

Left Ventricular Mass Formulae and Prevalence Rates of Echocardiographic Left Ventricular Hypertrophy in Nigerians with Essential Hypertension

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Abstract

Background: Left ventricular hypertrophy (LVH) as a marker of cardiac damage in hypertension has important prognostic implications. With high prevalence of hypertension in Nigeria and the untoward effect of LVH, it is essential that the prevalence of LVH be determined. **Aims:** To determine prevalence of LVH and its severity in clinical practice among hypertensive patients referred for echocardiographic assessment in Nigeria. **Materials and Methods:** Devereux and Troy formulae were used to calculate echocardiographic LV mass (LVM) in 401 subjects and thereafter normalized to body surface area (BSA), height² (ht²) and height^{2.7} (ht^{2.7}) to define LVH to standard gender-specific thresholds. **Results:** Mean age was 53.22 ± 16.56 years (male = 53.18 ± 15.80; female = 53.27 ± 17.43; *P* = 0.958) with a male:female ratio of 1.13:1. Prevalence rates of LVH ranged between 38.9-51.3% using the Devereux Formula and 62.4-71.1% using the Troy formula. LVM/(ht^{2.7}) using the Troy formula gave the highest prevalence rate of LVH. Majority of the patients with LVH had severe form of hypertrophy with the prevalence rates ranging from 22.3% (LVM/BSA; Devereux formula) to 47.1% (LVM/ht^{2.7}; Troy formula). **Conclusion:** Prevalence of LVH by any echocardiographic criteria is high. There is a need to come to a consensus on the best formula and indexing variables, that will unify the reporting of LVH.

Keywords: Essential hypertension, Left ventricular hypertrophy, Prevalence, Severity of left ventricular hypertrophy

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Introduction

The close association between established hypertensive disease and hypertrophy of the left ventricle and the vasculature has been known for more than a century. In hypertensive patients, left ventricular hypertrophy (LVH) is the earliest sign of cardiac damage, and significantly increases the risk of major cardiovascular events.^[1] The observation that LVH is twice as frequent in black

compared to white hypertensive patients with similar arterial pressures,^[2] suggests an ethnic determinant.

The two commonly used noninvasive methods of diagnosing LVH in clinical practice are electrocardiogram (ECG) and echocardiogram. Even though, the power of some of the more commonly used electrocardiographic criteria to rule out LVH in patients with hypertension is poor,^[3] the presence of electrocardiographic-LVH in hypertension nonetheless carries important prognostic information.^[4] In view of the low sensitivity of ECG in detecting LVH, echocardiography has become the preferred mode of investigation in patients with hypertension. In addition to the detection of LVH, echocardiographic examination of hypertensive patients or other individuals with cardiac complaints^[5,6] also provide additional information on cardiac structure and functions, such as the degree of atrial enlargement,

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ventricular geometric pattern and diastolic dysfunction. In terms of prognostic value, it is established that echocardiographically determined LVH is one of the powerful independent risk factors for cardiovascular morbidity, cardiovascular complications, and mortality.^[7] Total and cardiovascular mortality risk increases with increasing echocardiographic left ventricular mass index (LVMI), independent of other cardiovascular risk factors.

Calculations of LVM with M-mode echocardiography which has an advantage of being noninvasive, has led to the development of echocardiographic criteria for LV hypertrophy.^[8,9] The prevalence rates of LVH as assessed by echocardiography markedly varies among studies, ranging from 3 to 77%, depending on clinical characteristics of the population studied and diagnostic criteria applied.^[10,11] Available data on LVH prevalence are mostly derived from population-based studies and selected hypertensive cohorts with rather scanty data available from surveys conducted in clinical practice. Considering the high prevalence of hypertension in Nigeria,^[12] and the untoward effect of LVH, it is essential that the prevalence of LVH using echocardiography be determined. Consequently, the primary aim of the present study was to determine the prevalence of LVH and its severity in hypertensive patients referred for echocardiography assessment of LVH in Southwest Nigeria with the use of different LVM formulas (Troy^[13] and Devereux^[14]) as well as different indexation threshold values in these native Africans.

Materials and Methods

A cross sectional survey of 401 hypertensive subjects was carried out in the echocardiography laboratory of University Teaching Hospital, Ado Ekiti, Southwest Nigeria. This study was conducted between March 2009 and March 2011. Ethical clearance for the study was approved by the ethics and research committee of the hospital in conformity with ethical guidelines of the 1975 Declaration of Helsinki and all the participants gave written consent to participate.

