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EVALUATION OF LIPID PROFILE IN PATIENTS OF CHRONIC KIDNEY DISEASE

General Medicine

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ABSTRACT

Introduction: Chronic kidney disease (CKD) is one of the common serious health problem associated with increased morbidity and mortality, especially in developed countries. Progression of CKD is associated with having a number of complications, including thyroid dysfunction, dyslipidemia and cardiovascular diseases. This study was conducted to investigate lipid profile in CKD patients.

Methods: The present study was a prospective observational study conducted on 150 adult patients of CKD, 50 each from stage 3, 4, and 5 on regular follow up of kidney and dialysis clinic. Demographic features and medical history of diabetes mellitus, hypertension and cardiovascular diseases of each patient were noted, and blood samples were analyzed for serum urea, creatinine, total cholesterol, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol and triglyceride.

Results: In our study hypertriglyceridemia was found in 37%, hypercholesterolemia in 36%, low HDL in 36%, high LDL in 36%, and high VLDL in 36% of the patients. There was a significant rise in LDL and VLDL, and significant decrease in HDL from CKD stage 3 to 5.

Conclusions: CKD patients have increased risk of CVD and higher prevalence of dyslipidemia as compared to the general population. The majority of patients die because of cardiovascular related moratlity before even reaching to the ESRD. Therefore it is necessary to identify dyslipidemia in early stages to prevent the further progression of kidney dysfunction, leading to better quality of life and improved morbidity and mortality. So, regular checkup of lipid profile is recommended in patients with CKD.

KEYWORDS

Chronic kidney disease, Dyslipidemia, cardiovascular diseases.

INTRODUCTION

Chronic kidney disease (CKD) is one of the common serious health problem associated with increased morbidity and mortality, especially in developed countries. The major cause of mortality in CKD is increased incidence of cardiovascular diasese.[1] The risk of cardiovascular disease (CVD) related mortality increases with decline in the renal function.[2] CVD and CKD are interlinked conditions because each condition increases the risk of other condition and most of the common risk factors such as age, smoking, hypertension, diabetes mellitus and dyslipidemia are shared by them. CKD patients have similar or higher risk of coronary events as seen in the other high risk individuals of diabetes, metabolic syndrome, cigarette smoking. Most of the patients with CKD die from cardiovascular system related complications before even reaching Stage 5 CKD.[3,4]

Dyslipidemia is a disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency. Dyslipidemia is characterized by elevation of the total cholesterol, low-density lipoprotein (LDL) cholesterol and the triglyceride concentrations, and a decrease in the high-density lipoprotein (HDL) cholesterol concentration in the blood. Dyslipidemia has been known to increase the cardiovascular risk in CKD patients with an associated increase in mortality.[5] Various studies have shown that total and LDL cholesterols are the most independent predictors of mortality.[5] It has been suggested that various lipid abnormalities are associated with increase in the risk of development and progression of renal disease in diabetic as well as in non diabetic patients. Experimental studies have shown that lipids are associated with glomerular and tubulointerstitial injury resulting in renal disease progression. Dyslipidemia may cause damage to glomerular capillary endothelial and mesangial cells as well as podocytes. Hypercholesterolemia and hypertriglyceridemia are also associated with mesangial sclerosis secondary to podocyte injury.[6] Decline in renal function is associated with specific alteration in the lipid proile.[7] There is reduction in the activity of lipoprotein lipase and hepatic triglyceride lipase in CKD leading to decreased uptake of triglyceride rich, apolipoprotein (Apo B) containing lipoprotein's by the liver and in peripheral tissues, yielding increased circulation of these atherogenic lipoproteins.[8] Thus CKD patients are at increased risk of CVD. So it is better to screen these patients for dyslipidemia to prevent the further disease progression and the CVD related mortality. Thus present study was done to emphasise the importance of dyslipidemia in CKD.

MATERIAL AND METHODS

The present study was a prospective observational study conducted on 150 adult patients of CKD, 50 each from stage 3, 4, and 5. Patients were on regular follow up of Kidney and Dialysis clinic of Pt. B.D. Sharma PGIMS, Rohtak. After taking written informed consent and a thorough history, each participant had undergone detailed clinical, biochemical and radiological examination to establish the stage of CKD. All the patients were assessed for lipid profile. Patients on lipid lowering agents were excluded. The study was approved by ethical committee of Pt. B.D. Sharma University of Health Sciences. The patients were divided into three groups: Group A, B and C based on CKD staging.

Group A consisted of 50 patients with eGFR (estimated glomerular filtration rate) between 30-59 ml/min/1.73m^2 (CKD Stage III).

