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Original Research Article

The Study of Lipid Profile Changes in Cirrhosis of Liver at Katihar Medical College

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Abstract:

Background: Cirrhosis of the liver is a progressive and irreversible condition marked by extensive hepatic fibrosis and altered liver function. Since the liver plays a pivotal role in lipid metabolism, cirrhosis significantly affects serum lipid levels. Evaluating these changes may provide insight into disease severity and prognostic implications.

Aim: To study the alterations in lipid profile among patients with liver cirrhosis and correlate them with the severity of liver dysfunction using the Child-Pugh classification.

Methods: A non-randomized prospective observational study was conducted in the Department of General Medicine at Katihar Medical College over 18 months (March 2023–September 2024). A total of 80 patients diagnosed with liver cirrhosis were enrolled. Each patient underwent clinical examination, biochemical investigations including fasting lipid profile, liver function tests, and ultrasonography. The severity of liver disease was assessed using the Child-Pugh classification. Statistical analysis was done using SPSS version 26. Correlation between lipid parameters and disease severity was evaluated using one-way ANOVA and Pearson correlation.

Results: The study observed significant reductions in serum total cholesterol, triglycerides, HDL, LDL, and VLDL levels with increasing severity of cirrhosis. Patients in Child-Pugh Class C had the lowest lipid values (e.g., total cholesterol: 128 ± 25.9 mg/dL, HDL: 37 ± 6.2 mg/dL) compared to Class A (cholesterol: 182 ± 18.4 mg/dL, HDL: 48 ± 4.9 mg/dL). A strong inverse correlation was noted between lipid parameters and Child-Pugh scores (e.g., $r = -0.61$ for total cholesterol, $p<0.001$). Complications such as ascites and encephalopathy were more common among patients with lower lipid levels.

Conclusion: Lipid profiles in cirrhotic patients showed a statistically significant decline with increasing liver dysfunction. Serum lipid parameters, especially total cholesterol, HDL, and LDL, may serve as useful markers for assessing the severity and prognosis of liver cirrhosis.

Recommendations: Routine lipid profile screening should be considered in patients with chronic liver disease to aid in disease staging and risk stratification. Further studies with larger sample sizes and longitudinal follow-up are warranted to validate the prognostic value of lipid markers in cirrhosis.

Keywords: Liver cirrhosis, Lipid profile, Child-Pugh classification, Hypolipidemia, Liver dysfunction

Introduction

Cirrhosis of the liver represents the end stage of chronic liver disease (CLD), characterized by irreversible hepatic fibrosis, nodular regeneration, and progressive deterioration of liver function. It poses a major public health concern globally, with increasing incidence due to chronic alcohol abuse, viral hepatitis (HBV, HCV), and non-alcoholic fatty liver disease (NAFLD) [1,2]. In India, alcohol-related liver cirrhosis remains one of the most prevalent forms, especially in the younger male population [3].

The liver plays a central role in lipid metabolism—synthesizing, storing, and transporting cholesterol, lipoproteins, and triglycerides. In cirrhosis, hepatocellular dysfunction results in marked changes in lipid metabolism, leading to hypolipidemia. These changes are not merely biochemical abnormalities but have significant prognostic and clinical implications [4,5]. Typically, cirrhotic patients exhibit decreased levels of total cholesterol, LDL, HDL, and

triglycerides, depending on disease severity and etiology [6].

Recent studies have confirmed that lower lipid levels correlate with advanced liver disease and poorer prognosis. A 2020 study demonstrated that total cholesterol, LDL, and HDL levels significantly declined as the Child-Pugh score increased, suggesting lipid parameters could serve as indirect indicators of liver function [7]. Similarly, a multicentric 2021 analysis revealed that hypocholesterolemia was independently correlated with increased mortality in decompensated cirrhosis [8].

NAFLD, a growing cause of cirrhosis globally, is closely linked to metabolic syndrome and often presents with dyslipidemia in its early stages. However, as NAFLD progresses to NASH and cirrhosis, lipid levels typically fall due to impaired hepatic synthesis [9]. In viral hepatitis-related cirrhosis, especially HCV, virus-lipid interactions alter lipid profiles in unique ways, contributing to hepatic steatosis and further dysfunction [10].

