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Original Research Article

Study of Lipid profile in chronic kidney disease patients

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Abstract

Background: Chronic kidney disease (CKD) is associated with premature atherosclerosis and increased incidence of cardiovascular morbidity and mortality. 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: To assess the pattern of lipid profile in chronic kidney disease patients along with comparison of lipid profile in chronic kidney disease patients with diabetes mellitus and without diabetes mellitus.

Methods: A total of 50 patients of chronic kidney disease (CKD)of age group 20-70 year and 50 age and sex matched healthy controls were included in this study. Blood specimens were collected from both groups and measurement of the lipid prfile, FBS, PPBS, S.creatinine and blood urea was done and compare results with the study group.

Results: Total cholesterol and LDL was significantly raised in patients of CKD compared to controls The triglycerides were elevated. However, the statistical analysis showed that it was not significant. There was statistically significant variation found in triglyceride, VLDL and HDL when compared with the comorbid conditions of hypertension.

Conclusion: There is significant amount of dyslipidemia is found in patients of CKD. so treatment of dyslipidemia will reduce mortality in CKD patients.

Keywords: CKD, lipid Profile, Hypertension, Diabetes mellitus.

Introduction

In chronic kidney disease the most prevalent lipid disorders are hypertriglyceridemia and decreased HDL Concentration. LDL levels are usually normal or marginally increased^[1,2]. Also there are reports available regarding accelerated atherosclerosis in chronic kidney disease due to

altered lipid metabolism. In recent years, the levels of high-density lipoproteins have gained importance in view of the fact that increasing reports are available incriminating decreased HDL levels as one of risk factors for cardiovascular disease.

Progressive renal failure especially when associated with proteinuria is accompanied by abnormalities of lipoprotein transport. Typically, dyslipidemia reflected the is predominantly in increased serum levels of triglycerides with high levels of VLDL, apoB and pre β HDL and low levels of HDL and of apoA. Cholesterol levels may be very high in proteinuric patients.

Increased levels of atherogenic lipoproteins, especially LDL and possibly chylomicrons remnants, contribute to the development of atherosclerosis. Increased plasma concentration and reduced diameter favor sub endothelial accumulation of these lipoproteins. Following chemical modification such as oxidation, the lipoproteins are no longer clear by normal mechanisms. They trigger a self-perpetuating inflammatory response during which they are taken up by macrophages to form foam cells – a hallmark of the atherosclerotic process. Atherogenic lipoproteins also have an adverse effect on the endothelial function ^[4]. The arterial narrowing that follows impairs the blood supply to various organs.

Materials and Methods

This cross sectional study was conducted at SBKS medical institute and research center, Dhiraj 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.

Study Design

Lipid profiles and classical risk factors were assessed in 50 consecutive samples of chronic

kidney disease patients (20 diabetics and 30 non diabetics) attending the DHIRAJ HOSPITAL, SUMANDEEP UNIVERSITY. Samples characteristics were compared to those of the general population derived from 50 individuals of age Group 20-50 years.

Exclusion Criteria: Renal transplant patients, Patients on lipid lowering drugs, Hemodialysis or peritoneal dialysis patients, Age < 20 yrs were excluded from our study.

Patients presenting to the hospital and diagnosed with CKD were included in the study after obtaining informed consent until 50 cases (20 diabetics and 30 non diabetics) were collected. The history of the onset, progression, duration of various symptoms, drug and diet history was noted. Overnight fasting samples were taken and sent to laboratory immediately. Serum was separated within 2 hours after collection to prevent artifactal change in concentration of HDL. After the clot retraction occurred, the serum was transferred out to a centrifuge tube and centrifuged at 2000 rpm for 5 minutes. The supernatant clear serum was then pipetted out using dry piston pipettes with disposable tips and stored in dry thin walled vials at 4°C. The samples were analysed on the same day or within 48 hours. Following parameters were analyzed by colorimetric method fully automated on biochemistry analyser.

