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VIRAL LOAD VARIABILITY IN CHRONIC HEPATITIS B: ROLE OF SOCIO-CLINICAL DETERMINANTS IN AN EASTERN INDIAN HOSPITAL BASED COHORT

Medicine

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ABSTRACT

Background: Viral load is a major determinant of disease progression and transmission in chronic hepatitis B (CHB), but its socio-clinical determinants remain poorly studied in resource-limited settings. This study assessed viral load distribution and associated factors in an eastern Indian cohort. **Methods:** An analytical cross-sectional study (2022–2023) was conducted at a tertiary care hospital in Kolkata among 210 adults with confirmed CHB. Socio-demographic, behavioural, and clinical data were collected using a structured schedule. HBV DNA levels were measured by real-time PCR and classified as low (<2000 IU/mL), moderate (2000–<20,000 IU/mL), or high (≥20,000 IU/mL). Associations were analysed using chi-square tests and multinomial logistic regression. **Results:** Participants were predominantly aged 20–40 years (57.6%) with slight male predominance (53.3%). Low viremia was most common. Viral load showed significant associations with marital status, comorbidity, blood transfusion, family history, illness duration, treatment history, and sexual exposure. High viral load was independently associated with comorbidity (aOR 2.96), family history (aOR 6.25), illness duration of 1–3 years (aOR 26.64), entecavir therapy (aOR 55.30), and sexual exposure (aOR 7.98). Moderate viremia was associated with unmarried status (aOR 3.25), family history (aOR 6.06), and illness duration (aOR 5.51). **Conclusions:** Viral load variability in CHB is shaped by familial, clinical, and behavioural factors. Family history and disease duration were consistent predictors, supporting risk-stratified management and targeted screening in endemic settings.

KEYWORDS

Viral Load, Chronic Hepatitis B, Multinomial Logistic Regression

INTRODUCTION

Chronic hepatitis B virus (HBV) infection remains a major global public health challenge, affecting an estimated 296 million people worldwide and contributing to nearly 820,000 deaths annually, primarily due to cirrhosis and hepatocellular carcinoma.[1-2] Despite the availability of effective antiviral therapies, the burden of disease remains disproportionately high in low- and middle-income countries, including India, where intermediate endemicity and large population size contribute to substantial morbidity.[3]

A key determinant of disease progression and transmission risk in chronic hepatitis B (CHB) is the level of viral replication, commonly assessed by serum HBV DNA (viral load). Elevated viral load has been consistently associated with increased risk of liver fibrosis, cirrhosis, and hepatocellular carcinoma, independent of other clinical factors.[4-5] Consequently, viral load measurement plays a central role in clinical decision-making, including initiation of antiviral therapy and monitoring of treatment response.[6]

However, viral load in CHB is not static and varies considerably across individuals and over time, reflecting the dynamic interplay between host immune response, viral factors, and environmental influences.[7] The natural history of CHB is characterised by distinct phases—immune-tolerant, immune-active, inactive carrier, and reactivation phases—each associated with differing levels of viral replication and disease activity.[8] While virological and immunological determinants of these phases are well described, the contribution of socio-demographic and clinical factors to viral load variability remains less clearly understood, particularly in resource-constrained settings.

Emerging evidence suggests that socio-clinical determinants such as age, sex, comorbidities, behavioural risk factors, and access to healthcare may influence HBV replication and disease progression.[9-10] Additionally, factors such as family history and mode of transmission may reflect early-life infection dynamics, which are known to affect long-term viral persistence and immune control.[11] Understanding these determinants is critical for identifying high-risk subgroups and tailoring public health interventions.

In eastern India, data on viral load distribution and its associated determinants among CHB patients are limited. Hospital-based studies provide an opportunity to characterise real-world patient populations and explore context-specific risk factors. Therefore, this study aimed to assess viral load variability and examine the role of socio-clinical determinants among patients with CHB attending a tertiary care hospital in eastern India. By integrating epidemiological, clinical, and virological data, the study seeks to inform risk stratification and optimise management strategies in similar settings.

METHODOLOGY

This analytical cross-sectional study was conducted between 2022 and 2023 at a tertiary care teaching hospital in Kolkata, catering to a large and diverse population from eastern India. The study was designed to evaluate socio-clinical determinants associated with viral load variability among patients with chronic hepatitis B (CHB) infection.

