Original Article

Diagnostic Performance of Serum Lipid Parameters for Identifying Severe Liver Cirrhosis: A Receiver Operating Characteristic Analysis

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Abstract

Background: Liver cirrhosis is characterized by progressive hepatic tissue disruption leading to portal hypertension and impaired metabolic capacity. Because the liver synthesizes, remodels, and clears serum lipids, cirrhosis alters circulating lipid homeostasis. This study aimed to evaluate fasting serum lipid profile abnormalities in cirrhotic patients and determine their diagnostic and prognostic performance in identifying severe liver disease. **Material and Methods:** This analytical cross-sectional study included 103 adult patients with confirmed liver cirrhosis admitted to the Department of General Medicine, Koppal Institute of Medical Sciences Teaching Hospital, Koppal, between July 2023 and January 2024. Fasting lipid profiles—total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein cholesterol (VLDL-C), and triglycerides (TG)—were measured and correlated with Child-Pugh classification (Classes A, B, and C) and Model for End-Stage Liver Disease (MELD) scores. Receiver Operating Characteristic (ROC) curve analyses were performed. **Results:** The study population was predominantly male (80.6%) with a mean age of 44.2 ± 11.8 years. Alcoholic etiology predominated (93.2%), and 48.5% of patients were classified as Child-Pugh Class C. Lipid parameters showed a significant progressive decrease across Child-Pugh Classes A, B, and C: TC (164.3, 151.7, 138.4 mg/dL; $p < 0.001$), HDL-C (52.6, 45.1, 39.4 mg/dL; $p < 0.001$), and LDL-C (86.4, 71.3, 58.2 mg/dL; $p < 0.001$). Strong negative correlations were observed between lipid parameters and both Child-Pugh and MELD scores. ROC curve analysis demonstrated robust discriminatory performance for detecting severe cirrhosis (Child-Pugh Class C or MELD ≥ 18), yielding Area Under the Curve (AUROC) values of 0.76 for TC, 0.75 for LDL-C, and 0.73 for HDL-C. **Conclusion:** Serum lipid parameters, particularly total cholesterol, LDL-C, and HDL-C, decline in direct proportion to advancing hepatic decompensation. Serum lipid profiling offers objective, accessible, and high-performing non-invasive biomarkers for assessing disease severity and stratifying risk in patients with liver cirrhosis.

Keywords: Liver cirrhosis, Lipid profile, Total cholesterol, HDL-C, MELD score, Child-Pugh score, ROC curve, Diagnostic performance.

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Introduction

Liver cirrhosis represents the common end-stage outcome for a broad array of chronic liver injuries, characterized by diffuse hepatic tissue remodeling, excessive extracellular matrix accumulation, and the formation of regenerative nodules.[1] Globally, cirrhosis accounts for over 1.2 million deaths annually, ranking among the leading causes of morbidity and mortality worldwide.[2] In clinical practice, the natural history of cirrhosis transitions from a prolonged compensated phase to a decompensated state marked by overt complications such as ascites, hepatic encephalopathy, variceal hemorrhage, and hepatorenal syndrome.[1,3]

The liver occupies a pivotal role in systemic lipid homeostasis.[4] It serves as the primary site for de novo synthesis, esterification, packaging, and clearance of cholesterol, triglycerides, apolipoproteins, and circulating lipoproteins.[5] Hepatocytes synthesize apolipoprotein B-100 (apoB-100) and assemble very low-density lipoproteins (VLDL), synthesize apolipoprotein A-I (apoA-I) to form nascent high-density lipoproteins (HDL), and regulate low-density lipoprotein (LDL) clearance via cell-surface LDL receptors.[4,6]

and Class C (10–15 points; severe decompensation).[1,11] In addition, another enzyme that is produced by hepatocytes, called lecithin-cholesterol acyltransferase (LCAT), has a fundamental role in the maturation of HDL and reverse

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cholesterol transport.

