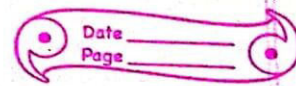


Dermatology

1. Acne Vulgaris
2. Alopecia
3. Pityriasis alba
4. Tinea versicolor
5. Melasma
6. Acanthosis nigricans
7. Atopic dermatitis
8. Contact dermatitis
9. Urticaria
10. Psoriasis
11. Lichen planus
12. Pityriasis rosea
13. Pemphigus
14. Dermatitis herpetiformis
15. Epidermolysis bullosa
16. Erythema multiforme
17. Darrier's d/s
18. Leprosy
19. Cutaneous TB
20. Dermatophytes
21. Scabies
22. Syphilis
23. Donovanosis
24. LGV
25. Gonococcal infection
26. HSV
27. Molluscum contagiosum
28. LE
29. Cutaneous reactions to drugs
30. Tuberculous sclerosis
31. Actinic keratosis

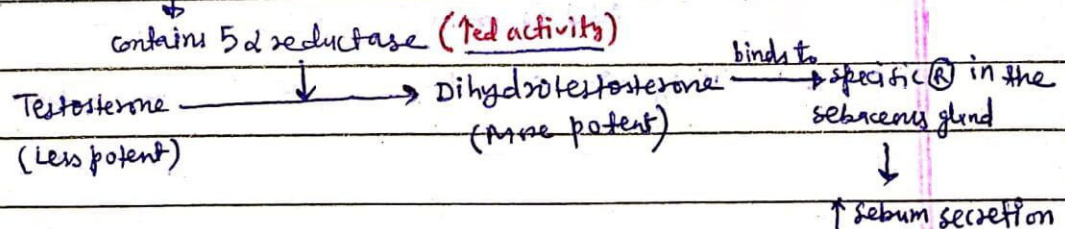


ACNE VULGARIS

- a disorder of pilosebaceous glands.
- Mainly affects adolescents, but may +nt into adulthood.
- characterised by Papules, Pustules, nodules, cyst and special lesions called comedones
 - closed (white head) (CW)
 - open (Black head)
- ↑ androgen production at puberty results in ↑ lipid rich sebum secretion which causes induction of propionibacterium.
- Propionibacterium causes lysis of triglycerides by producing lipase and release FFAs.
- FFAs cause follicular plugging and formation of acne vul.
- ABs used in acne are - Tetracycline, Minocycline, Doxycycline, Erythromycin, Clotrimoxazole, Dapsone.
 - Most effective
- T/T
 - Predominantly comedonic = Retinoids + Benzoyl peroxide
 - Predominantly inflammatory = AB + Benzoyl peroxide
- DOC for nodulocystic acne = Oral isotretinoin (13-*cis* retinoic acid)
 - other indications
 - Severe acne (>25 lesions)
 - Moderate acne not responding to oral AB (Refractory acne)

→ sebaceous gland activity is controlled by androgens.

Though most pts w/ acne have (N) levels of circulating androgens, their sebaceous glands are unusually sensitive to androgens due to an enhanced end organ sensitivity.



→ Vulgaris → means Common.



ALOPECIA

FOCAL

DIFFUSE

Rash in area of hair loss

YES

- Severe itching
- Painful boggy swelling
- Multiple discharging sinus

TINEA CAPITIS

Non cic. Alopecia

NO

Exclamation mark hair at the periphery of lesion

+nt

Not +nt

ALOPECIA AREATA

- young adults
- associated hair loss on beard, eyebrows, eyelashes
- Gray hairs in area of hair loss
- Nail changes (Pitting, Thinning)

H/O tying hairband very tightly

TRACTION ALOPECIA

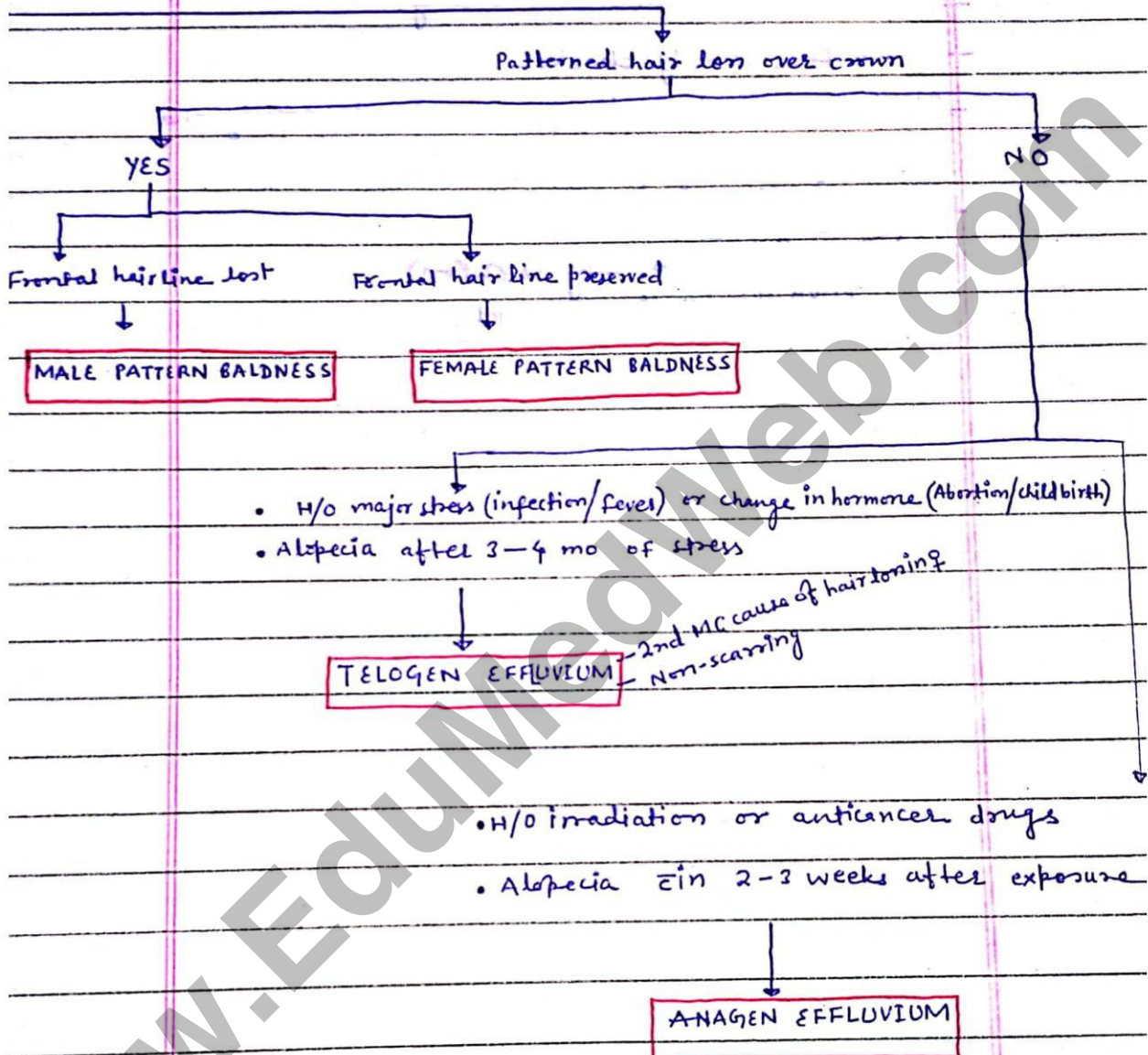
• Causes of cicatricial (scarring) alopecia - CLL - SAD

LP, Lupus vulgaris, Sarcoidosis, Aplasia cutis, DLE

• SLE can cause both cic. as well as Non cic. alopecia.



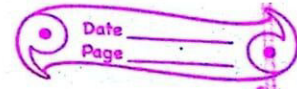
• MC type of hair loss in \rightarrow f \rightarrow = Androgenetic Alopecia or Patterned hair loss.





