Case Report

COVID-19 as a trigger for lichen planus: A case report

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ABSTRACT

Lichen planus (LP) is an inflammatory mucocutaneous dermatosis believed to result from immune-mediated damage to basal epidermal cells, potentially triggered by an exaggerated cytotoxic T-lymphocyte response. We report the case of a middle-aged woman presenting with widespread erythematous-brown macules and papules, sparing her face and hands, which appeared 2 days after a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and were histologically diagnosed as LP. She had received three doses of the Comirnaty (Pfizer-BioNTech) coronavirus disease (COVID-19) vaccine without prior skin manifestations and had no previous COVID-19 infection. The close temporal association between SARS-CoV-2 infection and the onset of LP suggests a potential trigger. The immunological alterations associated with COVID-19, particularly immune dysregulation mediated by cytotoxic T-lymphocytes, may explain the increased propensity for LP development in such cases. Further studies involving larger populations are required to statistically validate this association.

Key words: Coronavirus disease-19, Cytotoxic T-lymphocytes, Dermatosis, Lichen planus

Lichen planus (LP) is an inflammatory mucocutaneous dermatosis attributed to immune-mediated damage targeting basal epidermal cells, possibly initiated by an exaggerated cytotoxic T-lymphocyte response [1]. LP affects approximately 1–2% of the global population, with most cases developing between the ages of 30 and 60 and an almost identical prevalence in both genders [2,3]. Typically presents with distinctive skin and mucosal lesions, usually in the form of violaceous, flat-topped papules with Wickham's striae. The disease is often chronic, with periods of flare-ups, and its diagnosis is based on clinical and histological findings. Its etiopathogenesis remains only partially understood, with most cases considered idiopathic. However, infections, medications, and vaccines can serve as potential triggers [4,5]. Considering the increasing acknowledgment of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) as a factor in triggering immune-mediated dermatological conditions, this case contributes to the expanding literature on coronavirus disease-19 (COVID-19)-related cutaneous manifestations. The immune dysregulation caused by SARS-CoV-2 may play a role in predisposing individuals to inflammatory or autoimmune skin diseases, such as LP. Recognizing this potential link is essential for improving early diagnosis and optimizing treatment strategies in similar clinical scenarios.

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CASE REPORT

A 45-year-old female patient with no relevant medical history presented to the dermatology emergency service with widespread erythematous-brown macules and papules, sparing her face and hands. The lesions appeared 2 days after a SARS-CoV-2 infection. She had previously received three doses of the Comirnaty (Pfizer-BioNTech) COVID-19 vaccine, with the last dose administered 9 months prior, without developing skin lesions, and had no prior history of COVID-19 infection. She denied any other symptoms.

On physical examination, pruritic, violaceous, scaly, flat, and polygonal papules of varying sizes (6–10 mm) were observed on the forearms and flexor surfaces of the wrists, legs, and ankles, suggestive of LP (Fig. 1).

Laboratory investigations revealed normal blood counts (hemoglobin: 12.4 g/dL; leukocytes [white blood cell]: 5.0×10^9/L; neutrophils: 2.16×10^9/L; platelets: 186,000/μL; erythrocyte sedimentation rate: 18 mm [<30 mm]). Biochemical parameters showed negative autoantibody screening (anti-dsDNA antibodies: Negative; antinuclear antibody screening [SSA, SSB, Sm, RNP-Sm, HSP70, Jo-1]: negative [<20]), normal renal function and electrolytes (urea: 33 mg/dL, creatinine: 0.69 mg/dL, sodium [Na^+]: 141 mmoL/L, potassium [K^+]: 4.4 mmoL/L, chloride [Cl^-]: 103 mmoL/L, osmolality: 283 mOsmoL/kg, uric acid: 4.2 mg/dL), and normal liver function. Enzyme activity revealed

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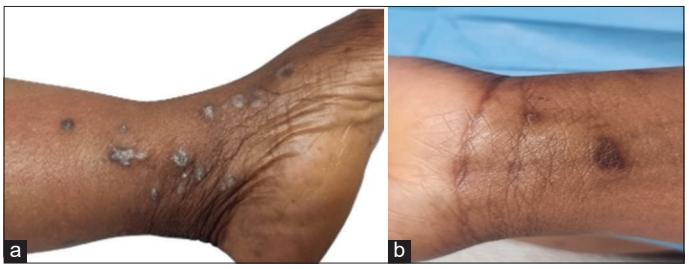

Figure 1: (a) Lichenoid eruption with small, striated, polygonal, flat, violaceous, scaly papules, some isolated, others confluent, distributed over the ankles. (b) Lichenoid eruption with small, striated, polygonal, flat, violaceous, and scaly papules distributed over the flexor surface of the wrist

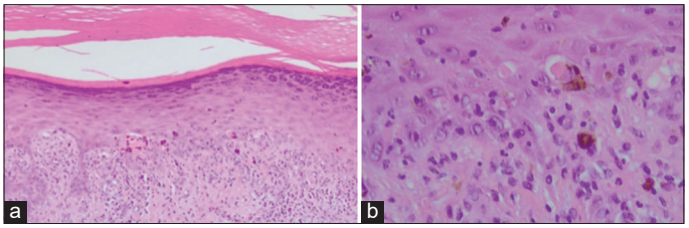

Figure 2: Histopathological examination of the lesion on the right lower limb. (a) Orthokeratotic hyperkeratosis, focal granular layer thickening, irregular acanthosis, and a dense lymphocytic band-like infiltrate at the dermoepidermal junction (H and E, magnification: ×100); (b) Colloid bodies in the superficial dermis (H and E, magnification: ×400)

elevated creatine kinase total at 230 U/L and lactate dehydrogenase at 327 U/L. Serologies for retroviruses, hepatitis, and syphilis were negative. The patient had a high titer of anti-SARS-CoV-2 (S1-RBD Ig: 12,500 U/mL) and elevated total immunoglobulin E (769 U/mL).

