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Case Report

Dermoscopic features of pediatric epidermodysplasia verruciformis in dark skin: a case report with clinicopathological correlation

Kawtar El Fid*, Hanane BayBay, Imane Denni, Ikram Bejja, Saadia Boughaleb, Zakia Douhi, Meryem Soughi, Sara Elloudi, Fatima zahra Mernissi

Department of Dermatology, Hassan II University Hospital, Faculty of Medicine, Pharmacy and Dentistry, Sidi Mohamed Ben Abdellah University, Fez, Morocco

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***Correspondence:**
Dr. Kawtar El Fid,
E-mail: kawtar.elfid2@gmail.com

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ABSTRACT

Epidermodysplasia verruciformis (EV) is a rare inherited genodermatosis associated with increased susceptibility to β-human papillomaviruses and an increased risk of cutaneous malignancy. We report the case of an 8-year-old child with Fitzpatrick skin phototype VI presenting with multiple hypopigmented verrucous lesions involving the face and upper trunk. Dermoscopy revealed well-demarcated structureless hypopigmented areas, fine white scales, and peripheral leukotrichic vellus hairs. Histopathological examination confirmed the diagnosis. The patient showed marked clinical improvement following topical adapalene. This case expands the limited literature on the dermoscopic features of EV in dark skin and highlights the diagnostic value of dermoscopy as a non-invasive tool in pediatric patients.

Keywords: Case report, Skin of color, Dermoscopy, Epidermodysplasia verruciformis, Pediatric

INTRODUCTION

Epidermodysplasia verruciformis (EV) is a rare inherited genodermatosis characterized by increased susceptibility to β-human papillomaviruses, resulting in persistent pityriasis versicolor-like macules and flat wart-like lesions from early childhood, with an increased risk of cutaneous squamous cell carcinoma.[1] Mutations in TMC6 (EVER1) and TMC8 (EVER2), and subsequently CIB1 deficiency, have established the importance of keratinocyte-intrinsic antiviral defense in the disease.[2,3] This susceptibility helps explain why otherwise healthy individuals may develop extensive and persistent cutaneous lesions.

Early recognition is important because the clinical course extends beyond childhood and may include malignant transformation later in life.[4]

However, hypopigmented, finely scaly lesions can resemble common pediatric dermatoses, delaying diagnostic consideration of EV. Although the diagnosis is mainly based on clinical and histopathological findings, dermoscopy can help identify subtle surface and pigmentary changes and guide clinicopathological assessment.[5,6]

The dermoscopic features of EV remain poorly characterized, particularly in patients with dark skin.7,8 Published observations suggest that the visibility of erythema, vessels, and hypopigmented structures may vary with the background pigmentation.

We report a pediatric case of EV in a Fitzpatrick phototype VI child, highlighting its clinicodermoscopic and histopathological correlation.

CASE REPORT

An 8-year-old child with no significant past medical history presented with a progressive eruption of asymptomatic cutaneous lesions that had been evolving since early childhood. There was no family history of similar lesions or evidence of immunosuppression.

Dermatological examination revealed multiple well-defined hypopigmented macules and papules with a verrucous, finely scaly surface involving the forehead, cheeks, and upper trunk in a child with Fitzpatrick skin phototype VI. Marked cutaneous xerosis was noted, whereas the mucosae and nails unaffected (Figure 1). Dermoscopy demonstrated well-demarcated structureless hypopigmented areas on a dark background with interruption of the facial pseudopigment network, associated with fine white scales and peripheral leukotrichic vellus hairs, without detectable vascular structures (Figure 2).

Histopathological examination showed hyperkeratosis, mild acanthosis, and enlarged keratinocytes with pale grayish, glassy cytoplasm and occasional perinuclear halos. The nuclei showed mild atypia, and the dermis exhibited a mild predominantly lymphocytic inflammatory infiltrate, confirming the diagnosis of EV (Figure 3).

