
**E-ISSN:** 2706-9575
**P-ISSN:** 2706-9567
IJARM 2024; 6(3): 118-121
www.medicinepaper.net
Received: 14-06-2024
Accepted: 20-07-2024

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# A cross-sectional study for lipid profile as an indicator of severity in cirrhosis of liver

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**DOI:** https://doi.org/10.22271/27069567.2024.v6.i3b.579

**Abstract**
**Background:** Various scoring systems are available to assess the severity of cirrhosis, that is, the Child-Pugh score and Model for End-Stage Liver Disease (MELD) score. Since the liver is the major site for converting excess carbohydrates into various lipids, the deranged lipid profile can act as a prognostic biomarker of cirrhosis. We assessed the lipid profile abnormalities among patients with cirrhosis of the liver and correlated them with the severity of cirrhosis.
**Materials and Methods:** This study is an analytical cross-sectional examination of lipid profiles as a determinant of severity in liver cirrhosis among patients hospitalized in the medical ward of a tertiary care teaching hospital in Mandya. Subsequent to a thorough investigation and verification of cirrhosis, a fasting serum lipid profile was assessed in all qualifying individuals diagnosed with cirrhosis. Total serum cholesterol, triglycerides (TGL), and high-density lipoprotein (HDL) were assessed using direct methods, while serum low-density lipoprotein (LDL) and very-low-density lipoprotein (VLDL) were computed utilizing the Friedwald formula.
**Results:** A total of 70 patients were studied. Of them, 64(91.4%) were male. Of them, alcohol (59, 85.0%), hepatitis B (6, 8.5%), and nonalcoholic steatohepatitis (NASH) (4, 5.7%) were the most common cause of cirrhosis. A clear dose-response association (decreasing trend) is observed in lipid levels corresponding to escalating severity according to the Child-Pugh score. Moreover, cholesterol, LDL, and HDL levels were markedly reduced in patients with ascites or spontaneous bacterial peritonitis compared to their respective cohorts.
**Conclusion:** This study found that when the severity of cirrhosis develops, there is a significant decrease in the levels of lipid profile indices such as blood total cholesterol, LDL, VLDL, TGL, and HDL in patients. A more accurate evaluation of the prognosis of patients in light of morbidity and mortality might result from further development of the scoring system in conjunction with an already-existing scoring system.

**Keywords:** Cirrhosis, Child-Pugh score, chronic liver disease

## Introduction
The advanced stage of hepatocellular injury induced by a variety of etiologies is represented by liver cirrhosis, which may eventually lead to hepatocellular cancer and liver failure. Cirrhosis is present in 4.5% to 9.6% of the world's population, or more than 50 million people [1-3].
National statistics in the United Kingdom indicate that liver illnesses are the sixth leading cause of mortality [4]. In the United States of America, it is the second leading cause of death [5]. In 2015, cirrhosis affected 2.8 million people and resulted in 1.3 million deaths [6, 7]. Annually, almost 1 million fatalities result from cirrhosis complications, positioning cirrhosis as the 11th leading cause of mortality worldwide [8]. Approximately 1.5 billion individuals globally suffer from chronic liver disease (CLD), with an age-standardized incidence of CLD and cirrhosis at 20.7 per 100,000, reflecting a 13% increase since 2000. Likewise, the prevalence and mortality rates of cirrhosis have risen in recent years in the United States [9].
Multiple grading methods exist to evaluate the severity of cirrhosis, namely the Child-Pugh score and the MELD score. The liver is crucial for lipid metabolism, serving as the primary location for the conversion of surplus carbohydrates into triglycerides and fatty acids. The liver produces substantial amounts of cholesterol and phospholipids. The synthesis and metabolism of cholesterol are compromised in chronic liver disease (CLD). This ultimately leads to a reduction in plasma levels.

In cirrhosis, HDL cholesterol and its principal apolipoproteins are diminished due to significant metabolic disturbances, along with a decrease in blood LDL cholesterol levels. In this context, we evaluated the lipid profile anomalies in individuals with liver cirrhosis and their link with the severity of the condition.

## Materials and Methods

**Study Design:** It is an analytical cross-sectional study

### Study Setting

The study was conducted in a tertiary care teaching hospital in Mandya, Karnataka, India. Outpatient and inpatient services related to hepatology services are provided by the Department of Internal Medicine of the hospital. All clinically diagnosed patients with cirrhosis are admitted as inpatients and investigated in detail for further management.

### Study Population and Period

We included patients with cirrhosis admitted to the medical wards of the study hospital between April 2023 and July 2023.

