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Case Report

Coexistence of nail psoriasis and cutaneous lichen planus in a single patient: An uncommon entity

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ABSTRACT

Psoriasis and lichen planus (LP) are common, routinely encountered autoimmune disorders. However, their coexistence is relatively uncommon. Both diseases have an autoimmune and complex genetic background. There are few case reports on the coexistence of LP and psoriasis vulgaris. However, to the best of our knowledge, there are no case reports on the coexistence of nail psoriasis and cutaneous LP. Many speculations have been put forward about the pathogenesis of their coexistence in a single patient. However, certain genetic predispositions, T-cell-mediated autoimmunity, and true Koebner phenomenon play a significant role in disease development. We present the details of a patient with isolated nail psoriasis who subsequently developed cutaneous and oral LP. Awareness of such a possibility can help avoid misdiagnosis in clinical practice.

Keywords: Nail psoriasis, Cutaneous lichen planus, Oral lichen planus, Autoimmune, Coexistence

INTRODUCTION

Psoriasis is a chronic inflammatory disease; nail involvement being seen in around 80–90% of patients.[1] It can be due to genetic predisposition or certain environmental triggers, including infections, drugs, and psychological stress. The coexistence of psoriasis and lichen planus (LP) is rarely reported.[2,3] Although the etiopathogenesis of both diseases is not completely understood, autoimmunity and genetic predisposition play a major role in development of both. Both are common cutaneous inflammatory diseases, where a plethora of altered cytokine expression and T-cell-mediated autoimmunity play a pivotal role in their pathogenesis. With significant differences in their aetiology, presentation, histology, and immunology, the coexistence of psoriasis and LP is rarely described.[2-4] However, to the best of our knowledge, there are no case reports on the coexistence of isolated nail psoriasis and cutaneous LP. We present the clinical details of a patient with isolated nail psoriasis with subsequent development of cutaneous LP.

CASE REPORT

A 42-year-old female, a teacher by occupation, presented to our dermatological outpatient department with complaints of discoloration, thickening and difficulty in cutting her fingernails and toenails for 2 years. She had also noticed the development of multiple skin-coloured raised, itchy lesions over the legs and feet for 2 months. These lesions were not associated with any pain, scaling, or bleeding. The patient had hypothyroidism and hypertension, and she was on thyroxine100 µg once a day and amlodipine 5 mg twice a day, respectively, for 5 years.

On cutaneous examination, multiple, discrete hyperpigmented to violaceous papules were present over the legs and feet [Figure 1a]. Palms, soles, and scalp were normal. Mucosal examination revealed a whitish lacy reticular patch in the right retromolar trigone region of buccal mucosa [Figure 1b]. On examination of nails, her fingernails showed Grade 2 onycholysis with a regular edge [Figure 2a], yellowish discoloration of the nail plate with pitting and splinter haemorrhages. The toenails showed pachyonychia, Grade 2 onycholysis with a regular edge, splinter haemorrhages, pincer nail morphology [Figure 2b] with compact subungual hyperkeratosis. There were multiple hyperpigmented to violaceous papules present involving the nail folds.

Dermoscopy (Dinolite AM7515MZT USB dermatoscope, Anmo Corporation, Taiwan) revealed transversal onycholysis