PITYRIASIS ALBA (PITYRIASIS SIMPLEX)

- extremely common cause of hypopigmentation in childhood.
- a chronic condition that affects children (6-12 years).
- White macule or patch which is scaling.
- affects face usually cheeks.
- Patch, over the time disappears spontaneously and recurs frequently.
- T/T
 - No T/T required
 - only reassurance
 - Tacrolimus (speeding resolution)
 - PUVA therapy (in exceptionally severe cases)



TINEA VERSICOLOR (PITYRIASIS VERSICOLOR)

- Malassezia furfur
- scaly hypopigmented macule (may be hyperpigmented also), Macules start around hair follicles and then merge $\bar{\text{c}}$ each other
 - Large areas
- Scales over trunk (mainly chest & back)
 - Furfuraceous or rice powder like
- Lesions are recurrent in nature.
- Examination of scales
 - Spaghetti and Meat ball appearance (characteristic) in 10% KOH.
 - SDA $\bar{\text{c}}$ olive oil \rightarrow Fried egg colonies
 - Wood's lamp examn. \rightarrow Golden yellow fluorescence
- T/T
 - systemic \rightarrow Ketoconazole, Fluconazole or Itraconazole
 - Topical
 - Azoles \rightarrow Clotrimazole, Econazole, Miconazole, KTZ
 - Others \rightarrow Selenium sulfide, Sod. thiosulphate, Whitfield ointment (3% Salicylic acid + 6% Benzoic acid)

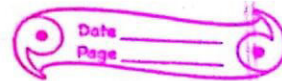


MELASMA

- MC cause of facial pigmentation in Indian patients.
- Brown macular pigmentation on the malar area of face, forehead and sometimes chin.
- usually affects young and middle aged women.
- When melasma results from pregnancy it is referred as chloasma.
- 2 major etiological factors
 - Estrogen (Pregnancy/oral CPs)
 - UVB (sun exposure)
- T/T → Combination of topical hydroquinone (2-4%), retinoic acid and a topical corticosteroid.

Adv

GETLITE Cream (HQ-topical 2%, Mometasone-topical 0.1%, Tretinoin topical 0.025%)



ACANTHOSIS NIGRICANS

- Brown to black, velvety hyperpigmentation of the skin.
- usually found in body folds, such as posterior & lateral folds of neck, axilla, groin, umbilicus, forehead.
- typically occurs in Pt. < 40 yrs of age.
- H/p Papillomatosis is characteristically seen; however, there is NO hypermelanosis and there is only mild acanthosis, if present.

- a/w $\left\{ \begin{array}{l} \text{Obesity (MC)} \\ \text{Endocrineopathies} \rightarrow \text{Hypo/Hyper th., insulin resist. diabetes,} \\ \text{Cushing's d/s, PCOD, Bloom syndrome} \\ \text{Internal malignancy} \rightarrow \text{Gastric adenoCa.} \end{array} \right.$

⊙ HAIR-AN syndrome - in women, the triad of $\left\{ \begin{array}{l} \text{Hyperandrogenism} \\ \text{Insulin-resistance} \\ \text{Acanthosis nigricans} \end{array} \right.$



ATOPIC DERMATITIS

Syn → Neurodermatitis / Besnier's prurigo / Infantile eczema

- +ve family h/o allergic rhinitis, asthma or eczema.

- Pruritis is the MC symptom.

- Course marked by exacerbations and remissions

- clinical course lasting longer than 6 weeks

- Infantile pattern - involves face, neck, extensor surfaces of arms

- Childhood/Adolescent pattern - involves flexural skin (Antecubital fossa & Popliteal fossa)

- Cutaneous stigmata

- ↳ Perioral pallor
- ↳ Extra fold of skin beneath the lower eyelid (Dennie-Morgan folds)
- ↳ Red palmar skin markings
- ↳ ↑ incidence of cutaneous infections, particularly staphylococci.

- Δ → clinically (dry skin, severe itching, flexural lichenification, eczema)
h/o atopy in the family/pt., and raised IgE serum levels.



CONTACT DERMATITIS

1. IRRITANT CD

- syn. Occupational D. / Housewife's D.
- MC site affected = Hand.
- MC cause in Indian women = Detergent

2. ALLERGIC CD

- It is due to delayed HS (type IV HS) to a particular antigen in a sensitised individual.

- MC metal causing C.D. = Ni
- MC cause of airborne C.D. = Parthenium

- $\Delta \rightarrow$ Patch test: Application of suspected allergens as a patch, usually over upper back.
 - \rightarrow Skin HS test (delayed type)
 - \rightarrow Readings are made after 48 hrs.

The patch is removed after 48-72 hrs and reading should be taken after 20 min to 1 hr after patch removal

- T/T
 - Topical corticosteroids + oral antihistaminic (in extensive cases, short course of oral corticosteroids)
 - Airborne C.D. \rightarrow Azathioprine

② Nummular (Discoid) Eczema -

- characterized by circular or oval coin-like lesion.
- MC sites = Trunk and Extensor surfaces of extremities (pretibial skin and dorsum of hands)
- MC affects men in middle age group.

- ② Urticaria Pigmentosa — Generalized distribution of red brown macules
 — Each lesion represents a collection of mast cells in the dermis & hyperpigmentation of overlying epidermis
 — These lesions urticate on scratching. [DARTRIER sign]

URTICARIA

- Urticaria, commonly referred to as HIVES, is type I HSR.
- Basic pathogenesis = Release of Histamine.
- Lesions begin as pruritic ill defined erythematous macules & patches. They rapidly develop to form the typical lesions called wheals.

- Rounded/Oval
- Pale coloured papules/plaques
- with a halo of erythema

- Trunk is involved more commonly than extremities.
- Wheals last a few hours and resolve in 24-48 hr leaving behind \emptyset skin.
- Edema is superficial, mainly dermal layer (In angioedema, edema involves deeper layers, i.e. subcutaneous tissue)

Causes

1. Drugs — Aspirin, NSAIDs, Penicillin, ACE-i, opiates.
2. Food — Meat, Fish, Egg, Milk.
3. Systemic d/s — SLE, serum sickness, Thyrotoxicosis.
4. Pregnancy
5. Infections — HBV, HCV, TB, Helicobacter, Worm infestation
6. Physical urticaria

A. Dermographism [skin writing] — stroking of the skin causes it to wheal on the line of stroke. It is exaggerated axon reflex (Triple response)

B. Cold urticaria

C. Heat urticaria

D. Solar urticaria — in 1-30 min. of exposure to sunlight & disappear in 24 hr. on the V area of the neck and the arms may be the tinting sign in PCT, SLE, EBstein-Barr virus

E. Cholinergic urticaria (Micropapular urticaria) — Urticaria develops in response to a rise in the core body temperature in 2-30 min. The

lesion lasts for 20-90 min. It is a/w cholinergic sympathetic fibres.

- They are provoked by — Generalized heating
 Exercise and physical exertion
 Emotional stress
 Hot food

T/T: Only Antihistaminics

- For rapid relief of acute severe urticaria & laryngopharyngeal edema 0.5 to 1ml of epinephrine should be used.
- A short burst of corticosteroids is reserved for very severe episodes of urticaria.



PSORIASIS

- chronic dermatoses characterised by unpredictable course of remission and relapse and presence of well defined, silvery white scaly papules and plaques on erythematous base.
- Psoriasis is aggravated in winter.
- Pathology - There are 2 vital pathological processes in psoriasis

1. Epidermal proliferation

2. Dermal inflammation - manifested by (i) Increased No. of dilated tortuous capillaries in dermal papillae. (ii) Lymphocytes in superficial dermis

Manifested by (i) Parakeratosis - Persistence of nuclei in str. corneum.

(ii) Hyperkeratosis - Thickening of str. corneum (on Palmar and Plantar psoriasis)

(iii) Munro's microabscess - str. corneum shows collection of polymorphs.