### Inclusion Criteria

- Patients of age ≥18 years with cirrhosis.
- Admitted to the medical ward

### Exclusion Criteria

- Diabetes mellitus/hypertension.
- Cerebrovascular disease.
- Patients on lipid-lowering drugs.
- Pancreatitis. Chronic kidney disease.
- Hypo/hyperthyroidism

### Sample Size and Sampling

Based on results reported by Suman *et al*. [10] we calculated the sample size considering the mean [±standard deviation (SD)] difference of VLDL between the patients with MELD score 19-24 (16.8±2.7) and >24 (15.0±2.0), at 95% confidence interval and 80% power using Open Epi software. The calculated (minimum) sample size was 60.

### Study Procedure

All patients diagnosed with cirrhosis were administered a comprehensive hemogram, renal function assessment, liver function evaluation including serum proteins, abdominal ultrasonography, coagulation profile, and upper gastrointestinal endoscopy as part of standard care. Additionally, disease severity is classified by the Child-Turcotte-Pugh (CTP) score. The CTP score is determined by the following parameters: grading of ascites, serum albumin, serum bilirubin, prothrombin time, and severity of encephalopathy. CTP scoring has three grades; score 5-6 is class A, score 7-9 is class B, and score 10-15 is class C. After informed consent, the fasting serum lipid profile is measured among patients with cirrhosis. Total serum cholesterol, TGL, and high-density lipoprotein (HDL) were measured by the direct method, and serum LDL and VLDL were calculated using Fried Wald formula (LDL cholesterol = Total cholesterol − [HDL cholesterol − TGL/5] and VLDL = Sr. TGLs/5)

### Statistical Analysis

The primary data was collected, and it was analyzed using Statistical Package for the Social Sciences 16.0 version software. Multiple variables between variable groups of a single population are done by using the chi-squared test. Quantitative data between two or more groups were analyzed using the analysis of variance (ANOVA) test. A p-value of <0.005 is considered significant.

## Results

A total of 70 patients with cirrhosis were included in the study. Of them, 64 (91.4%) were males, and 6 (8.6%) were females. Alcohol (59, 87.0%), hepatitis B (6, 8.5%), and NASH (4, 5.7%) were the most common cause of cirrhosis. A total of 26 (37.1%), 29 (41.4%), and 26 (21.4%) patients were classified with Child-Pugh score categories A, B, and C, respectively. The mean and SD of each lipid level for each category based on the Child-Pugh score is given in Table 1. A clear dose-response relationship (decreasing trend) is seen in the levels of lipids for increasing severity based on the Child-Pugh score. The distribution and association of lipid profile with the presence of ascites and spontaneous bacterial peritonitis are given in Tables 2 and 3, respectively. The cholesterol, LDL, and HDL were significantly lower among patients with ascites and among patients with spontaneous bacterial peritonitis compared to their respective groups.

## Discussion

In this tertiary care hospital-based study, we found significantly low levels of lipids among patients with severe cirrhosis based on the Child-Pugh score.

**Table 1:** Lipid profile according to Child-Pugh score classification

| Lipid Profile Characteristics | A (n=26) | B (n=29) | C (n=15) |
|---|---|---|---|
| Cholesterol | Mean ± SD: 180.4±12.24 | Mean ± SD: 151.6±11.42 | Mean ± SD: 123.8±9.69 |
| Range | 155-214 | 132-182 | 104-137 |
| 95% Confidence Interval | 176.2-184.6 | 148.9-154.3 | 120.3-127.3 |
| p-value (ANOVA) | <0.001 | <0.001 | <0.001 |
| TGL | Mean ± SD: 155.1±9.18 | Mean ± SD: 132.7±8.73 | Mean ± SD: 94.5±9.9 |
| Range | 127-178 | 112-148 | 75-114 |
| 95% Confidence Interval | 151.0-159.2 | 129.2-136.2 | 88.8-100.2 |
| p-value (ANOVA) | <0.001 | <0.001 | <0.001 |
| LDL | Mean ± SD: 103.5±12.68 | Mean ± SD: 88.3±12.74 | Mean ± SD: 75.8±10.51 |
| Range | 79-143 | 71.0-119 | 58.7-96 |
| 95% Confidence Interval | 99.2-107.8 | 83.7-92.9 | 69.7-81.9 |
| p-value (ANOVA) | <0.001 | <0.001 | <0.001 |
| VLDL | Mean ± SD: 31.0±1.84 | Mean ± SD: 26.5±1.73 | Mean ± SD: 18.9±1.72 |
| Range | 25.5-35.6 | 22.4-34.5 | 15.1-22.8 |

| | | | |
|---|---|---|---|
| 95% Confidence Interval | 29.8-32.2 | 26.1-26.9 | 18.1-19.7 |
| p-value (ANOVA) | <0.001 | <0.001 | <0.001 |
| HDL | Mean ± SD: 45.9±5.30 | Mean ± SD: 36.7±4.41 | Mean ± SD: 29.2±3.67 |
| Range | 35-56 | 33-48 | 23-38 |
| 95% Confidence Interval | 44.5-47.3 | 35.4-38.0 | 27.7-30.7 |
| p-value (ANOVA) | <0.001 | <0.001 | <0.001 |

