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Original Article

Left Ventricular Geometry and Left Ventricular Hypertrophy Phenotype in Newly Diagnosed Hypertension in North-eastern Nigeria

***Ibrahim Abubakar Galtimari[1,2], Chiroma Ijuptil[2,3], Sulaiman Mohammad Maina[2,4], Faruk Buba[1,2], Mohammed Abdullahi Talle[1, 2], Albert Imhoagene Oyati[5,6].**

[1]Cardiology Unit, Department of Internal Medicine, University of Maiduguri Teaching Hospital, Nigeria. [2]Department of Medicine, Faculty of Clinical Sciences, College of Medical Sciences, University of Maiduguri, Nigeria. [3]Endocrinology Unit, Department of Medicine, Internal Medicine, University of Maiduguri Teaching Hospital, Nigeria. [4] Nephrology Unit, Department of Internal Medicine, University of Maiduguri Teaching Hospital, Nigeria. [5]Department of Medicine, Faculty of Clinical Sciences, College of Medical Sciences, Ahmadu Bello University, Zaria, Nigeria. [6]Cardiology Unit, Department of Medicine, Ahmadu Bello University Teaching Hospital, Shika, Nigeria.

Abstract

Background: Left Ventricular Hypertrophy (LVH) is a key component of hypertensive heart disease. The prevalence of hypertensive LVH ranges from 19% to 48 % in untreated hypertensive patients amongst the Western population but is 46% to 63% in Nigeria. The study aims to highlight the prevalence of LVH and to determine the pattern of LV geometry and the LVH phenotype in newly diagnosed hypertensive patients.

Methodology: The study was cross-sectional, and observational between June 2019 and June 2021. The study population comprised 300 newly diagnosed hypertensive adult patients aged 18 years and above, and 300 Healthy age, sex-matched non-hypertensive adults as control groups. An echocardiography was performed and the diagnostic criteria for LVH, LV Geometry and LVH phenotype were used based on the American Society of Echocardiography and the European Association of Cardiovascular Imaging.

Results: The total number of study participants was 600, three hundred newly diagnosed hypertensive patients and three hundred normotensive controls. The male participants comprised 180 (60%) of the newly diagnosed hypertensive cases and 120 (40%) of the normotensive controls, while the female participants accounted for 168 (56%) of the hypertensive group and 132 (44%) of the control group, respectively. Overall, 59% of newly diagnosed hypertensive patients had LVH. Concentric LVH was the commonest LV geometry with a prevalence of 37% among newly diagnosed hypertensive patients. Fifty-five point four per cent (55.4%) of newly diagnosed hypertensive patients had concentric non-dilated hypertrophy.

Conclusion: LVH is highly prevalent and occurs in more than half of newly diagnosed hypertensive patients. The commonest LV geometry is concentric hypertrophy, and the LVH phenotype of concentric non-dilated Hypertrophy accounts for more than half of LVH.

Keywords: Left Ventricular Hypertrophy; Hypertensive Heart Disease; Left Ventricular Remodeling; Left Ventricular Geometry.

***Correspondence:** Dr. Ibrahim Abubakar Galtimari, Department of Medicine, Faculty of Clinical Sciences, College of Medical Sciences University of Maiduguri, Nigeria. **Email**: dr.galtimari@gmail.com

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Quick Response Code:

Introduction

Left Ventricular Hypertrophy (LVH) is a major and one of the earliest complications of systemic arterial hypertension[1,2]. LVH is a strong predictor of cardiovascular morbidity and mortality such as heart failure, coronary artery disease and stroke[3,4]. It is a key component of hypertensive heart disease[4], and several factors influence the increase in the prevalence of LVH among hypertensive. These include obesity, older age, duration of hypertension, valvular heart diseases and chronic kidney disease[5,6].

The prevalence of hypertensive LVH ranges between 19%-48% in untreated hypertensive patients and 58%-77% in high-risk hypertensive patients in a review of echocardiographic data of 37700 individuals amongst the Italian population[5]. The prevalence of LVH in newly diagnosed hypertensive patients in Nigeria ranges between 46% to 63%[7–9].

