Comparison of lipid parameters in acute viral Hepatitis and normal individuals

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Abstract

Introduction: The liver is a major organ of the body playing an important role in the maintenance of metabolic homeostasis especially lipid metabolism. As viral hepatitis is an inflammatory condition of the liver, it might interfere with the lipid and lipoprotein metabolism and cause a disturbance in their regulation in vivo and thereby cause change in normal levels of lipids and lipoproteins.

Materials and Methods: It is a case control study was designed to carry out the work with a total of 30 cases of acute viral hepatitis and 30 healthy controls were taken belonging to the age group 19-35 years. A detailed history, clinical examination, liver function test, lipid profile, viral markers and other relevant laboratory investigations were done. Appropriate statistical methods were applied to analyze the results.

Results: The study shows that there is a statistically significant increase in the triglycerides, LDL, VLDL and a decrease in HDL. The follow up analysis showed that serum bilirubin, SGOT, SGPT and triglycerides were decreased and total cholesterol and HDL were increased. Comparison of serum bilirubin with lipid parameters shows no correlation with total cholesterol and inverse correlation with TGL which is statistically insignificant. Comparison of SGOT and SGPT with lipid parameters shows inverse correlation with triglycerides which is statistically significant.

Conclusion: From the study it can be concluded that acute viral hepatitis does affect the lipid profile and HDL levels might be used as a prognostic marker to assess severity of liver disease.

Keywords: Lipid profile, Hepatitis, SGOT, SGPT, Bilirubin.

Introduction

Acute viral hepatitis is defined as an inflammatory process of liver lasting less than 6 months, commonly caused by viruses.[1] The liver is a major organ of the body playing an important role in the maintenance of metabolic homeostasis.[2] The liver is largely involved with the synthesis and regulation of various lipids, lipoproteins and apolipoproteins.[1] Normally most plasma endogenous lipids and lipoproteins are synthesized in the liver and are then secreted into circulation.[2] Plasma lipoproteins are also catabolised by the liver for maintenance of balance of lipids and lipoprotein metabolism in vivo. It also functions in metabolism and regulation of amino acids, carbohydrates, proteins, hormones and metabolism of durgs.[2]

As viral hepatitis is an inflammatory condition of the liver, it might interfere with the lipid and lipoprotein metabolism and cause a disturbance in their regulation in vivo and thereby cause change in normal levels of lipids and lipoproteins.[3] For more than 50 years, the above hypothesis has gained importance among the practitioners. Several reports have been published describing the changes in serum lipids in different animal species infected by various viruses, bacteria and protozoans.[4] Various studies till date have provided positive relation between lipid profile and various forms of viral hepatitis. Even though most of the studies agree that there will be change in lipid profile in viral hepatitis, some data till date disagree with the above hypothesis.

Because of the inconsistencies in the present available data, we are studying the lipid profile in a group of patients with viral hepatitis. We are studying the effect of viral hepatitis on lipid profile and whether the degree of hepatic damage in acute viral hepatitis can be assessed based on impairments in serum lipid composition.

Materials and Methods

Study was conducted with thirty patients with acute viral hepatitis and thirty age and sex matched controls were taken from healthy donors.

Case Group

Inclusion Criteria

Patients diagnosed with acute viral hepatitis in the age group 19-35 years by positive HBsAg for hepatitis virus.

Exclusion Criteria

1. Patients who consume alcohol
2. Smokers
3. Patients with diabetes mellitus, hypertension or ischemic heart disease
4. Chronic use of drugs which affect lipid profile
5. Hepatitis due to non-viral aetiology

Control Group

Inclusion Criteria

Healthy individuals coming for voluntary blood donation to blood bank.

Exclusion criteria

1. Hepatitis due to viral and non-viral etiology
2. Patients who consume alcohol
3. Smokers
4. Patients with diabetes mellitus, hypertension or ischemic heart disease

5. Chronic use of drugs which affect lipid profile

The present study was done on a total 60 patients, 30 cases with signs and symptoms suggestive of acute hepatitis (duration of illness <6 months).Cases presenting to the General Medicine OPD with signs and symptoms suggestive of acute hepatitis, who satisfy the inclusion criteria and voluntarily agree to be involved in the study were admitted. They were undergoing thorough history taking and clinical examination. Venous blood sample was drawn under aseptic precautions and collected in a pro-coagulation tube and the sample was sent for: Complete blood count a nd HBsAg for hepatitis virus and the following:

Fasting Lipid Profile
(a) Total Serum Cholesterol
(b) Triglycerides
(c) HDL-Cholesterol
(d) LDL-Cholesterol
(e) VLDL-Cholesterol

Liver Function Test
(a) Total Protein
(b) Albumin
(c) Globulin
(d) A/G Ratio
(e) Bilirubin-Total
(f) Bilirubin-Direct
(g) Bilirubin-Indirect
(h) SGOT (AST)
(i) SGPT (ALT)
(j) ALP

The results of these tests were recorded in a proforma.

