

Clinical and Histologic Diagnostic Guidelines for Psoriasis: A Critical Review

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Abstract Psoriasis is a common inflammatory skin disease that is associated with joint, psychiatric, and cardiovascular comorbidities. Diagnosis of plaque psoriasis is dependent primarily on characteristic physical findings and history. Given the varied clinical presentations of psoriasis and its mimicry to other papulosquamous skin diseases, it may be difficult for nondermatologists to diagnose psoriasis. Currently, no diagnostic criteria for plaque psoriasis have been validated in clinical studies. In this paper, we provide diagnostic guidelines for the nondermatologist to aid them in recognizing psoriasis.

Keywords Psoriasis · Diagnosis · Histopathology · Diagnostic criteria

Introduction

Psoriasis is a chronic, inflammatory skin disease that affects approximately 2–3% of the population in the USA and over 125 million people worldwide [1–3]. Psoriasis is associated with significant morbidity [4] and decreased quality of life [5, 6]. The associated comorbidities include psoriatic arthritis, cardiovascular and metabolic risks, and psychiatric disorders [7–9].

The clinical phenotype of psoriasis may manifest in several forms, which include plaque, guttate, pustular, and erythrodermic psoriasis. The plaque variant of psoriasis is the most common presentation, affecting 80–90% of psoriasis patients [10]. Clinicians have traditionally made the

diagnosis of plaque psoriasis based on pattern recognition from a thorough physical examination. To confirm the diagnosis of plaque psoriasis, clinicians have also relied on history and, if necessary, dermatopathology.

While some patients present with classic manifestations of psoriasis, diagnosis can be challenging in selected patients without complete, characteristic findings. Studies have demonstrated that nondermatologists experience difficulties diagnosing psoriasis [11, 12]. Although diagnostic criteria have been proposed for psoriatic arthritis, no validated diagnostic criteria exist for skin-limited psoriasis to date [13]. Few studies have investigated the sensitivity and specificity of signs and symptoms of plaque psoriasis. The aim of this article is to provide nondermatologists with key clinical and histological characteristics to help them diagnose plaque psoriasis. The diagnostic guidelines for plaque psoriasis provided in this paper are based on the epidemiologic data of characteristic signs and symptoms (Table 1).

Epidemiology

Prevalence

The prevalence of psoriasis in the USA is reportedly 2.6–3.15% [1–3]. One study estimated that 0.4–2.24% of the US population remains undiagnosed [2]. Worldwide, the prevalence rates range from 0.6% to 4.8% [14]. The highest psoriasis prevalence was observed in the Arctic Kasach'ye population (11.8%) [15], whereas the lowest rates of psoriasis were seen in certain African populations (<0.1%) [12, 16] and Samoan populations (0%) [17].

Caucasians are more frequently affected than any other racial or ethnic groups [1]. For example, the prevalence of psoriasis in African Americans is 52% lower compared to Caucasians [18]. Lower rates of psoriasis in African

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Table 1 Diagnostic guidelines for plaque psoriasis

Lesion Morphology. Characteristic morphology of erythema, scaling, and induration • Presence of Koebner Phenomenon (lesions develop following trauma) • Presence of Auspitz Phenomenon (pinpoint bleeding with removal of scale)	
Scalp Involvement Characteristic psoriasis lesions on the scalp • Scale usually thick and silvery. • Scale should not be yellow and greasy.	
Nail Involvement • Pitting • Onycholysis • Hyperkeratosis • Oil spots (yellowish-brown discoloration)	
Intertriginous Involvement Involvement of the body folds • Groin • Axilla • Intergluteal fold • Umbilicus • Inframammary folds • Genitalia	
History: • Gradual onset of skin lesions over the course of weeks to months • Age of onset between 20–30 years or 50–60 years • Presence of psoriasis in first-degree relatives • Improvement with UV exposure or exacerbation with lack of UV exposure	

Americans may be related to their West African ancestry, where prevalence rates are also observed to be lower [16, 18]. Studies investigating Asian populations also report lower prevalence rates than observed in Caucasians (0.1%–0.3%) [19, 20].

Gender Distribution

Most studies have found that psoriasis is equally distributed between males and females [17, 21], while women may experience earlier onset of disease [22]. No differences exist in quality of life decrements between male and female patients with psoriasis [6, 23, 24].