Demographic parameters of subjects were noted and recorded. All subjects were clinically examined to evaluate their body mass index (BMI) (weight (kg)/height² (m)), body surface area (BSA) was calculated using the formula of Dubois.^[15] The cardiovascular status of the subjects was also evaluated. Subjects were considered hypertensive if they had a resting SBP \geq 140 mmHg and/or DBP \geq 90 mmHg on two occasions measured after at least 3 min of rest with a mercury sphygmomanometer or if they were on antihypertensive therapy.^[16] Korotkoff phase 1 was used for systolic and phase 5 for diastolic blood pressure. Excluded were the patients with evidence

of valvular abnormality, congestive heart failure, ischemic heart disease, renal failure, hemoglobinopathy, and diabetes mellitus.

They also had transthoracic two-dimensional (2-D) and 2-D derived M-mode echocardiography performed according to standard procedure,^[13] while in the left lateral decubitus position using the (SonoScape 1000 Ultrasound Imaging System) with 4-2 MHz transducer. Left ventricular end diastolic measurements were taken during at least three cardiac cycles according to American Society of Echocardiography convention.^[17] This included the left ventricular internal diameter (LVIDD), posterior wall thickness (PWT), and interventricular septal thickness (IVST). LVM was estimated from the Devereux's formula^[14] $0.80 (1.04 (LVIDD + PWT + IVST)^3 - (LVIDD)^3)$ g and Troy's formula^[13] $1.05 ((LVIDD + PWT + IVST)^3 - (LVIDD)^3)$ g and normalized to BSA, height² (ht²) and height^{2.7} (ht^{2.7}).

LVH was defined by absolute and normalized LVM according to the following gender-specific thresholds: (A) LVM \geq 225/163 g; (B) LVMI \geq 116/96 g/m²; (C) LVMI \geq 77.7/69.8 g/m² (D) LVMI \geq 49/45 g/m^{2.7}^[18] in men/women, respectively. The values of LVM and LVMI were graded^[18] as mildly abnormal (A) 225-258 g in men, 163-186 g in women; (B) 116-131 g/m² in men, 96-108 g/m² in women; (C) 49-55 g/m^{2.7} in men, 45-51 g/m^{2.7} in women), moderately abnormal (A) 259-292 g in men, 187-210 g in women; (B) 132-148 g/m² in men, 109-121 g/m² in women; (C) 56-63 g/m^{2.7} in men, 52-58 g/m^{2.7} in women), and severely abnormal (A) \geq 293 g in men, \geq 211 g in women; (B) \geq 149 g/m² in men and \geq 122 g/m² in women; (C) \geq 64 g/m^{2.7} in men, \geq 59 g/m^{2.7} in women.

Statistical analysis

SPSS version 13.0 software (SPSS, Chicago, IL, USA) was used in the analysis of the data. Categorical variables were expressed as counts (percentages) while continuous variables were expressed as mean \pm standard deviation (SD). Chi-square analysis was done to compare proportions. A $P < 0.05$ was taken as statistically significant.

Results

A total of 401 patients with a male:female ratio of 1.13:1 were studied. Their mean age was 53.22 ± 16.56 years (male = 53.18 ± 15.80 ; female = 53.27 ± 17.43 ; $P = 0.958$). There were significant gender differences in the anthropometric indices of height and BSA as shown in [Table 1].

As shown in [Table 2], prevalence rates of LVH ranged between 38.9-51.3% using the Devereux formula and 62.4-71.1% using the Troy formula. On the whole,

Table 1: Clinical characteristics of the study population

	Total (n=401)	Male (n=213)	Female (n=188)	P value
Age (years)	53.22±16.56	53.18±15.80	53.27±17.43	NS
Weight (kg)	71.26±16.12	72.67±16.10	69.69±16.03	NS
Height (m)	1.66±0.09	1.68±0.10	1.64±0.07	<0.001
BMI (kg/m ²)	26.16±6.64	26.25±7.21	26.05±5.88	NS
BSA (m ²)	1.81±0.23	1.84±0.23	1.77±0.22	0.008
SBP (mmHg)	140.20±23.41	141.08±22.63	139.24±24.28	NS
DBP (mmHg)	87.37±14.85	87.02±14.94	87.75±14.79	NS
Treatment (%)	90.8	92.5	88.0	NS
% BMI ≥25 kg/m ²	52.1	54.5	45.4	NS

BMI: Body mass index; BSA: Body surface area; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; NS: Not significant