Serum Bilirubin Estimation And Direct Bilirubin by Photometric test using 2,4-dichloroaniline (DCA) (Wahlefeld method)[5,6]

SGOT (ASAT) KIT:[5,6] SGOT is an enzyme found mainly in heart muscle, liver cells, skeletal muscle and kidneys. Injury to these tissues results in the release of the enzyme in blood. Elevated levels are found in myocardial infarction, cardiac operations, hepatitis, cirrhosis, acute pancreatitis, acute renal diseases, primary muscle diseases. Decreased levels may be found in pregnancy, Beriberi and diabetic ketoacidosis.

SGPT (ALAT) KIT:[5,6] SGPT is found in a variety of tissues but is mainly found in the liver. Increased levels are found in hepatitis, cirrhosis, obstructive jaundice and other hepatic diseases. Slight elevation of the enzymes is also seen in myocardial infarction.

Lipid Profile Estimation[7]
1. Total cholesterol levels were estimated by CHOD-PAP method
2. Triglyceride levels were estimated by GOP-PAP method
3. HDL cholesterol levels were estimated by CHOD-PAP method
4. LDL cholesterol levels were derived by calculations.

Statistical Methods
Results on continuous measurements are presented on Mean $\pm$ SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance.

Student t test has been used to find the significance of study parameters on continuous scale between two groups (Inter group analysis) on metric parameters. Student t test (two tailed, dependent) has been used to find the significance of study parameters on continuous scale within each group. Chi-square/Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

Statistical Software: The Statistical software namely SPSS 15.0 was used for the analysis of the data and Microsoft word and Excel have been used to generate tables, graphs etc.

Results
In case group, majority of individuals belong to the age group 21-30 years (86.7%) followed by 31-40 years (10%) and 19-20 years (3.3%). The average age of case group is 25.71$\pm$3.91.

In control group, majority of individuals belong to the age group 21-30 years (70%) followed by 19-20 years (16.7%) and 31-40 years (13.3%). The average age of control group is 24.55$\pm$4.08. Samples are age matched with P=0.257

20(66.7%) individuals were females and 10(33.3%) were males in case group. 20(66.7%) individuals were females and 10(33.3%) were males in control group.

Table 1: Clinical characteristics

Clinical characteristics	No. of patients (n=30)	%
Jaundice	30	100.0
Fever	21	70.0
Vomiting	19	63.3
Malaise	15	50.0
Encephalopathy	4	13.3

Assessing the clinical features of the case group, 30(100%) individuals had jaundice, 21(70%) individuals had fever, 19(63.3%) individuals had vomiting, 15(50%) individuals had malaise and 4(13.3%) individuals had encephalopathy.

Table 2: Comparison of lipid parameters between case and control groups

Lipids parameters	Cases (n=30)		Controls (n=30)		P value
	No	%	No	%	
Total cholesterol (mg%)					
1. <200	28	93.3	27	90.0	1.000
2. 200-280	2	6.7	3	10.0	
3. >280	0	0.0	0	0.0	
TGL (mg%)					
1. <150	6	20.0	24	80.0	<0.001**
2. 150-200	23	76.7	6	20.0	
3. >200	1	3.3	0	0.0	
HDL (mg %)					
1. <35	16	53.3	5	16.7	
2. 35-60	14	46.7	25	83.3	
3. >60	0	0.0	0	0.0	
LDL (mg %)					
1. <70	1	3.3	7	23.3	0.028*
2. 70-190	27	90.0	23	76.7	
3. >190	2	6.7	0	0.0	
VLDL (mg %)					
1. <30	2	6.7	20	66.7	<0.001**
2. 30-60	28	93.3	10	33.3	
3. >60	0	0.0	0	0.0	

28(93.3%) individuals of cases and 27(90%) individuals of control has total cholesterol of <200mg%, 2(6.7%) individuals of cases and 3(10%) individuals of controls has total cholesterol of 200-280mg% and no individuals in cases and controls has total cholesterol of >280mg%. The p value is 1.000 which is statistically not significant.

