# Association of Dyslipidemia & Chronic Kidney Disease Presenting to Tertiary Care Hospital.

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ABSTRACT:

**Objective:** Chronic kidney disease (CKD) is associated with dyslipidemia and cardiovascular risk. Evidence describing lipid abnormalities across CKD stages remains limited. To determine the frequency of LDL-C/HDL-C-defined dyslipidemia and assess its association with CKD stage among patients presenting to a tertiary care hospital.

**Methodology:** This cross-sectional study included 84 patients aged 20–80 years with CKD stages 3–5 admitted to the medical units of Ayub Teaching Hospital, Abbottabad. Patients were enrolled through non-probability consecutive sampling. A fasting lipid profile was measured. The primary dyslipidemia outcome was defined as low-density lipoprotein cholesterol (LDL-C) ≥115 mg/dL and/or high-density lipoprotein cholesterol (HDL-C) ≤40 mg/dL. Hypercholesterolemia and hypertriglyceridemia were assessed separately as secondary lipid abnormalities. Data were analysed using comparative tests and binary logistic regression. A p-value <0.05 was considered significant.

**Results:** LDL-C/HDL-C-defined dyslipidemia was present in 66 patients (78.6%). Its frequency increased from 68.0% in stage 3 and 69.2% in stage 4 to 93.9% in stage 5 CKD (p=0.022). Median LDL-C increased across the stages (p=0.035), while median HDL-C decreased (p=0.001). Patients with dyslipidemia had a lower median estimated glomerular filtration rate than those without dyslipidemia (15.8 versus 26.2 mL/min/1.73 m²; p=0.008). After adjustment for age, gender, diabetes mellitus, and current lipid-lowering therapy, stage 5 CKD remained associated with LDL-C/HDL-C-defined dyslipidemia (adjusted odds ratio 6.20, 95% confidence interval 1.29–29.89; p=0.023).

**Conclusion:** LDL-C/HDL-C-defined dyslipidemia was common and increased with advancing CKD. Regular lipid assessment is particularly important in stage 5 disease.

**Keywords:** Chronic kidney disease; Dialysis; Dyslipidemia; Lipid profile; Renal function

Cite as: Mulk TU, Gillani SYH, Shah S, Zulfiqar A, Ahmed N, Zulfiqar T. Association of Dyslipidemia & Chronic Kidney Disease Presenting to Tertiary Care Hospital. J Muhammad Med Coll. 17(1). May-Oct 2026

## Introduction:

Chronic kidney disease (CKD) is a progressive disorder characterized by persistent abnormalities in kidney structure or function. It causes a gradual decline in renal function and increases the risk of cardiovascular morbidity and premature death. A national analysis of health outcomes in Pakistan identified CKD among the ten leading causes of years of life lost in the country.[1] A recent study from Lahore also found CKD in approximately one-quarter of individuals belonging to a high-risk urban population.[2] These findings indicate that CKD is an important and growing public health concern in Pakistan.

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*Received: July 2026, Revised : August 2026*

*Accepted: October 2026*

Dyslipidemia is a common metabolic abnormality in CKD. Earlier evaluation focused mainly on total cholesterol and low-density lipoprotein cholesterol (LDL-C). More recent evidence has shown that CKD is also associated with elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), accumulation of atherogenic lipoproteins, and impaired HDL function.[3] These abnormalities may appear before kidney failure and often become more pronounced as renal function declines. A study conducted among adults with CKD found that the triglyceride-to-HDL-C ratio was negatively correlated with estimated glomerular filtration rate (eGFR) and was associated with a greater risk of CKD progression.[4]

The relationship between dyslipidemia and CKD may be bidirectional. Reduced renal function alters lipid synthesis, metabolism, and clearance. In contrast, atherogenic lipid abnormalities may promote inflammation, oxidative stress, endothelial injury, and progressive renal damage. A prospective cohort study reported that the combined presence of dyslipidemia and systemic inflammation was associated with a higher risk of developing CKD.[5] These findings support the hypothesis that lipid abnormalities are not merely consequences of CKD but may also contribute to its progression.

The pattern of dyslipidemia also differs according to disease severity and treatment modality. A comparative study reported significantly higher LDL-C, very-low-density lipoprotein cholesterol, and triglyceride levels among patients receiving hemodialysis than among those receiving conservative treatment. HDL-C was significantly lower in the hemodialysis group.[6] A recent Pakistani study of stage 5 CKD found elevated LDL-C in 60.0% of non-dialysis patients and 74.0% of dialysis-dependent patients. Low HDL-

C was also more frequent among dialysis-dependent patients.[7]

Despite this evidence, local data remain limited. Most Pakistani studies have examined the general prevalence of CKD or have focused only on patients with stage 5 disease and hemodialysis.[2] Limited information is available regarding the frequency and pattern of dyslipidemia across CKD stages 3, 4, and 5 among patients admitted to general medical units.[7] Stage-wise assessment may help identify patients who require earlier lipid screening and cardiovascular risk management. It was hypothesized that dyslipidemia would become more frequent with advancing CKD and would be most pronounced in stage 5 and dialysis-treated patients.

## Objective:

The objective of this study was to determine the frequency of LDL-C/HDL-C-defined dyslipidemia and assess its association with CKD stage among patients presenting to a tertiary care hospital.

