



ACUTE VIRAL HEPATITIS AND ITS ASSOCIATION WITH LIPID PROFILE

Gastroenterology

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ABSTRACT

Background: Acute viral hepatitis is a parenchymal liver disease, characterized by widespread liver damage in which variable number of hepatocytes undergo necrosis. This results in different pattern of lipid abnormalities, which may be a prognostic factor in deciding the severity of acute Viral Hepatitis.

Aims: To know the pattern of lipid anomalies and severity of hepatic damage in acute viral hepatitis from the quantum of lipid profile. In addition, to find out if lipid profile helps in prognostication.

Methods: Case control study of patients of acute viral hepatitis was done. Seventy cases of acute viral hepatitis were studied along with seventy age and sex matched controls. All routine investigations, serological viral markers and fasting serum lipid profiles were analysed. All data was analysed using t test. A p value of <0.05 was considered statistically significant.

Results: Baseline parameters were similar between cases and control. On comparing the results of lipid profiles in both, the Serum Triglycerides, Cholesterol and LDLc levels were significantly raised ($p < 0.001$) and HDLc levels were significantly decreased ($p < 0.001$) in acute viral hepatitis when compared to controls. HDLc was significantly lower ($p < 0.001$) in cases of acute viral hepatitis with hepatic encephalopathy in contrast to those without it.

Conclusion: Triglycerides Cholesterol and LDLc were significantly raised and HDLc level were significantly decreased when compared to controls. HDLc was significantly low in cases with hepatic encephalopathy. In conclusion, estimation of serum HDLc allows better assessment of hepatic function and evaluation of prognosis of patients with acute viral hepatitis.

KEYWORDS

hepatitis, lipid profile, prognosis

INTRODUCTION

Viral hepatitis is a diffuse necroinflammatory disease of the liver as a result of infection by one of the five known primary hepatotropic viruses, namely HAV, HBV, HCV, HDV, HEV¹. Acute Viral hepatitis and Acute liver failure, caused by hepatitis viruses A through E, still remains a major public health problem in India. HAV is responsible for 10-30% of acute hepatitis and 5-15% of acute liver failure cases in India. HEV is responsible for 10-40% of acute hepatitis and 15-45% of acute liver failure in India². The disease spectrum in Acute viral hepatitis may range from mild illness with spontaneous recovery within a few weeks, to severe form, i.e. Acute Liver Failure which is characterized by altered sensorium and Coagulopathy has a high mortality in absence of liver transplantation. The liver is a major source of plasma lipoproteins including biosynthesis of VLDL, HDL, LDL, and also the most important organ for metabolism of lipids, lipoproteins and apolipoproteins. Therefore liver diseases could lead to change in the serum levels of lipids and lipoproteins^{3,4}. Varied patterns of Lipid abnormalities in obstructive and chronic parenchymal liver diseases is now firmly established⁵. Since changes in the concentration and composition of plasma lipids and lipoproteins occur frequently in liver diseases, these findings may be useful in following the clinical course of patients in the liver disease of various causes⁶. In the recovery phase, as clinical improvement occurs, simultaneous improvement is seen in the levels of lipoproteins. In this study, we have compared the lipid profile of patients suffering with Acute Viral Hepatitis with that of healthy people and also explored the possibility of using lipid profile as a prognosticating factor in these patients.

MATERIAL AND METHODS

Study Sample

The present study was a case control prospective study carried out at Shri Sayaji General Hospital and Medical College Baroda, Gujarat carried out between October 2013 to November 2014. Seventy patients above the age of 18 years having acute viral hepatitis, admitted to the medical wards of the hospital were enrolled as cases and seventy

healthy individuals were enrolled as controls after approval was received from the Institutional Ethics Committee For Human Research. Cases and Controls were matched according to their age, sex and socio-economic status. Both the cases and controls were explained about the study in detail, following which an informed written consent was taken regarding permission for inclusion in the study. For all Cases, detailed clinical history was taken, clinical examination was done and laboratory investigations that included Complete Blood Count, routine Urine examination, Blood Glucose, Serum Creatinine, Blood Urea, Liver Function Tests, Lipid Profile, Prothrombin Time, Serum Electrolytes, Serological testing for Hepatotropic Viruses and necessary radiological investigations were done. Blood collection for Lipid Profile was done after proper overnight fasting. During hospitalization, all cases were given standard treatment protocol for management of acute viral hepatitis and their clinical condition closely followed. For Controls, healthy individuals were enrolled which were confirmed with detailed history and clinical examination. Blood collection after proper overnight fasting was done for lipid profile estimation.

