



Inflammatory Markers Esr, Crp & Serum Ferritin and Their Correlation to Child-Pugh Scoring in Chronic Liver Disease

Dr. Rajanala Tejaram¹, Dr. Uthaya Sankar M.K², Dr. Athisankaran³

¹Department of General Medicine, Aarupadai Veedu Medical College and Hospital, Puducherry, India

²Professor, Department of General Medicine, Aarupadai Veedu Medical College and Hospital, Puducherry, India

³Associate Professor, Department of Biochemistry, Aarupadai Veedu Medical College and Hospital, Puducherry, India

Article Information

Received: 13-06-2026

Revised: 26-06-2026

Accepted: 16-07-2026

Published: 27-07-2026

Keywords

Chronic liver disease; Child-Pugh score; ESR; C-reactive protein; Serum ferritin; Inflammation; Ascites; Variceal bleeding

ABSTRACT:

Background: Chronic liver disease progression is largely driven by systemic inflammation. While the Child-Pugh score is widely used for disease severity assessment, it lacks inflammatory markers. Erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and serum ferritin are inexpensive inflammatory markers that may provide additional prognostic information. **Aims:** To investigate the correlation between inflammatory markers (ESR, CRP, serum ferritin) and Child-Pugh score, and their association with clinical decompensation features in chronic liver disease. **Methods:** Hospital-based cross-sectional observational study at Aarupadai Veedu Medical College and Hospital, Puducherry, from January 2024 to January 2025. One hundred fifty adult inpatients with confirmed chronic liver disease (compensated or decompensated) were enrolled. Patients with concurrent infections, systemic inflammatory conditions, severe renal insufficiency, malignancy, thyroid dysfunction, or receiving warfarin or steroids were excluded. ESR, CRP, and serum ferritin levels were measured, and Child-Pugh score was calculated. Pearson or Spearman correlation analysis was performed. Group comparisons between Child-Pugh classes B and C were analyzed using t-tests and Mann-Whitney U tests. **Results:** Mean age was 52.3±9.8 years, with 93.3% males. Seventy-three percent belonged to Child-Pugh Class B and 27% to Class C. Ascites was present in 86.7% of patients and variceal bleeding in 13.3%, with zero mortality. ESR, CRP, and serum ferritin were significantly higher in Child-Pugh Class C compared to Class B ($p<0.001$). All three inflammatory markers demonstrated strong positive correlations with Child-Pugh score (ESR $r=0.62$; CRP $r=0.71$; serum ferritin $r=0.68$, all $p<0.001$). Inflammatory markers were significantly elevated in patients with ascites and variceal bleeding ($p<0.001$). **Conclusion:** ESR, CRP, and serum ferritin are accessible, inexpensive inflammatory markers that effectively reflect disease severity in chronic liver disease and correlate strongly with Child-Pugh score. These markers may complement existing prognostic systems and provide

©2026 The authors

This is an Open Access article

distributed under the terms of the Creative Commons Attribution (CC BY NC), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers. (<https://creativecommons.org/licenses/by-nc/4.0/>)

additional information for disease monitoring and risk stratification in clinical practice.

INTRODUCTION:

Chronic liver disease remains a major global health burden responsible for substantial morbidity and mortality. According to the Global Burden of Disease Study 2016, India accounted for approximately 200,000 deaths attributed to chronic liver disease, representing about six percent of global liver cirrhosis and chronic liver disease-related fatalities. Chronic liver disease is the third leading cause of death in individuals aged 45-64 years in India.^{1,3,7} The progression of chronic liver disease is characterized by progressive hepatic fibrosis and eventual cirrhosis, with clinical manifestations ranging from compensated disease to life-threatening decompensation.

The major etiological factors include chronic viral hepatitis (B and C viruses), alcohol-related liver disease, and non-alcoholic fatty liver disease. In the Indian population, hepatitis B virus remains the predominant driver of chronic liver disease, while alcohol is the leading cause of cirrhosis.^{6,7} Hepatitis C, now potentially curable with direct-acting antivirals, continues to cause significant morbidity due to delayed diagnosis and limited accessibility to treatment. Non-alcoholic fatty liver disease and its progressive form, non-alcoholic steatohepatitis, are rapidly emerging as major contributors to chronic liver disease, particularly in populations with obesity, diabetes mellitus, and metabolic syndrome.

