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REVIEW

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A review on interleukins (IL10 and IL17) as biomarkers for hepatitis C-associated oral lichen planus

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Abstract

Background: Hepatitis C virus is a viral infection associated with autoimmune disorders. This virus has hepatic and extrahepatic manifestations. One of the extrahepatic manifestations associated with the hepatitis C virus includes oral lichen planus. Oral lichen planus is an autoimmune disorder mainly affecting the tongue and buccal mucosa. It clinically represents grayish-white striae bilaterally on the buccal mucosa. The pathogenesis involves the progression of the hepatitis C virus, and oral lichen planus affects T lymphocytes. Specific proteins and cytokines activate these T lymphocytes, which act as biomarkers to detect certain diseases. Interleukin 10 is an anti-inflammatory cytokine, whereas interleukin 17 is a pro-inflammatory cytokine. These cytokines have a pathophysiological role and act as biomarkers for many diseases. Therefore, this review article aims to establish the role of interleukin 10 and interleukin 17 as biomarkers for hepatitis C-associated oral lichen planus.

Conclusion: Hepatitis C virus is an infectious disease that can lead to liver cirrhosis, and oral lichen planus is a premalignant lesion that can lead to oral carcinoma. As interleukin 10 lessens the immune pathologies and interleukin 17 mediates proinflammatory response, therefore, these biomarkers have a role in progression of these diseases.

Keywords: Interleukin 10 (IL-10), Interleukin-17 (IL-17), Oral lichen planus (OLP), Hepatitis C (HCV)

Background

Hepatitis C virus (HCV) is a viral infection related to liver carcinoma and other chronic liver conditions [1]. It was first discovered in 1989 that HCV is an RNA virus and is transmitted through contaminated blood of that virus [2–4]. The diagnosis of hepatitis in the patient remains unknown and is identified when he suffers from other diseases like OLP [5]. This virus also affects hepatic and non- hepatic cells [1, 6]. Geographic location impacts HCV as it shows the highest number of patients in Egypt and the lowest in the UK [7]. Its histological picture shows mono-nucleated and lymphoid follicular cells in the liver of a deceased patient [1].

Lichen planus (LP) is a dermatological disease affecting the skin and oral mucosa. Oral lichen planus (OLP) is mainly a disease of oral mucosal involving the tongue and buccal mucosa [3, 4]. Middle-aged females are at higher risk of having this disease with 0.2–2.3%, affecting the general population worldwide. OLP should be distinguished from oral lichenoid lesion which occurs due to dental material specially metallic material, whereas the etiology of OLP is still unknown. It appears as a white lesion on the buccal mucosa [3, 6]. Histopathological factors involved in OLP are CD8-mediated T-cells and macrophages, which target the basal layer, causing apoptosis of cells [5, 8, 9].

Cytotoxic and helper T cells are precisely involved in the pathophysiology of OLP. Sugerman et al. presented CD4+ and CD8+ T cell interaction through cell surface molecules involving non-specific and specific mechanisms [10]. These immune cells synthesize numbered

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proteins that act as cytokines to promote the recruitment and migration of T lymphocytes and macrophages [11]. Interleukin 10 is an anti-inflammatory cytokine [12], and interleukin 17 is a pro-inflammatory cytokine [13].IL10 also regulates the production of Th1 and Th2. These cells help in the host mechanism against infection. Therefore, the imbalance between these cells can cause many chronic infections and immunological disorders [14]. This function has also been linked with interleukin 17 (IL17), secreted during inflammation in many infectious diseases. Although both are inflammatory diseases leading to carcinomas in chronic stages, pathogenesis involving certain specific biomarkers needs to be studied. Therefore, this study aims to review biomarkers involved in the pathogenesis of HCV and OLP.

