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Research Article

LIPID RATIOS, CLINICAL MANIFESTATIONS AND SEVERITY STRATIFICATION IN ALCOHOL-RELATED LIVER CIRRHOSIS: INSIGHTS FROM A TERTIARY TEACHING HOSPITAL

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Abstract

Background: Alcohol consumption is a leading cause of chronic liver disease worldwide. Chronic alcohol toxicity alters lipid homeostasis, hepatic vascular resistance, and systemic metabolism. This study investigated clinical characteristics, portal hypertensive manifestations, lipid profiles, and the Total Cholesterol to HDL-C ratio (TC/HDL ratio) across disease severity stages in patients with alcohol-related liver cirrhosis.

Methods: A prospective analytical study evaluated 103 cirrhotic patients admitted to Koppal Institute of Medical Sciences (KIMS) Teaching Hospital, Koppal. Clinical signs, ultrasonographic markers, baseline laboratory values, and fasting lipid profiles were documented. Etiology was stratified, and lipid derangements—including the TC/HDL ratio—were analysed relative to alcohol exposure duration, volume, and severity markers (Child-Pugh classification and MELD score).

Results: Alcohol abuse was the primary cause of cirrhosis in 93.2% (n = 96) of patients, who had an average drinking history of 7.8 ± 5.2 years and a mean daily alcohol intake of 156.3 ± 42.7mL. Decompensation features were frequent: ascites was present in 86.4% (n = 89; moderate 43.7%, severe 27.2%), splenomegaly in 79.6% (n = 82), portal hypertension on ultrasonography in 65.0% (n = 67), and abdominal collateral vein dilation in 35.9% (n = 37). Overall Child-Pugh distribution was Class A: 16.5%, Class B: 35.0%, and Class C: 48.5%. Fasting lipid parameters decreased with

advancing Child-Pugh class: Total Cholesterol (164.3 ± 18.2 to 138.4 ± 19.8 mg/dL, $p < 0.001$), HDL-C (52.6 ± 14.3 to 39.4 ± 17.5 mg/dL, $p < 0.001$), and LDL-C (86.4 ± 22.5 to 58.2 ± 27.9 mg/dL, $p < 0.001$). In contrast, the TC/HDL ratio increased significantly with advancing disease severity (3.2± 0.8 in Class A vs. 3.6 ± 1.3 in Class B vs. 4.2 ± 1.7 in Class C, $p < 0.001$). The TC/HDL ratio positively correlated with both Child-Pugh score ($r = 0.41$, $p < 0.001$) and MELD score ($r = 0.36$, $p < 0.001$). Duration of alcohol consumption was an independent positive predictor of MELD score ($\beta = 0.211$, $p = 0.017$). Comparison between alcoholic (n = 96) and non-alcoholic (n = 7) cirrhosis showed slightly lower mean TC (146.8± 22.7 vs. 153.1± 21.4 mg/dL) and LDL-C (67.2 ± 28.6 vs. 71.8 ± 26.3 mg/dL) in the alcoholic group, though differences did not reach statistical significance due to small non-alcoholic sample size.

Conclusion: Advanced alcohol-related cirrhosis is characterized by marked hypolipidemia accompanied by a paradoxical rise in the TC/HDL ratio. This shift reflects a disproportionate loss of functional HDL relative to total cholesterol. Alcohol exposure duration correlates directly with end-stage disease severity, emphasizing the value of monitoring lipid ratios alongside clinical markers of portal hypertension.

Keywords: Alcoholic Liver Disease, Liver Cirrhosis, Total Cholesterol/HDL Ratio, Ascites, Portal Hypertension, Disease Severity, Alcohol Exposure.

INTRODUCTION

Alcohol-related liver disease (ALD) remains a major global public health challenge, accounting for nearly half of all cirrhosis-related mortality worldwide [(1)]. Chronic ethanol consumption induces a spectrum of hepatic pathologies, progressing from steatosis and alcoholic hepatitis to irreversible cirrhosis and hepatocellular carcinoma [(1,2)]. Chronic exposure to alcohol and its toxic metabolite, acetaldehyde, promotes oxidative stress, mitochondrial damage, lipopolysaccharide (LPS)-mediated Kupffer cell activation, and profibrogenic stimulation of hepatic stellate cells [(2)].

