



**Research Article**

**STUDY OF LIPID PROFILE IN PATIENTS WITH CHRONIC LIVER DISEASE**

**RavikantSoni, \*BushraFiza, Maheep Sinha and Jai Shree Choudhary,**

Biochemistry, Mahatma Gandhi Medical College & Hospital, Jaipur

**ARTICLE INFO**

**Article History:**

Received 15<sup>th</sup> October, 2019

Received in revised form 7<sup>th</sup>

November, 2019

Accepted 13<sup>th</sup> December, 2019

Published online 28<sup>th</sup> January, 2020

**Key words:**

Lipid profile, plasma lipids, triglycerides, Liver

**ABSTRACT**

Chronic liver disease is a disorder that presents with progressive destruction of liver tissue. Liver has a central role in body metabolism and performs some very important metabolic functions. It has a key role in lipid metabolism including synthesis, storage, break down as well as lipid disorders. Lipids are an important component of biological membranes and have other functions like steroid hormone synthesis etc. The present study was planned to evaluate the various lipid profile components in patients diagnosed with chronic liver disease. Fifty patients diagnosed with chronic liver disease were enrolled for the study. Fifty age and gender matched individuals constituted the control group. For all subjects thus enrolled, fasting blood samples were collected and evaluated for serum lipid profile. Results obtained were later subjected to statistical analysis. It was observed that CLD patients had lower concentration of lipid components as compared to healthy control group. The study recommends regular screening of lipid profile and to identify more biochemical markers for evaluation of the severity of disease.

Copyright©2020 **RavikantSoni et al.** This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

**INTRODUCTION**

Chronic liver disease (CLD) is a clinical condition in which progressive destruction and regeneration of liver parenchyma leads to fibrosis and further cirrhosis. The common symptoms of CLD are jaundice, fatigue, nausea, poor appetite, abdominal distension and intestinal bleeding (Khare S *et al*, 2015). Several etiological factors can lead to development of CLD. These include alcoholism, portal hypertension, Autoimmune, Hepatitis B, C and others (Radicheva MP *et al*, 2018).

A range of different radiological and blood investigations are helpful in detection and diagnosis of various hepatobiliary abnormalities. These investigations also help in identifying the basis of clinically suspected disease of liver and to figure out the severity of liver disease (Al-Jumaily EF *et al*, 2012). For predicting prognosis of end-stage liver disease and severity, various prognostic models are recommended. Child Pugh (CP) score and Model for end stage liver disease are the generally prognostic scores. Lipids are essential component of biological membranes, free molecules and metabolic regulators which control homeostasis and cellular function (Chiang JY *et al*, 2005). Liver is a major organ which plays a crucial role in lipid metabolism & several stages of lipid synthesis and transportation. Lipoproteins, endogenous lipids and apolipoproteins are synthesised in liver, liver is the site where 80% cholesterol is synthesised from hepatocellular microsomes, as serum lipid panel expect to be abnormal during the progression damage of liver (Jarikre AE *et al*, 1996).

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 (Poynard T *et al*, 1997 and Bellentani S *et al*, 1999). Cirrhosis is an advanced stage of liver fibrosis that is accompanied by distortion of the hepatic vasculature. Frequently multiple etiological factors contribute to the development of cirrhosis, as exemplified in epidemiological studies that identified regular (moderate) alcohol consumption, age above 50 years, and male gender as risk factors in chronic hepatitis C or older age obesity, insulin resistance/type 2 diabetes, hypertension and hyperlipidemia (all features of the metabolic syndrome) in NASH (Bellentani S *et al*, 1997, Clark JM 2006 and Farrell GC *et al* 2006).

The present study was planned compare compoaints of lipid profile in CLD patients healthy controls.

**METHODOLOGY**

**Study Design**

This prospective study was conducted in the Department of Biochemistry in collaboration with Department of Gastroenterology at Mahatma Gandhi Medical College and Hospital, Jaipur after seeking approval from the Institutional Ethics Committee (IEC).

Fifty diagnosed patients of CLD, age 20 to 65 years, either gender, were enrolled for the study. Pregnant and lactating females and patients with acute liver disease and malignancies were excluded.

