International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: 6.614
Available Online at www.journalijcar.org
Volume 7; Issue 11(A); November 2018; Page No. 16220-16223
DOI: http://dx.doi.org/10.24327/ijcar.2018.16223.2988

Research Article

ASSESSMENT OF CASES OF ACUTE VIRAL HEPATITIS IN CORRELATION WITH SERUM LIPOPROTEINS

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A R T I C L E I N F O

Article History:

Received 10th August, 2018
Received in revised form 2nd September, 2018
Accepted 26th October, 2018
Published online 28th November, 2018

Key words:

Acute viral hepatitis, serum lipid profile, lipoproteins, liver profile

A B S T R A C T

Introduction: "Hepatitis" means inflammation of the liver. The liver is a vital organ that processes nutrients, filters the blood, and fights infections. When the liver is inflamed or damaged, its function can be affected. Heavy alcohol use, toxins, some medications, and certain medical conditions can cause hepatitis. However, hepatitis is most often caused by a virus. Acute viral hepatitis (AVH) continues to be a major public health burden in developing countries like India. In the present study, we assessed correlation between clinical presentation of acute viral hepatitis and alterations in serum lipoprotein levels.

Materials and Methods: It was a case control analytical study, conducted among 30 presumptive cases of acute viral hepatitis, admitted under department of general medicine, KIMS, Karad during the period of October 2015 to March 2017.

Results: In this study, different mean parameters of lipid profile in cases and controls as well as differences were calculated and compared to each other. Mean total cholesterol was 169.700 ± 33.225 and mean HDL was 25.10 ± 3.467. Both were significantly lower ($p < 0.05$) in cases than controls.

Conclusions: Lipid parameters (particularly HDL) are deranged in acute viral hepatitis as compared to controls, more deranged during acute phases of viral hepatitis than recovering phases and in complicated cases as compared to uncomplicated cases, so we can use serum lipid profile as prognostic marker in acute viral hepatitis.

INTRODUCTION

"Hepatitis" means inflammation of the liver. The liver is a vital organ that processes nutrients, filters the blood, and fights infections. When the liver is inflamed or damaged, its function can be affected. Heavy alcohol use, toxins, some medications, and certain medical conditions can cause hepatitis. However, hepatitis is most often caused by a virus. Acute viral hepatitis (AVH) continues to be a major public health burden in developing countries like India [1]. 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]. This study focus on the alterations of serum lipids in patients suffering from acute hepatitis due to hepatotropic virus and to find out the significance of serum lipid levels in active and recovering phases of acute viral hepatitis. High-density lipoprotein cholesterol (HDL-C), which consists mostly of cholesterol, phospholipid, and protein, is produced and secreted by the liver and intestine. [121]

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LDL-C is one of the major culprits in the development of atherosclerotic heart disease. Goal LDL (to prevent atherosclerotic plaque formation) is between 50-70 mg/dL. A higher value confers increasing risk for the development of coronary artery disease and needs to be remedied. This is based on The Framingham Heart Study, which was the first study to reveal a positive association between total cholesterol and coronary artery disease (CAD). [118]

HDL-C transports cholesterol from tissues to the liver. In this reverse cholesterol transport process, it performs a "clean-up" function. This process is called reverse cholesterol transport because cholesterol synthesized in peripheral tissues is eventually returned to the liver for its disposal from the body. HDL-C levels are decreased in association with recent illness; starvation and stress; smoking; obesity and lack of exercise; medications such as thiazide diuretics, steroids, and beta-blockers; hypertriglyceridemia; and in elevated immunoglobulin levels.

Liver is a main organ for synthesis and degradation of lipoproteins.

Therefore diseases of the liver may lead to quantitative and possible qualitative changes in plasma lipid profile.Defective cholesterol esterification of hepatic disease can be attributed to impaired lecithin cholesterol acyl-transferase (LCAT)

synthesis secondary to hepatocellular disease; Several studies have documented low plasma LCAT activity in patents with liver disease and have shown that this decrease is associated with impaired plasma cholesterol esterification.

In the present study, we assessed correlation between clinical presentation of acute viral hepatitis and alterations in serum lipoprotein levels.

MATERIALS AND METHODS

It was a case control analytical study, conducted among 30 presumptive cases of acute viral hepatitis, admitted under department of general medicine, KIMS, Karad during the period of October 2015 to March 2017.

Probable cases of acute viral hepatitis with clinical symptoms suggestive of hepatitis or liver Function Test Reports consistent with Acute Viral Hepatitis, AND those cases that were sero-positive for either Hepatitis-A, B, C or E were included in the present study.Similar number of controls (n=30) were selected in the present study. All the controls were matched for non-modifiable risk factors such as age, gender.

