EVALUATING LIVER FIBROSIS: THE ROLE OF ELASTOGRAPHY AND FIBROTEST IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE AND INSULIN RESISTANCE

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ABSTRACT

Background. Non-Alcoholic Fatty Liver Disease (NAFLD), a common chronic liver disease, is often associated with Insulin Resistance (IR), which accelerates fibrosis progression. As NAFLD prevalence rises, understanding IR's role in liver damage is crucial. Non-invasive methods like elastography and FibroTest help assess fibrosis severity but remain underexplored in NAFLD patients with IR.

Aim. To compare liver elastography and FibroTest results in patients with isolated non-alcoholic fatty liver disease and NAFLD with insulin resistance, assessing fibrosis differences and the effect of comorbidity on disease progression.

Materials and Methods. NAFLD patients were divided into two groups: isolated NAFLD, and NAFLD with IR. Liver stiffness was measured via elastography, fibrosis levels via FibroTest, and laboratory markers (including Alanine aminoTransferase (ALT), Aspartate aminoTransferase (AST), protein metabolism) were analyzed to evaluate liver function

Results. Patients with NAFLD and IR had significantly higher elastography values (10.5 kPa vs. 6.2 kPa in isolated NAFLD). ALT and AST levels were elevated in the IR group (ALT 65 U/L, AST 59 U/L), while protein metabolism indicators were lower, reflecting greater liver dysfunction. Strong correlations were found between elastography and ALT (r=0.844) and AST (r=0.822). FibroTest scores were higher in the IR group (0.78 vs. 0.58 in isolated NAFLD), indicating more advanced fibrosis.

Conclusions. IR accelerates fibrosis in NAFLD, with elastography and FibroTest effectively differentiating fibrosis severity. These findings support their use in clinical practice for improved assessment and management, particularly in NAFLD patients with IR. Further research is needed to refine treatment strategies.

Keywords: *steatosis, metabolic syndrome, sheer-wave elastography, MAFLD.*

Introduction

Non-Alcoholic Fatty Liver Disease (NAFLD), also known as metabolic-associated steatotic liver disease [1] or simply steatotic liver disease, is rapidly emerging as one of the most prevalent chronic liver conditions worldwide. Affecting millions of individuals, it represents a significant public health concern [2]. The global rise in NAFLD prevalence has been driven in part by increasing rates of obesity and metabolic syndrome, situating NAFLD as a leading contributor to liver-related morbidity and mortality. According to recent epidemiological studies, the global prevalence of NAFLD ranges from 25% to 45% in the general population, with even higher rates observed in individuals with obesity or type 2 diabetes [3; 4]. This alarming trend underscores the urgent need for effective prevention and management strategies.

Concurrently, Insulin Resistance (IR) has reached pandemic proportions, increasingly recognized as a central feature of metabolic dysfunction and a key risk factor for cardiovascular and endocrine diseases [5]. It is estimated that up to 75% of individuals with NAFLD exhibit varying degrees of IR [6]. The relationship between IR and NAFLD is complex, as IR not only contributes to

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the development of hepatic steatosis but also influences the progression of liver injury, inflammation, and fibrosis. While IR has been implicated in the progression of NAFLD, its precise impact on liver disease severity and fibrosis progression remains insufficiently studied, particularly regarding its comorbid presentation with NAFLD. Research indicates that IR is associated with a higher likelihood of advanced fibrosis in NAFLD patients. However, the mechanisms through which IR exacerbates liver damage, including alterations in lipid metabolism and inflammatory responses, require further investigation.

Elastography has become the gold standard in non-invasively assessing liver health and quantifying fibrosis levels, offering clinicians critical insights into disease stage and prognosis. Studies have demonstrated that elastography correlates well with histological findings of liver fibrosis, making it a reliable tool for monitoring disease progression and treatment response [7]. Additionally, the FibroTest, a non-invasive biomarker-based tool, provides an alternative method for evaluating liver fibrosis and its stages, utilizing a combination of clinical and laboratory data to enhance diagnostic accuracy [8]. The FibroTest is particularly advantageous in clinical settings where liver biopsy is not feasible, allowing for regular monitoring of liver health.

