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PREDICTORS OF LIVER FIBROSIS IN CHRONIC HEPATITIS B PATIENTS ATTENDING A TERTIARY CARE HOSPITAL IN EASTERN INDIA: AN ANALYTICAL CROSS-SECTIONAL STUDY

Medicine

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ABSTRACT

Background: This study investigated socio-demographic, clinical, and virological predictors of hepatic fibrosis among chronic hepatitis B patients attending a tertiary care hospital in Kolkata, Eastern India. **Methods:** This analytical cross-sectional study included 194 patients with CHB attending a tertiary care hospital in Kolkata between 2023 and 2024. HBsAg screening was performed using immunochromatographic test and confirmed by ELISA. Viral load was quantified using the GeneXpert real-time PCR system. Liver fibrosis was assessed using transient elastography (FibroScan) and classified as no fibrosis (<7.25 kPa), significant fibrosis (7.25–12.4 kPa), and cirrhosis (>12.4 kPa). Associations were analyzed using chi-square test, followed by multivariable logistic regression. Correlation and linear regression were used to assess relationships with liver stiffness (kPa). **Results:** Migration history (OR 4.61, p=0.024), entecavir-based therapy (OR 4.53, p=0.026), and high viral load (OR 8.14, p<0.001) were independent predictors of fibrosis. The model explained 20.3% of variability in fibrosis status with high specificity (96.9%) but low sensitivity (37.1%). A significant positive correlation was observed between HBV DNA levels and liver stiffness. Migrant patients and those with high viremia demonstrated higher disease severity. INR showed a weak association with fibrosis, while antiviral-treated patients likely reflected confounding by indication. **Conclusion:** High viral load, migration history, and treatment profile are key determinants of hepatic fibrosis in CHB. Viral replication remains the strongest driver of disease progression. Early identification of high-risk groups and timely antiviral therapy are essential for preventing fibrosis progression. A multimodal approach combining virological and non-invasive markers is recommended for optimal risk stratification.

KEYWORDS

Liver Fibrosis, Viral Load, Chronic Hepatitis B, Logistic Regression

INTRODUCTION

Chronic hepatitis B (CHB) remains a significant global health issue, impacting approximately 296 million individuals worldwide and contributing notably to liver-related illnesses and deaths.[1] The primary pathway through which CHB causes adverse outcomes involves progressive hepatic fibrosis, which can lead to cirrhosis and hepatocellular carcinoma.[2] Early detection of patients at risk for fibrosis progression is essential for timely treatment and reducing disease burden.[3] However, the natural course of CHB varies greatly among individuals, influenced by a complex interaction of host, viral, and environmental factors.[4]

In settings with limited resources, such as many regions in India, access to invasive diagnostic procedures like liver biopsy is often restricted. Consequently, non-invasive tools such as transient elastography have become increasingly important.[5] Despite advances in antiviral treatments and diagnostic methods, there is a lack of region-specific data on factors influencing liver fibrosis among CHB patients, especially in eastern India. Factors such as sociodemographic characteristics, behavioral risks like alcohol consumption and migration, comorbidities, and virological markers including viral load and hepatitis B e antigen (HBeAg) status may all impact fibrosis progression, but their relative importance remains unclear in this population.[6]

Understanding these predictors is vital for risk stratification, optimizing treatment strategies, and guiding public health policies. Identifying modifiable risk factors could enable targeted interventions to slow disease progression. Given the diversity in epidemiology and healthcare disparities across regions, locally derived evidence is crucial to complement global data.

This study was conducted to evaluate the socio-demographic, behavioral, clinical, and virological profiles of CHB patients and to identify independent predictors of liver fibrosis using non-invasive assessment methods. The primary goal was to determine factors associated with significant fibrosis among CHB patients attending a tertiary care hospital in eastern India, while the secondary goal was to assess the relationship between key predictors and liver stiffness measurements.

