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Dermatology: how to manage psoriasis and recognize differences in pathophysiology and presentation in patients with skin of colour

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Abstract

Psoriasis is a chronic inflammatory skin condition that affects diverse ethnic groups with a wide spectrum of skin colours. There are significant differences in how psoriasis presents and impacts the quality of life in non-White individuals. Genetic variations as well as cultural and socioeconomic factors all play a role in such differences and have important implications for the management of psoriasis in skin of colour. Despite these differences, the current psoriasis management is similar across different ethnic backgrounds and is mainly guided by factors such as disease severity, medical comorbidities and patient preferences. This is largely due to the lack of sufficient evidence for psoriasis treatment tailored for patients with skin of colour as most clinical trials are composed of mainly White individuals. Therefore, the focus of this article is to review the current evidence on how epidemiology, clinical presentation and genetic differences in patients with skin of colour with psoriasis may impact treatment strategies. Additionally, pharmacological therapies available to date in these diverse patient cohorts are summarized in this article. The limited data published on this topic reveal a significant need for more investigations with the ultimate goal of incorporating recommendations for patients with skin of colour into the current guidelines for psoriasis treatment. Moreover, awareness of differences in psoriasis presentation amongst individuals with skin of colour may support patients to seek medical care sooner, which could result in earlier diagnosis and lead to improved patient outcomes.

Keywords: psoriasis, therapeutics, skin of colour

Introduction

Psoriasis is a chronic autoimmune inflammatory skin condition affecting patients from diverse ethnic groups with a wide spectrum of skin colours. Whilst there are some similarities in psoriasis between ethnic groups, there are significant differences in epidemiology and clinical presentation that may impact management. Current guidelines on psoriasis treatment have no specific recommendations for patients with skin of colour,[1–5] which reflects the scarce evidence available in the literature to date. Focused studies are needed in this patient cohort to elucidate evidence-based and patient-centred strategies when caring for a diverse patient population. The goal of this article is to provide a thorough review of current literature and summarize unique characteristics of psoriasis presentation and management in patients with skin of colour, whilst highlighting the sparse knowledge in this area of research.

Methods

This study is a review of psoriasis in skin of colour with a primary focus on treatment strategies. On June 13, 2021, OVID PubMed, Medline and Google Scholar were used to conduct a search using the following key words: “psoriasis”, “management”, “treatment” and “skin of colour”. Only studies involving human patients published in the English language or with official English translations were included. In addition, the references of identified articles were reviewed for pertinent studies.

Review

Epidemiology

Psoriasis impacts 2–3% of the population worldwide, with the most common type being plaque psoriasis in 85–90% of cases.[6–10] The overall prevalence of psoriasis, however, varies between 0.05% and 3.7% in different ethnic groups.[6,11–17] Most data to date suggest that psoriasis is more prevalent in individuals with White skin compared to non-White skin; however, statistics in the latter may be underestimated due to the lack of data in this area as well as underreporting and underrepresentation of individuals with skin of colour in studies.[6,11–18] Additionally, National Health and Nutrition Examination Surveys in 2003–2006 and 2009–2010 reported that the highest proportion of patients with psoriasis were White (3.7%), followed by Black (2.0%) and Hispanic (1.6%).[14,15] Paediatric studies report a similar proportional pattern with the prevalence of psoriasis in White children being 0.29% and in Black children 0.06%.[19]

It is also important to note that the prevalence of psoriasis may vary by region, even between individuals with the same skin colour. For example, whilst 1.9–3.5% of individuals from Eastern Africa (Kenya, Uganda and Tanzania) have psoriasis, in Western Africa (Ghana, Nigeria, Senegal and Mali) the prevalence is 0.025–0.9%.[14] Such differences in prevalence may be attributed to genetic variations and other unknown factors. Interestingly, it was found that African Americans are more closely related to West Africans, which may help explain the comparably lower prevalence of psoriasis in individuals with Black skin in the data reported from the United States.[16]

