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Lichen Planus following COVID-19 Infection: A Case Report

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ABSTRACT

The ongoing COVID-19 pandemic has posed considerable challenges to healthcare systems worldwide, with numerous clinical manifestations, not limited to pulmonary symptoms being reported post-infection. Lichen planus (LP) has garnered significant attention due to its plausible link with viral infections such as COVID-19. Our case report aims to describe a case of lichen planus caused by SARS-CoV-2, focusing on pathophysiology, diagnosis, and disease management. This potential correlation requires exploration, given the ability of the virus to induce immune dysregulation. Though there have been numerous case reports detailing the development of LP after COVID-19 infection, further research is required to establish causality.

Here, we present a case of lichen planus with lesions typically involving the upper and lower extremities, occurring one month after COVID-19 infection. Clinical examination raised suspicion of lichen planus, which was further confirmed by histopathological evidence. Despite the viral etiology likely playing a critical role in the development of LP, only a few cases have been reported in association with COVID-19.

Our case thus highlights the importance of physicians considering this possibility to prevent treatment delays, decrease disease burden and improve quality of life.

Keywords: Lichen planus; COVID-19 infection; COVID-19 vaccination; Post COVID-19 syndrome

INTRODUCTION

Lichen planus (LP) is an autoimmune mucocutaneous condition found in 1-2% of the population, typically involving skin, nails, hair, and mucous membranes. It is caused due to destruction of basal keratinocytes by inflammatory T lymphocytes[19]. Females are more commonly affected than males. LP usually occurs in immunosuppressed individuals and occurs secondary to numerous genetic and environmental factors including infectious pathogens such as EBV, hepatitis C virus, among others[20].

Here, we report a noteworthy case of Lichen planus in a 22-year-old man following COVID-19 infection. After recovering from COVID-19 infection, many individuals experience additional symptoms, collectively known as post-COVID-19 syndrome. While literature exists on post-COVID vaccine-associated lichen planus, there are limited publications on lichen planus following COVID-19 infection. This report aims to add to the growing understanding of the dermatological sequelae of COVID-19, emphasizing the importance of vigilance in identifying and managing these manifestations in clinical settings.

CASE PRESENTATION

A 22-year-old man presented with chief complaints of extensive pruritic skin lesions over the dorsal aspect of the left hand, volar aspect of the right forearm and dorsal aspect of both lower extremities in February 2022 at the dermatology outpatient center of a tertiary care hospital in Chennai. The patient revealed that he initially noticed a small, solitary lesion on the dorsal aspect of the left hand shown in (Figure 1). A few days later, he developed new lesions on the volar aspect of the right forearm similar to the earlier eruption. He also noted purplish discoloration and scaling of the lesions. Subsequently, new lesions began to emerge on the dorsal aspect of both feet (Figure 1), which gradually spread to the anterior aspect of both lower extremities. The lesions were associated with intensive itching, exacerbated by sweating. His past medical history was unremarkable except for COVID-19 infection in January 2022. Family history was negative for similar illnesses. The patient had no significant history of allergies in the past. The patient denied any active medications.