(iv) Acanthosis - Thickening of str. spinosum.

(v) Suprapapillary thinning of str. malpighi.

- The basic defect in psoriasis is rapid replacement of epidermis in the site of lesions, i.e., epidermal cell cycle becomes 3-4 days duration instead of normal 28 days duration.

Lesion are Non-pruritic or only mildly pruritic.

C/F of Psoriasis (Psoriasis Vulgaris)

- Typical lesion = Nummular round plaque which has following features
- (i) Well defined (ii) Profuse, Silvery white, powdery scales (Candle drop scales) - loosely adherent and easily drops (iii) Bright red erythematous base
- (iv) Plaque is often surrounded by a hypopigmented halo → Ring of wormholes



③ Scaling is cont or minimal

- (i) Flexures (Groins, axillae, inframammary folds, Vulva, Gluteal cleft)
- (ii) Glans in uncircumcised patients
- (iii) Guttae psoriasis

③ Not found in Psoriasis

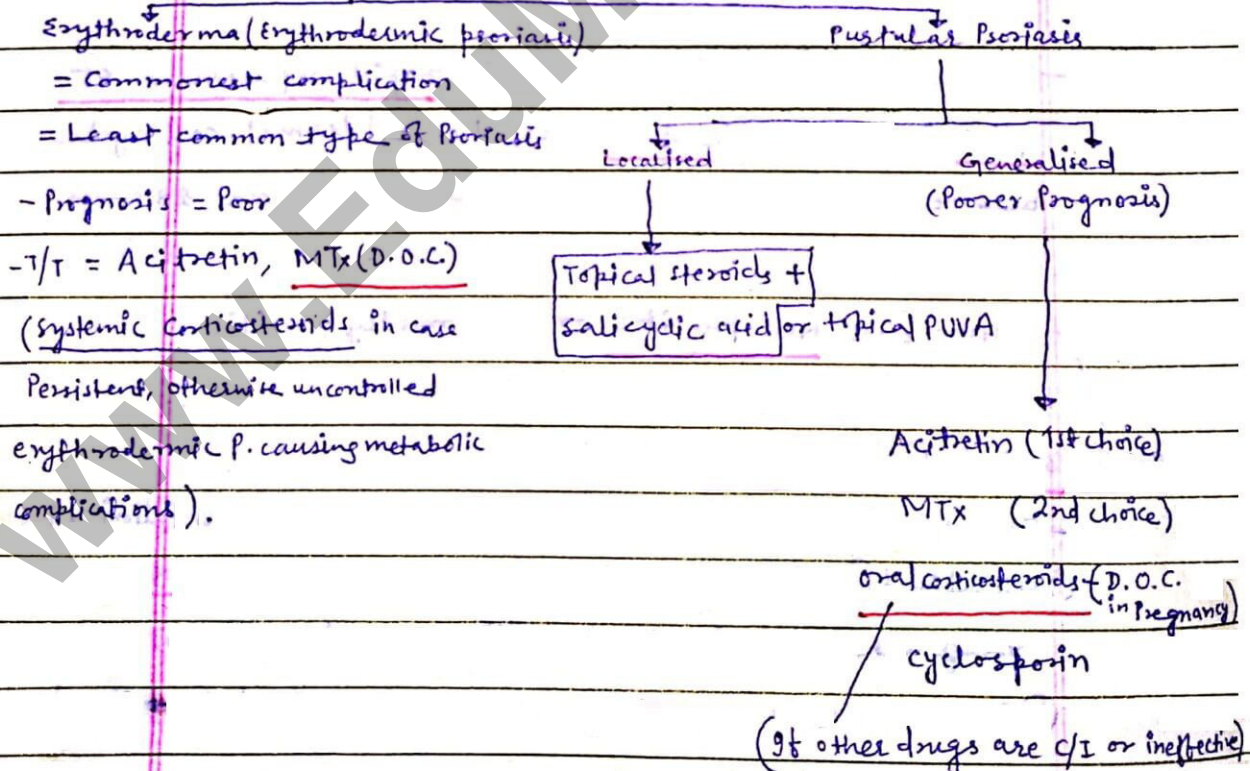
Psoriasis NOT (MAF) रोग

- ~~Mucosal involvement~~
- ~~Alopecia~~ (despite the involvement of scalp)
- ~~Face involvement~~

③ Uncommon in Psoriasis

- Joint involvement
- Pruritis

③ Complications of Psoriasis

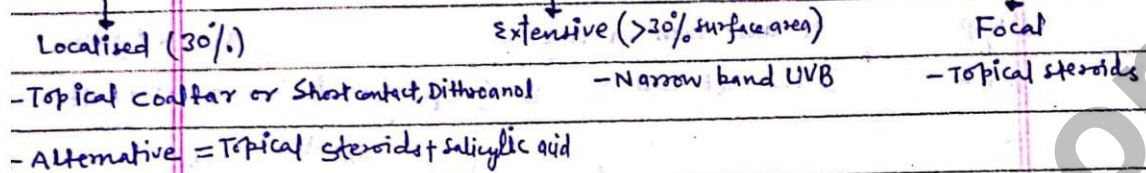




② Clinical variant

1. plaque type (Plaque Psoriasis/Psoriasis vulgaris) = MC

T/T:



2. Flexural P.

3. Scalp P.

4. Penile P.

5. Palmo-plantar P.

6. Guttate P. Pptd. by self limited

URT inf. (Streptococcal tonsillitis)

7. Rubroid P.

8. Arthropathic P./ Arthritis mutilans → T/T = MTx (Systemic corticosteroids in case of severe psoriatic arthritis)

② Auspitz sign

- clinical Δ of P.
- Removal of scale leads to pinpoint bleeding
- characteristically seen in plaque P.

② Impetigo herpetiformis

- some consider it as Generalised pustular P. of pregnancy.
- commonly seen in 3rd trim.
- Treated w/ topical & systemic corticosteroids.

② Imp. drugs which cause exacerbation of P. → Antimalarials, Li, Corticosteroid withdrawal, NSAIDs, β#.



LICHEN PLANUS

- Common Infl. d/s that affects skin, mucous memb., nails, hair.

- characterised by a lesion which has following features (SPs)
PRURITIC, POLYGONAL, PURPLE (violaceous), PLANE (flat topped),
PAPULE or PLAQUE.

- H/P suggests an immunological response to an antigen
+nt in the basal cell layer of epidermis → AUTOIMMUNE disorders

⇒ Causative factors - Drugs
Thiazides, Gold, Antimalarials,
Penicillamine, Phenothiazines,
Quinidine, ACE-i
Infections (HCV)
Immunological disorders (GVHD/s, Pr. Bil. cirrhosis)

⇒ H/P

(i) Damage to basal cell layer of epidermis → Hydropic degeneration of basal cells

(ii) Epidermal thickening esp. granular cell layer → Hypergranulosis

(iii) Hyperkeratosis

(iv) Acanthosis

(v) Subepidermal-lichenoid band (due to deposition of lymphocytes and histiocytes in upper dermis)

(vi) Pigment incontinence (Dropping of melanin from damaged keratinocytes of epidermis into dermis).

(vii) This melanin is engulfed by macrophages which results in formation of cytoid bodies (Giraffe or Colloid bodies).

(viii) Mox Joseph histological cleft (separation of epidermis in small clefts).

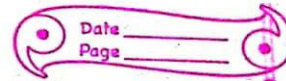
⇒ Clinical presentation

- 30-50 yrs ± slight ♀ predominance.

- Most cases are insidious.

- Lesions mainly on flexor surfaces of upper extr. (Wrist, Forearms, arms) and legs (shin).

- The surface has white streaks when examined under magnifying lens → WICKHAM'S STRIAE.



- Koebner's phenomenon.

- Mucous membranes are also involved esp of oral mucosa and less commonly of genital tract → lesions not as a white net like eruption (LACEY Pattern)

- The lesions heal spontaneously in 6 mo to 2 yrs. Lesions heal w/ hypopigmentation.

