ISSN : (Online): 3008-038X
Abbreviation: J Derm Cse Rep
DOI: 10.61705/jdcr.18.2.2025.239.247

Journal of Dermatological Case Reports

Differential Impact of Hepatitis B and C on Insulin Resistance: A Cross-sectional Study

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Abstract:

Background
Chronic hepatitis B (HBV) and hepatitis C (HCV) infections are increasingly recognized for their metabolic implications, particularly their association with insulin resistance. This study aimed to evaluate the relationship between insulin resistance and chronic viral hepatitis.

Methods
This cross-sectional study included 167 participants comprising HBV (n=58), HCV (n=54), and healthy controls (n=55). Fasting glucose and insulin levels were measured, and insulin resistance was assessed using the Homeostatic Model Assessment (HOMA-IR). Statistical analyses included ANOVA, chi-square test, correlation, and multivariate logistic regression.

Results
HOMA-IR was significantly higher in HCV (3.67 ± 1.28) and HBV (2.51 ± 1.01) compared to controls (1.96 ± 0.74) ($p<0.001$). Insulin resistance prevalence was highest in HCV (72.2%), followed by HBV (46.6%) and controls (20.0%) ($p<0.001$). HCV infection was a strong independent predictor of insulin resistance (AOR: 4.28), along with BMI >25 kg/m² and age >40 years. Significant positive correlations were observed between HOMA-IR and BMI, fasting glucose, and liver enzymes.

Conclusion
Chronic viral hepatitis, particularly HCV infection, is strongly associated with insulin resistance. Early metabolic screening in these patients is essential for comprehensive disease management and prevention of complications.

Keywords:

Insulin resistance, Hepatitis B, Hepatitis C, HOMA-IR, Chronic liver disease

Received : 25-03-2025 **Revised : 30-03-2025** **Accepted: 01-04-2025** **Published : 05-04-2025**

ISSN : (Online): 3008-038X
Abbreviation: J Derm Cse Rep
DOI: 10.61705/jdcr.18.2.2025.239.247

Introduction

Chronic viral hepatitis remains a major global health burden, with an estimated 296 million individuals living with chronic hepatitis B virus (HBV) infection and approximately 58 million with chronic hepatitis C virus (HCV) infection worldwide [1,2]. These infections are not only leading causes of cirrhosis and hepatocellular carcinoma but are increasingly recognized for their systemic metabolic implications, particularly their association with insulin resistance (IR) and related metabolic disorders [3,4].

Insulin resistance, defined as a diminished biological response to circulating insulin, plays a central role in the pathogenesis of type 2 diabetes mellitus and metabolic syndrome [5]. Emerging evidence suggests that chronic liver diseases, especially viral hepatitis, may directly contribute to the development of IR through complex mechanisms involving hepatic inflammation, cytokine release, and interference with insulin signaling pathways [5,6].

Hepatitis C virus infection has been more consistently associated with insulin resistance [7]. HCV is known to exert a direct cytopathic effect on hepatocytes and disrupt insulin signaling via upregulation of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), leading to impaired glucose metabolism [8]. Literature have demonstrated that the prevalence of insulin resistance in patients with chronic HCV infection ranges from 30% to 70%, significantly higher than in non-infected populations [7,8]. Furthermore, IR in HCV patients has been linked to increased hepatic steatosis, accelerated fibrosis progression, and reduced response to antiviral therapy [8].

In contrast, the relationship between hepatitis B virus infection and insulin resistance is less clearly defined and appears to be more heterogeneous [9]. While some report a modest association between HBV infection and altered glucose metabolism, others suggest a neutral or even protective effect [9,10]. The underlying mechanisms remain uncertain but may involve differences in viral biology, host immune response, and degree of hepatic inflammation compared to HCV infection [10].

