ORIGINAL ARTICLE

LEFT VENTRICULAR GEOMETRY AMONG CHRONIC KIDNEY DISEASE PATIENTS: THE ROLE OF ANEMIA.

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ABSTRACT

Background: Anaemia and abnormal left ventricular (LV) geometric pattern are common findings in Chronic Kidney disease (CKD) patients.

Objectives: To assess LV geometric pattern and its relationship with anaemia among CKD patients.

Methods: A cross sectional study of 163 subjects (102 and 61 CKD subjects with and without anaemia respectively). Echocardiography determined the LV geometric pattern while packed cell volume (PCV) levels determined anaemia.

Results: The mean age of subjects with and without anaemia was 54.04 ± 14.47 and 54.92 ± 15.67 years respectively (p = 0.717) while the prevalence of LVH among the two groups was 68.8% and 57.9% respectively (p = 0.174). The most frequent LV geometry in both groups was concentric LVH (53.8% and 43.9% respectively). Prevalence of LV systolic dysfunction was 45%, higher among anaemic subjects (58(61.7%) vs 10(17.5%)) p < 0.001. There was a strong negative correlation between PCV and left ventricular mass index (r = -0.345, p = 0.001) among anaemic subjects, but weak positive correlation among patients without anaemia (r = 0.001, p = 0.993).

Conclusion: Anaemic CKD patients had a high prevalence of abnormal LV geometry with significant contribution from anaemia. Early management of anaemia may thus improve cardiovascular outcomes.

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INTRODUCTION

Chronic kidney disease (CKD) is a global problem with worsening prevalence. [1-6] It is associated with several cardiovascular risks, with estimated death rates up to 10-20 times those of an age-matched population. [4, 7] Many of these patients develop cardiomyopathy, left ventricular dilation, left ventricular hypertrophy and systolic dysfunction which ultimately lead to cardiac failure, haemo-dynamic overload, maladaptive cardiac responses and death, if untreated. [4, 7-9]

Cardiovascular disease (CVD) is highly prevalent among CKD patients and it is the main reason for the high mortality and morbidity rates among end-stage renal disease (ESRD) patients. [10] Many factors account for the strong relationship between CKD and CVD, but the contribution of anaemia and hypertension to the development of cardiac abnormalities and CKD have been proven to be

significant, with hypertension being a common etiology as well as a complication of CKD. [9-15]

Left ventricular hypertrophy (LVH) is an eventuality in untreated and poorly controlled hypertension and an independent cardiovascular risk factor in hypertensive patients, LVH also puts additional burden on the cardiovascular system of CKD patients and thus worsens prognosis. [9, 13, 16-18] Anaemia, advancing age, dyslipidemia, diabetes mellitus, glomerulonephritis and atherosclerosis are also commonly associated with CKD as well as LVH. [8-12, 14]

It is therefore important to find the association of anemia, left ventricular hypertrophy, left ventricular systolic dysfunction and the various LV geometric patterns in CKD patients, while controlling for these many confounders.

This study therefore sets out to assess the prevalence of the various geometric patterns of LVH, the prevalence of LV systolic dysfunction and to determine the relationship between LVH and anemia among CKD patients seen in our facility.

METHODS

STUDY DESIGN

The study was a descriptive cross sectional study conducted among patients with the diagnosis of chronic kidney disease (CKD), at the adult nephrology out-patient clinic of Lagos State University Teaching Hospital (LASUTH), Ikeja, Lagos.

STUDY POPULATION

Adult CKD patients in stages 3 to 5 with anaemia were consecutively recruited into the anaemia group while age and sex-matched CKD patients in stages 3 to 5 without anaemia were recruited into the non-anaemic group to serve as control. Patients with underlying cardiac disease such as valvular heart disease, congenital heart disease and cardiomyopathy, those with arteriovenous fistula and patients with features of volume overload (pulmonary oedema, peripheral congestion and congestive cardiac failure) were all excluded.