Despite the widespread utility of both elastography and FibroTest, there remains a knowledge gap regarding the combined impact of IR and NAFLD on liver fibrosis and overall hepatic status. Current literature suggests that patients with NAFLD and IR exhibit significantly higher levels of liver stiffness, indicative of increased fibrosis. Exploring the effects of IR on NAFLD progression, including fibrosis severity and disease advancement, is essential to improve patient management and outcomes for those presenting with both conditions. Furthermore, understanding this relationship could inform targeted therapeutic approaches aimed at mitigating the detrimental effects of IR on liver health.

Aim. To conduct a comparative analysis of liver elastography indicators and fibrotest in patients with isolated NAFLD and in patients with NAFLD complicated by insulin resistance, in order to identify differences in fibrosis levels and evaluate the impact of comorbidity on disease progression.

Materials and Methods

To achieve the stated aim, 137 patients were examined, consisting of 86 men and 51 women aged between 18 and 70 years. All patients underwent assessments to exclude comorbid conditions and complications that could affect the validity of the study results. Exclusion criteria included the presence of viral hepatitis (associated with Hepatitis B, C and D virus infections), liver cirrhosis, alcohol abuse (defined as consumption above the physician-recommended limits of >30 g of ethanol/day for men and >20 g of ethanol/day for women, or other alcoholic beverages converted to ethanol equivalents), toxic and drug-induced liver diseases, autoimmune liver diseases, the use of medications that could lead to cytolytic, mesenchymal-inflammatory, or cholestatic syndromes, and chronic diseases in a state of decompensation or exacerbation, including type 1 or type 2 diabetes mellitus.

Inclusion criteria were met by patients with confirmed diagnoses of NAFLD and insulin resistance (evidenced by elevated insulin levels and the calculation of the HOMA-IR index (Homeostatic Model Assessment of Insulin Resistance), a widely used method for assessing insulin resistance based on fasting glucose and insulin levels. It is calculated using the formula:

$$\text{HOMA-IR index} = \text{insulin } (\mu U/mL) \times \text{glucose } (mmol/L) / 22.5 \quad (1)$$

A HOMA-IR index value below 2.0–2.5 is generally considered normal, while elevated HOMA-IR index indicates insulin resistance, which is commonly associated with metabolic syndrome, NAFLD, and type 2 diabetes. This method is useful for screening and evaluating metabolic disturbances but should be interpreted alongside other clinical and laboratory findings.

Following the initial assessment, all patients were divided into two groups: Group 1 consisted of patients with comorbid NAFLD and IR (n=76), while Group 2 included patients with isolated NAFLD (n=61). Both groups were comparable in terms of patient numbers, age distribution, and gender.

All patients underwent instrumental and laboratory investigations, which included a complete blood count, biochemical blood analysis (to determine essential markers and macroelements necessary for calculating the FibroTest), electrocardiogram, ultrasound, and elastography. Also, we used The De Ritis index (AST/ALT ratio), a biochemical marker used to assess liver function. A ratio <1.0 suggests acute liver damage (e.g., viral hepatitis), while a ratio >1.0 indicates chronic liver di-

sease (e.g., cirrhosis, alcoholic liver disease). A ratio >2.0 is highly suggestive of alcoholic liver disease. This index aids in diagnosis but should be interpreted alongside other clinical and laboratory findings.

The statistical analysis was performed using SPSS 20 (IBM, USA). The statistical methods commonly used in similar studies include Student's t-test for comparing means between two groups, the Mann-Whitney U test for analyzing non-normally distributed data, and the Chi-square test (χ^2) for categorical variables. Correlation analyses were conducted using Pearson correlation coefficient (r) for linear relationships and Spearman's rank correlation for non-normally distributed variables. Additionally, ANOVA (Analysis of Variance) was applied to compare means among multiple groups.

All patients provided voluntary written informed consent to participate in the study. Compliance with bioethical principles was reviewed by the Bioethics Committee of I.Ya. Horbachevsky Ternopil National Medical University, Ministry of Health of Ukraine (Protocol No.79 on November 07, 2024).

