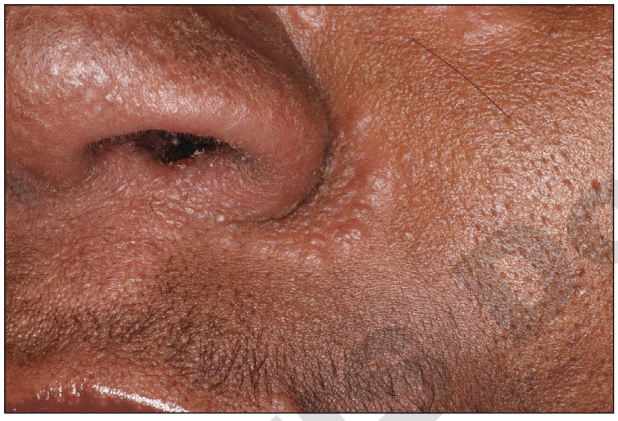
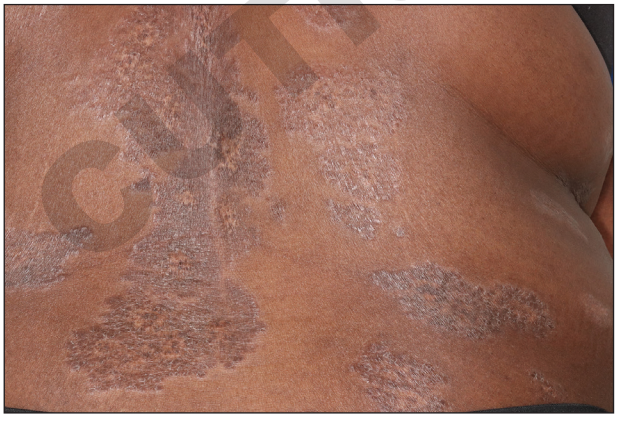
Asymptomatic Violaceous Plaques on the Face and Back

Ansley C. DeVore, MD; Dirk M. Elston, MD

Eligible for 1 MOC SA Credit From the ABD
This Photo Challenge in our print edition is eligible for 1 self-assessment credit for Maintenance of Certification from the American Board of Dermatology (ABD). After completing this activity, diplomates can visit the ABD website (http://www.abderm.org) to self-report the credits under the activity title "*Cutis* Photo Challenge." You may report the credit after each activity is completed or after accumulating multiple credits.





A 35-year-old Black woman presented to dermatology as a new patient for evaluation of an asymptomatic rash that had enlarged and spread to involve both the face and back over the last 4 months. She had not tried any treatments. She had no notable medical history and was uncertain of her family history. Physical examination showed indurated, flesh-colored to violaceous plaques around the alar-facial groove (top), nasal tip, chin, and back (bottom). The mucosae and nails were not involved.

WHAT'S THE **DIAGNOSIS?**

a. cutaneous sarcoidosis
b. discoid lupus erythematosus
c. mycosis fungoides
d. psoriasis
e. tinea infection

PLEASE TURN TO PAGE 27 FOR THE DIAGNOSIS

From the Medical University of South Carolina, Charleston. Dr. DeVore is from the College of Medicine, and Dr. Elston is from the Department of Dermatology and Dermatologic Surgery.
The authors report no conflict of interest.
Correspondence: Ansley C. DeVore, MD, Medical University of South Carolina, Department of Dermatology, 135 Rutledge Ave, 3rd Floor, Charleston, SC 29425 (devorea@musc.edu).
doi:10.12788/cutis.0925

