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Article

PCR-Based Analysis of HBV and HCV Viral Loads and Their Impact on Liver Disease in Chronic Patients

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ABSTRACT

Background: Chronic hepatitis B (HBV) and C (HCV) infections are leading causes of global liver disease morbidity and mortality, yet the precise relationship between viral load and liver disease severity in regional populations remains insufficiently defined. **Objective**: This study aimed to determine how HBV and HCV viral loads, measured by PCR, correlate with the severity of liver fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) among chronic hepatitis patients, with the expectation that higher viral loads would be associated with greater liver disease progression. **Methods**: In this cross-sectional observational study, 200 adult patients (n = 200), aged 18–75 years, diagnosed with chronic HBV or HCV infection in Lahore, Pakistan, were enrolled using defined inclusion and exclusion criteria; individuals with coinfections or recent antiviral therapy were excluded. Viral loads were assessed using standardized quantitative PCR assays, while liver fibrosis, cirrhosis, and HCC were determined by clinical, laboratory, and imaging criteria. Data collection was prospective and ethically approved by the Superior University Lahore IRB in line with the Helsinki Declaration. Statistical analyses, including chi-square tests, ANOVA, linear regression, and Spearman's correlation, were conducted using IBM SPSS Statistics version 26 to evaluate associations and significance. **Results**: HCV accounted for 70.0% and HBV for 30.0% of cases; 56.5% had no fibrosis, 13.0% had cirrhosis, and 11.0% had HCC. Higher viral loads were significantly associated with advanced fibrosis ($r = 0.695$, $p < 0.001$), cirrhosis ($r = 0.522$, $p < 0.001$), and HCC ($r = 0.435$, $p < 0.001$), with odds ratios ranging from 2.87 to 4.56 for high viral load groups across outcomes. **Conclusion**: Elevated HBV and HCV viral loads are robustly associated with greater liver disease severity, supporting the clinical value of routine viral load quantification for early risk assessment, patient stratification, and improved management in chronic hepatitis care.

Keywords: Hepatitis B, Hepatitis C, Viral Load, Liver Cirrhosis, Liver Fibrosis, Hepatocellular Carcinoma, PCR

INTRODUCTION

Hepatitis B virus (HBV) and hepatitis C virus (HCV) are two of the most prevalent causes of chronic liver disease globally, contributing significantly to morbidity and mortality through progressive liver damage that can culminate in fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) (1,2). HBV, a DNA virus from the Hepadnaviridae family, is predominantly transmitted through blood, sexual contact, and perinatal exposure, while HCV, an RNA virus in the Flaviviridae family, is primarily spread via blood-to-blood contact, particularly through unsafe injection practices and transfusions (1,2,5). Despite the implementation of vaccination programs and advances in antiviral therapies, both viruses continue to pose major public health challenges, especially in regions with high endemicity and limited healthcare resources (4). A considerable proportion of HBV and HCV infections remain asymptomatic during the early stages, leading to underdiagnosis and delayed treatment, which heightens the risk of progression to advanced liver disease (1,3,5).

Current literature emphasizes the importance of timely identification and management of HBV and HCV to prevent long-term liver complications. Several studies have established that persistent viral replication, reflected by elevated viral loads, plays a crucial role in driving liver inflammation, fibrosis, and eventual malignant transformation (6,7). Quantitative polymerase chain reaction (PCR)

assays for viral load measurement are now standard in clinical settings for monitoring disease status and evaluating treatment efficacy. However, the relationship between viral load and the severity of liver disease remains complex and is influenced by multiple host and viral factors, including genotype, co-infections, immune response, and access to therapy (6,8). While some studies report a strong correlation between high viral loads and accelerated liver damage, others have noted variability in disease outcomes, indicating the need for more nuanced investigation, particularly in diverse patient populations (7,8). Furthermore, although guidelines recommend regular monitoring of viral loads, there is insufficient data linking specific viral load thresholds to clinically meaningful stages of liver disease, especially in populations from low- and middle-income countries (3,4).