## Methodology:

A hospital-based cross-sectional study was conducted in Medical Units A, B, C, and D of Ayub Teaching Hospital, Abbottabad. The study was completed over a period of six months dated from 20 September 2025 to 20 March 2026. Ethical approval was obtained from the Institutional MERC (Medical and Ethics Review Committee) of AMTIA (Ayub Medical Teaching Institution, Abbottabad) RC-EA-2025/133. Written informed consent was obtained from every participant before enrolment. The sample size was calculated using the OpenEpi sample-size calculator for estimation of a single population proportion. An anticipated dyslipidemia prevalence of 80% was used, based on a Pakistani study conducted among patients with chronic kidney disease receiving maintenance hemodialysis. At a 95% confidence level and an absolute precision of 9%, the minimum calculated sample size was 76 patients. After adding a 10% allowance for incomplete data and non-response, the final required sample size was increased to 84 patients.[8] Participants were enrolled using a non-probability consecutive sampling technique.

Patients of either gender, aged 20–80 years, who were admitted to Medical Units A, B, C, or D of Ayub Teaching Hospital, Abbottabad, were included. Patients were required to have an established diagnosis of chronic disease of the kidney, defined as an estimated glomerular filtration rate below 60 mL/min/1.73 m² persisting for at least three months. Accordingly, patients with CKD stages 3, 4, and 5 were enrolled. Patients receiving either conservative treatment or dialysis were eligible for inclusion. Patients with acute kidney injury were excluded because an acute decline in renal function did not fulfil the definition of chronic kidney disease. Patients with renal carcinoma were also excluded from the study.

Data were collected by the principal investigator using a predesigned proforma. Demographic information included age, gender, medical record number, residence, monthly income, and socioeconomic status. Weight and height were recorded, and BMI (body mass index) was calculated as weight in kilograms divided by height in metres squared. Smoking status, physical activity, dietary habits, and daily water intake were also documented. Clinical information included the duration, suspected cause, and current stage of CKD. Any documented decline in eGFR and its estimated rate were recorded. Treatment modality was classified as dialysis or conservative management. Treatment adherence was categorized as strict, partial, or non-adherence. Kidney-biopsy status and available biopsy findings were documented. The duration, medication use, and level of control of hypertension, diabetes mellitus, vascular disease, and any previous history of dyslipidemia were also recorded. CKD-related complications, including proteinuria, electrolyte imbalance, edema, and encephalopathy, were documented. Current lipid-lowering therapy, including the drug name, dose, and reported adverse effects, was recorded. Treatment duration and therapy-specific adherence were not available for analysis.

After a 10–12-hour overnight fast, a 3 mL venous blood sample was collected from each participant. In patients receiving maintenance hemodialysis, blood was collected before the scheduled dialysis session and before heparin administration. Serum lipid parameters were analysed on a fully automated Atellica CH 930 clinical chemistry analyser (Siemens Healthineers, USA) in the clinical chemistry laboratory of Ayub Teaching Hospital, Abbottabad. Total cholesterol was measured using an enzymatic cholesterol esterase–oxidase colorimetric method. Triglycerides were measured using an enzymatic lipase and glycerol kinase method. High-density lipoprotein cholesterol was measured using a direct homogeneous selective-detergent assay, while low-density lipoprotein cholesterol was measured using a direct homogeneous enzymatic assay. Calibration and internal quality control were performed according to the manufacturer's instructions and the laboratory's standard operating procedures. Patient results were accepted only when the quality-control values were within the established laboratory limits. The primary outcome was LDL-C/HDL-C-defined dyslipidemia. It was considered present when the LDL-C level was ≥115 mg/dL and/or the HDL-C level was ≤40 mg/dL. Total cholesterol and triglycerides were evaluated separately as secondary lipid abnormalities and were not used to determine the primary dyslipidemia outcome. Hypercholesterolemia was defined as total cholesterol ≥200 mg/dL, while hypertriglyceridemia was defined as triglycerides ≥250 mg/dL. A mixed lipid abnormality was recorded when two or more measured lipid parameters were outside their specified limits. Mixed dyslipidemia was recorded when more than one lipid abnormality was present.

SPSS Statistics version 25.0 were used to enter and analyse the data. All continuous variables were tested for normality using test of Shapiro Wilk. The values of normally distributed variables have been reported as mean and deviation of the standard. Data for variables that did not follow distribution of a normal were given as median and interquartile range. Categorical data is shown as percentages and frequencies. T-test was used to compare age and BMI between the patients with and without dyslipidaemia. The Mann-Whitney U test was used to compare the duration of the event of CKD and eGFR. The Kruskal-Wallis statistical test was used to compare lipid levels between the different stages of CKD. The chi-square test or Fisher's exact test was used for comparison of categorical variables, as appropriate. Dyslipidaemia was analyzed using binary logistic regression to identify the factors associated with dyslipidemia. The main demographic and clinical confounding factors were calculated using crude odds ratios and their 95% confidence intervals. Multivariable model: age, gender, is diabetic mellitus (DM), is in CKD stage 5, and current lipid-lowering therapy. Dialysis was not included in the adjusted model as all the patients receiving dialysis were patients with stage 5 CKD. The model was evaluated

by the likelihood-ratio chi-square test, area under the receiver operating characteristic curve, and Hosmer-Lemeshow goodness-of-fit test. The p-values corresponding to the markers were calculated, and $p<0.05$ was taken as statistically significant.