Fasting blood samples were collected for all enrolled subjects and control and evaluated for serum Lipid profile including

\*Corresponding author: **BushraFiza**

Biochemistry, Mahatma Gandhi Medical College & Hospital, Jaipur

serum Cholesterol, serum triglycerides (TG), High density lipoprotein (HDL), Low density lipoprotein (LDL) and Very low density lipoprotein (VLDL).

Lipid profile

- Serum Cholesterol -(Enzymatic method)
- Serum Triglycerides -(Enzymatic method)
- High density lipoprotein (HDL) -(Phosphotungstic acid method)
- Low density lipoprotein (LDL) -(Calculated by Cholesterol-{HDL+VLDL})
- Very low density lipoprotein (VLDL) -(Calculated by Triglycerides/5)

Observation

	Control group (n=50)	Patient group (n=50)	t-value	p-value
Cholesterol (mg/dL)	150.06 ± 26.92	112.15 ± 43.01	5.283	0.0001
Triglycerides (mg/dL)	128.48 ± 24.35	113.44 ± 28.61	2.831	0.006
HDL (mg/dL)	43.67 ± 8.62	29.31 ± 14.63	5.980	0.000
LDL (mg/dL)	80.70 ± 26.42	60.16 ± 35.96	3.255	0.002
VLDL (mg/dL)	25.69 ± 4.87	22.69 ± 5.72	2.824	0.006

	No. of participants	Percentage
Child Pugh A	4	8%
Child Pugh B	26	52%
Child Pugh C	20	40%

RESULT

In the present study mean age in control group (47.85 ± 12.77 years) and CLD patient group (48.18 ± 11.89 years) was comparable. Majority of CLD patients were categorized as CP grade B (52 %) or C (40%). Only 8% of the patients were grouped as CP grade A. All components of lipid profile were significantly lower in the CLD patients as compared to healthy controls.

DISCUSSION

In this present study comparison of lipid profile including serum Cholesterol, TG, HDL, LDL and VLDL in between liver cirrhotic patients and control group was done. It is observed after comparison that low levels of serum cholesterol, TG and HDL were found in cirrhotic patient with the progression of liver disease. Hypercholesterolemia and hypertriglyceridemia are strongly associated with the progression of liver disease.

A reduction in TC serum levels is believed to be a consequence of decreased synthesis or partial blockage of the same esterification processes, likely due to a decline in the production of the enzyme ACAT (acyl CoA: cholesterolacyl transferase). Decreased VLDL levels are associated with deficiencies in the microsomal triglyceride transfer protein (MTP) and a partial inhibition of cholesterol synthesis (Habib A *et al*, 2005 and Jiang M *et al*, 2010). The formation of LDL is directly related to the production of VLDL and, when the metabolism of this lipoprotein is impaired, the other downstream lipid fractions also undergo changes. The drop in HDL levels suggests that there is a strong correlation between prognosis and decreased synthesis of Apoprotein AI (Apo AI), the major HDL lipoprotein (Mirandola S *et al*, 2010, Nashaat EH *et al*, 2010 and Petit JM *et al*, 2003).

Sachdeva S *et al*, 2018 showed in their study that mean of serum total cholesterol in cirrhotic study group was 147.29+17.14 mg/ dl and in control group was 163.86+17.63 mg/dl. Mean of total cholesterol was higher in control group as compare with control group as it was statistically significant as p value <0.05. Another study by Nangliya VJ *et al*, 2015 presented similar findings that mean of serum total cholesterol in cirrhotic study group was 141.06+22.64 mg/dl and in control group was 175.69+16.41 mg/dl, statistically significant as p value is <0.05. LDL cholesterol in patient group was 82.81+13.17 mg/dl and in control group was 107.28+9.04 mg/dl.

Mandal SK *et al*, 2013 reported in their study that serum total cholesterol in patient group was 141.5+46.69 mg/dl and in control group was 192+21.34 mg/dl. Mean of serum total cholesterol was observed higher in control group as compared to patient group, that was statistically significant as p value <0.05 as same with the mean of LDL cholesterol in cirrhotic study group was 83.55+16.08 mg/dl and in control group was 92.88+17.15 mg/dl. Mean of LDL cholesterol was higher in control group than patient group that was statistically significant as p value 0.0014.