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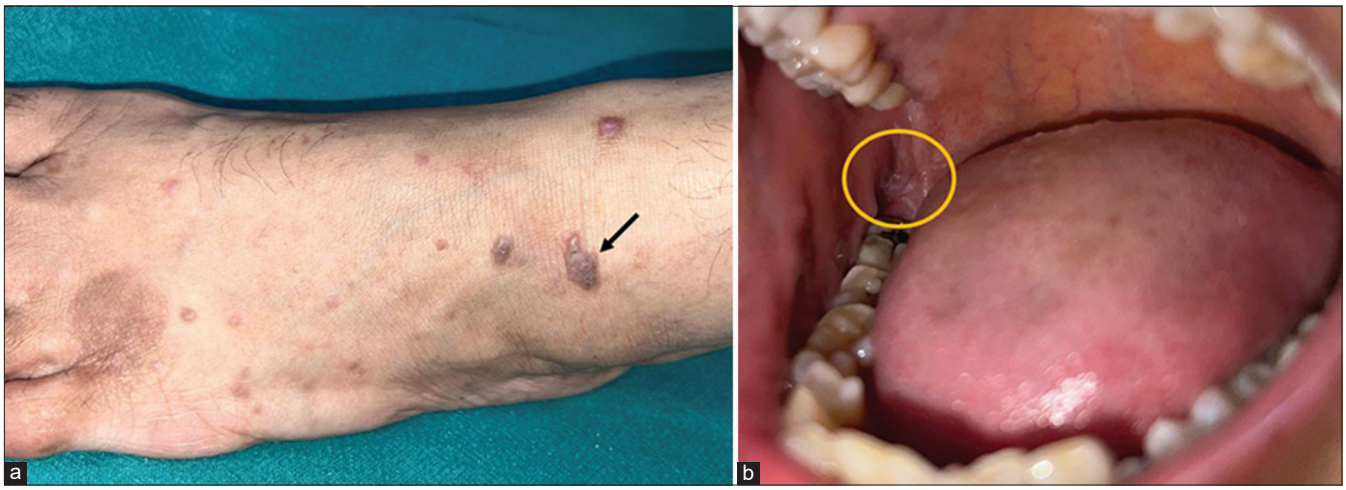

Figure 1: (a) Clinical image showing multiple hyperpigmented to violaceous discrete papules (black arrow) over dorsum of foot and ankle. (b) White lacy reticular patch (yellow circle) in the right retromolar trigone region of the buccal mucosa.

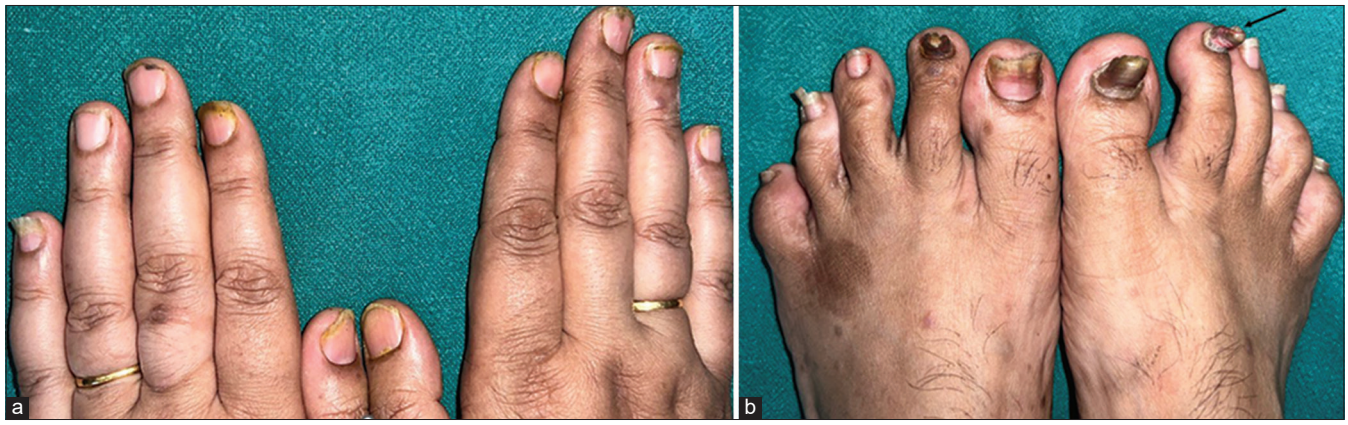

Figure 2: (a) Clinical image showing onycholysis with proximal erythematous border in multiple fingernails. (b) Multiple toenails with pachyonychia, yellowish-brown discoloration of the nail plate and onycholysis with massive subungual hyperkeratosis (black arrow).

