(International Print/Online Journal)

SJIF IMPACT FACTOR: 4.617
GADIF IMPACT FACTOR: 1.49

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume1, Issue 2, Page No: 165-173
July-August 2018

Co-relation of Lipid Profile In Patients With Acute Viral Hepatitis- A Hospital Based Study

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Type of Publication: Original Research Paper
Conflicts of Interest: Nil

ABSTRACT

Background: Viral hepatitis is a disease of antiquity. Viral hepatitis which is one of the commonest disease in day to day practice causes considerable mortality and morbidly. Early prediction of impending complications is imperative to modify its course and prognosis.

Methods: A case-control study on acute viral hepatitis was conducted in which forty cases of acute viral hepatitis and forty 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. Students T-test and Chi-square test was used for comparing variables. A 'p value' <0.05 was considered statisticallysignificant.

Results: Baseline parameters didn't differed significantly between cases and controls (p>0.05). The triglycerides, cholesterol and LDLc were significantly raised when compared to control. HDLc was significantly decreased when compared to the control in the present study. HDLc values were significantly much lower in patient with viral hepatitis with encephalopathy and in patients who expired as compared to those who recovered thus underlying the prognostic utility of HDLc.

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, cases, control

INTRODUCTION

The study of liver is as ancient as 2000 BC when during the reign of King Hammurabi of Babylon priests used it for predicting things to come. Liver is the most important organ for the metabolism of lipids, lipoproteins and apolipoproteins. Under normal circumstances, most plasma endogenous lipids and lipoproteins are synthesized in the liver and then are secreted into the blood circulation [1, 2]. And plasma lipoproteins are also mainly catabolism by the liver to maintain the relative balance of lipid and lipoprotein metabolism in vivo [3]. It has been well documented that chronic liver dysfunction might interfere lipid metabolism in vivo and could change plasma lipid and lipoprotein patterns [4]. Viral hepatitis is one of the frequently seen diseases in day to day practice and causes considerable morbidity and mortality. Early prediction of impending complication is imperative to modify its cause and prognosis. Acute viral hepatitis (AVH) continues to be a major public health burden in developing countries like India [5]. 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%) [6,

7].

In China, the most common etiology of acute hepatitis is viral infection, in which hepatitis A and hepatitis E are the most common causes. In clinical, the courses of acute hepatitis may vary widely from mild symptom that does not require treatment to the fulminant hepatic failure that needs emergency liver transplantation. Acute viral hepatitis is more likely to be asymptomatic in younger people. In addition, acute hepatitis may occur less commonly with infections such as Epstein-Barr virus, cytomegalovirus, adenovirus, herpes simplex and Coxsackie virus or with other noninfectious reasons. It has been demonstrated that in the acute and/or chronic liver diseases, hepatic function could be impaired and the circulating lipids and lipoproteins are not only present in abnormal amounts but they frequently also have abnormal composition including electrophoretic mobility and appearance [4].

A vast array of biochemical 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 a concomitant recovery of lipoproteins. In patients 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 patients of viral hepatitis [8]. Hence investigations of the lipid profile in viral hepatitis could aptly assess the prognosis. Therefore, this study was carried out to study the patterns of lipid profile abnormalities in patients with acute viral hepatitis

MATERIALS AND METHODS

The present study was a hospital based case control prospective study carried out at Government Medical college , Jammu (J&K), India , in Department of internal medicine. Forty patients, age 18 years and above, diagnosed as cases of acute viral hepatitis as per the selection criteria and forty healthy (controls) age and sex matched controls were enrolled for the study after approval was received from the Institutional Ethics Committee for Human Research. Both the cases and controls were explained about the study in detail, following which an informed written consent was taken regarding permission for inclusion in the study. For all Cases, detailed clinical history was taken, clinical examination was done and laboratory investigations that included Complete Blood Count, routine Urine examination, Blood Glucose, Serum Creatinine, Blood Urea, Liver Function Tests, Lipid Profile, Prothrombin Time, Serum Electrolytes, Serological testing for Hepatotropic Viruses and necessary radiological investigations were done. Blood collection for Lipid Profile was done after proper overnight fasting. During hospitalization, all cases were given standard treatment protocol for management of acute viral hepatitis and their clinical condition closely followed. For Controls, healthy individuals were enrolled which were confirmed with detailed history and clinical examination. Blood collection after proper overnight fasting was done for lipid profile estimation.

