



LIPID PROFILE CHANGES IN ACUTE VIRAL HEPATITIS: A STUDY FROM A TERTIARY CARE CENTRE OF EASTERN INDIA

Medicine

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ABSTRACT

Background: Liver is the most important organ for the metabolism of lipids, lipoproteins and apolipoproteins. Acute viral hepatitis is associated with significant hepatic dysfunction including lipid metabolism due to hepatic injury which may serve as prognostic marker for severity of hepatic injury.

Methods: The present Hospital-based retrospective study examined plasma lipid profiles in thirty patients suffered from acute viral hepatitis. Patients' blood samples were collected at both the debut and recovery of diseases. Routine biochemical investigation, hepatotropic viral serology and fasting serum lipid fractions were analysed for changes in patients with acute viral hepatitis.

Results: Jaundice (100%) and high coloured urine (100%) were the commonest presentations. Hepatitis A virus (47%) and Hepatitis B virus (33%) were commonest aetiological agents. Serum cholesterol, triglycerides, low density lipoprotein (LDL) were significantly higher while high density lipoprotein (HDL) was lower or normal in acute viral hepatitis compared to normal value and those became normal after recovery from the acute hepatitis. Complications were seen in 3(12%) cases with hepatic encephalopathy being the commonest (8%).

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

Viral Hepatitis, Lipid profile, Prognosis

INTRODUCTION:

Liver inflammation leads to disrupt lipid profile. Acute viral hepatitis mainly caused by Hepatitis A, B and E viruses and very rarely by Hepatitis C virus. 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 hepatitis¹. Serum levels of total cholesterol, HDL-C and apoAI were significantly lower in the acute hepatitis patients, whereas serum levels of TG and LDL-C were obviously higher in those patients². It has been well documented that chronic liver dysfunction might interfere lipid metabolism in vivo and could change plasma lipid and lipoprotein patterns³. Previous studies pay more emphasis on changes of lipid metabolisms under chronic hepatitis and cirrhosis with or without hepatocellular carcinoma^{4,5}.

In this study we have investigated the levels of cholesterol, triglycerides, high – density lipoprotein and low – density lipoprotein in sera of 30 acute viral hepatitis patients. We will compare the correlations between lipids parameters and hepatitis indices (bilirubin total and direct, albumin, alanine transaminase, aspartate transaminase, and alkaline phosphatase).

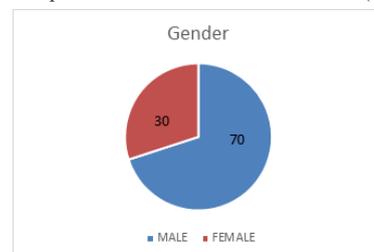
MATERIALS AND METHODS:

This is a retrospective study was conducted at School of Tropical Medicine, Kolkata on consecutive 30 patient admitted with jaundice with positive any viral serology (HBsAg, Anti HAV, Anti HEV) admitted from 1st January 2016 to 31st December 2016. The serological diagnosis was done using Rapid Immunochromatographic Card Test (RICT) in the Virology laboratory of the hospital. A pre-designed proforma was used to collect information from the hospital records. By using the proforma which included history, clinical manifestations, biochemical tests and serological tests, data were collected from hospital records of total 30 serologically diagnosed cases. Blood was collected from all subjects under standardized conditions after overnight fasting after admission and recovery. Standard statistical methods were used for data analysis. Inclusion criteria for this study was any patient above 18 years of age admitted at Carmichel Hospital

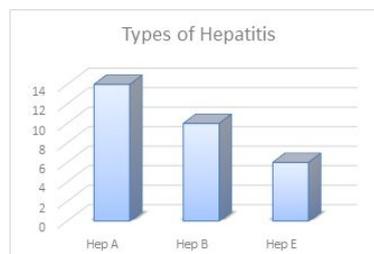
for Tropical Diseases with clinical and laboratory evidence of acute viral hepatitis and exclusion criteria was patients with established chronic liver disease and others co-morbidities which can affect blood lipids and lipoproteins levels were excluded.

RESULTS:

In this study age of the patients range from 20- 68 years with mean 37.5 and median 35 years. Most of the patients were in 2nd and 3rd decade of life. Most of the patients were male with male: female (2.3:1).



Hepatitis A virus (47%) and Hepatitis B virus (33%) were commonest aetiological agents.



Jaundice (100%) and high coloured urine (100%) were the commonest presentations. Most common findings were high triglyceride in all patients in acute phase of disease. High triglyceride level (161-

449mg/dl) was found in 22 patients and more than 450mg/dl was found in 8 patients. Total cholesterol level more than 200mg/dl was found in 17 patients. LDL level more than 100mg/dl level was found in 20 patients. HDL level less than 44mg /dl were found in 26 patients. Serum lipid profile with respect to total cholesterol, triglyceride, low density lipoprotein (LDL) and high density lipoprotein (HDL) of the patients with acute viral hepatitis and convalescent phase are shown in table 1&2. Complications were seen in 3(12%) cases with hepatic encephalopathy being the commonest (8%).

Table 1: Serum lipid profile in acute viral hepatitis and after recovery

parameter	Acute stage (mg/dl)	During discharge (mg/dl)
T cholesterol	212±112	176±100
Triglyceride	377±183.39	170±69
L D L	150±70	96±30
H D L	35±10	40.5±17

Table 2 : Lipid profile of acute and convalescent phase

	Acute (patients)	Convalescent (patients)	
T chol	≤200mg/dl	13	27
	≥201mg/dl	17	3
TG	≤160mg/dl	0	4
	161-449mg/dl	22	26
	≥450mg/dl	8	0
L D L	≤99mg/dl	10	19
	≥100mg/dl	20	11
H D L	≤44mg/dl	26	22
	≥45mg/dl	4	8

DISCUSSION:

Liver forms the central organ in the metabolism of lipid. Majority of the plasma lipids have their synthetic pathway in the liver and thus an intact cellular function is needed for balance lipid metabolism. Besides the synthesis of lipoproteins certain key enzymes of lipid metabolism including lecithin cholesterol acyltransferase (LCAT), hepatic lipase are also synthesized in the liver.⁸ As in blood circulation lipids do not dissolve in plasma, they need in combination with different apolipoproteins to form lipoproteins that may transfer endogenous or exogenous lipids to different organs or tissues for further metabolism. Under normal physiological conditions, liver plays an important role to regulate lipid and lipoprotein metabolisms. Liver not only synthesizes and secretes endogenous lipoprotein, synthesis of key enzyme for the LDL metabolism, i.e., lecithin cholesterol acyltransferase (LCAT), hepatic lipase and apolipoproteins, but also regulates catabolism of various plasma lipoproteins via hepatic cellular surface lipoprotein receptors, which may maintain relative equilibrium of plasma lipids and lipoproteins in vivo.⁹ These processes could be interfered or impaired when hepatic cellular damage, which leads to an alteration of plasma lipid and lipoprotein patterns. Apart from disruption of liver function test, dyslipidemia also a marker of impaired liver function in acute hepatitis.

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

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