

Original Research Article

To study the changes in lipid profile induced after ingestion of single high-cholesterol test meal in subjects of chronic liver disease and chronic renal disease

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Abstract

Chronic kidney disease (CKD), also known as chronic renal disease, is progressive loss in kidney function over a period of months or years. The symptoms of worsening kidney function are not specific, and might include feeling generally unwell and experiencing a reduced appetite. Chronic liver disease in the clinical context is a disease process of the liver that involves a process of progressive destruction and regeneration of the liver parenchyma leading to fibrosis and cirrhosis. "Chronic liver disease" refers to disease of the liver which lasts over a period of six months. It consists of a wide range of liver pathologies which include inflammation (chronic hepatitis), liver cirrhosis, and hepatocellular carcinoma. The entire spectrum need not be experienced. Lipid profile or lipid panel is a panel of blood tests that serves as an initial broad medical screening tool for abnormalities in lipids, such as cholesterol and triglycerides. The results of this test can identify certain genetic diseases and can determine approximate risks for cardiovascular disease, certain forms of pancreatitis, and other diseases. Aim of the present study To study the changes in Lipid Profile

Induced after Ingestion of Single High-Cholesterol Test Meal in Subjects of Chronic Liver Disease and Chronic Renal Disease. Materials and methods, present study was conducted in the department of General Medicine, RIMS, Kadapa, by taking 50 patients of both the sexes. Serum was separated within four hours by centrifugation and the tests are serum total cholesterol (STC), serum Triglycerides (STG), HDL-cholesterol, LDL-cholesterol and VLDL-Cholesterol. Statistical analysis of the data was done by using paired 't' test and student 't' test. As no such comparative prior studies have been done on COPD patients, it was strongly urged that further studies with larger sample groups be carried out to elucidate the quantitative and qualitative significance of these changes.

Key words

Chronic liver disease, Chronic renal disease, HDL-C, LDL-C.

Introduction

The relation between the levels of dietary and serum cholesterol has attracted the attention of the scientific community since Anichkovs cholesterol feeding experiments in rabbits at the beginning of this century.

The clinical importance of plasma lipoprotein levels derived from the ability of plasma lipoproteins to cause two life threatening diseases, pancreatitis and atherosclerosis. Among the most important risk factors are diabetes, hypertension, stress, obesity Hypercholesteremia and predisposition [1]. An abnormal lipid level is a factor that may be a factor that common to several of them. Diet is also plays a major role in modifying the lipid profile of both healthy as well as disease persons. Modification of diet has been Shows to result in progression or regression of atherosclerotic lesions in several experimental models.

Chronic liver diseases due to various causes are often associated with dramatic reductions in plasma triglyceride and cholesterol level due to reduced lipoprotein biosynthetic capacity [2]. Cholestasis is associated with hypercholesterolemia as the major excretory pathway of cholesterol is blocked in this disorder. Apart from the various complications seen in cirrhotic patients, chronic dyslipoproteinemia is one which can lead to alterations in cellular membrane lipids, that result in formation of abnormal RBCs, such as

echinocyte, and alterations in membrane function with potential pathophysiologic consequences [3]. Although several studies have been conducted on dyslipidemia in cirrhotics in developed countries, there is a paucity of data in this regard in India. As there is a high prevalence of chronic liver disease in our country, we conducted this study to determine lipid profile in patients with cirrhosis and to assess if it relates to the severity of chronic liver disease [4].

Chronic Kidney Disease (CKD) is becoming a worldwide health problem due to increasing incidence and prevalence, high cost, and poor outcomes [5]. It is a pathophysiological process with multiple etiologies resulting in the inexorable attrition of functional nephrons and frequently leading to End Stage Renal Disease (ESRD) necessitating hemodialysis as a mandatory therapeutic measure. The Kidney Disease Outcomes Quality Initiative defines chronic kidney disease as either kidney damage or a decreased kidney Glomerular Filtration Rate (GFR) of less than 60 ml/min/1.73 m² for 3 or more months [6].