Table 2: Prevalence rates of LVH according to different LVM indexations using Devereux and Troy formulae in the study population (n=401)

	Devereux (%)	Troy (%)	P value
LVM (g)	43.7	67.1	<0.001
LVM/BSA (g/m ²)	43.8	66.5	<0.001
LVM/ht ^{2.7} (g/m ^{2.7})	51.3	71.1	<0.001
LVM/ht ² (g/m ²)	38.9	62.4	<0.001

LVM: Left ventricular mass; BSA: Body surface area; ht: Height

Table 3: Prevalence rates of LVH according to gender

	Male n=213 (%)	Female n=188 (%)	P value
†LVM (g)	73 (34.3)	104 (55.3)	<0.001
†LVM/BSA (g/m ²)	88 (41.3)	90 (47.9)	0.168
†LVM/ht ^{2.7} (g/m ^{2.7})	109 (51.2)	97 (51.6)	0.529
†LVM/ht ² (g/m ²)	82 (38.5)	75 (39.9)	0.426
‡LVM (g)	128 (60.1)	141 (75.0)	0.001
‡LVM/BSA (g/m ²)	137 (64.3)	130 (69.2)	0.222
‡LVM/ht ^{2.7} (g/m ^{2.7})	154 (72.3)	131 (69.7)	0.390
‡LVM/ht ² (g/m ²)	131 (61.5)	119 (63.3)	0.410

†Represents derivatives of Devereux formula; ‡Represents derivatives of Troy formula. LVM: Left ventricular mass; BSA: Body surface area; ht: Height

LVM/height indexed to the power of 2.7 using the Troy formula gave the highest prevalence rate of LVH.

When LVM and its different indexation factors using the Devereux and Troy formulae were stratified according to gender [Table 3], prevalence rates of LVH were significantly higher in females than males only when LVM was used without any form of indexation.

Table 4 shows the echocardiographic findings of the study population. Aortic root dimension, posterior wall thickness (PWD), and IVST were significantly higher in males compared with females. The mean LVM calculated with Troy formula was higher than the mean LVM calculated with Devereux formula. As shown in [Figure 1], irrespective of the formula and partitioning criteria used to define LVH, majority of the patients with LVH had severe form of hypertrophy with the

prevalence rates ranging from 22.3% (indexing the LVM to BSA; Devereux formula) to 47.1% (indexing LVM to 2.7 the power of height; Troy formula). The prevalence of the severe form of LVH for males ranged between 16.4% with LVM/BSA with Devereux formula and 40.4% with LVM/ht^{2.7} with Troy formula while it ranged between 23.4% with LVM/ht^{2.7} (Devereux formula) and 51.1% with unindexed LVM with Troy formula in females.

Discussion

We found the prevalence of LVH in hypertension to be high irrespective of the echocardiographic criteria used. This is similar to previous findings by Cuspidi *et al.*, who reviewed 30 studies with a total of 37,700 hypertensive patients who were assessed for LVH using echocardiography. LVH was defined by 23 criteria and its prevalence ranged from 36-41%.^[19] We also found that the prevalence of LVH varied depending on the type of indexed LVM. This is similar to the work by Salvetti *et al.*,^[20] who found that the prevalence of LVH was drastically different depending on the type of indexed LVM, being 19.9% when the LVM was indexed for BSA and 72.3% when indexed for height.^[20]

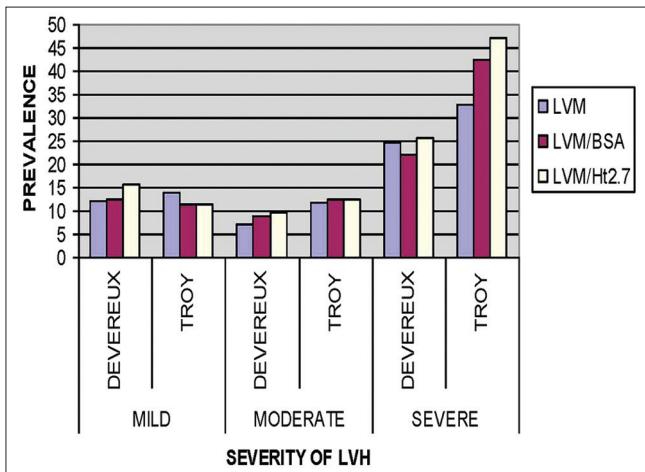
The Devereux formula is more conservative in identifying LVH compared with the Troy formula regardless of the indexation factor used in our study population. With allometric height-based adjustment, ht² gave a more restrictive prevalence rate of LVH than ht^{2.7} with both Devereux and Troy formulae. It has been suggested that ht^{2.7} adjustment model offers the most accurate estimation of LVH and risk factors for pathologic changes in the heart structure, particularly in obese subjects.^[21]

