

Erosive Pustular Dermatitis of the Scalp Treated with 0.1% Mometasone Furoate Cream

INTRODUCTION

Erosive pustular dermatosis of the scalp (EPD) is a seldom reported skin disease characterized by sterile pustules, chronic crusted erosions, and skin atrophy following physical or chemical trauma. EPD is seen mostly in elderly patients with long-term sun damage to the skin. We report on a case of a middle-aged man with androgenic alopecia who developed EPD lesions confined to the bald scalp.

CASE REPORT

A 49-year-old, Caucasian man was referred to our department with a 2-week history of erythema, multiple pustules and crusted erosions limited to the scalp (Figure 1). The lesions had gradually spread and converged, but remained confined to the site of previous androgenic balding of the scalp. According to the referral notes, the patient was treated for 8 days with oral limecycline (300mg), topical erythromycin (Davercin), and topical octenidine (Octenisept) with

no effect. The patient had a history of cranial trauma 1 year ago, which resulted in two corrective surgeries 7 months and 1 month before admittance to our department, but was otherwise healthy and denied taking any medications. In routine laboratory examinations, only C-reactive protein was found to be moderately elevated (10.4 mg/L; normal <5 mg/L).

After referral to the our clinic, topical treatment with fusidic acid (Fucidin), 0.1% amikacin ointment, and terbinafine cream (Terbiderm) was initiated with only moderate effects. Consequently, microbiological swabs and a skin biopsy were performed.

Single colonies of coagulase negative Staphylococci were found in cultures from lesion swabs, what was considered physiologic microflora. Direct and indirect immunofluorescence microscopy studies of lesional skin biopsy were negative. Light microscopy of skin biopsy specimens revealed areas of epidermal erosion accompanied by a polymorphous dermal

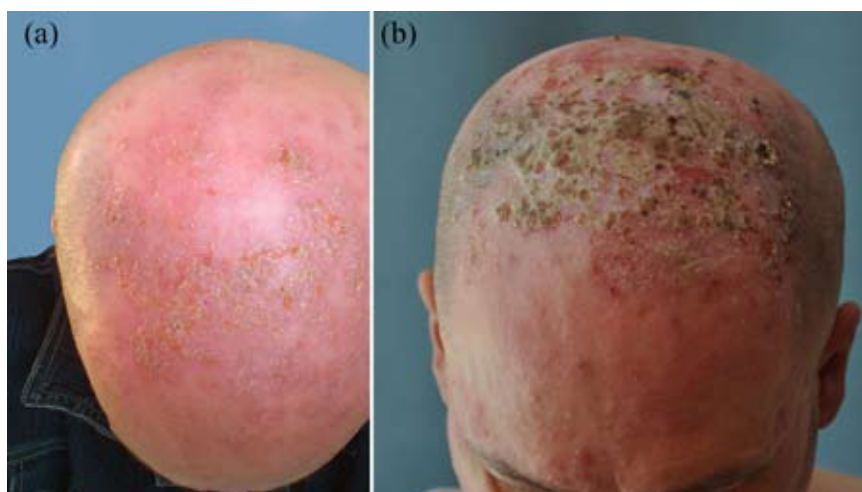


Figure 1. Clinical appearance of the scalp: a) multiple pustular and exudative lesions. Patient's self-documentation of early-onset lesions; b) diffuse crusting and erosions of the scalp. Photograph taken 2 weeks into the course of the disease, after 8 days of unsuccessful treatment with oral limecycline (300mg), topical erythromycin (Davercin), and topical octenidine (Octenisept).

