ORIGINAL ARTICLE

JIACM 2025; 26 (3): 126-31

Neutrophil-to-Lymphocyte Ratio as a Prognostic Marker in Alcoholic Liver Cirrhosis Complicated by Hepatic Encephalopathy: A Prospective Observational Study from North India

Ambadas Rathod, Pritam Singh**, Kamal Singh**, Anshu Palta***, Parth Sharma**

Abstract

***Background:** The neutrophil-to-lymphocyte ratio (NLR) reflects systemic inflammation in cirrhosis, but its prognostic role in Indian patients with alcoholic cirrhosis (ALC) and hepatic encephalopathy (HE) remains unclear. We compared NLR with Model for End-stage Liver Disease (MELD) and Discriminant Function (DF) scores for predicting 90-day mortality.*

***Methods:** This prospective cohort study enrolled 60 male patients with ALC and HE at GMCH, Chandigarh between July 2023 and January 2025. NLR, MELD, and DF scores were calculated at the time of admission. Outcomes were tracked for 90 days. ROC analysis and Cox regression were used to assess predictive performance.*

***Results:** Mean NLR was comparable between survivors (9.7 ± 11.3) and non-survivors (10.1 ± 7.3; p = 0.885). All markers showed poor discrimination (AUC: NLR = 0.587, MELD = 0.633, DF = 0.626; p >0.05). Sensitivity was high (NLR: 95.5%; MELD: 100%), but specificity was low (13.2 - 15.8%). Negative Predictive Value (NPV) was robust (NLR: 85.7%; MELD: 100%), suggesting utility in ruling out mortality.*

***Conclusion:** NLR did not significantly predict mortality but demonstrated high NPV, supporting its role in risk stratification. MELD/DF scores also lacked precision, underscoring the need for more accurate prognostic tools in ALC with HE.*

Introduction

Alcoholic Liver Disease (ALD) is a common global health issue that comprises a wide spectrum of conditions ranging from mild hepatic steatosis to cirrhosis and hepatocellular carcinoma (HCC). Long-term consumption of alcohol initiates macrovesicular fatty changes, followed by hepatic necrosis. Finally, it reaches irreversible diffuse fibrosis, disrupting chronic liver disease (CLD), characterised by parenchymal distortion and regenerative nodules[1].

The primary cause of cirrhosis is alcohol in Indian adults (43.2%), followed by NAFLD (14.4%), Hepatitis B virus (HBV) (11.5%), and Hepatitis C virus (HCV) (6.2%). Viral hepatitis-related cirrhosis is declining, while alcohol and NAFLD-related cases are rising[2].

ALD contributes significantly to the worldwide illness burden and is one of the main reasons for hospitalisation. The Asia-Pacific area is responsible for 54.3% of all cirrhosis-related deaths, making cirrhosis the primary cause of liver-related mortality[1].

Liver cirrhosis is characterised by an early phase of compensation which has a better outcome, followed by an advanced phase of decompensation which is associated with complications such as upper GI bleeding, portal hypertension, and HE[3].

HE is a reversible neuropsychiatric condition linked to liver cirrhosis. As a major complication of cirrhosis, HE contributes to high mortality rates and imposes a substantial economic burden on healthcare systems[1,4]. Studies indicate that around 30 - 40% of cirrhosis patients develop HE as a result of impaired liver function and portosystemic shunting[4]. The condition manifests with prominent clinical symptoms, leading to a worsened prognosis and a marked deterioration in patients' quality-of-life[5]. Research has shown that overt hepatic encephalopathy carries a grim outlook, with one-year mortality rates reaching 64%, escalating to as high as 85% within three years[2,6].