The coexistence of insulin resistance in patients with chronic viral hepatitis has important clinical implications [5]. IR not only contributes to disease progression but also increases the risk of developing type 2 diabetes mellitus, cardiovascular disease, and non-alcoholic fatty liver disease (NAFLD), thereby compounding the overall morbidity and mortality associated with these infections [5]. Early identification of insulin resistance in patients with HBV and HCV infection may therefore aid in risk stratification and guide therapeutic interventions. Hence, this study aimed to evaluate the association of insulin resistance with hepatitis B and hepatitis C infections in a cross-sectional cohort, thereby contributing to a better understanding of the metabolic consequences of chronic viral hepatitis.

Material and methods

Study Design and Setting

This cross-sectional, observational study was conducted in the Department of Biochemistry in collaboration with the Department of General Medicine at Vyas Medical College and Associated Hospital. over a period of 12 months, from January 2023 to December 2023. The study aimed to evaluate the association between insulin resistance and chronic viral hepatitis B and C infections among adult patients attending outpatient and inpatient services.

Study Population

The study population comprised adult patients aged $\geqslant$ 18 years who were diagnosed with chronic hepatitis B virus (HBV) or hepatitis C virus (HCV) infection. Chronic HBV infection was defined as persistence of hepatitis B surface antigen (HBsAg) positivity for more than six months, while chronic

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HCV infection was confirmed by the presence of anti-HCV antibodies with detectable HCV RNA. A control group of apparently healthy individuals, matched for age and sex, and negative for both HBsAg and anti-HCV antibodies, was also included for comparison. Exclusion criteria comprised patients with known diabetes mellitus, metabolic syndrome, obesity (body mass index $\geqslant$30 kg/m^2), alcohol use disorder, co-infection with both HBV and HCV, human immunodeficiency virus (HIV) infection, pregnancy, use of medications affecting glucose metabolism (such as corticosteroids), and those with other chronic liver diseases including autoimmune hepatitis or non-alcoholic fatty liver disease.

Sample Size and Sampling Technique

The sample size for the present study was calculated using the standard formula for estimating a single proportion in cross-sectional studies: n=Z2×P×(1−P)d2, where n is the required sample size, Z is the standard normal deviate corresponding to the desired confidence level (1.96 for 95% confidence), P is the expected prevalence of insulin resistance among patients with chronic viral hepatitis derived from previous studies, and d is the allowable margin of error (precision). Based on prior study by Mishra et al., the prevalence of insulin resistance among patients with chronic hepatitis (particularly HCV) has been reported to be approximately 30% (P = 0.30) [7]. Considering a 95% confidence level (Z = 1.96) and a margin of error of 7%, the sample size was calculated as 167 participants and were enrolled, including patients with hepatitis B infection, hepatitis C infection, and healthy controls. Participants were recruited using a consecutive sampling technique until the desired sample size was approached.

Data Collection and Clinical Assessment

Detailed demographic data including age, sex, and relevant clinical history were recorded using a structured proforma. A thorough clinical examination was performed for all participants. Anthropometric measurements including height, weight, and body mass index (BMI) were recorded using standardized techniques. Blood pressure was measured using a calibrated sphygmomanometer.

Laboratory Investigations

Venous blood samples were collected from all participants after an overnight fast of at least 8–10 hours. Laboratory investigations included fasting plasma glucose (FPG), fasting serum insulin levels, liver function tests, and viral markers. HBsAg and anti-HCV antibodies were detected using enzyme-linked immunosorbent assay (ELISA). HCV RNA testing was performed for confirmation of active infection where applicable.

Insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), calculated using the formula: fasting insulin (μIU/mL) × fasting glucose (mg/dL) / 405. A HOMA-IR value greater than 2.5 was considered indicative of insulin resistance.

