RESEARCH PAPER

APO A1 and LIPID Profile in Patients With Cirrhosis of Liver and Its Correlation With Prognostic Scores: Hospital Based Cross Sectional Study

Dr. Ashokavardhana S[1], Dr. Raksha Salian[2], Dr. Yashwanth N S[3], Dr. Ashok M L[4]

[1]*Assistant Professor, Dept Of General Medicine, Bangalore medical college and research institute.*
E mail - ashokavs1995@gmail.com
[2]*Senior Resident, Dept of General Medicine, Father Muller Medical college, Mangalore, Karnataka*
[3]*Senior Resident, Department of general medicine, Sri Siddhartha Medical College, Tumkur, Karnataka.*
E - mail yashwanth2302@gmail.com
[4]*Professor, Department of general medicine, Bangalore medical college and research institute.*

***Corresponding author:** Dr. Raksha Salian Senior Resident,*
Dept of General Medicine,
Father Muller Medical college, Mangalore, Karnataka

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ABSTRACT

Background: Liver cirrhosis causes progressive impairment of hepatic synthetic function and produces substantial alterations in lipid and lipoprotein metabolism. Apolipoprotein A1 (Apo A1), the principal protein component of high-density lipoprotein, may decrease with worsening hepatic dysfunction and could serve as a supplementary marker of disease severity.

Aim and Objectives: To estimate serum Apo A1 and fasting lipid-profile parameters in patients with liver cirrhosis, calculate Child–Pugh and Model for End-Stage Liver Disease–Sodium (MELD-Na) scores, and determine the correlation of Apo A1 and lipid parameters with these prognostic measures.

Materials and Methods: This hospital-based cross-sectional study included 80 patients with liver cirrhosis attending or admitted to hospitals attached to Bangalore Medical College and Research Institute between August 2022 and January 2024. Clinical assessment and relevant laboratory investigations, including fasting lipid profile and Apo A1, were performed. Child–Pugh classification and MELD-Na scores were determined, and their relationships with Apo A1 and lipid parameters were analysed.

Results: The mean age was 44.53 ± 10.51 years, and 82.5% were males. Alcohol was the predominant aetiology (82.5%). Child–Pugh classes A, B and C comprised 1.3%, 23.8% and 75.0% of patients, respectively. The mean MELD-Na score was 24.28 ± 8.15. Mean total cholesterol, triglycerides, LDL, VLDL and HDL were 98.09 ± 42.13, 116.71 ± 43.81, 55.03 ± 29.39, 23.86 ± 9.28 and 17.54 ± 10.28 mg/dL, respectively. Mean Apo A1 was
48.22 ± 31.49 mg/dL among 79 patients. Apo A1 differed significantly across Child–Pugh classes (p=0.013). All lipid parameters and Apo A1 differed significantly across MELD-Na categories (p=0.001 each). Apo A1 demonstrated the strongest inverse correlation with MELD-Na score (, p<0.001).

Conclusion: Apo A1 and lipid-profile parameters declined with increasing severity of liver cirrhosis. Apo A1 showed a particularly strong inverse relationship with MELD-Na score and may serve as a useful supplementary biochemical indicator of hepatic dysfunction when interpreted alongside established prognostic scores.

Keywords: Apolipoprotein A1; liver cirrhosis; lipid profile; Child–Pugh class; MELD-Na score; hepatic dysfunction; prognosis; cholesterol; high-density lipoprotein; chronic liver disease.

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INTRODUCTION

Liver cirrhosis is the advanced stage of progressive hepatic fibrosis and is characterized by distortion of normal liver architecture, formation of regenerative nodules, and impairment of hepatic function. It represents a major cause of morbidity and mortality worldwide and may result from chronic alcohol consumption, viral hepatitis, metabolic dysfunction-associated steatotic liver disease, autoimmune disorders, and other chronic hepatic conditions (1). As cirrhosis progresses, patients may develop portal hypertension, ascites, variceal bleeding, hepatic encephalopathy, spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatocellular carcinoma. Accurate assessment of disease severity is therefore essential for prognostication, treatment
planning, and prioritization for liver transplantation (2).

