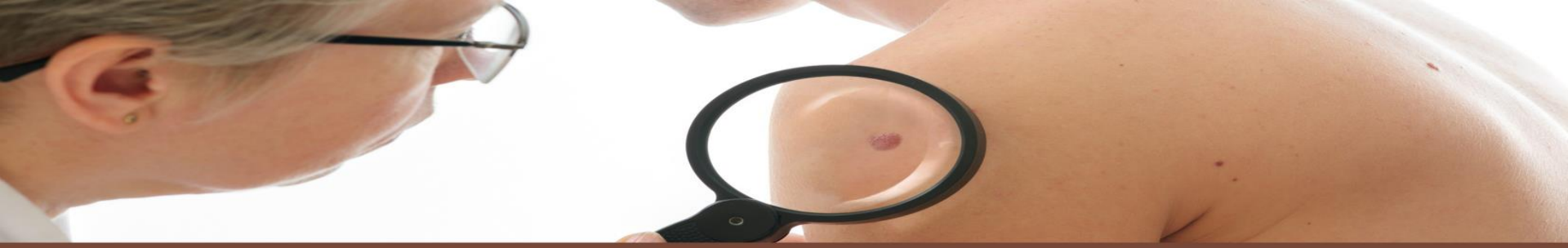


433 Teams

DERMATOLOGY

L4-Psoriasis and other Papulosquamous disorders

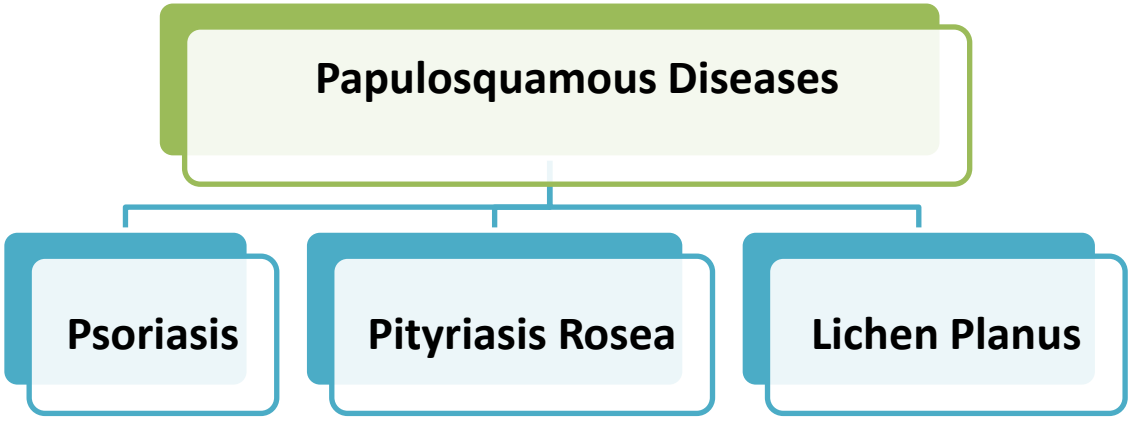
derm433team@gmail.com

جامعة الملك سعود
King Saud University

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

Mind Map



Color Index: Slides, Important, 432 Notes Doctor's Notes (Group F)

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
- Pityriasisrubrapilaris
- Secondary syphilis
- Miscellaneous mycosis fungoides, discoid lupus erythematosus, ichthyoses.

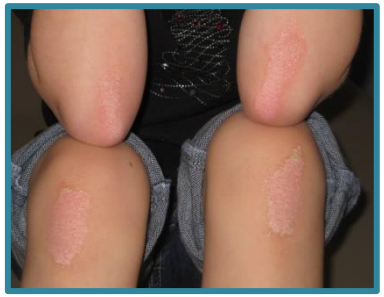
Psoriasis is characterized by an abnormally excessive and rapid growth of the epidermal layer of the skin.
Abnormal production of skin cells

1-Psoriasis

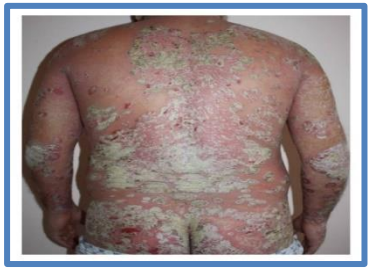
Definition:

