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Original Research Article

LIPID PROFILE ALTERATIONS IN CHRONIC LIVER DISEASE: ASSOCIATION WITH CHILD-PUGH SCORE

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ABSTRACT

Background: Chronic liver disease (CLD) is a progressive condition that alters hepatic lipid metabolism and leads to characteristic changes in serum lipid levels. The Child–Turcotte–Pugh (CTP) score is widely used to grade liver dysfunction, and correlation of lipid parameters with this score may provide a simple adjunct for assessing disease severity. The objective is to study serum lipid profiles in patients with chronic liver disease and evaluate their correlation with Child-Pugh score.

Materials and Methods: This hospital-based cross-sectional study was conducted on 250 patients with diagnosed CLD at Dr. Susheela Tiwari Memorial Government Hospital, Haldwani. Clinical evaluation, laboratory investigations, and fasting lipid profiles were performed. Disease severity was graded by the Child-Pugh classification. Spearman's rank correlation was applied to assess the relationship between lipid fractions and Child-Pugh score.

Results: Of the 250 patients, 68.4% were male and 31.6% female. The majority belonged to Child-Pugh class C (56%), followed by class B (36.8%) and class A (7.2%). Mean lipid values were: total cholesterol 136.44 ± 12.40 mg/dL, LDL 89.43 ± 19.20 mg/dL, HDL 31.50 ± 19.85 mg/dL, and triglycerides 114.07 ± 15.50 mg/dL. A statistically significant negative correlation was observed between Child-Pugh score and HDL, LDL, and total cholesterol, while triglycerides showed a weak, non-significant positive correlation. Ascites was the most common complication (64.4%).

Conclusion: Lipid abnormalities, particularly reduced cholesterol and HDL levels, were common in CLD patients and correlated inversely with Child-Pugh score. Lipid profile estimation may serve as a cost-effective adjunct to clinical and biochemical assessment of disease severity.

Keywords: Chronic liver disease, lipid profile, Child-Pugh score, cirrhosis, dyslipidemia.

INTRODUCTION

Chronic liver disease (CLD) is a major global health concern that significantly contributes to morbidity and mortality worldwide. It is characterized by progressive and long-standing hepatic injury, inflammation, and fibrosis that eventually compromise liver function. If left untreated, CLD can advance to cirrhosis, liver failure, and hepatocellular carcinoma (HCC), which together constitute some of the leading causes of death globally.[1] Recent estimates indicate that liver-related diseases account for more than two million deaths annually, making CLD one of the most pressing challenges in modern hepatology.[2]

The burden of CLD is diverse in its etiology. Common causes include chronic infections with hepatitis B virus (HBV) and hepatitis C virus (HCV), alcoholic liver disease (ALD), non-alcoholic fatty liver disease (NAFLD), autoimmune liver disorders, and cholestatic conditions such as primary biliary cholangitis (PBC) and primary sclerosing cholangitis (PSC).[3] Of these, NAFLD is emerging as a significant public health issue, largely due to

increasing rates of obesity, type 2 diabetes, and metabolic syndrome worldwide.[4] In many regions, particularly developing countries, alcohol-related liver disease and viral hepatitis continue to be leading contributors, while autoimmune and hereditary conditions such as Wilson's disease and hemochromatosis also play important roles. Despite advances in medical care, the silent and progressive nature of CLD means that diagnosis often occurs late, when disease has already progressed to cirrhosis or decompensation. This delayed recognition is associated with high treatment costs, prolonged hospital stays, reduced quality of life, and poor survival outcomes.[5]

The liver is central to metabolic regulation, particularly in lipid metabolism. It synthesizes, stores, and regulates cholesterol, triglycerides, and lipoproteins that are essential for energy balance, cell membrane integrity, and hormone synthesis. Dysregulation of lipid metabolism is a common feature of CLD and manifests as characteristic changes in lipid profiles. These alterations vary depending on the etiology and stage of disease. For example, in advanced cirrhosis, levels of total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) are often significantly reduced due to impaired hepatic synthetic capacity.[6] Conversely, in early NAFLD, triglyceride and cholesterol levels may be elevated due to excessive hepatic fat accumulation, before declining with worsening fibrosis and hepatocellular dysfunction.[7,8] Thus, lipid derangements in CLD are not uniform but instead reflect underlying pathophysiology and disease stage.