## Results:

LDL-C/HDL-C-defined dyslipidemia was identified in 66 (78.6%) of the 84 patients with CKD. Among patients with dyslipidemia, 37 (56.1%) were male and 29 (43.9%) were female, whereas the group without dyslipidemia comprised 6 (33.3%) males and 12 (66.7%) females; however, the gender-wise difference was not statistically significant (p=0.149). Mean age and BMI were also comparable between the two groups. Patients with dyslipidemia had a significantly lower median eGFR than those without dyslipidemia, 15.8 (9.8-32.7) compared with 26.2 (23.5-43.5) mL/min/1.73 m² (p=0.008). CKD stage was significantly associated with dyslipidemia (p=0.022), with stage 5 disease present in 47.0% of patients with dyslipidemia compared with 11.1% of those without dyslipidemia. Dialysis was also more frequent among patients with dyslipidemia, 33.3% compared with 5.6% (p=0.019). Current lipid-lowering therapy was used by 33 (39.3%) patients, including 5 (27.8%) patients without dyslipidemia and 28 (42.4%) patients with dyslipidemia; however, the difference was not statistically significant (p=0.392). Among treated patients, 16 (48.5%) received atorvastatin 20 mg, 10 (30.3%) received rosuvastatin 10 mg, 6 (18.2%) received atorvastatin 40 mg, and 1 (3.0%) received fenofibrate 145 mg. Residence, socioeconomic status, smoking, physical activity, CKD etiology, hypertension, diabetes, vascular disease, and previous dyslipidemia did not differ significantly between the groups, as shown in Table 1.

**Table 1. Demographic and clinical characteristics according to LDL-C/HDL-C-defined dyslipidemia status (n=84).**

| Characteristics | Overall (n=84) | Without dyslipidemia (n=18) | With dyslipidemia (n=66) | p-value |
|---|---|---|---|---|
| Age, years | 53.2 ± 8.1 | 51.8 ± 7.3 | 53.5 ± 8.4 | 0.403 |
| **Gender** | | | | |
| Male | 43 (51.2) | 6 (33.3) | 37 (56.1) | 0.149 |
| Female | 41 (48.8) | 12 (66.7) | 29 (43.9) | |
| BMI, kg/m² | 24.4 ± 3.2 | 24.3 ± 3.5 | 24.4 ± 3.2 | 0.891 |
| **Residence** | | | | |
| Rural | 49 (58.3) | 10 (55.6) | 39 (59.1) | 1.000 |
| Urban | 35 (41.7) | 8 (44.4) | 27 (40.9) | |
| Socioeconomic status | | | | |
| Low | 27 (32.1) | 5 (27.8) | 22 (33.3) | 0.871 |
| Middle/high | 57 (67.9) | 13 (72.2) | 44 (66.7) | |
| **Smoking status** | | | | |
| Current/former smoker | 32 (38.1) | 6 (33.3) | 26 (39.4) | 0.845 |
| Never smoked | 52 (61.9) | 12 (66.7) | 40 (60.6) | |
| **Physical activity** | | | | |
| Sedentary | 36 (42.9) | 7 (38.9) | 29 (43.9) | 0.908 |
| Moderate/active | 48 (57.1) | 11 (61.1) | 37 (56.1) | |
| Duration of CKD, years | 3.4 (1.6-5.1) | 2.0 (1.0-3.8) | 3.8 (1.9-5.6) | 0.073 |
| Current eGFR, mL/min/1.73 m² | 20.3 (11.2-35.4) | 26.2 (23.5-43.5) | 15.8 (9.8-32.7) | 0.008 |
| **Underlying cause of CKD** | | | | |
| Diabetic nephropathy | 32 (38.1) | 7 (38.9) | 25 (37.9) | 0.969 |
| Hypertensive nephropathy | 30 (35.7) | 6 (33.3) | 24 (36.4) | |
| Other causes | 22 (26.2) | 5 (27.8) | 17 (25.8) | |
| **CKD stage** | | | | |
| Stage 3 | 25 (29.8) | 8 (44.4) | 17 (25.8) | 0.022 |
| Stage 4 | 26 (31.0) | 8 (44.4) | 18 (27.3) | |
| Stage 5 | 33 (39.3) | 2 (11.1) | 31 (47.0) | |
| **Treatment modality** | | | | |
| Conservative treatment | 61 (72.6) | 17 (94.4) | 44 (66.7) | 0.019 |
| Dialysis | 23 (27.4) | 1 (5.6) | 22 (33.3) | |
| Hypertension | 51 (60.7) | 13 (72.2) | 38 (57.6) | 0.392 |
| Diabetes mellitus | 41 (48.8) | 11 (61.1) | 30 (45.5) | 0.362 |
| Vascular disease | 15 (17.9) | 5 (27.8) | 10 (15.2) | 0.296 |
| Previous history of dyslipidemia | 14 (16.7) | 5 (27.8) | 9 (13.6) | 0.167 |
| Current lipid-lowering therapy | 33 (39.3) | 5 (27.8) | 28 (42.4) | 0.392 |

