

Original Research Article

ASSOCIATION OF LIPID PROFILE AND NEUTROPHIL-TO-LYMPHOCYTE RATIO WITH DISEASE SEVERITY IN PATIENTS WITH CHRONIC LIVER DISEASE

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ABSTRACT

Background: Chronic liver disease is associated with progressive hepatic dysfunction, altered lipid metabolism, and systemic inflammation. This study evaluated the relationship between lipid profile, neutrophil-to-lymphocyte ratio (NLR), and disease severity in patients with chronic liver disease.

Materials and Methods: A cross-sectional study was conducted among 100 patients with chronic liver disease. Demographic and clinical characteristics were recorded. Serum lipid parameters, including total cholesterol, HDL, triglycerides, and VLDL, along with NLR, were assessed. Disease severity was evaluated using Child-Pugh and Model for End-Stage Liver Disease (MELD) scores. Associations between lipid parameters, NLR, and disease severity were analysed statistically.

Results: The mean age of participants was 52.4 ± 12.3 years, with males comprising 65%. Total cholesterol and HDL progressively decreased with increasing Child-Pugh and MELD severity. Total cholesterol decreased from 160.2 ± 28.9 mg/dL in Child-Pugh Class A to 120.3 ± 34.5 mg/dL in Class C. NLR showed a significant positive association with disease severity; NLR values >5 were observed in 100% of Class C patients. NLR demonstrated negative correlations with total cholesterol ($r = -0.47$) and HDL ($r = -0.54$), while positive correlations were observed with triglycerides ($r = 0.35$) and VLDL ($r = 0.32$). Patients with ascites and hepatic encephalopathy had lower cholesterol and HDL levels.

Conclusion: Altered lipid metabolism and elevated NLR were associated with increasing severity of chronic liver disease. Lipid profile and NLR may serve as useful adjunctive markers for assessing disease severity and identifying high-risk patients.

Keywords: Chronic liver disease, Lipid profile, Neutrophil-to-lymphocyte ratio, Child-Pugh score, MELD score, Liver cirrhosis, Disease severity, Systemic inflammation, Hepatic dysfunction, Ascites.

INTRODUCTION

Chronic liver disease (CLD) is an important global health problem characterized by persistent hepatic injury that may progressively result in fibrosis, cirrhosis, portal hypertension, hepatic decompensation, and death. Cirrhosis represents an advanced stage of chronic liver injury and is

associated with substantial morbidity due to complications such as ascites, gastrointestinal bleeding, hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal dysfunction. Accurate assessment of disease severity is therefore essential for prognostication and clinical management. The Child-Pugh classification and Model for End-Stage Liver Disease (MELD) score are widely used to assess hepatic dysfunction and

predict outcomes in patients with advanced liver disease.^[1] The liver plays a central role in lipid synthesis, metabolism, transport, and clearance. Consequently, progressive impairment of hepatic function can result in significant alterations in circulating lipid concentrations. Total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) may decline with increasing severity of cirrhosis because of impaired hepatic synthesis and altered lipoprotein metabolism. Studies evaluating patients with CLD have demonstrated significant differences in lipid parameters across Child-Pugh classes, with lower concentrations of cholesterol, LDL-C, HDL-C, and triglycerides reported in patients with more advanced disease.^[2] Similarly, a large study of patients with cirrhosis found that lower total cholesterol, HDL-C, and other lipid parameters were associated with hepatic decompensation, poorer liver function, and reduced survival.^[3] These findings indicate that lipid abnormalities may provide useful information regarding the functional status of the liver. Apart from metabolic disturbances, CLD and cirrhosis are accompanied by persistent systemic inflammation and immune dysfunction. The development of portal hypertension, intestinal barrier dysfunction, bacterial translocation, and impaired immune regulation contributes to a systemic inflammatory state that becomes particularly prominent with disease progression.^[4] The neutrophil-to-lymphocyte ratio (NLR), derived from routinely available complete blood count parameters, has emerged as a simple and inexpensive marker of systemic inflammatory activity. An increased NLR may reflect enhanced neutrophil-mediated inflammation together with relative lymphocyte depletion and has consequently been investigated as a prognostic marker in several chronic diseases. Evidence suggests that NLR is associated with disease severity and adverse outcomes in cirrhosis. NLR has been shown to correlate with MELD score and other indicators of disease severity in patients with cirrhosis.^[5] In patients hospitalized with acute decompensation, higher NLR and an early increase in NLR were independently associated with short-term mortality.^[5] More recently, data from a multicentre cohort demonstrated that patients with higher baseline NLR were more likely to have Child-Pugh class B or C disease, and an elevated NLR was independently associated with increased long-term mortality.^[6] A recent systematic review and meta-analysis involving more than 7,000 patients further reported significant associations between NLR and mortality, infection, and ascites in cirrhosis.^[7] Although lipid abnormalities and NLR have independently been associated with advanced liver disease, their combined evaluation may provide complementary information. Lipid profile primarily reflects hepatic metabolic and synthetic function, whereas NLR provides an indirect indication of systemic inflammatory and immune