- 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.**

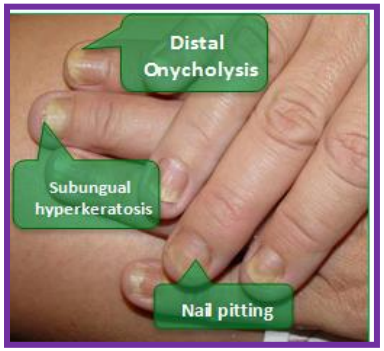
Well defined regular brownish to reddish scaly papule (classical presentation of chronic plaque Psoriasis)
Crust: dried fluid regardless the type of fluids, **scales:** dead keratocytes

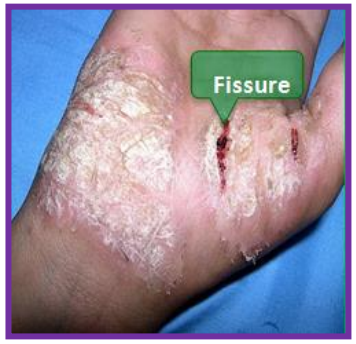


bilateral symmetrical Well defined regular erythematous scaly plaques on elbows and knees



Well defined Generalized dull red scaly plaques on the back





Fissures are an additional feature of the Psoriasis in the Palms & Soles. It develops becausethe 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!

Nail changes:
1-distal onycholysis →the separation of the distal nail plate from the nail bed
2-subungual hyperkeratosis→ scales under the nail plate
3-nail pitting(important sign) → severe nail pitting indicate **psoriatic arthritis**

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 **(early onset) usually associated with family history ,more severe**
  - and over 50 years of age.**(late onset)**
- 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.(**Strong genetic association).**
- 75% has nail changes
- 30 % of patients with Pso will develop **psoriatic arthritis**
- 75 % of psoriatic arthritis : the skin disease precedes arthritis, while in 15 % of patients Pso appears after PsA and in 10 % the cutaneous and articular involvement are simultaneous

Skin changes appear before psoriatic arthritis in most cases.

Pathogenesis: (important part)

-Who is the pathogenic driver in psoriasis: keratinocyte cells or T lymphocyte cells **Until today no one knows the answer for this question but they believe that the two cells play a role in the pathogenesis of psoriasis.**
-Considered to be an **autoimmune disease**

1-Genetic factor:- There are two types:-

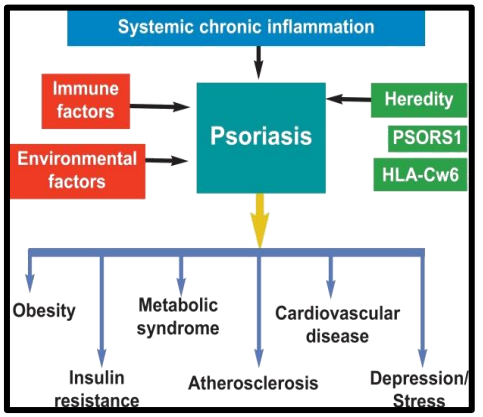
1-type I psoriasis(early onset): more likely to be familial, have a severe clinical course and is associated with **HLA-Cw6, B13 and B57**

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%

2-Environmental factors:-→ Next slide



2- Environmental factors:-

-**Infection**: streptococcal infection **(Especially beta hemolytic streptococcus infection)** ,Physical agents: stress, alcoholism, smoking

-**Drugs**: **lithium, anti- malarials (given to SLE+RA patients), NSAID, beta-blockers (it may cause psoriasis)**

- trauma (Koebner phenomenon): occurs in skin diseases specially psoriasis and lichen planus, in which the characteristic lesions of the disease appear in linear form in response to such trauma as cuts burns scratches.

Genetically predisposed individuals and **triggering factors** lead to **stressed keratinocytes.**
(**exogenous triggers and indigenous factors**)

↓

trauma to keratinocytes (Stressed keratinocytes)will produced: cathelicidin **(LL-37)**anti microbial**)**

↓

LL-37 binds to self-DNA and self-RNA (released from stressed or dying keratinocytes.)

