Original Research Article

Lipid profile in assessing the severity of cirrhosis

C. Suman[1*], B. Ramesh Kumar[2], B. Prabhakar[3]

[1]Consultant Gastroenterologist, Apollo Hospitals, Jubilee Hills, Hyderabad, India
[2]Associate Professor, Department of Gastroenterology, Osmania Medical College, Hyderabad, India
[3]Professor, Department of Gastroenterology, Osmania Medical College, Hyderabad, India
[*]Corresponding author email: **sumanandyou@gmail.com**

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Abstract

Background: Cirrhosis is a common hepatological disorder seen in clinical practice. Liver plays an essential role in lipid metabolism, several stages of lipid synthesis and transportation. It is reasonable to expect an abnormal lipid profile in those with severe liver dysfunction

Aim: To assess the lipid profile abnormalities in patients with cirrhosis of liver correlate with the severity of cirrhosis and whether lipid profile can be used to predict severe cirrhosis of liver.

Materials and methods: This is a cross sectional study performed for a period of 2 years. Age and sex matched controls were selected from the patients attending Out Patient Department of Gastroenterology. 150 patients with cirrhosis of liver were included in the study. Diagnosis of cirrhosis of liver was based on characteristic findings.

Results: Mean serum total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol and serum triglycerides were significantly ($p < 0.05$) lower in patients with cirrhosis of liver when compared to healthy controls. Serum lipids progressively decreased with increasing severity of cirrhosis as assessed by Child criteria and MELD score. There was no statistically significant difference in serum total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol and triglycerides among alcoholic and non alcoholic cirrhotic patients with similar severity of cirrhosis. Among the various parameters of lipid profile, total cholesterol and LDL cholesterol had the highest areas under the roc curve; hence may be considered as the best predictors of Child C cirrhosis and MELD > 24 respectively.

Conclusion: Among the various parameters of lipid profile, total cholesterol and LDL cholesterol have the highest areas under the roc curve; hence may be considered as the best predictors of Child C cirrhosis and MELD > 24 respectively.

Key words

Cirrhosis, Model for End Stage Liver Disease (MELD), Child pough classification.

Introduction

Cirrhosis is a common hepatological disorder seen in clinical practice. Cirrhosis is a pathologically defined entity that is associated with a spectrum of characteristic clinical manifestations. It is the histologic end point of varied chronic insults resulting in necrosis of the cell followed by fibrosis and nodular regeneration with gross distortion in liver architecture. Cirrhosis is defined by the World Health Organization (WHO) as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules [1]. This peculiar transformation of the liver was identified by the first anatomic pathologist, Gianbattista Morgagni in his 500 autopsies published in 1761 but the name of "cirrhosis" (greek=orange color) was given by Laennec in 1826 because of the yellowish-tan color of the cirrhotic liver [2]. Only in 1930, one hundred years later, however, the first theory as to the pathogenesis of this disorder was advanced by Roessle: parenchymal degeneration, regeneration and scarring. Cirrhosis affects hundreds of millions of patients worldwide. The overall burden of liver disease in the United States – the vast majority of which is due to chronic disease with fibrosis – continues to expand, exacting an increasing economic and social cost [3]. Chronic liver diseases and cirrhosis result in 26,000-35,000 deaths each year in the United States. Cirrhosis is the 12thleading cause of death in the US and is responsible for 1.3% of all US deaths [4]. The clinical manifestations of cirrhosis vary widely, from no symptoms at all to liver failure, and are determined by both the nature and severity of the underlying liver disease as well as the extent of hepatic fibrosis [5]. Cirrhotic patients need frequent visits and multiple hospitalizations for management of cirrhosis or its complications. However, choosing the proper treatment plan depends on the severity, type of liver damage and possibility of assessing its extent. To evaluate the severity of cirrhosis, Child-Turcotte-Pough (CTP) criteria can be used [6]. In addition, Model for End Stage Liver Disease (MELD) criteria is used to choose liver transplantation candidates which are substituted by Pediatric End Stage Liver Disease Model (PELD) criteria for children less than 12 years [7]. With continuous emergence of new treatments for liver disease in recent years, the evaluation system used for the severity and prognosis of patients with decompensated cirrhosis is needed urgently. Lipids are essential component of biological membranes, free molecules and metabolic regulators that control cellular function and homeostasis [8]. Liver plays an essential role in lipid metabolism, several stages of lipid synthesis and transportation. Endogenous lipids, lipoproteins and apolipoproteins are largely produced in the liver. More than 80% of endogenous cholesterol is synthesised in the hepatocellular microsomes, therefore, it is reasonable to expect an abnormal lipid profile in those with severe liver dysfunction [9]. Although, several studies have been performed on lipoprotein profile alterations in those with liver disease, contributions from India are scarce. Due to the high prevalence of cirrhosis in our country, the present study was conducted to determine lipid profile in patients with cirrhosis and to assess if it relates to the severity of cirrhosis.