The course of HE is related to systemic inflammation activation and immune disorders[3]. Immunodeficiency and systemic inflammation are concurrent variables that exacerbate liver cirrhosis. Inflammation is indicated by the increased production of pro-inflammatory cytokines and their increased blood levels[7]. The lymphocyte count is related to the regulatory immune pathway, whereas the neutrophil count provides information on ongoing inflammation. Raised NLR has been demonstrated to predict medium and long-term mortality in liver cirrhosis more recently[7]. NLR is a simple inflammatory marker derived from

**Junior Resident, **Professor, Department of General Medicine, ***Professor, Department of Pathology, Government Medical College and Hospital (GMCH), Chandigarh - 160 030.*

Corresponding Author: Dr Ambadas Rathod, Junior Resident, Department of General Medicine, Government Medical College and Hospital (GMCH), Chandigarh - 160 030. Tel: 7989799724, E-mail: ambadasrathod100@gmail.com

differential white blood cell count. It is linked to poor prognosis in several cancers, (e.g., colorectal, hepatocellular, and pancreatic) and cardiovascular conditions, including peripheral vascular disease, coronary artery ectasia, and hypertension[8].

Limited data is available in the literature on the usage of NLR as a prognostic marker in patients of ALC with HE, particularly in the context of the Indian population. Traditional scores such as MELD and Child Turcotte Pugh (CTP) did not include the inflammatory state of the patient. Hence, we conducted this study to predict the outcome of patients of alcoholic cirrhosis with HE by using NLR as a prognostic marker.

Material and Methods

Study population

A total of 60 patients diagnosed with alcoholic cirrhosis and hepatic encephalopathy were enrolled in this prospective cohort study, conducted at Government Medical College and Hospital, Sector 32, Chandigarh, India.

Inclusion criteria

- Age >18
- Patients diagnosed with alcoholic cirrhosis with hepatic encephalopathy (West Haven Criteria).
- Radiological imaging findings suggestive of Chronic Liver Disease (CLD) on USG.

Exclusion criteria

- Pregnant and breastfeeding female patients
- Malignancies such as HCC
- Other causes of Liver cirrhosis (viral hepatitis, autoimmune hepatitis, drug-induced liver disease).
- Other causes of altered mental status (uremic encephalopathy, CO2 narcosis, hypoglycaemia).

The study protocol conformed to the ethical guidelines of the Declaration of Helsinki and was approved by the Ethical Review Committee of the GMCH Chandigarh.

Data Collection and Clinical Definitions

- Clinically relevant variables were collected from all enrolled patients, including patient characteristics, complications and laboratory data. Routine investigations (CBC, RFT, LFT, and Coagulogram) were sent for the patients who enrolled in the study. MELD and DF scores were calculated on the day of enrolment.
- NLR ratio is calculated by using differential WBC count for Neutrophils and Lymphocytes.
- The performance of the NLR ratio in predicting mortality was compared with the Discriminant Function score and MELD score at the end of 90 days.
- Alcoholic liver cirrhosis was diagnosed using clinical history, laboratory tests, and ultrasonographic findings.
- Hepatic encephalopathy was diagnosed following American Association for the Study of Liver Diseases (AASLD) guidelines and the West Haven Criteria (WHC). Affected patients exhibited notable personality shifts, erratic behaviour, dyspraxia, disorientation, lethargy, and confusion regarding time and space, with some progressing to coma.

Statistical analysis

Mortality in patients of alcoholic cirrhosis with hepatic encephalopathy from the day of admission till 90 days post-discharge was described using proportions, percentages, and distribution. Significance of differences between mean values of NLR, MELD score and Discriminant function were tested by using the Kolmogorov–Smirnov test or Mann–Whitney 'U' test. The chi-square test was employed to assess the significance of associations between clinical outcomes and independent variables. Sensitivity, specificity, area under the curve (AUC), positive predictive value (PPV), and negative predictive value (NPV) were calculated to evaluate the NLR in comparison to the MELD score and DF score. Data analysis was carried out using SPSS 26.0 software.

