Nephrotic Syndrome Accompanying Hepatitis-B-Related Liver Cirrhosis and Delta Hepatitis

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Dear Sir,

Various pathological patterns of hepatitis B virus (HBV)-related glomerulonephritis (GN), including membranous glomerulonephritis (MGN), membranoproliferative glomerulonephritis (MPGN) and mesangial proliferative GN, have been described [1]. It has been established that circulating immune complexes trapping and formation of antigen-antibody complexes against major HBV antigens (HbsAg, HbeAg, HbcAg) may induce HBV-associated GN [2]. In most cases, patients with HBV-associated GN have no history of hepatitis despite having abnormal function tests on presentation [3]. Delta virus is superinfection to HBV infection. Due to delta virus, the HBV-related clinical course is more complicated such as in short-term occurrence of liver cirrhosis or fulminant hepatitis. Also, HBV replication has been reported after immunosuppressive treatment of HBV-associated MGN [4, 5]. We describe a patient with hepatitis-related MPGN, liver cirrhosis and delta hepatitis.

A 20-year-old woman was admitted to hospital with malaise, jaundice and edema of the lower extremities of 1 month duration. She had a history of hepatitis infection 5 years ago. Her physical exam revealed ascites, splenomegaly, jaundice and bilateral pretibial edema. Laboratory analysis showed hematocrit 35%, BUN 59 mg/dl, serum creatinine 4.2 mg/dl, total protein 6.3 g/dl, albumin 2.5 g/dl, bilirubin 2.8 mg/dl, direct bilirubin 1.6 mg/dl, SGOT (AST) 505 IU/l, SGPT (ALT) 212 IU/l, alkaline phosphatase (ALP) 907 IU/l on admission. Urinalysis revealed 4+ protein with 15-20 red cells, 4-5 white cells, 2-3 granular casts. Daily proteinuria was 10 g. Antinuclear antibody and rheumatoid factor were negative. HbsAg and antidelata were detected in the serum. Four weeks later, her laboratory findings were: BUN 20 mg/dl, serum creatinine 0.9 mg/dl, creatinine clearance 85 ml/min. Liver biopsy and renal biopsy showed micronodular cirrhosis and MPGN, respectively. Deposits of IgG, IgA and IgM were detected in the glomerulus and the capillary loops at renal biopsy. Immunofluorescence for HbsAg was positive in the glomerulus.

In our patient we diagnosed nephrotic syndrome due to postinfectious MPGN and acute hepatitis and liver cirrhosis. Hepatitis, acute renal failure and nephrotic syndrome regressed spontaneously. HBV- and HCV-related GN have been described in the literature. In our patient, MPGN may be secondary to HBV or HBV + delta superinfection. Or delta hepatitis caused exacerbation of GN, because impaired renal function and proteinuria resolved in a short time with decreasing liver enzyme. Two months later, proteinuria was negative and renal function was normal at outpatient follow-up. In summary, since our patient had a history of hepatitis, her liver biopsy showed cirrhosis, and she had clinical features of hepatitis and HBV-related MPGN at the same time, we suggested that replication of HBV or acute delta superinfection might be responsible of MPGN.

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