CASE REPORT

# MANAGEMENT OF EROSIVE ORAL LICHEN PLANUS ASSOCIATED WITH CHRONIC HEPATITIS C

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Hepatic disorders are generally silent, with slow progress, and are usually detected when the organ's function is severely affected. Several studies have shown that oral lichen planus (OLP) is one of the most Hepatitis C-associated diseases. The emergence of such an extrahepatic manifestation is frequent and can lead to an early diagnosis. Patients with chronic hepatitis C virus (HCV) infection exhibit extensive forms of OLP, with frequent periods of exacerbation of symptoms refractory to treatment, which is commensurate with the severity of the liver disease. When treating OLP in HCV patients, researchers have diverged results. This case report presents an extensive, erosive OLP in an HCV-positive female patient for whom clinical management was tremendous. Full remission was obtained after topical corticosteroid application and several sessions of oral cavity sanitation. This clinical case demonstrates that dentists play a major role in OLP diagnosing, managing, and malignant transformation preventing. They may also contribute to the screening of hepatitis C infection.

**Keywords:** Lichen Planus; Oral; Hepacivirus; Virus Diseases; Pathology

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## INTRODUCTION

The first histologically confirmed case of Oral Lichen Planus (OLP) in a patient with chronic hepatitis C was reported in 1991.[1] A meta-analysis integrating around forty studies showed that the hepatitis c virus (HCV) - OLP association was significant.[2] In addition, the erosive form of OLP has been reported to be preferentially associated with HCV infection.[1] However, more recent studies have shown significant variations in the OLP-HCV association according to patients' age and geographic origin.[3] In particular, the presence of the broad-antigen serotype HLA-DR6 allele would be partly responsible for variations in the HCV-OLP association depending on the geographical area.[4]

Hepatitis C virus is an enveloped RNA virus with an essentially hepatic tropism.[5] This virus, capable of causing chronic infection in humans, is responsible for the hepatitis C epidemic, a severe consequence of which concerns the occurrence of hepatic carcinoma. The recent development of very effective antiviral molecules makes it possible today to have very effective treatments to fight against hepatitis C. However, some of these antiviral therapies such as interferon-alpha (IFN-α) and ribavirin therapy were incriminated in the OLP onset.[6]

This paper aims to highlight the importance of dentists' role in screening HCV infection associated with OLP, and the value of dental care and follow-ups in relieving the patient and preventing malignant transformation.

## CASE REPORT

A 70-year-old female patient consulted the oral medicine and oral surgery department of the academic dental clinic of Monastir, Monastir, Tunisia, with a chief complaint of 5 months on defusing oral lesions causing pain, burning mouth sensation, and discomfort, especially with chewing and speaking.

The medical history was significant to hypothyroidism, high blood pressure, Cardiac arrhythmia, and type II diabetes mellitus. Her medication included Levothyrox® 125µg per day, Actopril®, and insulin injections.

Extraoral examination showed itchy purplish patches over the arms and legs, and diffuse crusty ulcerative lesions over her lower lip (Figure 1A). These lesions were painful, bleeding with a tendency to infiltrate the adjacent skin.

The intraoral examination revealed poor oral hygiene, a deteriorated metallo-resinous bridge extending from the right mandibular canine to the second left premolar, and multiple amalgam fillings.

Diffuse erosive areas extending from the pre molars region up to the retromolar area of the buccal mucosa were detected (Figures 1B and 1C). These erosions were surrounded by white radiating striae from the periphery of the lesions symmetrically on both sides. Similar lesions with an erythematous background were noted over the upper and lower, anterior and posterior vestibular regions, dorsal and ventral surfaces of the tongue, and the interior surface of the lower lip (Figures 1D and 1E), and posterior maxillary edentulous ridge (Figure 1F). Gingiva showed marginal erythema with superficial erosion on the attached gingiva. The lesions were extremely painful and sensitive to touch.

Although a secondary infection with candidiasis was suspected, the initial diagnosis was highly suggestive of erosive oral lichen planus.

Oral swabs were obtained from the above-mentioned locations for microbiological examinations which showed numerous pale yellowish dome-shaped colonies of *C. albicans* when cultured in Chromagar® medium.

