Tropical Journal of Nephrology Vol.11 No 2, December, 2016; 67 – 80 Original article

# Prevalence and geometric pattern of left ventricular hypertrophy and function in Hypertensive Chronic kidney disease patients and Hypertensive patients without Chronic kidney disease a comparative study

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## Abstract

Systemic hypertension is a global health burden with attendant systemic complications, which manifest as hypertensive target organ damage (TOD). The spectrum ranges from stroke, chronic kidney disease (CKD), cardiovascular disease (CVD) such as left ventricular hypertrophy (LVH) and heart failure, and retinopathy. Patients with CKD have higher frequency of adverse cardiovascular outcomes than the general population. This study aims to determine the prevalence and pattern of left ventricular hypertrophy and function among hypertensive CKD patients and those without CKD.

**Methodology**: This was a cross-sectional analytical study over a two year period between January 2014 to December 2015, and recruited a total of 778 patients. And they include 300 hypertensive CKD subjects attending the nephrology clinic of the Lagos State University Teaching Hospital (LASUTH), 297 hypertensive subjects without CKD attending the hypertension clinic and 181 o healthy controls. All subjects underwent clinical, laboratory and echocardiographic evaluations, and their results were analyzed with SPSS version 20.

**Results:** The prevalence of LVH among hypertensive CKD subjects was 165(65%), and was significantly higher compared to hypertensive without CKD 93(34.1%) and controls 7(4.2%) with $x^2 = 155.34$ and $P < 0.001$. The most common left ventricular geometric pattern was concentric hypertrophy 131(50.4%) among CKD subjects, concentric remodelling 107(37.8%) among hypertensives without CKD, while normal LV was observed in 124(73.8%) healthy controls ($P< 0.001$).

The frequency of left ventricular systolic dysfunction among CKD subjects, hypertensives without CKD and healthy controls were 68(25.9%), 13(4.3%) and 2(1.2%) respectively. The predictors of increasing left ventricular mass index (LVMI) were analyzed using multiple linear regression, and identified predictors were declining estimated glomerular filtration rate (eGFR), increasing systolic blood pressure (SBP) and male gender, which accounted for 20.3%, 30.3% and 30.8% respectively of the deviation in LVMI.

**Conclusion:** This study demonstrated high prevalence of LVH and poor left ventricular function among CKD patients compared to hypertensives without CKD and healthy controls. While reduced eGFR, elevated SBP and male gender were independent predictors of left ventricular hypertrophy and dysfunction.

**Key words:** Chronic kidney disease, Hypertension, Left ventricular hypertrophy.

## Introduction

Systemic hypertension is a global health burden with attendant systemic complications, which manifest as hypertensive target organ damage(TOD), which spectrum ranges from stroke, retinopathy, chronic kidney disease (CKD) and cardiovascular disease (CVD)[1–3].

However, chronic kidney disease patients are at higher risk of developing hypertension complications, particularly those associated with TOD[4,5]. Furthermore, hypertensive CKD patients have higher prevalence of left ventricular hypertrophy than hypertensive patients without chronic kidney disease[4]. Moreover, studies have shown that the prevalence of LVH among subjects starting dialysis was 42%[6], and increases to about 75% among subjects on haemodialysis for more than 10 years[7]. Moreover, the pattern of LVH seen among hypertensive CKD patients encompass similar spectrum among both groups; however, higher frequency of eccentric hypertrophy among hypertensive CKD patients.

The explanation for the high prevalence of LVH among hypertensive CKD patients has been attributed to other non-traditional risk factors, such as uraemic cardiomyopathy, fibroblast growth factor (FGF-23), mineral bone disease, anaemia, malnutrition, and others[7,8], as shown in figure 1. However, the high frequency of eccentric LVH has been attributed to factors such as anaemia, volume overload, arterio-venous fistula, recurrent blood transfusion, inadequate haemodialysis, hypoalbuminaemia, and fluid retention[9].

Left ventricular hypertrophy is an independent risk factor for cardiac mortality among end stage renal disease (ESRD)[10]. However, CKD patients have higher incidence of cardiovascular morbidity and mortality than the general population[4,11]. Furthermore, CKD patients have higher risk of death from cardiovascular disease than progression to end stage renal disease[3]. Therefore, early identification and management of cardiovascular disease is essential in the management of CKD patients.

There are few studies in this environment that have compared the prevalence of LVH and left ventricular function between CKD and hypertensive patients[12–14], most other studies compared either hypertensive or CKD patients with healthy individuals[15–19]. Furthermore, these studies had small sample size, and their method for assessing left ventricular systolic function is not the current recommendation by guideline[20,21].