Observation and Results

Table I: Clinical features of patients with ALC and HE

Variable	Total (n = 60)	Survival group (n = 38)	Non-survival group (n = 22)	P*
Age (years)	47.6 ± 10.9	46.4 ± 10.4	49.8 ± 11.7	0.266
BMI (kg/m^2)	26.0 ± 1.9			
Diabetes	12 (20%)	6 (15.8%)	6 (27.3%)	0.284
Hypertension	15 (25%)	6 (15.8%)	9 (40.9%)	*0.030*
CAD	3 (5%)	0 (0%)	3 (13.6%)	*0.020*
CKD	4 (6.7%)	2 (5.3%)	2 (9.1%)	0.567
Alcohol consumption duration	19.6 ± 7.2	18.4 ± 6.4	21.7±8.2	0.113
Precipitating factors for HE				
SBP	16 (26.7%)	10 (26.3%)	6 (27.3%)	0.936
UGIB	21 (35%)	15 (39.5%)	6 (27.3%)	0.340
Infections other than SBP	9 (15%)	6 (15.8%)	3 (13.6%)	
Constipation	10 (16.7%)	4	6	0.131
Other causes	4 (6.6%)	1	3	0.099

HE		2 (1.3-2)	2 (1.8-3)	0.114
Grade 1	15 (25%)	10 (26.3%)	5 (22.7%)	
Grade 2	30 (50%)	21 (55.3%)	9 (40.9%)	
Grade 3	12 (20%)	5 (22.7%)	7 (18.4%)	
Grade 4	3 (5%)	0 (0%)	3 (13.6%)	
Hypotension on presentation		0 (0%)	4 (%)	0.007
HB (gm/dL)	8.5 ± 2.3	8.8 ± 2.1	8 ± 2.6	0.217
TLC (x10^3/µL)	13.3 ± 8.06	13.7 ± 8.9	12.7 ± 6.6	0.638
PLT (x10^3/µL)	110.2 ± 71.7	118.7 ± 66.4	97.4 ± 80.9	0.302
NLR	9.91± 9.99	9.7 ± 11.3	10.1± 7.3	0.885
Total Bil (mg/dL)	10.36 ± 11.70	8.7 ± 10.1	13.2 ± 13.8	0.217#
SGOT ((U/L)	240.73 ± 434.24	217.7 ± 479.2	280.6 ± 350.1	0.217#
SGPT ((U/L)	123.73 ± 328.93	140.8 ± 393	94.2 ± 174.5	0.939#
Albumin (g/dL)	2.33± 0.5	2.4± 0.5	2.3± 0.5	0.379
Creatinine (mg/dL)	1.93 ± 1.80	1.8 ± 1.9	2.1 ± 1.7	0.480#
Sodium (mEq/L)	133.58 ± 8.28	133.6 ± 8.3	133.5 ± 8.5	0.842#
PT (seconds)	21.85 ± 8.79	23.6 ± 12	20.8 ± 6.2	0.236
INR	1.71 ± 0.894	1.9 ± 1.2	1.6 ± 0.6	0.196
MELD Score	23.8 ± 8.7	22.3 ± 8.6	26.3 ± 8.6	0.87
DF score	44.7 ± 38.3	37.5 ± 24.9	57.2 ± 52.5	0.55

Patients were divided into two groups based on 90-day survival outcomes: survivors (n = 38) and non-survivors (n = 22). Their clinical characteristics are presented in Table I.

The survival group had a mean age of 46.4 ± 10.4 years, compared to 49.8 ± 11.7 years in the non-survival group. The most prevalent co-morbidities in the study population were hypertension (25%), diabetes mellitus (20%), followed by chronic kidney disease (6.7%) and coronary artery disease (5%).

