SERUM LIPID PROFILE IN HEPATITIS C PATIENTS

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ABSTRACT*: Metabolic profiles are correlated with infection of the hepatitis C virus (HCV). The Hepatitis C virus changes the normal level of lipids in Hepatitis C patients. This study was performed to find the levels of HDL, LDL, VLDL, Total Lipids, Cholesterol, and Triglyceride in Hepatitis C patients. Lower levels of HDL, LDL, VLDL, Total Lipids, Cholesterol and Triglyceride in HCV patients cause severe complications. Blood samples of sixty hepatitis C patients were taken from Jinnah Hospital Lahore and blood samples of normal individuals were taken from the general public of Lahore. The enzymatic Photometric Assay Technique was used to determine the levels of plasma HDL, LDL, VLDL, Total Lipids, Cholesterol, and Triglyceride in the blood. It was found that levels of HDL, LDL, VLDL, Total Lipids, Cholesterol and Triglyceride were significantly lower as compared to control subjects. Results were compared with the previous reports of different countries and discussed. These results suggested that hypolipidemia may consider one of the clinical features in Hepatitis C patients probably related to hematopathology.*

Key Words*:* Serum, Lipid, Hepatitis *C*

INTRODUCTION

Hepatitis C severely affects the world population and according to the World Health Organization, 130-170 million people are infected with the hepatitis C virus (HCV), which is 2-2.5% of the world's total population [1]. Hepatitis C virus (HCV) causes changes in the lipid profile of the host ([2] and is a major cause of chronic liver disease [3].

Many studies have been conducted to observe the lipid profile of Hepatitis C patients. These previous studies demonstrated the lipid-lowering effect [4], lower serum levels of LDL [5, 6], cholesterol [7], Total lipids [8] and TG levels [9]. Another study further demonstrated that hepatitis C infection causes dyslipidemia [10] and hyperlipidemia [6].

We here conducted a study to demonstrate the relationship between HCV infection and alteration in HDL, LDL, VLDL, Total Lipids, Cholesterol, and Triglycerides. We hypothesized that chronic HCV infection decreases the levels of HDL, LDL, VLDL, Total Lipids, Cholesterol, and Triglycerides in Hepatitis C patients' blood.

MATERIALS AND METHODS

The clinical facility was available at Jinnah hospital Lahore. Hepatitis C patients with changed lipid profiles were admitted to different medical wards of Jinnah hospital. Hepatitis C patients were already diagnosed by hepatologists based on ELISA, PCR, SCREENING & BIOPSY. The clinical history and other details of patients were obtained from the different medical units of the Jinnah hospital. Blood samples of Hepatitis C patients were collected in a fasting state from Medical unit II and the special ward of endoscopy of Jinnah hospital. Blood samples of control subjects were also collected in a fasting state from the different areas of the city.

The study included 20 control subjects and 60 patients with Hepatitis C belonging to different age groups. Blood samples were collected in the fasting state of each of the control subjects and Hepatitis C patients. The lipid profile of Hepatitis C patients was compared with control subjects to find out the relationship between lipid profile and Hepatitis C infection. Blood samples were collected for 20-25 minutes allow clotting at room temperature and centrifuged for 2 minutes for the separation of serum. The speed of the centrifuge machine was 40 rotations per minute (rpm). The serum was floated on top of the blood clot. A dropper was placed in the tube and the serum was sucked and added to the labeled tubes. A commercially available kit was used, employing the Direct Enzymatic colorimetric liquid method, to determine the lipid profile. Liquid method. Experimental data were analyzed by student t-test.

RESULTS

The study included twenty control subjects and sixty hepatitis C patients. The average age was 41.45 ± 2.82 and 50.36 ± 1.23 years for control subjects and Hepatitis C patients respectively (Table 1).

The mean levels of serum HDL were observed in control subjects and Hepatitis C patients. Fasting serum HDL level in hepatitis C patients was less as compared to control subjects. These values were 39.5 ± 1.1 mg/dl in control subjects and 29.33 ± 1.33 mg/dl in Hepatitis C patients. The difference was significantly lower ($p < 0.001$) in Hepatitis C patients (Table 1).

