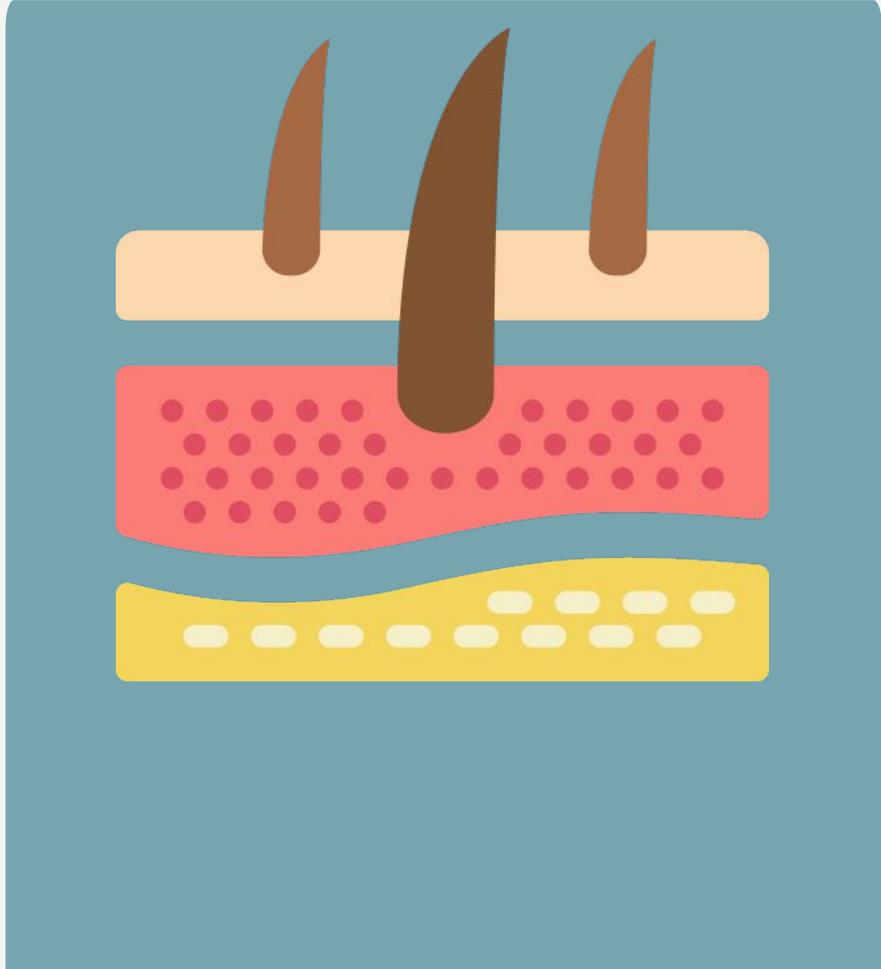

3-Psoriasis & Other Papulosquamous disorders

Objectives:

- To know the definition of papulosquamous pattern.
- To know the group of diseases known as papulosquamous diseases.
- Psoriasis pathogenesis, clinical presentation, and management.
- Lichen planus pathogenesis, clinical presentation, and management.
- Pityriasis rosea pathogenesis, clinical presentation, and management

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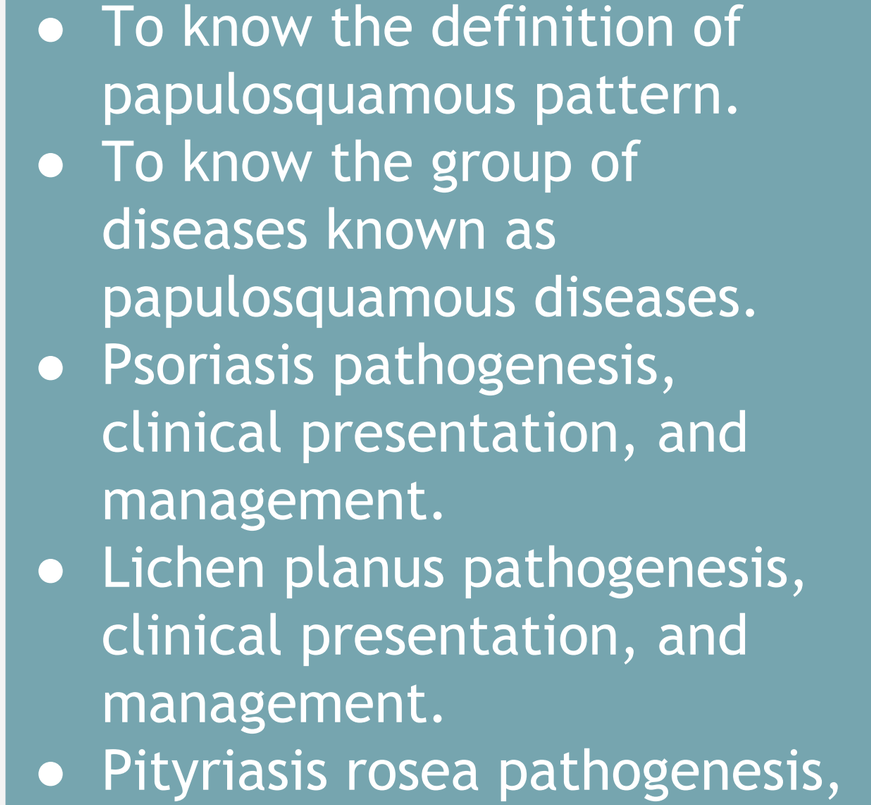
Revised by: Saif Al Meshari, Rotana Khateeb

References: Doctor slides, Team 436

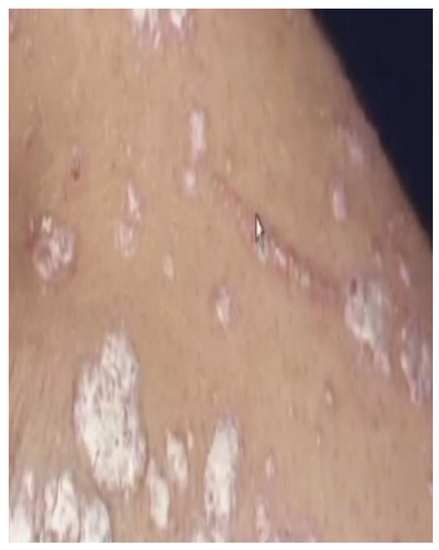
Color Index:

- Important
- Doctor's Notes
- Extra
- Extra

Editing File



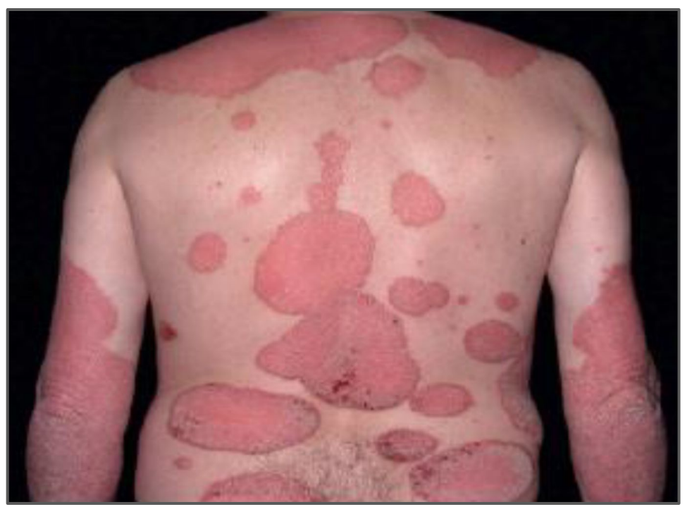
Papulosquamous diseases:



- The term squamous refers to scaling that represents thick Stratum Corneum and thus implies an abnormal keratinization process.
  - Keratinization is the differentiation of basal keratinocytes.
  - The basal keratinocytes as they move upward, they accumulate keratin inside them & their organelles totally die.

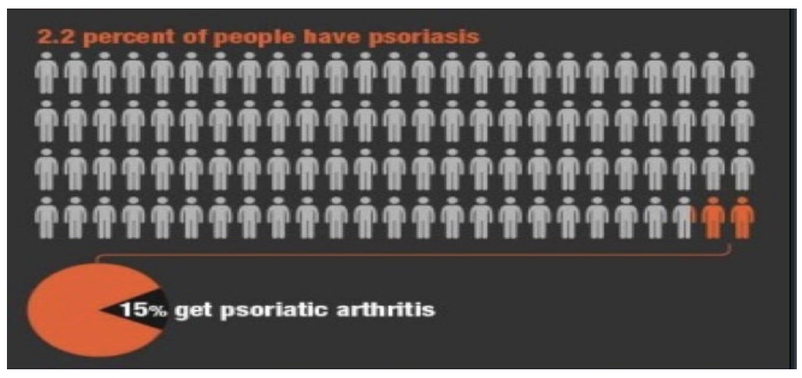
- Papulosquamous diseases are typically characterized by scaly papules (papule = elevated lesion).
- It could be papule or plaque, but the papulosquamous is the reaction pattern of the disease which means a reaction (inflammation) inside the skin (within the epidermis & dermis) to a specific thing
- that presents itself on the surface of the skin with a certain morphology.
- Other disease patterns include psoriasiform, lichenoid, bullous, pustular as well as papulosquamous pattern & each has many differential diagnoses.

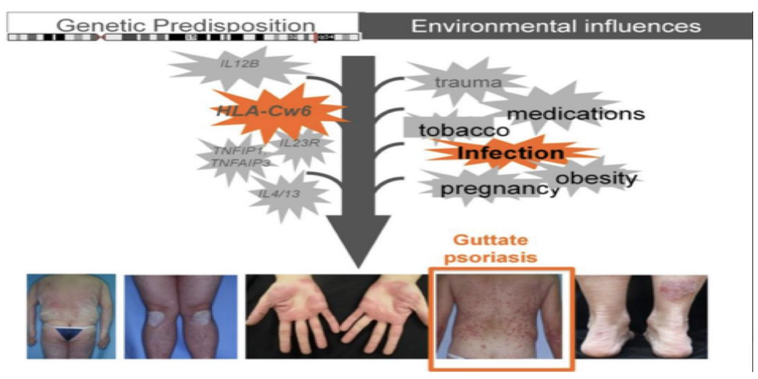
Psoriasis (الصدفية):

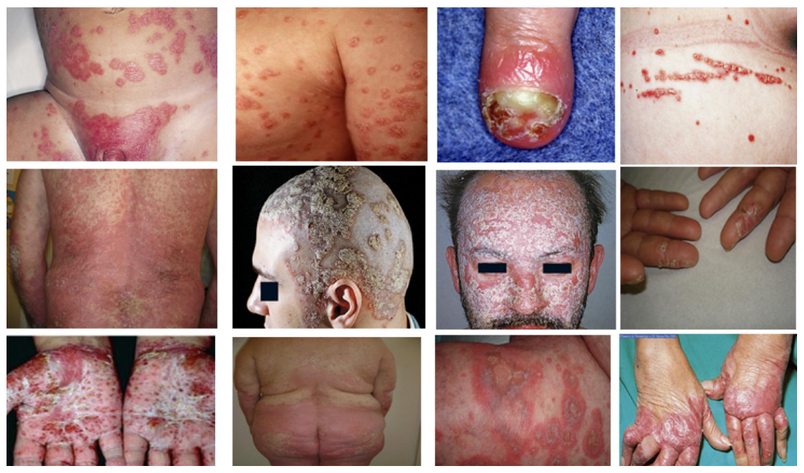


-Chronic plaque type
-Well demarcated erythematous scaly plaques

- Chronic non-contagious polygenic multisystem inflammatory disease.
- Psoriasis can be triggered by infections, trauma, stress and medications.
- The characteristic lesion (classical psoriatic lesion) is a sharply demarcated erythematous scaly plaque that may be localized or generalized.
- The natural history follows a chronic course with intermittent remissions.
- Has two peaks in age of onset (bi-model age presentation): one at 20-30 years and a second at 40-50.
- Genetic factors play a role in psoriasis proved by:
  - -Positive family history by 36-91%.
    - One affected parent: 16%
    - Both parents: 50%
    - Non-psoriatic parents with sibling: 10%
    - Monozygotic twins: 70%
    - Dizygotic twins: 20%
  - -There are multiple susceptibility genes called (PSORS 1-9).
  - -HLA studies showed high risk of psoriasis with HLA-Cw6 (also called PSORS 1 & is present on chromosome 6).
  - -HLA type influences the type of psoriasis and the course of the disease.
- Also involve extracutaneous manifestation







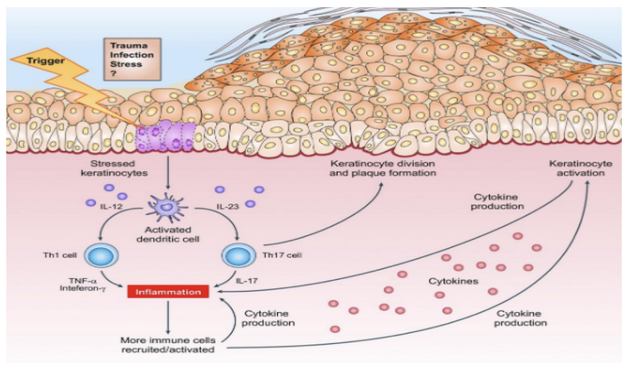
TYPE	HLA
Pustular psoriasis	B27
Guttate psoriasis	B13, B17
erythrodermic	B13, B17

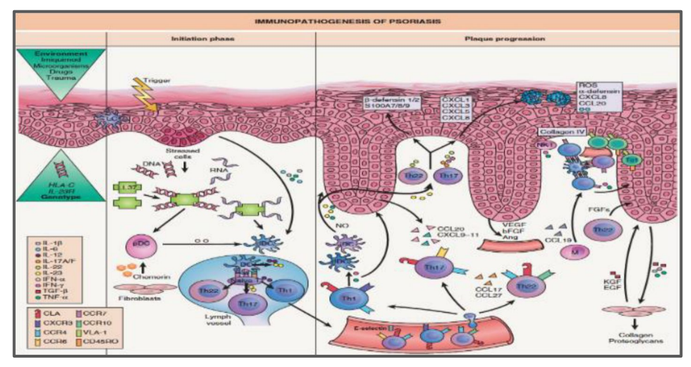
COURSE	HLA
Early onset (positive family history).	Cw6 (type 1 psoriasis) 90% of psoriasis patients have Earlier than 20-30
Late onset (negative family history, negative Cw6).	(type 2 psoriasis)

❖ Triggering factors:

- Trauma (25% of patients) sunburn, viral exanthem, any kind of trauma, physical trauma.
  - If psoriasis is induced by trauma, it is called koebnerization (also called isomorphic phenomenon).
  - Ddx of koebnerization: psoriasis, lichen planus or warts.
  - Trauma-induced psoriasis occurs in patients already having psoriasis.
- Infections (streptococcal pharyngitis). Guttate psoriasis
- Drugs: lithium, B-blockers, antimalarials, IF (interferon), systemic steroids may trigger pustular psoriasis
  - Antimalarials can either trigger or exacerbate psoriasis. Trigger psoriasis means that after taking antimalarials, psoriasis develops; however, exacerbation of psoriasis means it makes the condition worse in patients already having psoriasis.
- Irritating topicals like tar and dithranol. Exacerbation
- Hypocalcemia.
- Pregnancy.
- Obesity.

Psoriasis patients have lots of co-morbiditis





❖ Pathogenesis of psoriasis:

-In psoriasis Th1 is dominant with some cytotoxic cells IL-2 & 12

The trigger acts on the basal keratinocyte ---> keratinocytes become stressed & start to present some of its antigen to dendritic cells ---> the dendritic cells get activated IL-23 & IL-12 under the effect of dendritic cell stimulate Th17 cell & Th1 cell respectively ---> both of them induce inflammation & recruit more cells from the endothelium to keep the inflammation going on + plaque formation.

-Th17 cells will also cause excessive proliferation of the basal keratinocytes. The elevated lesion results from the cells building up. All of the basal keratinocytes underneath the lesion are contributing to the proliferation. This is what we call growth fraction.

❖ Epidermal cell kinetics:

- The growth fraction of basal cells is increased to almost 100% (in psoriatic patients) compared with 30% in normal skin.
  - Growth fraction is a ratio of dividing cells over the population cells.
  - 100% growth fraction means all the population cells are dividing.
- The epidermal turnover time is shortened to less than 10 days compared with 30 to 60 days in normal skin.

❖ Tools to measure severity index:

You are not supposed to memorize, know it

- Psoriasis is a disease of 10
- Psoriasis Area and Severity Index (PASI).
  - It is an equation that measures the erythema, scaling & induration of the lesion.
  - We sum the 3 measures & multiply them to the surface area involved.
  - It ranges from 0-72; anything above 10 is a severe psoriasis.
- Physician global assessment (PGA).

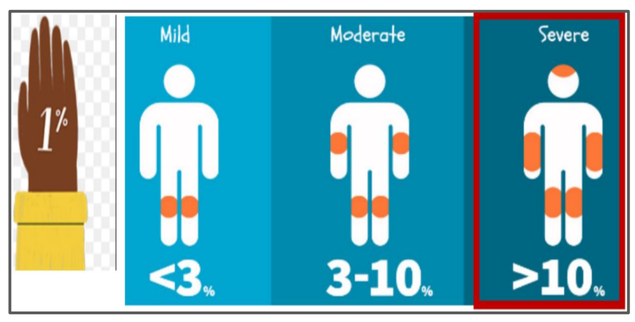
Psoriasis		
0	Clear	No signs of psoriasis, but postinflammatory discoloration may be present
1	Almost clear	Only minimal plaque elevation, scaling, and erythema
2	Mild	Slight plaque elevation, scaling, and erythema
3	Moderate	Moderate plaque elevation, scaling, and erythema
4	Severe	Very marked plaque elevation, scaling, and erythema

- Dermatology life quality index (DLQI).
  - 10 questions, covering the following topics: symptoms, embarrassment, shopping and home care, clothes, social and leisure, sport, work or study, close relationships, sex, treatment.
  - Each question refers to the impact of the skin disease on the patient's life over the previous week.
  - >10 is considered severe psoriasis.
- Patient global assessment.
  - How the patient is assessing the severity of psoriasis.

For example, the patient may have psoriasis limited to his hands, but is very distressing him.

- Body surface area (BSA) affected: role of hand.
  - Hand = Equal 1% BSA (the patient's hand).

Very important to ask the patient if it affect his\her quality of life



Types of psoriasis :

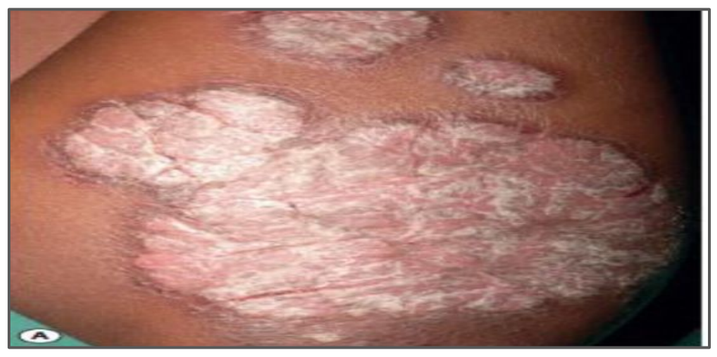
❖ **There are five types of psoriasis:**
- Plaque (most common).
- Guttate.
- Pustular.
- Erythrodermic.
- Psoriatic arthritis.