The LV geometry is classified into normal geometry, concentric remodeling (CR), concentric hypertrophy (CH) and eccentric hypertrophy (EH). This classification is based on the LV mass index and Relative Wall Thickness (RWT)[10,11].

Left ventricular hypertrophy (LVH) is a compensatory mechanism in response to increased blood pressure and afterload. LVH helps to reduce wall stress and initially maintains left ventricular systolic function. However, if the elevated blood pressure and afterload continue and overwhelm the heart's compensatory abilities, the left ventricular chamber may eventually dilate. This dilation can lead to impaired left ventricular ejection fraction and contribute to the development of systolic heart failure over time[12].

In recent years, left ventricular hypertrophy (LVH) has been categorised into concentric dilated hypertrophy (CDH), concentric non-dilated hypertrophy (CNH), eccentric dilated hypertrophy (EDH) and eccentric non-dilated hypertrophy (ENH)[13]. It has been observed that eccentric dilated hypertrophy, concentric non-dilated hypertrophy, and concentric dilated hypertrophy had an increased risk of CVD events among African Americans [13].

ECG is the most accessible and cost-effective option to diagnose LVH but its utility is limited due to its low sensitivity, making it a good starting point but not a definitive diagnostic tool[14]. The echocardiogram is the test of choice in establishing the diagnosis of LVH, it is more accurate than an electrocardiogram (ECG)[15].

There is a paucity of data on LV geometry and LVH phenotype among never-treated hypertensive patients in North-Eastern Nigeria. Therefore, the study aims to highlight the prevalence of LVH and to determine the pattern of LV geometry and the LVH phenotype in newly diagnosed hypertensive patients.

Methodology

This was a hospital-based, cross-sectional, observational study. The study population comprised 300 consecutively newly diagnosed hypertensive adult patients aged 18 years and above who presented to the Cardiology Clinic from June 2019 to June 2021. The methodology includes details on anthropometric measurements, blood pressure assessment, biochemical analysis, and echocardiographic evaluation, as well as ethical clearance, which was previously published [16].

The inclusion criteria for the study encompassed adults aged 18 years and older with an average office blood pressure (OBP) of ≥140/90 mmHg. Exclusion criteria included individuals with hypertension currently on antihypertensive medications, valvular heart disease, an estimated glomerular filtration rate (eGFR) below 60 ml/min/1.73 m², diabetes mellitus, or suboptimal echocardiographic image quality.

Echocardiography

Participants underwent evaluation in the left lateral decubitus position, with images captured from standard parasternal and apical views using second-harmonic two-dimensional imaging. M-mode echocardiography was employed to measure left ventricular dimensions, interventricular septal thickness (IVST), and left ventricular posterior wall thickness (LVPWT) in the parasternal long-axis (PSLAX) view. The left ventricular mass (LVM) was calculated using the Devereux regression formula. Left ventricular dilation is indicated by a Left Ventricular end-diastolic diameter (LVEDD) equal to or greater than 53mm in females and 59mm in males. A normal LV chamber is characterised by an LVEDD of less than 53mm in females and less than 59mm in males[17,18].

Definition of LVH and LV geometry

Left ventricular hypertrophy, (LVH) was defined by LV mass indexed to body size: to body surface area (BSA) or height or height to the allometric power of 2.7. Cut-off criteria for LVM/BSA(g/m^2) of greater than 115 g/m^2 and greater than 95g/m^2 were considered left ventricular hypertrophy (LVH) for males and females respectively[18]. Similarly, LVH was considered when LVM-indexed to a height greater than 99g/m and greater than126g/m for females and males respectively. The cut of the criterion of LVH ($g/m^{2.7}$) was considered LVM indexed to the allometric power of 2.7 of greater than 44 ($g/m^{2.7}$) and 48($g/m^{2.7}$)[19]. Relative wall thickness (RWT) was calculated using the formula $(2PWTd)/LVIDd$). This was used for the categorisation of an LVH as either concentric (RWT ≥0.42) or eccentric (RWT <0.42) hypertrophy. Concentric remodeling was considered when LVM was normal with increased RWT while those with normal LVM and RWT <0.42 were considered to be normal LV geometry[19]. The concentric LVH (CH) was further divided into concentric dilated hypertrophy (CDH) and concentric non-dilated hypertrophy (CNH) by the LV chamber dilation, likewise, the Eccentric LVH (EH) was also classified as eccentric dilated hypertrophy (EDH) and eccentric non-dilated hypertrophy (END) whether the LV is dilated or not[18].

