“Persistent Scabs: A Case of Eruptive Pustular Dermatosis”

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Introduction
Erosive pustular dermatosis (EPD) is a superficial skin disorder usually arising on the scalp of the elderly. It was first reported in Britain in 1977.1 It lacks specific clinical and histopathological findings. It is characterized as chronic intermittent crusted papules or plaques, pustules and erosions developed in local traumatized skin. High potency topical steroids have the most success in curing the condition. At some point during the clinical course, a scarring alopecia develops that can involve adjacent areas. EPD of the scalp is diagnosis of exclusion after neoplastic, infectious or other inflammatory conditions are ruled out. The predisposing factors include age and a history of various types of local skin trauma, thus, the incidence of EPD of the scalp is most likely underestimated in American literature.2

Case Presentation
A 92 year old Caucasian female presented to a dermatology office with the complaint of intermittent dry areas and pimples on the top of her head for over a year. The patient had a history of androgenic alopecia affecting the vertex and apical portions of the scalp. Upon initial presentation she had several yellow crusted papules and pustules located in the thinning portion of her apex (Figure 1). A presumptive diagnosis of folliculitis and impetigo was made and she was placed on topical clindamycin and mupirocin twice a day. Her lesions did not improve and she returned in two months for reevaluation. Upon examination she had several rough scaly papules on an erythematous base, a few erosions and a dark crusted hyperkeratotic plaque (Figure 2). She was thought to have actinic keratosis and treated with cryotherapy. A biopsy was done on the larger plaque to rule out neoplastic behavior. The histopathology revealed a mixed inflammatory infiltrate without any atypia (Figures 5-8). A diagnosis of EPD was considered and the patient was placed on Clotetasol 0.05% ointment. The patient showed clinical improvement, although throughout the following year she continued to have flares. Over time she developed fibrosis of her scalp (Figures 3, 4).

Histology
- Recognizing characteristic histological patterns with microscopy with staining or immunofluorescence assist in ruling out other possible diagnoses.2
- Histological findings of erosive pustular dermatosis are non-specific with parakeratosis, hyperkeratosis, and inflammatory cell infiltration.
- There is epidermal atrophy with loss of sebaceous glands.
- In long standing lesions there is also a loss of hair follicles and sebaceous glands with dermal fibrosis associated with resulting scarring alopecia.4

Discussion
Symptoms & Clinical Course: Lesions appear as superficial yellow-brown crusts with underlying erythema. The borders are irregular and may have smaller areas of preserved skin. The number of lesions and surface area it covers varies from case to case.1 Primary lesions are usually sterile, but samples from the area may be positive for secondary bacterial or mycological colonization. Lesions are generally asymptomatic but may be associated with pain or pruriens.2 There is often a scarring alopecia with destruction of the hair follicles and follicular stem cells due to chronic inflammation. Due to the chronic nature of EPD, a clinical suspicion of neoplasia formation should be considered during future evaluations of the area. Populations at Risk: EPD is typically seen in elderly patients, with women more commonly diagnosed than men.3 Ultradamage along with other etiologies and modalities causing skin injury are predisposing factors. There is also association in patients with autoimmune disorders such as Hashimoto’s thyroiditis, autoimmune hepatitis, rheumatoid arthritis, myelodysplastic syndrome, and myasthenia gravis.6,8 Pathogenesis: EPD is believed to be due to local skin trauma.9 The pathogenesis of the disorder is poorly understood. Published cases include patients with a history of sun exposure, CO2 laser therapy, topical ingenol mebutate, topical tretinoin, hair transplantation, surgery for cochlear implants, skin grafts, cryotherapy, radiation, contact dermatitis from a prosthetic hair piece, and post-herpes zoster.10,19

Immunosenescence, an age-associated decline in the immune system, is a hypothesized mechanism that leads to an abnormal immune response to wound healing.20 An additional association with autoimmune disorders is supported with a link to neutrophil-stimulating cytokines and chemokines.5,8

Diagnosis: The differential diagnosis for EPD includes squamous cell carcinoma, basal cell carcinoma, localized cicatricial pemphigoid, chronic discoid lupus erythematosus, folliculitis decalvans, ulcerative dermatitis and bacterial or fungal infections.2 EPD is a diagnosis of exclusion. Treatment: EPD is treated with the most success with high potency topical steroids. Other pharmaceutical treatments include topical tacrolimus, isotretinoin, aricetin, calcipotriol, and diprose.21 Even though EPD could result from photodynamic therapy, there has been success with its use.22 Surgical excision of the lesions is another treatment option; however, it may cause recurrence due to its traumatic nature.23 Scarring alopecia is usually a permanent consequence of the disorder no matter the treatment modality.

Conclusion
EPD presents as superficial crusts and ulcerations in epithelial areas with a history of local trauma typically in elderly females. Because of these non-specific symptoms, diagnosis of EPD is delayed and a diagnosis of exclusion. EPD resolves with a topical steroid regime and heals with scarring alopecia in the affected area.

References