An initial prescription of antiseptic mouth wash (chlorhexidine, 0.12%) along with a topical antifungal agent (Fungizone®) 3 times a day for 2 weeks was made to treat the fungal infection. She was subjected to a complete hemogram, hepatitis B and C serology blood test, and Thyroid blood tests. After 2-week's follow-up, the patient reported a slight improvement in symptoms (Figures 2A, 2B, 2C, and 2D).

Sanitation of the oral cavity was initiated with periodontal scaling, root planning, and the removal of the bridge. Regarding the blood test results, all the hemogram parameters were found to be within the normal range. Thyroid blood tests revealed a hypothyroidism FT4: 23,88 pmol/L (10-22) and TSH<0,05 µL/ml (0.27–4,2), and an HCV serology using ELFA (Enzyme-Linked Fluorescent Assay) was positive.

An incisional biopsy of the lesion was performed under local anaesthesia (Medicaine 2% with adrenaline 1/100.000®, Medis, Tunisia) from the left buccal mucosa. The specimen was divided into two parts, the first was kept in 10% formalin and submitted to the anatomopathological examination and the second was immediately placed in saline solution and immediately transported for direct immunofluorescence analysis.

The anatomopathological results revealed a band-like infiltrate made up of lymphocytes and plasma cells associated with liquefaction degeneration of the basal cell layer confirming the diagnosis of erosive OLP (Figure 3).

Topical corticosteroids (Solupred® 20 mg) were prescribed as a mouth rinse 3 times a day. The patient was subjected to regular follow-ups (Figures-4: A, B, C, D, E, F) and dose tapering after 6 months when the erosive lesions and symptoms completely regressed (Figures-5: A, B, C, D, E).

The patient is followed up with the collaboration of the gastroenterology and hepatology department.

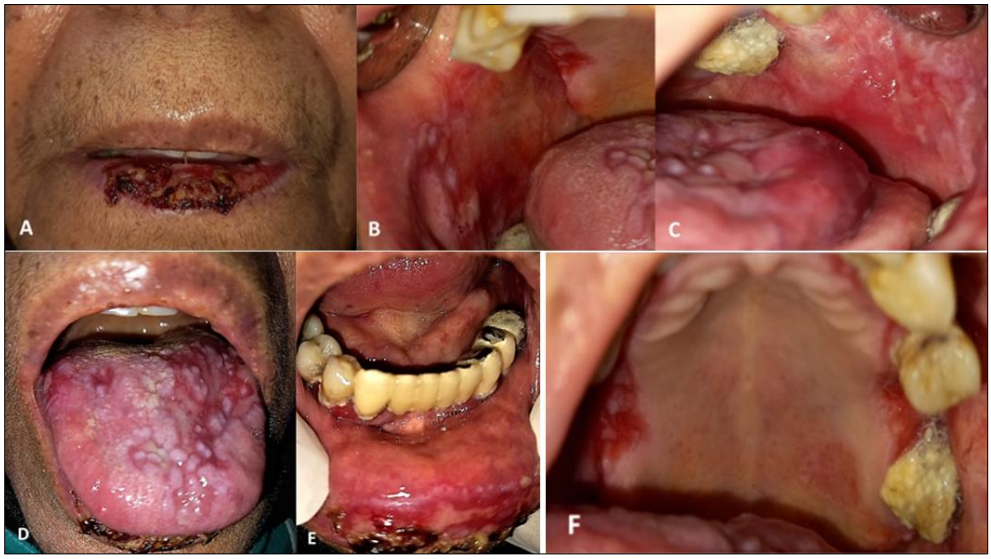
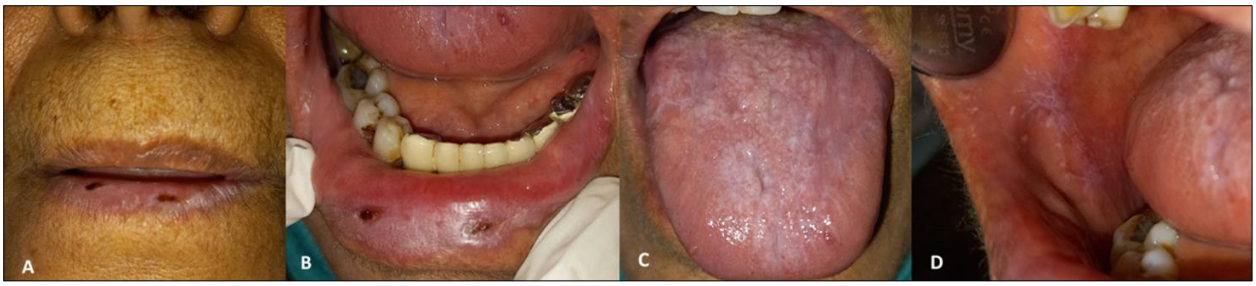