Data analysis

The Statistical Package for Social Science (SPSS) software version 26 (Chicago, Illinois, USA) was used for the statistical analysis were expressed as mean ± SD after using the Kolmogorov–Smirnov test for normal distribution. The student's t-test was used to compare the mean ± SD of the newly diagnosed hypertensive patients and the non-hypertensive controls. At the same time, the categorical variables were expressed as absolute values and percentages and χ^2 was used to determine the difference. Analysis of variance (ANOVA) one way was used to determine the difference among the LV geometry and LVH phenotype. A *P* value of ≤0.05 was considered significant.

Results

The total number of study participants was 600, three hundred newly diagnosed hypertensive patients and three hundred normotensive controls. The males constitute 180 (60%) and 168 (56%) and females accounted for 120 (40%) and 132 (44%) of the newly diagnosed hypertensive patients and the normotensive controls respectively. There was no significant difference in the mean age of the study groups (46.1±11.2 versus 46.7±12.4, p =0.93). The mean weight was significantly higher amongst the hypertensive group than the control group (72.6±15.7 versus 66.9±10.1, p = 0.020). There was no significant difference in mean height of the hypertensive and normotensive group (1.687±0.076 versus 1.667±0.076, p = 0.995), the mean BMI of the hypertensive group was higher than the controls (25.6±5.6 versus 23.6±3.7, p = 0.008) and the mean BSA was significantly higher in the hypertensive group than the normotensive group (1.83±0.25 versus 1.75±0.14 p = 0.030).

The mean SBP was significantly higher amongst the newly diagnosed hypertensive patients (150.1±9.7 versus121.4±8.4, p<0.001), a similar pattern was observed in the DBP (93.6±4.7 versus 76.2±5.6, p<0.001), the PP (56.4±9.2 versus 45.2±9.3, p <0.001) and the MABP (112.52 ± 11.9 versus 91.2 ± 9.5, p < 0.001) as summarised in table 1.

Table 1. The demographic and clinical characteristics of the study population

Characteristics	Newly diagnosed hypertension (n=300)	Non-hypertensive control (300)	P- value
Male	(180) 60%	(120) 40%	
Female	(168) 56%	(132) 44%	0.384
Age	46.1±11.2	46.7±12.4	0.930
Weight (Kg)	72.6±15.7	66.9±10.1	0.020
Height (m)	1.687±0.076	1.667±0.076	0.995
BMI(Kg/m^2)	25.6±5.6	23.6±3.7	0.008
BSA(m^2)	1.83±0.25	1.75±0.14	0.030
PR(b/min)	83.3±13.5	82.6±7.9	0.699
SBP(mmHg)	150.1±9.7	121.4±8.4	<0.001
DBP(mmHg)	93.6±4.7	76.2±5.6	<0.001
PP (mmHg)	56.4±9.2	45.2±9.3	<0.001
MABP (mmHg)	112.4±5.2	91.2±9.5	<0.001

BMI: Body mass index: BSA: Body surface area; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; PP: Pulse pressure; MABP: Mean arterial blood pressure

Table 2. demonstrates the echocardiographic characteristics of the study population, there was no significant difference between the two groups in the LVEDd (47.7±6.8 versus 46.4±4.1, p=0.200), the LVEDs (31.7±6.9 versus 29.9±4.5, p = 0.550), the IVSDs (14.8±3.2 versus 14.3±1.8, p = 0.255), the LVPWDS (14.8±3.2 versus 14.5±1.7, p = 0.377), the LVEF (67.3±10.5 versus 70±4.4, p = 0.082) and the LVFS (34.4±7.7 versus 34.4±3.0, p = 0.999). The mean LVM, LVMI and RWT were statistically significantly higher among the newly diagnosed hypertensive patients than the normotensive control (222.2±77.4 versus 178.2±43.9, p<0.001), (144.5±35.0 versus 103.8±27.6, p = 0.044) and (0.449±0.12 versus 0.413±0.063, p = 0.054) respectively.