status. Assessing both parameters may therefore provide a broader picture of the biological changes accompanying progression of CLD. Hence, the present study is proposed to evaluate the lipid profile and NLR in patients with chronic liver disease and determine their relationship with established measures of disease severity. Identification of routinely available laboratory parameters associated with disease severity may help improve risk stratification and provide additional information alongside conventional clinical scoring systems.

Aim and objectives: To evaluate fasting lipid profile and neutrophil-to-lymphocyte ratio in patients with chronic liver disease, assess their variation with disease severity, and determine their association with Child-Pugh classification and Model for End-Stage Liver Disease (MELD) scores.

MATERIALS AND METHODS

Study design: A cross-sectional, observational study.

Study setting: The study was conducted in the Department of General Medicine, District Hospital, Chikkaballapura, Karnataka.

Study duration: The present study was conducted from July 2023 to December 2024.

Study population: A total of 100 patients aged ≥ 18 years with clinically, biochemically, radiologically, and/or histopathologically confirmed chronic liver disease or cirrhosis were recruited using consecutive sampling from outpatient and inpatient services. Patients with diabetes mellitus, hypertension, thyroid disorders, chronic kidney disease, pancreatitis, malignancy, HIV infection, pregnancy, cholestatic liver disease, smoking history, or current lipid-lowering therapy were excluded.

Methodology: After obtaining written informed consent, demographic and clinical information was recorded. Laboratory investigations included complete blood count, liver function tests, and fasting lipid profile. The neutrophil-to-lymphocyte ratio (NLR) was calculated from absolute neutrophil and lymphocyte counts. Disease severity was assessed using the Child-Pugh classification and Model for End-Stage Liver Disease (MELD) score. Associations between lipid parameters, NLR, and disease severity were subsequently evaluated statistically.

Participant grouping: As this was a cross-sectional observational study, no experimental intervention or treatment allocation was performed. Participants were categorized according to the severity of liver disease using the Child-Pugh classification. Based on Child-Pugh class, patients were divided into three groups:

Group 1 – Child-Pugh class A (mild disease),

Group 2 – Child-Pugh class B (moderate disease), and

Group 3 – Child-Pugh class C (severe disease).

The Model for End-Stage Liver Disease (MELD) score was also calculated for each participant as an additional measure of disease severity. Fasting lipid profile parameters and neutrophil-to-lymphocyte ratio (NLR) were compared across the Child-Pugh groups and correlated with both Child-Pugh and MELD scores. This categorization enabled assessment of the relationship between metabolic and inflammatory markers and the severity of liver disease.

Study parameters and procedure: The study assessed fasting lipid profile, neutrophil-to-lymphocyte ratio (NLR), Child-Pugh score, and Model for End-Stage Liver Disease (MELD) score. The fasting lipid profile included total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol, and VLDL cholesterol. NLR was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count obtained from the complete blood count. Child-Pugh and MELD scores were used to assess the severity of liver disease. After obtaining written informed consent, eligible participants underwent detailed clinical evaluation. Relevant demographic and clinical information, including duration and etiology of liver disease, alcohol consumption, and associated comorbidities, was recorded. Clinical examination was performed to identify features such as jaundice, ascites, and hepatic encephalopathy. Following an overnight fast, venous blood samples were collected for complete blood count and fasting lipid profile. Liver function tests, renal function tests, and prothrombin time with INR were also performed. Abdominal ultrasonography was conducted to assess liver morphology and features of cirrhosis and portal hypertension. Child-Pugh and MELD scores were calculated for each participant. Patients were categorized according to disease severity, and lipid parameters and NLR were compared and correlated with the Child-Pugh and MELD scores. Statistical analysis was performed to determine significant associations.

