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Original Article

Correlation of Lipid Parameters with Severity of Hepatic Fibrosis in North East India

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Abstract

Background: Chronic liver disease (CLD) is a major global health burden with diverse etiologies. Alterations in lipid metabolism are common in CLD, but their association with hepatic fibrosis severity remains unclear.

Objectives: This study aimed to evaluate the clinical, etiological, and biochemical profile of CLD patients and to determine the correlation between lipid parameters and the severity of hepatic fibrosis, assessed by the aspartate aminotransferase to platelet ratio index (APRI).

Materials and Methods: A cross-sectional observational study was conducted on 100 consecutive CLD patients admitted to the Department of Medicine, Agartala Government Medical College. Demographic details, etiological classification, hemoglobin levels, lipid profile (triglycerides [TG], low-density lipoprotein [LDL], and high-density lipoprotein [HDL]), and APRI scores were recorded. Statistical analysis included Pearson's and Spearman's correlation tests.

Results: The majority of patients were middle-aged (50–59 years), with a male predominance (81%). Alcoholic liver disease (ALD) (58%) was the leading etiology, followed by metabolic dysfunction-associated steatotic liver disease (28%), hepatitis B virus (11%), and hepatitis C virus (3%). Moderate-to-severe anemia was common (91.7%). The mean TG, LDL, and HDL were 106.7 ± 55.0 mg/dL, 52.1 ± 22.9 mg/dL, and 25.7 ± 10.3 mg/dL, respectively. The mean APRI score was 1.44 ± 1.11. TG showed no significant correlation with APRI ($P > 0.05$). In contrast, both LDL ($r = -0.43$, $P = 0.019$) and HDL ($r = -0.54$, $P = 0.003$) demonstrated significant negative correlations with APRI, with HDL showing a stronger association ($\rho = -0.76$, $P < 0.0001$).

Conclusion: ALD was the most common etiology in this study group of North East India. LDL and HDL levels were significantly and inversely correlated with fibrosis severity, whereas TG showed no significant association. HDL, in particular, may serve as a sensitive, non-invasive biomarker of hepatic fibrosis.

Key words: Chronic liver disease, Hepatic fibrosis, Lipid profile, Liver stiffness measurement, Non-invasive testing of hepatic fibrosis

INTRODUCTION

Chronic liver disease (CLD) represents a spectrum of progressive hepatic insults, ultimately leading to fibrosis, cirrhosis, and its complications.[1] Hepatic fibrosis is the common pathway from different CLDs to compensated advanced CLD (cACLD), decompensated cirrhosis, and end-stage liver disease. The term cACLD is relatively new and was introduced at Baveno VI consensus guidelines in 2015 to denominate patients with CLD at risk of developing clinically significant portal hypertension based on liver stiffness measurement (LSM), one of the most well-recognized non-invasive assessments for liver fibrosis. Early diagnosis of hepatic fibrosis is crucial to halt the worsening of liver fibrosis, given its important prognostic implications. Many non-invasive tests have been developed over the years, trying to assess liver fibrosis in a practical and accurate way. The tests are mainly laboratory- or imaging-based, or in combination. However, there are questions about the availability of tests such as magnetic resonance (MR) elastography or even FibroScan [Table 1]. Hence, laboratory-based tests can be derived from simply routine blood tests and can be of paramount importance in many underserved areas. Nonetheless, non-invasive tests do not

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always apply to all liver diseases, nor does a common cutoff value of a test mean the same degree of liver fibrosis in different scenarios. CLD forms a big healthcare burden, and diagnosing and assessing the severity of liver fibrosis to provide appropriate management is the need of the hour.

Simultaneously, the etiology of liver disease is different in different geographical areas. The North Eastern region of India has its own diversity, and there is frequent change in demography, food habits, migration patterns, and addiction.

Lipid metabolism is significantly impacted in liver disease, given the liver's central role in lipid synthesis, storage, and transport.[2] Alterations in lipid fractions have been documented in various hepatic pathologies, but their correlation with fibrosis severity remains debated.[3] Non-invasive fibrosis assessment tools, such as the aspartate aminotransferase (AST) to platelet ratio index (APRI) score, offer a practical method for clinical evaluation.[4]

This study aims to delineate the clinical and biochemical profile of CLD patients in the specific study population and to examine the association between lipid parameters and fibrosis severity.

