

Lipid profile in acute viral hepatitis: A study from north eastern India

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Abstract

Background: Acute viral hepatitis is associated with significant hepatic dysfunction including lipid metabolism. Variations in the serum lipid fractions in acute viral hepatitis due to hepatic injury may serve as prognostic marker for severity of hepatic injury.

Methods: Hospital-based prospective study on acute viral hepatitis. Fifty cases of acute viral hepatitis and 50 age and sex matched controls, (>18 years) were studied. Routine biochemical investigation, hepatotropic viral serology and fasting serum lipid fractions were analysed for changes in patients with acute viral hepatitis. Chi-square test was used for comparing variables. A 'p value' <0.05 was considered statistically significant.

Results: Baseline parameters were similar between cases and controls ($p>0.05$). Jaundice (100%) and high coloured urine (100%) were the commonest presentations. Hepatitis A virus (52%) and Hepatitis E virus (30%) were commonest aetiological agents. Serum cholesterol, triglycerides, low density lipoprotein (LDL) were significantly higher ($p<0.01$) while high density lipoprotein (HDL) was significantly lower ($p<0.01$) in acute viral hepatitis compared to controls. There was no statistically significant difference in the levels of very LDL between cases and controls ($p>0.05$). Complications were seen in 7 (14%) cases with hepatic encephalopathy being the commonest (6%). Serum cholesterol, triglycerides, LDL were significantly higher and HDL was significantly lower in hepatitis with complications compared to uncomplicated hepatitis ($p<0.01$).

Conclusion

Acute viral hepatitis leads to significant alterations of serum lipid fractions which may serve as an indicator of severity of liver damage and be helpful in assessing the prognosis of patients with acute viral hepatitis.

Keywords: Acute viral hepatitis, serum lipids, comparison

1. Introduction

Acute viral hepatitis (AVH) continues to be a major public health burden in developing countries like India [1]. Studies have previously documented a variable prevalence of hepatotropic viruses: Hepatitis A Virus (HAV) (1.7-67%), Hepatitis B Virus (HBV) (7.3-42%), Hepatitis C Virus (HCV) (1.16-10.6%) and HEV (Hepatitis E Virus) (16.3-66.3%) [2-5].

Liver is the principal organ involved in lipid metabolism. In physiological circumstances most lipids are initially synthesized in liver and then introduced in the systemic circulation [6]. In the setting of acute or chronic hepatic dysfunction circulating lipids and lipoproteins are altered with respect to quantity as well as electrophoretic mobility and appearance [7].

Previous studies have documented the alterations of serum lipids in patients suffering from acute hepatitis due to hepatotropic virus [8]. However, there is a scarcity of literature especially from the north-eastern part of India about the changes in serum lipid profiles in the setting of acute viral hepatitis. Therefore, this study was carried out in a tertiary care teaching hospital situated in north east India to study the patterns of lipid profile abnormalities in patients with acute viral hepatitis.

2. Materials and Methods

The present study was a hospital-based prospective study, carried out between July 2012 and June 2013, in patients with acute viral hepatitis admitted to general medical ward (MW) of Gauhati Medical College & Hospital, Guwahati, a tertiary care hospital in northeast India catering to several north eastern states of the country.

Fifty patients, age 18 years and above, diagnosed as cases of acute viral hepatitis as per the selection criteria and fifty healthy age and sex matched controls were enrolled for the study. Cases and controls were matched according to their age, sex, body mass index (BMI), socioeconomic status.

2.1 Inclusion Criteria

Any patient above >18 years of age admitted to the general MW with clinical and laboratory evidence of acute viral hepatitis.

2.2 Exclusion Criteria

Patients with co-morbidities which can affect blood lipids and lipoproteins levels were excluded.

Detailed history, clinical examination and laboratory tests (complete blood counts, blood urea, serum creatinine, blood glucose, liver function tests, lipid profile, serological testing for hepatotropic viruses and coagulogram), were done in all cases. Blood samples were collected from all subjects under standardised conditions after overnight fasting. All laboratory parameters were directly determined on an automatic biochemical analyzer. Demographic data, clinical features, relevant laboratory parameters and the clinical course of the cases and controls were recorded in a pre-tested structured proforma.

2.3 Ethics

Ethical clearance was taken from the Institutional Ethical Committee and written informed consent was taken from all the patients included in the study.

2.4 Statistics

Statistical Analyses were done using Statistical Package for Social Survey (SPSS) for Windows version 17.0. A 'p value' <0.05 was considered as statistically significant. The results were tabulated and graphically represented using Microsoft Office for Windows 2008.

3. Results

Based on the selection criteria 50 cases of acute viral hepatitis and 50 controls were included in the study. The baseline characteristics of the cases and controls did not have any significant differences ($p>0.05$), (Table 1). Jaundice (100%) and high coloured urine (100%) were the commonest clinical manifestations (Table 2). Patients with acute viral hepatitis had a significantly higher level of serum bilirubin and alanine transaminase ($p<0.01$) (Table 3). Hepatitis A virus (52%) was the commonest aetiological agent followed by Hepatitis E virus (30%), (Table 4).

Serum lipid profile with respect to total serum cholesterol, triglycerides, high density lipoprotein, low density lipoprotein and very low density lipoprotein of the patients with acute viral hepatitis and the controls are shown in Table 5. Serum cholesterol, triglycerides, low density lipoprotein were significantly higher ($p<0.01$) in patients with acute viral hepatitis while high density lipoprotein was significantly lower ($p<0.01$), compared to controls. There was no statistically significant difference in the levels of very low

density lipoprotein between the two groups. Complications were seen in 7 (14%) of the cases of hepatitis with hepatic encephalopathy being the commonest (6%) (Table 6). Serum cholesterol, triglyceride and LDL levels were significantly higher in patients with complications and HDL were significantly lower ($p<0.01$) in comparison to those with uncomplicated hepatitis (Table 7).

