

Study of Neutrophil-To-Lymphocyte Ratio (NLR) and Its Correlation with Severity of Decompensated Cirrhosis of Liver Based on Child-Turcotte Pugh (CTP) Score

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Abstract: Background: Cirrhosis of the liver is the end stage of chronic liver disease marked by fibrosis, regenerative nodules, and architectural distortion. Disease severity is graded by the Child–Turcotte–Pugh (CTP) score, while the neutrophil-to-lymphocyte ratio (NLR) has recently emerged as a simple biomarker of systemic inflammation and immune imbalance. Since inflammation drives hepatic decompensation, NLR may serve as a prognostic indicator of cirrhosis severity. Objective: To assess the relationship between NLR and the severity of decompensated cirrhosis based on the CTP score. Methods: A cross-sectional study was performed on 65 patients with decompensated cirrhosis admitted to General Hospital, Jayanagar, Bengaluru, from August 2022 to December 2023. NLR was derived from complete blood counts, and CTP scores were calculated using bilirubin, albumin, INR, ascites, and encephalopathy parameters. Correlations were analyzed using Pearson's coefficient, with $p < 0.05$ considered significant. Results: The mean age was 44.9 ± 11.5 years; 89.2% were males. Most patients (61.5%) had $NLR > 3.53$, and CTP classes A, B, and C comprised 9.2%, 43.1%, and 47.7% respectively. NLR correlated positively with CTP score ($r = 0.345$, $p = 0.005$), prothrombin time, INR, bilirubin, and encephalopathy, and negatively with hemoglobin and albumin. Conclusion: Elevated NLR reflects greater disease severity in decompensated cirrhosis and may serve as a rapid, inexpensive prognostic marker complementing the CTP score.

Keywords: Neutrophil-to-lymphocyte ratio, Child-Turcotte-Pugh score, decompensated cirrhosis, inflammation, prognosis.

1. Introduction

Cirrhosis of the liver represents the end stage of chronic liver disease, characterized by fibrosis, nodule formation, and irreversible architectural distortion of hepatic parenchyma [1]. It results from long-standing hepatic injury due to diverse etiologies such as chronic viral hepatitis (HBV, HCV), alcoholic liver disease, non-alcoholic steatohepatitis (NASH), autoimmune hepatitis, and metabolic or genetic disorders including Wilson's disease and hemochromatosis [2]. Globally, cirrhosis is a leading cause of morbidity and mortality, accounting for nearly 1.3 million deaths annually, and its burden continues to rise due to increasing cases of fatty liver and viral infections [3]. According to the World Health Organization (WHO), liver disease contributes to approximately 2.95% of total deaths in India, representing a major public-health challenge in the country [4].

Cirrhosis culminates in portal hypertension, hepatocellular failure, and systemic inflammation, leading to hepatic decompensation manifested by ascites, jaundice, variceal bleeding, or hepatic encephalopathy [5]. The Child–Turcotte–Pugh (CTP) score is a well-validated and widely used clinical tool to evaluate hepatic reserve and predict prognosis in cirrhotic patients [6]. It combines five clinical and biochemical parameters—serum bilirubin, albumin, prothrombin time, ascites, and encephalopathy—to classify

patients into CTP classes A, B, and C, corresponding to mild, moderate, and severe disease, respectively [7].

Recently, the neutrophil-to-lymphocyte ratio (NLR) has emerged as a simple, cost-effective biomarker of systemic inflammation and immune imbalance [8]. An elevated NLR indicates neutrophilia, a reflection of ongoing inflammation, coupled with lymphopenia, denoting immune dysregulation and exhaustion—both key mechanisms underlying hepatic injury and disease progression [9]. Several studies have demonstrated that higher NLR values are associated with more advanced cirrhosis, increased risk of complications, and higher short-term mortality, making it a promising prognostic indicator [10,11].

Considering the pivotal role of inflammation in hepatic decompensation and outcome prediction, the present study was undertaken to evaluate the correlation between NLR and the CTP score in patients with decompensated liver cirrhosis, and to assess the utility of NLR as a rapid, inexpensive marker for grading disease severity and predicting prognosis.

