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Chronic Hepatitis C and Its Dermatological Impact: Focus on Lichen Planus

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Abstract **Case Report**

Lichen planus (LP) is a chronic inflammatory condition affecting the skin and mucosal surfaces, its association with liver diseases is well estiblashed particulary with hepatite c and it has been widely approched since 1990 here we report a two clinical cases of OLP in an HCV-positive patient. This cases report demonstrates not only the importance of diagnosing EHMs for identification of HCV infection, but also the importance of controlling it for management of OLP and EHMs.

Keywords: Oral Lichen Planus (OLP), Hepatitis C Virus (HCV), Chronic Inflammatory Disease, Immunological Response, Liver Disease.

INRODUCTION

Oral lichen planus (OLP) is a chronic inflammatory condition affecting the mucous membranes inside the mouth, characterized by white, lacy patches and painful sores.

Its a reaction immunologically mediated by antigens and especially orchestrated by T CD8+ lymphocytes, which promotes destruction of the keratinocytes in the basal layer of the epithelium [11]. While its exact cause remains elusive, some hypotheses have been suggested. A viral origin is suspected due to the presence of inclusion bodies observed under electron microscopy [2]. Other proposed causes include genetic predisposition (family history), as well as psychogenic, immunological, and neurological factors. Recently, researchers have also investigated various chronic liver diseases as potential contributors to the development of lichen planus. Hepatitis C is a viral infection that affects the liver [2]. It can cause both acute (short term) and chronic (long term) illness.

It can be life-threatening. Hepatitis C is spread through contact with infected blood. This can happen through sharing needles or syringes, or from unsafe medical procedures such as blood transfusions with unscreened blood products [19]. Symptoms can include fever, fatigue, loss of appetite, nausea, vomiting, abdominal pain, dark urine and yellowing of the skin or eyes (jaundice). talking about OLP, HCV is a major etiological factor under investigation due to its potential to trigger autoimmune responses and chronic inflammation in various tissues, including the oral mucosa. Studies have shown an association between HCV infection and an increased prevalence of OLP, suggesting a possible link between hepatic conditions and the development of oral manifestations [21]. Understanding these connections could lead to improved management strategies for both hepatic diseases and oral lichen planus.

The aim of this world is to report a two cases report of two patients in which hepatitis C infection was detected while complementary investigations required in case of OLP in order to highlights the diagnostic procedures and treatment strategies adopted for oral lichen planus associated to hepatits C virus (HCV) infection.

CLINICAL CASES

First Clinical Case

A 64-year-old female patient consulted the department of oral medicine and surgery at the university hospital clinic of dental medicine in Monastir for a sensation of burning and tingling in the oral cavity and painful lesions on the inner surfaces of the cheeks her familial and pas medical history was non-contributory.

On exobuccal clinical examination, the lower level of the face was sagging, and the labial mucosa was

atrophic.

Endo Buccally, the patient was totally edentulous. On the inner surfaces of the cheeks, erosions and bullae associated with whitish striae arranged in a meshwork pattern were observed. Lesions were bilateral and symmetrical. The rest of the oral mucosa was normal except for simple atrophy (Figure 1).

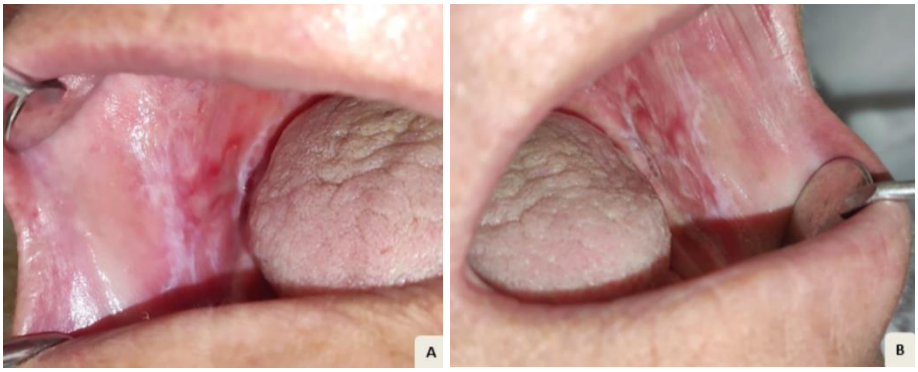

Fig 1: A/B: Erosions and bullae associated with a whitish network on the inner cheeks

The diagnosis of oral lichen planus super infected by candidiasis was suspected. A biopsy for histological examination and direct immunofluorescence was performed.