Sample size was calculated by using open-epi software, considering the difference of 10.4mg/dl in the values of serum cholesterol in patients of acute hepatitis as compared with normal (control) individuals from a previous study.

Case definition of Acute phase of acute viral hepatitis: Symptoms (Anorexia, Nausea, Vomiting, Alteration of taste, Arthralgia, Malaise in Prodromal phase. Dark urine, Pale color stool , Prostration , Yellow eyes, Abdominal pain and Pruritus in Icteric phase) and Liver Function Test Reports consistent with Acute Viral Hepatitis.

Recovering phase of acute viral hepatitis: Absence of constitutional symptoms like anorexia, nausea, vomiting, fatigue, malaise and arthralgia.

Patients having coexistent history of diabetes mellitus, coronary heart disease, smoking, alcoholism, familial hypercholesterolemia, taking lipid lowering agents (any condition affecting serum lipid levels) or patients having hypotension or history of ingestion of hepatotoxic drugs or toxins were excluded from the cases selected in the present study. Whereas, patients having coexistent history of diabetes mellitus, coronary heart disease, smoking, alcoholism, familial hypercholesterolemia, taking lipid lowering agents (any condition affecting serum lipid levels) were excluded from the controls selected in the present study.All the patients were enrolled after written and informed consent. Detailed history was taken. Thorough general and systemic examination was carried out. All findings were recorded in the Patient's Proforma. Investigations, as mentioned in the Patient's Proforma, were carried out on admission and during recovering phase of acute viral hepatitis. Fasting serum lipid profile levels of study group were compared with controls two times, once during the acute phase and then in the recovering phase of viral hepatitis.

Data was entered in using Microsoft Excel software and analysed with the help of Open-epi software. Descriptive statistics was explained by frequency and percentage with the help of tables and graphs. Tests of significance (t-test and Anova test) were applied to draw the conclusions. P-value less than 0.05 were considered as significant.

RESULTS

The present study was conducted among 30 probable cases of acute viral hepatitis admitted under the department of general medicine, KIMS, Karad. In this study, age of the patients was ranging from 21-70 years. The maximum incidence of acute viral hepatitis was in 3rd decade (50%). Age group distribution was almost equal in both groups. Differences of age between two groups were not significant (P value > 0.05). In this study average age of the patients was 35.5 years ±13.89. Out of 30 both among cases and controls, 22 (73.3%) were male and 08 (26.6%) were female. Sex distributions in case and control groups were comparable.(Table 1) (Figure 1)

Table 1 Age Distribution of cases and controls

Age	Cases	Controls
21-30	15 (50%)	14 (46.6%)
31-40	7 (23.3%)	7 (23.3%)
41-50	3 (10%)	4 (13.3%)
51-60	2 (6.6%)	2 (6.6%)
61-70	3 (10%)	3 (10%)
Total	30 (100%)	30 (100%)

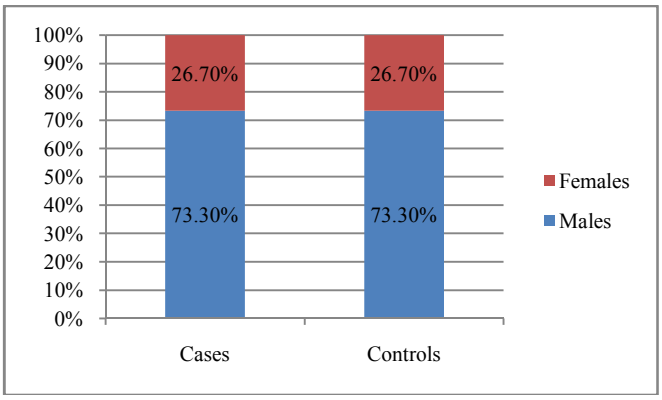

Figure 1 Sex distribution of cases and controls

In this study, HEV infection in 73.3% (22) cases was found to be the most common viral infection followed by 13.3 % (04) HAV infection, 6.66 % (02) HBV infection, and 6.66 % (02) HCV infection (Table 2). In this study, the majority of the cases presented with nausea/vomiting and dark yellow urine 86.6% (26) cases, followed by anorexia and icterus were in 83.3% (25) cases, abdomen pain was in 60% (18) cases, fever was in 53.3% (16) cases, hepatomegaly was in 36.6% (11) cases and pruritus was in 23.3.% cases. There was no splenomegaly in any cases.(Figure 2)

