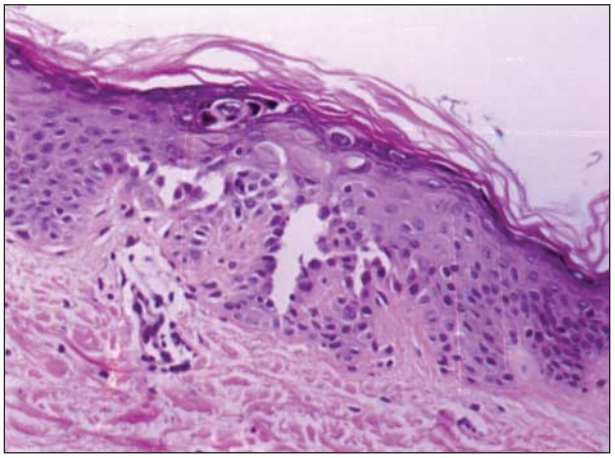

Figure 2. Suprabasilar acantholysis with corps ronds (hematoxylin-eosin, original magnification ×10).

the fifth to sixth decade of life. Initially thought to be a transient condition, persistent cases exist.[4] The cause is uncertain, but UV radiation, radiation therapy, xerosis, hot environmental conditions, and increased sweating have been implicated.[2,5]

The present findings suggest that increased sweating and dysfunction of the postganglionic sympathetic fibers that innervate the eccrine glands may cause transient acantholytic dermatosis and secondary hyperhidrosis. Physicians caring for patients with spinal cord trauma should be aware of this possibility to avoid erroneous alternative diagnoses such as drug eruptions or viral exanthems.

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Clinical and Dermoscopic Features of Porokeratosis of Mibelli

Porokeratosis is a rare, genetically determined disorder of epidermal keratinization characterized by lesions with keratotic borders corresponding histopathologically to compact columns of parakeratotic cells called *cornoid lamellae* that extend through the stratum corneum.[1] The clinical variants include porokeratosis of Mibelli (PM), disseminated superficial actinic porokeratosis,

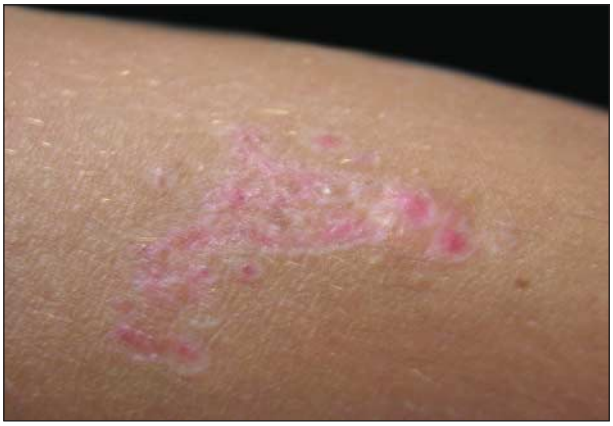

Figure 1. Clinical image revealing a whitish-red plaque on the right forearm of a 15-year-old boy. The affected area is made up of numerous whitish-red round papules that coalesce into irregular plaque and single papules, the area perimeter defined by a whitish border and cleaved by a central furrow. Slightly raised whitish-red portions can also be observed (original magnification ×20).

linear porokeratosis, porokeratosis palmaris, plantaris et disseminate, and punctate porokeratosis.[1]

Report of a Case. A 15-year-old boy presented with an area of asymptomatic dermatosis on the anterior right forearm that had first developed 12 years earlier and enlarged slightly over the years. No family history of porokeratosis was reported. Clinical examination revealed whitish-red round papules that coalesced into an irregular plaque and single papules, the overall patch extending 3.5 cm in length on the forearm. The area of plaque and papules had an annular appearance with whitish borders. It was cleaved by a central furrow with central, slightly raised whitish-red portions on either side of the furrow (**Figure 1**). Clinical differential diagnoses included patch dermatitis, actinic keratosis, and Bowen disease.

Dermoscopic evaluation of the lesion (**Figure 2**) revealed a thin, whitish rim surrounding the entire perimeter of the affected area; in some sections of this perimeter, the typical brown globules and/or dots conjoined to form a continuous line inside a whitish rim. In the central portion, a diffuse whitish-brown pigmentation with brown globules and/or dots was seen. Moreover, we observed dotted and linear vessels, regular and irregular, straight and curved, partially obscured by erythematous patches.

A 5-mm punch biopsy specimen of the peripheral hyperkeratotic ridge was obtained, and histopathologic examination showed parakeratotic columns within the stratum corneum of the epidermis, horny cells with pyknotic nuclei (cornoid lamella), and cytoplasmic features of premature keratinization. The underlying granular layer was thin or absent (**Figure 3**). No lymphoid infiltrate was present in the dermis. Gram stain highlighted the blue cornoid lamellae (Figure 3B). Based on clinicodermoscopic and histopathologic assessment, the lesion was diagnosed as PM.

