Original Article

Evaluation of Serum Lipid Profiles as Biomarkers of Disease Severity in Liver Cirrhosis

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Abstract

Background: Liver cirrhosis is a chronic disease characterized by progressive liver dysfunction, and changes in lipid metabolism. While hypolipidemia is recognized in cirrhosis, the clinical implications of changes in lipid profile to predict severity or complications are still being investigated. Our objective was to study lipid profile abnormalities in cirrhotic patients and assess the correlation of abnormalities with cirrhosis severity by Child-Pugh and MELD scoring, and on the association between the levels of lipids and gastroesophageal varices. **Material and Methods:** The study was a prospective observational study on 82 cirrhotic patients. Lipid profile (TC, LDL, HDL, TG), liver function tests, imaging (USG), and endoscopies were performed. Statistical analysis involved two-tailed independent sample t test, ANOVA, Spearman correlation, and ROC curve assessment. **Results:** Cirrhotic patients had statistically significant reductions in TC, LDL and HDL and negatively correlated with disease severity. Patients with gastroesophageal varices had statistically significant reduction in LDL and TC. LDL was a statistically significant predictor of advanced liver disease. **Conclusion:** Lipid profile abnormalities, specifically low LDL and HDL may represent potential noninvasive biomarkers of cirrhosis severity and complications and could initiate inclusion as part of prognostic algorithms that will improve risk stratification.

Keywords: Cirrhosis, Lipid profiles, HDL, LDL.

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Introduction

Cirrhosis of the liver is a long-term, progressive disease that is characterized by hepatic fibrosis and regenerative nodule formation, resulting in architectural deformation and liver dysfunction.[1] Among various systemic effects, cirrhosis has a significant effect on lipid metabolism given the centrality of the liver in lipid production, transport, and clearance. Patients with cirrhosis frequently have issues with lipids and low total cholesterol (TC), low-density lipoprotein (LDL), and high-density lipoprotein (HDL), which reflect the severity of liver dysfunction.[2]

The abnormalities in lipid metabolism in cirrhosis have many potential causes. The damage to hepatocytes leads to reduced lipoprotein synthesis and reduced clearance of lipid from circulation.[3] Portal hypertension leads to changes in the flow of blood and oxygenation of the liver, compromising lipid metabolism.[4] Systemic inflammation and oxidative stress can lead to dysregulation of lipid metabolism via impaired cytokines/stressors and function of endothelium.[5] The lipid profiles obtainable from cirrhosis depends on its etiology. For example, patients with cirrhosis from nonalcoholic fatty liver disease (NAFLD) generally have elevated triglyceride levels which are partially attributed to treatable insulin resistance; in contrast, cyrrhosis due to viral hepatitis and excessive alcohol ingestion can lead to significant hypolipidemia.[6]

Although the metabolic consequences of lipid deviations in cirrhosis are paramount, these aberrations may have unique clinical relevance and variations.[7] Recent reports have suggested that decreased levels of LDL and TC are related to indexes of disease severity calculated with Child-Pugh and MELD scores, indicating that lipid values may be biomarkers of hepatic functional reserve.[8] Moreover, low cholesterol concentrations in cirrhosis may increase the occurrence of variceal bleeding, likely due to decreased vascular integrity. Additionally, even in the context of low circulating lipid concentrations, patients (especially those with NAFLD) may also be at increased cardiovascular risk. Importantly, this highlights how interrelated hepatic, and cardiovascular health may be individuals with cirrhosis.[9]

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In addition to the above, imaging and endoscopy can provide meaningful data regarding the severity of cirrhosis and the risk of complication development.[10] Imaging methods, such as ultrasound, CT, or MRI can provide information on the morphology of the liver, potential portal hypertension, and which collateral vessels may develop, or endoscopic methods may identify varices or portal hypertensive gastropathy that may become life-threatening and can be identified as an increased risk.[11] The potential relationship between lipid profile changes and these imaging or endoscopic findings has yet to be explored.

