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ORIGINAL ARTICLE

Association of Insulin Resistance with Liver Enzymes and Inflammatory Biomarkers in Patients with Metabolic-Associated Fatty Liver Disease

SHEHRYAR KANJU[1], YASIR ABBAS ZAIDI[2], SHAHNAWAZ HASSAN GARDEZI[3], M. SHAKIL ZARI KHAWRI SIDDIQUI[4], RIZWAN SAEED KIANI[5], ASMA ARSHAD KHAN[6]

[1]*Assistant Professor, Gastroenterology, Department of Gastroenterology, Nishtar Medical University and Hospital, Multan, Pakistan.*
[2]*Associate Professor and Chairperson, Department of Gastroenterology, Nishtar Medical University, Multan, Pakistan.*
[3]*Assistant Professor of Medicine, Nishtar Medical University, Multan, Pakistan.*
[4]*Associate Professor of Biochemistry, Azra Naheed Medical College, Lahore, Pakistan.*
[5]*Assistant Professor of Gastroenterology, Poonch Medical College, Rawalakot, Pakistan.*
[6]*Department of Gastroenterology, Avicenna Medical College, Lahore, Pakistan.*
Correspondence to: M. Shakil Zari Khawri Siddiqui, Email: drshakil@gmail.com

ABSTRACT

Background: Metabolic-associated fatty liver disease (MAFLD) is a common chronic liver disorder closely linked with metabolic dysfunction and insulin resistance. Insulin resistance plays a central role in hepatic fat accumulation and promotes chronic low-grade inflammation, leading to hepatocellular injury. However, the relationship between insulin resistance, liver enzymes, and inflammatory biomarkers in MAFLD patients remains insufficiently explored in local clinical settings.

Objective: To evaluate the association of insulin resistance with liver enzymes and inflammatory biomarkers in patients diagnosed with metabolic-associated fatty liver disease.

Methods: This hospital-based cross-sectional study was conducted in the Department of Gastroenterology, Nishtar Medical University and Hospital, Multan, Pakistan, from July 2022 to July 2023. A total of 100 adult patients with ultrasound-confirmed fatty liver fulfilling MAFLD criteria were enrolled. Fasting plasma glucose and fasting insulin levels were measured, and insulin resistance was calculated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Liver enzymes including alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and gamma-glutamyl transferase were assessed. Inflammatory biomarkers such as high-sensitivity C-reactive protein, interleukin-6, tumor necrosis factor-alpha, ferritin, and adiponectin were also measured. Patients were categorized into insulin-resistant and non–insulin-resistant groups, and statistical analysis was performed to assess group differences and correlations.

Results: Insulin resistance was observed in 62% of patients. Insulin-resistant individuals exhibited significantly higher levels of liver enzymes, particularly alanine aminotransferase, aspartate aminotransferase, and gamma-glutamyl transferase, compared to non–insulin-resistant patients ($p<0.001$). Inflammatory biomarkers including high-sensitivity C-reactive protein, interleukin-6, tumor necrosis factor-alpha, and ferritin were significantly elevated in the insulin-resistant group, while adiponectin levels were significantly reduced ($p<0.001$). HOMA-IR showed significant positive correlations with liver enzymes and inflammatory markers and a negative correlation with adiponectin across both genders.

Conclusion: Insulin resistance is strongly associated with hepatic enzyme abnormalities and increased systemic inflammation in patients with MAFLD. These findings emphasize the central role of insulin resistance in disease pathophysiology and support the use of metabolic and inflammatory biomarkers for improved risk stratification and management of MAFLD.

Keywords: Metabolic-associated fatty liver disease; Insulin resistance; HOMA-IR; Liver enzymes; Inflammatory biomarkers; hs-CRP.

