BMJ Best Practice

Psoriasis

Straight to the point of care

Last updated: Sep 30, 2022

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Summary

Psoriatic lesions are red, inflamed, silvery-white scaly, and circumscribed papules and plaques; often affecting elbows, knees, extensor limbs, and scalp, and, less commonly, nails, ear, and umbilical region.

Psoriasis is a multifactorial disease with a genetic basis. Exacerbations of disease may be related to infection, alcohol, medications, stress, and intercurrent illness.

Diagnosis is usually clinical.

Mild or limited psoriasis is treated with topical corticosteroids and/or vitamin D analogues.

Moderate to severe and/or extensive psoriasis may require phototherapy, and systemic agents such as methotrexate, ciclosporin, acitretin, or biological agents.

Definition

Psoriasis is a chronic inflammatory skin disease characterised by erythematous, circumscribed scaly papules, and plaques. It can cause itching, irritation, burning, and stinging. Although approximately 30% of people with cutaneous psoriasis also have psoriatic arthritis, this topic only discusses cutaneous psoriasis.[1]

Epidemiology

More than 80% of countries in the world lack information on the epidemiology of psoriasis.[4] Published data report prevalence ranging from 0.09% to 11.43%.[5] [6]

A systematic analysis and modelling study reported psoriasis incidence of 30.3 per 100,000 person-years to 321.0 per 100,000 person-years, and prevalence of 0.14% to 1.99%.[4] Incidence and prevalence were relatively low in regions with young populations (e.g., south Asia and sub-Saharan Africa) and higher in regions with older populations (i.e., high-income regions).[4]

In the US, a population-based cross-sectional study (using National Health and Nutrition Examination Survey data) suggested that psoriasis affects 3% of the adult population (>7.5 million adults).[7] Psoriasis prevalence was similar between women and men.[7]

A UK population-based cohort study estimated psoriasis incidence to be 129 per 100,000 person-years.[8]

Peak incidence increases up to 39 years of age then decreases; there may be a second peak between 50 to 59 years or 60 to 69 years.[4]

Psoriasis is uncommon in children. Patients presenting at a younger age are more likely to have an affected parent and to demonstrate human leukocyte antigen association.[9]

Aetiology

Factors including genetics, immunology, and infection may contribute.

Genetics

Psoriasis heritability is between 60% to 90%, which is higher than most other multifactorial diseases.[10] Studies of monozygotic twins, linkage studies, and genome-wide association studies provide evidence that psoriasis has a genetic predisposition.[11] [12] [13]

Most genes associated with psoriasis are involved in the immune response, and relatively few encode for skin-specific proteins. HLA-Cw6 encodes an antigen involved in T-cell activation. There is an increased prevalence of HLA-Cw6 in people with psoriasis compared with controls. Tumour necrosis factor (TNF)-alpha, another protein-coding gene associated with innate and adaptive immune response, is implicated in the aetiology of psoriasis. Pathogenic involvement of genes related to Th17-cell activation have been demonstrated in people with psoriasis.[12] [13] [14] [15] [16]

Immunology

Psoriasis is believed to be triggered by external insults in genetically susceptible individuals. Recognised triggers include trauma, infection, and medications (e.g., beta-blockers, lithium). Following initiation by an insult, the host DNA forms complexes with antimicrobial peptides released from keratinocytes (skin cells), which results in inflammation and keratinocyte proliferation to cause disease presentation.[16] [17] [18]

Infection

Guttate psoriasis is often observed subsequent to upper respiratory infection, such as streptococcal pharyngitis. It may also be associated with HIV infection. Viral infection, immunisation, and any intercurrent illness have been linked to flares of guttate and plaque psoriasis.[16] [19]

Pathophysiology

Psoriasis is a hyperproliferative disorder, involving a complex cascade of inflammatory mediators. Mitotic activity of basal and suprabasal cells is significantly increased, with cells migrating from the basal layer to the stratum corneum in just a few days. The silver scale on the surface of psoriasiform lesions is simply a layer of dead cells.[16] [20]

Early clinical studies of TNF inhibitors demonstrated the important role of these cytokines in psoriasis, prompting the condition to be regarded as primarily driven by T-helper-1 (Th-1) cells.[21] However, evidence supports the pivotal involvement of a different immunological axis underlying the pathogenesis of psoriasis; namely, T-helper cells producing interleukin (IL)-17 and IL-23.[22] [23] [24] IL-17 and IL-23 expression is increased in the serum, lesional skin, uninvolved skin, and even tear liquid of patients with psoriasis compared with patients without psoriasis. These cytokines are now considered to be central to the pathogenesis of psoriasis as demonstrated by the efficacy of therapeutics inhibiting IL-17 or IL-23 pathways.

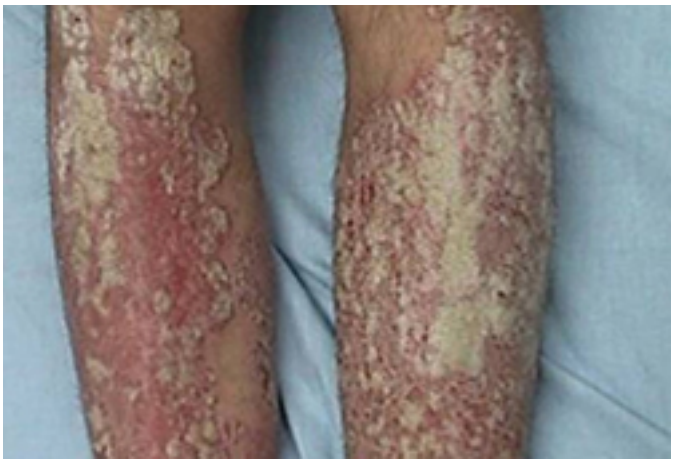
The presence of T-cells reacting against autoantigens has been detected; three autoantigens have been identified: LL37, ADAMTS-like protein 5, and phospholipase-2-derived products.[25] [26] [27] [28]

Classification

International Psoriasis Council[2]

1. Plaque psoriasis

- Raised inflamed plaque lesions with a superficial silvery-white scaly eruption. The scale may be scraped away to reveal inflamed and sometimes friable skin beneath.



Plaque psoriasis on legs

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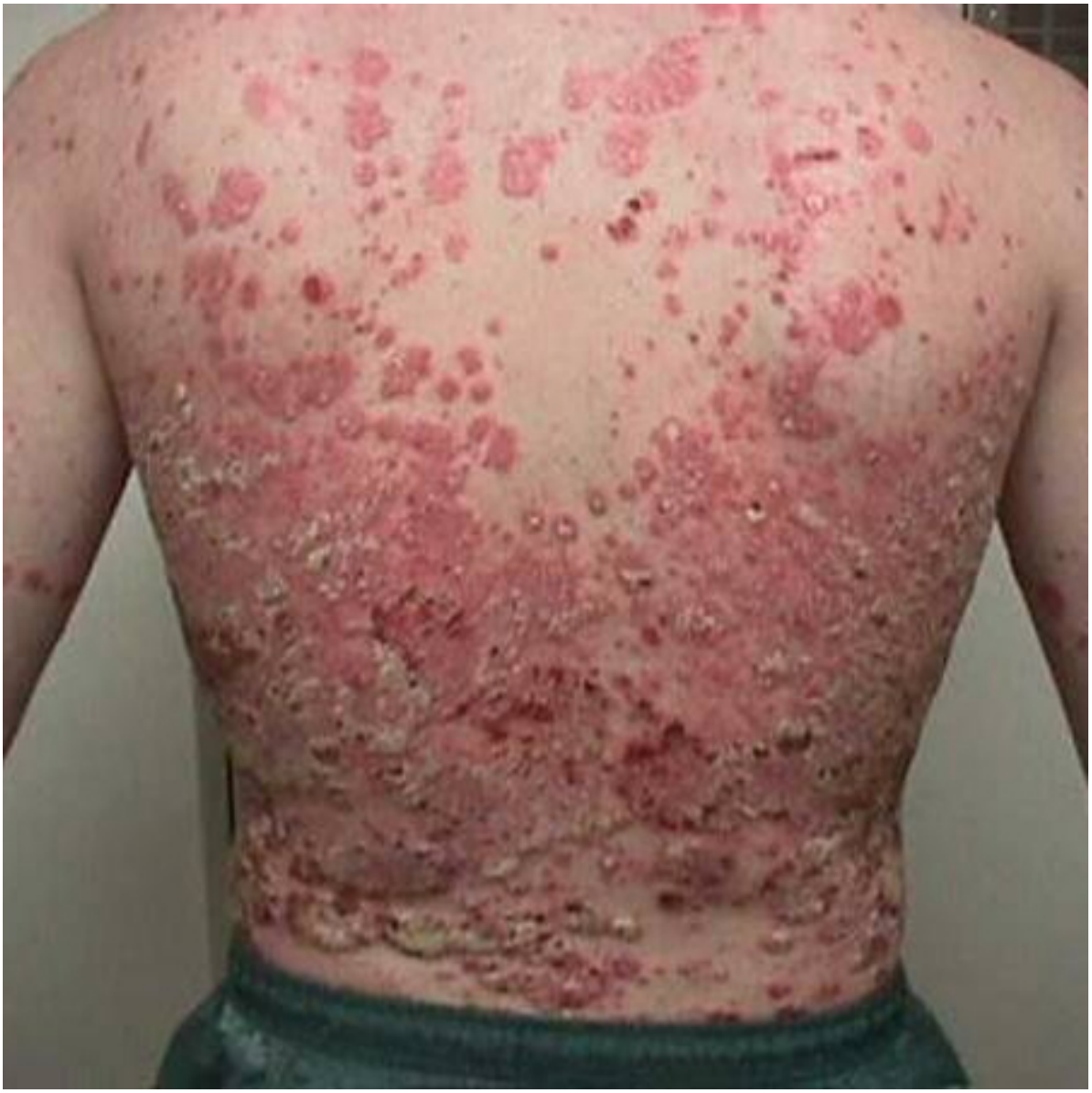

Plaque psoriasis on back

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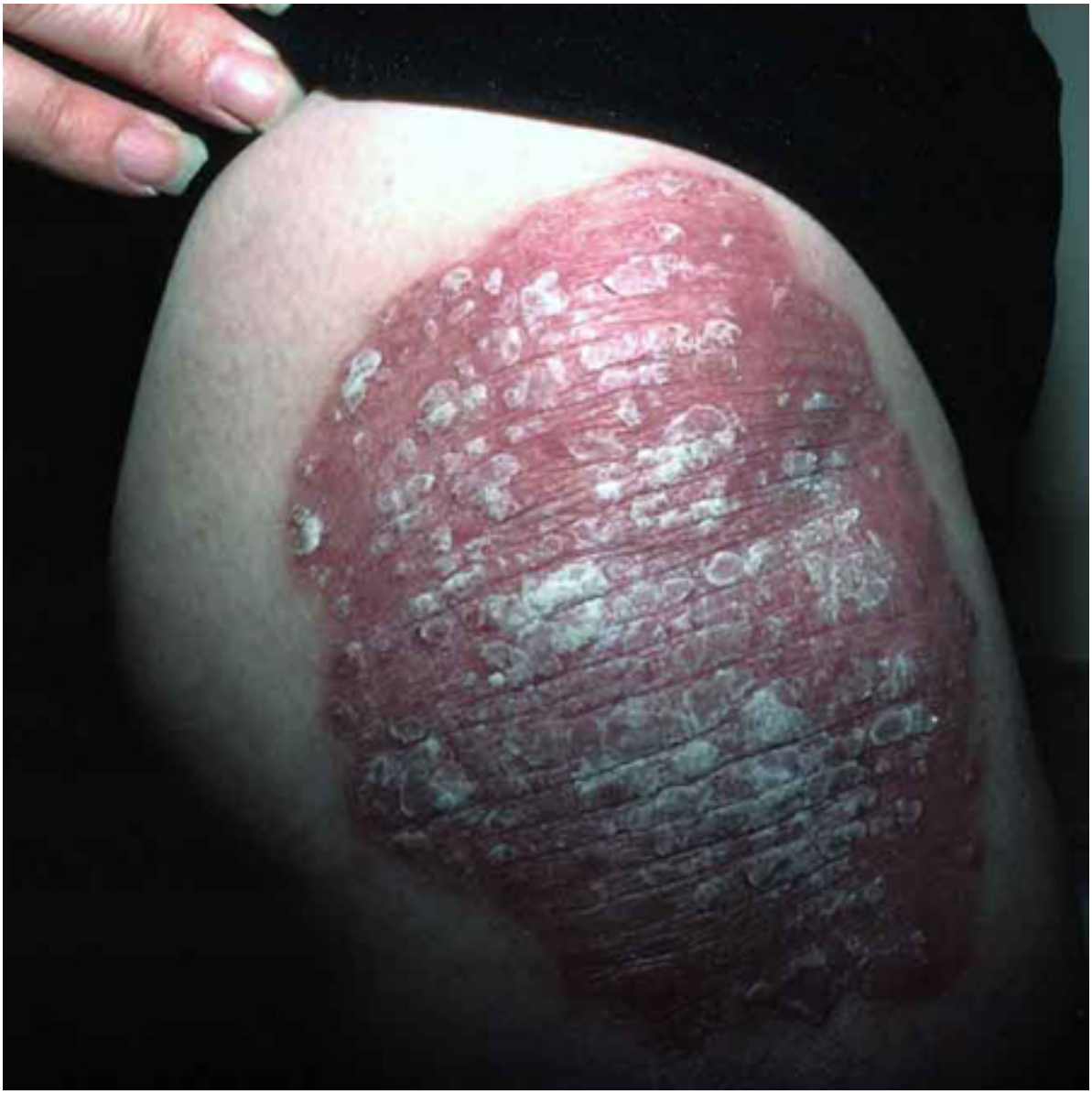

Plaque psoriasis on knee

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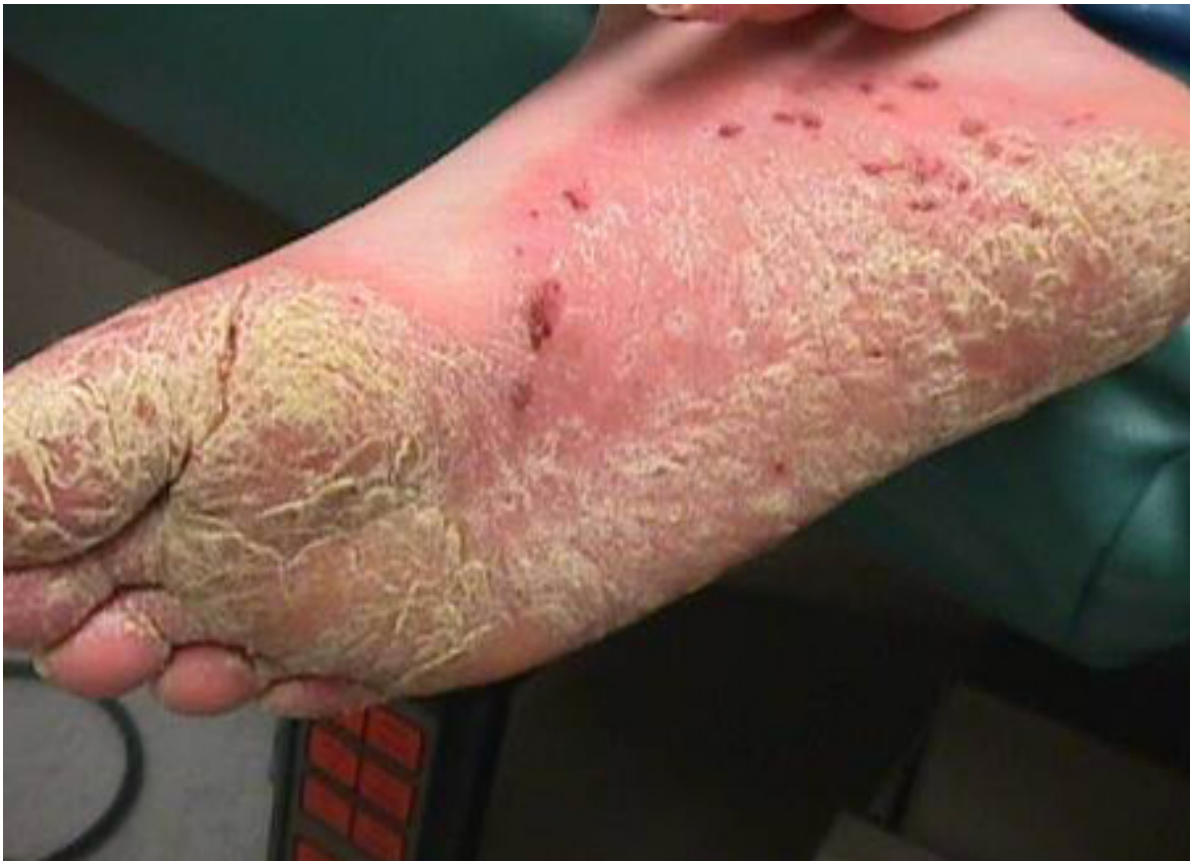

Plaque psoriasis on foot

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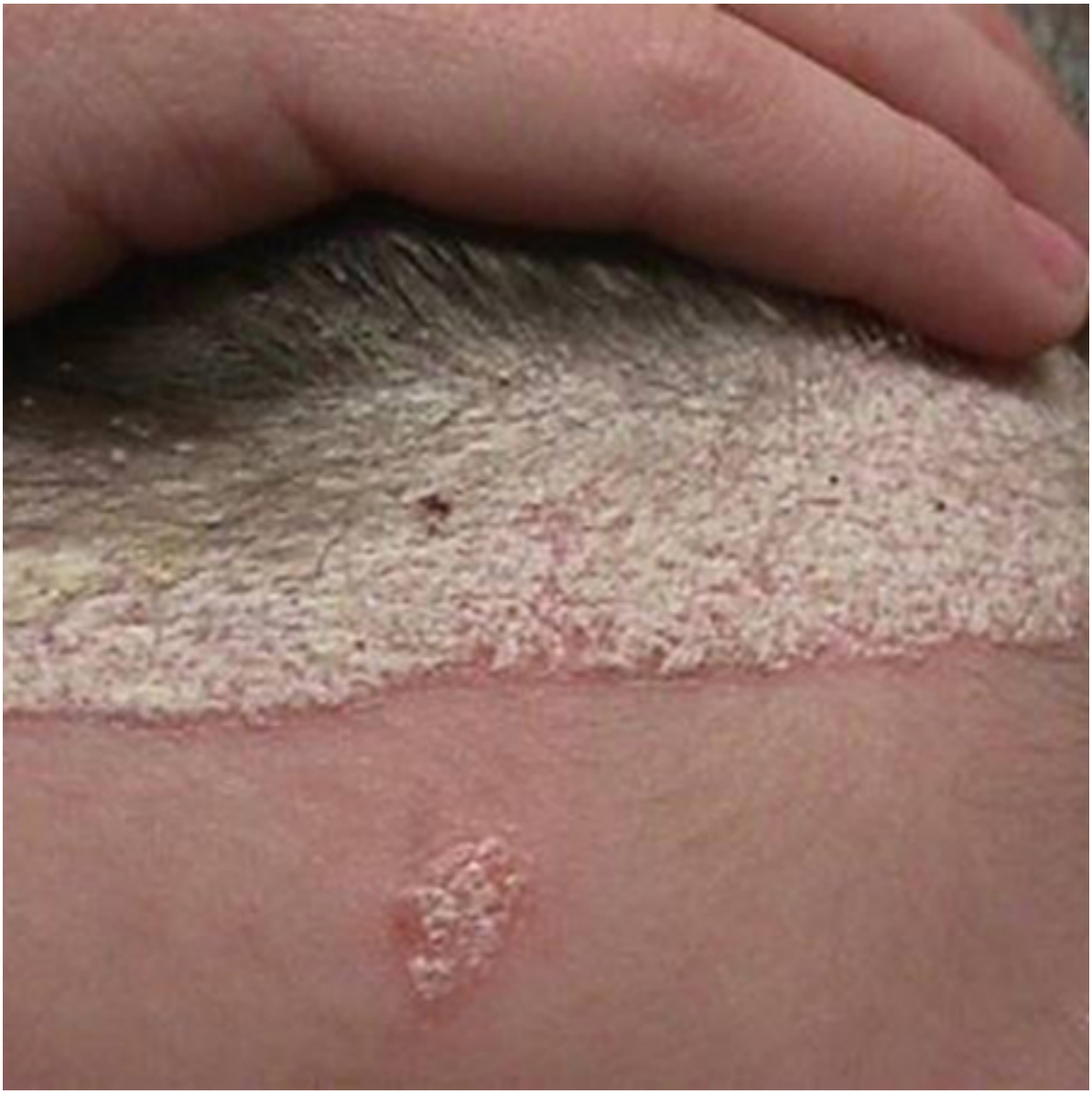

Plaque psoriasis on scalp

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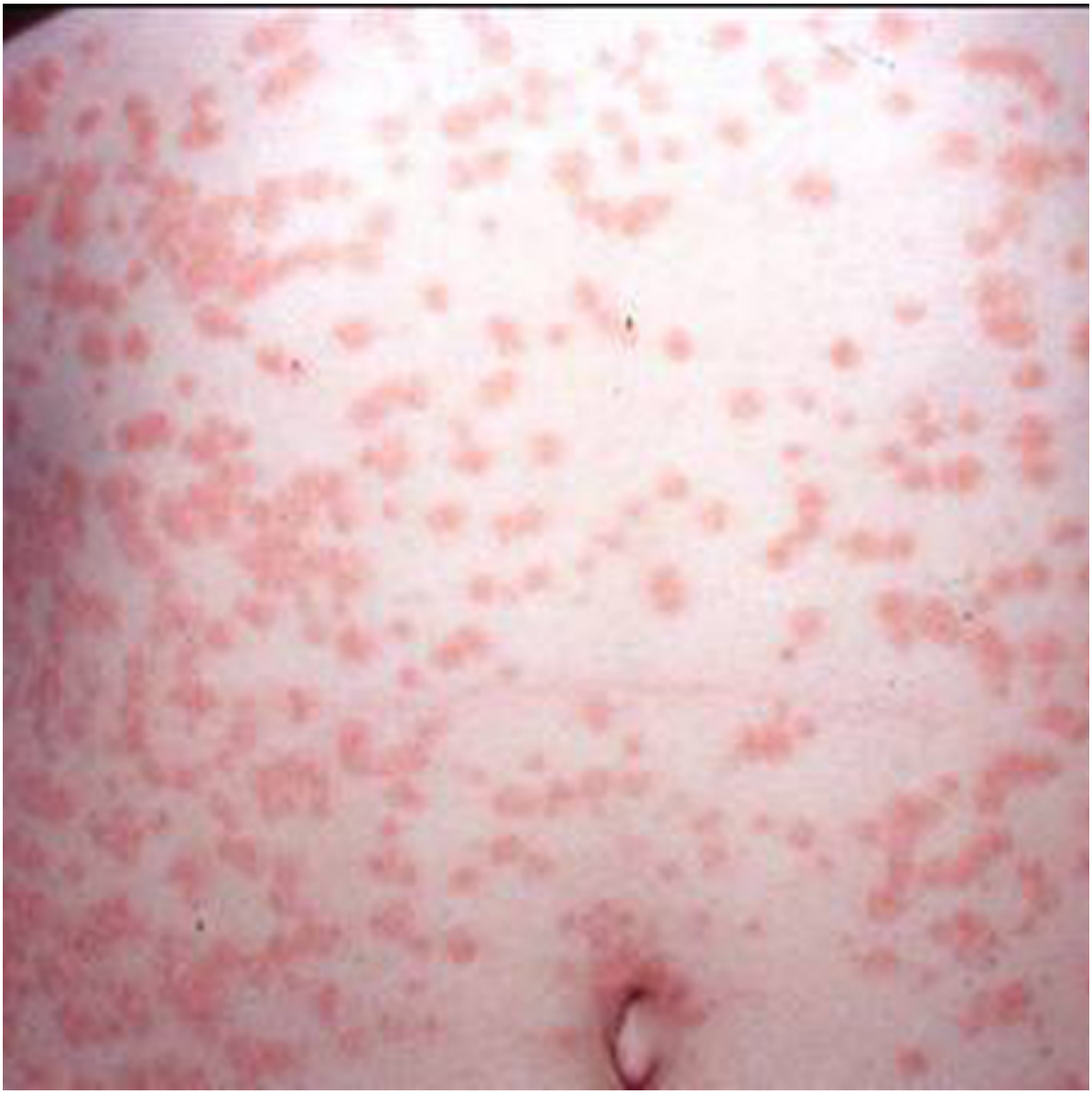
2. Guttate psoriasis

- Widespread, erythematous, fine, scaly papules (water drop appearance) on trunk, arms, and legs. The lesions often erupt after an upper respiratory infection.



