Dermatology Practical & Conceptual

Erosive Pustular Dermatosis of the Scalp Mimicking Squamous Cell Carcinoma: Can Dermoscopy Be Helpful?

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ABSTRACT

Introduction: Erosive pustular dermatosis of the scalp (EPDS) presents as a rare and challenging skin disorder marked by erosions, pustules, and crusted lesions on the scalp. Diagnosing EPDS is complex, often mimicking other dermatological conditions like actinic keratosis (AK), basal cell carcinoma (BCC), or squamous cell carcinoma (SCC). Therapeutic challenges arise from EPDS's chronic, relapsing nature, which requires long-term management.

Objective: This study aimed to identify and compare dermoscopic features of EPDS mimicking SCC of the scalp to improve diagnostic accuracy and support non-invasive differentiation.

Methods: A retrospective descriptive study, conducted in two Italian dermatological centers from 2017 to 2023, included 43 patients initially diagnosed with SCC of the scalp that was afterwards confirmed as EPDS through either histology or clinical evaluation and follow-up. Clinical and dermoscopic criteria were evaluated. A comparison of dermoscopical criteria with the same number of confirmed SCC of the scalp was performed.

Results: A total of 43 patients were included, predominantly male (42:1). Androgenetic alopecia was present in 65% of cases, predominantly in the parietal area (44%). Hairpin and dotted vessels, along with white and combined lesion colors, were strongly associated with SCC, while polymorphic and branched vessels, orange tones, and targetoid hair follicles predominated in EPDS.

Conclusions: EPDS poses diagnostic and therapeutic challenges due to its unique features and elusive etiology. Distinguishing SCC from EPDS is crucial to avoid unnecessary treatments; dermoscopy can serve as an instrument in this process.

Introduction

Erosive pustular dermatosis of the scalp (EPDS) is a rare and challenging skin disorder that manifests clinically in the form of erosions, pustules, and crusted lesions on the scalp [1]. It predominantly affects bald areas in older individuals, often in areas subjected to prior trauma or treatment. This condition is distinguished by its unique clinical presentation, enigmatic etiology, and the substantial impact it has on affected individuals [2]. The hallmark of EPDS is the presence of persistent, painful, ulcerative lesions on the scalp, typically occurring in elderly individuals [3]. These erosions are prone to pustule formation and often resist conventional treatment measures [2]. As a result, patients often experience a significant reduction in their quality of life due to discomfort, pain, and cosmetic concerns [4]. The exact cause of EPDS remains uncertain. However, various factors are believed to contribute, including a history of trauma, chronic irritation, and a potential link to sun damage. Moreover, immunological mechanisms might play a role in the pathogenesis of this condition [1]. One common observation in cases of EPDS is the often-reported occurrence of mechanical or chemical scalp trauma preceding its manifestation [5,6,7]. Some researchers have postulated that the development of this condition might be triggered by an autoimmune reaction to hair follicles induced by trauma, resulting in persistent inflammation and scarring. This theory gains support from the frequent co-occurrence of EPDS with other autoimmune conditions [1]. Diagnosing EPDS can be a complex process, as it often mimics other dermatological conditions like actinic keratosis (AK), basal cell carcinoma (BCC), or squamous cell carcinoma (SCC) [8]. The scalp area is also a body site with a particular predisposition to skin malignancies due to exposure to UV radiation [9]. Differential diagnosis, coupled with a thorough examination and, in some cases, skin biopsy, is essential to confirm the condition [8]. In histopathology, EPDS is characterized by atrophic epidermis and chronic inflammation marked by the presence of lymphocytes, neutrophils, and occasionally, foreign body giant cells [10]. The clinical characteristics and diagnostic criteria can help in distinguishing EPDS from other conditions. Recent advancements in diagnostic techniques, such as reflectance confocal microscopy and dermoscopy, have also been shown to improve the accuracy of skin tumor diagnosis [7, 8], but no specific criteria for EPDS have been described. Even if recent studies have understated the importance of trichoscopy and dermoscopy of EPDS [3, 9], histological examination is usually essential to distinguishing it from SCC [4,5,9]. Prompt and accurate diagnosis is crucial to prevent inappropriate treatments, especially since biopsy findings in EPDS can vary depending on the lesion stage and are often nonspecific [3]. A deep, representative sample is recommended over shave biopsies, as EPDS may involve mid- and deep-dermal layers, and its inflammatory infiltrate, which often includes plasma cells, can mimic malignancy, necessitating careful exclusion of SCC or infiltrative BCC [10]. After a diagnosis is established, managing EPDS is also challenging. Therapies often involve a combination of topical antibiotics, steroids, wound dressings, and sometimes surgical procedures [4]. Since EPDS is a chronic, relapsing condition, long-term management and monitoring are essential to provide relief to affected individuals. Despite the difficulties in treatment, most patients have experienced improvement and even remission with the right combination of therapies.