**Figure 1A - Diffuse crusty ulcerative lesions over the lower lip; 1B, 1C - Diffuse erosions surrounded by white radiating striae extending from the premolars region up to the retromolar region bilaterally on the buccal mucosa; 1D, 1E - Diffuse erosions with an erythematous background over the lower, anterior, and posterior vestibular regions, dorsal and ventral surfaces of the tongue; 1F - Erosive lesions on the posterior maxillary edentulous ridges with erythematous aspect in the marginal gingiva.**




**Figures-2: A, B, C, D - Two weeks' follow-up with symptoms regression**

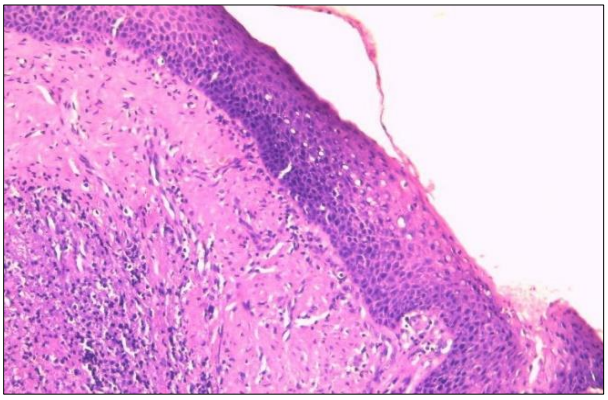
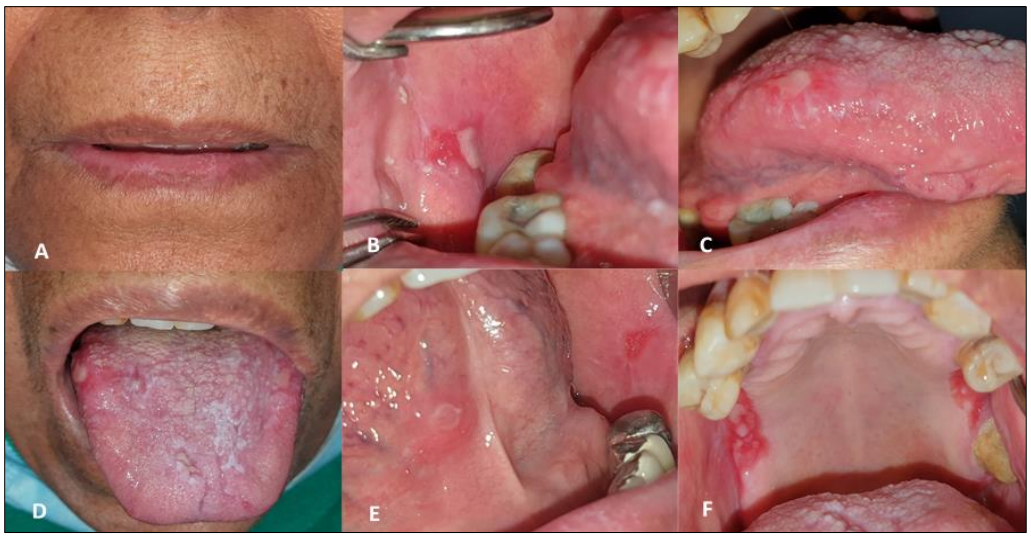
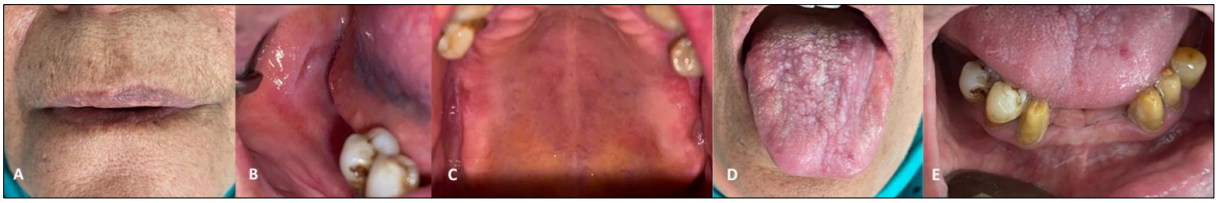


