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Original Article

Prevalence of echocardiographic left ventricular hypertrophy among hypertensives in a tertiary health institution in Nigeria

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ABSTRACT

Objective: Systemic hypertension remains an important risk factors for cardiovascular diseases and a major global public health problem. Left ventricular hypertrophy (LVH) is a recognized complication of hypertension and strongly predicts cardiovascular morbidity and mortality. In Nigeria, few studies evaluated the role of echocardiography in the diagnosis of LVHs among hypertensives. This study sets out to determine the prevalence of LVH among hypertensives as determined by echocardiography.

Material and Methods: One hundred and seventy-eight hypertensives and eighty-nine age and sex-matched controls were recruited consecutively into the study. They all had echocardiography done to determine which among had LVH. The partition value for LVH for hypertensives was determined using the 97th percentile of the left ventricular mass for controls as a cutoff point.

Results: Echocardiographic determined the prevalence of LVH among hypertensives was 32.4%.

Conclusion: The echocardiographic prevalence of LVH was 32.4% in the study population. This is a significant proportion among the study population considering the clinical impact of LVH among patients with hypertension.

Keywords: Echocardiography, Left ventricular hypertrophy, Hypertension, Nigeria

INTRODUCTION

Hypertension is defined as persistent systolic blood pressure (BP) of >140 mmHg and/or diastolic BP of 90 mmHg or higher.[1] It remains one of the most important risk factors for cardiovascular disease and a major global public health problem.[2] Worldwide, approximately 62% of strokes and 49% of cases of ischemic heart disease are attributable to suboptimal BP control. Hypertension accounts for more than 7 million deaths annually.[2]

Left ventricular hypertrophy (LVH) is a recognized complication of systemic hypertension and the best-studied marker of hypertensive heart disease.[3] It refers to an abnormal increase in the mass of the left ventricular myocardium caused by a chronically increased workload on the heart which may be directly due to hypertension.[4] Increased workload on the heart causing LVH can also be caused by comorbid conditions such as obesity, chronic kidney disease, and diabetes mellitus.

LVH is, however, a modifiable risk factor as treatment causes its regression thereby decreasing the rate of adverse cardiovascular events and improving survival independent of the degree of BP lowering hence the need for its early diagnosis.[5] The prevalence of LVH among hypertensive varies widely between 20 and 50% depending on the population and the tool applied for diagnosis.[6]

LVH strongly predicts cardiovascular morbidity and mortality in hypertensive patients and is also an independent risk factor for overall cardiovascular mortality and morbidity.[7] It is an important factor used for risk stratification of hypertensives[8] and is known to cause a reduction in myocardial coronary reserve, predisposes to myocardial ischemia and left ventricular dysfunction thereby causing an increased incidence of coronary heart disease among hypertensives.[9] It has been determined that there is a 4–8 fold increase in the chance of strokes after adjusting for an increase in BP among hypertensives with LVH compared to those without it.[10]

LVH can be diagnosed by ECG or echocardiography.[11] The sensitivity of various ECG criteria remains low, ranging from 7 to 35% in mild hypertension and 10–50% in moderate/severe hypertension,[12] thus echocardiography remains the more sensitive and acceptable modality for diagnosing LVH, though it is, however, not yet widely available in many developing countries.[4]

This study determined the prevalence of echocardiographic LVH in the study population and thus hasten the detection of this important cardiovascular risk factor, thereby helping to attenuate the burden associated with it.

MATERIAL AND METHODS

This was a prospective, cross-sectional, and descriptive study. A total of 178 subjects and 89 age- and sex-matched controls were recruited for the study. Subjects that were considered for this study were consecutive eligible adult hypertensives seen at the cardiology clinic of the medical outpatient department of University of Abuja Teaching Hospital.

A subject was considered hypertensive if the BP measured on two or three occasions of at least 5 min apart was consistently greater than or equal to 140 mmHg systolic and/or greater than or equal to 90 mmHg diastolic or if patients are on the treatment for hypertension. Control populations were chosen among apparently healthy volunteers consisting of nurses, doctors, other hospital workers in UATH, and patient relations. Eligible subjects were recruited after written informed consent was obtained. Patients with chronic renal failure, anemia, valvular heart disease, pregnancy, diabetes, and athletes were excluded from the study.

Demographic data and anthropometric measurements were recorded. BP measurements were taken in the standard fashion. Blood samples of patients were taken to the determination of serum electrolytes, urea, creatinine, fasting blood sugar, and fasting lipid profile.