Objectives

In this study, we aimed to analyze and compare the dermoscopical characteristics of EPDS and SCC of the scalp using visual data from patient images, looking to identify distinguishing features between these conditions.

Methods

Study Design and Setting

We conducted a retrospective descriptive study based on chart and image reviews from two distinguished dermatological

centers in Italy, covering cases between 2017 and 2023. Patients included in the study were initially diagnosed with either SCC or EPDS of the scalp. Data extracted from our databases included sex, age, initial diagnosis, anatomical area of the scalp, histological diagnosis, and the treatment regimen that was followed. All patients had undergone incisional biopsy and were evaluated by an experienced dermatopathologist to exclude the presence of a squamous cell carcinoma. The study population comprised patients with histologically-confirmed EPDS and patients with non-specific inflammation on histology. However, the latter category of patients had to demonstrate significant improvement following a course of corticosteroid therapy to be included in the study, thus supporting an EPDS diagnosis. Clinical and dermoscopic images were obtained for all patients. Two independent, blinded dermatologists evaluated the images for dermoscopic criteria potentially indicative of SCC, including ulceration, targetoid hair follicles, predominant vascular pattern (e.g., hairpin, glomerular, linear-irregular, or polymorphic vessels), presence of pigment, presence of scales/keratin, and the predominant background color of the lesion (white, yellow, red, orange/salmon, or combination). To enhance comparative analysis, a separate dataset of 43 dermoscopic images from patients with confirmed SCC of the scalp were reviewed under the same criteria. This control group was used to identify potentially distinguishing dermoscopic features between SCC and EPDS.

Statistical Analysis

Descriptive statistics were used to summarize baseline demographic and clinical characteristics. To assess differences between the EPDS and SCC groups, Chi-squared tests or Fisher's exact tests were applied for categorical variables and t-tests (or Mann-Whitney U tests if non-normally distributed) were used for continuous variables. A logistic regression model was performed to analyze the predictive power of dermoscopic features in distinguishing between EPDS and SCC. The following features were included: vessel types (absent, hairpin, glomerular, linear irregular, polymorphic, branched, dotted), lesion colors (white, yellow, red, orange, combined), ulceration, scales/keratin, pigmentation, and targetoid hair follicles. To address the issue of perfect separation and multicollinearity among features, Ridge regularization (L2 penalty) was applied to the logistic regression model. All predictors were standardized prior to model fitting to ensure comparability. All analyses were performed using R Studio 4.4.1 (2024-06-14), with statistical significance set at $P<0.05$