SAMPLE SIZE DETERMINATION

The minimum sample size of 100 for this study was calculated using the fisher's formula. [19] A standard normal deviation of 1.96 with a confidence level of 95% and a precision of 5% was used. A proportion of 7% obtained from a previous study on prevalence of CKD was used. The targeted sample size was achieved within a 10 month period (April 2016 to January 2017). A total sample size of 102 anaemic CKD subjects were recruited as well as 61 CKD subjects without anaemia, to serve as control; with a case: control ratio of approximately 2:1.

SAMPLING TECHNIQUE

A stratified systematic sampling technique was used for the selection of subjects. Patients were stratified into different stages of CKD 3 to 5 based upon their eGFR. A systematic random sampling technique was used to recruit patients, in which every 3rd patient that qualified were recruited into CKD subjects with anaemia group until the sample size was achieved. This also involved recruitment of at least 26 patients into each stages of CKD 3 to 5.

However, CKD subjects without anaemia were recruited using stratified random sampling technique into CKD stages 3 to 5, and simple random sampling technique was used to recruit them into each of the stages, and they were matched for age and gender. This technique was employed because of the challenges of achieving equal proportion of subjects in each of the stages especially in stage 5 CKD.

STUDY PROCEDURE

Clinical history was obtained using a structured questionnaire. Recruited subjects were stratified into appropriate CKD stages 3-5, in accordance with their eGFR (MDRD). [1] CKD was defined as abnormalities of kidney structure or function present for more than 3 months with implication for health and was classified into stages 3 to 5 of chronic kidney disease as follows: stage 3 with eGFR of 30 – 59 ml/min/1.73M^2, stage 4 with eGFR of 15 – 29 ml/min/1.73M^2, stage 5 with eGFR of < 15 ml/min/1.73M^2. [1]

Anaemia was defined as haemoglobin (Hb) concentration less than 13.0 g/dl (<130 g/l) in males and less than 12.0 g/dl (<120 g/l) in females. [14] Height and weight were measured using standard procedures, while Body mass index (BMI) was calculated using the formula: weight (kg)/ Height^2 (M^2) and Body surface area (BSA) was calculated using Dubois formula. [20] Blood pressure was measured in accordance to the recommendation of the American Heart Association guidelines. [21] Hypertension and Obesity were defined according to standard reference ranges. [19-21]

A total of 10mls of venous blood was collected from the antecubital fossa of each subject for estimation of fasting lipid profile (total cholesterol-TC, high density lipoprotein- HDL, low density lipoprotein- LDL, and triglycerides- TG), random blood glucose (RBG), packed cell volume (PCV) and serum electrolytes, urea and creatinine (E,U,Cr). [19, 22]

Transthoracic echocardiography (M-mode, two dimensional and Doppler) was performed with the General electric vivid Q echocardiographic machine, using 3.5 MHz phased array probe (cardiac probe) transducer following the American Society of Echocardiography and the European Association of Cardiovascular Imaging convention (ASE/EACI). [23] Two cardiologists read the echocardiograms to reduce intra-observer bias. Left ventricular mass (LVM), Left ventricular mass index (LVMI) and Relative wall thickness (RWT) were calculated by using the validated formula of American Society of Echocardiography and the European Association of Cardiovascular Imaging convention (ASE/EACVI). [23]

Left ventricular hypertrophy was defined in absolute terms as Left ventricular mass index >115 g/m2 in men and >95 g/m2 in women. [23] Left ventricular geometric pattern was classified as follows: Eccentric hypertrophy was defined as relative wall thickness (RWT) less than or equal to 0.42 in the presence of LVH, while concentric hypertrophy was defined as RWT greater than 0.42 in the presence of LVH, and concentric remodelling was defined as RWT greater than 0.42 in the absence of LVH, and normal left ventricular geometry was defined as RWT less than or equal to 0.42 in the absence of LVH. [23] Left ventricular systolic function was classified according to the standard values of the American Society of Echocardiography. [23]

DATA ANALYSIS

This was computed using statistical package for social science (SPSS) version 20. Continuous variables were described by calculating the means and standard deviation, and was compared using unpaired student t test in which normal distribution is assumed while screwed data (duration of hypertension and duration of CKD) were described with median and percentile, and compared using Mann Whitney U test.