Table 1: Baseline demographic characteristics of patients with acute viral hepatitis and controls

Parameter	Cases	Controls	P value
Mean age (years)	25.68	24.2	>0.05
Sex			
Male	24	22	>0.05
Female	26	28	>0.05
Mean body mass index (kg/m ²)	19.61	19.26	>0.05
Per capita income (INR)	3356	3255	>0.05

Table 2: Clinical manifestations of patients with acute viral hepatitis

Clinical Presentation	No. of Cases	
	n	%
Anorexia	45	90
Nausea/ Vomiting	35	70
Weakness	35	70
Fever	35	70
Jaundice	50	100
Dark Yellow Urine	50	100
Pain Abdomen	15	30
Pruritus	10	20
Hepatomegaly	36	72
Splenomegaly	7	14

Table 3: Liver function tests in patients with acute viral hepatitis and controls

Parameter	Cases	Controls	P value
Mean Total Serum Bilirubin	9.90±4.64	0.748±0.35	<0.01
Mean Alanine Transaminase (ALT)	963.14±12.38	48.38±14.19	<0.01

Table 4: Aetiological agent of acute viral hepatitis

Sero-type of hepatitis	No of cases	
	n	%
Hepatitis-A	26	52
Hepatitis-B	7	14
Hepatitis-C	2	4
Hepatitis-D	0	0
Hepatitis-E	15	30
Total	50	100

Table 5: Serum lipid profiles of patients with acute viral hepatitis and controls

Parameter	Cases	Controls	P value
Total cholesterol	228.84±31.34	90.68±24.19	<0.01
Serum triglycerides	164.6±70.82	111.18±24.15	<0.01
High density lipoprotein	12.21±5.59	47.18±8.37	<0.01
Low density lipoprotein	148.53±25.93	77.04±20.92	<0.01
Very low density lipoprotein	28.44±9.46	30.54±12.17	0.3381

Table 6: Complications in acute viral hepatitis

Complications	No. of Cases (%)	
	n	%
Bleeding	2	4
Hepatic encephalopathy	3	6
Fulminant hepatic failure	2	4
Hepato renal syndrome	0	0

Table 7: Lipid profile in patients with complicated viral hepatitis and those without complications

Parameter	Complicated viral hepatitis	Uncomplicated viral hepatitis	p value
Total cholesterol	250.71 ±80.80	150.04±8.73	<0.01
Serum triglycerides	323.25±144.31	149.93 36.40	<0.01
Low density lipoprotein	150.23±21.42	66.04±20.44	<0.01
High density lipoprotein	5.5±0.52	12.64±5.60	<0.01

4. Discussion

Liver forms the central organ in the metabolism of lipids. Majority of the plasma lipids have their synthetic pathway in the liver and thus an intact cellular function is a pre-requisite for a balanced lipid metabolism [6]. Besides the synthesis of lipoproteins certain key enzymes of lipid metabolism including lecithin cholesterol acyltransferase (LCAT), hepatic lipase are also synthesized in the liver [9]. Furthermore, liver also regulates the catabolism of plasma lipoproteins by lipoprotein receptors located on the hepatic cell surface thus maintain a degree of equilibrium between the lipoprotein fractions [10]. Therefore, analysis of plasma lipids and lipoprotein levels becomes helpful to ascertain the extent of the hepatic damage which occurs in patients with acute viral hepatitis.

In our study the cases of acute viral hepatitis were matched with appropriate controls which form an essential pre-requisite prior to comparison of differences in lipid profiles. Hepatitis A virus was the commonest aetiological agent followed by Hepatitis E virus. Similar results have been documented in previous studies from India [1,2]. All the

patients of acute hepatitis were icteric and had high coloured urine. The other common symptoms were anorexia, nausea and vomiting, hepatomegaly and fever. A previous study from India also has shown similar results with jaundice being the commonest symptom [1].

The alterations in the serum lipid fractions showed significant differences in patients with acute viral hepatitis and those without in our side. While, total cholesterol, triglycerides and low density lipoprotein levels were significantly higher in patients with hepatitis the levels of high density lipoproteins were significantly lower. In as early as 1862, Austin Flint suggested that blood cholesterol level were raised in hepatic [11]. However, in another study it was shown that total cholesterol levels were lower in patients with acute viral hepatitis [12]. Elevated levels of serum triglycerides and very low density lipoproteins in acute hepatitis have also been documented in a previous study which is in conjunction with our findings [8].

Low levels of high density lipoproteins as documented in our study have also been reported from previous studies [8, 12]. According to available literature high density lipoprotein may serve as one of the best indicator of liver damage [8, 12]. While evaluating liver functions literature suggests that an overall combination of prothrombin activity, serum bilirubin and hepatic transaminase levels and the high density lipoprotein fraction may serve to provide a useful index for evaluating the prognosis in acute hepatitis [8].

5. Conclusion

Acute viral hepatitis leads to significant alterations of the lipid fractions in blood. While total cholesterol, triglycerides and low density lipoprotein levels are higher, the levels of high density lipoproteins are lower. Variations in lipid fractions may serve as an indicator of severity of liver damage and be helpful in assessing the prognosis of patients with acute viral hepatitis.

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