A growing body of evidence suggests that lipid profile alterations may hold prognostic significance in CLD. Reduced cholesterol and lipoprotein levels have been correlated with advanced disease and poor survival, making them potential biomarkers for hepatic reserve and prognosis.[9] Moreover, lipid abnormalities in CLD carry systemic implications. While NAFLD patients with hyperlipidemia are at elevated risk of cardiovascular disease, cirrhotic patients with hypolipidemia paradoxically continue to face cardiovascular and infectious risks due to systemic inflammation, oxidative stress, and endothelial dysfunction.[10] Recognizing and monitoring these lipid changes is therefore important not only for liver disease assessment but also for broader clinical management.

The Child–Turcotte–Pugh (CTP) score remains a cornerstone in the clinical evaluation of CLD. Initially developed in the 1960s for surgical risk assessment, it has since been refined and is widely used for staging the severity of cirrhosis. The CTP score incorporates five clinical and laboratory parameters: serum bilirubin, serum albumin, prothrombin time (international normalized ratio, INR), ascites, and hepatic encephalopathy. Based on the total score, patients are categorized into Child-Pugh class A (mild disease), class B (moderate disease), or class C (severe disease). This classification not only predicts survival but also guides therapeutic decisions and transplant prioritization.[11]

Several studies have explored the association between lipid profiles and CTP classes, consistently demonstrating that advancing Child-Pugh class is associated with worsening lipid derangements. Patients in class C often have the lowest levels of cholesterol, LDL, and HDL, reflecting severely impaired hepatic synthetic function. In contrast, those in class A may retain relatively preserved lipid levels.[12,13] These findings suggest that lipid profile assessment, when combined with CTP classification, may provide a more comprehensive understanding of disease status. It also highlights the potential of lipid parameters as accessible, cost-effective, and non-invasive adjuncts in prognostic evaluation.

Despite extensive research, gaps remain in fully understanding the interplay between lipid abnormalities and liver disease severity. Findings regarding triglyceride and very-low-density lipoprotein (VLDL) levels have been inconsistent, and the role of advanced lipid subfractions such as apolipoproteins remains underexplored. Furthermore, most studies to date have been cross-sectional, limiting the ability to assess longitudinal trends in lipid changes during disease progression. Variability in results across populations also underscores the influence of etiology, nutritional status, and comorbidities, emphasizing the need for more standardized and comprehensive investigations.

Against this backdrop, the present study aims to evaluate lipid profile alterations in patients with chronic liver disease and assess their correlation with the Child-Pugh score. By systematically analyzing changes in cholesterol, triglycerides, LDL, HDL, and VLDL in relation to hepatic function, this study seeks to clarify the role of lipid profiles as indicators of disease severity. Understanding these associations may enhance early detection of decompensation, improve risk stratification, and inform both clinical and therapeutic decision-making. Ultimately, the integration of lipid profile monitoring into routine practice could provide a simple yet effective tool in the holistic management of chronic liver disease.

Several studies have highlighted the association between lipid profile alterations and the severity of chronic liver disease. Pandey et al. (2023) observed that all lipid parameters were significantly reduced in cirrhotic patients compared to healthy controls, with the decline most marked in advanced Child-Pugh Class C cases. Similarly, Verma et al. (2023) reported a significant inverse correlation between serum lipid levels and Child-Turcotte-Pugh (CTP) score, suggesting that lipid profiles can serve as simple adjunct markers for staging chronic liver disease severity. Badawi et al. (2021) demonstrated that cholesterol, low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL) decreased

with worsening cirrhosis, and these reductions correlated negatively with both fibrosis and inflammatory indices. In a rural tertiary care setting, Som et al. (2019) found that both alcoholic and non-alcoholic cirrhotic patients had significantly lower levels of cholesterol, LDL, and high-density lipoprotein (HDL) compared to controls, regardless of disease etiology. Likewise, Jaiswal and Choubey (2018) observed a progressive decline in cholesterol, HDL, VLDL, and triglycerides with advancing Child-Pugh class, reinforcing the role of lipid profiling in assessing liver dysfunction. Jatav et al. (2018) also reported that lipid levels were markedly lower in cirrhotic patients than in controls, with significantly reduced cholesterol, HDL, and LDL in Child-Pugh Class C compared to Class B, underscoring their importance as indicators of disease severity.