The mean levels of serum LDL were studied in control subjects and Hepatitis C patients. Fasting serum LDL level in Hepatitis C Patients was less as compared to control subjects. These values were 140.2 ± 6.2 mg/dl in control subjects and 95.03 ± 4.98 mg/dl in Hepatitis C patients. The observed difference was significantly lower ($P < 0.001$) in Hepatitis C patients (Table 1).

The mean levels of serum VLDL in Hepatitis C patients were decreased as compared to control subjects. These values were 41.06 ± 4.31 mg/dl in the control group and 20.98 ± 1.39 mg/dl in Hepatitis C patients. The observed difference was significantly lower ($P < 0.001$) in Hepatitis C patients (Table 1).

The mean level of serum total Lipids in Hepatitis C patients was low as compared to control subjects. In control subjects, these values were 606.5 ± 31.27mg/dl in the control group and 363.1 ± 16.55 in Hepatitis C patients. The observed difference was significantly lower ($P < 0.001$) in Hepatitis C patients (Table 1).

The mean levels of serum cholesterol were observed in control subjects and Hepatitis C patients. Level of serum cholesterol in Hepatitis C patients was lower as compared to control subjects. These values were 200.55 ± 8.77 mg/dl in the control group and 117.3 ± 6.00 mg/dl in Hepatitis C patients. The observed difference was significantly lower ($P < 0.001$) in Hepatitis C patients (Table 1).

The mean serum triglyceride level of control subjects and Hepatitis C patients was also studied. Fasting serum triglyceride level in Hepatitis C patients was lower. These

values were 205.3 ± 21.58 mg/dl in the control group and 104.93 ± 6.97 mg/dl in Hepatitis C patients. The observed difference was significantly lower (P < 0.001) in Hepatitis C patients (Table 1).

Table 1. Lipid profile of Hepatitis C patients and control subjects.

Lipids	Hepatitis c	Control	P value
HDL	29.33 ± 1.30	39.50 ±1.10	< 0.001
LDL	95.03 ± 4.98	140.20 ± 6.20	< 0.001
VLDL	20.98 ± 1.39	41.06 ± 4.32	< 0.001
Total Lipids	363.10 ± 16.55	606.50 ± 31.27	< 0.001
Cholesterol	117.30 ± 6.00	260.55 ± 8.77	< 0.001
Triglyceride	104.93 ± 6.97	205.30 ± 21.58	< 0.001

DISCUSSION

In this study lipid profiles (HDL, LDL, VLDL, total lipids, cholesterol, and triglycerides) of twenty control subjects and sixty, Hepatitis C patients were observed for comparison. Significantly lower levels (p < 0.001) of serum HDL were found in Hepatitis C patients as compared to control subjects. These results are in line with some previous studies. Many researchers in their studies described lower levels of HDL in Hepatitis C patients in comparison with control subjects in the Pakistani population [11], Egyptian population [12], New York population [13], and Turkish population [14].

Although changed serum lipids are commonly found in a patient with acute hepatitis C infection. It seems that there is a specific association between lipid metabolism and HCV infection [15]. Many researchers described that HCV replication is the major cause of a decrease in intrahepatic cholesterol synthesis leading to decrease serum HDL levels [16]. In post-alcoholic liver diseases, activities of HDL-lipoproteins, hepatic lipase, and lecithin were studied in different studies which showed the lower HDL level in acute hepatitis patients [17]. Some studies in recombinant cells indicated that SR-B1 can promote cholesterol efflux [18]. Deficient in SR-B1 indicates it physiologically functions selectively sequestering cholesterol esters from HDL [19]. This results in decreased HDL levels in the plasma by specific receptors [20].

Significantly lower levels (p < 0.001) of serum LDL were demonstrated in Hepatitis C patients in comparison with control subjects. These results are in line with some previous studies. Many researchers in their studies described lower levels of LDL in Hepatitis C patients in comparison with control subjects in the Egyptian population [21], Pakistani population [11], New York population [6], and in Turkish population [14].