Liver cirrhosis results in a progressive loss of parenchyma and architectural destruction, leading to loss of liver synthetic and clearance function.[7] The metabolic derangements seen following the event, result in marked decreases in total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C).[8,9] Although the clinical importance of these metabolic changes is increasing, the traditional composite scores like the model for end-stage liver disease (MELD) score.[10] and the Child Pugh classification.[1] are mostly used in clinical risk stratification in cirrhosis. Although these models are useful, there are certain limitations, such as the subjective scoring for ascites and encephalopathy for Child-Pugh, and the variation in the measurement of prothrombin time/INR between laboratories.[11]

Biochemical markers with objective and standardized values that are easily measurable are continuously looked for to supplement existing markers of prognosis.[12] While a few studies have reported an inverse correlation between serum lipid levels with liver dysfunction, [8-10,13] there have been only a few studies that tried to assess the diagnostic accuracy and decision values of specific circulating lipid parameters to detect severe liver disease, especially in the alcoholic population. It was conducted to assess the fasting serum lipid profile derangements in the different child-pugh classes and the different MELD scores spectrums and the diagnostic value of the individual parameters, using the Receiver Operating Characteristic (ROC) curve analysis, for identifying severe liver cirrhosis.

Materials and Methods

Study Design and Setting: It was an analytical cross-sectional study done at Department of General Medicine, Koppal Institute of Medical Sciences (KIMS) Teaching Hospital, Koppal, Karnataka, India. KIMS Teaching Hospital is a tertiary care referral centre providing health care services to the rural and semi-urban communities in the north of Karnataka. This study was carried out for 18 months starting July 2023 and continuing until January 2024.

Ethical Considerations: The study protocol was approved in formal manner by the Institutional Ethics Committee of Koppal Institute of Medical Sciences. Written informed consent was provided for all individual participants, or their legally authorized representative, before the participants participated.

Study Population and Selection Criteria: All adult patients (age 18 and above) who were admitted to the medical wards and diagnosed with liver cirrhosis were consecutively screened. Cirrhosis was diagnosed by a combination of clinical signs, biochemical parameters, abdominal ultrasound and/or computed tomography (CT) scan features and/or histopathology, if present. Total 103 patients were included in the study.

Contamination by coexisting systemic metabolic diseases and drugs was avoided by strict exclusion criteria.

Exclusion criteria were: Known diabetes mellitus or systemic arterial hypertension; Preexisting cerebrovascular or coronary artery disease; Administration of lipid-lowering pharmacotherapy (e.g. statins) within the previous six months or acute or chronic pancreatitis; Primary thyroid disorders including hypothyroidism or hyperthyroidism; Active pregnancy; and inability to give informed consent.

Clinical evaluation and severity assessment: To perform clinical evaluation and clinical severity assessment.

Details of the demographic, clinical, lifestyle and physical examination data were collected on a structured proforma. The etiology of cirrhosis was determined by history, laboratory data, serological testing and imaging. Liver dysfunction was **categorised based on two clinical scoring systems:**

1. The Child-Pugh score and classification is made up of five factors: Total Bilirubin, Serum Albumin, Prothrombin time/INR, ascites severity, hepatic encephalopathy grade. Class A: mild/compensated (5-6 points), Class B: moderate decompensation (7-9 points), Class C: severe decompensation (10-11 points) and Class D: moribund or decompensation (12 points).
2. Model for End-Stage Liver Disease (MELD) and MELD-Na Scores: MELD score was calculated using the standardized logarithmic equation: MELD = 3.78 × ln[Total Bilirubin (mg/dL)] + 11.2 × ln[INR] + 9.57 × ln[Serum Creatinine (mg/dL)] + 6.43 [10]. MELD-Na score was calculated using the refined formula: MELD-Na = MELD + 1.32 × (137 - Na) - [0.033 × MELD × (137 - Na)], with serum sodium bound between 125 and 137 mEq/L.