- Broca phenomenon (Despite severe pruritis, pts. don't scratch much; because scratching can lead to pain).

⇒ Associated C/F in LP

Ⓐ Nail changes -

(i) Pterygium - Proximal nail fold extends on to and fuses w/ nail bed. (characteristic lesion)

(ii) Onychomatrix - splitting of nail

(iii) Atrophy (thinning)

Ⓑ Scalp - cicatricial alopecia w/ perifollicular blue grey patches.

Ⓒ Very rarely chronic ulcerative lesions may develop malignant changes, i.e. Sq-cell. Ca.

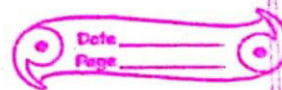
⇒ T/T of L.P.

- 1st line T/T = Topical Corticosteroids

- 2nd choice = Systemic CS for symptom control (possibly more rapid resolution)

- Oral antihistaminics for Pruritis

LESSIONS	T.O.C.
1. Localised LP	Topical CS + oral antihistaminic
2. Extensive LP	Oral CS / PUVA / Acitretin
3. LP of nail & scalp	Oral CS
4. Mucosal LP	Acitretin / Dapsone + oral CS



PITYRIASIS ROSEA

- Scaly disorder
- 10-35 yr.
- Round/oval pink/brown patches w/ a superficial, centrifugal scale, distributed over trunk in a Christmas tree pattern.
(Sometimes lesions predominantly on extremities and neck. (Inverse pattern))
- Viral d/s. (HHV6/HHV7)
- Self limiting (subsides in 6-12 weeks). Lesions subside w/ hyperpigmentation.
- Starts w/ UR prodrome or a mild flu.
- After 1-2 wks, Annular erythematous plaque appears on trunk, that is referred to as mother patch or herald patch.
- Over the next 1-2 wks, fresh patch appear all over in a Christmas tree configuration or fir tree configuration.
- The lesions are pinkish in white skin → "ROSEA"
- On the dark indian skin → lesions are skin coloured or brown.
- Most characteristic clue for Δ → the presence of a fine scale at the edge of the lesion referred to as centrifugal scale or collarette scales or cigarette paper scales.
- Drug induced P.R.
- ACE-i, Metronidazole, MTx, Isotretinoin, Gold, D-Penicillamine, P#
- Just discontinuation of the offending drug → Resolution of lesion



PEMPHIGUS

- an autoimmune blistering disorder, characterised by acantholysis (Loss of intracellular cohesion b/w keratinocytes), induced by deposition of IgG autoantibodies in the intercellular substance of epidermis [in Fish-net pattern].

- The split occur in epidermis (intraepidermal bullae).

- The characteristic c/f is Flaccid bullae on previously normal skin. [In contrast to bullous pemphigoid and eczema herpeticum, where the bullae occurs on underlying inflamed erythematous skin.]

④ main types -

- ① P. vulgaris
 - MC type
 - Antibodies form ag. Desmoglein 3 & 1
 - Found in epidermis of mucosa eg. oral
 - Found in skin epidermis
- ② P. foliaceus
 - Abs form ag. Desmoglein 1
- ③ P. vegetans
 - Least common type
- ④ P. erythematosus

split occur in suprabasal layer

split occur in subcorneal layer

⑤ Acantholytic cells (= Tzanck cells) are separated keratinocytes.

(found cells with hyperchromatic nuclei & perinuclear halo)

⑥ Carcinomatous P. (Paraneoplastic P.) is c/b NHL, CLL, Thymoma (EMG).

⑦ Drug induced P. is c/b Captopril, Bcin and Penicillamine. (CRP)



Dermatitis HERPETIFORMIS

- Vesiculo-bullous disorder.
- Almost all pt. have an associated gluten sensitive enteropathy.
- c/b deposition of IgA in papillary dermis and along the epidermal basement memb. zone (Dermoepidermal jn.).
- The deposits are Granular.
- IgA → triggers an immunological cascade → Neutrophil recruitment & Complement activation.
- split is subepidermal.
- DH
 - Celiac d/s (Gluten sensitive enteropathy) } w/ Ted extension of HLA — A1, B8, DR3 & DQ2
- Dominant autoantigen of DH = epidermal transglutaminase-3.
- DH is c/b dermal deposition of circulating immune complex containing both IgA and transglutaminase-3.
- Pr. lesion = Papule/papulo-vesticle.
- Pruritis is prominent
- Lesions are symmetrically distributed over extensor surfaces (elbow, knees, buttocks, back, scalp, posterior neck).
- Lesion is usually polymorphic and occurs in crops.
- Mucosa is never involved.
- T/T
 - Put the pt. on Gluten free diet
 - Dapsone = D.O.C.
 - (sulphapyridine (a sulfonamide can also be used))
 - Local application of antipruritic (1% phenol in calamine lotion is also used)
 - Vitamins & minerals are also given for supplementation.



EPIDERMOLYSIS BULLOSA

- a group of inherited bullous disorders chr. by blister formation in response to mechanical trauma. Family H may be +ve.

→ (N) basement memb is b/w epidermal basal layer and dermis.

→ The BM (basal lamina) is attached to basal cells hemidesmosomes & the help of Keratin containing intermediate filaments and is attached to dermis (dermal papillary layer) & the help of type VII collagen containing fibrils.

→ Any defect in this anchoring complex leads to separation of skin. (vulnerability to trauma and blisters)

(i) EB simplex - Mutation in gene coding for Keratin 5 & 14 [Separation → epidermal]

(ii) EB junctional - Mutation in Laminin α-3 (LAM α-3), LAM β-3, LAM γ-2 genes

is a part of BM

[Separation → at DEJ]

(iii) EB dystrophic - Mutation in Collagen VII-A1 gene. [Separation → in dermis]

Simple DJ
K C L

Simple DJ
(KCL)



ERYTHEMA MULTIFORME

- acute, often self limited eruption.

- Hallmark → Target lesion (Iris lesion). Bull's eye lesion

- classified into -

EM minor	SJS	TEN (= Lyell's synd.)
<ul style="list-style-type: none"> • ERYTHEMA MULTIFORME • Localized eruption of skin & or 2 out mucosal (limited to oral mucosa) involvement. [cutaneous d/s] • Self limiting (resolves in 10 days) 	<ul style="list-style-type: none"> • Mucosal erosions along • Pruritic macules. • Epidermal detachment affecting 10-30% of BSA. 	<ul style="list-style-type: none"> • SJS and TEN → single syndrome = EM Major or SJS-TEN synd. • Healing starts in a couple of days and is completed in 3 weeks

- causes

Idiopathic (MC)
 HSV (Most imp.), HBV, streptococci, TB, HIV, plasmid
 Sulphonamide, Phenytoin, NSAIDs.
 SLE, Pregnancy - Malignancy

- C/F

• Typical lesion = Target lesion (Iris lesion)

consists of 3 concentric components

central dusky purple
 central dusky erythema surrounded by vesicle bulla
 erythematous halo
 peripheral erythema
 Pale edematous ring
 Pale middle zone

• The distribution is on face and acral parts
 B/L symmetrical
 Palm, sole, Dorsum of hands/feet, distal part of forearms/legs

• Non pruritic & painless



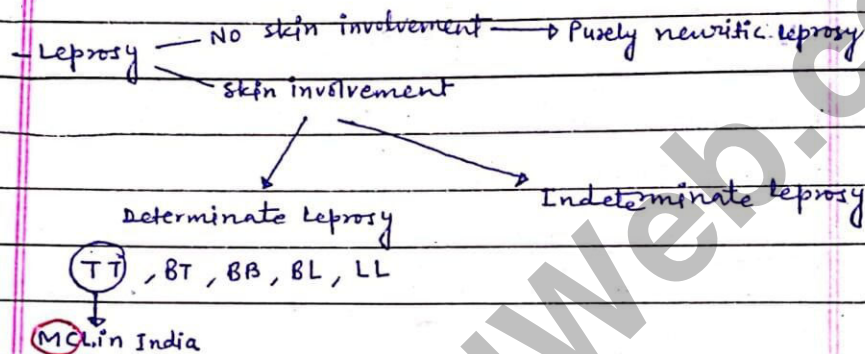
DARRIER'S DISEASE

- Keratosis Follicularis
- AD d/s
- begins in 1st and 2nd decade
- Waxy papules and plaques in sebaceous sites, i.e., face, forehead, scalp, chest and back.
- Histology shows suprabasal acantholysis in epidermis & dyskeratotic cells.