MATERIALS & METHODS

This cross-sectional analytical study involved 194 patients with chronic hepatitis B attending a tertiary care hospital in Kolkata (Medical College, Kolkata) between 2023 and 2024. Participants were recruited through routine clinical services, with eligibility determined based on predefined inclusion and exclusion criteria. Initial screening for hepatitis B surface antigen (HBsAg) was performed in the department of Microbiology using an immunochromatographic card test (ICT) (BIOTECH Pvt. Ltd.), and reactive samples were confirmed via enzyme-linked immunosorbent assay (ELISA) (MEDSOURCE OZONE BIOMEDICALS Pvt. Ltd.).

Confirmed HBsAg-positive samples underwent quantitative viral load estimation using the GeneXpert system, an automated real-time PCR platform that combines nucleic acid extraction, amplification, and detection within a closed, cartridge-based system. Serum samples of at least 600 µL were processed using disposable cartridges preloaded with assay reagents, and results were obtained through dedicated software.

Liver fibrosis was assessed non-invasively with transient elastography (FibroScan). Based on validated cut-offs for chronic hepatitis B, fibrosis was categorized as no fibrosis (12.4 kPa; METAVIR stage F4).[7]

Participants with confirmed CHB were included, while those with other chronic liver diseases (including alcoholic, autoimmune, or malignant causes) or on hepatotoxic medications were excluded.

The sample size was calculated using the formula $n = Z^2pq/d^2$, assuming a 34% prevalence of high viremia,[8] with 95% confidence and 7% precision, resulting in a minimum of 176 participants. After accounting for a 10% non-response rate, the final sample size was set at 194.

Data were entered into Microsoft Excel and analyzed with Jamovi software (version 2.7.28). Descriptive statistics summarized baseline characteristics. Associations between variables and fibrosis stages were tested using the chi-square test. Variables significant in bivariate analysis were included in a multivariable logistic regression to identify independent predictors. Correlation analysis evaluated the strength of association between variables and liver stiffness, followed by simple linear regression to quantify the effect of predictors on fibrosis severity measured by elastography (kPa). Statistical significance was set at $p < 0.05$.

All procedures adhered to the Declaration of Helsinki. Informed

consent was obtained from all participants, and confidentiality was maintained. The study followed STROBE guidelines to ensure methodological rigor and transparency.

RESULTS

The baseline characteristics of the 194 CHB patients showed that most were aged 20–40 years (58.8%), followed by 41–59 years (25.8%), with those ≤19 and ≥60 years each constituting 7.7%. Disease severity increased with age: 93.3% of those ≤19 years had no fibrosis, while cirrhosis was most prevalent (12%) in the 41–59 year group; patients ≥60 years had higher significant fibrosis (20%) but no cirrhosis.

Males comprised 54.1% and females 45.9%. Significant fibrosis was more common in males (18.1%) than females (6.7%), while females had a higher proportion of no fibrosis (86.5%). Cirrhosis was slightly more frequent among females (6.7%) than males (3.8%).

Hindu participants made up 58.8%, with minorities at 41.2%. Cirrhosis was marginally higher among minorities (6.2%) compared to Hindus (4.4%).

Occupationally, homemakers/retired individuals accounted for 44.3%, laborers 17%, students 14.9%, service workers 12.4%, and high-risk occupations (e.g., blood donors, truck drivers, sex workers) 11.3%. High-risk groups had lower no fibrosis (72.7%) and higher cirrhosis (9.1%), while students and laborers had no cirrhosis, though laborers showed 18.2% significant fibrosis. Homemakers/retired individuals had 7% cirrhosis.

Married individuals represented 71.1%, unmarried 28.9%. Cirrhosis was observed only in married patients (7.2%), whereas unmarried patients had higher significant fibrosis (23.2%).

Migration status revealed that 91.2% were non-migrants, and 8.8% were migrants. Migrants had lower no fibrosis (58.8%) and higher significant fibrosis (29.4%) and cirrhosis (11.8%).

