Original Research Article

Comparative study of serum lipid profile level between chronic kidney disorder patients and healthy controls

Ketan Mangukiya¹, Sarita H Patel²*, Milav Bhavsar³, Alok Parekh², Rinku Bhanavadiya⁴

¹Dept. of Biochemistry, Parul Institute of Medical Sciences & Research, Vadodara, Gujarat, India
²Dept. of Biochemistry, Government Medical College, Surat, Gujarat, India
³Dept. of Biochemistry, C.U Shah Medical College, Surendranagar, Gujarat, India
⁴Dept. of Biochemistry, GMERS Medical College, Junagadh, Gujarat, India

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A B S T R A C T

Introduction: Several factors contribute to atherogenesis and cardiovascular disease in patients with CKD. Notable among the CKD-induced risk factors are lipid disorders, oxidative stress, inflammation, physical inactivity, anemia, hypertension, vascular calcification, endothelial dysfunction, and depressed nitric oxide availability.

Objective: The objective is to Measure lipid profile in a patients of chronic kidney disorder (CKD) and compare their level with healthy control.

Materials and Methods: Study includes total 100 participants. Among them 50 patients of CKD of age group between 20 to 70 year and 50 healthy controls. Fasting blood samples were collected from both groups and measurement of the lipid profile, FBS, PPBS, serum creatinine and blood urea was done.

Results: There is increased level of serum cholesterol and triglyceride was found in CKD patients as compared to control group but when we compared it with control group by calculating p value the difference of significance was found only in case of cholesterol level. Serum LDL was also significantly raised in patients of CKD compared to controls.

Conclusion: There is significant amount of dyslipidemia is found in patients of CKD. So treatment of dyslipidemia will reduce mortality in CKD patients.
2. Materials and Methods

This cross sectional study was conducted at SBKS medical institute and research center, Dhraj hospital, Piparia, Vadodara, Gujarat from January 2011 to January 2012 after obtaining informed consent of the patients.

This study includes all the patients with chronic kidney disease (CKD) of age group 20-70 year irrespective of cause and sex who visited our institute.

2.1. Study Design

Study includes total 100 participates of age 20-70 year (n=100).

Group 1: confirm cases of CKD (n=50)
Among them 20 diabetics and 30 non diabetics

Group 2: Healthy controls (n=50)

2.2. Exclusion criteria

Renal transplant patients, patients on lipid lowering drugs, hemodialysis or peritoneal dialysis patients, age < 20 yrs were excluded from the study.

Consent was taken from all participants before taking blood samples of participants. Detail history of all patients was taken. An Advice was given to all participants of the study for a fasting of 10 hr and sample was collected from all participants in fluoride and plain vacutainer.

An Individual ID was given to all participants to hidden identity of the patients and all collected samples were centrifugated at clinical biochemistry laboratory at 3000 RPM for a period of 10 minutes.

Plasma was separated from fluoride vacutainer and serum was separated from plain vacutainer.

Following parameters were analyzed by colorimetric method on fully automated biochemistry analyser.

1. S. Cholesterol (CHOD PAP method
2. S. Triglyceride (GPO PAP method
3. S.HDL (Precipitation method
4. S.LDL ( Friedewald formula
5. S.VLDL (calculati on method
6. FBS(GOD POD method
7. PPBS(GOD POD method
8. S.Creatinine (Modifie d Jaffe’s reaction
9. B.urea (GLDH-Urease Method

2.3. Statistical analysis

Mean and standard deviation was calculated and the same represented by graphs. Student’s t test was used to calculate the significance of difference between two group by calculating p value.

3. Result

Among total 100 participants, they were divided in to various age group as mentioned in Table 1.

Total male in CKD group is 31(62%) and total female is 19(38%).

Average age of male CKD patients is 44.46 year while that of female is 40.26 year.

In Table 2, Mean values for urea in controls and patients showed a considerable difference, which was found to be highly significant (P<0.001).

Creatinine levels in CKD patients were very high as compared to controls. This difference was statistically significant (P<0.001).

Table 3 shows that there is increase level of cholesterol found in CKD patients as compared to control group and the difference between them is significant.

Samething found in serum triglyceride level but difference among them is not significant. Serum LDL cholesterol is significantly increased in CKD patients as compared to control group and p value is <0.05.

The variation in level of serum HDL was not found significant.

Serum VLDL level is found to be increased in CKD group and when it compared with control group but the difference among them is not significant.

