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Original Article

Correlation of Serum Ferritin with Child-Pugh and MELD Scores in Decompensated Liver Cirrhosis

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Abstract

Decompensated cirrhosis is associated with substantial morbidity and mortality. The Child- Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores are established tools for severity assessment, but inexpensive adjunctive biomarkers may be useful in resource-limited settings. Serum ferritin, an iron-storage protein and acute-phase reactant, may reflect inflammation, hepatocellular injury, and worsening liver disease.This cross-sectional study was conducted at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, from April 2021 to March 2022. A total of 33 patients with decompensated liver cirrhosis were enrolled consecutively. Patients were categorized by ferritin level (<200, 200-400, and >400 ng/mL). Correlation was assessed using Spearman's rank test. Multivariable analyses were performed using binary logistic regression for high MELD score (>15) and multiple linear regression for continuous MELD score, adjusting for age, sex, serum bilirubin, and serum albumin. A p-value <0.05 was considered statistically significant. The mean age was 52.7 ± 14.07 years; 63.6% were 41-60 years old, and 72.7% were male. Hepatitis B virus was the commonest etiology (45.5%), followed by cryptogenic cirrhosis (24.2%) and hepatitis C virus (21.2%). Ascites was present in 97.0%, jaundice in 48.5%, hepatic encephalopathy in 33.3%, and anemia in 57.6%. Serum ferritin correlated strongly with CTP score ($r=0.70$, $r^2=0.49$, $p<0.001$) and moderately with MELD score ($r=0.50$, $r^2=0.25$, $p<0.001$). Across ferritin categories, grade 3 ascites increased from 12.5% to 25.0% and 70.6% (p=0.014), prothrombin time from 13 to 15 and 21 seconds (p=0.024), MELD score from 13.75 ± 5.85 to 20.13 ± 6.40 and 22.71 ± 5.03 (p=0.003), and CTP score from 8.00 ± 1.19 to 9.63 ± 1.85 and 11.18 ± 1.70 (p<0.001). In multivariable logistic regression analysis, serum ferritin >200 ng/mL was independently associated with a high MELD score (adjusted OR 5.92; 95% CI: 1.03–33.98; p=0.046). Serum bilirubin was also independently associated with a higher MELD score (p=0.037), while serum albumin showed an inverse association (p=0.031). In multiple linear regression, serum ferritin remained a significant independent predictor of MELD score ($\beta = 0.38$; p=0.021), with the model explaining 42% of the variance (adjusted $R^2 = 0.42$). Serum ferritin was significantly associated with CTP and MELD scores and with clinical markers of decompensation. It may serve as an accessible supplementary biomarker for severity assessment in decompensated cirrhosis, but its nonspecific inflammatory nature requires interpretation alongside established scoring systems.

Keywords: *Serum ferritin; decompensated liver cirrhosis; child-Pugh score; MELD score; biomarkers; liver disease severity.*

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INTRODUCTION

Liver cirrhosis is the advanced stage of chronic liver injury, characterized by progressive fibrosis, architectural distortion, regenerative nodule formation, and gradual loss of functional hepatic reserve. It remains a major contributor to morbidity, mortality, repeated hospital admission, and health-care expenditure worldwide. Chronic liver disease is also an important public health problem in Bangladesh, where viral hepatitis, metabolic liver disease, alcohol- related liver disease, and cryptogenic causes continue to contribute to the burden of cirrhosis and its complications.[1,2,3,14]

The transition from compensated to decompensated cirrhosis marks a clinically important turning point.

Decompensation is usually manifested by ascites, jaundice, hepatic encephalopathy, variceal bleeding, coagulopathy, renal dysfunction, or severe hypoalbuminemia. Once these features develop, patients are at higher risk of short-term mortality, infection, hospitalization, and decline in quality of life. Accurate grading of disease severity is therefore central to clinical decision-making, prognostic counselling, referral, transplant prioritization, where applicable, and follow-up planning.[6,16,18,19]

The Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores are among the most widely used tools for assessing the severity and prognosis of cirrhosis. The CTP score integrates serum bilirubin, albumin, prothrombin time or international normalized ratio, ascites, and encephalopathy, thereby combining biochemical and clinical information. It is simple and clinically familiar, but its ascites and encephalopathy components are partly subjective. The MELD score, based on bilirubin, INR, and creatinine, provides a more objective biochemical estimate of short-term mortality risk and has been widely validated in advanced liver disease.[4,5,15]

Despite their utility, both scoring systems have limitations in everyday practice. CTP may vary according to clinical interpretation, while MELD may not fully capture systemic inflammation, immune dysfunction, iron overload, nutritional status, or other biological processes that influence decompensation. In resource-limited settings, a readily available, inexpensive, and interpretable supplementary biomarker could be helpful, particularly when it reflects inflammatory activity or hepatocellular injury beyond conventional severity scores. Such a marker would not replace clinical scoring, but it could help identify patients who need closer observation, more frequent laboratory follow-up, or earlier referral for specialist care.[7,8,18,19]

Serum ferritin is traditionally used as a marker of body iron stores, but it is also an acute-phase reactant. Ferritin may rise in response to inflammation, oxidative stress, hepatocellular necrosis, immune activation, infection, alcohol-related injury, and altered iron metabolism. The liver has a central role in iron homeostasis; therefore, chronic liver disease can disturb ferritin concentrations through several overlapping mechanisms. Raised ferritin has been reported in chronic hepatitis, alcoholic liver disease, metabolic liver disease, hemochromatosis, and advanced cirrhosis.[7,8,11]

Previous studies have suggested that higher serum ferritin may be associated with more advanced cirrhosis, higher CTP or MELD scores, and adverse clinical outcomes. Sungkar et al. reported serum ferritin as a biomarker related to CTP score, while Umer et al. and Maiwall et al. described ferritin as a predictor of mortality or poor outcome in cirrhosis. However, the strength and independence of this association vary across populations, and ferritin remains nonspecific because it can be increased by inflammation and other comorbid conditions.[9,10,12,17]

Data on serum ferritin in Bangladeshi patients with decompensated cirrhosis remain limited. Local evidence is important because the etiological distribution, nutritional background, infection burden, access to advanced diagnostics, and timing of hospital presentation may differ from other settings. The present study was therefore conducted to evaluate the correlation of serum ferritin with CTP and MELD scores among patients with decompensated cirrhosis and to explore whether higher ferritin levels are associated with clinical and laboratory markers of more severe disease. By linking ferritin with demographic profile, complications, grouped severity parameters, and regression-based outcomes, this study aimed to clarify whether ferritin may be useful as a practical adjunct in the routine assessment of decompensated cirrhosis. The focus was not to propose ferritin as a substitute for established scores, but to assess whether it adds clinically interpretable information when used alongside them.

MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Department of Gastroenterology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, from April 2021 to March 2022. The study evaluated the relationship between serum ferritin concentration and liver disease severity among patients with decompensated cirrhosis, using the Child-Turcotte-Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scoring systems.

A total of 33 patients were enrolled consecutively according to predefined eligibility criteria. Adults aged 18 years or above with decompensated liver cirrhosis diagnosed by clinical evaluation, laboratory assessment, and ultrasonographic evidence were included. Decompensation was defined by the presence of one or more features such as ascites, jaundice, hepatic encephalopathy, gastrointestinal bleeding, and/or hypoalbuminemia.

Patients were excluded if they had acute liver failure, hepatocellular carcinoma, severe infection, pregnancy, medical emergencies requiring immediate intervention, active heart failure, chronic kidney failure, diabetes mellitus, recent blood transfusion, hemochromatosis, or thalassemia. Written informed consent was obtained from all participants before enrollment.

The sample size was calculated using a correlation coefficient of 0.48 reported in previous work by Sungkar et al. on serum ferritin and CTP score in decompensated cirrhosis. With a 5% level of significance and 80% power, the required sample size was 33 patients. The study was approved by the Institutional Review Board of BSMMU (Reference: BSMMU/2021/1935) and was conducted in accordance with the Declaration of Helsinki.

