3 Parts of Skin

1> Epidermis → ← DEJ (Dermo-epidermal Jn)

2) Derma → ← Lobules, septa

3) Fat (panniculus)

epidermal Rete Ridge

← dermal papilla

EPIDERMIS

4 Layers

1> STRATUM CORNEUM

Thickest Layer

Max. Keratin

Corneum → means Keratin.

2> STRATUM GRANULOSUM

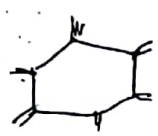
↳ granule

↓

Keratohyaline granule

Thinnest Layer

3> STRATUM SPINOSUM (SS)



← Desmosome

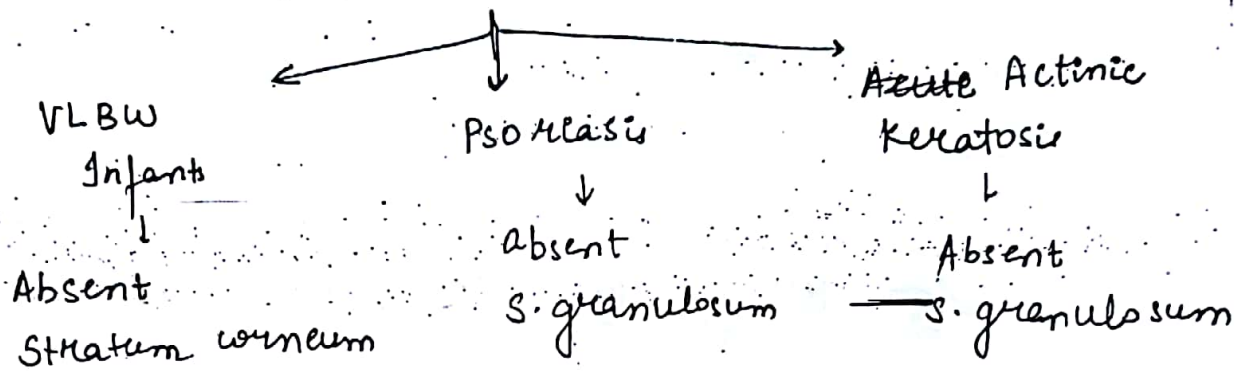
Spinous projection coming out

Max. Desmosome are present in SS AIIMS

4) BASAL LAYER

For Division

SITUATIONS For 3 LAYERS



5 LAYERS → palm + sole

- S corneum
- S. Lucidum ⇒ non nucleated layer
  - ? compression
  - ? artefact
- S. granulosum
- S. spinosum
- S. Basal

pressure (trauma)

Keratinisation of epidermal cells

- Sc → 1000 mg
- Sg → 100 mg
- Ss → 10 mg Keratin
- S. Basal → 1 mg Keratin

lots of pressure

Sc - 1000 mg Keratin (thick Sc)

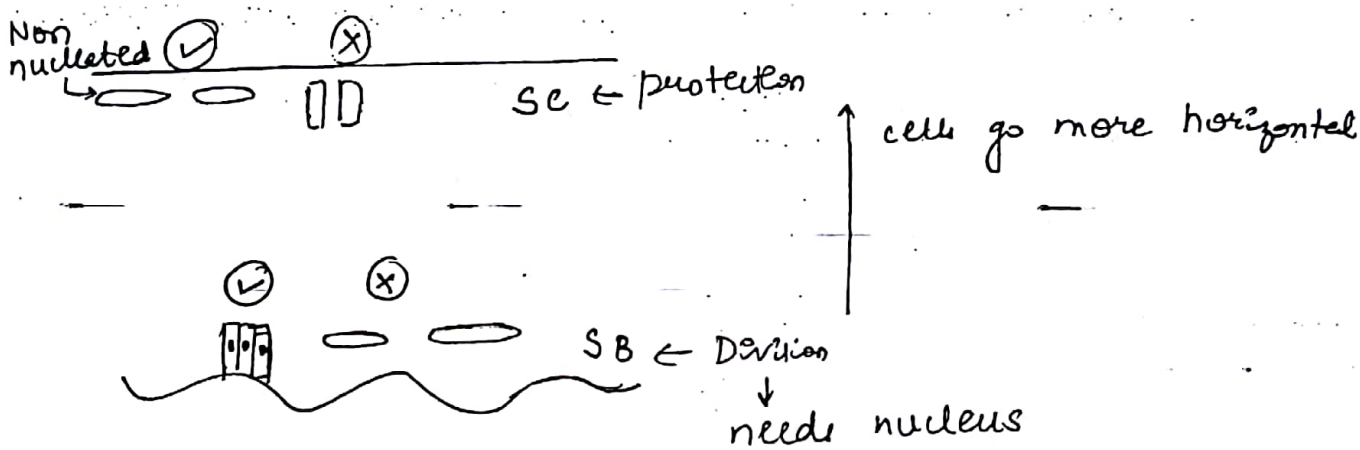
↓

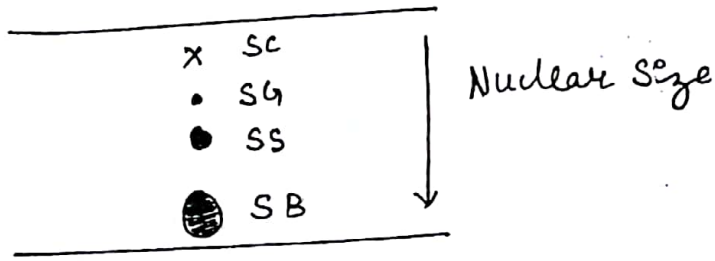
Hyperkeratosis

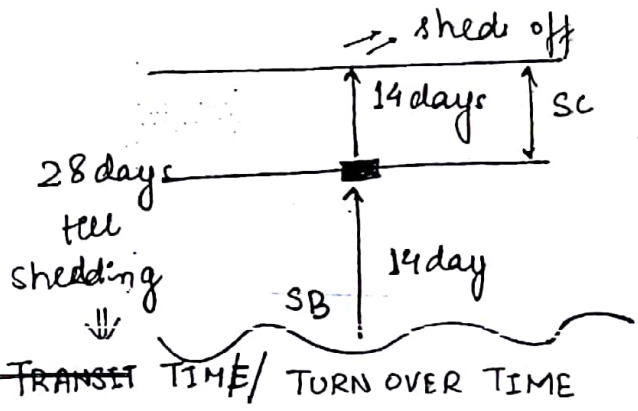
Thick SG ⇒ Hyper granulosis
↓
Seen in LICHEN PLANUS.

Thick SS ⇒ Acanthosis
↓
Seen in LICHEN PLANUS
PSORIASIS
ECZEMA

SS + SB = Malphigian Layer

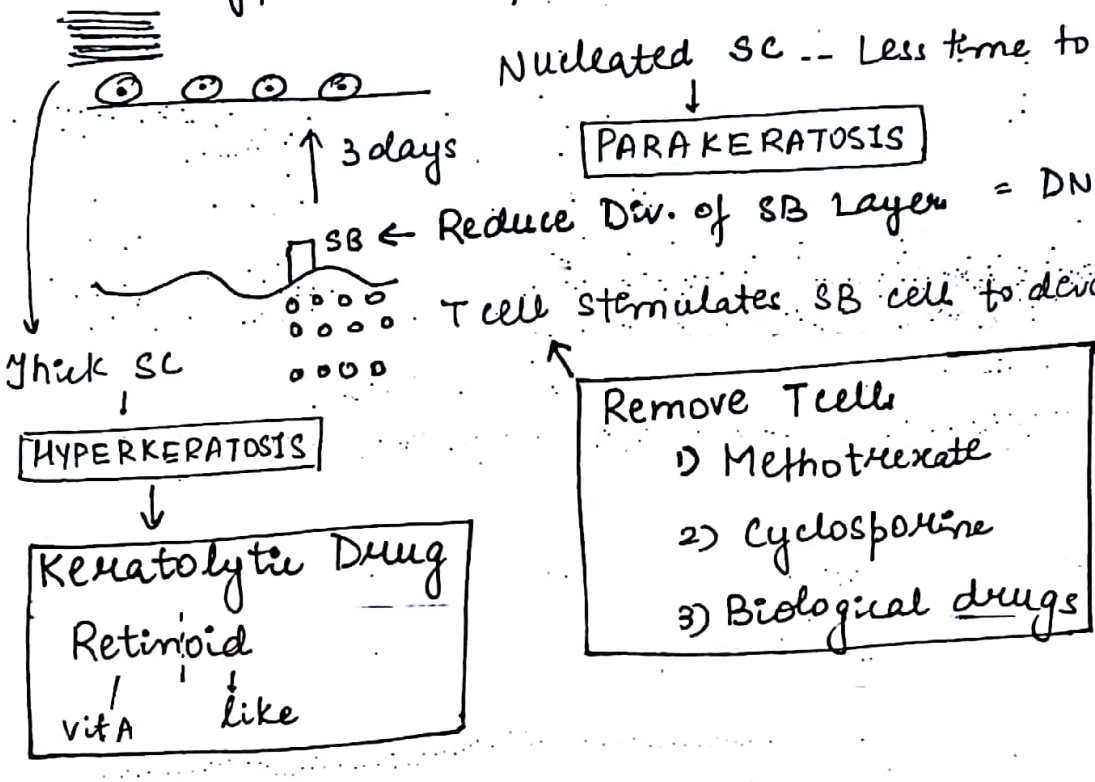






Psoriasis

Hyperdivision of SB Layer.



EPIDERMAL CELLS

1) 95% ⇒ has keratin, cell are called

KERATINOCYTES

- Func^{n} :- 1) Protection
  2) Immunological Role → secretion of cytokines → Innate Immunity
- Derived from ectoderm.

2> Rest 5%

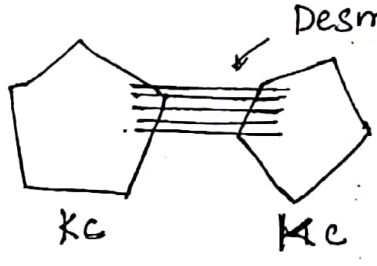
MELANOCYTES (ML)
- Make melanin
- UV light protection
- derived from Neural crest

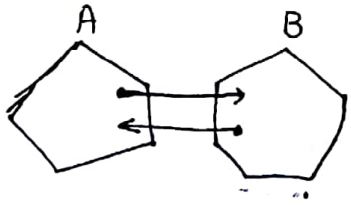
LANGERHANS CELL (LC)
- Ag presenting cell
- from Bone marrow

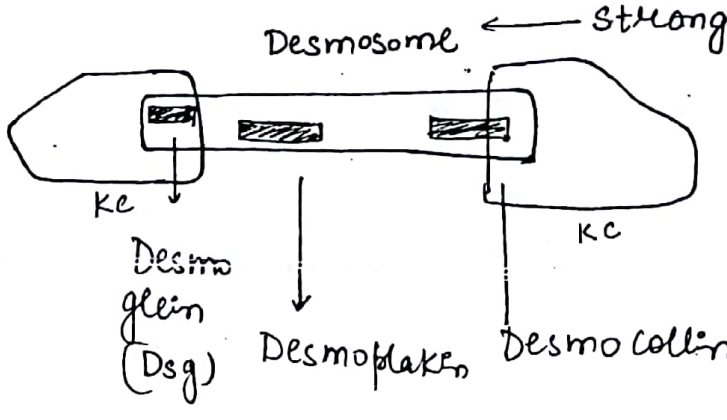
MERKEL DISC
- touch Receptors
- either from ectoderm or Neural crest

Keratinocyte : Merkel's cell have Desmosome on their surface.

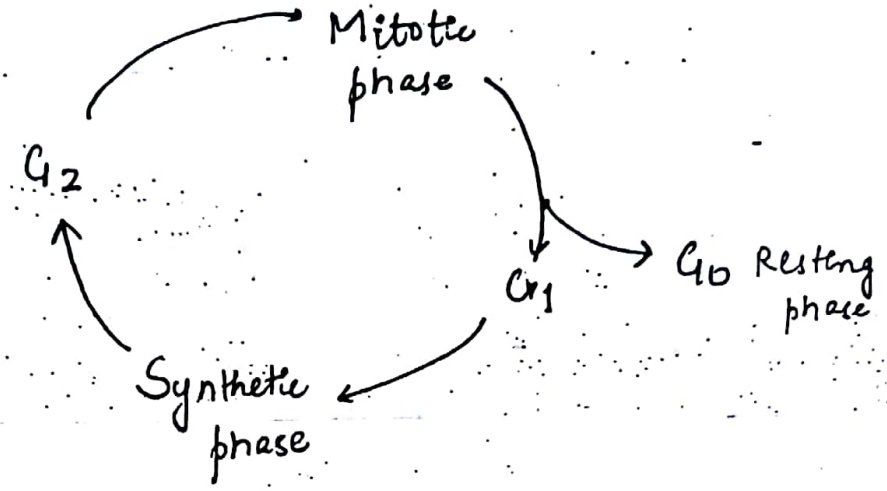
Melanocyte : Langerhans cell do not have Desmosome







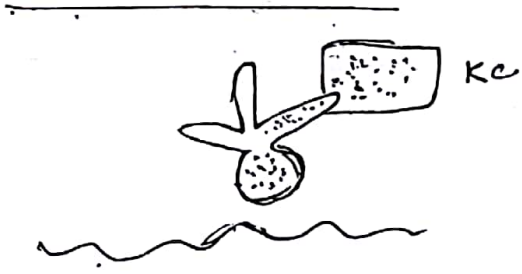
Keratinocyte cell cycle Time = 311 hrs



In Psoriasis ⇒ cell cycle time is ↓ to 36 hours.

MELANOCYTES

Melanocytes are Dendritic cells
They make melanin & transports it via dendrite processes into KC [Epidermal melanin unit = 1:36]



Indians have Type 5 skin (Brown skin)
Less melanin (fair skin) or No melanin (albinism)

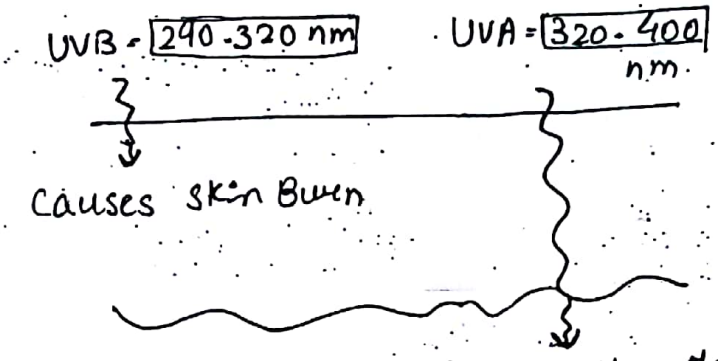
↓

Chronic DNA damage

↓

Premalignant → Malignancy (SCC, BCC, Melanoma)

Photo carcinogenesis
↓
Chr. sun damage
[cumulative.. or light]



Fairer skin
↳ More Burning

Darker skin
↳ More Tanning

PREMALIGNANT SKIN DISEASES

(A) SUN EXPOSURE

eg. ACTINIC KERATOSIS
↓
means sun
↓
Lead to SCC

(B) May or Maynot be SUN Induced

1) Bowen's Disease -
SCC - In - Situ
↓
Restricted to epidermis

~~2) Oral leukoplakia~~

3) Oral erythroplakia

4) Oral submucous fibrosis
5) Oral ulcerative lichen planus

MALIGNANT SKIN DISEASES

1) **BCC**

H/C Skin Cancer
H/C type of BCC ⇒ NODULO-ULCERATIVE (Rodent Ulcer)

Locally aggressive skin cancer
Metastasis is rare

C/F :-

1) Nodules ± ulcerate on sun-exposed sites
2) Ulcers have
   - Beaded
   - Rolled
   - Pearly

   } edge
3) Telangectasia on its surface

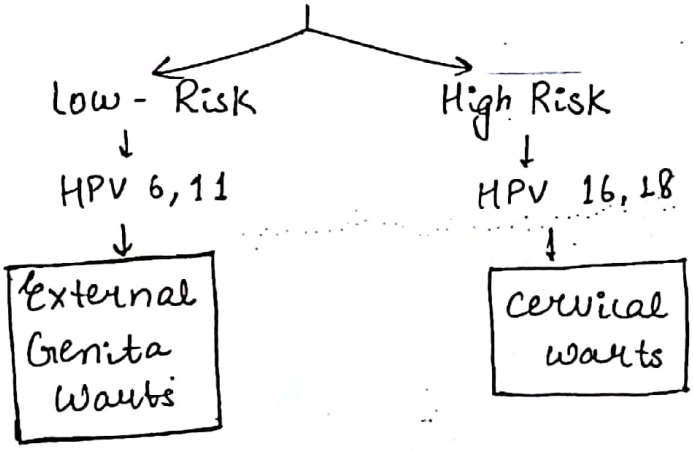
Rx :- Moh's microsurgery
↓
pathology controlled dissec^{n}.

(II) SCC

H/c in organ transplant pts.
↓
due to immunosuppression.

ETIOLOGY

1) Sun
2) Immunocompromised
3) HPV - DNA virus
   ↳ onchogenic virus
   Integrates its DNA into KC gene & divides along ē it.



C/F

a) Cauliflower masses

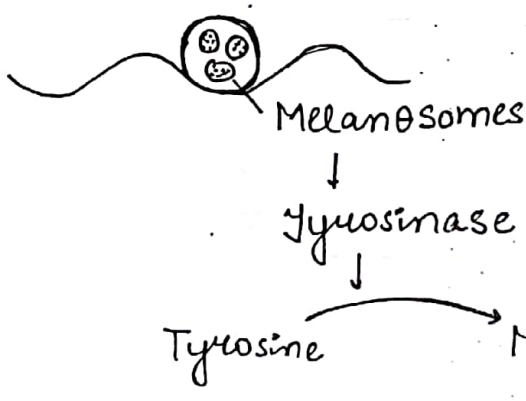
b) Hyperkeratotic plaques
c) ulcers
d) metastasis

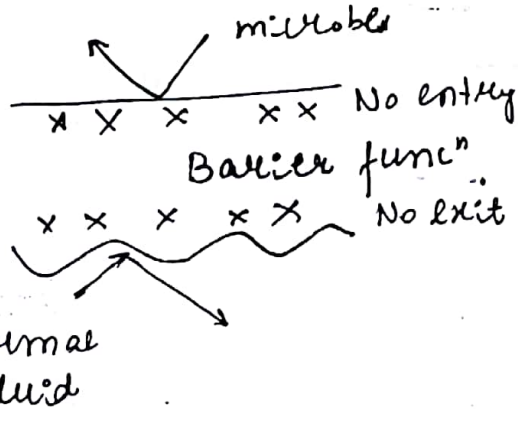
(III) Melanoma - Later.

* COLOUR OF SKIN depends on :-

1) Melanin production
2) Transfer of melanin to KC
3) No. of melanosomes

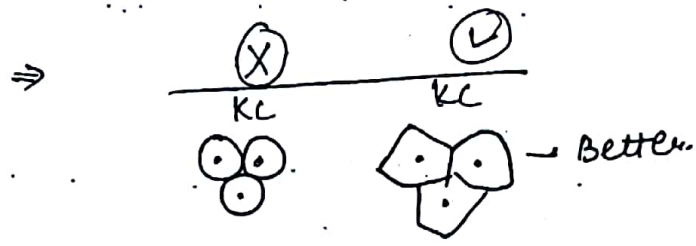
Not the no. of melanocytes

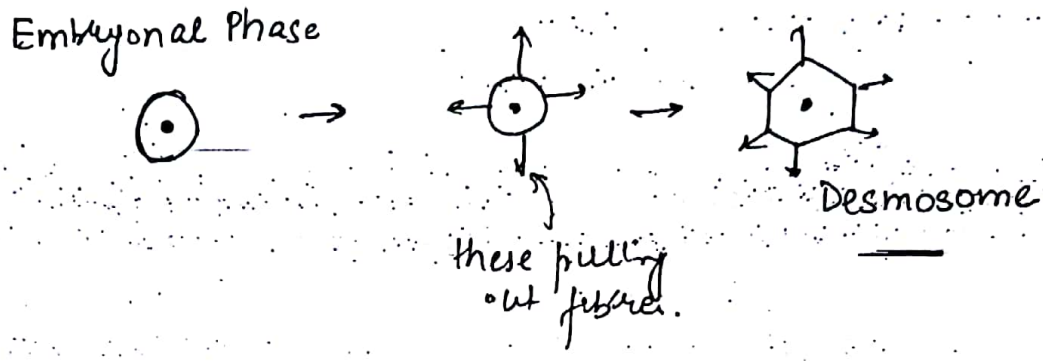




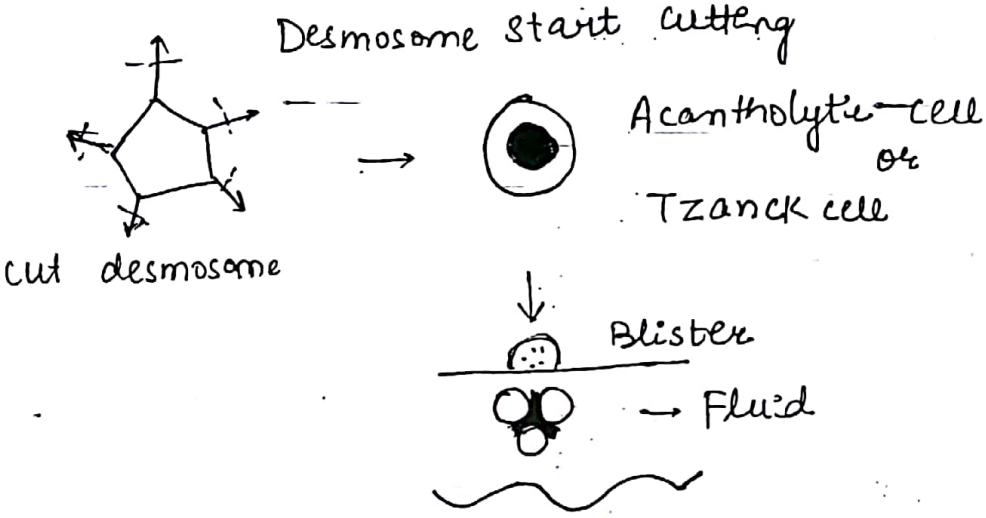
→ Gets altered where epidermis is damaged

a) Burn
b) wound.



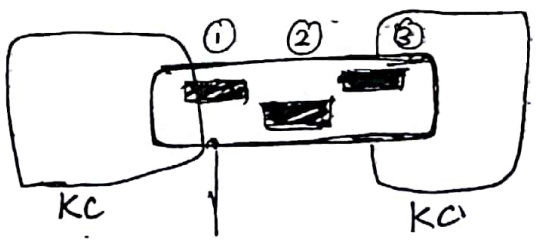


PEMPHIGUS



"pemphis" means Blister ⇒ Intercellular Disease

Y Desmosomes BREAK ?



IgG Ab formed against Desmoglein.

