

Study of Insulin Resistance in Hepatitis C Positive Patients**Dipendu Saha¹, Ajit Kumar Pegu², Rima Moni Doley³, Aparna Sonowal⁴, Lipee Nath⁵**¹Post Graduate Trainee, Department of Medicine, Assam Medical College, Assam, India^{2,3}Professor, Department of Medicine, Assam Medical College, Assam, India⁴Associate Professor, Department of Microbiology, Assam Medical College, Assam, India⁵Professor, Department of Radiology, Assam Medical College, Assam, India

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Abstract

Background & Objective: Hepatitis C virus (HCV) infection is a major cause of chronic liver disease and is increasingly recognized for its metabolic effects, particularly insulin resistance (IR). IR contributes to hepatic steatosis, accelerated fibrosis, and poorer treatment outcomes. The Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) provides a simple method to assess insulin resistance using fasting glucose and insulin levels. The study aims to evaluate insulin resistance using HOMA-IR among non-diabetic HCV-positive patients and to correlate IR with disease severity.

Materials and Methods: This hospital-based observational study was conducted in the Department of Medicine at Assam Medical College and Hospital, Dibrugarh. A total of 85 HCV-positive patients attending the outpatient department were included. Fasting blood samples were collected to estimate plasma glucose and serum insulin levels. Insulin resistance was calculated using the HOMA-IR formula. Clinical, biochemical, and radiological parameters, including liver function tests and FibroScan findings, were analyzed to assess disease severity and its correlation with insulin resistance.

Results: A significant proportion of HCV-infected patients demonstrated insulin resistance. Higher HOMA-IR values were observed in patients with advanced liver disease, increased fibrosis stages, and hepatic steatosis. Statistical analysis showed a positive correlation between insulin resistance and disease severity indicators, suggesting that metabolic dysfunction plays a key role in the progression of HCV-related liver pathology.

Conclusion: Insulin resistance is a common and clinically significant metabolic abnormality in patients with chronic hepatitis C infection. HOMA-IR serves as a practical and effective tool for its assessment. Early identification of insulin resistance may aid in risk stratification, guide therapeutic decisions, and improve overall disease outcomes.

Keywords: Hepatitis C Virus, Insulin Resistance, HOMA-IR, Chronic Hepatitis C, Liver Fibrosis, Hepatic Steatosis.

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Introduction

Hepatitis C virus (HCV) infection continues to pose a worldwide public health problem because it affects approximately 71 million people who represent about 1 percent of the global population. After acute exposure to the virus about 70 percent to 85 percent of infected individuals develop chronic infection which leads to extended periods of liver inflammation and fibrosis that ultimately results in liver failure.[1]

Chronic HCV infection shows different biochemical patterns in patients because some maintain normal alanine aminotransferase (ALT) levels even though they experience histological damage, which highlights the necessity of metabolic and molecular markers for complete

disease evaluation.[2] HCV creates insulin resistance through both direct and indirect molecular mechanisms. The viral core protein disrupts the insulin-signalling pathway by inducing serine phosphorylation of insulin receptor substrate-1 (IRS-1) at Ser312, leading to its ubiquitination and degradation.

HCV also suppresses the TSC1/TSC2 complex, resulting in overactivation of the mTOR/S6K1 pathway and further phosphorylation of IRS-1 at Ser1101, thereby aggravating post-receptor insulin-signalling defects. In addition, HCV upregulates proteins that inhibit IRS-1 function and suppress phosphoinositide-3-kinase (PI3K)/Akt signalling pathways. These alterations impair glycogen

synthesis and glucose uptake while enhancing gluconeogenesis. [3] Through increased hepatic glucose-6-phosphatase and phosphoenolpyruvate carboxykinase activity. Simultaneously, reduced expression of GLUT-2 and GLUT-4 decreases hepatic and peripheral glucose uptake. Hyperinsulinaemia further promotes oxidative stress, inflammation, and lipotoxicity, contributing to progressive liver injury. Thus, insulin resistance (IR) represents a central pathogenic mechanism in chronic HCV infection. HCV-related IR is strongly associated with hepatic steatosis, increased necroinflammation, accelerated fibrosis progression, and reduced sustained virologic response (SVR) to antiviral therapy. Therefore, early identification and management of IR remain essential components of HCV care.[4]

Assessment of IR in HCV-positive patients has important clinical implications, including early identification of individuals at high risk for type 2 diabetes mellitus (T2DM), a common extrahepatic manifestation of chronic HCV infection.