Comment. Porokeratosis of Mibelli was first described by Mibelli in 1893 as single or multiple annular and gy-

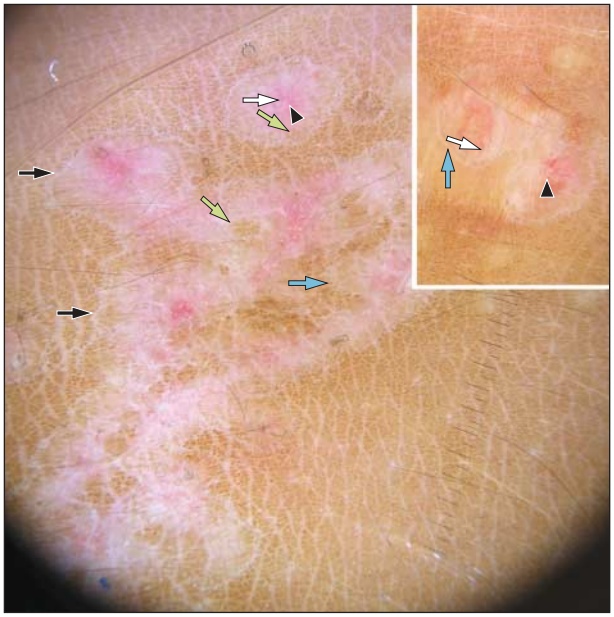

Figure 2. Dermoscopic images of the affected area exhibiting a cluster of lesions coalescing into plaque on the right forearm of a 15-year-old boy (original magnifcation of the main photograph ×10; inset, ×20). A thin whitish rim surrounds the entire perimeter of the affected area (black arrows), and brown close dots join to form a continuous line present inside the whitish rim (blue arrows). In the central portion of the lesions, a diffuse, whitish-brown pigmentation, globules, and dots (green arrows), red dots (arrowheads), and red lines (white arrows) (enlarged in the inset) can be recognized.

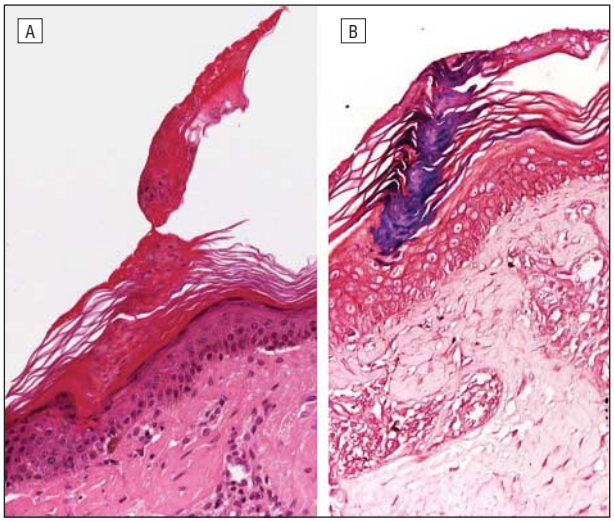

Figure 3. Histopathologic images under hematoxylin-eosin (A) and gram (B) stains (original magnification ×250 for both panels). The cornoid lamella (bright blue under gram stain) is characterized by a column of parakeratosis with a normal orthokeratotic epidermal stratum corneum above a thin granular layer. A few vacuolated and dyskeratotic cells are evident in the spinous layer.

rate plaques with central atrophy and elevated keratotic borders containing a longitudinal furrow.[2] It usually has onset in childhood but may appear at any age, especially in nonhereditary cases, with unilateral localized lesions. Various clinical types of PM were later described.[1,2]

Our case involved lesions clinically similar to those originally described by Mibelli; in fact, even in his patient, the lesions were whitish-red and bordered by an elevated ridge.[2] Dermoscopically, our case clearly displayed the thin, whitish peripheral rim corresponding histopathologically to cornoid lamella as reported in the literature.[3-5] The typical brown globules and/or dots seen within the whitish rim are histopathologically associated with tiny melanophages in the subepidermal region (Figures 2 and 3) and probably represent their postinflammatory accumulation.[3] Moreover, red dots and red lines suggest the vascular pattern seen in atrophic and inflammatory skin.[6]

Use of dermoscopy improves our ability to recognize the hyperkeratotic border, which may aid in the accurate diagnosis of PM and exclude patch dermatitis, actinic keratosis, and Bowen disease, conditions that may clinically mimic PM. In conclusion, the peripheral whitish rim represents the morphologic hallmark of porokeratosis, an essential pathognomonic dermoscopic feature for its diagnosis.

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Progression of Undiagnosed Cutaneous T-Cell Lymphoma During Efalizumab Therapy

Report of a Case. A 32-year-old man presented with a 2-year history of a pruritic, erythematous dermatitis. A skin biopsy was performed, and he was diagnosed with psoriasis. Treatment was administered with topical corticosteroids, acitretin, UV-B twice per week, and efalizumab, 80 mg, subcutaneous injections each week for 4 months without success. Within 3 months of stopping efalizumab therapy, he developed tumors on his face and ears and presented to the university clinic for evaluation. Other physical findings included alopecic scalp plaques, erythroderma sparing skin folds, palmar and plantar desquamation, erythematous plaques on his back, and no palpable lymphadenopathy.