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Assessment of lipid profile in patients suffered from hepatitis C viral disease

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Abstract
The liver is the principal site for formation and clearance of lipoproteins. It receives fatty acids and cholesterol from peripheral tissues and diet, packages them into lipoprotein complexes, and releases these complexes back into the circulation. Thus, in severe liver disease, lipid metabolism is profoundly disturbed. Hepatitis C viral (HCV) disease has been linked with dyslipidemia. Based on above literature findings the present study was planned to assess the effect of HCV on lipid metabolism by observing the variations in serum lipoproteins (total cholesterol, low-density lipoprotein [LDL], high-density lipoprotein [HDL], very LDL [VLDL], and triglycerides [TGs]) in chronic hepatitis C viral patients.
The study was conducted in the Career Institute of Medical Sciences And Hospital. We had conducted the study on about 60 patients admitted. The patients involved were suffering from HCV irrespective of their etiology.
Hence from above findings it can be concluded that the advancement of Hepatitis C viral infection can lead to decrease level of lipid profile. Lipid profile monitoring may help in the diagnosis of hepatic infection severity and may also act as a good prognostic sign, so it must be analysed in all advanced hepatic infection cases.

Keywords: lipids profile, hepatitis C viral (HCV) disease, HDL, LDL, TG, VLDL

Introduction
Hepatitis C virus (HCV) causes both acute and chronic infection. Acute HCV infection is usually asymptomatic, and is only very rarely (if ever) associated with life-threatening disease. About 15–45% of infected persons spontaneously clear the virus within 6 months of infection without any treatment.
The remaining 60–80% of persons will develop chronic HCV infection. Of those with chronic HCV infection, the risk of cirrhosis of the liver is between 15–30% within 20 years.
Hepatitis C is found worldwide. The most affected regions are WHO Eastern Mediterranean and European Regions, with the prevalence of 2.3% and 1.5% respectively. Prevalence of HCV infection in other WHO regions varies from 0.5% to 1.0%. Depending on the country, hepatitis C virus infection can be concentrated in certain populations (for example, among people who inject drugs) and/or in general populations. There are multiple strains (or genotypes) of the HCV virus and their distribution varies by region.
The hepatitis C virus is a bloodborne virus. It is most commonly transmitted through:
- Injecting drug use through the sharing of injection equipment;
- The reuse or inadequate sterilization of medical equipment, especially syringes and needles in healthcare settings; and
- The transfusion of unscreened blood and blood products.

HCV can also be transmitted sexually and can be passed from an infected mother to her baby; however these modes of transmission are much less common.

Hepatitis C is not spread through breast milk, food, water or by casual contact such as hugging, kissing and sharing food or drinks with an infected person.
Estimates obtained from modelling suggest that worldwide, in 2015, there were 1.75 million new HCV infections (globally, 23.7 new HCV infections per 100 000 people).
The incubation period for hepatitis C is 2 weeks to 6 months. Following initial infection, approximately 80% of people do not exhibit any symptoms. Those who are acutely symptomatic may exhibit fever, fatigue, decreased appetite, nausea, vomiting, abdominal pain, dark urine, grey-coloured faeces, joint pain and jaundice (yellowing of skin and the whites of the eyes).
Due to the fact that acute HCV infection is usually asymptomatic, few people are diagnosed during the acute phase. In those people who go on to develop chronic HCV infection, the infection is also often undiagnosed because the infection remains asymptomatic until decades after infection when symptoms develop secondary to serious liver damage.
HCV infection is diagnosed in 2 steps:
- Screening for anti-HCV antibodies with a serological test identifies people who have been infected with the virus.
- If the test is positive for anti-HCV antibodies, a nucleic acid test for HCV ribonucleic acid (RNA) is needed to confirm chronic infection because about 30% of people infected with HCV spontaneously clear the infection by a strong immune response without the need for treatment. Although no longer infected, they will still test positive for anti-HCV antibodies.

After a person has been diagnosed with chronic hepatitis C

infection, they should have an assessment of the degree of liver damage (fibrosis and cirrhosis). This can be done by liver biopsy or through a variety of non-invasive tests.

In addition, these people should have a laboratory test to identify the genotype of the hepatitis C strain. There are 6 genotypes of the HCV and they respond differently to treatment. Furthermore, it is possible for a person to be infected with more than 1 genotype. The degree of liver damage and virus genotype are used to guide treatment decisions and management of the disease [1].

The liver is the principal site for formation and clearance of lipoproteins. It receives fatty acids and cholesterol from peripheral tissues and diet, packages them into lipoprotein complexes, and releases these complexes back into the circulation [2]. Thus, in severe liver disease, lipid metabolism is profoundly disturbed [3]. Chronic hepatitis C viral (CHCV) disease has been linked with dyslipidemia [4].