Table 2 Etiological agent of acute viral hepatitis: Sero-type of hepatitis

Type of Hepatitis	No. of cases	Percentage
HAV	04	13.3 %
HBV	02	6.7 %
HCV	02	6.7 %
HEV	22	73.3 %
TOTAL	30	100 %

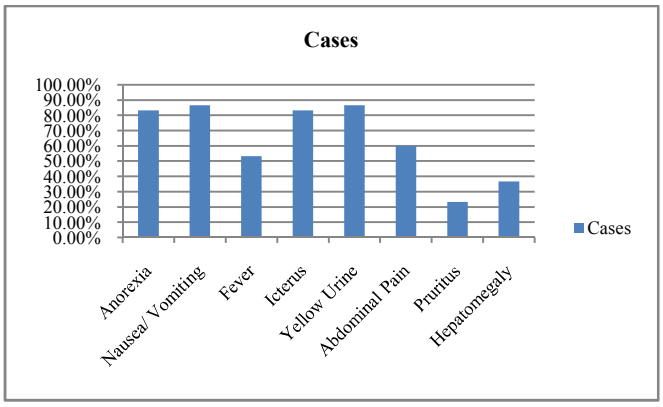

Figure 2 Clinical manifestations of patients with acute viral hepatitis

In this study, different mean parameters of lipid profile in cases and controls as well as differences were calculated and compared to each other. mean total cholesterol was 169.700 ± 33.225 and mean HDL was 25.10 ± 3.467. Both were significantly lower ($p < 0.05$) in cases than controls.The mean values of HDL in cases was 12.933± 8.304 mg/dl, while in LDL it was + 21.034± 36.666 mg/dl, which were statistically significant.In this study among the cases, Mean HDL was significantly lower ($p < 0.05$) in acute phase than recovering phase. Mean LDL was higher in acute phase than recovering phase but was not statically significant.The mean difference in HDL it was 2.866 mg/dl, which was statistically significant, whereas the difference in mean LDL was 7.367 ± 31.268, which was not significant (Table 3).

Table 3 Comparison of various clinical parameters with lipid profile mean values

Lipid Parameter	Present study		Bhattacharya *et al* [128]		Abbas al-Tamimi *et al* [127]	
	Cases	Controls	Cases	controls	Cases	Controls
Mean HDL (mg/dl)	25.100±3.467	38.033 ± 7.911	12.21 ± 5.59	47.18 ± 8.37	48.91±14.88	44.22±8.71
Mean LDL (mg/dl)	135.900 ± 28.779	114.866 ± 18.543	148.53 ± 25.93	77.04 ± 20.92	49.77±25.08	125.91±66.19

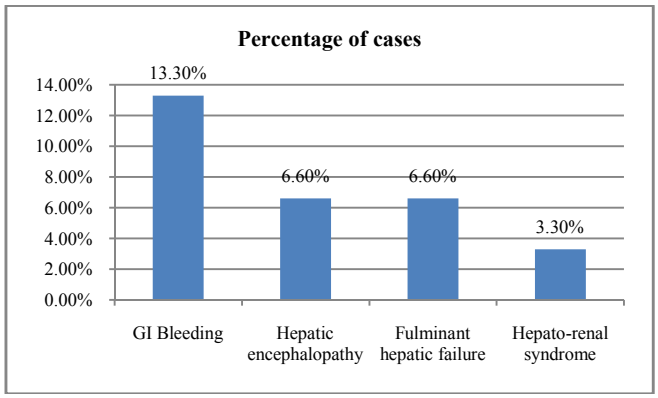

Figure 3 Distribution of cases according to complications

In the present study, the mean HDL was lower among the complicated cases than uncomplicated cases of acute viral hepatitis but only difference of HDL between complicated and uncomplicated cases was statically significant, while difference of Total cholesterol was not significant (Table 3). Serum LDL were increased in complicated cases as compared to uncomplicated cases, but differences of both were not significant. However, there was no significantly difference in any lipid profile parameters in relation to etiological agent of viral hepatitis.There was significantly lower mean HDL value in patients who required longer duration of hospital stay. There were no significant differences in other lipid parameters in relation to requirement of hospital stay (Table 3).

