

## Study of Lipid Profile in Acute Viral Hepatitis

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### ABSTRACT

**Introduction:** The viral hepatitis is an infectious disease caused by various hepatitis viruses which affect liver predominantly. Early prediction of impending complication is imperative to modify the cause and prognosis. Viral hepatitis may disturb metabolism of variable substances. This study ascertains the plasma lipid and lipoprotein pattern in subjects suffering from acute viral hepatitis and the change at six weeks follow up.

**Aims and Objectives:** (1) To study the changes in lipid and lipoprotein in acute viral hepatitis. (2) To study correlation between lipid and lipoprotein changes and severity of acute viral hepatitis. (3) To study the utility of lipid profile changes in prognosis of acute viral hepatitis.

**Materials and Methods:** This was hospital based prospective and follow up study, conducted on 50 patients who attended the Medicine department at R N T Medical College and MBGH, Udaipur.

**Results:** Out of 50 patients, majority were in between 21-40 years. 36(72%) were male. 29(58%) were positive for Hepatitis A, 2(4%) for Hepatitis B and 19(38%) for hepatitis E virus. Total cholesterol, triglyceride, LDL-C and VLDL-C were significantly raised, and HDL-C was significantly decreased at presentation as compared to follow up. There was no significant difference in lipid profile changes between viral hepatitis caused by different viruses. HDL-C was also significantly low in subjects who expired in comparison to who recovered.

**Conclusion:** The estimation of lipid levels is required for better assessment of hepatic function; prognostic assessment and management of viral hepatitis subjects. Increase in HDL-C during follow up in viral hepatitis appears to be a bad prognostic sign.

**Key words:** Viral Hepatitis, Lipid Profile, HDL-C.

### INTRODUCTION

Acute viral hepatitis is one of the most frequently seen diseases in day to day practice and causes considerable mortality and morbidity. Early prediction of impending complication is imperative to modify the cause and prognosis.

Liver plays an important role in metabolism of carbohydrates, lipids, proteins, drugs and hormones; hence disease of liver parenchyma like viral hepatitis may disturbs normal homeostasis of metabolism of variable substances.

Liver is most important organ for metabolism for lipids, lipoproteins and apolipoproteins. Under normal circumstances, most plasma endogenous lipids and lipoproteins are synthesized in the liver and then secreted into blood circulation. <sup>[1,2]</sup> Plasma lipoproteins are mainly catabolized by liver to maintain the relative balance of lipid and lipoprotein metabolism in vivo. <sup>[3]</sup>

It is not surprising that liver diseases often not only affect the concentration of various lipids but also may alter their distribution within lipoproteins. It has been

well documented that liver dysfunction might interfere lipid metabolism in vivo and could change plasma lipid and lipoprotein patterns. [4] The obstructive and chronic liver parenchymal diseases cause different patterns of lipid abnormalities is now firmly established. [5,6]

A vast array 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 patient. As the patient begins to recover from viral hepatitis, there is concomitant recovery of lipoproteins. In subjects with fulminant disease who fail to recover, the lipoproteins fail to come back to normal values. Because of this it has been suggested that absence of lipoproteins may be of prognostic significance in subjects of viral hepatitis. [7] Hence investigations of lipid profile in viral hepatitis could aptly assess the prognosis.

Previous studies [8,9] paid more emphasis on changes of lipid metabolisms in chronic hepatitis and cirrhosis with or without hepatocellular carcinoma. The present prospective study ascertains the plasma lipid and lipoprotein pattern in subjects suffering from acute viral hepatitis and the change at six weeks follow up.

**Aims and Objectives**

1. To study the changes in lipid and lipoprotein in acute viral hepatitis.
2. To study correlation between lipid and lipoprotein changes and severity of acute viral hepatitis.
3. To study the utility of lipid profile changes in prognosis of acute viral hepatitis.

**MATERIALS AND METHODS**

This was hospital based prospective and follow up study, conducted on 50patients who attended the Medicine department at R N T Medical College and MBGH, Udairpur (Raj.). Diagnosis of acute viral hepatitis was established by history, physical examination and biochemical parameters.

