CLINICAL MANAGEMENT extra

Diagnosis and Management of Cutaneous Psoriasis: A Review

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GENERAL PURPOSE:

To provide information about the diagnosis and management of cutaneous psoriasis.

TARGET AUDIENCE:

This continuing education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES:

After completing this continuing education activity, the provider should be better able to:

1. Describe the epidemiology, pathophysiology, clinical presentation, assessment, and diagnosis of the types and subtypes of psoriasis.

2. Explain the use of topical treatments, intralesional steroids, phototherapy, conventional systemic treatments, biologic agents, and pain medicines for psoriasis.

ABSTRACT

Psoriasis is a chronic inflammatory disease that is characterized by plaque, inverse, guttate, pustular, and erythrodermic variants. This review focuses on the epidemiology, diagnosis, and treatment of cutaneous psoriasis. Other related topics discussed include peristomal psoriasis, the Koebner phenomenon, and the relationship between biologic therapy and wound complications.

KEYWORDS: biologic therapy, cutaneous psoriasis, erythrodermic psoriasis, intertriginous psoriasis, Koebner phenomenon, psoriasis, psoriasis vulgaris, pustular psoriasis, topical corticosteroids

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INTRODUCTION

Psoriasis is a chronic inflammatory disease, with a reported prevalence of 1% to 3% in Europe and the US.[1] It may present at any age, but has a bimodal distribution of first presentation at between 15 to 20 and 55 to 60 years of age. Younger age at onset is associated with more severe disease and a family history affecting more family members.[2] In general, approximately 36% of patients have a family history of psoriasis, and multiple genetic susceptibility loci have been identified.[3,4]

Metabolic syndrome (three of five components: central obesity, high blood pressure, high blood sugar, high serum triglycerides, and low serum high-density lipoprotein)[5] has been associated with psoriasis. Psoriasis is also associated with chronic obstructive pulmonary disease, nonalcoholic fatty liver disease, and coronary artery disease.[6–8] Persons with psoriasis may also have a significantly decreased quality of life and psychological burden including anxiety, depression, and suicidal thoughts and behavior.[2,9,10] Reviewing this article will facilitate provider knowledge of psoriasis pathophysiology, diagnosis, and management.

PATHOPHYSIOLOGY

Psoriasis is a chronic autoimmune disease with multiple leukocytes and cytokines interacting to produce the disease process (Figure 1). The inflammatory cascade of psoriasis begins when antigens in the skin activate dendritic cells and neutrophils, which release cytokines including tumor necrosis factor α (TNF-α), interleukin 23 (IL-23), and IL-12. These cytokines participate in positive feedback loops by activating leukocytes, which then release more cytokines, resulting in continuous inflammation. For example, IL-23 converts cluster of differentiation 4–positive cells into T-helper 17 (T_H17) cells that release IL-17A; T_H17 cells and IL-17A act to upregulate TNF-α. These cytokines also exert effects on the skin with IL-17A, IL-20, and IL-22, and TNF-α contributing to the modified keratinocyte function and the T_H17 cells promote angiogenesis.[11,12]

CLINICAL PRESENTATION

Psoriasis is a relapsing-remitting disease that often improves with warmer weather and relapses during stressful life events or in conjunction with infections. Common presentations include (Figure 2):

1. Plaque psoriasis, with elevated areas of more than 1 cm. This is the most common subtype and presents with well-demarcated annular lesions comprising an erythematous base and thick silvery scale. These lesions are often found on the extensor surfaces (elbows, knees), scalp, lumbosacral area, and intergluteal cleft.
2. Inverse psoriasis, seen in the body folds. Also called flexural psoriasis, it is characterized by red shiny lesions devoid of scale in the inframammary, perineal, and axillary areas.
3. Guttate psoriasis, presenting as teardrop-shaped lesions. Acute guttate psoriasis is often preceded by a sore throat associated with group B streptococcal infection, and consists of multiple, small (2–10 mm) psoriatic lesions, most often on the trunk.
4. Erythrodermic psoriasis, in which 90% or more of the body is red. This variant consists of complete or almost complete involvement of the skin and is characterized by gradual coalescence of plaques caused by infection, drugs, systemic disease, or withdrawal of corticosteroids.
5. Generalized pustular psoriasis manifests as multiple uniform sterile pustules on the body and is often accompanied by fever. It may also be precipitated by withdrawal of systemic corticosteroids or infection and represents unstable disease that often requires hospitalization.
6. Palmoplantar pustulosis psoriasis presents on the hands and feet as sterile pustules on a base of erythema and scale.[2,13]