The median LDL-C increased from 118.0 mg/dL in stage 3 and 118.5 mg/dL in stage 4 to 134.0 mg/dL in stage 5 (p=0.035), while median HDL-C progressively decreased from 47.0 to 42.0 and 37.0 mg/dL, respectively (p=0.001). Dyslipidemia was present in 68.0% of patients with stage 3 CKD, 69.2% with stage 4 CKD, and 93.9% with stage 5 CKD (p=0.022). Elevated LDL-C and low HDL-C were also significantly more frequent in stage 5 disease, affecting 84.8% and 69.7% of patients, respectively. Although total cholesterol and triglyceride concentrations were generally higher in advanced CKD, the differences did not reach statistical significance. Current lipid-lowering therapy was used by 10 (40.0%) patients with stage 3 CKD, 9 (34.6%) with stage 4 CKD, and 14 (42.4%) with stage 5 CKD, with no significant difference across the stages (p=0.827). Median eGFR declined significantly across the three stages (p<0.001), while 23 (69.7%) patients with stage 5 CKD were receiving dialysis, as shown in Table 2.

**Table 2. Lipid profile and frequency of dyslipidemia according to CKD stage (n=84).**

| Variable | Overall (n=84) | Stage 3 (n=25) | Stage 4 (n=26) | Stage 5 (n=33) | p-value |
|---|---|---|---|---|---|
| Total cholesterol, mg/dL | 204.5 (182.8-232.2) | 198.0 (176.0-223.0) | 198.5 (189.2-219.0) | 213.0 (195.0-243.0) | 0.150 |
| LDL-C, mg/dL | 127.5 (108.8-142.8) | 118.0 (107.0-134.0) | 118.5 (108.0-130.8) | 134.0 (122.0-149.0) | 0.035 |
| HDL-C, mg/dL | 41.0 (36.0-47.0) | 47.0 (40.0-52.0) | 42.0 (37.2-46.5) | 37.0 (33.0-42.0) | 0.001 |
| Triglycerides, mg/dL | 180.0 (154.0-210.2) | 167.0 (141.0-191.0) | 191.0 (156.8-203.8) | 182.0 (169.0-232.0) | 0.062 |
| Current eGFR, mL/min/1.73 m² | 20.3 (11.2-35.4) | 48.3 (38.8-53.5) | 23.5 (20.1-25.7) | 9.7 (7.7-12.5) | <0.001 |
| LDL-C/HDL-C-defined dyslipidemia | 66 (78.6) | 17 (68.0) | 18 (69.2) | 31 (93.9) | 0.022 |
| Hypercholesterolemia | 48 (57.1) | 12 (48.0) | 12 (46.2) | 24 (72.7) | 0.067 |
| Elevated LDL-C | 55 (65.5) | 13 (52.0) | 14 (53.8) | 28 (84.8) | 0.011 |
| Low HDL-C | 41 (48.8) | 7 (28.0) | 11 (42.3) | 23 (69.7) | 0.005 |
| Hypertriglyceridemia | 8 (9.5) | 0 (0.0) | 3 (11.5) | 5 (15.2) | 0.138 |
| Receiving dialysis | 23 (27.4) | 0 (0.0) | 0 (0.0) | 23 (69.7) | <0.001 |
| Current lipid-lowering therapy | 33 (39.3) | 10 (40.0) | 9 (34.6) | 14 (42.4) | 0.827 |

In the crude analysis, every 5 mL/min/1.73 m² increase in eGFR was associated with a 17% reduction in the odds of dyslipidemia (OR=0.83, 95% CI: 0.71-0.97; p=0.023). Patients with stage 5 CKD had 7.09 times higher crude odds of dyslipidemia, while those receiving dialysis had 8.50 times higher odds. After adjustment for age, gender, diabetes mellitus, and current lipid-lowering therapy, stage 5 CKD remained significantly associated with dyslipidemia (adjusted OR=6.20, 95% CI: 1.29-29.89; p=0.023). Current lipid-lowering therapy was not independently associated with dyslipidemia (adjusted OR=1.93, 95% CI: 0.57-6.58; p=0.291). The adjusted model was statistically significant ($\chi^2$=12.86; p=0.025), demonstrated acceptable discrimination with an area under the receiver operating characteristic curve of 0.773, and showed satisfactory calibration on the Hosmer-Lemeshow test (p=0.659), as shown in Table 3. Adjusted model performance details are attached with the supplementary file.

**Table 3. Crude and adjusted logistic regression analysis of factors associated with LDL-C/HDL-C-defined dyslipidemia (n=84).**

| Predictor | Crude OR (95% CI) | p-value | Adjusted OR (95% CI) | p-value |
|---|---|---|---|---|
| Age, per one-year increase | 1.03 (0.96-1.10) | 0.432 | 1.02 (0.95-1.10) | 0.580 |
| Male gender | 2.55 (0.85-7.62) | 0.093 | 2.29 (0.70-7.57) | 0.172 |
| BMI, per 1 kg/m² increase | 1.01 (0.86-1.19) | 0.881 | – | – |
| Current or former smoking | 1.30 (0.43-3.90) | 0.639 | – | – |
| CKD duration, per one-year increase | 1.18 (0.93-1.48) | 0.167 | – | – |
| eGFR, per 5 mL/min/1.73 m² increase | 0.83 (0.71-0.97) | 0.023 | – | – |
| Hypertension | 0.52 (0.17-1.63) | 0.264 | – | – |
| Diabetes mellitus | 0.53 (0.18-1.54) | 0.243 | 0.77 (0.23-2.57) | 0.674 |
| Vascular disease | 0.46 (0.14-1.59) | 0.222 | – | – |
| Previous history of dyslipidemia | 0.41 (0.12-1.43) | 0.162 | – | – |
| CKD stage 5 | 7.09 (1.51-33.30) | 0.013 | 6.45 (1.35-30.88) | 0.020 |
| Receiving dialysis | 8.50 (1.06-68.09) | 0.044 | – | – |
| Current lipid-lowering therapy | 1.92 (0.61-6.00) | 0.264 | 1.93 (0.57-6.58) | 0.291 |

