

# Factors Associated with Dyslipidemia in Adults with Chronic Kidney Disease at the Borgou Teaching Hospital (Benin)

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## Abstract

**Introduction:** Cardiovascular disease (CVD) is very common in chronic kidney disease (CKD). Lipoprotein metabolism is impaired in CKD patients, which is a significant risk factor for atherosclerosis and thus CVD. **Objective:** To investigate the serum lipid profile of patients with chronic kidney disease at CHUD-B/A in 2021. **Method:** A cross-sectional, descriptive and analytical study was conducted from January 2 to June 30, 2021, at the CHUD-B/A, in adult patients with CKD received in consultation or hospitalized. After ethical consultation, a venous blood sample was taken, followed by the determination of lipid parameters (total cholesterol, HDL cholesterol, triglycerides) by enzymatic colorimetric methods. LDL-cholesterol was calculated according to the formula of Friedwald *et al.* The plasma atherogenic index (PAI) was obtained by the formula  $\log(\text{triglycerides}/\text{HDL-cholesterol})$ . Dyslipidemias was defined according to the *National Cholesterol Education Program Adult Treatment Panel III (NCEP/ATP III)* classification. Logistic regression was used to identify factors associated with dyslipidemia at the 5% level. **Results:** A total of 54 patients with CKD were enrolled. The hospital prevalence of dyslipidemia was 64.81%. The different types of dyslipidemia found were HDL hypocholesterolemia (40.74%), LDL hypercholesterolemia (33.33%), hypertriglyceridemia (29.63%), total hypercholesterolemia (24.07%), mixed hyperlipidemia (12.96%), atherogenic dyslipidemia (7.41%). High PAI was the most common with 64.81%. Abdominal obesity was associated with mixed hyperlipidemia ( $p = 0.0043$ ). HDL hypocholesterolemia was associated with end-stage CKD ( $p = 0.0001$ ). Obesity (BMI) was a risk factor for LDL hypercholesterolemia ( $p = 0.0082$ ). **Conclusion:** The hospital prevalence of dyslipidemia is high in pa-

tients with CKD. Serum lipid parameters should be included in the follow-up consultations of CKD patients.

## Keywords

Dyslipidemia, Atherogenic Plasma Index, Chronic Kidney Disease, Benin

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## 1. Introduction

Chronic kidney disease (CKD) is a major public health problem, with high morbidity and mortality [1], and imposes a significant burden on patients and healthcare systems [2]. Diabetes and hypertension are the leading causes of CKD [3]. Chronic kidney disease (CKD) is a risk factor for atherosclerotic cardiovascular disease (ASCVD), with a 10-fold higher prevalence of ASCVD in CKD. In addition, the risk of ASCVD increases progressively as renal function declines [4]. At least 50% of all deaths in people with end-stage renal disease (ESRD) are related to ASCVD [4]. Existing data show that patients with CKD have a much higher incidence, prevalence and severity of ASCVD, with the incidence of de novo cardiovascular disease in patients with CKD reported to be 41% in men and 19% in women [5]. Age, high body mass index (BMI) and smoking are consistently associated with the development of CKD [6]. CKD is associated with several complications, including hypertension, cardiovascular disease, disorders of phosphocalcium metabolism, metabolic acidosis, anemia, hyperkalemia and malnutrition [3]. Dyslipidemia is also not uncommon in CKD. In fact, several studies have demonstrated significant lipid abnormalities in CKD patients, depending on the stage of the disease [7]. Adejumo *et al.* [8] in Nigeria and Ganta *et al.* [9] in India reported overall prevalences of dyslipidemia of 60% and 65% respectively. In Nigeria, Chiji-oke *et al.* [10] and Mshelia *et al.* [11] found that mean serum triglycerides and LDL cholesterol were significantly higher in patients with chronic kidney disease.

The progression of CKD may be related to these abnormalities, and consequently to the development of cardiovascular disease (CVD). Therefore, the present study was initiated with the aim to determine the serum lipid profile in CKD, to identify the major lipid abnormalities during the course of these diseases and the associated factors for a comprehensive and appropriate management of patients with CKD.

