

Annular Atrophic Plaque with Peripheral Crusted Papules

A 50-year-old male presented to the dermatology outpatient with a dark elevated lesion over left hand for 15–20 years. He had received 6 months of antitubercular therapy for pulmonary tuberculosis, 8 years ago. On cutaneous examination, there was an ill-defined atrophic plaque, roughly measuring 4 × 5 cm, over the dorsum of left hand with few hyperpigmented papules at its disto-lateral end, some of which had areas of depression [Figure 1]. Laboratory workup was significant for raised glycosylated hemoglobin (9.1%), fasting and postprandial plasma glucose (216 mg/dL and 395 mg/dL respectively). Histopathological analysis was subsequently performed from skin biopsy sample that revealed necrobiotic granulomas in upper dermis focally transgressing through the epidermis. The granulomas had a central zone of necrobiosis with mucin deposition and were surrounded by palisading histiocytes and perivascular lymphocytes [Figures 2-4].

Diagnosis?

Perforating granuloma annulare.

Discussion

Granuloma annulare (GA) is a relatively common skin condition with a study reporting up to 0.1% to 0.4% of new patients being diagnosed with GA in dermatology setup.^[1] It is a spectrum of disease with significant overlap between various subtypes, most commonly recognized of which is the localized variant. Perforating GA is one of the less common subtypes.

It has been hypothesized that perforating GA undergoes four stages of progression, thereby leading to its varied clinical picture.^[2] The most commonly reported feature is an umbilicated papule with

crust.^[3] The lesion may initially be pustular or may ulcerate to later regress with punctate scarring. The above hypothesis may explain the presentation with papules and scar in the current case that gave it the appearance of having an advancing edge.

The clinical picture in this patient mimicked lupus vulgaris that usually presents as a plaque with an advancing edge and areas of scarring. A past history of tuberculosis added to the already present confusion, especially in a set-up of tuberculosis-endemic region, but the absence of caseating granuloma and failure to demonstrate acid-fast bacilli on biopsy favored against this diagnosis. Palisaded neutrophilic granulomatous dermatitis (PNGD) forms a close differential, as it presents as linear cords, papules, plaques or nodules, mainly over acral areas. Histological features depend upon the stage of the disease evolution. As compared to GA, PNGD shows more neutrophils, karyorrhectic debris, and fibrin, while mucin is less prominent. In our case, the clinicopathological features were characteristic of GA. Elastosis perforans serpiginosa presents clinically as small grouped papules in circinate pattern mostly on neck and upper trunk, which is unlike present case. In addition, histologically it shows altered brightly eosinophilic elastic fibers, instead of necrobiotic palisading granulomas. Verhoeff's Van Gieson stain failed to demonstrate altered elastic fibers in our case, thereby ruling out this possibility.

The clinical morphology simulated keratoacanthoma centrifugum marginatum, discoid lupus erythematosus, and sarcoidosis, but their respective characteristic histological features are pseudoepitheliomatous hyperplasia with dense dermal mixed inflammatory infiltrate, interface dermatitis with thickening of basement membrane, and noncaseating

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Figure 1: A 4 × 5 cm atrophic plaque over the dorsum of left hand with few hyperpigmented papules at disto-lateral end

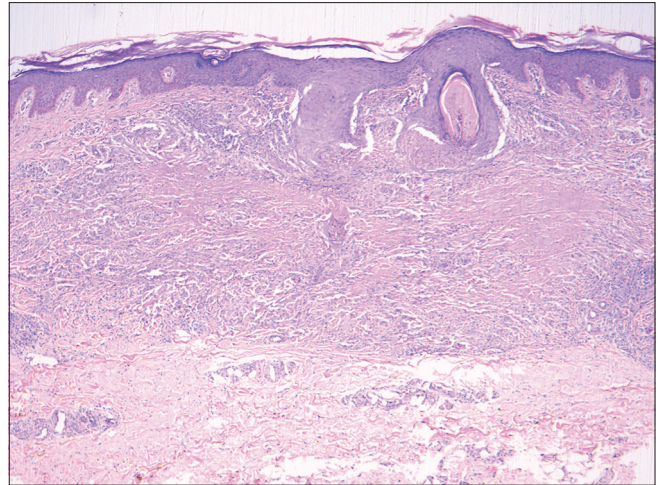


Figure 2: Upper dermis shows large palisading granulomas with central zone of necrobiosis surrounded by histiocytes (H & E, 40×)

