Hepatitis C Virus Infection and Membranous Glomerulonephritis

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Dear Sir,

Chronic hepatitis B virus (HBV) infection is known to be associated with membranous glomerulonephritis (MGN), where it represents one of the etiologic factors identified up to now [1]. The frequency of association varies according to different authors and is particularly high in childhood [2]. HBs, HBe and HBc antigens have been identified in subepithelial deposits [3–5]. Whether other forms of hepatitis can also be associated with this nephropathy is still unknown. As new tests for the detection of anti-hepatitis C virus antibodies (HCV-Abs) have become available recently, we looked for a possible association between HCV and MGN. We tested sera of 27 adult patients (16 males, 11 females; mean age 49.8 ± 12.2 years) with biopsy-proven MGN. None of them exhibited systemic lupus erythematosus, diabetes mellitus, syphilis, malignancy or exposure to heavy metals or drugs known to induce MGN. HBV antigen and antibody were negative in all the patients.

The presence of HCV-Abs was evaluated by the enzyme-linked immunosorbent assay 'Abbott HCV EIA', which employs a recombinant antigen of HCV. The neutralizing confirmatory Abbott test was used to bear out the positive results.

Only 1 of 27 patients showed the presence of HCV-Abs in several sera; this result was corroborated by the confirmatory test.

The onset of MGN in this patient occurred in July 1989. At the time of the admittance to our nephrology department (April 1990), laboratory investigations showed slight elevation of serum transaminases; alkaline phosphatase and prothrombin time were within the normal range, proteinuria was 4,400 mg/24 h, and renal function was normal. The patient was given a treatment with 3×1 g methylprednisolone pulses followed by oral prednisone 25 mg daily for 1 month and by chlorambucil 10 mg daily for the next month. The treatment was repeated 3 times and lasted on the whole 6 months [6]. At the end of the therapy, complete remission of the nephropathy (proteinuria 0.2 g/day) was achieved and transaminases were within the normal range.

The relationship between hepatitis and occurrence of the nephrotic syndrome is not simply defined. At the time of the admittance to our department, we probably might have observed a late

phase of the HCV infection. Besides, the remission of the nephropathy was achieved when transaminases returned to a normal range. This chronologic sequence is intriguing and suggests an etiologic and not only incidental relationship between hepatitis and MGN. The improvement of the hepatitis might have induced the remission of the nephropathy, according to a mechanism which is known to play a role in other similar associations.

The future systematic research of HCV-Abs in patients affected with MGN is needed in order to evaluate the possible association between HCV infection and MGN and to identify one more possible etiologic factor of this nephropathy.

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