↓

This component (LL-37 + self-DNA and self-RNA of Stressed keratinocytes) go to pDCs (Plasmacytoid dendritic cells) and mDCs (Myeloid dendritic cells)

↓

then →**Activates** pDCs via **TLR9** and **TLR7** also **activates** mDCs via **TLR8**

↓

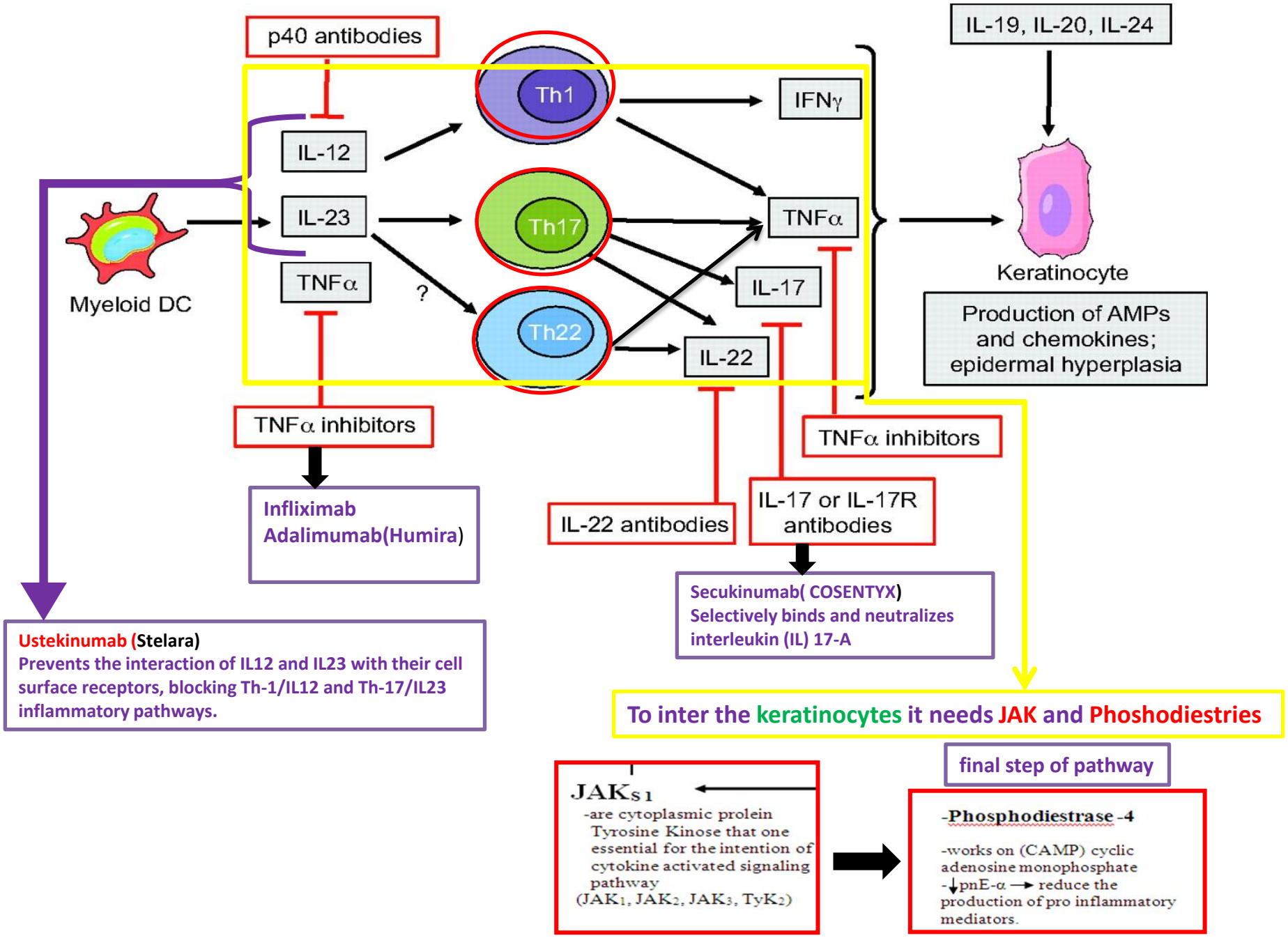
Activated dendritic cells in lymph nodes will release **IL12, IL23 and TNF-Alf**

