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Erosive Pustular Dermatosis of the Scalp

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Erosive pustular dermatosis of the scalp is a rare clinical entity with unclear etiology and pathogenesis. A very few cases have been described worldwide. We present a case of a 62-year-old woman with burning pustular lesions, erosions and crusts on scalp, following a seborrheic keratosis cryotherapy, dated 6 years ago. The persisting subjective symptoms and the conventional therapeutic refractivity classified the patient as having a psychosomatic disorder. Histology examination together with a thorough work-up resumed the diagnosis. Disulone was successfully introduced. The patient improved dramatically at the end of the first month and was advised to continue the therapeutic modality for 6 months.

Key words: erosive pustular dermatosis

Introduction

Erosive pustular dermatosis, also known as erosive pustulosis and erosive pustular dermatitis of the scalp is a chronic skin condition usually affecting the scalp, presented by pustules and eroded plaques, covered with crusts [8]. Most patients are elderly men and women that have suffered previous injury, skin cancer surgery or herpes zoster. A case of epidermal growth-factor receptor inhibitor – gefitinib – induced erosive pustular dermatosis is also anecdotally described [10]. The etiology of the disease remains obscure. Possibly, it is triggered by a minor injury, followed by impaired wound healing process [4]. Although inevitable by-stander, infection is not the primary cause, since the erosive lesions persist despite antibiotic therapy. Erosive pustular dermatosis usually starts with tiny pustules on the scalp, evolving into lakes of pus, erosions and erythematous patches, covered with yellowish crusts. Extensive disease often complicates with scarring and extensive balding [2].

Case report

A 62-year-old Caucasian female reported a 6-year history of pustular lesions on the scalp that have been extended to a different level, but persist chronically together with erosions (Fig. 1), pus lakes and areas of scarring alopecia. The lesions evolved abruptly upon cryotherapy of seborrheic keratosis of the right temporal zone. Various therapeutic modalities have been tried during the years including topical and systemic antibiotics, topical corticosteroids and calcineurin inhibitors. The patient claimed to have burning and tingling sensation, due to which she had been referred to as having artificial dermatitis (pathomimia). Histologic examination of a skin biopsy specimen revealed a thick parakeratotic crust on top, next to superficial acantholysis at the level of spinous keratinocytic layer, atrophic epidermis and a chronic inflammatory dermal infiltrate composed of many neutrophils, lymphocytes and a few tissue macrophages in the papillary dermis (Fig. 2). Fibrous and thickened reticular dermis was presented with a reduced number of hair follicles. Microbiological swabs and direct immunofluorescence microscopy were negative. Her routine blood examination results were within normal ranges. No evidence of monoclonal gammopathy, nutrition and vitamin deficits was detected. Superficial pyoderma gangrenosum was excluded due to the absence of an underlying systemic disease. The diagnosis of erosive pustular dermatosis of the scalp was made. Systemic therapy with



Fig. 1. Erythema-based erosion in the front part of the capillitium

disulone 25 mg daily was introduced in attempt to cease neutrophilic haemotaxis at the site of inflammation. At the follow-up visit one month later the pustular lesions were replaced by atrophic scars and minor zones of cicatricial alopecia. The subjective symptoms were resolved and the patient felt in good shape. Six month-continuation of therapy was recommended.

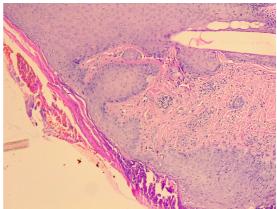


Fig. 2. Parakeratotic squamous crust, subcorneal acantolysis, neutrophil exocytosis with pronounced spongiosis of the epidermis, mixed inflammatory infiltrate with abundant polymorphonuclear cells in the papillar derma, interstitial and perivascular. Hematoxylin-eosin staining, \times 400

Discussion

Erosive pustular dermatosis of the sclap (EPDS) is a rare clinical entity. Probably, it is underestimated, since there are no more than 50 cases reported worldwide. The disease usually presents with sterile pustules, erosions and crusts, developing on sun-exposed areas of scalp after a minor injury. Most cases are reported to occur after the treatment of actinic keratoses or squamous cell carcinoma by X-ray radiation therapy, skin grafting, fluorouracil cream, or topical tretinoin [3, 6, 7, 9, 11]. To our knowledge, the first case of EPDS occurring after cryotherapy of seborrhoic keratosis of the scalp is herein reported.

The etiology and pathogenesis of the disease is unclear. In all cases, histology shows profound superficial or deep dermal neutrophilic infiltrate with a spectrum of epidermal changes from atrophy, erosions and ulcerations to hyperkeratosis and irregular acanthosis. In long-standing cases hair follicles are being reduced in number and replaced by fibrous tracts. Some authors speculated that EPDS might affect the legs of patients with chronic venous insufficiency thus raising the hypothesis that ineffective wound healing process provokes the inflammatory reaction [1]. We rather consider EPDS as an entity in the spectrum of auto-inflammatory disorders.

EPDS is a diagnosis of exclusion. The clinical presentation is more superficial than the one of folliculitis decalvans, more extended than the cicatricial pemphigoid and less severe than the pyoderma gangrenous [2, 4]. However, all those entities have to be ruled out via sophisticated broad-spectrum examinations, together with a thorough work-up for an underlying systemic disease. Negative microbiological skin cultures are always expected, however, not persistently presented due to secondary super-infections.

The most provocative EPDS issue is the therapeutic implications. It is a real treatment challenge since many different modalities are being tested but none of them showed to be beneficial enough. Response of EPDS has been variable with oral isotretinoin, zinc sulfate or aspartate, dapsone and topical calcineurin inhibitors [2, 5, 6, 7]. Although topical potent corticosteroids have been reported to be the most effective, the risk of atrophy on a prolonged use is crucial [3, 5]. Our patient proved to be refractory to topical corticosteroids and tacrolimus, therefore, our treatment choice was sulfones. Disappearance of the pustular and erosive lesions was observed within 1 month. A long follow-up is needed, however, to fully judge the therapeutic effectiveness.

EPDS is a rare and rather unknown clinical entity. Patients are often classified as having psychosomatic or obsessive-compulsive disorders. The proper recognition, verification and beneficial therapeutic approach are essential for restoring the physical and emotional integrity as well as the quality of life of the affected persons.

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