Guttate psoriasis

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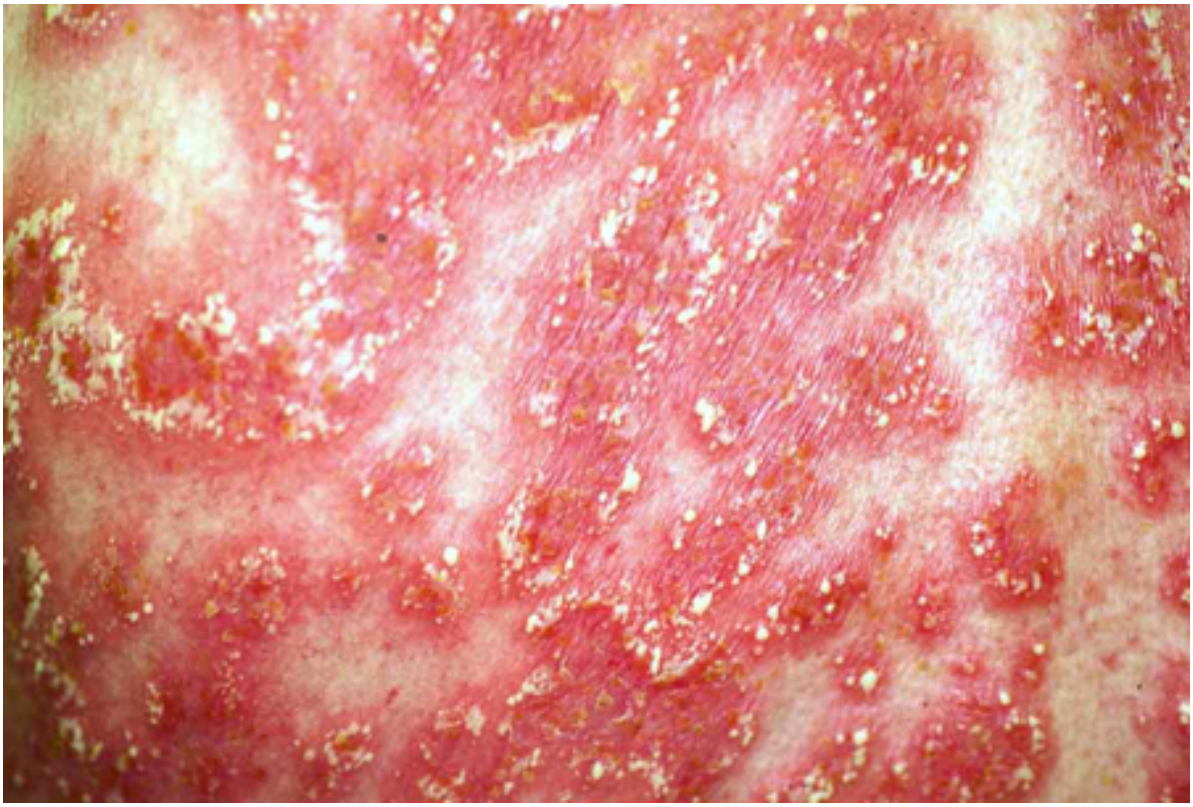
3. Pustular psoriasis

- Acute generalised pustular psoriasis (von Zumbusch): rare, severe, urgent.
- Palmoplantar pustulosis: chronic involvement of hands and feet.



Pustular psoriasis

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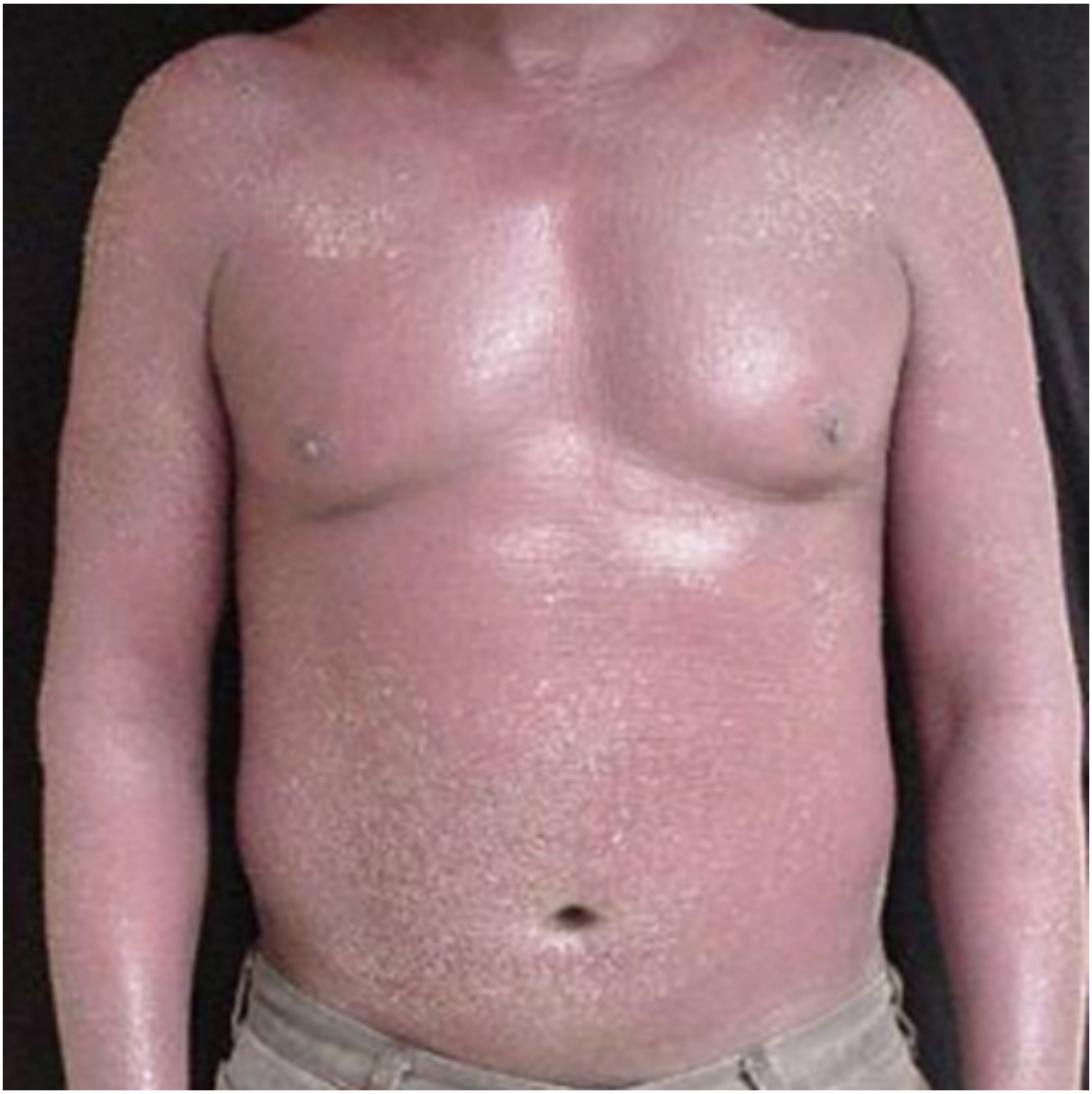
4. Erythroderma (erythrodermic psoriasis)

- Generalised erythema with fine scaling. It is often associated with pain, irritation, and sometimes severe itching.



Erythroderma

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5. Psoriatic arthritis

- Unique in that, in addition to skin lesions, there is joint involvement that causes inflammatory damage and deformity. It affects approximately 20% of people with psoriasis, as reported by a 2019 epidemiological study.[3]
- Most people with nail psoriasis have psoriatic arthritis. The arthritis most commonly involves fingers, hands, toes, and feet, and, less commonly, knees, elbows, and axial and sacroiliac joints.
- Cutaneous psoriatic lesions precede arthritis in 70% of cases.
- Psoriatic arthritis causes stiffness, inflammation, pain, and progressive and permanent joint damage. The arthritis is asymmetrical in around 50% of cases.

Psoriatic arthritis

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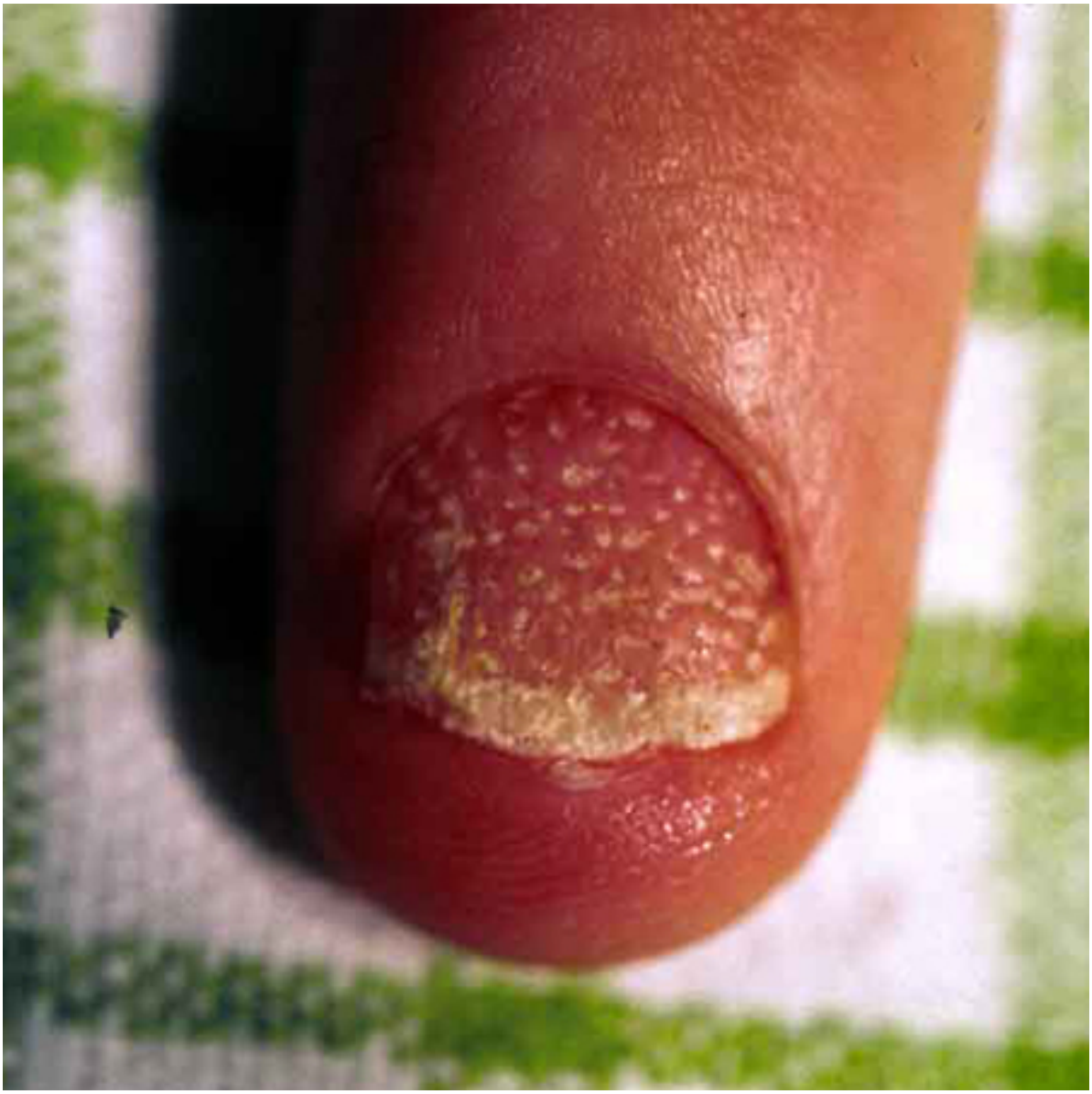

Nail psoriasis - pitted nails

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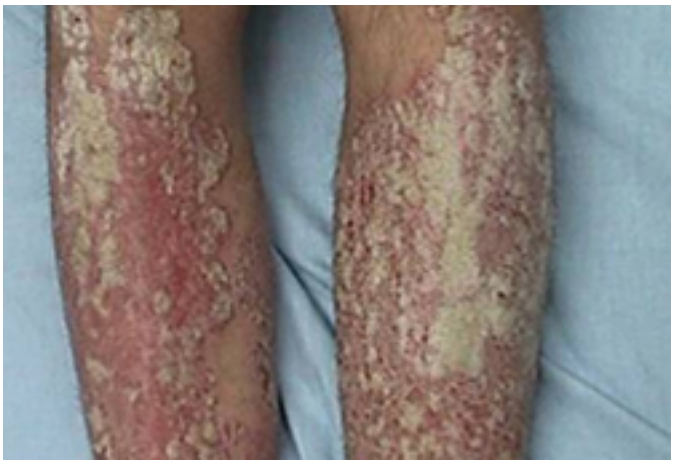
6. Keratoderma blennorrhagicum (reactive arthritis)

- Reactive immune disorder characterised by psoriasiform plaques, urethritis/cervicitis, conjunctivitis, and arthritis.
- Circular, scaly, scalloped-edged hyperkeratotic psoriasiform papules and plaques, which is sometimes painful and pustular (at the centre of lesions), appears on soles and toes, and, less commonly, legs, palms, scalp, and penis.
- Occurs in genetically susceptible people with HLA-B27 following an infection (particularly *Yersinia enterocolitica* and *Yersinia pseudotuberculosis*).

Case history

Case history #1

A middle-aged man with a known history of psoriasis presents with white scaly papules and plaques on his elbows, extensor arms, knees, and shins. In the past 6 months, these lesions have become much worse and have started to appear on his waist and hip. Scaly and flaky eruptions are also present on his scalp, ears, and eyebrows. He describes the lesions as being itchy and irritating. He is a heavy smoker and has been unsuccessful in a previous attempt at smoking cessation.



Plaque psoriasis on legs

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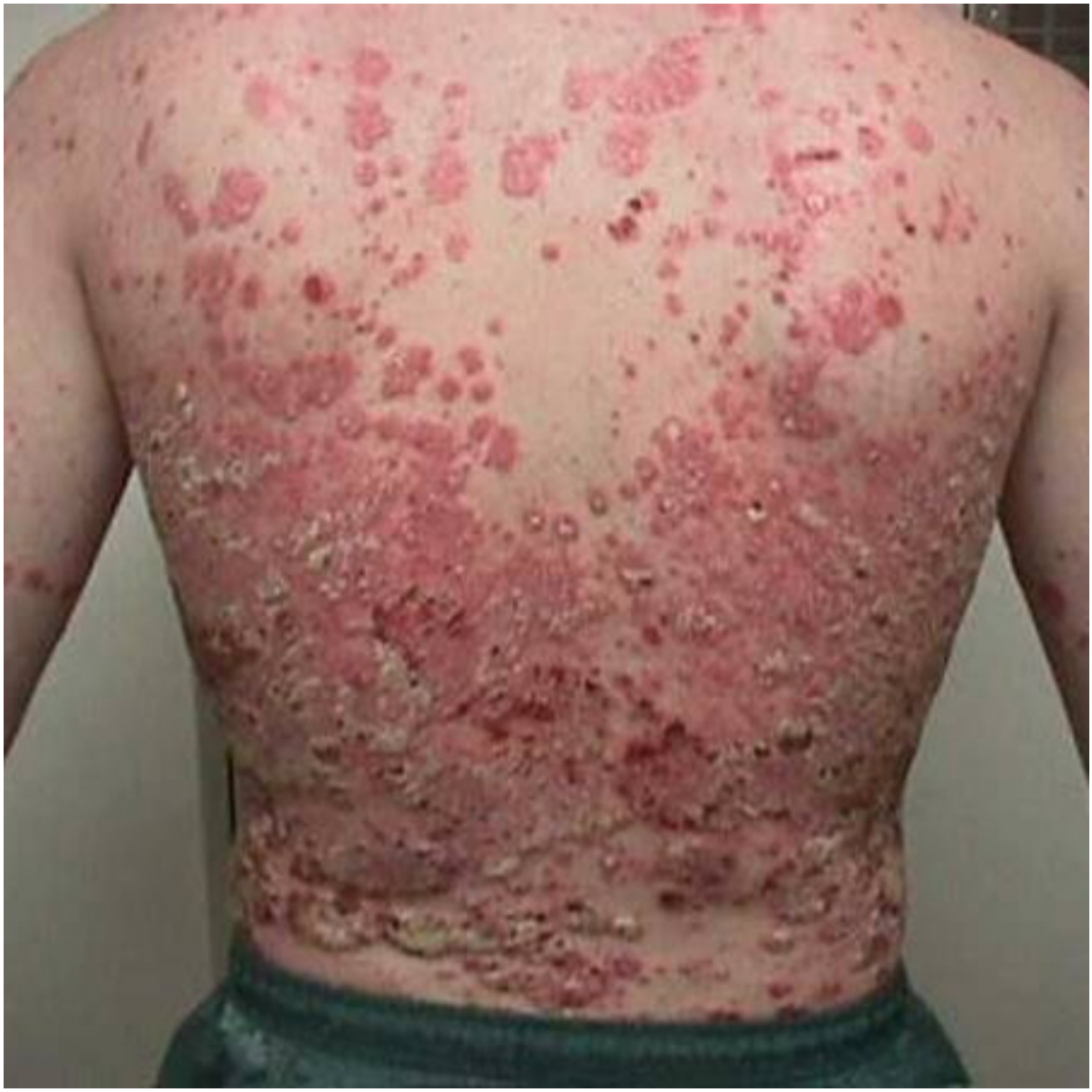

Plaque psoriasis on back

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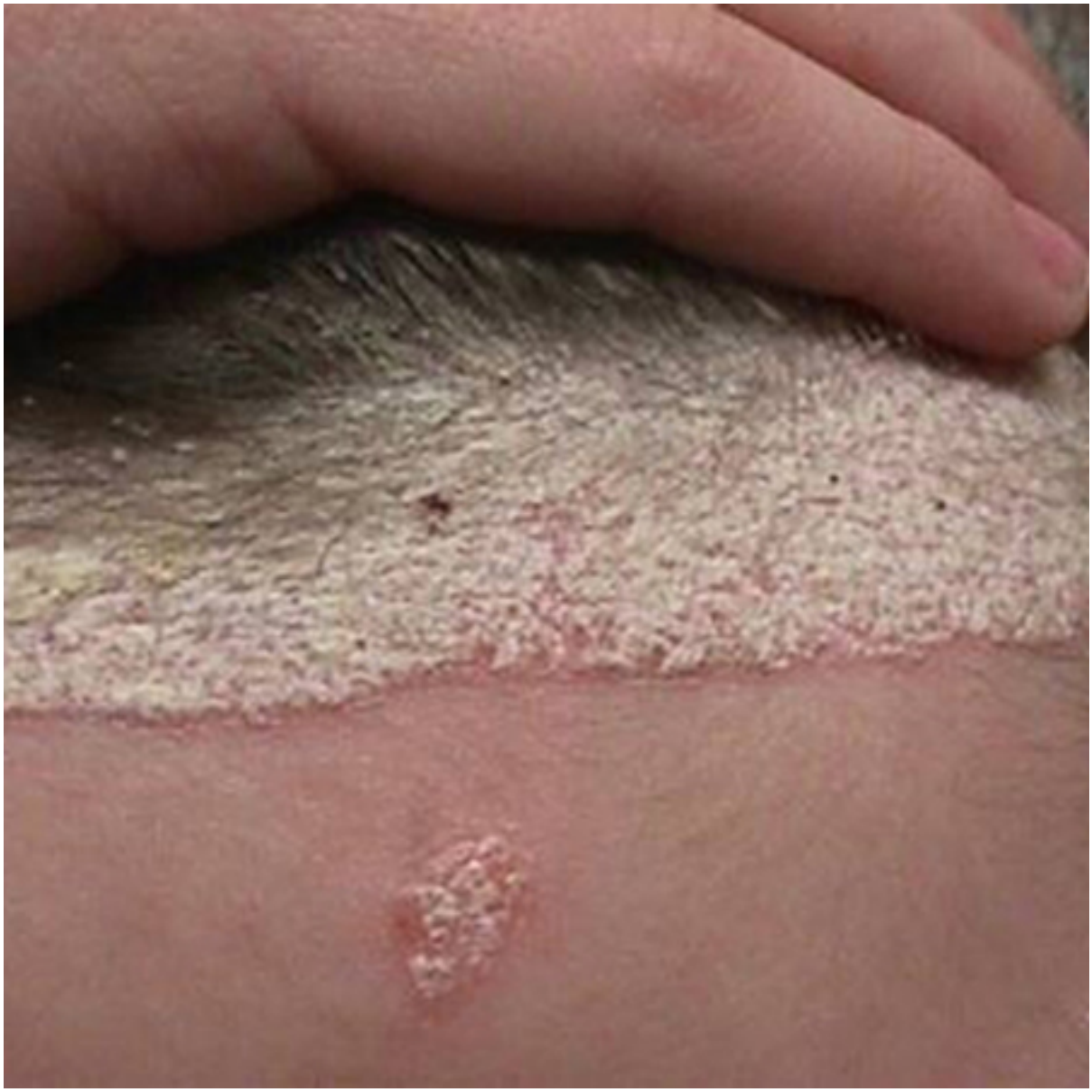

Plaque psoriasis on scalp

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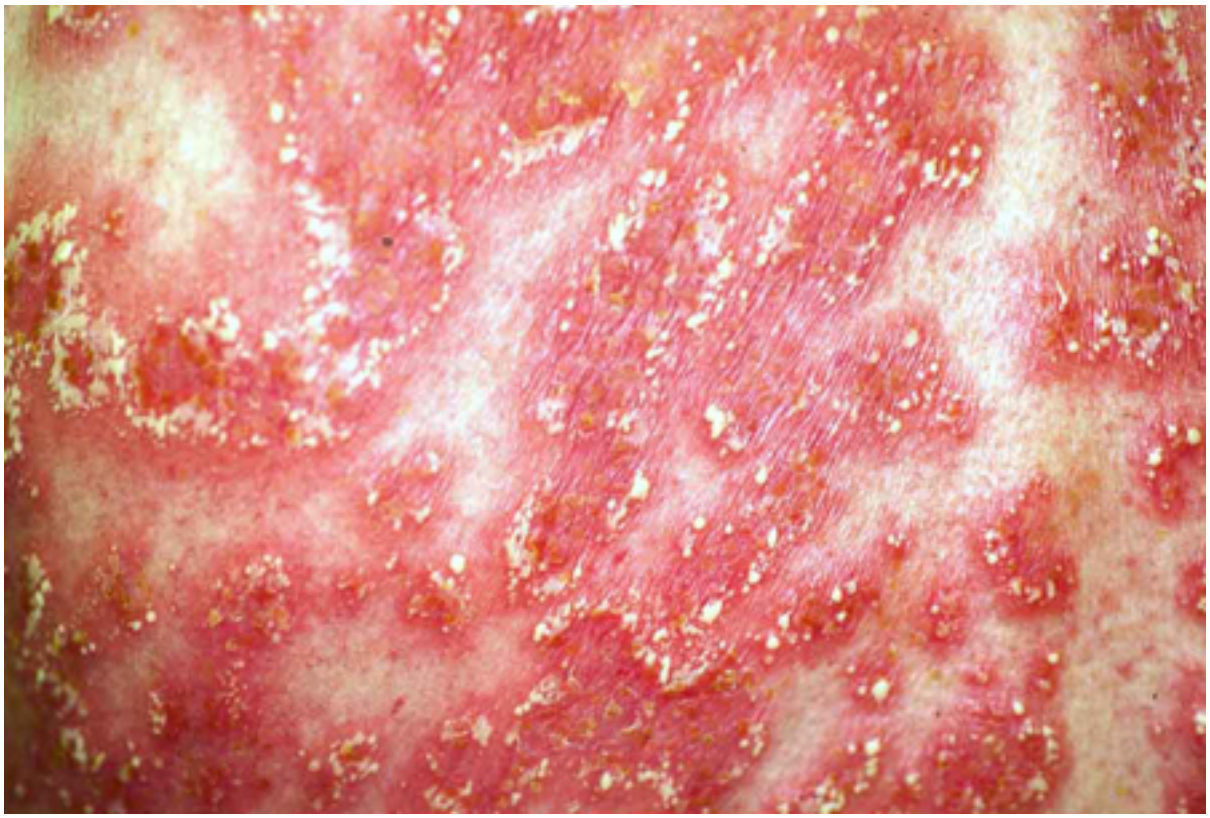
Case history #2

A young woman without a known history of psoriasis or skin disorder had a sudden onset of wide-spreading, white-scaly, oval- to round-shaped erythematous papules, which have been present for 2 weeks. Lesions are primarily on her trunk but also appear scattered on her arms and legs. She recalls a recent episode of sore throat and upper respiratory tract infection. A short course of antibiotics seemed to help, but did not clear the lesions.

Other presentations

Inverse psoriasis may present in the genital skin, the gluteal cleft and skin folds of the axillae, and under breasts. Palmar plantar psoriasis presents on palms and soles of feet. Pustular psoriasis presents as

sudden-onset disease with generalised pustulosis. However, the pustules carry no bacteria and patients are not febrile. Psoriatic arthritis is often insidious, with stiffness and inflammation around finger and toe joints only. The associated cutaneous lesions may be very minor, which makes it difficult to establish the diagnosis of psoriasis.