Comparisons in psoriasis prevalence between Asian countries are also limited. One large retrospective Malaysian study reported that Malay adults had a high rate of psoriasis (8.6%), followed by Chinese (6.0%). One major limitation of this study was that the patients included were selected from a hospital

clinic; as such, the cohort may not be reflective of the actual distribution of psoriasis in the population within the region.[20] Paediatric studies report similar findings with Malay and Indian children being 3–4 times more likely to be diagnosed with psoriasis compared to Chinese children.[13,21] Studies in South America suggest a prevalence of 1.3–4.2%, in Trinidad and Tobago the prevalence is 5.1% and in Cuba it is 6%.[22,23] Individuals of East Asian ethnicity were found to have a greater prevalence of psoriasis than those with African ancestry.[22] However, ethnic minorities may have barriers to healthcare and are more likely to have undiagnosed psoriasis.[18] Overall, psoriasis prevalence studies in patients with skin of colour are sparse. Large population-based studies are needed to improve our understanding of psoriasis prevalence in ethnicities across the globe.

Pathophysiology

Psoriasis is associated with interactions of keratinocyte-immune cells that are driven by T helper 1 (T_H1) and T_H17 cells.[24] The inflammatory cascade begins with skin antigens activating neutrophils and dendritic cells, which in turn release cytokines such as TNF, IL-23 and IL-17. These cytokines activate leucocytes and result in a perpetuated positive feedback loop.

Pathophysiology is a complex interaction between environmental and genetic factors. Known environmental psoriasis triggers include bacterial infections, stress, smoking, obesity, cold weather, HIV and, perhaps, diet.[16,25–29] Interestingly, in the African American diet, the presence of linoleic acid may be protective.[25] Given that obesity is a risk factor for psoriasis, it is thought that the low prevalence of obesity in African American and Asian women, as well as differences in metabolic factors, may reflect the relatively lower prevalence of psoriasis in these groups.[30–33] A number of studies have also demonstrated differences in genetic susceptibility to psoriasis amongst ethnic groups that have been reviewed elsewhere.[23]

Clinical presentation

Classically, psoriasis presents with hallmark features of well-demarcated erythematous plaques with silvery scales. Plaques are typically located in the scalp, extensor surfaces and buttocks but can be present anywhere on the body. These plaques may vary in size, thickness, shape, degree of scaling and distribution amongst different ethnic groups. For example, erythema on Black skin manifests as violaceous or hyperpigmented compared to the red or pink lesions typically seen in lighter skin tones.[18,34] Therefore, in individuals with darker skin tone, the active psoriatic lesions are more challenging to diagnose and could be mistaken for post-inflammatory hyperpigmentation.[18] Additionally, patients with Black skin usually have thicker plaques with more scaling compared to individuals with White skin.[34–36] Furthermore, resolution of skin lesions in darker skin tones may lead to lasting dyspigmentation that is very bothersome for patients and may take years to fade (Figure 1).[18,30,36]

