

## Lip Lichen Planus Following COVID-19 Infection

Niall Hamad<sup>1</sup>, Zinah Sharara<sup>2</sup>, Madhavi Maheshwari<sup>3</sup> and Mohammed S Al Abadie<sup>4\*</sup>

<sup>1</sup>School of Medicine, Keele University, United Kingdom

<sup>2</sup>Community Dermatology, National Health Service, (HealthHarmonie) and The Midlands Medical Academy, United Kingdom

<sup>3</sup>Histopathology Department, Royal Wolverhampton NHS Trust, Wolverhampton, United Kingdom

<sup>4</sup>University of Wolverhampton, North Cumbria Integrated Care NHS Foundation Trust, United Kingdom

### \*Corresponding Author

Professor Mohammed S Al Abadie, PhD, FRCP. Clinical Professor of Dermatology, University of Wolverhampton, United Kingdom.

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### Abstract

Lichen planus is a dermatological disorder believed to be induced by various factors including viral infections, drug exposure, and contact with metals. The condition manifests with diverse patterns and can affect multiple body sites. Here, we describe a patient with histopathology proven lip lichen planus occurring after a coronavirus infection.

**Keywords:** Lichen Planus, Lichenoid Eruption, Wickham's Striae, Pathology

### 1. Introduction

Lichen planus is a chronic inflammatory disorder involving the skin, mucous membranes, hair and nails, predominantly attributed to an autoimmune mechanism. Although its precise aetiology remains unclear, various triggers have been implicated. Factors identified are viral infections, certain medications such as antihypertensives, and exposure to metals like mercury, gold, copper. Psychological stress is also deemed a potential contributing factor [1]. The condition primarily involves individuals between the age of 30 and 60 years. The female to male ratio is approximately 1.5:1 [2].

Clinically, lichen planus presents with diverse morphologies, with the hallmark lesion being clusters of polygonal, purple papules that can be very pruritic. These papules often display a glistening surface characterized by Wickham's striae [3].

Lesions may manifest in various distribution patterns, ranging from isolated to extensive, and may appear in distinct configurations

such as annular, linear, or actinic arrangements. Commonly affected sites include the flexor aspects of the wrists, dorsal hands, lower back, shins, and ankles [2,3].

Mucosal involvement is frequent, particularly affecting the oral cavity, where six clinical subtypes of oral lichen planus have been described [4-6].

- Reticular (most common) - characterised by asymptomatic, white lines on both sides of the buccal mucosa
- Erosive and atrophic – often cause a burning sensation
- Papular, plaque-like and bullous forms are also recognised but less commonly observed

Biopsy and histopathology typically reveal degeneration of the basal layer of the epidermis and a lymphocytic infiltrate along the dermo-epidermal junction. The epidermis shows irregular hyperplasia, producing a saw-tooth pattern accompanied by

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wedge-shaped areas of hypergranulosis [7].

Lichen planus (LP) has been associated with hepatitis C virus (HCV) infection, though prevalence varies widely by region, from 4% in Northern France to 62% in Japan [8].

Despite recent advances in understanding the immunopathogenesis of oral lichen planus (LP), the initial triggers of lesion formation and the essential pathogenic pathways are unknown [9].

## 2. Treatment

The treatment of oral and cutaneous lichen planus (LP) primarily focuses on alleviating symptoms and managing inflammation. Various therapeutic modalities are employed, with topical corticosteroids being the first-line treatment for both oral and cutaneous forms. Clobetasol propionate is commonly used, demonstrating significant improvement in lesions. Other options include topical tacrolimus for mucosal lesions and systemic corticosteroids for severe cases [10,11]. Additionally, alternative treatments such as retinoids, immunosuppressants, and photodynamic therapy have shown promise [12,13]. In refractory cases, oral alitretinoin has been effective in resolving symptoms [14].

Despite these treatment options, lichen planus can be persistent and resistant, particularly in mucosal areas, leading to a need for ongoing management and monitoring [11]. This highlights the complexity of LP and the necessity for individualized treatment plans.

## 3. Case Report

A 60-year-old male with a history of coronavirus infection in November 2021 was referred to community dermatology services in July 2022 presenting with swelling, depigmentation, and thickening of the upper and lower lips. The patient reported accompanying symptoms of lip cracking, redness, and significant soreness, which had persisted since his COVID-19 illness. His medical history was otherwise unremarkable, with no ongoing medications or known allergies. He had undergone a two-week wait referral to maxillofacial surgery, where no signs of skin malignancy were identified. Notably, the patient was a smoker.

On clinical examination, lichenoid skin changes were observed on both the upper and lower lips, with pronounced thickening noted particularly on the lower lip.



**Figure -1:** Swelling, thickening and leukoplakia of the lips

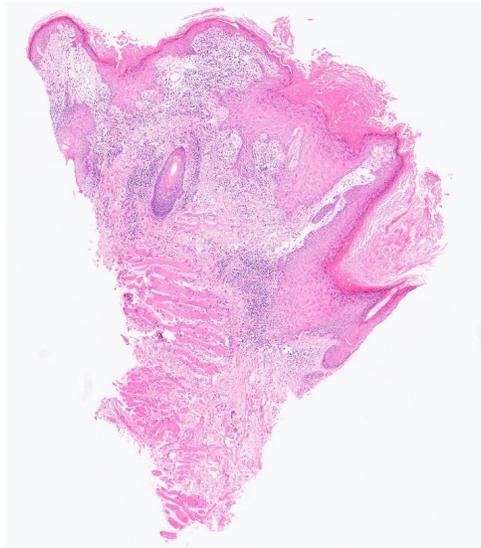
Investigations revealed a normal complement C3 but a raised complement C4. A speckled staining pattern was seen when testing anti-nuclear antibodies, which may be seen with several connective tissue diseases, often associated with Ro and La in Sjögren's syndrome.

