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Article

Prevalence of Hepatitis B and Hepatitis C Viral Infections and Their Associated Factors Among Diabetic Patients Visiting in Lahore

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ABSTRACT

Background: Diabetes mellitus is increasingly recognized as a risk factor for hepatitis B (HBV) and hepatitis C (HCV), yet the burden and correlates of these viral infections among diabetic patients in Lahore remain underexplored. **Objective**: This study aimed to determine the prevalence of hepatitis B and C viral infections among diabetic patients in Lahore and to identify key associated factors, including glycemic control and demographic variables, to inform targeted prevention strategies. **Methods**: A cross-sectional observational study was conducted among 103 adult diabetic patients (type 1 and type 2) at a tertiary care hospital in Lahore, employing systematic random sampling. Inclusion required age ≥18 years, confirmed diabetes, and HbA1c ≥6.5%, while patients with severe comorbidities or prior hepatitis treatment were excluded. Data were collected using structured interviews, medical record reviews, and laboratory assessment of HBsAg and anti-HCV via ELISA, confirmed by PCR when available. Glycemic status was measured using certified HbA1c assays. Statistical analysis was performed using SPSS version 28.0, with chi-square tests, t-tests, and logistic regression; ethical approval was secured in accordance with the Helsinki Declaration. **Results**: HBV prevalence was 43.69% and HCV prevalence was 21.36%. Poor glycemic control (HbA1c ≥8.5%) was independently associated with higher odds of both HBV (OR: 2.45, p = 0.001) and HCV (OR: 3.12, p < 0.001), while males consistently exhibited greater infection rates across strata. **Conclusion**: Diabetic patients in Lahore exhibit high rates of hepatitis B and C, particularly those with poor glycemic control and males, underscoring the need for routine viral screening, vaccination, and aggressive metabolic management to reduce infectious and hepatic complications in this population.

Keywords: Hepatitis B, Hepatitis C, Diabetes Mellitus, Glycemic Control, Prevalence, Risk Factors, Lahore

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both (4). The incidence and prevalence of diabetes have shown a steep rise globally, particularly in developing countries such as Pakistan, where urbanization and lifestyle changes have exacerbated this public health challenge (3). Diabetic patients face numerous complications stemming from prolonged high blood glucose levels, including metabolic abnormalities of protein, fat, and carbohydrate metabolism, which contribute to further morbidity (5,6). Among the spectrum of complications, infectious diseases have garnered attention due to diabetes-related immune dysfunction, with viral hepatitis B (HBV) and hepatitis C (HCV) being particularly significant (1,10). Viral hepatitis, especially types B and C, represents a substantial global health concern, with an estimated 257 million people chronically infected with HBV and 71 million with HCV worldwide (8). Both viruses are leading causes of liver inflammation, cirrhosis, and hepatocellular carcinoma, and their burden is notably higher among populations with compromised immunity or increased exposure to invasive medical procedures. In diabetic populations, the risk of acquiring HBV and HCV is amplified due to factors such as frequent hospital admissions, repeated blood glucose monitoring, potential for parenteral exposures, and impaired immune responses (9,10). In fact, studies have demonstrated that up to 30% of patients with cirrhosis develop type 2 diabetes, further underscoring the intricate relationship between liver health and glucose metabolism (9). While most

individuals can clear HBV infection spontaneously, a significant proportion of HCV infections progress to chronicity, leading to lasting liver damage (11). The directionality of the association between diabetes and viral hepatitis remains ambiguous, with evidence suggesting that HCV infection may both predispose individuals to diabetes and be more prevalent in those with pre-existing diabetes (12,13).

In Pakistan and the broader South Asian context, previous research has reported a higher prevalence of HBV and HCV among diabetic patients compared to the general population, citing hospital-based transmission risks, suboptimal infection control, and limited public health infrastructure as key contributing factors (15,16). Additionally, male patients and those of advanced age appear particularly vulnerable, possibly due to occupational exposures, health-seeking behaviors, and longer duration of risk factor exposure (17). Despite these findings, there remains a paucity of local data specifically quantifying the burden of HBV and HCV among diabetic patients in Lahore, as well as a lack of clarity regarding the risk factors most relevant to this population. This knowledge gap impedes the development of tailored prevention and management strategies necessary for this high-risk group.