Laboratory and Lipid Profile Measurements: Venous blood samples were collected after a mandatory 12-hour overnight fast. Hematological parameters were analysed using an automated hematology analyzer. Serum biochemical tests—including liver function assays (total and direct bilirubin, AST, ALT, ALP, GGT, total protein, albumin), renal parameters (blood urea, serum creatinine), and electrolytes (sodium, potassium)—were measured. Coagulation profiles (prothrombin time, INR) and viral serologies (HBsAg, Anti-HCV) were determined using standard reference assays.

Serum lipid parameters were measured on an automated clinical chemistry analyzer (Beckman Coulter AU680, USA):

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

Statistical Analysis: Receiver Operating Characteristic (ROC) curve analysis was conducted to establish the diagnostic

accuracy, Area Under the Curve (AUROC), sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of lipid parameters for identifying severe liver cirrhosis (defined as Child-Pugh Class C or MELD score ≥ 18). A two-tailed p-value < 0.05 was defined as statistically significant. [14,15]

Results

Baseline Demographic and Clinical Characteristics

A total of 103 cirrhotic patients were enrolled. The cohort had a mean age of 44.2 ± 11.8 years (median: 42 years, IQR: 35–51 years). Patients in their 4th and 5th decades constituted the majority of cases (67.0% combined). Males dominated the cohort, representing 80.6% (n = 83) of subjects, yielding a male-to-female ratio of 4.15:1.

Clinical presentation was dominated by decompensation features: abdominal distension was reported in 87.4% (n = 90) and clinical jaundice in 85.4% (n = 88) (Table 1). Alcohol consumption was recorded in 93.2% (n = 96) of subjects, with a mean duration of 7.8 ± 5.2 years and mean daily intake of 156.3 ± 42.7 mL.

Table 1: Clinical characteristics and etiology of cirrhosis.

Clinical Characteristic / Etiology	Frequency n (%) / Mean ± SD
Alcoholic Etiology	96 (93.2%)
Hepatitis B Virus (HBV)	3 (2.9%)
Other / Cryptogenic	4 (3.9%)
Abdominal Distension	90 (87.4%)
Jaundice	88 (85.4%)
History of Recurrent Admissions	26 (25.2%)
Blood Transfusion History	14 (13.6%)

Examination Findings and Biochemical Parameters: Physical examination demonstrated icterus in 89.3% (n = 92), pallor in 83.5% (n = 86), peripheral edema in 73.8% (n = 76), ascites in 86.4% (n = 89; moderate 43.7%, severe 27.2%), splenomegaly in 79.6% (n = 82), and abdominal collateral vein dilatation in 35.9% (n = 37). Summary laboratory investigations confirmed significant synthetic dysfunction, hyperbilirubinemia, anemia, and mild hyponatremia. [Table 2]

Table 2: Baseline laboratory and biochemical parameters of study subjects.

Laboratory 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
Total Bilirubin (mg/dL)	8.4 ± 7.3
Direct Bilirubin (mg/dL)	4.6 ± 4.2
Total Serum Protein (g/dL)	6.2 ± 1.1
Serum Albumin (g/dL)	2.4 ± 0.6
Prothrombin Time / INR	2.1 ± 0.8
Serum Sodium (mEq/L)	132.8 ± 6.4
Serum Creatinine (mg/dL)	1.2 ± 1.3

Disease Severity Distribution and Lipid Profile: According to Child-Pugh criteria, 15.5% (n = 16) were Class A, 35.9% (n = 37) Class B, and 48.5% (n = 50) Class C, reflecting advanced liver disease in nearly half of the study population. The overall mean MELD score was 18.4 ± 6.8, and mean MELD-Na score was 20.8 ± 7.2.

Evaluation of fasting serum lipid parameters demonstrated generalized hypolipidemia across the cohort. Mean lipid concentrations were: Total Cholesterol 147.2 ± 38.6 mg/dL, Triglycerides 108.4 ± 32.5 mg/dL, HDL-C 43.8 ± 12.4 mg/dL, LDL-C 67.2 ± 26.8 mg/dL, and VLDL-C 21.7 ± 6.5 mg/dL.