Aims and Objectives

Aims

This study aimed to evaluate the clinical profile of patients with CLD and determine the correlation between lipid parameters and the severity of hepatic fibrosis.

Objectives

1. To assess the demographic, clinical characteristics, and etiology of patients with CLD, including age, sex distribution, and etiological spectrum
2. To evaluate the hematological profile, particularly hemoglobin levels, in the study population
3. To determine the lipid profile parameters (triglycerides [TG], low-density lipoprotein [LDL], and high-density lipoprotein [HDL]) in patients with CLD
4. To measure the severity of hepatic fibrosis using the APRI score.
5. To analyze the correlation between each lipid parameter (TG, LDL, and HDL) and the APRI score to assess their potential as non-invasive markers of fibrosis severity.

MATERIALS AND METHODS

Study Design

The study design was cross-sectional.

Study Type

The study type was observational.

Study Setting

General Medicine Department of Agartala Government Medical College, Tripura.

Study Population

Indoor patients of CLD in the Medicine Department of Agartala Government Medical College.

Study Duration

One year (January 2025–December 2025)

Sample Size

A total of 100 consecutive patients of CLD admitted in medicine indoor.

Inclusion Criteria

Adults (>18 years) with confirmed CLD by clinical, biochemical, and radiological means.

Exclusion Criteria

Acute liver failure, concurrent severe systemic illness, and recent lipid-lowering therapy.

Data Collection

Demographic details, etiology, hemoglobin levels, lipid profile (TG, LDL, HDL), and APRI scores were recorded. Etiology was classified into alcoholic liver disease (ALD), metabolic dysfunction-associated steatotic liver disease (MASLD), hepatitis B virus (HBV), hepatitis C virus (HCV), autoimmune hepatitis, and others [Table 2].

Anemia severity was categorized per World Health Organization criteria. APRI score was calculated using the standard formula:

$$\text{APRI} = (\text{AST}/\text{ULN AST}) \times 100/\text{platelet count } (10^9/\text{L}).$$

Statistical Analysis

Data entry and analysis were performed using the Statistical Package for the Social Sciences version 21.0 in Windows PC. Data were expressed as mean ± standard deviation or median where appropriate. Pearson's and Spearman's correlation coefficients were computed to evaluate the association between lipid parameters and APRI. $P < 0.05$ was considered statistically significant.

RESULTS

In this cohort of 100 patients, the majority were in the middle-aged group. The most represented age category was 50–59 years (36%), followed closely by 40–49 years (33%). Patients aged 60–69 years constituted 17% of the sample, and those aged 30–39 years accounted for 11%. Only 3%

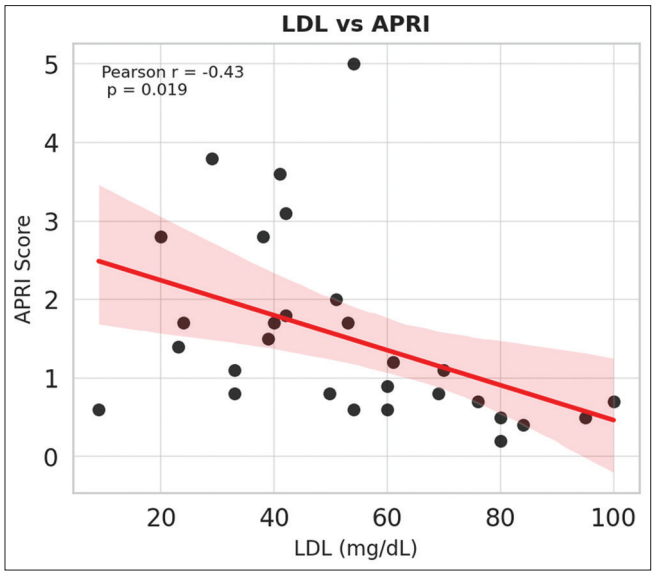

Figure 1: Scatter plot showing the correlation between low-density lipoprotein and aspartate aminotransferase to platelet ratio index score

