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Original Research Article

Lipid profiles of patients as markers for the severity of liver cirrhosis

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ABSTRACT

Background: The lipoproteins, endogenous lipids and apolipoproteins are formed in the liver and lipid metabolism, stages of lipid synthesis and transportation are also control by the liver. Degenerated serum lipoprotein pattern can be observed during the acute metabolic impairment in cirrhosis hence, it is reasonable to expect an abnormal lipid profile in those with severe liver dysfunction.
Methods: This cross-sectional study was carried out at a tertiary care hospital, in central Rajasthan among 360 cases of cirrhosis of liver from all causes admitted in Medicine and Gastroenterology wards of Jawaharlal Nehru Medical College, Ajmer. Venous blood sample was collected after overnight fasting of 12 h in all the patients diagnosed with cirrhosis for estimation of hemoglobin, total white blood cell (WBC) count, total platelet count, erythrocyte sedimentation rate (ESR), random blood sugar, blood urea, serum creatinine, liver function tests and serum lipid profile.
Results: The mean high-density lipoprotein (HDL) levels were decreasing statistically with increasing model for end stage liver disease (MELD) score while low-density lipoprotein (LDL) and triglyceride (TG) showed no significant association with increasing MELD score. The sensitivity of HDL in detecting the liver cirrhosis at cut off of 19.3 MELD score was estimated to be 93% and specificity of 82.1%.
Conclusions: The mean HDL levels were significantly decreasing with increasing MELD score and the study of lipid profile of patients may be an alternate to assess the severity of liver cirrhosis.

Keywords: MELD, HDL, LDL, TG, Liver disease

INTRODUCTION

Chronic liver disease is becoming major public health issues across the world.[1-3] The liver is a chemical factory performing more than 500 chemical functions in the human body and plays an important role in cholesterol and lipid synthesis, transport, and metabolism. Balance between the biosynthesis, utilization and transport of lipids is highly crucial in a healthy body. But the liver cirrhosis may lead to significant reduction in the glycogen reserves that may induce lipolysis and malnutrition.[4] The cirrhosis has been reported to be linked with reduction in serum levels of high-density lipoprotein (HDL), and low-density lipoprotein (LDL).[5] Lipoprotein levels are inversely proportionate to the severity of liver disease and lower level of HDL may worsen the situation leading to liver transplantation in patients with non-cholestatic cirrhosis.[6,7]

Cirrhosis is a common hepatological disorder associated with a wide range of distinguishable clinical indicators and depends mainly on the etiology and deformity in the hepatic architectural. The morphological deformity in the hepatic architectural may include necrosis of the cells followed by fibrosis and nodular regeneration, modified lobular architecture and formation of intrahepatic vascular shunts between portal vein and hepatic artery and vein vessels of the liver.[8,9] Studies also reported that capillarization of sinusoids and perisinusoidal fibrosis, vascular thrombosis and obliterative lesions in portal tracts

and hepatic veins, under-perfusion of lobular parenchyma and consequent tissue hypoxia may be other characteristics of hepatic architecture.[10,11] These changes increase the portal hypertension and other relative complications causing the death of cirrhotic patients.[12]

Several studies have reported chronic hepatitis B and C, fatty liver disease, autoimmune hepatitis, chronic biliary diseases and several other inherited metabolic disorders as major reasons of liver cirrhosis that may lead to hepatic failure and hepatocellular carcinoma.[13,14] Cirrhosis causes 1.16 million deaths per year and has been reported to be the 11th most common reason of death worldwide. India accounts for 18.3% of all cirrhosis deaths globally.[15] Cirrhosis is fourth prominent cause of deaths among Asian males. Liver cirrhosis cannot be a solitary disease unit but involves different clinical prognostic stages with one-year mortality fluctuating between 1-57% based on the predictive stages.

Diagnosis of cirrhosis mainly depends on histopathological evidence of stage 4 fibrosis using METAVIR system, or stages 5 or 6 in the Ishak scoring system. Liver fibrosis is divided in to 5 groups i.e., F0 to F4 in METAVIR system whereas, it is divided in 7 categories i.e., 0 to 6 in Ishak system. These are static diagnosis that reflect the end stage of wound healing process but fails to signify the complexity of pathogenesis, or functional, hemodynamic and prognostic correlates. The evident of understandings on the biological mechanisms of fibrogenesis, angiogenesis and involvement of extra-hepatic inflammation suggest the inadequacy of one-stage description such as METAVIR and Ishak scoring system for advanced fibrotic liver disease.

