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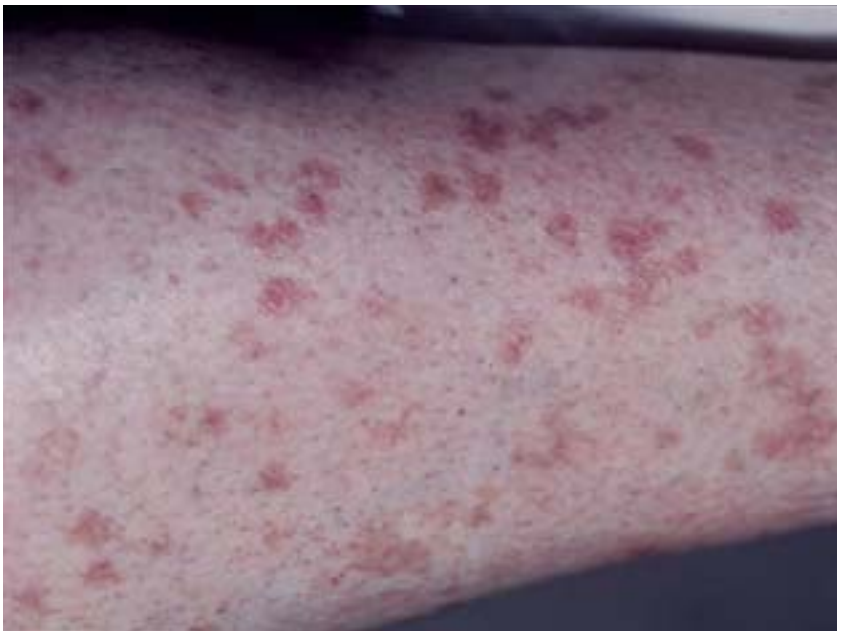
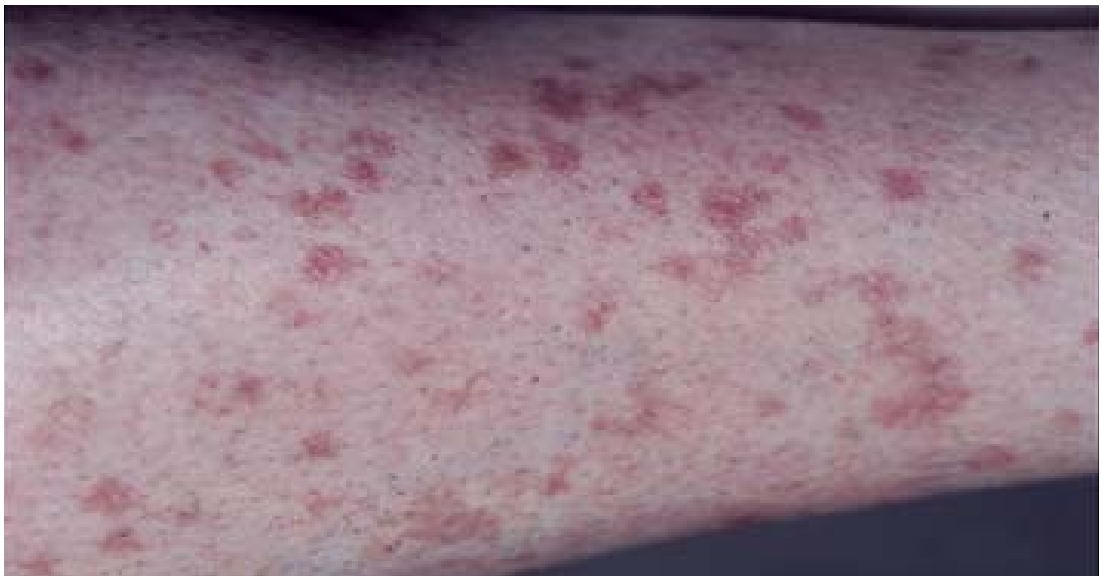


A 68-year-old woman complains of unsightly spots on her legs. She reports that she was living in Texas when she first noticed the lesions.

What is your diagnosis?

PLEASE TURN TO PAGE 296 FOR DISCUSSION

The Diagnosis: Disseminated Superficial Actinic Porokeratosis



Disseminated superficial actinic porokeratosis is a generalized process that involves sun-exposed skin.[1] A symmetric bilateral distribution involving the extremities is most characteristic. Non–sun-exposed sites also may be involved. Furthermore, the lesions may worsen during the summer months. Many small red-brown lesions with keratotic rims, as well as central hyperpigmentation, are characteristic. Lesions may slowly enlarge and take on an annular appearance. Patients typically present for evaluation in the third to fourth decade of life because the lesions are unsightly. Lesions of porokeratosis are most often asymptomatic and only slowly progressive. An autosomal dominant mode of inheritance has been described. Exposure to ultraviolet light, as well as phototherapy, has been noted to bring out this condition.

Treatment with liquid nitrogen cryotherapy, topical tacalcitol,[2] topical 5-fluorouracil, as well as destructive laser therapy may be of value. Keratolytic agents may lead to transient improvement but not to meaningful resolution. Autotransplantation studies suggest that changes in the graft bed may predispose to formation of disseminated superficial actinic porokeratosis. Immunosuppressive medication has been associated with the development of disseminated superficial actinic porokeratosis.[3] Likewise, immunosuppression in the setting of renal transplantation has been associated with other forms of porokeratosis, such as the Mibelli variant, as well as an increased number of cutaneous nonmelanoma malignancies. Abnormalities in p53 tumor suppressor protein levels may play a role in these cutaneous changes.[4] Affected keratinocytes exhibit abnormal DNA ploidy; overexpression of p53; and abnormal expression of cytokeratins, filaggrin, and involucrin. Keratinocytes exhibit chromosomal instability and increased fragility when exposed to radiography.[5]

The differential diagnosis of disseminated superficial actinic porokeratosis includes porokeratosis of Mibelli. Classic porokeratosis of Mibelli presents as a few nontender papules, with elevated borders on the extremities. The palms, soles, and mucosal surfaces are commonly involved. Lesions develop in childhood, and an autosomal dominant mode of inheritance is typical. All forms of porokeratosis are thought to develop from an abnormal clone of

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keratinocytes. Immunosuppression, burns, and ultraviolet light also can lead to the development of lesions.[6] Even a disseminated superficial actinic porokeratosislike drug eruption has been noted. Malignant degeneration has been reported with all types of porokeratosis. Summer exacerbation has been noted with disseminated superficial actinic porokeratosis and porokeratosis palmaris et plantaris disseminata but not with linear porokeratosis or the classic porokeratosis of Mibelli.

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