1 BASICS OF DERMATOLOGY

Skin and Its Appendages

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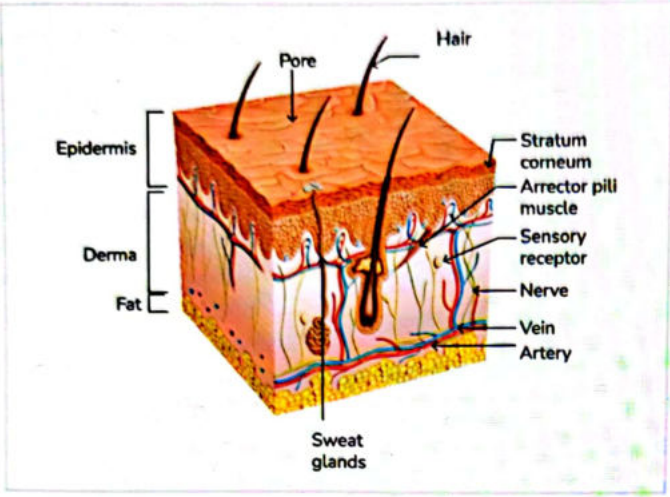
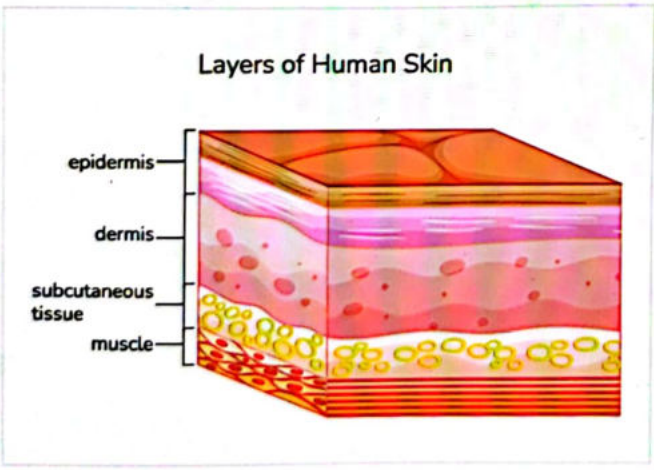


- Skin is the largest organ in the human body.
- Skin and its appendages are referred to as the Integumentary System.
- It weighs 4-5 kgs, and its BSA (body surface area) is 1.2-3 sq.m.
- The human body also has glabrous skin, which is non-hairy.
- It is present in Palms and Soles. Its appendages include:
  - Hair,
  - Nails,
  - Glands

Different Layers of Skin

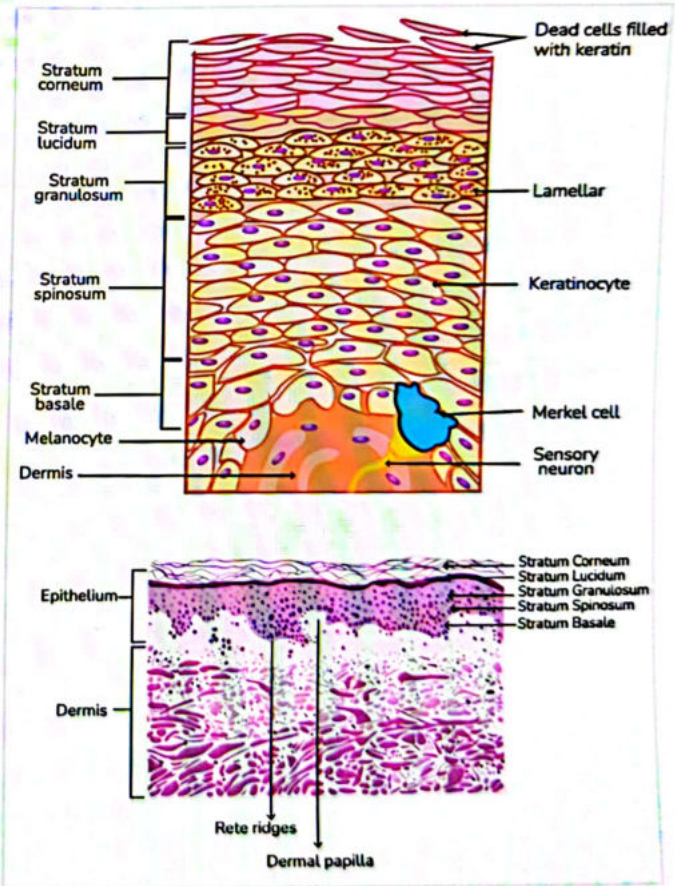


- Although the skin is visible as single-layered, it is made up of three layers, namely
  - Epidermis
  - Dermis
  - Subcutaneous tissue
  - Hypodermis
  - → Subcutaneous Fat
  - → Muscle

Epidermis

00:02:57

- The topmost layer of human skin is called Epidermis.
- It has a thickness of 0.5 - 1 mm.
- For example, the skin on our eyelids is extremely thin (0.5mm), and the skin on our hands and soles is quite thick (1 mm).
- Since Epidermis is the topmost layer, it acts as a major barrier-forming layer.
- Epidermis is formed of different layers that collectively build its complex structure. Each layer has its function and importance.
- Given below are the different layers of the Epidermis:



- Stratum corneum (come)
- Stratum lucidum (let's)
- Stratum granulosum (get)

- Stratum spinosum (sun)
- Stratum basale (burn)

Tip for learning:
Mnemonics - COME LET'S GET SUNBURN

Important Information

The layer Stratum Lucidum is only present in Palms and Soles. Since palms and soles are the thickest, they need an extra layer, Stratum Lucidum. This layer is not present in any other body parts.

- In the human body, skin is formed from bottom to top Basal layer to Corneum layer
- The skin undergoes a process known as cell Differentiation.
- Steps are as follows:
  - As the cell Differentiate, it losses nuclei
  - Next thing, the Cell becomes flattened
  - This results in loss of mitosis, and cells are dead
  - Then the surface area increases
  - Finally, cells become dehydrated.

Q. Describe Cell Differentiation

Ans:
- As the cell Differente, they lose nuclei
- Next thing, the Cell becomes flattened
- Loss of mitosis results in cell death.
- The surface area increases
- Cells become dehydrated.

Q. If a child is born preterm, which layer of skin is absent from the body?

Ans: Since the child is born preterm, his/her cell cannot differentiate; hence the Stratum Corneum will be absent.

Cell Kinetics
- Cell cycle of Keratinocytes - 300 hours

Epidermal Turnover time:
Epidermal Turnover time is the time cells travel from the base layer to the top for differentiation.
- Cells take 14 days to travel from Stratum Basale to Stratum Corneum.
- They stay at Stratum Corneum for another 14 days.
- After that, Cells exfoliate. It takes approximately 28 days.
- In total, it takes 56 days. But the range can vary from 52- 75 days.
- Skin remains in a continuous process of renewal and sheds old skin.

Stratum Corneum
- Corneocytes / Keratinocytes
- Dead Cells / Dead Keratinocytes
- No Nuclei/ No mitosis
- Its major importance is it acts as a barrier.
- Psoriasis - the Epidermal Turnover time in the disease is 4 days or 36 hours.

Pathological Findings:

Refer Table 1.1

Stratum Lucidum 00:19:17
- Found in Palms and Soles only, skin is thick on these parts.
- Also called clear cell layer
- Reason for being called a clear cell layer is that it has Refractile Granules of Elecidin.

Stratum Granulosum
According to the name, this layer is majorly made up of granules. The two most important granules of this layer are:

Types of Granules present in Stratum Granulosum

Keratohyalin Granules
- Responsible for forming Profilaggrin
- Profilaggrin forms Filaggrin
- Profilaggrin is Filament Aggregating Protein
- Filaggrin binds the keratocytes together to form a structure
- It has an important role in Barrier Functioning.

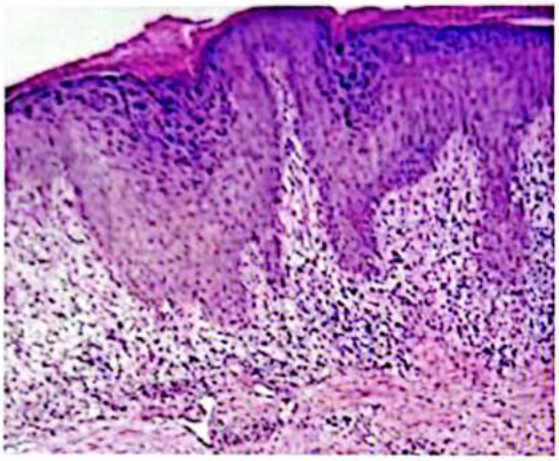
Lipid Coating Granules
- Responsible for providing moisture
- Odland bodies/ Lamellar Bodies
- Asteatotic dermatitis

Hypergranulosis
- Thickening of granular layer

E.g: Lichen planus

Agranulosis
- Absent granular layer

Eg.: Psoriasis

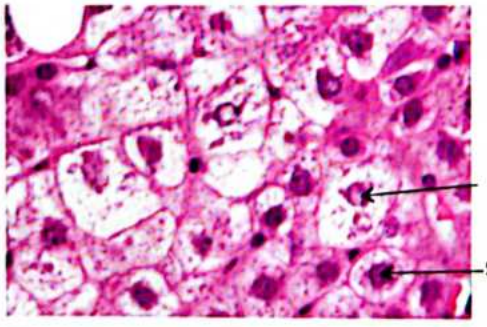


Stratum Spinosum
- Prickle cell layers- boldly visible
- Desmosomes
- Thickest layer of Epidermis
- Looks like spines assembled architecturally
- Keratonises

Pathological Findings:

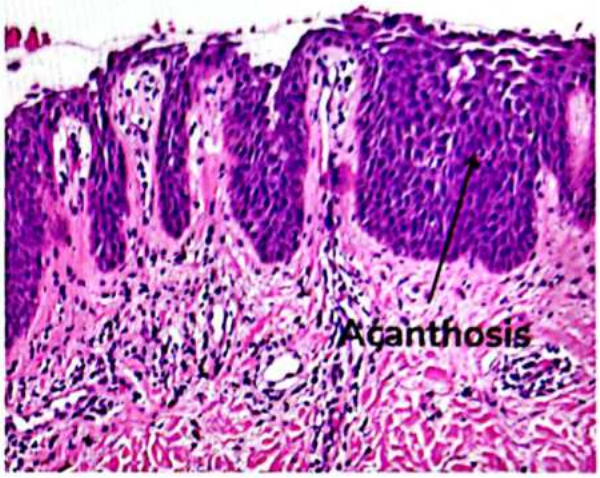
Cells have interlinking fluid
- **Spongiosis** - intercellular Edema
  - it occurs between the cell
- **Ballooning** - Intracellular Edema
  - occurs inside the cell

Seen in acute eczema



- **Acanthosis** - Occurs when Stratum Spinosum thickens. Keratocytes - Chronic Eczema

prince
ankitkarnawat9@gmail.com
9818635293

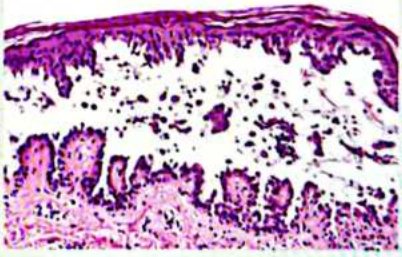


- Malphigian Layer - Mitotically Active layer of Epidermis
- Found in Stratum Basal and Stratum Spinosum

Stratum Basale
- Most Mitotically Active layer
- Provides one extra layer thickness
- Very important layer

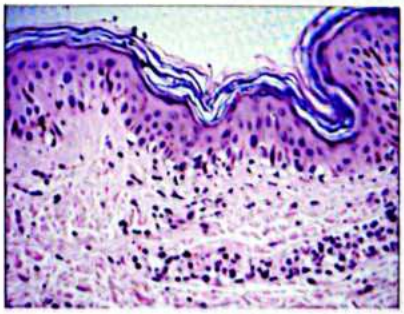
Pathological Findings:

Acantholysis



- Separation of keratinocytes
- Pemphigus group

Basale Degeneration



- Inflammation infiltrate
- Lichen planus

Types of Cells

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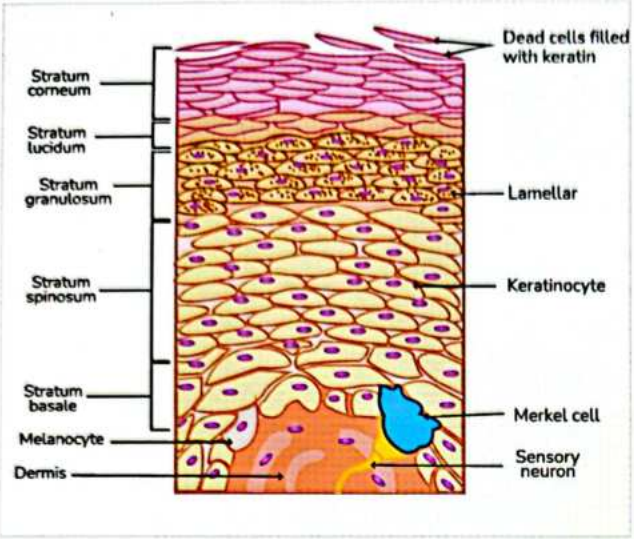
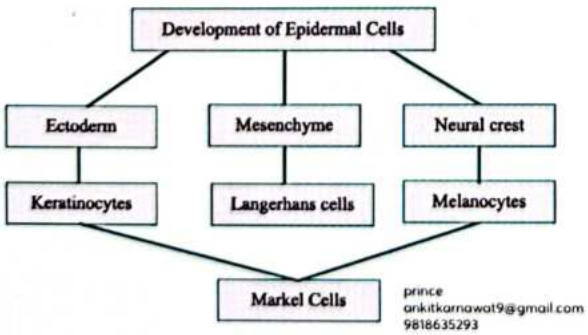


Layers of Epidermis are made up of cells, there are four types of cells:

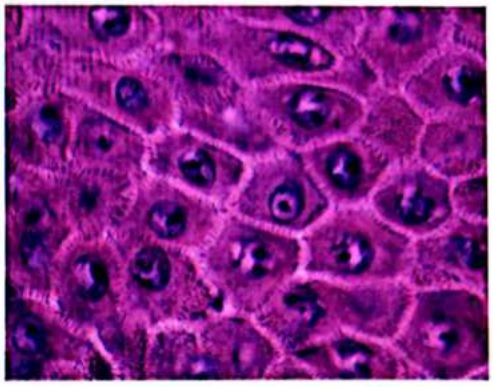
- **Keratinocytes - 95% of Epidermis**
- **Langerhan Cells - Stratum Spinosum**
  - Antigen Presenting cells
  - Birbeck Granules /Racquet Shaped
  - Chemicals - CDIA, CD207, Si00
- **Melanocytes - Stratum Basale**
  - Pigment forming cell
  - Dendritic cell
  - Ratio is 1 melanocyte for 10 Keratinocytes
- One melanocyte will transfer melanosomes to 36 Keratinocytes known as Epidermal Melanin Unit
- Responsible for forming skin color
- **Merkel Cells - Stratum Basale**
  - Ectoderm Neural Crest
  - Touch receptors
    - → Slow adapting touch receptors
    - → Fast adapting receptors responsible for hair cells pacinian corpuscle

Development of epidermal cells

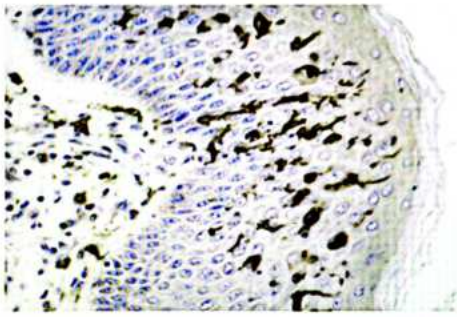


1. Keratinocytes

90-95% Epidermis

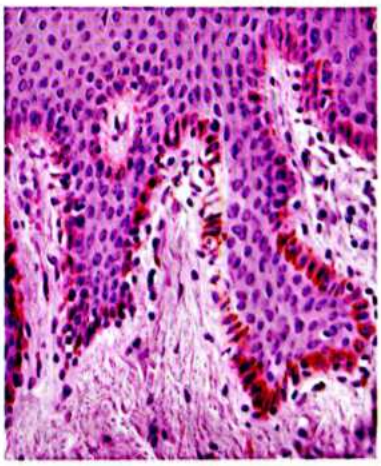


2. Langerhan cell



- Stratum Spinosum mesenchyeme
- Present in SS
- APC
- Birbeck granules / magnet shaped
- CD1A, CD207, S100
- Antigen presenting cells

3. Melanocytes



- Stratum Basale
- Pigment forming
- Dendritic cells
- 1:10 (10 keratinocyte to 1 melanocyte)
- EMU
  - One melanocytes transfer melanosomes to 36 keratinocytes
  - EMU → epidermal melanin unit (1:36)
  - EMU → responsible for uniform skin colour.

4. Melansomes

- Light skin →distributed as membrane- bound claster
- Dark skin → Be large and distributed individually

5. Merkel cell

- Ectoderm Neural Crest
- Touch receptors
  - Slow adapting touch receptors
  - Fast adapting receptors responsible for hair cells : pacinian corpuscle

Development of Epidermal Cells

- SB
- Ectoderm > Neural crest
- Touch receptors: Slow adapting Pacinian corpuscle

Touch Receptors (MRF)
- Fast adapting
- Slow adapting
  - Merkel cells
  - Ruffinis ending
  - Free nerve ending

Nerve and innervation

Mechanoreceptors	Merkel cells in epidermal
Nociceptors	Meissner corpuscles in dermis

Nerves & Innervation

- Rich network of nerves with 2 types of sensory endings – 'The Corpuscles (Mechano-receptors)' and 'Free nerve endings (Nociceptors)'

Mechano-receptors

- Light touch: Merkel cells of the epidermis, Meissner's corpuscles in dermal papilla
- Pressure: Pacinian corpuscles in deep dermis or subcutaneous tissue

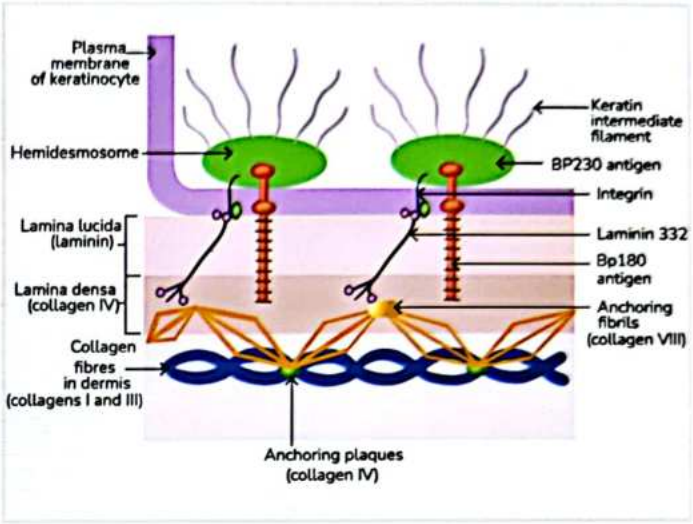
Nociceptors

- Pain and Itch: Transmitted through naked fine free nerve endings located in the basal layer of the epidermis close to the dermo-epidermal junction

Temperature

- Krause bulbs detect cold, Ruffini end organs detect heat
- Heat, cold and proprioception also located in the superficial dermis

Dermo epidermal junction



- A part of epidermis that invaginates into the dermis is called Rete Ridges.
- And the part of the Dermis that invaginates into the epidermis is called Dermal Papilla.
- The junction between Epidermis and Dermis is called Dermo Epidermal Junction.
- It is not just a line or connection; it's a complex structure.
- It is also known as the Basement Membrane Zone.
- Predominantly formed by type IV collagen
- Main function of BMZ is to provide adhesion between the two layers and signaling.

Dermis

- Dermis is the layer next to the epidermis. It contains all the Nerve Endings, Blood Cells, and other important skin components.

Dermis has two parts

- Papillary Dermis - the part that is invaginating into the epidermis
- Reticular Dermis - the part that contains all the fibers.

Why is Dermis Important?

Dermis is important because it has lots of structures that comprises the major part of the skin. The major components of the Dermis are:

- Cells
- Fibre
  - Collagen (Predominant)
  - Elastin

- Ground Substance
  - Hyaluronic Acid
  - Heparan Sulphate
- Nerves
- Vessels
- Hair Follicles
- Lymphatics.

- In Human skin, Collagen is considered a Predominant Fiber.

Furthermore, the cells that are present in Dermis are:

- Fibroblast
- Langerhan Cell
- Mast cell
- Lymphocytes
- Phagocyte

Subcutaneous Fat

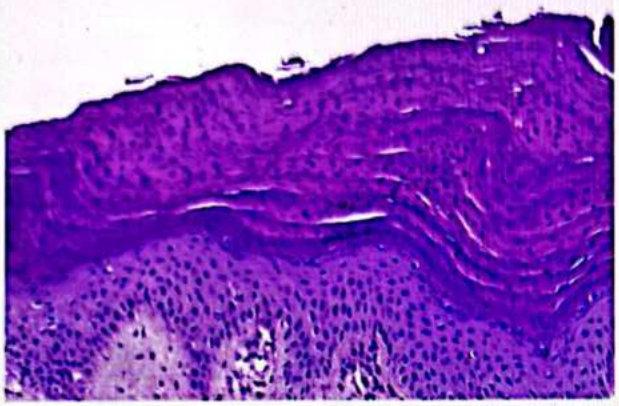
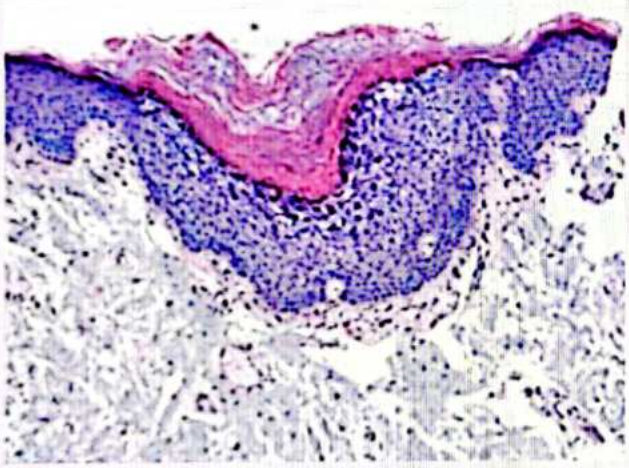
- Subcutaneous fat is all blood and lymphatics. It provides cushioning where the skin is thick. Therefore, it is absent where the skin is very thin. For example, Eyelids and Genitalia.

Functions of Skin

Most important function of the skin is the formation of Vitamin D. It is formed in Stratum Basal or Stratum Spinosum. Other functions are:

- Temperature Control
- Water Control
- Cushioning

Table 1.1

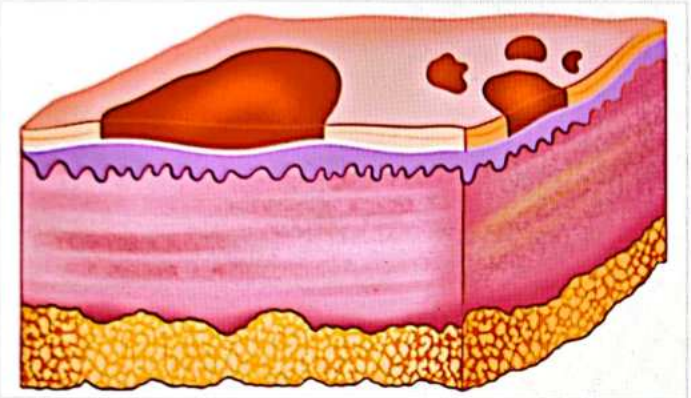
Histopathology Findings	Reasons	Physiological findings	Pathological Findings
Parakeratosis 	Retention of Nuclei In Stratum Corneum	Mouth and Vagina	Psoriasis, Eczema, Squamous Cell Carcinoma, Actinic Keratosis, Seb. Dermatitis
HyperKeratosis 	Thickness of Stratum Corneum	NA	Lichen Planus, Psoriasis

2 SKIN LESIONS IN DERMATOLOGY

- **Types Of Skin Lesion**
  - **Primary** - They appear first in the disease.
  - **Secondary** - Changes that develop over the primary lesions
  - **Special** - Characteristics of a particular disease. (Specific to certain dermatological disorders)

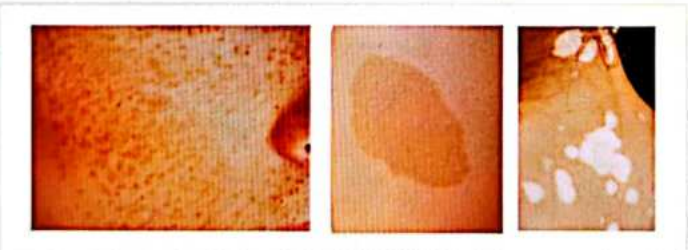
Primary lesion 00:01:50

Macule And Patch



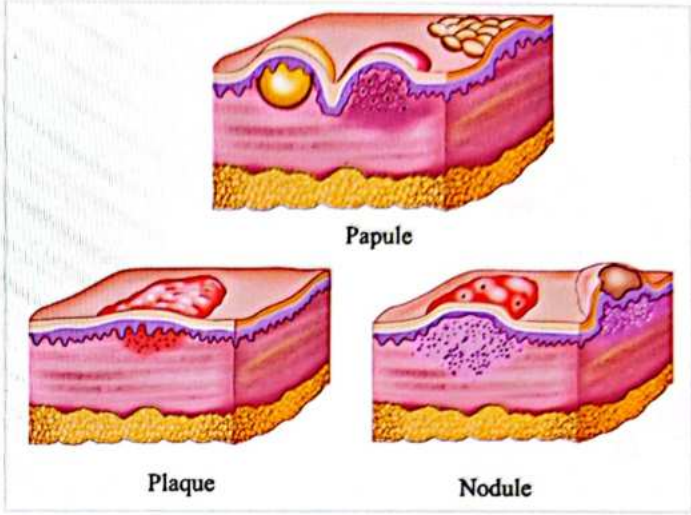
Macule and Patch

- There is a change in skin color.
- Cannot be felt.
- Better seen than felt.
- Neither raised nor depressed
  - If lesion < 0.5 cm = Macule
  - If lesion > 0.5 cm = Patch
- Change in skin color can be hyperpigmented (more color) or depigmented (absence of color)
- Sometimes lesions can be hypopigmented (decreased color)

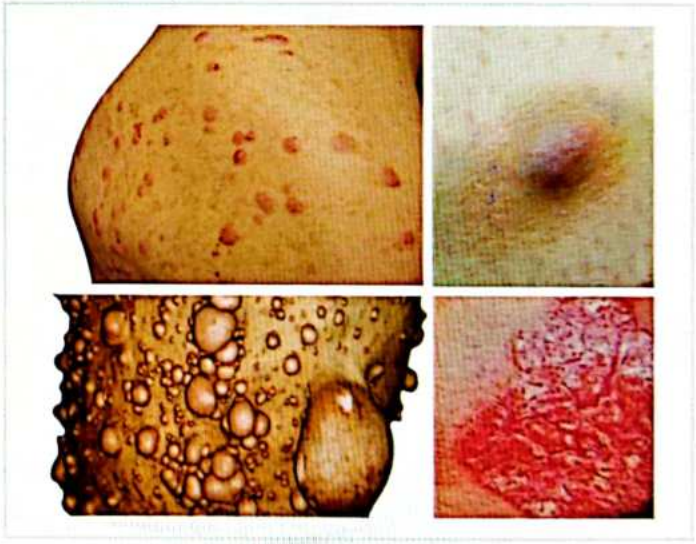


- There is no change in textures

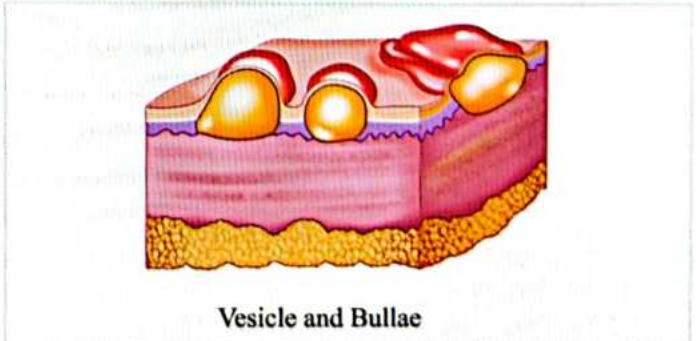
Papule

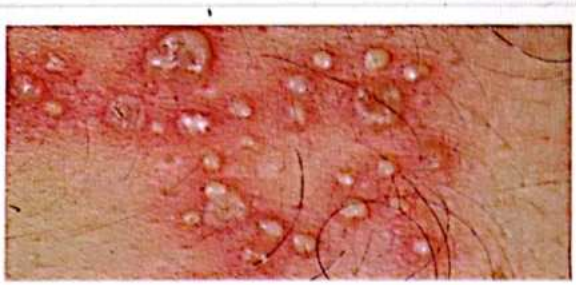


- It is a circumscribed solid raised lesion.
- Three types:
  - If they are < 0.5 cm = papule
  - If they are > 0.5 cm = plaque (there is a change in texture)
  - If they are > 0.5 cm & more depth = Nodule (better felt than seen)



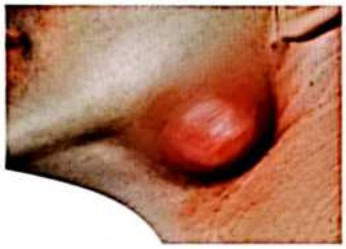
Vesicle and Bullae 00:06:30

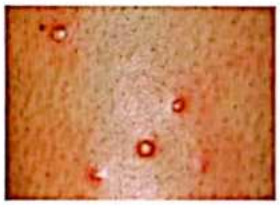


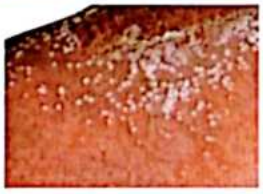


- These are fluid-filled lesions.
  - If lesion is < 0.5 cm = Vesicle
  - If lesion is > 0.5 cm = Bullae

Pustule







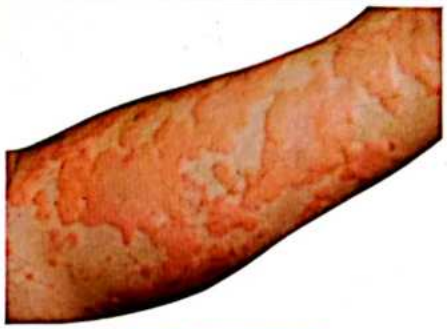
pustule can be primary or secondary

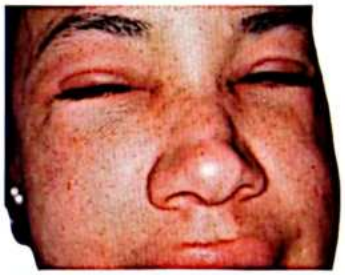
- They are pus-filled lesions.
- The collection of pus in a cavity is called Abscess.
- They can be primary or secondary lesions.
- For example, Pus arising from vesicles is a secondary pustule, as it develops over primary lesions.

Extravasation of RBCs in skin

- Due to any reason or clotting disorder, RBCs settle down on the skin.
- These are non-Blanchable.
  - If these lesions are 1-2 mm = Petechiae.
  - If > 3 mm = Purpura
  - If it is 1-2 cm = Ecchymosis

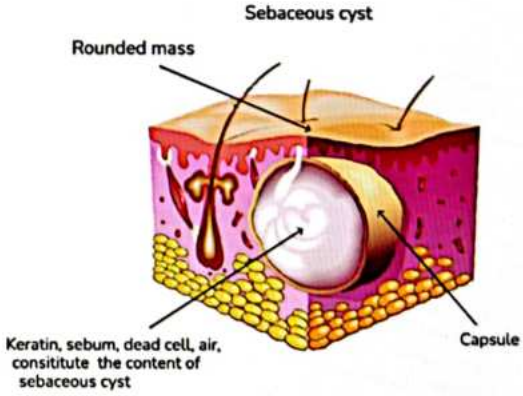
Urticaria and Angioedema

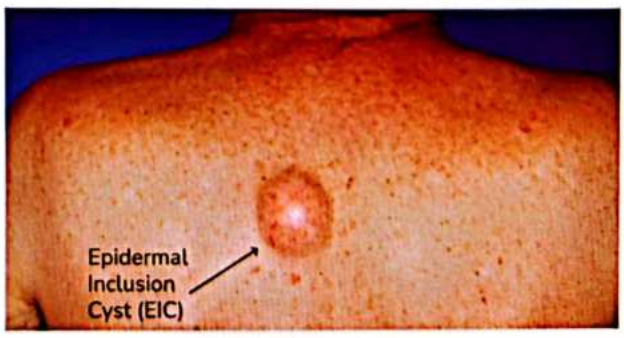




- Urticaria - referred to as wheals
- Wheal is erythematous, edematous, & evanescent.

Cyst

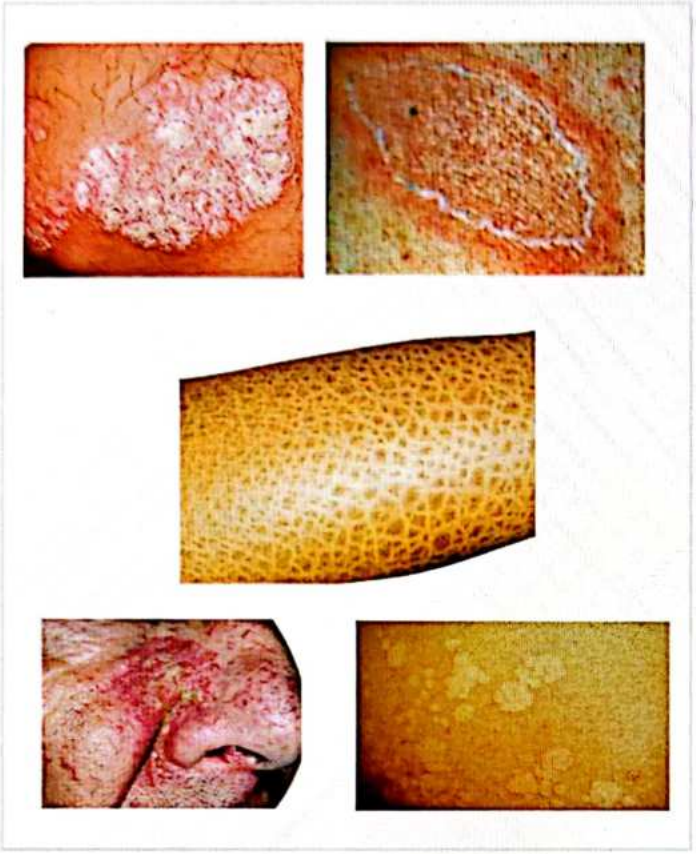




- It is an enclosed cavity with a lining filled with fluid or semisolid material.
- Example - Epidermal Inclusion Cyst (EIC)

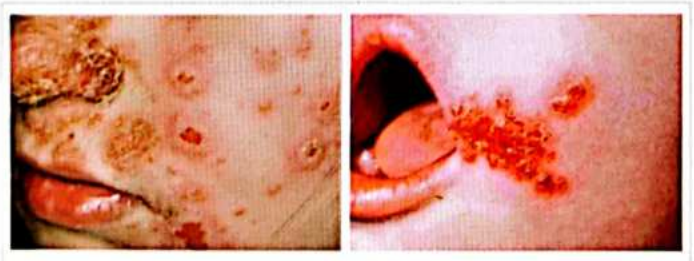
Secondary Skin Lesions 00:12:06

Scale



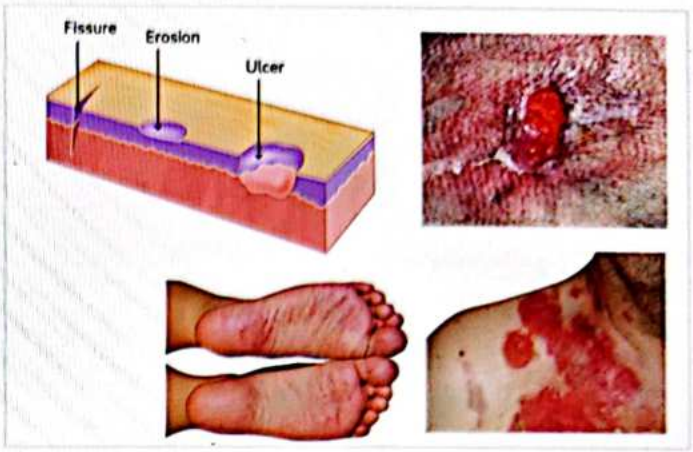
- These are visible exfoliation of the skin.
- They involve the stratum corneum skin layer.
- Examples:
  - Silvery white scale
  - Fish-like scale
  - Greasy scales

Crust



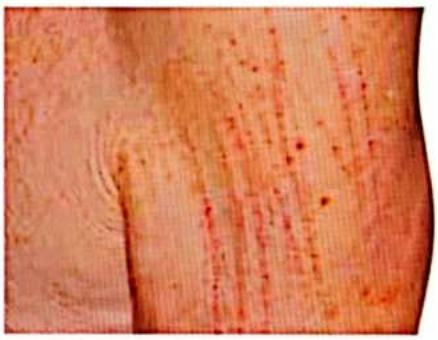
- Dried-up exudate is called Crust
- Exudate can be pus, serum & blood.
- Honey-coloured crust is seen in Non-bullous impetigo.

Erosion, Ulcers, and Fissures



- Erosions are raw, moist areas formed by the denudation part of the epidermis.
- Erosions are superficial and have no base.
- An ulcer is also denuded, but along with the epidermis, part of the dermis is also involved.
- They can even go up to subcutaneous tissue.
- Ulcers have a base and margin.
- Fissure is a linear crack in the skin.

Excoriations 00:17:52

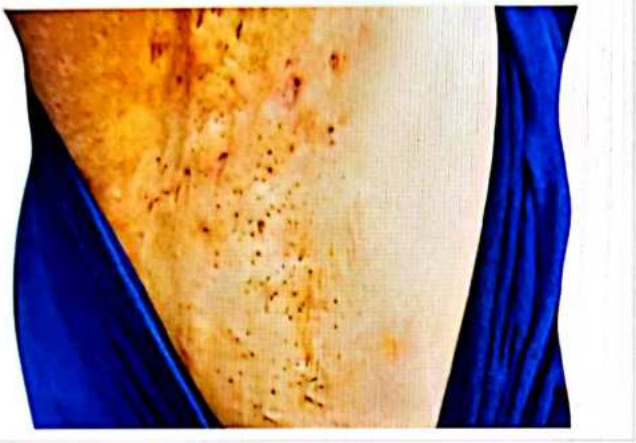


- They are surface excavations on the skin.
- Mainly caused by itching.

Lichenification

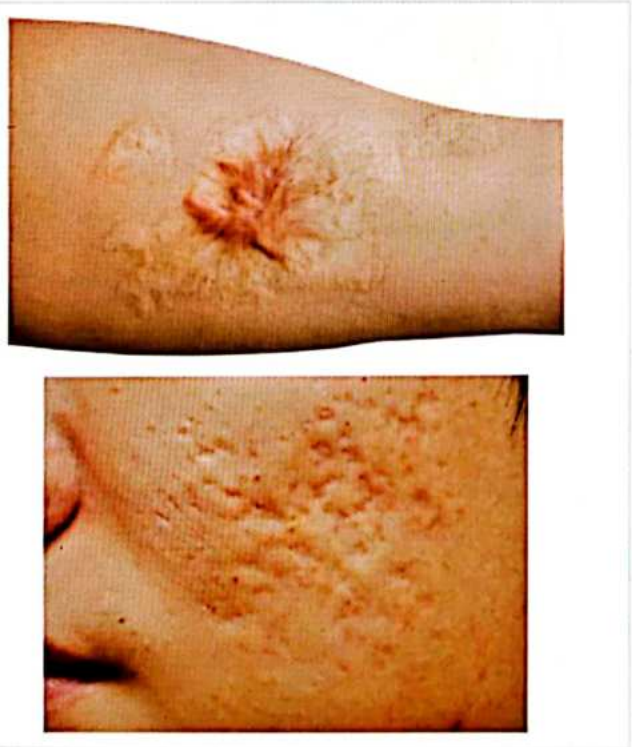
- They happen due to chronic itching.
- Acanthosis will be seen here.
- They involve certain features, such as:
  - Hyperpigmentation
  - Increase in skin markings
  - Thickening of skin

Sinus



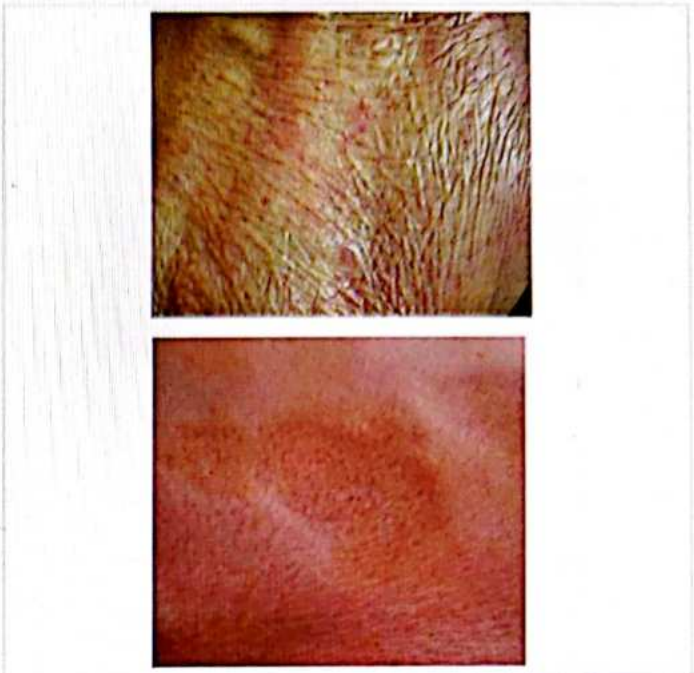
- They are blind tract that connects skin to a deeper cavity.

Scars



- Abnormal proliferation of fibrous tissue replaces normal collagen in the skin.
- Two types of scarring:
  - Hypertrophic: When scarring is high
  - Atrophic: When scarring is depressed.

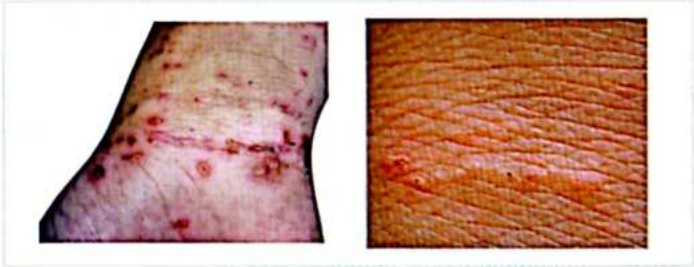
Atrophy



- It can be epidermal Atrophy.
  - In Epidermal Atrophy, one gets wrinkled skin.
- It can also be Dermis Atrophy.
  - In Dermis Atrophy, overall skin is normal, but there is depression.

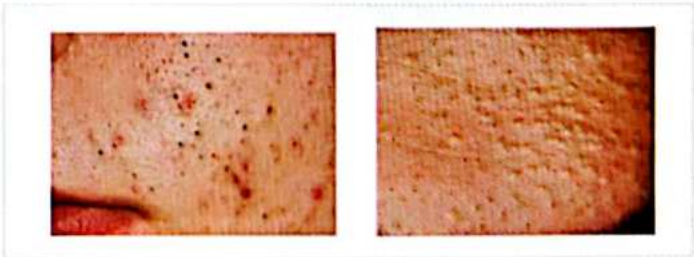
Special Lesions 00:21:07

Burrow



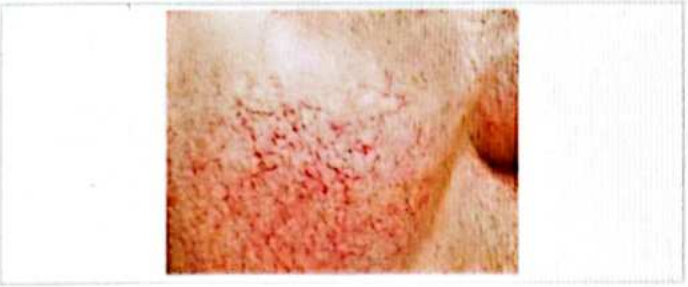
- They are mostly seen in Scabies.

Comedones

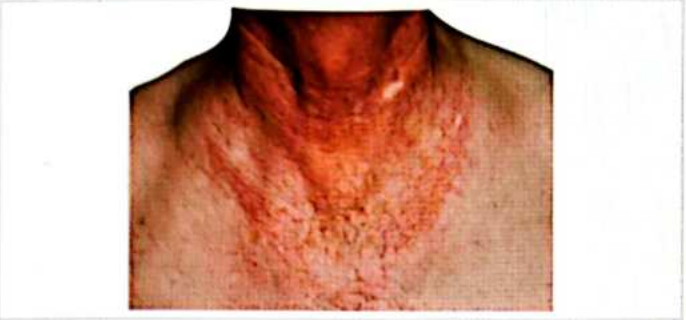


- They are blocked dilated Pilosebaceous glands.
- They can be seen as Acne.
- Comedones can be opened or closed.
  - Open Comedones are white.
  - Closed Comedones are black.

Telangiectasia

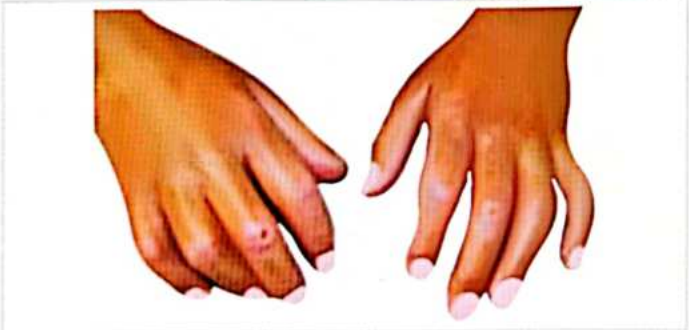


- Telangiectasia: visible dilatation of dermal capillaries



- Poikiloderma: telangiectasia, atrophy, skin pigmentation
- They are visible dilatation of dermal capillaries.
  - They appear in a condition named Poikiloderma.
- Poikiloderma has three components:
  - Telangiectasia
  - Atrophy
  - Skin pigmentation.

Sclerosis

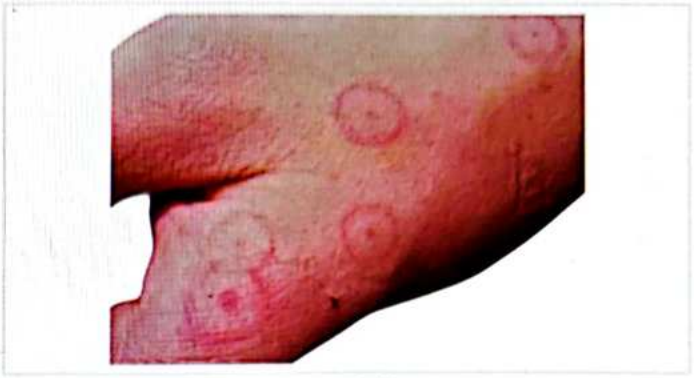


- When the skin is bound down.
- They are seen in Systemic Scleroderma.

Milia

- They are keratin-filled cysts.
- They appear as white lesions.
- They are asymptomatic.

Target lesion



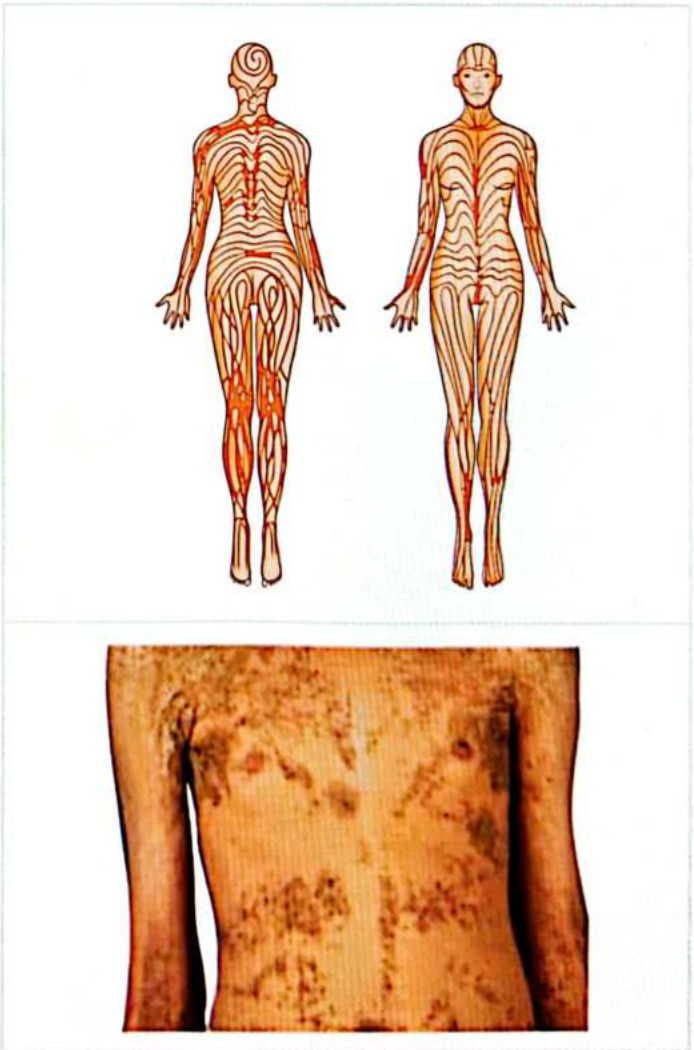
- They are seen in Erythema multiforme.

Lines In Dermatology

00:24:07

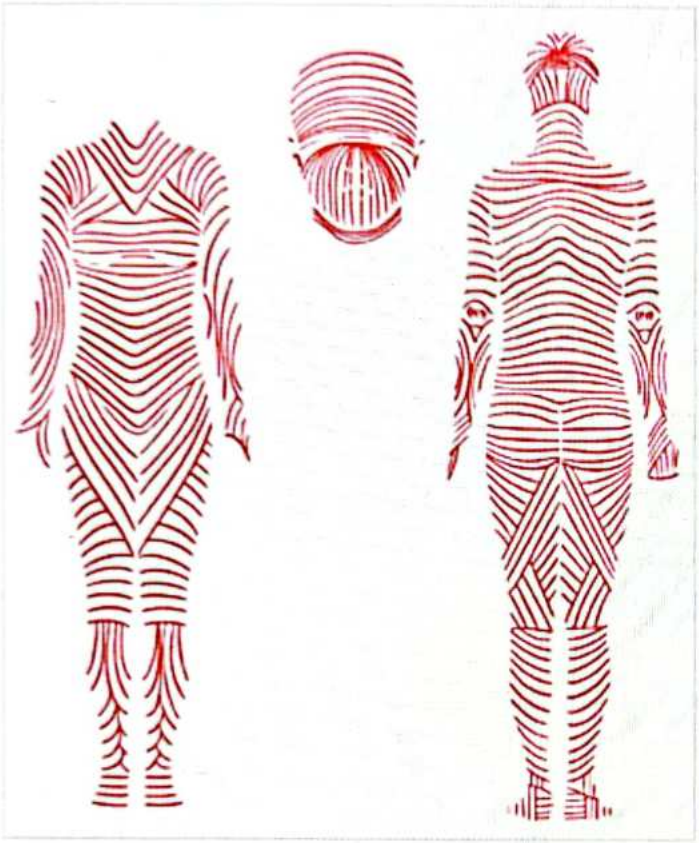
- There are two lines in dermatology. They are
  - Blaschko's lines
  - Langer's lines

Blaschko's Lines



- They are the lines of embryonic development.
- They are the lines along which keratinocytes migrate.
- These lines are constant.
- They have a strict midline demarcation.
- Spiral pattern of lines in the posterior part and the linear pattern on the limbs can be seen.
- They are important because of a certain dermatosis, which presents along the Blaschko lines.
- Some Examples - Verrucous epidermal nevus (VEN), Incontinentia pigmentation.

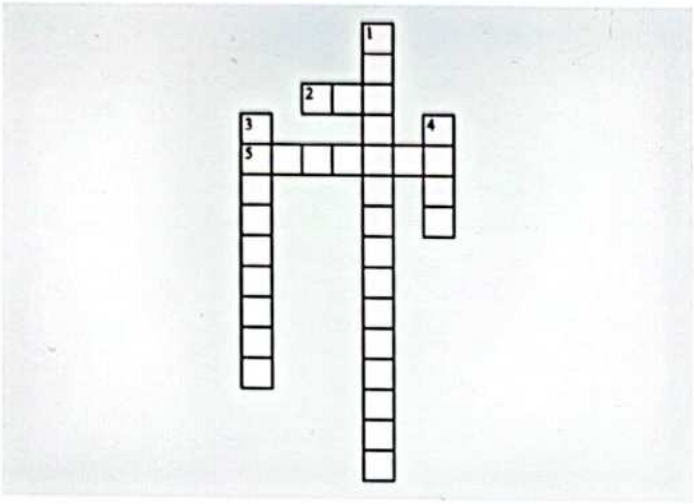
Langer's Lines



- They are the lines of the orientation of collagen and muscle fibers in our body.
- They are important to know at the time of making surgical incisions.
- Knowing about langer's lines helps dermatologists to determine how the incisions should be done in the skin.
- If done correctly, the healing would be better as there will not be much damage to collagen and muscle fibers.
- These lines are called the Relaxed Skin Tension Lines (RSTL).
- Since these lines represent collagen and muscle fibers, they are not constant.
- They do not have strict midline demarcation.

CROSS WORD PUZZLES

Crossword Puzzle



Across

2. Epidermal Inclusion Cyst
5. Lines collagen and muscle fibers

Down

1. chronic itching
3. Lines embryonic development
4. Relaxed Skin Tension Lines

3 BACTERIAL INFECTIONS OF SKIN

Flora

00:00:19

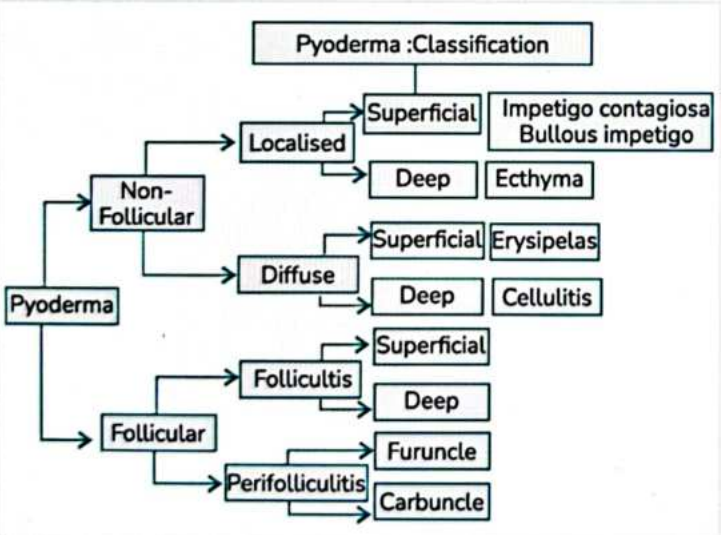
Two types

- Resident flora
  - It is always present
  - Acts as the first line of defense
  - Forms a protective barrier.
- Transient flora
  - It comes and goes
  - Pathogenic flora: causes diseases.
  - Example
    → Staphylococcus aureus
    → β hemolytic streptococci
- Normal resident flora in more quantity can also sometimes lead to disorders.
- Important resident flora which can cause problems are
  - Propionibacterium acne
  - Corynebacterium
  - Malassezia in the fungus anomalous flora is a resident flora which can sometimes become pathological
  - Staph epidermidis

Classification of bacterial infection

00:01:47

- Bacterial infections are also called pyoderma
- Types

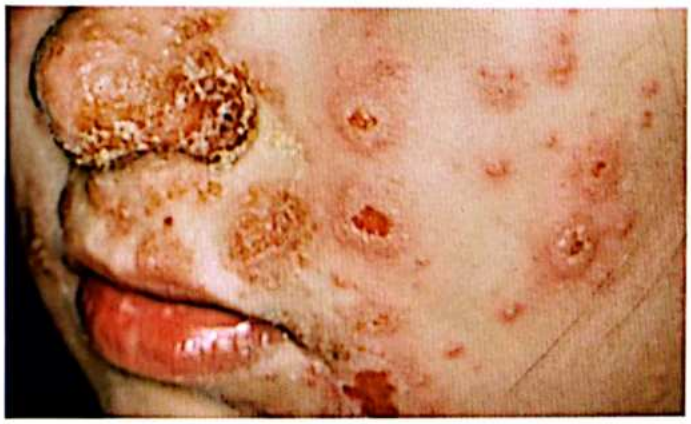


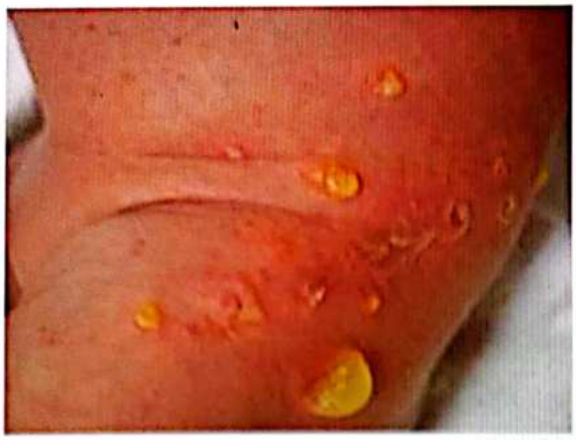
  - Non follicular
    → Not happening around the follicles
    → Can be caused by both strep and staph
  - Follicular
    → Always caused by Staph or Staphylococcus bacteria aureus
    → They can be folliculitis, furuncle, carbuncle

Impetigo

- Most seen in children on the face.
- Impetigo is a superficial infection
- Two types
  - Non bullous impetigo - more common also known as impetigo contagiosum
  - Bullous impetigo

	Bullous impetigo	Non bullous impetigo /IC
Age group	Usually seen in newborn	Usually seen in preschoolers/toddlers
Organism	Staph aureus	Both by streptococcus and staphylococcus • staphylococcus is more commonly seen in developed nations. • streptococcus is more commonly seen in developing nation If both are in option one should mark strep
C.F/type of crust	Varnish crust Superficial bulla	Crusted erosions covered by honey colored crust.
Complication,	These patients may land up into Staphylococcal scalded skin syndrome (SSSS).	The common complication is Poststreptococcal glomerulonephritis (PSGN)



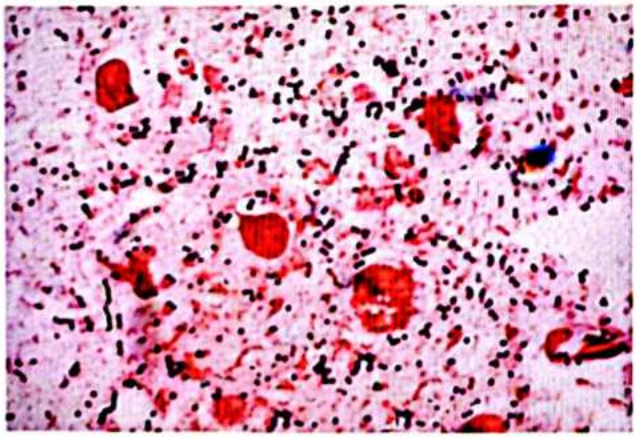


- Non-Bullous impetigo is also called Impetigo contagiosa because it spreads.
- Staph aureus produces exfoliative toxin, binds to Desmoglein-1 (DSG1) and causes Pemphigus foliaceus.
  - It causes separation of keratinocytes leading to formation of bulla

Q. In bullous impetigo, what is the target?
Ans. it is desmoglein-1

Q. What is the toxin?
Ans. It is the exfoliative toxin or ET-1.

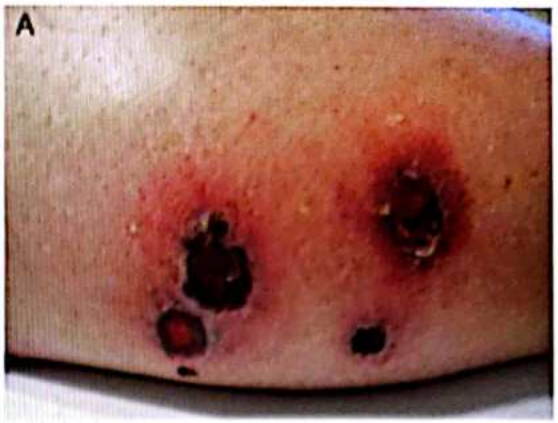
- Here, we will see superficial bulla, which will rupture to give rise to crust which are usually called as varnish like crust.
- Gram stain
  - Gram positive cocci present in cluster
  - They call it grape like cluster

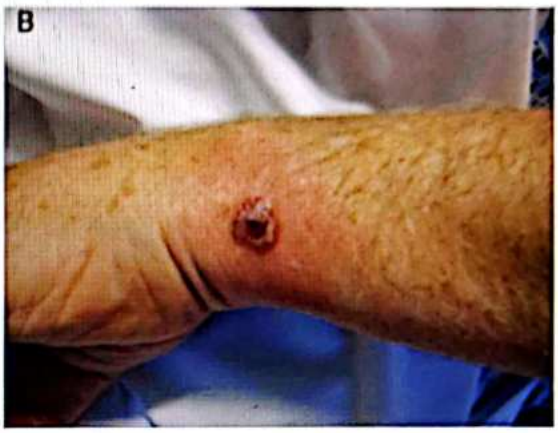


Ecthyma

00:08:19

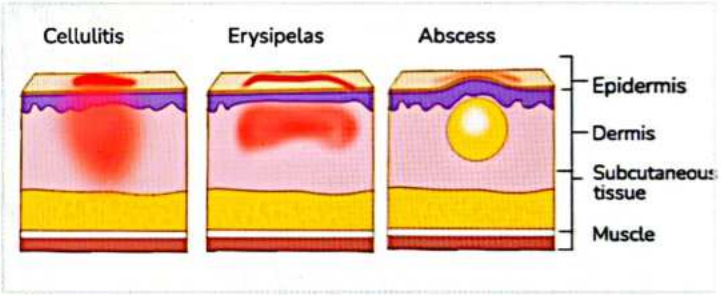
- It is a deep infection, but it is localized and is mostly caused by streptococcus and can also be caused by staphylococcus.
- Seen on buttocks, thighs, and legs
- In ecthyma, the ulcers are covered by thick, necrotic crust and painful.



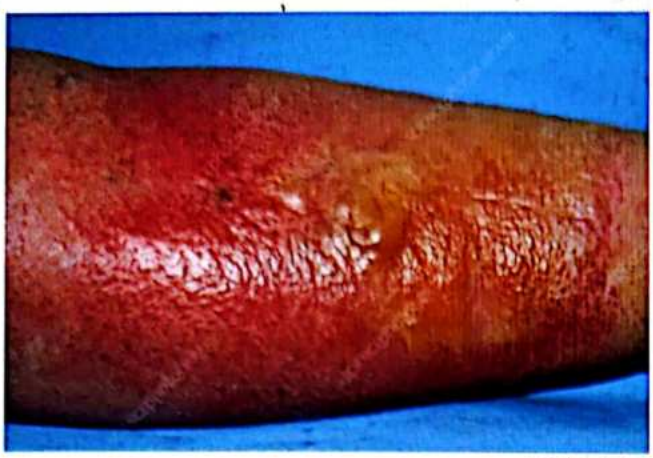


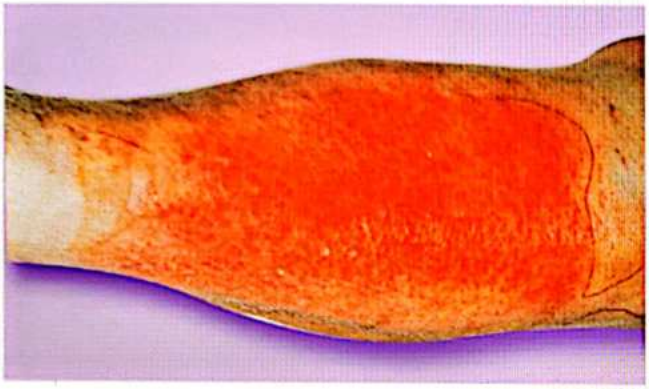
Erysipelas and cellulitis

00:09:07



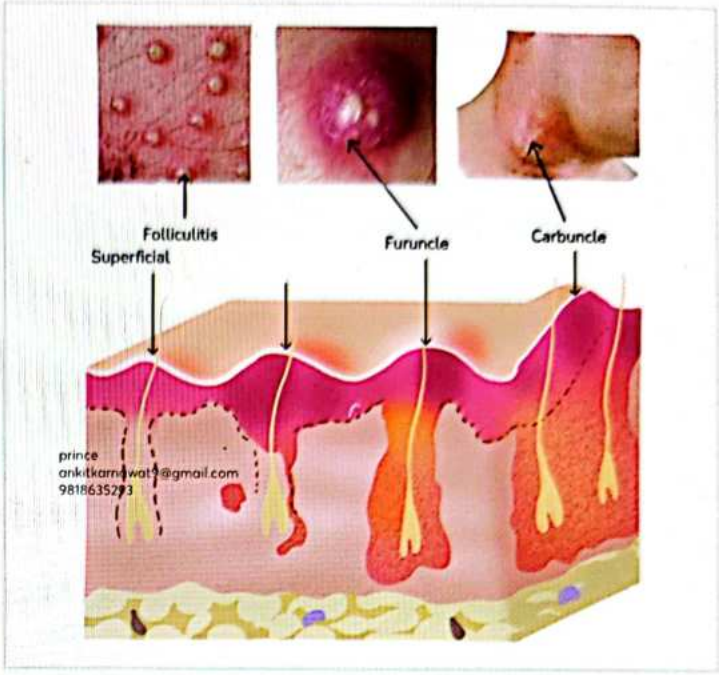
- These are generalized kind of infection (not localized)
- Soft tissue infection
- Present usually on the lower limb/ upper limb with warm tender plaques
- Associated with fever and constitutional symptoms
- Erysipelas
  - It is more superficial
  - Affects superficial dermis and lymphatics
  - Well defined margins
  - Ear involvement seen
- Cellulitis
  - Is deeper
  - Involve the whole of the dermis and the subcutaneous tissue.
  - ILL defined
  - Ear involvement is not seen





Pyoderma: Follicular infection

00:12:58



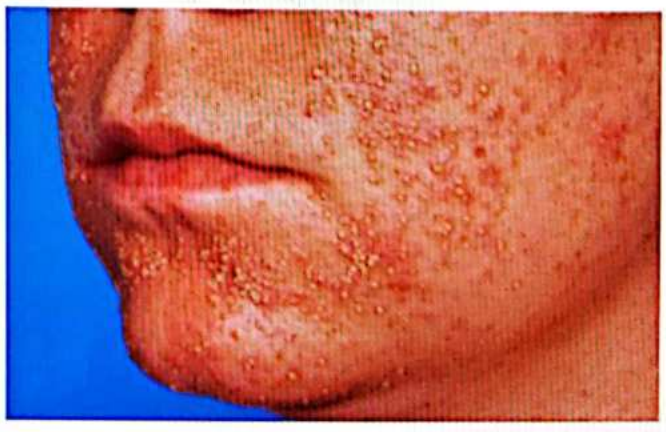
- Superficial folliculitis: Localized infection of upper part of the hair follicle
- Deep infection around the hair follicle, called deep folliculitis.

Folliculitis

- Types
  - Superficial
  - Deep
- Furuncle: severe infection, involves peri follicular area
- Carbuncle: infection involves contiguous hair follicles
- In superficial folliculitis
  - Superficial lesions
  - Located around hair margin
  - Pustular cause little discomfort, not very painful - or is also called Bockhart's impetigo

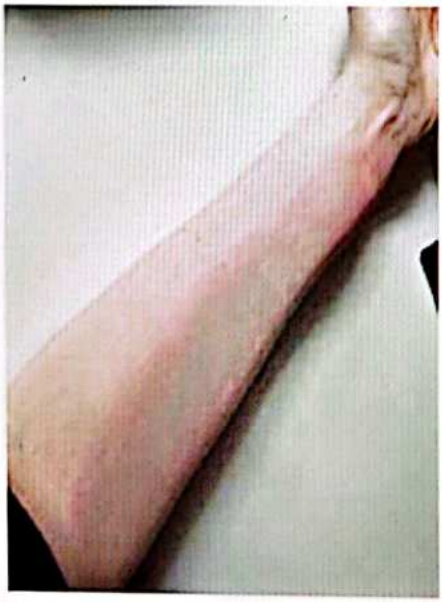
Bockhart's impetigo

00:14:50



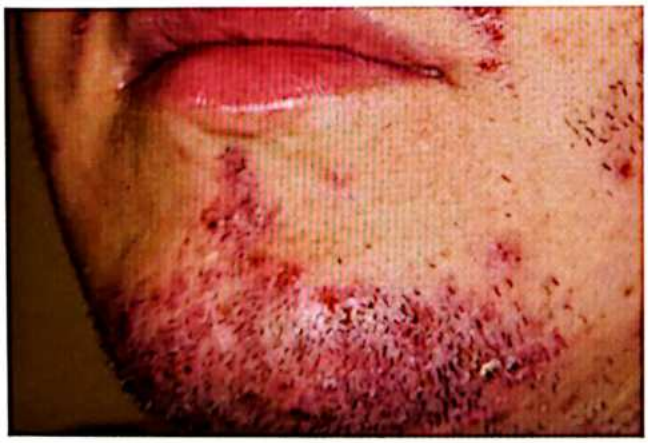
Important Information

- Acute lymphangitis is a streptococcal infection
  - Affects the lymphatic vessels of subcutaneous tissue
  - Seen as an erythematous linear streak
  - Starts from the portal of the entry of the organism
  - Tender and enlarged
  - Management will be like the treatment of bacterial infection.



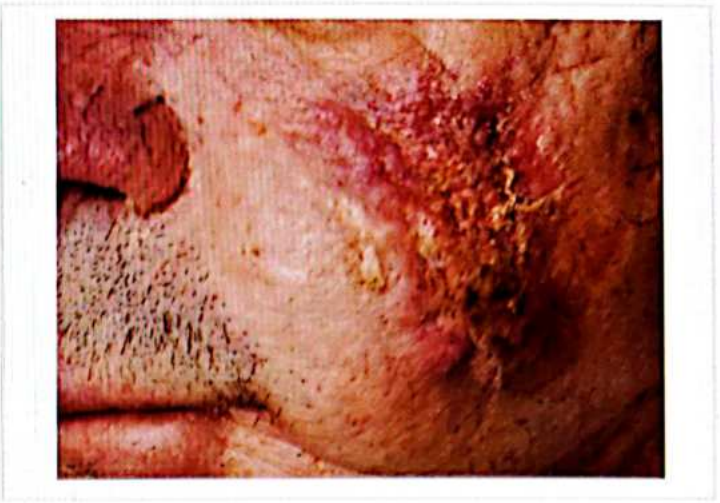
- Bockhart's impetigo is a superficial folliculitis (not an impetigo)
- Also known as Staphylococcus aureus superficial folliculitis
- caused by Staphylococcus aureus
  - Seen mostly in children.
  - Pustules are seen
  - Develop in crops over the scalp and hair margin
  - Also present in the extremities.
  - They heal within a week.
- Chemical folliculitis: Folliculitis caused due to irritation from chemicals
- Pseudofolliculitis: This happens after waxing if it is not done properly.
  - The hair gets damaged, and this leads to inflammation of the hair follicle.

Deep folliculitis 00:16:18



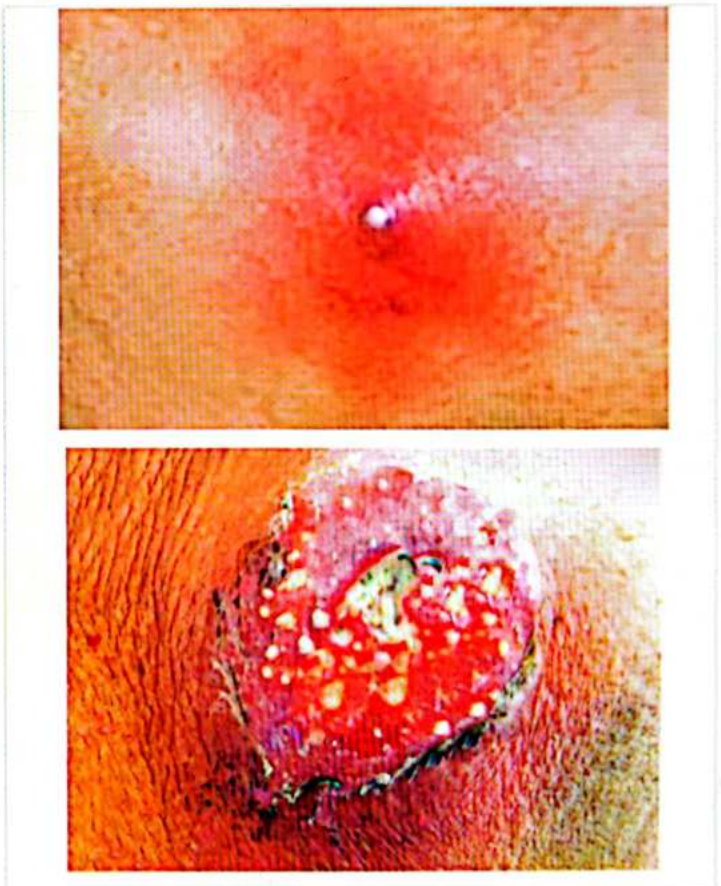
- Folliculitis can be very deep and is called sycosis barbae.
- Misnomer: sycosis means a fungus, but it is a bacterial infection usually seen in the beard area.
- It is differentiated from Tinea barbae, which is usually itchy and has a KOH positivity.
- They appear like a fig tree, fig like appearance

Lupoid sycosis



- Severely deep folliculitis leads to lupoid-like scarring or lupus like scarring.

Peri folliculitis

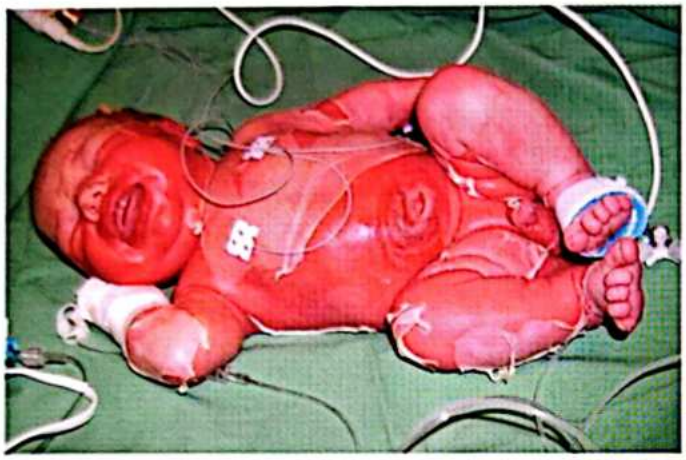


- Furuncle
  - Lot of inflammation around a hair follicle
- Carbuncle
  - Multiple hair follicle being affected
  - It is usually seen in diabetic and in the back
- Both are very painful conditions and requiring oral antibiotic

Bacterial toxin mediated dermatoses

- Staphylococcus causes Staphylococcal scalded skin syndrome.
- Streptococcus causes scarlet fever
- Both can cause toxic shock syndrome
- Staphylococcal scalded skin syndrome is also called Ritter's disease
  - It is different from Reiter's disease which is to be discussed in systemic disease in skin.

Staphylococcal scalded skin syndrome



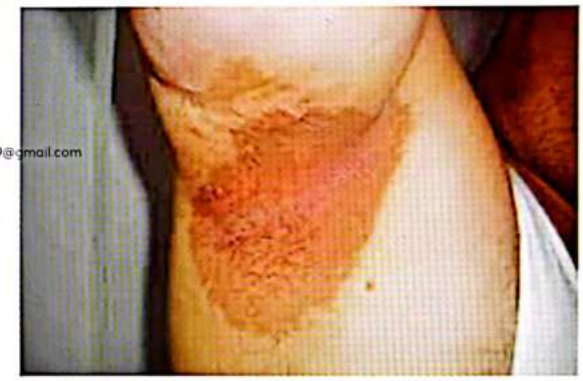
- This is seen in newborn
- The primary in the form of Bullous impetigo, ear infection caused by Streptococcus, which releases toxin called exfoliative toxin 1, which binds to Desmoglein-1 causing intraepidermal split (sub corneal split)
- Presentation:
  - Newborn with fever
  - Constitutional symptoms and might have had a Bullous impetigo
  - Ear infection
  - Peeling of skin.
- Positive nikolsky sign is seen
  - Also present in pemphigus group of disorder
  - Positive in Staphylococcal scalded skin syndrome
  - Positive in a drug reaction called toxic epidermal necrolysis
    - → Pseudo Nikolsky sign because it is not happening because of antigen or antibody deposition but because of keratinocyte necrosis and deposition.
- Infections caused by staphylococcus and streptococcus

• Staphylococcus aureus • Impetigo • Follicular infections, e.g. superficial folliculitis, deep folliculitis (sycosis barbae and sycosis nuchae), furuncles and carbuncles • Ecthyma (strep>staph). • SSSS, TSS	Streptococcus Pyogenes NBI Ecthyma Erysipelas Acute lymphangitis,

Corynebacterium infections

00:23:21

- Resident flora
- Etiology:
  - Healthy adults
  - Hot and humid climates
  - Asymptomatic/pruritic
- Site:
  - Most common - toe webs followed by the groin crease
- Three infections:
  - Erythrasma
  - Pitted keratolysis
  - Trichomycosis axillaris
- Erythrasma is caused by specific species of Corynebacterium minutissima in presence with asymptomatic or hyperpigmentation that may be present on the axilla, groin or interweb space.



- Wood's lamp test: To differentiate it with tinea

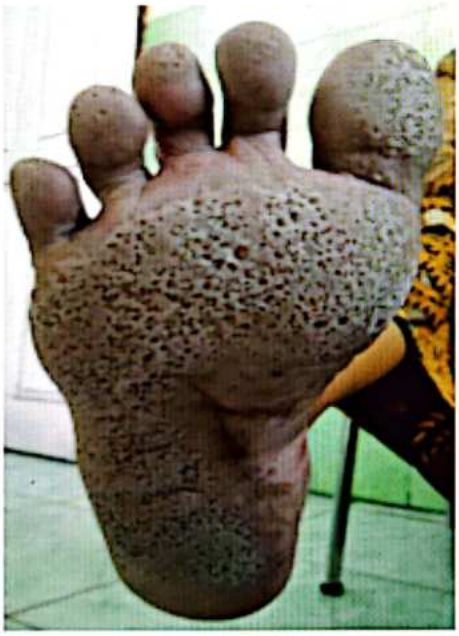
- One sees coral red fluorescence because this Corynebacterium produces coproporphyrin 3.
- Treatment:
  - Tropical therapies include fusidic acid, clindamycin, or erythromycin

- Oral options: clarithromycin, erythromycin

Trichomycosis axillaris 00:25:55

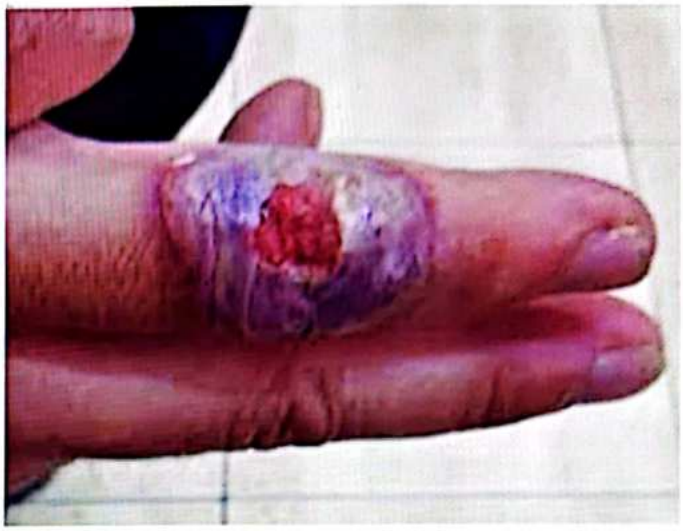
- It is misnomer and not caused by fungal infection
- Corynebacterium causes this.
  - Corynebacterium tenuis (yellowish/brown granules present in axillary hair)
  - Corynebacterium propinguum
  - Corynebacterium flavescens
  - Serratia marcescens

Pitted keratolysis 00:26:53



- Micrococcus sedentarius (now renamed to kytococcus sedentarius)
  - leads to Pitts in feet.
  - These superficial pits are asymptomatic on feet.
- Dermatophilus congolensis or species of Corynebacterium, Actinomyces, or streptomyces.
- Seen in individuals who have
  - Hyperhidrosis
  - Swimmers
  - Wet work
- Feet will be macerated here.

Anthrax 00:28:39

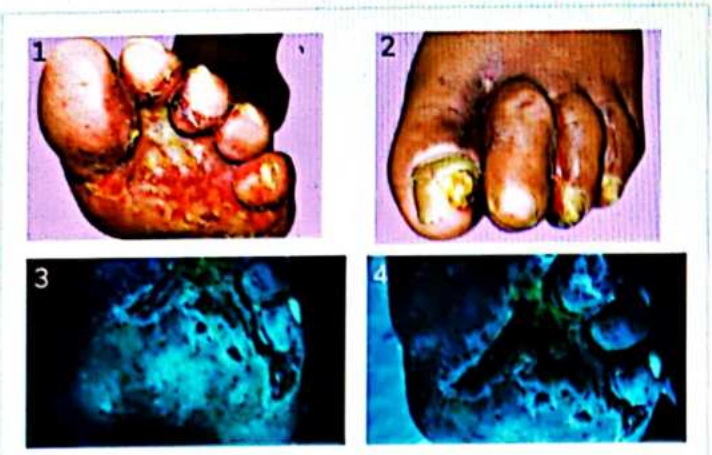


- Caused by:
  - Bacillus anthracis
  - Zoonotic
  - Livestock handlers
  - Malignant pustule - no puss or no pain
- Development of papules or nodules which increase in size with pus to leave behind bluish regions on the exposed part.

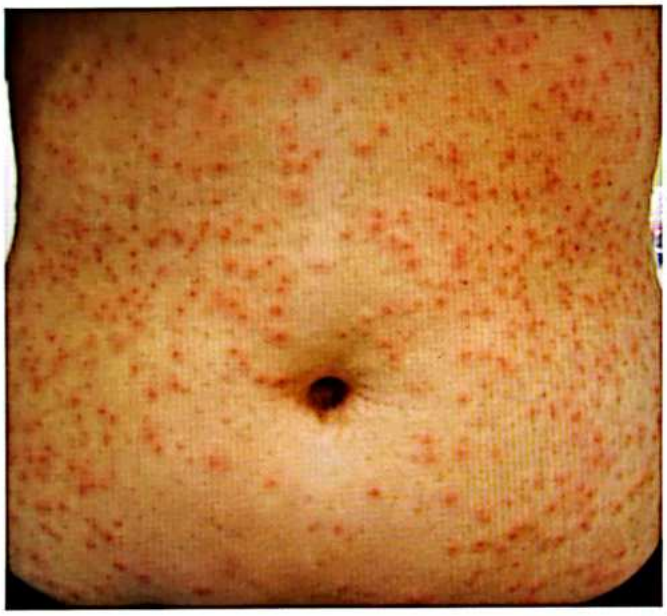
Pseudomonas skin infections 00:29:32

- Pseudomonas aeruginosa
- It can cause green nails Syndrome

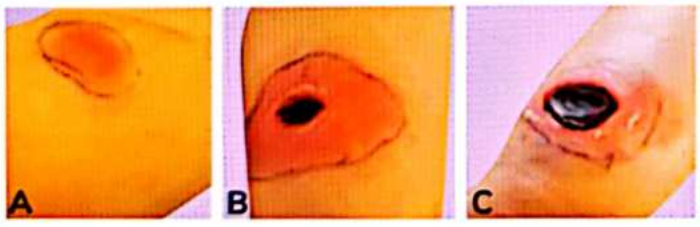
- Pseudomonas produces a toxin (pyoverdin) which is green in colour.
- Sometimes, inter web infection can happen.



- To differentiate with other disease:
  - Do wood's test - greenish fluorescence.
- It also causes hot tub folliculitis.



  - History of taking baths in Jacuzzi or contaminated swimming pools.
  - On trunk: patient develop - multiple monomorphic erythematous papules on trunk.



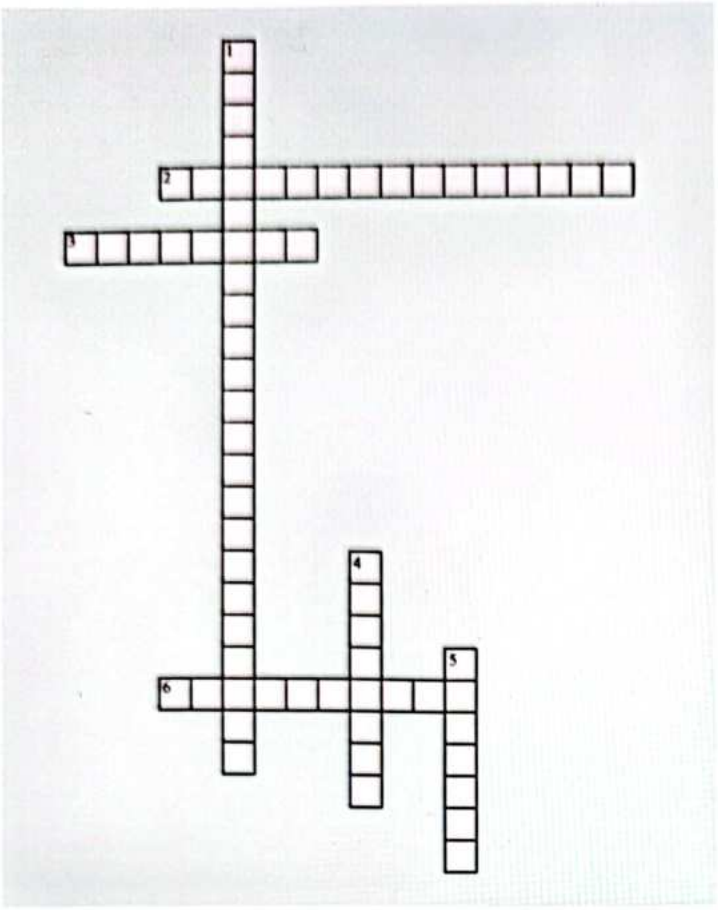
- Three types of Ecthyma:
  - Ecthyma Contagiosum - parapox
  - Ecthyma Infectiosum - streptococcus
  - Ecthyma Gangrenosum - pseudomonas
- Critically ill/immunocompromised
- Hemorrhagic pustules that evolve into necrotic ulcers
- Eschar - Hemorrhagic pustules ruptures and causes black heaped up crust known as Eschar.
  - Some others:
  - Necrotising fasciitis
  - Gangrenous cellulitis
  - Ear infection
  - Treatment antipseudomonal beta-lactam (penicillin or cephalosporin), aminoglycoside, carbapenems (imipenem, meropenem)

Management 00:33:22

- Local hygiene
- Elimination of immunocompromising factors such as
  - Malnutrition, steroid therapy, and other systemic conditions like diabetes
- Remove crust by using condy's compresses –
  - $KMNO_4$ (potassium permanganate)
  - $KMNO_4$ has very good antiseptic and astringent activities.
  - In water, put 1 or 2 crystals and dilute them to the tune of 1:1000.
  - And then use compresses to remove the crust.
  - This way, remove the crystal and crust.
- It is a localized infection; topical antibacterial agents can be used:
  - Bacitracin
  - Polymyxin B
  - Neomycin
  - Sodium fusidate
  - 1% framycetin
  - Mupirocin
  - Retapamulin - newer agent
  - Ozenoxacin - newer agent
- Deep infections, immunocompromised patients use:
- Systemic treatment:
  - → Beta lactamase resistant penicillin, e.g., methicillin, nafcillin cloxacillin, dicloxacillin
  - → Amoxicillin
  - → Erythromycin, cotrimoxazole and the cephalosporins.
  - → These are oral groups of drugs.
- Recurrent infections:
  - Increased bacterial carriage
  - Intranasal mupirocin (apply it inside the nose)
    - → To stop colonization of streptococcus
    - → Stop recurrent infection.
  - Clindamycin (if serious, orally) - 150 gm of BD for 3 months
  - Rifampicin 600 mg od for 10 days

CROSS WORD PUZZLES

Crossword Puzzle



Across

2. infection caused by resident flora which can turn into pathogens by increasing the quantity
3. most common bacterial infection which is usually seen in children on the face
6. a kind of flora that causes diseases

Down

1. infection is localised on the upper part of the hair follicle
4. called bacterial infections
5. a deep infection caused by streptococcus

4 FUNGAL INFECTIONS OF SKIN

Classification of the fungal infection

- **Superficial:**
  - It is on the upper surface of the skin, i.e., on the epidermis.
  - Transmitted by formide or person to person.
- **Subcutaneous:**
  - It is deep into the dermis of the skin.
  - It is caused due to the implantation injury.
- **Systemic**
- **Opportunistic**

Superficial fungal infection

00:00:58

- It is further of two types:
  - **Superficial**
    - → It occurs on the Stratum corneum.
    - → It will be asymptomatic.
    - → There will be no inflammation.
  - **Cutaneous:**
    - → It will go deeper into the stratum corneum
    - → It will be symptomatic.
    - → It might show inflammation.

Pityriasis Versicolor

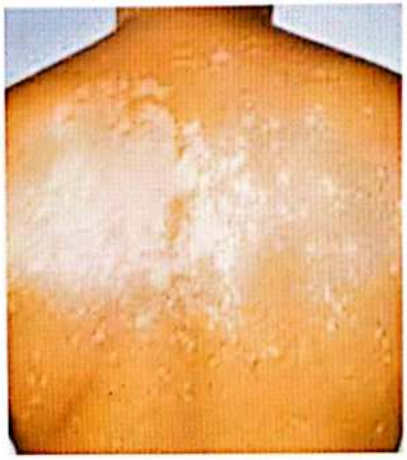
Name:

- It is also known as Tinea Versicolor - Misnomer

Organism:

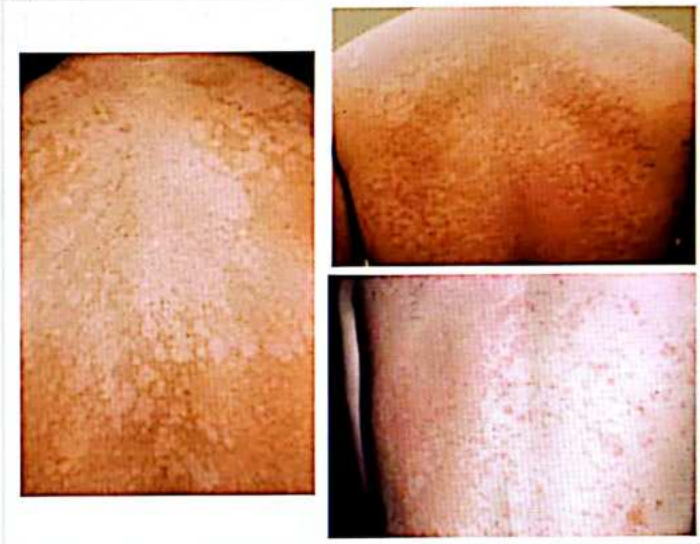
- It is due to Malassezia.
- The species are, Globosa, furfur and sympodialis.
- They occur in Seborrheic areas.
- It mostly occurs in the hot, humid season.
- It affects adolescents and adults.
- It relapses every year.

Presentation:



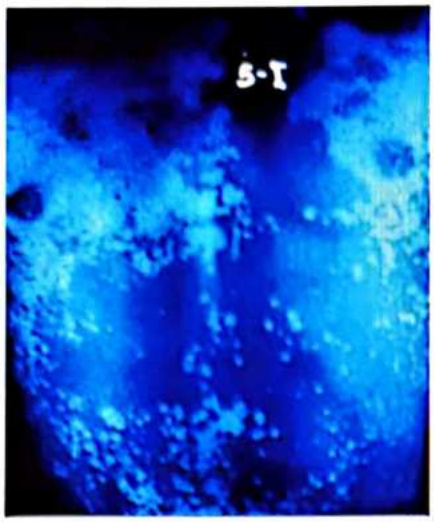
- Pityriasis means scaly, versi means various.
- Therefore, it is presented as asymptomatic hypopigmented and sometimes hyperpigmented.
- The hypopigmented is more common.
- Multiple patches will be seen in the upper trunk.
- Scales will appear as fine granny scales.
- It is also known as a nail scratch sign or Bernier's sign.
- Hyperpigmentation occurs because the fungus produces carboxylic acid or azelaic acid.
- Hyperpigmentation occurs due to the omission of giant melanosomes.

Examination:



- In order to examine the infection, KOH is tested.
- In KOH, clustered yeast will be seen.
- They are present in the mycelial form.
- It appears as spaghetti, where the meatball represents the yeast and spaghetti represents the mycelial forms. Therefore, it is called spaghetti and meatball appearance or Banana and Grapes appearance.

- If it is seen under the wood's lamp, it will give a yellowing fluorescence.



Treatment:

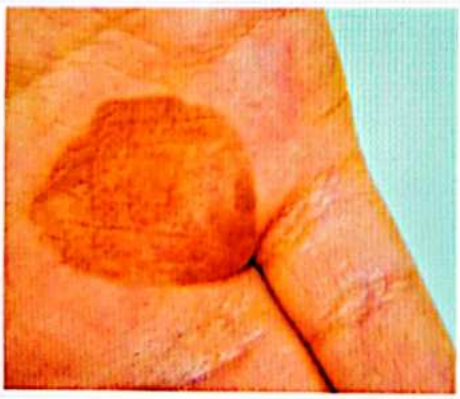
- Mostly topical treatment is given.
  - All kinds of azoles can be given.
  - Zinc Pyrithione.
  - Selenium Sulfide
  - Ciclopirox olamine
- In severe conditions, systemic treatment is given:
  - Azoles.
    - → Fluconazole
    - → Itraconazole

Other Malasszia-associated disorders:

- Malassezia Folliculitis
- Dandruff
- Seb Derm
- CRP (Confluent Reticulate Papillomatosis)

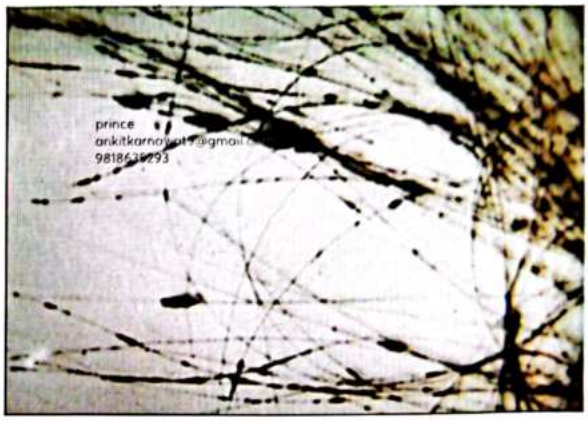
Tinea Nigra: 00:10:55

- It is caused by Hortaea Werneckii, also called Exophiala Werneckii.
- Nigra means Black.
- It is presented as:
  - Asymptomatic
  - Hyperpigmented
  - An annular or round plague on the palm.

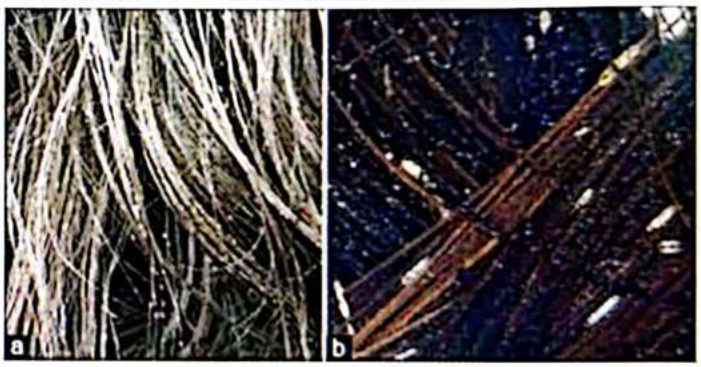


Piedra

- It is further divided as:
  - **Black Piedra:**
    - → It is caused by Piedraia Hortae
    - → It is presented as:
      a. Hard gritty nodules.
      b. Appears on the hair shaft, like the scalp.
  - **White Piedra:**
    - → It is caused by Trichosporon Asahii, also called Trichomycosis Nodosa.
    - → It is presented as:
      a. Soft white nodules.
      b. Appears on other hair-bearing areas.



Piedraia Hortae



Trichosporon Asahii/ Trichomycosis Nodosa

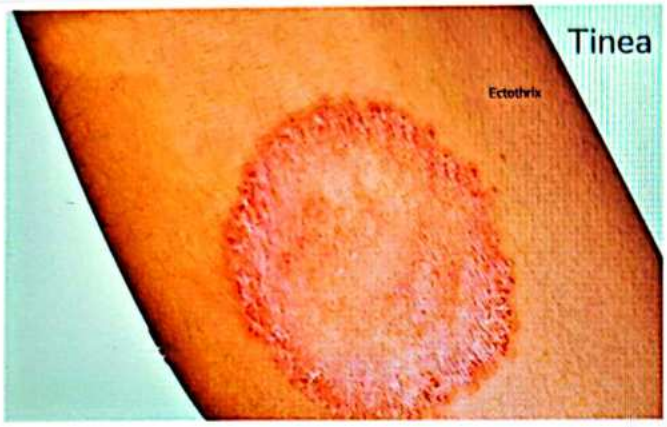
Dermatophytosis

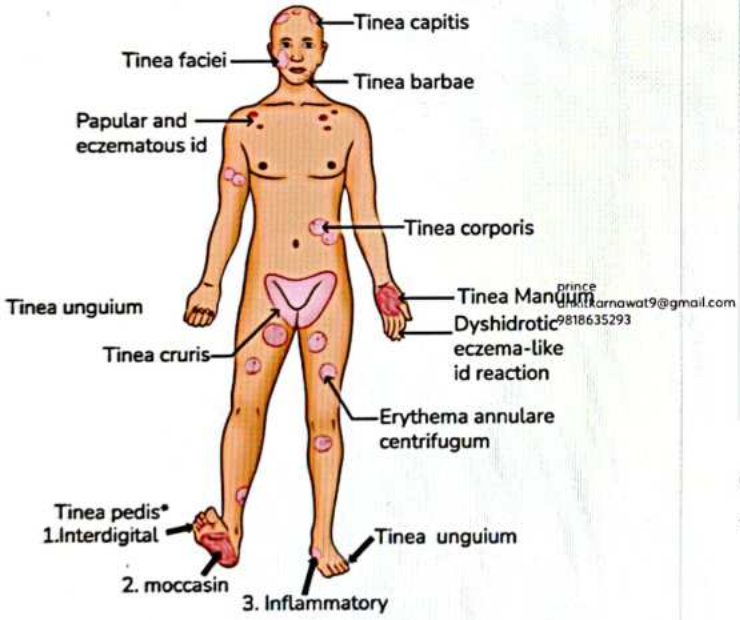
- It is caused by dermatophytes.
  - Keratinophilic.
- They affect the skin, nails and hair.
- They do not affect mucosa.
- They transfer from person to person and through fomites.
- Species that cause the infection are:
  - Trichophyton:
    - → It affects skin, hair and nails.
  - Epidermophyton
    - → It affects the skin and nails.
  - Microsporum

→ It affects skin and hair.

Tinea

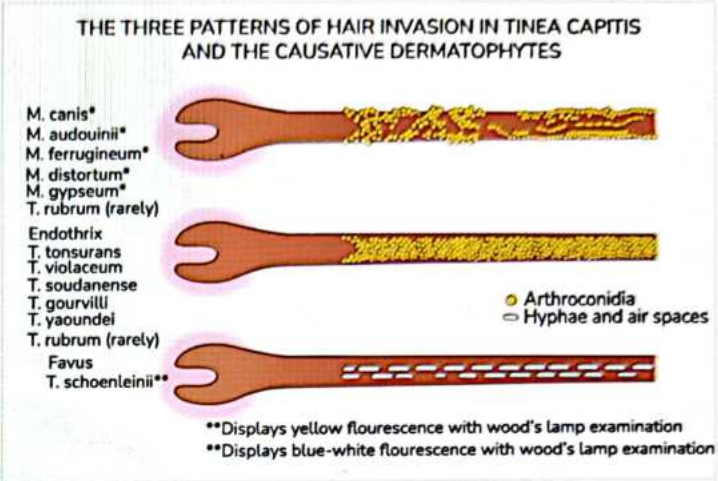
- There will be an erythematous plaque that shows central clearing.
- Peripheral extensions along with active margins:
  - Scales
  - Vesicles
  - Crusts

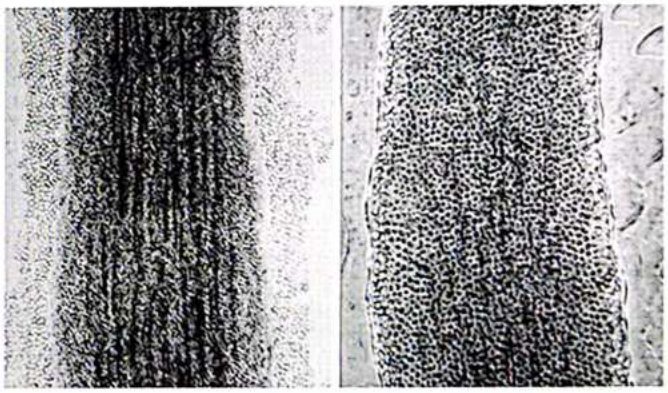




Classification of tinea by sites

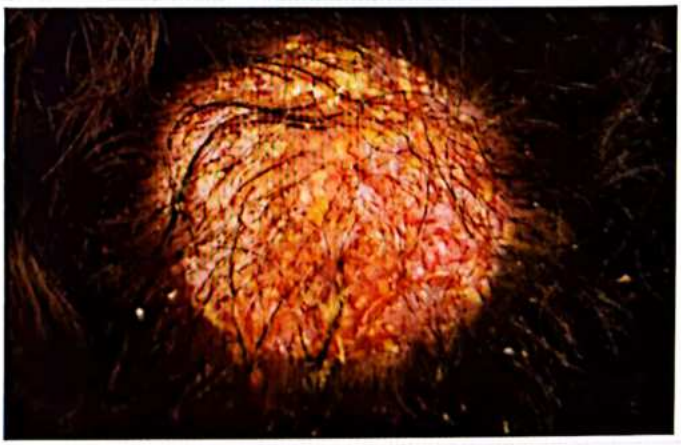
- **Tinea capitis:**
  - It is seen in the scalp
  - Most common mycosis in children.
  - Epidermophyton will not be affected.
  - The most common species that cause the infection is:
    → T. Tonsurans (Most common in developed countries.)
    → M. Cains (Most common in developing countries.)
  - The invasion of the fungus:
    → If the fungus is invading from outside and goes inside and releases some spores along with some spores outside, it is called Ectothrix, and Microsporum usually causes it.
    → If the fungus invades the hair shaft and they stay inside, it is called an Endothrix, and Trichophyton usually causes it.
    → If there is a presence of air spaces along with the fungal colony, it is called Favus, and it is usually caused by T. Schoenleinii





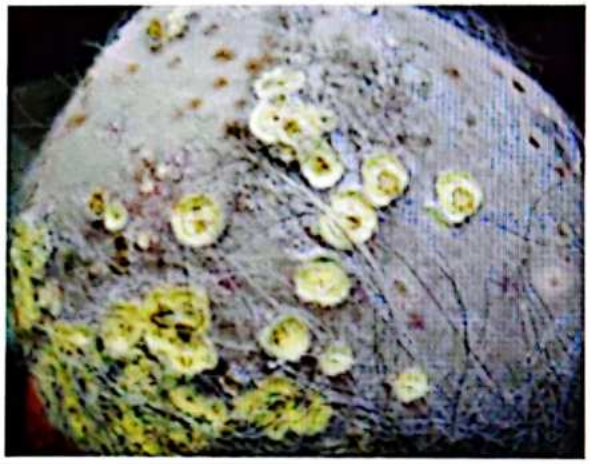
Clinical Classification

- Inflammatory
  - There will be scarring.
  - Some examples:



1. **Kerion:**
- Shows painful boggy swelling.
- Broken hair, alopecia, pustules and folliculitis matting of the hair can be seen.
- Second-degree infection and lymphadenopathy can be seen.
- Most common species are T. ferrugineum, and verrucosum.
- KOH and wood's lamp test will be negative.

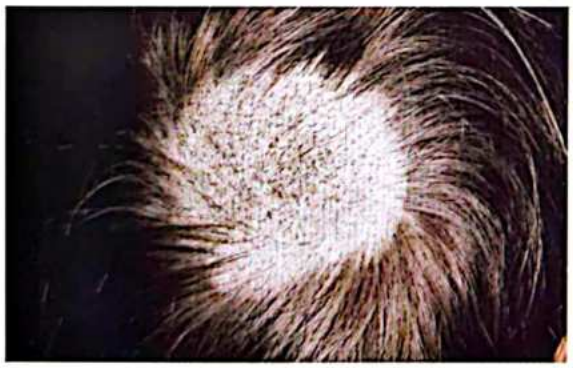
2. **Favus:**



- Shows cup-shaped crusts, also called scutula.
- It is caused by T. Schoenleinii
- Wood's lamp will be positive with dull green fluorescence.

Non-inflammatory.
- There will be non-scarring alopecia.
- Some examples:

1. Gray Patch:



- Most common cause due to the outbreak in school.
- Gray scales are visible.
- Broken hair can be seen
- The most common symptom is itching.
- The most common species that causes Gray Patches is M.canis
- If the wood's lamp is done, it will be positive, showing a bright green fluorescence.

2. Black Dot:

- Angular patches can be seen.
- Broken hair is visible.
- Swollen hair shafts can be seen.
- It is caused by tronsulans, violaceous, and sondenes,
- Wood's lamp test will be negative.

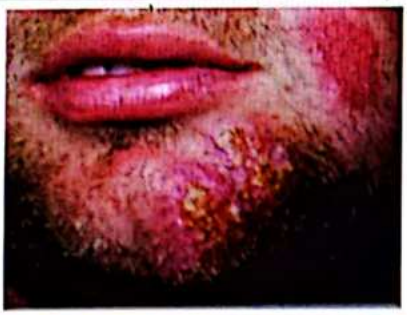
Hair Perforation Test For Dermatophytes
- To distinguish between isolates of dermatophytes, particularly Trichophyton Mentagrophytes and its variants

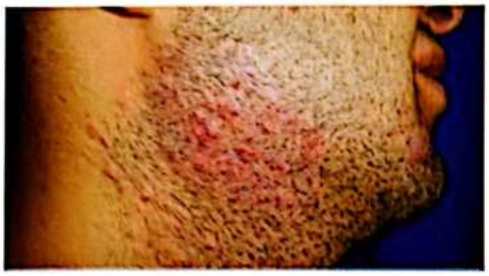
Ingredients
- Autoclaved blonde pre-pubital hair cut into short pieces (1cm)
- Sterile distilled water 5 ml in a suitable vial

Method
1. Place hair in water in vial
2. Inoculate with small fragments of the test fungus
3. Incubate at room temperature
4. Individual hairs are removed at intervals up to 4 weeks and examined microscopically in lactophenol cotton blue. Isolates of T. Mentagrophytes produce marked localized areas of pitting and marked erosion whereas those of T. Rubrum do not

Management:
- Topical shampoos are given to prevent the outbreak in society.
  - Azole
  - Ciclopirox Olamine
  - ZPTO
  - Selenium.
- Systemic:
  - Griseofulvin, which is given 15-20mg/Kg/day
  - Terbinafine works for Trichocytones.
  - Itraconazole.
  - The treatment should be for 4-6 weeks.

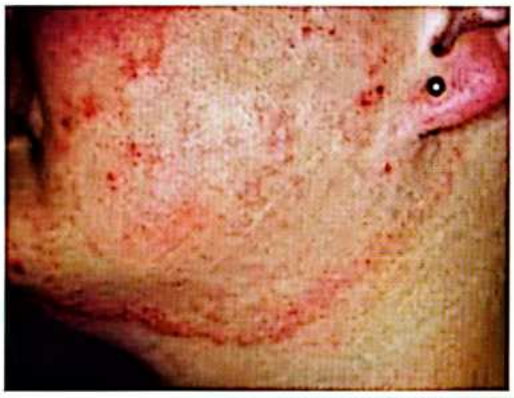
Tinea barbae:





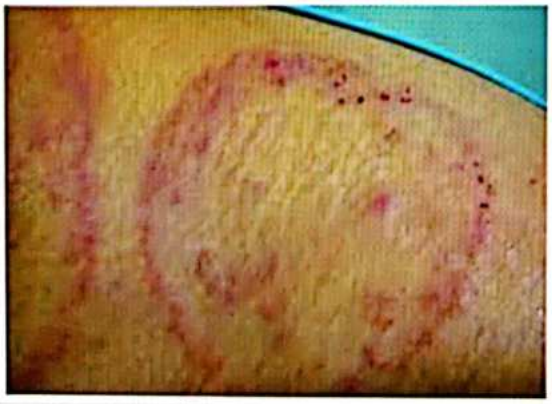
- It needs to be differentiated from Sycosis Barbae.
- Tinea barbae is itchy, and KOH is positive.
- Sycosis Barbae is painful and is seen in perioral areas.

Tinea faciei:

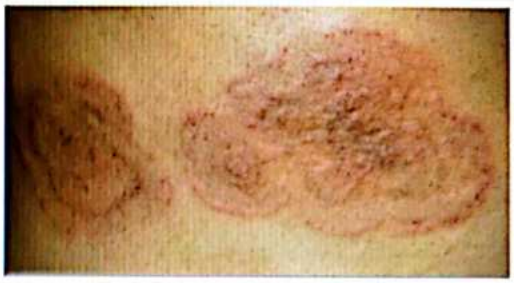


- It is present on the face.
- If it is present as isolated, then rule out an immuno-compromised state.
- Papular and eczematous id

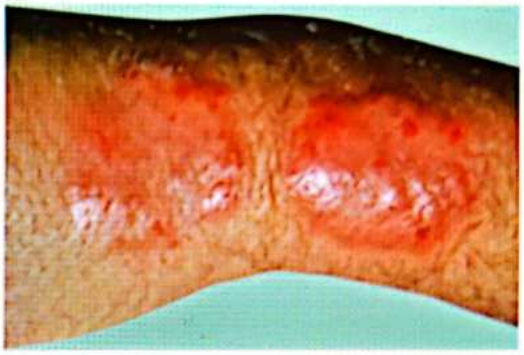
Tinea corporis:



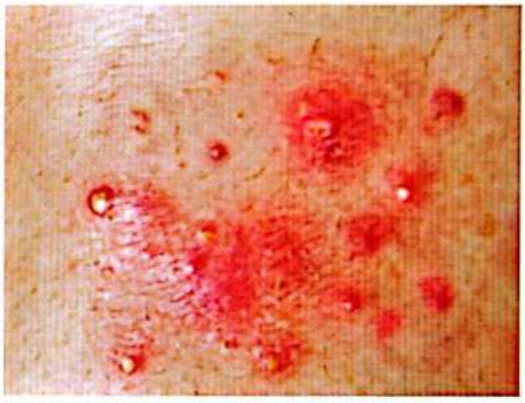
- Most common species is T. Mentagrophytes.
- Different variants are:
  - Tinea Circinata or Tinea Imbricata, caused by T. Concentricum.



