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Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Saboraud R. Manuel élémentaire de dermatologie topographique régionale. Paris: Masson and Cie; 1905; p. 197.
2. Trakimas CA, Sperling LC. Temporal triangular alopecia acquired in adulthood. *J Am Acad Dermatol.* 1999;40:842-4.
3. Elmer KB, George RM. Congenital triangular alopecia: a case report and review. *Cutis.* 2002;69:255-6.
4. Akan IM, Yildirim S, Avci G, Aköz T, Karadayi N. Bilateral temporal triangular alopecia acquired in adulthood. *Plast Reconstr Surg.* 2001;107:1616-7.
5. Ruggieri M, Rizzo R, Pavone P, Baieli S, Sorge G, Happel R. Temporal triangular alopecia in association with mental retardation and epilepsy in a mother and daughter. *Arch Dermatol.* 2000;136:426-7.
6. Trakimas C, Sperling LC, Skelton HG III, Smith KJ, Buker JL. Clinical and histologic findings in temporal triangular alopecia. *J Am Acad Dermatol.* 1994;31:205-9.
7. Kenner JR, Sperling LC. Pathological case of the month. Temporal triangular alopecia and aplasia cutis congenita. *Arch Pediatr Adolesc Med.* 1998;152:1241-2.
8. Barrera A. The use of micrografts and minigrafts in the aesthetic reconstruction of the face and scalp. *Plast Reconstr Surg.* 2003;112:883-90.
9. Haber RS, Stough DB. *Trasplante de pelo.* Madrid: Elsevier; 2007.
10. Bargman H. Congenital temporal triangular alopecia. *Can Med Assoc J.* 1984;131:1253-4.

Lichen Planus and Lichen Striatus: Opposite Ends of the Same Spectrum?

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To the Editor:

Lichen planus is an inflammatory dermatosis with an immunological pathogenesis that is not yet fully understood. It is characterized by an eruption consisting of pruritic violaceous polygonal papules that can affect the trunk or limbs and that may often have whitish linear striae, called Wickham striae, on their surface. Involvement of the mucosae is common.¹

Lichen striatus is an asymptomatic dermatosis that generally occurs in childhood. It consists of small, scaly, erythematous papules of 1 to 2 mm in diameter, occasionally with a vesicular component, distributed in a linear band along the Blaschko lines. The lesions are usually distributed unilaterally on a limb and, unlike those of lichen planus, usually leave a residual hypopigmentation.¹ Bilateral or multiple lesions are very rare.^{2,3}

We present a case that started as a linear eruption and subsequently became generalized.

The patient was a 7-year-old girl weighing 20 kg and with a history of celiac disease. She was seen for a pruritic linear eruption that had appeared 3 months earlier on the left upper limb, with involvement of the ipsilateral thumb nail. There was no fever or other clinical symptoms. The condition was diagnosed as lichen striatus, and we prescribed treatment with topical corticosteroids (0.1% methylprednisolone aceponate in a once-daily application).

After a month of topical treatment the lesions were no longer linear and localized, but had spread irregularly over the body. No mucosal lesions were detected. The lesions were shiny violaceous erythematous papules with a lichenoid appearance (Figures 1 and 2) that coalesced to form plaques spread randomly over the trunk, legs, and lumbar region. Some, such as those affecting the antecubital fossae, showed a degree of desquamation.



Figure 1. Erythematous violaceous papules with a lichenoid appearance.



Figure 2. Nail dystrophy on the left thumb.

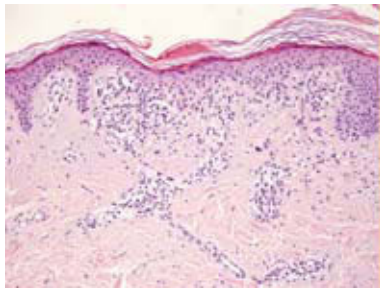


Figure 3. Focal parakeratosis and lymphocytic infiltrate involving the glands and the dermal-epidermal junction. Hematoxylin-eosin, original magnification $\times 10$.

