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Research Article

PATTERN OF LIPID PROFILE CHANGES IN PATIENTS SUFFERING WITH CHRONIC LIVER DISEASE

Manabendra Nayak., Chabukswar Sandip Vithalrao and Rahul Nayak

[1]Teacher of Medicine, National Board of Examination, HOD, Department of Medicine, Down Town Hospital, Guwahati
[2]DNB Medicine, down town Hospital, Guwahati
[3]Department of Microbiology, Assam Down Town University, Guwahati

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ABSTRACT

A malabsorption of lipids frequently occurs in patients with liver disease, not only in those with cholestasis but also in those with parenchymal liver disease. Moreover, lipid synthesis and transfer rates are impaired in cirrhotic patients. They have both impaired FFA synthesis and impaired VLDL production. In cirrhosis, plasma triglycerides are elevated and are carried by LDL rather than VLDL. In advanced liver disease, however, both plasma triglycerides and VLDL levels are reduced**.** In 1862 Austin Flint had suggested that the blood cholesterol level was affected by diseases of the liver. He found that blood cholesterol was raised in three patients with parenchymal liver diseases. Lipid synthesis is decreased in cirrhotics. In cirrhotics, triglycerides are increased and are carried by LDL rather than VLDL. However in advanced cirrhosis of liver, both plasma triglycerides and VLDL are found to be reduced. In patients with cirrhosis, the total serum cholesterol level was decreased. There was a significant decrease in serum HDL and LDL cholesterol. The serum triglyceride levels were significantly increased in alcoholic cirrhotic patients, the amount of decrements measured in the levels of serum total cholesterol; LDL and HDL in patients with cirrhosis are related to the progress in cirrhosis. Further studies are needed to assess the predictive values of measuring lipid profiles as a mean to estimate the extent of liver damage in cirrhotic patients. A total of 50 patients of age group >18 years, diagnosed as Cirrhosis with regards to both history and clinical examination were studied for 12 months in down town hospital, Guwahati, Assam. Lipid profiles of cases were compared with age and sex matched 50 controls patients. The age of the patients ranged from 18 to 60 years, with a mean age of 48.09 ± 9.3 years. Male patients predominated in this study. Maximum numbers of cases were in the age group 41 to 60 years. The commonest cause of cirrhosis in this study was alcoholism, followed by viral hepatitis C. Abdominal distension, fever, yellowish discoloration of eyes/urine & leg were the commonest presenting symptoms in this group of patients. The commonest signs were pallor, pedal edema, icterus and ascitis. Most of the patients in this study showing moderate to severe ascites. Maximum number of patients belonged to Child Pugh Class C. Total cholesterol levels showed highly significant reduction in cirrhosis when compared to controls ($p<0.001$) and showed positive correlation with the stage of Cirrhosis. Triglycerides level showed very significant reduction in cirrhosis when compared to controls ($p<0.01$) and was found to have significant correlation with the severity of Liver Disease. HDL cholesterol levels showed highly significant reduction in cirrhosis when compared to controls ($p<0.001$) and showed positive correlation with the severity of Liver disease. LDL cholesterol levels showed highly significant reduction in cirrhosis when compared to controls ($p<0.001$) and showed positive correlation with the severity of Liver disease. VLDL levels were decreased very significantly when compared to control & showed positive correlation with the severity of Liver disease. There is a graded response of the lipid profile depending on the Grade of Cirrhosis.

INTRODUCTION

Liver diseases have been found to contribute markedly to the global burden of mortality and morbidity. In the 2010 Global Burden of Disease (GBD) study, more than one million deaths (1,030,800 deaths representing 2.0% of all deaths, 1.4% of all deaths of women and 2.4% of all deaths of men) were due to liver cirrhosis.[1] The prevalence of chronic liver disease is high

*Corresponding author: **Manabendra Nayak***
Teacher of Medicine, National Board of Examination, HOD, Department of Medicine, Down Town Hospital, Guwahati

in our country and most common causes are chronic hepatitis C (HCV), alcoholic liver disease, non alcoholic steatohepatitis/ non alcoholic fatty liver disease (NASH/ NAFLD), chronic hepatitis B (HBV), autoimmune hepatitis, sclerosing cholangitis, primary billiary cirrhosis, hemochromatosis, Wilsons disease, Veno-occlusive disease, Drug induced hepatitis.