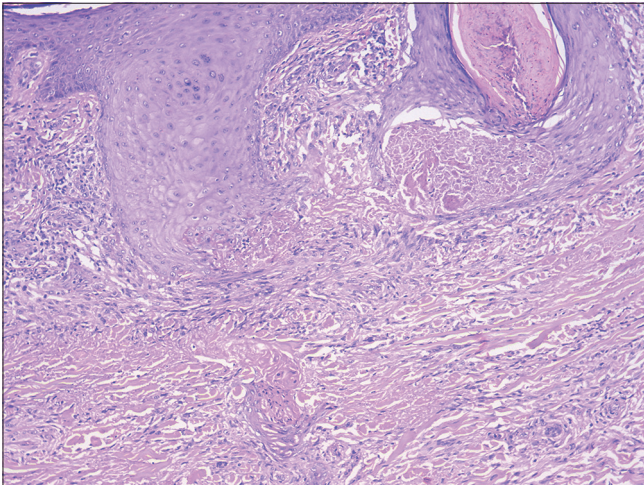


Figure 3: Necrobiotic collagen is seen transgressing through a perforating channel formed by epidermal downgrowths (H & E, 100×)

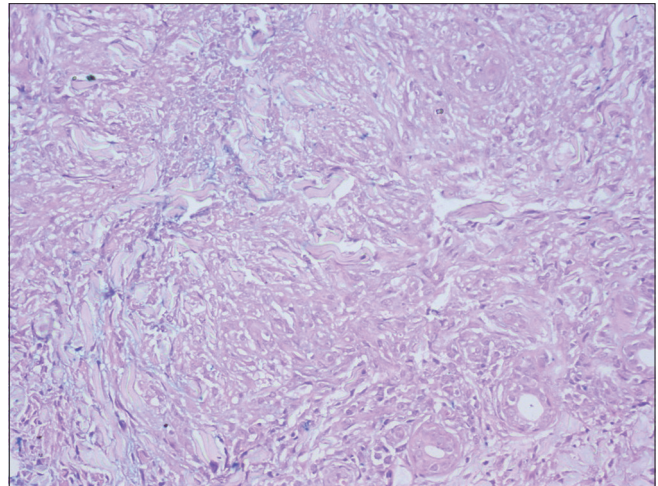


Figure 4: Mucin deposition in the area of necrobiosis (Alcian Blue, 200×)

granuloma with paucity of lymphocytes, which were all absent in our case. Acquired perforating dermatosis that presents clinically as itchy umbilicated papulonodules with central keratin plug, and transepithelial elimination of dermal elements on histology were differentiated on clinical grounds as well as by presence of palisading necrobiotic granuloma that is the characteristic histological feature of GA.

The histological picture of GA shows granulomatous inflammation in palisading or interstitial pattern and a peculiar feature of perforating GA is transepithelial

elimination of altered collagen.^[3,4] Histopathology of this case revealed features that were typical of perforating GA and did not leave any ground for doubt. Perforating disorders have also been found to be associated with diabetes mellitus, which was incidentally found in our patient as well.^[5] A number of therapeutic modalities have been tried for perforating GA with variable results, most of which are unsatisfactory.^[6] Our patient received topical tacrolimus to which he was unresponsive.

This patient under consideration presented with a morphology resembling lupus vulgaris and would have been missed if not for the typical histology features. To the best of our knowledge, this presentation is rare with perforating GA manifesting as annular atrophic plaque. He did not give any history of extrusion of material from the lesion, nor did he complain of polyuria and polydipsia, that could be attributed to his inattention due to low literacy. This is to assert that not every atrophic plaque with an apparent advancing edge is tuberculosis, even in

an endemic state. A broader outlook, a lower threshold for biopsy, and a wider set of differentials are necessary to cover the other possibilities.

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Conflicts of interest

There are no conflicts of interest.

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