❖ **Special locations:**
- Nail psoriasis.
- Palmoplantar pustulosis.
- Flexural psoriasis.

-Flexures = الثنيات .

-Flexural areas in the body: axilla, under the breasts & the groin.

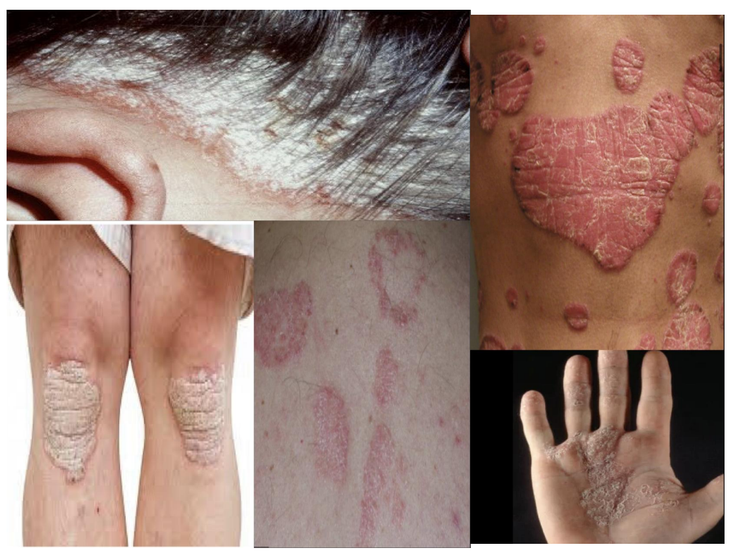
- Oral psoriasis.
- Napkin psoriasis.
- At any point of time different variants may coexist.



-If scratched causes candle sign
-if pealed causes auspitz sign

❖ Chronic plaque psoriasis:

- Characterized by sharply demarcated scaly erythematous plaques.
- Relatively symmetric distribution.
- Scalp, elbows, knees, hands and presacral are sites of predilection.
- When healing occurs, it starts in the center producing annular lesions.
- The most common type of psoriasis ranges from mild to severe.
- 65% complain of itching.
- The disease may worsen in winter; improve in summer.
- The major symptom is disfigurement that affects the quality of life.
- Signs of exacerbation and unstable disease include pinpoint papules surrounding existing plaques or expansion of the lesion with more intense erythema and tenderness.
- Koebner phenomenon is positive.

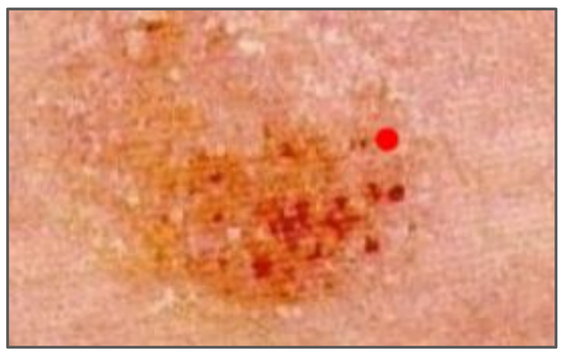


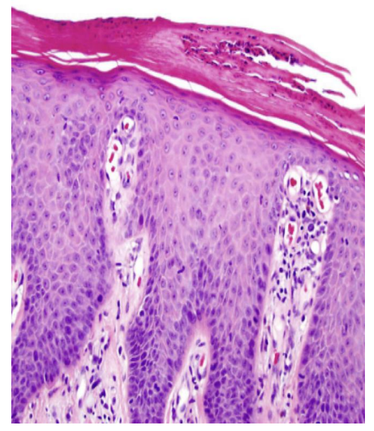
◆ Auspitz sign is positive:

-The appearance of small bleeding points (pin-point bleeding) after removal of scale from the surface of psoriatic papules or plaques.

-This sign is characteristic but NOT pathognomonic.

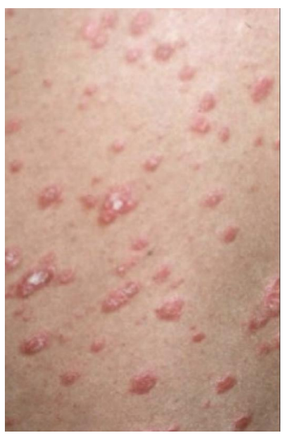
-It is a reflection of dilated vessels of the papillary dermis and thinning of the suprapapillary epidermis.

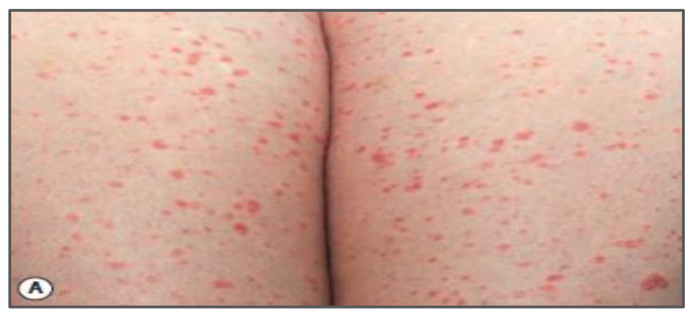




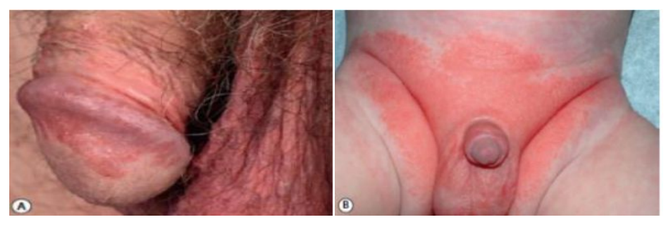
❖ Types of chronic plaque psoriasis:

❑ **<u>Guttate psoriasis</u>** (guttate = drop)
- Small papules over the trunk and extremities.
- Seen commonly in children and adolescent preceded by URTI.
- In over half of patients an elevated antistreptolysin O titer indicating recent streptococcal infection.





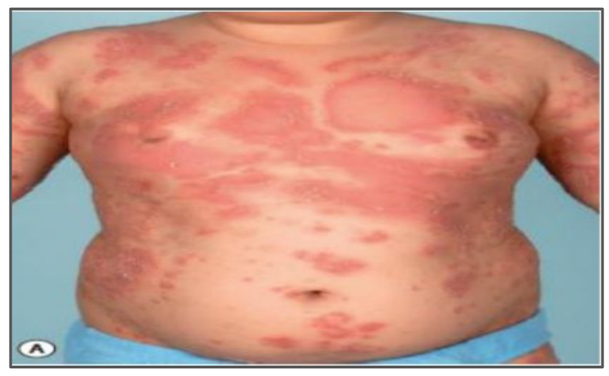
❑ Flexural psoriasis:
affects Axillae, groin and genital areas and presents as well demarcated erythematous plaque without scales.



Flexural psoriasis. notice there isn't any scaling

❑ Sebopsoraisis:
indeterminate stage (too advanced for us)

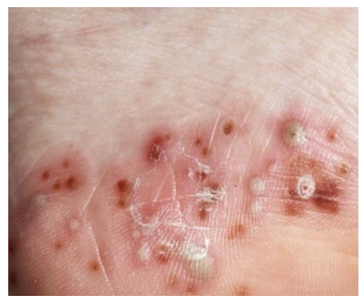
❖ Pustular psoriasis



Pustular psoriasis. sheets of erythema with pustules

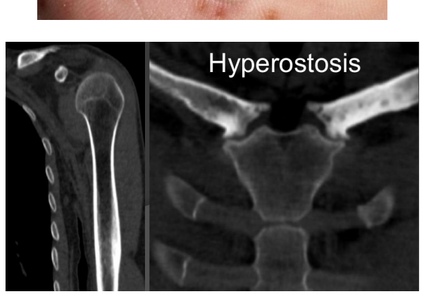
❖ **Generalized pustular psoriasis (Von Zumbusch):**

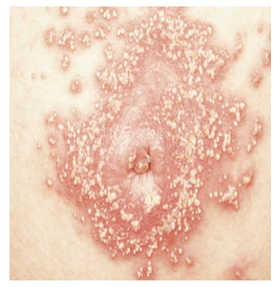
- Pustules over the body including nail beds, palms, soles.
- Skin is tender and may proceed to erythroderma.
- Fever, malaise and leukocytosis occurs.
- May form annular plaques.
- Resolves with extensive scaling.
- Onycholysis (separation of nail from it nail bed), shedding of the nails and hair loss (telogen effluvium) may follow 2-3 months later.
- Tongue may show circinate desquamation. In CBC you will find leukocytosis



❖ **Generalized pustular psoriasis (impetigo Herpitiformis in pregnancy):**

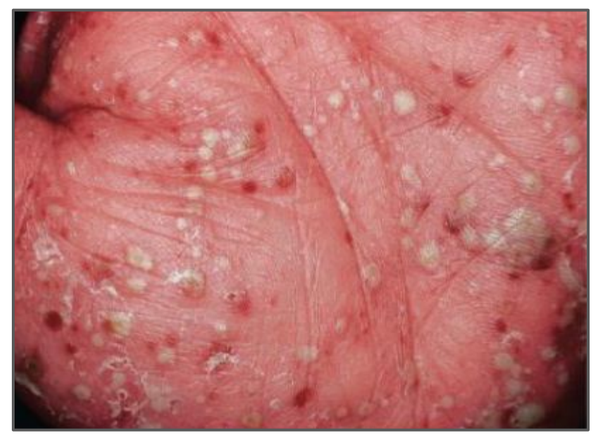
- Acute onset of pustules with annular configuration during the third trimester associated with hypocalcemia. - It may start in the 1st trimester.
- There is no personal history or familial psoriasis.





❖ **Localized pustular psoriasis (Palmoplantar Pustulosis):**

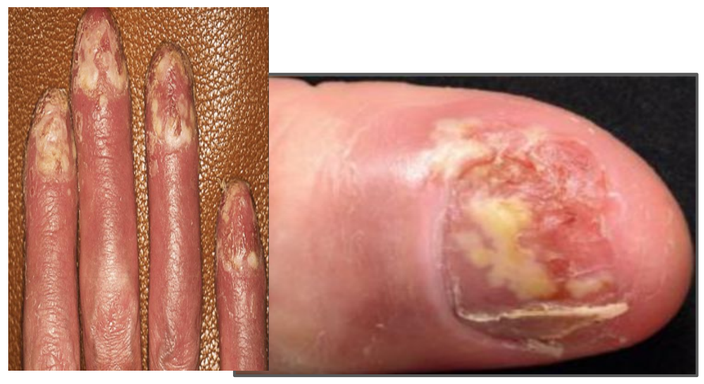
- Palmoplantar Pustulosis. Sterile pustules admixed with yellow brown macules.
- Sterile deeply-seated pustules and mixed with yellow brown macules.
  - Pustules sharply localized to the palms & soles (cannot extend further).
- Triggered by stress or infection.
- May be associated with bone lesions SAPHO syndrome (Synovitis, Acne, Pustulosis, Hyperostosis, Osteitis).
  - Hyperostosis in the medial ends of clavicle presented by tenderness. It can be erythematous & it is recurrent.
- Minority of patients have plaque or pustular psoriasis.



Palmoplantar pustulosis. some pustules are brown

❖ **Localized Pustular Psoriasis (Acrodermatitis Continua of Hallopeau):**

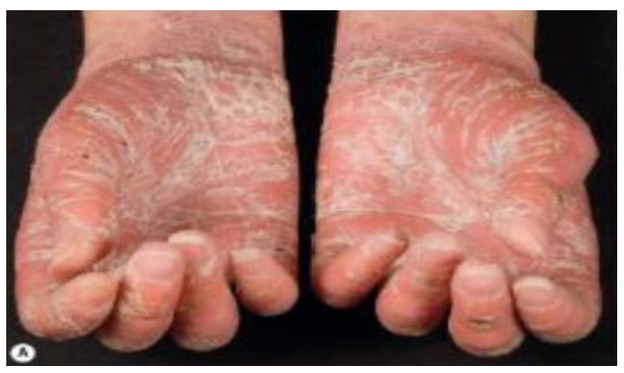
- Acro = pointed ends of the body such as the nose, ears & extremities.
- Pustular eruption of distal portions of fingers or toes that extends proximally.
- Most commonly affects the thumb.
- The phalanx becomes red, scaly and studded with pustules. Later the pustules burst leaving glazed painful skin and another bout of pustules appear.
- Can result in nail dystrophy or loss of nail.

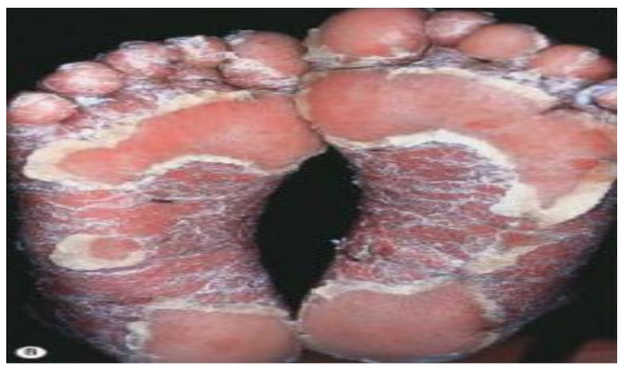


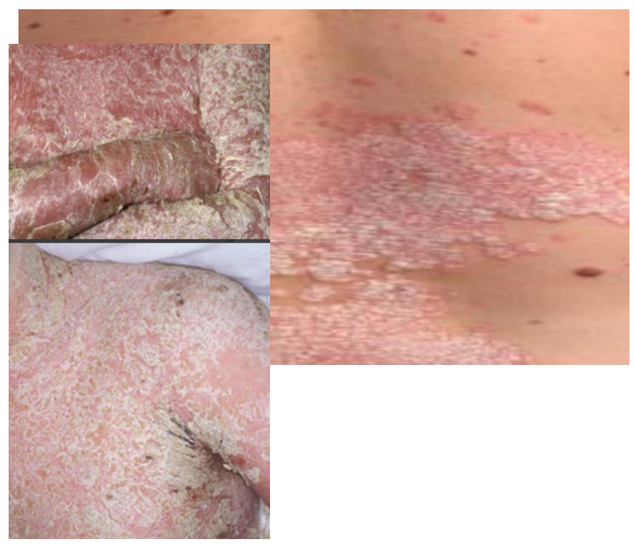
Acrodermatitis

❖ Erythrodermic psoriasis

- Erythroderma is a descriptive term characterized by erythema & scaling that can be caused by medications, lichen planus, ectopic dermatitis as well as psoriasis.
  -How to differentiate? by ruling out previous diagnosis of psoriasis or family hx of psoriasis.
- It affects all body sites ⩾ 90 %.
- Erythema is most prominent feature with scaling.
- Erythroderma is a state of skin failure in which the skin loses its thermo-regulatory function & becoming poikilothermic.
- Patients suffer from hypothermia due to vasodilation.
- Lower limb edema secondary to vasodilation and loss of proteins (due to loss of keratin from the skin & it should be compensated by high protein diet).
- High-output cardiac failure and impaired liver and renal functions also occur thus it is a systemic disease.
- Onset may be gradual or acute.
- Clues to diagnosis of psoriatic erythroderma mid include facial sparing (not always but most of the cases), characteristic psoriatic nail changes and previous plaques in classic location.



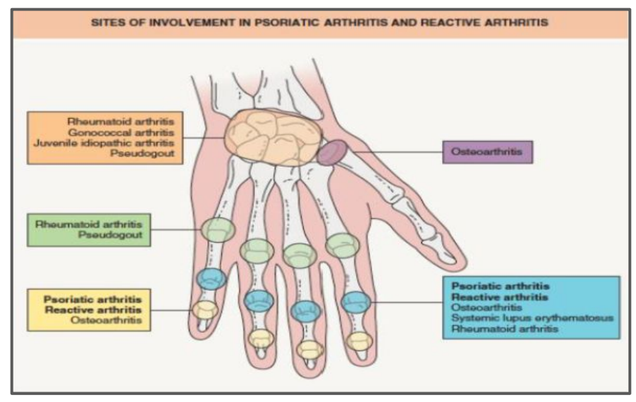


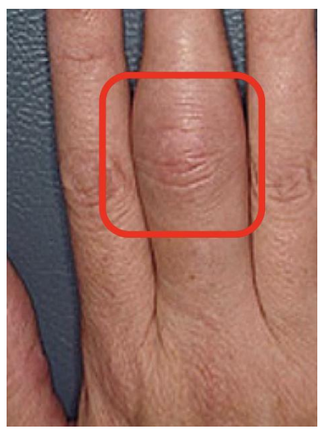


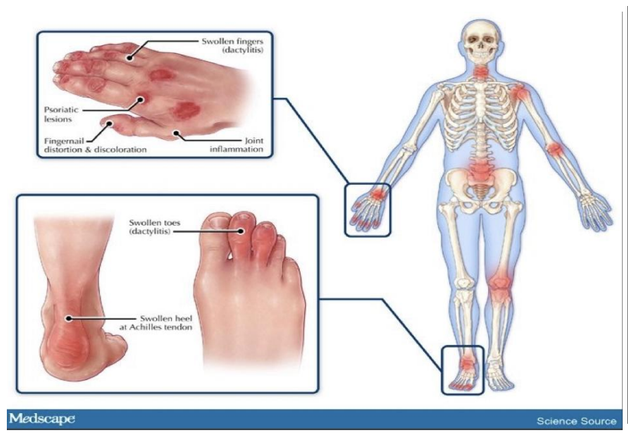
❖ Clinical features :

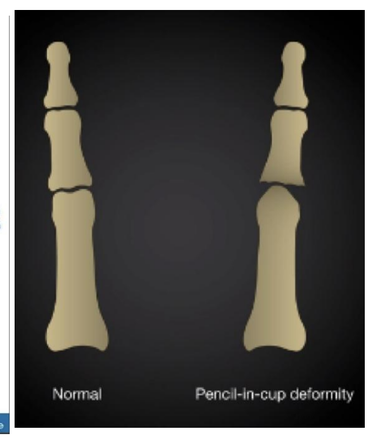
A. Psoriatic arthritis :

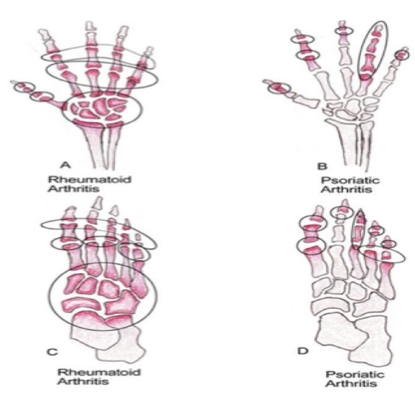
- 5 types;-
- Mono- and asymmetric oligoarthritis (oligo = 2-4).
- Arthritis of distal interphalangeal joints.
- Least common presentations:
  -Rheumatoid arthritis like.
  -Arthritis mutilans; it results in deformity of the hand.
  -Spondylitis and sacroiliitis.
- The most common presentation is inflammation of the distal (DIP mainly) and proximal (PIP) interphalangeal joints.
- Sausage digit if both DIP&PIP affected.
- Early diagnosis is important to avoid loss of function.
- Radiology shows:
  - Enthesitis is inflammation of the tendon insertion points (very important to ask about joint pain to prevent loss of joint function).
  - Periosteal bone formation and erosions (the only disease that can cause both bone erosions & formation is psoriasis).
  - Pencil in cup deformity distal part widening, proximal part thinning.

