Case Letter

Pityriasis Rubra Pilaris Triggered by Photodynamic Therapy With Response to Tumor Necrosis Factor α–Blocking Agents and Acitretin

To the Editor:

A 62-year-old man presented with an erythematous and scaly plaque measuring 11×9 mm on the forehead of 3 months' duration. A diagnosis of actinic keratosis was confirmed by histopathologic analysis of a punch biopsy specimen, and the patient received 1 treatment with methyl aminolevulinate photodynamic therapy (PDT). One week later, the patient presented with tender, dark red, erythematous, scaly plaques on the right hemifacial area, extending to his neck (Figure 1) and upper trunk within days after presentation.

Histopathologic analysis of a punch biopsy specimen was thought to be consistent with the clinical diagnosis of psoriasis, with a psoriasis area and severity index score of 24.8. Treatment with cyclosporine A 300 mg daily (3.5 mg/kg daily), omeprazole 20 mg daily, and triamcinolone acetonide cream 0.1% was initiated 4 months after presentation. One month later, the initial plaques remained unchanged and hyperkeratotic palmoplantar plaques appeared. Intravenous infliximab 5 mg/kg was started and cyclosporine was withdrawn.

One month later, after 2 infusions of infliximab, acitretin 25 mg once daily was added because of persistent palmoplantar hyperkeratosis. The psoriasiform plaques had become confluent, involving the trunk, scalp, and face, and spared islands of uninvolved skin had become apparent. A 4-mm punch biopsy was performed, which showed a hyperplastic epidermis with alternating hyperkeratosis and parakeratosis with some follicular plugging. In the upper and mid dermis, a dense lymphocytic infiltrate was present (Figure 2). These histopathologic findings were reevaluated and found to be consistent with a diagnosis of pityriasis rubra pilaris (PRP). Because infliximab has been reported to be effective in the treatment of PRP,[1,2] the patient continued infliximab and acitretin therapy. A month later, his rash was cleared, but infliximab was stopped because the patient developed infusion reactions with rash, dyspnea, and bradycardia on the third and fourth infusions, despite premedication with

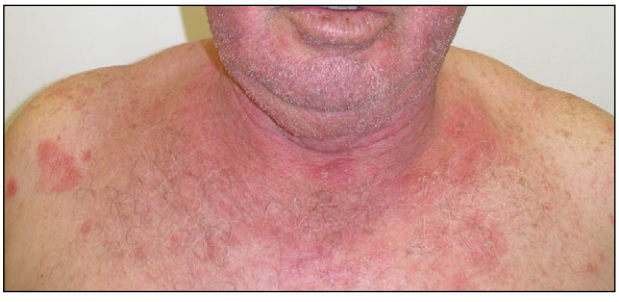

Figure 1. Erythematous and scaly plaques on the neck after 1 session of methyl aminolevulinate photodynamic therapy.

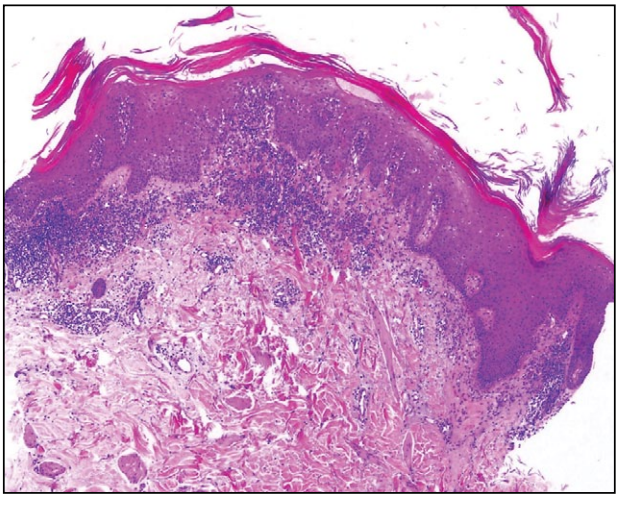

Figure 2. Histology showed a hyperplastic epidermis with hyperkeratosis and parakeratosis. In the upper and mid dermis, a dense lymphocytic infiltrate was present (H&E, original magnification ×100).

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diphenhydramine hydrochloride 50 mg and hydrocortisone acetate 100 mg. One month later, there was a partial relapse with flexural involvement, and etanercept 50 mg once weekly was started in combination with acitretin 25 mg once daily, leading to complete resolution of skin lesions, except for minimal plantar hyperkeratosis.

Photodynamic therapy has been shown to be an effective treatment of actinic keratoses. The most common adverse event is the pain that occurs during light exposure, but excessive photosensitivity may appear in some cases.[3] Pityriasis rubra pilaris is a papulosquamous disease that shares histologic and clinical features with psoriasis, and it occasionally has been reported to be aggravated by UVB therapy.[4]

Photoaggravation, photodistribution, and triggering of PRP has been reported following UV light exposure.[4-6] Although rechallenge was not attempted in our case, we believe the temporal and topographic associations provide substantial support for a likely role of localized PDT in triggering PRP. A report on the strong induction of tumor necrosis factor (TNF) α, IL-1, and IL-6 by PDT might provide a pathogenetic explanation of this phenomenon in susceptible individuals, and generalized psoriasis induced by imiquimod treatment might represent a similar scenario.[7] Nevertheless, anti-inflammatory effects of PDT also have been described.[8,9]

The pathogenesis of PRP is unknown; however, Zhang et al[10] reported a substantial elevation of TNF messenger RNA in the lesional skin of PRP. Thus, TNF-α–blocking agents may be useful for the treatment of photoaggravated PRP, though other causes of widespread papulosquamous eruptions with lymphocytic infiltrates, such as cutaneous lymphoproliferative disorders, should be ruled out prior to initiating therapy with TNF-α inhibitors.

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