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Association between Serum Ferritin level and Liver Fibrosis Severity in Non-Alcoholic Fatty Liver Disease Patients

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ABSTRACT

Background & Objective: Non-alcoholic fatty liver disease (NAFLD) is a prevalent condition characterized by hepatic fat accumulation, which can progress fibrosis and cirrhosis. Serum ferritin has been proposed as a biomarker for liver disease, but its relationship with fibrosis severity in NAFLD remains unclear. This study explored the relationship between serum ferritin levels and liver fibrosis severity in NAFLD patients.

Methods: In this cross-sectional study, 204 NAFLD patients were enrolled, including 139 with mild fibrosis and 65 with severe fibrosis. Baseline and demographic characteristics were compared between groups. Serum ferritin and other biochemical parameters were measured. Logistic regression analyses assessed the predictive value of serum ferritin levels for liver fibrosis severity, and a receiver operating characteristic (ROC) curve determined the optimal ferritin cutoff for identifying sever fibrosis.

Results: Patients with severe fibrosis had significantly higher serum ferritin levels than those with mild fibrosis (197.70 ± 79.40 vs 95.70 ± 73.20, P<0.001). Logistic regression analysis confirmed a significant association between ferritin levels and fibrosis severity (OR = 1.015, 95% CI = 1.009–1.02, P<0.001). ROC analysis showed that ferritin distinguished severe from mild fibrosis with 80% sensitivity and 80% specificity at a cutoff of 129 ng/ml (AUC = 0.86).

Conclusion: Elevated serum ferritin levels are associated with more severe liver fibrosis in NAFLD patients, supporting ferritin's potential as a non-invasive biomarker for fibrosis severity. Further studies are needed to validate these findings and explore ferritin's diagnostic and prognostic utility in diverse populations.

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Introduction

Non-alcoholic fatty liver disease (NAFLD) is a growing public health concern, with its associated mortality and morbidity primarily linked to liver fibrosis (1). Giving the rising prevalence of NAFLD, early detection of fibrosis is critical. Recent guidelines emphasize risk stratification for fibrosis in NAFL patients, highlighting the need for effective non-invasive screening tools (2, 3). Such tools are essential to reduce the costs and risks of liver biopsy, enabling broader screening and diagnosis (4).

Elevated hepatic iron levels are associated with reduced antioxidant protection, increased oxidative stress, insulin resistance, and hepatic fibrosis, contributing to liver disease progression (5). Serum ferritin, a marker of iron overload, is often elevated in NAFLD patients (6), with some studies suggesting that hyperferritinemia may independently indicate NAFLD (7, 8). As an acute-phase protein, ferritin is elevated in conditions such as oxidative stress, inflammation, and metabolism dysregulation, all of which are linked to NAFLD (9-11). Moreover, ferritin has been implicated in liver fibrosis and cirrhosis through its association with genetic variants, such as PNPLA3-rs738409-G and TM6SF2-rs58542926-T (12).

Although several studies have examined the prognostic value of serum ferritin in NAFLD, the findings remain inconsistent. Some studies report a significant association between serum ferritin and liver

fibrosis or survival (13-15), while a recent large cross-sectional study of 3689 NAFLD patients found no such correlation (10). The diagnostic value of serum ferritin for severe liver fibrosis is also debated, with some studies suggesting it may be insufficient as a standalone tool (16-18). Therefore, further research is needed to clarify serum ferritin's utility as a non-invasive biomarker for NAFLD-associated fibrosis.

This study aimed to investigate the association between serum ferritin levels and established factors influencing liver fibrosis in NAFLD patients. In particular, this study aimed to evaluate the hyperferritinemia's predictive value for severe fibrosis, and determine the optimal ferritin cut-off, along with its sensitivity and specificity, for diagnosing severe liver fibrosis.