Inclusion criteria

Any patient above >18 years of age confirmed with acute viral hepatitis through clinical and laboratory evidence were included in the study.

Exclusion criteria:

Subjects suffering from diabetes mellitus, nephrotic syndrome, thyroid dysfunction, cirrhosis of liver, chronic smokers, hepatitis due to other causes and those patients taking drugs which might affect blood lipids and lipoproteins were excluded from the study.

Data analysis:

The descriptive statistics was done using mean with standard deviation (SD) for quantitative variables and categorical variables were presented in frequencies along with respective percentages. The statistical comparisons for quantitative variables was done using Student's 't' test and for categorical variables Chi-square was used as per the nature of data. All statistical analyses were performed by using SPSS software (Version 22, SPSS Inc, Chicago, IL, USA). The p value less than 0.05 was considered as statistically significant.

RESULTS

Based on the selection criteria 40 cases of acute viral hepatitis and 40 controls were included in the study. The baseline characteristics of the cases and

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controls did not showed any significant differences (p>0.05) (Table 1 & 2). The mean age of the patients in cases group was 33.85 ± 13.80, while in control group it was 37.58 ± 13.37. The laboratory findings of liver function tests i.e. serum bilirubin, SGPT, lipids and lipoproteins obtained from controls and subjects are presented in Table 3 and Fig. 1, 2 and 3. It is evident from the Table that in viral hepatitis the levels of triglycerides in cases (168.1 ± 12.9) were significantly higher as compared to control group (138.8 ± 21.7). The levels of LDLc among cases (171.4 ± 13.8) was higher as compared to the control group (77.8 ± 25.7). This increase in LDLc levels in cases with viral hepatitis was highly significant (P<.001). Similarly, the levels of HDLc among cases (20.1 ± 6.9) was significantly decreased when compared to the control group (39.3 ± 7.1), however, the levels of very low density lipoprotein among cases (32.1 ± 2.3) was lower as compared to controls (32.9 ± 2) and the magnitude of decrease of VLDL in cases was not statistically significant. Table 3 also indicates that the levels of serum bilirubin in patients who expired were in the range of (10.8 ± 5.5) when compared to those patients who recovered completely (6.5 ± 3.5) and this increase in serum bilirubin in cases was statistically significant (P<0.05). Further, the difference in the range of hyperbilirubinaemia between the fatal outcome group and recovered group of cases was found to be statistically significant (P<0.05).

Table 1: Age and sex distribution

Age (years)	**Cases**		**Control**	
	Number	**%**	**Number**	**%**
≤20	7	17.50	5	12.50
21-30	11	27.50	8	20.00
31-40	13	32.50	12	30.00
41-50	4	10.00	6	15.00
>50	5	12.50	9	22.50
Mean age ± SD	33.85 ± 13.80		37.58 ± 13.37	
χ^2 value	5.97			
p-value	0.207			

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Table 2: Sex distribution

Sex distribution	Cases		Control	
	Number	%	Number	%
Male	28	70.00	30	75.00
Female	12	30.00	10	25.00
χ^2 value	0.63			
p-value	0.428			

Table 3: Comparison of biochemical parameters between control and viral hepatitis