Liver plays a central role in lipid metabolism as several pathways are at least in part dependent to this site. Major metabolic processes take place at this level involving the production transportation and storage of apoproteins and lipoproteins as well as catabolism of various lipids and excretion of cholesterol and phospholipids and alteration in liver function resulting from cellular injury leads to change in the serum concentration of cholesterol and lipoproteins. Infection with (HCV) leads to hepatic damage which in turn relates to change in alterations of lipid metabolism. Different mechanisms involved dependent on the stage of the liver disease and the metabolic state. Low levels of plasma cholesterol and lipoproteins as well as lower triglyceride (TG) values are usual in chronic liver disease. However, the number of studies which included patients with advanced cirrhosis remains low [5].

Based on above literature findings the present study was planned to assess the effect of HCV on lipid metabolism by observing the variations in serum lipoproteins (total cholesterol, low-density lipoprotein [LDL], high-density lipoprotein [HDL], very LDL [VLDL], and triglycerides [TGs]) in chronic hepatitis C viral patients.

Methodology

The study was conducted in the Career Institute Of Medical Sciences And Hospital, Lucknow, We had conducted the study on about 60 patients admitted. The patients involved were suffering from HCV irrespective of their etiology.

The patients suffering from diseases like which can alter the lipid profiles are not involved in the study. The examples of the lipid lowering diseases like diabetes mellitus, cancer, acute pancreatitis, recent parenteral nutrition and acute gastrointestinal bleeding, renal failure, patients who were on glucose or lipid lowering drugs.

The lipid profiles of the patients were done on fasting state. The samples were sent for the analysis and results were compiled and studied further.

Results & Discussion

The data from the 30 patients found positive for the HCV were collected and presented as below. The 30 patients were as studied parallel as a control group. The assessment parameters includes the mainly the variations in serum lipoproteins (total cholesterol, low-density lipoprotein [LDL], high-density lipoprotein [HDL], very LDL [VLDL], and triglycerides [TGs]).

Table 1: Demographic Details

	HCV group	Control Group
Number of Cases	30	30
Age of the Patients (years)	29 – 43	30 – 45
Waist (Cm)	65 – 74	63 – 75
BMI (kg/cm2)	21.5 – 27.6	22.2 – 28.3

Table 2: Blood Pressure

	HCV group	Control Group
Number of Cases	30	30
Systolic BP (mmHg)	115-131	117-133
Diastolic BP (mmHg)	76-91	79-93

Table 3: Lipid Profile

	HCV group	Control Group
Total cholesterol	150.6- 175.3	191.6- 226.8
LDL	88.3 – 103.5	98.6 – 117.4
HDL	43.2 – 50.2	46.2 – 51.5
Triglycerides	106.8 – 121.3	152.8 – 176.3

Our study found a statistically low total and HDL-C in the ungrouped, asymptomatic, and symptomatic CHC disease compared with their matched controls. Low LDL-C was also observed in all the groups compared with their matched controls, the decrease was, however, not statistic.

The study observed no disparity in the levels of VLDL-C and TG of the groups compared with their controls. Our study is consistent with the works of Maggi *et al*., Fabris *et al*., and Serfaty *et al*., who reported a higher prevalence of hypocholesterolemia and low LDL levels in HCV-infected patients compared to control groups [6].

The low HDL-C observed in our study is consistent with a study led by Li *et al*., reporting LDL and HDL values to be lower in HCV-infected patients [7]. The study is also in line with that of Nogueira *et al*., which evaluated 150 HCV-infected patients' genotypes 1, 2, and 3, showed that serum HDL values were lower in genotype 2 HCV-infected patients [8].

The spectrum of severity of chronic hepatitis C varies widely as done the rate of its progression to the cirrhotic stage. This heterogeneity largely depends on host and environmental factors, although the contributing role of viral features such as the HCV genotype has recently been revisited. Cofactors influencing hepatitis C severity and progression include age, gender, and excess alcohol consumption, co infection with other hepatotropic viruses and/or HIV and the metabolic syndrome. The role of the latter in the pathogenesis of hepatitis C has attracted considerable attention in recent years. HCV virion circulate in serum bound to lipoproteins; lipids have been shown to modulate (and indeed are essential for) the HCV life cycle and an occasionally severe accumulation of triglycerides in hepatocytes is observed in a distinct subgroup of patients in the form of fatty liver. In summary lipid metabolism shows widespread alteration conferring an idiosyncratic profile to HCV infection. This review will

discuss these aspects focusing on both their molecular mechanisms and their clinical consequences.

Conclusion

Hence from above findings it can be concluded that the advancement of Hepatitis C viral infection can lead to decrease level of lipid profile. Lipid profile monitoring may help in the diagnosis of hepatic infection severity and may also act as a good prognostic sign, so it must be analysed in all advanced hepatic infection cases.

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