Table 2. Echocardiographic characteristics of the study population

Characteristics	Newly diagnosed hypertension (n=300)	Non-hypertensive control (300)	*P*
LVEDd(mm)	47.7±6.8	46.4±4.1	0.200
LVEDs(mm)	31.7±6.9	29.9±4.5	0.550
IVSDd(mm)	10.2±2.1	9.5±1.5	0.033
IVSDs(mm)	14.8±3.2	14.3±1.8	0.255
LVPWDd(mm)	10.8±2.5	9.5±1.2	0.001
LVPWDS(mm)	14.8±3.2	14.5±1.7	0.377

LVEF(%)	67.3±10.5	70±4.4	0.082
LVFS(%)	34.4±7.7	34.4±3.0	0.999
LVM(Kg)	222.2±77.4	178.2±43.9	<0.001
LVM/BSA(Kg/m^2)	144.5±35.0	103.8±27.6	0.044
RWT	0.449±0.12	0.413±0.063	0.054

LVEDd: left ventricular end-diastolic diameter; LVEDS: left ventricular end-systolic diameter; IVSDd: interventricular septum diameter in diastole; IVSDs: interventricular septum diameter in systole; LVPWDd: left ventricular posterior wall diameter in diastole; LVPWDs: left ventricular posterior wall diameter in systole; LVEF: left ventricular ejection fraction; LVFS: left ventricular fraction shortening; LVM: left ventricular mass; RWT: relative wall thickness.

The distribution of LVH[LVM/BMI(g/m^2)] was 58.8% and 20.0% among newly diagnosed hypertensive patients and the normotensive controls, the distribution of LVH [LVM/Height(g/m)] was 59.3% and 14.3% among the hypertensive and normotensive group. The prevalence of LVH [LVM/Height$^{2.7}$(g/m$^{2.7}$)] was 63.0% and 15.0% among the newly diagnosed hypertensive patients and normotensive control respectively (χ^2 =29.537, p<0.001) as summarised in table 3.

Table 3. Comparison of the LVH determined by various methods

LVH by Various methods	**Newly diagnosed hypertension (n=300)**	**Non-hypertensive control (n=300)**	***χ²***	***p***
LVH/BSA(g/m^2)	173 (58.8%)	60(20%)		
LVH/Height(g/m)	178(59.3%)	43(14.3%)	29.537	<0.001
LVH/Height$^{2.7}$(g/m$^{2.7}$)	189(63%)	45(15%)		

LVH: left ventricular Hypertrophy; BSA: Body Mass Index

The mean IVSDd was observed to be significantly different across the left ventricular geometry (p = 0.001), similarly, the mean LVPWDD was also found to be a statistically significant difference across the LV geometry (p< 0.001). The mean of IVSDS and LVPWDS were significantly different across the group with a p-value of 0.010 and 0.001 respectively. The mean LVEF and LVFS were statistically different across the LV geometry. The mean LAA was observed to be not different across the group (p = 0.095). There were significant differences across the LV geometry in the mean of LVM (p = 0.001), the mean of LVM/BSA (P < 0.001), the mean of LVM/Height (p< 0.001) and the mean of the LVM/Height$^{2.7}$ (p< 0.001), as summarised in table 4.

Table 4. The left ventricular geometry of newly diagnosed hypertensive patients

Variables	**Normal LV Geometry (n=51)**	**Concentric Remodeling (n=72)**	**Concentric LVH (n=111)**	**Eccentric LVH (N=66)**	***F***	***P***
IVSDD(mm)	8.6±1.1	10.1±2.6	11.1±2.0	10.2±2.1	6.158	0.001
LVPWDD(mm)	8.1±3.0	11.0±2.0	11.9±2.0	11.0±1.7	11.807	<0.001
IVSDS(mm)	13.5±2.4	14.1±2.9	16.2±3.3	14.5.1±3.2	4.019	0.01
LVPWDS(mm)	12.6±2.3	14.5±3.2	16.3±3.2	14.7±2.9	6.036	0.001
LVEF(%)	61.4±8.7	67.8±10.7	71.9±8.3	64.1±11.8	5.352	0.002

