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Letter to the Editor

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Lichen striatus successfully treated with oral cyclosporine

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Abstract

Lichen striatus is an acquired, benign, linear inflammatory dermatosis characterized by a sudden skin eruption along Blaschko's lines that usually is not associated with specific etiologic agents. In most cases, it is a self-limited dermatosis, but may relapse. Topical steroids are its first-line therapy, but this treatment is not always effective. We describe the case of a 45-year-old woman affected by a lichen striatus on her right limb resistant to topical corticosteroid therapy. The patient was successfully treated with cyclosporine (4 mg/kg/die) for 4 weeks with no recurrence of the dermatitis during the subsequent 1-year follow-up period.

Keywords

corticosteroids, cyclosporine, lichen striatus

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Lichen striatus (LS) is an acquired, benign, linear inflammatory dermatosis characterized by a sudden eruption of asymptomatic small, flat-topped, lichenoid, scaly papules along Blaschko's lines. The skin lesions are frequently localized on the limbs, distributed as a continuous or interrupted linear band of pink, red, tan, or skin-colored papules.[1,2] It often occurs in children, most commonly in 2- to 3-year-old, although it is infrequently observed also in adults.[2] Digital involvement may lead to onycholysis, longitudinal ridging, splitting, and nail loss.[3] Although LS is not associated with specific etiologic agents, it could be triggered by infections, trauma, hypersensitivity reactions, vaccines, medications, and pregnancy.[1] In most cases, LS is a self-limited dermatosis and resolves within 1 year, but it may relapse. Topical steroids are first-line therapy for LS, but this treatment is not always effective and prolonged use of corticosteroids may be associated with adverse effects such as cutaneous atrophy.[1] Cases of LS unresponsive to topical steroids have been successfully treated with other therapies such as oral acitretin[1] and photodynamic therapy.[4] We describe the case of an atopic 45-year-old woman presented with a 1-year history of an asymptomatic linear skin eruption on the right limb (Figure 1). During this period of time, the lesions had been treated unsuccessfully with mometasone furoate cream 0.1% for three cycles of treatment (once daily for 2 weeks). Skin examination revealed erythematous and skin-colored papular lesions along the right lower limb following Blaschko's

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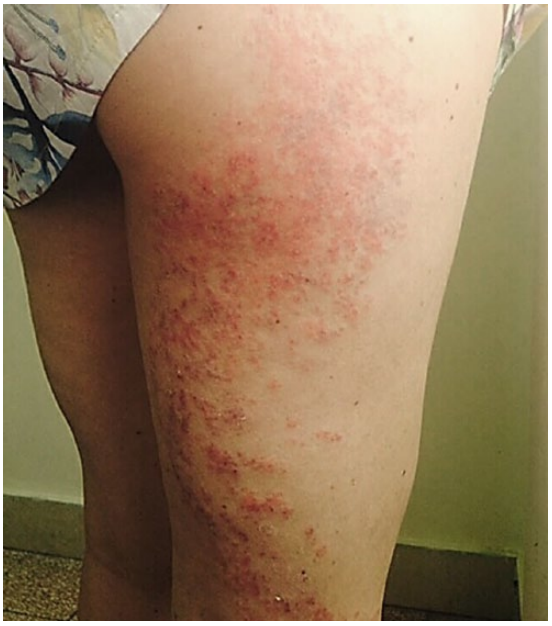

Figure 1. Asymptomatic linear skin eruption on the right lower limb of the patient.

lines. No other lesions in any other site was observed. Histopathologic examination of one of these lesions revealed a predominant pattern of interface dermatitis consisting of a dense band-like perivascular inflammatory infiltrate, also round appendages (Figure 2), composed of histiocytes and T lymphocytes CD3 (PanT) and CD8. Hyperparakeratosis and focal spongiosis of epidermis were also observed. On the basis of the histopathological findings, suggestive for LS,[5] since little improvement had been obtained with mometasone furoate cream, and after receiving an informed consent by the patient, we decided to administer systemic therapy with cyclosporine (4 mg/kg/die). The LS regressed completely after only 4 weeks of therapy. No drug side effects and no recurrence of LS were observed during the subsequent 1-year follow-up period.

LS is in most cases an asymptomatic and self-limited dermatosis but may resolve leaving post-inflammatory hyper or hypopigmentation causing significant psychological distress for the patient. Moreover, it can relapse and be resistant to topical corticosteroid therapy.[6] In our case, the patient was resistant to topical steroid treatment while she had a complete response to oral cyclosporine. Although the pathogenesis of LS remains unclear, it has been considered a CD8+ T-cell-mediated inflammatory reaction with a cytotoxic reaction directed toward mutated post-zygotic skin cell.[7] We may speculate that the therapeutic efficacy of cyclosporine is due to its immunomodulatory activity. This treatment approach could reduce morbidity and prevent complications of LS.

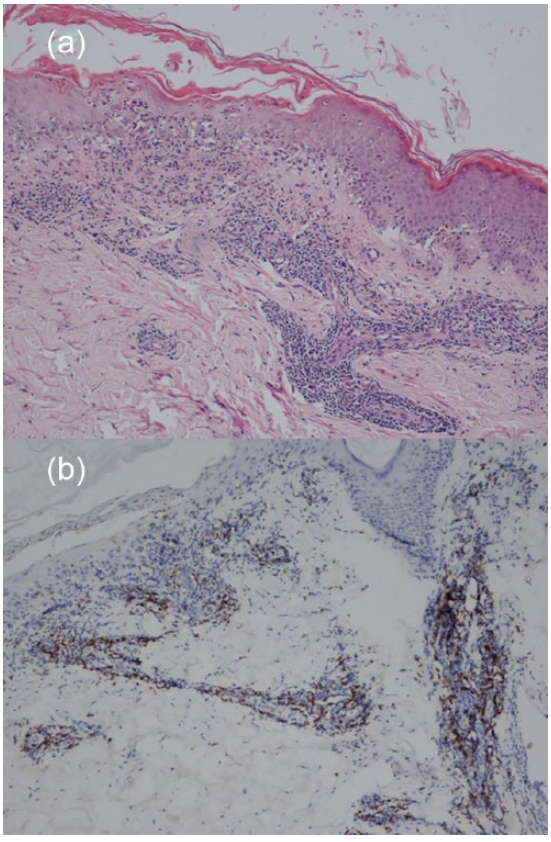

Figure 2. (a) A dense perivascular inflammatory infiltrate focally with the features of the interface dermatitis (hematoxylin–eosin, 100× original magnification) and (b) most of the perivascular inflammatory cells show intense immunohistochemical expression of CD8 (100× original magnification).

Declaration of conflicting interests

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