Materials and methods

This is a cross sectional study performed from December 2011 to November 2013 at Department of Gastroenterology, Osmania General Hospital. This study was approved by the ethical committee of the hospital. Age and sex matched controls were selected from the patients attending Out Patient Department of Gastroenterology. Informed consent is taken from all the subjects included in the study.

Inclusion criteria were patients with cirrhosis of liver and age of more than 18 years.

Exclusion criteria were age of less than 18 years, diabetes mellitus, hypertension, cardiovascular disease, cerebrovascular disease, chronic kidney disease, use of lipid regulating drugs, acute pancreatitis, hypothyroidism/ hyperthyroidism, smoking, biliary cirrhosis (Primary and secondary), Primary sclerosing cholangitis and other cholestatic liver diseases.

The diagnosis of cirrhosis of liver was based on characteristic findings including physical stigmata of cirrhosis, liver function tests, prothrombin time, ultrasonographic findings (like nodular liver surface, coarse echotexture of liver parenchyma, splenomegaly etc.), upper gastrointestinal endoscopy findings (varices and portal hypertensive gastropathy) and liver biopsy where required. 150 patients with cirrhosis of liver were included in the study. All patients with cirrhosis were considered for detailed medical history, complete physical examination, standard laboratory tests including complete blood picture, biochemical tests of liver and kidney function, serum electrolytes, thyroid profile, fasting and postprandial blood sugar, ECG, chest X ray, ultrasound of the abdomen and ascitic fluid analysis, upper GI endoscopy. For assessment of severity, cirrhotic patients were divided into A, B and C classes according to Child Pugh criteria. They were also divided into 4 MELD groups (as per UNOS[80]) as follows: Group I: MELD ≤ 10, Group II: MELD: 11 – 18, Group III: MELD: 19 – 24, Group IV: MELD > 24. Lipid profile including serum total cholesterol, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, very low density lipoprotein (VLDL) cholesterol and serum triglycerides were measured in all cases after a 12 hour overnight fast. A similar group of 50 healthy persons, age and sex matched, served as controls. Controls were non-smokers, non-alcoholic, non-obese (BMI 18.0 – 23.0 kg/m^2, waist circumference ≤ 90 cm in males, ≤ 80 cm in females) and had no history of Coronary artery disease. Diabetes mellitus, Hypertension, Chronic Kidney Disease, Liver disease and Thyroid disorders were ruled out before performing fasting lipid profile.

Quantitative data were described as mean and standard deviation with 95% confidence intervals (CIs). Lipid profile was compared between the control group and the cirrhotic group and was also compared among various subgroups of cirrhotic patients. For comparison of nominal data, chi – square ($\chi 2$) test was used. Continuous variables between two groups were compared using the Student t test. For comparison of continuous variables between more than 2 groups, one-way analysis of variance (ANOVA) was used. Receiver Operating Characteristic curves was calculated for various lipid parameters. *P* value <0.05 was considered statistically significant.

Results

In this study, 150 patients with cirrhosis of liver and 50 age and sex matched controls were included. There were 121 male and 29 female patients. The age of the patients ranged from 20 - 65 years. Mean age of the patients was 44.02± 10.62 years (**Table – 1**).

Control group included 39 (78%) males and 11 (22%) females. Mean age of the control group was 45.7 ± 11.66 years. There was no significant difference between the cirrhotic and control group in terms of age and sex distribution (**Table – 1**).

The most common cause for cirrhosis in the study was alcohol (64 %). Other common causes were Hepatitis B and Hepatitis C. Maximum number of patients belonged to Child class B (48%) and Child Class A patients were least in number (18%). Most of the patients (66%) belonged to the MELD group of 11 – 18 followed by MELD group of ≤ 10 (14%) and 19 – 24 groups (13.3%) while only 6.66% of the patients belonged to the high MELD group of > 24 (**Table – 2**).

The mean values of serum total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol and triglycerides were lower in cirrhotic patients when compared to the control group (**Table – 3**).

Table – 1: Age and sex distribution of the cirrhotic group and the control group.

Age group (years)	Male n (%)	Female n (%)	Total n (%)
Age and sex distribution of the cirrhotic group (N=150)			
20 – 30	11(07.33)	4 (02.66)	15 (10.00)
31 – 40	34 (22.66)	9 (06.00)	43 (28.66)
41 – 50	42 (2.80)	11 (07.33)	53 (35.33)
51 – 60	23 (15.33)	4 (02.66)	27 (18.00)
61 – 70	11 (07.33)	1 (0.66)	12 (08.00)
Total	121 (80.66)	29 (19.33)	150 (100)
Age and sex distribution of the control group (N=50)			
20 – 30	4 (8)	1 (2)	5 (10)
31 – 40	12 (24)	2 (4)	14 (28)
41 – 50	10 (20)	2 (4)	12 (24)
51 – 60	9 (18)	5 (10)	14 (28)
61 – 70	4 (8)	1 (2)	5 (10)
Total	39 (78)	11 (22)	50 (100)

Table - 2: Etiology of Cirrhosis, Distribution of patients according to Child pugh classification and according to MELD Score.

