Dermatology Team 441

MED441
KING SAUD UNIVERSITY

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REVIEWED BY

Psoriasis & other papulosquamous disorders

- No objectives were found

Color index:

- Main text
- Important
- Dr's explanation
- Golden notes
- Extra

Dermatology Team 439

DERMATOLOGY TEAM 438

This lecture was originally done by both 438 & 439 teams.
So great thanks to them

Psoriasis

Introduction:

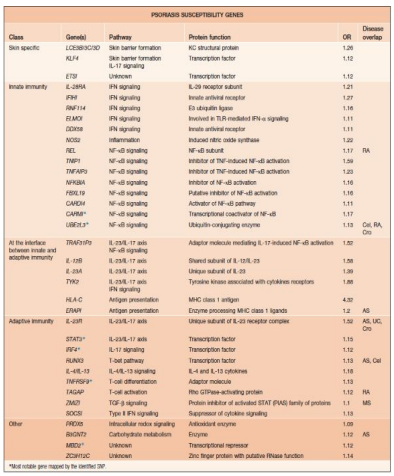
- Psoriasis is an **Complex** (Genetic + environmental factors) **immune-mediated polygenic** (Multiple genes involved) **skin disorder** (Multisystemic mainly involves the skin and joints). Various environmental triggering factors, e.g. trauma , stress, infections and medications, may elicit disease in genetically predisposed individuals.

Epidemiology:

- 2% of population (LP is less than 1%) (0.7% of Asian/African).
- 0.7% juvenile psoriasis.
- Only 25% have severe psoriasis.
- Bimodal disease (20-30s/50-60s).
- Two third of patients have family history of psoriasis (Strong family history in comparison to LP).
- Child risk : one parent 14% two parent 40%

Genetics: very imp

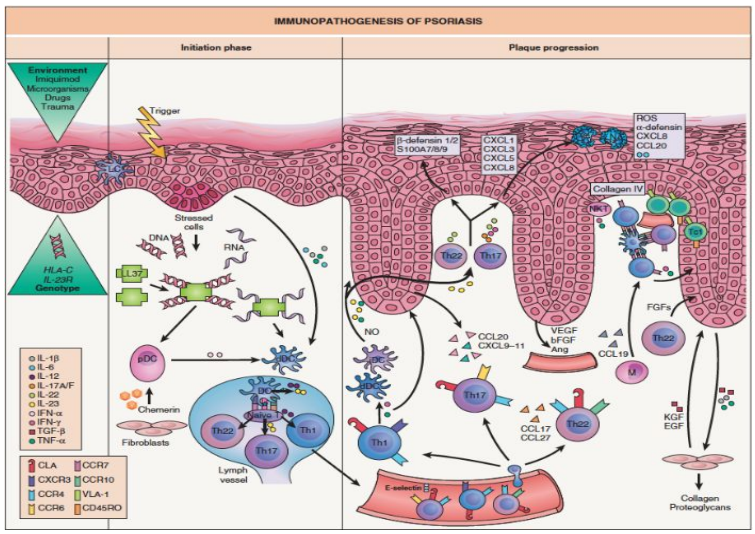
- Nine psoriasis susceptibility regions **(PSORS1–9)** in different chromosomal locations.
- **PSORS1** (on chromosome 6p), account for up to 50% of psoriasis risk.
- **PSORS1** contains genes such as **HLA-Cw6** which is important protein in immune system (from every two patients, one has this gene).
- **HLA-Cw6** is strongly linked to the age of onset of psoriasis.
  - 90% of the patients with early-onset psoriasis.
  - 50% of the patients with late-onset psoriasis.
- Early-onset psoriasis, positive family history and expression of HLA-Cw6 **(type I psoriasis).** (Aggressive type)
- Late-onset disease, no family history and a lack of expression of HLA-Cw6 **(type II psoriasis)**


