RESEARCH PAPER

Association of Serum Ferritin Levels with Disease Severity in Non-Alcoholic Fatty Liver Disease

Madhu Sharma[1], Dr. Bushra Fiza[2], Bhavana Singhal[3], Kavita Sharma[4], Shilpa Dhameja[1]
[1]PhD Scholar, Department of Biochemistry, Mahatma Gandhi Medical College & Hospital, MGMUST, 302022, Jaipur, Rajasthan, India.
[2]Department of Biochemistry, Mahatma Gandhi Medical College & Hospital, MGMUST, 302022, Jaipur, Rajasthan, India.
[3]Department of Allied Health Sciences, Mahatma Gandhi Institute of Allied Health Sciences, MGMUST, 302022, Jaipur, Rajasthan.
[4]Department of Allied Health Sciences Jagannath University, Sitapura, 302022, Jaipur, Rajasthan, India.
Corresponding Author*
Dr. Bushra Fiza
Professor
Department of Biochemistry, Mahatma Gandhi Medical College & Hospital, MGMUST, Jaipur, 302022, Rajasthan, India.
ORCID ID: https://orcid.org/0000-0001-8265-1226
Email ID: bushrafiza786@gmail.com

Abstract
Background: Non-alcoholic fatty liver disease (NAFLD) is a leading cause of chronic liver disease worldwide and is closely associated with metabolic syndrome. Serum ferritin, an acute-phase reactant and marker of iron stores, is frequently elevated in NAFLD patients.
Objective: To evaluate the association between serum ferritin levels and disease severity in patients with NAFLD.
Materials and Methods: This observational study included 50 patients diagnosed with NAFLD based on clinical, biochemical, and ultrasonographic findings. Serum ferritin levels were measured and correlated with liver enzymes, metabolic parameters and NAFLD severity grading. Disease severity was assessed using liver function tests, ultrasonography grading and non-invasive fibrosis markers. Statistical analysis was performed to determine the association between serum ferritin levels and NAFLD severity.
Results: The serum ferritin levels in the non-alcoholic fatty liver disease group were significantly higher than those without non-alcoholic fatty liver disease group and levels were elevated in patients with moderate to severe NAFLD compared to those with mild disease. Serum ferritin levels were significantly associated with the risk of non-alcoholic fatty liver disease in both men and women. Ferritin showed a positive correlation with alanine aminotransferase (ALT), aspartate aminotransferase (AST) and body mass index (BMI). Higher ferritin levels were associated with increased hepatic inflammation and fibrosis risk.
Conclusion: Serum ferritin levels were higher in patients with non-alcoholic fatty liver disease than in those without non-alcoholic fatty liver disease and were associated with the risk of non-alcoholic fatty liver disease in both men and women and may serve as a useful non-invasive biomarker for assessing hepatic inflammation and fibrosis progression.
Keywords: *MASH, Acute-phase reactant, Iron metabolism, Fibrosis, Metabolic syndrome*
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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is defined as excessive liver fat accumulation, with more than 5% of hepatocytes containing triglycerides, free fatty acids, ceramides, and free cholesterol [1] in the absence of heavy alcohol consumption or medication to induce liver steatosis [2]. The global prevalence of NAFLD is approximately 25.24%, and NAFLD has gradually become the common cause of morbidity and mortality in patients with liver-related chronic diseases [3]. In recent years, a considerable amount of evidence has shown the complex interactions between NAFLD and type 2 diabetes and obesity. Therefore, NAFLD has a significant impact on human health, and the early diagnosis and prevention of NAFLD are of utmost importance.

Non-alcoholic fatty liver disease includes a wide range of histological changes, mainly simple steatosis and Metabolic dysfunction associated steatohepatitis (MASH) [4]. MASH, previously known as NASH (Non-Alcoholic Steatohepatitis), refers to Metabolic Dysfunction-Associated Steatohepatitis. Steatosis can occur when the amount of fat in the liver cells is ≥5%, and the MASH can be diagnosed when any degree of hepatic steatosis, hepatocellular ballooning, or lobular inflammation occurs [5]. As MASH progresses, the liver becomes rigid, and liver function declines, leading to cirrhosis, hepatocellular carcinoma (HCC), death, or liver transplantation [6]. The MASH is the active period of NAFLD, and so we divide NAFLD into MASH and no-MASH. Compared with other liver diseases, 5%-50% of patients with HCC present with MASH before developing cirrhosis and routine safety screening, and these tumors tend to be larger and less treatable [7].