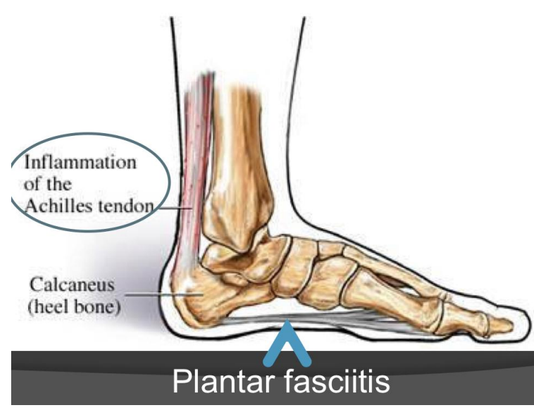






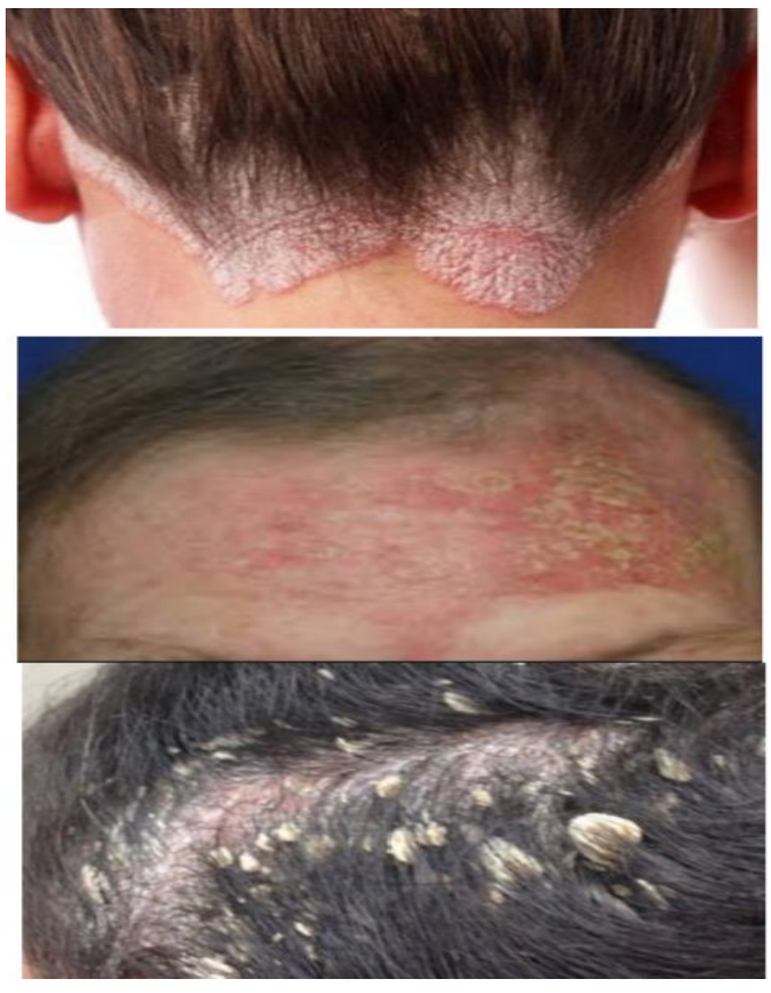






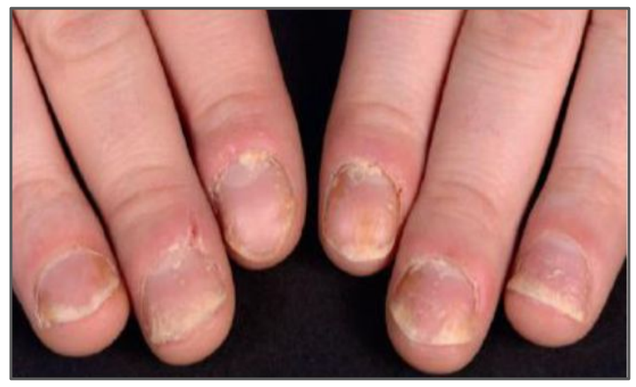
B. Scalp psoriasis

- The back of the head is a common site.
- May involve the whole scalp.
- Appears as well-defined erythematous plaques with thick silvery white scale.
- May extend beyond the hairline.
- May coexist with seborrheic dermatitis called sebopsoriasis.
  -Scales of seborrheic dermatitis is greasy yellowish scales.
- Sebopsoriasis the lesions are localized to seborrheic areas like; scalp,nasolabial fold,eyebrows,retrosternal region, folds.
- Scales sometimes are asbestos like (thick) and attached to the hairs (Pityriasis amintecea).
  -Pityriasis amintecea can be seen in seborrheic dermatitis; secondary infected atopic dermatitis and T. capitis (ask about contact with animals).
  - Pityriasis amintecea in seborrheic dermatitis occurs in seborrheic areas such as the nasolabial folds, the eyebrows, behind the ears & chest.
  -In atopic dermatitis, we should ask about hand eczema.

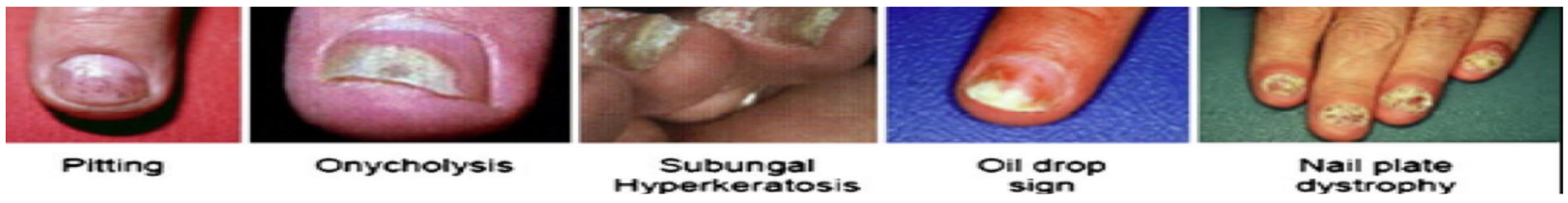


C. Nail psoriasis

- Psoriatic nail disease occurs in 10-70% of all patients with psoriasis.
- Less than 5% of psoriatic nail disease cases occur in patients without other cutaneous findings.
- Fingernails are more affected than toenails.
- Psoriasis affects nail matrix (pitting most common), nailbed (oil-spot) and hyponychium (subungual hyperkeratosis).
  - > 25 pits are more in favor of psoriasis.
- Pneumonic (STOP)
  - S: Subungual hyperkeratosis, Splinter hemorrhage
  - T: Thickening
  - O: Onycholysis, Oil spots
  - P: Pitting



- Pitting
- Oil-spot
- Onycholysis
- subungual hyperkeratosis and thickening of the nail in general



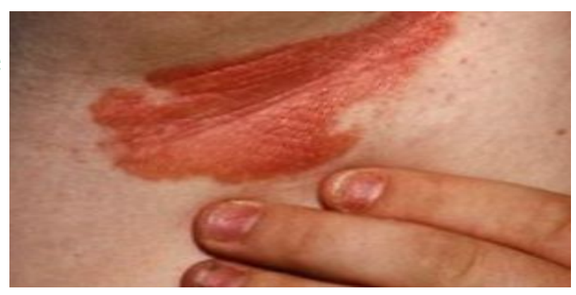
D. Oral mucosa:

- Migratory annular erythematous lesions with hydrated white scale over the tongue (geographic tongue) observed mainly in pustular psoriasis patients. psoriasis rarely affects mucosa, and most common presentation in mucosa is geographic tongue

E. Inverse psoriasis

- Psoriasis of the axilla

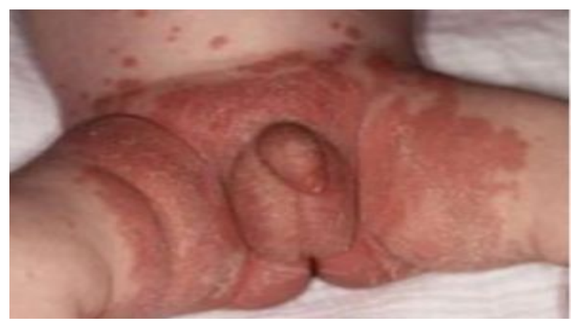
no scales because the area is wet



F. Napkin psoriasis

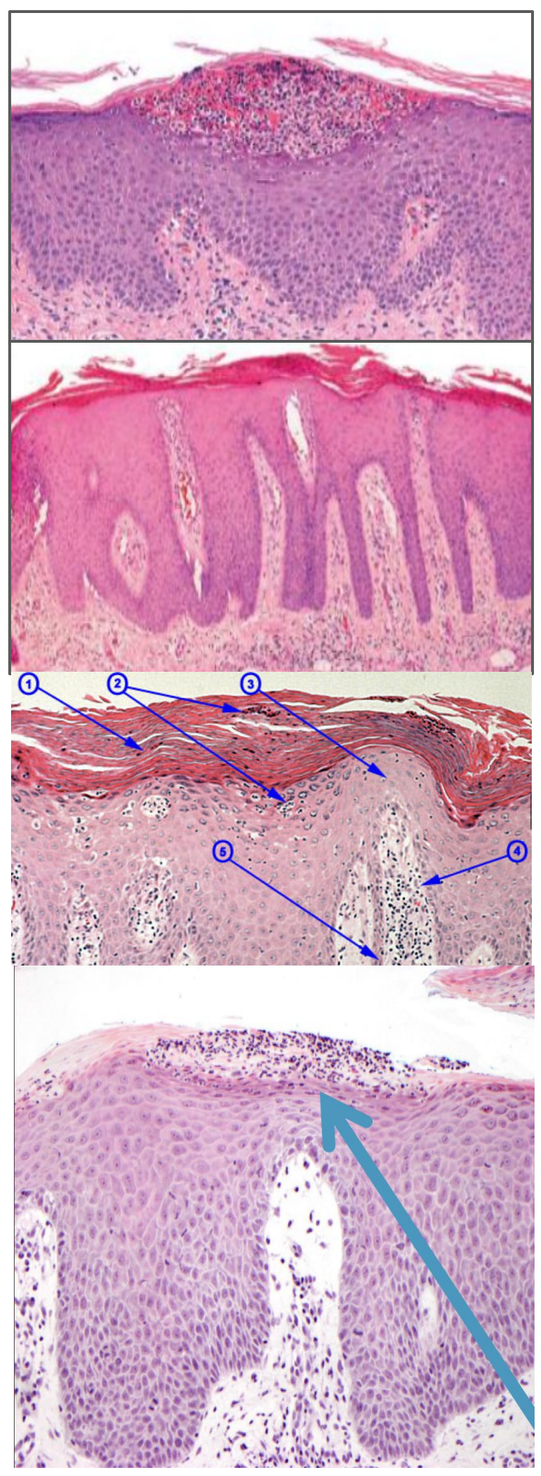
- Ddx of napkin psoriasis: irritant contact napkin dermatitis or candidal napkin dermatitis.
- How to differentiate?
  - Well-defined papules or plaques of psoriasis.
  - Erythema in psoriasis is severe (vivid color).

If you leave the area open for some time, it will show scales.

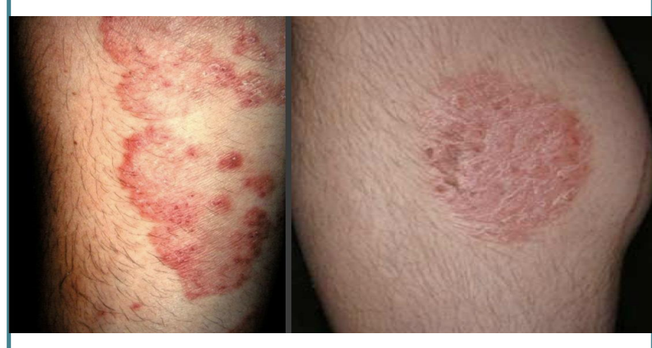
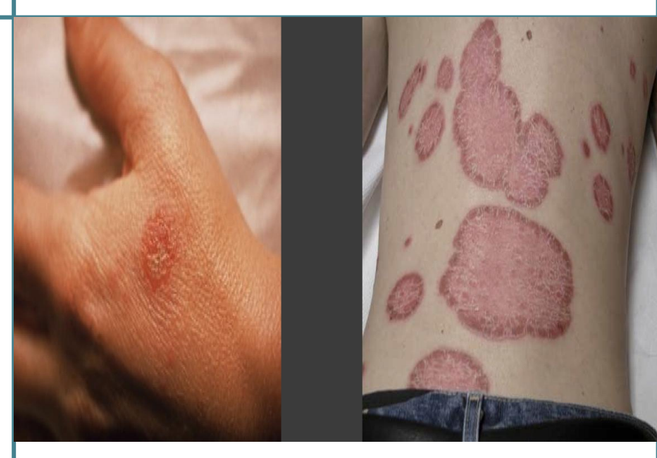
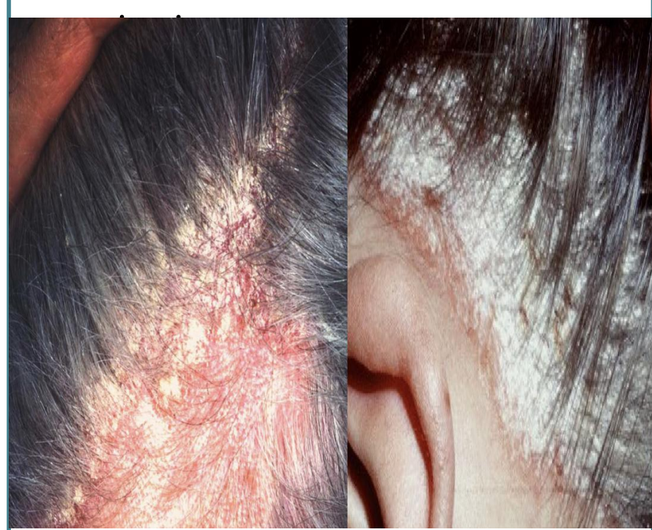
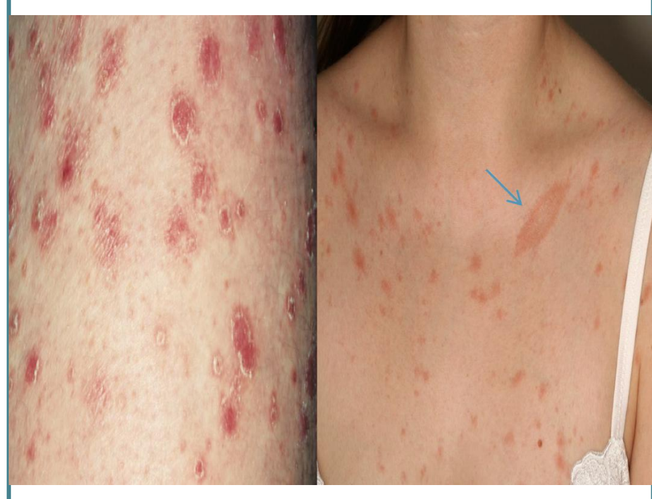
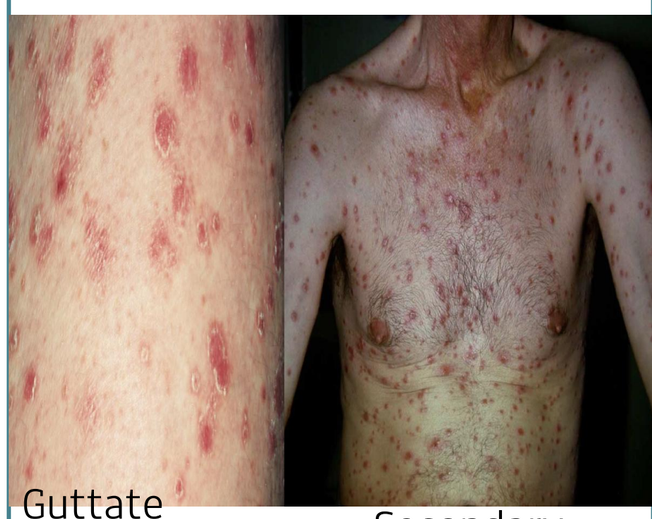


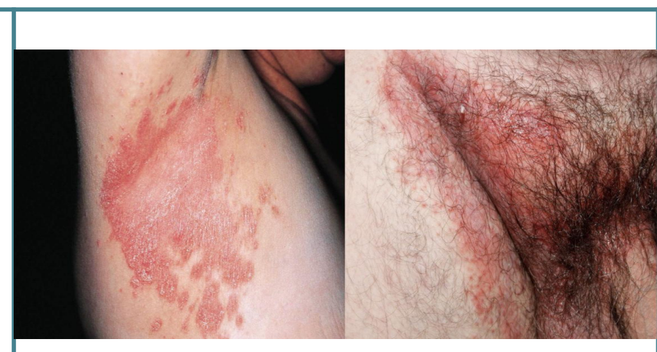
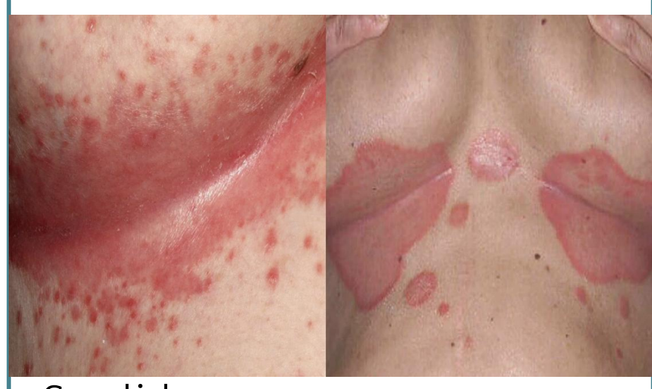
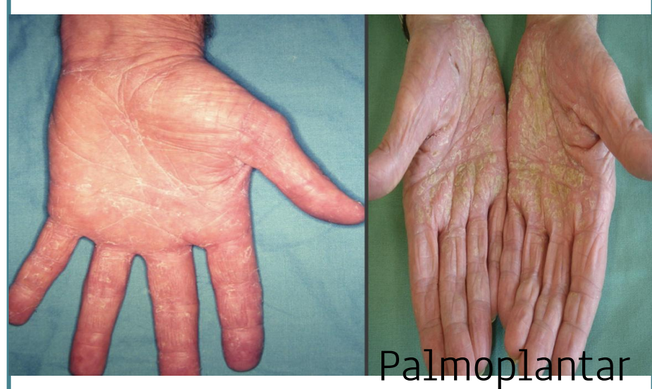
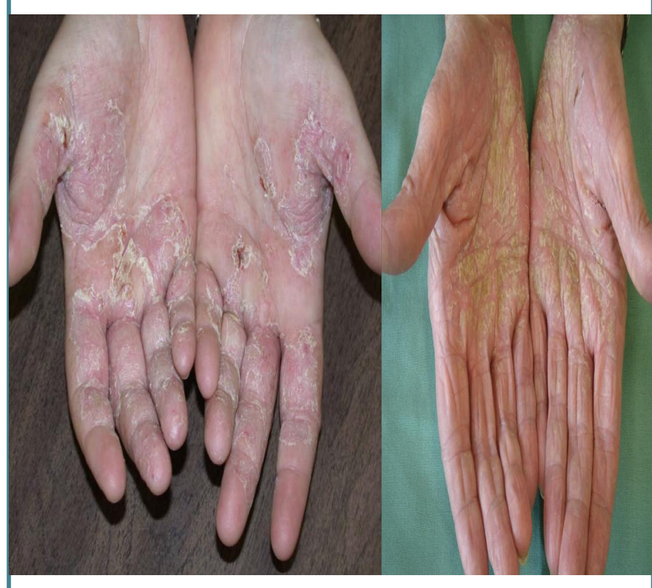
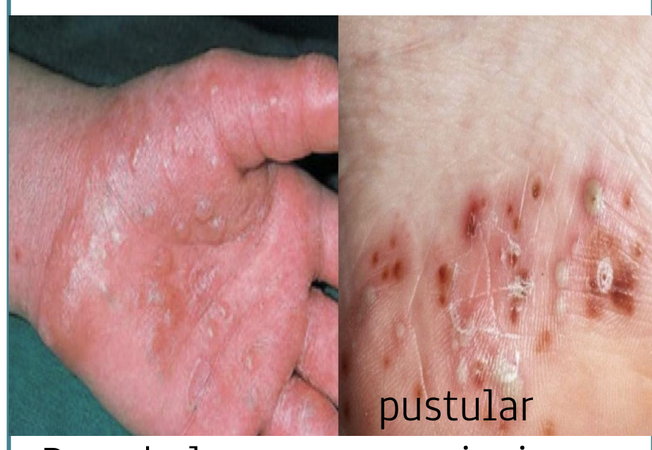
❖ Psoriasis pathology

