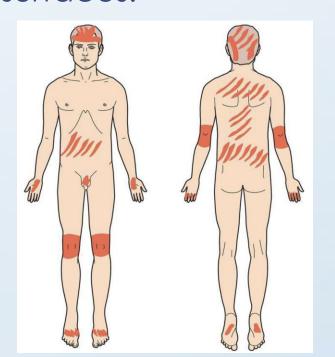


Psoriasis

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Definition

Psoriasis is a chronic noncontagious autoimmune disorder that
causes a rapid buildup of skin cells. This buildup of cells leads to
the formation of well demarcated erythematous plaques with
scaling on the surface of the skin, most commonly on the
extensor surfaces.



• Systemic disease, may involve the joints (Psoriatic Arthritis [PA]) Associated with T2DM, CVD, metabolic syndrome, NAFLD, depression

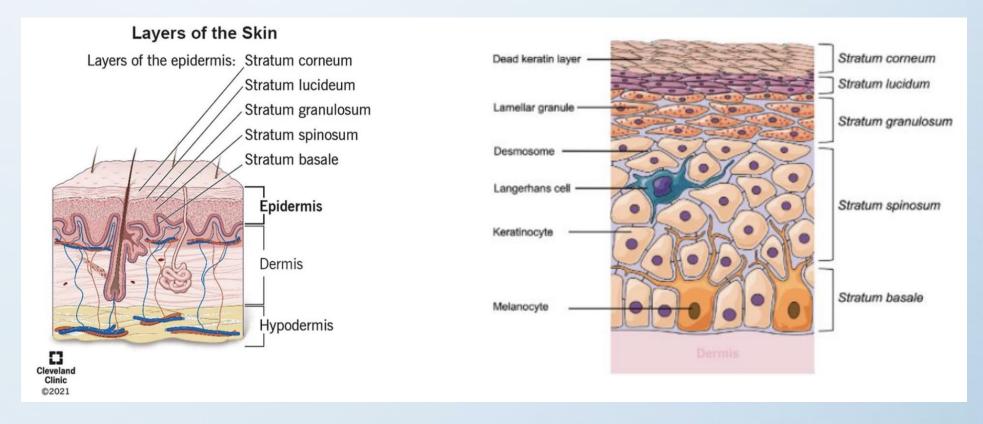
- There is no clear gender predilection
- Psoriasis can begin at any age, though it is less common in children than adults
- Median age is 28 years; can occur at any age
- Multifactorial; genetic & environmental factors play a role
- High Genetic association

Triggers

- Infections, such as strep throat or skin infections (S. aureus)
- Weather, especially cold, dry conditions
- Injury to the skin, such as a cut or scrape, a bug bite, or a severe sunburn
- Stress
- Vaccination
- Smoking and exposure to secondhand smoke
- Heavy alcohol consumption
- Certain medications (β-blockers, lithium, anti-malarial drugs, interferons, angiotensin-converting enzyme inhibitors, terbinafine, tetracycline, nonsteroidalanti-inflammatory drugs)
- Rapid withdrawal of oral or systemic corticosteroids
- Intrinsic factors: (obesity, DM, stress, hypertension)

Pathophysiology

 The pathogenesis of psoriasis is complex & the development of psoriasis is multifactorial with multiple potential susceptibility factors in a genetically at-risk individual.



- This combination of susceptibility factors and genetic predisposition results in an interactive web of immunecells/chemical cytokines impacting on skin cells and leading to disease.
- It's largely accepted that the disease is mediated by the dysregulation of T-helper lymphocytes (Th1/Th17)
- Keratinocytes are the skin cells that predominate in the epidermis; they grow from the basal layer and slowly migrate to the surface.
- In normal skin, this process of cell turnover takes about 23 days;
 however, in psoriasis cell turnover rapidly accelerated, taking only 3–5
 days for cells to reach the surface and accumulate in large numbers
 (hyperkeratosis).

 Keratinocytes normally lose their nuclei as they move to the skin surface; however, in psoriasis they move so quickly that the cells retain their nuclei throughout the epidermis, seen as parakeratosis histologically.

 This rapid turnover and failure of proper maturation result in defective keratinocytes, which are poorly adherent and easily scraped off ('Auspitz sign') revealing underlying dilated blood

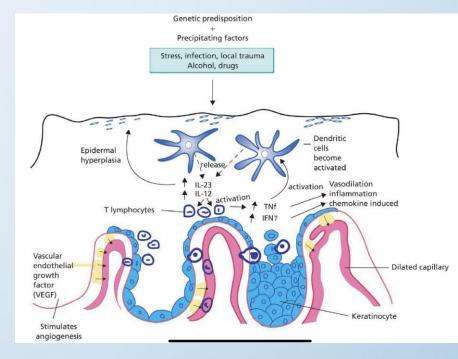
vessels.



Clinical Presentation

- Plaques of psoriasis are well defined raised lesion highly infiltrated with CD3+ T-cells and CD11c+ dendritic cells which produce pro-inflammatory cytokines
- Scaling: scratching surface
- Erythema, (especially in erythrodermic psoriasis where >90% of body surface is affected)
- Pustules: deep-seated yellowish sterile pustules (palmo-plantar pustular psoriasis)











Classification

- Plaque Psoriasis (Psoriasis vulgaris)
- Guttate Psoriasis
- Psoriasis Inversa
- Pustular Psoriasis
- Erythrodermic Psoriasis

PLAQUE PSORIASIS

- Most common type
- Raised inflamed red skin, covered by silvery scales
- Scalp, extensor elbows, knees, and gluteal cleft are commonsites for involvement
- Symmetrically distributed



