

Serum Iron indices and C-Reactive protein in Alcoholic Liver Disease

Authors:

Davina Hijam^{1*}, Mairembam Jamuna Devi², Ng. Arunkumar Singh³, Nungshitombi Ngangbam⁴,
Bidyarani Haobam⁵, Karam Romeo⁶, Babina Sarangthem⁷

¹⁻⁵Department of Biochemistry, Regional Institute of Medical Sciences, Imphal, India.

⁶Department of Medicine, Regional Institute of Medical Sciences, Imphal, India.

⁷Department of Pathology, Regional Institute of Medical Sciences, Imphal, India, 795004.

*Corresponding Author:

Dr. Davina Hijam, Professor, Department of Biochemistry, Regional Institute of Medical Sciences, Imphal, Manipur

Email ID: davina_hijam@yahoo.co.in

<https://doi.org/10.5281/zenodo.21453057>

Received: 11 June 2026, Accepted: 19 June 2026, Online First: 20 July 2026

ABSTRACT

Introduction: Alcoholic liver disease (ALD) is a leading cause of chronic liver morbidity in India, driven by metabolic dysfunction and systemic inflammation. Chronic alcohol intake suppresses hepatic hepcidin, causing unregulated iron absorption and oxidative stress while C-reactive protein (CRP) serves as a critical marker of systemic inflammation. **Objectives:** To estimate and compare iron indices and C-reactive protein levels in individuals with and without ALD and to determine their relationship with disease severity. **Materials and Methods:** An analytical cross-sectional study was conducted in the Department of Biochemistry in collaboration with Department of Medicine and Multi-Disciplinary Research Unit (MRU), Regional Institute of Medical Sciences (RIMS), Imphal, Manipur from February 2024 to February 2026. It included 60 diagnosed cases of ALD and 60 individuals without ALD. Serum iron indices (iron, ferritin, total iron binding capacity, transferrin saturation), CRP, LFT, KFT, PT-INR were assessed. Data were analysed using Student's t test and Spearman's correlation. **Results:** The mean age of the study participants was 51.3 years with 96.7% males. Serum iron, ferritin, transferrin saturation and CRP were significantly higher among the ALD patients, while TIBC was significantly lower in the cases compared to controls. Serum iron, transferrin saturation and ferritin demonstrated a very strong positive correlation with ALD severity (Child-Pugh score), whereas TIBC showed a negative correlation among the cases. CRP also exhibited a significant positive correlation with Child-Pugh scores. **Conclusion:** Coexisting iron overload and systemic inflammation, evidenced by elevated ferritin and CRP, synergistically accelerate hepatic fibrosis and cirrhosis. Combined assessment of iron indices and CRP provides a robust framework for improving diagnostic accuracy and risk stratification in ALD.

Keywords: Alcoholic liver disease, Iron indices, CRP, Child-Pugh score, Biomarkers

INTRODUCTION

Alcoholic liver disease (ALD) represents one of the leading causes of chronic liver morbidity like liver fibrosis, cirrhosis and mortality worldwide.^{1,2} In 2024, ALD remains a growing public health concern in India, driven by the steady rise in alcohol consumption and it continues to rank among the leading contributors to the global burden of alcohol-related liver disease. There is a substantial increase in per capita alcohol consumption between 2000-2019, in Southeast Asia region.³ The global burden of ALD has steadily increased over the past two

decades, with an estimated 3 million annual deaths attributable to harmful alcohol use.⁴ Also, the mortality from liver disease in India accounts for 3.17% of total deaths.⁵ The spectrum of ALD ranges from simple steatosis to alcoholic steatohepatitis, progressive fibrosis and hepatocellular carcinoma (HCC).⁶ Notably, 5-15% of patients progress to cirrhosis even after alcohol abstinence, suggesting that persistent oxidative stress and inflammation are critical drivers of the disease.^{7,8} Among the numerous biochemical alterations in ALD, disturbances in iron metabolism and systemic