PSORIASIS SUSCEPTIBILITY GENES

Class	Gene(s)	Pathway	Protein function	OR	Disease overlap
Skin specific	*LCE3B/3C/3D*	Skin barrier formation	KC structural protein	1.26	
	KLF4	Skin barrier formation IL-17 signaling	Transcription factor	1.12	
	ETS1	Unknown	Transcription factor	1.12	
Innate immunity	*IL-28RA*	IFN signaling	IL-29 receptor subunit	1.21	
	IFIH1	IFN signaling	Innate antiviral receptor	1.27	
	RNF114	IFN signaling	E3 ubiquitin ligase	1.16	
	ELMO1	IFN signaling	Involved in TLR-mediated IFN-α signaling	1.11	
	DDX58	IFN signaling	Innate antiviral receptor	1.11	
	NOS2	Inflammation	Induced nitric oxide synthase	1.22	
	REL	NF-κB signaling	NF-κB subunit	1.17	RA
	TNIP1	NF-κB signaling	Inhibitor of TNF-induced NF-κB activation	1.59	
	TNFAIP3	NF-κB signaling	Inhibitor of TNF-induced NF-κB activation	1.23	
	NFKBIA	NF-κB signaling	Inhibitor of NF-κB activation	1.16	
	FBXL19	NF-κB signaling	Putative inhibitor of NF-κB activation	1.16	
	CARD14	NF-κB signaling	Activator of NF-κB pathway	1.11	
	*CARM1**	NF-κB signaling	Transcriptional coactivator of NF-κB	1.17	
	*UBE2L3**	NF-κB signaling	Ubiquitin-conjugating enzyme	1.13	Cel, RA, Cro
At the interface between innate and adaptive immunity	*TRAF3IP2*	IL-25/IL-17 axis NF-κB signaling	Adaptor molecule mediating IL-17-induced NF-κB activation	1.52	
	IL-12B	IL-23/IL-17 axis	Shared subunit of IL-12/IL-23	1.58	
	IL-23A	IL-23/IL-17 axis	Unique subunit of IL-23	1.39	
	TYK2	IL-23/IL-17 axis IFN signaling	Tyrosine kinase associated with cytokines receptors	1.68	
	HLA-C	Antigen presentation	MHC class I antigen	4.32	
	ERAP1	Antigen presentation	Enzyme processing MHC class I ligands	1.2	AS
Adaptive immunity	*IL-23R*	IL-23/IL-17 axis	Unique subunit of IL-23 receptor complex	1.52	AS, UC, Cro
	*STAT3**	IL-23/IL-17 axis	Transcription factor	1.15	
	*IRF4**	IL-17 signaling	Transcription factor	1.12	
	RUNX3	T-cell pathway	Transcription factor	1.13	AS, Cel
	IL-4/IL-13	IL-4/IL-13 signaling	IL-4 and IL-13 cytokines	1.16	
	*TNFRSF9**	T-cell differentiation	Adaptor molecule	1.13	
	TAGAP	T-cell activation	Rho GTPase-activating protein	1.12	RA
	ZMIZ1	TGF-β signaling	Protein inhibitor of activated STAT (PIAS) family of proteins	1.1	MS
	SOCS1	Type II IFN signaling	Suppressor of cytokine signaling	1.13	
Other	*PRDX5*	Intracellular redox signaling	Antioxidant enzyme	1.09	
	BIN3NT2	Carbohydrate metabolism	Enzyme	1.12	AS
	*MEDI1**	Unknown	Transcriptional repressor	1.12	
	ZC3H12C	Unknown	Zinc finger protein with putative RNase function	1.14	

*Most notable gene mapped by the identified SNP.

Pathogenesis:



Color Index:
Pathogenesis
Pathology
Clinical feature

1- A Trigger (auto antigen or exogenous source) will cause protein changes in epidermis in susceptible people (who have defect in some genes) start to present some of its antigen to APC.
2- Antigen Presenting cells (langerhans cells and keratinocytes) in the skin will recognize the antigen and present it to the naive lymphocyte.
3- APC will make the T helper cell attack the antigen which is your own epidermis (Whenever this triggers come back again it will trigger the immune system continuously).
This will lead to superficial perivascular infiltrate.
4-Lymphocyte will secret cytokines and this will lead to vasodilation (erythema).
5- Too much cytokines will stimulate the epidermis and it will lead to Acanthosis (which is elongation of rete ridges in the papalis) (induration and Plaque).
6- Continuous stimulation of cytokines will accelerate the journey of Immature cells in basal layer to epidermis from 14 days to 3-4 days which will lead to Parakeratosis (the cells are not mature enough to become Corneocytes) (scale).

