

Seborrheic Dermatitis in Skin of Color: Clinical Considerations

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ABSTRACT

Seborrheic dermatitis is a common, relapsing, inflammatory skin condition of unclear etiology. The *Malassezia* yeast genus are believed to play a role. Seborrheic dermatitis commonly affects areas of the skin with high sebum production, including the scalp, nasolabial folds, glabella, eyebrows, beard, ears, retroauricular skin, sternum, and other skin folds. Seborrheic dermatitis may present differently in individuals with skin of color. Darker-skinned individuals may present with scaly, hypopigmented macules and patches in typical areas of involvement. Arcuate or petal-like patches may be seen, specifically termed petaloid seborrheic dermatitis. Children of color often do not experience the classic "cradle cap" appearance of seborrheic dermatitis, and have erythema, flaking, and hypopigmentation of the affected areas and folds of skin. Seborrheic dermatitis tends to respond well to conventional treatments, although it tends to recur. Skin of color patients may require a modified treatment approach which takes into account differences in hair texture and hair washing frequency. This paper aims to highlight these differences to help reduce disparities in the management of seborrheic dermatitis in patients of color.

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INTRODUCTION

Scalp and hair disorders are among the most common concerns in patients of color, particularly African-Americans.[1] Seborrheic dermatitis (SD) is a chronic, inflammatory condition that affects areas of high sebum production and is a common reason for dermatologic consultations in skin of color patients.[1] A paucity of literature exists regarding the presentation and treatment of SD in this population. As more African-Americans seek dermatologic care, it becomes crucial for dermatologists to be trained in recognizing and addressing the concerns of diverse patient populations. The goal of this paper is to address the differences in the presentation and treatment of SD in skin of color patients. Highlighting these differences can allow for an effective approach and ultimately reduce the current disparities in the management of skin of color patients with SD.

Epidemiology and Causes

Seborrheic dermatitis (SD) is a common, chronic, benign inflammatory skin disorder of unclear pathophysiology affecting 3% to 12% of the population.[2,3] SD may have slightly increased incidence among African Americans (6.5%)[4] and West Africans (2.9-6%).[5]

In a study that compared the most common diagnoses for patients of various ethnic groups in a hospital-based dermatology practice, SD was among the five most common diagnoses observed in black patients.[1] SD is prominent among black women and can be exacerbated by excessive use of hair oil and pomade, and infrequent shampooing.[6] Some of the recognized risk factors for SD include immunodeficiency (HIV), neurological (Parkinson's disease) or cardiac disease, as well as alcoholic pancreatitis.

Several factors have been proposed to play a role in the development of SD including the proliferation of the commensal yeast genus, *Malassezia*, the host immune response, and the composition of sebaceous gland secretions. The population density of *Malassezia furfur* has been shown to be highest in anatomic sites most highly populated with sebaceous glands,[7] overlapping with areas where SD tends to occur. However, it is unclear if this has a direct impact on the pathogenesis of SD. In 1989, Bergbrant and Faergemann[8] found no difference in the number of yeast cells in patients with SD and healthy controls, but several other studies since that time have suggested a positive correlation between yeast density and severity of SD.[9,10] A study conducted by Nakabayashi et al[9] compared lesional and non-lesional skin in patients with SD and healthy controls. The authors found that *M. furfur* and *M. globosa* were isolated at much higher rates from lesional skin on the face in SD patients than in healthy controls, which suggests that yeasts may have a pathogenic role in SD. Zaidi et al[10] conducted a study that compared *Malassezia* yeast density to SD disease severity and found that when comparing SD patients, those with more severe clinical disease had a greater density of *Malassezia* yeast

cells per high power field. In addition, the high therapeutic efficacy of antifungal agents in the treatment of SD seems to support Malessezia yeast as a contributing factor.[11]

The host immune response has been proposed to play a role in the pathogenesis of SD. SD is more common in immunosuppressed patients than immunocompetent patients with rates up to 83% in the former versus 1-3% in the latter.[11] The presentation of HIV-associated SD is distinct, and has unique clinical and histopathological features. HIV-infected patients may present with erythroderma or may develop concurrent Kaposi varicelliform eruption, also known as eczema herpeticum, both of which may be recalcitrant to standard treatment and tend to recur much more frequently.[12] This clinical presentation of SD may be a marker of underlying HIV infection and should prompt the clinician to screen for HIV status. In a study comparing lesional skin in SD patients to non-lesional skin in healthy controls,[13] it was found that SD involves a strong inflammatory reaction with an increase in NK1+ and CD16+ cells, complement activation, and increased inflammatory interleukins. It has also been shown that T-cell function is depressed in patients with SD while natural killer cell, IgA or IgG antibody levels may be increased.[8] Together these findings highlight an immune or inflammatory reaction in patients with SD, possibly towards Malessezia yeast.

