Dyslipidemia in patients with hepatitis C virus infection

Dr. Laith Aldabbagh1*, Dr. Abdulhamza Rajooj Hmood2, Dr. Ahmed Khalid Faleeh Aldahalemi3, Dr. Sadiq Almuhana4, Dr. Ali Sadiq Almussawi5

1. University of Kufa, College of medicine
2. Kerbella College of Medicine
3. Lecturer in College of pharmacy , university of Kufa.
4. University of Kufa, College of medicine,
5. Najaf teaching hospital

*Corresponding author: laitha.aldabbagh@uokufa.edu.iq (Dr. Aldabbagh)

Abstract

Background:
Hepatitis C is a blood-borne viral illness responsible for significant morbidity and mortality all over the world. Hepatitis C virus (HCV) interacts with the lipid receptors and is secreted as very low density lipoprotein (VLDL). It enters the hepatocytes, circulating as lipoviroparticles (LVP) so HCV may leads to disturbance of serum lipid levels.

Aims of the Study:
We aim to investigate the effect of HCV on lipid profile of patients with HCV infection and degree of dyslipidemia in relation to viral load, disease duration, and viral genotype

Patients and methods:
Total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), and low density lipoprotein (LDL) were measured in (100) patients with hepatitis C infection and (100) normal persons.

Results:
The mean total cholesterol (TC) level was lower in HCV cases than non-infected persons; (174.8 ± 37.9) mg/dL vs. (189.3 ± 21.4) mg/dL, respectively, HDL-C (42.3 ± 9.5 vs 52.8 ± 11.8) respectively, VLDL (27.2 ± 6.6 vs 32.6 ± 12.8) respectively, LDL (106.3 ± 35.4 vs 110.3 ± 25.2) respectively, triglycerides (132.7 ± 34.2 vs 163.1 ± 31.6) respectively. HCV cases were more likely to have hypolipidemia (TC< 150 mg/dL) than non-infected persons; 19% and 6%, respectively, while normal levels and hyperlipidemia were more frequent among non-infected persons. No statistically significant correlation had been found between lipid profile and duration of HCV or viral load. No statistically significant differences in all lipid profile except HDL-C levels (Fig. 3.5) between both genotypes subgroups

Conclusions:
Hepatitis C virus affect lipid levels and can cause low TC, low HDL, low LDL, low TG. and lipid profile is not significantly affected by viral load or disease duration.

Keywords: dyslipidemia, HCV, cholesterol, patients

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1. **Introduction**

Hepatitis C virus is recognized as a major public health problem worldwide affecting mostly low economic countries[1]. The virus is a member of the Flaviviridae family of viruses and lone member of the genus Hepacivirus. single-stranded, positive sense, RNA genomes. Replication of HCV is rapid and 1012 virus particles can be created per day in an individual. HCV-related liver disease is mostly asymptomatic. Consequences of hepatitis C infection are:

1. Chronic hepatitis
2. Liver cirrhosis
3. Hepatocellular carcinoma (HCC)
4. Membranoproliferative glomerulonephritis (MPGN)
5. Membranous glomerulonephritis (MG)
6. Non cryoglobulenic (MPGN)

An estimated 170 million people worldwide have been infected, most of whom are chronically diseased and at risk of liver cirrhosis and hepatocellular carcinoma (HCC)[2]. Over time, 20% will develop cirrhosis and its related complications and about 1% to 2% of subjects may develop hepatocellular carcinoma (HCC), after 2 to 3 decades of infection[3]. The virus is classified into six major genotypes, many of which include a number of closely related subtypes[4] (designated a, b, c and so on). The efficacy of the standard treatment with ribavirin and pegylated interferon depends on the genotype, infection with either genotype 1 or 4 leading generally to a poorer treatment response than infection with genotype 2 or 3[5]. HCV has 4 modes of transmission between humans, these include:

1. Injecting drug use
2. Transfusion of blood and blood products
3. Sexual transmission
4. Perinatal transmission.

**Clinical presentation:**

The acute HCV infection is usually asymptomatic and accidentally diagnosed. Chronic HCV infection also may be asymptomatic but some may experience fatigue, malaise, nausea, upper right quadrant pain. Over time, 20% will develop cirrhosis and its related complications. Some will progress to hepatocellular carcinoma after 2 to 3 decades of infection[6]. The prognosis of disease, either resolution or chronic infection depend on body immune response to infected hepatocytes.

