International Journal of Health Sciences and Research
Vol.10; Issue: 11; November 2020
Website: www.ijhsr.org
Original Research Article *ISSN: 2249-9571*

Serum Lipids and Apolipoprotein B/A1 Ratio Pattern in Chronic Kidney Disease Patients

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ABSTRACT

Background: Chronic kidney disease (CKD) has become a major health problem in developing countries, especially in Africa due to the burden of both infectious and non-communicable disease. Dyslipidemia is an established risk factor and independent predictor of cardiovascular disease (CVD) which is the principal cause of morbidity and mortality among CKD patients. The apolipoprotein B/A-1 ratio has been reported to be one of the strongest risk predictors of cardiovascular events. The aim of this study is to determine the plasma lipids and apo B/A-I ratio and its role as cardiovascular risk indicator in patients with chronic kidney disease.
Methods: 80 diagnosed CKD patients attending Nephrology Clinic were recruited with 80 age and sex matched apparently healthy adult controls between the ages of 35 – 70 years were recruited over a period of 8 months. Serum lipids, apolipoprotein A1 and B were determined after 8-10 hour fasting using enzymatic and immunoturbidimetry methods.
Results: apo B/A1, Triglyceride (TG), non-HDL, TC/HDL ratios are significantly higher among subjects with CKD than in healthy controls (P <0.001). The mean apo B/A1 ratio for CKD is 1.04 while for control is 0.60. HDL-C and apo A1 are significantly lower among the cases (P< 0.001). The commonest pattern of dyslipidemia in this population is hypertriglyceridemia and low HDL-C with a prevalence of 85%. The mean and prevalence of dyslipidemia increases as the albuminuria worsens
Conclusion: This study has demonstrated that dyslipidemia is common in CKD with predominantly hypertriglyceridemia and low HDL-C. Apo B/A1 is a strong predictor of cardiovascular events that can be utilized as a valuable tool in CVD risk assessment in patients with CKD

Keywords: Dyslipidemia, Chronic kidney disease, apolipoprotein B/A1 ratio

INTRODUCTION

Chronic kidney disease (CKD) is a major health problem affecting both developed and developing countries. It presently affects about 10% of the world's population and also a leading cause of global death accounting for most of the mortality and morbidity from non-communicable disease [(1,2)]. There is high burden of CKD in Africa, with prevalence of 15.8% for stages 1 to 5 in general African population, higher in Sub-Saharan African countries including Nigeria [(3,4)]. Community studies in Nigeria show a prevalence of 10-19.9% [(5-7)]. The reason for this rise in this environment is the double burden of both communicable and non-communicable disease attributed to rapid urbanization, changes in culture to western lifestyle including consumption of unhealthy diet. In consequence, there is rise in the prevalence of type 2 diabetes mellitus (T2D), hypertension obesity and with also the already high prevalence of infectious

diseases, especially HIV in sub-Saharan Africa [8,9], CKD has become a major threat to public health in developing countries.

Cardiovascular disease (CVD) is the leading cause of death in patients with CKD at every stage of the disease which implies most patients will die of CVD before reaching end-stage renal failure or dialysis becomes necessary [10]. CVD risk increases as the GFR declines and on average, tenfold higher in CKD Stage 5. Uremia, oxidative stress and inflammation in additional to the traditional risk factors have all play central role in atherogenesis contributing to the CVD in CKD patients [11].

Dyslipidemia is an established risk factor and independent predictor of CVD in general population. Studies has suggested the central role dyslipidaemia plays in the development and progression of renal disease contributing to the high cardiovascular morbidity and mortality of chronic kidney disease patients [12,13].

The dyslipidemia of renal disease has characteristic metabolic abnormalities. It usually involves all lipoprotein classes and shows considerable variations depending on the stage of CKD. It develops during the asymptomatic stages of renal insufficiency and becomes more pronounced as renal failure advances [14]. The most characteristic feature of the CRF-associated dyslipidemia is the accumulation of apolipoprotein B-containing triglyceride-rich lipoprotein particles in the very low density (VLDL) and intermediate density range (IDL).

Initially the estimation of serum lipids like cholesterol, triglycerides, LDL and HDL were used to assess the risk of coronary heart disease. However, the inconsistency in the correlation between serum lipid profile and coronary heart disease, led to the development of better indicators. Among them the estimation of serum apolipoproteins as a risk factor in coronary heart disease and also as a marker has shown great promise [15].