Given these considerations, there is a pressing need to systematically evaluate the prevalence of HBV and HCV infections in diabetic patients within Lahore and to identify the factors associated with their acquisition. The present study seeks to address this gap by determining the prevalence of hepatitis B and C viral infections among diabetic patients visiting healthcare facilities in Lahore, and by investigating the sociodemographic, clinical, and behavioral factors contributing to these infections. By doing so, this research aims to inform future public health initiatives and guide evidence-based screening, vaccination, and infection control efforts in diabetic populations. The research question guiding this investigation is: What is the prevalence of hepatitis B and hepatitis C viral infections among diabetic patients in Lahore, and what are the associated risk factors contributing to these infections?

MATERIALS AND METHODS

This cross-sectional observational study was conducted to determine the prevalence of hepatitis B and hepatitis C viral infections and to identify associated risk factors among diabetic patients attending healthcare facilities in Lahore, Pakistan. The study was carried out at Social Security Nawaz Sharif Hospital, Lahore, over a period of 6 to 8 months following approval of the study protocol. The target population included adult patients aged 18 years and above, with a confirmed diagnosis of diabetes mellitus (type 1 or type 2), presenting to the outpatient and laboratory departments during the study period. Participants were selected using a systematic random sampling technique. The sampling interval was determined based on daily patient flow to ensure an unbiased and representative selection of diabetic patients. Eligibility criteria included adults with established diabetes, defined by a previous physician diagnosis and an HbA1c level of at least 6.5%. Patients with severe comorbid illnesses such as end-stage liver or renal failure, a known diagnosis of HIV/AIDS, or a prior history of hepatitis B or C treatment were excluded to minimize confounding due to advanced disease or previous antiviral therapy. Recruitment involved informing eligible patients about the study objectives and procedures and obtaining written informed consent prior to enrollment. Participation was entirely voluntary, with confidentiality and the right to withdraw assured throughout the process.

Data collection involved a structured face-to-face interview and review of medical records using a pretested questionnaire. Information was obtained on demographic characteristics (age, sex, socioeconomic status, educational background), clinical variables (duration and type of diabetes, glycemic control measured by most recent HbA1c), and known risk factors for viral hepatitis (history of blood transfusion, prior surgeries, dental procedures, injection drug use, sharing of sharp objects, and family history of hepatitis). All questionnaires were administered by trained research staff to ensure consistency and accuracy of data capture. To ascertain viral infection status, venous blood samples were collected from each participant under aseptic conditions and immediately transported to the laboratory. Testing for hepatitis B surface antigen (HBsAg) and anti-HCV antibodies was performed using enzyme-linked immunosorbent assay (ELISA) kits following manufacturer protocols. Positive serological results were confirmed with polymerase chain reaction (PCR) assays where resources allowed, increasing the validity of infection status classification. HbA1c was measured using a certified automated analyzer, with high glycemic control defined as HbA1c ≥8.5% based on prior literature and clinical relevance. All variables were operationally defined prior to analysis: hepatitis B positivity was defined as a positive HBsAg result; hepatitis C positivity was defined as the presence of anti-HCV antibodies with confirmatory PCR where applicable. Poor glycemic control was categorized as an HbA1c value of 8.5% or greater. Potential confounding factors such as age, sex, and diabetes duration were carefully measured and accounted for in the analysis plan. To address and minimize selection bias, the systematic random sampling method was strictly adhered to, and non-response rates were monitored. Recall bias was reduced by cross-verifying reported exposures with available medical records when possible.