Alcohol is consumed widely in most parts of the world and has long been identified as a major risk factor for all liver diseases.[2] For specific secondary causes of liver cirrhosis and liver cancer, liver cirrhosis secondary to alcohol use for 2010 was estimated to be 282,800 deaths (representing 19.8% and 19.8% of all liver cirrhosis deaths). The mortality from liver cancer secondary to alcohol for 2010 was estimated to be 149,000 deaths and 3,782,000 Disability adjested life years (DALYs) (representing 27.4% and 27.6% of all liver cirrhosis deaths). [1] NAFLD are now being increasingly recognized as a major health burden. The prevalence of fatty liver in India has been shown to be as high as 15%-30%.[5] Earlier reports indicated that majority of cases of NAFLD are relatively mild and have a benign course. However, now it has been documented that number of these cases can progress to fibrosis, cirrhosis, liver failure and hepatocellular carcinoma and thus contributes to liver related mortality and morbidity.[3, 4]

Liver is the principal site of formation and clearance of lipoproteins. This shows liver is involved in many steps of lipid metabolism and lipid transport. Thus in severe liver disease, lipid metabolism is profoundly disturbed. In chronic liver disease due to decreased biosynthetic capacity of liver unusually low levels of cholesterol and triglycerides is found. [5] In Patients with HBV cirrhosis and HCV related cirrhosis, showed that lower levels of total cholesterol and cholesterol ester, as well as HDL and LDL-cholesterol except VLDL cholesterol and TG levels were influenced by aetiology and severity of chronic viral hepatitis. [6, 7] Patients with alcoholic cirrhosis and post alcoholic liver cirrhosis showed that total cholesterol, LDL, VLDL, HDL-cholesterol were all decreased. [8] Lipid abnormalities in chronic hepatitis, liver cirrhosis and hepatocellular carcinoma showed that the triglyceride and cholesterol levels decreased while LDL-triglyceride fraction increased.[9] LDL from cirrhotic patients contained more triglycerides and less esterified and free cholesterol. Patients with NAFLD the levels of serum triglycerides, total cholesterol, LDL and VLDL cholesterol were raised with low serum HDL levels. [10]

This study aims at studying the pattern of lipid profile changes in patients suffering with chronic liver disease.

METHODOLOGY

It was a Cross Sectional descriptive study in the Department of Medicine, Down Town Hospital, Guwahati, Assam, between May 2013 to April 2014.

This study includes 50 known cases with signs, symptoms & investigations suggestive of chronic liver disease were taken in age group of 18- 60 years irrespective of gender were included in the study & lipid profile was compared with age & sex matched 50 normal control patients. Control patients were randomly selected from down town hospital without history of liver disease & having normal liver function tests. Child pugh score was calculated for each cirrhosis case and they were divided into child pugh class A, B, C & lipid profile was compared within the child pugh classes.

Inclusion criteria

- Patients with age greater than 18 yrs.
- Known and established cases of chronic liver disease diagnosed by ultrasound and biochemical criteria.

Exclusion criteria

- Case that had used oral hypoglycemic agents or insulin within previous 30 days.
- Cases with chronic renal failure.
- Patient who had used lipid lowering agents within previous 30 days.
- Patient with diabetes mellitus, hypertension.
- Patients with other disease likely to result in alteration of lipid levels like nephrotic syndrome, thyroid disorders, cancer were excluded.
- Refusal to participate in the study

Data collection technique

Primary data: History and clinical examination. Patients were asked to sign a written informed consent form.
Secondary data: Systematic reviews and research data.

Tools – Clinical performance

Child Pugh score to assess the severity and prognosis of cirrhosis of liver

Primary Data collection

A hospital based descriptive study was conducted. All patients having age between 18-60yrs having the symptoms and signs of chronic liver disease presenting during the course of hospital stay were evaluated and studied. 50 patients were included in the study according to the above described inclusion and exclusion criteria. Proper Consent was taken and patient identification numbers were given to each patient to protect their identity. Lipid profile and relevant liver function tests were performed in cases & severity of liver cirrhosis has been assessed through Child Pugh score system in the present study. Liver function tests (S. Bilirubin and SGPT) and lipid profile were also estimated in 50 healthy controls consisting of 40 males and 10 females. Lipid profile of 50 cases was determined & compared with 50 normal ages & sex matched normal control patients randomly selected from hospital.

