Wadi Alshatti University Journal of Pure and Applied Sciences

مجلة جامعة وادي الشاطئ للعلوم البحتة والتطبيقية

Volume 4, No. 1, January-June 2026 Online ISSN: 3006-0877 المجلد 4، الإصدار 1، يناير- يونيو 2026

RESEARCH ARTICLE **CLINICAL CHEMISTRY**

The Relationship Between Hepatitis C Virus (HCV) Infection and Insulin Resistance (IR)

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ARTICLE HISTORY
Received 24 November 2025
Revised 12 March 2026
Accepted 21 March 2026
Online 26 March 2026

KEYWORDS
Hepatitis C Virus (HCV);
Insulin Resistance;
HOMA-IR;
Type 2 Diabetes Mellitus.

ABSTRACT

Background: There is a well-established correlation between Hepatitis C Virus (HCV) infection and the development of diabetes mellitus, with increased prevalence observed among diabetic patients.

Objective: This cross-sectional study was conducted on patients attending the Medical Reference Laboratory in Sebha-Libya.This study aimed to investigate the relationship between Hepatitis C Virus (HCV) infection and insulin resistance (IR), as one of the major metabolic complications associated with chronic infection.

Method: A total of 115 participants were enrolled and divided into two groups: 90 patients with confirmed HCV infection and 25 healthy controls. Clinical and laboratory assessments included fasting blood glucose, fasting insulin, glycated hemoglobin (HbA1c), liver function tests, body mass index (BMI), and calculation of HOMA-IR as an index for insulin resistance.

Results: The results showed significantly higher levels of fasting blood glucose, insulin, HbA1c, and HOMA-IR in patients group compared with controls(P-value<0.05), indicating a strong association between HCV infection and insulin resistance. Notably, 52.2% of HCV patients were found to have insulin resistance (HOMA-IR > 2.4), while no significant differences were observed with respect to age or gender. Moreover, alterations in liver function parameters were observed, reflecting the interplay between viral infection and metabolic dysregulation.

Conclusion: HCV infection plays a pivotal role in promoting metabolic disturbances, particularly insulin resistance, which may increase the risk of type 2 diabetes mellitus and accelerate liver-related complications.

العلاقة بين عدوى فيروس التهاب الكبد الوبائي C (HCV) ومقاومة الأنسولين (IR)

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الكلمات المفتاحية

فيروس الكبد الوبائي C
مقاومة الإنسولين
السكر التراكمي
داء السكري النوع الثاني

الملخص

هناك ارتباط راسخ بين عدوي فيروس التهاب الكبد الوبائي C وتطور مرض السكري, مع ملاحظة زيادة في معدل الانتشار بين مرضى السكري.

الهداف من الدراسة : تهدف هذه الدراسة إلى دراسة العلاقة بين الإصابة بفيروس التهاب الكبد الوبائي سي HCV ومقاومة. الأنسولين IR، كأحد المضاعفات الأيضية الرئيسية المرتبطة بالعدوى المزمنة.

المواد والطرق:أجريت هذه الدراسة المقطعية علي المرض المترددين علي المختبر الطبي المرجعي في مدينة سبها- ليبيا و تم تسجيل ما مجموعه 115 مشاركًا وتقسيمهم إلى مجموعتين: 90 مريضًا مصابًا بعدوى مؤكدة بفيروس التهاب الكبد الوبائي سي و25 من الأصحاء. تضمنت التقييمات السريرية والمخبرية نسبة الجلوكوز في الدم أثناء الصيام، والأنسولين أثناء الصيام، والهيموغلوبين السكري HBA1C، واختبارات وظائف الكبد ، ومؤشر كتلة الجسم BMI، وحساب HOMA-IR كمؤشر لمقاومة الأنسولين

النتائج :أظهرت النتائج مستويات أعلى بكثير من جلوكوز الدم الصائم، والأنسولين، والهيموغلوبين السكري، ومقاومة الأنسولين HOMA-IR في مجموعة التهاب الكبد الوبائي سي مقارنةً بالمجموعة الضابطة(P<0.05)، مما يشير إلى وجود ارتباط قوي بين عدوى التهاب الكبد الوبائي سي ومقاومة الأنسولين. والجدير بالذكر أن 52.2% من مرضى التهاب الكبد الوبائي سي يعانون من مقاومة الأنسولين (HOMA-IR > 2.4)، بينما لم تُلاحظ أي فروق ذات دلالة إحصائية فيما يتعلق بالعمر أو الجنس. علاوة على ذلك، لوحظت تغيرات في معايير وظائف الكبد، مما يعكس التفاعل بين العدوى الفيروسية واضطراب التمثيل الغذائي.