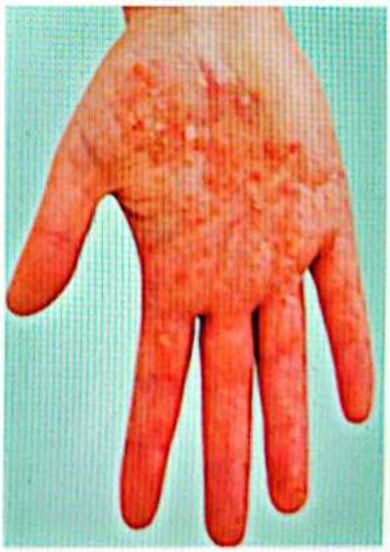
  - Tinea Profunda



  - Majochhi's Granuloma.

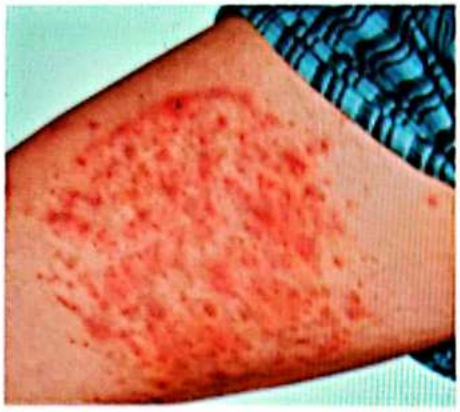


Tinea manuum:



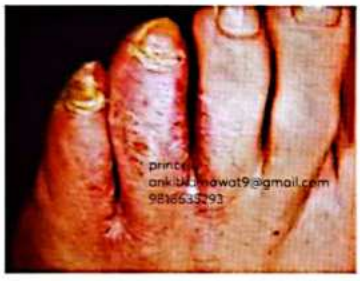
- It happens on the palmar aspects of the hand.
- Dyshidrotic eczema-like id reaction
- Erythema annulare centrifugum

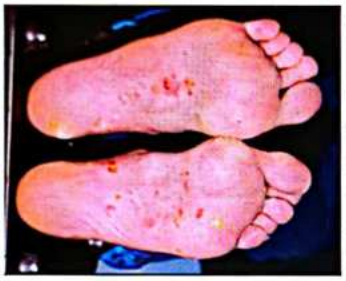
Tinea Incognito:

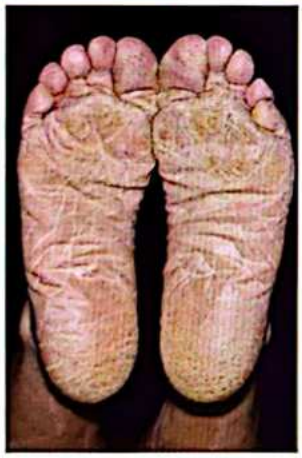


- It is also called as Steroid modified tinea.
- It is caused by the excessive use of Corticosteroids on tinea.
- There is no central clearing or scaling.

Tinea pedis:

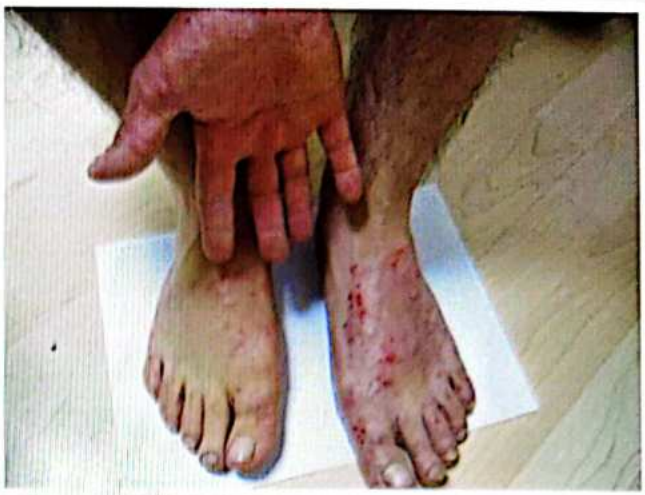




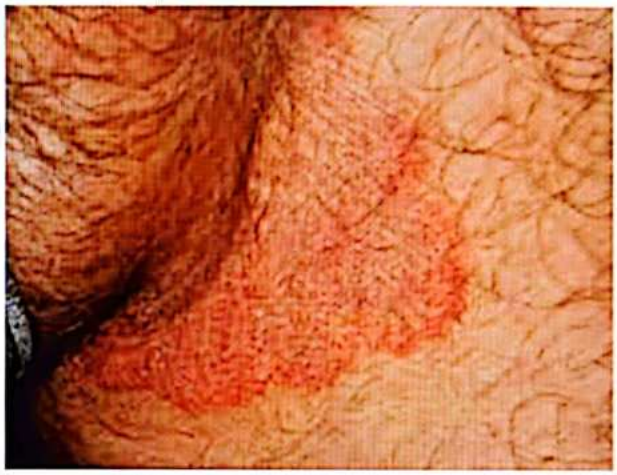


- It happens on the Dorsal aspect of the feet.
- It is also called Athlete's foot.
- Types:
  - Interdigital, it affects the fourth interweb space. (It is the most common type of Tinea Pedis.)
  - Moccasin
  - Inflammatory/ Hyperkeratotic
- It is also called Two feet, one hand syndrome, caused by T. Rubrum.

Two feet one hand syndrome.

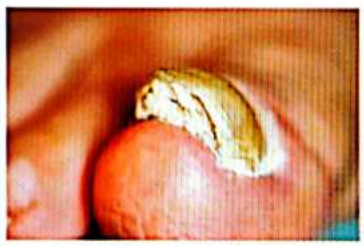


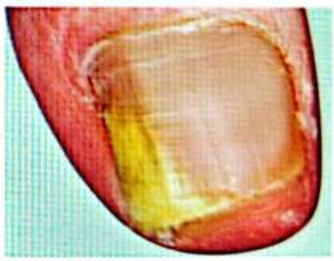
Tinea cruris:

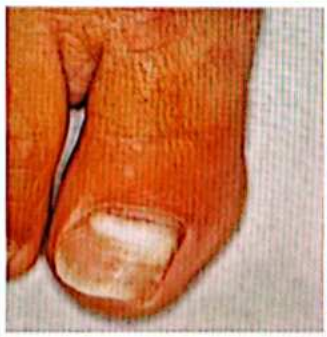


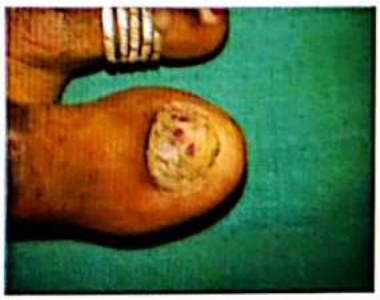
- Cruris means crural folds, which means groin areas.
- This is also called Dhobi's Itch or Jogger's itch because it happens due to a lot of sweating.
- It shows less scaling.

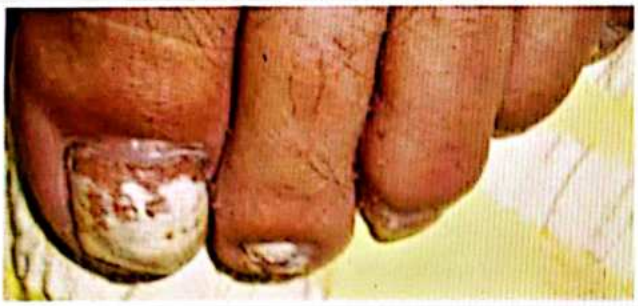
Tinea unguium:











- It happens in nails.
- The terms Onychomycosis is the fungal infection of nails that can be caused by:
  - Tinea
  - Candida
  - Other molds.
- It is asymptomatic.
- There is a yellow discolouration of the nails with the deposition of the Subungual debris.
- It shows subungual hyperkeratosis, which causes Onycholysis.
- It mostly involves toenails.
- The KOH will be positive.
- Types:
  - The most common type is Distal lateral Subungual Onychomycosis.
  - Proximal subungual onychomycosis. It is basically seen in HIV patients.
  - Superficial white Onychomycosis. It shows white crumbly material on the top of the nail, which can be scraped off.
  - Total dystrophic onychomycosis.

Treatment:

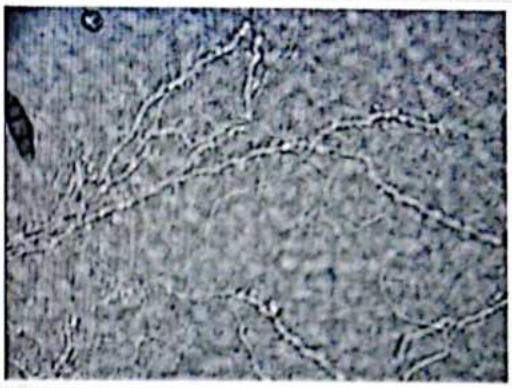
- Topical treatment:
- Nail lacquers.
  - Ciclopirox Olamina which is applied daily.
  - Amorolfine, which is applied weekly.
- Systemic treatment.
  - Fluconazole (160 to 200 milligrams per kg for 6 months for fingernails and 12 months for toenails.)
  - Terbinafine (250 mg OD, 6 weeks for fingernails and 12 weeks for Toenails.)
  - Itraconazole. (200 mg OD 3 months for fingernails and 4 to 5 months for toenails.)
- Pulse therapy:
  - Terbinafine 250 mg BD 1 week per month.
  - itraconazole 250 mg 1 week per month.

Notes:

M. Ferrugineum is a species that will show no fluorescence.

Diagnosis: Dermatophytosis

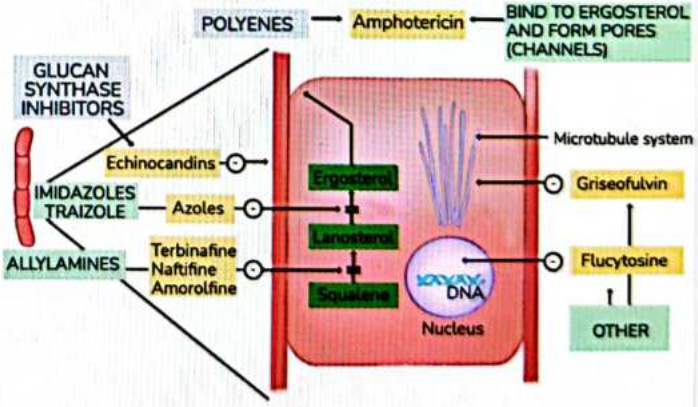
- KOH



- Fungal culture

Treatment: dermatophytosis

- The types of treatment that is most preferably adopted are:
  - Topical
  - Systemic
- The most common type of drug that is being used is:
  - Azoles
    - → Itraconazole
    - → Fluconazole
  - Allylamines
    - → Terbinafine.
    - → Salicylic acid
    - → Whitfield' Ointments: it consists of a benzoic acid.
    - → Castellani's Paint: It consists of:
      - Basic fuchsin
      - Ethyl alcohol
      - Boric acid
      - Acetone
      - Phenol
      - Resorcinol
      - Water



Candidiasis

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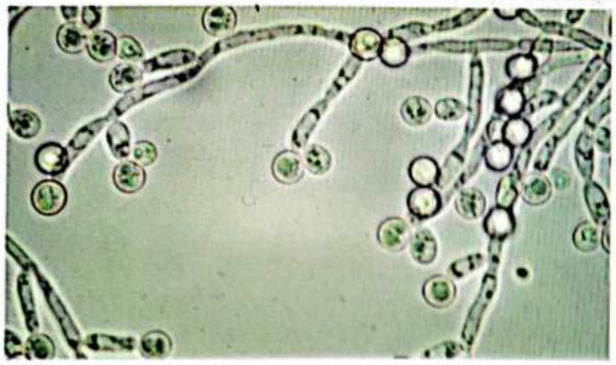
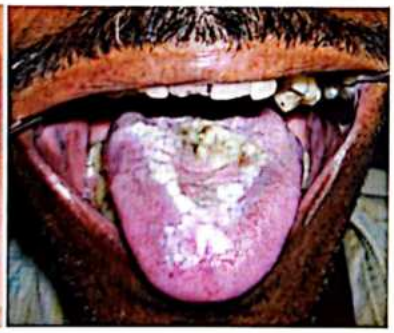


- It is caused by Candida
- The most common species that causes this infection is candida albicans
- It appears as budding yeasts.
- It sometimes forms pseudohyphae
- It affects:
  - Skin: It can cause intertrigo or
  - Mucosal : it can cause an oral infection or genital infection.
  - Nails: It might cause paronychia.
- It does not affect the hair.

Oral candidiasis:

- The patient is symptomatic
- It also shows whitish plaques.
- The two types are:
  - Immunocompetent
  - Immunocompromised.
- Oral Candidiasis is present as:
  - White lesions:

Acute pseudomembranous / oral thrush

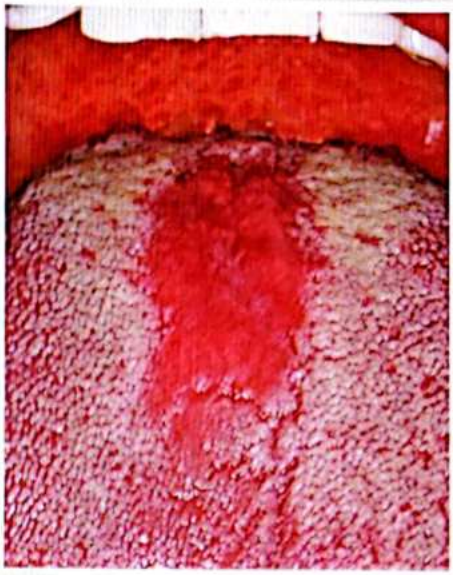


Chronic plaque/candidal leukoplakia

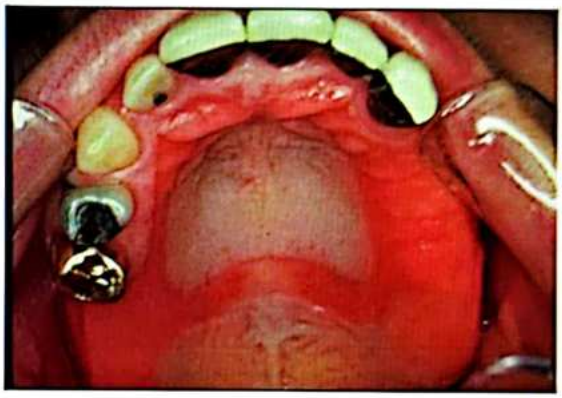
→ Acute pseudomembranous, it is also called oral thrush. It is the most common type. These are the white deposits which can be scraped off.

→ Chronic Plaque, it is also called candidal leukoplakia. These cannot be scraped off.

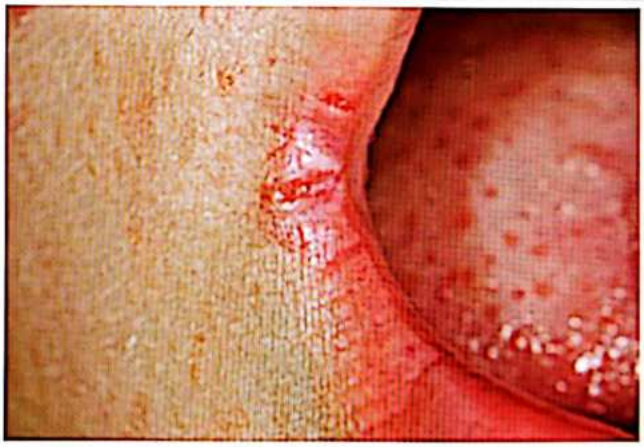
  - Red lesions:



Acute erythematosus candidosis/ Antibiotic sore mouth



Chronic erythematosus/ Denture stomatitis



Angular cheilitis/perleche

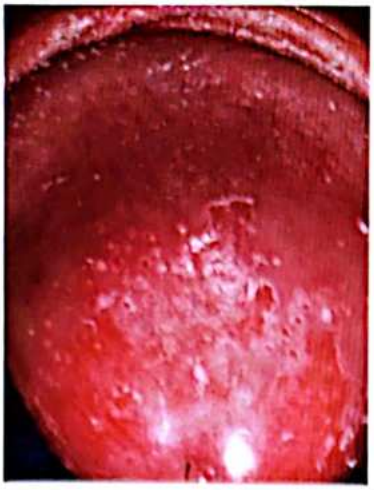
Candida MC infectious cause

→ Acute erythematosus. It is because of the antibiotics.

→ Chronic erythematosus. It is because of the dentures.

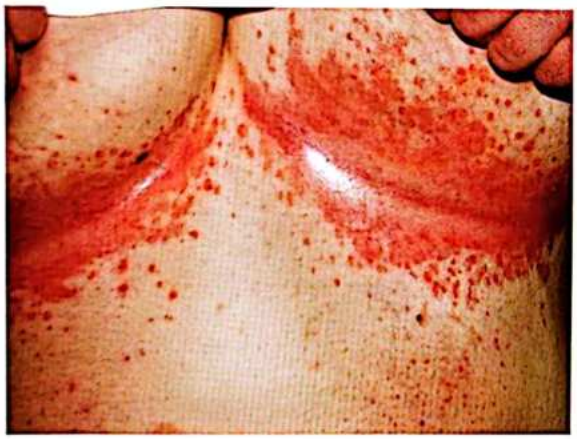
→ Angular cheilitis, it is also called perleche. It appears as fishers at the angle of the mouth.

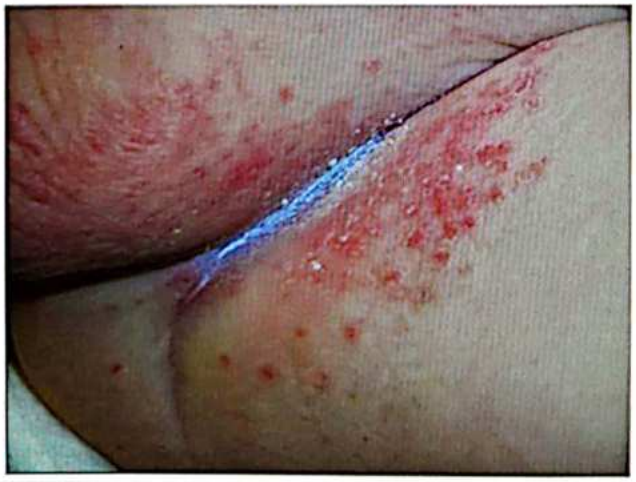
Candidal Balanoposthitis

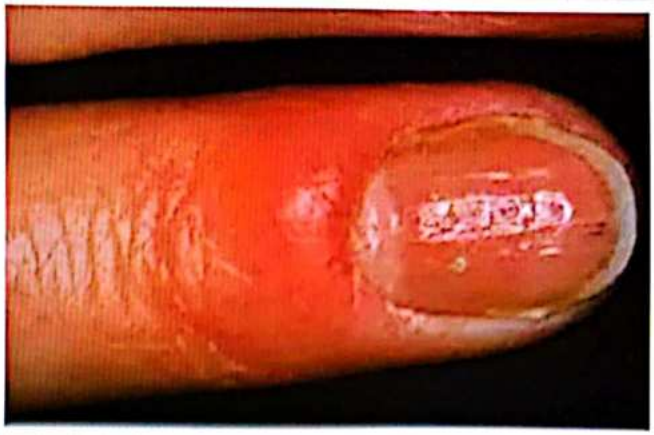


- It is mostly seen in uncircumscribed diabetics.
- It is present as superficial pustules which rupture to get a frayed appearance.
- It also causes superficial erosion, which comes with itching or discomfort sensation.
- In females, it is caused as vulvovaginal Candidiasis, and it comes up as a curdy white itchy discharge in the premenstrual period.

Intertrigo



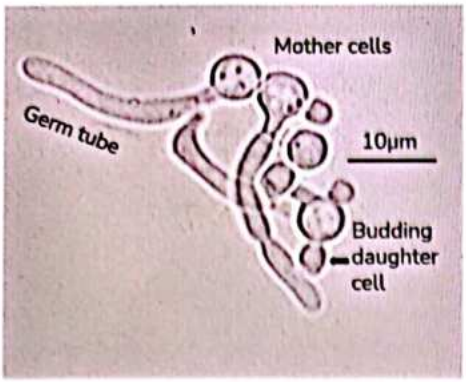


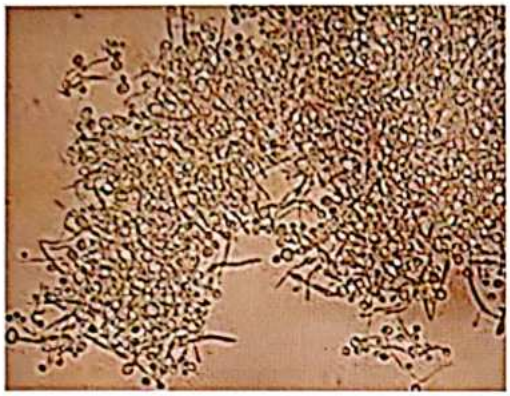


- It causes inflammation of folds
- Candida is one of the causes of this infection.
- These infections occurs in the areas of moisture.
- It shows satellite Pustules.
- It also shows a concave surface of infection.
- It also shows free scaling at the edges.
- Granuloma gluteale infantum is an infection that is caused in infants. Mostly this is also called diaper rash. Sometimes it can also cause nodular lesions.
- In the case of nails, it may cause paronychia which shows inflammation of nail folds. This may lead to pain, tenderness or redness of the nail folds.

Chronic Mucocutaneous Candidiasis

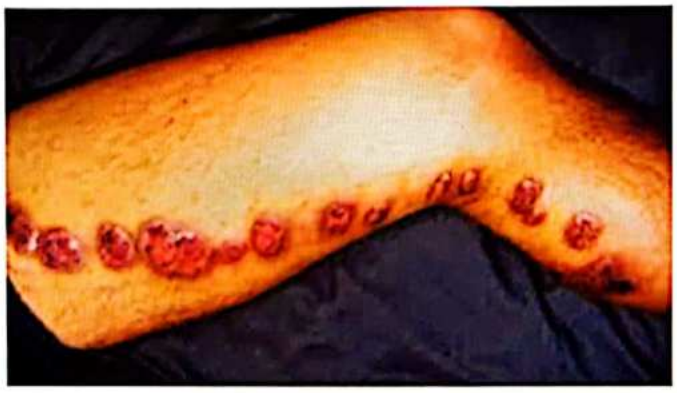
- Persistent Candida infection of the mouth, skin and nails that is refractory to conventional therapy.
- Characterized by a massive invasion of the epithelial cells of the host by C. Albicans.
- Associated with other cutaneous and/or systemic infections, the latter being common in immuno- suppressed patients
- Many patients of CMC show significant systemic abnormalities, e.g. hypothyroidism, hypoadrenalism, hypoparathyroidism, diabetes mellitus, iron deficiency, chronic hepatitis, hemolytic anemia and hypovitaminosis A.
- Investigation:
  - KOH: This shows budding yeast cells
  - Fungal culture: It shows a creamy white colony.
  - Germ Tube Test is a screening test which is used to differentiate Candida Albicans from other yeast. When Candida is grown in human or sheep serum at 37°C for 3 hours, they forms a germ tubes, which can be detected with a wet KOH films as filamentous outgrowth extending from yeast cells.



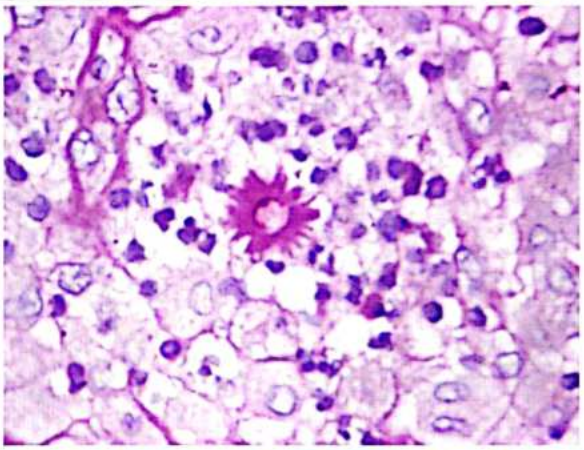


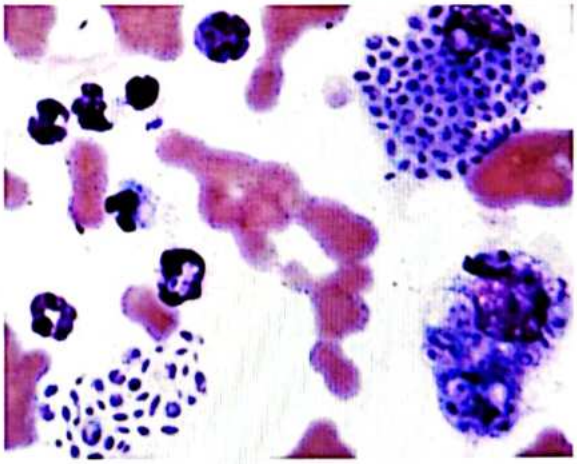
- Treatment:
  - Only azoles are used, and allylamine is not used.
    → Itraconazole and fluconazole are used.
  - Sometimes Nystatin is used with azoles.

Subcutaneous fungal infections



- These are the infections that go deep into the dermis.
- They are caused due to some implantation injury.
- There are 3 types of subcutaneous infection.
  - **Sporotrichosis:**
    → It is also called the Rose gardener's disease.
    → The basic agent for this infection is the sporothrix schenckii.
    → These are categorized into further 2 types:
      a. Fixed type
      b. Lymphangitic type:
        1. It is the most common type of infection.
        2. It shows papules or ulcerated nodules along the lymphatics.
        3. This fungus has a tendency to spread by lymphatics.
        4. This will cause the appearance of numerous lesions along the lymphatics.
        5. It is also called a Sporotrychoid pattern.
        6. If a biopsy is done on these patients, they show asteroid bodies.





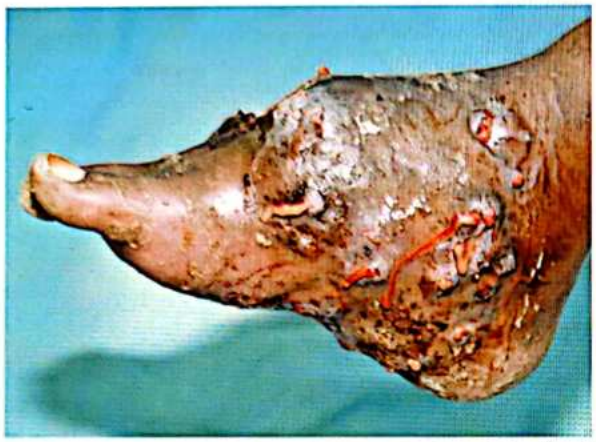
        7. This phenomenon is called Splendor Floppy Phenomena
        8. In the PAS stain, a cigar-shaped body is seen.
      c. Treatment:
  - Itraconazole (4 to 6 months of treatment)

- Potassium iodide
- Terbinafine.

Note:

- Germ Tube Test is a screening test which is used to differentiate Candida Albicans from other yeast.
- When Candida is grown in human or sheep serum at 37°C for 3 hours, it forms a germ tube, which can be detected with a wet KOH film as filamentous outgrowth extending from yeast cells.

Mycetoma: 01:17:50

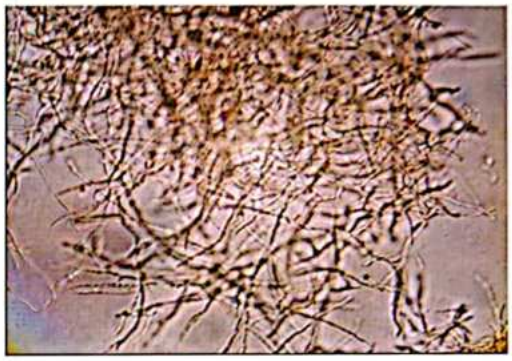


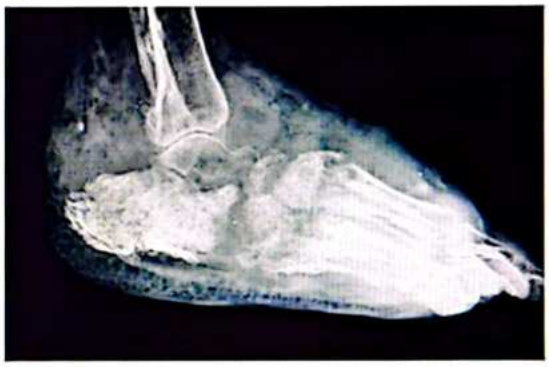
- It is also called Madura Foot or Maduromycosis.
- It is mostly seen in farmers, in their lower limbs.
- The basic and most common agent is M. Mycetomatis.
- It can be caused by bacteria or fungi
- If it is caused by bacteria, it is called actinomyces. And if it is caused by the fungus, it is called eumycetoma.
- It shows painless swelling.
- The mycetoma triads are:
  - Tumefaction: It shows the induration of the skin.
  - Sinuses: it shows multiple discharging sinuses.
  - Grains: These are fungal or bacterial colonies.
- It can also go deep into the bone and can cause Bony deformity.

Difference:

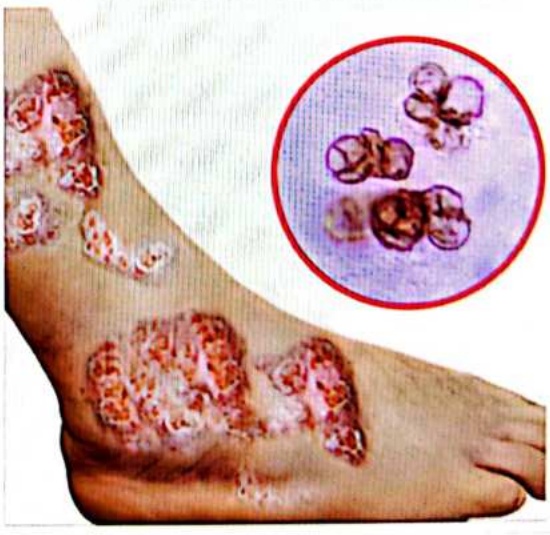
Eumycotic Mycetoma	Actinomycotic Mycetoma
• Slowly invasive • Late presentation, as it is relative • asymptomatic • No pus • Black brown granules • More deformity • Granules 4-5 microns, in clusters	• Rapidly invasive • Early presentation • Pus present • Granules yellowish white • Less deformity • Granules < 1 micron lie singly

• Gram -ve GMS, PAS +ve
• Responds to antifungals (Itraconazole, amphotericin B)
• KOH: Branched Thick Hyphae
• Antifungals for a long time or surgical treatment.
	• Gram + ve , GMS PAS - ve
	• Responds to antibiotics (sulphonamides, doxycyclines)
	• KOH: Thin Hyphae
	• Treatment:
	  ○ Dapsone + Strepto, Cotrimoxazole + Strepto
	  ○ 2nd line - Rifampicin

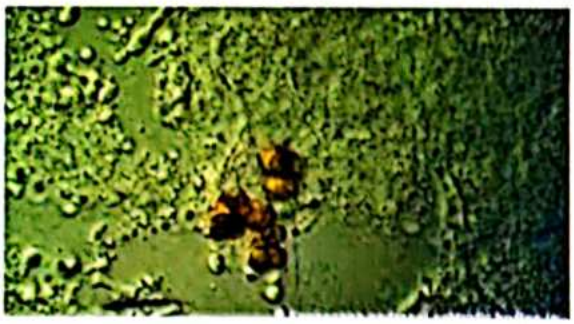




Chromoblastomycosis



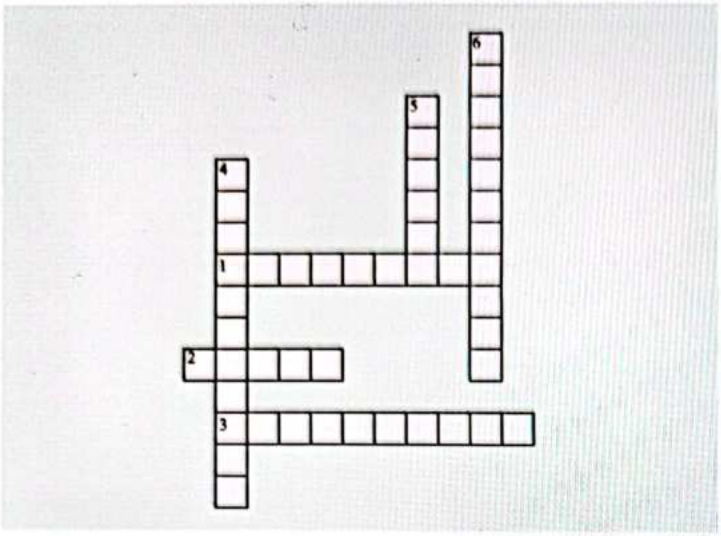
- Cromo means colorful, blasto means vegetative.
- It is presented as pigmented verrucous plaque on the lower limbs.
- If KOH is done, Pigmentation cells will be seen.



- There will be the appearance of a sclerotic copper penny or muriform body.
- Treatment:
  - Itraconazole
  - Potassium iodide
  - Heat therapy.

CROSS WORD PUZZLES

Crossword Puzzle



Across

1. Tinea Nigra is aka = Exophiala
2. Pityriasis Versicolor is aka = Tinea
3. Candida is one of the causes of this infection = Intertrigo

Down

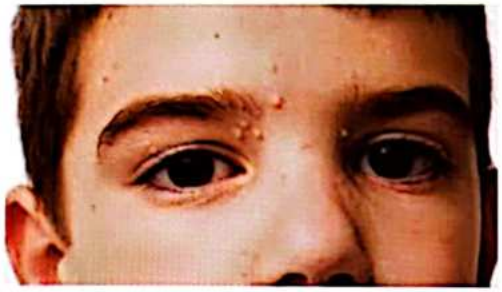
4. Classification of the fungal infection which is on the upper surface of the skin = Superficial
5. Dermatophytosis don't affect = mucosa
6. The most common species that causes this infection is candida albicans = Candidiasis

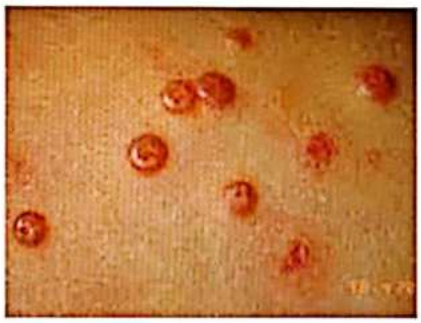
5 VIRAL INFECTIONS OF THE SKIN

Molluscum Contagiosum

00:00:16

- Virus - **MCV** - Molluscum contagiosum virus
- Family - **POXVIRUS**
  - Largest known viruses
  - DNA viruses
- Types
  - **MCV 1** - children
  - **MCV 2** - adults
    → Sexually transmitted





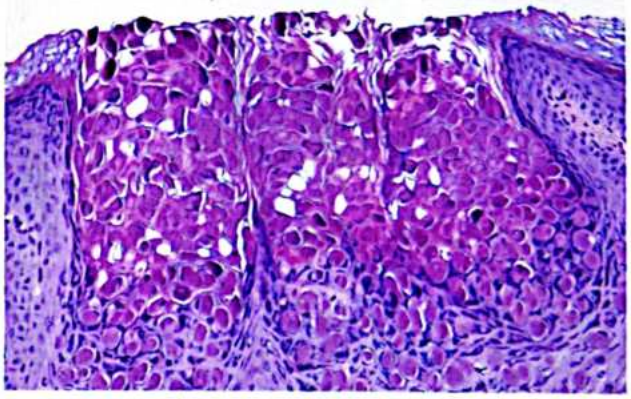
- The lesion in molluscum - **umbilicated papule**
- Usually seen in **children**

Clinical Features

- Asymptomatic
- Common in children
- Positive family history
- Face and other body parts
- **When present in the genital area → rule out sexually transmitted infection**
- Umbilicated papules - skin colored /red
  - **Central indentation**
- Shows Pseudo Koebner's Phenomenon

Important Information

- Pseudo Koebner's Phenomenon - seen in infections
  - Molluscum
  - Warts
  - New lesions arise along the line of trauma
  - Due to-**Autoinoculation/seeding of viral organisms**
- Koebner's phenomenon - psoriasis



Histopathology - molluscum

- Eosinophilic intracytoplasmic inclusion bodies - Henderson Patterson Bodies

Treatment

- Self-resolution
- Many lesions/adults / do not respond
- No oral treatment

Chemical method

1. Trichloroacetic acid - TCA
2. KOH

Physical method

1. Needle extirpation
2. Electrocautery
3. Radiofrequency
4. Cryotherapy

- Systemic treatment
  - in HIV positive
  - Severe lesions
  - Immunomodulators
    → Levamisole
    → Cimetidine

Other Poxvirus Infections

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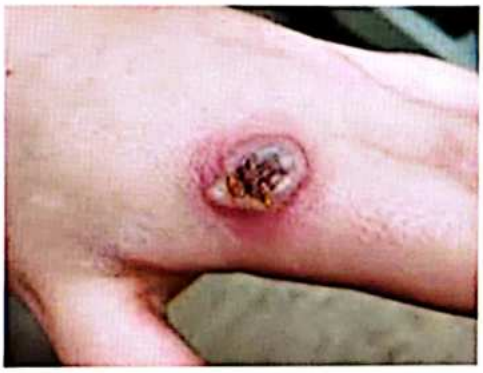
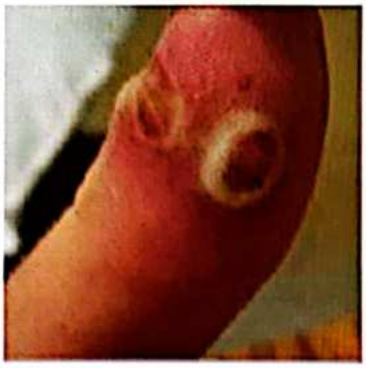
Ecthyma Contagiosum



- Zoonotic infections - Transferred by animals
- **Para poxvirus / ORF virus**
- Sheep

Milker's Nodule



Pseudopox Milker's nodule cow

- Pseudopox
- Affects those who rear cows
- Erythematous bluish papule→ pus → crusted lesions
- On the exposed part of the body

Ecthyma

- Ecthyma infectiosum - streptococcus
- Ecthyma contagiosum - parapoxvirus/orf virus
- Ecthyma gangrenosum - pseudomonas
- Pseudomonas also cause pyoderma gangrenosum

Human Papillomavirus

- Warts/verruca
- HPV > 200 subtypes
- Different subtypes cause different types of warts
- Asymptomatic
- Growths in the skin - **verrucous - hyperkeratotic**

HPV Types of Viral Infection

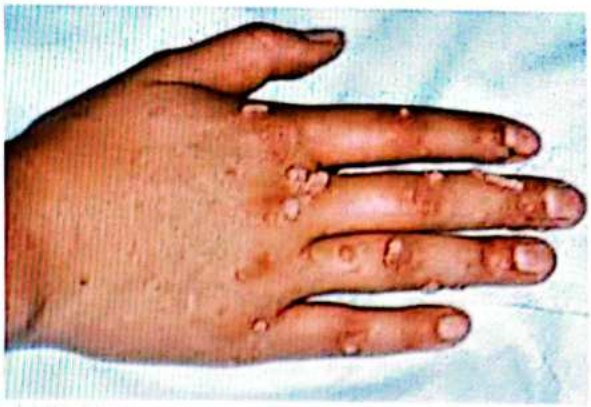
- 1 - Deep plantar wart
- 2 - Superficial plantar warts
- 3 - Plane warts
- 4 - Common warts
- 5 - EDV- epidermodysplasia verruciformis
- 6 - Anogenital warts
- 7 - Butcher's warts

Mnemonic - DSP Come Eat A Burger

Additional Types

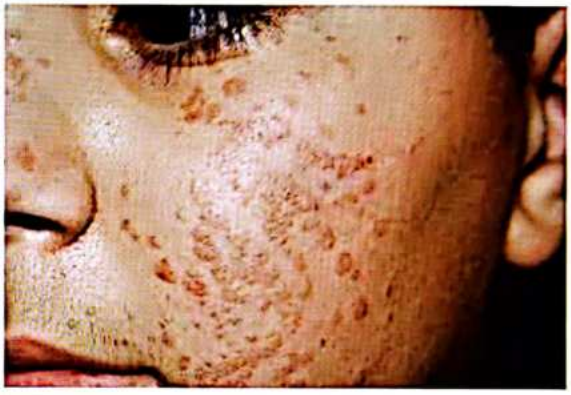
- EDV - is also caused by HPV - 8
- Common warts - HPV 4,2
- **Anogenital warts - HPV 6,11,16,18**
- Plane warts - HPV 3,10

Common Warts - Verruca Vulgaris



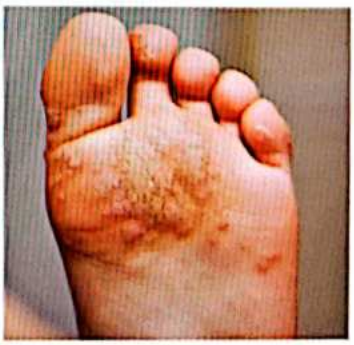
- Asymptomatic
- Hyperkeratotic growths
- Pseudo Koebner's phenomenon

Plane Warts - Verruca Plana



- Plane top
- Commonly seen in face
- HPV 3,10
  - In HIV and immunocompromised patients
  - Kidney transplant
  - Chemotherapy
- Transmitted from person to person or fomites - if there is a breach in skin

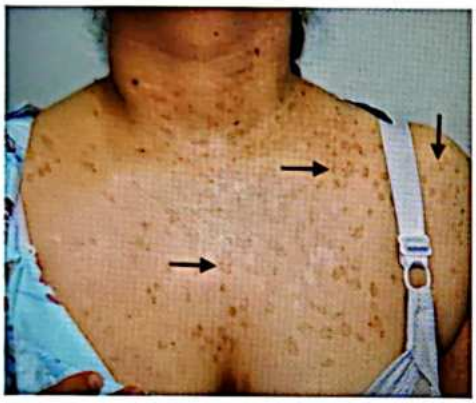
Plantar Warts



1. Superficial - HPV 2 - **mosaic pattern**
2. Deep - HPV 1 - **myrmecia**

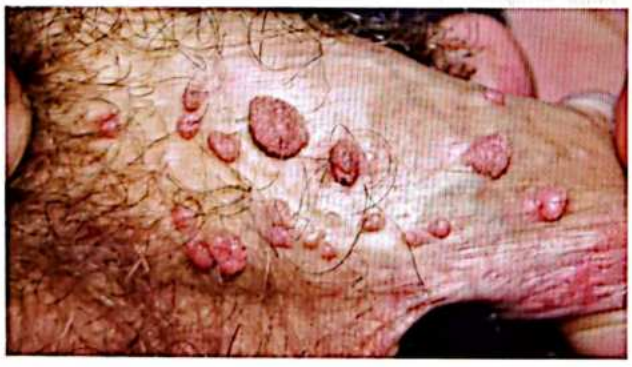
EDV - Epidermodysplasia Verruciformis

- HPV 5,8
- Genetic tendency - Autosomal recessive
- Risk of **squamous cell carcinoma**



- Presents with 3 Ps
  - **Plane warts**
  - **Pityriasis - versicolor like lesions**
  - **Reddish plaques**

Anogenital Warts

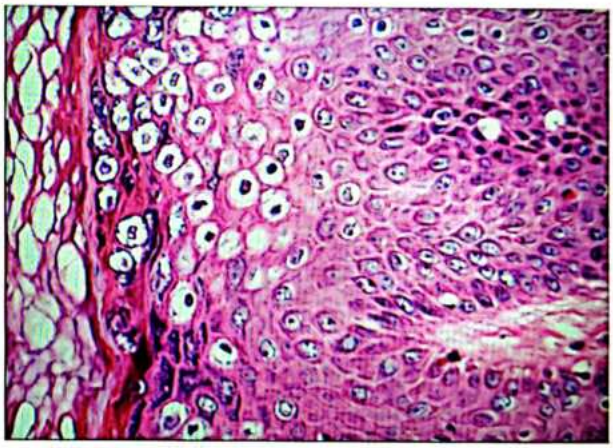


- Rule out sexually transmitted infections
- Also called **Condyloma Accuminata**
  - Accuminata - pointed
  - Condyloma - broad based
- **Males - sulcus**
- **Female - fourchette**
  - Site of sexual trauma
- More softer than normal warts
- HPV - 6, 11 - low risk
- HPV 16,18, 31, 33 - high risk
- Can predispose to malignancy

Giant Condyloma Accuminata

- Buschke Lowenstein Tumor
- Increased risk of malignant transformation

- **Histopathology – HPV**



  - **Clear looking cells with nuclear condensation in center and perinuclear halo**
  - Koilocytes
  - Transformation of keratinocytes

Treatment

- No oral treatment for HPV
  - Levamisole
  - Zinc
  - Can be given for immunomodulation
- Major therapy - destroying the virus

Chemical methods	Physical methods

• TCA
• 5-FU - 5-fluorouracil
• Imiquimod - TLR7 agonist
  ○ Toll like receptor
• Podophyllotoxin - podophyllin resin
  ○ Plant derived
• Cantharidine
• Salicylic acid
	• Electrocautery
	• Radiofrequency
	• Cryotherapy
	• Surgical excision

- Chance of recurrence
- Treatment of choice in case of
  - Genital lesions - podophyllin, imiquimod, TCA
  - Pregnancy - cryotherapy, TCA
  - Intraurethral, rectal – TCA

HHV - Human Herpes Virus

00:28:02

HHV 1 and 2 - Herpes Simplex Virus

HSV 1 and 2

- **HSV 1 - above waist infections**
- **HSV 2 - below waist**
  - Genital lesions
- The site of infection is however not exclusive

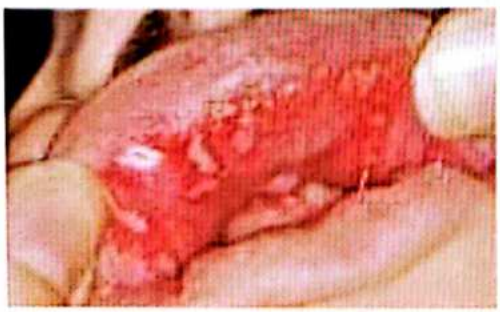
HSV Cause Three Kinds of Infections

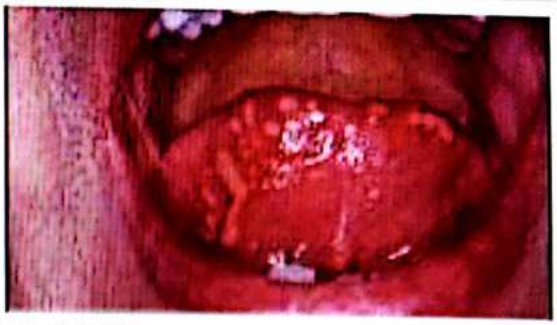
- Primary infections
- Secondary / recurrent
- Subclinical shedding
- HSV get latent in the dorsal root ganglion

Primary infection
↓
May not represent clinically
↓
Stays in the body
↓
Recurrent infections

- Even when they are clinically normal / asymptomatic, they continue to shed the virus
- Primary - more severe
- Secondary – milder

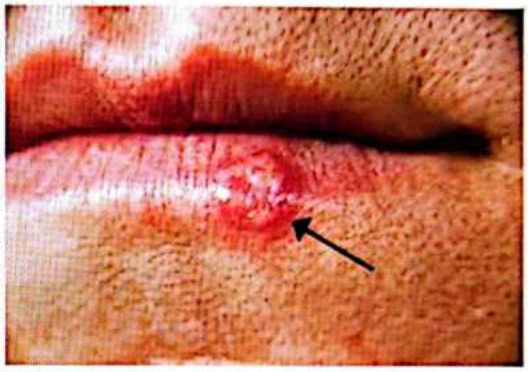
Oral Mucosa - Herpetic Gingivostomatitis





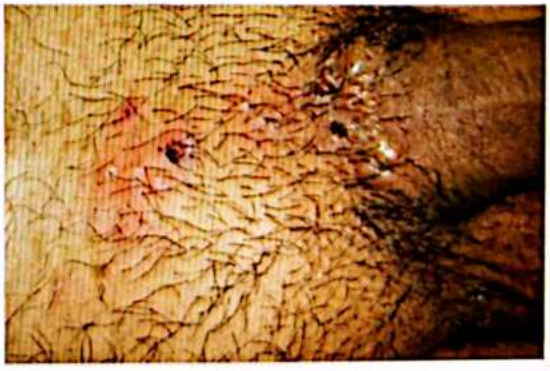
- Most common cause of gingivostomatitis in children
- Coalescing vesicles which rupture to form polycyclic erosions
- Painful

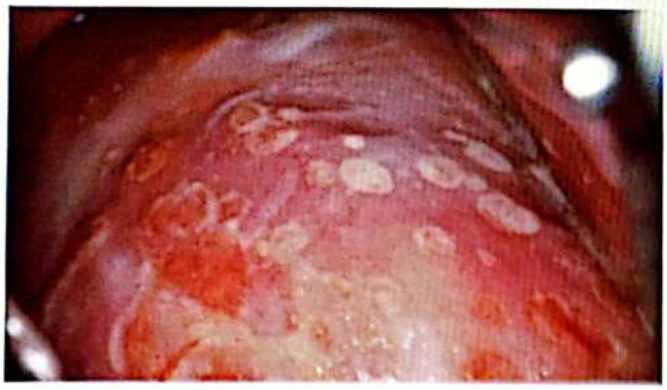
Herpes Labialis



- Cold sore
- Virus may stay latent and cause secondary infection
- Less severe
- On the **vermillion border of lips**
- Slightly painful - mild discomfort
- Predisposing factors
  - Fever
  - Stress
  - Menstrual cycle
  - Surgery
- Latent in Trigeminal ganglion

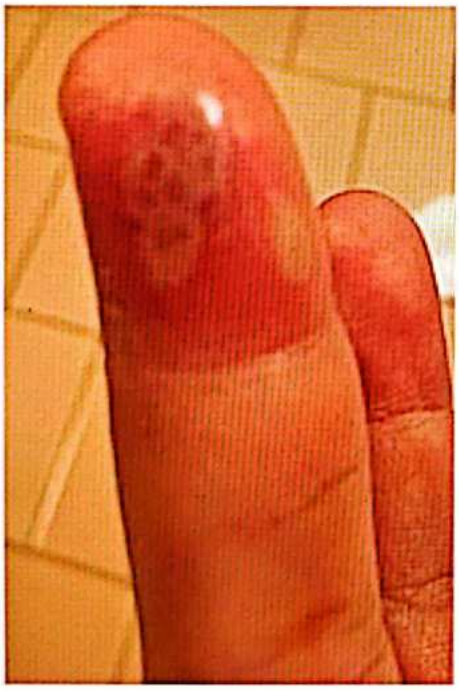
Genital Herpes





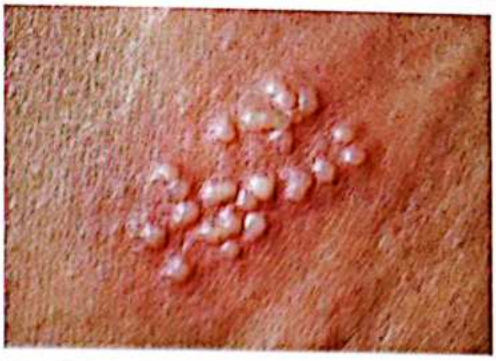
- Herpes genitalis
- HSV 2
- STI
- GUD - genital ulcer disease
  - Primary
  - Recurrent
- Subclinical shedding
- Painful coalescing vesicles → rupture to form polycyclic erosions
- Associated painful lymphadenopathy
- 7 days to heal - primary
- 3-5 days to heal - secondary
- Incubation period 2-7 days
- Recurrent herpes genitalis
  - **>6 episodes / year**

Herpetic Whitlow



- Fingertip
- Vesicles → polycyclic erosions

Herpetic Gladiatorum

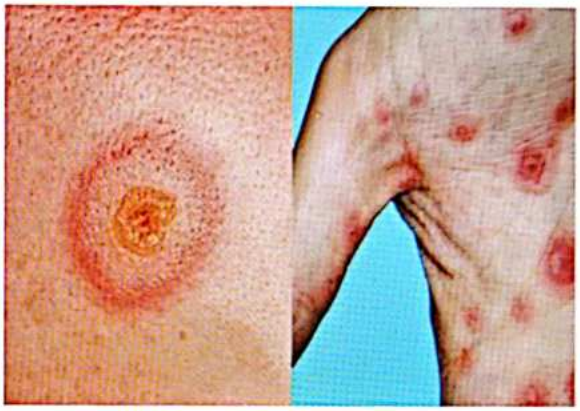


- Trunk of boxers / wrestlers

Complications of HSV

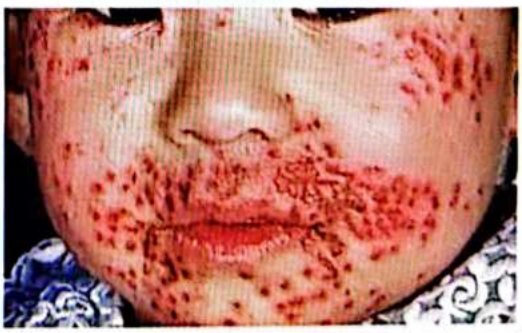
- Bells' Palsy
- Hepatitis, encephalitis
- Erythema Multiforme
- Eczema Herpeticum

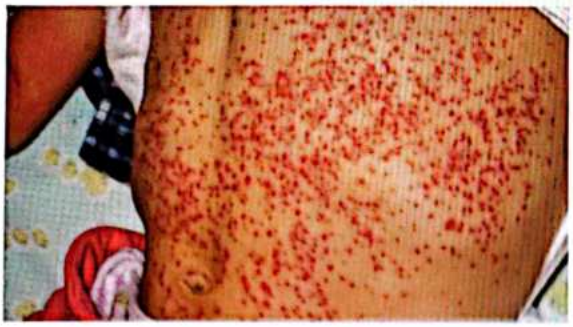
Erythema Multiforme - EM



- Reaction pattern - to drugs or HSV or other causes
- **Most common infectious cause of EM - HSV**
- Target lesion - Palms, soles / trunk
  - Multiple zones
  - Central area of necrosis / vesiculation
  - Central zone of edema / pallor
  - Outer zone of erythema
- History of HSV → target lesion → EM

Eczema Herpeticum / Kaposi Varicelliform Eruption

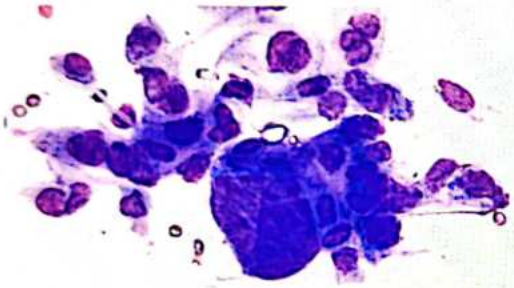




- Usually in children
- Preexisting dermatosis-mostly **Atopic Dermatitis / Darier's / Pemphigus**
- On compromised skin barrier→ HSV → numerous vesicles suddenly on preexisting dermatosis
- Treatment
  - If oral steroids are given → flare up -As this is HSV infection

  So, treatment – **Acyclovir**

Diagnosis of HSV



- **Tzank smear for:**
  - Pemphigus group if infections
  - HSV
  - Smear from base of vesicle
  - Acantholytic cells -seen in pemphigus and HSV
  - Multinucleated giant cell specific for HSV
- **Serology**
  - IgG - past infection
  - IgM - recent infection

Treatment

- TOC - Acyclovir 400mg TDS / 200 mg five times a day

Antiviral	Primary 7 - 10 days	Recurrence 5 days	Suppressive 6 month s- 1 yr
Acyclovir	**200 mg 5 times /day**	**400 mg TID**	400 mg BD
Valacyclovir	1 gm BD	500 mg BD	500 - 1000 mg BD
Famciclovir	250 mg TID	125 mg BD	250 mg BD

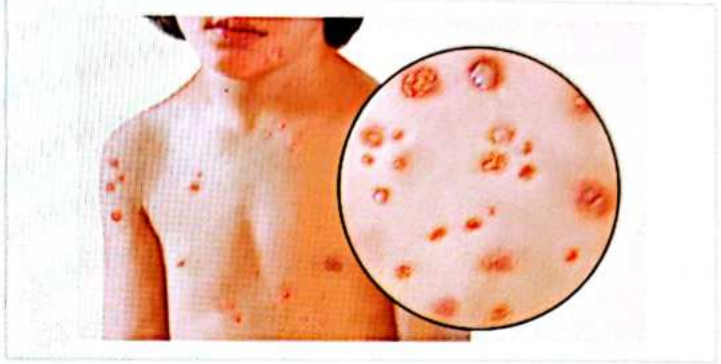
- Acyclovir resistant cases - FOSCARNET

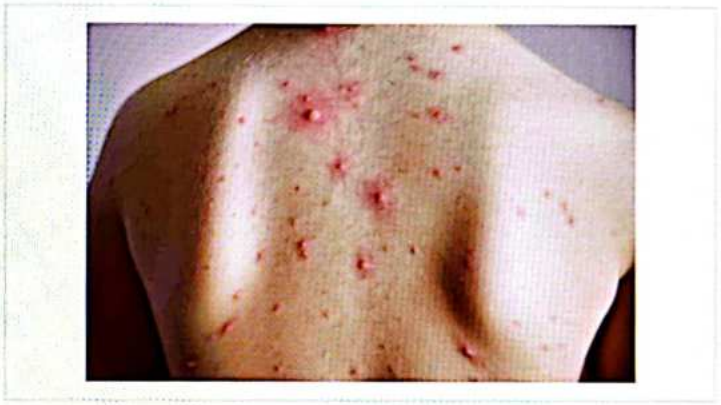
HHV 3 - Varicella Zoster Virus - VZV

- Varicella / chickenpox
- Herpes zoster

Chickenpox

- Primary infection
- Virus stays latent in dorsal root ganglion → Herpes Zoster
- Incubation - 2-3 weeks
- Usually in children
- Prodrome
  - Fever
  - Malaise
- Constitutional symptoms

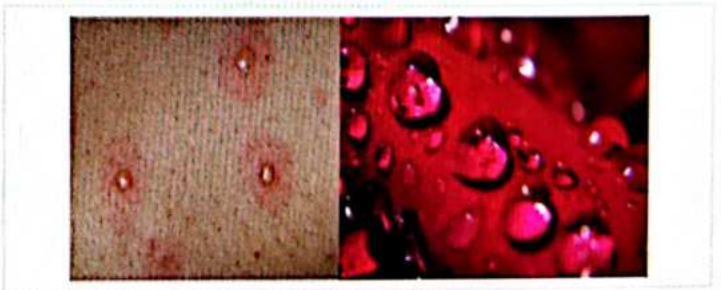




Rash - characteristics

- Polymorphic
  - Multiple morphology of lesion at the same time
  - Vesicles
  - Papules
  - Macules
  - Crusted erosions
- Morbilliform - red
- Centripetal - start from trunk spread peripherally

Vesicles on an erythematous background - Dewdrop on a Rose Petal appearance



Symptoms
- Itching / discomfort
- Usually, painless
- High transmission rate

Complications
- Encephalitis
- Meningitis

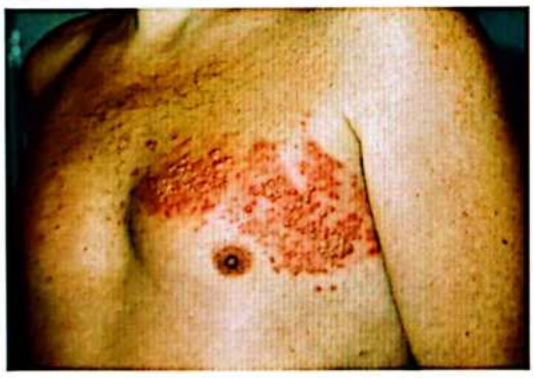
Pre exposure prophylaxis
- Vaccine
- **Oka strain**
- 2 doses - 3 months apart

Post exposure prophylaxis
- Acyclovir
- IV Immunoglobulins

Treatment
- Supportive
- Topical antibiotics
- Acyclovir only given in
  - Severe cases
  - In immunocompromised

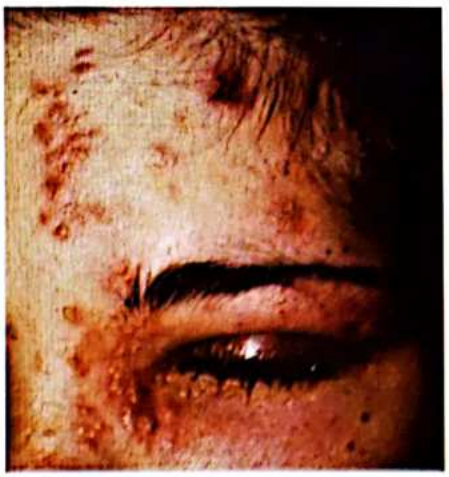
Herpes Zoster
- Varicella virus latent and get reactivated
- Latent in dorsal root ganglion
- Seen in
  - Elderly
  - Immunocompromised
  - Patients on Chemotherapy

Herpes Zoster - Dermatomal Distribution

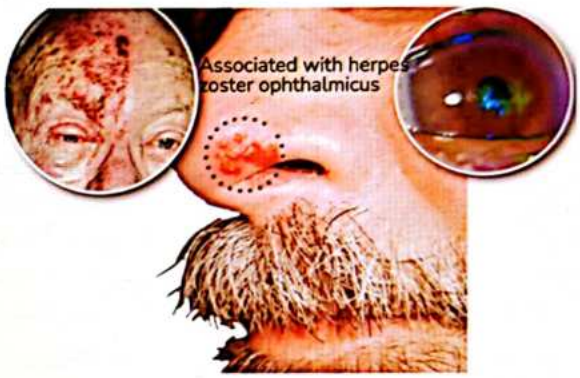


- Present along one dermatome - one side of body
- Very Painful
- Nerve trunk involvement
- Paraesthesia / altered sensation
- **Most common dermatome-thorax > cervical > trigeminal > lumbosacral**

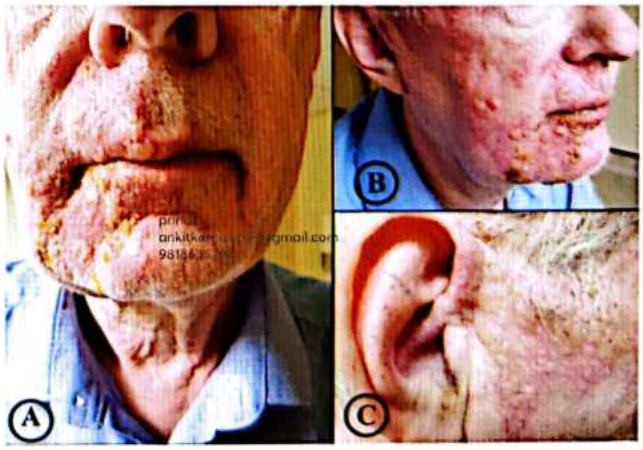
Complications of Herpes Zoster



- **Herpes zoster ophthalmicus**
  - Conjunctivitis
  - Uveitis
  - Indication for oral corticosteroids



  - Hutchinson's Sign
    → Vesicles on tip or side of nose - there is a high chance of ophthalmic involvement
    → Tip of the nose-Supplied by **nasociliary nerve** - a branch of the **ophthalmic division of trigeminal nerve**
- Ramsay Hunt Syndrome



- Geniculate ganglion
- **Trigeminal and facial nerve**
  → Vesicles on tympanic membrane→ ear pain, tinnitus
  → Facial palsy, vesicles on tongue

- **Post herpetic Neuralgia**
  - If pain persists for more than 3 months after herpes zoster
  - Pain / neurological symptoms
  - Extremely painful
  - **Gabapentin**
  - **Pregabalin**
  - **Tricyclic antidepressants**

Treatment of Herpes Zoster

- Acyclovir - 800 mg five times a day for 7 - 10 days
- Within 48 - 72 hours

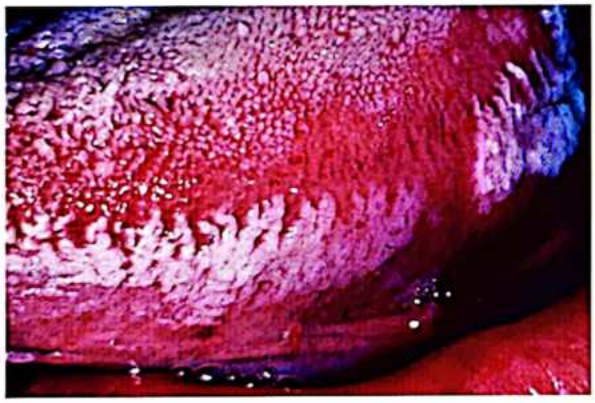
HHV - 4 - Epstein Barr Virus

01:00:16

IMN - Infectious Mononucleosis

- Glandular fever /kissing disease
- Exanthem
- Forscheimer spots
- Fever, sore throat
- If the patient is given antibiotics → amoxicillin, ampicillin → rash - **AMPI RASH**

Oral Hairy Leukoplakia



- EBV
- Lateral border of tongue
- Whitish patches - cannot be removed by scraping
- In HIV patients

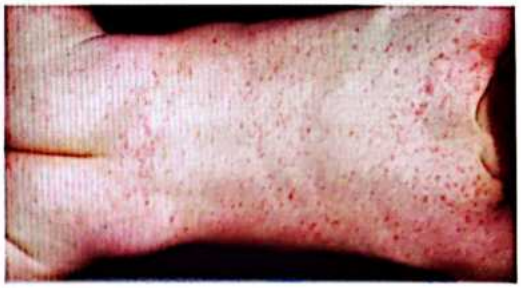
Other EBV Associated Diseases

- Burkitt's Lymphoma
- Hodgkin's Lymphoma
- Nasopharyngeal Carcinoma

HHV - 6 and 7

- PR - pityriasis rosea
- Trigger for LP - lichen planus
- Roseola infantum

Roseola Infantum

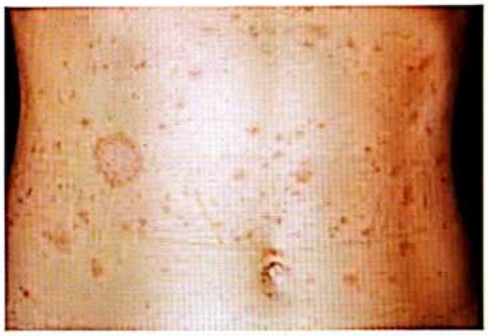


- Exanthem subitum
- Roseola - red
- In infants - < 2 years
- Exanthem - red rash
- Subitum - sudden

Fever
↓
Subsides in 3- 5 days
↓
Suddenly rash appears on trunk
↓
Rose red-coloured papules

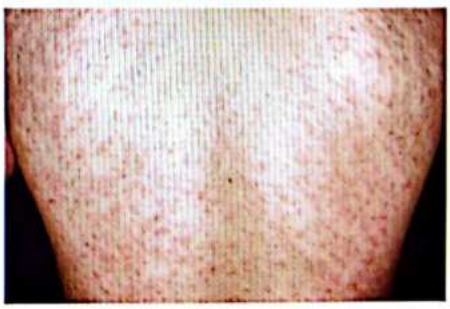
Pityriasis Rosea

- HHV - 6 and 7
- PITYRIASIS - scaly
- 2 -3 weeks post URTI / GI infection



- Lesion on trunk
- Appear Erythematous - Herald Patch
- Scale - like a collar - Collarette of Scales
- 1-2 weeks later numerous secondary lesions all over the body

Christmas Tree / Fir Tree Pattern

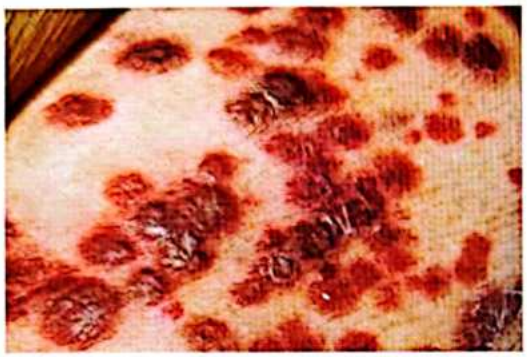


- Along lines of cleavage

Mnemonic - Pityriasis Rosea - HRCT

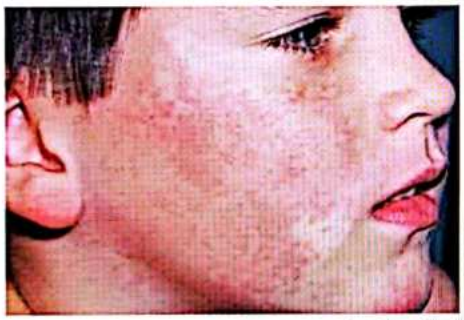
- **H**
  - HHV
  - Herald Patch
  - Hanging Curtain Sign
    → Scales are in the periphery
    → Stretch the lesion → appear like a hanging curtain
- **R** - resolute on its own
- **C**
  - Collarette of Scales
  - Christmas Tree Pattern
- **T** - Trunk

HHV - 8 Kaposi Sarcoma



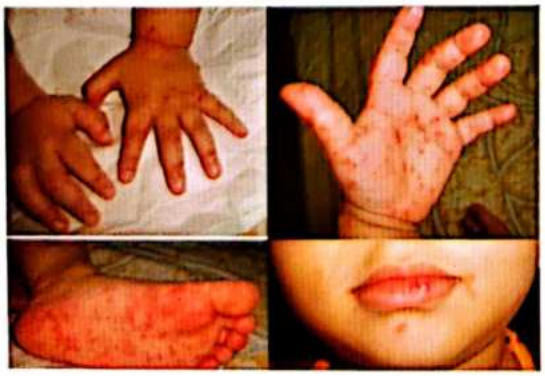
- Vascular lesion
- Seen in HIV patients

Parvovirus B19



- **Erythema Infectiosum**
- Slapped cheek appearance

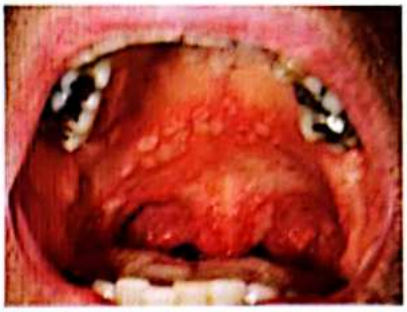
Enteroviral Disease 01:10:37



HFMD - Hand Foot and Mouth Disease

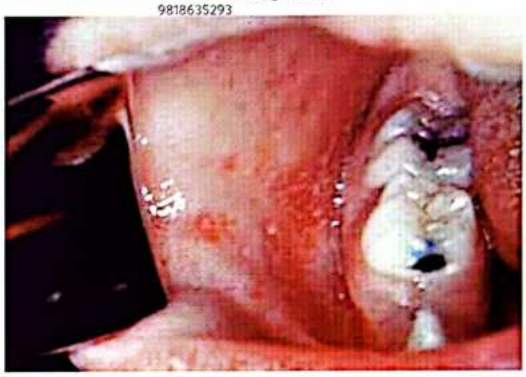
- **Coxsackie A16**
- **Enterovirus 71**
- Vesicles and papules - children
- Other constitutional symptoms

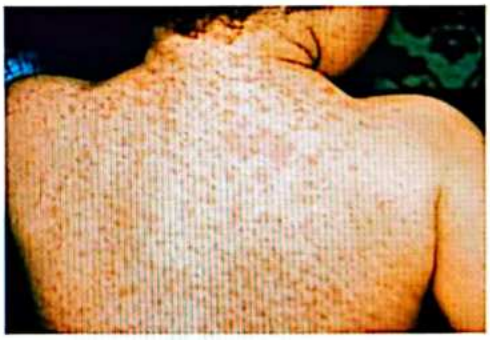
Herpangina



- Enterovirus 71
- Coalescing erosions on the **palate**

Measles

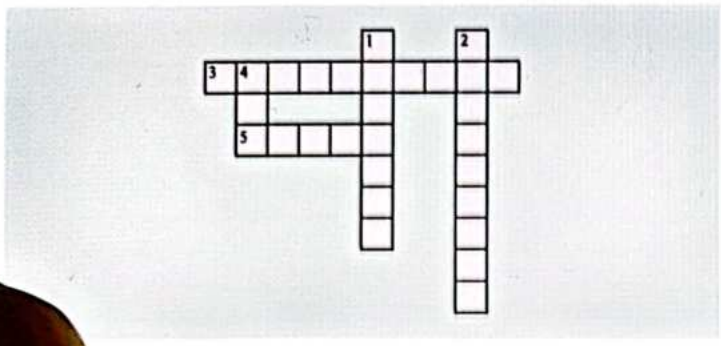




- Morbilliform rash
- Koplik's spots
- 1st disease - Measles
- 2nd disease - Scarlet Fever
- 3rd - Rubella
- 4th - SSSS, Reiter's -staphylococcal scalded skin syndrome
- 5th - Erythema Infectiosum
- 6th - Roseola Infantum
- **Mnemonic - MS Run 2 ER**

CROSS WORD PUZZLES

Crossword Puzzle



Across

3. What causes rash?
5. Measles is caused by?

Down

1. Which disease causes total-body rashes and flu.
2. Which virus is usually seen in children?
4. There are no oral treatments for?

6 PARASITIC INFESTATIONS

- Infections vs Infestations
  - Infestations are present on the surface of the skin but do not reach the bloodstream.

Scabies

- **Other Name:**
  - 7-year itch, water wash disease
- It is a parasitic infestation caused by a mite: Sarcoptes scabiei
- Variety: Humanus
- High transmission
- Transmission:
- From one family to the other,
- Through linens, through clothes and bedding.
- Found in overcrowded and low sunlight areas
- **Immunity:** Immediate immunity and delayed hypersensitivity reaction (generated)
- Water washed disease: go away on bathing and washing the clothes
- Occurs because of unhygienic conditions

Mite Image

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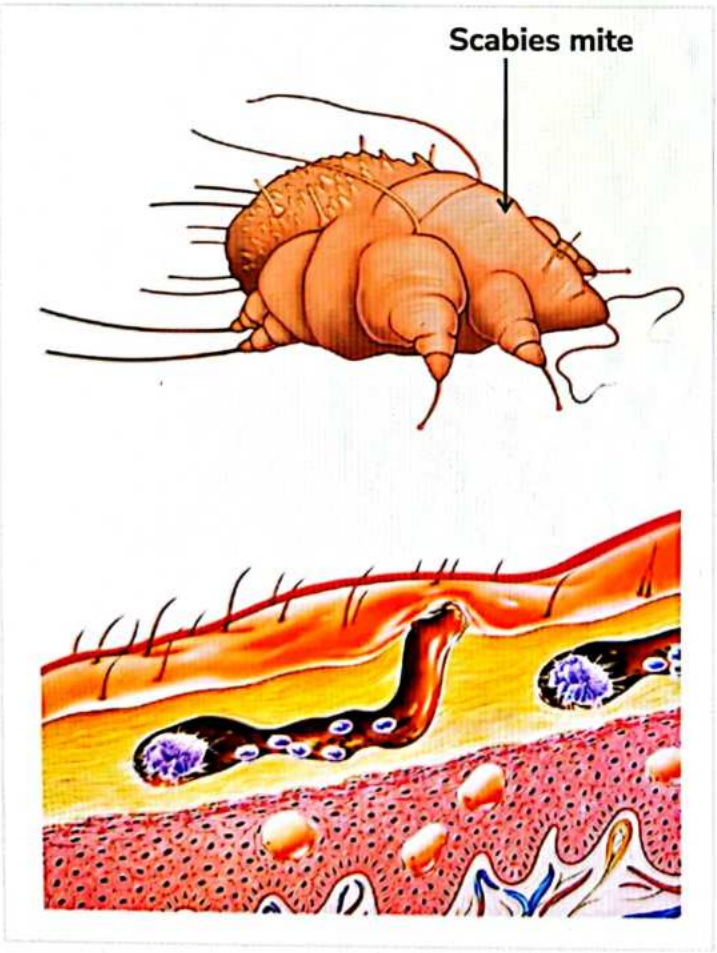


Sarcoptes scabiei

- Mite has the four pairs of legs
- It has both
  - Male mite
  - Female mite
- After copulation male mites die.
- Hence, the female mites are responsible for infestations
- Normally, in scabies patients 10-12 mites are found.

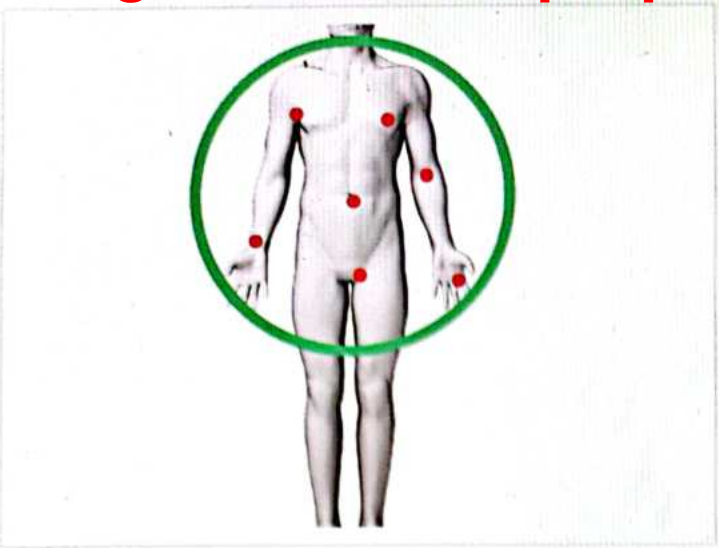
Important Information

- Female mite infests the skin and then it starts the forming a tract
- The formation of the tract starts from the stratum corneum and goes deeper
- At the end of the tract, it lays down its eggs
- The track laid down by the female mite in our skin is called the burrow
- This burrow starts from the stratum corneum and goes up to stratum Malpighi

- The lifecycle of the mite is like
  - Egg
  - Larva
  - Nymph
  - Adult

Clinical Presentations

- **Incubation period:**
  - 3-4 weeks
- If the patient is getting 2° infection
  - Incubation period: 2 weeks
- **Symptoms**
  - Nocturnal itching (increased itching at night)
  - Positive family history
  - These are used to diagnose Scabies
- **Site**
  - Interweb space (fingers)
  - Wrist
  - Popliteal fossa
  - Axilla
  - Chest
  - Umbilicus
  - Genitals
  - Mites will form an imaginary circle in the body called as Circle of Hebra



Presentation

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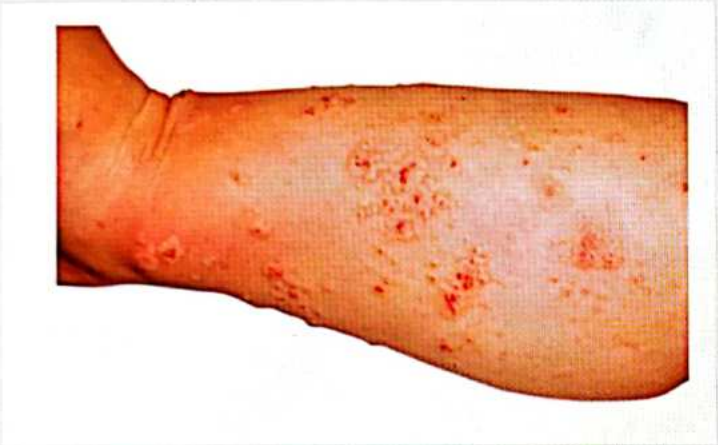
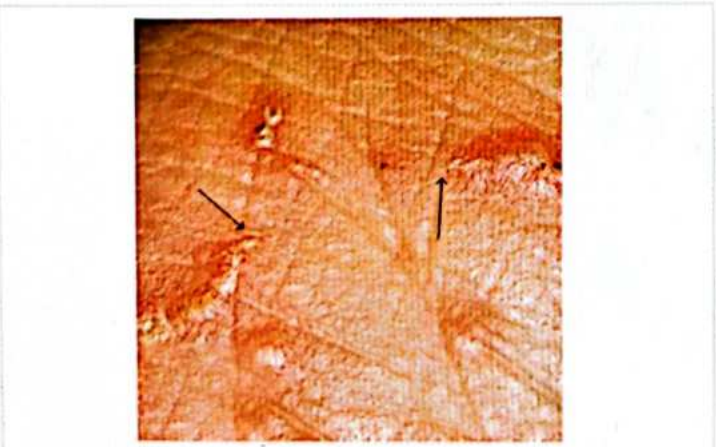


- **Scabies- papules**
  - Itchy
  - Excoriated papules
  - Vesicles (rarely)
  - Not seen because patients excoriate them
  - Present on interweb fingers with nocturnal itching
  - Positive family history

Other classical lesions are

- **Burrow**

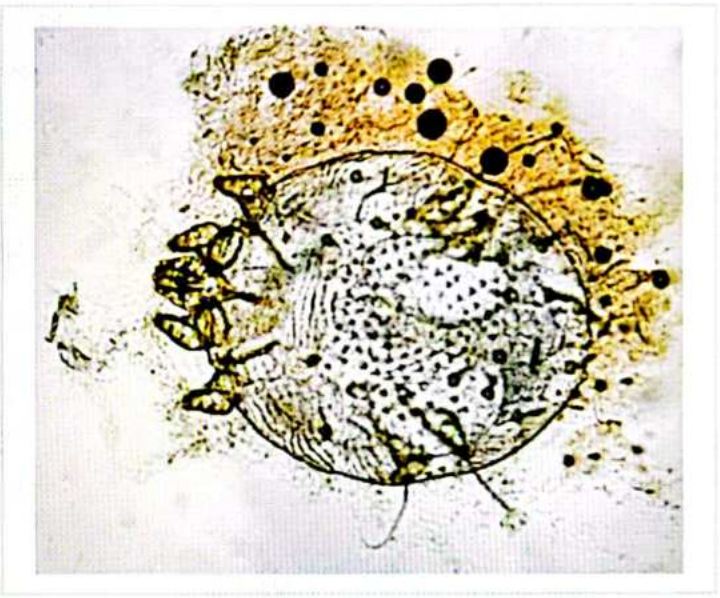


  - Burrow, it is a special lesion in scabies
  - It is tract created by the female mite
  - Clinically it appears as a linear wavy lesion
  - These are seen in
    → Interweb spaces
    → Wrist
  - Not seen commonly because we scratch the burrow
  - Scratching is a protection phenomenon through which you are destroying the path where the female lays the eggs.
  - Stratum corneum is affected but it can go up to stratum Malpighi.

Diagnosis

- Mostly, diagnosis is based on the clinical examination
- Clinical diagnosis can be done based on
  - Nocturnal itching
  - Positive family history
  - Typical sites being involved
  - Presence of the burrow

Slide preparation



- A wet mount can be made if we are not clear about the symptoms
- From the burrow, scrap the skin and mount it under the normal saline or cuvettes
- On the wet mount we will find
  - Mites
  - Sometimes eggs
  - Mite's eggs and fecal material is known as scybala.

Treatment

00:09:48

- General measures
  - Treat all family members to avoid transmission irrespective of their symptoms.

- Treat linen and clothing
- Treatment is available against the mite, not against the eggs.
- So, the patient should take the treatment after 1 week (because the eggs will eventually convert to mite)
- Hence, repeat the treatment for 1 week

Topical drugs for Scabies

- 5% Permethrin
- 1% GBHC (Gamma benzene hexa chloride)
- 25% Benzyl benzoate
- Malathion
- Crotamiton
- Precipitated sulfur
- Permethrin is the only drug of choice for all age group patients.
- Except, if the child is less than 2 months then the recommended drug of choice is Precipitated sulfur.

Drugs	Description
Permethrin	• Safest drug in all the scenarios. • **MOA:** It acts on the sodium gated channels that leads to the paralysis of the mite. • **Application:** minimum 8-12 hours repeated after a week. • Scabies does not affect the facial region (due to increased secretion of sebum). • It is recommended to apply below the neck to whole body parts (not just where you have the lesions). • After application it should not be cleaned for 8-12 hours. • Apply the cream during the night and let it stay overnight and clean it in the morning. • Repeat the same treatment after 1 week.
GBHC	• **MOA:** Acts on the chloride gated channels • Initially, it is used a lot, but it cannot be used in the certain conditions • **Contraindications** o Pregnant women o Children o Neurological disorders • It is preferred while choosing the cost-effective treatment or drugs • So, these are used in government hospitals and in other health care centers • If Permethrin is not in the option, then you can go for the GBHC
Systemic drug: Ivermectin	• Even Ivermectin is given, Permethrin will be the drug of choice • Oral drug given at the dose of 200 mcg per kg • It is used when topical drugs are not preferred. • It is also used in case of severe scabies where it needs both topical and oral agents • It is also used in o Strongyloidiasis o Onchocerciasis o Pediculosis

Types of Scabies

These are of different types:

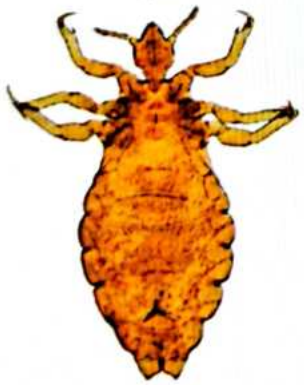
- Crusted Scabies
- Atypical scabies: It includes
  - Infantile scabies
  - Nodular scabies
  - Animal scabies
  - Genital scabies

Types of scabies	Explanation
Crusted Scabies	• **Other Name:** Norwegian Scabies (first found in Norway) • Most severe form • This occurs in the patients who cannot scratch as scratching is a protective phenomenon. • It mainly occurs in the patients with o Neurological disorders o Down syndrome • It also occurs in o Immuno-compromised patients o HIV patients o Older patients • As these patients will not scratch, the burrows will be accumulated. • Hence, heaped up crusts will be formed which are known to be crusted scabies. • In this conditions, millions of mites are present (Severe scabies) • It is responsible for institutional outbreaks • These patients admitted into hospitals • They are treated with both o Topical and o Oral drugs

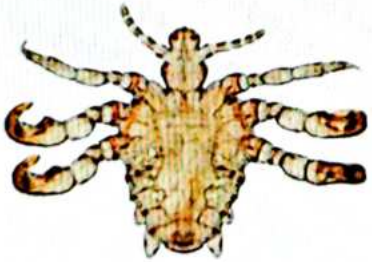
Infantile scabies	• Infants are affected with the scabies. • Regions involved o Face o Palms o Soles • Vesicles are also formed as the infant is unable to scratch. • These regions' involvement is not seen in adults. • **Treatment:** Cream is applied on the face
Nodular scabies	• Nodules are formed in the genital and axillary regions. • This results in delayed type hypersensitivity reaction.
Animal scabies	• Burrows are absent.
Genital scabies	• In this patient is presented with genital manifestations. • We should diagnose the patient for STI (both children and adults).

Pediculosis

- **Other Name:** Lice
- Caused by two organisms
- **Pediculus humanus**
  - Pediculus humanus capitis
    → These are head lice
    → Causes pediculosis capitis
  - Pediculus humanus corporis
    → These are body lice
    → Causes pediculosis corporis
- **Phthiriasis pubis**
  - It is a pubic louse
  - It causes pediculosis pubis and pubic lice



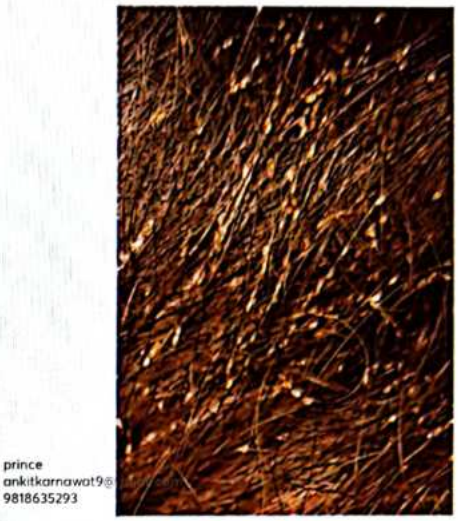
Pediculus humanus



Phthiriasis pubis

- Pediculus humanus
  - It has three pairs of legs
  - Suckers are present in the mouth
- Phthiriasis pubis
  - It is stouter and smaller
  - It has claw like legs

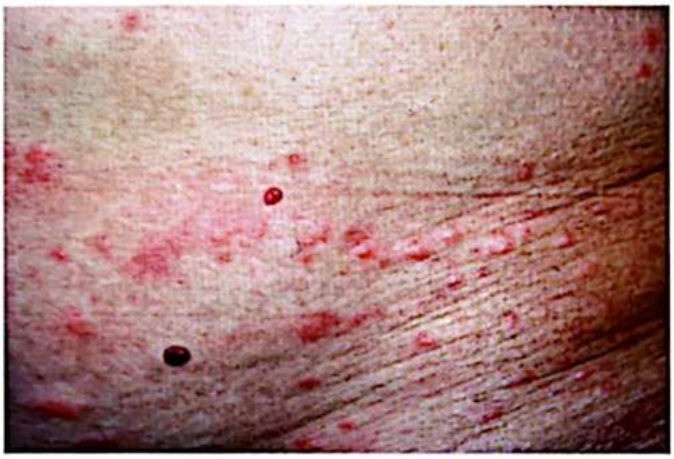
Pediculosis Capitis



- It is caused by Pediculus humanus capitis
- **Other Name:** Head lice
- It is usually present in the school going girls.
- Due to long hairs, parasitic infestations are easily transmitted.
- Presented with Itching on the scalp
- On examination, lice or nits are present.
- Nits are empty eggshells.
- Secondary infection can occur due to excessive itching
- Leading to the crusting of lesions.
- Crusting leads to the matting or coming of hair, called Plica polonica.
- Causes recurrent folliculitis in school going girls.
- Scratching causes secondary infection (recurrent folliculitis)
- Antibiotics should not be recommended for similar complaints.
- Treat the Pediculosis Capitis first.

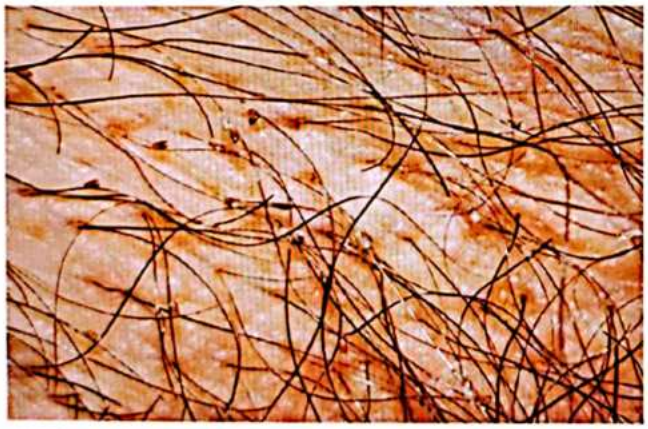
Pediculosis Corporis

- **Other Name:** Body Lice
- It is commonly found in the Destitutes
- Destitutes:
  - Those who do not have clothes, home- don't wash their Clothes
- Found in the people who does not change their clothes for longer time
- lice infest on the clothes, on seams of clothing.
- This is also referred to as Vagabond's disease.
- At night,
  - Lice attacks on the skin
  - Sucks the blood
  - Returns into the seams of clothing.
- In the body it causes the excoriated itchy papules, that are sometimes healed with pigmentation.
- It is called as Morbus Errorum (post inflammatory hyperpigmentation).



Pediculosis Pubis

- **Other Name:** Pubic lice



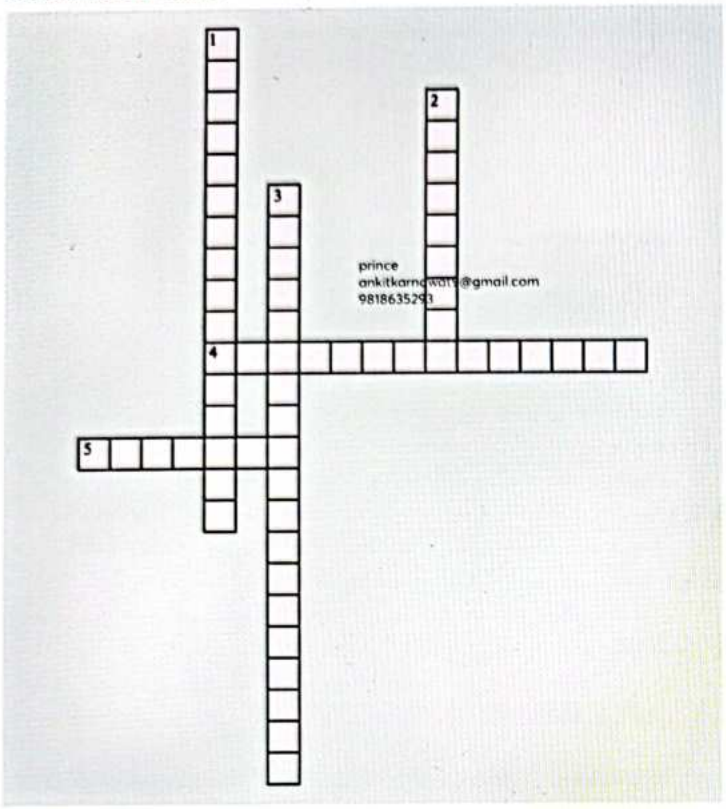
- It is caused by Phthiriasis pubis
- It is considered as a type of STI.
- If you find any pubic lice, then rule out sexual transmission.
- These are seen in
  - Pubic hair (Usually present)
  - Eyelashes
  - Axilla
- When the lice suck the blood sometimes extravasated blood is left in the skin that appears in blue color.
- It is known as Maculae Cerulae (Bluish color lesions on the skin adjoining the pubic hair).
- Bluish macules occur due to extravasation of the blood.

Treatment

- Family history of the patient is collected and treated.
- If you find any pubic lice the also observe eyelashes, axillary areas and treat them
- **Drug of choice:** 1% Permethrin (topical lotion)
- **Oral:** Ivermectin
  - **MOA:** It inhibits the Gamma butyric acid
  - Ivermectin shampoos can also be used
  - On the body you can use permethrin lotion (pubic area or hair)
- **Cotrimoxazole**
  - Used to kill symbiotic bacteria in the gut of lice.
  - Used in Pediculosis Pubis.
  - For eyelashes: petroleum jelly or Vaseline is used.
    → This will occlude the opening of the lice
    → So, that lice do not get the air
    → And finally, it dies due to suffocation.
  - You can also perform the mechanical removal of the lice by forceps.

CROSS WORD PUZZLES

Crossword Puzzle



Across

4. NorwegianScabies
5. Sarcoptesscabiei

Down

1. Infants
2. scabiei humanus.
3. BodyLice

7 CUTANEOUS TUBERCULOSIS

- Caused by Mycobacterium tuberculosis.
  - The bacteria causes:
    → TB
    → Leprosy.
- Type of extra pulmonary tuberculosis.

Classification of Cutaneous Tuberculosis 00:00:37

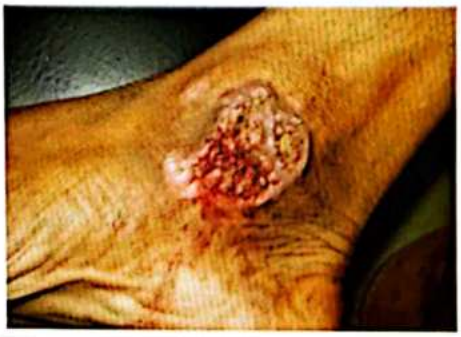
- Classified into 3 types:
  - Exogenous
  - Endogenous
  - Tuberculids

Exogenous	Endogenous
• Source of MTB is from outside the body. • First exposure to TB - new to TB infection is Naive infection. o Called TB chancre. o No immunity. • Once had TB but now reoccurred due to Exogenous causes: o Tuberculosis Verrucous cutis (TBVC). o Immunity present.	• Source is within the body. • Transmitted to skin by: o Contiguous (ex: lymphatic TB breaking into skin) - scrofuloderma o Hematogenous - Lupus vulgaris, TB gumma and miliary TB. o Auto inoculation - or orificial TB.

- Auto Inoculation
- Auto inoculation of TB occurs when MTB is present in GIT, lungs or rectum.
- When the contents are released out through mouth like vomiting and sputum:
  - Causes infection at skin around the mouth.
  - Called orificial TB.

Exogenous TB 00:05:30

1. TB Chancre / Primary Inoculation TB



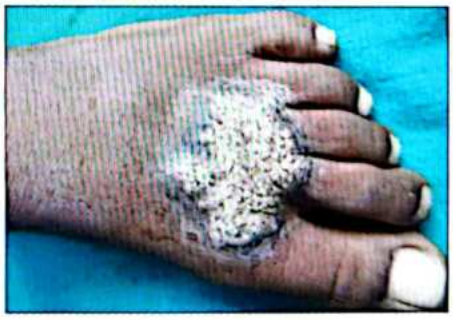
- It is a Naive infection.
- The source of infection is exogenous.
- Affected areas are extremities and face.
- First exposure to TB therefore no immunity.
- When First exposure of TB occurs ghon's focus in the pulmonary system is the first response.
- In case of cutaneous tuberculosis the ghon's focus is skin.
- Therefore, TB chancre is analogous to ghon's focus in pulmonary TB.
- Hence called primary inoculation TB.
- Patient present with an ulcer with undermined edges.
  To Remember: Any type of cutaneous TB is painless.

Important Information

Three ulcers with undermined edges in dermatology
- TB Chancre
- Chancroid
- Pyoderma gangrenosum

2. TBVC or Tuberculous Verrucousa cutis

- Occurs on skin and appears as verrucous.
- Exogenous resource.
- Body has high immunity against TB bacteria.
- Also called post primary cutaneous TB.
- Also called anatomists or pathologists wart because Verrucus occurs as warts on skin mainly in health care workers.
- Predisposing population
  - Health care workers
  - Farmers
  - Children - vermiculite occurs mainly on buttocks.



verrucous plaque

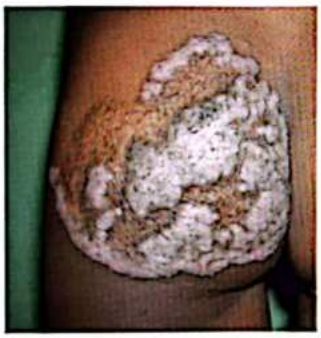
- Shows induration.
- Sometimes associated with some type of discharge or crusting.
- No pain.

Normal wart	Pathologists wart
• Multiple in number	• Single in number with large plaque
• No induration	• Induration occurs
• No discharge	• Discharge occurs

Endogenous TB

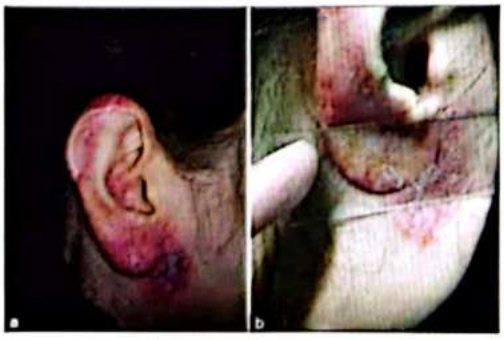
1. Lupus Vulgaris

- Most common form of cutaneous TB in adults in India.
- It is a plaque type TB.
- Endogenous type of TB.
  - Hematogenous spread (common)
  - Lymphatic spread
  - Auto inoculation
- **Most commonly seen on**
  - Head and neck
  - Arms
  - Legs
  - Buttocks
- Sometimes carcinoma may develop on top of Lupus vulgaris like:
  - Squamous cell carcinoma (SCC)
  - B cell carcinoma (BCC)



Lupus vulgaris plaque

- It is an erythematous plaque.
- Extends peripherally on one side and scarring on the other side or in the center.



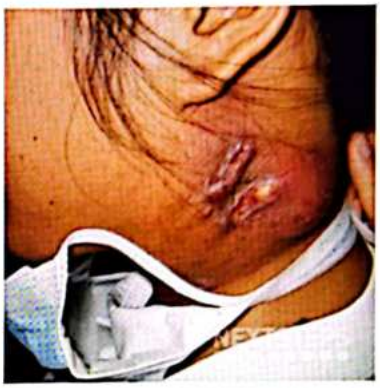
Diascopy picture

- Slide is pressed on the lesion and is examined.
- Yellowish to brownish discolouration is seen.
- All cutaneous TB are granuloma lesions and these appear yellowish brown.
- Typically called Apple jelly nodules.
- Therefore, Apple jelly nodules are the feature of Lupus vulgaris on diascopy.

Important Information

- Center scarring - Lupus vulgaris
- Center crusting - leishmaniasis
- Center clearing - Taenia

2. Scrofuloderma

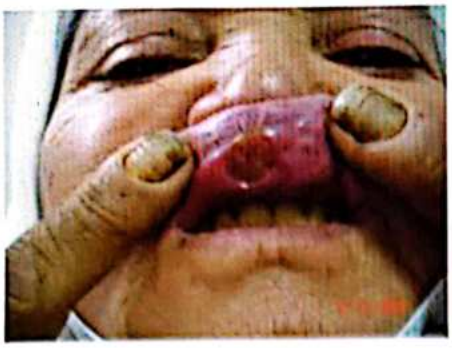


- Most common form of TB in children.
- Transmitted by contagious spread.
- **Underlying focus of TB is in:**
  - Lymph nodes
  - Bone
  - Lacrimal glands
- Most common lymph nodes included - cervical lymph nodes.
- Patient presents with:
  - Ulcerating lesions
  - Sinuses
  - Underlying lymphadenopathy.
- Painless.
- Discharge may also occur sometimes.

Lymphnode present below the skin
↓
Infected with TB
↓
The infected lymph starts invading the skin
↓
Forms ulcer on the skin
↓
Connected to the skin hence forms sinuses

3. Orificial Tuberculosis

- Pulmonary/intestinal/genitourinary immunocompromised
- Painful TB

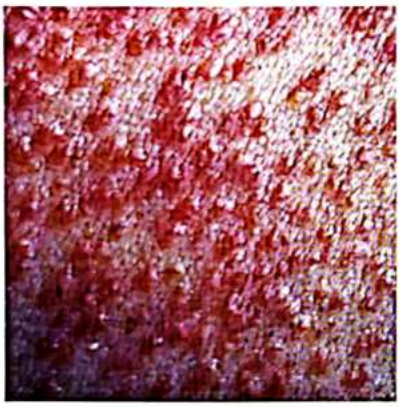


patient with orificial TB

- Infection occurs around the orifices.
- Oral - due to mucus of pulmonary TB.
- Genital - in case of TB in GIT the excretion of feces causes Genital TB.
- Only form of TB that is very painful.
- Seen in immunocompromised patients.

Acute Cutaneous Miliary TB

- Low immunity hematogenous spread



Acute cutaneous miliary TB

- It occurs due to low immunity hematogenous spread.

Tuberculids

- Type of Id reaction: Hypersensitivity reaction to the underlying microbe.
- Hypersensitivity reaction to Mycobacterium.

Mycobacterium tuberculosis infects the person - primary focus

↓

MTb or its remnants enters the blood from primary focus

↓

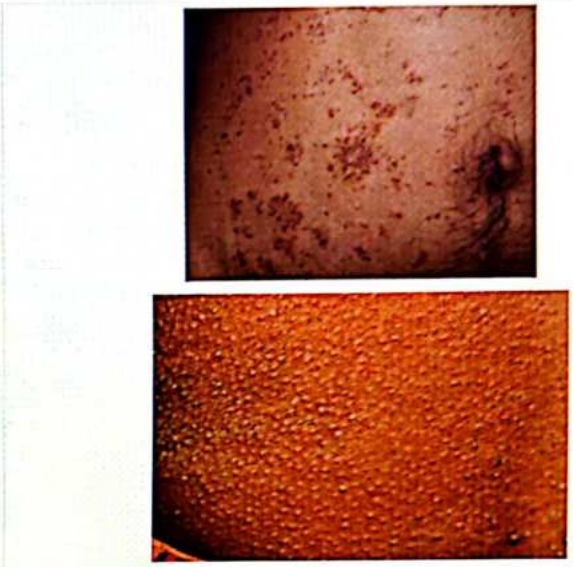
Initiates cell mediated immune response (CMI) - delayed type hypersensitivity reaction

↓

Leads to tuberculids

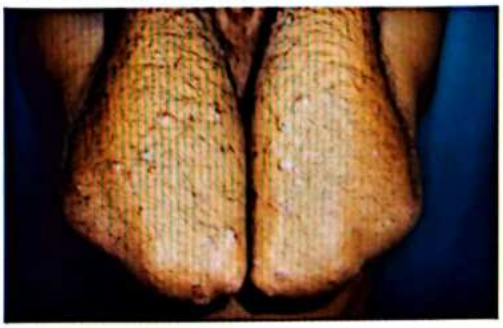
- MTb doesn't cause the tuberculids directly.
- Mantoux test is positive in tuberculids.
- Culture test and AFB test shows negative because there is no MTb, there is only a hypersensitivity lesion.
- Biopsy shows tuberculid histology.
- Responds to ATT - the primary focus is improved therefore CMI decreases.
- Tuberculids are:
  - **True:** Caused by only Mtb. Includes two types:
    - → Lichen scrofulosorum
    - → Papulonecrotic tuberculid
  - **Facultative:** Mtb is one of the causes. Includes:
    - → Erythema induratum

1. Lichen Scrofulosorum



- Contains grouped lichenoid papules on the trunk.
- Most common in children.
- Heals without scarring.
- Biopsy shows superficial dermal granuloma.
- The lesions are mainly perifollicular - present around the follicles.

2. Papulonecrotic Tuberculid

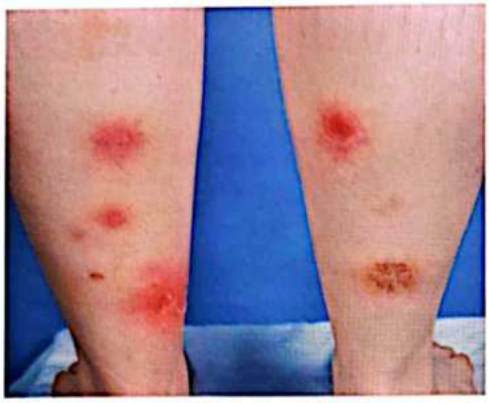


Papulonecrotic Tuberculid

- Necrotic papules appear on the extremities.

- Most commonly found in adults.
- Heals with scarring.
- Biopsy shows obliterative vasculitis (vascular inflammation) and Thrombosis.

3. Erythema Induratum of Bazin



Erythema Induratum of Bazin

- Type of nodular tuberculid.
- Most common in old aged and middle aged females.
- Occur in posterior calves.
- Nodular lesions are found on the posterior calves.
- Heals with scarring.
- Biopsy shows lobular panniculitis and vasculitis.
- The induration is deep till the fat cells.
- Only Mtb is the cause of infection.

Other Nodular Tuberculids

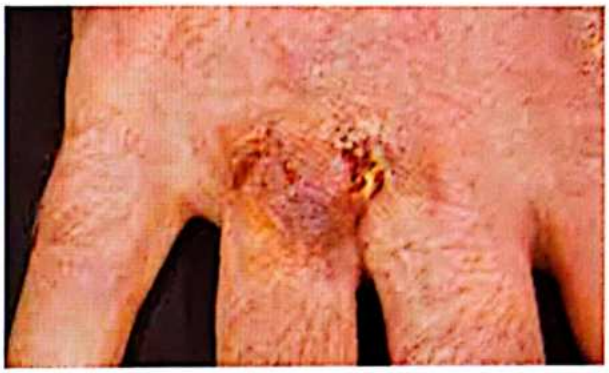
- **Erythema nodosum:**
  - Mtb is one of the causes of infection.
  - Also Caused due to other causative agents.
  - Never heals with scarring.
  - Present on the Anterior part of shin.
- **Nodular vasculitis:** Cause is other than Mtb.
- **LMDF:** Lupus Miliaris Disseminatus Faciei.
  - Earlier it was thought that it was caused by Mtb.
  - Now, it's confirmed that it isn't caused by Mtb.
  - On face - monomorphic papules are present in butterfly distribution.

Management

- All forms of cutaneous TB have the same treatment.
- Total 6 months treatment.
  - 2 months: Intensive phase
    → HERZ for 2 months.
  - 4 months: Maintenance phase
    → HRE for 4 months.
- HERZ - isoniazid, ethambutol, rifampicin and pyrazinamide.
- **Doses**
  - **Isoniazid (I):** 5mg/kg/day
  - **Rifampicin ®:** 10 mg/kg/day
  - **Ethambutol (E):** 15 mg/kg/day
  - **Streptomycin (S):** 15 mg/kg/day
  - **Pyrazinamide (P):** 25 mg/kg/day

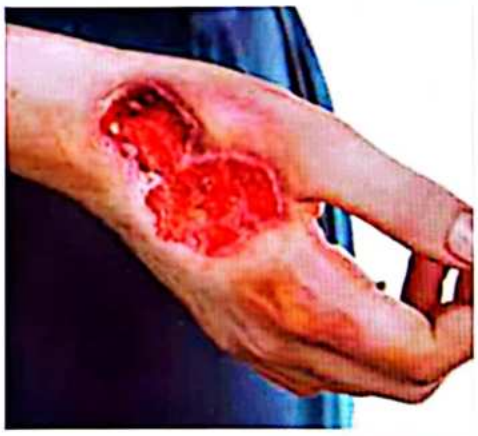
Atypical Mycobacterium Infections

00:33:33



Swimming pool granuloma

- **Swimming pool granuloma**
  - Caused by M. Marinum
  - Due to the exposure to fish tanks and contaminated water.
  - Scar forms on the skin.
  - Treated with clarithromycin + ethambutol.
- **Buruli ulcer**



  - Caused by M. Ulcerans.
  - Ulcers are formed on the skin.
  - Treated with rifampicin + streptomycin.

8 LEPROSY

- Leprosy is caused by mycobacterium leprae.

Cardinal signs of leprosy 00:00:39

- Diminution or loss of sensation in atypical skin lesions or an area supplied by one of the peripheral nerves.
- Enlargement and tenderness in a peripheral nerve.
- The finding of acid-fast bacilli in smears.

Other names for leprosy

- Hansen's disease
- Kushtha rog (Hindi name)

Important points

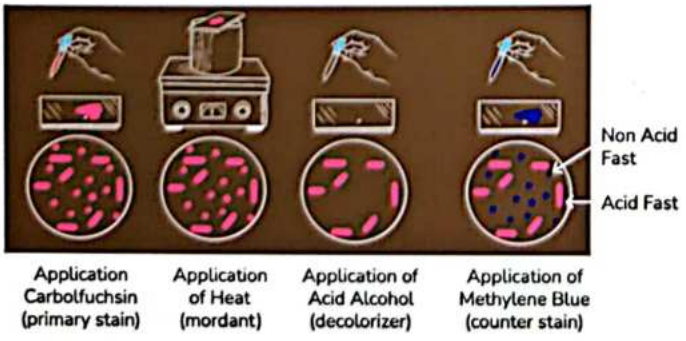
- **Route - transmits by droplet infection.**
- Incubation period - 3 to 5 years (even 10 years)
- Reservoir - Man
- Generation time - 10 to 13 days
- Culture - Non cultivable. Can grow in mouse foot pad or NBA
- New species - Mycobacterium lepromatosis.

