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Original Research Article

DYSLIPIDEMIA AND CARDIOVASCULAR CHANGES IN CHRONIC KIDNEY DISEASE

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ABSTRACT

INTRODUCTION

This study was carried out to assess the lipid profile and cardiovascular complications in patients with chronic kidney disease.

METHODS

This was a cross-sectional study carried out over a period of 2 years involving 60 patients with chronic kidney disease who were admitted to the Government General Hospital at Kurnool. Patients with bilaterally contracted kidneys, those with a GFR < 60 mL/min/1.73m² and adults aged > 18 years with a history of kidney disease for a duration of > 6 months were included in the study.

RESULTS

The mean total cholesterol of overall patients was 189.8 ± 14.72 mg/dl. The mean total cholesterol was significantly higher in patients with stage 5 CKD compared to stage 4 and stage 3 CKD patients. ($p < 0.05$; significant). The mean triglycerides of overall patients were 174.8 ± 16.29 mg/dl. There was no significant difference in mean triglycerides between different stages of CKD patients. ($p > 0.05$; not significant). The mean HDL-cholesterol of overall patients was 37.50 ± 7.56 mg/dl. There was no significant difference in mean HDL-cholesterol between different stages of CKD patients. ($p>0.05$; not significant). The mean LDL-cholesterol of overall patients was 132.2 ± 15.74 mg/dl. There was no significant difference in mean LDL-cholesterol between different stages of CKD patients. ($p>0.05$; not significant). The mean VLDL-cholesterol

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of overall patients was 37.50 ± 12.36 mg/dl. There was no significant difference in mean VLDL-cholesterol between different stages of CKD patients. ($p > 0.05$; not significant).

CONCLUSION

CKD continues to be a major disease affecting males in the age group of 41 to 50 years. Dyslipidemia in the form of higher cholesterol and triglyceride levels and cardiovascular changes are observed more frequently in CKD patients, especially in stages 4 and 5. Further research is warranted towards mitigating these important risk factors in order to reduce mortality in this cohort of patients.

KEY WORDS

Dyslipidemia, Chronic Kidney Disease (CKD), Cardiovascular Complications.

INTRODUCTION

CKD is a global public health problem, especially in developing countries like India. According to the National Kidney Foundation of India, behind cancer and heart disease, kidney diseases are the third most serious illnesses that can lead to death. Every year, some 200,000 people get renal failure, and millions more experience kidney disorders that are less severe.[1]

Some of the known risk factors for chronic kidney disease (CKD) include age, male sex, smoking, dyslipidemia, obesity, hypertension, diabetes, hyperparathyroidism, hyperhomocysteinemia, anemia, hypoalbuminemia, oxidative stress, and chronic inflammation. Dyslipidemia is one of the main consequences of chronic renal illness. Changes in lipoprotein metabolism may occur early in the course of chronic renal disease. These alterations usually worsen with time, reflecting the deterioration of renal function.[2] A number of recently published studies have indicated that dyslipidemia is a major factor in the development of cardiovascular disease and the decline in renal function. However, it appears that the pattern of dyslipidemia that the various researchers have identified differs significantly in some respects.[3]

Cardiovascular illness is increasingly recognised as the primary cause of death in people with end-stage renal failure. Age-adjusted cardiovascular problems and death from end-stage renal disease are approximately 30 times greater than in the general population.[4] Peripheral vascular disease, angina pectoris, myocardial infarction, dysrhythmia, cardiac failure, and stroke are all more prevalent in end-stage renal disorders.[5] Whether or not cardiomyopathy exhibits clinical silence, it is a reliable indicator of heart morbidity and death.[6]

This study was therefore carried out to assess the lipid profile and cardiovascular complications in patients with chronic kidney disease.

METHODS

This was a cross-sectional study carried out over a period of 2 years involving 60 patients with chronic kidney disease who were admitted to the Government General Hospital at Kurnool. Patients with bilaterally contracted kidneys, those with a GFR < 60 mL/min/1.73 m² and adults aged > 18 years with a history of kidney disease for a duration of > 6 months were included in the study. Patients with a history of parenteral iron injections in the last 14 days, those already on lipid-lowering agents and patients with ischemic heart disease were excluded from the study. A

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detailed history, physical examination and investigations were recorded. The gathered data was entered into Microsoft Excel. Percentages were used to express frequencies. The standard deviation and mean were used to express continuous data. The student T-test and the Chi-square test were employed as significance tests. Software for statistical analysis, SPSS version 26, was used. The level of significance was taken as $p < 0.05$.

RESULTS

Patients included in the study were between 25 and 72 years of age. The mean age of the CKD patients was 48.9 ± 11.42 years, with a majority of 35% being between 41 and 50 years old.

Out of 60 CKD patients studied, 40 (66.7%) were males and 20 (33.3%) were females.

