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LIPID PROFILE AS AN INDICATOR OF SEVERITY IN CIRRHOSIS OF LIVER AND ITS CORRELATION WITH CHILD PUGH SCORE

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Abstract

Background: Liver cirrhosis is the final stage of chronic liver disease and is associated with progressive impairment of hepatic metabolic functions, including lipid metabolism.The liver serves as the primary site for synthesis, processing, and distribution of lipoproteins, making lipid metabolism highly susceptible to hepatocellular dysfunction. Progressive deterioration of liver function in cirrhosis leads to significant alterations in serum lipid profiles. The Child-Pugh scoring system remains a widely utilized clinical tool for assessing cirrhosis severity based on clinical and biochemical parameters.Alterations in serum lipid profile may reflect the severity of liver dysfunction and could serve as potential biochemical markers in cirrhotic patients.Understanding the relationship between lipid profile changes and disease severity may provide valuable insights into disease progression and offer additional objective markers for prognostication in cirrhotic patients. ***Objective:*** To evaluate lipid profile abnormalities in patients with liver cirrhosis and determine their association with disease severity using the Child-Pugh score. ***Methodology:*** This was a cross-sectional observational study included 75 patients diagnosed with cirrhosis of liver conducted at BLDE University Shri BM Patil Medical college Vijayapura over a period of 18 months from March 2024 to December 2025 to evaluate lipid profile abnormalities in patients with liver cirrhosis and to determine their correlation with disease severity using the Child-Pugh class. Comprehensive clinical history, physical examination, and laboratory investigations were conducted including liver function tests (total bilirubin, serum albumin, PT/INR) and complete lipid profile (total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, and VLDL cholesterol). Patients were classified into Child-Pugh Class A, B, and C. Statistical analysis employed one-way ANOVA and chi-square testing to examine correlations between lipid parameters and Child-Pugh classification. ***Results:*** The mean age of patients was 47.61 ± 11.34 years, with a predominance of males (93.3%). Alcohol was the most common etiology of cirrhosis (82.7%). The majority of patients were classified as Child-Pugh class C (57.3%), followed by class B (28.0%) and class A (14.7%). The mean levels of total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, and VLDL cholesterol were 116.36 ± 23.90 mg/dL, 75.51 ± 37.97 mg/dL, 27.21 ± 14.95 mg/dL, 68.45 ± 25.89 mg/dL, and 27.69 ± 21.11 mg/dL respectively. Total cholesterol ($p < 0.001$), HDL cholesterol ($p = 0.006$), LDL cholesterol ($p = 0.046$), and VLDL cholesterol ($p = 0.024$) showed significant associations with Child-Pugh classification, while triglycerides did not demonstrate a significant relationship ($p = 0.824$).***Conclusion:*** Patients with liver cirrhosis demonstrate significantly altered serum lipid profile parameters, particularly total cholesterol, HDL cholesterol, and LDL cholesterol, which exhibit strong inverse correlations with disease severity as determined by Child-Pugh classification. These alterations reflect the progressive deterioration of hepatic synthetic and metabolic functions. Lipid profile parameters, especially total cholesterol and HDL cholesterol, may serve as valuable objective, cost-effective, and readily accessible biomarkers for assessing disease severity and prognosis in cirrhotic patients. Incorporating lipid profile assessment into routine evaluation of cirrhotic patients may enhance clinical decision-making and risk stratification. Lipid profile evaluation may therefore serve as a simple and accessible supplementary tool for assessing the severity and progression of liver cirrhosis.

Keywords: Liver cirrhosis, lipid profile, Child-Pugh classification, total cholesterol, HDL cholesterol, LDL cholesterol.