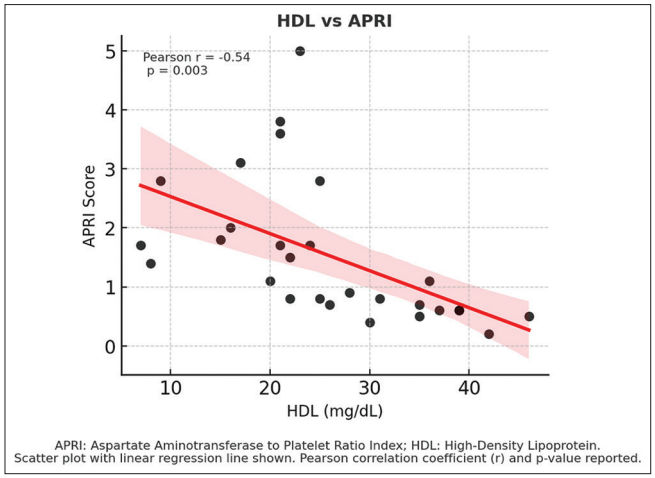

Figure 2: Scatter plot showing the correlation between high-density lipoprotein and aspartate aminotransferase to platelet ratio index score

of cases were aged 70 years or above. No patients were younger than 30 years.

The study population was predominantly male, with 81% males and 19% females. This male predominance was consistent across the major etiological subgroups [Table 3].

ALD was the most common underlying etiology, accounting for 58% of cases. MASLD was the second most common, observed in 28% of patients. Viral etiologies were less frequent, with HBV infection in 11% and HCV in 3% of patients.

The mean hemoglobin concentration in the study population was 7.87 ± 2.02 g/dL, with a median value of

Table 1: Etiological distribution of study population (*n*=100)

Etiology	Percentage	n (patients)
ALD	58.3	58
NASH/MASLD	27.8	28
HBV	11.1	11
HCV	2.8	3

ALD: Alcoholic liver disease, HBV: Hepatitis B virus, HCV: Hepatitis C virus, NASH: Non alcoholic steatohepatitis, MASLD: Metabolic dysfunction-associated steatotic liver disease

Table 2: Hemoglobin parameters in study population (*n*=100)

Hemoglobin category	Percentage	n (patients)
7–9.9 (Moderate anemia)	63.9	64
<7 (Severe anemia)	27.8	28
10–12.9 (Mild anemia)	5.6	6
≥13 (Normal)	2.8	3

Table 3: Lipid profile of study population (*n*=100)

Parameter	Mean±SD (mg/dL)	Median (mg/dL)
TG	106.7±55.0	92.5
LDL	52.1±22.9	51.0
HDL	25.7±10.3	24.5

TG: Triglycerides, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, SD: Standard deviation

7.95 g/dL. A large proportion of patients had moderate anemia (7–9.9 g/dL), comprising 64% of the sample. Severe anemia (<7 g/dL) was present in 28% of patients. Only 6% had mild anemia (10–12.9 g/dL), and 3% had hemoglobin in the normal range (≥13 g/dL).

The mean TG level in the study population was 106.7 ± 55.0 mg/dL (median 92.5 mg/dL). The mean LDL level was 52.1 ± 22.9 mg/dL (median 51.0 mg/dL). The mean HDL level was 25.7 ± 10.3 mg/dL (median 24.5 mg/dL).

The mean APRI score was 1.44 ± 1.11, with a median of 1.10. Intermediate APRI scores (0.5–1.5) were the most common (57%), followed by high APRI scores (>1.5) in 37%, and low APRI scores (<0.5) in 6%.

Correlation analysis showed that TG levels had no significant association with APRI (Pearson's $r = -0.15$, $P = 0.43$; Spearman's $\rho = -0.20$, $P = 0.30$). In contrast, LDL levels demonstrated a significant negative correlation with APRI (Pearson's $r = -0.43$, $P = 0.019$; Spearman's $\rho = -0.57$, $P = 0.001$). HDL levels demonstrated a statistically significant negative correlation with APRI score. The Pearson correlation coefficient was $r = -0.54$ ($P = 0.003$), and Spearman's rank correlation coefficient was $\rho = -0.76$ ($P < 0.0001$). These findings indicate that lower HDL levels were associated with higher APRI scores, with

the strength of this relationship ranging from moderate (Pearson) to strong (Spearman).