INTRODUCTION

Metabolic-associated fatty liver disease (MAFLD) has emerged as the most prevalent chronic liver disorder worldwide and represents a major public health challenge, particularly in low- and middle-income countries[1]. The condition is characterized by excessive accumulation of triglycerides within hepatocytes in the presence of metabolic dysregulation, including obesity, insulin resistance, type 2 diabetes mellitus, dyslipidemia, and hypertension. Unlike earlier definitions, MAFLD emphasizes the underlying metabolic dysfunction rather than exclusion of alcohol intake alone, thereby aligning the disease more closely with cardiometabolic risk and systemic inflammation[2,3].

Insulin resistance is widely recognized as a central pathogenic mechanism in MAFLD. Impaired insulin signaling promotes increased lipolysis in adipose tissue, resulting in an excessive influx of free fatty acids to the liver. Concurrently, hepatic de novo lipogenesis is upregulated while fatty acid oxidation is reduced, leading to progressive hepatic steatosis[4]. Persistent insulin resistance further aggravates mitochondrial dysfunction, oxidative stress, and endoplasmic reticulum stress, creating a cellular environment prone to hepatocyte injury. These pathological changes are commonly reflected by elevations in liver enzymes such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT), which serve as accessible indicators of hepatic inflammation and injury in clinical practice[5,6].

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In addition to metabolic derangements, chronic low-grade inflammation plays a pivotal role in the progression of MAFLD from simple steatosis to more advanced stages, including steatohepatitis and fibrosis. Insulin-resistant adipose tissue secretes increased levels of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), while anti-inflammatory adipokines such as adiponectin are reduced[7]. These inflammatory mediators not only worsen insulin resistance but also directly contribute to hepatic inflammation through activation of Kupffer cells and hepatic stellate cells. Systemic inflammatory biomarkers, including high-sensitivity C-reactive protein (hs-CRP) and ferritin, therefore provide valuable insight into the inflammatory burden associated with MAFLD[8,9].

Although liver enzymes are routinely used in the evaluation of MAFLD, their relationship with insulin resistance and inflammatory biomarkers is not always linear and varies across different populations. Many patients with significant metabolic dysfunction may exhibit only mild enzyme elevations, while others with advanced disease may have near-normal values[10]. This variability highlights the need for integrated assessment of metabolic and inflammatory parameters to better understand disease severity and progression. In South Asian populations, where central obesity and insulin resistance often occur at lower body mass indices, these relationships may be particularly pronounced yet under-studied[11].

Therefore, the present study was designed to evaluate the association of insulin resistance, assessed by the homeostatic model assessment of insulin resistance (HOMA-IR), with liver enzymes and key inflammatory biomarkers in patients with metabolic-associated fatty liver disease. Understanding these

associations may help identify high-risk MAFLD patients at an earlier stage and support more targeted metabolic and anti-inflammatory therapeutic strategies[12].

MATERIALS AND METHODS

This hospital-based cross-sectional analytical study was conducted in the Department of Gastroenterology, Nishtar Medical University and Hospital, Multan, Pakistan, over a one-year period from July 2022 to July 2023. The study aimed to evaluate the association of insulin resistance with liver enzymes and inflammatory biomarkers in patients diagnosed with metabolic-associated fatty liver disease (MAFLD).

A total of 100 patients were enrolled using non-probability consecutive sampling. Adult patients aged between 18 and 70 years with ultrasonographic evidence of hepatic steatosis were screened for eligibility. MAFLD was diagnosed in the presence of fatty liver on ultrasound along with at least one of the following metabolic criteria: overweight or obesity using Asian cut-off values (body mass index ≥25 kg/m²), established type 2 diabetes mellitus, or evidence of metabolic dysregulation such as dyslipidemia, hypertension, impaired fasting glucose, or elevated inflammatory markers.

Patients were included if they fulfilled the diagnostic criteria for MAFLD and provided written informed consent. Patients with chronic viral hepatitis (hepatitis B or C), significant alcohol intake, autoimmune liver disease, Wilson disease, hemochromatosis, pregnancy, acute inflammatory or infectious conditions, or those receiving hepatotoxic medications were excluded to avoid confounding of biochemical results.