The liver is central to lipid biosynthesis, apolipoprotein secretion, and lipoprotein remodelling [(3)]. Chronic heavy alcohol consumption initially stimulates hepatic triglyceride accumulation and VLDL secretion [(2)]. However, as progressive fibrosis replaces functional parenchyma and portal hypertension develops, hepatic synthetic capacity declines [(3)]. This leads to marked alterations in circulating lipid fractions, characterized by decreased Total Cholesterol, LDL-C, and HDL-C [(3)].

While absolute lipid concentrations decline in advanced liver disease, lipoprotein ratios—specifically the Total Cholesterol to High-Density Lipoprotein Cholesterol ratio (TC/HDL ratio)—provide insights into the relative decline among lipid fractions [(3)]. In non-cirrhotic populations, the TC/HDL ratio serves as an established index of cardiovascular and metabolic risk. In cirrhotic patients, however, shifts in this ratio reflect disproportionate derangements in specific lipoprotein classes, particularly functional HDL particles synthesized by the liver [(3,4)].

This study evaluated clinical presentation, ultrasonographic features of portal hypertension, and lipid parameter changes in patients with alcohol-related liver

cirrhosis, with a focus on the TC/HDL ratio across Child-Pugh stages and its relationship with alcohol exposure metrics [3,4].

MATERIALS AND METHODS

Study Population and Design

This prospective analytical study evaluated 103 consecutive patients admitted with liver cirrhosis to the Department of General Medicine at Koppal Institute of Medical Sciences (KIMS) Teaching Hospital, Koppal, India, from July 2023 to January 2024. The study protocol was approved by the Institutional Ethics Committee (DTHK/IEC/2023/076), and informed consent was obtained from all participants.

- **Inclusion Criteria**: Patients aged 18 years or older with clinically, laboratory and radiologically confirmed liver cirrhosis.
- **Exclusion Criteria:** Primary dyslipidemia, diabetes mellitus, systemic hypertension, active cardiovascular or cerebrovascular disease, thyroid pathology, underlying renal failure, chronic pancreatitis, pregnancy, or current use of lipid-modulating therapy, inability to provide informed consent.

Clinical and Alcohol Exposure Assessment

A detailed clinical history was taken, including specific documentation of alcohol use: duration (years), daily quantity (mL/day), and beverage type. Clinical signs of hepatic decompensation (jaundice, ascites, pedal edema, abdominal wall collateral dilation, splenomegaly, and hepatic encephalopathy) were documented. Ascites was clinically graded as mild, moderate, or severe.

Laboratory and Imaging Protocol

Fasting blood samples (12-hour fast) were collected for automated biochemical testing (Beckman Coulter AU680):

1. Standard Laboratory Testing: Complete blood count, liver function tests (bilirubin, albumin, total protein, transaminases, alkaline phosphatase), renal profile, serum electrolytes, and coagulation parameters (PT, INR).
2. Lipid Profile: Total Cholesterol (CHOD-POD method), Triglycerides (GPO-POD method), HDL-C (direct enzymatic method), and LDL-C (calculated via Friedewald formula).
3. Lipid Ratio Determination: Total Cholesterol to HDL-C ratio was calculated.
4. Ultrasonography: Abdominal ultrasound evaluated liver morphologic changes, portal vein diameter, presence of abdominal collaterals, spleen size, and ascites volume.

Severity Stratification

Severity was classified using:

- Child-Pugh Classification: Stratified into Class A (5–6), Class B (7–9), and Class C (10–15) based on bilirubin, albumin, INR, ascites, and encephalopathy.
- MELD Score: Calculated using standard logarithmic parameters (bilirubin, creatinine, INR).

RESULTS

Clinical Presentation and Etiology

Alcohol consumption was identified as the primary etiological factor in 93.2% (n = 96) of patients. The mean duration of alcohol use was 7.8 ± 5.2 years, with an average daily consumption of 156.3 ± 42.7mL. Non-alcoholic etiologies accounted for 6.8% (n = 7; Hepatitis B: n = 3, Cryptogenic: n = 4). The mean age of the cohort was 44.2± 11.8 years, with a male-to-female ratio of 4.15:1 (80.6% male).

