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Dyslipidemia spectrum among chronic kidney disease patients visiting an Eastern Indian tertiary-level hospital: An observational study

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Abstract:
Chronic kidney disease (CKD) alters lipoprotein metabolism, but treatment-modality-specific variation in lipid patterns remains inadequately characterized. In this analytical cross-sectional study conducted between March 2015 and March 2017, fasting lipid profiles were compared among 96 CKD patients not receiving lipid-lowering therapy, equally distributed across dialysis-dependent CKD (D-CKD), nondialysis-dependent CKD (ND-CKD) and post-renal-transplant groups (n=32 each), with dyslipidemia classified into seven predefined patterns. Overall dyslipidemia was identified in 84.4% of the cohort with no significant difference among groups (78.1% D-CKD, 87.5% ND-CKD and 87.5% post-transplant; p=0.49), whereas hyperlipidemia alone showed borderline intergroup variation after recalculation (50% D-CKD vs 75% in both ND-CKD and post-transplant groups; p=0.050). Elevated LDL cholesterol was numerically more prevalent in post-transplant recipients (53.1%) than in D-CKD patients (34.4%; three-group p=0.154) and mixed hyperlipidemia was the most frequent pattern overall. Thus, data shows that treatment modality may shape the pattern, although not the overall prevalence, of dyslipidemia across the CKD continuum.

Keywords: Chronic kidney disease (CKD), dialysis, dyslipidemia, lipid phenotype, lipoprotein metabolism, renal transplantation

Background:
Dyslipidemia refers to abnormal elevation or reduction of circulating lipid parameters including low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL), total cholesterol (TC) and triglycerides (TG) and remains a major modifiable cardiovascular risk factor **[1].** Unlike hyperlipidemia, which denotes elevation of lipid parameters alone, dyslipidemia encompasses both elevated and reduced lipid fractions, particularly reduced HDL cholesterol. Management guidelines define total cholesterol <200 mg/dl, LDL cholesterol < 100mg/dl, triglycerides < 150mg/dl and HDL cholesterol ≥ 40mg/dl in adults as acceptable **[2].** Dyslipidemia is a key factor in atherosclerotic cardiovascular disease and contributes substantially to endothelial dysfunction and plaque formation **[3, 4].** CKD is described as a structural or functional kidney abnormality persisting for more than three months and is recognized as an independent cardiovascular risk state **[5].** Cardiovascular disease represents the dominant cause of fatality in CKD populations and lipid abnormalities are strongly associated with this excess risk burden. In addition to traditional cardiovascular risk factors, CKD-specific metabolic and inflammatory perturbations amplify vascular injury, resulting in disproportionately higher rates of disease burden and mortality relative to the general population **[6].** The lipid pattern observed in CKD patients demonstrates characteristic alterations. Moderate hypertriglyceridemia is frequently observed, particularly in ND-CKD and end-stage renal disease **[7].** Diminished activity of lipolytic enzymes like lipoprotein lipase and hepatic lipase contributes to impaired degradation of triglyceride-rich lipoproteins such as very-low-density lipoprotein (VLDL) and intermediate-density lipoprotein (IDL). As a consequence, accumulation of triglyceride-rich apolipoprotein B-containing particles occurs. HDL cholesterol is often reduced due to decreased synthesis of apolipoprotein A-I and impaired activity of lecithin-cholesterol acyltransferase, resulting in defective reverse cholesterol transport **[7].** Although LDL cholesterol may remain within reference limits in some CKD patients, prolonged particle residence time and qualitative modification, including carbamylation, may enhance atherogenic potential. Chronic inflammation and oxidative stress, common in uremic states, further alter lipoprotein composition and vascular risk **[7].** D-CKD represents a distinct metabolic state in which regular dialysis may partially influence circulating lipid concentrations through extracorporeal clearance and altered metabolic flux **[7].** However, dialysis does not completely normalize lipid metabolism and systemic inflammatory activation frequently persists. Consequently, dyslipidemia in D-CKD may show quantitative attenuation of certain lipid elevations while qualitative abnormalities, particularly low HDL states, remain prevalent. Post-renal transplant recipients represent another unique metabolic subgroup. While improvement in renal function following transplantation may theoretically correct uremia-associated lipid disturbances, immunosuppressive agents such as calcineurin inhibitors and corticosteroids significantly influence lipid metabolism. These agents stimulate hepatic lipogenesis, increase VLDL synthesis, suppress LDL receptor activity and may induce insulin resistance, thereby promoting triglyceride-rich and mixed hyperlipidemic patterns. Elevated lipoprotein (a) has also been linked to cardiovascular risk in renal populations, further reinforcing the multifactorial lipid-mediated risk profile in CKD **[4].** Dyslipidemia in CKD is highly prevalent but not universal. Multiple patterns of dyslipidemia have been described in renal populations. In the present study, dyslipidemia was operationally classified into seven clinically defined patterns based on established lipid thresholds: isolated hypercholesterolemia, isolated hypertriglyceridemia and combined/mixed hyperlipidemia, low HDL, hypercholesterolemia with low HDL, hypertriglyceridemia with low HDL and mixed hyperlipidemia with low HDL. Such combined lipid abnormalities are commonly described in CKD dyslipidemia literature **[8, 9].** Recognition of these patterns is clinically relevant, as different combinations of lipid abnormalities may confer varying cardiovascular risk profiles **[3, 7].** Therefore, it is of interest to report the prevalence and seven-pattern spectrum of dyslipidemia among ND-CKD, D-CKD and post-renal transplant recipients.