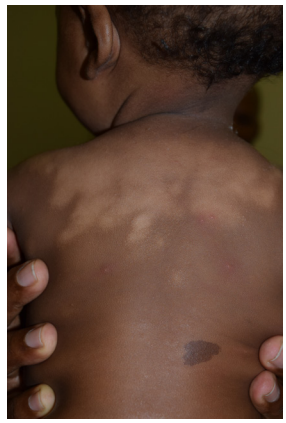
Clinical Manifestations and Diagnosis

SD is generally a clinical diagnosis based on the typical location and morphology of lesions. In adults, the classic presentation of SD consists of erythematous patches with greasy scales on areas of the body highly populated with sebaceous glands: scalp, glabella, eyebrows, nasolabial folds, paranasal skin, cheeks, bearded areas of the face, upper chest, back, and skin flexures.[11] HIV-associated SD may be quite extensive, spreading well beyond the commonly affected sites.[14] In children, SD typically presents as thick white or yellow greasy scale on the scalp, commonly called "cradle cap." SD may be asymptomatic or can be intensely itchy.

Darker-skinned adults may have additional presentations of SD including hypopigmentation in the classic areas of involvement (Figure 1) and underlying erythema may be difficult to appreciate.[15] This population may also present with arcuate and petaloid lesions that commonly involve the hairline but may affect other areas of the face.[16] This form of SD, called "petaloid seborrheic dermatitis," appears as polycyclic coalescing rings that are pink or hypopigmented with minimal scale.[17] The associated hypopigmentation typically improves with treatment[18] and is thought to result from inhibition of melanocyte tyrosinase function and pigment production by yeast metabolites.[18] In children of color SD generally does not have the standard "cradle cap" appearance that is seen in Caucasians. Children of color often present with erythema, flaking, and hypopigmentation of the affected areas and overlying atopic dermatitis,

FIGURE 1. Hypopigmented macules and patches on the face of an African female with SD *(image courtesy of Dr. Ncoza Dlova).*

FIGURE 2. Coalescing hypopigmented patches on the back of an infant with SD *(image courtesy of Dr. Ncoza Dlova).*



which accentuates hypopigmentation (Figure 2).[19] Meanwhile, adolescents of Hispanic, Asian, and African origin may present similarly to adults, with hypopigmented scaly plaques on the eyebrow and perinasal region.[20]

The differential diagnosis for SD is lengthy (Table 1) and includes psoriasis, atopic and contact dermatitis, and rosacea.[3] In children, tinea capitis, particularly the seborrheic type, is an important diagnostic consideration for scalp involvement as it is more common than SD in black and Hispanic children.[21] Langerhans cell histiocytosis is an additional consideration in children.[3] If uncertain, a biopsy can be obtained demonstrating parakeratosis in the epidermis, spongiosis, and plugged follicular ostia.[22]

TABLE 1.

Differential Diagnosis of Seborrheic Dermatitis	
Tinea capitis	Erythrasma
Psoriasis	Wiskott-Aldrich cutaneous lupus
Atopic dermatitis	Dermatomyositis
Contact dermatitis	Vitamin B deficiency
Rosacea	Zinc deficiency
Langerhans cell histiocytosis	Drug eruption

Adapted from "Seborrheic Dermatitis," by *Drs. Thomas Berk and Noah Scheinfeld, June 2010. (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2888552/)*

Prognosis

SD is a benign, though chronic, relapsing condition.[18] It usually responds well to conventional treatments, although it can recur. In adolescents with SD, the course is similar to adults, and relapse is common. Infantile SD is typically asymptomatic and resolves spontaneously in weeks to months.[23] Given the chronic and relapsing nature of SD, it may affect the quality of life of those affected. In a study of South Africans with SD, it was found that SD in visible body areas and with groin involvement had a greater impact on a patient's quality of life (QOL).[12] The goal of treatment of SD is to control and manage rather than cure.