LEPROSY

- Iris pearls (or Military Leprosia) → Pathognomonic
- Largely confined to Skin, PNS, URT, Eyes, Testes (because M. leprae grows better in cooler places)
- ♀ genital tract rarely involved in leprosy, when involved OVARY is the MC.
- Virchow's Lepra cells or Foamy cells are large undifferentiated histiocytes.
Abundant in LL



Minimally infectious	TT (Polar Tuberculoid)	<ul style="list-style-type: none"> • Increase in bacterial load • Increase in No. of lesions
	BT (Borderline T.)	<ul style="list-style-type: none"> • Decrease in cellular immunity
	BB (Mid borderline)	<ul style="list-style-type: none"> • Lesions become Asymmetric to symmetric
	BL (Borderline Lepromatous)	<ul style="list-style-type: none"> • Definitive sensory loss to Glove stocking anaesthesia
Maximally infectious	LL (Polar L.)	<ul style="list-style-type: none"> • Lesions become well defined to Poorly defined

- TT → Granuloma
- BT → Satellite lesion
- BB, BL → Punched out or inverted saucer shaped
- LL → Globi, Free subepidermal Grenz zone



① Nerve commonly involved - High Ulnar, Low Median

- N. involvement in Leprosy is 100% (However 100% pts. do not develop symptoms of neural involvement)

② Sensation lost → Temp. > Light touch > Pain

- Proprioception and vibration is not affected as Leprosy spares post. column.

③ Lucio's phenomenon

- seen in LL.

- occurs in pts. who are untreated.

- Pts. develop recurrent crops of large sharply margined, ulcerative lesions, particularly on lower extremities.

④ T/T

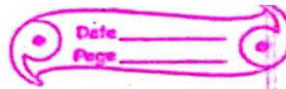
Single skin lesion	Paucibacillary (Ind, T1, B1)	Multibacillary (BB, BL, LL)
R.cin 600	R.cin 600 monthly	R.cin 600 monthly
Ofloxacin 400	Dapsone 100 daily	Dapsone 100 daily
Minocycline 100		Clofazimine 50 daily
ROM - single dose		
* Under LEP, Mx is same as Paucibacill.		

1st line antileprostatic drugs
(only R. cin = Bactericidal)

⑤ Post T/T surveillance

Paucibacillary → 2 yrs (once a yr)

Multibacillary → 5 yrs (once a yr)



LEPRO REACTION

- Sudden exacerbation in clinical activity of the d/s process in leprosy.
- represent HSR that occur in the d/s course due to abrupt change in immunological response of the body. eg. M. leprae.
- Rkns. may develop any time (before/during/after completion of Treatment)

② types

Type I	Type II (ENL)
- occurs during first 6 mo.	- occurs in lepromatous end (BL, LL)
- occurs in BT, BB, BL (Borderline)	- TNF- α plays central role
- Type IV HS	- a Jarisch Herxheimer skn. (Arthus skn. / type III HS)
- classical signs of inflamm. (Redness, Pain & Tenderness) \bar{c} in previously involved macule, papules or plaque.	- follows institution of antileprotic T/F
- systemic features do Not occur	- Presenting symptoms
- Neuritis is common (MC = ULNAR N.)	Crops of fresh pinkish erythematous papules or nodules (MC)
- ② types	systemic features: Fever, Arthritis, Hepatitis, Lymphadenitis, Glomerulonephritis
Downgrade	Reversal skn.
- occurs before initiation of anti-leprotic T/F.	- occur after initiation of anti-leprotic T/F.
- Histologically becomes more lepromatous.	- Histologically becomes more tuberculoid.
- T/T — Glucocorticoids along \bar{c} continuation of antileprotic drugs	- T/T
	• D.O.C. = Glucocorticoids
	• For mild cases — NSAIDs (Aspirin)
	• If 2 courses of Glucocorticoids fail, Thalidomide can be used.
	↓ Dose = 200-300mg
	• other drugs = Clofazimine, Chloroquine
	Thalidomide has NO role.

CUTANEOUS TB



— Based on the manner in which the skin is invaded by the tubercle bacilli, cut. TB is divided into —

Exogenous source	Endogenous	Hypersensitivity
<ul style="list-style-type: none"> • <u>TB chancre</u> (Pr. inoculation TB) • <u>TB verrucosa cutis</u> (Warty TB) — in pt. w/ good CMI to M.TB. — single indolent verrucous (warty) nodule is characteristic. 	<ul style="list-style-type: none"> • <u>Lupus Vulgaris</u> — MC cut. TB — Nonscaly annular erythematous lesion — Lesions are solitary — well defined Plaque. — crusting & induration at periphery — central scarring — on face, buttock — slowly ↑ in size. — <u>Blanching w/ glass slide</u> (diascopy) — reveals grey green foci — Apple jelly nodules — Reappearance of new nodules in prev. atrophic or scarred lesions is characteristic. — Δ is confirmed by Bx. • <u>Scrofuloderma</u> — 2nd MC cut. TB — MC cut. TB in children — due to direct extension of infection from an underlying TB nodule either in a LN, bone or a jt. 	<ul style="list-style-type: none"> • Tuberculids 1. <u>Lichen scrofulariformis</u> — involvement of sweat glands, and hair follicles w/ Noncaseating epithelioid granuloma. 2. Papulo-necrotic tuberculids → MC 3. <u>Erythema induratum</u> — Bazin's d/s or Nodular tuberculid 4. <u>Erythema nodosum</u> — example of facultative tuberculids — are lesions in which M.TB is one of the several etiological agents.



DERMATOPHYTES

- infect str. Corneum.

- cause a variety of clinical conditions, collectively k/a Dermatophytoses/
Tinea/Ringworm

- 3 genera

1. Trichophyton

→ All 3 ^{skin} Hair Nail

2. Microsporum

→ M does not affect N ^{skin} Hair

3. Epidermophyton

^{skin} Nails

Types of ring worm

Causative agents

1. Tinea capitis	Scalp & hair	Ectothrix → M. canis (MC) Endothrix → T. tonsurans (MC common cause of T.C. hus) ↳ T. violaceum (MC cause of T.C. in India) T. schoenleinii → Favus (Scutula / ^{fruiting} crusty form. over scalp) ⊕ inflammatory T. capitis → c/b M. canis ↳ Boggy swelling pustulation (KERIO)
2. Tinea imbricata (Hanuman RW)		T. rubrum, T. montagnophytes
3. Tinea barbae	Beard	T. mont.
4. Tinea corporis	Nonhairy skin of trunk & limbs	T. rubrum, T. montagnophytes, E. floccosum
5. Tinea unguium (Onychomycosis)	Nail	
6. Tinea manuum	Palms	T. rubrum
7. Tinea cruris (Jock Itch)	Groin, perineum, Thigh, scrotum	T. rubrum, T. mont., E. fl.
8. Tinea pedis (Athlete's foot)	4th web space	

⊙ Tinea incognita - due to inappropriate use of topical corticosteroids.
 Extensive RW & atypical appearance suppress the protective intl. response of the skin to the RW fungus



T/T of Dermatophytosis

1. Ordinary RW of hairy skin → Topical
 - Azoles (mic, eco, clotri)
 - Terbinafine
 - Cyclopirox oleamine

- Griseofulvin is not used topically.