1)S. Cholesterol (CHOD PAP method- Normal range 150-250 mg/dl)

2)S.Triglyceride (GPO PAP method-Normal range : 40 – 160 mg/dl)

3)S.HDL (Precipitation method -Normal range : 35 – 79 mg/dl)

4)S.LDL(Friedewald formula-Normal range: Upto 130 mg/dl

5)S.VLDL (calculation method-Normal range:<32 mg/dl

6)FBS (GOD POD method- Normal range: 70 – 110 mg/dl)

7)PPBS (GOD POD method- Normal range: 70 – 140 mg/dl)

8)S.Creatinine (Modified Jaffe's reaction -Normal

range:0.6 - 1.3 mg/dl)

9)B.urea (GLDH-Urease Method -Normal range: 0.6 – 1.3 mg/dl)

Mean and standard deviation was calculated and the same represented by graphs. Student's t test was used to calculate the significance between means.

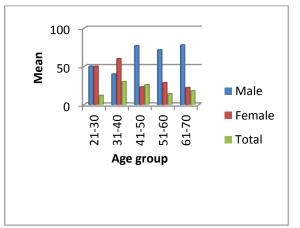
Results

Fifty patients of chronic kidney disease and 50 normal subjects (controls) were taken for present study.

Lipid levels like TC, TG, LDL and VLDL were estimated for both controls and CKD patients, and were compared.

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%)	039(23.08%
51-60	07(14%)	05(71%)	02(28.57%)
61-70	099(18%)	07(77%)	02(22.22)
Total	50(100%)	31	19

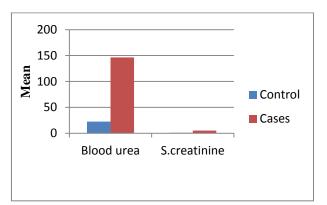


Graph 1: Age and sex distribution among CKD patients

In the present study, 50 patients of CKD were included, out of which 31 patients (62%) were male and 19 patients (38%) were females. On decade wise grouping, we found maximum number of patients between 31-40 years (60%). The mean age for the total number of patients was 44.46. The mean age for male patients was 47.25. The mean age for female patients was 40.26. **Table 2:** Biochemical data in controls and CKD patients (Mean±SD) mg/dl.

	, 0	
Groups	Blood urea	Serum creatinine
Controls	22.5±7.3	0.72±0.16
(n=50)		
Patients n=50	146.42±82.67	5.01±2.09
t-value*	10.558	14.47
Significance	Highly	Highly
_	significant	significant

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



Graph 2: Biochemical data in controls and CKD patients.

In Table/Graph 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: Biochemical (lipid profile) data incontrols and CKD patients (Mean± SD)

Group (mg/dl)	Control (n=50)	Case (n=50)	t- value *	Significanc e
Cholester ol	162.48±46.7 7	197.88±57.49	3.378	Sig (P = 0.0011)
TG	140.46±64.4 8	169.38±102.3 3	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	Sig (P = 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)

200 180 160 140 120 Mean 100 Control 80 Cases 60 40 20 0 Cholesterol Sr. 4D1 ND 26

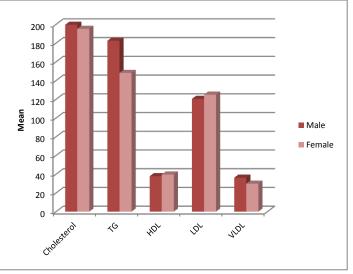
Graph 3: Biochemical (lipid profile) data in controls and CKD patients (Mean)

Table/Graph-3 shows Biochemical (lipid profile) data in controls and CKD patients. Total cholesterol value in controls and CKD patients and 197±57.49 were 162.48±46.77 mg/dl respectively (P<0.05) and this was statistically significant. Triglyceride values in patients and controls were 169±102.33 and 140±64.48 mg/dl respectively. Triglycerides values in patients of CKD were significantly high compared to controls. However this difference was statistically not significant (P>0.05). HDL values were almost similar in CKD patients and control, 38.52±9.04 and 38.56±6.82 mg/dl respectively (P>0.05). This was also statistically not significant. VLDL values in patients and controls were 33.82±20.54 and 29.1±13.64 mg/dl respectively. VLDL values in patients of CKD were higher than controls but this was statistically not significant (P>0.05).

LDL- Significant increase in LDL were found in CKD patients as compared to controls, 122.14±58.18and 102.2±43.39 respectively and this difference was statistically significant (P=0.05).

