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Ultrasonic Measurement of Left Ventricular Mass Index of Hypertensive and Normotensive Adults in a Tertiary Hospital in South-South Nigeria

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Hypertension is a known cause of heart disease which may manifest as an increase in left ventricular mass index. Echocardiography is a painless, safe and reliable test using sound waves for measuring Left Ventricular Mass (LVM) from which Left Ventricular Mass Index (LVMI) can be derived.

Aims and Objectives: This study was aimed at evaluating and comparing the ultrasonic measurement of the left ventricular mass index of hypertensive patients and normotensive participants and correlating the Left Ventricular Mass Index (LVMI) to the Body Mass Index (BMI) and Gender in the study populations.

Methodology: This was a prospective hospital-based case-control study of adult hypertensive participants and normotensive controls. Echocardiography was done with measurements of the Left

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Ventricular Mass (LVM) obtained. The LVMI of the study groups was correlated to their BMI and gender.

Results: The mean BMI was statistically higher in the hypertensive group $(25.98 \pm 4.40 \text{ kg/m}^2)$ (p-value=0.001).The mean Left Ventricular Mass Index (LVMI) was significantly higher in the hypertensive group $(79.59 \pm 61.67 \text{g/m}^{2.7})$ than in normotensive group $(55.36 \pm 34.77 \text{g/m}^{2.7})$ (p-value 0.0001). In this study, the high BMI category (obese/overweight) was the strongest predictors of LVMI above 51g/m^{2.7} (p-value= 0.0001) and females were almost 1.8 times more likely to have increased LVMI than men

Conclusion: This study revealed that the female gender, BMI and hypertension were significant predictors of increased left ventricular mass index. High BMI category (obese/overweight) was the strongest predictors of LVMI above 51g/m^{2.7}. Periodic ultrasonic measurement of the ventricular mass index will help in early detection and reducing this common and potentially modifiable risk that leads to increased morbidity and mortality.

Keywords: Hypertension; normotensive; left ventricular mass index; left ventricular hypertrophy.

1. INTRODUCTION

Left ventricular mass index is a relationship between the left ventricular mass to the body size of an individual to determine if the person has left ventricular hypertrophy [1]. Left ventricular mass index of more than 51g/m^{2.7} in an adult defines left ventricular hypertrophy [2]. This can be calculated as left ventricular mass (measured using Ultrasound scan) divided by the individual's height^{2.7}, where 2.7 is an exponent [3]. It is a common and potentially modifiable cardiovascular risk factor overlooked in clinical practice that leads to morbidity and mortality [3].

Causes of left ventricular hypertrophy include pressure overload as seen in hypertension and aortic stenosis; volume overload which may be due to ventricular septal defect, aortic or mitral regurgitation; abnormal heart wall as in hypertrophic cardiomyopathy and left ventricular aneurysm [4]. Other causes of LVH are genetic predisposition [3] and physiologic activities [4]. Usually hypertension results in increase of left ventricular wall thickness with or without increase in size of the cavity (left ventricular internal diameter) [5].

The prevalence of left ventricular hypertrophy using echocardiographic measurement is 16% in men and 19% in women; that is (i.e.) nearly 1 of every 5 American adult in the general population [6]. However, it is two times more frequent in blacks than in white hypertensive patients who have similar arterial blood pressure [7-9]. In Nigeria, the prevalence of left ventricular hypertrophy measured by echocardiography is also high with values of 34% and 1.67% in the hypertensive and normotensive adult populations respectively [10]. Akpa MR et al. [11] in their study showed that the prevalence of hypertension in adults in Port Harcourt, Rivers State, Nigeria was 40.82% in the general public.

Echocardiography is the modality of choice for assessing left ventricular hypertrophy [5]. When using echocardiography to assess for LVH, it is important to use left ventricular mass index (LVMI) instead of just left ventricular wall thickness [3]. Using wall thickness alone is not a good indicator and it tends to underestimate left ventricular hypertrophy in women and overestimate it in men [4]. Meanwhile, left ventricular mass indexed to height to the power 2.7 [LVM/h^{2.7}] is a better assessment of left ventricular hypertrophy in individuals with high body mass index and in those with hypertension [12]. In addition to hypertension, BMI and gender are also thought to affect the left ventricular mass index [13].

