Original Research Paper

A Study On Lipid Profiles In Chronic Liver Diseases

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Abstract

Background: Chronic liver diseases, including cirrhosis, are often associated with alterations in lipid metabolism. This study aimed to evaluate the lipid profiles of patients with cirrhosis and compare them to healthy individuals.

Methods: This case-control study included 25 patients with diagnosed cirrhosis (case group) and 25 age- and sex-matched healthy individuals (control group). Fasting lipid profiles, including total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), were measured in both groups using standard laboratory techniques. Differences in lipid parameters between the two groups were analyzed using appropriate statistical tests.

Results: Patients with cirrhosis exhibited significantly lower levels of total cholesterol (135 ± 30 mg/dL vs. 180 ± 25 mg/dL, $p < 0.001$) and LDL-C (70 ± 25 mg/dL vs. 110 ± 20 mg/dL, $p < 0.001$) compared to the healthy controls. HDL-C levels were also decreased in the cirrhosis group (30 ± 10 mg/dL vs. 45 ± 12 mg/dL, $p < 0.001$). However, triglyceride levels were significantly higher in patients with cirrhosis (180 ± 80 mg/dL vs. 120 ± 50 mg/dL, $p < 0.001$).

Conclusions: Patients with cirrhosis exhibited a distinct lipid profile characterized by decreased levels of total cholesterol, LDL-C, and HDL-C, along with elevated triglyceride levels compared to healthy individuals. These alterations in lipid metabolism may contribute to the pathogenesis and complications associated with chronic liver diseases. Further research is warranted to explore the underlying mechanisms and potential therapeutic implications of these findings.

Keywords: Chronic liver disease, cirrhosis, lipid profile, total cholesterol, triglycerides, HDL-C, LDL-C.

INTRODUCTION:

Chronic liver diseases, particularly cirrhosis, represent a significant global health burden, affecting millions of individuals worldwide.[1] These conditions are characterized by progressive liver damage, leading to impaired hepatic function and various systemic complications.[2] The liver plays a crucial role in lipid metabolism, and consequently, chronic liver diseases often result in alterations of lipid profiles.[3]

The relationship between liver function and lipid metabolism is complex and multifaceted. The liver is responsible for the synthesis, storage, and catabolism of various lipoproteins, including cholesterol and triglycerides.[4] As liver disease progresses, these metabolic processes can become

dysregulated, leading to changes in circulating lipid levels.[5]
Previous studies have reported conflicting results regarding lipid profiles in patients with chronic liver diseases. Some researchers have observed decreased levels of total cholesterol and low-density lipoprotein cholesterol (LDL-C) in cirrhotic patients[6], while others have reported elevated triglyceride levels.[7] The precise nature and extent of these lipid alterations, as well as their clinical significance, remain subjects of ongoing investigation.
Understanding the lipid profile changes in chronic liver diseases is important for several reasons. First, it may provide insights into the pathophysiology of liver disease progression and associated complications.[8] Second, altered lipid profiles may serve as potential biomarkers for disease severity or prognosis.[9] Finally, elucidating these changes could inform therapeutic strategies and nutritional management in patients with chronic liver diseases.[10]
This study aims to evaluate the lipid profiles of patients with cirrhosis and compare them to healthy individuals. By examining total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and LDL-C levels, we seek to contribute to the growing body of knowledge on lipid metabolism in chronic liver diseases and its potential clinical implications.

METHODS:

This case-control study was conducted to evaluate and compare the lipid profiles of patients with cirrhosis to those of healthy individuals. The study included a total of 50 participants, divided equally into two groups: 25 patients with diagnosed cirrhosis (case group) and 25 age- and sex-matched healthy individuals (control group).
Patients for the case group were recruited from the hepatology outpatient clinic and inpatient wards of the hospital. The diagnosis of cirrhosis was established based on a combination of clinical, laboratory, and imaging findings, and in some cases, liver biopsy results. The control group consisted of healthy volunteers with no history of liver disease or other significant medical conditions. Exclusion criteria for both groups included pregnancy, use of lipid-lowering medications, and any acute illness or condition that could affect lipid metabolism.
All participants underwent a comprehensive clinical evaluation, including medical history and physical examination. Demographic data, including age, sex, and body mass index (BMI), were recorded for each participant. For the cirrhosis group, the etiology of liver disease and Child-Pugh score were also documented to assess the severity of liver dysfunction.
Blood samples were collected from all participants after an overnight fast of at least 12 hours. The lipid profile parameters measured included total cholesterol, triglycerides, high- density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). These were analyzed using standard enzymatic methods on an automated biochemistry analyzer. LDL-C was calculated using the Friedewald formula for participants with triglyceride levels below 400 mg/dL.
In addition to the lipid profile, other relevant laboratory tests were performed, including liver function tests (alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, gamma-glutamyl transferase, total bilirubin, and albumin), complete blood count, and prothrombin time. These additional tests were conducted to assess liver function and to exclude other conditions that might affect lipid metabolism.
Statistical analysis was performed using SPSS version 21 software. Continuous variables were expressed as means ± standard deviations, and categorical variables were expressed as frequencies and percentages. The differences in lipid parameters between the cirrhosis group and the control group were analyzed using the independent t-test or Mann- Whitney U test, depending on the distribution of the data. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS:

The study compared 25 patients with cirrhosis to 25 healthy controls, matching them for age, gender, and BMI to ensure comparability between the groups. As shown in Table 1, the cirrhosis

group had a mean age of 55.4 years, with a slight male predominance (60%).
The etiology of cirrhosis was diverse, with alcoholic liver disease being the most common cause (40%), followed by viral hepatitis (32%) and non-alcoholic steatohepatitis (20%). The severity of liver disease was assessed using the Child-Pugh classification, with 40% of patients in Class A, 36% in Class B, and 24% in Class C, representing a range of disease severity within the study population.

Table 1: Demographic and Clinical Characteristics of Study Participants

Characteristic		**Cirrhosis Group (n=25)**	**Control Group (n=25)**	**P value**
Age (years)		55.4±10.2	54.8±9.7	0.83
Gender	**Males**	15 (60%)	15 (60%)	1
	Females	10 (40%)	10 (40%)	
BMI (kg/m^2)		26.3±4.1	25.8±3.7	0.65
Etiology of cirrhosis	**Alcoholic**	10 (40%)	NA	-
	Viral hepatitis	8 (32%)	NA	-
	NASH	5 (20%)	NA	-
	Others	2 (8%)	NA	-
Child-Pugh score	**A**	10 (40%)	NA	-
	B	9 (36%)	NA	-
	C	6 (24%)	NA	-

The lipid profile comparison between the cirrhosis and control groups, presented in Table 2, revealed significant differences across all parameters. Patients with cirrhosis exhibited markedly lower levels of total cholesterol (135 ± 30 mg/dL vs. 180 ± 25 mg/dL, $p < 0.001$), LDL-C (70 ± 25 mg/dL vs. 110 ± 20 mg/dL, $p < 0.001$), and HDL-C (30 ± 10 mg/dL
vs. 45 ± 12 mg/dL, $p < 0.001$) compared to healthy controls. These findings suggest a general reduction in cholesterol synthesis or metabolism in chronic liver disease. In contrast, triglyceride levels were significantly elevated in the cirrhosis group (180 ± 80 mg/dL vs. 120
± 50 mg/dL, $p < 0.001$), potentially indicating impaired triglyceride clearance or altered lipid metabolism in the context of liver dysfunction.

Table 2: Comparison of Lipid Profiles between Cirrhosis and Control Groups

Lipid parameter	**Cirrhosis Group (n=25)**	**Control Group (n=25)**	**P value**
Total Cholesterol (mg/dL)	135±30	180±25	**<0.001**
LDL-C (mg/dL)	70±25	110±20	**<0.001**
HDL-C (mg/dL)	30±10	45±12	**<0.001**
Triglycerides (mg/dL)	180±80	120±50	**<0.001**

Table 3 provides insight into the relationship between lipid parameters and liver function in cirrhosis patients. Total cholesterol, LDL-C, and HDL-C showed positive correlations with serum albumin ($r = 0.52$, 0.48, and 0.45 respectively, all $p < 0.05$) and negative correlations with total bilirubin, prothrombin time, and Child-Pugh score.
This suggests that as liver function deteriorates, indicated by lower albumin levels, higher bilirubin, prolonged prothrombin time, and higher Child-Pugh scores, cholesterol levels tend to decrease. Conversely, triglycerides showed an opposite trend, with a negative correlation with albumin ($r = -0.30$) and positive correlations with bilirubin, prothrombin time, and Child-Pugh score, although not all of these correlations reached statistical significance.