Early detection and management of IR through lifestyle modification and insulin-sensitizing agents may improve antiviral response and liver outcomes. Persistent IR also contributes to ongoing hepatic inflammation and steatosis even after viral clearance. These strategies support the goals of the National Viral Hepatitis Control Program and the National Viral Hepatitis Action Plan (NVHAP) in reducing HCV-related morbidity and mortality.[5]

The present study aims to evaluate insulin resistance using HOMA-IR among non-diabetic HCV-positive patients and to correlate IR with disease severity.

Materials and Methods

The hospital based cross-sectional study was conducted at the Department of General Medicine which is located at Assam Medical College and Hospital Dibrugarh, for period of one year (October 2024- September 2025).

A convenience based non-probability sampling method was employed and a total of 85 participants fulfilling the inclusion criteria were selected for the study. Ethical clearance was obtained from the Institutional Ethics Committee before starting the study.

Inclusion Criteria: Participants aged 13 years and above, with chronic hepatitis C infection, and non-diabetic, willing to provide written informed consent.

Exclusion Criteria: Patients with autoimmune hepatitis, alcoholic hepatitis and other causes of

hepatitis, Pregnant and lactating mothers and those who were unwilling to give informed consent.

The researchers conducted patient interviews through a pre-tested proforma which they used for data collection. The researchers documented all demographic information together with the complete clinical history and HCV transmission risk factors of the subjects.

Blood samples were collected by aseptic puncture following overnight fasting for 8 hours. Approximately 5 mL of venous blood was drawn—2 mL into a fluoride vial for glucose estimation and 3 mL into a plain vial for insulin and biochemical assays. Samples were centrifuged at 3000 rpm for 10 minutes, and serum was separated immediately.

Fasting plasma glucose was measured using the hexokinase enzymatic method on an automated analyzer. Fasting insulin was measured by a chemiluminescent immunoassay using standardized commercial kits calibrated against WHO reference standards. Liver function tests and lipid profiles were performed simultaneously.

For virological confirmation, anti-HCV antibody was detected by third-generation ELISA, and quantitative HCV RNA was determined by real-time PCR. Elastography (FibroScan) was conducted in the Department of Radiology using a 3.5 MHz probe to assess liver stiffness.

The main purpose of FibroScan® is to determine the stage of liver disease and the extent of liver scarring. Patients with chronic liver illness, such as fatty liver, chronic alcoholism, chronic hepatitis C, and chronic hepatitis B, can be benefitted greatly from this.

Each HOMA-IR value was calculated and recorded in the case proforma. Patients with abnormal biochemical findings were referred for appropriate medical management within the hospital.

Data was collected using a pre-designed structured proforma and entered into Microsoft Excel. Statistical analysis was conducted using the SPSS 25.0 version. Independent-sample t-test or Mann–Whitney U-test was used to compare continuous variables between normal and insulin-resistant groups.

Chi-square test or Fisher's exact test is used to compare categorical data. Pearson or Spearman correlation coefficients were used to find associations with HOMA-IR and liver function parameters or viral load. Statistical significance was set at $p < 0.05$.

Result

Table 1: Demographic Characteristics of Study Participants (n = 85)

Variable	Category	Number (n)	Percentage (%)
Age Group (years)	13–20 years	15	18
	21–30 years	47	55
	31–40 years	17	20
	41–50 years	5	6
	>50 years	1	1
Gender	Male	76	89
	Female	9	11
Grand Total		85	100

The majority of study participants belonged to the 21–30 years age group (55%), followed by 31–40 years (20%). Male participants predominated the study population, accounting for 89% of cases, while females constituted 11%.

Table 2: Mode of Transmission of Hepatitis C Infection (n = 85)

Mode of transfusion	Number (n)	Percentage (%)
IV drug abuse	78	92
Blood transfusion	3	4
Unidentified	2	2
Tattooing	1	1
Haemodialysis	1	1
Grand Total	85	100

Intravenous drug abuse was reported in 78 patients (92%). Blood transfusion accounted for 3 cases (4%). Tattooing, hemodialysis, and unidentified causes were each observed in 1–2% of cases, indicating IV drug abuse as the predominant transmission route.

Table 3: FibroScan Findings and Fibrosis Staging Among Study Participants (n = 85)

FibroScan Category	Corresponding Fibrosis Stage	Number (n)	Percentage (%)
Normal	F0	30	35
Dysfunction	F1	19	22
Fibrosis	F2–F3	31	35
Cirrhosis	F4	5	6
Grand Total		85	100

FibroScan assessment demonstrated that fibrosis (F2–F3) and normal liver status (F0) were equally prevalent, each accounting for 35% of the study population. Mild fibrosis/dysfunction (F1) was observed in 22% of participants, while cirrhosis (F4) was present in 6% of cases.

