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ORIGINAL ARTICLE

Comparative Analysis of Left Ventricular Geometry in Adult Nigerians with and without Chronic Kidney Disease: Results from Ibadan CRECKID STUDY

Analyse Comparative de la Géométrie Ventriculaire Gauche chez les Nigérians Adultes Avec et Sans Maladie Rénale Chronique: Résultats de L'étude Ibadan CRECKID

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ABSTRACT

BACKGROUND: Chronic kidney disease (CKD) is associated with increased risk of cardiovascular morbidity and mortality. Left ventricular hypertrophy (LVH) is considered the strongest independent predictor of cardiovascular disease and events among CKD patients. We reported the echocardiographic left ventricular geometry in CKD patients compared to non-CKD hypertensive and apparently healthy controls in Ibadan.

MATERIALS AND METHODS: A total of 683 participants in the CRECKID STUDY comprising 220(32.2%) CKD patients, 281(41.1%) non-CKD hypertensive patients and 182(26.6%) healthy controls were included in this analysis. Basic demographic and clinical information with echocardiographic parameters were obtained.

RESULTS: Study participants in the non-CKD hypertensive group were on average older than the CKD and the healthy controls (56.2±13.1 vs 47.2±14.6, and 46.8±13.3 years, respectively; p<0.01). Compared with other groups, greater proportions of participants with CKD were men (40.5% vs.38.1% and 21.3%; p<0.0001). The left atrial and left ventricular dimensions were significantly higher in CKD compared with others. LVH was significantly more prevalent among CKD patients (68.2%) compared to hypertensive (43.9%) and normotensive (19.5%) group (p<0.01). The participants with CKD had a greater proportion of abnormal LV geometry with concentric LVH predominating (p<0.0001). Having LVH was associated with lower mean estimated glomerular filtration rate (eGFR) (40.6±37.71 vs 67±37.38, p<0.0001)

CONCLUSION: In our study, patients with CKD had the highest prevalence of abnormal LV geometry and functions. A unit decrease in eGFR was associated with increased left ventricular mass. Early detection and prompt management of abnormal LV geometry may help in reducing adverse cardiovascular outcome in patients with CKD. **WAJM 2022; 39(4): 336–342.**

Keywords: Chronic kidney disease, Hypertensives, left ventricular geometry.

RÉSUMÉ

CONTEXTE: L'insuffisance rénale chronique (MRC) est associée àrisque accru de morbidité et de mortalité cardiovasculaires. Gauche l'hypertrophie ventriculaire (LVH) est considérée comme la plus forte prédicteur indépendant des maladies cardiovasculaires et des événements chez Patients atteints d'IRC. Nous avons rapporté l'échocardiographie ventriculaire gauche géométrie chez les patients atteints d'IRC par rapport aux patients hypertendus non atteints d'IRC etcontrôles apparemment sains à Ibadan.

MATÉRIAUX ET MÉTHODES: Un total de 683 participants à la ÉTUDE CRECKID portant sur 220 (32.2%) patients atteints d'IRC, 281 (41.1 %) patients hypertendus non atteints d'IRC et 182 (26.6 %) en bonne santé ont été inclus dans cette analyse.

Démographieet clinique de base des informations avec des paramètreséchocardiographiques ont été obtenues.

RÉSULTATS: Participants à l'étude dans le groupe hypertendu non atteint d'IRC étaient en moyenne plus âgés que l'IRC et les témoins sains(56.2±13.1 vs 47.2±14.6 et 46.8±13.3 ans, respectivement; p<0.01). Par rapport à d'autres groupes, plus grande proportion de participants avec l'IRC étaient des hommes (40.5 % contre 38.1 % et 21.3 %; p<0.0001). Les dimensions auriculaire gauche et ventriculaire gauche étaient significativement plus élevées chez CKD par rapport à d'autres. La LVH était significativement plus répanduechez les patients atteints d'IRC (68.2 %) par rapport aux patients hypertendus (43.9 %) et le groupe normotensif (19.5 %) (p<0.01). Les participants avec CKD avait une plus grande proportion de géométrie LV anormale avec LVH concentrique prédominante (p<0.0001). Avoir LVH était associé à un débit de filtration glomérulaire estimé moyen plus faible (DFGe)(40.6±37.71 contre 67±37.38, p<0,0001).

