A study for the changes of lipid profile in sera of patients infected with viral hepatitis type B and C infections

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Abstract

The main target of Viral hepatitis B and C is the liver, the most important organ of lipid and lipoprotein metabolism. Liver inflammation leads to disrupt lipid profile. In this study we investigated the levels of cholesterol, triglycerides, high – density lipoprotein and low – density lipoprotein in sera of 33 HBV patients and 30 HCV patients against 31healthy persons as control group. Correlations between lipids parameters and hepatitis indices (bilirubin total and direct, albumin, alanine transaminase, aspartate transaminase, and alkaline phosphatase) were statistically examined. Cholesterol and low-density lipoprotein found to significantly decreased in both groups of patients while triglycerides decreased in HCV patients. Spearman's coefficient test revealed significant correlation of triglycerides decrease with bilirubin (total and direct) and albumin increase, high-density lipoprotein decrease with direct bilirubin increase in HBV patients. In HCV patients, cholesterol found to be significantly decrease with alanine transaminase and direct bilirubin increase, triglycerides with direct bilirubin, low-density lipoprotein decrease with aspartate transaminase increase. These results clearly reveal that hepatitis B and C virus patients develop hypolipidemia and its severity positively correlate with hepatitis progression. We suggest that hepatitis B and C patients, especially those in chronic stages, should be examined for the concentrations of serum lipids and maintain their levels at the desired ones to avoid the risk of many disorders associated with these disturbances and, most importantly, to relieve the patients at advanced stages of hepatitis B and C virus infections.

Introduction

Hepatitis B and C are global health problems. It is estimated that 40% of the world's population has had contact with or are carriers of the hepatitis B virus (HBV). This corresponds to an estimated 350 million HBV carriers (1). There is a wide range of HBV prevalence rates in different parts of the world. HBV prevalence varies from 0.1% up to 20%.; intermediate prevalence (3-5%) are the Mediterranean countries, Japan, Central Asia, the Middle East, and Latin and South America (2). However, exact data are difficult to generate as many cases will remain undetected due to the asymptomatic nature of many acute and chronic infections (3). On the other hand, according to the World Health Organization there are 170 million people infected with the hepatitis C virus (HCV), corresponding to 3% of the world's total population. There are considerable regional differences. It is difficult to determine the number of new HCV infections, as most acute cases will not be noticed clinically. Fewer than 25% of acute cases of hepatitis C are clinically apparent. In addition, the age of infection upon diagnosis is not possible to determine in most cases (2).These two types of viruses sharing many properties like certain routes of transmission, many signs and symptoms, extent of severity, long period of incubation, mostly leading to chronicity that may ends with fatality because of liver cirrhosis or hepatocellular carcinoma.

The main target of these viruses is the liver, which is the most important organ for the metabolism of lipids, lipoproteins and Apo lipoproteins. Under normal circumstances, most plasma endogenous lipids and lipoproteins are synthesized in the liver and then are secreted into the blood circulation (4, 5). Plasma lipoproteins are also mainly catabolized by the liver to maintain the relative balance of lipid and lipoprotein metabolism in vivo (6). It has been well documented that chronic liver dysfunction might interfere lipid metabolism in vivo and could change plasma lipid and lipoprotein patterns (7).

Interactions between chronic hepatitis C virus infection and lipid metabolism are well noticed (8, 9). Important lipid –HCV interactions have been found: host serum lipid play a role in hepatitis C virion circulating and hepatocyte entry. A proportion of circulating hepatitis C viral particles are complexed with host triacyl glycerol-rich lipoproteins, known as lipo-viroparticles (10). These particles use LDL receptors on hepatocytes as points of entry and are associated with high rate of infectivity(11).Once hepatitis C virions have enter the hepatocytes their replication is again dependent on host lipid interactions. New hepatitis C virion formation

requires viral binding either to an endoplasmic reticulum phospholipid membrane or to an endoplasmic reticulum –associated membranous web (12). Dubuisson et al reported that HCV replication could decrease intrahepatic cholesterol synthesis through two possible pathways; first, it may shunt geranylpyrophosphate that leads to decreasing the quantity of this necessary intermediate available for cholesterol synthesis. Second, it may divert cholesterol to the synthesis of intracellular membranes that are necessary for the viral replication complex. The net effect of these diversions is the decrease of available cholesterol for physiologic intracellular processes and for peripheral delivery via VLDL, ultimately resulting in decreased serum cholesterol levels. The decrease in available intracellular cholesterol may also lead to an increase in LDL receptors and intrahepatic LDL. This increase in LDL uptake may account for the decreased serum LDL levels in HCV infection (12). In addition, the metabolic processes that is associated with viral replication may be associated with a drop in triglycerides levels (13, 14). Therefore, chronic HCV infection by interrupting cholesterol synthesis and using host lipids for replication, decreasing circulating lipids, and clearance of the virus results in rebound increase of lipid levels.