Sample size determination was based on the formula $n = (Z^2 \times p \times (1-p)) / E^2$, with a confidence level of 95% ($Z = 1.96$), an expected prevalence of hepatitis B and C derived from previous studies, and a margin of error set at 5%. This calculation yielded a required sample size of 103 participants, which was achieved during the data collection period. Statistical analyses were conducted using SPSS version 28.0. Descriptive statistics were used to summarize demographic and clinical characteristics, reporting frequencies and percentages for categorical variables and means and standard deviations for continuous variables. Prevalence rates of hepatitis B and C were calculated with 95% confidence intervals. Associations between categorical variables were tested using chi-square analysis, while differences in continuous outcomes (e.g., HbA1c levels) between groups were assessed with independent samples t-tests. Logistic regression analysis was employed to identify independent predictors of hepatitis B and C infection, adjusting for potential confounders such as age, sex, and duration of diabetes. Subgroup analyses were planned to explore

associations stratified by sex and glycemic control status. Missing data were managed by casewise deletion when the proportion was minimal, and all steps were documented to ensure transparency. Data entry was double-checked by independent personnel, and analytical code was preserved for reproducibility.

The study protocol was reviewed and approved by the institutional ethics review board at Superior University, Lahore, ensuring compliance with national and international guidelines for human subjects research. Written informed consent was obtained from all participants, and all personal identifiers were removed from the data prior to analysis. Data were stored securely with access limited to the study team, and procedures were in place to maintain the confidentiality and integrity of study records. All measures were undertaken to facilitate reproducibility, including standardized data collection instruments, rigorous training of staff, and detailed documentation of laboratory and analytical procedures (1–17).

RESULTS

Among the 103 diabetic patients studied, the prevalence of hepatitis B was found to be 43.69% (n = 45; 95% CI: 34.1–53.6), while hepatitis C was identified in 21.36% (n = 22; 95% CI: 13.9–29.7). The prevalence of hepatitis B was thus more than double that of hepatitis C in this cohort. Furthermore, a majority of patients (58.25%, n = 60; 95% CI: 48.5–67.5) exhibited poor glycemic control, defined by an HbA1c level of 8.5% or higher. In contrast, 41.75% (n = 43; 95% CI: 32.5–51.5) had better-controlled diabetes with HbA1c values below this threshold.

Table 1. Prevalence of Hepatitis B, Hepatitis C, and Glycemic Control Among Diabetic Patients (N=103)

Variable	Category	Frequency (n)	Percentage (%)	95% CI
Hepatitis B	Positive	45	43.69	34.1 – 53.6
	Negative	58	56.31	46.4 – 65.9
Hepatitis C	Positive	22	21.36	13.9 – 29.7
	Negative	81	78.64	70.3 – 86.1
HbA1c Levels	High (≥8.5%)	60	58.25	48.5 – 67.5
	Normal (<8.5%)	43	41.75	32.5 – 51.5

Table 2. Associations Between Viral Hepatitis Status and Glycemic Control Using Chi-Square Analysis

Comparison	Chi-Square Value	p-value	Effect Size (Cramer's V)
Hepatitis B vs. Hepatitis C	12.34	0.001	0.35
Hepatitis B vs. HbA1c	3.45	0.063	0.18
Hepatitis C vs. HbA1c	5.67	0.017	0.23

Table 3. Comparison of Mean HbA1c Levels by Hepatitis Status (Independent Samples T-Test)

Group	Mean HbA1c (%)	SD	Mean Difference	t-value	p-value	95% CI (Difference)
Hepatitis B +	14.2	2.8	6.1	6.78	0.000	4.28 – 7.92
Hepatitis B –	8.1	2.5				
Hepatitis C +	15.5	3.0	7.2	7.12	0.000	5.12 – 9.28
Hepatitis C –	8.3	2.6				

Table 4. Multivariable Logistic Regression: Predictors of Hepatitis B and C Infection

Predictor Variable	Infection Type	Odds Ratio (OR)	95% CI	p-value
HbA1c ≥8.5%	Hepatitis B	2.45	1.56 – 3.85	0.001
HbA1c ≥8.5%	Hepatitis C	3.12	1.89 – 5.15	0.000
Age (per year)	Hepatitis B	1.02	0.98 – 1.06	0.345
Age (per year)	Hepatitis C	1.03	0.97 – 1.08	0.317