Therefore, this study aimed to determine the prevalence and geometric pattern of LVH among hypertensive CKD patients and hypertensive patients without CKD as compared to healthy controls, and to also assess their left ventricular function, which will further enlighten us on the burden of cardiovascular disease especially among CKD patients.

## Methodology

### *Study population*

This is a cross sectional analytical study that was carried out at the nephrology and hypertension clinics of Lagos State University Teaching Hospital (LASUTH), Nigeria. The study population were chronic kidney disease (CKD) patients that attended the nephrology clinic as well as hypertensive patients that attended hypertension clinic in LASUTH. .

### *Inclusion and exclusion criteria for the subjects*

Inclusion criteria were CKD patients with hypertension who have been followed up for

at least 3 months, ESRD patients with hypertension and post renal transplant patients with hypertension. While inclusion criteria for hypertension arm was hypertensive patients without evidence of CKD, and healthy control.

Exclusion criteria were CKD patients without evidence of hypertension, pregnant women and patients with valvular heart disease and patients who do not consent to participate.

***Clinical variables***

Questionnaire was used to obtained relevant information for all participants. Clinical information obtained include bio-data (age, sex), history of hypertension with duration, cardiac symptoms, history of chronic kidney disease with, duration..

Social history of alcohol intake, smoking habit, and family history of cardiovascular disease were identified. Use of antihypertensive, antiplatelet, and lipid lowering agents were documented.

Examination involved weighing of subjects using a Hansen's weighing sale, they wore light clothes with no footwear and measurements were approximated to the nearest 0.5kg. Height was measured with a stadiometer with the subject standing erect backing the stadiometer such that the occiput, back and heel makes contact with the stadiometer and measurements was in meters. Body mass index (BMI) was calculated using the formula: weight (kg)/ Height$^2$ (m$^2$). The body surface area (BSA) was also calculated using the formula BSA= (.0001)(71.84) (Weight$^{0.425}$) (Height$^{0.725}$), with weight in kilograms and height in centimetres[22]. The blood pressure was measured after five to ten minutes of rest using the accoson's sphygmomanometer with appropriate cuff size and subjects were seated comfortably. The average of two blood pressure readings taken minutes apart in the right hand was used for analyses.

Laboratory sample and analyses included random plasma glucose, fasting lipid profile (total cholesterol- TC, high density lipoprotein- HDL, low density lipoprotein- LDL, and triglycerides- TG), serum electrolyte, urea and creatinine.

***Echocardiography***

Transthoracic echocardiography (M-mode, two dimensional and Doppler) was performed with the Sonoscape echocardiographic machine, which is equipped with 3.5 MHz phased array probe (cardiac probe) transducer. It has capabilities for M-mode, two-dimensional, pulsed-wave, continuous-wave Doppler echocardiography, colour flow and tissue Doppler imaging (TDI).

All patients underwent two dimensionally guided 2D, M-mode echocardiogram and Doppler recording. Linear internal measurements of the left ventricular wall and chamber size was measured through the two dimensional mode which was translated to the M-mode echograms, perpendicular to the left ventricular long axis, and measured at the level of the mitral valve leaflet tips, and images was frozen to take measurement. Electronic callipers was positioned on the interface between myocardial wall and cavity and the interface between wall and pericardium following the American Society of Echocardiography and the European Association of Cardiovascular Imaging convention (ASE/EACI)[20].

Two cardiologists performed the echocardiograms to reduce intra-observer bias. Left ventricular mass was calculated by using an anatomically validated formula American Society of Echocardiography and

the European Association of Cardiovascular Imaging convention (ASE/EACVI)[20]:
LV mass (g)= 0.8(1.04(IVS + LVID + PWT)$^3$ – LVID$^3$) + 0.6g.
(Where IVS= interventricular septal thickness, PWT= posterior wall thickness in diastole LVID= left ventricular internal diameter, all measurement taken in diastole).
Left ventricular mass index (LVMI) was calculated by dividing the left ventricular mass in grams by body surface area[20].
Relative wall thickness (RWT) was calculated with the formula[20]:
(2 × posterior wall thickness)/(LV internal diameter at end diastole).

### *Definition of terms*

Hypertension was defined as Blood Pressure greater than or equal to 140/90 mmHg, taken on at least two occasions and/ or use of antihypertensive therapy[23].

Estimated glomerular filtration rate was estimated using Modification of Diet in Renal Disease (MDRD-4) formula which is accurate for GFR estimation[24] which is as follows

GFR (mL/min/1.73 m$^2$) = 175 x (Scr)$^{-1.154}$ x (Age)$^{-0.203}$ x (0.742 if female) x (1.212 if black).