inflammatory activation have emerged as important contributors to disease progression. Iron is considered a risk factor in the progression of many liver diseases.^{9,10} Iron absorption from the gastrointestinal tract is increased due to the suppressive effects of alcohol on hepcidin, the master regulatory hormone of iron balance.¹¹ Hepcidin, produced by hepatocytes, normally inhibits ferroportin-mediated iron export, and reduced hepcidin levels in alcoholics, thus promote iron accumulation in the liver.¹² Furthermore, alcoholics often have coexisting increased intestinal permeability and impaired transferrin synthesis, all of which contribute to dysregulated iron indices.¹³ This accumulation of iron reflected by the elevated ferritin works synergistically with alcohol to amplify oxidative stress-creating a “second hit” that activates hepatic stellate cells and accelerates the transition to fibrosis.¹⁴ C-reactive protein (CRP) is a widely used inflammatory marker and an important biomarker in various liver diseases. The hepatocytes are the primary site of C-Reactive protein (CRP) synthesis.¹⁵ CRP is a significant acute-phase plasma protein that rises sharply and quickly in response to infection or tissue damage. It serves as a vital biomarker for the accompanying systemic inflammatory burden. Because CRP production by the hepatocytes remains relatively stable even as other liver functions decline, it is considered to be a reliable marker for assessing inflammatory burden in late-stage ALD.¹⁶ While iron indices and inflammatory markers are independently linked to liver injury, there is limited data on their combined significance within the Indian demographic, particularly in Northeast region. This study evaluates the relationship between serum iron parameters and CRP to determine their collective efficacy in predicting ALD severity and improving clinical outcomes at a tertiary care center in Manipur.

MATERIALS AND METHODS

The study was conducted in the Department of Biochemistry, in collaboration with the Department of Medicine and Multi-Disciplinary Research Unit (MRU) of Regional Institute of Medical Sciences (RIMS), Imphal from February 2024 to February 2026.

ETHICS APPROVAL

The study protocol was approved by the Research Ethics Board, Regional Institute of Medical Sciences (RIMS), Imphal, India (Ref. No.: A/REB/Prop(FP)211/138/17/2023). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

INFORMED CONSENT

Written informed consent was obtained from all study participants prior to enrolment in the study.

Inclusion Criteria

Individuals who were 18 years and above, diagnosed cases of Alcoholic Liver Disease (ALD) and age and sex matched individuals without ALD which was confirmed by history and LFT report.

Exclusion Criteria

Viral hepatitis B and C, Autoimmune hepatitis, Hepatocellular carcinoma (HCC), Haemochromatosis, GI bleeding within past 3 months, On iron supplements, T2DM, CKD, Ischaemic Heart Disease and existing active infection at any site eg. Peritonitis, UTI, pneumonia were excluded from the study.

Eligible participants with ALD were recruited consecutively from the General Medicine OPD/Wards of RIMS, Imphal. When one ALD case was recruited, one eligible participant without ALD (confirmed by history and LFT report) were also recruited conveniently from the OPD attendees in General Medicine department. After acquiring the informed consent, the blood sample of 5ml was drawn from the individuals included in the study. The sample was centrifuged at 3000 rpm and store at -20° C. Serum Iron was measured Colorimetrically using a Colorimetric Assay Kit (Cat.No:E-BC-K139-S, Elabscience). Serum ferritin, transferrin and C-Reactive protein (CRP) was estimated by ELISA using the following kits from Elabscience: Human FE(Ferritin) Sandwich ELISA Kit (Catalog No: E-EL-H0168), Human TF(Transferrin) Sandwich ELISA Kit (Catalog No: E-EL-H6028) and Human CRP Sandwich ELISA Kit (Catalog No: E-EL-H0043). Total Iron Binding Capacity (TIBC) was calculated using the formula: $TIBC = \text{Serum Transferrin} \times 1.389$. Transferrin saturation was subsequently calculated by using the formula: $\text{Transferrin saturation (\%)} = \frac{\text{Serum iron}}{TIBC} \times 100$.

Statistical Analysis

The collected data was analysed using IBM SPSS version 26.0 for Windows. Categorical variables like gender are presented as frequency and percentages. Continuous variables like age, serum iron indices and C-reactive protein level are summarized as mean and standard deviation or median and interquartile range, depending on the type of distribution. Inferential statistics like Student's t test and Spearman's correlation was used to represent the data. A p value of <0.05 was considered statistically significant.

RESULTS

A total of 120 participants (60 patients with ALD and 60 healthy controls) were included in the study. The mean age of the study participants was 51.3 ± 10.1 years. The mean age of the patients with ALD was 51.4 ± 10.5 years, compared to 51.2 ± 9.7 years for the healthy controls

(p=0.921). Both groups consisted of 58 males (96.7%) and 2 females (3.3%).