Cirrhosis, the terminal stage of chronic liver disease, is increasingly recognized as a dynamic condition marked by periods of stability and acute decompensation rather than a fixed irreversible state. Decompensated cirrhosis presents with serious complications including ascites, variceal bleeding, hepatic encephalopathy, and hepatorenal syndrome, with substantially higher mortality rates. The pathophysiology of cirrhosis progression is complex, involving portal hypertension and systemic inflammation. Persistent systemic inflammation in chronic liver disease drives hepatic fibrosis, portal hypertension development, and increased susceptibility to bacterial infections and complications.

The Child-Pugh scoring system remains the most widely used tool for assessing disease severity and prognosis in chronic liver disease. It incorporates five clinical and biochemical parameters: serum bilirubin, serum albumin, prothrombin time, ascites, and hepatic encephalopathy, stratifying patients into classes A, B, and C, which represent increasing severity.^{10,12,13} However, the Child-Pugh score does not account for systemic inflammation, which is now recognized as a crucial driver of disease progression and adverse outcomes.

Erythrocyte sedimentation rate, C-reactive protein, and serum ferritin are readily available, inexpensive acute-phase proteins and markers of systemic inflammation. These markers are produced in response to hepatic injury and inflammatory cytokine activation. Erythrocyte sedimentation rate reflects the clumping of red blood cells due to elevated acute-phase proteins such as fibrinogen. C-reactive protein is a sensitive marker of acute inflammation with a short half-life and rapid response to inflammatory stimuli. Serum ferritin reflects both iron metabolism and systemic inflammation, with elevated levels in chronic liver disease indicating hepatic injury and immune activation rather than solely iron overload.

Recent studies demonstrate that inflammatory markers predict disease severity, complications, and mortality in cirrhosis. However, the specific correlations between ESR, CRP, and serum ferritin and the Child-Pugh score, and their association with clinical decompensation features such as ascites and variceal bleeding, require further investigation. This study aims to clarify these relationships in a well-characterized cohort of chronic liver disease patients, providing clinicians with evidence-based guidance for incorporating these simple, inexpensive markers into routine clinical assessment.

MATERIALS AND METHODS:

Study Design: Hospital-based cross-sectional observational study.

Study Setting: Department of General Medicine, Aarupadai Veedu Medical College and Hospital, Puducherry, India, supported by the Department of Biochemistry.

Study Population: Adult inpatients aged ≥ 18 years admitted to the Department of General Medicine with clinical and/or radiological diagnosis of chronic liver disease (compensated or decompensated) who provided informed written consent.

©2026 The authors

This is an Open Access article

distributed under the terms of the Creative Commons Attribution (CC BY NC), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers. (<https://creativecommons.org/licenses/by-nc/4.0/>)

Inclusion Criteria: Age ≥ 18 years; confirmed diagnosis of chronic liver disease (compensated or decompensated); inpatient status during study period; informed consent.

Exclusion Criteria: Concurrent infections; systemic inflammatory or autoimmune conditions; significant hematological disorders affecting erythrocyte sedimentation rate; thyroid dysfunction; severe renal insufficiency; non-hepatic malignancy; current use of warfarin, systemic corticosteroids, or hormone replacement therapy.

Study Period: January 2024 to January 2025 (12 months).

Sample Size: One hundred fifty patients were included based on sample size calculation using the formula: $N = Z(1-\alpha)^2 pq/d^2$, with 95% confidence level, 6% significance level, and expected prevalence derived from published literature.

Data Collection: Structured clinical proforma documented demographic characteristics, clinical history, and examination findings. Laboratory investigations included complete blood count, liver function tests, serum electrolytes, renal function, blood glucose, lipid profile, serum albumin and globulin, viral markers, coagulation profile (prothrombin time/international normalized ratio), erythrocyte sedimentation rate, C-reactive protein, and serum ferritin. Child-Pugh score was calculated for all patients. During hospitalization, patients were monitored for clinical decompensation features including ascites, variceal bleeding, and hepatic encephalopathy.

Statistical Analysis: Continuous variables were presented as mean $\pm$ standard deviation or median (interquartile range) depending on normality of distribution. Categorical variables were presented as frequencies and percentages. Correlation between inflammatory markers and Child-Pugh score was analyzed using Pearson or Spearman correlation coefficient based on distribution. Group differences between Child-Pugh classes were analyzed using independent samples t-test or Mann-Whitney U test. Categorical comparisons were performed using chi-square test or Fisher's exact test. P-value < 0.05 was considered statistically significant. Data analysis was performed using statistical software.