Etiology	N =150 (%)
Alcohol	96 (64.00)
Hepatitis B	19 (12.66)
Hepatitis C	9 (06.00)
Alcohol + Hepatitis B	6 (04.00)
Alcohol + Hepatitis C	1 (0.66)
Budd Chiari Syndrome	1 (0.66)
Autoimmune Hepatitis	2 (1.33)
Cryptogenic	14 (9.33)
Wilson's Disease	2 (1.33)
Child's Class	
Class A	27 (18)
Class B	72 (48)
Class C	51 (34)
MELD Score	
≤ 10	21 (14)
11-18	99 (66)
19-24	20 (13.3)
>24	10 (6.66)

The mean values of serum total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol and triglycerides were progressively lower as the MELD score increased. The differences among the MELD subgroups were statistically significant ($p < 0.001$) as per **Table - 4**.

There was no statistically significant difference in the lipid profile between alcoholic and non alcoholic cirrhotic patients belonging to Child A ($p > 0.05$), alcoholic and non alcoholic cirrhotic patients belonging to Child B ($p > 0.05$) and alcoholic and non alcoholic cirrhotic patients belonging to Child C ($p > 0.05$) as per **Table - 5**.

The area under the curve to predict Child C cirrhosis was highest for total cholesterol (0.79) and least for HDL cholesterol (0.61) as per **Table – 6** and **Graph – 1 to Graph - 5**. The area under the curve to predict MELD > 24 was highest for LDL cholesterol (0.86) and least for HDL cholesterol (0.73) as per **Table – 7** and **Graph – 6 to Graph -10**.

Discussion

The present study found that all the five studied variables, (serum total cholesterol, LDL cholesterol, VLDL cholesterol, HDL cholesterol and triglycerides) were significantly lower in cirrhotic patients than in the comparison group. The amount of decrement in the serum total cholesterol, LDL, HDL, VLDL cholesterol and triglycerides had a positive correlation with the

severity of liver disease as assessed by Child's classification and MELD score. In our study, we found that mean serum total cholesterol in cirrhotic patients (147.54 mg/dl) was significantly lower than healthy controls (190.55 mg/dl) ($p < 0.001$). In study done by M R Ghadir, et al. [10], it was found that the mean serum total cholesterol in cirrhotic patients (138.9 mg/dl) was significantly lower than in healthy control group (184.6 mg/dl) (p=0.03).In study done by Fazle Subhan, et al. [14], it was found that the mean serum total cholesterol in cirrhotic patients (140.9 mg/dl) was significantly lower than in healthy control group (186.6 mg/dl) (p=0.031).In study done by S K Mandal, et al. [15], it was found that the mean serum total cholesterol in cirrhotic patients (141.5 mg/dl) was significantly lower than in healthy control group (192.0 mg/dl) (p<0.001).In study done by Edith Okeke, et al. [12], it was found that the mean serum total cholesterol in cirrhotic patients (143.85 mg/dl) was significantly lower than in healthy control group (158.93 mg/dl) (p=0.08).In study done by A C Sposito, et al. [13], it was found that the mean serum total cholesterol in cirrhotic patients (134.0 mg/dl) was significantly lower than in healthy control group (194.0 mg/dl) (p<0.003).In our study, we found that mean serum LDL cholesterol in cirrhotic patients (89.37 mg/dl) was lower than healthy controls (120.28 mg/dl).

Table - 3: Mean values of lipid profiles in control group and cirrhotic patients according to childs criteria.

Lipid	Control group Mean ± Standard deviation	Cirrhotic patients Mean ± Standard deviation	p value
Total Cholesterol (mg/dl)	190.55 ± 39.82	147.54 ± 35.46	< 0.001
LDL Cholesterol (mg/dl)	120.28 ± 27.01	89.37 ± 25.97	<0.001
VLDL Cholesterol (mg/dl)	28.04 ± 6.81	21.20 ± 6.25	<0.001
HDL Cholesterol (mg/dl)	42.33 ± 15.23	36.98 ± 11.94	0.012
Triglycerides (mg/dl)	140.12 ± 33.99	105.99 ± 31.32	<0.001

Table – 4: Lipid profile in cirrhotic patients according to MELD score.

