Annular Erythematous Plaques on the Back

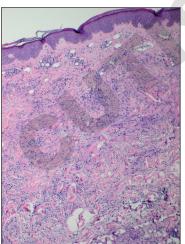
Lauren Schwartzberg, DO; Emily McEldrew, DO; Marisa Baldassano, MD; Howard Altman, MD; Anthony Gust, MD

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An 84-year-old man presented to the clinic for evaluation of a pruritic rash on the back of 6 months' duration that spread to the neck and chest over the past 2 months and then to the abdomen and thighs more recently. His primary care provider prescribed a 1-week course of oral steroids and steroid cream. The oral medication did not help, but the cream alleviated the pruritus. He had a medical history of coronary artery disease, hypertension, and diabetes mellitus. He also had a rash on the forearms that had waxed and waned for many years but was not associated with pruritus. He had not sought medical care for the rash and had never treated it. Physical examination revealed pink to violaceous annular plaques with central clearing and raised borders that coalesced into larger plaques on the trunk (top). Dusky, scaly, pink plaques were present on the dorsal forearms. Three punch biopsies—2 from the upper back (bottom) and 1 from the left forearm—all demonstrated consistent findings.



H&E, original magnification ×10.

THE BEST **DIAGNOSIS IS:**

- a. erythema gyratum repens
- b. granuloma annulare
- c. lepromatous leprosy
- d. mycosis fungoides
- e. sarcoidosis

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Drs. Schwartzberg, McEldrew, Altman, and Gust are from the Department of Dermatology, Lehigh Valley Health Network, Allentown, Pennsylvania. Dr. Baldassano is from Baldassano Dermatopathology, Blue Bell, Pennsylvania.

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Correspondence: Lauren Schwartzberg, DO, 1259 S Cedar Crest Blvd #100, Allentown, PA 18103 (Ischwartzberg@adaltd.com). doi:10.12788/cutis.0941

THE **DIAGNOSIS**:

Granuloma Annulare

he biopsies revealed palisading granulomatous dermatitis consistent with granuloma annulare (GA). This diagnosis was supported by the clinical presentation and histopathologic findings. Although the pathogenesis of GA is unclear, it is a benign, self-limiting condition. Primarily affected sites include the trunk and forearms. Generalized GA (or GA with ≥10 lesions) may warrant workup for malignancy, as it may represent a paraneoplastic process. Histopathology reveals granulomas comprising a dermal lymphohistiocytic infiltrate as well as central mucin and nuclear debris. There are a few histologic subtypes of GA, including palisading and interstitial, which refer to the distribution of the histiocytic infiltrate.^{2,3} This case—with palisading histiocytes lining the collection of necrobiosis and mucin (bottom guiz image)—features palisading GA. Notably, GA exhibits central rather than diffuse mucin.4

Erythema gyratum repens is a paraneoplastic arcuate erythema that manifests as erythematous figurate, gyrate, or annular plaques exhibiting a trailing scale. Clinically, erythema gyratum repens spreads rapidly—as quickly as 1 cm/d—and can be extensive (as in this case). Histopathology ruled out this diagnosis in our patient. Nonspecific findings of acanthosis, parakeratosis, and superficial spongiosis can be found in erythema gyratum repens. A superficial and deep perivascular lymphohistiocytic infiltrate may be seen in figurate erythemas (Figure 1). Unlike GA, this infiltrate does not form granulomas, is more superficial, and does not contain mucin.

Histopathology also can help establish the diagnosis of leprosy and its specific subtype, as leprosy exists on a spectrum from tuberculoid to lepromatous, with a great deal of overlap in between.⁶ Lepromatous leprosy has many cutaneous clinical presentations but typically manifests as erythematous papules or nodules. It is multibacillary, and these mycobacteria form clumps known as globi that can be seen on Fite stain.⁷ In lepromatous leprosy, there is a characteristic dense lymphohistiocytic infiltrate (Figure 2) above which a Grenz zone can be seen.^{4,8} There are no well-formed granulomas in lepromatous leprosy, unlike in tuberculoid leprosy, which is paucibacillary and creates a granulomatous response surrounding nerves and adnexal structures.⁶

Mycosis fungoides (MF) is the most common cutaneous lymphoma. There are patch, plaque, and tumor stages of MF, each of which exhibits various histopathologic findings. In early patch-stage MF, lymphocytes have perinuclear clearing, and the degree of lymphocytic infiltrate is out of proportion to the spongiosis present. Epidermotropism and

Pautrier microabscesses often are present in the epidermis (Figure 3). In the plaque stage, there is a denser lymphoid infiltrate in a lichenoid pattern with epidermotropism and Pautrier microabscesses. The tumor stage shows a dense dermal lymphoid infiltrate with more atypia and typically a lack of epidermotropism. Rarely, MF can exhibit a granulomatous variant in which epithelioid histiocytes collect to form granulomas along with atypical lymphocytes.¹⁰

The diagnosis of cutaneous sarcoidosis requires clinicopathologic corroboration. Histopathology demonstrates epithelioid histiocytes forming noncaseating

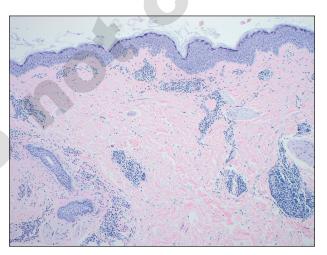


FIGURE 1. Figurate erythema demonstrates a superficial and deep perivascular lymphocytic infiltrate (H&E, original magnification ×10).

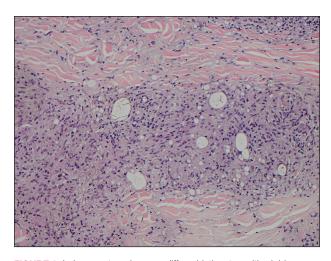


FIGURE 2. In lepromatous leprosy, diffuse histiocytes with globi are seen (H&E, original magnification ×20).

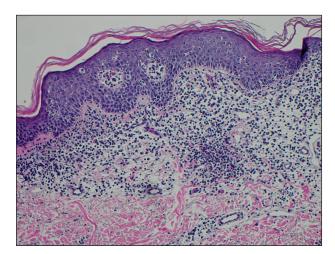


FIGURE 3. In patch-stage mycosis fungoides, atypical lymphocytes migrating from the dermis to the epidermis, consistent with epidermotropism, are seen (H&E, original magnification ×20).

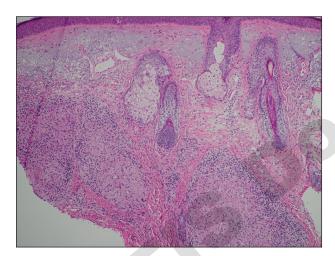


FIGURE 4. In sarcoidosis, epithelioid histiocytes form granulomas with minimal surrounding lymphocytic infiltrate (H&E, original magnification ×10).

granulomas with little to no lymphocytic infiltrate (Figure 4). There typically is no necrosis or necrobiosis as there is in GA. The diagnosis of sarcoidosis can be challenging histopathologically, and stains should be used to rule out infectious processes. Asteroid bodies—star-shaped eosinophilic inclusions within giant cells—may be present but are nonspecific for sarcoidosis. Schaumann bodies—inclusions of calcifications within giant cells—also may be present and can aid in diagnosis.

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