INTRODUCTION

The final common pathway of chronic liver injury is liver cirrhosis, which is marked by architectural distortion, progressive hepatic fibrosis, and the development of regenerating nodules that significantly alter the hepatic parenchyma and its functional capacity. This irreversible disorder has a continuously rising incidence in both developed and developing nations, making it one of the leading causes of morbidity and mortality worldwide. Millions of people globally are thought to be affected by cirrhosis, which raises healthcare costs and reduces the quality of life for affected patients and their families. The fourth stage of chronic liver disease is liver cirrhosis which is caused by fibrosis, regeneration nodules, and disruption in the hepatic architecture and leads to progressive hepatic dysfunction. It is still one of the greatest causers of morbidity and mortality in the world, a fact that contributes satisfactorily to the deaths caused by liver globally every year [1].Due to the progressive nature of cirrhosis and the possibility of life-threatening complications like hepatocellular carcinoma, spontaneous bacterial peritonitis, hepatic encephalopathy, and variceal hemorrhage, it is essential to accurately assess and stratify the disease's severity in order to guide appropriate clinical management and forecast patient outcomes.[1]

As the primary location for the synthesis, processing, and export of several lipoproteins that are necessary for maintaining systemic lipid homeostasis, the liver plays a crucial and central role in lipid metabolism. Hepatocytes are responsible for producing apolipoprotein B-containing lipoproteins, such as very low-density lipoproteins and intermediate-density lipoproteins, as well as the synthesis of triglycerides, cholesterol, and phospholipids.

Additionally, the liver is involved in the production of bile acids from cholesterol, the secretion of biliary lipids, and the uptake and degradation of circulating lipoproteins. Since the liver is essential for maintaining lipid homeostasis, it stands to reason that cirrhosis and chronic liver disease would have a major effect on blood lipid levels and lipoprotein composition, thereby offering useful biomarkers for determining the extent of hepatic dysfunction. Significant changes in serum lipid profiles have been reported by numerous researchers in cirrhotic individuals, with the severity of the disease as determined by standard grading systems progressively worsening.[2]

Proper evaluation of the severity of the disease is needed to prevent complications in the circulatory and predict the prognosis, as well as the approach to treatment of cirrhosis patients [2]. Liver is very important in the metabolism of lipids, synthesis, processing and transportation of cholesterol, triglycerides and lipoproteins. Hepatocytes control the circulating lipid fractions which include very-low-density lipoprotein (VLDL), low-density lipoprotein (LDL) and high-density lipoprotein (HDL) [3].Over the course of several decades, clinical observations and biochemical studies have consistently shown that patients with cirrhosis have significantly different lipid profiles than healthy individuals. These disorders include qualitative abnormalities in the composition and function of lipoproteins as well as quantitative alterations in different lipid fractions. The lipid abnormalities in cirrhosis are caused by a variety of complex and multifactorial pathophysiological mechanisms including nutritional and metabolic factors, including reduced hepatic synthesis of lipoproteins and apolipoproteins, decreased activity of the liver-synthesized enzyme lecithin-cholesterol acyltransferase, decreased hepatic uptake of lipoproteins, portal-systemic shunting that bypasses hepatic metabolism, malnutrition and decreased dietary intake, and possible side effects of medications used to treat cirrhosis and its complications. Serum lipid characteristics may be easily accessible and reasonably priced indicators of the severity of cirrhosis, as the degradation of hepatic synthetic function appears to be correlated with increasingly severe disturbances in lipid metabolism.[3,4]

Since the metabolism of lipids is mostly dependent on the proper functioning of the hepatocytes, the chronic liver disease usually entails serious changes in the serum lipid profiles [5]. A number of studies have also documented that patients with cirrhosis often exhibit low levels of total cholesterol, LDL cholesterol, and HDL cholesterol through compromised hepatic production and the derailment of metabolite processes [6]. Such lipid disorders could indicate the extent of hepatocellular injury and the progressive worsening of liver functioning [7].

