Clinical Images

Presentations of Cutaneous Disease in Various Skin Pigmentations: Plaque Psoriasis

Ashleigh E. Hermann, DO1,2; Daniel A. Nguyen, DO, PharmD1,2; Christopher M. Wong, DO1,2; Christian J. Scheufele, DO1,2; Michael Carletti, DO1,2; Stephen E. Weis, DO1,2

Abstract

Description
Recent studies estimate that more than 8 million Americans have psoriasis. The prevalence of psoriasis in African Americans is 1.5% compared to 3.6% of Caucasians. Psoriasis is likely to be underdiagnosed among African Americans and other individuals with darker pigmented skin due to variations in clinical presentation in addition to disease distribution and severity. We present images of psoriasis vulgaris in a variety of Fitzpatrick skin types. Differences in the biology of skin pigmentation may explain the clinical masking of erythema in darker-skinned individuals. Recognition of this important difference will help clinicians utilize additional clues to identify and diagnose this entity correctly.

Keywords
dermatology; psoriasis; psoriasis/diagnosis; psoriasis/ethnology; plaque psoriasis; skin pigmentation; skin of color; papulosquamous skin diseases; Fitzpatrick skin types

Introduction
Psoriasis is likely to be underdiagnosed among African Americans and other individuals with skin of color due to variations in clinical presentation.1 Additionally, disease distribution and severity may contribute to decreased rates of diagnosis.1 Differences in skin pigmentation can be described using the Fitzpatrick scale (Figure 1). Psoriasis is commonly associated with comorbidities such as chronic kidney disease, inflammatory bowel disease, hepatic disease, certain malignancies, and/or mood disorders.2,3 It is also a risk factor for cardiometabolic disease.2,3 The ability to accurately diagnose psoriasis is critical to appropriately screen these patients for the above comorbidities and provide treatment. Additional background information on the Fitzpatrick scale and a description of the classification of skin types are discussed in further detail in the article "Presentations of

Figure 1. The Fitzpatrick scale provides a classification system for an individual’s skin type based on the ability to burn and/or tan when exposed to ultraviolet light. It is used to approximate the degree of skin pigmentation.
Cutaneous Disease in Various Skin Pigmentations: An Introduction.

Clinical Images

Plaque psoriasis typically presents as sharply demarcated, erythematous, scaly plaques. Commonly affected areas include the scalp, trunk, gluteal fold, and extensor surfaces such as the elbows and knees. Psoriasis can have many different scale types, including micaceous, thick, and thin.

Figure 2 exemplifies the bright red erythematous nature of the plaques in a patient with Fitzpatrick skin type II (mostly burns, rarely tans). Also depicted are the varying degrees

Figure 3. Fitzpatrick type III (sometimes burns, often tans) skin demonstrating plaque psoriasis involving the trunk and upper and lower extremities. The lesions have bright red erythema. This is in contrast to the erythema observed in Fitzpatrick type V (almost never burns, always tans) skin. The photo on the left depicts a large bright red plaque (raised, >1 cm) on the right lateral thigh. There is an overlying micaceous scale. The photo in the middle depicts two large plaques (raised, >1 cm) on the lateral arm. As mentioned above, there is a micaceous scale, and the lesions are well demarcated (distinctly different from the adjacent normal skin where the border is readily identified). The photo on the right depicts a large plaque (raised, >1 cm) on the back with multiple satellite papules (raised, <1 cm) on the periphery.
of thickness of scale. On the left are multiple plaques with thick white scales, and on the right side, micaceous scales are depicted. The term micaceous refers to the mica stone with a silvery-white lamellated scale.

**Figure 3** depicts a classic example of the appearance of micaceous, thick, and thin scale in Fitzpatrick skin type III (sometimes burns, often tans). The left picture depicts a large bright red plaque with an overlying thick white scale on the right lateral thigh. The image in the middle is an example of a micaceous scale on the left forearm of the same patient. The image on the right depicts a large plaque on the back with a thin overlying scale.