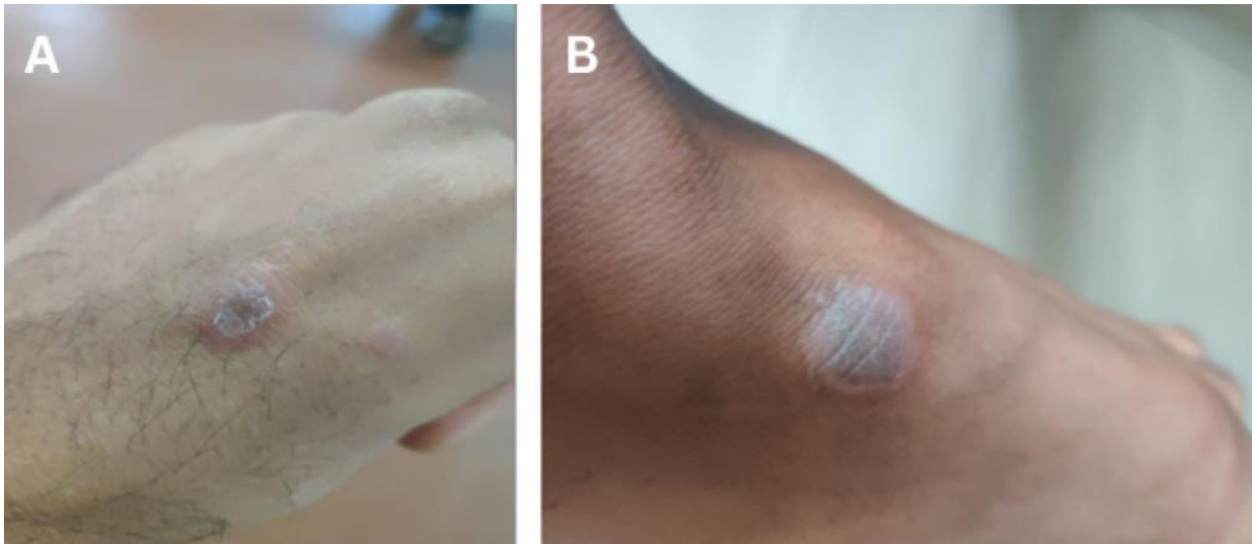

Figure 1: Clinical presentation of Lichen planus skin lesion: (A) A small, single, polygonal, flat-topped lesion with scaling was noted on the dorsum of the left hand (B) A single lesion with similar features of A is noted on the dorsum of the left foot.

On examination, numerous small papules and a few large, coalesced plaques were observed. These were polygonal flat-topped lesions with a violaceous discoloration (Figure 2). On closer inspection, fine white lines termed as Wickham's striae were noted, accompanied by scaling. The patient sought medical care for the above condition and a punch biopsy was ordered. Meanwhile, he was prescribed low-potency topical corticosteroids while awaiting biopsy results. Biopsy result showed hyperkeratosis, acanthosis, wedge shaped hypergranulosis, saw toothing of rete ridges and basal cell vacuolar degeneration features in the epidermis along with the dermis exhibiting dense band like lymphohistiocytic infiltrate consistent with the diagnosis of lichen planus.

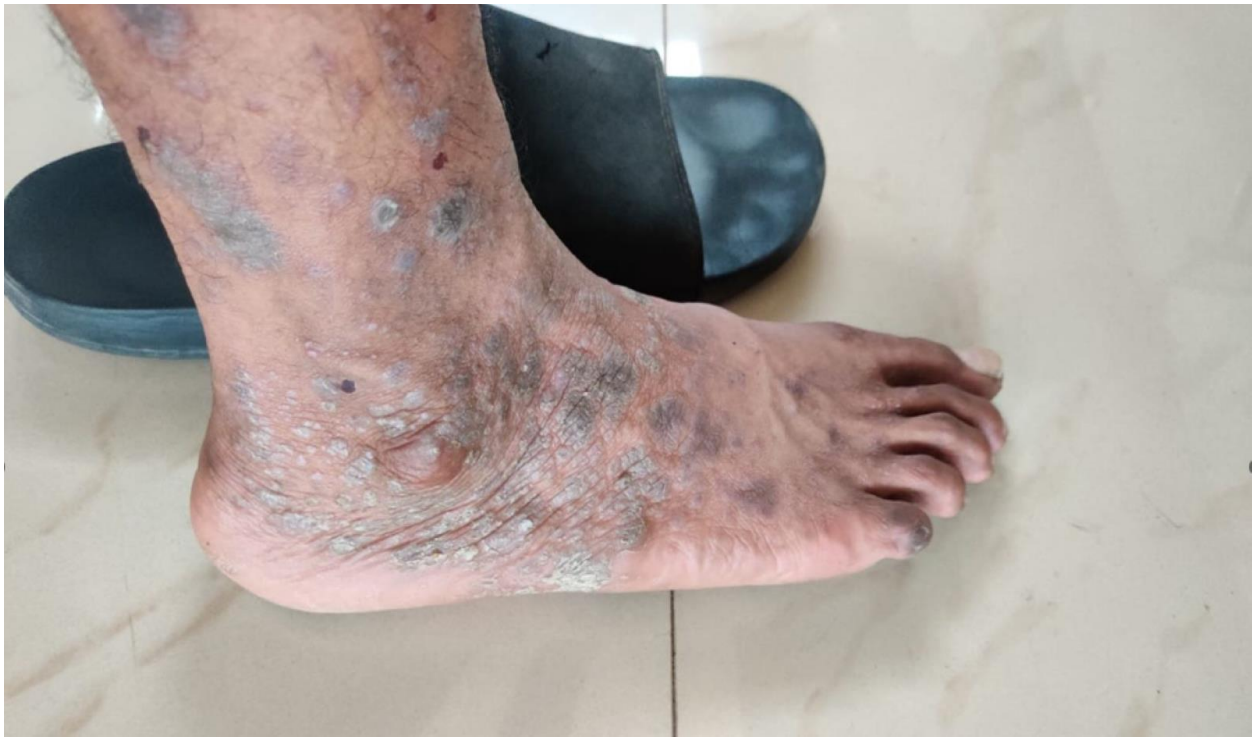

Figure 2: Advanced Lichen Planus Lesions: Severe Dermatological Manifestation on the Foot: Planar, Polygonal, flat-topped, purple confluent plaques with lacy, reticular whitish lines (Wickham's striae) were observed over the right foot, extending to the antero-lateral aspect of the leg.

