



433 Teams

DERMATOLOGY

Lecture (4+5)

Psoriasis and other Papulosquamous disorders

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1-Papulosquamous diseases(Psoriasis)

Objectives:

- 1- **Define** the papulosquamous disease.
- 2- Highlight on **the pathogenesis** of papulosquamous diseases.
- 3- Discuss the **clinical features** of papulosquamous diseases.
- 4- Highlight on the papulosquamous diseases **treatment**.

Papulosquamous disease:

The term squamous refers to scaling that represents thick stratum corneum and thus implies an abnormal keratinization process.

Papulosquamous diseases are typically characterized by **scaly papules**.

Papulosquamous diseases:

- Psoriasis
- Pityriasis rosea
- Lichen planus
- Seborrheic dermatitis
- Pityriasis rubra pilaris
- Secondary syphilis
- Miscellaneous mycosis fungoides, discoid lupus erythematosus, ichthyoses.

1-Psoriasis

Definition: is a common, chronic and **non-infectious** disease. It is a systemic complex disease. Primarily affects skin and joints. It may be a **risk factor** for **metabolic syndrome** and its components (abdominal obesity, insulin resistance, hypertension and dyslipidemia, as well as an independent risk factor for myocardial infarction). It causes rapid skin cell reproduction resulting in **red, dry patches** of thickened skin. **They have less infection rate than other normal people.**



Well defined regular brownish to reddish scaly papule

Crust: dried fluid regardless the type of fluids, **scales:** dead keratocytes

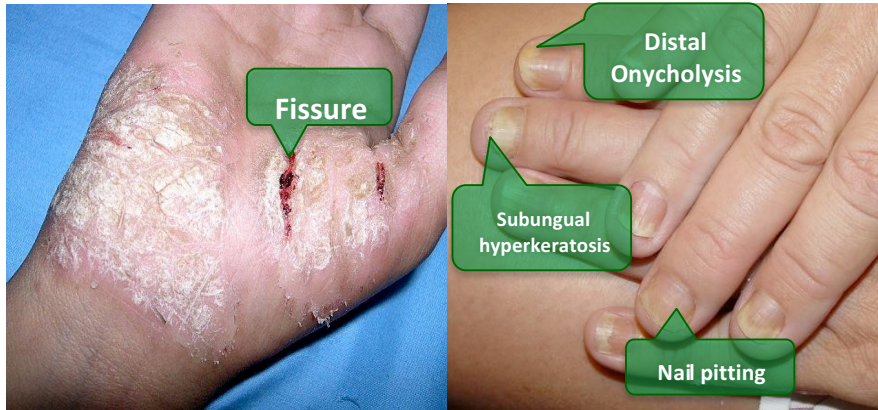
- The treatment improves some of the clinical presentation with no 100 %
- It can also affect the eye, GI & Liver but with lower incidence compared to the Skin & Joints



Bilateral, symmetrical, well-defined, regular, erythematous, scaly, plaques on elbows and knees.



Well-defined, generalized, dull red, scaly, papules and plaques on the back.



Fissures are an additional feature of the Psoriasis in the Palms & Soles. It develops because the palms and soles already have a thick skin, when Psoriasis occurs in it (in a thick skin) the scales accumulate; and with recurrent mechanical movements of the hands or soles the fissures develop!

- The thickest skin is in the Palms & Soles while the thinnest is in the eyelids.
- **Distal Onycholysis** is the separation of the distal nail plate from the nail bed.
- **Subungual hyperkeratosis** is scales under the nail plate.

✚ Epidemiology:

- The disease prevalence remains a questionable: **2% - 4%** in adult and 0.5–1% of children. **It is underestimated because of misdiagnosis.**
- The onset: any age, but two peaks were observed: around 20–30 and over 50 years of age.
- Pediatric psoriasis: up to 30% of all cases.
- Race: any race but higher prevalence in western European and Scandinavian populations
- Low risk in Asians and Africans.
- **75% has nail changes**
- 30 % of patients with Pso will develop PsA
- 75 % of PsA: the skin disease precedes arthritis, while in 15 % of patients Pso appears after PsA and in 10 % the cutaneous and articular involvement are simultaneous

✚ Pathogenesis:

It is considered to be an **autoimmune disease**.

Q: Who is the **pathogenic driver** in psoriasis? keratinocyte cells **or** T lymphocyte cells? **keratinocyte**.

❖ Genetic factor:

There are two types:-

1-type I psoriasis(early onset): more likely to be familial, have a severe clinical course and is associated **with HLA-Cw6**, B13 and B57.

2-type II psoriasis(Late onset): ages 50 to 60 and is correlated with **HLA-Cw2** and B27.