Participants were consecutively recruited from both outpatient and inpatient departments during routine clinical care. Individuals aged ≥18 years with confirmed CHB infection, defined by persistence of hepatitis B surface antigen (HBsAg) for at least six months, were included. Patients with coexisting chronic liver diseases (including alcoholic liver disease, autoimmune hepatitis, or hepatic malignancy), those receiving hepatotoxic medications, and individuals unwilling to provide informed consent were excluded to minimise confounding.

The sample size was calculated using the standard formula $n = Z^2pq/d^2$, assuming a prevalence of clinically significant high viremia of 41.5%, with a 95% confidence level and 7% absolute precision.[12] The minimum required sample size was estimated to be 191. After accounting for a 10% non-response rate, the final sample size was rounded to 210 participants, all of whom were included in the analysis.

Data were collected using a structured and pretested schedule capturing socio-demographic variables (age, sex, religion, occupation, marital status, and migration history), behavioural risk factors (alcohol use and sexual exposure), and clinical characteristics (presence of comorbidities, history of blood transfusion, needle stick injury, family history of hepatitis B, duration of illness, and treatment history). Information was obtained through a combination of patient interviews and review of medical records to enhance validity and completeness.

Laboratory confirmation of HBsAg was performed using a two-step approach. Initial screening was conducted using an immunochromatographic card test (ICT), and all reactive samples were confirmed by enzyme-linked immunosorbent assay (ELISA). Quantitative estimation of HBV DNA (viral load) was performed using the GeneXpert system, a cartridge-based real-time polymerase chain reaction (RT-PCR) platform that integrates nucleic acid extraction, amplification, and detection within a closed system. Serum samples with a minimum volume of 600 µL were processed in single-use cartridges containing preloaded reagents, and results were generated automatically. HBV viral load was expressed as DNA levels (IU/mL) and categorized based on widely accepted clinical thresholds: low viremia (<2,000 IU/mL), moderate viremia (2,000 to <20,000 IU/mL), and high viremia (≥20,000 IU/mL), consistent with international guideline recommendations.

The primary outcome was viral load variability, operationalized as categorical levels of viremia (low, moderate, and high). Data were entered into Microsoft Excel and analysed using Jamovi software (version 2.7.28).[13] Descriptive statistics were used to summarise baseline characteristics, with categorical variables presented as frequencies and percentages. Associations between socio-clinical variables and viral load categories were assessed using the chi-square test. Variables demonstrating statistical significance in bivariate analysis were subsequently entered into a multinomial logistic regression model to identify independent predictors of moderate and high viral load, using low viremia as the reference category. Model adequacy was evaluated using deviance, Akaike Information Criterion (AIC), and pseudo R^2 statistics (McFadden and Cox–Snell). Adjusted odds ratios (aORs) with 95% confidence intervals (CIs) were reported, and a two-sided p-value <0.05 was considered statistically significant.

Ethical approval for the study was obtained from the Institutional Ethics Committee of the participating hospital. All procedures were conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment, and strict confidentiality of patient information was maintained throughout the study.

The study was designed and reported in accordance with the STROBE Statement (Strengthening the Reporting of Observational Studies in Epidemiology) to ensure methodological rigor, transparency, and reproducibility.

RESULTS

A total of 210 individuals with chronic hepatitis B infection were included. The cohort was predominantly young to middle-aged, with over half aged 20–40 years (57.6%) and a further 26.7% aged 41–59 years; adolescents and elderly individuals comprised relatively small proportions (7.6% and 8.1%, respectively). There was a slight male predominance (53.3%), and most participants were Hindu (59.0%).

Nearly half of the cohort were not engaged in outdoor occupations (45.7%), while the remainder were distributed among labourers (16.7%), students (14.3%), service holders (11.9%), and high-risk occupational groups (11.4%). A large majority were married (71.9%) and reported no migration history (91.0%).

Behavioural risk factors were infrequent: only 3.8% reported alcohol use and 5.7% reported unsafe sexual exposure. Clinical comorbidities were present in 35.7% of participants, although only 7.6% were on chronic medication. A history of blood transfusion was noted in approximately one-quarter (25.7%), while needle stick injury (3.3%) and family history of hepatitis B (9.5%) were relatively uncommon.