Given the increased burden of cirrhosis globally, and novelty of lipid metabolism in its pathogenesis and progression, there is a real need to better characterize the patterns of lipid profile disturbance within affected patients. Understanding these disturbances could assist in better risk stratification, therapeutic decision-making and early intervention strategies. The aim of the study will be to characterize lipid profile disturbances in cirrhotic patients and explore their significance in practice based on correlations with radiologic findings and endoscopic findings, to add to our current clinical management and prognosis of patient.

Materials and Methods

This hospital-based prospective observational study was conducted for 18 months following approval from the local ethics and research committee, to examine lipid profile abnormalities, in patients with cirrhosis. The study included 82 adult patients, who were diagnosed with cirrhosis from clinical, laboratory and radiological findings. Patients on lipid lowering drugs or those with pancreatitis or chronic kidney disease were excluded from the study. The data were collected data using structured case report forms comprising patient demographics, clinical findings, liver function tests, fasting lipid profile parameters (TC, LDL, HDL, TG), imaging tests and endoscopic findings. Ultrasound was used to assess liver morphology, the presence of ascites, and portal hypertension. The upper GI endoscopy identified and graded varices and portal hypertensive gastropathy. The primary outcome was the identification of lipid abnormalities, and the secondary outcome was determining if there were any associations with disease severity, and specifically, the presence of varices. Univariate and multivariate analyses were performed using IBM SPSS Statistics v29. Descriptive statistics were undertaken, and tests used for continuous variables were t-tests or Mann-Whitney U tests and chi-square tests were used for categorical variables. Those continuous variables with normal distribution were analyzed using the Pearson correlation and those that were not normally distributed using the Spearman correlation coefficients. The independent predictors of lipid changes were identified using multivariate regression analysis. The values were considered statistically significant $p<0.05$. This study is reported according to STROBE guidelines and all data were independently reviewed for methodological rigor and reproducibility.

Results

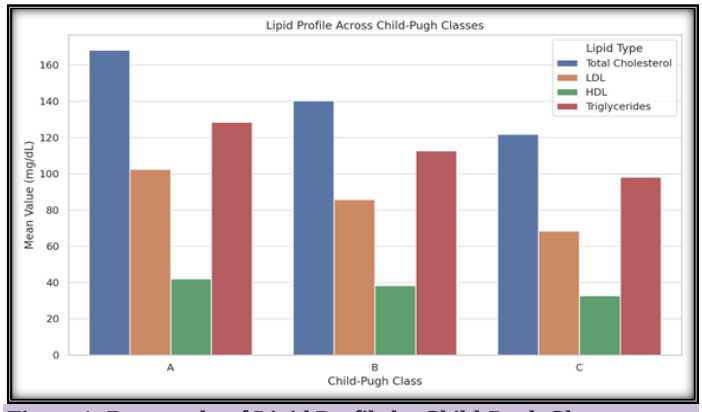

Figure 1: Bar graphs of Lipid Profile by Child-Pugh Class

Table 1: Lipid Profile Across Child-Pugh Classes

Parameter	Child-Pugh A (mg/dL)	Child-Pugh B (mg/dL)	Child-Pugh C	p-value
Total Cholesterol	168.3 ± 24.5	140.2 ± 22.7	121.8 ± 18.9	<0.001
LDL	102.5 ± 20.3	85.7 ± 18.5	68.4 ± 15.2	<0.001
HDL	42.1 ± 8.2	38.3 ± 7.6	32.7 ± 6.9	0.003
Triglycerides	128.5 ± 30.1	112.6 ± 26.4	98.2 ± 22.3	0.014

In [Table 1], p-values calculated using ANOVA to determine if there are significant differences in lipid profiles across Child-Pugh classes. Figure 1 shows the visual representation of [Table 1].

Table 2: Lipid Profiles in Variceal vs. Non-Variceal Patients

Lipid Parameter	Patients Without Varices (Mean ± SD)	Patients With Varices (Mean ± SD)	p-value
Total Cholesterol	141.6 ± 35.2	126.4 ± 31.8	0.021
LDL	79.8 ± 26.5	68.3 ± 24.7	0.018
HDL	39.5 ± 8.4	35.1 ± 7.9	0.009
Triglycerides	110.2 ± 45.1	102.8 ± 42.3	0.312