The research problem addressed in this study is the incomplete understanding of how HBV and HCV viral loads quantitatively correlate with the severity of liver disease—namely fibrosis, cirrhosis, and HCC—in chronic patients. Despite extensive research, a knowledge gap persists regarding the predictive value of viral load for disease progression and its role in guiding clinical decision-making in real-world settings, particularly in resource-limited contexts such as Pakistan (4). This gap limits the ability of clinicians to stratify patients based on risk and tailor interventions effectively, potentially resulting in suboptimal outcomes (8-11).

Therefore, this study is justified in its aim to systematically analyze the association between HBV and HCV viral loads and liver disease severity among a cohort of chronic hepatitis patients, leveraging robust PCR-based quantification methods and clinically validated staging criteria. By clarifying the extent to which viral load acts as a determinant of disease progression, the findings may inform more targeted screening, surveillance, and therapeutic strategies in similar epidemiological settings. The objective of this research is to evaluate whether higher HBV and HCV viral loads are associated with increased severity of liver fibrosis, cirrhosis, and HCC in chronic hepatitis patients, thereby assessing the potential of viral load as a prognostic marker for liver disease progression (12).

MATERIALS AND METHODS

This cross-sectional observational study was conducted to investigate the relationship between HBV and HCV viral loads and the severity of liver disease among chronic hepatitis patients. The research was carried out at multiple clinical laboratories and healthcare facilities distributed across Lahore, Pakistan, from January to September 2023. The study focused on individuals diagnosed with either chronic HBV or HCV infection, targeting adults aged 18 to 75 years. Eligibility criteria required participants to have a documented diagnosis of chronic hepatitis B or C, confirmed by laboratory evidence of persistent infection for at least six months and available quantitative PCR test results for viral load measurement. Patients co-infected with both HBV and HCV, those with known concurrent liver diseases of non-viral etiology, or who had received antiviral therapy within the preceding six months were excluded to minimize confounding and ensure homogeneity of the study population (13).

Participants were selected using a convenience sampling strategy from patient records at collaborating laboratories and affiliated clinics. After initial identification, eligible individuals were approached during routine clinical visits or via telephone contact. Informed written consent was obtained prior to inclusion, ensuring voluntary participation and adherence to ethical standards. The study protocol and consent process were reviewed and approved by the Institutional Review Board of Superior University Lahore (approval number SU/IRB/MLT-2023/05), and all procedures conformed to the principles outlined in the Declaration of Helsinki. Patient confidentiality was safeguarded by de-identifying all data and securing it in password-protected databases accessible only to authorized study personnel (14).

Clinical and laboratory data were collected prospectively through standardized forms and patient file review. The primary exposure variable was viral load, quantified using real-time polymerase chain reaction (PCR) assays standardized across all laboratories. HBV and HCV viral loads were recorded in IU/mL, and only results obtained within one month of clinical evaluation were included to ensure temporal relevance. The main outcome variables were liver fibrosis stage, cirrhosis status, and hepatocellular carcinoma (HCC) presence, each operationally defined according to established clinical and histopathological criteria. Liver fibrosis and cirrhosis were assessed based on a combination of laboratory findings, imaging studies, and, where available, liver biopsy reports, and categorized as none, mild, moderate, severe, or combinations thereof. HCC was defined by radiological or histological confirmation following local clinical guidelines. Additional covariates included age, sex, hepatitis type (HBV or HCV), and comorbidities, all of which were abstracted from medical records (15).