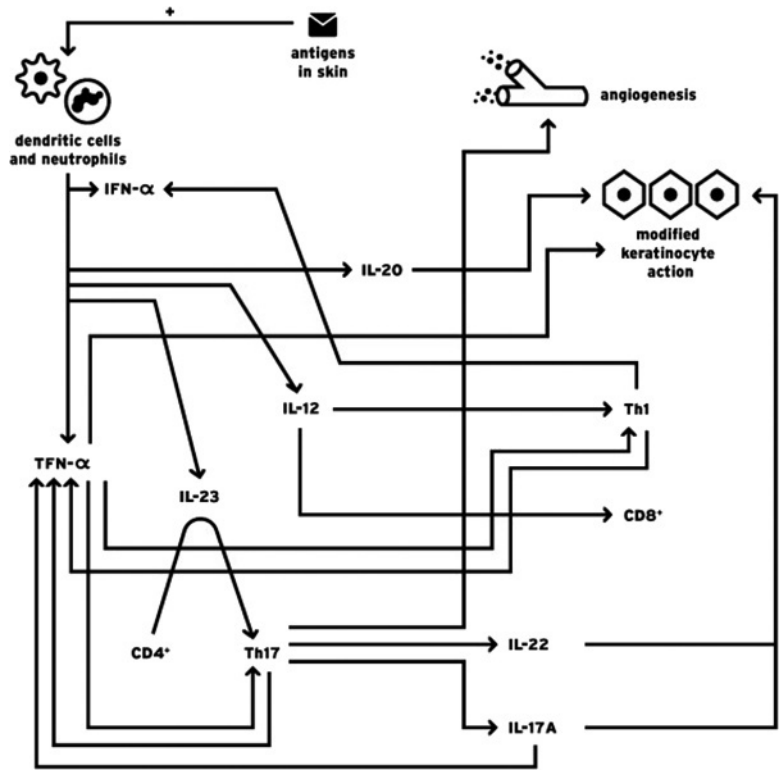
Extracutaneous manifestations of psoriasis include nail abnormalities and psoriatic arthritis. About 80% of patients with psoriasis have nail involvement including pitting (small depressions on the nail surface), onycholysis (distal nail separation from the nail bed), subungual hyperkeratosis, and orange-yellow spots beneath the nail plate (oil spots).[2,13]

The prevalence of psoriatic arthritis among patients with cutaneous psoriasis is 30%,[14,15] with patients developing arthritis an average of 12 years after the onset of cutaneous psoriasis.[16] There are five forms of psoriatic arthritis. From most common to least, these include distal oligoarthritis (inflammation that involves four or fewer joints), rheumatoid factor–negative polyarthritis, arthritis mutilans (resorption and shortening of finger bones), sacroiliitis, and ankylosing spondylitis.[13] The most common form of arthritis affects the distal joints of the digits in an asymmetric pattern. The soft tissue may become swollen, producing sausage-like digits called dactylitis.[2,13]

ASSESSMENT AND DIAGNOSIS

The diagnosis of psoriasis is usually clinical. The physical examination should include an examination of the primary lesion and other

Figure 1.
THE BASIC PATHOPHYSIOLOGY OF PSORIASIS



common areas affected by psoriasis including the scalp. The nails and joints should be examined for any changes consistent with psoriasis, and a family history should be taken to further elucidate the diagnosis.[13] Diagnosis can be further supported by the Auspitz sign or Koebner phenomenon. The Auspitz sign occurs because an excess of small surface capillaries results in multiple bleeding points when the silver-gray scale is lifted off.[17] The Koebner phenomenon consists of the appearance of psoriatic lesions on previously normal skin because of prior trauma; clinical psoriasis lesions appear after 7 days or more.[18] This phenomena may cause psoriatic lesions to appear around wound sites, under dressings, and around ostomy sites. Finally, if there is still doubt about the diagnosis, a simple punch biopsy can be performed.[13]

Chronic Plaque Psoriasis

Classification of plaque psoriasis severity can guide appropriate treatment. Commonly used tools for classification of plaque psoriasis include the Psoriasis Area and Severity Index (PASI), body surface area (BSA), and the Dermatology Life Quality Index, with a score of more than 10 on each of these parameters indicating moderate to severe psoriasis. Treatment may also be guided by the location of the plaques, associated pruritus, functional and psychosocial limitations, associated psoriatic arthritis, nail or scalp psoriasis, previously prescribed treatments, and patient preference.[13,19]