Pustular psoriasis

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Approach

Characteristic history and examination findings are often sufficient to diagnose the condition. Skin biopsy is reserved for atypical cases when lack of treatment response brings the diagnosis into question.

History

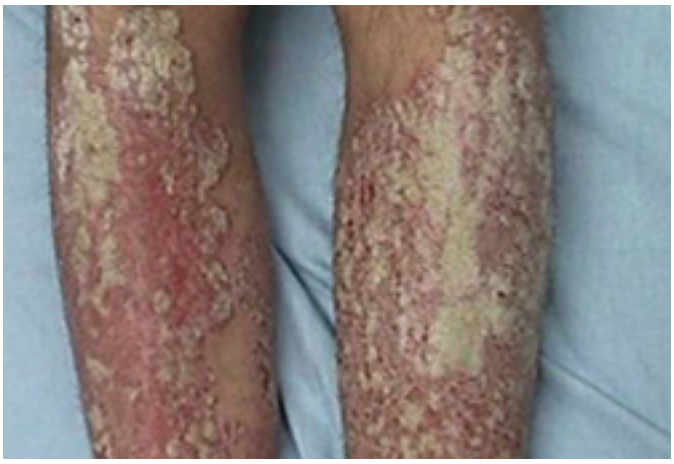
Most people with psoriasis have a positive family history.[11] [16]

Psoriasis usually begins as 1 or 2 limited lesions on elbows or scalp. In the majority of cases, psoriasis remains a limited disease. However, in some people, it may spread to involve other body sites over time.

Psoriasis has a fluctuating course of flares and remission but seldom completely subsides. Patients may describe the skin as highly sensitive, and itching can be severe. Bleeding may occur if the lesions are scratched. The skin can be painful, particularly if joints are involved. It may be aggravated by environmental, emotional, or infectious factors. As part of the diagnostic work-up, determine what, if any, therapies have been used by the patient and how effective they have been. Abruptly stopping corticosteroid therapy for psoriasis or adding a known irritant medication might lead to sudden worsening.

Physical examination

The typical appearance of psoriasis is of erythematous, circumscribed, scaly papules and plaques on elbows, knees, extensor surfaces of limbs, and scalp. To help differentiate from eczema, examine the scalp, behind the ears, and the nails for pitting.



Plaque psoriasis on legs

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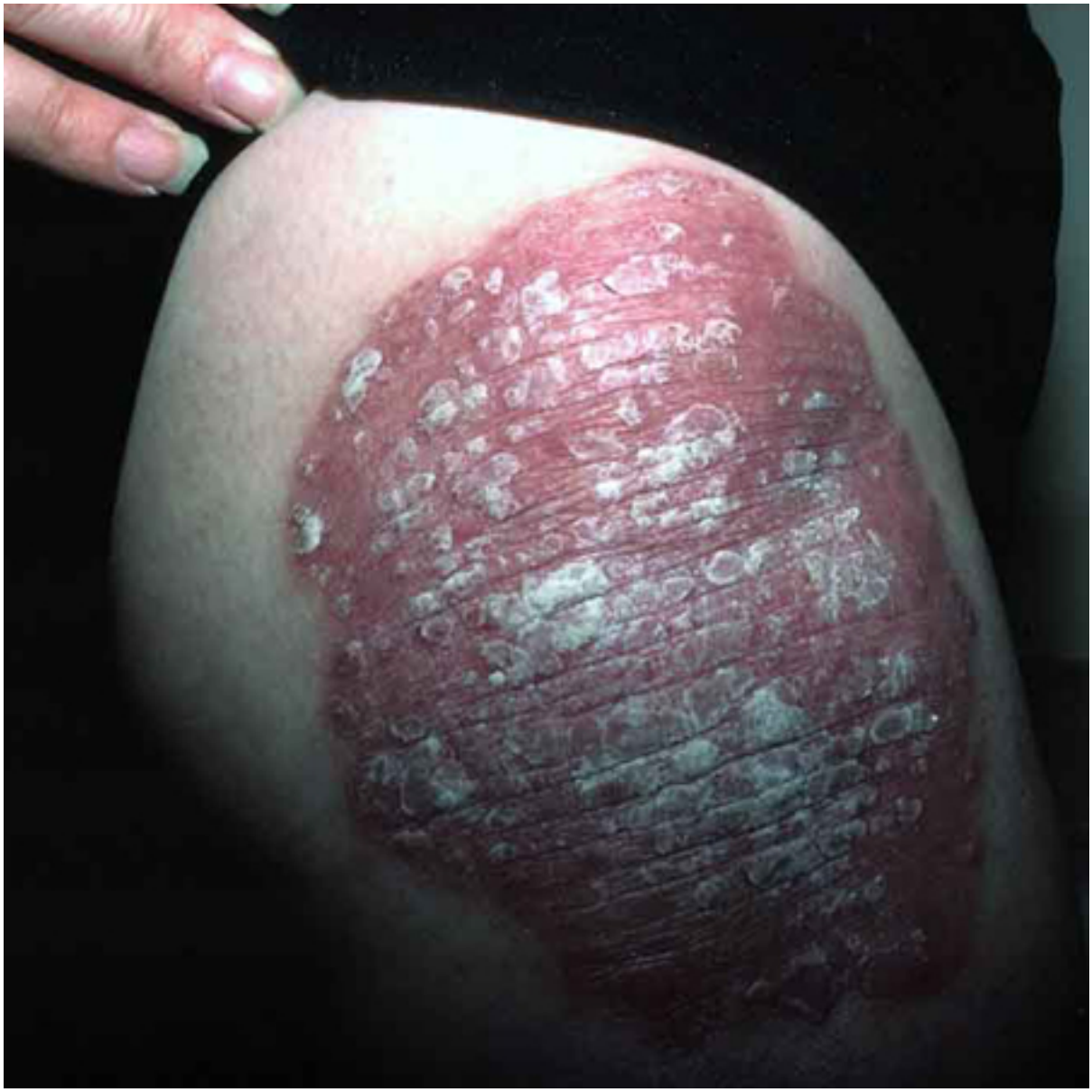

Plaque psoriasis on knee

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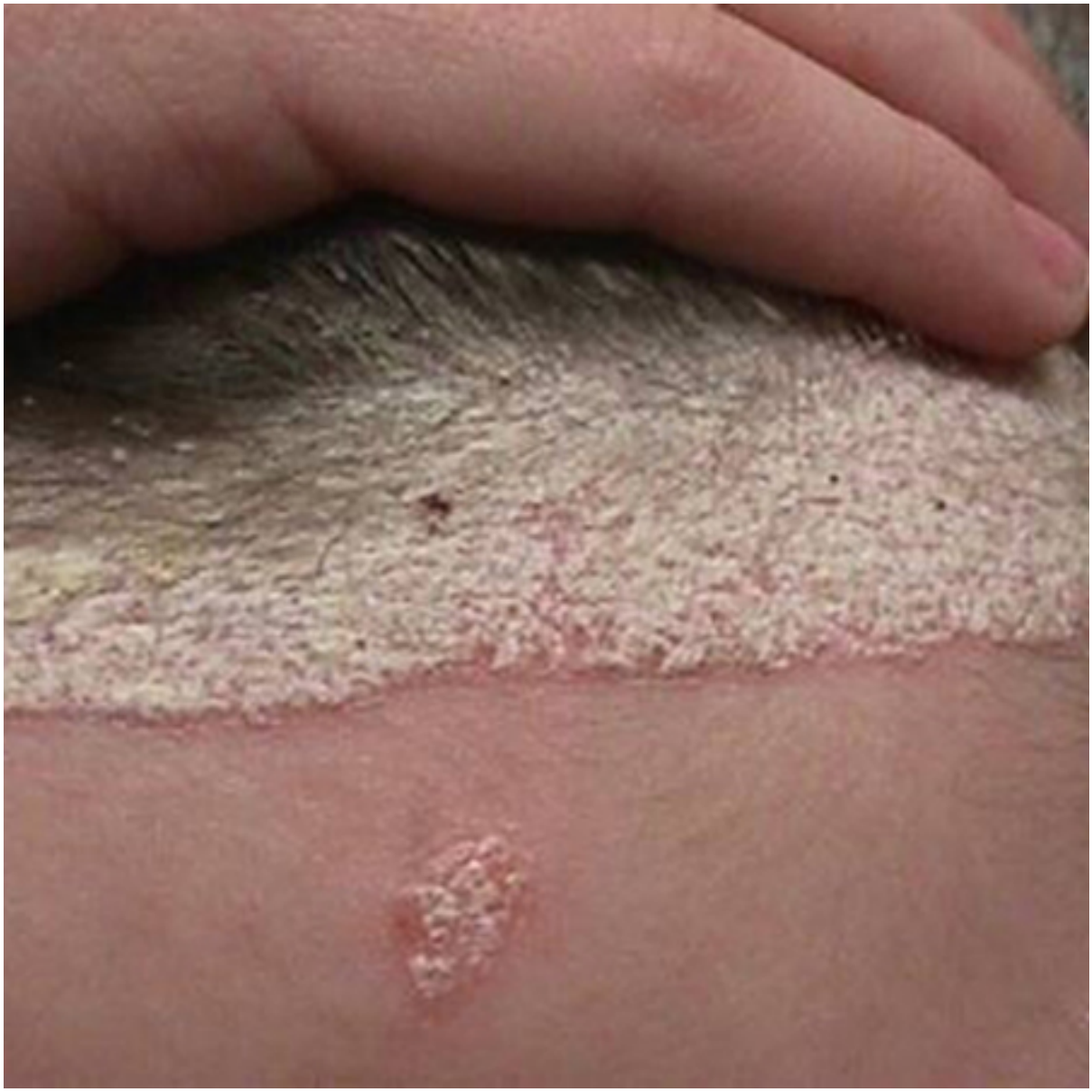

Plaque psoriasis on scalp

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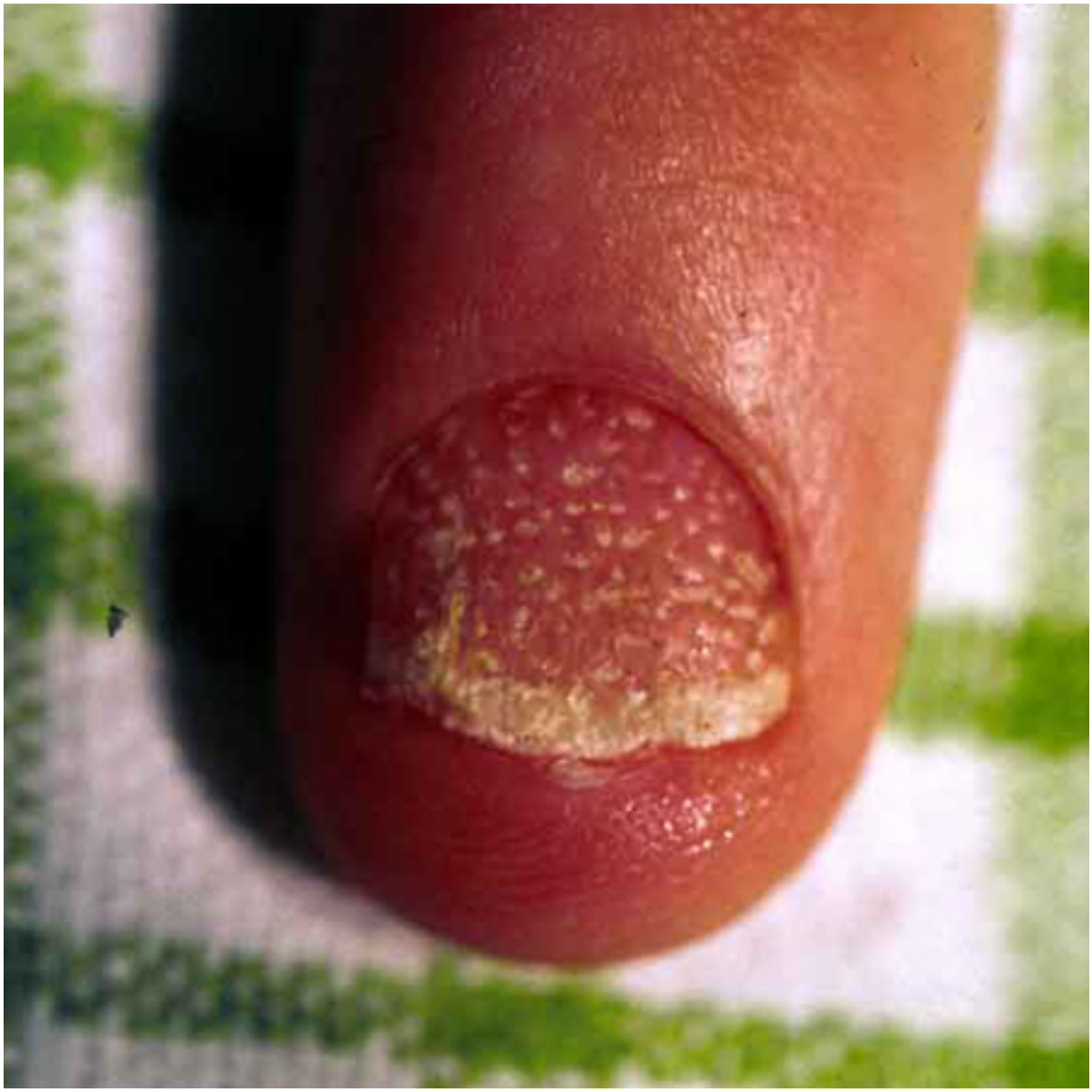

Nail psoriasis - pitted nails

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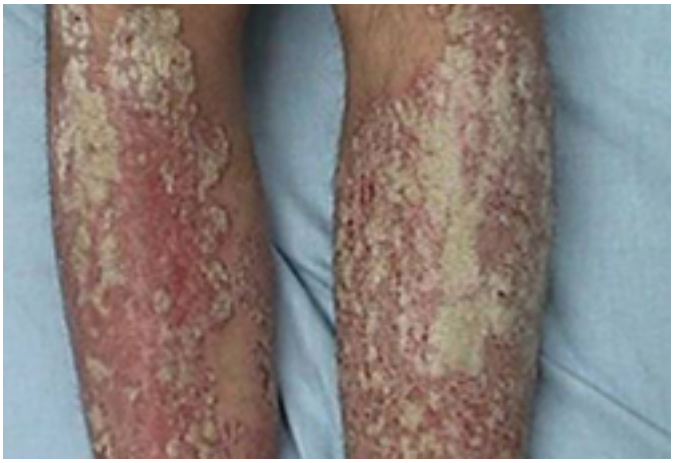
Subtypes of psoriasis have a typical presentation:[2]

Plaque psoriasis

- Raised inflamed plaque lesions with a superficial silvery-white scaly eruption. The scale may be scraped away to reveal inflamed and sometimes friable skin beneath.



Plaque psoriasis on legs

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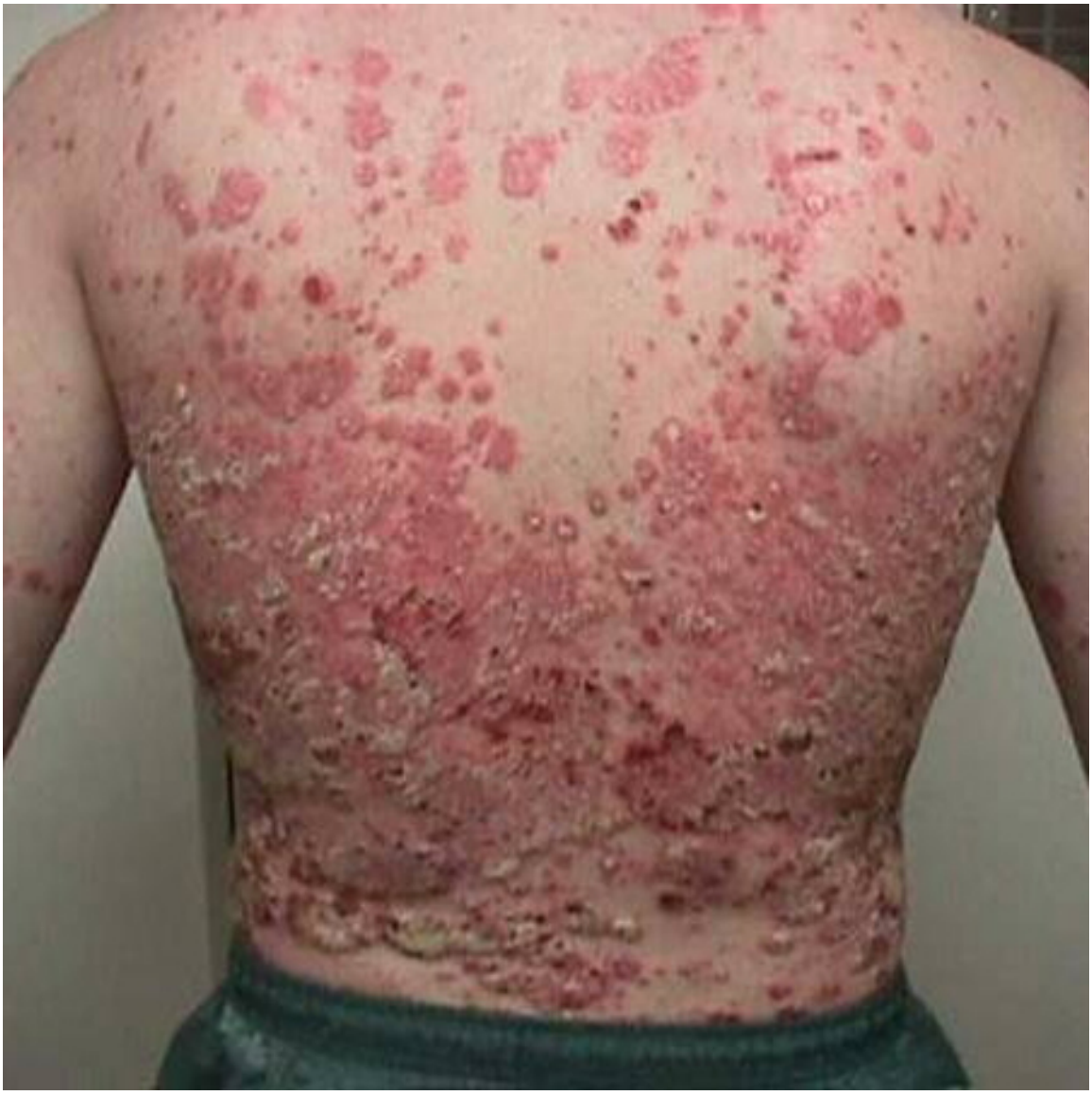

Plaque psoriasis on back

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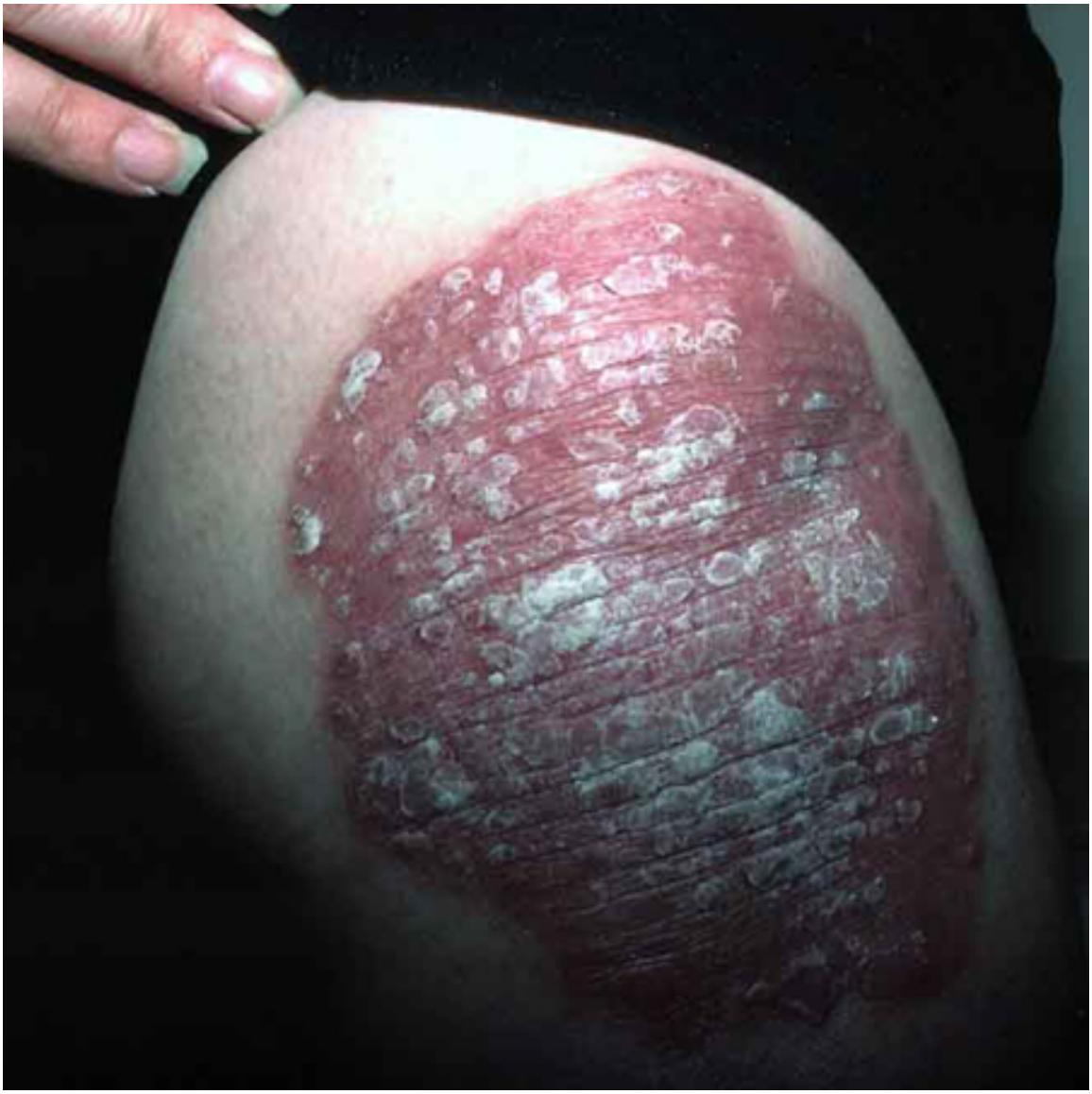

Plaque psoriasis on knee

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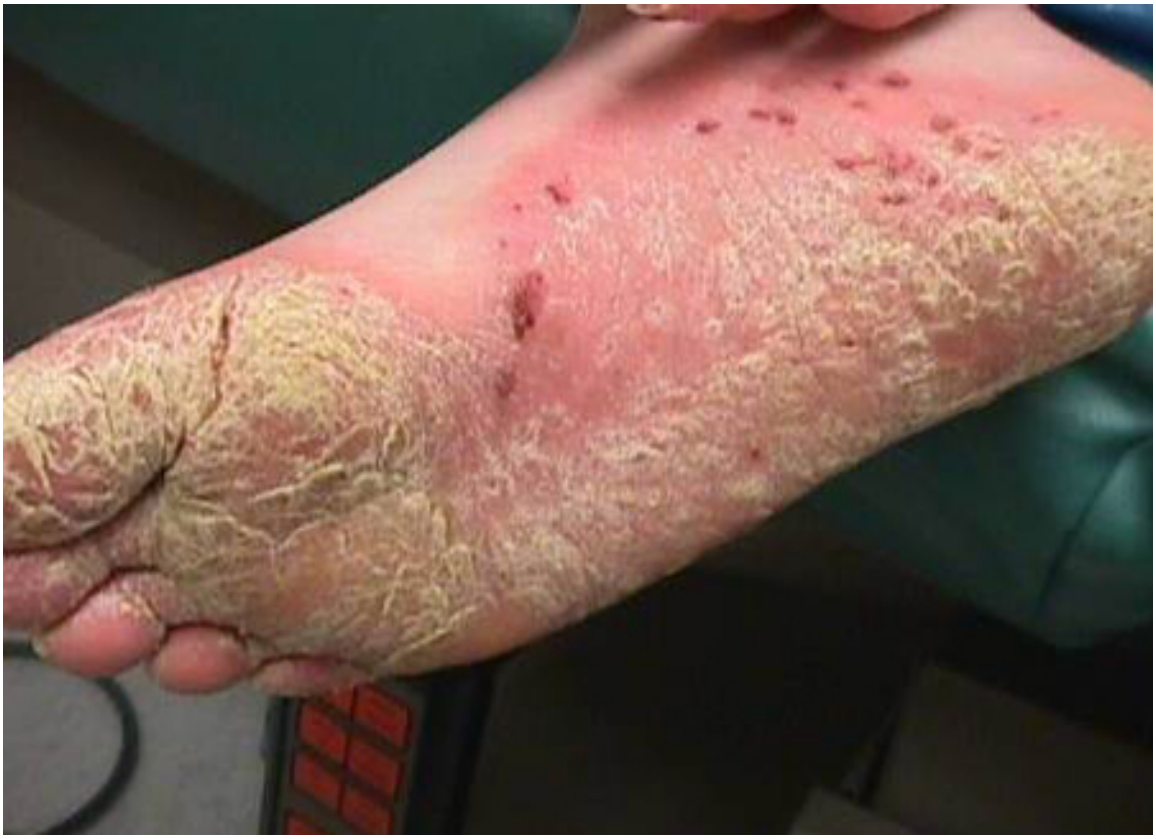