The Child-Pugh scale is among the most common scoring systems that are used to determine the degree of severity and prognosis of cirrhosis. It comprises five clinical and biochemical parameters, including serum bilirubin, serum albumin, prothrombin time or INR, ascites, and hepatic encephalopathy [8]. According to these parameters, patients are classified into category A, B, and C that indicate mild, moderate, and severe liver dysfunction respectively [9]. Recent investigations indicate that the serum lipid levels decrease gradually with the deteriorating cirrhosis and can be associated with the ChildPugh classification [10]. Since the lipid profile tests are cheap, readily accessible, and common tests in clinical practices, they can be used as effective biochemical indicators of assessing the severity of diseases [11]. These findings could enhance the clinical examination and surveillance of cirrhotic patients by identifying the connection between the changes in the lipid profile and the severity of the disease [12].The ability to determine the connection between the changes in the lipid profile and the severity of the disease could

help provide the clinical practice with improved risk stratification and disease monitoring [13]. The lipid abnormality can also indicate the metabolic effects of the progressive hepatocellular damage and low hepatic synthetic capacity [14]. Past researches have thus indicated that lipid parameters can add more information as to the disease progression in cirrhotic patients [15]. The lipid profile monitoring can also be a part of the improved clinical assessment and management of the chronic liver disease patients [16]. Thus, the current research was carried out to assess the lipid profile changes in the liver cirrhosis patients and to identify how these changes are correlated with severity of the disease according to the Child-Pugh classification system [17].

Objective: To evaluate lipid profile abnormalities in patients with liver cirrhosis and determine their association with disease severity using the Score.

METHODOLOGY

This was a cross-sectional observational study conducted at BLDE University Shri BM Patil Medical college Vijayapura over a period of 18 months from March 2024 to December 2025 to evaluate lipid profile abnormalities in patients with liver cirrhosis and to determine their correlation with disease severity using the Child-Pugh Score. The study sample comprised 75 patients diagnosed with liver cirrhosis who were admitted to or attending the outpatient department during the study period.

Inclusion Criteria

- Patients aged 18 years or older diagnosed with liver cirrhosis based on clinical, laboratory, and radiological findings.
- Patients willing to participate and who provided informed written consent.
- Patients with stable chronic liver disease undergoing routine clinical evaluation.

Exclusion Criteria

- Patients with hepatocellular carcinoma or acute liver failure.
- Patients receiving lipid-lowering medications such as statins.
- Patients with metabolic or endocrine disorders known to affect lipid metabolism.
- Patients with severe systemic illness or active infection.
- Patients with Cerebrovascular accident.
- Chronic kidney disease
- Patients diagnosed with Acute or Chronic Pancreatitis

Data Collection

After obtaining informed consent, demographic and clinical data including age, gender, and etiology of cirrhosis were recorded using a structured questionnaire. Detailed clinical examination was performed to identify features of chronic liver disease such as ascites and hepatic encephalopathy. Blood samples were collected after overnight fasting for laboratory investigations. Liver function tests including serum bilirubin, serum albumin, and prothrombin time/INR were measured. A complete lipid profile was performed for each patient including total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, and VLDL cholesterol. The severity of liver disease was assessed using the Child-Pugh scoring system, which categorizes patients into Child-Pugh class A, B, and C based on five clinical and biochemical parameters.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentage. The association between lipid profile parameters and Child-Pugh classification was analyzed using one-way analysis of variance (ANOVA) and chi-square test where appropriate. A p-value of <0.05 was considered statistically significant.

RESULTS

The average age of the participants was 47.61 ± 11.34 years, indicating that most patients were middle-aged adults. A strong male predominance was observed, with 70 patients (93.3%) being male and only 5 patients (6.7%) female. Regarding the underlying cause of cirrhosis, alcohol-related liver disease was the most common etiology, present in 62 patients (82.7%), while other causes of cirrhosis accounted for 13 patients (17.3%).