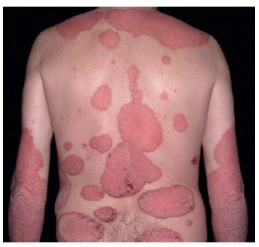
Psoriasis

Triggers:

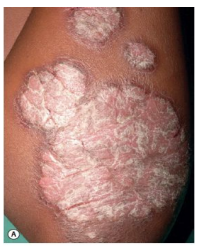
- Trauma
- Stress (emotional stress eg. alopecia areata/vitiligo)
- Infections: e.g. Streptococcal infections (group A), especially pharyngitis (most common infection to cause psoriasis).
- Drugs: e.g. lithium, IFNs, β-blockers, and antimalarial (IFNs and antimalarial drugs cause **lichen planus**).
- Hypocalcemia has been reported to be a triggering factor for generalized pustular psoriasis (happen in pregnancy).

1- Chronic Plaque Psoriasis: (~90%)

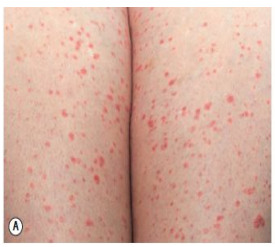
- Symmetric sharply defined erythematous plaques with thick silvery scale over the scalp, elbows, knees and lumbosacral area which is associated sometimes with hyperkeratosis of the hands and feet.
- Has these signs:
  - **Auspitz sign:** when removing a scale pinpoint bleeding occurs which represents the dilated capillaries.
  - Candle sign.
  - Woronoff sign: hypopigmented rim due to topical steroids use.
- **Types chronic plaque psoriasis:**
  - **Guttate psoriasis** looks like rain (**more common in children** and adolescents and preceded by an upper respiratory tract infection "strep") ; **REMEMBER IN CHILDREN**
  - **Flexural psoriasis** affects Axillae, groin and genital areas (humid areas masks the scales presented mistakenly as a resistant fungal infection> suspect psoriasis) and presents as well demarcated erythematous plaque without scales.
  - **sebopsoriasis:** indeterminate stage. (seborrheic dermatitis and psoriasis in the same patient)



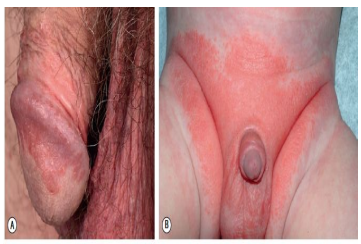
Classic Chronic Plaque type Psoriasis



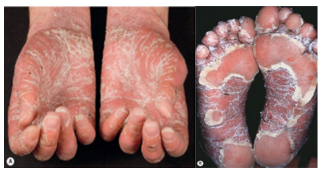
If scratched causes candle sign If peeled causes auspitz sign

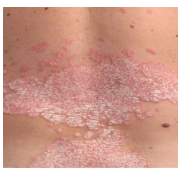


Guttate Psoriasis.



Flexural psoriasis, Notice there isn't any scaling

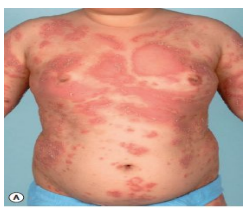




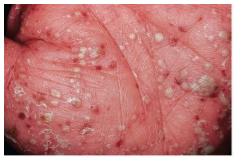
2- Pustular Psoriasis: (~9%)

- All are the same but named according to site of involvement.
- **Generalized pustular psoriasis** (von Zumbusch pattern):
  - ill patient with **constitutional symptoms** present with generalized abrupt painful eruption with erythema and **pustulation** starting over the intertriginous areas and trunk.
- **Palmoplantar pustulosis** (localized):
  - Associated with SAPHO syndrome (**S**ynovitis, **A**cne, **P**ustulosis, **H**yperostosis and **O**steitis).
- **Acrodermatitis continua of Hallopeau :**
  - Pustules over the distal portions of the fingers followed by scaling and crust formation. Pustules may also form subungual which **might cause shedding of nail plates.**

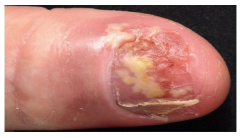
Pustular psoriasis. sheets of erythema with pustules



Palmoplantar pustulosis.



Acrodermatitis Continua of hallopeau (sama as Pustular Psoriasis but in the fingers) lead to anonychia.



