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Original Article

Neutrophil to Lymphocyte Ratio and its Correlation with Severity of Decompensated Liver Disease

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Abstract

Introduction: The neutrophil-to-lymphocyte ratio (NLR) is commonly used as a marker of inflammation and is independently associated with adverse clinical outcomes in patients with liver cirrhosis. This study aimed to assess the correlation between the NLR and complications in patients with decompensated liver disease (DCLD), including its role in determining disease severity.

Methods: This observational descriptive study included 100 patients admitted to Kanyakumari Government Medical College Hospital over 12 months. Demographic data and laboratory findings were collected, and NLR was calculated. Complications were identified and correlated with NLR.

Results: No significant differences in the NLR were observed based on age, sex, or DCLD etiology. Most patients (35%) were aged 41–50 years, with a male predominance (76%). Alcoholic liver disease was the most common cause of death (64%). Splenomegaly was absent in 20% of patients, mild in 38%, moderate in 24%, and severe in 18%. The Child-Turcotte-Pugh (CTP) classification was 54% for the CTP-B and 46% for the CTP-C. NLR analysis showed 32% with NLR <2, 50% within to 2.1–5, and 18% >5.1. Patients with upper gastrointestinal (GI) bleeding, ascites, and hepatic encephalopathy had significantly higher NLR ($P = 0.006$, $P < 0.0001$, and $P = 0.001$, respectively). Higher NLR levels were correlated with more severe disease stages.

Conclusion: The NLR is a valuable inflammatory marker in DCLD and correlates with complications such as ascites, hepatic encephalopathy, and GI bleeding. Higher NLR values were observed in patients with more severe clinical presentation, supporting its role as a prognostic indicator of disease severity and potential progression.

Key words: Alcoholic liver disease, Cirrhosis, Decompensated liver disease, Hepatic failure, Neutrophil-to-lymphocyte ratio

INTRODUCTION

The neutrophil-to-lymphocyte ratio (NLR) is a key inflammatory marker and is independently associated with poor clinical outcomes in liver cirrhosis. Admissions to acute medical units due to decompensated cirrhosis are frequent, presenting substantial medical challenges and often leading to prolonged hospital stays. In-hospital mortality rates for these patients typically range from 10% to 20%.[1] Early and accurate identification of underlying causes, coupled with the initiation of appropriate treatments, is crucial for managing patients during the early stages of decompensated cirrhosis.[2]

Decompensated cirrhosis refers to the rapid deterioration of liver function in individuals with pre-existing cirrhosis, and it commonly manifests through symptoms such as jaundice, ascites, hepatic encephalopathy, hepatorenal syndrome, and variceal bleeding.[1] Various triggers can precipitate hepatic decompensation, including infections, gastrointestinal bleeding, excessive alcohol consumption, alcohol-related hepatitis, and drug-induced liver injury.[2] Remarkably, nearly 50% of cases remain without an identified cause, highlighting the importance of a thorough clinical assessment, including detailed history-taking, examination, and diagnostic investigations,

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to identify the underlying trigger and guide appropriate treatment strategies.[3]

The management of decompensated cirrhosis necessitates prompt recognition and diagnosis, with non-specialist healthcare providers remaining vigilant for clinical signs, such as jaundice, ascites, altered mental status, and bleeding. Early identification of the root cause through careful history-taking and diagnostic workup is essential for guiding treatment decisions. In the case of jaundice, management primarily focuses on addressing the underlying cause while providing supportive care, which includes close monitoring of liver function, fluid balance, and potential complications.[4] Non-specialists should also be proficient in managing ascites through measures such as sodium restriction, diuretics, and, in severe cases, paracentesis. Furthermore, recognizing and treating hepatic encephalopathy, understanding hepatorenal syndrome, and managing variceal hemorrhage are fundamental aspects of care in these patients.[1]