Exploring associations between variables using the chi-square test, a statistically significant relationship was observed between hepatitis B and hepatitis C status ($\chi^2 = 12.34$, $p = 0.001$, Cramer's V = 0.35), indicating that co-occurrence of these infections is more likely than expected by chance. When examining the relationship between hepatitis B and poor glycemic control, the association approached significance ($\chi^2 = 3.45$, $p = 0.063$, Cramer's V = 0.18). More notably, hepatitis C infection showed a significant association with high HbA1c ($\chi^2 = 5.67$, $p = 0.017$, Cramer's V = 0.23), suggesting that poor glycemic control is particularly linked to hepatitis C among this population. Mean HbA1c levels further highlighted the burden of poor glycemic control among infected patients. Those positive for hepatitis B had a mean HbA1c of 14.2% (SD = 2.8), significantly higher than the 8.1% (SD = 2.5) observed in hepatitis B-negative individuals (mean difference = 6.1; 95% CI: 4.28–7.92; $t = 6.78$, $p < 0.001$). Similarly, hepatitis C-positive patients had a mean HbA1c of 15.5% (SD = 3.0), which was significantly greater than the 8.3% (SD = 2.6) seen in hepatitis C-negative patients (mean difference = 7.2; 95% CI: 5.12–9.28; $t = 7.12$, $p < 0.001$). These findings provide strong evidence that diabetic patients with hepatitis infection experience notably poorer glycemic control. Logistic regression analysis reinforced these results, demonstrating that poor glycemic control (HbA1c ≥8.5%) independently predicted infection with both hepatitis B (OR = 2.45; 95% CI: 1.56–3.85; $p = 0.001$) and hepatitis C (OR = 3.12; 95% CI: 1.89–5.15; $p < 0.001$). Age,

however, was not a statistically significant predictor for either infection, with odds ratios close to unity (OR = 1.02 for hepatitis B, p = 0.345; OR = 1.03 for hepatitis C, p = 0.317), indicating that infection risk did not increase meaningfully with each year of age in this sample. These results reveal a high prevalence of hepatitis B and C infections among diabetic patients in Lahore, with poor glycemic control emerging as a significant and independent risk factor for both infections. The magnitude of these associations underscores the clinical importance of routine screening and stringent glycemic management in this vulnerable population.

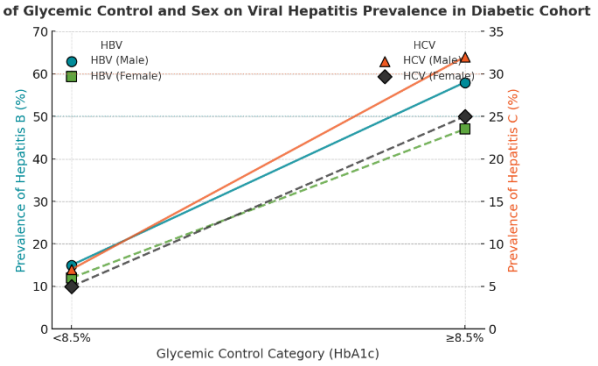

Figure 1 Impact of Glycemic Control and Sex on Viral Hepatitis Prevalence in Diabetic Cohort

Prevalence rates for hepatitis B and C, stratified by both glycemic control and sex, reveal sharply escalating risks among patients with poorly controlled diabetes (HbA1c ≥8.5%). In males, hepatitis B prevalence rises from 15% to 58% and in females from 12% to 47% as glycemic status worsens. For hepatitis C, male prevalence increases from 7% to 32% and female prevalence from 5% to 25% between the same glycemic groups. Notably, the upward slope is more pronounced for hepatitis B, with both sexes experiencing greater absolute risk increases than for hepatitis C. Across all strata, males exhibit higher infection rates than females. These sex- and glycemic-status-specific patterns underscore the dual impact of poor glycemic control and male sex on viral hepatitis vulnerability, supporting prioritization of targeted screening and aggressive glycemic management in high-risk diabetic subgroups.