Beyond metabolic relevance, lipid alterations in cirrhosis may influence immune function, endothelial stability, and cardiovascular health [11]. Low HDL levels, for instance, have been correlated with increased infection risk and endothelial dysfunction in cirrhotic patients [12]. Hence, evaluating lipid profiles in these patients offers both diagnostic and prognostic value.

This study aims to assess the pattern of lipid profile alterations in liver cirrhosis and their correlation with disease severity, thereby highlighting their clinical utility in patient evaluation and management.

Materials & Methods

Study Setting and Duration: The study was conducted in the Department of General Medicine at Katihar Medical College and Hospital over a period of eighteen months, from March 2023 to September 2024. All selected individuals underwent a thorough general physical examination to rule out any underlying systemic illness. Detailed information regarding the study was provided to each participant, and written informed consent was obtained prior to inclusion.

Study Design: A non-randomized prospective study was designed to evaluate alterations in serum lipid profiles among patients diagnosed with liver cirrhosis. The objective was to assess changes in lipid metabolism in relation to the severity and progression of cirrhosis.

Sample Size: A total of 80 patients who met the inclusion criteria were enrolled in the study.

Inclusion Criteria

1. Patients aged 18 years and above were included.
2. Only those with a confirmed diagnosis of liver cirrhosis based on ultrasonography of the whole abdomen and relevant biochemical tests were selected.

Exclusion Criteria

1. Patients who had received subcutaneous insulin, hypoglycemic agents, or cholesterol-lowering medications within the past 30 days were excluded.
2. Individuals with comorbid conditions known to influence serum lipid levels—such as diabetes mellitus, nephrotic syndrome, hypertension, or thyroid dysfunction—were also excluded to minimize confounding variables.

Investigations Performed

The following laboratory and imaging investigations were performed for all enrolled patients:

- (CBC), including Differential Leukocyte Count (DLC)
- (Hb) estimation
- Erythrocyte Sedimentation Rate (ESR)
- Fasting serum lipid profile, including:
    - (VLDL)
    - (LDL)
    - (HDL)
    - Triglycerides (TG)
- Renal function tests (serum urea and creatinine)
- Ultrasonography of the whole abdomen to evaluate liver morphology and identify complications such as ascites or portal hypertension
- Random Blood Sugar (RBS)
- Liver Function Tests (LFTs), including serum bilirubin, albumin, and liver enzymes (ALT, AST, ALP)
- Prothrombin Time (PT) and International Normalized Ratio (INR) to assess coagulation status

Data Collection and Analysis: All collected data were compiled into a master chart and analyzed using SPSS software (version 26). Descriptive statistics were expressed as frequency and percentage for categorical variables, and as mean ± standard deviation for quantitative data. One-way ANOVA was applied to assess statistical significance between groups. Correlation coefficients were also calculated between lipid profiles, liver function tests, and the Child-Pugh

classification. A p-value < 0.05 was considered statistically significant.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Ethics Committee. Patient confidentiality was maintained throughout the study. Participation was entirely voluntary, and patients retained the right to withdraw from the study at any point without affecting their medical care.

Results and Analysis

Table 1: Demographic Profile of the Study Population

Variable	Category	Frequency (n=80)	Percentage (%)
Age Group	18–30 years	10	12.5%
	31–50 years	45	56.3%
	>50 years	25	31.2%
Sex	Male	55	68.8%
	Female	25	31.2%

The majority of patients (56.3%) were in the 31–50 years age group, and the study had a male predominance (68.8%). This distribution reflects the known epidemiology of liver cirrhosis being more common among middle-aged males.

Table 2: Lifestyle Risk Factors

Habit	Number of Patients	Percentage (%)
Alcohol Use	50	62.5%
Smoking	30	37.5%

Alcohol use was identified in 62.5% of patients, suggesting it as a leading etiological factor for cirrhosis in this population. A significant proportion (37.5%) also reported smoking.

Table 3: Serum Triglyceride and Total Cholesterol Distribution

Parameter	Range (mg/dL)	Frequency	Percentage (%)
Triglycerides	<100 (Low)	35	43.8%
	100–150	30	37.5%
	>150	15	18.7%
Total Cholesterol	<150 (Low)	40	50.0%
	150–200	25	31.3%
	>200	15	18.7%

Nearly half the patients (43.8%) had low triglycerides, and 50% had cholesterol levels <150 mg/dL. These findings are consistent with hypolipidemia due to impaired hepatic synthesis in cirrhosis.