The cirrhotic patients need frequent visit and multiple hospitalizations for management of cirrhosis or related complications. The proper treatment plan of cirrhosis depends on the severity, type of liver damage and possibility of assessing its extent. The model for end stage liver disease (MELD) criteria is a chronic liver disease severity scoring system in which standards of serum bilirubin, serum creatinine, and the international normalized ratio (INR) for prothrombin time of the patients are used to decide three-month survival. The liver cirrhosis patients having higher MELD score is correlated with more severity of hepatic dysfunction and three-month mortality risk.[16]

Due to robustness of MELD scoring system in estimating the short-term survival among patients with cirrhosis, it has been approved by the United Network for Organ Sharing (UNOS) for prioritizing patients awaiting liver transplantation.[17] The emergence of new treatments for liver disease including licensed drugs, such as non-selective β blockers, statins, oral antibiotics, and anticoagulants in recent years, the evaluation system required to assess the severity and prognosis of patients with decompensated cirrhosis is an urgent necessity.[18]

Lipids are vital chemical compounds that control cellular function and homeostasis in the human body.[19] These are fatty substances and don't dissolve in water, therefore they are unable to move through blood itself. Hence, they combine with the proteins to make lipoproteins that can travel throughout the body. The lipid metabolism, stages of lipid synthesis and transportation are control by the liver. Basically, lipoproteins, endogenous lipids and apolipoproteins are formed in the liver. It has been observed that more than 80% of endogenous cholesterol is synthesized in the hepatocellular microsomes therefore, synthesis and metabolism of cholesterol are hampered in chronic liver disease leading to decrease in plasma levels. Degenerated serum lipoprotein pattern can be observed during the acute metabolic impairment in cirrhosis hence, it is reasonable to expect an abnormal lipid profile in those with severe liver dysfunction.[20]

Several studies have been carried out on lipoprotein profile alterations in those with liver disease throughout the world including from India.[21] Lower serum levels of HDL and LDL cholesterol have been reported in patients with liver cirrhosis.[22] The patients with chronic liver disease have been reported with significantly reduced serum total cholesterol (TC) and triglycerides (TG) levels and LDL, HDL, and very LDL (VLDL) levels.[23,24] The present cross-sectional study was conducted to determine the lipid profile in cirrhosis of liver patients and to assess their correlation with the severity of liver cirrhosis.

METHODS

The present study was carried out as a descriptive, cross-sectional study in the Department of Medicine and Gastroenterology at Jawaharlal Nehru Medical College, Ajmer, Rajasthan India during September 2021 to September 2022.

Prior ethical approval of the study was obtained from the Institutional Ethical Committee and written permission was undertaken from all the patients enrolled under the present study.

From the earlier studies, the occurrence of dyslipidmia with liver cirrhosis was 36.6%. The sample size was estimated based on prevalence of disease using following formula, where; n=sample size, z=1.96 (considering 0.05α, 95% confidence limits and 80% ß), p=assumed probability of occurrence or concordance of results, q=1–p and d=marginal error (precession). So in our study the estimated sample size was 360.

$$n = z^2pq/d^2$$

Inclusion criteria

All the ≥18 years old patients diagnosed with cirrhosis of liver due to any cause from clinical and investigative modalities were considered in the study.

Exclusion criteria

Patients with any of the following reasons were omitted from the study because these conditions may influence the lipid profiles: history of dyslipidemia prior to development of liver disease, patients on lipid lowering drugs such as statins, fibrates, drugs altering the lipid profile levels viz., amiodarone, loop and thiazide diuretics, diabetes mellitus, hypertension, cerebrovascular disease, cardiovascular disease, pancreatitis, and pregnant women.

