

**Dyslipidemia and Cardiovascular Changes in Chronic Kidney Disease**Sridhar Panda<sup>1</sup>, Sanjay Choudhuri<sup>2</sup>, Sujit Kumar Mohanty<sup>3</sup><sup>1</sup>Assistant Professor, Department of General Medicine, SCB MCH, Cuttack<sup>2</sup>Assistant Professor, Department of Urology & Kidney Transplantations, SCB Medical College & Hospital, Cuttack<sup>3</sup>Assistant Professor, Department of General Surgery, SCB Medical College & Hospital, Cuttack

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Conflict of interest: Nil

**Abstract:****Introduction:** This research evaluated the lipid profile and cardiovascular consequences in individuals with chronic renal disease.**Methods:** This 2-year cross-sectional research included 60 patients with chronic renal disease admitted to the SCB Medical College, Cuttack. The trial included patients with bilaterally constricted kidneys, a GFR < 60 mL/min/1.73m<sup>2</sup>, and people over 18 with a history of renal illness lasting more than 6 months.**Results:** The average total cholesterol for all patients was 189.8 ± 14.72 mg/dl. The mean total cholesterol in individuals with stage 5 CKD was considerably greater than in stage 4 and stage 3 patients. (p < 0.05, significant). The average triglyceride level across patients was 174.8 ± 16.29 mg/dL. There was no significant variation in mean triglycerides among CKD patients at various stages. (p > 0.05, not significant). The average HDL-cholesterol level across patients was 37.50 ± 7.56 mg/dL. There was no significant variation in mean HDL cholesterol levels across various stages of CKD patients. (p>0.05, not significant). The average LDL-cholesterol level across patients was 132.2 ± 15.74 mg/dL. There was no significant variation in mean LDL-cholesterol levels across various stages of CKD patients. (p>0.05, not significant). The mean VLDL cholesterol level across patients was 37.50 ± 12.36 mg/dL. There was no significant variation in mean VLDL-cholesterol levels between various stages of CKD patients. (p > 0.05, not significant).**Conclusion:** CKD is a serious illness affecting guys aged 41 to 50 years. Dyslipidemia (increased cholesterol and triglyceride levels) and cardiovascular abnormalities are more common in CKD patients, particularly in stages 4 and 5. Further study is needed to address these critical risk factors and minimize mortality in this patient population.**Keywords:** Dyslipidemia, Chronic Kidney Disease (CKD), Cardiovascular Complications.This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.**Introduction**

Chronic kidney disease (CKD) is a worldwide public health issue, particularly in poor nations such as India. According to the National renal Foundation of India, renal disorders are the third most deadly illnesses that may cause mortality, after only cancer and heart disease. Every year, around 200,000 individuals die because of renal failure, while millions more suffer from less serious kidney illnesses.[1]

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. One of the most common side effects of chronic kidney disease is dyslipidemia.

Changes in lipoprotein metabolism may appear early in the course of chronic renal illness. These changes often worsen with time, indicating impairment of renal function.[2] Several recently published research have shown that dyslipidemia has a significant role in the development of cardiovascular disease and the decrease of renal function. However, it seems that the dyslipidemia patterns reported by the different studies varied greatly in certain ways. According to [3], cardiovascular sickness is the leading cause of mortality in those with end-stage renal failure.

Age-adjusted cardiovascular issues and mortality from end-stage renal disease are almost 30 times higher than in the overall population.[4] End-stage renal diseases are more likely to cause peripheral vascular disease, angina pectoris, myocardial

infarction, dysrhythmia, cardiac failure, and stroke.[5]

Whether or whether cardiomyopathy is clinically silent, it is a consistent predictor of cardiac illness and mortality.[6] This research aimed to evaluate the lipid profile and cardiovascular consequences in chronic renal disease patients.

#### Methods:

This 2-year cross-sectional research included 60 patients with chronic renal disease admitted to the SCB Medical College and Hospital, Cuttack. The trial included patients with bilaterally constricted kidneys, those with a GFR < 60 mL/min/1.73 m<sup>2</sup>, and individuals over 18 with a history of renal illness lasting more than 6 months. Patients who had received parenteral iron injections within the previous 14 days, were already using lipid-lowering medicines, or had ischemic heart disease were excluded from the trial.