Clinical history and examination findings were recorded using a structured data collection form. Key clinical variables included ascites, jaundice, hepatic encephalopathy, gastrointestinal bleeding, anemia, and etiology of cirrhosis. Laboratory investigations included serum ferritin, complete blood count, serum bilirubin, serum albumin, serum creatinine, prothrombin time, international normalized ratio, and serum electrolytes. Abdominal ultrasonography was used to document ascites, and upper gastrointestinal endoscopy was used where indicated to assess esophageal varices.

CTP score was calculated from bilirubin, albumin, prothrombin time or INR, ascites, and encephalopathy. MELD score was calculated using the standard formula: MELD = 3.8 × ln(bilirubin) + 11.2 × ln(INR) + 9.6 × ln(creatinine) + 6.4. Serum ferritin was analyzed both as a continuous variable and by clinically relevant categories: <200 ng/mL, 200-400 ng/mL, and >400 ng/mL. Normal ferritin limits were considered 15-300 ng/mL for males and 15-150 ng/mL for females.

Data were analyzed using SPSS version 22. Continuous variables were summarized as mean ± standard deviation or median with range, as appropriate, while categorical variables were expressed as frequency and percentage. Spearman's rank correlation coefficient was used to assess the relationship of serum ferritin with CTP and MELD scores. Differences across ferritin categories were assessed using ANOVA or the Kruskal-Wallis test, according to data distribution. A p-value <0.05 was considered statistically significant.

To evaluate the independent association between ferritin and liver disease severity, binary logistic regression was performed with a high MELD score (>15) as the dependent variable. Covariates included serum ferritin category (>200 ng/mL versus ≤200 ng/mL), age, sex, serum bilirubin, and serum albumin. Adjusted odds ratios (AORs) with 95% confidence intervals were calculated. Model fitness was assessed using the Hosmer-Lemeshow goodness-of-fit test, and multicollinearity was evaluated using the variance inflation factor. Multiple linear regression was also performed using the MELD score as a continuous dependent variable.

RESULTS

A total of 33 patients with decompensated liver cirrhosis were included in the analysis. Serum ferritin levels were evaluated in relation to demographic characteristics, clinical manifestations, laboratory findings, CTP score, MELD score, and regression-based severity outcomes.

Table I shows the demographic characteristics and etiology of cirrhosis. The mean age was 52.7 ± 14.07 years, and 63.6% were 41-60 years old. Male patients were 72.7%, with a male-to-female ratio of approximately 2.7:1. Hepatitis B virus was 45.5%, followed by cryptogenic cirrhosis (24.2%), hepatitis C virus (21.2%), and other causes (9.1%).

Table I: Distribution of demographic characteristics and etiology of liver cirrhosis in the study population (n=33)

Variables	Frequency (n)	Percentage (%)
Mean age (years ± SD)	52.7 ± 14.07	
The majority of the age group (41–60 years)	21	63.6
Sex		
Male	24	73
Female	9	27
Etiology of cirrhosis		
Hepatitis B virus (HBV)	15	45.5
Hepatitis C virus (HCV)	7	21.2
Cryptogenic	8	24.2
Others	3	9.1

Age is expressed as mean ± standard deviation (SD).

Clinical and Laboratory findings of the study population

Table II summarizes the clinical and laboratory profile. Ascites was present in 97.0%, jaundice 48.5%, hepatic encephalopathy in 33.3%, and anemia in 57.6%. Ferritin level was >400 ng/mL in 63.6%. Mean hemoglobin was 9.59 ± 1.68 g/dL, serum albumin 20.94 ± 7.89 g/L, sodium 133.6 ± 5.4 mmol/L, serum bilirubin 5.09 ± 3.25 mg/dL, and prothrombin time 17.87 ± 5.23 seconds.

Table II Clinical and Laboratory findings of the study population (n=33)

Clinical feature	Frequency (n)	Percentage (%)
Ascites	32	97.0
Jaundice	16	48.5
Hepatic encephalopathy	11	33.3
Anemia	19	57.6
Ferritin levels above 400 ng/mL	22	63.6
Mean hemoglobin level	9.59 ± 1.68 g/dL	
Mean serum albumin level	20.94 ± 7.89 g/L	
Mean serum sodium level	133.6 ± 5.4 mmol/L	
Mean serum bilirubin Level	5.09 ± 3.25 mg/dL	
Mean prothrombin time	17.87 ± 5.23 seconds	

Association of Elevated Serum Ferritin with MELD Score

Table III shows the association between elevated serum ferritin and high MELD score. Among patients with MELD score>15, 84.6% had ferritin >200 ng/mL, whereas 15.4% had ferritin ≤200 ng/mL. Ferritin >200 ng/mL was associated with significantly higher odds of MELD >15.