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Lipid (patients) mg/dl	Male (n=31) Mean ±SD	Female (n=19) Mean±SD	ʻt'	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



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

Table/Graph-4 shows Biochemical (lipid profile) data among male and female CKD patients. On comparing Mean values of TC, TG, HDL, LDL, VLDL values between male and female patients, there was increase in TG, TC, VLDL and decrease in HDL in male patients and there was increase in LDL in female patients. However theses differences were statistically not significant. (P > 0.05).

Table 5: Number of DM and Hypertensionparticipants among CKD group

	YES	NO
Diabetes Mellitus	20(40%)	40(60%)
Hypertension	21(42%)	29(58%)

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: Total cholesterol levels among CKD patients

Total Cholesterol mg/dl	Male (%)	Female (%)	Total (%)
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%)
Total	31	19	50

Table 6 shows difference in total cholesterolvalues 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)

Table 7: Serum Triglycerides levels amongCKD patients

S. Triglycerides mg/dl	Male (%)	Female (%)	Total (%)
<150	15(30%)	13(26%)	28(56%)
Borderline high (150- 199)	07(14%)	00(0%)	07(14%)
High (200-499)	09(18%)	05910%)	14(28%)
Very high >500	00(0%)	01(02%)	01(02%)
Total	31	19	50

Table 8 shows difference in serum TG values among CKD patients. 28 patients (56%) had normal TG values (<150mg/dl), 22 patients (44%) had elevated TG values, among them 07 patients (14%) had TG in borderline high (150-199) range, 14 patients (28%) had TG in high (200-499) range and 01 patient (2%) had TG in very high (>500) range.

Table 8: Serum HDL cholesterol levels amongCKD patients

Serum HDL-C	Male (%)	Female (%)	Total (%)
<40	22(44%)	10(20%)	32(64%)
41-50	09(18%)	08(16%)	17(34%)
51-60	00(0%)	01(02%)	01(02%)
Total	31	19	50

Table 8 shows difference in serum HDL levelsamong CKD patients. 32 patients (64%) had HDL

values <40mg/dl.18 patients (36%) had HDL>40mg/dl

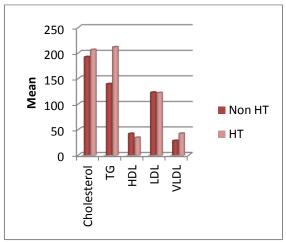
Table 9: Serum LDL cholesterol levels amongCKD patients

S.LDL-c mg/dl	Male (%)	Female (%)	Total (%)
<100	14(28%)	07(14%)	21(42%)
Near optimal (101-129)	04(08%)	04(08%)	08(16%)
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%)
Total	31	19	50

Table 9 shows difference in serum LDL levels among CKD patients. 21 patients (42%) had normal LDL levels (<100) ,08 patients (16%) had near optimal LDL levels (101-129) ,07 patients (14%) had borderline high LDL (130 - 159),14 patients (28%) had elevated LDL, among them 07 patients (14%) had high LDL (160 - 189) and 07 patients (14%) had very high LDL (>190). This was statistically significant (D <0.05)

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.

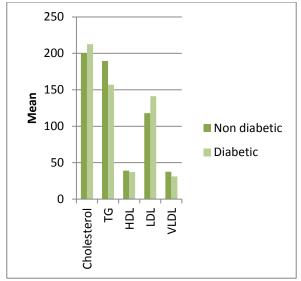


(HT- Hypertensive, Non HT- Non hypertensive)Graph 5 : Lipid profile in hypertensive and non hypertensive CKD patients

The mean values of triglyceride and VLDL in patients of CKD with hypertension were, 211.28 ± 124.61 and 42.04 ± 25.10 respectively which were

higher than patients of CKD without hypertension. HDL was reduced in patients of CKD with hypertension. The p value of triglyceride is 0.013, HDL is 0.008 and VLDL is 0.015 which is statistically significant. The p value of cholesterol is 0.400 and LDL is 0.95 which is insignificant. (Graph 5)

A similar comparison was done with diabetes mellitus as a comorbid condition to see if the glycemic status had a bearing on the lipid profile in the study population analysed. This is shown in the diagram below



Graph 6: Lipid profile in diabetic and Non diabetics

The p value of S.Triglyceride is 0.283, S.Cholesterol is 0.43, S.HDL is 0.49 and S.LDL is 0.116 which is insignificant. (Graph 6)

Discussion

The results of the study on the lipid disorders in patients with chronic kidney disease show that there are alterations in the lipid profiles of these patients as compared to controls.