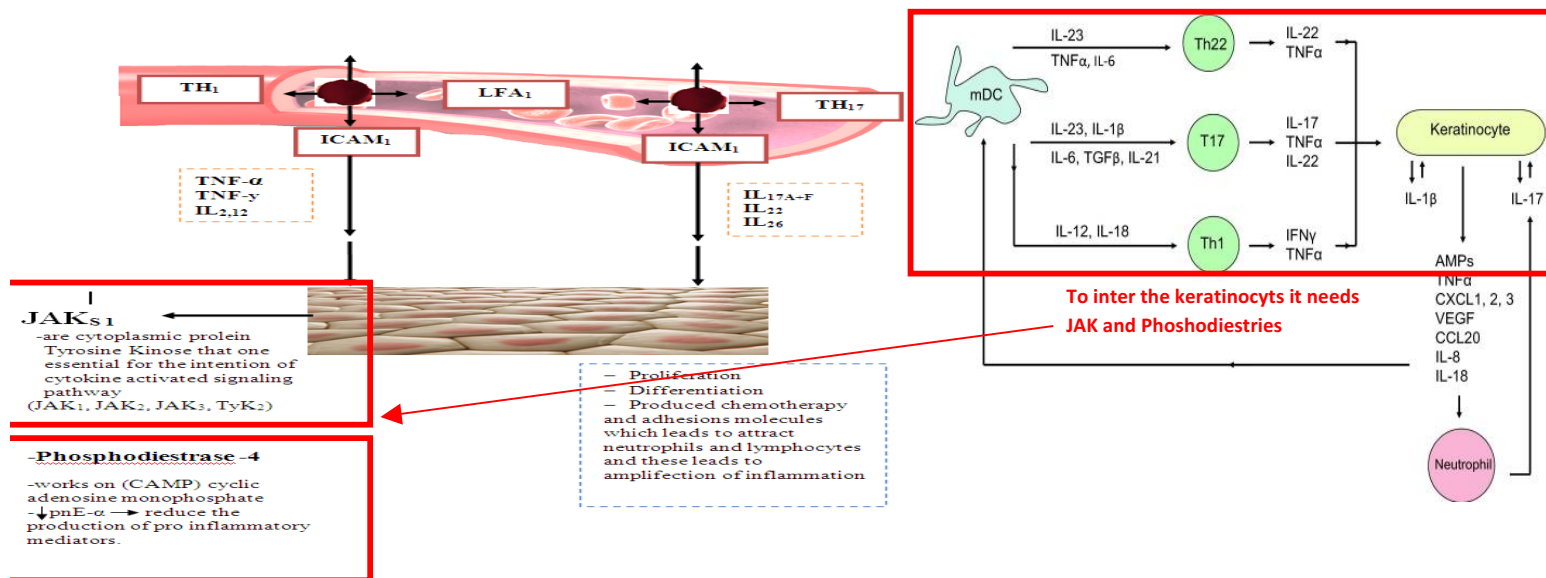
- Several genetic loci for psoriasis have been reported.
- There are at least 12 different PSORS loci.
- Recently, genome-wide association studies showed 50 regions associated with psoriasis risk.
 - ✓ One affected parent: 16%
 - ✓ Both parents: 50%
 - ✓ Non-psoriatic parents with affected child: 10%
 - ✓ Monozygotic twins: 70% -Dizygotic twins: 20%

❖ Environmental factors:

- Infection: **streptococcal infection**, beta hemolytic in children.
- Physical agents: stress, alcoholism, smoking
- Koebner phenomenon: presence of skin lesion in the site of trauma
- Drugs: lithium, anti- malarials, NSAID, **beta-blockers**.

□ This is important! to understand the drugs MOA

- Genetically predisposed individuals and triggering factors lead to **stressed keratinocytes**.(exogenous triggers and indigenous factors)
- 1) Stressed keratinocytes will produce: cathelicidin (LL-37) "highly anti microbial" that's way they don't have infection.
 - 2) LL-37 binds to self-DNA and self-RNA released from stressed or dying keratinocytes.
 - 3) Activates pDCs via TLR9 and TLR7 also activates mDCs via TLR8
 - 4) Activated **dendritic cells** in LN (lymph nodes) will release **IL12, IL23** and **TNF-Alpha**



❖ **Epidermal cell kinetics**

- The growth fraction of basal cells is increased to almost **100%** compared with only **30%** in a normal skin. (increase amount of production)
- The epidermal turnover time is shortened to less than **10 days** compared with 30 to 60 days in normal skin. (fast production)

✚ **Inflammatory factors:**

- Increase level of TNF
- TNF receptors are up-regulated
- Increase level of interferon gamma
- Increase level of interleukin 2, 12, 23 and 17

✚ **Immunological factors:**

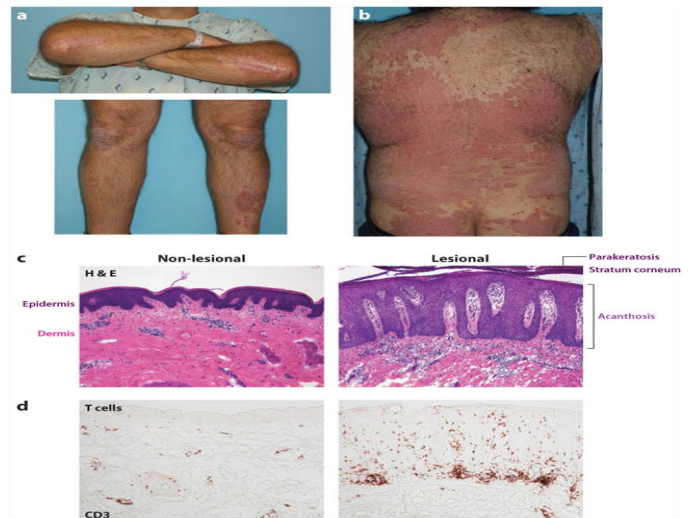
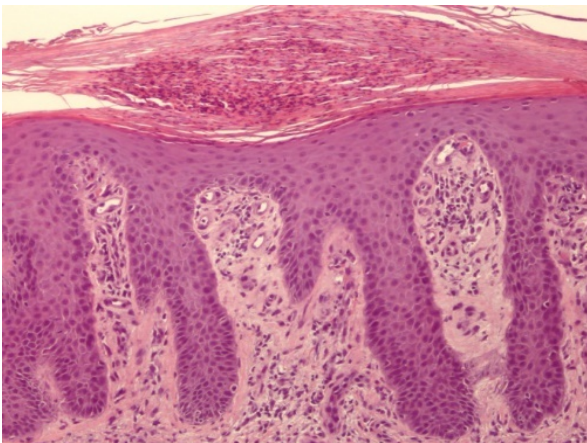
Psoriasis is fundamentally an inflammatory skin condition with reactive abnormal epidermal differentiation and hyper proliferation.

