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Original Research Article

A comparative study of lipid profile in patients with and without infective hepatitis

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ABSTRACT

Background: Liver plays a central role in lipid metabolism. Liver carries out some important functions in lipid metabolism like liver facilitates the digestion and absorption of lipids by the production of bile, which contains cholesterol and bile salts synthesized within the liver de novo or from uptake of lipoprotein cholesterol, the liver has active enzyme system for synthesizing and oxidizing fatty acids and for synthesizing triacylglycerols and phospholipids, synthesis of the ketone bodies, it plays an integral part in the synthesis and metabolism of plasma lipoproteins. The objective was to compare lipid profile in patients with and without infective hepatitis.
Methods: Hospital based cross sectional comparative study was carried out among 112 cases. The patients were divided as having infective hepatitis (69) called cases and not having infective hepatitis (43) called controls. Concentration of serum total cholesterol was determined by Carr and Drekter method. Concentration of serum HDL cholesterol was determined by Carr and Drekter method. Concentration of serum triglyceride level was determined by enzymatic end point peroxidase coupled method.
Results: Total cholesterol, VLDL, LDL and triglycerides have been found to be significantly higher in cases of infective hepatitis compared to control. The HDL value was also significantly deranged i.e. significantly lower in cases compared to controls ($p < 0.05$). Thus, it was clear that infective hepatitis deranges the lipid profile of the patients.
Conclusions: Lipid profile can be used as sensitive indicators of hepatic function and may have diagnostic and prognostic importance in infective hepatitis.

Keywords: Comparison, Controls, Cases, Infective hepatitis, Lipid profile

INTRODUCTION

Liver plays a central role in the maintenance of metabolic homeostasis. The biochemical functions in which the liver plays a major role include the intermediate metabolism of amino acids and carbohydrates, synthesis and degradation of proteins and glycoproteins, regulation of lipid and cholesterol metabolism, metabolism of drugs and hormones and contribution to immune system function through the hepatic immune response.[1] Viral hepatitis is a common and serious infectious disease caused by several viruses and marked by necrosis and inflammation of the liver. This disease was traditionally separated into two types: type A or infectious hepatitis caused by hepatitis A virus (HAV) and type B or serum hepatitis caused by hepatitis B virus (HBV). In last 25 years, it has become clear that there are many viruses that cause acute viral hepatitis. At present five hepatitis

viruses have been identified- hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis D virus (HDV), a parenterally transmitted classic non-A, non-B hepatitis virus (HEV). These five forms of hepatitis are primary infectious diseases of the liver. The five forms of viral hepatitis are similar clinically but the viruses that cause them are quite distinct. Manifestations of these viral infections are hepatocellular necrosis and hepatic inflammation and affect other organs to a major extent. Several other viruses can secondarily affect the liver and induce a viral hepatitis like syndrome. The most important of these viruses are Epstein Barr Virus (EBV), and Cytomegalovirus (CMV). Hepatitis can also occur with infections with herpes simplex virus, varicella zoster virus, measles, rubella, rubeola and coxsackie B viruses and adeno viruses.[2]

Liver plays a central role in lipid metabolism. Liver carries out some important functions in lipid metabolism like liver facilitates the digestion and absorption of lipids by the production of bile, which contains cholesterol and bile salts synthesized within the liver de novo or from uptake of lipoprotein cholesterol, the liver has active enzyme system for synthesizing and oxidizing fatty acids and for synthesizing triacylglycerols and phospholipids, synthesis of the ketone bodies, it plays an integral part in the synthesis and metabolism of plasma lipoproteins. Hence, infective hepatitis which is a parenchymal liver disease encountered quite frequently in medical practice alters the serum lipid and lipoprotein concentrations.[3]

Mechanisms responsible for these alterations are regurgitation of biliary cholesterol into circulation, increased hepatic synthesis of cholesterol, decreased plasma lecithin cholesterol acyltransferase activity, regurgitation of biliary lecithin which causes a shift of cholesterol from preexisting tissue cholesterol into the plasma. The most frequent disorder of serum lipids in hepatitis is a moderate increase in the ratio of free to total cholesterol.[4]

With this background, present study was carried out to compare lipid profile in patients with and without infective hepatitis.

METHODS

This was a hospital based cross sectional comparative study carried out at JJM Medical College and hospital over a period of one year from January 2002 to December 2002.

The patients admitted to Chigateri District Hospital, Bapuji Hospital and outpatient cases of Chigateri District Hospital and Bapuji Hospital, Davangere were selected for the present study.

The Ethical Committee of JJM Medical College had given the consent for conducting the present study. During the study, written informed consent was taken from each patient. During the study period it was possible to study a total of 112 cases as per the inclusion and exclusion criteria set for the present study.

The patients were divided as having infective hepatitis and not having infective hepatitis. Among the sample size of 112, 43 were not having infective hepatitis and they were recruited as controls and 69 were having infective hepatitis and they were recruited as cases for the present study.