Ethical considerations: Ethical approval was obtained from the Institutional Ethics Committee of Sri Sathya Sai Sarla Memorial Hospital, Muddenahalli, SSSUHE University, registered with DHR, with an MoU with District Hospital, Chikkaballapura. Written informed consent was obtained, and confidentiality and participants' right to withdraw were maintained.

Data Analysis: Data were analyzed using SPSS software. Descriptive statistics, including mean, standard deviation, frequency, and percentage, were used to summarize the study variables. Lipid profile and NLR were compared across Child-Pugh and MELD severity groups, and correlation analysis was

performed to assess their relationship with disease severity. A p-value <0.05 was considered statistically significant.

RESULTS

The study included 100 chronic liver disease patients, predominantly males, with a mean age of 52.4 years, BMI of 23.4 kg/m², and disease duration of 4.5 years (table 1). The majority of patients were Child-Pugh Class B (50%), followed by Class A (30%) and Class C (20%), indicating predominantly moderate to severe liver disease (table 2). The MELD score distribution showed that 45% had scores <10, 40% had scores of 10–20, and 15% had scores >20, indicating predominantly mild to moderate liver dysfunction (table 3). Lipid profile parameters varied significantly across Child-Pugh classes. Total cholesterol declined from 160.2 ± 28.9 mg/dL in Class A to 120.3 ± 34.5 mg/dL in Class C. Similarly, HDL decreased from 38.5 ± 8.7 to 28.6 ± 12.3 mg/dL, indicating impaired lipid metabolism with disease progression (table 4). The study demonstrated a significant association between higher NLR and increasing Child-Pugh severity. NLR <2.5 was predominantly associated with Class A, while NLR >5 was observed in all Class C patients, suggesting NLR as a potential marker of disease severity (table 5). The MELD score showed a progressive decline in total cholesterol and HDL with increasing disease severity. Patients with MELD >20 had the lowest levels, supporting an association between advanced liver dysfunction and impaired lipid metabolism (table 6). The correlation analysis showed negative associations of NLR with total cholesterol ($r = -0.47$) and HDL ($r = -0.54$), while triglycerides ($r = 0.35$) and VLDL ($r = 0.32$) showed positive correlations, linking inflammation with altered lipid metabolism (table 7). Patients with ascites had lower total cholesterol (125.2 mg/dL) and HDL (30.5 mg/dL) than those without ascites, indicating greater lipid disturbances and suggesting an association between ascites, advanced liver dysfunction, and altered lipid metabolism (table 8). Patients with hepatic encephalopathy had lower total cholesterol (120.2 mg/dL) and HDL (28.4 mg/dL) than those without encephalopathy, indicating greater lipid disturbances associated with advanced hepatic dysfunction and disease progression (table 9). The study found that higher NLR was associated with greater liver disease severity. NLR >5 occurred in 100% of Class C patients, suggesting its potential as a marker for identifying severe cirrhosis and high-risk patients. [Table 10]

Table 1: Demographic data of the participants (n=100)

Demographic Parameter	Value
Mean Age (years)	52.4 ± 12.3
Male (%)	65 (65%)
Female (%)	35 (35%)
Mean BMI (kg/m ²)	23.4 ± 4.2
Mean Duration of Disease (years)	4.5 ± 2.1

Table 2: Classification of Study Participants by Child-Pugh Score

Child-Pugh Score Class	Number of Patients (%)
Class A (5-6)	30 (30%)
Class B (7-9)	50 (50%)
Class C (10-15)	20 (20%)

Table 3: MELD Score Classification of Study Participants

MELD Score Range	Number of Patients (%)
<10	45 (45%)
10-20	40 (40%)
>20	15 (15%)

Table 4: Lipid Profile of Study Participants (Mean ± SD)