Most patients (95.9%) did not consume alcohol, while 4.1% did. Alcohol consumers showed higher rates of significant fibrosis and cirrhosis (25% each) compared to non-users (12.4% and 4.3%).

Comorbidities were present in 30.9%, with higher cirrhosis prevalence (8.7%) compared to those without comorbidities (3.2%). Chronic medication use was reported by 10.1%, with higher cirrhosis (18.8%) among users. Blood transfusion history was noted in 24.2%, with 85.1% having no fibrosis and 8.5% cirrhosis, versus 4% cirrhosis without transfusion history. Needle stick injury (3.1%) was associated with higher significant fibrosis (33.3%) but no cirrhosis. Family history of liver disease was present in 11.7%, with higher cirrhosis (15.5%) compared to 4.6% in those without family history.

Illness duration was mostly 1–3 years (82.5%), with cirrhosis highest (5.6%) in this group. Untreated patients made up 81.1%, while 9.4% each received tenofovir or entecavir. Patients on entecavir had higher significant fibrosis (33.3%) and cirrhosis (13.3%). Treatment duration was 0 in 81.1%, less than 3 years in 12.6%, and ≥3 years in 8.8%, with more severe disease in treated groups. Unsafe sexual exposure was reported by 7.5%, with slightly higher cirrhosis (8.3%) in this group.

HBeAg positivity was observed in 7.5%, with higher significant fibrosis and cirrhosis (25% each) compared to non-reactive patients (11.8% and 3.4%). Viral load levels showed that 68.6% had low viremia, 30.8% moderate, and 22.6% high. High viremia was associated with only 50% no fibrosis and 27.8% cirrhosis, indicating a strong link between viral load and fibrosis severity.

Bivariate analysis revealed significant associations of cirrhosis with female gender, married status, migration history, alcohol use, and regular medication for comorbidities. Significant fibrosis was associated with male gender, unmarried status, migration, and alcohol use. Treatment and virological factors, including entecavir therapy, active viral replication, and high viremia, showed strong associations with fibrosis. [Table 1]

Variables significant in bivariate analysis were included in a multivariable binary logistic regression model. The model demonstrated good fit, with a deviance of 139, AIC of 161, and Cox–Snell R^2 of 0.203, indicating that about 20.3% of variability in fibrosis status was explained by the predictors.

In the multivariable analysis, migration history was a significant independent predictor, with patients having a migration history showing 4.61 times higher odds of fibrosis (OR = 4.609, 95% CI: 1.223–17.368, p = 0.024). Treatment with entecavir was also significant, with an OR of 4.53 (95% CI: 1.203–17.066, p = 0.026). Viral load was the strongest predictor; high viremia increased odds of fibrosis by over eight times (OR = 8.141, 95% CI: 2.719–24.375, p < 0.001). Moderate viremia was not statistically significant.

The likelihood ratio tests confirmed the significance of migration (χ^2 = 4.75, p = 0.029), treatment history (χ^2 = 6.38, p = 0.041), and viral load (χ^2 = 14.87, p < 0.001). No multicollinearity was detected among predictors, with VIFs between 1.02 and 1.13.

The model's specificity was high, correctly classifying 96.9% of non-fibrosis cases, but sensitivity was low at 37.1%, indicating better performance in ruling out fibrosis than detecting it.

Overall, the analysis identified migration history, entecavir therapy, and high viral load as independent predictors of liver fibrosis, with viral load being the most influential factor. [Table 2]

Spearman's rank correlation showed a moderate positive correlation (ρ = 0.363, p < 0.001) between HBV viral load and liver stiffness, indicating that higher viral DNA levels are associated with increased fibrosis. [Figure 1]

A simple linear regression model quantified this relationship, with liver stiffness as the dependent variable and HBV viral load as the independent variable. The model was significant ($F(1,192) = 188$, p < 0.001), explaining 49.5% of the variance ($R^2 = 0.495$). The regression equation was:

Liver stiffness (kPa) = $5.02 + (3.58 \times 10^{-7} \times$ HBV viral load)

This indicates that each unit increase in viral load results in an increase of approximately 3.58×10^{-7} kPa in liver stiffness. The slope was highly significant ($\beta = 3.58 \times 10^{-7}$, p < 0.001), with a 95% confidence interval from 3.07×10^{-7} to 4.10×10^{-7}. The standardized coefficient (β = 0.703) confirms a strong positive association.

