Dear Sir,

Membranoproliferative glomerulonephritis (MPGN) associated with hepatitis C virus (HCV) infection has been recently reported [1] and its prevalence may be very high in primary MPGN [2]. This disease is clinically characterized by nephrotic syndrome, active HCV infection, frequent existence of cryoglobulinemia and hypocomplementemia and its pathogenesis is assumed to be caused by immune complex including HCV [1,2]. However, the glomerular deposition of HCV has not yet been demonstrated because the amount of HCV may be very small. We tried to detect the glomerular HCV deposition in this disease. Freezed kidney specimens obtained by renal biopsy in 6 patients were examined for glomerular HCV detection. Polyclonal rabbit antibody to HCV core antigen, which was provided by Dr. K. Shimotohno (National Cancer Center, Tokyo, Japan), was used as first antibody in the indirect immunofluorescence techniques. FITC-conjugated goat anti-rabbit IgG (Organon Teknika Co., Durham, N.C., USA) was used as second antibody. Negative control consisted of staining with normal rabbit serum or antibody to HCV absorbed with HCV core antigen, followed by FITC-conjugated goat anti-rabbit IgG. Glomerular HCV deposition was observed in 2 of 6 patients with granular manner along the capillary wall and in the mesangium (fig. 1). Doutrelepont et al.
[3] described the detection of HCV RNA by polymerase chain reaction (PCR) in urine and renal tissue from a patient with MPGN associated with mixed cryoglobulinemia and HCV infection. However, PCR is a very sensitive test and the existence of HCV RNA does not mean the glomerular HCV deposition. Misiani et al. [4] described PCR-detected HCV RNA in normal kidney of HCV carrier as well as in renal tissue from patients with cryoglobulinemic glomerulonephritis. We have firstly demonstrated the glomerular deposition of HCV in MPGN patients. This suggests the important role of HCV in the pathogenesis of this disease.

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