- Parakeratosis (nuclei retained in the horny layer).
  - Normally, there should be NO nuclei in stratum cornea.
  - The nuclei had no time to disappear (the turnover time of keratinocytes is shortened to 10 days in psoriatic patients).
- Irregular thickening of the epidermis over the rete ridges (to accommodate all the dividing cells) but thinning over dermal papillae.
- Epidermal polymorphonuclear leukocyte infiltrates (munro microabscesses).
- Dilated capillary loops in the dermal papillae
- T-lymph infiltrate in the upper dermis.
- Classically, Superficial perivascular lymphocytic infiltrate with even elongation of rete ridges ,dilated capillaries in papillary dermis which associated with spongiosis, acanthosis and parakeratosis
- In late lesions, accumulation of neutrophils within a spongiotic pustule "spongiform pustule of Kogoj" or sub-corneal accumulation of neutrophils "microabscess of Munro"



Differential diagnosis	
❖ **Localized plaques** ● Tinea ● Eczema ● Seborrhoeic dermatitis	❖ **Flexural** ● Tinea ● Eczema ● Candidiasis ● Seborrhoeic dermatitis
❖ **Guttate** ● Pityriasis rosea ● Drug eruption ● Secondary syphilis	❖ **Erythrodermic** ● Eczema ● Lichen planus ● Drug
❖ **Palmoplantar** ● Tinea	

LOCALISED PATCHES/PLAQUES	**Tinea corporis**	• Affects body. • Lacks symmetrical lesions. • Presence of peripheral scale and central clearing.	
	Discoid eczema	• Individualized plaques more pruritic than Psoriasis. • Lacks silvery scale. • Less vivid color than psoriasis.	 Discoid eczema psoriasis
	Seborrhoeic dermatitis	• Characterized by yellowish scaling and erythema. • Localized to many of the same areas as psoriasis. • Affects furrows of face Generally restricted to hairline.	 psoriasis
GUTTATE PSORIASIS	**Pityriasis rosea**	• Difficult to distinguish from acute guttate psoriasis. • Presents first as single large patch called herald patch. • Progresses to a truncal rash of multiple red scaly plaques (‘Christmas tree’ distribution)	 Guttate psoriasis Pityriasis rosea
	Secondary syphilis	• Search for characteristic primary syphilitic lesion, lymphadenopathy, and lesions on face, palm and soles. • Conduct serology and skin biopsies to confirm diagnosis.	 Guttate psoriasis Secondary syphilis

FLEXURAL PSORIASIS	**Tinea cruris**	• Affects groin area. • Characterised by central clearing with advancing edge with fine scale.	 Flexure psoriasis Tinea cruris
	Candidiasis	• Characteristic peripheral pustules and scaling different from psoriasis. • Yeast cultures are Diagnostic.	 Candida intertrigo Psoriasis
PALMOPLANTAR PSORIASIS	**Tinea manum**	• Ringworm of hands. • Fine powdery scale, particularly involving palms and palmar Creases. • Usually asymmetrical.	 Tinea Manum Palmoplantar psoriasis
	Hand and foot eczema	• Hyperkeratotic forms difficult to distinguish from psoriasis. • Biopsies may assist diagnosis. • Look for history of atopy, a lack of psoriasis elsewhere on body, and evidence of eczema elsewhere on skin.	 Hand eczema
	Pompholyx of palms and soles (dyshidrotic eczema).	• Presents as clear vesicles contrast to white/yellow pustules in pustular psoriasis. • Accompanied by intense pruritus.	 Pompholyx pustular psoriasis

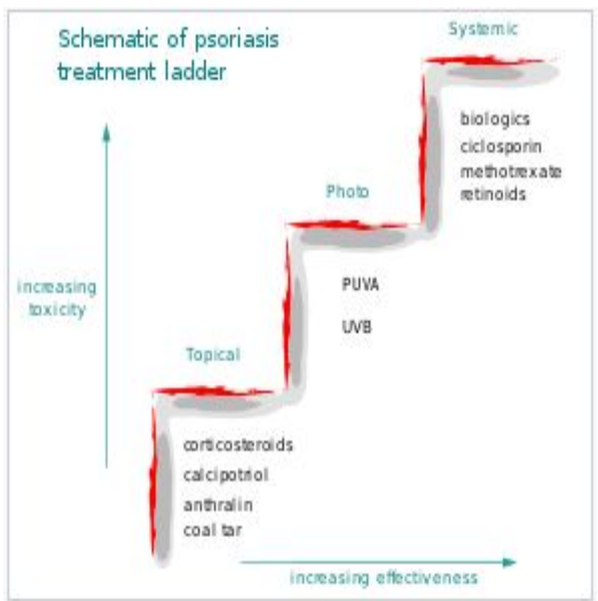
❖ Comorbidities :

- Cardiovascular disease.
- Metabolic syndrome and its individual components (i.e., hypertension, obesity, impaired glucose regulation, and low HDL levels).
- Higher risk of malignancies including lymphoma, and nonmelanoma skin cancer (PUVA, systemic therapy).
- Autoimmune diseases (e.g., inflammatory bowel disease, multiple sclerosis).
- Psychiatric conditions including anxiety and depression.

❖ Treatment:

Topical(for focal disease)	Biologic therapy	Phototherapy	Systemic
• Corticosteroids. • Vitamin D analogs. • A combination of the first two is called daivobet ointment • Retinoids. • Calcineurin inhibitors. • Anthralin. • Coal tar.	• TNF inhibitors: adalimumab, Etanercept, Infliximab • IL-12/IL-23: ustekinumab.	• UVB • UVA • PUVA	• Acetretin. • MTX (methotrexate). • Cyclosporin.

Chronic plaque type	Pustular type
Methotrexate first line	Retinoids(Acitretin) first line
NBUVB/PUVA	Cyclosporine
Anti-TNF	Methotrexate
Anti-IL 12/23	NBUVB/PUVA
Anti-IL 17A	
Cyclosporine	
Retinoids (Acitretin)	



Psoriasis topical therapy	
Topical Corticosteroids	**-Indication:** Plaque psoriasis<10 %. **-Dosing**: monotherapy 1-2 times daily. −Combined with other topical agents, UV light, and systemic agents. −Toxicity of topical steroid: • Local—skin atrophy,telangiectasia,striae, purpura, contact dermatitis, rosacea/acne if used on the face. • Systemic—HPA axis suppression may occur with use of medium-and high-potency topical steroids causing Cushing syndrome, DM or HTN. This will be lessened by intermittent or localized use. • Increased IOP,glaucoma, and cataract have been reported with use around eye.
Vitamin D analogues	Indication: Plaque-type psoriasis. relapses are less likely with this medication, prevent relapses Dosing: Twice daily. Calcitriol,calcipotriol, tacalcitol,maxacalcitol are used. Combination of calcipotriene and betamethasone ointment available. Adverse reactions: • Transient Irritation in lesional and perilesional skin. • Elevation of serum calcium more likely to occur in patients treated with > 100g/wk in adults and >50 g/wk in children. • Photosensitivity. • Since calcipotriene is inactivated by UVA, it is important to apply calcipotriene after and not before UVA exposure.

Tazoretene **Vitamin A analogues** Retinoid analogue	• Best used in combination with topical corticosteroids. Once daily for 12 weeks. • Probably best reserved for thick, recalcitrant plaques of psoriasis. unresponsive psoriasis Adverse reactions: • Skin irritation in lesional perilesional skin. • Photosensitizing. • Pregnancy and nursing: Category X (not used in pregnant/nursing lady)
Calcinurin inhibitors (tacrolimus and pimecrolimus)	-Indication: Intertriginous and facial psoriasis to avoid using steroids on the face because ofits side-effects (acne/rosacea/eye complications). -Side effects: stinging, burning,itching. - Better not used below 2 years of age.
Anthralin	Indications: Use has been declined these days. Dosing: short-contact therapy for 2h once daily. • 2% dithranol in Lassar's paste applied for 2 honce daily recommended optimal for home use. Adverse reactions: skin irritation and staining of skin and clothing.

❖ Psoriasis systemic therapy:

Agent	Administration (route; frequency)	Side effects
Acitretin	Oral; once daily	• Mucocutaneous changes; hypertriglyceridemia, elevated LFT • Enhanced efficacy when combined with UVB/ PUVA • Pregnancy category X
Cyclosporine	Oral; twice daily	• Nephrotoxicity, hypertension • Limited duration of continuous treatment (1 year) • Risk of skin cancer if history of PUVA • Reversible changes in serum lipids • Pregnancy category C
Methotrexate	Oral, SC, or IM; once weekly	• Myelosuppression, hepatotoxicity, pulmonary fibrosis • Parenteral administration may minimize GI side effects • Folate supplementation may be recommended • Pregnancy category X

Retinoids /Acitretin	Methotrexate
• VIT A analogue. ○ Inhibits abnormal keratinization. • **Indication:** ○ Severe plaque type psoriasis. ○ HIV-positive patients with severe psoriasis. Why used for HIV patients? It works only on keratinocytes & their differentiation & has no role in the immunity, does not suppress the immunity. ○ 10-75 mg/d given as a single dose. ○ The preferred schedule is acitretin monotherapy for 2 weeks followed by the addition of phototherapy. • **Adverse events:** ○ Dry eyes and lips ○ Lipid derangements ○ Pancreatitis ○ Hyperostosis ○ Pseudotumor cerebri ○ Hepatotoxicity ○ Teratogenicity (only female:3 years for wash out).	Folic acid antagonist blocks dihydrofolate reductase leading to inhibition of folic acid metabolism. • Antiproliferative, induces apoptosis and an immune and anti-inflammatory modulator. **Indication:** • Severe, recalcitrant, disabling psoriasis not adequately responsive. • Often used as the primary agent to treat psoriatic arthritis. • Absolute contraindications: 1. Pregnancy. 2. Nursing mothers. 3. Alcoholic liver disease or other chronic liver disease. 4. Immunodeficiency syndromes. 5. Bone-marrow hypoplasia, leukopenia, thrombocytopenia, or significant anemia. • Relative contraindications: 1. Abnormalities in renal function. 2. Abnormalities in liver function. 3. Active infection because it's an immune modulator. 4. Obesity. 5. Diabetes mellitus.

❖ Toxicity of methotrexate:

- Elevated LFT results - Minor elevations of LFT results are common; if elevation exceeds 2xnormal, must check more frequently; if exceeds 3xnormal, consider dose reduction; if exceeds 5 Discontinue.
- Anemia, aplastic anemia, leukopenia, thrombocytopenia.
- Interstitial pneumonitis.
- Ulcerative stomatitis. GI ulceration and bleeding.
- Nausea, vomiting, diarrhea.
- Alopecia.
- Pregnancy: category X; men and women considering conception should be off methotrexate for 3 months before attempting to conceive.
- How to monitor for methotrexate induced liver fibrosis: Procollagen 3 N terminal peptide(P3NP) unreliable in PsA. Liver biopsy. High risk and low risk? High risk are pts with comorbidities. High risk when the methotrexate cumulative dose reach 1-1.5 grams you better do liver biopsy, in low risk 3.5 grams of methotrexate cumulative dose do liver biopsy. Fibroscan type of ultrasound you can use it to see liver fibrosis

❖ Cyclosporin:

- Inhibits calcineurin phosphorylation, stop the division of cells. Carries the risk of cancer if combined with phototherapy.
- 2 to 5mg/kg/day for 12 to 24 weeks (maximum) to limit cumulative nephrotoxicity. -Shows quick improvement of the condition.
- Indication:
  - Erythrodermic psoriasis, Generalized pustular psoriasis, and Palmoplantar psoriasis, Severe recalcitrant plaque psoriasis. In other words, we use cyclosporine for severe entities of psoriasis.
  - Do baseline creatinine repeat twice.
  - Monitor creatinine for nephrotoxicity (very important). If increased by 30% or less, you should decrease the dose by 1 mg. If increased by >30%, you should discontinue the medication.
  - Monitor blood pressure for secondary HTN.
  - Monitor the lipid level, because the medication cause hyperlipidemia.

❖ Phototherapy

- Narrowband ultraviolet B: 311 nm. Broad band UVB 280.-320 nm
- PUVA (psoralen ultraviolet A): 320-400 nm.
- NB-UVB most commonly used due to: easier use, and fewer side effects when compared with BBUVB or PUVA.
- NBUVB (narrow band) leads to rapid clearance of lesions than BB-UVB (broad band). Best to treat psoriatic patient with narrow band.
- Broadband ultraviolet B: 290-313 nm.
- **Indications:**
  - Unresponsive generalized psoriasis/chronic plaque psoriasis (including guttate and seborrhoeic).
  - May aggravate psoriatic erythroderma and generalized pustular psoriasis.
  - Contraindications: Patients with known LE (lupus), H/O melanoma or NMSC (non-melanoma skin cancer), Xeroderma pigmentosum.
- **Toxicity of NBUVB:**
  - **Acute:**
    - Erythema, pruritus, burning, blister (NBUVB).
  - **Long term:**
    - Photoaging, lentigines, telangiectasias, cataract (especially patients using psoralen).
    - Advise use of protective goggles and genital shields during treatment. Protect the eye & genitalia
    - NB-UVB therapy should be considered first-line therapy in pregnant patients with plaque and guttate psoriasis who need systemic therapy.
    - Can be used with emollients, topical corticosteroid or VIT D analogues or systemic treatment like retinoids and MTX.
- **Mode of action NBUVB:**
  - Locally immunosuppressive.
  - Inhibition of epidermal hyperproliferation and angiogenesis.Selective reduction in T lymphocytes within psoriatic skin via apoptosis.

PUVA:

- Dosing: 8-MOP (Methoxypsoralen) 0.4-0.6 mg/kg, taken 1-2 h before exposure to UVA. Treatment 2-3 times/wk.
  - Psoralen is an oral medication taken 2 hours before being exposed to UV light.
- UV protective eye wear when outdoors for 12 hrs post ingestion.
  - Psoralen stays in the blood for 8 hours at least thus patients have to wear goggles even after the session to protect the eyes.
- Combination therapy with cyclosporin increases risk of squamous cell carcinoma.
- Side-effect of psoralen:
  - Gastric upset, patient should avoid sunlight for the whole day after receiving the
- MOA:
  - Has effects on epidermal keratinocytes and Langerhans cells (antigen-presenting cells) (similar to UVB irradiation).
  - Has effects on dermal cells including granulocytes and T Lymphocytes.
  - Psoralen intercalates between DNA base pairs and, on exposure to UVA, forms psoralen DNA crosslinks that prevent DNA replication.
  - Death of antigen presenting cells.

- Patients who are receiving more 200 sessions of PUVA are at risk of skin cancer.

❖ Topical PUVA:

- For localized psoriasis to palms and feet.
- Use 0.1% 8-methoxypsoralen in emollient and treat 2-3 /wk.
- Apply 30 min before UVA.
- Bath PUVA-50 mg of 8-Methoxypsoralen in 100 L of water, 20-30 min pre-exposure.

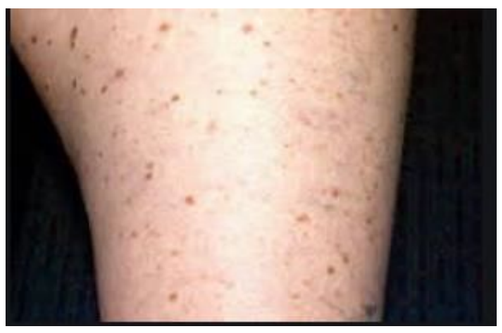
❖ Toxicity of PUVA:

- **Acute:**
  - Nausea and vomiting are common.
  - Dizziness and headache are rare.
  - Burns & erythema: peaks at 48-96 h (in UVA) while in narrowband in 8 hours (UVB).
  - Pruritus, Blisters
  - Tanning: starts 1 wk after PUVA. brown in color
  - Photo-onycholysis, melanonychia.
- **Chronic:**
  - Photocarcinogenesis (SCC, BCC, and possible melanoma).
  - Increased risk of photocarcinogenesis Caucasians with skin types I-III after 200 treatments; this risk not present for non-Caucasians.
  - Photoaging and lentigines are common, especially in patients of skin types I-III and are cumulative UVA dose dependent.

- If the patient carrying one of these 3 skin types & they are exposed to puva, the risk for cancerogenesis + lintigens increased.

Melanonychia

onycholysis



- **Excimer laser:** Used for resistant localized psoriasis such as scalp and palmoplantar psoriasis. Laser with a wavelength similar to narrow band.

❖ New oral medications:

- **Apremilast**:
  - Is a phosphodiesterase 4 (PDE4) inhibitor.
  - Promotes anti-inflammatory processes.
  - Used for moderate-to-severe plaque psoriasis.
  - 30 mg BID for 16 weeks.
  - Adverse events: nausea, diarrhea, nasopharyngitis, headache, **suicidal thoughts, depression**.

