Disseminated Porokeratosis and Myelodysplastic Syndrome

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The role of actinic radiations in the development of disseminated superficial actinic porokeratosis (DSAP) is well accepted [1, 2], Both the classic form of porokeratosis (Mi-belli) and DSAP have also been related to immunosuppression which in most cases reported was due to immunosuppressive therapy. We describe a patient with myelodysplastic syndrome (chronic refractory anemia) who developed DSAP when being treated exclusively with blood transfusions.

A 72-year-old man had had myelodysplastic syndrome (chronic refractory anemia) since March 1984. He was admitted at our institution in August 1989 for lobar pneumonia. During his stay, micropapular perifollicular lesions, slightly hyperpigmented and asymptomatic, were detected that did not worsen upon sun exposure; the patient denied having similar lesions among his relatives. Histology was compatible with porokeratosis. The patient was not given any treatment for his skin lesions; therapy for his anemia was red cell concentrates. In December 1989 he was readmitted in our hospital for a mucocutaneous jaundice due to a posttrans-fusional hepatitis. The physical examination showed a dramatic increase in the number, size and distribution of the porokeratotic skin lesions. A few days after his admission, there was an important deterioration in his general condition with serious pancytopenia; new skin elements continued to erupt. He soon died of sepsis, liver insufficiency and disseminated intravascular coagulation.

Most authors agree that therapeutic immunosuppression seems to either trigger or aggravate DSAP in genetically predisposed patients [3-7). How immunosuppressive therapy acts in the promotion of DSAP lesions is not fully understood. This case suggests a multifactorial origin for the failure in immunosurveillance mechanisms; it might be that sudden aggravation of DSAP justifies the search for an underlying immunosuppressive disease.

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