Lipid	MELD ≤ 10 (n = 21)	11 ≤ MELD ≤ 18 (n = 99)	19 ≤ MELD ≤ 24 (n = 20)	MELD > 24 (n = 10)	*p* value
Total cholesterol (mg/dl)	202.86± 27.32	146.74 ± 25.69	114.00 ± 10.50	106.40 ± 0.61	< 0.001
LDL cholesterol (mg/dl)	128.62 ± 24.22	88.27 ± 19.9	67.05 ± 7.80	62.40 ± 11.92	< 0.001
VLDL cholesterol (mg/dl)	26.95 ± 5.96	21.49 ± 5.83	16.77 ± 2.66	15.01 ± 2.01	< 0.001
HDL cholesterol (mg/dl)	47.28 ± 12.32	36.97 ± 11.33	30.18 ± 6.84	28.99 ± 6.89	< 0.001
Triglycerides (mg/dl)	134.9. ±30.10	107.46 ± 29.12	83.85 ± 13.29	75.06 ± 10.06	< 0.001

In study done by M R Ghadir et al. [10], it was found that the mean serum LDL cholesterol in cirrhotic patients (80.5 mg/dl) was significantly lower than in healthy control group (137.2 mg/dl) (p=0.025). In study done by Fazle Subhan, et al. [14], it was found that the mean serum LDL cholesterol in cirrhotic patients (82.5 mg/dl) was significantly lower than in healthy control group (139.2 mg/dl) (p=0.024). In study done by S K Mandal, et al. [15], it was found that the mean

serum LDL cholesterol in cirrhotic patients (86.58 mg/dl) was significantly lower than in healthy control group (122.8 mg/dl) (p<0.001). In study done by Edith Okeke, et al. [12], it was found that the mean serum LDL cholesterol in cirrhotic patients (87.39 mg/dl) was significantly lower than in healthy control group (90.10 mg/dl) (p=0.68).In study done by A C Sposito, et al. [13], it was found that the mean serum LDL cholesterol in cirrhotic patients (93.0 mg/dl) was significantly lower than in healthy control group (122.0 mg/dl) (p<0.003).

Table - 5: Comparison of lipid profile between alcoholic cirrhotic and non alcoholic cirrhotic.

Lipid	**Alcoholic cirrhotic patients (Mean ± SD) (n = 21)**	**Non Alcoholic cirrhotic patients (Mean ± SD) (n = 6)**	**p value**
Child-A			
Total Cholesterol (mg/dl)	170.47 ± 32.19	172.33 ± 38.85	0.90
LDL Cholesterol (mg/dl)	103.90 ± 23.37	109.5 ± 34.78	0.65
VLDL Cholesterol (mg/dl)	24.90 ± 7.53	23.0 ± 4.19	0.56
HDL Cholesterol (mg/dl)	41.66 ± 11.17	39.83 ± 7.14	0.70
Triglycerides (mg/dl)	124.71 ± 37.91	114.66 ± 21.0	0.54
Child B			
Total Cholesterol (mg/dl)	155.24 ± 35.17	153.59 ± 36.06	0.85
LDL Cholesterol (mg/dl)	95.51 ± 25.06	93.29 ± 26.42	0.72
VLDL Cholesterol (mg/dl)	23.02 ± 5.69	21.25 ± 5.93	0.29
HDL Cholesterol (mg/dl)	36.71 ± 13.19	38.77 ± 10.30	0.49
Triglycerides (mg/dl)	115.11 ± 28.47	107.59 ± 29.65	0.29
Child C			
Total Cholesterol (mg/dl)	126.94 ± 25.22	120.5 ± 16.71	0.38
LDL Cholesterol (mg/dl)	78.89 ± 18.64	69.85 ± 15.31	0.37
VLDL Cholesterol (mg/dl)	18.19 ± 5.43	16.5 ± 2.47	0.27
HDL Cholesterol (mg/dl)	33.89 ± 12.69	34.14 ± 9.7	0.95
Triglycerides (mg/dl)	90.86 ± 27.04	81.86 ± 12.65	0.24

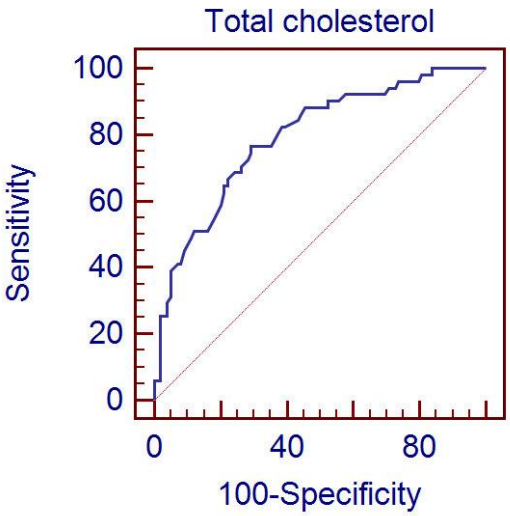
Table - 6: ROC curves analysis lipid profile to predict Child C cirrhosis.