Chronic kidney disease was defined as abnormalities of kidney structure or function present for more than 3 months with implication for health[24]. And was classified into the different 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$^2$ [24].

Left ventricular hypertrophy was defined in absolute terms as Left ventricular mass index >115 g/m$^2$ in men and >95 g/m$^2$ in women[20].

Left ventricular geometric pattern was classified as follows: Eccentric hypertrophy was defined if the relative wall thickness (RWT) is less than 0.42 in presence of LVH, while concentric hypertrophy was defined if RWT is greater than 0.42 in the presences of LVH, and concentric remodelling was defined as RWT greater than 0.42 in the absences of LVH[20].

Left ventricular systolic function was calculated using the modified Simpson method[20], and classified as follows; reference range for males was categorized as; normal was defined as left ventricular ejection fraction (LV EF) ≥ 52%, mildly abnormal was defined as LV EF between 41 – 51%, moderately abnormal was defined as LV EF between 30 – 40% and severely abnormal was defined as LV EF < 30%[20].
Reference range for females was categorized as; normal was defined as left ventricular ejection fraction (LV EF) ≥ 54%, mildly abnormal was defined as LV EF between 41 – 53%, moderately abnormal was defined as LV EF between 30 – 40% and severely abnormal was defined as LV EF < 30%[20].

Left ventricular diastolic dysfunction was defined as normal E/A ratio ≥ 0.8 and E/e′ ratio < 10, and grade I was defined as E/A ratio ≤ 0.8 and E/e′ ratio < 10, grade II was defined as E/A ratio 0.8 – 2 and E/e′ ratio 10 – 14, while grade III was defined as E/A ratio > 2 and E/e′ ratio > 14[21].

### *Statistical analysis*

The statistical methods used included frequency, percentages and bar charts to represent categorical variables, while mean, standard deviation and line graph was used to represent continuous variables. Chi square test and t- test were used to analyze categorical and continuous variables respectively. Pearson's correlation

coefficient was used to determine correlation, while multiple linear regressions were used to determine predictors of left ventricular mass index.

## Results

A total of seven hundred and seventy eight patients (778) were recruited, which included three hundred (300) hypertensive CKD patients, two hundred and ninety seven (297) hypertensive patients without CKD and one hundred and eighty one (181) healthy control subjects.

**Table 1: Showing age and gender distribution of subjects**

| Subjects | < 40 years n(%) | 40 – 59 years n(%) | ≥ 60 years n(%) | $x^2$ | p-value |
|---|---|---|---|---|---|
| **CKD** | | | | | |
| Female | 25(8.3) | 54(18) | 54(18) | 0.424 | 0.809 |
| Male | 30(10) | 63(21) | 74(24.7) | | |
| **Hypertension** | | | | | |
| Female | 19(6.4) | 105(35.4) | 49(16.5) | 5.154 | 0.076 |
| Male | 20(6.7) | 59(19.9) | 45(15.2) | | |
| **Control** | | | | | |
| Female | 39(21.5) | 51(28.2) | 17(19.4) | 1.019 | 0.601 |
| Male | 24(13.3) | 34(18.8) | 16(8.8) | | |

The mean age of hypertensive CKD subjects was 54.46 ± 15.25 years, while hypertensive subjects without CKD was 53.17 ± 11.54 years, mean age of control was 45.74 ± 13.40 years, and ($f = 25.54$ and $p < 0.001$), however post hoc analysis of mean difference between hypertensive CKD subjects and hypertensive subjects without CKD was not significant ($p = 0.569$). However, there was no significant difference in the mean duration of hypertension between CKD subjects 6.51 ± 7.05 years and hypertensive subjects 7.57 ± 7.46 years ($p = 0.203$). The mean duration of CKD is 99.84 ± 249.13 months.

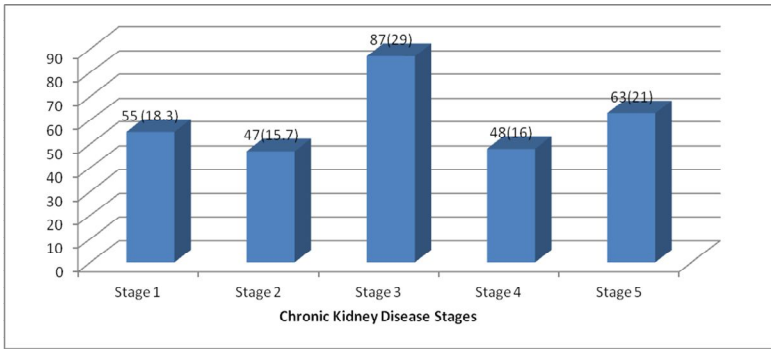


**Figure 1: Chart showing chronic kidney disease stratification.**

The most common aetiology of CKD was hypertension 119(39.7%), then diabetes 54(18%), and chronic glomerulonephritis 23(7.7%), the others are as represented in figure 2.