With respect to the duration of the disease, 13 (21.7%) patients had CKD from the past 6 to 12 months, 15 (25%) from 13 to 24 months, 26 (43.3%) from 25 to 36 months, and the remaining 6 (10%) patients for more than 36 months. Most of the patients had CKD for the past 25 to 36 months. The mean duration of CKD in the studied patients was 24.4 ± 11.29 months with a range of 7 to 54 months.

Out of 60 CKD patients, diabetic nephropathy (58.3%) was the leading cause of CKD, followed by chronic glomerulonephritis (21.7%), hypertension (8.3%), chronic interstitial nephritis (5%), obstructive nephropathy (5%) and autosomal dominant polycystic kidney disease (1.7%). Out of 60 CKD patients studied, 24 (40%) were in stage 3, 16 (26.7%) were in stage 4 and 20 (33.3%) were in stage 5 of chronic kidney disease. (Table 1)

CKD Stage	No. of Patients	Percentage
Stage 3	24	40%
Stage 4	16	26.7%
Stage 5	20	33.3%
Total	60	100%

Table 1: Distribution of CKD Patients based on the Stage of the Disease

The mean total cholesterol of patients with stage 3 CKD was 183.3 ± 13.15 mg/dl, for patients with stage 4 CKD, it was 191.3 ± 15.43 mg/dl and for patients with stage 5 CKD, it was 196.3 ± 13.30 mg/dl. The mean total cholesterol of overall patients was 189.8 ± 14.72 mg/dl. The mean cholesterol levels were thus higher in patients with stage 5 compared to stages 3 and 4. This difference was significantly significant ($p = 0.011$) (Table 2)

Stage of CKD	Mean of Total Cholesterol (mg/dl)	Standard Deviation	ANOVA (F) Value	P-Value
Stage 3	183.3	13.15	4.927	0.011; Significant
Stage 4	191.3	15.43		
Stage 5	196.3	13.30		
Overall	189.8	14.72		

Table 2: Distribution of CKD Patients by Total Cholesterol and Stage of CKD

The mean HDL-cholesterol of patients with stage 3 CKD was 39.75 ± 8.48 mg/dl, for patients with stage 4 CKD, it was 36.88 ± 7.13 mg/dl and for patients with stage 5 CKD, it was

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

35.30 ± 6.22 mg/dl. The difference was not significant (p = 0.141). The mean LDL-cholesterol of patients with stage 3 CKD was 127.7 ± 14.14 mg/dl, for patients with stage 4 CKD, it was 131.9 ± 17.26 mg/dl and for patients with stage 5 CKD, it was 137.8 ± 15.29 mg/dl. The mean VLDL-cholesterol of patients with stage 3 CKD was 34.96 ± 12.31 mg/dl, for patients with stage 4 CKD, it was 38.69 ± 12.60 mg/dl and for patients with stage 5 CKD, it was 39.60 ± 12.32 mg/dl. The mean VLDL-cholesterol of overall patients was 37.50 ± 12.36 mg/dl. Although, the levels were higher with higher stages, there was no significant difference in mean LDL or VLDL cholesterol between different stages of CKD patients. (p > 0.05; not significant)

The mean triglycerides of patients with stage 3 CKD were 171.6 ± 19.30 mg/dl, for patients with stage 4 CKD, it was 175.2 ± 14.29 mg/dl and for patients with stage 5 CKD, it was 178.2 ± 13.66 mg/dl. The mean triglycerides of overall patients were 174.8 ± 16.29 mg/dl. There was no significant difference in mean triglycerides between different stages of CKD patients. (p > 0.05; not significant) (Table 3)

Stage of CKD	Mean Triglycerides (mg/dl)	Standard Deviation	ANOVA (F) Value	P-Value
Stage 3	171.6	19.30	0.880	0.420; Not Significant
Stage 4	175.2	14.29		
Stage 5	178.2	13.66		
Overall	174.8	16.29		

Table 3: Distribution of CKD Patients by Triglycerides and Stages of CKD

Cardiovascular Changes

Out of 60 CKD patients studied, the majority, 40 (66.7%), showed left ventricular hypertrophy in the ECG, followed by ischemic changes in 36 (60%), ST-T changes in 20 (33.3%), occasional ventricular premature complexes in 15 (25%), low voltage complexes in 10 (16.7%) and only one (1.7%) patient had left bundle branch block. (Table 4)

ECG Changes	Number (n=60)	Percentage
Left Ventricular Hypertrophy (LVH)	40	66.7%
Ischemia changes	36	60%
ST-T changes	20	33.3%
Ventricular Premature Complexes (VPC)	15	25%
Low Voltage Complexes (LVC)	10	16.7%
Left Bundle Branch Block (LBBB)	1	1.7%