The main differential diagnoses included pityriasis versicolor, verruca plana, and epidermal nevus. However, the early onset, facial distribution, verrucous morphology, characteristic dermoscopic findings, and histopathological features supported the diagnosis of EV.

The patient was treated with topical adapalene applied once daily. After three months of treatment, marked clinical improvement was observed, with a significant reduction in lesion thickness and scaling. The improvement was maintained at the six-month follow-up, with no treatment-related adverse events (Figure 4).

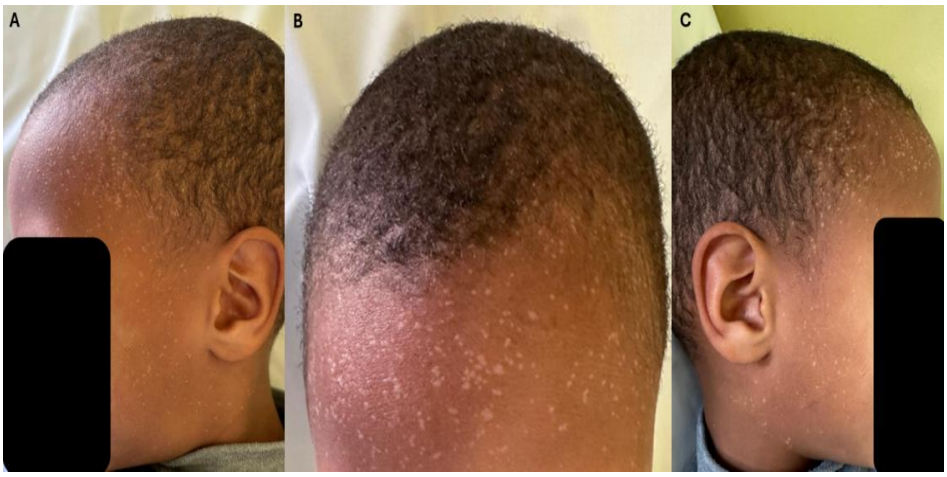


Figure 1 (A-C): Multiple well-defined hypopigmented macules and papules with a verrucous, finely scaly surface on the face (forehead and cheeks) in a child with phototype VI.

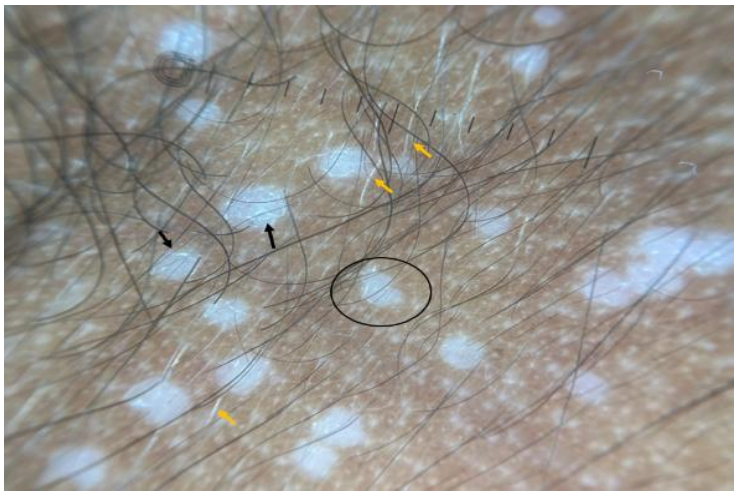


Figure 2: Dermoscopic examination of the forehead showing structureless white areas with interruption of the facial pseudopigment network (black circles), fine peripheral white scales (black arrows), and leukotrichic vellus hairs (orange arrows), without detectable vascular structures.

