A Review

Association of Lichen Planus With Hepatitis C Virus

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Abstract

The relationship between chronic hepatitis C virus infection (HCV) and oral lichen planus (OLP) is a current topic in the field of oral medicine. Many studies of this association have been made over time. 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 lichen planus with the hepatitis C virus. Approaching this issue is not to be neglected. 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. Background therapy for liver disease itself may influence lichen planus lesions. Thus, during therapy with interferon and ribavir in oral lesions may appear or become acute.

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INTRODUCTION

Lichen planus was firstly described in 1869 by Jonathan Hutchinson as a unique inflammatory cutaneous and mucous membrane reaction pattern of unknown etiology which is characterized by small violaceous flat angular scaly shiny pruritic papules and plaques on the skin and white papules in the mouth. [1]

There are several clinical forms of Lichen Planus and they are; actinic, annular, atrophic, erosive, follicular (lichen planopilaris), guttate, hypertrophic, linear, papular and vesiculobullous. Skin lesions are characterized by a white lace-like pattern (Wickham's striae) on papules and are almost associated with lichen planus. Mucous membrane involvement can be seen in 40–60% of patients with skin lesion.[2]

Hepatitis C virus (HCV) on the other hand is a single-stranded RNA virus that replicates in hepatocytes and parts of peripheral blood mononuclear cells. The diagnosis can be done on detection of antibodies against HCV (anti-HCV). The third-generation enzyme-linked immunosorbent assay (ELISA) has proved to be 99% sensitive in detecting total antibodies with 94% specific and it can further be confirmed by direct detection using HCV RNA.

The association of LP and HCV is very uncertain and controversial in literature because the prevalence of HCV infection in patients with LP varies considerably and depends on various factors including from one geographical area to another. It has been suggested and proven in literature that the geographical origin of patients would be an important factor in HCV prevalence among patients with LP.[3]

Discussion on Association Between LP & HCV

The pathogenesis of this association is very uncertain and there are several reasons that had been suggested by several researchers and studies have also been conducted to co relate the association between these two lesions. One of the more common given explanation is the reason that HCV infection precipitates an auto immune process were-as LP appears to be related to the pattern of immune dysregulation induced by HCV. The mechanism of HCV induced lichen planus is more possibly related to the viral replication in lymphocytes and it is well known that one of the common and characteristic histological features of LP is band like lymphocytic infiltration in the papillary dermis or which can possibly be the reason why HCV is related in a way or another to the basal cell layer of the epidermis to which the lymphocytic band

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like infiltrate seems to be directed and once all these cells are destroyed, the same infiltrate moves down to the upper dermis.[2]

It was Mokni et al who were the first to note the association between HCV and lichen planus (LP) in 1991. They have reported a case of a patient who was presented with an eruption consisting of violaceous papules disseminated on his arms and trunk. Laboratory findings were done and showed elevated transaminases and further tests also confirmed the diagnosis of HCV infection. A cutaneous biopsy was done which established the diagnosis of LP. Later on after three years, the first cases of oral lichen planus (OLP) in association with HCV infection were notified. These studies were later on published shortly after the isolation of HCV in 1989.[4]

It has been seen that in cases of LP that twenty percent of patients have mucous membrane lesions only and these mucosal lesions are usually asymptomatic and women outnumber men by more than 2:1. Ulcerative mucosal disease is considered to be premalignant in nature. Koebner's phenomenon which is appearance of lesions in areas of trauma is often seen. The cause of LP is unclear and unknown. The most acceptable hypothesis suggests a viral cause, immunological or emotional stress. Lichenoid eruptions include drug induced, chemical, bacterial and post bone marrow transplantation. Lichenoid drug eruptions are morphologically almost similar to LP and can be differentiated by drug history only. In recent years it has been seen that HCV induces a broad spectrum of extrahepatic manifestations including porphyria cutanea tarda and LP. These dermatological manifestations can serve as an early marker for HCV that can save lives. Therefore, evaluating the potential clinical role of LP in diagnosing HCV infection seems to be an extremely practical and pivotal task.[5]

There have been cases where lichen planus has been associated with liver disorders, specifically chronic active hepatitis, in several well-executed studies that relate to the discovery of hepatitis C virus (HCV). HCV infection is present in other diseases of altered immunity, such as ulcerative colitis, vitiligo, alopecia aerata, morphea, and lichen sclerosis. HCV initially was not seriously considered a precipitating factor of lichen planus until various serological tests for HCV became available in 1990 which supported the associated between the two. There is an important association between hepatitis C and lichen planus, especially in those countries which have a higher incidence of hepatitis C infection. Liver abnormalities are frequently reported in patients with lichen planus. Chronic active hepatitis, especially due to hepatitis C virus, is suspected to be a major contributing factor and reason. The significant association between erosive lichen planus and chronic hepatitis has also been seen in many cases. More recently there have been various studies suggesting that these two conditions might be interrelated.[6]

The prevalence of HCV in patients with lichen planus varies considerably from one geographic area to another and also the incidence of HCV infection was higher in the expatriate patients. The incidence and prevalence of HCV infection varies by geographic region and can be seen as very high in Africa and the Eastern Mediterranean and low in Western Europe and North America. The highest prevalence was reported in Egypt, where approximately 20% of blood donors had HCV- Antibodies (World Health Organization). Across Europe there is a North-South gradient (prevalence of 0.5% in the north compared to 2% in the south) and an east-west gradient, with a lower prevalence among Western countries. Although the prevalence of infection in the general population is not well known, the following endemic areas can be identified:

- Low (below 1.5%) North America, Northern Europe;
- Medium (1.5-3.5%): South Asia, sub-Saharan Africa, Central and Latin America, the Caribbean, Oceania, Australia, Central and Eastern Europe, Western Europe;
- High (over 3.5%): Central and Eastern Asia, Middle East, North Africa.[7]

The explanations for this observation could be that expatriates belong to the countries in which a higher incidence of HCV infection and lichen planus has been reported. Lichen planus may represent a mucosal reaction to a variety of factors, including hepatitis C virus (HCV) infection. Lichen planus can be considered the first presentation of HCV because of its significant morbidity and mortality associated with HCV.[8] It has become very important for clinicians to actively look for HCV infection in patients with lichen planus. Lichen planus which appears to be related to the pattern of immune dysregulation induced by HCV, probably in a host with an underlying susceptibility for autoimmune disease. It would be worth doing a more board-based multi-centre trial involving cases of lichen planus and hepatitis C diagnosed clinically from different geographical areas and ethnic groups to determine the association between these two conditions.[9]

CONCLUSION

Association of oral lichen planus with chronic hepatitis C may result in a longer evolution of oral lesions of lichen planus, with repeated exacerbations depending on the degrees of liver function decompensation. Chronic HCV has various social implications and involves response throughout the whole body, having many extrahepatic manifestations also. The main type of lichen planus frequently associated with chronic hepatitis C is mainly the erosive-ulcerative type and also associated with acute symptoms and significant functional disorders which also affect the patient's quality of life. Results of studies in 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 worldwide distribution which is different from one geographical region to another.

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