**Figure 4** exemplifies the salmon-colored, sharply demarcated plaques with an overlying silvery scale in patients with Fitzpatrick skin type IV (rarely burns, mostly tans). Well-demarcated refers to the distinct boundaries of the lesion that are clearly defined.

**Figure 5** depicts multiple large violaceous plaques and papules with well-demarcated borders and white scale on the trunk and buttocks of a patient with Fitzpatrick skin type V (almost never burns, always tans). The erythema in darker skin tones is often less conspicuous and may appear violaceous, violet in color, or hyperpigmented, appearing brown to dark brown. When assessing disease burden in patients, it is essential to consider that psoriatic plaques in patients with skin of color often resolve with either hypo- or hyperpigmented patches. The depigmentation may take anywhere from 6 to 12 months or more to resolve.

**Figure 6** provides a classic example of the appearance of a micaceous scale in a Fitzpatrick skin type V (almost never burns, always tans). There are large, violaceous papules and plaques on the thighs, extensor surface of the knees, and lower legs with thick, white micaceous scale overlying the plaques on the bilateral knees.
Figure 5. Fitzpatrick type V (almost never burns, always tans) skin demonstrating plaque psoriasis involving the trunk, buttocks, and upper extremities. The erythema is dark red to violaceous in color. The photo on the left depicts the generalized distribution of the psoriatic plaques. The photo in the middle is a close-up visualizing the violaceous erythema seen surrounding each papule (raised, <1 cm) and plaque (raised, >1 cm). There is an overlying white, silvery scale. The photo on the right shows the papules (raised, <1 cm) coalescing, or forming, into plaques (raised, >1 cm). The lesions are also involving his umbilicus, a common location for psoriasis.

Figure 7 shows classic examples of psoriasis affecting the scalp in Fitzpatrick skin types II (mostly burns, rarely tans), III (sometimes burns, often tans), and V (almost never burns, always tans). Of note, the picture on the far right depicts an example of festooning. The origin of the term “festooning” refers to “a garland or wreath hanging in a curve, which is used in decoration.” This can be visualized in the photo as the plaque suspended from the scalp, extending onto the forehead.

Survey results from national psoriasis experts found that African American patients experienced a higher degree of dyspigmentation (41% of responders) but a lower amount of erythema (24% of responders). African Americans
have also been reported to have more extensive body surface area (BSA) involvement. A survey found that African Americans reported 3 to 10% BSA involvement compared to only 1 to 2% BSA reported by Caucasian patients.

Discussion
The most current studies estimate that more than 8 million Americans have psoriasis. Psoriasis has a bimodal age distribution from 18 to 39 and 50 to 69, with similar occurrences in men and women. Psoriasis may present at an earlier age due to genetic factors. For example, the human leukocyte antigen (HLA)-C*06 allele is associated with an earlier age of onset of psoriasis.

The prevalence of psoriasis in African Americans is 1.5% compared to 3.6% of Caucasians. The numbers may be misleading as a national study analyzing NHANES data from 2003 to 2004 showed racial/ethnic disparities in access to a dermatologist have been reported in the United States, and undiagnosed psoriasis was more prevalent among African Americans.

The pathogenesis of psoriasis is complex; it is continually undergoing further research. Overactivation of certain parts of the adaptive immune system is the main component of its pathogenesis. Overactivation of the adaptive immune system leads to many downstream effects of psoriasis, including keratinocyte proliferation and infiltration of immune cells into lesional skin.

Psoriasis is a chronic skin disease associated with multiple systemic comorbidities. These include coronary atherosclerotic plaques, increased risk of myocardial infarction, depression, anxiety, and/or suicidal ideation. Additionally, approximately one-third of patients with psoriasis will develop psoriatic arthritis at some point during their lifetime.