2. When multiple areas are affected (T. unguium / T. capitis)

(or)

Failure of topical therapy



Systemic Terbinafine = DOC

systemic Griseofulvin (2nd choice)

Other → Systemic Ketoconazole / Itraconazole

③ Duration of systemic therapy

- skin → 2 wks
- Palm & soles → 4-6 wks
- Fingers nails → 4-6 months
- Toe → 8-12 months



SCALES

- intensely pruritic
- c/by Sarcoptes scabiei
 ↓
 an acarus (mite)
- usually affects children but can occur at any age.
- water washed d/s (occurs due to inadequate use of water or improper hygiene).
- After copulation, ♂ mite dies and then ♀ mite burrows into str. corneum at the rate of 2mm/d. ♀ mite lays eggs which hatch into larva, which moults and mature into adult mite.
- Burrow
 - visible clinically as an irregular grey brown line.
 - serpentine / snaked
 - Pathognomonic sign
 - difficult to demonstrate in infants
- I.P. = 4-6 weeks before itching begins.
 ↓
 due to the development of HS to some of the products (saliva-skin)
 ↓
 (4-6 wk are required)

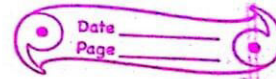
C/F 1. Severe itching

- Most notably along the web spaces of fingers, wrists, elbows, axillae and groin area → also k/a Circle of Hebra.
 - Worse at night
 - Generalised
 - affecting several family members.
2. Papules and papulovesicle due to HS to mite. Pustules can occur due to secondary infection.
- Excoriation and scratch marks

② Scalp, face, Palm, toes

Characteristically spared

Typically involved in young children & infant.



⊙ Atypical types

1. Norwegian scabies

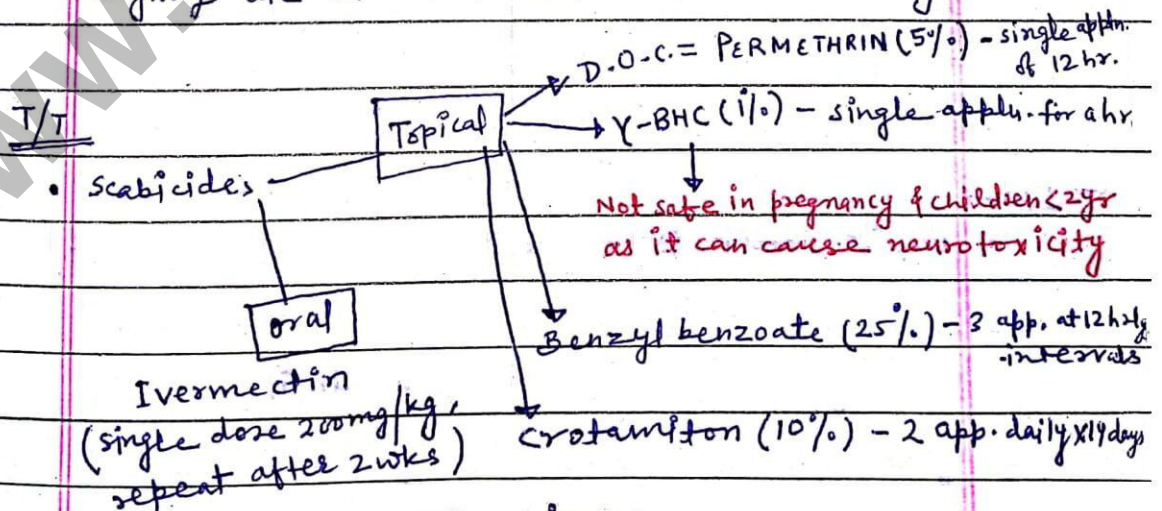
- Most severe form of scabies
- Itching not prominent
- crusted hyperkeratotic lesion on face, palms & sole, nails.

2. Nodular scabies

- Itchy nodule over scrotum and penis.

Principles of Mx

- After taking the proper bath, scabicides should be applied effectively to the whole body (BELOW THE JAW LINE IN ADULTS) including genitals, soles and skin under the free edge of nails.
- Reapply scabicides after 7-10 days. (since most scabicides are not ovicidal)
- Treat all member of the family whether symptomatic or not.
- All clothing need not to be treated, because the mites anyway die in unworn clothes in about 7 days.



- For itching → Antihistamines
- In sec. infections → i.v. antibiotics

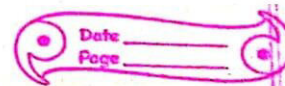


SYPHILIS

<u>PR. SYPHILIS</u>	<u>2° SYPHILIS</u>
<u>Lesion</u> → CHANCERE (Hard chancre) - occurs at the site of entry & spirochete - well defined punched out ulcer - single - Painless - Avascular (Non-bleeding) - Firm Induration.	• MC manifestation = Rash ↓ - B/L symmetrical macule or maculopapules on palms & soles. - Non pruritic and asymptomatic. • Generalized nontender LN pathy. • Condylomata lata - Most infectious lesion of syphilis ↳ Abundant spirochete in lesion • Superficial mucosal erosions in lips, oral mucosa, tongue, glans penis, inner prepuce, vagina.
<u>Lymphadenopathy</u> → Painless, firm & Non suppurative	• Constitutional symptoms - Sore throat, wt. loss, Malaise, anorexia, headache. • Mouth eaten alopecia (Patchy alopecia) • Hutchinson's sign.
<u>Sites</u> → • Penis in heterosexual • Rectum, Anal canal, mouth in homosexual • Cervix & Labia in ♀	• Deep dermal tenderness on pressure. • Complications - Proteinuria, Acute nephritis, Arthritis, Hepatosplenomegaly.

© T/T

1. Pr., Sec., Latent, Tert. (except Neurosyphilis) → D.O.C. = Benzathine
2. Neurosyphilis → Aqueous Penicillin G
3. Pregnancy → D.O.C. = Penicillin



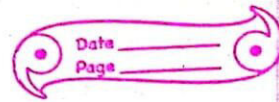
LATENT SYPHILIS	3° SYPHILIS
- Asymptomatic stage	- May +ve as
- occurs b/w 2° & 3° stages.	• Cardiovascular syphilis
- 1st stage - ① <u>Early Latent or relapsing syphilis</u>	• Neurosyphilis
- occurs during 1st 2yr	<div style="display: flex; justify-content: space-around;"> <div style="text-align: center;"> Asymptomatic ↓ Takes dorsalis General paresis Meningeal involvement </div> <div style="text-align: center;"> Symptomatic ↓ Takes dorsalis General paresis Meningeal involvement </div> </div>
- chancre may recur at its original place → <u>Chancre Redux</u>	
↓ It is due to relapse of original infection (not due to reinfection).	
	• Late benign syphilis (=Gumma)
② <u>Late Latent or Non-relapsing syphilis</u>	↓ Noninfectious
- occurs after 2yr	↓ Spirochetes are difficult to demonstrate in lesion
- No relapse	