Group B consisted of 50 patients with eGFR between 15-29 ml/min/1.73m^2 (CKD Stage IV)

Group C consisted of 50 patients with of eGFR < 15 ml/min/1.73m^2, not on Haemodialysis (HD) (CKD stage V)

Morning blood samples were taken after an overnight fasting for generation of plasma and serum for biochemical parameters analysis. Blood hemoglobin, blood urea, random blood sugar, serum creatinine, uric acid, sodium, potassium, calcium, phosphate, total protein and creatinine, fasting lipid profile (Triglycerides, Total Cholesterol, HDL, LDL and VLDL) were analyzed using certified methods at the department of biochemistry at PGIMS, Rohtak. Creatinine clearance was calculated using MDRD formulae. Apolipoprotein-B (apo-B) lipoprotein was measured by immunoturbidometry method.

STATISTICALANALYSIS

At the end of the study, the data was expressed as mean±1SD or range. Probability values of <0.05 were considered to be significant in all the analyses. ANOVA test was used to analyze differences in quantitative variables between the groups. The correlations were tested using Pearson correlation coefficient analysis. All statistical calculations were carried out using SPSS 21.0 software.

RESULTS

Study group comprised of 150 cases of chronic kidney disease. Out of total 150 patients, 94 were male and 56 were female. Majority of patients (84.67%) were above 40 years of age and were equally distributed in all groups. The mean age of study population was 52 years, ranging from 18 to 75 years. The most common cause of CKD in all groups was diabetes mellitus (38%) followed by hypertension (23.33%) and chronic glomerulonephritis (14%). General characteristics of the studied population are summarized in Table-1. Various biochemical parameter and lipid profile as shown in Table -2.

In our study hypertriglycerdemia was found in 37%, hypercholestrolemia in 36%, low HDL in 36%, high LDL in 36%, and high VLDL in 36% of the patients. There was a significant rise in LDL and VLDL, and significant decrease in HDL from CKD stage 3 to 5. Significant positive correlation of dyslipidemia was seen with hsCRP and serum apolipoprotein-B, and significant negative correlation was seen with serum albumin as shown in Table -3.

On performing multivariate linear regression, after adjusting for confounding factors, serum apolipoprotein-B was significantly affecting HDL, LDL, VLDL and cholesterol. With the increase in serum apolipoprotein-B by 1 unit, HDL significantly decreases by 11.902 units, LDL significantly increases by 18.042 units, VLDL by 17.3 units and cholesterol by 36.559 units.

On performing multivariate linear regression, after adjusting for confounding factors, hs-CRP, serum apolipoprotein-B and serum albumin was significantly affecting triglyceride. With the increase in hs-CRP by 1 unit, triglyceride significantly increases by 5.784 units, with the increase in serum apolipoprotein-B by 1 unit, triglyceride significantly increases by 40.997 units, with the increase in serum albumin by 1 unit, triglyceride significantly decreases by 28.994 units.

DISCUSSION

Dyslipidemia has been well known to increase risk for CVD development. As compared to general population, the risk of developing CVD is more in patients of CKD due to various risk factors associated with CKD and dyslipidemia being a major one amongst them.[9] The prevalence of dyslipidemia and thus CVD increases with deterioration in kidney function and is a major cause of morbidity and mortality in patients of CKD, particularly the patients of ESRD on renal replacement therapy.[3] CKD patients a have 7 to 10-fold higher mortality risk (40% death due to cardiovascular causes) compared to general population.[10] Increased oxidative stress, accumulation of uremic toxins, dyslipidemia and phosphocalcic metabolism disorders are the main underlying mechanisms for CVD risk in CKD patients.[11,12] CVD risk starts even at earlier stages (at a GFR of about 75 ml/min) of CKD and continuously increases with deterioration of renal function.[13,14] Multiple factors contribute to the development of dyslipidemia in CKD patients . Also, dyslipidemia is a common complication of CKD and lipoprotein metabolism alteration and is associated with the decline in GFR; hence, lipid profile depends on the level of kidney function and the degree of proteinuria.[15,16] The activity of lipoprotein lipase and hepatic triglyceride lipase is reduced in patients with CKD resulting in abnormalities in various lipid parameters. CKD patients who are not dependent on dialysis have low HDL and high triglyceride level and normal or even low total cholesterol and LDL cholesterol but a more atherogenic profile is hidden under this spectrum which includes increased apolipoprotein-B, lipoprotein(a) (Lp(a)), intermediate and very LDL and small dense LDL particles. Also in patients of advanced CKD stages, LDL and HDL particles are often modified by the oxidative process, that leads to the formation of small lipoproteins and increased formation of oxidized LDL.[17,18]

