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Original Research Article

EVALUATING FASTING LIPID PARAMETERS AS NON-INVASIVE BIOMARKERS OF HEPATIC DYSFUNCTION AND DISEASE SEVERITY IN LIVER CIRRHOSIS: A CROSS-SECTIONAL STUDY

Tejeshwini C[1], T. Tejaswini[2], Kiran B Budihal[3], Nagraraj H M[4], Dakshayini H S[5]

[1]Senior Resident, Department of General Medicine, Koppal Institute of Medical Sciences, Koppal, Karnataka, India.
[2]Senior Resident, Department of General Medicine, Sri Venkateswara Institute of Medical Sciences, Tirupati, Andhra Pradesh, India.
[3]Consultant Radiologist, Department of Radio Diagnosis, K.S Hospital, Koppal, Karnataka, India.
[4]Physician, Community Health Centre Karatagi, Koppal, Karnataka, India.
[5]Assistant Professor, Department of Radio Diagnosis, Alva's Medical College, Moodbidiri, Karnataka, India.

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Corresponding Author:
Dr. Kiran B Budihal,
Consultant Radiologist, Department of Radio Diagnosis, K.S.Hospital, Koppal, Karnataka, India.

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ABSTRACT

Background: The liver is the primary metabolic organ responsible for lipid synthesis, apolipoprotein assembly, and lipoprotein clearance. Progressive parenchymal loss and vascular architectural distortion in liver cirrhosis impair these metabolic pathways. This study evaluated fasting serum lipid profile alterations in patients with liver cirrhosis and assessed their correlation and diagnostic utility relative to established disease severity scoring systems (Child-Pugh classification and MELD score).

Materials and Methods: An analytical cross-sectional study was conducted among 103 adult patients with liver cirrhosis admitted to the Department of General Medicine, Koppal Institute of Medical Sciences (KIMS) Teaching Hospital, Koppal, Karnataka, India, between July 2023 and January 2024. Fasting serum lipid parameters—Total Cholesterol (TC), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), Very Low-Density Lipoprotein Cholesterol (VLDL-C), and Total Cholesterol/HDL-C ratio—were measured. Clinical severity was categorized using Child-Pugh classes (A, B, and C) and Model for End-Stage Liver Disease (MELD) scores. Comparative statistics, Pearson/Spearman correlation coefficients, multiple linear regression, and analyses were performed.

Results: The cohort was predominantly male (80.6%, n = 83) with a mean age of 44.2±11.8 years. Etiology was overwhelmingly alcoholic (93.2%, n = 96). According to Child-Pugh staging, 16.5% (n = 17) were Class A, 35.0% (n = 36) Class B, and 48.5% (n = 50) Class C; the overall mean MELD score was 16.8 2± 8.42. Progressive, statistically significant decreases were observed across Child-Pugh Classes A, B, and C for TC (164.3 2±18.2 vs. 151.7 2±21.5$ vs. 138.4 2± 19.8 mg/dL, $p < 0.001$), HDL-C (52.6 2±14.3 vs. 45. 2± 16.8 vs. 39.4 2±17.5$ mg/dL, $p < 0.001$), and LDL-C (86.4 2±22.5$ vs. 71.32± 26.2 vs. 58.22±27.9 mg/dL, $p < 0.001$). Strong negative correlations were documented between Child-Pugh score and TC ($r = -0.54$, $p < 0.001$), LDL-C ($r = -0.52$, $p < 0.001$), and HDL-C ($r = -0.47$, $p < 0.001$). Similarly, MELD score inversely correlated with TC ($r = -0.48$, $p < 0.001$), LDL-C ($r = -0.45$, $p < 0.001$), and HDL-C ($r = -0.42$, $p < 0.001$). Multiple linear regression identified HDL-C ($\beta = -0.142$, $p = 0.010$), TC ($\beta = -0.083$, $p = 0.008$), and LDL-C ($\beta = -0.067$, $p = 0.018$) as independent negative predictors of MELD score.

Conclusion: Serum lipid fractions—particularly TC, LDL-C, and HDL-C—decline in direct proportion to advanced hepatic functional impairment. These routinely measured biochemical markers serve as objective, low-cost non-

invasive indicators of disease severity in cirrhotic patients and offer dynamic prognostic information complementary to traditional scoring systems.
Keywords: Liver Cirrhosis, Lipid Profile, Child-Pugh Score, MELD Score, High-Density Lipoprotein, Total Cholesterol, Biomarkers.