This study aims at evaluating the relative impact of body mass index and gender on the left ventricular mass index of hypertensive and normotensive adults as well as determining the ultrasonic measurement of Left Ventricular Mass Index in adults with essential hypertension and that of non-hypertensive adults.

2. MATERIALS AND METHODS

2.1 Study Site

This study was conducted in the Radiology Department of University of Port Harcourt Teaching Hospital (UPTH), a tertiary hospital in southern Nigeria over a period of 12 months (January 2019-January 2020).

2.2 Study Design

This was a prospective case control study. The study population involved adult participants (males and females) with essential hypertension (case group) recruited from the consultant cardiology clinic of the Medical Out-Patient Department and apparently normal nonhypertensive adult volunteers (control group).

The echocardiographic measurement of left ventricular mass indexed to height was correlated with body mass index and gender in hypertensive participants and in normotensive controls. The results in both groups were compared using graphs, tables and Charts.

Inclusion criteria for participants were adult males and females who were known to have essential hypertension. While that for the control group were non-hypertensive and non-diabetic adults.

Participants noted to have any valvular defects or other cardiac defects during the scanning process were excluded from the study.

Exclusion criteria for participants were those having other causes of hypertension other than idiopathic or essential hypertension, like preeclampsia, pheochromocytoma or renovascular hypertension., active smokers, diabetic patients, those with clinical evidence of target organ damage by arterial hypertension such as stroke, coronary heart disease, heart failure and renal impairment.as well as evidence of heart disease other than hypertension and chronic lung disease. Other exclusion criterial used were competitive athletes, steroids use and pregnancy or lactating women.

Participants excluded from the study from the control group were active smokers, diabetic and hypertensive patients, those with evidence of cardiovascular disease and the presence of chronic lung disease or renal impairment, other exclusion criterial used were competitive athletes, steroids use and pregnancy or lactating women.

The consent was obtained with a signed form, confidentiality was ensured. Demographic data such as sex, age, weight, height, occupation and medical history were obtained using a structured interview form.

2.3 The Technique of Echocardiography

All the transthoracic ultrasound scans were performed using 3.5 MHz linear-array cardiac

transducer on a MINDRAY diagnostic ultrasound machine (Model: DC-8, SN – QE 3B001806, Year: 2013).

With a chaperon present in the ultrasound scan room, participants were asked to remove clothing from the upper body and covered by sheet of cloth to keep them comfortable and maintain privacy of females. Coupling gel was applied on the chest over the region of the heart to displace air and allow good contact between the transducer and the skin. The room was dark for better visualisation of the image on the screen. The participants were scanned lying down on the couch at left decubitus position.

The cardiac probe was placed at the left sternal edge in the left 3rd, 4th or 5th intercostal space with the marker oriented towards the right clavicle of the subject (approximately 11 o'clock position). This is known as the Parasternal Long Axis View (PLAX view).

During the scan, participants were sometimes asked to breathe slowly or hold their breath briefly as this helped to obtain higher quality images. The cursor was placed perpendicular to the long axis of the left ventricle approximately at the mitral valve leaflet tips. M-mode images were derived from two-dimensional (2D) images of the left ventricle at end inspiration from the PLAX view. The measurements were made according to the American Society of Echocardiography (ASE) leading edge-to-leading edge criteria [14]. All measurements were obtained at end diastole. the frame in the cardiac cycle in which the cardiac dimension was largest. The following left ventricular measurements were taken: Interventricular Septum Thickness in Diastole (IVSTD), Left Ventricular Internal Diameter in Diastole (LVIDD) and Posterior Wall Thickness in Diastole (PWTD). Each of the measurements was taken in three cardiac cycles, and average of the three values was calculated.

