IgA Nephropathy Associated with Liver Cirrhosis Presenting Antineutrophil Cytoplasm Antibody of IgA Class

M.	Minoru	Sakatsume ^a
S.	Satoru	Suzuki ^a
H.	Hisaki	Shimada ^a
M.	Mitsuhiro	Ueno ^a
S.	Shinichi	Nishi ^a
T.	Takashi	Miyakawa ^b
M.	Masaaki	Arakawa ^a

^aDepartment of Medicine (II), Niigata University School of Medicine and ^bDepartment of Internal Medicine, Saisei-kai Niigata General Hospital, Japan

Minoru Sakatsume, MD, Department of Medicine (II), Niigata University School of Medicine, 1-757 Asahimachi-dori, Niigata 951 (Japan)

Dear Sir,

Patients with liver cirrhosis are often associated with IgA nephropathy [1]. Recently, some reports revealed that antineutrophil cytoplasm antibody of IgA class (IgA-ANCA) might have a pathogenetic role in Henoch-Schönlein purpura and IgA nephropathy [2]. We treated a patient with liver cirrhosis who had IgA nephropathy presenting with ne-phrotic syndrome, progressive renal dysfunction and IgA-ANCA in serum.

A 63-year-old man with a large alcohol intake for 30 years presented with nephrotic syndrome and macroscopic hematuria. Laboratory investigations showed proteinuria (3.6 g/day), hypoalbuminemia (2.1 g/dl), elevated serum IgA (757 mg/dl) and liver dysfunction (the retention of indocyanine green in blood < 15 min > 29.3%; K value: 0.080). Serum creatinine increased from 0.9 mg/dl on admission to 1.5 mg/dl 5 weeks after. The levels of hemolytic complement activity C3 and C4 were normal, and anti-DNA antibody was negative. Viral markers of hepatitis (HBS antigen, anti-HBs and anti-HCV) were not detected. A liver biopsy confirmed the diagnosis of alcoholic liver cirrhosis.

A renal biopsy demonstrated IgA nephropathy with endocapillary proliferation and scattered duplication of capillaries. The electron-microscopic study showed that accumulated cells in glomeruli were mainly neutrophils and monocytes (fig. 1a). To find out the mechanism of glomerular injury in

this case, the serum was subjected to the detection of ANCA. Indirect immunofluorescence on fixed human neutrophils with the serum diluted at 1/20 proved that the ANCA of IgA class was positive (fig. 1b), whereas other isotypes (IgG or M) were negative. As a treatment, dipyridamole (300 mg/day p.o.) was administered, which was followed by decreased proteinuria (0.2 g/dl) and the improvement of renal function (serum creatinine: 1.2 mg/dl).

The pathogenicity of ANCA is the activation and degranulation of neutrophils [3]. We suppose that clinical manifestations of this case are related with IgA-ANCA. It might stimulate neutrophils and cause their accumulation and subsequent monocyte migration in glomeruli, and

then elicit massive proteinuria and hematuria. The pathogenetic role of IgA-ANCA, however, cannot be determined, because the reported antigens for IgA-ANCA are different from those for IgG-ANCA [2]. Regarding treatment, dipyridamole appeared to be effective in our case. It inhibits the active oxygen generation in neutrophils and directly scavenges a free radical, as we reported earlier [4]. It has already been widely used as an antiplatelet agent for treating various types of glomerulonephropathy. Furthermore it can be chosen to prevent the toxicity of activated neutrophils as in our case.

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0028-2766/94/

0683-0403\$8.00/0