Materials and Methods

This cross-sectional study included adult patients with NAFLD referred to the gastroenterology clinics at Mashhad University of Medical Sciences in Mashhad, Iran and the study protocol was approved by the institutional committee on human research (IR.MUMS.MEDICAL.REC.1402.056), ensuring that it conformed to the ethical guidelines of the Declaration of Helsinki. Patients were diagnosed with NAFLD using elastography (FibroScan, Echosens, Paris, France), based on the American Association for the Study of Liver Diseases (AASLD) guidelines, which define NAFLD as hepatic steatosis detected by imaging in the absence of secondary causes (19). No liver biopsy or non-invasive scoring systems (e.g., NAFLD Fibrosis Score, FIB-4) were used for diagnosis. Liver fibrosis severity was assessed using the same Fibroscan device, with the M-probe (for BMI < 32 kg/m²) or XL-probe (for BMI > 32 kg/m²), measuring liver stiffness in kilopascals (kPa). FibroScan measurements were performed by trained operators following standardized protocols provided by the manufacturer (Echosens). Quality control was ensured by obtaining at least 10 valid measurements per patient, with an interquartile range (IQR) to median ratio of ≤30%, to confirm data reliability. Fibrosis stages were categorized as follows: F0-F1 (mild fibrosis, 2-7 Kpa), F2 (7-10 Kpa), F3 (10-14 Kpa), and F4 (severe fibrosis ≥14 Kpa) (19).

To exclude patients with a excessive alcohol use, we applied the Asia-Pacific guideline, which defines excessive alcohol consumption as more than 2 standard drinks per day or 140 grams per week for men, and more than 1 standard drink per day or 70 grams per week for women (20). We excluded patients with excessive alcohol use, other hepatic conditions (e.g., autoimmune hepatitis, hepatitis B or C, baseline ALT > 120 U/L, hepatic vascular impairments, hereditary disorders like Wilson's disease or hemochromatosis (TSAT >45%), steatogenic or hepatotoxic medications, hepatobiliary cancer), and extrahepatic conditions that could affect results or elevate ferritin levels (e.g., cardiovascular disorders, cancers, untreated hypothyroidism, iron deficiency anemia (TSAT <20%), minor thalassemia, alpha-1 antitrypsin deficiency, celiac disease), per AASLD guidelines.

After obtaining written informed consent from each patient, who was informed about the study's objectives and procedures, a comprehensive questionnaire collected demographic and clinical information, including sex, age, and medical. We measured and recorded patients' height and weight, and calculated Body Mass Index (BMI) using the formula BMI = weight (kg) / height (m²). We also recorded liver fibrosis results. In addition, 5 mL of venous blood was collected from each patient after a 12-hour fast, and serum was separated and stored at -20°C until analysis. Serum levels of ferritin, iron, total iron-binding capacity (TIBC), transferrin saturation (TSAT), aspartate transaminase (AST), alanine transaminase (ALT), alkaline phosphatase (ALP), low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), fasting plasma sugar (FPS), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were measured to assess potential confounders, including metabolic and inflammatory factors.

Statistical Analysis

The sample size was calculated based on a previous study that reported a prevalence of elevated ferritin levels of 10% in NAFLD patients with fibrosis and 39% in those without fibrosis (21). Assuming a significance level of 0.05 and a desired power of 80%, a minimum of 32 participants per group was estimated to be sufficient to detect a significant difference. All statistical analyses were performed using SPSS software version 22.0 for Windows (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro-Wilk test. Continuous variables were reported as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on data distribution. Group comparisons were conducted using the independent-samples t-test or Mann–Whitney U test for continuous variables and the Chi-square or Fisher's exact test for categorical variables, as appropriate.

To evaluate the association between serum ferritin levels and liver fibrosis severity, both univariate and multivariable logistic regression analyses were conducted. The latter adjusted for potential confounders such as age, BMI, gender, FBS, and AST to assess the independent effect of ferritin. Odds ratios (OR) with 95% confidence intervals (CI) were calculated to measure effect sizes. Multicollinearity among independent variables was assessed using the variance inflation factor (VIF), revealing no problematic collinearity (all VIF < 2). Model adequacy was further evaluated using the Hosmer–Lemeshow goodness-of-fit test, which indicated acceptable calibration.

