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STUDY OF LIVER FUNCTION TEST AND LIPID PROFILE IN ALCOHOLIC LIVER DISEASE

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ABSTRACT

BACKGROUND: Alcoholic cirrhosis is quite often associated with impaired lipid metabolism.The aim of the study was to assess the degree of alteration of serum lipid profile in alcoholic cirrhotic patients and also to detect its association with the patients's age and the pattern of alcohol consumption . **METHOD:**This cross-sectional study was conducted in COM &JNM Hospital, Kalyani, Nadia, West Bengal for 1year on 50 cases with history of alcohol consumption. A questionnaire based on history of alcohol intake was made for each patient. LFT and Serum lipid profile (total, low-density lipoprotein (LDL), high-density lipoprotein (HDL) cholesterol and triglyceride) was recorded for each case and control. t test of significance was applied for statistical analysis. **RESULT:** Majority of the cases were in the 40-49 years age group. The study found a definite relationship with the quantity and the duration of alcohol consumption. In patients with cirrhosis, decrease in the total serum cholesterol level was noted. A significant decrease in serum HDL and LDL cholesterol was observed ($P < 0.001$). However, there were significant increase in the serum triglyceride levels in alcoholic cirrhotic patients compared with the control group ($P < 0.001$). **CONCLUSION:**In this study, we found that there was marked alteration of LFT and serum lipid profile values in patients with alcoholic cirrhosis. Therefore, a search for lipid profile abnormality along with LFT should be performed in every cirrhotic patient.

KEYWORDS : Alcohol consumption, alcoholic cirrhosis, serum lipid profile,LFT

INTRODUCTION

Alcohol consumption cause fatty liver, alcoholic hepatitis and ultimately, alcoholic cirrhosis in some patients.[1,2]In Western countries, alcohol is the major cause of liver cirrhosis, and it is gradually increasing in countries like Japan and India.[1] Alcohol-related liver deaths account for up to 48% of cirrhosis-associated deaths in the United States, and are also major contributors to liver disease-related mortality in other countries.[1]

Alcoholic cirrhosis is the end spectrum of alcoholic liver disease (ALD), which includes fatty liver or simple steatosis, alcoholic hepatitis, fibrosis, cirrhosis and superimposed hepatocellular carcinoma.[1]Fatty liver is the most common form of ALD, which occurs in more than 90% of heavy drinkers. But, only about 30% of heavy drinkers develop a more severe form of ALD, such as fibrosis and cirrhosis.[1] As about 30% of the heavy drinkers develop cirrhosis, there are many other factors that are involved in the development of alcoholic cirrhosis, which include sex, obesity, drinking patterns, dietary factors, non-sex-linked genetic factors and cigarette smoking.[3,4]

The liver plays a key role in the metabolism of plasma lipids and lipoproteins.[5] Severe metabolic impairment in cirrhosis can produce a worsening of the serum lipoprotein pattern. High-density lipoprotein (HDL) cholesterol and its major apolipoproteins have been shown to be reduced in cirrhosis, as also the serum levels of low-density lipoprotein (LDL) cholesterol.[6]

Despite the recent advances in the knowledge of alcohol induced liver damage, distance from alcohol and supportive therapy remains the mainstay of management for majority of patients. So it is essential to diagnose alcoholic liver damage earliest because fatty liver is totally reversible and alcoholic hepatitis to some extent on stoppage of alcohol.

It is also known that long-term ingestion of alcohol causes serum lipid profile abnormality.[7]The present study was undertaken with the following aims and objectives:

I. To assess the degree of alteration of serum lipid levels in alcoholic cirrhosis, and

II. To correlate the abnormality observed with the amount and duration of alcohol consumption.

METHOD

The present study was carried out in COM&JNM Hospital, Kalyani from April 2019 to March 2020 , on the patients attending the hospital. A total of 50 patients with alcoholic liver diseases were included in this study. The criteria for inclusion of cases were history of alcoholism($\geq$ 60gm per day) with clinical, biochemical and ultrasonographic evidence of cirrhosis. Detailed history of alcohol intake was taken in every patient.

Clinical signs of cirrhosis include hard and nodular consistency of liver, splenomegaly, ascites, varices and related hemorrhage, spider angiomata, palmar erythema, signs of hepatic encephalopathy, etc.

Biochemical tests including liver function tests were performed, which assisted in the diagnosis of alcoholic cirrhosis. These included serum levels of the enzyme aspartate aminotranferase (AST), alanine aminotransferase (ALT) and gamma glutamyltransferase (GGT). If the ratio of AST to ALT was greater than two in cirrhotic patients, the cause was most likely attributed to alcohol. Elevated GGT levels in blood also indicate heavy alcohol use and liver injury.

All cases were subjected to ultrasonographic examination. Typical imaging findings of alcoholic cirrhosis include hepatomegaly, bluntness of liver edges, irregular liver surface and coarse liver texture[8]. Caudate lobe hypertrophy is also a characteristic morphologic feature of cirrhosis. Computed tomography scan and magnetic resonance imaging were performed only in few cases.