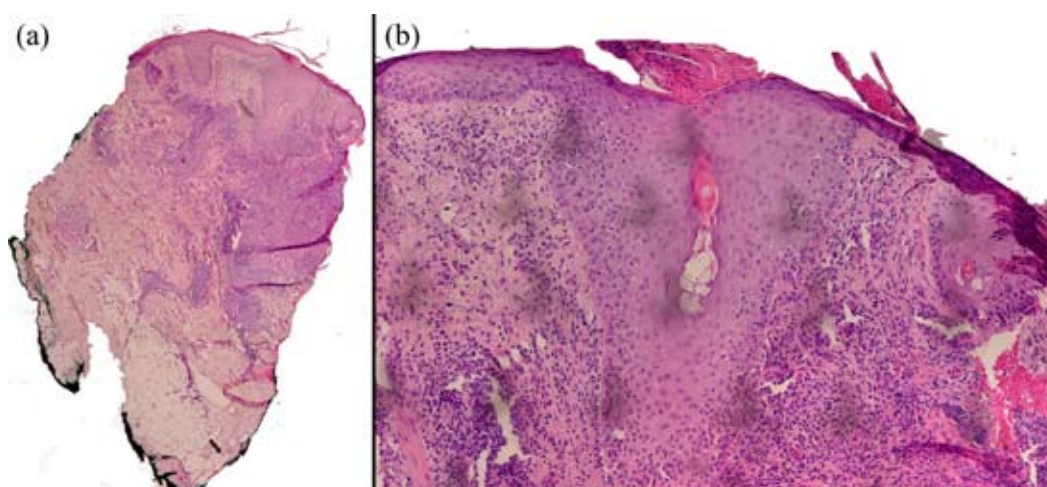


Figure 2. Hemalaun and eosin (H&E) staining of a patient's scalp skin biopsy.

(a) polymorphous dermal inflammatory infiltrate composed mainly of lymphocytes, neutrophils, and histiocytes. Low-power magnification $\times 40$; (b) medium-power magnification $\times 100$.

inflammatory infiltrate composed mainly of lymphocytes, neutrophils and histiocytes with occasional eosinophils, and foreign body type giant cells. Single subcorneal pustules were seen. In non-eroded areas, the epithelium was edematous with acanthosis and parakeratosis. There was a focal infiltration of epithelium by neutrophils (Figure 2).

Erosive pustular dermatosis of the scalp was diagnosed. We initiated treatment with topical 0.1% mometasone furoate cream (Elocom). The scalp responded well with no side effects. Lesions cleared within 6 days but the patient was lost to further follow-up (Figure 3).

DISCUSSION AND CONCLUSIONS

Erosive pustular dermatosis is an inflammatory skin disease of unknown etiology, characterized by multiple chronic, amicrobial pustules and crusted erosions occurring on the scalp or lower extremities. Since it was described, only approximately 150 cases have been reported in the literature. However Patton et al. suggest that EPD is a relatively common condition, though widely underrecognized (1,2).

Eruption of EPD lesions has been reported in relation to trauma to the previously atrophic skin. Typically, lesions arise in sites of cutaneous atrophy due to actinic damage or chronic venous insufficiency, although a Koebner-like phenomenon is sometimes reported, with lesions occurring in surgical or burn scars (3). List of triggering factors include herpes zoster infection and physical trauma such as surgery, cryosurgery, birth trauma, and compression therapy,



Figure 3. Clinical appearance of the scalp. Photograph taken 4 weeks into the course of the disease – improvement after 6 days of topical Elocom cream, only erythema remains.

radiation therapy and irritation due to topical 5-fluorouracil, tretinoin, imiquimod, and methyl aminolevulinate photodynamic therapy (4-6).

The clinical course of EPD is variable, and often includes chronic-relapsing. Successful topical therapies with potent corticosteroids, tacrolimus, calcipotriol, dapsone, and photolyase have been reported. Oral steroids, isotretinoin, and dapsone have been used with variable response (7-10). Although considered

a triggering factor, successful treatment with methyl aminolevulinate photodynamic therapy has also been reported.

The advantage of potent topical steroids, such as mometasone furoate, in EPD treatment is quick response and low cost. However, in refractory cases, topical steroids should be substituted with topical 0.1% tacrolimus to prevent exacerbation of skin atrophy with topical steroids.

Although EPD has been described mostly in the elderly, we believe that EPD should also be included in differential diagnosis of pustular lesions on the scalp in younger patients with sun damaged skin.

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