Non-alcoholic fatty liver disease (NAFLD) encompasses a spectrum of liver disorders ranging from simple steatosis to MASH, fibrosis, cirrhosis, and hepatocellular carcinoma. It is strongly associated with obesity, type 2 diabetes mellitus, dyslipidemia, and

insulin resistance, making it the hepatic manifestation of metabolic syndrome.

Iron is one of the most important micronutrients in living organisms and is an essential element for respiration and energy production in the mitochondria. Iron may cause oxidative damage to biomacromolecules by promoting the production of intracellular reactive oxygen species and changing the intracellular redox environment, eventually leading to chronic liver disease [8]. The liver plays a central role in iron metabolism and is the main source of hepcidin, a peptide hormone that regulates iron homeostasis. Excessive iron deposition, hepatocyte necrosis, and systemic inflammation caused by NAFLD may induce the overexpression of hepcidin, which decreases the intestinal iron absorption and increases the iron retention in macrophages and hepatocytes.

Ferritin is the primary storage mode of iron in the liver; once iron accumulates in the liver, it may damage the liver cell proteins and DNA through peroxidation stress. Ferritin can directly activate hepatic stellate cells and induce hepatic fibrosis through the nuclear factor κB cascade reaction [9].

The golden standard for diagnoses of NAFLD is liver pathological tissue biopsy however, it cannot be used as a routine screening method because of its invasive nature. Therefore, exploring a non-invasive, simple, and economical method for diagnosing NAFLD is a new direction of current research has found that hyperferritinemia, with or without increased iron concentration in the liver, is a common manifestation of NAFLD [10]. Iron metabolism abnormalities have gained increasing attention in the pathogenesis of NAFLD. Serum ferritin also acts as an acute-phase reactant and marker of systemic inflammation.

MATERIALS AND METHODS

Study Population and Clinical Assessment

This observational, cross-sectional study included NAFLD patients aged 18–60 years attending Mahatma Gandhi Medical College & Hospital, Jaipur, in collaboration with the Departments of Gastroenterology and Medicine.

Fifty adult patients diagnosed with NAFLD based on ultrasonography, minimal or no alcohol consumption (<20 g/day for women, <30 g/day for men) and willingness to participate in the study were included in the study. Patients with alcoholic liver disease, Viral hepatitis (HBV, HCV), autoimmune liver disease, hemochromatosis or other iron overload disorders and chronic inflammatory or malignant diseases were excluded. Fifty age and gender matched healthy adults constituted the Control group.

Demographic data, BMI and metabolic parameters were recorded. Blood samples were analyzed for Serum ferritin, ALT, AST, GGT, Albumin, Hba1c, Lipid profile by Chemiluminescence method using Vitros diagnostic reagents.

Statistical Analysis

Data were analyzed using appropriate statistical software. Continuous variables were expressed as mean ± SD. Correlation between serum ferritin and clinical parameters was assessed using Pearson correlation tests. A p-value <0.05 was considered statistically significant.

RESULTS

The present study has been conducted to evaluate ferritin level in NAFLD patients. A total number of 50 patients with NAFLD were included in the study. The study consisted of 50 subjects in Group-1(cases of NAFLD) and 50 subjects in Group-II (control). Among the NAFLD patients 68% were male and 32% were female.

