fibroblasts derived mainly from PK skin lesions, which suggests a relationship to malignant tumour development (8, 9). Although we examined chromosomal abnormalities in cells from various sites in the lesion and the normal-appearing skin of case 3, no clonal proliferation of such abnormal cells could be detected, nor could the specific site of such abnormalities be identified. However, various kinds of structural abnormalities were found in fibroblasts of this patient’s skin. These abnormal cells may explain the development of abnormal clones in the epidermis of the PK skin lesion, even though the mechanism or process has not been clarified.

Our present study suggests that large PK skin lesions contain proliferating clones in the epidermis, which are probably responsible for the formation of large skin lesions.

REFERENCES

Superficial Actinic Porokeratosis and Cystic Fibrosis

L. KLAPHOLZ,' M. GOLDENHERSH, Y. SHERMAN and V. LEIBOVICI
Departments of 'Dermatology and 2Pathology, Hadassah University Hospital, Jerusalem, Israel

A 24-year-old woman, presenting with cystic fibrosis, developed superficial actinic porokeratosis. Immunosuppression due to cystic fibrosis may be either the cause of or the exacerbating factor in superficial actinic porokeratosis in our patient.

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V. Leibovici, Post Office Box 12018, Hadassah University Hospital, Jerusalem 91120, Israel.

Porokeratosis is a distinctive skin eruption characterized by hyperkeratotic plaques with elevated borders and atrophic centres. In addition to the original plaque type described by Mibelli, several other varieties have been reported, including a superficial disseminated form, disseminated superficial actinic porokeratosis (DSAP), punctate porokeratosis of the palms and soles, and a linear type. Although each form represents a distinct clinical entity, they share similar histological changes, i.e. a characteristic column of parakeratotic cells (cornoid lamella).

Recently, several authors reported the association of porokeratosis with immunosuppression. To the best of our knowledge, this is the first case report of superficial actinic porokeratosis occurring in a patient with cystic fibrosis.

CASE REPORT
A 24-year-old woman suffering from multiple hyperkeratotic lesions on both legs, was referred to the Department of Dermatology at Hadassah Medical Center. The lesions, with a diameter of 0.5–1.0 cm, had an elevated border and were asymptomatic (Fig. 1). There was no family history of porokeratosis of Mibelli. Cystic fibrosis, which was diagnosed in her infancy, was complicated by liver cirrhosis at the age of 10 years. In 1975 an urgent porto-caval shunt was performed following acute hemorrhage of varicose veins in the esophagus. Since 1981 she has been treated with glibenclamide for diabetes mellitus. During the past 2 years she had twelve episodes of pulmonary infections, most of them

Acta Derm Venereol (Stockh) 71
The lesions were treated with liquid nitrogen, though with minimal response.

DISCUSSION

Recently, an association of porokeratosis of Mibelli with disseminated superficial actinic porokeratosis (DSAP) was reported in several immunosuppressed patients. The immunosuppression in these patients was due to renal transplantation (1, 2), malignancy and chemotherapy (3), mycosis fungoides (4), plasma exchange therapy for primary biliary cirrhosis (4), PUVA therapy for psoriasis (5) and immunosuppressive treatment for pemphigus foliaceus (6).

Read & Leone (7) have proposed that porokeratosis is a disease of the epidermis in which a mutant clone of epidermal cells is responsible for the formation of the parakeratotic column.

In our patient, aggravation of the pulmonary state seemed to correlate with the appearance of the superficial actinic porokeratosis. It is known that overstimulation of the immune system in cystic fibrosis, due to secondary bacterial infections, may result in immunosuppression (8). This could account for the porokeratosis of Mibelli observed in our patient.

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