

ASSESSMENT OF THE FREQUENCY AND PATTERN OF DYSLIPIDEMIA IN PATIENTS WITH EARLY-STAGE CHRONIC KIDNEY DISEASE

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ABSTRACT

Objective: To determine the frequency and pattern of dyslipidemia in patients with early-stage chronic kidney disease.

Study Design, Place and Duration: Descriptive cross-sectional study; Medicine Outpatient Department, Lahore General Hospital, Lahore, Pakistan; December 2025 to May 2026.

Materials and Methods: The supplied dataset comprised 248 adults aged 18–70 years with CKD stages G1–G3a. Fasting total cholesterol, triglycerides, LDL-C and HDL-C were assessed using the prespecified thresholds. Dyslipidemia was defined by at least one abnormal lipid component. Frequencies, Wilson 95% confidence intervals, chi-square tests, Kruskal–Wallis tests and multivariable logistic regression were calculated. The regression included age, sex, BMI, diabetes, hypertension, current smoking and CKD stage.

Results: Dyslipidemia was identified in 157/248 patients (63.3%; 95% CI 57.1–69.1%). Hypertriglyceridemia was present in 104 (41.9%) and low HDL-C in 95 (38.3%); mixed dyslipidemia occurred in 61 (24.6%). Prevalence increased from 31/60 (51.7%) in G1 to 74/118 (62.7%) in G2 and 52/70 (74.3%) in G3a ($\chi^2=7.15$; $p=0.028$). Relative to G1, adjusted odds were higher in G2 (aOR 2.14, 95% CI 1.06–4.29) and G3a (aOR 4.44, 95% CI 1.87–10.52). Diabetes (aOR 1.98; $p=0.021$) and hypertension (aOR 2.03; $p=0.013$) were also associated with dyslipidemia.

Conclusion: Dyslipidemia was common in early-stage CKD and more frequent at lower eGFR stages. Elevated triglycerides and low HDL-C were the most frequent lipid abnormalities. Diabetes and hypertension were associated with dyslipidemia after adjustment for the measured clinical factors.

KEYWORDS: Chronic kidney disease; dyslipidemia; early-stage CKD; triglycerides; HDL cholesterol; renal function.

INTRODUCTION

Chronic kidney disease (CKD) affects nearly 9% to 13% of adults worldwide—around 700 million people—with a higher prevalence among women and older age groups. ^{1,2} The burden is increasing globally, largely due to rising life expectancy and population growth, and is especially pronounced in low- and middle-income countries. South Asia, including Pakistan, reports some of the highest figures, with a pooled prevalence of 14%. ^{2,3} In Pakistan, studies estimate the CKD prevalence between 12.5% and 21.2%, with some high-risk populations reaching up to 29.9%. ¹ Among individuals aged over 50, the prevalence can climb to 43.6%. ¹ Gender-based trends remain variable, with different studies showing both male and female predominance. These patterns reflect the complex epidemiology of CKD in the region. ^{4,5} Importantly, metabolic complications such as dyslipidemia often remain unnoticed in early CKD, despite their known role in accelerating kidney damage and increasing cardiovascular risk, making early identification and intervention crucial.

Chronic kidney disease (CKD) leads to significant disruptions in lipid metabolism that accelerate both cardiovascular disease and kidney damage. Impaired fatty acid oxidation and mitochondrial dysfunction in CKD result in lipid buildup, oxidative stress, and inflammation in renal tissue. Common lipid abnormalities include elevated triglycerides, low HDL levels, and an increase in small, dense LDL particles, all of which are linked to higher cardiovascular risk. Changes in lipoprotein structure and function—such as altered composition and poor clearance—contribute to endothelial injury. Elevated free fatty acids and atypical sphingolipids add to lipotoxicity and fibrosis, worsening renal function. ^{6–8}

Liang et al. (2020) found a significant association between elevated lipid levels and incident CKD in a large Chinese cohort with normal baseline renal function. Higher triglycerides, LDL, and total cholesterol were linked to increased CKD risk, with statistically significant differences across lipid quartiles ($p < 0.001$). The study supports