The intercept of 5.02 (SE = 0.193, p < 0.001) represents the estimated baseline liver stiffness when viral load approaches zero. Diagnostic tests showed no autocorrelation (Durbin–Watson = 1.83, p = 0.254) and no multicollinearity (VIF = 1.00).

Similarly, INR showed a weak but significant positive correlation with liver fibrosis (ρ = 0.245, p < 0.001). Linear regression indicated that liver fibrosis explains about 11.1% of INR variability ($R^2 = 0.111$), with the equation:

INR = 1.0737 + (0.0496 × liver fibrosis score [kPa])

The slope was significant (β = 0.0496, p < 0.001), and residual diagnostics confirmed model adequacy, although residuals deviated from normality. [Table 3]

DISCUSSION

This study identified migration history, entecavir therapy, and high HBV viral load as key independent predictors of hepatic fibrosis, with high viremia showing the strongest association. The multivariable model explained a moderate portion of variability and demonstrated high specificity but limited sensitivity, indicating it is more effective at ruling out non-fibrotic cases than detecting early fibrosis. The significant linear relationship between HBV DNA levels and liver stiffness underscores the importance of viral replication in fibrogenesis.

The association between migration and advanced fibrosis aligns with prior research indicating that migrant populations often present with more severe liver disease, likely due to early-life infection in endemic regions, prolonged immune activation, and delayed diagnosis.[9–11] These findings highlight the need for targeted screening in migrant groups.[9,12]

The strong link between viral load and liver stiffness supports existing evidence that persistent HBV replication drives inflammation and fibrosis progression.[13] Longitudinal data suggest that sustained viral suppression can lead to fibrosis regression and improved liver stiffness.[14]

Interestingly, entecavir therapy was associated with higher odds of fibrosis, likely reflecting confounding by indication, as patients with more advanced disease are often started on potent antivirals.[15] Both entecavir and tenofovir are effective in viral suppression and fibrosis regression, with comparable long-term outcomes.[15,16]

Regarding biochemical markers, INR showed a weak association with fibrosis, consistent with its limited role in early disease detection. Its diagnostic utility is more prominent in advanced liver disease, and in this study, it did not reliably reflect early fibrotic changes.[17–20]

The high specificity of the predictive model suggests it can effectively exclude significant fibrosis, but its low sensitivity limits early detection. This aligns with known limitations of transient elastography, which can be influenced by inflammation and fibrosis heterogeneity.[13,21] Combining elastography with virological and biochemical markers may enhance diagnostic accuracy.

CONCLUSION

This study demonstrates that high viral load, migration history, and entecavir therapy are independent predictors of hepatic fibrosis in CHB patients, with viral replication being the most significant factor. The findings emphasize the central role of viral activity in fibrogenesis and highlight the vulnerability of migrant populations, necessitating targeted screening and early intervention. Although the predictive model has high specificity, its limited sensitivity underscores the need for multimodal assessment strategies to improve early fibrosis detection. Overall, prioritizing early antiviral therapy and integrated risk stratification combining virological, biochemical, and elastographic markers can enhance timely management of progressive liver disease.