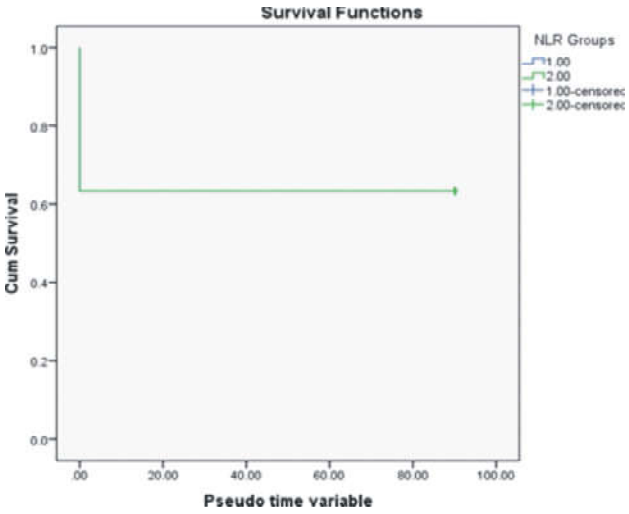

Fig. 1: *Kaplan-Meier survival analysis for 90-day mortality by NLR.*

The survival group had consumption of harmful alcohol for an average of 18.4 ± 6.4 years, while the non-survival group consumed it for an average of 21.7 ± 8.2 years.

The major precipitating factors for HE included: upper gastrointestinal bleeding (UGIB) (35%), spontaneous bacterial peritonitis (SBP) (26.7%), non-SBP infections (15%), constipation (16.7%) and other causes (6.6%). Regarding the severity of HE at presentation, grade 2 HE was the most common (50%), followed by grade 1 HE (25%), grade 3 HE (20%) and grade 4 HE (5%). The mean NLR showed no significant difference between groups (survivors: 9.7 ± 11.3 vs non-survivors: 10.1 ± 7.3; p = 0.885), despite being slightly elevated in non-survivors.

Patients were stratified by median NLR (≤7.38 vs >7.38; 30 each). Both groups had 11 deaths (63.3% 90-day survival). The Kaplan-Meier survival analysis (Fig. 1) found no statistically significant difference between groups (p >0.05), indicating that median-dichotomised NLR does not serve as a reliable prognostic marker for 90-day survival in this population.

Table II: Correlation between baseline NLR and 90-day mortality risk

NLR	Mortality at 90 days	Total	Chi-square value	p value
	Non-survivor	Survivor		
	N (%)	N (%)	N (%)	1.709 0.191
<2.59	1 (4.5)	6 (15.8)	7 (11.7)	
≥2.59	21 (95.5)	32 (84.2)	53 (88.3)	
Total	22 (100.0)	38 (100.0)	60 (100.0)	

The analysis examined the relationship between neutrophil-to-lymphocyte ratio (NLR) dichotomised at a cut-off of 2.59 and 90-day mortality. The association was evaluated using a Chi-square test ($\chi^2 = 1.709$, p = 0.191). While a trend towards increased 90-day mortality was observed in patients with NLR ≥2.59 (Table II), this difference did not reach statistical significance. Thus NLR dichotomised at this threshold does not demonstrate a significant association with 90-day mortality in our study population.

Table III: Cox proportional hazards regression analysis of 90-day mortality.

Predictor Variable	Hazard Ratio (HR)	p-value
Age (years)	1.03	0.126
MELD Score	1.03	0.331
DF Score	1.005	0.313
NLR	0.996	0.875

Table III shows a Cox proportional hazards regression model that was used to examine the relationship between baseline

factors and 90-day death rates. The analysis revealed the following trends, though none reached statistical significance:

- A one-year increase in age was found to increase the risk of death by 3% (HR = 1.03, p = 0.126).
- MELD Score: A one-unit increase in MELD score increased the risk of death by 3% (HR = 1.03, p = 0.331).
- A one-unit increase in the DF score led to a 0.5% increase in the risk of death (HR = 1.005, p = 0.313).
- A one-unit rise in NLR resulted in a 0.4% reduction in the risk of death (HR = 0.996, p = 0.875).

In this analysis, one of the analysed variables (age, MELD score, DF score, or NLR) showed statistically significant associations with 90-day mortality in this cohort. Further studies with larger cohorts may be needed to identify robust predictors of short-term survival in this population.

Fig. 2 shows poor mortality prediction by all markers: NLR (AUC = 0.587, p = 0.263), MELD (AUC = 0.633, p = 0.089), and DF scores (AUC = 0.626, p = 0.107), with none reaching statistical significance. All AUCs neared 0.5, indicating negligible discrimination between survivors and non-survivors. No variable demonstrated statistically significant predictive capability.