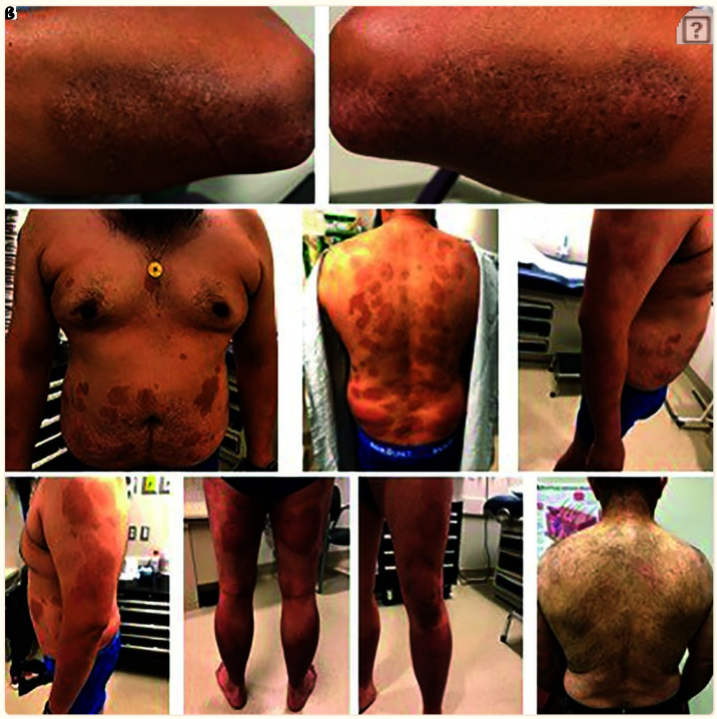

Figure 1

Post-inflammatory hyperpigmentation on the right (A) and left (B) legs of a patient with dark skin more than 2 years after achieving PASI 100. Examples of post-inflammatory hyperpigmentation on the torso (C), back (D), right flank (E), left flank (F), thighs on the back (G) and left thigh in the front (H). Patient with darker skin with PUVA-associated lentigo (I).

In addition to the lasting pigmentary effects after lesion resolution, individuals with Black skin have also been reported to have a larger surface area of skin involvement.[12,34–36] Whilst African Americans are documented to have an average of 3–10% body surface area involvement, White individuals have 1–2%.[12] Of the 525 individuals with psoriasis, only 36.3% with White skin tone present with severe psoriasis, whilst 54.2% of Asian and 51.7% of Hispanic patients have severe psoriasis.[37,38] These data suggest that Asian and Hispanic individuals are more likely to have a more severe psoriasis presentation.[37,38]

Psoriatic lesions on the scalp may also have a more severe presentation in African American women likely due to less frequent hair washing.[18,39,40] For example, women of African ancestry typically wash their hair once per week.[18,39,40] Some additional reports suggest that Asian individuals may have a slightly greater prevalence of scalp psoriasis compared to Western European but these data are limited.[41] On the contrary, inverse psoriasis is thought to be more common amongst White individuals compared to African or Asian.[30,42] Psoriatic arthritis is also reported to be twice as common in White compared to African individuals and also twice as common in Indian compared to Chinese ethnic groups.[43,44]

Psychosocial impact

As a result of differences in the clinical presentation of psoriasis amongst different ethnic groups, the quality of life was documented to be significantly

worse in non-White individuals compared to White.[12,18,43,45] Notably, the post-treatment dyspigmentation is not incorporated into the psoriasis severity scores, and the impact of treatments on such dyspigmentation has not been studied in individuals with skin of colour.[46] Therefore, the quality of life may be impacted more in individuals with darker skin tone even if the severity is not different from individuals with White skin tone.[18,30,36] Stress has also been shown to be a more likely trigger for psoriasis in Hispanic patients compared to White individuals.[47] Moreover, cultural variations in perception of appearance or differences in expression of emotions may further impact the quality-of-life scores.[18,48] Finally, the quality of life in patients with psoriasis may be significantly influenced by the differences in socioeconomic, educational and employment status in ethnic minorities.[43,48] Overall, the perception of patient's quality of life, depression and anxiety in patients of colour may impact their outlook on the condition.[49,50] It is therefore important to anticipate the impact that psoriasis has on patient quality of life and counsel the patient in a holistic way.

Treatment

Generally, treatment of psoriasis is similar across people of different ethnic backgrounds, and the choice of optimal therapy is mainly guided by disease severity, response to previous therapies, medical comorbidities, concomitant medications, impact on the quality of life and patient preferences.[51–53] First-line treatment for mild psoriasis is usually topical agents and, if insufficient, phototherapy and oral agents are considered.[51] In patients presenting with moderate-to-severe psoriasis, treatment entails phototherapy or systemic therapy, including biologic agents or a combination of these modalities.

The current treatment guidelines for psoriasis are based on randomized clinical trials (RCTs) composed mainly of White individuals. On average, about 84.3% (range 67.0–95.4%) of participants in psoriasis phase III pivotal trials were White.[23,54] As discussed earlier, there are important differences in pathophysiology, clinical presentation and severity of psoriasis in individuals with different ethnicities. Therefore, it is important to include a more racially diverse population in psoriasis trials and conduct subgroup analyses to widen the applicability of the trial outcomes to people with different skin colours. The following is a review of the evidence of psoriasis treatments available to date in individuals with skin of colour.

