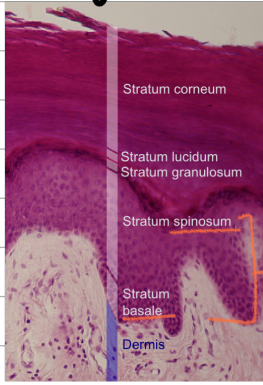


# Malpighian layer:



Malpighian layer = St. spinosum + st. basale

**Rx:**

- <20% Body Surface Area:
  - Topical tacrolimus
  - Topical steroids
- >20% BSA:
  - Narrow band Phototherapy
  - Systemic Steroids
  - Azathioprine

**Vitiligo:** onset: acquired disorder  
mechanism: autoimmune hypothesis (m/c)

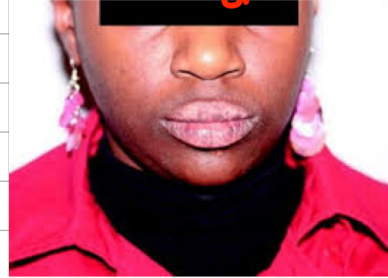
## Localized



## Segmental



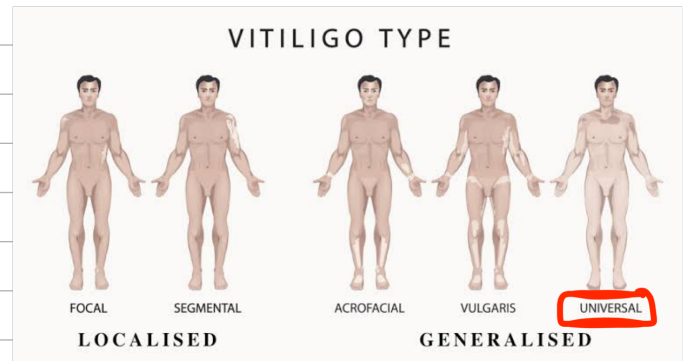
## Mucosal type



## Generalized



## Vitiligo (m/c type)



**O. Kobner's phenomenon:** along the line of trauma, new lesions start appearing in a vitiligo patient

**Leukoderma:** is induced destruction of melanocytes

Fig 14: hypopigmented macule over forearm



# Scleroderma: also known as systemic sclerosis

(thickening of skin)

↳ F >> M  
(mostly post menopausal women)

The limited symptoms of scleroderma are referred to as CREST

## Limited cutaneous

Skin thickening Restricted to distal to the elbow, knee & face

## Diffuse cutaneous

distal & proximal to the elbow & knee, trunk

**Pathogenesis:** Fibrosis - TGFβ  
Vascular dysfunction  
Immune dysregulation

**C**alcinosis- calcium deposits in the skin



**R**aynaud's phenomenon- spasm of blood vessels in response to cold or stress



**E**sophageal dysfunction- acid reflux and decrease in motility of esophagus



**S**clerodactyly- thickening and tightening of the skin on the fingers and hands



**T**elangiectasias- dilation of capillaries causing red marks on surface of skin



ADAM

Salt & pepper pigmentation

**Atopic Dermatitis:** It is classified as an endogenous eczema triggered by exogenous agent.

Chronic, Relapsing, inflammatory skin disease.

Atopy: Localized form of Type I reaction

**Pathogenesis:**

- Defective barrier function of skin
- Filaggrin mutation
- ↑ IgE response to an allergen

## Atopic triad:

- Allergic rhinitis
- Atopic Dermatitis
- Atopic Asthma (Bronchial asthma)



Indian Association of Dermatologists, Venereologists and Leprologists

**Nevus of Ota and Ito**

- Nevus of Ota (Blue-gray pigmented macule or patch on the upper half of face).
- Nevus of Ito : shoulder area
- Both are congenital dermal pigmentation.
- Pathogenesis - Migration arrest of melanoblast that arises from neural crest.













*epidermal nevi*

## skinScan™

### THE ABCDE SYSTEM OF MELANOMA DETECTION

The ABCDE criteria represent a commonly used clinical guide for early diagnosis of melanoma. The following features are considered suspicious:

A B C D E	<b>Asymmetry:</b> Moles that have asymmetrical appearance		
		Symetrical	Asymetrical
	<b>Border:</b> A mole that has blurry and/or jagged edges		
		Smooth borders	Irregular borders
	<b>Color:</b> A mole that has more than one colour		
	Single color	Multicolor	
<b>Diameter:</b> Moles with a diameter larger than a pencil eraser (6 mm or 1/4 inch)			
	Smaller than 6mm/0.2in	Bigger than 6mm/0.2in	
<b>Evolution:</b> A mole that has gone through sudden changes in size, shape or colour			
	No changes	Some changes	

## Erythema Multi-forme

• Acute, immune mediated, self limiting, cytotoxic dermatitis

- Aetiology:**
- Infections: HSV1 (m/c), Mycoplasma pneumoniae
  - Drugs: Antimalarials, NSAIDs, Sulphonamides
  - Connective tissue: Systemic Lupus Erythematosus (SLE) disorder
  - Neoplasia: Leukaemia, internal malignancies

**Special lesion:** Target lesion with 3 zones.

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

**Distribution:** Distal extremities (like palms, soles)

**Rx:** Treat the underlying case.

- Short course of systemic steroids if necessary.



