

ORIGINAL RESEARCH

A STUDY ON LIPID PROFILES IN CHRONIC LIVER DISEASES

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ABSTRACT

Background: Liver diseases can affect plasma lipid levels in a variety of ways. Chronic liver diseases due to various causes are often associated with dramatic reductions in plasma triglyceride and cholesterol level due to reduced lipoprotein biosynthetic capacity. The study was conducted to determine the lipid profiles in patients with chronic liver disease and to determine if it correlates with the severity of the chronic liver disease.

Materials and Methods: In an analytical cross-sectional study, 120 patients with chronic liver disease (case) and 50 age- and sex-matched healthy patients (controls) were studied. Serum triglyceride levels, total cholesterol, HDL, LDL and VLDL were then measured. Child-Turcotte-Pugh Score were calculated for each patient as an index for the extent of liver damage.

Results: In patients with chronic liver diseases, with the exception of triglyceride level, there was a significant decrease in total cholesterol, LDL, VLDL and HDL cholesterol levels compared to the control group ($p < 0.05$). However, no significant correlation was found between severity of cirrhosis and change in serum lipid levels.

Conclusion: Dyslipidemia exists in patients with liver cirrhosis and screening for the same is important for intervention with appropriate therapy to prevent cardiovascular events.

Keywords: Chronic liver disease; Lipid profile; Child Pughs score

INTRODUCTION

Liver is the principal site for formation and clearance of lipoproteins. It receives fatty acids and cholesterol from peripheral tissues and diet, packages them into lipoprotein complexes and releases these complexes back into the circulation. Hence it is not surprising that liver diseases can affect plasma lipid levels in a variety of ways. Chronic liver diseases due to various causes are often associated with dramatic reductions in plasma triglyceride and cholesterol level due to reduced lipoprotein biosynthetic capacity. Cholestasis is associated with hypercholesterolemia as the major excretory pathway of cholesterol is blocked in this disorder. Apart from the various complications seen in cirrhotic patients, chronic dyslipoproteinemia is one which can lead to alterations in cellular membrane lipids, that result in formation of abnormal RBCs, such as echinocytes, and alterations in membrane function with potential pathophysiologic consequences. Although several studies have been conducted on dyslipidemia in cirrhotics in developed countries, there is a paucity of data in this regard in India. As there is a high prevalence of chronic liver disease in our country, we conducted

this study to determine lipid profile in patients with cirrhosis and to assess if it relates to the severity of chronic liver disease.

MATERIALS AND METHODS

This was a cross-sectional study. The patients were selected from liver clinic at Medical College and Hospital, Kolkata, West Bengal, a tertiary care centre in the state. The study was approved by the ethics committee of the hospital and informed consent was obtained from all the subjects. We included 120 patients suffering from chronic liver disease (CLD) irrespective of etiology in the study period from January to December 2012. We excluded patients suffering from concomitant diseases, which can alter the lipid profiles, like diabetes mellitus, cancer, acute pancreatitis, recent parenteral nutrition and acute gastrointestinal bleeding, renal failure, patients who were on glucose or lipid lowering drugs. The diagnosis of chronic liver disease was based upon clinical features, liver function tests, prothrombin time, ultrasonography, upper gastrointestinal endoscopy and liver biopsy wherever

feasible. Other necessary investigations were done accordingly to find out the etiologies of CLD. A similar group of 50 healthy persons, age and sex matched, served as controls. Serum triglyceride level, total cholesterol, High density lipoprotein (HDL), Low density lipoprotein (LDL), very low density lipoprotein (VLDL) were then measured in all cases and controls by maintaining the standard protocol. Child-Turcotte-Pugh Score¹ were calculated for each patient as an index for the extent of liver damage.

Statistics: -

Data were analyzed by SPSS. χ^2 , one-way analysis of variance (ANOVA) and Student's *t* test were used. A *p* value <0.05 was considered statistically significant.

RESULTS

Total 120 patients with chronic liver diseases of different etiologies (Table 1) were included in the study. Among these etiologies, infection (hepatitis B and C) was the most common (52 patients, 43.33%) followed by Cryptogenic cirrhosis (51 patients, 42.50%). Alcoholic cirrhosis constituted 14.17% of patients (17 patients). Age and sex distribution of the patients were shown on table 2.

Table 1:-Causes of cirrhosis in study patients

Causes of Cirrhosis	Cases (%)
Alcoholic	17 (14.17)
Infective (B plus C)	52 (43.33)
Cryptogenic	51 (42.50)
Total	120 (100)

Table 2: Age and Sex distribution of cases (n= 120)

Age(Yrs)	Male (n=80) (%)	Female (n=40) (%)
<20	0 (0)	1 (2.5)
20-30	4 (5)	1 (2.5)
31-40	10 (12.51)	4 (10)
41-50	31 (38.75)	12 (30)
51-60	23 (28.75)	15 (37.5)
>=61	12 (15)	7 (17.5)

The most affected age group among male patients was 41 to 50 years (38.75%), and female patients of the age group 51 to 60 years were most sufferers (37.5%). Using Child Pugh's criteria, 18 (15%) patients had score A, 58 (48.33%) patients had score B, 44 (36.67%) patients had score C. We found that except triglyceride level, the other four variables (HDL, LDL, VLDL, total Cholesterol) were significantly lower in the study group compared to control group (*P* value<.05) (Table 3).