**Figure-3: Anatomopathological slides of the buccal mucosa showing a band-like inflammatory infiltrate predominantly made of lymphocytes and histiocytes.**
**(haematoxylin and eosin, original magnification 200x magnification)**




**Figures-4: A, B, C, D, E, F - Partial recovery of the ulcerative lesions over the palatal, labial, lingual and buccal mucosae after 1-month follow-up**




**Figures-5: A, B, C, D, E - Complete healing of symptoms after 6-months follow-up.**

## DISCUSSION

Oral lichen planus (OLP) is a chronic inflammatory disease of unknown aetiology that can occur as either a single condition of the oral mucosa or in association with lesions in genital mucosae, skin, or nails.[7] This disease is believed to affect around 0.2–2.3 percent of the overall population and accounts for nearly 0.6 percent of all conditions discovered by dentists regularly. OLP is widely distributed and affects all racial and ethnic groups, but it is most frequent in middle-aged female adults.[7]

The clinical manifestations of OLP encompass diverse forms, including keratotic lesions such as papules, reticular, or plaque-like lesions, often linked to atrophic, erosive, ulcerative, or bullous variants.[8] The erosive form prompts an array of symptoms, ranging from mild discomfort to profound functional disruptions that impact quality of life. Lesions commonly emerge bilaterally on the posterior buccal mucosa, with potential effects on the dorsal tongue, labial mucosa, and gingiva. Gingival mucosa involvement is termed desquamative gingivitis.[7]

Exogenous factors, including viral infections, are associated with OLP. Of the eight recognized human herpesviruses, Herpes simplex 1, Cytomegalovirus, Epstein-Barr virus, and Herpesvirus 6 have been implicated in OLP. Additionally, the human papillomavirus, human immunodeficiency virus, and hepatitis viruses B and C also show links to the condition.[9]

HCV infection is widespread, affecting around 71 million people worldwide[10], often causing extrahepatic manifestations (EHMs) in about 74% of cases[11]. EHMs include xerostomia, sialadenitis, Sjögren's disease, OLP, oral verrucous and squamous cell carcinomas, as well as bleeding disorders, cheilitis, gingivitis, and autoimmune bullous diseases like Pemphigus Vulgaris and bullous pemphigoid.[12,13]

These EHMs can serve as early signs of asymptomatic hepatitis C infection. Recent studies show a 22.3% prevalence of HCV among OLP patients[14], varying globally due to geographic differences. This co-occurrence could be tied to endemic HCV distribution in certain regions[15]. HCV prevalence worldwide fluctuates, averaging around 3%, although it's likely underestimated. The 2017 World Health Organization global hepatitis report indicated the highest prevalence in the Eastern Mediterranean (2.3%), followed by Europe (1.5%), and the lowest in South-East Asia (0.5%), with significant variation within these regions.[16,17] In North Africa, HCV prevalence ranges from 1.2–1.9%. However, a recent review spanning 1991–2019 found Tunisia's general population HCV prevalence to remain under 1%, classifying it as a low-endemic country.[18]

While the association between OLP and chronic HCV infection is well recognized, the underlying pathogenesis remains an intriguing subject of inquiry. Several hypotheses have been proposed to shed light on this association:

The Hepatitis C virus could initiate an autoimmune process, a proposition supported by the co-occurrence of LP with other autoimmune conditions like diabetes, vitiligo, and myasthenia gravis.[19]

HCV might directly impact cellular replication and induce immunological changes that contribute to the development of LP.[20]

In cases of chronic HCV infection, immunological alterations and the emergence of circulating autoantibodies are conceivable. These autoantibodies include anti-cardiolipin antibodies, anti-nuclear autoantibodies, anti-mitochondrial antibodies, anti-smooth muscle antibodies, rheumatoid factor, and anti-thyroperoxidase antibodies. The lymphotropism of HCV may underlie these serological autoimmune manifestations.[21]

Comparing OLP patients with liver diseases to those with normal liver function reveals notable distinctions. Those with chronic liver conditions often present more extensive and treatment-resistant OLP forms, mirroring the severity of their liver ailment. The acute erosive OLP variant is primarily associated with chronic HCV infection, marked by elevated serum transaminases and increased viral replication.[22]

Considering a recent study by Arduino *et al*.[23], the merits of automatic HCV screening in OLP patients warrant discussion, given the reduced prevalence of HBV in the younger population.

