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# Lipid Profile Alteration in CKD – ( A Case – Control Study)

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**ABSTRACT:**

**Background:** Chronic kidney disease (CKD) is characterized by decline in kidney function. The normal kidney function is lost in patients having chronic renal insufficiency; the most affected are the excretory and regulatory functions, as a result of infections, autoimmune conditions, diabetes, hypertension, cancer, or exposure to poisonous chemicals. One of the most prevalent side effects of chronic renal failure (CRF) is dyslipidemia, which is often associated with declining renal function and is evident even in early stages of CRF.
**AIM:** The study aimed to compare and correlate the pattern of low-density lipoprotein (LDL) and triglycerides (TG) in patients having CKD.
**Methods:** This case-control study was carried out at Integral Institute of Medical Sciences Lucknow on 100 patients with CKD in age group 18 to 75 years and divided in 2 groups (cases with CKD of stage-I to II (GFR of >60 ml /min/1.73m2) and case group included 50 cases with CKD of stage-IIIA-V (GFR <60 ml/min/1.73m2) on hemodialysis. Patients if who had Coronary Artery Disease, Heart Failure, fever, pyrexia of unknown origin (PUO), acute Poisoning, HIV positive patient, viral infection, skin infection, parasitic infestation, patients on anti-inflammatory drugs, any known malignancy were excluded.
**Result:** The age in the majority of them fell between 18 and 75 years, and the gender distribution was insignificantly distributed. Serum urea, serum creatinine, triglycerides and LDL was significantly higher ($p<0.05$) in case group than in control group. Estimated glomerular filtration rate (eGFR) of case group was lower than in control group and was found statistically significant ($p<0.05$). Age, Serum Urea, Serum Creatinine, Triglyceride and LDL has a negative correlation with the eGFR.
**Conclusion:** An increase in triglycerides and LDL levels are important risk factors for the development of CKD based on eGFR. Sustained monitoring and cautious interpretation of the triglycerides and LDL in CKD patients will be needed in clinical practice.

Keywords: chronic kidney disease, triglycerides, LDL, eGFR, Dyslipidemia.

## 1. Introduction

Chronic Kidney disease (CKD) is the condition causes damage to kidney or its slow impairment involving loss of glomerular, and tubular function which lasts longer than 3 months [1]. CKD has emerged as public health problem with worldwide prevalence of about 8.0%-16.0% and >10.0% prevalence in adult population of United States [2,3]. Due to the absence of a national registry, the incidence of CKD in India is not well known. Nonetheless, estimates suggest that as many as 785 persons per million may have CKD in India [4]. CKD progressively advances to end-stage renal disease (ESRD), typically with high rate of cardiovascular morbidity and death. Actually, cardiovascular problems are a greater cause of death for CKD patients than for ESRD patients [5].

In regard to lipid management, patients with CKD require special consideration when compared with other high-risk groups. Cardiovascular disease (CVD) is leading cause of the morbidity and the mortality in CKD people [6], with a continual increase in the risk of CVD from early stages of CKD along with a decline in renal function [7]. However, the association between LDL-C level and cardio-vascular outcomes in CKD population seems less apparent, and role of the statin treatment in this population is unknown than in other high-risk populations. Most notably, the 4D (Deutsche Diabetes Dialysis Study) and AURORA (A Study to Evaluate Use of Rosuvastatin in Subjects on the Regular Hemodialysis: Assessment of Survival, and Cardio-vascular Events) trials have unambiguously proven that statin treatment plays no role in reducing cardio-vascular events among ESRD patients, in contrast to the more prevalent benefit of LDL-C reduction in high-cardiovascular risk populations [8]. Moreover, subgroup analyses in SHARP (Study of Heart and Renal Protection) study and recent meta-analyses have revealed a trend of reduced benefit of statins as CKD progressed from stage III to stage V [9,10]. This trend of a weak association between cardiovascular risks and LDL-C levels or statin treatment in advanced CKD stages believed to be multi-factorial. Factors include changes in the cholesterol metabolism, like low LDL-C production, but the longer plasma residence-time, and the lower LDL-C but higher ratio of the oxidized and the small-dense LDL, that is more atherosclerotic [11], and increase in risk of non-atherosclerotic cardio-vascular disease, like calcium/phosphate imbalance induced arterial calcification, hyperkalemia induced arrythmia, and the uremic bleeding tendency induced hemorrhagic stroke in CKD progression [12]. However, in the current lipid management guidelines of the major medical societies, the CKD stages regarded as the crucial factors for the grouping, and the lower target LDL-C are set with the advancing CKD stages [13], mainly based on higher cardio-vascular risks from the early CKD to the advanced CKD in the observational studies [12], rather than on direct evidence between the lower LDL-C and cardio-vascular outcomes from the clinical trials of CKD. The SHARP research, the sole randomised controlled trial that addressed lipid management in the CKD population, lacked the capacity to discern the impact of statins on various stages of CKD independently and was not intended to assess the relationship between LDL-C and outcomes [14].