## 4. Histopathology

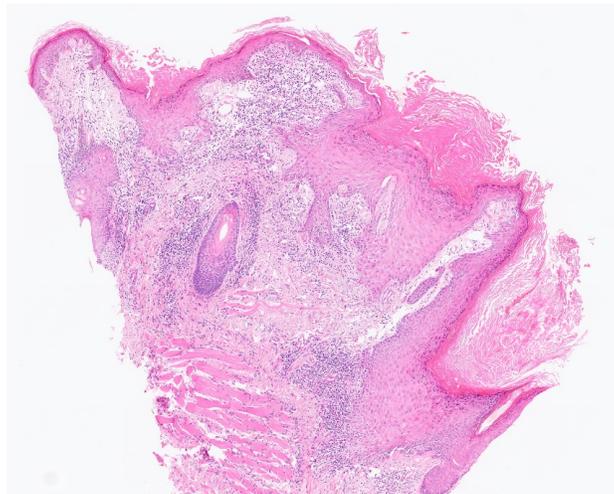
The patient had a 3mm punch biopsy performed on the left lower lip which revealed marked hyperkeratosis with focal follicular plugging and almost wedge-shaped hypogranulosis Figure -2, Figure -3, and figure -4. There was evidence of basal cell damage

with almost lichenoid inflammatory infiltrate at the epidermal dermal interface. Few apoptotic keratinocytes were noted in the basal epidermis.

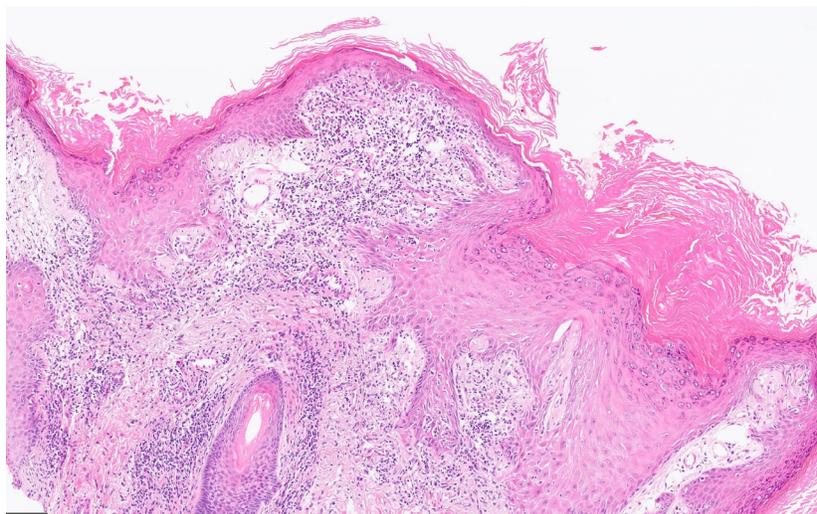
The dermis included showed no obvious peri-vascular or peri-adnexal inflammation. ABPAS stain showed some small rounded fungal debris admixed with keratin which appeared non-pathogenic while the dermis showed no obvious mucin. The appearances are of a lichenoid inflammation, and the features are suggestive of lichen planus. The patient was prescribed Elocon 0.1% ointment applied once a day on the lips with follow-up arranged.



**Figure 2:** Haematoxylin and Eosin staining (H&E) magnification 5.5x



**Figure 3:** Haematoxylin and Eosin staining (H&E) magnification 10x



**Figure 4:** Haematoxylin and Eosin staining (H&E) magnification 16x

## 5. Discussion

The aetiology of lichen planus (LP) remains poorly understood despite multiple known triggers. Current evidence supports LP as a T cell-mediated autoimmune disorder predominantly involving cytotoxic CD8+ T cells [15]. Potential triggers include viral infections such as COVID-19, certain medications, and exposure to metals, notably from dental restorations. The patient described above developed lip lesions following COVID-19 infection; although causality cannot be confirmed, similar associations have been frequently documented. An observational study demonstrated a statistically significant association between COVID-19 infection or vaccination and the onset of de novo LP, suggesting that COVID-19-related events may act as triggers [16]. A systematic review following PRISMA guidelines analyzed publications from December 12, 2019, to May 31, 2022, identifying 25 articles reporting 26 patients (mean age 55.3 years; 65.4% female) who developed LP after COVID-19 infection or vaccination.

## 6. Conclusion

The available evidence indicates a potential link between COVID-19 infection or vaccination and the onset of lichen planus, likely through immune-mediated mechanisms involving cytotoxic T cells. While causality remains to be definitively established, the consistent reporting of LP cases following COVID-19-related events highlights the need for heightened clinical awareness and further research to clarify these associations.

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