[Figure 3a] with a proximal erythematous rim, pitting. Additionally, there were multiple splinter haemorrhages [Figure 3b], dilated vessels, and presence of salmon patch [Figure 3c] in the nail bed. Massive subungual compact hyperkeratosis was visualised in the hyponychial view.

The patient's routine haematological and biochemical parameters were normal. Under local anaesthesia, a 4 mm skin biopsy was taken from the leg lesion. On histopathology, hyperkeratosis, hypergranulosis, basal layer vacuolar degeneration, and a band of lymphocytic infiltrate [Figure 4a] were seen in the dermis. Clippings from the nail plate, subjected to direct microscopy using 10% potassium hydroxide and culture on Sabouraud's dextrose agar (SDA) medium with cycloheximide, failed to reveal any fungal elements. A nail biopsy from the left great toenail was processed for histopathological examination and periodic acid-Schiff (PAS) staining. Histopathology revealed hyperkeratosis, parakeratosis, hypogranulosis, extensive neutrophilic infiltrate, and regular elongation of rete ridges. PAS staining did not reveal any fungi [Figure 4b].

Based on clinical examination and investigations, a diagnosis of nail psoriasis with cutaneous lichen planus (LP) was confirmed and the patient was started on topical steroids (mometasone 0.1% ointment) and tacrolimus (0.1% ointment) for LP lesions. A potent topical steroid (clobetasol 0.005%) with calcipotriol ointment was advised under occlusion for nail psoriasis. The patient did not consent for starting any systemic or injectable therapy for nail psoriasis.

DISCUSSION

Common skin conditions such as psoriasis and LP affect 2–3% and 1% of the population, respectively.[3] However, their coexistence in a single patient has been rarely seen, probably due to under-reporting. In a retrospective analysis, Poljacki *et al*. [5] identified five LP patients out of 1743 cases of

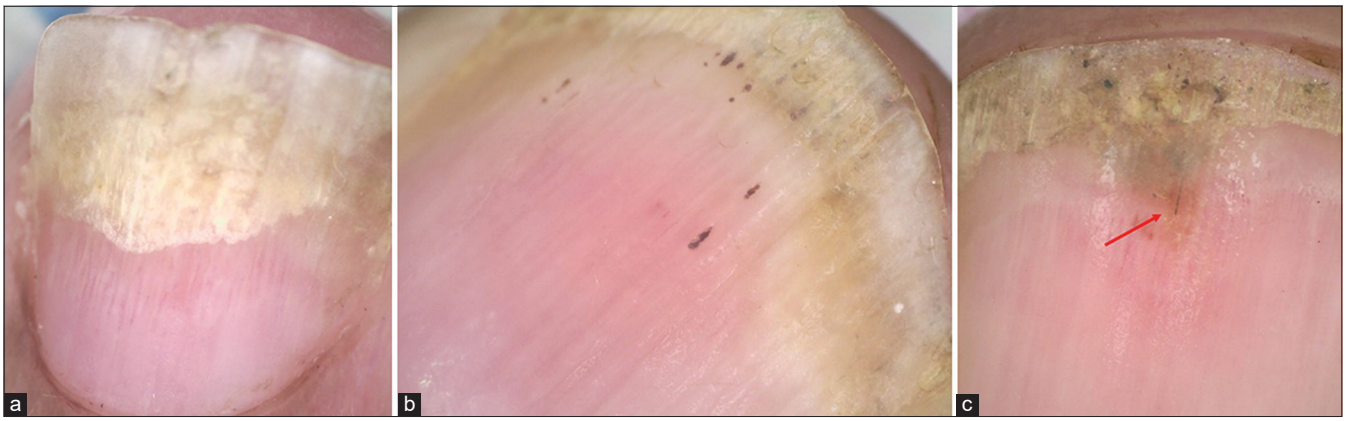

Figure 3: (a) Onychoscopic image showing linear transversal onycholysis with proximal erythematous border and multiple dilated vessels in the nail bed. (b) Multiple splinter haemorrhages in the nail bed. (c) Salmon patch (red arrow) in the nail bed (Dinolite AM7515MZT, polarised ×90).