The patients under the study were adults (≥18 years old) with cirrhosis of liver due to all causes admitted in the hospital. Their diagnosis was made by way of detailed history, clinical and histological examinations. Detailed history and physical examination was thoroughly recorded for all the patients under investigation. The patients were asked about past history of jaundice, blood transfusion, marital and sexual history and duration of alcoholism (if present), history of thyroid disease before the development of liver disease. Physical examination for the stigmata of chronic liver disease was routinely performed in all the patients. Ophthalmologic examination (slit-lamp examination) was also done to look for Kayser-Fleischer ring (KF ring). Detailed cardiovascular and neurological examination was also performed. Each patient was examined in the following order after the completion of physical examination:

Venous blood (5 ml) was collected after overnight fasting of 12 hours in all the patients diagnosed with cirrhosis for estimation of hemoglobin, total WBC count, total platelet count, ESR, random blood sugar, blood urea, serum creatinine, liver function tests and serum lipid profile (LDL, HDL, TG). Triglycerides (TG) and high-density lipoprotein (HDL) cholesterol was measured by direct method and serum low-density lipoprotein (LDL) was calculated by using Friedewald formula.

$$LDL\ cholesterol = total\ cholesterol - (HDL\ cholesterol - TG/5)$$

The ultrasound study of abdomen and pelvis region and Portal vein Doppler study was also conducted.

Model for end-stage liver disease (MELD) score was used to assess the severity of cirrhosis of liver. The components of MELD score considered under the study included bilirubin, creatinine and INR and the score was calculated using the following formula:

$$MELD = 3.78 \times loge\ serum\ bilirubin\ (mg/dl) + 11.20 \times loge\ INR + 9.57 \times loge\ serum\ creatinine\ (mg/dl) + 6.43\ (constant\ for\ liver\ disease\ etiology)$$

The range of MELD score was 6-40. The normal ranges for lipid profile in our laboratory were LDL: 20-150 mg, HDL (male: 30-65 mg and female: 35-80 mg) and triglycerides: up to 150 mg.

Statistical analysis was performed using statistical package for the social sciences (SPSS) 20 software and the analyzed data were expressed in percentages and in mean±SD. P value equal to or less than 0.05 was considered to be significant. The receiver operating characteristic (ROC) analysis was carried out for lipid profile to predict the MELD score.

RESULTS

The mean age of the patient under the study was 45.05±11.36 years wherein minimum age was 27 years while maximum was 76 years. The population under the study included 88.9% (n=320) males and 11.1% females (n=40). The majority (48.3%) of the study population were lying in the age group of 36-45 years (Figure 1).

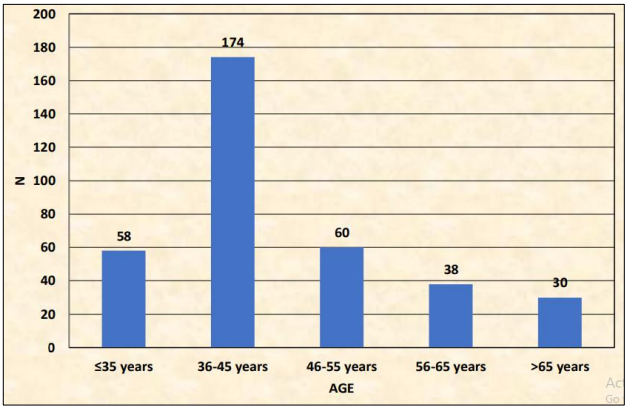

Figure 1: Age distribution of participants under the study.

There was no consistency in the MELD score with respect to the age group under the present study for example MELD score of ≤10 was observed higher in the age group 56-65 years, MELD score 11-18 was seen in age group ≤35 years, 19-24 in age group >65 years while MELD score >24 was recorded in the age group of 36-45 years (Table 1). However, except the patients of age group of >65 years, patients of all the age groups showed increasing MELD scores from ≤10 to >24. There was no significant difference in MELD score (severity of disease) with respect to age group in the present study. Similarly, there was no significant difference in number of females and males with varying MELD scores. The MELD scores invariably showed increasing trends from ≤10 to >24 across all the male and female patients.

The severity of cirrhosis according to the MELD scores showed that 47.8% of study population had MELD score ≥24 while only 2.8% of study participants had MELD score ≤10 (Table 2). The mean of MELD score recorded for total study participants was 24.11±7.53.