Using transthoracic two-dimensional (2D) guided M-mode echocardiography; Left ventricular mass was calculated with the corrected American Society of Echocardiography (ASE) formula described by Devereux et al. [15].

Left ventricular mass index was calculated as Left ventricular mass divided by the patient's height^{2.7} where 2.7 is an exponent (allometric sign) [16,17].

Left ventricular hypertrophy or increased left ventricular mass index was considered when Left

ventricular mass index exceeded 51g/m^{2.7} in both males and females.

2.4 Calculation of Left Ventricular Mass and Left Ventricular Mass Index

Left ventricular mass was calculated using the corrected American Society of Echocardiography (ASE) formula described by Devereux et al¹⁷ which has been shown to have closely related with values Left ventricular mass at necropsy as well as good inter-study reproducibility:

LVM (ASE) = $0.8 \times [1.04 \times (IVSTD + LVIDD + PWTD)^{3} - (LVIDD)^{3}] + 0.6 g.$

Where IVSTD is Interventricular Septum Thickness in Diastole, LVIDD is Left Ventricular Internal Diameter in Diastole and PWTD is Posterior Wall Thickness in Diastole.

Left ventricular mass index was calculated as Left ventricular mass divided by the participant's height^{2.7} where 2.7 is an exponent (allometric sign) [16]:

 $LVMI = LVM / Height^{2.7}$.

2.5 Data Analysis

Data was analyzed using IBM statistical Package for Social Sciences (SPSS) Windows Version 20; Chicago, IL, USA. Results were presented as mean ± standard deviation, percentages, tables, and graphs as appropriate. Means compared using Student's *t*-test. Pearson's correlation was used to assess the association between findings, sociodemographic factors and BMI. P < 0.05considered statistically significant. Binary logistic regression analysis was performed to determine the significant predictors of high LVMI and control for confounding influence. LVMI (categorized as >51g/m^{2.7}/≤51g/m^{2.7}) was the dependent variable while gender, blood pressure status (hypertensive/normotensive) and BMI $(\geq 25.0 \text{kg/m}^2/<25.0 \text{kg/m}^2)$ status were the independent variables in the logistic regression model. Odds ratio and 95% confidence interval were computed to determine the strength of association between the dependent and independent variables. A two tailed p value of less than or equal to 0.05 was considered statistically significant.



Fig. 1 showing two-dimensional (2D) guided M-mode echocardiographic measurements of left ventricular dimensions where point A to B is interventricular septum thickness in diastole (IVSTD), point B to C is Left Ventricular Internal Diameter in Diastole (LVIDD) and point C to D is Posterior Wall Thickness in Diastole (PWTD)

3. RESULTS

The study was conducted using 300 adults, made up of 150 hypertensive participants and 150 controls. In the overall study population, there were 147(49%) males and 153 (51%) females (Table 1). Among the hypertensive group, 72(48%) were males and 78(52%) were females while there were 75(50%) males and 75(50%) females among the control group (Table 1).

The age range of this study population was from 35years to 85 years with the mean ages of the hypertensive participants being 54.4 ± 16.2 years in males and 53.6 ± 15.0 years in females while

that of normotensives were 48.4 ± 13.8 years in males and 48.8 ± 14.3 years in females. These were not statistically significant (P-value 0.747 and 0.785 for hypertensives and normotensives respectively) (Table 2).

The mean BMI for the hypertensive group was 25.98 ± 4.40 while that of the normotensive group was 24.35 ± 3.88 ; which was statistically significant (p-value=0.001) (Table 4). Among the hypertensive group, 18% was obese while 8.7% of the normotensive participants were obese (Table 3). In the general population, 33.3% were overweight while 13.3% were obese (Table 3).