Topical therapies First-line topical therapies for the treatment of psoriasis include corticosteroids, vitamin D analogues, retinoids, and calcineurin inhibitors for the face and flexure areas.[51] A recently reported phase III study showed that 8-week treatment with a fixed-combination halobetasol propionate (0.01%) and tazarotene (0.045%) lotion reduced the severity of psoriasis across a range of skin colours in non-White, White and Hispanic/Latino participants.[55] Whilst topical corticosteroids are highly efficacious, they may induce hypopigmentation in individuals of colour.[56] Such a side effect may take years to fade and could have a significant impact on the patient's quality of life and satisfaction. It is important to counsel patients about this potential side effect and discuss alternative options.

Topical therapies are often used to treat scalp symptoms of psoriasis. Given that the hair texture and hair care patterns differ between individuals with different skin colours, it is important to individualize topical treatments for the scalp. Studies of calcipotriol and betamethasone have shown a significant improvement in redness, itching, thickness and scaling in a large proportion of individuals with non-White skin tone of African American, Latino and Asian descent.[57,58] After initial treatment, maintenance therapy with this topical agent led to better control of psoriasis symptoms in the Asian cohort.[59] The vehicle of topical scalp therapies should be compatible with the patient's hair texture, frequency of hair washing, and cultural hair styles in order to improve compliance to treatment. For African American individuals, usually oil-based vehicles, emollient foams and lotions are preferred.[18]

Phototherapy First-line phototherapy regimens for the treatment of psoriasis include narrowband ultraviolet (UV) B and psoralen with UVA (PUVA). Phototherapy has been shown to be effective in individuals with darker skin colours, albeit greater doses may be needed.[60,61] In individuals with skin types IV–V of Indian descent, narrowband UVB and PUVA were equally effective with the same number of treatment doses to achieve clearance and maintain remission of severe plaque psoriasis.[62,63] About 75% of Asian patients showed a good response to narrowband UVB.[61] The downside of phototherapy treatment in individuals with skin of colour is the temporary darkening of the skin, which may not be acceptable for some patients and may be impacted by cultural factors.[64–66] Whilst the effects of phototherapy on pre-existing psoriasis pigment changes have not been studied, exposure to ultraviolet radiation may exacerbate psoriasis-associated changes in pigmentation, especially in individuals with skin of colour. Suberythemogenic dosing with 70% of minimal erythema dose of narrowband UVB showed similar efficacy as full minimal erythema dose in both patients with fair and dark skin.[67] Whilst skin colour lightens after cessation of phototherapy, it is important to explore patient acceptability of the temporary tanning effects to ensure treatment compliance and satisfaction.

Oral therapies Current oral systemic therapies include methotrexate, acitretin, cyclosporine and apremilast. Patients with refractory psoriasis could also be treated with different combinations of such agents.

Methotrexate is a commonly prescribed medication for moderate-to-severe psoriasis. Ethnic differences in response to this therapy are not well studied, and limited data describing polymorphisms associated with methotrexate response are often unknown by dermatologists.[68–71] For example, it was shown that ABCC1 and ABCG2 are genes for efflux transporters that are known to be associated with a good methotrexate response.[68–71] Interestingly, these genes have different frequencies in a variety of populations and, hence, it may have an impact on methotrexate efficacy in some populations.[68–71] Polymorphisms in human leucocyte antigen (HLA)-Cw*06, *IL12*, LCE3C_LCE3B-del and *FOXP3* were shown to predict a superior methotrexate response in a South Indian Tamil cohort.[72] Finally, some polymorphisms in patients with rheumatoid arthritis (*MTHFR*, *FPGS*, *ABCC2* and *RFCl*) were shown to be associated with differences in response to methotrexate between Asian and European populations.[70,73] Such gene polymorphisms may contribute towards a greater methotrexate discontinuation rate due to attenuated efficacy or cutaneous side effects in the South Asian cohort.[74]

Data on ethnic differences in the treatment response to cyclosporine and acitretin are sparce. Polymorphisms in vascular endothelial growth factors, which differ in frequency between White and non-White individuals, were shown to predict the response to acitretin therapy.[75,76] Although some studies suggest the involvement of other gene polymorphisms, their frequency in specific ethnicities is not studied.[69]