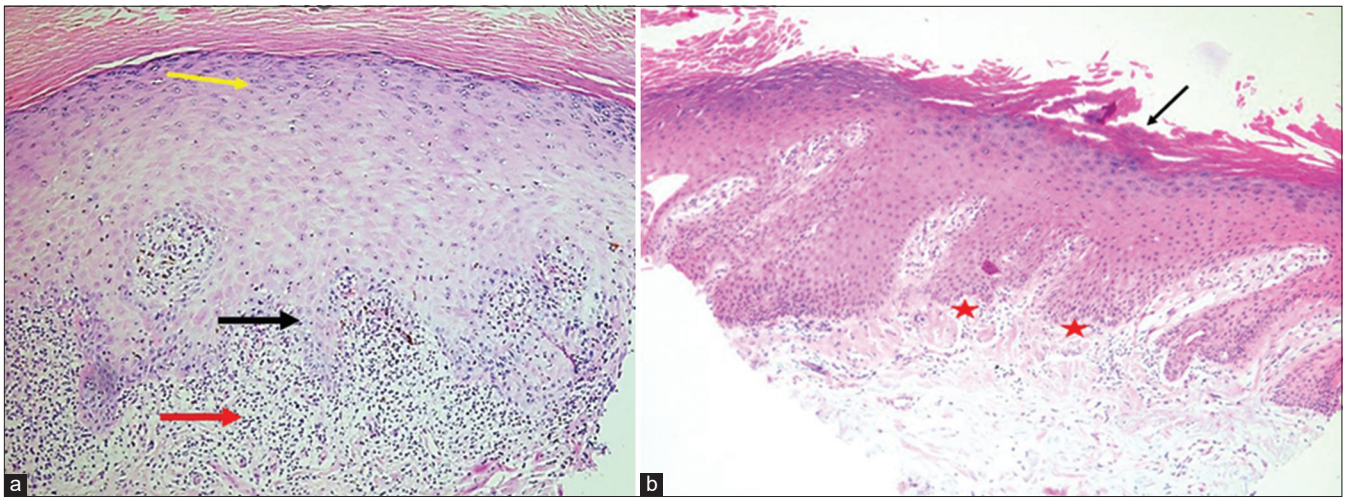

Figure 4: (a) Skin biopsy showing hyperkeratosis, hypergranulosis (yellow arrow), basal layer degeneration (black arrow) and a band of lymphocytic infiltration (red arrow) in the dermis (Haematoxylin and Eosin [H&E], ×400). (b) Nail bed biopsy showing psoriasiform hyperplasia of nail bed epithelium with hyperkeratosis and parakeratosis (black arrow) and regular elongation of rete ridges (red star) (H&E, ×400).

psoriasis. Naldi *et al*.[6] reported 711 cases of LP in the Italian population, of which 12 had psoriasis.