Collection of blood samples

Venous blood samples of the selected cases were taken for liver function tests, blood serum cholesterol, and triglyceride and lipoprotein fractions in morning after 10 hours or overnight fasting in same sitting. Blood samples were collected from the controls for measurement of liver function tests, serum cholesterol, triglycerides and lipoprotein fractions.
The different laboratory tests and methods done in our hospital for this study are:

- **Serum Ammonia-** Dry Bio-chemistry VITROS Method

- **Blood urea-** Dry Bio-chemistry VITROS Method
- **Serum creatinine-** Dry Bio-chemistry VITROS Method
- **Serum Sodium-** Ion Selective electrode method
- **Serum Potassium-** Ion Selective electrode method
- **Serum bilirubin-** Dry Bio-chemistry VITROS Method
- **Serum protein-** Dry Bio-chemistry VITROS Method
- **Serum albumin-** Dry Bio-chemistry VITROS Method
- **Serum globulin-** Dry Bio-chemistry VITROS Method
- **ALP (Alkaline phosphatase)-** Dry Bio-chemistry VITROS Method
- **AST (Aspartate transaminase)-** Dry Bio-chemistry VITROS Method
- **ALT (Alanine transaminase)-** Dry Bio-chemistry VITROS Method
- **HBsAg-** Electro Chemiluminisence
- **HCV-** Electro Chemiluminisence

Lipid Profile

The estimation of Serum Total Cholesterol, HDL, LDL, triglycerides, VLDL by CHOD-PAP: Enzymatic photometric method.[11]

RESULTS AND OBSERVATIONS

A total of 50 patients of age group >18 years, diagnosed as Cirrhosis with regards to both history and clinical examination were studied for 12 months in down town hospital, Guwahati, Assam. Fifty controls were selected randomly from patients admitted for other ailments not included in the exclusion criteria. Lipid profile of cases was compared with age and sex matched 50 controls patients.

The study shows the age incidence ranged from 20 years to 80 years. Cirrhosis was seen predominantly in older age group with 90% of patients >40 years, of which 76 % were in between 41 and 60 years of age. Distribution of patients according to Child-Pugh Score, the majority of the patients 48 % were found in Class C followed by 34 % in Class B and the remaining 18 % in Class A. Study showing alcohol was most common cause of cirrhosis (80%) followed by hepatitis C (10%) and hepatitis B (6%). 4 % cases were cryptogenic. Most common presenting symptoms were abdominal distension followed by fever, yellowish discoloration of eyes/urine & leg swelling. Most common sign's were pallor (66 %) followed by icterus (60 %) and pedal edema (56 %). Liver cell failure was seen in 50 % cases. About 76% of ascites in the patients were of grade 2 and 3, while encephalopathy was maximum of 46 % in grade 1.

Table 1 Showing liver function test in cases and controls.

Liver function tests	Cases (N- 50)		Control (N-50)		Statistical Analysis Unpaired t test
	Mean	Standard deviation	Mean	Standard deviation	
Total bilirubin	4.37	3.22	0.61	0.28	8.22, p<0.001
SGPT	70.44	50.34	36.06	8.45	4.76, p<0.001

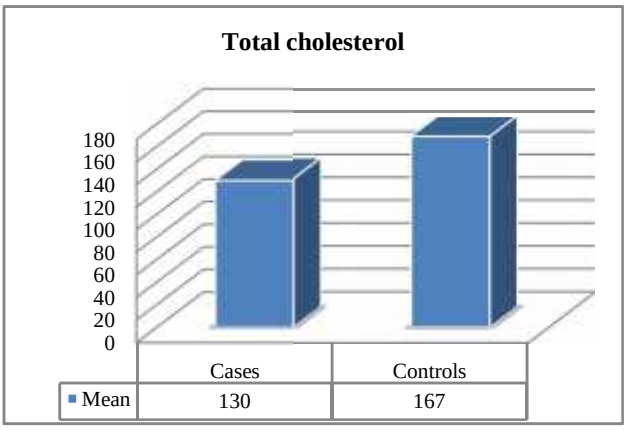
Jaundice was seen in 50 % of cases. Mean Bilirubin level was 4.37 mg/dl. proportionately liver enzyme was also found to be elevated in cases i.e. Mean SGPT was 70.44 U/dl.

Table 2 Showing Lipid profile in cases and controls

Lipid profile	Cases (N- 50)		Controls (N-50)		Statistical Analysis Unpaired t test
	Mean	Standard deviation	Mean	Standard deviation	
Total cholesterol	130.64	19.51	167.08	13.32	10.92 P <0.001
Triglycerides	87.34	16.57	97.38	16.16	3.06 P<0.01(0.002)
HDL	34.14	5.50	41.2	7.20	5.51 P<0.001
LDL	78.03	11.38	106.39	15.69	T 10.34 P<0.001
VLDL	17.46	3.31	19.46	3.21	T 3.06 P<0.01(0.002)

