SKIN

BRIEF ARTICLE

Petaloid Seborrheic Dermatitis: A Variant Described Primarily in Skin of Color

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ABSTRACT

Petaloid seborrheic dermatitis is a distinct variant of seborrheic dermatitis most commonly affecting patients with skin of color. It is characterized by arcuate coalescing lesions with fine scale and a raised border that symmetrically expands over the course of weeks in a seborrheic distribution. Two patients with extensive petaloid seborrheic dermatitis of the face are presented who quickly responded to a typical regimen for seborrheic dermatitis. Recognition of petaloid seborrheic dermatitis will prevent misdiagnosis and improper treatment of this common condition.

INTRODUCTION

Seborrheic dermatitis (SD) is a chronic condition characterized by erythematous patches and plaques with scale affecting body regions of high sebum production.[1] SD has a distinct appearance described primarily in patients with skin of color (SOC) termed "petaloid seborrheic dermatitis." Pink, hypopigmented polycyclic/arcuate coalescing rings with minimal scale slowly expand symmetrically over weeks from the classic SD distribution in the naso-labial folds, eyebrows, and hairline.[1,2] Two cases of petaloid SD are presented and the pathophysiologic basis for these findings is explored.

CASE REPORTS

A 29-year-old female presented with a 10-month history of pruritus, hypopigmentation, and flaking of the scalp and face. Ketoconazole 2% shampoo and ketoconazole 2% cream to affected areas resulted in little improvement. Inspection revealed symmetrical, erythematous, hypopigmented, petaloid patches with scale and raised advancing borders present symmetrically across the nasolabial folds, cheeks, temples, conchal bowls, and eyebrows (**Figure 1A-1C**). Xerotic plaques with scale were also present across the midline upper back and central chest. Recognizing the symmetry and classic distribution of petaloid seborrheic dermatitis, a KOH preparation was not performed. Treatment was initiated with ketoconazole 2% shampoo daily, ciclopirox 0.77% cream twice daily and hydrocortisone 2.5% ointment daily for facial plaques, and fluocinonide 0.05% solution daily and as needed for scalp itching. The condition cleared completely