Additionally, receiver operating characteristics (ROC) curve analysis was performed to identify the optimal ferritin cutoff for differentiating mild from severe fibrosis. Sensitivity, specificity, and the area under the curve (AUC) with 95% CI were calculated to evaluate diagnostic performance. A p-value < 0.05 was considered statistically significant.

Table 1. Baseline and Demographic Characteristics of NAFLD Patients Stratified by Fibrosis Severity

Variables		Low Fibrosis (F0/F1)	High Fibrosis (F2-F4)	P-value
		Mean±SD/Median (IQR)	Mean±SD/ Median (IQR)	
Gender	Male n (%)	47 (33.8)	30 (46.20)	**0.090**
	Female n (%)	92 (66.20)	35 (53.80)	
Age (years)		45.8 ± 12.10	11.9± 50.9	**0.006**
Past Medical History		59 (42.4)	32 (49.2)	**0.360**
BMI (kg/m2)		3.8± 30.1	5.0± 32.9	**0.001<**
Ferritin (ng/ml)		76.0 (52-106)	193 (142.9-234)	**0.001<**
TSAT		0.08 ±33.0	0.07± 34.0	**0.280**
FBS (mg/dl)		24.7± 105.9	40.7± 114.7	**0.060**
TG (mg/dl)		63.5± 158.9	61.1± 174.2	**0.100**
TC (mg/dl)		63.2± 186.6	45.7± 187.5	**0.870**
HDL (mg/dl)		9.6 ± 43.1	9.2± 42.0	**0.410**
LDL (mg/dl)		31.2± 108.9	39.6± 109.8	**0.850**
ALT (units/L)		40.2± 44.3	24.1± 43.8	**0.250**
AST (units/L)		17.2± 30.4	17.4± 36.2	**0.020**
ALP (IU/L)		79.5± 178.9	70.9± 190.4	**0.320**
SBP (mmHg)		8.6± 124.4	8.2± 127.1	**0.030**
DBP (mmHg)		3.7± 71.6	3.5± 73.3	**0.003**

Note: Data are presented as mean ± SD for normally distributed variables, median (IQR) for non-normally distributed variables (e.g., ferritin), or n (%) for categorical variables. Abbreviations: BMI, Body Mass Index; TSAT, Transferrin Saturation; FBS, Fasting Blood Sugar; TG, Triglycerides; TC, Total Cholesterol; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; ALP, Alkaline Phosphatase; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure.

Results

Baseline and Demographic Characteristics

This cross-sectional study included 204 NAFLD patients, with a mean age of 47.50 ± 12.28 years. Among them, 139 (68.1%) had mild fibrosis (F0-F1) and 65 (31.9%) had severe fibrosis (F2-F4). Most participants (n=127, 62.3%) were female. Table 1 presents baseline and demographic characteristics. Gender distribution (P=0.090) and past medical history (P=0.360) showed no significant differences between groups. However, age (45.08 ± 12.01 vs. 50.90 ± 11.90, P=0.006), BMI (30.10 ± 3.80 vs. 32.90 ± 5.00, P<0.001), AST (30.40 ± 17.20 vs. 36.20 ± 17.40, P=0.020), SBP (124.40 ± 8.60 vs. 127.10 ± 8.20, P=0.03), and DBP (71.60 ± 3.70 vs. 73.30 ± 3.50, P=0.003) differed significantly. No significant differences were found for TSAT (P=0.280), FBS (P=0.060), TG (P=0.100), TC (P=0.870), HDL (P=0.410), LDL (P=0.850), ALT (P=0.250), or ALP (P=0.320).