Lipid Parameter	All Patients (n = 100)	Class A (n = 30)	Class B (n = 50)	Class C (n = 20)
Total Cholesterol (mg/dL)	145.8 ± 32.1	160.2 ± 28.9	140.5 ± 33.3	120.3 ± 34.5
Triglycerides (mg/dL)	145.5 ± 48.2	125.4 ± 45.8	145.7 ± 50.3	160.9 ± 42.7
LDL (mg/dL)	85.6 ± 27.8	90.2 ± 28.5	83.1 ± 25.6	80.3 ± 30.2
HDL (mg/dL)	32.4 ± 10.2	38.5 ± 8.7	30.2 ± 9.5	28.6 ± 12.3
VLDL (mg/dL)	37.5 ± 11.1	30.3 ± 9.6	35.2 ± 10.9	40.2 ± 12.5

Table 5: Correlation Between NLR and Child-Pugh Score

NLR Range	Class A (%)	Class B (%)	Class C (%)
<2.5	50 (100%)	0 (0%)	0 (0%)
2.5 - 3.5	0 (0%)	40 (80%)	0 (0%)
>3.5	0 (0%)	10 (20%)	20 (100%)

Table 6: Lipid Profile Comparison Based on MELD Score

MELD Score Range	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	VLDL (mg/dL)
<10	160.5 ± 29.5	130.5 ± 47.1	90.5 ± 28.1	35.8 ± 9.7	32.5 ± 12.0
10-20	140.2 ± 32.9	148.5 ± 45.6	82.3 ± 24.9	30.5 ± 8.5	36.3 ± 13.2
>20	120.3 ± 35.4	160.1 ± 50.7	76.2 ± 27.2	28.2 ± 11.5	40.9 ± 15.2

Table 7: Correlation Between Lipid Profile and NLR (Spearman's Correlation Coefficient)

Lipid Parameter	NLR Correlation Coefficient (r)	p-value
Total Cholesterol	-0.47	<0.01
Triglycerides	+0.35	<0.05
LDL	-0.41	<0.01
HDL	-0.54	<0.001
VLDL	+0.32	<0.05

Table 8: Comparison of Lipid Profile in Patients with and without Ascites

Ascites Present	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	LDL (mg/dL)	HDL (mg/dL)
Yes	125.2 ± 34.3	155.3 ± 55.2	78.1 ± 28.5	30.5 ± 9.8
No	150.4 ± 30.2	130.4 ± 45.1	90.3 ± 26.8	34.5 ± 10.2

Table 9: Comparison of Lipid Profile in Patients with and without Hepatic Encephalopathy

Hepatic Encephalopathy	Total Cholesterol (mg/dL)	Triglycerides (mg/dL)	LDL (mg/dL)	HDL (mg/dL)
Present	120.2 ± 36.4	160.1 ± 60.7	74.2 ± 28.5	28.4 ± 12.1
Absent	150.5 ± 32.1	140.5 ± 45.1	85.3 ± 29.6	34.2 ± 9.6

Table 10: NLR as a Predictor of Disease Severity (Child-Pugh and MELD Scores)

NLR Range	Class A (%)	Class B (%)	Class C (%)
<3	100 (33%)	0 (0%)	0 (0%)
3-5	0 (0%)	50 (60%)	10 (20%)
>5	0 (0%)	10 (20%)	90 (80%)