Biochemical Parameter	Control	Cases	P- Value
Sr. Bilirubin (Mg/d)	0.55 ± 0.32	7.6 ± 4.5	P<.001
SGPT	29.9 ± 11.1	371.3 ± 244.9	P<.001
TC	149.3 ± 24.6	223.5 ± 19.1	P<.001
TG	138.8 ± 21.7	168.1 ± 12.9	P<.001
HDL	39.3 ± 7.1	20.1 ± 6.9	P<.001
LDL	77.8 ± 25.7	171.4 ± 13.8	P<.001
VLDL	32.1 ± 2.3	32.9 ± 2.0	NS

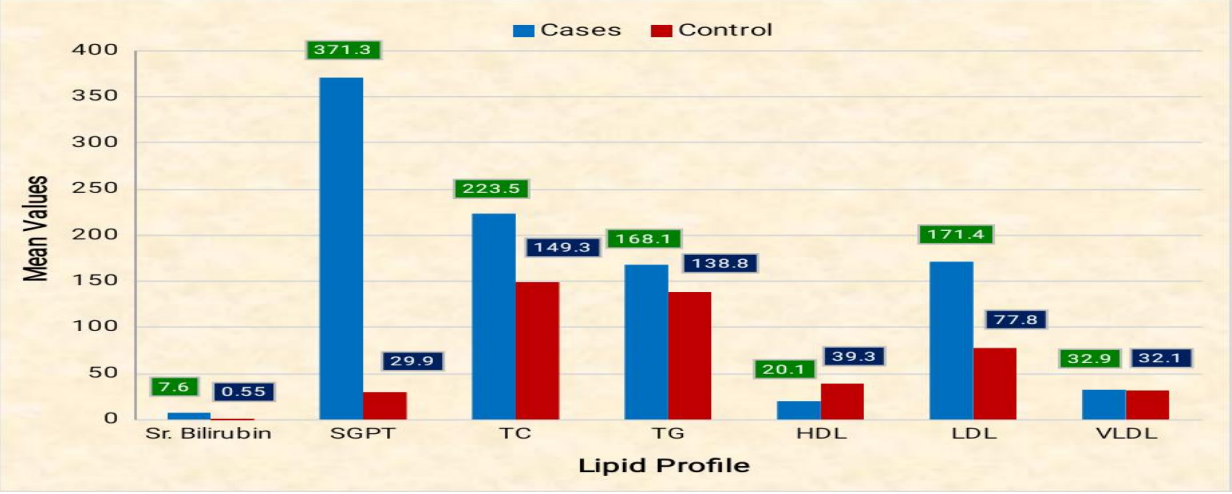

Fig. 1: Group comparison for lipid profile

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The levels of SGPT in patients who expired were in the range of (529.4 ± 336.2) as compared to those patients who recovered completely (323.6 ± 192.8) (P<0.05) (Table 4). This difference in the range of SGPT between the fatal outcome group and a recovered group of subjects was also found to be statistically significant (P<0.05). Similarly, the levels of HDLc in patients who expired were in the range of (13.0 ± 4.9) and differed significant when compared to those patients who recovered completely (21.41 ± 7.89). Perusal of the data presented in Table 5 reveled that as the patient improves, the high density lipoprotein increased significantly from 19.3 ± 8.0 on first day to 28.9 ± 5.7 on follow up, while the patients who expired showed a decrease in high density lipoprotein from 13.0 ± 4.9 on first day to 8.6 ± 2.6 in the follow up.

Table 4: Prognostic importance of HDLc and their correlation with conventional LFT

Biochemical Parameter	Cases Fully Recovered (n=12)	Cases Expired (n=6)	P- Value
Sr. Biluribin (Mg/d)	6.5 ± 35	10.8 ± 5.5	P<0.05
SGPT U/L	323.6 ± 192.8	529.4 ± 336.2	P<0.05
HDLc mg%	20.3 ± 7.3	13.0 ± 4.9	P<0.05