Table 4: Distribution of CKD Patients by ECG Changes

Out of 60 CKD patients studied, the majority i.e., 30 (50%) patients, showed concentric left ventricular hypertrophy in echocardiography, followed by dilated left ventricle in 14 (23.3%), dilated left atria in 10 (16.7%), and all chambers were dilated in 6 (10%) patients. (Table 5)

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

Chamber Dilatation	Number	Percentage
Concentric LVH	30	50%
Dilated Left Ventricle	14	23.3%
Dilated Left Atria	10	16.7%
All chambers dilatation	6	10%
Total	60	100%

Table 5: Distribution of CKD Patients by Chamber Dilatation

Mitral regurgitation (31.7%) was the major valve abnormality in the present study. The second major valve abnormality was posterior mitral annular calcification, reported in 30%. Other valve abnormalities reported in CKD patients were aortic sclerosis (26.7%), aortic regurgitation (12%), and tricuspid regurgitation (15%). Among patients with mitral regurgitation, 6 patients had trivial MR, 9 patients had mild MR, and 4 patients had moderate MR. Among aortic regurgitation, 5 patients had trivial AR and 7 patients had mild TR. In tricuspid regurgitation, 2 patients had trivial TR and 7 patients had mild TR. (Table 6)

Valve Abnormalities	Number	Percentage
Mitral Regurgitation	19	31.7%
Aortic Regurgitation	12	20%
Tricuspid Regurgitation	9	15%
Posterior mitral annular calcification	18	30%
Aortosclerosis	16	26.7%

Table 6: Distribution of CKD Patients by Valve Abnormalities

11 (18.3%) and 33 (55%) patients with CKD were reported to have systolic and diastolic dysfunction, respectively. In systolic dysfunction, 4 patients had mild and 7 patients had moderate dysfunction. Pericardial effusion was reported in 15 (25%) patients and left ventricular hypokinesis was reported only in 3 (5%) patients. (Table 7)

Left Ventricular and Other Abnormalities	Number	Percentage
Systolic dysfunction	11	18.3%
Diastolic dysfunction	33	55%
Left ventricular hypokinesia	3	5%
Pericardial effusion	15	25%

Table 7: Distribution of CKD Patients by Left Ventricular and Other Abnormalities

DISCUSSION

In the present study, the mean total cholesterol levels were higher in stage 5 compared to stages 3 and 4. This difference was significantly significant ($p = 0.011$). The mean HDL-cholesterol of patients with stage 3 was higher compared to the other two stages, with no statistically significant difference. Although the levels were higher with higher stages, there was no significant difference in mean LDL or VLDL cholesterol between different stages of CKD patients. ($p > 0.05$). There was no significant difference in mean triglyceride levels between

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

different stages of CKD patients ($p > 0.05$), although the levels were higher in stage 5 compared to stages 3 and 4.

A study conducted by Rashmi Rekha et al. found that the CKD group had higher triglycerides and VLDL and lower HDL-C compared to the control group.[7] Similar results were reported by Raju et al., who found that the CKD group's triglyceride and VLDL levels were higher and its HDL-C levels were lower than those of the control group. LDLC (Low-Density Lipoprotein Cholesterol) and serum total cholesterol in the two groups do not differ significantly from one another.[8] According to Chijioke et al., the TC, TG, HDL-C, LDL-C, and VLDL levels of people with chronic renal illness were very different from those in the control group. This was true for both men and women. The study group's cardiovascular risk indicators, such as TC/HDL-C and LDL-C/HDL-C, were greater than the control group's.[9]

According to Ekonoyan, one of the basic abnormalities of lipoprotein metabolism in renal illness is reduced catabolism of the lipoprotein-rich triglyceride. According to this study, triglyceride itself and triglyceride-rich lipoprotein (VLDL and LDL-C) were shown to be considerably greater in individuals with chronic renal illness, which predisposes them to cardiovascular disease.[[10] Low HDL levels in CKD patients were identified as one of the independent risk variables for the advancement of renal disease in the MDRD research.[11]

Chan MK et al.[12] also found hypertriglyceridemia to be the major abnormality in their studies and concluded that hypertriglyceridemia represents an early feature of renal failure. According to a study by Rajman I et al., uremic patients typically have normal or slightly reduced LDL-C concentrations and show significant abnormalities in the density distribution of the LDL subfraction, which are characterised by a predominance of small, dense LDL particles.[13] Massy et al.[14] observed no correlations between the individual classical lipid markers, such as LDL-C, and the development of ESRD (End-Stage Renal Disease), which is in contrast to the results of the current investigation. However, plasma triglyceride content was found to be an independent risk factor for a 25% drop in creatinine clearance in a post-hoc analysis of population research involving 12,728 participants with serum creatinine < 2.0 mg/dl.[15]