↓

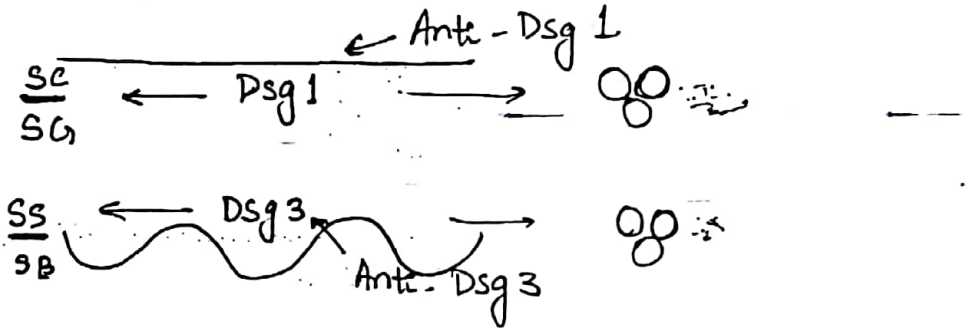
Weak Desmosome

TARGET → Desmoglein
Ab → Ante-Desmoglein (IgG)] IgG PEMPHIGUS

TARGET → Desmocollin
Ab → Ante-Desmocollin (IgA)] IgA PEMPHIGUS

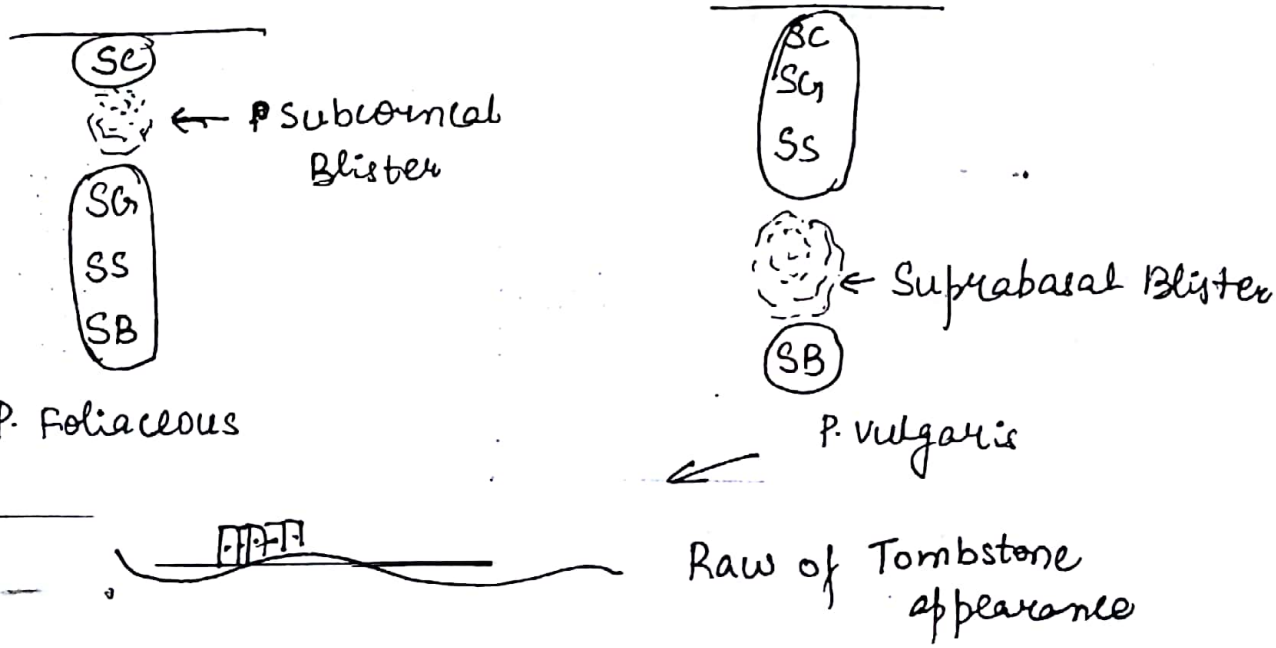
TARGET → Desmoplakin
Ab → Ante-Desmoplakin (IgA/IgG/IgM)] Paraneoplastic Pemphigus

IgG PEMPHIGUS



Subcorneal Blister = P. foliaceous

Suprabasal Blister - P. Vulgaris - common



INTERCELLULAR CEMENT

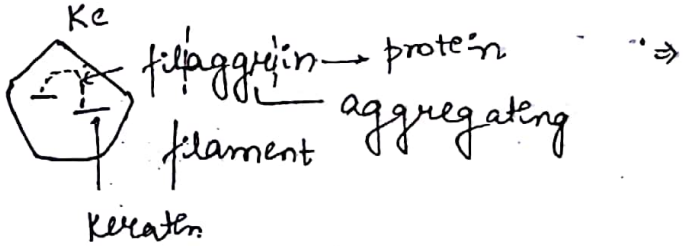
← Intercellular cement (Lipids)
like a glue

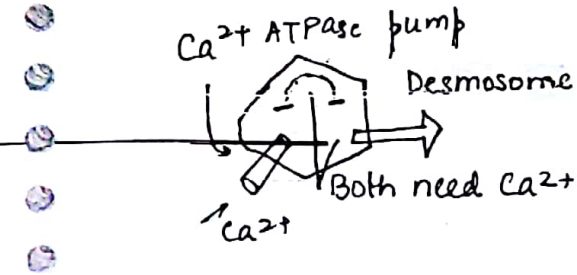
make the glue
Lamellar Body (Max. in granular layer)
Odland Body

ECZEMA/DERMATITIS

Desmosomes are (N)
Oedema or H_2O is more in Pemphigus
Oozing is a sign of acute eczema

CONGENITAL EPIDERMAL BLISTERS





DARIER's DISEASE (DD) & HAILEY-HAILEY DISEASE (HHD)

↓

mutation in Ca-ATPase pump ∴ birth

↓

weak KC

circular KC (Acantholytic cell)

DD

Keratosis Follicularis

↓ correct → there is hyperkeratosis

↓ Incorrect word: → No follicular involvement

Circular weak KC

Compensatory ↑ of keratin synthesis

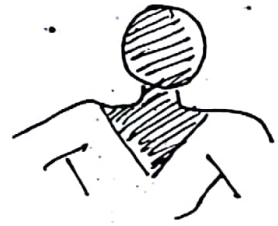
Circular strong KC

C/F :-

Hyperkeratotic spiny sharp papules on skin

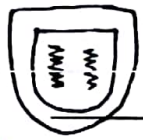
↓

More in seborrhoeic areas



NAILS :- 1) V shaped nicking of nails

2) ~~Red/white~~ longitudinal nail lines



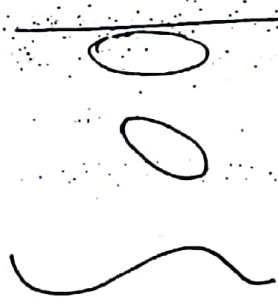
PALM- Palmar Pit

R_x = Retinoids.

DARIER'S DISEASE

Dyskeratotic cells in
S.C = CORPS GRAIN

Dyskeratotic cells in
SG, CORPS ROND



SC → 100000
SG → 10000
SS → 1000
SB → 1mg

Hyperkeratosis

Premature Keratinisation = Dyskeratosis

OTHER CAUSES FOR DYSKERATOSIS

1) Premalignant skin Disease
2) Malignant Skin Disease

HHD :- No compensatory hyperkeratosis
Hence presents ∴ Blisters

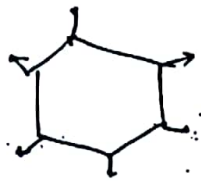
- GENE MUTATION :-

HHD
Ca ATPase 2C1 gene

Darier's
Ca ATPase 2A2 gene

EPIDERMOLYSIS BULLOSA SIMPLEX.

Trauma induced Blisters



No Keratin 5,14 since Birth.

(N) Desmosome

Fragile basal keratenocytes.

↓ TRAUMA

No acantholysis

Blister in basal layer

3 TYPES OF EPIDERMOLYSIS BULLOSA

EB SIMPLEX	EB JUNCTIONAL	EB DYSTROPHICA
S. Basal.	On DEJ"	in Dermis.

DIRECT IMMUNO FLOURESCENCE

Picks up Antibodies in Blistering Disorders

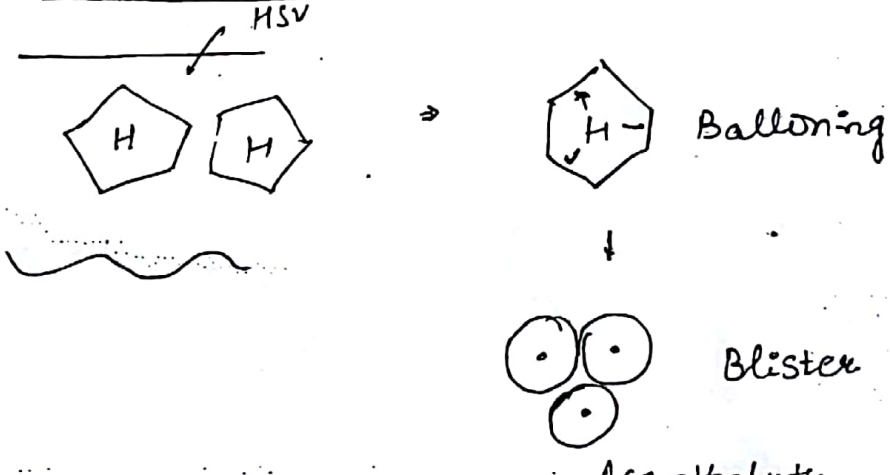
Pemphigus ⟶ DIF ⊕

DD/ HHD/ EBS | EBJ/ EBD ⟶ DIF ⊖

ACANTHOLYSIS

1° (Pulling Problem)	2° (Pushing Problem)
1) Pemphigus	1) HSV - Herpes Simplex
2) Dariers Disease	
3) HHD	
4) Bullous Impetigo	
5) Staphylococcal scalded skin Syndrome (SSSS)	

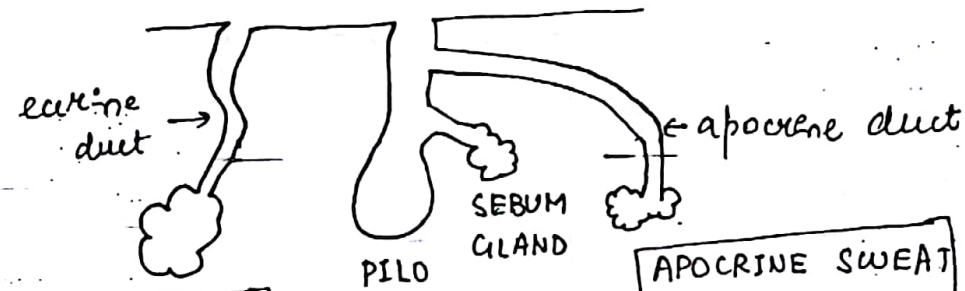
2° ACANTHOLYSIS



ACANTHOLYTIC CELL

1) Circular
2) Large nucleus
3) Narrow cytoplasm
4) Prominent nucleoli

APPENDAGE / ADNEXA



ECCRINE SWEAT GLAND

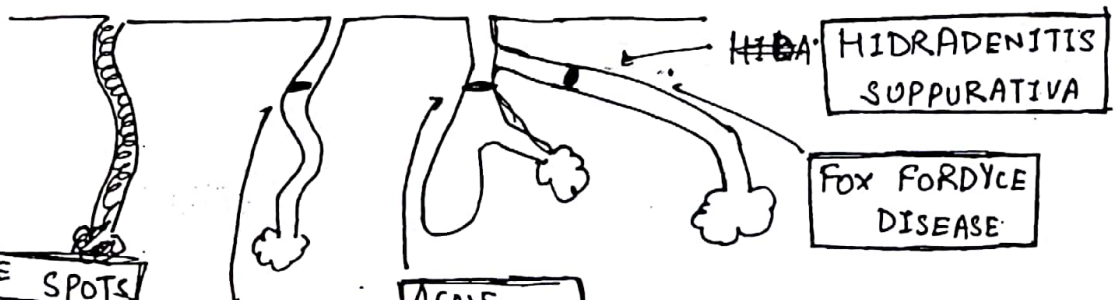
→ Everywhere else

Supplied by Cholinergic Nerves

APOCRINE SWEAT GLAND

→ Axilla
→ Perineum
→ Areola

Supplied by Adrenergic Nerves



Ectopic sebaceous GLANDS

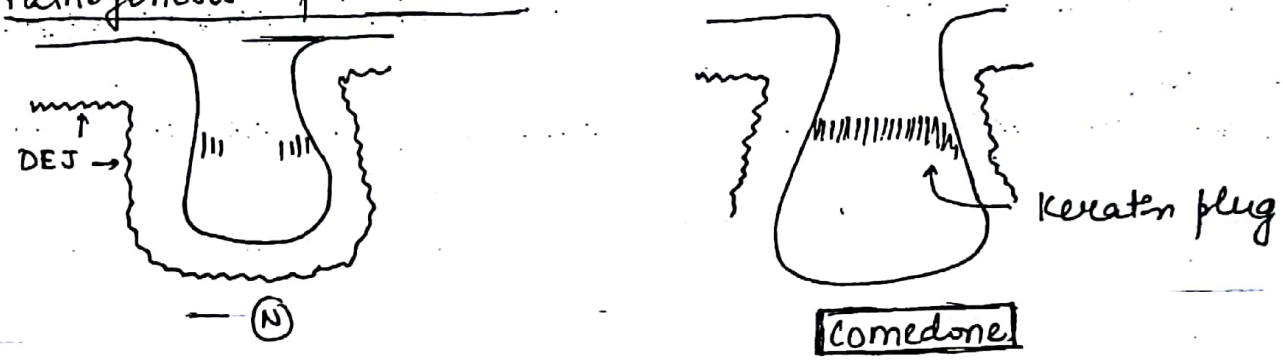
HILARIA (occlusion due to S. epidermidis)

① ACNE VULGARIS

PATHOGENESIS:-

1) Keratenisation of follicular Epithelium
2) Proliferation of propionibacterium acnes
3) ↑ sebum produc" in seborrhoeic area
4) Dermal inflammation.

Pathogenesis of comedone :-



(Hyperkeratenisation of follicular epithelium)

STAGE - 1 ACNE

2 TYPES

BLACK COMEDONE
(open comedone)
Black Head

WHITE COMEDONE
(closed)
White head

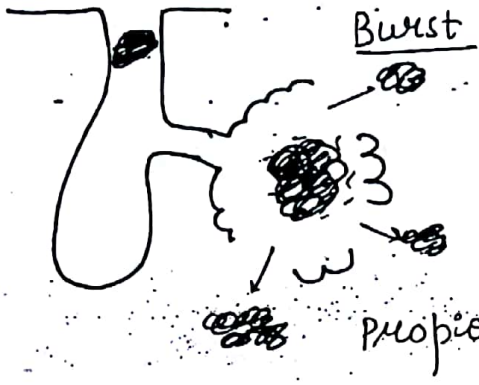
℞ = Topical Retinoids (Adapalene/ Tretinoin)

S/E → skin irritation
→ ~~photosensitivity~~
↳ hence applied at night

STAGE-2 = Inflammation

↓

[Red
Elevation] (Papule)
Pain



Stage 2 = (Stage 1 + papule)

R_x = Topical Retinoids + Topical Antibiotics
- clindamycin ✓
- clarithromycin ✓
- Dapsone. ✓

STAGE-3

Stage 2 + Pustule.

R_x = Topical retinoids + oral Doxy
Azithromycin
Minocycline → Most effective

More S/E
1) Hepatotoxic
2) Bluish skin pigment on long term use
- nail
- Acne scars

~~STAGE~~ / DRUG RESISTANT ACNE

1) Topical Benzoyl Perboxide
- Safe in ♀.

↓ ↓
release nascent [O] on skin surface
↓
[Bactericidal]

⇒ Topical Azelaic Acid
- Bactericidal
- Tyrosinase Inhibitor → Reduces post acne pigmentation.

STAGE 4 (stage 3 + Nodules / cysts)

Acne has polymorphic Lesions

Rx = Oral Retinoids
- Acitretin = Keratolytic → used in psoriasis
- Isotretinoin = Keratolytic + Sebolytic (AIIMS, NOV 15)

STAGE 5 (ACNE CONGLOBATA)

STAGE 4 + Severe Inflammation
- discharging sinuses
- Fever

Chest / back

Rx = Oral isotretinoin + anti inflammatory (steroids)

Recalcitrant Pustular Acne → Isotretinoin
↑ Not responding

ACNEIFORM ERUPTIONS :-

Drug induced Acne

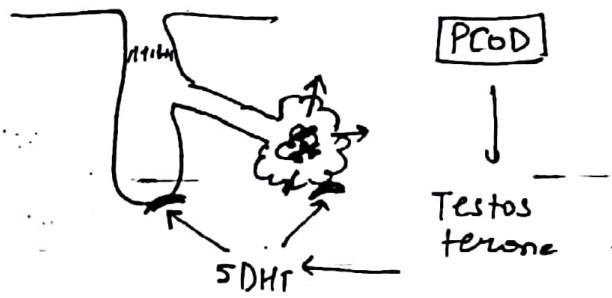
Monomorphic Lesions on chest & neck. (papules)

Causes :-
1) Oral & topical steroids ✓
2) Anabolic steroids ✓
3) INH, Rifampicin ✓
4) Phenytoin, Phenobarbitone ✓

HORMONAL ACNE

Eg. PCOD
presents as
- Acne
- Androgenetic Alopecia on scalp
- Hirsutism on face
- Irregular menses

R_x = Androgen (R) Blocker
- Cyproterone acetate
- Drosperinone



H/C SIDE EFFECT of Isotretinoin
= DRY LIPS (cheilitis)

Other S/E :-

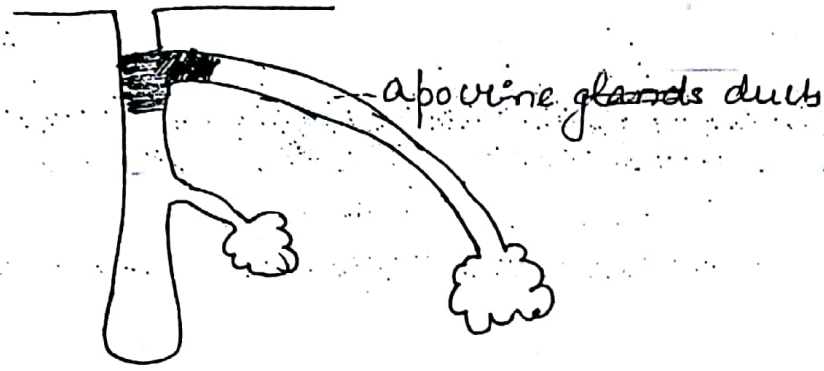
1) Hyperlipidemia
2) ♀ category 'X'
3) Period of contraception after stopping
   Isotretinoin → 1 month.
   Acitretin → 2 month.

② HIDRADENITIS SUPPURATIVA

water → sweat

gland → inflammation

pus → Discharge sinus



Keratin obstruction of apocrine ducts extending into hair follicles

↓

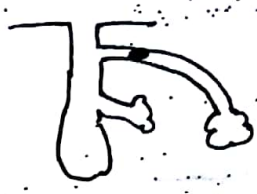
Lesions similar to Acne but in APOCRINE Areas. hence called INVERSE ACNE

2° Infn ⊃ S. Aureus ⇒ • Creates Abscesses +
• Draining sinuses in apocrine areas.

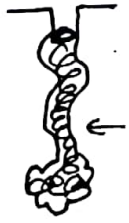
R_x = Retinoids + Broad spectrum oral Antibiotics, Surgical debridement of pus.

(III) FOX FORDYCE DISEASE

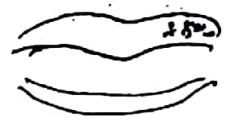
- Lesser keratin obstruction as compared to hidradenitis
- Only Inflammatory papules in apocrine areas
- No comedons seen.



(IV) FORDYCE SPOTS



← ectopic ~~sebaceous~~ sweat gland



Ectopic sebaceous gland on upper lip or Buccal mucosa
asymptomatic
No Rx required

LANGERHANS CELL

Derived from Bone Marrow
Picks up Ag in epidermis
↓
Sends to Local L.N. for processing

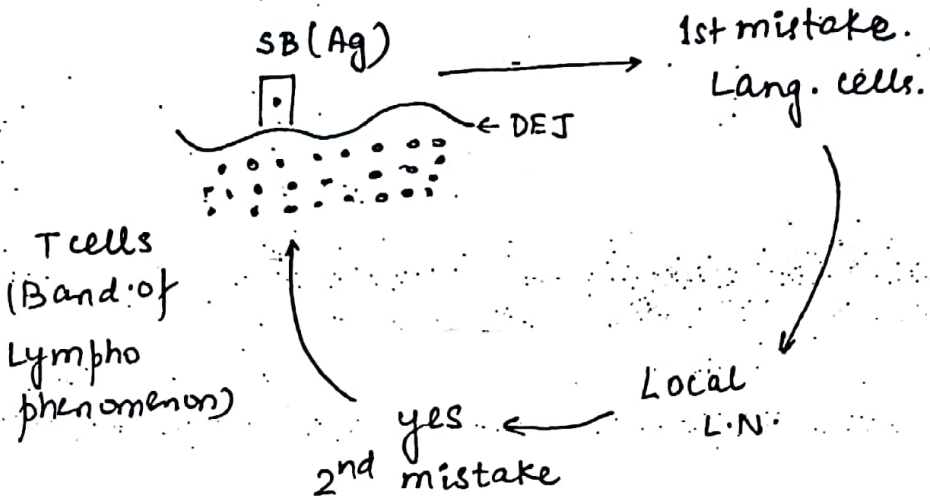
TH_1 response → (T cell)

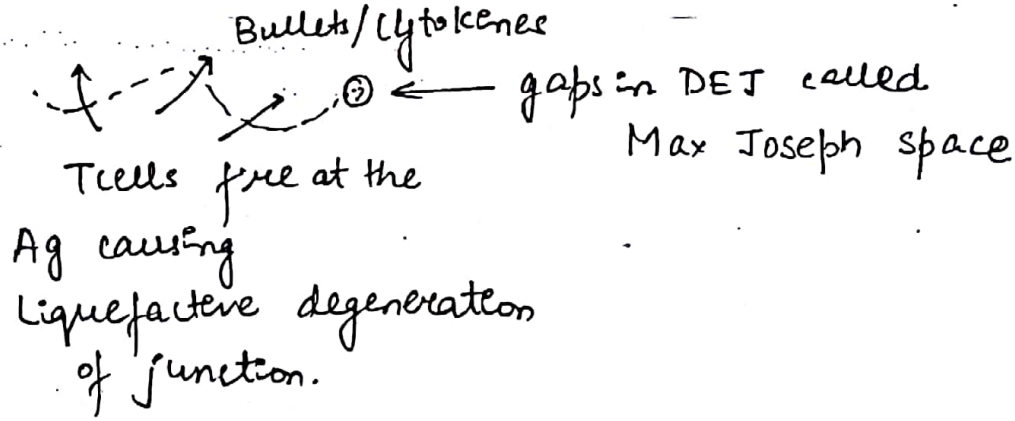
TH_2 response → (B cell)

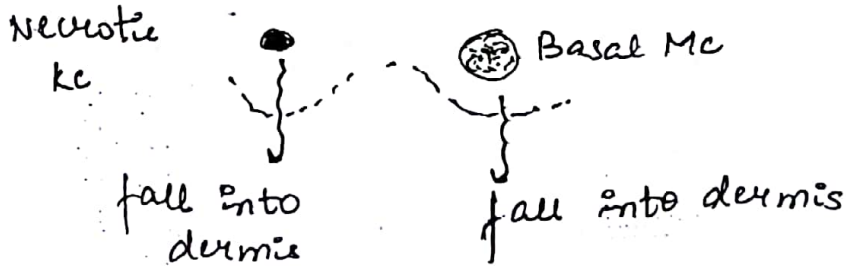
HPV puts Langerhans cell to sleep & creates infection in epidermis.

Hence Rx for warts is Langerhans cell stimulators [Topical Imiquimod]

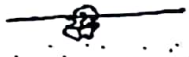
LICHEN PLANUS







Civatte bodies
or
Colloid bodies
or
cytoid bodies)

Melanin colour	Clinical skin colour
	BLACK
	BROWN
	PURPLE
	BLUE / GREY

Other changes seen in LP:-

→ Hyperkeratosis
→ Hypergranulosis
→ Pigment incontinance
→ Acanthosis (thickened spinous layer)

Band of Lymphocyte + Basal cell degeneration
= Interface Dermatitis

✓ Basal cell degeneration. (Most distinctive histology feature)

OTHER CAUSES OF INTERFACE DERMATITIS

→ Fixed Drug Eruption
→ Erythema multiforme
→ Graft vs Host Disease

MELASMA

Disorder of Pigmentation (Hyperfunctioning melanocytes)

TRIGGER FACTORS:-

1) Sun exposure
2) OCPs
3) ♀ (Chloasma)

♀ > ♂

C/F:-

1) Brown hyperpigmented patches at cheek & nose ± photosensitivity.
2) Chronic disease

Rx:-

1) Sunscreen
2) Tyrosinase Inhibitors eg. 1) Kojic acid
   2) Hydroquinone (2-4%) [gold Std]
   3) Azelaic acid
   4) Arbutin
3) Topical Retinoid
4) Topical Steroid (melanocyte inhibitors)

KLIGMAN REGIMEN

Topical hydroquinone
+
Topical Retinoid
+
Topical Steroid

SLE

Presents are persistent erythema on Malar area
i. photosensitivity
Rash is in a butterfly pattern.

ROSACEA

⇒ TRIGGER FACTOR
1) Sun
2) Alcohol
3) Hot spicy food
4) Emotional upset
5) Demodex mite
6) Exercise

⇒ STAGES
1) Telangectasia • Intermittent flushing (episodic flushing)
2) Papule • Pustules
3) Rhinophyma (Potato Nose)

⇒ Rx: avoid triggers
Topical steroids (C/I) ⇒ beco they cause telangectasea

Orally Doxycycline - Doc
↳ acts by anti-inflammatory effect

Topical Metronidazole or Clindamycin
(anti-inflammatory drugs)

ACANTHOSIS NIGRICANS → misnomer

Black velvety areas in flexures

PATHOLOGY :-

Insulin Resistance

↓

ILGF (Insulin like growth factor)

↓

thick skin.

