Lichen striatus associated with psoriasis vulgaris treated with oral acitretin

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ABSTRACT
Lichen striatus (LS) is an uncommon dermatosis of unknown etiology that presents as a continuous or interrupted linear band of pink, tan, red or skin-colored papules in a blaschkoid distribution. The lesions are generally solitary and unilateral, but unusual extensive cases with multiple and bilateral lesions have been also described. Albeit LS is typically an asymptomatic and self-limited dermatosis, it may cause a significant psychological distress in some patients, thus requiring an appropriate therapy. Topical steroid is the most commonly used treatment but it is not always effective. We report a case of LS unresponsive to topical steroid therapy associated with psoriasis vulgaris successfully treated with oral acitretin.

Key words: Acitretin, lichen striatus, psoriasis, therapy, treatment

INTRODUCTION
Lichen striatus (LS) is an uncommon, asymptomatic, self-limiting dermatosis which lasts an average of nine months and usually occurs in children, although it is also rarely seen in adults. It often arises without a clear trigger, but some cases would appear to be related to several possible precipitating events such as infections and, more rarely, cutaneous injury, trauma, hypersensitivity, or other unspecified factors.[1] LS typically presents as a continuous or interrupted linear band of pink, tan, red, or skin-colored papules that follow Blaschko’s lines,[1,2] which represent the pathways of cutaneous cell migration during embryonic development.[3] The lesions are generally solitary and unilateral, but unusual cases with multiple and bilateral lesions have been rarely reported.[1,2] Topical steroid is the most commonly used medication for the treatment of LS. However, this therapy is not always effective and its prolonged use may be associated with certain adverse effects including cutaneous atrophy.[4] Other anecdotal therapies include oral corticosteroid, photodynamic therapy, topical calcineurin inhibitors, and oral acitretin.[4,5]

We report a case of LS unresponsive to topical steroid therapy associated with psoriasis vulgaris, successfully treated with oral acitretin.
cyclosporine in the past with poor tolerance, we decided to start oral acitretin (0.5 mg/kg/day). Interestingly, we found that LS regressed completely and rapidly after only four weeks [Figure 2b]. The patient continued therapy with acitretin at the same dosage for other two weeks; thereafter, the drug was tapered over the subsequent four weeks due to resolution of psoriatic lesions. No recurrence of LS and psoriasis was observed during the subsequent four-month follow-up period.

**DISCUSSION**

The association between LS and psoriasis is a rare event, since, to the best of our knowledge, there are only two well-documented published reports in the literature. The first case was a 2-year-old boy with a LS localized to his left half of the body who developed an unusual form of unilateral eruptive psoriasis limited to the right half of the body 2 weeks later. The authors stressed that a common, but unknown, triggering factor may have been involved. The second report described a 58-year-old man suffering from plaque psoriasis who developed LS at the third session of narrow-band ultraviolet B phototherapy instituted for a psoriatic exacerbation that had manifested 5 months earlier. In this case, the authors hypothesized that the association between two dermatoses was a mere coincidence. We believe that also in our case the association between the two dermatoses is casual, since a wide time gap between the appearance of each.

Although LS is an asymptomatic and self-limited dermatosis, it may cause a significant psychological distress in some patients, thus requiring an appropriate therapy. Several treatments have been reported with various degrees of success including oral and topical corticosteroid, photodynamic therapy, topical calcineurin inhibitors, and oral acitretin. Regarding this last therapy, there is only one report describing an extensive case of LS markedly improved over the course of a few weeks with an attack dose of 0.6 mg/kg/die.

In our case of LS, the patient was found to be resistant to topical steroid treatment while she had a complete and rapid response to oral acitretin. The exact mechanism of action of this drug in LS is unclear.

LS has been considered to be the consequence of an acquired stimulus that induces a loss of immune tolerance to embryologically abnormal clones, resulting in a T-cell-mediated inflammatory reaction, which causing the typical Blaschko linear lesions histopathologically characterized by lichenoid, lymphocytic infiltrate with overlying epidermal acanthosis, dyskeratosis, hyperkeratosis, occasional parakeratosis, and lymphocytic exocytosis.

Therefore, it can be assumed that therapeutic efficacy of acitretin is attributable to its effect on cutaneous immunomodulation as well as epithelial cell proliferation and differentiation.

In conclusion, the clinical improvements observed in this report tend to further support the efficacy of oral acitretin in the treatment of LS and suggest its use not only in extensive and unaesthetic forms but also in cases unresponsive to steroid therapy. Further studies and reports are however needed in order to confirm these findings.

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