2. Materials and Methods

2.1 Study design and setting

This was a hospital-based cross-sectional observational study conducted in the Department of General Medicine,

General Hospital, Jayanagar, Bengaluru, over a period of **16 months (August 2022 to December 2023)**. The study protocol was reviewed and approved by the **Institutional Ethics Committee (IEC No: [insert number])**, and written informed consent was obtained from all participants prior to enrollment. The study was carried out in accordance with the **Declaration of Helsinki (2013 revision)**.

2.2 Sample size and participants

A total of **65 adult patients** (aged ≥ 18 years) diagnosed with **decompensated cirrhosis of liver** based on clinical, biochemical, and ultrasonographic findings were included. The sample size was calculated using a **95% confidence level and 80% statistical power**, yielding $n = 65$. Cirrhosis was diagnosed by characteristic clinical features, imaging evidence of a coarse echotexture with nodular liver surface, and biochemical derangements indicative of chronic hepatic dysfunction.

2.3 Inclusion criteria

- Age ≥ 18 years.
- Clinically and ultrasonographically diagnosed cases of cirrhosis of liver.
- Patients willing to provide informed written consent.

2.4 Exclusion criteria

- Patients with hepatocellular carcinoma or other malignancies.
- Diabetes mellitus or systemic infections that could alter inflammatory markers.
- Recent blood transfusion (<3 months).
- Acute liver failure or acute-on-chronic liver failure.
- Pregnancy.
- Chronic kidney disease, autoimmune disorders, or corticosteroid therapy.
- HBsAg or anti-HCV positive patients.

2.5 Data collection and clinical evaluation

A detailed **history and physical examination** were performed for all participants, documenting **age, sex, etiology of cirrhosis, clinical symptoms (jaundice, ascites, encephalopathy), and complications** such as variceal bleeding or spontaneous bacterial peritonitis.

Routine investigations included:

- **Complete blood count (CBC)** using an automated hematology analyzer (Sysmex XN-1000).
- **Liver function tests (LFTs):** Total bilirubin, direct bilirubin, AST, ALT, alkaline phosphatase, total protein, and albumin.
- **Renal function tests (RFTs):** Serum urea and creatinine.
- **Coagulation profile:** Prothrombin time (PT) and International Normalized Ratio (INR).

The **neutrophil-to-lymphocyte ratio (NLR)** was calculated as:

$NLR = \text{Absolute neutrophil count} / \text{Absolute lymphocyte count}$

All hematological analyses were performed on venous blood samples collected under aseptic conditions and processed within two hours of collection to avoid cellular degradation.

2.6 Assessment of disease severity (Child–Turcotte–Pugh score)

The **CTP score** was calculated for each patient using the following parameters: **serum bilirubin, serum albumin, PT/INR, ascites, and hepatic encephalopathy**, assigning 1–3 points for each variable according to standard scoring criteria.

Patients were categorized as:

- **Class A:** 5–6 points (well-compensated)
- **Class B:** 7–9 points (significant functional compromise)
- **Class C:** 10–15 points (decompensated)

2.7 Statistical analysis

All data were entered into Microsoft Excel and analyzed using **IBM SPSS Statistics version 20 (SPSS Inc., Chicago, IL, USA)**. Continuous variables were expressed as **mean \pm standard deviation (SD)**, while categorical variables were presented as **frequencies and percentages**. The **Pearson correlation coefficient (r)** was used to assess the relationship between **NLR and CTP score**, as well as between NLR and other laboratory parameters such as bilirubin, INR, and albumin. Comparisons between groups were made using the **Chi-square test** for categorical data and the **Student's *t*-test** for continuous variables. A p -value < 0.05 was considered statistically significant.