Outcome Measures

The primary outcome of the study was the prevalence of insulin resistance among patients with HBV and HCV infection compared to healthy controls. Secondary outcomes included comparison of mean HOMA-IR values among the study groups and assessment of the relationship between insulin resistance and clinical or biochemical parameters.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 20.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. The normality of data distribution was assessed using the Kolmogorov–Smirnov test. Comparisons between groups were performed using independent t-test or one-way analysis of variance

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(ANOVA) for continuous variables and chi-square test for categorical variables. Correlation between insulin resistance and clinical parameters was assessed using Pearson or Spearman correlation coefficient as appropriate. A p-value of <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee prior to commencement of the study. Written informed consent was obtained from all participants before enrollment. Confidentiality of patient information was strictly maintained, and the study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Results

The mean age of participants differed significantly among the groups (p=0.021), with patients in the HCV group being relatively older (44.2 ± 11.8 years) compared to HBV (39.8 ± 10.6 years) and controls (38.5 ± 9.9 years). Gender distribution was comparable across all groups (p=0.807), indicating no significant sex-related bias. The mean BMI did not differ significantly (p=0.099), suggesting comparable baseline anthropometry. However, both systolic and diastolic blood pressure were significantly higher in the HCV group compared to HBV and controls (p=0.041 and p=0.048, respectively), indicating a trend toward higher cardiovascular risk in HCV-infected individuals (Table 1).

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants.

Variable	HBV (n=58)	HCV (n=54)	Controls (n=55)	p-value
	Frequency (%)/mean ± SD			
Age (years)	39.8 ± 10.6	44.2 ± 11.8	38.5 ± 9.9	0.021
Gender				
Female	21 (26.2%)	22 (40.7%)	22 (40.0%)	0.807
Male	37 (63.8%)	32 (59.3%)	33 (60.0%)	
BMI (kg/m²)	24.1 ± 2.8	24.9 ± 3.1	23.8 ± 2.5	0.099
Systolic BP (mmHg)	122.6 ± 11.4	126.3 ± 12.8	120.4 ± 10.9	0.041
Diastolic BP (mmHg)	78.5 ± 7.2	80.8 ± 8.1	76.9 ± 6.8	0.048

Fasting plasma glucose, fasting insulin levels, and HOMA-IR values were significantly elevated in patients with HCV infection compared to HBV and control groups (all p<0.001). The HBV group also demonstrated higher values compared to controls, though to a lesser extent. Liver enzymes (ALT and AST) were markedly elevated in both HBV and HCV groups compared to controls, with the highest levels observed in HCV patients (p<0.001). These findings indicate a significant metabolic and hepatic derangement, particularly in HCV infection (Table 2).

Table 2. Comparison of Biochemical Parameters among Study Groups.

Parameter	HBV (n=58)	HCV (n=54)	Controls (n=55)	p-value
	mean ± SD			
FPG (mg/dL)	94.6 ± 11.8	101.2 ± 13.5	89.3 ± 10.2	<0.001
FI (µIU/mL)	10.8 ± 4.2	14.6 ± 5.3	8.9 ± 3.1	<0.001

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HOMA-IR	**2.51 ± 1.01**	**3.67 ± 1.28**	**1.96 ± 0.74**	**<0.001**
ALT (U/L)	**54.2 ± 21.3**	**68.7 ± 25.6**	**29.8 ± 10.5**	**<0.001**
AST (U/L)	**49.5 ± 18.7**	**62.4 ± 22.1**	**27.6 ± 9.8**	**<0.001**

The overall prevalence of insulin resistance was significantly higher among patients with viral hepatitis compared to controls ($p<0.001$). The highest prevalence was observed in the HCV group (72.2%), followed by the HBV group (46.6%), while controls showed a substantially lower prevalence (20.0%). This demonstrates a strong association between chronic viral hepatitis, particularly HCV infection, and insulin resistance (Table 3).

Table 3. Prevalence of Insulin Resistance among Study Groups.

