The need to comprehend erosive pustular dermatosis of the scalp is coming to a head

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Forty years ago, Pye et al[1] described "erosive pustular dermatitis of the scalp" (EPDS). "All the patients were elderly women who developed chronic, extensive, pustular, crusted and occasionally eroded lesions of the scalp which produced scarring alopecia. Investigations were essentially negative and skin biopsies showed only nonspecific changes of atrophy and chronic inflammation. … The condition did not respond to antibiotics, but was suppressed by potent topical steroids."

EPDS has a mean age of onset of 60 to 70 years, although it has been reported from infancy to 95 years. A female propensity is observed, with an estimated female-to-male ratio of 2:1.[2] In my experience, EPDS is more commonly observed in men with androgentic alopecia and a history of nonmelanoma skin cancer of the scalp. Erosive pustular dermatosis may also be observed on other sites (face, legs).

The diagnosis of EPDS is one of exclusion, after ruling out infectious causes (bacterial or fungal), autoimmune blistering diseases (pemphigus or cicatricial pemphigoid), or neoplastic processes (squamous cell carcinoma) by appropriate biopsies and cultures.

The pathogenesis of EPDS is unknown, but the disease has been attributed to trauma (secondary to surgical or topical chemotherapy for actinic damage or nonmelanoma skin cancer, burns, zoster, or contact dermatitis) and an altered immune response due to aging (immunosenescence). Ibrihim et al state,[3] "an aberrant wound healing response in the setting of actinically damaged skin seems to be a consistent theme in its presentation."

Histologic features of EPDS are considered nonspecific with acute to chronic inflammation and fibrosis. In this issue of *Journal of the American Academy of Dermatology*, Tomasini and Michelerio performed a retrospective study of 30 patients with EPDS (age range, 63-89 years; 22 men and 8 women). Androgenetic alopecia was present in 19 of 30 patients. Triggering factors included mechanical trauma in 10 of 30 patients, surgical procedures in 4 of 30 patients, and herpes zoster in 1 of 30 patients. Three patients were affected by autoimmune disorders (autoimmune hypothyroidism, collagenous colitis, and undifferentiated collagen vascular disease). The vertex was the most common location. Disease presentation varied from erosive, scaly lesions to crusted and hemorrhagic plaques, mimicking pustular pyoderma gangrenosum. The pathologic changes differed according to lesion type and disease duration. A spongiotic and suppurative infundibular folliculitis was observed in 8 of 30 patients. The authors proposed that the primary lesion of erosive pustular dermatosis of the scalp is a spongiotic, pustular superficial folliculitis. They suggested that EPDS is part of the neutrophilic dermatoses spectrum, related to pustular pyoderma gangrenosum, where pathergy plays a pathogenic role.[4]

This hypothesis, if correct, has important implications because clinicians would consider the possibility of associated disorders, rather than managing EPDS as a cutaneous disorder sui generis. Aside from reports of associated autoimmune disorders, EPDS has been observed in an 89-year-old man with myelodysplastic syndrome.[5] EPDS is difficult to treat, with the first-line therapies being ultrapotent topical steroids or calcineurin inhibitors. Gaining further insight into the pathogenesis of EPDS is essential to developing appropriate management strategies.

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