Inclusion criteria

All confirmed cases (age >18 years) of acute viral hepatitis, defined as a systemic infection predominantly affecting the liver caused most often by hepatotropic viruses (hepatitis A, B, C, D, E) according to the World Gastroenterology Organisation.

Exclusion criteria:

- Patients having pre-existing liver disease.
- Alcoholic liver diseases.
- Patients of Diabetes Mellitus, thyroid disorders and nephrotic syndrome.
- Patients on drugs that alter lipid profile which include: Statins, Fenofibrates, Beta blockers, Steroids, Amiodarone, Oral contraceptive pills, Thiazides (>25 mg/day), Pioglitazone, omega 3 fatty acids, bile acid sequestrants, orlistat, indapamide.

Data analysis:

Statistical analysis was done using t test using Microsoft Office Excel 2007 version. A p value of <0.05 was considered as statistically significant. The results were tabulated and graphically represented using Microsoft Office for Windows 2008.

Results

The results of the Lipid Profile, which included Serum Triglyceride, Serum Cholesterol, Serum LDL, Serum HDL and Serum VLDL, of the cases and controls, whose baseline characteristics did not have significant differences, were compared. Serum Triglyceride levels were significantly higher ($p < 0.001$) in the cases (165.74 ± 12.9) as compared to the control group (138.09 ± 21.06), as is shown in Fig.1.

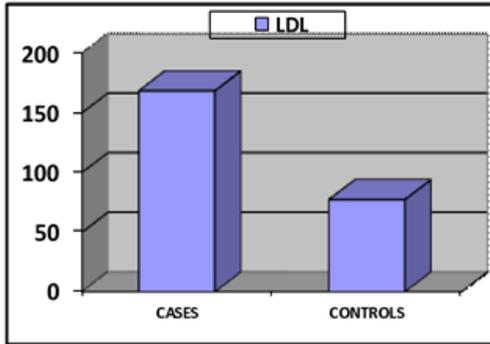


FIGURE-1 -COMPARISON OF SERUM LDL LEVEL BETWEEN CASE AND CONTROLS

Comparison of LDL- Cholesterol levels between cases (168 ± 12.8) and controls (77.3 ± 23.7) also revealed much higher levels in cases as against controls, showing significant difference ($p < 0.001$), as shown in fig.2.

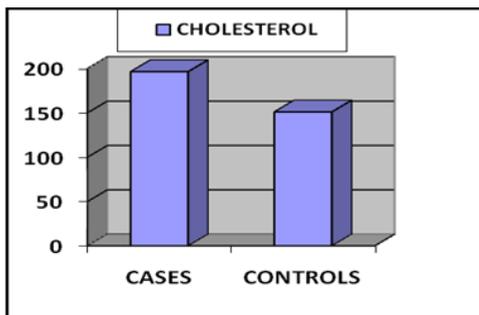


FIGURE-2 COMPARISON OF SERUM CHOLESTEROL LEVEL BETWEEN CASE AND CONTROLS

Fig 3 shows that the levels of Serum Cholesterol among subjects (196.3 ± 23.96) was significantly raised when compared to the control group (150.9 ± 22), this increase in cholesterol levels in subjects with viral hepatitis was highly significant ($P < 0.001$).

The levels of HDL Cholesterol among cases (22.7 ± 6.48) was significantly decreased when compared to the control group (39.4 ± 6.36), which was highly significant ($P < 0.001$) and consistent with a large volume of publications on this subject.

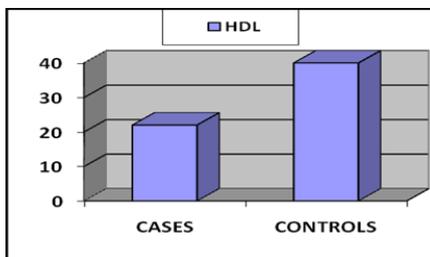


FIGURE-3 COMPARISON OF SERUM HDL LEVEL BETWEEN CASE AND CONTROLS

Out of 70 cases of viral hepatitis, 10 had developed hepatic encephalopathy. On comparing the HDLc level of cases with hepatic encephalopathy and with those without it, a significant fall in HDLc levels in cases with hepatic encephalopathy ($p < .001$) was noted and hence could be a predictor of poor prognosis.

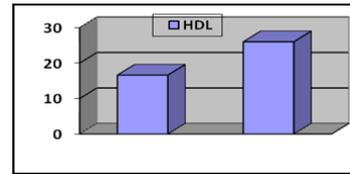


FIGURE-4 COMPARISON OF SERUM HDLc LEVELS BETWEEN CASES WITH AND WITHOUT HEPATIC ENCEPHALOPATHY.