الخلاصة: في الختام، تلعب عدوى التهاب الكبد الوبائي سي دورًا محوريًا في تعزيز الاضطرابات الأيضية، وخاصة مقاومة الأنسولين، مما قد يزيد من خطر الإصابة بداء السكري من النوع الثاني ويُسرّع من المضاعفات المتعلقة بالكبد.

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https://doi.org/10.63318/waujpasv4i1_32

Introduction

Hepatitis C Virus (HCV) remains a major global public health concern due to its widespread prevalence and significant clinical implications. With an estimated global infection rate of approximately 2.5%, HCV continues to affect more than 71 million individuals worldwide [1]. The virus was initially identified in 1989 by Choo and Kuo, and was soon recognized as the causative agent of non-A, non-B post-transfusion hepatitis [2]. Hepatitis C Virus (HCV) infection often begins without noticeable symptoms. Research indicates that approximately 85–90% of infected individuals remain asymptomatic, while only 10–15% experience mild and non-specific symptoms such as fatigue, muscle aches, and drowsiness. In some cases, these symptoms may be accompanied by jaundice [3]. Primarily targeting hepatocytes, HCV induces chronic liver inflammation and fibrosis, ultimately leading to cirrhosis and hepatocellular carcinoma. Beyond hepatic involvement, HCV has been linked to several extrahepatic manifestations, including metabolic syndromes, cardiovascular complications, and endocrine disorders [4]. HCV disrupts numerous intracellular processes, particularly those related to metabolic functions, including glucose metabolism, lipid regulation, and oxidative stress [5]. In recent years, increasingevidence has suggested a strong association between chronic HCV infection and the development of insulin resistance and type 2 diabetes mellitus (T2DM) [6]. Diabetes is a leading cause of death and illness worldwide [7]. Diabetes mellitus is a common non-communicable health problem that affects people all over the world. Many medical problems arise from long-term diabetes mellitus caused by insulin resistance [8] Diabetes mellitus (DM) is a metabolic disorder characterized

by chronic hyperglycaemia due to a defect in insulin production or action [9]. There is a well-established correlation between Hepatitis C Virus (HCV) infection and the development of diabetes mellitus, with increased prevalence observed among diabetic patients [10]. As a key hormone produced by pancreatic β-cells, insulin regulates glucose uptake primarily in skeletal muscle and adipose tissues. Insulin resistance impairs this regulatory pathway, resulting in chronic hyperglycemia and metabolic imbalance. Furthermore, insulin plays a critical immunomodulatory role, linking metabolic disruption with systemic inflammation [11]. Insulin resistance IR is defined by the reduced cellular responsiveness to insulin signals [12]. A decrease in sensitivity, activity, or reactivity to the metabolic effects of insulin is commonly used to describe IR.[13] Insulin resistance (IR) is a metabolic condition wherein higher-than-normal levels of insulin are required to elicit a standard metabolic response, or where normal insulin levels fail to promote adequate glucose uptake and utilization[14]. In muscular tissue, this leads to impaired glucose uptake, while excess glucose is redirected to the liver, enhancing lipid and glycogen synthesis. In hepatic insulin resistance, insulin fails to suppress gluconeogenesis, thereby exacerbating glucose accumulation and promoting steatosis [15]**.**

Recent research further emphasizes a strong correlation between HCV infection and metabolic disturbances, notably insulin resistance and type 2 diabetes mellitus (T2DM). This connection is primarily due to the virus's disruption of insulin signalling pathways, including the degradation of essential insulin adaptor proteins such as IRS-1 and IRS-2. Consequently, cellular insulin responses are weakened, contributing to the development of insulin resistance [16].

This study aims to achieve a set of scientific objectives designed to deepen the understanding of the link between viral infection and related metabolic disturbances. the relationship between (HCV) infection and insulin resistance. Assess gender-based differences(male vs female)in the degree of insulin resistance associated with HCV infection.

Materials and methods

This study was conducted on patients attending the Medical Reference Laboratory in Sebha. A total of 115 subjects were included and divided into two groups.

Patient Group

This group included 90 patients confirmed to be infected with HCV. Initial diagnosis was performed using an enzyme-linked immunosorbent assay (ELISA) to detect anti-HCV antibodies, utilizing a reagent from Biorex company and the Cobas e411 analyser. Positive results were further confirmed by polymerase chain reaction (PCR) testing using the GeneXpert system.