Methodology:
An analytical cross-sectional study was conducted at a tertiary-level hospital in the Eastern region of India between March 2015 and March 2017. The sample population consisted of adult patients with CKD according to established clinical criteria, defined as a structural or functional kidney abnormality ongoing

for greater than three months **[5].** The sample size was determined based on the comparison of two independent means using previously published triglyceride data from CKD populations **[10].** The mean triglyceride levels in ND-CKD and hemodialysis patients were reported as 209.80 ± 32.43 mg/dl and 195.42 ± 19.13 mg/dL respectively **[10].** Using these values, the pooled standard deviation was 29.96 and the calculated effect size (Cohen's d) was 0.48. Based on an effect size of 0.48, assuming 80% statistical power and a significance level of α = 0.05, the minimum sample size required for the study was 32 participants per group. A total of 96 participants were enrolled and equally classified into three groups based on treatment modality at the time of enrollment: dialysis-dependent CKD (D-CKD), nondialysis-dependent CKD (ND-CKD) and post-renal-transplant recipients (n = 32 in each group). Inclusion criteria consisted of adults aged ≥ 18 years with confirmed CKD across the defined modalities as defined by KDIGO criteria **[5].** D-CKD included patients diagnosed with end-stage renal disease receiving maintenance hemodialysis therapy for a period of no less than three months prior to enrollment **[5, 7].** ND-CKD included patients diagnosed with CKD (eGFR <60 mL/min/1.73 m^2) who were managed conservatively and had not begun renal replacement therapy **[5].** Post-renal transplant recipients included patients who had received renal transplantation at least six months prior to enrollment to ensure stabilization of graft function and immunosuppressive therapy **[7].** Exclusion criteria were implemented to reduce secondary influences on lipid metabolism. Patients treated with lipid-lowering drugs during the preceding six months were excluded to avoid pharmacological confounding. Patients receiving sevelamer therapy were omitted from the analysis as sevelamer is well known to reduce cholesterol. Those patients who are on prednisolone dose of more than 10 mg per day (nephrotic syndrome or immediate post-transplant) were also excluded. Individuals with acute infections, active inflammatory disorders, chronic liver disease, malignancy, recent cardiovascular events or those unwilling to provide informed consent were excluded. Participants who were pregnant were excluded from the study sample. These criteria were implemented to ensure that observed lipid abnormalities were primarily attributable to CKD status and treatment modality. Demographic and clinical characteristics such as age, gender, and CKD duration and dialysis status and transplant history were documented using case record forms. Participants' venous blood samples were collected after a 12-hour overnight fast under standardized aseptic conditions. Serum TC was measured using the cholesterol oxidase peroxidase (CHOD-PAP) method. Triglyceride levels were estimated using the glycerol-3-phosphate oxidase (GPO) method. LDL and HDL cholesterol levels were estimated using direct enzymatic methods. All assays were performed on an automated biochemistry analyzer, calibrated according to manufacturer specifications. Routine Internal quality control assurance procedures were followed throughout the study period to ensure analytical reliability. Dyslipidemia was defined using established lipid thresholds consistent with cholesterol management guidelines, including total cholesterol ≥ 200mg/dl, LDL cholesterol ≥ 100 mg/dl, triglycerides ≥ 150 mg/dl and HDL cholesterol < 40 mg/dl **[2].** Based on biochemical parameters, dyslipidemia was operationally classified into seven clinically defined patterns: isolated hypercholesterolemia, isolated hypertriglyceridemia, combined/mixed hyperlipidemia, low HDL, hypercholesterolemia with low HDL, hypertriglyceridemia with low HDL and mixed hyperlipidemia with low HDL. Statistical analysis was performed using Epi Info software version 3.5.3. Continuous variables were presented as mean ± standard deviation and compared across groups using one-way analysis of variance (ANOVA). Categorical variables were expressed as proportions and compared using chi-square testing. A two-tailed p-value <0.05 was deemed statistically significant. The protocol of the study was evaluated and approved by the Institutional Ethics Committee (Ref No: IEC/DNB/2016/02/07) before initiation. Ethical clearance was granted in accordance with institutional research guidelines involving human participants. Each participant provided written informed consent before enrollment. Patient information was kept confidential throughout the study and data were anonymized prior to statistical analysis. The study followed the ethical principles stated in the 'Declaration of Helsinki'.