Chrostek L *et al*, 2014 observed in their study that the concentrations of lipids and lipoproteins in the liver diseases are deranged. The mean concentrations of cholesterol, HDL-cholesterol, and LDL-cholesterol were significantly decreased in liver cirrhosis of both origin (alcoholic and non-alcoholic). Triglycerides-rich lipoproteins comprise very low-density lipoprotein (VLDL) and chylomicrons. The assembly and secretion of VLDL particles take place in the liver cells and both elements, apolipoprotein B and microsomal triglyceride transfer protein (MTP), are necessary for these processes. It has been shown that MTP plays a role in transferring lipid to nascent apolipoprotein B and hepatic induction of MTP, resulting in a reduction in hepatic TG accumulation and improvement of VLDL export, which increases the serum level of TG (Gordon DA *et al*, 2000 and Shindo N *et al*, 2010).

plasma lipids and lipoproteins tend to decrease with parenchymal liver disease, and the level and composition of the lipoproteins depends on the activity of enzymes involved in lipid metabolism. These include lipoprotein lipase (LPL), lecithin-cholesterol acyltransferase (LCAT), and hepatic triglyceride lipase (HTGL) (McIntyre N 1978 and Sabesin SM *et al*, 1980).

Ghadhir MR *et al*, 2010 showed that in patient their was a significant decrease in serum triglyceride, total, LDL and HDL cholesterol levels compared to the control group (mean of 82 vs 187, 138 vs 184, 80 vs 137, and 40 vs 44 mg/dL), respectively; all p= <0.05.

Boemeke L *et al*, 2015 concluded their study that hypercholesterolemia contributes to the evaluation of the severity of liver disease, due to the association between the reduction of cholesterol and the other lipid profile components as TG, HDL, LDL and VLDL.

CONCLUSION

There is a significant decrease in serum lipid profile in patients with Chronic liver disease when it is compared with the control group. Lipid profile including serum total cholesterol, TG, HDL, LDL and VLDL can be used to assess the severity

of liver disease; it could be a good reliable marker for the liver disorders.

#### Conflict of interest

The authors declare that they have no conflict of interest related to the publication of the manuscript.