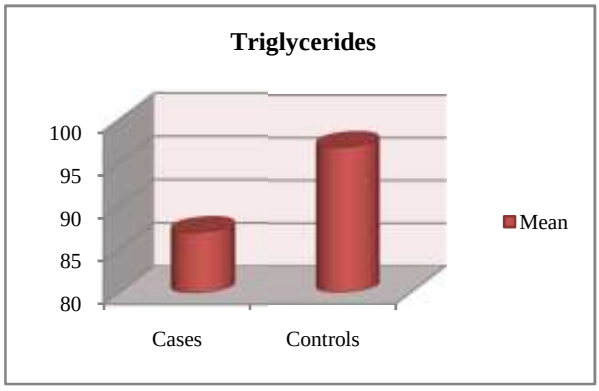
*(p < 0.05) significant, ** (P < 0.01) very significant, *** (P<0.001) indicates that highly significant and Rest are not significant (p>0.05)

It is evident from table 2 that the levels of total cholesterol, triglycerides, HDL, LDL & VLDL were significantly reduced in cases when compared to control group & it was statistically significant (p <0.05).



Graph 1 Showing comparison of Total cholesterol in cases and controls

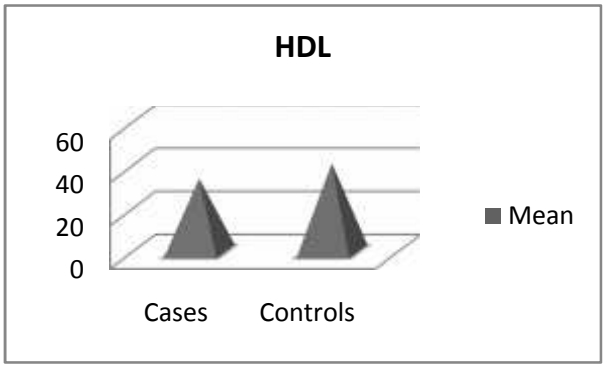
The level of total cholesterol among subjects (130.64 ± 19.51) was significantly lower when compared to the control group (167.08 ± 13.32) i.e. decrease in total cholesterol was highly significant (p<0.001).



Graph 2 Showing comparison of Triglycerides in cases and controls

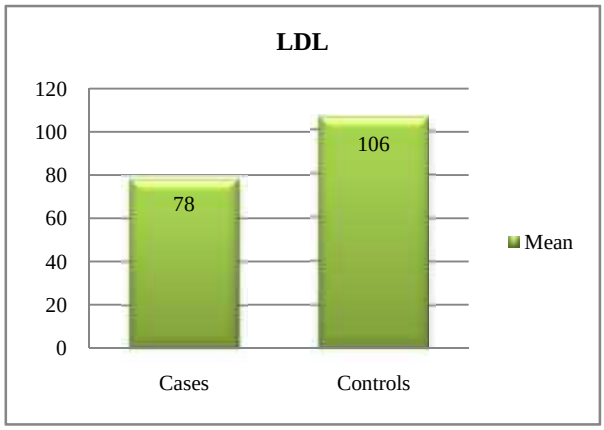
The level of triglycerides among subjects (87.34± 16.57) was significantly lower when compared to the control group

(97.38± 16.16). This decreased in LDL levels in subjects with cirrhosis was very significant (p<0.01).



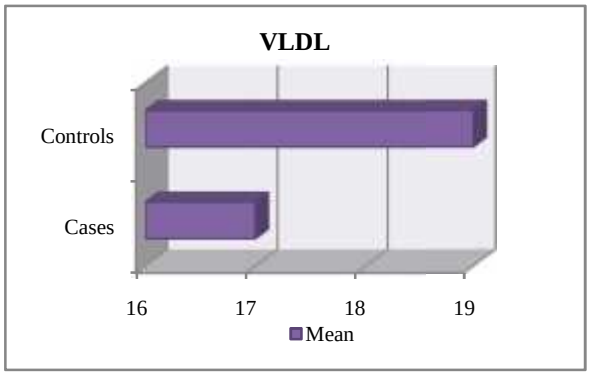
Graph 3 Showing comparison of HDL in cases and controls

The levels of HDL among subjects (34.14± 5.50) were significantly lower when compared to the control group (41.2± 7.20). This decrease in HDL levels in subjects with cirrhosis was highly significant (p<0.001).



Graph 4 Showing comparison of LDL in cases and controls

The levels of LDL among subjects (78.03± 11.38) were significantly lower when compared to the control group (106.39± 15.69). This decrease in LDL levels in subjects with cirrhosis was highly significant (p<0.001).



Graph 5 Showing comparison of VLDL in cases and controls

The levels of VLDL among subjects (17.46± 3.31) were significantly lower when compared to the control group (19.46± 3.21). This decrease in VLDL levels in subjects with cirrhosis was highly significant (p<0.01).

