- Females more commonly affected
- **On Histology:** Neutrophilic infiltrates.
- Types :
  - i) Classical
  - ii) Drugs
  - iii) Malignancies.

active space

Associations :

- Streptococcal Respiratory tract infections
- Gastro intestinal infections by Salmonella and Yersinia
- Mycobacterial infections
- IBD, RA, Sarcoidosis
- Hematological malignancies
- Drugs → GCSF





Clinical feature

- A/C Erythematous Rash
- H/O Fever + underlying condition
- In skin → A/c eruption of erythematous tender papules and plaques over upper part of body predominantly.
- pseudovesiculation → Edematous
- Associated findings
  - Joint pains
  - Eye involvement.

active space

Box 49.1 Diagnostic criteria for Sweet syndrome

Major

1 Acute onset of typical lesions
2 Histopathological findings consistent with Sweet syndrome

Minor

1 Fever >38°C
2 Association with malignancy, inflammatory disorder or pregnancy, or antecedent respiratory or gastrointestinal infection
3 Excellent response to systemic corticosteroids or potassium iodide (KI)
4 Abnormal laboratory values at presentation (three of four required: ESR >20 mm; leukocytes >8000; neutrophils>70%; elevated C-reactive protein)

Treatment

- Steroids
- Potassium iodide.
- Colchicine

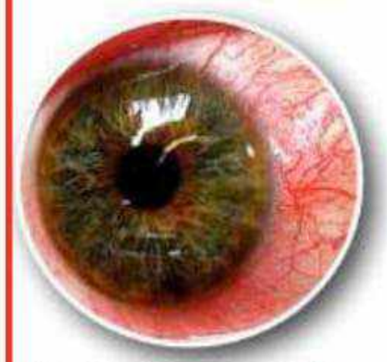
BEHCET'S DISEASE

47:10

- Multsystem inflammatory disease of unknown aetiology, classified as a systemic vasculitis and as a neutrophilic dermatosis involving all types and sizes of Blood vessels.
- On Histopathology: vasculitis and thrombosis

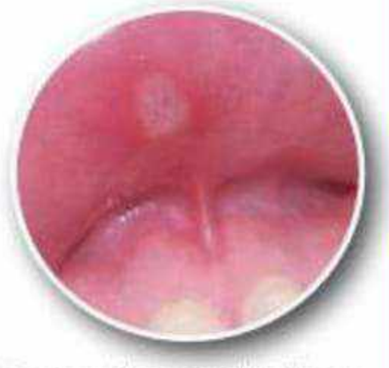
active space

Behçet syndrome

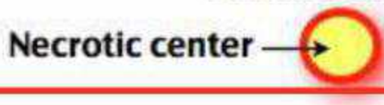


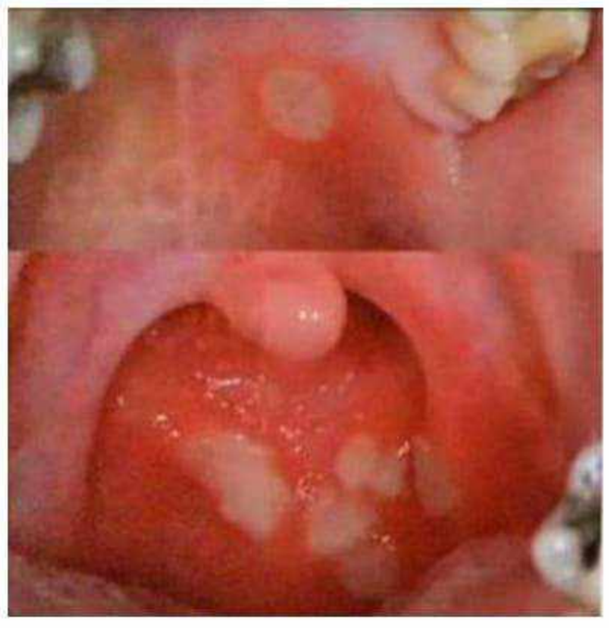
Relapsing uveitis
(Post > Ant).