↓

Next Slide

**TLR9 ,TLR7,TLR8 and LL-37 →highly anti microbial" that's way they don't have infection.

pDCs=Plasmacytoid dendritic cells
mDCs=Myeloid dendritic cells
TLR =Toll-like receptors


p40 antibodies
IL-19, IL-20, IL-24
Th1
IFNγ
IL-12
IL-23
Th17
TNFα
Myeloid DC
TNFα
?
IL-17
Th22
IL-22
Keratinocyte
Production of AMPs and chemokines; epidermal hyperplasia
TNFα inhibitors
TNFα inhibitors
Infliximab
Adalimumab(Humira)
IL-22 antibodies
IL-17 or IL-17R antibodies
Secukinumab(COSENTYX)
Selectively binds and neutralizes interleukin (IL) 17-A
Ustekinumab (Stelara)
Prevents the interaction of IL12 and IL23 with their cell surface receptors, blocking Th-1/IL12 and Th-17/IL23 inflammatory pathways.
To inter the keratinocytes it needs JAK and Phoshodiestries
final step of pathway
JAKs 1
-are cytoplasmic prolein Tyrosine Kinose that one essential for the intention of cytokine activated signaling pathway
(JAK1, JAK2, JAK3, TyK2)
-Phosphodiestrase -4
-works on (CAMP) cyclic adenosine monophosphate
-↓pnE-α → reduce the production of pro inflammatory mediators.

TNF-α
TNF-y
IL2,12

IL17A-F
IL22
IL26

JAKS 1

-are cytoplasmic prolein Tyrosine Kinose that one essential for the intention of cytokine activated signaling pathway
(JAK1, JAK2, JAK3, TyK2)

- Proliferation
- Differentiation
- Produced chemotherapy and adhesions molecules which leads to attract neutrophils and lymphocytes and these leads to amplifection of inflammation

-Phosphodiestrase -4

-works on (CAMP) cyclic adenosine monophosphate
-↓pnE-α → reduce the production of pro inflammatory mediators.

Epidermal cell kinetics

- The growth fraction of basal cells (the amount of activated keratinocytes)**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

Histopathology Of Psoriasis

- **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 keratocyts.

Parakeratosis →Diagnostic clue of Psoriasis

- 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 →Auspit 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 **pustural psoriasis.**
- Dilated capillary loops in the dermal papillae.
- T-lymph infiltrate in the upper dermis.

Types of Psoriasis:

A)Non- pustular psoriasis

1. **Psoriasis vulgaris**
2. **Guttate psoriasis**
3. **Erythrodermic psoriasis**
4. **Palmoplantar psoriasis**
5. **Psoriatic arthritis (PsA)**
6. **inverse psoriasis**

B) Pustular psoriasis

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

A)Non-pustular psoriasis

1-Plaque psoriasis (Psoriasis vulgaris)

The **most common** type of psoriasis.

It characterized by round-to-oval red **plaques** distributed over extensor body surfaces and the scalp.

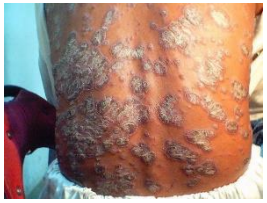
Up to 10-20% of patients with plaque psoriasis may evolve into more severe disease, such as **pustular** or **erythrodermic** psoriasis

2-Psoriasis, Guttate:

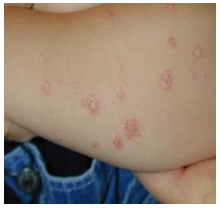
Small, droplike, 1-10 mm in diameter, salmon-pink papules "more red", usually with a fine "less" scale.

4 features of Guttate Psoriasis :

- **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.



