

An important factor in determining the impact of the development of resistance of *P. acnes* and aerobic micrococci to topical antibiotics, is the kinetics of reconstitution of an antibiotic-sensitive surface microflora. Our investigation did not include any follow-up after treatment was concluded. However, there is some preliminary evidence (2) that normally sensitive strains of *P. acnes* repopulate comedones 1–2 months after topical clindamycin or erythromycin is discontinued.

The widespread usage of topical antibiotics for the treatment of acne has profound effects on the resident microbial flora of the skin. Further study of the long-term ecological effect of these agents is warranted, particularly in view of their widespread use.

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Superficial Actinic Porokeratosis in a Patient Undergoing Long-term PUVA Therapy

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Abstract. A patient who was treated with PUVA for psoriasis developed disseminated superficial actinic porokeratosis. The significance of this complication is discussed.

Disseminated superficial actinic porokeratosis (DSAP) might be expected as a side effect of PUVA therapy.

CASE REPORT

Patient G. M. L., female, 67 years old, skin type III, has had psoriasis vulgaris since 1942. She has never been treated with arsenic for her disease. Her brother has psoriasis. There is no known family history of porokeratosis.

Standardized PUVA conditions had been used since November 1977, with 40 mg 8-MOP tablets (Meladinine; Promédica, France) and total irradiation 2 h later. The cumulative UVA dose was 611 j/cm². Localized PUVA therapy was also given to the legs after application of 8-MOP 1% solution. The UVA dose was 350 j/cm². Up to December 1979 the total dose applied to her legs was 961 j/cm².

At that time the psoriasis was under control, but various lesions appeared, localized essentially on the anterior surface of both legs. Clinically there were multiple superficial keratotic lesions surrounded by a slightly raised keratotic border. These small circinate lesions enlarged in a centrifugal fashion, the older central portion becoming atrophic and less keratotic (Fig. 1). A histological study of the keratotic rim of this lesion showed a well delimited parakeratotic zone above the spinous layer which displayed several isolated dyskeratotic cells (Fig. 2).

A diagnosis of DSAP was made.

DISCUSSION

This case of actinic porokeratosis during PUVA therapy is the first one observed, to our knowledge.

Disseminated superficial actinic porokeratosis (1) is a dominant autosomic genodermatosis, with some sporadic cases recorded. The disease begins

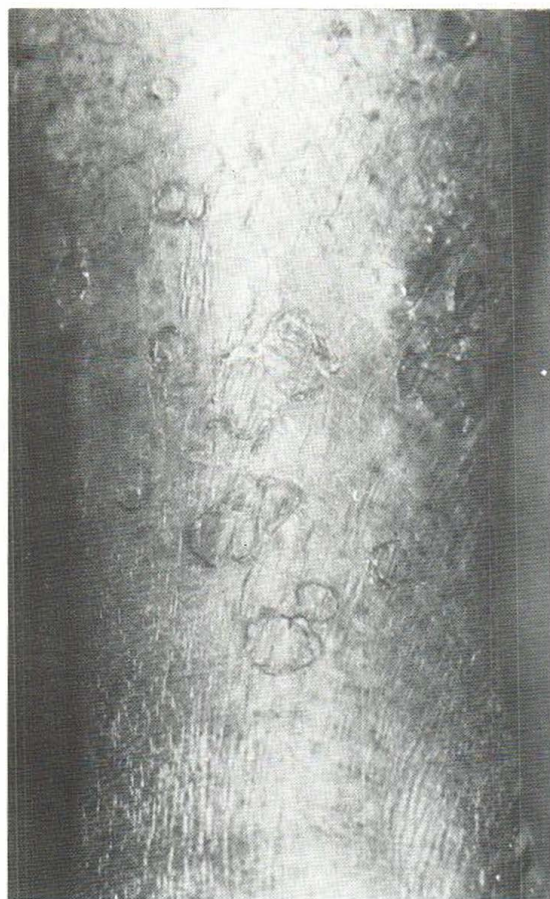


Fig. 1. Actinic porokeratosis on the anterior aspect of the legs after a cumulative UVA dose of 961 J/cm².

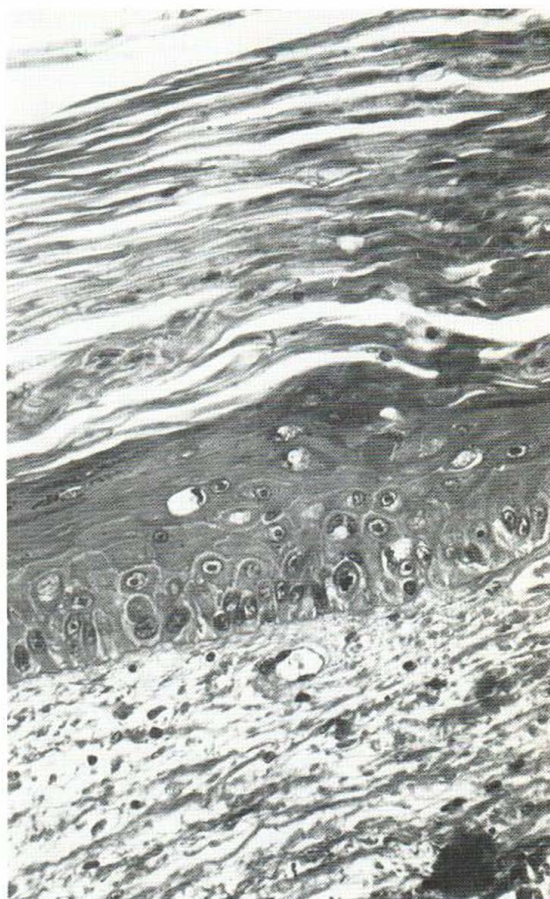


Fig. 2. Histological study of the keratotic rim, showing a parakeratotic column above the spinous layer displaying several isolated dyskeratotic cells.

and is always predominant on the sun-exposed skin; typical lesions have been experimentally provoked with prolonged artificial ultraviolet light (2). Clinical and histological characteristics are indistinguishable from Mibelli's Porokeratosis (PM) in its disseminated variant. PM and DSAP represent a clonal disease of the epidermis; the tendency of the clones to develop is probably inherited but in many cases the latent abnormal clones become clinically overt following actinic exposure (4).

The potential carcinogenicity of DSAP is unknown at present, while PM appearing as Bowenoid epithelioma is well documented (3).

Attention must be drawn to the potential risk of PUVA therapy due to the great similarity between these two diseases.

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