Bilateral Brownish-Red Indurated Facial Plaques in an Adult Man

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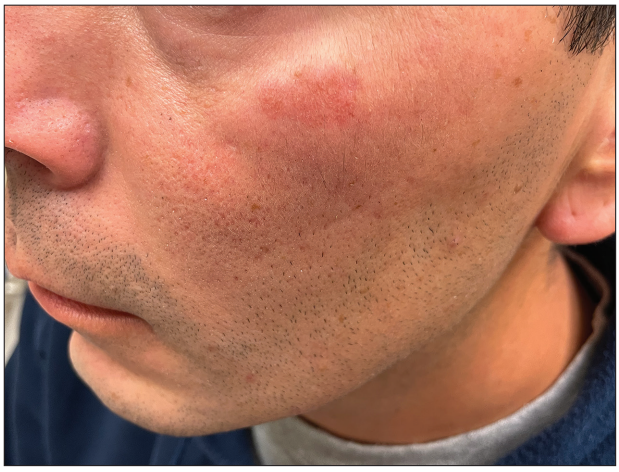

A 44-year-old man presented to the dermatology clinic with a facial rash of 2 years' duration. The patient reported associated pruritus but no systemic symptoms. His medical history was relevant for childhood eczema. He had tried various over-the-counter treatments for the facial rash, including topical hydrocortisone, neomycin/bacitracin/polymyxin antibiotic ointment, moisturizers, and antihistamines, with no success. Physical examination demonstrated symmetric, well-circumscribed, circinate, brownish-red, indurated plaques without scaling on the cheeks. A 4-mm punch biopsy was obtained from a plaque on the left cheek.

WHAT'S YOUR **DIAGNOSIS?**

a. cutaneous sarcoidosis
b. granuloma faciale
c. Jessner lymphocytic infiltrate
d. mycosis fungoides
e. tumid lupus erythematosus

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THE **DIAGNOSIS:** Granuloma Faciale

Histology revealed a dense mixed inflammatory cell infiltrate with conspicuous neutrophils and eosinophils in the upper to mid dermis with a narrow uninvolved grenz zone beneath the epidermis (Figures 1 and 2). These findings along with the clinical presentation (Figure 3) were consistent with a diagnosis of granuloma faciale (GF). Most often seen in middle-aged White men, GF is an uncommon localized inflammatory skin condition that often manifests as a single, well-defined, red-to-brown papule, nodule, or plaque on the face or other sun-exposed areas of the skin. Since numerous other skin diseases manifest similarly to GF, biopsy is necessary for definitive diagnosis.[1] Histopathology of GF classically shows a mixed inflammatory infiltrate with a narrow band of uninvolved dermis separating it from the epidermis (grenz zone). Dilated follicular plugs and vascular changes frequently are appreciated. Despite its name, GF does not include granulomas and is thought to be similar to leukocytoclastic vasculitis.[1] Reports of GF in the literature have shown immunohistochemical staining with the presence of CD4+ lymphocytes that secrete IL-5, a chemotactic agent responsible for attracting eosinophils that contributes to the eosinophilic infiltrate on histology.[2]

Topical corticosteroids and topical tacrolimus are the first-line treatments for GF. Intralesional corticosteroids also are a treatment option and can be used in combination with cryotherapy.[1,3] Additionally, both topical and oral dapsone have been shown to be effective for GF.[1] Oral dapsone is given at a dose of 50 mg to 150 mg once daily.[1] Clofazimine, typically used as an antileprosy treatment, also has been efficacious in treating GF. Clofazimine has anti-inflammatory and antiproliferative effects on lymphocytes that may attenuate the inflammation underlying GF. It is prescribed at a dose of 300 mg once daily for 3 to 5 months.[1]

The differential diagnosis for GF is broad and includes tumid lupus erythematosus, Jessner lymphocytic infiltrate (JLI), cutaneous sarcoidosis, and mycosis fungoides. Tumid lupus erythematosus is a subtype of cutaneous lupus erythematosus that rarely is associated with systemic lupus manifestations. Tumid lupus erythematosus manifests as annular, indurated, erythematous plaques, whereas JLI manifests with erythematous papular to nodular lesions without scale on the upper back or face.[4] Jessner lymphocytic infiltrate and tumid lupus erythematosus are histopathologically identical, with abundant dermal mucin deposition and a superficial and deep perivascular