Most participants had a disease duration of 1–3 years (82.4%), with fewer reporting longer (13.3%) or very recent illness (4.3%). The majority were not receiving antiviral therapy (77.1%); among treated individuals, tenofovir-based regimens were more common than entecavir-based regimens. Duration of treatment was generally short, with only 6.7% receiving therapy for three years or longer.

A total of patients with chronic hepatitis B were stratified into three viral load categories: high, moderate, and low viremia. Across all socio-demographic and clinical variables, the majority of participants were clustered in the low viremia group.

Age distribution showed that individuals aged 20–40 years constituted the largest proportion of the cohort, with 52.1% in the low viremia category, followed by those aged 41–59 years (60.7%) and ≥60 years (76.5%). Adolescents (≤19 years) also demonstrated a predominance of low viremia (62.5%). Male participants had a slightly higher proportion of high viremia (22.3%) compared to females (15.3%), though low viremia remained the most common category in both sexes (52.7% vs 62.3%).

With respect to religion, both Hindu and Muslim participants predominantly exhibited low viremia (60.5% and 52.3%, respectively). Occupationally, individuals not engaged in outdoor work (homemakers/retired) had the highest proportion of low viremia (63.5%), whereas students demonstrated a relatively higher proportion of moderate (36.7%) and high viremia (30%). High-risk occupational groups also showed a majority in the low viremia category (54.2%).

Married individuals were more likely to have low viremia (64.3%), whereas unmarried individuals had a comparatively higher proportion of moderate (40.7%) and high viremia (20.3%). Participants without a migration history, alcohol use, or chronic disease treatment also predominantly fell within the low viremia category.

Among clinical variables, individuals without comorbidities (60%), without blood transfusion history (51.9%), and without needle stick injury (56.7%) showed a predominance of low viremia. Similarly, those without a family history of hepatitis B had a higher proportion of low viremia (59.5%).

Regarding disease-related factors, patients with longer duration of illness (>3 years) showed the highest proportion of low viremia (78.6%). Those not receiving treatment constituted the largest group and had 57.4% in the low viremia category. Among treated individuals, those on tenofovir-based regimens had a higher proportion of low viremia (71%), whereas those on entecavir-based regimens showed a higher proportion of high viremia (53%). Individuals without unsafe sexual exposure predominantly had low viremia (59.1%).

Bivariate analysis was conducted to examine the association between socio-clinical determinants and viral load categories.

Several variables demonstrated statistically significant associations with viral load variability. Marital status was significantly associated with viremia levels (χ^2=14.6, p<0.001), with unmarried individuals showing a higher proportion of moderate and high viremia. Presence of comorbidities was also significantly associated (χ^2=10.9, p=0.004), with higher viremia observed among those with comorbid conditions.

History of blood transfusion showed a significant association (χ^2=8.03, p=0.018), with transfused individuals more likely to have low viremia. Family history of hepatitis B was significantly associated with viral load (χ^2=6.08, p=0.048), with a higher proportion of moderate viremia among those with positive family history.

Disease-related variables also showed significant associations. Duration of illness was significantly associated with viral load (χ^2=9.94, p=0.042), with longer duration linked to lower viremia levels. History of treatment demonstrated a strong association (χ^2=16.0, p=0.003), where individuals on entecavir-based regimens had higher proportions of high viremia, while tenofovir-based regimens were associated with lower viremia.

Finally, unsafe sexual exposure was significantly associated with viral load (χ^2=8.71, p=0.013), with exposed individuals demonstrating a markedly higher proportion of high viremia (50%). [Table 1]

In multinomial logistic regression analysis (N=210), the model demonstrated modest explanatory power (deviance=340; AIC=380; McFadden R^2=0.172; Cox–Snell R^2=0.106), indicating acceptable but limited discrimination between viral load categories.