The inflammatory mechanisms are:

- Immune based and most likely initiated and maintained primarily by T cells in the dermis.
- Antigen-presenting cells in the skin, such as Langerhans cells. - T-cells.
- Auspits sign.

✚ Histology

- **Parakeratosis (nuclei retained in the horny layer)** Normally when the cells reach to the horny layer it becomes Anucleated but in Psoriasis due to the rapid division of keratocytes.
- Cells in the horny layer retain some of its organelles including its nucleus.
- Irregular **thickening of the epidermis** over the rete ridges but **thinning over dermal papillae**.
- **Auspitz sign:** when you remove the scales, a pinpoint bleeding occurs.
- Epidermal polymorphonuclear leucocyte infiltrates (**Munro abscesses**)
 - **Epidermo-Tropism** is the process when the neutrophils migrate from the Dermis to the Epidermis (**MCQ**).
 - If the neutrophils migrate and accumulate it will result in the formation of Micro-abscesses called Munro Abscesses, if amount increased it will form pustular psoriasis.
- Dilated capillary loops in the dermal papillae.
- T-lymph infiltrate in the upper dermis.



✚ Types of Psoriasis:

1- Non-pustular psoriasis

- Psoriasis vulgaris
- Guttate psoriasis
- Erythrodermic psoriasis
- Palmoplantar psoriasis
- Psoriatic arthritis (PsA)
- inverse psoriasis

2-Pustular psoriasis

- Generalized pustular psoriasis (von Zumbusch type)
- Impetigo herpetiformis
- Localized pustular psoriasis (Palmoplantarpustular psoriasis and Acrodermatitis continua of Hallopeau).

Psoriasis can occur on any part of the body:

- Scalp psoriasis
- Genital psoriasis
- Around eyes, ears, mouth and nose
- On the hands and feet
- Psoriasis of the nails

1- Plaque psoriasis (Psoriasis Vulgaris):

The **most common** type of psoriasis. It is characterized by round-to-oval red **plaques** distributed over extensor body surfaces and the scalp.

Up to 10-20% of patients with plaque psoriasis may evolve into more severe disease, such as **pustular** or **erythrodermic psoriasis**



2-Psoriasis, Guttate:

Small, **droplike**, 1-10 mm in diameter, salmon-pink papules "more red", usually with a fine "less" scale. **Younger than 30 years**. **Upper respiratory infection** secondary to group A beta hemolytic streptococci. On the trunk and the proximal extremities "**in the hidden areas**". Resolution within few months.



3- Erythrodermic Psoriasis:

- Scaly erythematous lesions, involving 90% or more of the cutaneous surface.
- Hair may shed; nails may become ridged and thickened.
- Few typical psoriatic plaques.
- Unwell, fever, leukocytosis.
- **Excessive body heat and hypothermia** (increase heat on skin because dilatation of blood vessels but the patient will feel cold) (Low core temperature and high superficial temperature).
- Increase cutaneous blood flow (can cause heart failure).
- **Increase percutaneous loss of water, protein and iron** (iron deficiency anemia, because lose of keratin and hypoproteinemia)
- **Increase percutaneous permeability high absorption** (topical drugs toxicity).



4- Pustular Psoriasis:

The pustules are due to the Murno abscess (Micro-abscess due to the Epidermo-Tropism explained earlier). If the patient is presented with Pustular Psoriasis this means it is a **severe** type of psoriasis (Huge amount of Neutrophils are invading the skin!!)

- Uncommon form of psoriasis.
- Pustules **on an erythematous background.** imp
- Psoriasis vulgaris may be present before, during, or after it.
- Pus is sterile.

❖ Pustular psoriasis may be classified into several types:

1- Generalized type (von Zumbusch variant):

- Generalized erythema studded with interfollicular pustules.
- Fever, tachypnea and tachycardia.
- Absolute lymphopenia with polymorph nuclear leukocytosis up to 40,000/ μ L.

2- Localized form (in palms and soles).