CONCLUSION: Dans notre étude, les patients atteints d'IRC avaient le plus haut prévalence d'une géométrie et de fonctions LV anormales. Une diminution unitaire de Le DFG était associé à une augmentation de la masse ventriculaire gauche. Tôt la détection et la gestion rapide de la géométrie LV anormale peuvent aider à réduire les résultats cardiovasculaires indésirables chez les patients atteints de CKD. **WAJM 2022; 39(4): 336–342.**

Mots-clés: Maladie rénale chronique, Hypertensives, ventriculaire gauche géométrie.

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Abbreviations: CKD, Chronic Kidney Disease; **eGFR,** estimated Glomerular Filtration Rate **LVH,** Left Ventricular Hypertrophy.

INTRODUCTION

The burden of chronic kidney disease (CKD) is on the increase globally and driven majorly by increased prevalence of hypertension and diabetes mellitus which are major cardiovascular risk factors.[1,2] Kidney failure requiring renal replacement therapy is considered the most obvious outcome of poorly controlled blood pressure and blood glucose.[3] However, cardiovascular disease (CVD) is often associated with CKD and individuals with CKD are more likely to die from CVD events than from renal failure.[3] In the United States, the prevalence of CVD is estimated to be 69% in patients with CKD aged 66 years and above compared to 34.1% in those without CKD in the same age group.[4,5] While the burden of CKD has been well characterised among the Nigerian population, there is dearth of data on the incidence and prevalence of CVD in CKD patients.[6]

The spectrum of CVD in CKD includes; left ventricular hypertrophy (LVH) and dilatation, ischaemic heart disease (IHD) and peripheral vascular disease (PVD).[7] Aside from age, LVH is considered the strongest independent predictor of cardiovascular disease and events, cardiovascular death and total mortality.[8] Based on the Framingham study, for every 39g or standard deviation increase in left ventricular mass, there is a 40% rise in the risk of major cardiovascular event.[9] LVH is more common and more severe in CKD than in hypertensive patients with normal kidney function.[10] LVH was found to be present in over 70% of patients commencing dialysis and estimation of LV mass offers prognostic information better than other traditional cardiovascular risk factors in CKD patients.[11–13]

CVD in CKD is potentially preventable and treatable.[14] Despite this, there are no guidelines recommending echocardiography for early detection of abnormal LV geometry and aggressive management in CKD patients. There are few studies in our environment that have compared LV structure in CKD patients with those with hypertension without CKD and normal population. Many of these studies have relatively small sample size.[12]

This study aimed to assess echocardiographic left ventricular structure in CKD patients compared to hypertensive and healthy controls in Ibadan.

SUBJECTS, MATERIALS AND METHODS

Subjects and Methods

Cardiovascular and Renal events in people with Chronic Kidney Disease (CRECKID STUDY) is a prospective cross-sectional study approved by the Joint University of Ibadan/University College Hospital Ethics Committee, Ibadan, Nigeria with approval reference number UI/EC/14/0136. The main aim of the CRECKID study was to identify CKD patients at risk of major cardiovascular events for prompt management.

A total of 683 participants comprising 220(32.2%) CKD patients, 281(41.1%) non-CKD hypertensive patients and 182(26.6%) healthy controls recruited from Cardiology and Nephrology units at the medical out-patient department of the University College Hospital, Ibadan. The baseline characteristic data of all the participants were reported while the derived left ventricular structure and function measurements were limited to the participants with complete data set on echocardiography. Pre-tested questionnaires were administered by trained research assistants to collect basic demographic and relevant clinical information.