Group	IR Present (n=57)	IR Absent (n=110)	Prevalence (%)	p-value
HBV (n=58)	**27**	**31**	**46.60%**	**<0.001**
HCV (n=54)	**39**	**15**	**72.20%**	
Controls (n=55)	**11**	**44**	**20.00%**	

Post-hoc analysis revealed that both HBV and HCV groups had significantly higher HOMA-IR values compared to controls ($p=0.012$ and $p<0.001$, respectively). Additionally, HCV patients exhibited significantly higher insulin resistance than HBV patients (mean difference 1.16, $p<0.001$). These findings confirm a graded increase in insulin resistance from controls to HBV to HCV groups (Table 4).

Table 4. Post-hoc Analysis of Mean HOMA-IR between Study Groups.

Comparison	Mean Difference	p-value
HBV vs Controls	**0.55**	**0.012**
HCV vs Controls	**1.71**	**<0.001**
HCV vs HBV	**1.16**	**<0.001**

HOMA-IR showed a significant positive correlation with age ($r=0.21$, $p=0.007$), BMI ($r=0.34$, $p<0.001$), and fasting plasma glucose ($r=0.42$, $p<0.001$), indicating that insulin resistance increases with advancing age, higher adiposity, and glycemic levels. Additionally, moderate positive correlations were observed with liver enzymes ALT ($r=0.29$, $p<0.001$) and AST ($r=0.25$, $p=0.002$), suggesting a relationship between hepatic inflammation and insulin resistance (Table 5).

Table 5. Correlation of HOMA-IR with Clinical and Biochemical Parameters.

Variable	Correlation Coefficient (r)	p-value
Age	**0.21**	**0.007**
BMI	**0.34**	**<0.001**
Fasting Glucose	**0.42**	**<0.001**
ALT	**0.29**	**<0.001**
AST	**0.25**	**0.002**

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Multivariate logistic regression analysis identified HCV infection as a strong independent predictor of insulin resistance (AOR: 4.28, 95% CI: 2.01–9.12, p<0.001), followed by HBV infection (AOR: 2.11, 95% CI: 1.02–4.36, p=0.043). Additionally, BMI >25 kg/m² (AOR: 2.76, p=0.003) and age >40 years (AOR: 1.89, p=0.046) were also significant predictors. These findings highlight both viral and metabolic factors as independent contributors to insulin resistance (Table 6).

Table 6. Multivariate Logistic Regression Analysis for Predictors of Insulin Resistance.

Variable	Adjusted Odds Ratio (AOR)	95% CI	p-value
HCV Infection	**4.28**	**2.01 – 9.12**	**<0.001**
HBV Infection	**2.11**	**1.02 – 4.36**	**0.043**
BMI (>25 kg/m²)	**2.76**	**1.39 – 5.48**	**0.003**
Age (>40 years)	**1.89**	**1.01 – 3.52**	**0.046**

Discussion

The present study demonstrates a significant association between chronic viral hepatitis and insulin resistance, with a clear gradient observed across groups—lowest in controls, intermediate in HBV, and highest in HCV infection. The prevalence of insulin resistance was markedly higher in HCV patients (72.2%) compared to HBV (46.6%) and controls (20.0%) (p<0.001), indicating a strong metabolic impact of chronic HCV infection. These findings are consistent with previous studies by Shawky et al., Yu et al., and Lee et al., reporting insulin resistance prevalence ranging from 30% to 70% in HCV-infected individuals [11,12,13], and reinforce the concept that HCV has a more pronounced diabetogenic potential compared to HBV.

The significantly elevated fasting insulin levels and HOMA-IR in HCV patients (3.67 ± 1.28) compared to HBV (2.51 ± 1.01) and controls (1.96 ± 0.74) (p<0.001) further substantiate the metabolic derangement associated with HCV. Similar findings have been reported by Kukla et al., and Kralj et al., who demonstrated higher HOMA-IR values in HCV patients and linked them to disease severity and treatment response [14,15]. The underlying mechanism is thought to involve direct interference of HCV proteins with insulin signaling pathways, particularly through upregulation of pro-inflammatory cytokines such as TNF-α and IL-6, leading to impaired insulin receptor substrate (IRS-1) activity and increased hepatic gluconeogenesis [16]. Additionally, HCV-induced hepatic steatosis and mitochondrial dysfunction further exacerbate insulin resistance [17].

