

A Hospital Based Prospective Study to Evaluate the Serum Lipid Profile of Chronic Kidney Disease (CKD) Patients at District Hospital

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ABSTRACT

Background: Chronic kidney disease (CKD) encompasses group of distinct pathophysiological processes which are associated with abnormal kidney functioning and progressively reducing Glomerular Filtration rate (GFR). The widespread cause of mortality in patients with CKD is a spectrum of cardiovascular diseases. So, the aim of this study to evaluate the serum lipid profile of chronic kidney disease (CKD) patients at district hospital.

Materials & Methods: A prospective cross-sectional study was conducted among the 50 CKD patients admitted in the dept. of General Medicine, Government Hospital, Barmer, Rajasthan, India. Total cholesterol and triglycerides in the plasma were measured enzymatically and then the cholesterol in the supernatant was measured after precipitation of apolipoprotein B (Apo-B) containing lipoprotein to determine the HDL-C. The LDL-C is estimated using Friedewald formula. Kruskal–Wallis chi-square test was used to test the significance of difference between quantitative variables. A p-value less than 0.05 was significant.

Result: In the present study, 50 patients of CKD were included, out of which 34 patients (68%) were male and 16 patients (32%) were females. On decade wise grouping, we found maximum number of patients between 40-50 years (60%). The severity of CKD was positively correlated with total

cholesterol, triglyceride, low density lipoprotein and very low dentistry lipoprotein. High density lipoprotein was significantly negatively correlate with CKD severity.

Conclusion: We concluded that all the lipid abnormalities found in CKD were reduced HDL-C levels in serum along with a significant rise in Serum triglyceride, serum cholesterol serum LDL level and serum VLDL level.

Keywords: CKD, Lipid profile, HDL-C, LDL, Triglyceride, Total cholesterol.

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INTRODUCTION

Chronic kidney disease (CKD) encompasses group of distinct pathophysiological processes which are associated with abnormal kidney functioning and progressively reducing Glomerular Filtration rate (GFR). Various pathological processes in CKD ultimately results in loss of Renal metabolic, excretory, endocrine, and synthetic functions due to accumulation of various protein nitrogenous substances.^{1,2} The incidence of CKD in India, due to the lack of a national registry, is not well defined. However, it has been estimated that the prevalence of CKD in India may be up to 785 people per million populations.3 CKD gradually progresses towards end-stage renal disease (ESRD), generally in association with high cardiovascular morbidity and mortality. In fact, CKD patients are more likely to die of cardiovascular complications than ESRD.4 The prevalence of cardiovascular morbidity in patients of age group 25-34 years with CKD is 500 times that of people without CKD in similar age group and race.3

Hyperlipidemia, an abnormally high level of lipids in blood, is a well-known risk factor for early Atherosclerosis causing various cardiovascular diseases, is frequently seen in patients with CKD. Indian studies demonstrating pathophysiological relationship of CKD with Lipid profile have quoted almost nil Lipid profile abnormalities in CKD to pathophysiologically significant alterations in lipid profile in patients with CKD like high triglycerides and low HDL level. B Shah, S Nair⁵ studied the occurrence of lipid profile abnormalities in CKD and have demonstrated the significant hyper triglyceridemia in patients with CKD.

The patients have reduced HDL-C and increased plasma triglyceride concentrations and there is defect in the cholesterol transport. Other factors that may contribute to atherosclerotic coronary artery disease in ESRD are reduced HDL-C synthesis and reduced activity of the reverse cholesterol pathway. Also, a growing amount of clinical experience data suggests that lipids

may be important in the development and progression of chronic renal disease. Potentially injurious lipid abnormalities are invariably present in these patients more likely to progress to ESRD.

So, the aim of this study was to evaluate the serum lipid profile of chronic kidney disease (CKD) patients at district hospital.

MATERIALS & METHODS

A prospective cross-sectional study was conducted among the 50 CKD patients admitted in the dept. of General Medicine, Government Hospital, Barmer, Rajasthan, India.

Inclusion Criteria

- Patients age more than 20 years
- Patients with known CKD irrespective of the etiology
- Patients on conservative or dialysis treatment for CKD

Exclusion Criteria

- Known cases of dyslipidemia
- Pregnant women
- Patients with acute renal failure and nephrotic syndrome

Methodology: All specimens were analyzed within 4 to 6 hours of collection. Total cholesterol and triglycerides in the plasma were measured enzymatically and then the cholesterol in the supernatant was measured after precipitation of apolipoprotein B (Apo-B) containing lipoprotein to determine the HDL-C. The LDL-C is estimated using Friedewald formula.

Friedewald formula appears to be the most practical and reliable method for determining LDL-C in clinical practice.

LDL-C = Total cholesterol-[HDL-C + (Triglycerides/5)]

Very-low-density lipoprotein (VLDL) is estimated by dividing the plasma triglycerides by 5, reflecting the ratio of cholesterol to triglyceride in VLDL particles. This formula is reasonably accurate if the test resolution is obtained on fasting plasma and if the triglyceride level is less than 350 mg/dL. The accurate determination of LDL-C level in conditions with triglyceride levels greater than this requires application of ultra-centrifugation techniques (Beta quantification).

Statistical Analysis: The data collected regarding all the selected cases were recorded in a master chart. Kruskal–Wallis chi-square test was used to test the significance of difference between quantitative variables. A p-value less than 0.05 was significant.

