

Assessment of dyslipidemia in pre-dialysis patients in south-west Nigeria

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ABSTRACT

Background: Majority of chronic kidney disease (CKD) patients are more likely to die of cardiovascular complications before reaching end stage renal disease. The Kidney Disease Outcomes Quality Initiative (K/DOQI) recommends that all CKD patients should be evaluated for dyslipidemia and for treatment to reduce the risk of cardiovascular events. **Patients and Methods:** A cross-sectional case control study to determine the frequency of occurrence of lipid abnormalities in patients with CKD and compare these abnormalities with that of normal controls. A total of 100 patients and 100 controls were recruited for the study. Demographic and clinical data were obtained using structured questionnaire. Weight, height and waist circumference, body mass index (BMI) and blood pressure were also obtained. Subjects had their fasting lipid profile and fasting plasma glucose assayed after overnight fast of 8–14 hours. Low-density lipoprotein (LDL) was obtained using Friedwald formula. **Result:** The study revealed that total cholesterol (TC) was elevated above normal levels in 44% of cases compared with 6% in controls ($P < 0.001$), with the mean (SD) value of 5.82 ± 3.28 mmol/l for cases compared with 3.9 ± 1.0 mmol/l ($P < 0.001$) in controls. Low density lipoprotein was elevated in 48% of cases compared with 14% in controls ($P < 0.001$), with the mean (SD) values of 4.15 ± 2.74 mmol/l and 2.57 ± 0.95 mmol/l for cases and controls, respectively, ($P < 0.001$). Triglyceride (TG) was elevated above normal level in 26% of cases compared with none in the controls ($P < 0.001$), with the mean (SD) values of 1.41 ± 1.10 mmol/l and 0.64 ± 0.24 mmol/l for cases and controls, respectively ($P < 0.001$). All Lipid fractions except HDL also correlated significantly with levels of proteinuria TC ($r = 0.345$, $P = 0.001$), TG ($r = 0.268$, $P = 0.011$) LDL ($r = 0.366$, $P = 0.001$). **Conclusion:** Dyslipidemia is common among patients with CKD. Regular evaluation of all CKD patients for dyslipidemia and treatment need be instituted.

Keywords: Body mass index, chronic kidney disease, diabetes mellitus, dyslipidemia, friedwald formula low-density lipoprotein, hypertension

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INTRODUCTION

Chronic kidney disease (CKD) and indeed end stage renal failure are increasing in prevalence all over the world including Nigeria. Hospital-based data on the prevalence of kidney disease in Nigeria show a prevalence of 2–8%.^{1–4} However, emerging community studies show a prevalence of 10–19.9%.^{5–7} Cardiovascular diseases (CVDs) are common in patients with CKD and many of them are

more likely to die of cardiovascular complications before developing renal failure.⁸ One potential modifiable risk factor for CVD in patients with CKD is dyslipidemia. Evidence shows that CVD begins as soon as kidney function begins to deteriorate and increases in severity during progression of the disease.^{9,10} Given the overwhelming benefits regarding the treatment of dyslipidemia in non-renal populations at high risk for atherosclerotic disease as well as the high cardiovascular-related mortality of end stage renal disease (ESRD) patients, it is necessary that studies to determine the occurrence of dyslipidemia in CKD patients be undertaken. The increased cardiovascular and cerebrovascular complications in CKD may be due partly to accelerated atherosclerosis caused by dyslipidemia.¹¹ The common lipid abnormalities in CKD patients include low high-density lipoprotein (HDL), hypertriglyceridemia while low-density lipoprotein (LDL) and total cholesterol (TC) may be low, normal or high.^{12,13} These lipid abnormalities

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may persist in patients undergoing haemodialysis and may even get worse in those undergoing peritoneal dialysis and renal transplantation.^{14,15} Therefore to prevent the dire consequences associated with dyslipidemia, all patients with CKD should have routine assessment of their lipid level.