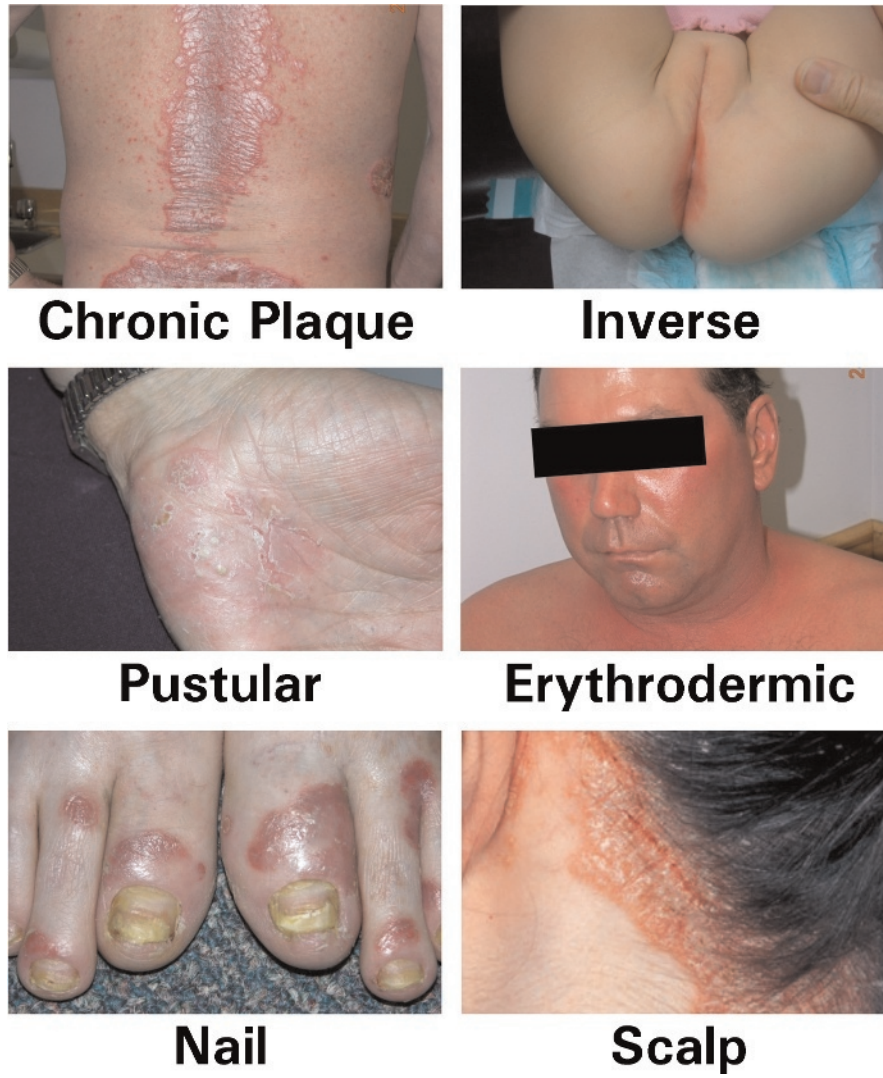
The PASI score is a formula calculated based on BSA affected, erythema, thickness of the plaques, and amount of scale, with each criterion rated on a 0- to 4-point scale. The PASI is often used to measure response rate, with PASI 90 scores (meaning a 90% reduction in severity from baseline) being a common target.[13,19] The most common method to estimate BSA is with the patient's full handprint (including the fingers) equating to 1% of the total BSA.[20]

TREATMENT

Topical Treatment

Mild disease may be effectively treated with topical therapies, including corticosteroids, vitamin D derivates, retinoids, tar,

Figure 2.
SKIN, NAIL, AND SCALP INVOLVEMENT WITH PSORIASIS VULGARIS



keratolytic agents that break down scale (urea, salicylic acid, α-hydroxy acid), and emollient moisturizers (Table 1). The choice of topical agent depends on anatomical area, size and thickness of the plaque, and whether the agent is being used for initiation or maintenance therapy.

A combination product of betamethasone dipropionate and calcipotriol is recommended to initiate treatment on the trunk or extremities because this preparation is more efficacious than monotherapy.[21] This product is too strong for the face or folds. When disease control has been established, vitamin D derivates are recommended for maintenance therapy. Further, thick plaques (clinical thickness >0.75 mm) respond to keratolytic agents including salicylic acid or urea, the use of emollients (lubricating moisturizers), and higher-strength topical corticosteroids (ointment formulation).[21]

The choice of topical corticosteroid depends on the anatomical location of the plaque, the thickness of the plaque, and the age of the patient. For thick plaques on the trunk or limbs, mid- to high-potency corticosteroids should be used. For infants and young children, body folds, and the face, low- to mid-potency corticosteroids should be used. The palms and soles require high- to very high-potency corticosteroids (Table 2).[13]

Table 1.
COMMON TOPICAL THERAPIES FOR PSORIASIS

Therapy Category	Mechanism of Action	Adverse Effects	Evidence Level
Corticosteroids	Anti-inflammatory[64]	Use of potent and superpotent class should be limited to 2–4 wk; inappropriate use may cause skin atrophy, contact dermatitis, rebound plaques, and systemic adverse effects[19]	A
Vitamin D analogs	Normalization of keratinocyte function and anti-inflammatory[64]	FDA Category C; usually well tolerated; most common adverse reaction is irritant contact dermatitis[19,65]	A
Tazarotene 0.05%–0.1%	Normalization of keratinocyte function and anti-inflammatory[64]	FDA Category X;[a] most common adverse effect is irritant contact dermatitis[66]	A
Salicylic acid 3%–10%	Degrades the stratum corneum by dissolving intracellular components holding keratinocytes together[11]	Concentration of ≥10% and application on ≥20% of body surface area may cause systemic adverse effects including metabolic acidosis and nausea[67]	C
Coal tar/LCD (20% LCD = 4% crude coal)	Decreased keratinocyte proliferation[19]	Irritant and allergic contact dermatitis, staining of clothes and furniture; possible increased risk of nonmelanoma skin cancer[24]	A
Calcineurin inhibitors (tacrolimus, pimecrolimus)	Anti-inflammatory[19]	FDA black box warning of increased lymphoma risk; however, no clinical or epidemiological evidence of increased risk;[68] usually well tolerated;[19] relatively safe, but use small quantities with caution during pregnancy	A

Abbreviation: LCD, liquor carbonis detergens.
[a]Fetal abnormalities have been demonstrated in animals or humans according to the FDA.

