Erosive pustular dermatosis of the scalp: a pathogenetic mystery and therapeutic challenge

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Abstract

Erosive pustular dermatosis of the scalp (EPD) is a rare condition that affects predominantly the adult population and occurs on a previously photo-damaged bald scalp. The physical examination is presented with large erythematous, erosive and crusted patches with granulation on an atrophic skin. The problem in patients with erosive pustular dermatosis of the scalp arises from the non-specific clinical and histopathological findings, which can be misleading. Biopsy followed by careful histopathological verification is mandatory, although the finding is nonspecific. The histopathology findings are characterized by superficial erosions with mild neutrophil infiltrate, mainly intravascular and focally with neutrophil exocytosis; focal parakeratosis, smoothed rete ridges without pronounced interface changes; pronounced lymphoplasmacytic infiltrate with focal distribution in the dermis and giant cell reaction with the formation of a "foreign body" granuloma.. We report a 58-year-old male patient with a 1-year-old lesion, suspected for skin cancer, later diagnosed with EPDS, which was successfully treated with topical clobetasol proprionate after 3-5weeks.

Introduction

Erosive pustular dermatosis of the scalp is a disease of unclear entity that predominantly affects the non-hairy area of the scalp in adults.[1] The pathogenetic mechanisms underlying this rare dermatosis remain unclear.[2]

The histopathological findings of the disease could be of mixed type and is often confusing,[2] but could include: i) epidermal ulceration, epidermal atrophy, ii) sterile pustules with variable localization, iii) reduced to completely absent hair follicles, as well as iv) nonspecific mixed inflammatory infiltrate.[3,4]

According to other authors, the histopathological findings could be even more extensive or non-specific, such as: i) perifollicular granulomas, including remnants of hair follicles and giant multinucleated cells, ii) intrafollicular spongiform pustules, iii) neutrophilic microabscesses in the epidermis, iv) granulation tissue, v) aggregates of macrophages and foreign body type giant cells, vi) diffuse dermal fibrosis, vii) dermal inflammatory infiltrate of lymphocytes and neutrophils.[1]

The problems in the treatment of this dermatosis arise, secondly, from the non-specific clinical picture, which makes clinicians quickly think in the direction of actinic keratoses and decide to start a radically different therapy. Thus, the histology itself subsequently becomes even more difficult to interpret.

Case report

A 58-year-old man presented a 1-year-old lesion on a previously photo-damaged bald scalp suspected for skin cancer. The physical examination showed large erythematous, erosive and crusted patches with granulation on an atrophic skin (Figure 1A,B). The biopsy results showed changes in the epidermis according to the vesicular-bullous model with basal/suprabasal epidermolysis without the presence of acantholysis, superficial erosions with mild neutrophil infiltrate, mainly intravascular and focally with neutrophil exocytosis. Focal parakeratosis, smoothed rete ridges without pronounced interface changes. Pronounced lymphoplasmocytic infiltrate with focal distribution in the dermis, giant cell reaction with the formation of a "foreign body" granuloma.

Immunohistochemistry: cytokeratin – negative, CD 138 – positive for plasma cell composition, CD 68 – marked the heterogeneous macrophage cell composition, CD 20 marked small lymphocytes.

The diagnosis of erosive pustular dermatosis of the scalp was made.

The patient started topical treatment with clobetasol proprionate 0.5 mg/g cream twice daily with favorable response after 4 weeks (Figure 2A,B).

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Discussion and conclusions

The provoking or triggering factors for erosive pustular dermatosis are different, often confusing for the clinician and include in turn: certain drug triggers, namely – after or within panitumumab therapy,[5] after local oxygen therapy,[6] after photodynamic aminolevulinic acid therapy,[7] treatment with gefitinib,[8] nivolumab,[9] sirolimus,[10] topical imiquimod,[11] ingenol mebutate,[12] and latanoprost.[13]

The triggering factors could also be of a mechanical or infectious nature such as trichotillomania *e.g.*,[14] past herpes zoster

infection,[15] post-traumatic,[16] after CO-2 laser resurfacing, after surgical interventions, or after hair transplantation.[17] Determining the importance of the above-mentioned factors in relation to the pathogenesis of the disease remains controversial as it cannot always be clearly/definitely demarcated whether it is a sporadic association between two diseases or a pathogenetic relationship.

The problem with the patients with erosive pustular dermatosis of the scalp is coming from the fact that the clinical manifestation of the disease mainly affects the non-hairy areas of the scalp and could hardly be differentiated clinically from a hypertrophic form of actinic keratosis or non-melanocytic skin tumors.[2] Besides the possible parallel manifestation of the two mentioned diseases, the transition of erosive pustular dermatosis to non-melanocytic skin tumors such as basal cell and squamous cell carcinomas has also been described.[18,19]

In practice, erosive pustular dermatosis (EPD) of the scalp could also be perceived as a precancerous condition. Repeat biopsies in this category of patients (with erosive pustular dermatosis of the scalp) are of significant prognostic importance.[2] Other important differential diagnoses that should not be neglected are: brunsting perry type of bullous membrane pemphigoid, bacterial or fungal infection.[20]

Scalp EPD therapy also remains a product of the individual approach of the clinician and has undergone some evolution in recent years.