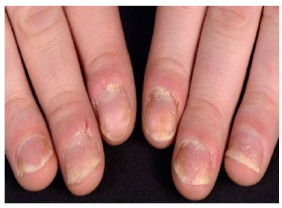
Psoriasis

3- Erythrodermic Psoriasis (EMERGENCY) (~1%). Confirm by skin biopsy

Involvement of >85-90% of body surface area with erythema>temp/electrolyte imbalance and risk of infection psoriasis is one of the pathologies that can develop into erythroderma

Nail: (more than ¾ of pt have nail involvement, ¼ of pt have joint involvement)

- Involved in 80% of patients (Thick and yellow nails).
- Patients with nail involvement appear to have an increased incidence of psoriatic arthritis.
  - **Pitting** (parakeratosis of the nail matrix).
  - **Oil-spot** (leukocytes beneath the nail plate).
  - **Onycholysis** (parakeratosis of the distal nail bed).



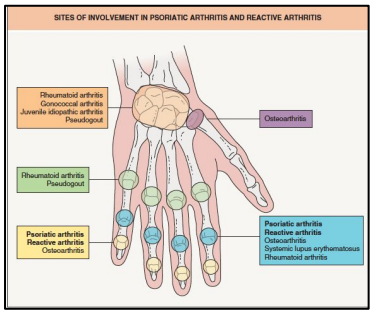
Pitting, Oil-spot, Onycholysis, Subungual hyperkeratosis and thickening of the nail

Oral mucosa: (not common in comparison to LP)

- Migratory annular erythematous lesions with hydrated white scale over the tongue **(geographic tongue)** observed mainly in pustular psoriasis patients.

Psoriatic Arthritis:

- 25% of patients
- Asymmetric distal mono-oligoarthritis (most common type) Inflammation of the DIP and PIP joints of the hands and feet (small joints). Involvement of both the DIP and PIP joints of a single digit can result in **"sausage" digit**.
- Least common presentations:
  - Rheumatoid arthritis-like:
    Symmetric polyarthritis that involves small and medium-sized joints.
  - Arthritis Mutilans (severe deformity in joints could cause shortening of digits).
  - Spondylitis and Sacroiliitis.

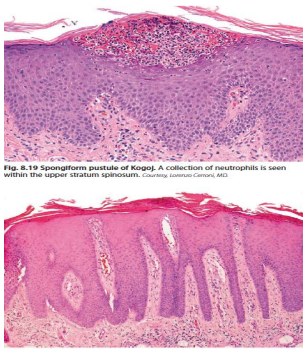


Associations: (psoriasis can increase risk of the following and vice versa)

- **Hyperlipidemia and metabolic syndrome.**
- **Diabetes Mellitus**
- **Atherosclerosis.**
- **Non-alcoholic steatohepatitis** we can't give methotrexate to Psoriatic arthritis patients because it might jeopardize the liver and cause further complications.
- **Depression and anxiety** that might lead to substance use.
- **Substance addiction.**
- **Parkinsonism.**

Pathology:

- 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 (Secondary phenomenon, not always happening), accumulation of neutrophils within a spongiotic pustule "spongiform pustule of Kogoj" or sub-corneal accumulation of neutrophils "microabscess of Munro" (exaggerated in pustular psoriasis).



Superficial perivascular infiltrate (lymphocyte), Dilated vessels and Elongation

Psoriasis

Treatment:

1. **Focal disease:**
   a. Topical corticosteroids.
   b. VitD3 analogues (calcipotriene). **The top treatment is Combination of vit D3 + Topical Corticosteroids. (Duobrii is a medication that combines steroid with vit d3 analogous)**
   c. Coal tar.
   d. Anthralin.
   e. Tazarotene.
   f. 10% salicylic acid.
   g. We apply peeling agents to increase the absorption of corticosteroids

2. **Widespread disease:**
   Systemic steroids are contraindicated for treating psoriasis because of conversion (turns chronic plaque type into pustular). can be used in pustular psoriasis of pregnancy for safety (biologic therapy is safer).

Widespread Disease Treatment	
Chronic plaque type	Pustular type
MTX First line	Retinoids First line
Anti-TNF	Cyclosporine
NBUVB/PUVA	MTX
Anti-IL 12/23	NBUVB/PUVA
Anti-IL 17A	
Cyclosporine "consider renal toxicity"	
Retinoids (acitretin) Kept in fat tissue for years; avoid it in child bearing age due to its teratogenicity	In contrast to isotretinoin (hydrophilic)which is cleared in a month

Lichen planus

Introduction:

- Lichen Planus is an Complex immune-mediated polygenic skin disorder. Various environmental triggering factors, e.g. Stress, infections and medications may elicit the disease in genetically predisposed individuals.