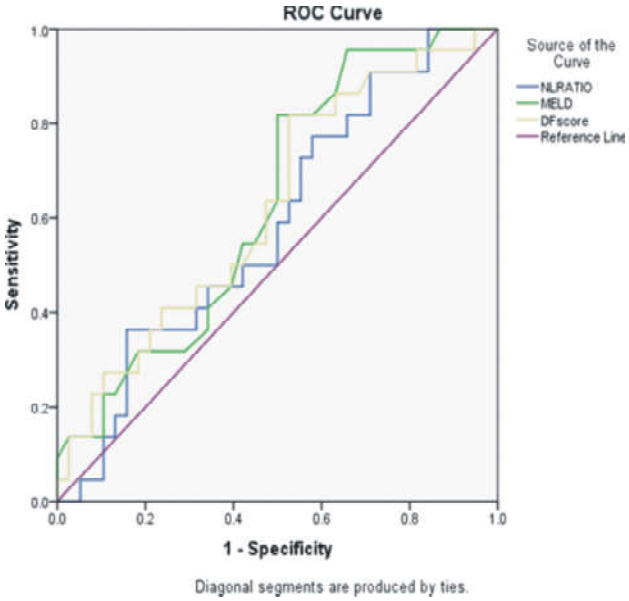

Fig. 2: *Assessment of 90-day mortality prediction using receiver operating characteristic (ROC) curves.*

Table IV: Comparison of predictive performance for 90-day mortality

Metric	NLR (Cut-off: 2.595)	MELD (Cut-off: 13)	DF Score (Cut-off: 10.85)
Sensitivity (%)	95.5	100	95.5
Specificity (%)	15.8	13.2	15.8
PPV (%)	39.6	40	39.6
NPV (%)	85.7	100	85.7

All three variables showed excellent sensitivity (MELD: 100%; NLR/DF: 95.5%) but poor specificity (MELD: 13.2%; NLR/DF: 15.8%) as shown in Table IV. While they effectively identified true positives (high sensitivity), false positives were common (low specificity). Positive predictive values were uniformly modest (~40%), indicating limited reliability for mortality prediction. However, negative predictive values were stronger (MELD: 100%; NLR/DF: 85.7%), suggesting better utility in ruling out mortality risk. The MELD score demonstrated perfect sensitivity and NPV, but all markers suffered from low specificity, limiting their clinical utility as standalone predictors. These patterns highlight a trade-off between sensitivity and specificity in mortality prediction.

Discussion

Alcoholic cirrhosis complicated by hepatic encephalopathy presents a significant clinical challenge, marked by high morbidity and mortality. Accurate prognostication in these patients is essential for optimizing clinical management and resource allocation. This study aimed to assess the prognostic value of the neutrophil-to-lymphocyte ratio (NLR) in predicting 90-day mortality among patients with alcoholic cirrhosis and HE, thereby addressing the utility of NLR as a potential risk stratification tool.

Systemic inflammation, as reflected by NLR, has emerged as a mortality predictor in various liver diseases. Contemporary research has established a significant association between elevated NLR and adverse outcomes in advanced liver disease. Specifically, Shi *et al* demonstrated its prognostic value in overt hepatic encephalopathy[9], while Liu *et al* validated its mortality prediction in decompensated cirrhosis[10]. These findings collectively highlight the clinical utility of inflammatory biomarkers for risk stratification in this high-risk patient population.

In addition to evaluating NLR's prognostic value, this study also aimed to compare its predictive performance against established clinical scoring systems (MELD and DF scores) for 90-day mortality. The MELD score, as evident by studies like Bohra *et al* has been routinely employed in clinical practice to evaluate disease progression and predicts

mortality risk in patients with cirrhosis[11].