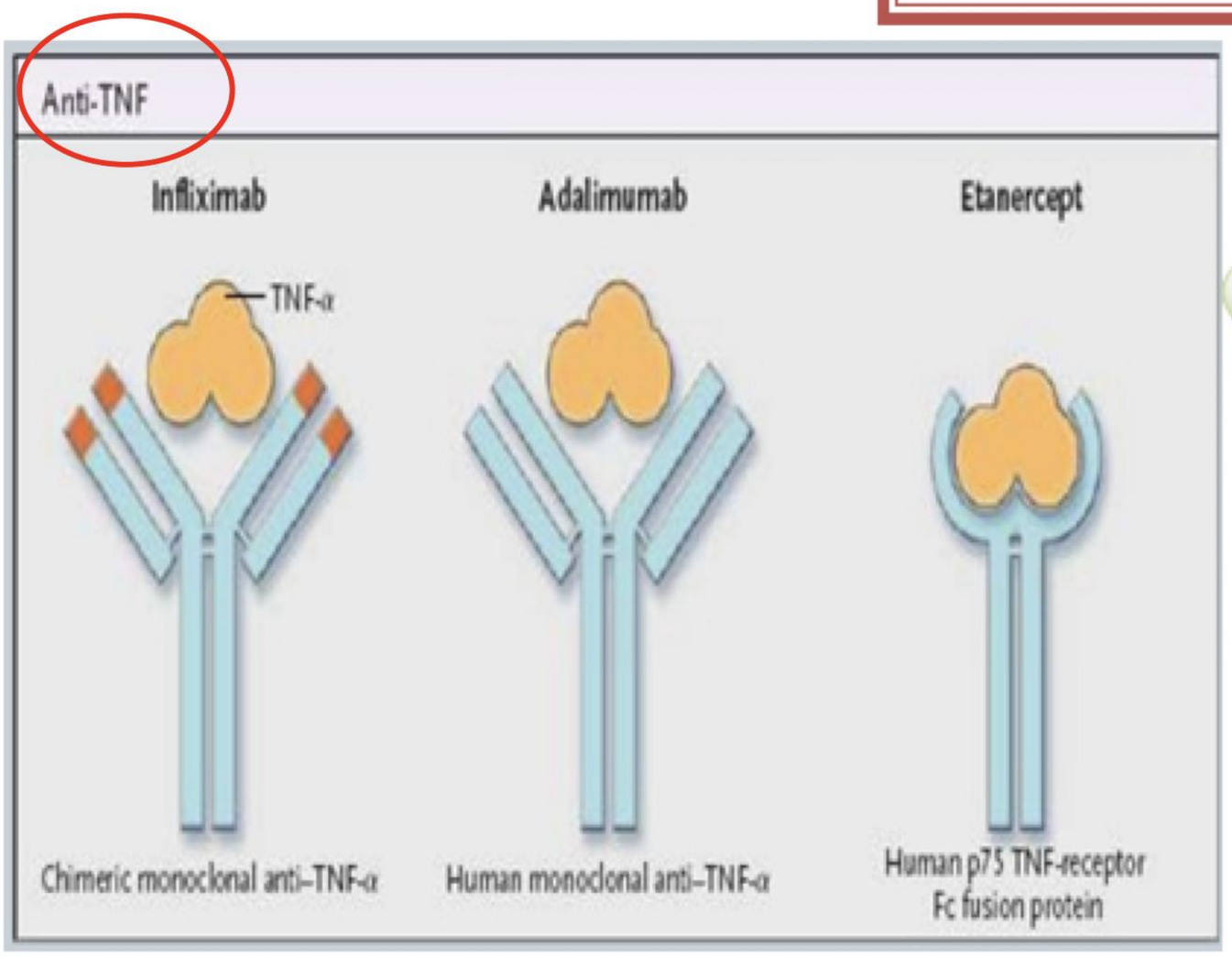
❖ Biologics:

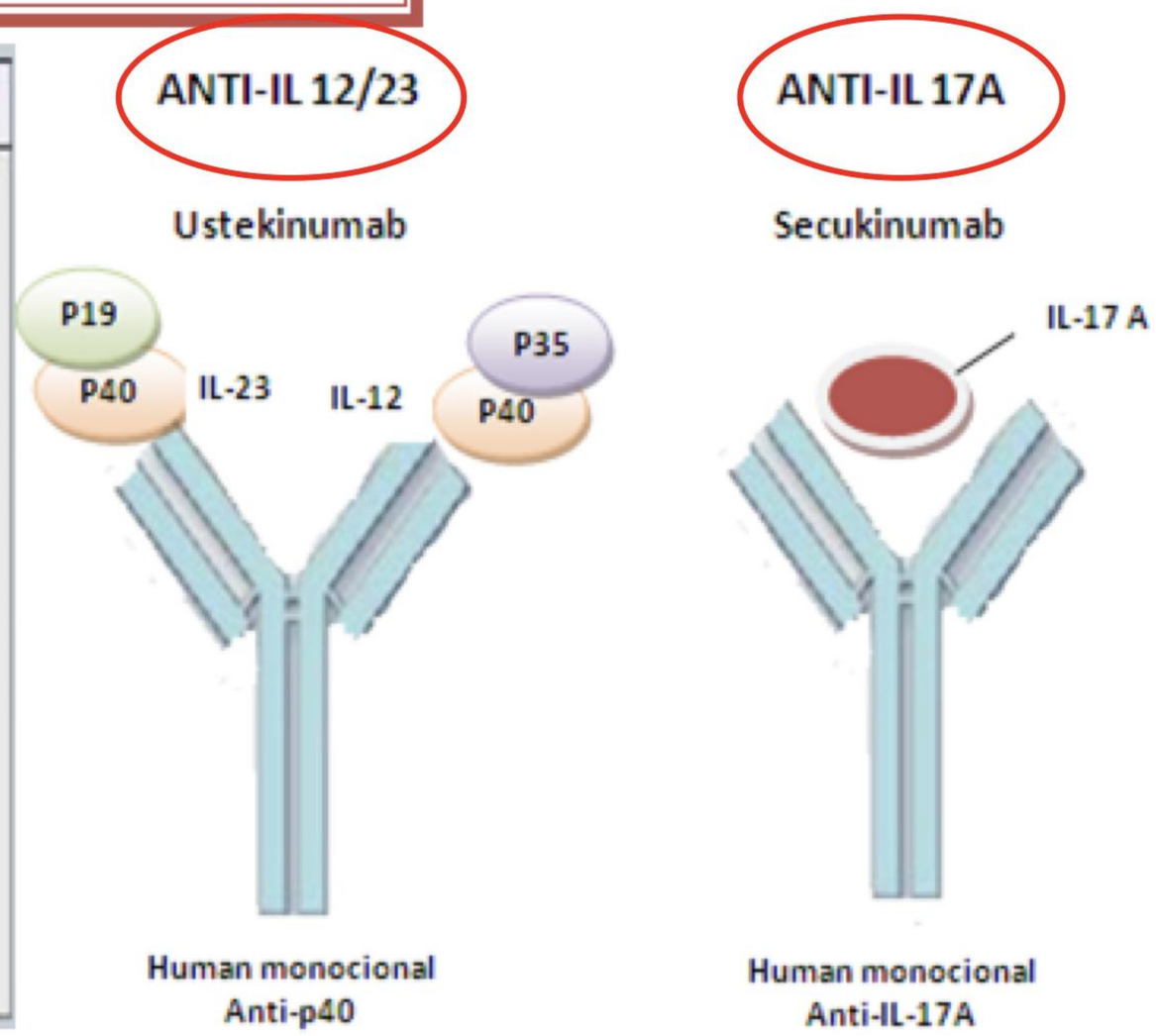
- Target key parts of immune system that drive psoriasis. Biological agents include:
  - Tumor necrosis factor-αinhibitors:
    - Etanercept.
    - Adalimumab.
    - Infliximab.
  - Non TNF inhibitors- αinhibitors:
    - Interleukin (IL-12 / IL-23) inhibitor (Ustekinumab).
    - Interleukin (IL-17) nhibitor (Secukinomab).

Indications of biologics:

- Severe disease: Psoriasis Area Severity Index (PASI) score of 10 or more (or a body surface area (BSA) of 10% or greater) AND a Dermatology Life Quality Index (DLQI) of >10.
- Phototherapy and alternative standard systemic therapy are contraindicated or cannot be used.
- Unresponsive to standard systemic therapy.

Anti-cytokine strategies





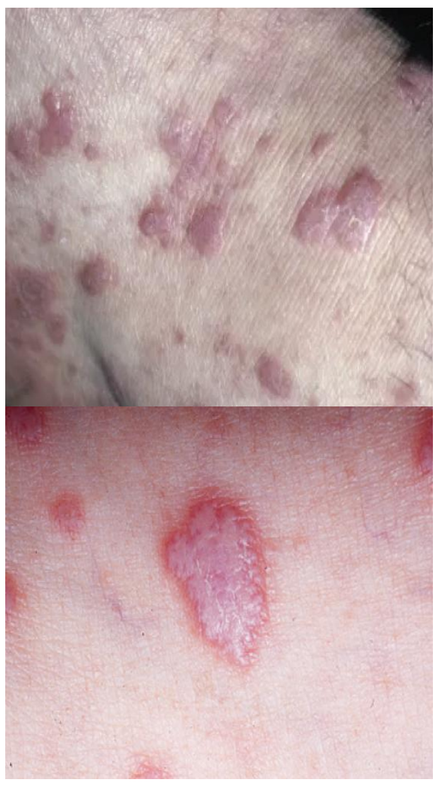
❖ Contraindications of biologics:

- Contraindications:
  - Patients with active, serious infections.
  - New York Heart Association class III or higher congestive heart failure.
  - History of demyelinating disease (e.g. multiple sclerosis).
  - Serious hematologic disease (e.g. aplastic anemia).
  - Current malignant tumor or prior malignant disease.
  - Immune-compromised by congenital or acquired immunodeficiency syndrome.

❖ Investigations for biologics:

- Complete blood cell count including platelet count.
- Chest x-ray
- Liver function tests.
- Renal panel.
- Hepatitis panel.
- Tuberclin test or Quantiferon Gold assay.
- HIV.
- Pregnancy test.
- Avoid vaccination with live vaccines (varicella, mumps, measles, rubella, oral typhoid, yellow fever).
- Avoid live-attenuated vaccines (including intranasal influenza and the herpes zoster vaccine).

Lichen planus



The classical picture.

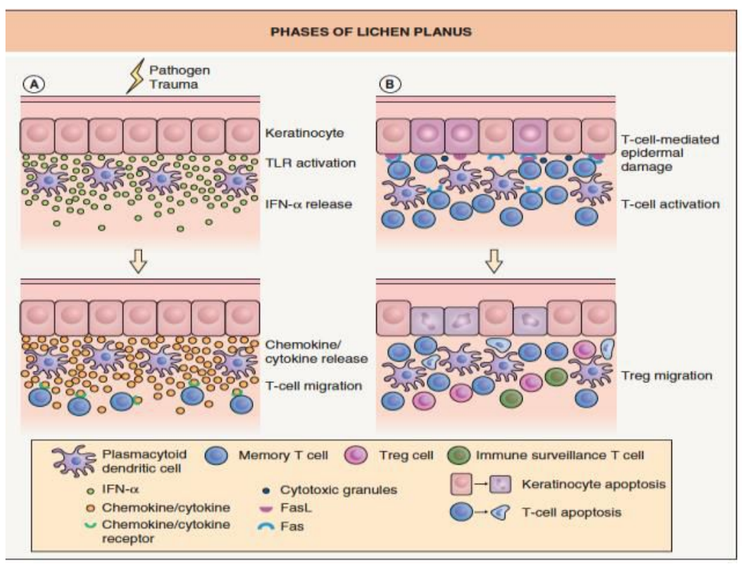
- An idiopathic inflammatory disease of the skin and mucous membranes. Classic LP is characterized by pruritic, flat topped plane, polygonal violaceous papules that favor the extremities. (5Ps)
- The onset of LP occurs most commonly during the 5-6 th decade. 2/3rd of patients developing the disease between the ages of 30 and 60 years.
- Increased prevalence of Hepatitis C.
- Lichen Planus is an immune-mediated polygenic skin disorder. Various environmental triggering factors, e.g. Stress, infections and medications may elicit the disease in genetically predisposed individuals.

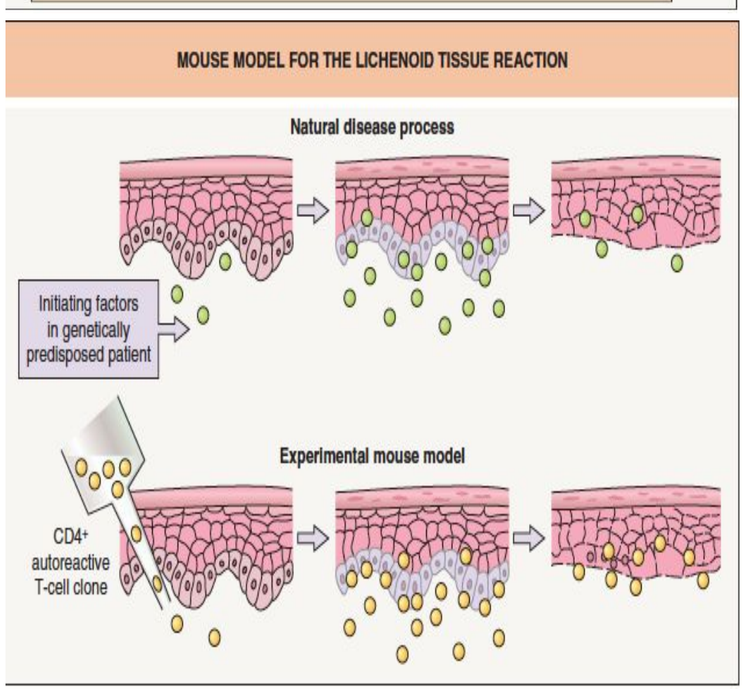
❖ Causes:

- Idiopathic complex polygenic condition.
- Genetic predisposition (HLA-A5, HLA-A3, 147,148, HLA-B7,143, HLA-DR1, 149,150 in the Arab population).
- Liver disease (sclerosing colangitis, chronic liver disease, Hepatitis C).
- Vaccination and drugs (IF, Antimalarials).
- Contact sensitizer(mercury amalgam, color film developers, methylacrylic esters like crowns ,veneers).
- Six single nucleotide polymorphisms (SNPs) were found to be associated the HLADQB1.
- 01:01 in Sardinian &Mexican population.
- 05:01 haplotype associated with Lichen planus.

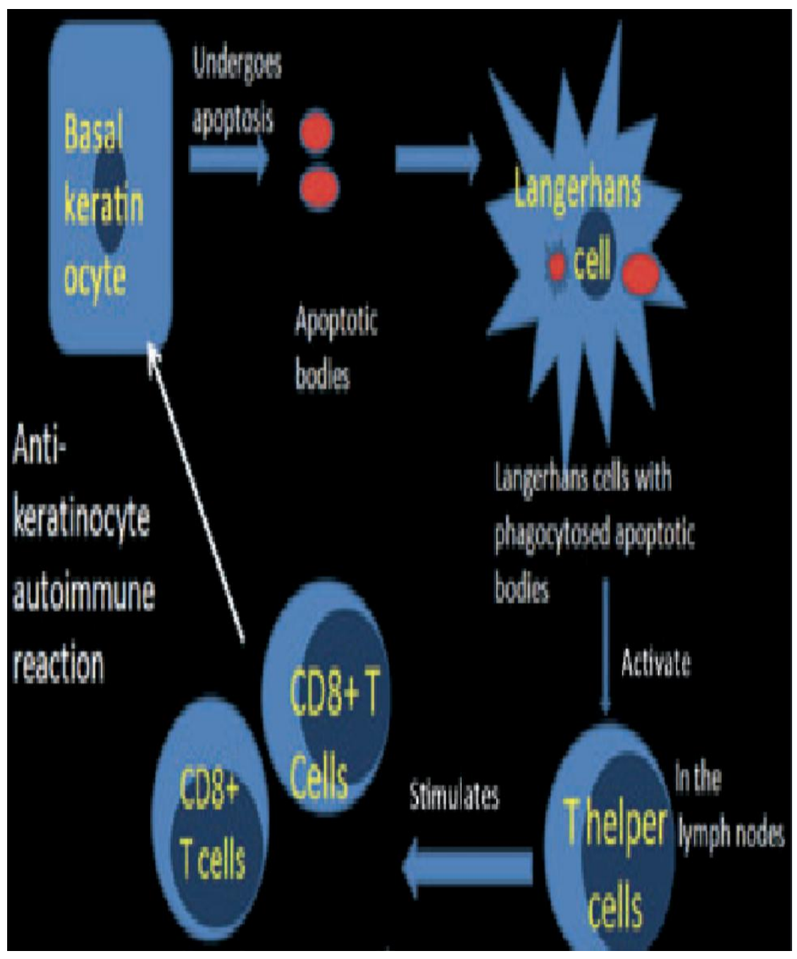
❖ Immunopathogenesis:

- The etiology of lichen planus is unknown.
- An immune-mediated mechanism involving activated T cells, particularly CD8+ T cells (cytotoxic cells which will kill the keratinocytes), directed against basal keratinocytes has been proposed.
- Upregulation of intercellular adhesion molecule-1 (ICAM-1) and cytokines associated with a Th1 immune response, such as interferon (IFN)-gamma, tumor necrosis factor (TNF)-alpha, interleukin (IL)-1 alpha, IL-6, and IL-8, may also play a role in the pathogenesis of lichen planus.
- The target in psoriasis and lichen planus is basal cells, the difference is what comes after. In psoriasis excessive cell division and differentiation, lichen planus the cells die and undergo macular degeneration.