Results Table

Table 1.Anthropometric and Biochemical Parameters

Parameter	Control (Mean ± SD)	Case (Mean ± SD)	p-value
BMI (kg/m²)	24.43 ± 5.52	33.98 ± 6.80	< 0.001
Total Cholesterol (mg/dL)	165.63 ± 40.34	189.52 ± 42.71	0.005
HDL (mg/dL)	59.58 ± 14.44	43.79 ± 14.35	< 0.001
Triglycerides (mg/dL)	90.76 ± 57.54	143.85 ± 138.82	0.014
HbA1c (%)	5.47 ± 0.66	5.99 ± 1.14	0.006
AST (IU/L)	20.57 ± 10.81	29.93 ± 9.31	< 0.001
ALT (IU/L)	19.27 ± 14.80	28.53 ± 16.24	0.004
GGT (IU/L)	23.11 ± 25.09	34.66 ± 33.61	0.054
Serum Albumin (g/dL)	40.92 ± 3.17	36.97 ± 3.07	< 0.001
Ferritin (ng/mL)	131.90 ± 142.36	178.41 ± 161.36	0.03

Values are expressed as mean ± standard deviation. Comparison between groups was performed using independent sample t-test. A p-value < 0.05 was considered statistically significant.

Figures

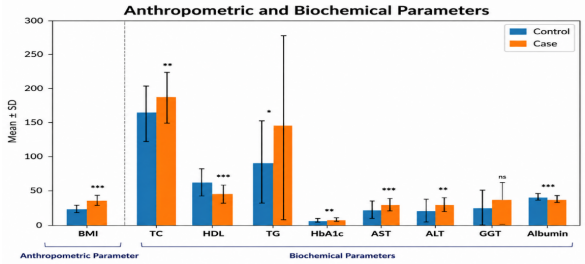

Figure 1. Comparison of Anthropometric and biochemical parameters between controls and NAFLD cases. *p < 0.05, **p < 0.01, ***p < 0.001, ns = not significant.

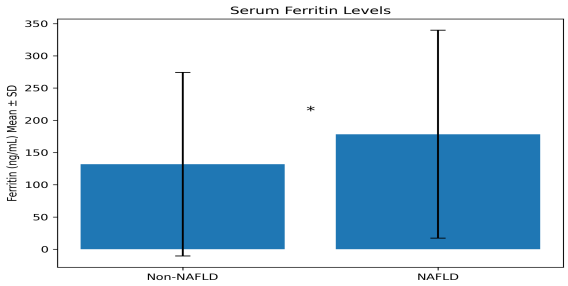

Figure 2. Comparison of serum ferritin levels between non-NAFLD and NAFLD subjects. *p < 0.05.

Serum ferritin levels increased progressively with NAFLD severity. Patients with moderate and severe NAFLD demonstrated significantly higher ferritin levels compared to those with mild disease ($p < 0.05$).

Anthropometric Characteristics and Biochemical Parameters

Patients with NAFLD were significantly had a higher BMI than controls ($p < 0.001$), indicating an association of increasing age and obesity with NAFLD**.** NAFLD patients showed significantly higher total cholesterol, triglycerides, HbA1c, AST, and ALT levels with significantly lower HDL and serum albumin levels compared to controls. GGT levels were higher but not statistically significant. These findings indicate dyslipidemia, altered glycemic status, and hepatic dysfunction in NAFLD patients**.** Serum ferritin levels were significantly higher in NAFLD patients compared to controls ($p = 0.03$), suggesting increased iron stores and inflammatory activity.

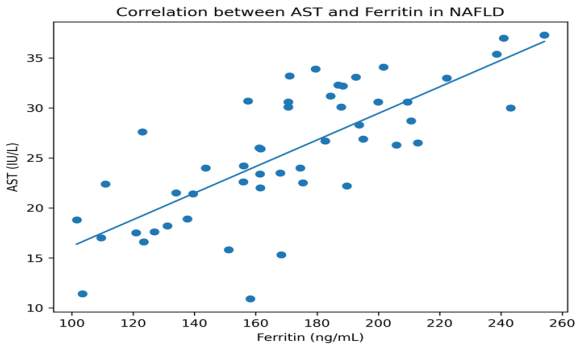

Figure 1: Scatter plot depicting the correlation between serum ferritin and AST levels in NAFLD patients (n = 50). A strong positive correlation was observed (r = 0.75, $p < 0.001$). The solid line represents the linear regression line and the shaded area indicates the 95% confidence interval.