Table 5: Follow up study of improved and expired patients

Outcome	No. of cases	Sr. Bilirubin		SGPT		HDL c	
		Day 1	Follow Up	Day 1	Follow Up	Day 1	Follow Up
Improved	12	6.4 ± 40	3.5 ± 2.6↓	270.3 ± 186.5	132.4 ± 100.9↓	19.3 ± 8	28.9 ± 5.7
Expired	6	10.8 ± 5.5	13.7 ± 6.3↑	529.3 ± 336.2	725.8 ± 898.5↑	13.0 ±4.9	8.6 ± 2.6

DISCUSSION

Liver forms the central organ in the metabolism of plasma apolipoproteins, endogenous lipids and lipoproteins. 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 [9]. 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. Besides the synthesis of lipoproteins. liver also 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 [10, 11]. These processes could be interfered or impaired when hepatic cellular damage, which leads to an alteration of plasma lipid and lipoprotein patterns. And syntheses of cholesterol, triglycerides,

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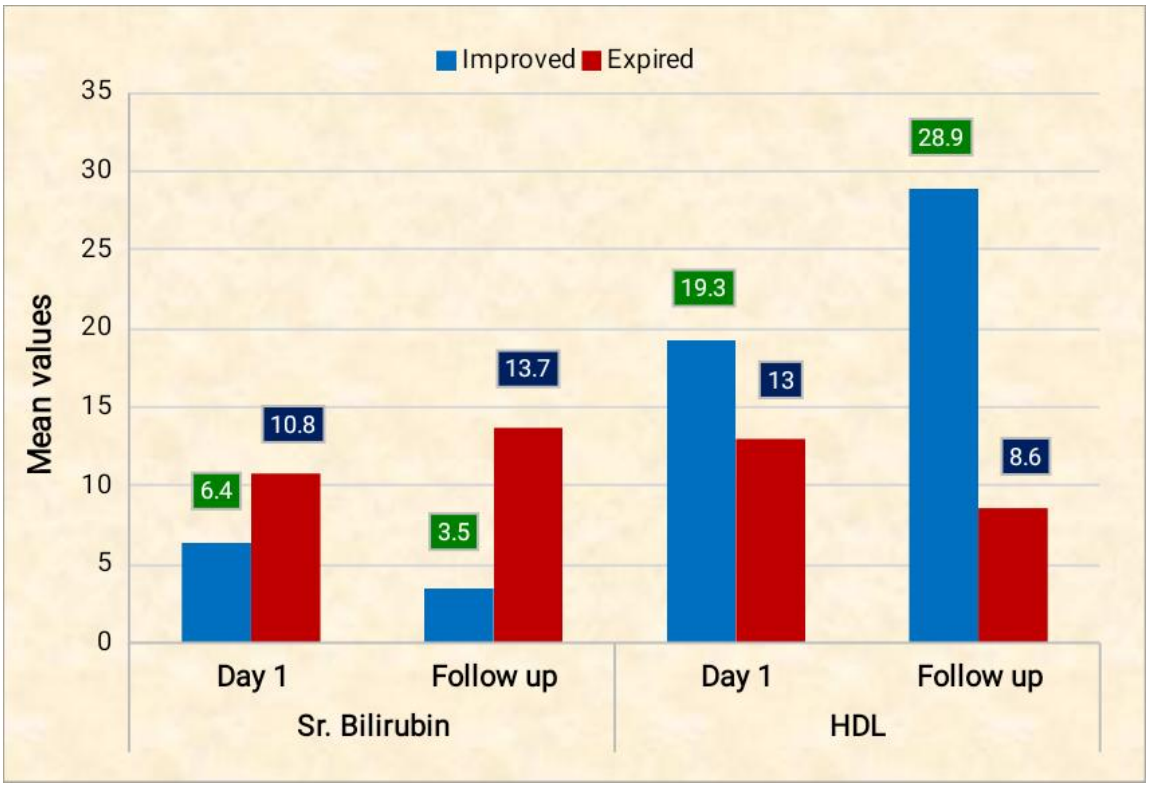