PATIENTS AND METHODS

The study was a single centre study involving adult patients with CKD seen at the University College Hospital, Ibadan. The study involved pre-dialysis patients with CKD from stage 1 to 5 according to the Kidney Disease Outcomes Quality Initiative (K/DOQI), classification of CKD. For each CKD subject selected, one age- and sex-matched individual without CKD was selected as control. Subjects were fully examined and anthropometric measurements including weight and height using a standard weighing scale and Seca stadiometer respectively. All subjects had their body mass index (BMI) calculated and their abdominal circumference was also measured. The blood pressure was taken with mercury sphygmomanometer using standard procedure. All subjects had urinalysis done using Combi — 10 strips. Patients with $\geq 1+$ proteinuria had their early morning spot urine sample taken for estimation of urine protein to creatinine ratio. Controls with overt proteinuria and microalbuminuria (urinary albumin concentration of 20 mg/L) were excluded from the study. Samples for serum lipid profile were collected from subjects after an overnight fast of 8-14 h using an anticoagulant bottle containing ethylene diaminetetracetic acid (EDTA) to prevent clotting. Samples were separated within 3 h of collection and kept in refrigerator at 4°C. Total cholesterol and HDL were assayed using enzymatic substrate method, while LDL was estimated using Friedwald formula¹⁶ as follows:

$LDL-C = TC - HDL-C - TG/2.19$ mmol/l. Where LDL= Low density cholesterol, HDL = High density cholesterol and TG = triglyceride.

Serum creatinine was estimated using the modified Jaffe kinetic reaction.¹⁷ Other laboratory investigations that were done included electrolytes, urea, packed cell volume and fasting plasma glucose. The glomerular filtration rate of each patient was estimated from serum creatinine using Cockcroft and Gault formula.¹⁸ Diagnosis of dyslipidemia was based on National cholesterol programme (NCEP) Adult Treatment Panel (ATP) III, which classifies TC level of < 200 mg/dl as 'desirable'.¹⁹ The atherogenic index (TC/HDL) and atherogenic index of plasma (\log_{10} TG/HDL) were also calculated. Patients on immunosuppressive drugs, for example corticosteroid, pregnant women, patients less than 18 years, those who refused to give consent and patients on lipid lowering drugs were all excluded from the study. Data were analysed using SPSS version 20. Variables were summarised using frequencies and proportions for

qualitative variables, and means and standard deviation for quantitative variables. The differences between groups concerning qualitative variable were tested using the chi-square test while mean differences were tested using the *t*-test (for two groups) and F test for analysis of variance for more than two groups. Levels of significance for all tests were at 5% level.

RESULTS

A total of 100 CKD patients and 100 controls were recruited into the study. The mean age of the CKD patients was 38.4 ± 12.6 years. Table 1 compares the socio-demographic and clinical characteristics of the CKD patients and their controls.

Figure 1 shows the proportion of individuals in each stage of chronic kidney disease. Males constituted 63% of the CKD patients while females were 37%.

Seventy-five (75%) participants had elevated blood pressure with a median duration since diagnosis of 2 (IQR 1-3 years) while 11% were diabetic with a mean duration since diagnosis of 4.7 ± 1.5 years. Chronic glomerulonephritis was the most common cause of CKD (43%) followed by hypertensive nephrosclerosis (21%), diabetes mellitus (11%), autosomal dominant polycystic kidney disease (4%) and human immunodeficiency virus (HIV)-related kidney disease in 6%. In 13% of the patients, the exact cause of the CKD could not be deciphered.