## Systemic Lupus Erythematosus (SLE)

Female: Male  
(9:1)

- Disease in females in Reproductive Age group
- Male SLE: Poor prognosis
- Childhood SLE: 100% Renal involvement (Lupus Nephritis)

Q. Strongest Genetic factor of SLE: Early component deficiency

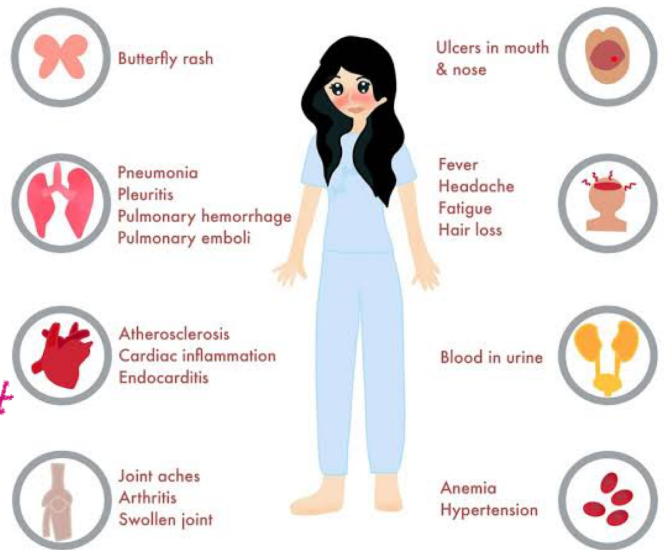
C1q, C2, C4

Q. Single most important genetic factor?

C1q deficiency

- TREX gene mutation (present on chromosome 3)

## Systemic LUPUS Erythematosus (SLE)



**SLICC<sup>†</sup> Classification Criteria for Systemic Lupus Erythematosus**

Requirements: ≥ 4 criteria (at least 1 clinical and 1 laboratory criteria)  
OR biopsy-proven lupus nephritis with positive ANA or Anti-DNA

**Clinical Criteria**

1. Acute Cutaneous Lupus\*
2. Chronic Cutaneous Lupus\*
3. Oral or nasal ulcers \*
4. Non-scarring alopecia
5. Arthritis \*
6. Serositis \*
7. Renal \*
8. Neurologic \*
9. Hemolytic anemia
10. Leukopenia \*
11. Thrombocytopenia (<100,000/mm<sup>3</sup>)

**Immunologic Criteria**

1. ANA
2. Anti-DNA
3. Anti-Sm
4. Antiphospholipid Ab \*
5. Low complement (C3, C4, CH50)
6. Direct Coombs' test (do not count in the presence of hemolytic anemia)

<sup>†</sup>SLICC: Systemic Lupus International Collaborating Clinics

\* See notes for criteria details

Petri M, et al. Arthritis and Rheumatism. Aug 2012

**Wiskott Aldrich Syndrome**

- X-Linked Recessive disorder
- More common in males >>> Females
- Pathogenesis: ↓

WASP gene defect on chromosome Xp11.23

- Decreased IgM, ↑ IgA4
- normal IgG.

- Mnemonic: **WAITER**
- W - Wiskott, WASP gene Defect
- A - Aldrich
- I - immunodeficiency
- T - T cell deficiency, Thrombocytopenia
- E - Eczema
- R - Recurrent infections.

• Clinical features:

- Affects 1 in 10 of every 1 million male newborns
- Combined immunodeficiency (humoral and cellular)
- Eczema
- Thrombocytopenia with small platelets. (↓production; ↑destruction)
- Increased risk of autoimmune disorders and cancers

Defective T cell development

Triad (only in 1/3<sup>rd</sup>)



**Leprosy / Hansen's disease:** is a chronic infectious granulomatous disease predominantly affecting skin & nerve.

• **Etiology:** *Mycobacterium leprae*  
MB Lepromatosis.

• **Incubation Period:** 2-5 years.

• **Transmission:** Respiratory Portal / inhalation (major) & skin to skin contact.

• **Staining:** Modified Ziehl-Neelsen stain Used (5% H<sub>2</sub>SO<sub>4</sub>)

• **Generation Time:** 11-13 days

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

### 5 types of Classifications in Ridley Jopling Classification:

TT - Tuberculoid type

BT - Borderline Tuberculoid

BB - Borderline Borderline

B<sub>L</sub> - Borderline Lepromatous

LL - Lepromatous leprosy

Decreasing  
Cell Mediated immunity  
(CMI)

→ TT: • No. of lesions - 1-3

• Morphology - Annular plaque

• Center lesion - cigar

• Periphery - Raised margins (Saucer Right way up)

• Associations - Granuloma forms in response to bacteria

• Loss of Nerve - Anesthesia

• Loss of Hair - Alopecia

• Loss of sweat glands - Anhidrosis

→ BT: • No. of skin lesions: 3-10

• Margins: Each lesion has well defined and ill defined margins

• Satellite lesions are also seen.

## Mid-borderline leprosy

03



→ BB: No. of lesions - 10-30

Most unstable form of Hansen's disease.

Polymorphic skin lesions appears like geographic map

• Skin lesions: Dimorphous skin lesions.

↓ Hence aka Dimorphous Leprosy.

• Inner border: Punched out.

Swiss cheese appearance

• Outer border: Sloping outwards

Inverted saucer appearance.

(Seen both in BB & BL)



→ BL: Skin Lesions: Numerous, B/L & almost Symmetrical

• Nerve involvement → B/L asymmetrical nerve thickening



→ LL: Symmetrical B/L @ thickening.

• Late features - Leonine Facies - occurs due to diffuse infiltration of the face

• Madrosis - Loss of eyebrows

• Glove & stocking Peripheral neuropathy

Scabies: Itchy contagious ectoparasitic infection

Etiology: itch mite → *Sarcoptes scabiei* var *hominis*.

Transmission:

- close personal contact: skin to skin contact.
- Also transmitted via fomites like clothes, bedding.

Classified as water washed disease.

- Seen with ↓ personal hygiene.

Incubation period:

- 1<sup>st</sup> time: 4 weeks
- Re-infection: 1-2 days.

Rx: 5% Permethrin: Best drug.

↓  
inhibits Na<sup>+</sup> channels in the parasite  
↳ induces paralysis.

• Below neck application, kept for 12 hours.

• Ivermectin - orally - 200 µg/kg

C/f: History of nocturnal pruritus.

- mite becomes active at night
- Pruritus happen due to delayed Hypersensitivity response to the mite.