The mean alcohol intake among the patients with ALD was 214.8 ± 73.5 gm/day. The median alcohol intake

among these patients was 200 (180-265) gm/day, with a minimum of 90 gm/day and a maximum of 375 gm/day. Around 35.0% of the patients with ALD had a history of alcohol consumption exceeding 10 years.

Table 1. Distribution of study participants by liver function between the groups (N=120)

Liver parameters	Group		p value
	Alcoholic liver disease (n=60) Mean (SD)	Healthy controls (n=60) Mean (SD)	
Total bilirubin (mg/dl)	8.4 (7.0)	0.7 (0.3)	<0.001
Direct bilirubin (mg/dl)	4.5 (4.4)	0.16 (0.05)	<0.001
Total protein (g/dl)	6.2 (0.7)	7.4 (0.5)	<0.001
Albumin (g/dl)	2.5 (0.7)	4.2 (0.3)	<0.001
Globulin (g/dl)	3.6 (0.3)	3.2 (0.4)	<0.001
AST (IU)	113.7 (89.2)	29.8 (7.8)	<0.001
ALT (IU)	51.0 (36.9)	34.7 (10.4)	0.001
ALP (IU)	115.2 (54.7)	76.5 (17.3)	<0.001
GGT (IU)	220.5 (185.1)	31.2 (9.9)	<0.001

Table 1 shows the liver function levels between the patients with alcoholic liver disease and healthy controls. It can be noted that the mean total and direct bilirubin was significantly higher among the patients with ALD compared to the controls (p<0.001). Total proteins and albumin were significantly lower among the patients with ALD (p<0.001); whereas globulin was significantly higher among the patients with ALD (p<0.001). Liver enzymes (AST, ALT, ALP and GGT), were significantly higher among the patients with ALD (p<0.001).

Table 2. Comparison of renal function parameters, serum electrolytes, clotting factors and blood count between the groups (N=120)

Parameters	Group		p value
	Alcoholic liver disease (n=60) Mean (SD)	Healthy controls (n=60) Mean (SD)	
Serum urea (mg/dl)	59.2 (36.2)	27.2 (9.2)	<0.001
Serum creatinine (mg/dl)	1.3 (0.5)	0.9 (0.2)	<0.001
Serum sodium (mEq/L)	135.3 (4.2)	139.1 (3.9)	<0.001
Serum potassium (mEq/L)	3.8 (0.6)	4.3 (0.5)	<0.001

Serum chloride (mEq/L)	100.1 (4.0)	100.9 (3.5)	0.231
Prothrombin time (seconds)	19.7 (2.7)	12.3 (1.1)	<0.001
INR	1.8 (0.4)	1.0 (0.09)	<0.001
Platelet count (10³/cu.mm)	170.8 (44.7)	301.5 (66.9)	<0.001

Table 2 shows that the mean serum urea (59.2 mg/dl vs 27.2 mg/dl) and serum creatinine (1.3 mg/dl vs 0.9 mg/dl) were significantly higher among the participants with ALD when compared to the healthy controls ($p<0.001$) while the mean serum sodium and potassium were significantly lower among the participants with ALD when compared to the healthy controls ($p<0.001$). However, there was no significant difference in mean chloride between the groups ($p=0.231$). The mean prothrombin time and INR were significantly higher among the patients with ALD when compared to the healthy controls in our study ($p<0.001$). Mean platelet count was significantly lower among the patients with ALD when compared to the healthy controls in our study ($p<0.001$).

Table 3. Distribution of the ALD patients according to Child-Pugh score (N=60)

Child-Pugh score	Frequency (n)	Percentage
Class A (5-6 points)	13	21.7
Class B (7-9 points)	9	15.0
Class C (10-15 points)	38	63.3

Table 3 shows the severity of alcoholic liver disease using Child-Pugh score. It can be seen that Class A (well compensated disease), B (moderately severe disease with functional compromise) and C (decompensated cirrhosis) was observed among 21.7%, 15.0% and 63.3% patients, respectively.