Table 3: Lipid profiles in cirrhotics and control group

	Control (n=50) (mean±SD)	Cirrhotics (n=120) (mean±SD)	P value
Total cholesterol (mg/dl)	192±21.34	141.5±46.69	<.0001
TG(mg/dl)	137.6±14.36	120.9±96.23	0.06
HDL (mg/dl)	41.78±5.04	33.50±12.78	<.0001
LDL (mg/dl)	122.8±19.29	86.58±35.63	<.0001
VLD (mg/dl)	27.52±2.87	23.53±15.04	.0058

Table 4:-Lipid profile in cirrhotics according to Child criteria

	Patients	LDL(mg/dl)	HDL(mg/dl)	VLDL(mg/dl)	TG(mg/dl)	Total CH(mg/dl)
Child A	18	88.33±40.37	32.78±13.37	21.5±6.18	106.1±30.16	139.8±36.56
Child B	58	89.69±33.36	32.98±11.42	25.22±18.65	132±127.5	146.7±50.38
Child A+B	76	90.31±34.97	33.16±11.86	24.36±16.72	125.8±112.6	146.3±47.16
Child C	44	81.05±36.99	34.09±14.36	22.15±11.93	112.6±58.13	134.1±45.61

All values are expressed as mean±SD

Table 5:- Relationship between lipid profile and severity of liver damage in cirrhotic patients (P values)

Lipid Profile	(A+B) versus C	A versus C
Total cholesterol	0.17	0.60
TG	0.40	0.56
LDL	0.18	0.51
VLDL	0.40	0.78
HDL	0.71	0.73

Lipid profiles in different groups of patients according to child pugh's criteria were shown in table 4. The change in the serum HDL, LDL, VLDL, total

cholesterol and triglyceride had no significant correlation with the severity of liver damage (table 5).

DISCUSSION

Derangement of serum lipid profile is a common observation in cirrhotics. To the best of our knowledge, there are very few studies on dyslipidemia in cirrhosis in India, but this subject has been dealt in detail worldwide. This study was conducted to document any derangement in lipid profile in cirrhotic patients and whether this derangement has any correlation to severity of liver damage. The results of this study showed that all the four studied variables (total

cholesterol, LDL, VLDL, HDL) were significantly low in the study population than in the control group (P value <.05) with the exception of serum triglyceride levels. Triglyceride values also showed a decline in CLD patients but it was not statistically significant.

Hypolipidemia is also seen in various other medical conditions like malnutrition, malabsorption, hyperthyroidism, renal failure, malignancy and immunoglobulin disorders². So we excluded patients suffering from these disorders from our study. One study conducted by Brier C et al³ on lipoproteins in the plasma of patients with post alcoholic liver cirrhosis, showed that in alcoholic cirrhosis, total cholesterol, HDL, VLDL, HDL-cholesterol were all decreased. LDL from cirrhotic patients contained more triglycerides and less esterified and free cholesterol. Selimoglu and colleagues⁴ in their study showed that with the exception of serum triglyceride levels, other variables like serum HDL, LDL level decreased in cirrhotics. This finding has some similarity with our results and hypolipidemia is expected in severe liver disease due to decline in synthetic function. However most of the studies conducted elsewhere showed all the lipid fragments in cirrhotics were lower than in control^{5,6}. Similar studies conducted by Edith N. Okeke⁷ and Mohammad Reza Ghadr⁸ showed significant derangement of lipid level in cirrhotics and a negative relation to extent of liver damage. Salimoghlu⁴ found that HDL level is lower in Child-Pugh B than Child-Pugh A and apo-A level is the most affected factor in those with liver damage. Perales and his colleagues⁹ showed that in chronic liver disease without cholestasis, there was a significant decline in lipid levels with the progression of disease process. This was another major difference with our results. We did not find any significant correlation of progression of liver damage with serum lipid levels. The lipid profile of patients with child A did not differ statistically with those of Child C (P value >.05%). One explanation of this major difference may be due to changes in patient characteristics and etiology of cirrhosis in our study. Cirrhosis was cryptogenic in majority of our patients which might have resulted from non alcoholic fatty liver disease. It is well known that NAFLD and NASH (nonalcoholic steatohepatitis) is associated with increased atherogenic risk and increased cardiovascular mortality from various studies¹⁰. An increase in TG secretion from the liver in the form of LDL is likely responsible for the increase in serum TG concentrations commonly noted in patients with NAFLD¹¹. Another possible cause of insignificant decrease of TG may be alcohol. In our study, alcohol contributed to cirrhosis in around 15% of our study subjects. Alcoholic cirrhosis is associated with increase in TG and LDL levels¹². Furthermore, our study was a hospital based study, which might have introduced some bias in patient selection. Hypolipidemia, in particular decreased HDL-C level is also an important risk factor for cardiovascular disease and vascular events^{13,14}.

CONCLUSION

In conclusion, dyslipidemia exists in patients with liver cirrhosis and screening for the same is important for intervention with appropriate therapy to prevent cardiovascular events. However, further studies are needed to assess the predictive value of dyslipidemia as a tool to forecast the progression of cirrhosis.

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