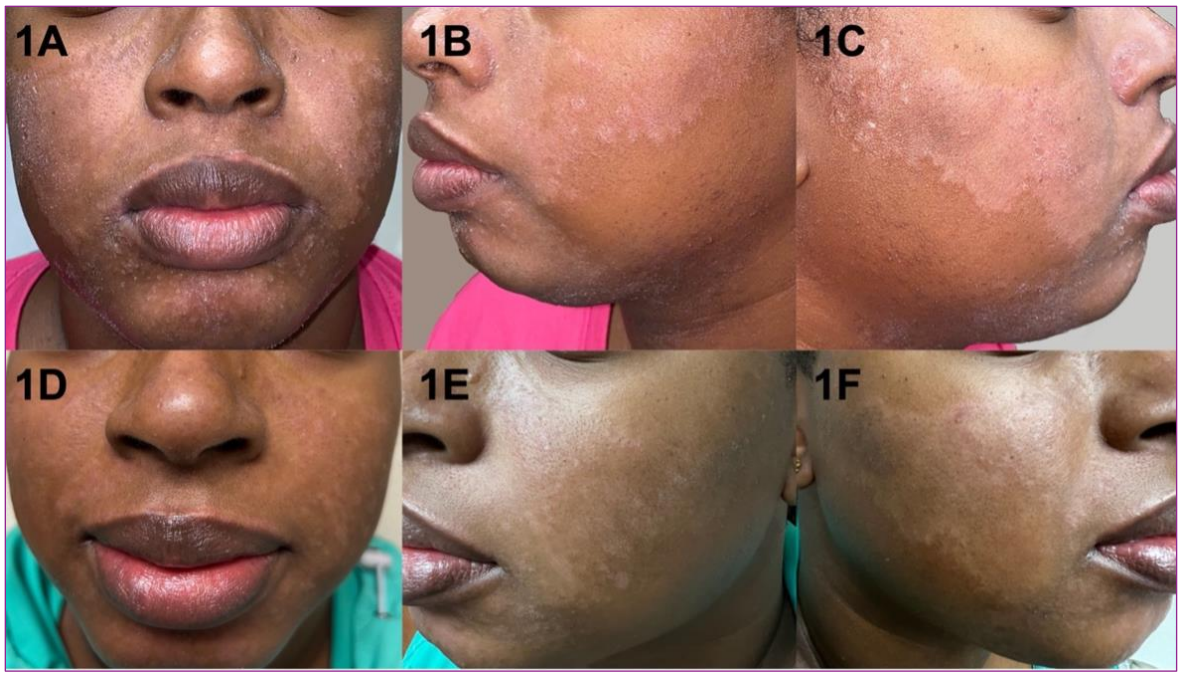

Figure 1. (A-C) Symmetrical, erythematous, hypopigmented, petaloid patches with scale and raised advancing borders present symmetrically across the nasolabial folds, cheeks, temples, conchal bowls, and eyebrows. **(D-F)** Clearance of plaques after ketoconazole 2% shampoo daily, ciclopirox 0.77% cream twice daily and hydrocortisone 2.5% ointment daily.

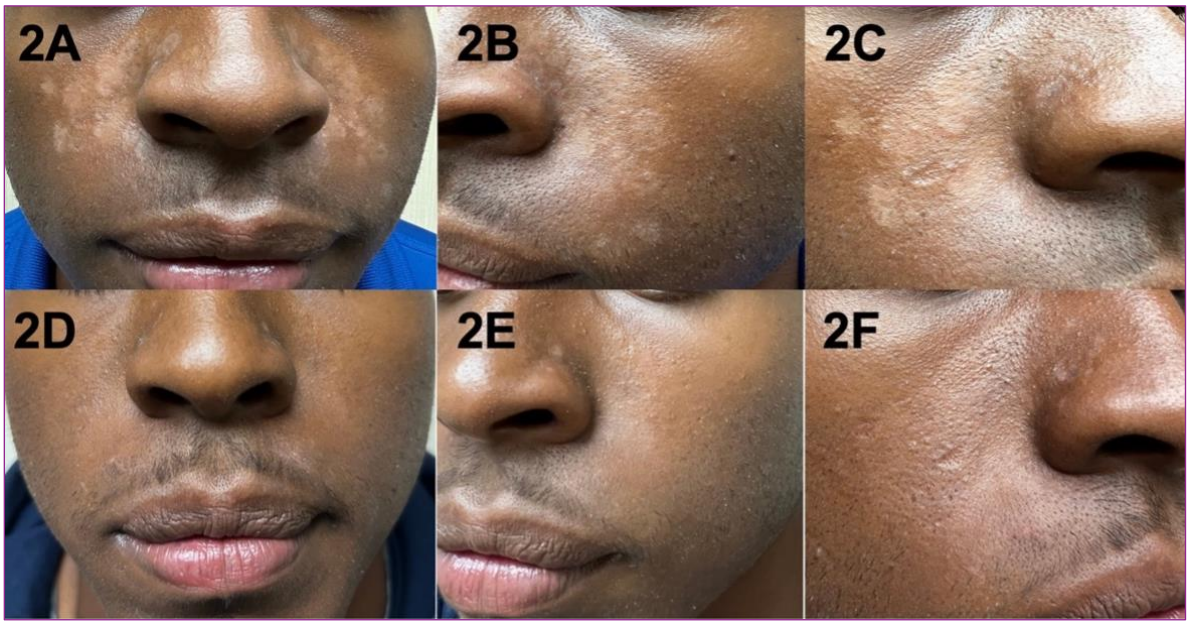

Figure 2. (A-C) Diffuse scale throughout the scalp and erythematous, greasy, petaloid patches with scale, some with a raised advancing border, symmetrically involving the nasolabial folds and eyebrows. **(D-F)** Complete clearance in one month with oral fluconazole 200 mg weekly, hydrocortisone 2.5% cream twice daily, ketoconazole 2% shampoo daily, and betamethasone dipropionate 0.05% ointment twice daily to the scalp and as needed for itching.