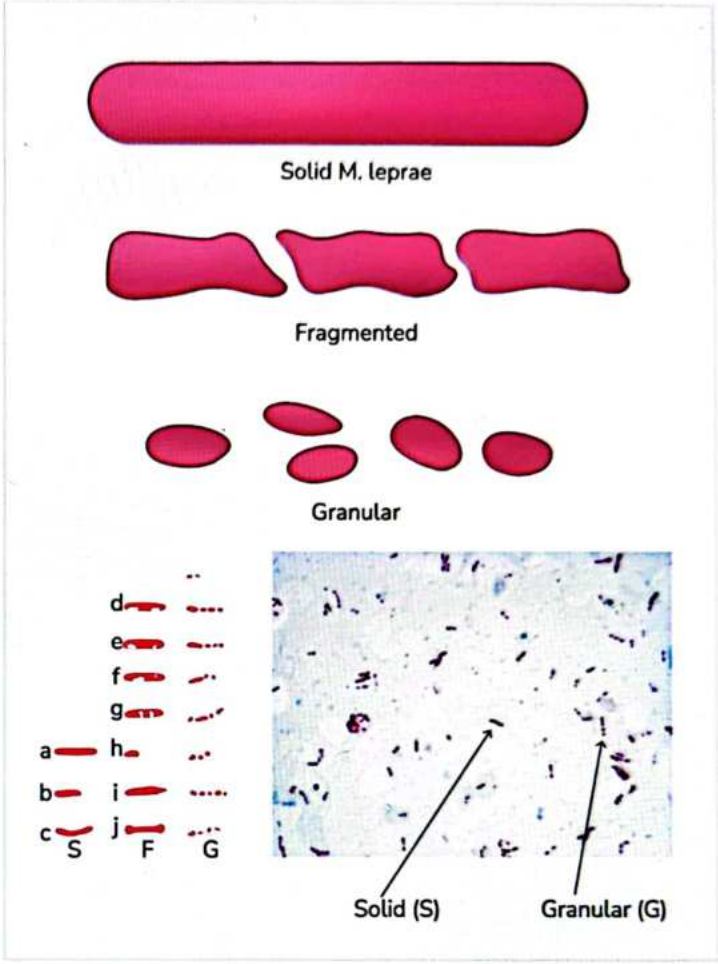
Bacteriology

- Mycobacterium leprae
- Acid-fast due to mycolic acid
- Virulent due to Phosphoglycolipids - 1 (PGL - 1)
- It looks like a solid rod with curved ends
- Tropism for Schwann cells

Slit Skin Smear 00:04:35

- **To identify, Procedure:**
  - Take a 15 mm blade and make an incision 5 mm long and 3 mm deep.
  - Rotate the blade and scrape out of the tissue. Smear the tissue and do ZN staining.
  - These bacilli being acid-fast, do not get colorized, so it appears pink on a blue background.



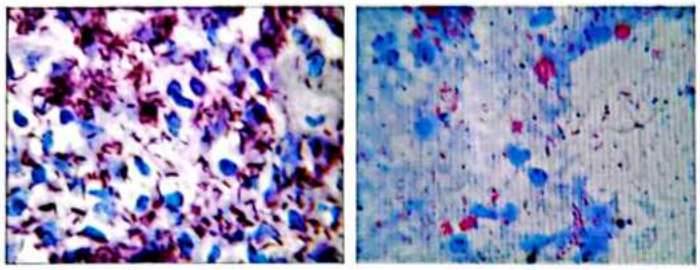


- **On Slit Skin Smear: 3 types of bacilli**
  - Solid M. leprae - It is living once it gets killed, and it remains in the tissue.
  - Fragmented - These dead bacilli are fragmented.
  - Granular - This dead bacillus can also be in granular form.
- **Identification criteria**
  - Site: Ear lobe
  - The density of bacilli in tissue: 10^4 bacilli/ gm
- **Bacteriological Index (BI):** density or concentration of bacilli, living and dead bacilli.

Refer Image 8.1

1-10 bacilli in 100 fields	1+
1-10 bacilli in 10 fields	2+
1-10 bacilli per field	3+
10-100 bacilli per field	4+
100-1000 bacilli per field	5+
>1000 bacilli per field	6+

- If BI is 1+, then bacillary concentration is very, whereas, in 6+, it is high.
- So, high BI means the patient has much more bacilli.
- Bacilli often form clumps, and it is not possible to count.
- These clusters are called globi, and if these are seen, they are 6+.

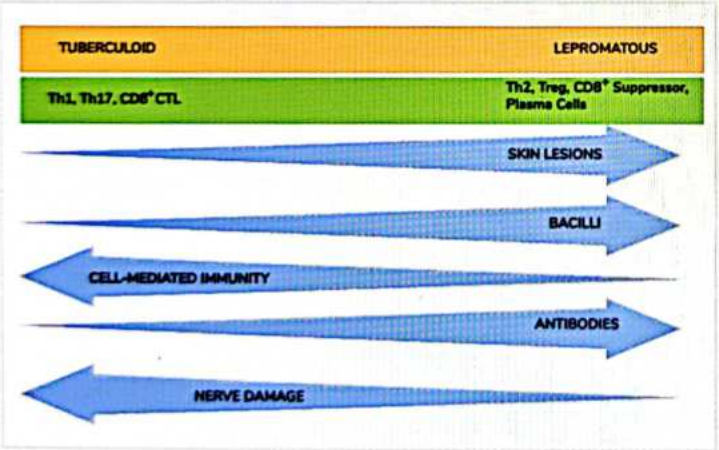


Morphological Index 00:12:14

- Percentage of solid stained bacilli, calculated after examining 200 bacilli lying singly.
- This index will help monitor response to treatment.
- It will also help with resistance to treatment.

Lepromin Test 00:13:31

- Delayed type of hypersensitivity reaction
- First described by Mitsuda in 1919
- Not for diagnosing leprosy
- Obsolete now
- Positive in tuberculoid pole
- Early reaction Fernandez
- Late reaction - Mitsuda



- This disease depends on the host immune factor, and that will decide the kind of clinical trial required.

Two poles of leprosy:

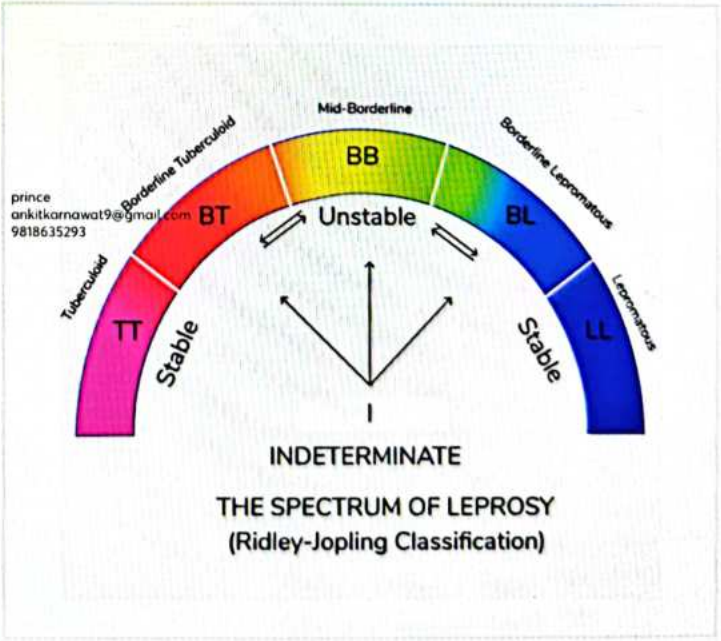
- **Tuberculoid**
  - Here immune system is good, possible to curtail the bacilli.
  - CMI or Cell-Mediated Immunity is protective against M leprae of TH1 type.
  - It has a TH1 response interferon gamma and interleukin 1.
- **Lepromatous**
  - Here the immune system is not good, It is not possible to curtail the bacilli.
  - The number of bacilli is more.
  - Skin lesions will be more.
  - It is more of a TH2 response.
  - TH2 is protective of bacteria and not against an individual.
  - There will be more antibodies.
- Nerve damage happens in both poles. But it is localized in the tuberculoid pole. In lepromatous pole, there will be multiple ones.

Contact with leprosy

- In 95% of cases, there will be no disease.
- In 5 % of cases, the body is in the indeterminate phase.
- In 70% of cases, the body will heal.
- 30% will go to a determinate form of leprosy.
- If the immune system is high, it will be TT (polar); if the immune system is low, it will be LL (polar).
- But if an immune system is in between, one can have other unstabilized leprosy called borderline leprosy.
- The most unstable phase is borderline borderline (BB).

Ridley Jopling Classification 00:35:14

- It is the most scientific classification as it considers the below-given parameters:
  - Clinical
  - Bacteriological
  - Immunological
  - Histopathology
- TT (stable) BT BB BL LL (stable)



THE SPECTRUM OF LEPROSY
(Ridley-Jopling Classification)

- 2 other types of leprosy are not included in Ridley Jopling

Classification
- Indeterminate
- Pure Neuritic Hansen

Classification

- Number
  - Lesions will be more towards Lepromatous pole
- Margins
  - Well-defined margin in tuberculoid and ill-defined in Lepromatous
- Glands
  - Dry-looking lesion in the tuberculoid and shiny lesion in the lepromatous
- Sensation
  - The sensation is localized in the tuberculoid and has widespread nerve involvement in lepromatous. The lepromin test is positive in the tuberculoid. The anesthesia is called gloves and stocking.
- Nerve involvement
- Bacterial load
  - Will increase from tuberculoid to Lepromatous.

Histopathology

00:30:18

	Tuberculoid	Lepromatous
Granuloma	Well-formed granuloma	Ill-defined granuloma
Epithelioid cells	More here	Less here
Macrophages	Less here	foamy macrophages
T-lymphocytes	More of CD4 cells	More of CD8 cells
Giant cells	More of Langerhans giant cells	Foreign body giant cells
Grenz Zone	Not present	Present here
Perineurial Lamination	Not present	Present here

Grenz Zone

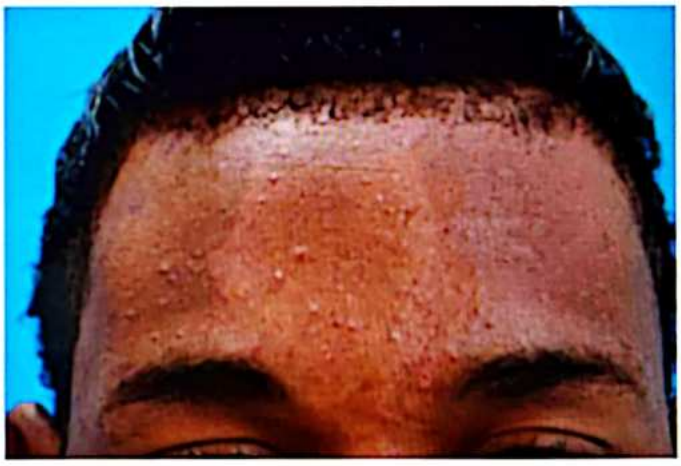
- In the tuberculoid pole
  - The granuloma is well-formed and targets the bacilli.
  - So, it's touching the epidermis.
- In the Lepromatous pole,
  - The bacilli are not controlled,
  - There are more and more non-competent cells that come out, so there is a lot of inflammation.
  - These inflammations settle down, and a Grenz zone in between the epidermis is formed
  - Onion skin appearance is seen

WHO classification

00:34:00

	MB (MULTIBACILLARY)	PB (PAUCIBACILLARY)
SKIN LESIONS	>5	1-5
NERVE INVOLVEMENT	>/=1	0
AFB	+	-

Indeterminate Leprosy



- Most Common form of leprosy in India
- Endemicity: Jharkhand, UP, Bihar
- Site: Face or extremities
- Clinical feature
  - Infiltration: ill-defined hypopigmented lesions which are single with no loss of sensation and no infiltration.
  - On HPE, peri appendageal granulomas.is seen

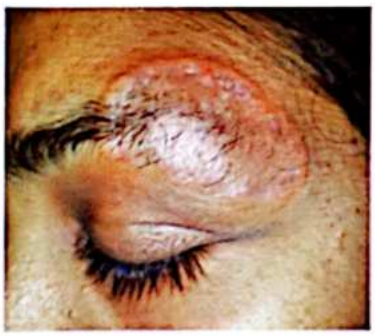
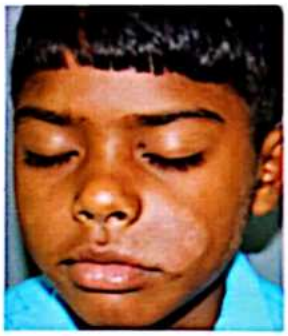
Differentiating from Pityriasis alba

- In Pityriasis Alba, multiple scaly lesions is seen.
- On histopathology, Spongiosis is seen

Determinate forms of leprosy

TT Leprosy

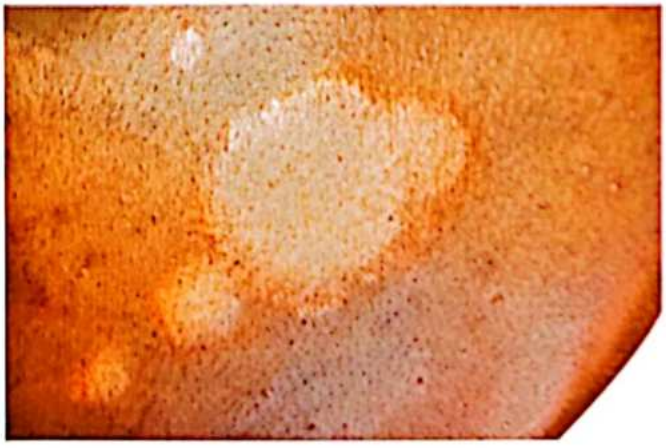
00:39:17

- Polar form
- Stable form
- High Immunity
- Only 1 to 10 lesions that are less than 10 cm in size.
- Well defined and scaly.
- Loss of hair
- 100% loss of sensation
- Dry looking lesion
- The saucer right way up is used for TT hansen - The lesion of TT is like a saucer.
- Autonomic damage
- Nerve - Nerve to patch
- Slit skin smear - Negative
- Lepromin test - Positive

BT (borderline hansen)

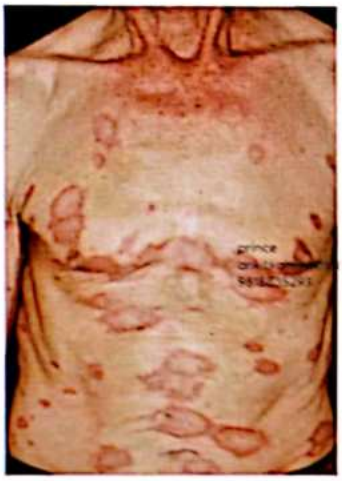
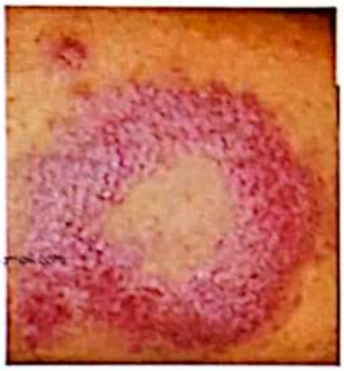
00:41:54



- Most common determinate form of Hansen
- Immunity is good but not as good as Tuberculoid leprosy
- Well to ill-defined lesion
- 10 to 20 lesions on the body of 10 to 20 cm in size.
- It is called Pseudopodia
- Smaller lesions on the periphery
- Satellite lesions suggest that it is spreading
- Marked loss of sensation but not 100%
- Nerve: will be present
- Anesthesia: will be present
- Swearing: will be lost but less than that of TT Hansen.

BL

00:44:17

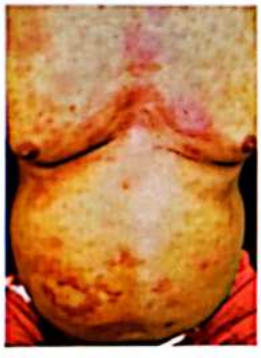
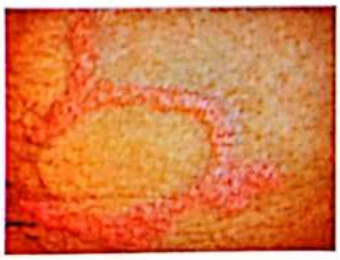



INVERTED SAUCER

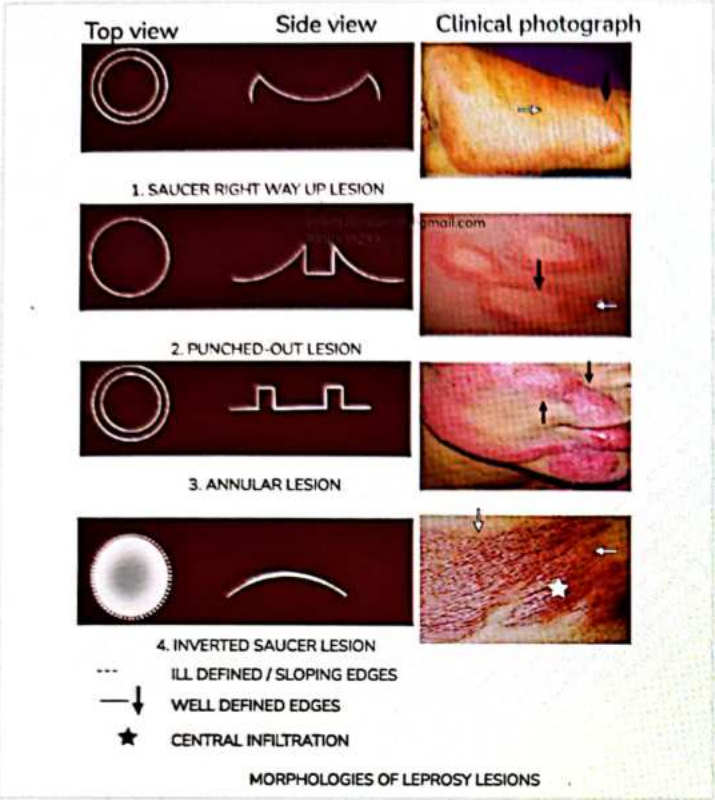
- Number:
  - This is more towards lepromatous, so numerous, small lesions with ill-defined margins which are tending to be symmetrical.
- Margin:
  - ILL-defined margins
- Tendency to symmetry:
  - Multiple lesions trying to be symmetrical
- Glove and stocking anesthesia
- Inverted saucer –
  - The bacilli are trying to invade the surrounding area, and there is a central invasion, so this is called an inverted saucer appearance.
  - It is also known as annular lesions, punched-out lesions, or dome-shaped lesions.
- An inverted saucer is more common in BL.
- Punched out is more in BB.

BB

00:46:25

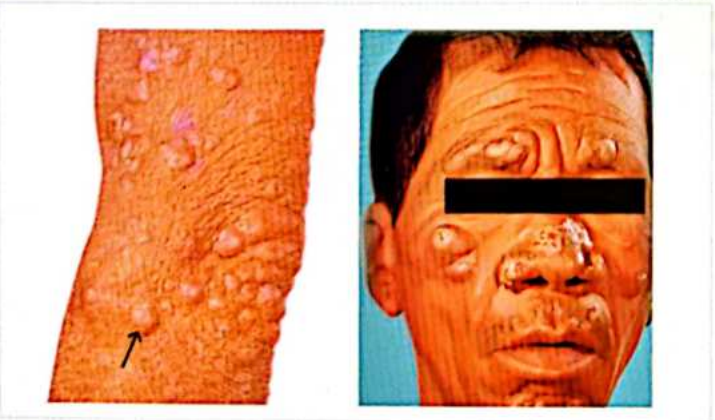
- Most unstable form. A patient does not stay in this phase for long.
- Lesions are spreading.
- Map-like or geographical lesions.
- More than 20 lesions will spread all over the body.



1. Saucer right way up lesion - TT
2. Punched out lesion - BB/BL
3. Annular lesion - BB/BL
4. Inverted saucer lesion - BL

LL

00:48:00



- The stable and immune system is low
- Numerous lesions like papules, nodules, and macules.
- Small symmetric ill-defined shiny ones
- In LL Hansen, bacilli infiltrates, so wrinkles are present. It infiltrates the skin. Follicle openings can be seen.
- Surface - shiny
- Leprous alopecia
- Anesthesia - Glove and stocking
- Slit skin smear - positive
- Lepromin test - negative

Facial deformities

00:50:00

Leonine Facies

- Madarosis - loss of eyebrows
- Sagging face
- Buddha's ears
- Rat bitten ears
- Collapsed Pinna
- Saddle nose deformities
- Looks like a lion called Leonine Facies.

Ocular Involvement trigeminal and facial line is involved

00:50:56

- Lagophthalmos - lower eyelid
- Corneal hypoesthesia
- Superficial punctate keratitis - malaria lepromata (chalk-like clusters seen with naked eye)
- Seen with slit lamp examinatio
- The most common cause of blindness in leprosy: cataract

Sanctuary sites

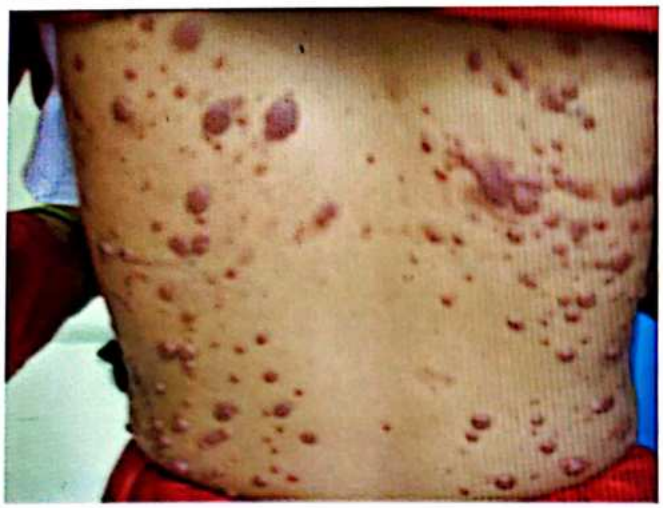
00:52:50

- Warmer area axilla, groin scalp
- CNS
- Post chamber of the eye
- Lower respiratory tract
- Female reproductive tract

Unusual presentations of leprosy

00:53:32

- **Histoid leprosy**



- Spindle-shaped cells in histopathology. nodular discrete lesions on clinically involved skin.
- This happens because of focal loss of immunity.
- AFB is longer and more slender
- Patients on Dapsone and monotherapy, which led to this.

- **LUCIO Leprosy**
  - This is called beautiful leprosy
  - Lepra Bonita
  - Diffuse infiltration with sensory loss
  - No skin lesions involvement/eye involvement

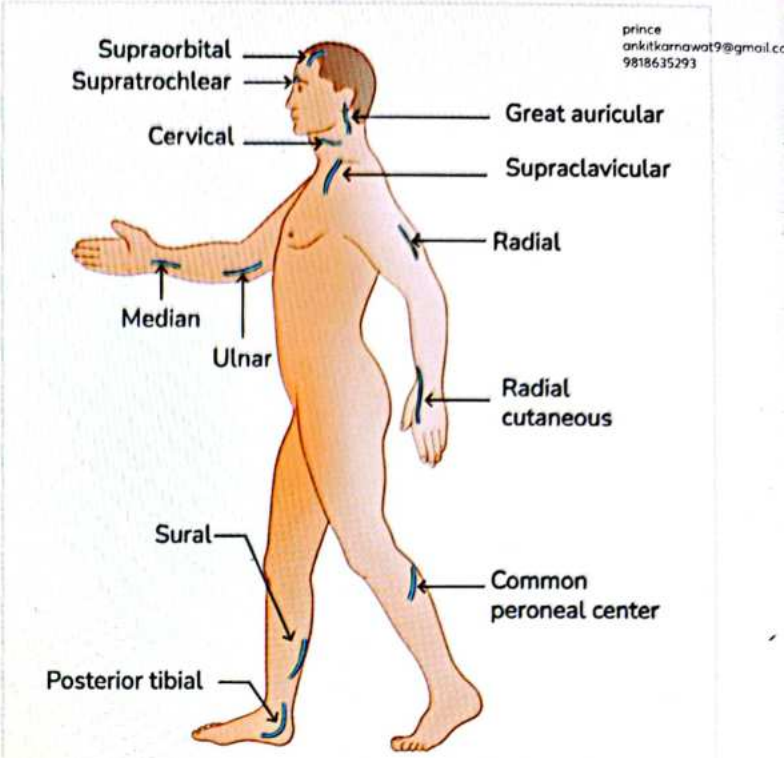
- **Lazarine Leprosy**
  - Malnourished, ulceration

Pure Neuritic Hansen

- Only nerve involvement and no skin lesions
- Endemic to India
- Neurological sensation loss
- Slit skin smear - Negative
- Absence of other causes of nerve involvement
- A nerve biopsy is diagnostic
- Take the nerves from purely sensory nerves, which are radial cutaneous and sural nerves.

Nerve Involvement in Leprosy

- Specific, paralytic, sensory
- Sensory abnormalities are the most common
- Loss temperature > fine touch > pain
- Not affected - deep touch, vibration, proprioception

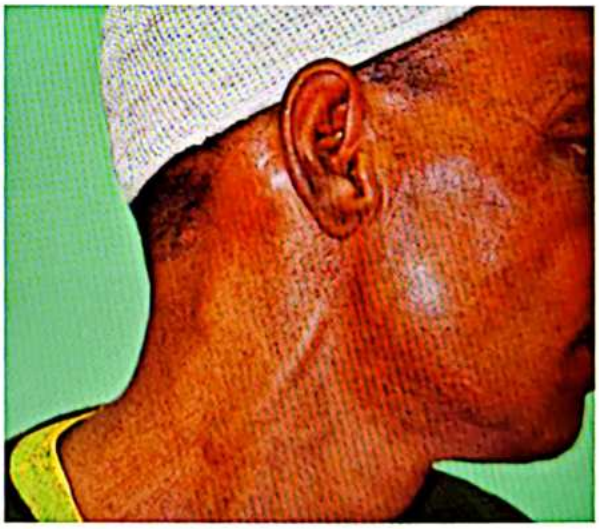


Surface marking of nerves commonly affected in leprosy

Important Information

Q. What nerve can you identify here?

- Greater curricular nerve - This nerve is not palpated but seen.



Q. Most common cranial nerve involved
Facial and trigeminal

Q. Most common peripheral nerve
Ulnar

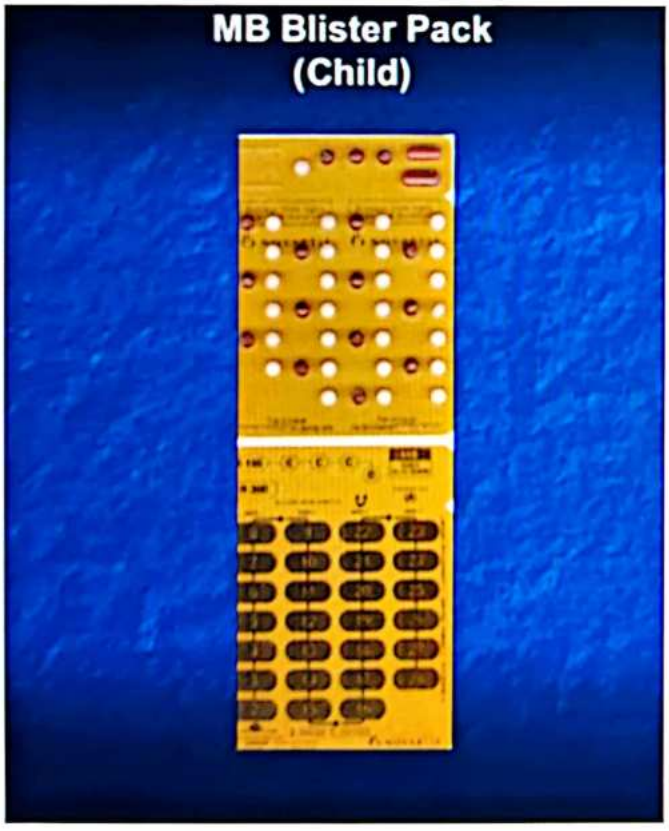
Q. Type of palsy
Ulnar (High ulnar)
Median (Low median)

Treatment of Leprosy

01:02:57

Multi-drug therapy

- Same treatment in both Lepromatous and Tuberculoid leprosy.
- This is to reduce the chances of resistance
- One dose of Rifampicin kills 99.9% of bacilli
- Clofazimine
- Dapsone
- Patients are given blister packs to ensure compliance
- Blister pack

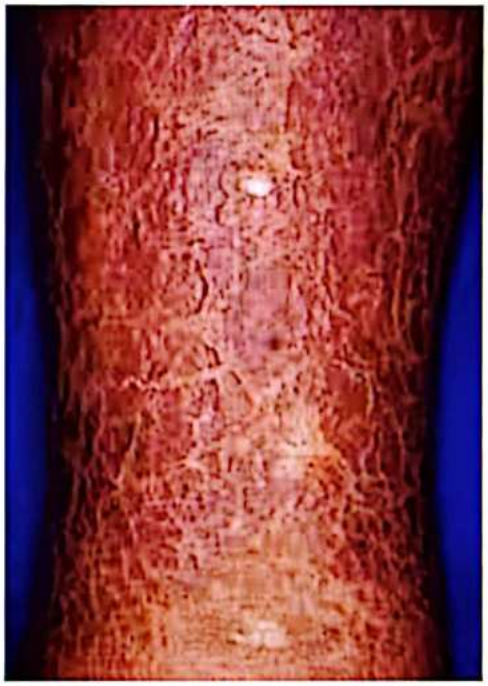


- It has medicine for one month, and everything is clearly written. This ensures compliance.
- The pink blister pack is for adults, and the yellow blister pack is for the child.

- Rifampicin - 600 mg monthly dose / 450 mg monthly for child
- Clofazimine - 300 mg monthly dose / 50 mg alternate for child
- Dapsone - 100 mg of daily dose / 50 mg alternate for child
- Medicines monthly
- Medicines on a regular basis
- Common blister packs, but for MB, it is for 12 months, and for PB, it is for 6 months.

Clofazimine

- Mechanism of Action:
  - Not very well known, acts on the outer membrane, interacts with respiratory chain and membrane phospholipid, causing cell death.
- Few isolated reports of resistance.
- Also used for type 2 reaction - 100 mg TDS
- Side Effect:
  - Reddish discoloration of the skin, ichthyosis



Newer Drugs

01:09:18

- Resistance and Compliance
  - Clarithromycin
  - Minocycline
  - Ofloxacin
  - Moxifloxacin
  - Rifabutin
- Pregnancy - Continue the same MDT
- Concomitant TB - Omit Rifampicin monthly dose. The rest remains the same.

Reactions

01:10:48

- The effect of the immune system on the patients
- Two types of reactions

- Type 1 reaction
- Type 2 reaction - Also known as ENL - Erythema Nodosum Leprosum

Type 1	Type 2
• Type 4 hypersensitivity reaction	• Type 3 hypersensitivity reaction
• Upper spectrum	• Lower end of the spectrum
• Predisposing factor: happens after initiation of MDT	• Predisposing factor: Stress, infection, pregnancy that brings down the immune system further.
• Exacerbation of existing lesions.	• New crops of lesions along with fever
• Nerve involvement: Tenderness, abscess formation	• Not seen much here.

Type 1 reaction is seen in the BT pole.

Type 1 Vs. Type 2

- Spectrum
- Type of reaction
- Onset
- Triggers
- Morphology
- Nerves
- Constitution symptoms
- Other organ involvement
- Treatment - continue MDT and give oral steroids
  - Nerve abscess:
    → Alternative drugs: AZA, cyclosporine, MTX, HCQS
  - For type 2, there are multiple crops of painful lesion which are papules or nodules associated with fever
  - They arise in the evening and settle in 24 to 48 hours with PIH
  - Management will be oral corticosteroids and MDT
  - Thalidomide is a second-line treatment for type-2.
  - It is because the patient is resistant to steroids or cannot be given medicine.

Thalidomide

- Type 2 reaction
- 100 - 400 gm
  - Start with 400 taper to 100
- Effect in 2 to 3 days
- Anti-TNF alpha
- Side effects: Sedation, constipation, sensory neuropathy

Lucio Phenomenon

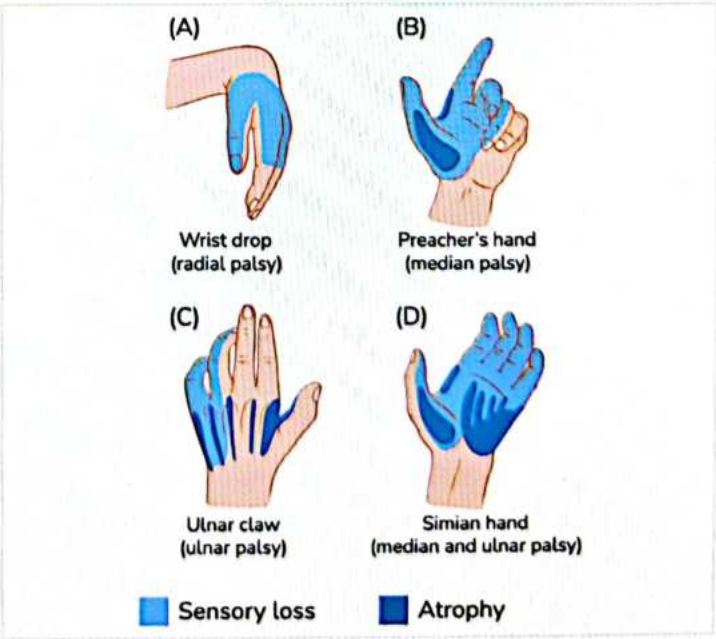
- Vasculitis and thrombus formation
- Ulceration with eschar in existing lesions
- Bacilli invade endothelium of vessels

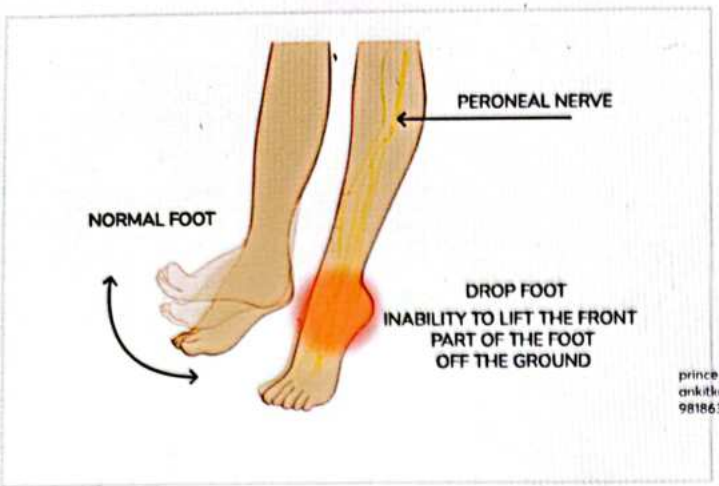
Deformities in Leprosy 01:20:17

- WHO classification and grinding
  - Hands and feet
    → Grade 0: no anesthesia, no visible deformity or damage
    → Grade 1: anesthesia present, but no visible deformity or damage
    → Grade 2: visible deformity or damage present
  - Eyes
    → Grade 0: no eye problem due to leprosy, no evidence of visual loss
    → Grade 1: eye problem due to leprosy present, but vision not severely affected as a result of these (vision: 6/60 or better, can count fingers at 6m)
    → Grade 2: severe visual impairment (vision: worse than 6/60; inability to count fingers at 6 m also includes lagophthalmos, iridocyclitis, and corneal opacities.

Motor Nerve Defects 01:21:27

- Ulnar n-partial claw hand
- Median total claw hand
- Radial wrist drop
- Common peroneal n-foot drop
- Post tibial n-claw toes/ hammer toes
- Facial n-lagophthalmos, facial palsy





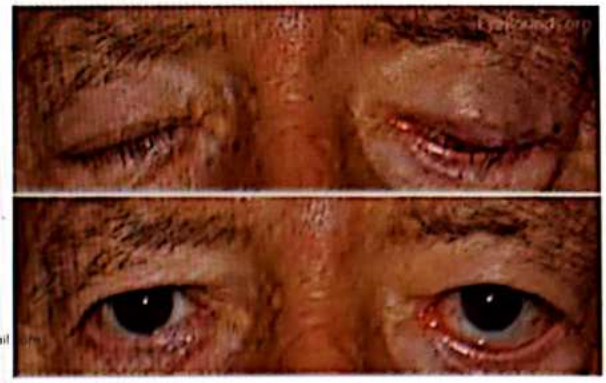
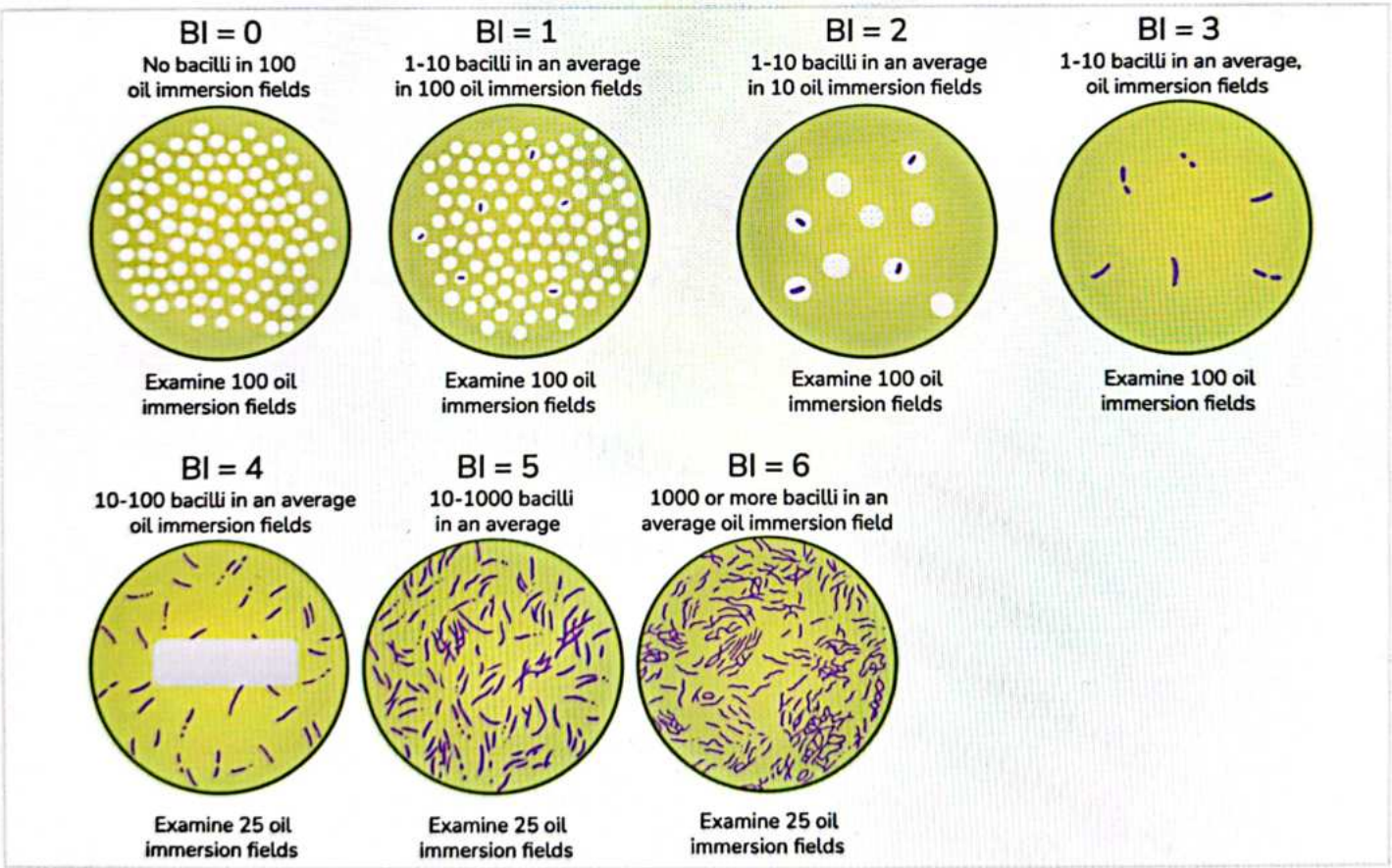
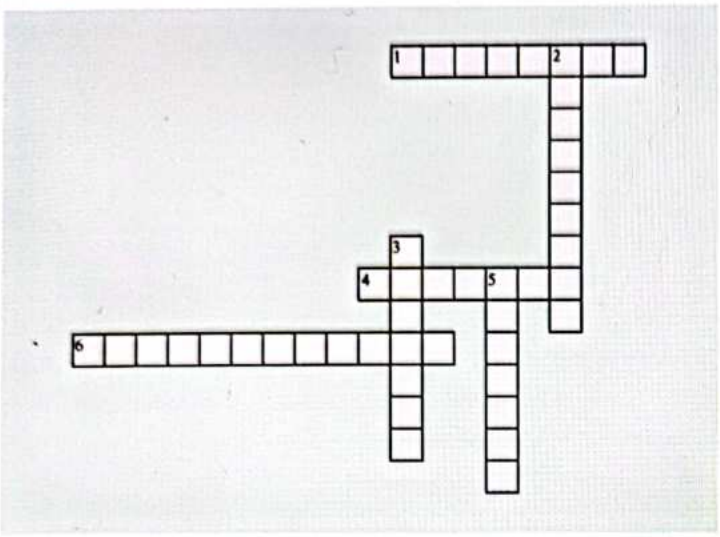


Image 8.1



CROSS WORD PUZZLES

Crossword Puzzle



Across

1. A test described by Mitsuda
4. Taxonomic class of bacteria
6. Called beautiful leprosy

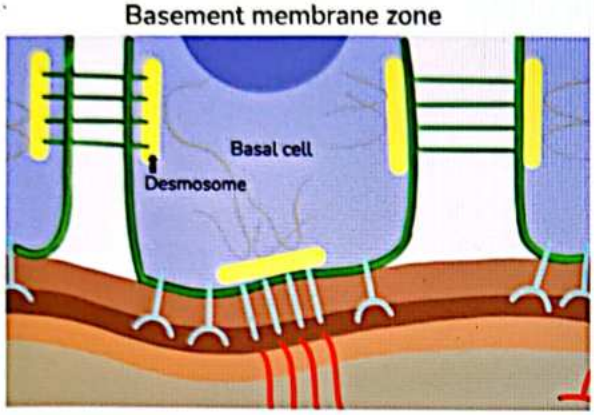
Down

2. Loss of eyebrows
3. Other name for Leprosy
5. A disease caused by mycobacterium leprae

prince
ankitkarnawat9@gmail.com
9818635293

9 IMMUNOBULLOUS DISORDERS

- It is referred to as 'Pemphigus Group of Disorders'.
- Immunological disorder.
- Both Vesicles and Bullae are formed.
- 2 Broad Categories:
  - Pemphigus group (Intraepidermal)
  - Pemphigoid group (Subepidermal)
- Ag-Ab complexes are formed - Type-III Hypersensitivity disorder.



- Basement Membrane Zone (BMZ) - Present between Dermis and Epidermis.

Pathogenesis or Mechanism 00:02:38

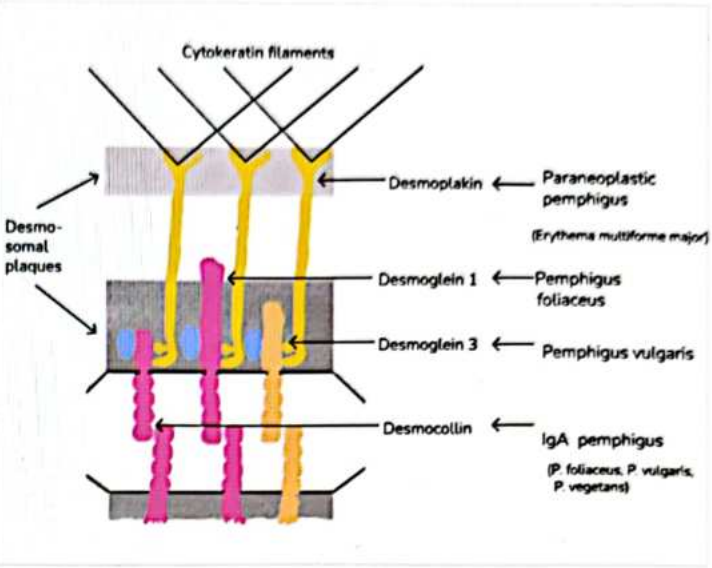
Ag-Ab complexes may form at the Keratinocytes or at Adhesions between Keratinocytes
↓
This will create an empty space between Keratinocytes
↓
The empty space will get filled with tissue fluids
↓
Leads to more separation of Keratinocytes (Acantholysis)
↓
This progresses to form a lesion filled with fluid
↓
Clinically appears as a Vesicle or Bullae

Types of Immunobullous Disorders 00:04:43

- 2 types:
  - **Intraepidermal:** Split is in the epidermis.
  - **Subepidermal:** Split is below the epidermis.

Intraepidermal Immunobullous Disorders 00:06:10

- Also called the Pemphigus group of disorders.
- Target proteins: Desmosomes (cementing substance between keratocytes)



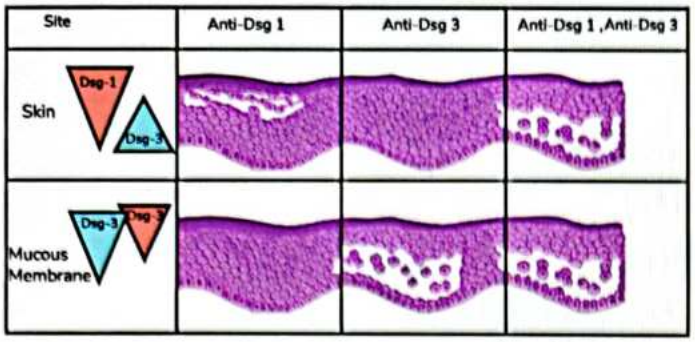
- Target Ag:
  - Desmoglein
  - Desmocollin
  - Desmoplakin
- Split:
  - Subcorneal - Beneath the corneal layer.
  - Suprabasal - Above basal layer.

Types of Intraepidermal or Pemphigus Immunobullous Disorders 00:10:00

- Depends on the type of target antigen (Ag).
- Usually 4 types:

Target Antigen	Type
Desmoglein-1 (DSG-1)	Pemphigus foliaceus
Desmoglein-3 (DSG-3)	Pemphigus vulgaris
Desmocollin	IgA type pemphigus
Desmoplakin	Paraneoplastic pemphigus

Desmoglein Compensation Theory 00:12:13



- DSG-1 and DSG-3 compensate for each other and can takeover each other's functions.
- **Skin**
  - DSG-1 is present throughout the skin (Concentration decreases from top to bottom).
  - DSG-3 is present at lower layers of skin (Max concentration is at stratum basale and spinosum).
- **Mucous Membrane**
  - DSG-1 is present only on the superficial layers.
  - DSG-3 is present throughout the membrane.
- **DSG-1 is Target**
  - Produces DSG-1 Antibodies.
  - Mucosa is not affected (DSG-3 compensates for DSG-1).
  - Skin is affected (Upper layer is affected as more DSG-1 is present).
  - Split is subcorneal
  - Seen in- Pemphigus foliaceus
- **DSG-3 is Target**
  - Produces DSG-3 Antibodies.
  - Skin is not affected (DSG-1 compensates for DSG-3).
  - Mucosa is affected (as DSG-3 is major).
  - Seen in Pemphigus vulgaris.
- **DSG-1 and DSG-3 Both are Targets**
  - Both skin and mucosa are affected.
  - Seen in Pemphigus vulgaris (DSG-3 >> DSG-1).

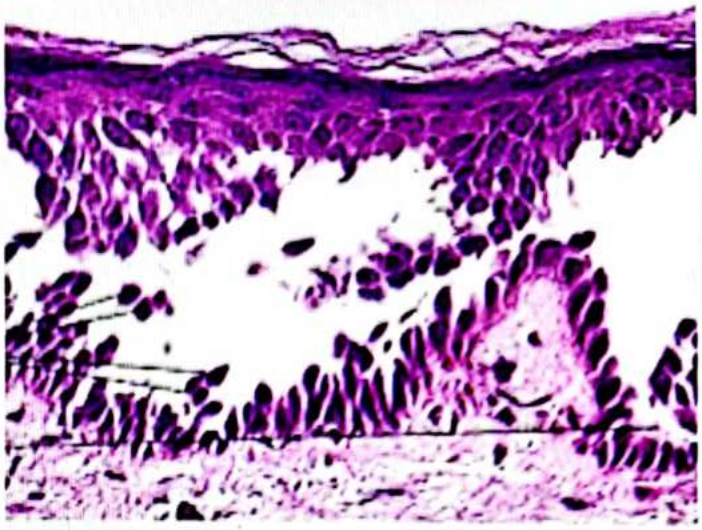
Antibodies 00:17:50

- Mostly IgG: IgG_1 and IgG_4.
- IgA and IgM are also seen.

Acantholysis 00:18:12

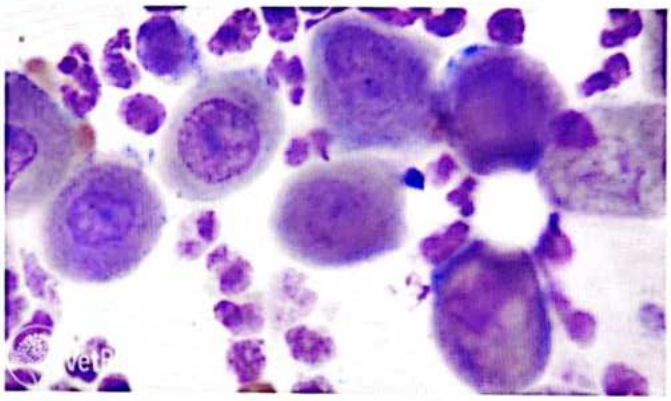
- It means seperation of keratinocytes from each other

Histopathology of Acantholysis or Pemphigus



- Findings are:
  - Acantholytic cells (separated keratinocytes)
  - Row of Tombstone Appearance (separated keratinocytes in basal layer)
  - Split

Tzanck Smear Test 00:22:43



- A Tissue smear test
- Performed to diagnose the Pemphigus disorders.

Bulla is ruptured
↓
Scraping off the base of Bulla with Scalpel
↓
Staining with Giemsa stain
↓
Acantholytic cells are seen

- Acantholytic Cell Characteristics
  - Round to oval shape, unlike keratinocytes.
  - Large nuclei (⅞ of cell surface).
  - Peripheral condensation of cytoplasm is seen.
- Tzanck smear test is also used in herpes - (Acantholytic Cells with Multinucleated Giant Cells).

Classification of Pemphigus 00:26:10

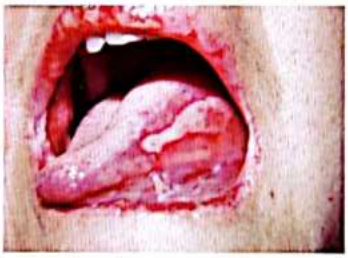
1. Pemphigus vulgaris (most common) - DSG-3 >> DSG-1.
2. Pemphigus foliaceus - DSG-1.
3. IgA pemphigus - Desmocollin
4. Drug induced pemphigus.
5. Paraneoplastic pemphigus - Desmoplakin.

Important Information

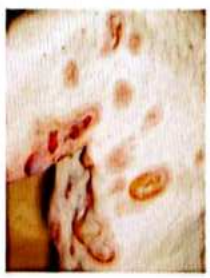
- They have an HLA association.
- Smoking helps.

Pemphigus Vulgaris 00:27:58

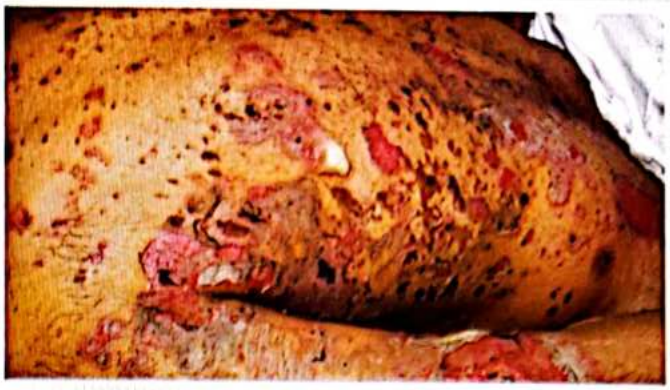
- Seen in the Middle Ages in many countries.
- **India:** Earlier ages.
- More commonly seen in the Ashkenazi Jews, Mediterranean, and Iranians.
- **Target Structure:** Desmosome.
- **Target Antigen:** DSG-1 and DSG-3.
- **Split:** Supra basal.
- **Layer:** Stratum spinosum.
- **Clinical features**
  - Involve mucosa (oral, vaginal, esophageal, rectal) and skin (overall).
  - **Mucosa:** Painful erosions with peripheral spreading tendency and yellowish sloughs.
  - Skin: Vesicle is formed which enlarges to form bulla
    → Bulla rupture results in crusted erosions
    → It heals without scarring
    → In severe cases, it involves the whole body and it heals with sudden pigmentary change.

Mucous spread

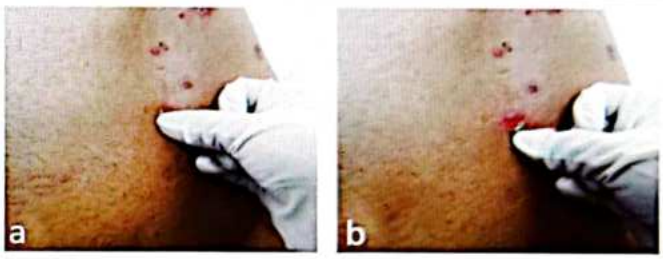
Skin spread



Case of Pemphigus Vulgaris

Bedside Tests or Signs for Pemphigus Vulgaris 00:36:13

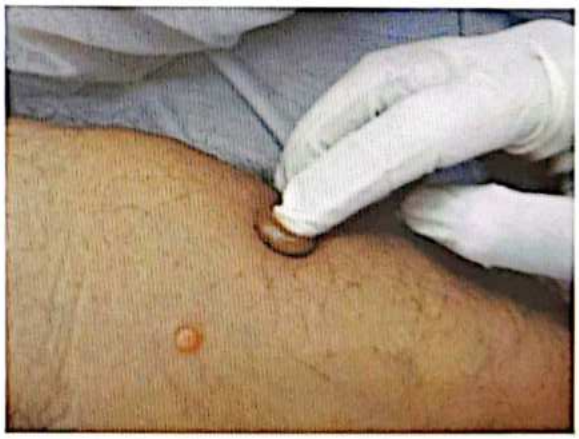
1. **Nikolsky's sign:** Development of bulla or erosion when a shearing force is applied on the skin surrounding a vesicle or bulla.



Importance of Nikolsky's Sign

- Deals with the severity and activity of the disease.
- It check response to treatment.
- Differentiate between intraepidermal positive and subepidermal disorders negitive.

2. **Bulla spread sign:** Bulla spreads over its margin when pressure is applied laterally.



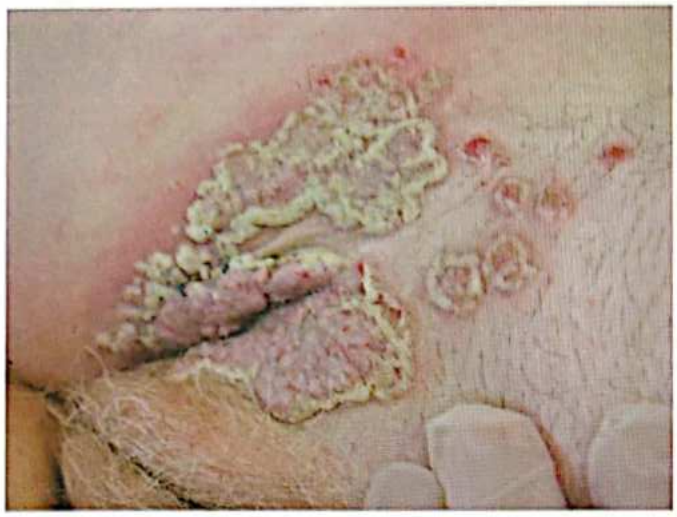
Importance of Bulla Spread Sign

- Deals with the activity of the disease.
- Differentiate between intraepidermal (angular spread) and subepidermal disorders (circular spread).

3. **Asboe Hansen sign:** The pressure is applied from the top for small bulla or vesicles.

Pemphigus Vegetans

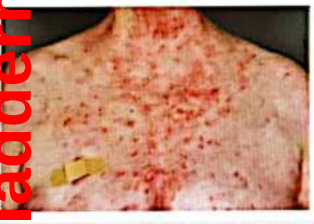
00:44:32

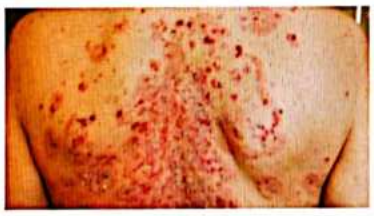


- Rare vegetative variant to Pemphigus vulgaris.
- Seen at scalp, face, and intertriginous areas.
- 2 types:
  - Neumann type (severe).
  - Hallopeau type (mild).

Pemphigus Foliaceus

- **Antigen**: Desmoglein 1
- **Split**: Subcorneal layer (Stratum Granulosum)
- No mucosal lesions (Desmoglein 3 compensates mucosa)
- **Site**: Seborrheic sites (face, scalp, upper trunk)





- **Clinical features:**
  - Vesicles and bullae are hardly present
  - Superficial vesicles and bullae rupture within a few hours which results in crusted erosions.

Important Information

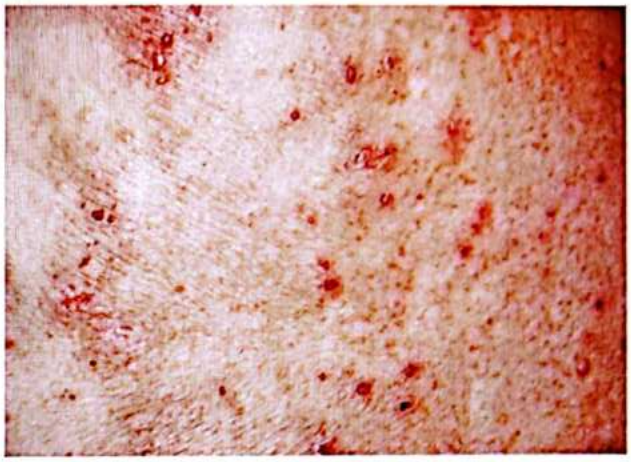
- In Pemphigus vulgaris, bullae rupture within 2-3 days.

	PV	PF
Antigen	Desmoglein 3>1	Desmoglein 1
Split	Suprabasal	Subcorneal
Skin	Vesicles and bullae rupture in few days	Crusted erosions in seborrheic distribution
Mucous membrane	Always present	Absent

Variants of Pemphigus Foliaceus

00:51:35

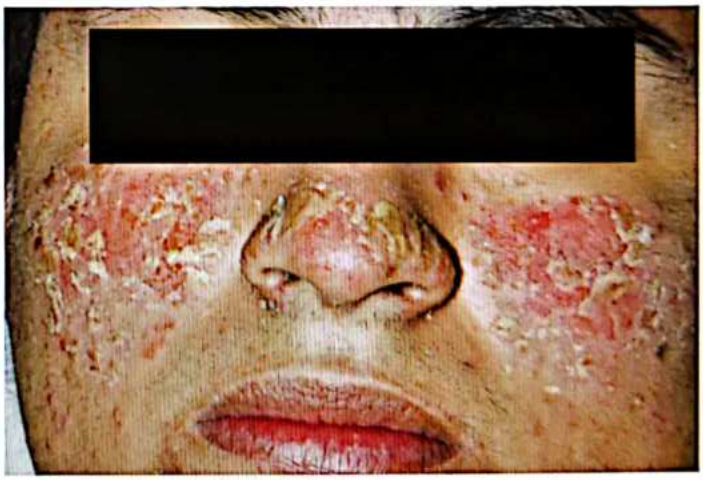
1. Pemphigus herpetiformis



- PF (Pemphigus Foliaceus) + DH (Dermatitis herpetiformis)
- Widespread clusters of pruritic papules and vesicles develop on an erythematous background.
- **Biopsy**: DH (Dermatitis herpetiformis)
- **Direct immunofluorescence** : PF (Pemphigus Foliaceus)

2. Pemphigus erythematosus

00:52:29



- PF + LE (Lupus erythematosus)
- Butterfly like rash - photosensitivity
- **ANA**: LE
- **ANA+Direct immunofluorescence**: PF (Pemphigus Foliaceus) +LE

3. Endemic PF (Fogo selvageum) 00:53:21

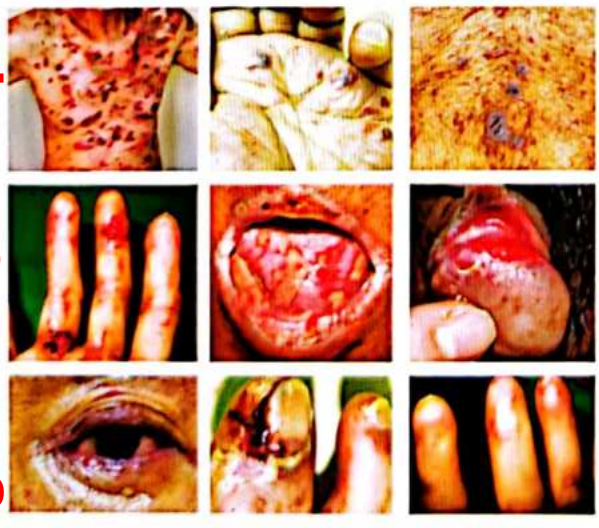
- **Endemic**: Certain parts of South America (Brazil, Columbia etc.)
- Common in children and young adults
- **Risk factor**: Arthropod bite (Blackfly- Simuliidae)
- Initial lesions are flaccid bullae
- **Nikolay sign**: positive
- Head and neck involved first
- Burnt appearance & burning sensation - Fogo selvagem, (Portuguese for 'wildfire')
- **Mucous membrane**: not involved

4. Drug induced Pemphigus 00:53:18

- Present as- PF>PV
- Thiol group: penicillamine
- Non thiol: ACE inhibitors, glibenclamide
- Phenol: cephalosporins, rifampicin, pyritinol, phenobarbital and aspirin

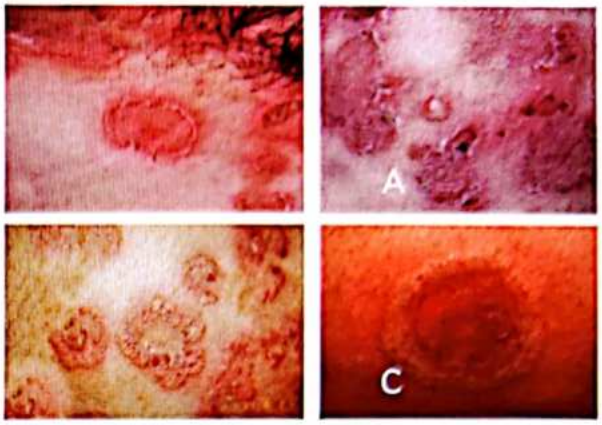
5. Paraneoplastic Pemphigus 00:57:11

- **Antigen**
  - DSG 1 and DSG 3
  - Desmoplakin 1 and 2
  - Envoplakin
  - Periplakin
  - 230 kDa bullous pemphigoid (BP) antigen 1
- **Association**: underlying neoplasms
- **Commonest**: Non hodgkin's lymphoma and B-cell lymphomas
- **Others**: chronic lymphocytic leukemia, Castleman disease, thymoma and Waldenstrom macroglobulinemia
- **Criteria**: Anhalt's criteria
- **Clinical features**:
  - Polymorphic
  - → Vesiculobullous
  - → Lichenoid
  - → Severe mucosal involvement



Golden Point: Paraneoplastic Pemphigus is the most severe type of Pemphigus.

6. IgA Pemphigus 01:01:28



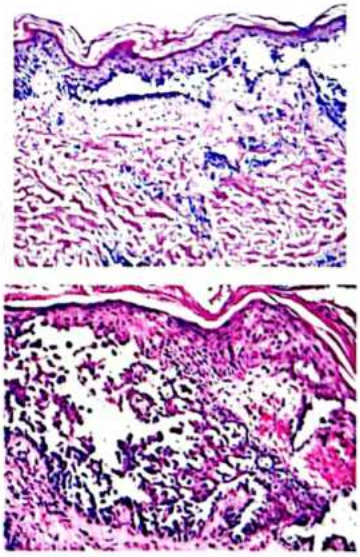
- **Antigen**: Desmocollin
- **Antibody**: IgA
- Children
- Circulate/ circinate lesions

Diagnosis of Pemphigus 01:02:27

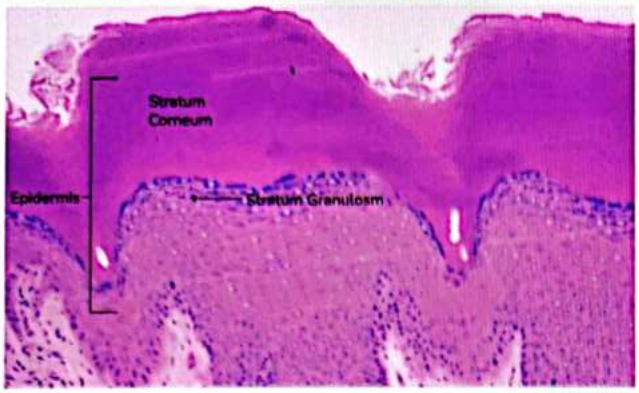
1. **Tzanck smear**: Acantholytic cells/ Tzanck cells

2. **Histopathology**

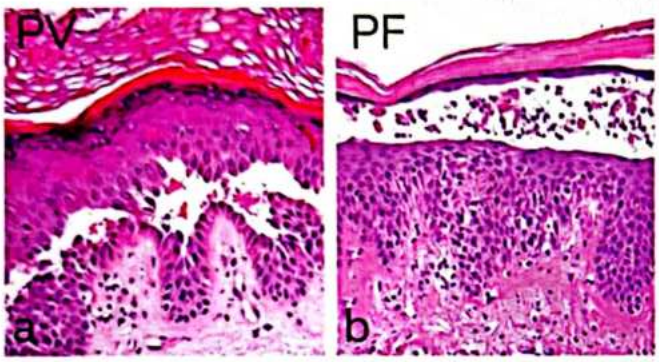
- Two samples are taken
  - Lesional sample: H&E staining
  - Perilesional sample: Direct immunofluorescence



- Pemphigus vulgaris-
  - **Split**: suprabasal
  - Row of tombstone appearance
  - Acantholytic cells

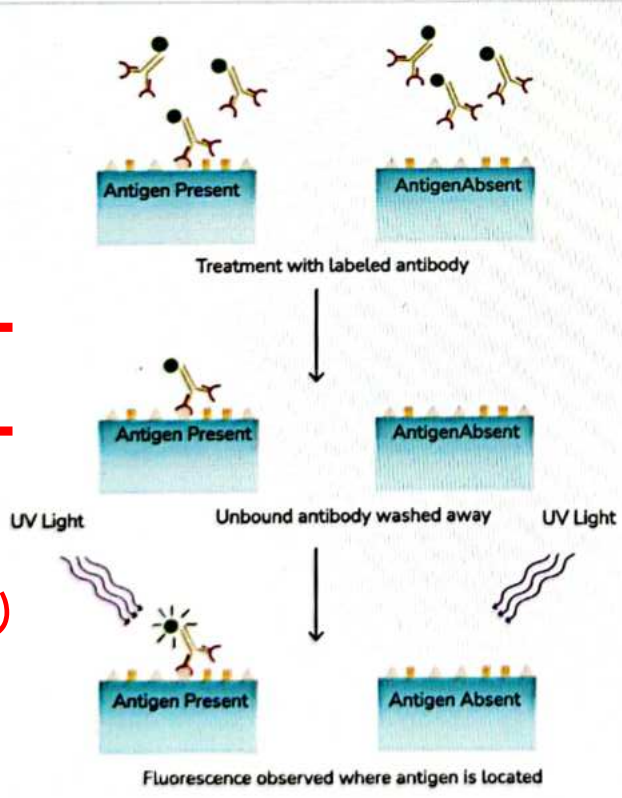


- Pemphigus Foliaceus-
  - **Split**: Subcorneal

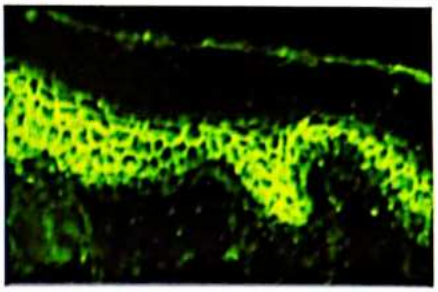


3. Direct immunofluorescence (DIF)

- Most diagnostic
- Demonstrate the presence of Ag-Ab complexes



- Principle of Direct immunofluorescence-
  - Fluorescent labeled antibodies bind to antigens.
  - Fluorescence is seen under UV light.



- Fluorescence passes between keratinocytes- Fishnet pattern.
- Fluorescence is more in suprabasal area- PV
- Fluorescence is more in Stratum corneum- PF

Important Information

Fishnet pattern is the characteristic feature of Pemphigus group of disorders.

4. Other tests

a. (Indirect immunofluorescence)

i. Blood samples containing pathogenic antibodies are collected

ii. Fluorescent labeled anti-antibody is placed

b. ELISA

Treatment of Pemphigus 01:12:13

a. Topical: Topical corticosteroids

b. Systemic

- Oral Corticosteroids and Immunosuppressants- Treatment of choice
  - → 1-2 mg/kg body weight
  - → 4-8 weeks and tapered off as needed.

Important Information

- **TOC:** Oral Corticosteroids and Immunosuppressants.
- Immuno-bullous disorders need treatment for a long time.

c. Adjunctive: Antibiotics

d. Pulse therapy

- High doses of corticosteroids are given for a few days in a month.
- In a 28 day cycle
  - **3 days:** High doses
  - **25 days:** No drugs
  - **3 days:** High doses
- **Two types**

- **DCP** (Dexamethasone Cyclophosphamide) pulse
- **MP** (Methylprednisolone) pulse

- **Advantages**
  - Less side effects
  - Diabetic or immunosuppressed patients are treated without giving corticosteroids on long term basis

	Azathioprine (AZA)	Mycophenolate Mofetil (MMF)	Cyclophosphamide
MOA	Inhibits purine synthesis	Inhibits IMP synthesis	Alkylating agent
Dose	2-3 mg/kg/day	1-3g/day	1-2 mg/kg
SE	Bone marrow suppression, Nausea, liver dysfunction	Bone marrow suppression, Gastrointestinal symptoms	Gonadal failure, hemorrhagic cystitis, bone marrow suppression and an increased risk of bladder cancer
Remarks	Enzyme thiopurine methyltransferase (TPMT)		Fertile women are contraindicated, may cause gonadal toxicity

Important Information

AZA, MMF, Cyclophosphamide are preferred along with corticosteroids.

Other drugs
- Gold
- Dapsone
- Methotrexate

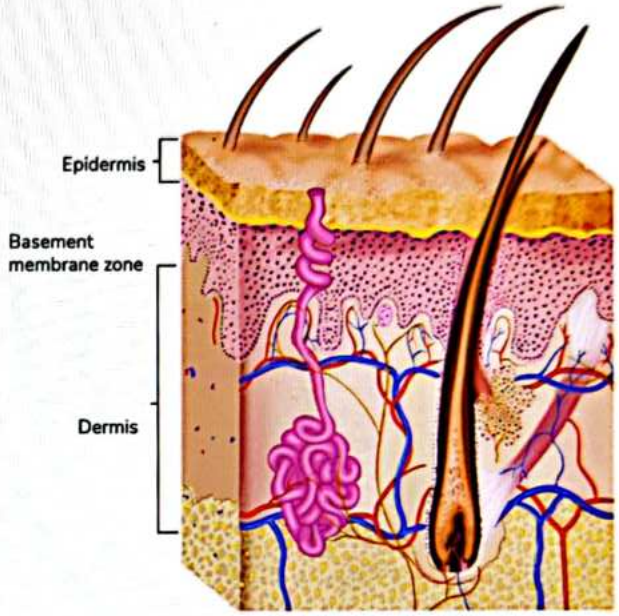
Biological agents (Rituximab)
- Anti-CD 20 molecule
- Forms immunoglobulins
- Binds with Pemphigus
- Intravenous agent
- Given at 0 and 15th day

Other methods
- IV Immunoglobulins
- Immunoadsorption
- Plasmapheresis

Subepidermal Disorders

01:23:06

- **Target:**
  - Basement membrane zone
  - Dermo epidermal junction (DEJ)
- **Types**
  - Pemphigoid group
  - Dermatitis Herpetiformis
- **Bulla**: tense (thick roof of epidermis)
  → Persist for few days
  → May be Hemorrhagic

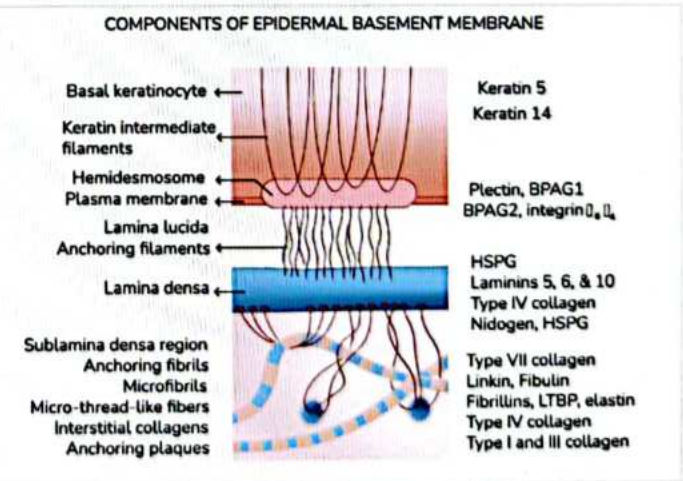


Important Information

Bulla in Pemphigus group are Flaccid (Old grape or old blown balloon)

Components of Basement Membrane Zone

01:27:28



- **Basal keratinocyte**: Keratin 5,14
- **Hemidesmosome:**

- Connects keratin intermediate filaments to Subepidermal layer.
- Agents- Plectin, BPAG1, BPAG2, integrin.
- Connected to Lamina lucida by Anchoring filaments.

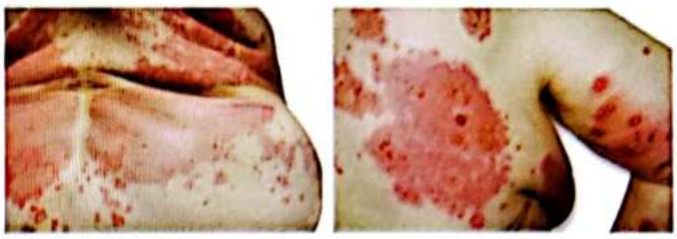
- **Lamina densa**: Laminins 5,6 &10, Type 4 collagen, Nidogen, HSPG
- → has Anchoring fibrils and Anchoring plaques.
- **Sublamina densa**: Type 7 collagen

Classification and Antigen 01:33:22

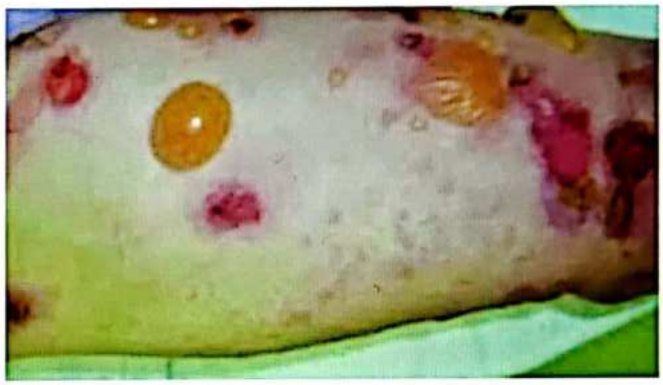
Disease	Target antigens
Bullous pemphigoid	BP 180, BP 230
Pemphigoid gestationis	BP 180, BP 230
Linear IgA bullous dermatosis (LABD)	BP 180 (LAD1)
Epidermolysis bullosa acquisita	Collagen VII
Bullous lupus erythematosus	Collagen VII
Cicatricial pemphigoid	BP 180, laminin, a_4 and b_6 subunits of integrin
MMP (Mucous Membrane Pemphigoid)	BP 180, laminin 332
Linear IgA	LAD1
DH (Dermatitis Herpetiformis)	Epidermal Transglutaminase (not from basement membrane)

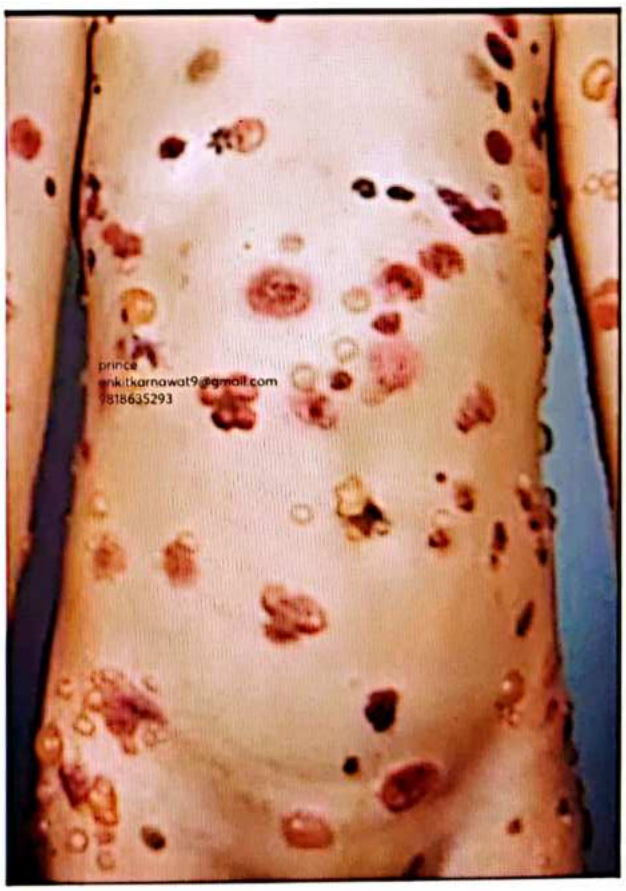
Bullous Pemphigoid 01:37:00

- **Antigen**: BPAG1 (230 kilo dalton), BPAG2 (180 kilo dalton)
- **Antibody**: IgG
- **Pathogenesis**:
  - Complement activation
  - Eosinophil recruitment
- **Age**: Elderly (60-80 years)
- **Association**: Neurological, autoimmune, psychiatric disorder
- **Triggers**: trauma, drugs
- **Site**: Trunk and near umbilicus
- Clinical features
  - Prebullous stage
    - Itchy urticarial lesions which are refractory to treatment

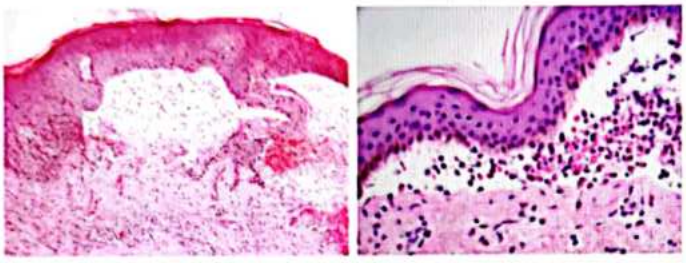


2. Bullous stage
   - Tense bullae (Source hemorrhagic)
   - Rupture in few days
   - Leave crusted erosions
   - Heal with milia formation
   - No scarring
   - No tendency to spread

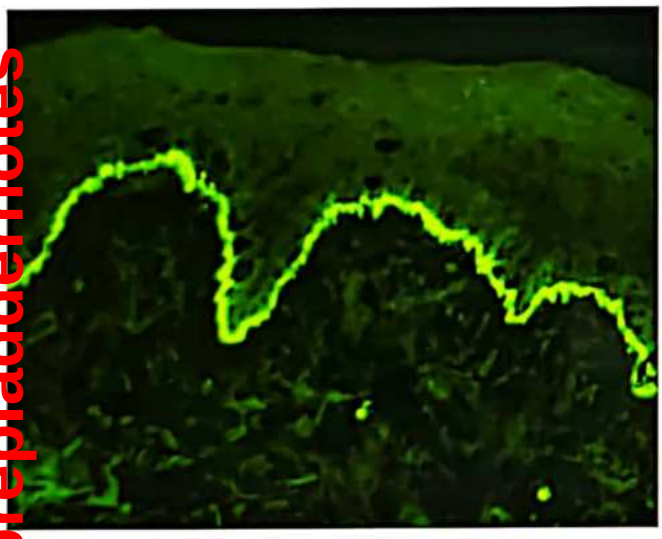




- **Mucosa**: rarely involved
- **Nikolsky sign**: Negitive
- **Bulla spread sign**: Positive, Round spread
- **Tzanck smear**:
  - No Acantholytic cells
  - Inflammatory cells are seen
- **Histopathology**
  - **Split**: Subepidermal with inflammatory cells

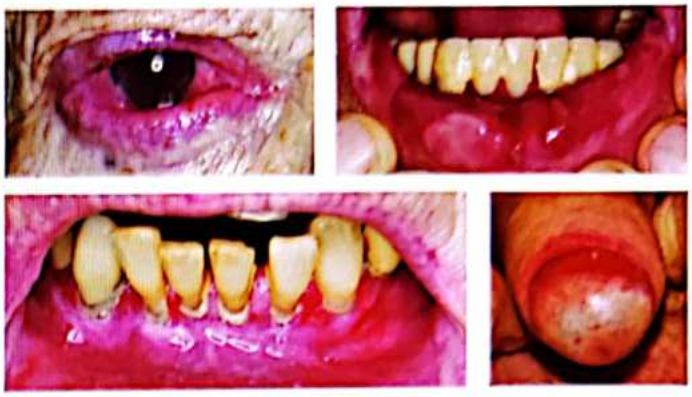


- **Direct immunofluorescence**
  - Anti fluorescent antibodies bind at dermo epidermal junction
  - Linear deposition of IgG and C3



- Treatment
  - **Mild**: topical corticosteroids (CS)
  - **Severe**:
    → Oral CS
    → Dapsone
    → Nicotinamide
    → Tetracyclines
    → Other immunosuppressants

Mucous Membrane Pemphigoid (MMP) 01:45:09



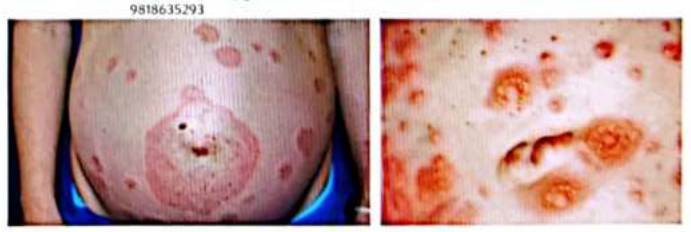
- **Antigen**: BP 180 and Laminin 332
- Scarring erosions are deep

Cicatricial Pemphigoid 01:45:48

- **Antigen**: BP 180
- Skin lesions
- No mucosal lesions
- Scarring is seen
- Brunsting-Perry Pemphigoid- **affects head and neck area**

Pemphigoid Gestationis 01:46:25

- Bullous Pemphigoid in pregnancy
- Also called as Herpes Gestationis (HG) just a missionary
- **Population affected**:
  - Pregnant females - 3rd trimester
  - Rarely, people with trophoblastic tumors
- Circulating complement fixing IgG antibodies of the subclass IgG1 (HG factor) bind to BP 180
- **Antibody**: C3>IgG
- Clinical features-
  - Starts in periumbilical area
  - Itchy urticarial plaques, eventually shows formations of tense vesicles and bullous.



- **Course**: Recur in subsequent pregnancy, can be associated with adverse fetal outcomes
- **Histopathology**
  - Split: Subepidermal
- **Direct immunofluorescence**: C3 deposit in 100% of cases
- Treatment:
  - **Mild**: topical Steroids

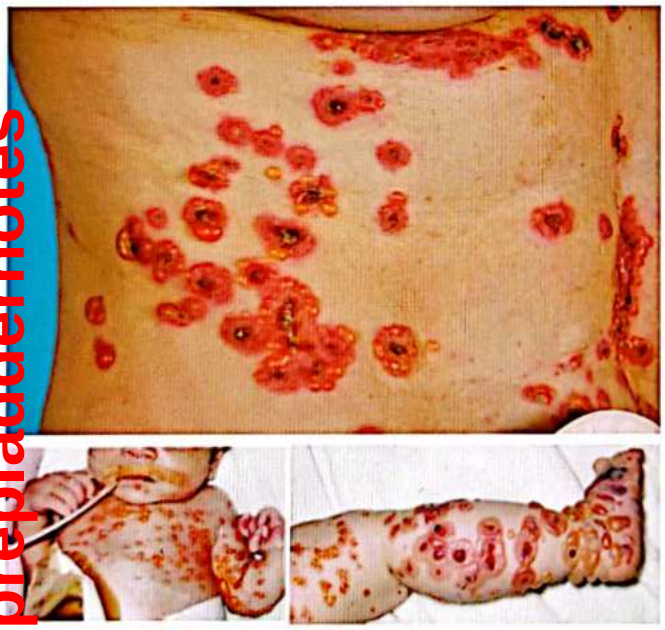
- **Severe:** oral steroids

Important Information

In pregnancy, other drugs like dapsone, immunosuppressants are toxic.

Linear IgA Disease (LABD) 01:50:44

- **Antigen:** LAD1, BP 180
- **Antibody:** IgA
- **Age:** Bimodal
  - Children (Chronic bullous disease of childhood-CBDC)
  - Adults (LAD)
- **Site**
  - **Adults:** trunk
  - **Children:** perioral area
- **Clinical features-**
  - Vesicles and tense bullae are in the form of rings (Cluster of jewels)
  - Perioral distribution
  - Cluster of jewels on body



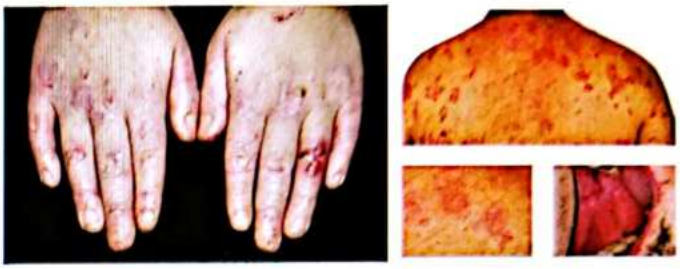
- **Histopathology**
  - Split: Subepidermal
- **DIF:** Linear IgA deposits
- **Treatment:** Dapsone

Important Information

DIF gives
- Antibody description
- Site of fluorescence
- Activity of disease

- DIF is done in patients whose treatment is about to complete.
- Negative DIF indicates to stop the treatment

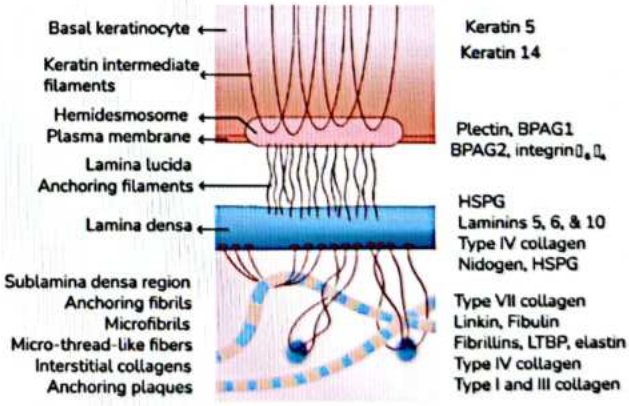
Epidermolysis Bullosa Aquisita (EBA) 01:55:32



- **Antigen:** Collagen type VII
- Acquired form
- Mechano bullous disorder- Bullae are formed at the site of trauma
- Heal with scarring

Salt-Split technique 01.57.46

- It helps to Differentiate between EBA and BP
- Splits the basement membrane zone (Lamina lucida), by adding 1M NaCl.
- Split happening at Lamina lucida
- In **BP**, immunofluorescence is present above the split (roof)
- In **EBA**, Immunofluorescence is present below the split (floor)

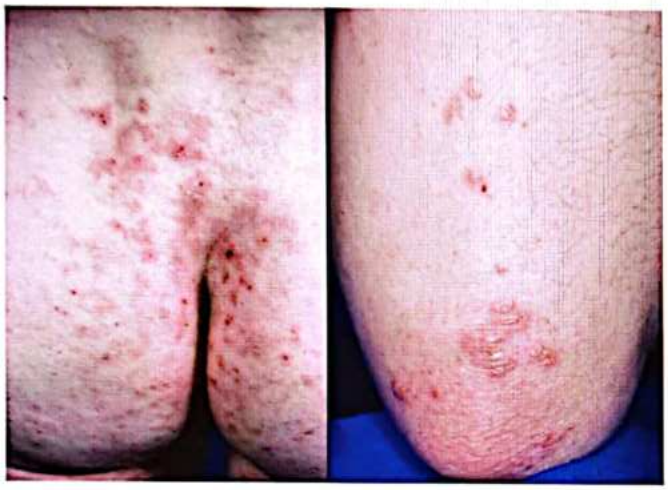


Dermatitis Herpetiformis 02:00:06

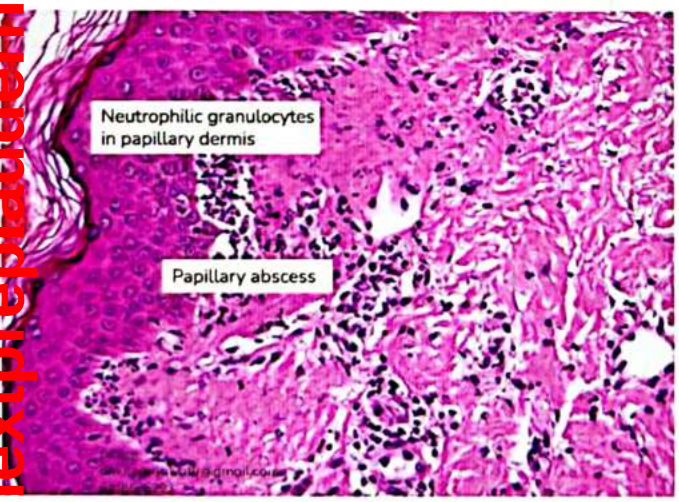
- Subepidermal Immunobullous Disorder
- **Association:**
  - Gluten sensitive enteropathy
    → Diarrhea

→ Abdominal distension
→ Bloating

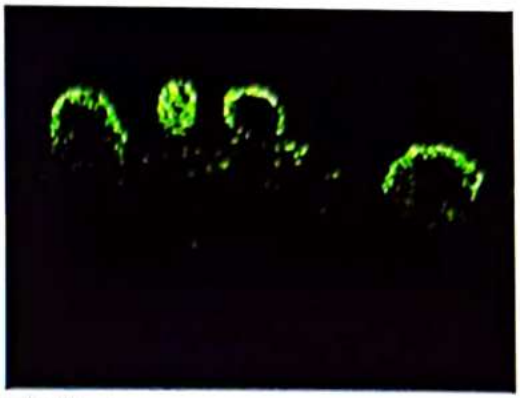
- **HLA association:** DQ2, B8
- Iodide can provocate the disease
- **Antigen:** Epidermal Transglutaminase
- **Clinical features**
  - Lesions are present on extensors and trunk
  - Itchy excoriated papulo vesicular lesions
  - No bulla
  - Refractory to treatment



- **Histopathology**
  - Papillary micro abscesses composed of neutrophils
  - Neutrophilic infiltration targets Epidermal Transglutaminase



- **DIF-**
  - Papillary tip immune fluorescence, papillary tip is involved.



- **Treatment:**
  - Dapsone
  - Avoid Gluten
    → Barley
    → Rye
    → Oats
    → Wheat
  - Eat maize and rice

Bullous SLE 02:07:48

- **Antigen:** Collage VII
- SLE (Systemic Lupus erythematosus) + Bulla
- **DIF**
  - Granular
  - Linear IgG and C3

PREVIOUS YEAR QUESTIONS

Q. Which of the following can be taken by a patient suffering with Dermatitis Herpetiformis?

- Barley
- Rye
- Oats
- Wheat

Ans: Oats

Q. Which of the following can be taken by a patient suffering with Dermatitis Herpetiformis?

- Dosa
- Chapati
- Noodles
- Pasta

Ans: Dosa

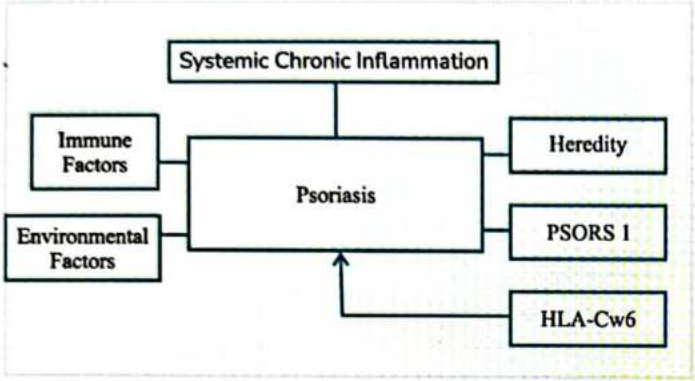
10 PAPULOSQUAMOUS DISORDERS

- Disorders with scaly papules and plaques. 00:00:06

Psoriasis 00:00:55

- Chronic disorder
- Relapsing remitting course
- Immune mediated

Etiology (Multifactorial)



- Factors which lead to systemic chronic inflammation are
  - Genetic factors (Heredity)
  - Immune factors
  - Environmental factors

Genetic Factors

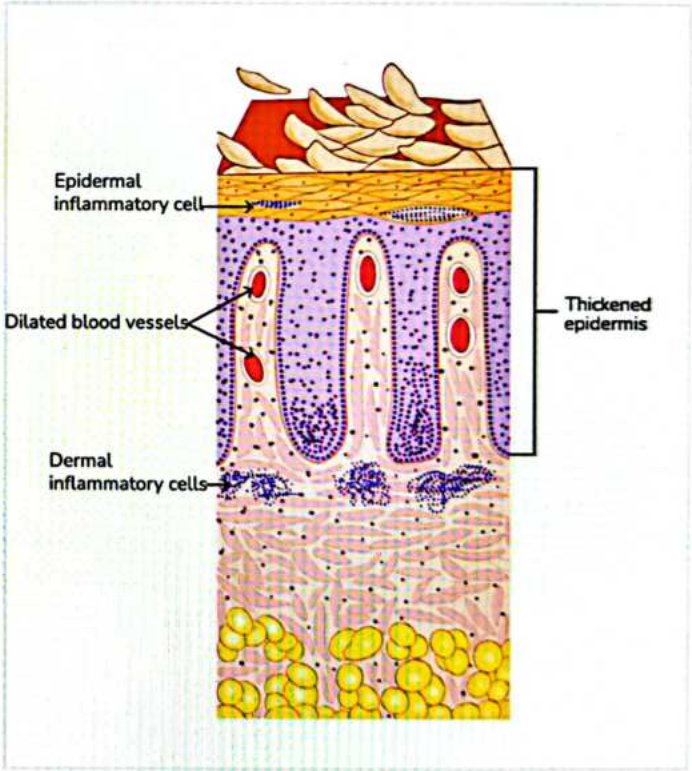
HLA Cw6	Early onset disease, positive family history
HLA B27	Psoriatic arthritis, nail changes
PSOR1	

Environmental Factors

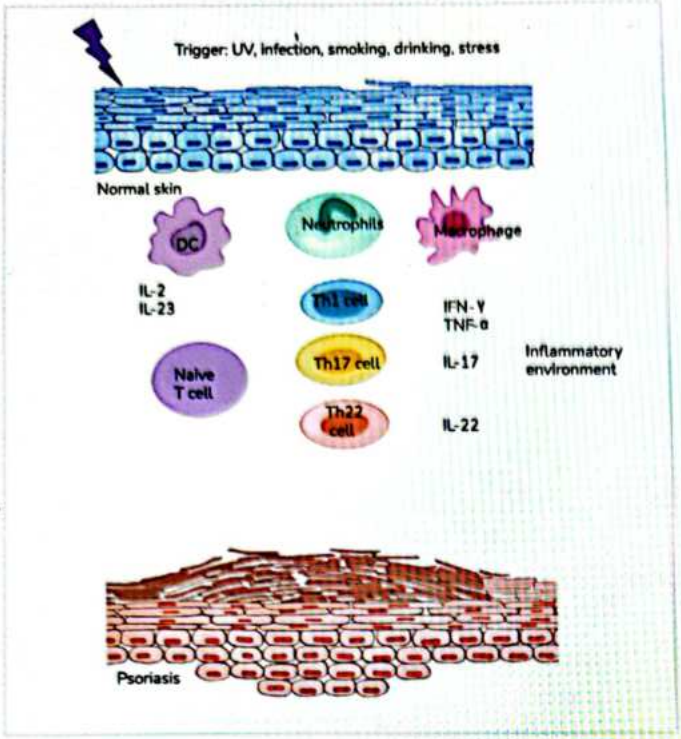
- **Infection**
  - Streptococcal throat infection which develops Guttate psoriasis
  - HIV positive aggravates psoriasis
- **Medications (BAT NAILS)**
  - Beta blockers
  - ACE inhibitors
  - TNA alpha inhibitors
  - NSAIDs
  - Antimalarials
  - IFN (Interferon) gamma
  - Lithium
  - Steroids

- Psoriasis aggravated by:
  - Alcohol
  - Cigarette smoking
  - Stress
  - Sunlight is protective, but in 20% cases it may aggravate psoriasis
  - Trauma

Pathogenesis 00:05:12



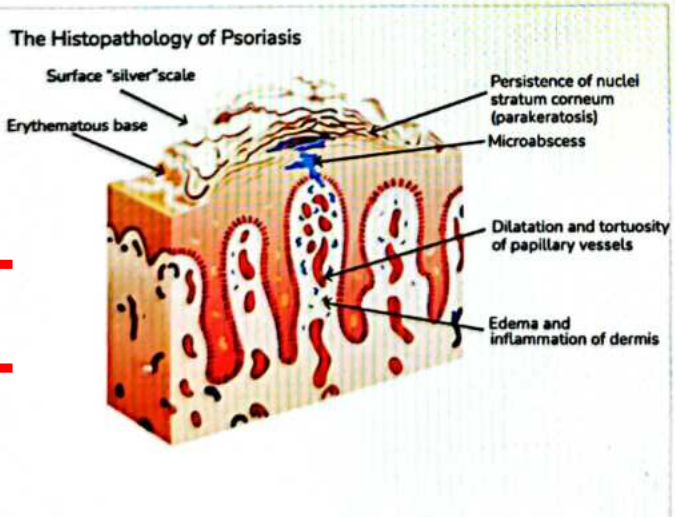
- Reduced epidermal turnover time to 4 days (normal 56 days) - This leads to hyperproliferation of keratinocytes
- Angiogenesis/ vasodilation - Increased blood flow
- Inflammation - TH-1 type of response is generated, no TH-2 response.



- **TH-1 has**
  - INF-γ
  - TNF-α
  - TH-17(IL-17)
  - TH-22 (IL-22)
- Inflammation causes vasodilatation and targets hyperproliferation in keratinocytes.

Histopathology

- Dermal changes precede epidermal changes

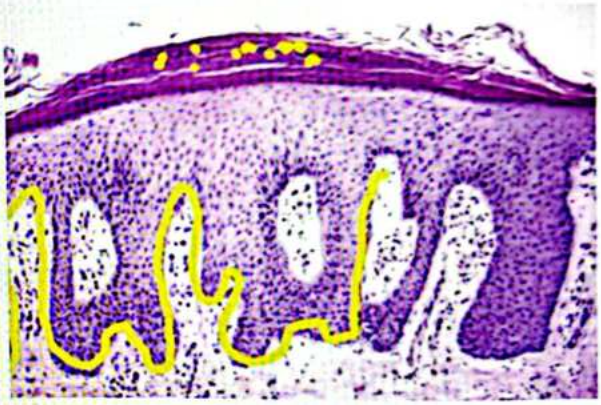


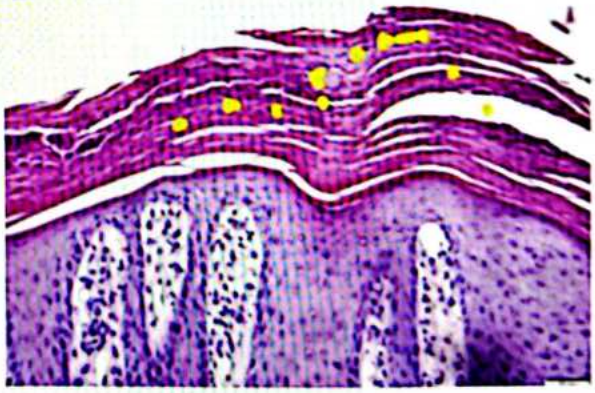
- **Stratum corneum**
  - Hyperkeratosis (Thickening of corneum)
  - Parakeratosis (Retention of nuclei in Stratum corneum)
  - Munro's microabscess (Formation of micro abscess-collection of neutrophils)
- **Stratum Granulosum**
  - Agranulocytosis/ Agranulosis (Absent granular layer)
- **Malpighian layer** (Stratum Spinosum and Basale)
  - Acanthosis (Increased thickness) of Stratum Spinosum
  - Kogoj's Spongiform pustules (Collection of neutrophils)

Important Information

- Rete ridges are the invaginations of epidermis into dermis.
- In psoriasis, rete ridges are elongated, and club shaped (Camel foot appearance).

- **Dermis**
  - Suprapapillary thinning
  - Dilated tortuous blood vessels
  - Inflammation

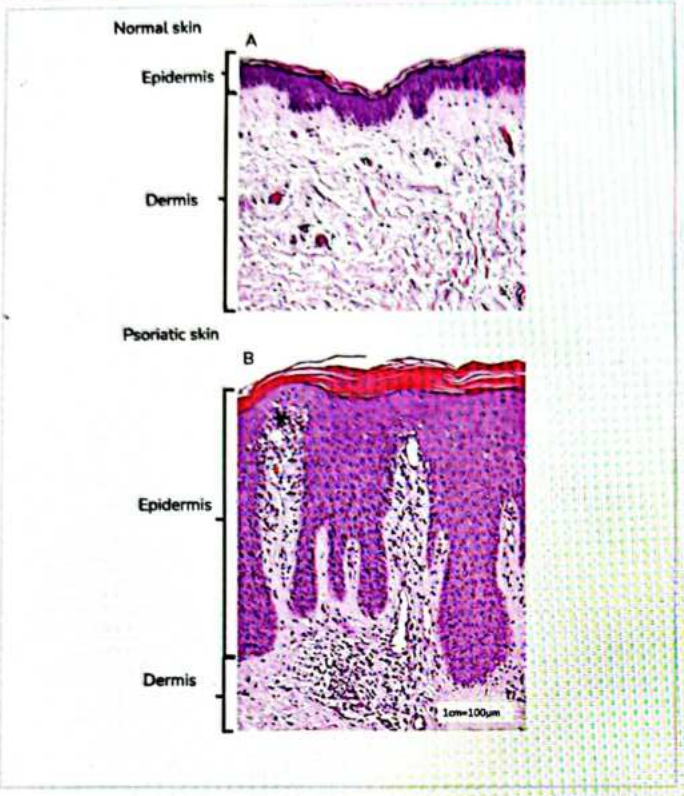




Important Information

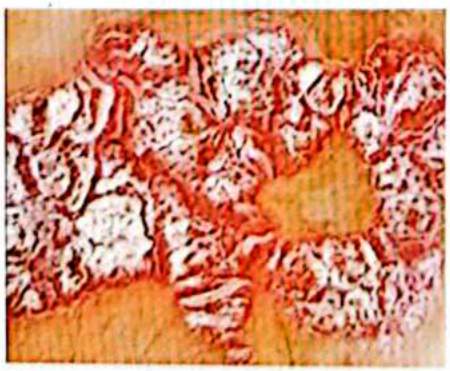
- Hyperkeratosis
- Parakeratosis is a giveaway in psoriasis
- Thickening of Stratum corneum, with many nuclei
- Rete ridges are club shaped, elongated and appear like camel foot

- No granular layer
- Stratum Spinosum is thick, Acanthosis
- Suprapapillary thinning
- Presence of inflammation and vasodilatation

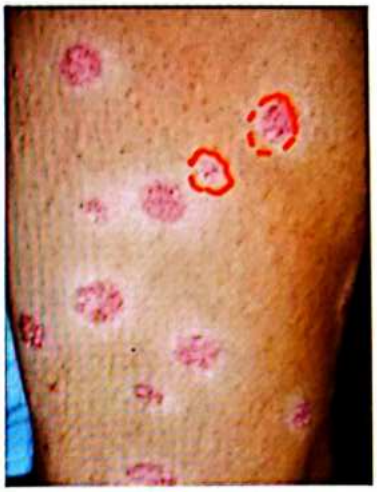


- Hyperkeratosis
- Rete ridges

- **Clinical Features of Psoriasis** 00:17:21
- **Symptoms**
  - No itching
  - Winter exacerbation
- **Site** - Extensors of the body
- **Symmetry of lesions** - Bilateral symmetrical
- **Color of lesions** - Salmon colored, due to increased inflammation and vasodilatation
- **Texture**

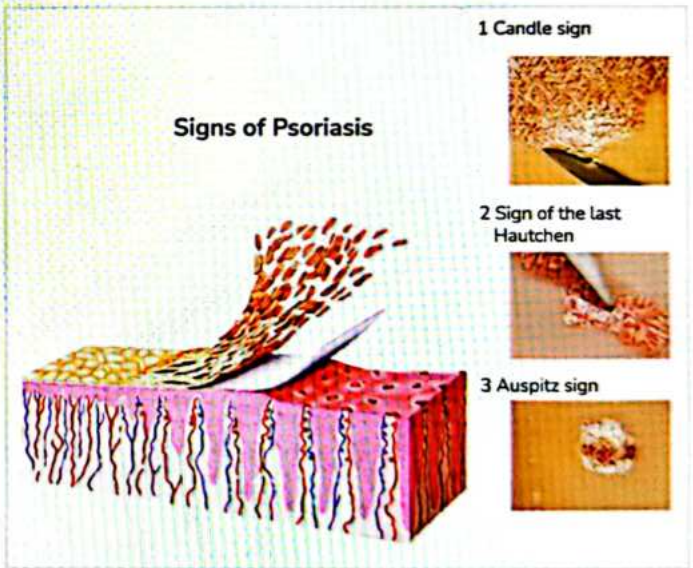


  - Well defined plaques and papules
  - **Erythematous:** Red
  - **Scaly:** Silvery white scale
  - **Indurated:** Thickened
- Woronoff's Ring



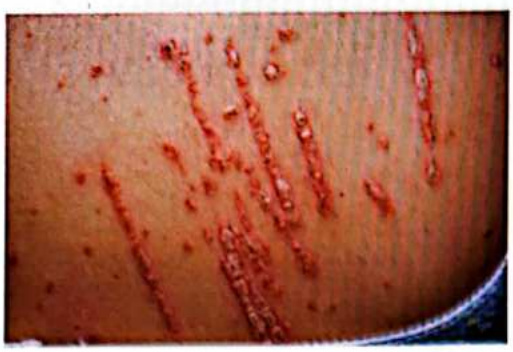
  - **Zone of pallor** occurs due to inability to synthesize PGE2.
  - This is seen in psoriatic patients.

Signs of Psoriasis



- Bedside tests are done with a blunt object scraping on the psoriatic skin. Continuous scraping produce:
  - **Grattage test/ Candle sign,** accentuation of scaling is increased.
  - **Berkley's membrane,** thin translucent membrane
  - **Auspitz sign,** pinpoint bleeding points
- The 3 tests are combinedly called Auspitz tests

- **Koebner's Phenomenon**



- Psoriatic lesions develop along the lines of trauma.
- Isomorphic response (similar morphology lesions are developed).
- All or none phenomenon.

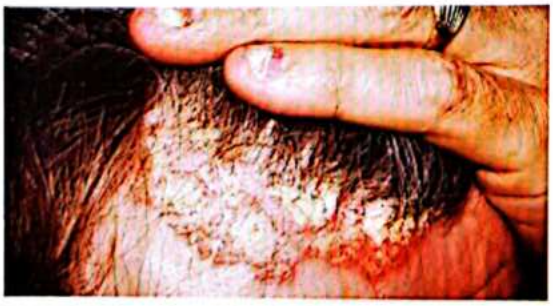
Category	Examples
Category 1 (True koebnerization)	Psoriasis, Lichens planus, Vitiligo
Category 2 (Pseudo koebnerization)	Warts, Molluscum
Category 3 (Occasional koebnerization)	Darier disease, Erythema multiforme
Category 4 (Dubious association with trauma)	Pemphigus vulgaris, Lupus erythematosus

In Pseudo koebnerization, autoinoculation or seeding of microbes is seen.

Classification of Psoriasis 00:27:49

Types
1. Based on sites
2. Based on morphology

Based on Sites
- **Scalp**
  - Scalp psoriasis



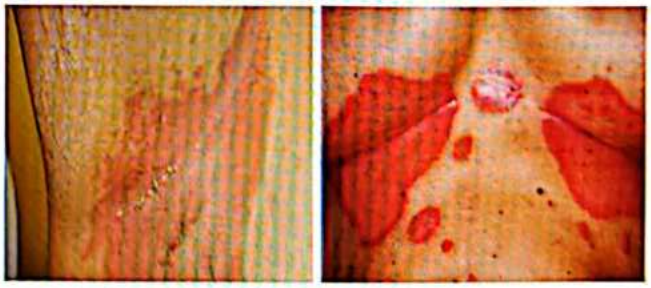
- Erythematous scaly plaques
- Cross hair line
- Not itchy
- No alopecia
- Extend up to forehead

Important Information
- Scalp psoriasis differs from Seborrheic dermatitis.
- Seborrheic dermatitis does not cross the hairline and itches.

- **Pityriasis amiantacea**

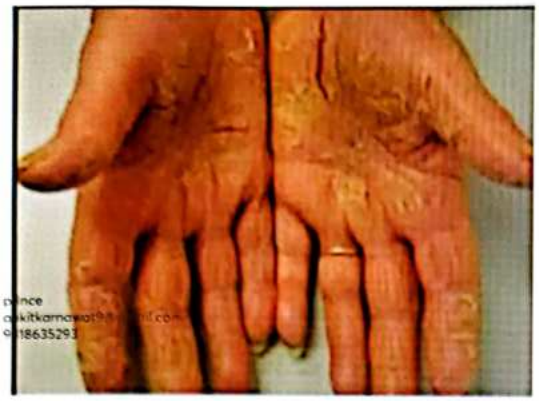
- Thick asbestos like scaling
- Also seen in infections and Lichen simplex

b. Flexures



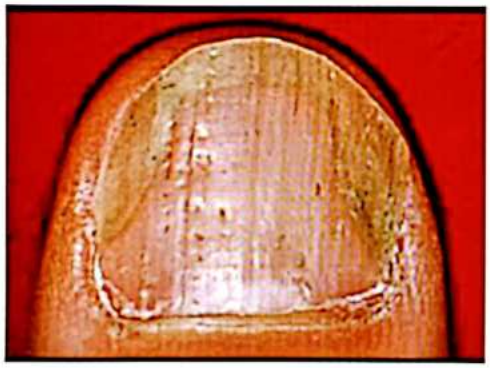
- Inverse/ flexural psoriasis
- Flexures like
  - Axilla
  - Groin
  - Infra mammary area
- No scaling
- Little induration
- Bilaterally symmetrical
- Not itchy

c. Palms and soles

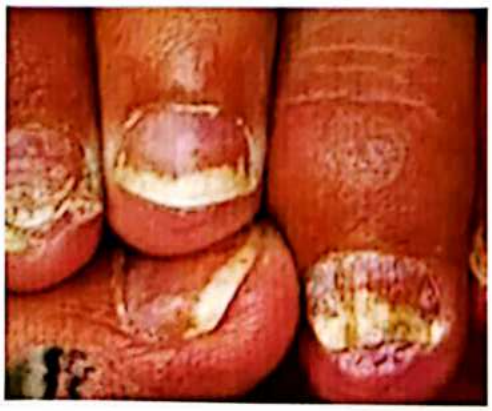


- Palmo-plantar psoriasis
- Hyperkeratosis
- Fissuring
- Hyerkeratotic plaque

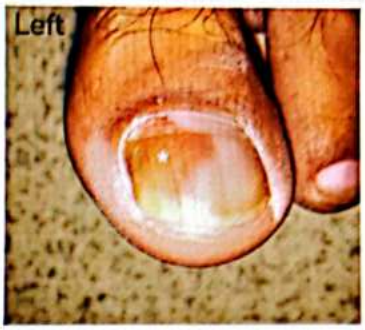
d. Nail (Nail psoriasis)



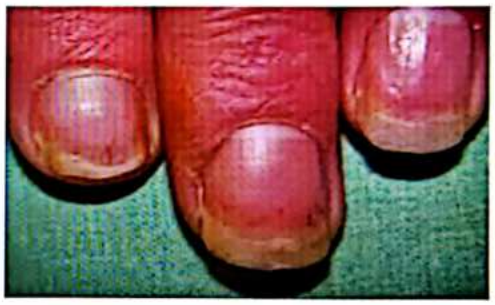
Association with Psoriatic arthritis
Pitting (shallow depressions) is the most common finding
Pits are
- Irregular
- Deep
- Random
- Large



- Onycholysis, separation of nail plate from bed.
- This appears white due to air and hyperkeratotic (subungual hyperkeratosis) materials.