## Discussion:

LDL-C/HDL-C-defined dyslipidemia was present in 78.6% of patients with CKD. This indicates a substantial burden of lipid abnormalities in the study population. A local cross-sectional study conducted at Ghurki Trust and Teaching Hospital reported dyslipidemia in 79.3% of Pakistani adults. Low HDL-C was the most frequent abnormality in that study.[9] The close agreement may reflect similar dietary patterns, physical inactivity, and cardiovascular risk factors in the two Pakistani populations. However, the current study specifically included hospitalized patients with stages 3-5 CKD, which may have further increased the observed frequency. Thus, dyslipidemia appears to be highly prevalent among Pakistani patients with CKD.

Low HDL-C was found in 48.8% of the overall population. Its frequency increased from 28.0% in stage 3 to 69.7% in stage 5 CKD. A national study from the Pakistan Institute of Medical Sciences found that metabolic syndrome was more frequent among patients with advanced CKD and those receiving dialysis.[10] Low HDL-C is one of the central components of metabolic syndrome and may contribute to the metabolic burden observed in advanced renal disease. The progressive reduction in HDL-C may result from impaired HDL formation and maturation in severe renal dysfunction. Therefore, declining HDL-C was an important component of the worsening lipid pattern across CKD stages.

Dyslipidemia was more frequent among patients receiving dialysis than among those managed conservatively. Dialysis was recorded in 33.3% of patients with dyslipidemia and in only 5.6% of those without dyslipidemia. Gupta et al. also found significantly higher triglyceride levels and lower HDL-C levels among Indian patients with CKD compared with healthy controls, with lipid disturbances remaining clinically relevant in patients managed with hemodialysis.[11] The similarity supports the effect of severe renal dysfunction on lipid metabolism. Dialysis itself may also influence the lipid profile through inflammation, nutritional changes, and repeated exposure to the extracorporeal circuit. Hence, dialysis dependence identified a subgroup with a particularly high burden of dyslipidemia.

Hypercholesterolemia was present in 57.1% of patients. Median total cholesterol increased from 198.0 mg/dL in stage 3 to 213.0 mg/dL in stage 5, although the stage-wise difference was not statistically significant. A study from Tamil Nadu reported a significant increase in total cholesterol with advancing CKD, reaching a mean of 261.7 mg/dL in stage 4.[12] The stronger association in that study may be related to differences in age, nutritional status, CKD-stage composition, lipid-lowering treatment, and the method used to summarize the data. The smaller sample in the current study may also have limited its ability to detect a moderate difference. Total cholesterol tended to increase with CKD severity, but it was not a reliable stage-specific marker in this population.

The frequency of LDL-C/HDL-C-defined dyslipidemia increased from 68.0% in stage 3 and 69.2% in stage 4 to 93.9% in stage 5. Kumar et al. similarly reported dyslipidemia in 71.7% of children with earlier CKD and in 95.7% of those with later-stage disease.[13] Although their population was pediatric, the direction and magnitude of the stage-related increase were similar. Progressive renal impairment disrupts lipid processing and promotes the accumulation of atherogenic particles. The marked increase in stage 5 supports CKD severity as the main clinical factor associated with dyslipidemia.

Among patients with stage 5 CKD, 69.7% were receiving dialysis. An Iraqi study of 111 maintenance-hemodialysis patients reported elevated LDL-C in 59.1% and an elevated triglyceride-to-HDL-C ratio in 64.5%.[14] The high frequency of lipid abnormalities in both studies supports the persistence of atherogenic dyslipidemia after dialysis has started. Differences in LDL-C frequency may be related to the much lower LDL-C threshold used in the Iraqi study and differences in statin use. Dialysis patients should therefore undergo careful assessment of the complete lipid pattern rather than LDL-C alone.

Elevated LDL-C was found in 65.5% of patients. Its frequency increased to 84.8% in stage 5, and the median LDL-C rose significantly from 118.0 mg/dL in stage 3 to 134.0 mg/dL in stage 5. In contrast, Hoca et al. observed that LDL-C declined as CKD advanced in a large Turkish cohort.[15] Lower LDL-C in advanced CKD may occur when inflammation, poor appetite, protein-energy wasting, and reduced hepatic lipoprotein production become prominent. Their cohort was older and included substantial differences in inflammatory and nutritional status. These factors were not fully assessed in the current study. The direction of LDL-C change in advanced CKD may therefore depend on nutritional condition, inflammatory burden, treatment exposure, and patient selection.

LDL-C may be affected by differences in statin prescribing and treatment adherence. The international CKD Outcomes and Practice Patterns Study included 8,194 patients from Brazil, France, Germany, and the United States. LDL-C was lower among treated patients and varied significantly between countries, but it did not differ consistently by CKD stage.[16] Current lipid-lowering therapy was included in the adjusted model and was not independently associated with dyslipidemia. However, the analysis did not account for treatment duration or therapy-specific adherence. Residual confounding related to treatment exposure therefore cannot be excluded. Medication history should be considered when interpreting LDL-C across CKD stages.