Table III: Association between elevated serum ferritin and high MELD score

Serum ferritin (ng/mL)	MELD >15 n (%)	MELD ≤15 n (%)	Odds ratio (95% CI)	p-value
>200	11 (84.6)	9 (42.9)	7.33 (1.17- 46.05)	0.022
≤200	2 (15.4)	12 (57.1)	Reference	-

Odds ratio calculated using binary logistic regression. p<0.05 is considered significant.

Serum Ferritin Levels and Correlation with CTP and MELD Scores

Table IV demonstrates that serum ferritin was significantly correlated with both severity scores. The correlation with CTP score was strong, while the correlation with MELD score was moderate. Thus, serum ferritin explained approximately 49% of the variation in CTP score and 25% of the variation in MELD score.

Table IV: Correlation between serum ferritin and liver severity scores (CTP and MELD Scores)

Parameters	(r)	(r^2)	p-value
Serum ferritin vs CTP score	0.70	0.49	<0.001
Serum ferritin vs MELD score	0.50	0.25	<0.001

Spearman's rank correlation test applied. A p-value <0.05 was considered statistically significant. Correlation coefficient (r), Coefficient of determination (r^2).

Scatter Plot Analysis

Scatter plot analysis supported these statistical findings. Figure 1A shows a clear upward relationship between serum ferritin and CTP score, while Figure 1B shows a positive but more dispersed relationship between serum ferritin and MELD score. Regression lines are displayed for visual trend assessment, with R^2 values shown in each panel.

Figure 1A displays that higher ferritin levels corresponded to higher CTP scores, indicating that ferritin was closely linked with clinical severity. The relatively tighter clustering of points is consistent with the strong correlation observed between ferritin and CTP score.

In Figure 1B, ferritin also increased with MELD score, although the wider spread of points suggests that bilirubin, INR, creatinine, and other biochemical factors contribute substantially to MELD variability.