Inclusion and Exclusion Criteria

Participants aged 18 years and above with at least stage 2 CKD (eGFR = 60–89 mL/min) diagnosed since at least 3 months earlier who consented were recruited. Consenting hypertensives without CKD and normotensives were also recruited. Individuals less than 18 years, or who had kidney transplantation or refused consent were excluded from the study.

Sample Size and Power Calculation

This cross-sectional and comparative study was commenced in December 2014 following ethics committee's approval. Using a 33% prevalence of CVD in CKD[15] and a study power of 80% to detect a difference of 10% in the prevalence of cardiovascular events and death among CKD cases and controls, the estimated number of participants for the main study was a minimum of 200 participants per group. The pilot study has been published.[10]

All participants had anthropometric measurements including height, weight, waist and hip circumferences. Height was measured to the nearest centimeter using a ruler attached to the wall, while weight was measured to the nearest 0.1 kg on an electronic scale with the subject wearing light outdoor clothing and barefooted. The waist circumference was measured as midway circumference between the rib cage and the iliac crest using an anthropometric measuring tape. The mean of measured waist circumferences taken at the end of expiration, recorded to the nearest tenth of a centimetre was obtained for analysis.

Laboratory Measurements

Fasting venous blood was obtained to determine serum creatinine, components of the lipid panel (total cholesterol, high-density cholesterol, low-density cholesterol, and triglycerides), and blood glucose. Plasma glucose was measured using glucose oxidase method and lipid profile by enzymatic colorimetric method.

Blood Pressure Measurements

Blood pressure was measured using a standard Omron (HEM711DLX) blood pressure apparatus on the left arm placed at heart level after a 5-minute rest and using a cuff of appropriate size with the subject in the sitting position and legs uncrossed. According to World Health Organisation Guidelines, three BP measurements were obtained with a minimum interval of one minute with the mean of the last two used in the definition of hypertension. Office/ clinic hypertension was therefore defined as systolic blood pressure (SBP) $\geq$ 140mmHg and/or diastolic blood pressure (DBP) $\geq$ 90mmHg or being on pharmacological treatment for hypertension. The Omron apparatus had been validated with the sensitivity and specificity of 88.2% and 98.6%, respectively, to detect hypertension.[16] The measurements were performed by three

well-trained research nurses and assistants with validation of their measurements by the investigators at the onset of the study.

Estimated glomerular filtration rate (eGFR) was calculated using Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) Creatinine Equation. CKD was defined as eGFR<60ml/min/1.73m^2. CKD-EPI is widely used in patients above age 18 years and, compared with MDRD, is more accurate in a subgroup with eGFR between 60 and 120mL/min/1.73m^2. [17] Using the eGFR, participants were classified as Stage 1 with normal or high GFR (GFR > 90 mL/min), Stage 2 Mild CKD (GFR = 60–89 mL/min), Stage 3A Moderate CKD (GFR = 45–59 mL/min), Stage 3B Moderate CKD (GFR = 30–44 mL/min), Stage 4 Severe CKD (GFR = 15–29 mL/min), and Stage 5 End Stage CKD (GFR <15 mL/min).

Echocardiography

To assess cardiac structure and function, transthoracic echocardiographic examination was performed for all the participants while lying in the left lateral decubitus position using a Toshiba Xario (Toshiba Medical Systems Corp) echocardiographic machine with a 3.5HZ transducer. All measurements were obtained as recommended by the American Society of Echocardiography.[18] Left ventricular mass was indexed by the allometric power of height (LVM/Ht$^{2.7}$).[19] LV hypertrophy was defined as left ventricular mass index (LVMI) $\geq$ 49.2 g/m $^{2.7}$ in males and $\geq$ 46.7 g/m$^{2.7}$ in females.[20] Relative wall thickness (RWT) was calculated as 2 multiplied by the posterior wall thickness divided by the LV internal diameter in diastole. Increased wall thickness was taken as RWT >0.42.