For cases, those with medical evidence of having infective hepatitis and for controls, those who are free from infective hepatitis were included.

Patients with those eligible for the present study but not willing and those eligible but bed ridden, or other severe complications or not able to give history and other details were excluded.

Procedure

After the patient being admitted and found eligible for the present study, he or she was explained the nature of the study and consent was sought. Those willing to give the consent and able to cooperate brief history was taken from each such patients. Complete clinical examination was carried out. Routine biochemical investigations like SGPT, SGOT, ALP, bilirubin and HBsAg were analyzed.

Five ml venous blood was collected by using sterile disposable syringe and taking all universal precautions. Later serum was separated from the clot within half an hour by centrifugation. Following biochemical analyses were made from the serum:

- Concentration of serum total cholesterol was determined by Carr and Drekter method.[5]
- Concentration of serum HDL cholesterol was determined by Carr and Drekter method.[5]
- Concentration of serum triglyceride level was determined by enzymatic end point peroxidase coupled method.[6]
- Concentration of serum LDL cholesterol was determined by Fried Wald's formula.[6]

The data was recorded and analyzed using mean and standard deviation (+2SD). Students t-test was applied to compare the mean values between the two groups and if it was found that the p value was less than 0.05, it was taken as statistically significant i.e. the difference in the values of the two groups are really present and the observation was not by chance.

RESULTS

Table 1 shows estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in controls. The mean serum levels of Tc, VLDLc, LDLc, HDLc and Tg were within normal limits. Even in the range the minimum value and the

maximum value were within normal limits. Table 2 shows comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in controls according to age. As the age increased, total cholesterol, VLDL, LDL and triglyceride increased and this difference was statistically significant but as the age increased, the HDL value decreased and this decrease was found to be statistically significant among the controls.

Table 1: Estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in controls.

Particulars	Tc (mg/dl)	VLDLc (mg/dl)	LDLc (mg/dl)	HDLc (mg/dl)	Tg (mg/dl)
Mean	158.4	21.6	78.7	58.2	108.1
±2SD	20.9	4.6	24.4	9.3	23.7
Range	122-210	28-46	72-139	35-72	74-168

Table 2: Comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in controls according to age.

Age (years)	Total number	Tc (mg/dl)	VLDLc (mg/dl)	LDLc (mg/dl)	HDLc (mg/dl)	Tg (mg/dl)
20-34	19	142±10.6	19.2±3.2	60.1±11.2	63.6±5.2	95.6±19.1
35-49	14	159±14.1	22±4.3	81.4±17.3	56.3±8.5	110.9±22.9
>50	10	186±11.6	25.4+3.8	110.4±15.6	50.5±10.7	127.8±18.8
P value		<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

Table 3: Comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in controls as per sex.

Sex	Total number	Tc (mg/dl)	VLDLc (mg/dl)	LDLc (mg/dl)	HDLc (mg/dl)	Tg (mg/dl)
Male	20	170±15.4	22.3±4.6	89.8±20.7	57.7±10	112.5±23.6
Female	23	148.8±199	20.9±4.6	58.6±8.9	58.6±8.9	104.2±23.5
P value		<0.0001	0.34	<0.0001	0.75	0.26

Table 4: Comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg between cases and controls.

Sex	Total number	Tc (mg/dl)	VLDLc (mg/dl)	LDLc (mg/dl)	HDLc (mg/dl)	Tg (mg/dl)
Controls	43	158.4±20.9	21.6±4.6	78.7±24.4	58.2±9.3	108.1±23.7
Cases	69	170±16	38.7±3.5	107.7±14.4	25±5.2	189±17.4
P value		<0.0001	<0.0001	<0.0001	<0.0001	<0.0001

Table 5: Comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in cases as per sex.

Sex	Total number	Tc (mg/dl)	VLDLc (mg/dl)	LDLc (mg/dl)	HDLc (mg/dl)	Tg (mg/dl)
Male	52	169.1±26.5	38±3.8	109.1±15.3	25.3±5.5	189.9±18.8
Female	17	164.3±13.5	37.7±2.6	104.7±11	24.6±4.1	188.5±12.9
P value		0.48	0.77	0.24	0.37	0.78

Table 3 shows comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in controls as per sex. Serum total cholesterol was significantly more in males compared to females and serum LDL was also significantly more in males compared to females but there was no difference in terms of VLDL, HDL and triglyceride values among males and females.

Table 4 shows comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg between cases and controls. Total cholesterol, VLDL, LDL and triglycerides have been found to be significantly higher in cases of infective hepatitis compared to control. The HDL value was also significantly deranged i.e. significantly lower in cases compared to controls ($p<0.05$). Thus, it was clear that infective hepatitis deranges the lipid profile of the patients.