Control Group

This group included 25 healthy subjects with no history of HCV infection, chronic diseases, diabetes, or obesity. They underwent the same laboratory tests.

Blood samples were collected from each patient using three types of blood collection tubes, depending on the type of analysis required

EDTA Tube: a total volume of 2 ml of venous blood was collected from each participant. This tube was used for the analysis of glycated hemoglobin (HbA1c) and for the polymerase chain reaction (PCR) test to detect HCV RNA, using the GeneXpert system after confirmation of HCV infection. Following the HbA1c analysis, the samples were centrifuged at 4000 rpm and the plasma was frozen for one week, after which PCR testing was performed.

Sodium Fluoride Tube: a total volume of 2 ml of venous blood was collected from each participant. This tube, containing sodium fluoride as an anticoagulant and glycolysis inhibitor, was used for the analysis of fasting blood glucose.

Clot Activator Tube: a total volume of 2 ml of venous blood was collected from each participant. This plain tube without anticoagulant was used to conduct a panel of biochemical tests, including Fasting insulin, Liver function tests (LFTs), and ELISA test for anti-HCV.

Additionally, the weight and height of all participants were measured to calculate the Body Mass Index (BMI) using the formula:

BMI = weight (kg) / height² (m²) [17] .

The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was also calculated using the formula:

HOMA-IR = (Fasting insulin (μIU/mL) × Fasting glucose (mg/dL)) / 405[17].

Statistical Analysis

The statistical analysis in this study was conducted using the Statistical Package for the Social Sciences (SPSS) software20. Data were analysed to compare the two study groups (patients and healthy controls).

Descriptive Statistics:

Mean and standard deviation were calculated for continuous variables such as age, weight, height, BMI, fasting blood glucose (FBS), insulin, HbA1c, HOMA-IR, liver function tests,and used to evaluate the frequency and proportion of participants with insluin resistance versus those without. Insulin resistance was considered when the HOMA-IR value exceeded 2.4, as reported by [18].

Independent Sample t-tests:

Used to compare means between the HCV patient group and the healthy control group in various parameters including anthropometric measurements, liver function tests.age, weight, height, BMI, fasting blood glucose (FBS), insulin, HbA1c, HOMA-IR, A p-value of less than 0.05 was considered statistically significant throughout the study

Results

The results showed a statistically significant difference in the mean weightbetween the patient group (73.47 kg) and the control group (67.96 kg) (P-value <0.05),. There is a slight difference in the mean height between the patient group (169.74 cm) and the control group (167.84 cm). However, this difference was not statistically significant. The BMI is higher in the patient group (25.73) compared to the control group (24.11). Though, there was no statistically significant difference between the groups (Table 1).

Table 1: Comparison between Patients and control group in Age, Weight, Height and BMI

	PATIENT	CONTROL	
	Mean±SD	Mean±SD	P value
Age	41.3±10.55	39.16±7.55	0.329
Weight	73.4±9.41	67.96±5.51	0.006
Height	169.7±10.47	167.8±5.80	0.238
BMI	25.73±5.000	24.11±1.100	0.112

The average fasting blood sugar (FBS) level was significantly elevated in patients compared to the control group (P<0.001). Similarly, insulin concentrations were notably higher in patients (11.54 µU/ml) than in controls (4.68 µU/ml), with a statistically significant difference (P<0.001). In addition, the mean HbA1c level was greater in the patient group (5.68%) than in the control group (5.38%) (P<0.001). The HOMA-IR index was also significantly increased in patients (2.88) compared to controls (1.08), reflecting higher insulin resistance among patients and indicating a strong association between HCV infection and insulin resistance (Table 2).

Table 2: Comparison between Patients and control in FBS, Insulin, Hba1c and HOMA IR

	PATIENT	CONTROL	
	Mean±SD	**Mean±SD**	**P value**
FBS mg/dl	100.93±11.99	93.64±7.31	0.000
Insulin µu/ml	11.5±6.38	4.68±1.09	0.000
HOMA-IR	2.88±1.67	1.08±0.269	0.000
HBA1C%	5.68±0.386	5.38±0.286	0.000

There was no statistically significant difference in the mean total bilirubin level between the patient group(0.40mg/dl) and the control group(0.37mg/dl) (P>0.05). However, there was a statistically significant increase in the mean ALP level in thepatient group(93.5u/l) compared with the control group(82.6u/l) (P<0.05).

The mean GOT level was significantly higher in the patient group(18u/l) than in the control group(13.6u/l) (P<0.05). Similarly, the mean GPT level showed a statistically significant elevation in the patient group(18.6u/l) compared to the control group (14.5u/l) (P<0.05).(Table 3).