Some researchers suggested that HCV infection is one of the causes of hypolipidemia in Hepatitis C patients. Virus interference with the mevalonate pathway cause decreased cholesterol production and compensatory upregulation of LDL receptors which result in decreased LDL levels. The Hepatitis C virus also reduces the levels of an enzyme, microsomal triglyceride transferase protein (MTTP). MTTP is involved in the production of VLDL and its inhibition decreases the levels of LDL [22]. It has been found in some studies that the liver is involved in the synthesis and secretion of lipoprotein and also synthesizes enzymes for LDL metabolism, i.e., lecithin cholesterol acyltransferase [23, 24]. Hepatic cellular damage disturbs the normal process of the liver which causes a change in serum LDL level and lipoprotein patterns [25}. It has been demonstrated that chronic hepatitis alters the plasma levels of cytokines, lipid peroxides, and anti-oxidant and disturbs the lipid metabolism which alters the level of LDL in Hepatitis C patients in vivo [26-28].

Our results also showed that serum VLDL level was significantly lower (p < 0.001) in Hepatitis C patients when compared with control subjects. These results are in line with some previous studies. Many researchers in their studies described lower levels of VLDL in Hepatitis C patients as compared to control subjects in the Turkish population [14] and in Japan [29]. The lower level of serum VLDL can be explained as HCV decreasing the activity of microsomal triglyceride transfer protein which decreases hepatic VLDL production [30, 31].

Significantly lower (p < 0.001) levels of serum Total Lipids were observed in Hepatitis C patients. These results are similar to previous studies which showed a lower level of Total Lipids in Hepatitis C patients when compared with control subjects in the population of Sindh Pakistan [11].

Previous reports suggested that Hypolipidemia is more common in patients having genotype 3a [32-34]. It was reported that in Pakistan the common type of HCV is genotype 3a. Therefore, a lipid profile was suggested for chronic liver disease patients having genotype 3a [35].

Our results showed that serum cholesterol in Hepatitis C patients was significantly lower (p < 0.001) if compared with control subjects. These results are in line with some previous studies. Many researchers in their studies described lower levels of cholesterol in Hepatitis C patients as compared to control subjects in the Japanese population [29], Egyptian population [21], and Turkish population [14].

The lower level of serum cholesterol in hepatitis C patients can be explained as HCV replication decreases cholesterol synthesis by two possible pathways; first, it may stop geranyl pyrophosphate production, required for cholesterol synthesis [21]. Second, it makes cholesterol available for the synthesis of intracellular membranes. These two factors ultimately decrease cholesterol levels [36]. Hepatitis severely disturbs liver function [37]. Many researchers demonstrated low serum levels of total cholesterol in liver damage patients [38-40].

The result of our tests revealed significantly lower (p < 0.001) levels of serum triglyceride in Hepatitis C patients when compared with controls. These results are in line with some previous works. Many researchers in their studies described lower levels of triglyceride in Hepatitis C patients as compared to control subjects in the Egyptian population [12], the New York population [13], and the Turkish population [14].

The life cycle of the virus has some effect on the maturation and excretion of VLDL which lower the level of TG [21]. It has also been observed that serum TG levels decreased in advanced chronic liver disease [41]. Changes in TG metabolism in the early stages of the liver disease remain unclear. Lipoproteins transport TGs in plasma and the measurement of these lipoproteins will provide the understanding of change in TG metabolism in liver injury [42]. Previous studies also demonstrated that HCV-associated

steatosis may be a result of less triglyceride export by Apo B-containing lipoprotein production [43]. Some previous studies showed that HCV protein has an effect on the secretion of Apo B containing VLDL [44]. These effects deposit TG in hepatocytes and decreased serum triglyceride levels, which may explain the relationship between HCV infection and lower TG levels. The low serum level of TG in HCV patients is due to HCV infection and liver cirrhosis [45].

Present studies suggested that lipid profile may be helpful in the diagnosis of Hepatitis C infection and the severity of the disease. Therefore, it must be analyzed in all hepatitis C patients. However, further studies are required to find out the other parameters as well for its diagnosis and severity of the infection.

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