Correspondence

Successful treatment of disseminated superficial actinic porokeratosis with calcipotriol

Editor,

Disseminated superficial actinic porokeratosis (DSAP) is the most common type of porokeratosis. It is related to sun exposure and an autosomal dominant inheritance pattern is recognized. No ideal treatment of this clonal disorder of keratinization exists. We describe a patient in whom the complete resolution of lesions was achieved with topical treatment with the vitamin D3 analog calcipotriol. Thus we add more evidence on the efficacy of calcipotriol to the literature.

We present a 73-year-old Caucasian woman with a 5-month history of lesions on her lower legs. No subjective complaints were reported. Family history was negative. The patient had no medical history of immunosuppression, x-ray treatment, or arsenic intake. Extensive sun exposure was evident.

On physical examination, multiple red–brown, annular macules were seen to be evenly distributed on the front and lateral surfaces of the lower legs (Fig. 1). Close examination showed central atrophy and an elevated hyperkeratotic ridge. Some scaly plaques were observed on the forearms.

A punch biopsy specimen taken from the keratotic border disclosed the characteristic cornoid lamella, as well as atrophy of the epidermis, flattening of the rete ridges, and absence of the granular layer. Perivascular lymphocytic infiltrate was present in the upper dermis (Fig. 2).

In accordance with the clinical presentation and histological findings, DSAP was diagnosed. Topical treatment with calcipotriol 0.005% cream twice daily for 3 months was started with excellent therapeutic outcome. At follow-up 6 months later, the patient was free of lesions.

Disseminated superficial actinic porokeratosis was first described by Chernosky and Freeman, since then various treatment options have been described but are poorly standardized. There is a lack of controlled studies and therapy is usually empiric. It encompasses potent topical steroids, keratolytics, topical retinoids, topical 5-fluorouracil, imiquimod 5%, anthralin, cryotherapy, carbon dioxide laser, pulsed dye laser, curettage, excision, dermabrasion, and oral retinoids. A summary of therapeutic regimens for DSAP, together with their advantages and disadvantages, is given in Table 1.

Treatment with calcipotriol and another vitamin D3 analog, tacalcitol, has been rarely reported. Vitamin D3 analogs induce genes critical for keratinocyte differentiation, such as transglutaminase or involucrin.

Figure 1 Multiple brownish annular, plaques distributed evenly on the lower legs. Close inspection of lesions revealed central atrophy with an elevated keratotic border

Figure 2 Histology showed the typical cornoid lamella. (Hematoxylin and eosin stain; original magnification ×100)
They may also inhibit proliferation by inducing sphingomyelin hydrolysis and modulation of protein kinase C activity. The significant effect of both vitamin D₃ analogs suggests that keratinocytes present in the lesional skin in DSAP express functional vitamin D₃ receptors.

By contrast, immunohistochemical studies show that keratinocytes beneath and central to the cornoid lamella in DSAP stain in patterns similar to those of squamous cell carcinoma and actinic keratosis, respectively. The good response of DSAP to calcipotriol raises the question of whether porokeratosis is a result of faulty maturation of keratinocytes or an increased rate of proliferation. In addition, the poor effects of photodynamic therapy with aminolevulinic acid (ALA-PDT) and 3% diclofenac gel confirm that DSAP does not have much in common with actinic keratosis in terms of its pathophysiology.

**Table 1** Treatment options for disseminated superficial actinic porokeratosis

<table>
<thead>
<tr>
<th>Treatment modality</th>
<th>Drug/treatment option</th>
<th>Advantages</th>
<th>Disadvantages</th>
<th>References</th>
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<tbody>
<tr>
<td>Topical</td>
<td>Potent steroids</td>
<td>Relief of pruritus</td>
<td>Low clearance rate</td>
<td>McDonald &amp; Peterka¹¹</td>
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<td>Keratolytics</td>
<td>Keratolytics</td>
<td>Reduce hyperkeratosis and roughness</td>
<td>No effect on erythema and pigmentation</td>
<td>Chernosky &amp; Freeman⁷</td>
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<tr>
<td>5-Fluorouracil</td>
<td>Simple</td>
<td>Occision</td>
<td>Long treatment course</td>
<td>Shelley &amp; Shelley¹²</td>
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<td>Calcipotriol</td>
<td>Simple</td>
<td>Insufficent data</td>
<td>Insufficent data</td>
<td>Harrison &amp; Stollery³</td>
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<tr>
<td>Imiquimod 5% cream</td>
<td>Simple</td>
<td>Strong inflammatory reactions</td>
<td>Expensive</td>
<td>Ahn et al.¹³</td>
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<td>Diclofenac 3% gel</td>
<td>Simple</td>
<td>No benefit</td>
<td>Side-effects (irritation, pigmentation, ulceration)</td>
<td>Vlachou et al.⁹</td>
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<tr>
<td>Systemic Retinoids</td>
<td>Most reproducible results</td>
<td>Without longterm remission</td>
<td>Side-effects</td>
<td>Hacham-Zadeh &amp; Holubar¹⁴</td>
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<td>Procedural</td>
<td>Cryotherapy</td>
<td>Few complications</td>
<td>Pretreatment may be needed to remove the hyperkeratotic border</td>
<td>Dereli et al.¹⁵</td>
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<td></td>
<td>Simple</td>
<td>High percentage of cure</td>
<td>Scar formation</td>
<td></td>
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<td></td>
<td>Low cost</td>
<td>Short treatment period</td>
<td></td>
<td></td>
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<tr>
<td>ALA-PDT</td>
<td>Field treatment</td>
<td>No benefit</td>
<td>Nayeemuddin et al.¹⁰</td>
<td></td>
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<tr>
<td>CO₂ laser</td>
<td>Short treatment period</td>
<td></td>
<td></td>
<td>Barnett¹⁶</td>
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<td>Pulsed dye laser</td>
<td>Cosmetic improvement</td>
<td>Scarring</td>
<td>Hypopigmentation</td>
<td>Alster &amp; Nanni¹⁷</td>
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<td>Dermabrasion</td>
<td>High cost</td>
<td>High cost</td>
<td>Campbell &amp; Voorhees¹⁸</td>
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<tr>
<td></td>
<td>Variable degree of success</td>
<td>Scarring</td>
<td>Hypopigmentation</td>
<td></td>
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<td>Excision</td>
<td>Required if any suspicion of malignant degeneration occurs</td>
<td>Not useful for multiple lesions</td>
<td>Scarring</td>
<td>Shelley &amp; Shelley¹²</td>
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ALA-PDT, aminolevulinic acid with photodynamic therapy

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By contrast, immunohistochemical studies show that keratinocytes beneath and central to the cornoid lamella in DSAP stain in patterns similar to those of squamous cell carcinoma and actinic keratosis, respectively. The good response of DSAP to calcipotriol raises the question of whether porokeratosis is a result of faulty maturation of keratinocytes or an increased rate of proliferation. In addition, the poor effects of photodynamic therapy with aminolevulinic acid (ALA-PDT) and 3% diclofenac gel confirm that DSAP does not have much in common with actinic keratosis in terms of its pathophysiology.

Ilko Bakardzhiev, MD PhD
Department of Dermatology and Venereology
Resort Clinic
Varna
Bulgaria

Svetlana Kavaklieva, MD
Georgy Pehlivanov, MD PhD
Department of Dermatology and Venereology
Faculty of Medicine
University of Sofia
Sofia
Bulgaria
E-mail: kavaklieva@abv.bg

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References