Categorical variables were analyzed using percentages and compared using Chi squared test. Analysis of variance (ANOVA) was used to compare means across groups. Pearson's correlation was used to assess the relationship between LVMI and selected variables (age, sex, body mass index, systolic blood pressure (SBP), diastolic blood pressure (DBP), haemoglobin and eGFR), while linear regression was used to control for confounders. Confidence levels was set at $p<0.05$ and taken to be statistically significant and confidence interval was set at 95%. Microsoft Excel was used to produce charts.

ETHICAL APPROVAL

Ethical approval was sought and obtained from the Health Research Ethics Committee of Lagos State University Teaching Hospital Ikeja, before the commencement of the study. The respondents were assured of strict confidentiality regarding all the information obtained throughout the study period. Written and verbal informed consent was obtained from all respondents before data collection.

RESULTS

A total of one hundred and sixty three subjects were recruited which included one hundred and two anaemic CKD subjects and sixty one CKD subjects without anaemia as controls. The mean age of anaemic CKD subjects was 54.04 ± 14.47 years,

while those of controls was 54.92 ± 15.67 years (p = 0.717). The gender distribution among anaemic CKD subjects was male 53 (52%) and female 49 (48%), while among controls male was 31(50.8%), and female was 30(49.2%) (p = 0.888).

Table 1 showed the baseline clinical profile of all subjects.

Table 1: Baseline Clinical Profile of Subjects

Parameter	Anaemic CKD mean ± SD	Control mean ± SD	T	p- value
Age (years)	54.04 ± 14.47	54.92 ± 15.67	-0.364[a]	0.717
Weight (kg)	69.85 ± 15.65	73.26 ± 16.66	-1.314	0.191
Height (m)	1.66 ± 0.09	1.65 ± 0.09	0.534	0.594
Body mass index (kg/m^2)	25.34 ± 5.49	26.98 ± 6.17	-1.766	0.079
Systolic blood pressure (mmHg)	144.13 ± 27.01	147.33 ± 28.87	-0.706	0.481
Diastolic blood pressure (mmHg)	83.40 ± 15.36	86.75 ± 15.78	-1.318	0.189
Body surface area (m^2)	1.77 ± 0.20	1.80 ± 0.21	-0.530	0.958
Stages of chronic kidney disease				
Stage 3 n(%)	30(29.4)	41(67.2)	29.699[a]	<0.001
Stage 4 n(%)	32(31.4)	17(27.9)		
Stage 5 n(%)	40(39.2)	3(4.9)		
Dialysis				
Yes n(%)	17(16.7)	1(1.6)	8.888[a]	0.003
No n(%)	84(82.4)	60(98.4)		
Blood pressure control				
Controlled n(%)	58(56.9)	26(42.6)	2.88[a]	0.90
Uncontrolled n(%)	44(43.1)	35(57.4)		
Body mass index (kg/m^2)				
< 18	7(6.9)	2(3.3)	1.592[a]	0.661
18 – 24.9	48(47.1)	25(41.0)		
25 – 29.9	28(27.5)	21(34.4)		
>30	19(18.6)	13(21.3)		

BMI; Body mass index, SBP; Systolic blood pressure, DBP; Diastolic blood pressure, BP; Blood pressure, BSA; Body surface area, a: statistics result derived with chi square test.