In the patients with MELD score ≤10, mean LDL, HDL, and TG levels were 92.8±5.13 mg, 38.6±8.65 mg and 188.2±66.52 mg, respectively (Table 3). The patients with MELD score 11 to 18 had mean LDL, HDL, and TG levels of 78.3±29.53 mg, 33.35±4.26 mg and 128.15±47.12 mg, respectively. In patients with MELD score 19 to 24, the mean LDL, HDL, and TG levels were recorded to be 83±29.16 mg, 29.12±6.27 mg and 124.82±47.71 mg, respectively while the patients with MELD score >24 showed mean LDL, HDL, and TG levels of 97.35±102.79 mg, 24.4±5.406 mg and 141.2±172.41 mg, respectively. The mean HDL levels decreased significantly with the increasing MELD score while LDL and TG had no statistically significant association with increasing MELD score. The mean TG levels showed decreasing trends with all the MELD scores except >24 where a slight increase in TG level was observed. However, all these changes in TG levels were statistically non-significant. Almost similar trends were observed with LDL levels.

Table 1: Relation of age with MELD score.

Age (years)		MELD score				Total
		≤10	11 to 18	19 to 24	>24	
≤35	N	2	18	12	26	58
	%	3.4	31.0	20.7	44.8	100
36-45	N	4	30	44	96	174
	%	2.3	17.2	25.3	55.2	100
46-55	N	2	18	16	24	60
	%	3.3	30.0	26.7	40.0	100
56-65	N	2	6	12	18	38
	%	5.3	15.8	31.6	47.4	100
>65	N	0	8	14	8	30
	%	0.0	26.7	46.7	26.7	100
Total	N	10	80	98	172	360
	%	2.8	22.2	27.2	47.8	100

P value=0.07

Table 2: Severity of cirrhosis by MELD score.

MELD score	N	%
≤10	10	2.8
11 to 18	80	22.2
19 to 24	98	27.2
>24	172	47.8
Total	360	100

The sensitivity of HDL in detecting the liver cirrhosis at cut off of 19.3 MELD score was estimated to be 93% and specificity of 82.1%. Similarly, LDL under the present study showed 95% sensitivity and 83.2% specificity with a cut off value of 14.34% with MELD score (Figure 2). While TG showed 100% sensitivity and 75.9% specificity with a value cut off 46.4% with MELD score.

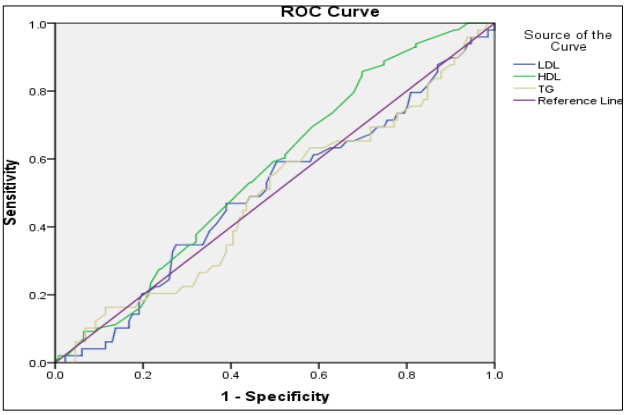

Figure 2: ROC analysis for lipid profile to predict MELD score.

Table 3: Mean lipid profile value with severity of cirrhosis (MELD score) of liver.

Variable	MELD score								P value
	≤10		11 to 18		19 to 24		>24		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
LDL (mg)	92.8	5.13	78.3	29.53	83	29.16	97.35	102.79	0.21
HDL (mg)	38.6	8.65	33.35	4.26	29.12	6.27	24.4	5.406	0.001
TG (mg)	188.2	66.52	128.15	47.12	124.82	47.71	141.2	172.41	0.37

DISCUSSION

In our study the mean age of the patients was 45.05±11.36 years which was in the range of 27 to 76 years. The population under the study included 88.9% males and 11.1% females. While, majority (48.3%) of the study population were in the age group of 36-45 years. Suman et al also reported age of the patients in the range from 20 to