Using LVMI and RWT, LV geometry was identified as normal geometry when LVMI and RWT were within limits of normal range; Concentric remodelling when there was Normal LVMI and increased RWT; eccentric hypertrophy when there was increased LVMI but RWT >0.43; and concentric hypertrophy when there was increased LVMI and RWT $\geq$0.43.[21] LV systolic function (ejection fraction) was calculated using the formula of Teicholz.[22] Two experienced cardiologists performed the echocardiography. In our laboratory, the intra-observer concordance correlation coefficient ranged from 0.76 to 0.98 while that of the inter-observer concordance ranged from 0.82 to 0.96.[23]

Data Management

Data were analysed using the Statistical Package for the Social Sciences for Windows version 22.0 (IBM, Armonk, NY, USA). Estimates were expressed as either mean values (standard deviation) for continuous variables or proportion (percentages) for categorical variables. Comparison for statistical significance was by independent students' t-test for continuous variables or chi-square for categorical variables. One-way analysis of variance with LSD post-hoc method was used to compare the demographic and echocardiographic parameters among various participants. Linear regression analysis was fitted to assess the determinants of LV mass. The level of significance was set at p> 0.05.

RESULTS

Table 1 depict the baseline characteristics of the study participants. The hypertensive without CKD group were the oldest with comparable mean age among CKD and healthy control participants (56.2±13.1 vs 47.2±14.6 vs 46.8±13.3 years, respectively, p< 0.0001). Compared with other groups, greater proportions of people with CKD were men (p<0.0001). Blood pressure parameters were comparable among hypertensives without CKD and CKD participants which were expectedly higher than the healthy control participants. Hypertensive participants had the highest mean BMI, waist and hip circumferences while the CKD participants had the highest waist-to-hip ratio. The CKD group had the highest mean serum urea, creatinine, and triglyceride with lowest mean eGFR.

As shown in Table 2, the LA and LV dimensions were significantly greater in CKD compared to other groups. Mean left atrial diameter for CKD patients, hypertensive without CKD and healthy subjects were 4.1cm, 3.6cm and 3.3cm, respectively; while the means for LVIDD were 5.0cm, 4.7cm and 4.6cm for the CKD, hypertensive without CKD and normotensive groups, respectively. As shown in Figure 1, the prevalence of LVH in CKD patients was 68.2% compared to 43.9% in hypertensive without CKD and 19.5% in healthy subjects. The increased relative wall thickness among CKD was 61.2% compared with 44.2% and 23.0% of hypertensive without CKD and normotensive patients, respectively (p<0.0001). The mean Ejection Fraction (EF) was lower in CKD group as compared

Table 1: Baseline Characteristics of the Study Participants

Variables	Hypertensive; (n=281) Mean±SD	Healthy Control; (n=182) Mean ± SD	CKD; (n=220) Mean ± SD	P-value
Female; n (%)	156(43.9%)	112(31.5%)	87(24.5%)	<0.001*
Age(years)	56.20±13.1^a	46.84±13.3^b	47.19±14.6^b	<0.001*
Systolic Blood Pressure(mmHg)	146.54±23.3^a	120.66±15.8^b	145.26±26.8^a	<0.001*
Diastolic Blood Pressure(mmHg)	90.38±14.2^a	77.71±11.7^b	90.44±18.4^a	<0.001*
Body Mass Index(kg/m^2)	26.54±6.2^a	25.70±4.3^b	25.09±4.7^b	0.001*
Hip Circumference(cm)	95.31±18.1^a	93.4±13.9^a	88.05±16.9^b	<0.001*
Waist Circumference(cm)	89.31±16.5^a	84.42±12.9^b	83.14±14.3^b	0.001*
Waist/hip ratio	0.94±0.10^a	0.91±0.1^b	0.96±0.2^a	0.001*
Urea(mg/dl)	27.21±19.8^a	17.23±5.2^{a,b}	134.33±91.9^c	0.001*
Creatinine(mg/dl)	2.11±9.6^a	0.888±0.2^a	10.04±11.2^b	0.001*
eGFR; mean±SD	70.9±30.65^a	94.2±23.85^b	14.4±19.37^c	0.001*
Total Cholesterol(mg/dl)	186.44±46.8^a	164.21±49.1^b	188.68±81.0^a	0.113
Triglycerides(mg/dl)	107.29±47.1^a	86.39±39.3^a	132.93±106.8^b	0.004*
HDL Cholesterol(mg/dl)	48.48±14.5^a	48.09±14.4^a	53.29±21.2^b	0.109
Fasting Plasma Glucose(mg/dl)	94.04±29.7^a	79.8±15.8^b	89.51±24.^{bc}	0.020*