**Investigations & Diagnosis:**

Serology is the main investigation of choice. Tests for antibodies to HCV are used to know if the individual is exposed to the virus. And can be performed with nucleic acid testing (NAT) available in several different forms:

1. Qualitative polymerase chain reaction using reverse transcriptase PCR (RT-PCR).
2. Qualitative PCR using transcription-mediated amplification (TMA).

Quantitative PCR tests use RT-PCR or branched RNA chain amplification techniques to measure the concentration of the virus in the serum. These tests are useful for measuring pre-treatment viral levels, and early virological response to therapy (measured at 12 weeks), defined as a 2 log drop in viral load.

**Treatment:**

The standard treatment of chronic HCV is a combination of pegylated interferon-α (PegIFN-α) and ribavirin. This treatment is associated with many side effects as flu-like symptoms, depression, nausea, and cytopenia. Genotype 2 and 3 associated with better prognosis than genotype 1 and 4. Thus Individuals with HCV G2 and G3 infection achieve SVR
(sustained virologic response)’s of 70-80% with 24 weeks of therapy whereas those with HCV G1 and G4 require at least 48 weeks of therapy with only approximately 50% achieving SVR. Recently, new antiviral drugs that target viral replication are developed. These drugs either inhibit the HCV NS3-NS4A protease (e.g. telepravir and bocepravir) or nucleoside or non-nucleoside analogues that target the NS5B RNA dependent RNA polymerase and are currently in phase II/III clinical trials[7].

HCV life cycle:

The HCV structural proteins and the receptor molecules at the surface of target cells are involved in the early entry of viral particles inside the cytoplasm, E1 and E2 glycoprotein are necessary for viral entry and fusion [8] (Figure 1.1)[9]. E2 interact with one or more components of the receptor complex [10] cell surface receptors like cell surface Glycosaminoglycans, Human CD81, Low density lipoprotein (LDL) receptors, while the non-structural protein (p7, NS2, NS3, NS4A, NS4B, NS5A, and NS5B) are essential for the intracellular aspects of the viral life cycle[11]. Scavenger receptor B type I (SR-BI) a cell surface transmembrane protein, primarily expressed in the liver, Claudin-1, and different co-receptors molecules have been showed to mediate HCV binding to host cell[12]

![Figure 1.1](image-url)

Following initial binding of the hepatitis C virus (HCV) particle to scavenger receptor class B member 1 (SRB1) and CD81, the particle engages in further interactions with the tight junction proteins claudin 1 (CLDN1) and occludin (OCLN) and finally enters cells by receptor-mediated endocytosis (step 1). The viral RNA genome is released into the cytoplasm and translated at the rough ER (step 2). Viral proteins, in conjunction with host cell factors, induce the formation of a membranous web (MW) composed of single-, double- and multi-membraned vesicles as well as lipid droplets (LDs) (step 3). RNA replication occurs at an unspecified site within the membranous web (step 4). Assembly of HCV particles probably initiates in close proximity to the ER and lipid droplets, where core protein and viral RNA accumulate. The viral envelope is acquired by budding through the ER membrane in a process that is linked to lipoprotein synthesis (step 5). HCV particles are released via the constitutive secretory pathway (step 6)[13]. the hallmark of viral replication cycle is its dependence on host cell lipids, which tightly depend on lipid synthesis pathway.[14]