Psoriasis vulgaris



Guttate psoriasis

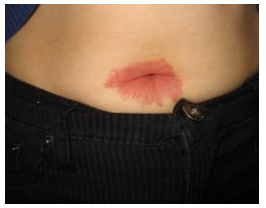
3- Erythrodermic Psoriasis:

- 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. mostly neutrophils
- **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- Psoriasis inversus (Sebopsoriasis):

Over body folds.

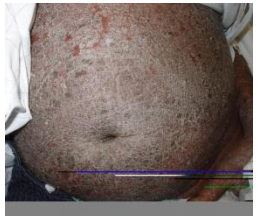
The erythema and scales are very similar to that seen in Seborrhoeic dermatitis (**it has no or very thin scales).**

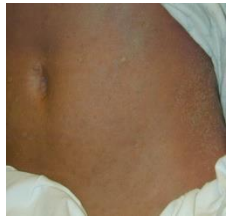


Psoriasis inversus (Sebopsoriasis):

5-Psoriatic Arthritis:

- Most commonly a seronegativeoligoarthritis.
- Classical PsA: interphalangeal joints of the hands and feet. I
- ncidence of nearly 10%.
- Asymmetric oligoarticular arthritis: It is the most characteristic form of joint involvement. 11% of cases.
- Symmetric poliarticular 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.





Erythrodermic psoriasis

7-Psoriatic nail:

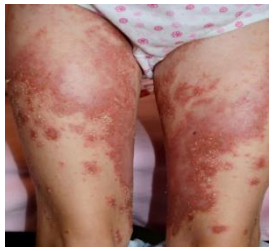
- Psoriatic nail disease occurs in 10-70% of all patients with psoriasis
- Less than 5% of psoriatic nail disease cases occur in patients without other cutaneous findings
- **(more risk for Psoriatic arthritis)**
- Oil drop or salmon patch/nail bed Pitting.
- Subungual hyperkeratosis.
- Onycholysis.
- Beau lines (longitudinal grove).

Differential diagnosis of Psoriasis *:*

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

B) Pustular Psoriasis:

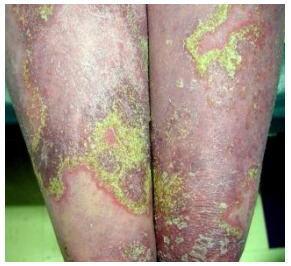
- The pustules are due to the Murno abscess (Micro-abscess due to the Epidermo-Tropism explained earlier).
- If the patient is presented with Pustular Psoriasis this means it is a **severe** type of psoriasis (Huge amount of Neutrophils are invading the skin)
- Uncommon form of psoriasis.
- **Pustules on an erythematous background. (important)**
- 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 interfolecular pustules.
- Fever, tachypnea and tachycardia.
- Absolute lymphopenia with polymorph nuclear leukocytosis up to 40,000/µL.



2-Localized form (in palms and soles).

Causes of Pustular Psoriasis:

Idiopathic in many patients but can be caused by:

- **Withdrawal of systemic steroids→So systemic steroids are contraindicated except in pregnancy.** .
- Drugs; including: Salicylates**,** **Lithium**, Phenylbutazone, Hydroxychloroquine, Interferon
- Strong, irritating topicals; including: Tar, Anthralin, Steroids under Occlusion, and Zinc Pyrithione in shampoo
- Infections.
- Sunlight (or Phototherapy).
- Cholestatic Jaundice
- Hypocalcemia

Investigations:

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).

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).**

Management of psoriasis

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 contraindecated 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 mainfestation "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 of Methotrexate

- 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 biologies
- **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 biologicals (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. **(Sensitive life affecting areas such as the face and hand will increase the DLQI even with small BSA).**
2- Phototherapy and alternative standard systemic therapy are contraindicated or cannot be used.
3-Unresponsive to standard systemic therapy