This work was initiated to study the frequencies and factors associated with different types of dyslipidemia in patients with chronic kidney disease at CHUD-B/A in 2021.

## 2. Methods

### 2.1. Nature, Scope and Study Period

This was a cross-sectional, descriptive, analytical study conducted from January 02 to June 30, 2021, in the Biomedical Laboratory and Nephrology Departments

of CHUD-B/A.

The study included adult subjects of both sexes, aged 18 years or older, who gave their free and informed consent to participate in the study, and who had impaired renal function for at least 3 months, whether on hemodialysis or not. Subjects with chronic kidney disease who were younger than 18 years of age or who had not given free and informed consent to participate in the study were not included.

## 2.2. Sampling, Parameters of Interest and Operational Definitions

The sample was non-probability and exhaustive. It consisted of considering all patients with CKD who came to CHUD-B/A either for consultation, hospitalization or dialysis and who met the above criteria during our study period.

The dependent variable was the presence of dyslipidemia. Dyslipidemia was defined according to the *National Cholesterol Education Program Adult Treatment Panel III (NCEP/ATP III)* [12]. Thus, dyslipidemia was considered present in an adult subject when at least one of these items (hypercholesterolemia > 2.00 g/L and/or hypertriglyceridemia > 1.50 g/L and/or decrease in HDL cholesterol < 0.40 g/L and/or increase in LDL cholesterol > 1.30 g/L) was detected.

- LDL cholesterol was calculated according to Friedwald *et al.* formula [13] if triglyceridemia was below 3.4 g/L.  $\text{LDL cholesterol} = \text{total cholesterol} - \text{HDL} - (\text{triglycerides}/5)$  in g/L [13].
- The plasma atherogenic index (PAI) is the log ratio (TG/C-HDL) and was used as a risk marker for cardiovascular disease [14]. For PAI between [-0.3 to 0.1[ for low risk, [0.1 to 0.24[ for intermediate risk and above 0.24 for high risk.
- Chronic kidney disease (CKD) was defined according to the KDIGO 2012 criteria [15]; *i.e.* the presence of renal insufficiency for more than 3 months, defined by a glomerular filtration rate (GFR) of less than 60 ml/min/1.73m<sup>2</sup> and/or a morphological or histological renal abnormality, provided that it is “clinically significant”, and/or an abnormality in the composition of the blood or urine as a result of renal damage.

The independent variables were sociodemographic factors (age, sex, marital status, occupation), terrain-related factors (diabetes mellitus and arterial hypertension), behavioral factors (alcoholism, smoking, sedentary lifestyle and dietary habits), CKD-related factors (relative to disease stage and treatment) and anthropometric factors (weight, height, body mass index, waist circumference).

The data collection tool used was a questionnaire with the following sections: sociodemographic characteristics, comorbidities, lifestyle, chronic kidney disease data, current treatment, clinical assessment, biochemical measurements.

Blood samples (4 mL) were obtained from each subject by superficial venipuncture on dry tubes. The samples were transported to the CHUD-B/A biochemistry laboratory within two hours of collection.

After centrifugation at 5000 rpm for five (5) minutes, the sera were decanted for use in the assay on the same day.

Total cholesterol was determined by the enzymatic endpoint method using cholesterol oxidase, while triglycerides were determined by glycerol phosphate oxidase [13]. HDL cholesterol was determined by the phosphotungstic acid precipitation method in the presence of magnesium ions [16], and serum LDL cholesterol concentration was calculated using the formula of Friedwald *et al.* [13]. Total cholesterol (TC), HDL cholesterol (HDL-C) and triglycerides (TG) were determined in each blood sample. Low-density lipoprotein cholesterol (LDL-C) was calculated.