**Author for Correspondence: principal@icon.ac.in*

The liver has a central role in lipid and lipoprotein metabolism. It is responsible for the synthesis, secretion, transformation, and clearance of cholesterol, triglycerides, phospholipids, apolipoproteins, and plasma lipoproteins. Structural and functional deterioration of the liver in cirrhosis may consequently produce substantial alterations in serum lipid concentrations (3). Reduced hepatic synthesis, impaired cholesterol esterification, altered lipoprotein assembly, malnutrition, and changes in biliary excretion may contribute to decreased levels of total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol. These abnormalities may become more pronounced with advancing hepatic dysfunction and could therefore reflect the functional reserve of the liver (4).

Apolipoprotein A1 (Apo A1) is the principal protein component of high-density lipoprotein and is synthesized predominantly in the liver and intestine. It has an important role in reverse cholesterol transport by facilitating the removal of excess cholesterol from peripheral tissues and its transport to the liver for metabolism and excretion (5). Apo A1 also possesses antioxidant, anti-inflammatory, and endothelial-protective properties. Because hepatic synthesis contributes substantially to circulating Apo A1, its concentration may decline when synthetic liver function deteriorates. Apo A1 may therefore serve as a potentially useful biochemical marker of hepatic dysfunction and disease severity in patients with cirrhosis (6,7).

The Child–Pugh score and Model for End-Stage Liver Disease–Sodium (MELD-Na) score are commonly used for prognostic assessment in cirrhosis. The Child–Pugh score incorporates serum bilirubin, serum albumin, prothrombin time or international normalized ratio, ascites, and hepatic encephalopathy to classify disease severity (8,9). The MELD-Na score uses objective laboratory parameters, including bilirubin, creatinine, international normalized ratio, and serum sodium, to estimate short-term mortality risk. Although these scoring systems are clinically valuable, investigating additional biochemical markers may improve understanding of the metabolic consequences of cirrhosis and provide supportive information regarding prognosis (10,11).

Assessment of Apo A1 together with the conventional fasting lipid profile may offer a practical and accessible means of evaluating altered lipid metabolism in cirrhosis. However, the relationship of these parameters with established prognostic scores requires further evaluation in the local clinical setting (12,13). The present hospital-based cross-sectional study was therefore undertaken to estimate Apo A1 and lipid profile levels, calculate Child–Pugh and MELD-Na scores, and determine their correlations in patients with cirrhosis of the liver. The findings may clarify whether declining Apo A1 and lipid levels correspond with increasing disease severity and may support their use as supplementary indicators for clinical assessment and prognostic stratification.

MATERIALS AND METHODS

This hospital-based cross-sectional study was conducted from August 2022 to January 2024 among patients with cirrhosis of the liver attending the outpatient department or admitted to the Department of General Medicine at hospitals attached to Bangalore Medical College and Research Institute (BMCRI). After obtaining approval from the Institutional Ethics Committee, eligible patients were enrolled following written informed consent. Patients aged 18–65 years who were willing to participate were included. Patients aged below 18 years, those who refused consent, and those with a previous history of dyslipidaemia, use of lipid-lowering medications, type 2 diabetes mellitus, hypertension, chronic kidney disease, hypothyroidism or hyperthyroidism, or pancreatitis were excluded.

A detailed clinical history was obtained, and a comprehensive physical examination was performed. Relevant demographic, clinical, and laboratory data were documented using a structured case record form. Investigations included complete blood count, renal function tests, liver function tests, prothrombin time–international normalized ratio, relevant serological tests, fasting lipid profile, serum apolipoprotein A1 levels, and abdominal ultrasonography. The diagnosis and aetiology of liver cirrhosis were determined using the clinical, biochemical, serological, and ultrasonographic findings. The severity and prognosis of cirrhosis were assessed using the Child–Pugh and Model for End-Stage Liver Disease–Sodium scores. Serum Apo A1 and fasting lipid profile parameters were correlated with these prognostic scores. Based on a previous study by Ahmed F. Gomaa et al., the sample size was calculated using the cross-sectional study formula , considering an expected proportion of 40%, absolute precision of 11%, and a 95% confidence level. The calculated sample size was 76, which was rounded to 80 participants.