Psoriasis is a T-cell-mediated autoimmune disorder; the exact pathogenesis of which is not fully understood. Patients with homozygous alleles of human leukocyte antigen (HLA)-Cw*0602 have a 2.5-fold increased risk of developing psoriasis than Cw6 heterozygous patients.[3] Both T-helper cell-17 (Th-17) and interleukin-22/23 (IL-22/23), along with a predominance of CD8+ lymphocytes, have been implicated in its pathogenesis. An altered cytokine milieu in the form of raised levels of interferons (IFN-α, -γ), tumour necrosis factor-α (TNF-α), IL-2, 6, 8, 12 and leukemic inhibitory factor-1, along with low levels of IL-1, 4, 5 and 10 have been reported.[7,8] LP is also a T-cell-mediated autoimmune disorder with cytotoxic CD8+ T-cells damaging the basal layer of keratinocytes. Genetic predisposition (mainly HLA-A3 and HLA-B7) along with environmental triggers such as infections (mainly *Helicobacter pylori*, Hepatitis C, dental amalgam), and drugs (such as lithium, beta-blockers, and non-steroidal anti-inflammatory drugs) induce cytokine production.[3] The altered cytokine milieu of TNF-α, IFN-α, IL-1, 6, 22, 23, and 31 plays a major role in the pathogenesis of LP.[3] These altered cytokines in the form of over-production of IFN-α and T-cell-mediated autoimmunity, as proven by leukocyte migration-inhibition assay, tend to occur in both conditions. Only a few hypotheses have been proposed to explain the coexistence of these diseases. The activation of both CD4+ and CD8+ T-cells in a concurrent manner and overproduction of proinflammatory molecules such as TNF alpha, IL-1, 6, 22, 23, and IFN-α, -γ could have played a key role in the coexistence of these diseases in a single patient.

The CD4+ T-cells play a crucial role in the development of LP. This auto-reactive CD4+ T-cell causes cytolysis of basal keratinocytes resulting in basal stem cell damage, decreasing the epidermal turnover. However, in a study by Shiohara *et al*.,[2] the CD4+ T-cells were also found responsible for the hyperproliferation of keratinocytes in psoriatic patients. Another hypothesis suggested that the overproduction and varied mechanisms incited by IFN-α can also be associated with the coexistence of LP and psoriasis.[4] In psoriasis, the activation of plasmacytoid dendritic cells (pDC) produces Type-I IFNs, mainly IFN-α, which promotes phenotypic maturation of myeloid dendritic cells, which further activates Th1 and Th17 differentiation and IL-17, 21, and 22 production. These cytokines are involved in the proliferation of epidermal keratinocytes in psoriasis. Similarly, in LP, there is an abundant secretion of IFN-α from pDCs, which upregulates chemokines such as chemokine (C-X-C motif) ligand (CXCL) 9 and chemokine (C-X-C motif) ligand (CXCL) 10, releases IL-12/IL-23, and promotes the differentiation and expansion of cytotoxic and helper T-cells, which ultimately results in basal cell damage.

Our case showed a rare coexistence of nail psoriasis and cutaneous LP based on clinical and histological evidence. No triggers were identified. We considered that a simultaneous activation of both cluster of differentiation 4 (CD4+) and cluster of differentiation 8 (CD8+) T-cells and some unknown antigenic stimuli may have initiated IFN-α production leading to co-expression of both diseases. The patient was also hypothyroid for a long duration, suggesting the role of autoimmunity as well as genetic predisposition, precipitated by environmental triggers. Most autoimmune conditions are associated with the above factors, explaining the higher susceptibility of an individual to develop multiple autoimmune diseases. More reports and immunohistochemical studies may help to understand the detailed pathogenesis behind this coexistence.

It has been suggested that the coexistence of LP and psoriasis can be difficult to manage. However, due to a lack of data, the selection of appropriate therapeutic agents and long-term prognosis remains conjectural. Koebner's phenomenon and pruritus can act as an exacerbating factor for both diseases, and may result in treatment failure or recalcitrance, affecting the quality of life.[9]

CONCLUSION

We report the features of coexistent isolated nail psoriasis and cutaneous LP. Although it is a rare combination with unclear pathogenesis, it is essential to be aware to avoid misdiagnosis. An appropriate diagnosis not only enables adequate treatment but also accentuates the patient's quality of life.

Authors' contributions

All the authors contributed to the manuscript preparation and design of the study, acquisition of data, analysis, and interpretation of data, drafting the article and revising it clinically for important intellectual content and final approval of the submitted version.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient's consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

Dr. Chander Grover is on the editorial board of the Journal.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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