**INCLUSION CRITERIA**

1. Patients aged above 18 years.
2. All patients suffering from acute viral hepatitis

**EXCLUSION CRITERIA**

1. Alcoholic
2. Cirrhosis of liver
3. Hepatitis due to other cause
4. Diabetes mellitus
5. Pregnancy
6. Nephrotic syndrome
7. Thyroid dysfunction
8. Subjects taking
  - o Lipid lowering agents
  - o Steroids
  - o Anti-neoplastic drugs

Routine blood investigations including liver function tests, PT/INR, and 8 hours fasting lipid profile were done. The lipid and lipoprotein assay were done in SIEMENS Dimension Clinical Chemistry System using Flex Reagent Cartilage. All subjects were followed up after 6 weeks and LFT, PT/INR and fasting lipid profile were repeated.

**OBSERVATIONS**

In the present study, analysis was carried out at baseline ad 6 weeks later. The demographic and investigational outcomes of 50 acute viral hepatitis subjects are as follows:

11 subjects (22%) were less than 20 years, 28 (56%) were between 21-40 years, 9 (18%) were between 41-60 years and 2 (4%) were above 60 years of age.

**Table 1: Gender wise distribution of causative virus**

		Causative Virus				Total
		HAV	HBV	HCV	HEV	
MALE	Count	24	2	0	10	36
	%	48	4	0	20	72
FMEALE	Count	5	0	0	9	14
	%	10	0	0	18	28
TOTAL	Count	29	2	0	19	50
	%	58	4	0	38	100

Out of 50 subjects, 36(72%) were males and 14(28%) were females. Among 36 males, 24 were due to Hepatitis A, 2 were due to Hepatitis B and 10 were due to Hepatitis E. Among 14 female subjects, 5 were due to Hepatitis A and 9 were due to Hepatitis E.

Total 29 were positive for Hepatitis A, 2 for Hepatitis B and 19 for hepatitis virus. Hepatitis C was not detected in any subjects.

**Table 2: Clinical features of acute viral hepatitis**

	Fever	Malaise	Vomiting	Jaundice	Encephalopathy
HAV	1	21	20	29	5
HBV	1	2	1	2	1
HEV	0	17	12	19	2
Others	0	0	0	0	0
Total	2	40	33	50	8

Based on symptomatology, all 50 patients presented with jaundice (100%). Malaise was present in 40 (80%) patients whereas vomiting reported in 33(66%) patients. 8(16%) had encephalopathy at onset and 2(4%) were febrile at presentation.

**Table 3: Comparison of lipid profile at baseline and 6-weeks follow up**

Biochemical parameter	Viral Hepatitis (n=50) Mean ± SD mg/dl	Follow up (n=50) Mean ± SD mg/dl	P -Value
Total CL	218.3±23.9	147.98±29.27	< 0.001
Triglyceride	147.9±32.57	139.86±21.55	< 0.001
HDL-C	21.53±7.02	39.70±7.33	< 0.001
LDL-C	162.1±27.17	80.56±25.52	< 0.001
VLDL-C	34.59±6.60	27.97±4.31	< 0.001

Total cholesterol at baseline (218.3±23.9 mg/dl) when compared to those at 6 weeks follow up (147.98±29.27 mg/dl) was significantly high (p<0.001).

Total triglycerides at baseline (147.9±32.57 mg/dl) when compared to those at 6 weeks follow up (139.86±21.55 mg/dl) was significantly high (p<0.001).

HDL-C at baseline (21.53±7.02 mg/dl) when compared to those at 6 weeks follow up (39.70±7.33 mg/dl) was significantly low (p<0.001).

LDL-C at baseline (162.1±27.17 mg/dl) when compared to those at 6 weeks follow up (80.56±25.52 mg/dl) was significantly high (p<0.001).

VLDL-C at baseline (34.59±6.60 mg/dl) when compared to those at 6 weeks follow up (27.97±4.31mg/dl) was significantly high (p<0.001).

**Table 4: Comparison of lipid profile in recovered and expired subjects**

Biochemical parameter	Subjects recovered (n=47) Mean ± SD mg/dl	Subjects expired (n=3) Mean ± SD mg/dl	P -Value
Total CL	218.38±23.34	188.0±72.77	NS
Triglyceride	174.90±32.57	197.0±40.95	NS
HDL-C	21.53±7.02	10.23±2.26	< 0.05
LDL-C	162.1±27.17	138.7±83.24	NS
VLDL-C	34.59±6.60	39.07±8.55	NS

Total cholesterol was low (188.0±72.77 mg/dl) in subjects who succumbed in comparison to those who completely recovered (218.38±23.34 mg/dl). This change was not statistically significant (p>0.05).