❖ Causes of Pustular Psoriasis:

1. Idiopathic in many patients but can be caused by:
2. **Withdrawal of systemic steroids.**
3. Drugs; including: Salicylates, Lithium, Phenylbutazone, Hydroxychloroquine, Interferon.
4. Strong, irritating topicals; including: Tar, Anthralin, Steroids under Occlusion, and Zinc Pyrithione in shampoo.
5. Infections.
6. Sunlight (or Phototherapy).
7. Cholestatic Jaundice
8. Hypocalcemia



5- Psoriasis inversus (Sebopsoriasis):

- Over body folds.
- The erythema and scales are very similar to that seen in Seborrhoeic dermatitis (it has no or very thin scales).



6- Psoriatic Arthritis:

- Most commonly a seronegativeoligoarthritis.
- Classical PsA: interphalangeal joints of the hands and feet. incidence of nearly 10%.
- Asymmetric oligoarticular arthritis: It is the most characteristic form of joint involvement. 11% of cases.
- Symmetric polyarticular form: It resembles rheumatoid arthritis.
- Incidence is between 15–61%.
- Arthritis mutilans is a rare form of psoriatic arthritis, occurring in 5% of patients with psoriatic arthritis
- Spondylitic form: Isolated spondylitis is rarely seen.

7-Psoriatic nail:

- 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.
- (more risk for Psoriatic arthritis)
- Oil drop or salmon patch/nail bed Pitting.
- Subungual hyperkeratosis.
- Onycholysis.
- Beau lines (longitudinal groove).



Differential diagnosis of :

- | | |
|---|----------------------------|
| 1-Bowes Disease | 10-Nummular Dermatitis |
| 2-Cutaneous T-Cell Lymphoma | 11-Parapsoriasis |
| 3-Drug Eruptions | 12-Pityriasis Rosea |
| 4-Erythema AnnulareCentrifugum | 13-Pityriasis RubraPilaris |
| 5-Extramammary Paget Disease | 14-Seborrheic Dermatitis |
| 6-Lichen Planus | 15-Syphilis |
| 7-Lichen Simplex Chronicus | |
| 8-Lupus Erythematosus, Discoid | |
| 9-Lupus Erythematosus, Subacute Cutaneous | |

+ Investigations:

1. Skin biopsy (**not needed for diagnosis** except in case there are differential diagnoses, It is preferable to do it for *documentation* because it is a chronic disease).
2. Others (imaging if there is joint involvement, CBC, Hg, LFT, Renal profile, Ca, VitD... **to assess the complications or to establish a baseline for treatment**).

+ Treatment

- What influence the choice of treatment? **Severity index.**
- Tools to measure severity index:
 - 1- Psoriasis Area and Severity Index (PASI)
 - 2- physician global assessment (PGA)
 - 3-Dermatology life quality index (DLQI) **if less than 20% but affecting life quality.**
 - 4-body surface area (BSA) affected: role of hand (hand = equal 1% BSA) **less than 10% to 20% topical, more than 20% systemic.**

What are the indications of Systemic Therapy in Psoriasis?

1) More than 20% of skin involvement.

2) Severe, We define it as Severe Psoriasis when its affect the Quality of Life e.g. a Female patient with Scalp Psoriasis or a Surgeon with Hand Psoriasis)

+ Management of psoriasis

- Educating the patient and family
- Psychosocial support
- **Smoking** and weight
- Several factors need to be taken into account when selecting a specific treatment: age, quality of life, severity of psoriasis, location of psoriasis, type of psoriasis, tolerability, safety and patient preferences.

✚ **Topical Treatments** If more than 20% of the body involved give systemic treatment.

- Vehicle for topical treatment: creams "flexers", ointments "palmes soles" , foams, gels and lotions.
- Emollients: فازلين
- Keratolytics كانسات: remove scales, urea and salicylic acid.
- **Topical Corticosteroids: "anti inflammatory" systemic steroids are contraindicated except in pregnancy.**
- Vitamin D Analogs(calcipotriol) **Alone or with Topical Corticosteroids.**
- Calcineurin Inhibitors: tacrolimus and pimecrolimus.
- Anthralin: Dithranol
- Tazarotene
- Tar

If the topical doesn't work or there is systemic manifestation "knee pain" no rule for topical go to systemic

✚ **Systemic: Phototherapy for psoriasis**

- broadband ultraviolet B: 290-313 nm
- narrowband ultraviolet B: 311 nm
- UVA: 320-400 nm
- psoralen plus ultraviolet A (PUVA)
- Excimer laser :308 nm
- NB-UVB is the most commonly used due to: easier to use, and has fewer side effects when compared with BB-UVB or PUVA
- Mechanism of actions: **induces apoptosis of keratinocytes and T cells, promoting migration of Langerhans cells out of the epidermis and induces alterations in the cytokine profile of psoriasis.**
- Typically requires 20–36 sessions for NB-UVB.
- **3 sessions per week**
- Minimum 24-h interval between each session
- Physician follow-up every 3 months for the first year
- Clearance rates range from 60 to 70%
- **Side effects of UVB phototherapy:**
 - Burning: Redness, tenderness, pain, tightness, itching, and rarely blistering Noticeable 4–6 h after treatment
 - Photoaging: wrinkling, laxity, increased fragility, mottled pigmentation, telangiectasias, and atrophic areas
 - Tanning: Skin darkening

If the phototherapy doesn't work go to Methotrexate

❖ Methotrexate

- is a **folic acid antagonist**: blocked dihydrofolate reductase and this will lead to inhibit the metabolism of folic acid.
- antiproliferative, induces apoptosis and an immune and anti-inflammatory modulator
- 7.5mg to 25mg weekly dose(orally or IM)
- **Folic acid supplement**
- adverse events:
 - infection, nasopharyngitis, headache.
 - **teratogenicity for male and female (3months for wash out)**
 - hepatotoxicity
 - myelo-suppression
 - ulcerative stomatitis, pulmonary fibrosis, skin reactions, and opportunistic infection.