Decompensation features were frequent: abdominal distension was present in 87.4% (n = 90), jaundice in 85.4% (n = 88), and a history of recurrent admissions for chronic liver disease in 25.2% (n = 26). Hematemesis or melena was recorded in 2.9% (n = 3). Physical examination revealed icterus in 89.3%, pallor in 83.5%, pedal edema in 73.8%, splenomegaly in 79.6%, and hepatomegaly in 11.7%. (Table 1)

Table 1: Per Abdomen Examination with Ultrasonography Findings

Decompensation / Imaging Features	Patient Count (n = 103)	Percentage (%)
Abdominal Distension	90	87.4%
Ultrasonographic Ascites	89	86.4%
- Mild Ascites	16	15.5%
- Moderate Ascites	45	43.7%
- Severe Ascites	28	27.2%
Splenomegaly	82	79.6%
Portal Hypertension	67	65.0%
Dilated Abdominal Collateral Veins	37	35.9%
Gallbladder Wall Edema	21	20.4%

Child-Pugh distribution assigned 16.5% (n = 17) to Class A, 35.0% (n = 36) to Class B, and 48.5% (n = 50) to Class C. The mean Child-Pugh score was 9.1± 1.6, and the mean MELD score was 16.8 ± 8.4.

Lipid Ratios Across Disease Severity

Total Cholesterol, LDL-C, HDL-C, and Triglycerides decreased progressively from Child-Pugh Class A to Class C. In contrast, the TC/HDL ratio increased significantly with advancing disease severity ($p < 0.001$). (Table 2)

Table 2: Comparison of Lipid Parameters across Child-Pugh Classes

Parameter	Child-Pugh A (n = 17)	Child-Pugh B (n = 36)	Child-Pugh C (n = 50)	p-value
Total Cholesterol (mg/dL)	164.3 ± 18.2	151.7± 21.5	138.4 ± 19.8	< 0.001
HDL-C (mg/dL)	52.6 ± 14.3	45.1 ± 16.8	39.4 ± 17.5	< 0.001
LDL-C (mg/dL)	86.4± 22.5	71.3 ± 26.2	58.2± 27.9	< 0.001
Triglycerides (mg/dL)	98.2 ± 36.4	89.6 ± 43.1	79.7 ± 42.8	0.018
TC / HDL Ratio	3.2 ± 0.8	3.6 ± 1.3	4.2 ± 1.7	< 0.001

Correlation analysis confirmed a significant positive relationship between the TC/HDL ratio and disease severity scores: Child-Pugh score ($r = +0.41$, $p < 0.001$) and MELD score ($r = +0.36$, $p < 0.001$). Multiple linear regression identified the duration of alcohol consumption as an independent positive predictor of MELD score ($\beta=0.211$, $p=0.017$, 95% CI: 0.038 to 0.384)

Comparison between Alcoholic and Non-Alcoholic Etiologies

Comparison of lipid parameters between patients with alcohol-induced cirrhosis (n = 96) and those with non-alcoholic etiologies (n = 7) revealed lower absolute lipid concentrations in the alcoholic group, though differences did not reach statistical significance (Table 3).

Table 3: Comparison of Lipid Parameters between Alcoholic and Non-alcoholic Cirrhosis

Parameter	Alcoholic Cirrhosis (n = 96)	Non-Alcoholic Cirrhosis (n = 7)	p-value
Total Cholesterol (mg/dL)	146.8 ± 22.7	153.1 ± 21.4	0.476
Triglycerides (mg/dL)	86.1 ± 42.6	90.7 ± 40.2	0.779
HDL-C (mg/dL)	43.6 ± 17.3	46.4 ± 16.8	0.679
LDL-C (mg/dL)	67.2 ± 28.6	71.8 ± 26.3	0.681
VLDL-C (mg/dL)	17.2 ± 8.5	18.1 ± 8.0	0.785
TC / HDL Ratio	3.8 ± 1.5	3.5 ±1.3	0.617

DISCUSSION

In this study, alcohol abuse was the primary cause of liver cirrhosis (93.2%), predominantly affecting middle-aged men (mean age 44.2 years). This finding highlights the substantial burden of alcohol-related liver disease among working-age individuals in this region [5].