The Chronic Kidney Disease (CKD) is characterized by specific metabolic abnormalities of plasma lipids both qualitatively and quantitatively [7]. Most common lipid abnormalities encountered are increased serum triglycerides and decreased serum HDL cholesterol with small alteration of other lipoprotein fraction in serum and in dialysis patients there is more of a dyslipidemia rather

than hyperlipidemia [8]. This may be a significant risk factor for vascular complications leading to increased morbidity and mortality in CKD patients [9].

The emulsification of dietary fats renders them accessible to various pancreatic lipases in the small intestine. These lipases, pancreatic lipase and pancreatic phospholipase A₂ (PLA₂) generate free fatty acids and a mixture of mono- and diglycerides from dietary triglycerides [10-13]. Phospholipids are degraded at the *sn*-2 position by pancreatic PLA₂ releasing a free fatty acid and the lysophospholipid. The products of pancreatic lipases then enter the intestinal epithelial cells via the action of various transporters as well as by simple diffusion. Within the enterocyte the lipids are used for re-synthesis of triglycerides [14].

Dietary triglyceride and cholesterol, as well as triglyceride and cholesterol synthesized by the liver, are solubilized in lipid-protein complexes. These complexes contain triglyceride lipid droplets and cholesteryl esters surrounded by the polar phospholipids and proteins identified as apolipoproteins [15]. These lipid-protein complexes vary in their content of lipid and protein.

A lipid disorder means that you have high levels of either low-density lipoprotein (LDL) cholesterol, or elevated levels of fats called triglycerides. If you have high LDL cholesterol or high triglycerides, you probably have an increased risk for developing heart disease [16]. The two major forms of cholesterol found in your body are high-density lipoprotein (HDL) and low-density lipoprotein (LDL). HDL or good cholesterol has a protective effect on your heart. HDL transports harmful cholesterol out of your arteries. Doctors usually recommend that you have a high level of HDL cholesterol. LDL or bad cholesterol is made by your body and also absorbed from cholesterol-rich foods such as red meat and dairy [17-19]. LDL can combine with other fats and substances, creating blockages in

your arteries. This can reduce your blood flow and cause serious health problems. A triglyceride is a type of fat you get from the food you eat. Your body also produces it when it converts excess calories to fat for storage. Some triglycerides are necessary for the proper cell functions, but too much is unhealthy. People with high cholesterol often have a raised level of triglycerides [20].

Materials and methods

The present study was conducted on Fifty (50) patients of Myocardial infarction admitted in Medical Wards and casualty of RIMS, Kadapa, (with mean age 15-60 years) during the period of 2015-2016.

The case subjects for the present study consisted of male and female patients suffering from chronic liver disease and chronic renal disease. An informed consent was taken from all the subjects who to be included in the study. In each case a detailed history was elicited and a meticulous clinical examination and investigations were carried out, and all basic clinical and biochemical parameters recorded.

The subjects are divided into two groups:

First group: In this group consisted of 25 patients of both sexes suffering from chronic renal failure. The age range of this group was 20-55 years.

Second group: In this group consisted of 25 patients of both sexes suffering from hepatic cirrhosis. The age range of this group was 15-65 years.

Design of test: All subjects were asked to have their dinner at 7.00 PM on the previous night and not to talk anything except water till the next morning. Fasting blood sample were taken at 8.00 AM the following morning in the recumbent posture without producing venous stasis. After this they were given a test meal consisting of 2 boiled eggs and 280 ml of whole fat buffalo

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milk. This supplied 500-550 mg of egg yolk cholesterol.

Postprandial blood samples were taken 1, 2 and 3 hours after the meals. During the test the subjects were not allow to talk anything except water. Smoking was prohibited during the test period. Serum was separated within four hours by centrifugation and the tests are serum total cholesterol (STC), serum Triglycerides (STG), HDL-cholesterol, LDL-cholesterol and VLDL-Cholesterol. Statistical analysis of the data was done by using paired 't' test and student 't' test.

Results

The present study was conducted in the department of General Medicine at RIMS,

Kadapa, Andhra Pradesh with 50 patients of both the sexes. These are divided into two groups, first group (chronic renal disease), second group (chronic liver disease).

