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Clinics in Dermatology

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Edited by Stephen P. Stone, MD

Lichen striatus after COVID-19

To the Editor:

Numerous cutaneous manifestations have been reported in association with COVID-19 infections, and some insight into their pathogenesis has been gained.[1,2] Among COVID-19–linked immune dermatologic conditions, lichen striatus (LS) was described after COVID-19 vaccination but never after COVID-19 infection.[3,4]

Four patients

We encountered four cases of LS post–COVID-19 in children. All of them were diagnosed clinically, and the diagnoses were histopathologically verified. COVID-19 was confirmed by molecular testing of viral RNA on nasopharyngeal swab. None of the patients was vaccinated against COVID-19. Three of the four children were girls, with a mean age of 12.5 years, in line with published data reporting LS especially in girls (75%) 5 to 15 years of age.[5]

Two patients were seen during summer 2022 with acute LS, shortly after having COVID-19. We observed characteristic lichenoid, erythematous, flat-topped papules forming a thin unilateral band along Blaschko lines of the lower extremities. These were chronic, hypopigmented lesions in linear distribution. There was also longitudinal nail ridging in one patient (Figure 1). Such characteristic clinical features suggested the diagnosis of LS. Histologic study confirmed the diagnosis by revealing lymphocytes surrounding subpapillary vessels, as well as hyperkeratosis and parakeratosis, a few necrotic keratinocytes, intercellular edema, and spongiosis (Figure 2). We prescribed topical corticosteroids and emollients for 3 months, after which the inflammation subsided with the lesions becoming flatter and hypopigmented.

The other two patients contracted COVID-19 during spring 2022, more than 3 months before presentation at the dermatologic unit. At the time of the dermatologic examination, there were only chronic lesions and no erythematous acute lesions. The lesions were hypochromic, with blaschkoid distribution, on both the arms and legs (Figure 3).

Histology confirmed the clinically established diagnosis, showing a lichenoid infiltrate, focal hyper-and-parakeratosis and periadnexial inflammatory lymphoplasmacytic infiltrate (Figure 4).

The eruption in the fourth case was prevalently hypopigmented from the beginning, suggesting lichen striatus albus (LSA). LSA has been described in literature as a possible initial LS manifestation.[5] It may begin with a hypopigmented maculopapular eruption and presents only a few characteristic red-lichenoid papules. In both LS and LSA, the eruption spontaneously regresses after 6 to 24 months, but recurrences may occur, mainly in the form of hypopigmented macules.[5]

Discussion

Characteristically, LS is usually unilateral and is typically located on the extremities, and less frequently, on the trunk or face. Rarely, the nails are involved, with thinning, onychodystrophy, onycholysis, and longitudinal ridging.[5] The present series is in line with the available literature in which arms and legs were involved in 100% of patients and nails in 25%.

The differential diagnoses of LS include linear lichen planus, which is characterized by intensely pruritic, violaceous, hyperkeratotic papules arranged in thick bands, and inflammatory linear verrucous epidermal nevus, which is characterized by markedly pruritic congenital lesions. In contrast with linear lichen planus and inflammatory linear verrucous epidermal nevus, the present lesions were acquired, being almost asymptomatic, flat-topped, and just slightly erythematous in the acute phase, with a hypopigmented evolution, forming a thin band of maximum 1.5-cm width along the lines of Blaschko.[6]

LS has an unknown etiology. In the literature, the occurrence of LS has been reported after such viral stimuli as influenza, varicella, and vaccinations against hepatitis B, yellow fever, and COVID-19.[4] It is assumed that an acquired stimulus, such as a viral infection or vaccination, may induce a loss of immunotolerance toward mosaic keratinocyte clones that undergo cytotoxic T cells' attack inducing LS.[4] Notably, in this series, COVID-19 was temporally close to

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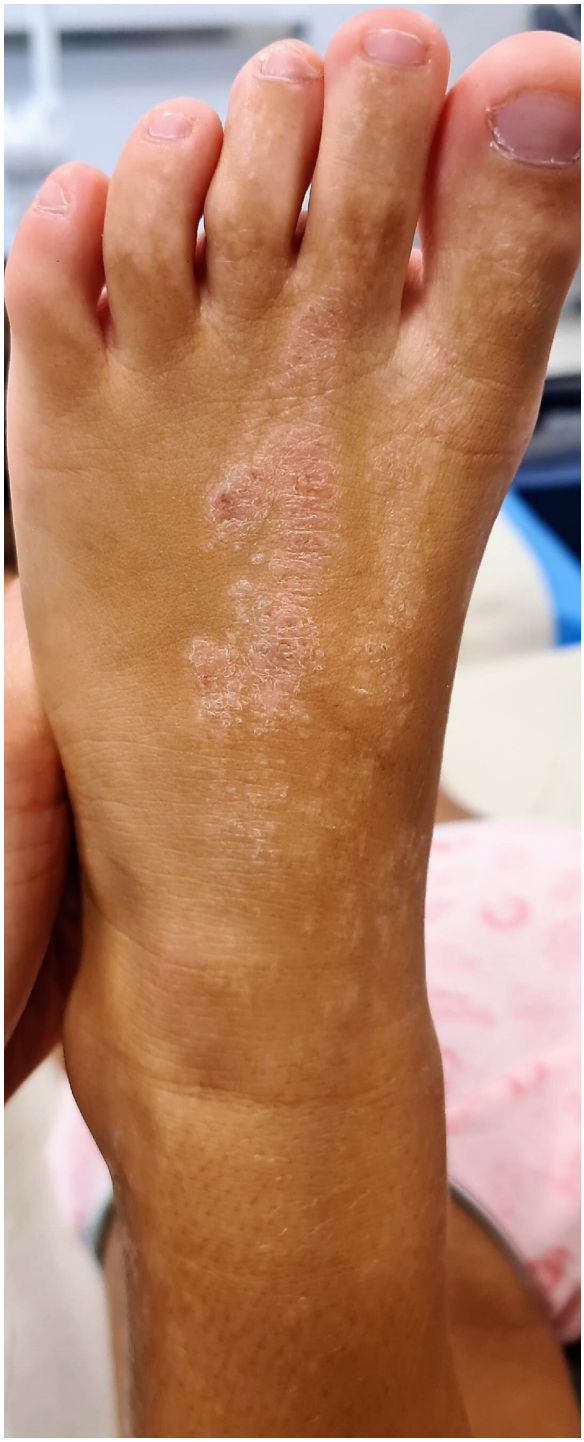

