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HBeAg negative chronic hepatitis B with persistently normal serum transaminase and low HBV DNA can cause significant liver disease

Hepatitis B virus (HBV) infects nearly 350 million people worldwide. Their clinical manifestations vary widely from asymptomatic acute viral B hepatitis to hepatocellular carcinoma (HCC). Almost 75-80% of HBV carriers worldwide reside in Asia and Western Pacific region. HBV is a major cause of cirrhosis of liver and HCC worldwide.1

Forty-two consecutive patients (age 21 - 48 years; 34 men) with chronic HBV infection (HBsAg positive for at least 6 months) attending our out-patient department were studied prospectively. Patients were enrolled only if they were HBeAg negative and had persistently normal serum alanine transaminase (ALT), serum HBV DNA level ≤ 10^5 copies/mL using a PCR assay, and negative anti-HCV and anti-HIV antibodies. Patients with clinical evidence of liver cirrhosis were excluded. None of the patients gave history of taking alcohol or any known hepatotoxic drug. Ethical approval for was obtained from our Departmental Review Committee and written informed consent was obtained from each patient. All the patients were detected incidentally at vaccination, health check up and blood donation. None of the patients had any symptom or clinical features of liver disease. All patients underwent percutaneous liver biopsy using a trucut biopsy needle under local anesthesia. Biopsy samples were scored using Knodell score and histologic activity index (HAI) score.2 The histological assessment was done by a pathologist who was unaware of the clinical findings. Necro-inflammation (HAI-NI) was given a score between 0 to 12, while the fibrosis (HAI-F) was scored as 0 to 4.

The ALT value was between 21 to 40 U/L, and quantitative HBV DNA between 1000 to 100,000 copies/mL of blood by PCR. The HAI score (necro-inflammatory score) was between 1-3 in 27 (64.3%) patients, between 4-8 in 11 (26.2%) patients and between 9-12 in 4 (9.5%). Fibrosis score was < 2 and ≥ 2 in 34 (81%) and 8 (19%) patients, respectively.

Our study reveals that CHB patients infected with HBeAg-negative HBV with persistently normal SGPT and low HBV DNA may develop significant necro-inflammation and fibrosis. Several studies have shown that patients with pre-core/core promoter mutant CHB tend to develop more severe liver disease than those infected with wild type CHB. In 2004, an Indian study involving 60 patients demonstrated significant difference in liver fibrosis between wild type and core/core promoter mutant CBH patients, with fibrosis score being higher in core/core promoter mutant CHB. Although these patients also had higher HAI score than those infected with wild type HBV, the difference was not significant.3 A Korean study in 85 young men also yielded similar results.4 A Turkish study that included 354 CHB patients revealed significant difference between necro-inflammatory activity, but not in fibrosis between precore/core promoter mutant and wild type CHB.5

In conclusion, our study shows that patients with HBeAg negative and low viral DNA may have marked necro-inflammation and fibrosis in the liver even when the HBV DNA load is low and ALT is persistently normal. This observation may well explain disease progression in HBeAg negative CHB patients.

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References

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