THE **DIAGNOSIS:** Cutaneous Sarcoidosis

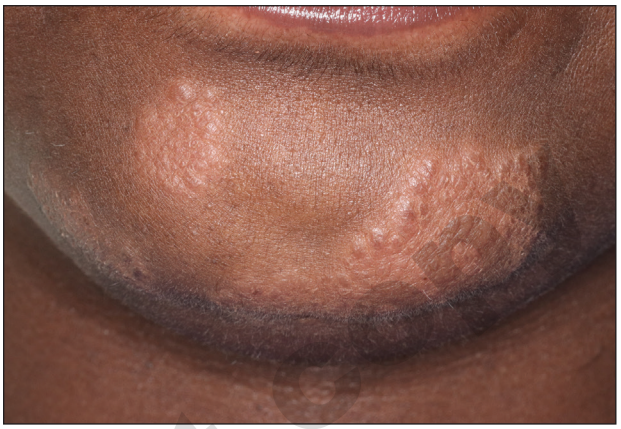
A biopsy of a plaque on the back confirmed cutaneous sarcoidosis (CS). A chest radiograph demonstrated hilar nodes, and a referral was placed for co-management with a pulmonologist. Histopathology was critical in making the diagnosis, with well-circumscribed noncaseating granulomas present in the dermis. The granulomas in CS often are described as naked, as there are minimal lymphocytes present and plasma cells normally are absent.[1] Because the lungs are the most common site of involvement, a chest radiograph is necessary to examine for systemic sarcoidosis. Laboratory workup is used to evaluate for lymphopenia, hypercalcemia, elevated blood sedimentation rate, and elevated angiotensin-converting enzyme levels, which are common in systemic sarcoidosis.[1]

Sarcoidosis is a multisystemic granulomatous disorder with an unknown etiology. It is believed to develop in genetically predisposed individuals as a reaction to unidentified antigens in the environment.[1] Helper T cells (T_H1) respond to these environmental antigens in those who are susceptible, which leads to the disease process, but paradoxically, even with the elevation of cellular immune activity at the sites of the granulomatous inflammation, the peripheral immune response in these patients is suppressed as shown by lymphopenia.[2]

Cutaneous sarcoidosis is found in approximately one-third of patients with systemic sarcoidosis but can occur without systemic involvement.[1,2] Sarcoidosis is reported worldwide and affects patients of all races and ethnicities, ages, and sexes but does have a higher prevalence among Black individuals in the United States, patients younger than 40 years (peak incidence, 20–29 years of age), and females.[2] In 80% of patients, CS occurs before systemic sarcoidosis develops, or they may develop simultaneously.[1]

Cutaneous sarcoidosis has a wide range of clinical presentations that are classified as specific and nonspecific. Specific lesions in CS contain noncaseating granulomas while nonspecific lesions in CS appear as reactive processes.[2] The most common specific presentation of CS includes papules that are brown in pigmentation in lighter skin tones and red to violaceous in darker skin tones (Figure). The most common nonspecific skin manifestation is erythema nodosum, which represents a hypersensitivity reaction. Cutaneous sarcoidosis can appear as hypopigmented or hyperpigmented patches or plaques.[1]

Treatments for CS vary based on the individual.[1] For milder and more localized cases, topical or intralesional steroids may be used. If systemic sarcoidosis is suspected or if there is diffuse involvement of the skin, systemic steroids, antimalarials (eg, hydroxychloroquine), low-dose methotrexate, minocycline, allopurinol, azathioprine, isotretinoin, tumor necrosis factor α inhibitors, or psoralen plus long-wave UVA radiation may be used. If systemic sarcoidosis is present, referral to a pulmonologist is recommended for co-management.[1]



Indurated, flesh-colored to violaceous plaques on the chin in a patient with cutaneous sarcoidosis.

Cutaneous sarcoidosis is known as the "great imitator," and there are multiple diseases to consider in the differential that are distinguished by the physical findings.[1] In our case of a middle-aged Black woman with indurated plaques, a few diagnoses to consider were psoriasis, discoid lupus erythematosus (DLE), mycosis fungoides (MF), and tinea infection.