The study assessed LDL-C but did not measure apolipoprotein B. A Chinese population study found apolipoprotein B to have a stronger association with CKD than several conventional lipid measures.[17] LDL-C measures the cholesterol content of LDL particles, whereas apolipoprotein B more directly reflects the number of circulating atherogenic particles. Patients may therefore remain at increased cardiovascular risk even when LDL-C is not markedly elevated. Conventional LDL-C measurement may not fully represent the atherogenic lipid burden in CKD.

Patients with dyslipidemia had a significantly lower median eGFR than those without dyslipidemia. Each 5 mL/min/1.73 m² increase in eGFR was associated with a 17% reduction in the crude odds of dyslipidemia. Oh et al. also found significant associations between atherogenic lipid indices and CKD in adults with metabolic abnormalities.[18] Reduced filtration may impair the metabolism and clearance of triglyceride-rich and other atherogenic lipoproteins. The inverse relationship between eGFR and dyslipidemia therefore supports the biological plausibility of increasing lipid disturbance as renal function declines.

Males represented 56.1% of patients with dyslipidemia, compared with 33.3% of those without dyslipidemia. However, gender was not significantly associated with dyslipidemia and was not an independent predictor after adjustment. Wang et al. reported that the relationship between the atherogenic index of plasma and CKD was stronger among males in a large United States popula-

tion.[19] The difference may reflect their much larger sample and use of a lipid ratio rather than a binary definition based on LDL-C and HDL-C. The present sample may have lacked sufficient power to identify a modest gender effect. Gender was therefore not established as an independent determinant of dyslipidemia in this study.

The cross-sectional design shows an association but cannot determine whether dyslipidemia preceded or resulted from renal impairment. A prospective Chinese cohort of 32,351 participants found that total cholesterol, triglycerides, HDL-C, and several lipid ratios were significantly associated with the subsequent development of CKD.[20] These findings support a possible bidirectional relationship. Renal impairment may disturb lipid metabolism, while atherogenic lipids may contribute to vascular and glomerular injury. The current findings are biologically plausible, but their temporal direction cannot be established from a single assessment.

Median triglycerides increased from 167.0 mg/dL in stage 3 to 191.0 mg/dL in stage 4 and remained elevated at 182.0 mg/dL in stage 5. However, the stage-wise difference did not reach statistical significance. Only 9.5% were classified as having hypertriglyceridemia because the study used a high threshold of 250 mg/dL. The 4C study reported hypertriglyceridemia as the most common lipid abnormality and identified it in 54.7% of children with CKD.[21] Its higher frequency may be explained by a lower diagnostic threshold, the pediatric population, and differences in proteinuria and treatment. Thus, the low frequency of hypertriglyceridemia in the current study mainly reflects the restrictive operational cutoff rather than generally normal triglyceride concentrations.

Conventional lipid concentrations may not capture all lipid-related risk in dialysis patients. Papadea et al. found increased oxidized LDL-based markers among patients receiving peritoneal dialysis and demonstrated their potential value for assessing atherosclerotic risk.[22] Oxidative modification can increase the atherogenicity of LDL particles without producing a corresponding increase in measured LDL-C. Although the dialysis modality differed from the current study, the findings suggest that a standard lipid profile may underestimate cardiovascular risk in advanced CKD. Dialysis-associated dyslipidemia should therefore be interpreted together with the wider clinical and inflammatory context.

Diabetic nephropathy was the most frequent suspected cause of CKD, followed by hypertensive nephropathy. However, CKD etiology did not differ between patients with and without dyslipidemia. Zitnik et al. found a greater burden of dyslipidemia among pediatric dialysis patients whose kidney failure resulted from glomerular disease.[23] The contrasting result may be explained by differences in age, dialysis duration, underlying renal diseases, and classification of etiology. Grouping several uncommon causes into one category may also have concealed cause-specific differences in the current study. CKD etiology was not a significant determinant of dyslipidemia in this adult population.

Mean age and BMI were comparable between patients with and without dyslipidemia. Neither variable was significantly associated with the outcome in crude regression, and age remained non-significant after adjustment. The narrow BMI distribution and predominance of middle-aged patients may have reduced the ability to detect these associations. The effect of age and adiposity may also have been overshadowed by the strong metabolic effect of advanced renal dysfunction. Age and BMI did not explain the occurrence of dyslipidemia in this hospital-based CKD population.

Diabetes and hypertension were common but were not significantly associated with dyslipidemia. Diabetes also remained non-significant in the adjusted model. This does not indicate that these conditions are clinically unimportant. Both were widely distributed across the dyslipidemia and non-dyslipidemia groups, which reduced the contrast required to detect an association. Their effects may also operate partly through CKD development and progression rather than independently of CKD stage. In this population, renal disease severity had a stronger relationship with dyslipidemia than diabetes or hypertension.

Stage 5 CKD remained independently associated with dyslipidemia after adjustment for age, gender, and diabetes. Patients with stage 5 disease had 6.45 times higher adjusted odds of dyslipidemia than those with stages 3–4. The confidence interval was wide, which reflects the limited sample and the small number of patients without dyslipidemia in stage 5. Dialysis was not entered into the adjusted model because every dialysis patient had stage 5 disease, creating substantial overlap between the two predictors. The regression findings therefore identify advanced CKD, rather than dialysis as a separate factor, as the dominant independent correlate of dyslipidemia.