Table 4: Liver Function Test (LFT) Profile among Study Participants (n = 85)

Overall LFT Status	LFT Category	Number (n)	Percentage (%)
Normal	Normal LFT	42	49
Deranged	Mild elevation ($\leq 2 \times$ ULN)	2	2
	Moderate elevation ($2-5 \times$ ULN)	2	2
	Severe elevation ($> 5 \times$ ULN)	3	4
	Other deranged LFT findings	36	43
Grand Total		85	100

Normal liver function tests were observed in 42 participants (49%), while deranged LFT findings were present in 43 participants (51%). Among abnormal cases, severe elevation of liver enzymes

was noted in 4% of participants, whereas mild and moderate elevations were observed in 2% each. Overall, abnormal liver function was slightly more prevalent in the study population.

Table 5: Descriptive Statistics of Metabolic and Glycaemic Parameters among Study Participants (n = 85)

Parameter	Mean	Standard Deviation	Minimum	Maximum	Number
HOMA-IR Score	1.72	2.49	0.2	12.5	85
BMI (kg/m ²)	24.53	3.31	18.5	29.9	85
Fasting Blood Sugar (mg/dL)	113.55	16.19	79	152	85
Fasting Insulin (μ IU/mL)	5.61	7.65	0.78	38.49	85
HbA1c (%)	5.56	0.49	4.8	6.4	85

The mean HOMA-IR score was 1.72 with a standard deviation of 2.49, ranging from 0.2 to 12.5. Mean BMI was 24.53 ± 3.31 , with values between 18.5 and 29.9. Mean fasting blood sugar was 113.55 ± 16.19 mg/dL, with values ranging from 79 to 152 mg/dL. Mean fasting insulin level was 5.61 ± 7.65 μ IU/mL, ranging from 0.78 to 38.49 μ IU/mL. Mean HbA1c was $5.56 \pm 0.49\%$, with values between 4.8 and 6.4 in the study population. Mean HbA1c was 5.24 ± 0.25 , ranging from 4.8 to 5.6 among the 85 participants.

Table 6: Distribution of Disease Severity and HOMA-IR Categories among Study Participants (n = 85)

Category	Subcategory	Number (n)	Percentage (%)
Disease Severity	Mild	67	79
	Moderate	12	14
	Severe	6	7
HOMA-IR Category	Mild	67	79
	Moderate	12	14
	Severe	6	7
Grand Total		85	100

Most study participants were classified under the mild category for both disease severity and HOMA-IR distribution (79%). Moderate and severe categories accounted for 14% and 7% of participants, respectively, indicating that the majority of hepatitis C–positive patients had lower grades of disease severity and insulin resistance.

Table 7: Insulin Resistance Status Based on HOMA-IR Score among Study Participants (n = 85)

Resistance Status Insulin	Number (n)	Percentage (%)
Normal	61	72
Significant IR	17	20
Early IR	7	8
Grand Total	85	100

Out of 85 HCV-positive patients, insulin resistance (IR) was identified in 24 patients (28%), including 17 patients (20%) with significant IR and 7 patients (8%) with early IR based on HOMA-IR scoring.

Normal insulin sensitivity was observed in 61 patients (72%). Insulin resistance showed a significant association with disease severity and FibroScan findings ($p < 0.001$).

Table 8: Hepatic Steatosis Status among Study Participants (n = 85)

Hepatic Steatosis	Number (n)	Percentage (%)
Present	47	55
Absent	38	45
Grand Total	85	100

Hepatic steatosis was present in 47 patients (55%) and absent in 38 patients (45%) across the study cohort.

Table 9: Clinical Presentation of Hepatitis C–Positive Patients (n = 85)

Clinical Presentation	Number (n)	Percentage (%)
Asymptomatic	11	13
Fatigue	11	13
Right upper quadrant pain	11	13
Jaundice	11	13
Loss of appetite	11	13
Nausea	10	12
Generalized weakness	10	12
Abdominal discomfort	10	12
Grand Total	85	100

Asymptomatic presentation was noted in 11 patients (13%). Fatigue, right upper quadrant pain, jaundice, and loss of appetite were each reported by 11 patients (13%). Nausea, generalized weakness, and abdominal discomfort were observed in 10 patients each (12%), indicating a varied but evenly distributed symptom profile.