Echocardiography

Subjects had echocardiography done at the cardiology laboratory of UATH using a Vivid e General Electronics CE 0197, Rev 4, 2010 echocardiography machine. Complete echocardiographic examination was performed in the left lateral decubitus position by the corresponding author adhering to the recommendation of the American Society of Echocardiography (ASE),[13] using the leading edge to leading-edge technique with ECG gating. M–mode images were derived from the 2D images. Measurements were averaged over three cardiac cycles.

The left ventricular mass (LVM) was calculated according to the Devereux – modified ASE cube formula.[14] The partition value for LVH by echo was determined using the 97th percentile of the LVM values for controls as a cutoff point, and LVH was deemed to be present by echo for each subject if calculated LVM exceeds this cutoff point. LVM was indexed to body surface area and height. The mean and 97th percentile of the LVM and LVM/BSA for both the subjects and controls were calculated.

97th percentile of LVM and LVM/BSA for the male control population was calculated to be 231.29 g and 124.6 g/m^2, respectively. LVM and LVM/BSA 97th percentile for female control was 168.2 g and 106.0 g/m^2, respectively.

All male hypertensives whose LVM exceeds 231.2 g were considered to have LVH by echo and hypertensive females with LVM ≥168.2 g were also considered to have LVH.

Statistical analysis

Data management and analysis were performed using SPSS version 19.0. Continuous variables were presented as means (±SD) while categorical variables were presented as proportions. Differences in the prevalence of LVH between subjects and controls were compared using the Chi-square test. The student's *t*-test was used to compare the means between the two groups. $P < 0.05$ was considered statistically significant.

RESULTS

A total of 178 subjects with hypertension and 89 controls were studied. The demographic and clinical characteristics of the study population are summarized in [Table 1].

The subjects with hypertension and controls did not differ significantly in age (mean ± SD) = 51.3 ± 11.2 years versus 52.7 ± 7.1 years ($P = 0.28$), or male: female ratio which was 1:1 in both groups. Subjects with hypertension as expected

had statistically significant higher systolic and diastolic BPs. There were also significant differences between subjects and controls in body mass index (BMI) (28.0 ± 6.3 kg/m^2 vs. 24.7 ± 3.9 kg/m^2, P = 0.001), body surface area (1.8 ± 0.19 m^2 vs. 1.7 ± 0.16 m^2, P = 0.001), waist circumference (93.77 ± 10.81 cm vs. 91.2 ± 9.95 cm, P = 0.0 05), and hip circumference (100.92 ± 11.83 cm vs. 102.6 ± 10.96 cm, P = 0.005).

A total of 81 (45.5%) subjects with hypertension had been diagnosed for >5 years, 71 (39.9%) were within 1–5 years of diagnosis, 10 (5.6%) were diagnosed <1 year, and 16 (8.9%) were newly diagnosed hypertensives. The majority 163 (91.6%) of the subjects with hypertension were on the treatment.

Echocardiographic parameters of hypertensive subjects and control population

The echocardiographic parameters of subjects and controls are shown in [Table 2].

Mean LVM was significantly higher among subjects with hypertension (178.7 ± 64.7 g) than controls (154.9 ± 91.1 g) P = 0.01. Mean LVM for male and female subjects (185.2 ± 63.7 g and173.8 ± 65.2 g), respectively, was also significantly higher than those of male and female controls (173.2 ± 51.9 g and 141.1 ± 32.1 g), P = 0.012 and 0.005, respectively.

The LVM indexed to body surface area (LVM/BSA) differed significantly among female subjects with hypertension (98.1 ± 40.1 g/m^2) compared to the female controls (85.3 ± 18.8 g/m^2) (P = 0.005) but not among the males. LVM/BSA for all subjects with hypertension was also not statistically different when compared to all controls.

LVM indexed to height (LVM/HT) for all, female, and male subjects with hypertension which were 119.4 ± 36.4 g/m, 118.8 ± 39.5 g/m, and 109.6 ± 41.9 g/m, respectively, were all significantly higher than those of all female and male controls which were 104.2 ± 32.2 g/m, 95.6 ± 26.8 g/m, and 89.0 ± 19.1 g/m (P = 0.005, 0.001, and 0.001, respectively).

The mean left ventricular internal dimension in diastole (LVIDd) for hypertensives (4.61 ± 0.89 mm) was significantly higher compared to that of the controls (4.33 ± 0.79 mm) (P = 0.005). The mean LVIDd for male subjects with hypertension (4.63 ± 0.92 mm) was significantly higher than that of male controls (4.38 ± 0.78 mm) (P = 0.04). Mean

Table 1: Comparison of the clinical characteristics of subjects and controls.