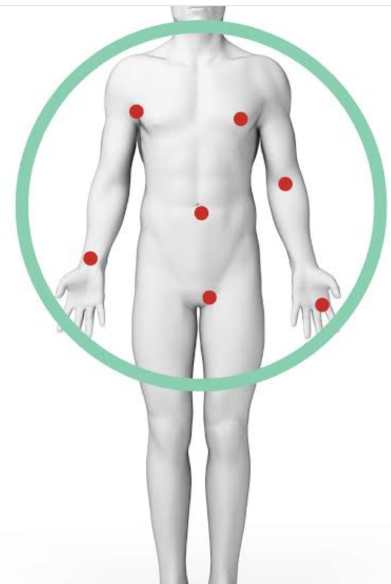
A. Pustule and Pimple-Like Lesions



B. Rash



C. Burrow Lesions (less commonly found)



Circle of Hebra

Distribution of scabies  
: circle of Hebra.

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

*Sarcoptes scabiei*  
itch mite

Note: face is spared in adults (due to red sebum activity that repels mites)



Investigations: KOH mount of skin scrapping.

- The mite is identified by:
  - Two anterior pair of legs.
  - Two posterior pair of legs.

**Alopecia** : Defined as loss of hair.

- Types :
- Non scarring (non-cicatricial)
  - Scarring (cicatricial)
- Cicatrix means scar.

**CAUSES OF NON - SCARRING & SCARRING ALOPECIA - MNEMONIC**  
WWW.OPENMED.CO.IN

NON - SCARRING ALOPECIA	SCARRING ALOPECIA
<p><b>MNEMONIC - THATS STD</b></p> <ul style="list-style-type: none"> <li>• Tinea capitis (Non Inflammatory Type)</li> <li>• Hormonal - Hypothyroidism, Androgenetic alopecia</li> <li>• Anagen effluvium, Alopecia areata</li> <li>• Trichotillomania</li> <li>• Secondary syphilis (moth eaten alopecia)</li> <li>• SLE</li> <li>• Telogen effluvium</li> <li>• Deficiency of Zinc, Iron</li> </ul> <p>WWW.OPENMED.CO.IN</p> 	<p><b>MNEMONIC - STD DVL</b></p> <ul style="list-style-type: none"> <li>• Scleroderma</li> <li>• Tinea capitis (Inflammatory Type - Kerion)</li> <li>• Folliculitis Decalvans</li> <li>• DLE</li> <li>• Lupus Vulgaris</li> <li>• Lichen Planopilaris</li> </ul> <p>WWW.OPENMED.CO.IN</p> 

Features	Non-scarring (non-cicatricial)	Scarring (Cicatricial)
• Hair follicles	Preserved	Destroyed
• visible sign of inflammation	Absent	Pustules/scaling present
• Course	Generally Reversible	Irreversible/permanent

**Alopecia Areata**

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

Rx : Localized disease :

↓  
intralesional steroids  
(Triamcinolone acetone)

• Extensive disease :

- Systemic
- Azathioprine
- Sulfasalazine
- Tofacitinib : Potent JAK-1/JAK-3



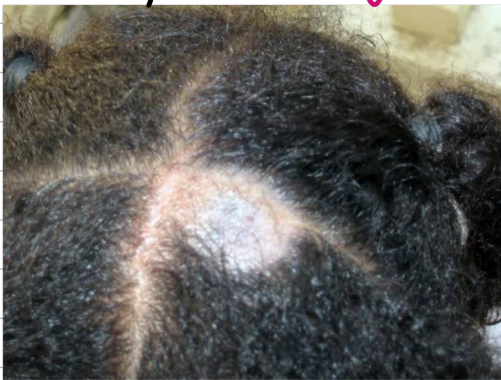
Geometrical pitting : fine, superficial, regular pitting.

**Trichotillomania** : an obsessive compulsive disorder characterized by pulling out one's own hair.



- incomplete loss of hair within a patch.
- Hair of varying length.
- Perifollicular haemorrhage.

**Tinea Capitis** : (non-inflammatory) : invasion of hair shaft.



- Types ::
- Black dot type. (endothrix)
  - Grey dot type. (ectothrix)

Causative organism ::

- *Trichophyton tonsurans*.
- *Trichophyton violaceum*.

**Secondary syphilis alopecia** : Moth eaten alopecia.



- Hematogenous dissemination of *Treponema pallidum*

**Anagen effluvium** : chemotherapy / radiotherapy (3-4 weeks)



The arrest of hair growth  
in anagen phase

Dystrophic anagen

Hair loss

**Telogen effluvium** : Major stress / Post pregnancy / Infections (malaria, typhoid, COVID-19) / Surgical procedures



Premature entry of hair into the telogen phase

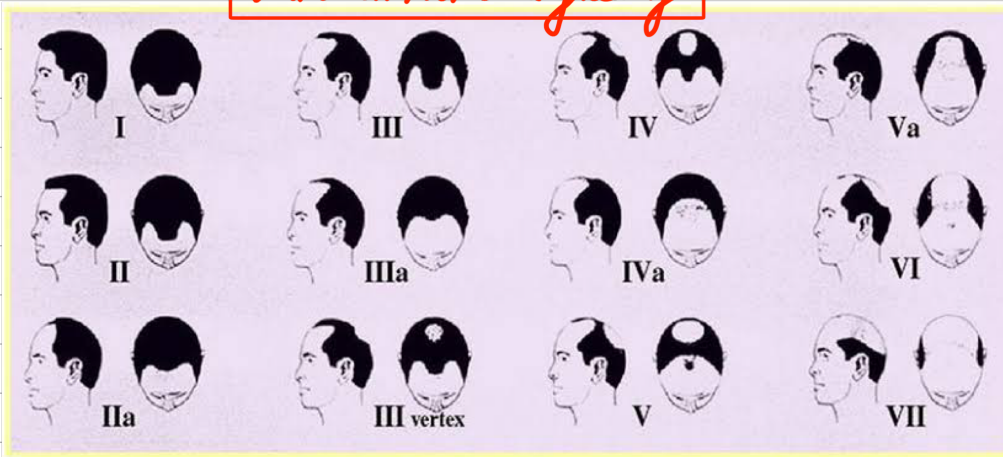
3 months later  
↓  
Hair loss

**Patterned type / Non-cicatricial alopecia** : Testosterone is converted to dihydrotestosterone, which is responsible for thinning of hair & alopecia.