The cubital side of the left thumb nail was dystrophic and had longitudinal striations (Figure 2). The face and popliteal fossae were affected, as were the soles of both feet. There was also extremely intense erythema and scaling on the scalp. A skin biopsy taken from the arm showed a lymphocytic infiltrate at the dermal-epidermal junction with vacuolar degeneration of the basal layer and a deep perifollicular and glandular infiltrate (Figure 3). In this first biopsy, the lesions were both clinically and histologically consistent with lichen striatus. In a second skin biopsy from the lumbar region, orthokeratotic hyperkeratosis, wedge-shaped hypergranulosis, acanthosis, papillomatosis, a bandlike lymphocytic infiltrate at the dermal-epidermal junction, pigmentary incontinence, and colloid bodies were observed. The infiltrate also extended towards deep glandular structures. High-dose (1 mg/kg/d) prednisone therapy was initiated, with no response after 3 weeks. In view of the inefficacy of corticosteroid therapy and the severe scalp involvement (with the consequent risk of scarring alopecia), we decided to initiate treatment with cyclosporine at 2.5 mg/kg/d and obtained a clear improvement of all the lesions, including the nail dystrophy, in 6 weeks.

Lichen planus and lichen striatus are entities that are well-defined clinically, although there may be cases in which the differential diagnosis is not really clear.

Generally, lichen striatus shows a linear distribution along the Blaschko lines,³⁻⁵ while lichen planus usually presents as papules or plaques that only occasionally

exhibit a linear distribution.⁶ The differential diagnosis between linear lichen planus and lichen striatus involves very subtle clinical distinctions. For this reason histology is necessary, although given the similarity between the 2 conditions, it is not always diagnostic.⁶ The absence of pruritus, a residual hypopigmentation, and a tendency to resolve spontaneously support the diagnosis of lichen striatus.¹ In our patient the initially linear eruption became generalized, though with no apparent trigger; there was pruritus, residual pigmentation, and no clear evidence of spontaneous resolution.

Histologically, lichen planus usually shows orthokeratotic hyperkeratosis, hypergranulosis, irregular sawtooth acanthosis, vacuolar degeneration of the basal layer of the epidermis, a bandlike infiltrate, apoptosis, pigmentary incontinence, and Civatte bodies.¹ The histology in lichen striatus varies somewhat depending on the stage of the disease. A dense and occasionally bandlike lymphohistiocytic perivascular infiltrate can usually be seen. Epidermal hyperkeratosis with focal parakeratosis is a typical finding in lichen striatus but rare in lichen planus. The infiltrate can extend as far down as the eccrine glands and ducts. Such a finding can help differentiate lichen striatus from lichen planus. However, the histologic variability of lichen striatus is considerable and the findings may sometimes be indistinguishable from those of lichen planus.¹

Cases of overlap between these 2 entities have been reported in the literature.

Herd et al⁶ described the case of a 37-year-old woman with an asymptomatic and spontaneously resolving linear eruption. Histologically, the condition was consistent with lichen planus and, in addition, showed occasional plasma cells in the papillary dermis. Rubio et al⁷ described the case of a 29-year-old woman with an asymptomatic and spontaneously resolving linear eruption that left residual hyperpigmentation. Histology showed acanthosis, hypergranulosis, basal layer degeneration, colloid bodies, and a bandlike lymphomononuclear infiltrate, more consistent with the microscopic diagnosis of lichen planus. Numata et al⁸ reported the case of a 21-year-old man who developed symptoms of linear lichen planus, but with histologic involvement of the eccrine sweat glands, following the intramuscular injection of triamcinolone acetonide.

In our patient, the first biopsy (obtained from the initial lineal eruption) was consistent with lichen striatus, given the presence of parakeratosis and a deep glandular infiltrate. The second biopsy (taken from one of the generalized lesions), however, showed epidermal changes that were more characteristic of lichen planus.

