
Lichen Planus and Hepatitis in Iraqi Patients

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Abstract

Background: With time, it is becoming that idiopathic lichen planus (L.P) is being observed more and more in conjunction with diseases of altered or disturbed immunity. Such autoimmune diseases include ulcerative colitis, alopecia areata, vitiligo, dermatomyositis, morphea and lichen sclerosus.

Objective: The aim of the study is to clarify the possible association between L.P and active chronic hepatitis especially hepatitis B virus (HBV) infection.

Patients & Methods: Fifty patients with lichen planus attending Department of Dermatology at Tikrit Teaching Hospital were enrolled in this study in the period December 2006 through to December 2006; their mean age (42.72), with standards deviation (± 12.72). A full history and clinical assessment for each patient with lichen planus and conforming diagnostic were performed. All patients were screened for hepatitis B surface antigen (HBs Ag), antibody to hepatitis B core antigen (anti-HBc), aspartate transaminase (AST), and alanine aminotransferase (ALT).

Results: were that lichen planus most common between 21 and 60 years were 60% males and 40% females. The percentage of new cases were 2%, the nonfamilial cases were 90% and familial cases 10%, most lesions on the cutaneous and mucosal (oral and genitalia) involvement (66%) and cutaneous only (34%). The dysfunction of liver test were positive in 16% and only (6%) from them have had HBsAg and anti-HBc positive.

Conclusions: It can be concluded that HBV infection is lower among lichen planus patients, only (6%) have had HBsAg and anti-HBc positive. Therefore, it seems prudent to screen all patients with lichen planus by liver function test. Liver disease may consider a risk factor for lichen planus although not a specific marker for it and should be receptive to signs or symptoms of liver disease when evaluating patients with lichen planus. The link between lichen planus and chronic active hepatitis infection continues to be investigated and debated.

Key Words: Lichen planus, Hepatitis.

Introduction:

Lichen planus (Greek *leichen*, "tree moss"; Latin *planus*, "flat") is a unique, common inflammatory disorder that affects the skin, mucous membranes, nails, and hair^[1].

Lichen planus is a noncontagious, clinically and histologically very typical, sub acute or chronically progressive, inflammatory, papulosequamous skin disease, which causes much itching, and also readily develops on the mucous membranes^[2].

Typical lesions are violaceous or lilac-colored, intensely itchy, flat-topped papules that arise usually on the extremities, particularly on the volar aspects of the wrists and legs. On must look closely to see a white streaky pattern on the surface of these papules (Wicham's striae)^[3]. The mucosae are often affected and lesions occur in the mouth in some 30% of patients. A white lacework pattern on the buccal mucosa is the most frequently observed type of lesion, but the tongue and elsewhere in the mouth may also be involved, with white lacework, whitish macule or punctuate lesions. The male genitalia are also sometimes affected. The nails develop longitudinal ridges in 5-10% of patients. Less commonly, a destructive process develops in which the nail plate is lost and the nail forming tissue (nail matrix) is damaged. The scalp is sometimes affected and then localized patches of hair loss and scalp scarring occur. As lesions

heal, they flatten and often leave a pigmented patch, which persists for some weeks^[4].

The etiology of the lichen planus is unknown, but cutaneous eruptions clinically resembling LP have been observed after administration of numerous drugs, including diuretics, gold, antimalarials, penicillamine, and phenothiazines, LP associated with abnormal liver function has been correlated with viral hepatitis, particularly hepatitis C infection^[5].

Rebora et al have reported that 81.81% patients with erosive lichen planus and 13.5 per cent of their patients with nonerosive lichen planus had evidence of chronic active hepatitis with negative hepatitis B surface antigen studies. Korkij et al found a similar incidence of documented liver disease, significantly higher than their control population. Two other studies have not confirmed these findings^[6].

LP may involve a cell-mediated immune response that occurs in the basal cells. There is basal cell degeneration with reduced cell mitosis^[7]. It is conceivable that disturbances in the polymorphonuclear neutrophil leukocytes system may trigger and sustain the inflammatory process in LP, which also involves macrophages/Langerhans cells and different T-cell subsets in the pathogenesis of this disease^[8].

Considerable evidence now exists that the underlying processes involved in the pathogenesis of LP are immunologically mediated; a virus has long been thought to be the 'triggering agent'. The

antigen carried on the cell surface could alter the T-cell interpretation of the histocompatibility complex so that it recognizes it as a foreign thereby attacking it ^[9]. The most consistent HLA association with LP is that of A3. Males with disease duration for greater than one year had Bw35 and Cw4 ^[10].

Cutaneous LP usually persists for months, but in some cases, for years ^[11].

Viral hepatitis is a systemic disease primarily involving the liver. Most cases of acute viral hepatitis in children and adults are caused by one of the following agents: hepatitis A virus (HAV), infectious hepatitis or short incubation hepatitis; hepatitis B virus (HBV), serum hepatitis or long incubation hepatitis; hepatitis C virus (HCV); or hepatitis E virus (HEV) ^[12].

About one third of the population has been exposed at some time to the hepatitis B virus (HBV) ^[13].

The diagnosis of chronic hepatitis B can be made, by definition, only after six months from the onset of acute hepatitis B. It is often difficult to suspect the diagnosis of chronic hepatitis B based just on the patient's symptoms. The reason for this difficulty is that those individuals, who develop chronic hepatitis, as indicated previously, are usually the same individuals who had few or no symptoms to signal the onset of their acute hepatitis B ^[14].