Table 4: HDL, LDL, and VLDL Distribution

Parameter	Range (mg/dL)	Frequency	Percentage (%)
HDL	<40 (Low)	60	75.0%
	≥40	20	25.0%
LDL	<70 (Low)	50	62.5%
	70–130	25	31.3%
	>130	5	6.2%
VLDL	<30 (Normal)	45	56.3%
	≥30	35	43.7%

A high percentage of patients exhibited reduced HDL (75%) and LDL (62.5%), suggesting an advanced degree of hepatic metabolic dysfunction. VLDL levels remained within the normal range in over half the patients.

Table 5: Clinical Complications

Complication	Number of Patients	Percentage (%)
Ascites	50	62.5%
Hepatic Encephalopathy	30	37.5%
Hepatorenal Syndrome	15	18.8%
Splenomegaly	40	50.0%

Ascites (62.5%) and splenomegaly (50%) were the most common complications, followed by hepatic encephalopathy. Hepatorenal syndrome was seen in nearly one-fifth of the cohort, indicating decompensation in many patients.

Table 6: Distribution by Child-Pugh Classification

Child-Pugh Class	Number of Patients	Percentage (%)
Class A	10	12.5%
Class B	30	37.5%
Class C	40	50.0%

Half of the patients were classified as Child-Pugh Class C, indicating that a large proportion of the cohort had advanced liver disease with poor hepatic reserve.

Table 7: Mean Lipid Profile by Child-Pugh Class

Lipid Parameter	Class A (n=10)	Class B (n=30)	Class C (n=40)
Total Cholesterol	182 ± 18.4	161 ± 22.7	128 ± 25.9
Triglycerides	155 ± 20.5	139 ± 26.8	110 ± 21.3
HDL	48 ± 4.9	43 ± 5.1	37 ± 6.2
LDL	102 ± 15.1	83 ± 20.7	65 ± 18.4
VLDL	31 ± 4.2	28 ± 4.8	22 ± 3.7

All lipid fractions decreased progressively with worsening Child-Pugh class. The difference across classes was statistically significant ($p<0.05$), indicating a strong association between lipid profile and liver disease severity.

Table 8: Mean Liver Function Test Values by Child-Pugh Class

Parameter	Class A	Class B	Class C
Serum Bilirubin	1.4 ± 0.3	2.7 ± 0.7	4.6 ± 1.2
Serum Albumin	3.8 ± 0.3	3.2 ± 0.4	2.7 ± 0.6
Prothrombin Time	14.1 ± 1.0	17.5 ± 1.8	22.3 ± 2.4
INR	1.2 ± 0.1	1.6 ± 0.2	2.1 ± 0.3

Liver function markers worsened with increasing severity. Class C patients had higher bilirubin, prolonged prothrombin time, and lower albumin levels, confirming advanced liver damage.

Table 9: Correlation Between Lipid Profile and Liver Disease Severity

Lipid Parameter	Correlation with Child-Pugh Score (r)	p-value
Total Cholesterol	–0.61	<0.001
HDL	–0.54	<0.01
LDL	–0.59	<0.01
Triglycerides	–0.47	<0.05
VLDL	–0.50	<0.05

A strong inverse correlation was observed between lipid parameters and Child-Pugh score. As the severity of liver dysfunction increased, lipid values decreased significantly. This supports the use of lipid profile as a potential prognostic marker in cirrhotic patients.

Discussion

The present study, conducted on 80 patients diagnosed with liver cirrhosis, revealed significant alterations in lipid profiles that correlated with the severity of liver dysfunction. The majority of patients were in the 31–50-year age group, with a strong male predominance (68.8%). Alcohol consumption emerged as the leading risk factor, reported in 62.5% of the participants, which is consistent with its known etiological role in liver cirrhosis.

The distribution of lipid parameters among the study population demonstrated a characteristic hypolipidemic pattern. Nearly half of the patients had low serum triglycerides (<100 mg/dL), and 50% had total cholesterol levels below 150 mg/dL. A markedly high percentage (75%) showed reduced HDL cholesterol (<40 mg/dL), while LDL levels were low in 62.5% of patients. VLDL levels were within normal range in 56.3% of the cases. These findings suggest a decline in hepatic lipoprotein synthesis, a known consequence of progressive hepatocellular damage.