In our cohort, although higher NLR values were observed in non-survivors compared to survivors, this difference did not achieve statistical significance (p = 0.885). The mean age of participants was 47.6 ± 10.9 years, consistent with the age range typically reported for alcoholic liver disease, which often presents in the fourth to fifth decades of life. Compared to our findings, Sahani *et al* reported a higher mean age (62.2 years), while Bohra *et al* reported a mean age of 57 years[11,12]. This relatively younger cohort may reflect regional variations in alcohol use patterns or earlier disease onset.

The predominant proportion of our study cohort (70%) had a BMI of 25.0 kg/m^2 or greater, indicating a high prevalence of overweight or obesity. The mean BMI of our study population was 26.0 ± 1.9 kg/m^2. In the study conducted by Berzigotti *et al*[13], the mean BMI was 27.9 ± 4.8 kg/m^2. Obesity can exacerbate liver inflammation and fibrosis, potentially influencing the severity of hepatic encephalopathy and overall prognosis[14].

In our study, all enrolled patients were male. This contrasts with the study conducted by Liu *et al* where 72.9% of the participants were male and 27.1% were female[10]. These findings highlight that alcoholic cirrhosis is more prevalent among males compared to females, likely due to higher rates of alcohol consumption among men in Indian society.

Our cohort demonstrated substantial co-morbidity burden, with hypertension predominating (25%), followed by diabetes mellitus (20%), while CAD (5%) and CKD (6.7%) were less prevalent. These findings align with Mukthinuthalapati *et al* reported atherosclerotic disease (89.7%), diabetes (27.4%), CKD (8.5%), and HF (9.1%) in similar patients[15].

Regarding alcohol exposure, survivors reported significantly shorter duration of consumption (18.4 ± 6.4 years) compared to non-survivors (21.7 ± 8.2 years, p = 0.113). Grade 2 HE was most frequent (30 patients: 21 survival, 9 non-survival), followed by Grade 1 (15: 10 survival, 5 non-survival), Grade 3 (12: 5 survival, 7 non-survival), and Grade 4 (3: all non-survival). Higher HE grades correlated with increased mortality. These findings differ from the study conducted by Shi *et al* which reported 68.2% of patients with Grade 2 HE, 25.1% with Grade 3 HE, and 6.6% with Grade 4 HE[9]. Similarly, Bajaj *et al*[16] noted that patients presenting with grade 3 - 4 hepatic encephalopathy demonstrated significantly increased 30-day mortality rates, reinforcing the prognostic significance of HE.

Notably, the mean NLR was 9.91 ± 9.9, with a wide range from 1.50 to 54.60. The mean NLR in the survival group was 9.77 and 10.16 in the non-survival group. These findings indicate significant haematological abnormalities in our cohort. Rice *et al*[17] also analysed haematological parameters, showing an increased risk of mortality with a rising NLR up to 8. Patients were divided into two groups using the median NLR (≤7.38 vs >7.38), with 30 patients in each. Over 90 days, 11 deaths occurred in both groups, yielding identical cumulative survival rates of 63.3%. Kaplan-Meier survival analysis revealed no statistically significant difference in 90-day mortality between NLR-stratified groups. These results suggest that NLR, when dichotomised at the median, does not predict short-term mortality in this cohort. This contradicts the findings of a study that suggested that NLR >4 was associated with a greater risk of 90-day mortality[18].

The analysis of serum electrolytes and renal function parameters showed a mean serum sodium level of 133.5 ± 8.5 mEq/L in the non-survival group and 133.6 ± 8.2 mEq/L in the survival group. This is inconsistent with a study where the mean sodium in the survival group was 136 mEq/L as compared to 132 mEq/L in the non-survival group[19].

The survival group had a slightly lower mean MELD score (22.3 ± 8.6) than the non-survival group (26.3 ± 8.6). This is in contrast to Mallik *et al's* findings, which showed that the median MELD score was 21.03 in the non-survival group and 10.36 in the survival group[20]. These findings align with Bohra *et al* who reported a median MELD score of 25, consistent with advanced liver disease in their study[11].

In our study, the non-survival group had a notably higher mean DF score (57.2 ± 52.5) than the survival group (37.5 ± 24.9). This is in contrast to Monsanto *et al's* findings, in which the mean DF score was 48 in the survival group and 96 in the deceased group[21].