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Table 1: Distribution of Socio-demographic, Behavioural, Clinical and Virological Profile of the Participants According to the Liver Fibrosis Status. N= 194

Variables	Sub-variables	No Fibrosis	Significant Fibrosis	Cirrhosis	Test of Significance
Age	≤19 years	14 (93.3)	1 (6.7)	0 (0)	χ^2=8.51, df=6, p=0.203
	20-40	94 (82.5)	16 (14)	4 (3.5)	
	41-59	39 (78)	5 (10)	6 (12)	
	≥60	12 (80)	3 (20)	0 (0)	
Gender	Male	82 (78.1)	19 (18.1)	4 (3.8)	χ^2=6.04, df=2, p=0.049*
	Female	77 (86.5)	6 (6.7)	6 (6.7)	
Religion	Hindu	95 (83.3)	14 (12.3)	5 (4.4)	χ^2=0.459, df=2, p=0.795
	Minority	64 (80)	11 (13.8)	5 (6.2)	
Occupation	High risk working group: blood donor/ Truck Driver/Sex Worker	16 (72.7)	4 (18.2)	2 (9.1)	χ^2=8.02, df=8, p=0.432
	Service	20 (83.3)	2 (8.3)	2 (8.3)	
	Student	24 (82.8)	5 (17.8)	0 (0)	
	Not in outdoor occupation : Homemaker/ Retired	72 (83.7)	8 (9.3)	6 (7)	
	Labourer	27 (81.8)	6 (18.2)	0 (0)	
Marital Status	Married	116 (84.1)	12 (8.7)	10 (7.2)	χ^2=10.8, df=2, p=0.004*
	Unmarried	43 (76.8)	13 (23.2)	0 (0)	
Migration History	Yes	10 (58.8)	5 (29.4)	2 (11.8)	χ^2=6.74, df=2, p=0.034*
	No	149 (84.2)	20 (11.3)	8 (4.5)	
Alcohol Addiction	Yes	4 (50)	2 (25)	2 (25)	χ^2=8.37, df=2, p=0.015*
	No	155 (83.3)	23 (12.4)	8 (4.3)	
Comorbidity	Yes	55 (79.7)	8 (11.6)	6 (8.7)	χ^2=2.81, df=2, p=0.245
	No	104 (83.2)	17 (13.6)	4 (3.2)	
Chronic disease on medication	Yes	11 (68.8)	2 (12.5)	3 (18.8)	χ^2=6.63, df=2, p=0.036*
	No	148 (83.1)	23 (12.9)	7 (3.9)	
History of Blood Transfusion	Yes	40 (85.1)	3 (6.4)	4 (8.5)	χ^2=3.47, df=2, p=0.177
	No	119 (81)	22 (15)	6 (4)	
History of Needle Stick	Yes	4 (66.7)	2 (33.3)	0 (0)	χ^2=2.5, df=2, p=0.286
	No	155 (82.4)	23 (12.2)	10 (5.3)	
Family History of Hepatitis B	Yes	15 (79)	2 (15.5)	2 (15.5)	χ^2=1.29, df=2, p=0.524
	No	144 (82.3)	23 (13.1)	8 (4.6)	
Duration of illness	<1 year	7 (87.5)	1 (12.5)	0 (0)	χ^2=0.747, df=4, p=0.945
	1-3 year	131 (81.9)	20 (12.5)	9 (5.6)	
	>3 year	21 (80.8)	4 (15.4)	1 (3.8)	
History of treatment	No treatment	129 (85.4)	16 (10.6)	6 (4)	χ^2=9.85, df=4, p=0.043*
	Entacavir-based regimen	8 (53.3)	5 (33.3)	2 (13.3)	
	Tenofovir-based regimen	22 (78.6)	4 (14.3)	2 (7.1)	
Duration of treatment	0	129 (85.4)	16 (10.6)	6 (4)	χ^2=5.82, df=4, p=0.213
	<3 year	20 (69)	6 (20.7)	3 (10.3)	
	≥3 year	10 (71.4)	3 (21.4)	1 (7.1)	
History of unsafe Sexual exposure	Yes	9 (75)	2 (16.7)	1 (8.3)	χ^2=0.468, df=2, p=0.791
	No	150 (82.4)	23 (12.6)	9 (4.9)	
Viral Replication / Infectivity status (HBe Ag)	Reactive	8 (50)	4 (25)	4 (25)	χ^2=17.5, df=2, p <0.001*
	Non-reactive	151 (84.8)	21 (11.8)	6 (3.4)	
Viral load	Low viremia < 2,000 IU/mL	100 (91.7)	9 (8.3)	6 (0)	χ^2=54.4, df=4, p <0.001*
	Moderate viremia 2,000 – < 20,000 IU/mL	41 (83.7)	8 (16.3)	0 (0)	
	High viremia ≥ 20,000 IU/mL	18 (50)	8 (22.2)	10 (27.8)	