Epidemiology:

- Types of Lichen planus : we can have 2 types at the same time
  - Cutaneous: it goes away usually after 4-5 years
  - Cutaneous Lichen planus affects **less than 1% of population** (Rare).
    - 75% of cutaneous Lichen planus have mucosal involvement specifically Oral.
  - Mucosal: Oral (Mucosal) Lichen planus (1-5%) more common and it's more resistant and chronic (Dentist encounter these patients more).
    - 25% of Oral Lichen planus have cutaneous Lichen planus.
  - Follicular.
- 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%).
- **Some experts consider them as separate disease.**

Causes:

- Idiopathic complex polygenic condition.

Lichen planus

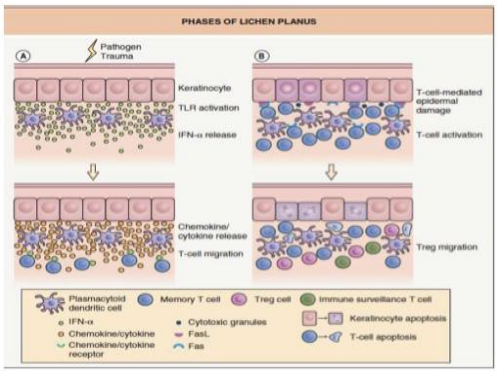
Genetic Predisposition: skipped by dr

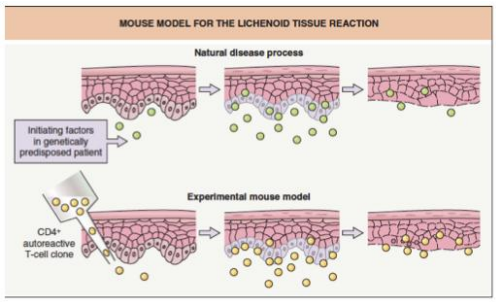
- Six single nucleotide polymorphisms (SNPs) were found to be associated the **HLA- DQB1**.
- 05:01 haplotype associated with Lichen planus.
- HLA-A5 ,HLA-A3,147,148, HLA-B7,143 HLA-DR1,149,150.
- HLA-DR10 in Arab population.
- **HLA-DRB1** 01:01 in Sardinien & Mexican population.

Antigenic Triggers:

- **Pathogens (Viral hepatitis-HCV)** (strongly associated mainly with cutaneous LP/order HCV panel).
- Geographic variation.
- **IFN therapy initiate or worsen LP.**
- Liver disease e.g. sclerosing cholangitis chronic liver disease.
- **Vaccination.**
- **Medication** (Lichenoid drug eruption e.g. antimalarials and IFNs).
- **Contact sensitizer** e.g. **mercury amalgam** حشوة الأسنان الزئبقية (it causes a lot of cases of LP, changing it to ceramic will improve the disease 50% will cure), color film developers, methacrylic acid esters, dimethyl fumarate in sofas and radiotherapy.

1- Superficial Perivascular infiltrate.
2- Sever inflammation cause thinning of epidermis (Sao tooth).
3- Retention of cells (immature).





An immune-mediated mechanism involving activated T cells (By the trigger), particularly CD8+ T cells (cytotoxic cells which will kill the keratinocytes), directed against basal keratinocytes has been proposed (In Psoriasis T helper CD4+ cell will be activated and it will cause inflammation). 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.

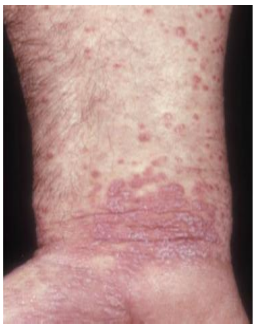
Clinical presentation: (5 Ps)

- Itchy (rubbing) not scratching **P**ruritic **P**olygonal **P**urple flat-topped (**plane**) **P**apule & **P**laques with **Wickham's striae** (Pathognomonic (wedge shaped hypergranulosis) it is a whitish lines visible in the papules of LP) over flexural surface of extremities ,wrist, legs, lower abdomen and genitalia.
- Pruritic well defined flat topped violaceous plaques and papules.

