No evidence of HCV infection or liver disease in British patients with oral lichen planus


Abstract. An association between chronic hepatic disease and/or hepatitis C (HCV) infection and lichen planus (LP) has been described in patients from Italy, Japan and Spain. There are no data on the frequency of the association with HCV in British patients. In the present investigation, the HCV seropositivity and liver function status of 55 British patients with oral LP were assessed and compared with these parameters in 110 healthy control subjects. None of the patients with LP or control subjects had serum IgG antibodies to HCV or had abnormal liver function. It was concluded that while LP may be associated with HCV infection and liver disease in some southern European and other patients, such a co-occurrence was not detected in British patients.

Key words: hepatitis C virus; lichen planus; oral.

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An association between lichen planus (LP) and chronic active hepatitis (CAH) or hepatic cirrhosis has been suggested mainly in studies of Italian patients and others of southern European or Japanese origin. There are conflicting data, principally from studies of patients from northern Europe and USA, concerning this possible association between LP and chronic hepatic disease. LP may accompany the development of hepatitis C virus (HCV)-associated hepatic disease, there can be coincident resolution of cutaneous and oral LP with interferon alpha therapy for chronic HCV-associated hepatic disease, and many patients with LP in Italy, Japan and Spain may be HCV-infected. The frequency of hepatic disease in UK patients with LP is low, but there are no data concerning the frequency of HCV infection in such patients, hence we have evaluated the hepatic enzymes and HCV status of a group of British patients with LP and compared it to control subjects.

Material and methods

Patient group

The study group comprised 55 British caucasian patients (44 women, 10 men; median age 46 years, range 32-75 years) with clinical and histological features of oral LP, who were otherwise well.

Control group

The volunteer control group comprised 110 (86 women, 24 men; median age 39, range 25-65 years), apparently healthy dental health care workers. None had mucocutaneous LP.

Serological estimation of hepatic function

Serological levels of total bilirubin, total protein, albumin, hepatic alkaline phosphatase, alanine transaminase and gamma glutamyl transpeptidase were estimated in all patients using conventional methods.

HCV antibody assay

Serum IgG antibodies to HCV were assayed using two different third-generation enzyme-linked immunosorbent assays (ELISA) (Ortho Diagnostic Systems, Emmeryville, CA, USA; and Sanofi Diagnostic Pasteur, Marnes la Coquette, France). Confirmatory assays were available but not required in view of the ELISA findings.

Results

None of the patients with LP or control subjects had serological abnormalities of hepatic disease.

None of the 55 patients with oral LP had any serological evidence of IgG antibodies to HCV. Likewise none of the control group were HCV seropositive.
Discussion

Evidence has been presented in previous studies that some patients with LP have concomitant clinical and/or serological evidence of chronic liver disease, particularly CAH or hepatic cirrhosis, and a causal link has been suggested. These associations have been mainly in Italian, other southern European or Japanese patients, but the results presented have shown neither a strong nor consistent association.

The present study was the first to examine the HCV status of a substantial group of British patients with oral LP, but no evidence of HCV infection was shown. None of these patients with LP had serological evidence of chronic hepatic disease and these data thus agree with those from previous studies on the prevalence of chronic hepatic disease in British patients with LP.

Although a positive association has been described for LP and HCV infection in some populations, an explanation for the mechanism of this association has not been found. It is known that the genotype of HCV can influence disease progression or response to treatment, but to date, no association has been found between the development of HCV-related LP and a specific HCV genotype. Likewise, while hepatitis G virus (HGV) may occasionally co-infect with HCV, limited data suggest that concurrent HGV infection does not contribute to the development of HCV-related LP (Lodi, Ibo & Porter, personal observations).

The apparent lack of an association between HCV infection and LP in British patients parallels the lack of an association between LP and chronic hepatic disease in that population.

The prevalence of HCV infection in the UK is low (0.088-0.55%) compared with countries such as Italy (0.7-1.3%). Thus the geographic differences in the prevalence of LP associated with HCV infection might simply reflect overall differences in HCV epidemiology against a background of similar prevalence of LP from country to country.

It was concluded from the present study that otherwise well patients with LP in Britain do not have evidence of HCV infection or of liver disease, a finding which must cast some doubt on the proposition that LP often has causal links with HCV infection and/or liver disease.

References


Address:
Professor S. R. Porter
Department of Oral Medicine
Eastman Dental Institute for Oral Health Care Sciences
University of London
256 Gray's Inn Road
London WC1X 8LD
UK
Internet: www.eastman.ucl.ac.uk