CAUSES :-

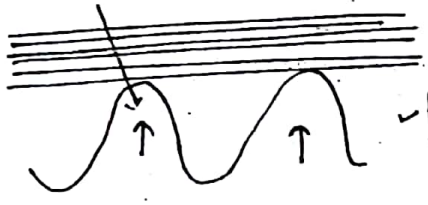
1) obesity Q
2) DM
3) PCOD
4) Drugs (systemic steroids, nicotinic acid)
5) Gastric Adeno Ca → Harest cause Q.

Not a melanin disease

ON BIOPSY :-

PAPILLOMATOSIS Q

HYPERKERATOSIS Q.



- papilla touches the sc

CLASSIFICATION OF SKIN LESIONS

1° 2°

PRIMARY LESIONS

	Less than 0.5cm	0.5cm – 1cm	More than 1cm
FLAT	MACULE		PATCH
PUS	PUSTULE		PUSTULE
FLUID	VESICLE		BULLA
SOLID ELEVATION	PAPULE		PLAQUE ↔ 1 / NODULE ↕

LEVEL OF BLISTERS

EPIDERMAL	DERMAL / DEJ"
Flaccid	Tense
Ruptures by itself	Doesn't rupture by itself
Doesn't heal ī scarring	Heals ī scarring, milia
Heals ī hyperpigmentation	Heals ī hypopigmentation

SECONDARY LESIONS

1) SCALE
   - visible exfoliation of skin
   - SILVERY SCALES :- Psoriasis
   - POWDERY » :- Pityriasis Versicolor
   - COLLARETTE » :- Pityriasis Rosea (small) → Hanging Curtain sign

P LEAF LIKE SCALES - Pemphigus follaceous

More fluid ○ PF → Raw always ruptures

Less fluid ○ PV

↓

ruptures, fluid released forms scale on raw surface

PF

Dsg I → Seborrhoeic areas

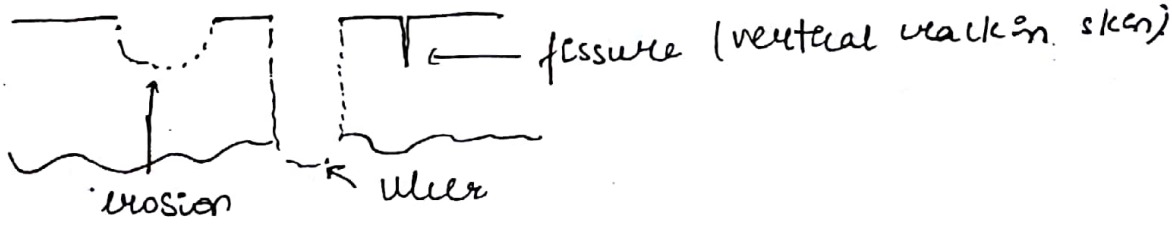
→ mucosa → absent

2> CRUST :-

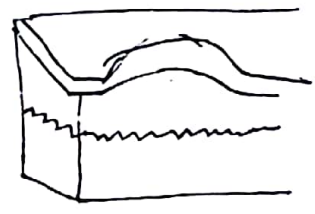
Dried exudate

Usually Black in colour

3> EROSION , ULCER , FISSURE :-



4> LICHEN SIMPLX CHRONICUS (Lichenification)



Thickening

Increased Skin marking ← △ → Hyperpigmentation

S/o Chronic itchy skin Disease

HAIRS

HAIRS 2 TYPES

- TERMINAL → Thick
- VELLUS → Thin

Terminal —miniaturisation→ vellus

Called as

ANDROGENETIC ALOPECIA (AGA)

MALE AGA

- → Starts ⇒ hair Line Recession
- → followed by frontal + vertex balding
- → Lateral + Post Density Ⓝ

℞ = 5% Minoxidil
oral finasteride 1mg/day

Male AGA GRADED from 1 ⟶ 7
Least most

Norwood-Hamilton Grading Scale

FEMALE AGA

No hair Line recession
widening of central parting

℞ = 2% Minoxidil
Androgen Ⓡ Blocker

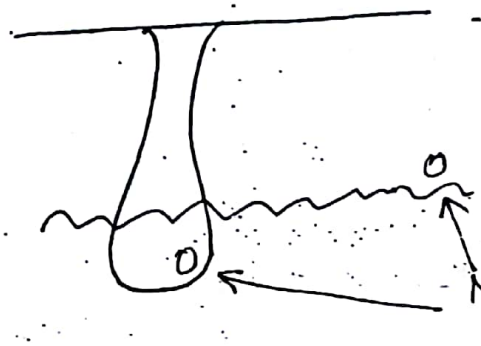
1 ⟶ 3

Ludwig Greading scale

DISORDERS OF MELANIN

(I) DISORDES of HYPO / DEPIGMENTATION

(A) ALBINISM



Tyrosine
↓
Tyrosinase x at birth
↓
Melanin.

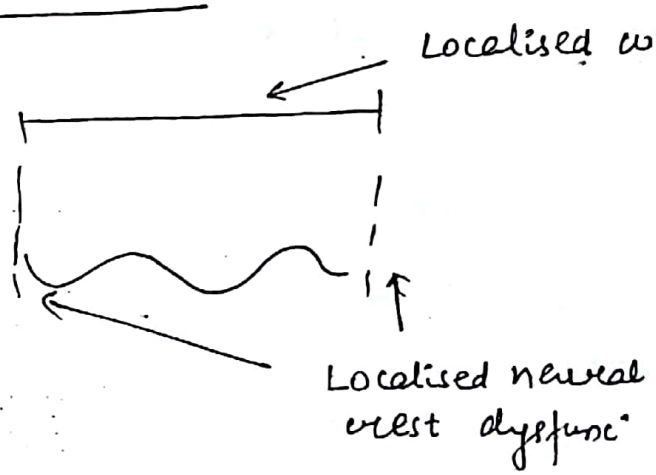
Melanocyte +nt
Melanin absent

Congenital

Diffuse white skin & white hair

No iris Pigmentation.

(B) PIEBALDISM



NO Melanocytes
NO melanin

Congenital

* Areas of skin without white patch
* White forelock
  - front
  - Lock of hair

WAARDENBERG SYNDROME

Piebaldism + Deafness + ↑ Inter pupillary Distance

(C) NEVUS DEPIGMENTOSUS / NEVUS ACHROMICUS :-

↓ Birth mark

No colour

Localised white patch since birth.

Pathology :-

Melanocyte ↓

Melanin transfer to keratinocytes ↓

(D) NEVUS ANEMICUS :-

Vascular Ab (N)

Faint hypopigmented patch since birth

Not a melanin disorder

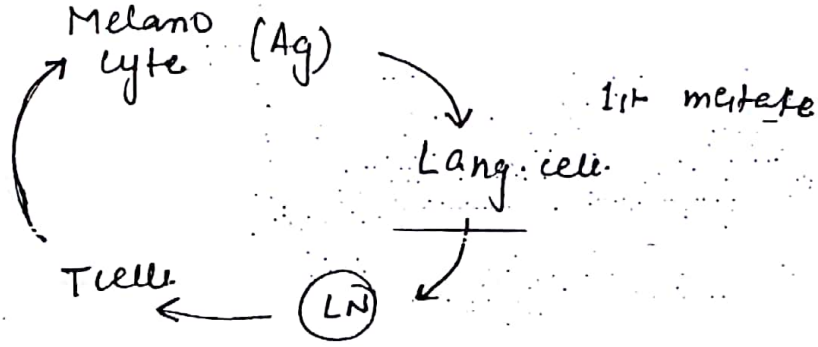
(E) VITILIGO :-

Acquired, not congenital

Autoimmune Disorder

Depigmented Lesion

M/c underlying Disease : Thyroid Disease



H/P :-

No melanocyte

No melanin.

Skin ← T cells → Skin

Hair

Hair

Depigmented skin

Black Hair.

Depigmented skin

>> Hair

↓

Leuco - Trichia

* POOR PROGNOSTIC FACTORS :-

1) On Bony prominences

2) Leucotrichia

3) Lip - Tip

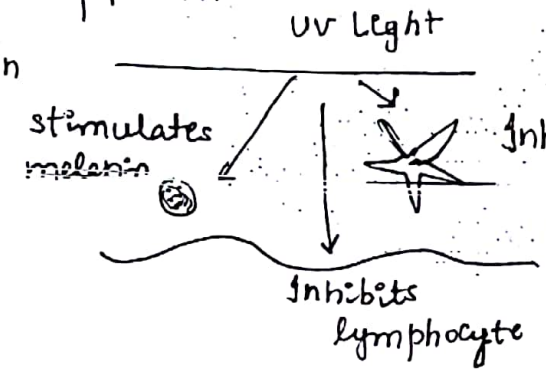
4) Thyroid Disease

CLASSIFICATION OF VITILIGO

LOCALISED	GENERALISED
Focal	Acrofacial
Segmental	Vitiligo vulgaris (M/c)
Mucosal	Universal
Lip - Tip	

Rx = Immunosuppresives

Photofherapy In Vitiligo



UVA Phototherapy

Need a sensitiser called 'PSORALEN'

- Topical (localised cases)
- Oral (generalised cases)

↓

Wait for 1-2 hours

↓

give UVA light

↓

[PUVA Therapy]

- Psoralen
- UVA

UVB Phototherapy

UVB (290-320nm)

No need for PSORALEN

- Broad Band UVB (290-320nm)
- Narrow Band UVB (311nm) → Best Therapy

For Localised cases:-

Topical immunosuppressives

- Steroid
- Tacrolimus

For Generalised case:-

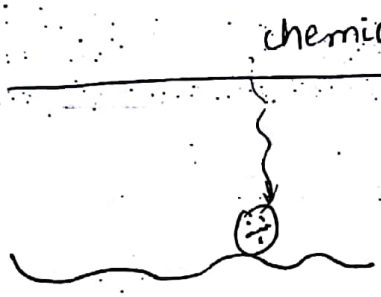
Systemic immunosuppresives

- Steroid
- Azathioprine
- Methotrexate

Surgical (Done ~~for~~ only for stable vitiligo)
↓
No new lesions
∵ past 2 years.

↳ Split skin Graft

(F) CONTACT LEUCODERMA



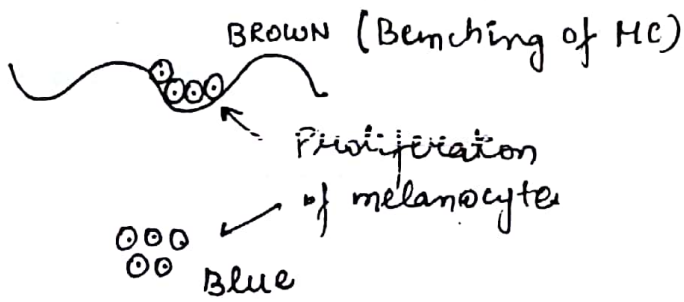
Agents causing Leucoderma

1) Bindi (commonest)
   - Para tertiary Butyl Phenol (PTBP)

2) Footwear / plastic
   - Monobenzyl ether of hydroquinone (MBEH)
     ↓ Most potent agent
     ↓
     useful in universal vitiligo - to depigment ⓝ Remaining skin

(II) DISORDERS OF HYPERPIGMENTATION

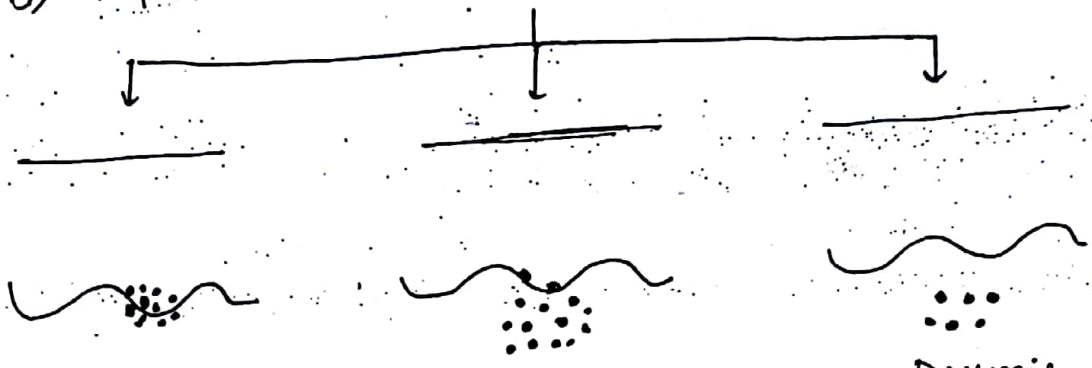
(A) MELANOCYTIC NEVUS (common mole)



1) CONGENITAL MELANOCYTIC NEVUS (CMN)

Giant Nevus (>20cm) has risk for malignancy
↓
Melanoma

B) ACQUIRED MELANOCYTIC NEVUS (AMN)



Junctional AMN
↓
BROWN MACULE

DEJ + Dermis
↓
Compound AMN
(Cells at Dermis are inactive)
↓
BROWN PAPULE

Dermis
↓
Intradermal AMN
↓
SKIN COLOURED NODULE

C) NEVUS OF OTA AIIMS MAY 2015

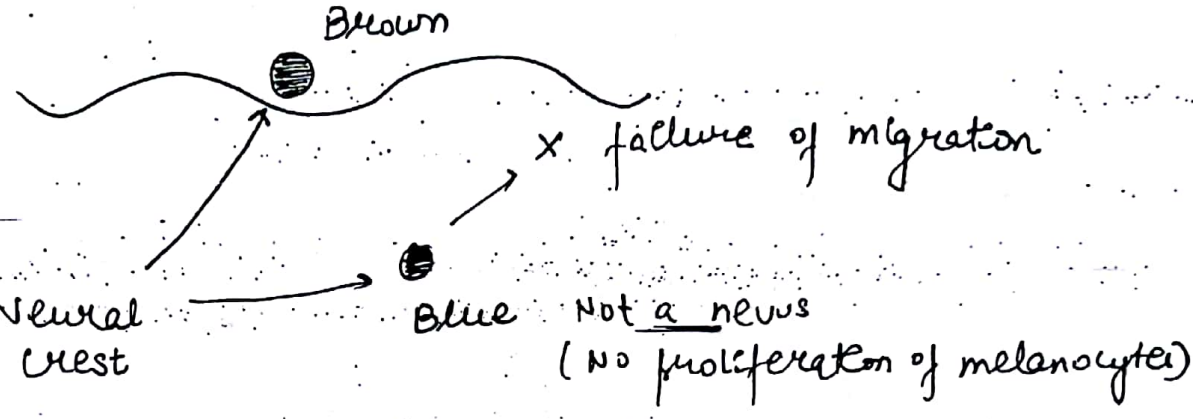
- Dermal melanocytic nevus
- Blue in colour
- along Trigeminal N/V
- U/L
- Congenital
- along ± Blue Sclera on same side

D) NEVUS OF ITO (similar to nevus of ota)

- Shoulder
- ~~Upper Back~~
- clavicular area

✓ Supraclavicular & Lateral Brachial N/Vs

E) MONGOLIAN SPOT



Site :- Lumbosacral area

Self resolving by puberty

F) BECKER's NEVUS Q.

→ Epidermal melanocytic nevus

↓

Hence Brown in colour

- On Shoulder, Chest & Upper Back
- ♂ > ♀
- → Onset → Adolescents
- → Due to androgen sensitivity causing hypertrichosis & Acne inside the Brown patch.

R_x = LASER.

G) MALIGNANT MELANOMA

R/F :- 1) Fair skin

2) Giant CMN

3) Atypical / Dysplastic Nevus

4) Family H/o.

5) Xeroderma Pigmentosum
(DNA repair disorders)

6)

CRITERIA

A → ASymmetry

B → Border (irregular)

C → Colour (multiple)

D → Diameter > 6mm

E → Evolution.

TYPES

1) **LENTIGO MALIGNA** (melanoma in situ)

Brown — Horizontal phase → cells travel in single line along basal layer

Best Prognosis

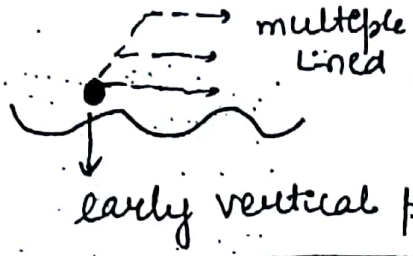
2) **LENTIGO MALIGNA MELANOMA**

10-15yrs → develops a vertical phase

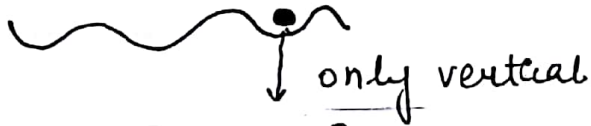
Not very good

3) **SUPERFICIAL SPREADING MELANOMA**

H/C type in world



4) **NODULAR MELANOMA**



POOREST PROGNOSIS

5) **AMELANOTIC MELANOMA**

→ Non pigmented

→ Variant of ~~no~~ nodular melanoma

6) **ACRAL MELANOMA**

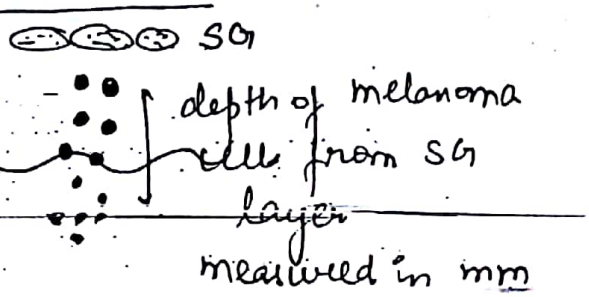
↓

extremity

IOC :- Excisional Skin Biopsy

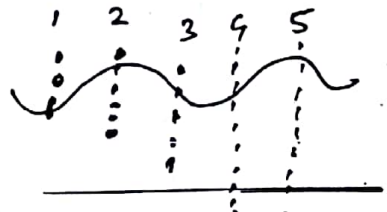
Histological Grading

→ **BRESLOW**



measured in mm

→ **CLARKE**



outdated

R_x = Surgical excision.

MAST CELL DISORDERS

(HL) mast cells ✗

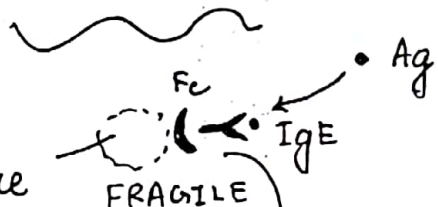
mast cell ✓ [Depth]

very tough membrane

(A) URTICARIA (HIVE)

Disease of fragile mast cell membrane

Depth is ok



Rupture

(H) Histamine (L) Leukotriene

create inflammation on mast cell surface

↓

weaken mast cell

1) Redness
2) oldema
3) Itching

→ Wheal (Transient) Lesions

TRIGGERS FOR MAST CELL RUPTURE

Ⓐ Acute Triggers

↓

Acute urticaria (< 6 week)

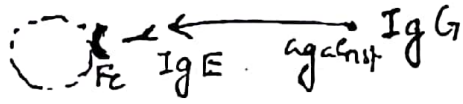
→ Food
→ Drugs
→ Infections

Ⓑ Chronic Triggers

↓

Chronic urticaria (> 6 weeks)

1) Autoimmune urticaria



A/c autoimmune thyroiditis.

2) Idiopathic Urticaria

Trigger is unknown.

3) Physical urticaria

↓

Trigger is physical

eg. cold → cold urticaria

sun → solar urticaria

sweat (eccrine) – cholinergic urticaria

scratch – Dermographism

↓ ↓

to skin to write

water → Acquagenic urticaria

Rx

Acute cases - Remove Ag

Chronic cases - Ag. Removal difficults

▷ Anti-(H_1) antihistamines.

1st gen
↓
More sedative
eg. Hydroxyzine

2nd gen
↓
Less sedative
eg. Levocitrezine
Fexofenadine
Loratidine
↓
preferred

2) Anti(H_2) Antihistamines
eg → Ranitidine
→ Cimetidine

3) Anti - Leukotriene
eg. → montelukast

4) For Autoimmune urticaria
↓
Immunosuppresives.
eg. → steroids
→ cyclosporine
→ Azathioprine
→ Methotrexate

5) Omalizumab ⇒ Anti IgE drug
↓ Ab
monoclonal

(B) ANGIOEDEMA

also called as **QUINCKE'S EDEMA**

Rupture of mast cells in subcutaneous **fat**

↓

Fat doesn't have itch n/vs.

No redness

But oedema is very prominent

Because fat is a loose tenue

↓

eyelids / lips

a/c resp. oedema → sudden death.

Rx = If lips, eyes involved → Inj Hydrocortisone

If resp. → Inj adrenaline

HEREDITARY ANGIOEDEMA

C_1 esterase inhibitor

↓ ⊖

Bradykinin

In HAE → C_1 esterase ~~enzyme~~ inhibitor enzyme deficiency

↓

Hence **Kinin Level ↑**

↓

Triggering angioedema

AD inheritance

Low complement (C_4) :- Screening Test

TYPES

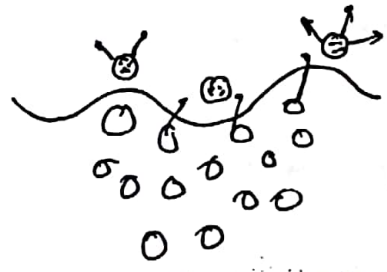
1 → Reduced amount of enzyme

2 → Amount (N), inactive enzyme

(C) MASTOCYTOSIS (URTICARIA PIGMENTOSA) (2nd, 1st)

mast cells ↑ no. — on rubbing

Brown.



producing melanin

Mast cell Proliferation → stimulate basal melanocytes

Presents as BROWN Hyperpigmented patches, plaques or nodules on TRUNK of a CHILD

Scratching on Brown patch → mast cell rupture (superficial) → Urticaria (red, elevated, itchy)

DARIER's sign (-ve)

Other causes:-

1) Xanthogranuloma
2) Histiocytosis
3) Leukemia

~~Pseudo-Darier's Sign~~

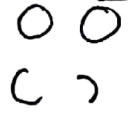
Smooth M/s Ha martorize.

SHAPES OF SKIN LESION

1> ANNULAR (Ring)

eg. a) Tinea [Ring worm]
b) BB Hansen
c) Herald Patch of Pityriasis Rosea

2) CIRCINATE
multiple circles



eg. Circinate Balanitis (Reiter's Disease)

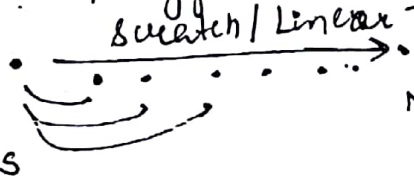
3) LINEAR NODULES , discharging sinuses along lymphatics

Causes

Sporotrichosis :- caused by sporothrix schenkii

FISH TANK GRANULOMA / SWIMMING POOL GRANULOMA
caused by Mycobacterium marinum.

4) ISOMORPHIC or KOEBNER's PHENOMENON
same morphology



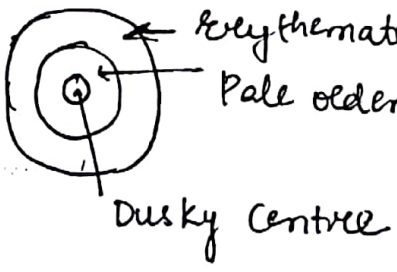
TYPES of KOEBNER's PHENOMENON

TRUE (autoimmune)	FALSE (Viral)	RARE
Psoriasis	Wart (verruca vulgaris)	Darier's Disease
Lichen planus	Molluscum	HHD
Vitiligo	Due to auto-inoculation while scratching.	Erythema multiforme
		Kaposi's sarcoma
		Lichen sclerosis
		Lichen nitedus

REVERSE KOEBNER -
Psoriasis

5> TARGET LESION/ BULL'S EYE /IRIS LESION

Eg. Erythema Multiforme.
Erythema Chronicum Migrans



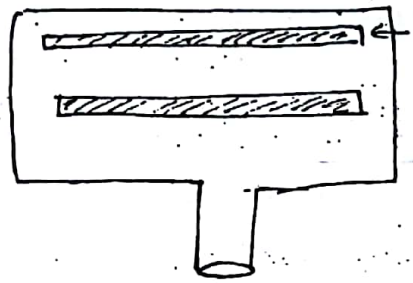
TESTS IN DERMATOLOGY

A) Wood's Lamp
B) Histopathology

WOOD'S LAMP

→ 365nm

→ Composition – Barium silicate + 9% nickel oxide



Use

1) **Tinea Capitis** →

Fungal (Dermatophyte)

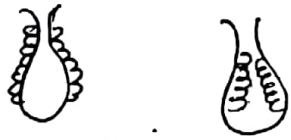
3 species

Trichophyton | Epidermophyton | Microsporum

Keratophilic fungus

↓

nail, Hairs.