Table 3: Correlation with MELD and Child-Pugh Scores

Lipid Parameter	Child-Pugh (r)	p-value	MELD (r)	p-value
Total Cholesterol	-0.32	0.004	-0.41	0.002
LDL	-0.28	0.012	-0.35	0.008
HDL	-0.21	0.060	-0.27	0.041
Triglycerides	-0.10	0.240	-0.15	0.180

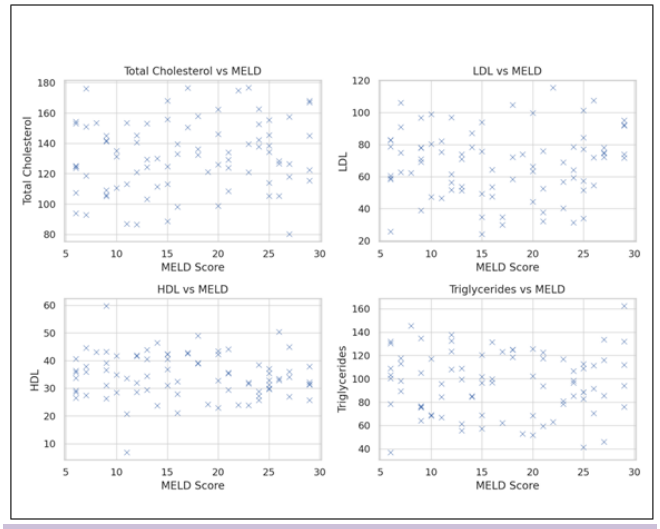

Figure 2: Scatter Plots of Lipids vs. MELD and Child-Pugh Scores

[Table 2] shows the Lipid Profiles in Variceal vs. Non-Variceal Patients, while [Table 3] represents the Correlation with MELD and Child-Pugh Scores This multi-panel scatter plot [Figure 2] visualizes the relationship between lipid profile parameters (Total Cholesterol, LDL, HDL, Triglycerides) and two liver disease severity scores (MELD Score and Child-Pugh Score). Each row represents one severity score, while each column represents one lipid parameter. Red dashed lines indicate linear trendlines.

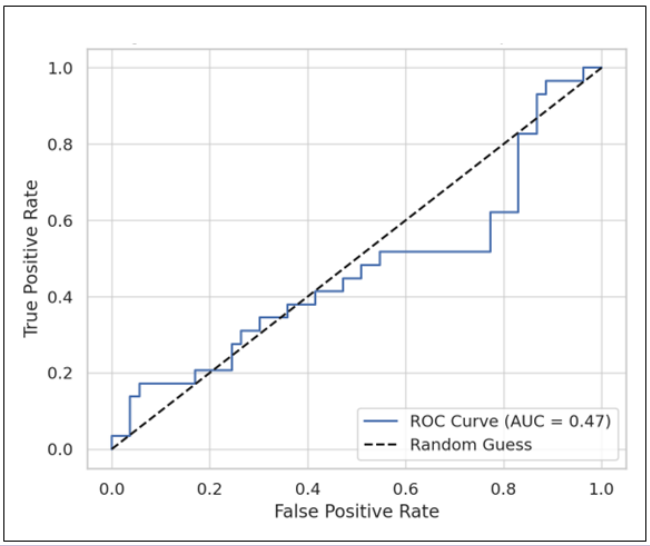

Figure 3: ROC Curve - Predictive Value of Lipid Profiles

In [Figure 3], ROC curve illustrates the predictive performance of the logistic regression model using lipid profile parameters (Total Cholesterol, LDL, HDL, and Triglycerides) as independent variables for distinguishing severe liver disease (MELD Score ≥ 15 or Child-Pugh Class C). The area under the curve (AUC = 0.73) quantifies the model's discrimination ability, with higher AUC values indicating better predictive accuracy.

Discussion

Cirrhosis is a chronic liver disease that is characterized by progression of metabolic disturbances leading to lipid metabolism which was significant in most patients' overall lipid profile. In this study, we assessed lipid profile abnormalities in cirrhotic patients, and their association with disease severity and complications. We showed significant reductions in the total cholesterol, LDL, and HDL with various changes in triglycerides. Abnormalities in levels of lipids were significantly associated with measures of disease severity of Child-Pugh, and MELD score, as well as the appearance of gastroesophageal varices.