❖ Cyclosporine

- Inhibits calcineurin phosphorylation
- 2 to 5mg/kg/day and from 12 to 24 weeks to limit cumulative nephrotoxicity
- Is used short-term for severe psoriasis flares, particularly pustular and erythrodermic psoriasis
- Monitoring for **hypertension, hyperlipidemia, hypomagnesemia, and hyperkalemia**
- Adverse events: **chronic nephrotoxicity**, hepatotoxicity, thrombotic microangiopathy, malignancies and serious infection

❖ Acitretin

- A vitamin A-derived retinoid
- works by stopping excessive growth and thickening of skin cells
- Doses from 10mg/day to 75mg/day
- To treat mild-to-moderate pustular, palmoplantar and erythrodermic psoriasis
- Or plaque psoriasis as combination therapy with phototherapy and biologics
- Adverse events: dry eyes, lipid derangements, pancreatitis, hyperostosis, pseudotumor cerebri, hepatotoxicity, and teratogenicity
(only female:2 years for wash out)

NEW ORAL THERAPY

1-Apremilast:

- is a phosphodiesterase 4 (PDE4) inhibitor
- promotes anti-inflammatory processes
- for moderate-to-severe plaque psoriasis
- 30mg BID for 16 weeks
- Adverse events: nausea, diarrhea, nasopharyngitis. headache,

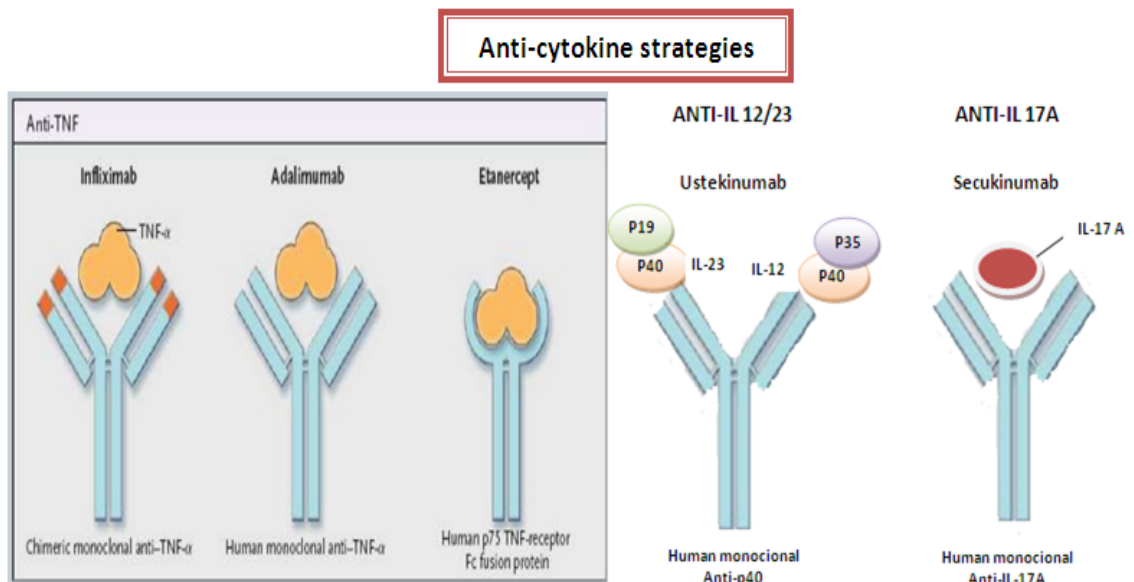
2- Tofacitinib:

- inhibits janus kinase (JAK) 1 and 3
- suppresses receptors for numerous cytokines
- 2mg-10mg BID daily
- due to Serious side effects, Tofacitinib remains under active investigation.

Biologics for Psoriasis

(for the treatment of moderate to severe Psoriasis) :

Check for hidden infection before start treating with biologics (TB, hepatitis...)



❖ Eligibility criteria

1-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.

2- Phototherapy and alternative standard systemic therapy are contraindicated or cannot be used.

3-Unresponsive to standard systemic therapy

❖ General recommendations for all patients who will be treated with biologics at baseline

-Complete blood cell count including platelet count

-Liver function tests

-Renal panel

-Hepatitis panel

-Tuberculosis (TB)

-HIV

- **Pregnancy test** because you have to stop it 3 months before delivery.

- Avoid vaccination with live vaccines (varicella;mumps, measles, and rubella; oral typhoid;yellow fever)

- Avoid live-attenuated vaccines (including intranasal influenza and the herpes zoster vaccine).

❖ Contra-indications for biologic therapy

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

➤ Etanercept

- **TNF inhibitor**
- Moderate-to-severe psoriasis
- recommended as first-line biologic therapy for psoriasis
- 50 mg twice/week given subcutaneously for 3 months followed by 50 mg once/week
- Side effects:
- Mildly pruritic injection site reactions
- Rare cases of serious infections (i.e., TB) and malignancies

➤ Adalimumab

- **TNF inhibitor**
- recommended as first-line biologic therapy for psoriasis
- induction dose: 80 mg at the start and 40 mg at week 1 Then maintenance dose: 40 mg every other week.