Table 1. Age and sex distribution of cases (hypertensives) and controls (normotensives	Table 1. Age and sex distribution of cases ((Hypertensives) and controls ((Normotensives)
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Hypertensives (n=150)	Normotensives (n=150)	Total (N=300)
n (%)	n (%)	n (%)
37 (24.7)	57 (38.0)	94 (31.3)
33 (22.0)	36 (24.0)	69 (23.0)
26 (17.3)	25 (16.7)	51 (17.0)
21 (14.0)	11 (7.3)	32 (10.7)
15 (10.0)	10 (6.7)	25 (8.3)
18 (12.0)	11 (7.3)	29 (9.7)
value =0.069		
72 (48.0)	75 (50.0)	147 (49.0)
78 (52.0)	75 (50.0)	153 (51.0)
lue =0.729	· ·	. ,
	Hypertensives (n=150) n (%) 37 (24.7) 33 (22.0) 26 (17.3) 21 (14.0) 15 (10.0) 18 (12.0) value =0.069 72 (48.0) 78 (52.0) lue =0.729	Hypertensives (n=150) n (%)Normotensives (n=150) n (%) $37 (24.7)$ $57 (38.0)$ $33 (22.0)$ $36 (24.0)$ $26 (17.3)$ $25 (16.7)$ $25 (16.7)$ $21 (14.0)$ $11 (7.3)$ $15 (10.0)$ $10 (6.7)$ $18 (12.0)$ $18 (12.0)$ $11 (7.3)$ $11 (7.3)$ value =0.06972 (48.0) $75 (50.0)$ $78 (52.0)$ 75 (50.0) lue =0.729

Table 2. Comparison of male and female ages by categories

Category	Males Mean age ± SD	Females Mean age ± SD	t	P-value	
Hypertensive	54.4 ± 16.2 years	53.6 ± 15.0 years	0.323	0.747	
Normotensive	49.4 ± 13.8 years	48.8 ± 14.3 years	0.273	0.785	
SD-Standard deviation					

Table 3. Body mass index categories of cases and controls

BMI Category	Hypertensive n (%)	Normotensive n (%)	Total n (%)
Underweight	3 (2.0)	8 (5.3)	11(3.7)
Normal	69 (46.0)	80 (53.3)	149 (49.7)
Overweight	51 (34.0)	49 (32.7)	100 (33.3)
Obese	27 (18.0)	13 (8.7)	40 (13.3)
Total	150 (100.0)	150 (100.0)	300 (100.0)

Chi Square =8.025; p value =0.046

Table 4. Comparison of body mass index values across cases and controls

Category		Body Mass Index		
		Mean ± Standard deviation		
Hypertensive		25.98 ± 4.40		
Normotensive		24.35 ± 3.88		
	t=3.401; p-value=0.001*	*Statistically significant		

3.1 Comparison of Left Ventricular Mass Index (LVMI) Values and Categories Across Cases and Controls

The mean LVMI was $79.59 \pm 61.67 \text{g/m}^{2.7}$ for hypertensives and $55.36 \pm 34.77 \text{g/m}^{2.7}$ for normotensives which was statistically significant (p-value 0.0001) (Table 5). The mean LVMI of the hypertensive group was almost two times that of the normotensive group (odds ratio 1.96; 95% confidence interval: 1.24 – 3.11) as seen in Table 6 (p value = 0.004).

3.2 Correlation between BMI (kg/m²) and LVMI (g/m^{2.7}) among Hypertensives and Normotensives

There is a significant positive correlation (r=0.469; p-value = 0.0001) between the left ventricular mass indexed to height^{2.7} (LVM/height^{2.7}) and the Body Mass Index (BMI) among the hypertensive group (Fig. 2). Similar but weaker correlation (r = 0.282; p-value = 0.0001) is seen in the LVM/height^{2.7} and the BMI of the normotensive participants (Fig. 3).

3.3 Comparison between BMI categories and LVMI in Hypertensives and Normotensives

The obese BMI category of both hypertensive and normotensive groups had high percentage of occurrence of increased LVMI with p values of 0.0001 and 0.025 respectively which were both statistically significant (Table 7). The percentage of increased LVMI was higher in the obesehypertensive group (92.6%) than in the obesenormotensive participants (76.9%) as also seen in Table 7.