Regarding laboratory parameters no significant differences were observed TSAT (P=0.280), FBS (P=0.060), TG (P=0.100), CHOL (P=0.870), HDL (P=0.410), LDL (P=0.850), ALT (P=0.250), and ALP (P=0.320) between the two groups. However, a significant difference was found in AST levels, with the severe fibrosis group exhibiting higher levels compared to the mild group (36.20 ± 17.40 vs 30.4 ± 17.20, P-value: 0.02, P=0.020).

Serum Ferritin Concentrations

Serum ferritin levels in NAFLD patients with different degrees of fibrosis severity are presented in Figure 1. The data indicated a statistically significant difference in serum ferritin levels between NAFLD patients with severe fibrosis and those with mild fibrosis. Specifically, the group with severe fibrosis had significantly higher levels compared to the group with mild fibrosis (197.70 ± 79.40 vs 95.70 ± 73.20, P<0.001).

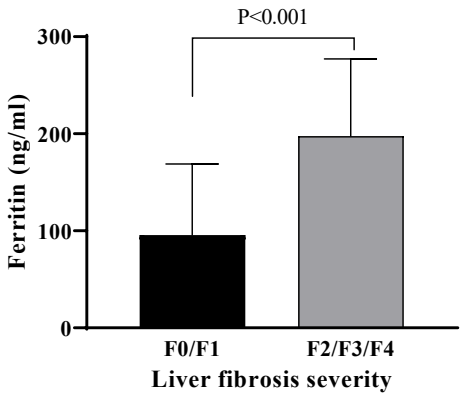

Fig. 1. Serum Ferritin levels in NAFLD patients with and without fibrosis severity

The Association of Ferritin Levels with Liver fibrosis severity

To evaluate the predictive value of serum ferritin levels for liver fibrosis severity in NAFLD patients, logistic regression analysis was performed in both univariate and multivariate models (Table 2). The results showed a significant association between serum ferritin levels and the risk of liver fibrosis development in NAFLD patients in both the univariate (OR = 1.016, 95% CI = 1.011–1.021, P < 0.001) and multivariate (OR = 1.015, 95% CI = 1.009–1.02, P < 0.001) models. The multivariate model adjusted for age, BMI, gender, FBS, and AST confirmed serum ferritin as an independent predictor of fibrosis severity. These findings indicate that serum ferritin levels are a significant independent predictor of liver fibrosis severity in NAFLD patients.

Table 2. Multivariable Logistic Regression Analysis of Factors Associated with Liver Fibrosis Severity

Variable	Adjusted OR 95% CI	P-value
Ferritin	1.018 (1.009-1.02)	<0.001
Age	1.04 (1.001-1.07)	0.033
Gender	1.41 (0.61-3.27)	0.421
BMI	1.20 (1.08-1.29)	<0.001
FBS	1.01 (0.99-1.02)	0.120
AST	1.02 (0.99-1.04)	0.227

Note: The model adjusted for age, BMI, gender, FBS, and AST. **Abbreviations:** OR, Odds Ratio; CI, Confidence Interval; BMI, Body Mass Index; FBS, Fasting Blood Sugar; AST, Aspartate Aminotransferase.

ROC Analyses of Ferritin Levels

To investigate the potential of ferritin as a novel biomarker for distinguishing NAFLD patients with severe fibrosis from those with mild fibrosis, ROC curve analysis was conducted. The results are presented in Figure 2. The optimal cutoff value of ferritin for distinguishing severe from mild fibrosis was found to be 129 ng/ml, with a corresponding sensitivity and specificity of 80%. Additionally, the area under the curve (AUC) was calculated to be 0.86, indicating a strong discriminatory ability of ferritin levels in predicting liver fibrosis severity.

