

Introduction

Hepatitis C Virus infection frequently causes chronic infection leading to liver damage, cirrhosis and hepatocellular carcinoma, Organ-specific and non-specific autoantibodies were first described in autoimmune disorders, but many of them may also be found during viral infections, Hepatitis C Virus (HCV) seems to be highly auto immunogenic because numerous autoantibodies have been detected in HCV-infected patients. (*Chretien et al., 2009*).

Hepatitis C virus (HCV) infection is the most common cause of chronic liver disease in the world. Several immunologic abnormalities, such as production of autoantibodies, like cryglobulin, are associated with HCV infection. Hepatitis C virus infection plays an important role in the pathogenesis of the immunologic derangement. (*Boyer and Marcellin, 2000*).

Autoantibodies include antinuclear antibody (ANA), anti-smooth muscle antibody (ASMA), anti-liver kidney microsomal antibody, antithyroglobulin antibody, antithyroid microsomes, and anticardiolipin antibodies. (*Clifford et al., 1995*)

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Antinuclear antibody is one of the most frequently detected autoantibodies. The prevalence of ANA in HCV infected individuals ranges from 21% to 34%. Although ANA is the diagnostic hallmark of systemic lupus erythematosus (SLE) and type 1 autoimmune hepatitis, its role in chronic HCV infection is unclear. (*Yen-Chun Peng et al., 2001*)

There are four major immunofluorescence patterns of ANA : nuclear, rim, Speckled and homogenous. Certain immunofluorescence patterns of ANA may indicate certain disease. For example, a rim pattern of ANA is often associated with SLE; speckled pattern SLE and scleroderma; nuclear pattern, scleroderma and sjögren syndrome; and homogenous, SLE and rheumatoid arthritis. (*Whittingham and McNeilage, 1988*)

The ANA pattern associated with HCV patients was speckled or granular but never homogenous, as observed in the case of autoimmune hepatitis. Similarly, the ASMA found here were not reactive with F-actin microfilaments. The precise antigen specificity of ANA detected in a context of chronic HCV remains unknown. (*Vergani et al., 2004*)

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The presence of serum ANA is associated with various factors including advancing age, genetic predisposition environmental agents, oestrogen-androgen balance, chronic infection and neoplasm. (*Hsieh et al., 2008*)

In some studies ANA positivity had no observed effect on HCV clinical outcome. ANA positivity was associated with being in the group of patient exhibiting quicker progression of HCV fibrosis although this did not reach statistical significance . Associations of ANA positivity with non-response to therapy were not observed. (*Yee et al., 2004*)

There were no differences observed with respect to the distribution of fat, eosinophils, and bile duct damage between ANA positive liver biopsies and ANA negative biopsies. Plasma cells were more frequent in those who are ANA positives. There was an association of ANA positivity with an almost twofold higher chance of having quicker fibrosis. (*Yee et al., 2004*)