LVFS(%)	33.4±5.9	34.6±9.0	36.7±6.9	31.3±7.7	2.487	0.065
LVIDD(mm)	46.5±4.3	44.2±4.8	46.1±6.9	55.1.±4.6	18.301	<0.001
LAA(m^2)	13.5±2.5	12.3±2.1	14.0±3.7	14.2±2.6	2.182	0.095
LVM(g)	176.5±49.4	198.7±86.5	233.5±76.1	264.4±61.0	6.101	0.001
LVM/BSA(g/m^2)	100.1±28.9	103.8±44.5	128.2±35.5	143.1±25.6	7.524	<0.001
LVM/HT(g/m)	105.8±28.3	115.9±47.2	138.7±42.5	154.1±30.3	6.687	<0.001
$LVM/HT^{2.7}$ ($g/m^{2.7}$)	44.4±11.3	46.7±17.6	57.4±15.9	62.0±10.6	7.408	<0.001

IVSDD: interventricular septum diameter in diastole; LVPWDd: left ventricular posterior wall diameter in diastole; IVSDS: interventricular septum diameter in systole; LVPWDS: left ventricular posterior wall diameter in systole; LVEF: left ventricular ejection fraction; LVFS: left ventricular fraction shortening; LIVDD: left ventricular internal diameter in diastole; LAA: left atrial area; LVM: left ventricular mass

The mean IVSDd was found to be significantly different across the LVH phenotype ($p< 0.001$), similarly, the mean LVPWDD was also observed to be a statistically significant difference across the LVH phenotype ($p = 0.001$). The means of IVSDS and LVPWDS were significantly different across the group with a p-value of 0.003 and 0.001 respectively. The mean LVEF was not statistically different across the groups, while the mean LVFS was statistically different across the LVH phenotype. The means LAA was observed to be different across the group ($p = 0.031$). There were significant differences across the LVH phenotype in the means of LVM ($p < 0.001$) and the mean LVMI ($p < 0.001$), as summarised in Table 5.

Table 5: The Left Ventricular Hypertrophy phenotype of newly diagnosed hypertensive patients

LVH Phenotype	**Concentric dilated hypertrophy (n=13)**	**Concentric non-dilated hypertrophy (n=98)**	**Eccentric dilated hypertrophy (n=14)**	**Eccentric non-dilated hypertrophy n=52**	***F***	***P***
IVSDD(mm)	10.5±1.0	11.4±2.5	10.0±2.0	10.2±2.1	5.604	<0.001
LVPWDD(mm)	11.9±1.6	11.9±2.1	11.8±1.6	10.6±2.4	4.800	0.001
IVSDS(mm)	16.9±1.6	16.2±3.4	16.4±3.8	13.9±3.0	4.406	0.003
LVPWDS(mm)	16.9±1.6	16.3±3.4	16.8±3.4	14.0±3.2	5.106	0.001
LVEF(%)	68.5±5.7	72.3±8.5	68.8±7.0	62.8±13.0	3.461	0.011
FS(%)	32.5±4.6	37.1±7.0	33.6±4.1	30.3±8.6	2.398	0.056
LVIDD(mm)	59.7±2.9	44.3±4.9	61.4±2.5	53.6±3.2	35.093	<0.001
LAA(m^2)	17.0±0.4	13.5±3.7	13.3±2.0	14.7±3.7	2.791	0.031
LVM(g)	332.2±64.9	224.3±71.4	323.9±100.8	245.6±32.6	11.282	<0.001
LVMI(g/m^2)	164.3±33.5	121.8±32.8	159.4±26.1	138.0±20.6	26.043	<0.001

Figure 1 depicts the distribution of the LV geometries, overall 59% and 61% of newly diagnosed hypertensive patients had LVH and increased RWT respectively. The concentric LVH was observed to be 37%, and the eccentric LVH accounted for 22% of the newly diagnosed hypertensive patients. Normal LV geometry accounted for 17% while concentric remodeling was found to be 24% of the newly diagnosed hypertensive patients.