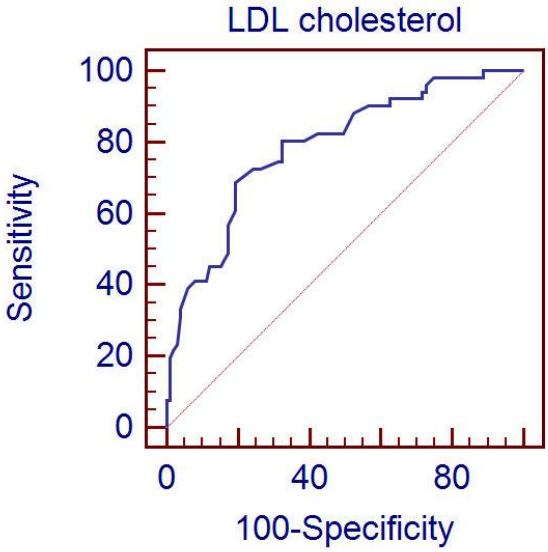
	Area under curve	**cut off value**	**Sensitivity**	**Specificity**
Total cholesterol	0.79	≤ 137 mg/dl	76.5 %	70.7 %.
LDL cholesterol	0.78	≤ 75 mg/dl	68.6 %	80.9 %.
VLDL cholesterol	0.70	≤ 19 mg/dl	78.4 %	68.7 %.
HDL cholesterol	0.61.	≤30 mg/dl	49 %	75.7 %.
Triglyceride	0.77	≤ 99 mg/dl	80.4%	67.7%.

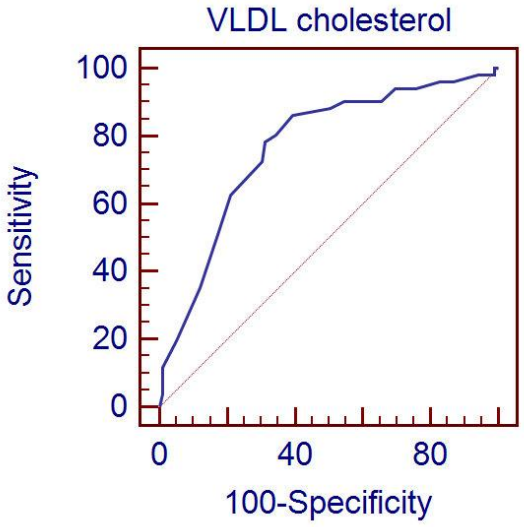
Table - 7: ROC curves analysis for lipid profile to predict MELD > 24.

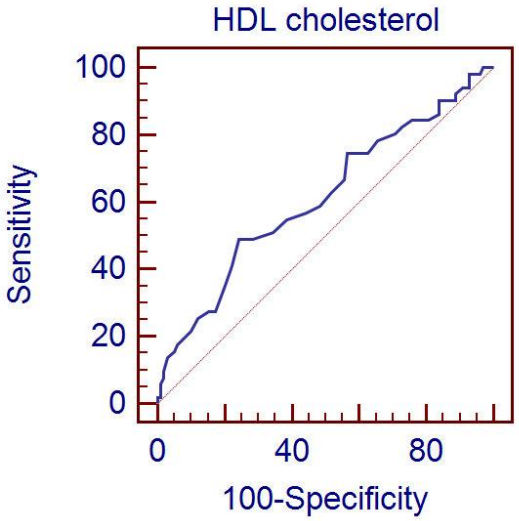
	Area under curve	**Cut off value**	**Sensitivity**	**Specificity**
Total cholesterol	0.74	≤ 123 mg/dl	100%	74.3%.
LDL cholesterol	0.86	72 mg/dl	90 %	73.6 %.
VLDL cholesterol	0.84.	≤ 18 mg/dl	100 %	59.3 %.
HDL cholesterol	0.73	30 mg/dl	60%	70 %.
Triglyceride	0.85	90 mg/dl	100 %	61.4 %.

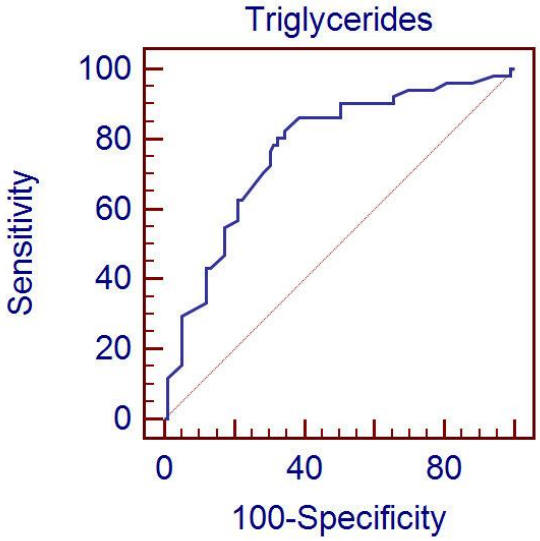
Graph – 1A to 1E: ROC curves for lipid profile to predict Child C cirrhosis.