Ectothrix

↓

usually by Microsporum

↓

⊕ on woodslamp

Bluish Green fluorescence

endothrix

↓

By Trichophyton.

↓

⊖ on wood's lamp

2) Erythrasma -
caused by Corynebacterium minutessimum
Red pathes → in groin & axilla
Asymptomatic
On Wood's Lamp :- "CORAL RED FLOURESCENCE"

3) Pityriasis Versicolor -
Fungal infecton by Malassezia
On WOOD's LAMP. YELLOW FLOURESCENCE

4) Burrow of Scabies -
on wood's lamp - GREEN.

5) Urine in Porphyria -
or Blister fluid
on Wood's lamp - PINK / RED

6) Vitiligo :-
WHITE

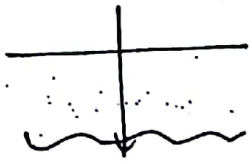
7) ASH LEAF MACULE.
WHITE & more prominent on Wood's

8)

PORPHYRIA

(N)

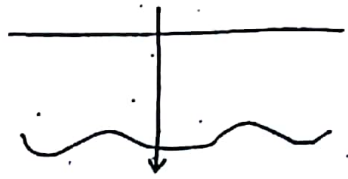
visible light



Neutralised by (N) Porphyrins in dermis

PORPHYRIA

visible soret light



Ab (N) Dermal porphyrin

↓

No neutralisation of light

↓

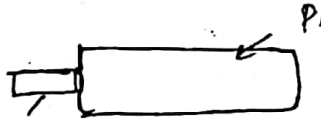
Dermal Damage

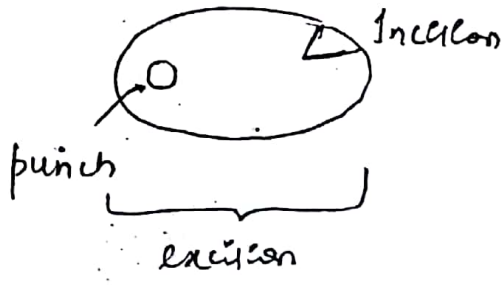
↓

Dermal tense bleister in light exposed areas.

B) HISTOPATHOLOGY-

1) By Punch Biopsy Instrument





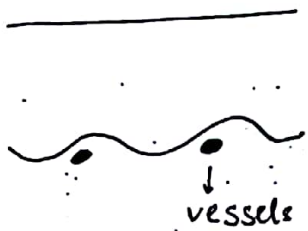
2) Incision Biopsy

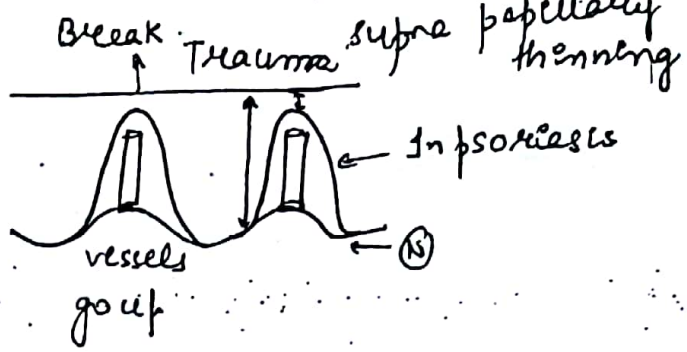
3) Excision Biopsy

4) Shave Biopsy ** (JIPMER)

Superficial Removal of skin ⊂ horizontal movement of Blade used for superficial elevation Lesions

1) Psoriasis :-





Intermittent pin point Bleed

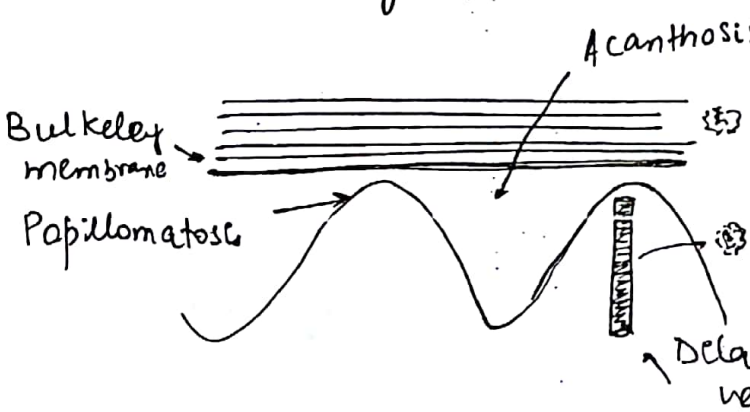
= AUSPITZ SIGN

Auspitz sign is demonstrated after GRATTAGE TEST (scraping)

↓

on scraping, Candle wax like scales are dislodged

Bulkley membrane is a thin membrane at the lower part of corneum & need to be dislodged to see bleeding points.



Munroe's microabscess (S. corneum)

S. granulosum → absent

Spongiform pustule of Kogoj (S. spinosum)

↑ neutrophils

Less neutrophils in corneum ⇒ MICROABSCESS.
not a macroabscess

More neutrophils in corneum ⇒ MICROABSCESS + MACROABSCESS

↓

Pustular Psoriasis

↓

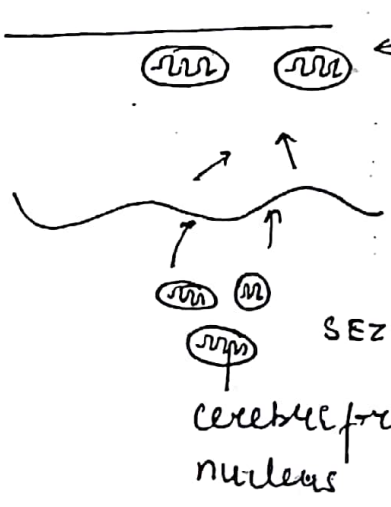
sterile pustule.

2> DERMATITIS HERPETIFORMIS :-

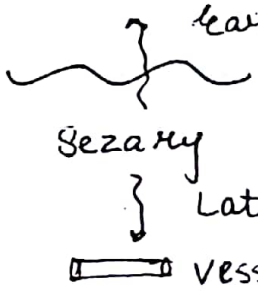
Papillary tip neutrophilic
" " microabscess
Not ~~macro~~abscess

3) MYCOSIS FUNGOIDES (MF)

Misnomer.
No fungus.
A type of CTCL (Cutaneous T. cell Lymphoma)
CD_4 ⊕ malignant dermal T cell.



when sezary cell go up ⇒ Early MF



FEATURES OF SEZZARY SYNDROME :-

1). Sezzary cells in Blood

2) Generalised Lymphadenopathy

3) Erythrodermal Exfoliative Dermatitis

4) means > 90% Body surface area involvement

Erythro - Red

Exfoliative - Scaly / falling off

4/F - Red scaly skin in > 90% Body surface

STAGES of MF :-

1) (I) → patch stage MF

(II) → plaque stage MF

(III) → Tumour

(IV) → Erythroderma stage

Rx of MF :-

1) EARLY :-

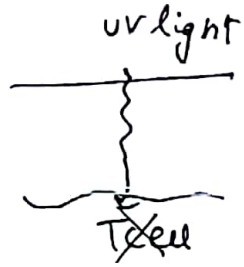
Rx from outside

Skin Directed Therapy (SDT)

a) Topical steroid

b) Phototherapy

c) Electron Beam Therapy (EBT)

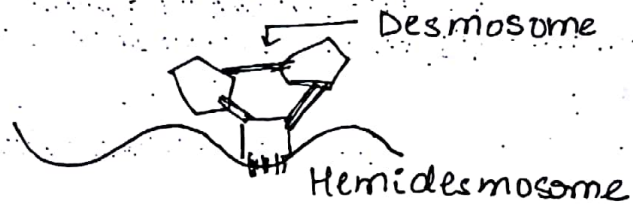


2) LATE :-

Rx from inside.

Chemotherapy

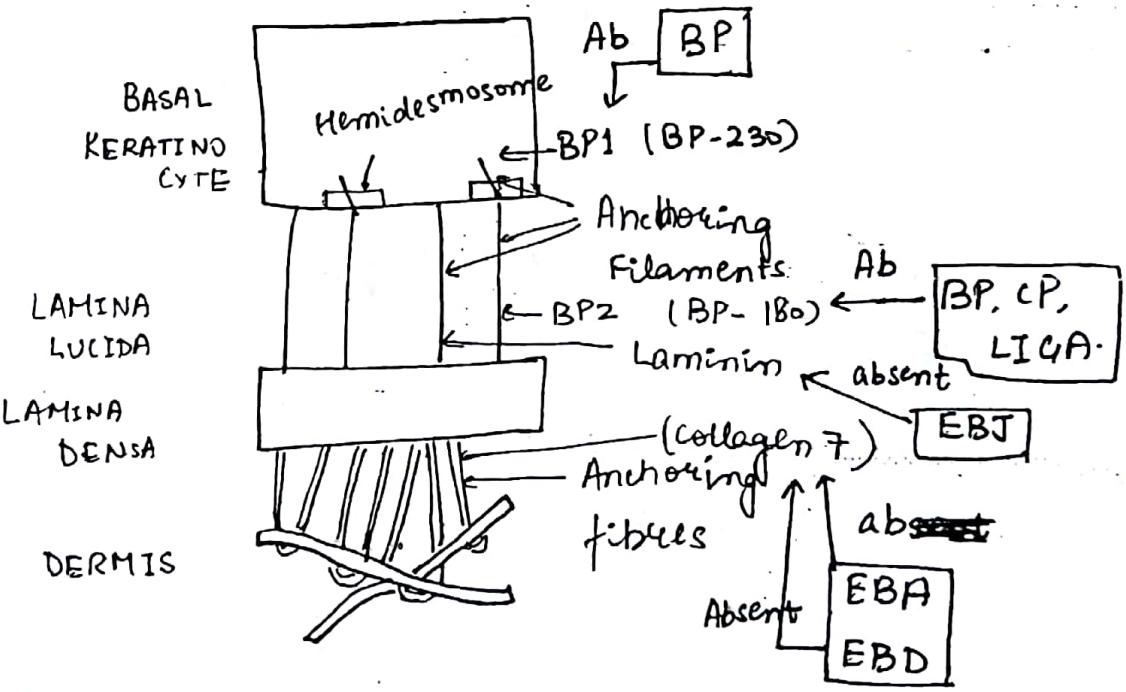
DERMO-EPIDERMAL JUNCTION (DEJ)



↓

contains Protein called

BP-1 (BP-230)



BP → Bullous Pemphigoid

CP → Cicatricial "

LIGA → Linear IgA disease

EBA → Epidermolysis Bullosa Acquisita

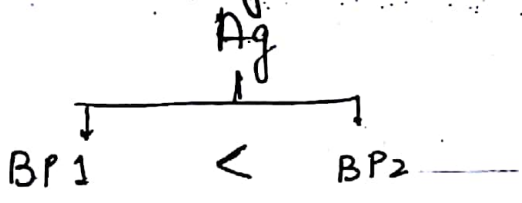
SALT SPLIT TECHNIQUE

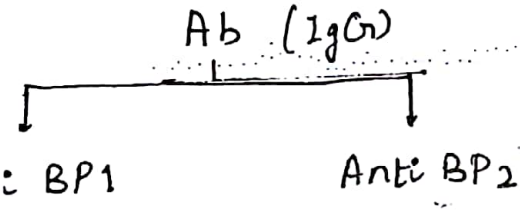
Splitting of skin @ Junc' of Lucida & Dense on putting the skin in saturated sol^{n} of salt

Roof Blisters → BP, CP, LIGA, EBJ

Floor Blisters → EBA, EBD

(I) Bullous Pemphigoid





Level of Blister = Lucida (Acquired)

DIF ⊕

(II) Cicatricial Pemphigoid

Ag → BP2

Ab → Anti BP2 (Ig G)

Level of Blister = Lucida (Acquired)

DIF ⊕

(III) LIGA

Ag = BP2

Ab = Anti BP2 (IgA)

Level = Lucida

DIF ⊕

(IV) EBJ

Ag = Nil

Ab = Nil

Absent Laminin since Birth

Level = Lucida

DIF = ⊖

(V) EBD

Ag = Nil

Ab = Nil (absent collagen 7 since Birth)

Level = Dermis

DIF = ⊖

(VI) EBA

Ag = Collagen 7

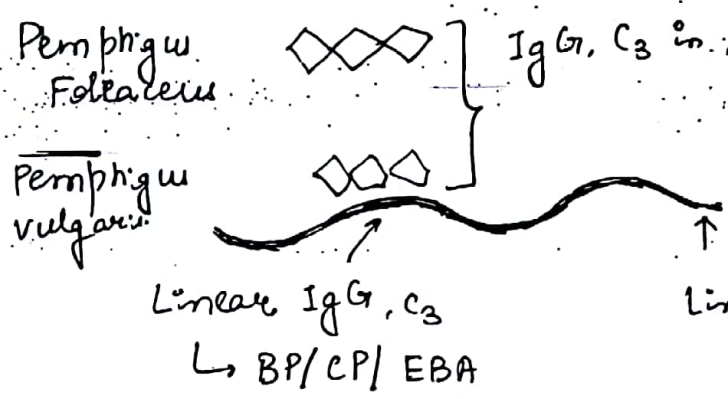
Ab = Anti collagen 7 (IgG)

Level = Dermis

DIF = ⊕

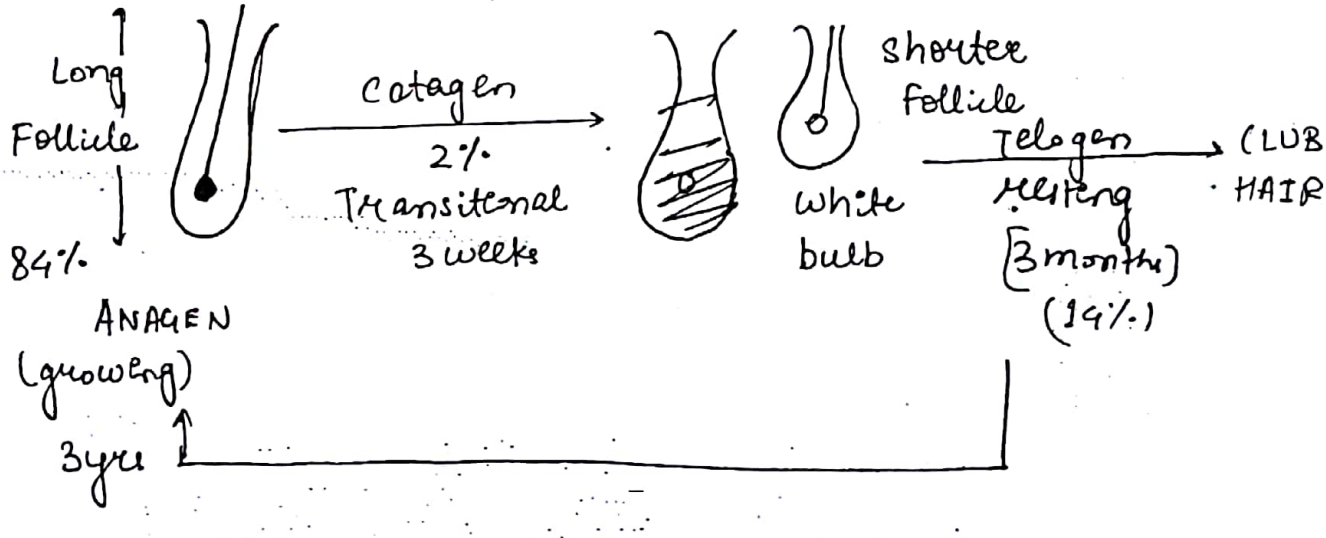
DIF

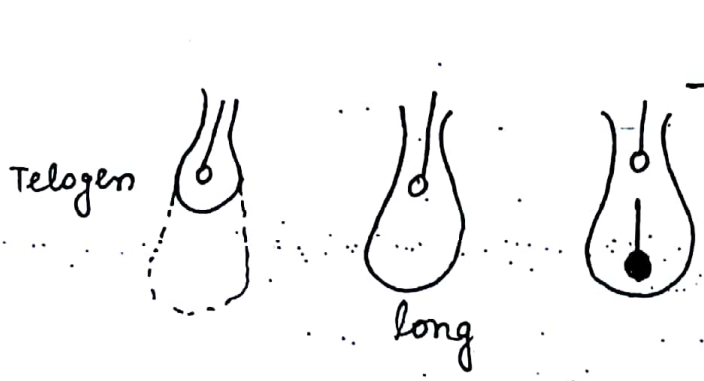
Sample is skin Biopsy from Perilesional skin for DIF. while for routine H & E Lesional Biopsy is taken



I Desmosome (Dsg-1) ⟷ IgG Ab ⟷ Anti-Anti IgG
green. green green

SCALP HAIR CYCLE





Anagen
→ pushes out the telogen.
↓
telogen loss
100 hairs/day

EFFLUVIUM (= Abn falling of Hair)

TELOGEN
>100 hairs/day
↓
Telogen effluvium

ANAGEN
1 hair/day
↓
Anagen effluvium
eg. Hair Loss in Chemotherapy

ACUTE TELOGEN EFFLUVIUN

Hair loss occurs acutely after 3 month of an acute Metabolic Insult to the body

eg severe Fever
Labour

No Rx Required, only Counseling.

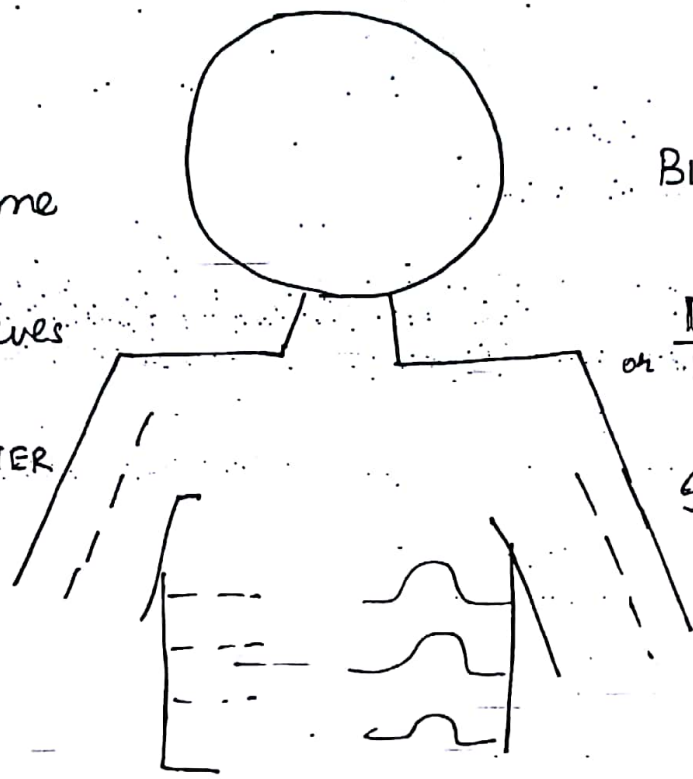
CHRONIC TELOGEN EFFLUVIUM

Hair Loss occurs chronically after chronic stressors

eg. Hypothyroidism
Anaemia
Nutritional Deficiency

Cause need to be Rx.

BLASCHKO LINES



Dermatome
↓
along nerves
↓
HERPES ZOSTER

BLASCHKO LINES
↓
Developmental Lines
or Keratinocyte Migration Line
↓
Incontinentia Pigmenti
Linear verrucous epidermal nevus

INCONTINENTIA PIGMENTI (AIIMS)

~~AD~~ ~~Dis~~ X-Linked Dominant Disorder

4 stages
Blister
↓
Verrucous (cauliflower like)
↓
Hyperpigmentation
↓
Hypopigmentation

LINEAR VERRUCOUS EPIDERMAL NEVUS Q. [AIIMS 65]

↓ ↓ ↓ ↓

Along BLASHCKO cauliflower epidermis since birth

Presents as cauliflower like masses along Blaschko line since birth. ↳ persists throughout life.

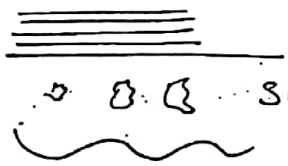
Histopath :-

Epidermolytic Hyperkeratosis

↓ ↘

Intra epidermis Breakdown,

In Stratum Granulosum.



NEUROFIBROMATOSIS

Skin Features :-

NF 1 → also called Von Recklinghausen Disease

1> AXILLARY FRECKLES (pathognomic)

↳ CROWE'S SIGN

2> CAFE-AU-LAIT MACULES (CALM)

↳ also seen in

a> Tuberous sclerosis

b> (N) people

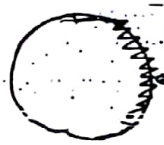
c) Mc-Cune Albright Syndrome

d) Fanconi anemia

← oval margin

Coast of California

NF



← Coast of Ireland

Mc-Cune

3) NEUROFIBROMA - BUTTON HOLE SIGN (+)

On pressing a blunt object on neurofibroma resistant is not felt in derma due to a dermal defect.

TUBEROUS SCLEROSIS / EPILOIA

Skin features:-

Tuber → potato like tumours in CNS.

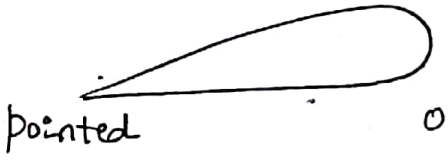
1) ASH LEAF MACULE
- Earliest
- H/c sign
- tnt at birth.
- Hypopigmented patch.
- Becomes more visible on wood's lamp.

Epi → epilepsy

Lol → Low

Io → IQ.

A → Adenoma sebacea.



pointed oval.

> 3 is significant

2) CONFETTI MACULE

↓

Small circulare.

~~hypo~~

→ Small hypopigmented Macule like confetti

3) ADENOMA SEBACEUM

- mimomer
- No sebum relation
- Skin coloured papules on face.

Onset - 2-5yr of age

Histopath → Angiofibroma.

4) **SHAGREEN PATCH/PLAQUE**

Shark skin → rough.

Roughened plaques on LS Region

H/P → collagenoma

5) **KOENEN'S TUMOUR**

→ periungual fibroma

→ at puberty

CONNECTIVE TISSUE DISEASE

(I) LUPUS ERYTHEMATOSIS

- SKIN → **DLE**
  - → Discoid (coin like)
    - periphery Active
    - centre active
  - → Nummular
- SKIN + SYSTEMIC → **SLE**
  - Cause (B)
    - cicatricial alopecia
    - non cicatricial alopecia.