Side effects:

- Mildly pruritic injection site reactions
- Rare cases of serious infections (i.e., TB) and malignancies

➤ Infliximab very fast action

- **TNF inhibitor**
- Severe psoriasis
- 5 mg/kg at weeks 0, 2, and 6 week then every 8-week intervals to maintain disease control up to 1 year (IV).
- Side effects: Infusion reactions and rare cases of serious infections (i.e., TB) and malignancies including hepatosplenic T-cell lymphoma (in children); there are rare reports of drug-induced, reversible side effects including lupus without renal, or CNS complications

➤ Secukinumab

- **Selectively binds and neutralizes interleukin (IL) 17-A**
- moderate-to-severe plaque psoriasis
- recommended as second-line biologic therapy for psoriasis
- 300 mg by subcutaneous injection with initial dosing at weeks 0, 1, 2, and 3 followed by 300 mg every 4 weeks.
- **Side effects:**
- nasopharyngitis, diarrhea, and upper respiratory tract infection. incremental risk in **Candida infections (limited to nonserious, localized mucosal, or cutaneous candidiasis)** has been noted.

➤ Ustekinumab

- Prevents the interaction of IL12 and IL23 with their cell surface receptors, blocking Th-1/IL12 and Th-17/IL23 inflammatory pathways.
- Recommended as second-line biologic therapy for psoriasis
- 45 or 90 mg at week 0 and 4 then every 12 weeks
- Ustekinumab may be associated with lower SAE rates, and lower infectious and serious infectious event rates compared to the TNF antagonists

➤ Alefacept (Amevive):

- It is the first biologic agent approved by the FDA for the treatment of Psoriasis.
- It works by blocking T cell activation and proliferation by binding to CD2 receptors on T cells.
- This stops the T cells from releasing cytokines, which is the primary cause of the inflammation.
- 7.5 mg by IV injection or 15 mg by IM injection once weekly for 12 weeks.
- **S/E:** dizziness, cough, nausea, itching, muscle aches, chills, injection site pain and injection site redness and swelling.
- Infections.
- Not used nowadays because its effect is very weak.

➤ Etanercept (Enbrel):

- This molecule serves as an exogenous TNF receptor and prevents excess TNF from binding to cell-bound receptors.
- 50mg SC given twice weekly for 3 months, then 50 mg SC qwk.
- **Contraindications:** Sepsis, active infection, concurrent live vaccination.
- **S/E:** injection site reactions (most common).
- Upper respiratory tract infections.

Adalimumab (Humira) SC, 80 mg € 40 mg

Infliximab (Remicade) IV, 5 mg/kg

Ustekinumab (Stelara) Anti-Interleukin (injection every 3 month)

Tofacitinip: Janus kinase (Jak) pathway inhibitor.

Lines of Treatment in Psoriasis:**Summary From 431 team work**

- 1- Topical Therapy.
- 2- Systemic Therapy (if more than 20% of surface area OR it affect the Quality of Life)
- 3- Phototherapy.
- 4- Biological Therapy
- 5- Cytotoxic Medications (Methotrexate, Vit A derivatives, Cyclosporine etc..)

Biological Therapies generally are safe but the most important side effect is the reactivation of chronic infections especially: TB so we need to do test for it (e.g. PPD or QuantiFERON)

PPD = Give 10 units of PPD and read it after 48 to 72 hrs.

The induration should be more than 15 to be positive, if it is from 5 - 10 = Gray area we should do QuantiFERON to confirm.

Lecture 2: Other Papulosquamous Diseases

Objectives:

- Define the papulosquamous disease.
- Highlight on the pathogenesis of papulosquamous diseases.
- Discuss the clinical features of papulosquamous diseases.
- Highlight on the papulosquamous diseases treatment.

Lichen Planus (الحرزاز)

- Lichen planus (LP) is a **pruritic**, papular eruption characterized by its **violaceous color**, **polygonal shape**, and sometimes fine scale.
- It is most commonly found on the **flexor** surfaces of the upper extremities, on the genitalia, and on the mucous membranes.

+ Epidemiology:

- Approximately 1% of all new patients in derma clinic.
- Rare in children
- **F=M**
- LP can occur at any age but two thirds of patients are aged 30-60 years
- **No racial predispositions have been noted**

+ Pathophysiology :

- The cause of LP is **unknown**
- LP may be a **cell-mediated immune** response of unknown origin
- LP may be found with other diseases of altered immunity like ulcerative colitis, alopecia areata, vitiligo, dermatomyositis.
- An association is noted between LP and **hepatitis C virus** infection, chronic active hepatitis, and primary biliary cirrhosis.
- Familial cases.
- Drug may induce lichenoid reaction like: **Thiazide, Antimalarials, Propranolol.**

+ Clinical features:

- Most cases are **insidious**.
- The initial lesion is usually located on the **flexor surface** of the limbs
After a **week or more**, a **generalized eruption** develops with maximal spreading **within 2-16 weeks**.
- **Pruritus** is common **but varies in severity**.
- **Deep pigmentation** may persist for long time.
- **LP With oral ulcers risk of Squamous cell carcinoma.**
- **Oral lesions** may be asymptomatic or have a burning sensation.
- In more than 50% of patients with cutaneous disease, the lesions resolve within 6 months, and 85% of cases subside within 18 months.
- The papules are **violaceous, shiny, and polygonal**. varying in size from 1mm to greater than 1 cm in diameter.