Table 1: Socio-demographic and clinical characteristics of respondents

	CKD patients (n = 100)	Controls (n = 100)	P
Age (years)	38.37±1.26	38.44±1.24	0.97
Female gender	37 (37%)	42 (42%)	0.47
Yoruba tribe	84 (84%)	80 (80%)	0.46
Religion			
Christianity	66 (66%)	81 (81%)	0.04
Islam	34 (33%)	19 (19%)	
Occupation			
Skilled	38 (38%)	58 (58%)	
Unskilled	41 (41%)	31 (31%)	0.01
Student	21 (21%)	11 (11%)	
Educational status			
Primary	21 (21%)	14 (14%)	
Secondary	38 (38%)	20 (20%)	0.002
Tertiary	41 (41%)	66 (66%)	
BMI(kg/m ²)	24.2±4.3	23.2±6.4	0.18
Waist circumference (cm)	82.2±12.4	81.9±11.0	0.88
Total cholesterol (mmol/L)	5.82±3.28	3.90±1.00	<0.001
LDL-C (mmol/L)	4.15±2.74	2.57±0.95	<0.001
HDL-C (mmol/L)	1.03±0.20	0.97±0.46	0.16
Triglyceride (mmol/L)	1.41±1.10	0.64±0.24	<0.001
High atherogenic index	84 (84%)	41 (41.0)	<0.001
Atherogenic index of plasma	35 (35%)	1 (1.0)	<0.001
Mean arterial blood pressure	108.5±25.0	90.4±11.1	<0.001

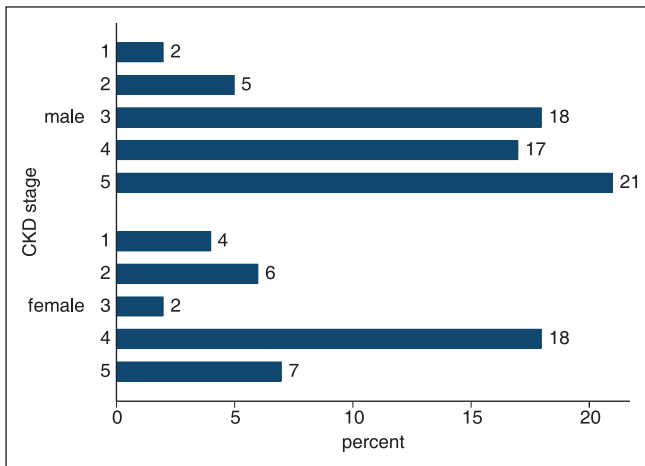


Figure 1: Proportion of individuals in each CKD stage by gender

Table 2: Proportions of lipid fraction derangements in subjects and controls

	CKD subjects n (%)	Controls n (%)	P
Total Cholesterol			
Desirable	56 (56)	94 (94)	<0.001
Non — desirable	44 (44)	6 (6)	
HDL			
Low	67 (67)	59 (59)	0.24
Normal	33 (33)	41 (41)	
LDL			
Optimal	47 (47)	86 (86)	<0001
Abnormal	48 (48)	14 (14)	
Triglyceride			
Normal	73 (73)	100	<0.001
Abnormal	26 (26)	0	

Table 3: Correlation of lipid fractions with selected cardiovascular parameters

	BMI	AER	SBP	DBP	WC	eGFR
Total Cholesterol	-0.01	0.49	0.16	0.20	0.09	-0.30
LDL-C	0.83	<0.001	0.02	0.004	0.40	<0.001
HDL-C	-0.02	0.48	0.18	0.23	0.03	-0.32
Triglyceride	0.74	<0.001	0.01	0.001	0.78	<0.001
	0.03	-0.18	-0.06	-0.01	0.14	-0.30
	0.63	0.01	0.37	0.88	0.17	0.004
	0.07	0.67	0.30	0.23	0.14	-0.47
	0.33	<0.001	<0.001	0.001	0.18	<0.001

BMI – Body mass index; AER – Albumin excretion rate; SBP – Systolic blood pressure; DBP – diastolic blood pressure; WC – Waist circumference; eGFR – Estimated GFR (Cocokroft–Gault). The upper figure in each cell is the Spearman rho while the lower figure is the P value