A study confirmed that NLR serves as an independent predictor of long-term survival, even when adjusted for established scoring systems such as the Child-Turcotte-Pugh (CTP) and model for end-stage liver disease (MELD) score.[5] Given its accessibility and cost-effectiveness, NLR shows potential as a widely applicable marker in routine clinical practice.[6]

NLR, a rising marker of systemic inflammation, has been associated with a broad range of medical conditions, including hypertension, diabetes, liver and renal failure, acute coronary syndromes, and metabolic syndrome.[6] However, some analyses of the study pointed out the lack of consideration for factors that could influence NLR levels, such as comorbidities, medications, and other underlying health conditions.[7] This underscores the need for further investigation into the relationship between NLR and the severity of decompensated liver disease, with particular attention to confounding factors that could affect its clinical utility.

Aim

This study aimed to assess the correlation between NLR and several complications in patients with decompensated liver disease.

MATERIALS AND METHODS

This observational descriptive study included 100 decompensated liver disease patients admitted to the medical wards of Kanyakumari Government Medical College Hospital, Kanyakumari, for 12 months. The study was approved by the Institutional Ethics Committee before initiation, and informed consent was obtained from all participants.

Inclusion Criteria

Patients aged 18–65 years with decompensated chronic liver disease were diagnosed based on their clinical history, examination, and ultrasound findings.

Exclusion Criteria

Patients with malignancy, immunosuppression (including those with HIV, solid organ transplant recipients, and post-splenectomy patients on steroids or chemotherapy), recent blood transfusions within the last 3 months, diabetes mellitus, dyslipidemia, autoimmune diseases, pregnancy, and sepsis were excluded.

Methods

Demographic data, including patient identity, history of specific diseases, and history of blood transfusions, were collected. Laboratory findings such as complete blood count, liver function tests, electrolyte levels, random and fasting glucose levels, lipid profile, and hemostasis function tests were recorded. Neutrophil and lymphocyte levels were calculated using the formula: (Absolute count of neutrophil)/(Absolute count of lymphocyte). Complications among patients were identified, and correlations between the calculated NLR and clinical outcomes were analyzed.

Statistical Analysis

The study employed descriptive statistics (frequencies and percentages) to summarize the data. Categorical variables were assessed using the Chi-squared test. Significance was defined as $P < 0.05$, using a two-tailed test. Data analysis was performed using IBM Statistical Package for Social Sciences version 21.0.

RESULTS

Our study included 100 patients, with most (35%) aged 41–50 years, followed by 51–60 years (30%). Patients under 40 years comprised 15%, while those over 61 years made up 20%. Males comprised the majority (76%), while females accounted for 24%. Alcoholic liver disease was the leading cause (64%), followed by hepatitis B virus (8%), non-alcoholic steatohepatitis (8%), cryptogenic (7%), hepatitis C virus (5%), autoimmune (3%), Wilson disease (3%), and biliary cirrhosis (2%).

Splenomegaly was absent in 20% of the patients, mild in 38%, moderate in 24%, and severe in 18%. CTP classification was 54% in CTP-B (moderate impairment) and 46% in CTP-C (severe dysfunction). NLR analysis

revealed 32% with NLR <2 (low inflammation), 50% within 2.1–5 (moderate), and 18% >5.1 (high inflammation) [Table 1].

Abdominal distention was reported in all the patients (100%). Jaundice was reported in 39 patients (39%); 61 patients (61.0%) reported no incidence of jaundice. Fever was observed in 26 patients (26%), and 74 patients had no fever. Upper gastrointestinal (UGI) bleeding was reported in 54 patients (54%), while 46 patients reported no such incidence. The UGI bleeding assessment revealed that 21% of the patients had UGI bleeding, whereas 79% did not report any incidence of UGI bleeding. Ascites were observed in 29 patients (29%), and 71 patients (71%) reported no incidences. Serious hepatic encephalopathy was observed in 13 patients (13%), and no incidence of hepatic encephalopathy was observed in 87 patients [Table 2].