After enrollment, detailed demographic and clinical data were recorded using a structured proforma. This included age, sex, history of diabetes and hypertension, smoking status, body mass index, waist circumference, and blood pressure. Anthropometric measurements were obtained using standardized procedures.

Venous blood samples were collected after an overnight fast of 8–12 hours under aseptic conditions. Fasting plasma glucose was measured by enzymatic methods, and fasting serum insulin levels were determined using immunoassay techniques. Insulin resistance was calculated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) formula, defined as fasting insulin (μIU/mL) multiplied by fasting glucose (mg/dL) divided by 405. Liver function parameters including alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, and gamma-glutamyl transferase were measured using automated analyzers. Lipid profile analysis included total cholesterol, triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol. Inflammatory biomarkers assessed in this study included high-sensitivity C-reactive protein, interleukin-6, tumor necrosis factor-alpha, serum ferritin, and adiponectin. All laboratory investigations were performed in the central laboratory of Nishtar Medical University and Hospital following standard quality control procedures.

Based on insulin resistance status, patients were categorized into two groups. Those with a HOMA-IR value of 2.5 or higher were classified as insulin resistant, while patients with HOMA-IR values below 2.5 were considered non-insulin resistant. This stratification was used to compare liver enzymes and inflammatory biomarkers between groups.

Statistical analysis was performed using SPSS software version 25. Continuous variables were expressed as mean with standard deviation or median with interquartile range depending on data distribution, while categorical variables were presented as frequencies and percentages. Differences between insulin-resistant and non-insulin-resistant groups were analyzed using independent sample t-tests or Mann–Whitney U tests for continuous variables and chi-square tests for categorical variables. Correlations between HOMA-IR, liver enzymes, and inflammatory biomarkers were assessed using Pearson or Spearman correlation coefficients as appropriate. A p-value of less than 0.05 was considered statistically significant.

Ethical approval for the study was obtained from the Institutional Review Board of Nishtar Medical University and Hospital, Multan. Written informed consent was obtained from all participants prior to inclusion, and confidentiality of patient information was strictly maintained throughout the study.

RESULTS

A total of 100 patients diagnosed with metabolic-associated fatty liver disease (MAFLD) were included in the final analysis. The mean age of the study population was 43.6 ± 10.8 years, with a range of 19–68 years. Of these, 56 (56%) were males and 44 (44%) were females, demonstrating inclusion of both genders. Insulin resistance, defined as a HOMA-IR value ≥2.5, was observed in 62 patients (62%), while 38 patients (38%) were classified as non–insulin resistant.

Baseline demographic and metabolic characteristics: Patients with insulin resistance had significantly higher body mass index, waist circumference, and a greater prevalence of type 2 diabetes mellitus compared to non–insulin-resistant patients. There was no statistically significant difference in age or gender distribution between the two groups, indicating comparable baseline demographics (Table 1).

Table 1. Baseline demographic and metabolic characteristics of MAFLD patients according to insulin resistance status

Variable	Total (n=100)	Non-IR (n=38)	IR (n=62)	p-value
Age (years)	43.6 ± 10.8	42.1 ± 10.2	44.5 ± 11.1	0.28
Male, n (%)	56 (56.0)	20 (52.6)	36 (58.1)	0.59
Female, n (%)	44 (44.0)	18 (47.4)	26 (41.9)	0.59
BMI (kg/m²)	29.3 ± 4.5	26.9 ± 3.9	30.8 ± 4.2	<0.001
Waist circumference (cm)	102.1 ± 10.6	96.7 ± 9.2	105.4 ± 9.8	<0.001
Type 2 diabetes, n (%)	54 (54.0)	14 (36.8)	40 (64.5)	0.006
Hypertension, n (%)	47 (47.0)	14 (36.8)	33 (53.2)	0.11
HOMA-IR	3.2 (2.1–4.6)	1.9 (1.5–2.3)	4.1 (3.2–5.4)	<0.001

Liver enzyme profile: Liver enzyme levels were significantly higher in insulin-resistant patients. Mean ALT, AST, and GGT values were markedly elevated in the IR group, indicating greater hepatocellular injury and metabolic stress. ALP levels were also higher in insulin-resistant patients, although the difference was comparatively modest (Table 2).