MATERIALS AND METHODS

This was a hospital-based cross-sectional study conducted in the Department of General Medicine at Dr. Susheela Tiwari Memorial Government Hospital, Government Medical College, Haldwani, Nainital, Uttarakhand. The study was carried out over a period of 18 months following approval from the Institutional Ethics Committee. Informed written consent was obtained from all participants prior to inclusion, and strict confidentiality was maintained throughout the study. Both indoor and outdoor patients attending the medicine department and diagnosed with chronic liver disease were considered for inclusion. A total of 250 patients were enrolled using convenient sampling. The inclusion criteria comprised patients aged above 16 years with a confirmed diagnosis of chronic liver disease who provided informed consent. Patients with comorbid conditions known to alter lipid metabolism, including diabetes mellitus, malignancy, acute pancreatitis, renal failure, recent parenteral nutrition, or those receiving lipid-lowering or glucose-lowering medications, were excluded.

A detailed clinical history and thorough physical examination were performed for each participant. Sociodemographic data including age, sex, and relevant risk factors were recorded. Routine laboratory investigations were carried out in the Departments of Biochemistry and Pathology at GMC Haldwani. These included hemoglobin, total and direct bilirubin, prothrombin time (PT), serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), and assessment for hepatic encephalopathy. Lipid profile parameters measured were total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL).

The severity of liver disease was assessed using the Child-Turcotte-Pugh (CTP) scoring system, which incorporates serum bilirubin, serum albumin, prothrombin time/international normalized ratio (INR), presence of ascites, and hepatic encephalopathy. Based on the total score, patients were categorized into Child-Pugh Class A, B, or C, reflecting mild, moderate, and severe liver dysfunction, respectively.

Data were entered into Microsoft Excel after proper coding and cleaning. Descriptive statistics such as mean, frequency, and percentages were calculated. The association between lipid parameters and Child-Pugh score was evaluated using Spearman's rank correlation coefficient (ρ), a non-parametric test suitable for ordinal and non-normally distributed data. A p-value of less than 0.05 was considered statistically significant.

RESULTS & DISCUSSION

In the present study, a total of 250 patients with chronic liver disease were included. Among them, 171 (68.4%) were males and 79 (31.6%) were females. The age distribution shows that most patients with chronic liver disease in this cohort were middle-aged to elderly. This is depicted by [Table 1].

The largest share fell in the 41–60 years group (41.6%), followed by 61–80 years (34.0%). Younger adults aged 21–40 years comprised 21.2% of the sample, whereas the extremes—0– 20 and 81–100 years—were minimally represented at 1.2% and 2.0%, respectively.

Table 1: Distribution of Patients by Age Group

Age group	Frequency	Percentage (%)
0-20	3	1.2
21-40	53	21.2
41-60	104	41.6
61-80	85	34.0
81-100	5	2.0
Total	250	100.0

The analysis of educational status among the 250 patients [Figure 1] with chronic liver disease in this study reveals that the largest proportion had completed high school education (38.4%), followed by those with no formal education (16.0%) and those educated up to the intermediate level (13.6%). Primary education accounted for 11.2% of the participants, while only 7.6% were graduates and 2.0% postgraduates. A smaller fraction of the cohort had attained higher secondary education (6.4%) or middle school education (2.4%), and 2.4% were completely illiterate.

In this study, the majority of patients with chronic liver disease (66.8%) were from rural areas, while 33.2% were from urban regions. This rural predominance may be attributed to factors such as higher prevalence of alcohol consumption, viral hepatitis, limited healthcare access, and lower awareness about liver disease in rural populations. This is depicted by [Figure 2].