Table 1: CKD grade in demographic variables

Demographic variables	CKD Grade				P-value
	Grade II (N=1)	Grade III (N=10)	Grade IV (N=33)	Grade V (N=6)	
Age (yrs)					
Mean±SD	63.8±4.78	45.26±9.24	49.67±8.26	52.3±10.34	>0.05
Gender					
Male	1	9	22	2	>0.05
Female	0	1	11	4	

Table 2: CKD grade in co-morbidity

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Co-morbidity	CKD Grade				P-value
	Grade II (N=1)	Grade III (N=10)	Grade IV (N=33)	Grade V (N=6)	_
Diabetes mellitus					
Yes	0	6	23	4	>0.05
No	2	4	10	2	
Hypertension					
Present	1	8	22	4	>0.05
Absent	0	2	11	2	

Table 3: CKD grade in Lipid profile

Lipid profile	CKD Grade				P-value
	Grade II (N=1)	Grade III (N=10)	Grade IV (N=33)	Grade V (N=6)	
Total cholesterol	180.25±16.78	204.56±18.94	239.67±30.26	252.8±35.84	0.001*
Triglyceride	147.5±6.34	168.8±30.26	179.3±46.25	192.7±15.62	>0.05
HDL	51.8±4.76	40.8±8.79	37.24±3.22	30.87±2.88	<0.001*
LDL	133.5±14.59	143.6±18.23	152.60±23.77	164.25±22.34	>0.05
VLDL	15.67±2.28	38.70±8.36	55.42±16.38	62.55±12.90	>0.05

RESULTS

In the present study, 50 patients of CKD were included, out of which 34 patients (68%) were male and 16 patients (32%) were females. On decade wise grouping, we found maximum number of patients between 40-50 years (60%). The mean age was 63.8±4.78 in grade II, 45.26±9.24 yrs in grade III, 49.67±8.26 yrs in grade IV and 52.3±10.34 yrs in grade V of CKD, which was statistical not significant (P>0.05) (table 1). Our study showed that mostly diabetes mellitus & hypertension patients occurred in grade IV CKD (table 2). The severity of CKD was positively correlated with total cholesterol, triglyceride, low density lipoprotein and very

low dentistry lipoprotein. High density lipoprotein was significantly negatively correlated with CKD severity (table 3).

DISCUSSION

Chronic kidney disease results when a disease process affects the structural or functional integrity of the kidneys. Cardiovascular disease is a major cause of mortality in patients with mild-to-moderate CKD and ESRD. Dyslipidemia has been established as a well-known traditional risk factor for cardiovascular disease in general population and it is well known that patient with CKD exhibits significant alterations in lipoprotein metabolism, which, in

their most advanced form, may result in the development of severe dyslipidemia. The low HDL levels in patients with chronic kidney disease in our study were in match with Diana M Lee LG et al⁶ who studied the abnormalities of lipid profile in CRF patients. This low HDL cholesterol levels were also an isolated independent risk factor for the development of CKD in the Framingham spring study. Several pathological processes may underlie the reductions in HDL cholesterol levels, which is usually an indication of dysfunctional reverse cholesterol transport. Apo AI, which is the activator of lecithin cholesterol acyltransferase (LACT), is decreased in CKD due to inverse regulation of hepatic Apo AI genes causing a decline in the function of LACT, which leads to decreased cholesterol esterification and abnormality in HDL maturation. The activity of LACT is persistently decreased in CKD, so there is reduced HDL level.

This was in concordance with the results obtained by Bhagwat et al where they found HDL-C to be significantly low (20±11) mg/dL (p-value less than 0.001) in CRF groups.7 Patients with CKD generally have reduced plasma HDL-C concentrations when compared with nonuremic individuals. Elevated triglyceride levels are implicated to impaired activity lipoprotein lipase (LPL)8 and direct inhibitory action of various uremic 'toxins' on the enzymes involved in lipid metabolism9 pinpointing the most important pathophysiological mechanisms causing the development of hypertriglyceridemia in renal failure. Chan MK et al¹⁰ also showed hypertriglyceridemia was the major abnormality in their studies. Hypertriglyceridemia may represent an early feature of renal failure. In another study done by Bhagwat et al,7 they concluded that CRF patients were having marked triglyceridemia of 232 mg/dL as compared with controls (p-value < 0.01). Another Indian study on dyslipidemia in patients with CRF and renal transplantation by Shah et al5 demonstrated that triglycerides level was elevated significantly in CRF patients on conservative management. These results show that hypertriglyceridemia is an important lipid abnormality in patients with CRF.

Most studies point out that Uraemic Patients commonly have normal to slightly decreased concentrations of LDL-C levels, and they exhibit significant disturbance in the density distribution of LDL sub fraction that is characterized by presence of predominantly small dense LDL particles. ¹¹ In the present study, we find significantly high levels of LDL cholesterol in the group with CKD stages IV & V.

Attman P.O, Alaupovic P et al12 stated that hypertriglyceridemia is the most common plasma lipid abnormality in patients of chronic kidney disease. He found a 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. P.O. Attman et al¹² showed no significant change in levels of total cholesterol in patients of CKD. Another study conducted by Gerald Appel et al13 showed an increase in very low-density lipoproteins (VLDL). In a study by Nayak et al14 and colleagues they found that the lipid profile in diabetic and nondiabetic 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.

CONCLUSION

We concluded that all the lipid abnormalities found in CKD were reduced HDL-C levels in serum along with a significant rise in Serum triglyceride, serum cholesterol serum LDL level and serum VLDL level.

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