Overall, the findings indicate that dyslipidemia is common among patients with CKD and becomes more pronounced as renal function declines. Elevated LDL-C and low HDL-C were the clearest stage-related abnormalities. The strong association with stage 5 CKD remained after adjustment for major demographic and clinical factors. These findings support regular lipid screening throughout CKD, with particular attention to patients with stage 5 disease and those receiving dialysis.

This study had several limitations. It was conducted at a single tertiary care hospital and included a relatively small sample. Therefore, the findings may not represent patients managed in primary care, other regions of Pakistan, or the general community. The cross-sectional design did not establish the temporal or causal relationship between CKD and dyslipidemia. Consecutive non-probability sampling may have introduced selection bias. Lipid measurements were obtained at one point, and temporary dietary or metabolic changes may have influenced the results. Although the type and dose of lipid-lowering medication were recorded, treatment duration and therapy-specific adherence were not available for analysis. Information on proteinuria severity, serum albumin, thyroid function, and inflammatory markers was also not included. The small number of patients without dyslipidemia also produced wide confidence intervals in the regression model. In addition, the triglyceride cutoff of 250 mg/dL was higher than that used in many contemporary studies and may have underestimated hypertriglyceridemia.

Future studies should use multicentre and prospective designs with larger samples from different regions of Pakistan. Patients should be followed across CKD stages to determine how lipid profiles change as renal function declines. Detailed information on diet, proteinuria, nutritional status, inflammation, dialysis duration, and lipid-lowering therapy should be collected. Future research should also assess non-HDL-C, apolipoprotein B, triglyceride-to-HDL-C ratio, lipoprotein(a), and oxidized LDL. These markers may identify residual cardiovascular risk that is not detected by the conventional lipid profile. Separate analyses of hemodialysis, peritoneal dialysis, and non-dialysis populations

would clarify the effect of treatment modality. Intervention studies are also needed to determine whether earlier detection and management of dyslipidemia can reduce cardiovascular events and slow CKD progression.

## Conclusion:

LDL-C/HDL-C-defined dyslipidemia was common among patients with CKD and became more frequent as renal function declined, particularly in stage 5 disease. Regular assessment of the complete lipid profile may support timely management of cardiovascular risk in patients with advanced CKD.