Table 4. Comparison of serum iron indices and C-Reactive protein between the groups (N=120)

Parameters	Groups		p value
	Alcoholic liver disease (n=60) Mean (SD)	Healthy controls (n=60) Mean (SD)	
Serum Iron (µg/dl)	274.8 (75.8)	99.2 (26.2)	<0.001
Serum Ferritin (ng/ml)	415.4 (132.6)	154.2 (45.5)	<0.001
TIBC (ng/ml)	259.3 (93.3)	364.0 (53.3)	<0.001
Transferrin saturation (%)	128.5 (67.6)	28.0 (9.3)	<0.001
CRP (ng/ml)	4218.3 (2327.1)	335.1 (189.0)	<0.001

Table 4 shows that the mean serum iron, serum ferritin, transferrin saturation and CRP level was significantly higher among patients with alcoholic liver disease compared to the healthy controls (<0.001); whereas TIBC was significantly lower in patients with alcoholic liver disease (<0.001).

Table 5. Correlation of serum iron indices and C-Reactive Protein with severity (Child Pugh score) of alcoholic liver disease (N=60)

Serum iron indices	Correlation co-efficient (r)	p value
Serum iron (µg/dl)	0.920	<0.001
Serum ferritin (ng/ml)	0.865	<0.001
Total iron binding capacity (µg/dl)	-0.872	<0.001
Transferrin saturation (%)	0.900	<0.001
C-Reactive Protein (ng/ml)	0.860	<0.001

Table 5 shows that there was a very strong positive correlation for the serum iron, transferrin saturation, serum ferritin and C-reactive protein with the severity of ALD as measured by Child Pugh score and it was found to be statistically significant ($p<0.001$). With regards to total iron binding capacity with the severity of ALD (Child Pugh score), there was a very strong negative correlation and it was found to be statistically significant ($p<0.001$).

DISCUSSION

ALD remains a major cause of chronic liver morbidity and mortality worldwide and continues to contribute substantially to cirrhosis, decompensation and liver-related deaths in India. Progressive hepatic injury due to sustained alcohol exposure is driven not only by direct hepatotoxicity, but also by oxidative stress, immune dysregulation, gut-derived endotoxemia and systemic inflammation. Chronic alcohol intake can alter hepcidin regulation and intestinal iron absorption, leading to increased circulating iron and hepatic iron deposition. Iron excess may amplify hepatocellular injury through free radical generation and lipid peroxidation, thereby accelerating fibrosis progression and worsening clinical outcomes.

The mean age of the study participants in our study was 51.3 years with almost all being males. The mean age and marked male predominance are consistent with typical clinical ALD cohorts, reflecting higher prevalence of heavy alcohol use among men and later presentation after years of exposure. From an epidemiologic perspective, comparability on age/sex is important because iron indices and CRP can vary with age, sex and comorbid inflammatory conditions.¹⁷

In this study, the mean amount of alcohol intake among the patients with ALD was 214.8 ± 73.5 gm/day and 35.0% of the patients with ALD had more than 10 years of alcohol consumption. These findings were consistent with the established thresholds indicating that consumption exceeding 80 g/day for over a decade substantially increases the risk of cirrhosis.¹⁸ The type of alcohol consumed and the proportion of alcohol in those drinks can both be used to explain the rise in alcohol

consumption in Manipur. Local alcoholic beverages with a high alcohol level are traditionally produced and consumed in the state. Traditional brews have such a high alcohol content that only a small portion can provide a large amount of alcohol. This adds to the number of grams of alcohol consumed, especially when people drink more than one serving.^{19,20}

One of the important findings in our study is that there is significant higher bilirubin (total/direct), AST, ALT, ALP, GGT and globulin in ALD patients, with lower total protein and albumin, which is a classic biochemical constellation in alcohol-associated liver injury. In ALD, AST typically exceeds ALT (often $AST/ALT > 2$) due to vitamin B6 deficiency reducing ALT activity and mitochondrial injury increasing AST release. GGT ($\pm$ ALP) rises with chronic alcohol use and a cholestatic/mixed pattern from ductular reaction/cirrhotic changes. Albumin and total protein fall due to reduced hepatic synthesis and inflammation, while globulins increase from immune activation.^{21,22} Consistent with our findings, studies by Jangam M et al²³, Chand A et al²⁴ and Mamatha BV et al²⁵ demonstrated that patients with ALD exhibit significantly elevated levels of liver enzymes, bilirubin and prothrombin time, alongside markedly reduced albumin and total protein levels.