Numerous studies demonstrate that patients with CKD have lower HDL levels than people with normal renal function. Given the findings of numerous epidemiological studies that identified HDL as a harmful risk factor for atherosclerosis, this condition puts them at a higher risk for the development of atherosclerosis. Reverse cholesterol transport, which involves moving cholesterol from the arterial wall to the liver for subsequent excretion, is the primary job of HDL. This works in conjunction with HDL-mediated inhibition of inflammation, platelet adhesion, and LDL oxidation to prevent atherosclerosis under normal conditions, but in CKD patients, HDL's protective function is diminished for a variety of reasons.[16-18]

According to numerous studies, CKD patients who are on HD typically have similar lipid profiles to those who are not dependent on dialysis for their care. Triglyceride levels are increased, HDL is low, and TC and LDL levels are often rather normal. Rarely, LDL in these people noticeably increased.

However, according to the K/DOQI guideline on dyslipidemias in CKD patients, 55.7% of patients with HD have LDL values that are greater than 100 mg/dL. Both these quantitative and qualitative lipid abnormalities contribute to atherosclerosis and cardiovascular death in HD

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patients. Cardiovascular mortality is 30 times greater in dialysis patients and accounts for about 50% of deaths in ESRD patients.[19-21]

Cardiovascular Changes

In the present study, a majority of 40 (66.7%) patients showed left ventricular hypertrophy in ECG .This observation of an increased prevalence of LVH in patients with CKD was similar to those described by others.[22,23] A higher prevalence of LVH in CKD patients has also been reported—up to 78% of patients with CKD stages 3–5 and up to 51% of patients with CKD stages 1–2.[24]

41% of patients in a study by Parfrey PS et al.[25] at the Salvation Army Grace General Hospital's Division of Nephrology in Canada exhibited concentric left ventricular hypertrophy. 52% of patients had an incidence of LVH, according to Dai Y. et al.[26] According to Gruppen MP et al., LVH was found in 39% of female patients and 47% of male patients.[27]

In the current study, 33.3% of patients had ST-T alterations, while 60% of patients had ischemia abnormalities. In contrast to previous stages of CKD, Akshat Jain et al.[28] found that the ST segment was significantly lowered in stages 4 and 5. In a similar vein, main T-wave inversion was more common in stages 4 and 5 of CKD compared to stage 1. Additionally, individuals with pathologic Q waves, slow R wave development, and low QRS voltage were only observed in stages 4 and 5, not in earlier stages.

In contrast to hypertensive patients without CKD, non-dialysis CKD patients with hypertension were observed to have a greater prevalence of LVH. In a 12-month longitudinal analysis of individuals with stage 3 CKD, LVH progression was noted despite stable blood pressure and kidney function.[29]

In the present study, concentric LVH was observed in 50% and a dilated left ventricle in 23.3% of CKD patients. The Parfrey PS et al.[30] investigation found that 28% of the subjects had dilated left ventricles. Systolic dysfunction was present in 4 out of 14 patients with a dilated left ventricle. Among the 14 patients with a dilated left ventricle, two additionally had pulmonary interstitial edoema and 10 showed cardiomegaly on a chest X-ray.

In the present study, systolic dysfunction was seen less frequently, in 18.3%, compared to diastolic dysfunction observed in 55% of patients. Park et al. looked at the CRIC cohort and found that irregular LV geometry, diastolic dysfunction, and LV hypertrophy were all connected to getting worse kidney function, but not systolic dysfunction.[31] In the PARAMOUNT experiment, 217 patients with heart failure and retained ejection fraction had their cardiac anatomy and function characterised by Gori et al. They discovered that lower mid-wall fractional shortening, greater LV mass or LV mass index, and aberrant LV shape were all linked to renal impairment.[32]

Only 16.7% of participants in the current research had dilated left atria, compared to 23.3% who had dilated left ventricles. In the VALIANT experiment, lower kidney function was linked to a smaller left ventricle, a bigger left atrial volume, and a higher left ventricle mass index in a group of people who had a myocardial infarction.[33]

In our study, pericardial effusion was seen in 15 patients (25%). 23 cases of mild pericardial effusion-defined as echo-free space less than 1 cm-and 2 cases of moderate effusion-defined as echo-free space between 1 and 2 cm-were found in the research by Frommer JP et al.[34]

ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

In our study, mitral valve abnormalities were more common compared to aortic valves. According to Raine, A.E.G., there was a 28-55% incidence of aortic valve calcification in end-stage renal disease, with aortic stenosis accounting for 3–13% of cases.[35]

CONCLUSION

CKD continues to be a major disease affecting males in the age group of 41 to 50 years. Dyslipidemia in the form of higher cholesterol and triglyceride levels and cardiovascular changes are observed more frequently in CKD patients, especially in stages 4 and 5. Further research is warranted towards mitigating these important risk factors in order to reduce mortality in this cohort of patients.

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ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 1, 2024

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