- They can be discrete or arranged in **groups of lines or Circles**.
- Characteristic **fine, white lines**, called *Wickham Stria*, are often found on the **papules**.
- Oral lesions are classified as **reticular, plaque-like, atrophic, papular, erosive, and bullous**.
- **Ulcerated oral lesions** may have a higher incidence of **malignant** transformation.
- **Genital involvement** is common in men with cutaneous disease
- **Vulvar involvement** can range from reticulate papules to severe erosions.

Variations in LP : skipped by the doctor

1- Hypertrophic LP:

These extremely pruritic lesions are most often found on the extensor surfaces of the lower extremities, especially around the ankles.

2- Atrophic LP:

Is characterized by a few lesions, which are often the resolution of annular or hypertrophic lesions.

3- Erosive LP: risk of squamous cell carcinoma

4- Follicular LP:

- Keratotic papules that may coalesce into plaques.
- A scarring alopecia may result.

5- Annular LP:

Annular lesions with an atrophic center can be found on the buccal mucosa and the male genitalia.

6- Vesicular and bullous LP:

Develop on the lower limbs or in the mouth from preexisting LP lesions.

7- Actinic LP:

- Africa, the Middle East, and India.
- Mildly pruritic eruption.
- Characterized by nummular patches with a hypo-pigmented zone surrounding a hyper-pigmented center.

8-LP Pigmentosus:

- Common in persons with darker-pigmented skin.
- Usually appears on face and neck.

✚ LP and nail :

- In 10% of patients
- Nail plate thinning causes longitudinal grooving and ridging
- Subungual hyperkeratosis and Onycholysis.
- Rarely, the matrix can be permanently destroyed with prominent Pterygium formation.
- Twenty-nail dystrophy

Pterygium unguis (Dorsal pterygium) forms as a result of scarring between the proximal nailfold and matrix.

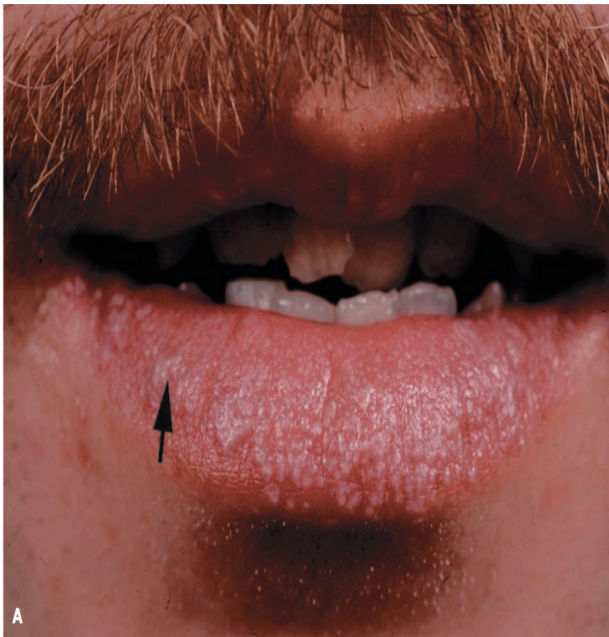


Figure 14-23. Lichen planus (A) Silvery-white, confluent, flat-topped papules on the lips. Note: Wickham striae (arrow). **(B)** Lichen planus, Koebner phenomenon. Linear arrangement of flat-topped, shiny papules that erupted after scratching.



Figure 14-20. Lichen planus (A) Flat-topped, polygonal, sharply defined papules of violaceous color, grouped and confluent. Surface is shiny and, upon close inspection with a hand lens, fine white lines are revealed (Wickham striae, arrow). **(B)** Close up of flat-topped shiny violaceous papules that are polygonal.

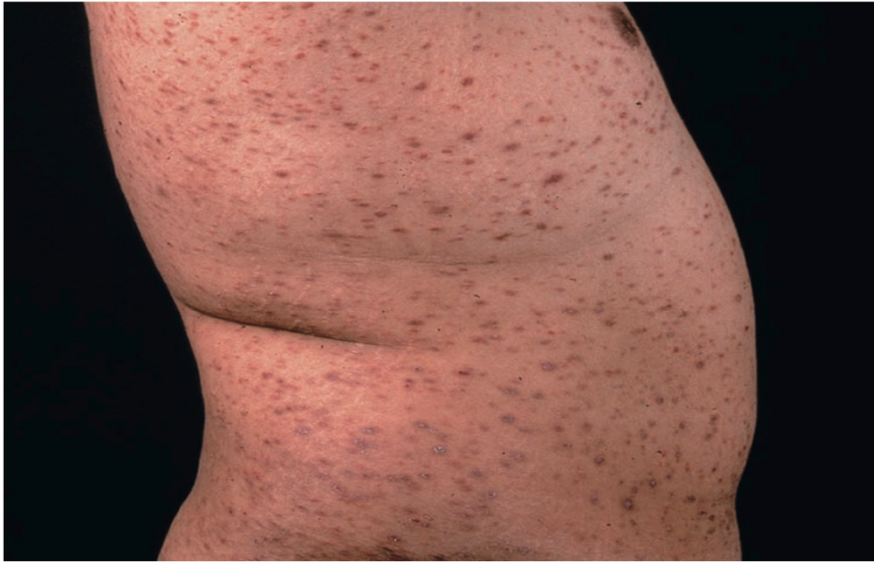


Figure 14-22. Disseminated lichen planus A shower of disseminated papules on the trunk and the extremities (not shown) in a 45-year-old Filipino. Due to the ethnic color of the skin, the papules are not as violaceous as in Caucasians but have a brownish hue.