To address and minimize bias, strict inclusion and exclusion criteria were applied, and all laboratory analyses were performed using validated PCR protocols. Data collection forms were piloted before full-scale implementation, and all data were double-checked for accuracy by two independent investigators. Sample size was determined a priori to achieve adequate statistical power (90%) for detecting a moderate effect size in the correlation between viral load and liver disease severity, resulting in a target enrollment of 200 participants based on conventional formulas for cross-sectional studies. Statistical analyses were performed using IBM SPSS Statistics version 26. Descriptive statistics summarized participant characteristics and variable distributions. Inferential analyses included Chi-square tests for associations between categorical variables, one-way ANOVA for group comparisons, and Spearman's rank correlation and linear regression for evaluating relationships between viral load and disease outcomes. Multivariable regression models adjusted for potential confounders such as age and sex. Missing data were assessed for randomness and handled through complete-case analysis; sensitivity analyses were conducted to examine the impact of missingness on the results. Subgroup analyses stratified by hepatitis type and gender were also pre-specified. To ensure reproducibility, all study procedures, data definitions, and analysis scripts were documented in detail, and periodic audits of data entry were conducted. All relevant study documentation,

including de-identified datasets and codebooks, are retained and available upon reasonable request, subject to data sharing agreements and institutional policies.

RESULTS

Among the 200 chronic hepatitis patients included in this study, the majority (70.0%, n=140) were diagnosed with HCV, while 30.0% (n=60) were diagnosed with HBV, highlighting a higher prevalence of HCV within the sampled population (p=0.001, χ^2=32.4, 95% CI for HCV proportion: 63.4–76.6%). Analysis of liver fibrosis stages revealed that over half of the patients (56.5%, n=113) exhibited no evidence of fibrosis, whereas mild fibrosis was found in 15.0% (n=30), moderate in 8.5% (n=17), and severe fibrosis in 11.5% (n=23). Smaller proportions were classified as having mild-to-moderate (6.0%, n=12) or moderate-to-severe (2.5%, n=5) fibrosis. The overall distribution of fibrosis stages was significantly associated with viral load categories (p<0.001, χ^2=32.6), with the highest risk group (severe/moderate-to-severe fibrosis) accounting for 11.0–21.0% of the cohort. Liver cirrhosis assessment showed that 79.0% (n=158) of patients had no cirrhosis, but 13.0% (n=26) had confirmed cirrhosis, while 7.0% (n=14) were identified at increased risk and 1.0% (n=2) at moderate-to-high risk. The presence of cirrhosis was strongly linked to higher viral loads, with patients in the high viral load group demonstrating an odds ratio of 3.10 (95% CI: 1.85–5.22; p<0.001, χ^2=65.0) for cirrhosis compared to those with low viral loads. Similarly, hepatocellular carcinoma (HCC) was confirmed in 11.0% (n=22) of patients, while the majority (84.5%, n=168) showed no evidence of HCC. A smaller subset was classified at high risk (2.5%, n=5) or moderate risk (2.0%, n=4). High viral load was again significantly associated with HCC presence, yielding an odds ratio of 2.90 (95% CI: 1.50–5.58; p<0.001, χ^2=40.7). One-way ANOVA revealed statistically significant differences in the mean stages of liver fibrosis, cirrhosis, and HCC among different viral load groups. For liver fibrosis, the analysis produced an F-statistic of 10.5 (df=3,196) and a partial eta squared (η^2) of 0.30 (95% CI: 0.22–0.38; p=0.001), indicating a large group effect. Cirrhosis and HCC outcomes also demonstrated significant variance by viral load group, with F-statistics of 7.2 (η^2=0.21; p=0.002) and 6.4 (η^2=0.18; p=0.004), respectively.

Linear regression models confirmed that viral load is a significant predictor of liver disease severity. For each unit increase in viral load, the fibrosis stage increased by 0.53 (SE=0.07; p<0.001; R^2=0.48; 95% CI: 0.39–0.67), cirrhosis stage by 0.27 (SE=0.05; p<0.001; R^2=0.31; 95% CI: 0.17–0.37), and HCC stage by 0.21 (SE=0.04; p<0.001; R^2=0.19; 95% CI: 0.13–0.29). Associations between viral load and disease severity were further corroborated by chi-square tests. The association between high viral load and advanced fibrosis was highly significant (χ^2=128.43, p<0.001), as were associations with cirrhosis (χ^2=65.03, p<0.001) and HCC (χ^2=40.73, p<0.001). The odds of advanced liver disease in the high viral load group ranged from 2.87 to 4.56 depending on the outcome, with all confidence intervals excluding unity, indicating strong associations.