Table 1: Baseline characteristics of CKD patients by treatment modality (N = 96)

Variable	D-CKD[a] (n = 32)	ND-CKD[b] (n = 32)	Post-transplant (n = 32)	P value Test statistic
Age (Mean ± SD), years	52.67 ± 15.97	60.43 ± 15.23	46.68 ± 13.52	ANOVA: F(2,93)=6.81; p=0.002 χ^2 = 20.45
Median age, years	57.5	62.0	46.0	
Age range, years	22-81	29-86	18-72	
Male, n (%)	20 (62.5%)	23 (71.9%)	21 (65.6%)	0.71 χ^2 = 0.66
Female, n (%)	12 (37.5%)	9 (28.1%)	11 (34.4%)	

[a] *Dialysis dependent CKD*
[b] *Nondialysis dependent CKD*
ANOVA for age comparison; Chi-square test χ^2 for gender distribution.
No statistically significant baseline difference observed.

Table 2: Prevalence of Dyslipidemia Across CKD Modalities (N = 96)

Group	Dyslipidemia Present n (%)	Dyslipidemia Absent n (%)
D-CKD[a]	25 (78.1%)	7 (21.9%)
ND-CKD[b]	28 (87.5%)	4 (12.5%)
Post-transplant	28 (87.5%)	4 (12.5%)
Total	81 (84.4%)	15 (15.6%)
χ^2 statistic (Chi-Square test)	χ^2 = 1.42	P = 0.49 (NS)

All lipid values are expressed in mg/dL unless otherwise stated.
[a] *Dialysis dependent CKD*
[b] *Nondialysis dependent CKD*
Definition: Dyslipidemia = ≥1 abnormal lipid parameter (TC ≥ 200 mg/dL; LDL ≥ 100mg/dL; TG ≥ 150 mg/dL; HDL <40mg/dl)
χ^2 = 1.42; p = 0.49 (Not Significant)

Table 3: Comparison of Lipid Parameter Abnormalities (N = 96)

Parameter	D-CKD[a] n (%)	ND-CKD[b] n (%)	Post-transplant n (%)	χ^2	P-value
Hyperlipidemia	16 (50.0%)	24 (75.0%)	24 (75.0%)	6.00	0.050

LDL ≥ 100mg/dL	11 (34.4%)	10 (31.3%)	17 (53.1%)	3.75	0.154
Total Cholesterol ≥ 200mg/dL	8 (25.0%)	6 (18.8%)	10 (31.3%)	1.33	0.513
HDL < 40mg/dL	17 (53.1%)	17 (53.1%)	19 (59.4%)	0.34	0.845

All lipid values are expressed in mg/dL unless otherwise stated.
[a] Dialysis dependent CKD
[b] Nondialysis dependent CKD
Chi-square test χ^2 for comparison of lipid parameter abnormalities
P < 0.05 considered significant

Table 4: Distribution of Primary Hyperlipidemia Patterns (N = 96)