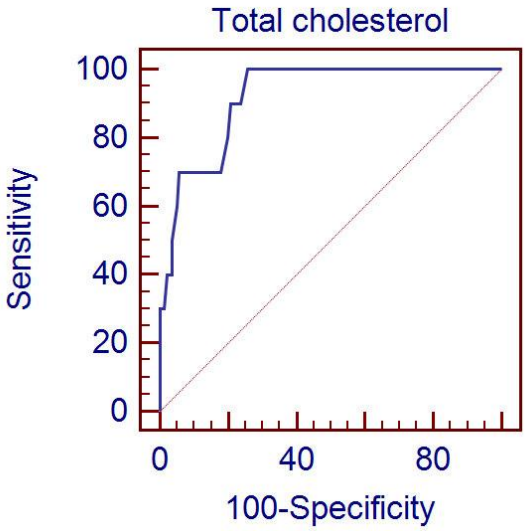


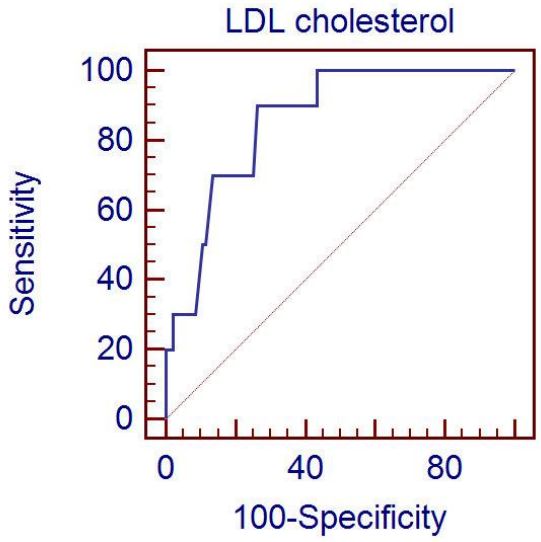


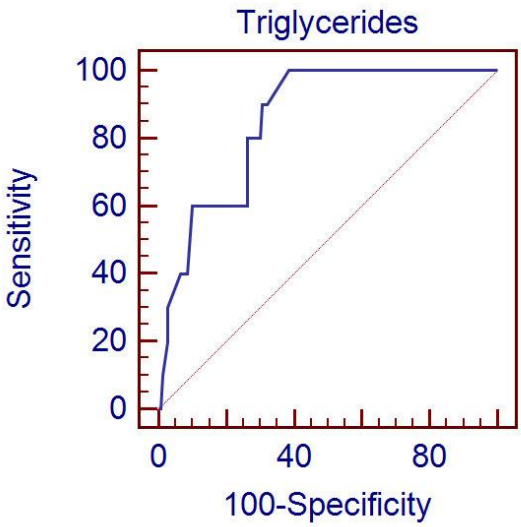


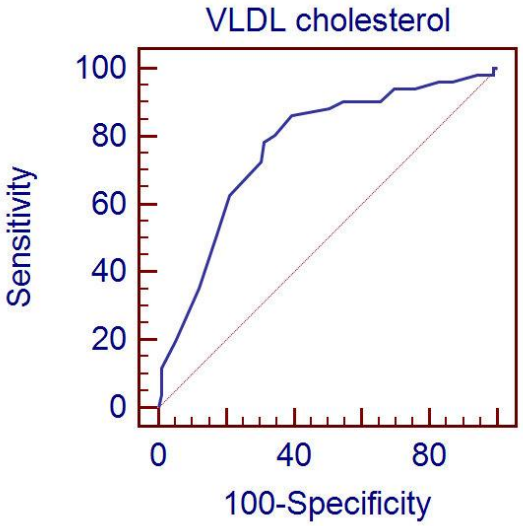


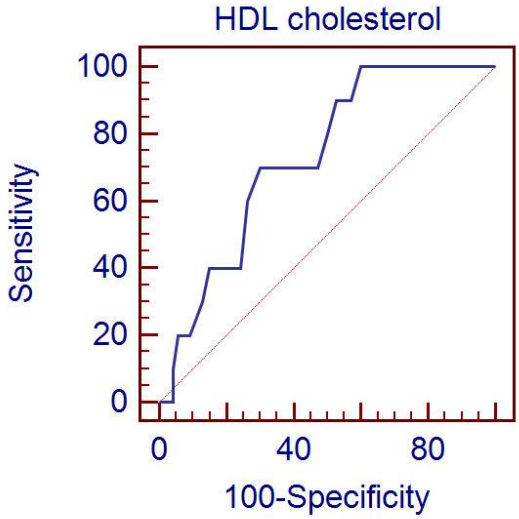
Graph – 2A to 2E: ROC curves for lipid profile to predict MELD > 24.