↓  
• **Synonym**: Androgenetic Alopecia.  
• **factors**: Genetic factor + androgens

• leads to the shortening of the anagen phase  
• Miniaturization of Hair follicles.

### Hamilton Norwood grading

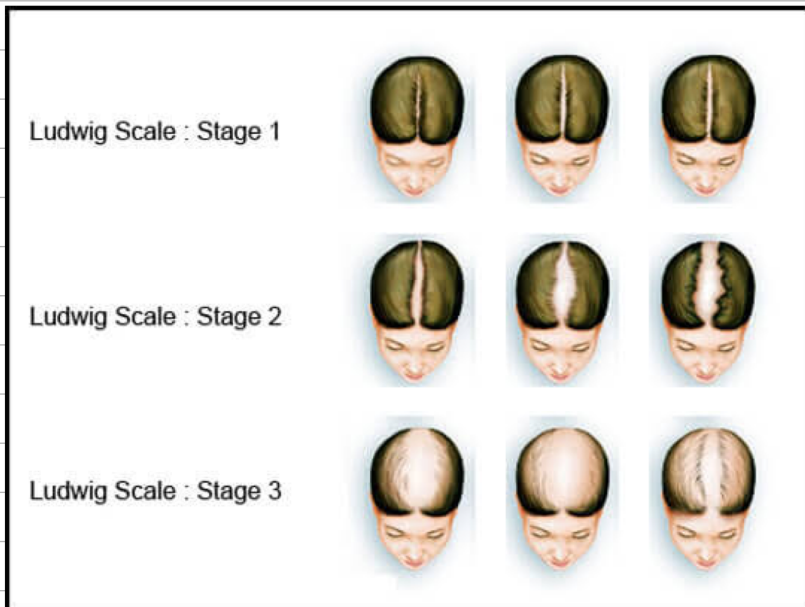


Rx: **Monoxidil** → ↑ anagen phase

2% BD (females)      5% BD (Males)

- **Finasteride** → ⊖ 5α reductase
- Platelet rich plasma therapy
- Hair transplantation.

**Ludwig's grading** : for female androgenetic alopecia : → Loss of hair over the central portion of the scalp.



• Frontal hair line maintained.

## Favus



Scutula  
(yellow cup shaped crust)

## Kerion



• *T. schoenleinii*

← Causative organism →

Zoophilic fungus • *T. Mentagrophytes*  
• *T. verrucosum*

• Yellow cup shaped crust.

← lesion →

Boggy indurated scalp swelling.

• endemic in Kashmir

• Commonly seen in children

Investigation: 10% KOH mount

Rx: overall drug of choice for *T. capitis*: Griseofluvin.

• for *T. capitis* with trichophyton species: Terbinafine.

Ichthyosis Vulgaris: • Disorder of keratinisation.  
• fish like scales + No inflammation.

• onset - 3-12 months

• inheritance - AD

• Defect - filaggrin (filament aggregating protein)

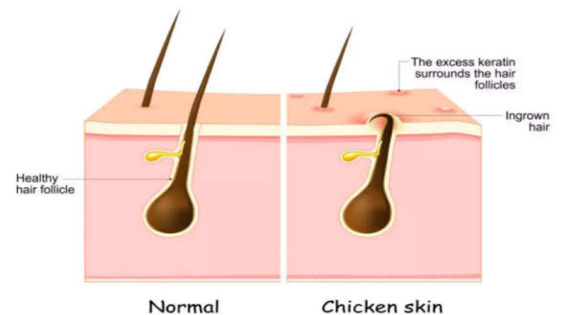
• fine white scales seen.

• Association - Keratosis pilaris (occlusion of follicles)

• Hyperlinear palms



## Keratosis pilaris



## Cutis Marmorata

Skin Marbling

C/F: Disease - Physiologic Vascular response to Cold.

Age group - Seen in Neonates

Lesion - Reticular (network like) Symmetric Marbling.

Site - Trunk, extremities.

Changes with temperature - Warming the child resolves cutis marmorata



## Varicella Zoster

The Primary infection is varicella / chicken pox.

On reactivation it can cause Herpes Zoster.

Varicella / chicken pox:

- incubation period: 14-17 days

- infectious period: 2 days before the rash develops till all the lesions have crusted.

- Lesions: dewdrop on rose petal appearance.

- Pleomorphic rash: characteristic of varicella.



Different stages of rashes in chicken pox are: macules → papules → vesicles → pustules  
↓  
Scabbing.

Atopic dermatitis - Chronic Relapsing inflammatory skin disease.

- Atopy: Localized Type 1 Hypersensitivity rxn.

- Triad: History of Allergic rhinitis,

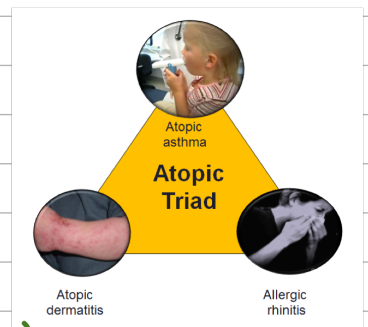
- Bronchial asthma

- Atopic dermatitis

Clinical phases - infantile: upto 2 years (Face, scalp, Extensors)

- childhood: 2 years to puberty (involvement of flexor)

- Adult: > puberty, Lichenification of flexor



## Key diagnostic criteria: Hanifin + Rajka Criteria.

- Pruritis.
- Typical morphology and distribution.
- Chronic/chronically relapsing dermatitis.
- Personal/ Family History of Atopy.



Skin changes -  
If u stroke skin & white lines are formed: white dermographism.



Facial Erythema sparing perinasal, perioral area: Headlight sign.