DISCUSSION

The present study was conducted among probable cases of acute viral hepatitis, with an objectives to study clinical presentations of various acute viral hepatitis (viz. Hepatitis A, B, C, D and E), and hence to correlate the diagnosis with liver profile levels. In this study we enrolled total 30 cases and 30 controls, who were matched for age, gender and risk factors. Mean age for these cases was 35.50 ± 13.89 years and in controls it was 35.86 ± 13.66 years. The mean ages are comparable between the cases and control groups. In a study by Abbas al-Tamimi *et al* [11], total number of cases were 63 and the mean age in cases was 30.3 years and in controls it was 36.16 years which is comparable to our study. In the Bhattacharya *et al* [12] study, total 100 subjects were studied (50 cases, 50 controls). The mean age in cases was 25.68 years and in controls it was 24.2 years (Table 5).In our study, out of 30 cases, 73% cases were male, while 27% were female, while in Bhattacharya [12] study 48% cases were male and 52% cases were female.

In the present study, the more common clinical manifestations were nausea/vomiting and dark yellow urine which were present in 86.6% (26) cases, followed by anorexia and icterus were in 83.3% (25) cases, abdomen pain was in 60% (18) cases, fever was in 53.3% (16) cases, hepatomegaly was in 36.6% (11) cases and pruritus was in 23.3.% cases. There was no splenomegaly in any cases. Most common symptoms in Bhattacharya *et al* [12] were jaundice and yellow colored urine –were present in all (100%) cases followed by anorexia in 90% cases, hepatomegaly in 72% cases, nausea/vomiting and fever in 70% cases, abdomen pain in 30% cases and pruritus in 20% cases (Table 4).

Mean HDL level in our study among cases during acute phase of viral hepatitis, was 25.100 ± 3.467 mg/dl, whereas it was 12.21± 5.59 mg/dl in the Bhattacharya [128] study and 48.91 ± 14.88 mg/dl in the Abbas al-Tamimi[127] study. Among the controls, mean HDL was 38.033 ± 7.911 mg/dl in our study whereas it was 47.180 ± 8.37 mg/dl in the Bhattacharya study[127] and 44.22 ± 8.71 mg/dl in the Abbas al-Tamimi [127] study(Table 4).Thus in our study mean HDL was lower in acute phase of viral hepatitis than control groups, which was same as Bhattacharya. [127] While in Abbas al-Tamimi [126] study mean HDL was higher in acute phase of viral hepatitis than control group.

Mean LDL level in our study among cases during acute phase of viral hepatitis, was 135.90 ± 28.779 mg/dl, whereas it was 148.53 ± 25.93 mg/dl in the Bhattacharya [127] study and 49.77 ± 5.08 mg/dl in the Abbas al-Tamimi[126] study. Among the controls mean LDL was 114.866 ± 18.543 mg/dl in our study whereas it was 77.04 ± 20.92 mg/dl in the Bhattacharya study[127] and 125.91 ± 66.19 mg/dl in the Abbas al-Tamimi[127] study. In our study, mean total LDL was higher in acute phase of viral hepatitis than control groups, which was same as Bhattacharya.[128] While in Abbas al-Tamimi[127] study mean LDL was lower in acute phase of viral hepatitis than control group. Mean serum HDL level in complicated cases was 21.25 ± 3.403 mg/dl which was lower than uncomplicated cases, same as the Bhattacharya *et al* [128] study. Mean serum LDL level in complicated cases was 140.50 ± 19.416 mg/dl

which was higher than uncomplicated cases, same as the Bhattacharya *et al* [128] study.

In our study, cases and controls were comparable according to age, sex and risk factor which could be confounding factors and distribution was equal according to these parameters in both the groups. The mean total cholesterol and mean HDL in cases were found to be lower than controls, while mean triglyceride and mean HDL found to be higher in cases than controls group. There was no correlation in any lipid profile parameters during acute phase in relation to etiological agent of viral hepatitis.This study was done upon a small number of subjects, so large amount of subjects required to establish strong relationship between these variables.

CONCLUSIONS

There is a significant decrease in mean HDL in acute phase of viral hepatitis than controls. While mean LDL was statistically significantly higher in acute phase of viral hepatitis than controls.Mean HDL levels were statistically significantly lower during acute phase than recovering phase of viral hepatitis. Hence, Mean HDL was statistically significantly lower among the complicated cases than uncomplicated cases of acute viral hepatitis.

Lipid parameters (particularly HDL) are deranged in acute viral hepatitis as compared to controls, more deranged during acute phases of viral hepatitis than recovering phases and in complicated cases as compared to uncomplicated cases, so we can use serum lipid profile as prognostic marker in acute viral hepatitis.

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How to cite this article:

Patel Pavan D and NairShruti S (2018) 'Assessment of Cases of Acute Viral Hepatitis In Correlation With Serum Lipoproteins', *International Journal of Current Advanced Research,* 07(11), pp. 16220-16223.
DOI: http://dx.doi.org/10.24327/ijcar.2018.16223.2988

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