LIST OF FIGURES

Figure 2.1: Geographic distribution and load of chronic HBV infection showing high prevalence and load in Asia Pacific regions. 6

Figure 2.2: Structural arrangement of Hepatitis B virus with respect to the viral proteins produced. 8

Figure 2.3: Structural and genetic arrangement of Hepatitis B virus genome 9

Figure 2.4 Representation of the entire Hepatitis B replication process. 15

Figure 2.5: Increased viral load is associated with increased risk for developing HCC 30

Figure 2.6: HBV quantification range via different strategies. (adapted from Mutimer D. EASL. 2001.). 30

Figure 5.1 Graphical representation of distribution of enrolled cases from different states of North east India viz Assam, Arunachal Pradesh, Manipur, Meghalaya, Mizoram, Nagaland and Tripura. Majority of the enrolled cases were from Assam and Arunachal Pradesh. 44

Figure 5.2: Family screening based distribution of acute and chronic HBV cases in family contacts of index patients infected with chronic HBV infection. 45

Figure 5.3: Clinical and histopathological examinations for assessment of liver disease severity. (A-C) Endoscopic examination. (D-F) CT analysis (G) H & E staining for histopathological examination and confirmation of chronic HBV related liver disease. 46

Figure 5.4: Distribution of cases based on the severity of liver disease i.e. chronic HBV and cirrhosis. 47

Figure 5.5: Graphical representation of distribution of cases based on e antigen and anti-HBe status in chronic HBV and cirrhosis cases. 48

Figure 5.6: Pie charts showing percentage distribution of cases based on e antigen and anti-HBe status in chronic HBV and cirrhosis cases highlighting that a high proportion of cases is associated with HBeAg positive status in both the groups. 48
Figure 5.7 A: Bar diagram representing percentage distribution of cases based on e antigen and anti-HBe status in chronic HBV and cirrhosis cases

Figure 5.7 B: Bar diagram representing comparative distribution of male and female cases based on e antigen and anti-HBe status in chronic HBV and cirrhosis cases

Figure 5.8: Difference and distribution of viral load (in log copies/ml) in the CHBV and cirrhosis patients in different sub groups based on HBeAg and anti-HBe status.

Figure 5.9: Comparative difference in viral load (in log copies/ml) in the CHBV and cirrhosis patients in different sub groups based on HBeAg and anti-HBe status

Figure 5.10: Representative post multiplex-PCR agarose gel electrophoresis results for HBV genotyping by virtue of PCR amplified products of different sizes showing presence of HBV genotype.

Figure 5.11: Agarose gel electrophoresis results showing PCR amplification of 742bp after the second round of amplification for the BCP, precore and core region of HBV

Figure 5.12: Representative electrophorogram of the sequences of the BCP, precore and core region of an HBV isolate of a chronic Hepatitis B patient.

Figure 5.13: Comparative analysis of sequence profile from randomly selected cases previously generated by direct sequencing of isolates with standard HBV genebank sequence of genotype A and D.

Figure 5.14: Representative agarose gel electrophoresis results showing second round PCR amplified product for the (A) BCP, precore and the core region and (B) surface region of HBV in the earlier non-genotyped cases.

Figure 5.15 A: Representative electrophorogram showing the direct sequencing results of the surface region of a HBV isolate isolated from a chronic HBV infected case.

Figure 5.15B: Representative electrophorogram showing the direct sequencing results of the surface region of HBV isolates isolated from a HBV related cirrhosis cases.

Figure 5.16: Sequence alignment of representative number of cases compared with the standard genebank sequence of HBV genotype.

Figure 5.17: Sequence alignment and phylogenetic tree analysis of HBV isolates based on the sequence of the surface region of HBV, which previously couldn’t be genotyped by multiplex-PCR method.
Figure 5.18: Prevalence of HBV genotypes in our HBV infected enrolled cases, showing high prevalence of HBV genotype D. Importantly, presence of HBV genotype C which is associated with greater risk of severity of liver disease was also found in high percentage of cases, along with genotype A and mixed genotype A+D, mixed genotype C+D and one case interestingly showing similarity with HBV genotype G.

Figure 5.19. Graph showing difference of genotype distribution in CHBV (group1) and cirrhosis group (Group2). HBV genotype 1, 2, 3, 4, 5, 6 represents genotype A, C, D, A+D, C+D and B+C respectively.

Figure 5.20. Graph representing distribution of HBV genotypes in e+, e- anti-HBe+, e- anti-HBe-ve, and e+ anti-HBe+ groups in the chronic HBV patient cohort.

Figure 5.21. Graph representing distribution of HBV genotypes in e+, e- anti-HBe+, e- anti-HBeve, and e+ anti-HBe+ groups in the cirrhosis patient cohort.

Figure 5.22: HBV genotype distribution as per e antigen positive status in chronic HBV and cirrhosis groups showing difference in prevalence of genotype A, C and D in chronic HBV and cirrhosis cases.