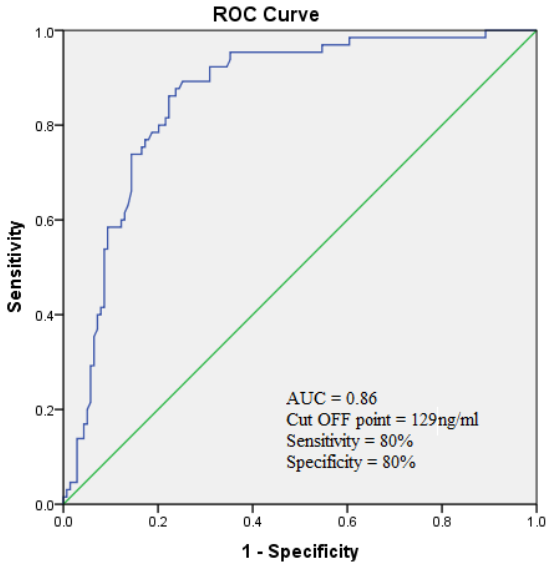

Fig. 2. Diagnostic value of serum Ferritin levels in distinguishing NAFLD patients with different fibrosis severity by ROC curve

Discussion

Our cross-sectional study identified a significant association between elevated serum ferritin levels and severe liver fibrosis in NAFLD patients. This finding is consistent with several prior studies but contrass with others, highlighting the complex role of ferritin in NAFLD progression. Specifically, our results show that patients with severe liver fibrosis had significantly higher serum ferritin levels compared to those with mild fibrosis. Furthermore, logistic regression analysis revealed that serum ferritin levels along with age and BMI, played a significant independent role in predicting liver fibrosis progression in NAFLD

patients, in both univariate and multivariate models. These findings suggest that serum ferritin levels may be a valuable biomarker for distinguishing NAFLD patients with severe liver fibrosis from those with mild fibrosis, with an optimal cutoff value of 129 ng/ml and corresponding sensitivity and specificity of 80%.

The mechanisms linking elevated serum ferritin to liver fibrosis in NAFLD are multifaceted, involving oxidative stress, inflammation, and iron dysregulation. In the context of NAFLD, ferritin is not only a marker of iron storage but also reflects the state of systemic and hepatic metabolic inflammation, a phenomenon known as “metabolic hyperferritinemia” (22). During states of chronic inflammation and insulin resistance, as observed in NAFLD, ferritin synthesis is upregulated in response to pro-inflammatory cytokines such as IL-6 and increased oxidative stress (22, 23). Reactive oxygen species (ROS), generated through mitochondrial dysfunction and iron-catalyzed Fenton reactions, promote hepatic stellate cell activation via the TGF-β/SMAD signaling pathway, driving collagen deposition and fibrogenesis (24, 25). Ferritin, in turn, serves as a compensatory mechanism to sequester excess iron, mitigating oxidative damage (26). Our study’s finding of elevated ferritin in severe fibrosis, despite normal transferrin saturation levels, suggests that ferritin’s role extends beyond iron overload to reflect inflammatory and fibrogenic processes in NAFLD (22, 27, 28).

Our results align with several studies that report a positive association between serum ferritin and liver fibrosis severity in NAFLD. For instance, Manousou et al. (2011) found that a ferritin cutoff of 240 ng/ml predicted hepatic inflammation and fibrosis with 91% sensitivity and 70% specificity, though their higher threshold may reflect the inclusion of patients with more advanced disease (16). Similarly, Yoneda et al. (2007) reported elevated ferritin levels in NAFLD patients with advanced fibrosis, supporting ferritin’s diagnostic potential (18). In contrast, Angulo et al. (2014) found limited diagnostic accuracy of ferritin for advanced fibrosis in NAFLD, possibly due to differences in study populations or diagnostic methods (e.g., biopsy vs. FibroScan) (29). These discrepancies may stem from differences in ethnicity, as our Iranian cohort may have unique genetic factors like PNPLA3-rs738409-G, or from methodological variations, such as our use of Fibroscan versus histological confirmation (12). Additionally, differences in age and sex distribution may influence ferritin levels (10). Our ferritin cutoff of 129 ng/ml, with 80% sensitivity and specificity, is lower than some prior thresholds, likely because we excluded patients with cirrhosis, which may elevate ferritin due to severe hepatocellular damage.