Results

The study included 43 patients that had initially received a diagnosis of SCC and had performed a biopsy that resulted suggestive of EPDS or nonspecific. Mean age was 77 years, and the predominant sex was male (42:1). In 12 cases, histology displayed only inflammatory aspects, with no indication of SCC, and the diagnosis of EPDS was therefore established based on the exclusion of SCC and improvement with topical steroid therapy, along with a clinical diagnosis rendered by two independent dermatologists. Contributing factors included pronounced androgenetic alopecia in 28 patients, with 16 of them exhibiting signs of actinic damage. None of the patients exhibited concurrent skin or mucosal disorders. The parietal area was the most frequently affected region (44%), followed by the frontal (30%) and vertex areas (26%) (Figure 1). All patients displayed signs of scarring alopecia, serum-hematic crusts, erosions, loss of follicular ostia, and visualization of hair bulbs through a thinned skin. Dermoscopic evaluation is presented in Table 1. Dermoscopic images of EPDS cases are displayed in Figure 2 and SCC cases in Figure 3. The Ridge logistic regression model achieved a classification accuracy of 97.6% on the training set and 96.7% on the validation set. The confusion matrix for the validation set showed 14 true negatives (EPDS correctly classified), 11 true positives (SCC correctly classified), and one false positive (EPDS misclassified as SCC). The most influential features for distinguishing SCC from EPDS included hairpin and dotted vessels, associated with SCC (positive coefficients) and polymorphic and branched vessels and orange tones, associated with EPDS (negative coefficients). The validation model demonstrated robust generalizability, achieving a mean accuracy of 75.3% (SD ±17.99%) across 5-fold cross-validation.

Conclusions

Dermoscopy plays an important role in differentiating EPDS from SCC, despite shared features such as erosions and crust formation. SCC criteria in our study were aligned with prior studies [14]–strongly associated with hairpin and dotted vessels–while in EPDS, polymorphic and branched vessels were predominant. In terms of color, SCC was significantly linked to white tones, whereas EPDS predominantly exhibited orange or salmon undertones, which were absent in SCC. Combined colors (white, yellow, or red) showed a trend toward SCC but without statistical significance. Targetoid structures and white circles, traditionally linked to SCC [15,16], were unexpectedly more common in EPDS ($P=0.0497$). These structures, previously considered a key indicator for distinguishing between in situ and invasive SCC, did not aid in differentiating between EPDS and SCC in our dataset. This might be explained by the fact that, as tertiary centers, most cases that reach our attention are more advanced and poorly differentiated SCC, which are not expected to display