Comparison of Lipid Parameters Across Child-Pugh Classes: When stratified by Child-Pugh severity, all lipid parameters showed a statistically significant, stepwise decline from Class A to Class C. [Table 3]

Table 3: Comparison of lipid profile parameters across Child-Pugh severity classes.

Lipid Parameter (mg/dL)	Class A (n=16)	Class B (n=37)	Class C (n=50)	Overall (N=103)	p-value
Total Cholesterol	164.3 ± 32.1	151.7 ± 35.4	138.4 ± 39.2	147.2 ± 38.6	< 0.001
Triglycerides	124.6 ± 28.4	111.2 ± 30.8	101.2 ± 33.1	108.4 ± 32.5	0.012
HDL-C	52.6 ± 10.2	45.1 ± 11.4	39.4 ± 12.1	43.8 ± 12.4	< 0.001
LDL-C	86.4 ± 22.1	71.3 ± 24.8	58.2 ± 26.3	67.2 ± 26.8	< 0.001
VLDL-C	24.9 ± 5.7	22.2 ± 6.2	20.2 ± 6.6	21.7 ± 6.5	0.012

Tukey's post-hoc pairwise testing revealed significant differences in TC, LDL-C, and HDL-C between Class A vs Class C ($p < 0.001$) and Class B vs Class C ($p < 0.01$).

Receiver Operating Characteristic (ROC) Curve Analysis: To evaluate the diagnostic capability of individual lipid profile parameters in identifying severe liver cirrhosis (defined as Child-Pugh Class C or MELD score ≥ 18), ROC curve analysis was performed. [Table 4]

Table 4: Receiver Operating Characteristic (ROC) performance of lipid parameters for predicting severe liver cirrhosis.

Lipid Parameter	AUROC	95% CI	Cutoff	Sens.	Spec.	PPV	NPV
Total Cholesterol	0.76	0.67–0.85	≤ 140 mg/dL	74.0%	71.7%	71.2%	74.5%
LDL-C	0.75	0.65–0.84	≤ 62 mg/dL	72.0%	69.8%	69.2%	72.5%
HDL-C	0.73	0.63–0.83	≤ 41 mg/dL	70.0%	67.9%	67.3%	70.6%

Total cholesterol exhibited the highest overall accuracy (AUROC = 0.76), closely followed by LDL-C (AUROC = 0.75) and HDL-C (AUROC = 0.73). A serum total cholesterol cutoff of ≤ 140 mg/dL provided a balanced sensitivity of 74.0% and specificity of 71.7% for identifying severe cirrhosis. [Figure 1]

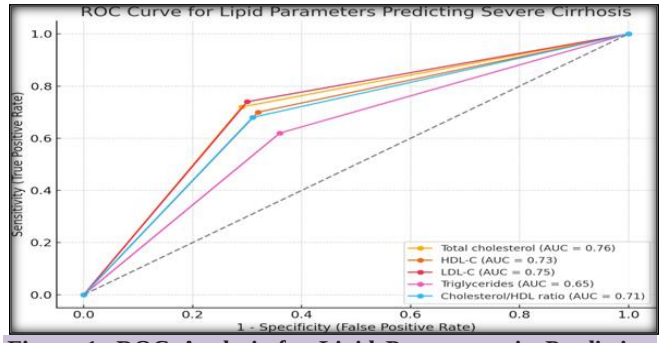

Figure 1: ROC Analysis for Lipid Parameters in Predicting Severe Cirrhosis

Discussion

These results of the analytical cross-sectional study show that the serum lipid profile parameters progressively and significantly decrease according to the severity of liver cirrhosis. All of the above were strongly correlated with both Child-Pugh and MELD severity scores and ROC curve analysis showed strong diagnostic power (AUROC 0.73-0.76) for severe liver decompensation.[1]