Table 10: Association between FibroScan Category and Insulin Resistance Status (n = 85)

FibroScan Category	Normal IR	Early IR	Significant IR	Total	χ^2 (df)	p-value
Normal	13	0	17	30	98.679	< 0.001*
Fibrosis	31	0	0	31		
Cirrhosis	0	5	0	5		
Dysfunction	17	2	0	19		
Total	61	7	17	85		

A statistically significant association was observed between FibroScan category and insulin resistance status ($\chi^2 = 98.679$, $df = 6$, $p < 0.001$). Normal FibroScan findings were associated with 17 cases

of significant insulin resistance, while fibrosis was predominantly associated with normal insulin sensitivity. Cirrhosis showed clustering with early insulin resistance.

Table 11: One-Way ANOVA Comparing Mean HOMA-IR Scores Across Disease Severity Groups.

Source of Variation	Sum of Squares	Mean Square	F value	p-value
Between Groups	440.116	220.058	218.387	< 0.001*
Within Groups	82.627	1.008		

One-way ANOVA demonstrated a statistically significant difference in mean HOMA-IR scores across disease severity groups ($F = 218.387$, $p < 0.001$). The between-group sum of squares was 440.116, while within-group variation was 82.627, indicating significant variability in HOMA-IR across severity categories.

Table 12: One-Way ANOVA Comparing Mean HOMA-IR Scores across Fibrosis Stages

Source of Variation	Sum of Squares	Mean Square	F value	p-value
Between Groups	20.615	6.872	1.108	0.351
Within Groups	502.129	6.199		

No statistically significant difference in mean HOMA-IR scores was observed across fibrosis stages ($F = 1.108$, $p = 0.351$). The between-group sum of squares was 20.615, while within-group variation was 502.129. Post-hoc Tukey HSD analysis did not demonstrate significant pairwise differences.

Table 13: One Correlation between BMI, HOMA-IR Score, and FibroScan Values

Variables	BMI	HOMA-IR Score	FibroScan
BMI	1	0.044 ($p = 0.690$)	-0.019 ($p = 0.860$)
HOMA-IR Score	0.044 ($p = 0.690$)	1	0.406 ($p < 0.001$)**
FibroScan	-0.019 ($p = 0.860$)	0.406 ($p < 0.001$)**	1

Pearson correlation analysis showed a significant positive correlation between HOMA-IR score and FibroScan value ($r = 0.406$, $p < 0.001$). No significant correlation was observed between BMI and HOMA-IR score ($r = 0.044$, $p = 0.690$) or between BMI and FibroScan value ($r = -0.019$, $p = 0.860$).

Discussion

This paper used the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) measurement to assess insulin resistance in chronic hepatitis C virus patients and its association with demographic data, viral features, liver hemodynamic variables, imaging and fibrosis variables, glycaemic level, and disease severity. The present study demonstrated that the majority of hepatitis C virus (HCV)-positive patients belonged to the 21–30 years age group, with a marked male predominance. Insulin resistance was identified even in this relatively young population, suggesting that metabolic derangements in chronic HCV infection may occur early and are not solely age-related. Similar observations were reported by Knobler et al., who demonstrated a higher prevalence of metabolic abnormalities and diabetes

mellitus among noncirrhotic chronic HCV patients compared to controls without liver disease [6]. Ziyadi et al. further reported an increased risk of diabetes among HCV-positive individuals, particularly with increasing age and disease duration [7]. The predominance of male participants in the present study is comparable with previous epidemiological studies conducted in high-risk populations. Bhandari and Priya also observed elevated HOMA-IR levels among HCV-infected patients irrespective of gender, indicating that viral mechanisms rather than sex-specific metabolic differences primarily contribute to insulin resistance [8]. Intravenous drug abuse was the predominant mode of HCV transmission in the present study, followed by blood transfusion, tattooing, haemodialysis, and unidentified causes. Although the route of transmission itself may not directly influence insulin resistance, chronic HCV infection appears to independently contribute to metabolic dysregulation. Ziyadi et al. emphasized that conventional metabolic risk factors alone could not explain the increased diabetes risk among HCV-positive individuals, supporting a virus-mediated metabolic effect [7]. Similarly, El-Meghawry et al. reported significant insulin

resistance among HCV-infected individuals even in the absence of metabolic syndrome or diabetes mellitus [9]. These findings support the hypothesis that chronic HCV infection interferes with insulin signalling pathways through inflammatory and hepatic mechanisms irrespective of transmission history.