Table 4: Lipid profile characteristics by CKD stage

	HighTC n (%)	HighLDL n (%)	LowHDL n (%)	HighTG n (%)	HighAI n (%)
Stage 1 (N=6)	4 (66.7)	5 (83.3)	3 (50.0)	3 (50.0)	5 (83.3)
Stage 2 (N=11)	6 (54.6)	9 (81.8)	4 (36.4)	3 (27.3)	11 (100.0)
Stage 3 (N=20)	13 (60.0)	18 (90.0)	8 (40.0)	6 (30.0)	17 (85.0)
Stage 4 (N=35)	11 (31.4)	23 (65.7)	18 (51.4)	8 (22.9)	29 (82.9)
Stage 5 (N=28)	8 (28.6)	23 (82.1)	18 (64.3)	6 (24.4)	22 (78.6)

TC – Total cholesterol; LDL – Low density lipoprotein; HDL – High density lipoprotein; AI – Atherogenic index

The differences in the proportions with dyslipidemia are shown in Table 2. Fifty-six percent of cases compared with 94% of controls had desirable cholesterol levels ($P < 0.001$). There was no significant difference in HDL levels between CKD cases and controls. The distribution of LDL between CKD patients and controls showed that a lower proportion of CKD cases (47.5%) had optimal levels of LDL compared with 86% of controls ($P < 0.001$). Twenty-six (26.3%) of CKD cases compared with none of the controls had abnormal TG levels and this difference was significant ($P < 0.001$).

Eighty-four (84%) of the participants had at least one lipid fraction deranged. This constituted 83.8% in females and 84.1% in males. All the participants with stage 1 CKD had dyslipidemia; 90.0% of stage 2, 85% of stage 3, 74.3% of stage 4 and 89.3% of stage 5 patients had at least one fraction of the lipids deranged. Table 3 shows the correlation between the various fractions of lipid profile and selected clinical parameters.

Eight-five (85%) of the hypertensive respondents were on drug therapy. The most common anti-hypertensive medications used were ACE-inhibitors (30.3%) and calcium channel blockers (33%). Twelve (13.5%) were on one anti-hypertensive agent; 31 (34.8% of the hypertensive patients) on two drugs; 27 (30.3%) on three drugs; 12 (13.5%) on four drugs and 7 (7.9%) on five anti-hypertensives. In the subjects, there was no statistically significant difference in lipid fractions among the hypertensives compared with the non-hypertensives ($P = 0.16$ for TC; 0.09 for LDL; 0.17 for HDL, 0.95 for TG, 0.22 for AI).

Eleven (11%) of the subjects were diabetic. There was no difference in the mean fasting plasma glucose levels of the diabetics between the subjects and controls (6.53 ± 0.85 vs. 6.08 ± 1.61 , $P = 0.66$). Table 4 shows the proportion of subjects with deranged lipid profile in each stage of CKD. There was no identifiable pattern of lipid profile fragment change across the CKD stages.

DISCUSSION

This study examined the occurrence of dyslipidemia among patients with CKD and compares the findings with those of the controls. Similar study was earlier done by Agaba *et al.*,¹² on pre-dialysis patients. Most of the patients who had CKD in the study were patients within an average age

of 38 years. This is consistent with previous studies on CKD in our environment.^{3,20,21}

Most of the cases were in CKD stages 4 and 5. This again was consistent with previous findings in our environment.^{3,12} A similar study on lipid profile in CKD patients in Yaounde, Cameroon showed that about 62% of patients presented in stage 5 of CKD.²² This is a reflection of the general late presentation of our patients to hospital.

The present study demonstrates that CKD is commonly accompanied by lipid abnormalities in the entire lipid fraction. The study revealed derangement in all the lipid fractions. These findings were consistent with previous studies.^{12,23} However, the derangements in all the lipid fragments in our study were more than that of Agaba *et al.*¹² This may be because we included all stages of CKD.