Recurring genital ulcers

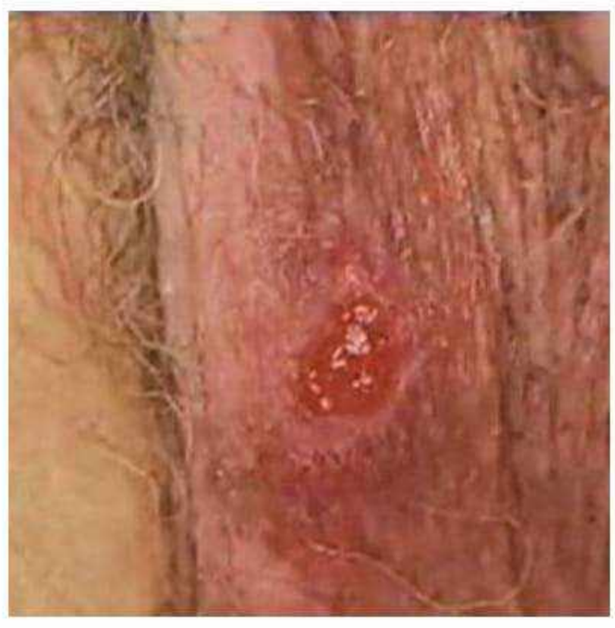


Recurring oral ulcers





Recurrent Oral Aphthae
↓
Heal without scarring



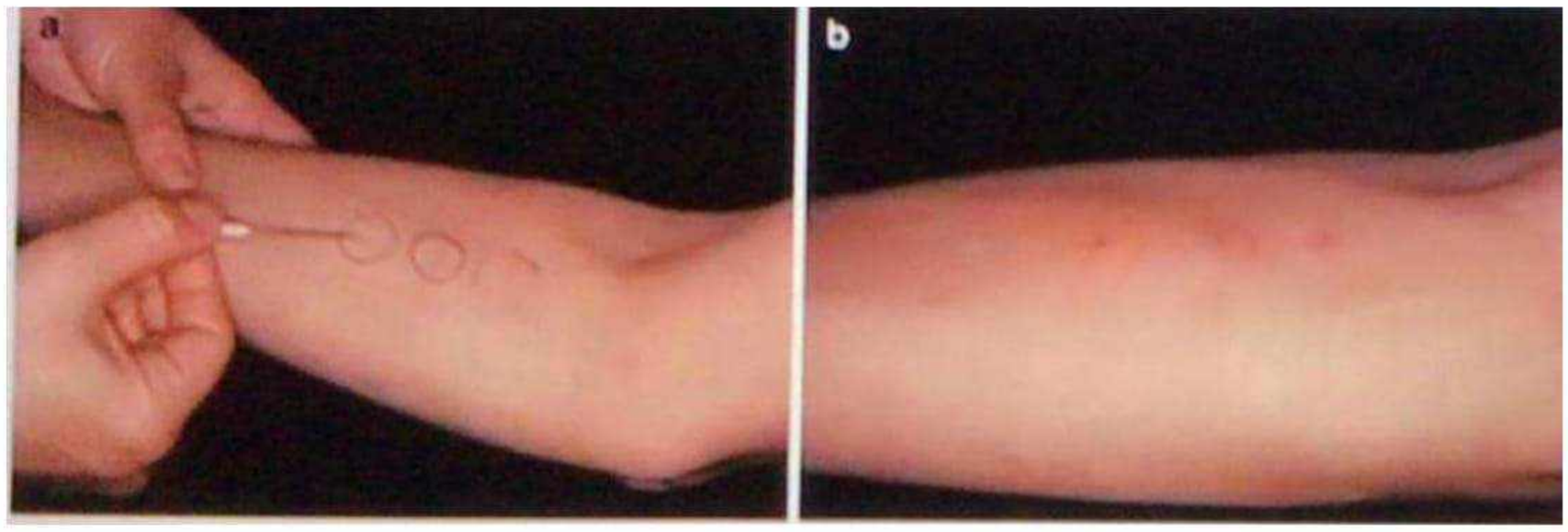
Recurrent Genital Ulcer
↓
Heal with scarring.