Histopathological examination of skin biopsies confirmed the diagnosis of LP (Fig. 2).

The patient was treated with oral corticosteroids and topical betamethasone, leading to clinical improvement. She was referred for follow-up in the dermatology consultation and re-evaluation 1 month after discharge.

DISCUSSION

LP is a chronic inflammatory autoimmune disorder of the skin and mucous membranes, primarily driven by cytotoxic CD8+ T lymphocytes that target basal keratinocytes [6]. These T-cells are activated by antigen-presenting cells, such as Langerhans cells, which present altered self-antigens, possibly induced by viral infections, drugs, or other environmental factors. The activated CD8+ T-cells release pro-apoptotic molecules, including granzyme B, perforin, and tumor necrosis factor (TNF-α), which induce keratinocyte apoptosis. This results in vacuolar degeneration of the basal layer, a hallmark feature of LP [7,8]. Inflammatory cytokines such as interleukin (IL)-1, IL-6, IL-17, and TNF-α amplify the immune response, leading to further recruitment of immune cells to the site of inflammation [9]. Histologically, LP shows interface dermatitis with a dense lymphocytic infiltrate at the dermoepidermal junction [8]. The specific autoantigens involved in LP remain unknown, but potential triggers include viral infections such as hepatitis C or SARS-CoV-2, as well as drugs that modify keratinocyte proteins [10]. Genetic predisposition plays a role, with familial cases and associations with certain human leukocyte antigen types suggesting a hereditary component. Chronic LP may involve dysregulated immune responses, such as persistent T-cell activation and inadequate regulatory T-cell function, leading to chronic inflammation and possible tissue remodeling. This chronicity can result in complications such as post-inflammatory hyperpigmentation or malignant transformation in rare cases [7,8]. Understanding the pathophysiology of LP informs treatment strategies, including the use of corticosteroids, calcineurin inhibitors, and biologics targeting specific immune pathways.

In this case, the temporal association between SARS-CoV-2 infection and the onset of LP suggests that the infection may have acted as a trigger. Dermatological manifestations associated with COVID-19 have been extensively documented, reflecting immune dysregulation induced by the virus. The pathogenesis of LP involves immune mechanisms akin to those observed in COVID-19, particularly the dysregulated activity of cytotoxic T lymphocytes, as previously described. This similarity supports the hypothesis that SARS-CoV-2 infection could predispose individuals to LP by inducing aberrant immune responses.

The management of cutaneous LP focuses on alleviating symptoms, reducing inflammation, and preventing complications such as scarring or post-inflammatory hyperpigmentation. The primary treatment modalities for LP include topical corticosteroids and topical calcineurin inhibitors [11]. Topical corticosteroids, such as clobetasol propionate and betamethasone, are effective for localized and mild-to-moderate cases. Topical calcineurin inhibitors, such as tacrolimus and pimecrolimus, are also commonly used for managing localized disease, particularly in sensitive areas like the face and genital regions. For more refractory or extensive cases of LP, systemic therapies may be required. Systemic corticosteroids, such as prednisone, are frequently utilized for widespread or severe disease, though their long-term use is limited due to potential side effects. Other systemic immunosuppressive agents include methotrexate, azathioprine, and hydroxychloroquine, which are effective in managing recalcitrant LP. Methotrexate is typically used for severe cases or those resistant to other treatments, whereas azathioprine and hydroxychloroquine are often considered for chronic, refractory diseases.

In addition, phototherapy, including ultraviolet light treatments, may be an option for widespread disease that does not respond to topical or systemic treatments. Finally, retinoids, such as acitretin, are reserved for severe or recalcitrant cases that fail to respond to other therapies [12]. These treatments are tailored to the patient's specific presentation and response to therapy, aiming for optimal management and symptom control.

Addressing emotional stress and providing psychological support are essential, as stress is a recognized exacerbating factor

in LP. Emerging therapies targeting specific immune pathways, including anti-TNF agents and IL-17 inhibitors, are being explored but have not yet been established as standard treatments for LP [13]. Further studies are required to validate their efficacy and safety in this context.

Regular follow-up is essential to monitor treatment response and side effects, particularly when using systemic therapies. Complications such as oral LP or malignant transformation (e.g., squamous cell carcinoma in chronic cases) should be carefully assessed [8]. Effective management of LP often requires a multidisciplinary approach tailored to the individual patient's needs.

CONCLUSION

This case report presents a possible link between SARS-CoV-2 infection and the development of cutaneous LP, emphasizing the temporal association between the infection and the onset of LP symptoms. Although the pathogenesis of LP remains incompletely understood, immune dysregulation, especially through the activation of cytotoxic T-lymphocytes, plays a central role in the development of the condition. The mechanisms underlying immune dysregulation in COVID-19 and LP share similarities, particularly in T-cell-mediated basal keratinocyte apoptosis. While this suggests a possible link, further studies are required to determine whether genetic predisposition plays a role in COVID-19-induced LP. This case also highlights the importance of recognizing emerging autoimmune manifestations in patients recovering from COVID-19. Early diagnosis and appropriate treatment, including corticosteroids and immunosuppressive agents, are crucial for managing LP and preventing complications. Further research involving larger cohorts is needed to clarify the association between COVID-19 and LP and to explore effective treatment strategies for affected patients.

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