dyslipidemia as an independent predictor of early CKD. ⁹ Garg et al. (2015) conducted a cross-sectional study of 100 CKD patients, reporting dyslipidemia in 97% with high TG, 61% with TC >200 mg/dL, 81% with LDL-C $\geq$ 100 mg/dL, and 87% with HDL-C <40 mg/dL. Mean lipid values were: TG 250.91 $\pm$ 56.15 mg/dL, TC 205.33 $\pm$ 27.02, LDL-C 120.29 $\pm$ 21.99, HDL-C 34.86 $\pm$ 3.99, VLDL-C 50.18 $\pm$ 11.23. Stage-wise TG $\geq$ 200 mg/dL increased from 0% (stage 1) to 92.9% (stage 5). Statistically significant increases were noted in TG ($p < 0.001$), TC ($p < 0.05$), VLDL-C ($p < 0.001$), and TC/HDL ($p < 0.05$). ¹⁰ Shumaila et al. (2020) studied 137 hemodialysis patients aged 20–60 years; dyslipidemia was observed in 63.5%, highest in stage I (73.7%), then stage III (69.2%), II (61.5%), IV (56.3%), V (50%), with no significant stage-wise association ($p = 0.745$). It was more common in females (73.8%) than males (55.3%; $p = 0.025$) and higher in older age groups (88.7% vs. 42.7%; $p < 0.0001$). ¹¹ Sah and Adhikary (2020) conducted a cross-sectional observational study in Nepal involving 136 patients to evaluate the prevalence of dyslipidemia across different stages of chronic kidney disease (CKD). The overall prevalence of dyslipidemia was 49.3% (67 out of 136), with a 95% confidence interval of 40.59–57.40%. When stratified by CKD stage, dyslipidemia was observed in 50.0% of patients in Stage 1/2 (29/58), 56.1% in Stage 3 (23/41), 50.0% in Stage 4 (14/28), and 11.1% in Stage 5 (1/9), though the association was not statistically significant ($P = 0.11$). Hypertension was significantly more prevalent among dyslipidemic patients (41.79%; 28/67) compared to non-dyslipidemic individuals (24.63%; 17/69), with a P-value of 0.034. Diabetes mellitus was more common in dyslipidemic patients (83.58%; 56/67) than in non-dyslipidemic ones (73.91%; 51/69), though this difference did not reach statistical significance ($P = 0.16$). ¹²

The rationale for this study stems from a critical gap in existing literature, wherein most previous investigations have predominantly focused on evaluating dyslipidemia in advanced stages of chronic kidney disease (CKD). In contrast, early-stage CKD often remains under-investigated with regard to lipid abnormalities, largely due to the absence of routine screening protocols at this stage. This oversight is clinically significant, as dyslipidemia is not only a common metabolic disturbance in CKD but also a key modifiable risk factor contributing to both disease progression and elevated cardiovascular morbidity and mortality. Timely detection and management of lipid abnormalities during early CKD could potentially delay the deterioration of renal function and reduce cardiovascular events. Furthermore, existing literature presents inconsistent findings regarding the correlation between CKD stage and dyslipidemia prevalence—some studies have demonstrated a rising trend of lipid disturbances with advancing CKD, whereas others have failed to establish a significant association. Without well-documented local data on the frequency and pattern of dyslipidemia in early CKD, it remains challenging to justify or implement evidence-based screening guidelines in clinical practice.

MATERIALS AND METHODS

This descriptive cross-sectional study was based on the protocol for the Medicine Outpatient Department, Lahore General Hospital, Lahore, Pakistan, with the study period specified as December 2025 to May 2026. The sample size of 248 was calculated in OpenEpi using a previously reported dyslipidemia frequency of 63.5%, 95% confidence level and 6% absolute precision. The study followed a non-probability consecutive sampling plan. Patients aged 18–70 years with chronic kidney disease of at least three months' duration and clinically stable stages G1, G2 or G3a were eligible. Stages were classified from eGFR as G1 $\geq$ 90, G2 60–89 and G3a 45–59 mL/min/1.73 m²; G1 and G2 required evidence of kidney damage. CKD staging was interpreted according to current KDIGO definitions.¹³

Patients receiving lipid-lowering medicines, those with known familial dyslipidemia, pregnancy or lactation, clinically significant liver disease, thyroid dysfunction, or recent immunosuppressive treatment were excluded according to the protocol. The study proforma included age, sex, BMI, CKD duration, serum creatinine, eGFR, albuminuria, renal ultrasonography findings, hypertension, diabetes, cardiovascular disease, smoking status, medication use and fasting lipid concentrations. The supplied analytical file contained one complete record per patient, with the primary outcome recorded for all 248 participants.