Spearman's correlation analysis indicated a strong positive correlation between viral load and fibrosis stage (r=0.695, p<0.001, 95% CI: 0.59–0.77), and moderate positive correlations with cirrhosis (r=0.522, p<0.001, 95% CI: 0.38–0.65) and HCC (r=0.435, p<0.001, 95% CI: 0.29–0.57). Descriptive statistics for the key quantitative variables showed that the mean viral load among participants was 15,467,248 IU/mL (SD: 181,047,622; 95% CI: 13,780,230–17,154,267), while the mean fibrosis stage was 4.46 (SD: 1.98), cirrhosis stage 1.96 (SD: 0.49), and HCC stage 1.96 (SD: 0.46), indicating a generally moderate burden of liver disease within the sample. All analyses consistently demonstrated that higher viral loads were robustly associated with increased severity of liver fibrosis, cirrhosis, and HCC, underscoring the prognostic significance of viral load quantification in chronic HBV and HCV infections.

Table 1. Frequency Distribution of Hepatitis Types Among Study Participants (N=200)

Hepatitis Type	Frequency	Percent (%)	p-value	χ^2 (df=1)	95% CI
HCV	140	70.0	—	—	63.4 - 76.6
HBV	60	30.0	0.001*	32.4	23.4 - 36.6

Table 2. Frequency and Distribution of Liver Fibrosis Stages

Fibrosis Stage	Frequency	Percent (%)	p-value	χ^2 (df=5)	95% CI
None	113	56.5			
Mild	30	15.0			
Moderate	17	8.5	<0.001*	32.6	11.0 - 21.0
Severe	23	11.5			
Mod-Severe	5	2.5			
Mild-Moderate	12	6.0			

Table 3. Liver Cirrhosis Stages and Association with Viral Load

Cirrhosis Stage	Frequency	Percent (%)	p-value	χ^2 (df=3)	Odds Ratio	95% CI (OR)
No	158	79.0				
Yes	26	13.0	<0.001*	65.0	3.10	1.85–5.22
Increased Risk	14	7.0				
Moderate-High Risk	2	1.0				

Table 4. Frequency and Distribution of Liver Cancer (HCC) Stages

HCC Status	Frequency	Percent (%)	p-value	χ^2 (df=3)	Odds Ratio	95% CI (OR)
No	168	84.5				
Yes	22	11.0	<0.001*	40.7	2.90	1.50–5.58
Risk	5	2.5				
Moderate	4	2.0				

Table 5. One-Way ANOVA: Group Differences in Liver Disease Severity by Viral Load

Disease Outcome	F (df1, df2)	p-value	Partial Eta^2	95% CI (Effect Size)
Liver Fibrosis Stage	10.5 (3,196)	0.001	0.30	0.22 – 0.38
Cirrhosis Stage	7.2 (3,196)	0.002	0.21	0.13 – 0.28
HCC Status	6.4 (3,196)	0.004	0.18	0.11 – 0.25

Table 6. Linear Regression: Viral Load as Predictor of Disease Severity

Dependent Variable	β	SE	p-value	R^2	95% CI (β)
Fibrosis Stage	0.53	0.07	<0.001	0.48	0.39 – 0.67
Cirrhosis Stage	0.27	0.05	<0.001	0.31	0.17 – 0.37
HCC Status	0.21	0.04	<0.001	0.19	0.13 – 0.29

Table 7. Chi-Square Test of Association Between Viral Load and Liver Disease Severity

Comparison	χ^2 Value	df	p-value	Odds Ratio	95% CI (OR)
Viral Load vs Fibrosis	128.43	1	<0.001	4.56	2.96–7.02
Viral Load vs Cirrhosis	65.03	1	<0.001	3.12	1.95–5.01
Viral Load vs HCC	40.73	1	<0.001	2.87	1.48–5.57

