Dear Sir,

A high prevalence of markers for hepatitis B virus (HBV) has been reported with great geographical variations in association with 3 types of immune complex glomerulonephritis (GN): membranous, membrano-proliferative and IgA GN [1,2]. Persistent HBV infection has also been shown to occur despite the absence of usual serological markers. HBV deoxyribonucleic acid (HBVDNA) detection in serum allows identification of HBV infection in patients negative for hepatitis B surface antigen (Hbs Ag) [3].

Since 1987, serum HBV-DNA has been tested in 70 idiopathic immune complex GN (membranous GN, n = 28; membranoproliferative GN, n = 10; IgA GN, n = 32) negative for Hbs Ag (radioimmunoassay).

HBV-DNA was detected in the serum by dot-blot hybridization using as probe as full-length cloned HBV-DNA obtained after separation from the cloning vector [3].

None of these 70 patients had detectable serum HBV-DNA. Therefore, our results indicate the unlikely role of HBV infection in negative Hbs-Ag idiopathic immune complex glomerulonephritis.

Nevertheless, the detection limit of this test ranges from 105 to 106 particles/ml, i.e. 103 above the infectivity limit concentration. Indeed, HBV-DNA sequences were found in the serum of 3 patients negative for HBV serological markers and HBV-DNA using polymerase chain reaction (PCR) [4]. A possible etiological role of HBV in these glomerulonephritides cannot be formally eliminated. The eventual HBV-DNA characterization of these sera by PCR is now undertaken.

References