GUTTATE PSORIASIS

- Characterized by the abrupt appearance of multiple small, psoriatic papules and plaques
- The papules and plaques are usually less than 1 cm in diameter
- The trunk and proximal extremities are the primary sites of involvement
- Typically occurs as an acute eruption in a child or young adult with no previous history of psoriasis
- Strong association between recent infection (usually streptococcalpharyngitis)



PSORIASIS INVERSA (FLEXURAL PSORIASIS)

- Presentation involving the intertriginous areas, including the inguinal, perineal, genital, intergluteal, axillary, or inframammary regions.
- Well-demarcated, smooth, shiny plaques with absent or minimal scale that are often misdiagnosed as intertriginous fungal or bacterial infection



PUSTULAR PSORIASIS

- Form of psoriasis that can have lifethreatening complications.
- Local(palmo-plantar) vs generalized
- This form of psoriasis can be associated with malaise, fever, diarrhea, leukocytosis, and hypocalcemia
- Renal, hepatic, or respiratory abnormalities and sepsisare potential complications.



ERYTHRODERMIC PSORIASIS

- Uncommon manifestation but very severe
- May involve 90% of total body surface
- Affected patients are at high risk for complications related to loss of adequate barrier protection, such as infection (including sepsis) and electrolyte abnormalities secondary to fluid loss



Figure 2.19 Erythrodermic psoriasis.

NAIL PSORIASIS

most often noted after the onset of psoriatic skin lesions (but not

nessecerely)

Types:

- Onycholysis
- Subungal hyperkeratosis
- Pitting
- Beau's lines
- Splinter hemorrhages









DIAGNOSIS:

1) History taking

- Worsening of a long-term erythematous scaly area.
- Sudden onset of many small areas of scaly redness.
- Recent streptococcal throat infection, viral infection or use of antimalarial drug.
- Family history of similar skin condition.
- Pruritus (especially in eruptive, guttate psoriasis).
- febrile (except in pustular or erythrodermic psoriasis in which the patient may have high fever).
- Dystrophic nails
- Long-term rash with recent presentation of joint pain.
- Joint pain without any visible skin findings.



Reported causes of psoriasiform drug eruptions

Common

Psychoactive drugs (lithium)

Antihypertensives (beta blockers)

Antimalarials (chloroquine, hydroxychloroquine, quinidine)

Nonsteroidal anti-inflammatory drugs, antibiotics (tetracycline)

Uncommon

Tumor necrosis factor inhibitors (infliximab, adalimumab, etanercept, certolizumab pegol)[1,2]

Antifungals (terbinafine)[3]

Rare

Antihypertensives (clonidine)

Antiarrhythmics (digoxin, amiodarone)

Antiepileptics (carbamazepine, valproic acid)

Psychoactives (fluoxetine)

Antihypertensives/diuretics (captopril, chlorthalidone, diltiazem, nifedipine, nicardipine, [4] acetazolamide)

Antibiotics (penicillin, amoxicillin, ampicillin)

Opioids (morphine)

Anesthetics (procaine)

Antihistamines (cimetidine, ranitidine*)

Heavy metals (gold, mercury)

Hormonal agents (oxandrolone, progesterone)

Fibrates (gemfibrozil)

Antithyroid (potassium iodide)

Cytokines/cytokine inducers (GM-CSF, imiquimod)

GM-CSF: granulocyte macrophage colony-stimulating factor.

* Ranitidine has been withdrawn from the United States market.

2) PHYSICAL EXAMINATION

- Findings depend on the type of psoriasis present.
- The most common skin manifestations are scaling, salmon-colored/erythematous, macules, papules, and plaques.

Typically, the macules are seen first. The area of skin involvement varies with the form of psoriasis.

- Nail pitting.
- Onycholysis.
- Scalp psoriasis.
- Psoriatic arthritis.

3) LABS

- CBC, LFTs, BUN and pregnancy test.
- Test result for rheumatoid factor (RF): negative.
- Erythrocyte sedimentation rate (ESR): usually normal (except in pustular and erythrodermic psoriasis).
- Uric acid: may be elevated in psoriasis (especially in pustular psoriasis) causing confusion with gout in psoriatic arthritis.
- Fluid from pustules is sterile with neutrophilic infiltrate.
- Perform fungal studies (this is especially important in cases of hand and foot psoriasis that seem to be worsening with the use of topical steroids.)

MANAGEMENT

Treatment modalities are chosen on the basis of disease severity, relevant comorbidities, patient preference (including cost and convenience), efficacy, and evaluation of individual patient response.

- 1) BSA: <10% topical, >10% systemic
- 2) PASI score: <10, >10
- 3) DLQI score

MANAGEMENT

- •It may be helpful for the clinician to touch the, patient's psoriasis lesions with an ungloved hand, when appropriate, to communicate physically that the skin disorder is neither repulsive nor contagious.
- Clinicians should lay out reasonable aims of treatment, making it clear to the patient that the primary goal of treatment is control of the disease. Although treatment can provide patients with high degrees of disease improvement, there is no definite cure for psoriasis.

TREATMENT

- 1- Topical –for limited disease-
- Emollients
- Corticosteroids
- Topical vitamin D analogs
- Topical vitamin A derivatives
- Tar

- 2- Systemic therapies
- Methotrexate
- Cyclosporine
- Biologic treatment
- Acitretin
- Ultraviolet light (phototherapy is administered 3 times per week during the treatment phase)



CANCER RISK

A concern with PUVA (psoralen + UV light) is an increased risk of non melanoma skin cancer and melanoma. Ongoing monitoring is indicated in patients who have received prolonged courses of PUVA.

* phototherapy is contraindicated in patients with a history of melanoma or extensive non melanoma skin cancer.

Thank U