Age of Onset

A bimodal age distribution exists for psoriasis presentation, where they typically present between 20 and 30 years and between 50 and 60 years of age. Approximately 75% of patients with psoriasis will present it prior to the age of 40 [25]. Early onset of disease is associated with female gender [1, 22] and affected first-degree relatives [26]. Disease onset at a younger age may predict more extensive disease with greater numbers of exacerbations in life [7]. In comparison, patients presenting with psoriasis at an older age usually have a milder and more stable course and lack affected relatives [7].

Genetic Epidemiology

Studies suggest that psoriasis is a polygenetic disease that is associated with known environmental triggers [26]. Several identified alleles (*HLA-Cw6*, *HLADQ* 0201*, *CCHCR1*, and *CYP1A1*) and loci (*PSORS1-9*, *PSORSASI*) confer genetic predisposition for psoriasis [27]. Overall, the risk of psoriasis is 41% if both parents are affected, 14% if one parent is affected, 6% if a sibling is affected, and 2% when no first-degree relative is affected [28]. Currently, *HLA-cw6* and *PSOR1* are the most strongly associated with increased risk for psoriasis [29–31].

Clinical Presentation

Clinical History

Affected individuals with plaque psoriasis often present with chronic, recurrent erythematous, scaly patches and plaques. Skin lesions tend to persist for months to years, and intermittent flares are common. Spontaneous remission occurs in up to 25% of patients [32] and may last for months [33].

Modifying Factors

A variety of environmental factors have been implicated in triggering plaque psoriasis. The onset of psoriasis is associated with therapy using several drugs (lithium, interferon, β -blockers and antimalarials), infections (i.e., streptococcal, HIV), withdrawal of corticosteroids, and stressful life events [1, 2]. While streptococcal infection is most closely associated with precipitating guttate psoriasis, chronic plaque psoriasis may be exacerbated by streptococcal infection [34]. Climatic changes may affect the clinical course resulting in improvement during the summer months and exacerbation during the winter [1]. Changes in disease severity based on seasonal variations have been attributed to the differential ultraviolet light exposure, which is known to ameliorate psoriasis. Scant data suggest that improvement of psoriasis can be seen during pregnancy [16].

Increasing epidemiological data suggest smoking and alcohol are associated with psoriasis. Mechanisms underlying the interaction between smoking and psoriasis include induction of oxidative damage, stimulation of proinflammatory cytokines, and enhancement of susceptibility gene expression [35]. Intensity of cigarette smoking may correlate with psoriasis severity; smoking more than a pack per day confers twice the risk of more severe involvement [36]. Alcohol intake may trigger psoriasis by impairing skin barrier function and altering immune function and keratinocyte activity [37, 38]. Consumption of

non-light beer may increase the risk for psoriasis among women [39].

Symptoms

Common symptoms associated with psoriasis include pruritus, irritation, burning, sensitivity, pain, and bleeding [40]. The discomfort caused by the skin lesions may result in impaired sleep, concentration, and overall reduced quality of life [41]. Pruritus is the most commonly reported symptom in psoriasis [3]. Studies report that as high as 80% of psoriasis patients experience pruritus [42]. Two thirds of patients with psoriasis experience itching often or constantly [40]. Psoriasis severity does not necessarily correlate with pruritus [43].

The prevalence and severity of pruritus associated with psoriasis appear to be different from those of other skin diseases. Compared to psoriasis patients, those with atopic dermatitis experience pruritus more frequently [44]. Pruritus is also more severe in patients with atopic dermatitis and lichen planus. Whereas significant improvement in itching can result from successful treatment of lichen planus, treatments that decrease affected psoriasis areas may not necessarily result in proportional relief of itching [45].

Physical Examination

Plaque psoriasis appears as sharply margined, erythematous patch or plaques with a characteristic silvery-white micaceous scale (Fig. 1) [46]. Affected persons may exhibit varying degrees of erythema, scaling, plaque thickness, and body surface involvement [47]. Lesions may initially manifest as erythematous, scaly papules that coalesce to form plaques. Affected areas may appear bright red to white, depending on the amount of scales. Approximately 80% of patients have a mild form of psoriasis (3% body surface area



Fig. 1 Large erythematous, well-demarcated plaque with coarse silvery-white scale on the abdomen

[BSA]), whereas the remaining 20% patients will have moderate (3–10% BSA) or severe forms (>10% BSA).