3. Results

3.1 Sociodemographic and clinical characteristics

A total of 65 patients with decompensated cirrhosis of liver were included in the study. The mean age (mean \pm SD) of the participants was 44.9 ± 11.5 years, ranging from 25 to 69 years. The number of male and female patients were 58 (89.2%) and 7 (10.8%), respectively. Among the enrolled participants, the maximum number belonged to the 40–54 year age group (50.8%), followed by the 25–39 year group (29.2%) and the 55–69 year group (20.0%). The most frequent presenting complaints included abdominal distension (100%), pedal edema (98.5%), yellowish discoloration of eyes (86.2%), fatigue (62%), and tremors (40%). Clinically, ascites was seen in all patients—grade 2 ascites in 87.7% and grade 3 ascites in 12.3%. Hepatic encephalopathy was observed in 63.1% at stage 1, 32.3% at stage 2, and 4.6% at stage 3. The mean serum creatinine level was 1.08 ± 0.38 mg/dL, and 61.5% had values between 0.7–1.2 mg/dL. These demographic and baseline characteristics are summarized in Tables 1 and 2.

Table 1: Age and gender distribution of study participants

Parameter	Category	n	%	Mean \pm SD
Age (years)	25–39	19	29.2	44.9 ± 11.5
	40–54	33	50.8	
	55–69	13	20.0	
Gender	Male	58	89.2	
	Female	7	10.8	

Table 2: Clinical characteristics of study participants

Parameter	Category	n	%
Stage of hepatic encephalopathy	Stage 1	41	63.1
	Stage 2	21	32.3
	Stage 3	3	4.6
Grade of ascites	Grade 2	57	87.7
	Grade 3	8	12.3
Serum creatinine (mg/dL)	<0.7	14	21.5
	0.7–1.2	40	61.5
	>1.2	11	16.9
Common presenting complaints	Abdominal distension	65	100
	Pedal edema	64	98.5
	Yellowish discoloration of eyes	56	86.2
	Fatigue	40	62.0
	Tremors	26	40.0

3.2 Hematological and biochemical findings

The mean hemoglobin level (mean \pm SD) among participants was 9.89 ± 2.14 g/dL, with 58.5% of cases between 5.1–10 g/dL and 41.5% between 10.1–15 g/dL. The total leukocyte count (TLC) ranged from 2400–37470/mm³, with 61.5% of patients showing TLC between 4000–11000/mm³, 29.2% above 11000/mm³, and 9.2% below 4000/mm³. The mean platelet count was 1.54 ± 0.42 lakhs/mm³, and thrombocytopenia (≤ 1.5 lakhs/mm³) was found in 64.6% of cases. Prolonged prothrombin time (>16 s) was present in 73.8% of patients, and INR > 1.7 in 33.9%. Biochemically, total bilirubin (mean \pm SD) was 3.42 ± 1.8 mg/dL, with 56.9% of participants showing values > 3 mg/dL. The mean serum albumin was 3.02 ± 0.64 g/dL, with 83.1% below 3.5 g/dL. These findings indicate a significant burden of anemia, coagulopathy, hypoalbuminemia, and hyperbilirubinemia among decompensated cirrhotics.

3.3 Distribution of NLR and CTP score

The mean neutrophil-to-lymphocyte ratio (NLR) (mean \pm SD) among the participants was 4.02 ± 1.83 , ranging from 1.5 to 8.5. None of the patients had NLR < 0.78 . A total of 25 (38.5%) participants had NLR between 0.78–3.53, while 40 (61.5%) had NLR > 3.53 . Based on the Child–Turcotte–Pugh (CTP) classification, 6 (9.2%) patients were in Class A, 28 (43.1%) in Class B, and 31 (47.7%) in Class C. The mean CTP score (mean \pm SD) of the study population was 9.24 ± 2.11 , ranging from 6 to 14.

The mean NLR values across the CTP classes were:

- Class A: 2.34 ± 0.71
- Class B: 3.71 ± 1.65
- Class C: 4.53 ± 1.22

The difference was statistically significant ($p = 0.002$), indicating a steady rise in NLR with increasing CTP score and hence worsening hepatic dysfunction. (Table 3).

