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Assessment of Lipid Profile in Chronic Kidney Disease Patients in a Tertiary Care Hospital Settings: A Prospective Study

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ABSTRACT

Introduction: Cardiovascular disease (CVD) is the leading cause of mortality in chronic kidney disease (CKD) patients as it is characterized by specific metabolic abnormalities of plasma lipids both qualitatively and quantitatively. Therefore, early determination and management of the risk factors for CVD in CKD patients play pivotal role to develop more effective screening and treatment strategies. Henceforth, the present study was conducted to assess the lipid profile in chronic kidney disease patients in tertiary care hospital setting.

Materials and Methods: The present prospective and observational study comprises of cases of chronic kidney disease patients admitted in a tertiary care centre. All patients were investigated for routine laboratory investigations, serum electrolytes, serum lipid profile, thyroid profile, ultrasonography of kidney size and cortico-medullary differentiation, diabetic profile, and other investigations as and when required. The statistical operations were done through SPSS (Statistical Presentation System Software) and Graph pad for Windows.

Results: The number of patients in stage 3 and stage 4 were more as compared to stage 5 population. The data suggests that there is high level of serum triglyceride level along with the low level of the serum HDL cholesterol in all the stages of CKD patients. There was rise in triglyceride level and VLDL level in all the stages of CKD. While there is decrease in the HDL level in all the stages of the CKD. There was mildly reduction or near normal level of serum cholesterol and LDL cholesterol in the present study.

Conclusion: There is significant alteration in lipid profile in CKD patients characterized by higher level of triglyceride and VLDL level, with substantial lower level of serum HDL level. Total cholesterol and LDL cholesterol show minimal to low alteration. Such abnormalities in lipid profile leads to the more prevalence of atherosclerosis in CKD patients and increase risks of cardiovascular complications.

Keywords: Cardiac Disorders, Comorbidities, Renal Disease.

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INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of mortality in chronic kidney disease (CKD) patients.[1] The Chronic Kidney Disease (CKD) is characterized by specific metabolic abnormalities of plasma lipids both qualitatively and quantitatively.[2] 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.[3] This may be a significant risk factor for vascular complications leading to increased morbidity and mortality in CKD patients. Dyslipidemia in CKD is one of the mechanism for the increased prevalence of the atherosclerosis. Data from the population-based Atherosclerosis Risk in Communities (ARIC) Study showed that risk factors for CHD in the general population also are associated with an increased risk for CHD among the population with CKD.[4]

Although in patients with CKD total and LDL cholesterol concentrations are usually within the target range or even lower, the serum concentrations of lipid subfractions may not fully reflect the CV risk attributed to lipid abnormalities. Compared to individuals with normal renal function, patients with CKD usually are characterized by a greater proportion of oxidized LDL particles, which are recognized by scavenger receptors and induce formation of foam cells in atherosclerotic plaques.[5,6] Furthermore, LDL particles in CKD patients tend to be smaller and denser, and, therefore, more atherogenic.[6,7]

Therefore, early determination and management of the risk factors for CVD in CKD patients play an important role to develop more

effective screening and treatment strategies to decrease cardiovascular mortality and morbidity in CKD patients.[1] Henceforth, the present study was conducted to assess the lipid profile in chronic kidney disease patients in tertiary care hospital setting.

MATERIALS AND METHODS

The present prospective and observational study comprises of cases of chronic kidney disease patients admitted in a tertiary care centre. Patients selected for the present study were clinically and laboratory proven cases of chronic kidney disease and were included in the study.

Inclusion criteria comprised of patients who were known case of chronic kidney disease with a diagnosed etiology, patient's whose diabetic and thyroid profile was normal and patient who were not on any hypolipidemic drugs. Exclusion criteria consisted of patients having age less than 12 years, patients having known case of diabetes mellitus, patients with hypothyroidism and patients having renal transplantation. All patients were studied in detail for history, clinical examination at the time of presentation, course of illness during hospital stay and at the time of discharge, associated risk factors and medical illness and on regular follow up.

All patients were investigated for routine laboratory investigations, serum electrolytes, serum lipid profile, thyroid profile, ultrasonography of kidney size and cortico-medullary differentiation, diabetic profile, and other investigations as and when required.