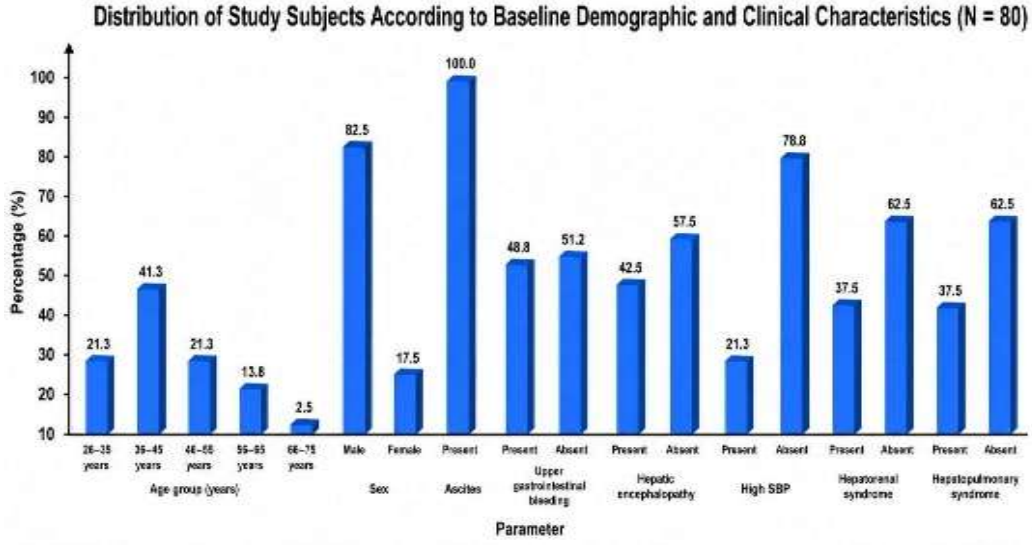
RESULTS

Table 1. Distribution of Study Subjects According to Baseline Demographic and Clinical Characteristics (N = 80)

Characteristic	Category	n	Percentage
Age group	26–35 years	17	21.3

	36-45 years	33	41.3
	46-55 years	17	21.3
	56-65 years	11	13.8
	66-75 years	2	2.5
Sex	Male	66	82.5
	Female	14	17.5
Ascites	Present	80	100.0
Upper gastrointestinal bleeding	Present	39	48.8
	Absent	41	51.2
Hepatic encephalopathy	Present	34	42.5
	Absent	46	57.5
High SBP	Present	17	21.3
	Absent	63	78.8
Hepatorenal syndrome	Present	30	37.5
	Absent	50	62.5
Hepatopulmonary syndrome	Present	30	37.5
	Absent	50	62.5

Graph 1: Distribution of Study Subjects According to Baseline Demographic and Clinical Characteristics (N = 80)