CKD chronic kidney disease, HDL cholesterol, high density lipoprotein cholesterol
^{a,b,c,} Means with the same superscript are not significantly different at P < 0.05 by LSD
"Statistically significant; SD – Standard deviation

Table 2: Echocardiographic Parameters across each Group

Variables	Hypertensive; (n=281) Mean±SD	Normal Control; (n=182) Mean ± SD	CKD; (n=220) Mean ± SD	P-value
Left Atrial Diameter(cm)	3.65±0.6[a]	3.33±0.4[b]	4.08±0.8[c]	<0.001*
Aortic Root Diameter(cm)	2.92±0.4[a]	2.78±0.4[b]	2.99±0.4[a]	<0.001*
LVIDD (cm)	4.65±0.6[a]	4.59±0.5[a]	5.04±1.0[b]	<0.001*
LVIDS (cm)	2.89±0.7[a]	2.90±0.6[a]	3.29±1.0[b]	<0.001*
IVSTD (cm)	1.05±0.7[a]	0.94±0.5[b]	1.32±0.5[c]	<0.001*
IVSTS (cm)	1.47±01.0[a]	1.43±0.9[a]	1.69±0.5[b]	<0.001*
LVPWTD (cm)	1.12±1.1[a]	0.91±0.6[b]	1.25±0.39[c]	<0.001*
Ejection Fraction (%)	67.47±12.6[a]	67.72±7.3[a]	61.13±13.9[b]	<0.001*
Fractional Shortening (%)	38.83±9.8[a]	37.86±5.7[a]	33.87±10.0[b]	<0.001*
LV mass Index ($ht^{2.7}$)	51.72±55.9[a]	42.29±46.65[a]	70.32±36.6[b]	<0.001*
RWT (Relative Wall Thickness)	0.45±0.2[a]	0.38±0.1[b]	0.52±0.2[c]	<0.001*
Increased RWT: No (%)	112 (56.3)	98 (77.8)	45 (38.8)	<0.001*
Increased RWT: Yes (%)	87 (43.7)	28 (22.2)	71 (61.2)	<0.001*
Dilated LV; No (%)	185 (93.0)	124 (98.4)	92 (79.3)	<0.001*
Dilated LV; Yes (%)	14 (7.0)	2 (5.0)	24 (20.7)	<0.001*
LV Geometric Pattern; (%)	70 (37.0)	79 (66.9)	21 (18.8)	<0.001*
Normal Geometry				
Concentric Remodeling; %	38 (20.1)	17 (14.4)	16 (14.3)	<0.001*
Eccentric LVH; %	38 (20.1)	13 (11.0)	24 (21.4)	
Concentric LVH; %	43 (22.8)	9 (7.6)	51 (45.5)	
Left Ventricular Hypertrophy: No (%)	108 (57.1)	96 (81.4)	34 (31.8)	0.0001*
Left Ventricular Hypertrophy: Yes (%)	81 (42.9)	22 (18.6)	73 (68.2)	<0.001*

LVIDD, Left Ventricular Internal Diameter at Diastole; LVIDS, Left Ventricular Internal Diameter at Systole; IVSTD, Intereventricular Septal Thickness at Diastole; IVSTS, Interventricular Septal Thickness at Systole; LVPWTD, LV Posterior Wall Thickness at Diastole; LVPWTS, LV Posterior Wall Thickness at Systole; [a,b,c] Means with the same superscript are not different at P < 0.05; "Statistically significant