The apolipoprotein B/A-1 ratio has been reported to be one of the strongest risk predictors of cardiovascular events [16]. It reflects the balance between atherogenic and anti-atherogenic particles and the incidence of cardiovascular risk is directly proportional to the value of this ratio which makes it a strong predictor of CVD than traditional lipid profile. More recent work however provides growing evidence that apo B and apo A-I are more effective indicators of Cardiovascular risk.

Increased urinary albumin excretion rate (UAER), even in the early macroalbuminuric range, is associated with progressive renal failure and increased cardiovascular morbidity and mortality. Microalbuminuria, an early marker of CKD, is an independent risk factor for cardiovascular disease. Microalbuminuria is excretion of albumin 30 to 300 mg/day in a 24-hour urine collection or 30 to 300 µgram/mg creatinine in a spot collection. Nephropathy is usually characterized by progressive increase of urinary albumin excretion rate (UAER). Urine albumin excretion above the normal range may imply altered renal function, either increased leakage across the glomerular filtration barrier, a decrease in proximal nephron reabsorption of the proteins that are normally present in glomerular ultrafiltrate, or possibly abnormalities in other renal cell types.

The purpose of the study was to determine the levels of serum Apolipoprotein A-I, Apolipoprotein B and serum lipid profile and to find out Apo B/Apo A-I ratio and its role as cardiovascular risk indicator in patients with Chronic Kidney disease. This study also aims to study the correlation between Apo B, apo B/A1 and various degrees of albuminuria in CKD patient.

METHODS

Study design

This was an observational case-control and a single center study done at University College Hospital Ibadan. It involves 80 consecutive diagnosed pre-dialysis CKD patients attending Nephrology

Clinic recruited over a period of 8 months. Target population are patients with eGFR <60 mls/1.73m^2 from stage 3-5 according to Kidney Disease Outcome Quality Initiative (K/DOQI) classification of CKD. 80 age and sex matched apparently healthy adult controls were also recruited over the same period. The inclusion criteria were adults ≥ 18 years with CKD stage 3-5 who is yet to commence dialysis. Patients who have had dialysis, renal transplant or who are on lipid lowering drugs were excluded from the study. The inclusion criteria for control subjects were adults ≥ 18 years, not hypertensive or diabetic with normal renal function who are not on steroid or lipid lowering drugs.

The study was approved by the Hospital Health Research Ethics committee. Informed consent was obtained from all the participants. Demographic and medical data were getting through the use of questionnaires.

Definition of variables

Chronic kidney disease stages

The CKD stages are stage 1 – eGFR ≥90 mL/min/1.73 m^2 with kidney damage, stage 2 – eGFR = 60–89.9 mL/min/1.73 m^2 with kidney damage, stage 3 – eGFR = 30–59.9 mL/min/1.73 m^2,
stage 4 – eGFR = 15–29.9 mL/min/1.73 m^2 and stage 5 – eGFR <15 mL/min/1.73 m^2.

Dyslipidemia

Dyslipidemia was defined according to the National Cholesterol Education Program/Adult Treatment Panel III (NCEP/ATP III) as raised TG level ≥ 150mg/dl, reduced HDL-C < 40mg/dl, LDL-C level > 130mg/dl and/or TC level ≥ 200mg/dl.

Albuminuria

A urinary albumin-creatinine ratio (UACR) of <30 mg albumin/g creatinine was regarded as normal. Microalbuminuria was defined as a UACR of 30–300 mg/g and overt albuminuria (macroalbuminuria) as a UACR of >300 mg/g creatinine.

Sample Collection and Storage

After informed consent was taken, 5 mls of blood was drawn from each participant after an overnight fast of 8-10 hours. It was collected in plain tube, allowed to clot and separated through centrifugation (1500× g for 30 minutes). It was then stored at -20^{o}C until assay were run. The assays were run in 3 batches, the duration storage of each batch of samples is between 6-8 weeks. Under an aseptic procedure 5 mls of freshly voided midstream urine was collected into a plain tube for determination of UACR.