Table 3 Showing lipid profile in child pugh class

Lipid profile	CHILD PUGH CLASS						One way Analysis of Variance (ANOVA) F value & sig.
	Class A (NO-9)		Class B (NO-17)		Class C (NO-24)		
	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation	
Total cholesterol	145.44	18.08	134.35	18.42	122.45	17.20	6.03 P< 0.0046
Triglycerides	96.88	20.43	91.82	15.78	80.58	12.98	4.73 0.013
HDL	38.1	6.001	36.96	4.789	32.73	4.90	5.32 0.0082
LDL	87.96	8.75	79.02	11.35	73.6	10.0	6.49 0.0032
VLDL	19.37	4.08	18.36	3.15	16.1	2.57	4.81 0.012

Comparison was done using ANOVA (Analysis of variance test) *(p < 0.05) significant, ** (P < 0.01) very significant, *** (P<0.001) indicates highly significant & (p>0.05) not significant.

The levels of all lipid parameters total cholesterol, triglycerides, HDL, LDL & VLDL in subjects was significantly reduced in Child Score C compared to B and B compared to A i.e. decrease in Lipids in patients with Cirrhosis was proportional to Child class.

The levels of total cholesterol, HDL & LDL showed very significant decrease (p**<0.01)** & triglycerides & VLDL showed significant decrease (**<0.05)** proportional to Child class.

Table 4 Showing lipid profile changes in males

Lipid profile	Cases Male (N- 42)		Control Male (N-40)		Statistical Analysis Unpaired t test
	Mean	Standard deviation	Mean	Standard deviation	t & p value
Total cholesterol	130.66	19.58	166.32	13.11	9.64 <0.001
Triglycerides	87.04	16.02	96.05	15.06	2.62 <0.05 (0.01)
HDL	35.19	5.60	40.89	6.86	4.11 <0.001
LDL	78.06	11.41	106.21	15.66	9.26 <0.001
VLDL	17.4	3.20	19.19	2.98	2.6 <0.05 (0.01)

Cases were compared with control among males in the above table-4. Male cases showing highly significant reduction in the levels of Total Cholesterol, HDL, LDL (p<.001) & significant reduction in triglycerides & VLDL levels (<0.05) when compared to control males.

Table 5 Showing lipid profile changes in Females

Lipid profile	Cases female (N- 08)		Control female (N-10)		Statistical Analysis Unpaired t test
	Mean	Standard deviation	Mean	Standard deviation	t & p value
Total cholesterol	130.5	20.52	170.1	14.47	4.8 <0.001
Triglycerides	88.87	20.39	102.7	20.02	1.44 0.16 (NS)
HDL	34.66	5.45	42.43	8.75	2.3 <0.05 (0.035)
LDL	77.85	12.03	107.13	16.60	4.17 <0.001
VLDL	17.77	4.07	20.53	4	1.4 0.16 (NS)

Cases were compared with control among females in the above table-5. Female cases showing highly significant reduction in the levels of Total Cholesterol, LDL (p<.001) & significant reduction in HDL levels (<0.05). Levels of Triglycerides & VLDL were not significantly reduced.

Table 6 Showing studies comparable to our study

Study	Total cholesterol	Triglycerides	HDL	LDL	VLDL
Spósito et al (1997)	134 ± 38	80 ± 35	25 ± 15	93 ± 28	16 ± 7
Vargese 2007	135.6 ± 50.50	86.56 ±41.32	33.51 ±10.20	72.09 ±45.58	14.59 ±12.03
Gadir 2010	138.9	82.2	40.7	80.5	-
Subhan 2012	140.6	84.2	34.7	82.5	-
Mandal et al 2013	141.5±46.69	120.9±96.23	33.50±12.78	86.58±35.63	23.53±15.04
Jagannatha (2013)	132 ± 12.6	79 ± 6.8	39 ± 2.3	80 ±12.3	-
Nangliya et al (2015)	141.06±22.64	118.52±24.88	34.54±4.50	82.81±13.17	-
Mohapatra (2015)	141.5±46.69	94.3±42.3	33.50±12.78	78.48±24.24	23.53±15.04

DISCUSSION

Chronic liver disease affects people in their most productive years of life and has a significant impact on the economy as a result of premature death, illness, and disability. [12] Earliest data regarding lipid levels in cirrhosis was available in 1862 when Flint *et al* had suggested that the blood cholesterol level was affected by the liver diseases. [13] Dyslipidemia in liver disease was studied earliest by Evelyn *et al* in 1944 which suggested liver diseases affecting liver parenchyma serum lipids tend to decrease. [14]