Table 1. Demographic and Clinical Characteristics of Patients with Liver Cirrhosis (N = 75)

Variable	Category	Patients n (%) / Mean ± SD	Total N	Percentage (%)
Age (years)	Overall patients	47.61 ± 11.34	75	100
Gender	Male	70 (93.3%)	75	93.3
	Female	5 (6.7%)	75	6.7
Etiology of Cirrhosis	Alcohol-related cirrhosis	62 (82.7%)	75	82.7
	Non-alcohol related cirrhosis	13 (17.3%)	75	17.3

Among the 75 patients studied, the majority were in Child-Pugh class C, representing severe cirrhosis, with 43 patients (57.3%). Moderate disease (Child-Pugh class B) was observed in 21 patients (28.0%), while only 11 patients (14.7%) were

classified as Child-Pugh class A, indicating mild disease. This distribution suggests that most patients in the study presented with advanced liver dysfunction.

Table 2. Distribution of Cirrhosis Severity According to Child-Pugh Classification (N = 75)

Child-Pugh Class	Score Range	Patients n (%)	Total Patients	Percentage (%)	Cumulative (%)
Class A	5–6	11 (14.7%)	75	14.7	14.7
Class B	7–9	21 (28.0%)	75	28.0	42.7
Class C	10–15	43 (57.3%)	75	57.3	100
Total	All patients	75 (100%)	75	100	100

The mean total cholesterol level was 116.36 ± 23.90 mg/dL, indicating overall hypocholesterolemia among cirrhotic patients. Triglyceride levels averaged 75.51 ± 37.97 mg/dL, remaining mostly within the normal range. HDL cholesterol was markedly reduced with a mean value of 27.21 ± 14.95 mg/dL, while LDL cholesterol also showed decreased levels with a mean of 68.45 ± 25.89 mg/dL. VLDL cholesterol levels averaged 27.69 ± 21.11 mg/dL and were relatively closer to normal. Overall, the findings demonstrate a pattern of reduced lipid parameters, particularly cholesterol fractions, in patients with liver cirrhosis.

Table 3. Serum Lipid Profile of Patients with Liver Cirrhosis (N = 75)

Lipid Parameter	Mean ± SD (mg/dL)	Total Patients	Percentage (%)	Clinical Range Category
Total Cholesterol	116.36 ± 23.90	75	100	Hypocholesterolemia
Triglycerides	75.51 ± 37.97	75	100	Normal range
HDL Cholesterol	27.21 ± 14.95	75	100	Markedly decreased
LDL Cholesterol	68.45 ± 25.89	75	100	Reduced levels
VLDL Cholesterol	27.69 ± 21.11	75	100	Near normal

Total cholesterol demonstrated a highly significant association with disease severity ($p < 0.001$), while HDL cholesterol ($p = 0.006$), LDL cholesterol ($p = 0.046$), and VLDL cholesterol ($p = 0.024$) also showed statistically significant relationships with worsening cirrhosis. In contrast, triglyceride levels did not show a significant association ($p = 0.824$). Liver function parameters including total bilirubin, serum albumin, and PT/INR all showed highly significant relationships with Child-Pugh class ($p < 0.001$). In addition, clinical complications such as ascites severity ($p < 0.001$) and hepatic encephalopathy ($p = 0.039$) were significantly associated with increasing disease severity. These findings indicate that worsening liver function is accompanied by progressive reductions in several lipid profile parameters.

Table 4. Association of Lipid Profile and Clinical Parameters with Child-Pugh Classification (N = 75)

Variable	Statistical Test	p-value	Total Patients	Percentage (%)	Statistical Significance
Total Cholesterol	One-way ANOVA	<0.001	75	100	Significant
HDL Cholesterol	One-way ANOVA	0.006	75	100	Significant
LDL Cholesterol	One-way ANOVA	0.046	75	100	Significant
VLDL Cholesterol	One-way ANOVA	0.024	75	100	Significant
Triglycerides	One-way ANOVA	0.824	75	100	Not significant
Total Bilirubin	One-way ANOVA	<0.001	75	100	Significant
Serum Albumin	One-way ANOVA	<0.001	75	100	Significant
PT/INR	One-way ANOVA	<0.001	75	100	Significant
Ascites Severity	Chi-square test	<0.001	75	100	Significant
Hepatic Encephalopathy	Chi-square test	0.039	75	100	Significant