First group: it comprised of 25 male and female patients of chronic renal disease, ranging in age from 20 to 55 years with a mean age of 38.88 ± 13.93 years, mean weight of 52.44 ± 7.5 kg and a mean height of 149.8 ± 7.0 cm.

Second group: it comprised of 25 male and female patients of chronic liver disease, ranging in age from 15 to 65 years with a mean age of 48.58 ± 17.93 years, mean weight of 42.44 ± 12.5 kg and a mean height of 150.38 ± 14.80 cm (**Table – 1 to 5**).

Table - 1: Effect of single high cholesterol test diet on mean STC levels in subjects of first group, second group (mg/dl). Mean \pm SD values.

| Groups | Fasting | After 1 hour | After 2 hours | After 3 hours |
|--------------|--------------------|--------------------|--------------------|--------------------|
| First group | 286.33 \pm 74.99 | 303.22 \pm 81.12 | 310.34 \pm 82.99 | 320.22 \pm 80.23 |
| Second group | 160.45 \pm 47.99 | 182.99 \pm 56.88 | 180.89 \pm 61.93 | 181.31 \pm 62.98 |

Table - 2: Effect of single high cholesterol test diet on mean HDL-Cholesterol levels in subjects of first group, second group (mg/dl). Mean \pm SD values.

| Groups | Fasting | After 1 hour | After 2 hours | After 3 hours |
|--------------|-------------------|-------------------|-------------------|-------------------|
| First group | 54.33 \pm 18.99 | 65.32 \pm 21.12 | 67.34 \pm 33.99 | 75.87 \pm 39.23 |
| Second group | 26.45 \pm 13.59 | 32.79 \pm 56.88 | 30.89 \pm 15.63 | 31.31 \pm 16.98 |

Table - 3: Effect of single high cholesterol test diet on mean LDL-Cholesterol levels in subjects of first group, second group (mg/dl). Mean \pm SD values.

| Groups | Fasting | After 1 hour | After 2 hours | After 3 hours |
|--------------|--------------------|--------------------|--------------------|--------------------|
| First group | 154.33 \pm 38.99 | 145.42 \pm 31.72 | 120.34 \pm 33.99 | 119.87 \pm 69.23 |
| Second group | 112.45 \pm 23.59 | 115.79 \pm 36.88 | 104.89 \pm 35.63 | 111.31 \pm 42.98 |

Table - 4: Effect of single high cholesterol test diet on mean STG -Cholesterol levels in subjects of first group, second group (mg/dl). Mean \pm SD values.

| Groups | Fasting | After 1 hour | After 2 hours | After 3 hours |
|--------------|---------------------|---------------------|---------------------|---------------------|
| First group | 454.63 \pm 138.99 | 545.42 \pm 221.72 | 584.34 \pm 233.69 | 619.97 \pm 269.23 |
| Second group | 142.05 \pm 73.59 | 165.79 \pm 96.88 | 174.82 \pm 95.63 | 115.71 \pm 98.68 |

Table - 5: Effect of single high cholesterol test diet on mean LDL/HDL-Cholesterol levels in subjects of first group, second group (mg/dl). Mean± SD values.

| Groups | Fasting | After 1 hour | After 2 hours | After 3 hours |
|--------------|-----------|--------------|---------------|---------------|
| First group | 4.63±1.99 | 3.42±1.72 | 2.34±1.39 | 2.47±1.23 |
| Second group | 4.05±1.29 | 3.79±2.98 | 4.62±2.37 | 5.71±2.68 |

Discussion

The present study was conducted in the department of General Medicine at RIMS, Kadapa, Andhra Pradesh with 50 patients of both the sexes. These are divided into two groups, first group (chronic renal disease), second group (chronic liver disease).

Changes in First Group

Changes in Serum total cholesterol: the highest fasting as well as postprandial STC levels were observed in this group of patients, although the postprandial rise in STC was only slight. Hyperlipidaemia is a well known feature of chronic renal disease. Cholesterol ester turnover has been found to be raised together with triglyceride turnover in patients with the nephrotic syndrome.