The gender distribution showed among CKD subjects there were 167(55.7%) males and 133(44.3%) females, among hypertensive subjects there were 124(41.8%) males and 173(58.2%) females, while among control subjects there were 74(40.9%) males and 107(59.1%) females, age and gender distribution are as shown in table 1

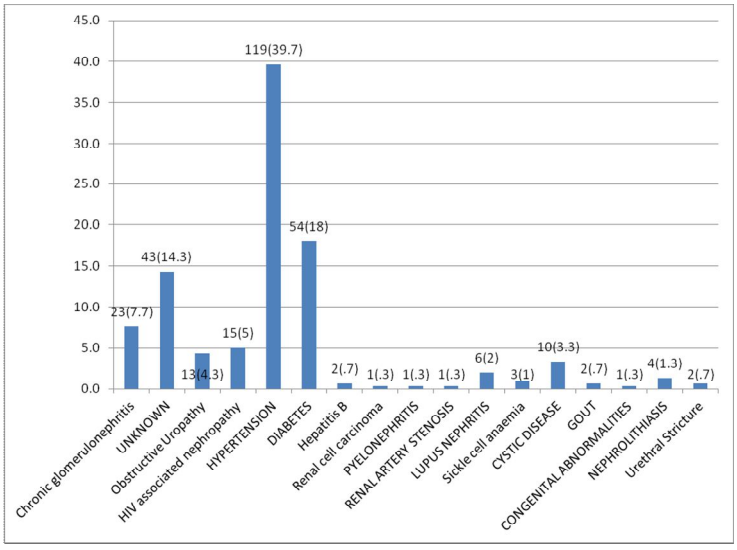


**Figure 2: Chart showing the distributions of the aetiology of chronic kidney disease.**

Duration of hypertension among CKD subject was 6.51 ± 7.05 years while among control was 7.57 ± 7.46 years (P= 0.203), mean duration of CKD was 99.84 ± 249.13 months, while figure 1 shows CKD stratification

The most common aetiology of CKD was hypertension 119(39.7%), then diabetes 54(18%), and chronic glomerulonephritis 23(7.7%), the others are as represented in figure 2.

**Table 2: Table showing the clinical and laboratory profile of subjects**

| **Parameter** | **CKD Subjects mean ± SD** | **Hypertensive Subjects mean ± SD** | **Control Subjects mean ±SD** | **F** | **P-value** |
|---|---|---|---|---|---|
| SBP(mmHg) | 143.80 ± 25.20 | 145.31 ± 2.98 | 123.63 ± 13.59 | 57.82 | < 0.001 |
| DBP(mmHg) | 85.76 ± 14.60 | 90.60 ± 13.79 | 77.94 ± 10.29 | 49.06 | <0.001 |
| BMI(kg/$m^2$) | 26.54 ± 5.74 | 29.69 ± 7.75 | 26.42 ± 4.73 | 14.89 | < 0.001 |
| Creatinine(mg/dl) | 3.77 ± 8.25 | 0.82 ± 0.25 | 0.74 ± 0.18 | 16.08 | < 0.001 |
| eGFR(ml/min/1.73$m^2$ | 43.87 ±33.68 | 108.55 ± 30.98 | 129.43 ± 34.41 | 5.95 | < 0.001 |
| Total Cholesterol(mg/dl) | 194.04 ± 62.47 | 213.01 ± 51.60 | 186.71 ± 35.58 | 8.56 | < 0.001 |
| LDL(mg/dl) | 125.40 ± 53.15 | 122.56 ± 46.33 | 114.08 ± 41.43 | 1.76 | 0.173 |
| HDL(mg/dl) | 49.77 ± 20.10 | 45.40 ± 11.75 | 43.75 ± 11.21 | 5.98 | 0.003 |
| Triglyceride(mg/dl) | 113.10 ± 58.91 | 101.12 ± 50.61 | 84.72 ± 34.91 | 9.94 | < 0.001 |
| FBS(mg/dl) | 107.25 ± 34.06 | 89.81 ± 9.66 | 86.61 ± 10.18 | 35.64 | < 0.001 |
| **BP Control** | | | | | |
| Controlled BP n(%) | 153(51.3) | 128(44.4) | 181(100) | 2.79[a] 1.32[b] | 0.095 |
| Uncontrolled BP n(%) | 145(48.7) | 160(55.6) | - | | |
| **Antihypertensive usage** | | | | | |
| **Chronic kidney disease subjects** | | | | | |
| Yes | 285(96) | 135(82.8) | - | 22.87[a] 4.93[b] | < 0.001 |
| No | 12(4) | 28(17.2) | - | | |