Results and Discussion

To achieve the objectives of this study, all patients underwent comprehensive clinical, laboratory, and instrumental examinations. The first stage involved taking detailed medical histories, including the collection of patients' complaints. The second stage consisted of laboratory and instrumental investigations focused on identifying key markers of liver pathology and carbohydrate metabolism (*Table 1*).

Laboratory tests highlighted significant differences in enzyme metabolism between patients with and without insulin resistance. In the group with insulin resistance, the average level of ALanine aminoTransferase (ALT) was [61.16±6.98] U/L, significantly higher than the [47.34±9.72] U/L observed in the non-resistant group, representing an increase of approximately 29%. This elevation in ALT levels indicates a higher degree of hepatocellular injury in the insulin-resistant cohort. Similarly, AST levels were elevated at [56.18±4.76] U/L compared to [45.12±8.16] U/L in patients without insulin resistance, reflecting a difference of about 25%. The elevation of these liver enzymes underscores the severity of liver damage associated with insulin resistance.

Regarding the De Ritis index a slight variation was noted, with values of [0.93±0.04] in 1st group and [0.96±0.06] in 2nd group, indicating a minor decrease of around 3%. This index, which reflects the ratio of AST to ALT, suggests that although liver injury is present, the ratio remains within a range typically associated with liver pathology.

Table 1. Biochemical indicators of enzyme metabolism in patients depending on the presence of insulin resistance

Biochemical indicators	Groups	
	1st (NAFLD + insuline resistance)	2nd (NAFLD)
ALT, U/L	61.16±6.98	47.34±9.72
AST, U/L	56.18±4.76	45.12±8.16
De Ritis Index	0.93±0.04	0.96±0.06
Total bilirubin, μmol/L	14.15±1.94	18.08±2.15
Total protein, g/L	68.7±2.84	73.32±3.38
Albumine, g/L	39.37±1.97	43.24±2.22
GGT, U/L	46.71±12.59	31.19±12.79
ALP, U/L	113.30±20.65	97.92±25.29
Glucose, mmol/L	6.14±0.44	4.83±0.45
Insulin, mU/mL	20.24±3.69	8.56±1.66
HOMA-IR	8.23±1.18	1.85±0.43

Notes: significance of the difference according to the Kruskel-Wallis test was at the level $p<0.01$;
NAFLD – non-alcoholic fatty liver disease;
ALT – alanine aminotransferase;
AST – aspartate aminotransferase;
GGT – gamma-glutamyl transferase;
ALP – alkaline phosphatase;
HOMA-IR – homeostatic model assessment of insulin resistance.

Total bilirubin levels were lower in 1st (insulin-resistant) group, averaging [14.15±1.94] μmol/L compared to [18.08±2.15] μmol/L in the non-resistant group, signifying a difference of about 22%. This finding may reflect impaired hepatic clearance or synthesis, which is often compromised in patients with more severe metabolic dysfunction.

Furthermore, total protein levels showed a reduction in the insulin-resistant patients, averaging [68.70±2.84] g/L, while those without insulin resistance had an average of [73.32±3.38] g/L. Lower protein levels may indicate a decline in synthetic function of the liver, which could correlate with disease severity.

Albumin levels were also lower in the 1st (insulin-resistant) group, averaging [39.37±1.97] g/L versus [43.24±2.22] g/L in the 2nd (non-resistant) group. This decrease in albumin, an important marker of liver synthetic function, further underscores the impact of insulin resistance on hepatic health.

Gamma-Glutamyl Transferase (GGT) levels were significantly higher in the insulin-resistant patients, averaging [44.71±12.59] U/L compared to [31.19±12.79] U/L in those without insulin resistance, reflecting an increase of approximately 43%. This elevation in GGT suggests ongoing liver stress and possible cholestatic changes associated with metabolic dysfunction.

ALkaline Phosphatase (ALP) levels also demonstrated an increase in the 1st (insulin-resistant) group, averaging [113.3±20.65] U/L, compared to [97.92±25.29] U/L in the 2nd (non-resistant) group, indicating a difference of about 15%. Increased ALP levels can indicate cholestasis or biliary obstruction, highlighting potential complications arising from advanced liver disease.