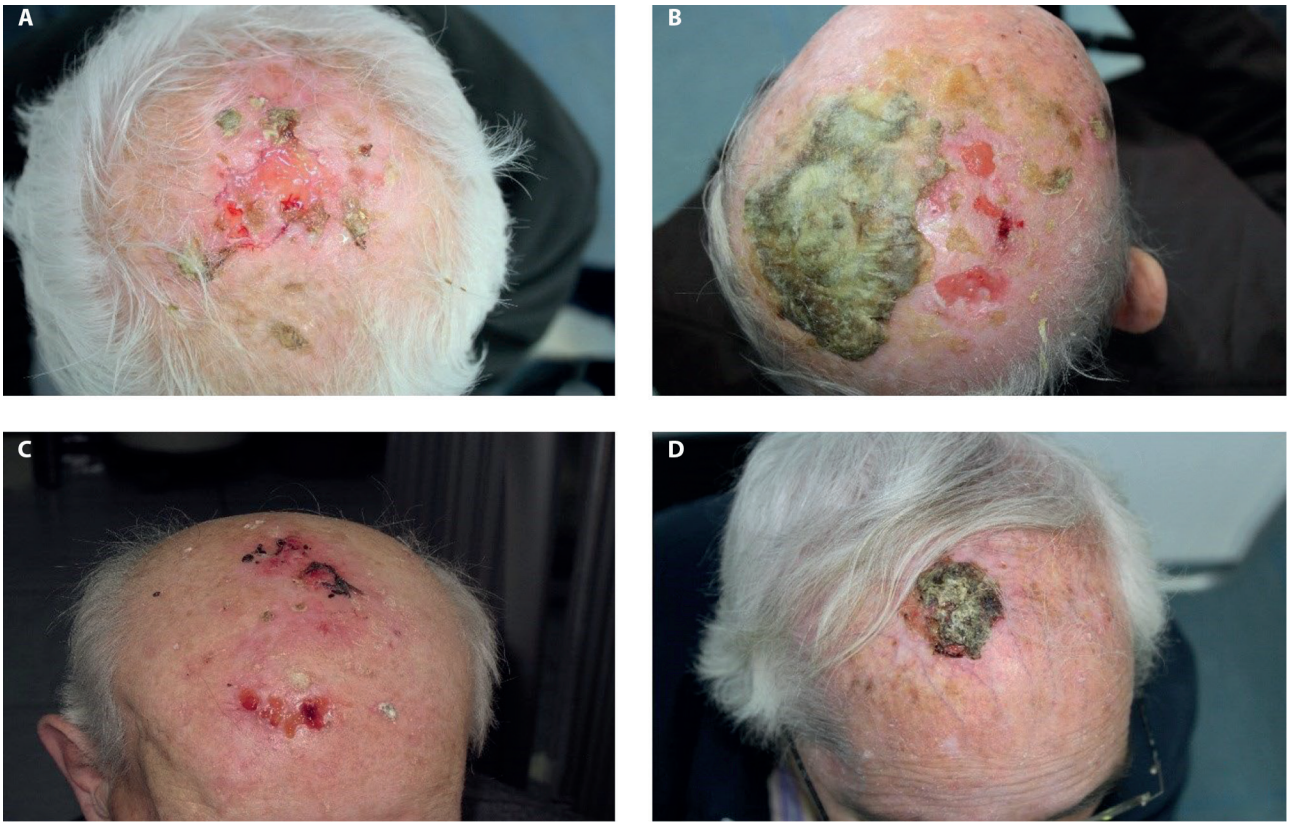

Figure 1. Clinical images of erosive pustular dermatosis of the scalp (EPDS) in four distinct patients. A-C are all males over 75 years old presenting erosions and crusted lesions on the scalp and signs of actinic damage. [D] An 82- year-old man presenting an eroded nodule in the frontal area, clinically mimicking a squamous cell carcinoma.

Table 1. Dermoscopic Evaluation of EPDS Mimicking SCC of the Scalp and Cases of Confirmed SCC of the Scalp.

Evaluated criteria	EPDS (n = 43)	SCC (n = 43)	p-value
Vascular pattern:			
• Absent	7 (16.3%)	0	**0.019**
• Hairpin	0 (0.0%)	17 (40.5%)	**<0.01**
• Glomerular	3 (7.0%)	2 (4.8%)	1.0
• Linear irregular	5 (11.6%)	0 (0.0%)	0.06
• Polymorphic	21 (48.8%)	8 (19%)	**<0.01**
• Branched	7 (16.3%)	0 (0.0%)	**0.019**
• Dotted	0 (0.0%)	15 (35.7%)	**<0.01**
Predominant color:			
• White	0 (0.0%)	13 (30.2%)	**<0.01**
• White-yellow	6 (14.0%)	10 (23.3%)	0.40
• Red	6 (14.0%)	3 (7.0%)	0.48
• Orange/salmon	22 (51.2%)	0 (0.0%)	**<0.01**0.1
• Combined	9 (20.9%)	17 (39.5%)	
Ulceration	43 (100%)	38 (90.5%)	0.11
Keratin/scales	39 (90.7%)	37 (88.1%)	0.97
Pigmentation	2 (4.7%)	2 (4.7%)	1.0
Targetoid hair follicles/white circles	20 (46.5%)	10 (23.8%)	**0.04**