Plaque psoriasis on foot

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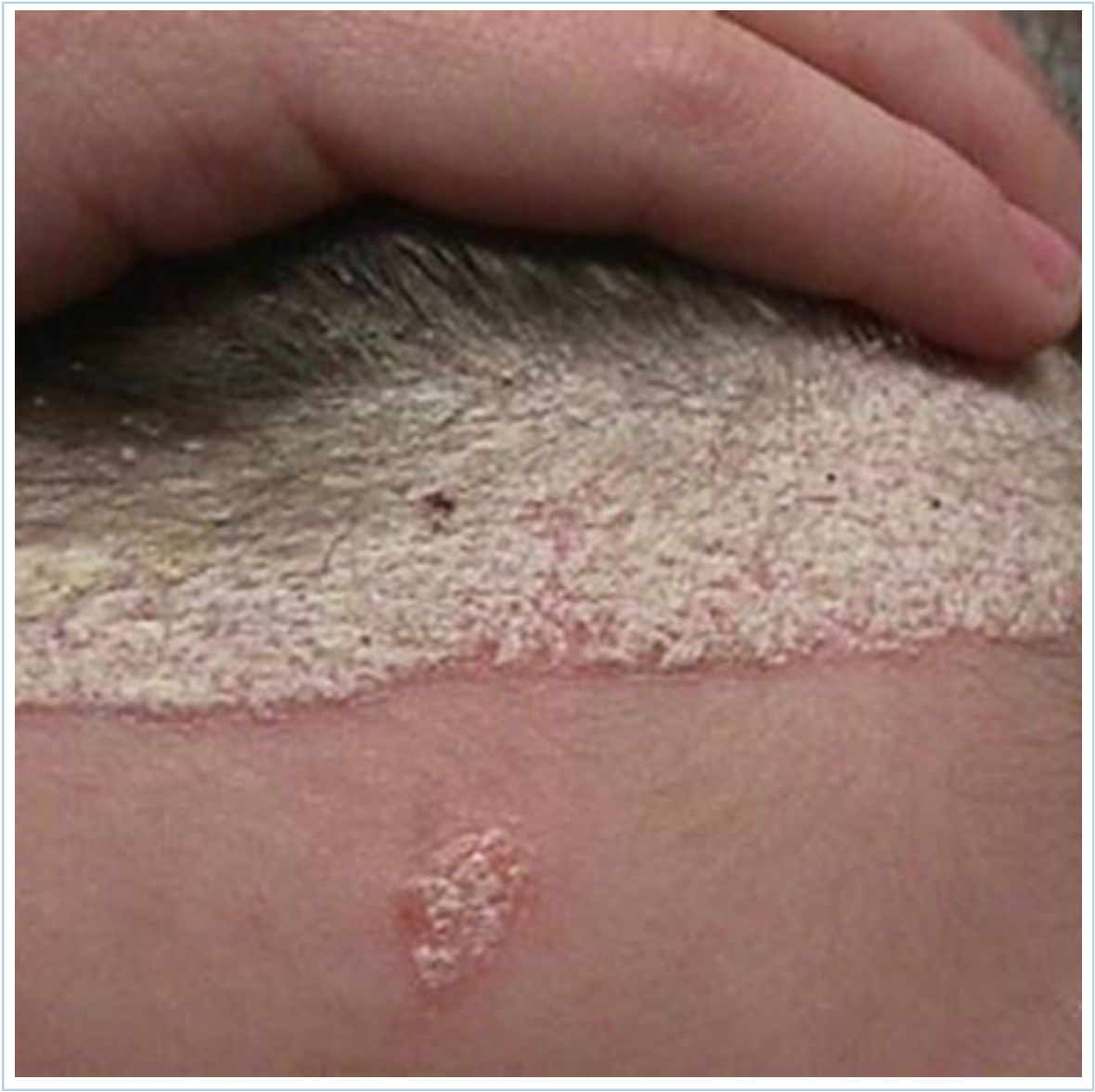

Plaque psoriasis on scalp

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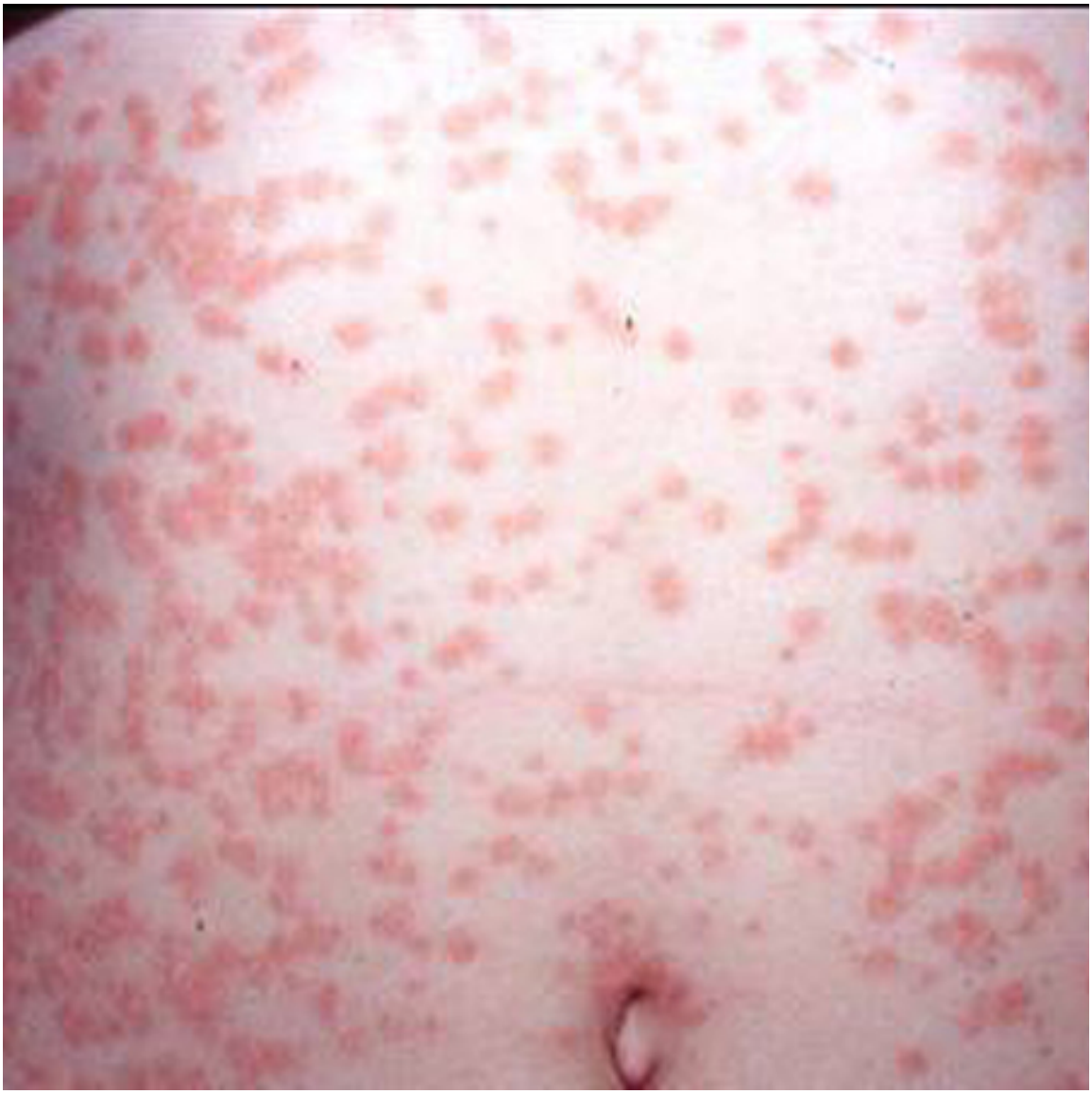
Guttate psoriasis

- Widespread, erythematous, fine, scaly papules (water drop appearance) on trunk, arms, and legs. The lesions often erupt after an upper respiratory infection.



Guttate psoriasis

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Pustular psoriasis

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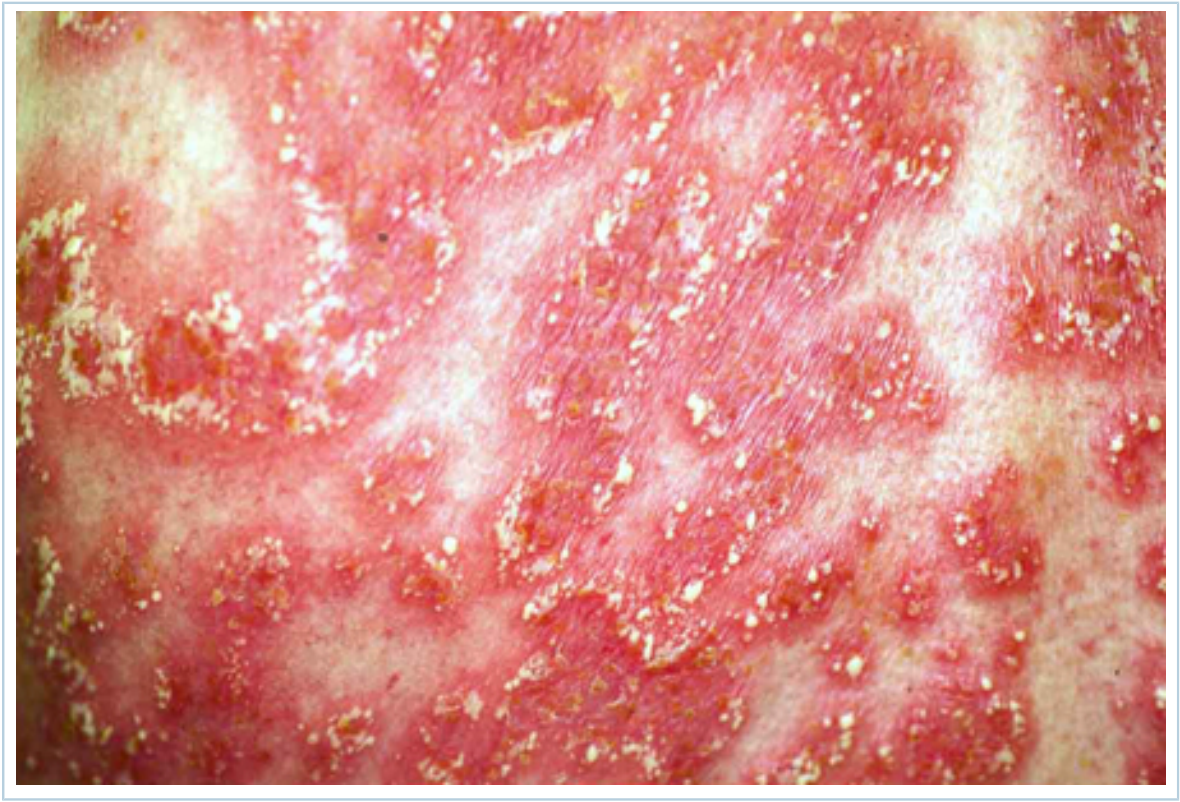

Pustular psoriasis

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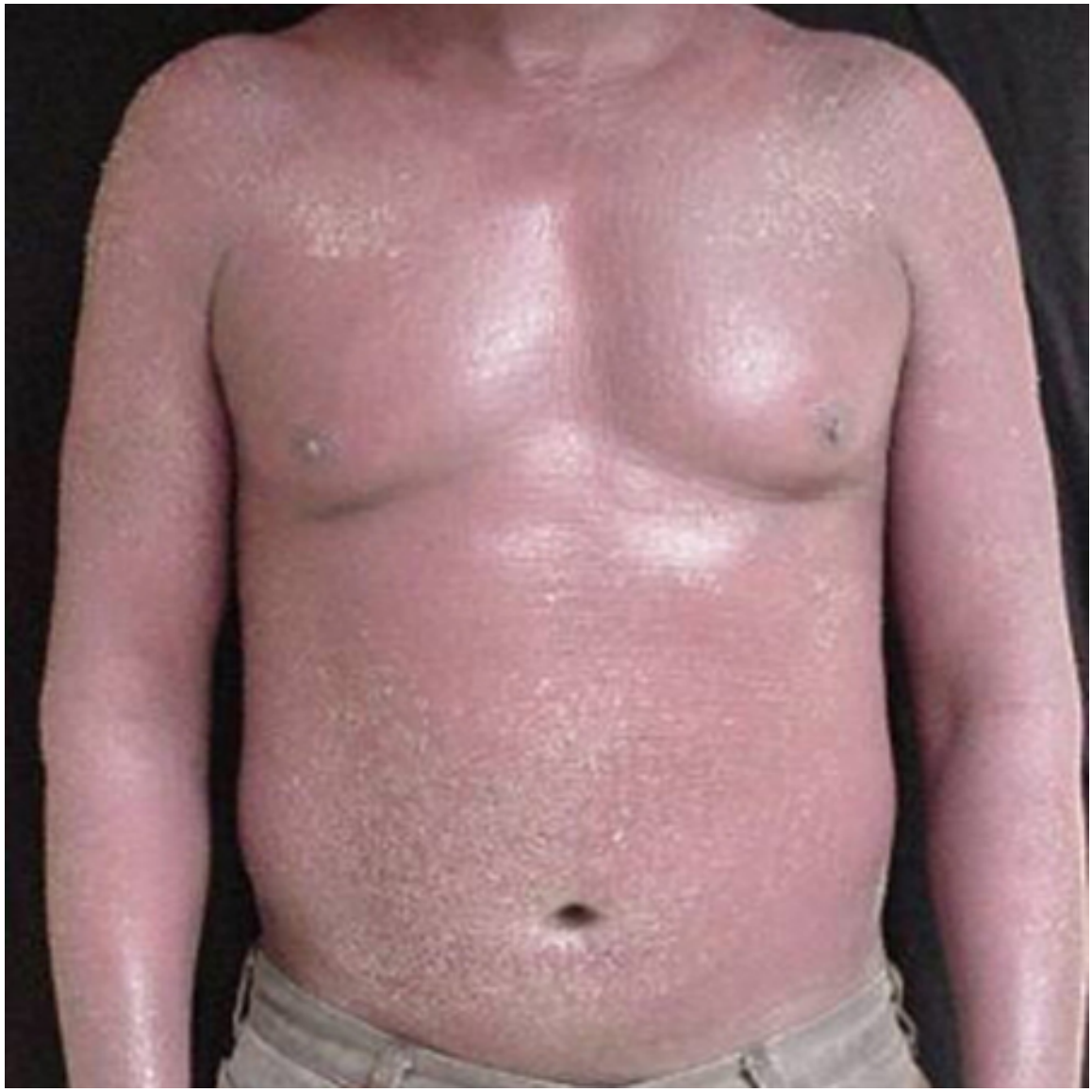
Erythroderma (erythrodermic psoriasis)

- Generalised erythema with fine scaling. It is often associated with pain, irritation, and sometimes severe itching.



Erythroderma

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Psoriasis Area and Severity Index (PASI)

PASI is the most widely used tool to measure severity and extent of psoriasis.[47] is a tool that can be used to measure severity and extent of psoriasis. It is a composite score grading severity in four body regions according to erythema, scaling, thickness, and the total area of skin affected. Severity of each of erythema, scaling, and thickness is graded from 0 to 4, and the extent of body surface area involvement in each body region is graded categorically from 1 to 6. The final composite score ranges from 0 to 72, with a higher score indicating a greater severity of psoriasis. A PASI of 10 or above indicates severe disease.[48] Online PASI calculators are available.

Other useful tools

- Body surface area: evaluates the extension on the body surface area without considering lesion features like erythema and scaling.
- Physician Global Assessment: a qualitative evaluation of the overall disease severity that is not sensitive to modification of disease severity over time (a rough estimation).

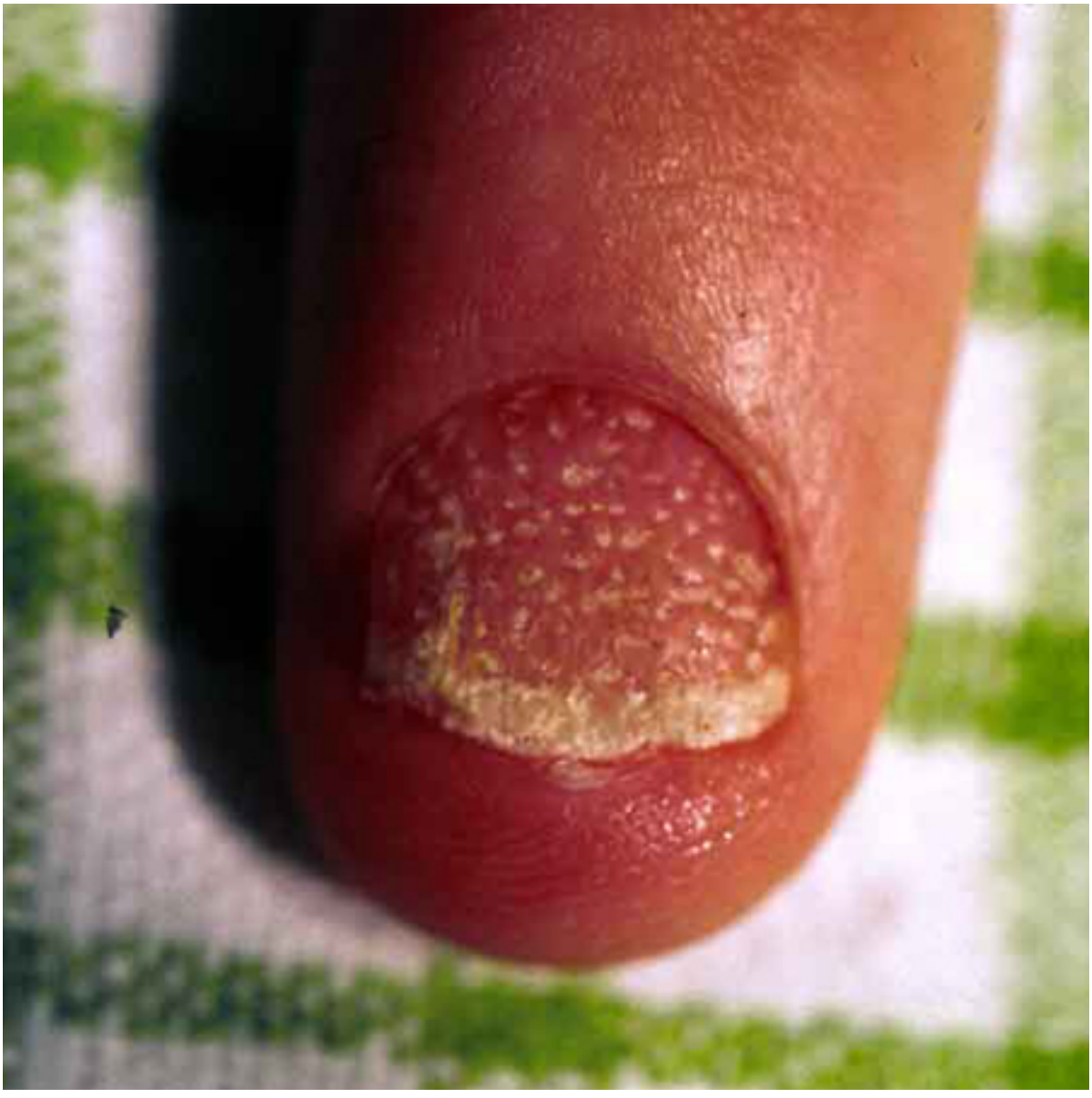
- Dermatology Life Quality Index: useful in assessing the impact of the disease on patient quality of life.

History and exam

Key diagnostic factors

skin lesions (common)

- Typically erythematous, circumscribed scaly papules and plaques on elbows, knees, extensor surfaces of limbs, scalp, and, less commonly, nails, ears, and umbilical region.



Nail psoriasis - pitted nails

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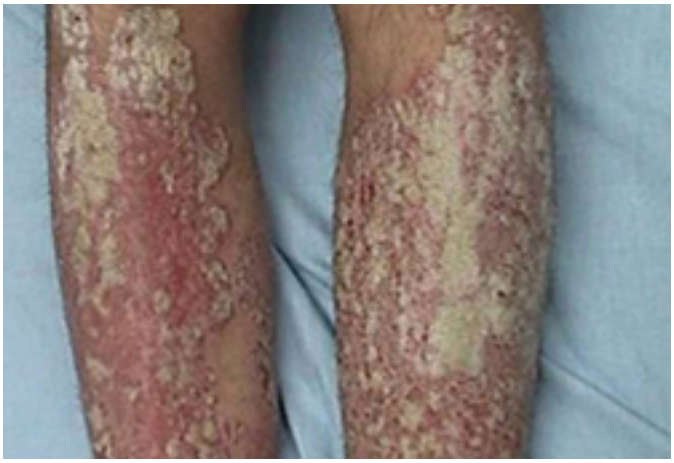
- In plaque psoriasis, there are raised inflamed plaque lesions with a superficial silvery-white scaly eruption. The scale may be scraped away to reveal inflamed and sometimes friable skin beneath.[2] Pinpoint bleeding points are known as Auspitz's sign.