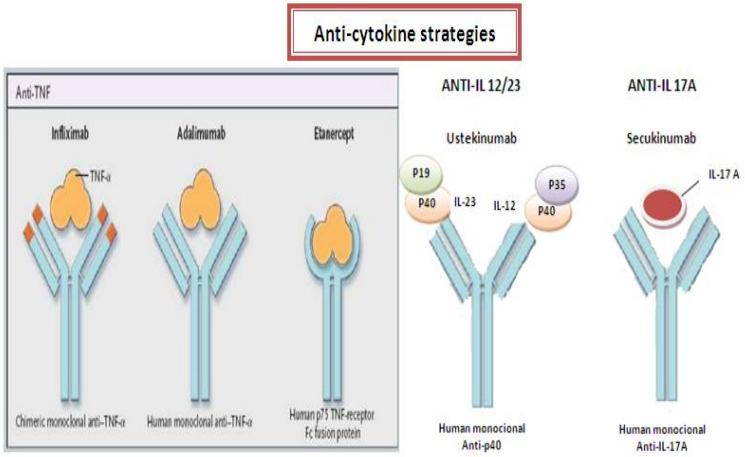
General recommendations for all patients who will be treated with biologics at baseline

-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.



Biological Therapy

1. 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

2-Adalimumab
TNF inhibitor
recommended as first-line biologic therapy for psoriasis
induction dose: 80 mg at the start and 40 mg at week 1Then
maintenance dose: 40 mg every other week.
Side effects:
Mildly pruritic injection site reactions
Rare cases of serious infections (i.e., TB) and malignancies

3-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

4-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.

5-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 o 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

2-Other Papulosquamous Diseases

Lichen Planus(الحزاز)

- Lichen planus (LP) is a **pruritic**, papular eruption characterized by its **violaceouscolor, 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

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 :

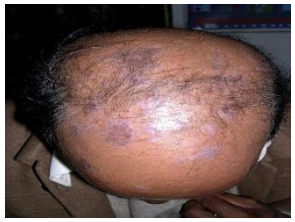
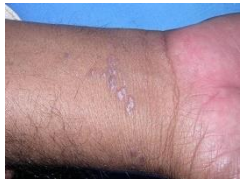
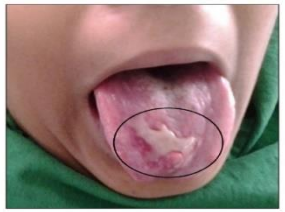
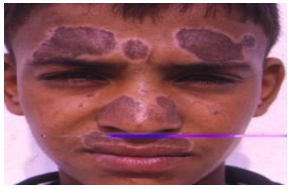
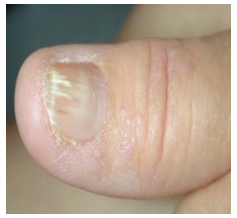
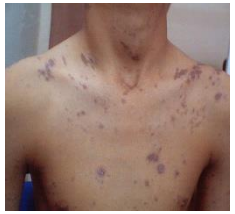
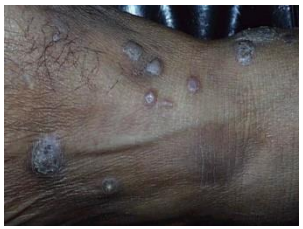
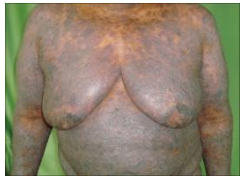
- In 10% of patients
- Nail plate thinning causes longitudinal grooving and ridging
- Subungual hyperkeratosis and Onycholysis.
- Rarely, the matrix can be permanently destroyed with prominent Pterygium formation.
- Twenty-nail dystrophy
- **Pterygium unguis (Dorsal pterygium) forms as a result of scarring between the proximal nailfold and matrix**

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

2-Other Papulosquamous Diseases

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:

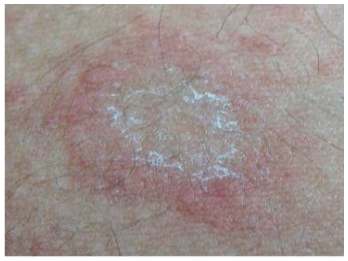
- PR is considered to be a viral exanthem
- Immunologic data suggest a viral etiology
- Families and close contacts
- A single outbreak tends to elicit lifelong immunity
- **Human herpes virus (HHV)–7 and HHV-6**
- PR-like drug eruptions may be difficult to distinguish from non–drug-induced cases.
-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

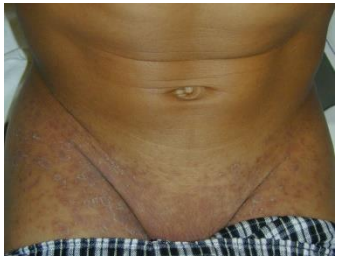
Clinical Features:

- 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**.