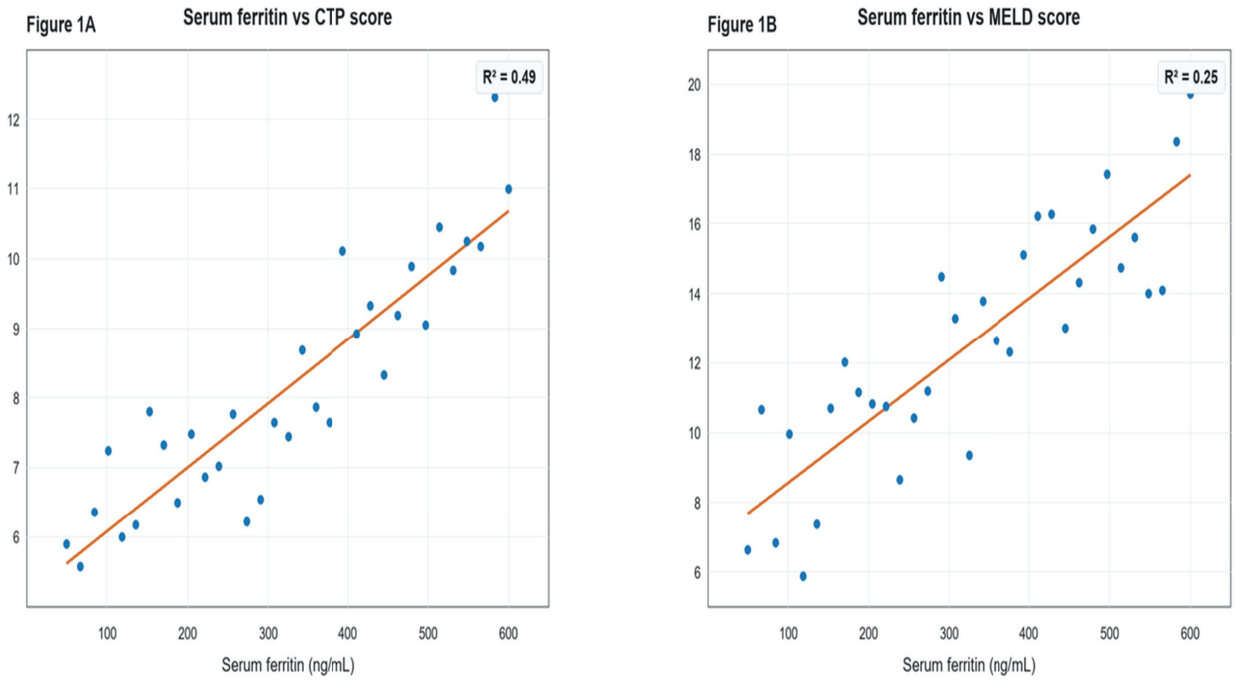

Figure 1. Scatter plots of serum ferritin with liver disease severity scores. Figure 1A: serum ferritin versus Child-Pugh (CTP) score (R^2=0.49). Figure 1B: serum ferritin versus MELD score (R^2=0.25).

Clinical and Laboratory Differences Based on Serum Ferritin Levels

Table V presents the clinical and laboratory parameters according to serum ferritin levels. Patients were stratified into three ferritin categories. Worsening clinical and laboratory parameters were observed with increasing ferritin. Grade 3 ascites increased from 12.5% to 25.0% and 70.6% across the <200, 200-400, and >400 ng/mL groups (p=0.014). Median prothrombin time increased from 13 seconds to 15 seconds and 21 seconds (p=0.024). Mean MELD score rose from 13.75 ± 5.85 to 20.13 ± 6.40 and 22.71 ± 5.03 (p=0.003), while mean CTP score rose from 8.00 ± 1.19 to 9.63 ± 1.85 and 11.18 ± 1.70 (p<0.001).

Table V: Clinical and laboratory parameters according to serum ferritin levels

Variables	Ferritin <200 ng/mL	Ferritin 200-400 ng/mL	Ferritin >400 ng/mL	p-value
Grade 3 ascites (%)	12.5	25.0	70.6	0.014
Prothrombin time (Median)	13 (11–19) Sec.	15 (12–33) Sec.	21 (13–29) Sec.	0.024
MELD score (Mean ± SD)	13.75 ± 5.85	20.13 ± 6.40	22.71 ± 5.03	0.003
CTP score (Mean ± SD)	8.00 ± 1.19	9.63 ± 1.85	11.18 ± 1.70	<0.001

Continuous variables expressed as mean ± SD or median (range). Kruskal-Wallis/ANOVA used as appropriate.

Figure 2 presents a grouped bar chart comparing MELD score (mean ± SD), CTP score (mean ± SD), grade 3 ascites (%), and prothrombin time (median seconds) across ferritin categories. The figure illustrates a consistent stepwise increase in disease severity with increasing ferritin level.