Levy *et al.*,^[22] reported a greater prevalence of LVH in women in the Framingham population, though some other studies failed to observe this gender trend. In this study, the prevalence of LVH is significantly higher in females than males by either of the Devereux and Troy formula only when there is no adjustment of LVM

Table 4: Echocardiographic findings of the study population

	Total (n=401)	Male (n=213)	Female (n=188)	P value
LVIDD (mm)	48.02±10.32	48.79±10.78	47.16±9.74	0.116
LVIDS (mm)	34.46±12.14	35.34±12.56	33.47±12.60	0.124
PWD (mm)	10.84±2.31	11.13±2.41	10.52±2.15	0.008
IVST (mm)	11.32±2.67	11.52±2.66	10.68±2.62	0.002
AOD (mm)	29.70±5.37	30.81±4.55	28.44±5.92	<0.001
LAD (mm)	37.87±7.90	37.51±7.96	37.24±7.85	0.732
EF (%)	60.90±18.68	59.47±18.53	62.52±18.77	0.105
FS (%)	29.62±11.90	28.75±11.65	30.60±12.13	0.122
E/A	1.30±0.84	1.32±0.90	1.26±0.77	0.490
RWT ₁	0.49±0.19	0.50±0.21	0.47±0.15	0.127
RWT ₂	0.49±0.19	0.50±0.21	0.47±0.16	0.233
†LVM (g)	199.87±80.81	213.32±84.20	184.81±75.47	<0.001
‡LVM (g)	252.22±101.98	269.21±105.00	233.24±95.24	<0.001
†LVM/BSA (g/m ²)	110.37±44.85	113.63±42.60	106.40±47.33	0.184
‡LVM/BSA (g/m ²)	139.29±56.60	143.41±53.76	134.28±59.73	0.184
†LVM/Ht ^{2.7} (g/m ^{2.7})	51.23±22.59	52.46±23.39	49.73±21.56	0.314
‡LVM/Ht ^{2.7} (g/m ^{2.7})	64.64±28.49	66.20±24.50	62.74±27.20	0.312
†LVM/Ht ² (g/m ²)	72.56±29.61	74.71±29.61	69.94±29.70	0.182
‡LVM/Ht ² (g/m ²)	91.58±37.47	94.28±37.36	88.27±37.48	0.182

†Represents derivatives of Devereux formula; ‡Represents derivatives of Troy formula; LVIDD: Left ventricular internal diameter in diastole; LVIDS: LVID in systole; PWD: P-wave dispersion; IVST: Interventricular septal thickness; AOD: Aortic root dimension; LAD: Left anterior descending; EF: Ejection fraction; FS: Fractional shortening; E/A: E wave/A wave; RWT: Relative wall thickness; LVM: Left ventricular mass; BSA: Body surface area; ht: Height

**Figure 1:** Severity of LVH in the study population

with body size such as BSA and height. In other words, adjustment for body size appear to obviate the gender differences that may occur with either the Devereux or Troy formulae in the determination of the prevalence rate of LVH.

In the present study, majority of the patients with LVH had severely abnormal form of LVH ranging from 22.3% (LVM/BSA; Devereux formula) to 47.1% (LVM/ht^{2.7}; Troy Formula). This is similar to the findings of Cuspidi *et al.*,^[23] where a prevalence of 20.8–28.9% was found. The severely abnormal form of LVH was more frequent in women than in men in our study. Women have been shown to have an

increased parietal hypertrophic responses to pressure overload,^[24,25] even after body size correction. This adaptive pattern has been demonstrated also in animal models.^[26] The unfavorable prognostic implications of this hypertrophic response are suggested by the findings of Liao and coworkers^[27] of a five-fold greater risk of death associated with LV hypertrophy indexed by BSA in woman compared to the risk associated to LVH in men. In type 2 diabetes mellitus, Tenenbaum and colleagues^[28] had also demonstrated that hypertensive women have significantly higher prevalence of LVH and left atrial enlargement compared to men.

In conclusion, the prevalence of LVH by any echocardiographic criteria is high and this points to the need for a more aggressive treatment of hypertension and related risk factors for LVH. There is also the need to come to a consensus on the best formula and indexing variables that will unify the reporting of LVH considering the prognostic significance of LVH.

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