65 years with mean age of the patients 44.02±10.62 years.[25] Similarly, age of the patients ranged from 40 to 60 years with mean age of the patients 48.27±11.12 years reported by Singh et al are comparable to our study.[26] Higher percentage of male (75.6%) patients were included in the earlier study.[26]

In our findings 47.8% of the participants had MELD score >24 and only 2.8% of the participants were reported with MELD score of ≤10. The mean of MELD score recorded for total study participants was 24.11±7.53 revealing no consistency in the MELD score with respect to the age group. This contrast may be due to involvement of more decompensated CLD patients in our study at baseline. Our study corroborates with the earlier studies in which only 6.6% of study subjects had very severe disease (MELD>24) and 13.3% of patients had MELD score in the range of 19 to 24.[25] Majority of the patients (66%) with cirrhosis had MELD score in the range of 11 to 18 which is in contrast to the present study where in majority of the cases had MELD>24. In patients under the present study with MELD score ≤10, mean LDL, HDL, and TG levels were 92.8±5.13 mg, 38.6±.65 mg and 188.2±66.52 mg, respectively. In patients with MELD score 11 to 18, mean LDL, HDL, and TG levels were 78.3±29.53 mg, 33.35±4.26 mg and 128.15±47.12 mg, respectively. In patients with MELD score 19 to 24, mean LDL, HDL, and TG levels were 83±29.16 mg, 29.12±6.27 mg and 124.82±47.71 mg, respectively. In patients with MELD score >24, mean LDL, HDL, and TG levels were 97.35±102.79 mg, 24.4±5.406 mg and 141.2±172.41 mg, respectively. The mean HDL levels were decreasing statistically with increasing MELD score while LDL and TG had no statistically significant association with increasing MELD score. The mean TG levels were also decreasing with all the MELD scores except >24 where a slight increase in TG level was observed. The changes under the study may be due to the impaired production of endogenous lipids, apoprotein, apo-lipoproteins in cirrhotic liver. Chrostek et al also reported decreased HDL and LDL levels in liver cirrhosis patients and suggested that it might be due to the decrease synthesis of apolipoprotein A and B.[27] It was also reported that decline in lipoprotein cholesterol may reflect deterioration in liver function in cirrhotic patients.[28] Significantly declined trend in HDL cholesterol was reported with increasing MELD scores and also the amount of decrease in serum LDL, total cholesterol, VLDL cholesterol, HDL and triglycerides showed positive correlation with severity of liver cirrhosis evaluated by MELD score.[25,29,30] Similarly, Jiang et al observed lower levels of serum triglyceride, total cholesterol, HDL and LDL level with increasing MELD score.[31] All these studies are comparable to our study in terms of HDL values.

A receiver operating characteristic (ROC) analysis is an efficient tool for quantifying the influence of variability among individuals' decision thresholds. The ROC analysis for lipid profile carried out to predict the MELD score have been presented in Figure 2. The ROC curve was obtained as the cumulative distribution function which was the area under the probability distribution from -∞ to the discrimination threshold against specificity on the y-axis and sensitivity on the x-axis to quantify the sensitivity as a function of false positive rate. The sensitivity of LDL in detecting the cirrhosis of liver at cut off of 14.35 based on ROC analysis under the present study was 95% with the specificity of 83.2% similarly, the sensitivity of HDL in detecting the cirrhosis of liver at cut off of 19.3 was 93% with the specificity is 82.1%. While, the sensitivity of TG in detecting the cirrhosis of liver at cut off of 46.4 was 100% with the specificity of 75.9% in the present study. ROC analysis revealed substantial variability in the diagnostic performance of these indices, underlining the intricacy of predicting complications in cirrhotic patients precisely. Suman et al observed that the area under the curve to predict cirrhosis was highest for total cholesterol (0.79) while it was least for HDL cholesterol (0.61).[25] They reported that the area under the curve to predict MELD >24 was highest for LDL cholesterol (0.86) and it was least for HDL cholesterol (0.73).

CONCLUSION

The mean HDL levels under the present study were significantly decreasing with increasing MELD score. The changes may be due to the impaired production of endogenous lipids, apoprotein, apo-lipoproteins in cirrhotic liver. Hence, the study of lipid profile of patients may be an alternate to assess the severity of liver cirrhosis.

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