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Hepatitis B virus precore and basal core promoter mutations in HBeAG negative inactive and chronic viral hepatitis

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Hepatitis B virus (HBV) infection can lead to a broad spectrum of clinical manifestations ranging from asymptomatic and self limiting infection to chronic liver disease, acute hepatitis, fulminant hepatitis, cirrhosis and hepatocellular carcinoma (HCC). Chronic HBV infection has high prevalence in our country accounting for almost 50 million subjects. Mostly chronic hepatitis B (CHB) infected subjects are asymptomatic inactive carriers (IC). Total 105 (37 IC and 68 CHB) subjects were enrolled in this study. The determination and confirmation of precore and basal core promoter sequence and mutation of HBV was done by using PCR followed by RFLP with restriction enzyme RsaI and Sau3AI and sequencing. In this study we observed the frequency of HBeAg negative and HBeAg positive in inactive carriers were 34(91.9%) and 3(8.1%) and in CHB subjects were 35 (51.5%) and 33 (48.5%) respectively. The prevalence of mutation in HBeAg negative IC was PC 3(8.8%), BCP 8(23.5%) and PC+BCP 12(35.3%) where as in HBeAg negative CHB subjects was PC 11(31.4%), BCP 8(22.8%) and PC+BCP 6(17.1%). Biochemical profile like ALT(IU/L), serum albumin(mg/dl), prothrombin time(INR) and HBV DNA(log10 copies/ml) in PC/BCP mutated HBeAg negative IC and CHB subjects was 31.00±9.62, 4.59±0.59, 1.11±0.15, 1.88±1.10 and 82.98±81.06, 3.56±0.92, 1.75±0.32, 5.66±1.64 respectively. In conclusion PC/BCP mutations were noted higher in both HBeAg negative IC and CHB subjects. Clinical and biochemical profile was similar irrespective of presence or absence of mutant strains.

Biography

Neha Gupta have completed her MSc from Dolphin Institute of Biomedical & Natural Sciences, Garhwal University, Dehradun in 2009 and pursuing PhD from Institute of Medical Sciences, IMS, BHU, Varanasi. She has published more than 6 abstracts in reputed journals.

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