Eye changes:

- Conical cornea - Keratoconus.
- Cataract - Sheild cataract (anterior subcapsular).
- Infraorbital skin fold - Dennie morgan folds.
- Lateral madarosis (AD) - Hertoghes sign.



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

- HLA Cw6: a/w early onset psoriasis.
- HLA B27: a/w psoriatic spondylitis.
- PSORS gene (Psoriasis susceptibility gene):
  - Expressed in Chromosome 6.
  - 50% Risk of developing psoriasis.



- Drugs that worsens psoriasis:
  - Lithium
  - Antimalarials
  - $\beta$ -blockers (anti HTNsive drug)
  - NSAIDs
  - Systemic painkillers
- Infections:
  - Group  $\beta$ -hemolytic streptococci can trigger guttate psoriasis.
  - HIV worsens psoriasis.

- Rx:
  - <10% BSA involved  $\rightarrow$  Topical
  - >10% BSA involved  $\rightarrow$  Phototherapy
  - Systemic therapy

\* Systemic steroids are contraindicated.  
 $\rightarrow$  only indication: pustular psoriasis of pregnancy.  
 (impetigo Herpetiformis)



DOC  $\rightarrow$  Acitretin (oral retinoid)

2nd DOC  $\rightarrow$  Cyclosporine

**Koebner's Phenomenon**: appearance of morphologically similar lesions along line of trauma over normal skin.

- Implies an active disease.

### True KP

- Immunologic
- e.g. Psoriasis,
- Lichen planus
- Vitiligo

### Pseudo KP

- Auto inoculation
- occurs due to viral infection.
- Lesion multiply in a line following trauma (scratching)
- e.g. molluscum contagiosum, viral warts.



## Acute Paronychia → Staphylococcus aureus.

- infection of nail fold.
- Nail fold shows tender erythema and purulent collection

Rx: if abscess → Topical/Systemic Anti-Biotics

- if no abscess → Surgical drainage



## Chronic Paronychia → candida Albicans

Rx: Anti-fungal + Steroids



## Primary Lesions:



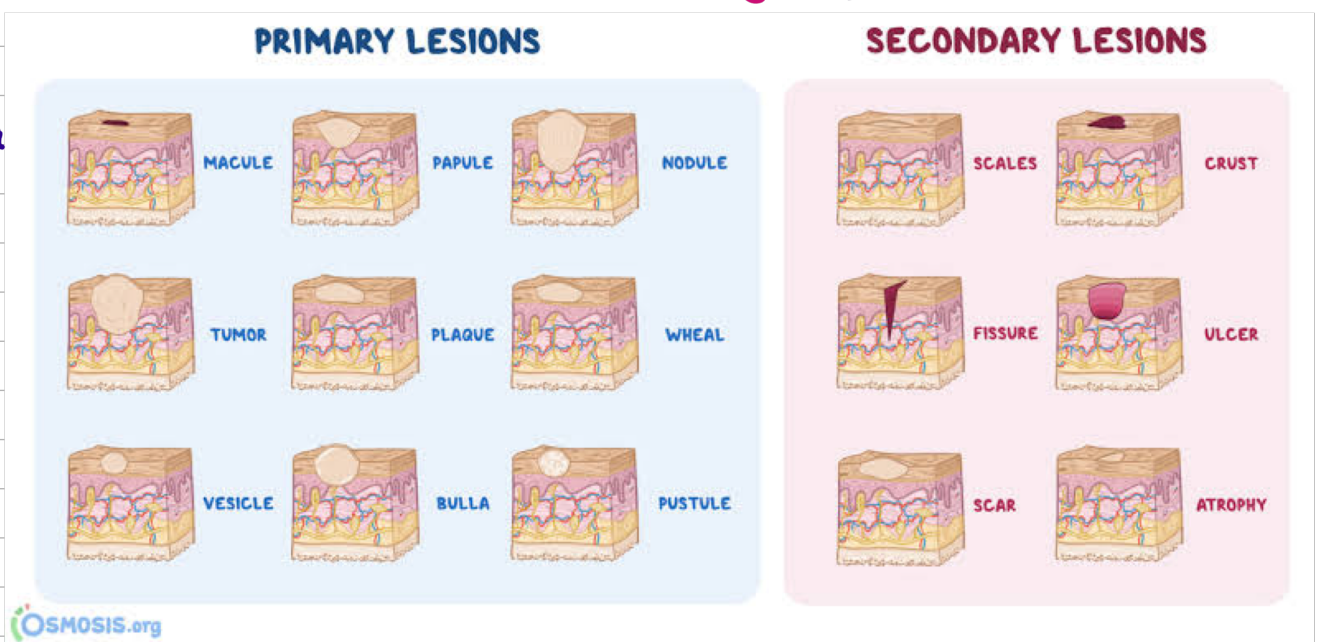
**mnemonic:** *Predatory Poisonous Wasps Viciously  
Bit on the Nose of My Patient  
Causing Purulent Pus.*

Papule  
Plaque  
Wheal  
Vesicle  
Bulla  
Nodule  
Macule  
Patch  
Purpura  
Pustule

## Secondary Lesions:

**mnemonic:** *'LEAF SUCKS water'*

L - Lichenification  
E - Erosion / Excoriation  
A - Atrophy  
F - Fissure  
S - Scale  
U - Ulcer  
C - Crust  
K - kind-of-hard-to-feel (Induration)  
S - Scar



## Chromoblastomycosis



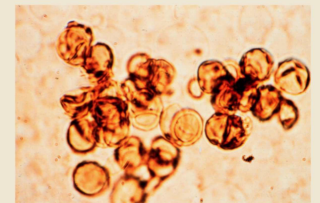
• Caused by a coloured fungus. When the fungus gets traumatically implanted into the skin it becomes blasto (rounded)

• It is a slowly progressive disease of the skin and subcutaneous tissue.

