Erosive Pustular Dermatosis of the Scalp

Treatment With Topical Tacrolimus

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REPORT OF CASES

CASE 1

A 65-year-old man had an 8-year history of erosive, pustular, and atrophic lesions on his alopecic scalp (Figure 1). The lesions developed since 1992, after treatment of actinic keratoses by topical fluorouracil and local trauma. Histologic examination of a skin biopsy specimen revealed an ulcerated atrophic epidermis with parakeratosis; a chronic inflammatory dermal infiltrate composed of lymphocytes, macrophages, and neutrophils; and a complete absence of hair follicles. Results of direct immunofluorescence microscopy and microbiologic examination were negative. Blood zinc level was within normal limits, and serum immunoelectrophoresis did not show a monoclonal gammopathy. No evidence for an underlying systemic disease was found to suggest superficial pyoderma gangrenosum. The diagnosis of erosive pustular dermatosis of the scalp (EPDS) was then made. The patient had been treated in 1993 with a 1-year course of topical 0.05% retinoic acid, and thereafter with a 5-month course of 30-mg/d oral isotretinoin, which resulted in worsening of the lesions. Furthermore, a 2-month trial with 100 mg/d of oral sulfapyridine was ineffective. From 1995 to 1999, repeated applications of potent topical corticosteroids (clobetasol propionate and mometasone furoate) improved the skin disease, but also increased the skin atrophy (Figure 2), with erythema and telangiectasias.

CASE 2

A 55-year-old man presented in 2001 with extensive actinic keratoses of his alopecic and sun-damaged scalp. From 1998 to 1999, he underwent several courses of topical fluorouracil and ablative treatment with an ultrapulse carbon dioxide laser. After laser therapy, the treated area developed an inflammatory reaction with a wide, erosive, pustular, crusted, and atrophic eruption. The lesions persisted for 2 years with regular flares, without any response to topical or systemic antibiotic therapy. General clinical examination was normal, and complete blood cell count was within normal limits. The diagnosis of EPDS was clinically made, and no skin biopsy was performed.

Figure 1. Atrophic, pustular, erosive, and crusted lesion of the scalp of patient 1.

Figure 2. Patient 1. After 4 years of treatment with topical clobetasol propionate or mometasone furoate, partial control of the eruption and worsening of the skin atrophy are observed.
Erosive pustular dermatosis of the scalp is a rare chronic disease with extensive pustular lesions, erosions, and crusting of the scalp, leading ultimately to scarring alopecia. Response of EPDS to therapy has been variable and different treatment regimens have been tried, including topical or systemic antibiotics, oral isotretinoin, zinc sulfate or aspartate, and dapsone. Topical potent corticosteroids have been reported to be the most effective therapy of this disease. However, there is a major risk of worsening atrophy of the treated skin after prolonged use.

Daily application of topical 0.1% tacrolimus ointment was started in both patients. This treatment resulted in a significant improvement of the first patient’s lesions within 2 weeks. Scarring and atrophic lesions were almost resolved after 6 months of topical tacrolimus use (Figure 3). Thereafter, the applications were progressively tapered to 3 times a week, without any recurrences after 1 year.

In the second patient, a prompt response to daily application of topical 0.1% tacrolimus ointment was observed after 2 days. To improve the atrophic condition of this sun-damaged scalp, daily application of topical 0.05% retinaldehyde cream (Ystheal) was added 2 weeks later, but after 1 week of use, the latter was stopped because of lesion relapse. Disappearance of the pustular and erosive lesions was observed within 1 month. After 8 months of therapy, a recovery of the skin atrophy was observed.

Erosive pustular dermatosis of the scalp is a rare condition, with approximately 40 cases reported. The disease is characterized by the development of sterile pustular lesions with a nonspecific inflammatory reaction of the scalp, which appears to be distinct from folliculitis decalvans, pyoderma gangrenosum, or cicatricial pemphigoid. Erosive pustular dermatosis of the scalp typically develops in long-standing atrophic sun-damaged skin changes. Local trauma to this atrophic skin acts as a triggering factor. Erosive pustular dermatosis of the scalp occurs rarely after the treatment of actinic keratoses or squamous cell carcinoma by x-ray radiation therapy, skin grafting, fluorouracil cream, or topical retinoin. Potent topical corticosteroids have been widely used in EPDS with a variable and inconstant response. Nevertheless, the prolonged use of potent topical corticosteroids on atrophic skin may be deleterious. Topical tacrolimus is a potent anti-inflammatory and immunosuppressive molecule, which has been shown to be effective in the management of chronic inflammatory skin diseases, such as atopic dermatitis. Topical tacrolimus is devoid of many side effects of topical corticosteroids such as epidermal atrophy, striae-distensae, or telangiectasia. However, there is concern about potential cutaneous carcinogenic effects associated with its long-term use on UV radiation–exposed skin, similar to those reported with systemic tacrolimus in transplant recipients.

Topical tacrolimus has been very effective in our 2 patients, with rapid control of the inflammatory process and an improvement of the atrophic condition. Our 2 observations strongly suggest that topical tacrolimus constitutes a novel therapeutic alternative for EPDS. However, neoplastic changes has been reported in EPDS. In the absence of data about a potential carcinogenic effect of this drug after long-term use on photodamaged skin, those patients should be carefully followed up, especially after tacrolimus treatment.

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REFERENCES


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