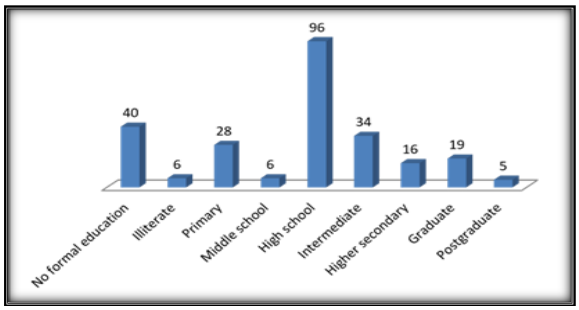

Figure 1: Educational Status of Patients

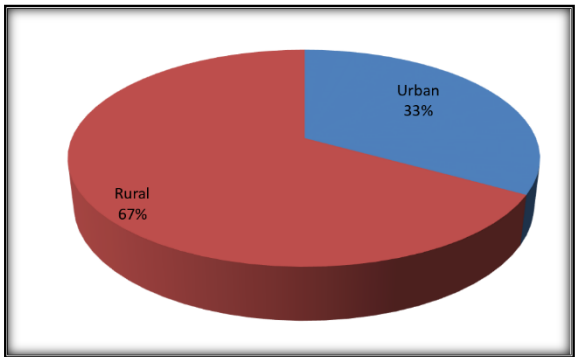

Figure 2: Distribution of Patients by Area of Residence

The analysis of socio-economic status reveals that nearly half of the patients (48.8%) belonged to the lower middle class, followed by 35.2% from the upper middle class. The lower socio-economic class accounted for 16.0% of the cases.

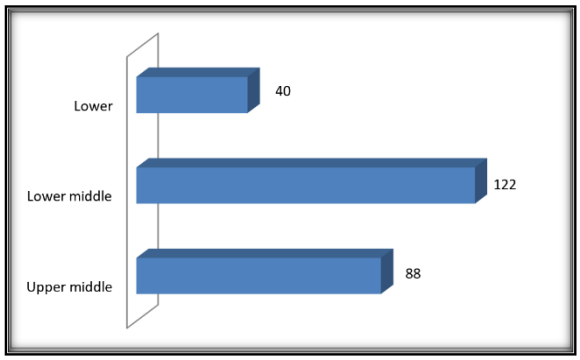

Figure 3: Socio-Economic Status of Study Population

The mean haemoglobin of the study population was 9.42 ± 2.43 g/dL, indicating that a significant proportion of patients were anemic, which is commonly observed in chronic liver disease due to nutritional deficiencies, chronic blood loss, or hypersplenism. The mean prothrombin time was 38.86 ± 20.38 seconds, and the mean international normalized ratio was 1.87 ± 0.78, both of which reflect impaired hepatic synthetic function and coagulation abnormalities in these patients. Albumin levels were reduced, with a mean of 2.72 ± 0.57 g/dL, consistent with diminished protein synthesis in chronic liver disease.

Table 2: Baseline Characteristics of Study Population

Parameter	Mean ± SD
Haemoglobin (g/dL)	9.42 ± 2.43
Prothrombin Time (sec)	38.86 ± 20.38
INR	1.87 ± 0.78
Albumin (g/dL)	2.72 ± 0.57
Total Bilirubin (mg/dL)	5.67 ± 2.75
Direct Bilirubin (mg/dL)	3.55 ± 1.43
SGOT / AST (U/L)	182.71 ± 20.83
SGPT / ALT (U/L)	113.04 ± 13.42

Bilirubin levels were elevated, with a mean total bilirubin of 5.67 ± 2.75 mg/dL and direct bilirubin of 3.55 ± 1.43 mg/dL, suggesting the presence of both hepatocellular dysfunction and cholestasis. The liver enzymes SGOT and SGPT were raised, with mean values of 182.71 ± 20.83 U/L and 113.04 ± 13.42 U/L respectively, indicating ongoing hepatocellular injury. Although these enzyme elevations are relatively moderate in the mean, some patients may have had higher levels, reflecting variability in the degree of hepatic injury.

The mean high-density lipoprotein cholesterol (HDL-C) was 31.50 ± 19.85 mg/dL, which is below the standard reference range of 40–60 mg/dL, indicating a reduction in the "good cholesterol" among these patients. Low HDL levels are commonly associated with impaired hepatic synthesis, malnutrition, and chronic inflammation in liver disease. The mean low-density lipoprotein cholesterol (LDL-C) was 89.43 ± 19.20 mg/dL, which falls within the normal upper limit (<100 mg/dL), suggesting that LDL levels are relatively preserved in this cohort despite the presence of chronic liver injury. Total cholesterol was 136.44 ± 12.40 mg/dL, which is lower than the normal range (<200 mg/dL), reflecting an overall reduction in circulating cholesterol, likely due to decreased hepatic production. Triglycerides had a mean value of 114.07 ± 15.50 mg/dL, which is within the normal reference range (<150 mg/dL), though a proportion of patients may still exhibit hypolipidemia in advanced liver disease.