Parameter	Subject *n*=178 Mean (SD)	Control *n*=89 Mean (SD)	*P*-value
Age (years)	51.3 (11.2)	52.7 (7.1)	0.280
Height (m)	1.64 (0.08)	1.61 (0.08)	0.004
Weight (kg)	75.9 (16.3)	64.8 (11.1)	0.001
BMI (kg/m^2)	28.0 (6.3)	24.7 (3.9)	0.001
BSA (m^2)	1.8 (0.19)	1.7 (0.16)	0.001
SBP (mmHg)	143 (19.5)	124.4 (4.8)	0.001
DBP (mmHg)	88.3 (10.2)	77.8 (7.6)	0.001
MAP (mmHg)	109 (12.6)	97 (8.3)	0.001
Pulse rate (b/min)	78.9 (18)	76.8 (17.4)	0.023
Waist circumference (cm)	93.77 (10.81)	91.2 (9.95)	0.005
Hip circumference (cm)	100.92 (11.83)	102.6 (10.97)	0.005
Waist/hip ratio	0.93 (0.10)	0.98 (0.12)	0.024
	n (%)	*n* (%)	
Male	76 (42.7%)	39 (43.8%)	
Females	102 (57.3%)	50 (56.2%)	
Duration of hypertension			
Newly diagnosed	16	8.9	
<1 year	10	5.6	
1–5 years	71	39.9	
>5 years	81	45.5	
Treated hypertensives	163	91.6	

BMI: Body mass index, BSA: Body surface area, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, SD: Standard deviation

Table 2: Echocardiographic parameters of subjects and controls.

Parameter	Subject *n*=178 Mean (SD)	Control *n*=89 Mean (SD)	*t*–test	*P*-value
LVM (g)				
Male	185.2 (63.7)	173.2 (51.9)	1.53	0.126
Female	173.8 (65.2)	141 (32.1)	4.46	0.0001
All	178.7 (64.7)	154.9 (91.1)	2.46	0.01
LVM/BSA (g/m^2)				
Male	99.4 (30.6)	99.5 (33.2)	0.336	0.715
Female	98.1 (40.1)	85.3 (18.8)	2.842	0.005
All	102.1 (36.2)	91.4 (26.7)	1.55	0.12
LVM/HT (g/m)				
Male	119.4 (36.4)	104.2 (32.2)	1.029	0.001
Female	109.6 (41.9)	89.0 (19.1)	1.203	0.001
All	118.8 (39.5)	95.6 (26.8)	2.84	0.005
LVIDd (cm)				
Male	4.63 (0.92)	4.38 (0.78)	2.005	0.04
Female	4.57 (0.83)	4.28 (0.74)	2.008	0.03
All	4.61 (0.89)	4.33 (0.79)	2.008	0.03
IVSTd (cm)				
Male	1.32 (0.21)	1.13 (0.15)	2.50	0.016
Female	1.27 (0.17)	0.99 (0.13)	0.93	0.354
All	1.28 (0.22)	1.12 (0.14)	1.69	0.02
LVPWd (cm)				
Male	1.22 (0.19)	1.11 (0.12)	2.58	0.010
Female	1.20 (0.13)	1.09 (0.11)	0.72	0.474
All	1.22 (0.21)	1.10 (0.13)	1.67	0.099

LVPWd: Left ventricular posterior wall in diastole, LVIDd: Left ventricular internal diameter in diastole, IVSDd: Interventricular septal diameter in diastole, SD: Standard error, LVM: Left ventricular mass, LVM/BSA: Left ventricular mass indexed to body surface area, LVM/HT: Left ventricular mass indexed to height

LVIDd was also significantly higher among female subjects with hypertension (4.57 ± 0.83 mm) compared with female controls (4.28 ± 0.74 mm) (P = 0.04). Mean interventricular septum dimension (IVSTd) for hypertensives (1.28 ± 0.22 mm) was significantly higher than that of the controls (1.12 ± 0.14 mm) (P = 0.02). Male hypertensives had a mean left ventricular posterior wall dimension in diastole (LVPWd) of 1.22 ± 0.19 mm which was significantly higher than a value of 1.11 ± 0.12 mm obtained for the male controls (P = 0.01). The LVPWd of female subjects with hypertension and controls was not significantly different.

