Membranoproliferative Glomerulonephritis and Hepatitis C Virus Infection

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Fig. 1. Glomerulus showing increased number of mesangial cells and matrix. HE. × 400.
remained raised at 100 U/l and a liver biopsy showed active chronic hepatitis. A severe
mononuclear cell infiltrate was observed in the portal tracts with rupture of the limiting plate
(piecemeal necrosis). Ground glass he-patocytes were absent and liver architecture was
conserved. Three months later, proteinuria and microhematuria persisted unchanged and a
renal biopsy was performed. It contained five glomeruli, all of them showing diffuse
mesangial cell proliferation and an increased mesangial matrix. Accentuation of the
glomerular lobular pattern as well as peripheral mesangial cell interposition was also
observed (fig. 1). Presence of

Dear Sir,

Membranoproliferative glomerulonephritis (MPGN) with subendothelial deposits (type I) is a
well-defined clinicopathological entity [1] which although it may be associated with several
conditions [2], is usually idio-pathic. Many cases of GN related to chronic hepatitis B virus
(HBV) infection have been classified as MPGN and the etiologic role of HBV in MPGN has
been considered. Rollino et al. [3] have recently described the association of hepatitis C virus
(HCV) infection and membranous nephropathy, stressing the importance of systematic
research of HCV antibodies in order to identify one more causal factor of this nephropathy.
We present a patient with HCV infection and type I MPGN.

A 28-year-old previously non transfused woman, consulted her physician in September 1990
because of asthenia, anorexia and weight loss during the preceding month. She did not have
arthralgias, Raynaud’s phenomenon or cutaneous lesions. On physical examination blood
pressure was 130/80 mm Hg, the liver was not palpable and she did not have edema.
Analytical studies disclosed serum creatinine 0.7 mg/dl, proteinuria 19 mg/kg/day and urine
red blood cells 375,000/min with hyaline and hyaline-granular casts. Alanine
aminotransferase (ALT) was 649 U/l, total serum bilirubin 0.9 mg/dl, serum albumin 32 g/l
and total protein 62 g/l. Serum IgG and IgA levels were in the normal range (1,070 and 113
mg/dl, respectively) and IgM was 348 mg/dl (normal 70-280 mg/dl). Serum complement
levels were reduced: C3 50 mg/dl (normal 55-120 mg/dl), C4 2.7 mg/dl (normal $10^{15}$ mg/dl) and CH50 25 UH50 (normal 150-250 UH50). Rheumatoid factor was 86 U/ml (normal < 40 U/ml) and a polyclonal mixed (IgM-IgG) cryoglobuline-mia was detected. Antinuclear antibodies, virus B markers (HBsAg, HBcAb and HBsAb) and IgM antihepatitis A virus yielded negative results. The anti-HCV test was positive. The presence of HCV antibodies was analyzed using a second generation ELISA tests (c200, c22-3) and confirmed with second generation RIBA tests (cl00-3, 5-1-1, c33c, c22-3) developed by Ortho Diagnostic Systems (Raritan, NJ) and Chiron Corporation (Emeryville, Calif.), respectively. In February 1991, the ALT level

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Book Review
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Renal Disease in the Aged
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This is a serious book written by the two authors in its entirety. It covers the literature well as well as citing the authors’ vast experience. The advice given is sound. The book is suitable for fellows in nephrology as well as internists and geriatricians. The writing is lucid, the printing is clear and pleasant and the price is reasonable.