Figure 32-9. Lichen planus (A) Middle finger: involvement of the proximal fold and matrix has caused trachonychia, longitudinal ridging, and pterygium formation. Index finger: destruction of the matrix and nail plate is complete with onychia. Seven of ten fingernails are involved; the others are normal. (B) Involvement of the nail matrix with scarring or pterygium formation proximally dividing the nail plate in two. (C) Early involvement of the matrix with thinning of the thumbnail plates. (D) Same patient as Fig. 32-8C 2 years later, the nail plate is completely destroyed, i.e., onychia.

+ Differentials diagnoses:

- Graft Versus Host Disease
- Lichen Nitidus
- Lichen Simplex Chronicus
- PityriasisRosea
- Psoriasis, Guttate
- Psoriasis, Plaque
- Syphilis
- TineaCorporis

+ Treatment:

- Self-limited disease that usually resolves within 8-12 months.
- Treat to prevent hyperpigmentation.
- Anti-histamine (for pruritus).
- Topical steroids, particularly class I or II ointments
- Systemic steroids for symptom control and possibly more rapid resolution
- Oral Acitretin (Retinoid).
- Photo-therapy
- Others

Pityriasis Rosea: (النخالية الوردية)

+ Definition:

- Acute mild inflammatory exanthem.
- Characterized by the development of erythematous scaly macules on the trunk.

+ Epidemiology:

- In children and young adult
- Increased incidence in Spring and Autumn
- PR has been estimated to account for 2% of dermatology outpatient visits.
- PR is more common in women than in men

+ Pathophysiology:

- PR is considered to be a viral exanthem
- Immunologic data suggest a viral etiology
- Families and close contacts
- A single outbreak tends to elicit lifelong immunity
- **Human herpes virus (HHV)–7 and HHV-6**
- PR-like drug eruptions may be difficult to distinguish from non–drug-induced cases.
- **Captopril**, metronidazole, **isotretinoin**, penicillamine, bismuth, gold, barbiturates, and omeprazole.

+ Clinical Features:

- **Begins** with a **solitary** macule that heralds the eruption (**herald spot/patch**).
- Usually a salmon-colored macule.
- Over a few days it become a patch with a collarette of fine scale just inside the well-demarcated border.
- Within the next 1-2 weeks, a generalized exanthem usually appears.
- Bilateral and symmetric macules with a collarette scale oriented with their long axes along cleavage lines.
- Tends to resolve over the next 6 weeks.
- Pruritus is common, usually of mild-to-moderate severity.
- Over trunk and proximal limbs.

+ Atypical form of PR:

- Occurs in 20% of patients
- Inverse PR
- Unilateral variant
- Papular PR
- Erythema multiforme–like
- Purpuric PR
- **If it appears in palms and soles —>DDx: secondary syphilis.**



+ Differential diagnosis:

- Lichen Planus
- Nummular Dermatitis
- PityriasisLichenoides
- Psoriasis, Guttate
- Seborrheic Dermatitis
- Syphilis
- TineaCorporis (Scenario: Patient presents in early stage (only herald patch) is misdiagnosed to have a fungal infection. The physician prescribes an anti-fungal agent. After a few days the patient returns upset with full exanthem and is assuming that the prescribed medication worsened their condition).



+ Treatment:

- Reassurance that the rash will resolve
- Relief of pruritus
- Topical menthol-phenol lotion
- Oral antihistamines
- Topical steroids
- Systemic steroids
- Ultraviolet B (UV-B) light therapy
- Antiviral

Summery

Lichen Palnus

Pruritic papular eruption characterized by its violaceous color, polygonal scale, sometimes fine

Scale Involves skin, nails and mucous membranes.

Rare in children. Two thirds of patients are aged 30-60 years.

Pruritus and deep pigmentations are the most important complaints.

5 P's to describe LP: Plentiful, Purple, Pruritic, Polygonal, Papules.

Clinical features:Violaceous, shiny, and polygonal papules of varying size. They can be discrete

Or arranged in groups of lines or circles + Characteristic fine white lines (Wickham stria).

Nail changes: longitudinal grooving and ridging+ subungal hyperkeratosis + onycholysis + pterygium.

Management:self-limited resolves in 8-12 months but leave hyperpigmentation.

Pityriasis Rosea

Acute, self-limiting, mild inflammatory exanthema of unknown origin. May be precipitated by a

Viral infection or drug reaction.

Clinical features: Begins with a herald patch followed by generalized exanthema after 1-2 weeks.

Oval well demarcated patch with fine scale running along cleavage lines.

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