Most patients across the different age groups had an NLR between 2.1 and 5, with no significant variation ($P = 0.527$). Gender also did not significantly impact NLR distribution ($P = 0.129$); however, more females (37.5%) had an NLR <2 than males (30.3%), while a higher percentage of males (22.4%) had an NLR >5.1. Among the patients with alcoholic liver disease, 53.1% had an NLR between 2.1 and 5, with no significant difference observed ($P = 0.678$). Similarly, the distribution of NLR across different etiologies of DCLD showed no significant differences, with most autoimmune-related DCLD patients (66.7%) falling within the 2.1–5 range.

There were no significant differences in NLR distribution among patients with jaundice ($P = 0.861$) or fever ($P = 0.901$). Likewise, splenomegaly severity did not influence NLR distribution ($P = 0.983$), as the percentages remained relatively stable across different severity levels. Although the study found no significant differences in NLR distribution between the CTP-B and CTP-C groups ($P = 0.231$), patients with higher NLR values (>5.1) had more severe complications, including hepatic encephalopathy and UGI bleeding. This suggests that an elevated NLR may reflect increasing systemic inflammation and worsening disease severity, warranting further investigation into its role as a prognostic marker alongside established severity scores, such as MELD and CTP.

Patients with UGI bleeding had a significantly higher proportion of NLR >5.1 (38.1%) than those without UGI bleeding (12.7%) ($P = 0.006$). Additionally, NLR distribution differed significantly in patients with ascites ($P < 0.0001$), where only 6.9% had an NLR <2, whereas 37.9% had an NLR >5.1. Hepatic encephalopathy was also significantly associated with elevated NLR levels ($P = 0.001$), as 53.8% of the affected patients had an NLR >5.1 compared to 12.6% of those without hepatic encephalopathy [Table 3].

Table 1: Demographic and clinical characteristics

Parameters	Frequency (%)
Age (in years)	
<40	15 (15)
41–50	30 (30)
51–60	35 (35)
>61	20 (20)
Sex	
Female	24 (24)
Male	76 (76)
Cause for DCLD	
Alcoholic	64 (64)
Autoimmune	3 (3)
Biliary cirrhosis	2 (2)
Cryptogenic	7 (7)
HBV	8 (8)
HCV	5 (5)
NASH	8 (8)
Wilson	3 (3)
Splenomegaly	
Absent	20 (20)
Mild	38 (38)
Moderate	24 (24)
Severe	18 (18)
CTP	
B	54 (54)
C	46 (46)
NLR	
<2	32 (32)
2.1–5	50 (50)
>5.1	18 (18)

DCLD: Decompensated liver disease, HBV: Hepatitis B virus, NASH: Non-alcoholic steatohepatitis, HCV: Hepatitis C virus, NLR: Neutrophil-to-lymphocyte ratio, CTP: Child-Turcotte-Pugh

DISCUSSION

In our study, 100 decompensated liver disease patients comprised a predominant age group of 41–60 years (35%) and a male majority (76%). The relationship between NLR and a range of demographic and clinical variables

Table 2: Clinical symptoms and conditions

Parameters	Frequency (%)	
Abdominal distension	No	0
	Yes	100 (100)
Jaundice	No	61 (61)
	Yes	39 (39)
Fever	No	74 (74)
	Yes	26 (26)
UGI bleeding	No	46 (46)
	Yes	54 (54)
UGI bleeding	No	79 (79)
	Yes	21 (21)
Ascites	No	71 (71)
	Yes	29 (29)
Hepatic encephalopathy	No	87 (87)
	Yes	13 (13)