(**Figure 1D-1F**). The topical steroids were tapered, and clearance was maintained with topical antifungals alone.

An 18-year-old male presented with a hypopigmented, erythematous and flaky rash affecting the eyebrows, nose, and appearing diffusely in the scalp for five years. Despite treatment with clotrimazole 1% cream, ketoconazole 2% shampoo, and betamethasone dipropionate 0.05% ointment, the rash intermittently flared. Physical examination revealed diffuse scale throughout the scalp and erythematous, greasy, petaloid patches with scale, some with a raised advancing border, symmetrically involving the nasolabial folds and eyebrows (**Figure 2A-2C**). A KOH preparation was not performed because of the classic presentation of petaloid seborrheic dermatitis. Treatment was initiated with oral fluconazole 200 mg weekly, hydrocortisone 2.5% cream twice daily, ketoconazole 2% shampoo daily, and betamethasone dipropionate 0.05% ointment twice daily to the scalp and as needed for itching. The condition cleared completely within one month (**Figure 2D-2F**) and was well-controlled with ketoconazole 2% shampoo twice weekly applied to the face and scalp.

DISCUSSION

Petaloid SD is a distinct presentation of SD almost always affecting patients with SOC.[1,2] Given the presentation of annular lesions and a raised edge, petaloid SD may be mistaken for tinea faceii, which can be excluded with a potassium hydroxide preparation, if needed. The symmetrical, central facial distribution and diffuse, fine scale argued against this diagnosis in our patients. Syphilis may present similarly to petaloid SD with arcuate or annular plaques or patches on the face.[3] That our patients had no lesions except in the seborrheic distribution, specifically no palm and sole involvement, argues against this diagnosis. Psoriasis/sebopsoriasis could be considered, but our patients did not have the classic white micaceous scale on extensor surfaces or nail changes consistent with psoriasis. Atopic dermatitis commonly occurs on the face in infants but would be unlikely to produce the chronic annular symmetrical patches seen in our patients. Several scarring conditions could produce annular, dermal lesions including cutaneous sarcoidosis and discoid lupus erythematosus, but no scarring process was evident. Finally, cutaneous T-cell lymphoma (CTCL) could be considered, but lesions would not be restricted to the seborrheic distribution and the poikiloderma typical of CTCL was not present. Occasionally, a punch biopsy could exclude these dermal infiltrative or neoplastic conditions.

The etiology of SD is unclear and undoubtedly multifactorial. Development of symptoms is strongly linked to the presence of *Malassezia* species.[1] Overactivity of sebaceous glands may also play a role, particularly given that excess sebum promotes *Malassezia* growth.[1] Finally, an immunological response to the presence of *Malassezia* on the skin contributes to disease pathogenesis.

The underlying pathophysiology associated with petaloid SD in SOC may differ from classic SD in some ways. One potential mechanism may be related to an increased inflammatory response to *Malassezia* organisms in darker-skinned patients. SOC, for unknown reasons, is associated with an increased inflammatory response in several dermatological conditions, such as acne vulgaris.[4] Of particular interest is the presence of polymorphonuclear leukocyte cell infiltrates within comedones, lesions