A complete history, physical examination, and investigations were documented. The acquired information was put into Microsoft Excel. Frequencies were expressed as percentages. The standard deviation and mean were employed to represent continuous data. The student T-test and Chi-square test were used as significance tests. The statistical analysis software utilized was SPSS

version 26. The criterion of significance was set at  $p < 0.05$ .

#### Results

The research comprised patients aged 25-72. The average age of CKD patients was  $48.9 \pm 11.42$  years, with 35% aged 41-50. Out of the 60 CKD patients evaluated, 40 (66.7%) were men and 20 (33.3%) were women.

In terms of illness duration, 13 (21.7%) patients had CKD for the previous 6 to 12 months, 15 (25%) from 13 to 24 months, 26 (43.3%) from 25 to 36 months, and the remaining 6 (10%) for more than 36 months. The majority of the patients had CKD during the previous 25 to 36 months. The average duration of CKD in the study participants was  $24.4 \pm 11.29$  months, with a range of 7 to 54 months.

Diabetic nephropathy was the predominant cause of CKD in 60 patients, accounting for 58.3%, followed by chronic glomerulonephritis (21.7%), hypertension (8.3%), chronic interstitial nephritis (5%), obstructive nephropathy (5%), and autosomal dominant polycystic kidney disease (1.7%). Of the 60 CKD patients investigated, 24 (40%) were in stage 3, 16 (26.7%) were in stage 4, and 20 (33%) were in stage 5. (Table 1).

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

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

Patients with stage 3 CKD had an average total cholesterol level of  $183.3 \pm 13.15$  mg/dl, whereas those with stage 4 CKD had an average of  $191.3 \pm 15.43$  mg/dl and stage 5 CKD had an average of  $196.3 \pm 13.30$  mg/dl. The average total cholesterol for all patients was  $189.8 \pm 14.72$  mg/dl. Patients with stage 5 had greater mean cholesterol levels than those with stages 3 and 4. The difference was substantial ( $p = 0.011$ ) (Table 2).

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

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		

Patients with stage 3 CKD had a mean HDL-cholesterol of  $39.75 \pm 8.48$  mg/dl, whereas those with stage 4 CKD had a mean of  $36.88 \pm 7.13$  mg/dl. Patients with stage 5 CKD had a mean of  $35.30 \pm 6.22$  mg/dl.

The change was insignificant ( $p = 0.141$ ). Patients with stage 3 CKD had an average LDL-cholesterol of  $127.7 \pm 14.14$  mg/dl, whereas those with stage 4 CKD had a mean of  $131.9 \pm 17.26$  mg/dl and stage 5 CKD had an average of  $137.8 \pm 15.29$  mg/dl. Patients with stage 3 CKD had a mean VLDL-

cholesterol of  $34.96 \pm 12.31$  mg/dl, whereas those with stage 4 CKD had a mean of  $38.69 \pm 12.60$  mg/dl and stage 5 CKD had a mean of  $39.60 \pm 12.32$  mg/dl. The average VLDL cholesterol level across patients was  $37.50 \pm 12.36$  mg/dl. Although the levels increased with each stage, there was no significant difference in mean LDL or VLDL cholesterol levels among CKD patients. ( $p > 0.05$ , not significant)

Patients with stage 3 CKD had a mean triglyceride level of  $171.6 \pm 19.30$  mg/dl, whereas those with

stage 4 CKD had  $175.2 \pm 14.29$  mg/dl and stage 5 CKD had  $178.2 \pm 13.66$  mg/dl, respectively. Overall, patients had mean triglyceride levels of  $174.8 \pm 16.29$  mg/dL. There was no significant

variation in mean triglycerides among CKD patients at various stages. ( $p > 0.05$ , not significant). (Table 3.)

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

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		

**Cardiovascular Changes:** Out of 60 CKD patients studied, the majority, 40 (66.7%), had 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).