- **Other Skin lesions:** Erythema nodosum, Papulopustular lesions
- **Pathery positive**
- **Ocular involvement:** Posterior uveitis > Anterior uveitis
- **Joints →** Asymmetric Seronegative Oligoarthritis.
  ↳ Knee joint.

active space

- Multisystem involvement: Pulmonary, Cardiac, G.I, Neurological.

Pathergy Test:



- also positive in PG, RA, Crohn disease and genital herpes infection

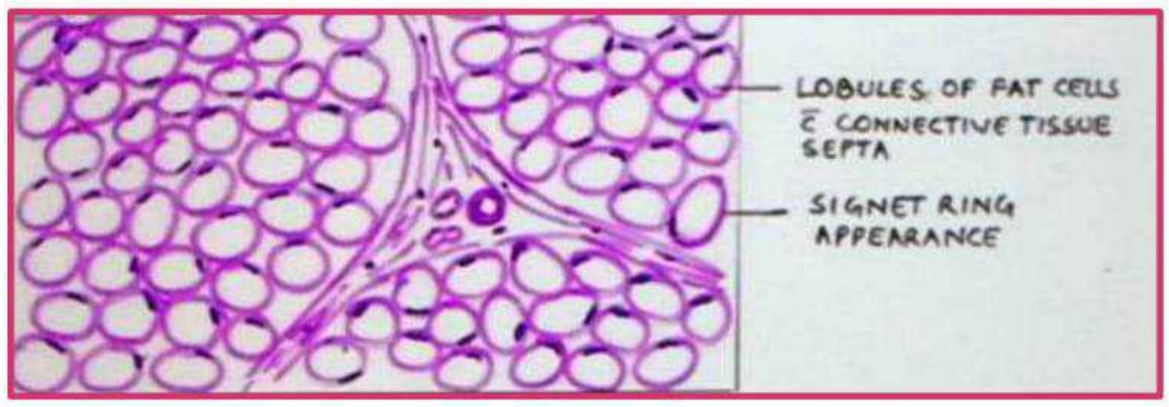
Treatment of Behcet's

- Oral Anesthetics
- Topical steroids
- Colchicine
- Dapsone.

PANNICULITIS

52:15

- Inflammation of panniculus (s/c fat)



- 2 Types :

  i) Lobular panniculitis

  ii) Septal panniculitis

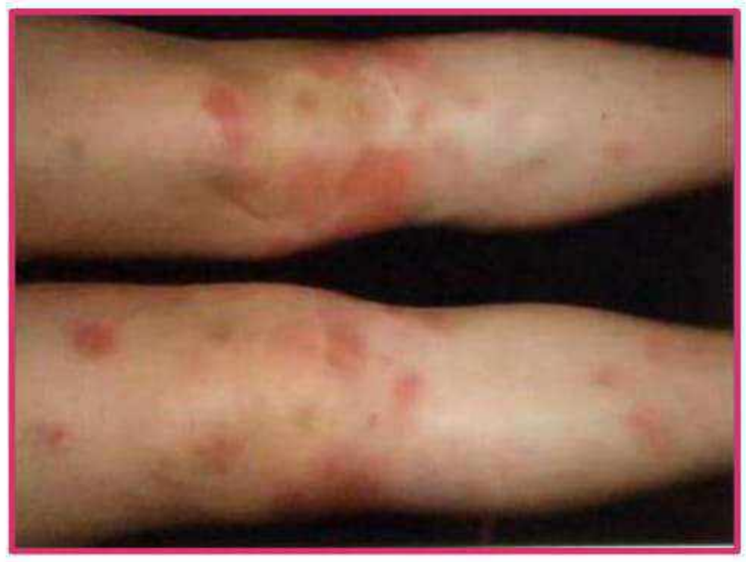
Erythema Nodosum

- (MC) panniculitis
- A/c onset and self limited course, Relapsing
- Cutaneous reactive process triggered by a wide variety of infectious and inflammatory disorders.
- (MC) Triggers :
  - Bacterial infections
  - Sarcoidosis
  - Inflammatory Bowel Disease
- URTI by Group A β Hemolytic Streptococcus : frequent cause in children and young adults
- Tuberculosis in areas of High endemicity.