DLE :- → only cicatricial alopecia

Photosensitive

Autoimmune

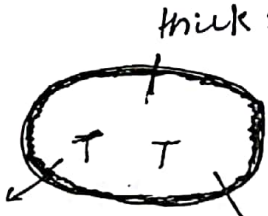
Hair follicle Disorder

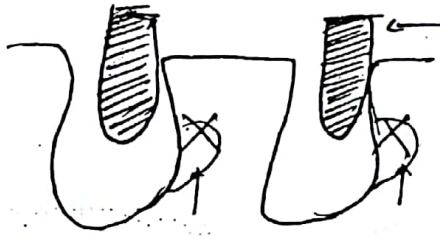
Scalp Hair / Facial Hair :-

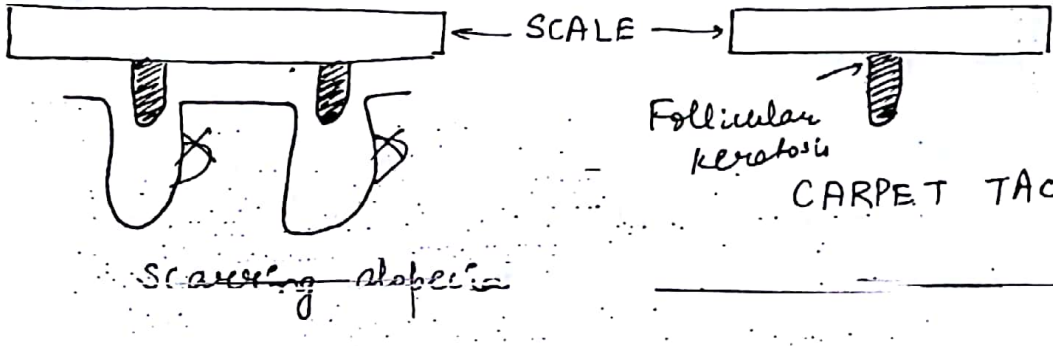
⊗ stem cells → Permanent Scarring Alopecia
(has hair stem cells)

C/F :-

Discoid plaque







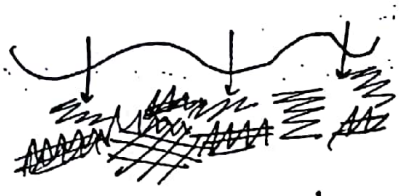
CARPET TAC SCALE

SLE

SLE pts sometimes have thin / fragile / easily broken hair
↳ [LUPUS HAIR]

(II) SCLERODERMA

excess collagen ↕ unpinchable. / Hard Bound Down skin.



pulled down.

collagen

- only SKIN → MORPHEA
  - M/C site = Trunk
  - Linear morphea on scalp causes Linear areas of cicatricial Hair Loss. resembling cut ē sickle.
    - ⇓
    - en-coup-d-sabre
      - a
      - cut
      - ē
      - sickle
- SKIN + SYSTEMIC
  - Few systems → LIMITED SYSTEMIC SCLEROSIS
  - Many systems → DIFFUSE SYSTEMIC SCLEROSIS

DIFFUSE SYSTEMIC SCLEROSIS → LIMITED SYSTEMIC SCLEROSIS

LIMITED SYSTEMIC SCLEROSIS → CREST SYNDROME

CREST SYNDROME
- C = Calcinosis
- R = Raynaud's phenomenon
- E = Esophageal dysmotility
- S = Sclerodactyly
- T = Telangiectasia

SYSTEMIC SCLEROSIS

CRITERIA for Diffuse

MAJOR	MINOR
Essential	2 or 3
• Scleroderma proximal to the metacarpophalangeal Jt.	• Sclerodactyly • Digital pitted scars • Bibasilar pulmonary fibrosis

SKIN FEATURES –

1) Mask like face due to facial tightening
2) Purse string mouth / microstomia 
3) Peri-oral Rhagades
4) Frequent Raynaud's phenomenon
5) Salt & Pepper Pigmentation

(III) DERMATO MYOSITIS

Skin — Proximal M/s weakness

↓

(A) Lilac/Purple Colour

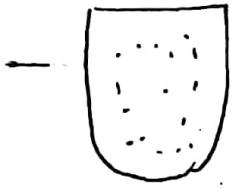
- around eyes ⊃ photosensitivity → Heliotrope Rash.
- in shawl area → SHAWL SIGN
- on Interphalangeal or Metacarpophalangeal Jts → GOTTRON's PAPULE

(B) Mechanics Hand.
↳ Rough Hand due to palmar Hyperkeratosis

NAIL DISORDER

1) PITING DISEASE.

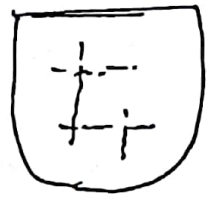
Due to involvement of proximal nail matrix.



RIDL

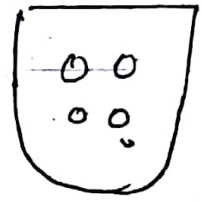
Random
Irregular.
Deep
Large

Psoriasis



Superficial
Regular
Geometric

Alopecia areata



Coarse large pits.

Eczema

H/c sign of nail psoriasis.

2) NAIL PSORIASIS

a) pitting is the H/c sign but not specific to psoriasis
Having pits → ↑ chance of getting jt involvement in psoriasis pt.

b) Salmon Patch / Oil Drop sign :- ~~(Pathognomic of psoriasis)~~
Red faint

3) Subungual hyperkeratosis
4) Onycholysis
5) Splinter heamorrhage.

3> NAIL LICHEN PLANUS (JIPMER)

Longitudinal Ridge
Pitted nail
Thinning of nail plate
Dorsal Pterygium (extension of proximal nail fold to nail bed.)

2o Nail Dystrophy. (Trachyonychia) JIPMER
or Sand Paper nail
↓
also seen in psoriasis
& alopecia areata

~~Inverse~~

4> INVERSE (VENTRAL) PTERYGIUM Q.

Skin from nail bed fuses with the undersurface of nail bed seen in scleroderma.

5> HALF & HALF NAIL

Seen in Chronic Renal Failure
Reversible on hemodialysis

due to anemia
↓
Proximal 50% white

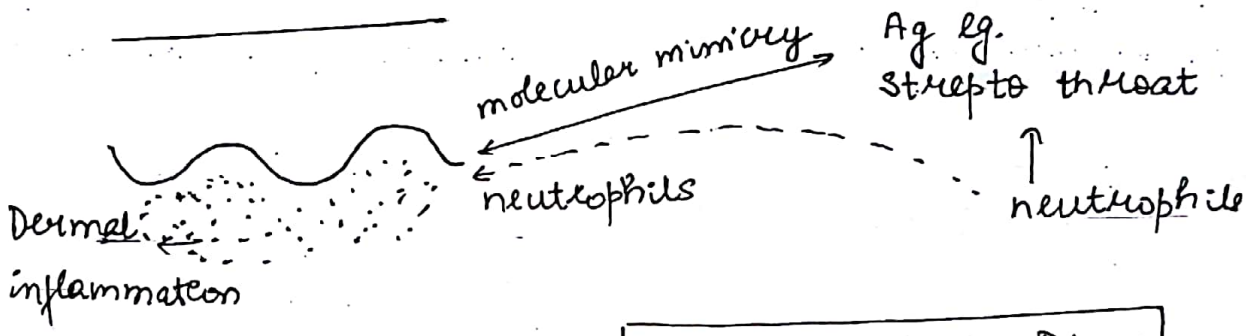
Distal 50% Brown.
↓
due to melanin deposits on nail bed
⋮
due to THSH secretion.
in CRF

6) BEAU'S LINE

Horizontal groove on the nail plate.
Due to temporary suppression of nail growth due to past fever or Local nail fold trauma
No Rx Required.

NEUTROPHILIC DERMATOSIS

Neutrophilic accumulation in Dermis



Rx = Antigen Removal + Anti-Neutrophilic Drug

1) DAPSONE
2) COLCHICINE
3) STERDIDS - oral

Dermatitis Herpetiformis → DOC :- Dapsone
Behcet syndrome
Sweets syndrome
Pyoderma Gangrenosum } DOC: steroids

PYODERMA GANGRENOSUM

No pyoderma, no gangrene
Present as Very Painful leg ulcer ~ Purple Margin around it
ulcer → undermined

Associated c
- IBD
- Haematological malignancy

SWEET SYNDROME / ACUTE FEBRILE NEUTROPHILIC DERMATOSIS.

Presents acutely c red, oedematous, painful plaque on extremities c fever & Jt pains.

Resembles cellulitis.

DOC :- Steroids.

Associated c
— Strepto (H/c)
others — AML, Drugs ♀

Histopath :-
Plenty of neutrophils on dermis

Beh

BEHCET'S DISEASE

MAJOR

Recurrent aphthous ulcers
↓
Superficial, round/oval, painful c a red margin around it.

MINOR (any 2)

+ Recurrent genital aphthous ulcers
→ Eye Lesions (Panuveitis)
→ Skin Lesions (Erythema nodosum, pustules)
→ ⊕ Pathergy test

PATHERGY TEST :-

Inflammatory Papule or Pustule at the site of intradermal Injⁿ on the forearm. (after 48 hrs)

Seen in-

1) Behcet's
2) Pyoderma Gangreenosum
3) Sweet's syndrome (rarely)
4) RA.
5) IBD

CUTANEOUS TB

A) EXOGENOUS TB

1) TB chancre
   ↳ means ulcer.
   Flask shaped undermined

2) TB verrucosa cutis (TBVC)
   ↓ cauliflower skin.

3) LUPUS VULGARIS
   ↳ H/c typ of cut. TB in adults.

Sputum → TB

4)

Previous exposure to TB
↓
Post 1°
↓
High immunity
TBVC
(paucibacillary)

No previous exposure to TB
(TB naive, patient)
↓
1° TB
↓
Pt. has low immunity to TB
↓
TB chancre
MULTIBACILLARY (MB)

LUPUS VULGARIS ✪ AIIMS.

Healing ☰ central scarring

Progressive Lesions

Buttocks.

DIAGNOSIS :- 1) DIASCOPY

Pressing ☰ a glass slide

↓

— Yellow Brown Nodules visible

(APPLE JELLY NODULES)

↳ also seen in

Sarcoidosis

Leishmaniasis

2) SKIN BIOPSY

Non-caseating tuberculoid Granuloma

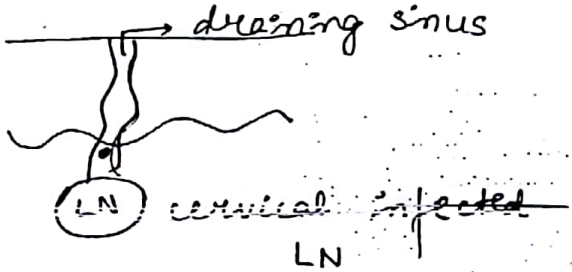
Lupus vulgaris ☰ paucibacillary

Central clearing	Central scarring	Central crusting
↓	↓	↓
Tinea	Lupus vulgaris	Leishmaniasis

(B) ENDOGENEOUS TB

1) SCROFULODERMA → M/C in children.

2) PERI-ORIFICIAL



Perl-oral & Perl-anal ulcer ⇒ severe int. TB.

(C) TUBERCULID → daughter Lesion
↓
mother Lesion.

* Daughter Lesions initiate Hypersensitivity in skin causing inflammation also leading to fragmented bacilli in skin & can't be cultured

Mantoux test is strongly positive in Tuberculid.

3 TYPES (Depending on size of daughter Lesions)

Daughter is very tiny (micropapule size)

Micropapular Tuberculid or Lichen scrofulosorum

C/F- Grouped micropapules on trunk

H/P- Perifollicular non-caseating TB granuloma

Daughter is small

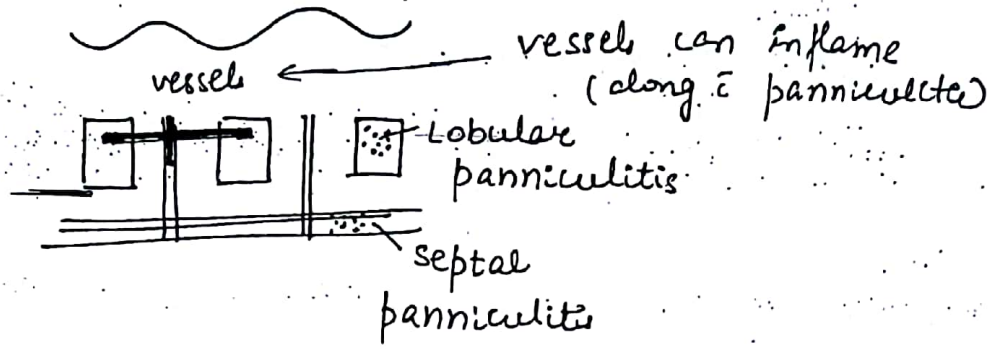
Papulo-necrotic Tuberculid

Daughter is Large

- Nodular tuberculid Erythema induratum
- Bazin's Disease

C/F- Red, tender, nodule on calf & ulcerates

PANNICULITIS



SEPTAL PANNICULITIS

- ī vasculitis
- ī out vasculetis
  - eg. Erythema Nodosum

LOBULAR PANNICULITIS

- ī vasculitis
  - eg. Nodular Tuberculoid
- ī out vasculitis
  - eg. 1> Pancreate Panniculitis (Acute/chr. pancreatetis/ Cancer)
  - 2> Post steroid panniculetis
  - 3> Lupus paniculite
  - 4> Subcutaneous fat necrosis of newborn

ERYTHEMO NODOSUM

Red, Tender nodules on **shin** & **never ulcerate**

CAUSES

NO - No cause, Neutrophilic Dermatoses
(Behcet's Disease, Sweet Syndrome)

D - Drugs (iodides, bromides, sulfonamides)

O - OCP

S - Sarcoidosis

U - Ulcerative Colitis (also Crohn's)

M - Microbes (strepto)
Maternity
Malignancy (Hematological)

FEATURES	EN	ENL	SWEETS SYNDROME
Neutrophils	+	+	⊕
Histiocytes	+	+	⊖
Vasculitis	−	⊕	⊖

Rx of EN:-

1) Bed Rest
2) Neutrophil Removal Drugs (steroids, Dapsone, colchicine)
3) Removal of cause

HANSENS DISEASE

M. Leprae
↳ grows in (cool) areas
- Skin
- n/v (superficia)

Nerve 1st —then→ Skin.
(thicken the n/v)

Nerve involved but doesn't involve skin = Pure neural Leprosy

H/c peripheral n/v involved (UL) = Ulnar
LL = Post Tibial

H/c deformity = CLAW HAND

M/c cranial n/v = FACIAL N/V
↳ Lagophthalmos

Biopsy taken from Radial cut > Sural n/v

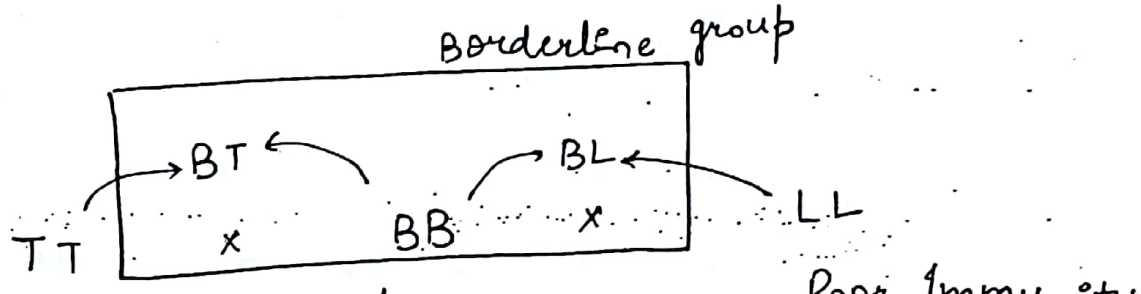
Commonest Hansen in India = BT HANSEN

Commonest Int. Organ Involved = Testis (bcoz it has low temp)

Organ never involved in HANSEN = uterus → ♀
CNS → ♂

Earliest sensation ~~lost~~ lost = Hot & cold differentiation > Cold > Hot > light touch > pain > crude touch

Sensation never Lost = Proprioception, vibration.



Good Immunity
Tuberculoid
Tuberculoid

Intermediate Immunity
Borderline
Borderline
(mid Borderline)

Poor Immunity
Lepromatous
Lepromatous

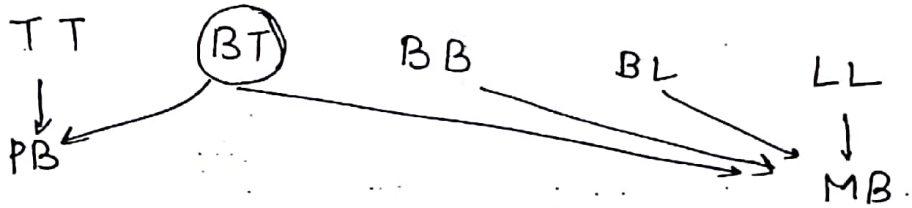
RIDLEY JOPLING CLASSIFICATION

Based on.

1) Clinical
2) Bacteriological (slit skin smear - SSS)
3) Histological (skin Biopsy)
4) Immunological (Lepromin testing)

If there are >10,000 Bacilli / gm of skin → MULTIBACILLARY HANSEN

If there is <10,000 Bacilli/gm of skin → PAUCIBACILLARY HANSEN.

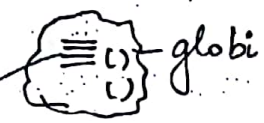


TT → Immunologically stable

BT → BB → BL → LL Downgrading [cout]

TT Hansen on Biopsy shows Perivascular, Periadnexal neural Granuloma

LL on Biopsy shows: Foam cell, Virchow cell, Lepra cells (Dermal macrophages full of leprosy Bacilli) —



cigarette shaped

Special stain

1) Zn Stain

2) Fite Stain.

↓

[Blue background
Red Bacilli.]

Image

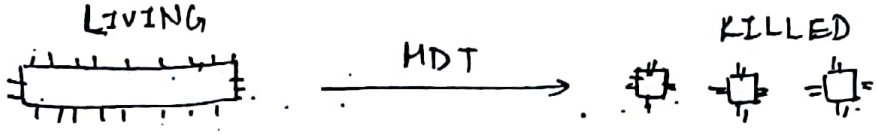
Entry of Organism

↓

sent to LN

Th1 (Tcells) → Toward TT

Th2 (Bcells) → Toward LL.



$10 cm^2$ Ag
↑
10 units of immunity

$100 cm^2$ of Ag
100 units of immunity
(Hypersensitivity)

T cell
Upgrading/Type 1 Lepra Reaⁿ/ Reversal Reaⁿ

B cell
Type 2 Lepra Reaⁿ

Hypersensitivity Towards TT side = TYPE IV
Presents as Neuritis
& Nerve Abscess
↓
also called TYPE 1 LEPRA RXN of N/V.

Rx for Neuritis ⇒ MDT + Oral steroids.
Rx for N/V Abscess ⇒ I & D.

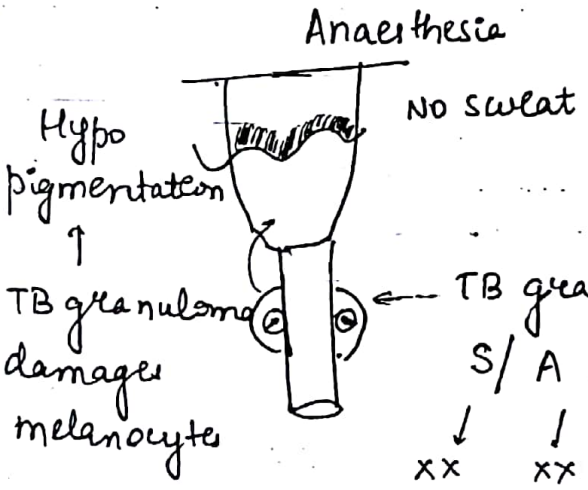
Hypersensitivity toward LL Side = TYPE III
Presents as vasculitis
Called as TYPE -2 LEPRA Rxn. or Erythema Nodosum Leprosum

Effect of MDT on-

TT	BT	BB	BL	~~BB~~ LL
↓	↓	↓	↙ ↘	↓
T cell hyper	T cell hyper	T cell hyper	T cell hyper	B cell hyper
↓	↓	↓	↓	↓
Type I Lepra	Type 1 Lepra	Type I Lepra	Type 1 Lepra	Type 2 Lepra

B cell hyper
↓
Type 2 Lepra

TT HANSEN :-



⇒ 1 thickened N/V + 1 skin Lesion (saucer Morphology)

INDETERMINANT HANSEN :-

Presents as Hypopigmented patch on cheek in children.
(N) sensation (N) sweating

D/D of Hypopigmented Patch on cheek in child

- C fine Scaling → Pityriasis alba
- Non scaly, atrophic ~~from endemic~~ area → Indeterminate Hansen.

FATE

Most pts. upgrade ← IH → some downgrade

TT ← IH → TT

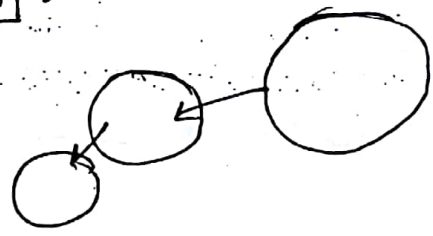
Histopath :-

Perivascular or Periadneral Lymphocytes

Bacillus not seen.

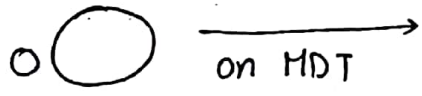
P **BT HANSEN** :-

Satellite Lesions (3-10)



few thickened n/vs

Original BT



on MDT

BT in type 1 Lepra

← ← ← ← ←

Slow upgrade

No clinical

Type 1

Fast upgrade

Clinical Type I.

BB HANSEN :-

sharp well defined

ill defined

1) Inverted saucer
2) Punched out
3) Annular (AIIMS).
4) Swiss-cheese

(10-30 Lesions)

TT
Granuloma size ↓, Hence
Sensations improve
Sweating "
Dry Lesions become shiny Lesions
Hypopigmentation ↓
Symmetry of patches ↓ N/Vs ↑
LL Patches ↑ in no. but ↓ in size

BL HANSEN

Many, almost symmetrical Lesions · almost symmetrical n/vs

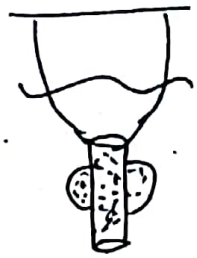
Inverted saucer / punched out Lesions

Uncomfortable Lesions.

LL HANSEN

Diffuse infilteration of skin · · Peripheral N/vs

1) (N) sensations (N) sweating, ill defined Borders
2) Ear Lobe infilteration
3) Lateral Madarosis
4) Gynaecomastia due to testicular involvement
5) Saddle nose - collapse of bridge of nose
6) Nasal Septal Perforation
7) B/L lagophthalmose ← due to facial n/v involvement [corneal ulcer]
8) Ulcers · Stocking Anaesthesia ← due to peripheral neuropathy



B/L symmetrical n/v involvement

Thickening of n/v in LL is due to invasion.

EARLY SIGNS:-

Nasal stuffiness

Epistaxis (JIPMER 2014)

Leg oedema

Nodular LL = LEONINE

↳ painless nodules

Due to unequal invasion of by bacilli

site of Biopsy:-

Non nodular LL = LUCIO

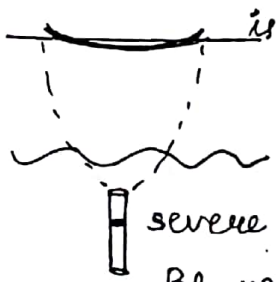
↓

means Beautiful (mexican)

also called BEAUTIFUL LEPROSY (Lepra Bonita)

wrinkle less / shiny skin, look younger] due to subcutaneous invasion by bacilli thus stretching skin

LUCIO REACTION:-



HISTOID LEPROSY:- Q.

type of LL ~ dapsone ~~resistance~~

(N) skin along ~ papulo-nodules

ERYTHEMA NODOSUM LEPROSY

Red — Nodule

Tender Nodule on extremities (New Lesions)

SYSTEMIC FEATURES

Immune Complex Deposit →
- Fever ✓
- Jt Pain ✓
- Uveitis ✓
- GN ✓
- Orchitis ✓
- Hepatitis ✓

Cytokine Involved in ENL = TNF α

Hence TNFα Inhibitors (Thalidomide) is given in ENL

TYPE 1	TYPE 2
NSAIDS	NSAIDS
Oral steroids - Doc	Oral steroid - Doc
Chloroquine	Chloroquine
Azathioprine	Azathioprine
Cyclosporine	Thalidomide
	Clofazimine

RECURRENT ENL

Step 1 - Prednisolone 3 months + Clofazimine
1mg/kg/day — 100 mg TDS - 3 months

↓

Step 2 - only clofazimine 100mg BD - 3 months

↓

Step 3 - ~~Clofazimine~~ 100 mg OD - 3 months

HISTOPATH OF LL

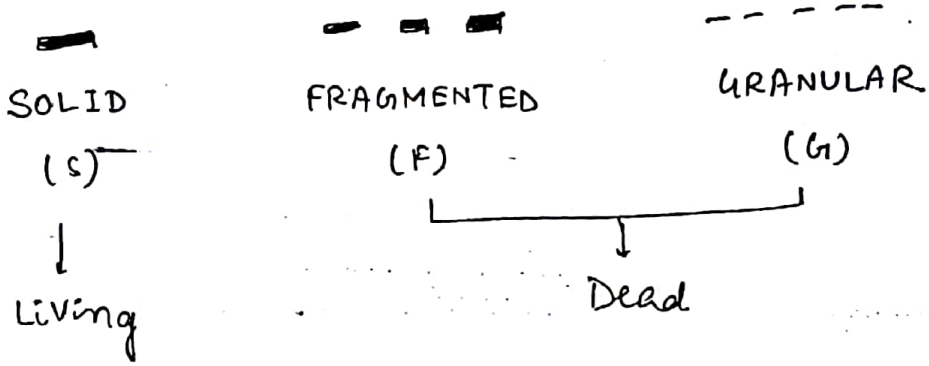
GRENZ ZONE

zone of sparing in upper dermis c out foam cells. This may be a zone of better Immunity in the dermis

Slit Skin Smear

Sites : Skin Lesions & Ear Lobe → Best site

3 types of staining Pattern



S + F + G = Bacteriological Index (BI)

S = Morphological Index (MI)

BI remains +ve even after Rx [↓ by 1+ every year on Rx]

BI is measured from 1+ to 6+

eg. BI = 3+ before MDT

↓ ← Rx eg for 1 yr

(2+) ← stop Rx

↓

1+ automatically after 1 yr.

