Imiquimod Therapy for Elastosis Perforans Serpiginosa

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The Cutting Edge: Challenges in Medical and Surgical Therapeutics

REPORT OF A CASE

A 14-year-old girl was first seen with an 8-month history of an asymptomatic “rash” on her chin and a “wart” on her right hand. At physical examination, she had several pink papules coalescing into annular and arcuate plaques on the central aspect of her chin (Figure 1). She was also noted to have a single flesh-colored verrucous papule on her right palm, which was treated with liquid nitrogen cryotherapy.

A biopsy of her chin was performed to rule out annular verruca plana. This biopsy specimen demonstrated narrow channels in the epidermis filled with orthokeratotic and parakeratotic cells, refractile eosinophilic elastic fibers extending into the papillary dermis, and a few neutrophils (Figure 2). An elastic stain demonstrated an increased concentration of clumped and thickened elastic fibers in the middle dermis extending to the epidermis. These findings were consistent with the clinical diagnosis of elastosis perforans serpiginosa (EPS). After careful review of the patient’s medical history and medication intake and a thorough evaluation for comorbid systemic disorders, she was diagnosed as having idiopathic EPS.

THERAPEUTIC CHALLENGE

Multiple therapies have been described for the treatment of EPS, but often this condition is difficult to manage. Reported treatments include liquid nitrogen cryotherapy, tazarotene gel, and isotretinoin. Laser resurfacing was found to be beneficial in some patients, and cellophane tape stripping is sometimes helpful. However, topical or intralesional corticosteroids and curettage and electrodesiccation are ineffective.

SOLUTION

Our patient was promptly and successfully treated with imiquimod cream applied every night for the first 6 weeks and then 3 times weekly for 4 weeks. She reported some redness of her chin but otherwise tolerated this topical therapy. Clinical improvement was noted after 2 weeks of therapy and complete clearing after 10 weeks (Figure 3). Imiquimod therapy was discontinued, and the patient’s skin remained clear 3 months later.

COMMENT

In EPS, altered elastic fibers are recognized as foreign material and are extruded through the epidermis by transepidermal eosinophilic elastic fibers (hematoxylin-eosin, original magnification ×10).
Imiquimod functions as an immune system modulator, with stimulation of the T<sub>γ</sub>1 response. It up-regulates the generation of interferon γ, and interleukin 12, among others, while inhibiting the production of interleukin 4 and interleukin 5. In addition, imiquimod stimulates Langerhans cell migration to lymph nodes, allowing increased antigen presentation, also contributing to modulation of the immune response. The mechanism of action of imiquimod in the treatment of EPS is unclear. Perhaps the transepidermal elimination in EPS is secondary to an antigenic change in the elastic fibers, and imiquimod causes a T-cell response that clears the antigen. Although not previously described, imiquimod therapy is a swift and easy way to manage EPS with minimal irritation. Randomized double-blind, placebo-controlled trials of patients with EPS treated with imiquimod would be helpful in confirming this outcome.

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