Vitamin D analogs (calcipotriol, calcitriol, and tacalcitol) are more efficacious than topical vitamin A derivatives (retinoids), coal tar, and placebo for trunk and limb psoriasis.[22] Further, the combination of a potent topical corticosteroid and betamethasone dipropionate once daily is more efficacious than topical corticosteroids, coal tar, or vitamin D analogs alone and topical retinoid once daily.[22] In addition to their efficacy, vitamin D analogs have a good long-term safety profile, making them an ideal choice for long-term maintenance therapy and for use in combination with phototherapy.[21,23]

Topical calcineurin inhibitors are currently available as tacrolimus ointment (0.03% and 0.1%) and pimecrolimus cream (1%). While there is no FDA approval for their use in psoriasis, they may be used as topical steroid-sparing medication for the acute and maintenance treatment of plaques on the face, genital, or intertriginous areas. Unlike topical corticosteroids, prolonged use does not result in increased absorption and thinning of the skin.[21,24]

Tazarotene is a topical retinoid (vitamin A derivative). There is limited evidence for its efficacy as a monotherapy often because of contact irritation. Therefore, it is usually used in combination with a topical corticosteroid; that said, topical retinoids should be avoided by pregnant women.[13,22]

Emollients limit the evaporation of water from the skin, increasing stratum corneum hydration. Keratolytic agents including salicylic acid, urea, and α-hydroxy acids (glycolic acid and lactic acid) may have some clinical benefit for hyperkeratotic psoriasis lesions, including the reduction of erythema, desquamation, and pruritus. The efficacy of salicylic acid to reduce scale is supported by the greatest amount of evidence, and it is most commonly used for this indication.[25] A further benefit of keratolytic agents is the associated increased penetration of other active topical agents.[13,21,25]

Coal tar is considered an alternative topical therapy for psoriasis, and may be beneficial when treatments such as corticosteroids and vitamin D analogs do not produce desired results. It is also a component in shampoos. Patients may be wary of the messiness, staining, and folliculitis associated with coal tar that limits its use.[26]

Intralesional Steroids

Intralesional steroid injections may be used for recalcitrant psoriasis plaques. Although to the authors' knowledge there are no intralesional psoriasis clinical studies published since the 1960s, clinical experience suggests almost 100% efficacy for small

Table 2.
RECOMMENDED CORTICOSTEROIDS BY BODY AREA

Application	Corticosteroids (Vehicles)
Face, body folds, and infants/young children	Hydrocortisone 1% (cream, lotion)
	Desonide 0.05% (cream, lotion)
	Mometasone furoate 0.1% (cream, lotion)
Plaques on trunk and limbs	Betamethasone valerate 0.1% (cream, ointment)
	Fluocinonide 0.05% (cream, ointment)
	Triamcinolone acetonide 0.5% (cream)
Palms and soles	Clobetasol propionate 0.05% (cream, ointment)
	Betamethasone dipropionate 0.05% (cream, ointment)
Scalp	Betamethasone valerate 0.1%, 0.05% (lotion)
	Fluocinonide 0.05% (gel)
	Mometasone furoate 0.1% (lotion)

plaques. Doses of up to 25 mg of total intralesional triamcinolone (usually injected as 4–8 mg/mL) are unlikely to have systemic effects.[27] Triamcinolone acetonide can be diluted with sterile saline or 1% lidocaine. Ethyl chloride spray may also be used before injection to reduce pain.[28]

Phototherapy

Photochemotherapy modalities commonly used to treat psoriasis include narrowband ultraviolet B (NB-UVB; 311–313 nm), broadband ultraviolet B (BB-UVB; 280–320 nm), targeted or excimer UVB laser (308 nm) and a combination treatment of oral or topical 8-methoxypsoralen and UVA (PUVA; 320–400 nm).[29] Initiation of BB-UVB, NB-UVB, or PUVA is usually considered when at least 10% of the BSA is involved or in patients who have not responded to topical therapies. In contrast, the excimer laser may be utilized as a third-line treatment of localized or treatment resistant lesions.[29,30] Both NB-UVB and PUVA are similarly efficacious; however, NB-UVB is the most commonly used first-line photo(chemo)therapy because of a decreased photodamage profile.[29–32]

Ultraviolet light exerts its effects by inhibiting the ability of epidermal Langerhans cells to present antigens to T cells, thus downregulating the immune response.[29] The PUVA therapy works as photochemotherapy by crosslinking DNA and inducing apoptosis.[29] Absolute contraindications to phototherapy include systemic lupus erythematosus, xeroderma pigmentosum, and porphyria. Relative contraindications include a history of skin cancer, extensive photodamage, immunosuppression, and use of photosensitizing medication other than the prescribed topical or systemic psoralens.[13,31] Phototherapy is usually well tolerated. However, acute adverse effects may include erythema, a burning sensation, blisters, and pruritus.[33] In addition, PUVA may be associated with an increased risk of skin cancer because the psoralens act as a photosensitizer and UVA penetrates deeper into the skin.[34,35]