Apremilast and tofacitinib have mostly been studied in patients with White skin colour in phase II RCTs with White study participants comprising 82–90% of the entire study cohort.[77–80] Efficacy of apremilast in a Japanese cohort of a phase IIb RCT was shown to be numerically similar to the phase III global trials.[77,81] On the contrary, the efficacy of tofacitinib in a Chinese, Taiwanese, Korean and Japanese mixed cohort exhibited a better efficacy response compared to phase III global trials, whilst the safety outcomes did not differ.[82,83] Some studies report that the risk of herpes zoster infection while taking tofacitinib is three times greater in Asian individuals compared to North American, European and Latin American individuals.[82,84,85] However, such conclusions should be interpreted with caution given the lack of direct comparisons and subgroup analyses of responses by different ethnic groups within a trial. It should also be noted that tofacitinib is not approved for the treatment of psoriasis.

Biologic therapies The list of biologics indicated for psoriasis treatment continues to expand. Currently, approved biologics in many countries include anti-TNF (adalimumab, certolizumab, etanercept, infliximab), anti-IL-12/IL-23 (ustekinumab), anti-IL-23 (guselkumab, risankizumab, tildrakizumab), anti-IL-17 (secukinumab, ixekizumab) and anti-IL-17R (brodalumab). With the rapidly increasing use of biologic therapies, it is important to consider potential differences in the efficacy of these medications between different ethnic groups to facilitate the choice of optimal therapy.

Whilst some inhibitors of TNF have shown differences in efficacy in some ethnic groups, others appeared to have similar efficacy. In the phase III REVEAL trial, 12 weeks of adalimumab treatment was shown to be more efficacious in White participants (Psoriasis Area and Severity Index score with 75% or greater improvement from baseline (PASI 75) of 72%) compared to non-White participants (PASI 75 of 63%).[86] In a Japanese cohort, adalimumab treatment for 16 weeks resulted in a PASI 75 of 59.5–62.8%.[87,88] In contrast, in a study of Chinese patients treated with adalimumab of the same dosing

regimen for 12 weeks, greater efficacy was achieved (PASI 75 of 77.8%).89 A post hoc analysis of the EASE study revealed that treatment with etanercept 50 mg twice a week found similar efficacy and safety across ethnic groups. However, 86% of the study cohort were White and only 7% were Hispanic, 4% Black and 3% Asian.45 In a group of Korean patients, half of this dose was shown to have similar efficacy as the phase III EASE trial.90–92

To date, the IL-12/IL-23 inhibitor has not shown evidence of efficacy that varies in different ethnic groups. Ustekinumab treatment with 45 mg was reported to be effective in Korean, Taiwanese, Japanese and Chinese individuals; with the greatest efficacy in Chinese patients.93–95 Treatment with an inhibitor of IL-23 (guselkumab) for 24 weeks was superior to adalimumab in White and Asian study participants but not in individuals with Black skin colour.96

Inhibitors of IL-17A (secukinumab and ixekizumab) have demonstrated efficacy in Japanese cohorts with generalized pustular psoriasis.97–99 On the other hand, treatment with inhibitors of the IL-17 receptor (ixekizumab and brodalumab for 12–52 weeks) were reported to result in a PASI 75 of between 95% and 100%, which was superior to the overall study population of different ethnic backgrounds.99–105 A more recent pooled analysis of AMAGINE-2/−3 RCTs composed of patients with psoriasis who received brodalumab reported similar skin clearance rates across the different ethnic subgroups studied (Black, Asian, White, Hispanic/Latino) at weeks 12 and 52.106 Hispanic patients were shown to have efficacy greater than that of non-Hispanic patients after 12 weeks of secukinumab treatment in pooled data from phase III RCTs (PASI 90 achieved by 70.6% *versus* 58.0%, respectively).107 Finally, a subgroup analysis of Taiwanese and Japanese patients from the ERASURE trial reported that treatment with secukinumab resulted in efficacy similar to that observed in the entire study population.102,108,109

