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Reduced cortisol in the absence of bacterial infection in patients with hepatitis B virus cirrhosis

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ABSTRACT. In liver cirrhosis with bacterial infection, hepatoadrenal syndrome has been described recently as a progressive impairment in the adrenocortical reserve, with deficient production or action of glucocorticoids resulting in adrenal insufficiency. The aim of this study was to explore the characteristics of cortisol in hepatitis B virus (HBV) cirrhosis patients in the absence of bacterial infection. Fasting peripheral venous blood samples were collected from 107 patients with HBV cirrhosis in the absence of bacterial infection and 18 patients with chronic hepatitis B (CHB) infection at 7 a.m. in the morning. The carbohydrate, cortisol-binding globulin, routine chemistry, liver function, and hepatitis B indicators were tested, and free cortisol was calculated. Cortisol (COR) levels were 18.72 ± 6.60 μg/dL in the CHB group and 14.20 ± 7.55 μg/dL in the HBV cirrhosis group (P = 0.002). COR levels were 15.11 ± 5.56, 14.88 ± 6.96, and 12.68 ± 8.36 μg/dL in Child-Pugh class A, B, and C cirrhotic patients, respectively (P = 0.006). Adrenocorticotropic hormone levels were 35.42 ± 24.49, 26.57 ± 15.72, and 19.65 ± 10.72 pg/mL in Child-Pugh class A, B, and C cirrhotic patients, respectively (P = 0.000). Patients with HBV

cirrhosis had significantly lower serum COR levels compared with those of CHB patients, even if they are in the absence of bacterial infection. COR levels negatively correlated with Child-Pugh scores. The hypothalamic-pituitary-adrenal axis might be damaged in patients with HBV cirrhosis.

Key words: Hepatitis B; Cirrhosis; Cortisol; Unbacterial infection; Patient; Liver