DISCUSSION

Liver cirrhosis represents the end-stage of chronic liver disease and is characterized by extensive fibrosis and nodule formation, leading to profound alterations in hepatic architecture and function. The liver plays a pivotal role in lipid metabolism, being the primary site for synthesis, storage, and distribution of lipoproteins. Given this central role, it is logical to expect that progressive hepatocellular dysfunction in cirrhosis would manifest as significant alterations in serum lipid profiles. In the current research, lipid profile abnormalities in patients with liver cirrhosis and their correlation with the severity of the disease were assessed (Child-Pugh classification). The results revealed that majority of the patients were middle aged with the mean age of 47.61-11.34, and a high male dominance was found (93.3%). Alcohol-related liver disease was found to be the most frequently occurring etiology of cirrhosis with 82.7 percent being the most frequent. The distribution of patients across Child-Pugh classes revealed that the majority (57.3%) were classified as Child-Pugh Class C, followed by Class B (28.0%) and Class A (14.7%), suggesting that patients in our setting often present late in the disease course.[8]Our study revealed markedly reduced lipid profile parameters, with all patients (100%) having total cholesterol levels in the desirable range with a mean of 116.36 ± 23.90 mg/dL, demonstrating universal hypocholesterolemia. Total cholesterol showed a highly significant correlation with Child-Pugh classification ($p < 0.001$), with levels declining from 142.45 mg/dL in Class A to 105.86 mg/dL in Class B and 114.81 mg/dL in Class C . These findings are strongly supported by numerous studies in the literature. Ghadir et al. demonstrated a significant negative correlation between liver damage severity and serum total cholesterol levels, with more severe liver damage associated with greater declines in cholesterol.[10]Similarly, Cicognani et al. reported significantly decreased total cholesterol levels in patients with cirrhosis compared to control subjects, with progressive reduction correlating with disease severity.[11] The pathophysiological basis for hypocholesterolemia in cirrhosis is multifactorial, involving decreased synthesis of cholesterol, reduced activity of lecithin-cholesterol acyltransferase (LCAT), and impaired formation of lipoproteins due to progressive hepatocellular dysfunction.[12]

HDL cholesterol levels were markedly reduced in our study, with 94.7% of patients having low HDL (<50 mg/dL) and a mean value of 27.21 ± 14.95 mg/dL. The correlation with Child-Pugh classification was highly significant ($p = 0.006$), with mean HDL levels progressively declining from 40.18 mg/dL in Class A to 24.35 mg/dL in Class C . These findings align remarkably well with published literature. Chrostek et al. reported that HDL cholesterol correlated significantly with the severity of liver disease in both alcoholic and non-alcoholic cirrhosis, indicating that HDL-cholesterol may serve as a biomarker of liver dysfunction severity.[13] Habib et al. demonstrated that HDL cholesterol was independently predictive of mortality in non-cholestatic cirrhotic patients, with low HDL levels being a strong prognostic indicator.[14] The mechanisms underlying HDL reduction in cirrhosis include decreased hepatic synthesis of apolipoprotein A-I, reduced LCAT activity, and altered HDL composition and functionality.[15,16]