The method for measuring total cholesterol (TC) in serum involved the use of three enzymes: cholesterol esterase (CE), cholesterol oxidase (CO) and peroxidase (POD). The reagent consisted of: Pipes buffer pH 6.7 (50 mmol/L), phenol (24 mmol/L) sodium cholate (5 mmol/L), cholesterol esterase ( $\geq 180$  U/L); cholesterol oxidase ( $\geq 200$  U/L), Peroxidase ( $\geq 1000$  U/L), 4-AAP (0.5 mmol/L) and sodium Azide ( $< 0.1\%$ ). Reagent R was placed in a container in the automaton on the spot it occupies. We took 1000  $\mu\text{L}$  of serum from each patient, placed in a vial and placed it in the machine, following the identification number of each sample, and noted the results on the on-board computer a few minutes later. For triglyceride determination, the reagent consisted of Pipes buffer pH 7.00 (50 mmol/L); p-Chlorophenol (2.7 mmol/L); magnesium (14.8 mmol/L); lipase ( $\geq 2000$  U/L); peroxidase ( $\geq 500$  U/L); 4-AminoAntiPyrin (0.31 mmol/L); glycerol-3-Phosphate Oxidase ( $\geq 4000$  U/L); glycerol kinase ( $\geq 500$  U/L); ATP (3.15 mmol/L); sodium azide ( $< 0.1\%$ ); and potassium ferrocyanide (10 micromol/L).

Once the container had been filled with reagent, 1000  $\mu\text{L}$  of serum from each patient was collected in the automated system. The results were recorded on the on-board computer a few minutes later. For cholesterol-HDL determination, the precipitation method was used. Chylomicrons, LDL and VLDL in serum were precipitated by the addition of phosphotungstic acid in the presence of magnesium ions. The fraction corresponding to HDL cholesterol contained in the supernatant obtained after centrifugation was determined by the enzymatic endpoint method. In hemolysis tubes numbered according to the number of samples, we took 75  $\mu\text{L}$  of serum from each sample and 150  $\mu\text{L}$  of precipitating reagent. After homogenization by inversion and a 10-minute rest at laboratory temperature, the mixture was centrifuged at 5000 rpm for 10 minutes. We recovered the supernatant for HDL-C determination. Hemolysis tubes were numbered according to the number of samples plus two others for the blank and the standard. *Magnesium chloride* (2 mol/L) was withdrawn in 1000  $\mu\text{L}$ , 100  $\mu\text{L}$  of standard solution was added to the corresponding tubes, and 100  $\mu\text{L}$  of precipitation supernatant from each sample was added to the corresponding tubes. The resulting mixture was incubated for 10 minutes at room temperature in the laboratory; and then read by spectrophotometer at 500 nm.

### 2.3. Data Collection and Analysis

Once the data were collected, they were double entered into Epi info version

7.2.3.1 and then exported to Microsoft Excel 2013 for analysis. This was also done with Epi-Info version 7.2.3.1. Categorical variables were expressed as percentages with confidence intervals; quantitative variables were expressed as means with standard deviation or median. Proportions were compared on a case-by-case basis using statistical tests such as Chi-square, Fischer or ANOVA. The measure of association between the dependent and independent variables was determined by logistic regression. A probability of  $p < 0.05$  was considered statistically significant.

## **2.4. Ethical and Deontological Considerations**

The research protocol was approved by the Local Ethics Committee for Biomedical Research of the University of Parakou. Oral consent was obtained from the study subjects. The data collected from the subjects in this study were kept strictly confidential and anonymous.

## **3. Results**

### **3.1. General Characteristics of the Sample**

A total of 54 patients whose records met our selection criteria were included in the study. Of these, 33 (61.1%) were male, resulting in a sex ratio of 1.57. The mean age was  $49.44 \pm 15.29$  years [extremes 20 and 90 years]. Of the 54 patients, 55.6% lived in couples, 35.19% were uneducated, 46.30% were farmers and 14.81% were shopkeepers. Among the 54 participants, hypertension, heart disease and diabetes accounted for 68.5%, 11.1% and 10.3% respectively. Practitioners of herbal medicine, those who consumed alcohol and those who were exposed to tobacco were 35.2%, 26.0% and 14.8% respectively. In terms of body mass index, 38.9% were overweight and 14.8% were abdominally obese.