The mean SBP for CKD subjects was 143.80 ± 25.20mmHg, while that of hypertensive subjects was 145.31 ± 22.98mmHg, and control was 123.62 ± 13.59mmHg, there was significant

difference in the mean SBP (F = 57.82, and P< 0.001), however a Post Hoc analysis The blood pressure control profile shows no significant blood pressure control among CKD subjects 153(51.3%) than hypertensive subjects 128(44.4%) with $x^2 = 2.79$ and P = 0.095, other blood pressure parameters are as represented in table 2.showed no significant difference between CKD subjects and hypertensive subjects (P = 0.69), and other clinical profile is as shown in table 2, and laboratory parameters of subjects in table 2.

The echocardiographic parameters among subjects revealed significantly higher thickness of interventricular septum (IVS) among CKD subjects mean IVSd 12.29 ± 2.51mm, than hypertensive subjects 11.14 ±

2.03mm, and controls 8.95 ± 1.34mm, with $x^2$ = 108.16 and P < 0.001. Other echocardiographic parameters are as represented in table 3.

Left ventricular function among subjects revealed significantly lower ejection fraction among CKD subjects (EF was 60.51 ± 15.58) than hypertensive subjects (EF was 69.74 ± 8.78) and control subjects (EF was 70.87 ± 7.23), with F = 61.32 and P < 0.001. Other echocardiographic parameters are as shown in table 3, while figure 3 shows higher degree of left ventricular mass and left ventricular mass index among CKD subjects than other groups.

The prevalence of left ventricular hypertrophy (LVH) is significantly higher among CKD subjects 165(63.5%), than hypertensive subjects 98(34.1%) and control subjects 7(4.2%), with $X^2$ = 155.34 and P < 0.001, and the pattern of LVH shows higher frequency of concentric LVH among CKD subjects 131(50.4%), while concentric remodelling was the most frequent pattern among hypertensive subjects, this is as represented in table 4. Left ventricular systolic dysfunction among subject shows that CKD subject had 68(25.9%) significantly higher than hypertensive subjects 13(4.3%) and controls 2(1.2%) $x^2$= 89.46, and P < 0.001, as shown in table 4.

