## Solitary Lesion on the Left Ankle



A 69-year-old white man presented with a solitary lesion on the left ankle. His medical history included hypertension and arthritis. He resided in Florida for 11 years but denied tanning and has had sensitive skin throughout his life. He had no other notable skin conditions, except for nummular eczema. He did not have a family history of skin cancer. Physical examination showed the single lesion on the left ankle.

## What's the diagnosis?

- a. disseminated superficial actinic porokeratosis
- b. linear porokeratosis
- c. porokeratosis of Mibelli
- d. porokeratosis palmaris et plantaris disseminata
- e. punctate porokeratosis

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The authors report no conflict of interest.

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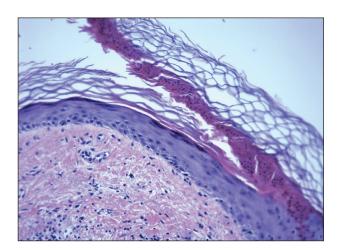
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## The Diagnosis: Porokeratosis of Mibelli

There are 5 variants of porokeratosis: disseminated superficial actinic porokeratosis (DSAP), linear porokeratosis, porokeratosis of Mibelli, porokeratosis palmaris et plantaris disseminata, and punctate porokeratosis. The most common type is DSAP,<sup>1</sup> which is characterized by multiple lesions on the body, particularly in sun-exposed areas. The distinguishing feature of porokeratosis is the cornoid lamella, which is made up of parakeratotic cells extending through the stratum corneum. There also is a thin or absent granular layer beneath it (Figure).<sup>2</sup>

Patients generally present in the third and fourth decades of life. Risk factors for porokeratosis include sun exposure, immunosuppression, and genetics. Overexpression of the protein p53 in porokeratosis lesions has been demonstrated in studies investigating the genetics of porokeratosis. A study of Chinese families with DSAP identified 3 different loci associated with DSAP: DSAP1, DSAP2, and DSAP3. The progression to cancer has been noted in all types of porokeratosis lesions. Malignancies include squamous cell carcinoma, Bowen disease, and basal cell carcinoma.

Many treatments have been tried for DSAP including cryotherapy, topical 5-fluorouracil, photodynamic therapy, and topical imiquimod with



A punch biopsy of the lesion demonstrated histology that corresponded with disseminated superficial actinic porokeratosis including a characteristic cornoid lamella; however, the clinical diagnosis was determined to be porokeratosis of Mibelli (H&E, original magnification ×200).

varying success.<sup>1</sup> Our patient was treated with cryotherapy but had side effects from treatment including cellulitis and local infections with ulceration before finally healing.

Interestingly, our patient had a single lesion with pathology findings most consistent with DSAP at a later age. Although the pathology suggested DSAP, the size and solitary lesion was more consistent with porokeratosis of Mibelli. Porokeratosis of Mibelli can occur concurrently with DSAP, but we have not seen other lesions in this patient. We have educated our patient to be aware of other lesions that may occur in the future. Due to risk for malignant conversion, it is generally viewed as beneficial to treat patients who present with porokeratosis lesions. Our patient's lesion ultimately cleared and he has not developed new lesions at 1-year follow-up.

Although DSAP generally presents in the third and fourth decades of life and porokeratosis of Mibelli during childhood, it is important to educate both dermatologists and primary care physicians to be aware of the possibility of both diagnoses in the elderly population.

## **REFERENCES**

- 1. Rouhani P, Fischer M, Meehan S, et al. Disseminated superficial actinic porokeratosis. *Dermatol Online J*. 2012;18:24.
- 2. Murase J, Gilliam AC, et al. Disseminated superficial actinic porokeratosis co-existing with linear and verrucous porokeratosis in an elderly woman: update on the genetics and clinical expression of porokeratosis. *J Am Acad Dermatol.* 2010;63:886-891.
- 3. Lederman JS, Sober AJ, Lederman GS. Immunosuppression: a cause of porokeratosis? *J Am Acad Dermatol*. 1985;13:75-79.
- Hernandez MH, Lai CH, Mallory SB. Disseminated porokeratosis associated with chronic renal failure: a new type of disseminated porokeratosis? Arch Dermatol. 2000:136:1568-1569.
- Magee JW, McCalmont TH, LeBoit PE. Overexpression of p53 tumor suppressor protein in porokeratosis. Arch Dermatol. 1994;130:187-190.
- 6. Arranz-Salas I, Sanz-Trelles A, Ojeda DB. p53 alterations in porokeratosis. *J Cutan Pathol*. 2003;30:455-458.
- 7. Curnow P, Foley P, Baker C. Multiple squamous cell carcinomas complicating linear porokeratosis. *Australas J Dermatol.* 2003;44:136-139.

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- 8. Lee HR, Han TY, Son SJ, et al. Squamous cell carcinoma developing within lesions of disseminated superficial actinic porokeratosis. *Ann Dermatol.* 2011;23:536-538.
- 9. Mehta V, Balachandran C. Simultaneous co-occurrence of porokeratosis of Mibelli with disseminated superficial actinic porokeratosis. *Indian J Dermatol.* 2009;54:390-391.