RESULTS:

Table 1. Demographic and Clinical Characteristics of the Study Population (N=150)

Variable	Category	No. of Patients (n)	Percentage (%)
Age (years)	40–60	120	80
	Other age groups	30	20
Sex	Male	140	93.3
	Female	10	6.7
Comorbidities	None	75	50
	Type 2 diabetes mellitus	45	30
	Hypertension	30	20
Child-Pugh Class	Class B	110	73.3
	Class C	40	26.7
Decompensating feature	Ascites	130	86.7
	Variceal bleeding	20	13.3
Mortality	No mortality	150	100

Table 2. Comparison of Inflammatory Markers According to Child-Pugh Class

Inflammatory Marker	Overall Mean $\pm$ SD	Range	Child-Pugh B (n=110) Mean $\pm$ SD	Child-Pugh C (n=40) Mean $\pm$ SD	p-value
ESR (mm/hour)	45.6 $\pm$ 12.4	20–80	41.2 $\pm$ 10.5	56.8 $\pm$ 11.3	< 0.001
CRP (mg/L)	28.3 $\pm$ 9.6	5–60	24.1 $\pm$ 7.8	38.6 $\pm$ 9.2	< 0.001
Serum ferritin (ng/mL)	410.5 $\pm$ 110.2	180–850	360.4 $\pm$ 85.6	540.8 $\pm$ 120.3	< 0.001

Table 3. Correlation of Inflammatory Markers with Child-Pugh Score

Inflammatory Marker	Correlation coefficient (r)	p-value	Strength of Correlation
ESR	0.62	< 0.001	Strong positive
CRP	0.71	< 0.001	Strong positive
Serum ferritin	0.68	< 0.001	Strong positive

Table 4. Association of Inflammatory Markers with Ascites

Inflammatory Marker	Ascites Present (n=130)	Ascites Absent (n=20) Mean $\pm$ SD	p-value
---------------------	-------------------------	-------------------------------------	---------

©2026 The authors

This is an Open Access article

distributed under the terms of the Creative Commons Attribution (CC BY NC), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers. (<https://creativecommons.org/licenses/by-nc/4.0/>)

	Mean $\pm$ SD		
ESR (mm/hour)	48.2 $\pm$ 11.8	29.0 $\pm$ 7.6	<0.001
CRP (mg/L)	30.1 $\pm$ 9.2	16.7 $\pm$ 5.4	<0.001
Serum ferritin (ng/mL)	435.6 $\pm$ 105.4	245.8 $\pm$ 70.2	<0.001

Table 5. Association of Inflammatory Markers with Variceal Bleeding

Inflammatory Marker	Variceal Bleeding Present (n=20) Mean $\pm$ SD	No Variceal Bleeding (n=130) Mean $\pm$ SD	p-value
ESR (mm/hour)	55.6 $\pm$ 10.9	43.8 $\pm$ 11.6	<0.001
CRP (mg/L)	41.2 $\pm$ 8.7	26.3 $\pm$ 8.9	<0.001
Serum ferritin (ng/mL)	565.4 $\pm$ 118.6	385.2 $\pm$ 96.4	<0.001

Table 6. Distribution of Variceal Bleeding According to Presence of Ascites

Clinical Feature	Variceal Bleeding Present (n=20)	Variceal Bleeding Absent (n=130)	Total (N=150)
Ascites present	20 (100.0%)	110 (84.6%)	130 (86.7%)
Ascites absent	0 (0.0%)	20 (15.4%)	20 (13.3%)
Total	20 (100.0%)	130 (100.0%)	150 (100.0%)