The clinical utility of serum ferritin as a non-invasive biomarker is supported by its association with histological markers of hepatic injury, including inflammation, hepatocellular ballooning, and fibrosis (13, 16). Our study’s cutoff of 129 ng/ml align with the range reported in other NAFLD cohorts, reinforcing its potential as a practical, cost-effective marker for fibrosis stratification, especially in resource-limited settings where biopsy or advanced imaging is unavailable.

Current clinical guidelines for non-invasive assessment of liver fibrosis in NAFLD recommend tools like the NAFLD Fibrosis Score (NFS), AST-to-platelet ratio index (APRI), the FIB-4 index, and Enhanced Liver Fibrosis (ELF) panel (2). Among these, the NFS incorporates six variables (age, BMI, hyperglycemia, AST/ALT ratio, serum albumin, and platelet count) and is effective for ruling out advanced fibrosis and predicting mortality (30). Likewise, FIB-4, based on age, AST, ALT, and platelet count, is widely used but can be confounded by age-related variability (31). The ELF panel, combining biomarkers such as hyaluronic acid, PIIINP, and TIMP-1, shows promise but struggles with consistent accuracy across populations (32). Importantly, none of these tools currently include serum ferritin, despite growing evidence of its role in metabolic inflammation and hepatic injury. Armandi et al. (2021) demonstrated that incorporating ferritin thresholds into FIB-4 and NFS enhances their prognostic capacity for liver-related and systemic complications in metabolic-associated steatotic liver disease (MASLD) (15). Our findings support this approach, suggesting that ferritin could be incorporated into composite scoring systems to improve fibrosis detection in NAFLD.

This study has several limitations that should be acknowledged. The cross-sectional design of our study prevents us from establishing a causal relationship between serum ferritin levels and liver fibrosis severity. Longitudinal studies are needed to investigate potential causal relationships. The relatively small sample size and the use of convenience sampling from a single center may limit the generalizability of the findings and reduce the statistical power to detect subtle associations. The reliance on FibroScan as the sole method for fibrosis assessment, while validated, may introduce variability due to operator skill and patient factors such as BMI or recent food intake, and future studies should incorporate additional diagnostic methods, such as liver biopsy or non-invasive scores, to enhance diagnostic accuracy. Additionally, the absence of liver biopsy as the gold standard for confirming NAFLD and fibrosis stages is a notable limitation, potentially affecting the accuracy of fibrosis classification. Future research with larger cohorts and biopsy-confirmed diagnoses is warranted to validate the clinical utility of serum ferritin as a non-invasive biomarker. Moreover, such studies should explore the establishment of fibrosis stage-specific ferritin cut-off values to improve diagnostic precision and enable better risk stratification of patients with NAFLD.

Conclusion

In conclusion, our findings indicate that serum ferritin levels may be a valuable non-invasive

biomarker for assessing liver fibrosis in patients with NAFLD. The significant association between elevated ferritin concentrations and fibrosis severity, along with its acceptable sensitivity and specificity in distinguishing mild from severe fibrosis, supports its clinical potential. These results encourage the consideration of ferritin in future diagnostic algorithms or fibrosis risk stratification models. However, larger, multicenter studies across diverse populations are needed to validate these findings and to further investigate the prognostic and diagnostic utility of serum ferritin in the context of NAFLD-related liver fibrosis.

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Data Availability

The data supporting the results of this study are available upon request from the corresponding author.

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Ethics Approval

Written informed consent was obtained from all patients, and the study protocol was approved by the institutional committee on human research (IR.MUMS.MEDICAL.REC.1402.056), ensuring that it conformed to the ethical guidelines of the Declaration of Helsinki.

Conflict of Interest

The authors declared no conflict of interest.

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