Table 3: Hematological and biochemical parameters of study participants

Parameter	Range / Category	n	%	Mean \pm SD
Hemoglobin (g/dL)	5.1–10	38	58.5	9.89 ± 2.14
	10.1–15	27	41.5	
Total leukocyte count (/mm ³)	<4000	6	9.2	
	4000–11000	40	61.5	
	>11000	19	29.2	

Platelets (lakhs/mm ³)	≤ 1.5	42	64.6	1.54 ± 0.42
	> 1.5	23	35.4	
Prothrombin time (s)	12–16	17	26.2	18.1 ± 2.9
	> 16	48	73.8	
INR	< 1.7	43	66.1	1.72 ± 0.36
	1.7–2.3	16	24.6	
	> 2.3	6	9.3	
Total bilirubin (mg/dL)	< 2	18	27.7	3.42 ± 1.8
	2–3	10	15.4	
	> 3	37	56.9	
Serum albumin (g/dL)	≤ 3.5	54	83.1	3.02 ± 0.64
	> 3.5	11	16.9	

3.4 Correlation between NLR and clinical–biochemical parameters

Pearson’s correlation analysis (Table 4) revealed a significant positive correlation between NLR and CTP score ($r = 0.345$, $p = 0.005$). NLR also showed positive correlation with total bilirubin ($r = 0.250$, $p = 0.045$), direct bilirubin ($r = 0.263$, $p = 0.035$), prothrombin time ($r = 0.169$, $p = 0.178$), INR ($r = 0.199$, $p = 0.112$), and stage of hepatic encephalopathy ($r = 0.344$, $p = 0.005$). Conversely, a negative correlation was found with hemoglobin ($r = -0.044$, $p = 0.728$) and serum albumin ($r = -0.169$, $p = 0.179$). The findings imply that higher inflammatory burden (NLR) parallels deterioration in liver function and increased clinical severity as reflected by CTP scoring.

Table 4: Correlation of NLR with clinical and biochemical parameters

Parameter	Correlation coefficient (r)	p-value
CTP score	0.345	0.005
Hemoglobin	-0.044	0.728
Total leukocyte count	0.121	0.338
Absolute neutrophil count	0.141	0.264
Absolute lymphocyte count	-0.043	0.734
Platelets	0.050	0.695
Prothrombin time	0.169	0.178
INR	0.199	0.112
Total bilirubin	0.250	0.045
Direct bilirubin	0.263	0.035
Serum albumin	-0.169	0.179
Stage of hepatic encephalopathy	0.344	0.005
Grade of ascites	-0.218	0.082
Serum creatinine	0.032	0.801

3.5 Comparative analysis and trends

On stratification, patients with NLR > 3.53 had significantly higher CTP scores and more advanced ascites and encephalopathy stages compared to those with NLR ≤ 3.53 . The mean NLR progressively increased with the grade of hepatic decompensation. No significant association was noted between NLR and age ($p = 0.73$) or platelet count ($p = 0.69$). However, elevated NLR was consistently associated with increased prothrombin time, INR, and bilirubin, and decreased albumin levels—parameters integral to the CTP scoring system.

Collectively, these results highlight that NLR correlates positively with liver disease severity and may serve as an adjunctive prognostic indicator in patients with decompensated cirrhosis.

4. Discussion

The present study demonstrates a significant positive correlation between neutrophil-to-lymphocyte ratio (NLR) and Child–Turcotte–Pugh (CTP) score ($r = 0.345$, $p = 0.005$), indicating that NLR closely reflects the degree of hepatic decompensation in cirrhosis. This finding supports the hypothesis that systemic inflammation and immune dysregulation play key roles in disease progression and clinical severity in chronic liver disease.

4.1 Correlation with previous studies

Our results are consistent with earlier reports. Sungkar et al. observed a similar correlation ($r = 0.326$, $p = 0.008$), while Tandale et al. found a stronger association ($r = 0.47$, $p = 0.0001$) [12,13]. Wadhvani et al. also demonstrated a positive correlation ($r = 0.572$, $p = 0.002$), confirming NLR's predictive role in hepatic dysfunction [14].

