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Elevation of ALT Fails to Predict Significant Histologic Abnormalities in Chronic Hepatitis B Patients

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BACKGROUND: We determined the association between various clinical parameters and significant liver injury in both hepatitis B e antigen (HBeAg)-positive and HBeAg-negative patients. METHODS: From 1994 to 2008, liver biopsy was performed on 319 treatment-naı¨ve CHB patients. Histologic assessment was based on the Knodell histologic activity index for necroinflammation and the Ishak fibrosis staging for fibrosis. Liver biochemistry, HBeAg status and HBV DNA levels were checked. RESULTS: 211 HBeAg-positive and 108 HBeAg-negative patients were recruited, with a median age of 31 and 46 years respectively. 9 out of 40 (22.5%) HBeAg-positive patients with normal ALT had significant histologic abnormalities (necroinflammation grading C7 or fibrosis score C3). There was a significant difference in fibrosis scores among HBeAg-positive patients with an ALT level within the Prati criteria (30 U/L for men, 19 U/L for women) and patients with a normal ALT but exceeding the Prati criteria (p = 0.024). Age, aspartate aminotransferase and platelet count were independent predictors of significant fibrosis in HBeAg-positive patients with an elevated ALT by multivariate analysis (p = 0.007, 0.047 and 0.045 respectively). Serum HBV DNA and platelet count were predictors of significant fibrosis in HBeAg-negative disease (p = 0.020 and 0.015 respectively). An elevated ALT was not predictive of significant fibrosis for both HBeAg-positive (p = 0.345) and –negative (p = 0.544) disease. There was no significant difference in fibrosis staging among ALT 1-2 x upper limit of normal (ULN) and [v2 ULN for both HBeAg-positive (p = 0.098) and –negative (p = 0.838) disease. CONCLUSION: An elevated ALT does not accurately predict significant liver injury. Decisions on commencing antiviral therapy should not be heavily based on a particular ALT threshold. HBV DNA and platelet count are more important determinants of significance fibrosis in HBeAg-negative disease.