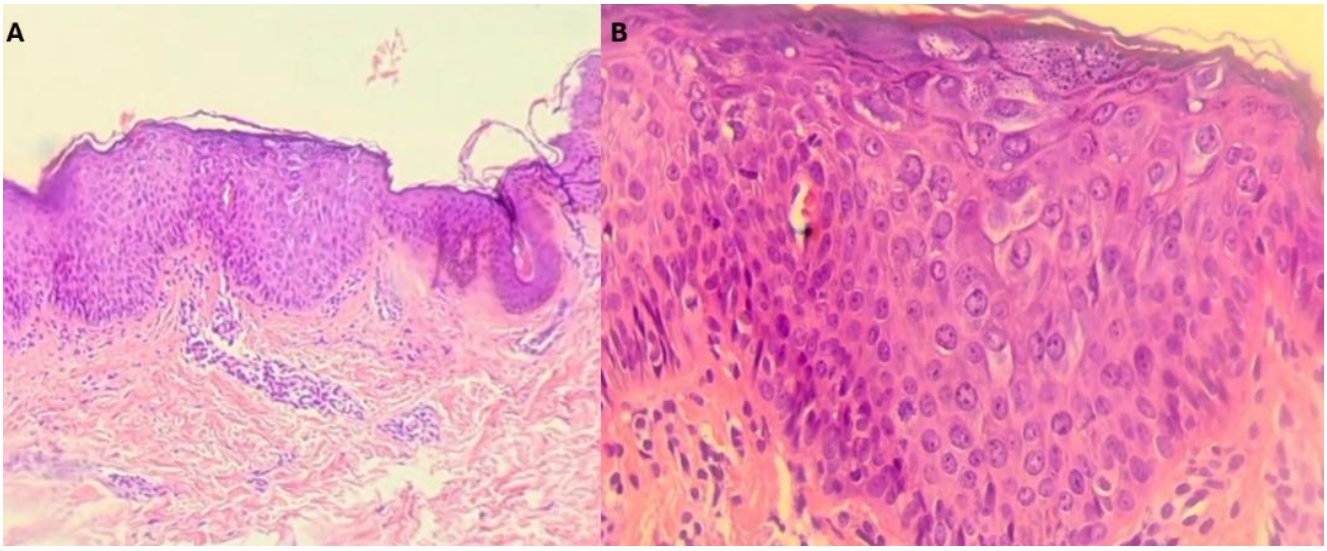


Figure 3 (A and B): Histopathological features of EV (hematoxylin–eosin stain). (A) Low-power view (×100) showing hyperkeratosis, mild acanthosis, enlarged keratinocytes within the epidermis, and a mild superficial lymphocytic inflammatory infiltrate in the dermis. (B) High-power view (×200) demonstrating enlarged keratinocytes with pale grayish glassy cytoplasm, round slightly atypical nuclei, and occasional perinuclear halos, characteristic of EV.



Figure 4 (A-C): Clinical follow-up at 6 months showing marked improvement of the cutaneous lesions, with significant regression of verrucous and flat wart-like lesions and no evidence of progression.

DISCUSSION

EV is an uncommon genodermatosis that typically presents during childhood with hypopigmented macules, flat wart-like papules, or pityriasis versicolor-like lesions, making early diagnosis challenging, particularly in patients with skin of color.[1,4] The clinical spectrum also includes EV associated with broader inherited immune defects and acquired EV in immunocompromised individuals.[9] In our patient, the early onset and absence of a reported history of immunosuppression support consideration of an inherited form, although clinical presentation alone cannot establish the underlying molecular defect. The absence of similarly affected relatives does not exclude a recessively inherited disorder.[2,3]

Although dermoscopy has become an essential tool in the evaluation of pigmented and inflammatory dermatoses, its application in EV remains poorly documented. Segura et al. described variable dermoscopic appearances across different lesion types, emphasizing the importance of histopathological correlation.[5]

Afra et al reported unfocused dotted vessels, a hypopigmented or erythematous background, whitish scales, and pigment-diluted vellus hairs in pityriasis versicolor-like lesions.[6] Sachdeva et al described the red dots, white scales, continuous marginal scaling, and brownish-black areas admixed with hypopigmented areas.[7] These observations suggest that a single uniform dermoscopic pattern should not be expected in every lesion.