Vigneshwaran et al. and Meena D et al. further reported progressively higher NLR values with increasing CTP class, reinforcing its value as a prognostic indicator [15,16]. The consistent pattern across studies suggests that NLR may serve as a reliable adjunct to established scoring systems for stratifying patients according to disease severity.

4.2 Pathophysiological explanation

Cirrhosis is marked by persistent hepatocellular injury, chronic inflammation, and immune dysfunction. Portal hypertension and bacterial translocation from the gut lead to activation of **Kupffer cells** and release of **cytokines** such as TNF- α , IL-6, and IL-8, resulting in neutrophilia and lymphocyte apoptosis [17].

Elevated neutrophil counts indicate ongoing systemic inflammation, while lymphopenia reflects impaired adaptive immune response—together producing a raised NLR. The significant positive correlation between NLR and CTP score in this study underscores that higher inflammatory burden is associated with worsening hepatic synthetic function, as evidenced by elevated bilirubin, prolonged PT/INR, and hypoalbuminemia.

4.3 Comparison of mean NLR across CTP classes

In the present study, mean NLR increased significantly from 2.34 in Class A to 4.53 in Class C ($p = 0.002$), consistent with previous literature. Vigneshwaran et al. reported mean values of 2.57, 3.18, and 4.99 for Classes A, B, and C respectively ($p < 0.05$), while Meena D et al. observed 2.8, 6.13, and 9.42 ($p < 0.0001$) [15,16]. Although our values were lower—likely due to smaller sample size ($n = 65$) and exclusion of concurrent infections—the upward trend remains comparable, reaffirming that NLR increases with disease severity.

4.4 Correlation with other clinical and biochemical parameters

NLR showed significant positive correlations with total bilirubin, prothrombin time, INR, and hepatic

encephalopathy, and negative correlations with hemoglobin and serum albumin—parameters integral to the CTP scoring system. These findings are in agreement with Biyik et al., who found that high NLR predicted both mortality and complications such as spontaneous bacterial peritonitis and variceal bleeding [18].

Anemia and hypoalbuminemia are well-recognized consequences of advanced liver disease and further reflect systemic inflammation and poor hepatic synthetic capacity.

4.5 Clinical and prognostic implications

NLR is an attractive biomarker because it is simple, inexpensive, and derived from a routine complete blood count. It may complement established models such as CTP and MELD scores in assessing disease severity and predicting prognosis. Elevated NLR values have been linked to increased mortality, longer hospital stays, and higher risk of complications in decompensated cirrhosis [19, 20].

Compared to more costly inflammatory markers like C-reactive protein (CRP) or IL-6, NLR offers a practical bedside index of systemic inflammation and immune imbalance. Identifying patients with elevated NLR may enable early risk stratification, closer monitoring, and timely therapeutic interventions.

4.6 Limitations

This study is limited by its single-center design and small sample size, which may affect the generalizability of findings. Potential confounders such as subclinical infections, nutritional status, or hematologic variations were not fully controlled. Additionally, this was a cross-sectional study, so dynamic changes in NLR during treatment or follow-up could not be evaluated. Larger, multicentric, longitudinal studies are warranted to validate cutoff values and assess the predictive accuracy of NLR compared to other scoring systems.

4.7 Summary

In summary, the results of this study reinforce that NLR correlates significantly with CTP score and other biochemical indicators of hepatic dysfunction, highlighting its potential as a rapid and reliable biomarker of disease severity in decompensated cirrhosis.

5. Conclusion

The neutrophil-to-lymphocyte ratio (NLR) shows a strong positive correlation with Child–Turcotte–Pugh (CTP) score in patients with decompensated liver cirrhosis. Higher NLR values reflect greater systemic inflammation and hepatic dysfunction, signifying a worse prognosis.

NLR is a simple, non-invasive, cost-effective marker that can be used alongside CTP or MELD scores for rapid assessment of cirrhosis severity in resource-limited settings. Routine inclusion of NLR in laboratory work-ups may facilitate early risk stratification and improved patient care.

Conflict of Interest

The authors declare no conflict of interest.

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