Main text

Hepatitis C virus (HCV) was first discovered in 1989 as a blood-borne disease, non-A and non-B hepatitis [15]. It is an RNA virus belonging to the Flaviviridae family [16], which has a severe disease burden on the population worldwide. This virus can also be symptomatic and asymptomatic, infecting 50% of the population [17]. Mainly, two types of hepatitis virus are present acute, which is usually asymptomatic with no progression towards liver cirrhosis, and chronic which can lead to liver cirrhosis [18]. The prevalence of HCV is 2.2% globally, whereas, in Pakistan, it reaches up to 4.5 to 8% of the population [19]. The transmission of this virus is due to the pricking of used needles and blades. It can also transfer from the infected mother to the child. Intravenous drug users, hemodialysis patients, and people with high sexual activity has higher risk of transmitting this virus [20]. Certain extrahepatic manifestations can occur following the hepatitis C virus. Of these extrahepatic manifestations, lichen planus (LP) has a higher prevalence [21].

Lichen planus (LP) is mainly a disease infecting the skin and oral mucosa. LP involving the oral mucosa is oral lichen planus (OLP) which can be seen on the tongue and buccal mucosa [5]. According to WHO, OLP can be diagnosed clinically and histologically [22]. Clinically, it presents as a bilateral lesion with a mild burning sensation. OLP is divided into reticular, erosive, bullous, and papular types. They are present bilaterally on the buccal mucosa, gingiva, and at the dorsum of the tongue. Histologically, it manifested as breakage of epithelium and basal layer. This lesion should have well-defined cellular penetration of lymphocytes with the liquefaction and disintegration of the basal cell layer [23]. In the general population, the prevalence of OLP is 0.1 to 4% [24]. Since OLP has an unclear etiology, certain factors are involved in its pathogenesis, which includes trauma, stress, and infectious viruses like herpes virus, cytomegalovirus, Epstein bar virus, and hepatitis viruses [25].

The first association between HCV and LP was published in 1991, and between HCV and OLP was published in 1994 [7]. Many studies have been reported to find the association between the two diseases. Sharma et al. studied the hepatitis C virus among lichen planus patients in 2020 in Nepal. In his study, a total of sixty-eight confirmed cases of different types of lichen planus were included, of which 30 were OLP, and 36 were cutaneous LP. He concluded only two cases were positive for hepatitis C virus, which is a limited resource to support the association [26]. Another study was conducted in Turkey in 2019 by Fatma et al. to find an association between HCV and LP. In this study, 145 patients diagnosed with LP were included, and their anti-HCV test was done. He stated no detection of chronic HCV infection in LP-diagnosed patients showed no association between HCV and LP, which could be because of the lower prevalence of HCV in their country [27]. The occurrence of the hepatitis C virus with oral lichen planus was assessed by Alaparthi et al. in India in 2020. This study includes 50 OLP patients and does not detect any association between OLP and HCV, and this could be due to genotyping discrepancy of HCV [28]. Another study conducted in 2021 on the Kashmir population in India by Chalkoo et al. showed no strong correlation between OLP and HCV with insignificant results compared to controls [29]. Malik et al. studied oral lichen planus and hepatitis C virus in Lahore, Pakistan. In this study, 103 patients were selected and screened for OLP and HCV, of which only 16.5% showed the presence of both diseases. Therefore, Malik concluded the occasional presence of OLP with HCV [30].

Many studies have been conducted to study the pathogenesis of OLP as it is an autoimmune disease mediated by T lymphocytes [31]. In contrast, the hepatitis C virus is mainly triggered by T lymphocytes [32]. Georgescu et al. conducted a review in 2019 on the pathogenesis involved between LP and HCV and concluded that the lesion is because of the immune response to viral components [33]. Carrozzo et al. reviewed in 2019 in which he described the role of increased secretion of Th1 and Th2 mediated cytokines in OLP pathogenesis [34]. In contrast, Cerny et al. did a review in 1999 and reported clearance of virus from Th1 and Th2 cells [32]. Data from the literature indicated that pro-inflammatory and anti-inflammatory cytokines have a role in infectious and autoimmune-mediated conditions [35]. In 2021, Zheng-Da Zhu et al. studied salivary cytokines in patients with OLP. He concluded the positive role of the granulocyte-macrophage colony-stimulating factor in the severity

of OLP [36]. In 2018, Azab et al. conducted a study on interferon-gamma and interleukin 8 in HCV related to OLP patients and concluded that HCV infection could increase the severity of OLP [37]. Femiano et al. studied the functions of different cytokines with oral lichen planus and hepatitis C virus in 2005. They reported a decrease in pro-inflammatory cytokines and an increase in immunomodulant cytokines in erosive lichen planus and hepatitis C virus infection patients [38].