After an overnight fast of approximately 12 hours, blood samples were specified for total cholesterol, triglycerides, LDL-C and HDL-C measurement by enzymatic colorimetric methods in the institutional laboratory. Dyslipidemia was recorded if total cholesterol exceeded 200 mg/dL, triglycerides exceeded 150 mg/dL, LDL-C exceeded 130 mg/dL or HDL-C was below 40 mg/dL in men or below 50 mg/dL in women. Individual lipid abnormalities were not mutually exclusive. The pattern variable distinguished isolated hypercholesterolemia, isolated hypertriglyceridemia, isolated low HDL-C and mixed dyslipidemia involving two or more abnormalities. The workbook categories were checked against the recorded lipid values and showed complete agreement with the prespecified cut-offs.

The protocol specified IBM SPSS Statistics version 26; the supplied spreadsheet was analysed using equivalent statistical procedures in Python with SciPy and statsmodels. The Shapiro–Wilk test was used to assess distribution. Continuous data were reported as mean $\pm$ standard deviation or median (interquartile range), as appropriate. Categorical data were expressed as frequency and percentage, and Wilson 95% confidence intervals were calculated for the main prevalence estimates. Pearson's chi-square test compared CKD categories and clinical groups; lipid concentrations across CKD stages were compared by Kruskal–Wallis tests because distributions were non-normal. Binary logistic regression was fitted for dyslipidemia using age, male sex, BMI, diabetes, hypertension, current smoking and G2/G3a stage (G1 as reference). Adjusted odds ratios (aORs) with 95% confidence intervals were calculated. Tests were two-sided and $p < 0.05$ was considered statistically significant.

RESULTS

The dataset contained 248 complete patient records. The median age was 51.0 (44.0–57.2) years, and 132 (53.2%) were men. Median CKD duration was 23.0 (16.0–31.2) months and mean BMI was 28.64 ± 4.25 kg/m². Stage G2 accounted for 118 (47.6%) patients, followed by G3a in 70 (28.2%) and G1 in 60 (24.2%). Hypertension was recorded in 139 (56.0%) and diabetes in 106 (42.7%). Baseline characteristics are presented in Table 1.

Table 1. Demographic, clinical and lipid characteristics of the study population (n=248)

Characteristic	Result
Total patients	248 (100%)
Age, years, median (IQR)	51.0 (44.0–57.2)
Male / female	132 (53.2%) / 116 (46.8%)
BMI, kg/m ² , mean $\pm$ SD	28.64 ± 4.25
CKD duration, months, median (IQR)	23.0 (16.0–31.2)
Serum creatinine, mg/dL, median (IQR)	1.6 (1.3–2.0)
eGFR, mL/min/1.73 m ² , median (IQR)	72.1 (56.8–88.8)
CKD G1 / G2 / G3a	60 (24.2%) / 118 (47.6%) / 70 (28.2%)
Albuminuria	181 (73.0%)
Abnormal renal ultrasound	78 (31.5%)
Hypertension	139 (56.0%)
Diabetes mellitus	106 (42.7%)
Cardiovascular disease	49 (19.8%)
Current smoker / former smoker	45 (18.1%) / 35 (14.1%)
Total cholesterol, mg/dL, median (IQR)	192.6 (186.3–199.8)
Triglycerides, mg/dL, median (IQR)	143.1 (120.6–240.0)
HDL-C, mg/dL, median (IQR)	42.4 (36.3–50.5)
LDL-C, mg/dL, median (IQR)	123.2 (118.9–128.2)

Note. Quantitative variables are mean $\pm$ SD or median (IQR). The denominator for categorical data is 248 unless stated otherwise.

Dyslipidemia was recorded in 157 patients (63.3%; Wilson 95% CI 57.1–69.1%). The most frequent individual abnormalities were triglycerides >150 mg/dL in 104 (41.9%) and low HDL-C in 95 (38.3%). Total cholesterol >200 mg/dL was present in 61 (24.6%) and LDL-C >130 mg/dL in 36 (14.5%). Mixed dyslipidemia was found in 61 (24.6%), isolated hypertriglyceridemia in 43 (17.3%), isolated low HDL-C in 34 (13.7%) and isolated hypercholesterolemia in 19 (7.7%). Table 2 describes the pattern distribution and lipid-specific frequencies.

Table 2. Frequency and pattern of dyslipidemia (n=248)

Lipid abnormality or pattern	n	%	95% CI, %
TC >200 mg/dL	61	24.6	19.7–30.3
TG >150 mg/dL	104	41.9	36.0–48.2
LDL-C >130 mg/dL	36	14.5	10.7–19.4
Low HDL-C	95	38.3	32.5–44.5
Any dyslipidemia	157	63.3	57.1–69.1
Pattern classification (mutually exclusive)			
Isolated Hypercholesterolemia	19	7.7	—
Isolated Hypertriglyceridemia	43	17.3	—
Isolated Low HDL-C	34	13.7	—
Mixed Dyslipidemia	61	24.6	—
No dyslipidemia	91	36.7	—

Note. Individual abnormalities overlap; one patient may have more than one. The five pattern categories are mutually exclusive. CI: confidence interval; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol.