INTRODUCTION

Liver cirrhosis represents the final common pathway for a wide variety of chronic liver diseases and constitutes a major cause of morbidity and mortality worldwide. The global prevalence of cirrhosis is estimated to be approximately 4.5% to 9.5% of the general population, with significant geographical variations reflecting differences in the prevalence of underlying etiological factors.[1] This progressive condition is characterized by the replacement of normal liver architecture with fibrotic tissue and regenerative nodules, leading to impaired hepatic function and potentially life threatening complications.[2] The liver plays a central role in lipid metabolism, including the synthesis, storage, transport, and clearance of various lipoproteins. It is responsible for the production of cholesterol, triglycerides, and phospholipids, which are essential components of cell membranes and serve as precursors for bile acids, steroid hormones, and vitamin D.[3]

Multiple studies have documented significant alterations in lipid metabolism in patients with liver cirrhosis. These changes are predominantly characterized by decreased serum levels of total cholesterol, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C).[4] The pathophysiological mechanisms underlying these alterations are multifaceted and include reduced hepatic biosynthetic capacity, impaired lipoprotein assembly and secretion, altered apolipoprotein synthesis, and enhanced LDL receptor-mediated clearance of lipoproteins from circulation.[5]

The severity of liver cirrhosis is traditionally assessed using clinical scoring systems such as the Child-Pugh classification and the Model for End-Stage Liver Disease (MELD) score. The Child Pugh classification incorporates parameters such as serum bilirubin, serum albumin, prothrombin time, the presence and severity of ascites, and hepatic encephalopathy to stratify patients into three classes (A, B, and C) of increasing severity.[2] The MELD score, initially developed to predict mortality in patients undergoing transjugular intrahepatic portosystemic shunt (TIPS) procedures, uses serum bilirubin, serum creatinine, and international normalized ratio (INR) to calculate a score that predicts three-month mortality in patients with end-stage liver disease.[6] While these scoring systems have proven valuable in clinical practice, they have certain limitations, including subjective components (in the case of Child-Pugh) and variability in laboratory measurements. Consequently, there is ongoing interest in identifying additional objective biomarkers that could enhance the accuracy of prognostication in cirrhotic patients.

Recent evidence suggests that the degree of lipid profile alterations may correlate with the severity of liver dysfunction in cirrhosis. Chrostek et al. observed a progressive decline in serum total cholesterol, LDL-C, and HDL-C levels with advancing Child-Pugh class in patients with alcoholic cirrhosis.[7] Similarly, Bassani et al. reported significant negative correlations between MELD scores and serum lipid parameters in a cohort of patients with cirrhosis of various etiologies.[8] These findings raise the intriguing possibility that serum lipid parameters could serve as additional prognostic markers in cirrhotic patients, potentially complementing established scoring systems. The relationship between lipid profile alterations and complications of cirrhosis is another area of growing interest. Hepatic encephalopathy, a neuropsychiatric syndrome that occurs in patients with advanced liver disease, has been associated with more pronounced reductions in serum cholesterol levels compared to cirrhotic patients without encephalopathy.[4] Similarly, patients with cirrhosis who develop hepatorenal syndrome or spontaneous bacterial peritonitis have been reported to exhibit more severe derangements in lipid metabolism than those without these complications.[9] These observations suggest that lipid parameters might not only reflect the severity of hepatic dysfunction but could also serve as predictors of specific complications in cirrhotic patients.

Interestingly, the etiology of cirrhosis may influence the pattern and magnitude of lipid profile alterations. For instance, patients with nonalcoholic steatohepatitis (NASH)-related cirrhosis may exhibit a different lipid profile compared to those with alcoholic or viral hepatitis-related cirrhosis, reflecting the distinct pathophysiological mechanisms involved in these conditions.[10]

Our study aims to address these knowledge gaps by comprehensively evaluating the relationship between serum lipid profile parameters and the severity of liver cirrhosis as assessed by clinical, biochemical, and imaging criteria. We hypothesize that specific lipid parameters, or combinations thereof, will demonstrate strong correlations with established markers of disease severity and could potentially enhance the accuracy of prognostication in cirrhotic patients. Additionally, we aim to explore whether the etiology of cirrhosis influences the pattern and magnitude of lipid profile alterations, which could have implications for patient stratification and management.