A mycological examination for Candidas Albicans was requested. Anatomopathological examination confirmed the diagnosis of bullo-erosive oral lichen planus. Mycological examination was negative.

A local corticosteroid: Solupred® 20 mg, 2 Cp*3/day was prescribed. Biological tests were required: fasting blood glucose, thyroid functions tests, Hepatitis C serology.

Fasting blood glucose and thyroid function tests were within normal limits, but hepatitis C serology was positive. The patient was then referred to the gastrology department for further investigation and was declared cured after genotyping, which did not detect HCV RNA, and viral load determination, which was also undetectable (Figure 2).

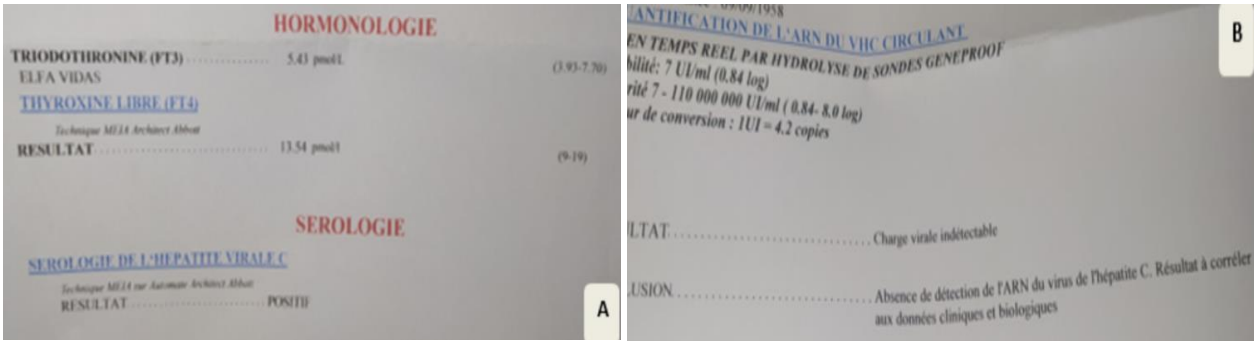

Fig 2: A/B: hepatitis c serology, viral load detection and genotyping

The check-up sessions were reassuring, with a favorable evolution under treatment (Figure 3).

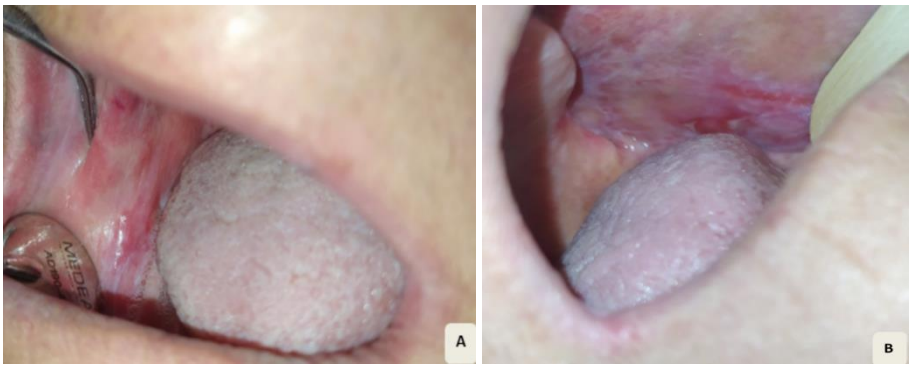

Fig 3: A/B: favorable outcome, beginning of lesion regression

Second Clinical Case

a 55-old-female patient consulted the department of oral medicine and surgery at the university hospital clinic of dental medicine in Monastir for a chief complain of burning sensation throughout the oral cavity with bilateral white lesions in a network on the inner face of the cheeks and lower lip.