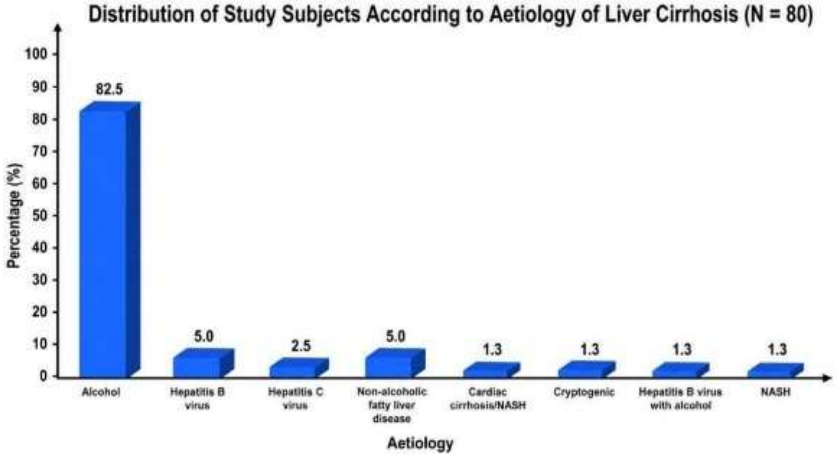


In the present study, a total of 80 patients with liver cirrhosis were included. The most common age group was 36–45 years, comprising 33 (41.3%) patients, followed by 26–35 years and 46–55 years, each with 17 (21.3%) patients. 11 (13.8%) patients belonged to the 56–65 years age group, while 2 (2.5%) patients were in the 66–75 years age group.
With respect to sex distribution, 66 (82.5%) patients were male and 14 (17.5%) were female.
Ascites was present in all 80 (100.0%) patients.

Table 2. Distribution of Study Subjects According to Aetiology of Liver Cirrhosis (N = 80)

Aetiology	n	Percentage
Alcohol	66	82.5
Hepatitis B virus	4	5.0
Hepatitis C virus	2	2.5
Non-alcoholic fatty liver disease	4	5.0
Cardiac cirrhosis/NASH	1	1.3
Cryptogenic	1	1.3
Hepatitis B virus with alcohol	1	1.3
NASH	1	1.3
Total	**80**	**100.0**

Graph 2: Distribution of Study Subjects According to Aetiology of Liver Cirrhosis (N = 80)

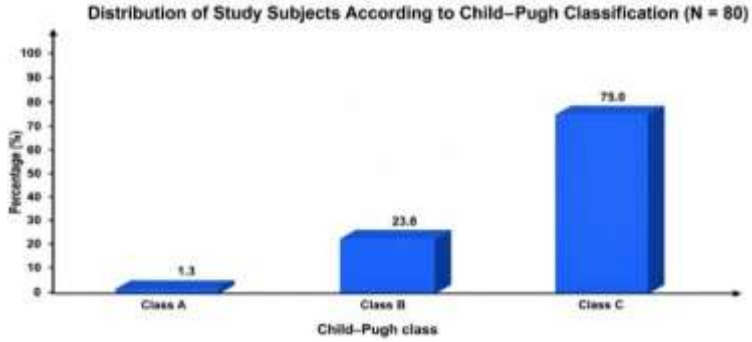


In the present study, alcohol was the most common aetiology of liver cirrhosis, accounting for 66 (82.5%) patients. Hepatitis B virus and non-alcoholic fatty liver disease were each observed in 4 (5.0%) patients, while hepatitis C virus was present in 2 (2.5%) patients. Cardiac cirrhosis/NASH, cryptogenic cirrhosis, hepatitis B virus with alcohol, and NASH were each identified in 1 (1.3%) patient. Overall, the study included 80 (100.0%) patients.

Table 3. Distribution of Study Subjects According to Child–Pugh Classification (N = 80)

Child–Pugh class	n	Percentage
Class A	1	1.3
Class B	19	23.8
Class C	60	75.0
Total	**80**	**100.0**

Graph 3: Distribution of Study Subjects According to Child–Pugh Classification (N = 80)

