

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 10¹⁵ mg/dl) and CH50 25 UH50 (normal 150-250 UH50). Rheumatoid factor was 86 IU/ml (normal < 40 IU/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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focal interstitial mononuclear cell infiltrates and fibrosis was found. Direct immunofluorescence showed intense diffuse granular IgG deposits, and less intense IgA, IgM and C3 deposits, along the peripheral glomerular capillary wall.

The coexistence of type I MPGN and HCV infection in our patient may be coincidental, since the incidence of HCV antibodies in the blood donor population in Spain is found to be 1.5% [4]. On the other hand, the possibility of a preexisting MPGN must be considered.

Microscopic hematuria

and nonnephrotic proteinuria were present at the onset of the disease in around 30% of the adult patients with type I MPGN [5]; these patients were, therefore, usually diagnosed on routine (or chance) urinalysis. However, we cannot exclude the possible pathogenetic link between MPGN and HCV infection. The beneficial effect of α -in-terferon therapy, with normalization of serum transaminase values and decrease of proteinuria, in a patient with chronic HCV and MPGN [6], supports a causal relationship between these two conditions. Johnson and

Couser [7] proposed that the characteristic pattern of MPGN-related HBV might be caused by a mesangial and subendothelial passive trapping of HBV antigen-containing immune complexes. A similar phenomenon involving HCV antigen could also explain the association of HCV infection and type I MPGN. Further studies are necessary in order to identify HCV in kidneys of the patients with such nephropathological manifestations, as recently shown.

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Book Review

Jerome G. Porush Pierre F. Faubert

Renal Disease in the Aged

Little Brown, Boston 1991. IX+ 400 pp.; E46.00

ISBN 0-316-71401-1

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.

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52. Agarwal N, Handa R, Acharya S et al. Membranoproliferative glomerulonephritis (MPGN) is a type of immune mediated glomerular disease. Its pathology is thickening of the basement membrane and proliferative changes. Other names for MPGN include mesangiocapillary glomerulonephritis and lobar glomerulonephritis.

Epidemiology of Membranoproliferative Glomerulonephritis (MPGN). MPGN is a rare glomerular disease, and seems to be decreasing in prevalence. Systemic infections such as hepatitis B, hepatitis C, endocarditis, chronic abscesses, Human Immunodeficiency Virus and schistosomiasis. Malignancies such as chronic lymphocytic leukemia, and α 1-antitrypsin deficiency.