CORRELATION OF HBSAG QUANTITATION BY ELISA IMMUNOASSAY WITH SERUM HEPATITIS B VIRUS DNA QUANTITATIVE PCR IN CHRONIC HEPATITIS B PATIENTS

Sandesh Kolassery, George Sarin Zacharia

Department of Medical Gastroenterology, Government Medical College, Calicut, Kerala, India

Background and objectives: Serum HBV DNA is a useful and reliable marker to diagnose and monitor CHB on treatment. The limitation of HBV DNA is that it is expensive and that the assays lack uniformity and standardization. Hence there is a need for more economical and reliable marker. HBsAg quantitation is one such surrogate serological marker. The objective of the current study is to compare and correlate the serum hepatitis B DNA quantitative PCR with HBsAg quantitation.
Methods: Patients with CHB attending to the outpatient clinic of Gastroenterology department were enrolled in the study. Patients with undetectable HBV DNA levels and those co-infected with HCV or HIV were excluded from the study. All patients were tested for serological markers like HBsAg (rapid), HBeAg, Anti HBe and HBV DNA-PCR. HBsAg quantification was done using conventional ELISA immunoassay. HBV-DNA and qHBsAg levels were expressed in $\log_{10}$IU/ml. Pearson correlation was used to estimate correlation between HBV DNA and HBsAg quantitation. Statistical analysis was done using SPSS and P value of <0.05 was considered significant.
Results: A total of 38 patients were enrolled in the study. 23.62% were females and mean age of patients in the entire study group was 35.72 years. The mean ALT level was 103.80U/L. 26.32% (n = 10) were HBeAg positive. Mean HBV DNA and qHBsAg levels for the entire cohort were 5.81 $\log_{10}$IU/ml and 5.83 $\log_{10}$IU/ml respectively with a correlation coefficient of 0.318 (P = 0.130). For HBeAg positive patients the mean HBV DNA and qHBsAg levels were 7.90 $\log_{10}$IU/ml and 5.91 $\log_{10}$IU/ml respectively with a correlation coefficient of 0.722 (P = 0.043). HBV DNA levels were significantly higher in HBeAg positive patients compared with HBeAg negative patients (7.9 vs 4.01; P = 0.002). qHBsAg levels were also marginally high in HBeAg positive patients (5.91 vs 5.8; P = 0.136). Neither HBV DNA levels nor qHBsAg levels correlated with serum ALT levels.
Conclusion: There is a significant correlation between quantitative HBsAg levels and HBV-DNA levels in HBeAg positive patients with chronic Hepatitis B but not in HBeAg negative patients. HBV-DNA levels are significantly higher in HBeAg positive patients.

Corresponding author: Sandesh Kolassery.
Email: drsandeshk@gmail.com

GENE EXPRESSION CHANGES IN LIVER TISSUE FROM FULMINANT HEPATITIS E

Anshu Naik*, Amit Goel*, Vinita Agrawal*, Aditya Narayan Sarangi*, Nanda Chhavi*, Vineeta Singh†, Shahid Jameel‡, Rakesh Aggarwal*

**Department of Gastroenterology and Pathology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India, †Department of Virology, Sandor Proteomics Pvt Ltd, Hydrabad, Andhra Pradesh, India, and ‡Virology group, International Centre for Genetic Engineering and Biotechnology, New Delhi, India*

PLENARY SESSION

Background and Aim: It is unclear whether liver injury in acute hepatitis E is due to virus-induced cytolysis or the host immune response. We therefore studied host gene expression and enumerated immune cells in liver tissues from fulminant hepatitis FHE (FH-E) patients, in comparison with healthy livers and those from fulminant hepatitis B (FH-B) patients.
Methods: Microarray-based expression profiling was done on post-mortem liver tissue from 5 FH-E and 6 FH-B patients, and normal liver tissue from 6 persons. Differential expression was defined as ≥2.0-fold change with Benjamini-Hochberg false discovery rate below 0.05. $CD4^+$, $CD8^+$ and $CD56^+$ cells were counted using immunohistochemistry.
Results: Compared to normal, the livers from FH-E and FH-B showed differential expression of 3377 (up-regulated 1703, down-regulated 1674) and 2572 (up 1164, down 1408) entities, respectively. This included 2142 (up 896, down 1246) entities that were common between the two sets; most of these belonged to metabolic, hemostatic and complement pathways. An analysis of 1235 (up 807, down 428) entities with differential expression in FH-E but not in FH-B showed activation of several immune response pathways, particularly those involving cytotoxic T cells. $CD8^+$ T cells showed similar increase in both FH-E (median 53.4 per arbitrary unit area [range 31.2-99.9]) and FH-B (49.3 [19.3-51]; P = 0.005) compared to controls (6.9 [3.1-14.9]).
Conclusion: Liver tissue from FH-E patients showed increased expression of genes belonging to cytotoxic T cell effector pathways, accompanied by $CD8^+$ T cell infiltration. This suggests that $CD8^+$ T cells play a role in the pathogenesis of hepatitis E.