HBV entry to the hepatocytes is still unclear, no study had determined the type of receptor responsible of this process, however, a recent study identified a protein named sodium taurocholate cotransporting polypeptide (NTCP)as a receptor for HBV on hepatocytes−(15). This protein is known to transport bile acids from the blood into the liver. Other recent studies have shown that the initial attachment of HBV to susceptible cells depends upon glycosaminoglycans present on the cell surface (16, 17). Although this is a necessary but not sufficient step. Furthermore, even after virus has attached, a number of agents can significantly inhibit entry (18).

HBV is a DNA virus related to hepadnaviridaea, while HCV is a RNA virus belongs to flaviviridaea and the attachment sites of these viruses on hepatocytes membranes are among many differences between these viruses.

In this study we have investigated the effect of HBV and HCV infections on lipid profile (cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL) in viral hepatitis B and C patients' sera in comparison to sera of control group. Moreover, Study the correlations between lipid parameters and certain hepatitis indices (bilirubin total and direct, alanine transaminase (ALT), aspartate

transaminase (AST), albumin, and alkaline phosphatase) and find the association, if any, between parameters studied and viral hepatitis B and C infections.

It is well known that maintaining lipids in desired levels avoids patients the risk of many heart and coronary diseases. Therefore, determination of disorders in lipid parameters may help in proper management and treatment of HBV and HCV patients.

Materials & Methods

The current study included 63 hepatic patients: (33 HBV+30 HCV) belonging to various risk groups including (thalassemic-samples, hemodialysis-samples, liver problems' patients–samples and general population-samples) who attending to Azadi Teaching Hospital (Gastroenterology-Liver Disease Unit and Thalassemia Center), Kirkuk General Hospital (Hemodialysis Center), The General Salaheddin hospital and Tikrit Educational Hospital in Tikrit .Their age ranged between 1-75 years old. These patients were tested against hepatitis free apparently healthy control group including 31 persons of comparable age, BMI, gender, socioeconomic status and locations. The study was a cross sectional carried out in Tikrit City from 15st of October 2013 to 30th of March 2014.

Tests for Alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin (total and direct), albumin, alkaline phosphatase, cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL) were done using kits provided by Agappe Diagnostics Switzerland GmbH, Knonauerstrasse 54/6330 Cham (Switzerland).

Tests for HBsAg and Hepatitis C Virus Antibodies were done using ELISA technique (ACON Laboratories, Inc.10125 Mesa Rim Road, San Diego, CA 92121, USA). Mini-Vitek Immuno Diagnostic Assay System (Mini-VIDAS) for Anti-HCV detection was supplied from Biomerieux-Marcy-Etoile (France).

Statistical calculations were made using SPSS 21 (version 21; SPSS, Inc., Chicago, Illinois, USA) computer program. The results were got as mean ± Standard deviation. For statistical analysis, Student t test was used for the significance of differences between parameters and the relation between variables was examined with Spearman correlation. $P<0.05$ value was accepted meaningful statistically.

Results & Discussion

Liver function parameters significantly increased in both HCV and HBV patients. Therefore, these parameters were used to assess parameters of interest for comparison between both HBV and HCV patients and control group as used by other researches(19, 20).

Table (1): Mean± SD of HBV & HCV patients tested parameters comparing to control group.

parameter	HCV N=30		HBV N=33		Control N= 31
	values	Sig. 2-tailed	values	Sig. 2-tailed	values
Age (years)	25.03±17.60 yrs	.002	35.12±14.02 yrs	.673	36.16±15.84 yrs
BMI (kg/m²)	24.18±4.65 kg/m²	.000	28.95±4.13 kg/m²	.288	28.18±6.43 kg/m²
AST (u/l)	49.13±3.11 u/l	.000	67.18±36.21 u/l	.000	28.70±11.03 u/l
ALT (u/l)	45.29±4.81 u/l	.000	44.45±7.87 u/l	.000	36.48±12.15 u/l
Triglycerides (mg/dl)	254.86±120.8 mg/dl	.724	157.51±88.9 mg/dl	.000	247.04±135.3 mg/dl
Bilirubin total (mg/dl)	3.84±2.55 mg/dl	.000	3.83±2.62 mg/dl	.000	1.112±.37 mg/dl
Bilirubin direct (mg/dl)	2.46±2.013 mg/dl	.000	1.62±1.38 mg/dl	.000	.525±.269 mg/dl
ALKaline Phosphatase (u/l)	379.75±134.49 u/l	.000	220.24±73.58 u/l	.000	85.29±37.46 u/l
Albumin (g/dl)	2.276±0.199 g/dl	.000	2.84±0.71 g/dl	.000	3.87±0.62 g/dl
LDL (mg/dl)	49.77±25.08 mg/dl	.000	41.99±40.15 mg/dl	.000	125.91±66.19 mg/dl
Cholesterol (mg/dl)	148.36±42.26 mg/dl	.000	117.69±48.62 mg/dl	.000	215.34±75.85 mg/dl
HDL (mg/dl)	48.91±14.88 mg/dl	.095	44.18±8.06 mg/dl	.978	44.22±8.71 mg/dl