- Salmon patch (Oil drop sign) is the most specific manifestation of Nail psoriasis.
- Translucent yellow-red discoloration in the nail bed proximal to onycholysis.
- Nail bed parakeratosis and psoriasiform hyperplasia is seen.



- On nail bed- dilated tortous blood vessels.
- Major findings in Nail psoriasis, POLISH
  - Pits
  - Onycholysis
  - Leukonychia
  - Salmon patch
  - Subungual hyperkeratosis
- Differences between Psoriatic nails and Onychomycosis

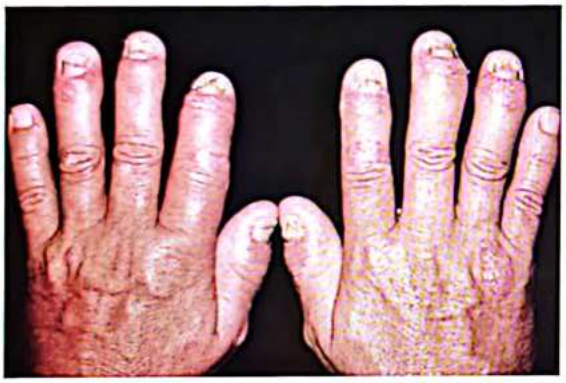
Psoriatic nails	Onychomycosis
Fingernails	Toenails
All the fingers	Few fingers
Pits and oil drops	No pits and oil drops
KOH: Negative	Positive KOH

e. Joints
- Psoriatic arthritis
- Seronegative
- HLA b27 gene association

Moll and Wright classification of Psoriatic arthritis	
Clinical pattern on presentation	**Percentage of patients**
Asymmetrical oligoarthritis	50
Symmetrical polyarthritis	40
Distal interphalangeal arthritis	5
Arthritis mutilans	5
Spinal column or axial involvement	40

Most common pattern - asymmetrical oligoarthritis
Most common joint involved- distal interphalangeal joint

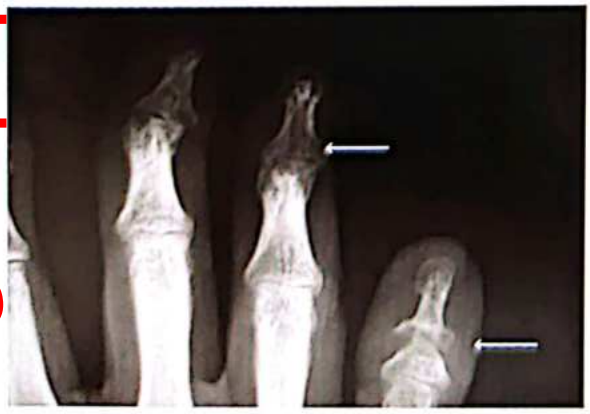
- Sausage digits, club shaped



Sausage digits

Pencil in cup deformity

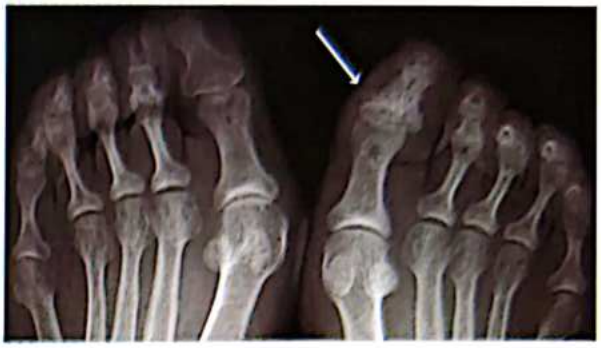
(a)



More osteochondritis

- Bone erosions
- New bones formation

- Ivory phalanx

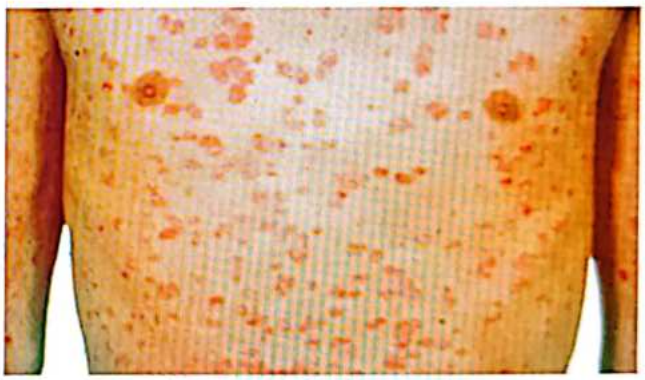


- Because of new bone formation, phalanx appears whiter in color

- Mucosa is generally not involved in psoriatic arthritis.
- But the tongue has some geographic tongue appearance.
- Ocular mucosa may be involved.
- Uveitis is associated with Psoriatic arthritis.

2. Based on Morphology

a. Guttate Psoriasis



- Raindrop like appearance
- Commonly seen in children.
- Preceding streptococcal infection.
- Small erythematous scaly plaques on the trunk.
- **Investigations**
  - ASLO swab
  - Throat culture
- **Treatment** with antibiotics
  - Penicillin
  - Amoxicillin
- If a recurrent episode occurs, Tonsillectomy is done.

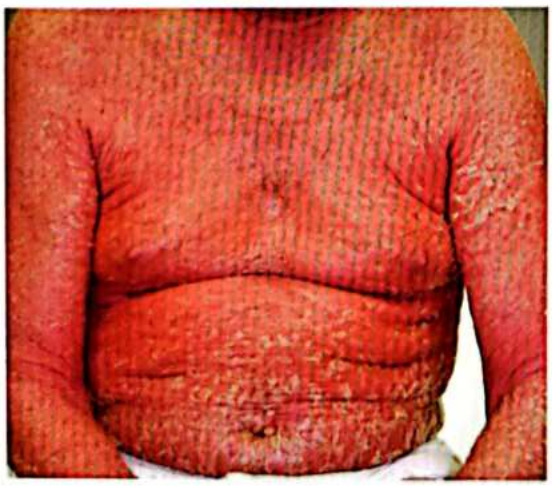
b. Unstable Psoriasis

- Constitutional symptoms
  - Fever
  - Malaise
  - Pain
  - Tenderness
- **Two types**
  - Erythrodermic psoriasis
  - Pustular psoriasis
- **Aggravating factors**
  - Withdrawal of steroids
  - Coal tar
  - Pregnancy
  - Stress
  - Infections
  - Hypocalcemia

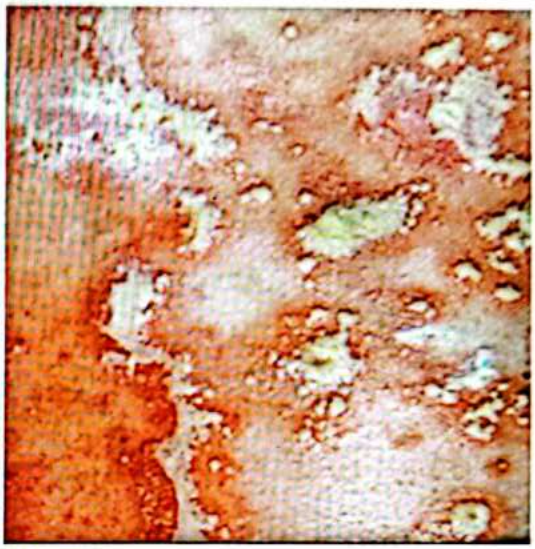
Important Information

Oral steroids are completely contraindicated in Unstable psoriasis.
Because they may lead to Rebound phenomenon.

Erythrodermic psoriasis

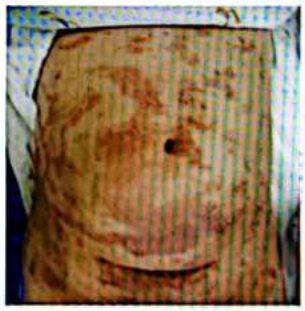


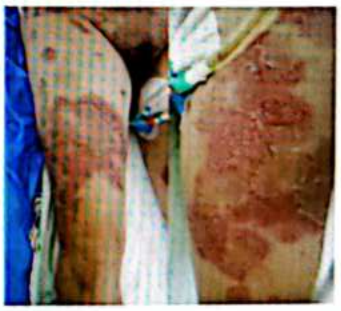
  - Erythema, scaling of > 90% of the BSA.
  - Red scaly skin
- **Pustular psoriasis**
  - Presence of visible Sterile pustules (collection of neutrophils)
  - Generalized or localized palms and soles
- Generalized
  - Acute generalized psoriasis, Von Zumbusch



→ Erythematous plaques studded with sterile pustules
→ Tenderness, discomfort
→ Constitutional symptoms
→ Lakes of pus
→ Treatment: Acitretin

  - Impetigo Herpetiformis





→ Pustular psoriasis of pregnancy
→ Seen in last trimester
→ Associated with adverse fetal outcomes
– Low birth weights
– Mortality
→ Recur in subsequent pregnancy
→ **Treatment**: Steroids

Important Information

- Steroids are contraindicated in psoriasis
- But in pregnancy they are considered as treatment of choice.
- Cyclosporine is considered as the second line of choice.

- **Management of unstable psoriasis**
  - Admit in ICU
  - Fluid, temperature monitoring
  - Avoid aggravating factors

c. Other typical forms

- Elephantine psoriasis
- Rupioid psoriasis -Limpet/ cone like scales

Associations of Psoriatic Patients

- Metabolic syndrome- Psoriatic March
- Other autoimmune disorders

Treatment of Psoriasis 00:53:43

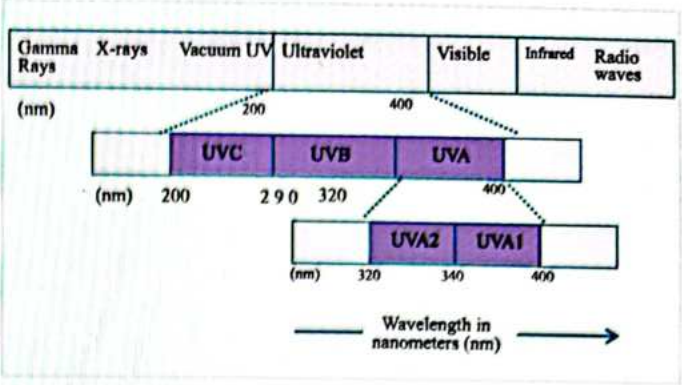
1. Topical Treatment

- <10% BSA
- <10 PASI (Psoriasis Area Severity Index)
- **Treatment of choice**
  - **Emollients**, moisturization and hydration of scaly skin
  - **Corticosteroids** are given topically, but not systematically
  - **Topical Retinoids**: Tazarotene, improve proliferation or keratinization
  - **Vitamin D analogues:** Calcipotriol, for keratinization.
  - **Coal tar** (Goeckerman's regimen)
  - **Anthralin** (Ingram's regimen)
  - **Salicylic acid**
  - **Calcineurin inhibitors** (Tacrolimus, Pimecrolimus) for face and flexures.

Important Information

- Goeckerman's regimen, apply coal tar paste to the affected areas and phototherapy is performed the next day.
- Coal tar is smelly, used hardly nowadays.
- Ingram's regimen, apply Anthralin paste to the affected areas and phototherapy is performed the next day.
- Anthralin produces staining on the clothes and is irritating.
- Steroids in long use can cause atrophy, hence Calcineurin inhibitors are given.

2. Phototherapy/Light therapy



- Wavelengths of light are used to cure.
- UV light has 3 spectrums
  - UV-A (320-400)
  - UV-B (290-320)- Broad band UV-B
  - UV-C (200-290)
- In phototherapy we use UV-A and UV-B.
- Most used phototherapy is NV (Narrow Band) UVB therapy.
  - 311-313 nm
  - Very specific for psoriasis
  - Patient must visit 3 times a week
  - Reduces inflammation
  - Acts at level of DNA.
  - Compliance is difficult
  - Not combined with cyclosporine, it may increase the risk of carcinogenesis

PUVA (Psoralen UVA) phototherapy

- Topical and Oral forms
- 5-MethoxyPsoralen (5-MOP), 8-MethoxyPsoralen (8-MOP) and Tri-Methoxy psoralen (tri-MOP) are used
- Psoralens taken by the patient bind to DNA.
- When exposed to UVA, DNA damage occurs.
- Patient must visit 2 times a week
- Longer exposure may lead to
  - Cataracts, due to their deposition.
  - Skin cancers (SCC and BCC)

Important Information

- Usually, 300 treatments are to be done using phototherapy.
- UVB is preferred over UVA, due to adverse effects of UVA on longer exposure.

Targeted phototherapy

- Excimer lasers/ lamps, 308 xenon-chloride excimer laser
- Targeted UVB therapy consists of a noble gas and halide.

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3. Systemic Treatment

a. Main Drugs

- **Methotrexate (MTX)**
  - Dihydrofolate (DHFA) reductase inhibitor
  - 7.5-25 mg once a week
  - Treatment of choice in
    - → Chronic plaque psoriasis
    - → Psoriatic arthritis
    - → Erythrodermic psoriasis
  - **Side effects**
    - → Nausea
    - → Vomiting
    - → Bone marrow suppression
    - → Teratogenesis
    - → Liver toxicity and fibrosis (dose related)
  - **Instructions**
    - → Take folic acid supplements on the other days
    - → Take MTX subcutaneously
    - → Monitor platelets count regularly
    - → Contraindicated in pregnancy
    - → Monitor LFT, if toxic, stop the usage.
- **Cyclosporin**
  - Inhibits Il2
  - Crisis drug, only used to control severe acute episodes
  - Not used of long term
  - Side effects

Side Effects of Cyclosporin

Involved Organs and System	Adverse Effects
Metabolism and nutrition disorders	Hyperlipidemia
Nervous system disorders	Tremor, Headache, Paresthesia
Cardiovascular disorders	Hypertension
Gastrointestinal disorders	• Nausea • Vomiting • Abdominal pain • Diarrhea • Gingival hyperplasia
Hepatobiliary disorders	Abdominal hepatic function
Skin and subcutaneous tissue disorders	Hypertrichosis
Musculoskeletal disorders	Muscle cramps myalgia
Renal disorders	Renal dysfunction

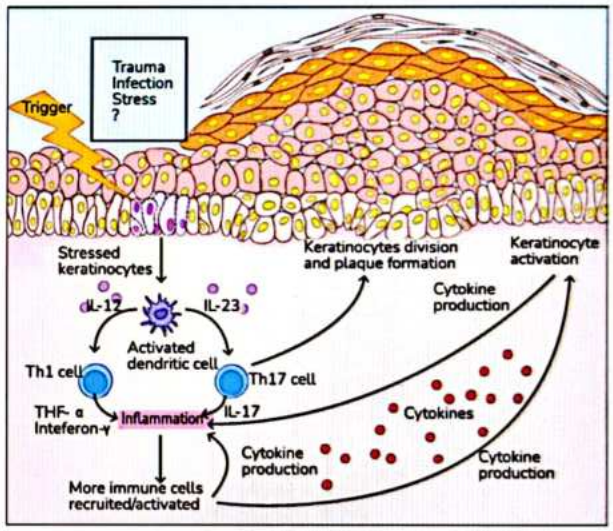
- **Acitretin**
  - Vitamin A analogue
  - Retinoids, two types
    - → Isotretinoin (Acne)
    - → Acitretin (Psoriasis)
  - Acitretin is derived from etretinate
  - Improve proliferation and keratinization
  - Reduce inflammation
  - Treatment of choice in
    - → Pustular psoriasis
    - → HIV
    - → Immunosuppression

- **Fumaric acid esters**
- **Hydroxyurea**

- **Oral drugs**
  - **Apremilast** - PDE 4 inhibitor
  - **Tofacitinib** - JAK kinase inhibitor

b. Biologicals

- Derived from living organisms



- Specific target drugs
- Used in patients not responding to conventional therapy
- Have fewer side effects, safer and effective
- Costly
- **Drugs**
  - Anti TNF alpha drugs
    - → Etanercept
    - → Infliximab
    - → Adalimumab
  - IL 12/23 Inhibitor
    - → Ustekinumab
  - LFA-1 (Lymphocyte Function Associated antigen)
    - → Alefacept

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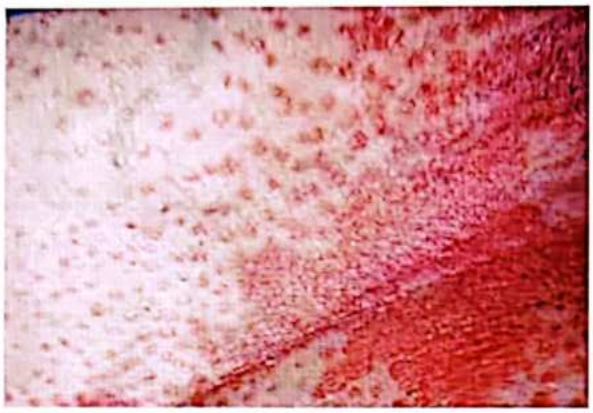
- IL 17A inhibitor
  → Secukinumab

Pityriasis Rubra Pilaris (PRP) 01:15:17

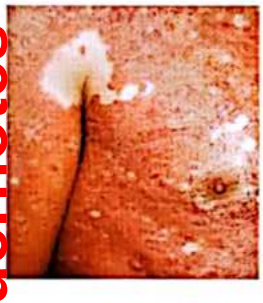
- Scaly red follicular papules or plaques
- Papulosquamous dermatitis of unknown cause

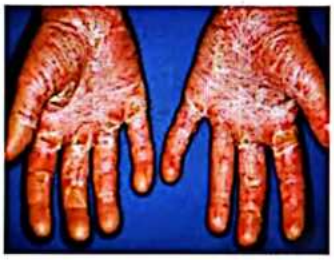
Histopathology

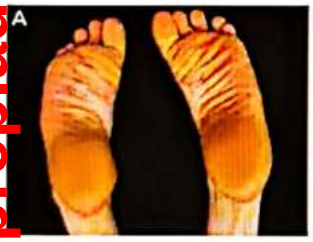
- Checkerboard pattern, alternate hyperkeratosis, and parakeratosis.

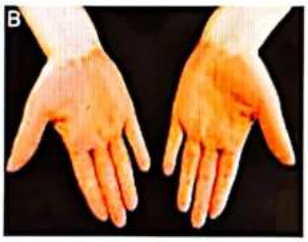


- Erythema is seen between the papules
- Salmon coloured erythema.
- Islands of sparing, which differs from psoriasis

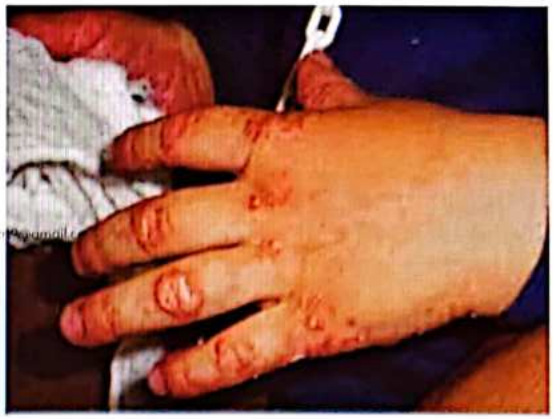








Palms and soles have hyperkeratosis, erythematous plaques. Appears to be a person wearing follicular sandals.



- Erythematous, scaly plaques on knuckles are known as Nutmeg crater papules .

Types of PRP

- Classical adult onset PRP (type 1)
- Atypical adult onset PRP (type 2)
- Classical juvenile onset PRP (type 3)
- Circumscribed juvenile PRP (type 4)
- Atypical juvenile PRP (type 5)
- HIV-related PRP (type 6)

Treatment

- Topical agents (same as psoriasis)
- **Oral agents**
  - MTX
  - Acitretin

Important Information

- HIV-related PRP (type-6) is seen in HIV patients.

87

PREVIOUS YEAR QUESTIONS

Q. Describe the pits in Alopecia areata.
Ans:
- Regular
- Geometric
- Shallow
- Superficial

Q. Most common clinical pattern in psoriatic arthritis is
Ans: Asymmetrical oligoarthritis

Q. Most common joint involved in psoriatic arthritis is
Ans: Distal interphalangeal joint

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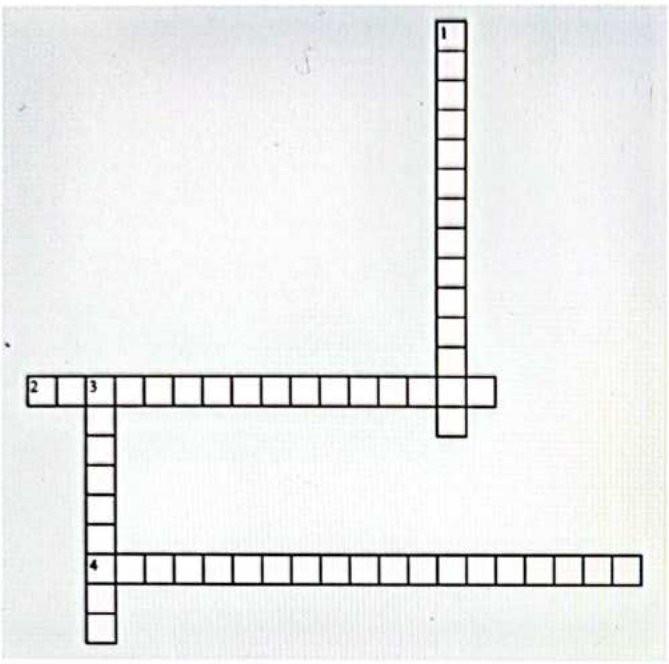
CROSS WORD PUZZLES

Crossword Puzzle



Across

2. Limpet
4. Guttatepsoriasis

Down

1. Seborrheicdermatitis
3. Chronicdisorder

prince
ankitkarnawat9@gmail.com
9818635293

11 LICHEN PLANUS

Lichen Planus

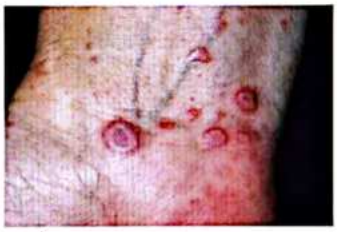
00:00:17

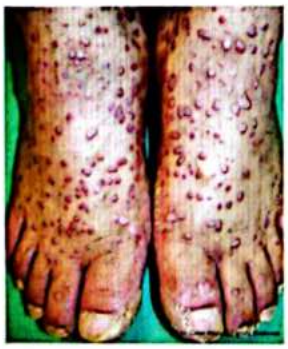
- Lichen: Lichenified (Purplish appearance with infiltrate at basement membrane).
- Planus: Plane.
- Autoimmune disorder.
- It is mediated by T cells.
- It affects the Basement Membrane Zone.
- Etiology: Unknown.
- In an external agent it can be an Infection (upto check for Hepatitis B, C in patients) or it can be a Drug.
- Some basic drugs which can cause Lichen Planus are:
  - Antibacterials
  - Anti-Tubercular Drug
  - Antihypertensive
  - Antimalarials
  - Anticonvulsants
  - Heavy metals-gold, lithium
  - Dental Amalgams:
  - In oral Lichen Planus, rule out dental Amalgams
    → If dental amalgam was placed before the development of Lichen Planus, tell the patients to get the dental amalgam removed.

Clinical Features

00:02:28

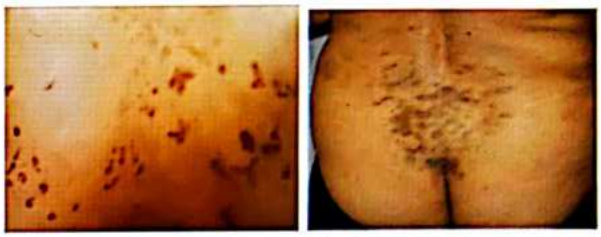
- Papulo squamous disorder which affects
  - Skin
  - Mucosa
  - Hair and nails.
- It is Pruritic and it affects Flexures.





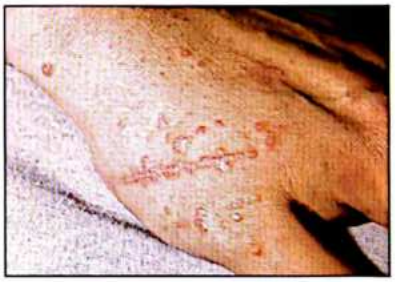
- The characteristic lesion of Lichen Planus is 5 p's-
  - Purplish
  - Plane topped
  - Pruritic
  - Polygonal
  - Papules and Plaque.
- It affects Flexures more than extensors but can be present anywhere on the body.
- The common sites are flexor of the wrist or on the ankle.

Post inflammatory Hyperpigmentation:



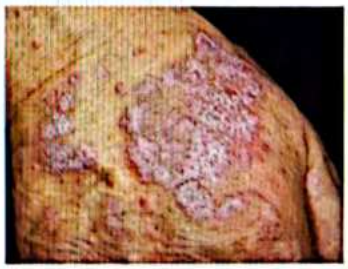
- The Lichen Plane healed with hyperpigmentation, which is called post Inflammatory Hyperpigmentation. hyperpigmented Macules are known as healed legion of Lichen Planus.

Koebner's phenomenon:



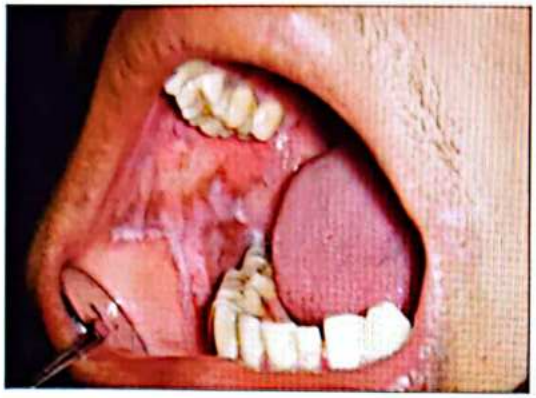
- Lichen Planus also shows Positive Koebner's phenomenon.
- Papules developed along the line of trauma.

Wickham's Striae



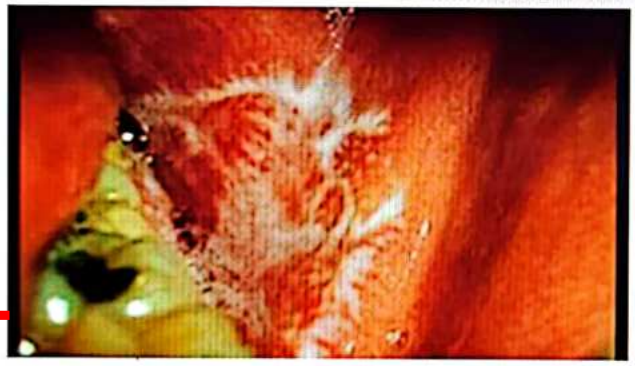
- The whitish streaks on the regions of Legion Planus seen on hand lens.
- This happens because of hyper granulosis (the granular layer is increased in thickness)

Oral lesions:



- In the mucosa, oral lesions which are symptomatic. The symptoms Include:
  - Pain & burning on eating hot spicy food.
- History of eating of hot spicy food,
- On examination: lacy reticular white pattern (lacy white streaks) like patterns in the oral mucosa, especially on the mucosa.

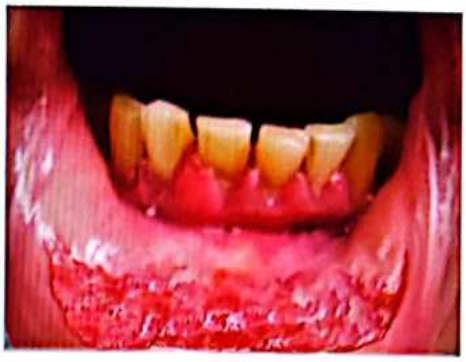
Dental amalgam



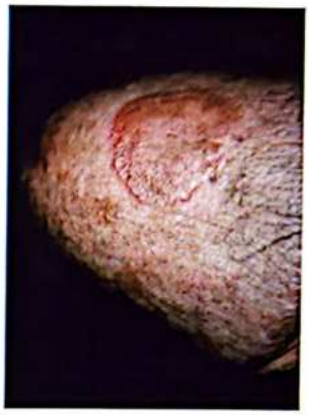
- Whitish reticular lacy pattern on the mucosa
- Oral Lichen Planus can affect another mucosa also such as genital or rectal mucosa.

Variants in oral mucosa:

- Reticular (most common),
- Erosive
- Atrophic
- Ulcerative.
- Ulcerative is a premalignant condition.



Annular LP



- Ring like region on glance with central clearing.
- Can also be present on the other parts of the body,
- Morphological variant.

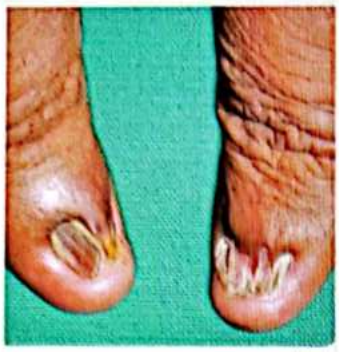
Trachyonychia:

- Rough, dry, brittle nails

- Longitudinal ridging of nails (most common finding).
- The nails look rough and there is dryness known as Sand blasted nails.
- Trachyonychia means rough, dry, brittle nails.
- Lichen Planus is one of the causes of Trachyonychia.

Pterygium:

- Most specific finding



- Pterygium is the wing shaped extension of the proximal nail fold on the nail plate.
- It causes tent-like appearance of the nail which is known as the tenting of the nail.

Cicatricial / Scarring alopecia:


- Lichen Planus is an important cause of Scarring alopecia
- The hair follicles are destroyed.
- Scarring occurs with no growth of hair.
- This is irreversible.

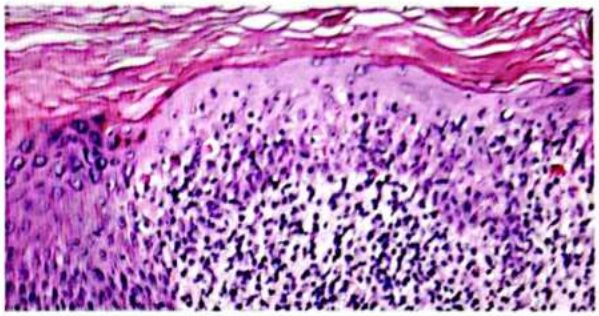
ther Types of Lichen Planus 00:12:22

- **Actinic Lichen Planus**
  - Associated with solar exposure
  - Seen in photo exposed areas.
- **Hypertrophic Lichen Planus**
  - Lichen Planus is thickened. (Appears Hypertrophic)
- **LP pemphigoides**
  - Combination of Lichen Planus and over that we get pemphigoides regions.
- **Annular Lichen Planus**

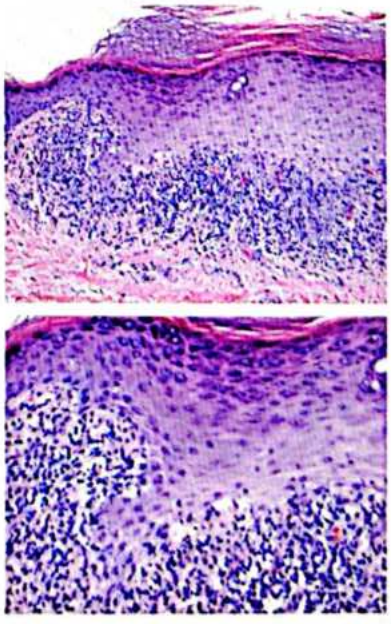
n Tuberculosis: 00:12:55

- Lichen Scrofulosorum and Lichen Amyloidosis.
- These are not Lichen Planus.
- Lichen is something which is Purplish or has a Lichenified or lichenoid histology.

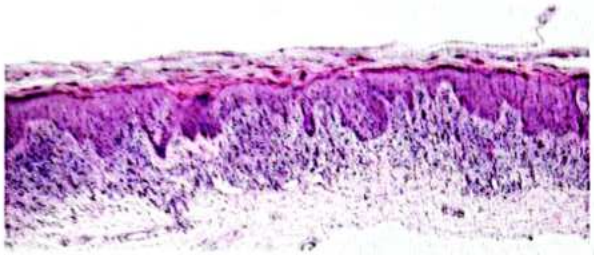
Histopathology 00:13:49



- In Stratum Corneum:
  - Hyperkeratosis (increased thickening)
- In Stratum Granulosum:
  - Hypergranulosis. And this is typically focal, wedge shaped hypergranulosis.
- In Starting Spinosum:
  - Acanthosis is seen.
- The most important changes occur in Stratum Basale.
- The infiltrate targets the Stratum Basale layer.
- Basal Cell Degeneration is seen.
- Keratinocyte turns into apoptotic bodies (undergoes apoptosis) which are called civatte bodies.
- Melanin (dendritic melanocytes) also targeted.
- They release their melanin which falls into the dermis.
- Leads to melanin incontinence.
- Melanin is engulfed by Macrophages forms melanophages.
- The rete ridges are saw toothed.
- Max Joseph Space: Space which is created between epidermis and dermis by the infiltrate of lymphocytes.
- The Basale cell Degeneration and the infiltrate in dermis are together referred to as Interface Dermatitis.



- In the first image, it is Hyperkeratosis.
- Then the band-like infiltrate around the epidermal junction.
- In the second image, it is Focal wedge shaped Hypergranulosis.



- Saw toothed rete ridges.

Treatment 00:19:46

- Treatments we use in Lichen Planus can be:
- **Tropical:**
  - Corticosteroids
  - Calcineurin Inhibitors (such as Tacrolimus, Pimecrolimus).
  - Antihistamine
  - If oral mucosa is affected, you need to give local anesthetics.
- **Intralesional:**
  - For Hypertrophic lesion: Intralesional injections of Corticosteroid.

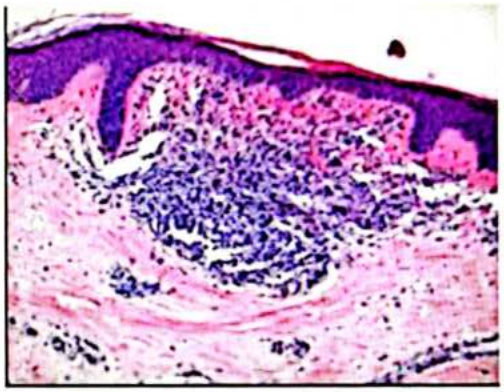
- **Systemic:**
  - Systemic treatment for more severe form.
  - Commonly used systemic agents are:
    - → Oral Corticosteroid
    - → Dapsone
    - → HCQS
    - → Azathioprine
    - → MMF (Mycophenolate mofetil)

All these can be used and does systemic therapy of Lichen Planus.

- **Phototherapy:**
  - NBUVB Phototherapy.
  - It is used especially in children who have a lot of regions in the body.

Lichen Nitidus: 00:22:32

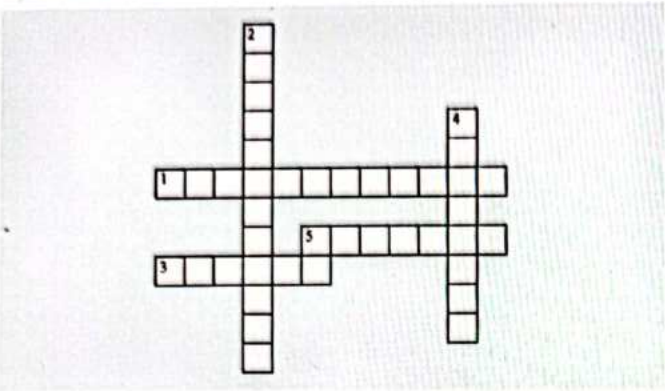
- It is usually seen in children.
- It is shiny, skin coloured Papules which can be present on elbows, knees and glans.
- They may or may not be symptomatic.
- The Papules on elbows, knees are also known as Lichenoid Papules.



- On Histopathology, you see that the rete ridge is engulfing a ball of inflammation.
- It appears like the epidermis is engulfing this Infiltrate.
- So, this is called a ball in claw appearance.
- Treatment is when you give Topical steroids, Topical Calcineurin.

CROSS WORD PUZZLES

Crossword Puzzle



Across

1. What does Lichen mean?
3. What type of Phototherapy is generally preferred in Lichen Planus?
5. What causes Lichen Planus?

Down

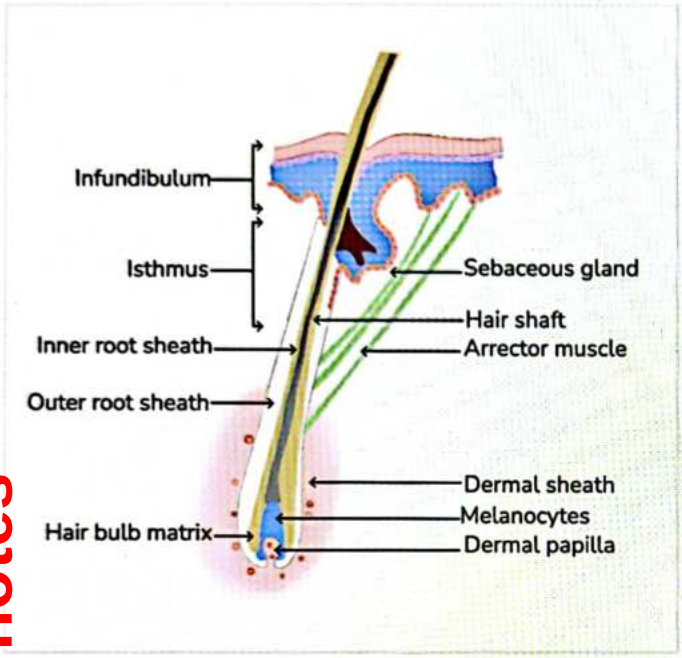
2. What type of disorder is Lichen Planus?
4. What does Lichen Planus affect ?

prince
ankitkarnawat9@gmail.com
9818635293

12 HAIR DISORDERS

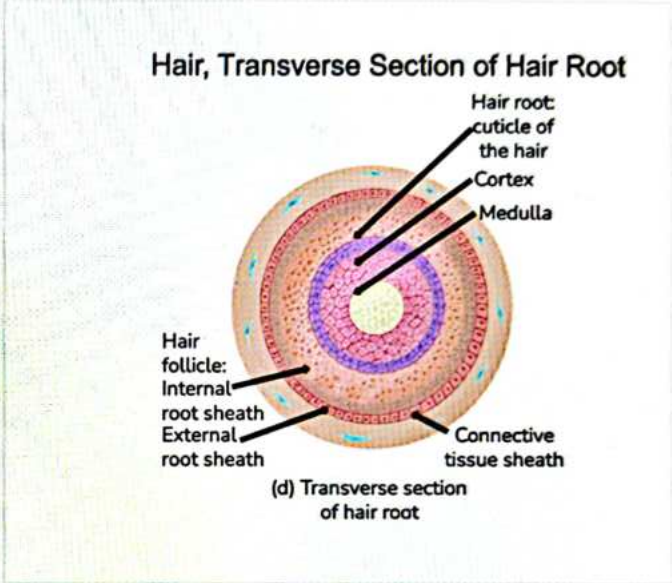
- Integumentary system is composed of
  - Skin
  - Appendages
- Appendages composed of
  - Hair
  - Nails
  - Different glands.

Structure of Hair 00:00:23



- Hair bulb
  - Present at the root of hair
- Epidermis and sebaceous gland
  - present at the level of hair follicle
  - Arrector Pili Muscle, present below the attachment of sebaceous follicles.
  - It attaches from the hair follicle from one end and the lower part of the epidermis at the other end.
- Based on their attachment, hair follicle gets divided into two parts:
  - Upper - Hair strand part
  - Lower - determines the type of hair.
  - Lower part keeps changing.
- Upper part: (two parts based on the attachment of sebaceous glands)
  - Infundibulum: Above the attachment of sebaceous glands
  - Isthmus: Below the attachment of sebaceous gland
- Lower part
  - Bulb region
  - Supra bulbar region.
- Stem cells are located at supra bulbar region. This region is also known as Bulge.
- **Cicatricial alopecia**
  - Anything that causes damage to outer root sheath will cause alopecia and where the hair will not grow back is cicatricial alopecia
- **Non cicatricial alopecia**
  - Here, hair will grow back because stem cells are not damaged.

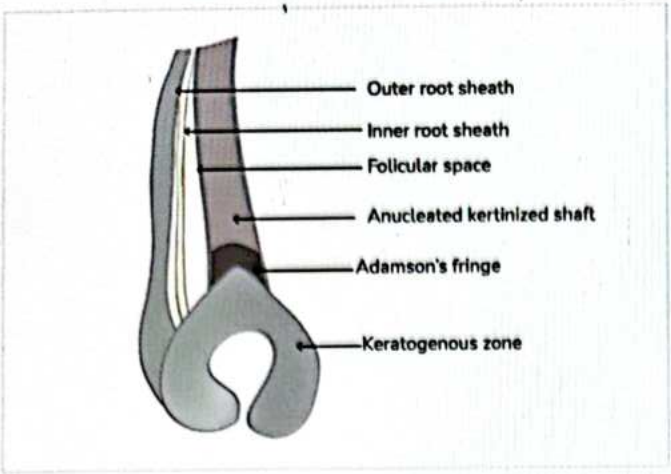
Hair, transverse section of hair root 00:04:51



- Cross section of hair: layers are as follow
  - Medulla - inner most layer
  - Cortex
  - Cuticle (part of inner root sheath) and on the outside, there is outer root sheath.
  - The inner root sheath has three parts:
    - → Henley's layer
    - → Huxley's layer
    - → Cuticle, cortex, and medulla.
    - → Mnemonic - hens hugs cute chicken mom's

Medullary index

- The thicker the hair, the thicker the medulla will be.
- Animal hairs are thicker than human hairs
- Medullary index = Diameter of medulla / diameter of whole shaft
- Humans: medullary index < 1/3
- Animals: medullary index > 1/2



- Adamson's fringe is just around the bulge, this area is known as keratogenous zone
- It differentiates between the keratogenous and non keratogenous parts
- Dermatophytes are keratinophilic, so dermatophytes will not enter this area because this will be limited by Adamson's fringe that marks the end of the keratogenous zone of the hair follicle.

Types of hair

- Three types of hair
  - Lanugo hair
  - Vellus hair
  - Terminal hair

Lanugo hair

- Seen in premature baby
- Soft non, medullated - thin
- Unpigmented hair
- In utero
- Premature babies

Vellus hair

- Hair on the face, forehead
- Soft
- Non medullated
- Less pigmented
- <2cm long
- Till adolescent

Terminal hair

- Coarser hair
- Hair on bearded area, pubic area
- Medullated
- Pigmented
- Longer
- Present in androgen-dependent areas because it has hormones.

Hair Cycle 00:12:43

- Growth phase is followed by the transition phase which is followed by the falling phase.
- This is the continuous cycle of growth.
- Growth phase - Anagen hair
- Transition phase – Catagen hair
- Falling phase – Telogen hair
- When entering the Telogen phase, the club root shape is formed.
- When hair shades off, it will go again to anagen stage.

Anagen	Catagen	Telogen
Growth phase	Transition phase	Falling phase
Lasts for 3 years	Lasts for 3 weeks	Last for 3 months
Is responsible for length of hair		Is responsible for daily hair fall
85-90% of total hair	1%	10%

- That is why we have 50-100 hairs falling per day.
- Anagen: Telogen = 9:1
- Hair growth rate: 1cm/month

Hair Disorders 00:17:28

- Loss of hair - Alopecia
- Excessive hair growth

Tests for hair

- Hair pull test -
  - Pull hair and see how many hairs are coming
  - If it is more than 6, it is insignificant
- Trichogram
  - Pull and take out certain hair and mount it to count no. of anagen and telogen based on the hair bulb
- Trichoscan
  - Like dermoscope
  - Computer based program where one can see hair growth in magnified form
- Scalp biopsy
  - Helps us to know different disorders.

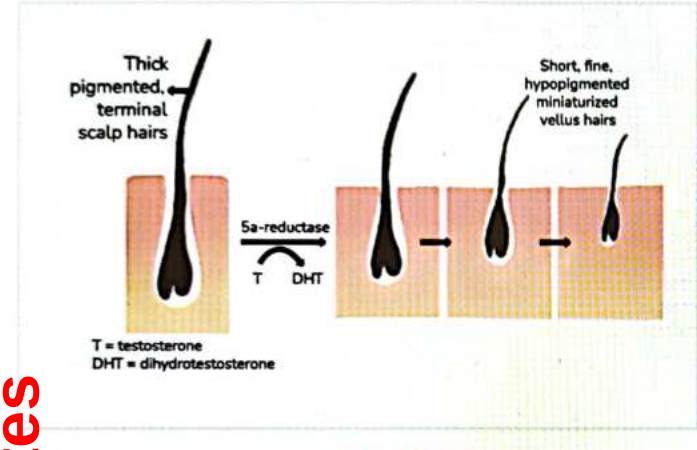
Alopecia 00:18:50

- Stem cells
  - When stem cells are affected, one gets Cicatricial/scarring area.
  - Here, the hair does not come back.
  - If stem cells are not damaged, one can get hair.
- Non Cicatricial/ non scarring
  - Also known as Non Cicatricial alopecia

- Patterned -
  → Androgenetic Alopecia
- Diffuse -
  → Effluvium
- Patchy -
  → Alopecia
  → Areata
  → Trichotillomania
  → Tinea capitis
  → Moth eaten alopecia

Androgenetic Alopecia

- Dependent on androgen
- In males - male patterned hair loss
- In female - female patterned hair loss
- These are non-genetic.
- Genetic susceptibility: polygenic inheritance



- Testosterone converts to dihydrotestosterone
  - The enzyme used is 5 alpha-reductase.
  - It reduces the anagen phase
  - Hair becomes short and causes miniaturisation of hair.

So, terminal hair is converting into vellus hair now.

In males, bitemporal recession happens - from temple and vertex balding happens.

If they progress, they will get little hair while keeping the vertex empty.

This **is male Androgenetic Alopecia**

- The vertex has no hair
- But little fringe of hair is present around the side because this area is occipital, which is not androgen dependent.
- Graded into I to VII sections and is given by Norwood-Hamilton

Refer Image 12.1

- In female patterned hair loss:
  - Widening of central parting.
  - There are three divisions of female Alopecia.
  - Type I
  - Type II
  - Type III
- This pattern was given by Ludwig
- Grade III looks like an air tree / Christmas tree pattern in females.

Management of these patient:

- Counselling - it requires long period treatment.
- Topical management
  - Minoxidil
  - Potassium channel opener
  - It causes Vasodilation (increase in blood flow of scalpy)
- 2% Minoxidil - female pattern hair loss
- 5-10% Minoxidil - male pattern hair loss
- Side effects of Minoxidil:
  - Contact Dermatitis
  - Headache
- This is FDA approved treatment.
- Systemic management
  - Finasteride - 5 alpha reductase inhibitor - type 1 & 2 given 1 mg/day
  - Dutasteride - type 1 & 2 inhibitor. Dose 0.5 mg/day
- For females
  - Oral anti-androgens, which can be cyproterone acetate, spironolactone, and flutamide.
- Surgical management:
  - Hair transplantation
  - Transplant hair from occipital area because it is not androgen dependent
  - When transplantation from the donor area, it retains the quality of the donor area. So if it is androgen dependent, it will remain androgen dependent.

Effluvium 00:31:57

- Diffused hair fall
- If Effluvium affects anagen - Anagen Effluvium
- If Effluvium affects telogen - Telogen Effluvium
- When a patient is given Chemotherapy.
  - Chemotherapy acts against all the mitotically active cells.
  - So, in the hair, it will act against anagen and thus patient losses all the hair.
- When all the hair is gone - anagen Effluvium
- Telogen Effluvium
  - Physical stress in the form of surgery, trauma, or prolonged illness.
  - Prolonged illness can be Dengue, malaria, Covid, TB, chikungunya, pregnancy.
  - When it happens in pregnancy, we call it Telogen gravidarum.
  - So, when stressed, Anagen prematurely converts into Telogen.
  - Hair in the telogen phase stays for three months and then hair fall happens.

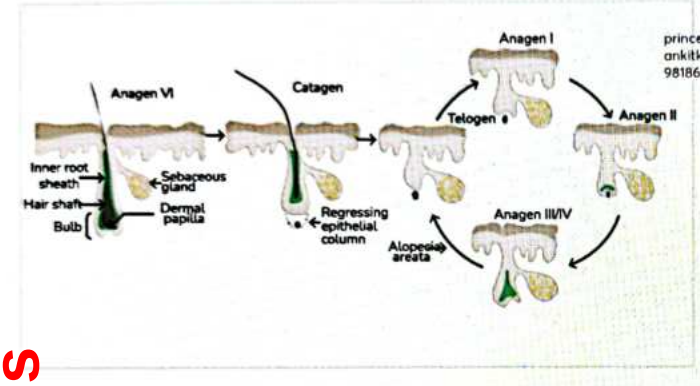
- Chronic TE:
  - Iron deficiency, thyroid, crash diet or malnutrition.
  - If repeated insults, patients will face hair fall for many years.
  - Then, there will be subsequent thinning of the ponytail.

Alopecia Areata 00:37:21

- It is a patchy kind of hair fall
- It is a non-cicatricial alopecia
- It is an autoimmune T-cell mediated disorder where the T-cell acts against hair follicles.

Association

- Other autoimmune disorder
  - Type 1 DM
  - Thyroid disorders
  - Pernicious anemia
- There are certain HLA association too



- The Anagen are being targeted
- T cell starts acting against the anagen hair root.
- The hair compensates and converts the anagen hair into the telogen hair.
- The telogen hair falls and goes back to the anagen hair.
- This happens repeatedly, and eventually, the person is left with a bald patch.
- Single or multiple patches of hair loss are seen in this condition

Refer Image 12.2

- These patches are smooth
- No scaling, and no itching, no redness and are well defined
- Also called Pelade

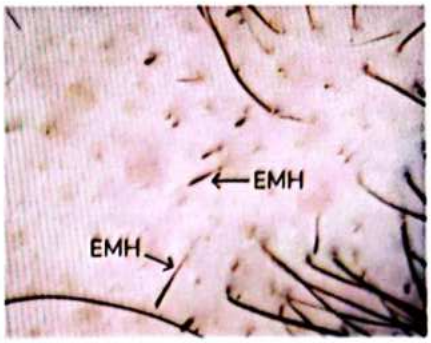
Inflammation affects the pigment in the hair and does not affect the grey area

- The person will keep on losing hair
- But the grey hair will stay there.
- All the grey hairs are retained in a person suffering from Alopecia Areata.
- It is called Turning grey syndrome
- Alopecia Areata does not affect grey hair.

It is asymptomatic.

Exclamation mark hair

Recreate this image or confirm its presence



- Broken hair present on the periphery of the lesion.
  - These hairs will appear tapering down and with a little bulbar area below.
  - These exclamation mark hairs are seen on the periphery of the patches of Alopecia Areata.
- Variants:
  - Complete hair loss from all over the body - Alopecia Universalis

Refer Image 12.3

  - Complete hair loss over scalp - Alopecia Totalis
  - Band of Alopecia Areata occurs called Ophiasis pattern and forms an O on the scalp.
    In Sisaipho, only an occipital band is left, and all the hair is gone.
- Sometimes the Alopecia Areata does not affect the scalp and may have associated involvement with .

Refer Image 12.4

  - Eyebrows
  - Eyelashes
  - Beard area
- Nail findings in the patients of Alopecia Areata (AA)

Refer Image 12.5

  - Pits
    → In psoriasis pits were deep random, and irregular
    → Here the pits are superficial, geometric and they are not random (regular)
  - Longitudinal ridging or Trachyonychia - all 20 nails may be affected.
- In 80 per cent of cases AA spontaneously resolves.

Differential diagnosis 00:46:52

- Tinea capitis
  - Also present with non-cicatricial, patchy hair fall, non-scarring alopecia, broken off hair - This is common
  - Scaling, itching, KOH positive (absent in alopecia areata)
  - It does not have exclamation mark hair

- Moth eaten alopecia
  - Associated with syphilis in the secondary stage
  - It will not be a standalone finding, and the patient will have other symptoms including VDRL positive
- Trichotillomania

Treatment

- Spontaneous resolution is an option

Topical treatment

- Corticosteroids - It can be used topically
- Intralesional - Intralesional steroids (ILS) is the treatment of choice.
- Triamcinolone or kenacort of the strength 10 mg/ml dilute it 1:4 or 1:2 and it is injected intradermally in the Alopecia Areata.
- Contact immunotherapy
  - Like simulating a contact irritant reaction
  - Three agents are used
    - → Di nitro chlorobenzene
    - → Squaric acid dibutyl ester
    - → Diphencyprone
  - A little concentration is applied on the patch, and these agents act as contact irritants which generate an irritant reaction and divert the T cells here, and they free the hair follicle, and they improve.

Oral agents

- Systemic therapy - for extensive alopecia areata
  - Levamisole
  - Cyclosporine
  - Oral mini pulse therapy - Betamethasone or methylprednisolone given twice a week to minimise the side effect.
- JAK kinase inhibitor called tofacitinib is a new agent increasingly being used.

Trichotillomania

Refer Image 12.6

- It is Differential Diagnosis of alopecia
- It is a psychiatric disorder and OCD of pulling hair.
- It is classified into DSM 4 criteria
- History will not be given by patients, some others will give hints about psychiatric issues.
- Often seen in adolescence or young adults.
- Site:
  - Happens on an accessible site which can be fronto parietal area.
  - Patchy hair loss.
  - This patchy hair loss will be broken hairs of variable lengths.
  - Happens on the dominant part of the scalp.
- Sometimes, because of the trauma, one may see perifollicular haemorrhages.
- Patients may also eat hairs which leads to trichophagia or trichobezoar obstruction.
- It is also called the sign-friar tuck sign.
- On skin biopsy
  - Empty cast, p/f hemosiderin
- Treatment:
  - Cognitive behavioural therapy (CBT)
  - Antipsychotic Medicines like SSRI, paroxetine, or NAC - N-acetyl cysteine.

Cicatricial or scarring alopecia 00:57:40

- Damaged Stem cells
  - Leads to permanent hair loss.
  - The area will look scarred.
- Counselling for patient
  - The purpose of counselling would be to avoid new patches of hair loss.
- Role of scalp biopsy
  - To know what kind of infiltrate it is and how deep it is to provide treatment to the patient.

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

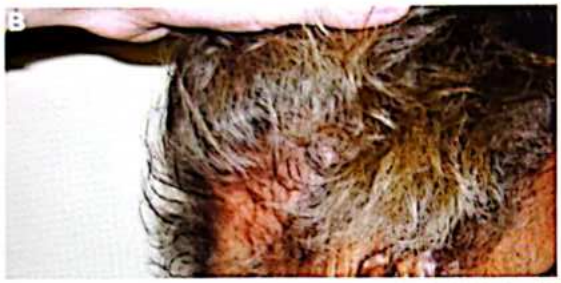
Neutrophilic primary Cicatricial alopecia

- Folliculitis decalvans
- Dissecting cellulitis/folliculitis (perifolliculitis abscedens et suffodiens)

Mixed Cicatricial alopecia

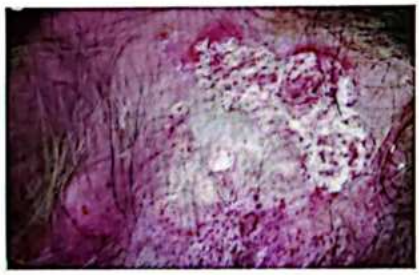
- Folliculitis (acne) keloidalis
- Folliculitis (acne) necrotica
- Erosive pustular dermatosis

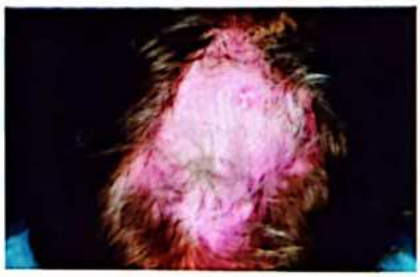
Follicular LP



- It is called Lichen Plano pilaris.
- It is affecting the hair or follicular lichen planer.
- There will be violaceous lesions and plaques, present in the perifollicular distribution.
- Itching, scaling present
- A patch of cicatricial alopecia will be left behind.

Lupus erythematosus





- Another cause of cicatricial alopecia.
- SLE or ACLE causes non scarring alopecia.
- DLE/ chronic cutaneous LE causes scarring alopecia
  - Scarred patches of alopecia which are associated with scaling and follicular plugging and can be seen on scalp.

Pseudopelade of BROCQ

- Smooth patches due to scarring alopecia.
- It is an idiopathic condition, no inflammation, no scaling, no erythema present in the biopsy report.
- The only thing visible is patches of cicatricial alopecia which is spreading.
  - Footprint in snow appearance
- It is chronic and slowly progressive.

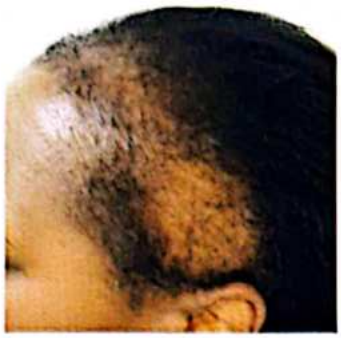
Folliculitis Decalvans / Tufted Folliculitis

- Neutrophil disorders that can lead to Cicatricial alopecia.
- Kind of deep folliculitis caused by bacteria.
- Pustules are found which go deep and damage the stem cells and cause scarring alopecia.
- Hair tufts are formed.
- It heals with boggy scars.
- Treatment - antibiotics

Dissecting cellulitis

- Deep folliculitis
- Causes cicatricial alopecia.
- This is a part of a triad - follicular occlusion has three features.
  - Dissecting cellulitis of the scalp
  - Acne conglobata
  - Hidradenitis suppurativa

Traction Alopecia



- Because of traction there is excess force applied on the hair follicle, leading them to causing Traction Alopecia
- It is a hairstyle disorder.
- Hair is damaged due to constant pressure of force applied to them.

Excessive Hair Growth 01:05:43

- Hypertrichosis - if it happens on the body.
  - Seen commonly congenitally over the knee.
  - Can be seen with use of topical steroid.

Hirsutism - when hair growth happens on androgen dependent areas.

Hirsutism

- Growth of unwanted hair on androgen dependent sites
- Female - growth of terminal hair in beard area
- Feature of female
- Vellus hair is converted to terminal hair

Causes

- Androgens are being produced by two sources.
  - Ovaries
  - Adrenals
- If there is PCOS or ovarian tumour or any cause which is stimulating the ovaries one can have Hyperandrogenism.
- Adrenal glands produce more androgen if there is
  - Congenital adrenal hyperplasia (CAH)
  - Virilizing tumours
- Sometimes there is idiopathic and no symptoms are found and perfect androgen levels.
- SAHA syndrome has four components
  - Seborrhoea
  - Acne
  - Hirsutism
  - AGA
- There is a scoring which has been given by Ferriman and Galway

Investigation

- PCOS - LH, FSH, DHEAS
- Adrenals - Level of 5 DHT is seen, look for thyroid, look for prolactin
- All these hormonal tests are done in a patient of Hirsutism to rule out the cause of Hyperandrogenism.

Treatment

- Topical agent - Eflornithine
  - Not very effective
- Antiandrogen
  - Spironolactone
  - OCP's - if patient has PCOS
  - Dutasteride
  - Flutamide
  - Cyproterone acetate

- Physical
  - Hair removal lasers
    → Diode and long pulse ND Yag

Image 12.1

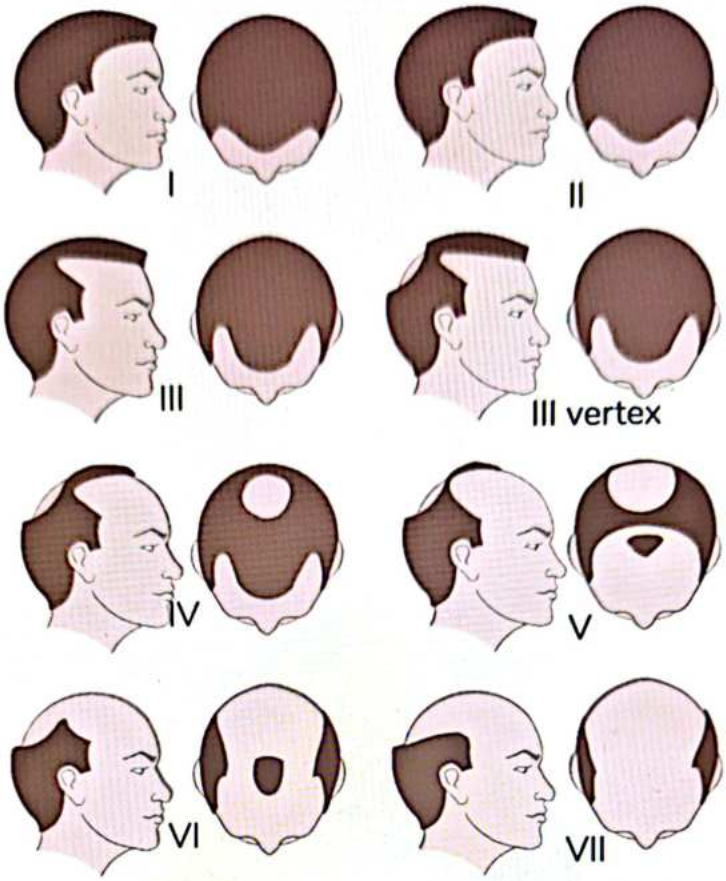
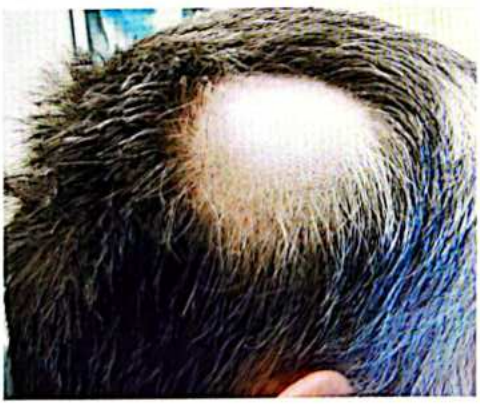


Image 12.2



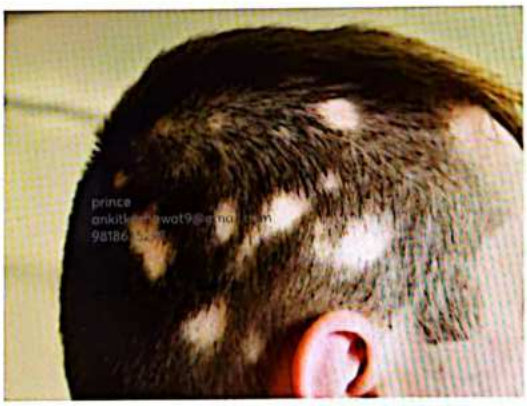


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Image 12.3

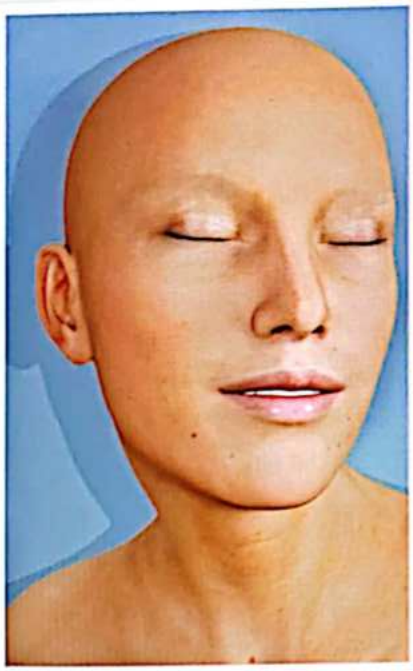


Image 12.4

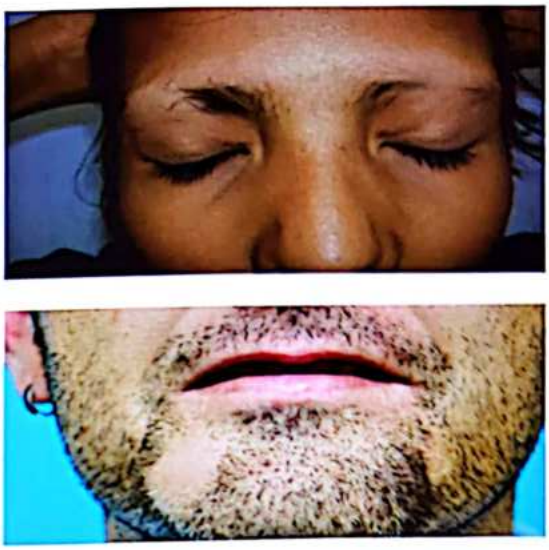


Image 12.5

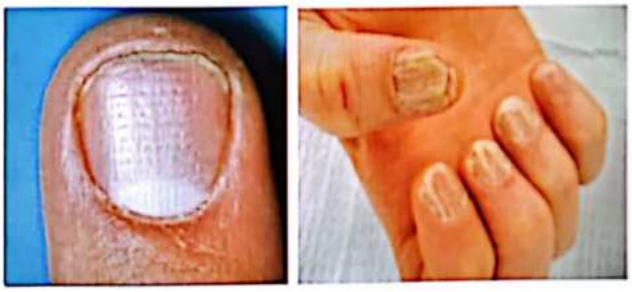
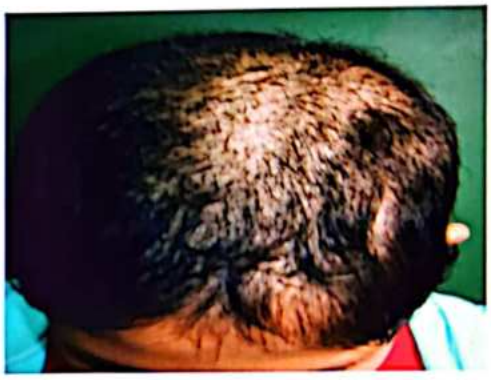
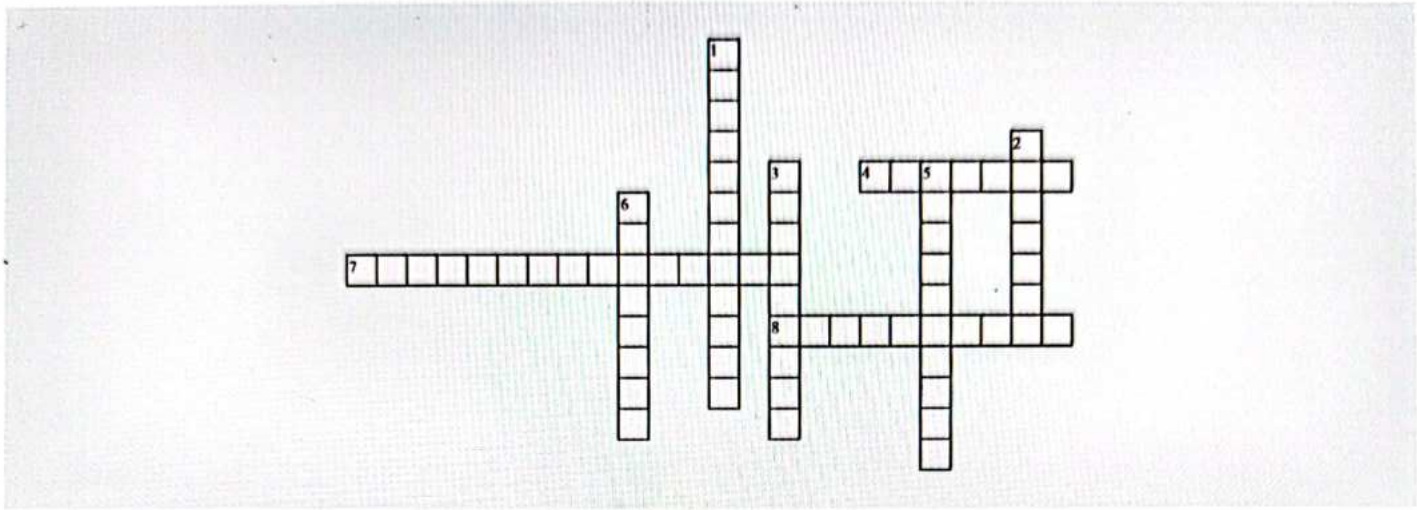


Image 12.6



CROSS WORD PUZZLES

Crossword Puzzle



Across

4. Which phase of hair cycle is responsible for daily hair fall
7. Complete hair loss over scalp
8. Computer based program where one can see hair growth in magnified form

Down

1. A type of hair present in androgen-dependent areas
2. Inner most part of the hair
3. Hair growth that happens on androgen dependent areas
5. A type of hair seen in premature baby
6. Damage to outer root sheath causes

prince
ankitkarnawat9@gmail.com
9818635293

13 NAIL DISORDERS

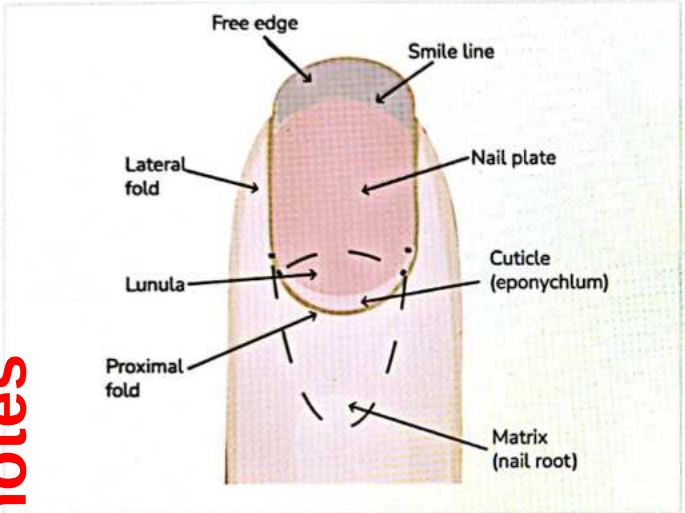
Nail 00:00:10

- Derived from ectoderm
- Composed of Hard keratin

- Keratin is of two types
  - Soft keratin
  - Hard keratin
- It protects nail bed
  - An important structure

- Growth rate
  - 3mm/month - finger
  - 1mm/month - toe

Parts of Nail 00:00:43



- Nail plate
- Lateral nail folds
- Proximal nail fold
- Cuticle- A curved part of the skin coming into the nail
  - Connects the proximal nail fold to the nail plate

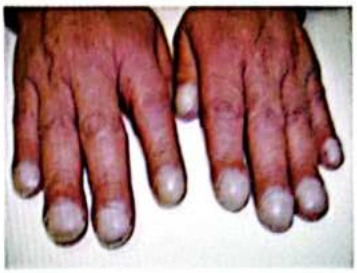
- Lunula - The semi - circular portion with different colour with the rest of the nail
  - Slightly pink or red
  - It is called the window to nail matrix.

- Nail matrix - Located below the proximal nail fold and lunula.
- Nail bed - The skin present beneath the nail plate.
- Hyponychium - The nail fold of skin which turns around, the distal end of the skin.

Refer Image 13.1

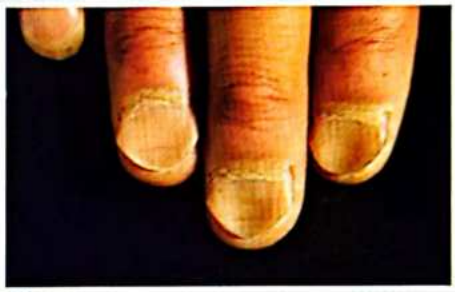
Nail Changes in Systemic Diseases 00:03:41

Clubbing 00:03:48

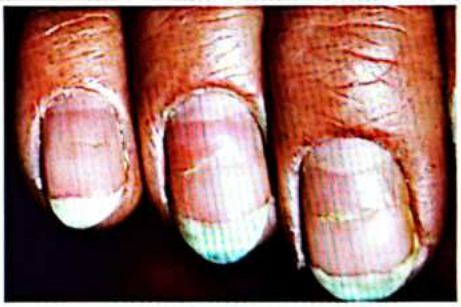


- Increased transverse and longitudinal curvature of the nail plate
- Idiopathic
- Hereditary
- Most common acquired cause is pulmonary disorders.

Koilonychia 00:04:30



- Spoon shaped nails
- Seen in
  - Iron deficiency
  - Surgery
  - MI

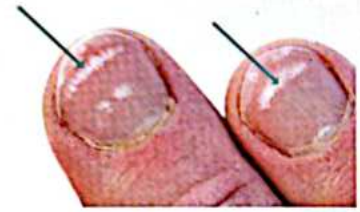


- Sometimes, transverse grooves are seen - Beau's lines
  - Indicates- Temporary arrest in nail production
  - It helps to calculate duration of insult
  - By measuring the distance, it will helps to asses when the insult, illness, MI or chemotherapy was given.
  - Time for insult lasted or how long back is calculated by measuring the width.

Leukonychia

00:06:26

- White spots or white lines on the nails.
- It can be
  - True leukonychia - due to matrix involvement
  - False leukonychia - due to nail bed involvement



A. **True leukonychia** are of two types

Transverse lines

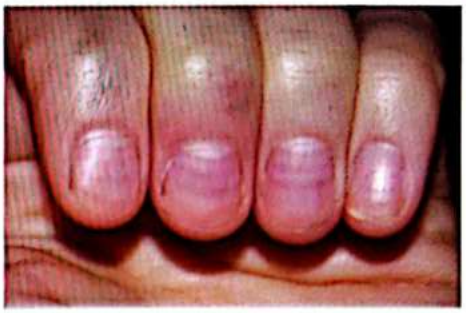
- Called as mees lines
- Seen in
  - → Arsenic poisoning
  - → Chemotherapy

Punctate lines

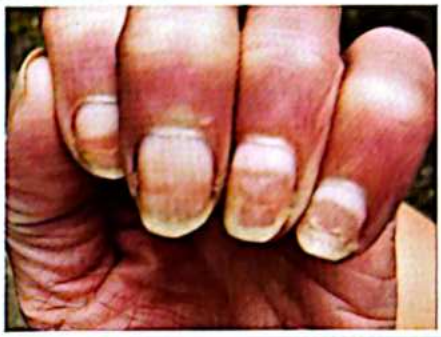
- Dotted spots
- Seen in
  - → Trauma
  - → Alopecia areata

B. **False leukonychia** are of three types

1. **Muehrcke's paired white bands**
   - Seen in hypoalbuminemia
   - Paired white lines are seen



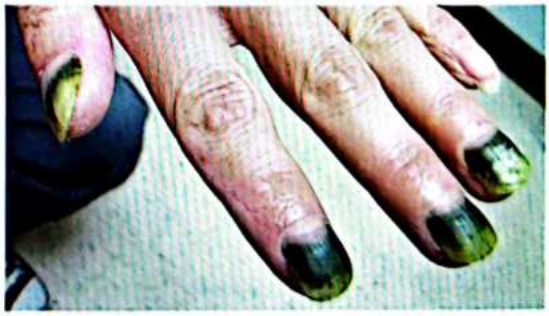
2. **Terry's lines**
   - Seen in cirrhosis, CHF and DM.
   - Nail is white proximally and normal distally

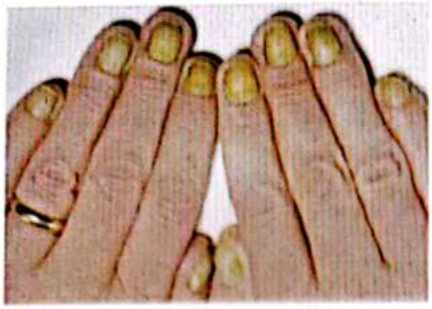


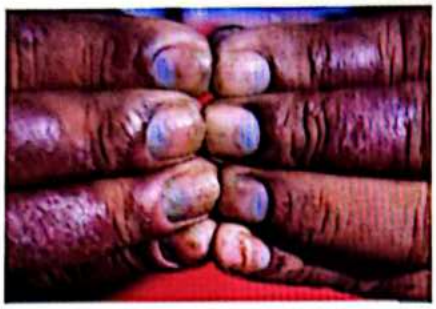
3. **Lindsay Nails or half and half nails**
   - Seen in chemotherapy and chronic renal failure
   - Proximal white zone and distal brownish sharp demarcation

Chromonychia

00:09:20



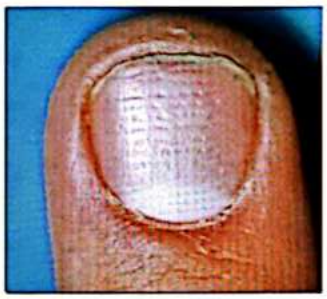




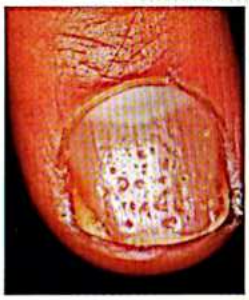
- Nails are coloured.
- Green nail syndrome- In pseudomonas infection
- Blue lunula - SLE
- Yellow nails- in pulmonary edema.

Pitting 00:10:15

- Regular pitting - Alopecia areata
- Irregular pitting - Psoriasis



Pitting



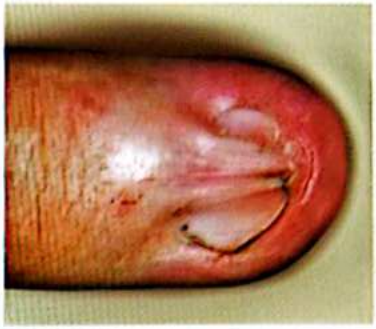
Psoriasis

Brachyonychia 00:10:36

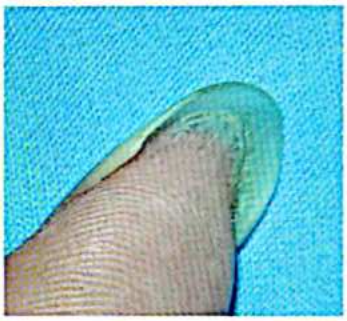
TND: Twenty nail dystrophy

- Sand blasted nails
- Idiopathic
- Seen in-
  - Alopecia areata; most common
  - Lichen planus
- Usually seen in children
- Self resolved or else steroids are given

Pterygium 00:11:35

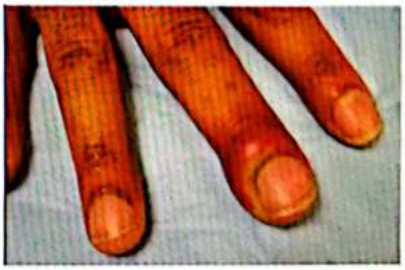


- Seen in Lichen planus
- The wing extension of proximal nail fold on the nail plate
- **Inverse pterygium**



  - Hyponychium is going and attaches to the nail plate distally.
  - Seen in
    - → Connective tissue disorders
    - → Trauma
    - → Scleroderma
    - → LE
    - → Raynaud's

Paronychia 00:12:15

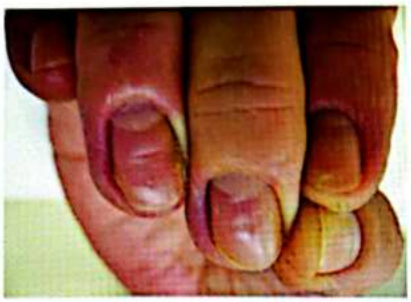


- Inflammation of nail folds
- Cuticle gets damaged and causes inflammation.
- Commonly seen in wet works, house wives, maids.
  - Due to more soaking in detergents

Acute Paronychia

- Severe swelling
- Redness
- Pain and pus is seen.

Chronic Paronychia

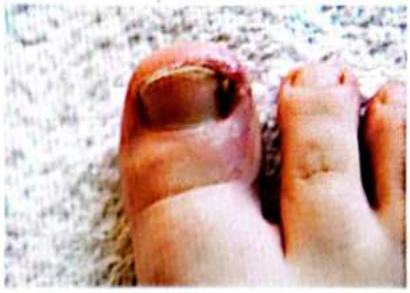


- No cuticle
- There is a gap between the proximal nail fold and nail plate.
- It is Eczematous in nature

Management of both Acute and Chronic Paronychia

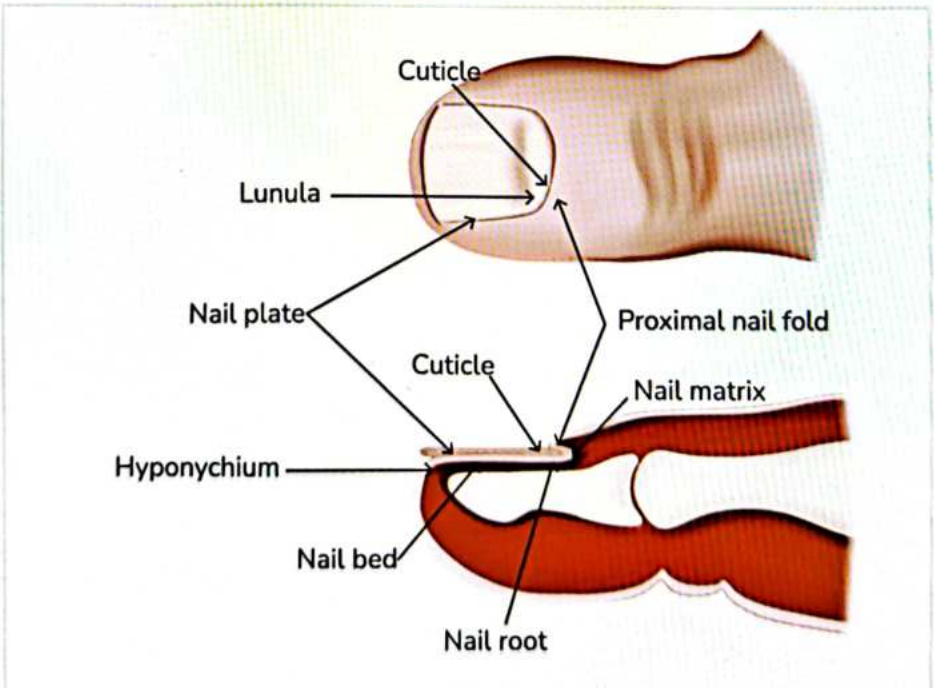
- Acute Paronychia is managed by antibiotics and painkillers
- Chronic paronychia patients must follow precautions
  - Wearing gloves
  - Avoid wet work
  - Not to soak in detergents
  - Topical steroids must be given.

Ingrown Toenail 00:14:50



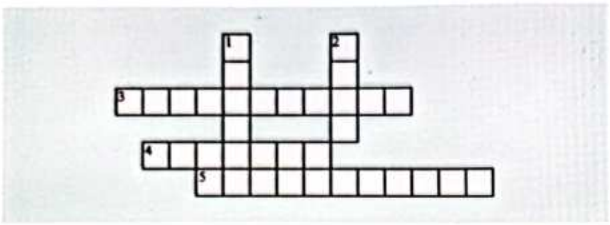
- If nails are cut at an angle - instead of growing straight, it grows different and nail gets into the lateral nail fold.
- Leads to inflammation of proximal nail fold.
- Leads to pain, infection and pus discharge
- Antibiotics, painkillers are given.
- Most importantly tell the patient to cut the nail straight.

Image 13.1



CROSS WORD PUZZLES

Crossword Puzzle



1. The semicircular portion with different colour with the rest of the nail
2. _____ is the dead structure.
3. The nail fold of skin which turns around, the distal end of the skin is called _____.
4. The skin present beneath the nail plate is called _____.
5. Nail Disorders is composed of _____.

prince
ankitkarnawat9@gmail.com
9818635293

14 DISORDERS OF GLANDS

Types of Glands in Skin

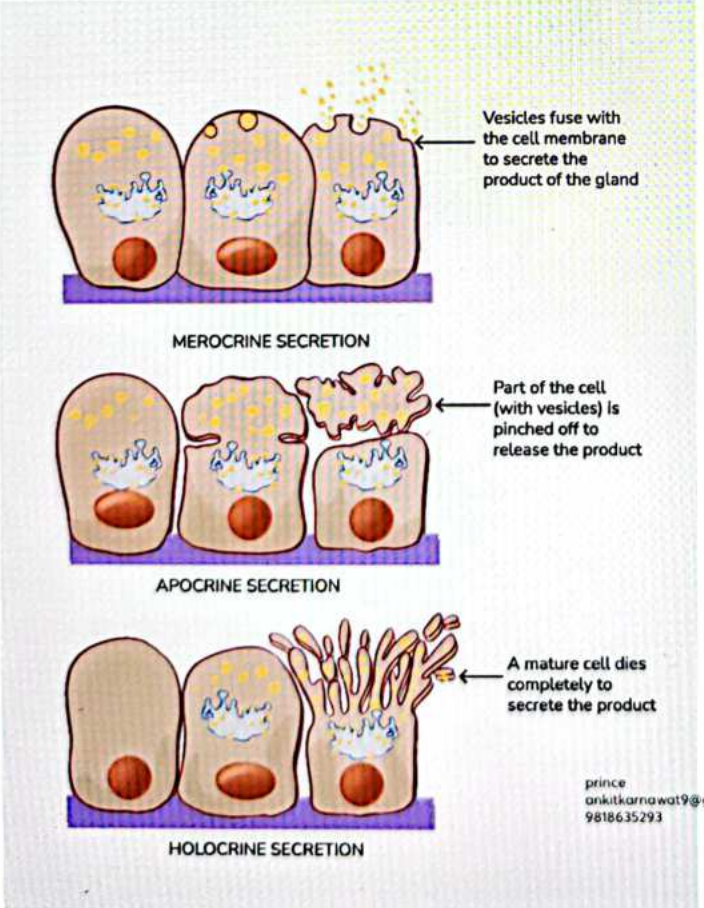
00:0013

- Sweat glands
  - Eccrine
  - Apocrine
- Sebaceous glands

Types of Skin Gland Secretions

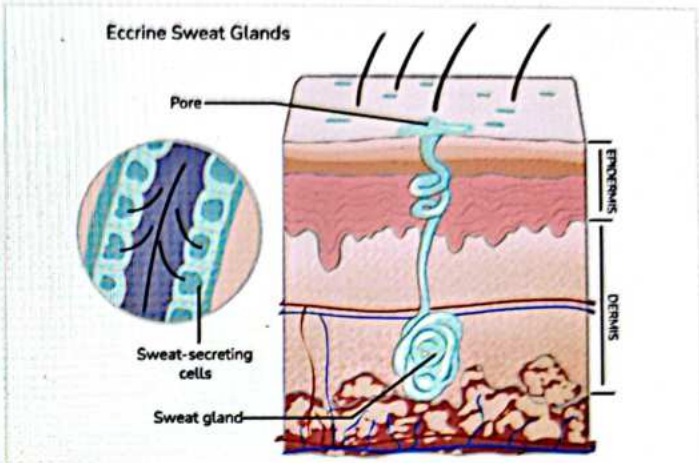
How Do Glands Secrete?

Gland	Secretion Name	Description
Eccrine	Merocrine secretion	Vesicles are formed and fused to cell membrane
Apocrine	Apocrine secretion	Apical part of gland is pinched off
Sebaceous	Holocrine secretion	Whole gland cell ruptures and dies

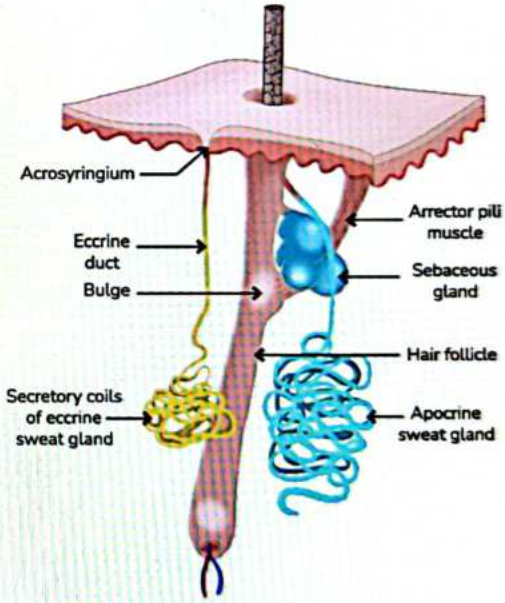


Location and Structure of Skin Glands

00:0138



PILOSEBACEOUS UNIT WITH APOCRINE AND ECCRINE SWEAT GLANDS



Eccrine gland

- Has two coiled structures
  - One below dermis - sweat gland
  - Another in epidermis - Acrosyringium
- Opens directly into the skin

Apocrine gland

- Single highly coiled structure
- Opens into hair follicle

Sebaceous glands

- Bulge structures
- Opens into hair follicle

Eccrine vs Apocrine

00:0853

Criteria	Eccrine Gland	Apocrine Gland
Location	All over body (more on palms and soles)	Axilla, groin, mammary area, umbilicus
Type of Secretion	Merocrine	Apocrine
Opening	Directly into skin	Into hair follicle
Innervation	Cholinergic	Adrenergic
Secretion	Watery	Viscous
Role	Sweating/ Thermoregulation	Body odor (no role in thermoregulation)
Onset of Activity	By birth	Puberty (Androgen dependent)

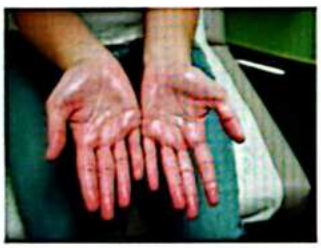
Eccrine Gland Disorders

00:07:28

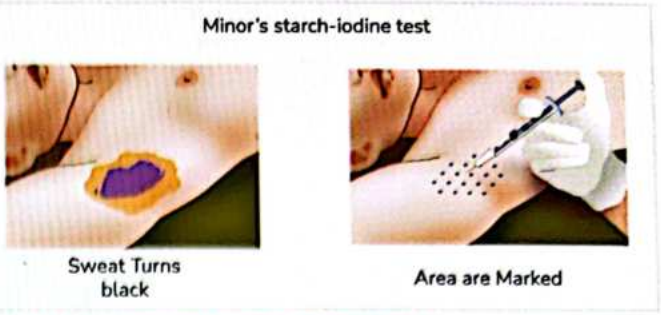
- Hyperhidrosis - Over sweating
- Hypo/Anhidrosis - Less/No sweating
- Miliaria - Blockage of Acrosyringium
- Neutrophilic Eccrine Hidradenitis - Seen in chemotherapy patients (characterized by painful red eruptions on trunk)

Hyperhidrosis

00:08:27




- **Cause:** Anxiety, stress, temperature differences
- **Locations**
  - Palms - Palmoplantar
  - Axilla - Axillary (usually in post pubertal individual)
- **Test for Hyperhidrosis**



- Iodine is painted on the affected area
- Starch is sprayed
- Black coloration is seen
- Black dots are formed (indicating hyperhidrosis)

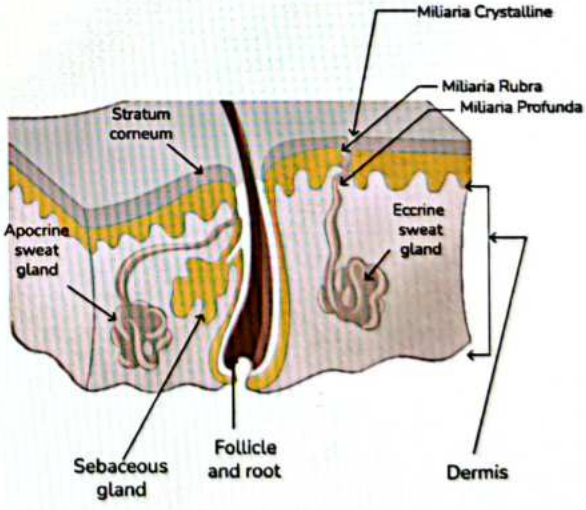
- **Treatment**
  - Aluminum hexachloride
  - Formaldehyde
  - Glutaraldehyde
  - Oral anticholinergic
  - Iontophoresis
  - Botulinum toxin
  - Surgery

Iontophoresis

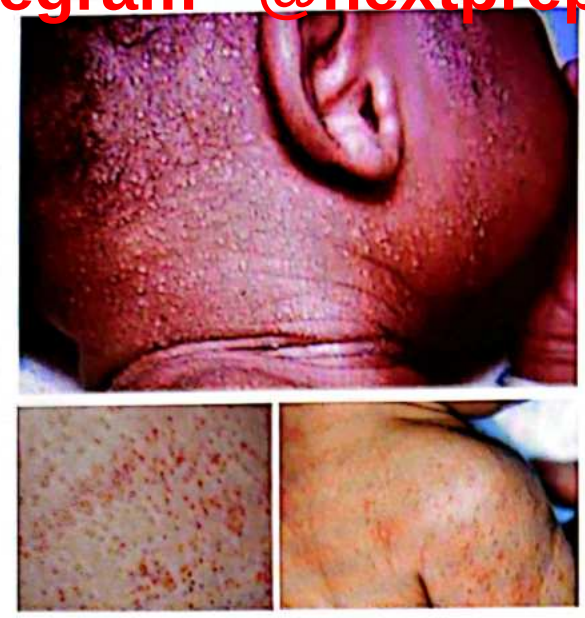
- Two bowls are water are placed
- Patient is allowed to put his hands into the bowls
- Current is passed to improve hyperhidrosis

Miliaria (IMP)

- Most common type of sweat gland disorders
- **Reason:** Blockage in the Acrosyringium
- **Types**



- Miliaria Crystallina - At levels of stratum corneum
- Miliaria Rubra - At levels of stratum spinosum
- Miliaria Profunda - At levels of basal layer (dermo-epidermal junction)

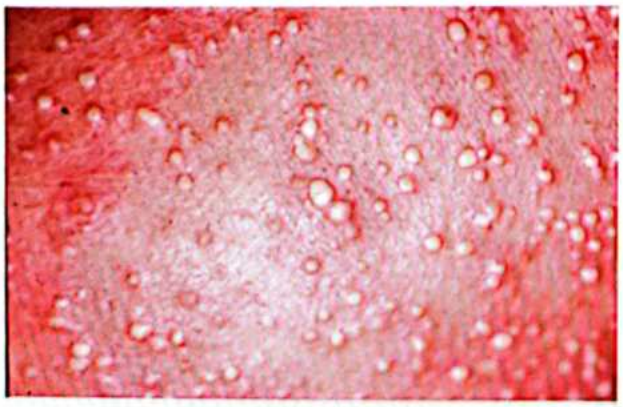


Miliaria Crystallina	Miliaria Rubra	Miliaria Profunda
• Usually, infants • Superficial • Clear fluid filled thin vesicles which ruptured easily	• **Other name:** Prickly heat • Commonest • Itchy or discomforting • Erythematous papules can be present on Trunk , arms or all over the body.	• Deeper • Nodular

- **Treatment**
  - Cooling advice
    - → Calamine lotion can be given
    - → Manthol
    - → Reassure to reduce the temperature

To Remember

- Miliaria rubra may get infected with staphylococcus aureus then it is called Miliaria pustulosa
- Pustules are seen.



Apocrine Gland Disorders

00:14:50

- Bromhidrosis
- Chromhidrosis
- Fox fordyce disease
- Hidradenitis Suppurativa

Bromhidrosis vs Chromhidrosis

00:15:13

Bromhidrosis	Chromhidrosis
• Brom - Odor • Abnormal sweat odor • Production of volatile chemicals by the actions of bacteria on apocrine sweat	• Chrom - Color • Vividly colored apocrine sweat (blue, yellow, green)
• **On set:** Pubertal individuals • **Management** o Dietary advice (avoid garlic) o Deodorants	• Result from the production of lipofuscin in apocrine sweat • **On set:** Pubertal individuals

Fox Fordyce Disease/Apocrine Miliaria

00:16:45

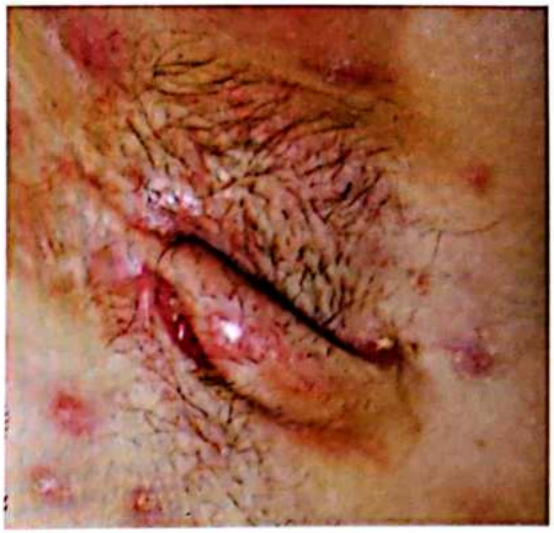
- **Reason:** Apocrine gland is blocked
- **On set:** Puberty
- **Gender:** Female >> Male
- **Site:** Mostly Axilla
- **Clinical presentation**
  - Skin colored raised discrete papules
  - Itchy or discomfort

Hidradenitis Suppurativa (HS)

00:17:55

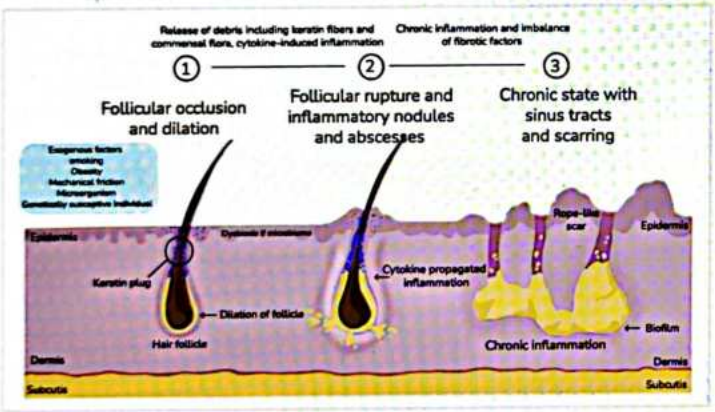
- Whole hair unit is affected, not only the apocrine gland
- Chronic inflammatory condition

- **Other name:** Acne inversa (seen at inverse locations to acne)
- **Site:** Axilla, groin, mammary, umbilicus
- **Age:** Pubertal age group
- **Predisposing factors**
  - Obesity
  - Smoking
  - DM
  - Insulin resistance
  - Metabolic syndrome
- **Associations**
  - Follicular occlusion triad (HS, acne conglobata, dissecting cellulitis of scalp)
  - **Tetrad:** Follicular occlusion triad + Pilonidal sinus
  - Crohn's disease

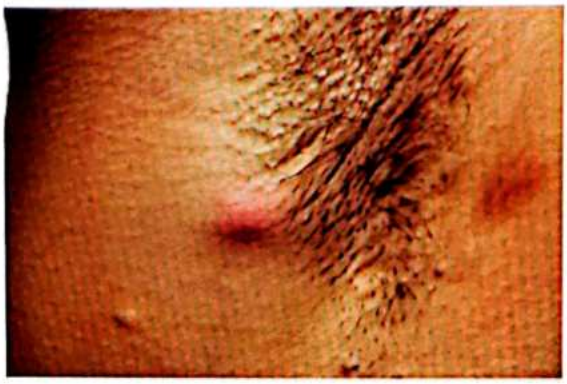


To Remember: Severity staging of HS was given by Hurley

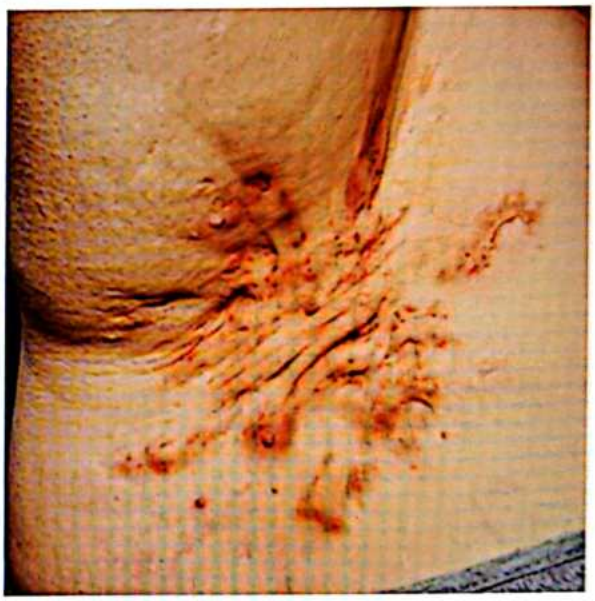
- Pathophysiology of HS
  - Follicular occlusion and dilation
  - Follicular rupture and inflammatory nodules and abscesses
  - Chronic state with sinus tracts and scarring.



- **Clinical presentation**
  - Polyporus Comedones (blocked sebaceous duct)
  - Inflammatory papules
  - Recurrent episodes
  - **If severe**
    - → Abscess
    - → Multiple discharge sinuses
    - → Heals with scarring (Bridging/ rope scars)



- Bridging/ Rope Scars: They are seen because of the sinuses formed, as they leave behind the bridge lines between the follicles.



Q1. Overweight diabetic male develops recurrent episodes of painful lesions at Axilla and Groin, what is your diagnosis?

Answer: Hidradenitis Suppurativa

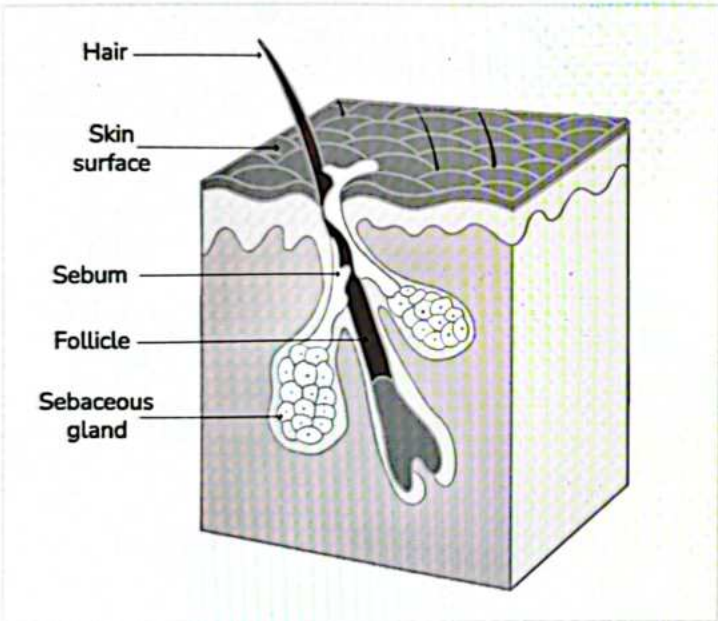
- **Criteria**

Essential criteria for a diagnosis of hideradenitis Suppurative to be made	
Typical lesions	Deep – Seated painful nodules, abscesses, draining sinuses, bridged scars and paired or multiheaded open pseudocomedones

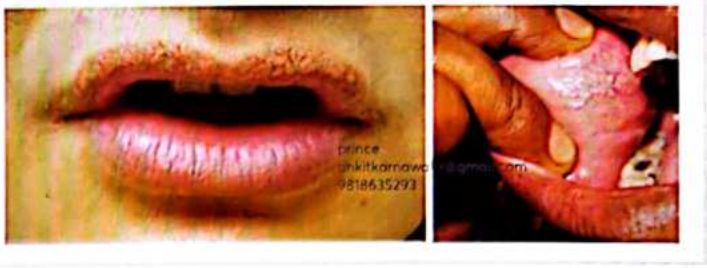
Typical topography	Axillae, groin, perineal and perianal region, buttocks, infra- and intermammary folds
Presence and recurrence of lesions	

- **Treatment**
  - **Advice:** Reduce weight, Diet modifications, quit smoking
  - **Analgesics:** For pain
  - **Antibiotics:** Doxycycline, Clindamycin
  - **Anti-inflammatory agents:** Dapsone
  - **Retinoids**
  - **Biologicals:** Adalimumab (TNF α inhibitor)
  - **Surgical therapy:** If HS is recurrent

Disorders of Sebaceous Glands 00:25:25



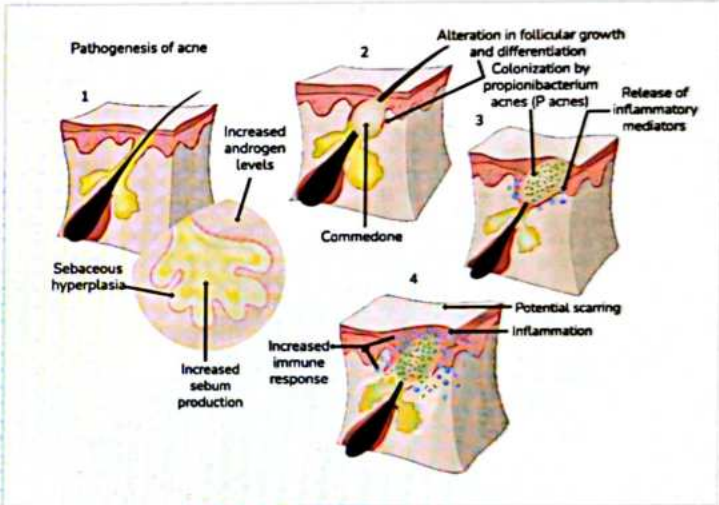
- **Location:** Along the side of hair follicle
- **Site**
  - All over the body (except palms and soles)
  - Increased Concentration in seborrheic areas (scalp, face, upper trunk, back)
- **Opening:** Into the hair follicle
- **Development:** Pubertal age group (androgen dependent)
- **Type of secretion:** Holocrine
- **Modifications:** Opening directly into skin (Ectopic sebaceous glands)
  - **Eyelids** – Meibornian glands
  - **Oral mucosa** - Fordyce spots
  - **Nipple** - Montgomery tubercles
  - **Penis** - Tyson's gland (**Mnemonic:** PT)



  - **Fordyce spots**
    → It is present on lips oral mucosa
    → Appearance is yellowish asymptomatic papules

Acne Vulgaris 00:29:41

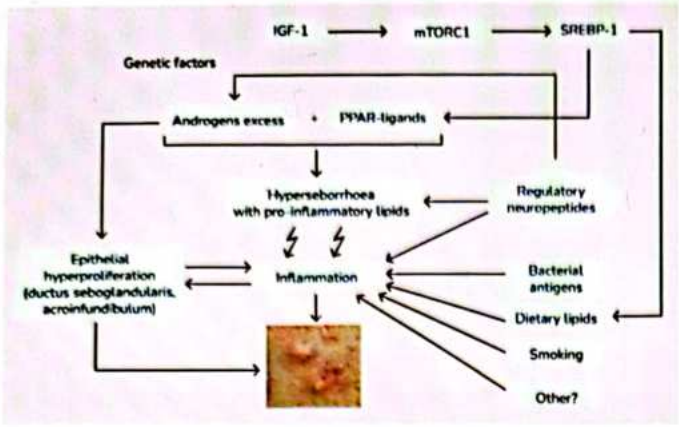
- Most common
- **Age**
  - Usually - Adolescent and Puberty
  - Modifications - Other age groups
- **Lesions:** Polymorphic lesions
- **Site:** Face, chest, back, upper arms
- **Pathophysiology of Acne**



Sebaceous hyperplasia (Increase sebum secreation)
↓
Altered follicular differentiation (keratinocytes)
↓
Blockage of sebaceous gland opening
↓
Colonization of bacteria (commonly Propionibacterium acnes)
↓
Triglycerides converted to Free fatty acids
↓
Inflammation

- **Clinical features**
  - Seborrhea (sebaceous hyperplasia)
  - Comedones (altered follicular differentiation)
  - Papules and Pustules (colonization of bacteria)
  - Scarring (inflammation)

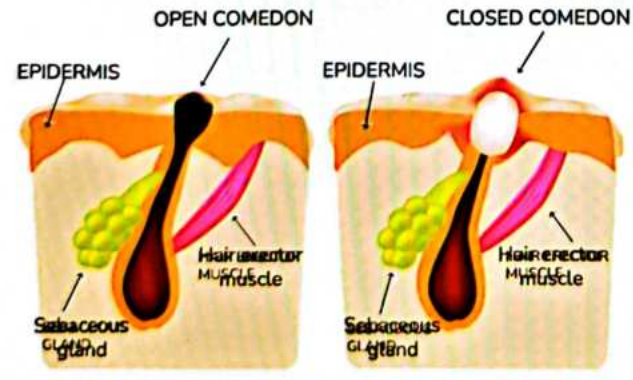
- **Other factors**



- **Genetic factors**
  - → IGF-1 increased
- Environmental factors
- Androgens (seborrhea)
- **Resident flora**
  - → Propionibacterium ovale
  - → Propionibacterium acnes
  - → Staph epidermidis

a. They release lipase that converts Triglycerides to free fatty acids resulting in blockade and inflammation
   - **Inflammatory markers**
     - → IL-1 (believed to be 1st step)
     - → TNF-alpha
   - **Others**
     - → Smoking
     - → Diet (High in fat)
     - → Pollution

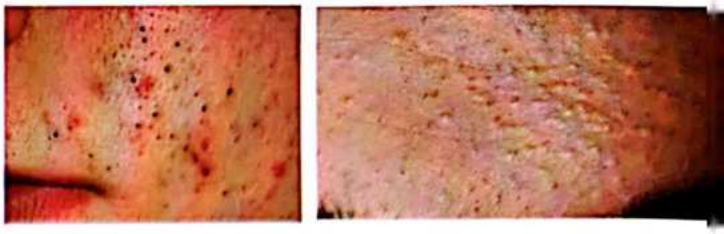
- **Clinical presentation**



- Comedones are characteristic to acne vulgaris
- Comedones are blocked pilosebaceous duct

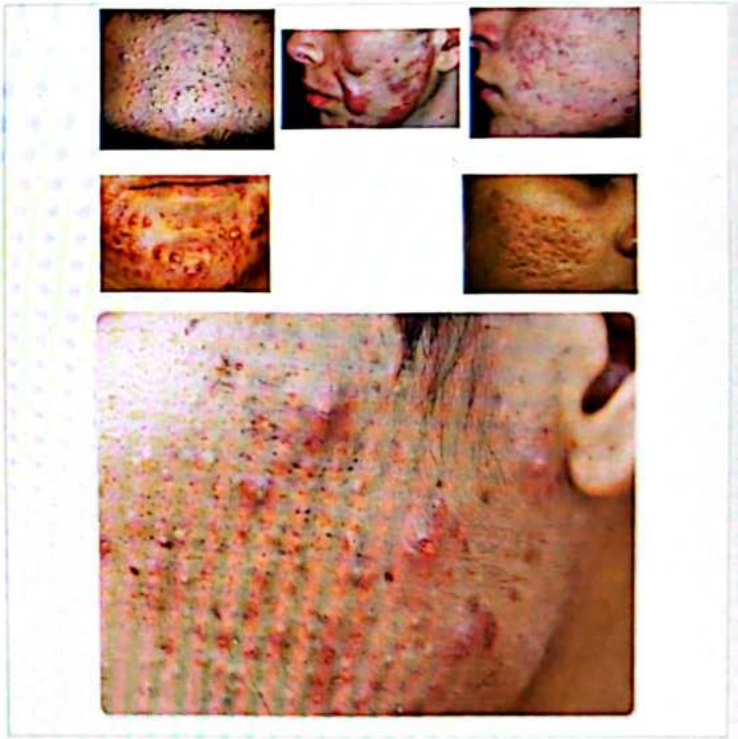
To Remember:

- Pseudo comedones are seen in Hidradenitis Suppurativa (HS) but Comedone are specific to Acne vulgaris.
  - → **Two types**



a. **Closed Comedone**
   i. Also called white comedones/ white heads
   ii. When epidermis is covered

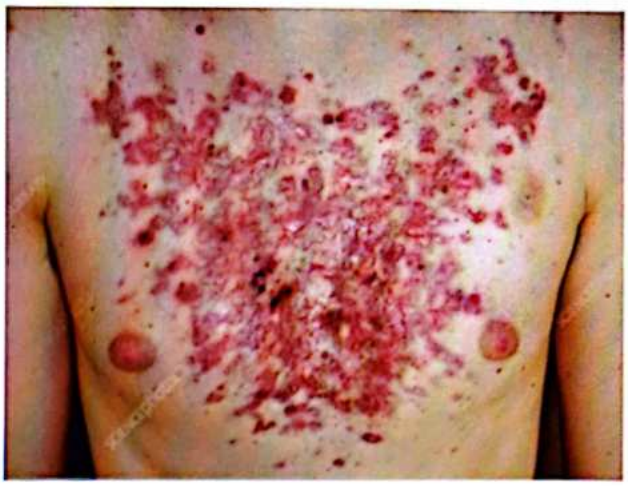
b. **Open Comedone**
   i. Also called black comedones/ black heads
   ii. When there is no epidermis covered
   iii. Keratin that is blocking the gland will get oxidized and becomes black
   - Pustules, papules and scarring can be observed



- Females may present with **Hormonal acne**

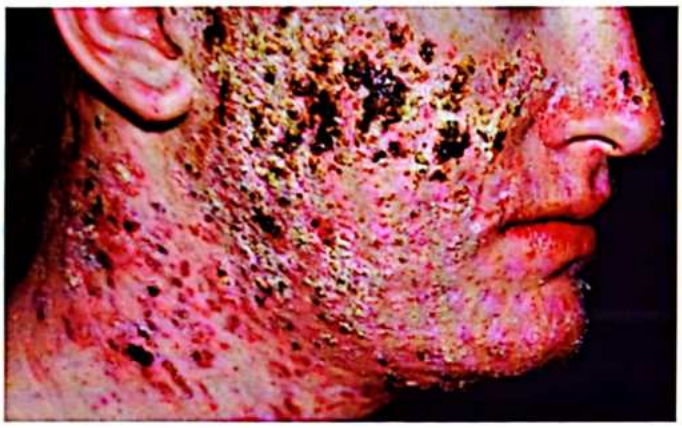
→ Due to overproduction of Androgens from ovaries or adrenals
→ Seen in patients with PCOs, Adrenal tumors, congenital adrenal hyperplasia, prolactinemia, etc.
→ More Acne on lower face
→ Association with signs of hyperandrogenism
- Hirsutism
- Seborrhea
- Female patterned hair loss

- Idiopathic syndrome: HAIR-AN - Hyperandrogenism with insulin resistance with Acanthosis nigricans



Other Types of Acne 00:42:12

- **Acne fulminans**

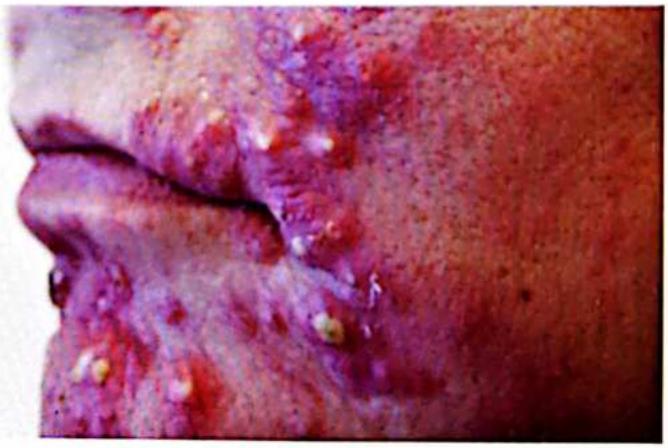


- It is ulcerative form of acne
- Acute in onset
- Systemic symptoms like fever or malaise can develop
- **Treatment:** Antibiotics with Steroids

To Remember:

- Steroids are not given in Acne vulgaris but in Acne fulminans and Acne conglobata

- **Acne conglobata**



- Most severe form of of acne
- Usually seen in males
- Observed on trunk
- Predominant nodules, abscesses, and intercommunicating sinuses
- Treatment includes Antibiotics + Oral retinoids + Short course steroids

- **Neonatal acne**

- Seen in neonates (usually less than 1 month of age)
- It is due to transfer of maternal androgens
- These subsides and no treatment is needed

- **Infantile acne**
  - Seen in infants (less than 1 year of age)
  - Usually subside and if not, can be treated with Benzoyl peroxide

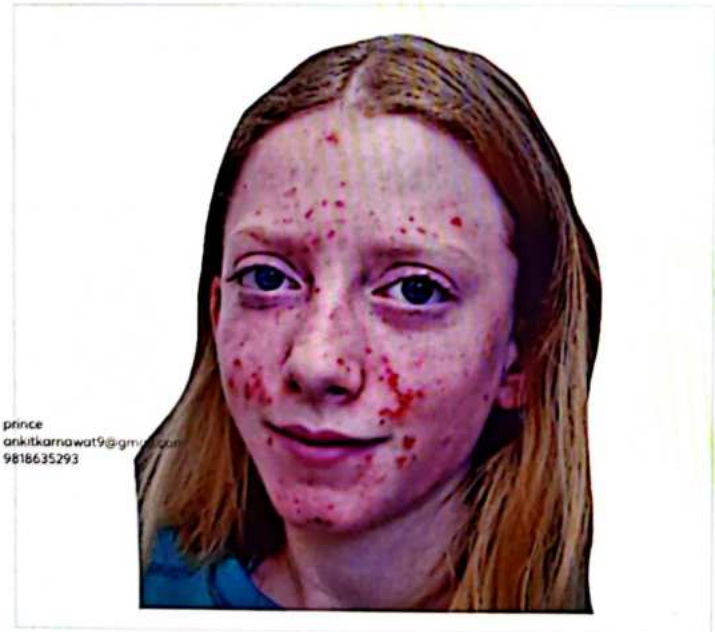
- **Mid childhood acne**
  - Seen in children between 1-7 years of age
  - Precocious puberty should be considered

- **Prepubertal acne**
  - May be due to Adrenarche
  - It may take 2 years to develop puberty after adrenarche.