In this study cirrhosis was seen predominantly in older age group with 82% of patients >40 years, of which 76 % were in between 41 and 60 years of age. This may be due high prevalence of undiagnosed cirrhosis in both NASH and hepatitis. Compensated cirrhosis often remains undetected for prolonged periods of time & delayed presentation of decompensated cirrhosis due to various etiologies. This is supported by the epidemiological studies done Clark *et al* and Poynard *et al*. [12, 15] Poynard *et al* extend their discussion to quote that three independent factors were associated with an increased rate of fibrosis progression in HCV patients: age at infection older than 40 years, daily alcohol consumption of 50 g or more, and male sex. [12] The value of serum total cholesterol was significantly lower in patients with cirrhosis when compared to controls in our study. (p <0.001) This observation supported by the studies done by Spósito *et al*, Mandal *et al*. [16, 17] Miller *et al* found that in cirrhosis without cholestasis, the patients are not usually significantly hyperlipidaemic and in advanced cases cholesterol and apo B levels may be reduced. Although LCAT activity and the proportion of plasma cholesterol esterified may also be markedly reduced. [5, 18]

Comparison of the total cholesterol values in different Child Pugh Classes showed a direct relation between the severity of Liver damage and reduction in the cholesterol level. This was supported by observations in study conducted by Spósito *et al*, Perales *et al*. [16, 19] They suggested that Cholesterol and lipid phosphorus are normal or subnormal in most patients with portal cirrhosis & with increase in parenchymal damage there is decrease in lipid phosphorus which is positively correlated with total cholesterol. The most frequent disorder of serum lipids in liver diseases is an increase of the ratio of free to total cholesterol. [14, 16] D'Arienzo *et al*, Jiang, *et al*, found in their study that a low serum cholesterol level is associated with a higher mortality rate in patients with liver cirrhosis.[20, 21] Janicko *et al* suggested that the total cholesterol level was a significant marker of mortality, independent of other predictive measures such as INR, bilirubin, creatinine, MELD & can be used to improve the predictive accuracy of MELD score.[22] Triglycerides levels were significantly lower in patients with cirrhosis when compared to controls in our study. (p <0.01) This was supported by studies done by Spósito *et al*, Ghadir *et al*. [16, 23] Zanatta et found that triglycerides were normal in hepatocellularcarcinoma and cirrhosis patients, this could be probably due to difference in the stages in cirrhosis and etiology.[24, 25] It is well known that NASH and Alcoholic cirrhosis is associated with increase in serum TG concentrations.[26] An increase in TG secretion form the liver in the form of LDL is likely responsible for the increase in serum TG concentrations commonly noted in patients with NAFLD.[27] Mandal *et al* didn't find significant difference in triglycerides in viral cirrhosis and control group.[17] This finding was contradicted by our study. The mechanism responsible for reduction of triglyceride level in patient with cirrhosis could be that the metabolism of free fatty acids might be reduced in cirrhotics due to decreased reserve of liver parenchyma as suggested by Evelyn and McIntyre *et al*.[14, 28] Poor nutrition, altered metabolism and abstinence from alcohol of cirrhosis patients may explain the lower triglyceride in cirrhosis in them. In our study we found that the level of reduction of the triglyceride was proportionate to the severity of the parenchymal liver disease (p<0.001). This was supported by the studies done by Perales *et al* and Varghese *et al* [19, 29] However Gadir *et al*, Subhan *et al* didn't find proportional decrease in triglycerides level according to severity of liver damage, [97, 30] this is probably due to the fact that in late stages of hepatic disease, the activity of TG lipase (the enzyme responsible for hydrolyzing ester linkages of TGs) is severely impaired.[6] The level of serum HDL in our study was significantly decreased in cases of Cirrhosis when compared to control (p<.001), are consistent with studies done by Spósito *et al*, Ghadir *et al*.[16, 23] The decrease in HDL in patients with cirrhosis can be attributed to decreased hepatic synthesis of HDL. This could be due to LCAT deficiency as liver is the only source of this enzyme (LCAT) and serum levels of this enzyme are decreased in liver disorders. [18, 31] We also found that the levels of HDL reduction were proportional to the severity of liver damage in cirrhosis. This HDL reduction is also suggested by Perales *et al*, Spósito *et al*.[16, 19] This is explained by the proportional decrease in serum levels of apoA I & apoA II according to increase in severity of liver damage.[24, 32] There was a significant decrease in levels of serum LDL in patients with cirrhosis, when compared to controls (p<0.001) in our study. This is in accordance with previous study by Spósito *et al* and Ghadir *et al*, [16, 23] We also found that the levels of LDL reduction were proportional to the severity of liver damage in cirrhosis. This HDL reduction is also suggested by