New psoriasis lesions can form at sites of trauma, which is termed Koebner phenomenon. Removal of scale often reveals pinpoint bleeding, which is called Auspitz phenomenon. With treatment, there is a decrease in erythema, scaling, and induration; areas can evolve to become hypo- or hyperpigmented patches without scarring.

Psoriatic lesions are distributed symmetrically (Fig. 2) and can affect any part of the body. The most commonly affected sites are the scalp, torso, and extensor surfaces of the extremities such as the knees and elbows (Fig. 3). Facial involvement is rare, and when present, is often associated with significant decrement in quality of life [48]. Although not always present, involvement of the intergluteal cleft can be a useful diagnostic clue.

Although plaque psoriasis typically involves the extensor surfaces, it can affect intertriginous folds in 3–7% of psoriasis patients [18], which has been termed “inverse psoriasis.” In inverse psoriasis, the groin, axillae, inframammary folds, and genitalia are commonly involved [19]. Clinical examination of inverse psoriasis reveals thin, well-defined erythematous plaques with little to no scale due to the often occluded locations (Table 1).

Nail Psoriasis

Nail disease occurs in 15% to 53% of psoriasis patients and is thought to correlate with more severe cutaneous and joint disease [8]. Up to 10% of psoriatic patients present with only nail manifestations with no other cutaneous or joint findings [2, 20]. Psoriatic nail disease occurs more commonly in the fingernails than the toenails [2] and manifests as pitting, onycholysis, oil spots, hyperkeratosis, or nail grooving (Table 1). Nail pitting occurs as a result of psoriasis affecting the nail matrix. Characteristic yellowish-brown



Fig. 2 Symmetrical distribution of discrete erythematous plaques on the back



Fig. 3 Bright pink plaques with minimal scale on the extensor surface of the left forearm

nail discoloration or “oil spots” on the nail of psoriatic patients result from psoriasis involvement of the nail bed. Onycholysis occurs at the distal nail plate at the point of separation from the hyponychium.

Scalp Psoriasis

Scalp psoriasis is the most common manifestation of plaque psoriasis and may be present in up to 79% of patients with psoriasis [49]. Similarly to other lesions of psoriasis, lesions on the scalp display erythema, induration, and scaling (Table 1). Scalp psoriasis is frequently pruritic and results in shedding of scale. The presence of scalp psoriasis is 66% sensitive (61–69) and 92% specific (87–95) for the diagnosis of psoriasis [11]; however, distinguishing scalp psoriasis from seborrheic dermatitis can be difficult in some patients. Scalp psoriasis is associated with significant impact on the quality of life [50].

Diagnostic Guidelines

The diagnosis of plaque psoriasis ought to be based on thorough clinical history and physician examination. Clinicians should inquire regarding the onset of lesions, possible triggering factors, associated symptoms (i.e., itch, pain, sensitivity, irritation), and family history. Associations of psoriasis with smoking and alcohol should prompt clinicians to inquire regarding social factors.

As reviewed previously, full cutaneous examination should include the nails, scalp, and intertriginous areas. We provide the following diagnostic guidelines for the nondermatologists: (1) characteristic morphology of erythema, scaling, and induration; (2) scalp involvement; (3) nail involvement (pitting, onycholysis, crumbling, or oil spots); (4) involvement of the intertriginous folds; and (5) family history of psoriasis.

Dominguez et al. have developed and proposed a psoriasis screening tool that incorporates skin and nail findings to produce good sensitivity and specificity [11]. The authors investigated the sensitivity and specificity of this screening tool to validate self-reports of psoriasis in the USA [11]. While isolated scalp, nail, or intertriginous psoriasis findings yielded fair sensitivity (66%, 51%, and 59%, respectively) and high specificity (92%, 93%, and 90%) [11], greater sensitivity (81%) and specificity (96%) can be achieved if scalp or nail disease is present in addition to intertriginous involvement [11].