Plaque psoriasis on legs

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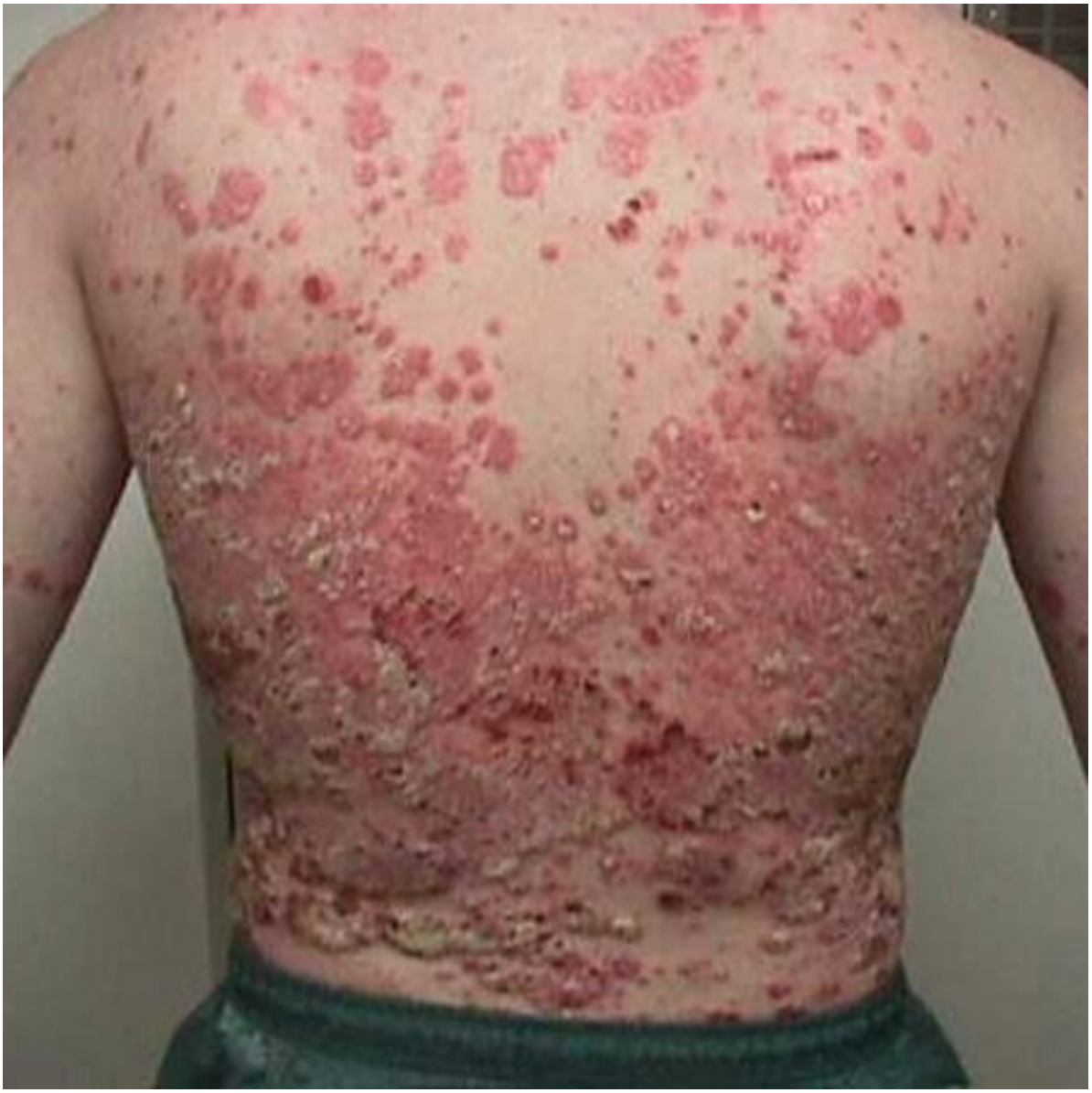

Plaque psoriasis on back

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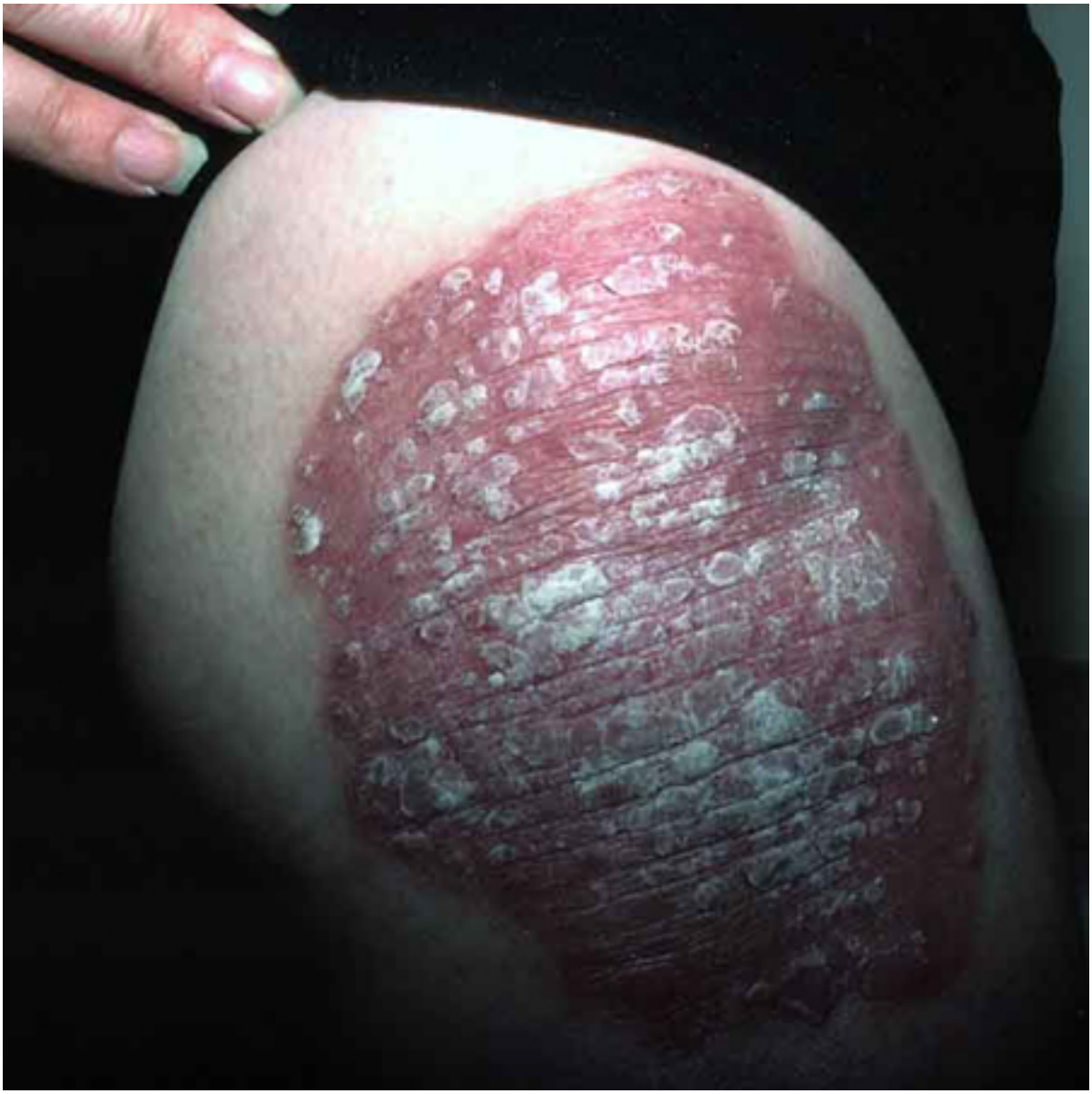

Plaque psoriasis on knee

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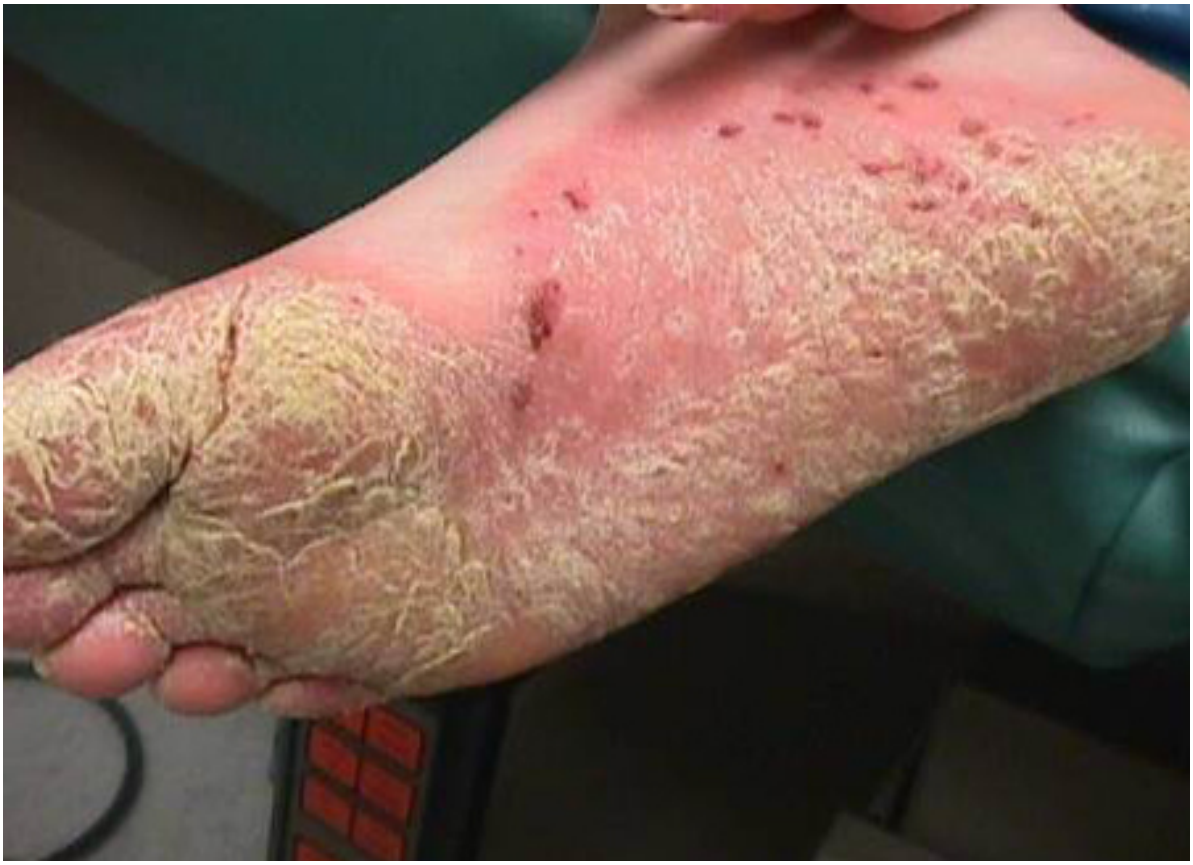

Plaque psoriasis on foot

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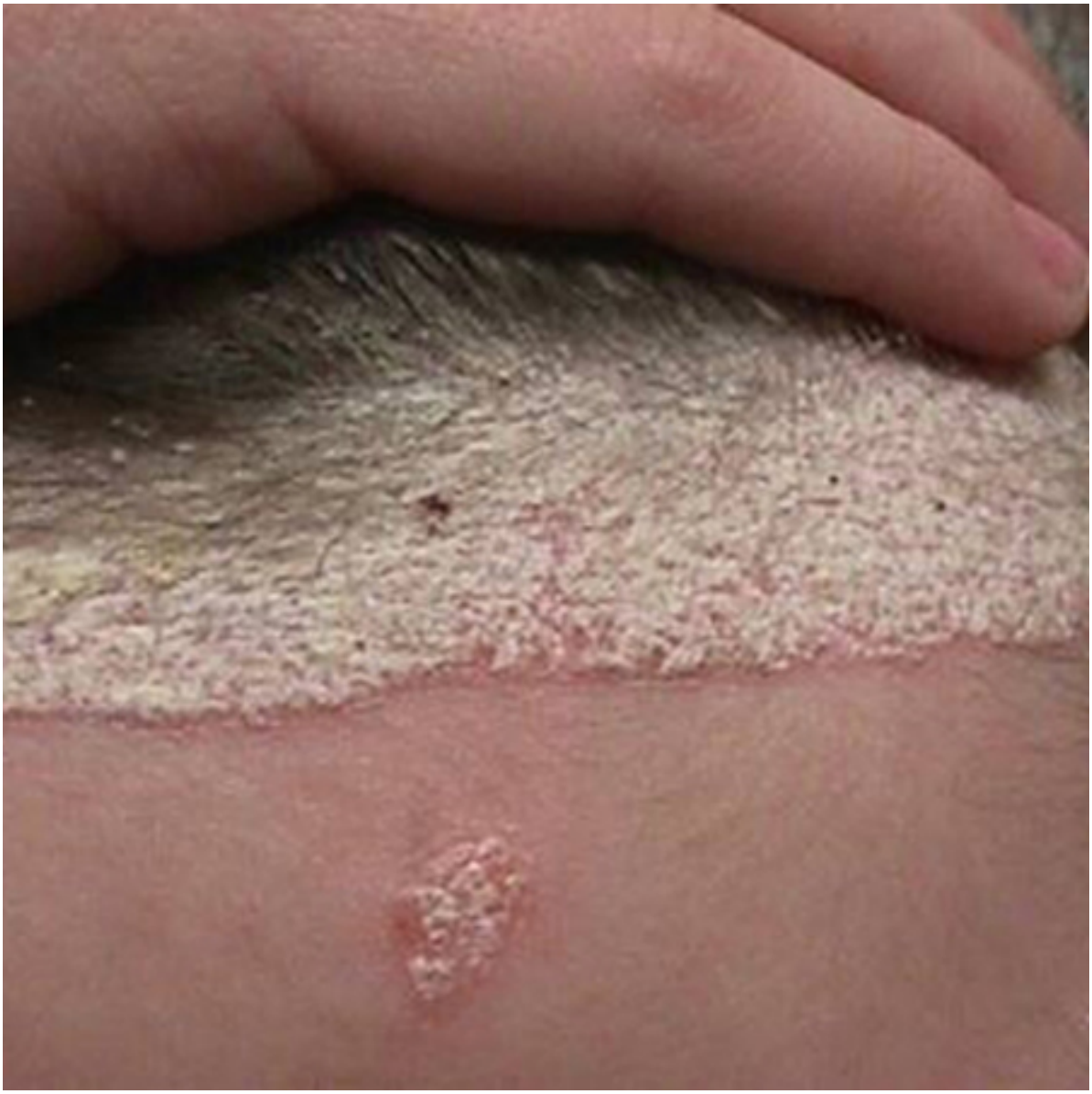

Plaque psoriasis on scalp

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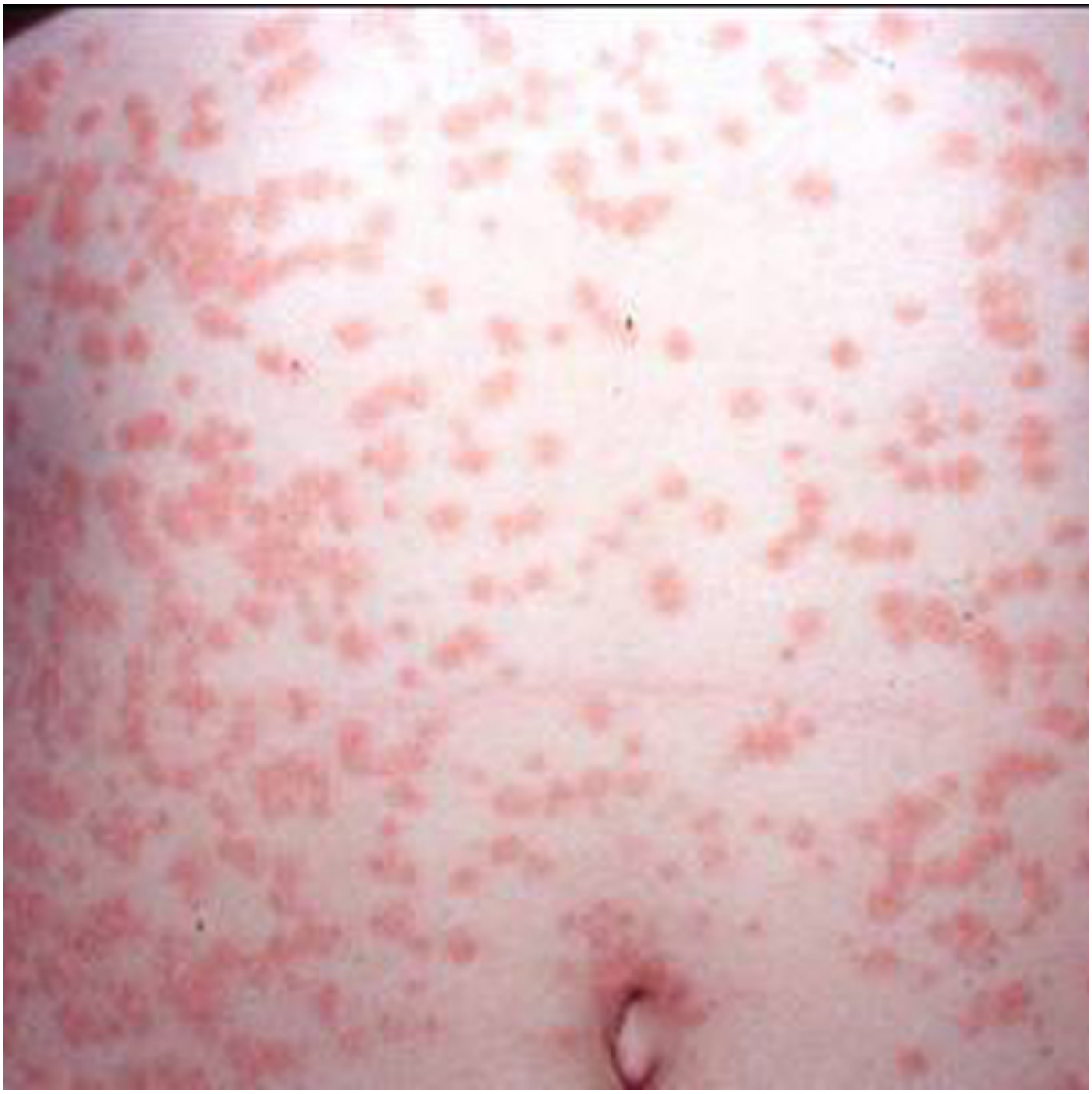
- In guttate psoriasis, there are widespread, erythematous, fine, scaly papules (water drop appearance) on trunk, arms, and legs. The lesions often erupt after an upper respiratory infection.[2]



Guttate psoriasis

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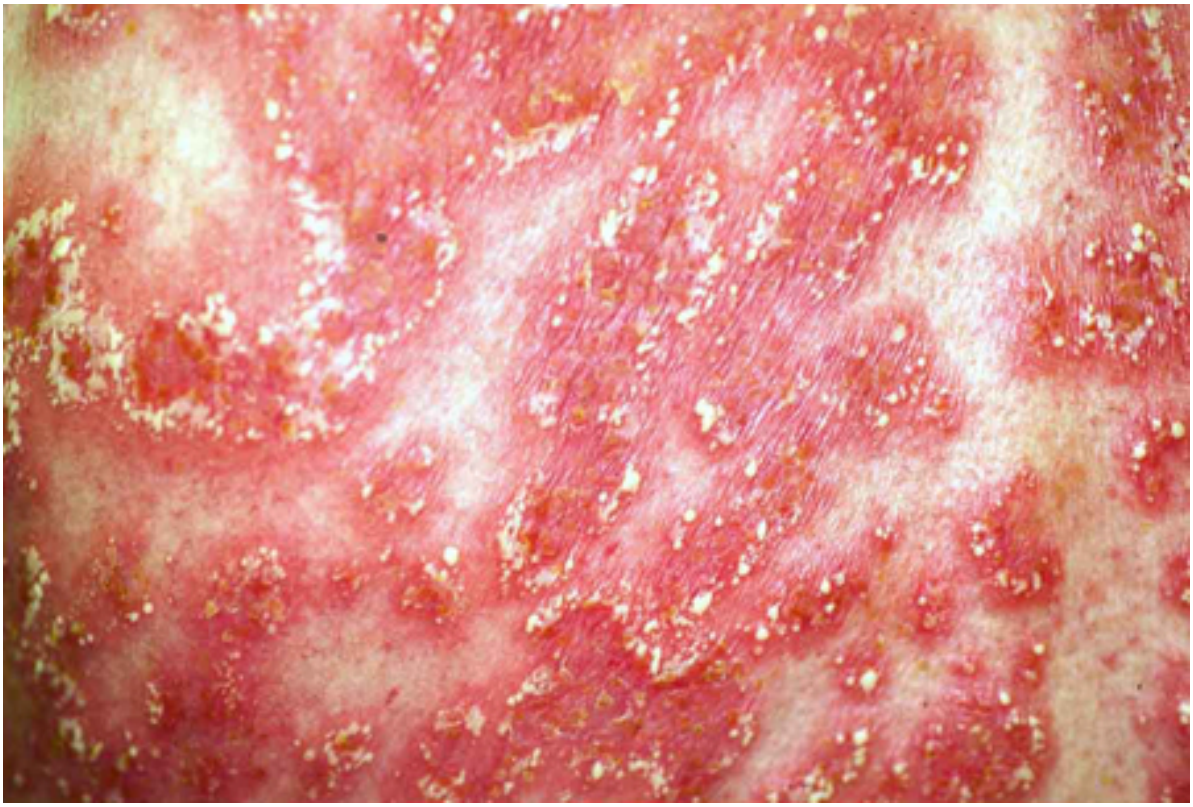
- In pustular psoriasis, acute generalised pustular psoriasis (von Zumbusch) is rare, severe, and urgent; palmoplantar pustulosis affects palms and soles and is chronic.[2]



Pustular psoriasis

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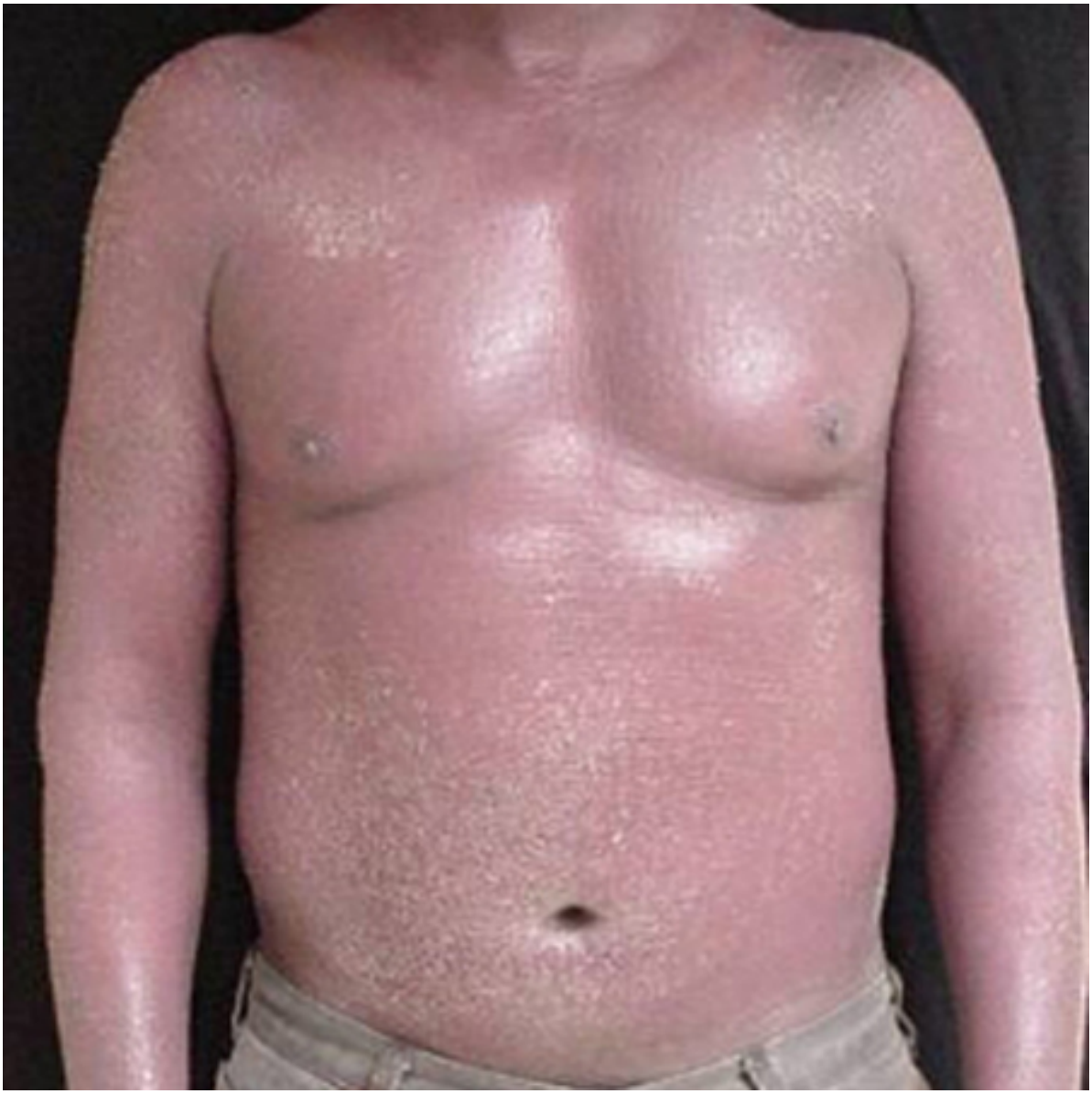
- In erythroderma (erythrodermic psoriasis), there is generalised erythema with fine scaling. It is often associated with pain, irritation, and sometimes severe itching.[2]



Erythroderma

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Other diagnostic factors

family history (common)

- Most people with psoriasis have a positive family history.[11] [16]
- Studies of monozygotic twins, linkage studies, and genome-wide association studies provide evidence that psoriasis has a genetic predisposition.[11] [12] [13]

joint swelling or pain (common)

- Psoriatic arthritis occurs in 20% of people with psoriasis and can point towards a diagnosis of cutaneous psoriasis.[3]
- Psoriatic arthritis has several presentations including joint pain, tendinitis, enthesitis, or dactylitis. In most cases arthritis presents after the onset of cutaneous psoriasis, but it may be a presenting sign.[49]
- Risk factors for psoriatic arthritis include early age at first presentation, female sex, polyarticular involvement, and genetic predisposition.[50]

Risk factors

Strong

genetic

- Most people with psoriasis have a positive family history.[11] [16]
- Studies of monozygotic twins, linkage studies, and genome-wide association studies provide evidence that psoriasis has a genetic predisposition.[11] [12] [13]
- Psoriasis has been linked to a number of genes, with the strongest association to those involved in the immune response, particularly IL23R, IL12B, and tumour necrosis factor (TNF)-alpha.[14]

infection

- Guttate psoriasis is often observed subsequent to upper respiratory infection, such as streptococcal pharyngitis. It may also be associated with HIV infection. Viral infection, immunisation, and any intercurrent illness have been linked to flares of guttate and plaque psoriasis.[16] [19]

local trauma

- Trauma, such as surgical scars and injection sites, may result in the appearance of new psoriatic lesions at the site of injury.[29] This is known as the Koebner phenomenon.

medications

- Several medications may induce or exacerbate pre-existing psoriasis (the incidence of psoriasis exacerbation is generally greater than that of psoriasis induction), including antihypertensives and lithium.[30]
- The latency period between drug ingestion and psoriasis flares varies, and can be considerable for certain medications.[31] [32]

Weak

stress

- Stress and sleep deprivation are recognised to exacerbate psoriasis. Stress reduction techniques may be useful in managing exacerbations.[33] [34]

smoking

- Systematic reviews and meta-analyses have found that that smoking is a risk factor for the development of psoriasis.[35] [36]

- Risk increases the greater the number of cigarettes smoked per day and in longer durations of smoking.[35]

ethnicity

- Psoriasis is reported to be twice as common in white people than in black people.[7] [37]

alcohol

- Alcohol consumption may be associated with increased risk of psoriasis.[38] [39]
- Heavy alcohol intake exacerbates psoriasis and complicates treatment by increasing the inflammatory response, altering pharmacology, and potentially reducing adherence to medication.[40] [41]

greater body mass index (BMI)

- Obesity is more prevalent in people with psoriasis (30% to 40%) compared with the general population.[42]
- Obesity as measured by BMI, waist circumference, waist-to-hip ratio, and weight gain is associated with increased risk of psoriasis and exacerbation of pre-existing psoriasis.[43] [44] [45] [46]

Investigations

1st test to order

Test	Result
clinical diagnosis • Usually no tests are necessary.	**features of psoriasis**

Other tests to consider

Test	Result
skin biopsy • Order skin biopsy only when diagnosis is in doubt. • Biopsy does not always show classic pathological features.	**intra-epidermal spongiform pustules and Munro neutrophilic microabscess within the stratum corneum; in addition to these classical features, others include focal parakeratosis and epidermal acanthosis with dilated capillaries within dermal papillae**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Eczema	• Dry, scaly, red skin sometimes with excoriations visible (scratch marks). Exacerbations may be associated with skin infection causing weeping or oozing skin. The border of eczema is usually less well-defined than a plaque of psoriasis.	• Skin biopsy shows changes consistent with atopic dermatitis.
Pityriasis rosea	• More common in children. • Lesions may show features of guttate psoriasis but are in a characteristic Christmas tree-shaped distribution. • Usually subsides within 8 weeks.	• Clinical diagnosis is usually sufficient.
Seborrhoeic dermatitis	• Scaly eruptions usually limited to scalp, eyebrows, paranasal region, ears, and chest, but can be widespread. • Scales are fine, not lamellar.	• Skin biopsy shows changes consistent with seborrhoeic dermatitis.
Mycosis fungoides	• Usually presents with patches and plaques on the lower half of the body but can be widespread. • Does not involve joints.	• Skin biopsy shows atypical lymphocytes and Pautrier abscess.
Tinea corporis	• Annular scaly patches.	• Skin scraping or biopsy confirms diagnosis.
Nappy dermatitis	• Oozy, weepy. • Only in nappy region.	• Clinical diagnosis is usually sufficient.
Onychomycosis	• Only involves nails.	• Culture of nail shows fungus.
Squamous cell cancer/ actinic keratosis	• Actinic keratosis or actinic field change often affect the forehead and dorsal aspect of hands, which are less common sites for psoriasis. • Usually presents at an older age.	• Skin biopsy shows proliferating atypical squamous cells.
Lichen planus	• Violaceous papules. Oral mucosa is more likely to be involved than in psoriasis.	• Skin biopsy shows lichenoid lymphocyte infiltrates under epidermis.