Numerous studies that have just been published suggest that dyslipidemia in people with CKD may play a proactive role in the development of CVD and the decline in renal function.[4] CVD is the primary cause of death for individuals with CKD, and its prevalence is significantly higher in hemodialysis patients [15]. Dyslipidemia is a recognised risk factor for CVD in general population, but further research and documentation are needed to fully understand the role dyslipidemia plays in the advancement of CKD. Therefore, the purpose of this study is to link cholesterol and LDL in CKD patients in the Lucknow community.

**AIM:** The study aimed to compare, and correlate pattern of low density lipoprotein and triglycerides in CKD patients.

## 2. Material and Methods

A case-control study took place at the Department of Medicine of Integral Institute of Medical Sciences, Lucknow. This study was conducted from September 2021 to august 2024. 50 with CKD, males and females between 18-70 years of age group patients and 50 healthy with similar gender and age group enrolled in this study. Patients were excluded if they had Coronary Artery Disease, Heart Failure, fever, viral infection, pyrexia of unknown origin (PUO), acute Poisoning, HIV positive patient, skin infection, parasitic infestation, patients on anti-inflammatory drugs, any known malignancy.
According to the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI)TM, GFR was used to categorise individuals with CKD into stages. The subjects were split up into two groups: Case and Control. Fifty people with CKD of stages I to II (GFR of >60 ml/min/1.73 m2) were included in control group, and fifty subjects on hemodialysis with CKD of stages IIIA to V (GFR <60 ml/min/1.73 m2) were included in case group. The HumaStar 200 automated analyzer (Human Diagnostics Worldwide, Wiesbaden, Germany) used enzymatic colorimetric method to measure serum total cholesterol, LDL, and triglycerides. Blood samples were taken from both groups using sterile tubes, following all aseptic precautions. Siemens' Dimensions RXL Max automated analyzer was used to measure serum urea and serum creatinine levels.

The different CKD stages form continuum. The stages of CKD classified as follows:[16]
**Stage I:** Kidney damage with normal, or increased GFR (>90mL/min/1.73 $m^2$)
**Stage II:** Mild reduction GFR (60-89mL/min/1.73 $m^2$)
**Stage IIIa:** Moderate reduction GFR (45-59mL/min/1.73 $m^2$)
**Stage IIIb:** Moderate reduction GFR (30-44mL/min/1.73 $m^2$)
**Stage V:** Severe reduction GFR (15-29mL/min/1.73$m^2$)
**Stage 5:** Kidney failure (GFR <15mL/min/1.73$m^2$ or dialysis)

Categorical/Ordinal data was expressed as percentage and compare by using Chi Square test. Independent Sample t test was used to test difference between quantitative data among groups. Bivariate analysis (Pearson correlation) was used to find the association of eGFR with age, LDL, triglycerides, S. urea, S. creatinine level. A p-value less than 0.05 ($P<0.05$) was considered as statistically significant.