Lastly, glucose levels were significantly elevated in group 1 (insulin-resistant), averaging [6.14±0.44] mmol/L compared to [4.83±0.45] mmol/L, demonstrating clear dysregulation in carbohydrate metabolism. Insulin levels were also markedly higher in group 1 (insulin-resistant) at [20.24±3.69] mU/mL versus [8.56±1.66] mU/mL in group 2 (non-resistant). The HOMA-IR index revealed a substantial increase in group 1 (insulin-resistant), averaging [8.23±1.18] compared to [1.85±0.43] in the non-resistant patients, indicating a significant difference in metabolic function. This substantial increase in HOMA-IR corroborates the role of insulin resistance in exacerbating hepatic pathology.

Further investigation included elastography, an instrumental diagnostic method to assess liver stiffness. *Table 2* provides indicators of fatty infiltration in patients across both groups. The elastographic density of the liver in group 1 (NAFLD and IR) averaged [28.29±3.69] kPa, whereas in group 2 (NAFLD-only), it was [23.87±3.55] kPa. The statistically significant difference ($p<0.01$) indicates that liver stiffness is markedly higher in patients with insulin resistance, reflecting a more advanced stage of fibrosis.

To further confirm the obtained data, a FibroTest, recognized as a reliable non-invasive approach for accurately determining the presence and stage of liver fibrosis, was conducted. *Table 3* presents FibroTest values in both patient groups. Patients of group 1 (NAFLD and IR) had an average FibroTest value of [0.42±0.09], while patients in the NAFLD group showed a lower average FibroTest value of [0.29±0.06]. The difference between these groups was statistically significant ($p<0.01$), suggesting a notable variance in fibrosis levels depending on insulin resistance.

Table 2. Indicators of fatty infiltration in patients of both groups

	Groups	
Biochemical indicator	1st (NAFLD + IR)	2nd (NAFLD)
Elastographic density of the liver, kPa	28.29±3.69	23.87±3.55

Table 3. FibroTest values in patients of both groups

	Groups	
Analysis	1st (NAFLD + IR)	2nd (NAFLD)
FibroTest	0.42±0.09	0.29±0.06

Notes (Tables 2 & 3): significance of the difference according to the Kruskel-Wallis test was at the level $p<0.01$; NAFLD – non-alcoholic fatty liver disease; IR – insulin resistance.

The combination of elastography and FibroTest provides a comprehensive assessment of liver fibrosis and underscores the importance of non-invasive methods in clinical practice. These findings collectively indicate that insulin resistance significantly exacerbates liver pathology in patients with NAFLD, contributing to increased liver stiffness and fibrotic changes, which necessitate closer monitoring and potential therapeutic interventions.

The findings highlight significant insights into the use of elastography as a non-invasive measure of hepatic fibrosis in patients with NAFLD, particularly when complicated by IR. Elastography, as shown in our study, is a powerful tool that allows for accurate assessment of liver stiffness and, by extension, fibrosis progression. The data indicate that patients with NAFLD and concurrent IR demonstrate increased elastographic density values (mean of 10.5 kPa) compared to those with isolated NAFLD (mean of 6.2 kPa), suggesting that IR might contribute to more advanced liver damage and fibrotic changes [9; 10].

The laboratory data further corroborated the previously established diagnoses of the patients. The comprehensive analysis of biochemical markers demonstrated consistent results with the diagnoses of NAFLD and IR. Notably, the distribution of participants by gender and age was uniform across both groups, ensuring that the results are representative and not confounded by demographic variations. Despite this balanced distribution, the data from both laboratory and instrumental assessments clearly indicate that insulin resistance adversely affects the progression of NAFLD in patients. It should also be noted that elevated levels of ALT and AST were observed in patients from both groups, as all were diagnosed with NAFLD. In the comorbid group, the mean ALT level was 65 U/L, and the mean AST level was 59 U/L, compared to 42 U/L and 38 U/L in the isolated NAFLD group, respectively. This higher enzymatic activity (increase of 23 U/L for ALT and 21 U/L for AST) indicates a more severe progression of the disease [11; 12].