Notes: ALT= alanine transaminase, AST= aspartate transaminase, BMI= body mass index, HDL=high-density lipoprotein, LDL= low-density lipoprotein

As shown in table (1), lipid profile tests revealed a significant decrease in cholesterol, triglycerides and Low Density Lipoprotein-C (LDL) levels compared to controls. Correlation coefficient between lipid profile parameters and hepatitis indices of HBV patients revealed that triglycerides inversely correlate with bilirubin (total and direct) and albumin levels, high-density lipoprotein with direct albumin, at significant level for both parameters. Other lipids (cholesterol and low-density

lipoprotein) were also decreased with hepatitis progression indices in these patients but at insignificant level, as shown in table (2).

Table (2): Correlation coefficient (r) between lipid profile parameters and hepatitis indices of HBV patients.

Spearman's rho		cholesterol	triglycerides	HDL	LDL
ALT	r	-.205-	-.130-	-.148-	-.100-
	Sig. (2-tailed)	.253	.472	.410	.579
AST	r	.130	.143	.094	.028
	Sig. (2-tailed)	.472	.428	.605	.879
Bilirubin	r	-.254-	-.392-	-.203-	.003
	Sig. (2-tailed)	.153	.024	.258	.986
Bilirubin direct	r	.265	.369	.357	-.036-
	Sig. (2-tailed)	.137	.034	.041	.841
HBValbumin	r	-.024-	-.350-	-.020-	.178
	Sig. (2-tailed)	.893	.046	.910	.321
alk. Phosphatase	r	-.102-	-.038-	.210	-.212-
	Sig. (2-tailed)	.574	.835	.242	.236

Interactions between chronic hepatitis C virus (HCV) infection and lipid metabolism have been described in some studies (8, 9, 21, 22). Some studies have reported a higher prevalence of hypocholesterolemia and low LDL levels in HCV-infected patients compared to control groups (8, 9, 21). Other studies showed decrease levels of triglycerides among chronic HCV patients, (14). Although changed serum lipid is commonly found in patients with chronic liver disease of any etiology, the relationship between HCV and lipid metabolism seems to be more specific: binding of HCV particles to human high density lipoprotein (HDL), low density lipoprotein (LDL), and very low density lipoprotein (VLDL) has been described (23). Moreover, the LDL receptors could permit the entry of HCV in hepatocytes (24, 25). In addition, HCV replication could decrease intrahepatic cholesterol synthesis. The decrease in available intracellular cholesterol may also lead to an increase in LDL receptors and intrahepatic LDL. This increase in LDL uptake may account for the decreased serum LDL levels in HCV infection (25).

Body mass index of hepatitis C patients was significantly lower than that of control group as shown in table (1), which agree with Alessandro et al.(26). This finding means that HCV infection may leads to lower body content of lipids, although this decrease did not associate with

the level of liver enzymes as the correlation between these parameters was insignificant.

Lipid profile tests showed a significant decrease in cholesterol and low-density lipoprotein (LDL) (table 1). These types of lipids found to be significantly associated with certain liver function parameters (table 3). Cholesterol decrease associated with increase of ALT and direct bilirubin levels. Low-density lipoprotein decreased significantly with AST increase. Triglycerides decrease associated significantly with direct bilirubin increase although this decrease is insignificant in comparison with control group. Although Guclu and others found no significant differences between HCV patients and control group in levels of cholesterol, triglycerides, LDL, HDL (27), but Nashaat reported that It is well recognized in many studies that hepatitis C chronic infection is associated with hypolipidemia. He also found that in 150 patients with chronic hepatitis C had significant lower LDL, cholesterol, and triglycerides than normal persons with comparable age, sex and BMI (28). Moreover, Luo et al found a significant decrease in cholesterol, triglycerides and Low Density Lipoprotein-C (LDL) levels compared to controls (29*)*. Moreover, he reported that patients' plasma levels of total cholesterol, (LDL), High Density Lipoprotein –C (HDL) were lower at the active phase of the diseases than at the recovering phase, which indicating that acute liver damage could significant influence lipid metabolism in vivo. While with Carlo et al it was only the triglycerides decreased (30).

