WART-LIKE POROKERATOSIS OF MIBELLI

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Abstract. Four members of a black family presenting wart-like lesions which proved to be porokeratosis of Mibelli are described.

This report of four members of a black family with porokeratosis of Mibelli illustrates two morphologic types of lesion in this disease, one of which is the easily overlooked warty variety.

Porokeratosis of Mibelli is classically inherited in autosomal dominant fashion (2, 15). However, non-familial cases have been reported (6, 14). Possibly in some of such cases lesions of the warty variety have been overlooked.

The type of eruption most often encountered is the round, oval, or irregular gyrate plaque with a thin guttered keratotic rim (2). Occasionally, disseminated eruptive very superficial lesions occur (5). Another type is the tiny keratotic wart-like papule several millimeters in diameter which is surrounded by a raised horny wall (2, 11). These small lesions have a tendency to coalesce to form linear streaks (2, 7). A third type presents larger wart-like horny formations (10). Different types of lesions may be present in different members of the same family. In the family studied by Bloom & Abramowitz (2), the father showed characteristic plaques on the fingers while the son showed linear type lesions on the neck.

Porokeratosis has been reported in negroes (1, 9).

CASE REPORT

This 23-month-old male infant presented with a “wart” on the left sole which appeared at the age of 3 months and gradually enlarged. At about the same time a similar lesion appeared on the medial left ankle. Both lesions were completely asymptomatic and no previous treatment had been given.

Family history revealed that the mother, age 19, maternal grandmother, age 48, and a maternal aunt, age 24, had also had “warts” since childhood (Fig. 1). Most of their lesions started as flat keratoses and tended slowly to develop a verrucous surface and then either remained stationary or flattened slightly. These lesions did not ex-
Aside from classic porokeratosis of Mibelli, two other types of porokeratosis have recently been described. These are disseminated superficial actinic porokeratosis (DSAP) (3) and porokeratosis plantaris, palmaris et disseminata (PPPD) (8). Histologically all types of porokeratosis are characterized by the presence of cornoid lamellae which consist of well defined, narrow columns of parakeratosis below which the granular cell layer is absent or diminished. Cornoid lamellae may or may not start in ductal or follicular ostia (13). DSAP and PPPD are said to have less prominent cornoid lamellae (8).

In the DSAP type of Chernosky, the keratotic ridges never exceed 1 mm in width. The lesions are acquired in later life and occur in sun-exposed areas. The patient's ankle lesion presented similar histopathological changes and showed several cornoid lamellae. All were highly characteristic of porokeratosis of Mibelli (Fig. 3).

DISCUSSION

Physical examination showed a 4 mm keratotic lesion without a peripheral ridge on the left medial ankle. A 1 x 0.5 cm irregular hard keratosis with a sharply defined keratotic rim was present on the left sole (Fig. 2). No puncta of degenerating hemorrhage were noted on paring the superficial keratin.

Except for the skin lesions, general physical examination was unremarkable.

Histopathologic examination of two biopsy specimens from the mother's knee and elbow showed alternate columns of parakeratosis and orthokeratosis. The granular layer was missing beneath the parakeratotic columns. A few sparse lymphocytic infiltrates were noted in the dermis. The patient's ankle lesion presented similar histopathological changes and showed several cornoid lamellae. All were highly characteristic of porokeratosis of Mibelli (Fig. 3).
exposed areas. They may be exacerbated after sun exposure and may even be induced by artificial ultraviolet irradiation (3, 4). Guss et al. suggested that PPPD was a distinctive type of porokeratosis because lesions appeared initially on the palms and soles in the late teens and early twenties and later became widely disseminated as hundreds of lesions over the body. They observed eight cases in four generations in one family which showed an autosomal dominant mode of inheritance. There has been some reluctance to accept PPPD as a type of porokeratosis distinctive from the classic Mibelli variety (12).

Wart-like lesions occurring in relatives of patients with porokeratosis of Mibelli may be a subtle manifestation of this genetic disorder which can be easily misinterpreted.

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REFERENCES

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