## 3. Results

In this study we categorise our study patients based on KDIGO 2012 CKD stages and we noted that in control group 53.0% patients in stage I and 42.0% patients were in CKD stage II. In case group 8.0% patients in stage IIIa, 36.0% patients were in CKD stage IIIb, 38.0% patients were in CKD stage IV and 18.0% patients were belonging to CKD stage V **[Figure 1]**.
In the control group, the average age of CKD patients was 44.80±14.99 years, whereas in the case group, it was 46.98±14.35 years. It was determined that there was no statistically significant difference in mean age of the two groups ($P<0.05$). There were more female patients than males in both groups. 34 (68.0%) females and 16 (32.0%) men made up the case group, whereas 33 (66.0%) females and 17 (34.0%) males made up the control group. Age and sex did not significantly correlate with the number of CKD patients in case or control groups. **[Table 1]**.
The mean Serum Urea, Serum Creatinine, eGFR, Triglyceride and LDL in case group were found 67.74±16.33 mg/dl, 2.66±0.87 mg/dl, 27.16±10.22 ml/min, 178.85±14.03 mg/dl, and 95.36±9.64 mg/dl, respectively, in control group, mean Serum Urea, Serum Creatinine, eGFR,

Triglyceride and LDL were found to be 12.96±4.44 mg/dl, 0.75±0.29 mg/dl, 102.02±28.67 ml/min, 76.74±14.65 mg/dl, and 64.66±18.86 mg/dl, respectively. To compare pattern of kidney and lipid profile in groups, Independent Sample t-test performed, and the P-value was calculated. As seen in **Table 2**, serum urea, serum creatinine, treglycerides and LDL were significantly higher ($p< 0.05$) in case group than in control group. eGFR of case group was lower than in control group and this was also found statistically significant (P-value <0.05).

On the conflicting with growing age, Serum Urea, Serum Creatinine, Triglyceride and LDL level; the eGFR was originate to be reduced. Based our study data, nevertheless these are statistically significant; age, Serum Urea, Serum Creatinine, Triglyceride and LDL has a negative correlation**[Table 3, Figure 2 and 3]**. In our study negative sign indicate the universally proportional associatio**]**.