### **3.2. Distribution of Patients According to Lesions and Stages of Chronic Kidney Disease**

Anatomo-clinically, 51.9% had benign nephroangiosclerosis, 9.6% had diabetic nephropathy, 13.5% had other chronic glomerulonephritis, 23.1% had tubulointerstitial nephropathy and 1.9% had polycystic kidney disease.

Stages 3, 4 and 5 of chronic kidney disease were found in 9.3%, 25.9% and 64.8% respectively.

### **3.3. Paraclinical Characteristics of the Patients**

Among the participants, 42.59% had hypocalcemia, 90.74% had a hemoglobin level below 10 g/dl, 11.1% had hyperkalemia, 3.7% had hypokalemia and 13.0% had hyponatremia.

### **3.4. Hospital Incidence of Dyslipidemia**

#### **3.4.1. Overall Hospital Frequency of Dyslipidemia**

Of the 54 patients, 35 had dyslipidemia. The overall hospital incidence of

dyslipidemia in CKD patients was therefore 64.8% (95% CI = [50.62% - 77.3%]).

### 3.4.2. In-Hospital Frequency of Different Types of Dyslipidemia

In terms of hospital frequency, 40.7% of the 54 patients had HDL hypocholesterolemia, followed by 33.3% with LDL hypercholesterolemia. The hospital frequencies for other dyslipidemias (hypertriglyceridemia, mixed hyperlipidemia, atherogenic dyslipidemia, total hypercholesterolemia) were 29.6%, 12.9%, 7.4% and 24.1% respectively. **Table 1** shows the described data.

**Table 1.** In-hospital frequency of different types of dyslipidemia in CKD patients at CHUD-B/A in 2021 (N = 54).

	Frequencies		95% CI
	Absolute	Relative (%)	
Hypertriglyceridemia	16	29.6	[17.98; 43.61]
LDL hypercholesterolemia	18	33.3	[21.09; 47.47]
Mixed hyperlipidemia	07	12.9	[5.37; 24.90]
Atherogenic dyslipidemia	04	07.4	[2.06; 17.89]
HDL hypocholesterolemia	22	40.7	[27.57; 54.97]
Total hypercholesterolemia	13	24.1	[13.49; 37.64]

### 3.5. Mean Lipid Parameters in Patients with CKD

The mean values of lipid parameters in (g/L) in patients with CKD are shown in **Table 2**.

**Table 2.** Mean values of lipid parameters in (g/L) for patients with CKD at CHUD-B/A in 2021.

	Mean ± standard deviation	[Extremes]
Total cholesterol	1.74 ± 0.65	0.55 - 4.61
HDL cholesterol	0.43 ± 0.16	0.10 - 0.79
LDL cholesterol	1.16 ± 0.58	0.10 - 3.53
Triglycerides	1.22 ± 0.62	0.48 - 3.09

In Stage 3 CKD, atherogenic dyslipidemia and mixed hyperlipidemia dominated, with hospital frequencies of 25.0% and 14.3%, while in stage 4 total hypercholesterolemia predominates with 30.8%. In stage 5, HDL hypocholesterolemia was more pronounced at 81.8%. **Table 3** shows the data described below.