Table 2: Biochemical and haematological parameters of subjects

Parameters	Anaemic CKD mean ± SD	Control mean ± SD	T	p – value
Sodium (mmol/l)	140 ± 6.51	141.33 ± 7.32	-1.176	0.241
Potassium (mmol/l)	4.99 ± 0.81	3.97 ± 0.66	4.105	< 0.001
Bicarbonate (mmol/l)	19.86 ± 4.58	22.39 ± 3.60	-3.509	0.001
Chloride (mmol/l)	102.41 ± 10.26	101.82 ± 4.83	0.407	0.685
Urea (mg/dl)	103.08 ± 66.13	51.90 ± 26.71	5.720	<0.001
Creatinine (mg/dl)	4.91 ± 4.03	2.37 ± 1.56	4.696	<0.001
Total Cholesterol (mg/dl)	191.16 ± 48.15	206.64 ± 59.63	-1.708	0.090
High density lipoprotein (mg/dl)	53.34 ± 18.69	58.73 ± 20.89	-1.616	0.108

Low density lipoprotein (mg/dl)	117.90 ± 37.32	128.09 ± 48.09	-1.428	0.155
Triglyceride (mg/dl)	112.84 ± 55.05	111.96 ± 52.28	0.095	0.924
Very low density lipoprotein (mg/dl)	21.83 ± 10.32	25.99 ± 15.81	-1.824	0.070
Packed cell volume (%)	28.48 ± 5.37	40.51 ± 3.29	-15.78	<0.001
Fasting blood sugar (mg/dl)	107.36 ± 36.71	103.37 ± 23.91	0.747	0.456
Estimated glomerular filtration rate (ml/min/1.73m^2)	22.67 ± 15.21	38.73 ± 14.79	-6.590	<0.001

Table 3 showed Prevalence and pattern of left ventricular hypertrophy and left ventricular function among participants. The overall prevalence of left ventricular hypertrophy among CKD subjects was 103(63.2%) with 95% C.I = 56.99 – 72.34. The prevalence of LVH among anaemic CKD subjects was 68(66.7%), while among CKD subjects without anaemia was 35(57.4%) (X^2 = 1.845, p = 0.174, O.R = 1.61, and 95% C.I was 0.81 – 3.17).

Table 3: Prevalence and pattern of left ventricular hypertrophy and left ventricular function among participants

Parameters	**Anaemic CKD n(%) N = 102**	**Control n(%) N = 61**	**X^2**	**O.R**	**p – value**	**95% C.I**
Left ventricular hypertrophy						
Present	68(66.7)	35(57.4)	1.845	1.61	0.174	0.81 – 3.17
Absent	34(33.3)	26(42.6)				
Left ventricular geometry						
Normal	12(11.8)	8(13.1)	2.385		0.497	
Concentric remodelling	20(19.6)	18(29.5)				
Eccentric hypertrophy	15(14.7)	9(14.8)				
Concentric hypertrophy	55(53.9)	26(42.6)				
Systolic function						
Abnormal	63(61.8)	11(18.0)	27.952	7.57	<0.001*	3.43 – 16.73
Normal	39(38.2)	50(82.0)				
Systolic dysfunction severity						
Normal	39(38.2)	50(82.0)	29.726		<0.001*	
Mildly abnormal	44(43.1)	8(13.1)				

Moderately abnormal	15(14.7)	1(1.6)
Severely abnormal	4(3.9)	2(3.3)

* $P < 0.05$

Table 4 showed the relationship between left ventricular hypertrophy and some clinical variables. However, none of the relationship was statistically significant.

