Reactivation of Hepatitis B Virus by Corticosteroids in a Case of Idiopathic Nephrotic Syndrome

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Dear Sir,

Lai et al. [1] recently reported their experience of corticosteroids in Hepatitis B virus surface antigenaemia (HbS) persistent carriers with associated membranous nephropathy [1]. They observed an increase in liver enzymes in 3/7 patients, an enhancement of Hbe antigenemia in 3, an increase in HBV-DNA in 5/6 patients with a decrease after the withdrawal of steroids. We treated an idiopathic nephrotic syndrome (segmental glomerulo-sclerosis) in a 20-year-old Malian patient having a persistent isolated HbS antigenemia unrelated to the nephropathy. Before therapy he was negative for both Hbe antigen and serum HBV-DNA.

He received steroids (1 mg/kg/day) for 4 months during which the proteinuria completely disappeared. He was then reevaluated for this HbS status: we observed the disappearance of anti-Hbe antibodies associated with the appearance of both Hbe antigen and serum HBV-DNA; the delta serology was negative. Concomitantly a 5-fold increase of alanine aminotransferase (ALAT) was noted. Steroids were then rapidly tapered to 10 mg/day and ciclosporin was introduced (4 mg/kg/day). 2 months later, ALAT was normal, HBV-DNA and Hbe antigen were no longer detectable while anti-Hbe antibodies reappeared.

We therefore conclude as Lai et al. [1] that corticosteroids may be harmful in HbS carrier nephrotic patients by inducing viral replication whatever the histological type of glomerular disease is and that liver enzymes, HBV serology, HBV-DNA and delta serology should be carefully monitored in these patients if steroids or immuno-suppressive agents have to be used [2, 3].

References

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