Figure 1: CKD stages Distribution in both groups

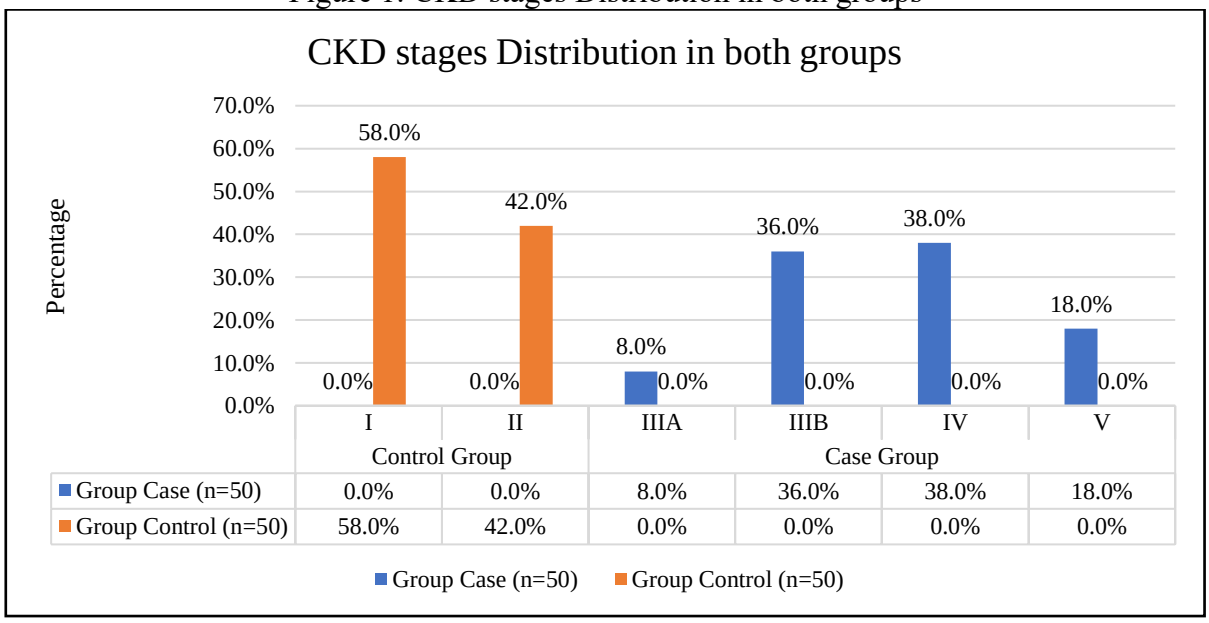



Table 1: Age and Sex Distribution

| | | Group | | $X^2$/t value | p value |
|---|---|---|---|---|---|
| | | Case (n=50) | Control (n=50) | | |
| Age Group (Years) | 18-30 | 8 (16.0%) | 12 (24.0%) | 1.272 | 0.866* |
| | 31-40 | 10 (20.0%) | 8 (16.0%) | | |
| | 41-50 | 12 (24.0%) | 12 (24.0%) | | |
| | 51-60 | 11 (22.0%) | 11 (22.0%) | | |
| | >60 | 9 (18.0%) | 7 (14.0%) | | |
| | Mean±SD | 46.98±14.35 | 44.80±14.99 | 0.743 | 0.459# |
| Sex | Male | 16 (32.0%) | 17 (34.0%) | 0.045 | 0.832* |
| | Female | 34 (68.0%) | 33 (66.0%) | | |

*Chi Square test; #Independent Sample t test

Table 2: Kidney function test variables Distribution

| | | **Case (n=50)** | **Control (n=50)** | **t value** | **p value#** |
|---|---|---|---|---|---|
| Kidney function test | Serum Urea (mg/dl) | 67.74±16.33 | 12.96±4.44 | 22.897 | <0.001 |
| | Serum Creatinine (mg/dl) | 2.66±0.87 | 0.75±0.29 | 14.680 | <0.001 |

| | | | | | |
|---|---|---|---|---|---|
| | eGFR (ml/min) | 27.16±10.22 | 102.02±28.67 | -17.393 | <0.001 |
| Lipid profile | Triglyceride (mg/dl) | 178.85±14.03 | 76.74±14.65 | 35.597 | <0.001 |
| | LDL (mg/dl) | 95.36±9.64 | 64.66±18.86 | 10.249 | <0.001 |

#Independent Sample t test

Table 3: Correlation of eGFR with age, S. urea, S. creatinine, LDL, and Triglyceride level.

| | eGFR (ml/min) | |
|---|---|---|
| | Pearson,s Correlation coefficient | Sig. (2-tailed) |
| Age (Years) | -0.213* | 0.033 |
| Serum Urea (mg/dl) | -0.792** | <0.001 |
| Serum Creatinine (mg/dl) | -0.865** | <0.001 |
| Triglyceride (mg/dl) | -0.813** | <0.001 |
| LDL (mg/dl) | -0.549** | <0.001 |
| **. Correlation is significant at 0.01 level (2-tailed). | | |
| *. Correlation is significant at 0.05 level (2-tailed). | | |

*Bivariate (Pearson Correlation) analysis;

Figure 2: Correlation of eGFR with Triglyceride level.