In an attempt to identify the etiology of lichen planus, a series of investigations were ordered, comprising a complete blood count, basic metabolic panel, thyroid function tests, and autoimmune profile. Additionally, a viral panel including Hepatitis B, Hepatitis C, syphilis, and HIV testing was conducted to rule out any possible association. All investigations turned out to be normal. The only potential etiology identified was his COVID-19 infection a month before being diagnosed with lichen planus.

Due to the severity of the lesions, he was prescribed topical high-potency corticosteroids (clobetasol) and tacrolimus. Additionally, tablet beta-histine 20mg once daily was given to combat intense pruritus. Despite adhering to the aforementioned treatment plan for a month, the patient did not observe any improvement. Therefore, he was transitioned from topical to oral corticosteroids in an effort to facilitate recovery. Prednisone 10 mg once daily for two consecutive days in a week for four weeks was initiated, while continuing existing topical medications. The patient reported drastic improvement in pruritus within just one week of starting the therapy accompanied by gradual healing of lesions. Complete resolution was noted by the end of four weeks, leaving behind scars (Figure 3). Oral corticosteroids were

stopped while topical medications were continued for an additional week with recommendations to adequately moisturize the affected area. At the two-week follow-up, no new or active lesions were observed, except for the presence of scars in the affected regions. Due to cosmetic concerns, the residual scar lesions underwent treatment with a topical formulation containing clobetasol (0.05% w/w) combined with salicylic acid (6% w/w), resulting in satisfactory outcomes in the following months.

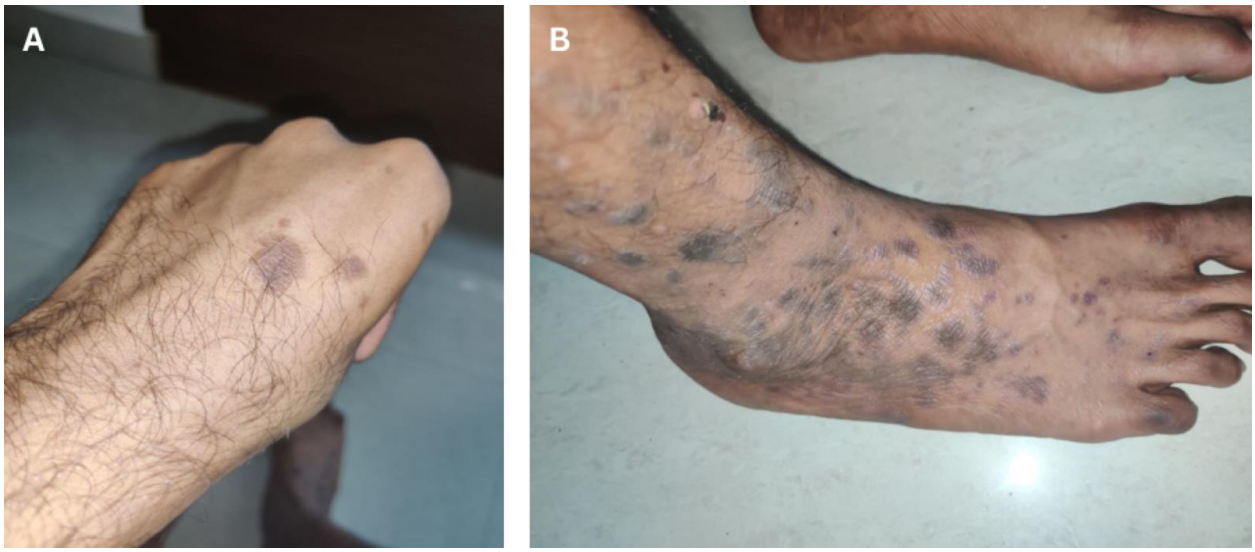

Figure 3: Healed lesions with hyperpigmented macules: (A) dorsum of left hand and (B) dorsal, antero-lateral aspect of the right leg.

DISCUSSION

Lichen planus (LP) is an autoimmune condition that specifically targets basal keratinocytes by the activation of cytotoxic (TH1) CD8+ cells leading to interface dermatitis. This chronic inflammatory process is aided by CD4+ helper T (TH2) cells which produce TH1 cytokines, eventually leading to destruction of basal keratinocytes[3,4]. Numerous factors such as viruses, medications, and contact allergens have been implicated in the development of lichen planus[4]. Lichen planus can develop secondary to genetic and environmental factors including stress, drugs (antimalarial agents, methyldopa, gold etc.), viral infections (HCV, EBV, HHV-7, SARS-CoV2 etc.), vaccination against hepatitis A and B, influenza, or SARS-CoV-2. Various haplotypes such as HLA - A3, -A5, -A28, -B8, -B16, -Bw35, -B7, -B18, -Aw19, -Cw8, are associated with different variants of LP. LP is associated with other autoimmune disorders like alopecia areata, ulcerative colitis, vitiligo, morphea, lichen sclerosus, and myasthenia gravis[5].