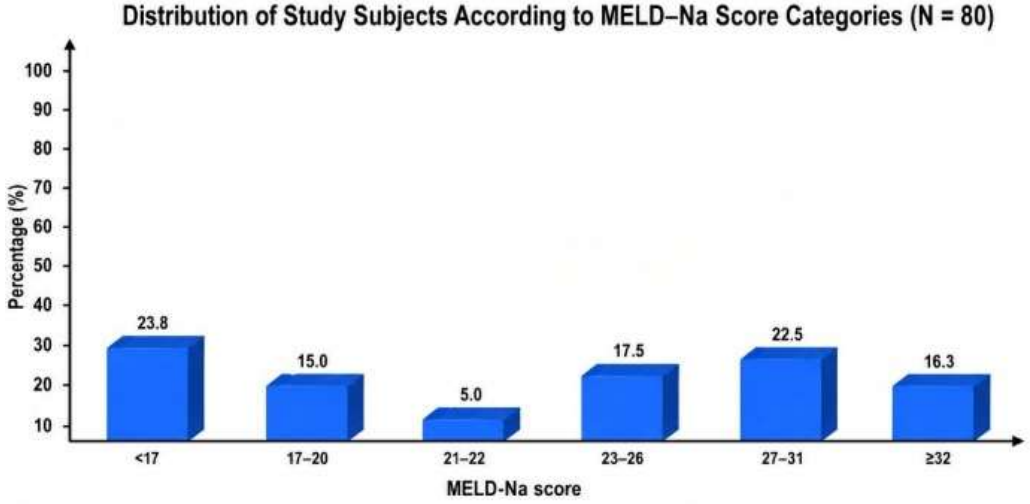


In the present study, according to the Child–Pugh classification, 1 (1.3%) patient belonged to Class A, 19 (23.8%) patients belonged to Class B, and 60 (75.0%) patients belonged to Class C. Overall, the study included 80 (100.0%) patients.

Table 4. Distribution of Study Subjects According to MELD–Na Score Categories (N = 80)

MELD-Na score	n	Percentage
<17	19	23.8
17–20	12	15.0
21–22	4	5.0
23–26	14	17.5
27–31	18	22.5
≥32	13	16.3
Total	**80**	**100.0**

Graph 4: Distribution of Study Subjects According to MELD–Na Score Categories (N = 80) Distribution of Study Subjects According to MELD–Na Score Categories (N = 80)



In the present study, according to the MELD–Na score, 19 (23.8%) patients had a score of <17, 12 (15.0%) patients had a score of 17–20, 4 (5.0%) patients had a score of 21–22, 14 (17.5%) patients had a score of 23–26, 18 (22.5%) patients had a score of 27–31, and 13 (16.3%) patients had a score of ≥32. Overall, the study included 80 (100.0%) patients.

Table 5. Descriptive Statistics of Serum Apo A1 and Fasting Lipid Profile Parameters Among Study Subjects (N = 80)

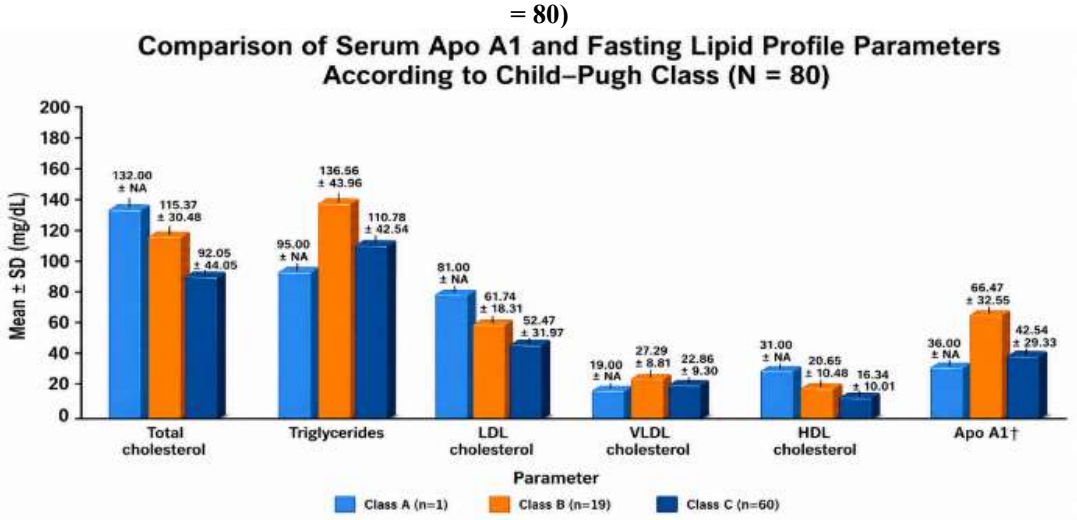
Parameter	n	Minimum	Maximum	Mean	SD
Total cholesterol	80	21.0	236.0	98.09	42.13
Triglycerides	80	37.0	232.0	116.71	43.81
LDL cholesterol	80	4.0	144.0	55.03	29.39
VLDL cholesterol	80	7.5	46.0	23.86	9.28
HDL cholesterol	80	5.0	61.0	17.54	10.28
Apo A1	79	8.0	179.0	48.22	31.49