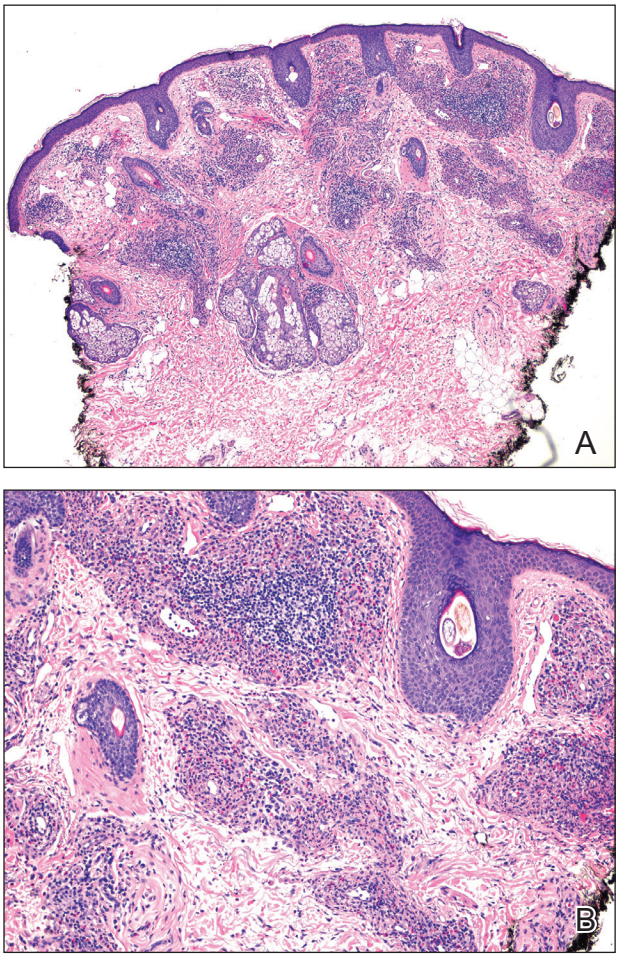

FIGURE 1. A and B, Dense mixed inflammatory cell infiltrate in the upper to mid dermis with a narrow uninvolved grenz zone beneath the epidermis (H&E, original magnifications ×4 and ×10).).

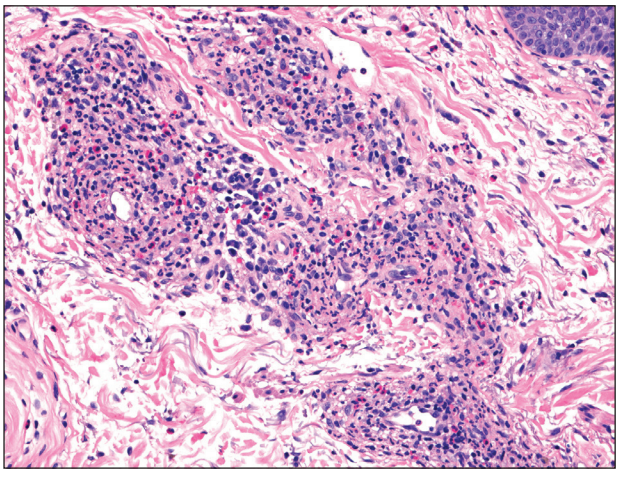

FIGURE 2. Higher-powered magnification revealed conspicuous neutrophils and eosinophils in the upper to mid dermis demonstrating perivascular accentuation (H&E, original magnification ×40).

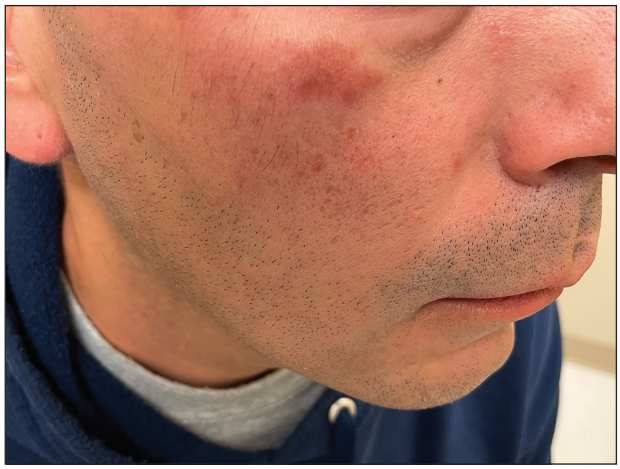

FIGURE 3. Granuloma faciale plaque on right cheek.

and periadnexal lymphocytic infiltrate. It is debatable whether JLI is a separate entity or a variant of tumid lupus erythematosus. Sarcoidosis is a granulomatous disease that manifests with a myriad of clinical features. The skin is the second most commonly involved organ.[5] The most common morphology is numerous small, firm, nonscaly papules, typically on the face. Histology in cutaneous sarcoidosis will show lymphocyte-poor, noncaseating epithelioid cell granulomas with positive reticulin staining, which were not seen in our patient.[6] Lastly, mycosis fungoides is the most common type of cutaneous T-cell lymphoma. It can manifest as patches, plaques, or tumors. The plaque stage may mimic GF as lesions are infiltrative, annular, and raised, with well-defined margins. Histopathology will show intraepidermal lymphocytes out of proportion with spongiosis.[7]

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