This study evaluated the dyslipidemia in different stages of CKD patients. In our study hypertriglycerdemia was found in 37%, hypercholesterolemia in 36%, low HDL in 36%, high LDL in 36%, and high VLDL in 36% of the patients. In this study there was a statistically significant decline in HDL level from stage 3 to 5. Low HDL has been recognized to be an independent risk factor in the development of kidney disease.[19] CKD patients have reduced level of HDL in contrast individuals with normal renal functions.[20,21] This predisposes the CKD patients to higher risk of atherosclerosis development. Various epidemiological studies results shown that HDL is negative risk factor for atherosclerosis.[22] CKD patients have reduced level of apolipoproteins AI and AII, the main components of HDL leading to reduced level of HDL.[17] Present study showed similar findings with the study of Sabeela et al.[23] We found significant increase in LDL level from group stage 3 to 5. Elevated plasma LDL cholesterol concentration is common in nephritoc syndrome, but it is not a typical feature of patients with advanced CKD, especially those who are on HD. Elevated LDL cholesterol levels seem to be mainly due to reduced LDL catabolism, while the actual role of LDL synthesis is currently debated. The LDL particles are smaller, denser and more atherogenic in CKD. Increased oxidative stress results in highly oxidized forms of LDL which are more atherogenic in their nature, are increased in CKD. In various studies it has been shown that increased level of these smaller dense LDL increases the risk of CVD development. Our results were consistent with the study done by Samuelsson et al in which they observed, low-density lipoprotein (LDL) cholesterol was significantly associated with a more rapid decline in renal function.[24] The VLDL levels were found to be significantly increased from stage 3 to 5. Increase in VLDL cholesterol in CKD is mainly due to their reduced clearance as well as insulin resistance impelled overproduction of VLDL.[25,26] Results of present study were similar to these of Raju et al who found a significant increase of VLDL in CKD patients.[27]

We observed positive correlation of dyslipidemia with hsCRP. Similar findings were seen with the study of Martinez-Hervas and Real in which they reported that patients with familial combined hyperlipidemia had increased levels of hsCRP, suggesting that primary dyslipidemia was associated with oxidative stress and inflammation.[28] Negative correlation of dyslipidemia with serum albumin was also seen in the present study. Hypoalbuminemia leads to increased hepatic synthesis of VLDL leading to accumulation of LDL particles. Study conducted by kaysen et al shown similar results.[29]

This study elaborated the importance of dyslipidemia in CKD. There was a significant rise in LDL and VLDL, and significant decrease in HDL from stage 3 to 5 which suggests that the dyslipidemia is a common complication of CKD and is associated with increased risk of CVD and renal disease progression. The severity of dyslipidemia correlates with degree of renal dysfunction.[30] Early detection and treatment of dyslipidemia in CKD patients will help in preventing further disease progression and CVD outcome and mortality.

The present study was associated with certain limitations. One of the limitations of this study was that it was a cross-sectional study and no follow up was done. No intervention was done in this study as well as sample size was small and no control group was included in this study. A longitudinal study can assess the association between thyroid dysfunction and long term outcomes in CKD patients.

CONCLUSION: CKD patients have increased risk of CVD. They have a higher prevalence of dyslipidemia as compared to the general population. The majority of patients die because of cardiovascular related moratlity before even reaching to the ESRD. Therefore it is necessary to identify dyslipidemia in early stages to prevent the further progression of kidney dysfunction, leading to better quality of life and improved morbidity and mortality. So, regular checkup of lipid profile is recommended in patients with CKD.

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Table 1- Baseline demographics and clinical parameters

Baseline parameters (Mean±SD)	Group A (n=50)	Group B (n=50)	Group C (n=50)	P value
Age(yrs)	52.82 ± 11.44	53.2 ± 11.04	51.32 ± 14.58	>.05*
Weight(kg)	65.26 ± 11.63	59.92 ± 8.92	58.42 ± 9.43	<.05*
Height(m)	166.18 ± 7.99	164.54 ± 8.92	163.51 ± 8.76	>.05*

Body Mass Index(kg/m2)	23.71 ± 3.61	22.12 ± 2.58	21.8 ± 2.58	<.05*
Duration of disease(yrs)	5.61 ± 4.63	5.84 ± 2.66	6.55 ± 5.42	>.05*
Pulse rate(bpm)	79.82 ± 8.59	77.6 ± 9.28	79.56 ± 7.52	>.05*
Systolic Blood Pressure (mmHg)	128.68 ± 15.58	128.68 ± 17.33	135.22 ± 21.91	>.05#
Diastolic Blood Pressure(mmHg)	81.28 ± 8.61	81.04 ± 9.21	82.14 ± 10.4	>.05#
Mean Arterial Pressure(mmHg)	97.08 ± 10.46	96.92 ± 11.42	99.83 ± 13.28	>.05#