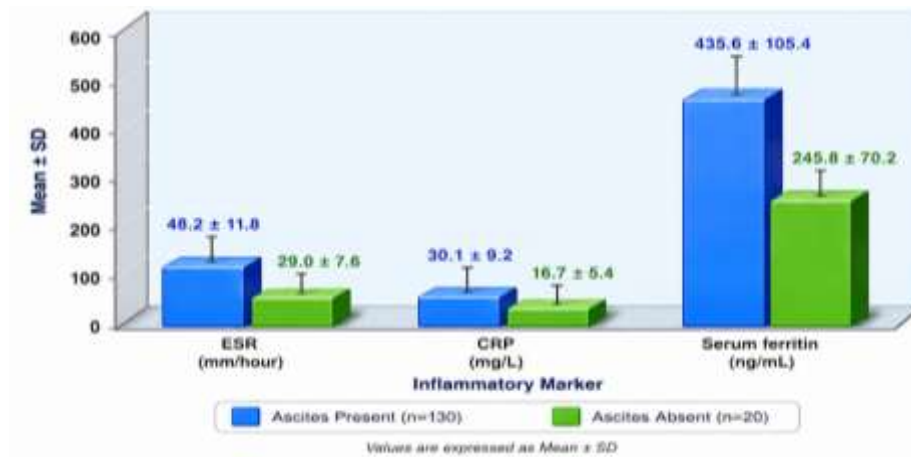


Figure 1. Association of Inflammatory Markers with Ascites

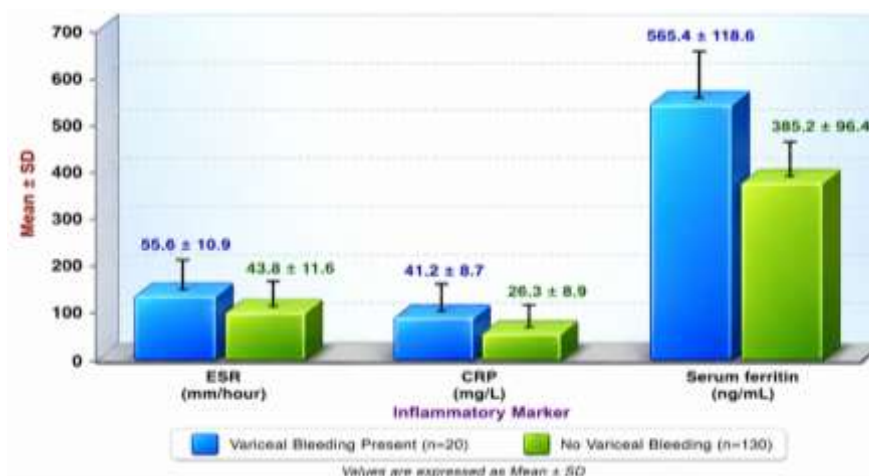


Figure 2. Association of Inflammatory Markers with Variceal Bleeding

©2026 The authors

This is an Open Access article

distributed under the terms of the Creative Commons Attribution (CC BY NC), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers. (<https://creativecommons.org/licenses/by-nc/4.0/>)

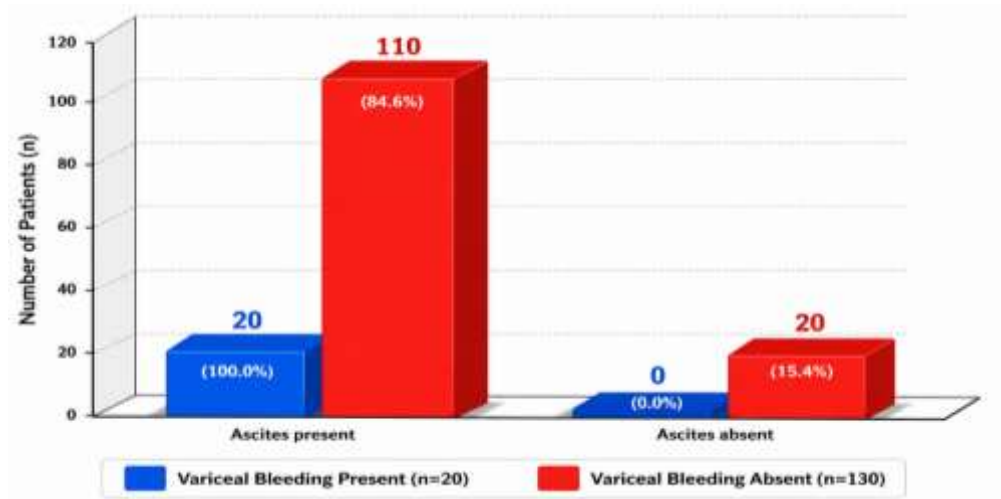


Figure 3. Distribution of Variceal Bleeding According to Presence of Ascites

Demographics and Clinical Characteristics:

One hundred fifty adults in patients with chronic liver disease were enrolled. The mean age was 52.3 ± 9.8 years, with eighty percent of patients in the 40-60 year age group. Male predominance was evident, with 93.3% of the cohort being male. Regarding comorbidities, 50% of patients had no associated comorbidities, 30% had type 2 diabetes mellitus, and 20% had hypertension. Child-Pugh Class B comprised 73% of the cohort ($n=110$), while Class C represented 27% ($n=40$). Ascites was the most frequent decompensating feature, observed in 86.7% of patients, followed by variceal bleeding in 13.3%. No mortality was recorded during the study period.

Inflammatory Markers and Child-Pugh Classification:

Mean ESR level was 45.6 ± 12.4 mm/hour (range 20-80). In Child-Pugh Class B patients, mean ESR was 41.2 ± 10.5 mm/hour, while in Class C, it was significantly higher at 56.8 ± 11.3 mm/hour ($p < 0.001$). Mean CRP level was 28.3 ± 9.6 mg/L (range 5-60). Child-Pugh Class B patients had mean CRP of 24.1 ± 7.8 mg/L, compared to 38.6 ± 9.2 mg/L in Class C patients ($p < 0.001$). Mean serum ferritin was 410.5 ± 110.2 ng/mL (range 180-850). Class B patients had mean serum ferritin of 360.4 ± 85.6 ng/mL, whereas Class C patients demonstrated significantly elevated levels of 540.8 ± 120.3 ng/mL ($p < 0.001$).

Correlation Analysis:

All three inflammatory markers demonstrated strong positive correlations with Child-Pugh score. Erythrocyte sedimentation rate showed a correlation coefficient of 0.62 ($p < 0.001$). C-reactive protein exhibited the strongest correlation with a correlation coefficient of 0.71 ($p < 0.001$). Serum ferritin demonstrated a correlation coefficient of 0.68 ($p < 0.001$). These findings indicate that inflammatory marker levels increase significantly with increasing disease severity as measured by Child-Pugh score.

Association with Ascites:

Patients with ascites ($n=130$) had significantly higher inflammatory markers compared to those without ascites ($n=20$). Mean ESR in ascites patients was 48.2 ± 11.8 mm/hour versus 29.0 ± 7.6 mm/hour in non-ascites patients ($p < 0.001$). Mean CRP in ascites patients was 30.1 ± 9.2 mg/L versus 16.7 ± 5.4 mg/L in non-ascites patients ($p < 0.001$). Mean serum ferritin in ascites patients was 435.6 ± 105.4 ng/mL versus 245.8 ± 70.2 ng/mL in non-ascites patients ($p < 0.001$).

Association with Variceal Bleeding:

Patients with variceal bleeding ($n=20$) demonstrated substantially elevated inflammatory markers. Mean ESR in patients with variceal bleeding was 55.6 ± 10.9 mm/hour versus 43.8 ± 11.6 mm/hour in those without bleeding ($p < 0.001$). Mean CRP in bleeding patients was 41.2 ± 8.7 mg/L versus 26.3 ± 8.9 mg/L in non-bleeding patients ($p < 0.001$). Mean serum ferritin in bleeding patients was 565.4 ± 118.6 ng/mL versus 385.2 ± 96.4 ng/mL in non-bleeding patients ($p < 0.001$). Notably, variceal bleeding was observed exclusively in patients with ascites.

©2026 The authors

This is an Open Access article

distributed under the terms of the Creative Commons Attribution (CC BY NC), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers. (<https://creativecommons.org/licenses/by-nc/4.0/>)

DISCUSSION:

This study demonstrates significant correlations between three readily available inflammatory markers (ESR, CRP, and serum ferritin) and disease severity as measured by Child-Pugh score in chronic liver disease. All markers showed strong positive correlations with disease severity, with C-reactive protein demonstrating the strongest association. These findings extend existing knowledge regarding the role of systemic inflammation in chronic liver disease pathogenesis and progression.

The predominance of male patients (93.3%) in this cohort reflects epidemiological patterns documented in multiple studies, attributable to higher alcohol consumption in males and delayed healthcare-seeking behavior in females. The age distribution, with 80% of patients in the 40-60 year age group, is consistent with the progressive nature of chronic liver disease, which typically develops over decades. The substantial prevalence of type 2 diabetes mellitus (30%) and hypertension (20%) as comorbidities is noteworthy and reflects the increasing overlap between metabolic disorders and liver disease.