*p<0.05 statistically significant

Table 2: Multivariable Binary Logistic Regression Model

							95% Confidence Interval	
Predictor		Estimate	SE	Z	p	Odds ratio	Lower	Upper
Intercept		-3.2004	0.533	-6.010	<.001	0.0407	0.0143	0. 116
Gender	Male Female (ref)	0.0871	0.507	0.172	0.864	1.0911	0.4039	2. 947
Marital Status	Unmarried Married (ref)	0.3291	0.499	0.660	0.509	1.3897	0.5230	3. 693
Migration history	Yes No (ref)	1.5281	0.677	2.258	0.024	4.6094	1.2233	17.368

Alcohol addiction	Yes No (ref)	0. 8575	0. 928	0. 924	0. 355	2. 3573	0. 3824	14. 533
Medication for Chronic Disease	Yes No (ref)	0. 7288	0. 726	1. 004	0. 315	2. 0726	0. 4999	8. 593
Treatment regimen	Tenofovir No (ref)	0. 9450	0. 626	1. 509	0. 131	2. 5727	0. 7538	8. 780
	Entacavir No (ref)	1. 5108	0. 677	2. 233	0. 026	4. 5302	1. 2025	17. 066
Viral Replication/ Infectivity status (HBe Ag)	Reactive Non-reactive (ref)	0. 9736	0. 678	1. 437	0. 151	2. 6476	0. 7016	9. 991
Viral load	Moderate Viremia Low viremia (ref)	0. 6836	0. 578	1. 184	0. 237	1. 9809	0. 6386	6. 145
	High Viremia Low Viremia (ref)	2. 0969	0. 560	3. 747	<. 001	8. 1406	2. 7188	24. 375

Note. Estimates represent the log odds of "Firosis yes = 1" vs. "Firosis no = 0" ref: Reference sub-variable

Table 3: Linear Regression Analysis Evaluating the Association of HBV Viral Load and INR with Liver Stiffness (kPa).

Model				Overall Model Test			
	R	R^2	Adjusted R^2	F	df1	df2	p
1. Liver stiffness (kPa) vs HBV viral load	0.703	0.495	0.492	188	1	192	<.001
2. INR vs liver stiffness (kPa)	0.333	0.111	0.106	23.9	1	192	<.001

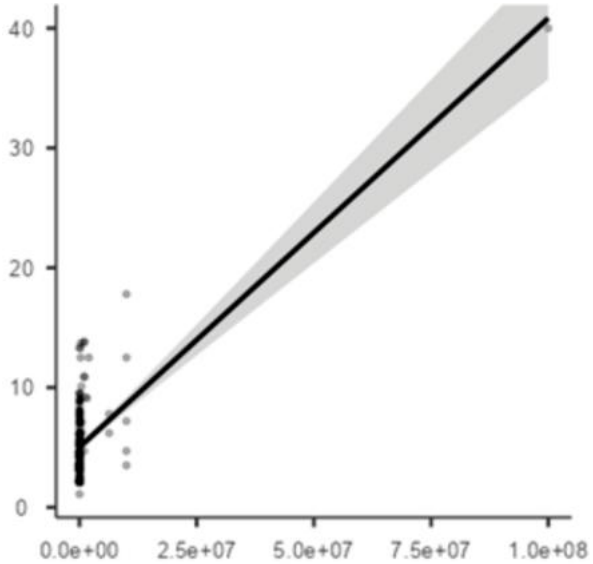

Figure 1: Scatterplot Showing the Correlation Between HBV Viral Load and Liver Stiffness (kPa)

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