Table 4: Relationship between left ventricular hypertrophy and clinical variables

	Anaemic CKD N = 102				Controls N =61			
Parameter	**LVH**	**No LVH**	**X^2**	**p value**	**LVH**	**No LVH**	**X^2**	**p value**
Age range								
< 40 years	16(15.7)	3(2.9)	6.793	0.033	10(16.4)	4(6.6)	1.83	0.40
40 – 59 years	32(31.4)	13(12.8)			8(13.1)	9(14.8)		
≥60 years	20(19.6)	18(17.7)			17(27.9)	13(21.3)		
Gender								
Male	33(32.4)	22(21.6)	3.396	0.065	15(24.6)	17(27.9)	3.27	0.07
Female	35(34.3)	12(11.8)			20(32.8)	9(14.8)		
Hypertension								
Yes	60(58.8)	24(23.5)	0.373	0.541	29(47.5)	22(36.1)	0.081	0.77
No	8(7.8)	10(9.4)			6(9.8)	4(6.6)		
Diabetes								
Yes	22(21.6)	15(14.7)	0.907	0.341	4(6.6)	2(3.3)	0.516	0.472
No	46(45.1)	19(18.6)			31(50.8)	24(39.3)		
Blood pressure control								
Controlled	33(32.4)	22(21.6)	1.77	0.183	18(29.5)	9(14.8)	0.151	0.698
Uncontrolled	35(34.3)	12(11.8)			17(27.9)	17(27.9)		
Dialysis								
Yes	16(15.7)	5(4.9)	1.86	0.173	2(3.3)	0(0)	0.740	0.390
No	52(51.0)	29(28.4)			33(54.1)	26(42.6)		
Antihypertensive								
Yes	53(52.0)	26(25.5)	0.08	0.76	29(47.5)	17(27.9)	3.745	0.053
No	15(14.7)	8(7.8)			6(9.8)	9(14.8)		

There was a significantly strong negative correlation between packed cell volume and left ventricular mass index among CKD subjects with anaemia (r = -0.345, p = 0.001), while in the control group there was a weak positive correlation (r = 0.001, p = 0.993). There was also a negative correlation between eGFR and left ventricular mass index among both CKD subjects with anaemia (r = -0.436 and p < 0.001) and controls (r = -0.363, p = 0.006), this is as shown in Table 5.

Table 5: Correlation between left ventricular mass index and selected variables among participants

	Anaemic CKD		Controls	
Parameters	**R**	**p – value**	**R**	**p – value**
Packed cell volume	-0.345	0.001	0.001	0.993
eGFR	-0.436	<0.001	-0.363	0.006
Systolic blood pressure	0.112	0.292	0.098	0.472
Diastolic blood pressure	0.212	0.043	0.078	0.567
Body mass index	-0.084	0.423	0.016	0.909
Age	-0.372	<0.001	-0.008	0.951

Furthermore, linear regression analysis revealed anaemia as a significant predictor of increased left ventricular mass index when compared to other selected variables; this is as represented in Table 6.

Table 6: Linear regression for the predictors of left ventricular hypertrophy among participants

Parameters	Unstandardized Coefficient (β)	Std error	Standardized Coefficient	T	p – value	95% C.I
Constant	152.793	35.897		4.256	< 0.001	81.813 – 223.773
SBP	-0.012	0.177	-0.008	-0.068	0.946	-0.361 – 0.337
DBP	0.452	0.316	0.159	1.433	0.154	-0.172 – 1.077
FBS	-0.139	0.120	-0.096	-1.158	0.249	-0.377 – 0.099
PCV	-1.170	0.495	-0.198	-2.362	0.020	-2.149 – -0.191
Hypertension	-6.495	11.296	-0.049	-0.575	0.566	-28.831 – 15.841

SBP; Systolic blood pressure, DBP; Diastolic blood pressure, FBS; Fasting blood sugar, PCV; Packed cell volume.

DISCUSSION

The objective of this study was to assess the prevalence and pattern of left ventricular geometry and determine its relationship with anemia, among established CKD patients seen at Lagos State University Teaching Hospital, Nigeria.