In 2003, Knapp et al. studied interleukin 10 promoter polymorphism and its outcome on hepatitis C virus. They concluded that interleukin 10 has a role in HCV infection and its progression towards liver fibrosis [39]. Serum levels of interleukin 4 and interleukin 10 were assessed in chronic hepatitis C virus patients by Reiser et al. and concluded elevated levels of both cytokines with chronic hepatitis C virus [40]. In another study, M.El Kady et al. studied cytokines patterns of interleukin 4, interleukin 10, interleukin 18, and interferon gamma in hepatitis C virus and *Schistosoma mansoni* infection patients. They observed increased serum levels of IL4 and IL10 in HCV coinfection [41]. Tarek Aboushosha et al. studied the expression of interleukin 4, interleukin 27, and CD163 in chronic hepatitis C patients and concluded higher scores of IL17 levels with HCV severity [42]. Another study was conducted in 2020 by Elbanan on interleukin 17 and transforming growth factor beta 1 in the hepatitis C patient group and observed increased levels of IL17 in the hepatitis C patient group compared to controls [43]. In 2019, Azatyan et al. conducted a study to evaluate levels of oral cytokines during the onset of inflammatory periodontitis among hepatitis C virus and other viruses and concluded a significant difference with higher mean levels of IL10 and IL2 in cases groups when compared to controls [44]. Afzal et al. reviewed the role of interleukin 10 in the hepatitis C virus among the Pakistani population and reported decreased levels of interleukin 10 with an increase in infection. He also stated that interleukin 10 could be used as antiviral therapy in chronic hepatitis C patients [45].

Just like hepatitis C virus, oral lichen planus is also involved with T lymphocytes. In 2009, Bai J et al. studied the association of polymorphism in tumor necrosis factor alpha and interleukin 10 with oral lichen planus patients in the Chinese population. They concluded the role of interleukin 10 in developing OLP [46]. Simark et al. studied the distribution of interleukin 2, interleukin 4, interleukin 10, tumor necrosis factor alpha, and transforming growth factor beta mRNA in oral lichen planus patients. They observed mRNA for pro-inflammatory and anti-inflammatory cytokines generated in chronic lesions of oral lichen planus [47]. Carrozzo et al., in 2004, studied the role of pro-inflammatory and anti-inflammatory cytokines in lichen planus and oral lichen planus patients and stated that pro-inflammatory cytokines have a role in the development of lichen planus and oral lichen planus [48]. Lu Rui et al. studied the regulatory roles of interleukin 23 and interleukin 17 and showed overexpression of IL17 levels in OLP patients compared to controls [49]. In Iran in 2017, Rezazadeh et al. studied salivary levels of interleukin 10 in oral lichen planus patients. They reported higher levels of interleukin 10 with a non-significant difference in the cases group compared to controls [50]. Another study was conducted in 2019, assessing serum and salivary levels of interleukin 17 in oral lichen planus patients. In this study, El-Refai et al. reported higher mean levels with significant results for the cases group compared to controls [51]. In 2017, Shiva Shirazian et al. studied salivary IL17 and IL22 levels in oral lichen planus and control groups. They found significant results for IL22 with increased mean levels and insignificant results for IL17 with increased mean levels [52]. Another study was conducted by Zhu et al. in 2015 in serum and saliva for interferon-gamma and interleukin 10 in different types of oral lichen planus. It detected higher levels of IL10 in serum and saliva in patients with erosive and reticular oral lichen planus [53].

We can summarize that IL10 has a role in immune responses by clearing out bacteria and viruses from the human body, whereas IL17 has a role in inflammation and autoimmune disease progression. Since HCV and OLP are inflammatory and autoimmune diseases respectively, these cytokines can progress the disease towards a chronic stage.