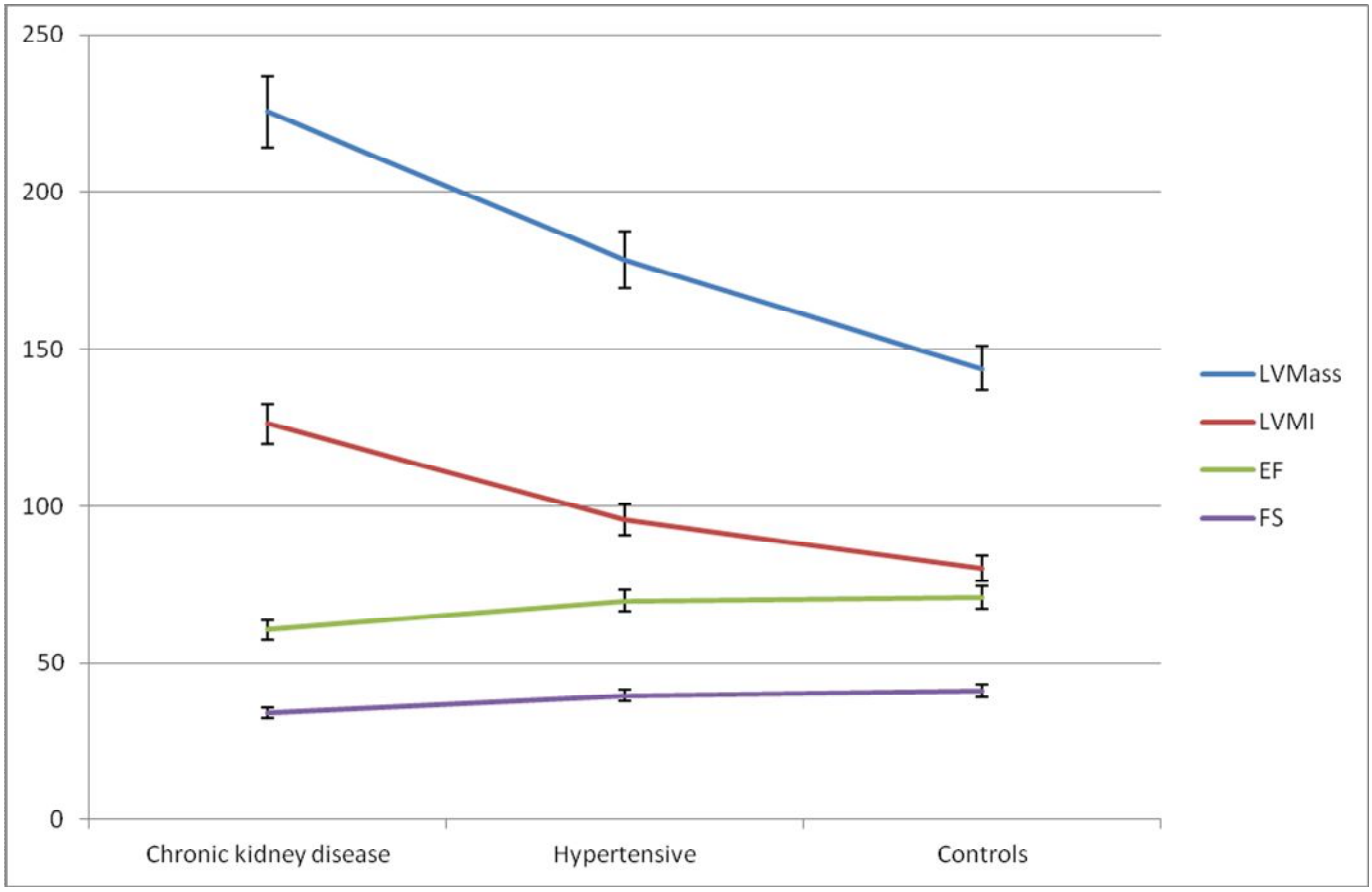


**Figure 3: Chart showing the distribution of left ventricular mass and function across different categories of subjects.**

**LVMass; Left ventricular mass, LVMI; Left ventricular mass index, EF; Ejection fraction, FS; Fractional shortening**

The severity of left ventricular systolic dysfunction among subjects revealed that only CKD subjects have severely abnormal systolic dysfunction 10(3.8%), and grade of left ventricular diastolic dysfunction among subjects revealed grade I diastolic dysfunction was the most common abnormality among both CKD subjects 159(65.2%) and hypertensive subjects 199(67.7%) than controls 53(29.4%), others are as represented in table 4.

The relationship between left ventricular mass index and estimated glomerular filtration rate (eGFR) revealed a negative correlation with correlation coefficient ($r = -0.271$ and $P < 0.001$) among CKD subjects, as shown in table 5 and figure 4.

**Table 3: Table showing echocardiographic parameters of subjects**

| Echocardiographic parameters | CKD Subjects mean ± S.D | Hypertensive Subjects mean ± SD | Controls Subjects mean ± SD | F | P – value |
|---|---|---|---|---|---|
| AoD (mm) | 31.85 ± 3.91 | 31.11 ± 3.40 | 30.28 ± 3.78 | 9.871 | < 0.001 |
| IVSd (mm) | 12.29 ± 2.51 | 11.14 ± 2.03 | 8.95 ± 1.34 | 108.16 | < 0.001 |
| IVSs (mm) | 16.71 ± 7.56 | 16.67 ± 3.32 | 14.09 ± 2.76 | 10.32 | < 0.001 |
| LVIDd (mm) | 47.28 ± 7.62 | 45.78 ± 5.18 | 45.05 ± 4.52 | 162.19 | < 0.001 |
| LVIDs (mm) | 31.79 ± 8.88 | 27.77 ± 5.36 | 26.99 ± 4.55 | 35.43 | < 0.001 |
| PWTd (mm) | 12.71 ± 8.56 | 10.50 ± 1.83 | 8.80 ± 1.13 | 28.43 | < 0.001 |
| PWTs (mm) | 17.94 ± 3.99 | 17.17 ± 2.63 | 15.18 ± 2.24 | 21.26 | < 0.001 |
| LAD (mm) | 37.16 ± 6.54 | 35.36 ± 4.34 | 32.08 ± 4.20 | 37.60 | < 0.001 |
| FS (%) | 34.28 ± 11.04 | 39.66 ± 7.33 | 41.08 ± 5.66 | 38.86 | < 0.001 |
| EF (%) | 60.51 ± 15.58 | 69.74 ± 8.78 | 70.89 ± 7.23 | 61.32 | < 0.001 |
| E- wave (m/s) | 0.64 ± 0.26 | 0.61 ± 0.17 | 0.72 ± 0.14 | 10.06 | < 0.001 |
| A- wave (m/s) | 0.67 ± 0.21 | 0.67 ± 0.15 | 0.59 ± 0.15 | 8.08 | < 0.001 |
| MV Dec T (ms) | 201.18 ± 61.24 | 211.74 ± 44.97 | 186.69 ± 35.30 | 9.21 | < 0.001 |
| IVRT (ms) | 113.38 ± 34.99 | 100.52 ± 18.79 | 89.98 ± 13.21 | 19.94 | < 0.001 |
| RWT | 0.53 ± 0.17 | 0.46 ± 0.11 | 0.39 ± 0.51 | 68.32 | < 0.001 |
| LV Mass (g) | 225.68 ± 86.03 | 178.25 ± 49.96 | 143.89 ± 35.35 | 95.44 | < 0.001 |
| LVMI ($g/m^2$) | 126.15 ± 51.17 | 95.58 ± 24.57 | 80.18 ± 17.88 | 95.49 | < 0.001 |