**Diagnosis:** KOH mount of the skin scrapings.



## COPPER PENNY BODIES



ALSO CALLED AS: **MEDLAR BODIES**  
**SCLEROTIC CELL**

SEEN IN CHROMOBLASTOMYCOSIS  
IT'S AN CHRONIC FUNGAL INFECTION

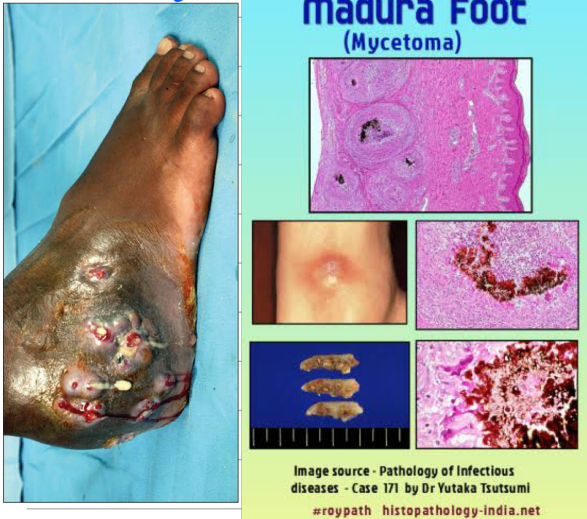
CAUSED BY  
**FONSECAEA PEDROSOI**  
**CLADOPHIALOPHORA**



# Mycetoma : aka Madura foot

- Typically described as a chronic granulomatous disease.
- Following the traumatic implantation of the causative implantation weeks to months later a chronic granulomatous disease characterized by manifestations :

- Swelling of the involved tissue.
- Sinus formation.
- Pus or discharge from the sinuses containing granules



Causative agent	Color of the grains
<b>Eumycetoma</b>	
Modurella mycetomatis	Black
M. grisea	Black
Pseudallescheria boydii	White -yellow
<b>Actinomycetoma</b>	
Actinomadura madurae	White -yellow
A. pelletieri	Red-yellow
Nocardia asteroides	White -yellow
Streptomyces somaliensis	yellow
<b>Botryomycosis</b>	
Staphylococcus species	White
Streptococcus species	White
Escherichia coli	White
proteus species	White
Pseudomonas aeruginosa	White
Actinobacillus lignieresii	yellow

## Annular lesions

are figurate lesions characterized by a ring-like morphology.



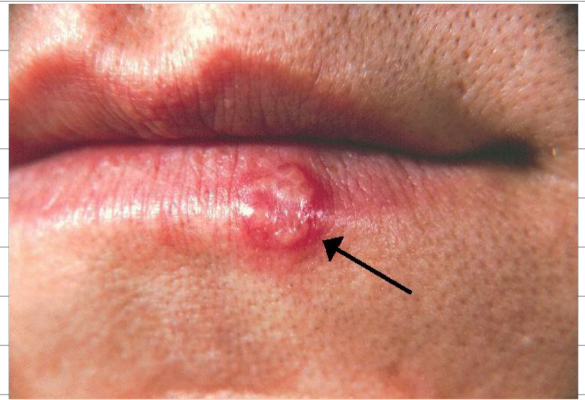
TABLE 1

### Diseases That Can Cause Annular Lesions

Common annular lesions	Uncommon annular lesions
Common diseases:	Common disease:
Erythema migrans	Nummular eczema
Erythema multiforme	Uncommon diseases:
Lichen planus	Immunoglobulin A vasculitis
Pityriasis rosea	Sarcoidosis
Plaque psoriasis	Secondary syphilis
Tinea corporis	
Urticaria	
Uncommon diseases:	
Fixed drug eruption	
Granuloma annulare	
Leprosy	
Subacute cutaneous lupus erythematosus	

## Herpes labialis

- Vesicles on the lips
- m/c reactivation lesion of HSV-1.



## Genital Herpes

m/c manifestation of primary and reactivation of HSV-2.

Diagnosis: Scraping from the base of ulcer  
↳ Tzanck smear → Stain with Methylene blue

PCR: most sensitive test

Rx: Acyclovir / Valacyclovir / Famciclovir for 2 to 3 weeks



## Candidiasis

Candidiasis:

### 1. Oral candidiasis

white plaques

Red plaques

white pseudomembranous candidiasis

Atrophic candidiasis

(false membrane which can be rubbed off).  
AKA oral thrush.



### 2. Candidial intertrigo

Disease: Candidiasis which involves the intertriginous areas.

Lesion: Red macerated plaques (wet and soft to touch), small satellite pustules in the periphery.



### 3. Candidial balanoposthitis

Feature: Red fissuring over prepuce.  
Association: Diabetes mellitus.



Treatment of candidiasis:  
Fluconazole.

# Pemphigus Vulgaris

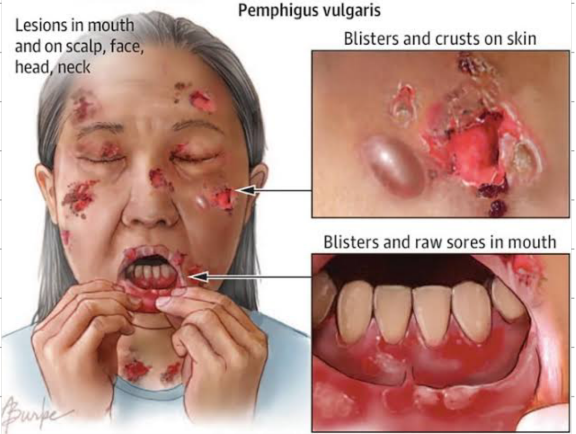
• Age grp: 40-60 years

• Clinical profile:

• Flaccid bullae

Erosions

Tend to extend  
No tendency to heal



## Clinical signs:

1. Nikolskiy Sign:
  - Procedure: Tangential pressure: Applied over skin.
  - Observation: upper layers of epidermis separate from lower layers due to acantholysis.