HCV circulates in the bloodstream in different forms; either free or in a complex with immunoglobulin or lipoprotein to form the so-called lipoviro particles (LVPs), either very low density lipoprotein (VLDL) or low density lipoproteins (LDL).[15] Two
lipoprotein receptors on hepatocytes are known to be of the receptors components that mediate viral entry: SR-B1 and LDL-R\textsuperscript{[16]} . Replication of HCV, like all positive-strand RNA viruses, occurs in association with cytoplasmic membranous vesicles\textsuperscript{[17]}. These membrane vesicles are enriched with proteins that are responsible for lipid metabolism and lipoprotein generation (i.e., apoB-100, Microsomal triglyceride transfer protein (MTP), apoE and Acyl-coA synthetase 3 (ACSL3)\textsuperscript{[18]} Animal studies showed an increased intrahepatic expression of genes involved in lipogenesis\textsuperscript{[19]} such as the ATP citrate lyase\textsuperscript{[20]}. This suggests possible links between viral replication and lipobiogenesis. low LDL-cholesterol level is one of the features of chronic HCV infection. Regarding cholesterol, its metabolism pathways are now known to impact on HCV replication, secretion, and entry\textsuperscript{[21]}. Viral RNA replication has been shown to be dependent on cholesterol \textsuperscript{[22]}. Cholesterol can be synthesized from acetyl-CoA via the mevalonate pathway. the mevalonate pathway is involved in HCV RNA replication. Although many studies have reported abnormal serum lipid levels in HCV infection, especially low levels of total cholesterol (TC)\textsuperscript{[23]} and low-density lipoprotein cholesterol (LDL-C)\textsuperscript{[24]} , little is known about the serum triglyceride (TG) profile in HCV infection. HCV infection by itself, or inflammation and fibrosis accompanied by chronic HCV infection might be affected by the disturbance of lipoprotein metabolism. As mentioned above, low LDL-cholesterol level is one of the features of chronic HCV infection. Serum levels of apo B (apo B-100) have been examined in connection with the persistence of HCV infection and treatment outcome of IFN-based therapy\textsuperscript{[25]} Serum apo B level is strongly correlated with LDL-cholesterol, and is partially determined by the amino acid changes of core and NS5A protein\textsuperscript{[26]}, suggesting a close interaction between HCV replication and lipoprotein production in the liver.

**Significance of hypolipidemia:**

hypolipidemia refers to reduced plasma cholesterol. Most authors use the total serum cholesterol (TC) to define this condition. most of the authors use a cut-off value between 120 mg/dl (3.1 mmol/l) and 150m/dl (3.88mmol/l)\textsuperscript{[27]} . However some authors use higher levels up to 190mg/dl (4.9mmol/l)\textsuperscript{[28]} while others use lower values such as 100mg/dl (2.59 mmol/l)\textsuperscript{[29]} .hypolipidemia is classified according to cause as primary and secondary. There are 3 rare primary disorders of hypolipidemia that result in lipid levels low enough to cause significant consequences.

**Consequences of hypolipidemia:**

1- **Effects on plasma membrane:** lipids serves as a major structural component of cell membranes It is not known how very low plasma cholesterol levels would affect membrane composition and function Acanthocytosis was reported with hypobetalipoproteinemia in advanced chronic liver disease ,coeliac disease.\textsuperscript{[30]}

2- **Intracerebral hemorrhage (ICH):** Several studies have demonstrated that low cholesterol is a risk factor for ICH\textsuperscript{[31]} studies described the association between low serum cholesterol level and cerebral hemorrhage in elderly men\textsuperscript{[32]}

3- **Adrenal failure:** The adrenal gland requires a continuous supplement of cholesterol for the biosynthesis ,hypcholesterolemia will be associated with hypocortisolemia, and during stress cortisol production may not be high enough to protect against the cell damage. Hence critically ill patients will be predisposed to adrenal failure\textsuperscript{[33]}.

4- **Sepsis:** Hypcholesterolemia in healthy men is reported to be associated with significantly fewer circulating lymphocytes, total T cells, and CD8+ cells\textsuperscript{[34]} , thus the host immunity will be altered and the patient may be prone to infection

5- **Disease mortality:** Epidemiologic studies have identified a relationship between hypcholesterolemia (< 130 mg/dL) and increased mortality from all causes \textsuperscript{[35]} . A low baseline serum cholesterol level is associated with higher mortality rates in patients with liver cirrhosis. There is a significant relationship and increased risk of mortality in patients with HIV and HCV co-infection.
2. **Aims of the Study:**

The aim of this study is to evaluate the lipemic profile in patients with HCV infection and to identify any association between serum lipid levels and viral load, viral genotype, disease duration.

**Patients and Methods**

- This is a Cohort study. Data were collected prospectively and retrospectively in Al-Sadr Medical City in Najaf between April 2015 and January 2016 from patients come to Gastroenterology (GIT) Centre. Patients files in that contain lipid profile and viral load were included in this study.

**Exclusion criteria:** HCV patients with liver cirrhosis, other types of viral hepatitis, HIV patients, patients with tuberculosis, any infection individuals taking lipid lowering drugs, diseases with malabsorption.

