HBV-DNA and Primary Immune Complex Glomerulonephritis

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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, membranoproliferative and IgA GN [1,2]. Persistent HBV infection has also been shown to occur despite the absence of usual serological markers. HBV deoxyribonucleic acid (HBV-DNA) 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 10^5 to 10^6 particles/ml, i.e. 10^3 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

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