Yotsu et al reported two siblings with Fitzpatrick phototype VI whose lesions appeared whitish against a dark background, with hypopigmented hairs arising within or around some lesions.[8] Our case similarly showed well-demarcated structureless hypopigmented areas and peripheral leukotrichic vellus hairs. Fine white scales were evident in our patient, whereas scaling was not prominent in their cases. Interruption of the facial pseudopigment network was an additional observation in our case. No vascular structures were detected; reduced visibility against heavily pigmented skin is a possible explanation, but cannot be established from this single observation. Consequently, the absence of visible vessels should not be treated as a defining feature of EV in phototype VI.

The clinicopathological findings provide complementary diagnostic information. The finely scaly surface is consistent with the histological hyperkeratosis, whereas mild acanthosis supports the epidermal thickening associated with the papular morphology. Enlarged keratinocytes with pale grayish, glassy cytoplasm and perinuclear halos provide characteristic histological evidence of EV in the appropriate clinical setting.[1,5,7] However, the microscopic findings described here do not directly establish the mechanism of the dermoscopic hypopigmentation or leukotrichia. These features should therefore be interpreted as associated observations rather than as proof of a specific pigmentary mechanism.

In our patient, the clinical differential diagnoses included pityriasis versicolor, verruca plana, and epidermal nevus. Hypopigmentation and fine scales overlap with pityriasis versicolor, while flat papules and verrucous surfaces can resemble plane warts. Dermoscopy helps characterize this overlap, but the published EV patterns are based mainly on small reports and their diagnostic specificity remains uncertain.[5-8] The combination of persistent lesions beginning in childhood, the observed dermoscopic findings, and confirmatory histopathology established the diagnosis in this case. Dermoscopy is therefore best considered an adjunct to clinical examination and biopsy rather than an independent confirmatory test.

Management of EV remains challenging because no curative treatment is currently available. Retinoids and other topical, systemic, or destructive approaches have been reported, with variable outcomes.[1] Our patient showed marked clinical improvement following topical adapalene, with good tolerability during follow-up. Nevertheless, the therapeutic efficacy of adapalene cannot be established from a single observation. The reduction in lesion thickness and scaling represents clinical improvement, but does not demonstrate viral clearance or correction of the underlying susceptibility. The six-month follow-up also does not establish whether improvement will persist over subsequent years.

Long-term care remains important even when childhood lesions improve. Clinical series have documented the subsequent development of cutaneous malignancies, particularly squamous cell carcinoma, highlighting the need for ongoing surveillance.[4,10] Photoprotection and continued dermatological follow-up should therefore accompany symptomatic management.[8] Changes such as persistent ulceration, rapid enlargement, or increasing induration warrant reassessment and consideration of biopsy. Short-term therapeutic improvement should not be interpreted as evidence of a reduced future cancer risk. These distinctions are particularly relevant when explaining the objectives of treatment and follow-up to a child's caregivers.

The main strength of this report lies in the clinicodermoscopic and histopathological documentation of EV in a child with Fitzpatrick phototype VI, a population that remains underrepresented in the literature. Its limitations include the single-patient design, descriptive assessment of treatment response, and limited follow-up. The report also provides no HPV typing or molecular confirmation, preventing genotype-specific interpretation. Further observations are needed to determine the reproducibility of these dermoscopic clues and their ability to distinguish EV from other hypopigmented pediatric dermatoses.

CONCLUSION

This case expands the limited literature on the dermoscopic features of EV in dark skin. The combination of structureless hypopigmented areas, interruption of the facial pseudopigment network, fine white scales, and peripheral leukotrichic vellus hairs may serve as useful dermoscopic clues, facilitating early recognition and appropriate histopathological confirmation. These findings remain descriptive and require validation in larger series. Clinical improvement with topical adapalene in this child supports further documentation of treatment outcomes, while photoprotection and long-term surveillance remain essential components of care.

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