Conventional Systemic Treatments

Conventional systemic treatments are usually initiated when 10% or more of the BSA is affected, when the psoriasis has a debilitating effect on the patient's quality of life (eg, involvement of the palms or soles), or when response to topical treatments and phototherapy is not sufficient.[13] The most commonly used conventional systemic medications include methotrexate, cyclosporine, acitretin, and sulfasalazine (Table 3). Methotrexate, cyclosporine, and acitretin are first-line systemic agents. Sulfasalazine and apremilast, among others, may be used when treatment with first-line systemic agents does not produce the desired effect, first-line therapy is contraindicated, or the medication causes undesirable adverse reactions.[36]

A 2018 review reported a strong consensus statement on the use of methotrexate and cyclosporine for first-line systemic induction therapy. Absolute contraindications to methotrexate include hepatic impairment (excessive alcohol consumption and active hepatitis B or C), pregnancy, and tuberculosis or other active infection.[37] Benefits of methotrexate include evidence of efficacy for psoriatic arthritis[36] and the best safety profile out of all commonly used systemic agents and biologic therapies.[38] Absolute contraindications to cyclosporine include renal impairment, uncontrolled hypertension, active tuberculosis or other infection, and some current and past malignancies. Further, cyclosporine is activated through the CYP3A4 pathway, resulting in multiple drug interactions.[37] A benefit of cyclosporine is the fast onset of action.[36]

The previously mentioned review concluded that acitretin is less efficacious than both methotrexate and cyclosporine for chronic plaque psoriasis. Further, acitretin has teratogenic potential; therefore, in women of childbearing age, it should be started on the second to third day of a menstrual period. Contraception should be initiated 1 month prior to and continued up to 3 years after discontinuation of the acitretin. Moreover, acitretin is contraindicated for women who are breastfeeding and patients with severe renal or hepatic dysfunction.[37] Acitretin and UVB phototherapy combination treatment may be used, especially for patients with thicker plaques, and results in lower doses of both acitretin and UVB.[29] However, acitretin (and the oral biologic agent apremilast) does not cause immunosuppression that is associated with methotrexate, cyclosporine, or the other biologic agents.[36]

Table 3.
TRADITIONAL SYSTEMIC MEDICATIONS FOR PSORIASIS

Medication	Mechanism of Action	Benefits	Common Dosing	Adverse Effects	Evidence Level
Methotrexate	Inhibits production of proliferating cells such as lymphocytes[35]	Some evidence for psoriatic arthritis[35] and best safety profile of systemic agents[37]	Once weekly 7.5–25 mg + 1–5 mg folic acid daily on other days[35]	FDA pregnancy category X;[a] hepatotoxicity, bone marrow suppression, pulmonary toxicity[35]	A
Cyclosporine	Inhibits immune response through inhibition of calcineurin[35]	Fast acting[35]	3–5 mg/kg daily[35]	Renal toxicity, hypertension, limit continuous long-term use[35]	A
Acitretin	Modulates epidermal differentiation and has anti-inflammatory effects[35]	Evidence for generalized pustular and erythrodermic psoriasis and patients who are HIV-positive[35]	10–50 mg/d[35] with meals	FDA pregnancy category X,[a] hypertriglyceridemia, dryness of oral and nasal mucosa, brittle nails, alopecia, hepatotoxicity[35]	A
Sulfasalazine	Exact mechanism unclear; has anti-inflammatory effects[35]	Low adverse effects profile; some evidence for psoriatic arthritis[11,35]	1–4 g daily[35]	Rash, nausea[35]	A

Abbreviation: HIV, human immunodeficiency virus.
[a]Fetal abnormalities have been demonstrated in animals or humans according to FDA
[b]No FDA approval in the US
[c]No FDA pregnancy category because not approved for use in the US

Biologic Agents

Over the past decade, several biologics have been licensed in the US and Canada as safe and effective treatments for moderate to severe plaque psoriasis (Table 4). Currently anti–IL-17 agents (secukinumab brodalumab, ixekizumab), anti–IL-23 inhibitors (guselkumab), and anti–IL-12-23 inhibitors (ustekinumab) are commonly used agents for plaque psoriasis and psoriatic arthritis. The anti-TNF agents (adalimumab, etanercept, infliximab, certolizumab pegol) were first on the market and may still be used for psoriatic arthritis, but newer agents have replaced the TNF inhibitors for increased efficacy in plaque psoriasis.

