



Case Report

Asymptomatic Hepatitis C in Patient with Oral Lichen Planus: A Case Report

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Abstract: Lichen planus (LP) is a chronic inflammatory condition that affects the oral mucous membrane/skin with a variety of clinical presentations. Oral Lichen Planus (OLP) is considered to be associated with numerous systemic conditions one of which includes hepatitis C virus (HCV) infection. The geographic variation of the hepatitis C prevalence proved to be an important factor influencing the statistical results of the studies analyzing the association of the oral plan lichen with the hepatitis C virus. LP patients have about a fivefold higher risk than the controls of being HCV seropositive, with geographic variability, being most prevalent in the region of Japan, Mediterranean countries and the USA. Treatment outcomes in patients with oral lichen planus associated with chronic hepatitis C virus are often unsatisfactory compared to patients suffering from idiopathic oral lichen planus. Also, the evolution of oral lesions is often fluctuating, with repeated periods of relapse according to the degree of liver function decompensation. Herewith, a case of oral lichen planus in a female patient is reported, which on investigation revealed hepatitis C antibodies.

Keywords: Female, Hepatitis C, Lichen Planus, Oral

1. Introduction

Lichen planus (LP) is a chronic inflammatory condition that affects the oral mucous membrane/skin with a variety of clinical presentations. Oral Lichen Planus (OLP) is considered to be associated with numerous systemic conditions one of which includes hepatitis C. Hepatitis C Virus (HCV) infection is one of the major causes of chronic hepatitis but if it is untreated may progress to cirrhosis or hepatocellular carcinoma over a period of time. Oral lichen planus is the one of the extra-hepatic disorders associated with chronic HCV infection. OLP patients have about a fivefold higher risk than the controls of being HCV seropositive, being most prevalent in the region of Japan, Mediterranean countries and the USA.

[1] The HCV positive patient may require particular

management when dental treatment is required due to bleeding disorders, defective metabolism of many drugs and the risk of nosocomial transmission of HCV. Thus testing for HCV patients with LP can lead to the early diagnosis and treatments and precautions can be useful to avoid further spread. Moreover, because chronic HCV infection can lead to cirrhosis and hepatocellular carcinoma and OLP is a potentially malignant disorder, an early diagnosis and a proper management might save lives and being beneficial in reducing health care costs. In the present case, a female reported with oral lichen planus, which on investigation revealed hepatitis C antibodies. Surprisingly, the patient was completely unaware of this condition, being discovered by chance during

diagnostic protocol for oral lichen planus.

2. Case Presentation

2.1. Ethics Statement

Informed consent has been obtained from the patient and this investigation has been conducted according to the principles expressed in the Declaration of Helsinki.

2.2. Case Report

A 50-year old female reported to the department of oral medicine and radiology with a chief complaint of burning sensation in mouth on eating spicy food since last six month. History revealed that burning was more on tongue and on the inner aspects of both right and left cheeks. Past medical and dental and the family history was not significant. Also the personal history did not reveal any deleterious habit. On general physical examination, patient was moderately built, well oriented to time, place and the person. On intra oral clinical examination, there was well defined grayish white patch on right buccal mucosa and linear reticular pattern on the left buccal mucosa. On the dorsum of the tongue similar grayish white areas were present, covering whole of the surface. On palpation all the patches were non scrapable, non tender and rough. Based on the history and clinical examination, a provisional diagnosis of oral lichen planus was made and routine blood investigations were advised. All the blood investigations were within the normal limit, except that the serum was positive for Hepatitis C antibodies, performed by Tridot test. Incisional biopsy of the lesion was done and the lesion was confirmed as lichen planus histopathologically. The patient was managed with topical Triamcinolone acetonide (0.1%) 3-4 times daily, as topical application after food and at bed time, to increase its retention. Also Lycopene, as an antioxidant was prescribed in the dose of 8mg once daily after food for three months. Within first week, there was symptomatic relief to the patient. Although the patient's medical history was not significant for any systemic condition, but being HCV positive on diagnostic protocol for OLP, patient was referred to the general physician for further evaluation of hepatic status. Patient was advised regular follow up too.

3. Discussion

Oral lichen planus is a chronic inflammatory condition of oral mucous membranes. Prevalence of oral lichen planus in the community is 1-4%. [2] The various etiological factors include autoimmune disease, drug reaction, diabetes mellitus, hypertension, kidney stones, psychological factors, bacterial infection and several viruses, including; herpes viruses, immunodeficiency virus, papilloma-virus, the hepatitis viruses B and C. [2, 3] Although the exact etiopathogenesis of OLP is not clear, but it is suggested that it has an auto-immune nature, mediated by T CD⁸⁺ cells, macrophages and Langerhans cells. Langerhans cells and macrophages in the

basal epithelial layer provide antigenic information for T CD⁸⁺ cells activated against basal layer. Cell destruction is done through immune mediated apoptosis, resulting in the appearance of characteristic histological changes. [4] OLP usually occurs bilaterally on the buccal mucosa and also involves the tongue, gingiva and other sites. OLP involves females 1.4 times more than males, mostly between the ages of 50 to 60. [5]

Clinically, OLP can present papular, reticular, plaque type, erosive and ulcerative form. The disease is usually asymptomatic, but patient may present with pain or irritation or a feeling of roughness of mucosa.