- The number of HCV infected patients was one hundred. Regarding the non-infected persons, we took one hundred apparently healthy individuals.

- Regarding cases, the patient was previously diagnosed as HCV infected by Enzyme-Linked Immuno-Sorbent test (ELISA) (Applied Biosystem) by detection of viral antibody in serum. The patient enrolled are either already registered in the centre with information about genotype, viral load, disease duration, or we send him to lab to do these investigations. We send the patient to lab for serum total cholesterol, serum HDL, serum TG, by dividing TG by 5 we get serum VLDL and to calculate LDL we use the following equation:

\[
LDL = TC - (HDL + VLDL)
\]

The same is done to control group regarding lipid profile.

**Statistical analysis:**

Data of the patients were entered and transformed into a computerized data base with statistical utilities, the statistical package for social sciences (SPSS) version 22, USA, was used in statistical procedures. Descriptive statistics were presented as frequencies (numbers of patients), proportions (%), mean and standard deviation, appropriate statistical tests were applied accordingly. Level of significance was set at 0.05 as significant. Finally, findings were presented in tables or figures with an explanatory paragraph for each.

4. **Results**

There were 100 HCV patients and 100 apparently healthy subjects enrolled in this study. Both groups almost matched for age and gender with no statistically significant differences in age or gender, (P>0.05).

The Comparison of mean values of lipid of the studied group is shown in table 3.2, it had been significantly found that the mean total cholesterol level was lower in HCV cases than non-infected persons; 174.8 ± 37.9) mg/dL vs. (189.3 ± 21.4) mg/dL, respectively, (P=0.002), (Fig 3.1). On the other hand HDL-C, the VLDL-C and triglycerides levels were significantly lower in HCV cases than the non-infected persons, (P<0.05), (Table 3.2 and Figs 3.2, 3.3 &3.4). Regarding the LDL-C it was lower in HCV cases than non-infected persons, however, the difference was statistically insignificant between both studied groups, (P=0.37).
As it shown in table 3.4, genotyping data were available for only 53 HCV cases, nonetheless, the genotype 4 was the dominant detected genotype among these cases, 44/53 (83%) while the genotype 1 represented only 17% of the detected genotypes. On the other hand no other genotypes were reported in this group of HCV cases.

Furthermore, the viral load of the 53 HCV cases is shown in table 3.5, the viral load was detected in 39 HCV cases only, while in the remaining 14 cases it was undetected. However, the mean viral load of the 39 cases with detected viral load was $(1626.75 \pm 1463.69 \times 10^3)$. 

According to the levels of total cholesterol and using a cutoff point of 150 mg/dL, it had been significantly found that HCV cases were more likely to have hypolipidemia (TC$<150$ mg/dL) than healthy persons; 19% and 6%, respectively, ($P=0.019$), while normal levels and hyperlipidemia were more frequent among non-infected persons, (Table 3.3).

Table 3.7 shows the correlation matrix for the Pearson’s correlation test for lipid profile and each of duration of HCV and viral load. No statistically significant correlation had been found between lipid profile and each of duration of HCV and viral load, in all correlations ($P>0.05$).

In table 3.8, the comparison of mean values of the component of lipid profile according to genotypes (1 and 4) revealed no statistically significant differences in all lipid profile except HDL-C levels(Fig. 3.5) between both genotypes subgroups. HDL-C levels were significantly higher in cases with genotype 4 than those with genotype 1; the mean was $(43.5 \pm 10.4)$ mg/dL and $(36.0 \pm 3.0)$ mg/dL, respectively, ($P=0.029$).
### Table 3.1 Age and gender distribution of the HCV patients and non-infected persons

<table>
<thead>
<tr>
<th>Variable</th>
<th>HCV (N=100)</th>
<th>Control (N=100)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Age (year)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 20</td>
<td>12</td>
<td>12.0</td>
<td>12</td>
</tr>
<tr>
<td>21 – 30</td>
<td>26</td>
<td>26.0</td>
<td>16</td>
</tr>
<tr>
<td>31 – 40</td>
<td>25</td>
<td>25.0</td>
<td>32</td>
</tr>
<tr>
<td>41 – 50</td>
<td>18</td>
<td>18.0</td>
<td>28</td>
</tr>
<tr>
<td>&gt; 50</td>
<td>19</td>
<td>19.0</td>
<td>12</td>
</tr>
<tr>
<td>mean ± SD</td>
<td>37.1 ± 15.1</td>
<td>36.9 ± 12.5</td>
<td>0.91</td>
</tr>
<tr>
<td>Range</td>
<td>19 – 72</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>64</td>
<td>64.0</td>
<td>65</td>
</tr>
<tr>
<td>Female</td>
<td>36</td>
<td>36.0</td>
<td>35</td>
</tr>
</tbody>
</table>