Conclusion

Hepatitis C virus is an infectious disease that can lead to liver cirrhosis, and oral lichen planus is a premalignant lesion that can lead to oral carcinoma. As interleukin 10 lessens the immune pathologies and interleukin 17 mediates pro-inflammatory response, therefore, these biomarkers have a role in the progression of both the diseases.

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Authors' contributions
First Author (SH) conducted the literature search and wrote an article's initial and final draft. Second Author (AC) conceived and designed the study and critically reviewed the final draft. Third Author (SS) conducted a literature search and wrote the initial draft of an article. Fourth Author (SF) wrote the initial draft of the article and provided a similarity index of the article. All the author(s) read and approved the final manuscript.

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References

1. Libra M, Indelicato M, De Re V, Zignego AL, Chiocchetti A, Malaponte G et al (2005) Elevated serum levels of osteopontin in HCV-associated lymphoproliferative disorders. Cancer Biol Therapy 4(11):1192–1194
2. Grossmann SMC, de Aguiar MCF, Teixeira R, do Carmo MAV. (2007) Oral lichen planus and chronic hepatitis C. Am J Clin Pathol 127(5):800–804
3. Gerayli S, Meshkat Z, Pasdar A, Mozafari PM, Banihashemi E, Khajavi MA, Rasekhi J. (2015) The association between oral lichen planus and hepatitis C virus infection; a report from northeast of iran. Jundishapur journal of microbiology. 8(4).
4. Lodi G, Giuliani M, Majorana A, Sardella A, Bez C, Demarosi F et al (2004) Lichen planus and hepatitis C virus: a multicentre study of patients with oral lesions and a systematic review. Bri J Dermatol 151(6):1172–1181
5. Gheorghe C, Mihai L, Parlatescu I, Tovaru S (2014) Association of oral lichen planus with chronic C hepatitis. Review of the data in literature. Maedica. 9(1):98
6. Song J, Zhang Z, Ji X, Su S, Liu X, Xu S et al (2016) Lack of evidence of hepatitis in patients with oral lichen planus in China: a case control study. Med Oral Patol Oral Cir Bucal 21(2):e161
7. Carrozzo M, Scally K (2014) Oral manifestations of hepatitis C virus infection. World J Gastroenterol. 20(24):7534–7543
8. Alaizari NA, Al-Maweri SA, Al-Shamiri HM, Tarakji B, Shugaa-Addin B. (2016) Hepatitis C virus infections in oral lichen planus: a systematic review and meta-analysis. Australian dental journal. 61(3):282-7.
9. Zhou ZT, Wei BJ, Shi P (2008) Osteopontin expression in oral lichen planus. J Oral Pathol Med 37(2):94–98
10. Alrashdan MS, Cirillo N, McCullough M (2016) Oral lichen planus: a literature review and update. Arch Dermatol Res 308(8):539–551
11. Onishi RM, Gaffen SL (2010) Interleukin-17 and its target genes: mechanisms of interleukin-17 function in disease. Immunology. 129(3):311–321
12. Moore KW, O'garra A, RdW M, Vieira P, Mosmann TR (1993) Interleukin-10. Ann Rev Immunol 11(1):165–190
13. Miossec P, Korn T, Kuchroo VK (2009) Interleukin-17 and type 17 helper T cells. New Engl J Med 361(9):888–898
14. Dogra G, Chakravarti A, Kar P, Chawla YK (2011) Polymorphism of tumor necrosis factor–α and interleukin-10 gene promoter region in chronic hepatitis C virus patients and their effect on pegylated interferon–α therapy response. Human Immunol 72(10):935–939
15. Choo Q-L, Kuo G, Weiner AJ, Overby LR, Bradley DW, Houghton M (1989) Isolation of a cDNA clone derived from a blood-borne non-A, non-B viral hepatitis genome. Science. 244(4902):359–362
16. Lindenbach B, Rice C (2001) Flaviviridae: the viruses and their replication, vol 1. Lippincott Williams & Wilkins, Philadelphia
17. Maticic M (2003) Hepatitis C virus infection: the dermatological perspective. Acta Dermatovenerol Alpina Panonica Adriatica. 12(1):19–27
18. Modi A, Liang T (2008) Hepatitis C: a clinical review. Oral diseases. 14(1):10–14
19. Jiwani N (2011) A silent storm: hepatitis C in Pakistan
20. Memon M, Memon M (2002) Hepatitis C: an epidemiological review. J Viral Hepatitis. 9(2):84–100