The mechanisms responsible for differences in the efficacy of biologics in individuals of different ethnic backgrounds are unknown. It is possible that differences in average body weight between populations could be a contributing factor. For example, Asian patients in clinical trials were found to be 15–20 kg lighter on average compared to White individuals, and thus receiving the same dose would have a greater dose per body weight and could result in greater efficacy.110 Similar to oral therapies, efficacy in response to biologic agents may be impacted by genetic polymorphism differences amongst ethnic groups. For example, polymorphisms in *TNF*, *IL12B*/*IL23R* and *TNFRSF1B* were shown to have improved efficacy of respective biologics in a European patient group.111–113 Genome-wide association studies in Japanese and Chinese patients with psoriasis reported 80 SNPs that were associated with the response to TNF inhibitors.114 Human leukocyte antigen polymorphisms may also play a role in the response of Chinese patients to etanercept and ustekinumab.115 Whilst there are reports of genetic polymorphisms that affect response to biologic therapy, the prevalence of such polymorphisms is not studied in different ethnic groups.111

Conclusion

Evidence shows that individuals with skin of colour have unique clinical presentations of psoriasis, susceptibility and response to treatment. Psoriasis often presents with more severe lesions and has a greater impact on the quality of life in patients with skin of colour. Moreover, genetic diversity may have an impact on drug metabolism, toxicity and response to treatment. When choosing optimal therapy in individuals with skin of colour, such differences in genetic diversity should be considered in addition to disease severity, impact on the quality of life, response to previous therapies and presence of other comorbidities. Unfortunately, data are sparse in these diverse populations, as over 80% of study participants in phase III RCTs have White skin tone. It is therefore important to emphasize the inclusion of individuals with diverse ethnic backgrounds into psoriasis clinical trials to enable subgroup analyses. Furthermore, the impact of treatments on the unique pigmentary changes observed with psoriasis in individuals with skin of colour needs to be studied. Awareness of differences in psoriasis presentation amongst individuals with skin of colour may support patients to seek medical care sooner, resulting in earlier diagnosis and, ultimately, in better patient outcomes. Evidence-based treatment guidelines that apply across a range of skin colours are needed to help dermatologists serve an increasingly diverse patient population.

Key practice points

- Current psoriasis treatment guidelines are based on randomized clinical trials composed of mainly White individuals; thus, these guidelines do not contain specific recommendations for patients with skin of colour.
- Erythema in darker skin tones manifests as violaceous or hyperpigmented compared to the red or pink lesions typically seen in lighter skin tones, making it harder to diagnose. Patients with darker skin usually have thicker plaques that cover larger surface areas with more scaling compared to individuals with White skin. Such lesions often resolve with lasting dyspigmentation that is very bothersome for patients and may take years to fade.
- The quality of life was documented to be significantly worse in non-White individuals compared to White.
- The current choice of optimal therapy is mainly guided by disease severity, response to previous therapies, medical comorbidities, concomitant medications, impact on the quality of life and patient preferences.
- First-line topical therapies for the treatment of psoriasis include corticosteroids, vitamin D analogues, calcineurin inhibitors and retinoids. Whilst topical corticosteroids are highly efficacious, they may induce hypopigmentation in individuals of colour.
- Phototherapy has been shown to be effective in individuals with darker skin colours. Whilst the effects of phototherapy on pre-existing psoriasis pigment changes have not been studied, exposure to ultraviolet radiation may exacerbate psoriasis-associated changes in pigmentation.
- Current oral systemic therapies include methotrexate, acitretin, cyclosporine and apremilast. Ethnic differences in response to this therapy are not well studied, and limited data exist describing the polymorphisms associated with the methotrexate response.
- Currently approved biologics in many countries include anti-TNF (adalimumab, certolizumab, etanercept, infliximab), anti-IL-12/IL-23 (ustekinumab), anti-IL-23 (guselkumab, risankizumab, tildrakizumab), anti-IL-17 (secukinumab, ixekizumab) and anti-IL-17R (brodalumab). The mechanisms responsible for differences in the efficacy of biologics in individuals of different ethnic backgrounds are unknown.
- It is important to include a more racially diverse population in psoriasis trials and conduct subgroup analyses to widen the applicability of the trial outcomes to people with different skin colours.

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