Relative to low viral load, high viral load was significantly associated with several clinical and exposure-related factors. Individuals with comorbidity had nearly threefold higher odds of high viral load (aOR 2.96, 95% CI 1.19–7.41; p=0.020). A positive family history showed a strong association (aOR 6.25, 95% CI 1.38–28.41; p=0.018), while longer duration of illness (1–3 vs >3 years) markedly increased the odds (aOR 26.64, 95% CI 2.33–304.14; p=0.008). Treatment with entecavir was associated with substantially higher odds (aOR 55.30, 95% CI 5.71–535.87; p<0.001), although the wide confidence interval suggests imprecision. Sexual exposure was also a significant predictor (aOR 7.98, 95% CI 1.70–37.50; p=0.009). Duration <1 year showed borderline significance (aOR 14.60; p=0.055). Marital status, blood transfusion history, and tenofovir therapy were not significantly associated with high viral load.

For moderate versus low viral load, being unmarried was associated with over threefold higher odds (aOR 3.25, 95% CI 1.45–7.27; p=0.004). A positive family history remained a consistent predictor (aOR 6.06, 95% CI 1.73–21.23; p=0.005), and illness duration of 1–3 years was also significant (aOR 5.51, 95% CI 1.11–27.31; p=0.037).

Other variables, including comorbidity, blood transfusion, antiviral treatment (tenofovir or entecavir), and sexual exposure, were not statistically significant in this comparison. Overall, family history and duration of illness emerged as consistent predictors across viral load strata, while associations with treatment variables should be interpreted cautiously given the wide confidence intervals. [Table 2]

DISCUSSION

In this hospital-based cohort from eastern India, viral load distribution among individuals with chronic hepatitis B (CHB) was predominantly skewed toward low viremia, with relatively fewer patients exhibiting moderate or high viral replication. This pattern is broadly consistent with the natural history of CHB, where a substantial proportion of patients remain in inactive carrier or low-replicative phases, particularly in endemic regions of Asia.[14,15] The modest explanatory power of the multinomial model further underscores the multifactorial and dynamic nature of viral replication in CHB, which is influenced by a complex interplay of host, viral, and environmental factors.[16]

A key finding of this study is the consistent and strong association between family history of hepatitis B and higher viral load categories. This observation aligns with existing evidence suggesting that intrafamilial transmission, particularly perinatal or early childhood acquisition, is associated with prolonged immune tolerance and sustained viral replication.[17,18] Individuals infected early in life are more likely to exhibit higher viral loads over time due to delayed immune clearance, which may explain the elevated odds observed in both moderate and high viremia groups.

Comorbidity emerged as another important determinant of high viral load. Patients with coexisting conditions had nearly threefold higher odds of high viremia, which may reflect immune dysregulation or metabolic influences on viral replication. Previous studies have demonstrated that metabolic comorbidities, including diabetes and fatty liver disease, can exacerbate HBV replication and impair immune-mediated viral control.[19,20] This highlights the need for integrated clinical management addressing both hepatic and extrahepatic conditions.

Duration of illness demonstrated a nuanced relationship with viral load. While longer duration (>3 years) was associated with lower viremia in bivariate analysis, multinomial regression indicated that intermediate duration (1–3 years) significantly increased the odds of both moderate and high viral load. This likely reflects transitional phases in the natural history of CHB, particularly the immune-active phase, during which viral replication and liver inflammation are heightened.[21] Over time, many patients may transition to inactive phases with lower viral loads, explaining the inverse association seen in descriptive analyses.

The association between antiviral therapy and viral load requires careful interpretation. Patients receiving entecavir had markedly higher odds of high viral load; however, this likely reflects confounding by indication, where individuals with more severe disease or higher baseline viral loads are preferentially initiated on potent antiviral therapy.[22] The wide confidence intervals further suggest limited precision due to small subgroup sizes. In contrast, tenofovir-based regimens were descriptively associated with lower viremia, consistent with their well-established antiviral efficacy.[23] These findings reinforce the importance of considering treatment selection bias in observational analyses.

Behavioural and social determinants also contributed to viral load variability. Unsafe sexual exposure was independently associated with high viral load, which may indicate recent infection or ongoing transmission dynamics. Similarly, unmarried individuals had higher odds of moderate viremia, potentially reflecting differences in social behaviour, healthcare access, or timing of diagnosis. Although these associations are less frequently highlighted in the literature, they underscore the role of social context in shaping disease patterns.[24]

Interestingly, traditional risk factors such as blood transfusion and needle stick injury were not independently associated with higher viral load in adjusted analysis, despite showing significance in bivariate comparisons. This suggests that while these exposures may influence acquisition risk, they do not necessarily determine subsequent viral replication dynamics once chronic infection is established.