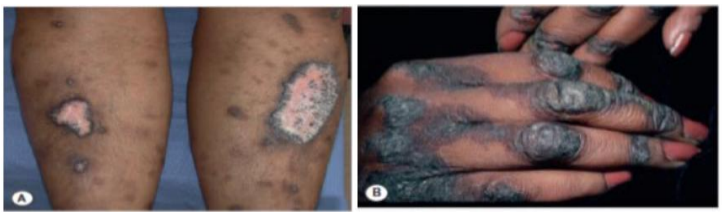
Variants: It's a littel advanced for you

By morphology	By configuration
Hypertrophic-leg	Annular-penis
Atrophic	Liner
Ulcerative-palm/soles	
Ballous	

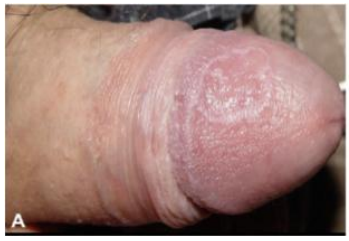
Lichen planus



Pruritic flat topped violaceous plaques and papules in the wrist.



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

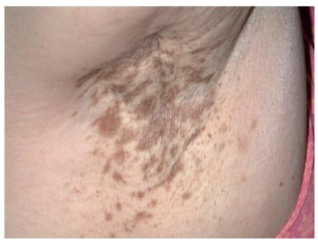


Annular Lichen planus of penis (never progress).
More common gentalia involvement in comparison to Psoriasis.

Types of LP: (by Presentation)

1- Lichen planus Pigmentosus:

- Not the same clinical features. Pigmentation is the prominent feature
- It is more common in dark skin people and it presents as **well- defined brown to slate-gray patches** with/without **violaceous** indurated border or hypopigmented halo over **axilla/groin** & proximal limbs (may come on the face).



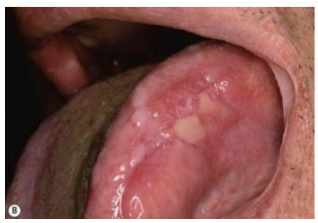
Pigmentosus (no rim neither halo).

2- Lichen planus actinicus: same as #1, but when it only affect the sun exposed area

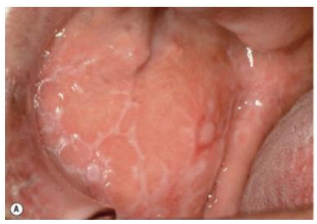
- Sun exposed area (same disease and presentation as Lichen planus Pigmentosus but named according to site of involvement).

3- Mucosal Lichen planus: more common variant

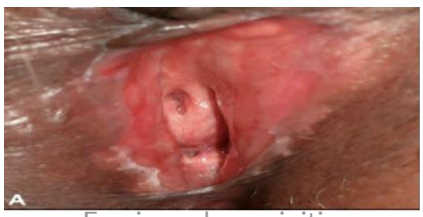
- The most common site of involvement is the oral mucosa which usually present as **well-defined reticulated violaceous plaques over buccal mucosa, lips and gingiva** (oral involvement is rare).
- **Varintes:**
  - **Reticulated.**
  - **Erosive (less than 1% risk of SCC)** If there is an oral ulcer you should take biopsy. This risk increased by smoking
  - **Atrophic.**
  - **Hypertrophic.**
- very itchy (genitalia+anal area) It can affect other mucosal surfaces e.g. vulvar, vaginal and penile- Vulvovaginogingival syndrome sever erosive.



reticulated



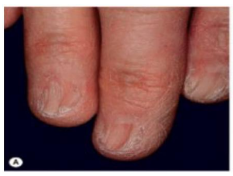
erosive

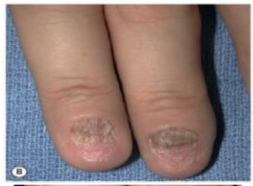


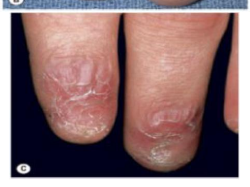
Erosive vulvovaginitis.

4- Nail Lichen planus: (Cutaneous)

- Nail involvement usually occur in 20% and it is more common in children
  - **Dorsal Pterygium** (Very specific to LP, but not pathognomonic) it happened when hyponychium attached to the Proximal nail fold.
  - **Lateral thinning.**
  - **Longitudinal ridging.**
  - **Distal splitting.**
  - Thinning of the nail.
  - 20 Nail Dystrophy when all 20 nails are destroyed. Can be caused by other diseases.





