IgA Nephropathy Associated with Hepatitis C Virus Infection

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Dear Sir,

Hepatitis C virus (HCV) is a major cause of chronic liver disease. Moreover, HCV infection has been related with immunologically mediated renal disease [1-5]. In addition, in chronic liver disease of any etiology, mesangial glomerular deposits of IgA can appear resulting in lesions similar to those observed in idiopathic IgA nephropathy (Berger's disease). In fact, IgA nephropathy has been asso-

Table 1. Clinical and serological data of 2 patients with IgA nephropathy and HCV infection

ciated with chronic hepatitis B virus (HBV) infection [6], at least in some parts of the world. We describe 2 patients with cryoglobulinemic IgA nephropathy and HCV infection. Table 1 shows the main clinical and serological data. The major clinical findings at presentation, in both patients, were purpura, arthralgias, weakness and mild hypertension. Analytical studies disclosed microhematuria, nonnephrotic proteinuria, renal insufficiency and mildly raised serum aminotransferase values. A polyclonal mixed (IgM-IgG) cryoglobulinemia was also detected. The apparent onset of renal involvement was coincident with that of liver disease. On liver biopsy, patient 1 had chronic active hepatitis and patient 2 micronodular hepatic cirrhosis. In patient 1 renal biopsy revealed an increase in mesangial matrix and mesangial cells and hyalinization and thickening of the arteriolar walls. In patient 2 there were glomerular matrix expansion and focal and segmental lesions of glomerular sclerosis, as well as patchy areas of interstitial fibrosis and tubular atrophy. In both patients, direct immunofluorescence showed intense diffuse granular IgA deposits and less intense C3 in the mesangium. Besides low protein diet and normalization of blood pressure, patient 1 was given interferon- α for 6 months (3 million units thrice weekly). The treatment was associated with an improvement in her general condition and decreased serum ALT up to 26 U/l. HCV-RNA and cryoglobulins were no longer detectable in serum although hypocomplementemia persisted. Renal function and proteinuria remained unchanged. There is a possibil-

ity that HCV infection may in some way be related to IgA nephropathy. Several other viral infections have also been postulated to be associated with glomerular injury involving mechanisms similar to that described for hepatitis B-associated glomerular disease.

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