MATERIALS AND METHODS

Study Design and Setting: This analytical cross-sectional study was conducted at Koppal Institute of Medical Sciences (KIMS) Teaching Hospital, Koppal, Karnataka, India—a tertiary care referral centre serving the northern regional population of Karnataka. The study was conducted over an 18-month period from July 2023 to January 2024 following approval by the Institutional Ethics Committee Written informed consent was obtained from all participants or their legal guardians prior to enrolment.

Inclusion Criteria

Adult patients (18 years) admitted to the medical wards with a confirmed diagnosis of liver cirrhosis based on combined clinical presentation, biochemical evidence, endoscopic features, and radiological findings (abdominal ultrasonography/CT showing nodular liver surface, coarsened echotexture, portal vein dilation, and splenomegaly).

Exclusion Criteria

Patients with pre-existing primary dyslipidemia, underlying diabetes mellitus, systemic hypertension, cerebrovascular accidents, primary thyroid disorders, chronic kidney disease, acute or chronic pancreatitis, concurrent pregnancy, or those receiving lipid-lowering medications (statins, fibrates) or nephrotoxic/hepatotoxic medications influencing lipid metabolism

Data Collection and Laboratory Measurements

Demographic information, clinical history, and clinical signs (jaundice, ascites, splenomegaly, peripheral edema) were systematically documented. Blood samples were drawn following a 12-hour overnight fast and processed within 2 hours using an automated chemistry analyser (Beckman Coulter AU680, USA). Biochemical Workup: Complete blood count, liver function tests (total and direct bilirubin, AST, ALT, ALP, total protein, serum albumin), renal function tests (blood urea, serum creatinine), serum electrolytes, coagulation profile (prothrombin time, INR), and viral markers (HBsAg, anti-HCV antibody).

Lipid Profile Assessment:

- Total Cholesterol (TC) measured via the cholesterol oxidase-peroxidase (CHOD-POD) enzymatic method.
- Triglycerides (TG) measured via the glycerol phosphate oxidase-peroxidase (GPO-POD) method.
- High-Density Lipoprotein Cholesterol (HDL-C) quantified directly using polyethylene glycol-modified enzymatic assays.
- Low-Density Lipoprotein Cholesterol (LDL-C) calculated using the Friedewald equation: LDL-C = Total cholesterol - [HDL-C + (TGL/5)]
- Very low-density lipoprotein cholesterol (VLDL-C) was calculated as: VLDL-C = TGL/5

Assessment of Severity Scores

Child-Pugh Score: Computed by assigning 1 to 3 points across five variables: total bilirubin, serum albumin, prothrombin time/INR, ascites severity, and encephalopathy grade. Patients were classified into Class A (5–6 points; mild), Class B (7–9 points; moderate), or Class C (10–15 points; severe).

MELD Score: Calculated using the baseline formula: MELD = 3.78 × ln [serum bilirubin (mg/dL)] + 11.2 × ln[INR] + 9.57 × ln [serum creatinine (mg/dL)] + 6.43

MELD-Na score: This was calculated using the formula: MELD-Na = MELD + 1.32 × (137 - Na) - [0.033 × MELD × (137 - Na)] Where Na is the serum sodium concentration (mmol/L) with a lower limit of 125 mmol/L and an upper limit of 137 mmol/L.

Radiological Assessment

All patients underwent abdominal ultrasonography to confirm the diagnosis of cirrhosis and to assess for complications such as ascites, splenomegaly, and portal hypertension. The ultrasonographic criteria for the diagnosis of cirrhosis included nodular liver surface, coarsened echotexture, attenuated hepatic veins, and the presence of collaterals. Portal vein diameter, splenic size, and the presence of collaterals were also recorded.

RESULTS

Baseline Characteristics

The cohort consisted of 103 patients with a mean age of 44.2 ± 11.8 years (range: 18 to 60 years; peak incidence in the 31–40 age bracket: 38.8%, n = 40). Males accounted for 80.6% (n = 83) and females 19.3% (n = 20). Etiology was predominantly alcohol-related (93.2%, n = 96), followed by Hepatitis B (2.9%, n = 3), and cryptogenic/other factors (3.9%, n = 4). Abdominal distension (87.4%) and jaundice (85.4%) were the most frequent presenting features. On clinical examination, icterus was present in 89.3%, pallor in 83.5%, ascites in 86.4%, splenomegaly in 79.6%, and peripheral edema in 73.8%. Laboratory parameters revealed significant abnormalities across multiple systems. [Table 1]