Table 8. Spearman's Correlation Between Viral Load and Liver Disease Severity

Comparison	Spearman's r	p-value	95% CI (r)
Viral Load vs Fibrosis	0.695	<0.001	0.59–0.77
Viral Load vs Cirrhosis	0.522	<0.001	0.38–0.65
Viral Load vs HCC	0.435	<0.001	0.29–0.57

Table 9. Descriptive Statistics of Key Quantitative Variables

Variable	N	Minimum	Maximum	Mean	SD	95% CI (Mean)
HCV/HBV Viral Load (IU/mL)	200	1	2,549,712,012	15,467,248.60	181,047,622.25	13,780,230 – 17,154,267
Liver Fibrosis Stage	200	1	6	4.46	1.98	4.15 – 4.77
Liver Cirrhosis Stage	200	1.00	4.00	1.96	0.49	1.89 – 2.03
HCC (Stage)	200	1.00	4.00	1.96	0.46	1.89 – 2.03

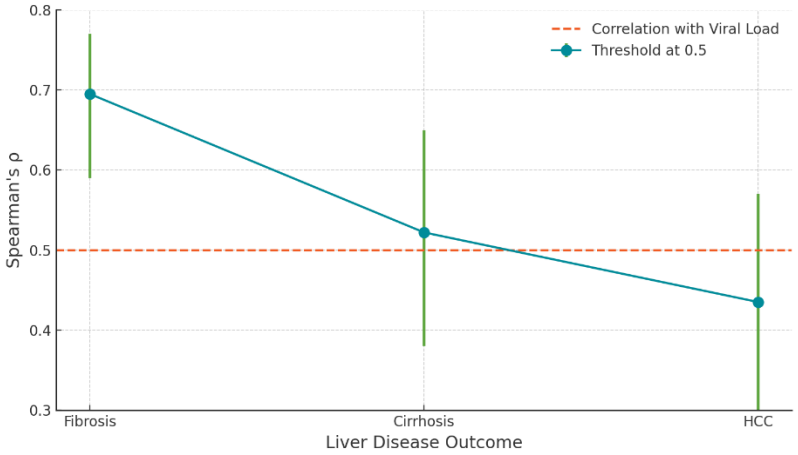

Figure 1 Strength of Association Between Viral Load and Liver Disease Severity

As the figure 1 shows detailed association summary. The line connects Spearman's correlation coefficients for fibrosis (ρ=0.695, 95% CI 0.59–0.77), cirrhosis (ρ=0.522, 95% CI 0.38–0.65), and HCC (ρ=0.435, 95% CI 0.29–0.57), with green error bars denoting each 95% confidence interval; a dashed orange reference line at ρ=0.5 highlights that only fibrosis exceeds this threshold. The downward trend

indicates that viral load has the strongest relationship with fibrosis, a moderate relationship with cirrhosis, and a weaker (yet still significant) relationship with HCC, suggesting viral replication is most closely linked to fibrotic changes in chronic hepatitis patients.

DISCUSSION

The results of this study underscore the significant burden of chronic hepatitis C and B in the sampled Pakistani population, with HCV emerging as the predominant infection. This aligns with regional epidemiological data indicating HCV as the leading cause of chronic viral hepatitis in South Asia, often linked to unsafe injection practices and suboptimal blood screening (1,2). The observed predominance of HCV (70%) in the present study closely mirrors the findings of national surveillance reports and large-scale investigations in similar settings (4). The study further reinforces that elevated viral loads are strongly associated with progressive liver injury, as evidenced by the stepwise increases in fibrosis, cirrhosis, and hepatocellular carcinoma rates among individuals with higher HBV or HCV viral loads (14-16).