Cytotoxic cells predominate here with Th cells



❖ Epidemiology:

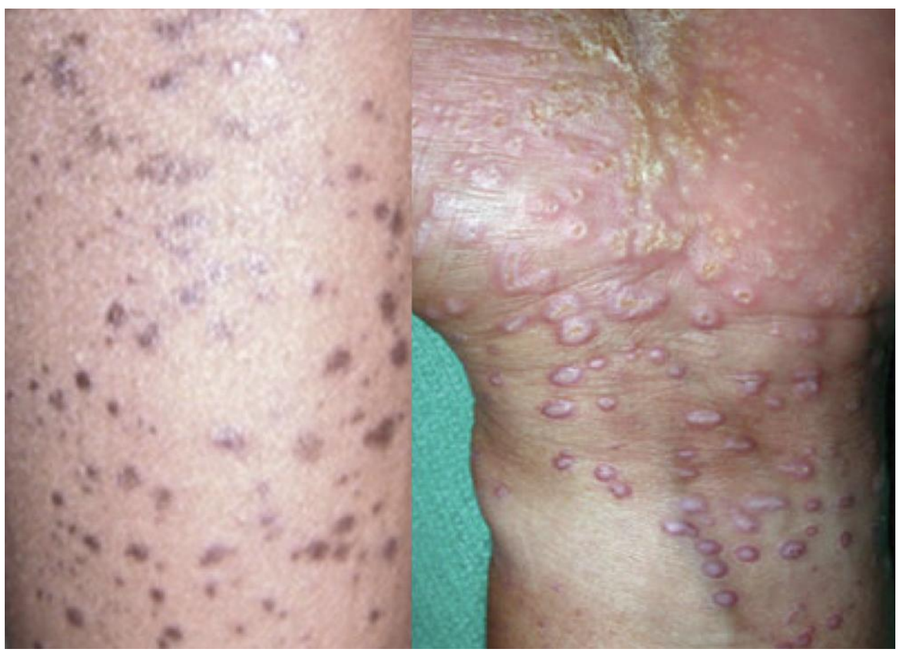
- Cutaneous Lichen planus affects less than 1% of population.
- Oral Lichen planus (1-5%).
- It usually affect adult (30-60 years) but it can rarely affect children.
- It doesn't have any gender/race predilection.
- Familial cases are underestimated (some about 10%)
- 75% of cutaneous Lichen planus have mucosal involvement sp. Oral
- 25% of Oral Lichen planus have cutaneous Lichen planus.Some expert consider them separate disease

❖ Antigenic Triggers:

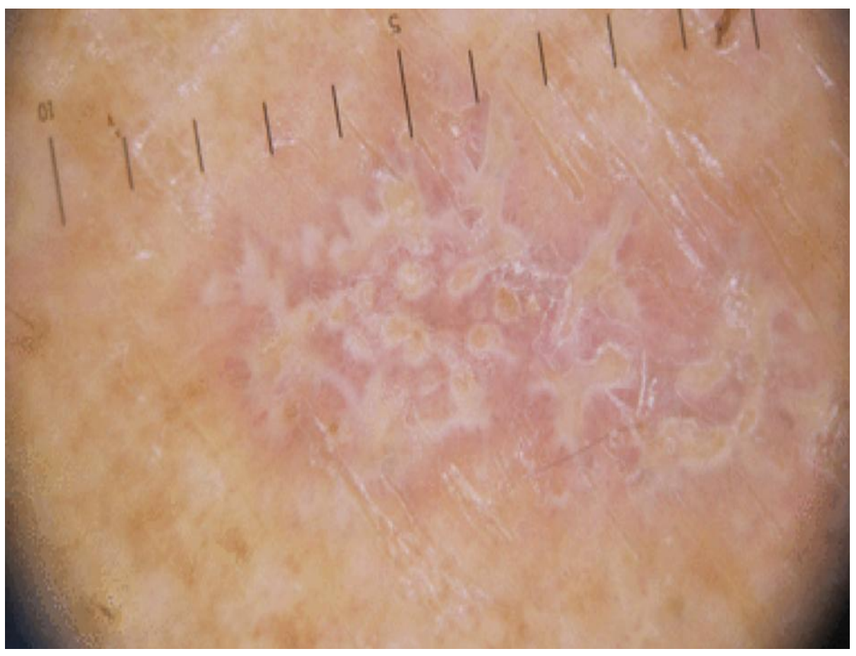
- Pathogens (Viral hepatitis-HCV) screen the patient for hepatitis C
- geographic variation
- IFN therapy initiate or worsen LP
- Liver disease e.g. sclerosing cholangitis chronic liver disease
- Vaccination
- Medication (Lichenoid drug eruption)(antimalarials and IFNs)
- Contact sensitizer e.g. mercury amalgam(changing it to ceramic will improve the disease), color film developers, methacrylic acid esters, dimethyl fumarate in sofas and radiotherapy.

❖ Clinical presentation:

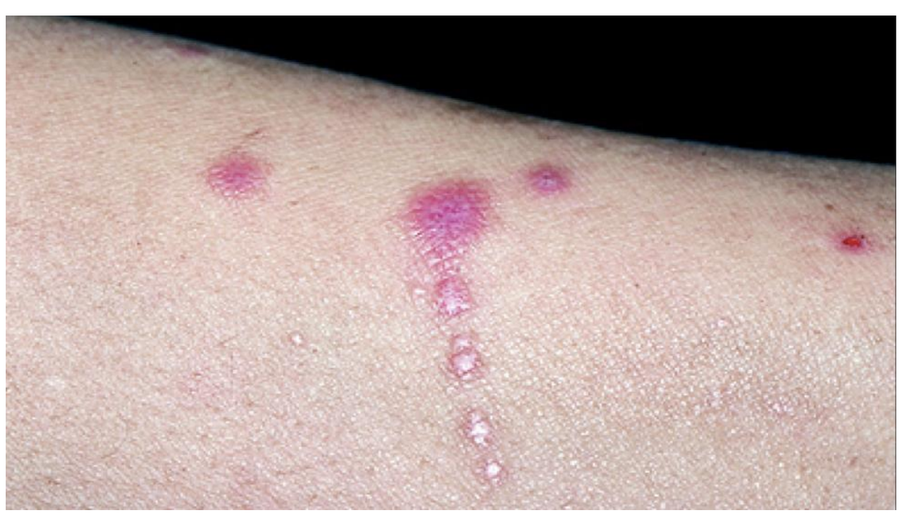
- The extremities, particularly the ankles and the volar surface of the wrists, are common sites for cutaneous involvement.
- Lesions heal with post inflammatory hyperpigmentation.
- Koebnerization is positive.
- Itchy (rubbing)not scratching pruritic polygonal purple flat-topped papule & plaques with Wickham's striae is pathognomic over flexural surface of extremities ,wrist, legs, lower abdomen and genitalia.



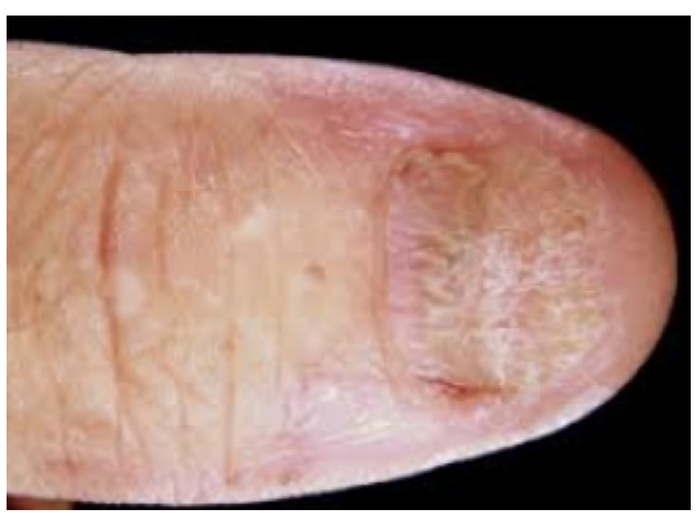
Flat topped polygonal violaceous papules



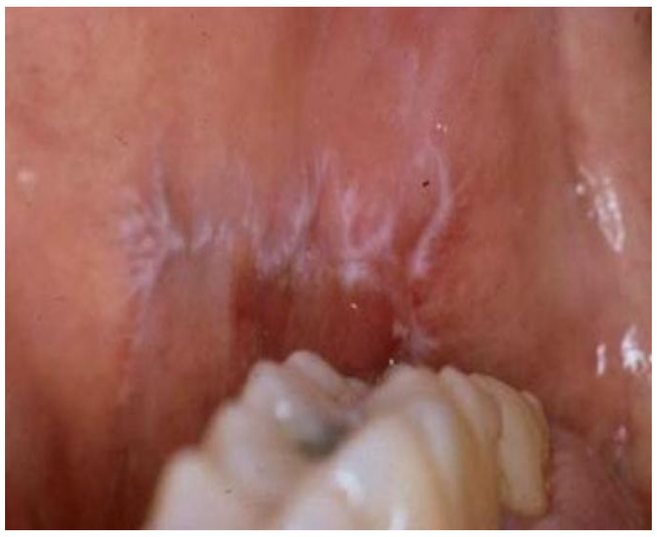
Dermoscopy showing Wickham's striae (WS)



Koebner phenomenon



Nail LP

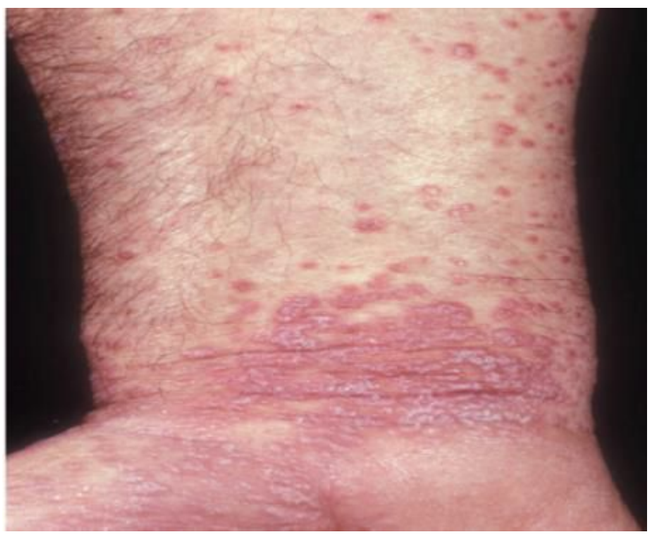


Oral LP

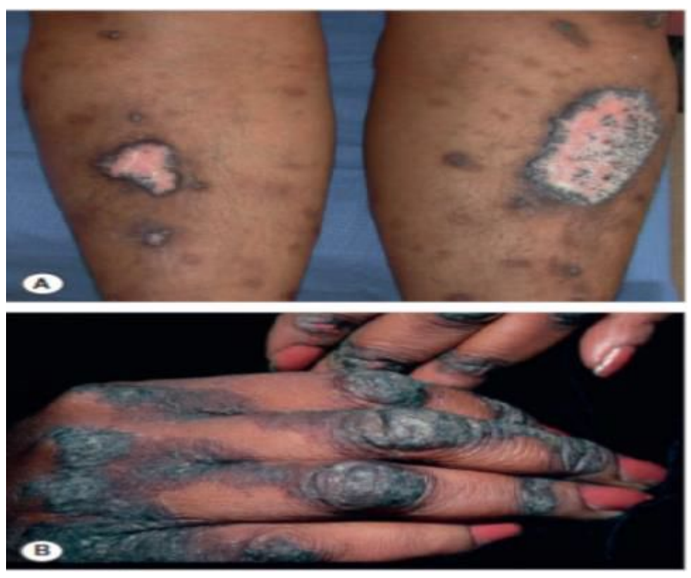
Variants:

By Morphology	By Configuration
Hypertrophic-leg thickening and scaling	Annular-penis few lesion and doesn't become lichen planus and involve other areas
Atrophic	Linear
Ulcerative-palm/soles ulceration	
Bullous	

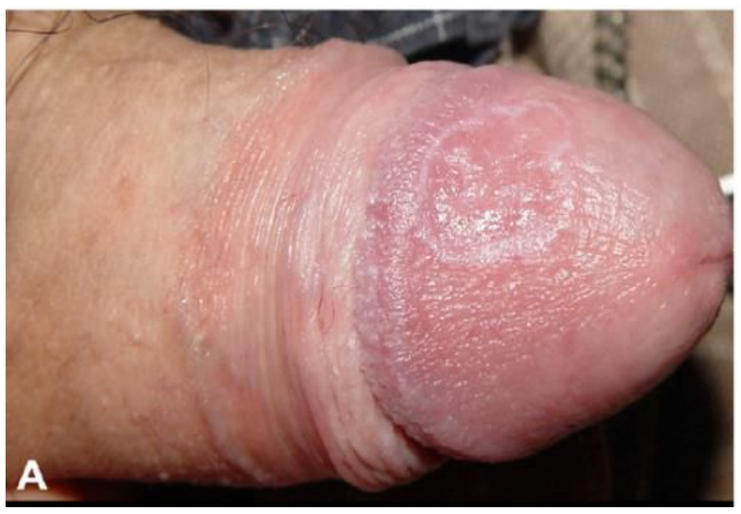
- Important types are hypertrophic, ulcerative, annular and pigmentosus
- Lichenoid morphology means flat topped



Pruritic flat topped violaceous plaques and papules in the wrist



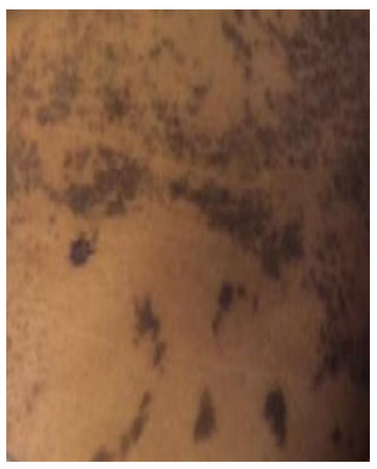
Lichen planus pigmentosus(generalized) and actinicus(sun exposed areas)
Well defined slate gray patches sometimes with violaceous rim or hypopigmented halo, but it's not in the picture



Annular Lichen planus of penis (never progress)

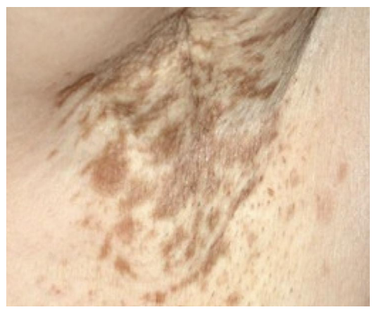
❖ Clinical Variants:

Lichen planus Pigmentosus:



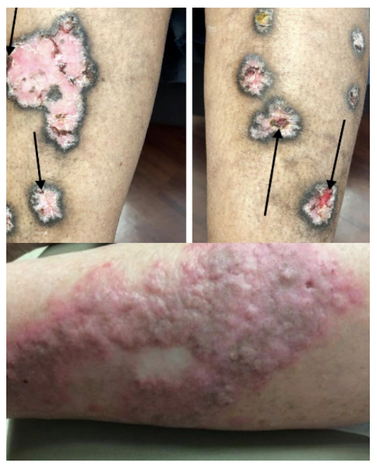
- Presents with gray-brown or dark brown macules or patches that are most commonly found in sun-exposed or flexural areas.
- Pruritus is minimal or absent.
- The term "lichen planus pigmentosus-inversus" is used to describe patients with primarily flexural involvement.
- It is more common in dark skin people.
- indurated border or hypopigmented halo over axilla/groin & proximal limbs.

Inverse lichen planus:



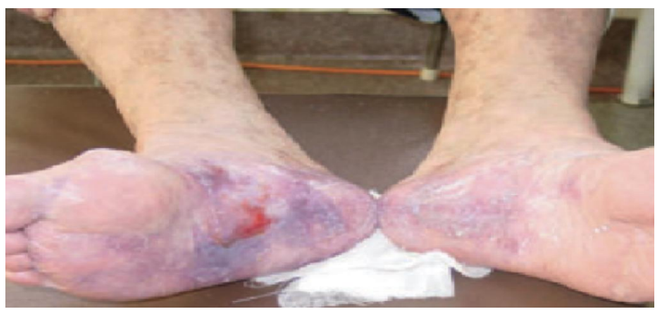
- Characterized by erythematous to violaceous papules and plaques in intertriginous sites, such as the axillae, inguinal creases, inframammary area, or limb flexures.
- Associated hyperpigmentation is common.

Hypertrophic lichen planus:



- Characterized intensely pruritic, flat-topped plaques.
- Involving typically the anterior lower legs.
- Cutaneous squamous cell carcinoma has been reported in patients with longstanding hypertrophic lichen planus lesions.

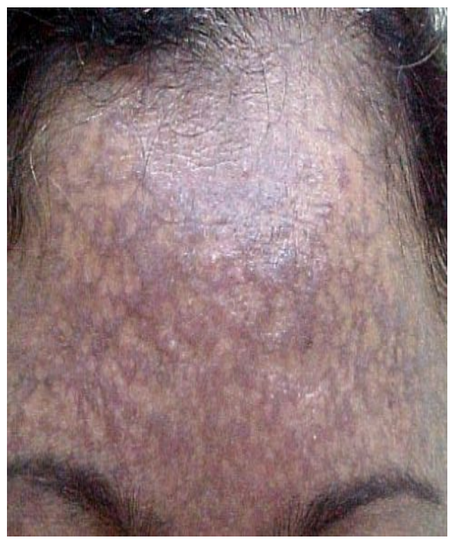
Palmoplantar lichen planus:



- A variant that may demonstrate ulceration.

Perforating lichen planus.

Lichen planus Actinicus:



(sun exposed area) same disease and presentation as Lichen planus Pigmentosus but named according to site of involvement. Known as lichen planus tropicus.

- Present with a photodistributed eruption of hyperpigmented macules, violaceous or
- annular papules, or plaques. - Photodistribution areas are the face, neck & upper trunk.
- It spears the eyelids, below the chin, and post auricular.

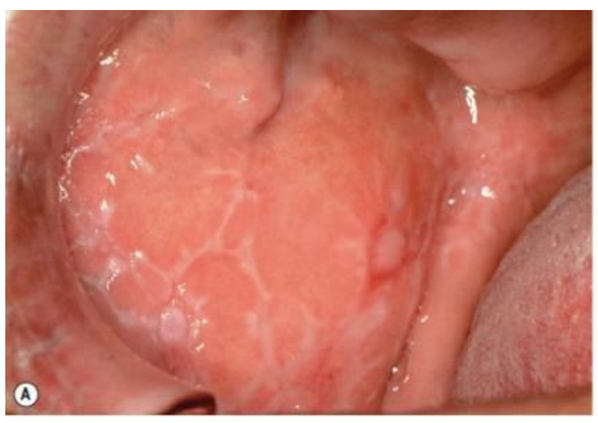
- Most commonly seen in the Middle East, India, and east Africa.

Mucosal Lichen planus:

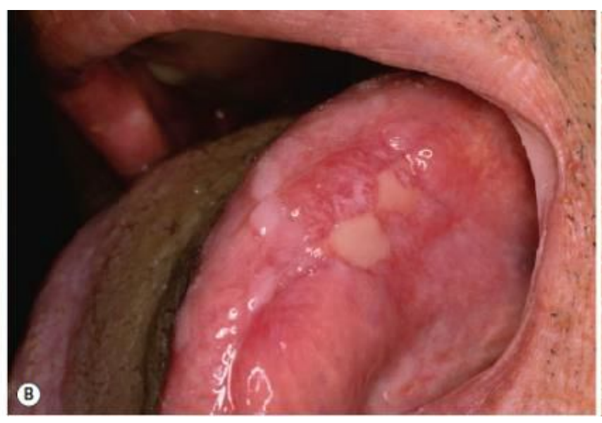
The most common site of involvement is the oral mucosa which usually present as well-defined reticulated violaceous plaques over buccal mucosa, lips and gingiva.