Dyslipidemia was present in 31/60 (51.7%) of G1 patients, 74/118 (62.7%) of G2 and 52/70 (74.3%) of G3a ($\chi^2=7.15$, df=2; p=0.028). Rates of individual lipid abnormalities did not differ significantly by CKD stage, although triglycerides tended to be higher and HDL-C lower at more advanced stages. The stage-specific findings are presented in Table 3.

Table 3. Dyslipidemia and lipid profile by CKD stage

Variable	G1 (n=60)	G2 (n=118)	G3a (n=70)	Test	p
Any dyslipidemia	31 (51.7%)	74 (62.7%)	52 (74.3%)	7.15	0.028
TG >150 mg/dL	20 (33.3%)	54 (45.8%)	30 (42.9%)	2.56	0.278

Low HDL-C	18 (30.0%)	48 (40.7%)	29 (41.4%)	2.32	0.313
TC >200 mg/dL	12 (20.0%)	30 (25.4%)	19 (27.1%)	0.97	0.615
LDL-C >130 mg/dL	6 (10.0%)	18 (15.3%)	12 (17.1%)	1.43	0.490
Total cholesterol, mg/dL	191.2 (184.1–198.1)	192.3 (186.9–215.2)	194.4 (187.6–224.1)	H=2.48	0.290
Triglycerides, mg/dL	129.4 (118.4–202.2)	145.2 (124.1–260.5)	145.6 (120.4–237.2)	H=5.27	0.072
HDL-C, mg/dL	42.5 (39.9–51.2)	42.6 (35.0–50.6)	41.8 (35.1–43.7)	H=5.32	0.070
LDL-C, mg/dL	123.4 (120.2–127.4)	123.2 (118.8–128.3)	122.8 (118.2–128.6)	H=0.50	0.780

Note. Categorical rows use *n* (column percentage), Pearson χ^2 with *df*=2. Continuous lipid rows use median (IQR) and Kruskal–Wallis *H* tests. The first row is the primary stage-wise comparison.

At the unadjusted level, dyslipidemia was associated with CKD stage ($p=0.028$), diabetes ($p=0.008$) and hypertension ($p=0.017$). In the multivariable model, CKD G2 (aOR 2.14, 95% CI 1.06–4.29; $p=0.033$) and G3a (aOR 4.44, 95% CI 1.87–10.52; $p<0.001$) remained associated with dyslipidemia relative to G1. Diabetes (aOR 1.98; $p=0.021$) and hypertension (aOR 2.03; $p=0.013$) also remained associated. Age had a small inverse adjusted association (aOR 0.97 per year; $p=0.031$). Sex, BMI and current smoking were not independently associated. Results are shown in Table 4.

Table 4. Adjusted associations with the presence of dyslipidemia

Variable	aOR	95% CI	p-value
Age (per additional year)	0.97	0.94–1.00	0.031
Male sex (vs female)	0.69	0.39–1.21	0.198
BMI (per kg/m ²)	1.03	0.97–1.10	0.325
Diabetes mellitus	1.98	1.11–3.54	0.021
Hypertension	2.03	1.16–3.54	0.013
Current smoker (vs other)	1.71	0.80–3.65	0.164
CKD G2 (vs G1)	2.14	1.06–4.29	0.033
CKD G3a (vs G1)	4.44	1.87–10.52	<0.001

Note. Binary logistic regression, $n=248$, 157 with dyslipidemia; all listed variables entered simultaneously. The global likelihood-ratio test $p=0.0003$; pseudo- $R^2=0.089$. aOR: adjusted odds ratio; CI: confidence interval.

DISCUSSION

Dyslipidemia was found in 63.3% of adults with CKD stages G1–G3a in this study. It was present in approximately half of the patients in G1 and nearly three-quarters of those in G3a. Hypertriglyceridemia and low HDL-C were the leading lipid abnormalities, while almost one-quarter of the sample had mixed dyslipidemia. Diabetes and hypertension were associated with dyslipidemia after adjustment for age, sex, BMI, smoking and CKD stage. These findings describe a substantial lipid burden in patients who had not yet reached advanced CKD.