#### References

1. Al-Jumaily EF, and Khaleel FM. The effect of chronic liver diseases on some biochemical parameters in patient's serum. *Curr Res J Biol Sci*, 2012; 4:5, 638-642.
2. Bellentani S, Pozzato G, Saccoccio G, Crovatto M, Croce M, Mazzoran L *et al*. Clinical course and risk factors of hepatitis C virus related liver disease in the general population: report from the Dionysos study. *Gut*. 1999; 44: 874-880.
3. Bellentani S, Saccoccio G, Costa G, Tiribelli C, Manenti F, Sodde M *et al*. The Dionysos Study Group. Drinking habits as cofactors of risk for alcohol induced liver damage. *Gut*. 1997; 41: 845-850.
4. Boemeke L, Bassani L, Marroni CA, Gottschall C.B.A. Lipid Profile In Cirrhotic Patients And Its Relation To Clinical Outcome. *AbcdArq Bras Cir Dig*. 2015; 28:132-135.
5. Chiang JY. Nuclear receptor regulation of lipid metabolism: potential therapeutics for dyslipidemia, diabetes, and chronic heart and liver diseases. *Curr Opin Investing Drugs*. 2005; 6: 994-1001.
6. Chrostek L, Supronowicz L, Panasiuk A, Cylwik B, Gruszevska E, Flisiak R. The effect of the severity of liver cirrhosis on the level of lipids and lipoproteins. *Clin Exp Med*. 2014; 14: 417-421.
7. Clark JM. The epidemiology of nonalcoholic fatty liver disease in adults. *J Clin Gastroenterol*. 2006; 40: S5-10.
8. Farrell GC and Larter CZ. Nonalcoholic fatty liver disease: from steatosis to cirrhosis. *Hepatology*. 2006; 43: S99-S112.
9. Ghadir MR, Riahin AA, Havaspour A, Nooranipour M, Habibinejad AA. The relationship between lipid profile and severity of liver damage in cirrhotic patients. *Hepat Mon*. 2010; 10: 285-288.
10. Gordon DA and Jamil H. Progress towards understanding the role of microsomal triglyceride transfer protein in apolipoproteins B lipoprotein assembly. *Biochim Biophys Acta*. 2000; 1486: 72-83.
11. Habib A, Mihas AA, Abou-Assi SG, Williams LM, Gavis E, Pandak WN, *et al*. High-density lipoprotein cholesterol as an indicator of liver function and prognosis in noncholestatic cirrhotics. *Clin Gastroenterol Hepatol*. 2005; 3: 286-291.
12. Jarikre AE, Momoh JAF. Plasma total cholesterol, high density lipoprotein cholesterol and low density lipoprotein cholesterol levels in liver cirrhosis in Nigerians. *Nig Q J Hosp Med*. 1996; 6: 157-159.
13. Jiang M, Liu F, Xiong WJ, Zhong L, Xu W, Xu F, *et al*. Combined MELD and blood lipid level in evaluating the prognosis of decompensated cirrhosis. *World J Gastroenterol*. 2010; 16: 1397-1401.
14. Khare S, Garg V K, and Jatav O. Serum iron and TIBC Parameters in Chronic liver disease. *Scholars Journal of Applied Medical Science*. 2015; 3: 2128-2131.
15. Mandal SK, Sil K, Chatterjee S, Ganguly J, Chatterjee K, Sarkar P *et al*. Study on Lipid Profiles In Chronic Liver Diseases. *National Journal Of Medical Research*. 2013; 3: 70-73.
16. McIntyre N. Plasma lipids and lipoproteins in liver disease. *Gut*, 1978, 19:526-530.
17. Mirandola S, Bowman D, Hussain MM, Alberti A. Hepatic steatosis in hepatitis C is a storage disease due to HCV interaction with microsomal triglyceride transfer protein (MTP). *Nutr Metab (Lond)*. 2010; 7:13.
18. Nangliya VJ, Sharma A, Mishra S. Evaluation of lipid profile in cirrhosis and their association with severity of the disease. *International Journal of Recent Trends in Science and Technology*. 2015; 16: 79-82.
19. Nashaat EH. Comparative study of serum lipid profile between chronic hepatitis C Egyptian patients and normal controls and the effect of viral eradication on lipids profile. *Report and Opinion*. 2010; 2: 14-20.
20. Petit JM, Benichou M, Duvillard L, Jooste V, Bour JB, Minello A, *et al*. Hepatitis C virus associated is correlated with plasma viral load, steatosis, and liver fibrosis. *Am J Gastroenterol*. 2003; 98: 1150-1154.
21. 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; 349: 25-32.
22. Radicheva MP, Andonova AN, Milcheva HT, Ivanova NG, Kyuchukova SG, Nikolova MS. Serum markers of iron metabolism in chronic liver disease. *Open access Macedonian Journal of Medical Science*. 2018; 6: 1-7.
23. Sabesin SM, Bertram PD, Freeman MR. Lipoprotein metabolism in liver disease. *Adv Intern Med*. 1980, 25:117-146.
24. Sachdeva S, Singh J, Kumar A, Singh J, Aggarwal J, Bansal G. Evaluation of Lipid Profile in Patients with Liver Cirrhosis. *International Journal of Contemporary Medical Research*. 2018; 5: 21-23.
25. Shindo N, Fujisawa T, Sugimoto K, Nojima K, Oze-Fukai A, Yoshikawa Y, *et al*. Involvement of microsomal triglyceride transfer protein in nonalcoholic steatohepatitis in novel spontaneous mouse model. *J Hepatol*. 2010; 52: 903-912.

#### How to cite this article:

RavikantSoni *et al* (2020) 'Study of Lipid Profile in Patients with Chronic Liver Disease', *International Journal of Current Advanced Research*, 09(01), pp. 20891-20893. DOI: <http://dx.doi.org/10.24327/ijcar.2020.20893.4091>

\*\*\*\*\*