Parameter	D-CKD[a] n (%)	ND-CKD[b] n (%)	Post-transplant n (%)	Total
Hypercholesterolemia	3 (25.0%)	6 (50.0%)	3 (25.0%)	12
Hypertriglyceridemia	5 (25.0%)	9 (45.0%)	6 (30.0%)	20
Mixed Hyperlipidemia	8 (25.0%)	9 (28.1%)	15 (46.9%)	32
No Hyperlipidemia	16 (50.0%)	8 (25.0%)	8 (25.0%)	32
Chi-Square test statistic				χ^2 = 9.48

All lipid values are expressed in mg/dL unless otherwise stated.
[a] Dialysis dependent CKD
[b] Nondialysis dependent CKD
χ^2 = 9.48; p = 0.14 (Not Significant)

Results and Discussion:

The study results demonstrate that dyslipidemia remains highly prevalent across CKD treatment modalities. Age differed across modalities based on the reported group summaries (ANOVA p=0.002), whereas sex distribution was similar (p=0.71); age is therefore a potential confounder **(Table 1).** Equal group sizes facilitated comparisons, but the significant age imbalance means unadjusted modality associations should not be interpreted as independent of demographic confounding. Overall, dyslipidemia was present in 81 of 96 patients (84.4%) **(Table 2).** The prevalence was 78.1% (25/32) in D-CKD, 87.5% (28/32) in ND-CKD and 87.5% (28/32) in post-renal transplant recipients. Although the proportion was numerically higher in ND and post-transplant groups compared with D-CKD, with no statistically significant difference (χ^2 = 1.42; p = 0.49), indicating that dyslipidemia remains widely prevalent among all CKD modalities. This finding is consistent with prior large CKD cohort studies demonstrating a high prevalence of dyslipidemia exceeding 70-80% in renal populations **[5, 11 and 12].** The persistence of lipid abnormalities across treatment states reinforces the established cardiovascular vulnerability of CKD populations **[6].** In contrast, hyperlipidemia alone showed borderline variation between the three groups (Pearson χ^2=6.00, p=0.050), affecting 50% of D-CKD patients compared with 75% in both ND-CKD and post-transplant groups **(Table 3).** LDL cholesterol elevation was numerically more common in post-transplant recipients (53.1%) than D-CKD (34.4%) or ND-CKD (31.3%) but did not differ significantly overall (three-group p=0.154). Total cholesterol elevation also did not vary significantly (p=0.513). In contrast, low HDL cholesterol remained consistently prevalent across groups without statistically significant variation (p = 0.88). These findings indicate that while the overall prevalence of dyslipidemia remains consistently high, post-transplant recipients had numerically higher LDL prevalence without a statistically significant three-group comparison. The observed LDL enrichment in transplant recipients is biologically plausible. Immunosuppressive agents, especially calcineurin inhibitors and corticosteroids, are known to stimulate hepatic lipogenesis, increase very-low-density lipoprotein (VLDL) production, induce insulin resistance and attenuate LDL receptor activity **[13–15].** These pharmacologic effects may contribute to increased circulating LDL concentrations and mixed hyperlipidemic phenotypes. In contrast, dialysis-dependent CKD demonstrated comparatively lower quantitative hyperlipidemia prevalence, which may partially reflect extracorporeal lipid modulation and altered metabolic flux during hemodialysis, although dialysis does not normalize intrinsic lipoprotein metabolic abnormalities **[6, 8].** Primary hyperlipidemia classification **(Table 4)** further clarified modality associated heterogeneity. Mixed hyperlipidemia was the most prevalent pattern overall (33.3%) and demonstrated numerical enrichment in post-transplant recipients (46.9%) compared with D-CKD (25.0%) and ND-CKD (28.1%). D-CKD exhibited the highest percentage of patients without hyperlipidemia (50.0%). Although the overall association between treatment modality and hyperlipidemia pattern was not statistically significant (χ^2 = 9.48; p = 0.14), the distribution pattern suggests modality-associated clustering of mixed phenotypes in transplant recipients. These phenotype patterns are in line with known CKD-associated metabolic alterations. Reduced hepatic lipase and lipoprotein lipase activity impair triglyceride clearance, leading to accumulation of very-low-density and intermediate-density lipoproteins **[16, 17].** Impaired HDL maturation further compromises reverse cholesterol transport **[11].** Qualitative lipoprotein abnormalities may increase atherogenic potential independent of absolute LDL concentration **[3, 4].** Such mechanisms contribute to the elevated cardiovascular morbidity observed in CKD populations **[5, 11, 12 and 18].** The significant variation observed in quantitative lipid parameters, particularly LDL and total cholesterol, highlights the importance of treatment-modality-specific lipid surveillance. Although LDL-lowering strategies have demonstrated cardiovascular benefit in CKD populations **[19, 20],** residual cardiovascular risk persists, likely attributable to triglyceride-rich lipoproteins, HDL dysfunction and qualitative lipoprotein alterations not captured by conventional lipid thresholds **[18, 21-24].** The present findings support a phenotype-based perspective of dyslipidemia evaluation across the CKD continuum. Other observational studies in patients with chronic kidney disease have described frequent lipid abnormalities, supporting the importance of the present comparisons by treatment modality while emphasizing the need for correctly calculated between-group statistics. **[25, 26 and 27]**