The study group was analysed with the risk factors associated with increased cardiovascular mortality. It was found that patients with diabetes comprised 40 percent of the study population while the remaining 60 percent were non diabetic.

Among the 50 patients included in the study 42 percent (21patients) had hypertension as a comorbid condition.

Table 6 shows difference in total cholesterol values among CKD patients.

27 patients (54%) had total cholesterol <200mg/dl (desirable range) and 23 patients had abnormal value, among them 13 (26%) patients had borderline high (200-239) and 10 patients had high values (>240) (20%). This was statistically significant (P<0.05)

The mean values of the different fractions were obtained with respect to the comorbid conditions present in the study sample. The comparative mean values of the different fractions of lipid in the hypertensive subjects with those who were not hypertensive is depicted in bar diagram below.

4. Discussion

In a study by Vaziri N D it was found that the patients had elevated serum triglycerides level, with high levels of VLDL and pre β HDL. However, the levels of HDL and apoA were reduced. This study included, primarily patients with nephrotic syndrome and the elevated triglycerides were found to be directly proportional to the levels of proteinuria.

Chan C M et al studied the lipid abnormalities in patients with renal failure due to nephrotic syndrome and also due to causes other than nephrotic syndrome. He found that the prevalence of hypertriglyceridemia and the elevation of HDL cholesterol were proportional to
Table 1: Age and sex distribution among CKD patients

| Age group | Total no of cases (%) | Male (%) | Female (%) |
|-----------|-----------------------|----------|------------|
| 21-30     | 06(12%)               | 03(50%)  | 03(50%)    |
| 31-40     | 15(10%)               | 06(40%)  | 09(60%)    |
| 41-50     | 13(26%)               | 10(76%)  | 03(23.08%) |
| 51-60     | 07(14%)               | 05(71%)  | 02(28.57%) |
| 61-70     | 09(18%)               | 07(77%)  | 02(22.22%) |
| Total     | 50(100%)              | 31       | 19         |

Table 2: Biochemical data in controls and CKD patients (Mean±SD) mg/dl.

| Groups | Blood urea | Serum creatinine |
|--------|------------|------------------|
| Controls (n=50) | 22.5±7.3 | 0.72±0.16 |
| Patients n=50 | 146.42±82.67 | 5.01±2.09 |
| P value | <0.001 | <0.001 |

Significance: Highly significant

*Student’s t-test (unpaired) P<0.001 highly significant

Table 3: Biochemical (lipid profile) data in controls and CKD patients

| Group (mg/dl) | Control (n=50) Mean ± SD | Case (n=50) Mean ± SD | t-value* | P value |
|---------------|-------------------------|-----------------------|----------|---------|
| Cholesterol   | 162.48±46.77            | 197.88±57.49          | 3.378    | 0.0011  |
| TG            | 140.46±64.48            | 169.38±102.33         | 1.691    | NS      |
| HDL           | 38.56±6.82              | 38.52±9.04            | 0.025    | NS      |
| LDL           | 102.2±43.39             | 122.14±58.18          | 1.943    | 0.05    |
| VLDL          | 29.1±13.64              | 33.82±20.54           | 1.354    | NS      |

*Students t-test (unpaired), Sig: significant, NS: not significant (P >0.05)

Table 4: Biochemical (lipid profile) data among male and female CKD patients

| Lipid (patients) mg/dl | Male (n=31) Mean ±SD | Female (n=19) Mean±SD | ‘t’ | p-value |
|------------------------|----------------------|-----------------------|-----|---------|
| Total cholesterol      | 199.51±57.49         | 195.21±61.93          | 0.249 | N.S     |
| Triglyceride           | 182.45±102.33        | 148.05±106.94         | 1.134 | N.S     |
| HDL                    | 37.83±9.93           | 39.63±9.68            | 0.673 | N.S     |
| LDL                    | 120.41±58.18         | 124.94±62.79          | 0.259 | N.S     |
| VLDL                   | 36.25±20.54          | 29.84±21.55           | 1.051 | N.S     |

Table 5: Number of DM and Hypertension patients among CKD group

| Disease             | Yes | No |
|---------------------|-----|----|
| Diabetes Mellitus   | 20(40%) | 40(60%) |
| Hypertension        | 21(42%) | 29(58%) |

The severity of renal impairment. He however noticed that the diabetic patients had increased triglycerides and lower HDL suggesting that diabetes itself exacerbated lipid abnormalities. In this study the p value of the CKD patients with diabetes and without diabetes were compared. The p values of S.triglyceride, S.cholesterol , S.HDL and S.LDL which was found to be non significant and not in accordance with the lipoprotein abnormalities seen in the study by Chan C M.