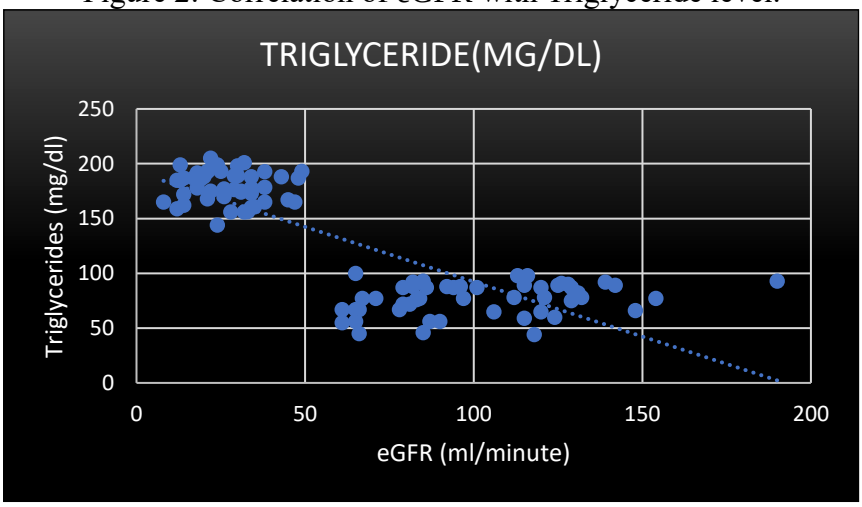
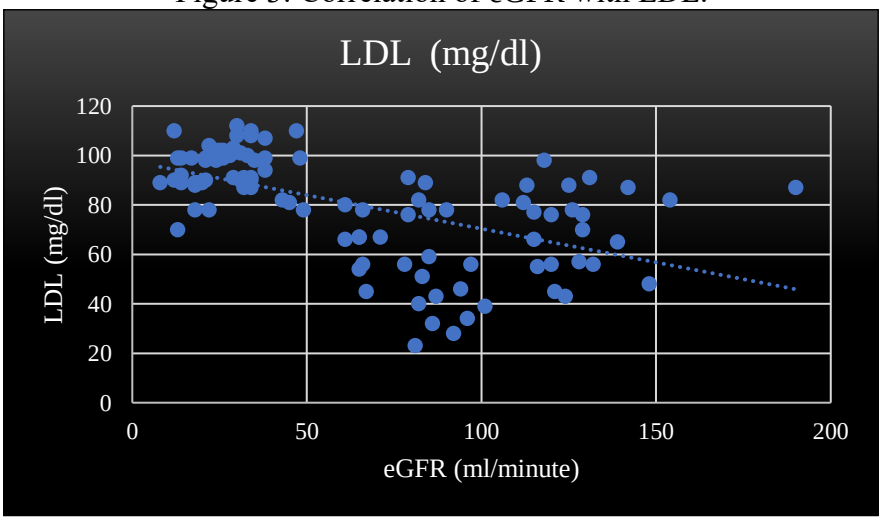



Figure 3: Correlation of eGFR with LDL.



## 4. Discussion

A dyslipidemia characterised by low HDL cholesterol and elevated triglycerides is associated with CKD. Triglycerides and proteinuria are related, however LDL-cholesterol levels are usually not elevated. The LDL-receptor and lipoprotein lipase are still directed in CKD, and increased triglycerides in CKD caused by the breakdown of triglyceride-rich lipoproteins with no change in the rate of synthesis [17]. CKD is connected with lesser levels of apoA-I (because of reduced hepatic expression [18], and advanced level apoB/apoA-I. Reduced LCAT activity and amplified cholesteryl ester transfer protein (CETP) activity donate to reduced HDL-cholesterol levels [19].

As CKD advancements the dyslipidemia frequently deteriorates. According to analysis of National Health and Nutrition Examination Survey (NHANES) data from 2001 to 2010, occurrence of dyslipidemia increased from 45.5% in stage-I CKD to 67.8% in stage-IV CKD; similarly, use of lipid-lowering medications increased from 18.10% in stage I CKD to 44.7% in stage IV CKD [20]. A bigger study appraising dyslipidemia in >21,000 incident dialysis cases found 82.0% incidence of dyslipidemia, and recommended threshold of the non-HDL cholesterol >100mg/dl (2.6mmol/L) to classify dyslipidemia in CKD stage-V patients [21].