**Table 3.** In-hospital frequency of different types of dyslipidemia by stage of CKD at CHUD-B/A in 2021.

	MRC stage 3		MRC stage 4		MRC stage 5		p
	n(%)	95% CI	n(%)	95% CI	n(%)	95% CI	
Hypertriglyceridemia	2(12.50)	[1.55 - 38.35]	2(12.50)	[1.55 - 38.35]	12(75.00)	[47.62 - 92.73]	0.0019
LDL hypercholesterolemia	2(11.11)	[1.38 - 34.71]	5(27.78)	[9.69 - 53.48]	11(61.11)	[35.75 - 82.70]	0.0302
Mixed hyperlipidemia	1(14.29)	[0.36 - 57.87]	1(14.29)	[0.36 - 57.87]	5(71.43)	[29.04; 96.33]	0.1017
Atherogenic dyslipidemia	1(25.00)	[0.63 - 80.59]	0(0.00)	-----	3(75.00)	[19.41 - 99.37]	0.2173
HDL hypocholesterolemia	2(9.09)	[1.12 - 29.16]	2(9.09)	[1.12 - 29.16]	18(81.82)	[59.72 - 94.81]	0.0001
Total hypercholesterolemia	1(7.69)	[0.19; 36.03]	4(30.77)	[9.09; 61.43]	8(61.54)	[31.58; 86.14]	0.0581

### 3.6. Factors Associated with Dyslipidemia

#### 3.6.1. Univariate Analyses

Medication use (adjusted OR = 8.75; 95% CI [1.04; 73.54],  $p = 0.0287$ ) and hypocholesterolemia (adjusted OR = 0.21; 95% CI [0.05; 0.87];  $p = 0.0270$ ) were associated with hypertriglyceridemia. A significant association was found between obesity and LDL hypercholesterolemia (adjusted OR = 0.13; 95% CI [0.02; 0.73];  $p = 0.0082$ ). Abdominal obesity and mixed hyperlipidemia (adjusted OR = 1.08; 95% CI [1.08; 36.65];  $p = 0.0043$ ) were significantly associated. **Table 4** shows the results of univariate analysis of factors associated with different types of dyslipidemia in CHUD-B/A in 2021.

**Table 4.** Factors associated with different types of dyslipidemia in CHUD-B/A in 2021, univariate analysis.

Variables and modalities	N	n	%	OR [95% CI]	p
<b>Hypertriglyceridemia</b>					
<b>Usual drug intake</b>					
No	15	01	6.67	1	
Yes	39	15	38.46	8.75 [1.04; 73.54]	0.0231
<b>Chloremia</b>					
Hypo	30	06	20.00	0.21 [0.05; 0.87]	0.0264
Hyper	11	03	27.27	0.32 [0.05; 1.79]	0.1882
Normal	13	07	53.85	1	
<b>LDL hypercholesterolemia</b>					
<b>Waist circumference</b>					
Normal	46	12	26.09	1	
Obesity	08	06	75.00	8.50 [1.50; 47.96]	0.0067

## Continued

Mixed hyperlipidemia					
<b>Waist circumference</b>					
Normal	46	4	8.70	1	
Obesity	8	3	37.50	6.30 [1.08; 36.65]	<i>0.0265</i>
Atherogenic dyslipidemia					
<b>Body mass index</b>					
Normal weight	22	1	4.55	1	
Undernutrition	5	3	60.00	31.5 [2.14; 463.16]	<i>0.0019</i>

### 3.6.2. Multivariate Analysis

A significant association was found between obesity and LDL hypercholesterolemia (adjusted OR = 0.13; 95% CI [0.02; 0.73];  $p = 0.0082$ ). Drug use (adjusted OR = 8.75; 95% CI [1.04; 73.54],  $p = 0.0287$ ) and hypochloremia (adjusted OR = 0.21; 95% CI [0.05; 0.87];  $p = 0.0270$ ) were associated with hypertriglyceridemia. Abdominal obesity and mixed hyperlipidemia (adjusted OR = 1.08; 95% CI [1.08; 36.65];  $p = 0.0043$ ) were significantly associated.

**Table 5** shows the results of the final multivariate analysis model of factors associated with different types of dyslipidemia in CHUD-B/A in 2021.

**Table 5.** Result of the final multivariate analysis model of factors associated with different types of dyslipidemia in CHUD-B/A in 2021 (N = 54).