Treatment and Clinical Considerations

There is limited literature on the treatment of SD in patients of color. While some components of treatment are similar to those used in other populations, special considerations should be considered given differences in hair type, hair washing frequency, and tendency for hypopigmentation. Traditionally, treatment includes the use of topical corticosteroids or antifungals such as ketoconazole, selenium sulfide, and zinc pyrithione-containing shampoos/creams/lotions. Immunomodulators, tar, and phototherapy may also be used. The goal of treatment is to inhibit yeast colonization, loosen crusts and scales, and reduce inflammation and pruritus.[3]

Care must be given to the treatment choice and vehicle type as some patients, particularly African-Americans, inherently have dry and brittle hair. In the treatment of SD of the scalp, this subset may find certain shampoos and solution-based topicals too drying or irritating. Often, patients may use over-the-counter dandruff shampoos which are exceptionally drying to the hair shaft, particularly in patients of color who may use heat or chemical relaxers,[24] thus resulting in fragility and hair breakage. In a study by Chappell et al[25] that compared SD treatment modalities, it was found that Caucasian patients preferred antifungal foams, gels, and sprays, while black patients preferred ointment or oil preparations. Emphasis on alternative, less drying treatment modalities may increase compliance and treatment success in skin of color patients, and prevent hair damage and breakage. In general, ketoconazole shampoo is utilized with caution in African-American women and care should be taken to instruct patients to apply directly to the scalp, rather than to the hair shaft.[6] In men and children of color, ketoconazole shampoo may be used with less concern for hair fragility given men and children are less likely to have chemically or heat-styled hair.[6]

It is also important to consider the difference in hair washing frequency in certain populations, as SD of the scalp may be difficult to treat in individuals who wash their hair less frequently. In the same study, 79.4% of African American women were found to report hair washing frequency as less than once a week.[25] The researchers evaluated ketoconazole 2% foam, which is applied to the scalp and left in, versus ketoconazole 2% shampoo, which requires washing, for treatment of SD in African-American females. The prospective, investigator initiated, parallel-group, open label, cross-over trial found that both shampoo and foam are effective at reducing SD disease severity, but foam users were more likely to be very satisfied with their results despite lower compliance. It is thought that these findings may be a result of better penetration of the foam due to its alcohol base and longer contact time. Thus, methods that require less frequent hair washing are likely to be more efficacious in this group.

Finally, hypopigmentation is a common occurrence in skin of color patients with SD. This tends to resolve with treatment. A study by High and Pandya[26] studied 1% Pimecrolimus in the treatment of SD for African American patients with associated hypopigmentation. In the study, five African American adults with SD applied a thin layer of pimecrolimus to the affected areas twice daily for 16 weeks. Measures of improvement examining erythema, pruritus, scaling, and hypopigmentation and were objectively measured using a mexameter. Improvement was seen not only in erythema and scaling, but also in associated hypopigmentation. Medications in this class, including tacrolimus, may be a good option for resistant cases of SD as well as to avoid the side effects of long-term topical steroid use such as skin atrophy, tachyphylaxis, and perioral dermatitis.

Prevention

Given the relapsing and remitting nature of SD, reducing the frequency of flare is key. Once SD is under control, it is important to emphasize that infrequent hair washing may lead to product build-up in the scalp which may further contribute to irritant dermatitis and seborrheic dermatitis.[27] Shampooing once weekly or once every two weeks is advised for women of African descent with tightly coiled hair.[6] A common practice in skin of color patients, particularly African-Americans, of applying pomades and oils to the scalp should also be avoided. Patients may use these products to mask dry flakes, or because they believe flaking represents a dry scalp, but these products often worsen SD by causing scalp irritation.[24] Instead, dermatologists can advise the use of hair emollients on the hair shaft as opposed to the scalp.[28]

Furthermore, hair styling practices and their effect on SD have been studied. A cross-sectional survey conducted by Rucker Wright et al[29] looked at various hair styling practices among African-American girls aged 1-15 years and the prevalence of scalp/hair disorders among this cohort. The study reported a significant association between SD and the use of added hair extensions and infrequent hair oil application (every two weeks). The authors propose hair extension use may lead to scalp irritation, which may cause scalp inflammation and SD.

They propose girls with SD may use hair oils less frequently due to physician advice that SD is exacerbated by hair oils/grease, and paradoxically, that infrequent hair oil use may lead to a dry scalp and SD. Interestingly, this study found no correlation between more frequent hair washing (more than once per week) and SD prevalence. Thus, for the prevention of SD, it appears as though natural hairstyles may prevent occurrence, although more studies are needed to fully access the effect of hair extensions on SD.

CONCLUSION

SD is a common condition that may present differently in children and adults of color. An understanding of these differences, as well as the various practical considerations that impact treatment choice can help dermatologists provide culturally competent care and may lead to better outcomes for patients. Given the chronic and relapsing nature of SD, more studies looking into preventative measures are warranted. In particular, studies investigating the role of various haircare practices in skin of color patients, such as hair washing frequency, hairstyle type, hair extension use, and haircare product type are recommended.

DISCLOSURES

Drs. Dlova, Ogunleye, and Taylor and May Elgash have no conflicts to disclose.

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