**Table 2:** Presence of ascites and lipid profile characteristics

| Lipid Profile Characteristics | Yes (n=54) | No (n=16) | p-value* |
|---|---|---|---|
| Cholesterol | Mean ± SD: 152.8±22.7 | Mean ± SD: 162.4±24.0 | 0.007 |
| TGL | Mean ± SD: 131.7±23.5 | Mean ± SD: 136.3±22.5 | 0.365 |
| LDL | Mean ± SD: 89.0±15.3 | Mean ± SD: 95.8±14.8 | 0.005 |
| VLDL | Mean ± SD: 26.3±4.9 | Mean ± SD: 27.0±4.7 | 0.365 |
| HDL | Mean ± SD: 37.6±7.6 | Mean ± SD: 39.7±8.0 | 0.032 |

**Table 3:** Presence of spontaneous bacterial peritonitis and distribution of lipid profile characteristics

| Lipid Profile Characteristics | Spontaneous Bacterial Peritonitis | Spontaneous Bacterial Peritonitis | p-value* |
|---|---|---|---|
| | Yes (n=16) | No (n=54) | |
| Cholesterol | 139.7 (20.6) | 152.5 (23.4) | 0.017 |
| TGL | 121.5 (24.7) | 128.9 (22.9) | 0.178 |
| LDL | 81.4 (11.4) | 89.1 (15.4) | 0.025 |
| VLDL | 24.3 (5.0) | 25.7 (4.6) | 0.178 |
| HDL | 34.0 (8.2) | 37.6 (7.4) | 0.045 |

Abnormalities in lipid profiles are significantly inversely correlated with the severity of cirrhosis. This study confirms that lipid profile abnormalities occur in cirrhosis due to reduced synthetic function in affected patients. The results aligned with prior studies. Our investigation revealed that measures such as blood total cholesterol, LDL, VLDL, HDL, and triglycerides dramatically decreased with the progression of cirrhosis severity. Fazl *et al*. did a comparable study, concluding that blood cholesterol, LDL, and HDL levels were considerably diminished in patients with cirrhosis compared to controls, whereas TGL levels were statistically insignificant. Serum cholesterol and other indicators are markedly diminished due to the substantial impairment of the liver's synthetic function in cirrhosis, resulting from fibrosis and nodule formation.

While Suman *et al*. documented a substantial area under the curve for blood cholesterol and LDL levels, which were statistically significant in relation to cirrhosis severity, they contrasted their findings with those of healthy persons, unlike our investigation.

The decreased levels of LDL and HDL might be attributed to the reduced synthesis of apolipoproteins A and B. Since apo B is involved in the synthesis of VLDL, the reduced level of TGLs is explained in cirrhosis. This can be due to insulin resistance found in liver cirrhosis. The insulin signaling mechanism in cirrhosis is found to be critical for lipogenesis regulated by phosphoinositide 3-kinase and AKT serine/threonine kinase 2 signaling pathways. Among the various transcription factors, sterol regulatory element binding protein-1c has a stimulatory effect on the genes involved in lipogenesis.

In contrast to earlier studies, our research demonstrates that all lipid profile indicators, including serum total cholesterol, serum HDL, triglycerides (measured directly), serum VLDL, and LDL (estimated using a formula), exhibit a negative correlation with the severity of cirrhosis. Furthermore, we have incorporated all qualifying patients admitted throughout the research duration.

Considering lipid profile abnormalities in CLD is of paramount importance to assess the severity since the changes are correlating statistically significant with previously existing severity assessment scores like the CTP score.

## Conclusion

Alterations in lipid profiles are frequently observed in individuals with cirrhosis. This study revealed a notable decrease in lipid profile markers, including serum total cholesterol, LDL, VLDL, TGL, and HDL, in individuals with cirrhosis as severity increases.

The presence of low lipid levels in cirrhosis patients presenting with altered sensorium and renal failure can aid in the diagnosis of hepatic encephalopathy and hepatorenal syndrome, respectively. The enhancement of the scoring system in conjunction with an existing scoring system may yield a more accurate evaluation of patients' prognoses concerning morbidity and death. It is also an economical approach. We advocate assessing the fasting lipid profile in all patients with cirrhosis to prognosticate disease development.

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**How to Cite This Article**
Manjesh KS, Chandregowda. A cross-sectional study for lipid profile as an indicator of severity in cirrhosis of liver. International Journal of Advanced Research in Medicine. 2024;6(3):118-121.

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