	ORa	95% CI	p
<b>Hypertriglyceridemia</b>			
Usual intake of medication	8.75	[1.04; 73.54]	<i>0.0287</i>
Hypochloremia	0.21	[0.05; 0.87]	<i>0.0270</i>
<b>LDL hypercholesterolemia</b>			
Obesity	0.13	[0.02; 0.73]	<i>0.0082</i>
<b>Mixed hyperlipidemia</b>			
Abdominal obesity	1.08	[1.08; 36.65]	<i>0.0043</i>

## 4. Discussion

### 4.1. Study Limitations

The main limitation of our study is that it was monocentric, which does not reflect the reality of the Parakou population. As a result, our sample size was among the smallest of all the studies. The study was cross-sectional and did not allow evaluation of patient follow-up. It was also difficult to reduce the risk of confounding.

Despite these weaknesses, we believe that the results of this study are reliable.

## 4.2. Comments on the Results Obtained and Comparison with the Literature

### 4.2.1. Overall Hospital Incidence

The overall hospital incidence of dyslipidemia was 64.81% (95% CI = [50.62%-77.32%]). Similar prevalences have been reported by Adejumo *et al.* [6] in Nigeria (60%) and Ganta *et al.* [7] in 2015 in India (65%). However, Ladhari *et al.* [17] in India reported a lower prevalence of 20%. Elmachtani *et al.* [16] in Morocco in 2008 found a much higher prevalence of 80%. The discrepancy between these results could be related to the small size of our sample, the hospital setting of the study and the study population consisting of all CKD patients.

### 4.2.2. HDL Hypocholesterolemia

Its frequency was 40.74%. Deepak *et al.* [18] in India reported a prevalence close to ours: 36%. However, Elmachtani *et al.* [16] in Morocco in 2008 and Ganesan *et al.* [19] in Bangladesh in 2017 found higher values 58.7%, 70% and 80%, respectively. Patients' diets differ from one continent to continent. It should also be noted that most of the work has been done in patients at an advanced stage of CKD, and at this stage, the decrease in HDL-C could be due to a decrease in the levels of apolipoprotein A-I and A-II, the main protein components of HDL and/or a decrease in the activity of LCAT, the enzyme responsible for the esterification of free cholesterol from HDL particles, and/or an increase in the activity of cholesterol ester transfer protein (CETP), which facilitates the transfer of HDL-C esters to triglyceride-rich lipoproteins. All of these factors combine to lower serum HDL-C concentrations.

### 4.2.3. LDL Hypocholesterolemia

The prevalence of LDL hypercholesterolemia in our study was 33.33%. Deepak *et al.* in India [18] and Aljabri *et al.* in Saudi Arabia [20] reported similar prevalences of 36% and 37.20% respectively. In contrast, Ganta *et al.* [9] and Mshelia *et al.* [11] reported much lower prevalences of 12.86% and 17.50% respectively. This shows that LDL hypercholesterolemia is always present, regardless of the stage of CKD. Differences in results may be due to genetic factors (race), diet and patient follow-up.

### 4.2.4. Hypertriglyceridemia

Contrary to several studies reporting hypertriglyceridemia as the most common dyslipidemia, especially those by Ganesan *et al.* [19] in India (62%), Aljabri *et al.* [20] in Saudi Arabia (47.30%) and Mondal *et al.* [21] in Bangladesh (89%), our study classified hypertriglyceridemia as the third lipid anomaly (29.63%), comparable to the research work of Deepak *et al.* [18] who reported a prevalence close to ours (37%). The accumulation of triglycerides leading to hypertriglyceridemia in CKD is thought to be the consequence of high triglyceride production and low triglyceride catabolism. Several factors contribute to this metabolic alteration in

CKD, including reduced lipoprotein lipase (LPL) activity as a result of down-regulation of the enzyme gene, and a disproportionate increase in plasma apolipoprotein C-III, a possible cause of lipoprotein lipase inactivation when urea levels rise.

#### 4.2.5. Total Hypercholesterolemia

In the present study, the hospital incidence of total hypercholesterolemia was 24.07%. Adejumo *et al.* [6] found a similar prevalence of 29.50%. In contrast, Mondal *et al.* [21] and Llisterri *et al.* [22] found higher prevalences of 62% and 65% respectively.