The clinical profile reflects advanced disease, with 86.4% presenting with ascites, 79.6% with splenomegaly, and 65.0% with ultrasonographic evidence of portal hypertension[5]. Chronic alcohol toxicity drives hepatic parenchymal destruction and fibrotic tissue deposition, which increases intrahepatic vascular resistance [6]. Subsequent splanchnic vasodilation and hyperdynamic circulation aggravate portal pressure, leading to ascites and collateral vein formation [6].

A key finding of this study is the behaviour of the TC/HDL ratio across disease stages. Although absolute levels of both Total Cholesterol and HDL-C declined as liver function worsened, the TC/HDL ratio increased from 3.2 ± 0.8 in Child-Pugh Class A to 4.2 ± 1.7 in Class C ($p < 0.001$). This rise indicates that HDL-C levels drop disproportionately faster than Total Cholesterol as hepatic impairment progresses [5].

This disproportionate loss of HDL-C reflects specific pathophysiological alterations in advanced liver disease:

1. **ApoA-I Synthetic Failure**: ApoA-I, the principal structural protein of HDL, is synthesized almost exclusively by hepatocytes. Advanced parenchymal loss markedly reduces ApoA-I production [7,8].
2. **LCAT Deficiency:** LCAT, the hepatic enzyme responsible for esterifying free cholesterol into mature HDL, shows reduced activity in cirrhosis, impairing HDL particle maturation [7,8].
3. **Pro-Inflammatory Remodelling:** Advanced cirrhosis is characterized by systemic inflammation and endotoxemia, which promote HDL consumption and structural remodelling [8].

These mechanisms lead to a relative depletion of circulating HDL. Because functional HDL mediates reverse cholesterol transport, neutralizes lipopolysaccharides, and protects vascular endothelial function, its disproportionate decline—reflected in the rising TC/HDL ratio—correlates with clinical decompensation and portal hypertensive complications [5,8].

Furthermore, duration of alcohol consumption was an independent predictor of higher MELD scores (beta = +0.211, p = 0.017). This reinforces the direct relationship between cumulative ethanol exposure, sustained hepatic necroinflammation, progressive fibrogenesis, and deteriorating hepatic synthetic reserve [5,6].

While comparisons between alcoholic (n = 96) and non-alcoholic (n = 7) subgroups showed lower mean TC (146.8 vs. 153.1 mg/dL) and LDL-C (67.2 vs. 71.8 mg/dL) in alcohol-induced cases, these differences were not statistically significant due to the small non-alcoholic sample size. However, the trend toward lower lipid concentrations in alcoholic cirrhosis aligns with previous observations by Phukan et al.[9] and Vere et al.[10], who reported that direct ethanol toxicity further suppresses lipoprotein synthesis beyond the impairment caused by chronic liver injury alone.

Clinical Utility and Implications

Monitoring lipid ratios alongside clinical signs of portal hypertension offers practical benefits. Unlike complex functional assays, standard lipid profiles are widely available, objective, and inexpensive. A rising TC/HDL ratio combined with declining total cholesterol provides a clear biochemical signal of deteriorating hepatic synthetic function and advancing disease severity in patients with alcohol-related cirrhosis.

LIMITATIONS

1. **Sample Imbalance**: The high proportion of alcohol-related cases (93.2%) limited the statistical power for subgroup comparisons with non-alcoholic etiologies.
2. **Cross-Sectional Assessment**: Serial lipid profiles over extended follow-up were not measured, preventing evaluation of dynamic changes during alcohol abstinence or medical management.
3. **Specific Assays**: Direct measures of apolipoproteins (ApoA-I, ApoB) and functional HDL subfractions were not performed.

CONCLUSION

Patients with alcohol-related liver cirrhosis present with high rates of decompensation and portal hypertensive complications. Advanced liver disease is characterized by marked hypolipidemia accompanied by a progressive increase in the Total Cholesterol to HDL-C ratio, driven by a disproportionate reduction in HDL-C relative to Total Cholesterol. Duration of

alcohol consumption independently predicts MELD score severity. The TC/HDL ratio serves as an accessible, objective marker of hepatic synthetic impairment and clinical severity in alcohol-related liver cirrhosis.

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