In patients with CKD, LDL-C may be elevated or appear within normal level. Several studies including those within and outside Nigeria have shown elevated LDL-C.^{12-14,22} We observed in this study that the prevalence of abnormal levels of LDL-C was statistically significant in cases compared with the controls. Apart from being a strong cardiovascular risk factor for coronary heart disease, because of its strong atherogenic profile, LDL-C also contributes adversely to progression of renal disease.^{9,24} It does this by inducing the formation of oxygen radicals in arteries, in glomeruli and in juxtaglomerular cells, which results in an inhibition of nitric oxide-mediated vasodilatation, stimulation of renin release and modulation of mesangial cell growth and apoptosis.²⁵⁻²⁷

There is strong epidemiological and clinical evidence that increase in plasma level of LDL-C and low plasma level of HDL are associated with increased atherosclerotic complications.^{9,11,24} These complications are potentiated through the synergistic action of both LDL and HDL. While increased serum concentration of LDL is believed to be associated with increased deposition in the vessel wall, low levels of HDL may decrease the reverse cholesterol transport from tissues to the liver leading to their accumulation in the tissue thereby losing its protective effect. When the two are taken together they become highly atherogenic. The logarithm of the ratio is often referred to as atherogenic index. This ratio has been shown in several studies to be a better index for prediction of the risk of coronary artery disease and its attendant morbidity and mortality in both the general population and in patients with CKD.^{13,27,28} We found significantly higher values in cases compared with controls. The atherogenic index found in this study was slightly lower than what was found by previous researchers.^{12,29} This is most likely because of the very high percentage of patients who had high levels of LDL-C and a corresponding low level HDL.

We also observed that a significantly higher proportion of cases had high TC level compared with controls. This finding has been observed by others.^{12,13,21,29} Although hypercholesterolemia is clearly associated with a significant increase in the risk of cardiovascular-related disease and mortality in the general population, in CKD patients especially those on haemodialysis, it is believed that low levels of serum cholesterol is associated with an increased risk of mortality.^{10,30-32} The inverse association of TC level with mortality in haemodialysis patients is likely due to the inflammatory lowering effect of cholesterol due to malnutrition and not its protective effect. A high prevalence of malnutrition among CKD patients in our environment has been noted.^{33,34} The finding of significantly elevated level of TC like in our study could be associated with severe cardiovascular risk.

The level of TG was not elevated in controls while 26% of cases had elevated TGs ($P = 0.001$). This was consistent with what was found by previous authors.^{12,13,29} Higher level TG is one of the most common lipid abnormality in CKD patients. This may have been because of the higher proportion of diabetics among the subjects compared with the controls.

We observed that levels of proteinuria correlated significantly with levels of TC, TG and LDL. To our knowledge this may be the first attempt in Nigeria to try and correlate the levels of proteinuria with lipid abnormalities in CKD patients. However, a study outside Nigeria has shown that levels of blood pressure, levels of urinary protein correlates significantly with serum TC, TG and lipoprotein (a), and inversely with the levels of HDL-C.³⁵ The positive correlation between proteinuria, blood pressure, TC and TG, and the inverse correlation with HDL-C was reported in the MDRD study.²⁵ This correlation is further strengthened by the fact that control of proteinuria in both nephrotic and non-nephrotic patients with angiotensin converting enzyme also effectively reduce levels of TC, TG, LDL-C and increased levels of HDL-C.^{36,37} In addition, control of dyslipidemia with statin has been noted to reduce levels of proteinuria through its pleiotropic effect.³⁸ We could not establish the inverse relationship between total urinary protein and HDL-C. The mechanism of this relationship is unclear but it may be the same mechanism as in nephrotic syndrome.²³

A weak correlation was observed between the entire lipid fraction and all parameters that were used to define obesity. There was no significant correlation between BMI and waist circumference and the lipid fractions. These findings were different from previous studies.^{29,39} This may be as a result of the fact that most of the cases were either in stage 4 or stage 5 of CKD or that only very few subjects were obese.

As in previous Nigerian studies, we could not correlate the abnormalities in lipid fraction with GFR^{12,13} and

CKD stage. There was no association in lipid fractions between hypertensive and non-hypertensive. This was not consistent with a previous study in Nigeria.³⁰ A lot of factors may have contributed to this finding, such as educational status, smoking and alcohol consumption. Others include drugs used in treatment of diabetes and hypertension especially angiotensin converting enzyme inhibitor and angiotensin receptor blockers, which most of the patients were taking.

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