The choice of biologic is often difficult for the provider and the patient.[39–41] Patients requiring systemic treatment may be offered biologics based on criteria including the failure of two or more conventional systemic treatments. The efficacy, safety, and ease of administration of each particular agent should be balanced against the coverage provided by the patient's healthcare insurance.[13]

Interleukin 17A plays a critical role in the pathophysiology of psoriasis, and it is the target of many newly developed biologic agents, including secukinumab, ixekizumab, and brodalumab. Unlike secukinumab and ixekizumab, which bind to IL-17A itself, brodalumab targets the IL-17A receptor on keratinocytes and immune cells, providing a more direct therapeutic target.[42] Infliximab, adalimumab, certolizumab pegol, and etanercept target TNF-α.[43] Ustekinumab prevents IL-12 and IL-23 from stimulating receptor complexes by binding to the p40 subunit common to both cytokines.[44] In contrast, guselkumab targets only target IL-23 by binding to its p19 subunit.[45]

A 2017 Cochrane review (prior to the marketing of some of the newer agents) concluded that ustekinumab, infliximab, and certolizumab have the best combination of efficacy and safety when prescribed for plaque psoriasis. Ixekizumab had the highest efficacy in terms of reaching PASI 90, whereas certolizumab had the lowest relative risk of serious adverse events.[38]

There are several safety considerations that are associated with biologic agents. All patients starting biologics should be given the opportunity to take part in long-term saferty registries. Live vaccines should be avoided in patients on biologic therapies and infants (up to 6 months of age) born to mothers taking biologic therapy beyond 16 weeks' gestation. Special care should be taken when prescribing biologics to patients with a history of cancer, particularly in the past 5 years, and patients starting biologic therapy should be tested for infection with hepatitis B and C, human immunodeficiency virus, varicella-zoster antibody, and latent tuberculosis (preferably with an interferon γ release assay).[19,46]

Table 4.
BIOLOGIC AGENTS FOR MODERATE TO SEVERE PLAQUE PSORIASIS

Biologic Agent	Target	Adverse Effects[a]
Secukinumab	IL-17A	Nasopharyngitis, upper respiratory tract infection, other infection, nausea, diarrhea, urticaria, rhinorrhea
Ixekizumab	IL-17A	Injection site reaction, upper respiratory tract infection, tinea infection, other infections, nausea
Brodalumab	IL-17A receptor	Suicidal ideation, arthralgia, headache, fatigue, diarrhea, oropharyngeal pain, nausea, myalgia, injection site reactions, influenza, neutropenia, and tinea infections
Infliximab	TNF-α	Serious infections, malignancy, hepatotoxicity, cytopenia, hypersensitivity reactions, cardiovascular events, cerebrovascular events, neurologic disorders, lupus-like syndrome, reactivation of latent hepatitis B infection, infusion-related reactions, headache, and abdominal pain
Adalimumab	TNF-α	Serious infections, malignancy, hypersensitivity reactions, cytopenia, neurologic disorders, congestive heart failure, lupus-like syndrome, reactivation of latent hepatitis B infection, injection site reactions, headache, and rash
Etanercept	TNF-α	Serious infections, malignancy, neurologic disorders, congestive heart failure, pancytopenia, hypersensitivity reactions, lupus-like syndrome, autoimmune hepatitis, injection site reaction, and infections, reactivation of latent hepatitis B infection
Cetrolizumab pegol	TNF-α	Serious infection, congestive heart failure, malignancy, hypersensitivity reaction, hepatitis B virus reactivation, neurologic reactions, pancytopenia, lupus-like syndrome, upper respiratory tract infections, rash, and urinary tract infections
Ustekinumab	P40 subunit of IL-12 and IL-23	Infection, reactivation of latent infection, nonmelanoma skin cancer, hypersensitivity reactions, posterior leukoencephalopathy syndrome, noninfectious pneumonia, nasopharyngitis, upper respiratory tract infection, headache, and fatigue
Guselkumab	P19 subunit of IL-23	Upper respiratory tract infections, gastroenteritis, tinea infections, herpes simplex, headache, injection site reactions, arthralgia, diarrhea
Apremilast	Inhibits phosphodiesterase-4 resulting in a build-up of cyclic adenosine monophosphate and a downregulation of the immune response[69]	Gastrointestinal adverse effects, weight loss, depression, headaches[69]

Abbreviations: IL, interleukin; TNF-α, tumor necrosis factor α.
[a]Adverse effects listed obtained from pharmaceutical company websites.

PSORIASIS SUBTYPES

Scalp Psoriasis

Psoriasis of the scalp is common, with approximately half of patients with cutaneous psoriasis exhibiting some scalp involvement.[47] The scalp should be examined in all patients with psoriasis. While scalp psoriasis responds to the same topical and systemic treatments as psoriasis on other parts of the body, patients often find topical treatments on the scalp undesirable because of cosmetic concerns. Lotions and gels are usually preferred to thicker creams and ointments. Excimer laser therapy may be used if the hair can be parted sufficiently. Keratolytic and tar-based shampoos may also be used with optimal effect from longer contact time (10-20 minutes is recommended).[13,48]

Nail and Cuticle Psoriasis

Nail involvement is seen in approximately a quarter of patients with psoriasis.[47] Nails exhibiting psoriatic changes may respond

to systemic therapies used to control psoriasis on other body sites. To specifically target cuticle psoriasis, providers should consider high-potency topical corticosteroids alone or in combination with calcipotriol around the nail fold. In selected cases, intralesional steroid injections can supplement treatment, but they may be very painful.[48]