Triglyceride was low (197.0±40.95 mg/dl) in subjects who succumbed in comparison to those who completely recovered (174.90±32.57 mg/dl). This change was not statistically significant (p>0.05).

HDL-C was low (10.23±2.26 mg/dl) in subjects who succumbed in comparison to those who completely recovered (21.53±7.02 mg/dl). This low HDL-C was statistically significant (p<0.05).

LDL-C was low (138.7±83.24 mg/dl) in subjects who succumbed in comparison to those who completely recovered (162.1±27.17 mg/dl). This change was not statistically significant (p>0.05).

VLDL-C was high (39.07±8.55 mg/dl) in subjects who succumbed in comparison to those who completely recovered (34.59±6.60 mg/dl). This change was not statistically significant (p>0.05).

**Table 5: Comparison of lipid profile changes between different viruses**

Biochemical parameter	HAV (n=29) Mean ± SD mg/dl	HBV (n=50) Mean ± SD mg/dl	HEV (n=50) Mean ± SD mg/dl	P -Value
Total CL	214±25.19	231.50±0.7	218.89±34.31	NS
Triglyceride	179.45±39.46	169.50±12.02	172.0±22.44	NS
HDL-C	22.11±7.79	14.95±9.69	19.55±6.2	NS
LDL-C	157.76±32.17	185.25±4.31	162.59±32.36	NS
VLDL-C	35.73±7.92	31.30±6.08	33.91±4.40	NS

Total cholesterol in subjects with Hepatitis A was 214±25.19 mg/dl, Hepatitis B was 231.50±0.7 mg/dl and Hepatitis E was 218.89±34.31 mg/dl. The variation in increase of total cholesterol among subjects with different hepatitis was not statistically significant (p>0.05).

Triglyceride in subjects with Hepatitis A was 179.45±39.46 mg/dl, Hepatitis B was 169.50±12.02 mg/dl and Hepatitis E was 172.0±22.44 mg/dl. The variation in increase of triglyceride among subjects with different hepatitis was not statistically significant (p>0.05).

HDL-C in subjects with Hepatitis A was 22.11±7.79 mg/dl, Hepatitis B was 14.95±9.69 mg/dl and Hepatitis E was 19.55±6.2 mg/dl. The variation in decrease of HDL-C among subjects with different hepatitis was not statistically significant (p>0.05).

LDL-C in subjects with Hepatitis A was 157.76±32.17 mg/dl, Hepatitis B was 185.25±4.31mg/dl and Hepatitis E was 162.59±32.36 mg/dl. The variation in increase LDL-C among subjects with different hepatitis was not statistically significant (p>0.05).

VLDL-C in subjects with Hepatitis A was 35.73±7.92 mg/dl, Hepatitis B was 31.30±6.08 mg/dl and Hepatitis E was 33.91±4.40 mg/dl. The variation in increase of VLDL-C among subjects with different hepatitis was not statistically significant (p>0.05).

## DISCUSSION

The viral hepatitis is one of the infectious diseases caused by various

hepatitis viruses (HAV, HBV, HCV, HDV, HEV) which affect liver predominantly. The incidences of this disease are increasing worldwide and also in developing countries like India. These increase incidences may be due to poor sanitation, overcrowding, contaminated blood and blood product transfusion, use of unsterilized instruments and sexual promiscuity.

The present study was carried out in 50 patients above the age of 18 years. Majority of patients in present study were in between 21-40 years of age group, 28 patients (56%) followed by 11 patients (22%) in age group less than 20 years. Basavraj Patil et al <sup>[10]</sup> studied 30 patients of age group 19-35 years and he observed that the maximum numbers of the patients were in 21-30 years of age group and it was 26(86%). The results of above study didn't match with our result due to selection of the patients between 18-70 years of age.