### Table 3.2. Comparison of mean lipid profile of HCV patients and non-infected persons

<table>
<thead>
<tr>
<th></th>
<th>HCV (N = 100)</th>
<th>Control (N=100)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>mean ± SD</td>
<td>174.8 ± 37.9</td>
<td>189.3 ± 21.4</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>79 – 290</td>
<td>142 – 239</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>mean ± SD</td>
<td>42.3 ± 9.5</td>
<td>52.8 ± 11.8</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>28 – 67</td>
<td>23 – 88</td>
</tr>
<tr>
<td>Component</td>
<td>Mean ± SD</td>
<td>Range</td>
<td>P-value</td>
</tr>
<tr>
<td>--------------------</td>
<td>---------------</td>
<td>-------------</td>
<td>---------</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>106.3 ± 35.4</td>
<td>41 – 182</td>
<td>0.37</td>
</tr>
<tr>
<td>VLDL-C (mg/dL)</td>
<td>27.2 ± 6.6</td>
<td>16 – 43</td>
<td>0.011</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>132.7 ± 34.2</td>
<td>78 – 217</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Figure 3.1. Comparison of mean Total cholesterol of HCV patients and healthy persons

Figure 3.2. Comparison of mean HDL-C levels of HCV patients and healthy persons.
Figure 3.2. Comparison of mean HDL-C of HCV patients and non-infected persons

Figure 3.3. Comparison of mean VLDL-C of HCV patients and non-infected persons
Figure 3.4. Comparison of mean Triglycerides of HCV patients and non-infected persons

Table 3.3. Distribution of Hypolipidemia among HCV patients and non-infected persons

<table>
<thead>
<tr>
<th></th>
<th>HCV (N = 100)</th>
<th>Non-infected (N=100)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Hypolipidemia (TC&lt;150mg/dL)</td>
<td>19</td>
<td>19.0</td>
</tr>
<tr>
<td>Normal</td>
<td>59</td>
<td>59.0</td>
</tr>
<tr>
<td>Hyperlipidemia (TC&gt;200 mg/dL)</td>
<td>22</td>
<td>22.0</td>
</tr>
</tbody>
</table>

P. value = 0.019

Table 3.4. Distribution of genotyping of HCV patients

<table>
<thead>
<tr>
<th>Genotype</th>
<th>NO.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9</td>
<td>17.0</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>83.0</td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>100.0</td>
</tr>
<tr>
<td>Unavailable</td>
<td>47</td>
<td>47.0</td>
</tr>
</tbody>
</table>

Table 3.5. Distribution of Viral load of HCV patients.

<table>
<thead>
<tr>
<th>Viral load x 10³</th>
</tr>
</thead>
</table>
The mean HCV disease duration was $1.5 \pm 0.9$ years (range: one month – 2.3 years).

Table 3.6. Distribution of Duration of disease of the HCV patients

<table>
<thead>
<tr>
<th>Duration of HCV (year)</th>
<th>Statistics</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td></td>
<td>1.5 year</td>
<td>0.9 year</td>
<td>one month</td>
<td>2.3 year</td>
</tr>
</tbody>
</table>

Table 3.7. Correlation of viral load and duration of HCV infection with lipid profile among HCV patients

<table>
<thead>
<tr>
<th>Lipid profile</th>
<th>Statistics</th>
<th>Duration of HCV</th>
<th>Viral load *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>Pearson Correlation (r)</td>
<td>-0.02</td>
<td>-0.07</td>
</tr>
<tr>
<td></td>
<td>P.value</td>
<td>0.86</td>
<td>0.61</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>Pearson Correlation (r)</td>
<td>0.08</td>
<td>-0.03</td>
</tr>
<tr>
<td></td>
<td>P.value</td>
<td>0.40</td>
<td>0.83</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>Pearson Correlation (r)</td>
<td>0.01</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>P.value</td>
<td>0.94</td>
<td>0.50</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>Pearson Correlation (r)</td>
<td>-0.08</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>P.value</td>
<td>0.43</td>
<td>0.86</td>
</tr>
</tbody>
</table>
Table 3.8. Comparison of mean values of lipid profile of HCV patients according to genotyping *