6(20%) individuals of cases and 24(80%) individuals of controls has triglycerides of <150mg%, 23(76.7%) individuals of cases and 6(20%) individuals of controls has triglycerides of 150-200mg% and 1(3.3) individuals of cases and no controls has triglycerides of >200mg%. The p value is <0.001 which is statistically significant.

16(53.3%) individuals of cases and 5(16.7%) individuals of controls has HDL of <35mg%, 14(46.7%) individuals of cases and 25(83.3%) individuals of controls has HDL of 35-60mg% and no individuals of cases and controls has HDL of >60mg%. The p value is 0.003 which statistically significant.

1(3.3%) individuals of cases and 7(23.3%) individuals of control has LDL of <70mg%, 27(90%) individuals of cases and 23(76.7%) individuals of controls has LDL of 70-190mg% and 2(6.7%) individuals of cases and no controls has LDL of >190mg%. The p value is 0.028 which is statistically significant.

2(6.7%) individuals of cases and 20(66.7%) individuals of controls has VLDL of <30mg%, 28(93.3%) individuals of cases and 10(33.3%) individuals of controls has VLDL of 30-60mg% and no cases and controls has VLDL of >60mg%.The p value is <0.001 which is statistically significant.

Table 3: Comparison of lipid parameters between case and control groups

Lipid parameters	Cases	Controls	P value
Total cholesterol (mg %)	166.58±19.44	154.52±31.10	0.072+
TGL (mg %)	158.10±38.87	115.35±39.53	<0.001**
HDL (mg %)	33.81±4.12	41.16±6.25	<0.001**
LDL (mg %)	149.26±30.50	86.45±23.28	<0.001**
VLDL (mg %)	39.10±6.31	26.58±12.27	<0.001**

The average of total cholesterol of 30 individuals in case group is 166.58±19.44 and of 30 individuals in control group is 154.52±31.10 with a p value of 0.072+ which is nearing significance. The average of triglycerides of 30 individuals in case group is 158.10±38.87 and of 30 individuals in control group is 115.35±39.53 with a p value of <0.001 which is statistically significant.

The average of HDL of 30 individuals in case group is 33.81±4.12 and of 30 individuals in control group is 41.16±6.25 with a p value of <0.001 which is statistically significant.

The average of LDL of 30 individuals in case group is 149.26±30.50 and of 30 individuals in control group is 86.45±23.28 with a p value of <0.001 which is statistically significant.

The average of V LDL of 30 individuals in case group is 39.10±6.31 and of 30 individuals in control group is 26.58±12.27 with a p value of <0.001 which is statistically significant.

Follow up analysis of cases

Table 4: Follow up analysis patients studied

Variables	First day	Follow up day	Difference	t value	P value
S.Bilirubin (mg/dl)	6.86±4.23	4.07±3.61	2.79	6.546	<0.001**
SGOT U/L	312.78±217.96	180.35±139.29	132.43	4.666	<0.001**
SGPT U/L	330.56±226.29	191.21±149.71	139.35	4.983	<0.001**
Cholesterol (mg/%)	166.06±17.72	176.34±18.65	10.26	4.892	<0.001**
TG (mg/%)	156.91±38.75	128.52±39.47	28.39	3.936	0.001**
HDL (mg/%)	33.35±4.33	40.48±4.29	7.13	7.112	<0.001**

Out of 30 cases only 23 came for follow up. Out of the 23 cases, the average of serum bilirubin on 1st day is 6.86±4.23 and on follow up day is 4.07±3.61 with a difference of 2.79, t value of 6.546 and p value of <0.001.

The average of SGOT on 1st day is 312.78±217.96 and on follow up day in 180.35±139.29 with a difference of 132.43, t value of 4.666 and p value of <0.001.

The average of SGPT on 1st day is 330.56±226.29 and on follow up day is 191.21±149.71 with a difference of 139.35, t value of 4.983 and p value of <0.001.

The average of cholesterol on 1st day is 166.06±17.72 and on follow up day is 176.34±18.65 with a difference of 10.26, t value of 4.892 and p value of <0.001.

The average of triglycerides on 1st day is 156.91±38.75 and on follow up day is 128.52±39.47 with a difference of 28.39, t value of 3.936 and p value of <0.001.

The average of HDL on 1st day is 33.35±4.33 and on follow up day is with a 40.48±4.29 difference of 7.13, t value of 7.112 and p value of <0.001.