The analysis revealed non-significant statistical correlation between dichotomised baseline NLR at a cut-off value of 2.59 and 90-day mortality. The NLR was less than 2.59 in 15.8% of survivors and 4.5% of non-survivors. Whereas, it was greather than 2.59 in 84.2% of the survivors and 95.5% of the non-survivors. The data suggests a trend where a larger proportion of patients with NLR ≥2.59 died after 90 days compared to those with NLR <2.59, although the difference is not statistically significant (p = 0.191). In contrast to Biyik *et al* results showing markedly worse survival for NLR ≥2.72 (p <0.001) and consistent independent mortality prediction (OR 1.2, 95% CI, 1.2 - 1.3), our analysis failed to replicate these significant associations at similar NLR thresholds[22]. Therefore, while the observed frequencies suggest a possible relationship, there is not enough evidence to conclude a statistically significant association between NLR (using this cut-off) and 90-day mortality.

In our study ROC curve analysis yielded optimal cut-off values of 2.59 for NLR, 13 for MELD, and 10.85 for DF scores.

However, these thresholds demonstrated inadequate discriminatory power for 90-day mortality prediction (all AUCs <0.65, p >0.05), potentially reflecting the study's limited sample size, population heterogeneity (variations in co-morbidities, age, and disease severity), or the dynamic nature of cirrhosis-related complications.

The MELD score demonstrated 100% sensitivity but very low specificity (13.2%), while NLR and DF scores each showed high sensitivity (95.5%) but similarly low specificity (15.8%). The positive predictive values (PPV) for all three markers were modest (~40%), suggesting limited reliability in confirming mortality risk. In contrast, the negative predictive values (NPV) were relatively high 100% for MELD and 85.7% for NLR and DF, indicating that negative results were more reliable in predicting survival.

These findings differ from prior studies. Mallik *et al* reported MELD sensitivity, specificity, PPV, and NPV of 55.38%, 93.33%, 87.8%, and 70.7%, respectively[20]. Similarly, Maccali *et al* found that at an NLR cut-off of 3.6, sensitivity was 69%, specificity was 65%, PPV was 38%, and NPV was 87% for 90-day mortality prediction[6]. While our results align with previous studies in terms of sensitivity and NPV, all three scores exhibited poor specificity and PPV, limiting their utility as standalone prognostic tools.

Conclusion

This study reveals that while the neutrophil-to-lymphocyte ratio (NLR) lacks significant predictive power for mortality in alcoholic cirrhosis patients with hepatic encephalopathy, its exceptionally high negative predictive value (85.7%) offers crucial clinical utility by reliably identifying low-risk patients who may not require intensive intervention. Although traditional scores (MELD/DF) similarly showed limited prognostic accuracy, NLR's strength in ruling out mortality risk provides a simple, cost-effective tool for risk stratification in resource-limited settings. These findings highlight both the challenges in prognostication for this high-mortality population and the potential for NLR to optimise clinical decision-making, while underscoring the urgent need for more robust predictive models through future multicenter validation studies.