Table 5 shows comparison of estimated serum levels of Tc, VLDLc, LDLc, HDLc and Tg in cases as per sex. The values of lipid profile of all cases among the sexes were not found to be statistically significant ($p>0.05$). The mean values of triglycerides and HDL were abnormal in both the males and females. But other mean values like total cholesterol, VLDL, LDL were within normal limits. Table 6 shows inter correlations between Tc, VLDLc, LDLc, HDLc and Tg in infective hepatitis cases. Serum total cholesterol has significant positive

correlation with triglycerides, VLDLc, LDLc levels. Serum HDLc has been found to have significant inverse correlation with triglycerides and LDLc levels. Serum triglycerides have significant positive correlation with LDLc, and VLDLc levels and serum LDLc has significant positive correlation with VLDLc levels.

Table 6: Inter correlations between Tc, VLDLc, LDLc, HDLc and Tg in infective hepatitis cases.

Particulars	Tc	HDLc	Tg	LDLc	VLDLc
Tc	-	0.06	0.49*	0.87*	0.48*
HDLc	-	-	(-) 0.16*	(-) 0.28*	(-) 0.04
Tg	-	-	-	0.33*	0.99*
LDLc	-	-	-	-	0.33*
VLDLc	-	-	-	-	-

DISCUSSION

Infective hepatitis which is a parenchymal liver disease is encountered quite frequently in clinical practice. As liver plays a central role in lipid metabolism, serum lipids and lipoproteins concentration are known to change in infective hepatitis.

As the vast array of biochemical tests are available for diagnosing and assessing severity of liver cell damage, in liver disease desired sensitivity and specificity are lacking. So, a new test which is better suited for the above-mentioned functions is always welcome. In this present study the clinical utility of the measurement of serum lipid profile as sensitive indicator of hepatic function in infective hepatitis was assessed.

Author found that the value of serum total cholesterol was significantly elevated in patients with infective hepatitis when compared to controls ($p<0.05$). This observation supports the earlier reports.[7-12] However, in contrary the studies done by McIntyre NE et al, and Papadopoulos NM et al, showed that the total serum cholesterol remained unaltered in infective hepatitis when compared to controls.[13,14] The probable explanation for the raised serum total cholesterol is that it was because of increase in serum free cholesterol to cholesterol ester, which resulted due to decreased lecithin cholesterol acryl transferase (LCAT) activity in viral hepatitis, due to impaired hepatic LCAT synthesis which is responsible for esterification of free cholesterol to cholesterol ester.[9]

Author observed that the levels of VLDLc in patients with infective hepatitis significantly increased when compared to controls ($p<0.05$). This observation was in agreement with earlier report of Gallin JI et al, however, in contrary to this there are reports stating decreased serum VLDLc levels.[11,13-15] However, this decrease in serum VLDLc levels is not accounted for in these earlier reports. Mehrotra TN et al, reported no significant difference in serum VLDLc levels in infective hepatitis.[16] VLDL production is stimulated by conditions that elevate free fatty acid levels. As reported earlier in viral hepatitis fatty acids are mobilized from adipose tissue and are re-esterified to triglyceride in liver and transported back to the peripheries after being secreted in the form of VLDL.[16]

Possibly this may lead to the increased serum VLDLc levels in infective hepatitis. Apo C-III carried in VLDL may have an inhibitory effect on hepatic VLDL uptake. This may tend to prolong circulation interval of VLDL, thereby elevating the levels of VLDL. Defective clearance of VLDL remnants may also be a cause for increase in serum VLDLc levels in infective hepatitis.

There was significant increase in levels of serum LDLc in patients compared to controls. This is in accordance with previous studies.[15,17] However, this increase in LDLc is not accounted for in these earlier reports.[11,18] In the present study, there was significant increase in serum VLDLc levels, which may lead to increase in serum LDLc concentration as a result of metabolic transformation of VLDL into LDL.

The levels of serum HDLc which was significantly decreased in cases of infective hepatitis when compared to controls is in accordance with earlier reports.[13,14,16] The decrease in serum HDLc in patients with infective hepatitis can be attributed to decreased production of enzyme LCAT by the diseased liver. Liver is the only source of enzyme LCAT and serum levels of this is decreased in liver diseases. It seems probable that LCAT plays a necessary role in the normal conversion of the nascent HDL to the mature HDL.

In deficiency of LCAT there is impairment of conversion of nascent HDL to mature HDL, resulting in an increase in immature HDLc, which is more prone for degradation, resulting in decreased levels of serum HDL.[13,18] Decreased production of Apo lipoprotein A and all by liver may also contribute for decreased serum HDLc production.[15]

The levels of serum triglycerides were significantly higher in cases compared to controls. This observation is in accordance with earlier reports.[8-11]

CONCLUSION

Increase in serum concentrations of Tc, VLDLc, LDLc, Tg and decrease in HDLc levels indicate that there is impairment in the lipid metabolism in infective hepatitis. Hence, these parameters can be used as sensitive indicators of hepatic function and may have diagnostic and prognostic importance in infective hepatitis.

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