For this study, HDL was measured by accelerator selective detergent method. Urea was measured by urease method. Serum cholesterol was measured by enzymatic method. Serum triglyceride was measured by Glycerol Phosphate Oxidase member method. The main method for measurement of all these parameters were spectrophotometry. The commercial kit for measurement of all these parameters used was Architect c-8000 machine. The statistical operations were done through SPSS (Statistical Presentation System Software) and Graph pad for Windows.

Table 1: CKD stage wise distribution of population

CKD stage	Frequency	Percentage
3	18	36
4	18	36
5	14	28

Table 2: Year wise distribution of morbidity of population

Years of suffering from disease	Frequency	Percentage
Up to 1 year	8	16
>1 year to 2 year	18	36
>2 year to 3 year	13	26
>3 year	11	22

Table 3: The distribution of lipid profile values according to the CKD stage types in their mean and standard deviation.

	Total cholesterol	HDL	VLDL	LDL	Triglyceride
Stage 3	170+/- 16.18	31.89+/- 3.51	45.38+/- 13.11	81.27+/- 11.59	156.50+/- 11.61
Stage 4	165.11+/- 20.25	27.50+/- 5.35	44.63+/- 15.99	77.22+/- 20.29	184.79+/- 65.37
Stage 5	176.46+/- 14.81	20.46+/-5.15	44.15+/-9.76	85.85+/- 32.60	223+/- 81.35

Table 4: The values of lipid profile in population with HD and without HD.

	Total cholesterol	HDL	VLDL	LDL	Triglyceride
Without hemodialysis	169.19+/-18.44	27.74+/-5.97	44.42+/-13.35	79.95+/-21.15	182.88+/-60.51
With hemodialysis	180+/-15.62	23.29+/-8.4	46.43+/-12.42	91.71+/-25.36	201.86+/-69.98

(The values are in the Mean+/-SD in this table.)

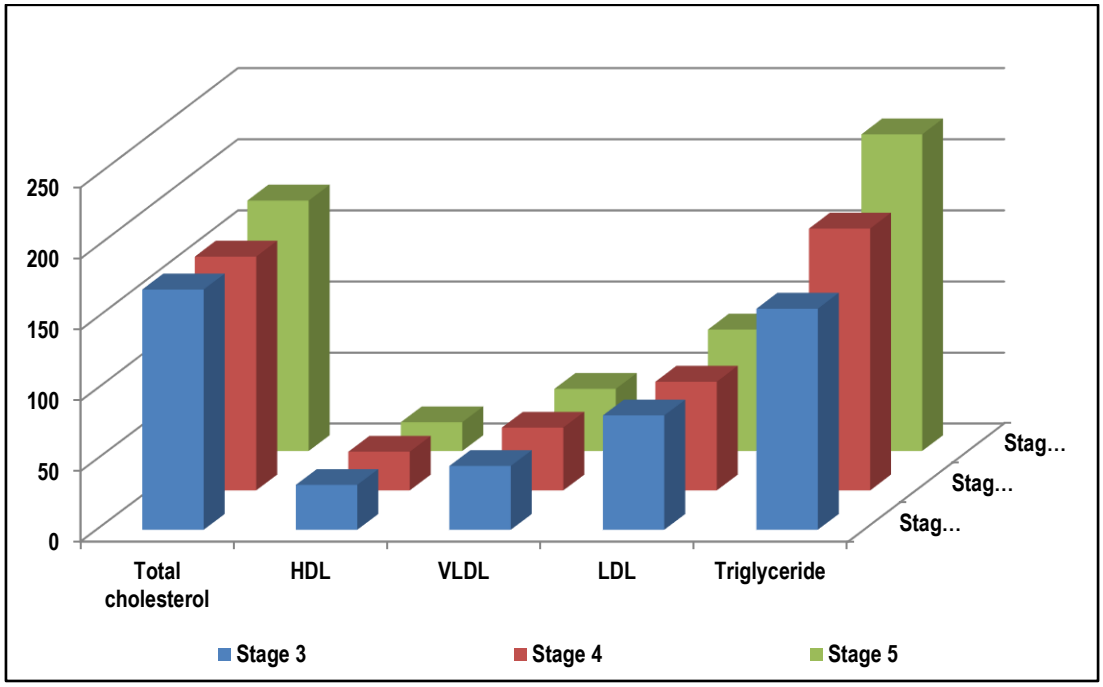
RESULTS

The patients included in our study were suffering from various stages of chronic kidney disease, mainly stage 3, stage 4, and stage 5 (table 1). The number of patients in stage 3 and stage 4 were more as compared to stage 5 population.