The urea and creatinine levels among ALD patients was significantly higher which suggests a higher burden of renal dysfunction, which is common in decompensated cirrhosis. The renal dysfunction in ALD could reflect pre-renal azotemia or acute kidney injury from infection, bleeding, nephrotoxic exposure or Hepatorenal syndrome.^{26,27} Studies by Jangam M et al²³ and Chand A et al²⁴, also had supported this evidence. Patients with

ALD also have significantly reduced serum sodium and potassium levels due to factors like dilutional hyponatremia, malnutrition and diuretic use. These electrolytes deficiencies serve as critical markers of severe liver decompensation and are strongly linked to increased mortality and complications, including encephalopathy and ascites and arrhythmias.^{28,29}

The mean prothrombin time and INR were significantly higher among the patients with ALD; whereas, platelet count was significantly lower among the patients with ALD which is consistent with the findings of Chand A et al²⁴. Higher prothrombin time and INR in ALD reflects reduced hepatic synthesis of clotting factors and overall synthetic dysfunction. Hence, INR is a key component in both Child–Pugh and MELD, making it a validated marker of advanced disease.^{26,30} Reduced platelet count is commonly due to portal hypertension related hypersplenism, reduced hepatic thrombopoietin production, bone marrow suppression from alcohol toxicity and consumptive processes. Consistent with our findings Das SK et al³¹ also documented similar alterations in PT, INR and platelet count.

Regarding iron indices, ALD patients exhibited significantly higher mean serum iron, transferrin saturation and ferritin, but significantly lower TIBC. This aligns with established pathophysiology showing that alcohol disrupts iron homeostasis and chronic liver disease impairs transferrin synthesis. Physiologically, iron suppresses hepcidin expression, increasing intestinal iron absorption and macrophage iron release via ferroportin. The resulting elevated circulating iron contributes to hepatic iron loading, which synergises with alcohol to exacerbate oxidative stress and liver injury.^{32,33} concurrently, elevated ferritin reflects both increased iron stores and its role as an acute phase reactant. In ALD it rises due to hepatic necrosis, systemic inflammation and reduced clearance.³⁴ Conversely, low TIBC reflect impaired hepatic synthesis and a negative acute-phase response. This dual pattern of high ferritin and low TIBC is well documented in cirrhosis capturing both compromised liver function and active inflammation.³⁵ In many studies elevated serum iron, ferritin and transferrin saturation have been consistently reported in ALD patients compared to controls with ferritin highlighted as a key marker for monitoring hepatic injury.^{23,24,36} Even when serum iron levels fluctuate, markedly reduced transferrin or TIBC alongside elevated ferritin remain consistent signatures of advanced disease and systemic inflammation.³⁷

There was a strong correlation between altered iron indices and Child–Pugh severity score: serum iron, transferrin saturation and ferritin correlated positively, while TIBC correlated negatively. This aligns with prior research demonstrating that serum iron and ferritin rise progressively with advancing liver dysfunction, with

ferritin serving as an independent predictor of severity.^{38,39,40} This relationship reflects worsening hepatic function and inflammation. Advanced cirrhosis impairs the hepatic synthesis of transferrin, driving down TIBC as severity scores rise. Simultaneously, accelerating hepatocyte necrosis and systemic inflammation stimulate the release of ferritin a key acute-phase reactant resulting in marked hyperferritinemia in severe disease.^{41,42}

Lastly, CRP levels were significantly higher in ALD patients than in controls and correlated strongly with Child–Pugh severity. Advanced ALD particularly decompensated cirrhosis and severe alcoholic hepatitis triggers systemic inflammation driven by altered gut permeability, endotoxemia, bacterial translocation and cytokine activation. As a hepatic acute-phase protein induced by IL-6, elevated CRP directly reflects this inflammatory state. Our findings align with extensive literature: significantly elevated CRP levels in ALD cohorts are well-documented, with levels peaking in Child–Pugh Class C patients.^{23,24} Furthermore, prior studies corroborate that CRP correlates significantly with disease severity scores like MELD, acute alcoholic hepatitis severity and iron indices such as ferritin.^{43,44,45}

LIMITATIONS

Our study has few limitations. First, its cross-sectional design prevents establishing causality between exposure (iron indices/CRP) and outcome (severity). Second a high proportion of severe cases (Child-Pugh C) introduces selection bias, limiting generalizability to early-stage of ALD. Finally, hepcidin levels were not measured, which could have clarified the link between alcohol intake and iron overload.