The predominance of low viremia across most demographic groups—including age, sex, and occupation—suggests a relatively stable CHB population with limited progression to high replicative states. However, the higher proportions of moderate and high viremia among younger individuals and students may indicate ongoing transmission or delayed diagnosis in these subgroups, warranting targeted screening and intervention strategies.

CONCLUSION

This study has several limitations. The cross-sectional design precludes causal inference, and temporal relationships between predictors and viral load cannot be established. The relatively small sample size, particularly within subgroup analyses, is reflected in wide confidence intervals for several estimates. Additionally, virological parameters such as HBV genotype, e-antigen status, and adherence to therapy were not assessed, which may have provided further insights into viral dynamics. Despite these limitations, the study offers valuable real-world evidence from an underrepresented region.

In conclusion, viral load variability in CHB is strongly influenced by familial, clinical, and behavioural factors. Family history and duration of illness emerged as consistent predictors across viral load strata, while comorbidity and sexual exposure were specifically associated with high viremia. These findings highlight the need for risk-stratified management approaches that integrate socio-clinical determinants with virological assessment. Strengthening early detection, family-based screening, and comprehensive care models may improve long-term outcomes in CHB, particularly in resource-constrained settings.

Table 1: Distribution of Socio-demographic, Behavioural, Clinical and Virological Profile of the Participants According to the Viral Load Level. N= 210

Variables	Sub-variables	High Viremia	Moderate Viremia	Low Viremia	Test of Significance
Age	≤19 years	2 (12.5)	4 (25)	10 (62.5)	χ^2=8.56, df=6, p=0.200
	20-40	23 (19)	35 (28.9)	63 (52.1)	
	41-59	14 (25)	8 (14.3)	34 (60.7)	
	≥60	1 (5.9)	3 (17.6)	13 (76.5)	
Gender	Male	25 (22.3)	28 (25)	59 (52.7)	χ^2=2.33, df=2, p=0.312
	Female	15 (15.3)	22 (22.4)	61 (62.3)	
Religion	Hindu	21 (16.9)	28 (22.6)	75 (60.5)	χ^2=1.49, df=2, p=0.474
	Muslims	19 (22.1)	22 (25.6)	45 (52.3)	
Occupation	High risk working group: blood donor/ Truck Driver/Sex Worker	6 (25)	5 (20.8)	13 (54.2)	χ^2=10.2, df=8, p=0.248
	Service	5 (20)	5 (20)	15 (60)	
	Student	9 (30)	11 (36.7)	10 (33.3)	
	Not in outdoor occupation : Home-maker/ Retired	13 (13.5)	22 (23)	61 (63.5)	
	Labourer	7 (20)	7 (20)	21 (60)	
Marital Status	Married	28 (18.5)	26 (17.2)	97 (64.3)	χ^2=14.6, df=2, p<0.001*
	Unmarried	12 (20.3)	24 (40.7)	23 (39)	
Migration History	Yes	4 (21)	6 (31.6)	9 (47.4)	χ^2=0.923, df=2, p=0.630
	No	36 (18.9)	44 (23)	111 (58.1)	
Alcohol Addiction	Yes	3 (37.5)	2 (25)	3 (37.5)	χ^2=2.05, df=2, p=0.358
	No	37 (18.3)	48 (23.8)	117 (57.9)	
Comorbidity	Yes	23 (30.7)	13 (17.3)	39 (52)	χ^2=10.9, df=2, p=0.004*
	No	17 (12.6)	37 (27.4)	81 (60)	
Chronic disease on medication	Yes	5 (31.3)	4 (25)	7 (43.8)	χ^2=1.91, df=2, p=0.385
	No	35 (18)	46 (23.7)	113 (58.3)	