Table 1: Baseline Laboratory and Biochemical parameters of study subjects

Parameter	Mean ± SD
Hemoglobin (g/dL)	8.5 ± 2.1
Total leukocyte count (×10³/µL)	10.8 ± 6.1
Platelet count (×10³/µL)	146.7 ± 84.3
Urea (mg/dL)	35.8 ± 23.5

Serum creatinine (mg/dL)	1.2 ± 1.3
Sodium (mEq/L)	132.8 ± 6.4
Potassium (mEq/L)	3.8 ± 0.8
Total bilirubin (mg/dL)	8.4 ± 7.3
Direct bilirubin (mg/dL)	4.6 ± 4.2
Total protein (g/dL)	6.2 ± 1.1
Serum albumin (g/dL)	3.1 ± 0.6
AST (U/L)	114.3 ± 88.6
ALT (U/L)	123.5 ± 103.2
ALP (U/L)	168.4 ± 76.3
PT (seconds)	22.8 ± 8.3
INR	1.8 ± 0.8

Lipid Parameters Across Disease Severity

Mean baseline lipid values for the entire cohort were: Total Cholesterol 147.2 ± 22.6 mg/dL, Triglycerides 86.4 ± 42.3 mg/dL, HDL-C 43.8 ± 17.2 mg/dL, LDL-C 67.5 ± 28.4 mg/dL, VLDL-C 17.3 ± 8.5 mg/dL, and TC/HDL Ratio 3.8 ± 1.5.

Child-Pugh stratification assigned 16.5% (n = 17) to Class A, 35.0% (n = 36) to Class B, and 48.5% (n = 50) to Class C. The mean Child-Pugh score was 9.1 ± 1.6, and the mean MELD score was 16.8 ± 8.4. Stratification across Child-Pugh classes demonstrated a progressive decrease in all lipid fractions with advancing disease severity. Total cholesterol, HDL-C, LDL-C, and TC/HDL ratios showed highly significant differences ($p < 0.001$). [Table 2]

Table 2: Comparison of Lipid Parameters across Child-Pugh Classes

Parameter	Class A (n=17)	Class B (n=36)	Class C (n=50)	p-value
Total cholesterol (mg/dL)	164.3 ± 18.2	151.7 ± 21.5	138.4 ± 19.8	<0.001
Triglycerides (mg/dL)	98.2 ± 36.4	89.6 ± 43.1	79.7 ± 42.8	0.018
HDL-C (mg/dL)	52.6 ± 14.3	45.1 ± 16.8	39.4 ± 17.5	<0.001
LDL-C (mg/dL)	86.4 ± 22.5	71.3 ± 26.2	58.2 ± 27.9	<0.001
VLDL-C (mg/dL)	19.6 ± 7.3	17.9 ± 8.6	15.9 ± 8.6	0.031
Cholesterol/HDL ratio	3.2 ± 0.8	3.6 ± 1.3	4.2 ± 1.7	<0.001

Table 3: Correlation with Child-Pugh and MELD Scores

Parameter	Child-Pugh Score (r, p-value)	MELD Score (r, p-value)
Total cholesterol	-0.54, <0.001	-0.48, <0.001
Triglycerides	-0.31, 0.002	-0.28, 0.005
HDL-C	-0.47, <0.001	-0.42, <0.001
LDL-C	-0.52, <0.001	-0.45, <0.001
VLDL-C	-0.29, 0.003	-0.25, 0.012
Cholesterol/HDL ratio	0.41, <0.001	0.36, <0.001

Bivariate analysis revealed significant inverse correlations between serum lipid parameters and clinical severity metrics. Total cholesterol, LDL-C, and HDL-C demonstrated strong negative correlations with both Child-Pugh and MELD scores. Conversely, the TC/HDL ratio showed a positive correlation with increasing score values. [Table 3]

Regression and Discriminatory Performance

Multiple linear regression analysis evaluated independent predictors of MELD score. HDL-C, Total Cholesterol, LDL-C, and duration of alcohol consumption emerged as statistically significant independent predictors. [Table 4]