Penicillin



Allergy

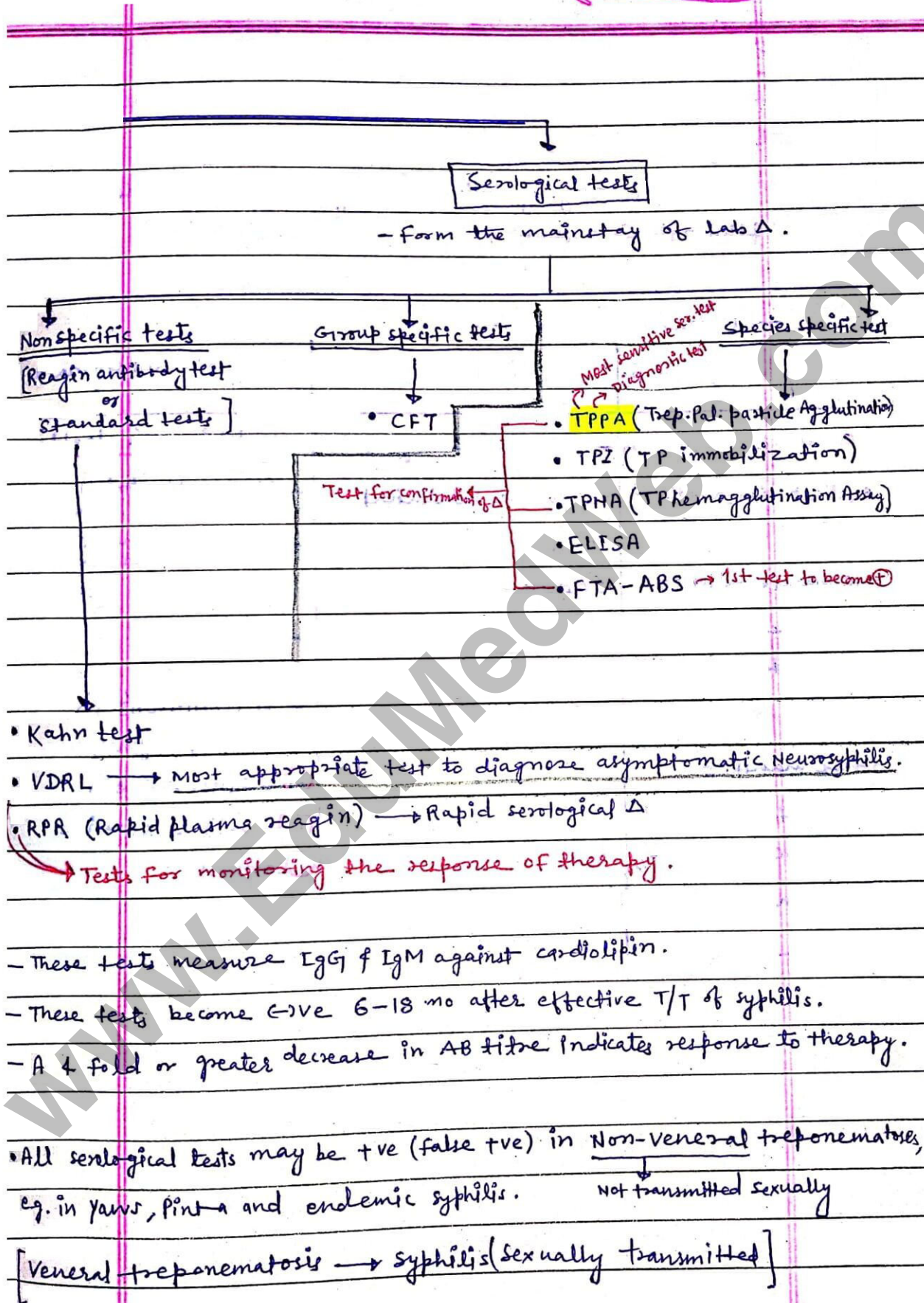
→ D.O.C. = Tetracycline or Doxycycline.



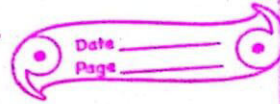
LAB Δ of syphilis

Demonstration of spirochetes under microscopy

- applicable in Pr. & sec. stages as spirochetes are abundant during Pr. & sec. stages.
- Dark field microscopy & Immunofluorescence microscopy



best diagnosed by $\begin{cases} \text{IgM FTA-ABS} \\ \text{syphilis capita M test} \end{cases}$

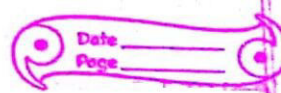


Congenital syphilis

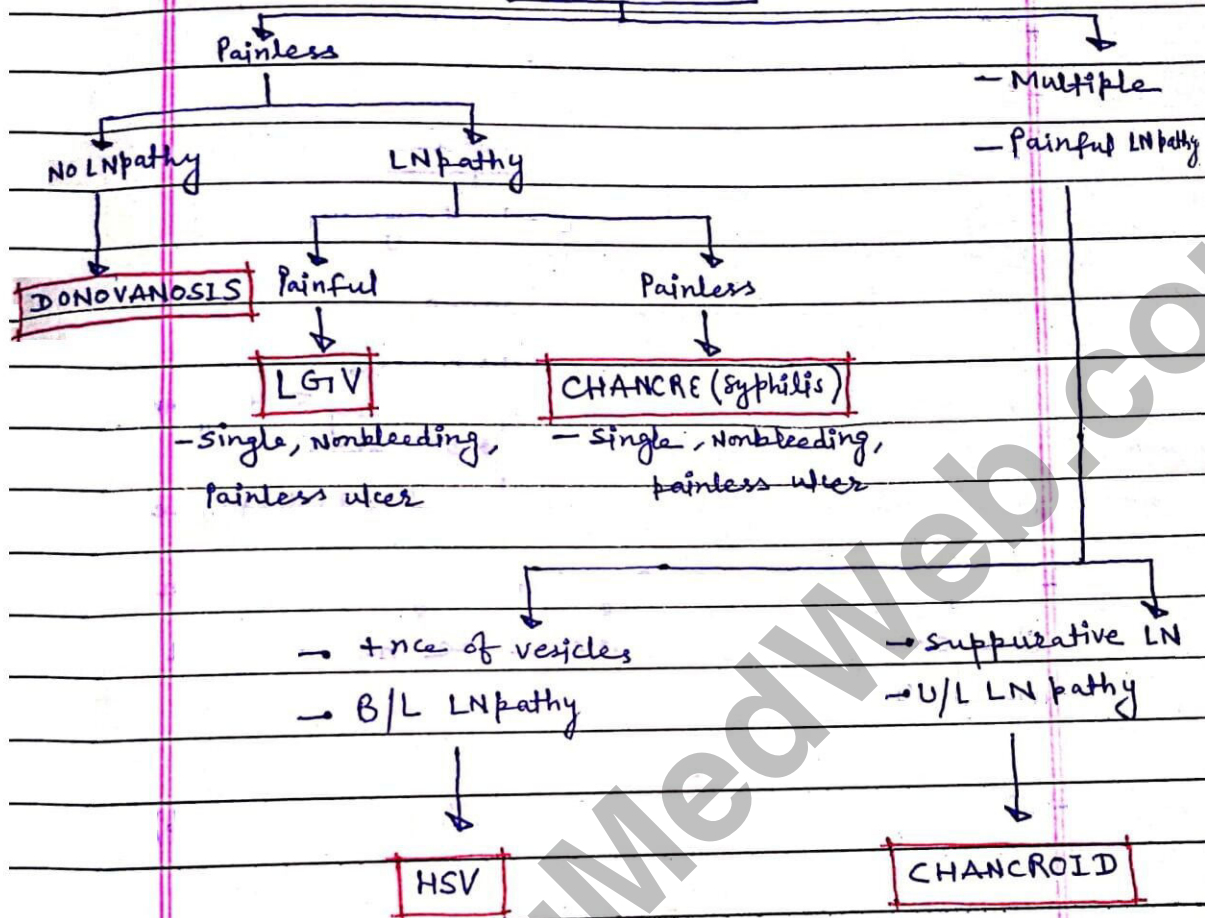
— Transmission of *T. pallidum* across the placenta may occur at any stage of pregnancy but the lesions of cong. syphilis generally have their onset after 4th month of gestation.