Condition	Differentiating signs / symptoms	Differentiating tests
Lichen simplex chronicus	• Usually limited to a few areas easily reached by hands. • Lesions are thick and mostly without scaly or desquamated appearance	• Skin biopsy shows chronic dermatitis with epidermal acanthosis.
Subcorneal pustular dermatosis	• A differential for pustular psoriasis. • Pustular lesions are subcorneal and in annular/ serpiginous forms, present on the abdomen, axillae, and groin.	• Culture of pustules shows no bacteria. Skin biopsy shows predominantly neutrophilic perivascular infiltrate; minimal spongiosis.
Keratoderma blennorrhagicum (reactive arthritis)	• Lesions are circular, scaly, scalloped-edged hyperkeratotic psoriasiform papules and plaques, which are sometimes painful and pustular (at the centre of lesions); appear on soles and toes, and, less commonly, legs, palms, scalp, and penis.	• Skin biopsy may be done, but may show overlapping features with psoriasis.

Criteria

Psoriasis Area and Severity Index (PASI) score[47]

Composite score grading severity of psoriasis in four body regions according to erythema, scaling, thickness, and the total area of skin affected. Severity of each of erythema, scaling, and thickness is graded from 0 to 4, and the extent of body surface area involvement in each body region is graded categorically from 1 to 6. The final composite score ranges from 0 to 72, with a higher score indicating a greater severity of psoriasis. Online PASI calculators are available.

Physician Global Assessment (PGA)

The PGA was introduced in 1998 by a US Food and Drug Administration panel as the preferred tool to assess and record the severity of disease in clinical studies.[47] Typically rates a patient's disease from 'clear' to 'severe' or 'very severe'.

Body surface area (BSA)

BSA percentage is a rapid and easy method to score psoriasis; BSA >10% is considered severe disease.[51]

Approach

The aim of treating psoriasis is to decrease the percentage of body surface involved (aiming for complete disease clearance) in as short a time as possible, and to maintain remission. Effectiveness of therapy is usually monitored by both a disease severity tool such as the Psoriasis Area and Severity Index (PASI) and a quality-of life index, usually the Dermatology Life Quality Index.[48]

Mild psoriasis

Topical treatments are the mainstay of therapy.[52] [53]

Choice of formulation depends on the area of cover (e.g., lotion for scalp; cream for moist weeping lesions; and ointment for dry, lichenified, or scaly lesions).

For patients with limited psoriasis involvement, start with topical corticosteroids and a topical vitamin D analogue.[54] [55] [56] [Evidence A] Topical calcineurin inhibitors are second-line agents. Emollients may be considered by those averse to pharmacological options.

Topical corticosteroids

- A topical corticosteroid in combination with a vitamin D analogue is more effective in treating disease than either treatment alone.[55] [57]
- Combination therapy may help to reduce potential adverse effects associated with extensive use of topical corticosteroids.
- The potency of topical corticosteroid used is determined by the extent of disease and the responsiveness of the patient to medications. Low-potency treatments are appropriate for lesions on the face or intertriginous areas.[58]
- The combination product halobetasol/tazarotene has been approved for the treatment of plaque psoriasis in adults in some countries.

Topical vitamin D analogues

- Agents such as calcipotriol bind with vitamin D-selective receptors and inhibit the hyperproliferation and abnormal differentiation of keratinocytes characteristic of psoriatic lesions.[58]
- Calcipotriol has a relatively slow onset of action and its maximal effect is after 6 to 8 weeks. A two-compound formulation with betamethasone dipropionate appears to be superior to other topicals in scalp psoriasis and psoriasis vulgaris.[59] [60]
- Topical vitamin D analogues can be used alone for chronic therapy when psoriasis is under good control or when treatment needs to be applied long-term to the face or intertriginous areas.

Topical calcineurin inhibitors

- Tacrolimus or pimecrolimus are often used as second-line agents in the treatment of psoriasis, especially facial, flexural, and genital psoriasis; however, this use is off-label.[61] [62]

Patients should be referred to a dermatologist for consideration of systemic therapy if:[63]

- Diagnosis is unclear
- Psoriasis does not respond to topical therapy
- Psoriasis is widespread
- Psoriasis is on parts of the body that are highly visible or difficult to treat (including face, scalp, genitals), or

- Psoriasis is having an adverse impact on the mood or mental health of the patient (contributing to anxiety or depressive symptoms).

Moderate to severe psoriasis

Treatment options for moderate to severe psoriasis include phototherapy, conventional systemic therapy (including methotrexate, ciclosporin, or acitretin), apremilast, fumaric acid esters, and biological therapy.[64]

Treatment should be supervised by a dermatologist.[65]

Phototherapy

- Phototherapy for moderate to severe psoriasis includes narrow-band UVB or PUVA.[66]
- Phototherapy is an effective treatment for psoriasis with skin clearance rates of 50% to 75% with narrow-band UVB, and up to 85% with PUVA.[67]
- Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.
- Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning if the dose is not adequately controlled. There is a small increased risk of skin cancer; the risk is higher in Fitzparick skin types I and II.

Conventional systemic therapy

- Methotrexate
    - A folic acid antagonist that works as an antiproliferative and anti-inflammatory agent that is considered a first-line systemic drug.
    - Methotrexate may increase the incidence of liver fibrosis in people who are overweight or who have diabetes.[68]
    - Folic acid is usually co-prescribed with methotrexate to minimise adverse effects (such as gastrointestinal symptoms and deranged liver function tests).[69]
    - Subcutaneous methotrexate may be used in people who fail to respond to oral therapy or have nausea with oral treatment.
- Ciclosporin
    - Suppresses T cells and pro-inflammatory cytokines (such as interleukin 2), inhibits antigen-presenting capacity of Langerhans cells, and impedes mast cell function of degranulation and cytokine production.
    - Ciclosprin is an effective treatment for psoriasis but has significant adverse effects, such as nephrotoxicity and hypertension.[70] It is, therefore, generally reserved for very extensive psoriasis requiring rescue to bring disease severity under relative control.
    - Long-term use (i.e., >12 months) is not recommended.
- Acitretin
    - An oral retinoid chemically related to vitamin A that helps to regulate epithelial cell growth.
    - Moderately effective in many cases and often combined with other treatments.
    - Do not use oral retinoids in women of childbearing age, as they are teratogenic.
    - Monitor liver function and blood lipid concentration.
- Apremilast

- An oral phosphodiesterase-4 inhibitor that works by modulating cyclic adenosine monophosphate levels, which in turn down-regulates inflammatory cytokines including tumour necrosis factor (TNF)-alpha and interleukins 23 and 17.
- Clinical trials have shown apremilast to have modest efficacy in patients with moderate to severe psoriasis.[71] [72] [73]
- Common adverse events included nausea, diarrhoea, nasopharyngitis, and upper respiratory tract infection.[71] [72] [73]
- Apremilast should be used with caution in patients with a history of depression.

- Fumaric acid esters

  - Fumaric acid esters have immunosuppressive and anti-inflammatory properties.
  - Licensed for moderate to severe psoriasis in European countries. In the UK, dimethyl fumarate is licensed for the treatment of moderate to severe plaque psoriasis in adults.
  - Not approved in the US for cutaneous psoriasis but may be prescribed off-label in the US and other countries.[74] [75] [76] [77]

Biological therapy

Biologicals have been transformative in the management of psoriasis, clearing widespread severe disease and improving psoriatic arthritis. They act at a cellular level and target particular steps in the immunological processes key to psoriasis activity.

A 'living' (regularly updated) Cochrane network meta-analysis has demonstrated that all biologicals are effective in improving psoriasis (90% or 90% improvement in PASI compared with baseline).[78] At class level, the biological treatments that target interleukin (IL)-17, IL-12/23, IL-23, and TNF-alpha were significantly more effective than the small molecules and conventional systemic agents.[78]

The results from another network meta-analysis of randomised controlled trials suggest that brodalumab, guselkumab, ixekizumab, and risankizumab are associated with the highest PASI response rates for both short- and long-term therapy.[79]

Rare adverse effects include drug-induced lupus (associated with TNF-alpha inhibitors) and *Candida* infections (with IL-17 inhibitors, typically mucocutaneous).[80]

Tuberculosis screening (e.g., tuberculin skin test, interferon-gamma release assay, asking about exposure and travel history, and chest x-ray) is recommended prior to initiation of biological therapy.[80] [81] Screening prior to initiation also includes an HIV and hepatitis B/C test.[80] [81]

All biologicals are given as subcutaneous injections (patients administer themselves) except infliximab, which is given as an intravenous infusion.

- TNF-alpha inhibitors

  - Include adalimumab, etanercept, infliximab, certolizumab.[82] [83] [84] [85] [86] [87] [88]
  - If clinically needed, certolizumab may be used in pregnancy.

- Interleukin-12/23 inhibitors

  - Ustekinumab: a human monoclonal antibody that inhibits interleukins 12 and 23.[89] [90] [91] [92]
  - Guselkumab: a monoclonal antibody that inhibits IL-23; believed to provide similar health benefits to ixekizumab and secukinumab.[93] [94]

- Risankizumab: a human monoclonal antibody that targets IL-23; significantly improved symptoms of moderate to severe psoriasis in clinical trials.[95]
- Tildrakizumab: an IL-23 antagonist approved for the treatment of moderate to severe plaque psoriasis; efficacious when compared with placebo and etanercept in two phase 3 trials.[96]

- Interleukin-17 inhibitors
  - Secukinumab: a human monoclonal antibody; efficacious in clearing psoriasis plaques.[97] [98] [99]
  - Ixekizumab: a monoclonal antibody; clinical trial data indicate that ixekizumab is highly effective in the treatment of moderate to severe psoriasis for up to 60 weeks of treatment.[100]
  - Brodalumab: a monoclonal antibody that targets the IL-17 receptor, blocking the signalling pathway of interleukins 17A, 17F, and 25. Appears to be well tolerated and efficacious over a 2-year period.[101] [102] [103]

Principles of biological therapy management

When considering a biological agent, factors to take into account include:[65]

- The goal of therapy (e.g., Physician Global Assessment, PASI, or body surface area)
- Disease phenotype and pattern of activity
- Disease severity and impact
- Individual factors including age, comorbidities, conception plans, and body mass index.

Biological therapy in patients with comorbid conditions

In patients with multiple sclerosis, TNF-alpha inhibitors are not recommended, while IL-17 inhibitors and ustekinumab are recommended first-line.

In patients with hepatitis B infection or latent tuberculosis, ustekinumab and IL-17 inhibitors are recommended, while TNF-alpha inhibitors should be used with caution.[104]

Biosimilars

Biosimilars are available for some biological agents. The International Psoriasis Council has published a consensus statement to guide prescribing of biosimilars (generic agents highly similar to the originator biological agent that can be prescribed at reduced cost).[105]

A 2021 systematic review in a small sample of psoriasis patients determined that switching between reference adalimumab and biosimilars has no impact on efficacy, safety, and immunogenicity.[106]

Erythrodermic psoriasis

Patients with erythrodermic psoriasis may need admission to hospital for intense topical treatment, fluid replacement, and electrolyte monitoring. Rapid and aggressive control is essential.

Initial treatment is often with ciclosporin for around 3 weeks to manage the flare. Patients who are more stable can be started with a biological agent (e.g., a TNF-alpha inhibitor, ustekinumab).

Guttate psoriasis

The recommended treatment approach for guttate psoriasis largely mirrors the strategies employed for plaque psoriasis. Important differences include investigating for an infectious trigger, which may include a throat swab for streptococcal infection and a screen for HIV.

First-line treatment is phototherapy; oral systemic therapies (e.g., ciclosporin, methotrexate, acitretin) are second- and third-line options.[66] [69] [70] [107] [108] [109] Ciclosporin is often prescribed first if guttate psoriasis is widespread and has not responded to phototherapy.[65]

Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.

Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning if the dose is not adequately controlled. There is a small increased risk of skin cancer; risk is higher in Fitzparick skin types I and II.

Pustular psoriasis

Pustular psoriasis may require hospital admission if widespread. Fluid replacement, electrolyte monitoring, and supportive care is required for patients with extensive disease.

Pustular psoriasis may be treated with intestine topical therapy, acitretin, or a combination of acitretin and phototherapy. Other systemic agents such as methotrexate and ciclosporin may be prescribed. Cases are managed on a case-by-case basis under the supervision of a dermatologist.

Managing patients with comorbidities

Comorbidities in patients with psoriasis contribute to poorer health outcomes and have a significant health economic burden. Guidelines encourage physicians to address comorbidities when managing psoriasis.[63] [110]

Screen people with moderate to severe psoriasis for comorbidities annually. The most common comorbidities associated with psoriasis are hyperlipidaemia, hypertension, obesity, type 2 diabetes, and depression.[111] [112]

People with psoriasis are also more likely to have non-alcoholic fatty liver disease and liver fibrosis, which may impact treatment with methotrexate.[113]

Management of psoriasis during the COVID-19 pandemic

- The International Psoriasis Council is recording data on psoriasis and the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic and will provide updates to the global dermatology community.[114]
- Data suggests that treatments for psoriasis, including biologicals, do not alter the risk of acquiring COVID-19 or having worse outcomes. It is recommended that patients who are not infected continue their biological or oral therapies in most cases.[115] [116]
- Established risk factors (being older, being male, being of non-white ethnicity, and having comorbidities) have been associated with higher hospitalisation rates.[117]
- Infection with COVID-19 may cause a flare of psoriasis. Resumption of psoriasis treatments withheld during SARS-CoV-2 infection should be decided on a case-by-case basis.[115]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoing			(summary)
plaque			
	mild	**1st**	**topical therapies**
	moderate to severe	**1st**	**phototherapy**
		1st	**methotrexate**
		1st	**ciclosporin**
		1st	**acitretin**
		1st	**apremilast**
		1st	**biological agent**
		2nd	**fumaric acid esters**
	erythrodermic	**1st**	**ciclosporin or biological agent**
guttate			
		1st	**phototherapy**
		2nd	**ciclosporin**
		2nd	**methotrexate**
		3rd	**acitretin**
pustular			
		1st	**supportive care, phototherapy, or systemic agents**

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Ongoing

plaque

mild — **1st** — **topical therapies**

Primary options

» hydrocortisone topical: (2.5%) apply sparingly to the affected area(s) two to four times daily
-or-
» triamcinolone topical: (0.025 or 0.1%) apply sparingly to the affected area(s) two to four times daily
-or-
» betamethasone dipropionate topical: (0.05%) apply sparingly to the affected area(s) once or twice daily
-or-
» clobetasol topical: (0.05%) apply sparingly to the affected area(s) twice daily for a maximum of 2 weeks, maximum 50 g/week

--AND/OR--

» calcipotriol topical: (0.005%) apply sparingly to the affected area(s) once or twice daily

OR

» calcipotriol/betamethasone dipropionate topical: apply sparingly to the affected area(s) once daily for up to 4 weeks

OR

» halobetasol/tazarotene topical: (0.01%/0.045%) apply sparingly to the affected area(s) once daily, maximum 50 g/week

Secondary options

» tacrolimus topical: (0.1%) apply sparingly to the affected area(s) twice daily

OR

» pimecrolimus topical: (1%) apply sparingly to the affected area(s) twice daily

Ongoing

» Topical treatments are the mainstay of therapy.[52] [53]

» Choice of formulation depends on the area of cover (e.g., lotion for scalp; cream for moist weeping lesions; and ointment for dry, lichenified, or scaly lesions).

» For patients with limited psoriasis involvement, start with topical corticosteroids and a topical vitamin D analogue.[54] [55] [56] [Evidence A] Topical calcineurin inhibitors are second-line agents. Emollients may be considered by those averse to pharmacological options.

» Topical corticosteroid: a topical corticosteroid in combination with a vitamin D analogue is more effective in treating disease than either treatment alone.[55] [57] Combination therapy may help to reduce potential adverse effects associated with extensive use of topical corticosteroids. The potency of topical corticosteroid used is determined by the extent of disease and the responsiveness of the patient to medications. Low-potency treatments are appropriate for lesions on the face or intertriginous areas.[58] The combination product halobetasol/tazarotene has been approved for the treatment of plaque psoriasis in adults in some countries.

» Topical vitamin D analogues: agents such as calcipotriol bind with vitamin D-selective receptors and inhibit the hyperproliferation and abnormal differentiation of keratinocytes characteristic of psoriatic lesions.[58] Calcipotriol has a relatively slow onset of action and its maximal effect is after 6 to 8 weeks. A two-compound formulation with betamethasone dipropionate appears to be superior to other topicals in scalp psoriasis and psoriasis vulgaris.[59] [60] Topical vitamin D analogues can be used alone for chronic therapy when psoriasis is under good control or when treatment needs to be applied long-term to the face or intertriginous areas.

» Topical calcineurin inhibitors: tacrolimus or pimecrolimus are often used as second-line agents in the treatment of psoriasis, especially facial, flexural, and genital psoriasis; however, this use is off-label.[61] [62]

moderate to severe **1st** **phototherapy**

» Phototherapy for moderate to severe psoriasis includes narrow-band UVB or PUVA.[66]

MANAGEMENT

Ongoing

» Phototherapy is an effective treatment for psoriasis with skin clearance rates of 50% to 75% with narrow-band UVB, and up to 85% with PUVA.[67]

» Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.

» Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning if the dose is not adequately controlled. There is a small increased risk of skin cancer; the risk is higher in Fitzparick skin types I and II.

1st **methotrexate**

Primary options

» methotrexate: 10-25 mg orally/ subcutaneously once weekly on the same day of each week

» A folic acid antagonist that works as an antiproliferative and anti-inflammatory agent that is considered a first-line systemic drug.

» Methotrexate may increase the incidence of liver fibrosis in people who are overweight or who have diabetes.[68]

» Folic acid is usually co-prescribed with methotrexate to minimise adverse effects (such as gastrointestinal symptoms and deranged liver function tests).[69]

» Subcutaneous methotrexate may be used in people who fail to respond to oral therapy or have nausea with oral treatment.

1st **ciclosporin**

Primary options

» ciclosporin: 2.5 to 5 mg/kg/day orally given in 2 divided doses

» Ciclosporin suppresses T cells and pro-inflammatory cytokines (such as interleukin 2), inhibits antigen-presenting capacity of Langerhans cells, and impedes mast cell function of degranulation and cytokine production.

» Ciclosporin is an effective treatment for psoriasis but has significant adverse effects, such as nephrotoxicity and hypertension.[70] It is, therefore, generally reserved for very extensive psoriasis requiring rescue to bring disease severity under relative control.

Ongoing

» Long-term use (i.e., >12 months) is not recommended.

1st **acitretin**

Primary options

» acitretin: 25-50 mg orally once daily

» An oral retinoid chemically related to vitamin A that helps to regulate epithelial cell growth. Moderately effective in many cases and often combined with other treatments.

» Do not use oral retinoids in women of childbearing age, as they are teratogenic.

» Monitor liver function and blood lipid concentration.

1st **apremilast**

Primary options

» apremilast: 10 mg orally once daily in the morning on day 1, followed by 10 mg in the morning and 10 mg in the evening on day 2, then 10 mg in the morning and 20 mg in the evening on day 3, then 20 mg in the morning and 20 mg in the evening on day 4, then 20 mg in the morning and 30 mg in the evening on day 5, then 30 mg twice daily thereafter

» An oral phosphodiesterase-4 inhibitor that works by modulating cyclic adenosine monophosphate levels, which in turn down-regulates inflammatory cytokines including tumour necrosis factor (TNF)-alpha and interleukins 23 and 17.

» Clinical trials have shown apremilast to have modest efficacy in patients with moderate to severe psoriasis.[71] [72] [73]

» Common adverse events included nausea, diarrhoea, nasopharyngitis, and upper respiratory tract infection.[71] [72] [73]

» Apremilast should be used with caution in patients with a history of depression.

1st **biological agent**

Primary options

» adalimumab: 80 mg subcutaneously on day 1, followed by 40 mg every other week starting 1 week after initial dose

OR

MANAGEMENT

Ongoing

» etanercept: 50 mg subcutaneously twice weekly for 3 months, followed by either 50 mg once weekly or 25 mg twice weekly, each dose should be 3-4 days apart

OR

» infliximab: 5 mg/kg intravenously at weeks 0, 2, 6, and then every 8 weeks thereafter

OR

» certolizumab pegol: 400 mg subcutaneously every 2 weeks
Can consider giving 400 mg subcutaneously at weeks 0, 2, and 4, then reducing dose to 200 mg every 2 weeks starting from week 6 in patients who weigh ≤90kg, as these patients may achieve an acceptable response with the lower dose.

OR

» ustekinumab: patient weight ≤100kg: 45 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter; patient weight >100kg: 90 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter

OR

» guselkumab: 100 mg subcutaneously at weeks 0 and 4, then every 8 weeks thereafter

OR

» risankizumab: 150 mg subcutaneously at weeks 0 and 4, and then every 12 weeks thereafter

OR

» tildrakizumab: 100 mg subcutaneously at weeks 0 and 4, then every 12 weeks thereafter

OR

» secukinumab: 300 mg subcutaneously at weeks 0, 1, 2, 3, and 4, followed by 300 mg every 4 weeks thereafter

Ongoing

OR

» ixekizumab: 160 mg subcutaneously at week 0, followed by 80 mg at weeks 2, 4, 6, 8, 10, and 12, then 80 mg every 4 weeks thereafter

OR

» brodalumab: 210 mg subcutaneously at weeks 0, 1, and 2, followed by 210 mg every 2 weeks thereafter; consider discontinuing treatment if inadequate response within 12-16 weeks

» Biologicals have been transformative in the management of psoriasis, clearing widespread severe disease and improving psoriatic arthritis. They act at a cellular level and target particular steps in the immunological processes key to psoriasis activity.