The results indicate that 52.2% of the participant's exhibit insulin resistance (HOMA-IR > 2.4), while 47.8% do not exhibit insulin resistance (HOMA-IR < 2.5) (Figure 1).

Discussion

The results of this study indicate that the mean age of patients with hepatitis C virus (HCV) infection (41.31 ± 10.55 years) was slightly higher than that of the control group (39.16 ± 7.55 years), without statistical significance (P=0.252). The mean weight was significantly higher in patients (73.47 ± 9.41 kg) compared to controls (67.96 ± 5.51 kg) with clear statistical significance (P=0.006). However, height (169.74 ± 10.47 cm vs. 167.84 ± 5.80 cm, P=0.238) and body mass index (BMI) (25.73 ± 5.00 vs. 24.11 ± 1.10, P=0.112) did not show statistically significant differences. When comparing these results with the study by Ezzat et al, [10] the mean age in the patient sample was higher (47 ± 12 years), which may reflect the inclusion of more advanced age cases in that study. The mean BMI was 25.6 ± 1.5 kg/m², close to the results of this study, supporting the hypothesis that BMI in HCV patients tends to remain within the overweight range rather than obesity, especially when excluding patients with BMI ⩾ 30 kg/m². Height was not directly reported in Ezzat et al.'s study, but the mean waist circumference (84.6 ± 6.3 cm) indicated a relative increase in central body mass[10].

Overall, weight and BMI in HCV patients are generally higher than in control group or reference values, but these increases do not reach obesity levels. This pattern may suggest a role of overweight status and visceral fat in influencing insulin resistance in HCV patients, warranting further longitudinal studies to establish causality, especially considering the demographic differences between research samples in various studies.

In the present study, fasting blood sugar (FBS) was significantly higher in HCV patients (100.93 ± 11.99 mg/dL) compared to controls (93.64 ± 7.31 mg/dL, P=0.008). Serum insulin levels were markedly elevated in the HCV group (11.54 ± 6.38 µU/mL) versus controls (4.68 ± 1.09 µU/mL, P<0.001), and HOMA-IR values were significantly higher (2.88 ± 1.67 vs. 1.08 ± 0.269, P<0.001), indicating a strong association between HCV infection and insulin resistance.These findings are consistent withstudy of Mishra et al. [19] who reported significantly higher fasting insulin (4.91 ± 2.29 µU/mL vs. 3.87 ± 2.07 µU/mL, P=0.02) and insulin resistance (HOMA-IR: 5.91 ± 1.21 vs. 3.55 ± 1.17, P=0.001) in HCV-positive patients, alongside elevated

Table 3: Comparison between patients group and control group in LFT

	PATIENT	CONTROL	
	Mean±SD	Mean±SD	P value
T.BLI mg/dl	0.40±0.209	0.37±0.102	0.335
ALP U/L	93.5±23.09	82.6±10.62	0.001
GOT U/L	18.0±9.38	13.6±6.22	0.008
GPT U/L	18.6±10.99	14.5±5.16	0.009

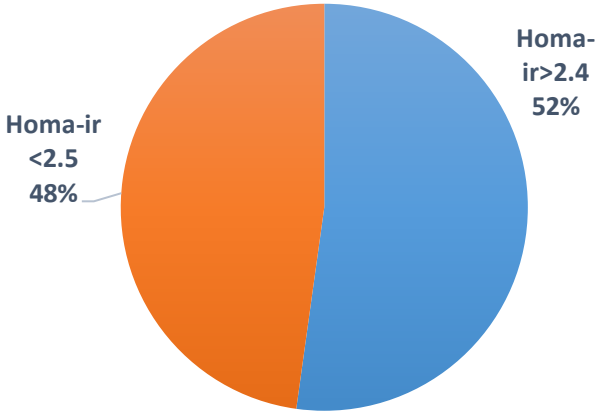

Figure 1: Distribution of Insulin Resistance among study participants.

ferritin levels [14]Chen *et al*. [20] demonstrated that glucose tolerance status in HCV patients correlates strongly with IR severity, with higher HOMA-IR values observed in impaired fasting glucose (IFG), impaired glucose tolerance (IGT), and diabetic subgroups compared to normal glucose tolerance. This supports the finding in the present study that higher fasting glucose parallels increased HOMA-IR in HCV infection[20].