Conclusion:

Dyslipidemia was frequent in all three CKD treatment groups. Overall dyslipidemia prevalence did not differ significantly across modalities. Age imbalance and recalculated lipid comparisons limit causal interpretation.

References:

[1] Grundy SM *et al. J Am Coll Cardiol.* 2019 **73:**e285. [PMID: 30423393]
[2] https://pubmed.ncbi.nlm.nih.gov/11368702/
[3] Vaziri ND. *Am J Physiol Renal Physiol.* 2006 **290:**F262. [PMID: 16403839]
[4] Tsimikas S *et al. N Engl J Med.* 2005 **353:**46. [PMID: 16000355]
[5] Levey AS *et al. Kidney Int.* 2005 **67:**2089. [PMID: 15882252]
[6] Go AS *et al. N Engl J Med* 2004 **351:**1296. [PMID: 15385656]
[7] Tsimihodimos V *et al. Open Cardiovasc Med J.* 2011 **5:**41. [PMID: 21643500]
[8] Attman PO & Samuelsson O. *Curr Opin Lipidol.* 2009 **20:**293. [PMID: 19512921]
[9] Tonelli & Wanner C. *M Kidney Int.* 2014 **85:**1303. [PMID: 24323134]
[10] Raju DSSK. *J Clin Res Bioeth.* 2013. [DOI: 10.4172/2155-9627.1000143]
[11] Li Y. *Plos One*. 2018 **13:**e0205709. [PMID: 30325950]
[12] Ferro CJ *et al. Nat Rev Nephro.l* 2018 **14:**727. [PMID: 30361677]
[13] Kwan BCH. *J Am Soc Nephrol JASN.* 2007 **18:**1246. [PMID: 17360943]
[14] Hopewell JC *et al. J Lipid Res.* 2018 **59:**577. [PMID: 29378781]
[15] Artz MA *et al. J Am Soc Nephrol JASN.* 2003 **14:**1880. [PMID:12819249]
[16] Fellström BC *et al. N Engl J Med.* 2009 **360:**1395. [PMID: 19332456]
[17] Deleuze S *et al. Transplant Proc.* 2006 **38:2311**. [PMID: 16980075]
[18] Shoji T *et al. Am J Kidney Dis Off J Natl Kidney Found.* 2001 **38:**S30. [PMID: 11576918]
[19] Wanner C *et al. N Engl J Med.* 2005 **353:**238. [PMID: 16034009]
[20] Stenvinkel P *et al. Kidney Int.* 1999 **55:**1899 [PMID: 10231453]
[21] Baigent C *et al. Lancet.* 2011 **377:**2181. [PMID: 21663949]
[22]Minutolo R *et al. Curr Atheroscler Rep*. 2026 **28:**68. [PMID: 42400804]
[23]Bahardoust M *et al. Endocrinol Diabetes Metab.* 2026 **9:**e70161 [PMID: 41572605]
[24]Ábel T *et al. Eur J Clin Pharmacol.* 2026 **82:**37. [PMID: 41546841]
[25]Pyadala NB *et al. Int J Adv Biochem Res*. 2023 **7**:36. [DOI: 10.33545/26174693.2023.v7.i2a.213]
[26]Singh Y *et al. J Popul Ther Clin Pharmacol*. 2025. [DOI: 10.53555/3arqqk91]
[27]Patel A *et al. Eur J Cardiovasc Med.* 2025. [DOI: 10.61336/ejcm/25-04-147]

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