Fig. 1 Flat-topped erythematous papules forming a continuous thick band along Blaschko lines on the patient's shin, dorsum of the foot, and first three toes, also involving the nail of the third toe, causing longitudinal ridging.

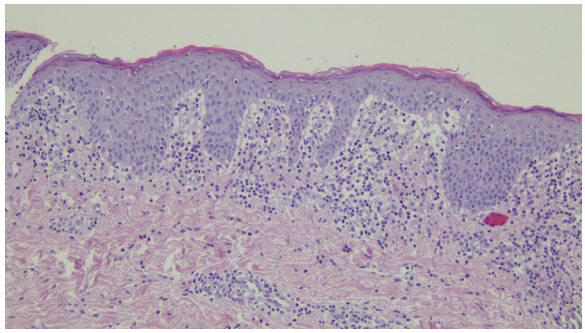

Fig. 2 Biopsy showed a pattern of interface dermatitis with basal layer vacuolization, superficial lymphoplasmacytic inflammatory infiltrate, mainly around subpapillary vessels and intraepidermal exocytosis and edema (histologic section in hematoxylin and eosin).

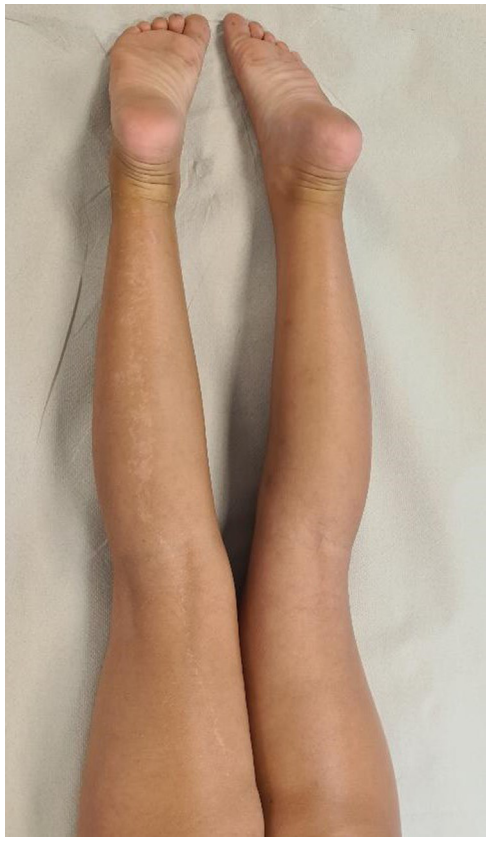

Fig. 3 Hypopigmented, small, monomorphic, flat-topped papules, forming a band with blaschkoid distribution on the lower limb.

the development of LS, suggesting a possible role of the infection in triggering autoimmunity.

The same process is supposed to take place after COVID-19 vaccination, which has been seen with LS. Cross-reactivity to viral antigens, inducing a pathogen-specific immune response that targets also mosaic host's keratinocytes, has been suggested as the pathogenic mechanism.[4]

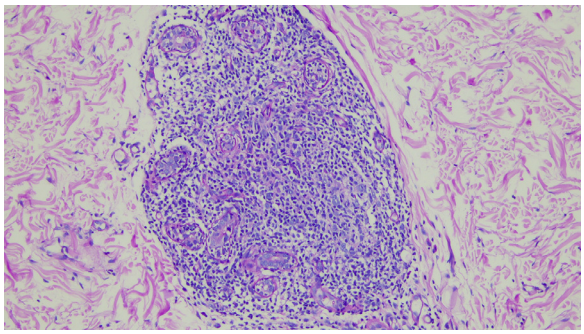

Fig. 4 A periadnexial inflammatory lymphoplasmacytic infiltrate can be observed (histologic section stained by Periodic Acid Schiff (PAS) histochemical technique).

Conclusions

COVID-19 infection has been widely documented in the past, allowing the possibility of identifying associations with immune-mediated cutaneous manifestations.[1-4] Possibly, lichen striatus, as well as other COVID-19 associated autoimmune phenomena, may be virally triggered. An increased awareness toward this particular infection may suggest an otherwise underestimated association.

Conflict of interest

The authors declare no conflict of interest.

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