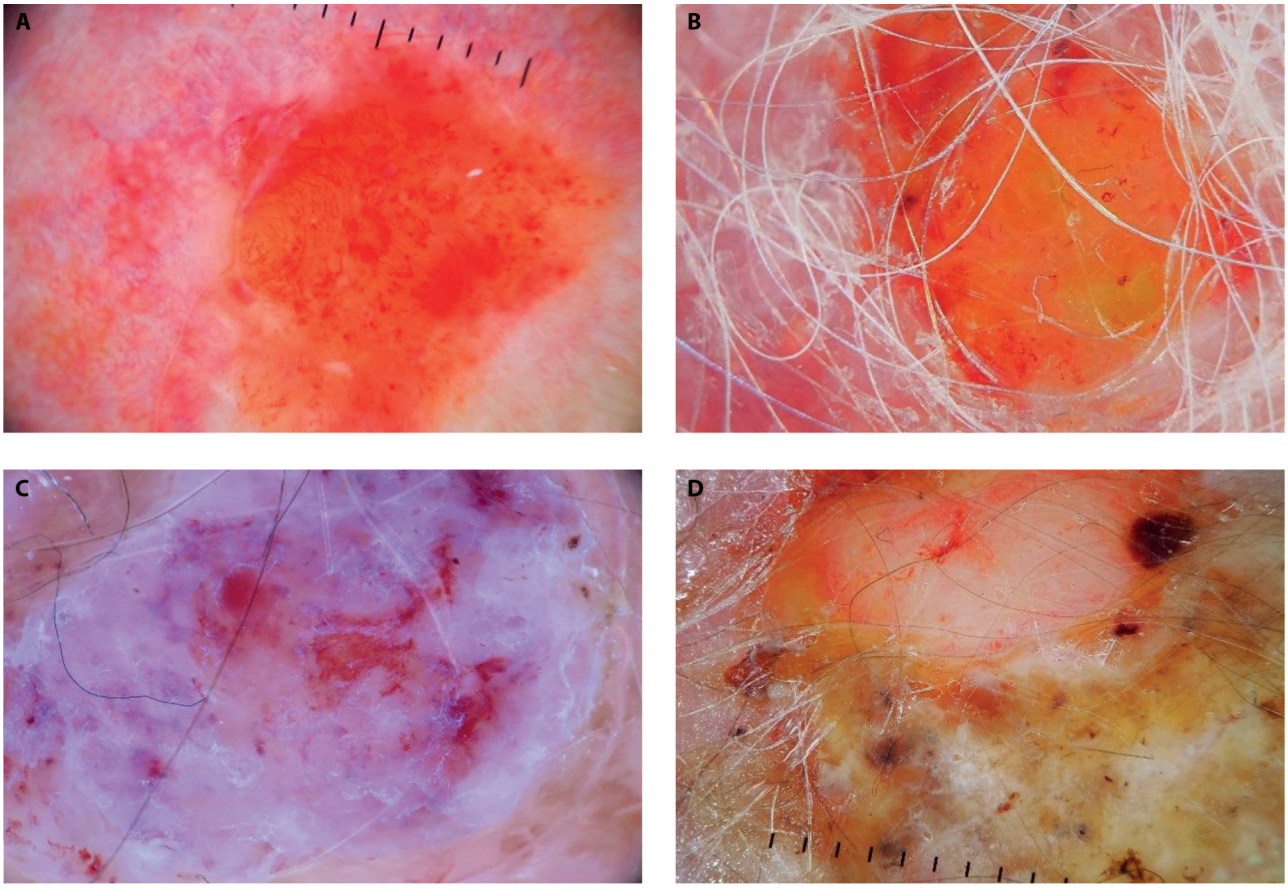

Figure 2. (A, B) Dermoscopy showing four different patients with similar patterns of orange background, absence of follicles, polymorphic vessels, erosion, and targetoid white peripheric structures. (C) Whitish eroded nodule with polymorphic vessels. (D) Pinkish nodule with polymorphic vessels and brown scattered pigment.