The therapeutic options described in the world literature are variable and include i) topical zink oxide,[21] ii) the systemic application of sulfasalazine in combination with 308 nm monochromatic excimer light,[22] iii) oral application of retinoid and a bi-layered skin substitute,[23] local application of novel silicone gel,[24] local treatment with 0.1% mometasone furoate cream,[25] systemic therapy with oral prednisone and topical tacrolimus,[26] local monotherapy with tacrolimus,[27] systemic dapsone therapy,[28] systemic therapy with acitretin,[29] photodynamic therapy,[30] isotretinoin systemically,[31] local therapy with calcipotriol cream,[32] and oral zinc sulphate.[33]

First-line therapy remains the local application of potent corticosteroids and topical calcineurin inhibitors.[2] Systemic administration of corticosteroids and topical calcipotriol are considered as second-line therapy.[2] A third line is reserved for systemic retinoids and topical dapsone in the form of a 5% gel, for example.[2]

The opinion regarding the application of photodynamic therapy with aminolevulinic acid, as well as the surgical approach, should be considered with particular attention as possible therapeutic options, since there are conflicting clinical observations in the literature that describe these two options as more of a triggering/inducing factor.[8,34-38]

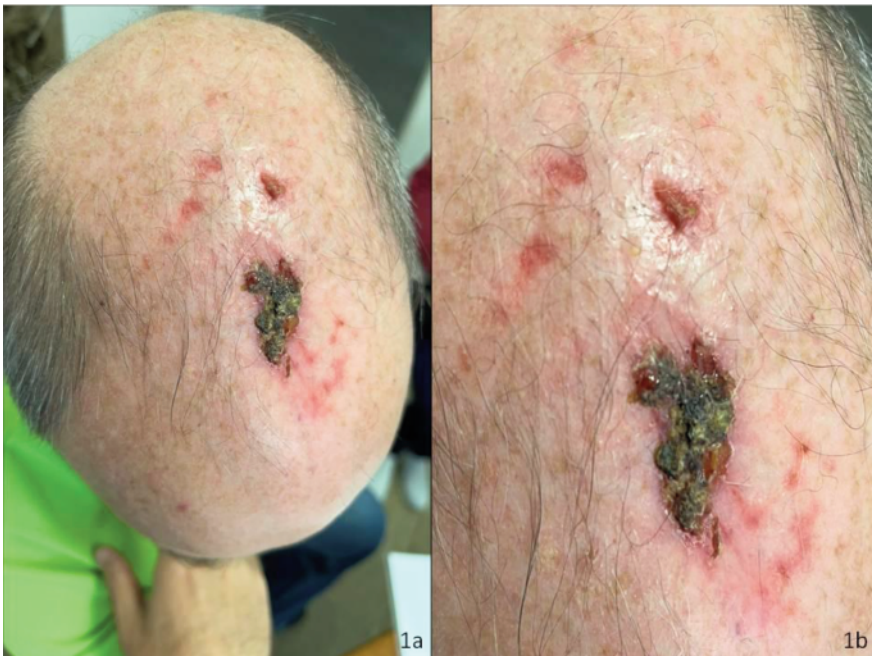

Figure 1. A,B) Clinical picture of a histopathologically proven form of erosive pustular dermatosis of the scalp with large erythematous, erosive and crusted patches with granulation on an atrophic skin with androgenetic alopecia.

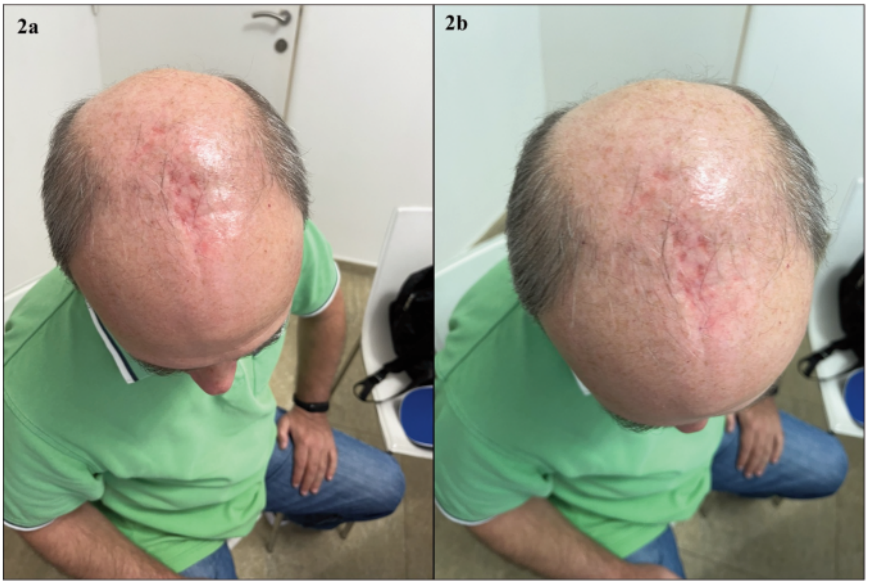

Figure 2. A,B) Improvement of the patient after starting therapy with topical clobetasol proprionate 0.5mg/g after 3-5 weeks. Residual erythema, with slight scarring can be seen.

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