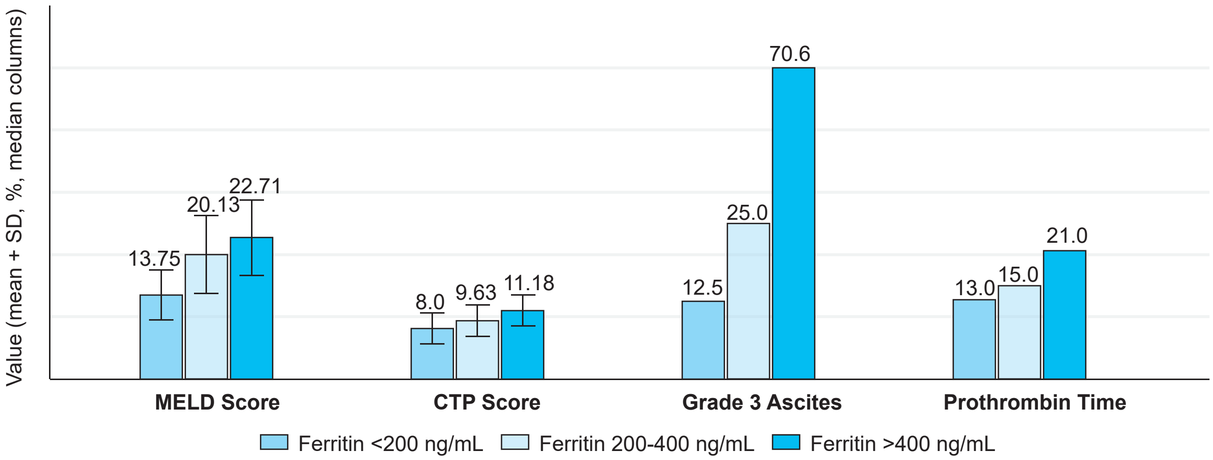

Figure 2: Unified grouped bar diagram comparing MELD score (mean ± SD), Child-Pugh (CTP) score (mean ± SD), grade 3 ascites (%), and prothrombin time (median seconds) across serum ferritin categories. Error bars indicate SD.

Multivariable Logistic Analysis of Factors Associated with High MELD Score

Binary logistic regression was performed with MELD score >15 as the dependent variable. Adjusted odds ratios (AORs) with 95% confidence intervals were calculated after controlling for age, sex, serum bilirubin, and serum albumin. Model calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test, and $p<0.05$ was considered significant.

Table VI shows that serum ferritin >200 ng/mL remained independently associated with high MELD score after adjustment for age, sex, serum bilirubin, and serum albumin. Serum bilirubin was also positively associated with high MELD score, whereas serum albumin showed a protective association. Age and sex were not statistically significant, and the Hosmer-Lemeshow test indicated acceptable model fit ($p>0.05$).

Table VI: Multivariable logistic regression analysis of factors associated with high MELD score (>15)

Variables	Adjusted OR (95% CI)	p-value
Serum ferritin >200 ng/mL	5.92 (1.03–33.98)	0.046
Age (years)	1.02 (0.96–1.08)	0.51
Male sex	1.34 (0.32–5.61)	0.69
Serum bilirubin (mg/dL)	1.41 (1.02–1.96)	0.037
Serum albumin (g/dL)	0.71 (0.52–0.97)	0.031

Binary logistic regression model with MELD score >15 as the dependent variable.

Adjusted for clinically relevant covariates. OR = odds ratio; CI = confidence interval.

Multiple Linear Regression Analysis

Multiple linear regression analysis was performed with the MELD score as a continuous dependent variable. Regression coefficients (β), standard errors, and p-values were reported after adjustment for age, serum bilirubin, and serum albumin.

Table VII shows that log serum ferritin was an independent predictor of MELD score. Serum bilirubin was positively associated with MELD score, while serum albumin was negatively associated. Age was not significant. The model explained a moderate proportion of MELD variability (adjusted $R^2=0.42$).

Table VII: Multiple linear regression analysis for predictors of MELD score

Variables	β coefficient	Standard error (SE)	p-value
Serum ferritin (log)	0.38	0.15	0.021
Serum bilirubin	0.44	0.17	0.014
Serum albumin	-0.36	0.14	0.019
Age	0.09	0.11	0.41

Multiple linear regression model with MELD score as the dependent variable. Adjusted $R^2 = 0.42$.

DISCUSSION

This study evaluated the association between serum ferritin and established liver severity scores among 33 patients with decompensated cirrhosis. The study population was predominantly middle-aged, with a mean age of 52.7 ±

14.07 years; 21 patients (63.6%) were 41-60 years old, and 24 (72.7%) were male. Hepatitis B virus was the leading etiology (45.5%), followed by cryptogenic cirrhosis (24.2%), hepatitis C virus (21.2%), and other causes (9.1%). This pattern is clinically relevant for Bangladesh, where viral hepatitis remains an important contributor to chronic liver disease.[1,14]