References

1. Omar YA, Randhawa T, Attar B *et al*. Prognostic value of neutrophil-lymphocyte ratio in patients with severe alcoholic hepatitis. *Cureus* 2019; 11 (11).
2. Swaroop S, Vaishnav M, Arora U *et al*. Aetiological spectrum of cirrhosis in India: a systematic review and meta-analysis. *J Clin Exp Hepatol* 2024; 14 (2): 101291.
3. Maccali C, Augustinho FC, Zocche TL. Neutrophil-lymphocyte ratio predicts short-term mortality in patients hospitalised for acute decompensation of cirrhosis. *Arq Gastroenterol* 2021; 58: 131-8.
4. Younossi ZM, Wong G, Anstee QM. The global burden of liver disease. *Clin Gastroenterol Hepatol* 2023; 21 (8): 1978-91.
5. Mokdad AH, Forouzanfar MH, Daoud F *et al*. Global burden of diseases, injuries, and risk factors for young people's health during 1990 - 2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 2016; 387 (10036): 2383-401.
6. Sharma A, Nagalli S. Chronic liver disease. In: StatPearls [Internet]. StatPearls Publishing; 2023.
7. Chen L, Lou Y, Chen Y. Prognostic value of the neutrophil-to-lymphocyte ratio in patients with acute-on-chronic liver failure. *Int J Clin Pract* 2014; 68 (9): 1034-40.
8. Lin L, Yang F, Wang Y *et al*. Prognostic nomogram incorporating neutrophil-to-lymphocyte ratio for early mortality in decompensated liver cirrhosis. *Int Immunopharmacol* 2018; 56: 58-64.
9. Shi K, Huang Y, Zhang Q. Neutrophil–lymphocyte ratio and the risk of 30-day mortality in patients with overt hepatic encephalopathy. *Eur J Gastroenterol Hepatol* 2022; 34 (5): 529-36.
10. Liu J, Li H, Xia J *et al*. Baseline Neutrophil-to-Lymphocyte Ratio Is Independently Associated With 90-Day Transplant-Free Mortality in Patients With Cirrhosis. *Front Med* 2021; 8: 726950.
11. Bohra A, Worland T, Hui S. Prognostic significance of hepatic encephalopathy in patients with cirrhosis treated with current standards of care. *World J Gastroenterol* 2020; 26 (18): 2221-31.
12. Sahani S, Das D. Neutrophil to lymphocyte ratio (NLR) and its correlation with Child-Turcotte-Pugh (CTP) score in predicting severity of decompensated liver cirrhosis. *J Assoc Physicians India* 2022; 70: 11-2.
13. Berzigotti A, Garcia-Tsao G, Bosch J *et al*. Obesity is an independent risk factor for clinical decompensation in patients with cirrhosis. *Hepatology* 2011; 54 (2): 555-61.
14. Younossi ZM, Koenig AB, Abdelatif D. Global epidemiology of nonalcoholic fatty liver disease – Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology* 2016; 64 (1): 73-84.
15. Mukthinuthalapati PK, Syed M, Salazar M *et al*. Prevalence of co-morbidities in patients with cirrhosis admitted to US safety net hospitals and their impact on clinical phenotype of admission. *Am J Gastroenterol* 2018; 113: S566-7.
16. Bajaj JS, O'Leary JG, Tandon P *et al*. Hepatic encephalopathy is associated with mortality in patients with cirrhosis independent of other extrahepatic organ failures. *Clin Gastroenterol Hepatol* 2017; 15 (4): 565-74.
17. Rice J, Dodge JL, Bambha KM *et al*. Neutrophil-to-lymphocyte ratio associates independently with mortality in hospitalised patients with cirrhosis. *Clin Gastroenterol Hepatol* 2018; 16 (11): 1786-91.
18. Kalra A, Wedd JP, Bambha KM *et al*. Neutrophil to lymphocyte ratio correlates with proinflammatory neutrophils and predicts death in low model for end stage liver disease patients with cirrhosis. *Liver Transpl* 2017; 23 (2): 155-65.
19. Londono MC, Cardenas A, Guevara M. MELD score and serum sodium in the prediction of survival of patients with cirrhosis awaiting liver transplantation. *Gut* 2007; 56 (9): 1283-90.
20. Mallik M, Singhai A, Khadanga S. The significant morbidity and mortality indicators in patients of cirrhosis. *Cureus* 2022; 14 (1).
21. Monsanto P, Almeida N, Lrias C. Evaluation of MELD score and Maddrey discriminant function for mortality prediction in patients with alcoholic hepatitis. *Hepatogastroenterol* 2013; 60 (125): 1089-94.
22. Biyik M, Ucar R, Solak Y *et al*. Blood neutrophil-to-lymphocyte ratio independently predicts survival in patients with liver cirrhosis. *Eur J Gastroenterol Hepatol* 2013; 25 (4): 435-41.