CONCLUSION

The present study evaluated the clinical and biochemical profile of patients with ALD compared to healthy controls. There were significant impairments in hepatic, renal and haematological functions among ALD patients, with a high prevalence of advanced disease severity and clinical complications. ALD patients exhibited significantly elevated CRP and iron indices, which demonstrated a strong correlation with Child-Pugh score. Based on these findings, we suggest that iron indices and CRP can serve as effective adjunct markers for assessing ALD severity. However, longitudinal studies incorporating oxidative stress markers and hepcidin are needed to further validate our findings.

CONFLICT OF INTEREST

The authors declare no potential conflicts of interest with respect to the research, authorship or publication of this article.

FUNDING STATEMENT

The authors gratefully acknowledge the financial and infrastructural support provided by the Multi-Disciplinary Research Unit (MRU), RIMS, Imphal, under Department of Health Research, the Ministry of Health and Family welfare, Government of India, which facilitated the execution, laboratory investigations and completion of this research project. The funding agency provided the necessary resources to conduct the study but had no direct involvement in the study design, data collection, statistical analysis, interpretation of the results or the decision to submit this manuscript for publication.

DATA AVAILABILITY STATEMENT

The datasets generated and analysed during the current study are available from the corresponding author upon reasonable request.

ACKNOWLEDGEMENT

First and foremost, we would like to express our deepest gratitude to the Department of Health Research, Ministry of Health and Family welfare, Government of India, for the financial support of this research. We are also grateful to the Regional Institute of Medical Sciences, Imphal, for allowing the utilization of resources for research, capacity building on research work and strengthening the research environment in the institute.

Finally, we would like to thank the Nodal officer and the staff of the Multi-Disciplinary Research Unit (MRU), RIMS, Imphal, for invaluable guidance, access to MRU laboratory facilities and constant support throughout the research work.

AUTHOR CONTRIBUTIONS

Davina Hijam: Conceptualization, Methodology, Investigation, Data Curation, Formal Analysis, Writing – Original Draft, Supervision.

Mairembam Jamuna Devi: Conceptualization, Methodology, Supervision, Writing Review & Editing.

Ng. Arunkumar Singh: Clinical Investigation, Patient Recruitment, Data Collection, Writing Review & Editing.

Nungshitombi Ngangbam: Laboratory Investigation, Data Collection, Validation.

Bidyarani Haobam: Laboratory Investigation, Data Curation, Validation.

Karam Romeo: Clinical Investigation, Patient Management, Resources, Writing Review & Editing.

Babina Sarangthem: Laboratory Investigation, Methodology, Validation, Writing Review & Editing.

All authors reviewed, critically revised, approved the final version of the manuscript, and agreed to be accountable for all aspects of the work.

REFERENCES

1. Acharya P, Chouhan K, Weiskirchen S, Weiskirchen R. Cellular Mechanisms of Liver Fibrosis. *Front Pharmacol*. 2021 May 6;12:671640.
2. Paik JM, Golabi P, Younossi Y, Mishra A, Younossi ZM. Changes in the global burden of chronic liver diseases from 2012 to 2017: the growing impact of NAFLD. *Hepatology*. 2020;72(5):1605-16.
3. World health statistics 2023: Monitoring Health for the SDGs, Sustainable Development Goals. Geneva: World Health Organization; 2023. Available at: <https://www.who.int/data/gho/publications/world-health-statistics>. Last accessed on November 20, 2025.
4. World Health Organization (WHO). Global status report on alcohol and health 2018. WHO; 2018. Available at: <https://www.who.int/publications/i/item/9789241565639>. Last accessed on November 20, 2025.
5. World life Expectancy India : Liver disease. In: World Health Rankings. World Life Expectancy. World Health Organisation 2020. Available at: <https://www.worldlifeexpectancy.com/india-liver-disease>. Last accessed on November 20, 2025.
6. Rehm J, Shield KD. Global Burden of Alcohol Use Disorders and Alcohol Liver Disease. *Biomedicines*. 2019;7(4):99-102.
7. Menon KVN, Gores GJ, Shah VH. Pathogenesis, Diagnosis, and Treatment of Alcoholic Liver Disease. *Mayo Clin Proc*. 2001;76(10):1021-29.
8. Sakaguchi S, Takahashi S, Sasaki T, Kumagai T, Nagata K. Progression of alcoholic and non-alcoholic steatohepatitis: common metabolic aspects of innate immune system and oxidative stress. *Drug Metab Pharmacokinet*. 2011;26(1):30-46.
9. Thursz M. Iron, haemochromatosis and thalassaemia as risk factors for fibrosis in hepatitis C virus infection. *Gut*. 2007;56(5):613-14.
10. Husić-Selimović A, Huskić J, Vukobrat-Bijedić Z, Mesihović R, Gribajcević M. The role of nitric oxide and ferritin in the pathogenesis of alcoholic liver disease: a controlled clinical study. *Bosn J Basic Med Sci*. 2009;9(3):204-09.
11. Harrison-Findik DD. Role of alcohol in the regulation of iron metabolism. *World J Gastroenterol*. 2007;13(37):4925-30.
12. Nemeth E, Tuttle MS, Powelson J, Vaughn MB, Donovan A, Ward DM, et al. Hcpidin regulates cellular iron efflux by binding to ferroportin and inducing its internalization. *Science*. 2004;306(5704):2090-93.