Biochemical Analysis

The lipid profile (Total Cholesterol, TG, HDL) was done using the enzymatic method (CHOD-PAP) on Landwind C 100 Plus Auto analyzer. LDL was calculated using the Friedewald formula. Serum Apolipoprotein B and Al concentrations were determined using immunoturbidimetry methods on Landwind C. 100 plus automated analyzer (Shenzen Landwind Industry Co. China). Urine Albumin-Creatinine-Ratio (UACR) was determined by estimating urine albumin was using the turbidimetric immunoassay on the automated chemistry analyzer. Urine and serum creatinine concentrations were analysed using the enzymatic creatinase method and the serum value obtained was used to calculate the eGFR of each participant using the abbreviated Modification of Diet in Renal Disease formula: GFR (mL/min/1.73 m²) = 175 × (Scr)-1.154 × (Age)-0.203 × (0.742 if female) × (1.212 if black)

Data Analysis

All statistical analysis was done using the IBM statistical package for social science (SPSS) version 20 software. All tests of statistical significance were 2 sized with 95% confidence interval. Continuous data are presented as mean and standard deviations (SD) while proportions are presented as numbers and percentages. Comparison of means was performed using the student t-test for unskewed data and Mann-Whitney U for skewed data. The chi - square test was used to determine the significance of observed differences for

categorical variables. P values < 0.05 were considered significant.

RESULTS

A total of 80 CKD patients and 80 age and sex-matched healthy controls were recruited into this study. CKD patients had a mean age of 55yrs and comprises of 41 males (56.3%) and 39 females (44.8%). The healthy controls are made up of 32 males (43.8%) and 48 females (55.2%) with an average age of 52yrs.

Table 1: Biochemical characteristics of study population

Variables	CKD patients mean (SD)	Healthy controls mean (SD)	P- value
Age (years)	55.1 (9.8)	52.3 (9.2)	0.080
TC (mg/dl)	183 (42)	176 (30)	0.230
TG (mg/dl)	104 (43)	63 (23)	<0.001*
HDL-C (mg/dl)	35 (7)	44 (7)	<0.001*
LDL-C (mg/dl)	127 (38)	119(28)	0.125
Non-HDL-C (mg/dl)	148 (40)	132 (31)	0.006
Apo B (mg/dl)	112 (27)	86 (22)	<0.001*
Apo A1 (mg/dl)	113 (23)	146 (22)	<0.001*
Apo B/A1 ratio	1.04 (0.42)	0.60 (0.17)	<0.001*
TC/HDL ratio	6.35 (2.10)	4.05 (0.85)	<0.001*
Serum creatinine (mg/dl)	4.22 (2.61)	0.82 (0.21)	<0.001*
eGFR(ml/min.1.73m^2)	28.8 (26.8)	105 (25.3)	<0.001*
UACR (mg/g)	1141.4 (1751)	38.7 (46.2)	<0.001*

*LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol; Apo B=Apolipoprotein B; Apo A1=Apolipoprotein A1; TG=Triglycerides; eGFR= estimated glomerular filtration rate; UACR= urine albumin creatine ratio TC=Total cholesterol. *P < 0.05*

Table 1 outlines the biochemical characteristics of the study population. No significant difference in the age between the two groups. Triglyceride (TG), non-HDL and apo B, apo B/A1 and TC/HDL ratios, are significantly higher in subject with CKD. HDL-C is significantly reduced in CKD group. However, there is no difference observed in total cholesterol and LDL-C between the two groups.

Table 2: Prevalence of dyslipidemia in healthy and controls

Variables	Prevalence (%)		Total (n=160)	P- value
	CKD Cases (n=80)	Control (n=80)		
TC (mg/dl)				0.715
<200	59 (73.8)	61 (75.0)	120 (75.0)	
≥ 200	21 (26.2)	19 (25.0)	40 (25.0)	
Apo B/A1				<0.001*
<0.8	19 (23.8)	69 (86.2)	88 (55.0)	
≥0.8	61 (76.2)	11 (13.8)	72 (45.0)	
LDL-C (mg/dl)				0.191
< 130	46 (57.5)	54 (67.5)	100 (62.5)	
≥130	34 (42.5)	26 (32.5)	60 (37.5)	
TG (mg/dl)				<0.001*
<150	68 (85.0)	80 (100.0)	148 (92.5)	
≥150	12 (15.0)	0 (0.0)	12 (7.5)	
HDL-C (mg/dl)				<0.001*
< 40	68 (85.0)	20 (25.0)	88 (55.0)	
≥40	12 (15.0)	60 (75.0)	72 (45.0)	
eGFR(ml/min/1.73m^2)				<0.0001*
< 60	70 (87.5)	4 (5.0)	74 (46.2)	
≥60	10 (12.5)	76 (95.0)	86 (53.8)	

*TC=Total cholesterol LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol; Apo B=Apolipoprotein B; Apo A1=Apolipoprotein A1; TG=Triglycerides*P < 0.05*

The prevalence of dyslipidemia among CKD cases and control is shown in Table 2. The commonest form of dyslipidemia in this study is elevated TG and reduced HDL with 85% prevalence. 76.2% of subjects have increased apo B/A1 ratio and 15% have a high TG. These dyslipidemia are statistically significant compared to control group. Although the number of CKD patients with higher TC, LDL, are more than the control group.