Histopathology

Skin biopsy is not routinely required for diagnosis of psoriasis. Histological findings in psoriasis vary depending on the age of the lesions [2]. Early lesions display edema and dilated capillaries in the papillary dermis, perivascular lymphocytic infiltrate, early Munro abscesses (neutrophilic infiltrates in parakeratotic foci), epidermal spongiosis, and loss of the granular layer [27].

As the lesions progress, elongation and widening of the deep portion of the rete ridges in the epidermis occur. The rete ridges may become fused with one another (Fig. 4). Other characteristic findings are parakeratosis with neutrophils, suprapapillary thinning, increased number of mitotic figures, and superficial pallor of the epidermis [2]. Pathognomonic findings include microabscess of Munro (collection of neutrophils surrounded by parakeratosis in the stratum corneum) and the spongiform pustule of Kojog (spongiotic pustule with neutrophilic infiltration). In late lesions, there are reductions in leukocytes, neutrophils, suprabasal mitotic figures, and papillary edema [28]. The rete ridges may appear club shaped or squared at the bases (Fig. 5).



Fig. 4 Intermediate histological changes at medium power (courtesy of Keira L. Barr MD, University of California Davis Dermatology)

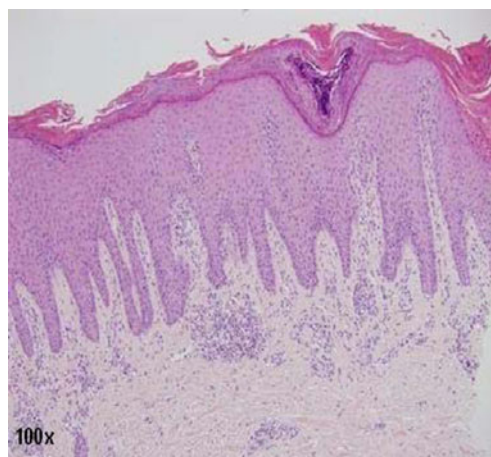


Fig. 5 Late histological changes at medium power (courtesy of Thomas Konia MD, University of California Davis Dermatology)

Differential Diagnoses

The differential diagnoses for plaque psoriasis includes nummular eczema, lichen simplex chronicus, atopic dermatitis, lichen planus, discoid lupus, and parapsoriasis. Distinguishing eczema from psoriasis can be challenging in some patients; some eczema lesions have different scale presentation and relative lack of sharp margination. Studies also suggest that the pruritus is experienced differently between the two conditions [45]. While psoriatic lesions devoid of scales may appear similarly to lichen simplex chronicus, the development of characteristic scale in psoriasis and presence in classic locations can aid in psoriasis diagnosis. Compared to psoriasis, lichen planus tend to have greater involvement of the mucous membranes. Unlike most cases of psoriasis, discoid lupus can be exacerbated by sun exposure and result in scarring. In addition, parapsoriasis is typically not associated with pruritus and lacks nail and joint involvement. In difficult cases, biopsy may be necessary to differentiate psoriasis from similar appearing conditions.

Additional differential diagnoses may be considered for location-classified plaque psoriasis. Inverse psoriasis may appear similarly to intertrigo, Hailey–Hailey disease, seborrheic dermatitis, and eczema. Scalp psoriasis may be misdiagnosed as tinea capitis, eczema, and seborrheic dermatitis. Differential diagnoses for psoriatic nail disease include fungal infections of the nail, lichen planus, and pityriasis rubra pilaris. The presence of classic psoriatic lesions in other cutaneous locations may be helpful in distinguishing psoriasis from similar appearing skin conditions.

Conclusion

Psoriasis is a common, immune-mediated disease with associated arthritis, depression, and cardiovascular comor-

bidities [29–31]. While no established diagnostic criteria exist for skin-limited psoriasis, trained clinicians can often diagnose psoriasis based on clinical history and skin examination. In challenging cases, histopathology can be helpful in distinguishing psoriasis from other types of inflammatory skin diseases. In the current literature, there is a dearth of studies examining the sensitivity and specificity of clinical signs and symptoms for psoriasis. In this paper, we have provided diagnostic guidelines to aid the nondermatologist in recognizing and diagnosing psoriasis. Future studies are necessary to investigate and validate screening tools for diagnosing psoriasis.

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