The synthesis, esterification, transport and catabolism of circulating lipids and lipoproteins primarily occurs in the liver. In cirrhosis, parenchymal injury, destruction of functional hepatocyte, and vascular distortion of cirrhosis hinder these metabolic processes.[5] HMG-CoA reductase activity is downregulated, which decreases de novo cholesterol production.[6]. Deficiencies in apolipoprotein production, such as apoA-I, apoA-II, and apoB-100 can impair the assembly and secretion of VLDL and HDL particles into the intravascular space.[6,7] In addition, the hepatic synthesis of LCAT is reduced which impairs HDL maturation, and LPL activity is decreased and portosystemic shunting further disturbs lipid kinetics in the periphery. [5,8]

Our findings corroborate with literature found in the international and national literature. Ghadir et al. found significant correlations between Child-Pugh classes and total cholesterol, LDL-C in 50 patients with cirrhosis.[8] Bassani et al. divided 74 cirrhotic patients and found that total cholesterol (TC), HDL-C, LDL-C significantly negatively correlated with Child-Pugh and MELD scores, and the mean value of TC was 117.6 mg/dL in cirrhotic patients.[9] Among the children with alcoholic cirrhosis, Chrostek et al. noted a gradual decrease in TC levels (5.53 mmol/L in Child A vs 2.91 mmol/L in Child C) and LDL-C.[10] The presence of HDL-C levels < 30 mg/dL was independently associated with high 18-month mortality rates, as emphasized by Habib et al.[16]

In Indian patients, Phukan et al. [17] also noted progressive decrease in all the lipid parameters with increasing Child Pugh functional class and similarly Mandal et al. [18] Khullar et al.[19] and Mohanty et al. [20] have documented this. In the UK, Mohanty et al. reported an AUROC of 0.752 for total cholesterol levels < 100 mg/dL in predicting 90-day mortality, similar to the diagnostic value in our ROC analysis (AUROC = 0.76 for TC ≤ 140 mg/dL).[20]

The diagnostic accuracy obtained in our study reinforces the clinical validity of fasting lipid profile. Routine lipid panels are inexpensive, fully automated, and easily available throughout the world, and the parameters they measure are consistently standardized. Routine lipid panels are not subjective as in the Child-Pugh score, do not have variable laboratory parameters, and are in standard automated and available worldwide. The addition of lipid parameters (50% total cholesterol ≤ 140 mg/dL or HDL-C ≤ 41 mg/dL) to clinical risk evaluation may provide an objective complementary tool for the early diagnosis of disease decompensation, clinical patient stratification on waiting lists for transplantation, and the follow-up for clinical disease progression.

This study has a few restrictions that should be taken into account. Firstly, the cross-sectional study design does not allow temporal sequence to be established or the long-term predictive value to be evaluated for hard clinical endpoints like 90 days or 1-year mortality. Second, the patients were from a single tertiary referral center in the southern part of India, which is predominantly alcoholic cirrhosis (93.2%), and may restrict direct generalizability to a population where viral cirrhosis or non-alcoholic steatohepatitis (NASH) is more common. Third, because of resource limitations it was not possible to measure directly apolipoproteins (apoA-I, apoB) and LCAT enzymatic activity. Further prospective studies, prospective multi-center studies, and long-term follow up studies with complete apolipoprotein profile are recommended.

Conclusion

As liver cirrhosis worsens in severity, there is a significant and predictable decrease in values of the serum lipid profile parameters, including the levels of total cholesterol, LDL-C and HDL-C. The fasting serum lipid profiling is an inexpensive, objective, reproducible, and widely available biochemical biomarker panel with good correlations to

Child-Pugh and MELD severity scores. Lipid profile (total cholesterol cut-off ≤ 140 mg/dL and HDL-C cut-off ≤ 41 mg/dL) as given in the routine clinical evaluation proved as a valuable non-invasive tool for the severity stratification and clinical management in cirrhotic patients.

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