<table>
<thead>
<tr>
<th>Lipid profile</th>
<th>Genotype 4 (n=44)</th>
<th>Genotype 1 (n=9)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>180.1 ± 36.1</td>
<td>162.8 ± 49.1</td>
<td>0.23</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>43.5 ± 9.3</td>
<td>36.0 ± 8.0</td>
<td><strong>0.029</strong></td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>111.5 ± 35.9</td>
<td>104.2 ± 43.5</td>
<td>0.60</td>
</tr>
<tr>
<td>VLDL-C (mg/dL)</td>
<td>33.2 ± 14.9</td>
<td>29.1 ± 11.8</td>
<td>0.44</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>133.8 ± 36.5</td>
<td>133.1 ± 45.2</td>
<td>0.96</td>
</tr>
</tbody>
</table>

*Correlation made for patients with available viral load results
5. Discussion
Hepatitis C virus (HCV) infection has been associated with alterations in lipid level. Previous studies and reports have studied the changes in lipid profile after HCV infection, other studies document changes in lipid profile after treatment of HCV and compare these changes according to demographical genotyping and viral load. However, to best of our knowledge, no similar studies have been published in Iraq, particularly in Al-Sadr medical city in Najaf concerned with this subject, and previous published studies found conflicting results. Therefore, the current study tried to assess the changes in lipid profile after HCV infection. previous studies do not inform us whether the changes in lipid levels are a direct consequence of HCV infection, or such changes are present at time of HCV infection.

The current case-control study included 100 HCV patients and 100 apparently healthy persons to compare these changes and we found that the mean total cholesterol level was lower in HCV cases than non-infected individuals. On the other hand HDL-C, the VLDL-C and triglycerides levels were significantly lower in HCV cases than healthy persons, (P<0.05). These findings are consistent with that reported by Butt et al. in 2009 which found that Mean plasma levels of total cholesterol, LDL-C, and triglycerides were significantly lower in the HCV-infected subjects, compared with HCV-uninfected subjects[37]. Other studies have shown similar associations[38]. Regarding viral genotype and its effect on the lipid profile, the comparison of mean values of the component of lipid profile according to genotypes (1 and 4) revealed no statistically significant differences in all lipid profile except HDL-C levels. Bridge SH et al[39] report that HDL-C concentrations were significantly lower in HCV-G3 compared to HCV-G1 but there is no HCV-G3 cases in our samples to compare, in previous studies there is significantly higher serum LDL and total cholesterol levels in patients with hepatitis C genotype 1 but not in patients with hepatitis C genotype 3, genotype 4 and control (non infected) population[40]. The effect of disease duration on lipid profile is not significant because our sample size is small and single reading. Some studies signify the effect of disease duration on the lipid levels[41]. they found that After HCV acquisition, TC, LDL, TG and non-HDL-C progressively decline over time independent of BMI and liver fibrosis.

Regarding HCV viral load, no statistically significant correlation had been found between lipid profile and HCV viral load (P>0.05). other studies show the levels of plasma LDL was negatively correlated with HCV RNA load in all correlations[42].

Conclusions and Recommendations

5.1. Conclusions
1. Serum total cholesterol(TC), low density lipoprotein(LDL), very low density lipoprotein(VLDL), high density lipoprotein(HDL), and triglycerides(TG) were lower in patients with HCV infection than non-infected individuals. It had been significantly found that HCV cases were more likely to have hypolipidemia (TC< 150 mg/dL) than non-infected persons; 19% and 6%, respectively, and lipid profile is not significantly affected by viral load or disease duration, the comparison of mean values of the components of lipid profile according to genotypes (1 and 4) revealed no statistically significant differences in all lipid profile except HDL-C levels

5.2. Recommendations

Indirectly, Low lipid profile can aware the medical team for the presence of HCV infection
Use of lipid profile as an indicator for severity and improvement in HCV infection.

Reference:


41. Butt A.A.2, Yan P., Tracey G. et al. Changes in circulating lipids level over time after acquiring HCV infection 5 November 2015