21. Zignego A, Ferri C, Pileri S, Caini P, Bianchi F (2007) Extrahepatic manifestations of Hepatitis C Virus infection: a general overview and guidelines for a clinical approach. Digest Liver Dis 39(1):2–17
22. Kramer I (1978) Definition of leukoplakia and related lesions: an aid to studies on oral precancer. Oral Surg Oral Med Oral Pathol 46:518–539
23. Krupaa RJ, Sankari SL, Masthan K, Rajesh E (2015) Oral lichen planus: An overview. J Pharm Bioallied Sci 7(Suppl 1):S158
24. Gichki AS, Ul-Haq Z, Ahmed K (2015) Oral Lichen Planus Among Dental Patients in Sandeman Provincial Hospital Quetta. Journal of khyber college of dentistry. 6:7
25. Qazi JA, Qazi AH. (2011) The Association Between Oral Lichen Planus and HCV Infection. Journal of the Dow University of Health Sciences (JDUHS). 5(1):3-5.
26. Sharma R, Paudel S (2020) Venereology, Leprology. Prevalence of hepatitis C among lichen planus patients. Nepal J Dermatol Venereol Leprol 18(1):15–17
27. Akyurek FT, Kurtipek GSJMS (2019) Is there really association between lichen planus and hepatitis. C? 8(4):913–915
28. Alaparthi R, Yalamanchili S (2020) Prevalence of hepatitis C virus infection in patients with oral lichen planus in Guntur, Andhrapradesh. Indian J Clin Res Dent 1(2):37–40
29. Chalkoo AH, Begum S (2021) Association between oral lichen planus and hepatitis C virus infection among Kashmir population
30. Anwar MA, Iqrar S, Akram Z, Tayyab MA, Afzal N (2018) Oral lichen planus and hepatitis C virus infection; a symbiotic relationship or a mere co-incidence? Int J Res Med Sci 6(3):772
31. Liu G-X, Sun J-T, Yang M-X, Qi X-M, Shao Q-Q, Xie Q et al (2011) OPN promotes survival of activated T cells by up-regulating CD44 in patients with oral lichen planus. Clin Immunol 138(3):291–298
32. Cerny A, Chisari FV (1999) Pathogenesis of chronic hepatitis C: immunological features of hepatic injury and viral persistence. Hepatology. 30(3):595–601
33. Georgescu SR, Mitran MI, Mitran CI, Sarbu MI, Nicolae I, Matei C et al (2019) Potential pathogenic mechanisms involved in the association between lichen planus and hepatitis C virus infection. Exp Ther Med 17(2):1045–1051
34. Carrozzo M, Porter S, Mercadante V, Fedele SJP (2019) Oral lichen planus: a disease or a spectrum of tissue reactions? Types, causes, diagnostic algorhythms, prognosis, management strategies. Periodontol 2000 80(1):105–125
35. Xavier GM, Sá AR, Guimarães ALS, Silva TA, Gomez RSJJoOP, Medicine. (2007) Investigation of functional gene polymorphisms interleukin-1β, interleukin-6, interleukin-10 and tumor necrosis factor in individuals with oral lichen planus. J Oral Pathol Med 36(8):476–481
36. Zhu ZD, Ren XM, Zhou MM, Chen QM, Hua H, Li CLJJoDS. (2021) Salivary cytokine profile in patients with oral lichen planus. Journal of Dental Sciences.
37. Azab NA, Abd El Salam L, Ahmed E, El Sharkawy M, ElSharkawy A, El Asheiry SGJ (2018) Interferon gamma and interleukin 8 gene polymorphisms in patients with hepatitis C virus related oral lichen planus. Arch Oral Biol 96:189–194
38. Femiano F, Scully C (2005) Functions of the cytokines in relation oral lichen planus-hepatitis C. Med Oral Patol Oral Cir Bucal 10(Suppl 1):E40–EE4
39. Knapp S, Hennig BJ, Frodsham AJ, Zhang L, Hellier S, Wright M et al (2003) Interleukin-10 promoter polymorphisms and the outcome of hepatitis C virus infection. Immunogenetics. 55(6):362–369
40. Reiser M, Marousis CG, Nelson DR, Lauer G, González-Peralta RP, Davis GL et al (1997) Serum interleukin 4 and interleukin 10 levels in patients with chronic hepatitis C virus infection. J Hepatol 26(3):471–478
41. El-Kady IM, Lotfy M, Badra G, El-Masry S, Waked I (2005) Interleukin (IL)-4, IL-10, IL-18 and IFN-γ cytokines pattern in patients with combined hepatitis C virus and Schistosoma mansoni infections. Scand J Immunol 61(1):87–91
42. Aboushousha T, Emad M, Rizk G, Ragab K, Hammam O, Fouad R et al (2021) IL-4, IL-17 and CD163 Immunoexpression and IL-6 gene