The present study was done in 50 patients. We found that 36 patients (72%) were male and rest 14 patients (28%) were female. Among 36 males, 24 were due to Hepatitis A, 2 were due to Hepatitis B and 10 were due to Hepatitis E. Among 14 female patients, 5 were due to Hepatitis A and 9 were due to Hepatitis E.

Basavraj Patil et al, <sup>[10]</sup> studied 30 patients of acute viral hepatitis and found 10(33.33%) patients were male and 20 (66.67%) were females.

Prasanta Kumar Bhattacharya et al, <sup>[11]</sup> studied over 50 patients and observed 26 (52%) patients were male and 24 (48%) were females.

Study was carried out in 50 patients of viral hepatitis and observed, 29 were positive for Hepatitis A (58%), 2 for Hepatitis B (4%) and 19 for hepatitis E virus (38%) and Hepatitis C was not detected in any of the patient.

Prasanta Kumar Bhattacharya et al, [11] studied and found similar type of results. In his study he found HAV (52%), HBV (14%), HCV (4%) and HEV (30%). In the present study, all 50 patients (100%) were icteric followed by malaise in 40 patients (80%), vomiting in 33 (66%), encephalopathy in 8 patients (16%) and 2 had fever (4%).

Prasanta Kumar Bhattacharya et al. [11] showed the similar results in which most common finding was icterus and high coloured urine. Other common symptoms were anorexia, nausea and vomiting, hepatomegaly and fever.

The present study which was carried out in 50 patients of viral hepatitis which undergone 8 hours fasting lipid profile at the time of presentation and at follow up after 6 weeks. Total cholesterol was significantly higher at presentation as compared to follow up ( $p < 0.001$ ). The probable explanation for this raised serum total cholesterol was due to decreased lecithin cholesterol acyl transferase (LCAT) activity in viral hepatitis and may be due to intrahepatic biliary obstruction.

McIntyre N et al, [12] showed that the total serum cholesterol remained unaltered in infective hepatitis as compared controls.

In yet another research, Goel VK et al, [13] showed that values of serum cholesterol remain unaltered in viral hepatitis irrespective to whether the patient is in coma or not.

Serum triglycerides levels were significantly higher during presentation when compared with follow up ( $p < 0.001$ ). Similar observations were found by study done by McIntyre et al. [12]

HDL-C at presentation when compared to those at 6 weeks follow up was significantly low ( $p < 0.001$ ). In addition, it was also observed that the HDL-C values

were significantly lower in subjects who expired as compared to those who recovered completely thus underlying the prognostic utility of HDL-C.

McIntyre N et al, [12] also observed that the HDL-C was decreased in subjects with acute viral hepatitis and attributed this decrease in HDL-C to decreased production of enzyme LCAT.

Irshad M et al, [14] studied on 50 patients of acute viral hepatitis, it was observed that HDL-C level was significantly decreased irrespective to viral etiology.

LDL-C at presentation was significantly high as compared to follow up ( $p < 0.001$ ). Goel VK et al, [13] found the same results.

VLDL-C in patients with acute viral hepatitis significantly higher when compared to follow up ( $p < 0.001$ ).

Goel VK et al, [13] observed that there was a significant decrease in VLDL cholesterol in subjects with viral hepatitis when compared to controls. Unlike the HDL-C which was significantly decreased in those subjects who expired when compared to those who recovered completely, no significant difference was observed in VLDL-C values in relation to mortality.

## **CONCLUSION**

The present study of lipid profile in viral hepatitis clearly establishes the clinical utility of estimation of lipid levels for better assessment of hepatic function, prognostic assessment and management of viral hepatitis subjects. Total cholesterol, triglyceride, LDL-C and VLDL-C were significantly raised, and HDL-C was significantly decreased at presentation as compared to follow up. There was no significant difference in lipid profile changes between viral hepatitis caused by different viruses. HDL-C was also significantly low in subjects who expired in comparison to who recovered. Hence, we conclude that increase in HDL-C during follow up in viral hepatitis appear to be a bad prognostic sign. Therefore, estimation of serum HDL-C allows better assessment

of hepatic function and evaluation of prognosis of subjects with acute viral hepatitis.

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How to cite this article: Dave M, Sharma SK, Surjan T. Study of lipid profile in acute viral hepatitis. *International Journal of Research and Review*. 2019; 6(7):339-344.

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