Table 4: Multiple Linear Regression Analysis for Predictors of MELD Score

Variable	β coefficient	Standard Error	p-value	95% CI
Total cholesterol	-0.083	0.031	0.008	-0.144 to -0.022
HDL-C	-0.142	0.054	0.010	-0.249 to -0.035
LDL-C	-0.067	0.028	0.018	-0.122 to -0.012
Triglycerides	-0.024	0.016	0.134	-0.056 to 0.008
Age	0.098	0.062	0.115	-0.024 to 0.220
Sex (Female)	-1.824	1.563	0.246	-4.925 to 1.277
Duration of alcohol consumption	0.211	0.087	0.017	0.038 to 0.384

DISCUSSION

This study demonstrates that advanced liver cirrhosis is characterized by marked hypolipidemia affecting all major lipoprotein fractions, with the magnitude of decline directly correlating with clinical and biochemical indices of disease severity. The biological mechanisms driving hypocholesterolemia and decreased lipoprotein concentrations in liver disease are multifactorial.

Synthetic failure: The liver is a major site of endogenous cholesterol synthesis, and progressive parenchymal destruction and fibrotic replacement

impair hepatic lipid synthesis and lipoprotein metabolism. [11-15]

Apolipoprotein deficiencies: ApoA-I, the main structural apolipoprotein of HDL, and ApoB-100, which is essential for VLDL assembly and secretion, are synthesized predominantly by hepatocytes. Loss of hepatic synthetic reserve reduces apolipoprotein availability and may impair the assembly and secretion of circulating lipoproteins.[15,16]

Enzymatic dysfunction: Lecithin-cholesterol acyltransferase (LCAT), which is synthesized by the liver and plays a major role in the esterification of free cholesterol and HDL maturation, is altered in chronic liver disease, thereby potentially impairing reverse cholesterol transport.[17]

Altered lipoprotein metabolism and splanchnic factors: Changes in hepatic lipoprotein receptor metabolism, together with portosystemic shunting and nutritional or intestinal disturbances associated with advanced portal hypertension, may further contribute to reduced circulating lipid concentrations.[15,18]

Our findings align with international literature. Ghadir et al. demonstrated a significant inverse relationship between serum lipid concentrations and the severity of liver damage in cirrhotic patients, with progressive reductions in TC, LDL-C, and HDL-C with worsening hepatic dysfunction. [11] Similarly, Chrostek et al. observed marked decreases in TC, LDL-C, and HDL-C in patients with alcoholic cirrhosis compared with controls, with greater lipid abnormalities corresponding to more advanced Child-Pugh classes.[12] Bassani et al. confirmed inverse associations between TC, HDL-C, and LDL-C and both Child-Pugh and MELD scores, supporting the potential role of lipid parameters as indicators of cirrhosis severity.[13]

Our multiple linear regression analysis demonstrated that HDL-C ($\beta = -0.142$) was the strongest independent lipid predictor of MELD score, consistent with Habib et al., who identified HDL-C <30 mg/dL as an independent predictor of mortality in patients with non-cholestatic cirrhosis.[14] Beyond acting as a lipid carrier, HDL exerts anti-inflammatory, antioxidant, and endotoxin-neutralizing effects and participates in the modulation of lipopolysaccharide (LPS)-induced inflammatory responses.[14,18,19] Lower HDL-C levels may therefore reflect impaired hepatic synthetic function and may be associated with impaired host defense and dysregulated systemic inflammatory responses, potentially contributing to bacterial translocation, spontaneous bacterial peritonitis, and acute-on-chronic liver failure.[18,19]

Limitations

Design Constraints: The cross-sectional design precludes evaluation of long-term mortality, dynamic temporal changes in lipid parameters, or survival rates across extended follow-up periods.

Etiological Homogeneity: Over 93% of the study cohort had alcohol-induced cirrhosis, limiting subgroup comparisons with non-alcoholic fatty liver disease (NAFLD) or chronic viral hepatitis.

Unmeasured Parameters: Specific apolipoprotein levels (ApoA-I, ApoB), LCAT activity, functional HDL assays, and precise nutritional assessment markers were not measured.

CONCLUSION

Serum lipid parameters—specifically Total Cholesterol, LDL-C, and HDL-C—decline in direct proportion to the clinical severity of liver cirrhosis. These lipid fractions demonstrate strong inverse correlations with both Child-Pugh and MELD scores, with Total Cholesterol 145 mg/dL and LDL-C 65 mg/dL offering solid diagnostic performance for identifying severe cirrhosis. Given their widespread availability, low cost, and reproducibility, fasting lipid profiles represent valuable non-invasive biomarkers of hepatic synthetic impairment and disease severity in patients with liver cirrhosis.

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