FibroScan assessment in the current study demonstrated that fibrosis and normal liver stiffness were equally prevalent, while cirrhosis was observed in a smaller proportion of patients. Although ANOVA did not demonstrate a statistically significant difference in mean HOMA-IR scores across fibrosis stages, a significant positive correlation between HOMA-IR and FibroScan values was observed. Sud et al. identified HOMA-IR as an independent predictor of significant fibrosis and incorporated it into a fibrosis prediction model with high diagnostic accuracy [10]. Ibrahim et al. also demonstrated progressive increases in insulin resistance with worsening hepatic fibrosis and disease severity [11]. These findings suggest that liver stiffness may better reflect metabolic impairment than categorical fibrosis staging alone.

Most patients in the present study demonstrated preserved liver synthetic function despite elevated transaminase levels and evidence of insulin resistance. Deranged liver function tests were observed in approximately half of the study population, indicating that metabolic abnormalities may coexist even with relatively preserved hepatic function. Knobler et al. reported that metabolic disturbances and diabetes mellitus may occur in noncirrhotic HCV patients without marked biochemical liver injury [6]. El-Meghawry et al. similarly demonstrated substantial insulin resistance in HCV patients independent of overt hepatic dysfunction [9]. These observations indicate that conventional liver function parameters alone may not adequately reflect metabolic risk in chronic hepatitis C infection.

The present study demonstrated near-normal glycaemic indices despite significant variability in fasting insulin and HOMA-IR values. A considerable proportion of patients exhibited insulin resistance despite maintaining relatively normal HbA1c levels, suggesting early metabolic dysfunction. Cheng et al. reported improvement in fasting glucose, HbA1c, and HOMA-IR following successful antiviral therapy, supporting the direct role of HCV in metabolic dysregulation [12]. Knobler et al. also demonstrated a higher prevalence of diabetes among chronic HCV patients, suggesting that insulin resistance may precede overt diabetes mellitus [6]. These findings highlight HOMA-IR as a sensitive marker for early metabolic abnormalities in HCV infection.

Most participants in the current study had mild disease severity and mild HOMA-IR categories; however, significant differences in HOMA-IR scores were observed across disease severity groups. Ibrahim et al. demonstrated that insulin resistance progressively increased with worsening hepatic disease severity and fibrosis burden [11]. Sud et al. similarly reported a close association between insulin resistance and disease progression in chronic HCV infection [10]. The present findings support the concept that insulin resistance is not merely an associated metabolic abnormality but may contribute to disease progression.

Abnormal insulin resistance patterns were identified in a substantial proportion of the study population, with significant insulin resistance observed in many patients despite preserved glycaemic control. Bhandari and Priya reported a high prevalence and severity of insulin resistance among HCV-infected patients, with significantly elevated HOMA-IR values compared to controls [8]. Interventional studies by Elhelbawy et al. and Khan et al. demonstrated significant improvement in insulin resistance following direct-acting antiviral therapy, supporting the role of active HCV replication in metabolic impairment [13,14]. Lamichhane et al., in a meta-analysis, further confirmed that HOMA-IR significantly decreases after viral eradication [15]. These findings establish insulin resistance as an important extrahepatic manifestation of chronic hepatitis C infection.

More than half of the patients in the present study demonstrated hepatic steatosis, suggesting a strong association between fatty liver changes and metabolic dysfunction in HCV infection. El-Meghawry et al. reported that chronic HCV infection promotes hepatic inflammation and steatosis, leading to impairment of insulin signalling independent of obesity [9]. Casas-Deza et al. further demonstrated persistent lipid abnormalities and altered hepatic lipid metabolism in HCV patients despite viral eradication [16]. The present findings support the role of hepatic steatosis as an important mediator linking chronic HCV infection and insulin resistance. The clinical presentation in the present study was variable, with a proportion of patients remaining asymptomatic. Despite mild or nonspecific symptoms, a considerable number of patients exhibited insulin resistance, indicating that metabolic abnormalities may occur silently during chronic HCV infection. Knobler et al. observed that metabolic disturbances frequently occur even in noncirrhotic HCV patients without severe clinical manifestations [6]. Ziyadi et al. similarly reported that diabetes risk among HCV-positive individuals may not correlate directly with symptom severity [7]. These findings emphasize the importance of metabolic screening even in asymptomatic HCV-positive individuals.