polymorphism in chronic hepatitis C patients and associated hepatocellular carcinoma. Asian Pac J Cancer Prev 22(4):1105–1113
43. Elbanan W, Fathy S, Ibrahim R, Hegazy MJTB (2020) Assessment of interleukin 17 and transforming growth factor-beta 1 in hepatitis C patients with disease progression. Trop Biomed 37(4):1093–1104
44. Azatyan V, Yessayan L, Shmavonyan M, Melik-Andreasyan G, Perikhanyan A, Porkshenyan KJTJoliDC. (2019) Evaluation of IL-2, IL-10, IL-4 and ϒ-interferon levels in the oral fluids of patients with hepatitis C. B and HIV. 13(05.1):069S–074S
45. Afzal A, Zain M, Sumra AA, Ali A, Akhtar T, Ashfaq T, et al. (2021) Role of Interleukin 10 in Hepatitis C Viral Clearance and Distribution of its Polymorphism in Pakistani Population. International Journal on Emerging Technologies. 12(1).
46. Bai J, Jiang L, Lin M, Zeng X, Wang Z, Chen Q (2009) Association of polymorphisms in the tumor necrosis factor-α and interleukin-10 genes with oral lichen planus: a study in a Chinese cohort with Han ethnicity. J Interf Cytokine Res 29(7):381–388
47. Simark-Mattsson C, Bergenholtz G, Jontell M, Eklund C, Seymour G, Sugerman P et al (1999) distribution of interleukin-2,-4,-10, tumour necrosis factor-α and transforming growth factor-β mRNAs in oral lichen planus. Arch Oral Biol 44(6):499–507
48. Carrozzo M, Dametto E, Fasano ME, Arduino P, Broccoletti R, Vezza D et al (2004) Tumor necrosis factor-α and interferon-γ polymorphisms contribute to susceptibility to oral lichen planus. J Invest Dermatol 122(1):87–94
49. Lu R, Zeng X, Han Q, Lin M, Long L, Dan H, et al. (2014) Overexpression and selectively regulatory roles of IL-23/IL-17 axis in the lesions of oral lichen planus. Mediators of inflammation Journal. 2014.
50. Rezazadeh F, Shahbazi F, Andisheh-Tadbir AJCCP (2017) Evaluation of salivary level of IL-10 in patients with oral lichen planus, a preliminary investigation. Comp Clin Pathol 26(3):531–534
51. El-Refai I, Maged A, El-Saady D (2019) Assessment of IL-17 in oral lichen planus and in pemphigus vulgaris. Egypt Dental J 65(1-January (Oral Medicine, X-Ray, Oral Biology & Oral Pathology):343–350
52. Shirazian S, Aghahosseini F, Salehi E, Vatanpour M, Banijamali SN, Pourshahidi SJBBRC (2017) Comparison of interleukin 17 and 22 in saliva of oral lichen planus patients with healthy people. Biosci Biotechnol Res Commun 10:587–591
53. Zhu J-h, Liu N, Zhao C-r, Liu J-G (2015) Interferon-gamma and interleukin-10 levels in serum and saliva are related to different types of oral lichen planus. Chin JTissue Eng Res 19(2):236

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