2. Bulla Spread Sign:
  - Procedure: Pressure over bulla.
  - Observation: Irregular angulated margin.

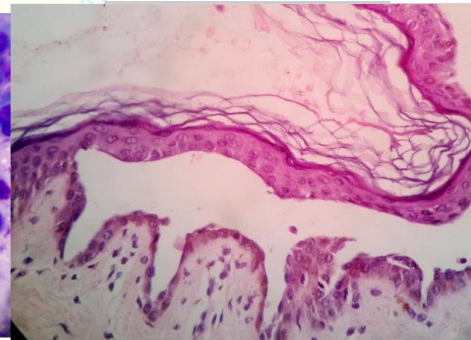
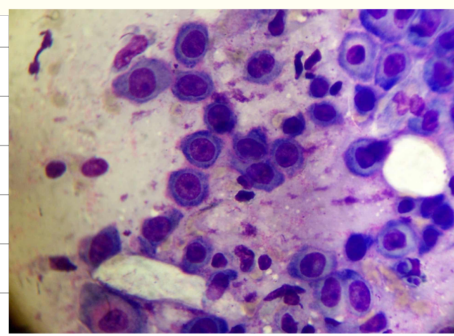
Oral mucosa:  
Oral lesions should not be misdiagnosed as aphthous ulcers.  
Oral pemphigus presents with painful erosions in 90-95% patients.



## Investigations:

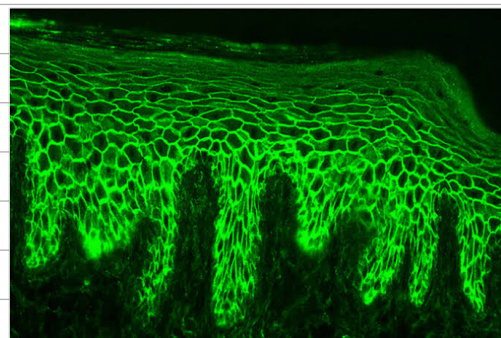
1. Tzanck cells or acantholytic cells.

2. Histopath → • Suprabasal split  
• Acantholytic cells  
• intact stratum basale: Row of tombstone appearance.



## 3. Direct immuno Fluorescence:

- Investigation of choice.
- Direct detection of pathogenic auto antibodies.
- IgG + C3 is deposited in P: vulgaris.
- Deposited in intraepidermal intercellular.
- Pattern: Fish net pattern.



## Rx:

1. High dose Systemic Steroids.
2. Cyclophosphamide:
  - Side effect: Hemorrhagic cystitis.
3. Rituximab:
  - Anti CD 20 used in the management of moderate to severe P. vulgaris.

## *Candidal intertrigo*

### Candidial intertrigo

Disease : Candidiasis which involves the intertriginous areas.

Lesion : Red macerated plaques (wet and soft to touch), small satellite pustules in the periphery.



## *Lupus vulgaris*

Route : Exogenous/ endogenous.

Bacillary status : Paucibacillary  
(good prognosis)

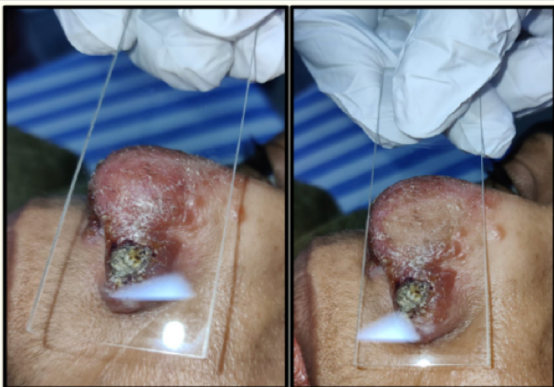
Lesion : Annular infiltrated plaque.

Centre shows atrophy + scar

Periphery shows extension

Sites : Buttocks, face, Extremities.

Diascopy : Apple jelly nodules.



## KIT 1/Grey



### KIT 1

Azithromycin 1 gm single dose +  
Cefixime 400 mg single dose  
For

Urethral discharge, Ano-rectal discharge,  
Cervicitis Syndromes and Asymptomatic infection  
Management

**IMPORTANT**  
NON-COMMERCIAL PRODUCT  
NOT FOR SALE  
TO BE DISPENSED ONLY AT RTI/STI  
CLINICS

## KIT 2/Green



### KIT 2

Secnidazole 1 gm BID dose +  
Fluconazole 150 mg single dose

For  
Vaginal discharge Syndrome

**IMPORTANT**  
NON-COMMERCIAL PRODUCT  
NOT FOR SALE  
TO BE DISPENSED ONLY AT RTI/STI  
CLINICS

## KIT 3/White



### KIT 3

Inj. Benzathine penicillin 2.4 MU (1) +  
Tab. Azithromycin 1 g single dose +  
Disposable syringe 10 ml with 21 gauge  
needle (1) +  
Sterile water 10 ml (1)

For  
GENITAL ULCER DISEASE – Non-  
HERPETIC SYNDROME

**IMPORTANT**  
NON-COMMERCIAL PRODUCT  
NOT FOR SALE  
TO BE DISPENSED ONLY AT RTI/STI  
CLINICS

## KIT 4/Blue



### KIT 4

Doxycycline 100 mg BID for 15 days +  
Azithromycin 1 gm single dose

For  
GENITAL ULCER DISEASE - Non-HERPETIC  
SYNDROME

**IMPORTANT**  
NON-COMMERCIAL PRODUCT  
NOT FOR SALE  
TO BE DISPENSED ONLY AT RTI/STI  
CLINICS

## KIT 5/Red



### KIT 5

ACYCLOVIR 400 MG ORALLY TID FOR 7  
DAYS

For  
GENITAL ULCER DISEASE - HERPETIC  
(GUD-HERPETIC) SYNDROME

**IMPORTANT**  
NON-COMMERCIAL PRODUCT  
NOT FOR SALE  
TO BE DISPENSED ONLY AT RTI/STI  
CLINICS

## Kit 7/Black



### KIT 7

Doxycycline 100 mg BID for 21 days +  
Azithromycin 1 gm single dose

For  
Inguinal Bubo Syndrome

**IMPORTANT**  
NON-COMMERCIAL PRODUCT  
NOT FOR SALE  
TO BE DISPENSED ONLY AT RTI/STI  
CLINICS

# Genital warts

Anogenital warts :

AKA condyloma acuminata (acuminata = pointed).