Clinical features

- fever, joint pains, Malaise,

  Acute erythematous painful tender nodules



active space

predominantly on lower limbs → Shins, ankles and knee.

- They donot ulcerate
- They donot heal with atrophy or scar.
- Tendency to Recurrence → Spontaneous Resolution.
- Relapsing course
- On HPE : Septal panniculitis without vasculitis.

  "Meischer's granuloma" also seen.

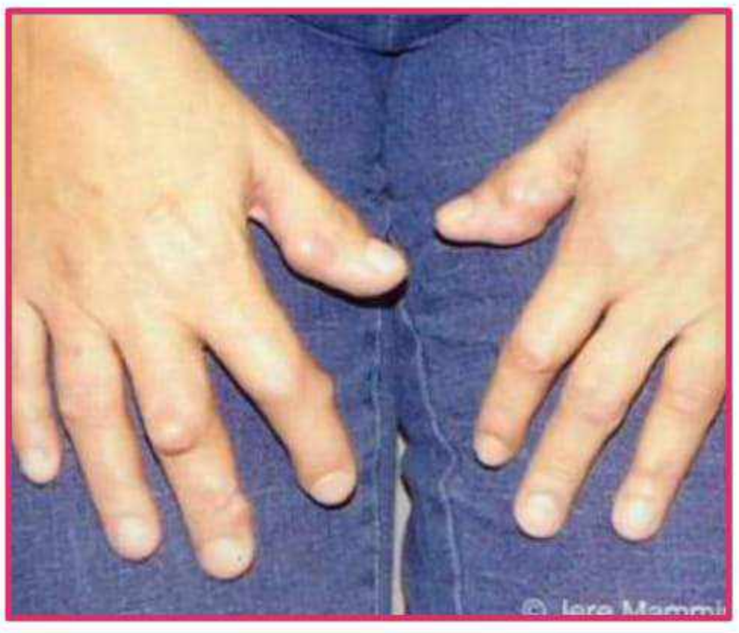
Treatment

- NSAIDS
- Leg elevation
- Acetaminophen, Aspirin
- In Resistant cases → Colchicine
- No steroids.

RHEUMATOID NODULE

59:30

- Extraarticular manifestations of RA
- Site : Skin of joints
- Accelerated Rheumatoid nodules



active space

- Accelerated Rheumatoid nodules
  ↳ Chronic RA → Give Methotrexate
  ↓
  Sudden eruption of Rheumatoid nodules.