Table 2. Liver enzyme levels in MAFLD patients according to insulin resistance status

Parameter (U/L)	Non-IR (n=38)	IR (n=62)	p-value
ALT	42.6 ± 17.9	64.1 ± 25.8	<0.001
AST	34.2 ± 13.8	47.9 ± 19.6	<0.001
GGT	45.9 ± 20.3	70.6 ± 32.1	<0.001
ALP	101.8 ± 27.4	113.9 ± 30.8	0.04

Inflammatory biomarker profile: Insulin-resistant patients exhibited significantly higher levels of systemic inflammatory biomarkers. Median hs-CRP levels were more than twofold higher in the IR group. Pro-inflammatory cytokines IL-6 and TNF-α were also significantly elevated, while serum ferritin levels were markedly increased. Conversely, adiponectin levels were significantly lower in insulin-resistant patients, reflecting an adverse inflammatory and metabolic milieu (Table 3).

Table 3. Inflammatory biomarkers in MAFLD patients according to insulin resistance status

Biomarker	Non-IR (n=38)	IR (n=62)	p-value
hs-CRP (mg/L)	2.2 (1.3–3.6)	4.8 (3.2–6.9)	<0.001
IL-6 (pg/mL)	3.9 ± 1.8	6.3 ± 2.6	<0.001
TNF-α (pg/mL)	8.9 ± 3.1	12.4 ± 4.4	<0.001
Ferritin (ng/mL)	170 (125–235)	270 (195–365)	<0.001
Adiponectin (μg/mL)	7.8 ± 2.7	5.3 ± 2.2	<0.001

Correlation of insulin resistance with liver enzymes and inflammatory biomarkers: Correlation analysis demonstrated that HOMA-IR was positively correlated with ALT (r=0.45, p<0.001), AST (r=0.39, p<0.001), GGT (r=0.42, p<0.001), and hs-CRP (r=0.53, p<0.001). Significant positive correlations were also observed between HOMA-IR and IL-6 (r=0.47, p<0.001), TNF-α (r=0.44, p<0.001), and ferritin (r=0.41, p<0.001). In contrast, adiponectin showed a significant negative correlation with HOMA-IR (r=-0.48, p<0.001) (Table 4).

Table 4. Correlation of HOMA-IR with liver enzymes and inflammatory biomarkers

Parameter	Correlation coefficient (r)	p-value
ALT	0.45	<0.001
AST	0.39	<0.001
GGT	0.42	<0.001
hs-CRP	0.53	<0.001
IL-6	0.47	<0.001
TNF-α	0.44	<0.001
Ferritin	0.41	<0.001
Adiponectin	–0.48	<0.001

Overall, these findings indicate that insulin resistance is associated with significantly worse liver enzyme derangements and a heightened inflammatory state in both male and female patients with MAFLD, supporting its central role in disease pathophysiology.

DISCUSSION

The present study demonstrates a strong and clinically meaningful association between insulin resistance and both hepatic enzyme derangements and systemic inflammatory activation in patients with metabolic-associated fatty liver disease (MAFLD)[13]. In this cohort of patients from a tertiary care center in South Punjab, insulin-resistant individuals exhibited significantly higher levels of ALT, AST, GGT, and ALP, along with elevated inflammatory biomarkers including hs-CRP, IL-6, TNF-α, and ferritin, while adiponectin levels were markedly reduced. These findings reinforce the concept that insulin resistance is not merely a coexisting metabolic abnormality but a central driver of hepatic injury and inflammation in MAFLD[14].