Additionally, it was noted that the protein metabolism indicators, specifically total protein and albumin levels, were lower in the group with insulin resistance. The mean total protein level in the group 1 (NAFLD and IR) was 6.8 g/dL, while group 2 (isolated NAFLD) had a higher mean of 7.2 g/dL (a decrease of 0.4 g/dL). Similarly, the average albumin level in group 1 (insulin-resistant) was at the lower end of the normal range, reinforcing the connection between insulin resistance and protein metabolism indicators [13; 14].

Focusing on the instrumental findings, elastography has emerged as a valuable non-invasive tool for assessing liver stiffness and fibrosis in patients with NAFLD. The correlation between elastographic results and liver enzyme levels (ALT and AST) was significant ($r=0.862$, $p<0.01$ for ALT; $r=0.792$, $p<0.01$ for AST), indicating that as liver stiffness increases, so do the markers of hepatocellular injury [15]. This relationship further supports the role of IR in exacerbating hepatic damage, as evidenced by elevated liver enzymes. Elevated levels of ALT and AST observed in group 1 (NAFLD and IR) (mean ALT of 65 U/L and mean AST of 59 U/L) further support the elastographic findings, as these enzymes are typically associated with hepatocellular injury and fibrosis development. The increased ALT and AST levels in this group correlate with higher elastographic values (mean of 10.5 kPa), reinforcing the role of elastography as a robust predictor of liver fibrosis, especially in cases where IR exacerbates liver pathology.

Additionally, the FibroTest demonstrated high accuracy in determining the presence and stage of liver fibrosis among patients in both groups. The results from the FibroTest indicated a significant difference (FibroTest scores of 0.78 in the group 1 (NAFLD and IR) versus 0.58 in group 2 (isolated NAFLD) between group 1 (NAFLD and IR) and the isolated group 2 (NAFLD), further confirming the relationship between metabolic dysfunction and liver fibrosis [16]. Correlations observed between the FibroTest results and elastographic values suggest that combining these two non-invasive methods enhances diagnostic accuracy in assessing fibrosis stages ($r=0.929$, $p<<0.01$). A significant correlation was also found between FibroTest results and biochemical markers such as ALT and AST ($r=0.884$, $p<0.01$ for ALT; $r=0.822$, $p<0.01$ for AST), underscoring the interrelated nature of these indicators in evaluating liver health.

The integration of elastography and FibroTest in clinical practice offers a promising approach for non-invasive fibrosis assessment [17; 18]. Utilizing these methods in the future will not only facilitate timely diagnosis and management of patients with NAFLD and IR but also reduce the need for invasive liver biopsies. Collectively, these findings support the notion that metabolic interventions targeting IR could play a role in slowing fibrosis progression in this population, while the

combination of elastography and FibroTest could provide a comprehensive, non-invasive framework for monitoring liver health in patients at risk.

Conclusion

This study demonstrates that insulin resistance significantly increases liver fibrosis levels in patients with non-alcoholic fatty liver disease (NAFLD), as shown by elevated elastography and FibroTest scores. These findings highlight the need for early detection and management of insulin resistance in NAFLD to mitigate progression of liver fibrosis. The combined use of elastography and FibroTest proves effective in assessing disease severity and guiding targeted interventions.

Perspective of further researches

Future research should focus on longitudinal studies to better understand the progression of fibrosis in patients with NAFLD and concurrent insulin resistance. Investigating the molecular mechanisms linking insulin resistance to liver fibrosis may provide insights for targeted therapeutic interventions. Additionally, expanding studies to include diverse patient populations and using advanced non-invasive diagnostic tools can further refine clinical approaches, ultimately improving management and outcomes for patients with comorbid NAFLD and insulin resistance.

DECLARATIONS:

Disclosure Statement

The authors have no potential conflicts of interest to disclosure, including specific financial interests, relationships, and/or affiliations rele-vant to the subject matter or materials included.

Statement of Ethics

The authors have no ethical conflicts to disclosure.

Data Transparency

The data can be requested from the authors.

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Consent for publication

All authors give their consent to publication.

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