An 84-year-old man presented with gradually enlarging, asymptomatic, erythematous to violaceous plaques on the face and scalp of 11 years’ duration ranging in size from 0.5×0.5 cm to 10×8 cm. The plaques were unresponsive to treatment with topical steroids. The lesions were nontender with no associated bleeding, burning, or pruritus. The patient denied any trauma to the sites or systemic symptoms. He had a history of essential hypertension and benign prostatic hyperplasia and had been taking ramipril, tamsulosin, and dutasteride for 5 years. His medical history was otherwise unremarkable, and routine laboratory findings were within normal range.

WHAT’S THE DIAGNOSIS?

- a. cutaneous lymphoma
- b. cutaneous pseudolymphoma
- c. cutaneous sarcoidosis
- d. erythema elevatum diutinum
- e. granuloma faciale

Please turn to page E12 for the diagnosis.
A biopsy from a scalp lesion showed an intense mixed inflammatory infiltrate mainly consisting of eosinophils, but lymphocytes, histiocytes, neutrophils, and plasma cells also were present. A grenz zone was observed between the dermal infiltrate and epidermis. Perivascular infiltrates were penetrating vessel walls, and hyalinization of the vessel walls also was seen (Figure 1). Direct immunofluorescence demonstrated IgG positivity on vessel walls (Figure 2). A diagnosis of granuloma faciale with extrafacial lesions was made. Twice daily application of tacrolimus ointment 0.1% was started, but after a 10-month course of treatment, there was no notable difference in the lesions.

Granuloma faciale (GF) is an uncommon benign dermatosis of unknown pathogenesis characterized by erythematous, brown, or violaceous papules, plaques, or nodules. Granuloma faciale lesions can be solitary or multiple as well as disseminated and most often occur on the face. Predilection sites include the nose, periauricular area, cheeks, forehead, eyelids, and ears; however, lesions also have been reported to occur in extrafacial areas such as the trunk, arms, and legs.1-4 In our patient, multiple plaques were seen on the scalp. Facial lesions usually precede extrafacial lesions, which may present months to several years after the appearance of facial disease; however, according to our patient’s history his scalp lesions appeared before the facial lesions.

The differential diagnoses for GF mainly include erythema elevatum diutinum, cutaneous sarcoidosis, cutaneous lymphoma, lupus, basal cell carcinoma, and cutaneous pseudolymphoma.5 Diagnosis may be established based on a combination of clinical features and skin biopsy results. On histopathologic examination, small-vessel vasculitis usually is present with an infiltrate predominantly consisting of neutrophils and eosinophils.6

It has been suggested that actinic damage plays a role in the etiology of GF.7 The pathogenesis is uncertain, but it is thought that immunophenotypic and molecular analysis of the dermal infiltrate in GF reveals that most lymphocytes are clonally expanded and the process is mediated by interferon gamma.7 Tacrolimus acts by binding and inactivating calcineurin and thus blocking T-cell activation and proliferation, so it is not surprising that topical tacrolimus has been shown to be useful in the management of this condition.8
REFERENCES