On exobuccal clinical examination, the lower level of the face was depressed, and the labial mucosa was atrophic (Fig 1).

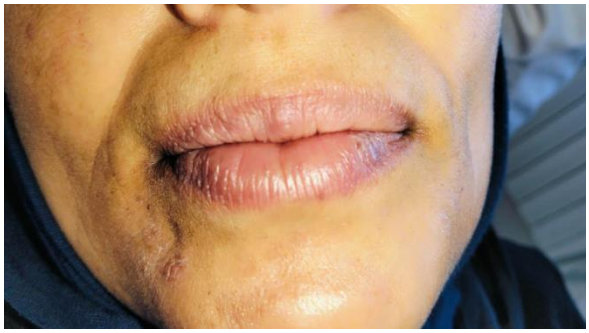

Fig 1: Exo buccal examination

On the inner surfaces of the cheeks, erosions and bullae associated with whitish striae arranged in a meshwork pattern were observed (Fig 2). Lesions were bilateral and symmetrical.

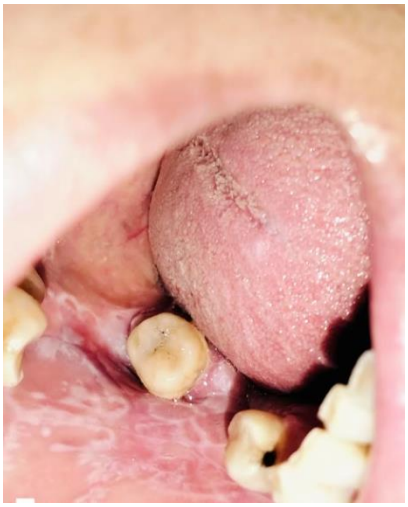

Fig 2: Endo buccal examination showing erosions and bullae associated with whitish striae arranged in a meshwork pattern

The diagnosis of erosive oral lichen planus was suspected.

A panoralic x-ray, CBC, ferritinemia, vitamin D dosage was requested. A biopsy for histological examination and direct immunofluorescence was done.

Anatomopathological examination confirmed the diagnosis of erosive oral lichen planus. Hepatitis C serology was positive with minimal viral load. The vitamin C dosage had a normal value of 650 IU/day

CBC and Panoramic x-ray didn't show anything particular or suspicious

The hepatic report through blood test showed normal values: ASAT (SGOT) = 18UI/L (usual value; less than 35)

ALAT (SGPT) = 15UI/L (usual value;less than 35)

GGT (gamma-glutamyl transferase) =18.00UI/I (usual value; 7-35) alkaline phosphatase = 85UI/I (usual value; 42-98)

A corticosteroid: Copred® (20mg) 3 times a day for 15 days as a mouthwash was prescribed.

Patient was recalled for regular follow-up which was uneventful.

DISSCUSION

Lichen planus is primarily diagnosed clinically, but biopsies can be crucial to confirm the diagnosis, particularly in cases where the presentation is atypical. Histological examination typically reveals characteristic features such as hyperkeratosis, basal layer vacuolization, hypergranulosis, "saw-tooth" shaped rete ridges, a band-like lymphocytic infiltrate at the dermal-epidermal junction, and apoptotic keratinocytes in the papillary dermis, known as Civatte bodies [19].

This disease is estimated to impact between 0.2% and 2.3% of the general population and represents approximately 0.6% of the conditions routinely identified by dentists [13]. Oral Lichen Planus (OLP) is prevalent across all racial and ethnic groups, though it is most commonly observed in middle-aged women [13].

This pathology is based on an autoimmune reaction, mediated by cytotoxic T lymphocytes directed against oral mucosal cells, for reasons that are not yet fully understood [16]. However, there appear to be several predisposing or aggravating factors.

Recently, associations with hepatitis C-related liver failure, primary biliary cirrhosis and other forms of hepatitis have been reported in the other side HCV infection is prevalent globally, affecting approximately 71 million individuals. It frequently leads to extrahepatic manifestations (EHMs) in about 74% of cases. These EHMs can include xerostomia, sialadenitis, Sjögren's syndrome, oral lichen planus (OLP), as well as oral verrucous squamous cell carcinomas Pemphigus Vulgaris and bullous pemphigoid [20]. These EHMs can aid clinicians in identifying HCV infection.