Spósito *et al* and Perales *et al*. [16, 19] The levels of VLDL in patients with cirrhosis decreased significantly when compared to controls in our study ($p<0.01$).This is in accordance with studies conducted by Mandal *et al* and Ghadir *et al* [17, 23] Studies by Perales J *et al* in 1994 had observed that there was significant decrease in VLDL cholesterol and apo E levels in patients with severe chronic liver disease when compared to controls. He suggested that the reduction was probably as a result of a defect in their synthesis.[19] VLDL also showed declining levels with progressive increase severity of liver disease. This is supported by studies done by Spósito *et al* and Perales *et al*. [16, 19]

CONCLUSION

Cirrhosis of liver is one of frequently encountered disease causing significant morbidity & mortality. Since compensated cirrhosis often goes undetected for prolonged periods of time, early diagnosis may result in reversal in fibrosis if cirrhosis is caused by hepatitis C & hemochromatosis & management of complications, results in improved management, quality of life and life expectancy. Dyslipidemia is common in cirrhosis & it is decline in lipid levels is negatively correlated with severity of liver damage in previous studies. This study was undertaken to study lipid profile in cirrhosis & there by to assess the clinical utility of the estimation of lipid and lipoprotein levels as a sensitive indicator of extent of liver damage. We found highly significant reduction in levels of total cholesterol, HDL & LDL & in cirrhotic patients when compared with normal controls. Triglycerides & VLDL showed very significant reduction compared to controls. All lipid parameters total cholesterol, triglycerides, HDL, LDL & VLDL showed a graded response to the severity of liver disease among the cases. There are many different biochemical tests are available for diagnosing and assessing the severity of liver cell damage. But these tests lack the desired sensitivity and specificity for assessing the prognosis of the patients. Thus serum lipid profile may serve as a sensitive indicator of severity in cirrhosis of liver.

Reference

1. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, *et al*. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012; 380: 2095–2128.
2. Rush B. An inquiry into the effects of ardent spirits upon the human body and mind: with an account of the means of preventing, and of the remedies for curing them. 8th ed. Exeter (NH): Richardson; 1785; Reprint.
3. Whey LC, Ping YL, Ching TC, Hamid JM, Siong LW. Prevalence of ultrasound diagnosed nonalcoholic fatty liver disease among rural indigenous community of sarawak and its association with biochemical and anthropometric measures. *Southeast Asian J Trop Med Public Health* 2013; 44(2): 309-317.
4. Baffy G, Brunt EM, Caldwell SH. Hepatocellular carcinoma in non-alcoholic fatty liver disease: an emerging menace. *J Hepatol* 2012; 56: 1384-1391.
5. Miller JP, Dyslipoproteinaemia of liver diseases. *Baillieres Clin Endocrinol Metab*. 1990 Dec; 4(4):807-32.
6. Hiraoka H, Yamashita S, Matsuzawa Y, Kubo M, Nozaki S, Sakai N, Hirano K, Kawata S,Tarui S: Decrease of hepatic triglyceride lipase levels and increase of cholesteryl ester transfer protein levels in patients with primary biliary cirrhosis: relationship to abnormalities in high-density lipoprotein. *Hepatology* 1993; 18: 103–110.
7. Vere CC, Neagoe D, Streba CT, Prejbeanu I Ianosi G, Comanescu V, Pirici D: Steatosis and serum lipid patterns in patients with chronic viral hepatitis: differences related to viral etiology. *Rom J Morphol Embryol* 2010; 51: 509–514
8. Breier C, Lisch HJ, Braunsteiner H. Lipoproteins, HDL apolopoproteins, activities of hepatic lipase and lecithincholesterol acyltransferase in the plasma of patients with postalcoholic end-stage liver cirrhosis. *Klin Wochenschr*. 1983 Sep 15; 61(18):929-31.
9. Ooik, Shiraki K, Sakurai Y, Morishita Y, Nobori T. Clinical significance of abnormal lipoprotein patterns in liver diseases. *Int J Mol Med*. 2005; 15:655-660.
10. Agrawal R, Mishra S, Dixit VK, Rai S. Association of non-alcoholic fatty liver disorder with obesity. *Indian J Prev Soc Med* 2009; 40:126-129.
11. Deeg R, Ziegenhorn J. "Kinetic enzymatic method for automated determination of total cholesterol in serum". *Clin. Chem*. 1983; 29:1798-802.
12. Poynard T, Bedossa P, Opolon P; Natural history of liver fibrosis progression in patients with chronic hepatitis C. The OBSVIRC, METAVIR, CLINIVIR, and DOSVIRC groups. *Lancet*. 1997 Mar 22; 349(9055):825-32.
13. Flint A. Experimental researches into a new excretory function of the liver. *Am J Med Sci* 1862; 44: 305-65.
14. Evelyn B. Man, Bernard L. Kartin, Stanley H. Durlacher, John P. Peter. The lipids of serum and liver in patients with hepatic diseases. *J Clin Invest* 1945; 24 : 623-43.
15. Clark JM; The epidemiology of nonalcoholic fatty liver disease in adults. *J Clin Gastroenterol*. 2006 Mar;40(3 Suppl 1):S5-10
16. A.C. Spósito, C.G. Vinagre, F.L. Pandullo, S. Mies, S. Raia and J.A.F. Ramires1Apolipoprotein and lipid abnormalities in chronic liver failure, *Brazilian Journal of Medical & Biological Research* 30(11) 1997.
17. Mandal SK, Koelina Sil, Chatterjee S, Ganguly J, Chatterjee K, Pankaj Sarkar *et al*. A Study on Lipid Profiles in Chronic Liver Diseases. *Natl J Med Res*. 2013; 3(1): 70-72.
18. Sasso GF, Ceccanti M, NardiE *et al*. Lecithin: cholesterol-acyltransferase (LCAT) activity in alcoholic liver disease. *Panminerva Med*. 1989 Jan-Mar; 31(1):30-3.
19. Perales J, Angel Lasuncion M, Cano A, Martin-Scapa MA, Maties M & Herrera E (1994). Changes in the lipid profile in chronic hepatopathies. *Medical Clinics of North America*, 102: 364-368.