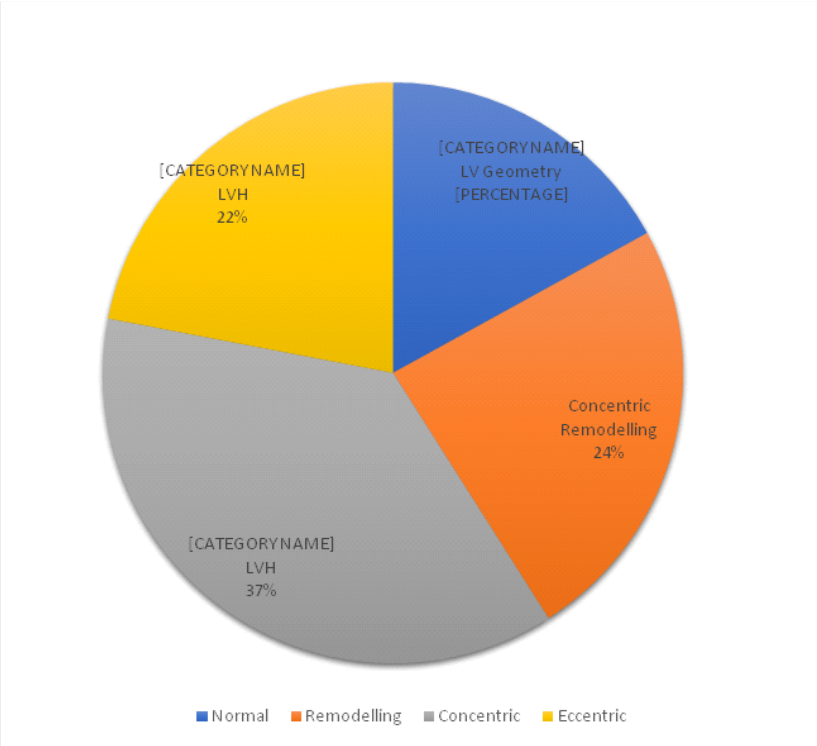

Figure 1. Distribution of various Left Ventricular Geometry among newly diagnosed hypertensive patients.

Figure 2 shows the distribution of the LVH phenotype, 55.4% and 7.3% of newly diagnosed hypertensive patients had concentric non-dilated hypertrophy and concentric dilated hypertrophy respectively. The eccentric non-dilated hypertrophy was observed to be 29.4%, and the eccentric dilated hypertrophy accounted for 7.9% of the newly diagnosed hypertensive patients with LVH.

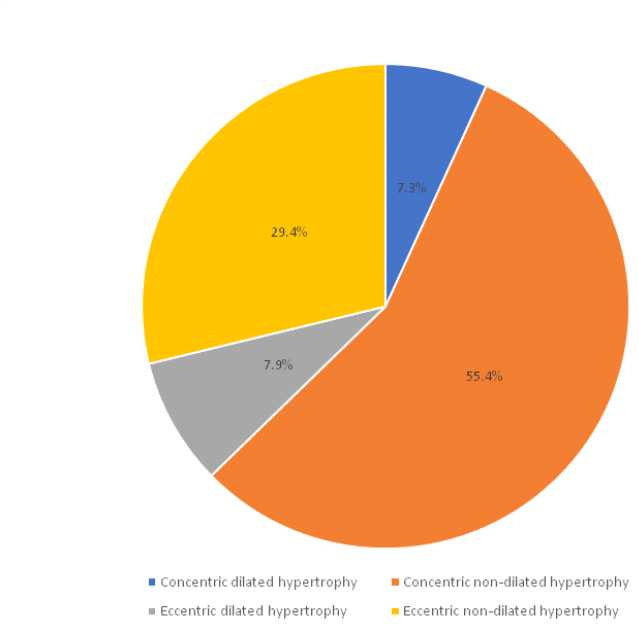

Figure 2. Distribution of various Left Ventricular Hypertrophy phenotype among newly diagnosed hypertensive patients.