In a study by Vaziri N D ^[5] 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.

In a study by Abrass et al ^[6] he found that the patients with chronic kidney disease had an elevated serum triglyceride level and decreased clearance of chylomicrons and VLDL.

Attman P.O, Alaupovic P et al^[7] stated that hypertriglyceridemia is the most common plasma lipid abnormality in patients of chronic kidney disease. He found decrease in plasma HDL cholesterol concentration in patients with CKD. It was also reported that decreased HDL was associated with decrease in both the fractional catabolic rate and the total synthetic rate of ApoA₁

and HDL. Another study conducted by P.O. Attman et al ^[7] showed no significant change in levels of total cholesterol in patients of CKD.

Gerald Appel et al ^[8] 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 was 0.094, S.Cholesterol was 0.001, S .HDL was 0.98, LDL was 0.05, and VLDL was 0.17.

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

But the rise in serum triglyceride level in CKD patients compared to control was statistically not significant, may be because of the limitation of

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this study like this study does not include a detailed dietary history and does not compare the caloric intake and the triglyceride levels.

Thomas Quasctining et al ^[9] reported combined hyperlipidemia (elevated total cholesterol and triglycerides) in their study.

Chan C M et al ^[10] 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 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 is 0.50, S.cholesterol is 0.81, S.HDL is 0.49 and S.LDL is 0.26 which was found to be non significant and not in accordance with the lipoprotein abnormalites seen in the study by Chan C M.

In a study by Nayak et al ^[11] 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.

However the elevation in the triglycerides when compared by statistical analysis was found to be insignificant. The statistical values were similar to previous Indian studies by Sharma et al ^[12]. As in these studies, this study showed these lipoprotein fractions were within the statistically normal range.

This could be attributed to low calories derived from carbohydrates and the high intake of polyunsaturated fatty acid 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.

Conclusion

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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Conflicts of Interest: None

References

- Lidner A, Charra B, Sherrard D. Accelerated atherosclerosis in prolonged Maintainance hemodialysis. N Engl J Med 1974;290:697-701.
- Tetsuo Shoji, Eiji Ishimura, Masaaki Inaba, Tsutomu Tabata, yoshiki Nishizawa. Atherogenic Lipoproteins in End-stage renal disease. Am J kidney dis 2001;38:S30-S33
- Foley RN, Parfrey PS, and Sarnak MJ. Clinical epidemiology of cardiovascular disease in chronic renal disease. Am J Kidney Dis 32: S112–S119, 1998
- David CW,Ravinder SC.Oxidation of LDL by mesangial cells may promote glomerular injury. Kidney Int 1994;45: 1628-36
- Vaziri ND, Sato T, Liang K. Molecular mechanisms of altered cholesterol metabolism in focal glomerulosclerosis. Kidney Int 2003;63:1756-63.

- Abrass CK. Cellular lipid metabolism and the role of lipids in progressive renal disease. Am J Nephrol 2004; 24:46-53.
- Attman PO, Samuelsson O, and Alaupovic P. Lipoprotein metabolism and renal failure. *Am J Kidney Dis* 21: 573– 592, 1993
- Gerald appel, Jordan J Cohen, John T Harrington, Cheryl J Zusman. Lipid abnormalities in renal disease. Kidney int 1991;39:169-183
- Thomas Quaschning, Vera Krane, Thomas Metzger, Christoph Wanner. Abnormalities in uremic Lipoprotein Metabolism and it's impact on cardiovascular disease. Am J kidney dis 2001;38:S14-S19
- CM Chan. Hyperlipidemia in chronic kidney disease. Ann AcadMed Singapore 2005;35:31-35
- Nayak KC, Saini MS, Singh VB, Verma SK, Tanwar RS, Charanjeet L. Carotid artery intima - media thickness and its relation with lipid profile in nondiabetic uremic patients. Indian J Nephrol 2006;16:170-3
- Sharma BK, Jindal SK, Rana DS, Gupta B, Kumar M. Absence of hyperlipidemia in patients of chronic renal failure in Chandigarh.Ind.J.Med. Res 1980; 72:461-64.