20. D'Arienzo A, Manguso F, Scaglione G, Vicinanza G, Bennato R, Mazzacca G. Prognostic value of progressive decrease in serum cholesterol in predicting survival in Child-Pugh C viral cirrhosis. *Scand J Gastroenterol*. 1998; 33(11):1213-8.
21. Jiang JT, Xu N, Zhang XY, Wu CP. Lipids changes in liver cancer. *J Zhejiang Univ Sci B*. 2007; 8(6):398-409.
22. Janicko M, Veseliny E, Lesko D, Jarcuska P. Cholestrol predicts cirrhosis mortality. Annals of Hepatology 2013; 12(4): 413-419.
23. Mohammad Reza Ghadir, Ali Akbar Riahin, Abbas Havaspour, Mehrdad Nooranipour, Abbas Ali Habibinejad. The Relationship between Lipid Profile and Severity of Liver Damage in Cirrhotic Patients. *Hepat Mon*. 2010; 10(4): 285-288.
24. Vergani C, Trovato, Delu, Pietrogrande, Diguardi N. Serum total lipids, lipoproteins, Cholestrol and apolipoprotein A - in acute viral hepatitis and chronic liver disease. *Journal of Clinical Pathology*, 1978; 31: 772-778.
25. Gabriela Zanatta PORT, Kalinca OLIVEIRA, Jonathan SOLDERA and Cristiane Valle TOVO. Biochemical Nutritional Profile of Liver Cirrhosis Patients With Hepatocellular Carcinoma. *Arq. Gastroenterol.* [online]. 2014, vol.51, n.1, pp. 10-15.
26. Valerio, Nobili V, Alkhouri N, Bartuli A, Manco M, Lopez R, Alisi A, Feldstein AE. Severity of Liver Injury and Atherogenic Lipid Profile in Children with Non-alcoholic Fatty Liver Disease. *Pediatric Research* .2010; 67: 665–670.
27. Elisa Fabbrini, Shelby Sullivan, and Samuel Klein. Obesity and Nonalcoholic Fatty Liver Disease: Biochemical, Metabolic and Clinical Implications. *Hepatology*. 2010; 51(2): 679–689. doi:10.1002/hep.23280.
28. Mclntyre N. *et al*. Plasma lipids and lipoproteins in liver diseases. *Gut* 1978; 19: 526-30.
29. Joye S. Varghese, Kolandaivel K, Ragul Upadhuyay, Revathy SM, Jayanthi V. Lipoprotein profile in cirrhosis of liver. European *Journal of Gastroenterology & Hepatology* 2007, 19:521–522.
30. Sushma B Jagannatha, Nagarajappa K & Mallikarjuna. C.R. Serum Paraoxonase-1 activity, Oxidative stress & Lipid profile in patients with Chronic liver disease *International Journal of Pharmacy and Biological Sciences* Volume 3| Issue 1 |JAN-MAR |2013|01-06.
31. Mandenhall CL *et al*. Alterations in serum triglyceride level in liver disease. *Gastroenterol* 1962; 14: 684-85.
32. Cordova C, Musca A, Violi F, Alessandri C, Iuliano L: Apolipoproteins A-I, AII and B in chronic active hepatitis and in liver cirrhotic patients. *Clinica chimica acta; international journal of clinical chemistry* 1984, 137(1):61-66.

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