In the present study, total cholesterol ranged from 21.0–236.0 mg/dL (mean 98.09 ± 42.13 mg/dL), triglycerides from 37.0–232.0 mg/dL (mean 116.71 ± 43.81 mg/dL), LDL cholesterol from 4.0–144.0 mg/dL (mean 55.03 ± 29.39 mg/dL), VLDL cholesterol from 7.5–46.0 mg/dL (mean 23.86 ± 9.28 mg/dL), and HDL cholesterol from 5.0–61.0 mg/dL (mean 17.54 ± 10.28 mg/dL), all estimated in 80 patients. Serum Apo A1 was estimated in 79 patients and ranged from 8.0–179.0 mg/dL, with a mean of 48.22 ± 31.49 mg/dL.

Table 6. Comparison of Serum Apo A1 and Fasting Lipid Profile Parameters According to Child–Pugh Class (N = 80)

Parameter	Class A Mean ± SD (n=1)	Class B Mean ± SD (n=19)	Class C Mean ± SD (n=60)	p-value
Total cholesterol	132.00 ± NA	115.37 ± 30.48	92.05 ± 44.05	0.078
Triglycerides	95.00 ± NA	136.56 ± 43.96	110.78 ± 42.54	0.071
LDL cholesterol	81.00 ± NA	61.74 ± 18.31	52.47 ± 31.97	0.333
VLDL cholesterol	19.00 ± NA	27.29 ± 8.81	22.86 ± 9.30	0.169
HDL cholesterol	31.00 ± NA	20.65 ± 10.48	16.34 ± 10.01	0.117
Apo A1†	36.00 ± NA	66.47 ± 32.55	42.54 ± 29.33	**0.013***

Graph 5. Comparison of Serum Apo A1 and Fasting Lipid Profile Parameters According to Child–Pugh Class (N = 80)



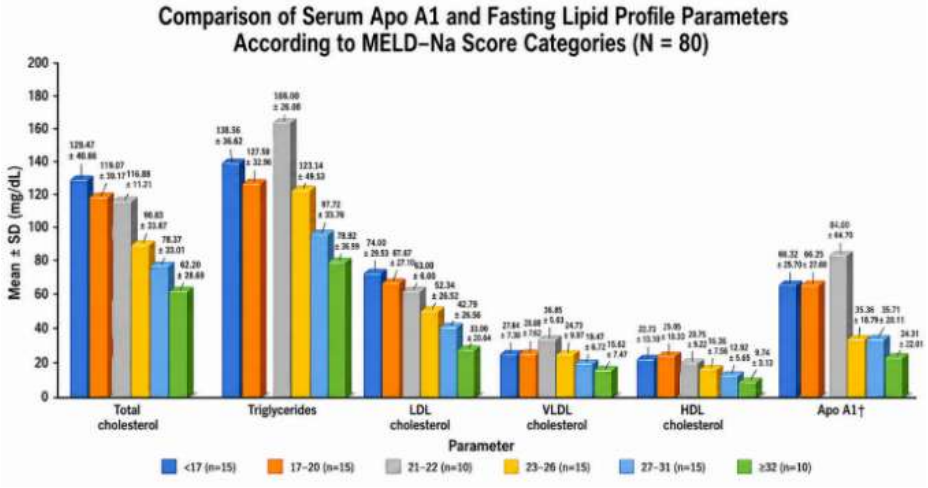
In the present study, the mean total cholesterol was 132.00 ± NA mg/dL in Child–Pugh Class A (n=1), 115.37 ± 30.48 mg/dL in Class B (n=19), and 92.05 ± 44.05 mg/dL in Class C (n=60), with no significant difference (p=0.078). Similarly, the mean triglyceride levels were 95.00 ± NA, 136.56 ± 43.96, and 110.78 ± 42.54 mg/dL (p=0.071); LDL cholesterol levels were 81.00 ± NA, 61.74 ± 18.31, and 52.47 ± 31.97 mg/dL (p=0.333); VLDL cholesterol levels were 19.00 ± NA, 27.29 ± 8.81, and 22.86 ± 9.30 mg/dL (p=0.169); and HDL cholesterol levels were 31.00 ± NA, 20.65 ± 10.48, and 16.34 ± 10.01 mg/dL (p=0.117) in Classes A, B, and C, respectively, with no significant differences. However, the mean Apo A1 level was 36.00 ± NA mg/dL in Class A, 66.47 ± 32.55 mg/dL in Class B, and 42.54 ± 29.33 mg/dL in Class C, showing a statistically significant difference across the Child–Pugh classes (p=0.013).