In our study, we found that mean serum HDL cholesterol in cirrhotic patients (36.98 mg/dl) was lower than healthy controls (42.33 mg/dl). This difference was statistically significant ($p < 0.001$). In study done by M R Ghadir, et al. [10], it was found that the mean serum HDL cholesterol in cirrhotic patients (40.7 mg/dl) was significantly lower than in healthy control group (44.5 mg/dl) ($p=0.043$). In study done by Fazle Subhan, et al. [14], it was found that the mean serum HDL cholesterol in cirrhotic patients (38.7 mg/dl) was significantly lower than in healthy control group (42.5 mg/dl) ($p=0.044$). In study done by S K Mandal, et al. [15], it was found that the mean serum HDL cholesterol in cirrhotic patients (33.5 mg/dl) was significantly lower than in healthy control group (41.78 mg/dl) ($p<0.001$).In study done by Edith Okeke, et al. [12], it was found that the mean serum HDL cholesterol in cirrhotic patients (33.25 mg/dl) was significantly lower than in healthy control group (43.31 mg/dl) ($p<0.001$).In study done by A C Sposito, et al. [13], it was found that the mean serum HDL cholesterol in cirrhotic patients (25 mg/dl) was significantly lower than in healthy control group (46 mg/dl) ($p<0.003$).

In our study, we found that mean serum VLDL cholesterol in cirrhotic patients (21.20 mg/dl) was lower than healthy controls (28.04 mg/dl). This difference was statistically significant ($p < 0.001$). In study done by S K Mandal, et al. [15], it was found that the mean serum VLDL

cholesterol in cirrhotic patients (23.53 mg/dl) was significantly lower than in healthy control group (27.52 mg/dl) (p=0.005). In study done by A C Sposito, et al. [13], it was found that the mean serum VLDL cholesterol in cirrhotic patients (16 mg/dl) was significantly lower than in healthy control group (25 mg/dl) (p<0.003). In our study, we found that mean serum triglycerides in cirrhotic patients (105.99 mg/dl) were lower than healthy controls (140.12 mg/dl). The difference was statistically significant (p< 0.001). In studies done by M R Ghadir, et al. (82.2), Fazle Subhan, et al. (84.2), A C Sposito, et al. (80), S K Mandal, et al. (120.9), it was found that serum triglycerides in cirrhotic patients was lower than healthy controls 187.2, 189.8, 126, 137.6 respectively and p was 0.011, 0.012, <0.003, 0.06 and <0.001 respectively.

In our study, among cirrhotic patients, mean serum total cholesterol was lower in Child B patients (154.63 mg/dl) than Child A patients (172.12 mg/dl) and mean serum total cholesterol was lower in Child C patients (125.18 mg/dl) than Child B patients. This difference was statistically significant (p< 0.001). In study done by M R Gadhir, et al. [10], mean serum total cholesterol was lower in Child B patients (161.2 mg/dl) than Child A patients (166.5 mg/dl) and mean serum total cholesterol was lower in Child C patients (121.2 mg/dl) than in Child B patients. This difference was statistically significant (p<0.05).In study done by Fazle Subhan, et al. [14], mean serum total cholesterol was lower in Child B patients (156.6 mg/dl) than Child A patients (165.6 mg/dl) and mean serum total cholesterol was lower in Child C patients (119.9 mg/dl) than in Child B patients. This difference was statistically significant (p<0.05).In study done by Andrzej Prystupa, et al. [11], mean serum total cholesterol was lower in Child B patients (162.57 mg/dl) than Child A patients (176 mg/dl) and mean serum total cholesterol was lower in Child C patients (157.16 mg/dl) than in Child B patients. This difference was not statistically significant (p>0.05).