The same trends in demographics have been reported in earlier studies where cirrhosis was mainly reported to be prevalent in middle aged men and alcohol use was found to be a significant cause of chronic liver disease in large groups of people [18]. The allocation of the patients based on Child-Pugh classification showed that most of the patients were in the advanced liver disease. In this study, 57.3 percent of the patients were in the Child-Pugh class C with the other 28.0 percent in class B and 14.7 percent in class A. This observation shows that a majority of the patients were brought with a severe state of hepatic dysfunction during the examination. Similar observations were previously noted in other studies, in which a great percentage of cirrhotic patients were reported to be of the higher Child-Pugh classes, indicating the late stage of the disease or late presentation of the disease [19]. Serum lipid profile analysis showed significant changes in the patients with cirrhosis. The overall cholesterol level was at 116.36 + 23.90 mg/dL suggesting the existence of hypocholesterolemia with the HDL cholesterol being at a significantly lower level with the mean of 27.21 + 14.95mg/dL. Further reduction of LDL cholesterol was also achieved with a mean of 68.45/25.89mg/dL. The level of triglycerides was 75.51 plus or minus 37.97 mg/dL, which was more or less normal and VLDL level had an average of 27.69 plus or minus 21.11mg/dL. These results contribute to the idea that cirrhosis is related to the broken lipid metabolism because of the reduced hepatic synthesis and lipoprotein processing. The same results have been observed in other researches in which the levels of total cholesterol, HDL, and LDL cholesterol were much lower in cirrhotic patients as the liver disease progressed [20].

Another finding of the current research was that there was a strong correlation between the parameters of lipid profile and the severity of the disease. There was a very significant correlation between Child-Pugh classification and total cholesterol ($p = 0.001$), and HDL cholesterol ($p = 0.006$), LDL cholesterol ($p = 0.046$), and the VLDL cholesterol ($p = 0.024$) demonstrated significant associations with progressive cirrhosis. Triglycerides, however, did not display any notable correlation with the severity of the disease ($p = 0.824$). The same has been noticed in earlier studies when the total cholesterol and HDL cholesterol decreased gradually as the child-Pugh classes increased and this suggests that lipid parameters can be used as an indicator of hepatic synthetic function [21]. Besides lipid parameters, traditional parameters of

liver functionality were also strongly associated with the severity of the disease. There were very significant relationships between total bilirubin, serum albumin, and PT/INR and Child-Pugh classification ($p < 0.001$). Moreover, a high level of ascites ($p < 0.001$) and hepatic encephalopathy ($p = 0.039$) were also potential clinical complications with a significant impact on advanced stages of the disease. The same past studies have also noted that increasing liver functioning indicators and occurrence of complications are also strongly associated with increasing Child-Pugh classes and low clinical prognoses [22]. In general, the results of the current research confirm the assumption that the level of liver cirrhosis is strictly linked to the abnormalities of lipid profiles. Lowering of the total cholesterol, HDL cholesterol, and LDL cholesterol levels seem to indicate a poor hepatic synthetic activities and hepatocellular dysfunction. The same trends have been being steadily reported in prior studies, indicating that the parameters of lipid profile can be used as helpful auxiliary indicators in the determination of the severity of cirrhosis. Since lipid profile is cheap and easily accessible, as well as a routine test, its inclusion in the clinical assessment of patients with cirrhosis could be used to enhance the assessment and management of the disease.

CONCLUSION

It is concluded that patients with liver cirrhosis demonstrate significant alterations in serum lipid profile parameters that correlate with the severity of liver disease. In the present study, reduced levels of total cholesterol (116.36 ± 23.90 mg/dL), HDL cholesterol (27.21 ± 14.95 mg/dL), and LDL cholesterol (68.45 ± 25.89 mg/dL) were observed, reflecting impaired hepatic synthetic and metabolic functions, while triglycerides (75.51 ± 37.97 mg/dL) remained relatively within the normal range and VLDL cholesterol (27.69 ± 21.11 mg/dL) showed mild alterations. A significant association was found between lipid parameters and Child-Pugh classification, with total cholesterol, HDL cholesterol, LDL cholesterol, and VLDL cholesterol decreasing as disease severity increased, whereas triglycerides showed no significant correlation. These findings indicate that lipid profile abnormalities mirror progressive hepatic dysfunction and may serve as simple, inexpensive, and readily available supplementary biomarkers for evaluating disease severity and monitoring progression in patients with liver cirrhosis.

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