Psoriasis is a common disease, and 90% of patients have chronic plaquelike disease with well-demarcated erythematous plaques that have a silver-gray scale and a positive Auspitz sign (also known as pinpoint bleeding).[3] Plaques often are distributed on the trunk, limb extensors, and scalp, along with nail changes. Some patients also have joint pain, indicating psoriatic arthritis. The etiology of psoriasis is unknown, but it develops due to unrestrained keratinocyte proliferation and defective differentiation, which leads to histopathology showing regular acanthosis and papillary dermal ectasia with rouleaux. Mild cases typically are treated with topical steroids or vitamin D, while more severe cases are treated with methotrexate, cyclosporine, retinoids, or biologics.[3]

Discoid lupus erythematosus occurs 4 times more often in Black patients than in White patients. Clinically, DLE begins as well-defined, erythematous, scaly patches that expand with hyperpigmentation at the periphery and leave an atrophic, scarred, hypopigmented center.[4] It typically is localized to the head and neck, but in cases where it disseminates elsewhere on the body, the risk

for systemic lupus erythematosus increases from 1.2% to 28%.[5] Histopathology of DLE shows vacuolar degeneration of the basal cell layer in the epidermis along with patchy lymphocytic infiltrate in the dermis. Treatments range from topical steroids for mild cases to antimalarial agents, retinoids, anti-inflammatory drugs, and calcineurin inhibitors for more severe cases.[4]

Although there are multiple types of cutaneous T-cell lymphoma, the most common is MF, which traditionally is nonaggressive. The typical patient with MF is older than 60 years and presents with indolent, ongoing, flat to minimally indurated patches or plaques that have cigarette paper scale. As MF progresses, some plaques grow into tumors and can become more aggressive. Histologically, MF changes based on its clinical stage, with the initial phase showing epidermotropic atypical lymphocytes and later phases showing less epitheliotropic, larger, atypical lymphocytes. The treatment algorithm varies depending on cutaneous T-cell lymphoma staging.[6]

Tinea infections are caused by dermatophytes. In prepubertal children, they predominantly appear as tinea corporis (on the body) or tinea capitis (on the scalp), but in adults they appear as tinea cruris (on the groin), tinea pedis (on the feet), or tinea unguium (on the nails).[7] Tinea infections classically are known to appear as an annular patch with an active erythematous scaling border and central clearing. The patches can be pruritic. Potassium hydroxide preparation of a skin scraping is a quick test to use in the office; if the results are inconclusive, a culture may be required. Treatment depends on the location of the infection but typically involves either topical or oral antifungal agents.[7]

REFERENCES

1. Tchernev G, Cardoso JC, Chokoeva AA, et al. The "mystery" of cutaneous sarcoidosis: facts and controversies. *Int J Immunopathol Pharmacol*. 2014;27:321-330. doi:10.1177/039463201402700302
2. Ali MM, Atwan AA, Gonzalez ML. Cutaneous sarcoidosis: updates in the pathogenesis. *J Eur Acad Dermatol Venereol*. 2010;24:747-755. doi:10.1111/j.1468-3083.2009.03517.x
3. Rendon A, Schäkel K. Psoriasis pathogenesis and treatment [published online March 23, 2019]. *Int J Mol Sci*. 2019;20:1475. doi:10.3390/ijms20061475
4. McDaniel B, Sukumaran S, Koritala T, et al. Discoid lupus erythematosus. *StatPearls* [Internet]. StatPearls Publishing; 2023. Accessed December 11, 2023. https://www.ncbi.nlm.nih.gov/books/NBK493145/
5. Bhat MR, Hulmani M, Dandakeri S, et al. Disseminated discoid lupus erythematosus leading to squamous cell carcinoma. *Indian J Dermatol*. 2012;57:158-161. doi:10.4103/0019-5154.94298
6. Pulitzer M. Cutaneous T-cell Lymphoma. *Clin Lab Med*. 2017; 37:527-546. doi:10.1016/j.cll.2017.06.006
7. Ely JW, Rosenfeld S, Seabury Stone M. Diagnosis and management of tinea infections. *Am Fam Physician*. 2014;90:702-710.