Table 2- Baseline biochemical parameters and lipid profile

Baseline parameters (Mean±SD)	Group A (n=50)	Group B (n=50)	Group C (n=50)	P value
Haemoglobin (g/dL)	11.06 ± 1.51	9.86 ± 1.78	8.47 ± 1.38	<.05*
Total Leukocyte Count	7246.8 ± 2088.02	7769 ± 2086.18	8113.4 ± 2864.54	>.05#
Blood sugar (mg/dL)	100.5 ± 21.7	111.52 ± 35.59	129.3 ± 38.95	<.05*
Blood urea (mg/dL)	83.93 ± 35.72	108.8 ± 38.79	196.34 ± 75.39	<.05*
S. uric acid(mg/dL)	5.83 ± 1.48	7.68 ± 2.32	8.08 ± 2.76	<.05*
Serum Sodium (mEq/L)	139.52 ± 3.27	139.92 ± 3.02	140.96 ± 4.81	>.05*
Serum potassium (mEq/L)	4.27 ± 0.54	4.17 ± 0.45	4.25 ± 0.6	>.05*
Serum creatinine (mg/dL)	1.98 ± 0.57	3 ± 0.59	7.43 ± 2.71	<.05*
Serum calcium (mg/dL)	9.2 ± 0.75	8.68 ± 0.66	7.77 ± 0.51	<.05*
Serum phosphate (mg/dL)	3.84 ± 1.11	4.75 ± 1.57	6.38 ± 1.81	<.05*
Serum protein(g/dL)	6.4 ± 0.67	6.6 ± 0.61	6.38 ± 0.6	>.05*
Serum albumin(g/dL)	3.36 ± 0.41	3.44 ± 0.36	3.24 ± 0.4	>.05*
eGFR (ml/min/1.73m2)	40.36 ± 7.34	21.58 ± 4.12	8.68 ± 3.18	<.05*
hs-CRP (mg/dL)	0.85 ± 0.84	1.24 ± 1.06	2.51 ± 1.39	<.05*
Serum Apo B(g/L)	1.21 ± 0.43	1.24 ± 0.36	1.27 ± 0.31	>.05*
S. Triglycerides (mg/dL)	154.16 ± 42.63	159.98 ± 46.43	165 ± 44.68	>.05*
S. Cholesterol(mg/dL)	199.78 ± 40.89	190.48 ± 36.25	192.66 ± 44.02	>.05*
S. HDL-Cholesterol(mg/dL)	39 ± 10.9	37.16 ± 12.44	30.88 ± 12.14	<.05*
S. LDL Cholesterol(mg/dL)	145.28 ± 23.29	151.48 ± 19.63	160.24 ± 23.13	<.05*
S. VLDL Cholesterol(mg/dL)	26.88 ± 7.83	31.34 ± 10.18	38.82 ± 14.74	<.05*

*eGFR–Estimated Glomerular Filtration Rate, Apo B–Apolipoprotein-B, HDL–High Density Lipoprotein, LDL–Low Density Lipoprotein, VLDL–Very Low Density Lipoprotein,hs-CRP-High Sensitivity C Reactive Protein
*-Analyzed by KruskalWalis test
#-Analyzed by ANOVA

Table 3- Correlation of bio chemical parameters with lipid profile.

		S. HDL-Cholesterol(mg/dL)	S. LDL Cholesterol(mg/dL)	S. Cholesetrol(mg/dL)	S. Triglycerides (mg/dL)	S. VLDL Cholesterol(mg/dL)
	Correlation Coefficient	0.073	-0.017	-0.045	0.023	-0.117
Blood urea (mg/dL)	P value	>.05	>.05	>.05	>.05	>.05
Serum creatinine (mg/dL)	Correlation Coefficient	0.051	0.038	-0.099	0.103	-0.107
	P value	>.05	>.05	>.05	>.05	>.05
	P value	>.05	>.05	>.05	>.05	>.05
Serum albumin(g/dL)	Correlation Coefficient	0.494	-0.524	-0.361	-0.496	-0.407
	P value	<.01	<.01	<.01	<.01	<.01
hs-CRP (mg/dL)	Correlation Coefficient	-0.433	0.414	0.357	0.455	0.354
	P value	<.01	<.01	<.01	<.01	<.01
Serum Apo B (g/L)	Correlation Coefficient	-0.622	0.636	0.469	0.635	0.622
	P value	<.01	<.01	<.01	<.01	<.01

*eGFR–Estimated Glomerular Filtration Rate, Apo B–Apolipoprotein-B
Spearman rank correlation coefficient

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