Association of Lichen Planus with HCV and HBV in Nepal

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ABSTRACT

INTRODUCTION: Lichen Planus (LP) is a papulosquamous disorder of skin and mucous membrane, nail, hair. Cell –mediated immunity plays the major role in triggering the clinical expression of the disease. The hypothesis of viral etiological agent has gained association of the hepatotrophic viruses namely Hepatitis B and C viruses with LP in the past one decade.

METHODS: One hundred fifty patients clinically and histologically proven cases of LP of any age, sex, were enrolled in this study. Patients who satisfied the inclusion criteria were enrolled for detection of HCV antibodies and HbsAg Antigen by HCV TRI-DOT and HEPACARD respectively.

RESULTS: Lichen Planus patients had a mean age of 38.47±11.8Year with the range of 9-70 years. Out of one hundred fifty patients 67(44.6%) were male and 83(55.3%) were female. Seventy three (48.6%) cases were in third and forth decades only 3 children were below 12 years. Similar history in family was present only in 9 cases. Mean duration of disease was six to seven months, 70 cases (46.6%) being in the range of 0-8 months. Classical Lichen planus was present in 130(86.6%), oral LP 12 (8.0%) and Genital LP was present in 8(5.3%). None of hundred fifty patient with LP showed positive reaction for HbsAg, similarly none of cutaneous LP show positive reaction of HCV antibodies, but only 5 (41.6%) patients out of 12 oral LP were positive for HCV antibodies.

CONCLUSION: No association between HBV and LP was found in our population, only few oral LP was positive for HCV. In view of our result, we recommend that viral serology for Hepatitis B and C for LP patient may not be done as routine screening process. However long term follow up studies are needed.

KEYWORDS: lichen planus, Hepatitis, virus, HCV, HBV

INTRODUCTION

Lichen Planus (LP) is a Papulosquamous disorder of skin, mucous membrane, nail and hair characterized by pruritic violaceous papules with whitish streaks known as Wickham’s stria. Cell –mediated immunity plays a major role in triggering the clinical expression of the disease. Both CD4+ and CD8+ T cells are found in the skin lesion of LP. The hypothesis of viral aetiological agent has gained association of the hepatotrophic viruses namely Hepatitis B and C viruses with LP in the past one decade. Various dermatological conditions including LP have been found in association with these viruses.

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Hepatitis B virus has been associated with a number of hepatic and extra hepatic manifestations. It is mainly transmitted through blood and blood products. The prevalence of Hepatitis B virus in general population in Nepal is 0.3 to 4.0%. There are at least 350 million carriers of HBV world wide. The global prevalence of HBV infection varies from more than 8% in Africa, Asia and Western Pacific and less than 2% in Western Europe, North America and Australia.

Since 1991, many reports began to emerge about coexistence of LP with HCV infection. In studies from Japan, Italy, Spain, and Germany, high levels of HCV infection have been described in patients with LP. On the other hand, studies from Northern Europe including the UK, USA show no association.
The present study was carried out to ascertain the association of LP with HCV, HBV infection in our Nepalese population.

METHODS

This is an open, cross-sectional, quantitative, non randomized study carried out in tertiary care centre at Kathmandu University Teaching Hospital, Dhulikhel after obtaining permission from the institutional ethics committee.

One hundred and fifty clinically and histologically proven cases of LP of any age, sex, were enrolled in this study. The inclusion criteria included patients with clinical, and histological proven LP. A history of blood transfusion, history of alcohol intake and intravenous drug abuse, history of medication (Anti malarial, Thiazide, Beta-blocker, NSAID, ACE inhibitor), dental amalgam, HBV vaccination, liver disease and lichenoid drug eruption were excluded.

All diagnosed cases of LP were enrolled for HCV antibodies and HBsAg antigen detection. The kit used was Hepatitis C virus encoded antigens NS3, NS4, NS5. The fourth generation HCV TRI-DOT is a rapid visual, sensitive and qualitative test for the detection of antibodies to Hepatitis C virus in human serum or plasma.

Second kit used was Hepatitis B virus encoded antigen adw, adr, ayw and ayr by HEPACARD. It is a visual, rapid, sensitive and accurate immunoassay for qualitative detection of Hepatitis B surface antigen in human serum or plasma.