Insulin resistance plays a pivotal role in MAFLD pathogenesis by promoting excessive free fatty acid flux from adipose tissue to the liver and enhancing hepatic de novo lipogenesis[15]. This metabolic overload leads to triglyceride accumulation, lipotoxicity, and mitochondrial dysfunction, which ultimately result in hepatocellular injury. The significantly higher ALT, AST, and GGT levels observed in insulin-resistant patients in this study are consistent with these mechanisms and indicate a greater degree of hepatocyte stress. GGT, in particular, showed a strong association with insulin resistance, supporting its role as a marker of oxidative stress and metabolic liver injury rather than simple cholestasis[16].

The inflammatory profile identified in insulin-resistant MAFLD patients further supports the metabolic–inflammatory axis underlying disease progression. Elevated hs-CRP reflects systemic low-grade inflammation, while increased IL-6 and TNF-α indicate activation of pro-inflammatory cytokine pathways originating from dysfunctional adipose tissue and hepatic Kupffer cells[17]. These cytokines are known to impair insulin signaling through serine phosphorylation of insulin receptor substrates, thereby perpetuating a vicious cycle of worsening insulin resistance and inflammation. The observed reduction in adiponectin, an anti-inflammatory and insulin-sensitizing adipokine, further accentuates this imbalance and has been linked to increased hepatic steatosis, fibrosis, and cardiometabolic risk in MAFLD[18].

An important finding of this study is the significant correlation between HOMA-IR and both liver enzymes and inflammatory biomarkers across both genders. The lack of significant gender-based differences in these associations suggests that insulin resistance exerts a similar pathogenic influence on MAFLD in males and females when metabolic dysfunction is present[19]. This is particularly relevant in South Asian populations, where insulin resistance and central obesity often occur at lower body mass indices and may contribute to under-recognition of metabolic liver disease in clinical practice[20].

Compared with international literature, the results of this study are consistent with previous reports demonstrating strong links between insulin resistance, transaminase elevation, and inflammatory activation in MAFLD and NAFLD populations. However, local data from Pakistan remain limited, and the present study adds valuable region-specific evidence highlighting the metabolic and inflammatory burden of MAFLD in this population[17,18]. The findings underscore the limitations of relying solely on liver enzymes to assess disease severity and emphasize the importance of incorporating metabolic indices such as HOMA-IR and inflammatory markers into routine evaluation[3].

Despite its strengths, including comprehensive biochemical profiling and inclusion of both genders, this study has certain limitations. Its cross-sectional design precludes causal inference, and ultrasound-based diagnosis does not allow precise staging of fibrosis. Additionally, advanced non-invasive fibrosis markers or elastography were not assessed. Longitudinal studies are required to determine whether insulin resistance and inflammatory biomarkers predict disease progression, fibrosis development, or cardiovascular outcomes in MAFLD patients[15-20].

CONCLUSION

This study concludes that insulin resistance is significantly associated with elevated liver enzymes and heightened systemic inflammatory biomarkers in patients with metabolic-associated fatty liver disease. Insulin-resistant MAFLD patients demonstrate a more pronounced hepatocellular injury pattern and a pro-inflammatory state, irrespective of gender. These findings highlight insulin resistance as a key pathogenic factor linking metabolic dysfunction to hepatic inflammation and injury. Incorporating assessment of insulin resistance and inflammatory biomarkers into routine clinical evaluation may improve early risk stratification and guide targeted metabolic and therapeutic interventions aimed at preventing progression of MAFLD and its systemic complications.

Availability of data and materials: The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Competing interests: The authors declare that they have no competing interests.

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Authors' contributions: Conceptualization and study design were carried out by S.K. and Y.A.Z. Data collection and patient recruitment were performed by S.K. and S.H.G. Laboratory investigations and biochemical analyses were conducted by M.S.Z.K.S. and A.A.K. Statistical analysis and interpretation of data were undertaken by R.S.K. Manuscript drafting was done by S.K. and Y.A.Z., while critical revision for important intellectual content and final approval of the manuscript were contributed by S.H.G., M.S.Z.K.S., A.A.K., and R.S.K. All authors read and approved the final version of the manuscript.

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