Original Research

Serum lipid profile in chronic kidney disease patients

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ABSTRACT:
Background: The illness known as chronic kidney disease (CKD) is characterized by a gradual decline in renal function over time. The present study was conducted to assess the serum lipid profile in chronic kidney disease patients. **Materials & Methods:**54 chronic kidney disease patients of both genders were selected. Group I had CKD patients and group II had healthy subjects. Assessment of blood urea, serum creatinine, fasting blood sugar, post prandial blood sugar, serum total protein, serum albumin, serum globulin, lipid profile: total cholesterol, triglycerides, HDLc, LDLc, VLDLc were done. **Results:** Out of 54 patients, there were 30 males and 24 female patients. The mean total cholesterol was 136.2 mg/dl in group I and 151.2 mg/dl in group II, TG was 158.4 mg/dl in group I and 104.4 mg/dl in group II, HDLc was 38.5 mg/dl in group I and 52.3 mg/dl in group II, LDLc was 68.1 mg/dl in group I and 84.4 mg/dl in group II, VLDLc was 39.4 mg/dl in group I and 25.4 mg/dl in group II. The mean blood urea was 130.1 mg/dl in group I and 32.7 mg/dl in group II. The mean serum creatinine was 7.5 mg/dl in group I and 0.98 mg/dl in group II. The difference was significant (P< 0.05). **Conclusion:** Patients with chronic kidney disease (CKD) are more likely to experience cardiovascular issues due to elevated triglycerides, elevated VLDL, and decreased HDL.
Key words: Chronic kidney disease, cardiovascular disease, renal tissues

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INTRODUCTION

The illness known as chronic kidney disease (CKD) is characterized by a gradual decline in renal function over time. Because of the less efficient functioning of the renal tissues, CKD causes a decline in renal function.[1] One of the main causes of morbidity and death in people with chronic kidney disease is cardiovascular disease. The majority of CKD patients pass away from problems with their cardiovascular systems before they ever reach stage 5. Since dyslipidemia is a significant risk factor for coronary heart disease, there is interest in identifying and treating abnormalities in lipoproteins and plasma lipids.[2] Higher triglycerides (TG), higher total cholesterol (TC), lowered high density lipoprotein cholesterol (HDL-C), and elevated high density lipoprotein cholesterol (LDL-C) are the hallmarks of dyslipidemia in individuals with chronic kidney disease (CKD).[3]

However, total cholesterol may be lower or normal, particularly when starvation is present. Patients with chronic kidney disease (CKD) have a dyslipidaemia pattern that is extremely atherogenic and linked to the development of atherosclerotic cardiovascular disease as well as all cause death.[4] Since dyslipidemia is a modifiable cardiovascular risk factor, reducing the risk of cardiovascular disease and its development to end stage renal disease (ESRD) can be achieved through early identification and care with lipid-lowering drugs as well as lifestyle modifications. It has been shown that treating dyslipidemia with statins slows down the rate at which glomerular filtration rate (GFR) declines in people with chronic kidney disease (CKD).[5]The present study was conducted to assess the serum lipidprofile in chronic kidney disease patients.

MATERIALS & METHODS

The present study comprised of 54chronic kidney disease patients of both genders. The consent was obtained from all patients.
Data such as name, age, gender etc. was recorded. Group I had CKD patients and group II had healthy subjects. Assessment of blood urea, serum creatinine, fasting blood sugar, post prandial blood sugar, serum total protein, serum albumin, serum globulin, lipid profile: total cholesterol, triglycerides, HDLc, LDLc, VLDLc were done by using semiautoanalyzer. Data

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thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS
Table I Distribution of patients

Total- 54		
Gender	Males	Females
Number	30	24

Table I shows thatout of 54 patients, there were 30 males and 24 female patients.

