or other tissues (somatic mosaicism). In the present case, the pattern of mosaicism is difficult to classify because mosaicism of adipocytes, which are mesodermal not ectodermal tissue, is currently not well understood. It is very likely, however, that this type of segmental lipodystrophy reflects mosaicism. Investigating genes known to cause inherited, systemic lipodystrophies (eg, AGTAT2, seipin, LMNA, PPARγ, and ZMPSTE24) could aid in proving a genetic basis of mosaicism in the present unclassified segmental lipodystrophy. However, we were unable to perform this genetic studies because our patient refused further evaluation.

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CASE LETTERS

Erosive pustular dermatosis of the scalp following topical methylaminolaevulinate photodynamic therapy

To the Editor: Erosive pustular dermatosis of the scalp (EPDS) is a rare inflammatory disease of unknown etiology that usually occurs in the elderly. It is characterized by sterile pustules, chronic crusted erosions, cicatricial alopecia, and skin atrophy.

A 93-year-old, otherwise healthy female presented to our department with a 2-month history of eroded, pustular, and crusted lesions limited to the scalp. According to the referral notes, she had longstanding female-androgenetic alopecia, and had actinic keratoses on the scalp that were treated with two sessions of topical methylaminolaevulinate photodynamic therapy (MAL-PDT), with improvement of the condition. The patient had superficial curettage of the lesions before the first session, followed by the application of methylaminolaevulinate cream (Metvix; Galderma, Fort Worth, TX) for 3 hours under an occlusive, light-protecting dressing; the area was then irradiated with 75 J/cm² of red light. The photodynamic therapy was repeated 1 week later. Twenty-eight days after the first treatment, burning erosions developed, extending slowly but progressively. Treatment with topical antibiotics (fusidic acid cream and mupirocin) and systemic antibiotics (amoxicillin/clavulanate) was unsuccessful.

The physical examination revealed multiple pustules, erosions, scales, and crusts on the frontoparietal and temporoparietal scalp; gentle removal of the crusts revealed a moist, atrophic surface with telangiectasias and minute erosions exuding yellowish seropurulent material. Areas of scarring alopecia were also evident (Fig 1). The remainder of the skin examination was essentially normal, with no features of a blistering disorder or psoriasis. No

Fig 1. Clinical appearance of the scalp: diffuse crusting associated with multiple pustular, exudative, and erosive lesions.
clinical evidence of the original actinic keratoses was detected.

Cultures from the pustules and erosions failed to show bacterial or mycological growth. Routine laboratory blood tests were normal except for elevated values of markers of inflammation (erythrocyte sedimentation rate, C-reactive protein, and hypergammaglobulinemia); autoantibodies (antinuclear antibodies, rheumatoid factor, thyroglobulin, and microsomal antibodies) were negative. A histologic examination of a 3-mm punch biopsy showed a dense, mainly perifollicular, dermal infiltrate of neutrophils and lymphocytes, with a loss of normal collagen architecture, vasodilatation, and angiogenesis (Fig 2). There was no evidence of malignancy or vasculitis, and a periodic acid-Schiff stain was negative.

The clinical and histopathologic features were consistent with a diagnosis of EPDS. The patient was treated with oral methylprednisolone (16 mg/day with progressive tapering) in combination with topical gentamycin-betamethasone cream, resulting in marked improvement of lesions and partial resolution of the cutaneous atrophy at 3 months of follow-up. Residual scarring alopecia was evident.

Since the first report by Pye et al. in 1979, several cases of EPDS have been reported. The disease is an uncommon condition that occurs mainly in the elderly, with a slight preference for females. It characteristically develops in atrophic sun-damaged skin. It is characterized by sterile pustules with a nonspecific inflammatory infiltrate. It must be differentiated from folliculitis decalvans, pyoderma gangrenosum, and cicatricial pemphigoid. The nonspecific histopathologic pattern, the evolution leading to scarring alopecia, and the resistance to antibiotics, with response to steroids, favor the diagnosis.

Although the pathophysiologic mechanisms remain obscure, it is generally believed that local trauma acts as a triggering factor. EPDS has been widely reported in the literature following the treatment of actinic keratoses or squamous cell carcinoma with topical 5% fluorouracil or tretinoin. It may also occur days to years after incidental blunt trauma, surgery, cryotherapy, or radiotherapy. Response to treatment is variable. Potent topical steroids have been widely used in EPDS, and anecdotal reports have described a partial response to oral isotretinoin, dapson, and nimesulide. Recently, calcipotriol cream and tacrolimus ointment have been used successfully as alternative therapies.

To our knowledge, none of the previous reported side effects of MAL-PDT have included chronic pustules, scale crusts, and nonhealing erosions. In our opinion, EPDS should be considered in any subject developing a chronic inflammatory response after MAL-PDT.

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