- **Drug induced acne**

  - Generally caused by drugs like
    - → Steroids (hence not a treatment choice in Acne vulgaris)
    - → Antitubercular drugs
    - → Anticonvulsants
    - → Antipsychotics
  - Differential features from normal acne
    - → Comedones are not seen
    - → Mostly observed on trunk and have temporal correlation
    - → Lesions will be monomorphic

- **Acne excoriee**



  - Seen in patients with psychiatric illnesses
  - Patients scratch the lesions leading to numerous scarring

Grading of Acne and Treatment 00:47:57

- **Grade-01**
  - Comedones
  - **Treatment:** Topical Retinoids
- **Grade-02**
  - Comedones + Inflammatory papules and few pustules
  - **Treatment**
    - → Topical Retinoids + Topical Antibiotics
    - → If necessary, Benzoyl peroxide can be given
- **Grade-03**
  - Comedones + Multiple Inflammatory papules and pustules
  - **Treatment:** Topical Retinoids + Topical Antibiotics + Oral Antibiotics
- **Grade-04**
  - Nodulocystic acne (Nodules + cysts)
  - Most severe form
  - **Treatment:** Oral Retinoids

Topical Retinoids

- They include
  - Tretinoin
  - Adapalene
- Side effects include
  - Dry skin
  - Irritation
  - Secondary bacterial colonization due to flaking of skin due to dryness
- Contraindicated in pregnancy

Benzoyl Peroxide

- Used as 2.5%, 5%, 10% face wash or body wash
- It is antimicrobial and anti-inflammatory
- Side effects include
  - Irritation
  - Stinging sensation
- It may cause staining of clothes

Topical Antibiotics

- They include
  - Clindamycin
  - Nadifloxacin
- Due to concern of antibiotic resistance, these are nowadays combined with Topical retinoids or Benzoyl peroxide
- Other topicals include
  - Azelaic acid
  - Nicotinic acid

- **Chemical peels**
  - These are concentrated chemicals that are applied on face and peeled off after 5 mins
  - Done in the clinic itself
  - Salicylic acid peel
    - → Used in active acne
  - Glycolic acid peel

→ Used to reduce pigmentation and post acne scarring

- **Intralesional steroids**
  - Used when deep nodules or cysts are present

- **Systemic antibiotics**
  - They include
    - → Minocycline
      a. May cause pigmentation
    - → Doxycycline
      a. Side effect is Photo Onycholysis
    - → Lymecycline
    - → In pregnant females, Erythromycin is given

- **Hormonal therapy**
  - Cause based therapy
  - OCP's, Antiandrogens are given

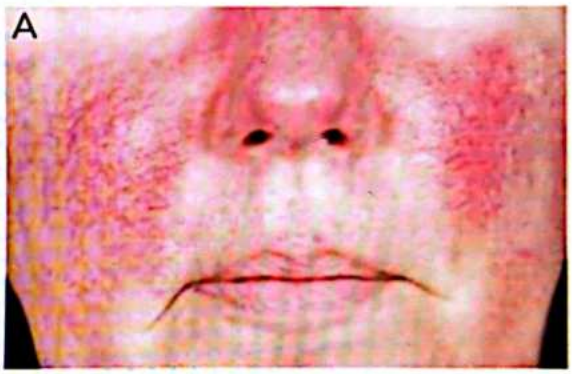
- **Metformin**
  - In patients with insulin resistance

- **Oral retinoids**
  - Treatment of choice is Isotretinoin
    - → It acts on all pathogenetic factors (Seborrhea, Bacterial colonization, occlusion, and inflammation)
    - → Dose is 0.5-1 mg/kg
    - → Stop when the dose range reaches to 120-150 mg/kg
    - → Side effects include
      a. Teratogenicity (Not given in pregnancy)
      b. Female patient should be advised to have gap of 1 month (Wash off period is from 1 month to 3 months) between Isotretinoin usage and conceiving
      c. Dryness
      d. Cheilitis
      e. Ocular dryness
      f. Headache
      g. Secondary bacterial colonization
      h. Rarely, pseudotumor cerebri (Not to be combined with tetracyclines)
      i. Rare but Severe side effect is Diffuse interstitial hyperostosis (DISH)
      j. Rarely depression

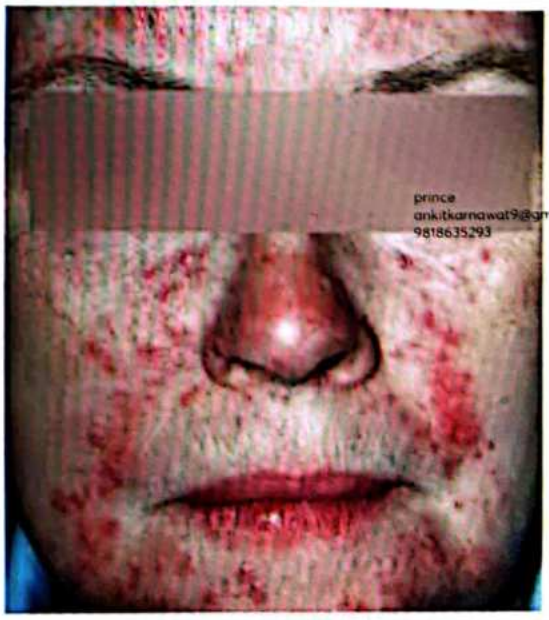
Rosacea 01:00:36

- It is also known as Acne rosacea
- It is misnomer and is not related to Acne
- In Rosacea, inflammation and vascular hyperactivity is seen
  - Flushing is seen
    - → When exposed to heat or spicy foods
    - → When felt anxious or stressed
- Predominantly seen in adults
- Affected areas are convexities of face
  - Cheeks
  - Nose
  - Chin

- **Etiology**
  - Due to innate immunity
  - Exposure to UV light
  - Stress
  - Hot spicy food
  - Organisms include
    - → Demodex
      a. Hence Ivermectin is used in treatment of Rosacea
    - → Staphylococcus epidermidis
    - → Chlamydophila pneumonia
- **4 Subtypes**
  - Subtype 1

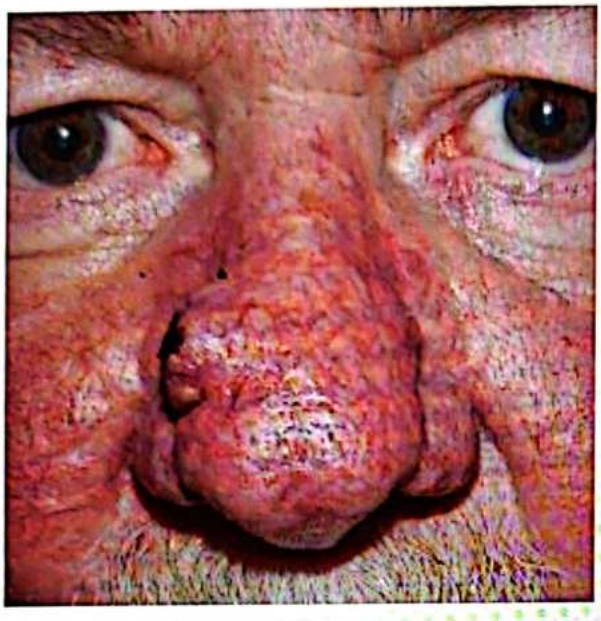


    - → Erythematotelangiectatic rosacea (ETTR)
    - → Erythema and telangiectasia (Dilated blood vessels) is seen
  - Subtype 2
    - → Papulopustular rosacea (PPR)



→ Papules and pustules are seen
→ No comedones are seen
→ Instead, telangiectasia is observed

- Subtype 3



→ Phymatous rosacea (PR)
→ Granulomatous lesion is seen on nose
→ Also called potato nose

- Subtype 4
  a. Ocular rosacea: have ocular signs & symptoms

- **Treatment**
  - Avoiding triggering factors
  - **For Subtype 1**
    → Vasoconstrictors
    a. Topical brimonidine
    b. Oral propranolol
  - **For Subtype 2**
    → Topical Metronidazole
    → Topical Ivermectin
    → Antibiotics
    a. Doxycycline
    b. Erythromycin
  - **For Subtype 3**
    → Additionally, Retinoids can be given
    → Surgical/Laser corrections if necessary

15 GENITAL ULCER DISEASES

STD vs STI

- STDs were considered diseases transmitted through the genital route.
- Certain infections infect the bloodstream transmitted through the sexual route, but they can be in blood also.
- Now, it is called Sexually Transmitted Infections.

Genital Ulcer Disease 00:02:08

- Syphilis
- Chanceroid
- Herpes genitalis
- Donovanosis
- LGV

Syphilis 00:03:00

- Caused by Treponema Pallidum
- One night with Venus, a lifetime with mercury
- If not treated, it can stay in the body for 10-40 years.

Bacteriology	
Family	Spirochaetacea
Species	Treponema pallidum "Turn and thread like"
Length	6-15um
Width	0.25 um
Coils	8-24
Culture	Not cultivable bacilli

Motility

Two types of motility:

- Locomotion - one place to another and it has corkscrew rotation
- Propulsion

Classification

Congenital	Acquired
Fetus from Mother	Sexual Blood Transfusion

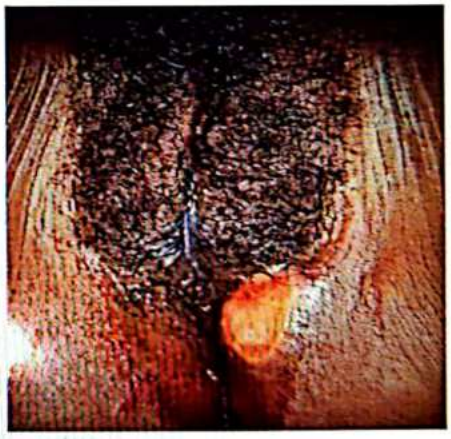
Exposure to Treponema pallidum 1/3 infected
↓ (10-90 days)
Primary (chance)
↓ (2-12 weeks)
Secondary (mucocutaneous lesions/organ involvement)
↓ (2-12 weeks)
Early laten ↕ relapsing (in 25%)
(1-2 year from contact)
↓
Late latent
(more than 2 years)

- Remission (2/3)
- Tertiary (1/3)
  - Late benign (16%)
  - Cardiovascular (10.4%)
  - Neurosyphilis (6.4%)

Acquired syphilis

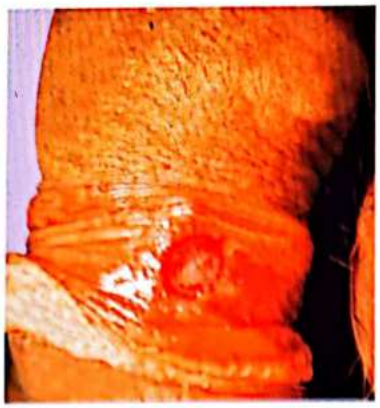
Primary Syphilis

- Incubation period of 10-90 days
- Chancre/ Hunterian chancre/ hard chancre
- Ulcer in the genital area



Characteristics of ulcer

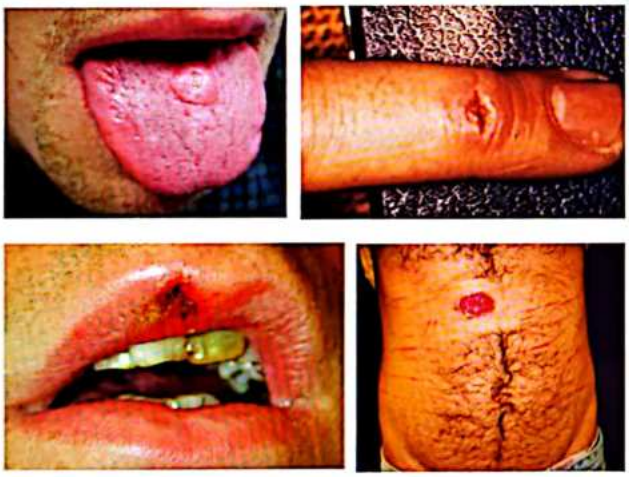
- Single
- Painless - the patient does not know hence go into the second stage
- Well-designed ulcer
- Rubbery margins
- Clean base
- Indurated– it thickens while touching
- Serious discharge may be there on pressing



- Button sign: palpating the chancre as if putting the button.
- Lymph nodes:
  - Bilateral
  - Painless
  - Enlarged
  - Shotty

Extra genital lesions

- They are transmitted through oral or other routes - chancre can be on the trunk, fingers, mouth etc.



Secondary Syphilis

- Primary syphilis
  ↓
- Spirochetemia
  ↓
- Treponema disseminates to all the tissues via the bloodstream
  ↓
- Multiply in the tissues
  ↓
- Secondary syphilis

Secondary syphilis

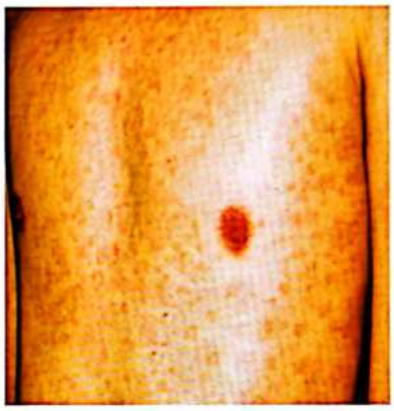
- Affects the skin
- Mucosa
- Systemic
- Lymph nodes
- Secondary stage is called the great imitator
  - It mimics lots of disorder - fever, skin rash, lymph nodes

Constitutional symptoms

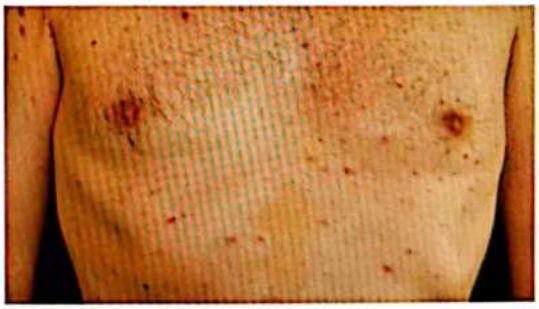
- Spirochaetemia involved, fever malaise
- All the systems is involved hence it is called The Great Imitator

Skin Lesions

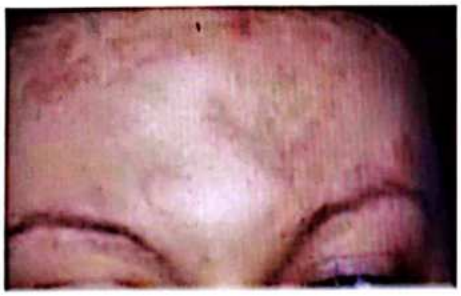
- Bilaterally symmetrical
- Asymptomatic
- Polymorphic
- Does not present with vesicles and bulla
- Ham/rose coloured

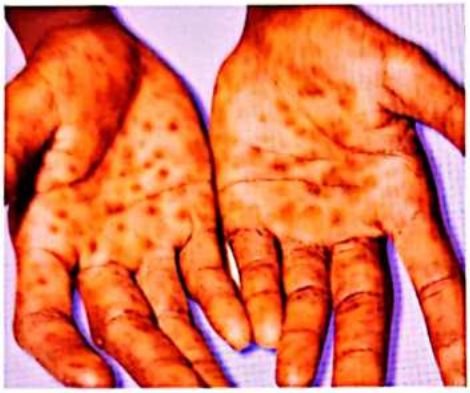


- Macular - because it is rose-coloured also called roseola syphilide
- first rash to appear–subsequently turning into papular rash hence the name papular syphilide– papular lesion



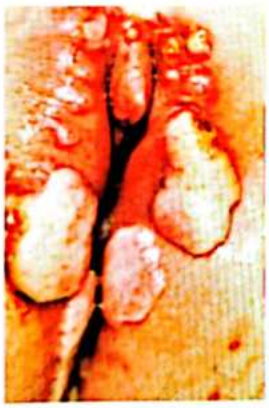
- These papules are present like a crown on the forehead, then it is called corona veneris. The infection is on the edge of the hairline.

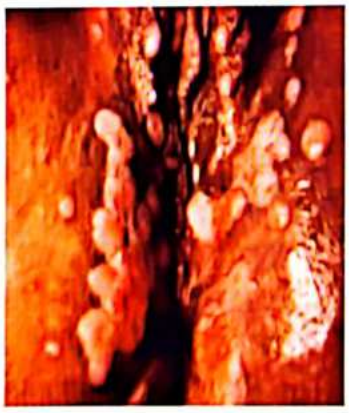




- When syphilis affects the palms and soles, hyperpigmented plaques are seen
- Pain on being pressed by blunt pin
- This is called the Buschke Ollendorff sign.

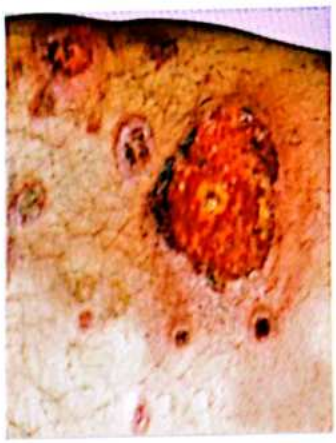
Asymptomatic- but tenderness, when pressed (because of endarteritis obliterans)



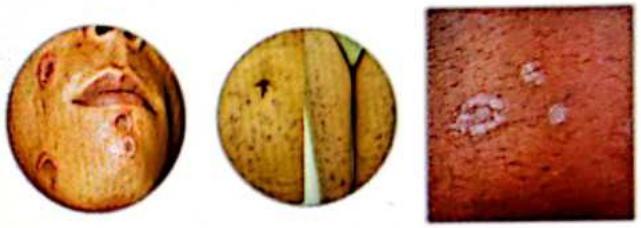


- Papular syphilides are present on the flexors like the perirectal area and vaginal area
- Because of constant friction, they get macerated
- Condyloma lata means broad and flat
- Moist papules present in flexural areas
- These are highly contagious
- Teaming with spirochetes – very very infectious
- Covered with grayish slough which is rich in spirochetes

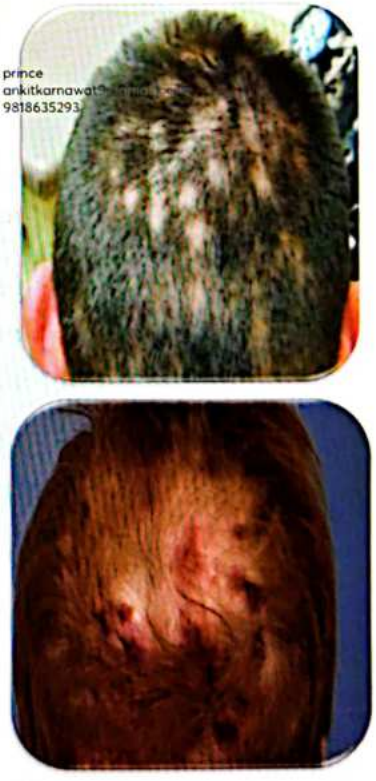
Lues Maligna



- These crusted necrotic plaques are called Lues Maligna.
- Secondary Syphilis is present in many forms except vesicles and bulla.
- Angular form /aneuploid forms/ psoriasis forms - secondary syphilis can be present.

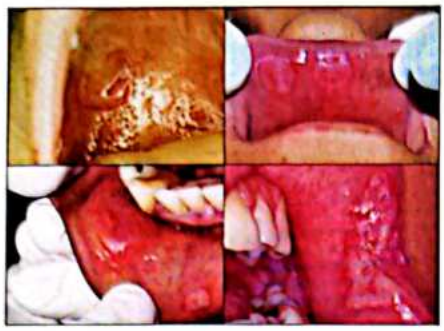


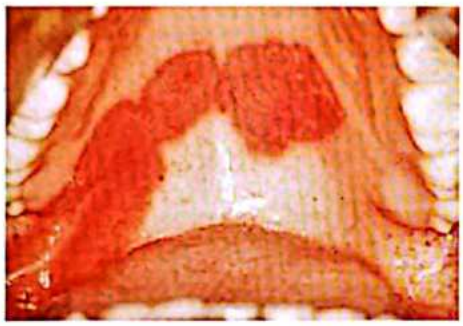
Hair:



- Moth-eaten alopecia WITH circular patches
- Non-scarring alopecia different from alopecia areata

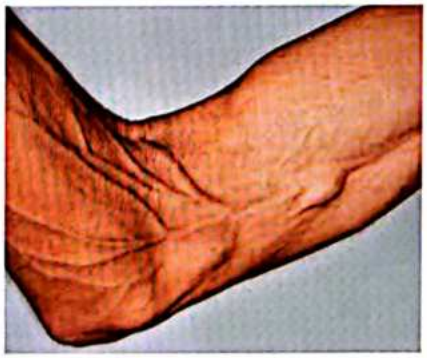
Mucosal lesions





- Secondary syphilis can lead to mucosal lesions
- Called as mucosal patches or snail track ulcer
- Well defined erosion on mucosal surface
- Painless erosion can be present all over the mucus membrane

Lymphadenopathy



- Bilateral
- Symmetrical painless wide-spread lymphadenopathy
- This is epitrochlear LAN- specific for leprosy caused by secondary syphilis

Systemic manifestations:

- Involvement of spleen
- Involvement of blood
- CNS manifestation
- Involvement of kidney, liver
- Different manifestations that can be there due to spirochete apnea.

Syphilis D' Emblee

- When treponema pallidum is transmitted through blood transfusion, there is no primary stage
- Patient is immediately present with the secondary stage – syphilis d' emblee
- Spirochetes are present throughout the blood

Latent Syphilis

- Asymptomatic
- No clinical signs or symptoms but if you do a serology, it will be positive- VDRL and TPHA
- Spirochetes are there but not manifesting themselves.
- There can be telltale signs - healed ulcers, lymphadenopathy is there
- Latent is divided into two types:
  - Early latent syphilis– less than 1 year- maybe less than 2 years
  - Late latent syphilis- more than 1 year– more than 2 years it is not infective
- Syphilis is infective
  - Infected stage of syphilis is Primary, Secondary, and early latent stage is infective
  - Late latent and tertiary stage is not infective

Important Information

- Sometimes when the patient is in the early latent stage- 25% may relapse back & go to the second stage.
- This can be a clinical relapse or serological evidence
- VDRL 1:8 will jump to 1:64
- Relapsing is present at the secondary stage
- Chancre redux– lesions develop at the site of the primary chancre; relapsing lesions will appear there calling it as chancre redux.

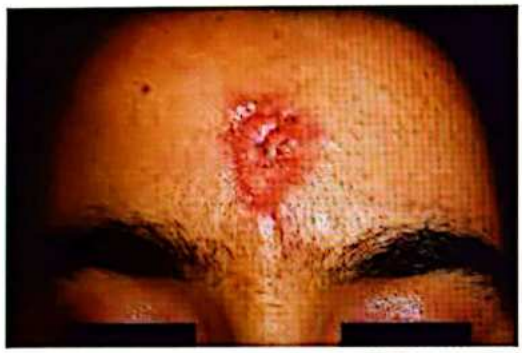
Tertiary Syphilis

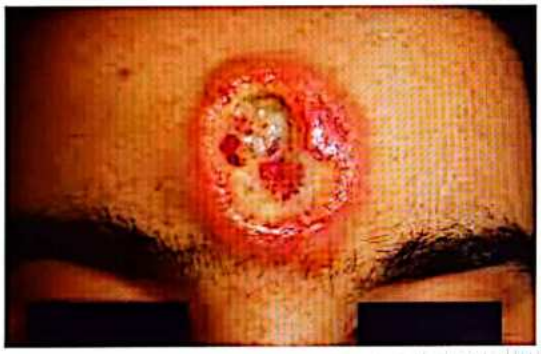
- After the patient has gone through early and late latent.
- 2/3rd will recover
- 1/3rd from late latent syphilis will go into the tertiary stage, which is a non-infective stage (Develops after 10-40 years)
- One night with Venus, a lifetime with mercury

- Hypersensitivity reaction which occurs to the reminiscence to the spirochetes

Types

- Benign- it can involve skin, bones, and lymph nodes
- CVS- aortitis and aortic aneurysms, ascending aorta
- CNS- different manifestation
  - General paresis of instate associates with psychiatric + affective issues
  - Tabes dorsalis which will manifest with Gait abnormalities





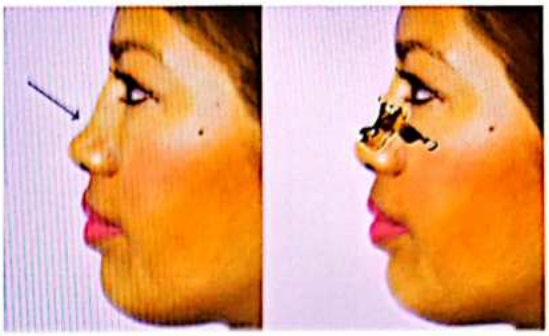
Skin lesions of the tertiary syphilis presents with

- Skin lesions of tertiary syphilis is called as gumma
- Gummy consistency
- Deep dusky red papule and nodules
- Heal with scars

Pseudo chancre redux

- Occurrence of gumma at the site of a chancre– tertiary syphilis lesions
- Chancre can be
  - Hard or Hunterian (are seen in primary syphilis)
  - Extragenital (primary syphilis)
  - Chancre redux (relapsing)
  - Pseudo chancre redux (tertiary syphilis)
  - Soft chancre redux (chanceroid)

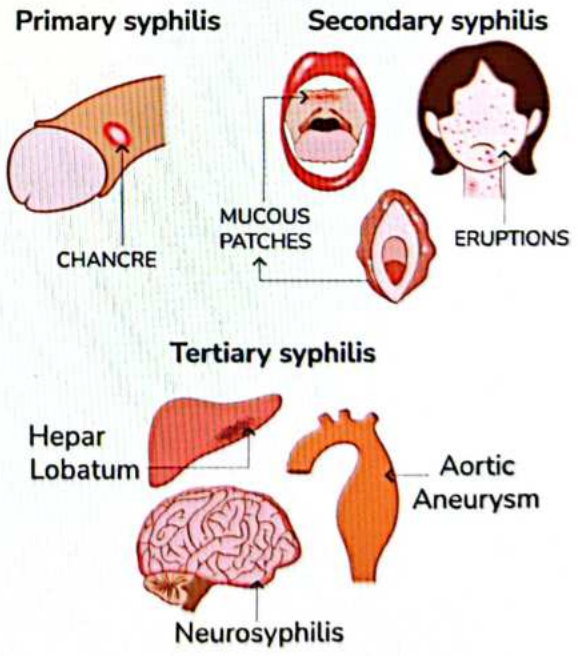
Destructive syphilis



- Saddle nose deformity is seen in tertiary syphilis.

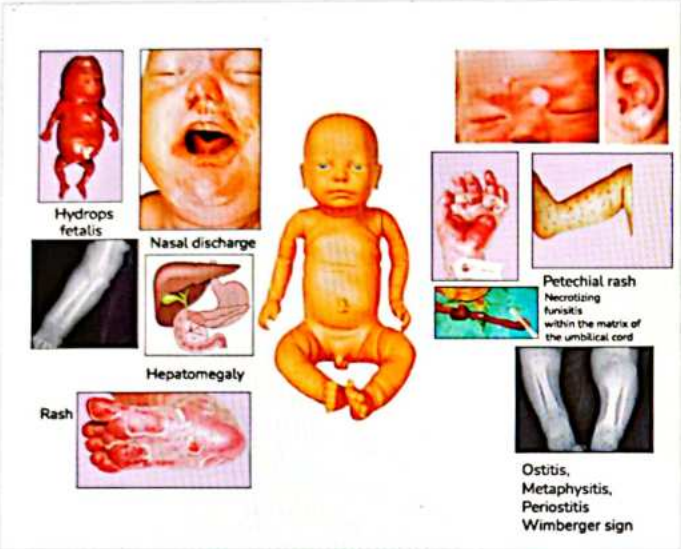
General Paresis of Insane

- **Progressive** Disease
- **Nerve** cells - brain - mental deterioration - psychiatric symptoms
- **Symptoms**
  - Paresis
  - Personality changes
  - Alteration of affect
  - Hyperactive reflexes
  - Alteration in eye function
  - Changes in sensorium
  - Decreased intellect
  - Slurred speech
- **Tabes dorsalis:** Post column and post root of spinal cord– ataxia, gait abnormalities (foot slap gait)
- **Charcot joints:** Enlarged, painless, inflamed joints of lower extremities and spine due to repeated traumatic injury

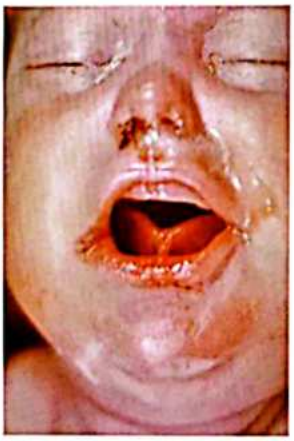


Congenital / Prenatal Syphilis 00:32:18

- Transmitted from the mother to the child after 20 weeks
- Divided into two stages:
  - Early congenital usually manifests at less than 2 years, infective
  - Late congenital usually manifests after two years - present with stigmata–not infective



- A little wrinkled potbellied old man with a cold in his head
  - Wrinkled - old age appearance
  - Pot Bellied because of hepatosplenomegaly
  - Cold head - nasal discharges– Snuffles
  - Bone changes
  - Hydrops fetalis
  - Rash
  - Necrotizing funisitis
- Snuffles is the nasal discharge



  - Which is seen at the early congenital syphilis
  - Discharge is teaming up with spirochetes and is very infective

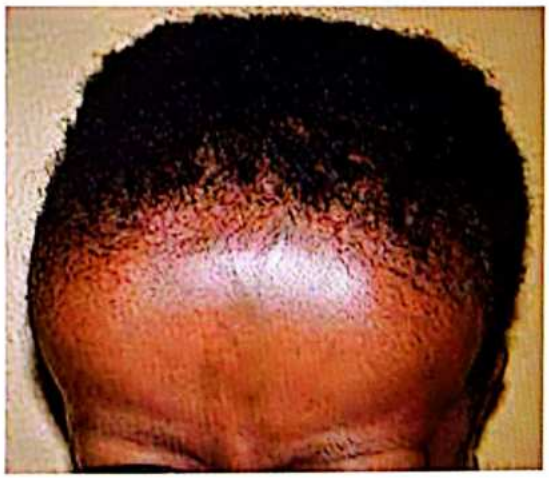
  - The cartilage gets affected leading to saddle nose or fleur de lis nose

- In skin the rash of syphilis presents at vesicles and bullae - early congenital syphilis
- Infants can have vesicles and bullae that is why syphilitic pemphigus in infants

Bone changes

- Pseudoparalysis of parrot: Because of osteochondritis and pain infant does not move his limb and it looks like paralyzed

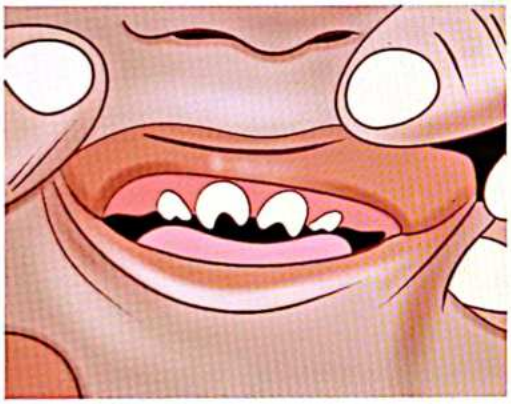
Late Congenital Syphilis



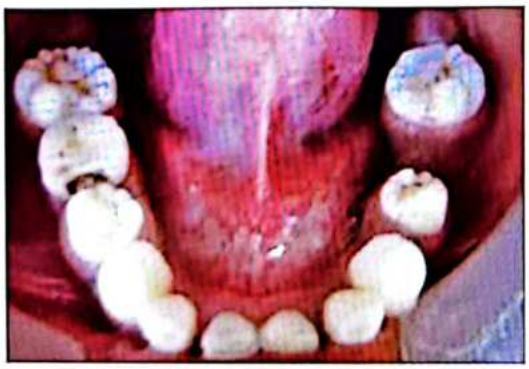
- Presence with stigmata
- What are the stigmas?
  - This frontal bone is increased due to new bone formation called frontal bossing
  - The scapula thickens and it is called Higoumenakis sign

Hutchinson's Triad

- Late congenital syphilis
- Teeth
- 8th nerve deafness
- Interstitial keratitis



- Abnormal permanent upper central incisors that are peg-shaped and notched– HUTCHINSON'S teeth

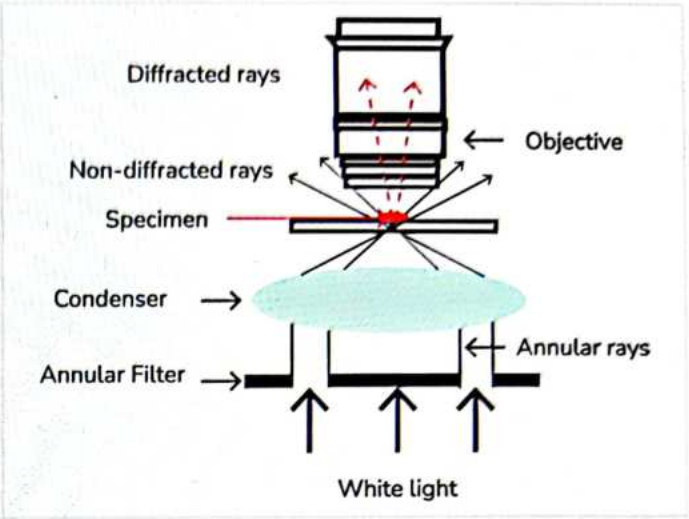


- Molar appears like mulberry
- Cusps on molars and are rounded
- Called as mulberry molars –a specific feature of congenital syphilis

Investigations

Identification of organs:

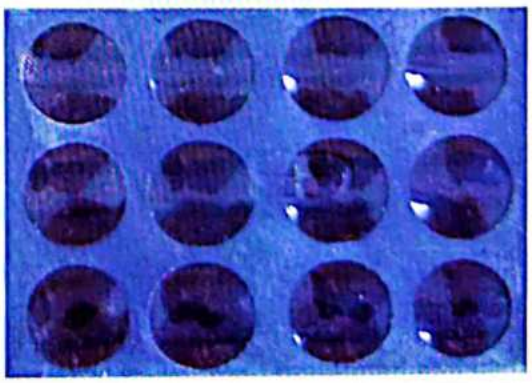
- By dark ground microscopy



- Use for an organism that cannot be stained
- Reflects the light transmitted by the organisms
- Treponema pallidum it shows fluorescent
- It appears spiral wavy slender fluorescent organisms– commonly done
- Take Chancre and take a smear and do the dark ground microscopy

Serological tests	
Treponemal / specific test • They become positive first • They stay positive, not helping in regular monitoring	Non- Treponemal/ Reagin Test • Become positive later • They become negative and helps in monitoring
TPHA, FTA- ABS, TPI, TPPA, ETA, latex agglutination	VDRL, RPR, WASSERMAN, TRUST, RST, UST

VDRL Slide



- Slide Flocculation test
- It becomes positive after 4-6 weeks of acquiring infections or 7-14 days of primary chancre
- Always given in dilution and > 1:8 dilution is considered positive
- It is important because it act as:
  - Screening test
  - Congenital syphilis- VDRL of mother and child – if the test is different from mother —child is infected
  - Monitor response to treatment– treponemal test is stay positive–
  - VDRL will show fall in dilution–treatment has worked
  - Reinfection indicator

Important Information

- Most specific test- DGI
- Most specific blood test- FTA-ABS, TPHA
- EARLIEST TO BECOME positive– IgM Capita (rarely done)> FTA-ABS
  - Mark Igm Captia
- Most sensitive test- IgM > FTAABS
- Screening - VDRL, RDR
- Field based screening- RPR card
- IOC in 1-DGI, 2- FTA ABS, 3- EIA, NEURO - CSF EXAM
- To monitor the treatment and disease activity–VDRL

Investigations in Congenital Syphilis:

- VDRL
- FTA-ABS
- IgM Captia
- CSF

Note: IgM is not transmitted, IgG is transferred through maternal blood

Treatment

- Injection is Benzathine Penicillin, Dosage - 2.4 million units Intramuscular
- Two divided doses after a test dose
- If primary, secondary or early latent syphilis – only one dose is given
- In case of late latent or tertiary syphilis (benign CVS) -- 3 doses at weekly interval
- For children: 50,000 units/kg IM up to the adult dose of 2.4 million units in single dose

Neurosyphilis and congenital syphilis:

- Benzathine penicillin is not given because it cannot cross blood-brain barrier
- Used agents:
  - Aqueous crystalline penicillin- Intravenous
  - Procaine penicillin probenecid

Q: What if the patient is allergic to penicillin?
Ans: Desensitize the patient because penicillin is the most effective drug

Q. How do you desensitize the patient?
Ans: Start giving the slow doses and then gradually increase the doses; if desensitization is not possible, then DOXYCYCLINE 100 mg twice daily

- Where 14 days is used for early syphilis
- And 28 days for late syphilis

Q. What if the patient is pregnant?
Ans: same treatment

Q. What if the pregnant patient is allergic to penicillin?
Ans: you can only desensitize because we don't want the risk of the mother having congenital syphilis
If desensitization is not possible, then you can give erythromycin.

Jarisch Herxheimer Reaction:

- When penicillin is injected at the early stages of syphilis, there is a huge spread of spirochetes which get killed, and it creates **Jarisch Herxheimer Reaction**
- Few hours after administering the penicillin, and this is because of the body's hypersensitivity reaction against killed treponemes
- When the benzathine penicillin is injected — a test dose is given, and the patient is kept for 3-4 hrs of daycare to avoid this reaction
- Present symptoms are fever malaise, headache, vomits, nausea, abdominal pain, rash
- **Treatment:** symptomatic treatment– crocin, antihistamines, oral steroids (short course)

Chancroid

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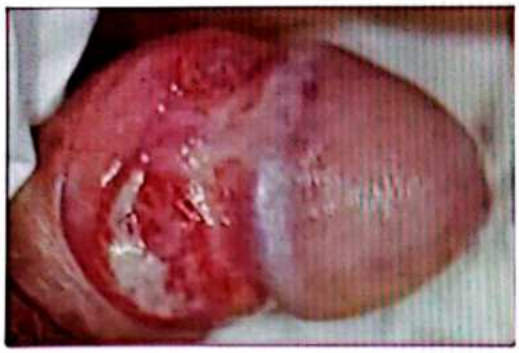
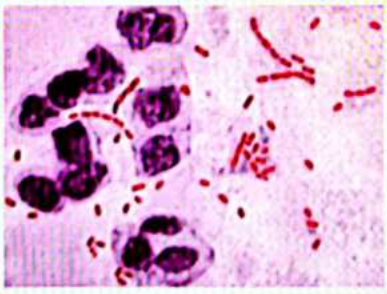


- Known as soft chancres because these are non-indurated
- Caused by: Haemophilus ducreyi
- Incubation period: 3-7 days

Present as:

- Multiple ulcers
- Painful
- Non indurated
- Undermined edges
- Greyish slough
- Bleeds on touch
- Lymph nodes are unilateral, painful
- Suppurative lymphadenopathy is called as 'BUBO'

Investigation	
Gram stain	Gram-negative bacilli arranged in chains, also called as railway tracks or school of fish appearance 
Culture	Mueller Hinton Agar supplemented with chocolate horse blood
Treatment	Azithromycin 1 g orally in a single dose Ceftriaxone 250 mg intramuscular in a single dose

Herpes Genitalis

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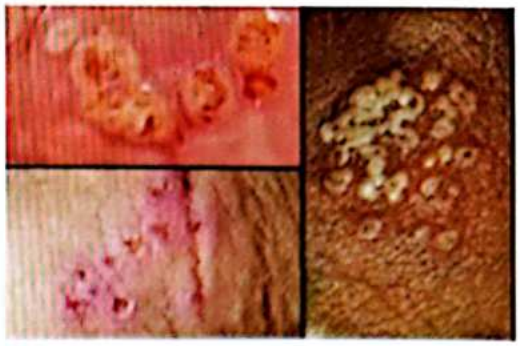
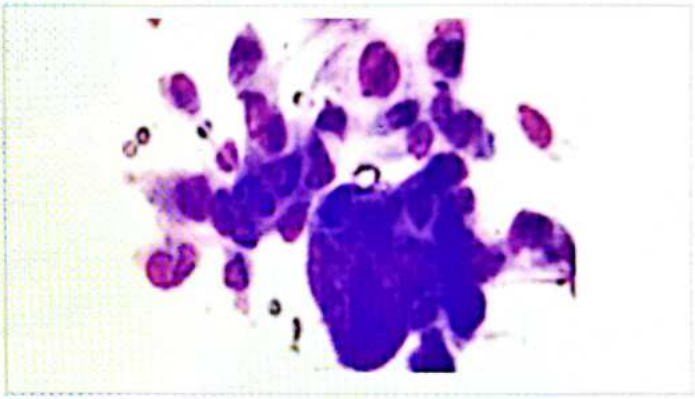


- Organism: HSV 2> HSV 1
- Incubation period: 2-7 days
- Present in primary recurrent stage latent infection
- Multiple painful; non- indurated vesicles
- They rupture to form coalescing vesicles or polycyclic erosions
- There are no ulcers like chancroid
- Lymph node: bilateral and tender Lymphadenopathy



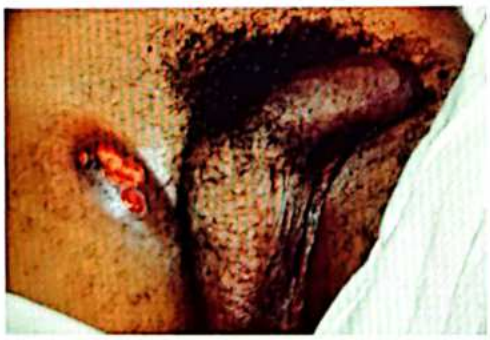
Investigation	
Tzanck Smear	Multinucleated giant cells are characteristics
Serology	ELISA - Ag- HSV glycoprotein
Treatment	Acyclovir – 200 mg 5 times a day Acyclovir – 400 mg 3 times a day 7-10 days for primary infection 5 days for secondary infection

- Its latent in Dorsal root ganglion

Donovanosis

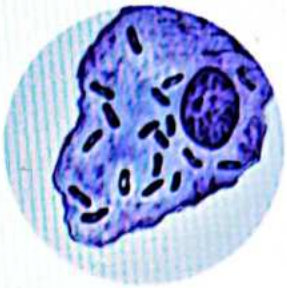
Known as	Granuloma venereum (std)or Granuloma Inguinale
Organism	Calymmatobacterium granulomatis/ klebsiella granulomatis
Incubation period	3 days to 3 months

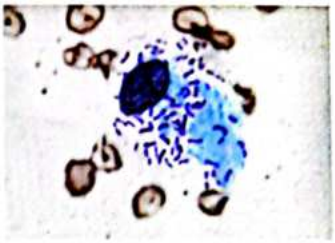
- Patients is present with
  - Beefy red granulomatous ulcer which bleeds very easily on touch.
  - Single painless ulcer due to many granules it bleeds very easily.



- Lymph nodes are not involved,
- But sometimes, donovanosis presents may develop on lymph nodes
- It is referred to as "Pseudo-BUBO"

Investigation:





- Take a tissue, crush it on a smear and see it on an organism that has peripheral condensation of cytoplasm
- Inside the cell these bodies are called as Donovan bodies
- Appearance is like safety pin appearance
- Test: NAAT- nucleic acid amplification test or CFT

Treatment:

- Azithromycin- 1 g per week or 500 mg daily for at least 3 weeks and until all lesions have completely healed

Alternative regimes

- Doxycycline 100 mg orally twice a day for at least 3 weeks and until all lesions have completely healed.

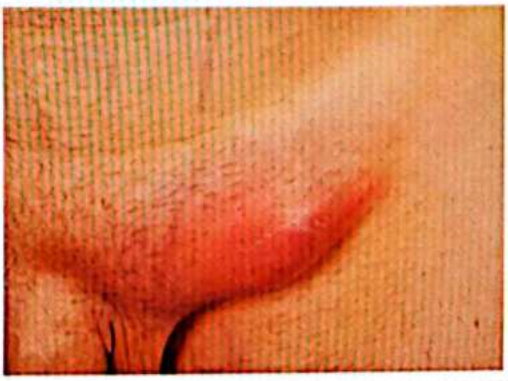
Lymphogranuloma Venereum

01:03:56

- Lymphatic involvement
- Also known as a climactic bubo, lymphogranuloma inguinale
- Organism: Chlamydia Trachomatis serovars L1, L2, L3 – L1 is most common
- Incubation period is 10-30 days

Clinical Stages and features:

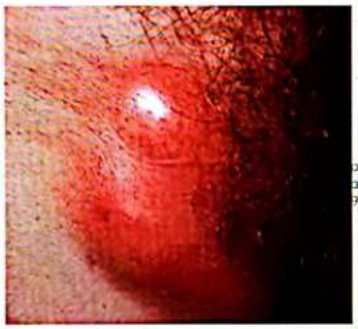
- First stage is the formation of the ulcer which is transient ulcer and patient is not present in this stage
- Secondary stage: patient presents mostly in this stage, Inguinal syndrome
- Tertiary: when the infection is spread in the larger area it is called Genito Acro Crural Syndrome



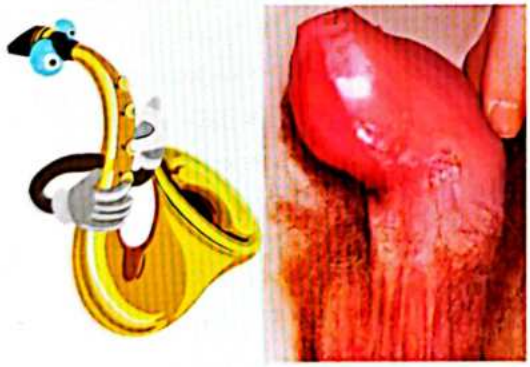
- Suppurative lymphadenopathy
- Multilocular
- Breaks down leads to the formation of discharging sinus
- Climatic BUBO

Lymphogranuloma venereum vs chancroid

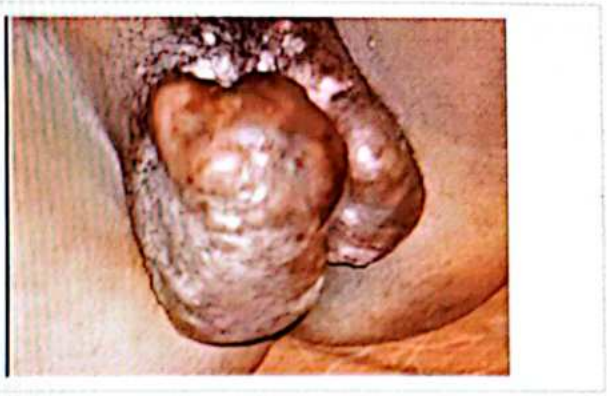
Chancroid	Lymphogranuloma venereum
Unilateral	Bilateral
Ulcer present	No ulcer
Abscess is unilocular	multilocular



- There is the involvement of femoral and inguinal node - inguinal ligament also called Poupert's ligament
- When both femoral node and inguinal node are involved
- It appears taut and forms a groove called the Groove sign of GreenBlatt.



- Third stage where there is excessive lymphatic involvement
- In males, the penis may look like a saxophone or ramus penis in the tertiary stage of LGV



- In females, the vulva may become enlarged, which is called Esthiomene

Investigation:

- NAAT - nucleic acid amplification test
- CFT- Complement fixation test

Treatment:

- Doxycycline 100 mg orally twice a day for 21 days

Alternative regime

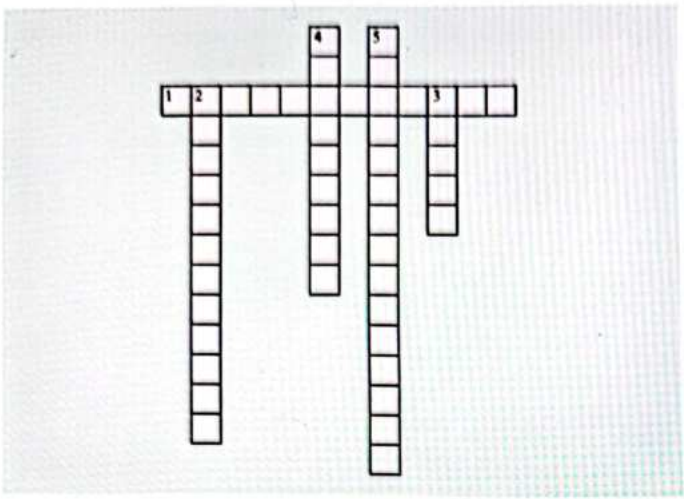
- Erythromycin base 500 mg orally four times a day for 21 days

Approach to Genital Ulcer Disease:

	Syphilis	Chancroid	HSV	Donovanosis	LGV
Incubation period	9-90 days	2-7 days	2-7 days	3-30 days	10-30 days
Ulcer number	One	Multiple,	Multiple	One	Transient
Pain	No	Yes	yes	No	No
Base	Clean	Covered with grayish slough	Erosion	Granules	Nothing
Edges	Rubbery	Undermined	Polycyclic	Overhanging GRANULES	Nothing
Induration	Positive	Negative	Negative	Ulcer may be firm	Nothing
Bleeds on touch	No	Yes	Yes	Yes very easily	Nothing
Lymph nodes					
Laterality	Bilateral	Unilateral	Bilateral	Pseudo Bubo	Bilateral
Pain	No	Yes	Yes	No	Yes

CROSS WORD PUZZLES

Crossword Puzzle



1. STDs were considered diseases transmitted through the _____.
2. What if the patient is allergic to penicillin and desensitization is not possible
3. The Characterstic of this disease are Indurated, Painless and Ruberry Margins
4. Known as soft chancres because these are non-indurated
5. Present in: primary recurrent stage latent infection

16 STD DISCHARGE

- **Two types**
  - Urethral discharge and Cervical discharge (taken together as causative organism is same)
  - Vaginal discharge
- **Causes**
  - STD causes
  - Non STD causes

This is only about STD causes

STD Causes of Discharge 00:00:36

- **Broadly two types**
  - Gonococcal
  - Non gonococcal

Refer Table 16.1

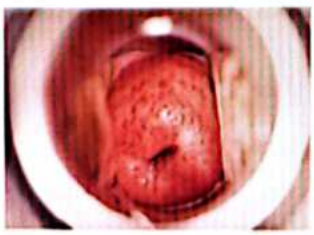
Disseminated Gonococcal Infection 00:10:49

- Seen in severe cases of GU.
- Disseminated into the bloodstream, thus has systemic complications.
- **Other name:** Acute arthritis dermatitis syndrome.
- **Joints involved in Arthritis:** Wrist, Metacarpophalangeal joint, knee, ankle
- Tenosynovitis
- **Skin:** Dermatitis, which has pustules especially localized at the distal portion of extremities.
- **Treatment:** Ceftriaxone 1g IM or IV every 24 hours + Azithromycin 1 gm oral x 7 days.

Vaginal Discharge 00:11:51

- **Most common cause:** Physiological.
- **Most common pathological cause:** Bacterial vaginosis.
- **3 major discharges:**
  - Vulvovaginal candidiasis
  - Trichomonal vaginalis
  - Bacterial vaginosis

Refer Table 16.2



Q. Strawberry cervix is a feature of?
Ans: Trichomonas vaginalis

Bacterial Vaginosis - Amesis criteria 00:21:20

Three of the following symptoms or signs:

- Grey/white, homogenous, thin, scanty discharge that smoothly coats the vaginal walls.
- Clue cells (e.g., vaginal epithelial cells studded with adherent coccobacilli) on microscopic examination, at least 20%.
- pH of vaginal fluid >4.5
- A fishy odor of vaginal discharge before or after addition of 10% KOH (i.e., the whiff test).

Other STIs 00:22:05

- Genital scabies
- Molluscum contagiosum
- Anogenital wart
- Pubic lice

Syndromic Management of STDs 00:22:24

- STD patients usually are not supportive for the treatment
- And most probably they prefer the treatment at PHC.
- For these reasons STDs are divided to syndrome
  - Urethral discharge
  - Cervical discharge
- Kits are given to patients for treatment.

Kits Used in STD Syndrome 00:23:50

There 7 kits for STD syndromes

Refer Table 16.3

Mnemonic: **great girls won't buy red yellow bags**

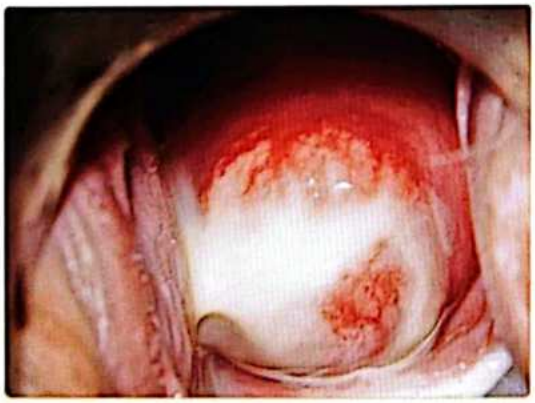
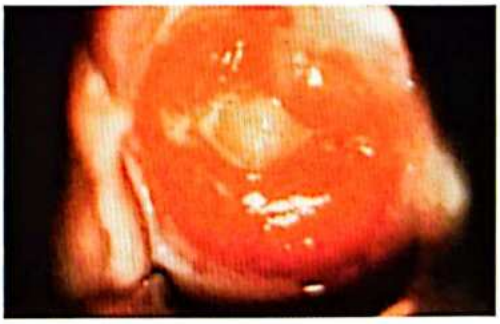
- **1st kit**
  - GREAT - Grey kit
  - GREAT - Gonorrhea and Trachomatis (Urethral discharge)
- **2nd kit**
  - GIRLS - Green kit
  - GIRLS have - Vaginal discharge
- **3rd kit**
  - WON'T - White kit
  - White (When board is white we write ABC)
    - → **A** - Azithro
    - → **B** - Benzathine
    - → **C** - Chancre/ Chancroid

- **4th kit**
  - BUY - Blue kit
  - Buy (when we buy, we spend money, spending is BAD)
    → BAD because the patient is allergic to penicillin.
    → A - Azithro
    → D – Doxy
- **5th kit**
  - RED - Red kit
  - Red - Means herpes (painful)
  - For herpes – Acyclovir
- **6th kit**
  - YELLOW - Lower abdominal pain
  - Pelvic inflammatory disease (PID)
  - For PID - Cefixime, Metronidazole, Doxy
- **7th kit**
  - BAGS - Black
  - Black is for Bubo.
  - BAD - For Bubo, we use Azithro and Doxy.

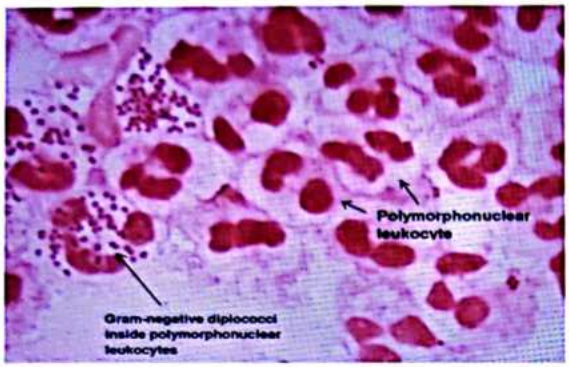
To Remember

- **BAD - Both B kits (Buy and Black) have Doxy and Azithro**
- **Buy is for genital ulcer disease.**
- **Black is for Bubo.**

Table 16.1

Gonococcal	Non Gonaococcal
Causative Organism	
Neisseria gonorrhoeae	**Main cause:** Chlamydia trachomatis (D-K) **Others:** Mycoplasma, ureaplasma, HSV, Adenovirus, Trichomonas vaginalis
Disease Name	
Gonococcal urethritis/ cervicitis (GU/ GC)	Non gonococcal urethritis/ cervicitis (NGU/ NGC)
Incubation Period	
3-5 days	3-5 days (little longer sometimes)
Presentation of Constitutional Symptoms	
+ve (fever)	-ve
Urinary Symptoms	
+ve (dysuria)	-ve
Clinical Features	
In penis: Thick copious purulent discharge 	**In penis:** Thin watery scanty clear looking discharge 
In cervix: Thick copious purulent discharge 	**In cervix:** Thin watery scanty clear looking discharge 
• Cervix is friable	• Cervix is friable
Investigations	

- **Gram stain**
  - Gram -ve diplococci
  - Bean or kidney shaped cells
  - May be intracellularly/ extracellularly
  - Seen inside the PolyMorphoNucleocytes(PMNs)
  - **Remember:** PMNs are +ve (>5/HPF)



- **Culture test** - Thayer Martin Medium
- Nucleic acid amplification test (NAAT) is performed.

- **Gram stain**
  - No diplococci
  - Only PMNs are seen
- **Culture test** - No culture test.
- Nucleic acid amplification test (NAAT) is performed.

Complications

- **Male**
  - Prostate urethritis
  - Prostatitis
  - Perianal abscess
  - Epididymitis
  - Testis are not involved.
- **Female**
  - Salpingitis
  - PID
  - Bartholin gland abscess
- **Infants**
  - Ophthalmia neonatorum

- Complications will be more in NGU, as it is not symptomatic.
- Patients take more time to consult a doctor.
- **Chlamydia** - Causes PID, which leads to secondary infertility.
- **High chances of**
  - Arthritis
  - Perihepatitis (Fitz hugh curtis syndrome)

Treatment

Treatment of choice: Inj. Ceftriaxone 250 mg IM stat + Azithromycin 1 gm

Ceftriaxone not available: Cefixime 400 mg (oral) + Azithromycin 1 gm

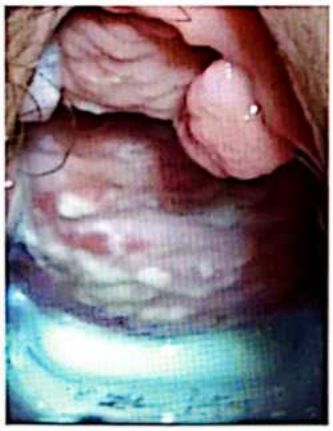
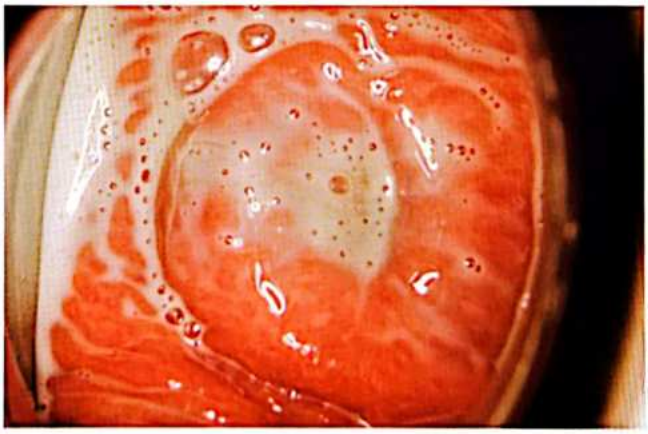
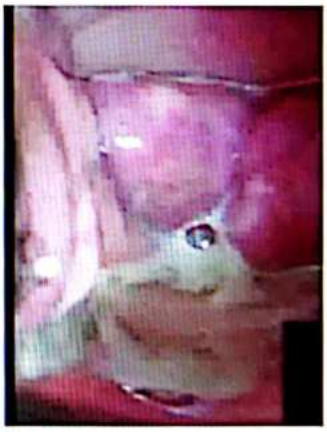
Treatment of choice: Azithromycin 1 gm stat

Azithromycin not available: Doxy 100 mg BD x 7days

To Remember:

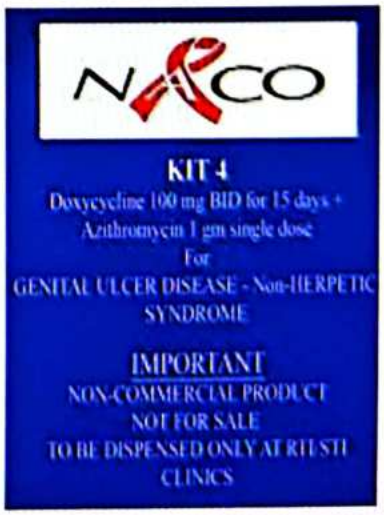
- Azithromycin is given, as there is always a chance that NGU is also present in GU.
- So for all GU we give NGU treatment as well.

Table 16.2

Vulvovaginal candidiasis	Trichomonal vaginalis	Bacterial vaginosis
Causative Organism		
Candida albicans (fungus)	Trichomonal vaginalis (protozoa)	• Altered flora (Anaerobic bacteria takes over the Lactobacilli) • Increase gram -ve anaerobes • **Examples** o Gardenella vaginalis o Mycoplasma hominis o Ureaplasma • Conc. of lactobacilli is reduced.
Population		
• More in DM • Immunocompromised	• Adverse pregnancy outcomes	• Not an STD • Nuns • It is an STI in which partner treatment is not done
To Remember: We do partner treatment in all STIs, except the Bacterial vaginosis.		
Symptoms		
Pruritus	Pruritus	May or may not be symptomatic
pH		
Acidic	Basic	Basic
Types of Discharges		
		
• Curdy white cheesy discharge • Premenstrual flare	Green frothy discharge	• Thin, homogenous, white discharge • Coating vaginal walls
Investigate by Smears		

• KOH smear • Pseudo hyphae & budding yeast cells	• Wet mount • Motile, pear shaped bodies Strawberry cervix is seen	• Gram stain • Clue cells (>20%) • Stippled appearance of vaginal squamous cells
Treatment		
Azoles	• Secnidazole 2g stat • Tinidazole 2g stat	• Secnidazole 2g stat • Metronidazole 500 mg TDS x 7 days

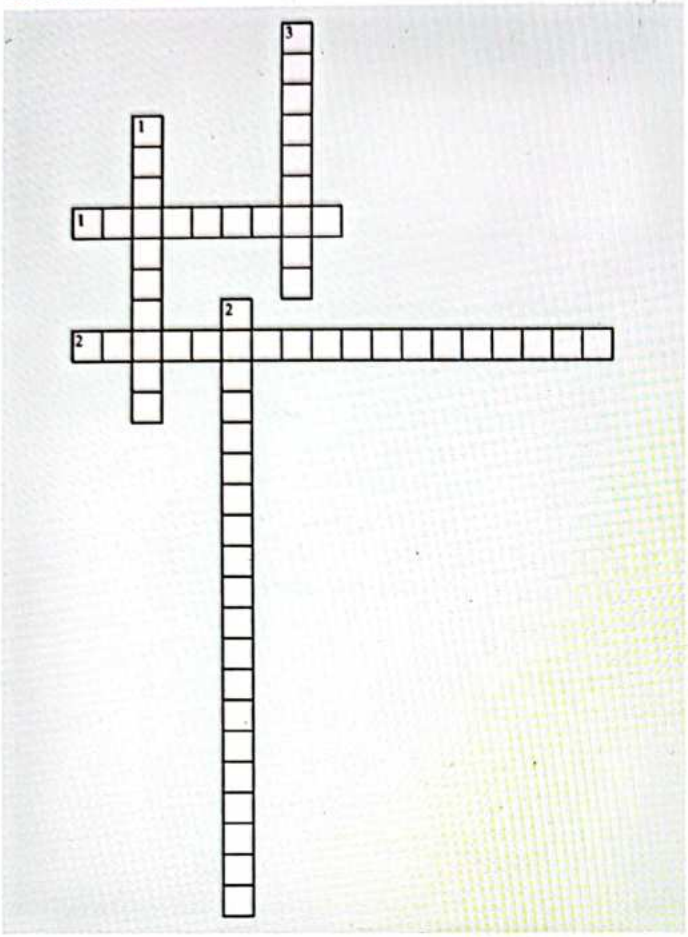
Table 16.3

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 tb)	
Genital Ulcer Disease (Non Herpitic)	KIT 2: 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	KIT4: Blue	Tab Doxycycline 100 mg (1 tab BD for 14 days) Tab Azithromycin 1g x 1 tab	

Genital ulcer disease (Herpetic)	KIT5: Red	Tab Acyclovir 400 mg x 1 tab TDS x 7 days	
Lower abdominal pain (Pelvic inflammatory disease)	KIT 6: Yellow	Tabl Cefixime 400 mg x 1 tab Tab Metronidazole 400 mg x (1 BD 14 days)Tab Doxycycline 1g (1 BD 14 days)	
Inguinal Bubo	KIT6: Black	Tb Doxycycline 100 mg (1 BD x 21 days) Tab Azithromycin 1g x 1 tab	

CROSS WORD PUZZLES

Crossword Puzzle



Down

1. Type of causes of STD discharge
2. Strawberry cervix is a feature of
3. STI Type

Across

1. Male complication
2. Vaginal discharge

17 ECZEMAS

- Usual meaning - to boil
- Dermatological meaning - to ooze

Dermatitis vs Eczema

00:00:29

Dermatitis	Eczema

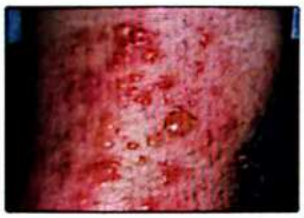
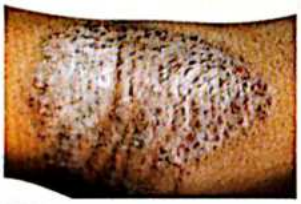
• Inflammation of skin.
• All dermatitis are not eczemas.
• Dermatitis can be
  o Psoriatic type
  o Eczematous type
• Broader term
	• All eczemas are dermatitis.
	• A part of dermatitis.

To Remember: All eczemas are dermatitis, but not all dermatitis are eczemas.

Clinical Stages of Eczema

00:01:17

- There are 3 stages.
  - Two are main

Acute	Chronic
	
Clinical Stage A • Recent • **Appearance** - Vesicles • **Oozing** - More • **Histopathology** - Ballooning (intracellularly) and spongiosis (intracellularly - stratum spinosum).	Clinical Stage B • Long term • **Appearance** - Dryer • **Oozing** - Less • Lichenification (pigmentation, more skin markings, skin thickening). • **Histopathology** - Acanthosis (increased thickness of stratum spinosum).

- **3rd stage** - Subacute eczema (not that important).

Classification of Eczemas

00:03:03

- This classification is based on
  - Exogenous - Outside the body.
  - Endogenous - Inside the body.

Classification	
Exogenous eczemas	**Endogenous eczemas**
• Irritant dermatitis • Allergic contact dermatitis • Photodermatitis • Phytodematitis	• Atopic dermatitis • Pityriasis alba • Seborrheic dermatitis • Discoid eczema • Hand eczema • Asteatotic eczema • Gravitational eczema • Lichen simplex chronicus • Prurigo nodularis

To Remember: Exogenous and Endogenous can occur with the other, as they are not mutually exclusive.

- **Exogenous**
  - Irritant - Contact with irritants.
  - Allergic contact dermatitis - Contact with external agents.
  - Photodermatitis - Aggravated by Sun.
  - Phyto dermatitis - Aggravated by Plant.
- **Endogenous** -
  - Maybe due to dry skin, or genes, or immunological responses that may predispose to eczema.
  - But all these are affected by external agents
  - Go through the classification flow chart.

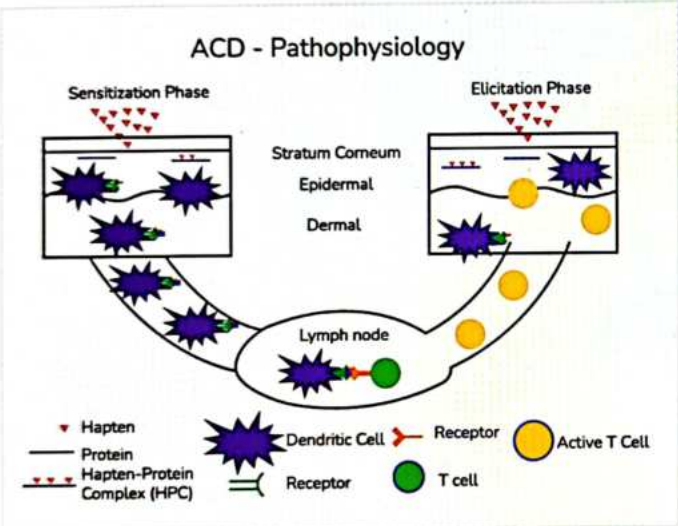
Exogenous Eczemas

- Two categories

ICD (Irritant Contact Dermatitis)	ACD (Allergic Contact Dermatitis)
Onset - Sudden (within a few hours). • **Example:** Going in the sun, exposure to lab chemicals. **Reason:** Non-immunological in nature.	**Onset** - Not Sudden (within a few days). **Reason** • Immunological in nature (Type-4 hypersensitivity). • Takes time to show immunological response.

Occurrence - First exposure	**Occurrence** - Subsequent exposure to antigens (in first exposure memory T Cells are formed). • Do not occur with 1st exposure.
Localized to the site of contact.	• Can spread beyond the area of content. • Gives immunological to second time exposure, which can spread beyond the contact location.
Depends on the nature of the irritant, so it is localized.	Depends on the nature of allergen, immunity of a person.

Pathophysiology of ACD



Explanation

1st Exposure
↓
Sensitization Phase
↓
Immune response by body (Antigen presenting cells, active T cells)
↓
T cells
↓
Memory T cells
↓
2nd Exposure
↓

Immune response
↓
Elicitation phase
↓
Allergic Contact Dermatitis

1. Irritant Contact Dermatitis (ICD) 00:09:59

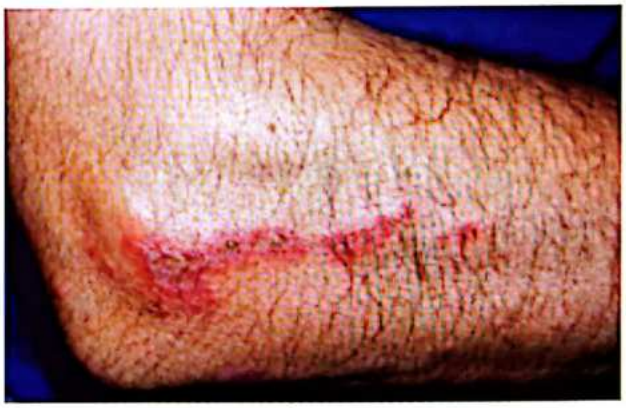
- **Main types**
  - Acute
  - Chronic

Acute ICD	Chronic ICD
Causes: Acids, Alkali, Solvents.	**Causes:** Detergents and Cutting oil. • Another name for detergent related ICD is Housewife dermatitis (seen mostly in housewives). • Cutting oil dermatitis - Seen in oil mechanics.

- **Other Type - Paederus Dermatitis**
  - **Other name:** Blister beetle dermatitis.
  - **Cause:** Blister beetle mosquito.
  - **Epidemiology:** Seen more in Monsoon.
  - **Irritant:** Blister beetle mosquitoes release Paedrin as irritant (thus the name Paederus Dermatitis).
  - **Appearance:** Overnight

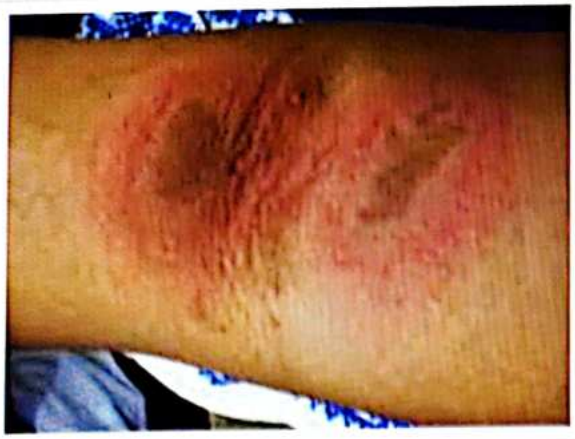
To Remember

Paederus Dermatitis (Whiplash dermatitis)



- Paederus Dermatitis also called Whiplash dermatitis.
- **Reason:** After mosquito bites people usually scratch along the irritant (Paedrin) and it creates a long whip like scratch on the skin.

Paederus Dermatitis (Kissing lesions)



Reason for Kissing Lesions: When the hand is bent at the elbow joint as a reflex due to mosquito bites, then the irritant will get settled on the opposite skin layer, leading to two lesions either side of the popliteal fossa.

2. Allergic Contact Dermatitis 00:13:18

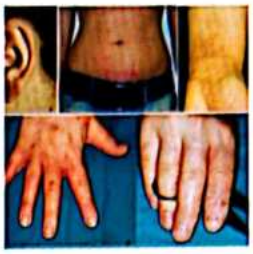
- Immunological - Type IV.
- Happens on subsequent exposure only.
- **Types**
  - Acute
  - Chronic
- **Causes - Allergens**
  - Metals
  - Cosmetics
  - Medicines
  - Clothing
- **Allergens and their Sources in ACD**
- Table of Allergens and their Sources in ACD

Allergens	Sources
Nickel, cobalt	Artificial jewellery, jean buttons
Chromium	Cement, Painting
Potassium dichromate	Leather, detergents, paint
Epoxy resins, phenols	Plastics
Parthenium	Plants
Propylene glycol	Cosmetics, medicaments
PPD	Hair dyes
Neomycin, gentamycin	Topical medications
Latex/ rubber	gloves, shoes, belts
PTBP	Bindi

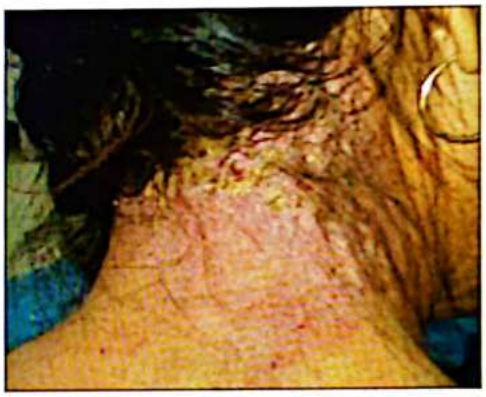
- Most common allergen is Nickel (artificial jewelry).
- In cementing - Potassium dichromate is common.
- Neomycin is a most common topical medication allergen.
- Many are allergic to Bindi due to PTBP (Para Tertiary Butylphenol).

Important Images

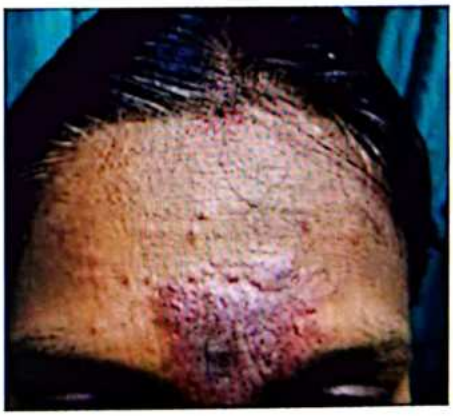
- Women with Artificial jewelry, Belt, Ring and Other



  - Explanation: Artificial jewelry has Nickel, which can cause ACD.
- Female with ACD along the hairline



  - Explanation: Hair dyes have PPD (Para Phenylene-diamine), which can cause ACD.
- Female with ACD around the Bindi Area



- Explanation: Bindi has PTBP (Para Tertiary Butylphenol) as an adhesive, which acts as an allergen.

One Liners

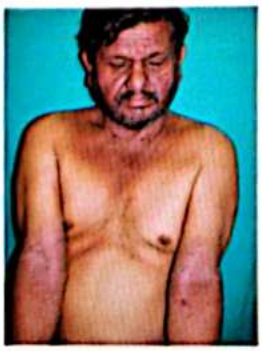
- Most common metal allergen: Nickel.
- Most common allergen in India: Nickel.
- Most common topical medicine causing ACD: Neomycin.

3. Phyto dermatitis 00:18:33

- Cause: Because of plants.
- Nature: Allergic/ Irritant/ combined with Photodermatitis.
- Common plants causing photodermatitis in India
  - Parthenium hysterophorus - Most common.
  - Xanthium strumarium
  - Chrysanthemums
  - Sunflower
  - Dahlia

Parthenium Hysterophorus

- Name of Disease: Parthenium dermatitis.
- Allergen: SQL (Sesquiterpene lactone)
- Population: Mostly farmers and people outside the city.
- Other names: Wild grass/ congress grass (Hindi: Junglee ghas, Safed phool wali ghas).
- Parts present: Pollens, stems, leaves, almost all parts of the plant.
- Clinical features: Only exposed parts of the body show dermatitis.
- Major affected parts: Face, neck, upper part of chest, upper back, flexures.

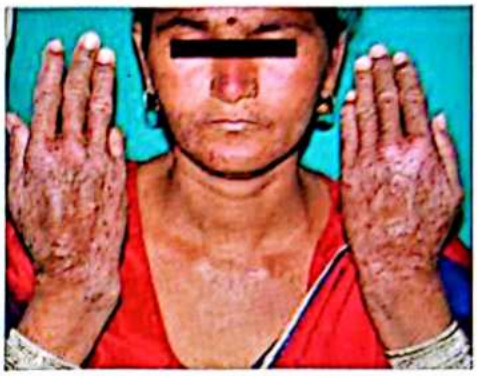


- Subacute eczema is majorly seen.

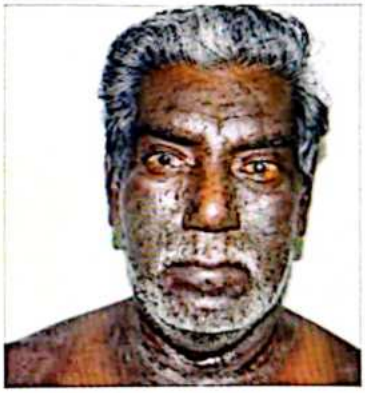
- **Subacute eczema**
  - Clinical Features: Dry and fissures
  - Locations: Face, cubital fossa, popliteal fossa, near collar bone.

To Remember: As Parthenium dermatitis is airborne, it is referred as Airborne contact dermatitis (APCD).

Exposed parts of hand



Leonine facies



Note: If Parthenium dermatitis is not treated on time, it may lead to Leonine facies or Lion like face.

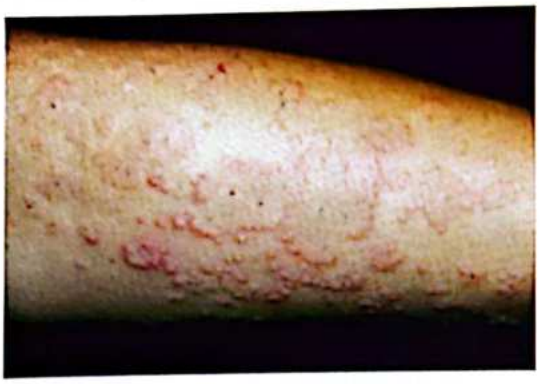
- **Other features**
  - Chronic relapsing
  - More in summer and monsoon (more pollens)
- **Treatment**
  - Remove the cause - Changing the occupation (practically not possible).
  - Pharmacotherapy - Antihistamines, corticosteroids, azathioprine.
  - Medical advice
    → Covering the body from exposure.
    → Taking a bath once home.

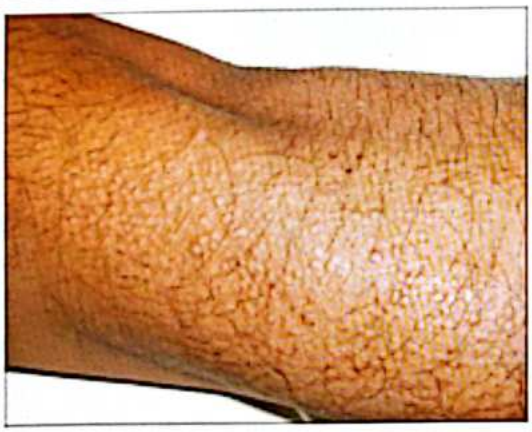
00:23:52

4. Photodermatitis

- Cause: Sun exposure.
- Can be
  - Phototoxic - Irritant in nature
  - Photoallergic - Allergic in nature.
  - PMLE

PMLE (Polymorphous Light Eruption)





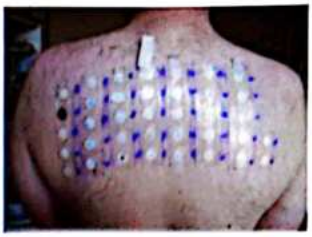
- Light Eruption - Eruption caused by light.
- Polymorphous - Different morphologies.
- Most common form of Immunologically mediated photosensitivity dermatitis.
- Appearance
  - Intense sunlight (summer and spring).
  - Recurring

- PMLE on Hand
- Features
  - Although the name is polymorphous, the lesions are monomorphic.
  - Shiny
  - Skin colored to erythematous itchy papules.
- Location (More exposed to sun)
  - Face
  - Neck
  - Forearms

Diagnostic Test for ACD

Patch Test

00:25:59



- Important diagnostic test for ACD.
- Indication
  - Helps to identify the allergen better, as it is difficult to rule out with just visual diagnosis.
  - Also used to rule out if it is ACD or not.
- Principle: Simulating the elicitation phase of ACD.
- Hypersensitivity: Type IV Hypersensitivity reaction.

Patient's back (Site)
↓
Patch with Finn chambers (white spots on the patch)
↓
Allergens (liquid or powder form)
↓
Placed in the chambers
↓
Labeled (1 to n)
↓
Patch placed on the site
↓
Secured using a micropore
↓
Patient sent home and returned after 48 hours
↓
Patch is removed
↓
Wherever there is erythema or vesiculation, patient is allergic to that

To Remember

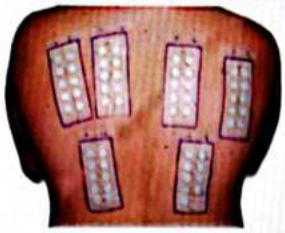
- Patch test is to be done on the back only, as it is easy, and the patient won't move more in that area.
- 48 hours are compulsory to elucidate the allergic reaction
- If nothing is found in 48 hours, the patient is to be called after 96 hours (as there are few allergens which are delayed reactors).

Q1. When do you take the patch test?
Ans

- If the option is 48 hours and 96 hours, then it is the best answer.
- If 48 hours and 96 hours are given separately, then the best answer is 96 hours (because at 48 hours some allergens may not show reaction).
- Sometimes it is done for 4-7 days as well.

Diagnostic Test for Photoallergic Dermatitis

Photopatch



- Antigen patches are applied in duplicate.
- One patch is covered with opaque material.
- Patient is called back after 48 hours and one of the patches is irradiated with UVA light.
- If positive - photoallergic dermatitis.

Interpretation (ACD & Photoallergic Dermatitis) 00:32:20

Reaction on non-irradiated	Reaction on irradiated side	Interpretation
Negative	Negative	No allergy, no photo allergy
Negative	Positive	Pure photo allergy
Positive	Negative	Allergy, no photo allergy
Positive	Positive	Allergy, with photo-exacerbation

Management of Exogenous Eczema 00:33:27

- Avoid irritant - Most important (treatment of choice)
- Topical steroids
- Oral steroids
- Azathioprine
- Cyclosporine

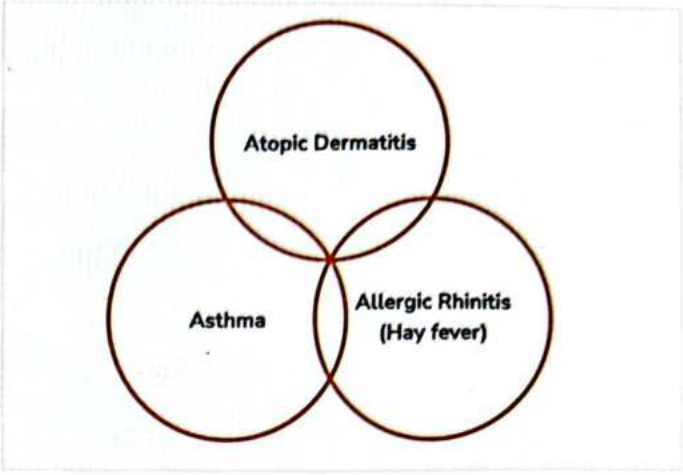
To Remember: Retinoids are not given, as they cause dryness which may lead to more irritation.

Endogenous Eczemas

- Cause: Due to the Predisposing factors Inside of the body

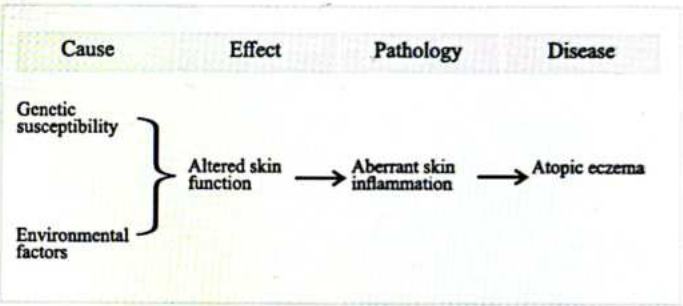
1. **Atopic Dermatitis**

- Chronic disorder - relapsing remitting.
- Always associated with itching (pruritus)
- Immunologically mediated
- Seen in atopic individual (proven to allergic)
- Other name: Itch that rashes.
- The itch is a disease here, as it is caused by a scratch and leaves rashes.
- Atopic individual
  - May have Atopic dermatitis/
  - Asthma/
  - Allergic rhinitis (Hay fever)
  - Or can have a combination of these three.
- Atopic Triad



  - Has any two, or all three of the above.
  - Personal or a family history of Atopic Triad may be seen.

- Pathogenesis of Atopic Dermatitis

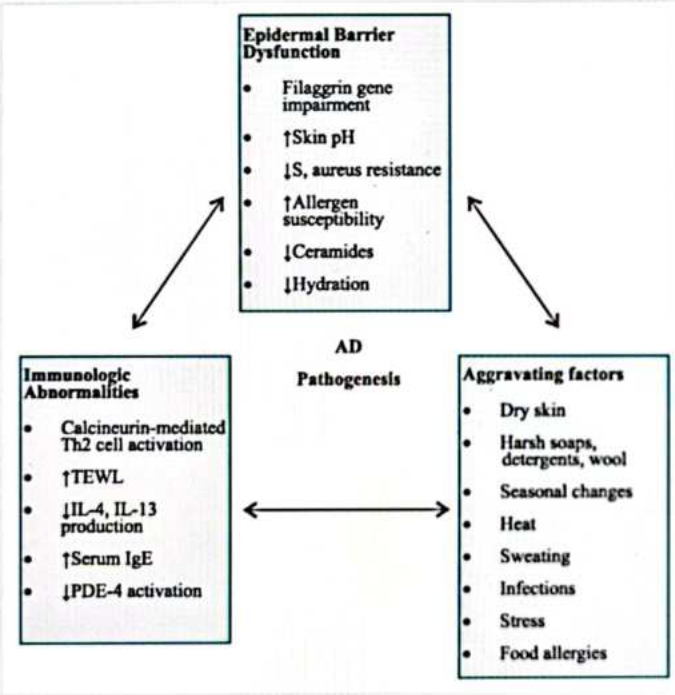


  - Genetic factors
    - → Filaggrin gene (FLG gene) defect (important in barrier function of skin)
    - → Immune genes - Increased IL-4, IL-13, leads to altered inflammation or immune response.
  - Environmental factors (Normally skin will not get affected with all these agents, but if the skin barrier is compromised, they show effects)
    - → Irritants
    - → Winter
    - → Wool clothing
    - → Excessive heat
    - → Sweating
    - → Some airborne allergens
    - → Food allergens
    - → Infections
    - → Stress
    - → Scratching

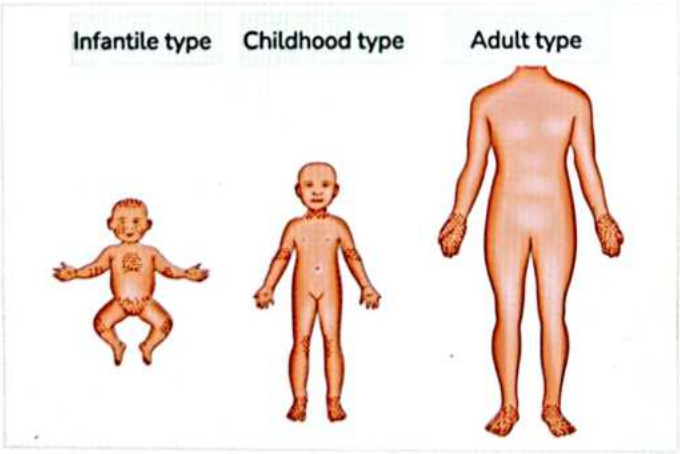
Pathogenesis of Atopic Dermatitis

Genetic factors
↓
Altered skin barrier (↓ Filaggrin, ↑Trans epidermal water loss)
↓ ← Environmental factors
Inflammation/ immune response
↓
TH_2 type response
↓
↑IL-4 and IL-13
↓
↑Eosinophils
↓
↑ Serum IgE

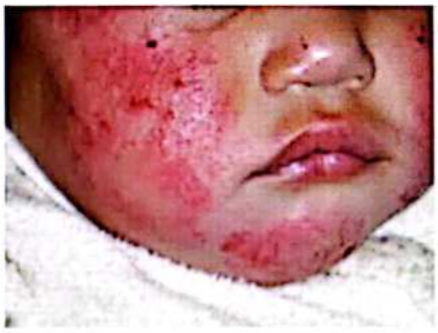
Atopic Dermatitis Pathogenesis



Clinical Features of AD 00:41:05



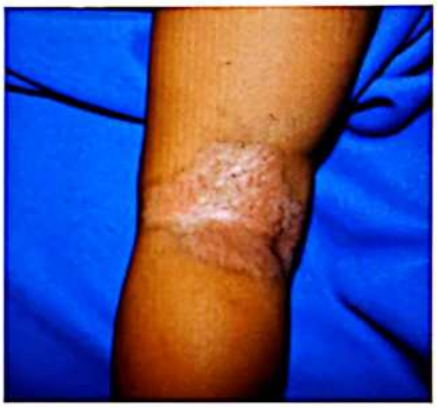
- 3 types based on age group
  - **Infants** - 3 months to 2 years



→ As they crawl, they come in contact with external factors
→ **Locations:** Face, scalp, trunk, diaper area, and extensor surfaces of extremities.

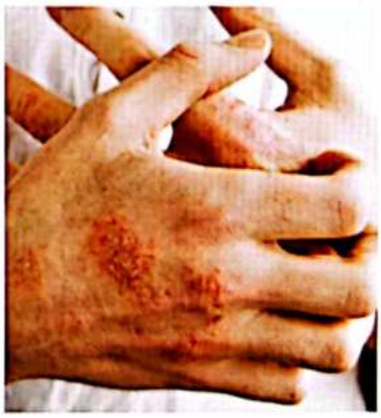
To Remember: AD is not present at birth, because it needs time to show.

- **Adolescent (childhood type)** - 2 to 12 years.



→ Allergens deposit on the flexural folds.
→ **Locations:** Antecubital fossa, popliteal fossa, neck, and ankles.

- **Adult type** - >12 years.



→ Indulge in lot of work
→ Locations: Hands, feet, also flexural folds.

To Remember: Whatever be the presentation, there will always be more itching.

Hanifin and Rajka Criteria for Diagnosis of AD (Major Criteria) 00:44:09

- Table of Hanifin and Rajka Criteria for Diagnosis of AD

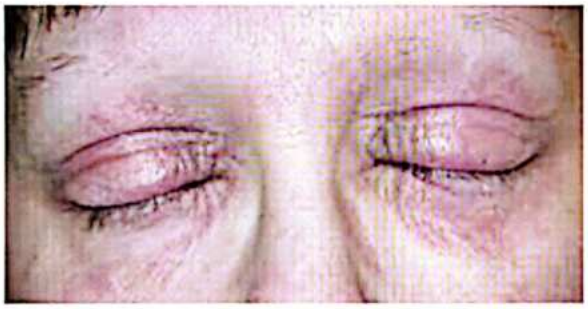
Major criteria

Hanifin & Rajka criteria for the diagnosis of AD

- Major criteria (must have three or more)
  1. Pruritus
  2. Typical morphology & distribution
     - Facial/extensor involvements in infants
     - Flexural lichenification in adults
  3. Chronic or chronically relapsing dermatitis
  4. Personal or family history of atopy (Asthma, allergic rhinitis, atopic dermatitis)
- AD is more a clinical diagnosis based on this criteria.

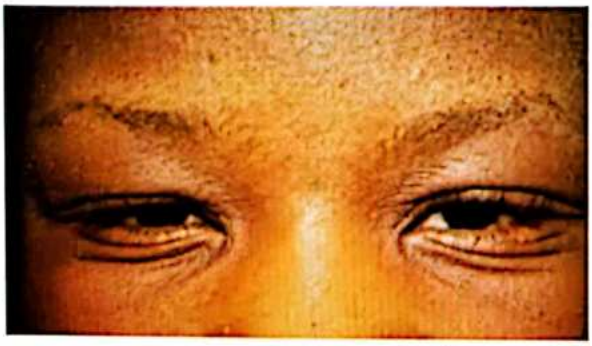
Minor Criteria

- Can have periorbital dermatitis.
  - Some can have Hertoghe's sign (loss of lateral 1/3rd of eyebrows).



  - Can have Dennie Morgan fold (extra fold of skin in the lower eyelid).

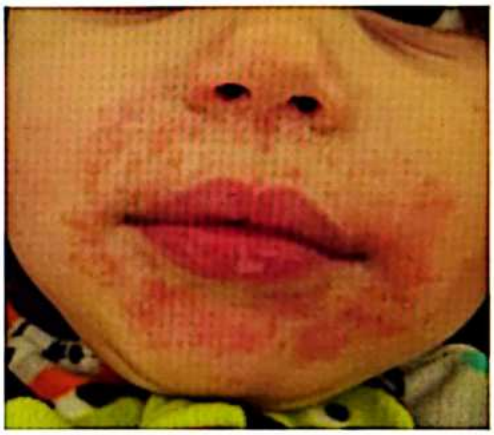
- Denny Morgan fold



  - Can also have allergic shiners.
  - Allergy in the eyes - leads to papillary conjunctivitis.
  - Also have Keratoconic cornea.
  - Anterior subcapsular cataract.

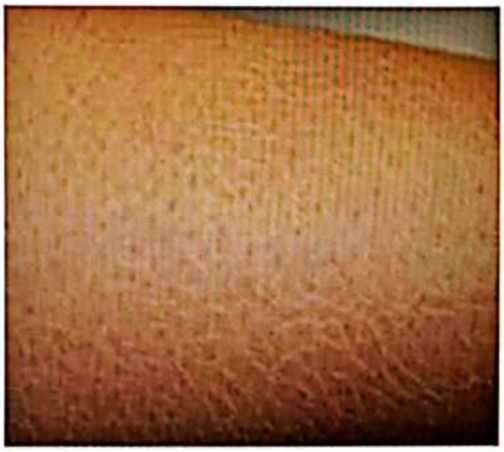
- Image of papillary conjunctivitis, keratoconic cornea, and anterior subcapsular cataract

- Can have perioral dermatitis (dermatitis around the oral mucosa)

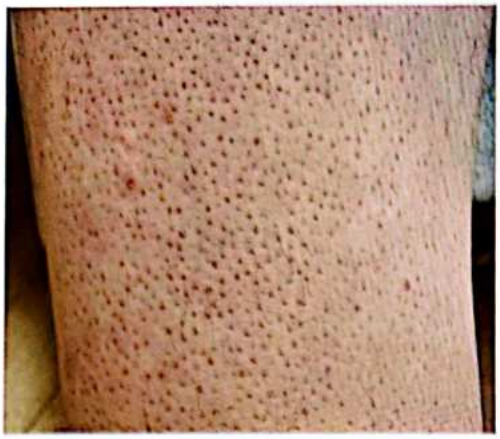


  - Cheilitis - Inflammation of the lips
  - Area of pallor - clear area.
  - Perioral dermatitis area
  - These 3 areas are arranged like a headlight, thus called a Head light sign.

- Dry skin or Ichthyosis

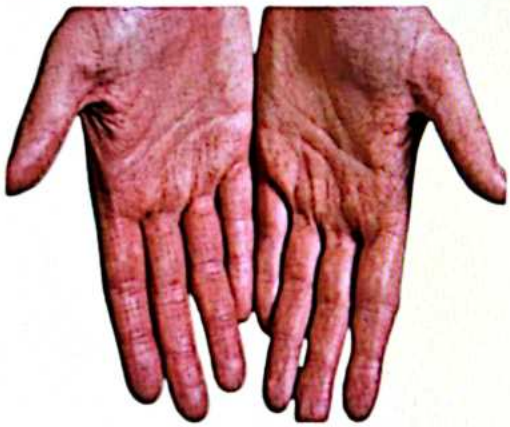


- Keratosis pilaris

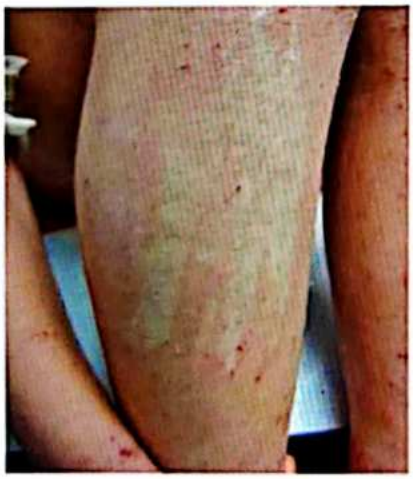


  - Hair follicles are occluded and hair gets coiled (also called strawberry skin)
  - Hyper keratotic follicular openings - gives a rough feel on the skin.

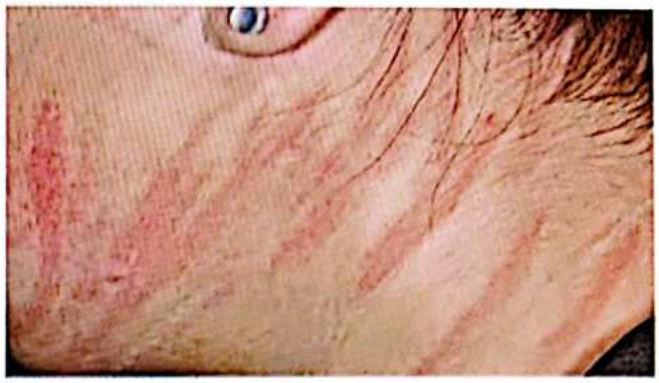
- Hyperlinear palms and soles



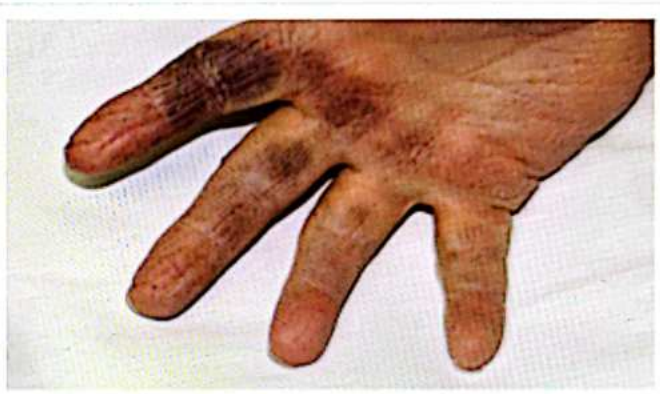
- White dermographism



  - Writing on the skin
  - Seen in AD
  - Due to vasoconstriction
- Red dermographism - seen in urticaria



- Black dermographism - seen in metal contact dermatitis



Complications of AD

- As skin barrier is compromised - AD patients are prone to get infections (Reason: Staph colonization).
- Eczema herpeticum can be seen (discussed in chapter viral infections)
- Can also lead to Erythroderma

Diagnosis of AD

- Clinical diagnosis - Choice
- IgE test high
- Hanifin and Rajka criteria can be used

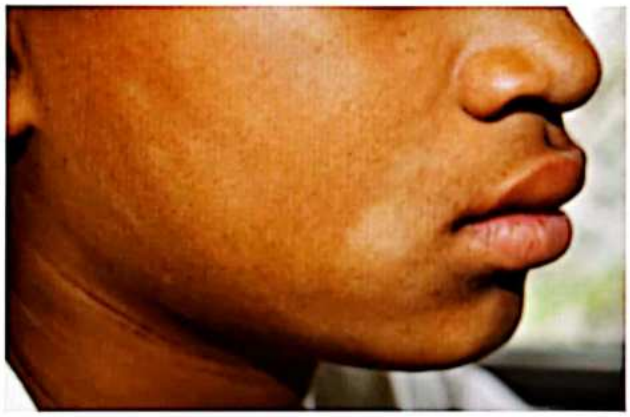
Management 00:51:24

- Genetic - Nothing much can be done.
- Environmental factors - Removing triggers
  - Avoid dust
  - Avoid extreme temperature
  - Avoid detergents
  - Avoid harsh soaps
- Barrier compromised - Hydration (to apply emollients and moisturizers)

- Immunological factors - Giving pharmacological treatment
  - Antihistamines
    - → Localized condition: Topical corticosteroids + Antibiotics
    - → For face: Calcineurin inhibitors (pimecrolimus and tacrolimus)
    - → Severe disease (systemic management): Oral corticosteroids, cyclosporine, and azathioprine.
  - Newer drugs
    - → Crisabole - PDE-4 inhibitor (topical).
    - → Dupilumab - IL-4 receptor alpha antagonist (systemic).
    - → Tofacitinib - JAK kinase inhibitors
    - → Apremilast - PDE-4 inhibitor (also used in psoriasis)

1. Pityriasis Alba

00:54:57



- Pityriasis - Scaly
- Alba - White
- **Clinical presentation**
  - Usually in children
  - Atopic
  - Hypopigmented asymptomatic macules on face
  - Macules are associated with mild scaling
- **Treatment**
  - Topical emollients
  - Topical calcineurin inhibitors

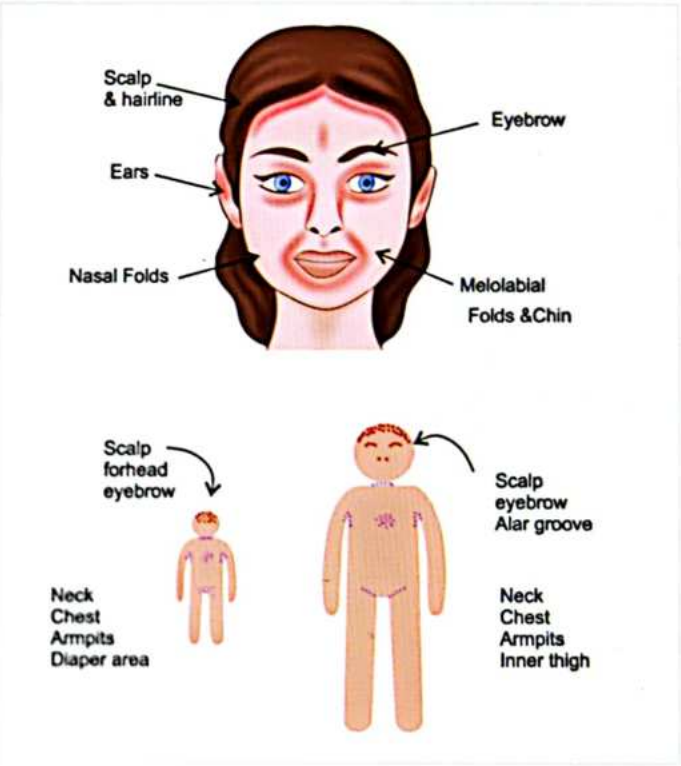
How is Pityriasis Alba different from other dermatological diseases?

- Vitiligo - Has deep pigmentation and it is not scaly.
- Indeterminate Hansen - Single lesion and is seen in the endemic population only.

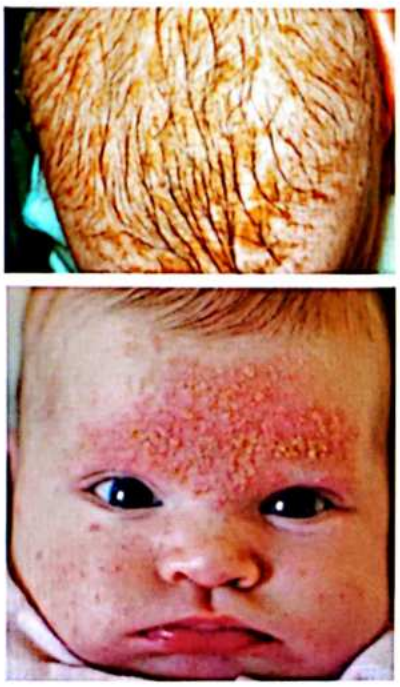
2. Seborrheic Dermatitis

00:57:22

- Present in Seborrheic area (scalp, face, chest, upper back, upper trunk)
- **Pathogenesis:** Increased sebum
- **Organism:** Malassezia
- **Age**
  - Infants - Usually happens in <3 months old due to maternal androgens in them.
  - Adults and Adolescent - Major activity of sebaceous glands is seen
- Sites of Seborrheic Dermatitis

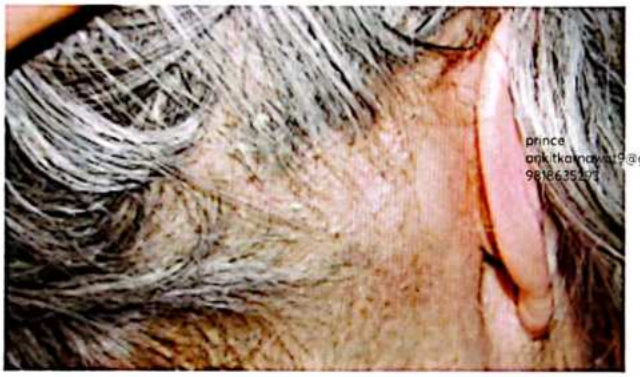


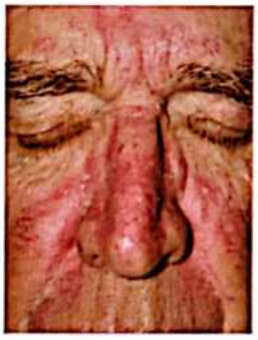
- **Clinical Presentations of Seborrheic Dermatitis**
- In <3 month old (Infantile Seborrheic Dermatitis)

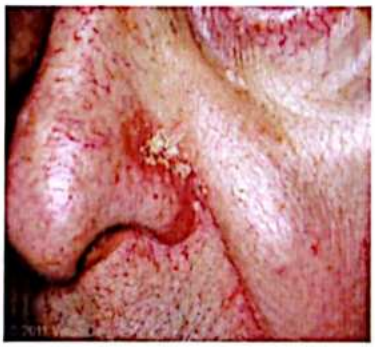


- Cradle cap is seen - Yellowish adherent crust or scales of scalp, also seen on the face.