these structures. Certain dermoscopic features, such as ulceration and scales/keratin, were equally prevalent in both conditions and thus non-discriminatory. Pigment was rare in both groups, further limiting its diagnostic utility. While trichoscopic features of EPDS, such as absent follicular ostia and perifollicular pigmentation, enlarged dermal vessels, perifollicular serous or brown–gray hyperpigmentation, *pili torti,* and broken hair shaft, tufted hairs or black crusts or yellow thick exudate, have been well documented [1,12], other overlapping criteria with SCC can complicate diagnosis, underscoring the need for histological confirmation. Dermoscopy is typically known to be a valuable tool for diagnosing AKs. However, the presence of widespread scaling and crusting can be a perplexing factor found in both multiple AKs and EPDS. Furthermore, it is important to note that EPDS and multiple AKs can co-occur in the same patient, as both conditions share a common underlying factor of actinic damage [13]. Our study population's mean age of 77 years is higher than that in previous reports [3], likely reflecting a selection bias, as patients were referred to tertiary centers for suspected SCC, which is more common in older individuals. Interestingly, while EPDS is reported to primarily affect females, with a 2:1 female-to-male ratio [10,11], our cohort was predominantly male. This could be attributed to androgenetic alopecia and sun exposure, factors that predispose individuals to skin thinning, hinder wound repair, and promote persistent erosions and ulcers, potentially triggering autoimmune responses, as noted in prior research [1]. One limitation of this study is the use of punch biopsy, that might not be enough for a clear histological evaluation. Considering that both EPDS and SCC tend to be extensive, and most affected patients are elderly, performing an excisional biopsy is not feasible, and partial biopsies are our only way to try to achieve histological diagnosis in a less invasive manner. However, long-term follow-up to clinical treatment is mandatory in all cases for a definitive diagnosis. Clinicians must remain vigilant about the potential causes and associations of EPDS. Topical treatments (e.g., ingenol mebutate, 5-fluorouracil, imiquimod), procedures (cryotherapy, CO_2 laser, photodynamic therapy), and systemic diseases have been