This difference could be explained by the size of our population and the shorter study period in the present study.

#### 4.2.6. Mixed Hyperlipidemia

The hospitalization rate was 12.96%. Kaba *et al.* [23] in Guinea found 14% of cases, a result close to ours. In contrast, Llisterri *et al.* [22] reported a much higher prevalence: 26.40%. These results may be explained by the choice of criteria used to define mixed hyperlipidemia. We used the NCEP/ATP III classification of dyslipidemias.

#### 4.2.7. Atherogenic Dyslipidemias

In our study, the hospital incidence of atherogenic dyslipidemia was 7.41%. To date, none of the studies that have addressed this issue have reported it.

It is characterized by the lipid triad of high LDL levels, low HDL levels and high TG levels, leading to increased development of cardiovascular disease. In the presence of abdominal obesity or diabetes, which usually accompany these lipid combinations, glucose is not readily utilized due to insulin resistance. Energy must therefore be obtained from fat reserves, with the release of free fatty acids, triggering increased hepatic production of TGs contained in large particles of highly atherogenic very low-density lipoprotein (VLDL). VLDL exchanges these TGs for cholesterol with both LDL and HDL particles, and the TGs in these smaller particles are then hydrolyzed, producing large numbers of even smaller, denser particles. Smaller, denser LDL particles dense LDL particles contain less cholesterol (hence lower LDL measurements), but they readily penetrate the vascular endothelium, are easily oxidized and are highly atherogenic [24]. Low LDL levels mask the importance of the increased particles number, which is the parameter most strongly associated with vascular events [25]. Small HDL particles do not function well, resulting in some loss of the protective function of HDL [26]. Because of the small particle size, a significant number of LDL particles are lost through the kidneys, resulting in lower measured LDL levels. The true marker of increased cardiometabolic risk thus becomes the triad of atherogenic dyslipidemia [27]. On the other hand, measured LDL may be low, but this distorts the assessment of true risk. The LDL level simply reflects the amount of cholesterol in LDL particles and is not a reliable measure when these particles become smaller and more numer-

ous, or when a substantial amount of cholesterol is transported in VLDL and remnant lipoproteins [28].

### 4.3. Associated Factors

Abdominal obesity was a factor associated with mixed hyperlipidemia in our study ( $p = 0.0043$ ). HDL hypocholesterolemia was associated with end-stage CKD ( $p = 0.0001$ ). Obesity was a risk factor for LDL hypercholesterolemia ( $p = 0.0082$ ). However, Adejumo *et al.* [6] reported that the prevalence of hypertriglyceridemia, HDL hypocholesterolemia and high atherogenic risk increased significantly between CKD stages 1 and 5, with  $p$  values of 0.02, 0.04 and 0.03 respectively. They also reported that female gender was associated with dyslipidemia with  $p = 0.02$ . In the study by Liang *et al.* [29] in China between 2011 and 2016, hypertriglyceridemia ( $p < 0.001$ ) and high levels of TC ( $p = 0.001$ ) and LDL ( $p < 0.001$ ) were found to be associated with CKD progression. Ritchy *et al.* [30] in Madagascar from 2008 to 2015 reported that, adjusted for proteinuria, lipid parameters except triglyceridemia were significantly associated with a decrease in GFR ( $p < 0.05$ ).

Most of the studies reported to date have highlighted the factors associated with dyslipidemia in general and have only demonstrated their impact on the development of CKD.

## 5. Conclusion

The hospital incidence of dyslipidemia is high in these patients. HDL hypocholesterolemia, LDL hypercholesterolemia and hypertriglyceridemia are the most common dyslipidemias. Obesity, abdominal obesity and end-stage CKD are associated with dyslipidemia. Two-thirds of patients have an elevated plasma atherogenic index, putting them at risk for cardiovascular disease. Determination of serum lipid parameters should be an integral part of follow-up visits for patients with CKD, with the aim of detecting and managing dyslipidemia.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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