Globally, the prevalence of HCV infection shows regional variations, averaging around 3%, though this is likely underestimated [1]. The incidence and prevalence of HCV infection differ by geographic region, being high in Africa and the Eastern Mediterranean and low in Western Europe and North America [4]. Egypt reports the highest prevalence, with approximately 20% of blood donors having HCV antibodies while recent review spanning 1991 to 2019 found Tunisia's general population HCV prevalence to

remain under 1%, classifying it as a low-endemic country.

Although the association between oral lichen planus and chronic hepatitis C virus is well documented, the pathogenesis remains unclear. Several hypotheses may explain this association [21]:

1. Hepatitis C virus may trigger an autoimmune process, supported by the association of lichen planus with other autoimmune diseases such as vitiligo, myasthenia gravis, or diabetes.
2. HCV may directly interfere with cellular replication and cause immunological changes that lead to the development of mucocutaneous lesions of lichen planus.
3. In chronic HCV infection, immunological changes and circulating autoantibodies can emerge, including anti-nuclear autoantibodies (ANA), anti-cardiolipin antibodies (ACL), anti-smooth muscle antibodies (ASMA), anti-mitochondrial antibodies (AMA), anti-thyroperoxidase antibodies (ATPO), and rheumatoid factor (RF). These serological autoimmune manifestations are attributed to the lymphotropism of the hepatitis C virus.

The association between lichen planus and chronic liver disease has been recognized since 1980. Studies from various geographic regions have found the prevalence of chronic hepatitis among lichen planus patients to range from 0.5% to 35% [6]. In 1991, Monki and collaborators reported the first histologically confirmed case of lichen planus in a patient with chronic active HCV hepatitis, suggesting a link between the two conditions. Subsequent research has supported this connection, with reported prevalence of chronic active hepatitis C ranging from 16% to 55% in study groups and 0.17% to 4.7% in controls.

A 2004 study by Lodi and collaborators investigated the presence of HCV antibodies in 581 patients, 303 of whom were diagnosed with oral lichen planus based on clinical and histopathological criteria [10]. The remaining 278 subjects, who did not have clinical oral lesions, served as the control group. The study found that 19.1% of lichen planus patients had HCV antibodies, compared to only 3.2% of the controls [17].

When comparing oral lichen planus patients with various liver diseases to those without significant general pathology, several differences were noted. Patients with chronic liver disease generally exhibited extensive forms of oral lichen planus, frequent exacerbations of clinical lesions, and symptoms refractory to treatment, aligning with the severity of their liver disease.

Initial treatment typically involves topical corticosteroids, which help reduce inflammation and modulate immune responses. If topical treatments are insufficient, systemic corticosteroids may be considered. alternative immunomodulatory treatments may be explored (8).

For patients with Oral Lichen Planus (OLP) who have localized plaques or persistent erosions, surgical excision can be a viable treatment option [18].

Cryosurgery is also used for managing persistent erosive OLP, although it may lead to scarring [18].

For severe and resistant cases of erosive OLP, both ultraviolet A (PUVA) therapy and photodynamic therapy have proven to be effective. This article is both interesting and useful, as it addresses the regular follow-up of patients for both oral disease and liver involvement. The acute, erosive form of lichen planus is often associated with active chronic hepatitis C, indicated by elevated serum transaminase levels and accelerated viral replication. In such cases, it is crucial to investigate hepatitis and collaborate with infectious disease specialists to balance the underlying condition and appropriate therapy.

CONCLUSION

HCV infection is often asymptomatic and largely underdiagnosed, potentially leading to severe consequences. Recognizing its extrahepatic manifestations can help identify asymptomatic HCV-infected patients.

Numerous studies globally support a strong link between oral lichen planus (OLP) and HCV infection, though this association may not be significant in some regions.

Therefore, further prospective and interventional studies are needed to better understand these controversial findings.

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