The differential diagnosis of psoriasis includes atopic dermatitis, seborrheic dermatitis, lichen planus, and cutaneous T-cell lymphoma. Atopic dermatitis can also affect a large body surface area. However, pruritis is typically more persistent, and these lesions lack the well-demarcated morphology of psoriatic plaques. Seborrheic dermatitis is also ill-defined and occurs in a seborrheic distribution (scalp, eyebrows, nasolabial folds, upper back and chest, axillae, etc.). Lichen planus and cutaneous T-cell lymphoma also lack the micaceous scale observed in psoriasis. Lichen planus presents with purple, polygonal, pruritic, flat-topped papules and plaques with overlying grayish-white streaks called Wickham's striae. Cutaneous T-cell lymphoma consists of patches and thin plaques with fine scales favoring sun-protected areas.

Informed Consent
Written informed consent was obtained from the patients for their anonymized information to be published in this article.
Conflicts of Interest
The authors declare they have no conflicts of interest.

Drs Carletti, Hermann, Nguyen, Weis, and Wong are employees of Medical City Fort Worth, a hospital affiliated with the journal’s publisher.

This research was supported (in whole or in part) by HCA Healthcare and/or an HCA Healthcare affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA Healthcare or any of its affiliated entities.

Author Affiliations
1. University of North Texas Health Science Center, Fort Worth, TX
2. Medical City Fort Worth, Fort Worth, TX

References
1. Alexis AF, Blackcloud P. Psoriasis in skin of color: epidemiology, genetics, clinical presentation, and treatment nuances. J Clin Aesthet Dermatol. 2014;7(11):16-24.
2. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis: a review. JAMA. 2020;323(19):1945-1960. doi:10.1001/jama.2020.4006
3. Scheufele CJ, Weis D, Weis SE. Presentations of cutaneous disease in various skin pigmentation: an introduction. HCA Healthcare Journal of Medicine. 2022;3(3):135-138. doi:10.36518/2689-0216.1483
4. Takeshita J, Grewal S, Langan SM, et al. Psoriasis and comorbid diseases: implications for management. J Am Acad Dermatol. 2017;76(3):393-403. doi: 10.1016/j.jaad.2016.07.065
5. McMichael AJ, Vachiramon V, Guzmán-Sánchez DA, Camacho F. Psoriasis in African-Americans: a caregivers’ survey. J Drugs Dermatol. 2012;11(4):478-482.
6. Gelfand JM, Stern RS, Nijsten T, et al. The prevalence of psoriasis in African Americans: results from a population-based study. J Am Acad Dermatol. 2005;52:23-26.
7. Parisi R, Symmons DP, Griffiths CE, Ashcroft DM: Identification and Management of Psoriasis and Associated Comorbidities (IMPACT) project team. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. J Invest Dermatol. 2013;133(2):377-385. doi:10.1038/jid.2012.339
8. Nair RP, Stuart PE, Nistor I, et al. Sequence and haplotype analysis supports HLA-C as the psoriasis susceptibility 1 gene. Am J Hum Genet. 2006;78(5):827-851. doi:10.1086/503821
9. Armstrong AW, Mehta MD, Schupp CW, Gondo GC, Bell SJ, Griffiths CEM. Psoriasis prevalence in adults in the United States. JAMA Dermatol. 2021;157(8):940-946. doi:10.1001/jamadermatol.2021.2007
10. Kurd SK, Gelfand JM. The prevalence of previously diagnosed and undiagnosed psoriasis in US adults: results from NHANES 2003-2004 [published correction appears in J Am Acad Dermatol. 2009 Sep;61(3):507]. J Am Acad Dermatol. 2009;60(2):218-224.
11. Nestle FO, Kaplan DH, Barker J. Mechanisms of disease: psoriasis. N Engl J Med. 2009;361(5):496-509. doi:10.1109/APCAS.2002.1114931
12. Mease P, Goffe BS. Diagnosis and treatment of psoriatic arthritis. J Am Acad Dermatol. 2005;52(1):1-19. doi: 10.1016/j.jaad.2004.06.013