In this study we noted mean values of triglyceride and LDL in case group were found 46.98±14.35 years 178.85±14.03 mg/dl, and 95.36±9.64mg/dl, respectively, and in control group, the mean values of age, triglyceride and LDL were found 176.74±14.65 mg/dl, and 64.66±18.86 mg/dl, respectively. As seen in **Table 2**, treglycerides and LDL were higher significantly ($p<0.05$) in case group than in control group. Mishra P et al [22], Verma J [23] and Vintha RK et al [24] also reported similar value of triglyceride and LDL in case and control groups in their respective study.

In our study, LDL in case group on hemodialysis found more than control group, similar to Saini M et al [25] and Singh S et al [26]. Enlarged LDL sub-fractions in dialysis patients were also noted by Morena et al [27]. In present study we noted that TGs to be greater in case group as compared to control group, which are supported by Saini M et al**Error! Bookmark not defined.** and Singh S et al [23] and was also supported by Zolezzi et al [28], who reported high TG in patients of CKD on hemodialysis. Because hemodialysis patients utilise heparin, that inhibits lipoprotein lipase, the enzyme that hydrolyzes triglycerides, their TG levels are higher than those of non-hemodialysis patients.

Hyperuricemia and dyslipidemia are significant illnesses that impair human health. As a result, we carefully investigated the correlation between triglycerides and LDL cholesterol levels with various stages of CKD. The goal of the current investigation was to determine if TG and LDL were linked to development of CKD. Specifically, even within the normal range of TG levels, our study's major findings reveal that triglycerides and LDL cholesterol indicate a risk of renal dysfunction and progression. On the conflicting with increasing age, Serum Urea, Serum Creatinine, Triglyceride and LDL level; the eGFR was originate to be reduced. Based our study data, nevertheless these are statistically significant; age, Serum Urea, Serum Creatinine, Triglyceride and LDL has a negative correlation. This result is different with previous studies in adult patients, the higher stage of CKD, the higher LDL and triglyceride level [22,23,24,28,29].

The dyslipidemia-to-CKD mechanism is currently being worked out. According to recent research, ectopic lipids may accumulate in nearly every type of cell, including mesangial cells, podocytes, and proximal tubular epithelial cells. This is caused by aberrant lipids in the blood [30]. Lipid-induced mitochondrial damage might also be more lethal to the proximal tubule cells [31]. In the kidney, high cholesterol leads to foam cell production and macrophage invasion. Patients with CKD have elevated blood levels of triglycerides and products of lipid metabolism, which have a severe atherosclerotic and pro-inflammatory effect on the renal

parenchymal vascular system [32]. Furthermore, renal tubular epithelial cells in the proximal and distal regions, podocytes, mesangial cells, microvascular endothelial cells, and interstitial macrophages—all of which have the ability to mediate the absorption of oxidised low-density lipoprotein (ox-LDL)—highly express CD36 [33], and also be combined with different circulating ligands to promote development of the kidney inflammation, the oxidative stress and fibrosis [34].

## 5. Conclusion

The present study concluded that an increase in triglycerides and LDL in CKD group patients as compared to control group. Alteration in lipid metabolism laterally with the development of stage of CKD may be led to rise risk of atherogenesis important to poor prognosis and amplified mortality rate. This emphasises how crucial it is to maintain lipid profiles and observe them intermittently in CKD patients so that they become unfluctuating as soon as possible to prevent cardio-vascular morbidity and death. It is advised that more focus be given to the multi-centric studies in order to have a compelling opinion on the design of lipid parameters in CKD cases, as despite the fact that there have been many studies on lipid parameters in CKD patients, inclination of lipo-protein pattern of the CKD patients on the hemodialysis remains unreliable.

**Declarations**

**Ethics approval and consent to participate**: Not applicable.

**Competing interests:** The authors declare that they have no competing interests.

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**Authors' contributions:** NM , KAM & MA conceived, received, wrote, and edited the article. The authors read and approved the final manuscript.

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