UGI: Upper gastrointestinal

Table 3: Comparison between NLR and clinical characteristics

Parameters	NLR			*P*-value
	<2 (%)	2.1–5 (%)	>5.1 (%)	
Age (in years)				
<40	5 (33.3)	9 (60)	1 (6.7)	0.527
41–50	9 (30)	13 (43.3)	8 (26.7)	
51–60	11 (31.4)	20 (57.1)	4 (11.4)	
>61	7 (35)	8 (40)	5 (25)	
Sex				
Female	9 (37.5)	14 (58.3)	1 (4.2)	0.129
Male	23 (30.3)	36 (47.4)	17 (22.4)	
Cause for DCLD				
Alcoholic	18 (28.1)	34 (53.1)	12 (18.8)	0.678
Autoimmune	1 (33.3)	2 (66.7)	0	
Biliary cirrhosis	1 (50)	1 (50)	0	
Cryptogenic	3 (42.9)	3 (42.9)	1 (14.3)	
HBV	4 (50)	1 (12.5)	3 (37.5)	
HCV	3 (60)	2 (40)	0	
NASH	2 (25)	5 (62.5)	1 (12.5)	
Wilson	0	2 (66.7)	1 (33.3)	
Jaundice				
No	19 (31.1)	30 (49.2)	12 (19.7)	0.861
Yes	13 (33.3)	20 (51.3)	6 (15.4)	
Fever				
No	23 (31.1)	37 (50)	14 (18.9	0.901
Yes	9 (34.6)	13 (50)	4 (15.4)	
UGI bleeding				
No	15 (32.6)	24 (52.2)	7 (15.2)	0.796
Yes	17 (31.5)	26 (48.1)	11 (20.4)	
Splenomegaly				
Absent	5 (25)	10 (50)	5 (25)	0.983
Mild	13 (34.2)	19 (50)	6 (15.8)	
Moderate	8 (33.3)	12 (50)	4 (16.7)	
Severe	6 (33.3)	9 (50)	3 (16.7	
CTP				
B	12 (22.2)	32 (59.3)	10 (18.5)	0.231
C	20 (43.5)	18 (39.1)	8 (17.4)	
UGI bleeding				
No	30 (38)	39 (49.4)	10 (12.7)	0.006
Yes	2 (9.5)	11 (52.4)	8 (38.1)	
Ascites				
No	30 (42.3)	34 (47.9)	7 (9.9)	<0.0001
Yes	2 (6.9)	16 (55.2)	11 (37.9)	
Hepatic encephalopathy				
No	31 (35.6)	45 (51.7)	11 (12.6)	0.001
Yes	1 (7.7)	5 (38.5)	7 (53.8)	

DCLD: Decompensated liver disease, HBV: Hepatitis B virus, NASH: Non-alcoholic steatohepatitis, HCV: Hepatitis C virus, NLR: Neutrophil-to-lymphocyte ratio, CTP: Child-Turcotte-Pugh, UGI: Upper gastrointestinal

revealed no differences in NLR distribution among diverse age cohorts, sexes, or specific etiologies of DCLD. This observation highlights the intricate and multifaceted nature of inflammatory processes in DCLD, which appear to be influenced by many factors rather than by demographic characteristics or the etiology of liver disease alone. This aligned with Sungkar *et al*., where no significant difference was reported between NLR and demographic or clinical characteristics, such as age, sex, etiologies of DCLD, and symptoms, and chronic alcoholism was the primary cause of liver cirrhosis in most patients.[8]

Abdominal distention was observed in patients with DCLD. However, the occurrence of jaundice, fever, and UGI bleeding varies among patients, showing diverse clinical manifestations associated with DCLD. The same clinical characteristics were reported by Sharma *et al*., where abdominal distention, fever, and UGI were most observed in patients with DCLD.[9] In addition, Prahraj and Anand, Sahani and Das reported that clinical conditions such as jaundice and fever are implications of ongoing infections in DCLD with severe disease progression and can lead to ascites.[10,11]