3.4 Comparison between Gender and LVMI in Cases and Controls

Table 8 showed that among the hypertensive group, gender was significantly associated with increased LVMI (p-value = 0.002) where more females (67.9%) had increased LVMI than males (43.1%). Meanwhile, there was no significant association between gender and LVMI among the normotensive participants.

Table 5. Comparison of left ventricular mass index (LVMI) values across cases and controls

Category		LVMI	
		Mean ± Standard deviation	
Hypertensive		79.59 ± 61.67	
Normotensive		55.36 ± 34.77	
	t=4.191; p-value=0.0001*	*Statistically significant	

Table 6. Comparison of category of left ventricular mass index (LVMI) among cases and controls

Category	LVMI values			
	>51 g/m ^{2.7} n (%)	≤ 51 g/m ^{2.7} n (%)	Total n (%)	
Hypertensive	84 (56.0)	66 (44.0)	150 (100.0)	
Normotensive	59 (39.3)	91 (60.7)	150 (100.0)	
Total	143 (47.7)	157 (52.3)	300 (100.0)	

Odds ratio=1.96 (95% confidence interval: 1.24 – 3.11); Chi Square =8.352; p value =0.004* *Statistically significant

Table 7. Comparison between bmi categories and lvmi in cases and controls

BMI category	Hypertensive		Normotensive	
	>51 g/m ^{2.7} n (%) ≤ 51 g/m ^{2.7} n (%)		>51 g/m ^{2.7} n (%)	≤ 51 g/m ^{2.7} n (%)
Underweight	0 (0.0)	3 (100.0)	3 (37.5)	5 (62.5)
Normal	26 (37.7)	43 (62.3)	26 (32.5)	54 (67.5)
Overweight	33 (64.7)	18 (35.3)	20 (40.8)	29 (59.2)
Obese	25 (92.6)	2 (7.4)	10 (76.9)	3 (23.1)
Total	84 (56.0)	66 (44.0)	59 (39.3)	91 (60.7)
	Chi Square=29.457; p value =0.0001*		Chi Square =9.320; p value =0.025	

*Statistically significant



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Fig. 2. Correlation between bmi (kg/m²) and lvmi (g/m^{2.7}) among hypertensives





Gender	Hypertensive		Normotensive		
	>51 g/m ^{2.7} n (%)	≤ 51 g/m ^{2.7} n (%)	>51 g/m ^{2.7} n (%)	≤ 51 g/m ^{2.7} n (%)	
Male	31 (43.1)	41 (56.9)	27 (36.0)	48 (64.0)	
Female	53 (67.9)	25 (32.1)	32 (42.7)	43 (57.3)	
Total	84 (56.0)	66 (44.0)	59 (39.3)	91 (60.7)	
	Chi Square=9.416; p value =0.002*		Chi Square =0.698	8; p value =0.403	
	$+\mathbf{O}(-i)$				

Table 8. Comparison between gender and lvmi in cases and controls

*Statistically significant

Table 9. Logistic regression of predictors of high LVMI values above 51g/m^{2.7}

Gender	B coefficient	Odds ratio	95% CI	P value
Female	0.605	1.83	1.13 – 2.97	0.014*
Male ^R				
BMI category				
Obese/overweight	1.104	3.02	1.86 – 4.89	0.0001*
Not obese/overweight	R			
Blood pressure categ	gory			
Hypertensive	0.608	1.84	1.13 – 2.98	0.014*
Normotensive ^R				
Constant	-1.086			0.001
R-Re	eference category CI-Confid	ence interval *Statisti	cally significant	

R-Reference category CI-Confidence interval *Statistically significant

3.5 Logistic Regression of Predictors of High LVMI Values above 51g/m^{2.7}

This study revealed that gender, BMI and blood pressure were significant predictors of LVMI above 51g/m^{2.7} (Table 9).

Females were 1.83 times more likely to have increased LVMI than males (Odds ratio 1.83; 95% confidence interval 1.13 – 2.97) (Table 9).