In contrast, although HBV infection was also associated with a significantly higher prevalence of insulin resistance compared to controls (46.6% vs 20.0%), the magnitude of association was comparatively lower (AOR: 2.11 vs 4.28 for HCV). This aligns with existing literature by Kaya et al., and Riveiro-Barciela et al., where HBV has shown inconsistent or weaker associations with insulin resistance [18,19]. The relatively modest effect observed in HBV patients in this study may be attributed to indirect mechanisms such as chronic low-grade inflammation rather than direct viral interference with insulin signaling, which is more characteristic of HCV infection [20].

The study also identified independent predictors of insulin resistance, including HCV infection (AOR:

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4.28, p<0.001), elevated BMI (>25 kg/m²) (AOR: 2.76, p=0.003), and age >40 years (AOR: 1.89, p=0.046). These findings are in agreement with earlier studies Bose et al., and Desbois et al., highlighting the synergistic effect of viral and host metabolic factors in the development of insulin resistance [21,22]. The significant positive correlation of HOMA-IR with BMI (r=0.34) and fasting glucose (r=0.42) (both p<0.001) underscores the role of adiposity and glycemic dysregulation in amplifying insulin resistance in these patients. Furthermore, the observed correlations between HOMA-IR and liver enzymes (ALT: r=0.29; AST: r=0.25) suggest that hepatic inflammation and injury may contribute to impaired insulin sensitivity, supporting the concept of "hepatogenous diabetes" [23].

The biochemical profile observed in this study further supports the metabolic burden of viral hepatitis. HCV patients exhibited significantly higher fasting glucose, insulin levels, and transaminases compared to both HBV and controls (p<0.001), indicating more severe metabolic and hepatic dysfunction. Similar patterns have been reported in studies by Yilmaz et al., and Zampino et al., where HCV infection is frequently associated with higher rates of insulin resistance, hepatic steatosis, and progression to fibrosis and cirrhosis [24,25]. The higher systolic and diastolic blood pressure observed in HCV patients (p=0.041 and p=0.048) also suggests a clustering of cardiometabolic risk factors in this group.

Importantly, the graded increase in HOMA-IR demonstrated through post-hoc analysis (HBV vs controls: p=0.012; HCV vs controls and HBV: p<0.001) strengthens the causal inference of viral hepatitis contributing to insulin resistance. This stepwise rise highlights the differential pathogenic mechanisms of HBV and HCV, with HCV exerting a more profound systemic metabolic effect [26]. From a clinical perspective, these findings have significant implications, as insulin resistance is known to accelerate hepatic fibrosis, reduce antiviral treatment response, and increase the risk of type 2 diabetes mellitus and cardiovascular disease in patients with chronic hepatitis [27,28].

Limitations

This study has certain limitations. Being a cross-sectional design, causal relationships between viral hepatitis and insulin resistance cannot be established. The sample size was modest and derived from a single tertiary care center, limiting generalizability. Potential confounders such as dietary habits, physical activity, and socioeconomic status were not assessed. Additionally, advanced markers of insulin resistance and hepatic fibrosis were not evaluated.

Conclusion

The present study demonstrates a significant association between chronic viral hepatitis and insulin resistance, with a higher burden observed in HCV compared to HBV infection. Patients with HCV exhibited markedly elevated HOMA-IR values and a greater prevalence of insulin resistance, highlighting its strong metabolic impact. HBV infection also showed a moderate but significant association. Additionally, age, BMI, and glycemic status emerged as independent predictors of insulin resistance. These findings emphasize the interplay between viral and metabolic factors in chronic liver disease. Routine screening for insulin resistance in patients with hepatitis B and C may facilitate early identification of metabolic risk and improve overall disease management and long-term outcomes.

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