REACTIVE ARTHRITIS

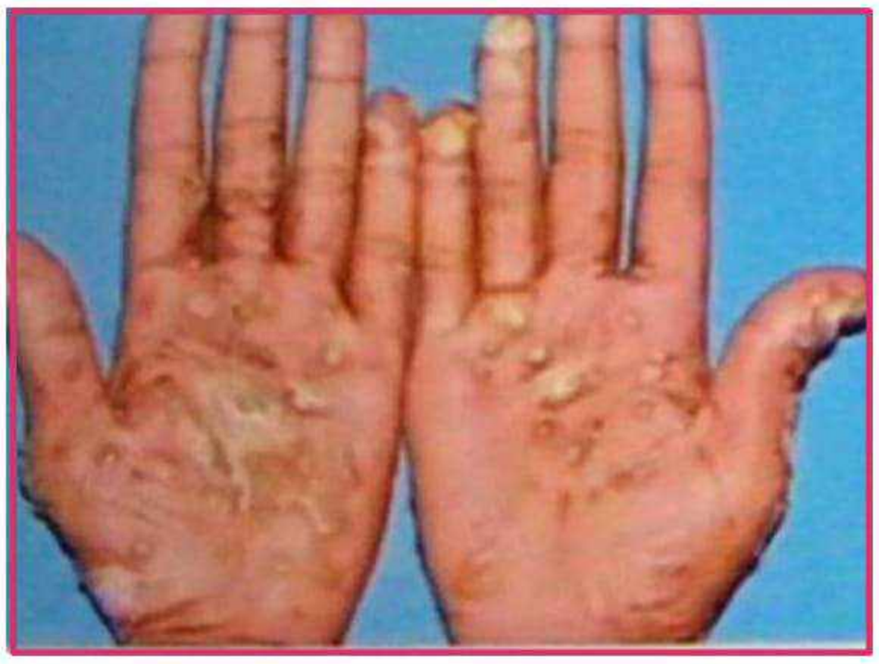
01:00:43

- a.k.a Reiter's disease
- HLA association → HLA B27, HLA B51
- Triggers:
  - GI Infection → Shigella, Salmonella, Yersinia, Campylobacter
  - STI (Lower Genital Tract Infection) → Chlamydia Trachomatis.
    ↓
    SARA
    (Sexually Acquired Reactive Arthritis)
  - Mycoplasma infection
  - HIV
- Triad (SARA) → i) Polyarthritis (>1 month duration following a lower genital tract infection)
  ii) Urethritis → Non-gonococcal conjunctivitis, Anterior uveitis.
  iii) Non-gonococcal conjunctivitis.

active space

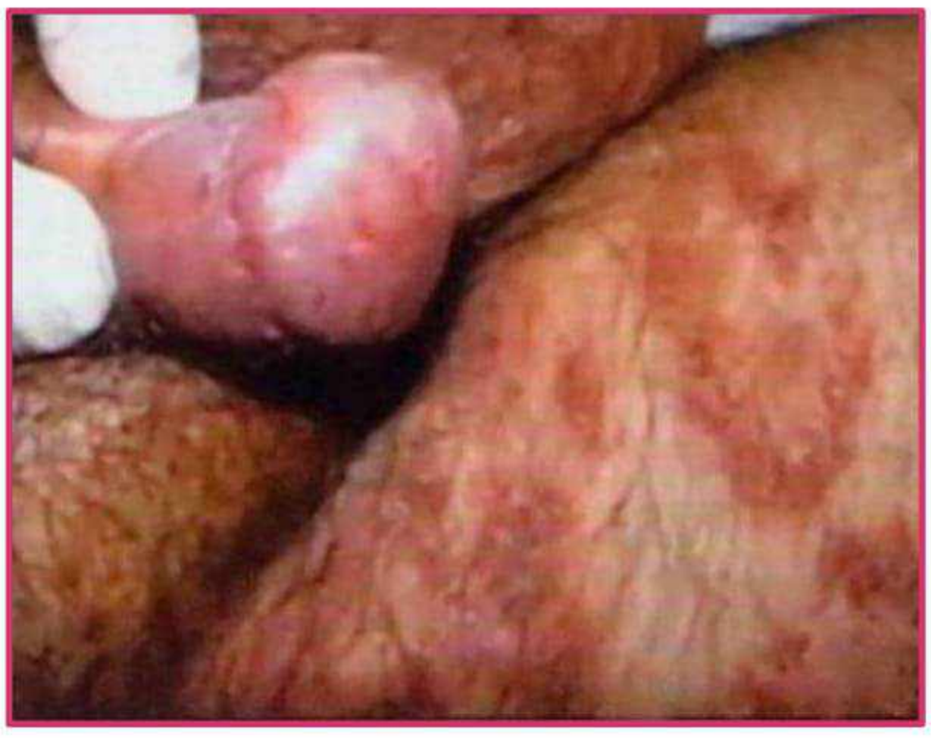
- **Polyarthritis** → Non-suppurative, joints of lower limbs, knee, ankle, Sacroiliac joint.
- **Skin manifestations:**

i) **Keratoderma blaenorrhagicum.**



- small vesicles surrounded by erythema on palms and soles.
  ↓
  Heaped up crusting
  ↓
  Keratoderma

ii) **Circinate balanitis :**



- Moist superficial erosions
  ↓
  coalesce to form circinate lesions

- **Renal involvement :** usually asymptomatic
  ↳ Proteinuria, Microscopic Hematuria and Aseptic pyuria.
- **Course** → **Chronic**

active space

Treatment :

- Topical Keratolytics
- Methotrexate
- In HIV ⊕ ⟶ Acitretin.