↓

0 " " "

MI becomes -ve after Rx

RELAPSE

BI ↑ by 2+ over the previous value
clinically by new skin lesions + new thickened n/vs

SSS is +ve if u have more than 10,000 bacilli/gm (Multibacillary) of tissue.

SSS is -ve if less than 10,000 bacilli/gm of tenue (Paucibacillary)

SS is -ve in ~~these in~~ -

TT

BT

Indeterminate Hansen

Pure neural Hansen

SS is +ve in -

BT

BB

BL

LL

LEPROMIN SKIN TEST

Intradermal ~~sts~~ test for immune status in Leprosy

⊕ ← → ⊖

⊕ Towards TT side → Normal People

⊖ Towards LL side

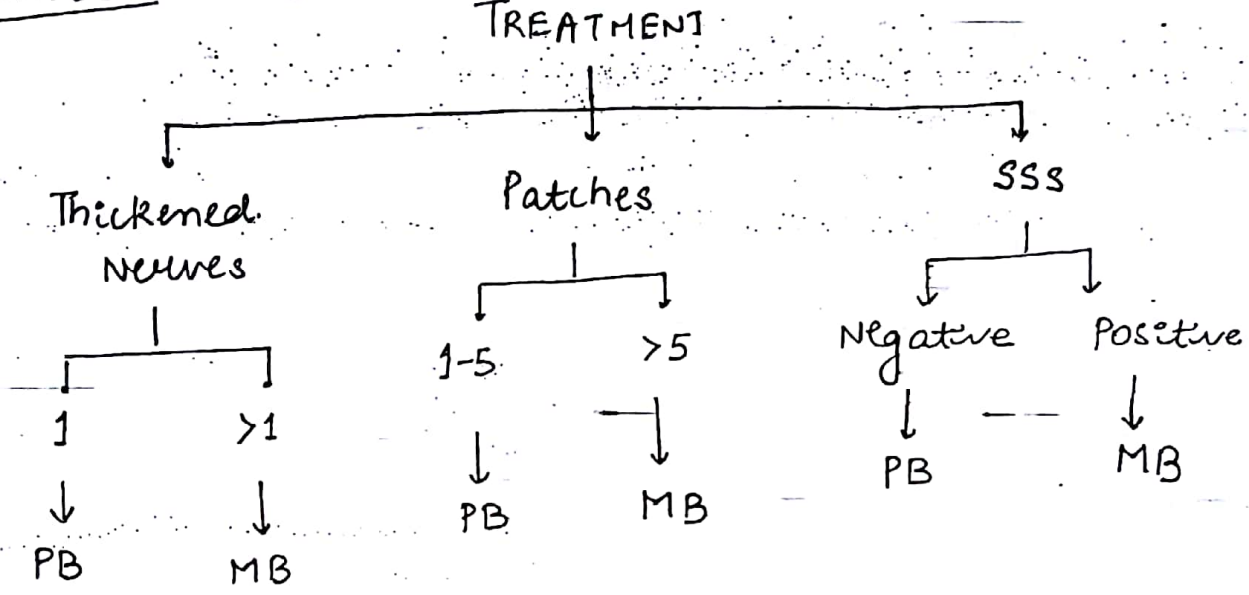
Not a diagnostic test but a prognostic test

Reading of Lepromin

1) Early response
↓
wheat at 48 hrs
↓
FERNANDEZ Rxn

2) Late response at 4 wks
↓
MITSUDA Rxn
(Better indicator of CMI)

SLIDE 1

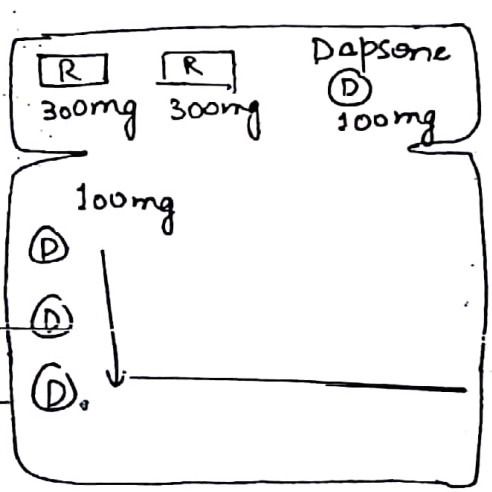


From a programme perspective only clinical Δ is enough to classify PB & MB

ROM = Rifampicin + ofloxacin + Minocycline ; discontinued

SLIDE-2 PB PACKET – GREEN

each packet = 28 days
Finish 6 pack max in a month

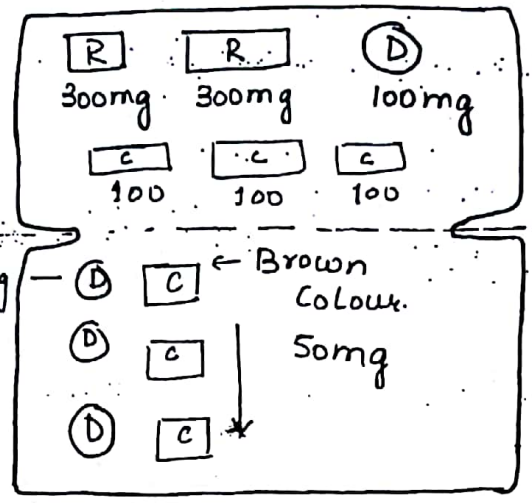


5 supervised monthly

Daily unsupervised

SLIDE - 3 MB - Red

12 packets max in 18 months.
Each pack = 28 Days



- Lesions often remain the same even after completion of MDT.

2nd LINE DRUGS

1) Quinolones
   Moxi/ Spar/ ofloxacin
2) Clarithromycin
3) Minocycline
4) Rifapentene

S/E of CLOFAZIMINE

Q Pigmentation - M/c
Ichthyosis (Dry Skin)
~~Intestinal obstruction~~

S/E of DAPSONE

1) Hemolytic anaemia
2) Peripheral neuropathy
3) **Dapsone Syndrome** ('5th WEEK SYNDROME')

↓

Skin Rash
Hepatitis
} after 5 weeks of taking Dapsone

STD

GENITAL ULCER DISEASES

① SYPHILIS

T. pallidum - Spirochete ∴ "cork-screw" motility

↓

enters Genitals

(1° chancre) I.P. - 9-90 days

↓

ulcer.

Extragenital chancre = M/C site = **LIP**

SLIDE-4

1° chancre
(Hard - Hard chancre)
painless
- Button like ⊙
- Round / oval
- Single
- Regular edges

I.P. = 9-90 days

IOC in 1° chancre - Smear from ulcer
↓ followed by
Dark Ground Illumination (DGI)
↓
MOST SENSITIVE & MOST SPECIFIC TEST IN 1° SYPHILIS

DGI can't be done from extragenital Lep chancre due to salivary contamination & commensal Treponemes.

SLIDES

Blood Test In 1° chancre

at 3 wks → EIA (Enzyme Immuno assay)
most sensitive screening test

at 3 wks → FTA-Abs → outdated.

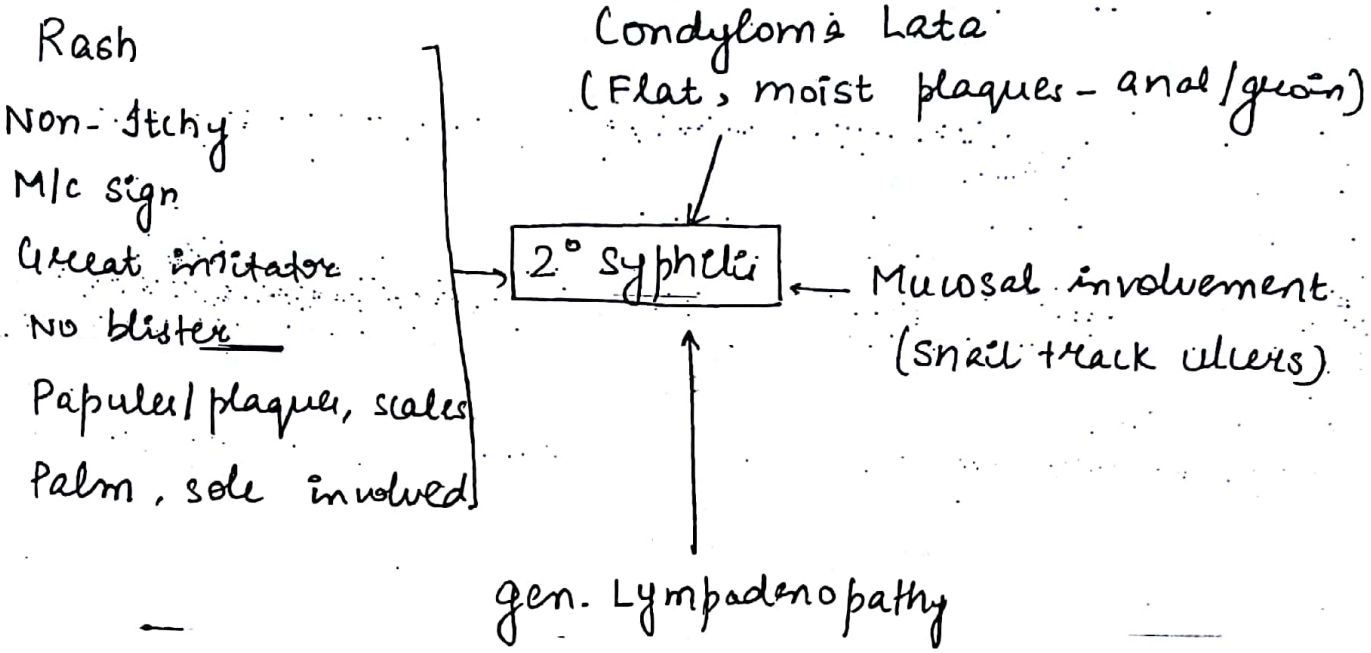
4 wks → VDRL

4-6 wks → TPPA / TPHA

(b) → Ing. L.N. ⟶ move into Blood (2° syphilis)
enlarged
painless
Rubbery
Shotty
IOC = VDRL

1° chancre self heals

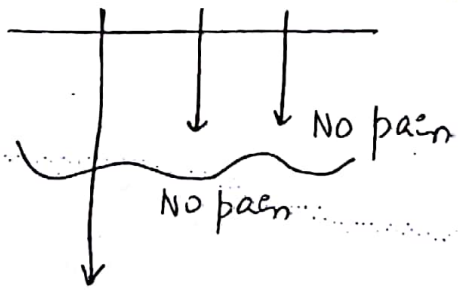
SLIDE-6

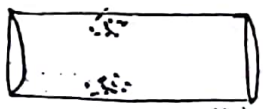


Deep Dermal Tenderness/ Buchke-ollendorf sign

On deep pressure c a blunt object on palm, sole

Lesion → there is deep tenderness





Deep pressure on palm, sole cause tenderness

End arteritis Obliterans

Condyloma Lata is full of Spirochete

↳ Hence DGI sample can be ✓ taken from it

MOTH-EATEN ALOPECIA

non-scarring alopecia → also seen in
- → alopecia areata
- → Trichotillomania

2° syphilis ⟿ spirochetes stay in blood but become inactive

(Pts → asymptomatic)

LATENT SYPHILIS → EARLY LATENT / LATE LATENT

↓

Spirochetes go into deep tissue.

[TERTIARY SYPHILIS]

3 types

- Into skin: Gummatous syphilis
- CNS: Cardiovascular
- CNS: Neurosyphilis

Ioc of neurosyphilis = CSF VDRL

SLIDE 7

EARY SYPHILIS <2yrs	1° 2° Early Latent	Inj^{n} Benzathine Penicillin 2.4MU IM Single dose
LATE SYPHILIS >2yrs	Late Latent 3°	Inj^{n} Benzathine Penicillin 2.4MU IM 3 doses at weekly intervals Neurosyphilis → I.V. aqueous crystalline penicillin

JARISCH- HERXHEIMER Rxn

Inflammation, Fever, ↑ of lesions after Rx in syphilis is max. in 2° syphilis.

Rx In Penicillin Allergy:-

Doxycycline (14 days - early syphilis
Incompliance ↓ — 28 days - Late syphilis)

Chancre (Redux)

Recurrent syphilis
Relapsing

Rx Pregnancy:-

→ Same as in non-pregnant pts.
→ If allergic to penicillin → Desensitize

VDRL - used to monitor response to therapy
↓
titre reduces 4 fold ī in 6 months of Rx.

1:64
↓ Rx
1:16 ī in 6 months

Specific Treponemal Test (TPPA, TPHA) remain +ve even after therapy often ~~long~~ life long
So, can't be used for prognostic purpose

CONGENITAL SYPHILIS

Early (1st 2yrs) — like adult 2° syphilis

Late (>2yrs) — like adult 3° syphilis

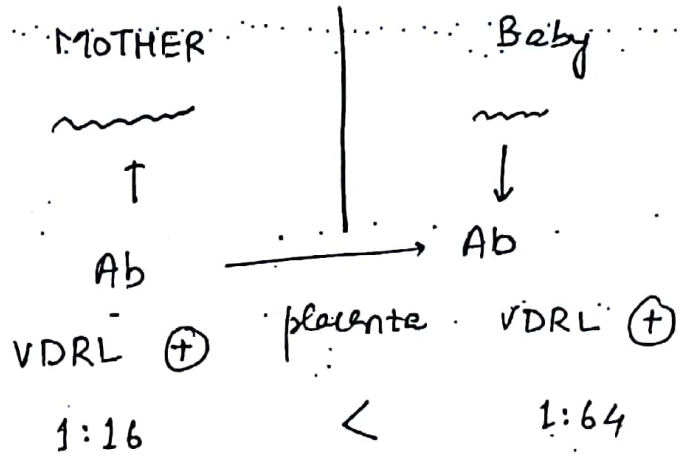
SLIDE-8

EARLY CONG.	LATE CONG.
1) Snuffles (Rhinitis) earliest & M/C sign	1) Clutton's Joint - Painless knee swelling
2) BLISTERS Q. (Syphilitic pemphigus)	2) Sabre Tibia (Ant. bowing of tibia)
3) Epiphysitis	3) Olympian's Brow - frontal Bossing
4) Osteochondritis cause pseudoparalysis	4) SADDLE NOSE
4) Condyloma Lata	

HUTCHNISON TRIAD of LATE CONG. SYPHILIS

Interstitial Keratitis

8th n/v disease — Hutchinson's tooth



⇒ VDRL is +ve in Baby at birth.

But if titre is more than mother
↳ Baby has syphilis

Δ = VDRL of Baby 4 times > mother's titre

DG1 from nasal secretions . blister fluid

(II) CHANCROID

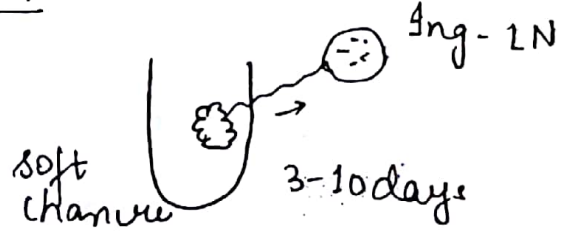
H. Ducreyi - extracellular gram -ve organism.

I.P. = 3 - 10 days.

Chancr|oid
chancre → like.

⇒ completely unlike chancre.

SLIDE-9



painful
Multiple
~~irregular~~ edges
Bleeds on touch

Inflammation
↓
Bubo (ipsilateral, may suppurate)

In chancroid, kissing ulcers are seen due to autoinoculation.

Δ =

1> Gram stain on smear ⇒ Gram -ve

School of fish
Rail road track

Extracellular

2> Intradermal test - ito test

↓

outdated

3> PCR on skin Biopsy or smear.

Rx -

Azithromycin 1gm stat

or

Inj^{n} ceftriaxone 250 mg im stat

(III) DONOVANOSIS

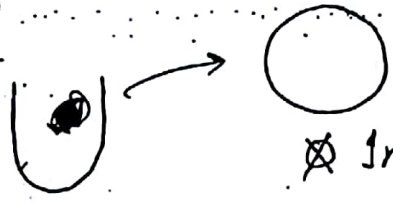
INTRACELLULAR

Calymmatobacterium granulomatis (Klebsella granulomatis)

I.P. = 8-80 Days.

→ Hypertrophic granulation tissue on ulcer floor
→ Beefy Red colour
→ Bleeds on touch
→ Painless

SLIDE - 10



Granuloma - pseudobubo

⊗ Ing. L-N.

Granuloma Inguinale

Crush smear.

↓

Giemsa stain

DONOVAN BODIES

Histocytes (closed safety Pin) arrangement

↳ AIIMS Nov 15, May 2017

⊕ Organism is intracellular

Rx = Azithromycin 1gm/week (preferred) } Yell ulcer heals

or

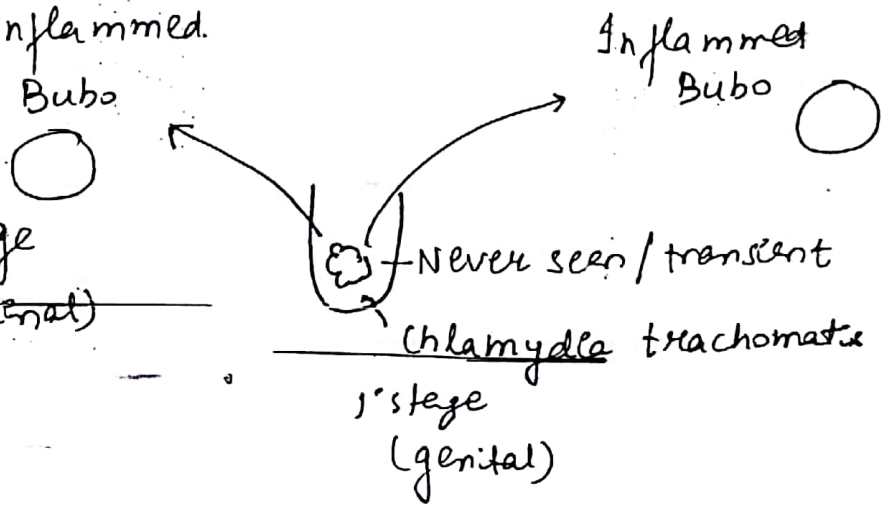
Doxy 100 mg BD

IV LGV

Chlamydia Trachomatis

I.P - 10 - 30 days.

SLIDE - 11



BuBos

U/L ← → B/L.

ing Bubo

Femoral Bubo

→ Inguinal Ligament (GROOVE)

↓

Also seen in

a) MONDOR's Disease

b) Eosinophilis fascitis

3° Stage of LGV

Elephantiasis due to Lymph oedema

RAMS HORN PENIS / SAXOPHONE

S shaped penile deformity

LYMPHANGIECTASIA

Bubbles of lymph on skin surface

LYMPHORRHOEA

oozing of lymph

ESTHIOMENE

(Lymphangiectasia + overlying ulceration)

Δ of LGV = 1> Frei test → outdated

2> PCR for chlamydia by NAAT ← Best Test

Most commonly done 3> CFT (complement fixation Test)

4> MIFT (micro immuno flouresent test)

ANORECTAL FEATURES

1) fissure
2) fistula
3) Sinuses
4) Strictures.

℞ of LGV -
Doxy 100 mg BD for 21 days

V HERPES GENITALIS

HSV-2 > HSV-1.

Recurrent Blister + Ulcers (painful, grouped) along ± painful inguinal Bubo always recurrent

℞ = Acyclovir group of Drugs.

URETHRAL DISCHARGE

Pathology = URETHRITIS

GONOCOCCUS

Gram -ve intracellular diplococcus

♂
often symptomatic
↓
Urethritis
(urethral discharge)

♀
1) often asymptomatic (carrier)
↓
In cervix
2) cervical discharge

Presumptive Partner Treatment (PPT)
is done in STDs to prevent recurrence in index STD pt.

SLIDE-12

GONOCOCCAL	NON-GONOCOCCAL
N. gonorrhoea	Chlamydia/Trichomonas/ mycoplasma/ureaplasma
~~IP~~ 2-8 days	1-3 weeks
Thick purulent urethral D/c	Thin mucopurulent D/c
Rx -	Rx
→ Inj^n ceftriaxone 250mg IM stat + Azithromycin 1gm stat (preferred)	Tab. azithromycin 1gm stat (preferred)
→ Tab cefixime 400mg stat + Azithromycin 1gm stat	Doxy 100mg BD for 7 days

SYNDROMIC APPROACHES

URETHRAL DISCHARGE

GONORRHOEA → CEFIXIME

CHLAMYDIA → AZITHRO

GREY PACKET

NACO KIT-1
AZITHRO + CEFIXIME

VAGINAL DISCHARGE

CANDIDA
- White creamy
- minimal D/c
- Itching

TRICHOMONIASIS
- Green frothy D/c
- Profuse D/c
- Strawberry Cx
- Itching

BACTERIAL VAGINOSIS
- Foul smelling D/c
- M/c cause
- No Itch
- No Inflammation

Candida → Tab. FLUCONAZOLE 150mg stat

Trichomonas, Gardenella → Tab Metronidazole 2g stat
or Tab Tinidazole 2g stat
or Tab Secnidazole 2g stat

NACO KIT-2
SECNIDAZOLE + FLUCONAZOLE
green packet

CERVICAL D/C

- GONORRHOEA → GREY KIT
- CHLAMYDIA → GREY KIT

green kit (for vaginal D/c)

Speculum Examn

BACTERIAL VAGINOSIS Q.

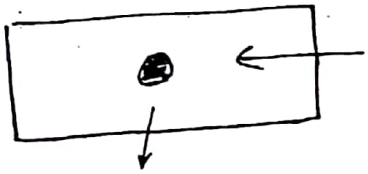
→ GARDENELLA VAGINALIS
→ Anaerobic Bacteria (Bacteroides, Peptococcus)
→ Mycoplasma

AMSEL CRITERIA Q.