We found splenomegaly, a common feature of liver disease with varying degrees of severity, with 38% of the cases presenting as mild, 24% as moderate, and 18% as severe. Kanakamedala *et al*. found Splenomegaly can be observed in patients with DCLD who have inadequate management.[12] Sultan *et al*. reported that lymphadenopathy was the primary finding in 43.1% of cases, followed by splenomegaly (23.5%) and hepatomegaly (21.5%).[13] The distribution of patients in the CTP classification was balanced between the CTP-B and CTP-C classifications, suggesting a mixed range of liver impairment within the study population. However, Sungkar *et al*. and Tanoglu and Karagoz stated that the NLR is significantly associated with CTP scores and can be used to predict DCLD.[8,14]

NLR has proven to be a useful inflammatory marker, with 32% of patients displaying an NLR <2, 50% exhibiting an NLR between 2.1 and 5, and 18% with an NLR >5.1. This categorization indicated varying levels of inflammatory response within the study population, implying potential implications for prognosis and treatment. Zhang *et al*. found that NLR was higher in decompensated patients than in compensated cirrhosis patients. This is due to the intricate relationship between systemic inflammation and the immune system in advanced or decompensated cirrhosis.[15]

In our study, intriguing correlations between NLR and specific complications of DCLD were observed; a distinct NLR distribution was observed in patients with ascites, showing a significant disparity between those with and without ascites. This suggests that the NLR may be a prognostic marker for ascites development in DCLD. In addition, patients with hepatic encephalopathy showed a significant difference in NLR distribution compared to those without this complication, implying a potential connection between systemic inflammation and the emergence of hepatic encephalopathy. Probowati *et al*. found a positive correlation between NLR and CTP scores among 33 patients.[16] Biyak *et al*. discovered that NLR is an independent predictor of mortality in Cox regression models, and a significant correlation was obtained with CTP score ($r = 0.25$; $P = 0.002$).[3] In Oh *et al*., NLR determined a linear relationship with CTP score as a prognostic factor for HCC in a different population with HCC.[17]

This study found a strong association between NLR and UGI bleeding in patients with DCLD, highlighting its potential as a predictive marker. A significant difference in NLR distribution was observed, correlating with disease severity and outcome. Additionally, NLR was positively correlated with CTP score, indicating systemic inflammation. Similar findings by Zhang *et al.* linked an elevated NLR to increased liver disease severity and higher short-term mortality. Other scores, such as MELD, also predict 30-day mortality.[15] Sun *et al.* further supported NLR as an independent marker for disease severity and short-term outcomes in DCLD patients.[18]

The association between NLR and complications, such as ascites, hepatic encephalopathy, and UGI bleeding, highlights its clinical relevance. These findings emphasize the importance of monitoring inflammatory markers in DCLD management and warrant further research into their potential roles in therapeutic strategies.

CONCLUSION

This study underscores the prognostic significance of NLR in DCLD, emphasising its correlation with key complications such as ascites, hepatic encephalopathy, and UGI bleeding. These findings suggest that NLR serves as a valuable inflammatory marker, independent of age, sex, or disease etiology, and could aid in risk stratification and complication management. Given its strong association with disease severity, the NLR may be a useful adjunct in clinical decision-making, helping to identify high-risk patients and guide timely interventions. However, further research is needed to validate these findings, explore the underlying pathophysiological mechanisms, and establish standardized thresholds for clinical application. Prospective studies could enhance the understanding of NLR's role of the NLR in predicting outcomes and optimizing management strategies for patients with DCLD. By integrating the NLR into routine assessments, clinicians may improve prognostication and treatment strategies, ultimately enhancing patient outcomes in decompensated liver disease.