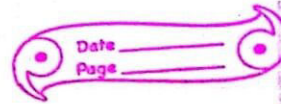
Clinical manifestation-

Early congenital S.	Late cong. S.	Residual stigmata
— occur \geq in 2yrs of birth	— occur after 2yrs of birth	— <u>Hutchinson's teeth</u>
— Earliest sign = Rhinitis or snuffles	— 60% are subclinical	— Shaker shins (Ant. tibial bowing)
— MC manifestations =	— Clinical manif. =	— Rhegades (linear scars at the angle of mouth and nose)
— Bone changes like	<u>Interstitial keratitis</u>	— <u>Deafness</u>
— osteochondritis, periostitis,	Clutton's jt (B/L knee effusion),	★ <u>HUTCHINSON'S TRIAD</u>
— osteitis.	8th N. deafness	of Cong. syphilis
— other manif. =		① Hutchinson teeth
Mucocut. lesions (Vesicles/bullae, petechiae, condyloma lata),		② Interstitial keratitis
Hepatosplenomegaly,		③ Deafness
Jaundice		<u>HIND</u>



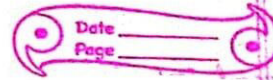
Genital Ulcers





DONOVANOSIS

- Granuloma inguinale / Granuloma venereum
- c/by *Calymmatobacterium granulomatis*.
- I.P. = 1-4 wks
- begins as one or more sc nodules
 - ↓
 - erode through skin
 - ↓
 - ULCER
 - Painless
 - Bleeding & red granulation tissue
 - Indurated
 - Red and velvety (Beefy red)
- S.C. granulomas of inguinal region in Donovanosis look like enlarged LN, but these are not enlarged LNs. ∴ these are not PSEUDOBUBOS.
- Site of lesions = Genitalia (90%), Inguinal (10%) & anal regions.
- C/C → Pseudoelephantiasis, Phimosis, Paraphimosis.
- Δ → Demonstration of intracellular Donovan bodies in large mononuclear cells visualized in smear prepared from lesions or biopsy specimens.
 - Bacteria has safety pin appearance.
- T/T
 - D.O.C. = Azithromycin
 - 2nd choice = Doxycycline & chloramphenicol
 - Streptomycin → NOT USED NOW

LGV

- c/by chlamydia trachomatis (serotypes - L₁, L₂, L₃)

- clinical course

3 stages

1st stage (Pr. LGV)

2nd LGV

3rd LGV
(Genitoanal syndrome)
- chr. by Proctocolitis

- self limited, single, - Painful inguinal LNopathy

asymptomatic, painless, - Swollen LN coalesce to form bubos, i.e. Matted LN
Nonbleeding genital ulcer

may rupture to form discharging sinus

- Groove's sign - Enlarged LN both above & below inguinal ligament

- C/C

1. Esthiomene - Enlargement, thickening and fibrosis of labia.

2. Elephantiasis of the genitals

3. Rectal stricture

4. Systemic → Arthritis, Pneumonitis, Perihepatitis

• Frei's test - was used for Δ (not used now)

• T/T → D.O.C. = Doxycycline / Tetracycline

Gonococcal Infection

In ♂	In ♀
<p>MC = <u>Acute Urethritis</u></p> <p>↓</p> <p>Purulent discharge per urethra</p>	<div> <div> <p><u>Gonococcal cervicitis</u></p> <ul style="list-style-type: none"> - N. gonorrhoeae primarily infects cervical os but can also infect more peripheral area due to presence of columnar cells. - c/c of cervicitis - Urethritis, Acute salpingitis etc. </div> <div> <p><u>Gonococcal vaginitis</u></p> <ul style="list-style-type: none"> - The vaginal mucosa of healthy women is lined by str. sq. epithelium hence usually not infected by N. gon. - In Anaestrogenic women (e.g. prepubertal girls and postmenopausal women) in whom the vaginal str. sq. epith. layers are often thinned down to basal layer, which can be infected by N. gon. </div> </div>

I/I

D.O.C. = Ceftriaxone 250

⊙ Gonococci affects epididymis but does not affect testis.

⊙ Syphilis involves testis but not epididymis.

⊙ NGU (or Non specific U)

↓
MC cause = Chlamydia trachomatis

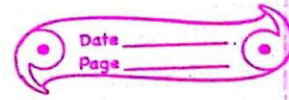
Other causes = Ureoplasma ureolyticum, Mycoplasma hominis, Trichomonas vaginalis, HSV.



HSV

- 2 types →

HSV-1	HSV-2
- causes lesions above waist.	- causes lesions below the waist.
	- <u>Herpes Genitalis</u>
	• STD
	• Often asymptomatic
	• When symptomatic it ppt as multiple vesicles, which are very painful.
	• B/L Painful inguinal LNopathy
	• characteristic feature = Frequent recurrences
	• T/T - Acyclovir (DOC)
	Valacyclovir
	Famciclovir
	In case of Acyclovir resistant mucocutaneous HSV infection - <u>Foscarnet</u> .



MOLLUSCUM CONTAGIOSUM

- A common viral infection in children.
- C/by Pox virus
- chr. by Multiple pearly white, dome shaped papules which are umbilicated centrally.
- on using a hand lens, many of the papules has a mosaic appearance.
- Epidermal cells contain eosinophilic intracytoplasmic inclusion bodies (Molluscum or Henderson-Paterson bodies).
- Autoinoculation can give rise to lesions arranged linearly along line of trauma. → Pseudoisomorphic phenomenon.
- May involve any part of body (In children, MC = Face).
- Anogenital mol. cont. is a STD.
- T/T
 - Curettage, Electrocautery, Cryotherapy.
 - Imiquimod (Immunostimulant).

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DLE	SLE
well defined coin shaped (discoid), annular, erythematous plaque, \pm adherent scales.	Butterfly rash Papulosquamous lesions on exposed parts
- central depigmentation, and	- DLE like discoid lesions
- peripheral hyperpigmentation & erythema.	- Photosensitivity, oral ulcers (painless)
- Central depigmented area is atrophic, scarred and telangiectatic.	Periungual telangiectasia, alopecia
- distributed predominantly on sun exposed areas of scalp and on external ear.	- systemic manifestations - Arthritis, serositis (Pleuritis, Pericarditis), Renal d/s, Neurological (seizure, Psychosis), constitutional symptoms like fever.
Mucosal involvement occurs in 25% of pt. \rightarrow Lips and buccal mucus.	- Drugs causing SLE - Procainamide, Hydralazine, INH, Methyldopa, Penicillamine, Chlorpromazine, Li, CBZ, Phenytoin.



CUTANEOUS REACTIONS TO DRUGS

- Most frequently observed adverse reactions to drugs.

FIXED DRUG ERUPTION

- Development of one or more annular or oval erythematous patches as a result of systemic exposure to a drug.
- Lesions develop soon after ingestion: occurs from 30 min to 8 hr after ingestion of drug, in a previously sensitized person.
- Lesions begin as annular or erythematous patch which sometimes develop central vesicle/bullae.
- Mostly lesions are solitary but they may be multiple.
- Mucocutaneous junctions (lip & glands) most frequently involved.
- Lesions heal w/ hyperpigmentation.
- Lesions are recurrent, occur everytime τ drug intake.
- Drugs implicated in FDE \rightarrow Salicylates and other NSAIDs (PCM, Ibuprofen, phenacetin, phenylbutazone), antibiotic (Sulfonamide, Dapsone, Tetracyclines), Anticonvulsants (Barbiturates).

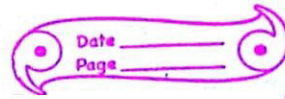
LICHENOID DRUG ERUPTION

- closely resembles lichen planus, but differs from LP in that mucosal lesion and nail involvement is rare.
- Drugs causing LDE \rightarrow Antimalarials (chloroquine), NSAIDs, Gold, Dapsone, Ketoconazole.

Date: _____
Page: _____

- characterized by

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ACTINIC KERATOSIS

- Solar Keratosis
- Paraneoplastic (Persistence of the nuclei of the keratinocytes into the str. corneum)
- UV-light induced lesion of the skin that may progress to invasive sq.c.c.
- MC lesion & malignant pot. arise on the skin.
- seen almost exclusively in whites.
- elderly →
- raised, pink or grey, scaly or warty hyperkeratotic plaque or papule.
- T/T
- ✓ Medical - Topical 5-FU (MC)
- ✓ Surgical destruction of the lesion - Cryosurgery (MC).