Overall, across all studies, the evidence consistently supports that HCV infection is associated with elevated fasting insulin and increased insulin resistance, often independent of overt hyperglycemia. The present study's observation of significantly higher FBS, insulin, and HOMA-IR in HCV patients aligns with most literature, though a study by Mohamed et al. [21] did not observe significant FBS differences, highlighting potential population and methodological variability. The convergence of results underscores the role of HCV in disrupting glucose metabolism through mechanisms likely involving both hepatic insulin extraction impairment and systemic inflammatory responses.

In the current study, liver function tests (AST, ALT,) showed clear alterations in HCV-positive patients compared to controls. The AST level in the HCV group was 18.03 ± 9.38 U/L versus 13.68 ± 6.22 U/L in the control group (P= 0.030). ALT (GPT) was similarly elevated at 18.68 ± 10.99 U/Lcompared to 14.52 ± 5.16 U/L (P= 0.021). For bilirubin, total bilirubin was 0.40 ± 0.209 mg/dL versus 0.37 ± 0.102 mg/dL (P= 0.335).These results are in strong agreement withthe finding of a study conducted byMohamed et al. [21], who reported AST at 42.45 ± 29.75 IU/L in HCV patients versus 23.00 ± 12.03 IU/L in controls (P< 0.05), and ALT at 43.77 ± 37.79 IU/L versus 20.24 ± 11.24 IU/L (P< 0.05). They also found higher total bilirubin (0.82 ± 0.42 mg/dL vs. 0.65 ± 0.36 mg/dL, P<0.05) and direct bilirubin (0.20 ± 0.14 mg/dL vs. 0.13 ± 0.13 mg/dL, P< 0.05),This discrepancy in bilirubin may reflect differences in disease stage or the proportion of patients with advanced fibrosis[21].

Similarly, Mishraet al. [19] demonstrated significantly increased ALT in HCV patients (54.2 ± 21.8 IU/L) compared to non-HCV controls (28.4 ± 14.5 IU/L, p < 0.05), although their total bilirubin values (0.90 ± 0.25 mg/dL vs. 0.85 ± 0.20 mg/dL, P< 0.05) did not show a significant difference.which parallels the present study's bilirubin findings[19].

Overall, across the present and previous studies, AST and ALT elevations are consistently observed in HCV infection, confirming their role as markers of hepatocellular injury. The variability in bilirubin outcomes between studies may be due to patient selection criteria, differences in disease severity, and the presence of comorbid conditions like insulin resistance or obesity.

The current study found that 52.2% of participants exhibited insulin resistance (HOMA-IR > 2.4), whereas 47.8% did not. This prevalence is within the range reported by multiple international studies, confirming that insulin resistance is a common metabolic complication of chronic hepatitis C virus (HCV) infection. Similarly, Kiranet al. [22] conducted a study in Pakistan on non-diabetic chronic HCV patients and found that 53% of the cohort had insulin resistance (HOMA-IR ≥ 2.5). This result closely matches the present study's finding of 52.2%, suggesting that the high prevalence is consistent across different populations and is not limited to those with comorbid diabetes [22].

Villar et al. [23] reported an even higher prevalence of 62% insulin resistance among chronic HCV patients in Brazil. They also observed that insulin resistance was significantly associated with more severe liver histopathological changes, reinforcing the link between metabolic and hepatic disease progression [23].

Conclusion

The prevalence rate of insulin resistance observed in the present study (52.2%) is consistent with rates reported in previous literature, ranging from 48% to over 60%. The consistency of these findings across different geographic regions and patient populations underscores the global relevance of insulin resistance in chronic HCV infection. Clinically, this highlights the importance of early metabolic screening and intervention strategies—such as lifestyle modifications, insulin-sensitizing therapies, and integrated hepatology–endocrinology management—to improve both hepatic and metabolic outcomes in this patient group.

Recommendations

These findings highlight the need to integrate metabolic evaluation into the clinical management of HCV patients, as well as the importance of post-treatment follow-up to assess improvements in insulin sensitivity after sustained virological response.

Author Contributions:"Albannai: Conceptualized the study, collected the data, performed the laboratory tests and data analysis, and wrote the manuscript draft. Eshnaf: edited and reviewed the manuscript.Both authors have read and agreed to the published version of the manuscript."

Funding: "This research received no external funding."

Data Availability Statement: "The datasets are not publicly available due to confidentiality and ethical restrictions. Data relating to test results are available fromthe corresponding author on reasonable request."

Acknowledgments: "The authors would like to express theirappreciation to all participants in this study.The authors wish to thank the administration and staff of the Medical Reference Laboratory, Sebha, Libya for their kind help and cooperation.

Conflicts of Interest: "The authors declare no conflict of interest."

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