» A 'living' (regularly updated) Cochrane network meta-analysis has demonstrated that all biologicals are effective in improving psoriasis (90% or 90% improvement in Psoriasis Area and Severity Index [PASI] compared with baseline).[78] At class level, the biological treatments that target interleukin (IL)-17, IL-12/23, IL-23, and tumour necrosis factor (TNF)-alpha were significantly more effective than the small molecules and conventional systemic agents.[78]

» The results from another network meta-analysis of randomised controlled trials suggest that brodalumab, guselkumab, ixekizumab, and risankizumab are associated with the highest PASI response rates for both short- and long-term therapy.[79]

» Rare adverse effects include drug-induced lupus (associated with TNF-alpha inhibitors) and *Candida* infections (with IL-17 inhibitors, typically mucocutaneous).[80]

» Tuberculosis screening (e.g., tuberculin skin test, interferon-gamma release assay, asking about exposure and travel history, and chest x-ray) is recommended prior to initiation of biological therapy.[80] [81] Screening prior to initiation also includes an HIV and hepatitis B/C test.[80] [81]

» All biologicals are given as subcutaneous injections (patients administer themselves)

Ongoing

except infliximab, which is given as an intravenous infusion.

» TNF-alpha inhibitors: these include adalimumab, etanercept, infliximab, certolizumab.[82] [83] [84] [85] [86] [87] [88] If clinically needed, certolizumab may be used in pregnancy.

» Interleukin-12/23 inhibitors: ustekinumab is a human monoclonal antibody that inhibits interleukins 12 and 23.[89] [90] [91] [92] Guselkumab is a monoclonal antibody that inhibits IL-23, and is believed to provide similar health benefits to ixekizumab and secukinumab.[93] [94] Risankizumab is a human monoclonal antibody that targets IL-23 and significantly improved symptoms of moderate to severe psoriasis in clinical trials.[95] Tildrakizumab is an IL-23 antagonist approved for the treatment of moderate to severe plaque psoriasis, and was efficacious when compared with placebo and etanercept in two phase 3 trials.[96]

» Interleukin-17 inhibitors: secukinumab is a human monoclonal antibody that is efficacious in clearing psoriasis plaques.[97] [98] [99] Ixekizumab is a monoclonal antibody; clinical trial data indicate it is highly effective in the treatment of moderate to severe psoriasis for up to 60 weeks of treatment.[100] Brodalumab is a monoclonal antibody that targets the IL-17 receptor, blocking the signalling pathway of interleukins 17A, 17F, and 25; it appears to be well tolerated and efficacious over a 2-year period.[101] [102] [103]

2nd **fumaric acid esters**

Primary options

» dimethyl fumarate: consult specialist for guidance on dose

» Fumaric acid esters have immunosuppressive and anti-inflammatory properties.

» Licensed for moderate to severe psoriasis in European countries. In the UK, dimethyl fumarate is licensed for the treatment of moderate to severe plaque psoriasis in adults.

» Not approved in the US for cutaneous psoriasis but may be prescribed off-label in the US and other countries.[74] [75] [76] [77]

erythrodermic **1st** **ciclosporin or biological agent**

Primary options

Ongoing

» ciclosporin: 2.5 to 5 mg/kg/day orally given in 2 divided doses

Secondary options

» adalimumab: 80 mg subcutaneously on day 1, followed by 40 mg every other week starting 1 week after initial dose

OR

» etanercept: 50 mg subcutaneously twice weekly for 3 months, followed by either 50 mg once weekly or 25 mg twice weekly, each dose should be 3-4 days apart

OR

» infliximab: 5 mg/kg intravenously at weeks 0, 2, 6, and then every 8 weeks thereafter

OR

» ustekinumab: patient weight ≤100kg: 45 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter; patient weight >100kg: 90 mg subcutaneously as a single dose on day 1, week 4, and week 16, then once every 12 weeks thereafter

» Patients with erythrodermic psoriasis may need admission to hospital for intense topical treatment, fluid replacement, and electrolyte monitoring. Rapid and aggressive control is essential.

» Initial treatment is often with ciclosporin for around 3 weeks to manage the flare. Patients who are more stable can be started with a biological agent (e.g., a tumour necrosis factor [TNF]-alpha inhibitor, ustekinumab).

guttate

1st **phototherapy**

» The recommended treatment approach for guttate psoriasis largely mirrors the strategies employed for plaque psoriasis. Important differences include investigating for an infectious trigger, which may include a throat swab for streptococcal infection and a screen for HIV.

» First-line treatment is phototherapy. Phototherapy for moderate to severe psoriasis includes narrow-band UVB or

Ongoing

PUVA.[66] Phototherapy is an effective treatment for psoriasis with skin clearance rates of 50% to 75% with narrow-band UVB, and up to 85% with PUVA.[67]

» Phototherapy requires the patient to attend the clinic or hospital several times a week for the duration of treatment.

» Adverse effects of phototherapy include phototoxicity (during and after treatment), and burning if the dose is not adequately controlled. There is a small increased risk of skin cancer; risk is higher in Fitzparick skin types I and II.

2nd **ciclosporin**

Primary options

» ciclosporin: 2.5 to 5 mg/kg/day orally given in 2 divided doses

» Ciclosporin suppresses T cells and pro-inflammatory cytokines (such as interleukin 2), inhibits antigen-presenting capacity of Langerhans cells, and impedes mast cell function of degranulation and cytokine production.

» Ciclosprin is an effective treatment for psoriasis but has significant adverse effects, such as nephrotoxicity and hypertension.[70] It is, therefore, generally reserved for very extensive psoriasis requiring rescue to bring disease severity under relative control.

» Long-term use (i.e., >12 months) is not recommended.

2nd **methotrexate**

Primary options

» methotrexate: 10-25 mg orally/ subcutaneously once weekly on the same day of each week

» A folic acid antagonist that works as an antiproliferative and anti-inflammatory agent that is considered a first-line systemic drug.

» Methotrexate may increase the incidence of liver fibrosis in people who are overweight or who have diabetes.[68]

» Folic acid is usually co-prescribed with methotrexate to minimise adverse effects (such as gastrointestinal symptoms and deranged liver function tests).[69]

3rd **acitretin**

Ongoing

Primary options

» acitretin: 25-50 mg orally once daily

» An oral retinoid chemically related to vitamin A that helps to regulate epithelial cell growth. Moderately effective in many cases and often combined with other treatments.

» Do not use oral retinoids in women of childbearing age, as they are teratogenic.

» Monitor liver function and blood lipid concentration.

pustular

1st **supportive care, phototherapy, or systemic agents**

Primary options

» acitretin: 25-50 mg orally once daily

Secondary options

» ciclosporin: 2.5 to 5 mg/kg/day orally given in 2 divided doses

OR

» methotrexate: 10-25 mg orally/ subcutaneously once weekly on the same day of each week

» Pustular psoriasis may require hospital admission if widespread. Fluid replacement, electrolyte monitoring, and supportive care is required for patients with extensive disease.

» Pustular psoriasis may be treated with intestine topical therapy, acitretin, or a combination of acitretin and phototherapy.

» Other systemic agents such as methotrexate and ciclosporin may be prescribed. Cases are managed on a case-by-case basis under the supervision of a dermatologist.

Emerging

Tapinarof

Tapinarof, a small-molecule topical aryl hydrocarbon receptor (AhR) agonist, is the first topical novel chemical entity (corticosteroid-free) treatment to be approved by the US Food and Drug Administration (FDA) in 25 years for adults with any severity of plaque psoriasis. Two identical phase 3 randomised controlled trials demonstrated tapinarof significantly reduced the severity of plaque psoriasis, compared with vehicle, in patients with mild to severe plaque psoriasis at 12 weeks.[118] Patients who completed the 12-week trial were eligible to be included in a 40-week phase 3 open-label trial with a 4-week follow up.[119] The trial reported that 41% of patients achieved complete disease clearance (physician global assessment [PGA] score 0) and that 58% of patients who entered the trial with PGA ≥2 achieved PGA of 0 or 1. The mean duration of remission for patients who achieved PGA 0 was 130 days. The most frequent adverse effects were folliculitis, contact dermatitis, and upper respiratory tract infection.[119]

Tofacitinib

Tofacitinib, an oral Janus kinase inhibitor, is approved for use in patients with psoriatic arthritis and has been evaluated in phase 3 randomised controlled trials of patients with moderate to severe chronic plaque psoriasis.[120] [121] [122] Systematic reviews conclude that tofacitinib is effective in reducing signs and symptoms of chronic plaque psoriasis.[123] [124] Tofacitinib appeared to be associated with an increased risk for infection (including serious infections and herpes zoster) in some studies.[120] [122] [123]

Deucravacitinib

Deucravacitinib, a first-in-class oral selective tyrosine kinase 2 (TYK2) inhibitor, has been approved by the FDA to treat moderate to severe plaque psoriasis in adults. Deucravacitinib improved clearing of psoriasis compared with placebo in two phase 3, double-blind, randomised controlled trials in patients with moderate to severe plaque psoriasis at 16 and 24 weeks.[125] [126] Efficacy continued to improve after 24 weeks, with 82% of patients who achieved Psoriasis Area and Severity Index (PASI) score of 75 with deucravacitinib at week 24 maintaining their response at week 52 in the first trial.[125] The second of the two phase 3 trials included a randomised withdrawal and retreatment after week 24, 80% of patients who continued with deucravacitinib maintained PASI 75 response compared with 31% of patients who were withdrawn.[126] The results of the trials were limited by a lack of cultural diversity, further phase 3 trials are underway.[127]

Bimekizumab

Bimekizumab, an immunoglobulin G1 monoclonal antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A and IL-17AF, is approved in Europe for the treatment of moderate to severe plaque psoriasis in adults who are candidates for systemic therapy. The FDA is currently reviewing an application for approval. In patients with moderate to severe psoriasis, treatment with bimekizumab resulted in greater skin clearance than treatment with secukinumab over 16 and 48 weeks; bimekizumab was associated with oral candidiasis.[128] A study on the 2-year safety profile of bimekizumab reported that the treatment was well tolerated with no increase in adverse effects with longer treatment duration for patients with plaque psoriasis, apart from an increased risk of mild to moderate oral candidiasis.[129]

Spesolimab

Spesolimab, a monoclonal antibody that inhibits the activation of the interleukin-36 receptor (IL-36R), is the first FDA-approved treatment specifically to treat generalised pustular psoriasis (GPP) flares in adults. The approval is based on the results of one phase 2 randomised controlled trial which demonstrated that spesolimab significantly increased lesion clearance at one week compared with placebo in patients with GPP.[130] Infections occurred in 47% of patients treated with spesolimab at 12 weeks, and anti-drug antibodies were detected in 46% of the patients treated with spesolimab. Longer and larger trials are needed to identify the efficacy and risks of spesolimab treatment.[130]

Roflumilast

Roflumilast, a topical phosphodiesterase type 4 (PDE-4) inhibitor, is approved by the FDA for the treatment of plaque psoriasis. A phase 2b, double-blind randomised controlled trial demonstrated that roflumilast significantly improved disease clearance at 6 weeks, compared with vehicle, in patients with plaque psoriasis.[131] Further trials are needed.

Patient discussions

Educate patients on diagnosis, step-wise therapeutic options, and the importance of adherence to both topical and systemic medications.

Patients should be weighed and have their blood pressure checked annually. If they are overweight or obese, they should be counselled on the potential impact on their psoriasis and overall health.[104]

Tell patients about support groups and services available including:

- [Psoriasis Association] (https://www.psoriasis-association.org.uk)
- [National Psoriasis Foundation (US)] (https://www.psoriasis.org)

Monitoring

Monitoring

Frequency of monitoring depends on disease severity and the type of therapy that the patients are taking. In general, monitor patients with moderate to severe psoriasis at 3- to 6-month intervals. Clinicians need to ensure that treatment goals are met and continually monitor for drug safety.

Complications

Complications	Timeframe	Likelihood
cardiovascular complications	**long term**	**high**
Patients with psoriasis or psoriatic arthritis have an increased incidence of cardiovascular disease (e.g., myocardial infarction and stroke) and cardiovascular risk factors such as smoking, hypertension, and metabolic syndrome.[35] [134] [135] [136] There is also evidence to suggest that psoriasis is associated with an increased prevalence and incidence of diabetes and obesity, particularly in patients with severe psoriasis.[137] [138] [139] The cause is not yet known.[140] [141]		
psoriatic arthritis	**long term**	**medium**
Up to 30% of people with psoriasis have psoriatic arthritis.[1] The arthritis most commonly involves fingers, hands, toes, and feet, and, less commonly, knees, elbows, and axial and sacroiliac joints.		
depression	**variable**	**medium**
Patients with psoriasis have an increased risk of depression, anxiety, and suicidality.[132]		
lymphoma	**variable**	**low**
Patients with psoriasis have an increased risk of developing lymphoma. The cause of this association is unknown, but is thought to result from pathophysiology, treatment, or a combination of both.[133] Absolute risk is low.		
secondary infection	**variable**	**low**
Pruritus may lead to skin breaks from scratching		

Prognosis

The exact natural history of psoriasis is poorly understood. However, psoriasis is generally considered a chronic disease with a fluctuating course. Long-term control with topical and/or systemic medications is necessary for many patients.

Treatment guidelines

United Kingdom

British Association of Dermatologists guidelines for biologic therapy for psoriasis (https://www.bad.org.uk/healthcare-professionals/clinical-standards/clinical-guidelines)

Published by: British Association of Dermatologists **Last published:** 2020

Psoriasis: assessment and management of psoriasis (https://www.nice.org.uk/guidance/CG153)

Published by: National Institute for Health and Care Excellence **Last published:** 2017

Europe

Guideline on the management of vulval conditions (https://iusti.org/guidelines-resources)

Published by: International union against sexually transmitted infections **Last published:** 2021

EuroGuiDerm guideline on the systemic treatment of psoriasis vulgaris – part 2: specific clinical and comorbid situations (https://www.eadv.org/clinical-guidelines)

Published by: European Dermatology Forum **Last published:** 2021

EuroGuiDerm guideline for the systemic treatment of psoriasis vulgaris – part 1: treatment and monitoring recommendations (https://www.eadv.org/clinical-guidelines)

Published by: European Dermatology Forum **Last published:** 2020

French guidelines on the use of systemic treatments for moderate-to-severe psoriasis in adults (https://www.sfdermato.org/page-24-recommandations)

Published by: French Society of Dermatology **Last published:** 2019

North America

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with topical therapy and alternative medicine modalities for psoriasis severity measures (https://www.aad.org/member/clinical-quality/guidelines/psoriasis)

Published by: American Academy of Dermatology **Last published:** 2021

Joint AAD-NPF guidelines of care for the management of psoriasis with systemic non-biological therapies (https://www.aad.org/member/clinical-quality/guidelines/psoriasis)

Published by: American Academy of Dermatology **Last published:** 2020

Joint AAD–NPF guidelines of care for the management and treatment of psoriasis in pediatric patients (https://www.aad.org/member/clinical-quality/guidelines/psoriasis)

Published by: American Academy of Dermatology **Last published:** 2020

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with phototherapy (https://www.aad.org/member/clinical-quality/guidelines/psoriasis)

Published by: American Academy of Dermatology **Last published:** 2019

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with biologics (https://www.aad.org/member/clinical-quality/guidelines/psoriasis)

Published by: American Academy of Dermatology **Last published:** 2019

Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities (https://www.aad.org/member/clinical-quality/guidelines/psoriasis)

Published by: American Academy of Dermatology **Last published:** 2019

Treatment targets for plaque psoriasis (https://www.jaad.org/article/S0190-9622(16)30909-4/fulltext)

Published by: National Psoriasis Foundation **Last published:** 2017

Online resources

1. Psoriasis Association (https://www.psoriasis-association.org.uk) *(external link)*

2. National Psoriasis Foundation (US) (https://www.psoriasis.org) *(external link)*

Evidence tables

How do topical corticosteroids affect outcomes in people with scalp psoriasis?

This table is a summary of the analysis reported in a Cochrane Clinical Answer that focuses on the above important clinical question.

Cochrane Clinical Answers

View the full source Cochrane Clinical Answer (https://www.cochranelibrary.com/cca/doi/10.1002/cca.1344/full)

Evidence A * — Confidence in the evidence is high or moderate to high where GRADE has been performed and the intervention is more effective/beneficial than the comparison for key outcomes.

Population: Adolescents and adults with scalp psoriasis
Intervention: Topical corticosteroid [a]
Comparison: Topical vitamin D (calcipotriol) [a]

Outcome	Effectiveness (BMJ rating)†	Confidence in evidence (GRADE)‡
Severity score (total sign score [TSS])	See Notes [b]	GRADE assessment not performed for this outcome
Clearance of symptoms (investigator assessed)	Favours intervention	Moderate
Clearance of symptoms (patient assessed)	Favours intervention	GRADE assessment not performed for this outcome
Response to treatment (investigator assessed)	Favours intervention	High
Response to treatment (patient assessed)	Favours intervention	Moderate
At least one adverse event	Occurs more commonly with topical vitamin D compared with topical corticosteroids (favours intervention) [c]	GRADE assessment not performed for this outcome
At least one adverse event	No statistically significant difference [d]	GRADE assessment not performed for this outcome
Withdrawals due to adverse events	Occurs more commonly with topical vitamin D compared with	Moderate

Outcome	Effectiveness (BMJ rating)†	Confidence in evidence (GRADE)‡
	topical corticosteroids (favours intervention)	
Disease-free period, duration of response, quality of life	-	None of the studies identified by the review assessed these outcomes

Note

[a] This evidence table summarises the findings for the comparison of topical corticosteroids versus topical vitamin D, which is the main comparison as stated in the Cochrane review Summary of Findings table. See the full Cochrane Clinical Answer (CCA) for information on other comparisons (topical corticosteroid versus placebo; topical corticosteroid plus vitamin D versus placebo; topical corticosteroid plus vitamin D versus topical vitamin D).

[b] Results reported narratively (five RCTs; all trials reported a greater reduction in TSS with topical corticosteroids compared with topical vitamin D).

[c] At least one adverse event occurred more commonly with topical vitamin D when compared with the following three corticosteroids: 1 mg/mL betamethasone valerate solution; 0.5 mg/g betamethasone dipropionate gel; and 0.05% clobetasol propionate shampoo). Results reported separately as subgroup analyses.

[d] No statistically significant difference was found when comparing 0.05% clobetasol propionate solution with topical vitamin D; result reported as a subgroup analysis.

* Evidence levels

The Evidence level is an internal rating applied by BMJ Best Practice. See the EBM Toolkit (https://bestpractice.bmj.com/info/evidence-tables/) for details.

Confidence in evidence

A - High or moderate to high

B - Moderate or low to moderate

C - Very low or low

† Effectiveness (BMJ rating)

Based on statistical significance, which demonstrates that the results are unlikely to be due to chance, but which does not necessarily translate to a clinical significance.

‡ Grade certainty ratings

High	The authors are very confident that the true effect is similar to the estimated effect.
Moderate	The authors are moderately confident that the true effect is likely to be close to the estimated effect.
Low	The authors have limited confidence in the effect estimate and the true effect may be substantially different.
Very Low	The authors have very little confidence in the effect estimate and the true effect is likely to be substantially different.

BMJ Best Practice EBM Toolkit: What is GRADE? (https://bestpractice.bmj.com/info/toolkit/learn-ebm/what-is-grade/)

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Images

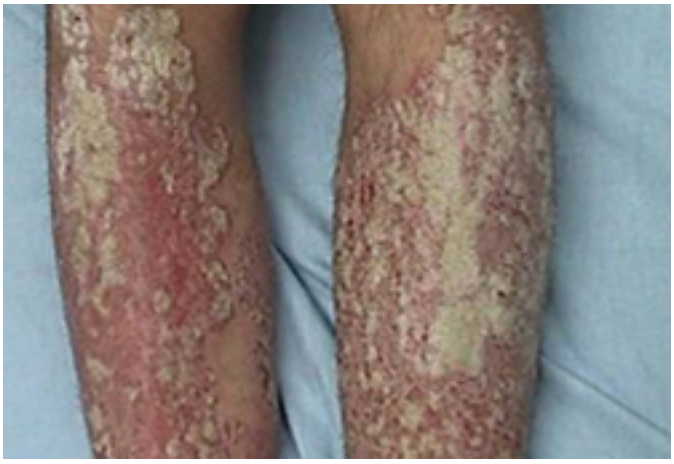

Figure 1: Plaque psoriasis on legs

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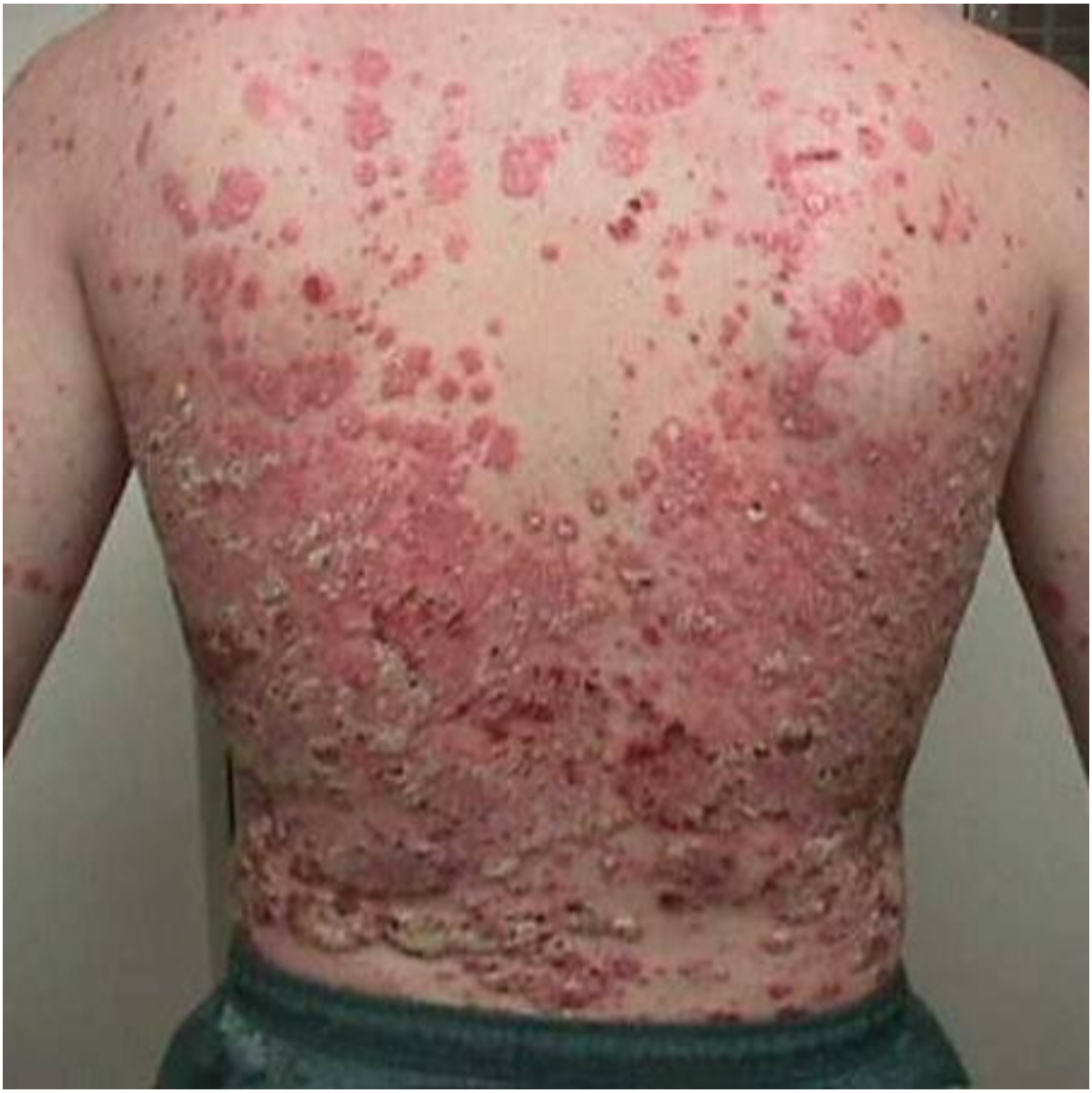