Also in Table 9, Obese/overweight BMI category was about 3 times more likely to have LVMI above $51g/m^{2.7}$ than the not obese/overweight category (odds ratio 3.02; 95% confidence interval 1.86 – 4.89). In this study, the high BMI category (obese/overweight) was the strongest predictors of LVMI above $51g/m^{2.7}$ (p-value= 0.0001).

When LVMI was correlated with blood pressure categories (Table 9), being hypertensive was also seen to be a significant predictor of high LVMI above $51g/m^{2.7}$ (p value = 0.014).

4. DISCUSSION

Left ventricular mass index which is a relationship of the left ventricular mass to the body size of an individual to determine if the person has left ventricular hypertrophy [1] is thought to be affected by hypertension, Body

Mass index (BMI) and gender [13]. Sorof et al. [2] defined increase in left ventricular mass index of more than 51g/m^{2.7} to be left ventricular hypertrophy, where 2.7 is an exponent.

The age range of this study population was from 35years to 85 years with the mean ages of the hypertensive participants being 54.4 ± 16.2 years and 53.6 ± 15.0 years in males and females respectively while that of normotensives were 48.4 ± 13.8 years and 48.8 ± 14.3 years in males and females respectively. These groups were comparable because their mean ages were not statistically significant (P-value 0.747 and 0.785 for hypertensives and normotensives respectively) (Table 2).

This study showed that 56% of the hypertensive subjects had increased left ventricular mass index of more than 51g/m^{2.7}. This corresponds with the 30.9% to 56.0% prevalence of increased left ventricular mass index among hypertensive subjects reported by Adewale et al. [18] in their study "Echocardiographic partition values and prevalence of left ventricular hypertrophy in hypertensive Nigerians" at the cardiology clinic of the University College Hospital Ibadan, Nigeria.

Hypertension was found to be an independent significant positive predictor of left ventricular mass index (p = 0.014) in this study. This is in

keeping with the finding in a study by Kuperstein et al. [19] "The importance of age and obesity on the relation between diabetes and left ventricular mass". Among hypertensive participants in this study, 56% were found to have increased LVMI as >51g/m^{2.7} of against 39.3% of the normotensive adults (p=0.004). This showed that left ventricular hypertrophy is commoner in adults with essential hypertension than in normotensive adults and this concurred with the study "left ventricular hypertrophy as a predictor of coronary heart disease mortality and the effect of hypertension" by David et al. [20] Also in this study, hypertensive adults were found to have significantly higher mean LVMI than the nonhypertensive adults (79.59 ± 61.67 and 55.36 ± 34.77 respectively; p-value = 0.0001) with the hypertensive group being almost two times likely to have LVMI of more than 51g/m^{2.7} than the normotensive group (odds ratio 1.96; 95% confidence interval: 1.24 - 3.11).

Body mass index (BMI) did not have independent relationship to LVM indexed to height^{2.7} in a study "Correlates of left ventricular mass in hypertensive Nigerians: an echocardiographic study" carried out by Ogah et al. [21] on a group of hypertensive Nigerian adults seen at the University College Hospital (UCH), Ibadan, Nigeria. In contrast, the index study showed significant positive correlation between body mass index and left ventricular mass indexed to height^{2.7} in both hypertensive and normotensive groups (p = 0.0001 in both groups), though stronger significant positive correlation was seen among the hypertensive group than in the normotensive participants. This however is in agreement with the study done by Fox et al. [22] in both male and female hypertensive groups (P = 0.0001) where BMI as an independent predictor of LVM/height^{2.7} was greater in high BMI groups for a given blood pressure. BMI was also seen to be a significant predictor of LVMI among normal subjects of Ibo descent in Nigeria in a study "Linear regression models for quantitative assessment of left ventricular function and structures using M-mode" by Okwor CA et al. [23].