The clinical profile reflected advanced decompensation. Ascites was present in 32 patients (97.0%), jaundice in 16 (48.5%), hepatic encephalopathy in 11 (33.3%), and anemia in 19 (57.6%). Laboratory findings also suggested substantial hepatic dysfunction, including low mean albumin (20.94 ± 7.89 g/L), hyponatremia tendency (133.6 ± 5.4 mmol/L), raised bilirubin (5.09 ± 3.25 mg/dL), anemia (hemoglobin 9.59 ± 1.68 g/dL), and prolonged prothrombin time (17.87 ± 5.23 seconds), supporting a clinically meaningful decompensated cirrhosis cohort.[6,16]

Serum ferritin showed a strong positive correlation with CTP score ($r=0.70$, $r^2=0.49$, $p<0.001$) and a moderate positive correlation with MELD score ($r=0.50$, $r^2=0.25$, $p<0.001$). These results indicate that increasing ferritin levels were associated with worsening liver disease severity, particularly with the CTP score. This may reflect the clinical components of CTP, such as ascites and encephalopathy, which can parallel inflammation and hepatocellular injury. MELD is driven mainly by bilirubin, INR, and creatinine; therefore, ferritin explained a smaller but still significant proportion of MELD variability. [4,5,15]

The graded analysis across ferritin categories further supports this interpretation. Grade 3 ascites increased from 12.5% in the ferritin <200 ng/mL group to 25.0% in the 200-400 ng/mL group and 70.6% in the >400 ng/mL group (p=0.014). Prothrombin time also increased from a median of 13 seconds to 15 seconds and 21 seconds across the same categories (p=0.024). Mean MELD score rose from 13.75 ± 5.85 to 20.13 ± 6.40 and 22.71 ± 5.03 (p=0.003), while mean CTP score rose from 8.00 ± 1.19 to 9.63 ± 1.85 and 11.18 ± 1.70 (p<0.001). These trends suggest a consistent relationship between higher ferritin and both clinical and biochemical severity.

The association with high MELD score was also observed in categorical and adjusted analyses. Ferritin >200 ng/mL was associated with MELD >15 in unadjusted analysis (OR 7.33; 95% CI: 1.17-46.05; p=0.022) and remained significant after adjustment for age, sex, bilirubin, and albumin (AOR 5.92; 95% CI: 1.03-33.98; p=0.046). Bilirubin was positively associated with high MELD (AOR 1.41; p=0.037), while albumin showed a protective association (AOR 0.71; p=0.031). Linear regression similarly showed that log ferritin independently predicted MELD score ($\beta=0.38$, $p=0.021$), with an adjusted R^2 of 0.42.

These findings are consistent with previous reports describing ferritin as a marker associated with cirrhosis severity and adverse outcomes. Sungkar et al. reported ferritin as a biomarker related to CTP score, while Umer et al., Maiwall et al., and Oikonomou et al. linked elevated ferritin with mortality or worse cirrhosis outcomes. Biologically, this is plausible because ferritin reflects iron storage as well as inflammatory and oxidative stress responses, immune dysfunction, and liver injury.[7,8,9,10,11,12,17,18,19]

However, ferritin should not be interpreted as a stand-alone severity marker. It is nonspecific and may increase with infection, inflammation, metabolic disease, alcohol-related injury, malignancy, and iron overload states.

LIMITATIONS OF THE STUDY:

The small sample size, single-center design, and cross-sectional nature of this study limit causal inference and generalizability. The adjusted findings suggest that the ferritin-severity relationship is not solely explained by conventional biochemical parameters, but the wide confidence interval indicates imprecision. Larger multicenter longitudinal studies are needed to validate ferritin as an adjunctive biomarker and define clinically useful cut-off values.

CONCLUSION

Serum ferritin was significantly associated with liver disease severity among patients with decompensated cirrhosis. It correlated strongly with CTP score and moderately with MELD score, and higher ferritin categories were associated with grade 3 ascites, prolonged prothrombin time, and higher MELD and CTP scores. Ferritin >200 ng/mL remained independently associated with high MELD score after adjustment for clinically relevant covariates. These findings suggest that serum ferritin may serve as an accessible supplementary biomarker for severity assessment, particularly in resource-limited settings. Because ferritin is nonspecific, it should be interpreted alongside established scores and clinical context rather than used as a replacement for CTP or MELD.

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