- **In adults**
- Usually dandruff (Malassezia over colonization)



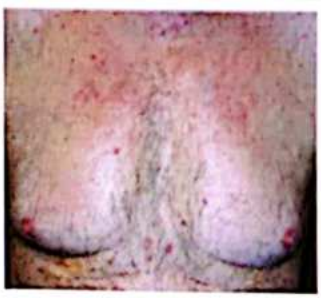


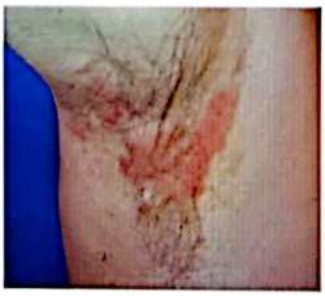


- Inflammation with greasy scales - around nasolabial folds, eyes, forehead

To Remember: Greasy looking scales are a typical feature of Seborrheic Dermatitis

- May be present at chest or underarms

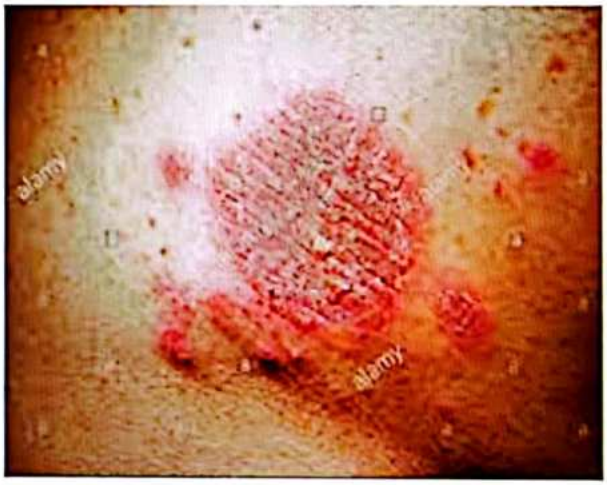




- **Management of Seborrheic Dermatitis**
- Stop Malassezia growth - Antifungals
- No oil application, as sebaceous gland activity is already increased.
- Topical - Azoles (Sertaconazole, Propiconazole, Luliconazole)
- Scalp - Shampoo
  - Azole based
  - Zinc pyrithione (ZPTO) based
  - Ciclopirox Olamine based
  - Selenium sulfide based
- If dry erythema
  - Topical corticosteroids + Shampoo or Azoles
  - Topical calcineurin inhibitors + Shampoo or Azoles
- If Itchy - Antihistamines
- If Severe - Oral antifungals (itraconazole, fluconazole).

To Remember: Only azole class of antifungals are used in Seborrheic Dermatitis.

3. **Discoid Eczema** 1:04:03



- Disc like or coin like
- Other name: Nummular Eczema (ring shaped)
- Present on any part of the body.

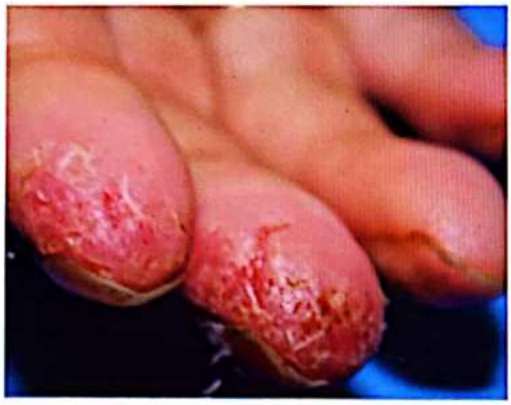
4. **Hand Eczema** 01:04:46

- Both exogenous and endogenous

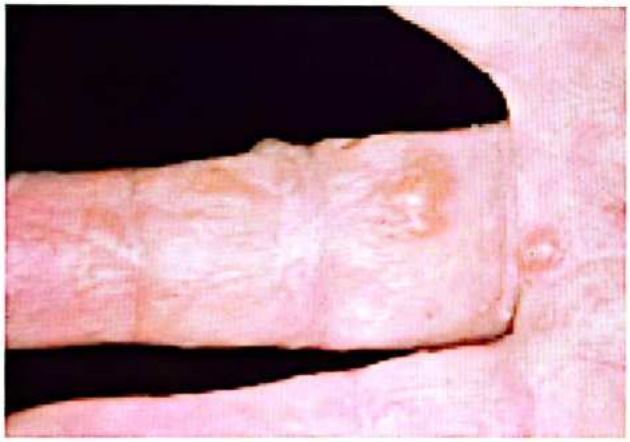
- **Manifestations**
  - ACD
  - ICD
  - Focal peeling
  - Hyperkeratotic
  - Pompholyx

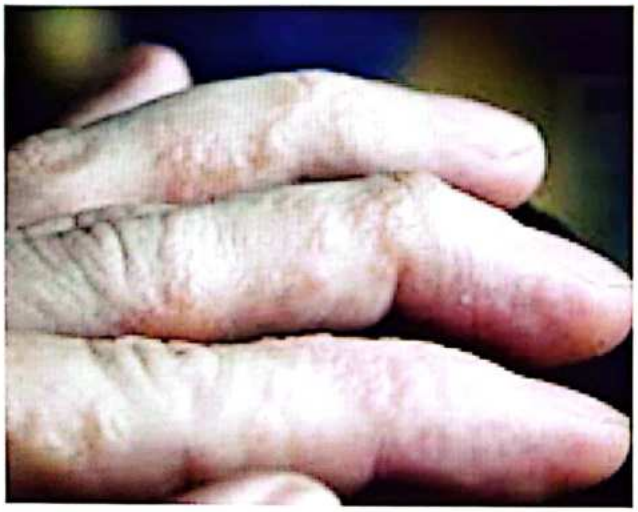
- **Fingertip Eczema**

  - Hand Eczema on Fingertips (Fingertip Eczema)



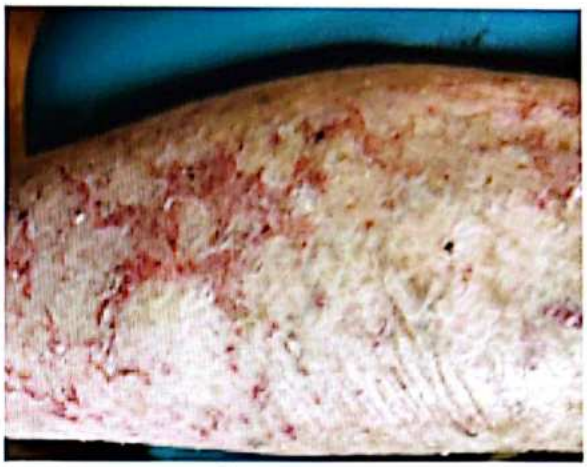
- Commonly seen in housewives
- In adults - when working for longer time
- **Pompholyx Eczema**





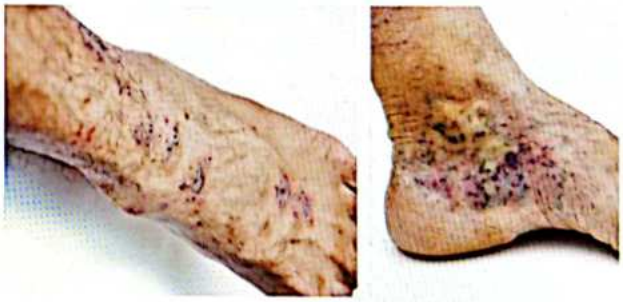
  - Endogenous hand eczema type
  - Present bilaterally
  - Deep seated itchy vesicles are seen
  - Look like sabudana, thus the name Sago grain vesicles
  - Very chronic in nature, if untreated.

5. Asteatotic Eczema 01:06:55



- **Asteatotic** - No fat, thus the name NO fast rash.
- **Age**
  - Seen in elderly people, usually lower legs.
  - Due to less fat and dry skin
- **Season:** Winters or Dry seasons
- **Appearance**
  - Cracked skin (cracked porcelain or crazy paving pattern, dried riverbed pattern)
- **Other names:** Eczema craquele, Winter eczema.

6. Venous Eczema 01:08:30



- Seen in venous insufficiency
- Usually seen in Varicose veins
- **Mechanism**

Blood pooling in the veins
↓
Skin becomes weak
↓
Eczema over the area

- **Other names:** Gravitational/ stasis eczema.
- **Location:** Medial side of leg or ankles.
- **Clinical presentations**
  - Dilated blood vessels
  - Atrophy
  - Hemosiderin deposition

153

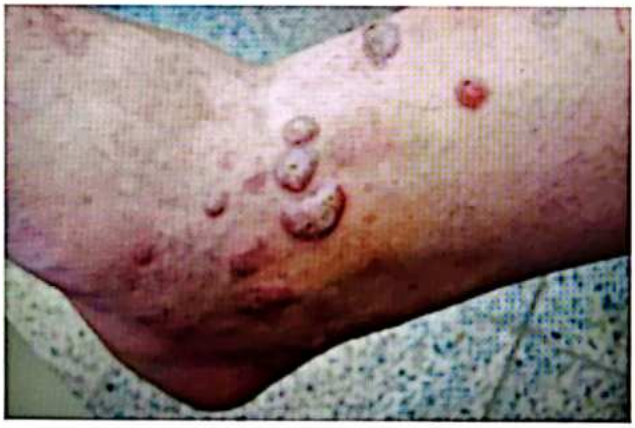
7. Neurodermatitis 01:09:41

- Neuro - Nerve

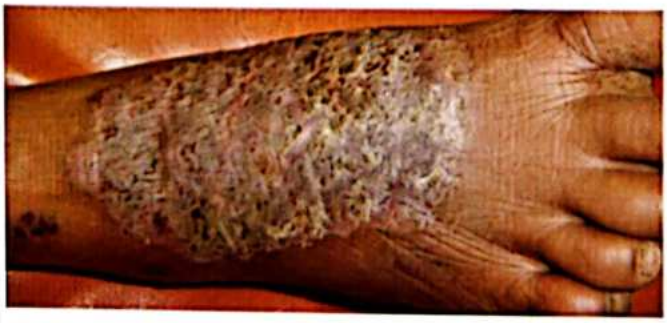
Itch Scratch Cycle

Because of neurological or psychiatric issues
↓
Patients scratch at particular areas more
↓
More itching
↓
More scratching
↓
Eczema
↓
Inflammatory response
↓
More itching
↓
More Eczema

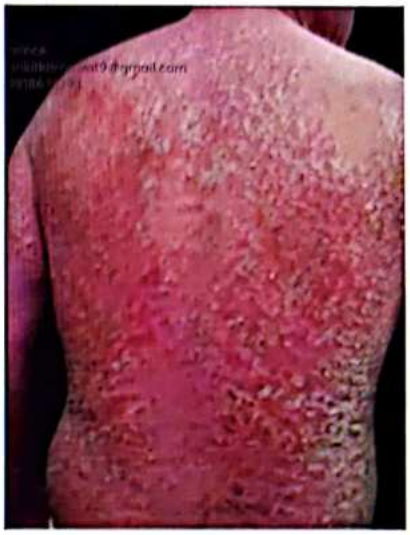
- Patient themself do it no other cause
- **Clinical manifestations**
- **Location:** Accessible parts of the body (as only these can be scratched)
- Prurigo Nodularis - Shows hyperkeratotic nodules



- Prurigo Simplex - Only papules are seen.
- Lichenification (Lichen simplex chronicus) - Severe itching



8. Exfoliative Dermatitis 01:12:39



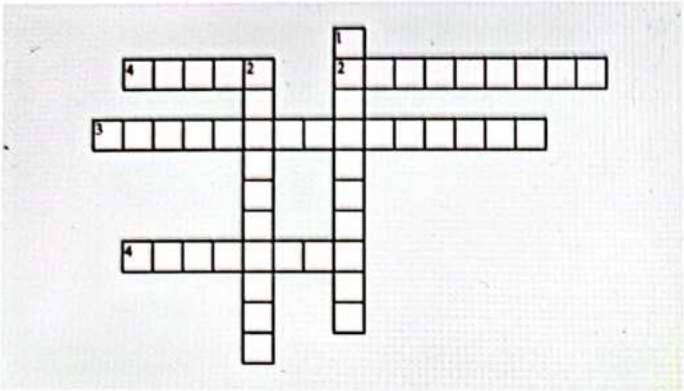
- **Other name:** Erythroderma (briefed in psoriasis lecture)
- Erythema + Scaling involving >90% of body surface area
- Underlying chronic inflammation
- Can lead to systemic complications
  - Thermoregulatory failures
  - Temperature differences
  - Loss of proteins
  - Loss of calcium
  - **May have**
    → Lymphadenopathy
    → Hepatosplenomegaly
- **Common causes**
  - Most common - Pre Existing dermatoses (Psoriasis, ACD, PRP, Ichthyosis, Scabies, Pemphigus foliaceous)
  - Drugs - Antiepileptics, Antibiotics (sulfonamides, penicillin, and vancomycin), lithium, and allopurinol.
  - Malignancies - Skin malignancies (CTCL, B-Cell CLL) and solid organ malignancies.
  - Idiopathic (unknown cause)
- **Clinical features**
  - Erythema + Scaling (>90% of body surface area)
  - Fever + Other constitutional symptoms
  - Lymphadenopathy (LAN)
  - Hepatosplenomegaly (HSM)
- **Management**
  - Can go into circulatory and thermoregulatory failures, thus daily monitoring is required.
  - Oral fluids are to be given
  - So, the majority are handled as inpatients.

To Remember

- If not managed can lead to septicemia.
- Most of the drugs are also avoided because it can be due to drugs.

CROSS WORD PUZZLES

Crossword Puzzle



Down

1 What is the condition that cause inflammation of skin?

Across & Down

2 What are the two classifications of eczemas?

Across

3 What is caused because of plant?

4 Name two clinical stage of Eczema?

18 PIGMENTARY DISORDERS

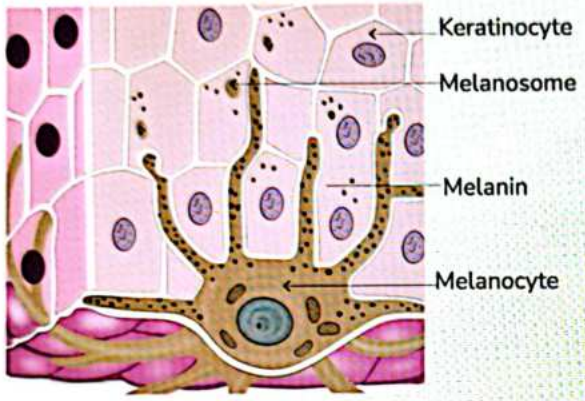
- Hyper pigmentary disorder: Increase in pigmentation
- Hypo pigmentary disorder: Decrease in pigmentation
- Depigmentation: Complete absence of pigmentation

Skin color

00:01:19

- Melanin
- Hemoglobin
- Carotenoids
- Constitutive (genetic) and facultative (modified: sun exposure, hormones)

00:02:30



- Melanocytes are pigment forming cells which are present in the basal layer of the skin. They are present in the ratio 1:10 and they transfer melanosomes to 36 keratinocytes which is known as epidermal melanin unit which is 1:36.
- Epidermal melanin unit makes skin color uniform.
- Skin color is not defined by melanocytes.
- Skin color is defined by type of melanin and distribution and size of the melanosomes.

Types of melanin

00:04:40

- Eumelanin: Brownish to blackish in color
- Pheomelanin: Orangish-red in color

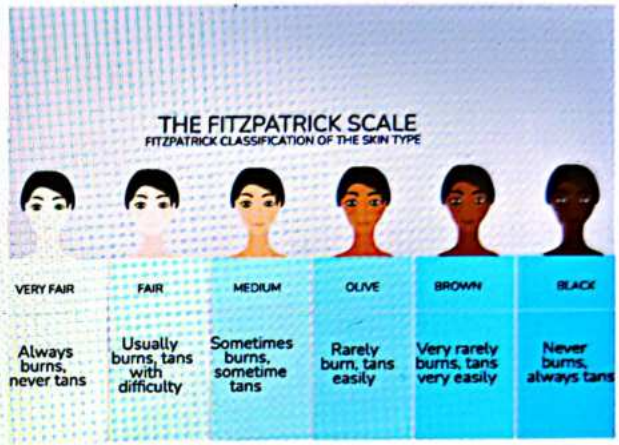
Formation of Melanin

00:05:47

- Tyrosine is the precursor.
- Tyrosine by the tyrosinase enzyme gets converted into L-DOPA, DOPAquinone that further forms eumelanin and pheomelanin.

The Fitzpatrick scale

00:06:25



- Lightest skin color means more pheomelanin and darkest skin color means more eumelanin.
- Indian comes in medium, olive, and brown skin type category
- Eumelanin is more protective that is why fitzpatrick 1 and 2 are more prone to skin cancers, and sunburns.
- Eumelanin has less risk of skin cancer but they have more tanning, which is a protective phenomenon.

Classification

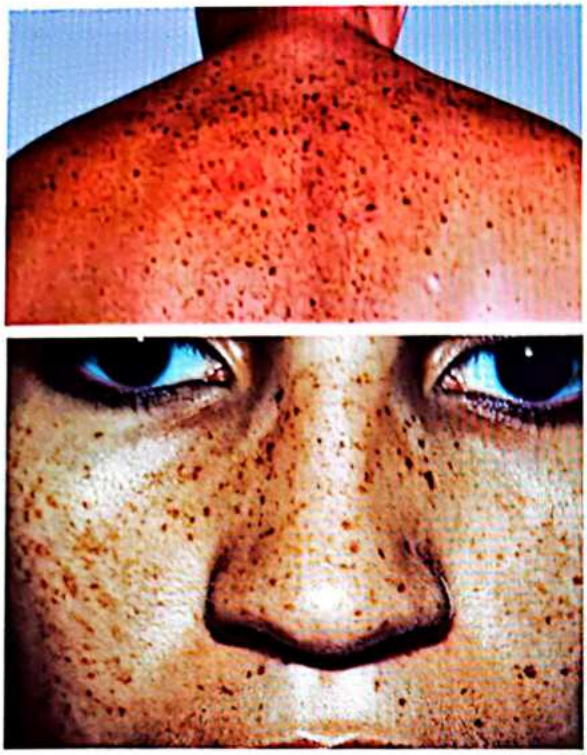
00:08:22

1. Hypo pigmented disorder
2. Hyper pigmented disorder
   a. Epidermal pigmentation: more brownish
   b. Dermal pigmentation: more blueish

- Whenever light falls on the skin, the light gets reflected back.
- If the pigment is deeper, the light that will reflect is of shorter wavelength.
- If the pigment is superficial, the light that will reflect is of longer wavelength.
- Tyndall effect: It is the differential reflection of the light based on the depth of the pigmentation that changes the color of lesion.

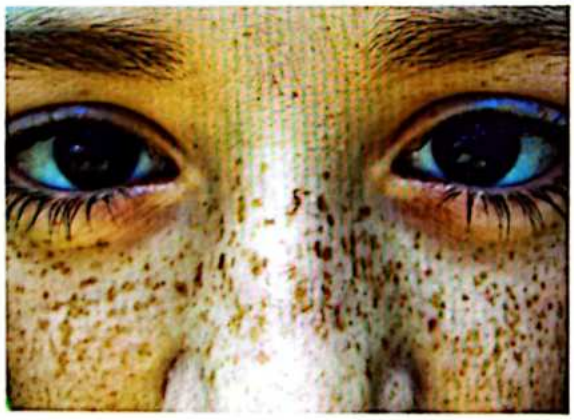
Disorders of hyperpigmentation

1. Lentigo 00:11:11



- Meaning: lentil like
- Clinical features: dark brown hyperpigmented macules which can be present anywhere on the body.
- Pathology: Increased melanocytes
- Syndromes
  - Leopard
  - Cronkite Canada
  - Peutz Jeger's (periorificial lentigines)

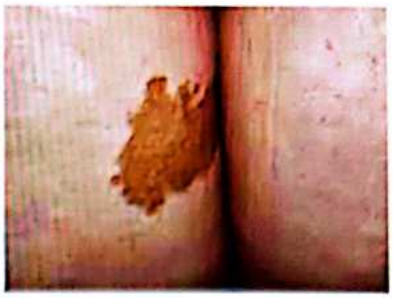
2. Freckles 00:12:00



- They are lighter brown in color (lighter than lentigines).
- They are asymptomatic macules.
- More common in summers or when there is direct sun exposure

Refer Table 18.1

3. Cafe AU LAIT Macules 00:13:08

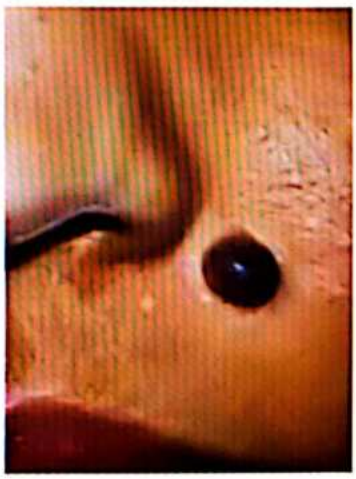


- Cafe Au Lait means coffee on milk.
- Clinical features: Hyperpigmented macules, asymptomatic.
- They have serrated borders.
- Age: At birth
- Pathology: Increased melanosomes
- If they are less than 3 in number and if they are 0.5 to 1.5 cm in nature, they are benign.
- If they are multiple in number then they may be associated with certain syndromes like neurofibromatosis, TS and others.

4. Melanocytic nevi 00:15:24

- They are also called moles.
- When the melanocytes derive from the neural crest, they are transferred from the neural crest . They decide to form a nest like place, this nest of proliferating Nevus cells is called a melanocytic nevi.
- Classification
  1. Congenital
  2. Acquired
- Nevus cells can proliferate and be there in the epidermis or dermis.

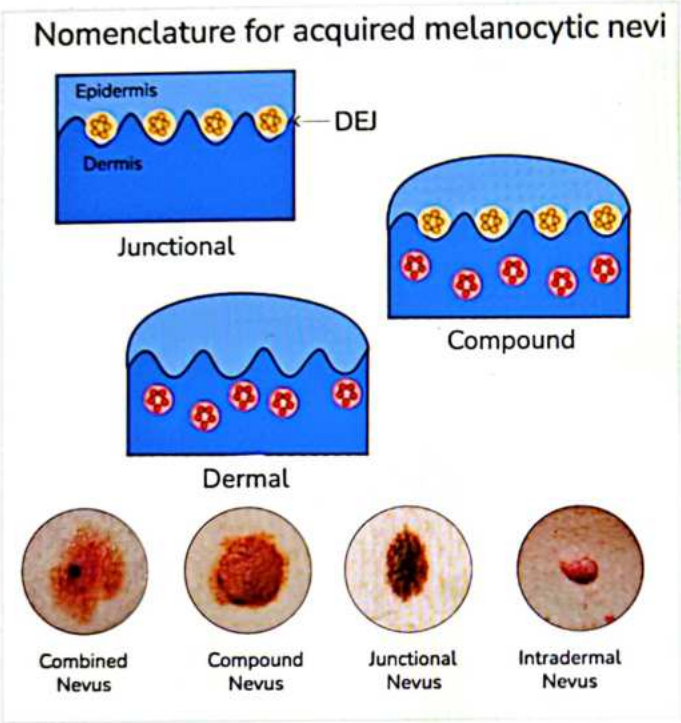
1. Acquired Melanocytic nevi 00:17:00



- Nest of proliferating nevus cells is seen
- Defect: Proliferation in epidermis

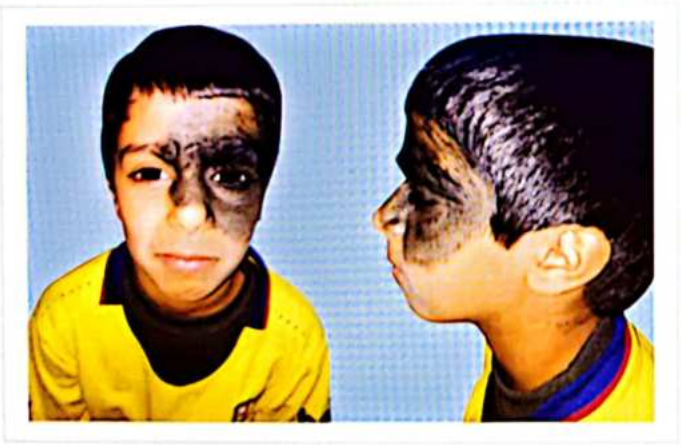
Types of acquired melanocytic nevi

- Junctional nevus: When they are present in the dermoepidermal junction. It is small and dark brown in color.
- Compound nevus: When they are present in the dermoepidermal junction and dermis. It is slightly raised and lighter in color.
- IntraDermal nevus: When it is in the dermis. It is raised papules and is skin coloured or red in color.



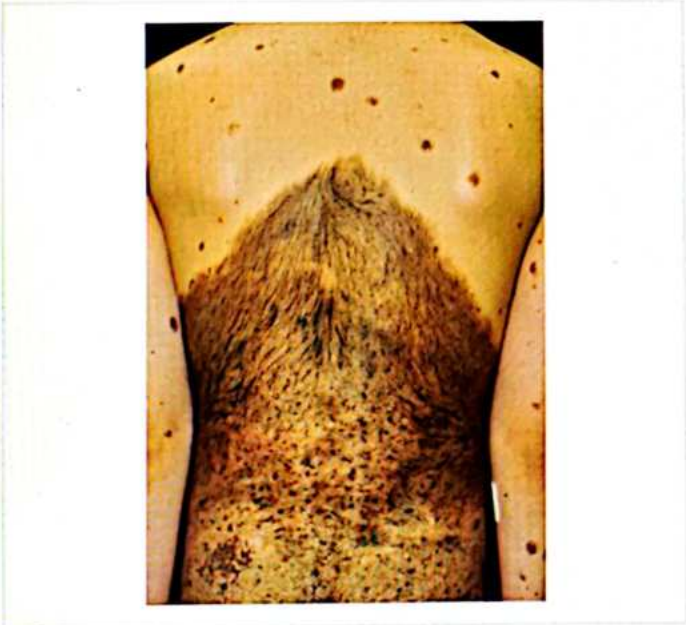
2. Congenital melanocytic nevi

00:19:19



- **Congenital melanocytic nevi**
  - Present at birth with a solitary lesion.
  - Associated with hypertrichosis and rugosities

- **Bathing trunk/ garment nevi**
  - A giant CMN
  - This might have some malignant potential.



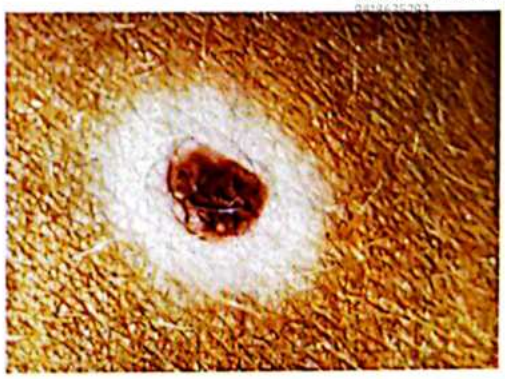
3. Nevus Spilus

00:21:05

- It is also known as speckled lentiginous nevus.
- It has a tan background and over it are speckled hyperpigmented macules.

4. Halo Nevi

00:22:00



- When a nevi has a halo aroùnd it.
- There is autoimmune destruction of melanocytes.
- When the body mounds, the body responds against this mole, so melanocytes have been destroyed but the surrounding skin is normal. But the autoimmune system does not know that it is normal. So as a result, the melanocytes in the surrounding skin also gets damaged. So it appears depigmented.
- The treatment is to leave it like this or remove the mole. After removing the mole, it will go away because the immune system is directed against this mole.

Treatment of epidermal melanocytic nevi

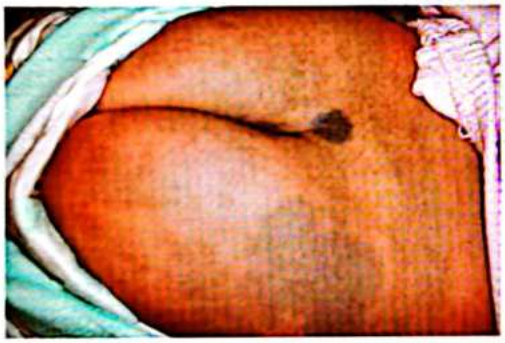
- It is treated only for cosmetic reasons unless there is a change in shape, color, size then there comes malignancy.
- It can be treated by electrocautery, laser, and surgical excision.

5. Dermal Melanocytic Nevi 00:24:33

- Here proliferating nevi cells are in dermal and they are bluish in color.
- Three types of dermal Melanocytic nevi are there:

1. Mongolian spots
2. Nevus of OTA
3. Nevus of ITO

1. Mongolian spots

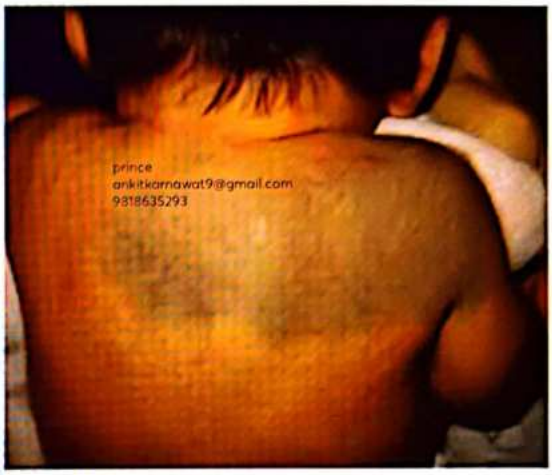


- Present at birth
- Commonly seen in mongoloid races.
- Bluish macules which are asymptomatic seen in sacral areas.
- Spontaneous resolve

2. Nevus of OTA

- It is also known as nevus fusco caeruleus opthalmomaxillaries.
- Most commonly seen in Japan
- They are unilateral and developed along the ophthalmic and maxillary division of the trigeminal nerve.
- It affects eyes, temple, spiral vital area, forehead and little part of the nose.
- Speckled brownish to bluish macules present not only on skin but also on sclera.

3. Nevus of ITO

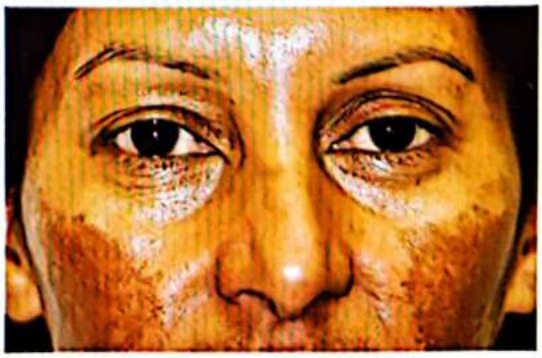


- Bluish macules seen in supraclavicular, scapular, and deltoid regions.
- It is called nevus fusco caeruleus acromeoclavicuralous.
- It is along the nerves of lateral brachial, post supra scapular nerves.

Treatment of Nevus of ITO and Nevus of OTA

- It does not resolve spontaneously, it needs to be treated unlike mongolian spots which resolves on it's own.
- It is treated for cosmetic reasons by using lasers that are pigment specific.
- These lasers are Ruby lasers, Alexandrite lasers, ND yag lasers.

5. Melasma 00:29:39



- Most common cause of facial pigmentation
- It presents with hyper pigmented macules on the face.
- It involves cheeks, forehead, chin, nose etc.
- Melasma is triggered by sun, pregnancy, OCP and in thyroid disorders. In pregnancy, it is referred to as chloasma.
- In melasma, melanocytes are hyperactive so they are larger and more dendritic so they produce more melanin. This defect is hyperactive melanocytes.
- Site: Convex areas of face and any other area.
  - Centro facial: Nose, cheeks, forehead, and chin.
  - Malar
  - Mandibular
- Type: based on the level of pigmentation
  1. Epidermal: More blackish-brownish color. It will be more amenable to treatment or easy to treat.
  2. Dermal: More bluish in color. It will be resistant to treatment. Lasers have to be used in this.
- Wood's lamp test is used to prove whether it is epidermal or dermal melasma.
- Accentuation will be seen in epidermal whereas dermal melasma will not be changed. Sometimes it can be both epidermal and dermal melasma.

Treatment of Melasma

- Sun protections are to be used such as sunscreen, goggles, hats, etc.

Topical treatments

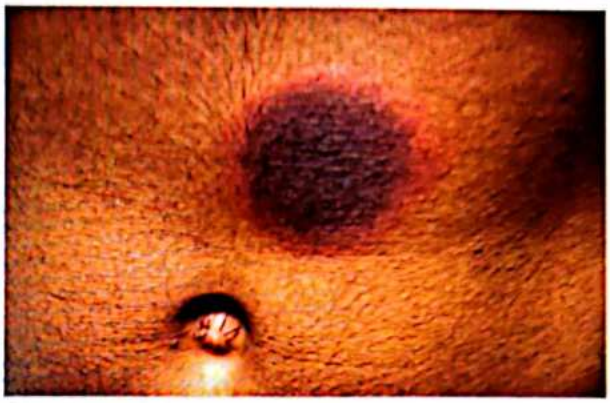
- Hydroquinone, glycolic acid, azelaic acid, and kojic acid are used.
- Kligman's formula is used in this, it is a combination of 3 agents- hydroquinone (act as depigmented agent), topical retinoid (act as exfoliant), and mild topical steroid (reduces the inflammation that is present and is caused by retinoids)
- Chemical peels are topical agents in a concentrated manner and done by physicians. The purpose of it is glycolic, azelaic acid, kojic acid, lactic acid, topical retinoids are all present in higher concentration, it is applied on patients face, kept for 5 minutes and then washed off.
- These are concentrated chemicals which penetrate deepers, causing exfoliation of the skin.

Systemic treatments

- Tranexamic acid, and glutathione are used.
- Lasers can also be used for these patients.

Agents used		
Tyrosinase inhibitors	**Melanocyte cytotoxic**	**Others**
• Hydroquinone, • Arbutin • Kojic acid • Licorice acid • Vitamin E	• Azelaic acid	• AHA • Resorcinol • Vitamin C • Tretinoin

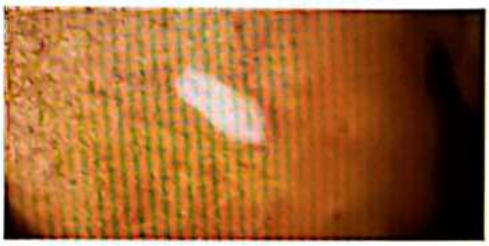
- **Fix Drug Eruption**



6. **Diffuse Hyperpigmentation** 00:38:00

- Addison's disease
- Haemochromatosis (bronze diabetes)
- CRF
- HIV infection and AIDS
- Drugs
  - Clofazimine
  - Antimalarials, minocycline
  - Chlorpromazine
  - Amiodarone
  - Anticancer agents

7. **Nevus Depigmentosus** 00:38:39



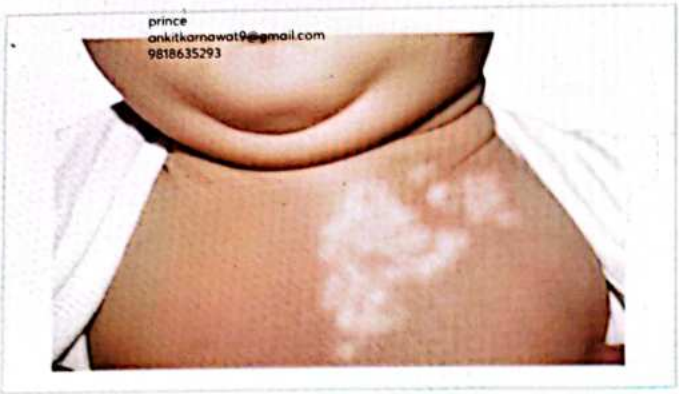
- Single hypopigmented macule in a dermatomal distribution.
- It is unilateral and present from birth.
- It is asymptomatic
- Seen in trunks and limbs.
- Congenital, stable, does not cross midline, melanosome not transferred to keratinocytes

Vs

- Congenital Vitiligo: Since birth, increase proportional to body growth, completed depigmented

8. Nevus Anemicus

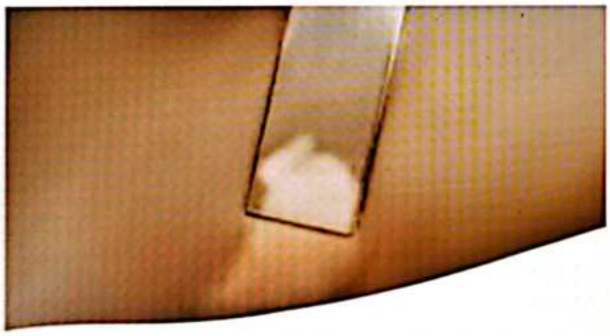
00:41:38



- It is a pharmacological nevus.
- Capillaries are hyper responsive to catecholamines due to this there is vasoconstriction of those capillaries. Because of this vasoconstriction, the blanching effect is there. This blanching effect appears as white areas known as nevus anemicus.

On Diascopy

- If there are depigmented macules, then it can be nevus anemicus or nevus depigmentosus. So to differentiate, a diascopy test is used.
- A glass slide is taken and pressed on the lesion.



Nevus depigmentosus
Margins do not change

Nevus Anemicus
Margins will merge with the normal skin

9. Albinism

00:43:59

- Congenital universal absence of eumelanin synthesis in the skin and hair follicles and eyes
- Pathogenesis: Failure of synthesis of tyrosinase or defect in tyrosinase activity

Types

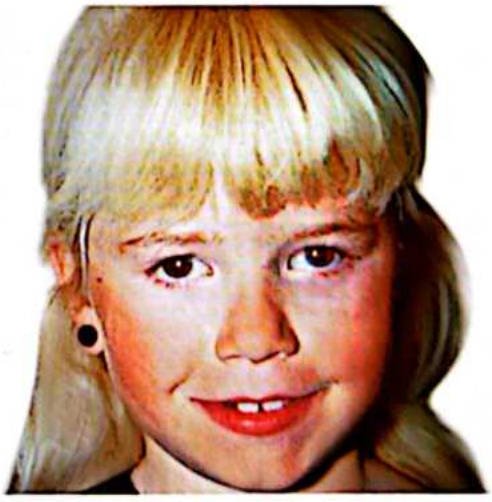
1. **Ocular albinism**: XLRI (X link recessive in nature)
2. **Oculocutaneous albinism (OCM)**: Affects skin and eyes.

Refer to as albino kids
Inheritance: Autosomal recessive

Types

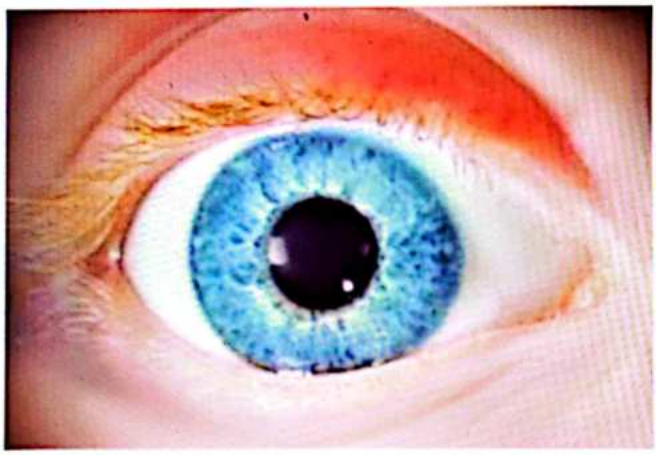
- Tyrosinase-negative type OCA(TNA)(OCA1)
- Tyrosinase-positive type OCA (TPA)(OCA2)

OCA 1.
No pigmentation on hair, skin or eyes.

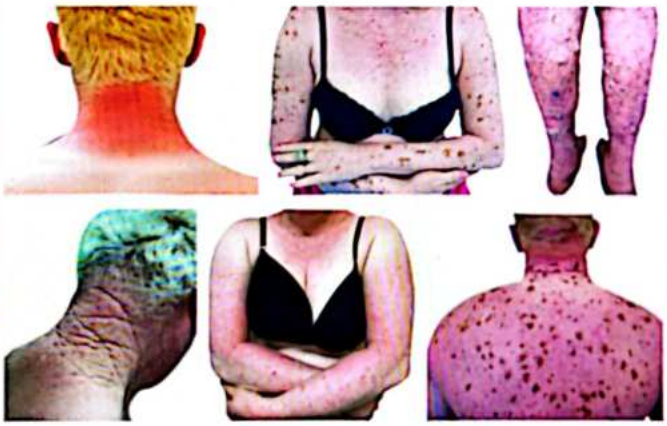


OCA 2
Some pigmentation on skin, eyes or skin

- At birth- OCA 1 & 2 will be similar. Gradually with age OCA 2 starts showing some pigmentation on hair, skin, and eyes.
- These kids always have eye Involvement



- Blue Iris
- Photophobia
- Nystagmus
- Impaired visual acuity
- Characteristic facial expression due to habitual squinting



- These kids are also prone to solar damage because there is no eumelanin so there may be skin cancers, sun burns etc.

Treatment

- Sun protection
- Genetic counselling
- Screening for malignancy annually

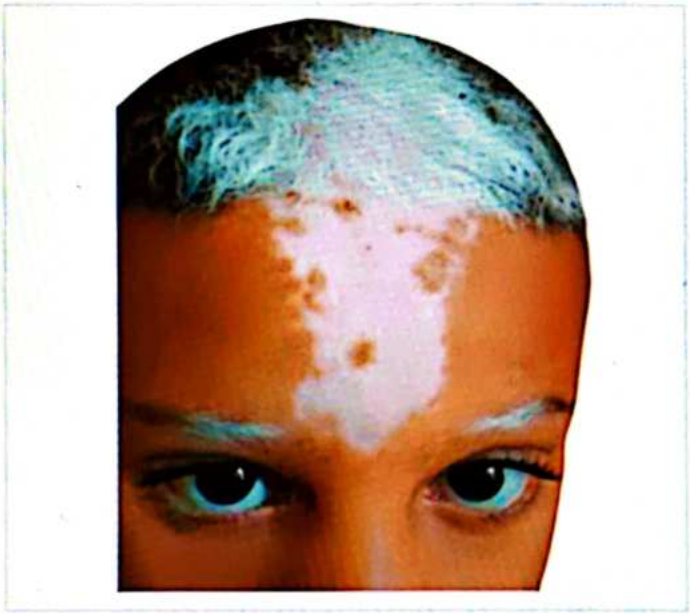
10. Hermansky Pudlak Syndrome

00:47:53

- Oculocutaneous albinism
- A bleeding diathesis
- In some individuals, pulmonary fibrosis, granulomatous colitis, or immunodeficiency.
- Demonstration of absent delta granules (dense bodies) on whole-mount electron microscopy of platelets.

11. Piebaldism

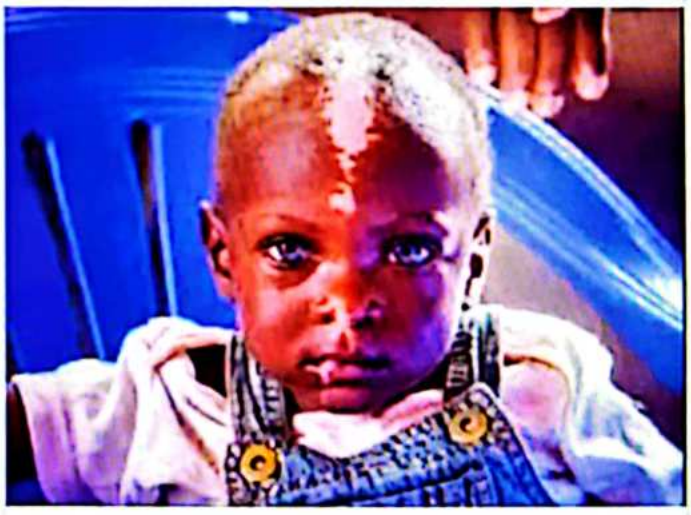
00:48:50



- It is autosomal dominant
- Defect: Mutations in the KIT gene on chromosome 14
- Clinical features
  - Triangular depigmented patch: Frontal or paramedian
  - White forelock of hair
  - Islets of hyperpigmentation

12. Waardenburg Syndrome

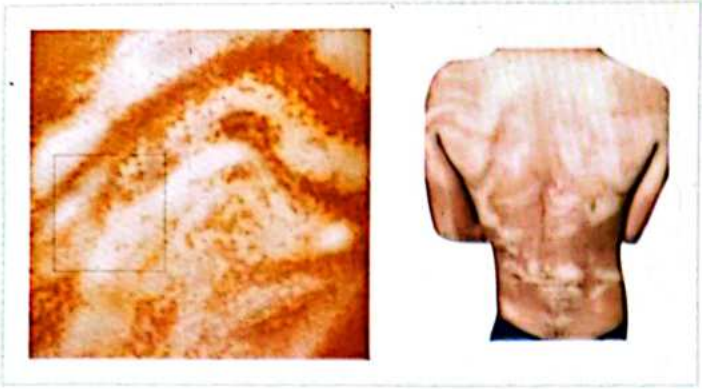
00:50:30



- Inherited syndrome
- Skin lesions resembling piebaldism
- Lateral displacement of the inner canthi (dystopia canthorum) with normal interpupillary distance
- Irides show partial or total heterochromia
- Cochlear deafness

13. Hypomelanosis of ITO

00:51:15



- Genetic mosaicism
- Onset: years after birth
- Clinical Features: Whorls of hypopigmentation along with the lines of blaschko.

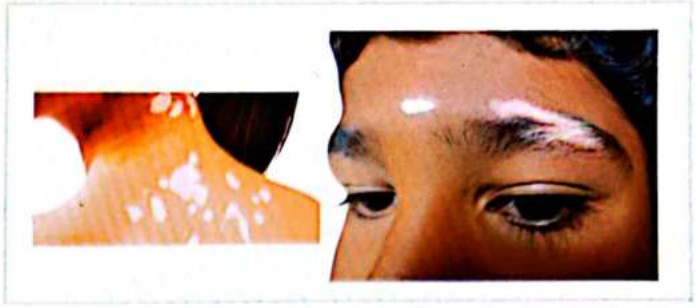
14. Vitiligo

00:52:10

- Autoimmune progressive acquired
- Defect: Melanocytopenia
- Family history 20-30%
- It is associated with other autoimmune disorders that can be diabetes, thyroid, pernicious anemia, alopecia , and down's. Most common association is Hashimoto's thyroiditis.
- **Pathogenesis**
  - Genetic factors: catalase gene
  - Immune hypothesis
  - Neural hypothesis: certain neurotransmitters are released, and destroy the melanocytes along with the particular nerve. This is responsible for segmental vitiligo.
  - Autotoxic self destructive or free radical hypothesis

Presentation of Vitiligo Patient

00:55:55

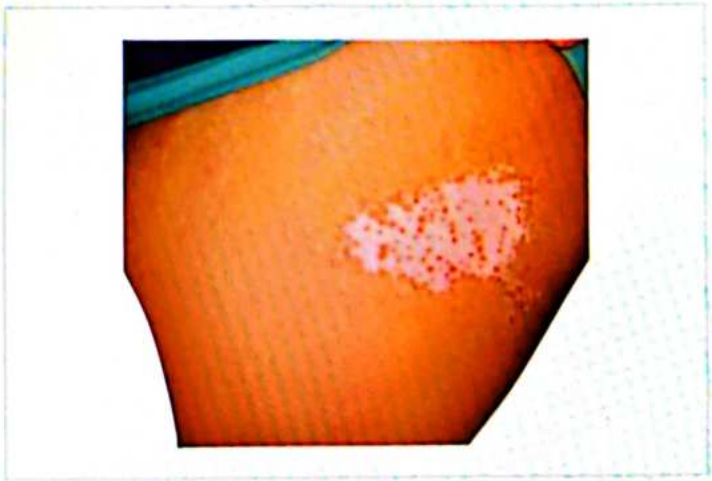


- Vitiligo patients present with totally depigmented and asymptomatic macules and patches.
- They are chalky white in colour.
- They are progressive and can be present anywhere on the body.
- Thy have scallopping or convex margins.
- Hairs are also whiten that is called leucotrichia (patchy white hairs)

Multiple colors are seen in vitiligo patches

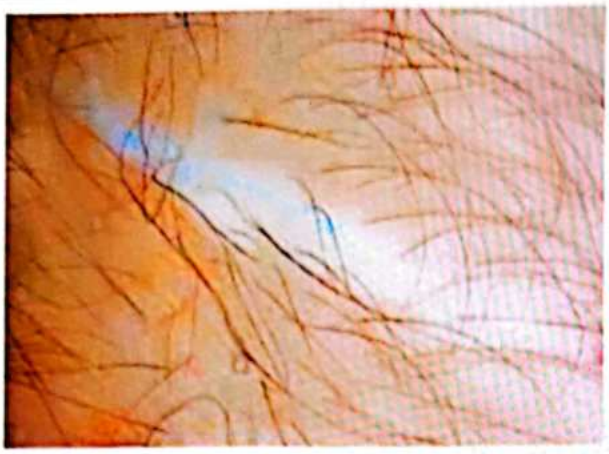
00:57:10

- Trichrome vitiligo
- Three zones are there
  - Center zone is of depigmentation
  - Surrounding is the zone of hypopigmentation
  - Again surrounding it is the zone of hyperpigmentation
  - It is of 3 colors



- Quadrichrome vitiligo
  - Perifollicular pigmentation is there
  - It is of 4 colors

koebner's phenomenon 00:58:18

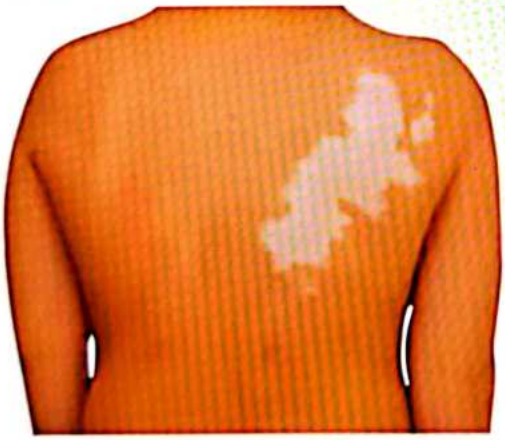


- Vitiligo shows koebner's phenomenon
- Very common in children
- Depigmented lines are there

Classification of vitiligo 00:58:50

1. Segmental
2. Non segmental: More common

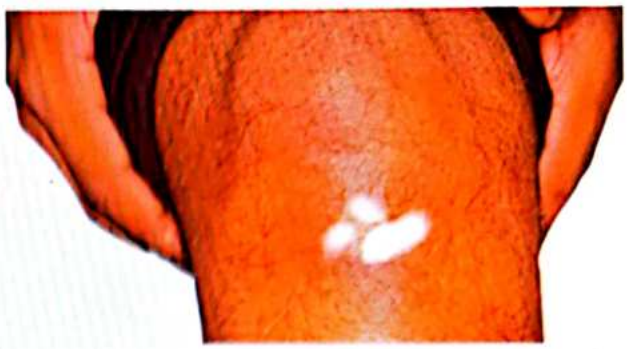
1. **Segmental Vitiligo**



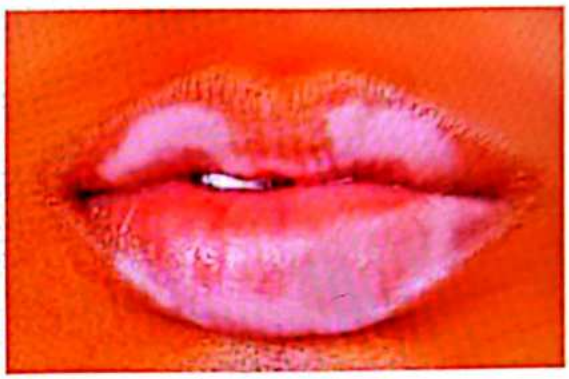
- Along a segment, or a dermatome
- Can develop in childhood, usually not at birth but after birth but in early childhood
- Depigmented macules are seen in particular dermato.
- Unilateral with midline demarcation
- Relatively stable course
- Frequent association of leucotrichia
- Absence of koebner's phenomenon,
- Neural hypothesis
- Not amenable to medical treatment hence surgical treatment

is required.

2. **Non segmental Vitiligo**



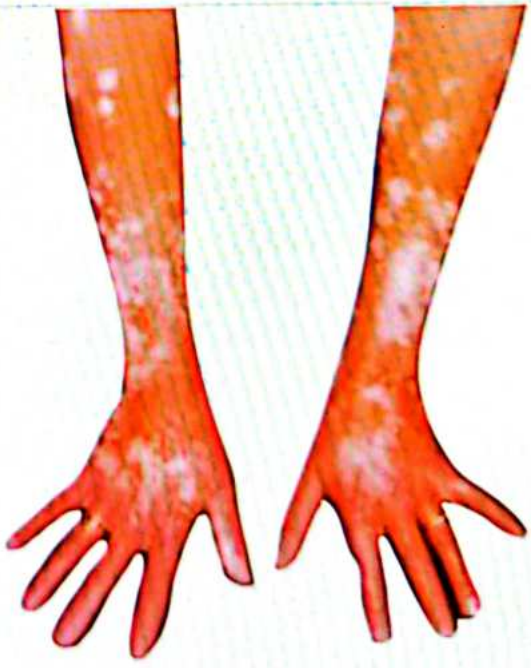
- Not along the segment, it can be focal (limited to one area)



- One or two lesions are there on mucosal area.

Generalized Vitiligo

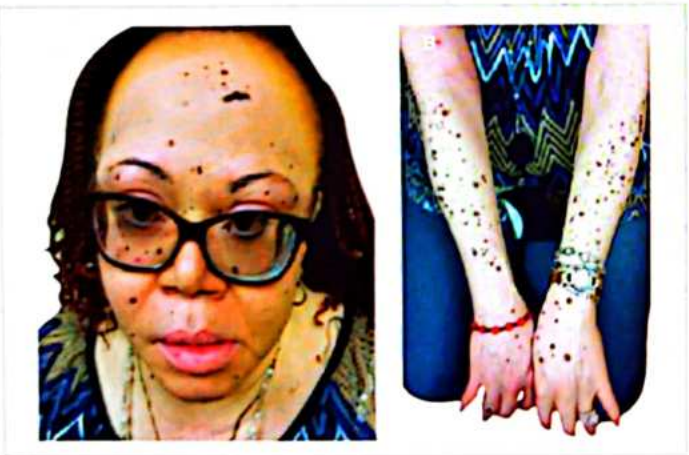
1. Vitiligo vulgaris (bilateral,symmetrical lesions)



2. Acrofacial (acral and face areas)

- Referred to as lip tip vitiligo.
- Very resistant to treatment because when the patient is treated there are no melanocytes in the vitiligo patch. Pigment is to be taken from surrounding skin or from the hair follicles which are present in that patch. But there are no hair follicles, and if there will be no hair follicles in the patch it will become too difficult to get the pigmentation.

3. It is called as vitiligo universalis

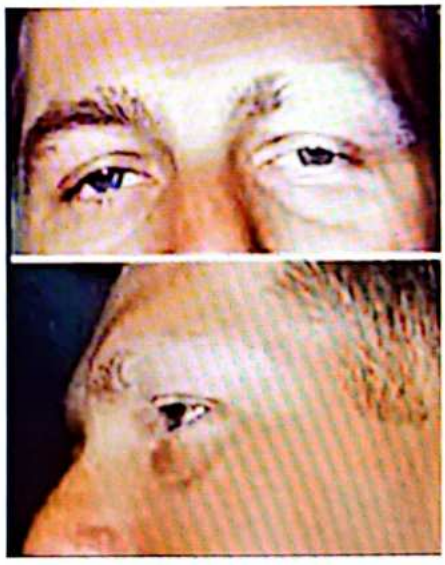


- When 90% of the body area is showing depigmentation.
- In this other 10% area of the body of the patient made to be depigmented such that the patient looks the same. It is done using monobenzyl ether of hydroquinone (MBEH)
- Prognostic factors are seen in acrofacial, vitiligo universalis, leucotrichia, presence of other autoimmune diseases, and progressive vitiligo.

Treatment

- General: avoid traumas
- Medical
  - Topical: Topical corticosteroids, Topical calcineurin inhibitors (tacrolimus, pimecrolimus). Mainly used for face and genetalia.
  - Systemic: Oral minipulse steroids (OMP), Betamethasone, methylprednisolone (twice a week), levamisole, azathioprine JAK inhibitor drug -tofacitinib.
  - Phototherapy: Topical (PUVA sol.) Oral psoralens 8 methoxy psoralens (8 MOP), 5 MOP, tri MOP NBUVB (narrow band UVB THERAPY) given 3times/week at wavelength of 311nm of UVB.
    Targeted phototherapy or Excimer laser..
- Surgical: When vitiligo is stable (not progressive) for more than one or two years. Donor areas are selected that have a good amount of pigmentation. For example: if the vitiligo is on the face the donor area will be behind the ears, if it is anywhere else on the body, the donor area will usually be thigh. A skin graft is taken from the donor area and put on the recipient area. There are various skin grafting techniques and cellular techniques where keratinocytes and melanocytes are separated and transferred as a suspension to the recipient side. Other surgical methods are : Skin grafting techniques and Cellular techniques.
- Cosmetic: Camouflage
- Bleaching: MBEH to treat vitiligo universalis.

15. **Vogt-Koyanagi-Harada Syndrome** 00:14:43

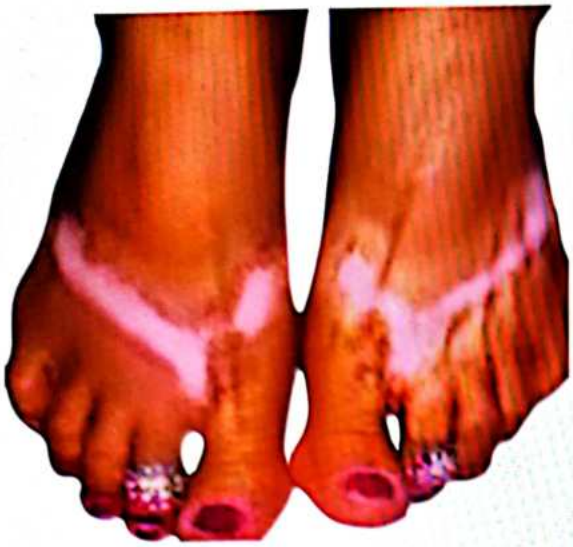


- Onset commonly in the third and fourth decades
- Encephalitis or meningitis symptoms Bilateral uveitis with choroiditis and optic neuritis.
- Dysacousia is usually bilateral.
- Poliosis (90%), leukoderma (63%) and alopecia

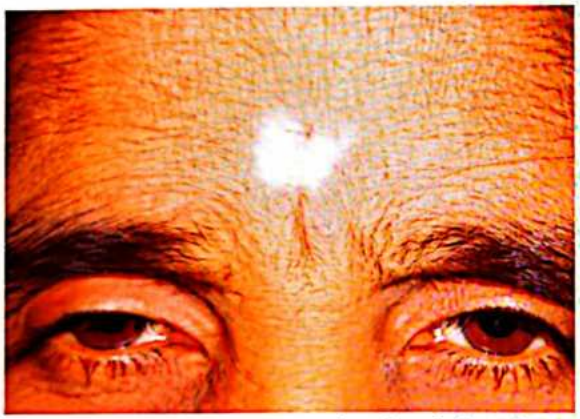
16. **Chemical Leukoderma** 01:15:12

- It is Because of chemical exposure, a person gets white skin.

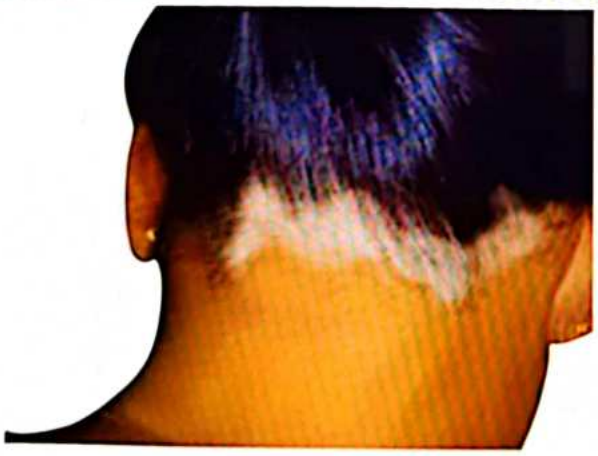
- It is also called as contact Leukoderma



Rubber chappals caused by MBEH



Bindi, caused by para tertiary butyl phenol

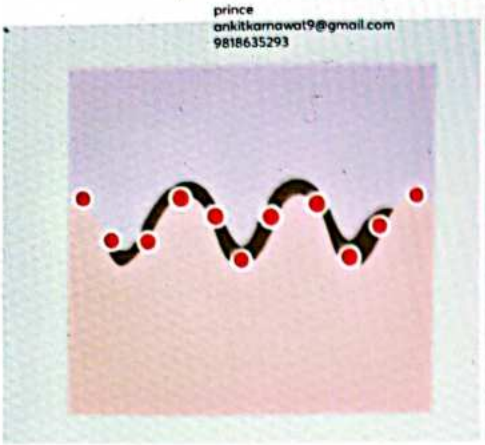
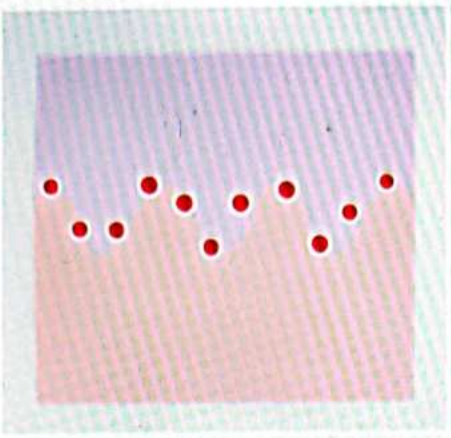
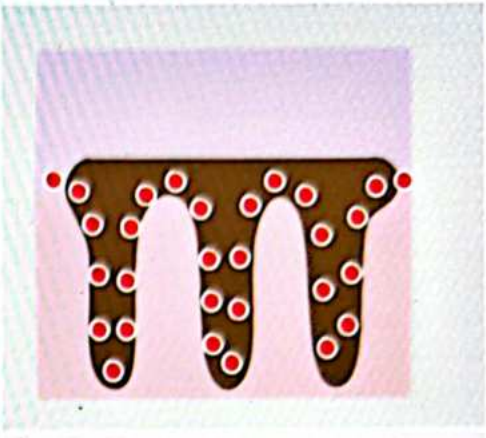


PPD
Hair dermatitis (hair dye)
Caused by para tertiary butyl catechol

Post Inflammatory Hypopigmentation

- MC of all acquired hypomelanotic disorders of the skin
- Causes
  - Mechanical and physical causes
  - Radiation injury, Thermal Injury
  - Infections: PV, Candidiasis, Leprosy, pinta, yaws, onchocerciasis Syphillis, Herpes simplex and zoster, Dermal leishmaniasis and vagabond's disease
  - Non infective inflammatory conditions : Pityriasis alba, pityriasis rosea, psoriasis, eczema, lichen striatus, lichen planus, discoid lupus erythematous, morphea, sarcoidosis and erythema multiforme.

Table 18.1

		
Freckle	Normal	Lentigo
Increased melanin production		Increased melanocytes

19 CONNECTIVE TISSUE DISEASES

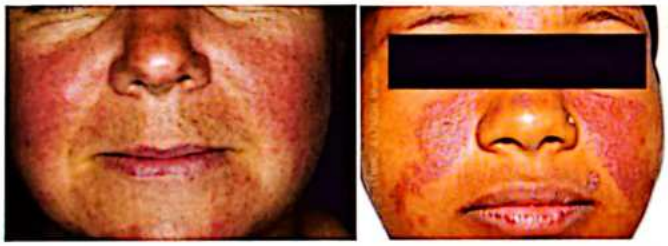
- Connective tissue is present everywhere in the body.
- Connective tissue disease are a group of disorders with skin manifestation, and internal organ manifestations.
- Associated with autoimmune inflammation.

Lupus Erythematosus

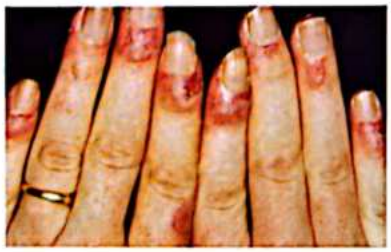
00:00:44

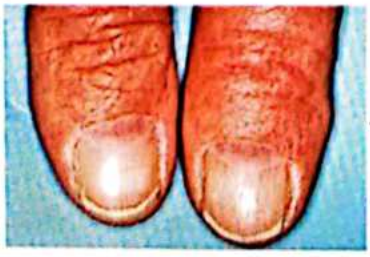
- Autoimmune connective disorder
- Associated with photosensitivity
- There is a role of genes and environment.
- Occurs more in females than male.
- Three Types of Erythematous
  - Acute Cutaneous Lupus Erythematosus - SLE
  - Subacute Cutaneous Lupus Erythematosus - SCLE
  - Chronic Cutaneous Lupus Erythematosus - DLE
- Skin lesions are Cutaneous Lupus Erythematosus.

SLE/ACLE

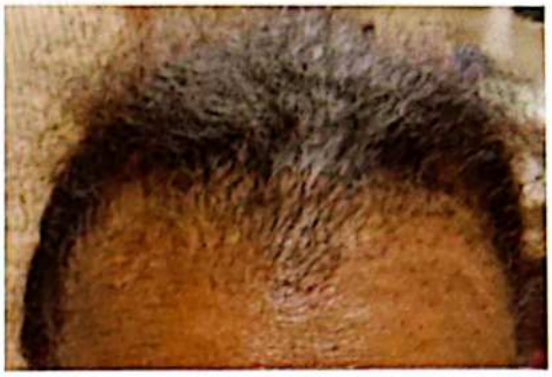


- In Acute Cutaneous Lupus Erythematosus: Malar Rash.
- In SLE - ARA criteria and SLICC criteria in medicine, one of them is associated with Malar Rash.
- Erythematous rash present in butterfly distribution,
  - It is the convex area of the space, such as the nose, cheeks.
- It spares nasolabial folds.
- In nails:





  - Red lunula with hyperkeratotic tissue and some ulceration and telangiectasia.
- In the head, SLE presents as hair fall.



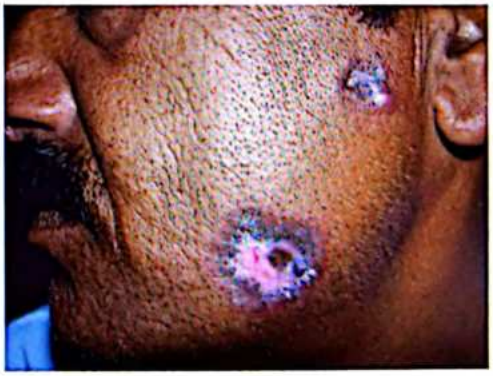
- SLE leads to non-scarring alopecia.
- Most common type of hair fall is Telogen Effluvium
- Most characteristic feature is lupus hair. These are short, rough hair in the frontal area.

SCLE

- It is present in the upper part of the body
- Present as Papulosquamous or annular
- One can see Positive anti Ro/anti SS-A

DLE

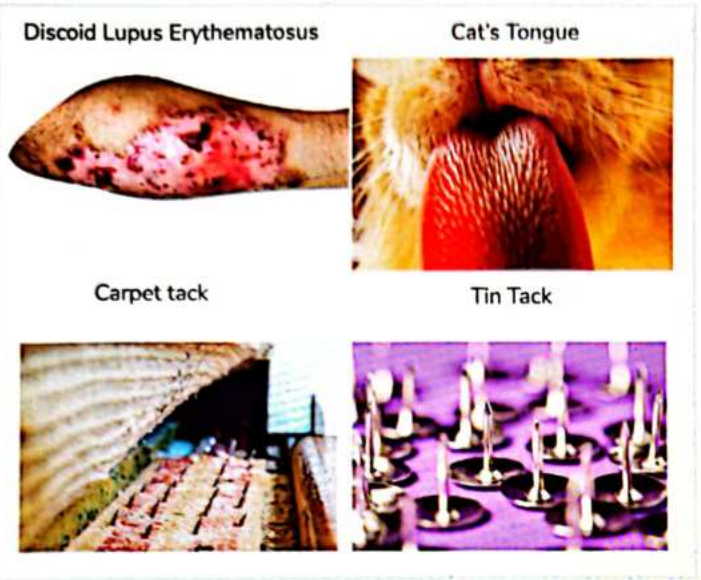
- Earlier known as DLE-Discoid Lupus Erythematosus because of discoid lesions.
- Now known as chronic cutaneous LE



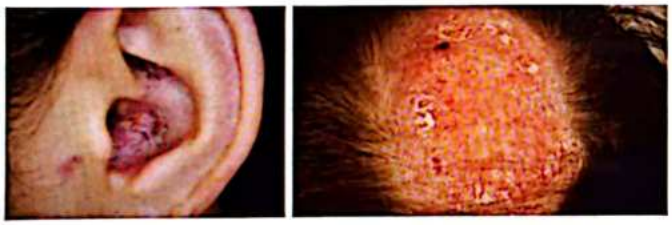
- **Site**: present in head or neck or disseminated in other areas also.
- **Sex**: most seen in Females than male
- **Age**: around middle age - 40 years.
- Chronic Cutaneous LE are characterised by three zones:
  - Central Atrophy area

- Middle activity-perifollicular plugging, scaling, telangiectasias
- Zone of hyperpigmentation

- Sign of DLE:
  - Perifollicular plugging - scales start depositing in it.



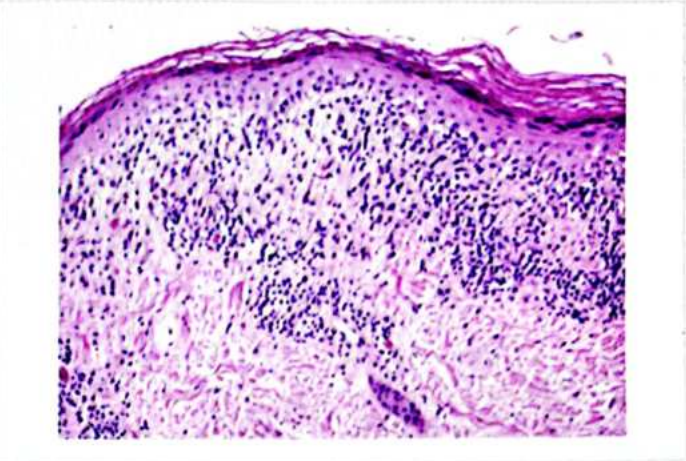
- Carpet Tack or Tin Tack Sign, also called Cat's tongue is characteristic of DLE
- Lesions in the ear (conchae region), it is called Schuster sign.



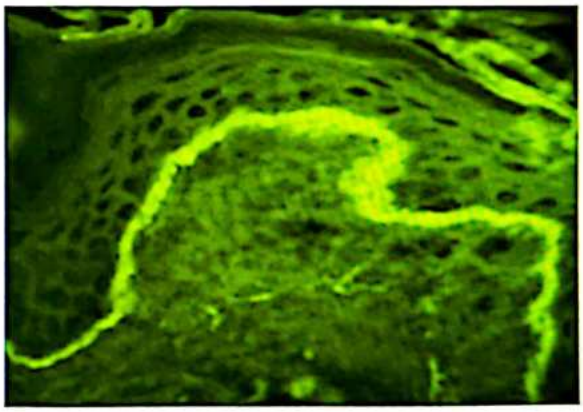
- DLE causes Cicatricial alopecia.

Drug Induced LE

- Drugs: Procainamide, hydralazine, quinidine, minocycline
- Temporal correlation
- Within four to six weeks, LE syndrome develops
- Drug induced LE vs SLE: Difference between drug Induced and normal LE:
  - Older Age Group
  - Renal and CNS Involvement Infrequent
  - Positive Anti histone Antibodies
  - Anti-DNA Antibodies Absent
  - Serum complement is normal
- Investigation of LE:
  - Skin biopsy- Histopathology and DIF (direct immunofluorescence)
  - In Histopathology, one sees:
    - → Interface Dermatitis –



      - Junction between the dermis and epidermis.
    - → BMZ thickening and degeneration
      - Basal cell will get degenerated.
      - Base membrane will get thickened
    - → Lymphocytic infiltration
      - Lots of lymphocytes will get infiltrated.
    - → Dermal mucin
      - Deposition of dermal mucin
  - In DIF

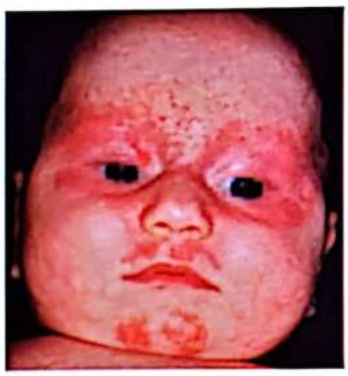


      - → Homogeneous linear granular IgG and C3.
  - Autoantibodies:
    - → ANA - Screening
    - → Anti-Smith (Anti-SM) antibody - more specific
    - → Anti-DS DNA - specific
  - Treatment:
  - Sun protection
  - Topical Corticosteroids or calcineurin inhibitors.
  - Drug of choice - Antimalarials (HCQs)
  - However, one can use other agents like Azathioprine, Methotrexate or Mycophenolic acid.

Neonatal LE

- Caused by Transplacental passage of maternal antibodies
- The patient or the child has two things.
  - Cutaneous lesions and Congenital heart block.

- Erythematous, slightly scaly eruption on the face and periorbital skin (racoon sign/owl eye/eye mask)
- These patients have positive Ro/SSA antibodies.



Important Information

Tip to remember -

- AML M1 - little brother - without maturation
- AML M2 - big brother - with maturation

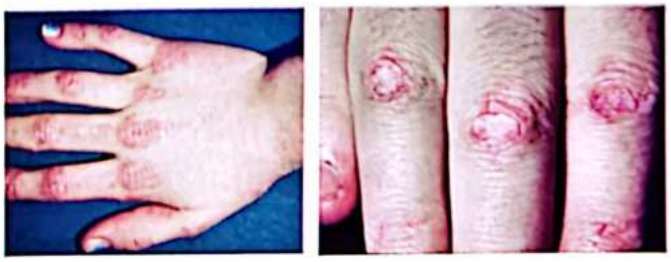
Dermatomyositis

00:14:13

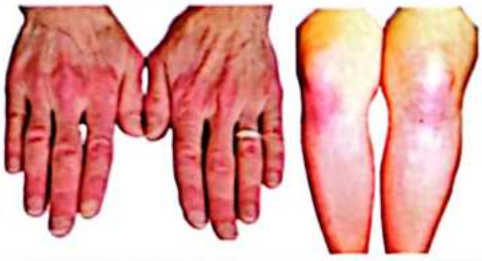
- Derma - skin, Myositis – Muscle inflammation
- These are seen in children and adults.
- Child form is mild
- Adult form can be associated with malignancy
- Age: bimodal
- Sex: Females more than males
- Muscle symptoms: proximal muscles, symmetrical involvement
  - It means thighs, buts, or shoulders will have problems.

Pathognomonic Lesions

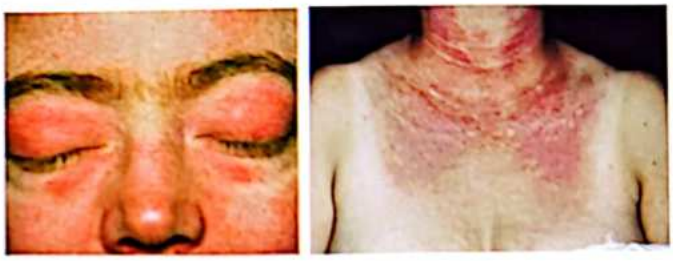
Gottron's papule



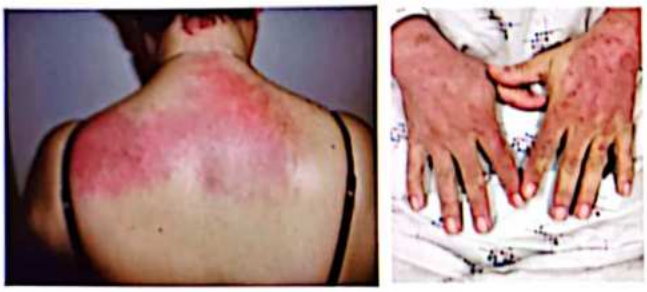
Gottron's Sign



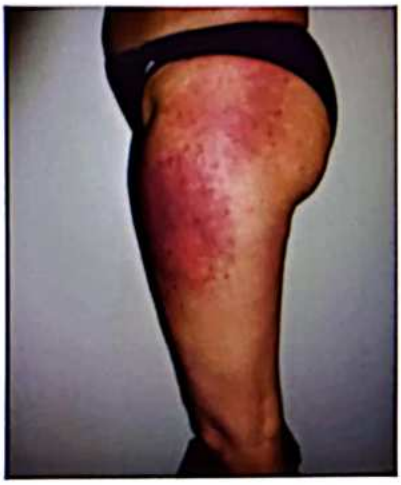
- There are two pathognomonic Lesions which are Gottron's Papules and Gottron's Sign.
- Gottron's Papules is the presence of Erythematous scaly plaques
- Associated with mild atrophy of interphalangeal and metacarpophalangeal joints.
- Violaceous confluent macular erythema (CMVE) on interphalangeal and metacarpophalangeal joints is known as Gottron's Sign.
  - And sometimes it happens on the knee.
- Gottron's Papules and Gottron's Sign are both present with confluent, macular, violaceous erythema or CMVE.
- CMVE is seen over other sites too, and it is named accordingly



  - Around eyelids Heliotrope rash (commonly asked)
  - At anterior aspect of the neck, it is called V-sign.

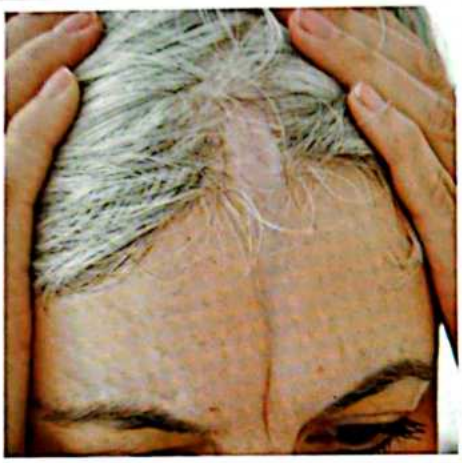


  - On the upper back it is called Shawl sign
  - It may also be present on the dorsa of hands - no name here
  - Lateral aspect of thigh - Holster sign



Criteria for DM

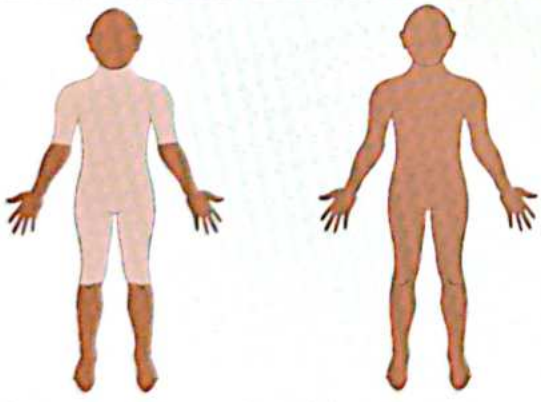
- Usually seen on trunk
- It can have single or multiple plaques
- Atrophic bound down plaque - start with an erythematous plaque
- Atrophy in the centre is seen and called ivory white scar or atrophy at the centre
- On the periphery, there is a violaceous border or erythematous border suggestive of activity called lilac border.
- This localised morphoea can affect the front of the parietal part of the scalp,



- There is an atrophic plaque sitting on it and extending downward, called En coup de sabre.

Systemic sclerosis or scleroderma

- It will be associated with skin manifestation where there will be sclerosis of skin and internal organ involvement.
- Vasculopathy involved here is aligned with inflammation.
  - These eventually lead to fibrosis
- The molecule involved here is TGF-beta.
- Present in two forms of Systemic sclerosis



Limited cutaneous systemic Diffuse cutaneous systemic

  - Limited cutaneous systemic sclerosis
    - → When it is involving the face and an area distal to the elbows and knee.
    - → Anticentromere antibody to be positive
  - Diffuse cutaneous systemic sclerosis
    - → If it involves the trunk, the proximal area above the elbow and knee.
    - → It will have more systemic manifestations and will be more complicated.
    - → Here it is anti Scl-70 positivity

Clinical features

- Raynaud's phenomenon

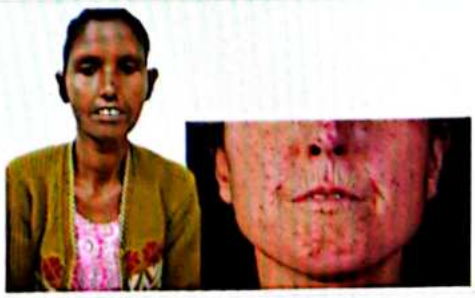
1. Fingers can become white due to the lack of blood flow
2. The fingers may turn blue as the blood vessels dilate to kept the blood in the tissue
3. Finally the fingers may turn red as the blood begins to return

  - Three phases
    - → Fingers become white
    - → Then they become blue because of the deoxygenated blood comes and flows in the periphery
    - → Then it becomes red when the normal flow of blood begins to return
- When there are repeated episodes of Raynaud's phenomenon, we see digital ulcers which heal with digital pitted scars.

- Dysphagia
- GI reflux
- Sclerosis of skin
- Internal organ involvement

Appearance

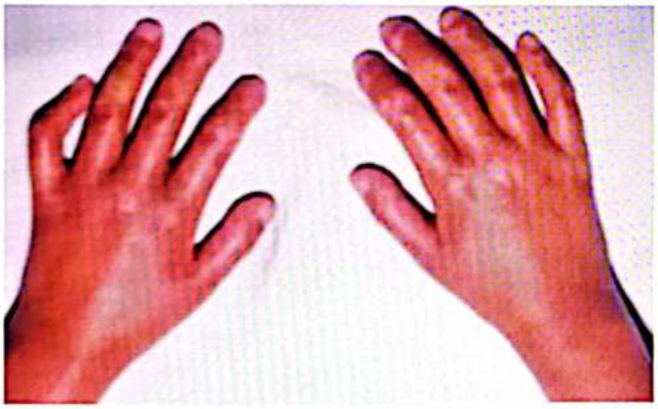
- Mask-like appearance

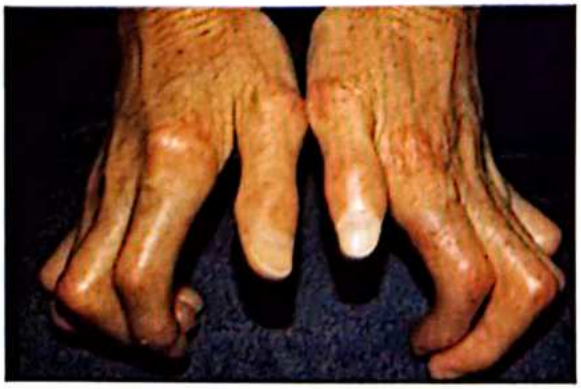


prince
ankitkarnawal9@gmail.com
9818635293

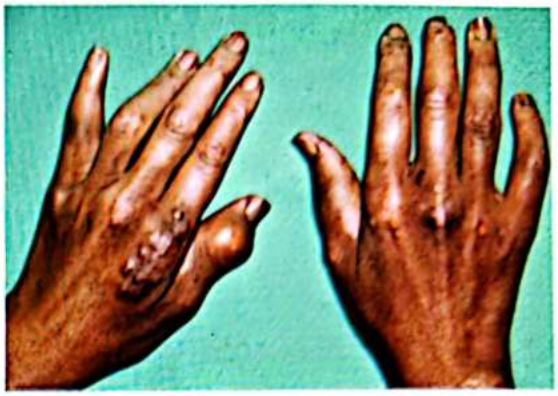
- Beaking of nose
- Stretched skin
- Central incisors are becoming more prominent
- Lips are thinned out and stretched
- Broad forehead
- Prominent jaw
- Bull dog mandible
- Telangiectasias
- Perioral furrowing
- Pigment loss, known as salt and pepper pigmentation
  - Because of perifollicular areas which are spared, there are areas of normal pigmentation called salt and pepper pigmentation.

- Sclerodactyly

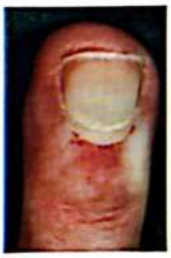


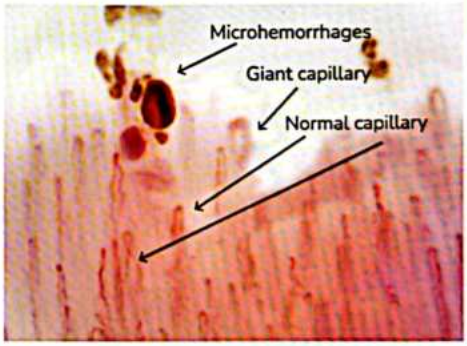


  - A feature of scleroderma where the skin is so bound down those contractures develop, and your finger becomes flexed permanently.
- Calcinosis cutis



  - White chalky material comes out of the skin, there can be the periungual nail changes where there are giant capillaries and haemorrhages.





American college of Rheumatology criteria for diagnosis of systemic sclerosis

- Major criterion
  - Presence of sclerodermatous skin proximal to the metacarpophalangeal joint
- Minor criteria
  - Sclerodactyly
  - Digital pitting scars or loss of tissue over the volar pads of fingertips
  - Bibasilar pulmonary fibrosis
- Diagnosis:
  - Presence of major criteria and two or more minor criteria is diagnostic of systemic sclerosis\

CREST syndrome

- It is a type of limited scleroderma where you see CREST features
- Calcinosis- Calcium deposits in the skin
- Raynaud's phenomenon
- Esophageal dysfunction
- Sclerodactyly
- Telangiectasias
- There will be a lot of systemic manifestations
- In the GI you will see dysphagia, reflux, and watermelon stomach because of vascular ectasia in the stomach.
- Pulmonary fibrosis
- Arrhythmias
- Scleroderma renal crisis
- Muscle involvement
- Investigation
  - Serology
    - → ANA
    - → Scl70 - diffused type
    - → Anticentromere antibody - limited type
- Investigations regarding inner organ involvement
- Treatment
  - Anti-inflammatory-penicillamine and cyclophosphamide, methotrexate
  - Vasculopathy - give calcium channel blockers
  - Antifibrotics

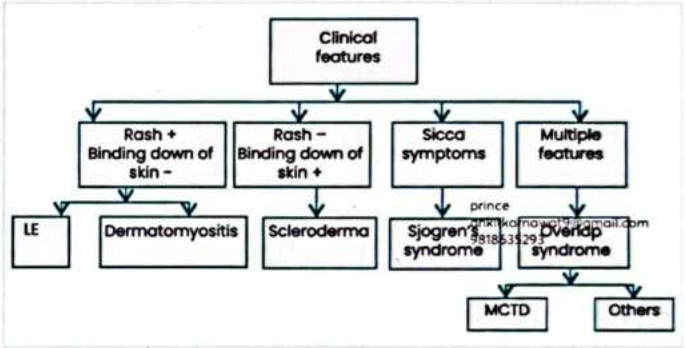
MCTD (Mixed Connective Tissue disorder) 00:33:17

- It has overlapping symptoms of LE, Scleroderma and DM (major connective tissue disorders) can happen.
- It is U1RNP positive

Sjogren's syndrome 00:33:53

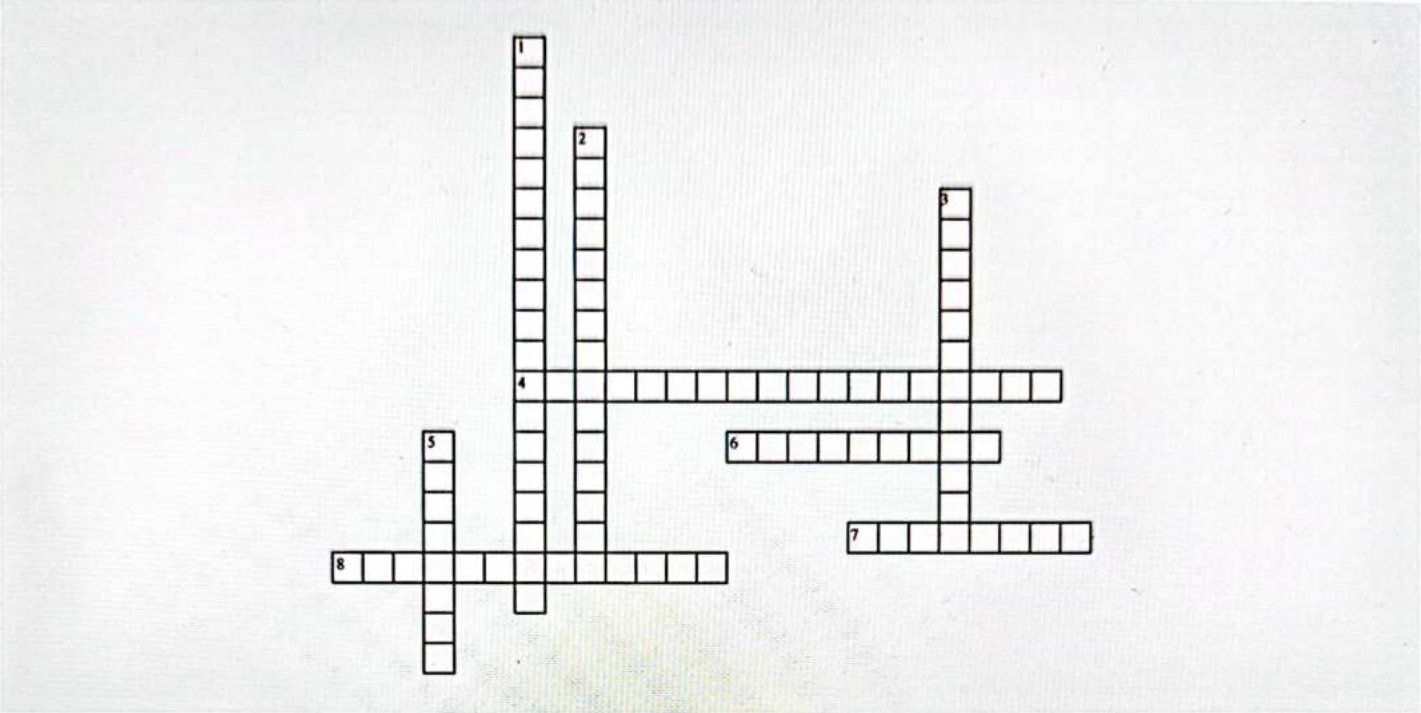
- It is a drying syndrome
- Here the glands are involved
  - Salivary gland
    - → Causing dry mouth
  - Lacrimal gland
    - → Dry eyes
- Dry mouth and dry eyes are called SICCA syndrome.
- Xerostomia (or dry mouth) with keratoconjunctivitis sicca

Diagram of summary of chapter 00:34:23



CROSS WORD PUZZLES

Crossword Puzzle



Across

4. Fingers become white and then they become blue because of the deoxygenated blood comes and flows in the periphery
6. Found in patients with Acute cutaneous lupus Erythematosus
7. Lupus Erythematosus caused by Transplacental passage of maternal antibodies
8. Hyperkeratosis on the edges of the finger

Down

1. Junction between the dermis and epidermis
2. Shows muscle symptoms like proximal muscles, symmetrical involvement
3. Lesions in the ear (conchae region)
5. localised form of scleroderma is called

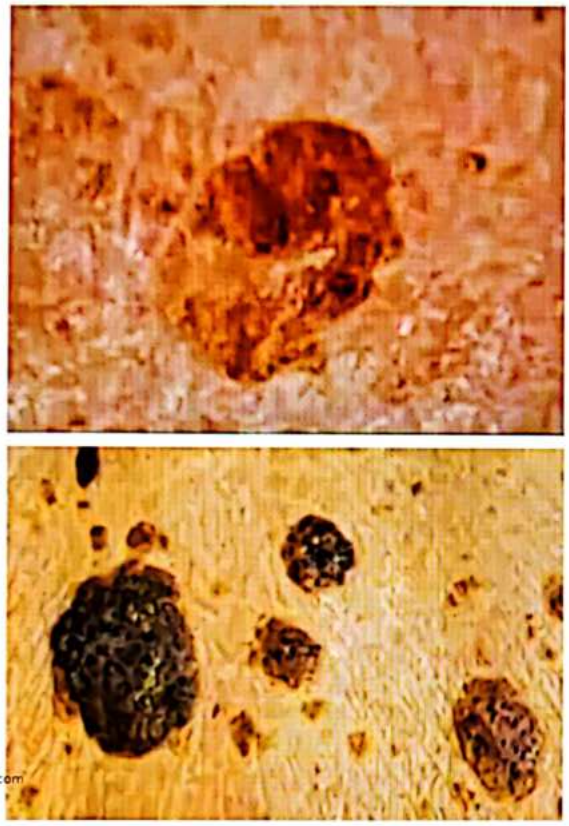
20 SKIN TUMORS

- Benign, premalignant, malignant
- Divided upon origin

Benign Keratinocytic Proliferation

00:00:54

Seborrheic Keratoses (SK)

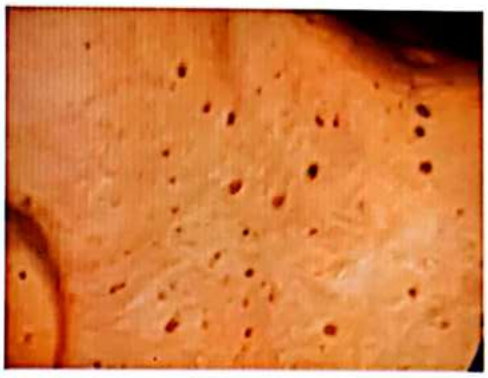


- Benign proliferation of keratocytes
- Older age groups
- Photo Exposed side (face, dorsa of hands, upper back)
- **Appearance**
  - Hyper keratotic plaques
    → Stuck on appearance
  - Follicular plugs
- **Sign of leser-Trelat**
- **Treatment**: for cosmetic purposes

Golden Points

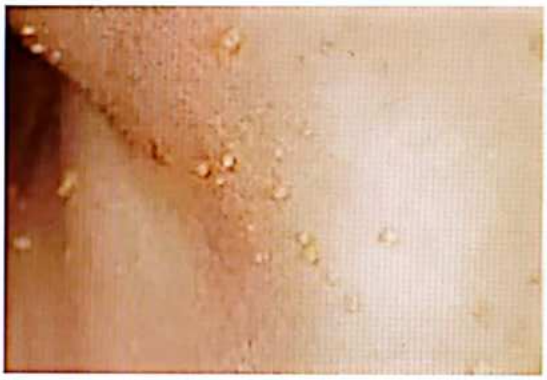
- Sign of leser-Trelat, sudden appearance of SK on body.
- Associated with underlying GI malignancy

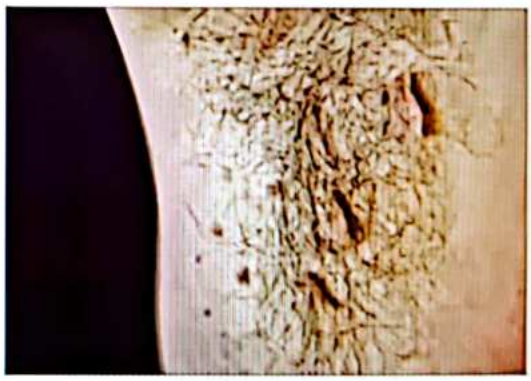
Dermatosis Papulosa Nigra (DPN)



- Variant of SK
- **Appearance**: Pigmented papules
- Common in
  - Dark skinned races
  - Face and neck

Skin Tags

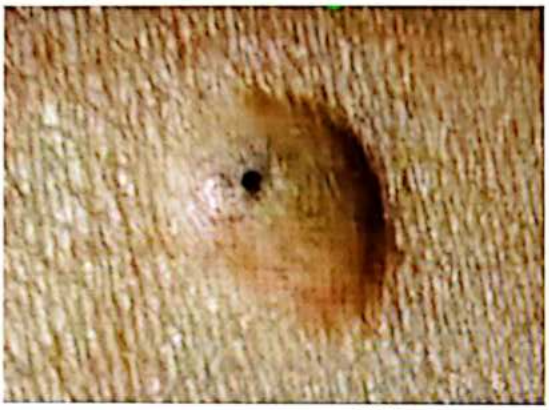




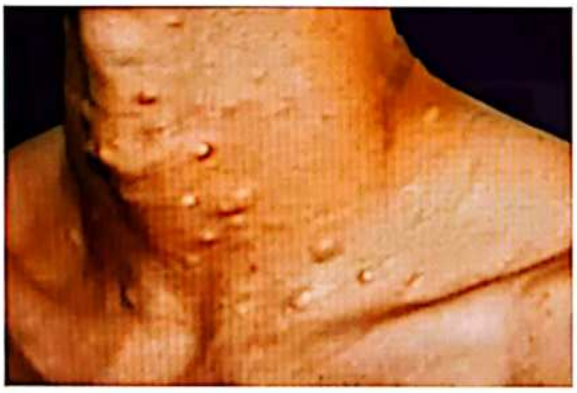
- Benign proliferation of loose fibrous tissue
- **Site**: neck, axilla, flexures
- **Appearance**: skin-colored pedunculated growths

- **Associations:**
  - Obesity
  - Diabetes mellitus
  - Insulin resistance
  - Acanthosis Nigricans

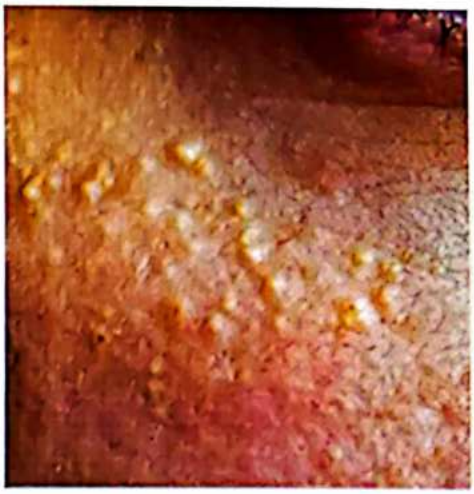
Cutaneous Cyst



- Epidermoid Cyst
  - Epidermal inclusion cyst
  - **Misnomer**: sebaceous cyst
  - Most seen in
    - → Back
    - → Nodules filled with foul smelling keratinous material and central plug or punctum
  - **Treatment**: excision



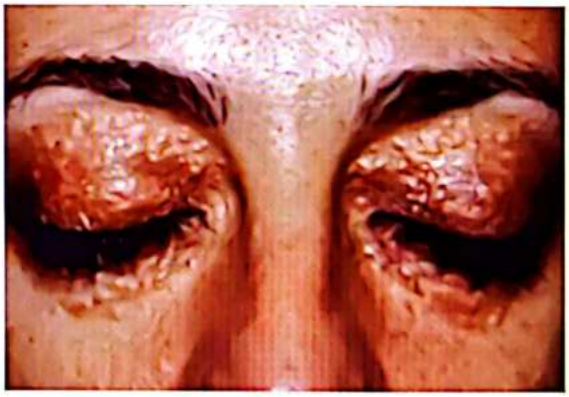
- Steatocystoma Multiplex
  - Multiple fatty cysts
  - True sebaceous cyst
  - Autosomal dominant condition
  - **Clinical features:** Multiple asymptomatic yellowish dermal nodules
  - Commonly seen
    - → Neck
    - → Scrotum
  - **Treatment**: removal of cyst



- Milium/ Milia
  - Benign keratinous cyst
  - Face-areas of vellus hair
  - Secondary to various dermatoses
  - **Appearance:**
    - → Whitish
    - → Pinpoint papules
    - → No central umbilication
    - → Single/ multiple lesions

Syringoma

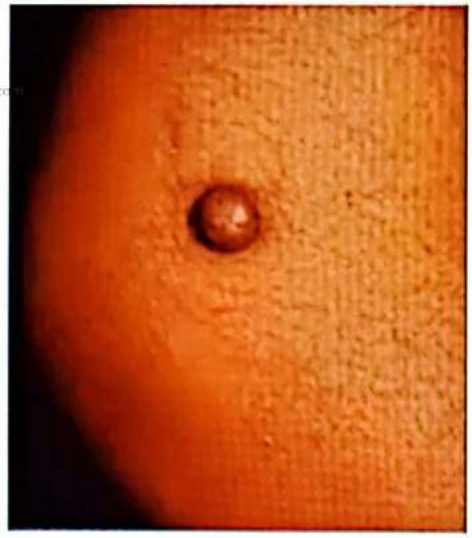
00:12:04



- **Origin**: acrosyringium of eccrine sweat glands
- **Site**: over eyelids
- **Clinical Features:**
  - Multiple Translucent papules
  - Asymptomatic
  - Eccrine (watery) secretions
  - Grouped
  - Angular borders
- **Histopathology:** Tadpole/comma shaped cells

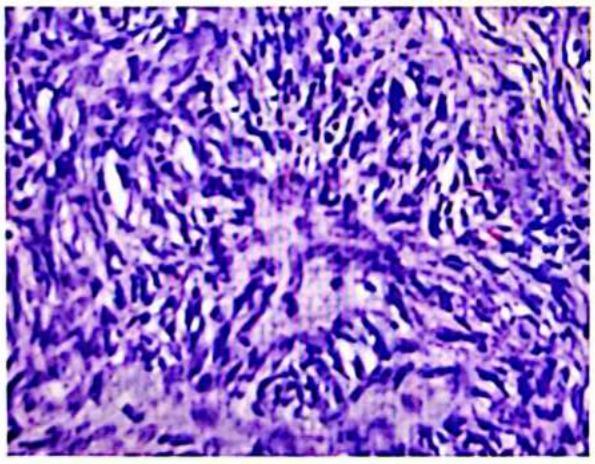
Dermatofibroma

00:13:53



- Benign Fibrous Histiocytoma
- **Clinical Features:**
  - Solitary
  - Hyperpigmented
  - Round
  - Oval or targetoid papule, plaque, or nodule
- Commonly seen on lower legs
- Central Dimple sign when lateral pressure is applied

- **Histopathology**
  - Storiform, cartwheel or whorled pattern
  - Consists of spindle cells with elongated nuclei radiating from center nuclei

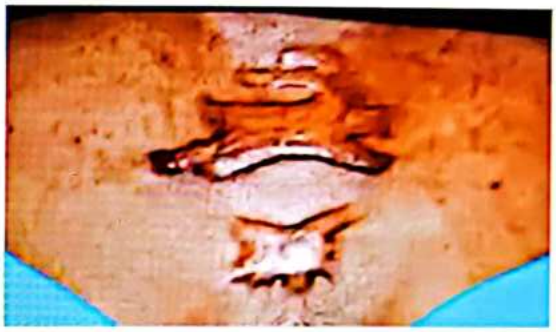


- Seen in
  - Dermatofibroma
  - Dermatofibrosarcoma protuberans

Keloid

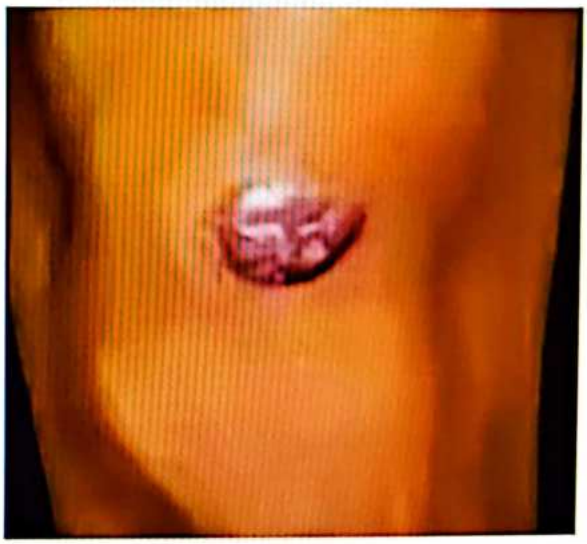
00:15:38

- **Also known as:** crab's claw
- Overgrowth of fibrous tissue after healing of skin injury extends beyond the original defect.
- **Association:** HLA
- Autosomal dominant inheritance
- **Sites**
  - Shoulders
  - Chest
  - Ear lobes (dumb bell configuration)
  - Upper arm
  - Cheeks



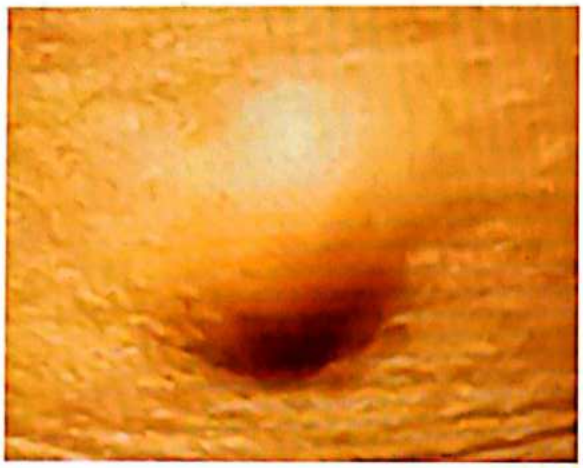
Golden Points

- Hypertrophic scar differs from Keloid
- Keloid
  - Extend beyond the original defect
  - Does not resolve spontaneously
  - Not corrected by surgery
  - Intralesional treatment
    - → Corticosteroids
    - → Fluorouracil
    - → Cryotherapy
- Hypertrophic scar



- Scar does not extend
- Can resolve spontaneously in 1-2 years on its own
- Corrected by surgery

Lipoma



- Benign tumor of adipose tissue
- **Clinical Features:**
  - Soft
  - Compressed
  - Subcutaneous swelling
  - Single/ multiple

Skin Cancers

- Usually, skin cancers are of two types
  - Non melanoma skin cancers (NMSC)
    - → BCC (Basal Cell Skin Cancer)
    - → SCC (Squamous Cell Skin Cancer)
  - Melanoma skin cancer
- Non melanoma skin cancers (NMSC)
  - More indolent
  - Less aggressive
  - Less chances of metastasis
  - More localized
- Melanoma skin cancers
  - More aggressive
  - More chances of metastasis

Non melanoma skin cancers:

SCC and its Precursors (Premalignant Dermatosis) 00:20:50

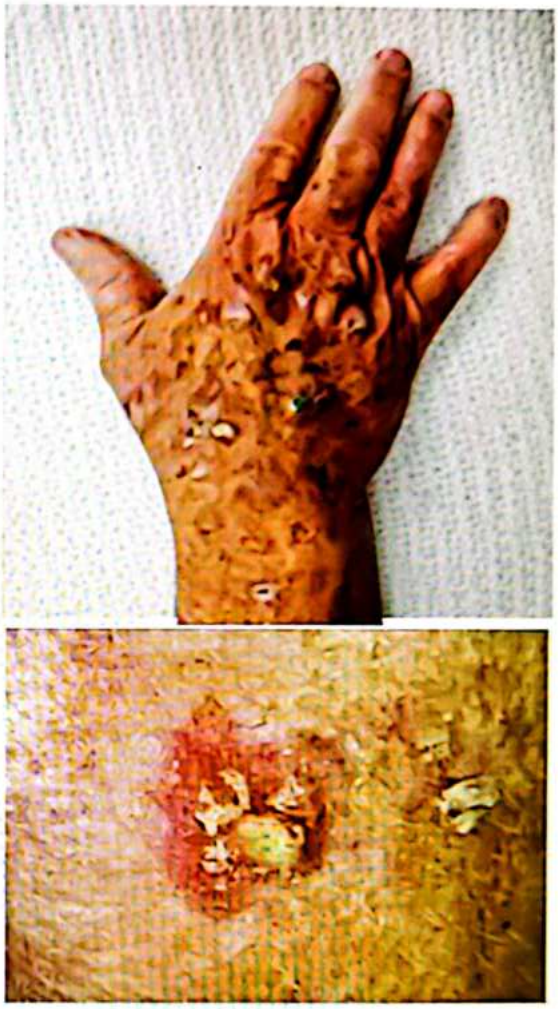
- Premalignant dermatosis has higher chances of SCC.

A. Actinic Keratoses

- Actinic - Sun
- Keratoses - Keratotic lesions
- Most common precursor.