Table 3: Correlation between Anthropometric, Laboratory Parameters and LVmass

Variable	Total Population		CKD		Hypertension Control		Normal	
	rho	p-value	rho	p-value	Rho	p-value	rho	p-value
Age	0.08	0.70	0.02	0.76	0.03	0.64	0.12	0.16
Sex	–0.21	<0.001	0.05	0.57	–0.30	<0.001	–0.20	0.02
Systolic blood pressure	0.32	<0.001	0.23	0.01	0.75	0.03	0.30	0.001
Diastolic blood pressure	0.33	<0.001	0.22	0.02	0.79	0.007	0.29	0.001
Weight	0.22	<0.001	0.14	0.17	0.21	0.002	0.57	<0.001
Height	0.22	<0.001	0.05	0.58	0.21	0.004	0.26	0.004
BMI	0.05	0.41	0.12	0.38	0.11	0.29	0.18	0.11
Hip Circumference	0.18	<0.001	0.18	0.17	0.23	0.001	0.32	<0.001
Waist Circumference	0.20	<0.001	0.10	0.32	0.25	0.001	0.41	<0.001
Waist/hip Circumference	0.07	0.19	0.02	0.87	0.008	0.91	0.11	0.25
Urea	0.35	<0.001	0.12	0.28	–0.003	0.77	–0.05	0.79
Creatinine	0.30	<0.001	0.20	0.06	0.18	0.11	0.001	0.99
Triglyceride	0.12	00.9	–0.08	0.49	0.16	0.18	0.54	0.001
Fasting blood glucose	0.03	0.70	0.14	0.21	–0.16	0.18	0.18	0.30
eGFR	–0.51	<0.001	–0.24	0.01	0.28	<0.001	–0.18	0.05

rho, Correlation Coefficient

with hypertensive without CKD and healthy groups (61.1%±13.9,67.5%±12.6 and 67.7%±7.3, respectively). Also, LVMI was significantly higher in patients with CKD as compared with patients with hypertension without CKD and healthy controls (p<0.001). Participants with CKD had higher proportion of abnormal LV geometry with concentric LVH predominating. Table 3 shows the relationship between anthropometric, laboratory parameters, and LV mass. Systolic blood pressure (rho= 0.23; p-value = 0.01) diastolic blood pressure (rho = 0.22; p-value = 0.02) were positively correlated with LV mass while eGFR (rho = –0.24; p-value = 0.01) was found to be inversely correlated with LV mass among CKD patients. eGFR (β = –0.23 p-value = 0.02) stood out as the only independent predictor of LV mass in the regression model (Table 4).

DISCUSSION

In this study, four out of five participants with CKD had associated abnormal left ventricular geometry and function compared with hypertensives without CKD and normotensive cohorts. Concentric LVH predominated the abnormal LV geometry which was common among male participants. Clinic blood pressure parameters were similar among participants with CKD and non-CKD hypertensive but higher than the normal controls. Abnormal e-GFR was an independent determinant of increased left ventricular mass.

Previous studies had established the detrimental effects of abnormal LV geometry on overall outcome in people with hypertension and CKD.[24–26] It has also been suggested that individuals with CKD are more likely to die from abnormal cardiac function-related major cardio-vascular events than from the pro-gression of the diseased kidney.[27,28] Compared with eccentric LVH, concentric LVH worsens the outcome in end-stage renal disease (ESRD) due to compromised coronary reserve in the stiff and thick myocardium, reduced cardiac output, and eventual hypotension.[29] Our findings of predominant concentric LVH is then worrisome and may account for the high mortality among the studied population.