Table II Assessment of parameters

Parameters	Variables	Group I	Group II	P value
Lipid profile	Total cholesterol	136.2	151.2	0.05
	TG	158.4	104.5	0.01
	HDLc	38.5	52.3	0.02
	LDLc	68.1	84.4	0.04
	VLDLc	39.4	25.6	0.02
RFT	Blood urea	130.1	32.7	0.001
	Serum creatinine	7.5	0.98	0.01

Table II shows that mean total cholesterol was 136.2 mg/dl in group I and 151.2 mg/dl in group II, TG was 158.4 mg/dl in group I and 104.4 mg/dl in group II, HDLc was 38.5 mg/dl in group I and 52.3 mg/dl in group II, LDLcwas 68.1 mg/dl in group I and 84.4 mg/dl in group II, VLDLc was 39.4 mg/dl in group I and 25.4 mg/dl in group II. The mean blood urea was 130.1 mg/dl in group I and 32.7 mg/dl in group II. The mean serum creatinine was 7.5 mg/dl in group I and 0.98 mg/dl in group II. The difference was significant (P< 0.05).

DISCUSSION
The primary cause of hospitalization and death for those with chronic renal disease is cardiovascular disease.[6,7]When renal replacement treatment (RRT) was initiated, the severity of cardiovascular illness suggests that the disease began early in the course of CKD. One of the established traditional cardiovascular risk factors in both the general population and CKD patients is dyslipidemia.[8] This cardiovascular risk factor is frequently seen in CKD patients. In patients with chronic kidney disease (CKD), dyslipidemia is linked to a quick deterioration in renal function and the start of RRT.[9] Although the exact process is uncertain, it has been hypothesized that mesangial cells bind to and absorb oxidized low-density lipoprotein (LDL), which subsequently damages mesangial, epithelial, and endothelial cells by encouraging the migration of inflammatory cells like macrophages that produce chemokines and cytokines.[10] This subsequently leads to glomerulosclerosis. Hypercholesterolaemia and hypertriglyceridaemia also cause podocyte injury and mesangial sclerosis, subsequently leading to glomerulosclerosis.[11,12]The present study was conducted to assess the serum lipidprofile in chronic kidney disease patients.

We found that out of 54 patients, there were 30 males and 24 female patients. Phukan et al[13] assessed the pattern of changes in the serum lipid profile in patients with chronic kidney disease (CKD) receiving conservative treatment as well as those who were hemodialyzed. The study comprised 51 seemingly healthy controls and 71 randomly chosen CKD patients who attended an Assamese tertiary care hospital over the course of a year (31 receiving conservative treatment and 40 receiving hemodialysate). All of the patients and controls had their serum lipid profiles, urea creatinine, FBS, PPBS, total protein, and albumin tested. The patient had marginally elevated levels of very low-density lipoprotein (VLDL) of CKD group 31.58±12.36, controls 19.39±7.50, and significantly reduced levels of high-density lipoprotein (HDL) of CKD group 33.40±9.06, controls 45.95±10.35. Triglyceride level (TGL) of CKD group 157.88±61.82, controls 96.98±37.52. The patient group had lower total cholesterol (CKD group: 128.2±53.57, controls: 142.53±31.44) and lower LDL (CKD group: 63.23±46.47, controls: 77.35±26.81), but the differences were not statistically significant (p values of 0.09 and 0.059, respectively). Between the hemodialyzed and conservatively treated CKD groups, there was no statistically significant difference in the lipid profile, and there was also no variation in the lipid profile associated to gender.

We found that the mean total cholesterol was 136.2 mg/dl in group I and 151.2 mg/dl in group II, TG was 158.4 mg/dl in group I and 104.4 mg/dl in group II, HDLc was 38.5 mg/dl in group I and 52.3 mg/dl in group II, LDLc was 68.1 mg/dl in group I and 84.4 mg/dl in group II, VLDLc was 39.4 mg/dl in group I and 25.4 mg/dl in group II. The mean blood urea was 130.1 mg/dl in group I and 32.7 mg/dl in group II. The mean serum creatinine was 7.5 mg/dl in group I and 0.98 mg/dl in group II.The prevalence and pattern of dyslipidemia in CKD patients prior to dialysis were ascertained by Adejumo et al.[14] A total of 105 age and sex matched control volunteers and 105 consecutive pre-dialysis CKD patients recruited over a two-year period were included. Participants provided information on their demographics, BMI, and CKD etiology. For the purpose of measuring creatinine and