**Table 4: Distribution of CKD Patients by ECG Changes**

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%

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)

**Table 5: Distribution of CKD Patients by Chamber Dilatation**

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%

Mitral regurgitation (31.7%) was the most common valve anomaly in the current research.

The second most common valve anomaly was posterior mitral annular calcification, which was seen in 30% of cases. Other valve abnormalities found in CKD patients were aortic sclerosis (26.7%), aortic regurgitation (12%), and tricuspid

regurgitation (15%). Among individuals with mitral regurgitation, 6 had trivial MR, 9 had mild MR, and 4 had significant MR.

Among the individuals with aortic regurgitation, 5 had minor AR and 7 had mild TR. In tricuspid regurgitation, there were two individuals with trivial TR and seven with mild TR. (Table 6).

**Table 6: Distribution of CKD Patients by Valve Abnormalities**

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%

11 (18.3%) and 33 (55%) CKD patients were found to have systolic and diastolic dysfunction. Four individuals had mild systolic dysfunction, whereas seven had significant dysfunction. Pericardial effusion was detected in 15 (25%) individuals, but left ventricular hypokinesia was identified in just three (5%) patients. (Table 7).

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

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%

## Discussion

In the current investigation, the average total cholesterol levels were greater in stage 5 than in stages 3 and 4. The difference was statistically significant ( $p = 0.011$ ). Patients in stage 3 had greater mean HDL cholesterol than those in the previous two stages, but the difference was not statistically significant. Although the levels increased with each stage, there was no significant difference in mean LDL or VLDL cholesterol levels among CKD patients. ( $p > 0.05$ ). The mean triglyceride levels did not vary significantly between

Different stages of CKD patients ( $p > 0.05$ ), while the levels were greater in stage 5 than in stages 3 and 4.

Rashmi Rekha et al. discovered that the CKD group had more triglycerides, VLDL, and lower HDL-C than the control group.[7] Similar findings were reported by Raju et al., who discovered that the CKD group had greater triglyceride and VLDL levels and lower HDL-C levels than the control group. The two groups had similar LDL-C (Low-Density Lipoprotein Cholesterol) and blood total cholesterol levels.[8] Chijioke et al. found that the TC, TG, HDL-C, LDL-C, and VLDL levels of persons with chronic renal disease differed significantly from those of the controls. This held true for both men and women. The study group had higher levels of cardiovascular risk markers, such as TC/HDL-C and LDL-C/HDL-C, than the control group did.[9] Ekonoyan identifies decreased catabolism of lipoprotein-rich triglycerides as a key abnormality in lipoprotein metabolism during renal disease. According to this research, triglycerides and triglyceride-rich lipoprotein (VLDL and LDL-C) levels are much higher in those with chronic renal illness, predisposing them to cardiovascular disease.[10] Low HDL levels in CKD patients were found as one of the independent risk factors for renal disease progression in the MDRD study.[11]

Chan MK et al.[12] discovered hypertriglyceridemia to be the most significant anomaly in their research and concluded that hypertriglyceridemia is an early indicator of renal failure. According to Rajman I et al., uremic patients often have normal or slightly lower LDL-C values and exhibit major anomalies in the density distribution of the LDL subfraction, characterized by a preponderance of tiny, dense LDL particles.[13] Massy et al.[14] found no link between specific conventional lipid markers, such as LDL-C, and the development of ESRD (End-Stage Renal Disease), which contradicts the findings of the present study. A post-hoc study of population research comprising 12,728 subjects with serum creatinine  $< 2.0$  mg/dl revealed that plasma triglyceride level was an independent risk

factor for a 25% reduction in creatinine clearance.[15]

Numerous studies have shown that individuals with CKD had lower HDL levels than persons with adequate renal function. Given the results of multiple epidemiological investigations identifying HDL as a hazardous risk factor for atherosclerosis, this condition increases their chance of developing atherosclerosis. HDL's principal function is reverse cholesterol transport, which entails transferring cholesterol from the artery wall to the liver for elimination. This acts in tandem with HDL-mediated suppression of inflammation, platelet adhesion, and LDL oxidation to prevent atherosclerosis under normal circumstances, but HDL's protective effect is reduced in CKD patients for a number of reasons. [16-18]

According to multiple studies, CKD patients on HD often have comparable lipid profiles to individuals who do not need dialysis for treatment. Triglyceride levels rise, HDL falls, while TC and LDL levels remain relatively normal. LDL levels in these persons have seldom grown appreciably.