Hepatitis C virus (HCV), being discovered by Patrick in 1989, belongs to the family *Flaviviridae* and has a positive sense single-stranded RNA genome. [6] It is estimated that 70% to 80% of the patient with HCV enter in the chronic phase of the disease. [7] HCV is known to be a leading cause of chronic hepatic diseases and hepatocellular carcinoma. Chronic hepatitis C is often asymptomatic and is usually discovered accidentally, like in the present case. The diagnosis is based on detection of antibodies against HCV (anti-HCV). The third-generation enzyme-linked immunosorbent assay (ELISA) has 99% sensitivity in detecting total antibodies with 94% specificity and can be confirmed by direct detection using HCV RNA. Extra hepatic complications of the HCV includes; thyroiditis, delayed skin porphyria, cryoglobulinemia, glomerulonephritis, sicca syndrome, thrombocytopenia, lichen planus, diabetes mellitus and lympho-proliferative disorders. These dermatological manifestations may serve as an early marker for HCV that may save lives. Therefore, evaluating the potential clinical role of OLP in diagnosing HCV infection seems to be an extremely practical and pivotal task. [8, 9]

The prevalence of the chronic hepatitis in the patients with OLP has been found to be between 0.5-3.5% in studies conducted in various geographic areas. [1] In 1991, Monki *et al* reported the first case of histologically confirmed OLP in a patient with chronic active HCV, suggesting an association between the two. These mucosal/dermatological manifestations may serve as an early marker for the diagnosis of HCV in asymptomatic carriers. Most cases of oral lichen planus associated with HCV have been reported from studies in Mediterranean area whereas, in countries with highest prevalence of HCV (such as Egypt and Nigeria), no significant correlation has been established between HCV and OLP. [10] Although the manner in which the HCV infection predisposes patients to the development of LP remains unclear. Some speculate that long-term infection may lead to an aberrant immunologic response so suggesting that OLP may be triggered by HCV. [15] Other studies [12, 1] suggest that host immune response rather than the viral factors is of greater importance in the development of OLP lesions in HCV infected patients. The role of environmental or geographical factors, however, is less clear. It has been suggested that geographic localization could explain the different association between HCV and OLP. A higher frequency of HCV infections in certain countries would increase the chance that a

patient with OLP would also be infected with HCV. [13] Mega *et al* [14] noted three types of OLP: OLP associated with a HCV infection (OLPHCV), Oral lichen-contact sensitivity reaction (OLCSR) and idiopathic oral lichen planus (IOLP), all of which exhibited a similar histopathology. However, some OLPHCV cases showed deeper lymphocyte infiltration in the lamina propria than in IOLP, and more extensive and aggressive lymphocyte infiltration could be associated with the erosive manifestation of OLP-HCV. Immunohistochemically, a similar distribution of the CD⁴ cells was observed in all the OLP groups. Although the pathogenesis of OLP-HCV remains to be unclear, some reports have supported a host factor caused by HCV infection rather than a reaction to local HCV as an exogenous antigen. This is supported by significantly increased levels of the soluble intercellular adhesion molecule (ICAM)⁻¹ and IgG in OLP-HCV patients. Furthermore, HCV replication has been reported in the epithelial cell of oral mucosa from HCV patients with and without OLP, using both reverse transcriptase PCR, and in-situ hybridization. HCV as well as other factors seem to be responsible for OLP-HCV. In the lamina propria of OLP-HCV, the different function of the CD⁸⁺ cells have been reported in vitro (20). Langerhans cells, which play a key role in the presentation of antigen in OLP have been reported as either increased in number or activated in OLP, showing the expression of S-100, HLA-DR and CD¹. [12] Lazaro *et al* [15] have demonstrated that HCV infects keratinocytes from cutaneous LP lesions and that the viral RNA is translated in these cells as demonstrated by the HCV incorporated in the skin biopsies. Klanrit *et al* [16] showed a small but significant percentage of HCV infected patients with OLP. The study of Harman *et al* [11] supported the view that the consistency of HCV and OLP is probably more than coincidental and they recommend that it is appropriate to screen all patients with LP for HCV infection.