In our study, among cirrhotic patients, mean serum LDL cholesterol was lower in Child B patients (94.68 mg/dl) than Child A patients (106.19 mg/dl) and mean serum LDL cholesterol was lower in Child C patients (73.51 mg/dl) than Child B patients. This difference was statistically significant (p< 0.001).In study done by M R Gadhir, et al. [10], mean serum LDL cholesterol was lower in Child B patients (96.4 mg/dl) than Child A patients (107 mg/dl) and mean serum LDL cholesterol was lower in Child C patients (59.5 mg/dl) than in Child B patients. This difference was statistically significant (p<0.05).In study done by Fazle Subhan, et al. [14], mean serum LDL cholesterol was lower in Child B patients (94.3 mg/dl) than Child A patients (110.6 mg/dl) and mean serum total cholesterol was lower in Child C patients (56.5 mg/dl) than in Child B patients. This difference was statistically significant (p<0.05).In study done by Andrzej Prystupa, et al. [11], mean serum LDL cholesterol was lower in Child B patients (83.75 mg/dl) than Child A patients (112 mg/dl) and mean serum total cholesterol was higher in Child C patients (84.89 mg/dl) than in Child B patients. This difference was not statistically significant (p>0.05).In our study, among cirrhotic patients, mean serum HDL cholesterol was lower in Child B patients (37.49 mg/dl) than Child A patients (41.88 mg/dl) and mean serum HDL cholesterol was lower in Child C patients (33.99 mg/dl) than Child B patients. This difference was statistically significant (p< 0.001).In study done by M R Gadhir, et al., mean serum HDL cholesterol was lower in Child B patients (40.0 mg/dl) than Child A patients (49.0 mg/dl) and mean serum HDL cholesterol was lower in Child C patients (37.4 mg/dl) than in Child B patients. This difference was statistically significant (p<0.05).In study done by Fazle Subhan, et al., mean serum HDL cholesterol was lower in Child B patients (42 mg/dl) than Child A patients (47 mg/dl) and mean serum HDL cholesterol was lower in Child C patients (35.3 mg/dl) than in Child B patients. This difference was statistically significant (p<0.05).In study done by Andrzej Prystupa, et al. [11], mean serum HDL cholesterol was lower in Child B patients (27.36 mg/dl) than Child A

patients (45 mg/dl) and mean serum HDL cholesterol was higher in Child C patients (32.56 mg/dl) than in Child B patients. This difference was statistically significant (p<0.001).In our study, among cirrhotic patients, mean serum triglycerides was lower in Child B patients (112.29 mg/dl) than Child A patients (120.35 mg/dl) and mean serum triglycerides was lower in Child C patients (88.38 mg/dl) than Child B patients. This difference was statistically significant (p< 0.001). Though studies by M R Ghadir and Fazle Subhan reported lower mean serum triglycerides in Child C patients when compared to Child A patients, this difference was statistically not significant. This may possibly be because ours being a tertiary care centre, patients with more advanced and long standing cirrhosis were included.

Further, we have excluded smokers in the present study which was not done in the previous studies. As smoking increases serum triglycerides, this may also have contributed to the difference in results. In our study, among cirrhotic patients, mean serum total choletsterol was progressively lower in sub groups of patients with increasing MELD scores, with lowest mean value noted in patients with MELD score > 24 (106.4 mg/dl) and highest mean value noted in patients with MELD score ≤ 10 (202.8 mg/dl). This difference in various subgroups of MELD was statistically significant (p < 0.001). Similar finding was reported by MR Ghadir, et al. and Fazle Subhan, et al. In our study, among cirrhotic patients, mean serum LDL cholesterol was progressively lower in sub groups of patients with increasing MELD scores, with lowest mean value noted in patients with MELD score > 24 (62.40 mg/dl) and highest mean value noted in patients with MELD score ≤ 10 (128.62 mg/dl). This difference in various subgroups of MELD was statistically significant (p < 0.001). This is in accordance with studies by MR Ghadir, et al. and Fazle Subhan, et al. In our study, among cirrhotic patients, mean serum HDL cholesterol was progressively lower in sub groups of patients with increasing MELD scores, with lowest mean value noted in patients with MELD score > 24 (28.99 mg/dl) and highest mean value noted in patients with MELD score ≤ 10 (47.28 mg/dl). This difference in various subgroups of MELD was statistically significant (p < 0.001). This is in accordance with studies by MR Ghadir, et al. and Fazle Subhan, et al. In our study, among cirrhotic patients, mean serum triglycerides was progressively lower in sub groups of patients with increasing MELD scores, with lowest mean value noted in patients with MELD score > 24 (75.01 mg/dl) and highest mean value noted in patients with MELD score ≤ 10 (134.9 mg/dl). This difference in various subgroups of MELD was statistically significant (p < 0.001). Studies by M R Ghadir, et al. and Fazle Subhan, et al. also reported progressively lower mean serum triglycerides within subgroups of patents with increasing MELD score, but it was not statistically significant. This may possibly be because ours being a tertiary care centre, patients with more advanced and long standing cirrhosis were included. Further, we have excluded smokers in the present study which was not done in the previous studies. As smoking increases serum triglycerides, this may also have contributed to the difference in results.

Conclusion

Lipid profile abnormalities are common in patients with cirrhosis of liver. Future classification criteria for assessment of severity of cirrhosis of liver and liver transplant listing criteria may incorporate serum lipid profile parameters to increase their accuracy, but further studies are needed to assess the predictive values of lipid profile to estimate the extent of liver damage in cirrhotic patients.

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