However, according to the K/DOQI guideline for dyslipidemias in CKD patients, 55.7% of HD patients had LDL levels more than 100 mg/dL. Both qualitative and quantitative lipid abnormalities lead to atherosclerosis and cardiovascular mortality in HD patients. Cardiovascular mortality is 30 times higher in dialysis patients and accounts for almost half of all ESRD fatalities. [19-21]

### Cardiovascular Changes

In the current research, the majority of 40 patients (66.7%) had left ventricular hypertrophy on ECG. This finding of a higher frequency of LVH in CKD patients was consistent with previous reports.[22,23] A greater 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 individuals with stages 1-2.[24]

In a research conducted by Parfrey PS et al.[25] at the Salvation Army Grace General Hospital's Division of Nephrology in Canada, 41% of patients had concentric left ventricular hypertrophy. Dai Y. et al. reported that 52% of patients developed LVH.[26] According to Gruppen MP et al., LVH occurred in 39% of female patients and 47% of male patients.[27]

In the present research, 33.3% of patients showed ST-T changes, and 60% had ischemic abnormalities. In contrast to earlier phases of CKD, Akshat Jain et al. [28] discovered that the ST segment was drastically reduced in stages 4 and 5. In a similar vein, primary T-wave inversion was more likely in stages 4 and 5 of CKD than stage 1. Furthermore, people with pathologic Q waves,

sluggish R wave development, and low QRS voltage were only seen in stages 4 and 5, not earlier stages.

In contrast to hypertensive individuals without CKD, non-dialysis CKD patients with hypertension were shown to have a higher incidence of LVH. In a 12-month longitudinal study of people with stage 3 CKD, LVH development was seen despite stable blood pressure and renal function.[29]

In the current research, 50% of CKD patients had concentric LVH, whereas 23.3% had a dilated left ventricle. The Parfrey PS et al.[30] study discovered that 28% of the individuals had dilated left ventricles. Systolic dysfunction was found in four of the 14 individuals with a dilated left ventricle. Two of the 14 patients with a dilated left ventricle also exhibited pulmonary interstitial edoema, and ten showed cardiomegaly on a chest X-ray.

In the current investigation, systolic dysfunction was identified in 18.3% of patients, whereas diastolic dysfunction was reported in 55%. Park et al. examined the CRIC cohort and found that irregular LV geometry, diastolic dysfunction, and LV hypertrophy were all linked to lower kidney function, but not systolic dysfunction.[31] Gori et al. investigated the cardiac structure and function of 217 individuals with heart failure and maintained ejection fraction in the PARAMOUNT study. They observed that reduced mid-wall fractional shortening, increased LV mass or LV mass index, and abnormal LV shape were all associated with renal impairment.[32]

Only 16.7% of the individuals in the present study had dilated left atria, whereas 23.3% had dilated left ventricles. In the VALIANT study, worse kidney function was associated with a smaller left ventricle, a larger left atrial volume, and a higher left ventricular mass index in a group of myocardial infarction patients.[33]

In our research, 15 individuals (25%) had pericardial effusion. The study by Frommer JP et al. revealed 23 instances of mild pericardial effusion (defined as echo-free space less than 1 cm) and 2 cases of severe effusion (defined as echo-free space between 1 and 2 cm).[34] Mitral valve anomalies were found to be more prevalent in our research than aortic valves. Raine, A.E.G. reported a 28-55% incidence of aortic valve calcification in end-stage renal illness, with aortic stenosis accounting for 3-13% of cases.[35]

### Conclusion

CKD is a serious illness affecting guys aged 41 to 50 years. Dyslipidemia (increased cholesterol and triglyceride levels) and cardiovascular abnormalities are more common in CKD patients, particularly in stages 4 and 5. Further study is

needed to address these critical risk factors and minimize mortality in this patient population.

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