Table 7. Comparison of Serum Apo A1 and Fasting Lipid Profile Parameters According to MELD–Na Score Categories (N = 80)

Parameter	<17	17–20	21–22	23–26	27–31	≥32	p-value
Total cholesterol	129.47 ± 40.66	119.07 ± 39.17	116.88 ± 11.21	90.83 ± 33.87	78.37 ± 33.01	62.20 ± 28.69	**0.001***
Triglycerides	138.56 ± 36.62	127.58 ± 32.96	166.00 ± 26.08	123.14 ± 49.53	97.72 ± 33.76	78.92 ± 36.99	**0.001***
LDL cholesterol	74.00 ± 29.53	67.67 ± 27.10	63.00 ± 6.00	52.34 ± 26.52	42.79 ± 26.56	33.00 ± 20.64	**0.001***
VLDL cholesterol	27.64 ± 7.30	28.08 ± 7.62	36.85 ± 5.03	24.73 ± 9.97	19.47 ± 6.72	15.62 ± 7.47	**0.001***
HDL cholesterol	22.73 ± 13.10	25.05 ± 10.33	20.75 ± 9.22	16.36 ± 7.56	12.92 ± 5.65	9.74 ± 3.13	**0.001***

Apo A1†	66.32 ± 25.70	66.25 ± 27.60	84.00 ± 64.70	35.36 ± 18.79	35.71 ± 20.11	24.31 ± 22.01	**0.001***

Graph 6. Comparison of Serum Apo A1 and Fasting Lipid Profile Parameters According to MELD–Na Score Categories (N = 80)



In the present study, the mean total cholesterol was 129.47 ± 40.66 mg/dL in patients with MELD–Na score <17, 119.07 ± 39.17 mg/dL in those with 17–20, 116.88 ± 11.21 mg/dL in those with 21–22, 90.83 ± 33.87 mg/dL in those with 23–26, 78.37 ± 33.01 mg/dL in those with 27–31, and 62.20 ± 28.69 mg/dL in those with ≥32, showing a statistically significant difference (p=0.001). Similarly, the mean triglyceride levels were 138.56 ± 36.62, 127.58 ± 32.96, 166.00 ± 26.08, 123.14 ± 49.53, 97.72 ± 33.76, and 78.92 ± 36.99 mg/dL (p=0.001); LDL cholesterol levels were 74.00 ± 29.53, 67.67 ± 27.10, 63.00 ± 6.00, 52.34 ± 26.52, 42.79 ± 26.56, and 33.00 ± 20.64 mg/dL (p=0.001); VLDL cholesterol levels were 27.64 ± 7.30, 28.08 ± 7.62, 36.85 ± 5.03, 24.73 ± 9.97, 19.47 ± 6.72, and 15.62 ± 7.47 mg/dL (p=0.001); HDL cholesterol levels were 22.73 ± 13.10, 25.05 ± 10.33, 20.75 ± 9.22, 16.36 ± 7.56, 12.92 ± 5.65, and 9.74 ± 3.13 mg/dL (p=0.001); and Apo A1 levels were 66.32 ± 25.70, 66.25 ± 27.60, 84.00 ± 64.70, 35.36 ± 18.79, 35.71 ± 20.11, and 24.31 ± 22.01 mg/dL across increasing MELD–Na score categories, respectively, all demonstrating statistically significant differences (p=0.001).