A statistically significant association was observed between FibroScan category and insulin resistance status in the present study. Furthermore, liver stiffness values positively correlated with HOMA-IR scores, suggesting progressive metabolic impairment with worsening hepatic stiffness. Sud et al. demonstrated that insulin resistance independently predicts significant fibrosis in chronic HCV infection [10]. Ibrahim et al. also reported increasing insulin resistance with greater fibrosis burden and disease severity [11]. These findings support combined metabolic and elastographic assessment in evaluating disease progression in chronic hepatitis C patients.

The current study demonstrated a statistically significant difference in mean HOMA-IR scores across disease severity groups, indicating progressive worsening of insulin resistance with increasing disease severity. Ibrahim et al. similarly reported greater insulin resistance among patients with advanced hepatic disease and fibrosis [11]. Sud et al. also highlighted the association between insulin resistance and hepatic disease progression [10]. These observations suggest that insulin resistance may serve as both a marker and contributor to disease progression in chronic hepatitis C infection.

No statistically significant difference in mean HOMA-IR scores was observed across fibrosis stages in the present study. However, continuous correlation analysis demonstrated a significant relationship between HOMA-IR and FibroScan values, suggesting that categorical fibrosis staging may not adequately capture metabolic variability. Similar findings have been reported by Sud et al., who emphasized the value of continuous fibrosis assessment in predicting metabolic impairment [10]. These findings indicate that liver stiffness measurements may better reflect ongoing metabolic dysfunction than fibrosis stage categories alone.

The present study demonstrated a significant positive correlation between HOMA-IR score and FibroScan values, whereas BMI showed no significant correlation with either HOMA-IR or FibroScan findings. El-Meghawry et al. similarly demonstrated insulin resistance among HCV-infected patients independent of obesity or metabolic syndrome [9]. Bhandari and Priya also reported elevated HOMA-IR values despite variable BMI measurements [8]. These findings suggest that insulin resistance in chronic HCV infection is driven predominantly by viral, inflammatory, and hepatic mechanisms rather than generalized adiposity alone.

The present study has several strengths that enhance its clinical relevance and scientific validity. A comprehensive evaluation of insulin resistance using HOMA-IR was performed in a

well-characterized cohort of hepatitis C virus-positive patients, incorporating demographic, biochemical, virological, imaging, FibroScan, and metabolic parameters. Standardized laboratory methods and non-invasive FibroScan assessment improved the reliability and objectivity of hepatic and metabolic evaluation. Statistical analyses including correlation analysis, chi-square test, and ANOVA strengthened the assessment of associations between insulin resistance and liver disease severity. The study also identified metabolic abnormalities in relatively young patients with preserved liver synthetic function and near-normal BMI, emphasizing insulin resistance as an important extrahepatic manifestation of HCV infection. However, the cross-sectional design limits causal inference, and the absence of a control group restricts comparison with the general population. Additionally, HOMA-IR is an indirect measure of insulin resistance, while lifestyle factors and histopathological liver biopsy correlation were not assessed, which may influence interpretation of the findings.

Conclusion

The present study identified insulin resistance as a prevalent and clinically relevant metabolic impairment in chronic hepatitis C virus infection. Insulin resistance was observed in 28% of study participants, including 20% with significant insulin resistance and 8% with early insulin resistance based on HOMA-IR assessment. Despite the relatively young study population (21–40 years), insulin resistance was observed in nearly one-fourth of patients, suggesting that metabolic dysfunction begins early in the disease process. Insulin resistance in patients with near-normal glycaemic indices and preserved liver synthetic function indicates that hepatitis C infection has systemic metabolic consequences independent of obesity and diabetes. The absence of a significant association between body mass index and insulin resistance further supports the role of virus-dependent and hepatic mechanisms rather than generalized adiposity.

A significant association was observed between insulin resistance and disease severity, with HOMA-IR scores increasing alongside clinical severity and liver stiffness values. Although categorical fibrosis staging did not show significant differences in mean HOMA-IR scores, the continuous positive relationship between FibroScan values and HOMA-IR suggests progressive metabolic dysfunction with increasing hepatic stiffness. The high prevalence of hepatic steatosis further supports the biological role of hepatitis C infection in disrupting insulin signalling and hepatic metabolism.

The findings emphasize the need for early

metabolic evaluation and intervention in chronic hepatitis C patients, even among young, non-obese, and non-diabetic individuals. Routine HOMA-IR assessment may help identify patients at risk of diabetes, cardiovascular disease, and progressive hepatic fibrosis. Comprehensive management of hepatitis C should therefore include both viral eradication and metabolic risk modification to improve long-term hepatic and systemic outcomes.

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