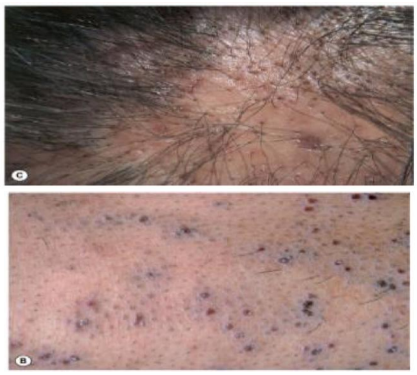


Lichen planus

Types of LP (cont'):

5- Lichen Planopilaris-LPP: (Follicular, scarring alopecia)

- It usually present as multiple, keratotic plugs surrounded by a narrow ill defined violaceous rim are observed primarily on the scalp around follicles, 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.



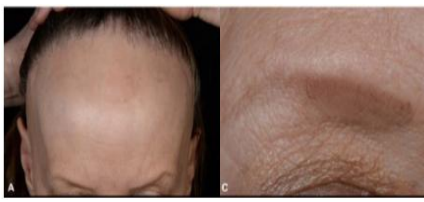
Lichen planopilaris Perifollicular
(Later causes scarring)

6- Graham-little-piccardi-lassueur syndrome:

- **Is a Triad of:**
  a. Non-scarring alopecia of pubic and axillary hairs and disseminated spinous or acuminated follicular papules.
  b. Typical cutaneous or mucosal LP.
  c. Scarring alopecia of the scalp.

7- Frontal fibrosing alopecia: (it was called Postmenposal frontal fibrosing alopecia)

- It usually affect **postmenopausal** female. >50 yo
- **It presents as frontal hairline scarring alopecia with eyebrows thinning.**



Frontal fibrosing alopecia
2ed Pic: Thinning of eyebrows

8- Lichenoid drug eruption:

- It is a drug eruption that resemble Lichen planus with the **following clinical differences:** imp
  - **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).

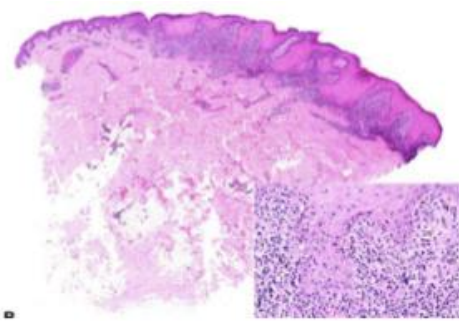
Pathology:

- **Classic Lichen planus pathology:**

  Superficial 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 "saw- tooth" rete ridges.**

  There's parakeratosis like psoriasis but (absence of microabscess and collection of neutrophils within epidermis)

  If shaggy band of fibrogen along with DEJ and colloid bodies staining with immunoglobulins at the papillary dermis



- **Lichenoid drug eruption:** The infiltrate is more deep with **eosinophils**/plasma cell with the presence of **parakeratosis** (which is Not in LP).

Lichen planus course:

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

Lichen planus

Treatment:

- **Focal:**
  - topical corticosteroids.
  - Intralesional corticosteroids -LPP.
- **Generalized**:
  - Systemic therapy:
    - Systemic Corticosteroids The first line of therapy for generalized.
    - Steroid sparing agents:
      - Systemic Retinoids e.g. **Acitretin** (avoid in female childbearing age)
      - Anti Malaria- Hydroxychloroquine- LPP.
      - Immunosuppressive therapy- MTX, Cyclosporine.
  - Phototherapy: not commonly used
    - NBUVB/PUVA

Pityriasis Rosea النخالة الوردية

Epidemiology :

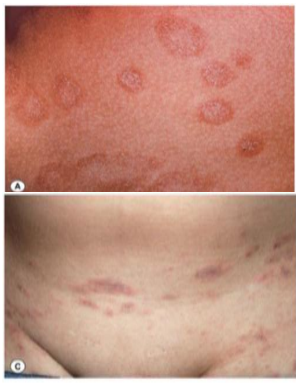
- Pityriasis Rosea is a **common acute self-limiting** (Viral) **exanthematous** eruption that usually affect adolescent (10-30 years) (Presented usually with prodrome). (Ones in your life)
- It doesn't have any gender/race predilection.
- Although, PR can be seen all year around, it is more frequent in winter, fall and spring.