Cases of cirrhosis arising due to reasons other than alcohol were excluded from the study.

Fasting blood samples were sent for lipid profile, both in cases and in controls. Lipid profile estimation was performed using a semi-automated biochemistry analyzer (Model: CHEM-7; ERBA diagnostic Mannheim GmbH-Transasia, Bio-Medicals

Ltd. Transasia House, 8 Chandivali Studio Road, Mumbai - 400 072, India. Total cholesterol was estimated by an enzymatic method (Cholesterol oxidase-Peroxidase), end point, and triglyceride by an enzymatic method (Glycerol phosphate oxidase-Peroxidase), end point. HDL cholesterol was estimated by the phosphotungstic acid precipitation method. LDL cholesterol was determined by Friedewald's equation[9]. Total serum cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride levels were recorded for cases and controls.

STASTICAL ANALYSIS

T test of significance was applied to determine whether the differences observed in the two groups of cases and controls were significant or not. A $P < 0.05$ was considered statistically significant.

RESULT

A total of 50 patients of alcoholic liver diseases were studied. Majority of the patients were in the age group of 40-49years (32%). All cases were male.

Majority of the cases (30%) consumed a daily amount of 81-90 g of alcohol, followed by 20% cases consuming 71-80 g of alcohol and 20% cases consuming 61-70 gm of alcohol.[Table 1]

Table 1:Distribution of alcoholics according to the quantity of alcohol consumption

Quantity (gm)	No. of cases	%
<60	6	12
61-70	10	20
71-80	10	20
81-90	15	30
>90	9	18

We observed that majority of the cases (32%) had consumed alcohol regularly for a period of 9-12 years, followed by 28% cases having consumed alcohol for 13-16 years.[Table2]

Table 2:Distribution of Alcoholics according to duration of Alcohol consumption (in years)

Duration of alcohol consumption in years	Number of cases	%
5-8	11	22
9-12	16	32
13-16	14	28
17-20	8	16
>20	1	2

Raised SGOT was seen in 84%, raised bilirubin 66%, hypoalbuminemia in 62%, raised PT 64% and hyperglobulinemia were seen in 62%. Raised alkaline phosphatase was seen in 34% of cases. Raised SGPT was seen in only 44% of cases.[Table 3]

Table 3: The incidence of various LFT abnormalities

LFT abnormality	Number of cases	%
Hypoalbuminemia (<3gm/dl)	31	62
Hyperglobulinemia(>3.5gm/dl)	31	62
↑SGOT (>50IU/dl)	42	84
↑SGPT (50IU/dl)	22	44
↑Alkaline phosphatise(>350)	17	34
↑PT (>3sec.thancontrol)	32	64
↑Bilirubin (>1.5mg/dl)	33	66

In patients with cirrhosis,there was a significant decrease in serum HDL and LDL cholesterol compared with the control group in all age groups ($P < 0.001$) . However, the serum triglyceride levels were significantly increased in 64% of alcoholic cirrhotic patients compared with the control group ($P < 0.001$).[Fig. 1]

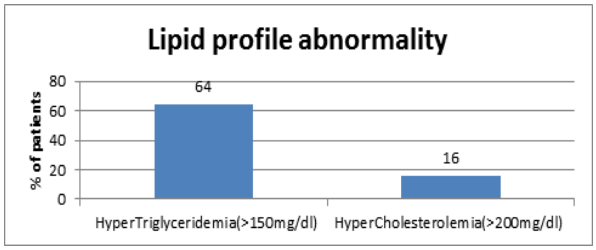

Figure 1: Lipid profile abnormality

DISCUSSION

In the present study, 50 alcoholics who were admitted in COM&JNM Hospital,Kalyani were studied .

Majority of the patients were in the age group of 40-49 years (32%) of age. This correlated with the study by D N Amarapurkar in which the mean age of presentation of alcoholics was 41-51years.[8]Gordon Bekett et al in their study showed that the common age group of presentation of alcoholics was 40-50 years.[9]

Majority of the patients (30%) consumed 81-90gms of alcohol per day. 20% of the patients consumed between 71-80 gms and 61-70gms per day respectively.Lena Van Waes, Charles S Leiber in their study of 100 alcoholic patients, the average alcohol intake was 90-180gms per day.[10]

Majority of the patients (32%) consumed alcohol for a period of 9-12yrs. 28% patients consumed alcohol for 13-16yrs & 0nly 2% of the patients consumed >20 yrs.

Our study correlated with Taorkild et al, who showed that mean duration of alcohol consumption was 10-13yrs[11]

Morgan and Sherlock in their study revealed that mean duration of alcohol intake was 20.4 yrs in men and 16.8yrs in women[12].

Out of 50 patients,7 patients (14%) had normal LFT. Other 43 patients had one or more abnormal LFT. LFT & other biochemical abnormality were also related to dose & duration of alcohol consumption by the patients.