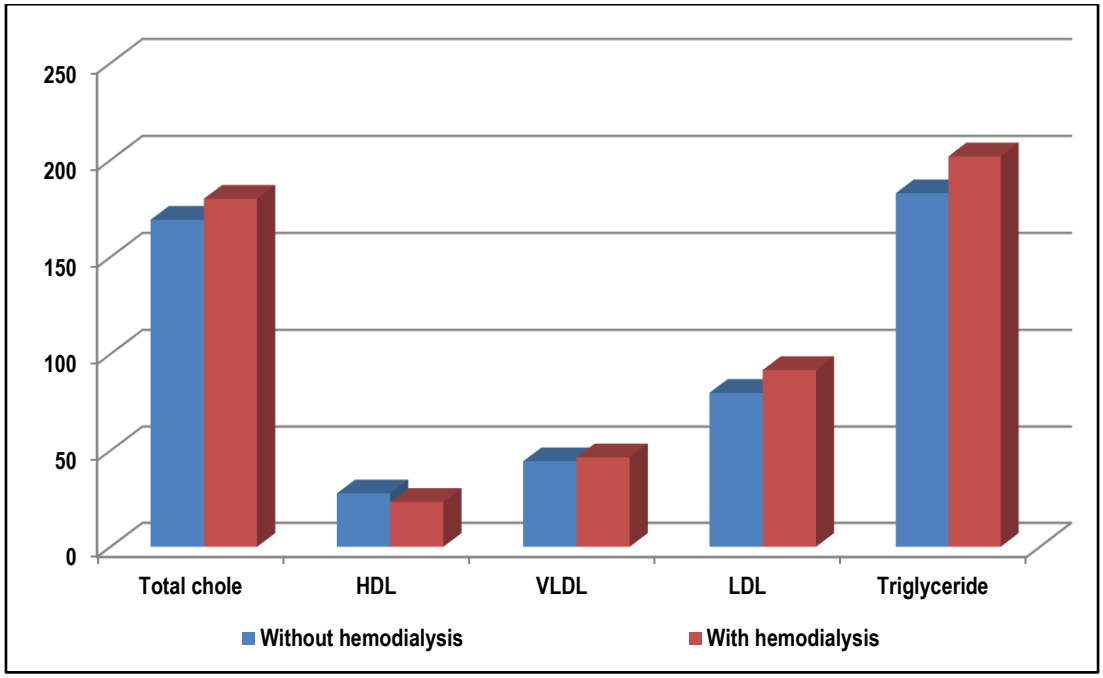
There were patients of chronic kidney disease suffering from variable periods of duration (table 2). The population having more patients suffering more than one year of disease and are on regular treatment.

There were 7 patients in our study who were on maintenance hemodialysis. And remaining were on conservative regular treatment, not on maintenance hemodialysis. According to MDRD formula, eGFR was calculated in all the subjects in our study and according to eGFR, we had distributed the patients in clinical CKD stage 3, 4, and 5. The data suggests that there is high level of serum triglyceride level along with the low level of the serum HDL cholesterol in all the stages of CKD patients (table 3, graph 1).

Thus, in this study, it was very clear that, there was rise in triglyceride level and VLDL level in all the stages of CKD. While there is decrease in the HDL level in all the stages of the CKD. There is mildly reduction or near normal level of serum cholesterol and LDL cholesterol in the present study. In addition to this, we have collected the data in maintenance hemodialysis patients, which also suggests the same picture of the lipid profile as in non-maintenance hemodialysis patients (table 4, graph 2).



Graph 1: CKD stage wise distribution of lipid profile.



Graph 2: The comparative graph between the lipid profile values in patients on HD and without HD.

DISCUSSION

In our present study, dyslipidemia was observed in CKD patients. There was a statistically significant increase of serum triglycerides and VLDL in almost all the patients. There was a substantial decrease in serum HDL-C in both non dialysis and hemodialysis groups when compared with the controls. There was no hypercholesterolemia and serum LDL-C was not significantly altered in CKD patients both in non-dialysis and hemodialysis groups when compared with controls.

