Original Research Article

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Correlation of lipid profile in patients with severity of liver disease: a cross sectional study in a tertiary care hospital

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ABSTRACT

Background: Many previous studies concluded variation in the lipid parameters such as total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) with severity of liver disease. Hence, this study was conducted to find out the correlation of lipid profile in patients with severe liver disease.

Methods: A cross sectional study which included 170 patients admitted with chronic liver disease. Severity of liver disease according to Child Pugh Turcotte Score. The patients were subjected to routine investigation and fasting lipid profile test. Correlation was studied using the Pearson correlation coefficient and the comparison of lipid parameters was also done.

Results: Total of 170 consecutive chronic liver disease patients were analysed over a period of one year. Majority of the patients were of the age 51 to 60 years (39.8%). Among the total, 24 patients were in Child Pugh Turcotte Score class A, 47 patients were in class B and 52 were patients in class C. We could observe a significant (p<0.001) negative correlation of all the lipid profile parameters with the severity of liver disease.

Conclusions: Serum TC, LDL TG and HDL were decreased in patients with cirrhosis and they are inversely correlated to severity of disease.

Keywords: Cirrhosis, Chronic liver disease, Child Pugh Turcotte Score, Lipid profile, Non-alcoholic seatohepatitis

INTRODUCTION

Cirrhosis is defined as a diffuse process characterized by fibrosis and the conversion of normal liver architecture into structurally abnormal nodules. Autopsy studies done globally showed prevalence of cirrhosis ranging from 4.5 to 9.5% of the general population. Hence more than fifty million adult population in the world would be affected with cirrhosis liver. ^{2,3}

In the Western world alcoholic liver disease and hepatitis C remains the most common cause of cirrhosis, while hepatitis B prevails in most parts of Asia and sub-Saharan Africa. Currently along with alcohol and viral hepatitis, nonalcoholic steatohepatitis (NASH) is another most

common etiology for cirrhosis.² Biological membranes, free molecules and metabolic regulators contains lipid and are essential in controlling cellular function and homeostasis. Liver has important role in lipid metabolism hence it is profoundly disturbed in a variety of ways in severe liver disease.⁵

Circulating lipoproteins are seen in abnormal amount, abnormal composition, electrophoretic mobility and appearance.⁵ Cardiovascular disease (CVD) risk stratification includes serum lipid profile. It is rarely considered a useful screening tool for the evaluation of liver diseases, yet there is reason to think otherwise.⁶ American Heart Association guideline, 2013 suggest once therapy for dyslipidemia is started there is no need

of monitoring.⁷ Many previous studies concluded an inverse association of lipid parameters such as total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) with severity of liver disease. However, some other studies did not find such correlation especially for the TG and HDL levels.^{8,9} Further, Sen et al showed that TC, HDL and TG were higher in grade 3 fatty liver.¹⁰ This warrant the significance of a population based evaluation of the lipid profile in subjects with liver disease. Hence, this study was conducted to find out the correlation of lipid profile in patients with severe liver disease.

METHODS

A cross sectional study which included the patients admitted in the department of General Medicine and Gastroenterology, Amala Institute of Medical Sciences Thrissur, Kerala, India from January, 2015 to January, 2016. Patients with age ≥18 years with chronic liver disease or documented case of chronic liver disease irrespective of aetiology were included in the study. Proven cases of dyslipedemia prior to detection of chronic liver disease, recent parenteral nutrition, patient on immunosuppressive drug, patients with BMI >30 or patients unwilling to participate in the study were excluded. Informed consent was obtained from either the patient or bystander. The study was conducted after acquiring consent from the institutional scientific and ethics committee, and by abiding the rules and regulations as per Helsinki Declaration.

Detailed history was taken from each patient to ascertain past and present illness. All the patients were subjected to a thorough physical examination using specific proforma. Severity of liver disease was calculated according to Child Pugh Turcotte Score. Patients were subjected to routine investigation and fasting lipid profile test. Routine test included complete blood count (CBC), urine routine, random blood sugar, renal function test, liver function test, HBsAg, HCV antibody, Prothrombin time (PT/INR), ultrasonography of whole abdomen. A fasting serum lipid profile included TC, TG, HDL and LDL.

Statistical analysis

The data were analysed using the SPSS software (version 16, IBM, New York, USA). Correlation was studied using the Pearson correlation coefficient. Comparison of lipid parameters with the severity of liver disease was done using Kruskal-Wallis Test. P<0.05 was considered significant.

RESULTS

Total of 170 consecutive chronic liver disease patients were analysed over a period of one year. Among them 47were excluded as per exclusion criteria. Among the remaining 123 patients, 104 were male (84.6%) and 19 were female (15.4%) (Figure 1). Majority of the patients

were of the age 51 to 60 years (39.8%) (Figure 2). We had 24 patients in CPT class A, 47 patients in class B and 52 patients in class C (Figure 3).

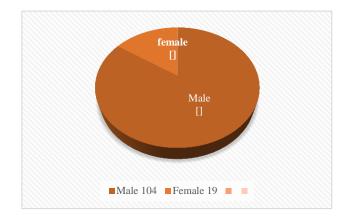


Figure 1: Distribution of gender.