The observed prevalence was similar to the 63.5% reported by Shumaila et al., although their report concerned a different clinical population and should not be treated as an estimate restricted to early CKD. The figure was higher than the 49.3% reported by Sah and Adhikary in 136 non-dialysis patients in Nepal. Garg et al. reported dyslipidemia in 97% of patients across a broader spectrum of CKD, including advanced stages. Direct comparison is limited by differences in stage distribution, lipid thresholds, diabetes prevalence and exclusion of patients already receiving lipid-lowering treatment. The current estimate is specific to stable patients with G1–G3a disease who met the protocol's eligibility criteria.^{11,12,10}

Raised triglycerides affected 41.9% and low HDL-C 38.3% of patients, whereas LDL-C exceeded 130 mg/dL in 14.5%. This pattern agrees with descriptions of CKD-associated dyslipidemia, in which impaired clearance of triglyceride-rich particles and changes in HDL composition may occur without a comparable increase in LDL cholesterol concentration. Suh and Kim described hypertriglyceridemia and altered HDL metabolism as common findings in CKD, and the review by Barbagallo et al. outlined similar abnormalities in lipoprotein turnover. The present study measured routine lipid concentrations, not particle size, apolipoproteins or lipoprotein function; the mechanisms cannot be tested directly with these data.^{14,6}

The frequency of any dyslipidemia increased from 51.7% in G1 to 74.3% in G3a. Compared with G1, both G2 and G3a remained associated with the outcome after adjustment for the prespecified clinical factors. Nevertheless, the individual triglyceride, HDL-C, LDL-C and total-cholesterol thresholds did not show significant stage-wise differences when examined separately. A composite outcome based on any abnormality can increase across stages even when changes in separate components are less precise. Liang et al. linked atherogenic lipid measures with later CKD development in a population initially free of kidney disease; that longitudinal relationship differs from the cross-sectional comparison in this article. Progressive metabolic changes in CKD are also described in recent reviews, but the present data do not establish whether declining filtration preceded dyslipidemia.^{9,15}

Diabetes and hypertension were each associated with approximately doubled adjusted odds of dyslipidemia. Both conditions are common in CKD and can influence lipid profiles independently of kidney function. Age showed a small inverse association after adjustment, despite older participants being more frequent in lower eGFR categories.

This conditional result should not be read as evidence that ageing protects against dyslipidemia; correlated age, CKD stage and comorbidity variables can change the direction of model coefficients. BMI and current smoking were not statistically significant after adjustment. The model therefore supports an association with the measured factors but does not identify causal effects.¹⁶

The clinical relevance of detecting lipid abnormalities early is the opportunity to assess cardiovascular risk before kidney function deteriorates further. KDIGO guidance recommends a lipid profile as part of CKD evaluation and bases lipid-lowering decisions on age, kidney function and overall cardiovascular risk rather than a single abnormal lipid component. In the SHARP trial, simvastatin plus ezetimibe reduced major atherosclerotic events in patients with CKD, although that trial did not evaluate whether lipid screening delays CKD progression. Contemporary reviews likewise stress that the cardiovascular evidence for LDL-lowering treatment should not be confused with proof that correcting every lipid abnormality improves renal outcomes. In this study, treatment effects were not measured because participants receiving lipid-lowering medicines were excluded.^{17,18,19}

The study used the planned sample size, applied uniform lipid thresholds and included patients across three early CKD stages. All 248 records had the primary lipid outcome and the covariates needed for the adjusted analysis. Its main limitations were the single-hospital design, consecutive rather than probability sampling, absence of repeat lipid testing, and restriction to patients not taking lipid-lowering treatment. Albuminuria was recorded as a yes/no variable rather than a quantitative urinary albumin-to-creatinine ratio, and diet, physical activity and glycaemic control could not be fully addressed. CKD stage was derived from recorded eGFR, and variation in kidney function over time was not assessed. The adjusted model used the available covariates, but residual confounding is possible. Because CKD prevalence and its clinical correlates vary between populations, these results should inform local assessment rather than be treated as a population prevalence estimate or evidence of CKD progression. Further studies with repeated kidney and lipid measurements would help examine longitudinal relationships.^{13,20}

CONCLUSION

Dyslipidemia was recorded in 63.3% of patients with CKD stages G1–G3a. Elevated triglycerides and low HDL-C were the most frequent abnormalities, and mixed dyslipidemia affected nearly one-quarter of participants. The frequency of dyslipidemia increased across CKD stages and was associated with diabetes and hypertension after adjustment for measured clinical factors. Fasting lipid assessment in early CKD may help identify patients requiring cardiovascular risk assessment and management. The cross-sectional findings do not establish whether dyslipidemia contributes to subsequent renal decline.

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