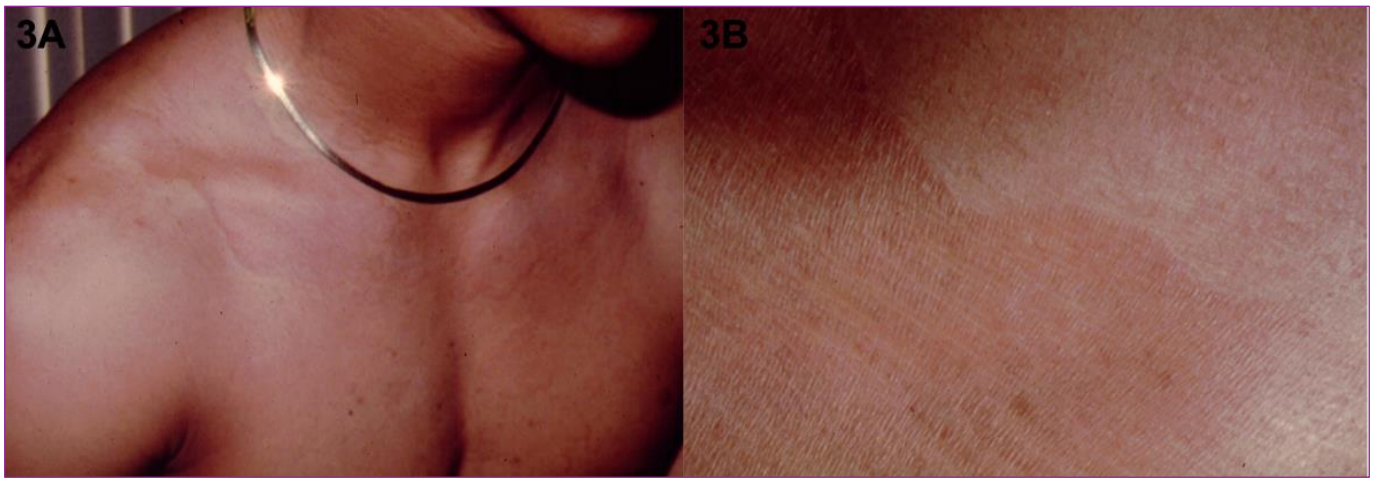

Figure 3. Petaloid appearance of tinea versicolor.

generally non-inflammatory in lighter-skinned patients.[4]

Inflammatory pityriasis rosea is also more common in SOC. It presents with a greater number of papular and plaque-like lesions of the scalp and face with post-inflammatory hyperpigmentation, more frequent involvement of oral mucosa, and more pruritus, when compared to light-skinned phenotypes.[5,6] While the Herald patch is often annular, multiple annular lesions occur in as many as 20% of patients.[7,8] It is unclear from the literature if these annular lesions occur more commonly in SOC. Tinea versicolor (TV), a condition which like SD is associated with *Malassezia*, may also produce greater inflammation in SOC.[6] When confluent, an exaggerated rim can have a petaloid appearance (**Figure 3**). Inverse TV is a variant commonly seen in SOC which occurs on flexural areas, extremities, and the face. The latter location is notably spared in the classic presentation.[9,6] In summary, pityriasis rosea, TV and SD can present with annular rings and a petaloid appearance that are not as commonly described in light-skinned patients.[1,8]

An increased inflammatory response in patients with SOC may be driven by various mechanisms. Genetic influences on sebum production may optimize skin for growth of resident flora.[9] Culturally promoted application of palm oil or cocoa butter may play a role as these substances are lipid-rich and may facilitate growth of *Malassezia* in TV by altering the carbon dioxide concentration, epidermal flora, and pH of the skin.[9,10]

CONCLUSION

Future research should explore histopathological features of inflammation at the advancing rim in patients with petaloid SD. It is also important to know more about the clinical response to anti-inflammatory topicals and more targeted anti-fungal agents designed to eliminate *Malassezia* yeast. Finally, an assessment of moisturizing products that may be preferentially utilized by patients with SOC is needed to determine if their avoidance minimizes petaloid SD development.

Conflict of Interest Disclosures: Robert T. Brodell is a principal investigator for clinical trials (Novartis and Pfizer) the Corevitas psoriasis biologic registry, and owns stock in Veradermic, Inc. He serves on editorial boards of American Medical Student Research (faculty advisor); Practice Update Dermatology (Editor-in-Chief); Journal of the American Journal Academy of Dermatology (Associate Editor); Practical Dermatology; Journal of

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