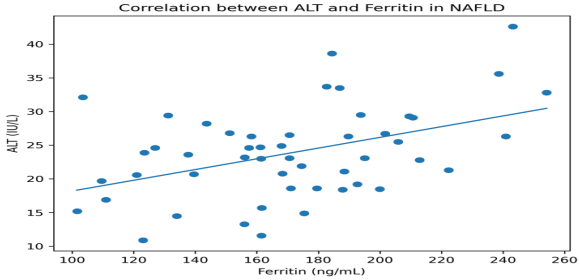

Figure 2: Scatter plot depicting the correlation between serum ferritin and ALT levels in NAFLD patients (n = 50). A moderate positive correlation was observed (r = 0.44, $p = 0.001$). The solid line represents the linear regression line and the shaded area indicates the 95% confidence interval.

Correlation Analysis Between Serum Ferritin and Liver Enzymes in NAFLD

Correlation analysis was performed to evaluate the association between serum ferritin levels and liver transaminases in patients with non-alcoholic fatty liver disease (NAFLD) (n = 50). Pearson's correlation coefficient was used for analysis.

A **strong positive correlation** was observed between serum ferritin and aspartate aminotransferase (AST) levels (**r = 0.75**, $p < 0.001$). Scatter plot analysis demonstrated a clear linear upward trend, with the regression line indicating a significant increase in AST levels with rising ferritin concentrations. The 95% confidence interval bands closely followed the regression line, suggesting a robust and consistent association across the observed range of ferritin values (Figure 1).

A **moderate positive correlation** was also identified between serum ferritin and alanine aminotransferase (ALT) levels (**r = 0.44**, $p = 0.001$). The scatter plot showed a positive linear relationship, though with greater variability compared to AST. The regression line and accompanying 95% confidence bands confirmed the statistical significance of this association (Figure 2).

Overall, these findings indicate that elevated serum ferritin levels are significantly associated with increased hepatic transaminase levels in NAFLD patients, supporting the role of iron overload and inflammation in hepatocellular injury.

DISCUSSION

The study demonstrated that elevated serum ferritin is associated with increased disease severity, metabolic abnormalities, and risk of fibrosis in NAFLD, supporting its potential role as a marker of disease activity.

Ferritin elevation in NAFLD likely reflects a combination of hepatic iron accumulation, chronic low-grade systemic inflammation, and insulin resistance. Iron-induced oxidative stress has been shown to promote lipid peroxidation, mitochondrial dysfunction, and activation of hepatic stellate cells, thereby contributing to hepatocellular injury and progression from simple steatosis to metabolic dysfunction non alcoholic steatohepatitis (MASH) and fibrosis [8–10]. Experimental studies suggest that excess hepatic iron exacerbates oxidative stress and inflammatory signaling pathways, accelerating NAFLD progression [11,12].

In the present study, Body mass index (BMI) was markedly higher in NAFLD cases (33.98 ± 6.80 kg/m²) compared to controls (24.43 ± 5.52 kg/m²), and this difference was highly significant ($p < 0.0001$). Obesity plays a central role in NAFLD pathogenesis by increasing free fatty acid flux to the liver, inducing insulin resistance, and promoting hepatic triglyceride accumulation. These findings are consistent with large epidemiological and longitudinal studies identifying

obesity as a key determinant of NAFLD development and progression [13,14].

Dyslipidemia was a prominent metabolic abnormality among NAFLD patients. Mean serum cholesterol and triglyceride levels were significantly higher in cases than in healthy controls, reflecting an atherogenic lipid profile. This pattern is characteristic of insulin resistance and altered hepatic lipid metabolism and has been consistently reported in NAFLD populations [15,16]. Given that cardiovascular disease is the leading cause of mortality in NAFLD, these findings highlight the importance of early identification and management of metabolic risk factors in this population [16,17].

Glycemic control, as assessed by HbA1c, was significantly worse in NAFLD patients, supporting the close bidirectional relationship between NAFLD and insulin resistance. NAFLD is now recognized as both a hepatic manifestation of metabolic syndrome and an independent risk factor for the development of type 2 diabetes mellitus. Meta-analyses and prospective studies have demonstrated that NAFLD confers a significantly increased risk of incident diabetes, which is in agreement with the findings of the present study [18,19].