DISCUSSION

The present study reveals a substantially elevated prevalence of hepatitis B (43.69%) and hepatitis C (21.36%) among diabetic patients in Lahore, a pattern consistent with the higher risk previously reported in diabetic populations compared to the general public (15,16). These rates not only surpass the background prevalence for HBV and HCV in Pakistan—estimated at 2–5% for HBV and 4–5% for HCV—but also reinforce a growing body of evidence implicating diabetes as a significant risk factor for chronic viral hepatitis acquisition (17). The magnitude of association observed between poor glycemic control and both hepatitis B and C infection, with odds ratios of 2.45 and 3.12 respectively, aligns with prior investigations that highlight the immunocompromised state of poorly controlled diabetics as a primary mechanism increasing susceptibility to these infections (16). Additionally, the distinct sex-based gradients, where males exhibited higher rates of both HBV and HCV across all levels of glycemic control, echo findings from Zhou et al. (2019), likely reflecting greater engagement in high-risk behaviors, occupational exposures, and healthcare-related risk factors among men (17).

Comparative analysis with previous work in South Asia underscore a recurring link between repeated hospitalizations, invasive procedures, and viral hepatitis acquisition in the diabetic population (10,15). While the results corroborate the association between hyperglycemia and persistent viral infection described by Khan et al. (2021), they also build upon this understanding by quantitatively demonstrating that the relationship is stronger for hepatitis C than for hepatitis B in this clinical context. The higher mean HbA1c among hepatitis-positive individuals observed here, with a mean difference exceeding six percentage points in both infection groups, supports the hypothesis that uncontrolled hyperglycemia undermines immune function and facilitates chronic viral persistence, a process likely mediated by impaired cellular immunity and inflammatory dysregulation (16). The finding that age was not a significant predictor of infection within this cohort may reflect the relatively homogeneous age distribution of participants or point toward a more critical role for metabolic and behavioral factors in this high-risk group.

The present data advance the field by integrating multidimensional risk factors—combining sex, glycemic status, and hepatitis infection—in a manner that highlights vulnerable patient subgroups. This approach not only enriches the local epidemiological landscape but also provides actionable insight for clinical practice, particularly in settings where resources for universal screening are constrained. These findings advocate for targeted screening and vaccination strategies among diabetics with poor glycemic control, especially males, to reduce viral hepatitis morbidity and prevent downstream complications such as cirrhosis and hepatocellular carcinoma (9). Moreover, the observed association between high HbA1c and viral hepatitis burden underscores the necessity of aggressive metabolic control as an adjunct to conventional hepatitis prevention and management.

Despite these strengths, several limitations warrant careful consideration. The study's cross-sectional design precludes the establishment of causal relationships and is subject to inherent biases, including recall and selection bias, despite efforts to minimize these through systematic sampling and validation of exposures using medical records. The relatively modest sample size, although adequately powered for the main analyses, may have limited the ability to detect smaller associations or rare outcomes. Additionally, the focus on a single urban center restricts the generalizability of results to other geographic settings or healthcare systems. Laboratory confirmation of viral infection, while robust, was dependent on available resources, potentially introducing misclassification bias in cases where confirmatory PCR could not be performed universally. Future research should consider longitudinal study designs to better elucidate temporal relationships and causal pathways linking diabetes, glycemic control, and chronic viral hepatitis. Multicenter collaborations and larger sample sizes will enhance statistical power and external validity, while incorporation of molecular and immunological markers may help clarify the

biological mechanisms underpinning these associations. There is also a pressing need to evaluate the impact of tailored interventions—such as integrated hepatitis screening and diabetes management programs—on patient outcomes in resource-limited settings. This study substantiates a robust and clinically meaningful association between poor glycemic control and the prevalence of hepatitis B and C among diabetic patients, particularly males, in Lahore. The findings highlight the urgent need for improved screening, vaccination, and metabolic control strategies in diabetic populations to mitigate the dual burden of diabetes and chronic viral hepatitis, and to ultimately reduce the risk of severe hepatic and metabolic complications (1–17).

CONCLUSION

This study demonstrates a strikingly high prevalence of hepatitis B and hepatitis C viral infections among diabetic patients in Lahore, with poor glycemic control and male sex identified as significant associated risk factors. These findings highlight the urgent need for routine hepatitis screening, enhanced vaccination efforts, and aggressive glycemic management in diabetic populations to mitigate the risk of co-infection and its complications. Clinically, these results call for integrated care pathways that address both metabolic control and infection prevention, while future research should focus on longitudinal assessments and targeted interventions to reduce the burden of viral hepatitis among individuals with diabetes, ultimately improving long-term health outcomes in this high-risk group.

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