fasting serum lipids, blood was drawn. With a male to female ratio of 1.7:1, the mean age of the CKD and control participants was 46.98±16.81 and 47.57±15.97 years, respectively. While mean high density lipoprotein-cholesterol (HDL-C) was significantly lower in CKD patients (p=<0.001), the median atherogenic index of plasma (AIP), low density lipoprotein-cholesterol, and triglyceride (TG) were significantly higher in these individuals. 60% of CKD patients had dyslipidemia overall, which was substantially greater than the control group's 39% prevalence (p=0.002). With declining renal function came an increase in the incidence of high AIP, elevated TG, and decreased HDL-C. Patients with CKD who were female (p=0.02) and older (p=0.94) had higher rates of dyslipidemia.

The shortcoming of this study is small sample size.

CONCLUSION

Authors found that patients with chronic kidney disease (CKD) are more likely to experience cardiovascular issues due to elevated triglycerides, elevated VLDL, and decreased HDL.

REFERENCES

1. Balode AA, Khan ZH. Serum lipid profile in chronic kidney disease patients on haemodialysis. Indian Journal of Applied Research. 2011;3(8):20–22.
2. Sumathi ME, Tembad MM, Jayaprakash Murthy DS, Preethi BP. Study of lipid profile and oxidative stress in chronic renal failure. Biomedical Research. 2010;21:451-56.
3. Mshelia DS, Buratai LB, Mamza YP. Lipid profile in pre dialysis chronic kidney disease patients attending University of Maiduguri Teaching Hospital, MaiduguriNigeria. Niger J Clin Pract. 2009;12(2):173-78.
4. Chijioke NN, Bartimaeus EAS, Okeke CU. Lipid profile in chronic renal failure patients on dialysis. European Journal of Cardiovascular Medicine, Original Research. 2012;2(2):106-09.
5. Miller M. Dyslipidemia and cardiovascular risk: The importance of early prevention. QJM. 2009;102(9):657–67.
6. Burtis CA, Ashwood ER, Bruns DE, Border BG, Sawyer B, Tietz NW. Tietz fundamentals of clinical chemistry and molecular diagnostics. 6th ed. United States: Elsevier Health Sciences; 2014 May 28. ISBN: 9780721638652.
7. Trevisan R. Lipids and renal disease. Journal of the American Society of Nephrology. 2006;17(4_suppl_2): 145–47.
8. Issa V, Andrade L, Bocchi E. Current strategies for preventing renal dysfunction in patients with heart failure: A heart failure stage approach. Clinics. 2013;68(3):401– 09.
9. Tsimihodimos V. Dyslipidemia associated with chronic kidney disease. The Open Cardiovascular Medicine Journal. 2011;5(1):41–48.
10. Ganta V, Yalamanchi RP, Mahanta KC, Sahu B, Raghvendar K, Anusha G, et al. A study of lipid profile in non- diabetic chronic kidney disease. International Journal of Advances in Medicine. 2016:965–70.
11. Vaziri ND. Causes of dysregulation of lipid metabolism in chronic renal failure. Seminars in Dialysis. 2009;22(6):644–51.
12. Rao AM, Bitla AR, Reddy EP, Sivakumar V, Srinivasa Rao PVLN. Lipid abnormalities, lipoprotein (a) and apoprotein pattern in non- dialyzed patients with chronic kidney disease. Indian Journal of Clinical Biochemistry. 2010;25(1):47– 50.
13. Phukan RR, Goswami RK. Unusual dyslipidemia in patients with chronic kidney diseases. Journal of Clinical and Diagnostic Research: JCDR. 2017 Jan;11(1):01.
14. Adejumo OA, Okaka EI, Ojogwu LI. Lipid profile in pre-dialysis chronic kidney disease patients in southern Nigeria. Ghana medical journal. 2016 Apr 7;50(1):44-9.