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REFERENCES

1. Mansour D, McPherson S. Management of decompensated cirrhosis. Clin Med (Lond) 2018;18:s60-5.
2. Moreau R, Jalan R, Gines P, Pavesi M, Angeli P, Cordoba J, *et al*. Acute-on-chronic liver failure is a distinct syndrome that develops in patients with acute decompensation of cirrhosis. Gastroenterology 2013;144:1426-37.e9.
3. Cholongitas E, Arsos G, Goulis J, Birtsou C, Haidich AB, Nakouti T, *et al*. Glomerular filtration rate is an independent factor of mortality in patients with decompensated cirrhosis. Hepatol Res 2014;44:E145-55.
4. Ginés P, Quintero E, Arroyo V, Terés J, Bruguera M, Rimola A, *et al*. Compensated cirrhosis: Natural history and prognostic factors. Hepatology 1987;7:122-8.
5. Biyik M, Ucar R, Solak Y, Gungor G, Polat I, Gaipov A, *et al*. Blood neutrophil-to-lymphocyte ratio independently predicts survival in patients with liver cirrhosis. Eur J Gastroenterol Hepatol 2013;25:435-41.
6. Balta S, Cakar M, Demirkol S, Arslan Z, Akhan M. Higher neutrophil to lymhocyte ratio in patients with metabolic syndrome. Clin Appl Thromb Hemost 2013;19:579.
7. Karaman M, Balta S, Seyit Ahmet AY, Cakar M, Naharci I, Demirkol S, *et al*. The comparative effects of valsartan and amlodipine on vWf levels and N/L ratio in patients with newly diagnosed hypertension. Clin Exp Hypertens 2013;35:516-22.
8. Sungkar T, Rozi MF, Dairi LB, Zain LH. Neutrophil-to-lymphocyte ratio (NLR) and its correlation with severity of decompensated liver cirrhosis based on child-turcotte pugh (CTP) score. J Clin Diagn Res 2019;13: OC29-31.
9. Sharma S, Ahuja V. Liver abscess: Complications and treatment. Clin Liver Dis (Hoboken) 2021;18:122-6.
10. Prahraj D, Anand AC. Tropical liver diseases: An overview. Clin Liver Dis (Hoboken) 2021;18:138-42.
11. Sahani S, Das D. Neutrophil-to-lymphocyte ratio and its correlation with child turcotte pugh (ctp) score in predicting the severity of decompensated liver cirrhosis. J Assoc Physicians India 2022;70:11-2.
12. Kanakamedala S, Danak SU, Conway EJ, Kotla V. Cirrhosis with splenomegaly and pancytopenia complicating a concurrent diagnosis of acute lymphoblastic leukaemia. Cureus 2022;14:e31707.
13. Sultan S, Irfan SM, Parveen S, Mustafa S. Acute lymphoblastic leukemia in adults - an analysis of 51 cases from a tertiary care center in Pakistan. Asian Pac J Cancer Prev 2016;17:2307-9.
14. Tanoglu A, Karagoz E. Neutrophil-to-lymphocyte ratio an emerging prognostic factor of cirrhosis? Eur J Gastroenterol Hepatol 2014;26:362.
15. Zhang H, Sun Q, Mao W, Fan J, Ye B. Neutrophil-to-lymphocyte ratio predicts early mortality in patients with HBV-related decompensated cirrhosis. Gastroenterol Res Pract 2016;2016:4394650.
16. Probowati W, Bayupurnama P, Ratnasari N. Correlation between neutrophil to lymphocyte ratio with Child Turcotte Pugh in liver cirrhosis patients. Actainterna. J Intern Med 2017;6:28-35.
17. Oh BS, Jang JW, Kwon JH, You CR, Chung KW, Kay CS, *et al*. Prognostic value of C-reactive protein and neutrophil-to-lymphocyte ratio in patients with hepatocellular carcinoma. BMC Cancer 2013;13:78.
18. Sun J, Guo H, Yu X, Zhu H, Zhang X, Yang J, *et al*. A neutrophil-to-lymphocyte ratio-based prognostic model to predict mortality in patients with HBV-related acute-on-chronic liver failure. BMC Gastroenterol 2021;21:422.

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