13. Fletcher LM, Halliday JW, Powell LW. Interrelationships of alcohol and iron in liver disease with particular reference to the iron-binding proteins, ferritin and transferrin. *J Gastroenterol Hepatol*. 1999;14(3):202-14.
14. Mehta KJ, Farnaud SJ, Sharp PA. Iron and liver fibrosis: Mechanistic and clinical aspects. *World J Gastroenterol*. 2019;25(5):521-38.
15. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. *N Engl J Med*. 1999;340(6):448-54.
16. Mariadi KI, Koncoro H, Wibawa DN. C-Reactive Protein and Interleukin-6 Correlated with Resistin Level in Liver Cirrhosis. *Indones J Gastroenterol Hepatol Dig Endosc*. 2020;21(1):17-21.
17. Bafei SEC, Yang S, Chen C, Gu X, Mu J, Liu F, et al. Sex and age differences in the association between high sensitivity C-reactive protein and all-cause mortality: A 12-year prospective cohort study. *Mech Ageing Dev*. 2023;211:111804.
18. Bruha R, Dvorak K, Petrtyl J. Alcoholic liver disease. *World J Hepatol*. 2012 Mar 27;4(3):81-90.
19. Kapai VY, Judith L, Muivah G. Indigenous fermented food and beverages of Manipur. In: Singh J, Pandey A, editors. *Microbial Products*. Boca Raton (FL): CRC Press; 2022. p. 119-35.
20. Singh PK. Traditional alcoholic beverage, Yu of Meitei communities of Manipur. *Indian J Tradit Knowl*. 2013;12(1):111-16.
21. Torkadi PP, Apte IC, Bhute AK. Biochemical Evaluation of Patients of Alcoholic Liver Disease and Non-alcoholic Liver Disease. *Indian J Clin Biochem*. 2014;29(1):79-83.
22. Sivasubramaniam R. A research study on the utility of GGT level and AST/ALT ratio in alcoholic liver diseases. *Int J Clin Biochem Res*. 2024;11(2):85-92.
23. Jangam M, Nandeesha H, Kadiravan T, Murugaiyan S. Association of Severity Serum Iron Indices and High-sensitivity C-reactive Protein with Disease Severity in Men with Alcoholic Liver Disease. *Indian J Med Biochem* 2021;25(3):96-99.
24. Chand A, Tahir FN, Shahbaz R, Salman R, Umar S, Abbas HB. Assessment of Serum Iron Indices and Levels of C-Reactive Protein in Patients of Alcoholic Liver Disease and their Relationship with Severity of Disease. *APMC* 2023;17(1):106-11.
25. Mamatha BV, Mahantesh SSV, ShankarPrasad DS, Kashinakunti SV, Manjula R. A comparative study of effect of alcohol on Serum Ferritin, LFT and GGT in a tertiary care hospital. *MedPulse Int J Biochem*. 2019;12(1):21-24.
26. Kamath PS, Wiesner RH, Malinchoc M, Kremers W, Therneau TM, Kosberg CL, et al. A model to predict survival in patients with end-stage liver disease. *Hepatology*. 2001;33(2):464-70.
27. Allegretti AS, Ortiz G, Wenger J, Deferio JJ, Wibecan J, Kalim S, et al. Prognosis of Acute Kidney Injury and Hepatorenal Syndrome in Patients with Cirrhosis: A Prospective Cohort Study. *Int J Nephrol*. 2015;2015:108139.
28. Rondon-Berrios H, Velez JCQ. Hyponatremia in Cirrhosis. *Clin Liver Dis*. 2022;26(2):149-64.
29. Kim WR, Biggins SW, Kremers WK, Wiesner RH, Kamath PS, Benson JT, et al. Hyponatremia and mortality among patients on the liver-transplant waiting list. *N Engl J Med*. 2008;359(10):1018-26.
30. Garcia-Tsao G. The Child-Turcotte Classification: From Gestalt to Sophisticated Statistics and Back. *Dig Dis Sci*. 2016;61(11):3102-04.
31. Das SK, Mukherjee S, Vasudevan DM, Balakrishnan V. Comparison of haematological parameters in patients with non-alcoholic fatty liver disease and alcoholic liver disease. *Singapore Med J*. 2011;52(3):175-81.
32. Li S, Hong M, Tan HY, Wang N, Feng Y. Insights into the Role and Interdependence of Oxidative Stress and Inflammation in Liver Diseases. *Oxid Med Cell Longev*. 2016;2016:4234061.
33. Harrison-Findik DD. Is the iron regulatory hormone hepcidin a risk factor for alcoholic liver disease? *World J Gastroenterol*. 2009;15(10):1186-93.
34. Wang W, Knovich MA, Coffman LG, Torti FM, Torti SV. Serum ferritin: Past, present and future. *Biochim Biophys Acta*. 2010;1800(8):760-69.
35. Viveiros A, Finkenstedt A, Schaefer B, Mandorfer M, Scheiner B, Lehner K, et al. Transferrin as a predictor of survival in cirrhosis. *Liver Transpl*. 2018;24(3):343-51.
36. Agrawal S, Chouhan DS, Sisodiya LS, Solanki J, Patel R. To Assess the Serum Ferritin Level Served as a Screening Test in Alcoholic Liver Disease. *J. Cardiovasc. Dis. Res*. 2024;15(5):1-5.
37. Ribot-Hernández I, Martín-González C, Vera-Delgado V, González-Navarrete L, de Armas-González JF, Viña-Rodríguez J, et al. Prognostic Value of Serum Iron, Ferritin, and Transferrin in Chronic Alcoholic Liver Disease. *Biol Trace Elem Res*. 2020;195(2):427-35.
38. Khare S, Garg VK, Jatav O. Serum Iron and TIBC Parameters in Chronic Liver Disease. *Sch J App Med Sci*. 2015;3(5E):2128-31.