Figure 2: Plaque psoriasis on back

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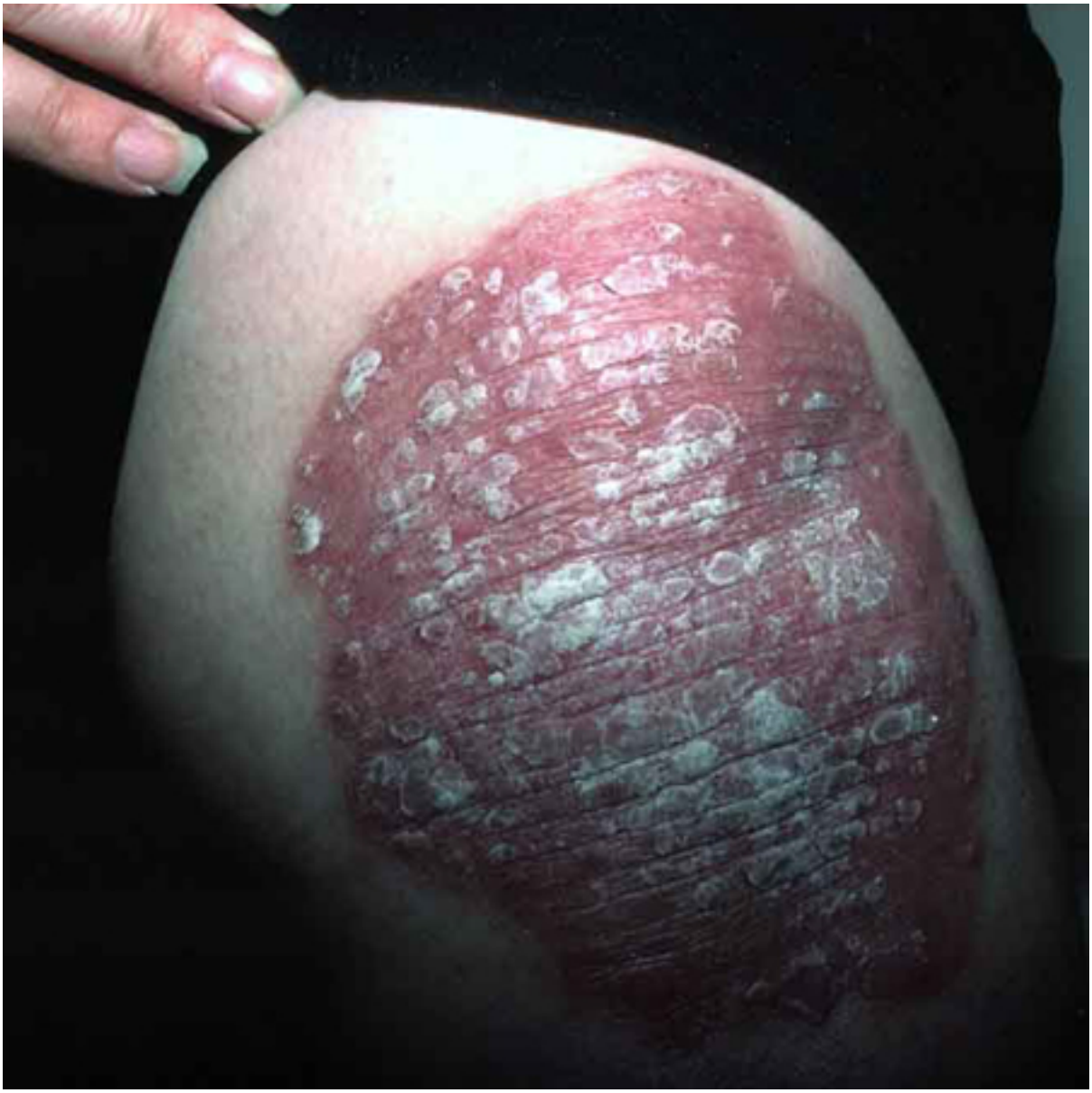

Figure 3: Plaque psoriasis on knee

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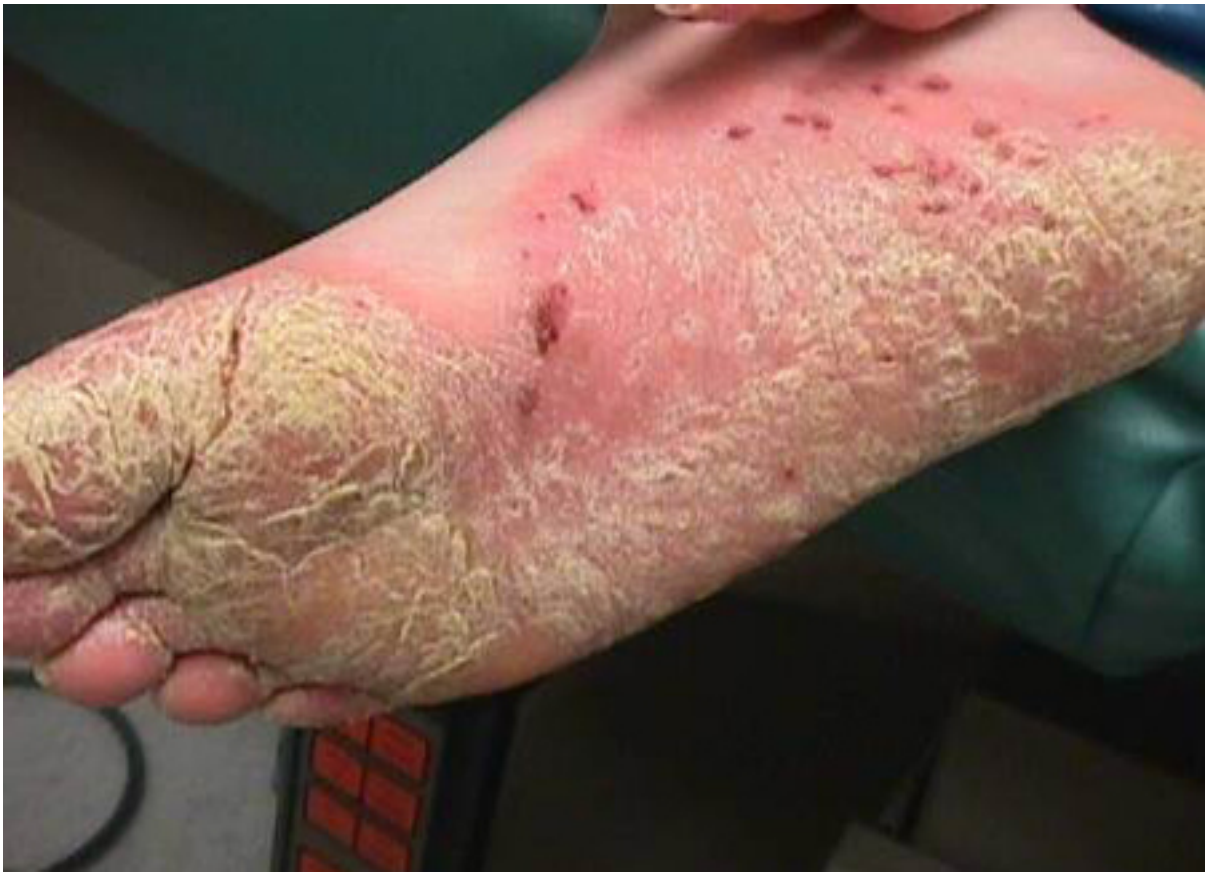

Figure 4: Plaque psoriasis on foot

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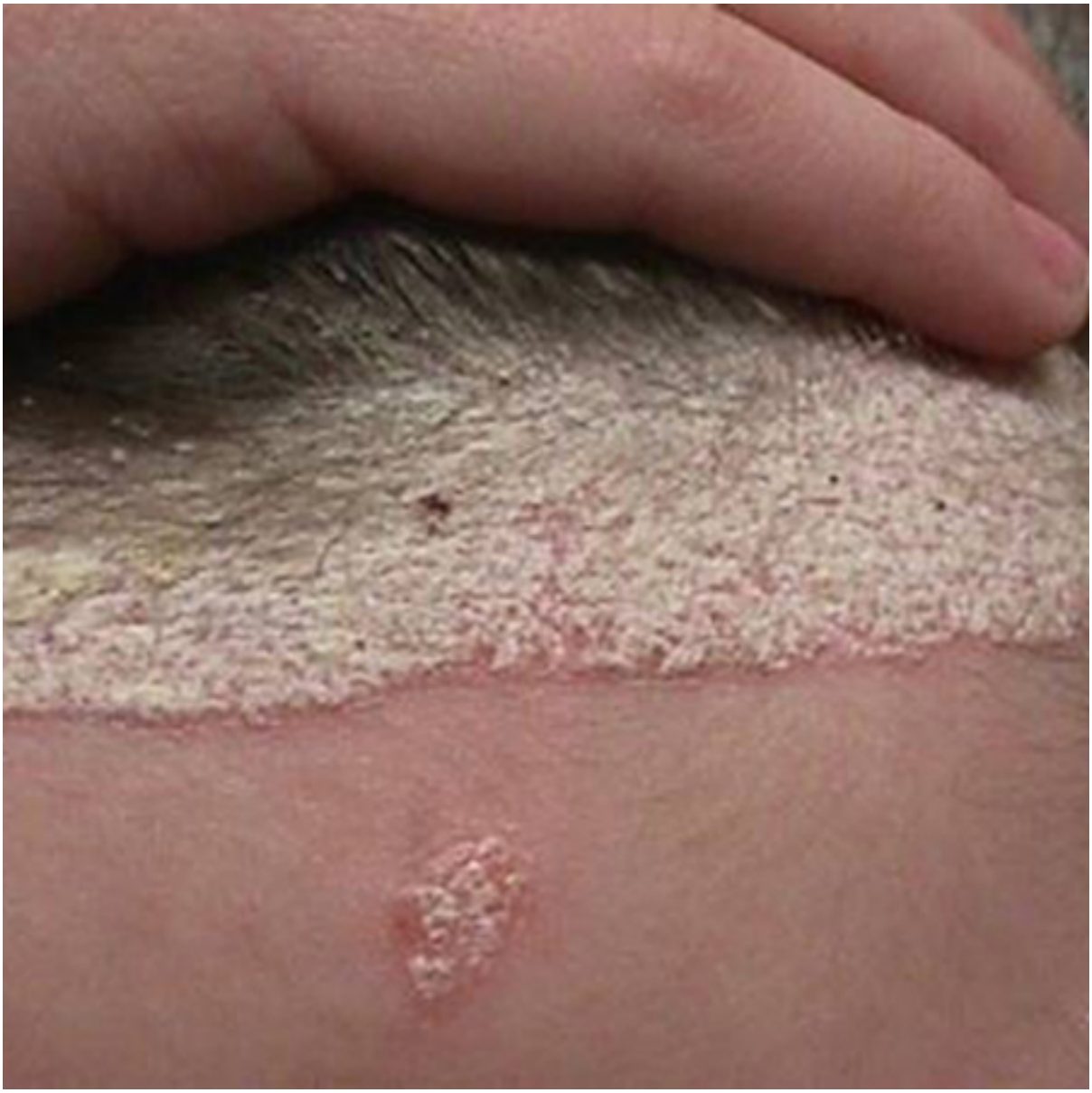

Figure 5: Plaque psoriasis on scalp

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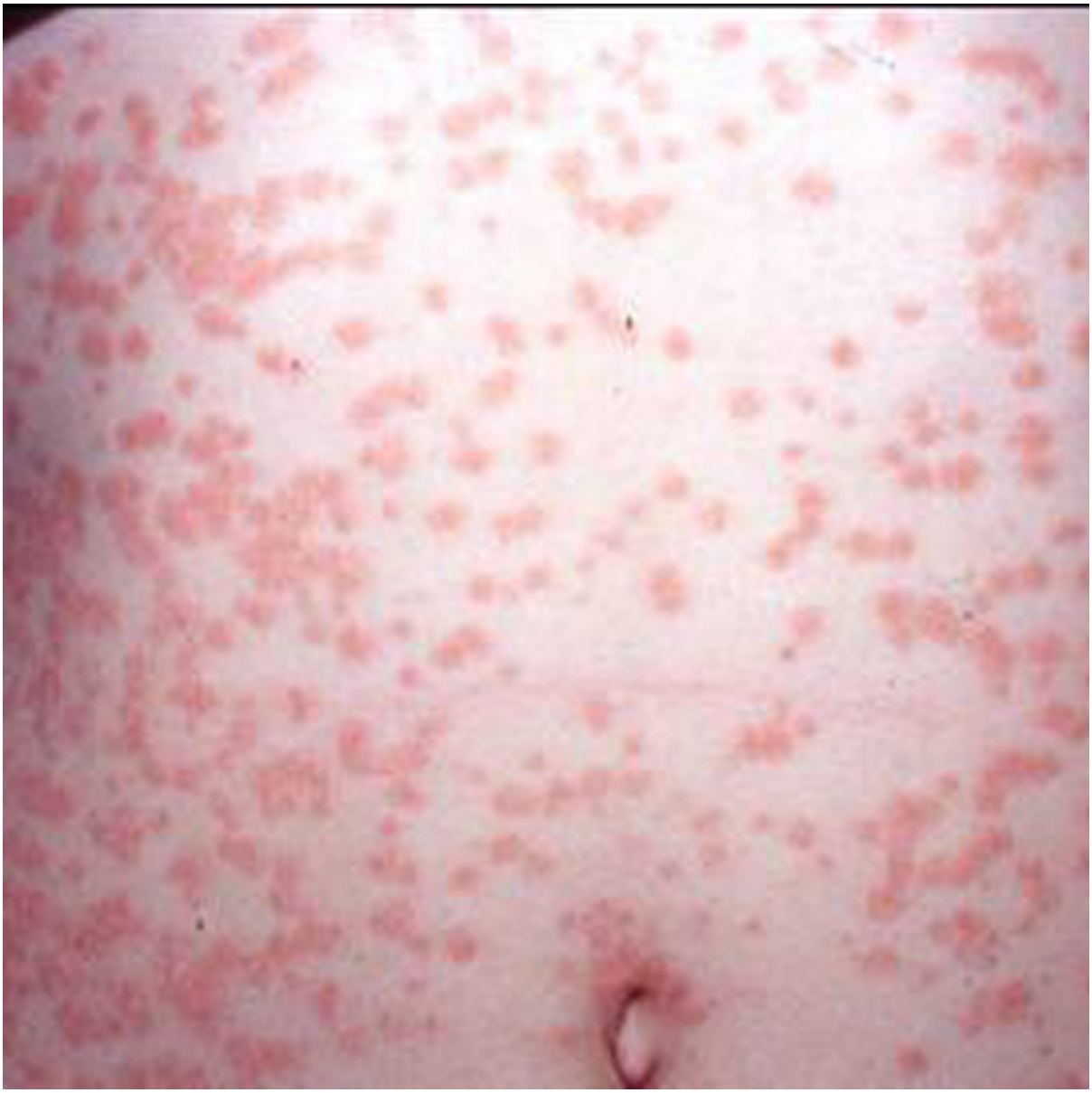

Figure 6: Guttate psoriasis

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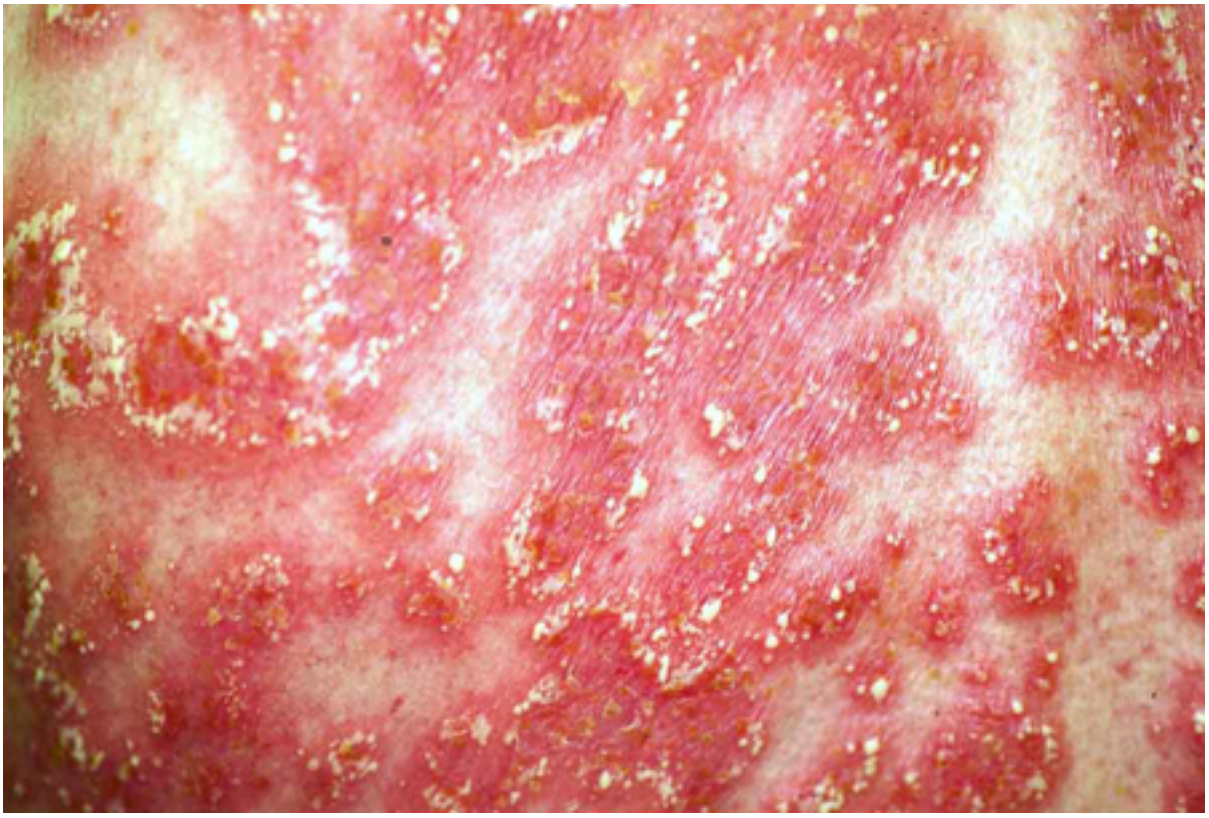

Figure 7: Pustular psoriasis

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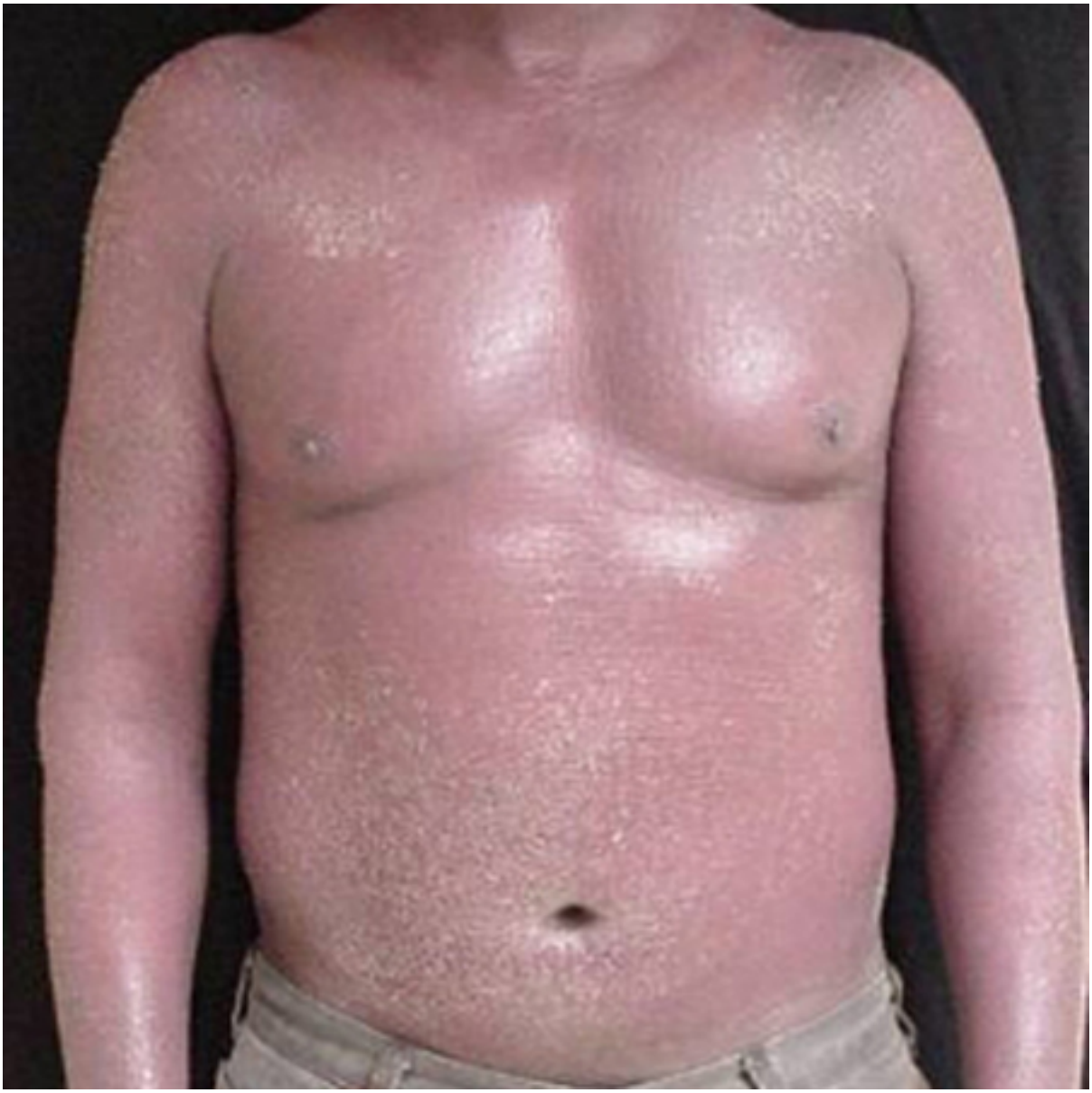

Figure 8: Erythroderma

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Figure 9: Psoriatic arthritis

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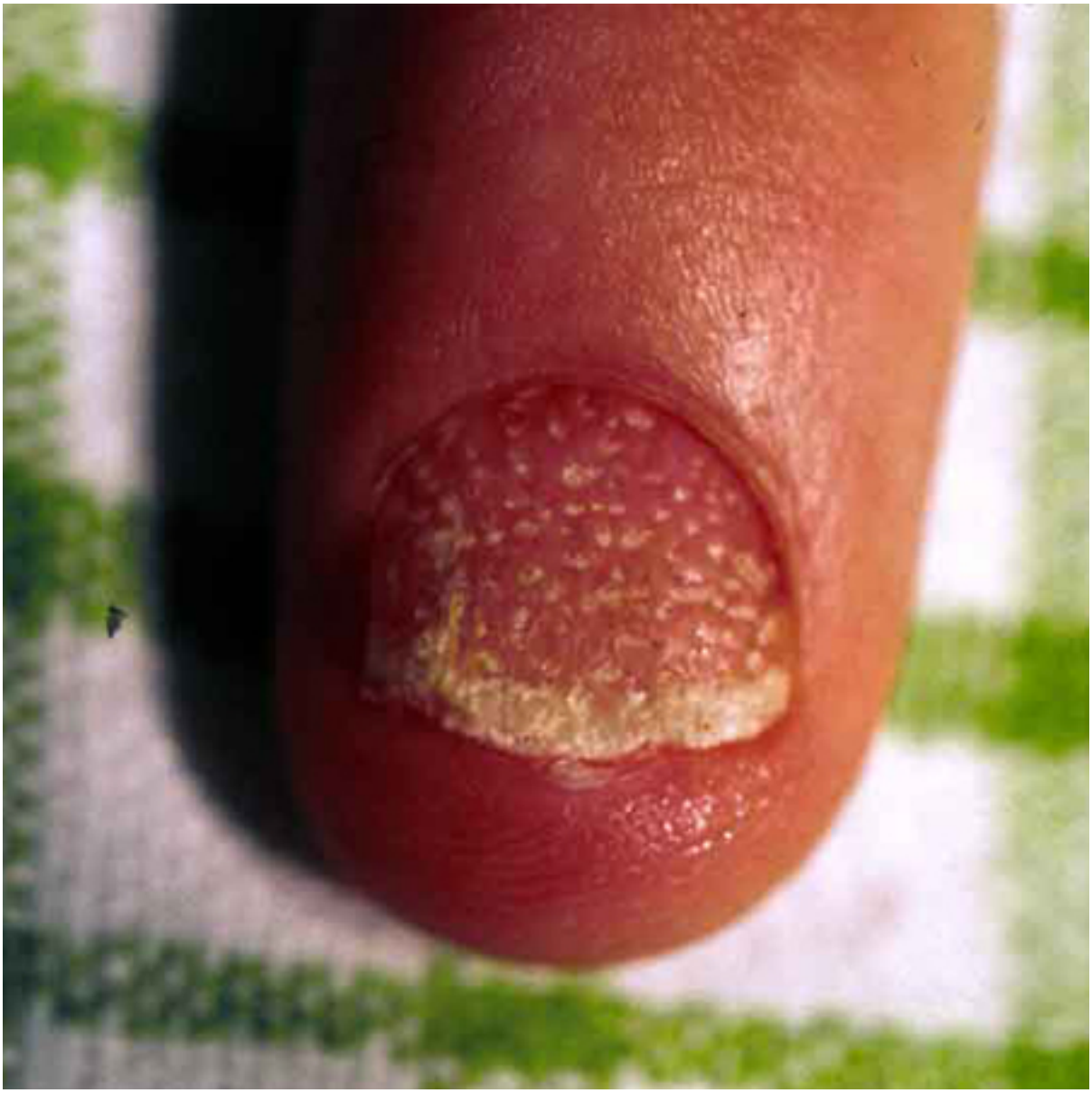

Figure 10: Nail psoriasis - pitted nails

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Figure 1 – BMJ Best Practice Numeral Style

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