Majority of the CKD patients were in their middle age with a slight male predominance. This is in keeping with the demographics of CKD patients in previous works. [3, 5, 7, 11, 24] Most of the anaemic CKD subjects were in stage 5 with a fraction of them on hemodialysis. This is not unexpected and has been documented in previous works because declining

renal function has been associated with anemia. [10-12, 14, 25]

The overall prevalence of left ventricular systolic dysfunction was 45.4% among studied participants, with significantly higher prevalence among anaemic CKD group (61.8%), than CKD subjects without anaemia (18%) with $p < 0.001$. This was significantly higher than the prevalence of 22% for systolic dysfunction reported by Foley et al. [12, 13] This difference could be explained by the diagnostic method used for defining systolic dysfunction, in which fractional shortening was used to assess ejection fraction, and this has been shown to be unreliable in the presences of asymmetric left ventricular geometry and regional wall motion abnormality from coronary artery disease or conduction abnormality, which are common in CKD patients, and thus, not currently recommended. [23]

The overall prevalence of LVH in the study population was 63.2%. The prevalence of LVH was slightly higher among anaemic CKD subjects (66.7%) than CKD subjects without anaemia (57.4%), though not significantly different. This is slightly lower to the works of Adejumo et al and Jesurobo et al who reported a prevalence of 76 and 77.6% respectively, and much lower to reports by Ulasi et al with a prevalence of 95.5%. [9, 15, 26] The difference in the prevalence could be attributed to subject selection because their studies had more patients with hypertension as the aetiology of CKD. Conversely, the prevalence of LVH in our study was however significantly higher than reports by Chijioke et al who reported a prevalence of 27.6% using electrocardiogram. [27] Electrocardiogram has been shown to have lower sensitivity and specificity for the detection of LVH compared to Echocardiogram. [28]

Prevalence of anaemia, hypertension and LVH increased as CKD stage advanced among both groups of studied participants, with a negative correlation between eGFR and LVMI among both anaemic CKD subjects ($r = -0.436$, $p < 0.001$) and CKD subjects without anaemia ($r = -0.363$, $p = 0.006$). For instance in this study, 42.3% of CKD stage 3 patients in this study were anaemic, and 83.1% were hypertensive, compared to 93% and 90.7% prevalence of anaemia and hypertension stage 5 CKD respectively. Ijoma et al and Akinsola et al had previously reported increasing prevalence of anemia as renal function worsens. [14, 24] This may suggest that the LVH seen down the CKD groups may be a result of anaemia and hypertension or other variables. LVH has been shown to be multifactorial. [15, 16] Our findings are also similar to reports by Adejumo et al and Levine et al, that both reported increasing prevalence of LVH as CKD stages advanced. [18, 26]

The most frequent left ventricular geometric pattern seen among both anaemic CKD group and those without anaemia were concentric hypertrophy (53.9% versus 42.6%) and concentric remodelling (19.6% versus 29.5%). These findings are comparable to reports from Foley et al, who reported higher frequency of concentric LVH (39.4%) among CKD patients with anaemia. [12, 13] However, this finding is at variance with the finding of Ulasi et al, who reported higher frequency of eccentric hypertrophy among CKD patients (54.6%). [15] Though Ulasi et al focused mainly on hypertensive CKD subjects, the variance in LV geometric pattern with our study may stem from the criterion used for the classification of left ventricular geometry. The diagnostic criteria used for the measurement of left ventricular dimensions was the Penn convention [29] which is now obsolete because of its inaccuracies, while the American Society of Echocardiography and the European Association of Cardiovascular Imaging convention [23] is now the recent recommendations of most guidelines because the LVMI assessment had good correlation with cardiac MRI which is the gold standard of cardiac measurements. [23]