DISCUSSION

The present study provides insights into the clinical and biochemical profile of CLD patients and explores the relationship between lipid parameters and fibrosis severity, as measured by the APRI score. The findings reinforce known epidemiological patterns while contributing novel observations on lipid alterations in advanced liver disease.

In our cohort, the majority of patients were middle-aged (50–59 years, 36%), with a male predominance (81% males and 19% females), reflecting the established epidemiology of CLD. ALD (58% of cases) was identified as the leading etiology, consistent with national and regional data from India, where alcohol misuse is a significant contributor to liver disease.[5] MASLD and viral hepatitis (HBV and HCV) were less common but still clinically important causes in our study population. These distributions align with previous literature, where alcohol, metabolic syndrome, and viral infections constitute the major drivers of CLD globally.[6]

A high prevalence of anemia was observed, with the majority of patients having moderate-to-severe anemia with a mean hemoglobin concentration of 7.87 ± 2.02 g/dL. This finding may be attributed to nutritional deficiencies, hypersplenism, chronic inflammation, and direct bone marrow suppression seen in advanced liver disease. The presence of significant anemia has prognostic implications and underscores the need for integrated management strategies in CLD.[7]

The study observed reduced mean levels of LDL and HDL, with TG being relatively preserved. Importantly, both LDL and HDL showed a statistically significant negative correlation with APRI score, suggesting that lower levels of these lipoproteins are associated with more severe hepatic fibrosis. Notably, HDL demonstrated a strong inverse correlation with APRI (Pearson $r = -0.54$, Spearman $\rho = -0.76$), indicating its potential as a sensitive biomarker of fibrosis severity [Figures 1 and 2]. These results can be explained pathophysiologically by the liver's central role in lipoprotein metabolism. Fibrosis and cirrhosis compromise hepatic synthetic capacity, leading to reduced production of apolipoproteins and lipoproteins. HDL, in particular, is significantly affected due to impaired synthesis of ApoA-I and alterations in reverse cholesterol transport, which may explain the stronger correlation observed.[8]

The lack of significant association between TG and APRI in this study parallels several previous works, suggesting that TG levels are less reliable indicators of fibrosis severity, possibly due to their dependence on multiple metabolic and dietary factors.

Our findings align with studies by Tauseef *et al*.,[3] which reported reduced LDL and HDL levels in advanced liver disease, with inverse associations with disease severity. Similarly, Chin *et al*.[4] emphasized the potential of lipid parameters as adjuncts to non-invasive fibrosis assessment tools. However, variations exist in the strength of these associations across populations, likely influenced by differences in etiology, nutrition, and comorbidities.

Clinical Implications

The significant correlation of LDL and HDL with APRI underscores their potential role as simple, cost-effective, and non-invasive markers for assessing fibrosis severity in CLD patients, especially in resource-limited settings where advanced imaging, FibroScan, MR elastography, or liver biopsy may not be feasible. While they cannot replace established methods, lipid parameters could complement existing non-invasive scores, enhancing clinical decision-making.

Ali Khan and Desale also showed that declining lipid profile parameters, particularly total cholesterol, HDL, and LDL, are significantly associated with increased severity of CLD. These changes reflect impaired hepatic synthetic function and may serve as simple, non-invasive markers to aid in staging and monitoring disease progression in clinical practice.[9]

Limitations

This study is limited by its cross-sectional design and single-center setting, which may restrict the generalizability of findings. The absence of longitudinal follow-up prevents assessment of causal relationships. In addition, potential confounders such as diet, comorbid metabolic conditions, and use of hepatotoxic drugs were not extensively evaluated.

Future Directions

Future multicentric studies with larger cohorts and longitudinal designs are required to validate these findings. Incorporating other non-invasive fibrosis indices, such as LSM, alongside lipid parameters may help develop composite models with greater diagnostic accuracy. Investigating molecular mechanisms linking HDL metabolism and fibrosis progression could also open new avenues for therapeutic interventions.

CONCLUSION

ALD was the most common cause of CLD in this region. However, the way metabolic disorders are increasing, in

the future MASLD may outnumber Alcohol associated liver disease. LDL and HDL levels showed a significant inverse correlation with fibrosis severity, while TG did not show any association despite its role in the pathobiology of ALD. HDL, in particular, may serve as a useful non-invasive marker of hepatic fibrosis.

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