Ascites was the predominant decompensating feature in 86.7% of patients, consistent with its recognition as the most common manifestation of portal hypertension in cirrhotic disease. The occurrence of variceal bleeding exclusively in patients with ascites underscores the association between progressive portal hypertension and the development of esophageal varices. The absence of mortality during the study period may reflect the relatively short follow-up duration and exclusion of patients with acute infections.

The significantly elevated inflammatory markers in Child-Pugh Class C compared to Class B patients provides objective evidence supporting the recognized role of systemic inflammation in advanced cirrhosis. C-reactive protein demonstrated the strongest correlation ($r=0.71$) with Child-Pugh score, followed by serum ferritin ($r=0.68$) and ESR ($r=0.62$). The relative strength of these correlations is noteworthy. C-reactive protein, as an acute-phase reactant with rapid kinetics, may more sensitively reflect active inflammatory processes. Serum ferritin, while influenced by both iron metabolism and inflammation, also demonstrates robust association with disease severity. Erythrocyte sedimentation rate, though less specific, maintains significant correlation with disease progression.

The substantially elevated inflammatory markers in patients with ascites reflect the relationship between systemic inflammation and the transition to clinical decompensation. Portal hypertension-induced mesenteric vasodilation and splanchnic vasodilation trigger compensatory systemic vasoconstriction and activation of neurohumoral systems, perpetuating systemic inflammation. The bacterial translocation from the gut and impaired host immunity further amplify the inflammatory burden.

Variceal bleeding, associated with the highest inflammatory marker levels in this study, reflects the severity of portal hypertension and underlying systemic inflammation. Enhanced inflammation may impair endothelial function and increase bleeding risk. These findings suggest that inflammatory markers may serve as complementary tools to the Child-Pugh score for risk stratification and disease monitoring in chronic liver disease. The accessibility and low cost of these markers make them particularly valuable for resource-limited settings.

Study limitations include the single-center design, cross-sectional nature precluding causal inferences, and inclusion primarily of Child-Pugh Classes B and C. The relatively short study duration prevented assessment of mortality and long-term outcomes. Additionally, inflammatory marker levels may be influenced by concurrent infections and other systemic conditions not captured in the exclusion criteria. Future prospective multicenter studies with extended follow-up are warranted to validate these findings and establish cutoff values for clinical application.

CONCLUSION:

Erythrocyte sedimentation rate, C-reactive protein, and serum ferritin demonstrate strong correlations with Child-Pugh score and disease severity in chronic liver disease. These readily available, inexpensive inflammatory markers significantly associate with clinical decompensation features including ascites and variceal bleeding. Integration of inflammatory markers with existing prognostic systems may enhance disease monitoring, risk stratification, and clinical decision-making in chronic liver disease management. These findings support the incorporation of simple inflammatory markers into routine clinical assessment protocols for patients with chronic liver disease, particularly in resource-constrained settings. Further prospective studies are recommended to establish optimal cutoff values and validate the clinical utility of these markers for predicting short-term outcomes.

©2026 The authors

This is an Open Access article

distributed under the terms of the Creative Commons Attribution (CC BY NC), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers. (<https://creativecommons.org/licenses/by-nc/4.0/>)

and mortality.

DECLARATIONS:

CONFLICT OF INTEREST: Not Applicable

FUNDING: Not Applicable

REFERENCES:

1. Schuppan D, Afdhal NH. Liver cirrhosis. *Lancet*. 2008;371(9615):838-851.
2. Bataller R, Brenner DA. Liver fibrosis. *J Clin Invest*. 2005;115(2):209-218.
3. Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. *Lancet*. 2014;383(9930):1749-1761.
4. Mokdad AA, Lopez AD, Shahraz S, et al. Liver cirrhosis mortality in 187 countries between 1980 and 2010. *BMC Med*. 2014;12:145.
5. Asrani SK, Larson JJ, Yawn B, et al. Underestimation of liver-related mortality in the United States. *Gastroenterology*. 2013;145(2):375-382.
6. Singh SP, Reddy KR. Epidemiology of chronic liver disease in India. *J Clin Exp Hepatol*. 2022;12(2):58-67.
7. Paik JM, Golabi P, Fazel Y, et al. Global epidemiology of chronic liver disease. *Frontline Gastroenterol*. 2021;13(3):215-223.
8. Angeli P, Bernardi M, Villanueva C, et al. From compensation to decompensation in cirrhosis. *Clin Gastroenterol Hepatol*. 2023;21(4):908-919.
9. GBD 2019 Cirrhosis Collaborators. Global burden of liver cirrhosis and other chronic liver diseases, 1990–2019. *Hepatol Int*. 2024;18(2):452-465.
10. Garcia-Tsao G. Child-Turcotte-Pugh classification. *Dig Dis Sci*. 2016;61(11):3102-3104.
11. Kamath PS, Wiesner RH. Model for End-Stage Liver Disease (MELD). *Hepatology*. 2001;33(2):464-470.
12. Durand F, Valla D. Prognosis assessment in cirrhosis: Child-Pugh versus MELD. *J Hepatol*. 2005;42(Suppl 1):S100-S107.
13. Christensen E. Prognostic models in liver disease. *J Hepatol*. 2004;41(2):344-350.
14. Brigden ML. Clinical utility of the erythrocyte sedimentation rate. *Am Fam Physician*. 1999;60(5):1443-1450.
15. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. *N Engl J Med*. 1999;340(6):448-454.
16. Kalaitzakis E, Olsson R, Henfridsson P, et al. Systemic inflammation in cirrhosis. *J Hepatol*. 2009;50(5):907-914.
17. Rimola A, Soto R, Bory F, et al. Reticuloendothelial system phagocytic activity in cirrhosis. *J Hepatol*. 1994;21(3):405-412.
18. Cazzaniga M, Dionigi E, Gobbo G, et al. SIRS and outcome in cirrhosis. *J Hepatol*. 2009;51(3):475-482.
19. Cervoni JP, Thévenot T, Weil D, et al. C-reactive protein predicts short-term mortality in cirrhosis. *J Hepatol*. 2012;56(6):1299-1304.
20. Thabut D, Massard J, Gangloff A, et al. Prognostic value of CRP in cirrhosis. *J Hepatol*. 2007;46(3):451-458.
21. Pieri G, Agarwal B, Burroughs AK. C-reactive protein and prognosis in cirrhosis. *J Clin Transl Hepatol*. 2014;2(1):1-6.
22. Arvaniti V, D'Amico G, Fede G, et al. Infections increase mortality in cirrhosis. *J Hepatol*. 2010;52(4):487-495.
23. Albillos A, Lario M, Álvarez-Mon M. Cirrhosis-associated immune dysfunction. *J Hepatol*. 2014;61(6):1385-1396.
24. Moreau R, Jalan R, Gines P, et al. Acute-on-chronic liver failure. *Gastroenterology*. 2013;144(7):1426-1437.
25. Maiwall R, Kumar S, Chaudhary AK, et al. Serum ferritin predicts early mortality in decompensated cirrhosis. *J Hepatol*. 2014;61(1):43-50.
26. Oikonomou T, Goulis I, Christodoulou D, et al. High serum ferritin and outcome in cirrhosis. *Ann Gastroenterol*. 2017;30(2):217-224.
27. Walker NM, Stuart KA, Ryan RJ, et al. Serum ferritin predicts mortality in transplant candidates. *Hepatology*. 2010;51(5):1683-1691.
28. Umar M, Bilal M, Khan FA, et al. Serum ferritin and disease severity in cirrhosis. *J Ayub Med Coll Abbottabad*. 2016;28(4):742-746.
29. Sungkar T, Lubis M, Gurning M, et al. Ferritin as a biomarker in decompensated cirrhosis. *Malays J Med Sci*. 2019;26(2):59-65.
30. Attallah KM, El-Fayoumi H, El-Shabrawy M. Acute-phase inflammatory markers in chronic liver disease. *J Am Sci*. 2013;9(10):25-32.
31. Kumar B, Kumari B, Kumari P. Inflammatory markers and Child-Pugh score. *Int J Acad Med Pharm*. 2024;6(1):1196-1202.

©2026 The authors

This is an Open Access article

distributed under the terms of the Creative Commons Attribution (CC BY NC), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers. (<https://creativecommons.org/licenses/by-nc/4.0/>)