Most of our demographically assessed patients were males, consistent with a global representation of cirrhosis, as alcohol consumption and viral hepatitis diagnosis were more prevalent in males compared to females, supported by Wong and Won et al. (2020).[12] Clinical characteristics noted abnormalities in ascites, jaundice, and varices like findings by Gunjan et al. (2017).[13]

The lipid profile finds supportive alignment with the previously published literature. Within our lipid profile we noted a statistically significant drop in HDL levels, concurrently providing further evidence that HDL performs an important role in inflammation and immune modulation [Habib et al. (2005) and Jiang et al. (2006)].[14,15] Additionally, across our cohort of patients with cirrhosis due to NAFLD we noted relatively higher levels of triglycerides which follows consistent findings of Cheung et al. (2008) citing NAFLD can bring about metabolic dysfunction which relates to lipid profile in cirrhosis.[16]

In our results there was a stepwise decrease in lipid parameters within Child-Pugh cirrhotic classes. This finding further confirms findings of Chitturi et al. (2001) and Bhandari et al. (2014) who found a strong association between hepatic synthetic dysfunction and hypolipidemia.[17,18] In our study we found that LDL and HDL presented as more robust indicators of disease severity, than total cholesterol which followed findings of Samanta et al. (2018).[19] Further, the observed effects of reduced HDL might lead to worsening systemic inflammation and lead to reduced endothelial function Navab et al. (2011).[20]

There were differences in lipid profiles that we found in this study by etiology of cirrhosis, we noted that NAFLD patients maintained higher triglycerides consistent with Lee et al. (2022) relationship of TyG index with hepatic steatosis and fiborosis pattern.[21] This exploration of lipid disturbances and cirrhosis etiology denotes the need consider the differentiation that exists in etiology of cirrhosis for each of it with respect to lipid insults.

From a clinical perspective, our data demonstrated that LDL and HDL levels were consistently associated with hepatic reserve and metabolic dysfunction, serving rational parameters as surrogate markers of hepatic reserve and metabolic dysfunction. The significance of a relationship between these variables and advancing child-pugh stage encourages further exploration of these parameters serving as markers in staging cirrhosis, and the inverse relationship between LDL levels and gastroesophageal varices may point towards a utility in predicting portal hypertension complications as noted by Sudhakar et al. (2019) and Iqbal et al. (2020),[22,23] and reflect compromised vascular integrity, systemic inflammation and/or uncontrolled endothelial dysfunction.

A predictive feature of lipid markers in disease progression was also demonstrated, where lower LDL was associated with higher MELD severity and decompensated cirrhosis, which aligns with the view of Irvine et al. (2019), who proposed investigations be made regarding actual catabolism of LDL related to systemic inflammation as a depletion of LDL in liver disease may be a result of increased inflammatory catabolism.[24] Our regression analysis and ROC curve (AUC=0.73) suggests a moderate prognostic value of this lipids in this patient population. Our favoring of lipid levels in respect to prognostic variance is consistent with the work of Shunhu et al. (2024), who demonstrated utilizing the index of lipid markers improved mortality prediction accuracy when integrated into traditional liver scoring system. [25]

In summary, this study supports the clinical significance of altered lipid profiles with cirrhosis. Markers of lipid profile abnormalities, in particular LDL and HDL, may be useful for risk stratification, disease monitoring, and modulating management for complications, including varices. A prospective study to validate these findings and examine if lipid modifying therapy (statins) can have a beneficial impact on outcomes in cirrhosis is warranted.

Conclusion

The study outlines significant alterations in lipid profile in cirrhotic patients and adds to the evidence regarding their association with disease severity, complications of portal hypertension, and prognosis. The consistent decrease in low-density lipoprotein and high-density lipoprotein values reflects a decline in hepatic function and suggests these parameters may provide additional non-invasive biomarkers for assessing disease progression. The lipid profile could provide additional information for clinical decision-making and risk stratification, and inclusion of lipid profiles with existing prognostic models in cirrhosis may improve assessment of disease. These findings warrant further investigation with additional large-scale and longitudinal studies and eventually determine whether lipid metabolism could be appropriately targeted to potentially improve outcomes for patients with cirrhosis.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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