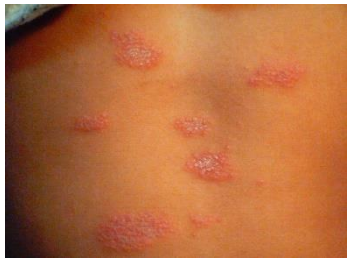
Differential diagnosis:

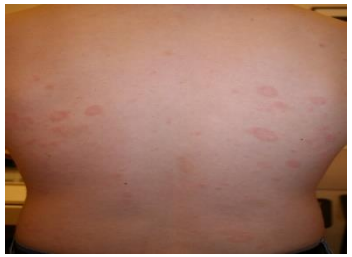
- Lichen Planus
- Nummular Dermatitis
- PityriasisLichenoides
- Psoriasis, Guttate
- Seborrheic Dermatitis
- Syphilis
- TineaCorporis (Scenario: Patient presents in early stage (only herald patch) is misdiagnosed to have a fungal infection. The physician prescribes an anti-fungal agent. After a few days 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

	Psoriasis	Lichen Planus	Pityriasis Rosea
pathogenesis	keratinocyte cells/T lymphocyte cells autoimmune disease Drugs: **lithium, anti- malarials ,nsaid, beta-blockers**	A cell-mediated immune response dermatosis. - Associated with: **ulcerative colitis, alopecia areata and vitiligo** - Increase in **hepatitis C virus** - Familial cases. - Drug may induce lichenoid reaction like: **thiazide, antimalarials, propranolol.**	Human herpesvirus (HHV)–7and HHV-6 drugs
clinical features	common, chronic and non-infectious disease. systemic complex disease. Primarily affects skin and joints. may be a risk factor for **metabolic syndrome ,myocardial infarction**). **higher prevalence** in western European and Scandinavian populations. **low risk** in Asians and Africans. 1-type I psoriasis(early onset): 20–30 ,more likely to be familial, a severe clinical course ,**HLA-Cw6, B13 and B57** 2-type II psoriasis(Late onset): ages 50 to 60 ,**HLA-Cw2 and B27** - **Koebner phenomenon** **parakeratosis (nuclei retained in the horny layer)**	F=M , 30-60 years No racial predispositions. - Started flexor surface of the limbs - After a week or more, a generalized eruption develops with maximal spreading within 2-16 weeks Characteristic **Wickham stria**, Ulcerated oral lesions may have a higher incidence of **malignant transformation(squamous cell carcinoma)** **Koebner phenomenon**	children and young adult Increased incidence in spring and autumn more common in women Begins with a herald spot/patch over trunck and proximal limbs Usually a salmon-colored macule with a collarette of fine scale Within the next 1-2 weeks, a generalized exanthem usually appears
Management	Educating the patient and family Psychosocial support Quit Smoking and decrease weight -Emollients: -Keratolytics: **urea and salicylic acid** - Topical Corticosteroids: -Vitamin D Analogs(**calcipotriol**) Alone or with Topical Corticosteroids: -Calcineurin Inhibitors: **tacrolimus** and **pimecrolimus** -Anthralin: Dithranol -Tazarotene, Tar	**self-limited disease that usually resolves** **within 8-12 months** - Anti-histamine - topical steroids, particularly class I or II ointments - systemic steroids - Oral acitretin - Photo-therapy	Reassurance that the rash will resolve -Relief of pruritus -Oral antihistamines -oral antiviral therapy -Topical or Systemic steroids -Ultraviolet B (UV-B) light therapy

Done By:

Yasmine Alshehri

Nawal Asiry

Revised by:

Nawal Asiry