LP is an uncommon consequence triggered by COVID-19 infection. SARS-CoV-2 has been found to affect multiple organs not limited to the respiratory system. Numerous dermatological manifestations have been increasingly reported, both due to infection as well as vaccination. COVID-19 induced lichen planus have often been overlooked due to their resolution with topical steroids, leading to a lack of investigation into the potential etiology[1,2].

After extensive clinical assessment and investigations, all potential causes of LP were excluded in our patient. The sole identified triggering factor was his COVID-19 infection one month prior to diagnosis.

Several pathological mechanisms have been identified. Overactivation of CD8+T-cells and uninhibited production of proinflammatory cytokines such as Interleukin-2(IL-2), tumor necrosis factor-alpha (TNF-) and interferon-gamma (IFN-) by CD4+T cells play a dominant role in the implication of the disease[6-10]. Some studies also suggest that COVID-19 has been linked with disruption of mammalian target of Rapamycin (mTOR) pathway leading to abnormal T-cell proliferation. Another widely proposed mechanism attributable to COVID-19 induced lichenoid eruption is molecular mimicry, a phenomenon wherein infectious pathogens/environmental agents resemble self-antigens, leading to cross reactivity and triggering immune response[11]. SARS-CoV-2 proteins exhibit similarity to human mitochondrial M2 proteins, F-actin, TPO proteins and specifically ACE-2 receptors which are abundant in skin and oral mucosa[6,12,13]. SARS-CoV-2 proteins binding with ACE-2 receptors initiate TH1 activation, thereby eliciting an immune response that contributes to the development of LP[12,13]. The viral protein also activates Toll-like receptor 9 (TLR9), prompting the release of cytokines such as tumor necrosis factor-alpha, gamma interferon, IL-2, and IL-6, further facilitating the disease progression[14].

Lichenoid dermatoses comprise of a spectrum of skin conditions including graft versus host disease, lichen sclerosus and lichen planus[15]. Based on the location involved, there are three widely identified subcategories comprising of cutaneous LP (CLP), mucosal LP (MLP) and LP of the scalp (lichen planopilaris, LPP). Clinical variants of CLP comprise of linear LP, annular LP, atrophic LP, hypertrophic LP, inverse LP, eruptive LP, erosive and ulcerative LP[16]. There is a slightly increased risk of squamous cell carcinoma in the mucosal and cutaneous hypertrophic LP types[3].

CLP typically presents red to brown, violaceous, polygonal, intensely pruritic flat papules with mild scaling. These papules coalesce to form larger plaques. White streaks in a lacy or fern-like pattern called the Wickham striae is noticed on closer inspection. The extremities are commonly involved with pruriginous papules found on the medial side of the wrist[15]. Individuals with darker skin tones may observe persistent hyperpigmentation despite resolution of lesions[3]. The diagnosis is primarily clinical, but confirmation requires biopsy and histopathological examination revealing acanthotic saw tooth rete ridges in the epidermis, hypergranulosis, and apoptotic keratinocytes (Civatte bodies)[17].

The treatment of LP remains the same regardless of etiology. The primary objective is to decrease the pruritus and promote faster resolution of lesions. The mainstay treatment of lichen planus is steroid therapy. At the outset, treatment is initiated with super potent topical steroids (triamcinolone, betamethasone and clobetasol). Lesions unresponsive to topical therapy are treated with intralesional Triamcinolone injection. If they persist without significant improvement, therapy is escalated to systemic corticosteroids (oral or intramuscular injections), acitretin, isotretinoin, or oral cyclosporine[18-20]. Topical calcipotriol, Mycophenolate mofetil, azathioprine, methotrexate, and cyclophosphamide, broad or narrowband UVB, topical calcineurin inhibitors like tacrolimus are implicated for refractory cases[3].

DISCUSSION

LP is an extremely rare complication that can arise either following a COVID-19 infection or vaccination. While immunocompromised patients are at increased risk, it can also occur in otherwise healthy individuals, highlighting the importance for healthcare providers to identify the lesions. The precise timeframe for onset of LP after COVID-19 is not clearly defined, but early diagnosis is crucial for appropriate management. While there hasn't been conclusive evidence establishing a direct link between COVID-19 and LP, some studies have suggested a potential association. However, these findings are preliminary, and further research is indeed necessary to explore any potential connections between the two. Understanding such links can provide valuable insights into the pathogenesis and manifestations of COVID-19 and its potential long-term effects.

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DISCLOSURE STATEMENT

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