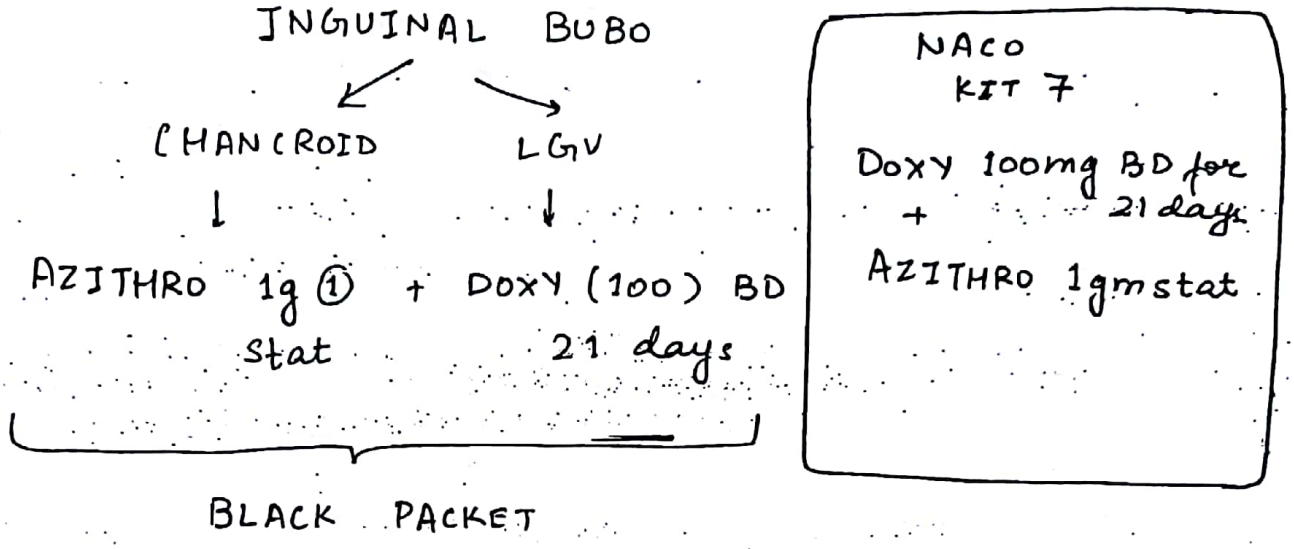
1> Thin homogeneous white adherent D/c
2> Vaginal fluid pH > 4.5
3> Fishy amine odour (WHIFF TEST)
4> Clue cells ≥20%

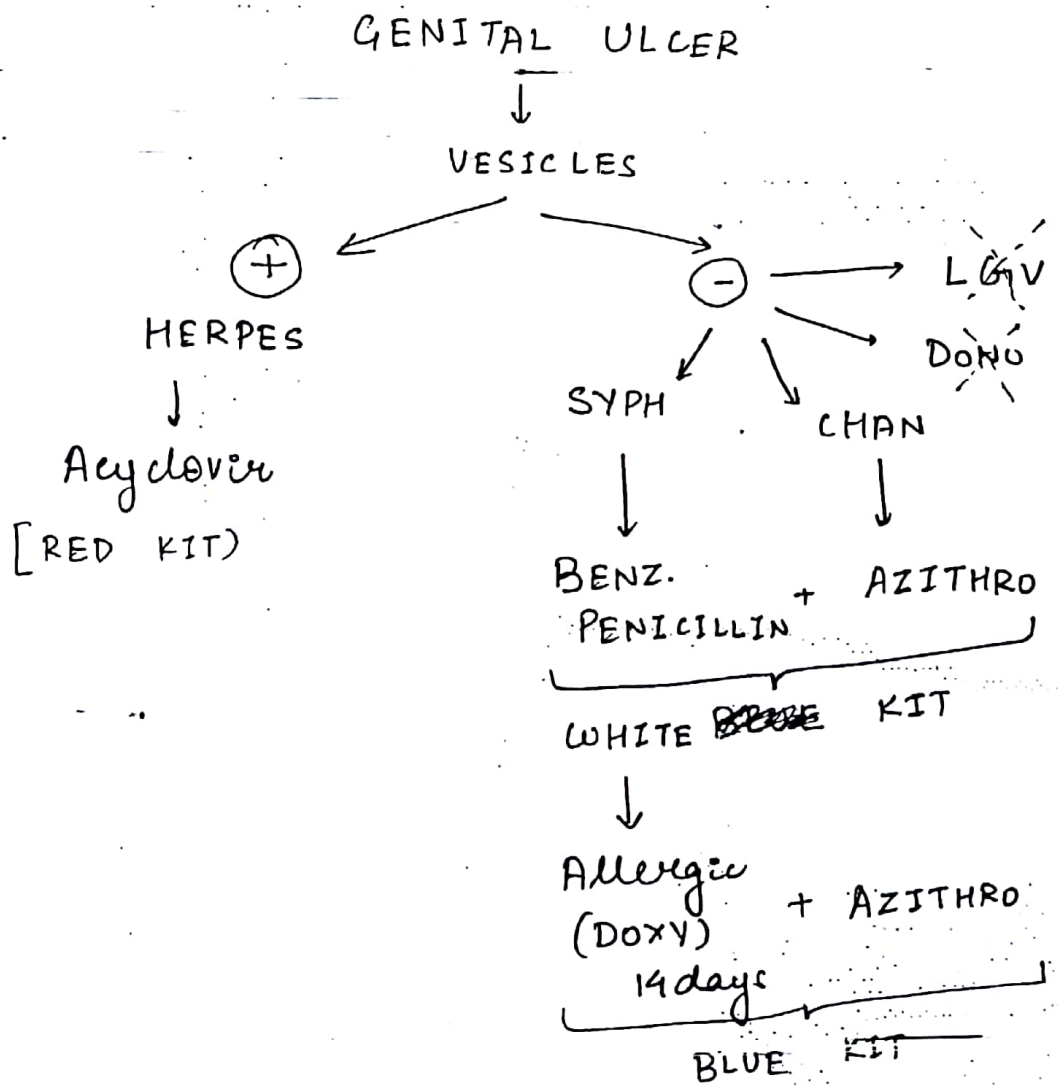
Clue cells on wet mount / GRAM.

Organism Epithelial cell



Amine / Fishy odour. (whiff test)


INGUINAL BUBO
CHANCROID
LGV
AZITHRO 1g ① stat
+
DOXY (100) BD 21 days
BLACK PACKET
NACO
KIT 7
Doxy 100mg BD for 21 days
+
AZITHRO 1gm stat


GENITAL ULCER
VESICLES
+
HERPES
Acyclovir
[RED KIT)
−
LGV
DONO
SYPH
CHAN
BENZ. PENICILLIN
+
AZITHRO
WHITE KIT
Allergic (DOXY)
+ AZITHRO
14 days
BLUE KIT

NO PARTNER T/T REQUIRED IN ♀

GARDENELLA

HERPES

CANDIDA [If partner is symptomatic at same time Rx partner]

M/C - WORLD (WHO - 2015)

STD (overall) - HSV2

Viral - HSV 2 > HSV 1

Bacterial - Chlamydia > Gonorrhoea

Protozoal - Trichomonas

BLANK

PSORIASIS

→ Autoimmune Disease

→ TH1 & TH17 mediated inflammation (T cells)

→ IL-(12), IL-(17), IL-(23) → are secreted by T cells. initiating inflammation

→ Associated ē HLA-Cw6

R_x = Immunosuppresives [systemic steroid is C/I]

STABLE

↓ ← Exacerbating factors

UNSTABLE

(- sudden many new Lesions
- pustular psoriasis
- erythrodermic psoriasis)

* Exacerbating Factors :-

✓ Sudden withdrawal of systemic steroids

✓ ♀

✓ Infn - streptococcus

✓ Drugs - (β blockers, Lithium, Chloroquine, NSAIDS, ACEI

TYPES

I> Ps. VULGARIS
↓
M/C type

on extensors silvery scales
itchy
chronic plaque form - M/C.

II> GUTTATE Ps.
↓
Raindrop

∴ associated ī streptococcal Pharyngitis
R_x - includes Antibiotic against strepto

III> ERYTHRODERMIC Ps. / EXFOLIATIVE DERMATITIS
↓ ↓
Red scaly

C/F- Red scaly plaques all over body (>90% of BSA)

R_x - 1st Line = Methotrexate
2nd " = ACITRETIN

IV> FLEXURAL PSORIASIS -
(inverse)
NO scaling
~~skin~~ erythematous shiny plaque in skin folds
(Inframammary area, groin) as scales get dislodged

V) SEBOPSORIASIS :
Silvery plaques on scalp
Thick scales

Seborrhoeic Dermatitis (SD)

↳ caused by MALASSEZIA.

↓

- yellow greasy scales.
- thin scales

VI) GEN. PUSTULAR PSORIASIS (GPP)

C/F Diffuse pus all over body

severe inflammation

Fever

"Sheet of pus"

"Lake of pus"

→ separate pustules on periphery

all pustules fuse together in centre to form "sheet of pus".

GPP

Non-Preg

↓

VON-ZUMBUSCH TYPE

↓

Rx - 1st Line = Acitretin

2nd Line = Mtx

♀

↓

IMPETIGO HERPETIFORMIS

↓

Doc = Syst. steroids

If ♀ is also diabetic

↓

Doc = Cyclosporin

VII) PSORIATIC ARTHRITIS

→ 5-30% of pts.

→ HLA B27, HLA B7

→ Nail psoriasis pt have ↑ risk of developing arthritis

→ Dactylitis, enthesitis

→ usually skin involvement precedes joint involvement

classical jt involvement = DIP.

DOC - Methotrexate. (except ARTHRITIS MUTILANS)

↓ DOC

[Etanercept]

SLIDE-14

Rx

<10%	10-30%	>30%
1st Line Topical steroids Topical calcipotriol (vit D derivative) ↳ DOC 2nd Line - coaltar - Salicylic acid coaltar is anti-division drug Salicylic acid is keratolytic	1st Line · Narrow band · UVB 2nd Line · PUVA	1st Line · Methotrexate · Acitretin 2nd Line · Cyclosporin · Fumaric acid ester · Biologics. (Etarnercept Infliximab, Adalimumab)

OLD REGIMENS Q

INGRAM

UV ↓

ANTHRALIN

GOECKERMAN

UV ↓

COALTAR

WORONOFF'S RING -

- Hypopigmented Ring around psoriasis Lesions indicating healing of Lesions
- Due to inhibition of PG synthesis ↓ resulting — **Vasoconstriction**

REITER'S DISEASE

REACTIVE

M/c cause

entry of **Chlamydia** through sexual route ↙

entry of Salmonella Shigella Yersenia Campylobacter → DIARRHOEA

URETHRITIS

3 common Symptoms afterwards

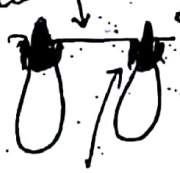
ARTHRITIS	RED EYE	SKIN LESIONS
- in wt bearing jts (large)	Conj^{n} / Uveitis	Circinate Balanitis — Keratoderma blenorrhagica (AIIMS, 08)
- HLA-B27		
- enthesitis, dactylitis		

Keratoderma blenorrhagica
- hyperkeratosis → on sole
- skin → pus → oozing.

PITYRIASIS RUBRA PILARIS

Red, Hair

(N) skin between follicle → ← Follicular Keratosis



Red/orange colour around hair

↳ very sharp on palpation → Feels like a "Nutmeg-grater" on palpation

---

(N) skin bet follicle = Island of sparing

→ Palmo-Plantar Hyperkeratosis

→ Erythroderma

→ Keratotic ~~sto~~ sandal → thick plantar keratin appearing like sandal of keratin.

LICHEN PLANUS

autoimmune disease

5Ps → PAPULES
- PURPLE
- POLYGONAL
- PRURITIC
- PLANE-TOPPED

NO scale

Flexural areas (M/c- wrist flexure)

A/c HCV, ~~HBV~~

Usually healing ⊂ hyperpigmentation.

← mineral oil

White criss-cross marks
(Wickham's striae)

↓

is due to **HYPERGRANULOSIS**

TYPES OF LP

1) **ORAL LP**

- White. criss cross (Lacy/ Reticular) pattern
- Buccal mucosa, tongue
- Associated ⊂ **Dental Amalgam** → contains Mercury
  - B/L ↳ U/L or B/L
  - have symptoms

AIIMS nov 15 (Image)

[Leukoplakia is U/L, not criss-cross, asymptomatic] –

* **Risk of malignant pot" in oral LP :-**

Lacy pattern doesn't have Risk

Ulcerative oral LP and LP on tongue have risk of 1-5%

2) **ACUTE WIDESPREAD LP**

Sudden eruption of multiple Lesions

DOC- systemic steroids

3> LICHEN PLANUS PIGMENTOSUS

Pigmentation in sun exposed areas (No itching)

4> LP PILARIS / PSEUDO PELADE

hair ↑ cells

Bulge (stem cells)

↑ Bulge is Ag

↓

Scarring alopecia

- ✓ patchy scarring alopecia
- ✓ perifollicular blue-grey macules
- ✓ "foot print in snow appearance"

Perifollicular Blue-grey macule

5> ACTINIC LP

- → sun induced LP
- → Itch
- → Hyperpigmented macule surrounded by a hypopigmented ring on sun exposed areas

6> HYPERTROPHIC LP

Hypertrophic plaques on lower legs

(thick, flat elevated)

Rx of LP

Oral LP
LP Pigmentosus → CHRONIC Ds.
LP Pilaris

⇒ **Localised LP** → Topical Rx
↓
Steroids
Calcipotriol
Tacrolimus

⇒ **Generalised LP** → Systemic Rx
→ Steroids
→ Non-steroidal immunosuppressive
Cyclosporine ← → Mtx
Phototherapy
Mycophenolate
Azathioprine mofetil

⇒ **For hypertrophic LP** → Acitretin (keratolytic)

ALOPECIA AREATA

autoimmune ds of hair

Ag :- melanin in hair bulb.

Bulge → Ⓝ

T cells

↓

destroy bulb

↓

pathy alopecia

H/c - scalp.

⇒ NON-SCARRING ALOPECIA → No wrinkling.

always sparing of white/ Grey hair in alopecia patch.

Rx :- LOCALISED PATCH

↳ Topical steroids

Minoxidil

Intralesional steroids - most effective

POOR PROGNOSTIC FACTORS

1) OPHIASIS

↳ areata at the hair line margin.

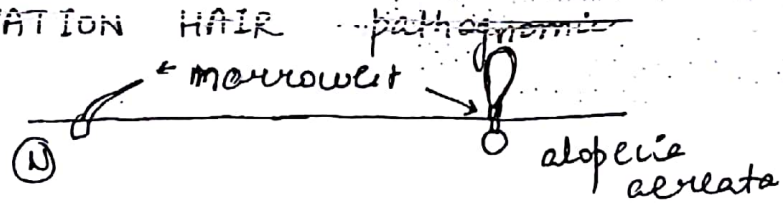
2) ALOPECIA TOTALIS

↳ loss of complete hair of scalp

3) ALOPECIA UNIVERSALIS.

↳ loss of total body hair

4) EXCLAMATION HAIR ~~pathognomic~~



! exclamation mark

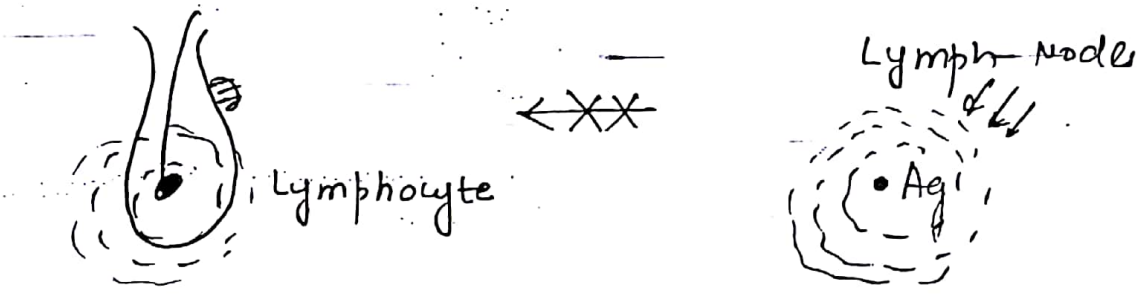
5) PRESENCE OF ATOPY

6) NAIL CHANGES (Regular pitting)

TOTALIS/ UNIVERSALIS

Contact ↑ Sensitizer

- Di Nitro Chloro Benzene (DNCB)
- Di-Phen Cyprone (DPC)
- Squaric Acid Di-Butyl-Ester (SADBE)



TRICHOTILLOMANIA

AIIMS MAY 2017

→ Obsessive compulsive Disorder of hair pulling

→ Patchy Hair Loss ↑ hairs of varying length in patch more on vertex & Dominant Hand side

→ TONSURE/ FRAIR TUCK SIGN:-
Loss of VERTEX & sparing of side

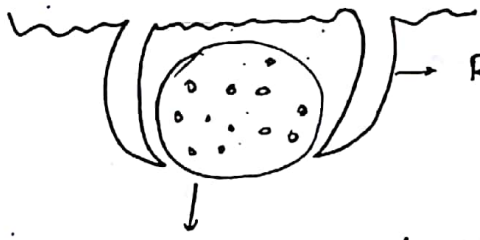
- HISTOPATH :- follicular H'ge

LICHEN NITIDUS AIIMS May 15

- Pin point papule on dorsum of hand + genitals
- asymptomatic
- self resolving

HISTOPATH

CLAW + BALL appearance
Clutching the ball +



Rete go down + curve inwards like a claw.

Lymphocytes look like Ball

FUNGAL DISEASES

1) PITYRIASIS VERSICOLOR

- powdery scale
- various color

caused by Malassezia furfur
Now Malassezia globosa

Both are commensal around hair follicle in the seborrhoeic areas.

↓

Chest
Back
Face

↓ overgrowth of malassezia

↓

release Azelic Acid [Tyrosinase Inhibitor]

↓

Perifollicular Hypopigmentation

↓

Later fuse to form Large Patches [asymptomatic]

Sometimes other colours [Brown, Red, Yellow] may also be seen

SCRATCH SIGN-

scratching of Lesions make powdery scale prominent in the scratch Line.

Δ -

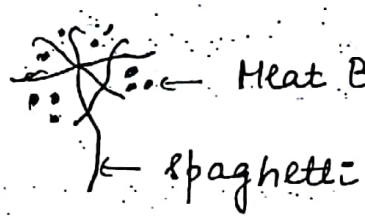
Scrapping

stain ⊂ KOH

↓

Spaghetti + MEAT BALL or

BANANA + GRAPES appearance Q.



Rx - 1) Oral + topical Azole group of drugs

2) Topical selenium sulphide

3) Oral griseofulvin / Oral Terbinafine do not work

* Organism is killed immediately but pigmentation problem takes longer (4-8 wks) to resolve.

27 **SEBORRHEIC DERMATITIS** (SD)

→ Malassezia overgrowth ⊂ Itching + yellow greasy scales in seborrhoeic areas.

→ SD in infants = **CRADLE CAP**

→ Extensive SD — HIV

Parkinson's Disease

Rx = Similar to pityriasis versicolor

3) CANDIDIASIS

Candida albicans (OPPORTUNISTIC Fungus)

↓

- DM
- Moisture
- Immunosuppression

TYPES

a> ORAL / THRUSH

↓

White creamy / white curdy plaques in oral cavity can be scrapped off (pseudomembrane)

↓

Leukoplakea can't be scrapped off.

b> CANDIDAL BALANITIS

↓

glans inflammation

Erythematous itchy papule or erosion on glans often ī repeated washing ī water.

c> CANDIDAL BALANO-POSTHITIS

↳ prepuce

Fissures on prepuce

If recurrent → s/o uncontrolled DM Q.

D> CANDIDAL INTERTRIGO

↳ fold

↓

Bet^n

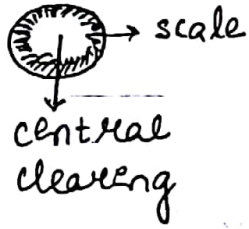
Moist erythema in folds ī satellite lesions

Δ -

- Smear
- Staining ⊼ KOH • grams stain to show budding yeast

4) **TINEA**

causes annular Lesions
↓
⊼ itching • peripheral scale



A/⊼ moist skin

Groin → Tinea cruris (Jack itch, Dhobi itch)

Body → T. corporis

Scalp → T. capitis

Feet → T. pedis / Athlete's Foot

Nail → T. unguium (onychomycosis)

Hand → T. manuum

Steroid modified Tinea → T. incognito

ONYCHOMYCOSIS (T. of nail)

→ Yellow Discolouration

→ Thickening of nail

→ Subungual hyperkeratosis

T. PEDIS

3 TYPES

INTER-DIGITAL — Trichophyton Rubrum

CHRONIC PLANTAR SCALING (MOCCASIN FOOT) — Trichophyton Rubrum

BULLOUS T. PEDIS — Trichophyton Mentagrophytes Q

Tinea caused by Dermatophytes

3 species

	TRICHOPHYTON	MICROSPORUM	EPIDERMOPHYTON
		Keratophilic	
Hair	✓	✓	⊗
Skin	✓	✓	✓
Nail	✓	⊗	✓

SLIDE 15

T. CAPITIS	ALL OTHER TINEAS
M/c organism	M/c organism
India - T. violaceum	T. Rubrum
World - Microsporum canis	
US/UK - T. Tonosurans	
DOC - Griseofulvin	DOC - Terbenafen

T. CAPITIS

Easy pluckability of hair in a **child**

Creates patchy hair Loss

ECTO-THRIX

↓

Caused by Microsporum

ENDO-THRIX

↓

Caused by Trichophyton

(16) T. CAPITIS

NON - INFLAMMATORY (Non-scarring alopecia)	INFLAMMATORY (scarring alopecia)
• GREY PATCH Microsporum canis M. audouinii M. ferrugineum	KERION (Boggy swelling) Poor prog T. Mentagrophyte M. canis T. verrucca
• BLACK DOT T. Tonsurans/ T. violaceum	—FAVUS (yellow scutulum) T. schoenleinii

5) SPOROTRICHOSIS

Ioc - Skin Biopsy

↓

H/P → Cigar shaped yeasts

↓

ASTEROID- Bodies in dermis

*

Rx = ORAL ITRACONAZOLE → DOC

Other — KI

↳ Amphotericin B.

6> EUMYCETOMA

Swollen foot ~ Discharging sinuses
In a farmer walking BARE FOOT.

TRIAD Q.

TUMEFACTION (swelling

SLIDE-1

Swollen Foot ~ Discharging sinus

- Botryomycosis [Staph]
- Eumycetoma
  Madurella mycetomatis
  ↓
  BLACK GRAINS
- Actinomycotic mycetoma
  a> Actinomadura madurae M/C
  b) Nocardia
  c) Streptomyces
  ↓
  WHITE GRAINS

EUMYCETOMA

Oral Itraconazole

KI.

Amputation.

ACTINOMYCOTIC MYCETOMA

Q. WELSH Regimen → Amikacin +
Rifampicin +
Cotrimoxazole.

7> CHROMOBLASTOMYCOSIS

Presents as cauliflower masses on feet in a barefoot farmer after a thorn prick

Smear shows → naturally yellow spore
SCLEROTIC BODIES
MURIFORM "
MEDLAR "
COPPER - PENNY

DOC- Oral ITRACONAZOLE + Sx excision of Mass.

VIRAL DISEASES

I) HPV

causes warts

- Warts comes on Non-genital skin → VERUCA
  - V. VULGARIS → HPV 2
  - PLANA → HPV 3, 10
- Genital skin (STD) → CONDYLOMA ACUMINATA
  - means pointed → HPV 6, 11

GENITAL WARTS

Imiquimod - Immunomodulator - DOC

Podophyllin, Podophyllotoxin } Anti-mitotics

(purified extract of podophyllin)

IMIQUIMOD is a TLR-7, TLR-8 agonist

↓

hence, activates Langerhans cells

VERRUCA

- Burning (Cautery)
- Freeze - cryotherapy (Liquid N_2)
- Acids [Salicylic acid - DOC, Trichloro acetic acid]

GENITAL WART In ♀

- Trichloro acetic acid
- Cryotherapy (BETTER ANS)

PLANTAR WARTS Q

- SUPERFICIAL MOSAIC — HPV-2
- DEEP MYRMECIA — HPV-1

Q BUSCHKE – LOWENSTEIN TUMOUR

Big cauliflower mass

mutated HPV-6, 11 → Creating Low grade Cauliflower shaped. SCC/ Verrucous carcinoma

SEBORRHEIC WART/ KERATOSIS.

BASAL CELL PAPILLOMA Q

Misnomer.

Sign of ~~Ageing~~

Due to benign proliferation of keratinocyte.

LESSER TRELAT SIGN

Sudden eruption of multiple seborrheic keratosis suggest underlying malignancy
(Adeno carcinoma of stomach > colon)

2) HUMAN HERPES VIRUS [HHV]

HHV 1 = HSV 1
cause **Herpes Labialis**
group blisters on the lip. + peri-oral area.
reactivating ~ fever [FEVER BLISTER]

HHV 2 = HSV2
causes **Herpes Genitalis**

ECZEMA HERPETICUM / KAPOSI'S VARICELLIFORM ERUP°
Disseminated HSV-1 in an atopic eczema patient who is immunosuppresed + is inoculated by HSV-1 through another patient
Also seen ~ DARRIER's DISEASE
P. FOLEACEOUS PI.

HHV 3 = Varicella Zoster virus
1st episode = Varicella [chicken pox]
Reactivation = Herpes Zoster [shingles]

Varicella presents as Vesicles (Dew Drop on Rose Peta) ~~pustule, finally crust (non-contagious)~~
It has polymorphic, centripetal lesions

After varicella gets → VZV remains hidden in spinal & cranial ganglia. & reactivates along dermatome. (immunosuppression)

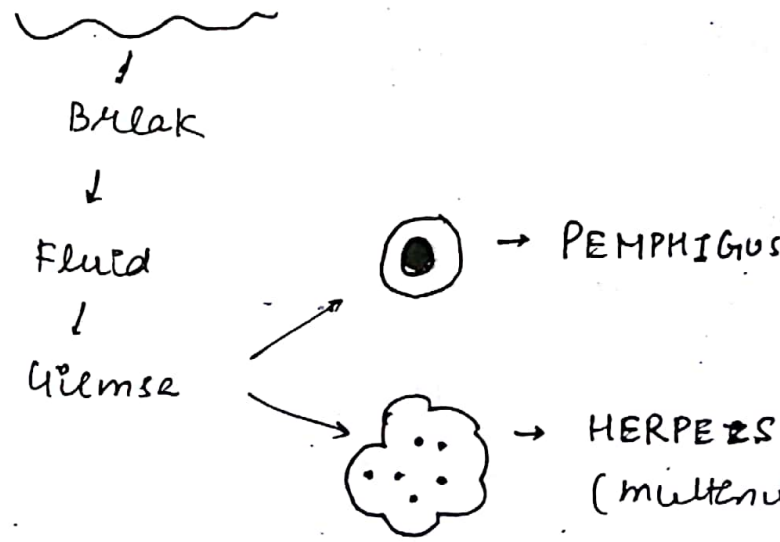
↓

HERPES ZOSTER

COMPLICATION OF HERPES ZOSTER

1) Post-herpetic neuralgia (PHN)
   defined as pain even after 4 weeks of resolution of herpes zoster
   DOC = GABAPENTIN

TZANCK SMEAR



PITYRIASIS ROSEA

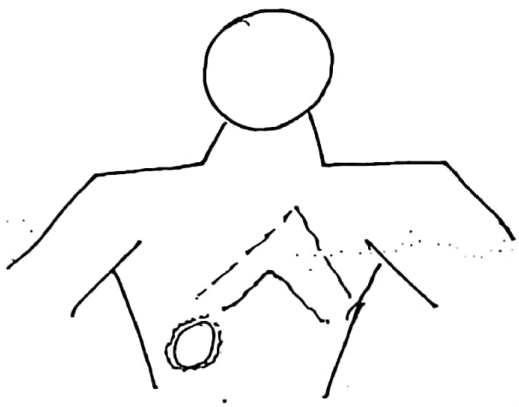
HHV 7 > HHV6

Rarely, Drug Induced ?