Table 5: Pearson correlation between Serum Bilirubin, SGOT and SGPT with Lipid parameters of case group

Lipid parameters	Serum Bilirubin		SGOT		SGPT	
	r-value	p-value	r-value	p-value	r-value	p-value
Total Cholesterol	0.272	0.138	-0.011	0.952	-0.027	0.884
TGL	-0.210	0.257	-0.463	0.009**	-0.466	0.008**
HDL	-0.009	0.962	-0.207	0.264	-0.202	0.276
Vs LDL	0.326	0.073+	0.297	0.105	0.295	0.107
VLDL	0.158	0.397	0.072	0.700	0.052	0.782

Serum Bilirubin: Correlating Serum Bilirubin of cases with total cholesterol (r value – 0.272 and p value - 0.138), HDL (r value – 0.009 and p value – 0.962), LDL (r value – 0.326 and p value – 0.073) and VLDL (r value – 0.158 and p value – 0.397) proves that no positive or inverse correlation exists between serum bilirubin levels and total cholesterol among the cases with hepatitis.
Whereas there is a inverse correlation shown between serum bilirubin and TGL among cases with an r value of -0.210 and p value of 0.257, which is statistically insignificant.
SGOT: Correlating SGOT of cases with total cholesterol (r value -0.011 and p value – 0.952), HDL (r value – 0.207 and p value – 0.264), LDL (r value – 0.297 and p value – 0.105) and VLDL (r value – 0.072 and p value – 0.700) proves that no positive or inverse correlation exists between SGOT and total cholesterol, HDL, LDL, VLDL. Correlating SGOT of cases with TGL (r value – 0.463 and p value – 0.009**) shows that SGOT and TGL have inverse correlation r=-0.463, r^2=0.214, p=0.009 (statistically significant) and it shows that 0.21 percent of the variation in SGOT is inversely related to the variation in TGL ie with every 0.21% rise in SGOT levels leads to a decrease in TGL levels.

SGPT: Correlating SGPT of cases with total cholesterol (r value -0.027 and p value – 0.884), HDL (r value -0.202 and p value 0.276), LDL (r value – 0.295 and p value – 0.107) and VLDL (r value – 0.052 and p value – 0.782) proves that no positive or inverse correlation exists between SGPT and total cholesterol, HDL, LDL, VLDL. Correlating SGPT of cases with TGL (r value – 0.466 and p value – 0.008**) shows that SGPT and TGL have inverse correlation r=-0.466, r^2=0.217, p=0.008 (statistically significant) and it shows that 0.21 percent of the variation in SGPT is inversely related to the variation in TGL.

Table 6: Comparison of Lipid parameters according to encephalopathy and non-encephalopathy patients

Lipid parameters	Encephalopathy		P value
	No	Yes	
Total cholesterol (gm/dl	165.33+17.03	175.00+34.02	0.362
TGL (mg/dl)	161.85+35.65	132.75+55.77	0.166
HDL (mg/dl)	34.52+3.87	29.01+2.16	0.010*
LDL (mg/dl)	147.89+32.03	158.50+16.84	0.525
VLDL (mg/dl)	38.44+6.45	43.50+2.65	0.137

The table shows the comparison of lipid parameters among patients with hepatitis, who developed encephalopathy and patients without encephalopathy. The average of total cholesterol (without encephalopathy – 165.33+17.03 and with encephalopathy – 175.00+34.02) showed p value of 0.362, which is statistically insignificant. Similarly, average of TGL (without encephalopathy – 161.85+35.65 and with encephalopathy – 132.75+55.77, p value of 0.166), LDL (without encephalopathy – 147.89+32.03 and with encephalopathy – 158.50+16.84, p value of 0.525) and VLDL (without encephalopathy – 38.44+6.45 and with encephalopathy – 43.50+2.65, p value of 0.137) does not show any statistical significance. There is a statistically significant reduction in HDL levels among the encephalopathy patients (29.01+2.16) with p value of 0.010, compared to non encephalopathy patients (34.52+3.87).

Discussion

As hepatitis is one of the most commonly encountered disease of today, a good understanding of its treatment modalities and prevention is important. Liver being the major organ involved in maintaining homeostasis of carbohydrates, proteins and fats, affection of the liver can cause a wide range of disturbances in the body. Hence, it is very vital to diagnose early and start appropriate treatment. Several studies have put forward theories on how lipid profile is affected in liver disease though a clear understanding is still lacking.

The study group consisted of individuals who aged between 19-35yrs, with an average age group of 25.71+3.91. Majority of the patients belongs to age group of 21-30 years i.e. 26(86.7%) in case group, whereas in control group, majority of the patients belongs to age group of 21-30 years i.e 21(70%). The average age of control group is 24.55+4.08.