- **Predisposing factors:**
  - Elderly
  - White races
  - UV radiation

Recall: Parakeratoses is seen in Actinic Keratosis

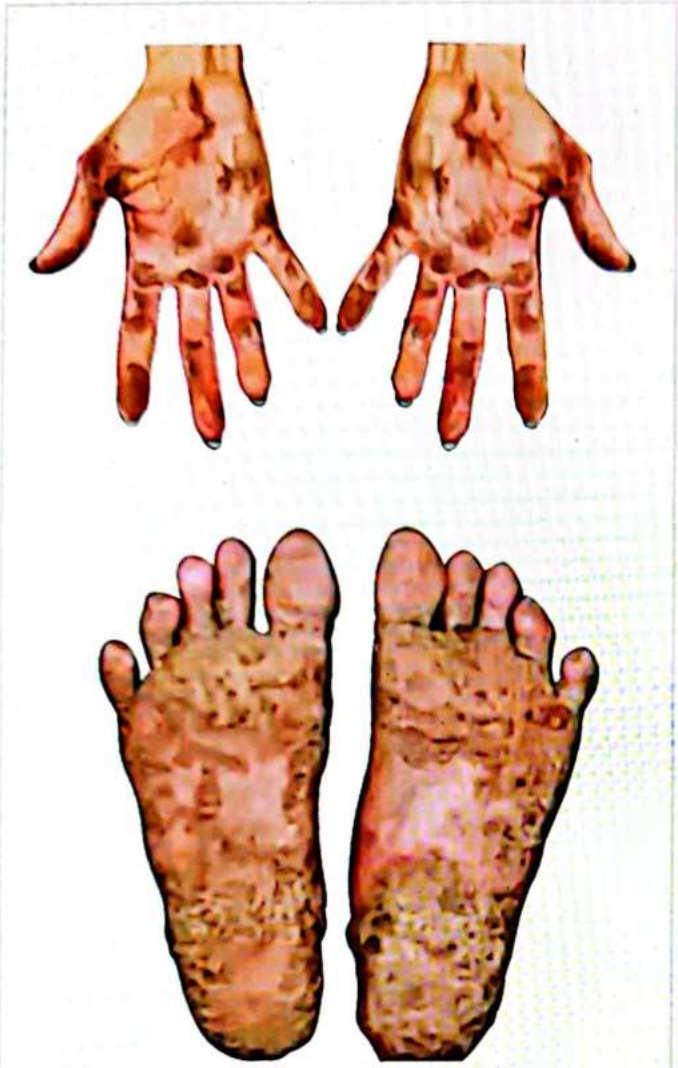


- **Pathology:** Parakeratosis (PK)+ Atypia
- **Site**
  - Photo exposed (dorsa of hands and face)
  - Lower lip (common)
  - Papules with rough scaly surface
  - Adherent yellowish crust
  - Pinpoint bleeding points

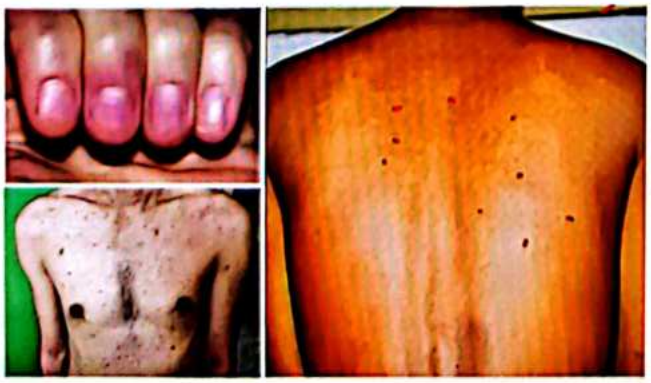
B. Arsenical Keratosis

- Premalignant precursor
- High exposure to Arsenic, through contaminated drinking water
- Seen at community level

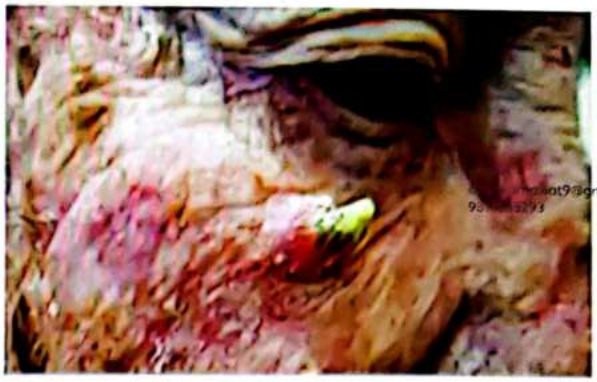
- **Site**
  - Palms and soles (rough Hyperkeratotic papules)



  - Nails (Mees lines- transverse white bands)
  - Trunk (Hypo or hyper Rain drop pigmented macules)



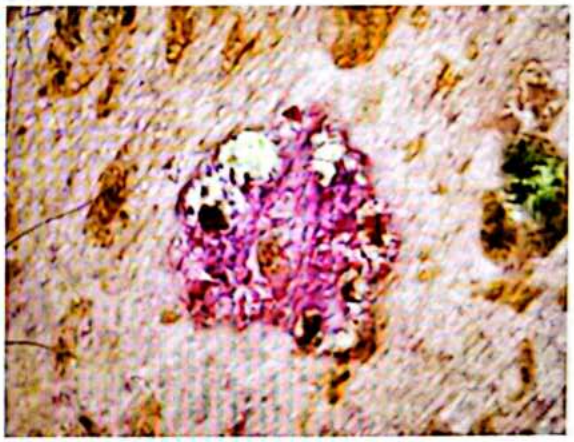
C. Cutaneous Horn

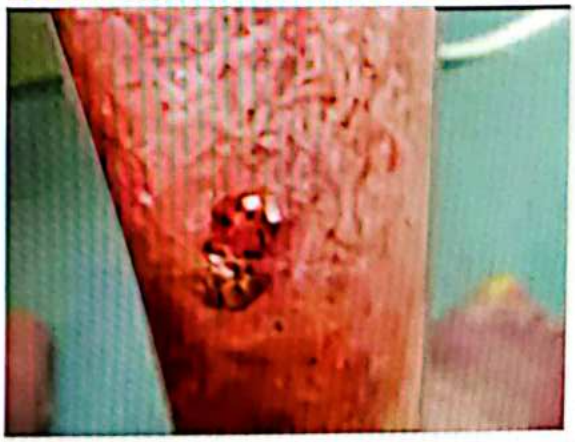


- Premalignant precursor
- Conical projection of hyperkeratotic skin
- Photodamaged area

D. Bowen's disease

- Intraepidermal (in situ) SCC
- Not a precursor
- Small potential for invasive malignancy
- **Population:**
  - White
  - Elderly
  - UV exposed





- **Site:** lower leg of elderly females
- **Clinical Features**
  - Hyperkeratotic
  - Rough plaque
  - Adherent crust
  - No bleeding points

Management of Bowen's disease and Premalignant Dermatoses

- Biopsy
- (PDT) Photo dynamic therapy
- 5-FU
- Imiquimod
- Cryotherapy

SCC (Squamous Cell Carcinoma)

- Malignant tumor arising from epidermal keratinocytes or its appendages
- Second most common skin tumor (First- BCC)

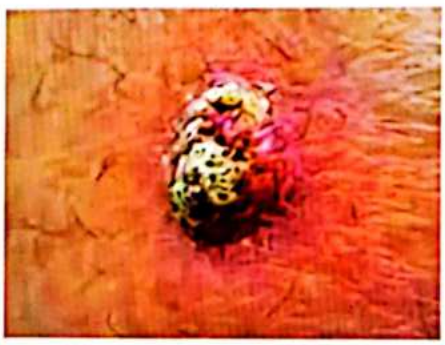
To Remember

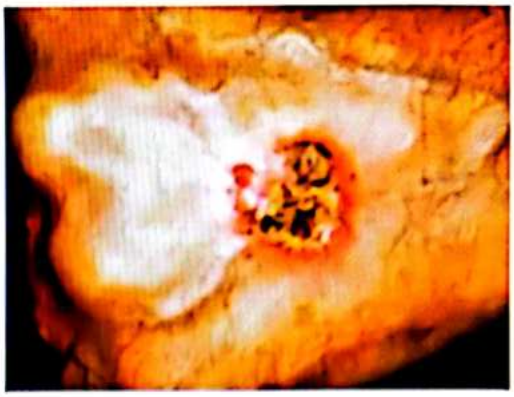
- SCC is the most common skin tumor in immunocompromised
- Normally SCC is 2nd most common

- **Association**
  - Immunocompromised-HIV
  - Renal transplant
- Low rate of local, regional, and distant spread
- **Predisposing factors**
  - UV exposure
  - Immunosuppressed individuals
  - Xeroderma pigmentosum (XP)
  - Albinism
  - LP, DLE

- **Site**
  - Exposed areas
  - Areas of photo damage

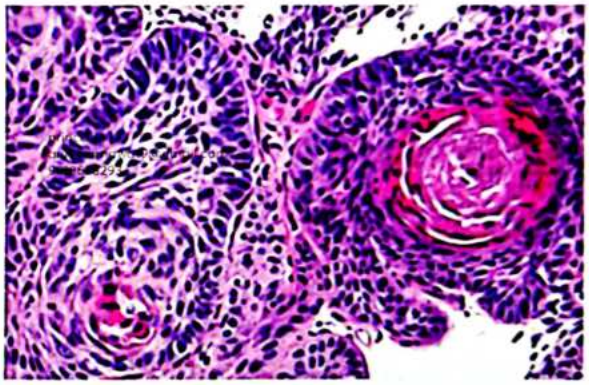
- **Clinical features:**





  - No classical feature
  - Papule or Nodule or Plaque
  - Presence of induration
  - Signs of photo damage
    - → Crusting
    - → Scaling

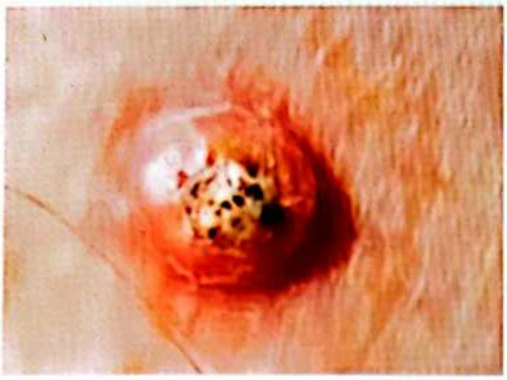
- **Histopathology:** Keratin pearls



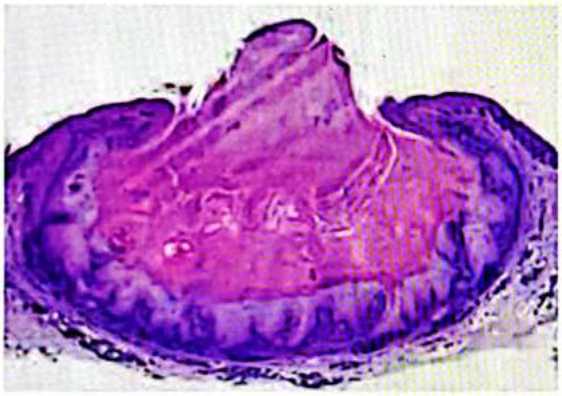
- **Treatment**
  - Wide surgical excision with margins
  - MMS (Mohs Micrographic Surgery) - Pathologist is involved while removing the tumor and checks if margins are involved are not.
  - **Advanced case**
    - → Radiation
    - → Chemotherapy

Keratoacanthoma

- Recently been classified as a well-differentiated SCC
- **Site:** Face>Hands
- **Phases**
  - Growing
  - Maturation
  - Resolution

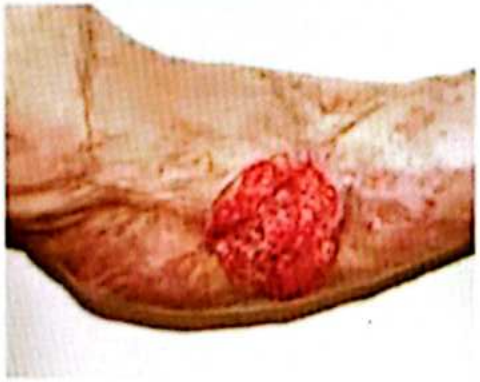


- **Clinical features**
  - Start as a papule
  - Develops to a Dome shaped nodule
  - Central keratin plug
  - Outer epidermal lining everts to surrounding skin
  - Plug falls off
  - Lesion disappears



- **Histopathology**
  - Epidermal lipping
  - Central keratotic plug

Recall: Molluscum has HP bodies

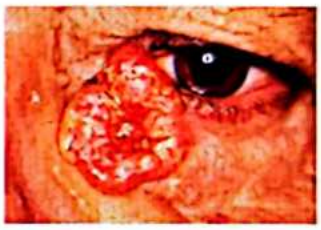


Marjolin's Ulcer

- Sites of burns, scars
- High tendency to develop into CA (SCC>BCC)
- High invasive potential

BCC (Basal Cell Carcinoma) 00:36:58

- **Also known as**: basalioma, rodent ulcer
- Most common human cancer
- Slow growing, locally invasive tumors rarely metastasize.
- It arises from pluripotent cells within the basal layer of the epidermis or follicular structures
- **Clinical Features**
  - No recognized premalignant stage
  - Small scaly plaque initially
  - Slow progressive course of peripheral extension
  - Nodulo ulcerative
  - Thready touch
  - Telangiectasia
  - Bleeds to touch



  - **Site**
    → Face and around eyes
    → Nose

Nodular NCC

Superficial BCC

Pigmented BCC

Morphoeic BCC

Basosquamous BCC

- **Types of BCC**
  - Nodulo ulcerative
    → Rodent ulcer, most common
  - Superficial BCC
  - Pigmented BCC
  - Morphoeic BCC
  - Basosquamous BCC
- **Association**

- Xeroderma pigmentosum
- Albinism
- Naevoid BCC syndrome/ Gorlin syndrome

- **Treatment**
  - **Medical:** low-risk superficial and small nodular BCC
    → Imiquimod, 5-FU
    → PDT
    → Intralesional interferon α-2b
  - **Hedgehog pathway inhibitors:** (Vismodegib)
    → Invasive BCC
    → Cannot do surgery

Golden Points

- 90% of human BCCs have loss of function of PTCH1
- The remaining 10% have gained a mutation in SMO
- This leads to unchecked activation of the Hedgehog pathway, resulting in unregulated proliferation of basal cells.
- Vismodegib (GDC-0449) is a small molecular Inhibitor of SMO
- Treats metastatic and locally advanced BCCs
- Orally administered at a dose of 150 mg daily.

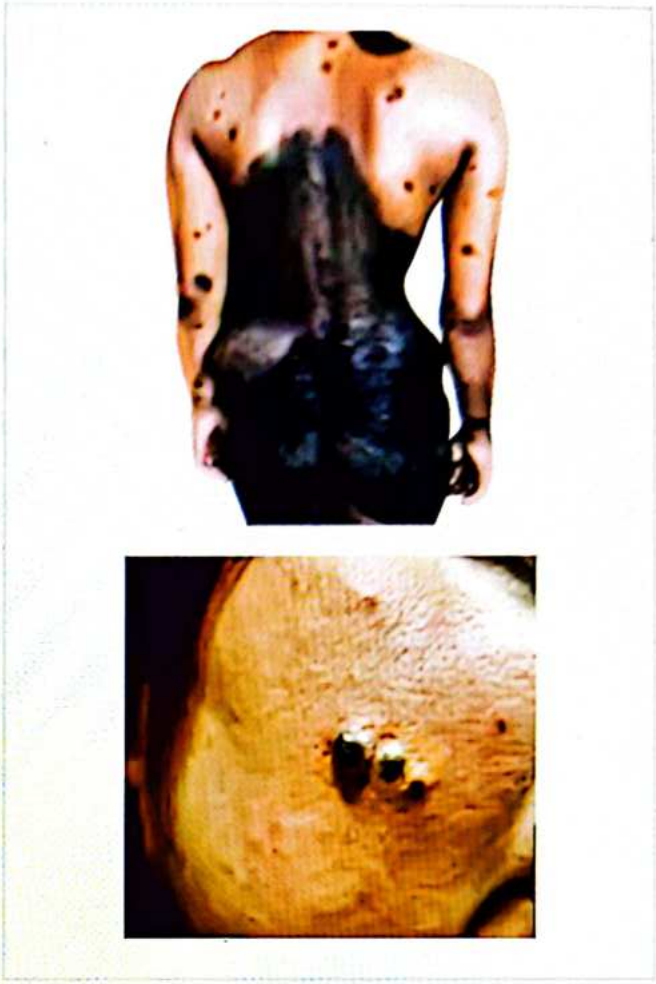
  - **Surgical**
    → Excision with predetermined margins
    → 4-5 mm surgical margin
  - **Mohs micrographic surgery**
    → Recurrent
    → Morphoeic or large BCC
    → 3-15 mm margin to obtain a similar clearance rate
  - Cryotherapy
  - **Superficial and electron beam radiotherapy or brachytherapy**
    → Primary or surgically recurrent BCC
    → High risk BCC in patients who are unwilling or unable to tolerate surgery

Melanomas:

Malignant Melanoma Skin Cancer

- A malignant tumor arising from melanocytes
- Metastasis is seen
- **Predisposing factors**
  - Precursor lesions
    → UV radiation (controversial)
    → Genetics (CDKN2A)
  - Western population

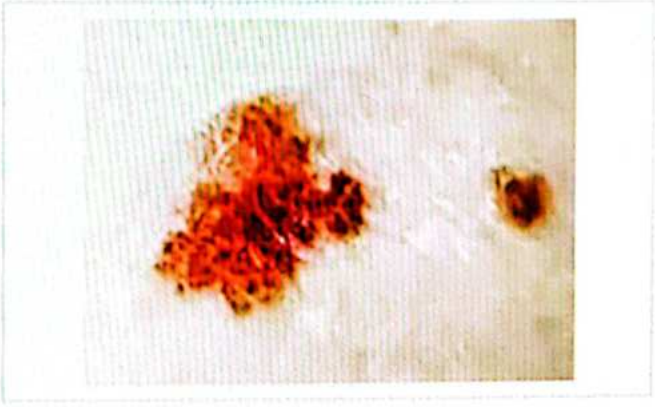
Precursor Lesions



- **CMN**
  - Large CMN (>20 cm)
  - More often located on the trunk and present with satellites

- **Common Nevi**
  - Risk of transformation very low
  - Highest risk is with junctional nevi

- **Dysplastic/Atypical Nevi**



- **Atypical Nevi**
  - → Usually large size (>5 mm)
  - → Irregularly distributed colors
  - → Tendency to emerge after young age
- **Dysplastic Nevi**
  - → Distinct patterns which are interpreted either as a pattern of naevus growth or as pre-malignant status

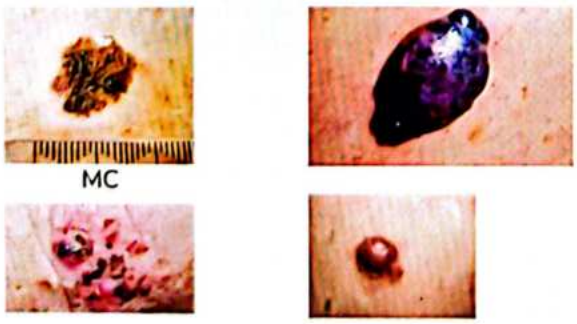
Identification of Malignant Melanoma

Refer Table 20.1

- **ABCDE rule**
  - **A:** Asymmetry
    - → Moles that have asymmetrical appearance
  - **B:** Border
    - → A mole that has blurry and/or jagged edges
  - **C:** Color
    - → A mole that has more than one color
  - **D:** Diameter
    - → Moles with a diameter larger than a pencil eraser (6mm or ¼ inch)
  - **E:** Evolution
    - → A mole that has gone through sudden changes in size, shape, and color

- Dermoscopy
- Histopathology - Gold standard

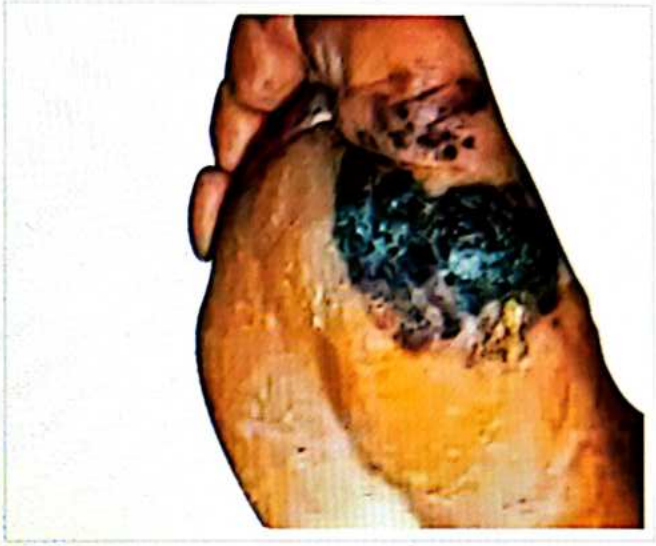
Types of Melanoma



- **4 types**
  - Superficial Melanoma (most common)
  - Nodular Melanoma
  - Acral lentiginous
  - Lentigo maligna

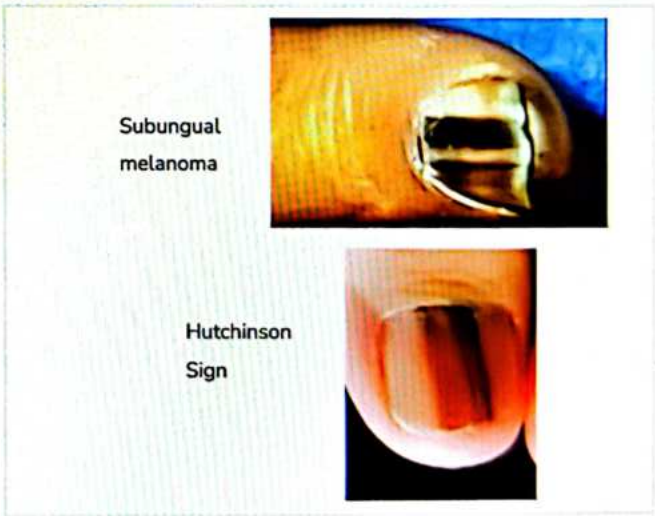
Golden Point: Worst prognosis is seen in Amelanotic melanoma

- **Acral lentiginous melanoma**



  - → Feet > Hands
  - → Most common type in south Asian people (India)
  - → Variably pigmented plaque

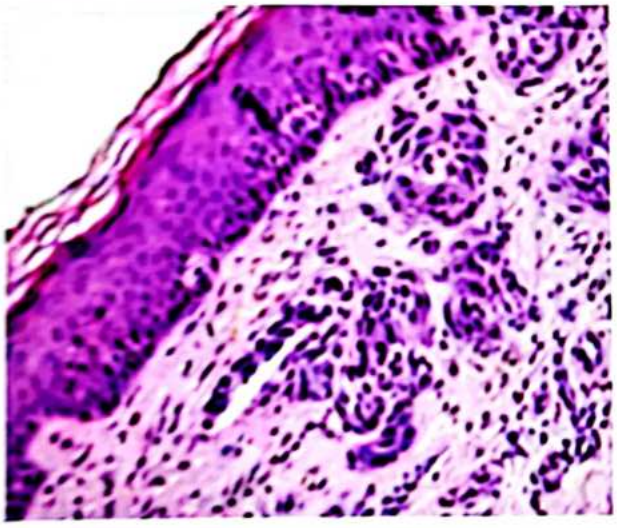
- **Subungual melanoma**



  - → Multiple, irregular bands of pigmentation on nails
  - → Hutchinson sign
    - Multiple irregular pigmentation of PNF (proximal nail fold)

Investigations of Melanoma

- **Histopathology**
  - Ideal biopsy is a narrow (2 mm) margin excision of the entire clinically apparent lesion.
  - Gold standard for melanoma diagnosis.
  - 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 and the deepest level of invasion of the primary lesion
  - Mitotic rate
  - Ulceration
  - Sentinel node (1st node affected in carcinoma)

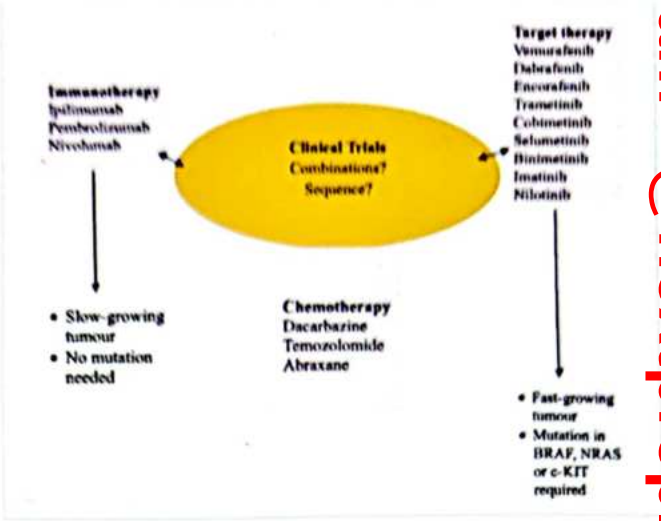


  - Clark's level
  - Immunohistochemistry
    - → Melanocytes stain with S-100, HMB 45 and MART-1 (also known as Melan-A)
  - FISH

- **Staging of melanoma (TNM)**
  - Tumor size
  - Lymph node involvement
  - Metastasis

Treatment of Melanoma

- Wide local excision of a melanoma (with margins measured before excision and determined by Breslow depth, with sentinel lymph node biopsy indicated for tumors with a depth of >1.0 mm)



- If tumor is aggressive
  - Immunotherapy
  - Target therapy
  - Chemotherapy

Mycosis Fungoides

- Tumor of T cell in skin
- Most common variant of primary CTCL (Cutaneous T cell Lymphoma)
- Monoclonal proliferation of predominantly CD4+/CD45R0+ helper T cells and the loss of mature T-cell antigens in the skin and other involved organs.

Course:

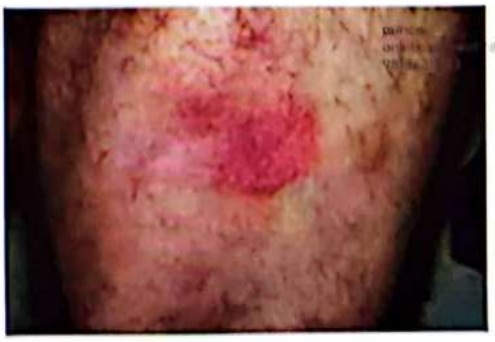
- Slow growing
- High chance of metastasis

Site:

- girdle (abdomen, back)

Clinical Features:

- Polymorphic
  - Starts as scaly macule
  - Turns into erythematous plaque
  - Tumor/nodular stage
  - Erythroderma



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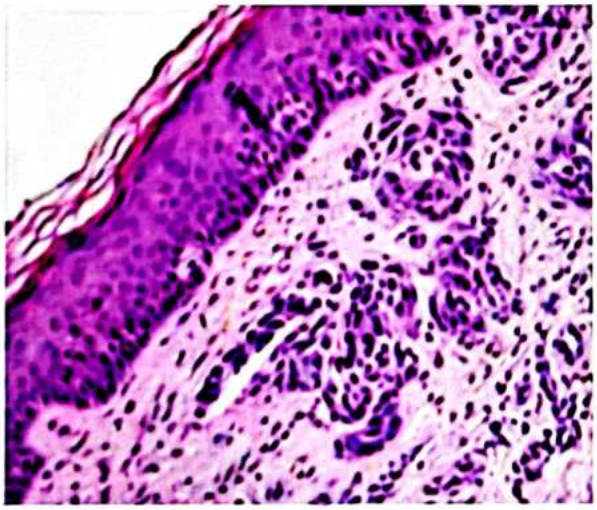

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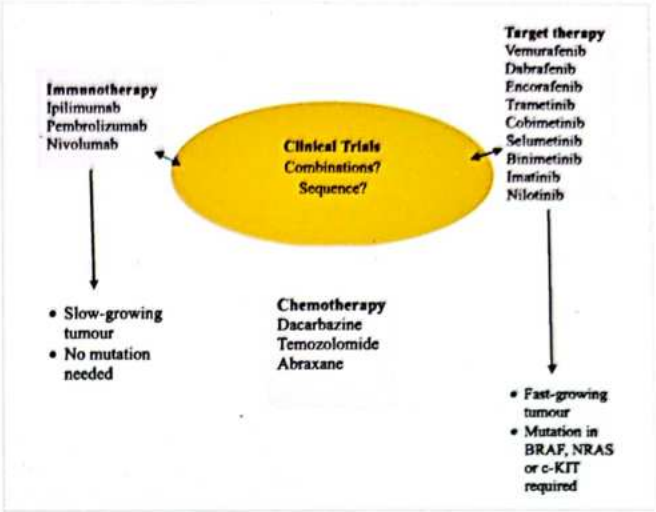

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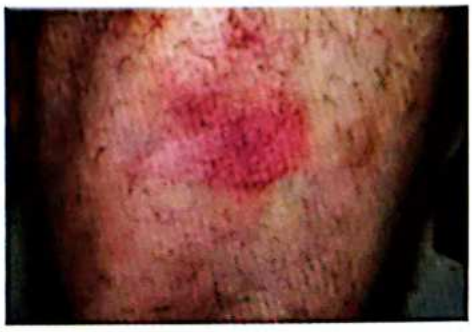
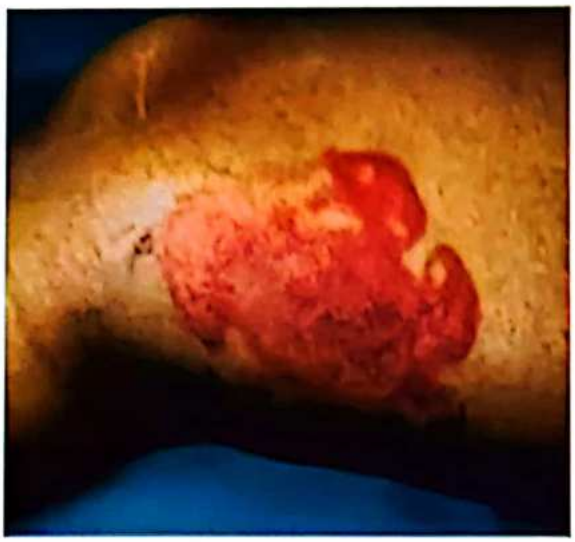
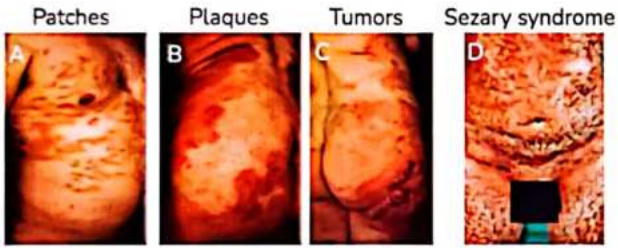
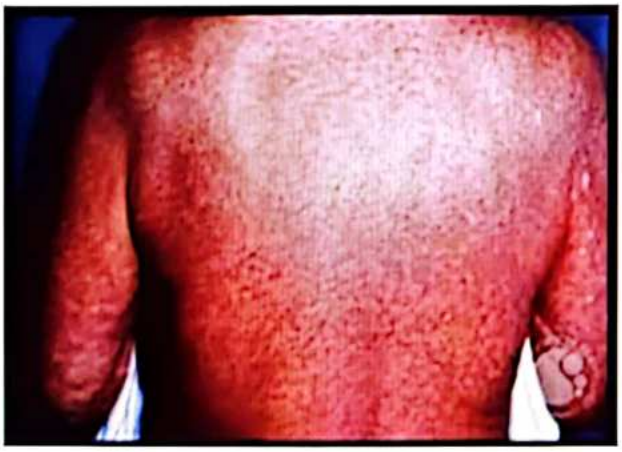




Mycosis Fungoides

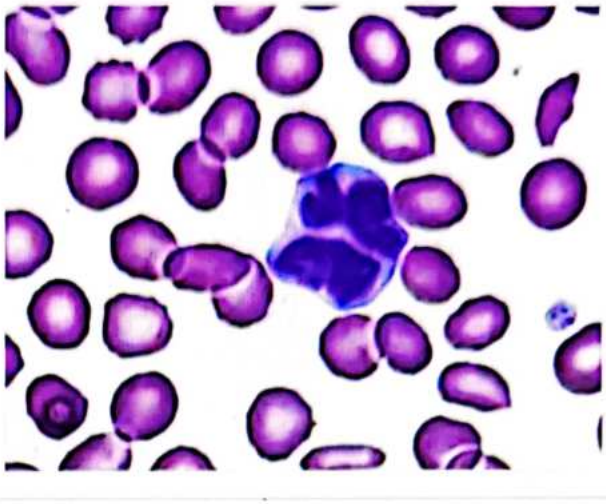


- Asymptomatic



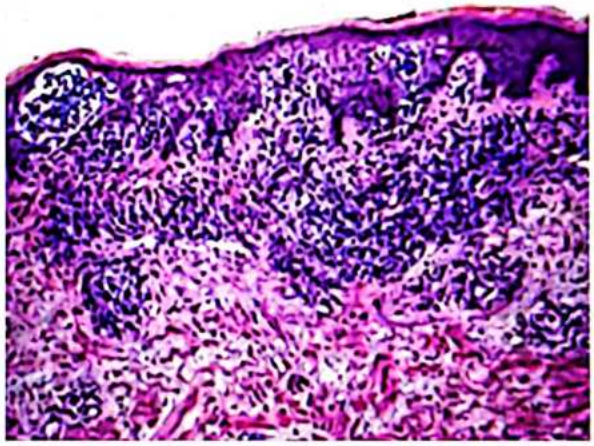
Golden Points

- **Sezary syndrome:** triad
  - Erythroderma
  - Peripheral lymphadenopathy
  - Atypical mononuclear cells (Sezary cells) comprising >20% of total lymphocyte count or total Sezary count of >1000 cells/cumm
- If only Erythroderma is seen, it is Mycosis Fungoides

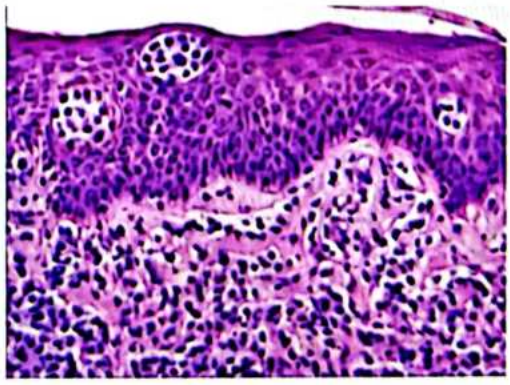


Golden Point: **Sezary cell**: atypical lymphocytes with a grooved or cerebriform nuclei seen both in tissue and blood

Histopathology:



- **Epidermotropism**
  - Invasion of epidermis with T-cells
- Pautrier's microabscess
  - Collection of T-cells



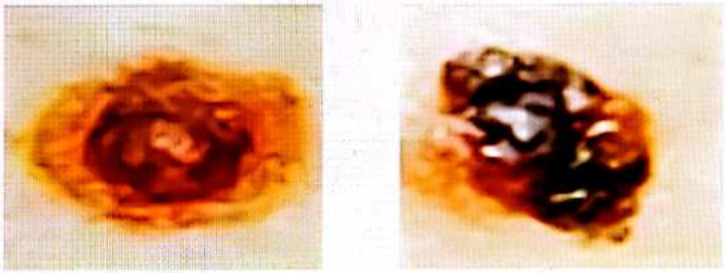
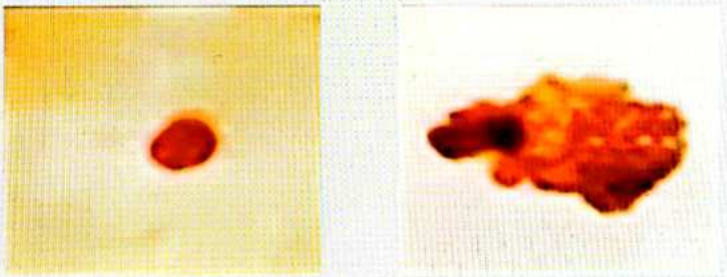
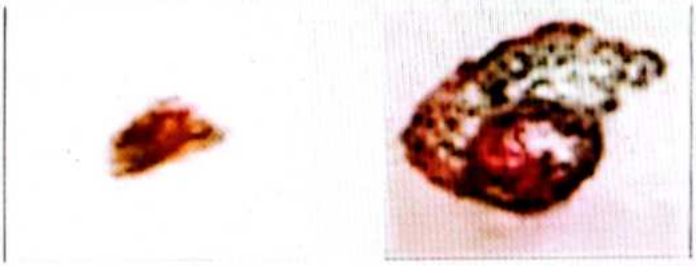
Treatment

- **Patch stage**
  - Topical steroids
  - Nitrogen mustard (mechlorethamine)
  - Carmustine (BCNU)
  - Bexarotene gel
  - PUVA
  - UVB
- **Plaque stage**
  - Total skin electron beam therapy (TOC)
  - Interferon-alpha
  - Oral retinoids
- **Nodal and visceral type**
  - Aggressive chemotherapy consisting of CHOP regimen
    → Cyclophosphamide
    → Doxorubicin
    → Vincristine
    → Prednisolone
- **NEWER**
  - Denileukin diftitox
  - DAB389-IL-2 fusion toxin
  - Histone deacetylase inhibitors

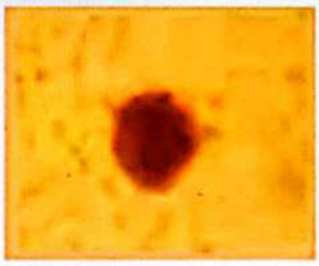
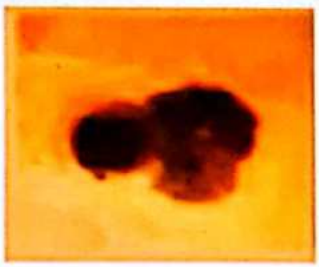
Golden Points - Parapsoriasis

- Scaly erythematous plaques
- Asymptomatic
- Scaling less than psoriasis
- Parapsoriasis can develop into Mycosis Fungoides

Table 20.1

A	Asymmetry: Moles that have asymmetrical appearance	 Symmetrical Asymmetrical
B	Border: A mole that has blurry and /or jagged edges	 Smooth borders Irregular borders
C	Colour: A mole that has more than one color	 Single Colour Multi Colour
D	Diameter: Moles with a diameter larger than a pencil eraser (6 mm or ¼ inch)	 Smaller than 6 mm/0.2 in Bigger than 6 mm/0.2 in

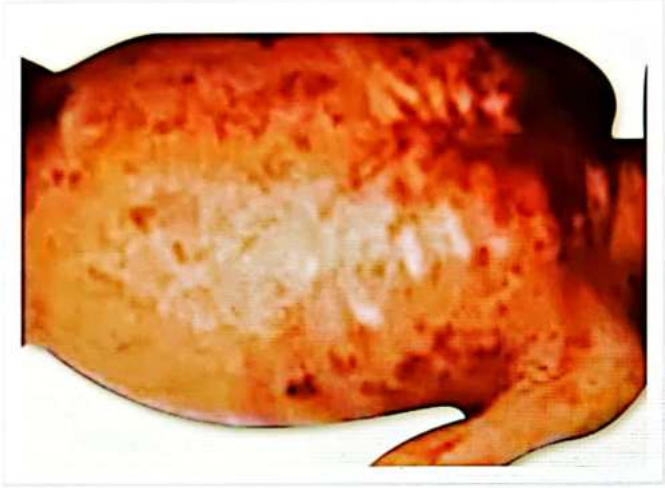
9818635293

E	Evolution: A mole that has gone through sudden changes in size, shape or colour	 No changes  Some changes

21 SYSTEMIC DISEASES AND SKIN

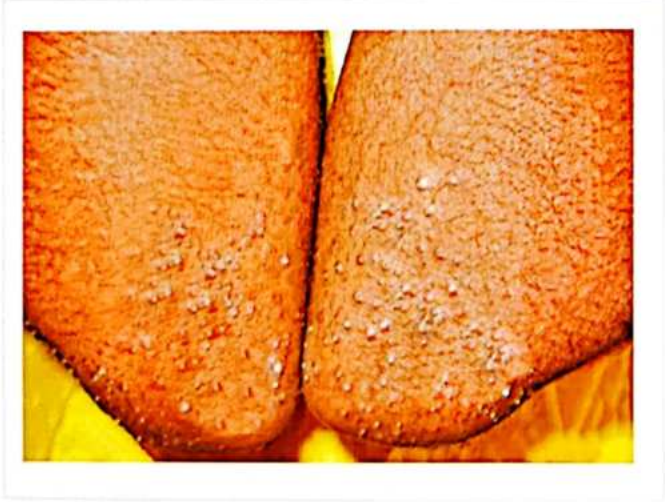
Nutritional Disorders 00:00:29

1. Kwashiorkor 00:00:38



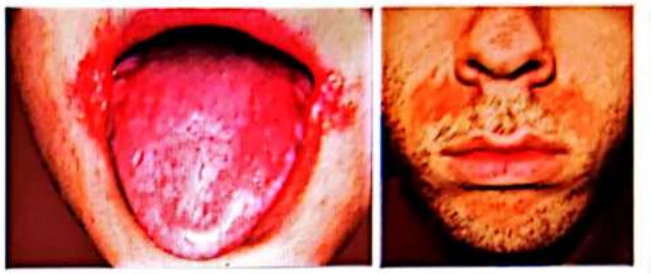
- "Flaky paint" or "Crazy pavement" or Enamel paint dermatoses.
  - It is a dry skin - Hyperpigmented scaly macule.
- FLAG SIGN-HAIR.
  - The part of the hair with malnutrition is lightly pigmented.

2. Vitamin A Deficiency 00:01:34



- **Earliest sign:** Night blindness
- **Earliest skin manifestation:** Dryness.
- Phrynoderma (Toad skin):
  - Mixed deficiency of vitamin A, B, D, E and essential fatty acids.
  - Asymptomatic hyperkeratotic rough papules on elbows and knees

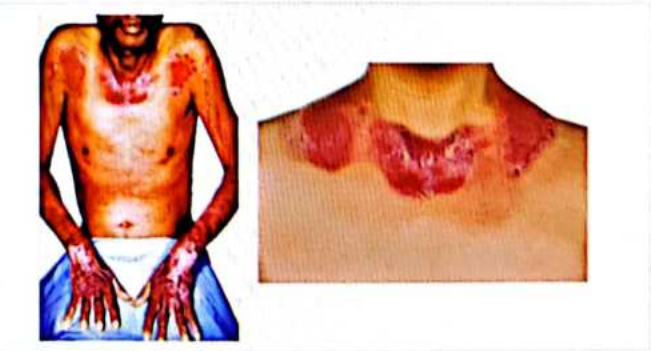
3. Vitamin B2 Deficiency (Riboflavin) 00:03:25



- It causes:
  - Angular cheilitis
  - Glossitis (Red tongue)
  - Dyssebacia (Altered sebaceous gland production) - Seborrheic dermatitis like manifestation.

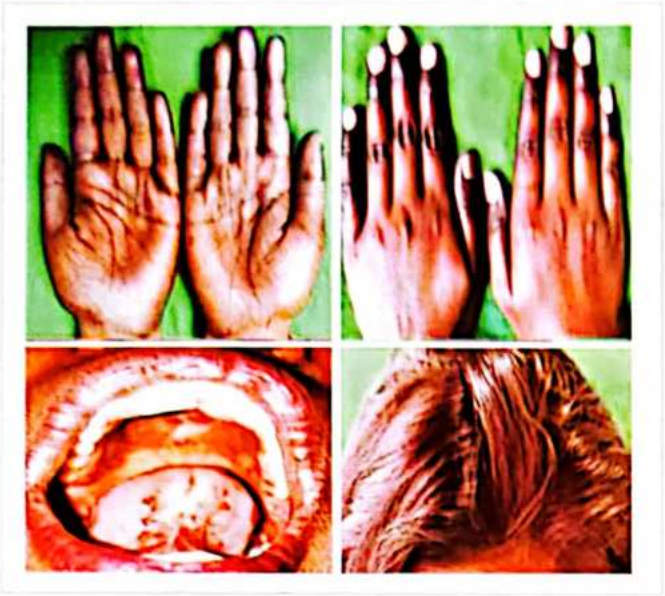
4. Vitamin B3 Deficiency 00:03:59

- Vitamin B3 is also called as nicotinic acid, nicotinamide, and niacin.
- The vitamin B3 deficiency - Pellagra.
- Pellagra can be represented as 4D's:
  - Dermatitis
  - Diarrhoea
  - Dementia- Psychiatric or neurological symptoms can be seen.
  - Death
- Predisposing population-
  - Maize eaters,
  - Chronic alcoholics.



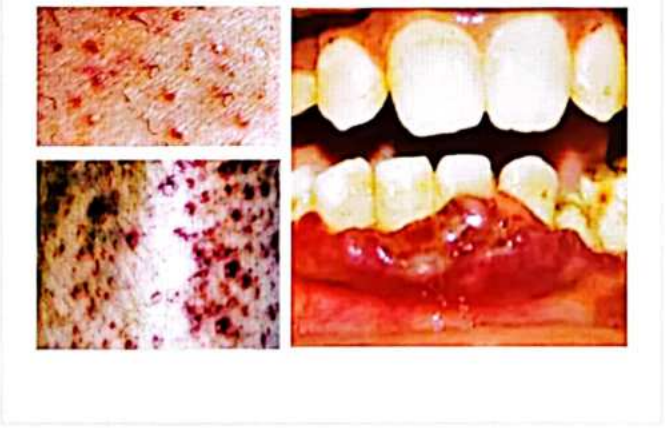
- Exposed parts of the patients - Eczematous lesions on the exposed part (Rashes).
  - dorsum of the hand.
  - V of the neck- lesions may extend to the anterior aspect of the neck to form the hyperkeratotic plaque - Cassal's Necklace.
  - Face
- **Treatment:** Nicotinic acid (given in the form of nicotinamide)

5. Vitamin B12 Deficiency 00:07:10



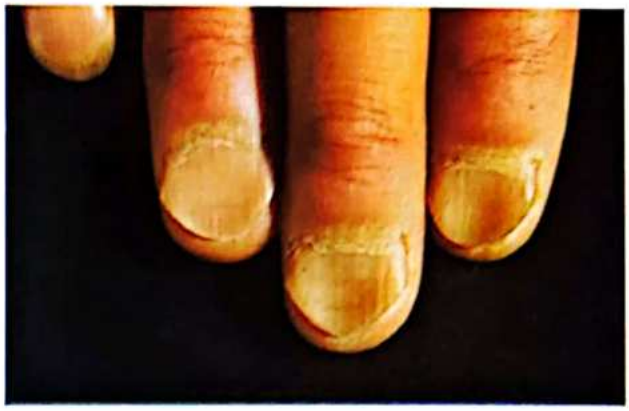
- Addisonian pigmentation (In Addison's disease). These can be seen;
  - On the oral mucosa.
  - Inside the oral mucosa.
  - Palmar creases.
  - Interphalangeal joints.
  - Dorsum of the hand.
  - Nails.

6. Vitamin C Deficiency 00:07:51



- Vitamin C is important to form the endothelium of the blood vessels.
- Presentation:
  - Perifollicular haemorrhages,
  - Corkscrew hair,
  - Spongy gums (Haemorrhage of the gums),
  - Follicular hyperkeratosis (Haemorrhage of nails).

7. Iron Deficiency 00:08:55



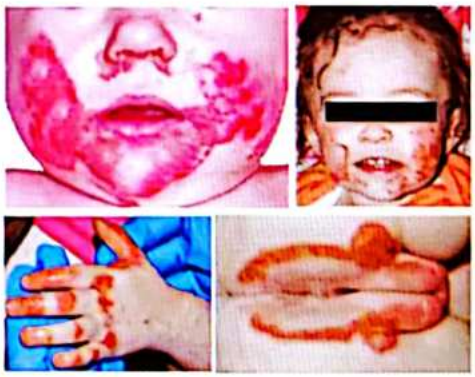
- Spoon shaped nails - Koilonychia.
- Hair fall.

8. Zinc Deficiency 00:09:20

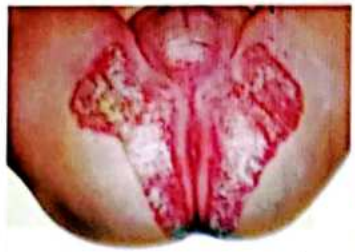
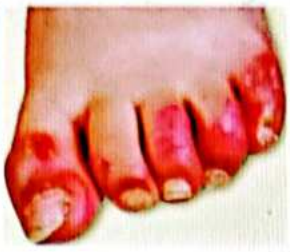
- It can be;
  - **Genetic** - Acrodermatitis Enteropathica
  - **Acquired** - It is seen in chronic alcoholics.

a. Acrodermatitis Enteropathica 00:09:52

- Inheritance - Autosomal recessive.
- Aetiology - ZIP4 transporter is defective in this condition causing inadequate zinc absorption.
- Onset –
  - After 6 months of age (Weaning of the child).
- Triad - DAD
  - D - Dermatitis
  - A - Alopecia
  - D - Diarrhoea.



- **A 6 month old child may present;**
  - Periorificial eczematous rash, also presented at acral areas.
  - Sometimes the periorificial eczematous rash may become psoriasiform seen at feet and hands of the child.

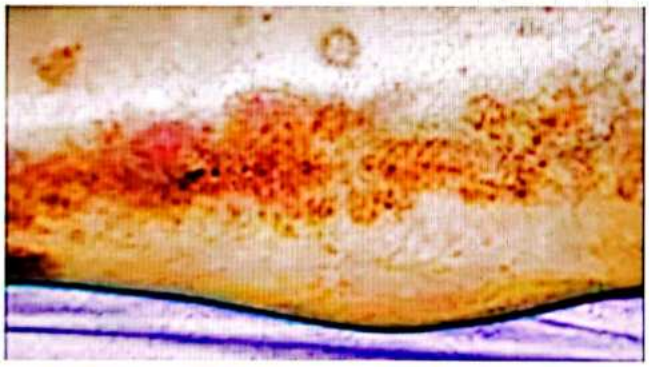
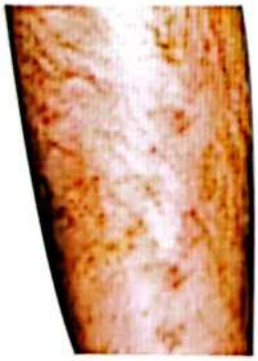
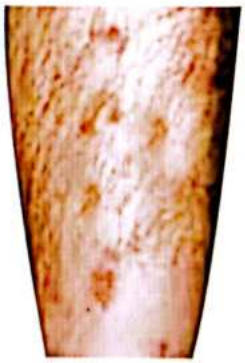
- **Treatment** - Zinc supplement.

Metabolic Disorders 00:12:55

1. Diabetes Mellitus 00:12:56

- Diabetes can lead to many skin manifestations;
  - Acanthosis nigricans,
  - Diabetic dermopathy,
  - Necrobiosis lipoidica,
  - Granuloma annulare,
  - Limited joint mobility
  - Scleredema diabeticorum,
  - Eruptive xanthomas,
  - Perforating disorders,
  - Bullous diabeticorum,
  - Bacterial and fungal infections,
  - Pruritus,
  - Diabetic bulla.

a. Diabetic Dermopathy 00:13:25

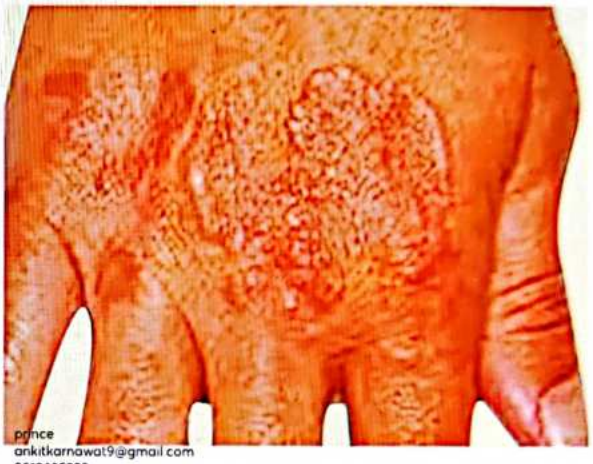




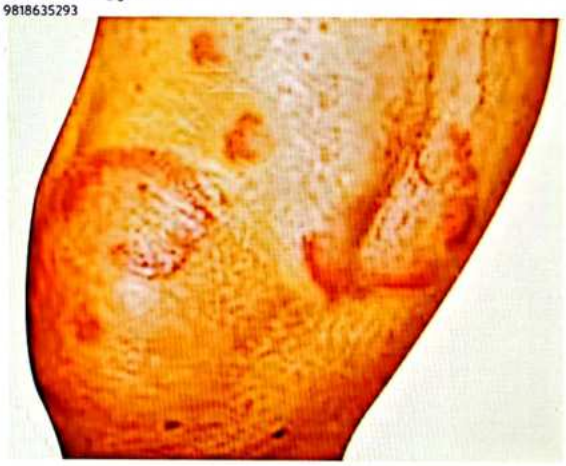
- Asymptomatic, oval, dull-red papules over shin.
- Heal with brown atrophic scar.
- **Marker for complications:**
  - Retinopathy,
  - Nephropathy
  - Neuropathy.

b. Granulomatous Disorders in DM 00:14:16

- There two granulomatous disorders in DM;
  - Granuloma annulare- nonspecific.
  - Necrobiosis lipoidica- specific.

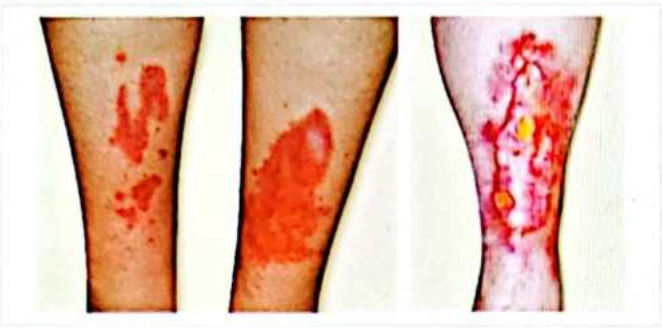
i. Granuloma Annulare 00:15:02





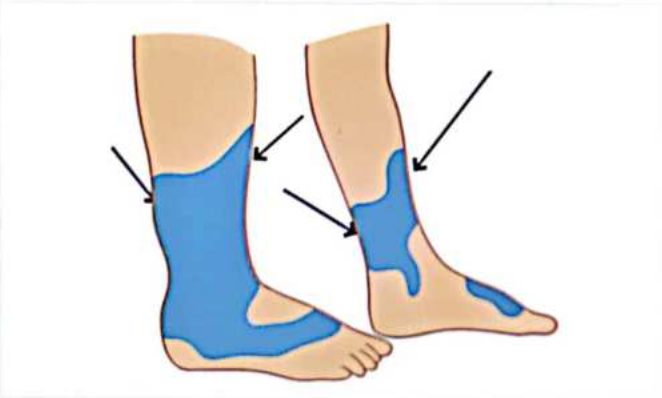
- Nonspecific - Can be seen in diabetic as well as thyroid patients.
- Symptoms: Asymptomatic.
- **Sites:** Dorsa of hands and feet.
- **C.F:** The patient may present with;
  - Annular lesions with raised erythematous, granulomatous borders, with typical central clearing.
- **Biopsy:** Necrobiotic granuloma (Degeneration of collagen).
- **Treatment:** Topical steroids.

ii. Necrobiosis Lipoidica 00:17:02



- **C.F:**
  - Well-demarcated waxy red-brown plaques with an atrophic centre, most commonly.
  - They are located on the shins.
- **Markers** of retinopathy and nephropathy.

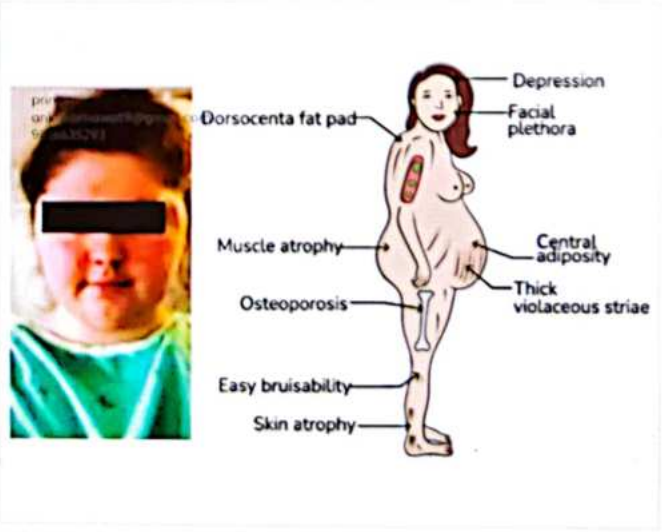
2. Disorders of Thyroid 00:18:01



- Pretibial myxedema.
  - Well demarcated, non-pitting edema on lower legs.
  - It is because of the deposition of glucose aminoglycans.
- Ophthalmopathy.

3. Disorders of Adrenal Gland 00:18:48

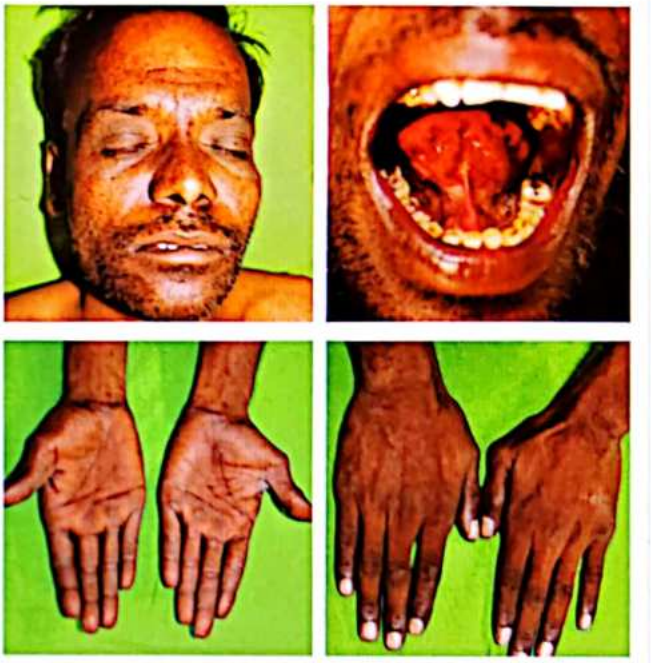
a. Cushing's Syndrome



- Hypertrichosis
- Moon like facies
- Buffalo hump on the back
- Telangiectasia
- Striae
- Central deposition of fat

b. Addison's Disease (Adrenal Insufficiency) 00:19:39

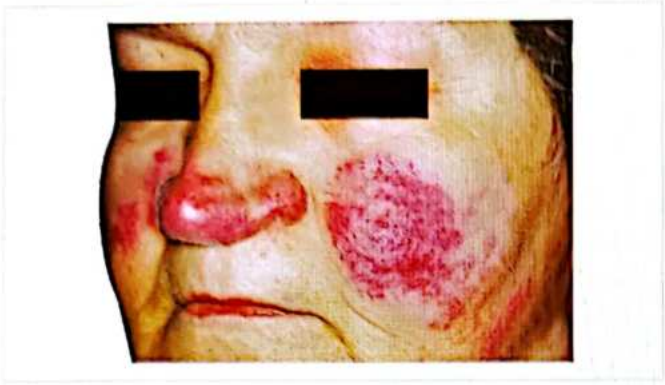
- Addisonian pigmentation can be seen.



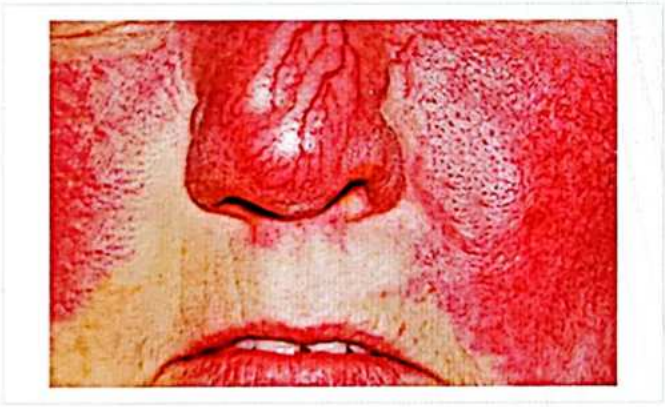
c. Sarcoidosis 00:19:46

- Characteristic feature – Naked granuloma present all over the body
- Multisystem granulomatous disorder which involves skin and many other systems.
  - Pulmonary - Most common organ involved.
  - Peripheral lymph nodes- Lymphadenopathy.
  - Skin
  - Eyes- Uveitis.
  - CVS, renal, liver, CNS manifestations.
- The antigen triggering the granulomatous process can be;
  - Dust,
  - Silica,
  - Infections,
  - Any other antigen.
- Granuloma on the skin can be used for early diagnosis.
- Cutaneous lesions: 2 types
  1. Nonspecific- Erythematous Nodusum.
  2. Specific-

- Sarcoidal granuloma on biopsy are seen.
- Red-brown papule with normal surface.
- Diascopy - Apple jelly nodules on the skin.
- **If present on the face** - Lupus pernio.
  → Erythematous, granulomatous red-brown papules (Smooth-surfaced) present on the convexities of the face



- Angio lupoid:
  → **On the face** - Erythema and telangiectasia are present on the face.
  → **On the body** - Erythematous red-brown papules and nodules with normal overlying surface.

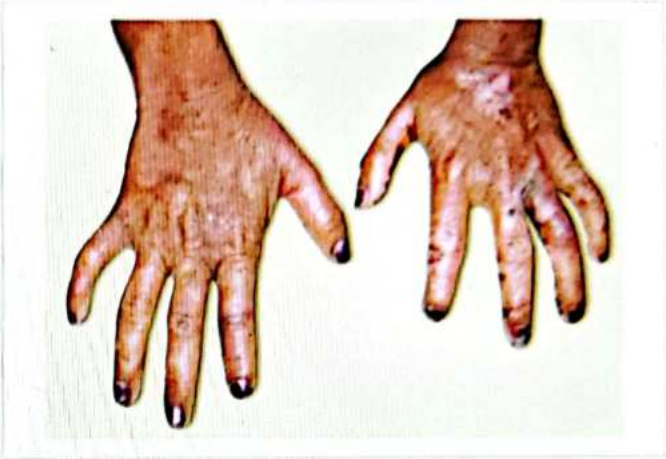


- **Biopsy**
  - Necrobiotic naked granulomas (Surrounded by sparse lymphocytic infiltrate).
  - Schaumann bodies.
  - Asteroid bodies.
- **Treatment:** Steroids and antimalarials.

4. Porphyria 00:24:58

- **Cutaneous disease only**
  - Porphyria cutanea tarda.
  - Congenital erythropoietic porphyria.
  - Erythropoietic protoporphyria.
- **Cutaneous disease and acute attacks**
  - Hereditary coproporphyria.
  - Variegate porphyria.
- **Acute attacks only**
  - Acute intermittent porphyria.

a. Porphyria Cutanea Tarda 00:25:09

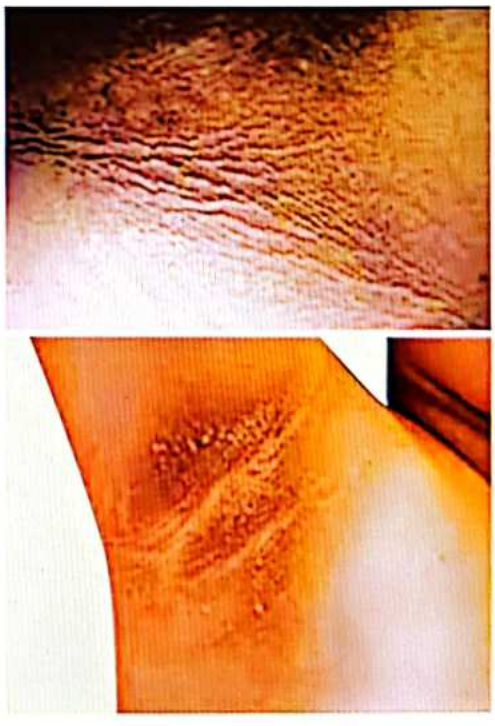


- Aetiology: Uroporphyrinogen decarboxylase deficiency.
- Age of onset: 30-40 years
- Clinical features:
  - Vesicular crusted lesions on the exposed parts.
  - Burning sensation.

5. Paraneoplastic Dermatoses 00:26:04

- These are associated with internal malignancies, but themselves are not malignant.

a. Acanthosis Nigricans 00:26:44

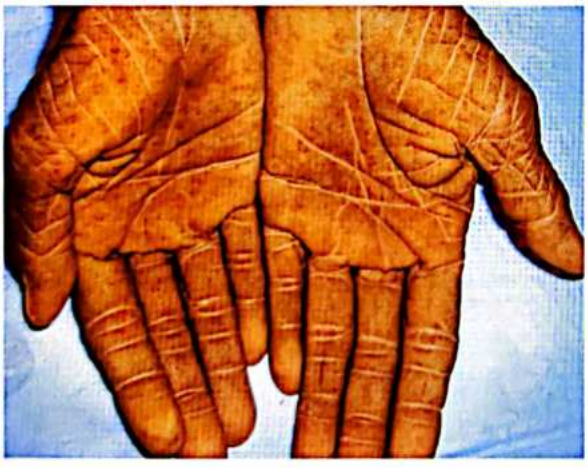


- It is a misnomer, because there is no acanthosis in biopsy.
- Characteristics feature - Velvety hyperpigmentation on flexors seen on;
  - Neck
  - Axilla
  - Groin area.
- Important marker for insulin resistance.
- Skin tags are the common skin condition associated with acanthosis nigricans.
- Pathogenesis: Increased production of IGF-1 (Insulin like growth factor 1).
- Commonly seen in associated conditions, like;
  - DM
  - Obesity
  - Metabolic syndrome
  - OCP
  - Insulin resistance
- It is of 2 types:

1. Benign
   - Mild
   - Localised
   - Associated with diabetes and MS.

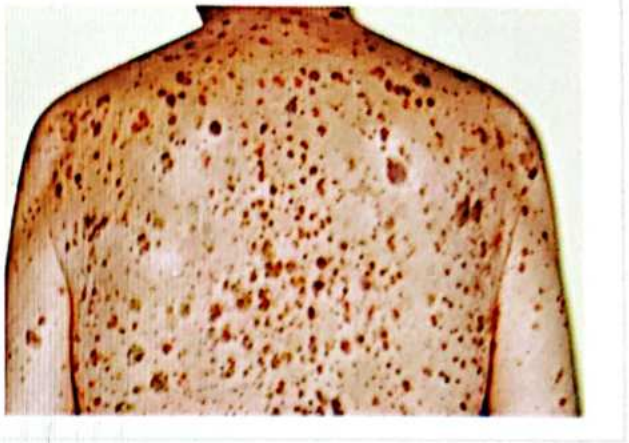
2. Malignant
   - Rapid in onset.
   - Generalised.
   - Bilaterally symmetrical.
   - Associated with Adenocarcinoma of GIT.

b. Tripe Palms 00:30:45



- Acanthosis nigricans on the palms
- If presented with clubbing- associated with bronchogenic carcinoma
- Otherwise seen in adenocarcinoma of GIT.

c. Sign of Leser-Trelat 00:31:07

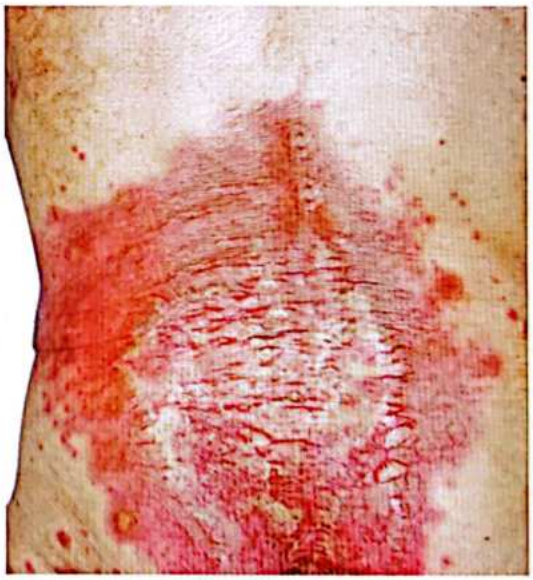


- Sudden eruption of seborrheic keratosis on the trunk of the patient
- It is associated with:
  - Adenocarcinoma of GIT.
  - Carcinoma of breast.

d. Migratory Erythema 00:31:50

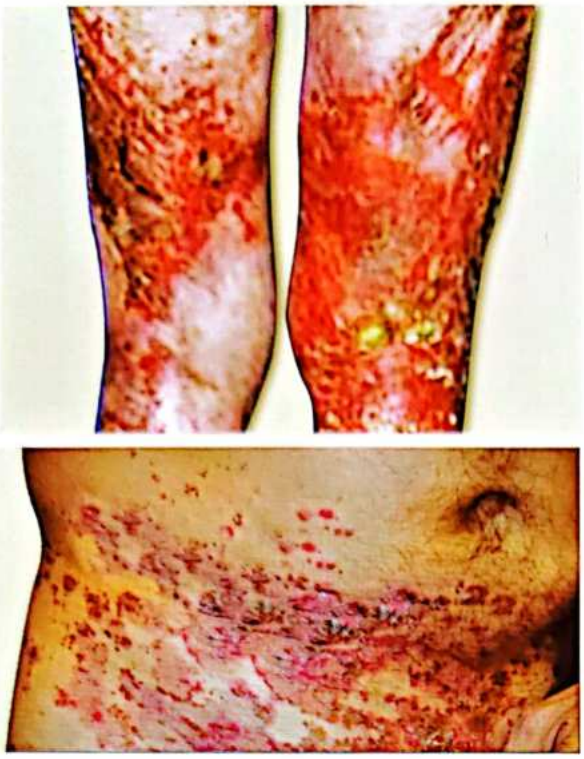
- Two types

1. EGR



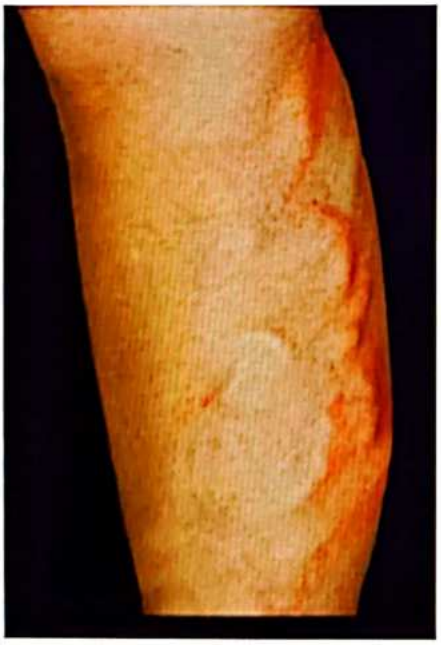
- Erythema Gyratum Repens.
- It is mobile, concentric, often palpable, erythematous, wave-like bands.
- Migrate from day to day, about 1 cm daily.
- Associated with lung cancer.

2. NME



- Necrolytic Migratory Erythema.
- Widespread painful polycyclic inflammatory migratory rash is present on the anogenital region and trunk.
- Associated with glucagon-secreting alpha-cell tumour of the pancreas.

e. Migratory Thrombophlebitis 00:34:02

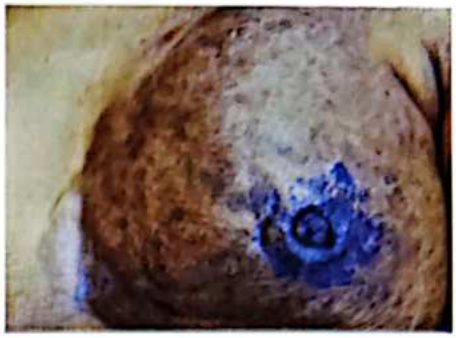


- Also known as Trousseau's sign
- **Associated with** pancreatic carcinomas.

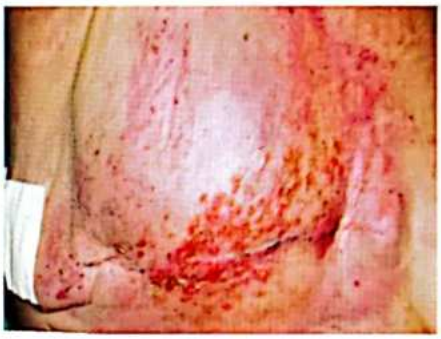
f. Breast cancer- 00:35:30

- Breast cancer has a direct invasion of the skin.

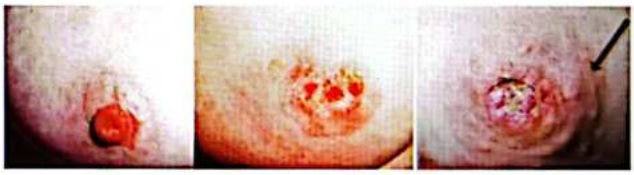
i. Sclerodermatous (Thickened indurated skin): Carcinoma en cuirasse.



ii. Skin may be inflamed - Carcinoma erysipeloides.



iii. Paget's Disease of Nipple 00:36:08



- Origin: Intraductal carcinoma of breast
- Characteristic cells upon biopsy: Paget cells in Stratum spinosum
- Seen on the nipple, unilaterally, with eczematous lesions, with a typical well-demarcated border.
- Part of areolar tissue is involved.

Important Information

- It can be confused with eczema, where upon misdiagnosis and treatment the patient can't resolve the symptoms.
- Eczema is presented bilaterally.

g. Cutaneous Metastasis 00:37:31

- Extramammary Paget's disease.
- Most common sources: BLOCK.
  - B - Breast
  - L - Lung
  - O - Oesophagus
  - C - Colon
  - K - Kidney
- Most commonly present on head, neck and upper trunk.
- Umbilicus- Sister Mary Joseph nodule, related most commonly to bowel tumours.

Summary on Cutaneous Conditions may be presented with the Underlying Visceral Malignancies.

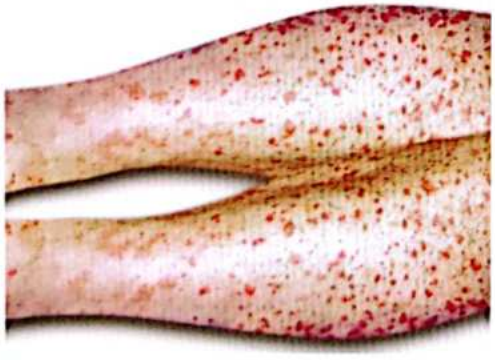
- **Hyperkeratotic diseases:**
  - Acanthosis nigricans,
  - Acquired ichthyosis,
  - Leser-Trelat sign.
- **Collagen vascular diseases:**
  - Dermatomyositis,
  - Scleroderma.
- **Reactive erythema:**
  - Erythema gyratum repens,
  - Necrolytic migratory erythema.
- **Neutrophilic dermatosis:**
  - Sweet's syndrome,
  - Pyoderma gangrenosum.
- **Bullous disorders:**
  - Paraneoplastic pemphigus,
  - Dermatitis herpetiformis.
- **Migratory thrombophlebitis.**

Inflammatory Dermatoses with Systemic Associations

00:38:21

Vasculitis

00:38:30



- Inflammation of vessels
- Involves both skin and systemic.
  - Only skin involved (Rare) - Leukocytoclastic or cutaneous small vessel vasculitis.
- Characteristic feature - Palpable purpura.
  - Purpura - If pressed with a diascopy, no blanching or extravasation of blood is seen.
  - Palpable - Lesions are felt.
- Located- on the lower limbs.
- Associated with constitutional features:
  - Fever
  - Malaise.

Important Information

- Upon **Diascopy**, vasodilation and erythema can be subsided due to peripheral spread of blood.
- Upon dioscopy, if the purpura remains intact, then the vasculitis should be checked for sensation.
  - **Palpable** - Vasculitis (Inflammation of the vessels).
  - **Nonpalpable** - Platelet or clotting disorders.

Areas in the history of a patient with cutaneous vasculitis that may give **clues indicating systemic disease**:

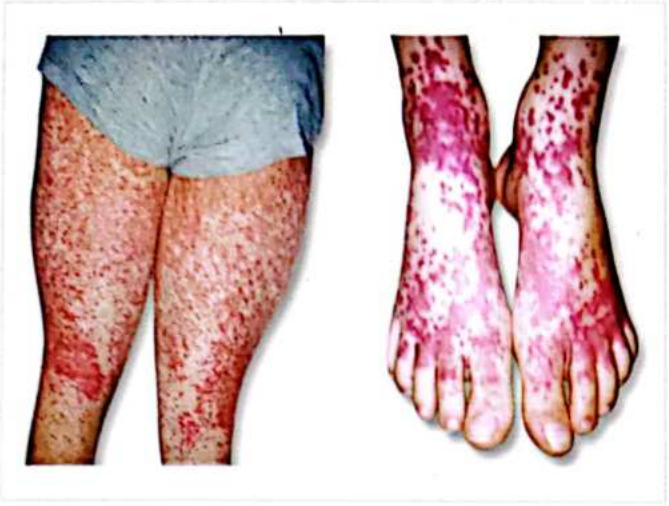
- Weight loss, fatigue, fever.
- Arthralgia, myalgia, arthritis.
- Dry eyes, dry mouth.
- Red eye, eye pain, vision loss.
- Nasal or sinus congestion.
- Ear pain.
- Oral / nasal ulcers.
- Chest pain / dyspnoea.
- Abdominal pain, blood in faeces.
- Blackouts, weakness, fits.

Cutaneous Small Vessel Vasculitis or Leukocytoclastic Vasculitis 00:41:34

- It is a pure skin manifestation.
- Idiopathic
- Self-limiting.

Henoch-Schoenlein Purpura (HSP) 00:41:50

- Systemic small vessel vasculitis.
- Usually seen in children.
- Aetiology: Infections, drugs.
- Palpable purpura.
- On biopsy - IgA-containing immune complexes are seen.



- Organs affected;
  - **Skin**
    → Palpable purpura on lower limbs and buttocks.
  - **GIT**
    → Nausea,
    → Vomiting,
    → Abdominal pain,
    → Bloody stools
  - **Joints**
    → Joint pains - Arthralgias.
  - **Kidneys**
    → Haematuria,
    → Proteinuria,
    → RBC casts in the urine.
- Treatment
  - Usually self-limiting.
  - In adults- colchicine and steroids can be given.

Neutrophilic Dermatoses 00:44:23

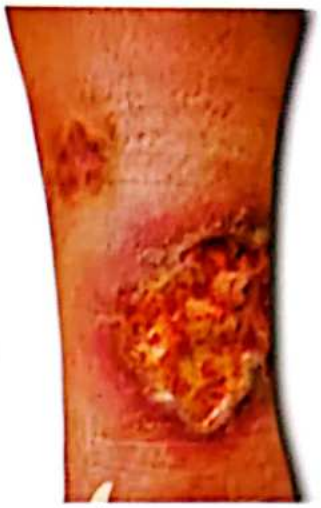
- Neutrophilic infiltration is seen.

Pyoderma Gangrenosum 00:44:32

- It is a misnomer - No bacterial infection is present.
- Ulcerations of the skin associated with an underlying systemic disorder.
  - **Association:** IBD, Rheumatoid arthritis; haematological malignancy or monoclonal gammopathy.
- It is a diagnosis of exclusion - No characteristic histopathology feature or no laboratory evaluation evidence.

Case: A patient may present the features of ulceration of skin. Many diagnostic tests are done but the test results are negative.

- Then it should be considered as PG, which has a diagnosis of exclusion.



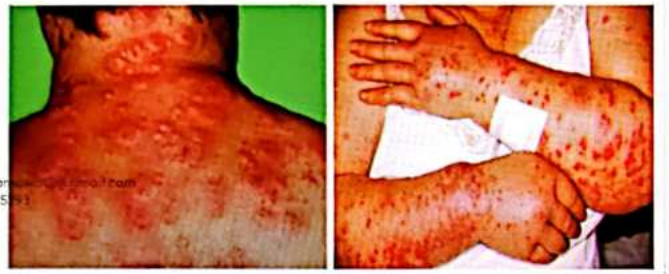
- Painful papules or lesions on lower limbs which break down to form ulcers.
- Ulcers - Violaceous undermined margin.
  - **Undermined margins are also seen in;**
    → TB chancre.
    → Chancroid
    → PG.
- Pathergy: positive

- PG can be healed with atrophic cribriform scarring - Characteristic Feature.
- **Treatment:**
  - Steroids
  - Dapsone

- Colchicine.

SWEET Syndrome 00:47:30

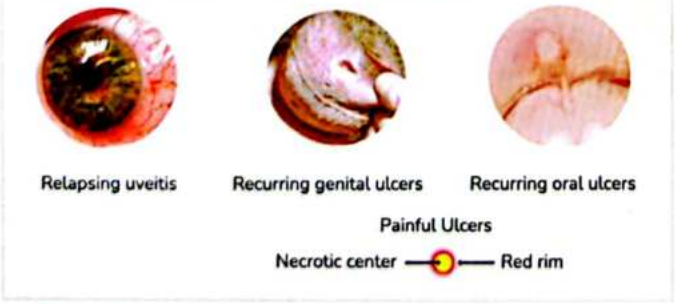
- Also called as acute febrile neutrophilic dermatosis.
- More common in females.
- Biopsy: Neutrophilic infiltrate is seen.
- **Associations:**
  - Streptococcal respiratory tract infections.
  - Gastrointestinal infections by Salmonella and Yersinia.
  - Mycobacterial infections.
  - IBD, RA, sarcoidosis.
  - Haematological Malignancies.
  - Drug - G CSF.



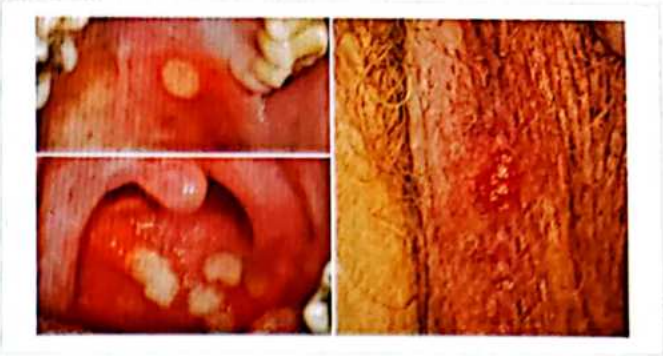
- Acute onset of fever and constitutional symptoms are present.
- On the upper part of the body or upper trunk;
  - Sudden appearance of erythematous papules occurs.
- Papules are oedematous - Pseudo vesiculation (Appearance - Fluid filled papules, but no fluid is present in actual).
- **Treatment:**
  - Steroids,
  - Potassium iodide.

Behcet's Disease 00:49:45

- Multisystem inflammatory disease of unknown aetiology.
- **On Biopsy** - Systemic vasculitis along with neutrophilic dermatosis involving all types and sizes of blood vessels are present.
- **Histopathology:** Vasculitis and thrombosis.

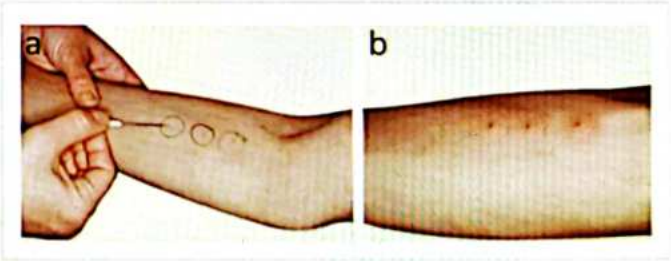


- Triad of symptoms:
  - Relapsing uveitis
  - Recurrent genital ulcers
  - Recurrent oral ulcer.



- Appearance of ulcer - Aphthae (Small erythematous erosions with necrotic centre).
- They usually heal within a week.
- Oral ulcers - Heals without scarring.
- Genital ulcers - Heals with scarring.
- Systemic features:
  - Ocular:
    - → Posterior (Retinal vasculitis) - Most diagnostic feature.
    - → Anterior uveitis, hypopyon.
  - Joint:
    - → Non-erosive, asymmetrical seronegative oligoarthritic (MC-knee).
  - Multisystem Involvement:
    - → Pulmonary, cardiac, GI, neurological involvement can also be seen.

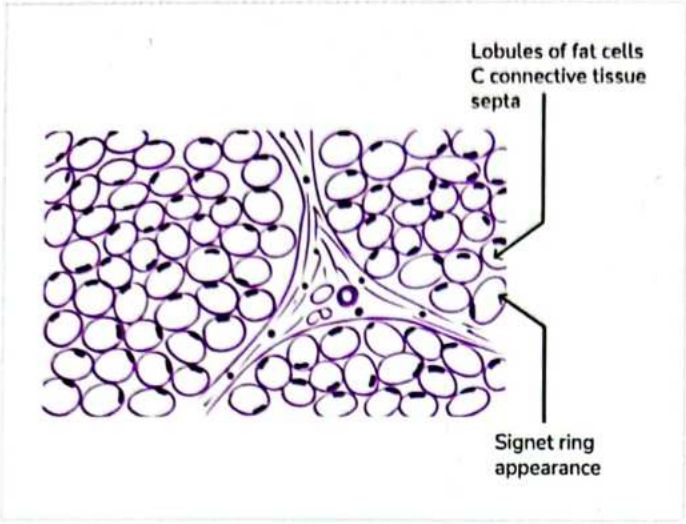
- **Pathergy Test**



  - Pathergy test is positive in Bechet's disease.
  - **Manifests within 48 hours,** as an erythematous papule (>2 mm) or pustule at the site of a skin needle prick or after intracutaneous injection.
  - **Also positive in:**
    - → PG
    - → RA
    - → Crohn disease and
    - → Genital herpes infection.
- **Treatment:**
  - Topical anaesthetics - Because it is painful.
  - Steroids or colchicine.

Panniculitis

00:53:25

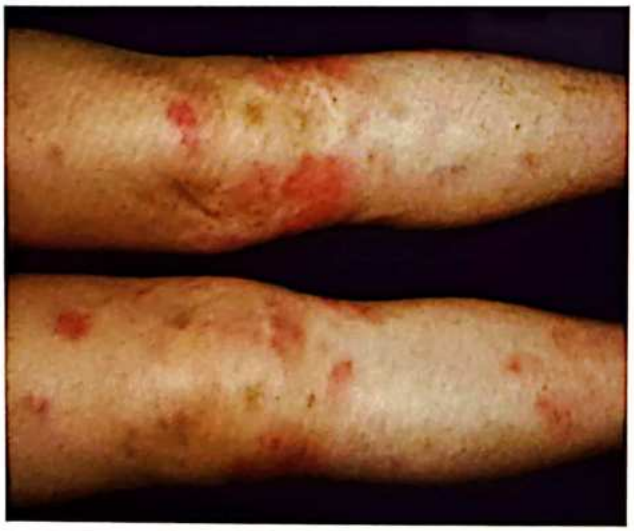


- Inflammation of the panniculus.
- **Panniculus** - Subcutaneous fat.
- It can be lobular or septal.
- There are many types of panniculitis
  - Erythema Nodosum (most common)

Erythema Nodosum

00:54:23

- Most common panniculitis.
- Cutaneous reactive processes triggered by a wide variety of infectious and inflammatory disorders, it becomes difficult to identify the cause.
- Symptomatic, acute onset and self-limited but it can recur.
- **Most common triggers:**
  - Bacterial infections
  - Sarcoidosis
  - Inflammatory bowel disease.
  - URTI by group A B-hemolytic Streptococcus.
    → Frequent cause in children and young adults.
  - Tuberculosis



- Presentation on anterior aspect of shin.
- Erythematous tender papules and nodules.
- **Recurrent and heal on their own** without any atrophy with normal overlying surface.

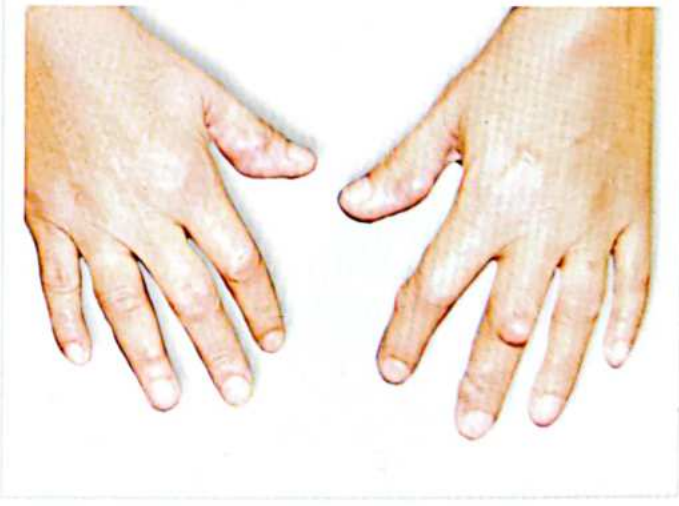
Important Information

- **Erythema Leprosum** (Discussed in leprosy) is a Type II reaction. The lesions are newly formed for every 24 hours in the lower limbs.
- **Erythema induratum** of basin, which is a nodular tuberculae seen in the posterior cast of the females, which heals with scarring.
- But in **EN**, healing occurs without scarring.

- **On biopsy:**
  - Septal panniculitis is present without vasculitis.
  - Miescher radial granulomas are seen.
- **Treatment:**
  - Leg elevation
  - NSAIDS
  - Colchicine
  - No steroids are given.
    → Reason:
    – In India, TB can be one of the causes of EN and other infections can cause EN.
    – If given, they aggravate the infections.

Rheumatoid Nodules

00:57:51



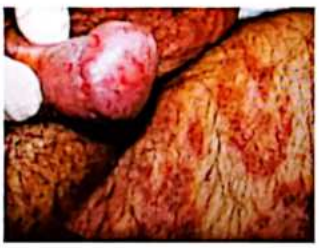
- Extra-articular manifestations of rheumatoid arthritis.
- **Site:** Dorsa of hands and appears as nodules.
- Accelerated rheumatoid nodulosis (ARN):
  - In patients with RA, methotrexate can be given for treatment.
  - Sometimes it may lead to multiple rheumatoid nodules - ARN.

Reactive Arthritis

00:58:49

- Also known as: Retiers disease.
  - It is **different from Ritter's disease** - A Staphylococcal Scarlet Skin Syndrome.

- **HLA association:** HLA B27, B51.
- Triggers:
  - **GI infection**
    - → Y shigella.
    - → Yersinia.
    - → Campylobacter.
  - **HIV** - Commonly seen.
  - **NGU-SARA** - Caused Chlamydia trachomatis, called sexually acquired reactive arthritis.
  - **SARA** can show triad of symptoms:
    - → Polyarthritis (Of over a month's duration following gastrointestinal or lower genital infection) - Non suppurative, lower limbs.
    - → Urethritis.
    - → Non-gonococcal conjunctivitis.



- **Reactive arthritis has two manifestations in skin;**
  - Keratoderma blenorrhhagicums.
    - → Patients develop small vesicles on the hand or palms and are covered by the hyperkeratotic heaped-up crusts.
  - Circinate balanitis.
    - → It is usually seen in the glans with asymptomatic circular erosions.
    - → It is not painful as in herpes (Vesicles are present - Reason for pain).

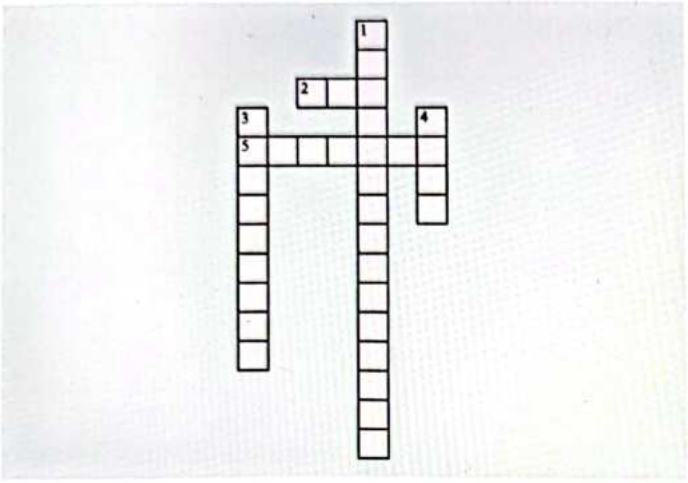
Important Information

- Circinate balanitis can be presented as a case like a truck driver with nongonococcal urethritis or a person who has come after a leisure trip with hyperkeratotic lesions on the hand or erosions in the genital tract.

- Renal involvement is usually asymptomatic - Proteinuria, microscopic haematuria and aseptic pyuria.
- **Treatment:**
  - Keratolytic
  - Methotrexate,
  - In patients with HIV - Acitretin.

CROSS WORD PUZZLES

Crossword Puzzle



Across

2. Epidermal Inclusion Cyst
5. Lines collagen and muscle fibers

Down

1. chronic itching
3. Lines embryonic development
4. Relaxed Skin Tension Lines

prince
9818635293

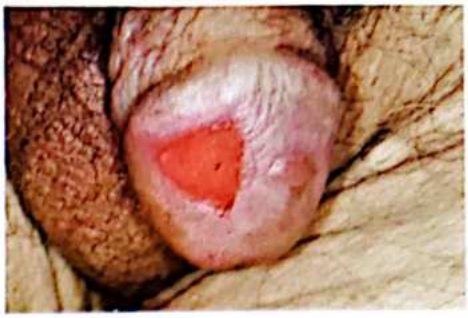
22 ADVERSE DRUG REACTION

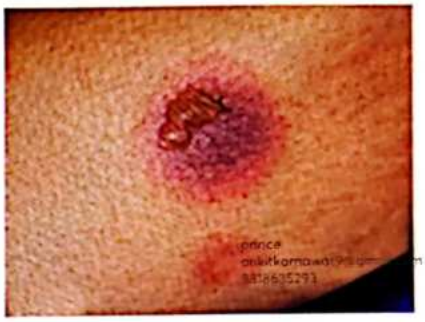
Adverse cutaneous drug reaction

Exanthematous reaction Maculopapular rash	Severe cutaneous adverse drug reaction(SCAR)
Urticaria, angioedema	Stevens johnson syndrome
Anaphylaxis	Toxic epidermal necrolysis(TEN)
Fixed drug eruption	Drug hypersensitivity reaction(DRESS/DHS)
Lichenoid eruptions	Acute generalized exanthem
Photosensitivity reaction	Exfoliate dermatitis
Acneiform eruption	Serum sickness-like rash
Erythema multiforme	
Psoriasiform rash	
Pigmentation	
Hair loss	

Fixed Drug Eruption

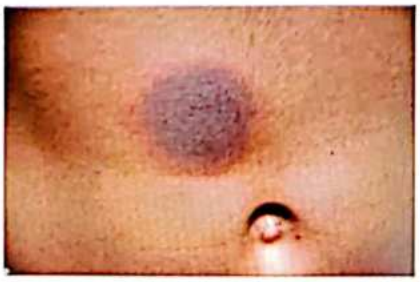
Type of HS	Type 4 hypersensitivity reaction
Characteristics	Same place, same morphology, same lesions When the same drug is taken regularly
Pathogenesis	Memory T-cells
drugs	Co-trimoxazole, tetracyclines, and NSAIDs

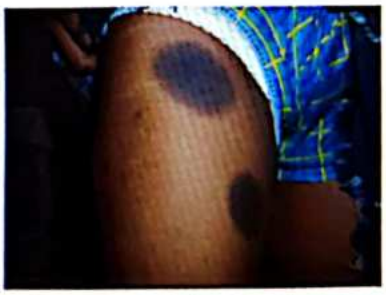




Images shows -

- circular well, defined plague
- Violaceous
- enthymemes
- Hyperpigmented
- With overlined blister
- He develops round lesions on trunks or genital areas(erosion always at the same site)
- It heals, leaving behind bad post-inflammatory hyperpigmentation.

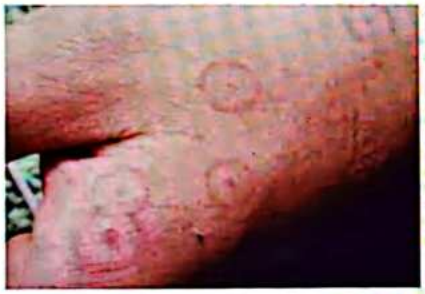


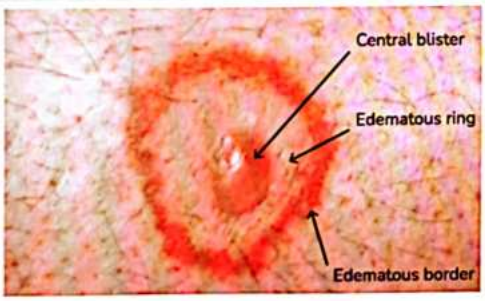


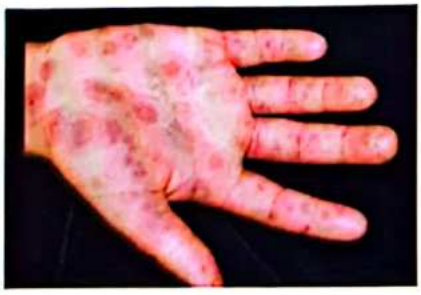
Treatment: topical sterpids and antibiotics combinations

Erythema Multiforme

Etiology	90% HSV, mycoplasma 10% penicillin, quinolones, tetracycline, rifampicin, sulfonamides, NSAIDs, anticonvulsants, etc.,
Onset	72 hours
Distribution	acral
Treatment	Acyclovir, topical steroids and antibiotics



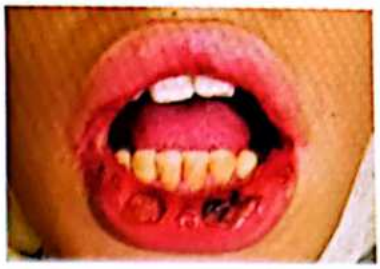




Target lesions are:

- Central purpura
- Middle edema
- Peripheral erythema
- Mucosal involvement can also be seen

prince
ankitkarnawat9@gmail.com
9818635293



Stevens johnson syndrome/ toxic epidermal necrolysis (SJS& TEN)

Body surface area <10% - call it is as SJS

Body surface area > 30% - call it as TEN

Body surface area - 10-30% SJS overlap

- TEN is also called **LYELL's disease**
- Important about SJS/TEN - skin +mucosa and systemic involvement
- Drugs involved: 85% of cases - idiosyncratic, not dose-dependent
  - Penicillin, sulfonamides, quinolones, cephalosporins, and acetaminophen
  - Longer periods: carbamazepine, phenobarbital, phenytoin, oxicam group of NSAIDs, antitubercular drugs, antiretroviral agents, and allopurinol.

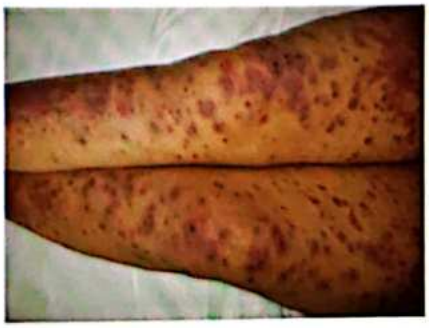
Question: what genetic factors predispose you to SJS/TEN drugs?

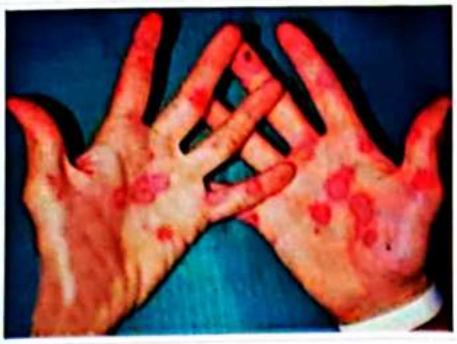
1. Carbamazepine- HLA-B *1502
2. Allopurinol- HLA-B* 5801

Patho- due to the drug, there is inflammation – apoptosis and necrotic keratinocyte that comes out as sheets

Drug stimulates CTCL- releases the enzymes

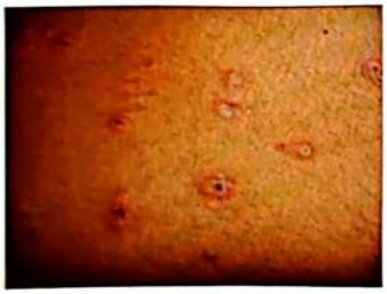
Fas ligands, perforin, granulysin, granzymes

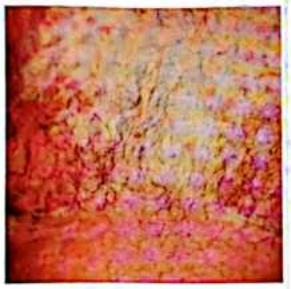


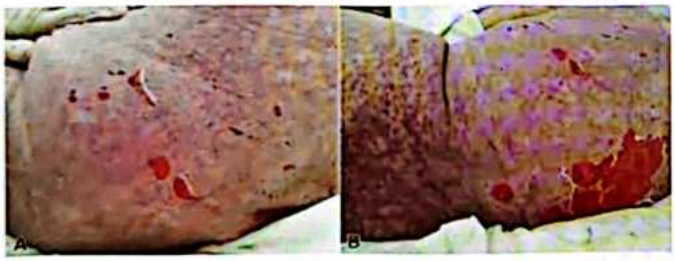


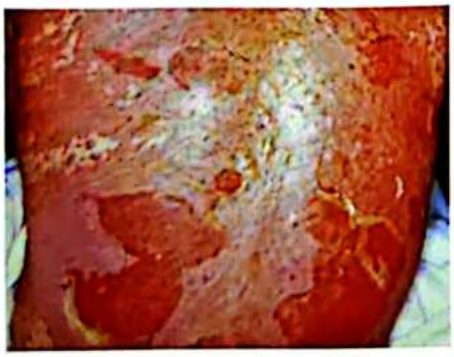
Patient will have

- Fever
- Constitutional symptoms
- Purpuric lesions on limbs and trunks
- Targetoid lesions: hands and feet: ⅔ zones

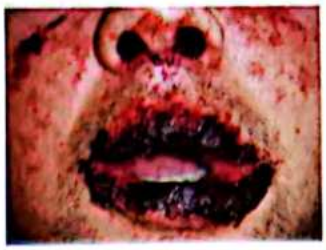


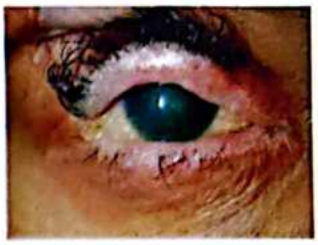






- Central necrosis
- Sheets of erythema developing over the body which breakdown at places of friction
- Eroded areas with erythema, this is where body surface area
- Severe kind of drug reaction.





- >/= 2 mucosal involvement
- Conjunctival mucosa. Oral mucosa, genital mucosa, esophagus mucosa
- Severe crusting and erosions
- Ectropion and conjunctivitis
- Prone to systemic complications:
  - Hypothermia
  - Sepsis
  - Hypocalcemia
  - Hypoalbuminemia
  - Septic shock

Chances of mortality:

- SJS - 5%
- TEN-30%
- That is classifying is important based on BSA

SCORTEN PARAMETERS	Score
Age years > 40 years	1
malignancy	1
tachycardia	1
Initial surface of epidermal detachment	1
Serum urea	1
Serum glucose	1
bicarbonate	1
SCORTEN SCORE	Predicted mortality (%)
0-1	3.2
2	12.1
3	35.8
4	58.3
5	90

SCORTEN predicts mortality

- if the score is 5, then mortality is 90%
- Prognostic marker
- Biochemical mediators are

Serum urea
Serum glucose
Bicarbonate

Treatment:

- Supportive treatment
- No antibiotics to be given
- Monitor under ICU setting
  - Fluid management
  - Temperature
  - Antipyretics
  - Genital mucosa
- In certain cases- IV immunoglobulin
- Cyclosporine
- Steroids - if the patient case worsens then only go for supportive treatment

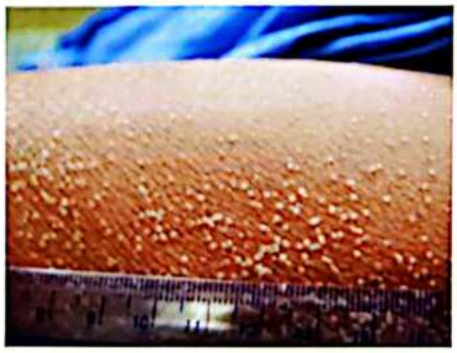
Dress

Drug reaction with eosinophilia and systemic symptoms

1. Anticonvulsants
2. DAPSONE

- Also called as Anticonvulsants hyper syndrome/dapsone hyper syndrome
- Form - hepatic and renal involvement
- Splenomegaly
- Hemolytic changes are there

Acute Generalized exanthematous Pustulosis



- Sudden appearance of sterile pustules
- Non-follicular confluent pustules
- Predominantly over face or flexures
- Accompanied with Leukocytosis and fever
- Confused with pustulosis psoriasis- but this happens after drug
- Resolves spontaneously and disseminates rapidly with desquamation
- Ampicillin, macrolide, tetracyclines are common culprits

Erythroderma

- Syn. exfoliate dermatitis
- Anticonvulsants, chloroquine, ATT, lithium etc,

Erythema Nodosum

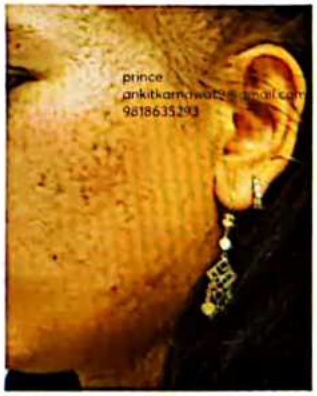
Drugs causing erythema nodosum -

- Acetylsalicylic acid
- Bromides
- Gold
- Iodides
- Penicillin
- Salicylates

Lichenoid Eruption

- Beta blocker
- Chloroquine
- Gold
- Carbamazepine
- Thiazides
- Quinine

Drug Induced Pigmentation



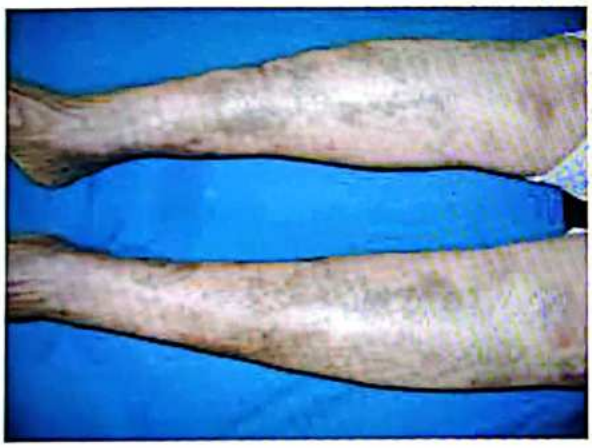
- Minocycline- given to acne patient
- Black blue pigmentation or scars
- Deposition of iron chelates
- Lupus-like reaction

Question: A patient gets treated for acne, develops black-blue scar, who is the culprit?
Answer: Minocycline

A patient with Melasma comes

1. You give hydroquinone
2. It increases pigmentation.
3. Dark bluish to brownish pigmentation
4. With these atrophic hypopigmented lesions in between
5. Ochronosis – drug-induced pigmentation by hydroquinone
6. Inhibits homogentisic acid oxidase on the skin
7. Biopsy shows ochre bodies

- HIV positive patient started on ART
- Pigmentation of nails and skin
- Usage of Zidovudine is the cause



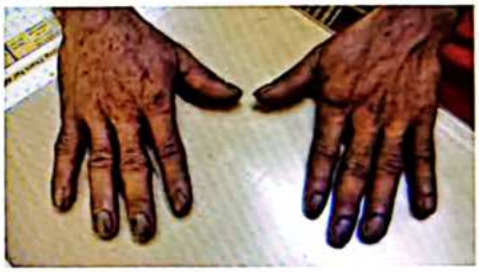
- A patient of Arthritis is given HCQS, he develops slate blue pigmentation of shins

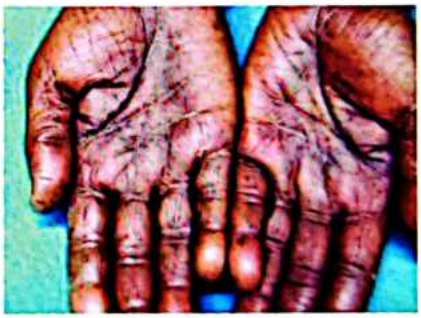
Question: what was the drug prescribed to a patient of Arthritis that he develops slate blue pigmentation of shins?

Answer: Bleomycin

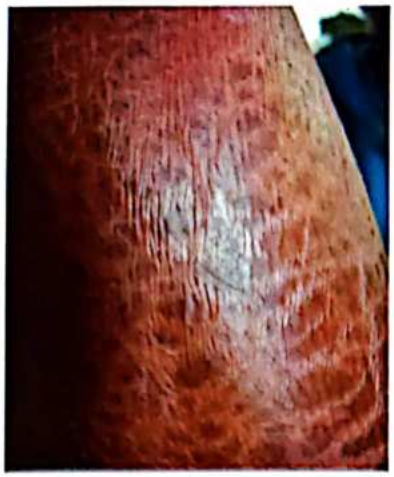
Develops a flagellate pigmentation that is characteristic of bleomycin

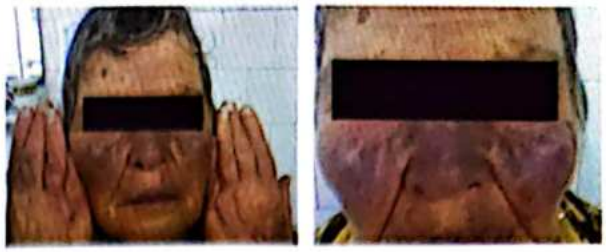
- Also causes flagellate erythema





- Clofazimine
- Causes ichthyosis
- Reddish discoloration
- Leprosy patient develops this lesion



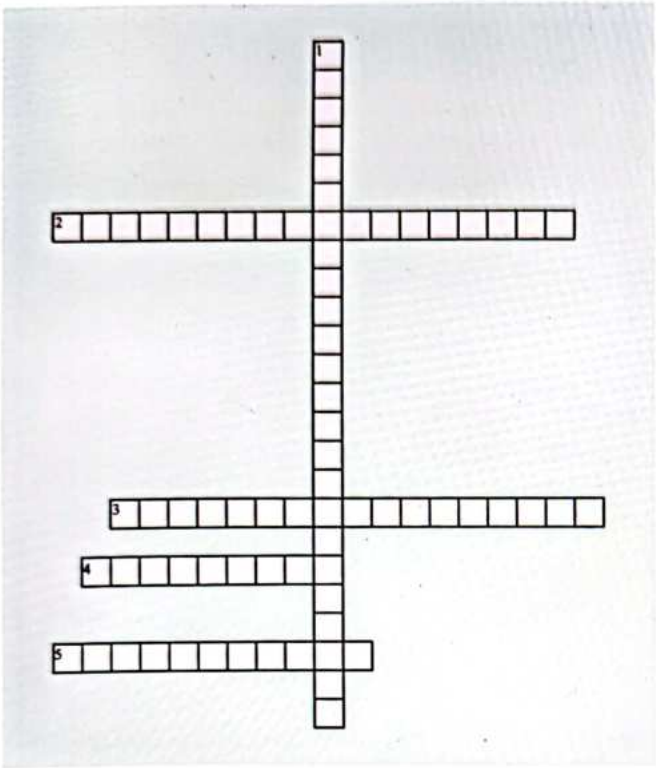


- Patient of ventricular erythema, started on drugs
- Develops pigmentation on photo exposed paths
- Amiodarone are the causative agents

prince
ankitkarnawat9@gmail.com
9818635293

CROSS WORD PUZZLES

Crossword Puzzle



Across

2. Acute Generalized Exanthem
3. Serum Sickness Rash
4. Stevens Johnson Syndrome
5. Toxic Epidermal Necrolysis

Down

1. Exfoliative Dermatitis

prince
ankitkarnawat9@gmail.com
9818635293

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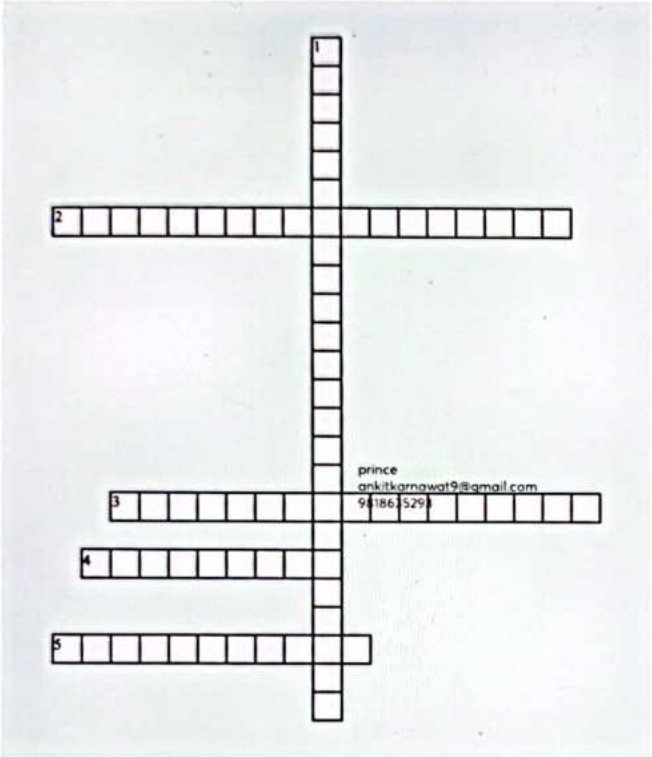


Across

2. Acute Generalized Exanthem
3. Serum Sickness Rash
4. Stevens Johnson Syndrome
5. Toxic Epidermal Necrolysis

Down

1. Exfoliative Dermatitis

23 PEDIATRIC DERMATOSES

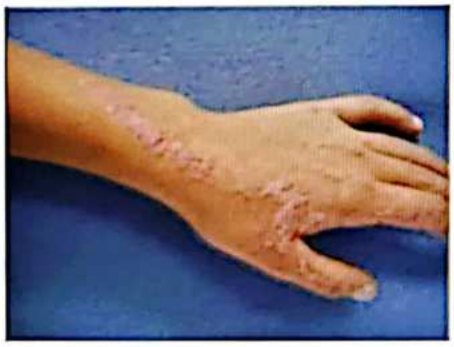
Nevi 00:00:12

- Birthmarks
- Due to genetic mosaicism
- Leads to formation of Blaschko's lines.

Verrucous Epidermal Nevi 00:00:35

- Common form
- Not only present during birth, the onset is within 2-3 months
  - Grows with age
- Hyper keratotic verrucous papules forming the plague.
- Follows Blaschko's lines.
- Increases in proportion to body growth
- Treatment
  - Laser
  - Electro frequency
  - Electrocautery.