History of Blood Transfusion	Yes	9 (17)	6 (11.3)	39 (73.7)	χ²=8.03, df=2, p=0.018*
	No	31 (19.9)	44 (28.2)	81 (51.9)	
History of Needle Stick	Yes	2 (28.6)	0 (0)	5 (71.4)	χ²=2.33, df=2, p=0.312
	No	38 (18.7)	50 (24.6)	115 (56.7)	
Family History of Hepatitis B	Yes	4 (20)	9 (45)	7 (35)	χ²=6.08, df=2, p=0.048*
	No	36 (18.9)	41 (21.6)	113 (59.5)	
Duration of illness	<1 year	4 (44.4)	1 (11.2)	4 (44.4)	χ²=9.94, df=4, p=0.042*
	1-3 year	34 (19.7)	45 (26)	94 (54.3)	
	>3 year	2 (7.1)	4 (14.3)	22 (78.6)	
History of treatment	No treatment	27 (16.7)	42 (25.9)	93 (57.4)	χ²=16.0, df=4, p=0.003*
	Entacavir-based regimen	9 (53)	3 (17.6)	5 (29.4)	
	Tenofovir-based regimen	4 (12.9)	5 (16.1)	22 (71)	
Duration of treatment	0	27 (16.7)	42 (25.9)	93 (57.4)	χ²=9.35, df=4, p=0.053
	<3 year	12 (35.3)	6 (17.6)	16 (47.1)	
	≥3 year	1 (7.1)	2 (14.3)	11 (78.6)	
History of unsafe Sexual exposure	Yes	6 (50)	3 (25)	3 (25)	χ²=8.71, df=4, p=0.013*
	No	34 (17.2)	47 (23.7)	117 (59.1)	

*p<0.05 statistically significant

Table 2: Multinomial Logistic Regression to Predict Viral Load Variability in Accordance with Socio-Clinico-Behavioural Profile of the Participants

	Model Coefficients - viral load						95% Confidence Interval	
viral load	Predictor	Estimate	SE	Z	p	Odds ratio	Lower	Upper
HV - LV	Intercept	-5.6103	1.295	-4.331	<.001	0.00366	2.89e-4	0.0463
	Marital Status: Unmarried – Married	0.1705	0.532	0.320	0.749	1.18584	0.41793	3.3647
	Comorbidity: Yes-No	1.0868	0.468	2.324	0.020	2.96472	1.18571	7.4129
	Blood transfusion: No-Yes	0.4131	0.506	0.817	0.414	1.51157	0.56119	4.0714
	Family history: Yes-No	1.8330	0.772	2.374	0.018	6.25274	1.37639	28.4053
	Duration Illness: <1 years – >3 years	2.6812	1.399	1.916	0.055	14.60267	0.94035	226.7643
	Duration Illness: 1 to 3 years - >3 years	3.2825	1.242	2.642	0.008	26.64223	2.33385	304.1358
	Treatment history: Tenofovir - No	0.2681	0.722	0.372	0.710	1.30754	0.31790	5.3781
	Treatment history: Entacavir - No	4.0127	1.159	3.463	<.001	55.29724	5.70623	535.8676
	Sexual exposure: Yes – No	2.0768	0.790	2.630	0.009	7.97887	1.69773	37.4986
MV - LV	Intercept	-3.5926	0.965	-3.723	<.001	0.02753	0.00415	0.1825
	Marital Status: Unmarried – Married	1.1781	0.411	2.868	0.004	3.24814	1.45191	7.2665
	Comorbidity: Yes-No	-0.0759	0.424	-0.179	0.858	0.92689	0.40391	2.1270
	Blood transfusion: No-Yes	0.8417	0.527	1.598	0.110	2.32025	0.82641	6.5144
	Family history: Yes-No	1.8024	0.639	2.819	0.005	6.06440	1.73202	21.2336
	Duration Illness: <1 years – >3 years	1.1438	1.378	0.830	0.407	3.13878	0.21066	46.7673
	Duration Illness: 1 to 3 years - >3 years	1.7062	0.817	2.088	0.037	5.50775	1.11059	27.3145
	Treatment history: Tenofovir - No	-0.6577	0.631	-1.042	0.298	0.51807	0.15032	1.7855
	Treatment history: Entacavir - No	1.6454	1.063	1.548	0.122	5.18301	0.64582	41.5959
	Sexual exposure: Yes – No	0.3040	0.881	0.345	0.730	1.35523	0.24125	7.6129

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