Serum aminotransferases (AST and ALT) were significantly elevated in NAFLD cases, indicating hepatocellular injury. Although aminotransferase levels may remain normal in a subset of NAFLD patients, elevations are commonly observed in individuals with steatohepatitis and multiple metabolic risk factors [20]. Gamma-glutamyl transferase (GGT) showed borderline elevation, which may reflect oxidative stress and hepatic fat accumulation. GGT has been proposed as a surrogate marker of oxidative stress and cardiometabolic risk in NAFLD [20,21]. Additionally, the significantly reduced serum albumin levels observed in NAFLD patients may suggest early impairment of hepatic synthetic function or a chronic inflammatory state, both of which have been associated with advanced fibrosis and poorer prognosis [22].

A key finding of this study is the significantly higher mean serum ferritin levels observed in NAFLD patients (178 ± 161.36 ng/ml) compared to healthy controls (131.40 ± 142.36 ng/ml; $p < 0.05$). Elevated ferritin in NAFLD is generally considered a marker of hepatic inflammation, insulin resistance, and altered iron metabolism rather than true iron overload. Several studies have demonstrated that hyperferritinemia is associated with increased necroinflammation, fibrosis severity and adverse metabolic outcomes in NAFLD patients [23-26].

Zelber-Sagi et al. reported that hyperinsulinemia and hepatic steatosis are major determinants of serum ferritin levels in NAFLD [23]. Kim et al. demonstrated that elevated ferritin levels independently predicted incident NAFLD in healthy individuals [24]. Manousou et al. further showed that serum ferritin was a strong discriminant marker for both fibrosis and inflammation in histologically proven NAFLD [25].

Kowdley et al. reported that elevated serum ferritin is an independent predictor of histological severity and advanced fibrosis in NAFLD [26]. These findings underscore the role of ferritin as a marker of disease activity rather than a simple indicator of iron overload.

Conversely, some studies have cautioned against the use of serum ferritin as a standalone diagnostic marker for fibrosis. Angulo et al. and Yoneda et al. reported that ferritin alone has limited diagnostic accuracy for staging fibrosis in NAFLD, emphasizing the need for its interpretation in conjunction with other clinical and non-invasive markers [27,28]. These observations suggest that ferritin is best utilized as part of a composite risk stratification strategy rather than as an isolated biomarker.

Population-based studies have further highlighted the influence of demographic factors on the ferritin–NAFLD relationship. Yang et al. reported significant variations in the association between ferritin and NAFLD based on age and sex [29]. Du et al., in a meta-analysis, found higher ferritin levels in MASH compared to non-MASH patients, although heterogeneity across studies was noted [30]. Jeong et al. demonstrated a dose–response relationship between ferritin quartiles and NAFLD severity, particularly in men [31]. Recent studies have also suggested sex-specific associations, with elevated ferritin being more strongly associated with NAFLD in postmenopausal women [32,33].

Overall, the findings of the present study reinforce the concept of NAFLD as a complex metabolic and inflammatory disorder. The coexistence of obesity, dyslipidemia, impaired glycemic control, hepatic dysfunction, and elevated ferritin highlights the importance of comprehensive metabolic evaluation in NAFLD patients. Identification of elevated ferritin may help recognize individuals at higher risk for progressive liver disease and cardiometabolic complications.

The use of serum ferritin as a non-invasive, inexpensive, and widely available biomarker may aid in identifying high-risk NAFLD patients who require closer monitoring or more aggressive lifestyle and pharmacological interventions.

CONCLUSION

Serum ferritin levels are significantly elevated in patients with NAFLD and are associated with adverse metabolic profiles and markers of disease severity. Elevated ferritin may serve as a valuable, cost-effective, non-invasive biomarker for identifying NAFLD patients at increased risk of hepatic inflammation and fibrosis. Incorporation of ferritin into routine clinical assessment may improve risk

stratification and guide management strategies in NAFLD.

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