Left ventricular hypertrophy was found in 52% of obese-hypertensive and 30% of normal-weight hypertensive group as against 14% in the obesenormotensive and 5% in the normal-weight normotensive group in a study by De Simone et al. [13]. In this study, 92.6% of obese hypertensive and 37.7% of normal-weight hypertensive groups as against 76.9% of obese normotensive and 32.5% of normal weight

normotensive groups had increased LVMI. This showed that being obese and at the same time hypertensive increases the chance of having increased LVMI. Similarly, several studies have shown that hypertension and obesity have additive effect on left ventricular mass index [13,22,24,25]. Meanwhile, this study showed that high BMI category alone (obese/overweight) had stronger prediction of having increased LVMI than hypertension alone (p= 0.0001 and 0.014 for high BMI category and hypertension respectively). In the index study, the obese BMI category had the highest frequency of LVMI of $>51g/m^{2.7}$ in both hypertensive and normotensive groups (Table 7). This frequency is higher in the obese-hypertensive group (92.6%) than in the obese-normotensive participants (76.9%). Similarly, in the study by De Simone et al. [13], obesity was found to be an independent stimulus to left ventricular mass index in the normotensives. Obese/overweight BMI category of the index study was about 3 times more likely to have LVMI above 51g/m^{2.7} than the not obese/overweight category (odds ratio 3.02; 95% confidence interval 1.86 – 4.89). Also in this study, the high BMI category (obese/overweight) was the strongest predictors of LVMI above 51g/m^{2.7} (pvalue= 0.0001).

Gender had a statistically significant positive correlation (p = 0.002) with LVMI in the hypertensive group of this study while there was no significant correlation (p= 0.403) between gender and LVMI for the normotensive group in this study. Females were almost 1.8 times more likely to have increased LVMI than men (Odds ratio 1.83; 95% confidence interval 1.13 - 2.97). Meanwhile, a study done by De Simone et al. [13] "Relation of obesity and gender to left ventricular hypertrophy in normotensive and hypertensive adults" showed that in both hypertensive and normotensive groups, obesity was associated with increase in left ventricular mass index in both gender but more in women than in men. Also in both gender there was almost equal significant positive linear correlation between body mass index and left ventricular mass index in a study by Rashid et al. [25] (P=0.00) while Gardin et al. [26] reported positive association between increased left ventricular mass index and being male in their study in healthy black and white adult men and women.

5. CONCLUSION

Ultrasonic measurement of Left ventricular mass index was significantly higher in the hypertensive

than the non-hypertensive adults with mean left ventricular mass index being 79.59 ± 61.67 and 55.36 ± 34.77 in the hypertensive and the non-hypertensive individuals respectively; p-value = 0.0001.

The high BMI category (obese/overweight) was the strongest predictors of LVMI above 51g/m^{2.7} (p-value= 0.0001). There was a stronger positive significant correlation between the Left Ventricular Mass Index (LVMI) and Body Mass Index (BMI) in hypertensive adults than in nonhypertensive group of this study.

Gender had a statistically significant positive correlation (p = 0.002) with LVMI in the hypertensive group while among the non-hypertensive group there was no significant correlation (p=0.403) between gender and LVMI. Females were almost 1.8 times more likely to have increased LVMI than men (Odds ratio 1.83; 95% confidence interval 1.13 – 2.97).

Therefore this study revealed that gender (being female), BMI (obese/overweight category) and blood pressure (hypertensive group) were significant predictors of LVMI above 51g/m^{2.7}.

6. RECOMMENDATION

Studies should be conducted in other regions of Nigeria and Africa to expand the body of knowledge of the relationship of left ventricular mass index to body mass index and gender in hypertensive and normotensive adults.

Echocardiography to measure left ventricular mass index should be routinely done for hypertensive adult males and females especially those with high body mass index category (obese/overweight) as this could help in early management decision, forecast of outcome and development of measures aimed at preventing left ventricular hypertrophy.

7. LIMITATION

Hypertensive participants with well controlled blood pressure who were included in the hypertensive group might have affected the result of this study as they may have normal left ventricular mass index. This is because increase in left ventricular mass index which is a complication of hypertension is reversible when blood pressure is well controlled [15].

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

Ethical approval was obtained from Hospital Ethical Committee before commencement of the study.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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