In contrast to these results, Radulescu MA. reported that the lipid profile show higher levels of cholesterol but with decreased (LDL) levels, also he found that HCV patients who respond to (interferon –α) therapy showing high levels of cholesterol (31). However, other risk factors might influence the results, such as food habits/lipid intake, abdominal obesity, exercise, antiviral treatment and treatment response history.

Table (3): Correlation coefficient (r) between lipid profile parameters and hepatitis indices of HCV patients.

Spearman's rho		cholesterol	triglycerides	HDL	LDL
ALT	r	.403	.200	.176	.279
	Sig. (2-tailed)	.027	.289	.352	.136
AST	r	.330	-.154-	-.089-	.372
	Sig. (2-tailed)	.075	.417	.639	.043
Bilirubin total	r	.214	.349	.094	.151
	Sig. (2-tailed)	.256	.059	.622	.425
Bilirubin direct	r	.394	.500	.014	.218
	Sig. (2-tailed)	.031	.005	.941	.246
albumin	r	-.287-	-.330-	.139	-.229-
	Sig. (2-tailed)	.124	.075	.465	.223
alk. phosphatase	r	-.288-	-.103-	.192	-.360-
	Sig. (2-tailed)	.122	.587	.311	.051

This study clearly indicates that hepatitis B and C virus patients, those in chronic stages as confirmed by hepatic inflammation indices, may be develop hypolipidemia or at least from disturbance of certain types of lipids that may worsen the symptoms in these patients or developing diseases associated with these disorders. Examinations for lipid profile in chronic HBV and HCV patients are required for proper management and treatment for these two types of chronic diseases.

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دراسة لتغيرات صور الدهون في امصال المرضى المصابين بالتهاب الكبد الفايروسي نمط ب وسي

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الخلاصة:

أن الهدف الرئيس لالتهاب الكبد الفايروسي هو الكبد, أهم الأعضاء المسؤولة عن أيض الدهون والبروتينات الدهنية. يؤدي ألتهاب الكبد عادة الى تخريب صور الدهون. في هذه الدراسة تم الكشف عن مستويات الكولستيرول, الدهون الثلاثية, البروتين الدهني عالي الكثافة والبروتين الدهني واطئ الكثافة في أمصال ٣٣ مصابا بالتهاب الكبد الفايروسي نمط ب و ٣٠ مصابا بالتهاب الكبد الفايروسي نمط سي مقابل ٣١ شخصا سليما كمجموعة سيطرة. تم اختبار العلاقات الإحصائية بين مؤشرات الدهون ومؤشرات التهاب الكبد (البيليروبين (الكلي والمباشر), الزلال, الالانين ترانساميناز, الاسبارتيت ترانساميناز والفوسفاتيز القاعدي) وقد وجد بان الكولستيول والبروتين الدهني واطئ الكثافة ينخفضان بمستويات معنوية في كلتا المجموعتين من المصابين بينما تنخفض الدهون الثلاثية معنويا في مرضى التهاب الكبد نمط سي. أظهر أختبار مكافئ سبيرمان وجود علاقة معنوية بين أنخفاض الدهون الثلاثية وأرتفاع البيليروبين (الكلي والمباشر) والزلال, كذلك بين أنخفاض البروتين الدهني عالي الكثافة وأرتفاع البيليروبين المباشر لدى مرضى ألتهاب الكبد نمط ب. في مرضى ألتهاب الكبد نمط سي تم الكشف عن وجود علاقة معنوية بين أنخفاض الكولستيرول وأرتفاع مستويات الالانين ترانساميناز والبيليروبين المباشر وبين أنخفاض الدهون الثلاثية وأرتفاع البيليروبين المباشر وبين أنخفاض البروتين الدهني واطئ الكثافة وأرتفاع الاسبارتيت ترانساميناز. تدل هذه النتائج بوضوح على ظهور حالة أنخفاض مستوى الدهون لدى مرضى التهاب الكبد الفايروسي نمط ب وسي وأن شدة الانخفاض ترتبط أيجابيا بتطور التهاب الكبد لذلك فأننا نقترح أجراء الكشف عن مستويات الدهون في أمصال مرضى التهاب الكبد الفايروسي نمط ب و سي, خاصة لذوي الطور المزمن, والحفاظ عليها ضمن المستويات المرغوبة تجنبا لمخاطر العديد من الامراض التي ترافق حالات أختلال الدهون, والاهم من ذلك كله, التخفيف عن مرضى المراحل المتقدمة من الإصابة بالتهاب الكبد الفايروسي نمط ب و سي.