Etiology :

- The etiology of PR is unknown but most experts believe that the **reactivation of HHV 6/7** play a significant role in the pathogenesis of the disease.

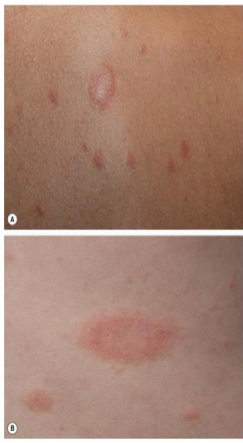
Typical pityriasis rosea: (Common)

- Single **well-defined** **oval** (4cm) **pink-erythematous patch** (or thin plaque) **with** **collarette scale** over the **trunk**/proximal limbs (**Herald patch** 50%). **(mother lesion) /common clinically diagnosed**
- followed **after average of 2 weeks** with similar **daughter lesions** on the **trunk**/proximal limbs satellite linear papules called **(christmas tree)** with whole illness ranging between **6-12 weeks**.
- The Scale in Psoriasis is **thick** whereas in PR is **thin**.
- 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 Pityriasis rosea: (50% present with this type)



- Herald Patch: mother patch
  - No HP.
  - Multiple HP.
  - **When we don't see clear herald patch we have to rule out secondary syphilis**
- Morphology
  - **papular** common in dark skin people and children **vesicular**.
  - Some PR have purpura or Targetoid lesion.
- Distribution
  - PR **Inversa** **"within skin folds"**affect axilla, groin and distal extremities sparing trunk

Pityriasis rosea النخالة الوردية

Course and duration:

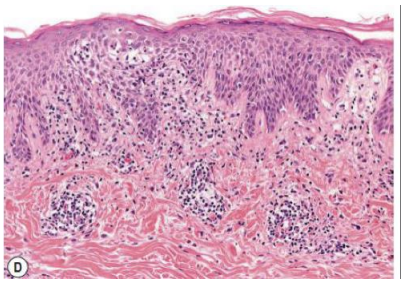
- **Persistent PR:** duration longer that 12 weeks and usually up to 6 months with aggressive course and presentation.
- **Relapsing PR: more than estimated (~5%) but carry a mild and shorter course with few attacks over 3-5 years duration (usually due to the time the immune system take to gain full control over HHV 6/7)**
- **Shall we look for HHV 6/7? we don't why would we? 1- it's not always there 2- it's a clinical diagnosis if in doubt do a biopsy to rule out other diseases.**

PR & Pregnancy: SKIPPED BY DR

- 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 gestational week, the abortion rate is higher probably because the risk of intrauterine transmission of HHV-6 (or less commonly HHV-7) is increased. Doesnt affect the course of the pregnancy

Histopathology:

- **focal parakeratosis** (it's focal so that's why we have scalcing only on the edges, not like psoriasis where we have homogenous), spongiosis and acanthosis of the epidermis with superficial perivascular lymphohistiocytic infiltrate accompanied by some extravasated RBC.



PR-like eruption: SKIPPED BY DR

- 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.**

Triggers:

- **Gold can lead to psoriasis**
- **Medications:**
  - Barbiturates, methopromazine, captopril, clonidine, **gold**, metronidazole, **D–penicillamine,** isotretinoin, levamisole, Pyribenzamine, NSAID, omeprazole, **terbinafine**, ergotamine tartrate, tyrosine kinase inhibitors & **Adalimumab**.
- **Vaccination:**
  - diphtheria, smallpox, pneumococcal, hepatitis B virus, BCG and HPV.

Treatment of PR:

- Symptomatic with **topical corticosteroids** and antihistamine (if there is Pruritus).
- Antiviral treatment (Acycolvir 800mg 5 times for 10 days) indicated in the following settings: (rarely used)
  - **Persistent PR.**
  - **Relapsing PR.**
  - Severe PR at the first trimester of pregnancy.

Quiz!

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- 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

4- 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

5- What's the most common type of psoriasis?

A) Plaque psoriasis
B) Erythrodermic Psoriasis
C) Pustular psoriasis
D) Plantopalmar Psoriasis

Answers:
1 : A, 2 : C, 3 : B, 4 : B, 5 : A

Thanks!!

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