Table 3: Relationship of plasma lipids, apolipoprotein B and A1 and various degree of microalbuminuria among patients with chronic kidney disease

Variables	UACR <30 (n=11)	UACR 30-299 (n=25)	UACR ≥300 (n=44)	P- value
Apo B Mean (SD)	105.5 (20.0)	108.1 (22.1)	115.3 (23.1)	0.393
Apo A1 Mean (SD)	115.3 (23.1)	112.3 (19.4)	112.1 (25.1)	0.918
ApoB/A1 Mean (SD)	0.94 (0.25)	0.98 (0.20)	1.10 (0.52)	0.352
Total cholesterol Mean (SD)	172.9 (30.6)	178.9 (35.2)	188.3 (47.7)	0.463
Triglyceride Mean (SD)	92.1 (26.9)	108.9 (38.0)	104.1 (48.0)	0.557
HDL-C Mean (SD)	35.8 (7.2)	34.9 (6.0)	35.2 (6.8)	0.924
LDL-C Mean (SD)	118.6 (28.1)	122.2 (31.2)	132.6 (42.9)	0.393
Non HDL-C Mean (SD)	137.0 (28.9)	144.0 (32.1)	153.4 (45.8)	0.395

*LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol; Apo B=Apolipoprotein B; Apo A1=Apolipoprotein A1; TG=Triglycerides; eGFR= estimated glomerular filtration rate; UACR= urine albumin creatine ratio TC=Total cholesterol. *P < 0.05**

Table 4: Relationship of plasma lipids, apolipoprotein B and A1 and various degree of microalbuminuria among patients with chronic kidney disease

Variables	UACR n (%)			Total (n=80)	P- value
	< 30	30-299	≥300		
Apo B/A1					0.553
<0.8	4 (36.4)	5 (20.0)	10 (22.7)	19 (23.8)	
≥0.8	7 (63.6)	20 (80.0)	34 (77.3)	61 (76.2)	
TC (mg/dl)					0.805
<200	9 (81.9)	18 (72.0)	32 (65.1)	59 (73.8)	
≥ 200	2 (18.1)	7 (28.0)	12 (34.9)	21 (26.2)	
LDL-C (mg/dl)					0.204
< 130	9 (81.9)	14 (56.0)	23 (52.3)	46 (57.5)	
≥130	2 (18.1)	11 (44.0)	21 (47.7)	34 (42.5)	
TG (mg/dl)					0.652
<150	10 (90.9)	20 (80.0)	38 (86.4)	68 (85.0)	
≥150	1 (9.1)	5 (20.0)	6 (13.6)	12 (15.0)	
HDL-C (mg/dl)					0.463
< 40	8 (72.7)	22 (88)	38 (86.4)	68 (85.0)	
≥40	3 (27.3)	3 (12)	6 (13.6)	12 (15.0)	
eGFR(ml/min/1.73m^2)					0.263
< 60	8 (72.7)	22 (88.0)	40 (91.9)	70 (87.5)	
≥60	3 (27.3)	3 (12.0)	4 (9.1)	10 (12.5)	

*LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol; Apo B/A1=Apolipoprotein B/A1 ratio TG=Triglycerides; eGFR= estimated glomerular filtration rate; UACR= urine albumin creatine ratio TC=Total cholesterol. *P < 0.05*

The mean apolipoprotein B, apo B/A1, TC and LDL-C increase has the degree of albuminuria worsens while Apo A1 and HDL reduce; these differences show no significance (Table 3). Among the 3 groups of various degrees of albuminuria, there is a significant difference with plasma, lipids and lipoproteins through average, which tends to increase apoA1, and tends to decrease at the severity of albuminuria progressively (Table 4).

Table 5: Prevalence of Lipid abnormalities across the CKD stages

Parameter	CKD Stage 1 n (%)	CKD Stage 2 n (%)	CKD Stage 3 n (%)	CKD Stage 4 n (%)	CKD Stage 5 n (%)	P -value
High apo B/A1	2 (50)	5 (83.3)	14 (70)	13 (76.5)	27 (81.8)	0.612
High TC	1 (25)	2 (33.3)	3 (15)	7 (41.2)	8 (24.2)	0.479
High TG	1 (25)	1 (16.7)	3 (15)	3 (17.6)	4 (12.1)	0.959
High LDL-C	1 (25)	3 (50)	5 (25)	9 (52.9)	16 (48.5)	0.356
Reduced HDL-C	3 (75)	6 (100)	15 (75)	14 (88.4)	30 (90.9)	0.414

*LDL-C=Low density lipoprotein cholesterol; HDL-C=High density lipoprotein cholesterol; Apo B=Apolipoprotein B; Apo A1=Apolipoprotein A1; TG=Triglycerides; TC=Total cholesterol. *P < 0.05*

The prevalence of dyslipidemia increases across the stages of CKD. (Table 5) Majority of end stage renal disease patient (90.9%) has reduced HDL-C and 81.8% have a high apo B/A1 ratio.