Changes in HDL cholesterol: this was the only group which the HDL-cholesterol shows a marked rise from the basal to the postprandial levels, a rise of almost 40 percent over the fasting level. Since 24 out of 25 patients were of nephrotic syndrome, understandably, the hyperlipidemia associated with this condition has overtaken the overall mean HDL-C levels. Joven and Villadona, et al. (1990) stated that overproduction of lipoproteins containing apo proteins is the principal cause of hyperlipidemia in these patients [21] but Karadi and Romics, et al. (1989) found that serum lipoprotein (a) levels may be increased [22].

Changes in LDL cholesterol: the fasting levels decreased gradually after 3 hours. The marked progressive rise of HDL-C levels could be held responsible for affecting the derivation of LDL cholesterol values.

Changes in Serum Triglycerides (STG): Hypertriglyceridemia is a characteristic feature of nephrotic syndrome as is apparent from the markedly high fasting and postprandial values rising at 3 hours.

Murase, et al. (1975) provided evidence of a factor in uremic plasma which inhibits lipoprotein lipase activity. This results in decreased clearance of triglycerides from plasma [23].

Changes in VLDL: changes in VLDL are exactly similar to changes in STG.

Changes in First Group

Changes in Serum total cholesterol: The mean fasting STC was lowest to the First group. There was a pronounced rise after 1 hour of diet ingestion, with marginally lower subsequent values—all postprandial values being above the fasting one. A rise in STC levels at first hour. Stigendahl, et al. (1984) have stated that there is reduced synthesis of endogenous cholesterol in cirrhotics [24].

Changes in HDL cholesterol: the HDL cholesterol increased after 1 hour but thereafter displayed only very marginal changes. The slight decrease shown by some patients at 2 hours appeared to be a transient phenomenon, as the values rise again at 3 hours. We recommend further studies to explain the significance of the above findings in our study.

Changes in LDL cholesterol: A slight rise in LDL-cholesterol at 1 hour, after 3 hours it shows rising the values.

Changes in Serum Triglycerides (STG): 24 of the 25 patients showed a gradual postprandial rise in STG at peaking at 3 hours. Schaefer, et al. (1985) have also reported increased levels of TG, chylomicrons and VLDL in patients of primary biliary cirrhosis and have attributed these abnormalities to hepatic lipase inhibition and altered cholesterol esterification [25].

Changes in VLDL: changes in VLDL are exactly similar to changes in STG.

This is the greatest magnitude of rise in STG in the postprandial phase seen in the two disease group in this study. Even though the rise was not statistically significant. We believe that the relatively large standard deviation along with the small sample size could be responsible for the same and therefore strongly urge that further studies with larger sample group be carried out to elucidate the qualitative and quantitative significance of these changes.

Conclusion

In the present work 25 male and female patients of chronic renal disease, 25 male and female patients of chronic liver disease were studied to see the response of an individual to stress of single high cholesterol test load in disease state.

Highest fasting as well as postprandial levels of STC, a marked rise from the basal to the postprandial levels of HDL- C, raising almost 42 percent over to the fasting levels, 3 hours after ingestion of the test diet. The LDL cholesterol concentration in first group patients showed a steady decline from fasting to the postprandial phase, reaching the lowest concentration 3 hours after ingestion of high cholesterol test diet. Highest fasting and postprandial levels of serum triglycerides were observed in first group patients of chronic renal disease with the 3 hours mean value being 34 percent higher than the mean fasting value. The difference was not statistically significant, hence further studies with large groups were recommended to properly quantify the changes.

The mean fasting STC was lowest in second group. However all patients in this group shows a rise in STC levels at 1 hour. Two distinct trends were noticed in the changes in LDL concentration in second group patients. Majority of the patients showed a rise in LDL levels at 1 hour and continued to show a further rise at 3 hours, some patients showed a fall in LDL at 1 hour and continued to show a further fall at 3 hours.

As no such comparative prior studies have been done on COPD patients, it was strongly urged that further studies with larger sample groups be carried out to elucidate the quantitative and qualitative significance of these changes.

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