1st Lesion of Disease ⇒ HERALD PATCH / MOTHER PATCH

↓

annular
Itchy
Peripheral scale (collarette)
TRUNK



Rest of lesion come in straight line meeting in centre

↓

FIR TREE / CHRISTMAS TREE pattern. (differentiate with Tinea)

→ Self limiting in 4-10 wks
→ Acyclovir shorten disease duration.

3) MOLLUSCUM CONTAGIOSUM

caused by MCV (DNA virus)

← Inclusion Body

↓

molluscum Body
[Henderson Peterson Body]

C/F - Shiny umbilicated dome shaped papule
- children, face
- genital molluscum → STD

Rx - same as for warts

PARASITIC DISEASES

I> SCABIES

Caused by female scabies mite
enters finger web or genitals through BURROWS
↓
S-shaped

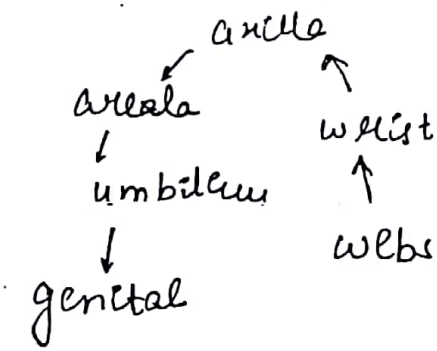
Avg. no. of mite on skin = 12

C/F - Itchy Papules in adults
Nocturnal Itch
Facial sparing in adults

Scabies → poor hygiene disease
[WATER WASHED DISEASE]

I.P. = 3-4 weeks → 1st episode
1-2 days - later episode
(due to memory T cells)

CIRCLE OF HEBRA



Rx - For pt + close contact + clothing

Rx for INFANT SCABIES

→ Face is involved
→ Palm / sole involved
→ Papules + vesicles

Rx - DOC → 5% Permethrin - single overnight application
[adults, infants, ♀]

other Rx -

Topical
1) Benzyl Benzoate 25%
2) Lindane
3) γ Benzene Hexachloride
4) Crotamiton
5) Sulphur

Oral → Ivermectin → 2 doses
14 days apart
(200 µg / kg / dose)

NODULAR SCABIES

Hypersensitivity response to scabies mite

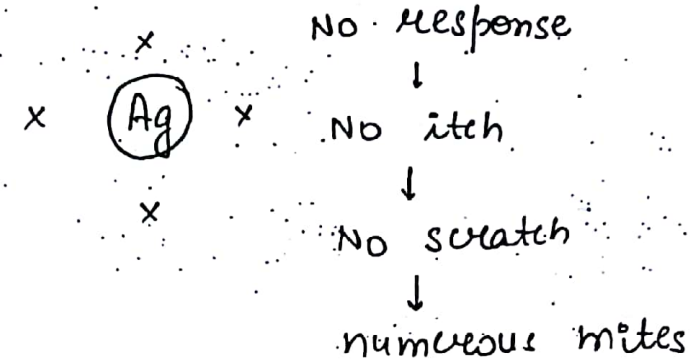
INTRALESIONAL STEROIDS

5% PERMETHRIN → Ag → more inflammation.
↓
more elevation (nodule)

Seen on genitals

NORWEGIAN / CRUSTED / KERATOTIC SCABIES

HIV ⊕ pts



c/F - Hyperkeratosis

R_x - Oral Ivermectin + Topical Permethrin.

PEDICULOSIS

caused by LOUSE

HEAD LOUSE

Long, slender, louse lays eggs (Nits) on scalp hairs.

Nit → adult → Itch

P. CAPITIS

R_x - 1% permethrin

BODY LOUSE

causes pediculosis corporis.

VAGABOND'S DISEASE

Not on Body, But on clothing → Bite skin → returns back to cloth

R_x = Disinfect of cloth

PUBIC LOUSE / CRAB LOUSE

Short, stocky louse

causes P. PUBIS

Louse bite mark → called Maculae cerulae

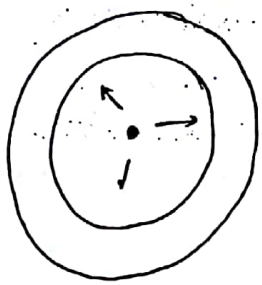
R_x = 1% permethrin

5% Permethrin

Oral ivermectin

III) ERYTHEMA CHROMICOM MIGRANS

Bite by a hard tick (IXODES)
↓
deposits
BORRELIA BURGDORFERI into skin
↓



red rings around Bite ⟹ TARGET LESION

↘ ANNULAR LESION

↓
Later Pt develops
LYME'S DISEASE

IV) PKDL

Post-Kalazar Dermal Leishmaniasis
Bite by a sandfly
↓
deposits Leishmania

- ↙ cutaneous Leishmaniasis → CRUSTED ULCER
- ↘ Visceral Leishmaniasis → Fever ± hyperpigmentation [KALA-AZAR] → after many yrs of Kalazer → PKDL

PKDL

Hypopig — Nodular

(B) forms resemble Leprosy

H/o Past fever in Q ⇒ Suggests PKDL

Δ of PKDL-

Crush smear (giemsa stain) for LD Bodies

Slit Skin smear (") for LD Bodies

DOC -

Oral MILTEFOSIN

V) INSECT BITE HYPERSENSITIVITY

Excessive Immune Response to Simple Insect Bite

→ Lesions on exposed areas.

→ ↑ in Rainy Season

ECZEMA / DERMATITIS

1) ATOPIC ECZEMA

TH2 mediated inflammation (B cell)

Δ :- Hanifen & Rajka criteria

SLIDE - 18 MAJOR CRITERIA - Any 3 out of 4.

1) Itching → Hallmark
2) Typical Sites
   → Extensor Dermatitis [children] 0-2yr
   → Flexor Dermatitis [Adults & children] 2-12yrs
3) Personal H/o / Family H/o of atopy
4) Chronic relapsing course

classical flexor involved : ante-cubital fossa
" extensor " children = cheek
[HEADLIGHT SIGN]
↓
sparing of nose & peri-oral area, ~~peri-orbital area~~

ACUTE STAGE → Oozing, crusting

CHRONIC STAGE → Lichenification

MINOR CRITERIA

1) Dennie MORGAN FOLD -
   Extra crease on Lower eyelid.

2) Pityriasis Alba -
   - Hypopigmented patch ± fine scaling on cheek in children
   - often recurrent / non-itchy

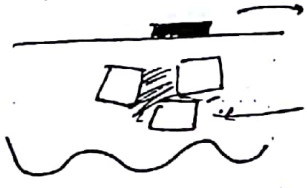
3) Peri-orbital Pigmentation

4) White Dermographism - Q
   Vasoconstriction on scratching.

5) Plantar Hyperlinearity

6) Palmar Hyperlinearity

7) Ichthyosis Vulgaris (Dry skin)

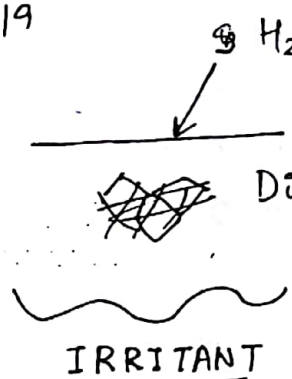
Rx -
1)  → Moisturising cream (lipid) / Lipid replacement

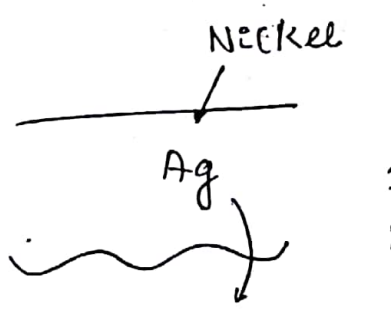
2) TH2 cell Inhibitors
   Localised D. → generalised Disease

Localised Disease	Generalised Disease
Steroid	steroid
Calcipotriol	Cyclosporine
Tacrolimus	Azathioprine
	Phototherapy
	Mycophenolate

II> CONTACT ECZEMA

SLIDE-19





1st exposure sensitizes
next exposure cause
clinical disease

IRRITANT	ALLERGIC
Not Immunological	Immunological Type-4 hypersensitivity
Due to toxic chemical	
No sensitization required direct clinical phase	sensitization phase 1st followed by clinical phase
Memory cells not involved	Memory cells involved In predisposed person.
Eg. 1) Detergents (HOUSEWIVES ECZEMA) 2) Acid & alkali Burn.	Δ - PATCH TEST Eg. 1) nickel → M/c overall 2) PPB → in hair dye 3) vegetable - M/c in Indian female

READINGS OF PATCH TEST

1st Read ↓ — 2nd Read ↓ — For neomycin, PPD - metals

Day 0 — Day 2 — Day 4 — Day 7 ↑ 3rd Reading

~~OCU~~ OCCUPATIONAL CONTACT DERMATITIS

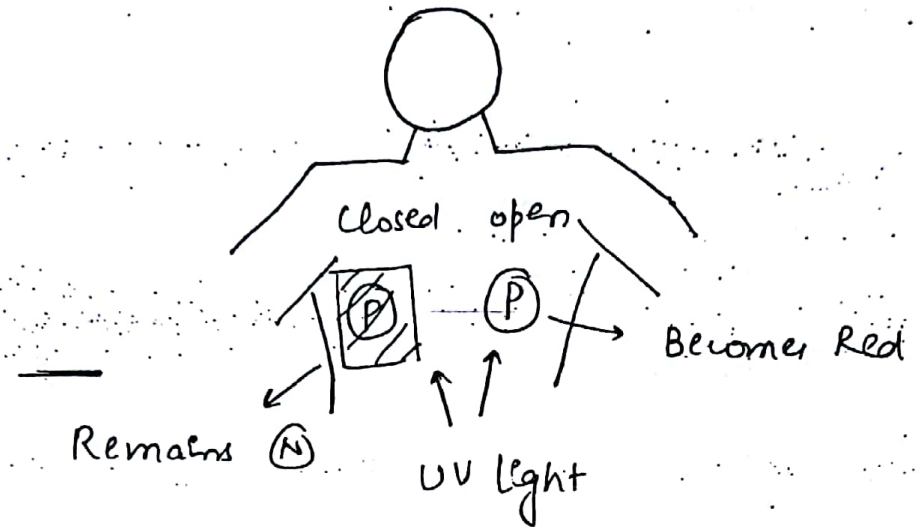
A) Air Borne Contact Dermatitis / Phytophoto dermatitis

seen in farmers.

exposed to Ag - from Parthenium Plant

coupled ± some exposure

A- Photo PATCH TEST AIIMS - May 16.



DOC - AZATHIOPRINE

B) CEMENT DERMATITIS

Ag → Potassium Dichromate

c) HAIR DRESSERS

Ag - PPD

D) TEXTILES

Ag - AZO DYES

III) POMPHOLYX

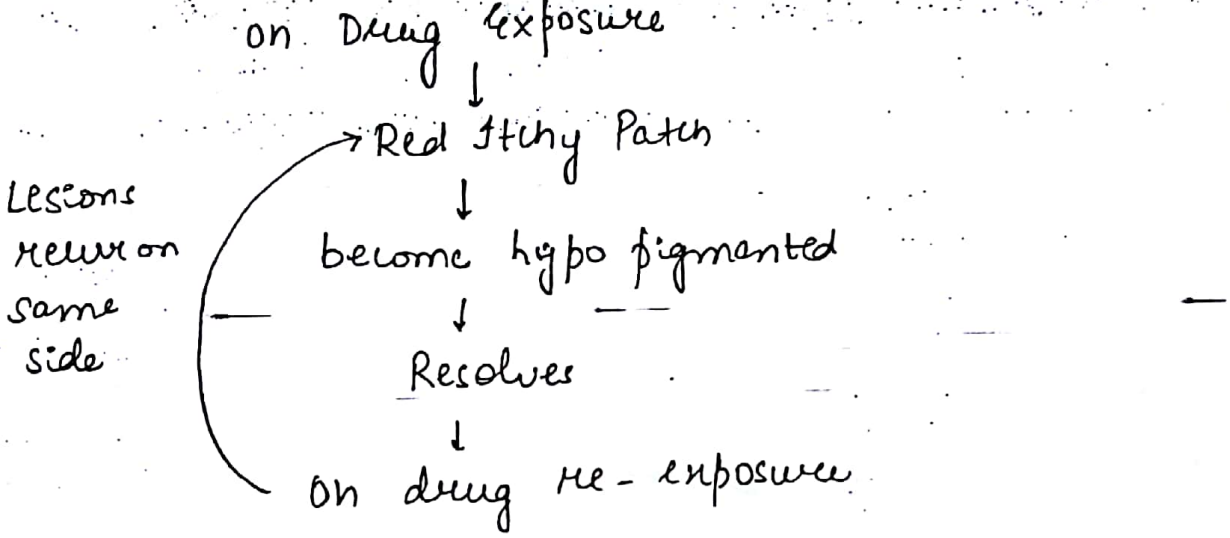
→ Form of HAND & FOOT ECZEMA ~ severe spongiosis

→ presenting as Deep seated Blisters on Palm & Soles ~ SAGO ~~GRAIN~~ like feel, severe Itching

DRUG REACTION

A> FIXED DRUG ERUPTION

- M/c cause - NSAIDS
  other tetracycline
  Metronidazole
  Sulphonamides



COMMON SITE = Lips, Genitals

Pigmentation is Bluish-Grey

[BROWN PIG. ON NOSE Post Fever = CHIK Sign seen in chickenguniya]

Bullous FDE is seen in Genitals.

On Genitals it comes ~~as~~ as recurrent blister / ulcer healing ↓ hyperpigmentation
(not ↓ Herpes genitalis)

B> **ERYTHEMA MULTIFORME**

C/F- Target Lesions ∈ 3 ZONES 

ETIO-

H/cc - HSV

other - Mycoplasma
Idiopathic
Drugs

Rx- self Limiting

BULLOUS EM ⇒ centre most area gets blister

2 types of EM

EM MINOR	EM MAJOR
Few target Lesions	Many target Lesions
No mucosal involvement	one mucosa involvement (oral)

C> **STEVENS JOHNSON SYNDROME (SJS)**
TOXIC EPIDERMAL NECROLYSIS (TEN)

ETIO → mainly Drug Induced
→ sometime mycoplasma

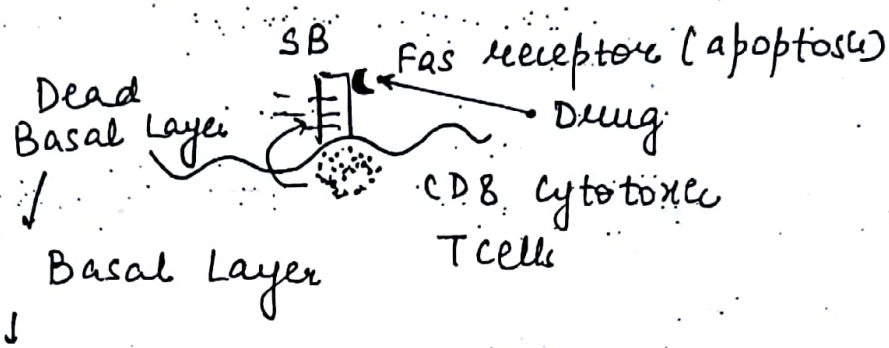
C/F- 1) TARGETOID LESION or atypical target Zones

2) ≥ 2 mucosa involved

Depending on % Body

<10%	10-30%	>30%
SJS	Overlap	TEN

TEN (LYELL'S SYNDROME)



cut in Basal Layer

↓

Blister

↓

Death extends to full epidermis (necrosis)

↓

falls off

↓

Barrier Loss

R_x- 1) FAS Ⓡ Antagonist ⇒ IVIg

2) CD8 cell Inhibitor ⇒ CYCLOSPORINE

~~NICHOLS~~ * NIKOLSKY SIGN

Tangential movement ε finger create epidermal movement ε a raw area underneath.

BLISTERING DISORDERS

A) IMPETIGO

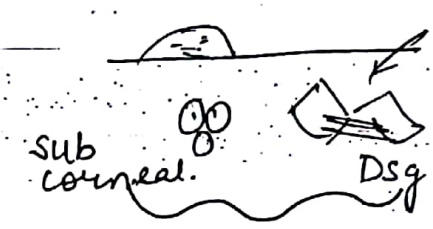
NON - BULLOUS

IMPETIGO CONTAGIOSA

- → Staph > Gr A strepto
- → Commonest skin infec" in children
- → Honey coloured crusts around mouth & nose

BULLOUS

PEMPHIGOUS NEONATORUM



E. Toxin. disseminates in Blood → SSSS

(Exfoliative) or (epidermolytic)

DIF = (-ve)

HYPOPYON SIGN - pus in Lower 1/2

Dsg 1 → Seborrheic areas
↘ mucosa - absent

Child

RITTER's Disease

Presents only in children as scale crust lesions at seborrheic areas [out mucosal involvement

Fever (+)

Nikolsky sign (+)

B) PEMPHIGUS

1) P. FOLIACEOUS

Seen in adults

No fever

Scale / crust in seb. area

No mucosa

2) P. ERYTHEMATOSUS

also called SENEAR- USHER SYNDROME

variant of PF

PF + SLE

↓

PE

3) P. VULGARIS

Dsg 3 Disorder

present all over body

also tnt in mucosa

→

Deep wounds open painful wound, slow to heal

Severe mucosal involvement.

2 TYPES

MUCOSAL	MUCOCUTANEOUS
Only Dsg 3 involved	Both Dsg 1 + 3 involved

4) P. VEGETANS

↳ like a vegetable

↓

— cauliflower-like masses in flexures

variant of PV.

Rarest pemphigus

5) PARANEOPLASTIC PEMPHIGUS

Resembles P. vulgaris but ⊂ Internal Malignancy

↓

M/c → NHL

Others → CLL

↳ Castleman Disease

↳ Thymoma

↳ Retroperitoneal sarcoma

Nikolsky sign ⊕ in all pemphigus.

R_x = 1) Systemic steroid - DOC

↓

High Dose

2) Non-steroidal immunosupp.

eg. Azathioprine, Mycophenolate, Cyclophosphamide

3) Rituximab → antibody
↓
monoclonal
against CD_{20} receptor on B cell surface

C> **BULLOUS PEMPHIGOID**

Tense/ Itchy blisters
Blisters come on red/ urticarial skin
extremities/ trunk
Elderly patients

H/P - Subepidermal blisters + Eosinophils

BULLA SPREAD SIGN/ LUTZ SIGN

Bullous Pemphigoid

P. vulgaris

ASBOE - HANSEN SIGN

Variant of Bulla sign
Pressing on top of blister not from one side

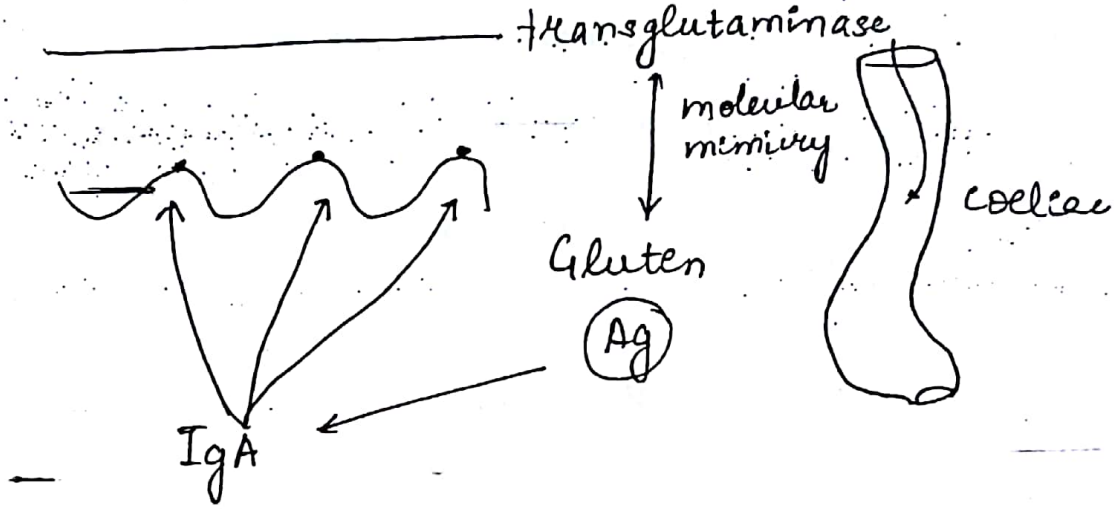
BP in ♀ ⇒ ~~GESTAT~~ PEMPHIGOID GESTATIONIS
(Herpes gestationis)
× → miscarriage
M/C site - Periumbilical Blisters

D) **DERMATITIS HERPETIFORMIS**

Papillary tip Blisters

↑ " " ↓ Microabscess Q

SLIDE - 20



DIF → IgA @ tip of Dermal Papilla in a granular pattern

Severe Itching

Grouped papulovesicles on elbow

DOC = Dapsone + Gluten free diet

E) **LIGA / Chronic Bullous Disorder of Childhood**

CBDC

↓ ADULTS

↓ children

→ Itchy tense blisters in a cluster of Jewel or string of Pearl appearance

→ 50% have mucosal involvement

→ DOC - DAPSONE

F) HAILEY / HALLEY DISEASE

Benign Familial Pemphigus

Age of presentation - 2-4th Decade

Flaccid Blisters in Flexors rupturing easily to create erosions & painful fissure.

H/P – Dilapidated Brick Wall Appearance

Level of Blister: SUPRABASAL

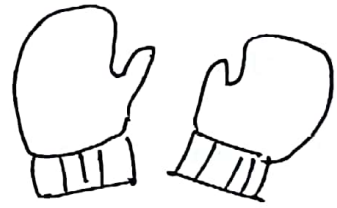
G) EPIDERMOLYSIS BULLOSA

(Trauma Induced Blister) —

Blisters @ site of handling

MITTEN HAND DEFORMITY seen in EBD.

Δ - Electron microscopy



VASCULAR LESIONS

CONG.

VASCULAR TUMOURS

Infantile Hemangioma
(strawberry hemangioma
capillary "
cavernous ")

Grows Rapidly - till 9 month
then plateau phase then
resolves

Blanches on pressure

Propranolol is Doc. if
→ Rapidly ↑
→ Ulcerating
→ near eye

VASCULAR MALFORMATION

- Port wine Stain

Persists throughout life
(portwine stain on glabella -
called a salmon patch
resolves)

Doesn't blanch

Associated with Sturge weber
Syndrome

Pulse Dye Laser (PDL)

STURGE WEBER SYNDROME

I/L Portwine Stain +
I/L Eye Involvement +
Same side CNS involvement

ICTHYOSIS

→ Generalised Dry Skin
→ Fish like scales
Ichthyos → means fish

A) CONGENITAL ICTHYOSIS

(I) ICTHYOSIS VULGARIS

H/c type
Flexures spared
Palms/Soles Involved
Small scales
Association - atopy
H/P → Absent granular Layer

(II) X- LINKED RECESSIVE ICTHYOSIS

Flexures Involved
Palms/Sole spared
Steroid sulphatase deficiency
Large Brown scales

(III) LAMELLAR ICTHYOSIS

Entire skin involved
Plate like Large scales (Lamella - Plate)
Born ∴ "COLLODION membrane" Q.

B) ACQUIRED ICTHYOSIS

CRF

AIDs

Hypothyroidism

Hansens

Drug Induced (Nicotenic acid, clofazemines