Fig. 2: Group comparison for serum bilirubin and HDL

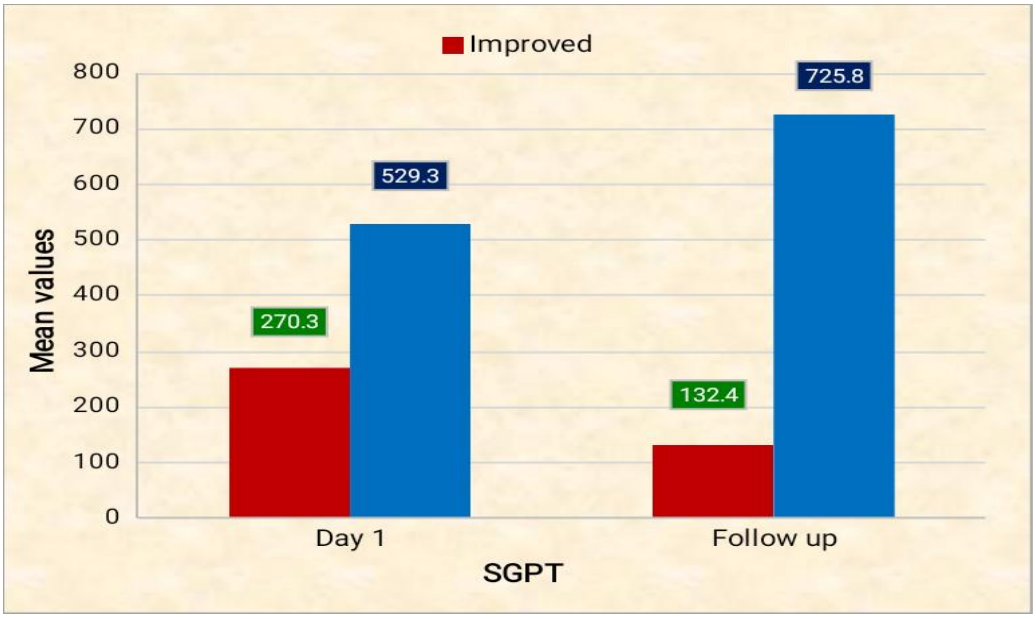

Fig. 3: Group comparison for serum bilirubin and HDL

apoAI, apoB and Lp(a) could be changed and their plasma concentrations will be altered correspondingly [12]. Therefore, analysis of plasma lipids and lipoprotein levels becomes helpful to ascertain the extent of the hepatic damage which occurs in patients with acute viralhepatitis.

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In the developed countries the incidence is attributable to sexual transmission rather than the other modes of transmission observed in developing countries. There are various liver function tests that are used in the assessment of liver function in patients with viral hepatitis. The routine liver function tests, i.e. serum bilirubin; SGPT used in the assessment of liver function may give abnormal results in various kinds of liver disorders. Furthermore these tests reflect the extent of hepatic cell damage, rather than hepatic function assessment which is more important to evaluate the patients condition and prognosis.

Data regarding lipid levels in viral hepatitis was available in 1862 when Austin Flint had suggested that the blood cholesterol level was affected by the liver. He found that blood cholesterol was raised in patients with parenchymal liver disease. It was in 1978 that Neil McIntyre studied the levels of plasma lipoproteins patterns in liver diseases [13]. Triglycerides and total cholesterol were the important lipid factors and HDL, VLDL and LDL were the important lipoproteins estimated in this study of 40 patients of viral hepatitis.

In the present study the serum total cholesterol was significantly elevated in patients with infective hepatitis when compared to controls (P<0.001). This observation supports the earlier reports. However, in contrary the studies done by Neil McIntyre and

N.M. Papadopoulous 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 decreased lecithin cholesterol acyl transferase (LCAT) activity in viral hepatitis and also part to intrahepatic biliary obstruction. Another study conducted by N.Pal, RC Misra et al have stated that serum cholesterol was low in cases of infective hepatitis [15]. This fall in serum cholesterol in severe parenchymal liver damage may be due to decrease in LCAT activity. Goel et al have found that the values of serum cholesterol remains unaltered in viral hepatitis irrespective of whether the patient is in coma or not. This finding is in contradiction with observations of some authors[16].