Chronic kidney disease (CKD) shares a common appearance of glomerulosclerosis, vascular sclerosis and tubulointerstitial fibrosis, suggesting a common final pathway of progressive injury.[8]

In the endogenous pathway, the liver assembles and secretes triglyceride-rich very low-density lipoprotein (VLDL) particles, which transport triglycerides from the liver to peripheral tissues. After hydrolysis of the triglycerides by LPL, the VLDL particles are

reduced to intermediate-density lipoproteins (IDL), which can be taken up by the liver or can be further hydrolyzed to LDL particles. During this conversion, the particles become depleted of triglycerides but retain considerable amounts of cholesterol.[9] LDL transports cholesterol primarily to hepatocytes but also to peripheral tissues. ApoB-100 is responsible for the recognition and uptake of LDL by the LDL receptor, which clears approximately 60 to 80% of LDL in normal individuals. The remaining LDL is removed by other specific receptors, such as LRP, or by scavenger receptors. Oxidized LDL (ox-LDL) in particular can be taken up by scavenger receptors on macrophages and vascular smooth muscle cells. When these macrophages become overloaded with cholesteryl esters, they transform into foam cells, which is a major step in the development of atherosclerosis.[10] When LDL becomes lipid depleted, small dense LDL (sdLDL) is generated, which has lower affinity for the LDL receptor but is more susceptible to oxidative modification. Thus, sdLDL are believed to be more atherogenic than larger LDL particles.[11]

High-density lipoprotein (HDL) plays an important role in reverse cholesterol transport, which shuttles cholesterol from peripheral cells to the liver,[12] an important step that relieves the peripheral cells from cholesterol burden. HDL precursor particles are secreted as disc-shaped structures by the liver and intestine and can absorb free cholesterol from cell membranes, a process that is mediated by ATP binding cassette transporter 1, apoA-I, and apoA-IV. ApoA-I is the major apolipoprotein of HDL and activates the enzyme lecithin:cholesterol acyltransferase (LCAT), which esterifies the accepted free cholesterol to allow more efficient packaging of the cholesterol for transport. By acquisition of additional apolipoproteins, cholesteryl esters, and triglycerides, HDL_3 particles are transformed into larger HDL_2 particles.[13]

The present study clearly correlates with the results of the study done by Raju DS et al[14] (table 5).

Table 5: The comparison table between the present study and the study done by Raju DS et al[14]

Lipid profile	Present study	Raju et al
Total cholesterol	176.46+/-14.81	182.46 ± 22.12
Triglycerides	223.0+/-81.35	209.80 ± 32.4
HDL	20.46+/-5.15	35.28 ± 5.67
LDL	85.85+/-32.60	105.22 ± 26.15
VLDL	40.0+/-9.76	41.96 ± 6.48

Values used in this table are in the Mean +/- SD form.

Thus, after the present study, we can understand that in chronic kidney disease patients, there is elevation in Serum Triglyceride level, along with elevation of serum VLDL level. Thus, in this study, it is very clear that, there is rise in triglyceride level and VLDL level in all the stages of CKD. While there is decrease in the HDL level in all the stages of the CKD. There is mildly reduction or near normal level of serum cholesterol and LDL cholesterol in the present study.

The data suggests that there is high level of serum triglyceride level along with the low level of the serum HDL cholesterol in all the stages of CKD patient. This data showed (table 3,4) the more worsening picture of the lipid profile in hemodialysis patients. A number of qualitative changes in individual parameters of lipid profile have been determined. These patients have elevated levels of small dense and oxidized LDL and Lp(a), whereas LDL cholesterol levels are within normal limits. Many studies also show the enhanced level of the oxidative stress and compromised antioxidant stress in hemodialysis patients leading to alteration in lipid profile level and all these factors contribute to the cardiovascular risk factor enhancement in CKD patients.[15,16]

In the present study, the HDL level is dramatically low in all the stages of the chronic kidney disease. While there is minimal to low alteration in the serum LDL and total cholesterol level in chronic kidney disease patients. The accompanying serum lipid alteration i.e. hypertriglyceridemia, increased serum VLDL and decreased serum HDL in CKD enhance the risk of atherosclerosis and favours higher incidence of cardiovascular complications. Therefore, lipid regulation must be instituted to decrease the risk of complications in CKD patients.

CONCLUSION

There is significant alteration in lipid profile in CKD patients characterized by higher level of triglyceride and VLDL level, with substantial lower level of serum HDL level. Total cholesterol and LDL cholesterol show minimal to low alteration. Such abnormalities in lipid profile leads to the more prevalence of atherosclerosis in CKD patients and increase risks of cardiovascular complications.

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