Discussion

This study shows a high prevalence of LVH with concentric Hypertrophy as the predominant LV geometry in newly diagnosed hypertensive patients. Concentric non-dilated hypertrophy (CNH) is the common LVH phenotype in this category of patients.

The gender and age distribution of the subjects and controls were similar. The prevalence of LVH among newly diagnosed hypertensive patients was 58.8%, 59.3% and 63.0% using LVM/BSA, LVM/Height and LVM/Height$^{2.7}$ respectively. Ajayi *et al* reported similar findings among hypertensive patients in southwestern Nigeria[20]. A similar prevalence (61.8%) was found by Karaye*et al* in a multicentre study in Kano northwest Nigeria[7], while in the northcentral region of Nigeria, Adamu *et al* also found a similar prevalence (62.7%)[8]. The prevalence of echocardiographic LVH in newly diagnosed hypertension is found to be lower in the southern region of Nigeria[9,21]. However, in the Western world, the prevalence of echocardiographic LVH is much lower[5], probably due to increased awareness and early screening of the population for hypertension.

It has been observed that the risk of developing LVH depends on the severity of hypertension not only the duration [22]. This high prevalence underscores the importance of public awareness of early detection and management of hypertension to prevent the development of adverse cardiac remodeling and associated complications.

In this study, the most common LV geometry among the patients with LVH was CH which accounts for 35% of the newly diagnosed hypertensive patients, followed by EH accounting for 24%. Similar patterns were reported in Nigeria[8,9]. However, Karaye *et al* found different patterns in their study which showed that EH was the common geometry[7]. The difference is probably due to the duration of hypertension, the study population in the Karaye *et al* were long-standing hypertensive patients. Long-standing hypertension is likely to cause LV remodeling leading to dilated LV diameter, unlike hypertension with short duration which may cause LV muscle hypertrophy leading to CH. This pattern which indicates an increased LV wall thickness and reduced or normal LV size is considered a more severe form of LV remodeling and is associated with a higher risk of cardiovascular events[23].

The study also found that 17% of newly diagnosed hypertensive patients exhibited a normal LV geometry, indicating that not all newly diagnosed hypertensive individuals develop LV structural abnormal remodeling (CR). This subgroup may have a lower risk of adverse cardiovascular outcomes compared to those with LVH[11].

We found concentric non-dilated hypertrophy (CNH) to be a common LVH phenotype (55.4%) among newly diagnosed hypertensive patients, followed by eccentric non-dilated hypertrophy with prevalence of 29.4%, the CDH and EDH accounted for 7.3% and 7.9% respectively. This is in contrast with Khouri *et al*, who found the common LVH phenotype was ENH which accounted for 52.3% among long-term hypertensive African Americans, followed by CNH which accounted for 40.3%, the EDH and CDH observed in the 6% and 1.5%[13].

This difference might be due to the duration of hypertension. The concentric non-dilated ventricular hypertrophy (CNH) is a compensatory mechanism in response to increased blood pressure and afterload. When the mechanism is overwhelmed, the heart's compensatory abilities fail, and the left ventricular chamber may eventually dilate hence eccentric dilated hypertrophy (EDH)[12].

This study's findings highlight the various LVH phenotypes, including concentric dilated hypertrophy, concentric non-dilated hypertrophy, eccentric dilated hypertrophy, and eccentric non-

dilated hypertrophy are associated with unique patterns of left ventricular remodeling, wall thickness, left ventricular function, and LV mass. The differences in parameters (interventricular septal, posterior wall thickness, left ventricular size, LV mass, ejection fraction, and fractional shortening) suggest that each LVH phenotype has specific structural and functional characteristics.

The limitation of the study was the inability to do Cardiac Magnetic Resonant imaging (MRI) to rule out other causes of LVH such as hypertrophic cardiomyopathy and athlete's heart.

Conclusion

Overall, our findings suggest that a considerable proportion of newly diagnosed hypertensive patients exhibit LV structural changes, with 59% showing evidence of LVH. The common LV geometry is concentric hypertrophy, and the LVH phenotype of concentric non-dilated Hypertrophy accounts for more than half of LVH.

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