The serum triglyceride levels were significantly higher in cases of viral hepatitis as compared to control (P<.001). This observation is in full agreement with the studies conducted by Bhattacharya et al. [17], Pathak et al. [18], McIntyre [19] and Goel et al., [20]. The mechanism responsible for elevation of triglyceride level in patient with viral hepatitis might be due to the fact that in acute viral hepatitis free fatty acids are mobilized from adipose tissue depots and they are re-esterified again to triglyceride in liver to be transported back to peripheries, as explained by Goel et al., [20]. It is also suggested that high triglycerides is due to decreased hepatic lipase activity. These patients also appear to have decreased level of lipoprotein lipase of non-hepatic origin and this could explain the increase in triglyceride equally well. Similarly, in the present study, the level of serum HDL was significantly decreased in cases as compared to control. In addition it was also observed that the HDLc values were significantly much more lower in patients who expired as compared to those who recovered completely thus underlying the prognostic utility of HDLc. The decrease in HDLc in patients with viral hepatitis can be attributed to decreased hepatic synthesis of HDLc. This could be due to LCAT deficiency [13, 14, 15]**.** Liver is the only source of this enzyme (LCAT) and serum levels of this enzyme are decreased in liver disorders. The mechanism of LCAT deficiency in acute viral hepatitis is obscure. It could be related to bile salts interaction with apoprotein activator or to subtle derangement in lipoprotein metabolism.

It seems probable that LCAT plays a necessary role in the normal conversion of "Nascent HDL" to the mature HDL. Therefore deficient LCAT results in impairment of conversion of nascent HDL to mature HDL resulting in an increase in immature HDLc in blood which is more prone for degradation, resulting in decreased levels of HDLc as suggested byVergani G.Trovati. Decreased production of apolipoprotein I and II by liver may also be responsible for decreased HDLc production. Goel et al. [20] also studied 35 patients with viral hepatitis and observed decreased HDL cholesterol. He also observed a significant decrease in HDLc levels in patients of viral hepatitis with coma who expired, when compared to patient of viral hepatitis without coma who recovered. Irshad [16] studied 50 patients of acute viral hepatitis and observed that HDLc level was significantly decreased irrespective of viral etiology.

There was a significant increase in levels of serum LDLc in patients with infective hepatitis, when compared to controls (P<0.001) in our study. This is

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in accordance with previous study by Goel et al. [20]. But studies by McIntyre [19] observed that the LDLc concentration was not increased in patients with viral hepatitis. As these patient had a very low VLDL which is thought to be the precursor of LDL, it seems likely that their LDL catabolism was greatly reduced resulting in normal level of LDL [19]. The levels of VLDLc in patients with viral hepatitis decreased marginally when compared to controls in our study. Studies conducted by Goel et al. [20] observed that there was significant decrease in VLDL cholesterol in patients with viral hepatitis when compared to controls [16]. No significant difference was observed in VLDLc values in relation to mortality which was observed by Goel et al. [20] unlike the HDLc which was significantly decreased in those patients who expired when compared to those who recovered completely [16].

The present study of lipid profile in viral hepatitis thus clearly establishes the clinical utility of estimation of lipid levels for better assessment of hepatic function, prognostic assessment and management of viral hepatitis patients.

CONCLUSION

Eighty subjects were studied which includes 40 control and 40 cases of acute infective hepatitis. The study concluded that a failure to increase in HDLc level during follow up in viral hepatitis appear to be a bad prognostic sign. Further, estimation of serum HDLc allows better assessment of hepatic function and evaluation of prognosis of patients with acute viral hepatitis.

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