Hyperbilirubinaemia (>1.5mg/dl) was present in 66% of patients where the incidence were 33%, 44%, 87.4% in fatty liver, alcoholic hepatitis & cirrhosis respectively.P-vaiue were 0.001 for both quantity and duration (significant).The Medenhall study showed that bilirubin level elevated in 65% of hepatitis and 90% in cirrhosis.[13]

SGOT was increased (>50IU/dl) in 84%of the patients. Leena Waes et al found SGOT raised in 50% of patients. [10] In this study the incidence of raised SGOT was 50%, 71% and 98 % in fatty liver, hepatitis& cirrhosis respectively. In the other study it was 59%, 95% and50%, in fatty liver, hepatitis& cirrhosis respectively.[14] P-value was <0.001 for both quantity and duration (significant).Abnormal biochemical results in the form of raised SGOT is not specific for fatty liver, hepatitis or cirrhosis. But elevated enzymes point more towards hepatitis and cirrhosis.

The raised SGPT (50IU) was seen in 44% of patients . The incidence of abnormality was 16.6%, 22% 64% in fatty liver, hepatitis and cirrhosis respectively whereas in Lieiber study[15] it was 50%, 95% and 50% in fatty liver, hepatitis and cirrhosis respectively. P-value was <0.001 for both quantity and duration (significant).

Daniel et al in their study, showed distinct patterns of serum SGOT and SGPT elevation. Ratio of SGOT/SGPT greater than 2 is highly suggestive of alcoholic hepatitis or cirrhosis which

is primarily due to diminished activity of SGPT in liver.[16]

In this study SGOT/SGPT >2 was present in 20% patients where the incidence was 16.5%, 64% in hepatitis & cirrhosis respectively and SGOT/SGPT>1 was in100% of patient.

Alkaline Phosphatase was raised (>350u/dl) in 34% patients. The incidence was 32% ,26.6 %, 44% in fatty liver ,hepatitis and cirrhosis respectively. P-value was 0.804 &0.920 for quantity and duration respectively i,e non significant. In the study by Medenhall, alkaline phosphatase was raised in 67% Of alcoholic hepatitis.[13] Elevated ALP more common in hepatitis than cirrhosis.

In this study hypoalbuminaemia (<3.0gm/dl) was observed in 62% of patients. The incidence was 60%, & 76% in hepatitis and cirrhosis respectively. P-value was <0.001 for both quantity and duration (significant). It was not a feature of fatty liver.In the study by Charles Leiber it was 50% in both alcoholic hepatitis and cirrhosis respectively.[17]

Hyper globulinemia (>3.5gm/dl) was observed in 62% of the patients. The incidence was 33%, 60% & 68% in fatty liver, hepatitis and cirrhosis respectively. P-value was <0.001 for both quantity and duration (significant).This correlated with Charles Leiber study in which it is 48% and 72% in hepatitis and cirrhosis respectively.[17]
Altered albumin globulin ratio present in 90% of patients.

P-Time was raised (3 sec. more than control) in 64%of patients. The incidence was 49.5% & 87% in hepatitis and cirrhosis respectively. P-value was <0.001 for both quantity and duration (significant). Prolonged PT is more favour in cirrhosis. Howeveri, it rules out the possibility of fatty liver alone as the diagnosis.In the study by Medenhall, PT was raised in 65% of patients with hepatitis and 90% patients with cirrhosis.[13]

Hyper triglyceridemia (>150mg/dl) was present in 64% of patients.The incidence was 50%, 33% & 87% in fatty liver, hepatitis and cirrhosis respectively. P-value was <0.001 for both quantity and duration (significant). This finding is similar to the results of a study done in India.[18]

In our study, serum total cholesterol values was lower in alcoholic cirrhotic patients compared with the normal, healthy individuals. The serum HDL cholesterol and LDL cholesterol levels was also significantly decreased compared with the normal controls. Dyslipidemia is seen in ALD, which progressively increases from steatosis to hepatitis to alcoholic cirrhosis.[19] Kackar et al. found that the serum cholesterol levels decreases progressively with the progress of alcoholic cirrhosis.[20] Few studies on cirrhosis of liver showed that serum HDL cholesterol, LDL cholesterol and total cholesterol values were significantly diminished.[21,22] In a Nigerian study, the median total cholesterol and HDL cholesterol levels were significantly higher in controls compared with cirrhotic patients; however, LDL cholesterol levels were higher in controls compared with cirrhotic patients and the difference was not statistically significant.[23The serum LDL cholesterol level is inversely proportional to the severity of liver damage and therefore, it is expected that the serum LDL cholesterol would be low in cirrhotic patients.[22] However, alcoholic cirrhosis may be associated with increased total cholesterol and LDL cholesterol levels, as found by Varghese et al.[24]

CONCLUSION

Dyslipidemia and abnormal LFTs are seen commonly in alcoholic cirrhosis. Therefore, alcoholic cirrhotic patients should be routinely screened for LFT & lipid profile abnormality. Further research in this field is justified. This may, in the future, provide a valid relationship between progression of alcoholic cirrhosis and severity of dyslipidemia and altered LFT. Thus, studies of LFT and lipid profile may guide us in the prognosis and treatment of alcoholic cirrhosis in the near future.

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