CKD; Chronic kidney disease, AoD; Aortic root diameter, IVSd; Interventricular septum thickness in diastole, IVSs; Interventricular septum thickness in systole, LVIDd; Left ventricular internal diameter in diastole, LVIDs; Left ventricular internal diameter in systole, PWTd; Posterior wall thickness in diastole, PWTs; Posterior wall thickness in systole, FS; Fractional shortening, EF; Ejection fraction, E – wave; Mitral peak E wave velocity, A – wave; Mitral peak A wave velocity, MV Dec T; E velocity deceleration time, IVRT; Isovolumetric relaxation time, RWT – Relative wall thickness, LV Mass; Left ventricular mass, LVMI; Left ventricular mass index.

Table 4: Table showing frequency of left ventricular hypertrophy and left ventricular geometric pattern

| **Parameter** | **CKD Subjects n(%) N=260** | **Hypertensive Subjects n(%) N=287** | **Controls n(%) N=168** | **$x^2$** | **P – Value** |
|---|---|---|---|---|---|
| **Left ventricular hypertrophy** | | | | | |
| Left ventricular hypertrophy | 165(63.5) | 98(34.1) | 7(4.2) | 155.34 | < 0.001 |
| Normal | 95(36.5) | 189(65.9) | 161(95.8) | | |
| **Left ventricular geometric pattern** | | | | | |
| Normal | 33(12.7) | 80(28.3) | 124(73.8) | 51.17 | < 0.001 |
| Concentric remodelling | 62(23.8) | 107(37.8) | 37(22) | | |
| Eccentric left ventricular hypertrophy | 34(13.1) | 28(9.9) | 5(3) | | |
| Concentric hypertrophy | 131(50.4) | 68(24) | 2(1.2) | | |
| **Systolic dysfunction (overall)** | 68(25.9) | 13(4.3) | 2(1.2) | 89.46 | < 0.001 |
| Normal | 194(74) | 283(95.6) | 179(98.9) | | |
| Mildly abnormal | 39(14.9) | 12(4.1) | 1(0.6) | | |
| Moderately abnormal | 19(7.3) | 1(0.3) | 1(0.6 | | |
| Severely abnormal | 10(3.8) | - | - | | |
| **Diastolic dysfunction (overall)** | 201(83) | 250(85) | 60(33.3) | 168.06 | < 0.001 |
| Normal | 43(17.6) | 44(15.0) | 120(66.7) | | |
| Grade I | 159(65.2) | 199(67.7) | 53(29.4) | | |
| Grade II | 28(11.5) | 48(16.3) | 6(3.3) | | |
| Grade III | 14(5.7) | 3(1.0) | 1(0.6) | | |

Table 5: Table showing correlation between left ventricular mass index and estimated glomerular filtration rate among subjects

| **Subjects** | **R** | **P – value** |
|---|---|---|
| Chronic kidney disease subjects | -0.271 | < 0.001 |
| Hypertensive subjects | 0.31 | 0.693 |
| Control subject | -0.026 | 0.804 |

**Discussion**

Hypertension is an independent risk factor for cardiovascular morbidity and mortality[2,25]. The main finding in this study were

The most common aetiologies for CKD were hypertension (39.7%), diabetes (18%) and chronic glomerulonephritis (7.7%). There was higher left ventricular mass index among CKD subjects than hypertensive subjects without CKD and controls. The prevalence of LVH was significantly

higher among CKD subjects 63.5%, than hypertensive subjects 34.1% and controls 4.2%, with $P < 0.001$. The most common geometric pattern of LVH among CKD subjects was concentric LVH 50.4%, among hypertensive subjects was concentric remodelling 37.8%, and normal among controls 73.8%. Left ventricular ejection fraction was significantly lower among CKD subjects (60.51 ± 15.58) than hypertensive subjects (69.74 ± 8.78) and controls (70.89 ± 7.23) with $P < 0.001$.