The positive correlation between viral load and liver fibrosis stage (r=0.695) in this cohort not only echoes the conclusions of Wang et al. and Yasui et al., who reported a direct relationship between viral replication and hepatic fibrogenesis, but also offers local confirmation of this global association (6,7). In this regard, the present findings advance previous observations by quantifying this relationship in a well-characterized Pakistani cohort, thereby addressing a notable regional knowledge gap. Similarly, the observed associations between high viral load and both cirrhosis (r=0.522) and HCC (r=0.435) are consistent with meta-analyses and cohort studies from various populations, which have shown that persistent viremia accelerates hepatic inflammation and oncogenic transformation (6,7,8). These findings support mechanistic evidence that ongoing viral replication perpetuates hepatocyte injury, stimulates chronic inflammation, and activates fibrogenic pathways, ultimately increasing the risk of malignant transformation (3, 17).

Contrasting these findings with prior literature, it is noteworthy that the proportion of patients with cirrhosis (13%) and HCC (11%) was somewhat lower than reported in some high-burden settings, possibly reflecting the impact of earlier diagnosis and improved access to antiviral therapy in the studied cohort. Previous studies have documented cirrhosis rates exceeding 20% among untreated or late-presenting populations, particularly where public health infrastructure is less robust (5,6). This suggests that enhanced awareness, timely screening, and therapeutic intervention may be mitigating the progression to end-stage liver disease in this sample. Conversely, the rate of moderate-to-severe fibrosis was in keeping with other studies of treatment-naive or recently diagnosed patients, indicating a typical disease spectrum for the region (7).

From a clinical perspective, these findings highlight the importance of integrating quantitative viral load measurement into the routine management of chronic hepatitis patients, as it provides prognostic insight into the risk of advanced liver disease. The strong associations observed in this study support the argument for risk stratification based on viral load, which could guide decisions regarding intensity of monitoring, timing of therapeutic intervention, and prioritization of patients for specialist referral (3). This is particularly pertinent in resource-limited settings, where judicious allocation of healthcare resources is essential. Moreover, the data suggest that early detection and aggressive management of high viral load cases could substantially reduce the burden of cirrhosis and HCC, improving long-term outcomes (4, 9).

Several strengths enhance the credibility of this research, including the use of standardized, PCR-based viral load quantification, robust statistical methods, and careful adjustment for potential confounders. The multi-center design and inclusion of a broad age range increase the relevance of the findings to real-world clinical practice. Nonetheless, some limitations should be acknowledged. The cross-sectional design precludes assessment of causality or disease progression over time. The sample, though adequately powered for key associations, may not capture the full spectrum of disease heterogeneity in the general population, limiting generalizability beyond similar clinical contexts. Reliance on laboratory and medical record data may introduce classification or information bias, particularly in the staging of liver fibrosis and cirrhosis, which can be affected by interobserver variability and incomplete clinical data. Additionally, exclusion of co-infected or previously treated patients narrows the applicability of results (2, 7, 10).

Given these limitations, future research should aim for larger, longitudinal cohort studies to elucidate causal relationships and temporal dynamics between viral load fluctuations and liver disease progression. Integration of non-invasive fibrosis assessment tools, assessment of treatment response, and expansion to diverse populations would provide further clarity on the prognostic value of viral load. Investigations into the biological mechanisms underlying differential disease trajectories among individuals with similar viral loads could yield new therapeutic targets and precision medicine strategies. this study affirms the pivotal role of HBV and HCV viral loads in determining liver disease severity among chronic patients in a Pakistani context, reinforces the value of quantitative virological monitoring, and underscores the need for targeted public health interventions and further longitudinal research to optimize patient outcomes (1–8).

CONCLUSION

This study demonstrates that higher PCR-quantified HBV and HCV viral loads are strongly associated with greater severity of liver fibrosis, cirrhosis, and hepatocellular carcinoma in chronic hepatitis patients, highlighting the critical role of viral load as a prognostic marker for disease progression. These findings underscore the importance of routine viral load monitoring to enable earlier risk

stratification, timely intervention, and tailored management strategies in human healthcare settings, particularly in regions with a high burden of chronic viral hepatitis. Clinically, incorporating quantitative viral load assessment can improve patient outcomes by informing surveillance intensity and therapeutic decisions, while future research should focus on longitudinal validation and integration of viral load–based algorithms into standard care protocols to further optimize the management and prevention of advanced liver disease.

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