In view of the intense involvement of the scalp and the lack of response to oral corticosteroid therapy, we decided to initiate treatment with cyclosporine. The result was spectacular and we were thus able to prevent scarring alopecia.

We present a case that had clinical and histologic characteristics both of lichen planus and of lichen striatus. This is exceptionally common, and supports the hypothesis that these 2 diseases represent the opposite ends of the same disease spectrum.

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Conflicts of Interest

The authors declare no conflicts of interest.

References

1. Tilly JJ, Drolet BA, Esterly NB. Lichenoid eruptions in children. *J Am Acad Dermatol.* 2004;51:606-24.
2. Aloï F, Solaroli C, Pippione M. Diffuse and bilateral lichen striatus. *Pediatr Dermatol.* 1997;14:36-8.
3. Mittal R, Khaitan BK, Ramam M, Verma KK, Manchanda M. Multiple lichen striatus—An unusual presentation. *Indian J Dermatol Venereol Leprol.* 2001;67:204.
4. Peramiqnel L, Baselga E, Dalmau J, Roé E, del Mar Campos M, Alomar A. Lichen striatus: clinical and epidemiological review of 23 cases. *Eur J Pediatr.* 2006;165:267-9.
5. Taniguchi Abagge K, Parolin Marinoni L, Giraldi S, Carvalho VO, de Oliveira Santini C, Favre H. Lichen striatus: description of 89 cases in children. *Pediatr Dermatol.* 2004;21:440-3.
6. Herd RM, McLaren KM, Aldridge RD. Linear lichen planus and lichen striatus—opposite ends of a spectrum. *Clin Exp Dermatol.* 1993;18:335-7.
7. Rubio FA, Robayna G, Herranz P, de Lucas R, Hernández-Cano N, Contreras F, et al. Linear lichen planus and lichen striatus: is there an intermediate form between these conditions? *Clin Exp Dermatol.* 1997;22:61-2.
8. Numata Y, Okuyama R, Tagami H, Aiba S. Linear lichen planus distributed in the lines of Blaschko developing during intramuscular triamcinolone acetonide therapy for alopecia areata multiplex. *J Eur Acad Dermatol Venereol.* 2008;22:1370-1.

Unilateral Allergic Contact Dermatitis of the Eyelid Caused by Iopimax

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To the Editor:

Apraclonidine is a drug belonging to the group of α -2 adrenergic agonists; it is widely used as short-term additional treatment for chronic glaucoma in patients receiving medical treatment and who require additional reduction of intraocular pressure in order to delay treatment with laser or surgery. The drug is also widely used to control pressure within the eye after glaucoma surgery.

We report the case of a 63-year-old woman with a history of hypertension on treatment with Ameride (amiloride plus hydrochlorothiazide); the woman suffered from long-term glaucoma in the left eye for which she had been receiving treatment for several years with Xalatan ophthalmic solution (latanoprost, 50 mg). Despite this treatment, the last ophthalmic check-up, performed in early 2007, revealed high intraocular pressure and treatment with Iopimax (apraclonidine, 0.5%) ophthalmic solution was therefore added. The main objective of this additional treatment was to control intraocular pressure, as well as to delay surgery because the patient had already undergone surgery for glaucoma in the right eye. The patient consulted for an 8-month history of progressive and recurrent appearance of intensely pruritic, eczematous

lesions on the upper and lower eyelids of the left eye; the lesions had developed after starting treatment with Iopimax and had improved substantially following application of different topical corticosteroids, although they recurred when corticosteroid treatment was suspended.

Examination of the skin revealed a small erythematous plaque with fine desquamation, located on each eyelid; desquamation was more accentuated on the internal surface (Figure 1). Laboratory tests (general analysis, immunologic study, and thyroid hormones) revealed no



Figure 1. Eczematous lesions on the upper and lower left eyelids.