A hepatitis B viral infection can progress from an immune tolerant phase (in which the immune system ignores the virus), through an immune clearance phase (in which the immune system attempts to eliminate the virus), to a quiescent phase (in which the virus is inactive). The course depends in large part on the interaction or balance between the immune system and the virus ^[15].

This suggests an immune complex mechanism for the pathogenesis of hepatitis B associated LP; it does seem that LP patients are more prone to develop hepatitis.

The diagnosis of hepatitis B infection is made primarily by detecting the hepatitis B

surface antigen (HBsAg) in the blood. Following an exposure to hepatitis B virus, HBsAg becomes detectable in the blood within four weeks. Chronic hepatitis B viral infection is defined as the persistence of HBsAg for more than six months. The hepatitis B core antigen can only be found in the liver and cannot be detected in the blood. The antibody to hepatitis B core, known as the hepatitis B core antibody (anti-HBc), however, is detectable in the blood ^[16].

Patients & Methods:

Fifty patients with LP attending Department of Dermatology at Tikrit Teaching Hospital were enrolled in this study in the period December 2005 through to December 2006. There were 30 males and 20 were females.

All patients were examined clinically, diagnoses with LP were confirm by histological findings then interviewed and detailed LP questionnaires were completed for each of them.

From each patients were taken two blood sample each contain 5cc, all serum isolated and kept freezing until examination.

First sample were tested for HBV serological markers, HBsAg is the antigen most routinely measured in blood was detected by Radioimmunoassay technique and anti-HBc, which is appear toward the end of the incubation period and persist for several months to years after that was tested by Enzyme linked immunosorbent assay technique . The positive sera were tested again for confirmation.

The second sample were tested for alanine aminotransferase (ALT, SGPT) and aspartate transaminase (AST, SGOT) are used to asses injury to liver cells, the liver's ability to synthesize proteins, and the excretory function of the liver.

Results:

Of fifty patients thirty were male (60%) and twenty were female (40%), their aged ranged (42.72) with SD (± 12.72) (table 1). The new cases were 2% for a period of one year from December 2005 to the December 2006 (table 2).

Table 1: Age and sex of patients with lichen planus.

Age	11-20	21-30	31-40	41-50	51-60	61-70	Total	
							No	%
Male	1	5	8	7	7	2	30	60
Female	1	2	3	8	5	1	20	40
Total	2	7	11	15	12	3	50	100

Table 2: The incidence of new cases of lichen planus.

Patients	No.	%
Chronic cases	49	98
New cases	1	2
Total	50	100

Familial cases were 10% and nonfamilial cases were 90% (table 3).

The sites of the lesions mostly were mucocutaneous 66% and only cutaneous 34 % (table 4).

Only eight patients (16%) had modification of laboratory test, were two patients (4%) positive for HBsAg and one patient (2%) with anti-HBc positive. Five patients (10%) with ALT and AST moderate higher (table 5).

Table 3: The incidence of familial and non-familial cases of lichen planus.

Patients	Numbers	%
Familial	5	10
Non-familial	45	90
Total	50	100

Table 4: The site of the lesions of lichen planus.

Site	Male	Female	Total	
			No	%
Cutaneous	12	5	17	34
Muco-cutaneous	18	15	33	66
Total	30	20	50	100

Table 5: The prevalence of HBsAg, anti-HBc, ALT and AST among lichen planus patients.

Test	Numbers	%
HBsAg(+ve)	2	4
Anti-HBc(+ve)	1	2
ALT,AST(highly moderate)	5	10
Total	8	16

Discussion:

With time, it is becoming apparent that lichen planus is being observed more and more in conjunction with diseases of altered or disturbed immunity; these conditions include ulcerative colitis, alopecia areata, vitiligo, dermatomyositis, morphea, lichen sclerosis, and myasthenia gravis^[17].

The etiology of the lichen planus is unknown. Viral infection has been suspected, because the disease develops symmetrically as in infectious exanthema.

It is probably immunologically mediated perhaps triggered by a virus. An association is noted between lichen planus and hepatitis C virus infection, chronic active hepatitis, and primary biliary cirrhosis. It is thought to be due to an abnormal immune reaction provoked by a viral infection (such as hepatitis) or a drug. Inflammatory cells seem to mistake the skin cells as foreign and attack them^[18].

Langerhans cells process antigens, which are then presented to T-lymphocytes. This stimulated lymphocytic infiltrate is epidermotropic and attacks keratinocytes. During this lymphocytotoxic process, the keratinocytes release cytokines that attract more lymphocytes^[19].

Rebora and Rongioletti in Italy consider that lichen planus can be regarded as a major risk factor for the development of liver cirrhosis. In Italy possibly because of a higher prevalence of HBV infection^[20].

In present study, fifty patients with confirming diagnosis lichen planus were performed. From test liver function we had 16% with dysfunction of tests liver and only 6% from 16% have had HBsAg and anti-HBc positive. We conclude that, the percentage 16% of abnormal liver test function in patients with lichen planus and 6% of them have had hepatitis B infection is low incidence. The explanation for this is a coincidental or due to the antibodies which result from hepatitis can deposit in small blood vessels, causing inflammation of the vessels in tissues throughout the body for example the skin and in some cases give arise the disease like lichen planus.

Therefore, it seems prudent to screen all patients with lichen planus by liver function test. Liver disease may consider a risk factor for lichen planus although not a specific marker for it. The link between liver disease and lichen planus continues to be investigated and debated.

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