It has been observed that, patients who have any form of chronic liver disease generally show extensive forms of oral lichen planus, with frequent episodes of exacerbation of lesions, which are refractory to treatment. Often the acute, erosive form of OLP associated with HCV in an active stage is biochemically revealed by elevated levels of serum transaminases and accelerated viral replication. So in such situations, it is mandatory to investigate the hepatitis and an interdisciplinary approach with the specialists of infectious diseases for appropriate therapy. Another important aspect is the need to test all patients diagnosed with oral lichen planus for the presence of hepatitis C virus as the patients are often asymptomatic and unaware of their condition, as reported in the present case. [17]

4. Conclusion

Oral lichen planus may be the first extra-hepatic manifestation of HCV infection and as HCV is associated with significant morbidity and mortality, it is important for the clinician to subject the patients suspected of OLP, for liver function tests and hepatitis C antibodies detection to prevent

any incidental transmission of HCV to health care workers. Also the association of oral lichen planus with HCV may result in a longer evolution of oral lesions of lichen planus, with repeated exacerbations. The type of OLP frequently associated with chronic hepatitis C is the erosive-ulcerative type, accompanied by acute symptoms. Results from literature have shown that the prevalence of hepatitis C in patients with oral lichen planus is higher compared to the general population; these values vary to a great extent because chronic HCV has a world-wide distribution which is different from one geographical region to another. Chronic hepatitis C virus can remain asymptomatic for a long time, but patients can present a number of extrahepatic manifestations such as those in the oral cavity. Dentist has an active role in detecting infection with hepatitis C. On the other hand, given the risks, the dental treatment of patients with known chronic HCV liver disease raise special problems.

References

- [1] Lodi G, Pellicano R, Carrozzo M. Hepatitis C virus infection and lichen planus: a systematic review with meta-analysis. *Oral Dis.* 2010; 16: 601-612.
- [2] Gupta S, Jawanda MK. Oral lichen planus: An update on etiology, pathogenesis, clinical presentation, diagnosis and management. *Indian J Dermatol.* 2015; 60: 222-229.
- [3] Shimoyama T, Horie N, Kato T, Kaneko T, Komiyama K. Helicobacter pylori in oral ulcerations. *J Oral Sci.* 2000; 42: 225-259.
- [4] Sugeran PB, Savage NW, Walsh LJ, Zhao ZZ, Zhou XJ, Khan A, Seymour GJ, Bigby M. The pathogenesis of oral lichen planus. *Crit Rev Oral Biol Med.* 2002; 13: 350-365.
- [5] Machado AC, Sugaya NN, Migliari DA, Matthews RW. Oral lichen planus: Clinical aspects and management in fifty-two Brazilian patients. *West Indian Med J.* 2003; 52: 203-207.
- [6] White CP, Hirsch G, Patel S, Adams F, Peltekian KM. Complementary and alternative medicine use by patients chronically infected with hepatitis C virus. *Can J Gastroenterol.* 2007; 21: 589-595.
- [7] Harden D, Skelton H, Smith KJ. Lichen planus associated with hepatitis C virus: no viral transcripts are found in the lichen planus, and effective therapy for hepatitis C virus does not clear lichen planus. *J Am Acad Dermatol.* 2003; 49: 847-852.
- [8] Pilli M, Penna A, Zerbini A, Vescovi P, Manfredi M, Negro F, Carrozzo M, Mori C, Giuberti T, Ferrari C, Missale G. Oral lichen planus pathogenesis: a role for the HCV-specific cellular immune response. *Hepatology.* 2002; 36: 1446-1452.
- [9] Cacoub P, Comarmond C, Domont F, Savy L, Desbois AC, Saadoun D. Extrahepatic manifestations of chronic hepatitis C virus infection. *Therapeutic Advances in Infectious Disease.* 2016; 3: 3-14.
- [10] Ibrahim HA, Baddour MM, Morsi MG, Abdelkader AA. Should we routinely check for hepatitis B and C in patients with lichen planus or cutaneous vasculitis? *East Mediterr Health J.* 1999; 5: 71-78.

- [11] Harman M, Akdeniz S, Dursun M, et al. Lichen planus and hepatitis C virus infection: an epidemiologic study. *Int J Clin Pract* 2004; 58: 1118-1119.
- [12] Nagao Y, Sata M, Itoh K, et al. Quantitative analysis of HCV RNA and genotype in patients with chronic hepatitis C accompanied by oral lichen planus. *Eur J Clin Invest* 1996; 26: 495-498.
- [13] Figueiredo LC, Carrilho FJ, de Andrade HF, Migliari DA. Oral lichen planus and hepatitis C virus infection. *Oral Dis* 2002; 8: 42-46.
- [14] Mega H, Jiang WW, Takagi M. Immunohistochemical study of oral lichen planus associated with hepatitis C virus infection, oral lichenoid contact sensitivity reaction and idiopathic oral lichen planus. *Oral Dis* 2001; 7: 296-305.
- [15] Lazaro P, Olalquiaga J, Bartolome J, et al. Detection of hepatitis C virus RNA and core protein in keratinocytes from patients with cutaneous lichen planus and chronic hepatitis C. *J Invest Dermatol* 2002; 119 (4): 798-803.
- [16] Klanrit P, Thongprasom K, Rojanawatsirivej S, et al. Hepatitis C virus infection in Thai patients with oral lichen planus. *Oral Dis* 2003; 9 (6): 292-7.
- [17] Beard LM, Kahloon N, Franco J, Fairley JA. Incidence of hepatitis c in lichen planus. *J Am Acad Dermatol.* 2001; 44: 311-312.