DISCUSSION

The role of lipids and lipoproteins in the development and progression of CKD has been well documented in observational studies and clinical trials [17,18,19]. The metabolic abnormalities in chronic renal patient, especially the altered lipid metabolism is specific in its patterns depending on the degree of impairment [19].

The mean age of our study population was 55. 1± 9.8 years, which were similar to studies by Okwuonu et al which reported an average of 54. 8±12. 8 in Umuahia, Southeast Nigeria [20] and 56. 3±19. 9 by Oluyombo et al in Ekiti South-west Nigeria [21].

Hypertriglyceridemia and reduced HDL are the most common dyslipidemia in these subjects. This is consistent among established findings among CKD patients. They are usually characterized by high plasma triglycerides and low HDL levels, normal or slightly reduced cholesterol-LDL [22]. Cholesterol-LDL is not a reliable predictive cardiovascular risk factor in patients with advanced CKD. The hypertriglyceridemia is due to delayed catabolism and increased TG rich lipoproteins due to decreased activity of hepatic lipase. The presence of hepatic lipase inhibitors can also be explained by significant increases in plasma levels of apolipoprotein C-III, which is a potent inhibitor of lipoprotein lipase (LPL) [23]. LPL, which is located in the capillary endothelium, is responsible for triglyceride and phospholipid hydrolysis of VLDL and chylomicrons, leading to their deposition in arterial vessels [24,25].

The reduced HDL-C has been attributed to impairment of activity of lecithin-cholesterol acyl-transferase (LCAT) and decreased levels of Apo A1. LCAT is responsible for the conversion of cholesterol into its esterified forms, allowing hepatic removal of cholesterol. LCAT dysfunction causes morphological changes to HDL, which acquire a spherical rather than disc shaped structure, with resulting alteration of their catabolism [26,27].

However, the total cholesterol and LDL-C are not significantly higher than in controls. This is also reported by Odum et al in Port-Harcourt, Nigeria [28]. Plasma total cholesterol and LDL-C is usually normal or low in advanced cases and has been found to be a poor predictor of cardiovascular risk in end stage renal disease (ESRD) [29].

In this study, the majority (63.2%) of CKD patients is in stages 4 and 5 which depict the late presentation in this part of the world.

The apolipoprotein B, B/A1 ratio is significantly higher in CKD patients while apo A1 is lower than in healthy controls. This is consistent with ARIC study that lower apo A1 and higher B/A1 ratio is strongly associated with CKD [30]. The high apoB/A1 ratio has been attributed to reduced Apo A1 from decline activity of LCAT and increased apo B levels from

reduced catabolism of TG rich particles (VLDL, IDL).

Albuminuria is a measure of endothelial damage which has been linked to atherogenesis and contributed immensely to nephropathy. It is an independent risk factor for cardiovascular events. It is often found in association with dyslipidemia especially in patients at risk of nephropathy [(31)]. Our study shows worsening dyslipidemia across the stages of albuminuria. TG and Apo B/A1 ratio is increased over the stages of albuminuria. 65% and 23% of total CKD patients in this study has microalbuminuria and macroalbuminuria respectively. On the other hand, only 15% of the control has microalbuminuria and none has macroalbuminuria which is similar to the study by Oluyombo et al among individuals in South-West Nigeria [(21)].

In conclusion, this study has demonstrated the pattern of dyslipidemia among CKD patients with predominant hypertriglyceridemia, low HDL-C. APO B/A1 is significantly higher in them, which increases as the albuminuria progresses and across the stages of CKD.

ACKNOWLEDGEMENTS

We are thankful to the study participants for making this study possible. We also acknowledged the help of the entire department of Chemical Pathology and Renal unit, Internal Medicine for their assistance.

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How to cite this article: Ayoade OG, Bello T, Motilewa O et.al. Serum lipids and apolipoprotein B/A1 ratio pattern in chronic kidney disease patients. Int J Health Sci Res. 2020; 10(11):90-97.

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