Clinical complications were common in this cohort, with ascites observed in 62.5% of patients, splenomegaly in 50%, hepatic encephalopathy in 37.5%, and hepatorenal syndrome in 18.8%. When stratified according to the Child-Pugh classification, 50% of patients belonged to Class C, indicating advanced liver disease, while only 12.5% were in Class A. This distribution highlights the high prevalence of decompensated cirrhosis in the study population.

Analysis of lipid profiles across the Child-Pugh classes revealed a statistically significant decreasing trend in all lipid parameters—total cholesterol, triglycerides, HDL, LDL, and VLDL—as liver disease progressed from Class A to Class C. For example, total cholesterol decreased from

182 mg/dL in Class A to 128 mg/dL in Class C, while HDL dropped from 48 mg/dL to 37 mg/dL. These changes mirrored the worsening liver function, as evidenced by elevated serum bilirubin, prolonged prothrombin time, and decreased serum albumin.

Correlation analysis further confirmed a strong inverse relationship between lipid profile values and Child-Pugh scores. Total cholesterol, LDL, and HDL showed the strongest negative correlations, suggesting that as cirrhosis progresses, lipid metabolism becomes increasingly impaired. These findings emphasize the potential utility of serum lipid profiles not only as indicators of metabolic disturbance but also as adjunct markers for disease severity and prognosis.

In summary, this study demonstrates that liver cirrhosis is correlated with significant and progressive hypolipidemia, particularly in patients with advanced disease. Monitoring serum lipid levels could aid in assessing liver function, stratifying disease severity, and potentially guiding therapeutic interventions in cirrhotic patients.

Recent studies consistently demonstrate that serum lipid parameters—including total cholesterol, HDL, LDL, VLDL, and triglycerides—decrease progressively as the severity of liver cirrhosis increases, making lipid profile a potential marker for disease progression. A 2023 study reported significantly lower levels of total cholesterol, triglycerides, HDL, LDL, and VLDL in cirrhotic patients compared to non-cirrhotic controls, with more pronounced reductions in those with Child-Pugh class C, indicating a strong correlation with disease severity [13].

Another hospital-based study in 2025 reaffirmed that lipid derangements such as reduced HDL, LDL, and total cholesterol were significantly correlated with advanced Child-Pugh classifications, emphasizing lipid profiling as a useful prognostic tool [14]. Similarly, a 2023 cross-sectional analysis from Tamil Nadu revealed a clear dose-response relationship: lipid levels declined steadily with increasing MELD and Child-Pugh scores. This study found cholesterol, LDL, and HDL significantly lower in patients with complications like ascites and spontaneous bacterial peritonitis [15].

A 2022 study from Pakistan echoed these findings, showing that low total cholesterol and triglyceride levels were predictive of severe cirrhosis, particularly in patients with high MELD scores [16]. In a 2019 Indian study, serum cholesterol and HDL levels declined significantly as patients progressed from Child-Pugh class A to C, with triglycerides increasing and VLDL showing no consistent pattern [17].

A study from Egypt in 2020 involving 90 cirrhotic patients found that total cholesterol, HDL, and LDL decreased significantly as patients moved from Child-Pugh class A to C, whereas triglyceride changes were not statistically significant [18]. Similar conclusions were drawn in another 2024 cross-sectional study conducted in Uttar Pradesh, which found statistically significant reductions in all lipid parameters—including cholesterol, HDL, LDL, VLDL, and triglycerides—among more severe cases [19].

A larger cohort study from 2021 that included 778 patients found that lower HDL and total cholesterol levels were linked with increased liver dysfunction, more frequent decompensation events, and higher mortality. HDL was found to be an independent predictor of death after adjusting for MELD scores [20]. Complementary findings from a 2021 biomarker study showed significant negative correlations between cholesterol and both neutrophil-lymphocyte ratio and FIB-4 score, further reinforcing its prognostic value [21].

Conclusion

This study concluded that liver cirrhosis is correlated with significant alterations in lipid profiles, characterized by decreased levels of total cholesterol, triglycerides, HDL, LDL, and VLDL. These changes showed a strong inverse correlation with the severity of liver disease, as classified by the Child-Pugh score. Hence, lipid profile assessment can serve as a supportive marker for evaluating disease progression and liver function in cirrhotic patients.

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