Table 3: Serum lipid profile in chronic liver disease patients

Parameter	Mean ± SD (mg/dL)	Reference Range (mg/dL)
HDL-C	31.50 ± 19.85	40–60
LDL-C	89.43 ± 19.20	<100
Total Cholesterol	136.44 ± 12.40	<200
Triglycerides	114.07 ± 15.50	<150

Overall, these results indicate that chronic liver disease is associated with a significant reduction in HDL and total cholesterol levels, while LDL and triglyceride levels are less markedly affected.

The correlation analysis [Table 4] between serum lipid parameters and ChildPugh score in patients with chronic liver disease shows interesting patterns. High-density lipoprotein cholesterol (HDL) exhibited a weak negative correlation with Child-Pugh score (Spearman's rho = -0.157, p = 0.020). This indicates that as the severity of liver disease increases, HDL levels tend to decrease slightly. The decrease in HDL can be explained by impaired hepatic synthesis and altered lipid transport that occurs in chronic liver disease, as the liver is the primary site for lipoprotein metabolism.

Table 4: Correlation between serum lipid profile and child pugh score

Lipid Parameter	Spearman Rho	p-value
HDL	-0.157	0.020
LDL	-0.077	0.015
Total Cholesterol	-0.061	0.001
Triglycerides	0.051	0.052

Low-density lipoprotein cholesterol (LDL) also showed a weak negative correlation with Child-Pugh score (rho = -0.077, p = 0.015). Total cholesterol similarly displayed a weak negative correlation with Child-Pugh score (rho = -0.061, p = 0.001), indicating that higher Child-Pugh scores are associated with lower total cholesterol levels. Whereas, triglycerides showed a very weak positive correlation with Child-Pugh score (rho = 0.051, p = 0.052), which was not statistically significant. This suggests that triglyceride levels do not follow a consistent trend with disease severity.

The distribution of patients according to the Child-Pugh class shows that a majority of the patients in this study belong to Class C, accounting for 140 out of 250 patients, which is 56% of the total study population. This has been indicated by [Figure 4].

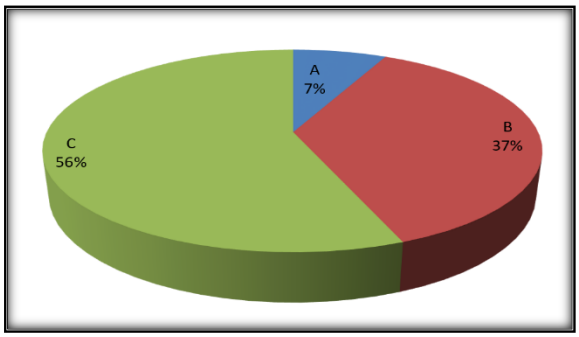

Figure 4: Distribution of Patients According to Child-Pugh Class

This indicates that more than half of the patients had severe chronic liver disease as per the Child-Pugh classification. Class B patients, representing 36.8% (92 patients), had moderate severity of liver disease, while Class A patients, who constitute only 7.2% (18 patients), had mild liver dysfunction.

CONCLUSION

This hospital-based cross-sectional study conducted at Dr. Susheela Tiwari Memorial Government Hospital, Haldwani, highlights the significant alterations in serum lipid profiles among patients with chronic liver disease and their correlation with disease severity as assessed by the Child-Turcotte-Pugh (CTP) score. The study observed a progressive decline in total cholesterol, low-density lipoprotein, and high-density lipoprotein levels with advancing stages of cirrhosis, particularly in patients belonging to Child-Pugh Class C. Triglyceride and very-low-density lipoprotein levels also showed variable reductions in advanced disease, reflecting impaired hepatic synthetic capacity. These findings confirm that lipid profile abnormalities are a consistent feature of chronic liver disease and worsen as hepatic dysfunction advances.

The results emphasize that serum lipid profile estimation, being simple, inexpensive, and widely available, can serve as a useful non-invasive marker for staging chronic liver disease and monitoring disease progression. Its correlation with the CTP score suggests that routine lipid profiling may complement existing prognostic tools in clinical practice. Early identification of dyslipidemia in cirrhotic patients not only aids in assessing liver disease severity but also provides opportunities for timely intervention to prevent associated metabolic and cardiovascular complications.

In conclusion, monitoring serum lipid profiles in chronic liver disease patients has both diagnostic and prognostic value, and integrating such testing into routine clinical evaluation can enhance patient care and long-term outcomes.

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