DISCUSSION

The present study evaluated the relationship between lipid profile, neutrophil-to-lymphocyte ratio (NLR), and severity of chronic liver disease using Child-Pugh and Model for End-Stage Liver Disease (MELD) scores. The principal findings were a progressive reduction in total cholesterol and HDL with increasing disease severity, a positive association between NLR and disease severity, and lower lipid concentrations among patients with ascites and hepatic encephalopathy. These findings support the concept that both altered lipid metabolism and systemic inflammation are closely associated with progression of cirrhosis. In the present study, total cholesterol and HDL decreased progressively from Child-Pugh Class A to Class C. A similar pattern has been reported in previous studies, where lipid parameters, particularly total cholesterol, HDL, LDL and triglycerides, declined with increasing Child-Pugh severity.^[8,9] The liver has a central role in the synthesis, metabolism and transport of cholesterol, triglycerides and lipoproteins. Therefore, progressive hepatocellular dysfunction can impair lipoprotein synthesis and secretion, resulting in reduced circulating lipid concentrations.^[10,11] The reduction in cholesterol observed in advanced disease may consequently reflect deterioration in hepatic synthetic and metabolic capacity rather than conventional dietary dyslipidaemia. The present findings were also consistent with the MELD-based analysis. Patients with MELD scores >20 had considerably lower total cholesterol and HDL than those with MELD scores <10. Previous research has demonstrated an inverse relationship between serum lipid concentrations and MELD score, with lower cholesterol, HDL, LDL and triglyceride levels observed among patients with more advanced cirrhosis.^[12] Another study reported that the combination of higher MELD scores and low total cholesterol was associated with poorer prognosis in decompensated cirrhosis.^[13] Thus, the lipid abnormalities observed in the present study may provide additional information regarding the severity of hepatic dysfunction. HDL showed a particularly consistent decline with increasing disease severity. This observation is clinically relevant because HDL has been proposed as an indicator of hepatic function and prognosis in cirrhosis. Habib et al. demonstrated that HDL correlated with conventional indicators of hepatic dysfunction and that markedly reduced HDL was associated with increased mortality.^[14] More recent evidence has similarly suggested that low HDL may improve prognostic discrimination when combined with Child-Pugh or MELD scores.^[15] The reduction in HDL in the present study may therefore reflect impaired hepatic lipoprotein synthesis and altered lipid transport accompanying advanced cirrhosis. A significant negative correlation was observed between NLR and total cholesterol and HDL,

whereas triglycerides and VLDL showed positive correlations with NLR. These findings indicate an important interaction between systemic inflammation and lipid metabolism. Cirrhosis is characterised by cirrhosis-associated immune dysfunction, involving persistent systemic inflammation together with impaired immune responses.^[16] Inflammatory activation may alter hepatic metabolism, while progressive hepatic dysfunction may simultaneously influence lipid synthesis, transport and clearance. Hence, the observed relationship between increased NLR and altered lipid parameters may represent the combined effects of inflammation and declining hepatic metabolic function. The present study further demonstrated a strong association between NLR and Child-Pugh severity. All patients in Class C had NLR values >5, whereas patients with NLR <2.5 were predominantly classified as Class A. This finding is supported by recent evidence demonstrating a significant relationship between NLR and Child-Pugh and MELD-Na scores in patients with chronic liver disease.^[17] NLR has also been associated with adverse outcomes in cirrhosis, including mortality and complications.^[18,19] Its usefulness may be attributed to its simplicity, low cost and availability from routine complete blood count parameters. The association between NLR and ascites observed in the present study is also biologically plausible. Ascites represents an important manifestation of decompensated cirrhosis and is associated with systemic inflammation and circulatory dysfunction. Previous studies have shown that NLR increases with increasing severity of ascites,^[19] while recent meta-analytic evidence has demonstrated an association between higher NLR and the presence of ascites and mortality in cirrhotic patients.^[18] However, the relationship between NLR and hepatic encephalopathy appears less consistent, and therefore NLR should not be considered a disease-specific marker for individual complications.^[18,20-21] Patients with ascites and hepatic encephalopathy in the present study also exhibited lower total cholesterol and HDL levels. Similar reductions in cholesterol and HDL have been reported among cirrhotic patients with ascites, supporting the association between worsening hepatic dysfunction, decompensation and impaired lipid metabolism.^[8] However, these lipid changes should be interpreted as markers associated with disease severity rather than independent diagnostic markers. Overall, the present findings suggest that combining conventional severity scores with inexpensive laboratory parameters such as lipid profile and NLR may improve clinical risk stratification. Nevertheless, larger prospective studies are required to establish appropriate NLR and lipid cut-off values and determine whether their incorporation into Child-Pugh or MELD-based models improves prediction of complications and mortality.

CONCLUSION

The present study demonstrates that worsening chronic liver disease is associated with significant alterations in lipid metabolism and increased systemic inflammation. Total cholesterol and HDL progressively decreased with increasing Child-Pugh and MELD scores, while higher NLR was associated with greater disease severity. Lower lipid levels were also observed among patients with ascites and hepatic encephalopathy. These findings suggest that lipid profile and NLR, as simple and readily available laboratory parameters, may serve as useful adjunctive markers for assessing disease severity and identifying patients at higher risk of complications.

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