Ilven 00:01:59

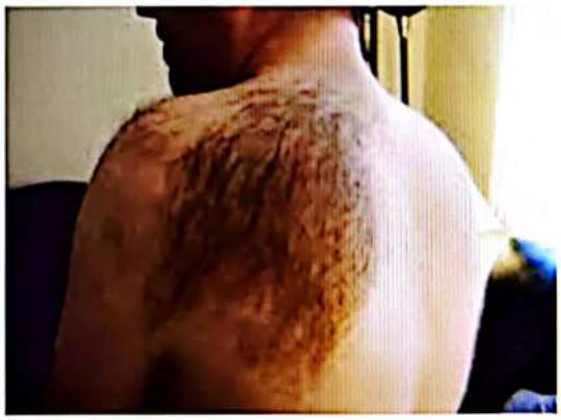


- Inflammatory linear verrucous epidermal nevi
  - It is eczematous
  - Erythema and itching.
- Papules are not hyperpigmented, they are in skin colour.
- Follows Blaschko's lines
- Start in 6 months of life
- Resistant to usual treatment regimen
- Treatment- topical steroids

Sebaceous Nevi 00:03:15

- Seen in scalp or face (more sebaceous glands)
- Accumulation of sebaceous glands.
- Clinical features-
  - Asymptomatic yellowish raised papules
  - Coalesce to form plagues
- <5% chances of developing neoplasm
  - BCC is a common malignancy.
- Certain benign tumours are present which form on them
  - Commonest one is syringocystadenoma papilliferum
- Treatment- When the child grows. remove it.

Becker's Nevus 00:04:48



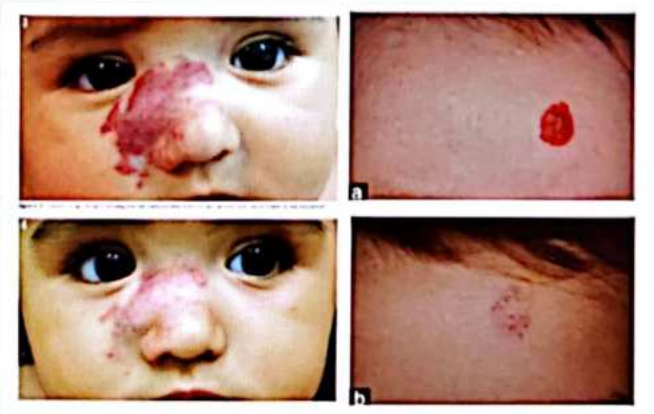
- Present at adolescence

- It forms as a hyperpigmented macule → patches→ hypertrichosis → plaque.
- Commonly seen on shoulders, chest
- They can be single or multiple.
- It also has hair growth over it.
- Also called as Becker's melanosis or pigmented hairy epidermal Nevus.
- There is role of androgens - More common in males
- Starts with pigmentary lesions and then forms hypertrichosis.
- No Treatment - Lasers are done but not that effective.

Vascular Nevi 00:06.51

- They can be classified into Hemangiomas (due to proliferation)
- Also called as vascular tumours
- There can be malformations
  - Capillary malformation
  - Venous malformation
- It can be proliferative or mal-formatted
- Vascular malformations are present at birth and Don't regress.
- Hemangiomas appear at 3 months of age and regress.
- Vascular Nevi are two types :

1. Infantile Haemangioma 00:08.10



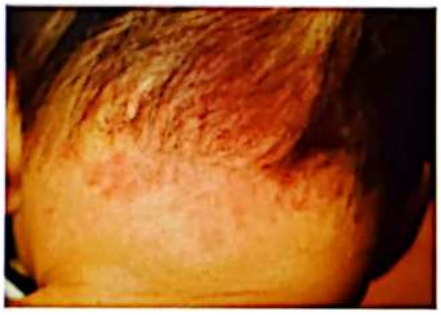
- Most common in childhood
- Starts at 2-3 months of age
- Have a course
  - Progress: By 9-10 months of age
  - Mature
  - Involute- They subside
- They can be superficial, deep or mixed.
- It grows and involute
- Leads to some amount of telangiectasias as remnant
- They start to lay, grow and involute.

- Hemangioma sitting on the nose,- Pinocchio nose or Cyrano nose
- Infantile Hemangioma sitting on the nose.

Treatment

- Indications for treatment- if hemangiomas are
  - Bleeding
  - Ulcerated
  - Present on vital organs like Pinocchio nose
  - Hampers breathing
  - Present in the mouth- hampers eating.
  - Present in the eyes- hampers seeing.
- In all the cases, systemic steroids and propranolol are used to treat.

Salmon Patch 00:10.24

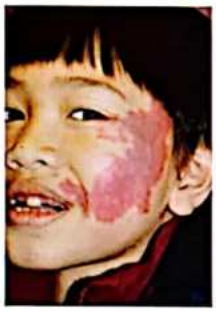


- Also called as stork bite, erythema nuchae
- Capillary malformation is present at birth and improves usually.
- Born child comes with a clinical features of
  - Little, superficial, reddish pink plaque
  - Salmon coloured irregular flat areas with telangiectasia.
  - Present of face, nape of the neck, upper eyelid, forehead and glabella areas.
- Face lesions improve by a year, neck lesions are persistent

- Shows autosomal inheritance dominance.

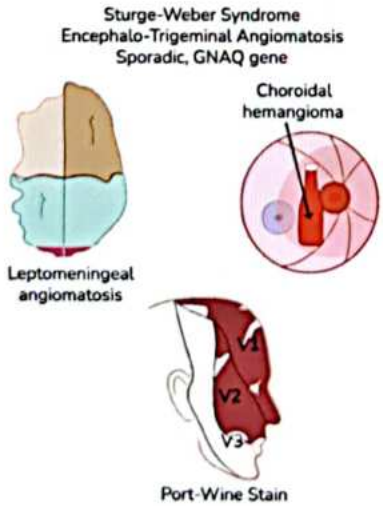
Port Wine Stain 00:11.18

- Also known as Nevus Flammeus
- Capillary malformations
- Present at birth and persists
- Do not resolute
- Seen in trunk and face
- Sharply unilateral distribution

- Present as erythematous macule which turns into plaque
  - Shows rugosities on surface
- With the proportion of growth of the child, it will progress and persist.
- It can be treated for cosmetic reasons.
- Treated with lasers
  - PDL laser (Pulse dye laser) is preferred.
- Port wine stains are associated with a few syndromes:

a) Sturge Weber Syndrome



- Port wine stain is present in the trigeminal distribution.
- It involves ipsilateral leptomeninges, microid
- A triad of port wine stain is present in the distribution of trigeminal nerve
- Ipsilateral leptomeningeal angiomatosis:
  - Convulsions
  - Behavioural problems
  - Subnormal intelligence
- **Ocular complications**
  - Ipsilateral glaucoma
  - Buphthalmos
  - Congestion
  - Strabismus and loss of vision

Q. A patient presents with a red patch on the face. Along with epilepsy and ocular complications

Ans: Sturge Weber syndrome is a triad of these three complications.

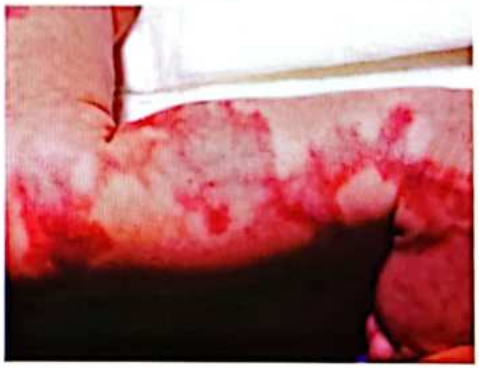
- Characteristic S- shaped intracranial calcifications in CT Scans and on X-rays.

b) Klippel-Trenaunay Syndrome (KTS)

- **Triad of**
  - → Port wine stain
  - → Venous and lymphatic malformation
  - → Bony and soft tissue hypertrophy of an extremity.
- Can be seen on leg showing hypertrophy with venous and lymphatic malformation.

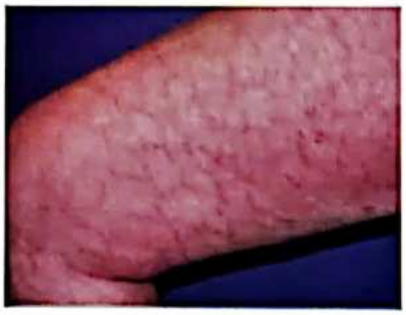
c) Cutis Marmorata Telangiectatica Congenita

- Mixed capillary and venous malformation.
- Most commonly involved in
  - → Trunk
  - → Face



- Due to mixed capillary and venous malformation.
  - A characteristic pattern is seen due to blood flow.
  - This network-like pattern is called Reticulate/ marbled appearance.
- This aggravates on cold exposure due to vasoconstriction
- This can be physiological and pathological
- Can be associated with underlying connective tissue disorder.

- If it is Persistent or leads to ulceration then think about connective tissue disorders.

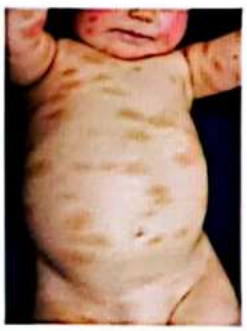


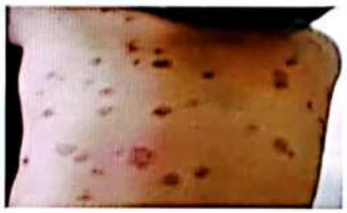
Mastocytosis 00:16.35

- Collection of mast cells
- When it is present in the skin- called Cutaneous Mastocytosis.
  - Mast cells aggregated in the skin.
- By biopsy, mast cells can be seen.
  - By using toluidine blue or Giemsa stain.
- **Types of Cutaneous Mastocytosis**
  a. Urticaria pigmentosa (it is not urticaria)
  b. Solitary Mastocytosis
  c. Diffuse cutaneous Mastocytosis
  d. Telangiectasia macularis eruptiva Perstans.

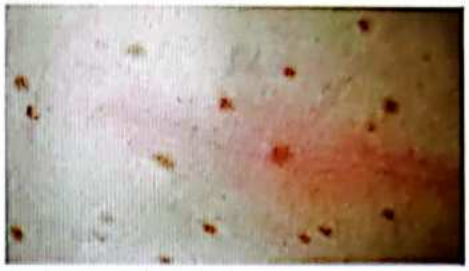
Urticaria Pigmentosa 00:17.14

- Common form of Mastocytosis
- No systemic involvement
- Only skin is involved
- **Onset:** before 2 years
- **Clinical features**
  - Urticarial lesions heal with hyperpigmentation
  - Stroking of lesions- develops Urticarial lesions.
- Due to the presence of mast cells, little trauma or stroking leads to development of Urticarial lesions.





- Hyperpigmented plaques present on trunk
  - Seen in <2yrs old
- When you stroke with a pen or key- erythematous wheals are seen- Darier sign



- Skin biopsy-
  - Mast cell is identified
  - Anaesthesia must be given to surroundings.
  - Avoid trigger factors which lead to mast cell degranulation.
- Mast cell stabilisers like montelukast, ketotifen are used.

Histiocytosis 00:20.19

- Includes disorders resulting from a proliferation of cells of the monocyte- macrophage lineage.
- **Class-1:** Langerhans cell histiocytosis.
  - Proliferation of Langerhans cells
  - These are antigen presenting cells.
- If they're formed in more numbers, it causes Langerhans cell histiocytosis.

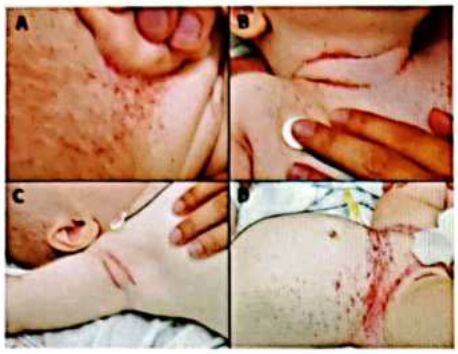
Langerhans Cell Histiocytosis

- Positive for S100 and CD1a
  - Which contains Birbeck granules
- Multi organ involvement
  - Bone, skin
  - Lymph nodes, lungs
  - Liver and spleen
  - Endocrine glands and nervous system.
- 3 types of LCH

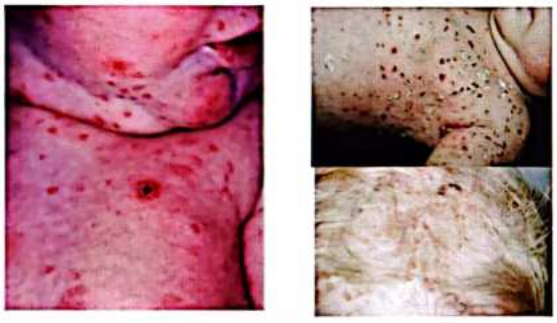
1. **Eosinophilic granuloma**
   - Seen in older children or adults.
   - Lesions are only confined to bones
2. **Hand-Schuller-Christian disease**
   - Seen in 2-6 years
   - Classical triad of skull defects, diabetes insipidus and exophthalmos
3. **Letterer-Siwe disease**
   - Seen in 0-2 years
   - Visceral and skin involvement

- All these three have Birbeck granules and rod shaped cytoplasmic cells.
- These are classified based on age and clinical presentation.
- **Age of presentation**: 1-3 years
- **Symptoms**: fever, failure to thrive

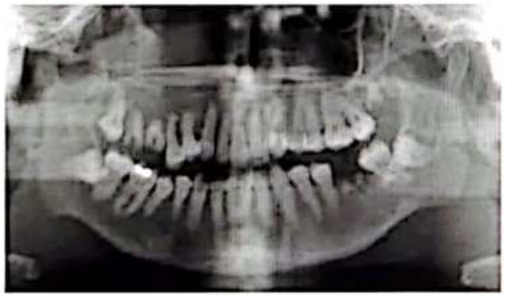
- **Cutaneous features**: Seborrheic dermatitis- papules, vesicles, pustules which may crust and ulcerate.



- Seborrheic dermatitis present of flexus, underarms
- Child has non healing cradle cap



- **X-ray**- Floating tooth
  - Lower molar areas- the alveolar bone is lost

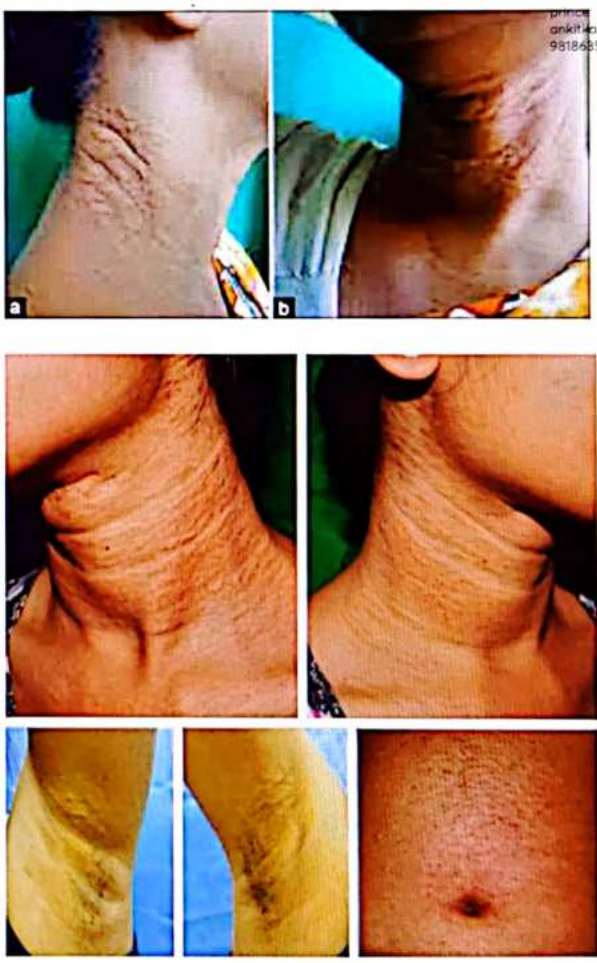


- **Visceral involvement**
  - Most commonly skull followed by long bones
  - Lymphadenopathy
  - Splenomegaly
  - Jaundice
- Three markers are expressed by LCH cells
  - Peanut agglutinin
  - An epitope shared with IFN-g
  - Placental alkaline phosphatase
- Treatment
  - Limited involvement of the skin is left untreated
  - **Extensive and symptomatic cutaneous changes:** Prednisolone therapy
  - **Recalcitrant ulcerated plaques:** PUVA Therapy and topical nitrogen beam therapy
  - **Chemo and radiation therapy:** Systemic disease
  - Vinblastine and corticosteroids are the current standards for treatment of multisystem disease in children.

Pseudoxanthoma Elasticum

00:25:25

- Genetic metabolic disease
- Autosomal recessive inheritance
- Mutations in ABCC6 gene- leads to ectopic mineralisation
- Involves elastic tissue of the skin, eyes and blood vessels.



- Small yellow papules on nape and sides of neck and in flexural
- Papules coalesce and the skin becomes loose and wrinkled.
- Eyes: Angioid streaks
  - Due to dystrophic calcification of Bruch's membrane
- Choroidal neovascularization
  - Due to elastic tissue involvement

- Arteries also involved and leads to cardiac complications
- Loss of central vision and blindness
- Lesions in small and medium sized artery walls may result in intermittent claudication peripheral artery disease.

Palpable Purpura 00:26.50

- 2 types of Purpura
  - Extravasation of blood cells
    → Non palpable
  - Palpable purpura

Palpable purpura

- Present in
  - Vasculitis
  - LCV, HSP, CTD, cryoglobulinemia, Hep C
  - PAN
  - Disseminated Gonococcal infection
  - Acute meningococcemia
  - Rocky Mountain spotted fever
  - Ecthyma gangrenosum

Non-Palpable Purpura

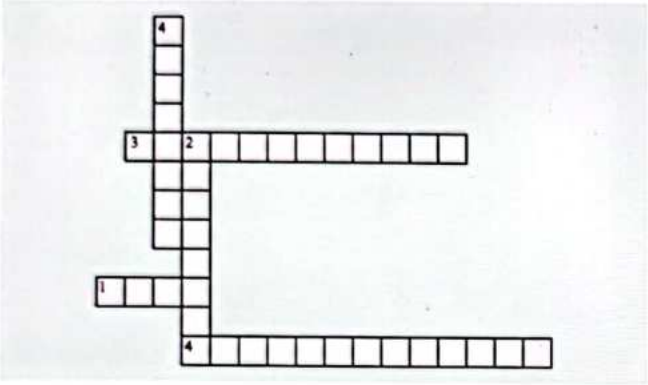
- ITP
- Platelet dysfunction
- DIC
- Fat or cholesterol emboli

Pinch Purpura

- Due to increased capillary fragility
- Seen in- Primary systemic amyloidosis, EDS, scurvy.

CROSS WORD PUZZLES

Crossword Puzzle



Across

1. Caused Due to genetic mosaicism
3. Collection of mast cells is aka
4. 2 types of purpura

Down

2. Salmon Patch is aka
4. 2 types of purpura

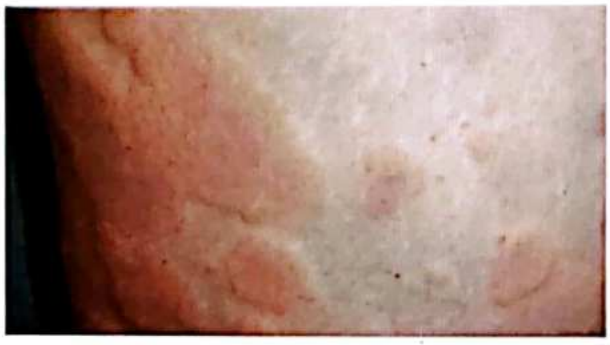
prince
ankitkarnawat9@gmail.com
9818635293

24 URTICARIA AND ANGIOEDEMA

- Wheals is the characteristic lesion seen in urticaria.
- In urticaria, vasodilation is present
  - It causes redness of skin
  - Increased vascular permeability leads to edema.
  - This is caused by the release of histamine.
  - Mast cells stimulate histamines.
  - Mast cells cause degradation and lead to release of histamines.
  - Mast cells are stimulated by IgE by binding to the allergen.
- Many times, mast cell stimulation can be Non IgE dependent
  - The above pathway also may not be present.
- Urticaria is mast cell dependent
  - Whether it may be IgE dependent or Non IgE dependent
- Mast cell-independent (bradykinin dependant)
- Mast cell-dependent is more common.
- IgE + allergen
  - It acts on nerve endings and causes axon reflex and pruritus.
- This is responsible for Lewis's triple response of erythema, flare, and wheal.

Etiology

- In 60% of the cases- unknown cause or idiopathic
- Allergic
- Mnemonic: All I's
  - **Ingestion:** common cause
    → Food is the cause
  - **Inhalation**
    → Pollens, air borne are responsible
  - **Infection**
    → Many infections can cause this
  - **Injection**
    → Many drugs can cause it
  - **Insects**
    → Insect bites can cause urticaria.



- It is typically present with wheals
- Wheals are erythematous(red), edematous(swollen), evanescent(transient), itchy lesions.
- Transient means they come and go in 15mins to 2hrs.

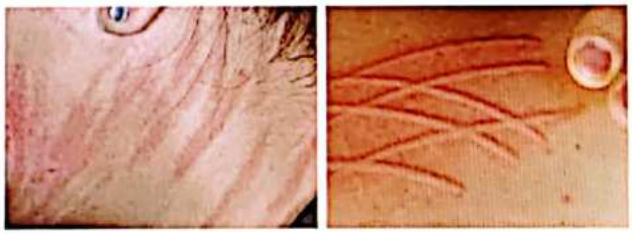
Urticaria is classified into two types based on the duration.

- Acute urticaria
- Chronic urticaria

Acute Urticaria	Chronic Urticaria
• Duration is less than 6 weeks.	• Duration is more than 6 weeks.
• In all cases, all **5 I's** are included. o Infections (40%)- viral infections o Drugs (9%)- penicillin, sulfa o Food items (1%)- additives o Inhalation- pollen o Idiopathic (50%)	• It can be chronic spontaneous urticaria driven by autoimmune system (65%) physical urticarias • (35%) • Urticarial vasculitis (5%).

Physical Urticaria

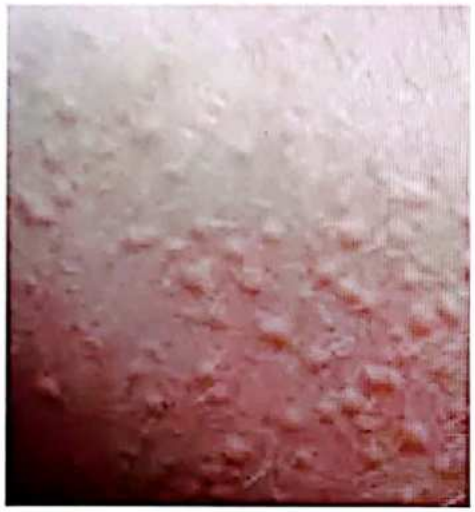
Dermographism



- The most common physical urticaria
  - Also called as dermatographism or dermographic urticaria.
- Derma: skin; graphism: writing
  - It means writing on skin.
- Caused by physical force.
  - Like sun, water, heat, etc.
- Linear streaks of urticaria on lines of stroking.
  - Whatever you write, it gets imprinted.
- As per the strokings, you get linear wheals of urticaria.

Cholinergic Urticaria

- The second form of physical urticaria



- It happens due to increased sweating
  - Patient goes to exercise, gym, etc. and gets urticaria.
  - Increase in core body temperature.
  - Sweat glands are stimulated by post ganglionic Cholinergic nerves.
    → They get stimulated.
- Small pinpoint urticarial papules are seen.
- Each type of urticaria develops in a few minutes and lasts for a few hours.

Delayed Pressure Urticaria

- It happens after 3-4 hours
  - Stays for 24 hours
- If you hold the hand of a patient tightly
  - Then he develops urticaria in that pressurised place.
- If the patient wears a watch
  - He gets the urticaria at that site.
- If he is sitting on a chair
  - Urticaria develops on the buttocks.
- In this case, pressure leads to urticaria.

Different types of Physical Urticaria

Type of Physical Urticaria	Eliciting Factors
Urticaria factitial/ dermographism (Most common type, red, itchy, linear wheal appearing immediately after light stroking of the skin.)	Mechanical shearing forces
Cold contact urticaria	Cold objects, air, fluids, wind
Heat contact urticaria	Localised heat
Solar urticaria	UV and / or visible light
Delayed pressure urticaria	Vertical pressure
Aquagenic	Water
Cholinergic	Increase in core body temperature by exercise or spicy food

Aquagenic Pruritus

- On contact with water, just itching will be present.
  - Wheals are absent
- This is seen in polycythemia Vera.

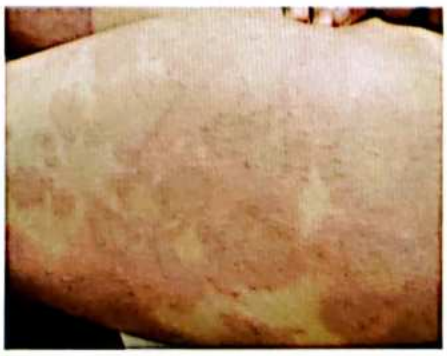
Management

- Avoid the trigger
- Antihistamines are used
- In the first 2 weeks, modern 2nd generation H1 antihistamines with standard dosages.
  - If the patient does not respond, increase the same to four folds.
- Even if they did not respond, add
  - Montelukast, cyclosporine
- **Biological agents** used in Urticaria are Omalizumab.

Urticarial Vasculitis

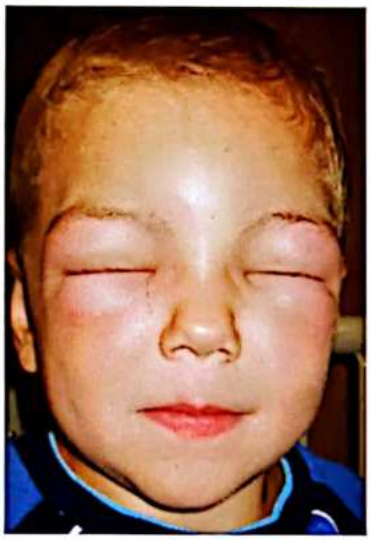
00:12:08

- A type of chronic urticaria
- Patient will have Urticarial wheals
  - But after biopsy, leukocytoclastic vasculitis.
  - These are little different from urticaria.
  - These lesions will persist for 24-48 hours.
  - They are less itchy and painful.
  - They heal with pigmentation.
- This is associated with connective tissue diseases, haematological malignancy.



Angioedema

00:13:58



- When a patient gets bitten by wasp or bee bite
  - Eyes, lips get swollen
  - This is angioedema
- This is much deeper.
- Less well defined that urticaria
- Painful rather than itchy
- Less erythematous
- Basically, it affects mucosa, submucosa, and hypodermis.
- Takes longer than 24 hours to resolve.

Types

- **With wheals**
  - Just treat it like urticaria
  - Mast cell dependent
- **Without wheals**
  - Role of bradykinin
  - Not mast cell-mediated
- angioedema without wheals is furtherly divided into two types.
  - **Hereditary**
    → C1 esterase inhibitor deficiency or impaired function
    → Due to genetic effect
  - **Acquired**
    → It may be drug induced (ACE inhibitors)
    → Idiopathic
- Kininogen gets converted to bradykinin
  - Bradykinin causes vasodilation and increases vascular permeability.
- C1 esterase inhibitor inhibits this conversion.
- If it is deficient, when a patient has acute episode of angioedema or an attack
  - Kininogen gets converted to bradykinin and then gets inhibited.
- If it does not get inhibited, then there is increased bradykinin formation
  - Leads to all these manifestations.

Role of ACE inhibitors:

- ACE inhibitors inhibits angiotensin converting enzyme
  - Converts bradykinin into inactive metabolites.
- Bradykinin does not get converted to inactive metabolites.
  - Gets angioedema.
- Complement cascade gets activated.
- Then deficiency of C4 and C2 occurs.
- Low C4 levels are used as screening tests for angioedema.

Hereditary Angioedema

- Also called as Quincke's disease.
- It has autosomal dominant inheritance.
- Bradykinin mediated.
- Hereditary angioedema can be of 3 types depending on C1 INH protein
- **C1 INH**
  - Can be low
  - Normal but function is low
  - Normal, function is low, still we get in
- Hereditary angioedema is 3 types
  - Type 1
  - Type 2
  - Type 3

Forms of Hereditary Angioedema

- C1 inhibitor deficiency
- Deficiency is seen in levels or functioning.
  - If levels are less - **Type 1.**
  - If levels are normal and functioning is low - **Type 2**
  - If everything is normal, but other mutation defects like factor 12 & 13 - **Type 3**
- Since it is autosomal and hereditary,
  - Family history is positive.
- **Other symptoms**
  - Recurrent laryngeal edema
  - Colicky abdominal pain
  - Recurrent angioedema

Screening tests

- **C4 levels**
  - During an acute attack, C4 levels go low
- **C1 inhibitor level**
  - It is not easily available
  - If function is low, it is type 2
  - If levels are low, then it is type 1.
- In type 3 HAE, the C4 levels are normal.

Treatment

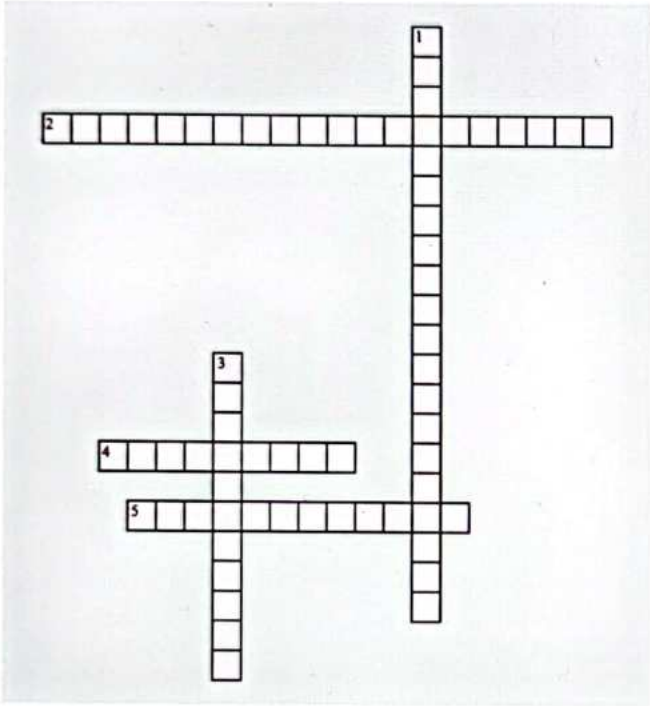
- If a patient has acute episodes,
  - Then he has laryngeal edema
- Airway management must be done
- Purified plasma derived C1INH
- Icatibant - bradykinin receptor 2 antagonist
- Ecallantide - a kallikrein inhibitor
- Fresh frozen plasma, containing C1INH.

Prophylaxis

- Can be given in recurrent angioedema
- Plasma derived C1INH replacement therapy
- Tranexamic acid
- Anabolic steroids like danazol and stanozolol

CROSS WORD PUZZLES

Crossword Puzzle



Across

2. Quincke'sdisease
4. Mast cell dependent
5. Increase in core body temperature by exercise or spicy food results in

Down

1. Associated with connective tissue diseases, haematological malignancy
3. Used as screening tests for angioedema

25 GENODERMATOSES

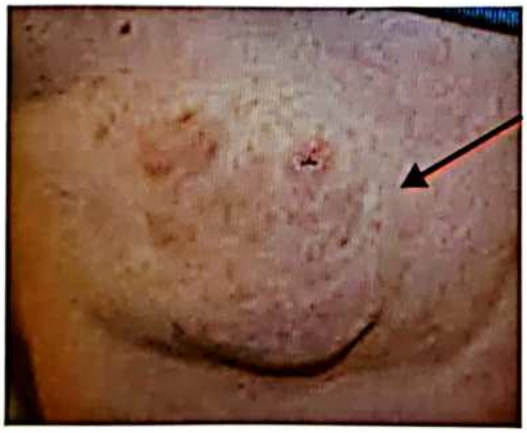
- Genetic disorders of the skin are called Genodermatoses.

Genodermatoses

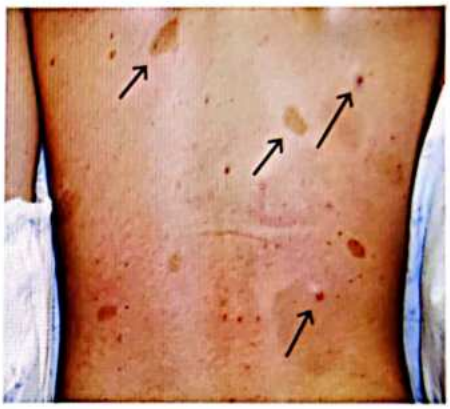
00:00:10

Neurofibromatosis 1

- Also known as Von reckling hausen's disease
- Inheritance: autosomal dominance
- Gene: NF1 - chromosome 17
- Protein: neurofibromin



- **Neurofibromas**
  - They are soft pedunculated growth in the skin.
- **Buttonhole sign**
  - If pricked, the button will go in.
- **Bag of worms**
  - It feels like a plexiform with lots of nerves.
- For it to be in the category of NF1, there should be more than or equal to 2 neurofibromas.
- Or the individual can have one plexiform neurofibroma along the nerve.



- CALM's– cafe au lait macules (coffee on milk) due to their coffee like color.
- They are hyperpigmented and asymptomatic lesions.
- More than 6 CALMs
- To fall under the category of NF1, they should be more than
  - 5 mm in prepubertal
  - 15 mm in post-pubertal

CALMs are associated with the following:

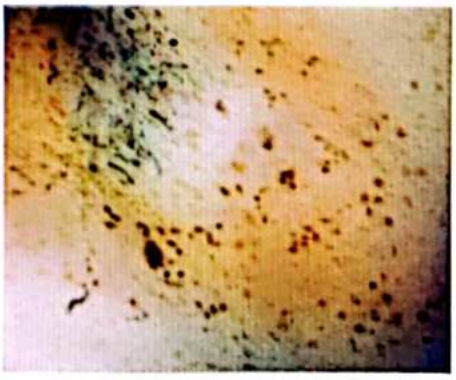
- Neurofibromatosis (NF1, Nf2)
- McCune - Albright Syndrome
- Tuberous sclerosis
- Fanconi anemia
- Bloom Syndrome
- Silver-Russell Syndrome

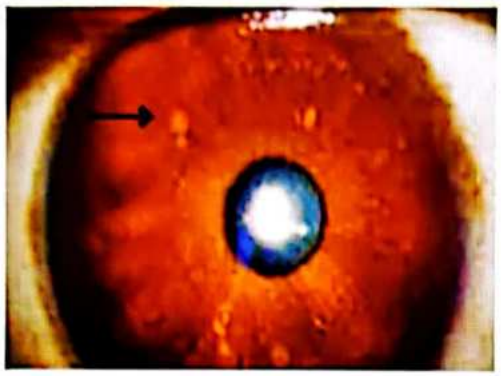
Important Information

- Calm was mentioned as a feature of NF, and it is also seen in other disorders.

Axillary and inguinal freckling

00:05:00





- Usually, you see freckles on the face, but if NF, freckles can be seen in the axillary and inguinal areas.
- A specific feature in NF that needs no number or size
- This freckle is called Crowe's sign
- In the eyes, one can see Lisch nodules (Iris hematoma—these are not in the line of vision) - **seen with a slit lamp**
- Skeletal features: sphenoid dysplasia, pseudoarthrosis, kyphoscoliosis
- CNS features: epilepsy, low intelligence
- Malignancies: gliomas, astrocytoma

A diagnosis of NI can be made in an individual with 2 or more of the following

>= 6 cafe au lait macules (> 5 mm in greatest dimensions for prepubertal and > 15 mm in post pubertal persons)
Axillary and inguinal freckles
>= 2 neurofibromas (any types) or plexiform 1 neurofibromas optic glioma
>= 2 lisch nodules
Sphenoid dysplasia, tibial pseudarthrosis, or other distinctive bone lesion
First degree relative with a diagnosis of NFI

Abbreviation: NF1, neurofibromatosis type 1

Remember it with

C- Cafe-au-lait-spots (greater than six)
A- Axillary or inguinal freckling
F- Fibromas or plexiform neurofibroma
E- Eye hamartomas
S- Skeletal abnormalities e.g., leg bowling, sphenoid wing dysplasia

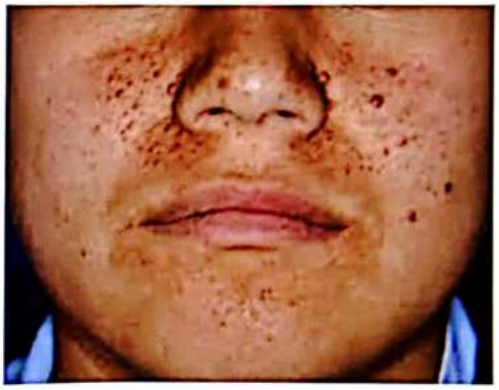
Treatment:

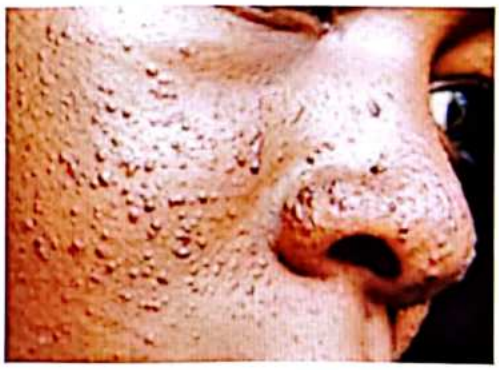
- Symptomatic with no cure
- Pirfenidone, a farnesyl transferase inhibitor used as an antibiotic agent, down regulates the RAS oncogene PEG

Tuberous Sclerosis Complex 00:08:30

- Also known as Bourneville's Disease, Epiloia
- Inheritance: autosomal dominant inheritance
- Chromosome: 9 & 16
- Gene: TSC 1 and 2
- Protein: tuberin and hamartin
- Growth
- Patients have epilepsy, low intelligence, and adenoma sebaceous - EPILOIA

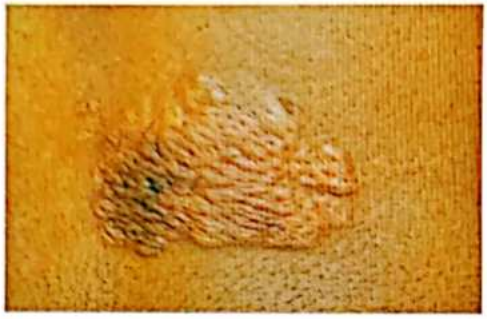
Adenoma sebaceum 00:09:40

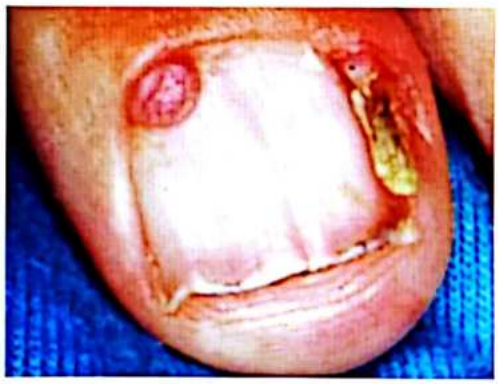




- It is a misnomer.
- Nothing to do with sebaceous glands.
- Why the name? Because it is present in sebaceous glands.
- It is a cutaneous angiofibroma, growth of blood vessels and fiber.
- Arrhythmia soft papules increased during puberty– seen in butterfly distribution.

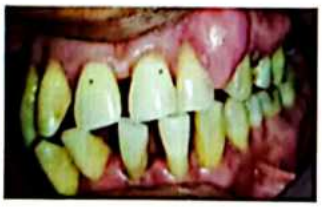
Other features are:

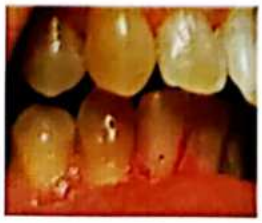




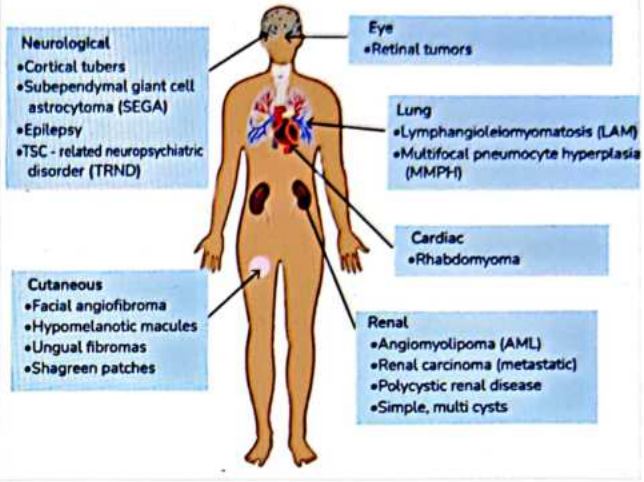
Koenen's tumours

- Ash leaf macules
- First thing to see – manifestation
- Wood's lamp– accentuated in the wood's lamp
- Hypopigmented leaf shaped macules on trunk.
- Presence of shagreen patch which is a collagenoma that is seen on the trunk.
- Koenen's tumors are the periungual fibromas.





- Gingival fibroma
- Dental pits
- CALM
- Hypopigmented to depigmented which are called confetti like macules– CLM



Hamartomas:

- Hamartoma
- Adenoma sebaceum
- Mental retardation
- Ash leaf spots
- Rhabdomyoma
- Tubers
- Optic hamartomas (phakomas)
- Mitral regurgitation
- Astrocytoma
- Seizures

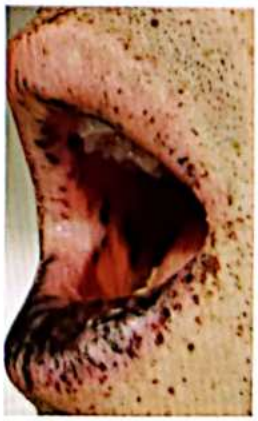
Treatment:

- Rapamycin (also called sirolimus)-an immunosuppressant- can induce regression of brain astrocytoma associated with TSC

GI Polyposis Syndrome 00:15:30

- Peutz- Jeghers Hamartomatous
- Cowden - Hamartomatous
- Gardner- Adenomatous Polyps

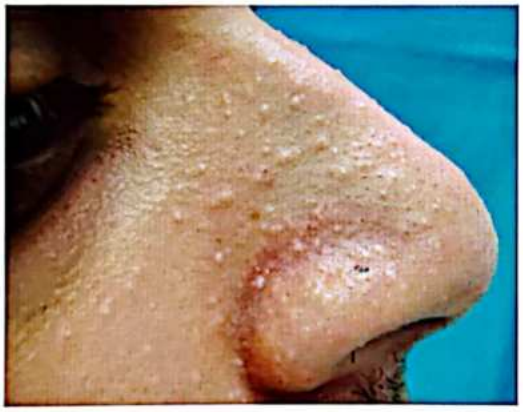
Peutz- Jeghers Hamartoma



- Important question- polyps do noT develop early in life
- Regular checkup is done- endoscopic examination
- Early in life- patients comes with periorificial lentiginosis

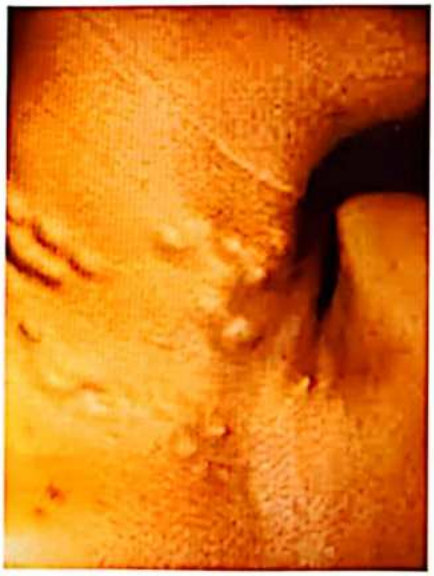
- Lentigines-hyperpigmented macules present on lips, around mouth, oral mucosa
- It might lead to GI cancer
- Autosomal dominant condition – family history
- Like the history of gastric cancer or lentiginosis.
- Patients need to come for regular checkups to rule out any symptoms of GI-polyposis.

Cowden Syndrome



- Trichilemmoma – shiny minute papules present on the nose

Gardner Syndrome



- APC Gene
- Epidermoid Cyst

Important Information

- Peutz-Jeghers: Characterized by multiple hamartomatous polyps in GI tract.

DNA Repair Defect

00:19:00

- XERODERMA PIGMENTOSUM– dry skin and pigmented skin
- Photosensitivity, oculocutaneous pigmentation and early neoplasia
- Inheritance: autosomal recessive condition
- **Defect:** nucleotide excision repair defect
  - Because of this, individual DNA is damaged and accumulates in body leading to photosensitivity and early neoplasia

Features: Xerotic dry

- Normal at birth
- Photosensitivity
- Persistent erythema
- Acute sunburn
- Xerosis
- Diffuse freckling
- Telangiectasis
- Atrophic lesions
  - Stay in dark room
  - Photosensitive children who are unable to open eyes if presented in sunlight
  - Lead to DNA defect- normal at birth but gradually the damage increases
  - Extreme photophobia

Malignancy:

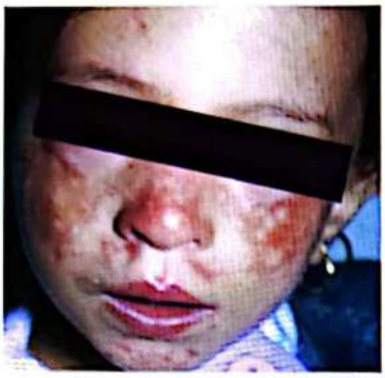
- **Skin malignancy:** Benign, keratoacanthoma, AK, BCC, SCC, malignant melanoma
- **Systemic malignancies:** Solid organ malignancies– medulloblastoma, astrocytoma, mental retardation and low intelligence

Treatment:

No cure available as this is a genetically inherited disorder

- Complete sun protection
- Genetic counseling
- Malignancy screening
- Oral isotretinoin
- Topical application of liposomal lotion of the microbial enzyme 14 endonuclease v

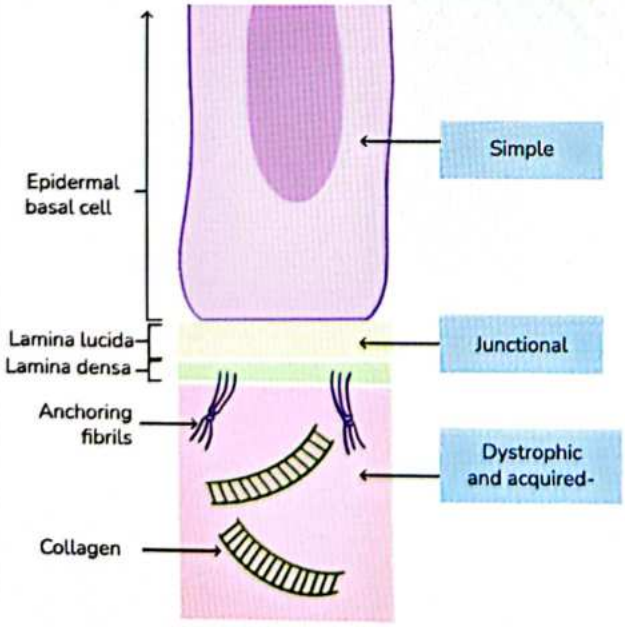
Bloom Syndrome (congenital telangiectatic erythema and stunted growth)



- Autosomal recessive
- Growth retardation
- On the face dilated blood vessels with redness and stunted growth
- Photosensitivity and early malignancy

Epidermolysis Bullosa

- Also known as mechano bullous disorders-mechanical trauma and gene defects
- **Types:**
  - **Congenital**
    - → EB simplex
    - → EB junctionalis
    - → EB distrophicans
  - **Acquired – EBA-** defects of **collagen 7**

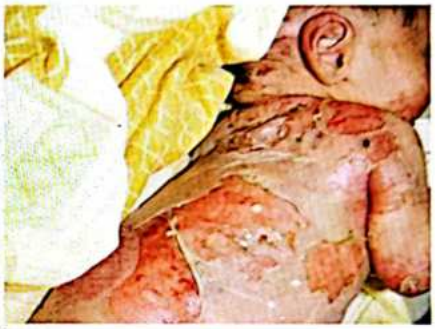


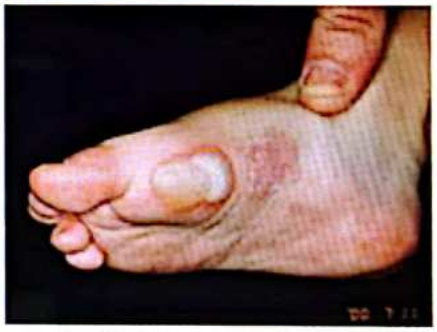
Levels of blister formation in epidermolysis bullosa at the demo - epidermal junction.

- **Three types of defects:**

	Ebs	Ebj	Ebd
defect	Krt 5 And 14	Laminin 5	Collagen Vii
split	Intra epidermal split	Sub epidermal bulla	Sub epidermal bulla
Inheritance	Ad	Ar	Ad/ar

- Usually, genetic defects - the child is born at an early stage of bulla which is formed on trauma prone site





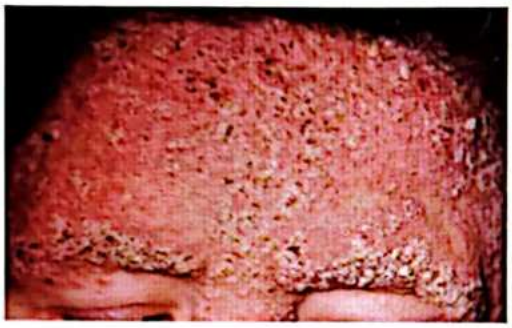
- Genetic disorder - positive family history
- Family member might say wherever they hold the baby- skin of the baby split, means trauma from touch or pressure
- Can be seen on trunk in baby
- In a grown kid - it can be seen on foot— site of trauma – may or may not have mucosa
- **EB junctionalis and EB distrophicans have mire severe lesions**
  - Mucosal lesions
  - Severe manifestations
  - Dental abnormalities
  - Periungual lesion
- **Diagnosis:**
  - No role of DIF
- **Skin biopsy:**
  - Split
- **EM- diagnostic:**
  - Electron microscopy
- **Treatment:**
  - **Gene** therapy/SYMPTOMATIC TREATMENT

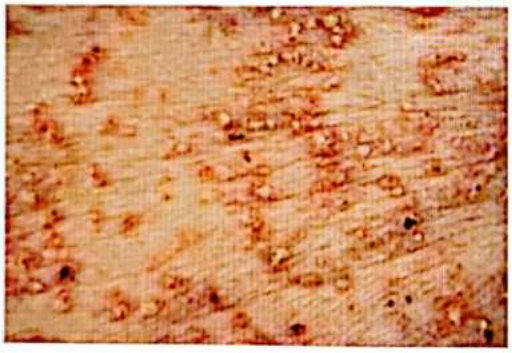
Inherited Acantholytic Disorder

- **Hailey Hailey**
- **Darier's disease**

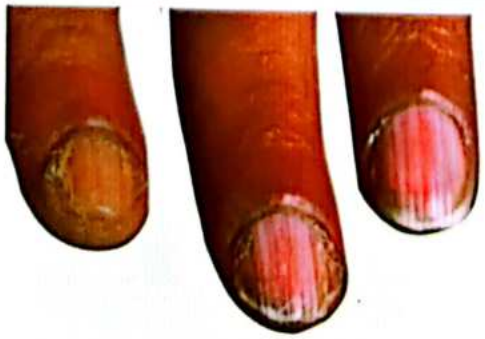
Darier's disease

- Inheritance: autosomal dominant inheritance
- Defect: ATP2A2 gene defect– responsible for circa -2 type calcium bound
- Onset: mostly in childhood
- Season: summer and monsoon

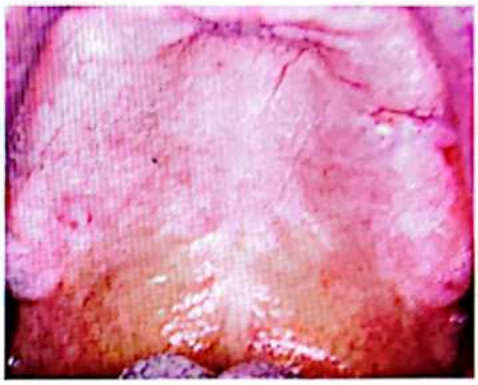




- Warty dirty papule – rough hyperkeratotic associated with discomfort itchiness
- Seborrheic areas
- Covered with dirty looking crust
- No bulla or vesicles is seen
- **Features:**
  - On the nails-longitudinal lines called erythro leukonychia– alternate longitudinal white and red bands



  - Distal notching – v sign on nails
  - Cobblestoning of palate is seen

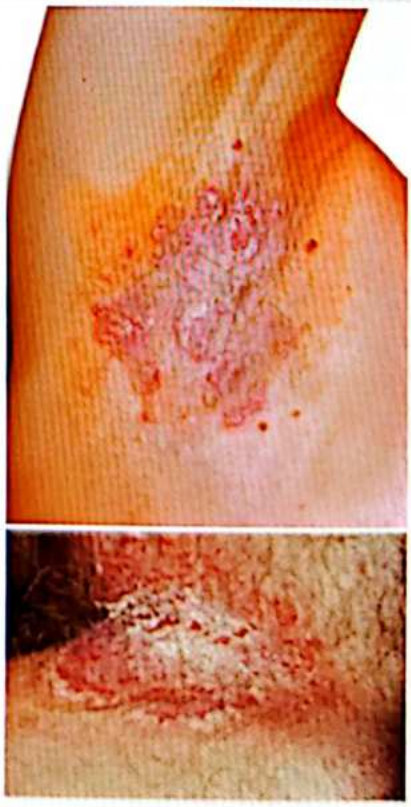


  - Histopathology: suprabasal acantholysis
  - Dyskeratosis - disordered keratinization
    → Leads to formation of dyskeratosis cells known as CORPS RONDS

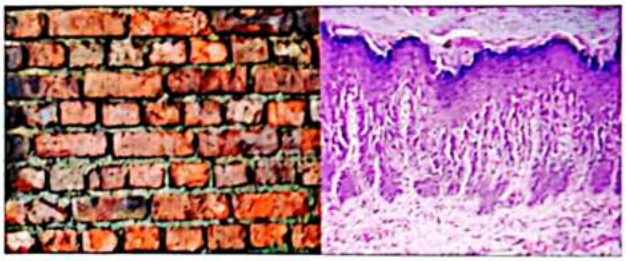
Hailey Hailey

- Also known as familial benign chronic pemphigus – acantholysis disorder not related pemphigus
- Inheritance: autosomal dominant inheritance
- Defect: mutation in the ATP2C1 gene

Features:



- Recurrent vesicular eruption
- Intertriginous areas
- Maceration
- Exacerbations and remissions
- Chronic causes
- No mucosal involvement
- Since its genetically inherited – it will come and go



Biopsy:
- supra basal acantholysis which is partial
- Dilapidated brick wall appearance

Clinical picture:
- Darier's and hailey hailey - both are inherited disorders can be easily differentiated
- Pemphigus vegetans - vegetating lesions with no family history and it was a variant of pemphigus vulgaris therefore mucosal involvement is present
- DIF- positive

Tinea: pruritus KOH+
No chronic course of disease

Ichthyosis

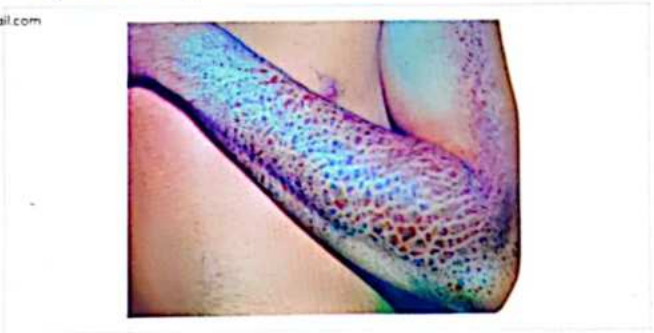
00:38:00

- Scaly dry and rough skin associated with persistent scaling
- Disorder where Defect is in keratinization which is very important for formation of skin ,if its altered skin will get dry and rough

TYPES (exam question)	
Congenital	**Acquired**
Ichthyosis vulgaris	Infections (leprosy), HIV
X- Linked Ichthyosis	Drugs (Clofazimine)
Lamellar Ichthyosis	Malignancies
	nutritional
	hypothyroidism
	Systemic diseases (sarcoidosis)

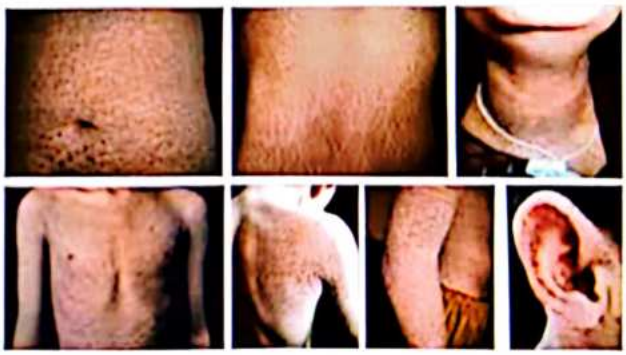
Important Information
- What are the causes of acquired ichthyosis?

Congenital Ichthyosis



Ichthyosis vulgaris
- Defect is in filaggrin gene, skin barrier will be altered causing dry scaly skin which are on extensors
- Gray fine scales upturned at edge
- Very dry skin associated with atopy there is hyperlinear palms and soles
- Present after 3 months of age
- Vulgaris is common
- It increases in winters

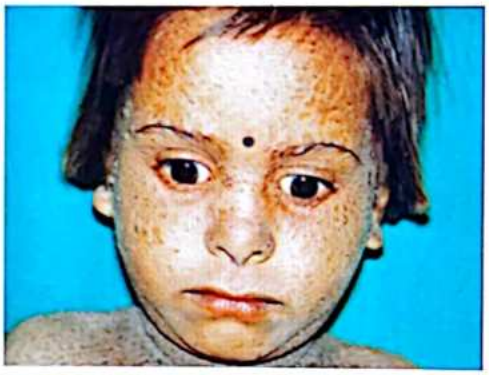
X- Linked recessive Ichthyosis

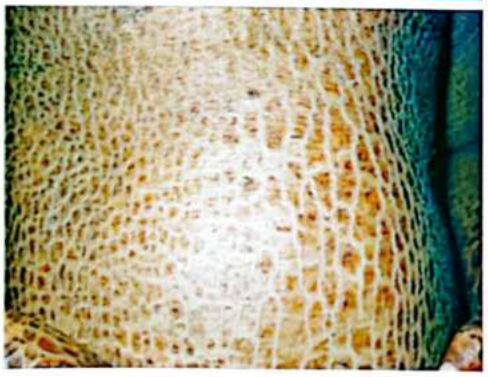


- Scales are dirtier looking
- Present in the preauricular area and
- Present on the flexors more than the extensors
- X-linked recessive ichthyosis
- Defect is in the STS- steroid sulfatase gene
- Defective gene leads to accumulated cholesterol which gives rise to such scales
- Palms and soles spared

Lamellar Ichthyosis

- Defect in transglutaminase
- Born as collodion baby
- Baby covered in Taut and shiny glistening membrane
- ectropion, eclabium- eyes and averting of mouth
- Patients are born with erythroderma having scales on lower legs
- Fish like pasted scales





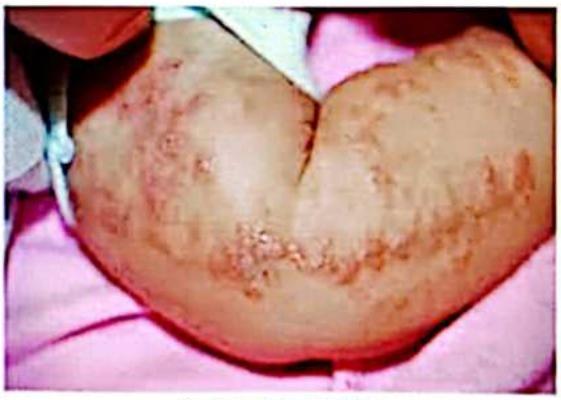
	IV	XLRI	LI
Inheritance	AD	XLR (males manifests)	AR
Defect	filaggrin	Steroid sulfatase	transglutaminase
Onset	After 3 months	Not at birth	At birth
Type Of Scales	Fine gray, upturned at edges	Dirty looking adherent	fish/plate like scales
Area Affected	Extensors, winter exacerbation	Flexural, pre auricular Palms and soles spared	Erythroderma Lower legs
Associations As	Atopy, KP, PP hyper linearity	Comma shaped corneal opacities, cryptorchidism	Collodion baby, ectropion, eclabion
Treatment	Keratolytics No retinoids	Keratolytics, retinoids, liarozole	Keratolytics Retinoids

Harlequin ichthyosis

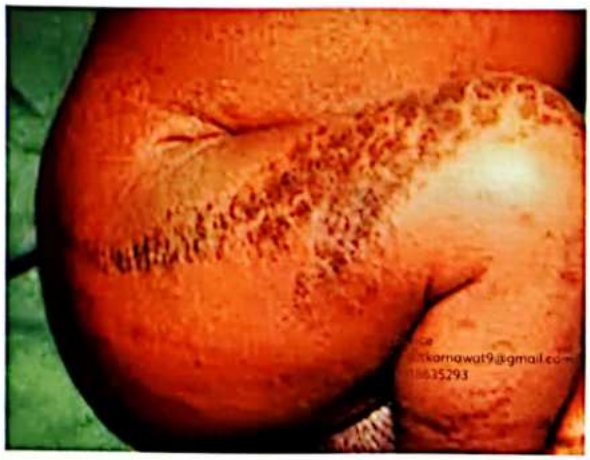
- Child is born in armor appearance
- Zero survival

Incontinentia Pigmenti

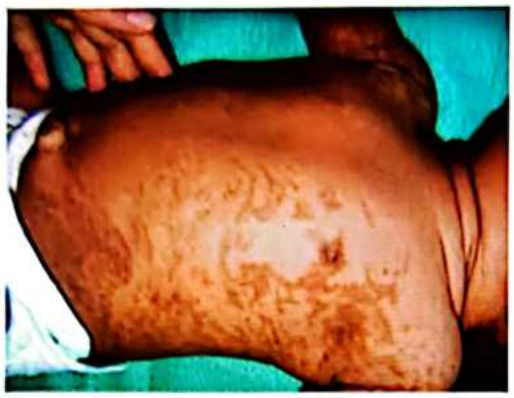
- Also called as Bloch- Sulzberger syndrome
- Inheritance: X-linked dominant
- Males will die in utero
- Females will present with this defect
- Gene: NEMO gene is defected.



0- 2 weeks persists



2 weeks-6 weeks

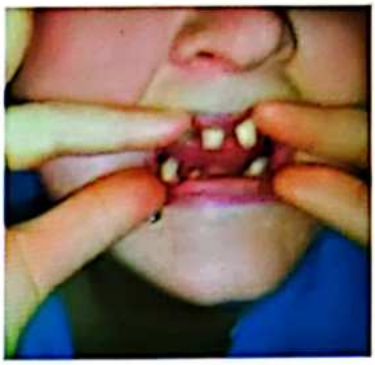


12-16 weeks

It is in three phases: defects along the dermatome/blaschko's line

- **Vascular**– 0-2 weeks persists for 2 months– in utero
- **Verrucous– 2 weeks - 6 weeks**
- **Pigmentation** – hyperpigmented macules along Blaschko's line— this is most commonly presents phase in a child
- Always a female will be affected

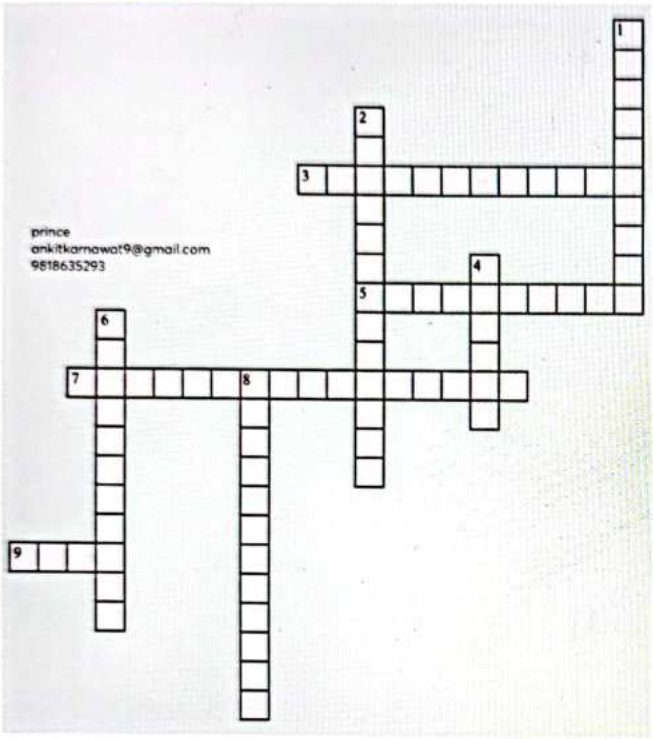
Extracutaneous features:



- Dental - peg/cone shaped crowns
- Ocular: blindness strabismus, nystagmus
- CNS: **MR**
- Skeletal: Skull deformity, spina bifida, dwarfism
- X-linked dominant gene defects– question
- Incontinentia pigmenti
- Focal dermal hypoplasia
- Oral-facial-digital syndrome
- Child syndrome.

CROSS WORD PUZZLES

Crossword Puzzle



Across

3 Mutation in the ATP2C1 gene
5 Hyperpigmented macules present on lips, around mouth, oral mucosa
7 Lamellar Ichthyosis is caused by defeat in this
9 Incontinentia Pigmenti is caused by defect in this gene

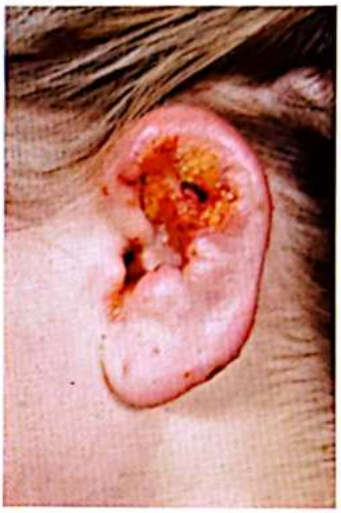
Down

1 Defect is in keratinization which is very important for formation of skin
2 Shiny minute papules present on the nose
4 Supra basal acantholysis which is partial
6 Used as an antibiotic agent, downregulates the ras oncogene PEG
8 Congenital telangiectatic erythema and stunted growth

26 VECTOR BORNE DISEASES

Leishmaniasis 00:00:13

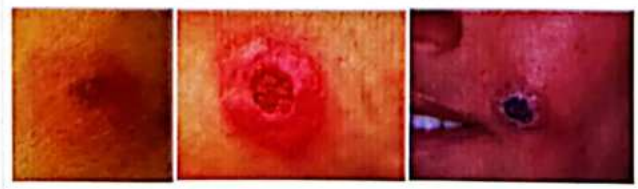
- Protozoal disease
- Caused by leishmania
- **Vector:** Sandfly
- **Classification**
  - Cutaneous
    - → Old world type
    - → New world type
  - Mucocutaneous
  - Visceral
    - → Commonly called as Kala Azar
    - → Can lead to a cutaneous Leishmaniasis
    - – Known as Post Kala Azar dermal leishmaniasis (PKDL).



Old World Cutaneous Leishmaniasis 00:01:20

- In developing countries
- Endemic to India.
  - Northeastern states
  - Rajasthan
- Also called as
  - Delhi boil
  - Baghdad boil
  - Oriental sore
- **Species:** Mnemonic: MAIT
  - L.major
  - L.tropica
  - L.aethiopica
  - L.infantum

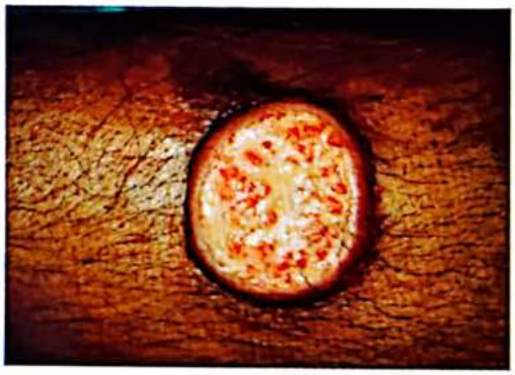
- Clinical features:
  - Erythematous papules
  - Ulcer
  - Central crusting on exposed parts.



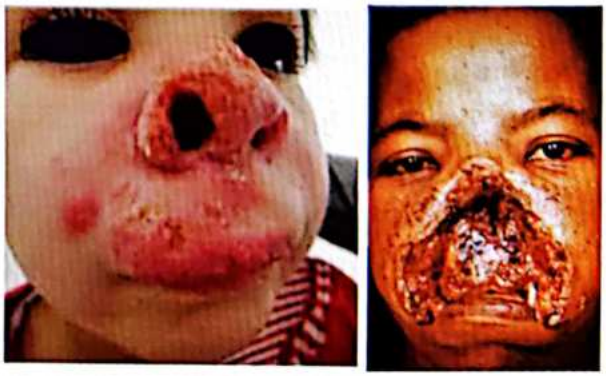
New World Cutaneous Leishmaniasis 00:03:04

- Seen in Mexico
- Species - L. Mexicana
- Ulcer typically affects the ear cartilage - called as Chiclero's ulcer
- Cartilage destruction and ulcer is seen.

Mucocutaneous Leishmaniasis 00:03:52



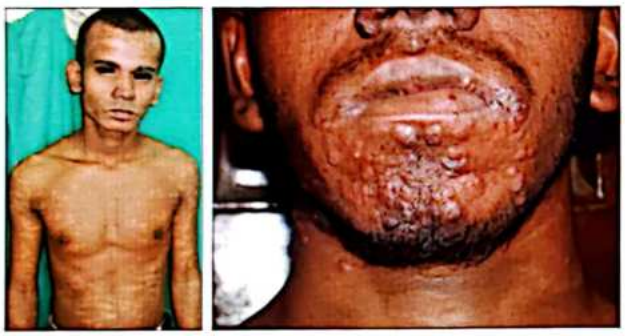
- Caused by L. braziliensis
- Commonly seen in Brazil.
- Starts with cutaneous lesion.
- Cutaneous ulcer is seen on the lower leg. Hematogenous spread enters mucosa Involves nose and oral mucosa destruction of cartilage destruction of the area (Espundia)



- Nose is called as Tapir nose

Post Kala Azar Dermal Leishmaniasis 00:05:15

- The visceral form of Leishmaniasis
- History of Kala Azar
  - Prolonged fever
  - Hepatosplenomegaly
- **Species**
  - L.infantum
  - L.Chagasi
  - L.donovani (Most common species)
- **Endemic areas**
  - North-eastern areas of India
- **Clinical presentations**



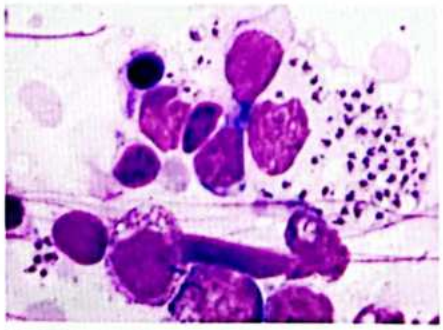
  - Hypopigmentation
  - Macules on face and trunk
  - Papules present on muzzle area of face - The butterfly area or beard area of face

Important Information

- In leprosy,
  - There is no nerve involvement
  - There is no sensory impairment
  - Slit skin smear can be done - There will be no AFPs.

Diagnosis:

- Smear
  - In the tissue - amastigote form of the Protozoa of leishmania will be present.



- Slit Skin Smear test
  - LD bodies: Leishman Donovan bodies
  - These are present in macrophages
- Culture
  - Novy McNeal Nicolle (NNN) media is commonly used
- Biopsy:
  - Diffuse infiltration of macrophages with amastigotes.
- Leishmanin test
  - Not done anymore
  - Tells about immunity

Treatment

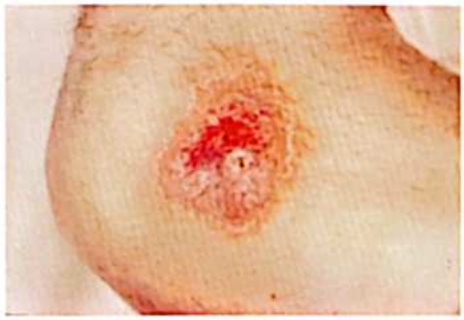
- Parenteral antimonial - Most successful but toxic
- Lipid based amphotericin B
- Miltefosine- Drug of choice

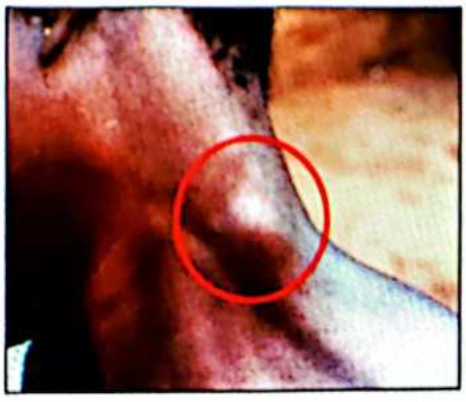
Trypanosomiasis 00:09:01

- Two types
  - African form
  - American form
- Trypanosoma is the protozoa involved.
- There are different vectors.

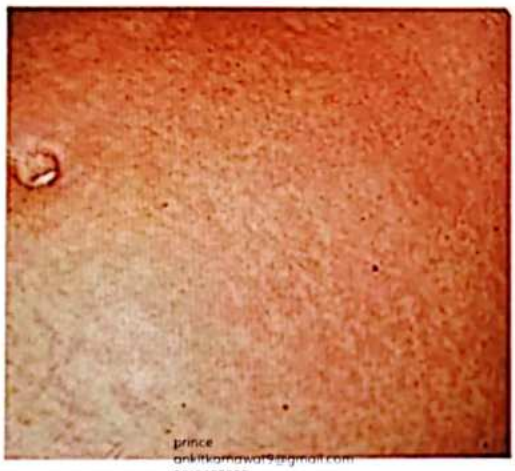
African form

- Also called as sleeping sickness
- Species: Trypanosoma brucei
- In African form, there are
  - West African form
  - East African form
- Vector: tsetse fly
- Clinical Features-





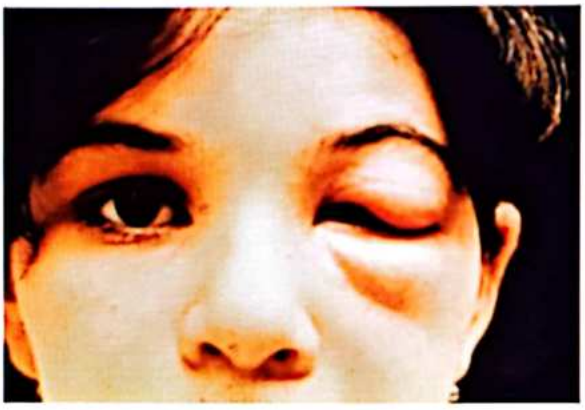
- Painful chancre on lower limbs
- Hematogenous and lymphatic spread
- Involvement of lymph nodes from cervical and post occipital areas
- Leads to a large lymph node - called as Winterbottom sign.
- Rash might be present due to Hematogenous spread



- Treatment-
  - Suramin
  - Pentamidine

American form 00:11:09

- Also known as Chagas' disease
- Species: T. Cruzi
- Vector: Reduviid bugs or kissing bugs
- Clinical features-
  - It leads to unilateral edema of eyelids and lacrimal glands.
  - Leads to Romana's sign

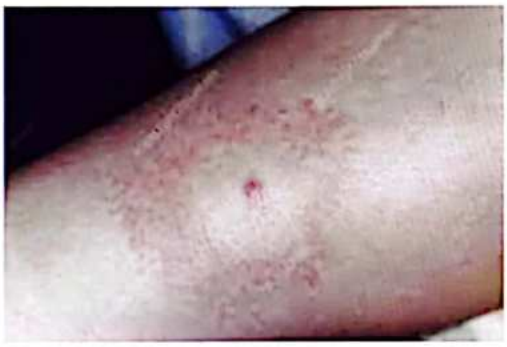


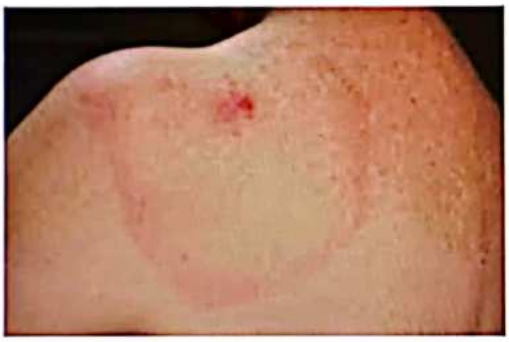
Treatment

- Nifurtimox
- Benznidazole

Erythema Chronicum Migrans 00:12:25

- Characteristic of Lyme disease
- Present in 3 phases
  - Phase 1: ECM
  - Phase 2: Cardiac and neurological disease
  - Phase 3: Arthritis and a chronic neurologic syndrome
- Caused by Borrelia burgdorferi.
- Vector: Ixodid ticks
- It is a migrating chronic erythematous rash.
- Clinical features:

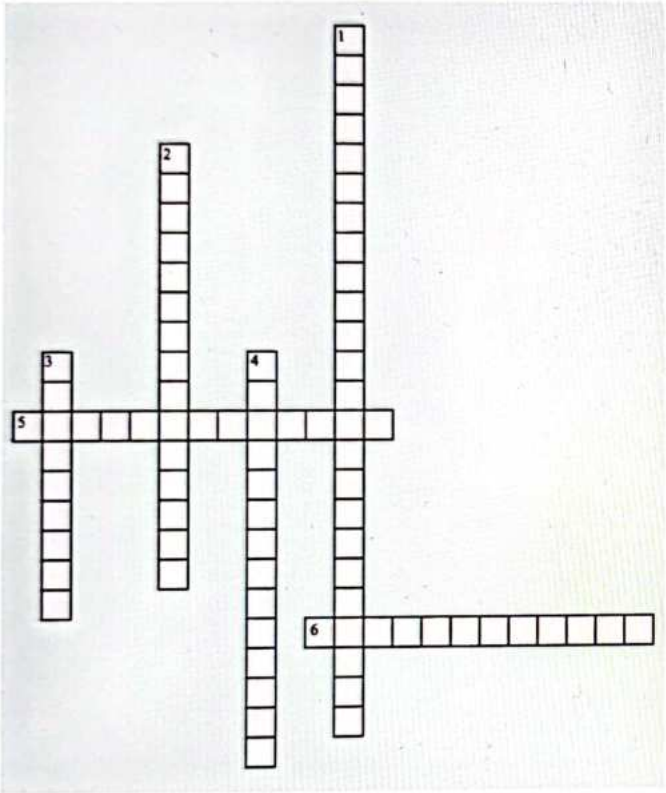




  - Erythema expands away from the central bite punctum centrifugally
  - Clears centrally within a week, leaving a red 1 to 2 cm ring that advances for days or weeks
  - May reach a diameter of 50cm.
  - There is erythema, migration and it is chronic.
  - It is persistent and has central punctum.

CROSS WORD PUZZLES

Crossword Puzzle



Across

5. Protozoal disease classified as cutaneous
6. Old World Cutaneous Leishmaniasisis also called as

Down

1. Characteristic of Lyme disease
2. Found in two types- African & American
3. Species of New World Cutaneous Leishmaniasis
4. Ulcer that typically affects the ear cartilage

27 BEDSIDE TESTS IN DERMATOLOGY

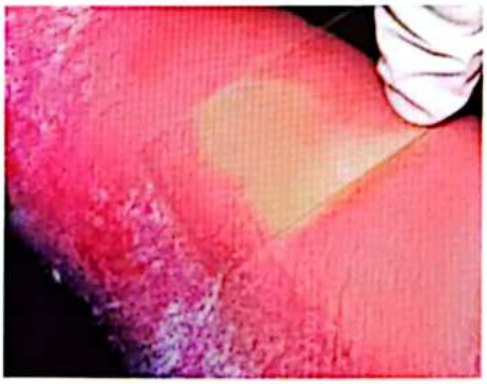
Magnification

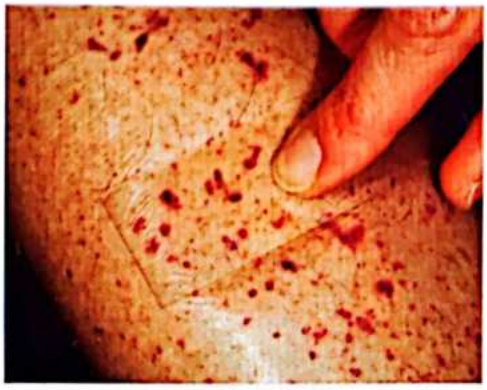
00:00:22

- The most important tool for a dermatologist is a hand lens.
- It is a convex lens
- Gives magnification of 5X-7X

Diascopy

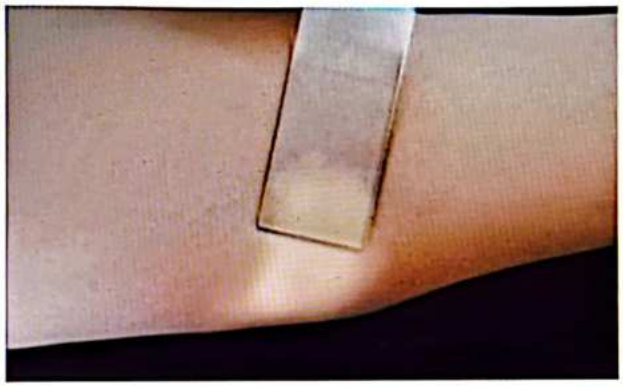
00:00:34

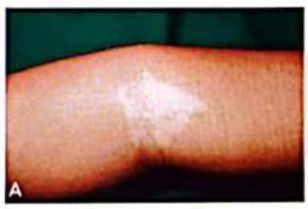




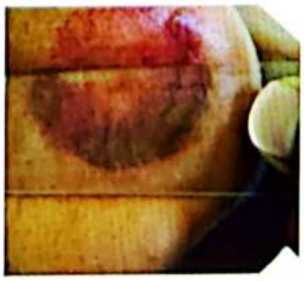
- Take a glass slide and press it on the skin.
- Uses:

1. To differentiate between erythema and purpura
   - When there is simple erythema due to vasodilation
     → If you press, the blood spills out to the sides.
     → So, there will be a blanching effect.
   - In purpura, there will be no change.
     → Because it is either due to vasculitis or extravasation of RBCs.





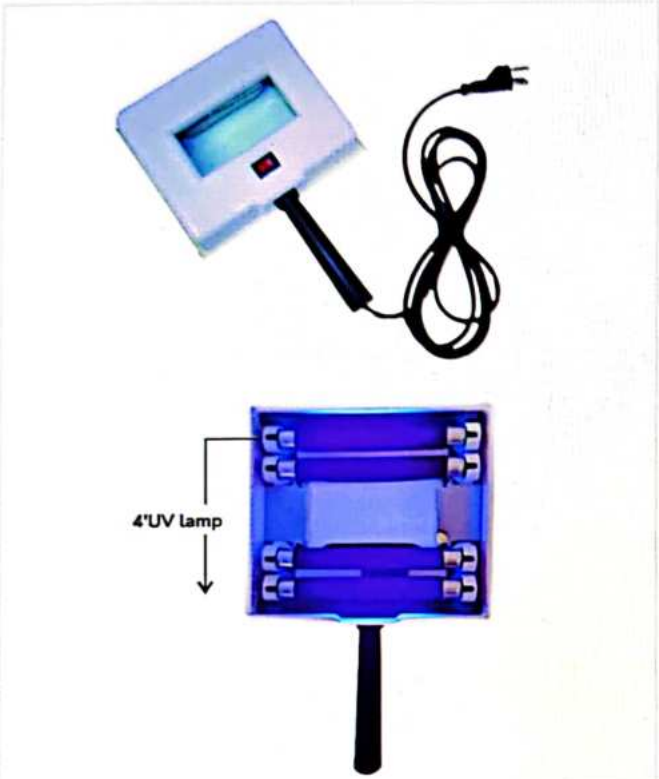
2. To differentiate between nevus anemicus and depigmentosis
   - **In depigmentosis-** no change.
   - **In nevus anemicus-** merge with the surrounding skin.



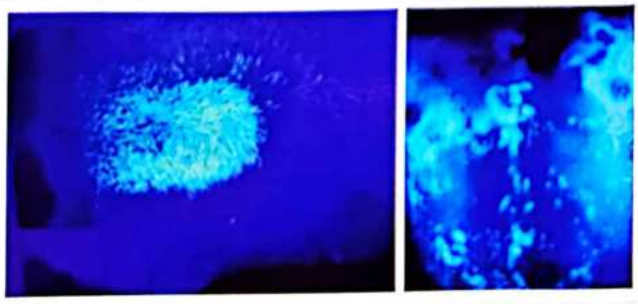
3. To see Apple jelly nodules- seen in
   - Lupus vulgaris
   - Sarcoidosis

Wood's Lamp

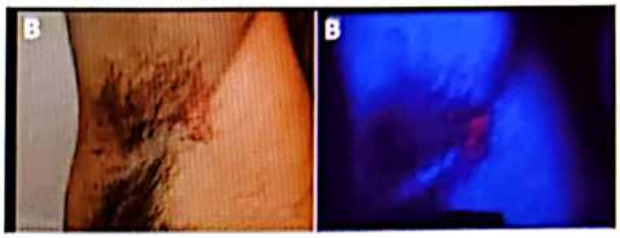
00:02:12



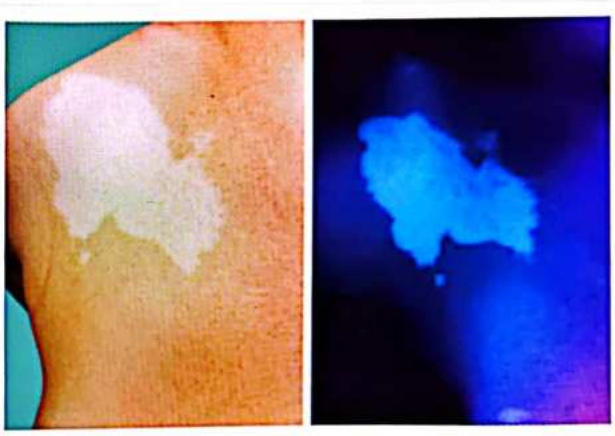
- Low output mercury arc amp
- Covered by wood's filter
  - Barium silicate
  - 9% nickel oxide
- Emits light of wavelength 320-450nm.
  - Peak 365nm
- It is done in dark room.
- Put on the lamp and let it get warmed for a minute on the suspected lesion
- Observe the fluorescence



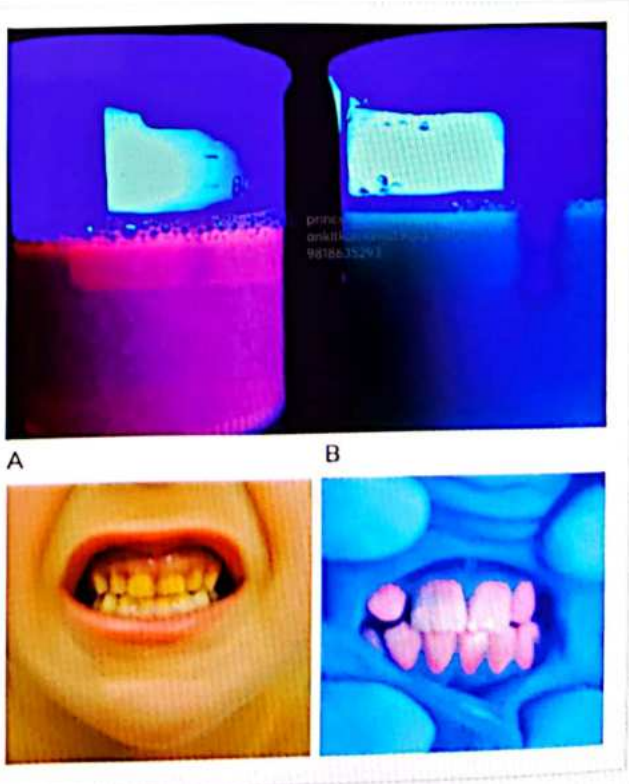
- Used in tinea capitis, pityriasis versicolor
  - Yellowish fluorescence in pityriasis versicolor
  - In tinea capitis, it shows positive fluorescence in grey patches and favours.



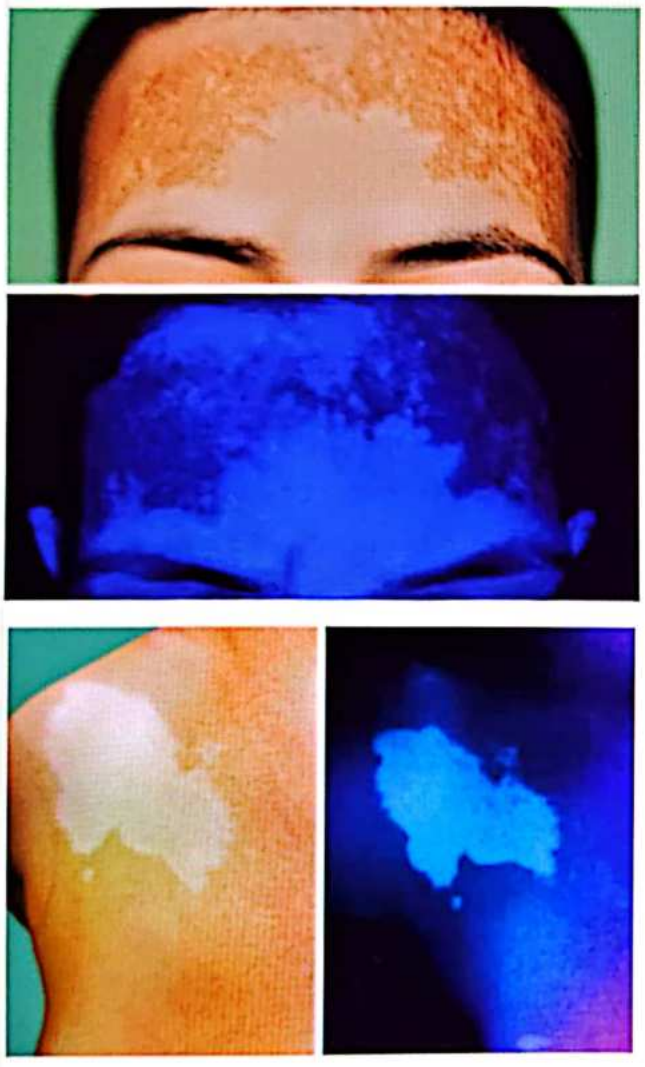
- Used it in erythrasma - Coral red fluorescence



- It helps to differentiate nevus de pigmentosus
  - In the presence of a wood lamp, it will accentuate.
- It can be used in vitiligo
  - It can accentuate on wood's lamp examination.



- It can be used in congenital erythropoietic porphyrias.
  - Fluorescence can be observed in teeth and urine.



- It is used in melasma
  - Epidermal pigmentation will accentuate
  - So, there will be no much change in dermal pigmentation.

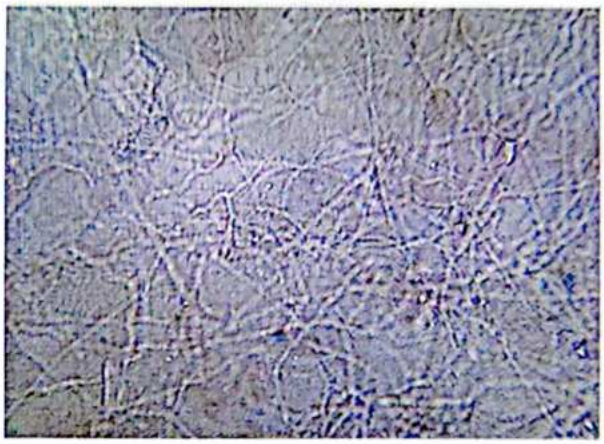
Smears

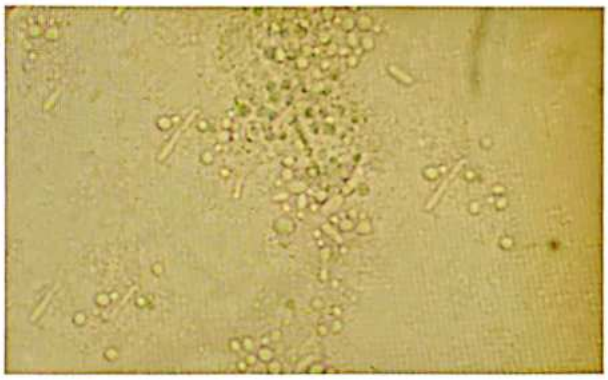
00:04:59

- KOH
- TZANCK
- GRAM's
- Wet preparation
- Tissue smear
- Mite demonstration
- Slit skin smear

KOH Smear

00:05:04



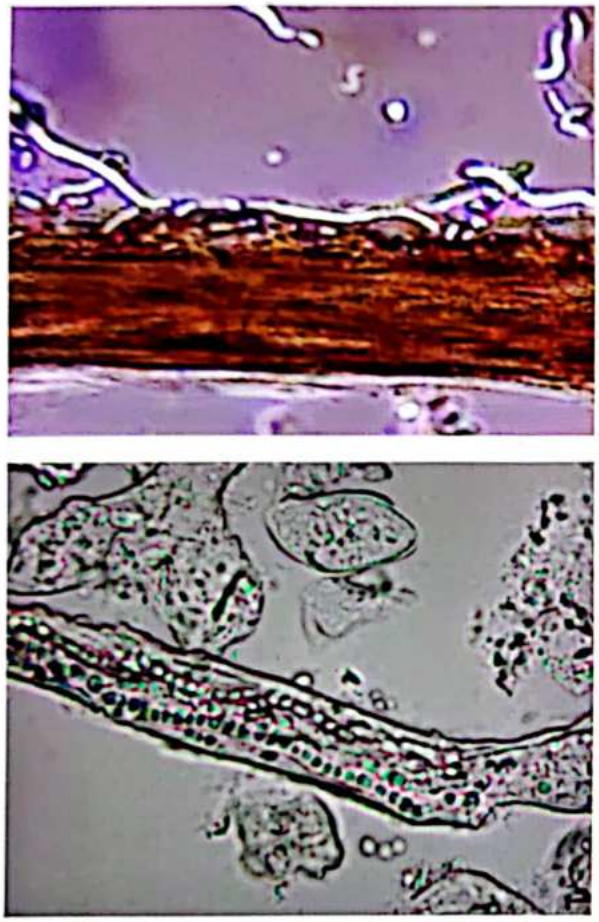


- Used in fungal infections
- Candida - Budding yeast cells
- Dermatophyte- multiple branching hyphae is seen

Pityriasis versicolor:

- Spaghetti and cheese ball appearance

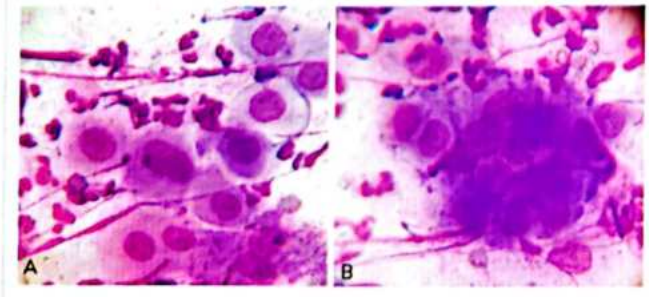
Tinea capitis:



- Then KOH is used to distinguish ectothrix and endothrix in Tinea capitis
- In ectothrix, spores are outside the hair shaft
- In endothrix, spores are inside the hair shaft

TZANCK Smear

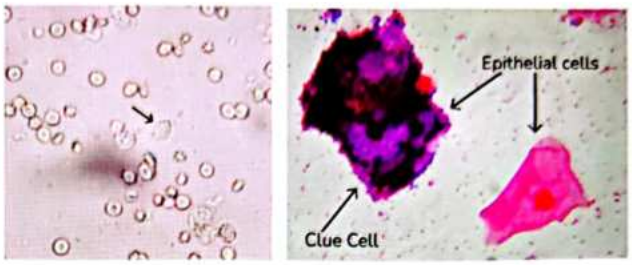
00:05:58



- Done for- Pemphigus and Herpes simplex
- It is done where the Acantholytic cells are along with multinucleated giant cells.
- In herpes simplex- both acantholytic and multinucleated giant cells are present.

Wet Smear

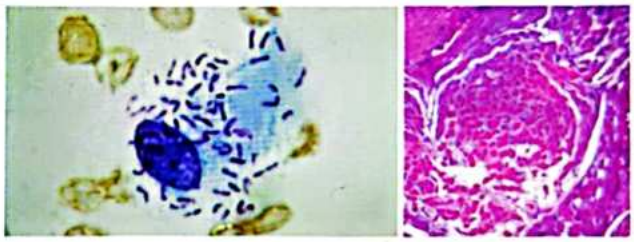
00:06:28



- Done for- Trichomoniasis and Bacterial vaginosis
- Trichomoniasis - Pear shaped jerky movements are seen.
- Bacterial vaginosis - Closed cells are seen.

Tissue Smear

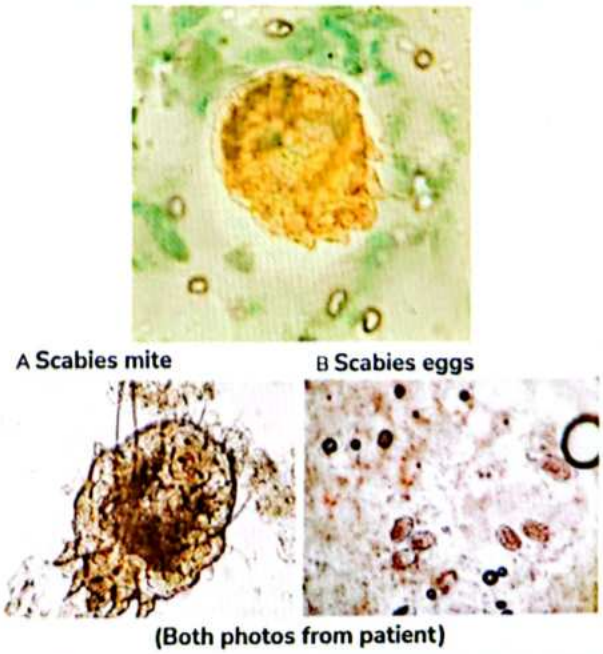
00:06:54



- Done in
  - Molluscum- LD bodies are seen
  - Donovanosis- Safety pin appearance is seen

Scabies Mite Demonstration

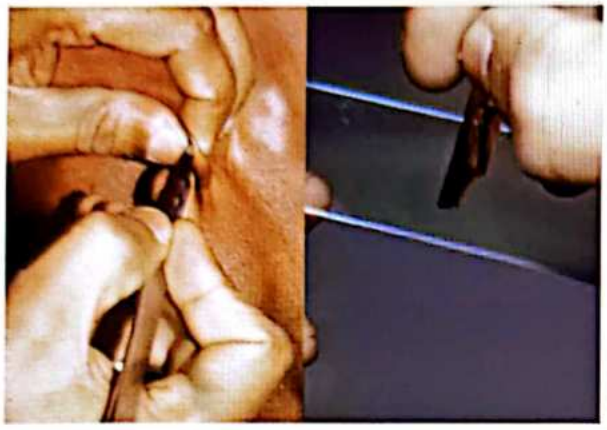
00:07:16



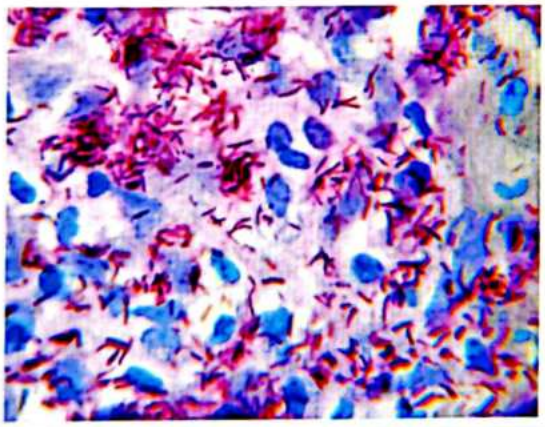
- Eggs, mites and skybella are seen.

Slit Skin Smear

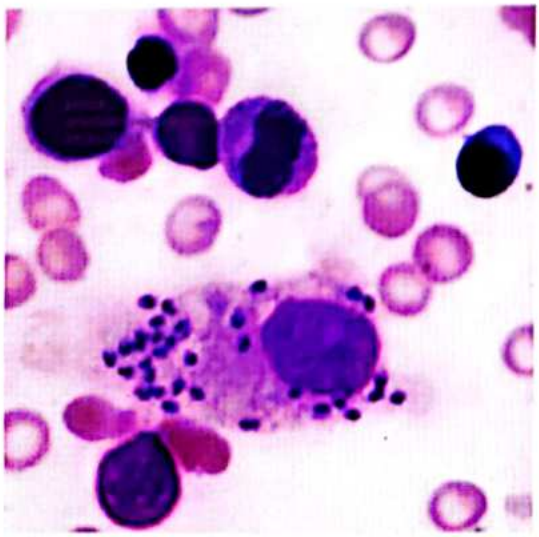
00:07:22



- Pinch the ear lobe and take a no.15 scalpel blade with 5mm length and 3mm width
- Then take a smear
- Done for
- Leprosy



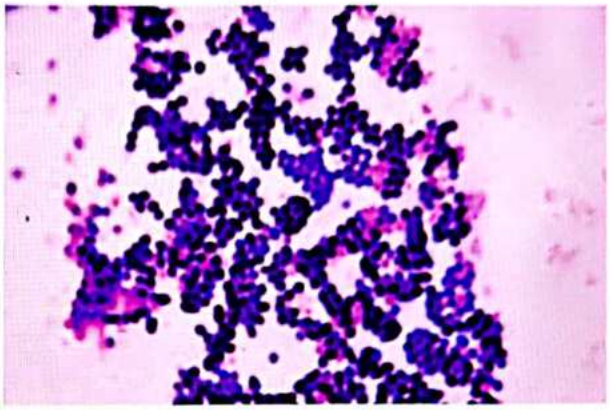
- Leishmaniasis to demonstrate LD bodies.

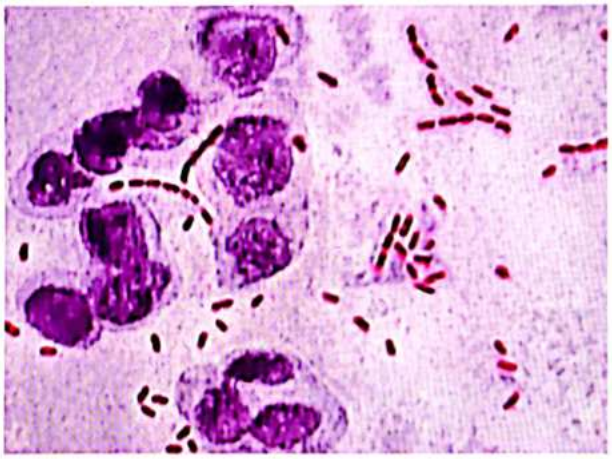


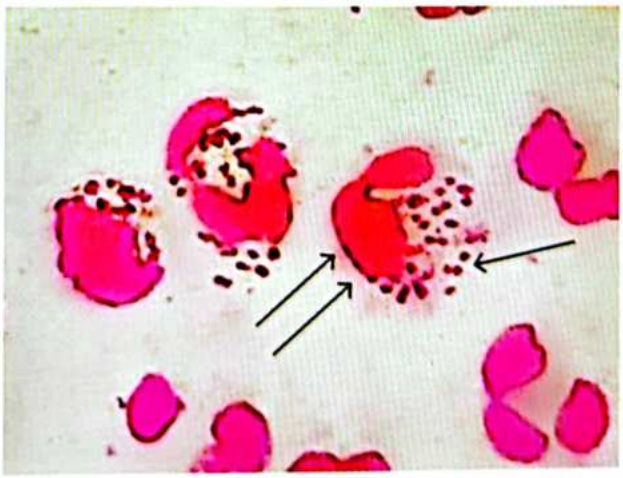
Gram Stain

00:07:56

- Used in various bacterial infections





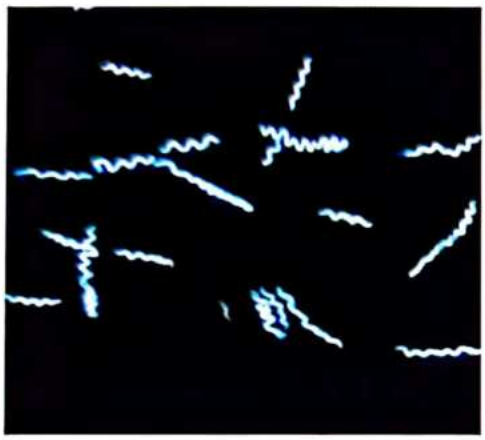


- **In impetigo-** Gram positive cocci in clusters is seen.
- **In chancroid-** The rail track appearance is seen
  - Or fish and stream appearance
- **In gonococcal urethritis-** Intracellular diplococci is seen.
  - These both are gram negative and the other is gram positive

Dark Ground Microscopy

00:08:28

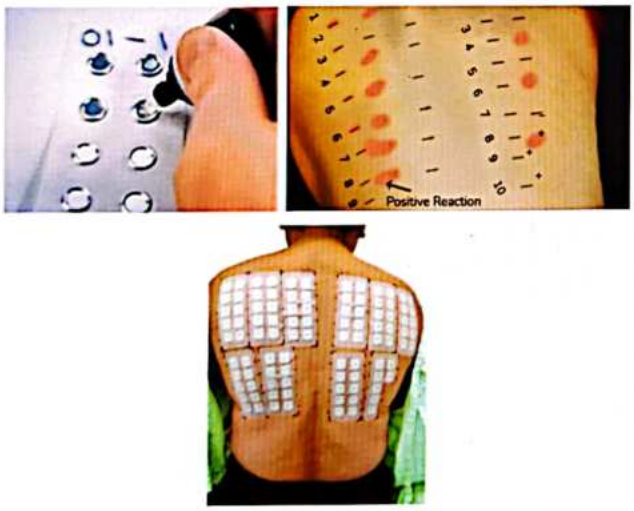
Done for- Treponema pallidum



Patch Test

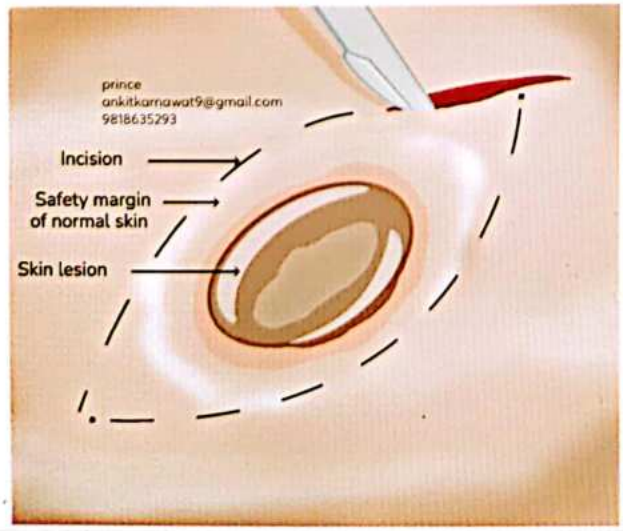
00:08:41

- Done for – Eczema



Skin Biopsy

00:08.50



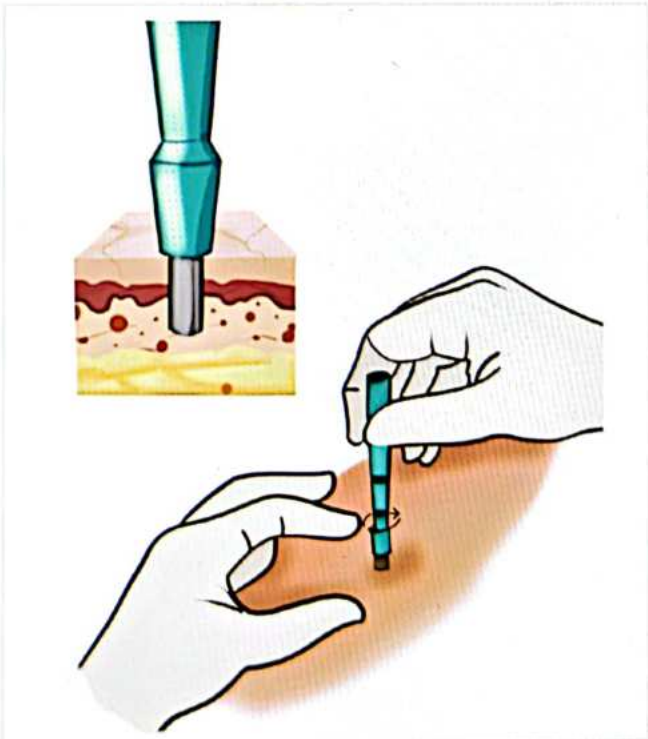
- **Incisional biopsy**
  - If there is a lesion, or plaque
- Take a small biopsy from the plaque
  - This is called an incision biopsy.
- **Excisional biopsy**
  - Lesion suspected as tumour or premalignant condition
- Whole lesion must be removed
  - This is called an excision Biopsy.
- **Shave biopsy**
  - If the lesion is present superficially on the skin- Take a scalpel or a blade and shave it off from the surface
  - This is called as shave biopsy
  - This can be done for
    - → Actinic Keratosis
    - → Naevi

Incisional Biopsy

00:10:29

- **Two ways** to perform incision biopsy:

1. **Incisional biopsy**
   - Taking small elliptical incision
   - Disadvantage- Ragged margins
2. **Punch biopsy**



- Punch is used and inserted on the skin to take a perfectly round biopsy - It must heal very well.
- Most common biopsy
- Advantages-
  - → Very convenient
  - → Punches come in various sizes like 3mm, 4mm, 5mm etc.

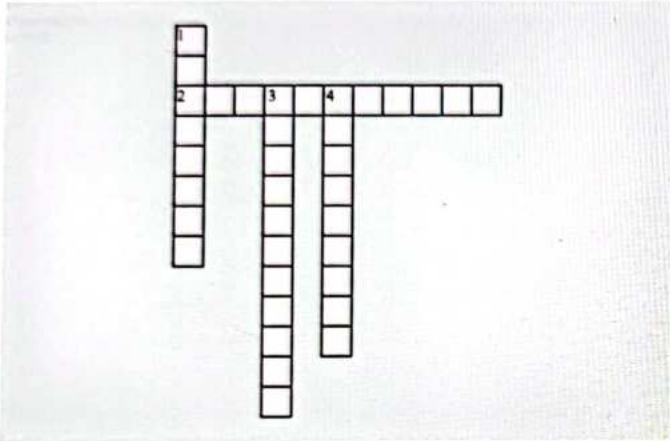
→ Healing is very good.
→ Has a perfect circular margin.

Important Information

- Whenever there is a tumour, excision biopsy must be done.
  - It may be therapeutic
  - Suspicion of malignancy

CROSS WORD PUZZLES

Crossword Puzzle



Across

2. It is done in Molluscum, leish and Donovanosis

Down

1. It helps to differentiate nevus de pigmentosa
3. Done for Actinic Keratosis
4. Spores outside the hair shaft