In a similar study done by Libo Luo et al,[1] 30 individuals of acute hepatitis were taken with 30 healthy controls. The individuals in study group had an average age of 41.7+17.3. The control group consisted of individuals in 41.7+17.3. The control group consisted of individuals in the age group of 20-63 years with an average age of 46.0+13.6.

In another study done by Fayda AH et al,[8] average age group of cases is 31.6+8.1. The number of controls taken was 21 with an average age group of 31.9+6.9.

In the present study, out of the cases, 30 cases had fever, 21 cases had jaundice, 19 had vomiting, 15 had malaise and 4 had encephalopathy. None of these features were there in the control group. In both the studies (Libo Luo et al[1] and Fayda AH et al[8]) all patients had moderate hepatitis but clinical features were not mentioned in the study. In the present study, all the cases were found to be having hepatitis B. In a similar study by Libo Luo et al,[1] there were 4 cases of hepatitis B, 19 cases of hepatitis E and 7 cases of non-A non-E. The study done by Fayda AH et al,[8] the type of hepatitis was not mentioned.

In the present study, the average of total cholesterol is 166.58+19.44, the average of triglycerides is 158.10+38.87, the average of HDL is 33.81+4.12, the average of LDL is 149.26+30.50 and the average of VLDL is 39.10+6.31.

In a similar study done by Libo Luo et al,[1] they have noticed that levels of total cholesterol, LDL, HDL and apoA1 were reduced.

In another study by Fayda AH et al,[8] out of the cases, the average of total cholesterol is 157.4+51.4, the average of HDL is 25.8+10, the average of ApoA is 183.8+74.2 and the average of ApoB is 128.2+38.8.

In this study, 23 individuals only have come for follow up. The values have shown that serum bilirubin, SGOT, SGPT has improved markedly. The level of total cholesterol and HDL has increased and triglyceride has decreased.

In a similar study by Libo Luo et al[1] and Fayda AH et al,[8] they have not done follow up study. In this study, 30 individuals were included in the study group along with an equal number of control groups. The serum bilirubin of the patients with hepatitis were correlated with total cholesterol, triglycerides, HDL, LDL and VLDL and it showed that no positive or inverse correlation exists between serum bilirubin levels and total cholesterol among the cases with hepatitis. Whereas there is a inverse correlation shown between serum bilirubin and TGL among cases, which is statistically insignificant.

In a similar study by Nayak et al,[9] showed that reduced HDL levels was associated with increased bilirubin levels but the correlation was found to be non significant.

In another study by Goel VK et al,[10] showed that, there was substantial correlation between HDL and serum bilirubin. In the present study, SGOT of the patients with hepatitis were correlated with total cholesterol, triglycerides, HDL, LDL and VLDL and it showed that no positive or inverse correlation exists between SGOT and total cholesterol, HDL, LDL, VLDL whereas SGOT and TGL have inverse correlation which is statistically significant. In a similar study done by Nayak et al,[9] showed that reduced HDL levels was associated with increased SGOT levels but the correlation was found to be statistically non significant. In the present study, SGPT of the patients with hepatitis

were correlated with total cholesterol, triglycerides, HDL, LDL and VLDL and it showed that no positive or inverse correlation exists between SGPT and total cholesterol, HDL, LDL, VLDL whereas SGPT and TGL have inverse correlation which is statistically significant. In a similar study by Nayak et al,[9] showed that reduced HDL levels was associated with increased SGPT levels but the correlation was found to be statistically non significant. In another study by Goel VK et al,[10] showed that there was low correlation of HDL with SGPT and alkaline phosphatise. LDL, VLDL and triglycerides also showed low correlation with conventional liver function test. In the present study, Lipid parameters of patients with hepatitis were compared with patients who developed encephalopathy and patients without encephalopathy. The average of total cholesterol showed p value which is statistically insignificant. Similarly, average of triglycerides, LDL and VLDL showed p value which is statistically not significant. There is a statistically significant reduction in HDL levels among the encephalopathy patients compared to non encephalopathy patients.

Conclusion

Comparison of SGOT and SGPT with lipid parameters shows an inverse correlation between SGOT, SGPT and triglycerides. Comparison of lipid parameters with patients who developed encephalopathy and without encephalopathy shows a reduction in HDL levels among the encephalopathy patients which indicating that it might be used as a prognostic marker to assess severity of liver disease.

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