DISCUSSION

The subjects and controls were similar in gender distribution with a male to female percentage of 42.7%:57.3% and 43.8%:56.2% for subjects and controls, respectively. This is important because gender has been shown to affect LVM values necessitating the use of different LVM partition values or cutoff points for males and females. Subjects and controls also had similar age distribution (51.3 ± 11.2 vs. 52.7 ± 7.1 years). The mean systolic and diastolic BPs of the hypertensive subjects as expected were significantly higher in the subjects than in controls (P = 0.001)

The mean BMI and body surface area (BSA) for the hypertensive subjects were also significantly higher in the subjects than in the controls. Obesity has been associated with increases in the left ventricular thickness, LVM, and prevalence of echocardiographic LVH independent of the impact of BP levels.[15]

Table 3: Prevalence of LVH by and echocardiography among the study population.

Echocardiographic LVH	Prevalence-*n* (%)	95% CI
Echocardiographic LVH	58 (32.4%)	25.9–39.6%

ECG: Electrocardiographic, LVH: Left ventricular hypertrophy, CI: Confidence interval

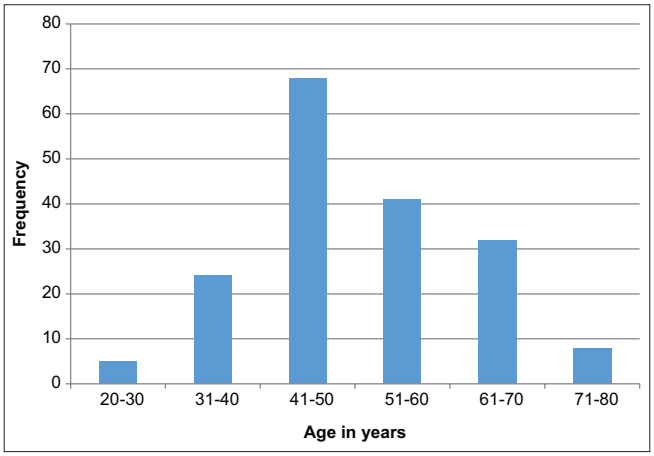

Figure 1: Age distribution of the study population.

The prevalence of LVH by echocardiography was 32.4%. This is similar to values obtained by Dada *et al*., in Ibadan[16] and Katibi and Adenle, in Ilorin[17] who reported echocardiographic LVH prevalence of 34% and 35%, respectively. A higher prevalence of echocardiographic LVH (61.8%) was, however, obtained by Karaye and colleagues in Kano in a previous study where the majority of participants were not on treatment.[18] A report involving Caucasian population demonstrated prevalence rates of echocardiographic LVH ranging from 20 to 50%.[19] Another study in a Caucasian population with a large percentage of newly diagnosed hypertensives obtained a prevalence of LVH by echocardiography of 50%.[20]

The present study involved a mixed group of hypertensives: Controlled, uncontrolled, newly diagnosed hypertensives, and hypertensives that have been in treatment for many years. The last group constituted a very large percentage of the study population. Some previous studies that stratified hypertensives into BP controlled and non-controlled groups obtained lower LVH prevalence for the controlled hypertensives.[21] This was due to the effect of long-term antihypertensive treatment on the regression of LVH.[22]

The duration of anti-hypertensive treatment also affects LVM in individuals with hypertension. The losartan intervention for endpoint reduction in hypertension (LIFE) study[13] showed that there was a significant reduction in the LVM with prolonged pharmacological treatment, especially with an angiotensin receptor blocker. About 45.5% of the hypertensives recruited in this study have been on the treatment for more than 5 years and this might have influenced the comparatively low prevalence of echocardiographic LVH.

The echocardiographic partition values of LVM for LVH among participating hypertensives in the present study were 231.29 g for males and 168.2 g for females while LVM/BSA partition values were124.6 g/m^2 for males and 106.0 g/m^2 for females. These were based on the 97th percentile echocardiographic LVM and LVM/BSA of the control subjects. Similar LVM/BSA partition values of 130 g/m^2 for males and 106 g/m^2 for females were utilized in a study on black hypertensives.[16] These values are higher than the standard cutoff values for determining LVH in the previous studies on Caucasian population. The Framingham heart study established partition values of 121 g/m^2 and 100 g/m^2 for males and females, respectively[14] whereas in the LIFE study, the values were 116 g/m^2 and 104 g/m^2 for males and females, respectively. The higher partition value of LVM and LVM/BSA used in the present study might, therefore, be an additional explanation for the observed relatively low echocardiographic LVH prevalence.

CONCLUSION

The echocardiographic prevalence of LVH was 32.4% in the study population. This is a significant proportion among the study population considering the clinical impact of left ventricular hypertrophy among patients with hypertension.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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