Gerald Appel et al 11 showed increase in very low density lipoproteins (VLDL).

In contrast to the above mentioned studies, in this study total cholesterol and LDL were markedly elevated in CKD patients as compared to control group and it was statistically significant. Triglycerides were elevated compared to control group but it was statistically not significant. The levels of the lipoprotein fractions like HDL and VLDL were within normal range and the p values of the mean were found to be insignificant. The p value of S. Triglycerides, S.Cholesterol, S. HDL, LDL, and VLDL was nonsignificant.

Increase serum triglyceride level in CKD patients as compared to control, may be due to the pathogenesis of most lipid abnormalities in patients with CKD primarily involves defective removal from the circulation. The diminished clearance of triglycerides, which can lead to hypertriglyceridemia, stems both from an alteration in the composition of circulating triglycerides (which become enriched with apolipoprotein C-III) and, perhaps later,
Table 6: Showing values (mg/dl) of S. total cholesterol, S. Triglyceride, S.HDL, S. LDL

| Parameter               | Range               | Male (%) | Female (%) | Total (%) |
|-------------------------|---------------------|----------|------------|-----------|
| Total Cholesterol mg/dl | Desirable <200      | 17(34%)  | 10(20%)    | 27(54%)   |
|                         | Borderline high 200-239 | 04(8%)  | 09(18%)    | 13(26%)   |
|                         | High>240            | 10(20%)  | 00(0%)     | 10(20%)   |
|                         | <150                | 15(30%)  | 13(26%)    | 28(56%)   |
| S. Triglycerides mg/dl  | Borderline high (150-199) | 07(14%)  | 00(0%)     | 07(14%)   |
|                         | High (200-499)      | 09(18%)  | 05(10%)    | 14(28%)   |
|                         | Very high >500      | 00(0%)   | 01(02%)    | 01(02%)   |
|                         | <40                 | 22(44%)  | 10(20%)    | 32(64%)   |
|                         | Borderline high     | 07(14%)  | 00(0%)     | 07(14%)   |
|                         | Near optimal (101-129) | 04(08%)  | 04(08%)    | 08(16%)   |
| S. HDL-C mg/dl          | Borderline high (130-159) | 04(08%)  | 03(06%)    | 07(14%)   |
|                         | High (160-189)      | 04(08%)  | 03(06%)    | 07(14%)   |
|                         | Very high>190       | 05(10%)  | 02(04%)    | 07(14%)   |

from reductions in the activity of lipoprotein lipase and hepatic triglyceride lipase which are involved in triglyceride removal.12

But the rise in serum triglyceride level in CKD patients compared to control was statistically not significant, may be because of the limitation of this study like this study does not include a detailed dietary history and does not compare the caloric intake and the triglyceride levels.

Thomas Quascetning et al13,14 reported combined hyperlipidemia (elevated total cholesterol and triglycerides) in their study.

In a study by Nayak et al15 and colleagues they found that the lipid profile in diabetic and non diabetic patients with CKD had elevated triglycerides, LDL cholesterol and VLDL. They found no statistically significant correlation between diabetic and the non diabetic patients. This study did not find the elevation in the lipoprotein fractions however there was no correlation found amongst the study group with diabetes as the comorbid condition.

This could be attributed to low calories derived from carbohydrates and the high intake of polyunsaturated fatty acids in the diet of most of the people residing in this region. Hence it can be seen that the degree of hypertriglyceridemia in our population is less although the type of hyperlipoproteinemia is the same as that in the Western population. This may be related to the dietary pattern in the form of high intake of polyunsaturated fatty acids.

However the study group in this study was heterogeneous hence the data collected should probably have included a larger group.

5. Conclusion
Cardiovascular disease is a major cause of morbidity and mortality in patients with impaired renal function. Dyslipidemia has been established as a well-known traditional risk factor for cardiovascular disease (CVD) in the general population and it is well known that patients with chronic kidney disease (CKD) exhibit significant alterations in lipoprotein metabolism. There is significant amount of dyslipidemia is found in patients of CKD. So treatment of dyslipidemia will reduce mortality in CKD patients.

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None.

8. Conflicts of interest
None

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Author biography

**Ketan Mangukiya** Associate Professor

**Sarita H Patel** Assistant Professor

**Milav Bhavsar** Associate Professor

**Alok Parekh** Assistant Professor

**Rinku Bhanavadiya** Tutor

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