Intertriginous/Inverse Psoriasis

Intertriginous psoriasis can occur at the point of contact of any two skin folds; the inguinal fold, axilla, and external genitalia are the most common sites. This site is characterized by well-demarcated shiny red plaques with minimal to no scale. Because of maceration of the skin, superinfection may occur. Any erythematous eruption in the folds can be called intertrigo, with a differential diagnosis that includes inflammatory conditions (psoriasis, lichen planus), fungal infection (dermatophytes, yeast), bacterial infections including erythrasma, and irritant and allergic contact dermatitis.[49,50] Intertriginous psoriasis can be distinguished from these entities based on a history of psoriasis combined with a physical examination that reveals the characteristic uniform pink-to-red involvement. Other criteria for differentiation include:

- microscopic potassium hydroxide examination and fungal cultures to rule out yeast or dermatophyte fungus;
- woods light examination (black light with coral fluorescence characteristic of erythrasma);
- bacterial swab examined microscopically with culture to identify organisms responsible for cellulitis/impetigo; and
- skin biopsy if necessary to rule out lichen planus, contact dermatitis, and others.[13,50]

Psoriatic intertrigo is often treated with low-potency corticosteroids (often combined with an antiyeast agent to prevent secondary colonization) continuously for 2 to 4 weeks and then once to twice weekly for maintenance therapy. Alternately, topical calcineurin inhibitors (pimecrolimus cream, tacrolimus ointment) can be used as a steroid-sparing agent or when the skin is thin or fragile. There is little evidence for the use of conventional systemic therapies or biologics.

Erythrodermic Psoriasis

Erythrodermic psoriasis is life-threatening and affects 75% or greater BSA with accompanying erythema, scaling, and occasionally pustules. It may be associated with systemic symptoms including fever, chills, fatigue, malaise, peripheral edema, heart failure, renal failure, electrolyte imbalances, difficulties with thermoregulation, and superinfection. Erythroderma can be induced by both the introduction and abrupt withdrawal of medications, overuse of topical corticosteroids, stress, and alcohol use, among other causes.[51] The differential diagnosis includes severe atopic dermatitis, drug eruptions, cutaneous lymphoma, and an uncommon psoriasis-like keratinizing disorder (pityriasis rubra pilaris) with a characteristic yellow hyperkeratosis of the palms and soles.[51]

Management involves the evaluation and treatment of systemic abnormalities and superinfection, as well as systemic medications. Hospitalization may be necessary. First-line therapies include methotrexate, acitretin, cyclosporine, and infliximab because of their rapid onset of action. Systemic corticosteroids should be generally avoided because they can be a trigger for erythrodermic psoriasis (but may be the only choice in pregnancy or breastfeeding mothers), and phototherapy is contraindicated as these patients are often photosensitive.[51] Mild- to moderate-potency topical corticosteroids are often safely used in combination with systemic treatments.

Pustular Psoriasis

Pustular psoriasis presents as erythematous skin covered with 2- to 3-mm pustules and has generalized (acute, subacute) and localized forms (palmoplantar pustulosis and acrodermatitis continua of Hallopeau). Generalized pustular psoriasis may be accompanied by systemic symptoms including fever, malaise, arthralgias, peripheral edema, conjunctivitis, iritis, uveitis, oral pustulosis, and cheilitis, among others. Both generalized and localized pustular psoriasis may be induced by initiation and withdrawal of systemic and topical medications, infection, the H1N1 vaccine, sunburn, stress, and hormone changes, among others.[52]

The differential diagnosis of generalized pustular psoriasis includes pustular drug eruption, dermatitis with secondary infection, immunoglobulin A pemphigus, and pustular tinea corporis. A definitive diagnosis can be made based on history, physical examination, and, if the diagnosis is still unclear, punch biopsy of an intact pustule. First-line treatments for generalized pustular psoriasis include acitretin, cyclosporine, and methotrexate, with infliximab reserved for severe cases. Second-line treatments include adalimumab, etanercept, and PUVA. Topical adjunctive treatments including corticosteroids, calcipotriene, tacrolimus, and pimecrolimus may be used in conjunction with systemic treatments.[52]

Localized pustular psoriasis has a debilitating impact on quality of life and is often resistant to treatment. For localized pustular psoriasis without psoriatic arthritis, the recommended first-line therapy is potent or ultrapotent corticosteroids with occlusion and, if applicable, smoking cessation.[53]

Guttate Psoriasis

Guttate psoriasis is characterized by an acute eruption of drop-shaped red scaling papules and plaques. It may occur 2 to 3 weeks after a streptococcal infection in children and young adults. The differential diagnosis includes pityriasis rosacea, tinea corporis, secondary syphilis, pityriasis lichenoides chronica, nummular

dermatitis, and drug eruptions. These entities can be differentiated from guttate psoriasis based on history and physical examination with skin biopsy when necessary.[54]

To the authors' knowledge, the last review of treatments specific to guttate psoriasis was published in 2001. Investigations of treatments specific to guttate psoriasis are limited. There is no evidence to suggest any efficacy for antibiotics. Poor-quality evidence suggests that tonsillectomy may be efficacious in some patients. The most commonly used treatments include phototherapy, topical corticosteroids, vitamin D analogs, and tar. The natural history of guttate psoriasis is varied; it may clear on its own, turn into chronic plaque psoriasis, or remit and reappear.[54]