There was a strong negative correlation between packed cell volume and left ventricular mass index among anaemic CKD patients ($r = -0.345$, $p = 0.001$), compared to those without anaemia ($r = 0.001$, $p = 0.993$). Furthermore, linear regression analysis to control for confounders such as hypertension revealed anaemia as the only predictor that significantly increased the risk of increased left ventricular mass index (β coefficient = -1.170, $p = 0.002$, and 95% C.I = -3.879 - -0.866). Therefore, there is a strong evidence that anaemia is associated with a 1.170g/m^2 increase in left ventricular mass per 1% decrease in packed cell volume (0.33g/dl of

haemoglobin) after controlling for other confounders such as hypertension, thus anaemia contributed to the development of LVH. This has also been supported by larger studies by Foley et al, which revealed 50% increase in the risk of developing LVH and systolic dysfunction with each decrease of 1g/dl of haemoglobin. [12]

From the foregoing, anaemia has been shown to have a role in the development of left ventricular hypertrophy in CKD patients and thus, early evaluation and treatment of anaemia will go a long way at mitigating LVH, which is an independent cardiovascular risk factor, and invariably reduce the incidence of cardiovascular deaths among CKD patients.

CONCLUSION

This study showed that chronic kidney disease patients had high prevalence of anaemia, left ventricular hypertrophy and left ventricular systolic dysfunction which is worse in anemic CKD patients. The commonest left ventricular geometric pattern was concentric left ventricular hypertrophy followed by eccentric left ventricular hypertrophy.

Anaemia contributed significantly to increased LV mass, LV mass index and poor left systolic function, therefore, early management of anaemia may improve cardiovascular outcomes.

CONFLICT OF INTEREST

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REFERENCES.

1. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis. 2002;39(2 Suppl 1):S1-266.

2. Akinsola W, Odesanmi WO, Ogunniyi JO, Ladipo GO. Diseases causing chronic renal failure in Nigerians--a prospective study of 100 cases. Afr J Med Med Sci. 1989;18(2):131-7.

3. Alebiosu CO, Ayodele OO, Abbas A, Olutoyin A. Chronic renal failure at the Olabisi Onabanjo University Teaching Hospital, Sagamu, Nigeria. Afr Health Sci. 2006;6 3:132-8.

4. Stevens PE, Levin A. Evaluation and management of chronic kidney disease: synopsis of the kidney disease: improving global outcomes 2012 clinical practice guideline. Ann Intern Med. 2013;158(11):825-30.

5. Ulasi, II, Ijoma CK. The enormity of chronic kidney disease in Nigeria: the situation in a teaching hospital in South-East Nigeria. J Trop Med. 2010;2010:501957.

6. Ulasi II, Ijoma CK, Onodugo O, Arodiwe EB, Ifebunandu NA, Okoye JU. Towards prevention of chronic kidney disease in Nigeria: a community-based study in Southeast Nigeria. Kidney International. 2013;3:195-201.

7. Levin A, Djurdjev O, Barrett BJ, Burgess E, Carlisle EJF, Éthier J, et al. Cardiovascular disease in patients with chronic kidney disease: getting to the heart of the matter. Am J Kidney Dis. 2001;38 6:1398-407.

8. Bayauli MP, Lepira FB, Kayembe PK, M'Buyamba-Kabangu JR. Left ventricular hypertrophy and geometry in type 2 diabetes patients with chronic kidney disease. An echocardiographic study. Cardiovasc J Afr. 2012;23(2):73-7.

9. Jesuorobo DE, Odia JO, Uchenna DI. Left Ventricular Hypertrophy and Its Correlates in Chronic Kidney Disease Patients in a Nigerian

Tertiary Hospital. Int. J. Intern. Med. 2012;1:11-6.

10. Locatelli F, Pozzoni P, Vecchio LD, Tentori F. Effect of anaemia on left ventricular hypertrophy in end-stage renal disease. Eur. J. Heart Fail. Supplements. 2003;2:207-12.

11. Donne R, Foley R. Anaemia management and cardiomyopathy in renal failure. Nephrol. Dial. Transplant. 2002;17(SUPPL. 1):37-40.

12. Foley RN, Parfrey PS, Harnett JD, Kent GM, Murray DC, Barre PE. The impact of anemia on cardiomyopathy, morbidity, and and mortality in end-stage renal disease. Am J Kidney Dis. 1996;28(1):53-61.