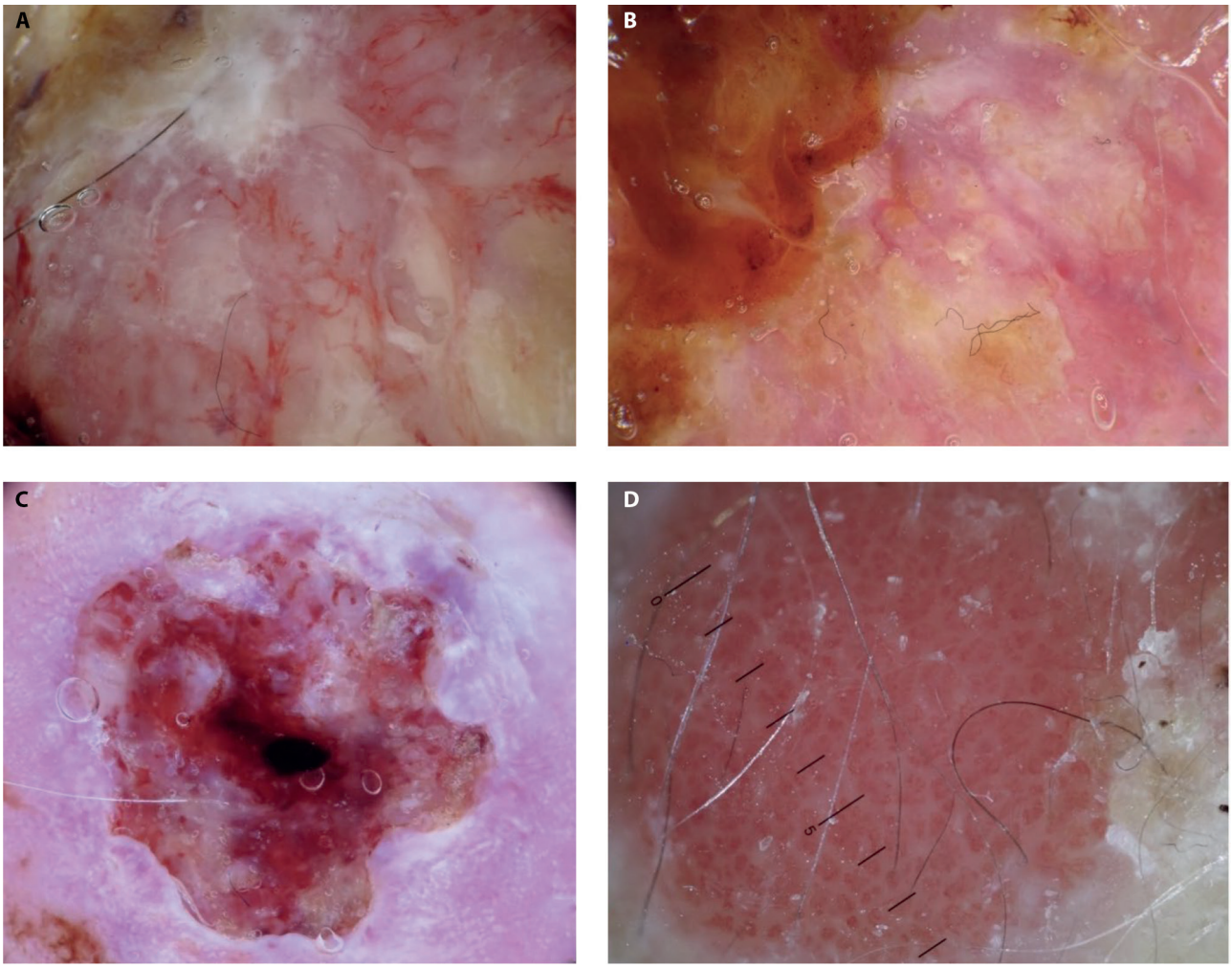

Figure 3. Dermoscopy showing four different patients with SCC of the scalp, with the presence of erosions and keratin and predominant patterns of combined colors, mostly red, yellow, and white, polymorphic vessels (A, B), dotted (C), and glomerular vessels (D).

linked to EPDS [3]. Many of these interventions, commonly used for Aks —a known SCC precursor—can also predispose to EPDS. This shared actinic damage explains why AKs and EPDS often co-occur, with scaling and crusting further complicating dermoscopic differentiation [13]. Treatment of EPDS includes high-potency topical corticosteroids, tacrolimus, oral steroids, and dapsone [10]. Early initiation of therapy is critical to mitigate symptoms and avoid invasive procedures. However, despite the efficacy of these treatments, relapse is common after discontinuation of corticosteroids or tacrolimus, emphasizing the importance of timely diagnosis and sustained management. In this study, some features demonstrated good performance in distinguishing between EPDS and SCC. The Ridge logistic regression model effectively leveraged a few characteristics: Hairpin and dotted vessels, along with white and combined lesion colors, were strongly associated with SCC, while polymorphic and branched vessels, orange tones, and targetoid hair follicles were more evident in EPDS. Additional features, such as ulceration and keratin, provided incremental diagnostic value, though their overlap between conditions occasionally contributed to misclassification. These findings highlight the potential of dermoscopy as a non-invasive diagnostic tool in clinical practice, aiding in the differentiation of EPDS from SCC. This approach could minimize reliance on biopsies only while providing interpretable insights into the relative importance of dermoscopic features. As seen in Figures 2 and 3, these conditions might have extremely overlapping criteria, and biopsies are still essential, but some features might aid not only in thinking about the condition but also in better organizing resources. Future research should focus on validating this model across larger samples and refining feature representations to further enhance its clinical applicability. The study's retrospective design and referral bias, as patients were selected from tertiary and dermato-oncology centers, limit generalizability. Further prospective studies are warranted to validate our findings.

EPDS is a complex, rare dermatological disorder that poses diagnostic and therapeutic challenges. The unique clinical features, non-conclusive dermoscopy, elusive etiology,

and profound impact on the quality of life of affected individuals make it a subject of interest and concern within the field of dermatology. The presence of orange colors can be indicative; also, it is important not to automatically associate the presence of white circles/targetoid hair follicles with the diagnosis of SCC, as they can also be present in EPDS. Accurately diagnosing SCC and EPDS is essential to avoiding unnecessary treatments and providing better patient care. Continued research efforts are needed to shed light on the underlying causes and to develop more effective management strategies.

Statement: The patients in this manuscript have given written informed consent to the publication of their case details.

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