- Reticulated
- Erosive (less than 1% risk of SCC)
- Atrophic
- Hypertrophic

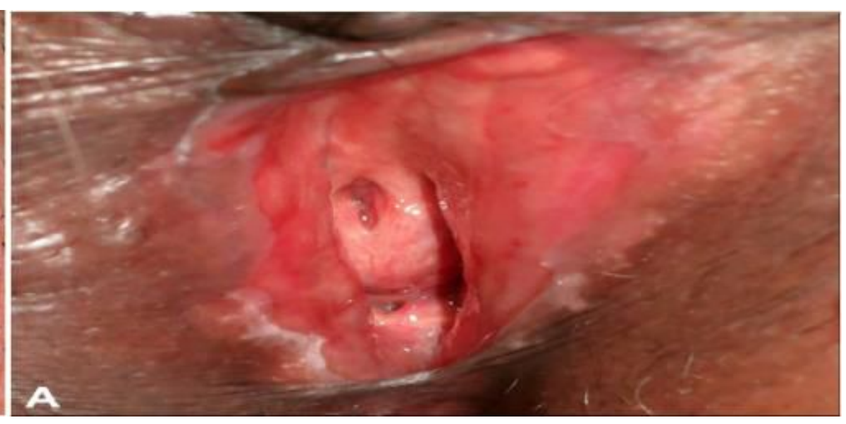
It can affect other mucosal surfaces e.g. vulvar, vaginal and penile- Vulvovaginogingival syndrome sever erosive



reticulated



erosive

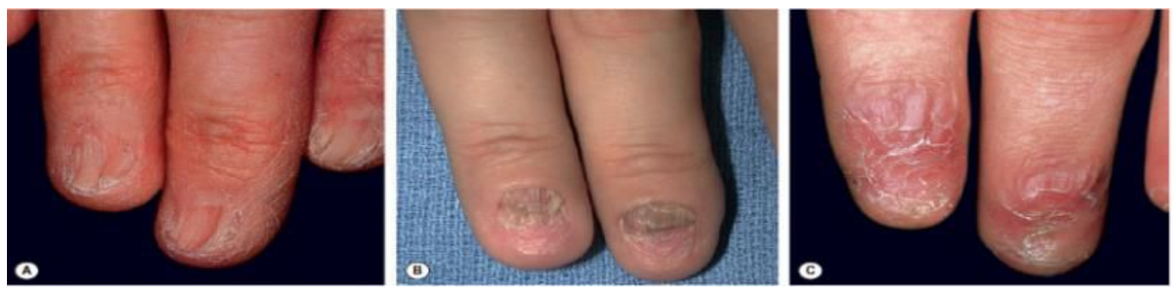


Erosive vulvovaginitis

Nail Lichen planus:

Nail involvment usually occur in 20% and it is more common in children

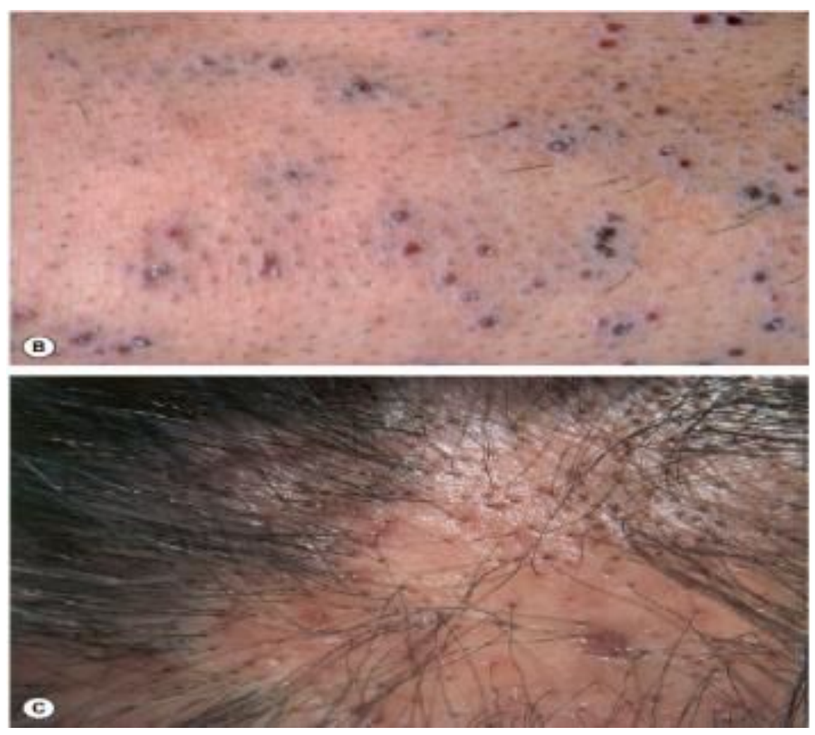
- Dorsal Pteryigum the adhesion of the hyponychium with the Proximal nail fold
- Lateral thinning
- Longitudinal ridging
- Fissuring
- Distal splitting the nail plate at the end will start to split
- thinning of the nail
- 20 Nail Dystrophy when all 20 nails are destroyed. Can be caused by other diseases



Lichen planopilaris-LPP(scarring):

The scalp is the classic site for lichen planopilaris. However, other body sites, particularly in patients with the Graham-Little-Piccardi-Lasseur syndrome (GLPL).

It usually present as multiple, keratotic plugs surrounded by a narrow violaceous rim are observed primarily on the scalp, although other hair bearing areas can also be affected. The inflammatory process usually result in scarring alopecia when it doesn't cause scarring it's called lichen spinulosus

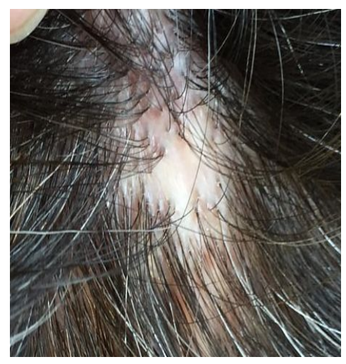


Lichen planopilaris
(Later causes scarring)

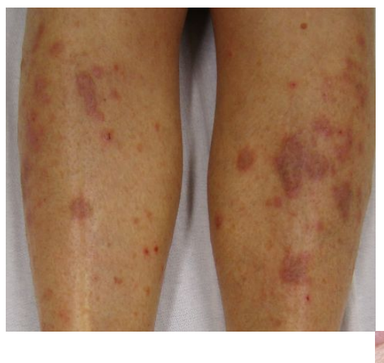
Graham-Little–Piccardi–Lassueur syndrome:

Is a Triad of:

Multifocal cicatricial alopecia of the scalp. Noncicatricial alopecia of axilla and groin. follicular lichen planus eruption on the body, scalp, or both.

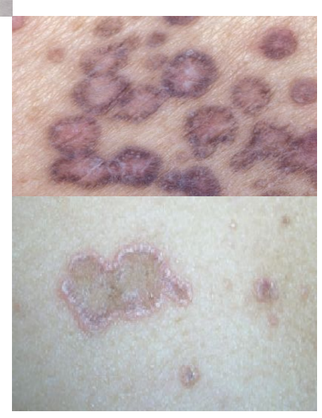


Atrophic lichen planus



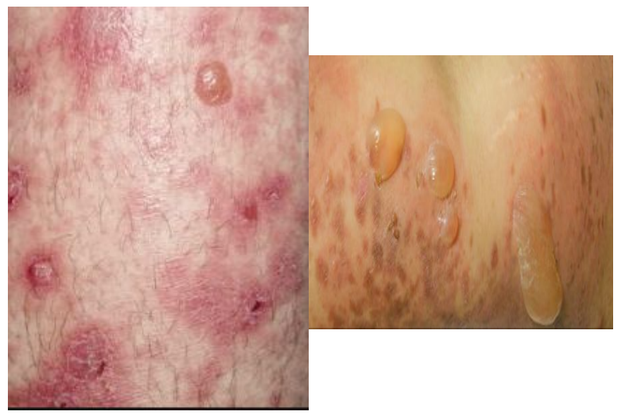
- Violaceous, round or oval, atrophic (depressed) plaques.
- The legs are a common site of involvement.

Annular lichen planus



- Characterized by the development of violaceous plaques with central clearing.
- The penis, scrotum, and intertriginous areas are common sites of involvement.
- Annular lesions may occur in other areas.
- Central atrophy may be present.

Bullous lichen planus



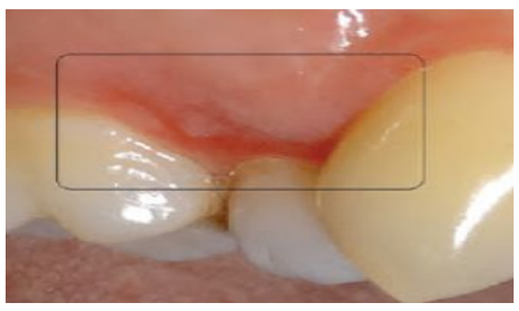
- Vesicles or bullae within the sites of existing cutaneous lichen planus lesions.

- Appear on diseased skin but NOT normal skin.

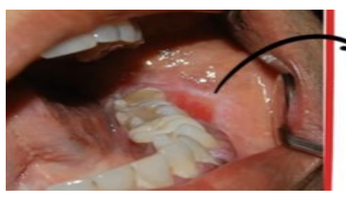
- The legs are a common site of lesion development.

Oral lichen

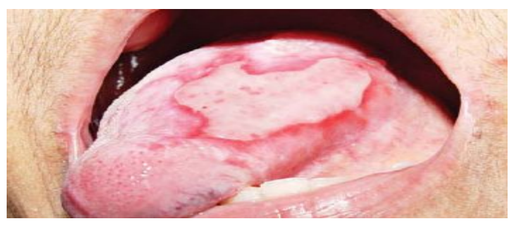
- Occur in conjunction with cutaneous disease or independently.



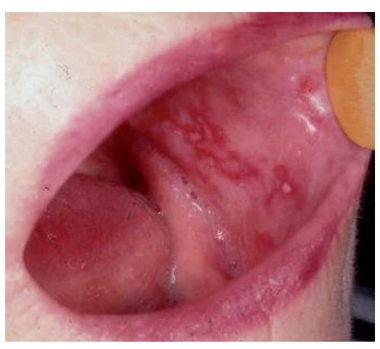
- Atrophic may lead to (desquamative gingevitis)



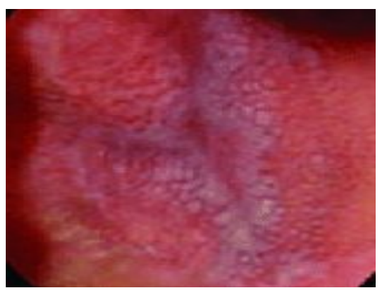
Atrophic erythematous



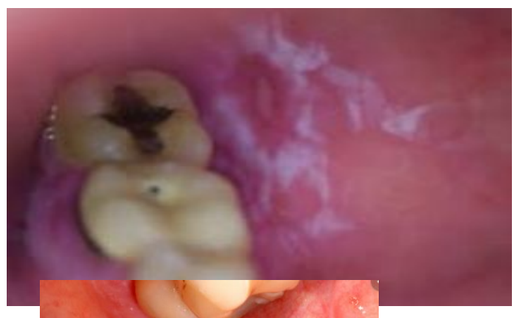
Erosive lesions

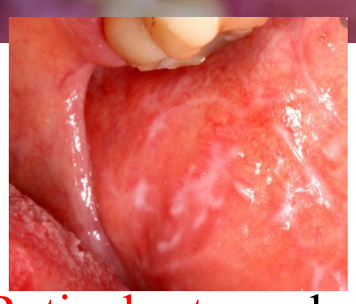


Ulcerative LP



Papular

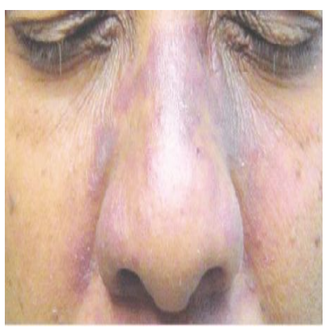




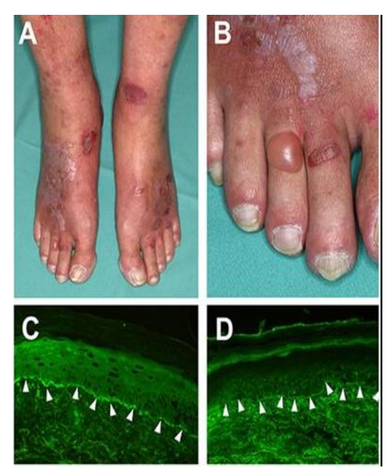
Reticular type; lacelike Wickham's striae /white patches that are particularly evident on the buccal mucosa.

Overlap syndromes:

- Are disorders that are characterized by the presence of features of cutaneous lichen planus and a second disease.
- Lichen planus pemphigoides and lichen planus-lupus erythematosus overlap syndromes.
  - **Lichen planus-lupus erythematosus overlap syndrome**
    - Rare condition, patients develop skin lesions with clinical, histologic, and/or immunopathologic features of both diseases.
    - Clinically, patients often present with discoid lupus erythematosus or malar erythema and cutaneous lichen planus. If you do serology the patient has SLE



  - **Lichen planus pemphigoides overlap syndrome**
    - Patients develop bullae in normal appearing skin and on top of lesions of lichen planus (abnormal lesion).
    - The presence of both the bullae & lichen planus lesions means it is NOT a pure lichen planus.
    - Similar to bullous pemphigoid, direct immunofluorescence studies of LPP demonstrate linear deposition of IgG and C3 at the dermoepidermal junction.
    - The natural history of most cases of cutaneous lichen planus is to remit within one to two years in up to two thirds of patients.
    - Oral, genital, scalp, and nail lichen planus tend to be more persistent.

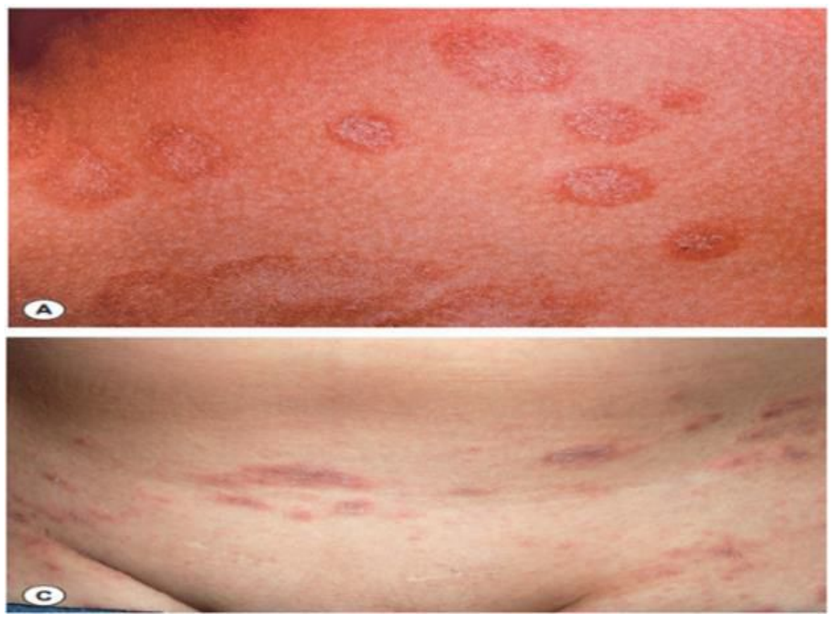


Complication of lichen planus: 1- squamous cell carcinoma in oral erosive LP. 2- scaring alopecia in lichen planopilaris. 3- post inflammatory hyperpigmentation.

Typical pityiasis rosea:

only 50% are typical Single well-defined oval (4cm) pink-erythematous patch with collarette scale over the trunk/proximal limbs (Herald patch 50%) when it's large it called PITYRIASIS ROSEA gigantea followed after average of 2 weeks with similar daughter lesions on the trunk /proximal limbs(christmas tree) with whole illness ranging between 6-12 weeks

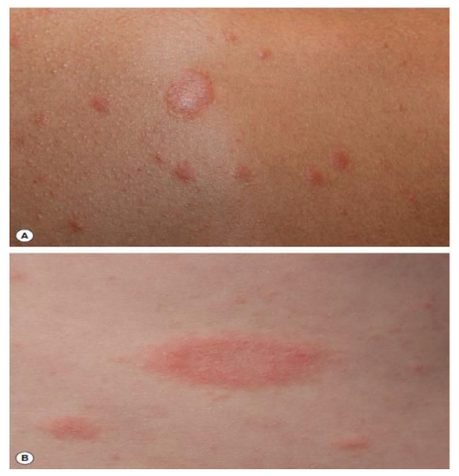
- 75% complain of pruritus
- 75% complain of Viral prodrome
- 10% oral lesions : erosion/ulcer (most common), purpura and erythematous patch



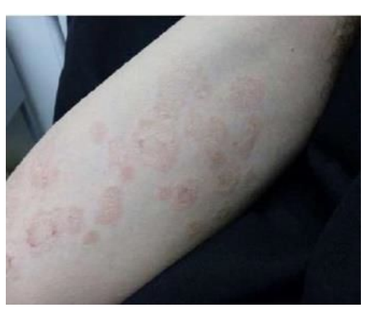
Oval pinkish collarette scales(pointing inwards)

Atypical forms of pityriasis rosea:

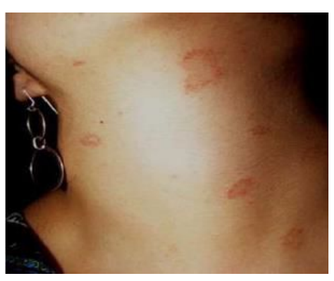
-Lacks herald patch, different morphology (purpuric, targetoid)
and different distribution (inversa over axilla, groin, distal extremities)
- papular is common in dark skin people and children vesicular



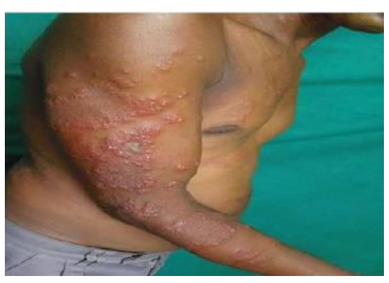
- Pityriasis rosea Inversa
- Involves the axillae and inguinal areas and sometimes the face.
- It is more common in younger children and in those with darkly pigmented skin.
- Urticarial.
- Erythema multiforme like (Targetoid).
- Vesicular.
- Pustular.
- Purpuric.



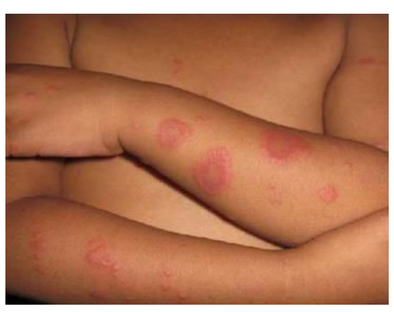
Urticarial



• Purpuric.



• Vesicular.

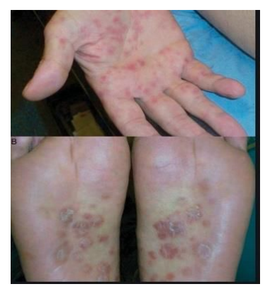


• Erythema multiforme like (Targetoid).

Diferential diagnosis:

• Secondary syphilis:

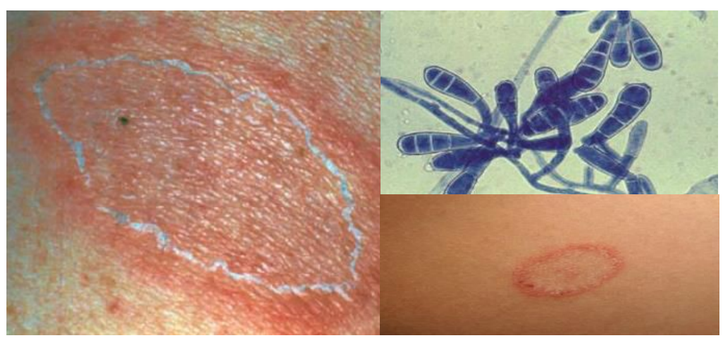
▪ Meticulous history taking, history of chancre, lymphadenopathy. ▪ Lesions are monomorphous and always asymptomatic; almost always affect palms and soles.
▪ Positive VDRL test.
▪ Histology showing plasma cells.