Table 4: Independent Predictors of Left Ventricular Mass among CKD Patient

Predictors	B	Std. Error	β	p-value
Age	0.13	1.17	0.01	0.11
Sex	3.13	25.39	0.01	0.90
Systolic blood pressure	0.18	0.75	0.41	0.81
Diastolic blood pressure	1.34	1.24	0.19	0.28
Waist circumference	0.95	1.60	0.11	0.56
Hip circumference	–0.26	1.76	–0.25	0.89
eGFR	–0.63	0.26	–0.23	0.02
R^2			0.13	0.05

In a multivariate linear regression model to predict left ventricular mass, a unit decrease in eGFR was associated with increased left ventricular mass ($\beta = –0.23$, $p=0.02$).

Similar to our finding of high prevalence of LVH among CKD, a study on a Nigerian population in a different settlement showed that, compared with controls, 65% of participants with CKD had LVH with 50% having concentric LVH while concentric remodelling predominated among the CKD-free hypertensive participants.[12] From our study, concentric LVH predominated among both hypertensive-without-CKD and CKD participants and the reason for this is not readily obvious. In contrast to our finding, Ulasi, *et al*[30] found eccentric hypertrophy predominating the abnormal LV geometry among pre-dialysis CKD patients. The reason for this disparity may be related to the differences in their ages and aetiologies of the CKD in the two studies. While their study population was younger (42±15.23 vs 47.19±14.6 years) with chronic glomerulonephritis as the leading aetiology, our study population was older with chronic hypertension as the main aetiology. Increasing age in patients with hypertension is associated with concentric LVH.[31] Ulasi, *et al's* finding also contradicted the studies that showed concentric LVH predomination among pre-dialysis CKD patients.[32] However, irrespective of the type, abnormal LV geometry is an independent risk factor for cardiac death with relative risk higher in concentric LVH.[11,13,33]

Studies have shown that long-term LVH leads to left ventricular dysfunction, resulting in the constriction of the renal arteries, hypoperfusion of the glomeruli, and alteration of the glomerular membrane filtration coefficient and tubular reabsorption.[34] Consistent with other studies, our participants with CKD, compared with controls, had higher cardiac chambers enlargement, lower LV ejection fraction and fractional shortening with eGFR as an independent determinant of increased LVM.[35–37] Our study findings were corroborated by another study with smaller sample size among another tribe in the country[6] From the aforementioned, cardiac structure and function are major determinants of renal function decline among patients with CKD; probably, prompt management of cardiac dysfunction may delay or prevent progression to end stage kidney disease or cardiovascular morbidity and mortality.

Cardiac remodelling and CKD interact in a vicious cycle to promote each other.[38] While the presence of LVH potentiates renal decline, renal deterioration even in the presence of normal blood pressure leads to abnormal LV geometry.[8] A population-based autopsy study has shown that lower eGFR was significantly associated with LVH, cardiomyocyte hypertrophy, and cardiac fibrosis after adjustment for age, sex, hypertension, obesity, diabetes mellitus, and haemoglobin and serum phosphate levels. In this present study, despite having comparable blood pressure with hypertensive without CKD, participants with CKD were associated with abnormal cardiac remodelling, implying additional risk factors. After adjusting for body mass index, blood pressure, gender, and age, eGFR was an independent predictor of left ventricular mass. The often less attended risk factors for cardiac remodelling in CKD are uremic toxins, oxidative stress, and fibroblast growth factor which were not the primary focus of this current study.[39,40]

Limitation

There is a need to highlight some limitations of the present study. This was a hospital-based study; thus, generalisation over the whole population should be with caution. Serial echocardiographic measurements at various CKD stages, pre- and post-haemodialysis would have helped to delineate uremic cardiomyopathy. Also, assessment of LV longitudinal strain and strain rate would have added more value to the study. Despite all these, our study had strength in the large sample size, making our findings comparable with larger multicentre studies.

CONCLUSION

Our study patients with CKD had higher prevalence of LVH, abnormal LV dimensions and function which carry high risk of cardiovascular morbidity and mortality. The early detection of left ventricular hypertrophy and both prevention of the deterioration of renal function and aggressive blood pressure control may help reduce cardiovascular morbidity and mortality in these patients. A larger longitudinal study is needed to confirm our findings.

Conflicts of Interest

There are no conflicts of interest.

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