39. Sudheendran P, Govindaraju C, Peter F, Suraj N, Iyer A, Narayanan P, et al. Is Serum Ferritin a Predictor of the Severity of Alcoholic Hepatitis. *J Clin Exp Hepatol* 2015;5(2):S15-6.
40. Siregar GA, Maail W. Serum iron parameters in liver cirrhosis. *IOP Conf Ser: Earth Environ Sci.* 2018;125:1-5.
41. Wang W, Knovich MA, Coffman LG, Torti FM, Torti SV. Serum ferritin: Past, present and future. *Biochim Biophys Acta.* 2010;1800(8):760-69.
42. Meire JA, Bokemeyer A, Cordes F, Fuhrmann V, Schmidt H, Husing-Kabar A, et al. Serum levels of ferritin and transferrin serve as prognostic factors for mortality and survival in patients with end-stage liver disease: a propensity score matched cohort study. *United European Gastroenterol J.* 2020;8(3):332-339.
43. Shardhul J, Haobam N, Haobam N, Vidhate A, Vidhate D. Elevated Ferritin/CRP Ratio: A Predictor of Liver Damage in Alcoholic Liver Disease Patients. 2024;14(6):673-81.
44. Vanbiervliet G, Le Breton F, Rosenthal-Allieri MA, Gelsi E, Marine-Barjoan E, Anty R, et al. Serum C-reactive protein: a non-invasive marker of alcoholic hepatitis. *Scand J Gastroenterol.* 2006;41(12):1473-79.
45. Mortensen C, Andersen O, Krag A, Bendtsen F, Møller S. High-sensitivity C-reactive protein levels predict survival and are related to haemodynamics in alcoholic cirrhosis. *Eur J Gastroenterol Hepatol.* 2012;24(6):619-26.