The strongest correlations were observed between lipid parameters and the Child-Pugh score, emphasizing the close relationship between overall liver function and lipid metabolism.

Table 3: Correlation between Lipid Parameters and Liver Function Tests in Cirrhosis Group

Parameter	Total Cholesterol (r)	LDL-C (r)	HDL-C (r)	Triglycerides (r)
Albumin	**0.52***	**0.48***	**0.45***	-0.30
Total Bilirubin	**-0.58***	**-0.50***	**-0.42***	0.35
Prothrombin Time	**-0.45***	**-0.40***	**-0.38***	0.28
Child-Pugh Score	**-0.60***	**-0.55***	**-0.50***	**0.40***

r: Pearson correlation coefficient. *$p<0.05$

DISCUSSION:

The present study demonstrated significant alterations in lipid profiles among patients with cirrhosis compared to healthy controls. Our findings of decreased total cholesterol,
LDL-C, and HDL-C levels, along with elevated triglycerides in cirrhotic patients, are consistent with several previous studies but also highlight some important nuances in lipid metabolism in chronic liver disease.

The observed reduction in total cholesterol and LDL-C levels in our cirrhotic patients aligns with the findings of Ghadir et al.[6], who reported significantly lower total cholesterol and LDL-C in cirrhotic patients compared to controls. This decrease in cholesterol levels is likely due to impaired hepatic synthesis capacity in advanced liver disease. Chrostek et al.[5] similarly found that the severity of liver cirrhosis was inversely correlated with total and LDL cholesterol levels, which is consistent with our observed correlations between lipid parameters and markers of liver function.

However, our finding of significantly lower HDL-C levels in cirrhotic patients contrasts with some earlier studies. For instance, Habib et al.[11] reported no significant difference in HDL-C levels between cirrhotic patients and controls. This discrepancy might be attributed to differences in study populations, cirrhosis etiology, or disease severity. Our results suggest that HDL-C metabolism may be more profoundly affected in our cohort of cirrhotic patients.

The elevated triglyceride levels observed in our cirrhotic patients are consistent with the findings of Mehboob et al.[7], who also reported significantly higher triglycerides in patients with chronic liver disease. This hypertriglyceridemia may be explained by decreased hepatic lipase activity and reduced clearance of triglyceride-rich lipoproteins in cirrhosis, as proposed by Jiang et al.[4]

Our study's demonstration of correlations between lipid parameters and markers of liver function (albumin, bilirubin, prothrombin time, and Child-Pugh score) provides valuable insights into the relationship between disease severity and lipid alterations. These findings are in line with those of Vere et al.[9], who reported that the degree of liver dysfunction correlates with the extent of lipid profile abnormalities in cirrhotic patients.

The clinical implications of these lipid alterations in cirrhosis are still debated. While lower cholesterol levels might intuitively suggest a lower cardiovascular risk, some studies have associated low cholesterol with increased mortality in cirrhosis. Feinleib[12] reported that very low cholesterol levels in cirrhotic patients were associated with a higher risk of death, emphasizing the complex relationship between lipid profiles and outcomes in chronic liver disease.

Furthermore, the elevated triglyceride levels observed in our cirrhotic patients may have implications for the management of these patients. Boemeke et al.[13] suggested that hypertriglyceridemia in cirrhosis might contribute to insulin resistance and hepatic steatosis, potentially exacerbating liver disease progression.

CONCLUSION:

In conclusion, our study confirms and extends previous findings on lipid profile alterations in cirrhosis. The distinct pattern of decreased cholesterol fractions and elevated triglycerides, along with their correlations with liver function parameters, underscores the profound impact of chronic

liver disease on lipid metabolism. These findings highlight the need for careful interpretation of lipid profiles in cirrhotic patients and suggest that these alterations could potentially serve as markers of disease severity or prognostic indicators.

Future research should focus on the longitudinal changes in lipid profiles as liver disease progresses and the potential use of these parameters in risk stratification and management of cirrhotic patients. Additionally, investigating the underlying mechanisms of these lipid alterations could provide new insights into the pathophysiology of chronic liver disease and potentially identify novel therapeutic targets

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