In our case, exploring HCV's viral involvement was crucial, especially for an elderly female residing in a rural community with limited access to disease-related initiatives. Collaborative efforts with infectious disease specialists were essential to balance effective OLP management and address the underlying condition.

Additionally, testing all diagnosed OLP patients for HCV merits attention, as this liver ailment can quietly progress for extended periods, evading detection.[1]

When devising OLP treatment strategies, factors like cost-effectiveness, patient history, drug interactions, treatment adherence, and psychological state are vital.[23] Early intervention, driven by a precise diagnosis, holds paramount importance.[23]

Primary therapy involves topical corticosteroids, reducing inflammation and immunological responses.[24] Systemic corticosteroids become an option if topical remedies prove inadequate. When corticosteroids fall short,

immunomodulatory alternatives like calcineurin inhibitors, retinoids, or systemic immunosuppressants may be considered.[25]

While biologic agents such as alefacept, efalizumab, and basiliximab, along with polysaccharide nucleic acid fraction of bacillus Calmette–Guerin (BCG-PSN), have emerged[24], their use in OLP remains reserved for severe or refractory cases due to high costs[25].

For OLP patients with localized plaques or resistant erosions, surgical excision stands as an option.[26] Cryosurgery has treated stubborn erosive OLP, yet scarring may result.[27]

Numerous studies have investigated desquamative gingivitis in OLP patients, revealing chronic and severe gingival lesions. Plaque accumulation, more prevalent due to decreased tooth brushing frequency, raises the risk of long-term periodontal issues. Effective management necessitates periodontal therapy, encompassing biofilm reduction, scaling, root planning, decay treatment, and strict oral hygiene. Combining this approach with topical corticosteroid prescription yields improved outcomes.[28]

In cases of severe and refractory erosive OLP, ultraviolet A (PUVA) therapy and photodynamic therapy have demonstrated efficacy.[29] Laser therapy's potential impact on erosive OLP is documented across diverse studies, involving Diode laser[30], pulsed diode laser biostimulation[31], carbon dioxide laser evaporation[32], and low dose excimer laser with ultraviolet B rays[33]. While encouraging results emerge, the definitive efficacy of this approach remains to be established.[29]

HCV treatment options include interferon-alpha (IFN-α) monotherapy or combined IFN-α and ribavirin therapy for chronic cases. Studies explored IFN-α's effect on HCV patients with OLP, yielding mixed outcomes—improvement, triggering, or exacerbation of OLP lesions.[6,29,34]

This suggests non-primary HCV causation, implicating other host-related factors in HCV-related OLP pathogenesis.[35]

Direct-acting antiviral (DAA) therapy is recommended for chronic HCV, considering factors like liver fibrosis, genotype, resistance, prior failures, and comorbidities. DAAs offer higher response rates and shorter treatment duration than IFN-α and ribavirin.[29,34] DAA effectiveness on OLP lesions varies; improvements documented[36], while cases of OLP worsening and new cutaneous/genital LP development were observed[37].

The studies of Nagao Y. and Tsuji M.[38] underscore dentists' role in detecting HCV in patients with oral mucosal disorders. In this context, HCV could be discovered alongside OLP during a dentist-initiated hepatic investigation. Dentists also contribute to patients' quality of life through adequate dental care, mandating regular follow-ups to avert malignant transformation. OLP, particularly with Hepatitis C involvement, is linked to oral cancer development.

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**Conflict of Interest.** The authors declare that there is no conflict of interest.

Informed consent was obtained from the patient. The treatment was conducted following the Helsinki Declaration.

## AUTHORS' CONTRIBUTION

TM study design, draft, and manuscript editing. AB patient management, study design, draft, and manuscript editing. TM and AB have the same contribution to this paper. WN study design, manuscript editing. NB study analysis. JS manuscript editing.

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