Condyloma lata

• Dermatophytosis:

▪ Difficult to differentiate from herald patch. However, a mycotic lesion expands progressively and shows a clear center (activity in the periphery).
▪ Positive KOH mount

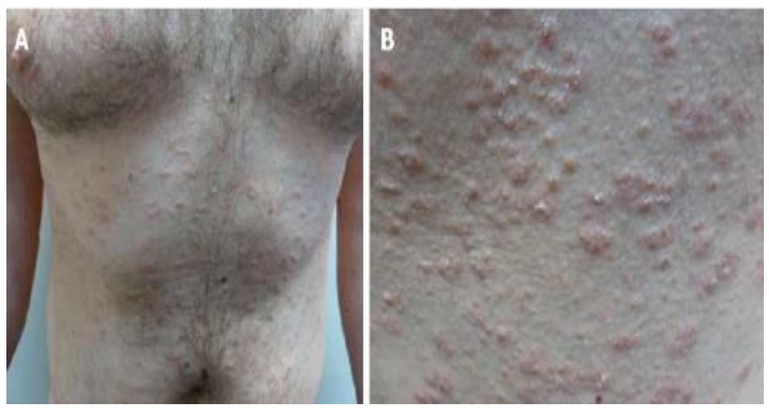


• Guttate psoriasis:

▪ History of sore throat.
▪ Presence of rain-drop pattern and histology are important clues.
▪ Scales are thicker and silvery-white..

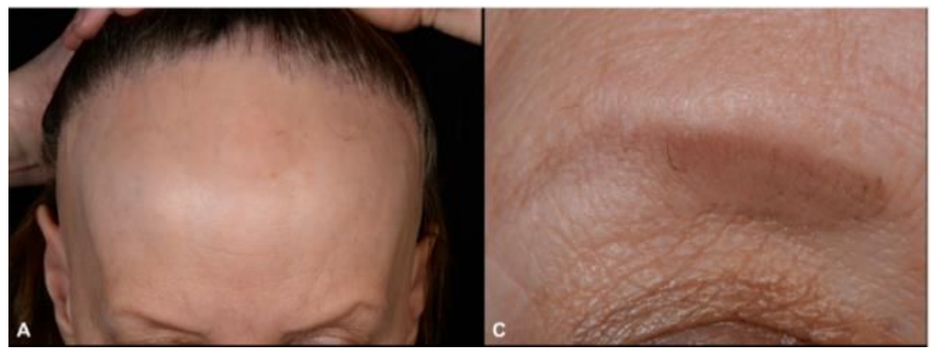
• PR like drug eruption:

An eruption that resemble PR with the following differences:
• lacking herald patch and viral prodrome
• Papular morphology
• Acrofacial distribution
• Histology : Superficial perivascular lymphocytic infiltrate with eosinophil and vacuolar interface reaction and necrotic keratinocytes within the epidermis
• HHV6/7 serology negative
▪ The lesions are usually monomorphic and lack herald patch. Slower to resolve than the idiopathic form.



Frontal fibrosing alopecia:

- It usually affect postmenopausal female
- It presents as frontal hairline scarring alopecia with eyebrows thinning



Lichnoid drug eruption: INFs and Antimalarials

It is a drug eruption that resemble Lichen planus with the following clinical differences

- Morphology: more psoriasiform/eczematous
- Distribution: start as photo-distributed then generalized
- Usually no mucosal involvement and Wickham's striae

It can appear anytime between weeks and years after exposure (commonly diuretics cause it)

• Lichenoid drug eruption (drug-induced lichen planus) should always be considered so that the offending agent can be withdrawn when possible.
- Always take history of medications in patients with lichen planus because it is difficult to differentiate between drug-induced & classic lichen planus except pathologically (there will be eosinophils in drug induced).
• The cutaneous manifestations closely resemble idiopathic lichen planus.
• The patient's history of drug exposure and a skin biopsy can aid in distinguishing lichenoid drug eruptions from idiopathic lichen planus.
• Lichenoid drug eruptions usually develop insidiously and can affect any area of the body surface.

FEATURES FOR DISTINGUISHING LICHENOID DRUG ERUPTION FROM LICHEN PLANUS

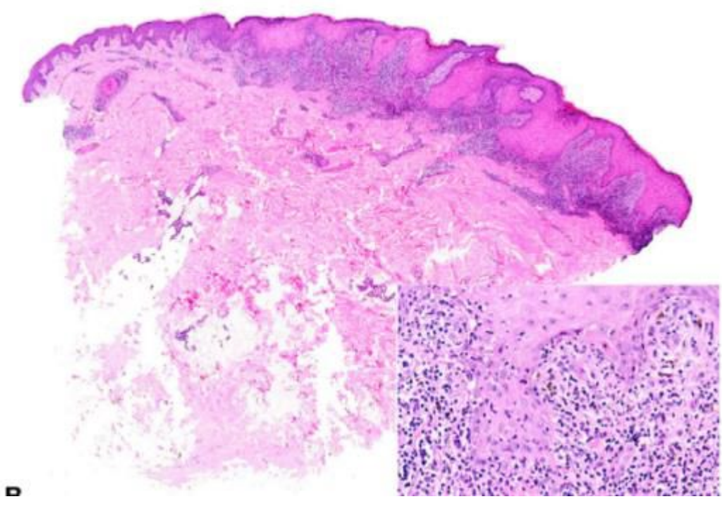
Feature	Lichenoid drug eruption	Idiopathic lichen planus
Mean age	65 years	50 years
Location	More generalized (including the trunk) and symmetric; often spares the "classic" sites of LP	Wrists, flexor forearms, presacral area, lower legs, genitalia
Morphology	More eczematous, psoriasiform or pityriasis rosea-like	Shiny, flat-topped, polygonal, violaceous papules
Wickham striae	Uncommon	Present
Hyperpigmentation	Very common, sometimes persistent	Common
Photodistribution	Frequent*	Unusual
Mucous membranes	Usually spared	Often involved
Histology	Varying degree of eosinophilic and/or plasma cell infiltrates	Eosinophils and plasma cells uncommon
	Deep perivascular infiltrate may be present (<50% of cases)	Dense band-like infiltrate of lymphocytes in the papillary dermis
	Focal parakeratosis and focal interruption of the granular layer	Parakeratosis uncommon
	Cytoid bodies in cornified, granular and upper spinous layers	Cytoid bodies in lower spinous layer

*Especially with medications such as hydrochlorothiazide.

Group of drug	
Antimicrobial substances	Aminosalicylate sodium, ethambutol, griseofulvin, ketoconazole, streptomycin, tetracycline, trovafloxacin, isoniazid
Antihistamines (H_2-blocker)	Ranitidine, roxatidine
Antihypertensives/antiarrhythmics	**ACE-inhibitors** (captopril, enalapril), doxazosin, **beta blockers** (propranolol, labetalol, sotalol), **methyldopa**, prazosin, nifedipine, **quinidine**
Antimalarial drugs	**Chloroquine**, **hydroxychloroquine**, quinine
Antidepressives/antianxiety drugs/antipsychotics/anticonvulsants	Amitriptyline, carbamazepine, chlorpromazine, levomepromazine, methopromazine, imipramine, lorazepam, phenytoin
Diuretics	**Thiazide diuretics** (chlorothiazide and hydrochlorothiazide), furosemide, spironolactone
Antidiabetics	Sulfonylureas (chlorpropamide, glimepiride, tolazamide, tolbutamide, glyburide)
Metals	**Gold salts**, arsenic, bismuth, mercury, palladium, lithium
Nonsteroidal-antiinflammatory drugs (NSAIDs)	Acetylsalicylic acid, benoxaprofen, diflunisal, fenclofenac, flurbiprofen, ibuprofen, indomethacin, naproxen, sulindac
Proton pump inhibitors	Omeprazole, lansoprazole, pantoprazole
Lipid lowering drugs	Pravastatin, simvastatin, gemfibrozil
Tumor necrosis factor-alpha antagonists	Infliximab, adalimumab, etanercept, lenercept

Pathology:

1- Classic Lichen planus pathology:
Superficial band like lymphocytic infiltrate with vacuolar interface reaction at the DEJ with dyskeratotic keratinocyte (Civatte bodies) & colloid bodies which is associated with wedge-shaped hypergranulosis (Wickham's striae) and sawtooth rete ridges "irregular acanthosis".
2- Lichenoid drug eruption:
The infiltrate is more deep with eosinophils/plasma cell with the presence of parakeratosis.



Lichen planus course:

It follow a relatively short course with relapse/remit nature that self-limit usually within 1-2 years except for oral and follicular LP which tend to be more chronic. Leave very bad pigmentation

Complication of lichen planus:

- Squamous cell carcinoma in oral erosive LP & hypertrophic LP.
- Scarring alopecia in lichen planopilaris.
- Post inflammatory hyperpigmentation.

Treatment:

- **Focal**

1) Topical corticosteroids
2) Intralesional corticosteroids -LPP

- **Generalized**

★ Phototherapy
1) NBUVB/PUVA
★ Systemic therapy
1) Systemic Corticosteroids if the disease is generalized steroids are the first line treatment
2) Systemic Retinoids e.g. Acitretin
3) Antimalaria- Hydroxychloroquine- LPP can treat it and cause
4) Immunosuppressive therapy- MTX
5) Cyclosporine

Pityriasis rosea

Epidemiology:

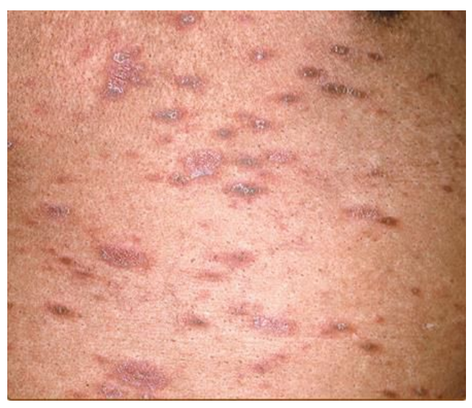
• Common, “acute”, self-limited (lasts for 2 months) papulosquamous eruption that favors healthy adolescents and young adults.It doesn't have any gender/race predilection.
• Distributed bilaterally over trunk and proximal extremities in christmas tree pattern.
- The long axis of the lesion follows the langer’s lines.
- Classical lesion: erythematous papules with Colarette scale.
• The classic presentation is readily recognized.
• Generally, it only appears once throughout life.
• Atypical forms may present a greater challenge.
• Persistent PR up to 6 months.
• Relapsing PR: 5%, mild shorter course, few attacks over 3-5 years.
• A viral etiology has been postulated, but this remains unproven.
• Seasonal variation. Although, PITYRIASIS ROSEA can be seen all year around, it is more frequent in winter, fall and spring.

pathogenesis:

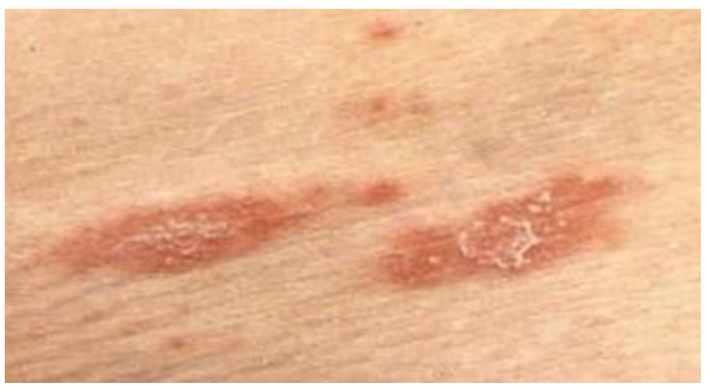
• The precise cause of pityriasis rosea unknown.
• A viral etiology is frequently proposed.
• Human herpesvirus-7(HHV-7) and, less so, on HHV-6.
• Attempts to identify HHV-7 DNA within cutaneous lesions have been unsuccessful.

Clinical presentation:

• Most patients with pityriasis rosea are asymptomatic.
• However, 25% of patients, pruritus ranges from mild to severe.
• Mild prodrome with headache, malaise, fever, arthralgia in 5% of patients.
• The classic situation, a solitary lesion “herald patch” appears on the trunk and enlarges over several days.
• Less often, this initial lesion is seen on the neck or proximal extremities.
• It predates the remainder of the eruption by hours to days.
• The herald patch incidence varies from 12% to 94%, but in most series, it has been seen in over 50% of cases.
• Multiple herald patches have also been reported.
• The herald patch is a skin- to pink- to salmon-colored patch or plaque with a slightly raised advancing margin.
• The size of herald patch varies from 2-4 cm, but it can be as small as 1 cm or large as 10 cm.
• Within the next few days, there is a blossoming of lesions on the trunk and proximal extremities.
• The lesions are usually round to oval papules or plaques, with their long axis following Langer’s lines of Cleavage, characteristic collarette scale.
• On the posterior trunk, the lesions show a “fir tree” or “Christmas tree "pattern.
• Minute pustules can also be seen during this initial phase of pityriasis rosea.
• The face, palms and soles are usually (but not always) spared.
• Oral lesions are uncommon.
• The eruption of pityriasis rosea usually persists for 6–8 weeks and then spontaneously resolves; however, occasional patients have lesions that may last 5 months or longer.
• In the latter situation, the possibility of pityriasis lichenoides chronica arises. Almost same presentation as pityriasis rosea but the duration differs.



Colarette scale



pityriasis lichenoides chronica

- **Medications causing PR drug eruption:**
  (ACE) inhibitors like captopril, metronidazole, isotretinoin, gold, arsenic, non-steroidal anti-inflammatory, terbinafin, omeprazole, bismuth, imatinib and clonidine, etanercept, tripelennamine, ketotifen, salvarsan, Barbiturates, methopromazine, D – penicillamine, levamisole, Pyribenzamine, ergotamine tartrate, tyrosine kinase inhibitors & Adalimumab

Vaccinations causing PR drug eruption:
diphtheria, smallpox, pneumococcal, hepatitis B virus, BCG and HPV

Course and duration:

• Persistent PITYRIASIS ROSEA duration longer that 12 weeks and usually up to 6 months with aggressive course and presentation.
• Relapsing PITYRIASIS ROSEA more than estimated (5%) but carry a mild and shorter course with few attacks over 3-5 years duration (usually due to the time the immune system take to gain full control over HHV6/7)

• Most patients with pityriasis rosea don't have biopsies because the clinical picture is characteristic, and the histopathology is relatively nonspecific.
• Small mounds of parakeratosis, spongiosis (epidermal edema), and a mild lymphohistiocytic perivascular and interstitial papillary dermal infiltrate.
• There may be mild erythrocyte extravasation.

PR and Pregnancy: not important

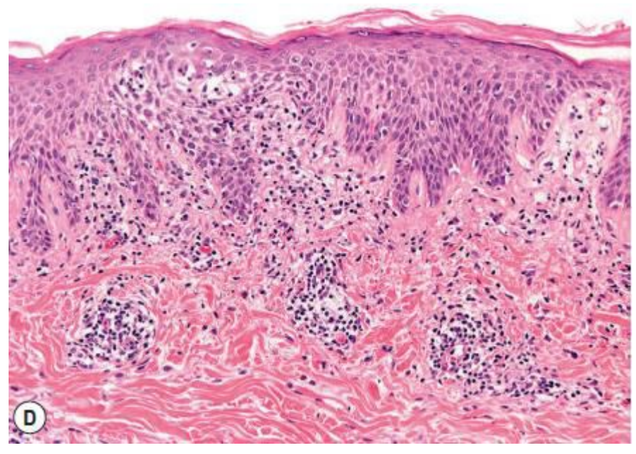
Since pregnancy is a state of altered immune response, a risk of viral reactivations and intrauterine transmission of HHV-6/7 exists.
On the whole, the total abortion rate among women with pregnancy PR is the same as that of the general population but noteworthy, when PR develops within the 15th Oval pinkish collarette scales(pointing inwards) gestational week, the abortion rate is higher probably because the risk of intrauterine transmission of HHV-6 (or less commonly HHV-7) is increased.

Histopathology of PR:

Most patients with pityriasis rosea don't have biopsied because the clinical picture is characteristic and the histopathology is relatively non specific

1. Small mounds of focal parakeratosis, spongiosis and acanthosis of the epidermis with mild superficial perivascular lymphohistiocytic and interstitial papillary dermal infiltrate. infiltrate
2. There may be mild erythrocyte extravasation

Spongiosis is water between the epidermal cells



Treatment:

• Because pityriasis rosea is often asymptomatic and self-limited, patient education and reassurance is sufficient.
• Symptomatic treatment with antihistamines and topical steroid.
• NBUVB.
• Acyclovir 400mg TDS x7 days. When to use?
  - Persistent PR.
  - Relapsing PR.
  - PR occurring in the 1st trimester of pregnancy.
• Short course oral steroids in severe cases.

Questions:

1- Which of the following is the primary lesion for lichen planus?

A. Papule
B. Nodule
C. Pustule
D. Macule

2- Pityriasis rosea is (associated) caused by reactivation of?

A. herpes 1
B. herpes 2
C. herpes 6
D. herpes 8

3- Most common nail change in lichen planus

A. Pitting
B. Oil drop
C. Longitudinal striations
D. Subungual hyperkeratosis

4- 30 years old male presents with multiple silvery white scaly plaques on his extensors. Which of the following is a common associated manifestation?

A. nephritis
B. Arthritis
C. Conjunctivitis
D. Interstitial pneumonitis

5- A skin lesion with a change in color and is more than 1 cm without elevation is called:

A. Papule
B. Macule
C. Plaque
D. Patch

6- which one type of psoriasis is considered serious or emergency?

A. Erythematous psoriasis
B. Pustular psoriasis
C. Psoriasis vulgaris
D. Guttate psoriasis

7-Which of the following organs is affected the most when using Cyclosporine as treatment for psoriasis?

A) Liver
B) Brain
C) Kidney
D) Heart

8- A 30 y/o male presented to you with multiple well-defined flat topped violaceous polygonal papules and plaques crossed with fine white lines over the trunk and extremities. What is your diagnosis?

a) Psoriasis
b) Lichen planus
c) Pityriasis rosea
d) Atopic dermatitis

9- Continued use of methotrexate will mostly likely cause damage to which of the following?

a) Liver
b) Kidneys
c) Joints

10- Elevated lesion less than 0.5cm what is the name of this lesion?

a) Macule
b) Patch
c) Papule
d) Plaque

11- What's the most common type of psoriasis

a) Plaque psoriasis
b) Erythrodermic Psoriasis
c) Pustular psoriasis
d) Plantopalmar Psoriasis

12- Pityriasis rosea is associated with which one of the following?

a) Herpes 1
b) Herpes 2
c) Herpes 7
d) Herpes 8

13- Which one of the following is present in nail lichen planus?

A- Splinter hemorrhage.
B- Oil drop.
C- Pterygium.
D- Clubbing.

14- Which one of the following is associated with lichen plans?

A- Hepatitis
C. B- HIV.
C- HPV.
D- HHV.

15- What is the clinical term that describes the presence of nucleated cell in the stratum corneum seen is psoriasis?

A- Parakeratosis.
B- Hyperkeratosis.
C- Acanthosis.

16- Old Patient who is in renal dialysis which one of the following is contraindication to give?

A- Cyclosporine.
B- Methotrexate.
C- Retinoid.

17- Erosive lichen planus has a risk of causing which of the following?

A- basal cell carcinoma
B- Squamous cell carcinoma

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
A	C	C	B	D	A	C	B	A	C	A	C	C	A	A	A	B