## References:


1. GBD 2019 Pakistan Collaborators. The state of health in Pakistan and its provinces and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet Glob Health. 2023 Feb;11 (2):e229-e243. doi: 10.1016/S2214-109X(22)00497-1. PMID: 36669807; PMCID: PMC10009760.
2. Khan A, Cheema MF, Fatima R, Cheema SS, Butt Z, et al. Prevalence of Chronic Kidney Disease in a High-Risk Population in Urban Lahore, Pakistan: A Cross-sectional Study. Cureus. 2024 Jun 27;16(6):e63296. doi: 10.7759/cureus.63296. PMID: 39077231; PMCID: PMC11284504.
3. Suh SH, Kim SW. Dyslipidemia in Patients with Chronic Kidney Disease: An Updated Overview. Diabetes Metab J. 2023 Sep;47(5):612-629. doi: 10.4093/dmj.2023.0067. Epub 2023 Jul 24. PMID: 37482655; PMCID: PMC10555535.
4. Nguyen HH, Tran HH, Nguyen LT, Nguyen T, Nguyen NA, Vi MT, Nguyen KT. TG/HDL-C Ratio Is a Risk Factor Associated with CKD: Use in Assessing the Risk of Progression of CKD. Pathophysiology. 2022 Jul 17;29 (3):374-382. doi: 10.3390/pathophysiology29030029. PMID: 35893599; PMCID: PMC9326757.
5. Yoon J, Han T, Heo SJ, Kwon YJ. Comprehensive assessment of the combined impact of dyslipidemia and inflammation on chronic kidney disease development: A prospective cohort study. J Clin Lipidol. 2024 Mar-Apr;18(2):e251-e260. doi: 10.1016/j.jacl.2024.01.002. Epub 2024 Jan 10. PMID: 38233308.
6. Manhas M, Shan R, Kotwal S, Kotwal SK, Gupta M. Lipid profile of chronic kidney disease patients on two different modalities of treatment: Conservative versus hemodialysis. Natl J Physiol Pharm Pharmacol. 2023;13(04):736-739. doi: 10.5455/njppp.2023.13.06308202218082022
7. Ansar M, Khan MNA, Farooq A, Saeed HAS, Alvi FN, Rana H. Association of dyslipidemia with chronic kidney disease in patients requiring renal replacement therapy. Pak Armed Forces Med J. 2025;75(5):965-968. doi:10.51253/pafmj.v75i5.10845.
8. Malik SI, Khan R, Imran A, Akram M, Maqbool S, Habib R. Frequency of dyslipidemia in patients undergoing maintenance hemodialysis at Bahawal Victoria Hospital, Bahawalpur. Professional Med J. 2020;27 (9):1829-1833. doi:10.29309/TPMJ/2020.27.09.3975.
9. Mushtaq T, Farooq A, Ambreen I, Usmani M, Irshad O, Salman M. Prevalence of dyslipidemia and its association with lifestyle choices among Pakistani adults: a cross-sectional study. Dev Med Life Sci. 2025;2(1):3-7. doi:10.69750/dmls.02.01.098.
10. Kanwal K, Abbasi MSR, Sultan K, Butt G, Ashraf MU. Prevalence of metabolic syndrome in patients with chronic kidney disease: a hospital-based cross-sectional study. Pak J Kidney Dis. 2025;9(2):28-33. doi:10.53778/pjkd92301.
11. Gupta N, Dahiya S, Bansal P, Kumar S, Agarwal AK. A Study of Thyroid Profile and Lipid Profile in Patients with Chronic Kidney Disease with or without Hemodialysis in a Tertiary Care Hospital. J Assoc Physicians India. 2023 May;71(5):11-12. doi: 10.5005/japi-11001-0254. PMID: 37355813.
12. Prabu V, Mohamed Rebayudeen SM, Geetha J, Prabakar B, Kulothungan. A study of thyroid profile and lipid profile abnormalities in chronic kidney disease patients in a tertiary health care centre in Chengalpattu District, Tamil Nadu. Afr J Biomed Res. 2024;27 (3S):1019-1024. doi:10.53555/AJBR.v27i3S.2206
13. M AK, Mantan M, Mahajan B. Serum apolipoproteins (apoA-1, apoB, and apoB/apoA-1 ratio) for early identification of dyslipidemia in children with CKD. Pediatr Nephrol. 2024 Mar;39(3):849-856. doi: 10.1007/s00467-023-06144-0. Epub 2023 Sep 27. PMID: 37752382.
14. Hayder Aledan HA, Zaid David ZD, Sara Haider SH, Zainab Almosawi ZA, Zinelabidin Saleh ZS. [The TG/HDL ratio as a marker of dyslipidemia among patients on maintenance hemodialysis. A cross-sectional study]. Urologiia. 2024 Nov;(5):97-101. Russian. PMID: 39564851.
15. Hoca E, Mermer HB, Kula AC, Ahbab S, Ataoglu HE. The effects of chronic kidney disease stages on dyslipidemia, cardiovascular disease prevalence and mortality. North Clin Istanb. 2025 Jan 28;12(1):95-102. doi: 10.14744/nci.2024.81582. PMID: 40838232; PMCID: PMC12364478.
16. Calice-Silva V, Muenz D, Wong MMY, McCullough K, Charytan D, et al. International practice patterns of dyslipidemia management in patients with chronic kidney disease under nephrology care: is it time to review guideline recommendations? Lipids Health Dis. 2023 May 25;22(1):67. doi: 10.1186/s12944-023-01833-z. PMID: 37231413; PMCID: PMC10210460.
17. Xu Y, Liu B, Lin L, Lei F, Sun T, et al. The association of apolipoprotein B with chronic kidney disease in the Chinese population. Front Endocrinol (Lausanne). 2023 Apr 12;14:1083614. doi: 10.3389/fendo.2023.1083614. PMID: 37124758; PMCID: PMC10132458.
18. Oh D, Lee S, Yang E, Choi HY, Park HC, et al. Atherogenic indices and risk of chronic kidney disease in metabolic derangements: Gangnam Severance Medical Cohort. Kidney Res Clin Pract. 2025 Jan;44(1):132-144. doi: 10.23876/j.krcp.23.043. Epub 2023 Dec 1. PMID: 38062621; PMCID: PMC11838861.
19. Wang Y, Cui J, Gao J, Liang S, Cai G, et al. Gender disparities in the association between atherogenic index of plasma and chronic kidney disease. BMC Public Health. 2025 Mar 1;25(1):825. doi: 10.1186/s12889-025-22087-7. PMID: 40025582; PMCID: PMC11871803.
20. Lyu K, Liu S, Liu Y, You J, Wang X, et al. The Effect of Blood Lipid Profiles on Chronic Kidney Disease in a Prospective Cohort: Based on a Regression Discontinuity Design. Biomed Environ Sci. 2024 Oct 20;37 (10):1158-1172. doi: 10.3967/bes2024.113. PMID: 39622665.
21. Mencarelli F, Azukaitis K, Kirchner M, Bayazit A,

Duzova A, et al. Dyslipidemia in children with chronic kidney disease-findings from the Cardiovascular Comorbidity in Children with Chronic Kidney Disease (4C) study. Pediatr Nephrol. 2024 Sep;39(9):2759-2772. doi: 10.1007/s00467-024-06389-3. Epub 2024 May 8. PMID: 38720111; PMCID: PMC11272819.

22. Papadea P, Kalaitzopoulou E, Skipitari M, Varemmenou A, Papasotiriou M, et al. Novel oxidized LDL-based clinical markers in peritoneal dialysis patients for atherosclerosis risk assessment. Redox Biol. 2023 Aug;64:102762. doi: 10.1016/j.redox.2023.102762. Epub 2023 Jun 2. PMID: 37302344; PMCID: PMC10363433.
23. Zitnik E, Streja E, Laster M. The Impact of Glomerular Disease on Dyslipidemia in Pediatric Patients Treated with Dialysis. Nutrients. 2025 Jan 27;17(3):459. doi: 10.3390/nu17030459. PMID: 39940317; PMCID: PMC11819668.

| Authors' contribution | |
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| Naveed Ahmed | Data interpretation, and manuscript editing |
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