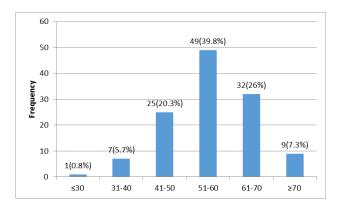


Figure 2: Distribution of age.

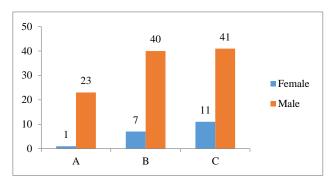


Figure 3: Distribution of severity of disease.

Table 1: Major symptoms of the patients.

Major symptoms of the patients	Percentage (%)
Abdominal distension	49.59
Jaundice	30.08
Altered sleep rythm	30.08
Malena	29.27
Altered behaviour	23.58
Hematemesis	18.70
Breathlessness	10.57

The main clinical symptoms for admission were abdominal distension (61%) and jaundice (37%) (Table 1). Co-morbidities were found in patients. Among the total patients, 63 % were using alcohol, 43% had diabetes mellitus, 37.40% had systemic hypertension, and 8% had

chronic kidney disease. Present study showed that 98 people had portal hypertension (as evidenced by USG). Serum lipid profile is given in Table 2. We could observe a negative correlation between all the lipid profile parameters with the severity of liver disease (Table 3).

Table 2: Serum lipid profile in patients with chronic liver disease.

	CPT		Calculated	P value	
	A	В	С	value	
Total cholesterol (mg/dl)	139.08±40.16	141.30±51.54	105.81±40.26	9.02	0.0001
LDL (mg/dl)	84.29±35.16	88.55±48.13	76.67±79.19	8.63	0.013*
HDL (mg/dl)	34.42±14.67	29.81±12.82	21.23±11.73	21.62	0.0001*
TG (mg/dl)	109.17±64.04	116.32±54.99	90.06±53.35	11.16	0.004*

Comparison done using Kruskal-Wallis Test; p< 0.05 is considered significant.

Table 3: Correlation between the laboratory parameters with the severity of disease.

	HB	MCV	PLT	PT INR	BIL-T	ALB	S CHL	LDL	HDL	TG
Pearson Correlation coefficient (r) With	-0.130	-0.018	-0.349	0.691	0.661	-0.706	-0.387	-0.129	-0.434	-0.191
score										
P value	0.152	0.840	0.0001	0.0001	0.0001	0.0001	0.0001	0.154	0.0001	0.034

HB: hemoglobin; MCV: Mean corpuscular volume; PT/INR: Prothrombin time in international normalized ratio; BIL-T: Total bilirubin; ALB: Albumin; S CHL: Serum cholesterol; LDL: Low density cholesterol; HDL: High density cholesterol; TG: Triacyl glycerol.

DISCUSSION

The result of the present study revealed that more number of patient were in class C. All the parameters of lipid profile were lower in the severe form of liver disease irrespective of the etiologies. Furthermore, the amount of decrement in the serum HDL, LDL, TC and TG had a negative correlation with the severity of liver disease. This indicated an inverse correlation of lipid parameters with the severity of the disease. The result was consistent to the previous studies. ^{5,11-13} They found a reduction in all parameters of lipid profile with severity of chronic liver disease. However, there is conflicting of observations on this regard. Previous study by Mandal et al found thatserum TG and HDL levelwere not reduced with severity of liver cirrhosis. ⁸

The decreased LDL and HDL levels found in patients with liver cirrhosis might be ascribed to the decreased synthesis of apolipoproteins (Apo) A and B. Habib et al previously reported that decline in lipoprotein cholesterol may reflect deterioration lipoproteins synthesis in liver. ^{14,15} Since the apo B is involved in the synthesis of very low density lipoproteins, the exhibited lowering of TG can be explained to its lowered synthesis in liver. This can be due to the insulin resistance (IR) found in liver diseases. Insulin signalling mechanism was found to be critical for the lipogenesis in hepatocytes by regulating the PI3K and AKT2 signalling pathways. ^{16,17} Among the

transcription factors, sterol regulatory element binding protein-1c (SREBP-1c) has stimulatory effects on the expression of genes involved in lipogenesis. ¹⁸ Insulin was found to stimulate the lipogenesis via activation SREBP-1c. ¹⁸ Therefore, the decrease in lipid parameters can be ascribed to the IR which possibly more in subjects with cirrhosis. ¹⁹ During IR states, AKT2 was involved in the hepatic lipid accumulation, explains one of the etiological factor for lipid accumulation in liver. ¹⁷ The pathophysiology of fatty liver disease was initiated from the deposited fat in hepatocytes followed by the inflammation and oxidative stress. ¹⁶ As the disease advances to liver fibrosis and cirrhosis, inflammation and oxidative stress were involved in the augmentation of the

CONCLUSION

In conclusion, lipid abnormalities exist in patients with liver cirrhosis. The levels of serum total cholesterol, LDL TG and HDL in patients with cirrhosis are inversely related to severity of disease.

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Institutional Ethics Committee

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