Discussion

The liver plays an indispensable role in lipid and lipoprotein metabolism, functioning as the center for lipoprotein uptake, formation and export to the circulation and also a source of some important apolipoproteins and enzymes which are requisite for lipid metabolism. Cholesterol and Triglycerides are insoluble in water and so for transportation in the circulation, they have to form complexes with proteins, which are known as lipoproteins. Enzymes such as Lipoprotein Lipase (LPL), Lecithin Cholesterol Acyl Transferase (LCAT), and Hepatic Triglyceride Lipase, play a key role in the metabolism of lipoproteins of which LCAT and Hepatic Triglyceride Lipase are synthesized only in the liver^{7,8}. LCAT, first described in 1962 by Glomset⁹, a key enzyme in lipoprotein metabolism, promotes formation of HDL. Liver plays a key role in maintaining a relative equilibrium of lipids and lipoproteins by regulating the lipoprotein receptors, which are membrane proteins, important for lipid metabolism. In hepatic parenchymal diseases, due to damage of the hepatocytes, these functions are disturbed, resulting in marked changes in the lipids and lipoproteins¹⁰. Disturbances in the metabolism of Cholesterol and Triglycerides lead to alteration in their levels as well. Therefore, estimation of Cholesterol, Triglycerides and Lipoprotein levels can give us an idea regarding the extent of hepatocellular damage and may also be of prognostic value.

Our study showed that the serum triglyceride levels were significantly raised in patients of acute viral hepatitis as compared to the controls with the p value <0.001. This result was similar to several other studies by Prasantakumar Bhattacharya et al¹¹, LiboLua et al¹², Goel et al¹³ and Neil McIntyre et al⁷. In contrast to this, a study by N M Papadopoulos¹⁴ demonstrated that triglyceride levels remained in the normal range in patients with Viral Hepatitis¹⁴. This rise in triglycerides is attributed to the decrease of hepatic lipase levels and also decreased levels of lipoprotein lipase^{15,16}. The other mechanism could be that, in acute viral hepatitis, free fatty acids are mobilized from adipose tissue depots to the liver to be reesterified to triglycerides to be transported back to periphery.

Our study showed significantly increased levels of LDLc in patients of acute viral hepatitis as compared to controls (p value <0.001). This is in accordance with the studies by Prasantakumar Bhattacharya et al¹¹, Goel et al¹³, LiboLua et al¹². This was in contrast to a study done by Neil McIntyre et al⁷, which showed no change in LDLc levels in patients of acute viral hepatitis when compared with the controls.

The level of serum cholesterol in patients of acute viral hepatitis was also significantly increased in comparison to controls in our study (p value <0.001). This observation is similar to that in a study by LiboLua et al¹². The probable explanation for the raised serum cholesterol is that, there is decreased activity of Lecithin Cholesterol Acyl Transferase in viral hepatitis and also in part to intrahepatic biliary obstruction. This is in contrast to the results observed in a study done by Neil McIntyre⁷ and N M papadopoulos¹⁴

The levels of serum HDLc in patients of acute viral hepatitis in our study were significantly decreased (p value <0.001) as compared to the controls which showed consistency with several studies by Neil McIntyre⁷, Goel et al¹³, GNKundaje et al¹⁷ and Mirshad et al¹⁸. HDLc levels were also observed to be significantly much lower in patients with hepatic encephalopathy as compared to those without, thus highlighting the significance of HDLc in prognostication of liver diseases. Thus, HDL levels can be used along with Prothrombin time

and Serum Bilirubin levels, which are established prognostic factors, to grade the severity of liver involvement. Decreased level of LCAT, whose only source is liver resulted in decreased HDLc production. Deficiency of LCAT causes impairment of conversion of nascent HDL to mature HDL, resulting in increased levels of immature HDL which are more prone for degradation and hence reduction in HDL levels.¹⁹

Conclusion

Triglycerides, Cholesterol and LDLc levels were significantly raised in acute viral hepatitis patients when compared to controls. HDLc levels were significantly decreased when compared to controls; more so in patients with hepatic encephalopathy as compared to those without it. Hence, estimation of serum HDLc allows better assessment of hepatic function and evaluation of prognosis of patients with acute viral hepatitis.

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PEER REVIEW

Not commissioned. Externally peer reviewed.

CONFLICTS OF INTEREST

we have no competing interests.

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