13. Foley RN, Parfrey PS, Harnett JD, Kent GM, Murray DC, Barre PE. Impact of hypertension on cardiomyopathy, morbidity and mortality in end-stage renal disease. Kidney Int. 1996;49(5):1379-85.

14. Ijoma C, Ulasi I, Ijoma U, Ifebunandu N. High Prevalence of Anemia in Predialysis Patients in Enugu, Nigeria. Nephrology Research & Reviews. 2010;2(1):61-5.

15. Ulasi, II, Arodiwe EB, Ijoma CK. Left ventricular hypertrophy in African Black patients with chronic renal failure at first evaluation. Ethn Dis. 2006;16(4):859-64.

16. Di Lullo L, Gorini A, Russo D, Santoboni A, Ronco C. Left Ventricular Hypertrophy in Chronic Kidney Disease Patients: From Pathophysiology to Treatment. Cardiorenal Med. 2015;5(4):254-66.

17. Eckardt KU, Scherhag A, Macdougall IC, Tsakiris D, Clyne N, Locatelli F, et al. Left ventricular geometry predicts cardiovascular outcomes associated with anemia correction in CKD. J Am Soc Nephrol. 2009;20(12):2651-60.

18. Levin A, Thompson CR, Ethier J, Carlisle EJ, Tobe S, Mendelssohn D, et al. Left ventricular mass index increase in early renal disease: impact of decline in hemoglobin. Am J Kidney Dis. 1999;34(1):125-34.

19. Ajibare AO, Adekoya AO, Dada AO, Fagbemiro EY, Odeyemi AS, Okogun EO, et al. Cardiovascular Risk Factors: A Silent Epidemic among Urban Adult Male Socialites. J Epid Soc Nig. 2021;4(2):61-70.

20. Kouno T, Katsumata N, Mukai H, Ando M, Watanabe T. Standardization of the body surface area (BSA) formula to calculate the dose of anticancer agents in Japan. Jpn J Clin Oncol. 2003;33(6):309-13.

21. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, Jr., et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003;42(6):1206-52.

22. Heider JG, Boyett RL. The picomole determination of free and total cholesterol in cells in culture. J Lipid Res. 1978;19(4):514-8.

23. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1-39.e14.

24. Akinsola A, Durosinmi MO, Akinola NO. The haematological profile of Nigerians with chronic renal failure. Afr J Med Med Sci. 2000;29(1):13-6.

25. Singh AK, Szczech L, Tang KL, Barnhart H, Sapp S, Wolfson M, et al. Correction of anemia with epoetin alfa in chronic kidney disease. N Engl J Med. 2006;355(20):2085-98.

26. Adejumo OA, Okaka EI, Madumezia G, Okwuonu CG, Ojogwu LI. Assessment of some cardiovascular risk factors in predialysis chronic kidney disease patients in Southern Nigeria. Niger Med J. 2015;56(6):394-9.

27. Chijioke A, Makusidi AM, Kolo PM. Electrocardiographic abnormalities among dialysis naïve chronic kidney disease patients in Ilorin Nigeria. Ann Afr Med. 2012;11(1):21-6.

28. Lepira FB, Mpembe CMB, Mbutiwi FIN, Makulo JRR, Kayembe PK, Kajingulu FM, et al. Comparison of the Performance of Three Commonly Used Electrocardiographic Indexes for the Diagnosis of Left Ventricular Hypertrophy in Black Hypertensive Patients with Reduced Kidney Function Managed at a Tertiary Healthcare Hospital: A Post-Hoc Analysis. World J Cardiovasc. 2017;07:105-18.

29. Foppa M, Duncan BB, Rohde LE. Echocardiography-based left ventricular mass estimation. How should we define hypertrophy? Cardiovasc Ultrasound. 2005;3:17.