The aetiologies of CKD shows hypertension 39.7%, diabetes 18% and chronic glomerulonephritis 7.7% as the most common causes of CKD, which is similar to the spectrum reported by other studies[26–28]. This emphasizes the importance of awareness and early detection of hypertension, to reduce the burden of cardiovascular disease among patients with CKD.

There is suboptimal blood pressure control among subjects which can worsen the progression of CKD and its outcome. However, high prevalence of uncontrolled hypertension among CKD subjects has been reported in other studies with a prevalence of 71.8%, and also high prevalence of antihypertensive use[1,29–31].

The echocardiographic features among participants showed significantly higher dimensions of left atrial diameter, septal wall thickness and left ventricular posterior wall thickness among CKD subjects, than hypertensives without CKD and healthy controls. Therefore, there was a resultant higher left ventricular mass index among CKD subjects 126.15 ± 51.17g/m$^2$, than hypertensive subjects 95.58 ± 24.57g/m$^2$ and controls 80.18 ± 17.88g/m$^2$, with $P < 0.001$. Moreover, there was higher prevalence of left ventricular hypertrophy among CKD subjects 63.5%, than hypertensive subjects 34.1% and controls 4.2% with $P < 0.001$.

This findings further emphasize the reports from other studies, which has shown higher prevalence of LVH among CKD patients than hypertensive patients and controls, with prevalence ranging from 47.1% to 95.5% among CKD patients to 32.8% among hypertensive patients[12,28,32], and higher left ventricular mass index seen in CKD patients was similar to other studies[13,33]. Left ventricular hypertrophy has been established as an independent risk factor for cardiovascular disease, and increased risk of adverse cardiovascular outcomes[34–38]. Therefore, the high prevalence of LVH in CKD patients predisposes them to higher risk of cardiovascular morbidity and mortality.

However, the most common pattern of left ventricular geometry seen among CKD subjects was concentric hypertrophy 50.4%, while among hypertensive subjects was concentric remodelling. This is similar to reports by Cerasola et al[32] and Mpembe et al[33]. However, this is in contrast to reports by Ulasi et al[12], who reported a higher prevalence of eccentric LVH (54.6%) among CKD predialysis patients. The reasons for the difference may not be understood, but could be attributed to the diagnostic criteria for left ventricular geometric pattern.

The prevalence of left ventricular systolic dysfunction among CKD subjects was 25.9%, which was significantly higher than hypertensive subjects 4.3% and controls 1.2% with $P < 0.001$. This emphasizes poor left ventricular function among CKD patients, and it is comparable to other studies which reported prevalence range of 15 to 30%[13,33,39–41].

The prevalence of left ventricular diastolic dysfunction was significantly higher among

both CKD subjects (83%) and hypertensive subjects (85%), than controls (33.3%) with P< 0.001. Although, higher than similar studies that reported a prevalence of 62.8% to 66.6% among CKD patients[14,39], and 62% to 79.1% among hypertensive patients without CKD[14,16,17]. The reasons for this disparity could be attributed to the old criteria used for establishing diastolic dysfunction in the other studies.

However, left ventricular systolic and diastolic dysfunction has been established as an independent predictor for cardiovascular mortality among patients with CKD, and other cardiovascular disease[42–45]. The proposed pathophysiology was that reduced systolic function is associated with diastolic dysfunction which can contribute to the progression of heart failure by limiting cardiac output and reserve, and acceleration of neuro-endocrine activation; which will further worsen difficulty in breathing, dyspnoea at rest, thus promoting physical inactivity, deconditioning and frailty, and therefore increase cardiovascular morbidity and mortality[45,46].

Furthermore, there was a negative correlation between estimated glomerular filtration rate and left ventricular index (r = -0.271 and P <0.001); while a positive correlation between eGFR and left ventricular ejection fraction (r = 0.171 and P = 0.08). Further analysis for predictor of left ventricular mass index showed declining eGFR and BMI among CKD subjects, and increasing systolic blood pressure among hypertensive subjects, and male gender among controls. This is similar to report from other studies[12,15,28,33,47].

This study is not without its limitation, in which other non-traditional risk factors for LVH among CKD subjects such as FGF-23, mineral bone disease, mineral bone disease, anaemia, and other risk factor could not be assessed because of cost, availability and standardization of FGF-23 in this environment. Furthermore, cohort study would have been essential to follow up the impact of therapy, adequacy of dialysis and management of other non-traditional risk factors in these patients.

In conclusion, this study has demonstrated the high prevalence of LVH among CKD patients, with poor left ventricular function as compared to hypertensive subjects and controls. Therefore, attention should be geared towards the cardiovascular status of CKD patients for early identification of declining cardiovascular function, and prompt therapy should be instituted.

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