ASSESSMENT AND MANAGEMENT OF PAIN IN PSORIASIS

Psoriasis is often associated with burning and stinging pain, especially with increased inflammation and the formation of fissures within plaques.[55] Pain control can be achieved through improved control of psoriasis with systemic and topical medications as well as analgesics. The World Health Organization's Pain Ladder is a frequently used tool for choosing analgesics for the nociceptive pain component (gnawing, aching, tender, throbbing). The Pain Ladder suggests starting with acetaminophen and nonsteroidal anti-inflammatory drugs and then, if necessary, adding mild opioids (codeine, tramadol, or hydromorphone).[55,56] For the neuropathic component (burning, stinging, shooting, stabbing), oral agents including pregabalin, gabapentin, nortriptyline, and carbamazepine can provide symptom control. Providers must address the psychological component of the pain, and any psychiatric comorbidities should be diagnosed and treated.[55]

PSORIASIS AND WOUNDS

Wound care specialists may be called to treat peristomal psoriasis. A recent study reported a prevalence of peristomal psoriasis of 5% in a general stoma clinic.[57] While the majority of these patients (70%) had widespread cutaneous psoriasis, 3% had psoriasis only in the peristomal region, and 27% only had subtle signs of psoriatic disease outside the peristomal area.[57] Recent recommendations from a team of dermatologists who manage peristomal psoriasis have been reported.[57] Simply covering the peristomal psoriasis with a thick hydrocolloid dressing may be effective in some cases. Occlusion and topical corticosteroid lotion, gels, and foam vehicles are also used; providers should avoid greasier creams and ointments because they prevent the adhesion of the ostomy appliance. Topical products should be left to dry before the stoma appliance is reattached. In resistant cases, intralesional steroid injections using triamcinolone 2.5 to 8 mg/mL using 1 to 3 mL can often control the disease. Minimizing peristomal trauma is imperative during application of topical treatments. To avoid Koebner phenomena, flat appliances are preferred to convex alternatives. Conventional systemic treatments, phototherapy (with the stoma protected), and biologics are also used.[57]

A topic of some debate is whether to recommend continuation or interruption of biologic therapy for patients with psoriasis who are undergoing surgery. The 2017 British Association of Dermatologists guideline recommends stopping biologic therapy for either three to five half-lives of the drug or the length of one treatment cycle, whichever is longer, prior to surgery.[46] However, two recent studies of patients with psoriasis managed by biologic therapy documented that 66% to 74% continued treatment through the perioperative period, with a greater number of patients suspending therapy for more extensive cardiothoracic and orthopedic surgery. Both studies reported no increased risk of wound complications or infections with continued biologic therapy. However, these results were based on 77 and 131 surgical procedures, respectively, and a larger patient population needs to be studied before making definitive alternate recommendations.[58,59]

In general, psoriasis does not appear to have a negative effect on wound healing.[60,61] However, the Koebner phenomenon may result in patients developing psoriatic lesions over surgery sites[62] and tattoos,[63] among other injuries. This phenomenon is more common in patients with unstable, undertreated disease. Both the epidermis and the dermis must be involved for clinical disease to occur.[62] Lesions typically arise 10 to 20 days after trauma but have been known to arise anywhere from 3 days to 2 years postinjury.[18,62]

CONCLUSIONS

In conclusion, psoriasis can be differentiated into plaque, inverse, erythrodermic, pustular, and guttate forms. In addition to cutaneous manifestations, nail, scalp, joint, and systemic abnormalities may be present. Therefore, patients presenting with a suspected diagnosis of psoriasis should undergo a thorough history and physical examination including the scalp, nails, and joints.

Optimal management depends on the form of psoriasis, severity, location, and patient preference. Psoriasis affecting less than 10% of the patient's BSA and without debilitating effect on the patient may be treated with topical therapies. A BSA involvement of more than 10%, debilitating impact on life, or suboptimal response with topical therapies should prompt consideration of phototherapy or conventional systemic therapy. Biologic therapy is usually reserved for patients with 10% or more of the body involved, a PASI score greater than 10, and a Dermatology Quality of life Index score greater than 10. These patients have usually failed or have a contraindication to two or more conventional systemic therapies and/or photochemotherapy.

Currently, psoriasis treatment comprises an effective toolkit of therapeutic options. There are even more new agents on the

horizon, each with the potential to give patients with psoriasis improved quality of life and control of key aspects of psoriasis.

PRACTICE PEARLS

- Psoriasis can manifest in plaque, inverse, guttate, pustular, or erythrodermic forms.
- Fully examine the skin, nails, scalp, and joints to document the extent of the disease.
- Patients with less than 10% BSA affected are usually treated with topical treatments: corticosteroids and vitamin D analogs are first-line therapies.
- For patients with moderate to severe disease (≥10 BSA or debilitating disease), consider phototherapy and conventional systemic treatments.
- Biologic therapies are usually reserved for patients who have failed or have contraindications to two or more conventional systemic therapies and with residual psoriasis impairing their quality of life.

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