DISCUSSION

The present hospital-based cross-sectional study evaluated Apo A1, fasting lipid profile, and their relationship with established prognostic scores among 80 patients with liver cirrhosis. The mean age was 44.53 ± 10.51 years, and 41.3% were aged 36–45 years. Males constituted 82.5% of participants. Ascites was present in all patients, while upper gastrointestinal bleeding, hepatic encephalopathy, hepatorenal syndrome, and hepatopulmonary syndrome were observed in 48.8%, 42.5%, 37.5%, and 37.5%, respectively. These findings indicate that decompensated cirrhosis and its complications were common in the study population.

Alcohol was the predominant aetiology, accounting for 82.5% of cases. Hepatitis B infection and non-alcoholic fatty liver disease each accounted for 5.0%, whereas hepatitis C infection accounted for 2.5%. Other causes were individually observed in 1.3% of patients. The predominance of alcohol-associated cirrhosis may partly explain the marked male preponderance.

Most participants had advanced hepatic dysfunction. Child–Pugh class C constituted 75.0% of cases, followed by class B in 23.8% and class A in 1.3%. The mean MELD-Na score was 24.28 ± 8.15, ranging from 11 to 48, while 38.8% had MELD-Na scores of 27 or higher. Ghadir et al. (2010) similarly demonstrated progressive lipid abnormalities with increasing Child–Pugh severity (14). Bassani et al. (2015) also reported lower lipid concentrations among patients with advanced cirrhosis (15).

The mean total cholesterol, triglyceride, LDL, VLDL, and HDL levels were 98.09 ± 42.13, 116.71 ± 43.81, 55.03 ± 29.39, 23.86 ± 9.28, and 17.54 ± 10.28 mg/dL, respectively. Mean Apo A1 was 48.22 ± 31.49 mg/dL among 79 patients. Poynard et al. (1986) also reported decreased Apo A1 levels in alcoholic liver disease and related these reductions to disease severity (16). These findings may reflect impaired hepatic synthesis and altered lipoprotein metabolism.

Apo A1 differed significantly across Child–Pugh classes (p=0.013), whereas differences in conventional lipid parameters were not statistically significant. This finding should be interpreted cautiously because only one patient belonged to class A.

All lipid parameters and Apo A1 differed significantly across MELD-Na categories (p=0.001 each). From MELD-Na <17 to ≥32, total cholesterol decreased from 129.47 to 62.20 mg/dL, LDL from 74.00 to 33.00 mg/dL,

HDL from 22.73 to 9.74 mg/dL, and Apo A1 from 66.32 to 24.31 mg/dL. Yamuna et al. (2023) similarly reported declining lipid levels with worsening cirrhosis (17).
Total cholesterol, triglycerides, HDL, and Apo A1 correlated negatively with advancing Child–Pugh class, while LDL and VLDL showed nonsignificant negative correlations. All parameters were significantly inversely correlated with MELD-Na. Apo A1 showed the strongest inverse correlation (, $p<0.001$), supporting its potential role as a supplementary biochemical indicator of cirrhosis severity.

CONCLUSION

The present hospital-based cross-sectional study demonstrated that Apo A1 and lipid-profile parameters were substantially altered in patients with liver cirrhosis. Most participants had advanced disease, with 75% classified as Child–Pugh class C and a mean MELD-Na score of 24.28 ± 8.15. Apo A1 levels differed significantly across Child–Pugh classes, while conventional lipid parameters showed no statistically significant differences. However, total cholesterol, triglycerides, LDL, VLDL, HDL, and Apo A1 differed significantly across MELD-Na categories and generally decreased with increasing disease severity. Apo A1, total cholesterol, triglycerides, and HDL showed significant inverse correlations with advancing Child–Pugh class. All evaluated biochemical parameters were significantly inversely correlated with MELD-Na score, with Apo A1 demonstrating the strongest correlation. These findings suggest that reduced Apo A1 and lipid levels are associated with worsening hepatic dysfunction. Apo A1 may therefore serve as a useful supplementary biochemical indicator for assessing disease severity alongside established prognostic scoring systems in patients with liver cirrhosis.

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