Etiology

HPV 6, 11

HPV 16, 18

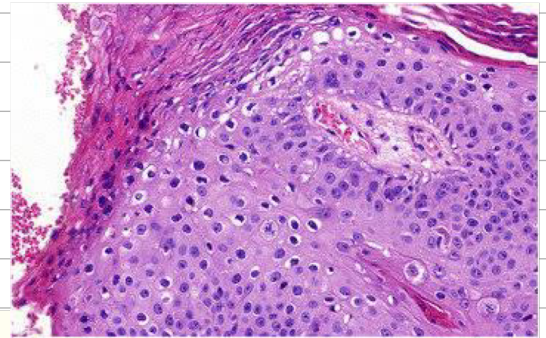
(low risk HPV, 90% cases)

(High risk HPV, 10% cases)

C/F: Asymptomatic, fleshy, pink pointed papules and plaques.  
 • Cauliflower like lesions.

Histopath: Koilocyte (HPV infected cell)

- Squamous epithelial cell
- Central Hyperchromatic nucleus
- Perinuclear halo.



Treatment

Non pregnant

Pregnant

Podophyllin

Imiquimod

Cryotherapy

TCA 70-80%

Targets :  
mitotic  
spindle

TLR-7 agonist,  
acts as immune  
response  
modifier

By Liquid N<sub>a</sub>  
at -196°C  
(TOC)

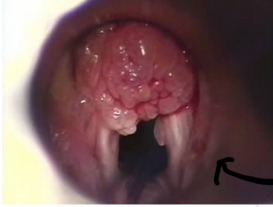
Chemical  
coagulation of  
the proteins

# Trichomoniasis Rx

Drug	Dose	Duration	+/- Notes
<b>1st choice options</b>			
Metronidazole	2g PO	Single dose	<ul style="list-style-type: none"> <li>• Avoid 2g dose metronidazole in pregnancy, breast-feeding and elderly.</li> <li>• Avoid alcohol during metronidazole treatment and for 48 hours afterwards (disulfiram reaction).</li> </ul>
<b>OR</b>			
Metronidazole	400mg every 12 hours PO	5-7 days	

## Human Papilloma Virus (HPV) and Associated Lesions

HPV serotype	Lesions
HPV 1,2,3,4	<ul style="list-style-type: none"> <li>Common warts (verruca vulgaris, plantar wart)</li> </ul>
HPV 6 and 11	<ul style="list-style-type: none"> <li>Condyloma acuminatum (genital warts)</li> <li>Sinonasal papilloma</li> <li>Squamous papilloma of the larynx</li> </ul>
HPV 16	<ul style="list-style-type: none"> <li>LSIL, HSIL, carcinoma cervix</li> <li>Vulval carcinoma</li> <li>Bowen disease (Carcinoma in situ of male external genitalia)</li> <li>Oropharyngeal squamous cell carcinoma</li> </ul>
HPV 18	<ul style="list-style-type: none"> <li>LSIL, HSIL, Carcinoma of the cervix</li> <li>Carcinoma of the anogenital region</li> </ul>
HPV 5 and 8	<ul style="list-style-type: none"> <li>Squamous cell carcinoma of the skin in epidermodysplasia verruciformis patients</li> </ul>



**Acanthosis nigricans** → Hyperpigmented velvety plaques.

• Sites: Neck, axilla.

• m/c/c: Obesity

• Mechanism: in obesity → increased like growth factor → Stimulate epidermal keratinocytes.

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



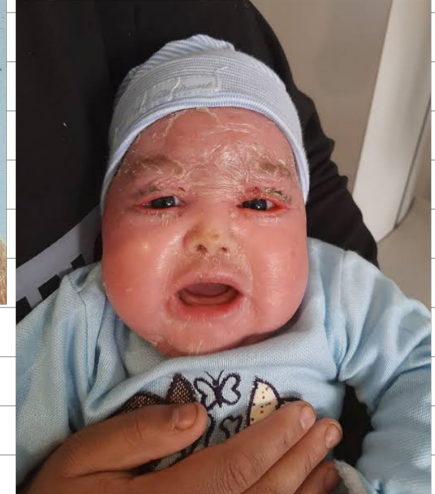
## Lamellar Ichthyosis

- Onset at birth.
- Autosomal Recessive
- Defect in Epidermal transglutaminase 1.

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

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

- Rx: Topical : Emollients  
Systemic : Retinoids (Acitretin).



## Melanoma

- Risk factors:
- UV radiation
  - White population.
  - Familial atypical mole melanoma Syndrome: Also predispose patient to (pancreatic adenocarcinoma)

- Biopsy : Melanin pigment seen.  
• Important prognostic factor : depth of invasion.

- Types :
- Superficial spreading : m/c type (seen in : young, sun exposed areas)
  - m/c melanoma developing in pre-existing mole.

- Lentigo maligna : aka Hutchinson's melanotic freckle.

• Insitu : Hence best Prognosis.

• Seen in Elderly.

- Acral melanoma : In dark skin patients.  
Seen in palms, soles.

Rapid Vertical phase : hence aggressive in nature.

- Nodular melanoma : Appear as Nodule.

Rapid vertical phase.

Worst prognosis

• **Desmoplastic melanoma** : Head and neck region.

• **Perineural invasion** present : Also seen in adenoid cystic carcinoma in salivary glands.

• **Increased local recurrence rate.**

• **Subungual melanoma** :

- Melanoma beneath nail bed.

- In it **superficial spreading type** is more common than acral melanoma beneath the nail bed.

**Changes in naevus** :  
Asymmetry  
Border - irregular  
Colour change  
Diameter > 6mm  
Evolution

**Immunohistochemical marker** : S-100

HMB-45

MELAN-A

**2 Staging classification** : TNM staging

• Clarke and Breslow staging.