RESULTS

LP patients had a mean age of 38.47±11.8 years with the range of 9-70 years. Out of 150 patients, 67(44.6%) were males and 83(55.3%) were females. Seventy three (48.6%) cases were in third and fourth decades of life, only three were children of below 12 years of age. Similar history in family was present in nine cases. Mean duration of disease was six to seven months; 70 cases (46.6%) being in the range of zero to eight months. Classical LP was present in 130(86.6%), oral LP in 12 (8.0%) and genital LP in eight (5.3%). None of the 150 patients with LP showed positive reaction to HBsAg. Similarly, none of the cutaneous LP showed positive reaction to HCV antibodies. However, five (41.6%) out of 12 OLP patients were positive for HCV antibodies.
DISCUSSIONS

LP is a chronic papulosquamous disease of unknown etiology with prevalence up to 2%. Theories of infectious, autoimmune, genetic metabolic cause have been proposed. The first description of association of LP with HCV was reported in 1991. So far, many case control studies have been undertaken implicating HCV association with LP. Hepatitis C virus is a single-stranded RNA virus. About 3% of world population is infected with HCV. Most of the positive studies are from Japan, Spain and Italy and few parts of India. On the other hand, Northern UK studies have persistently failed to depict an association between Hepatitis C infected and LP. It is believed that this association might be related to cytotoxic immune response to epithelial cells infected with HCV.

In our study we found that five OLP patients out of 12 (41.6%) had positive antibodies for HCV (OLP seems to be significantly associated with HCV antibodies with 41.6%). Similar study was carried out in Japan where they reported the prevalence of OLP with HCV. Similarly an Italian study showed weak relation between HCV infection with OLP. Similarly, another two Italian studies, one Brazilian study, one Turkish study, and one Serbian study were not able to find any correlation between HCV infection and LP.

Many mechanisms have been proposed to explain the correlation of LP with HCV such as HCV’s capability of cytopathic replication in cell type outside the liver, autoimmune process against antigens expressed on extrahepatic cell, activated CD8 T cells and cytokines. Prevalence of HCV in Nepal is eight percent. In our study, only 3.3% were positive for HCV, that also, in oral LP. Comparing with international results, the authors feel that the relevance of co-existent HCV infection in LP remains a mystery. Prevalence of HCV antibodies is constant worldwide, ranging from 0.3% to 1.5%. Host-related factors including host immune-dysregulation or concomitant immunomodulatory infection play a role.

Hepatitis B virus has been associated with a number of hepatic and extra hepatic manifestations. Dermatological syndromes associated with HBV infection include: Henoch- Schonlein purpura, erythema multiforme, serum sickness-like syndrome, polyarteritis nodosa, essential cryoglobulinemia, toxic erythema and lichenoid dermatitis resembling lichen planus.

In Nepal, sero-prevalence of HBsAg has been reported ranging from 0.3 to 4.0% of which 1% of the population are asymptomatic chronic HBsAg carriers. In our study, out of 150 patients none were positive for HBV infection. There are many reports from different regions of world suggesting and negating the concept of association of HBV infection and LP. Observation of LP following HBV vaccination has been reported. A chronic graft versus host reaction (GVHR) - like autoimmune reaction has been suggested as a pathogenic mechanism. Many vaccines share only protein S as a common component. HbsAg is a mosaic of epitopes (S, pre S1 and pre S2) each of which is immunogenic. LP may develop as immune reaction to keratinocytes expressing S epitopes. Therefore in patients with chronic post-viral hepatitis, LP would be cytotoxic reaction to keratinocytes expressing HBsAg.

However, since the prevalence of HBsAg in patients with LP in this study was not statistically significant and since other serological markers of HBV was not assayed, it is difficult to deduce any relationship between the two diseases.

CONCLUSIONS

No association between HBV and LP was found in our population; only few OLP was positive for HCV. In view of our results, we recommend that viral serology for Hepatitis B and Hepatitis C for LP patient may not be done as a routine screening process. In case of OLP, however, Hepatitis C screening is advised. Nonetheless, long term follow up studies are needed to consider routine screening.

REFERENCES