



Original Article

Determinants of Left Ventricular Mass Among Apparently Normal Children in Enugu State

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Abstract

Background: The left ventricular mass (LVM) index is a very crucial index used for risk stratification among children. This work aimed to document the LVM values among children and delineate the prevalence of LVH among healthy children in Southeast Nigeria. It elicited the correlation between Left ventricular mass index (LVMI) and age, gender and height.

Methodology: This is a descriptive study involving 218 children drawn from one public and 2 private hospitals over six years. Echocardiographic measurements which assessed various parameters and indices of LVH were ascertained. The data was analyzed with the IBM SPSS statistics for windows, version 20 (IBM Corp, Chicago).

Results: The prevalence of left ventricular hypertrophy among the respondents was 5.0%. There was a strong positive correlation between left ventricular mass and surface area, ($n=218$, $r=0.751$, $p<0.001$). There was a very strong positive correlation between left ventricular mass and weight, which was found to be statistically significant, ($n=218$, $r=0.755$, $p<0.001$). There was a very strong positive correlation between left ventricular mass and BMI, which was found to be statistically significant, ($n=218$, $r=0.34$, $p=0.004$). There was a positive correlation between left ventricular mass and height, which was found to be statistically significant, ($n=218$, $r=0.126$, $p=0.238$).

Conclusion: The mean values of LVM indexed to height, BMI, surface area, and weight and compared with gender were elicited in this study. These mean normative values could be a guide for the cardiothoracic surgeon and paediatric cardiologist in some clinical decision-making.

Keywords: Left Ventricular Hypertrophy; Healthy Children; Prevalence; Echocardiography.

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Introduction

Changes in left ventricular mass are used to ascertain disease prognosis in children with heart disease. [1] Among children with heart diseases, the risk of abnormally high LVM is usually associated with high morbidity and mortality. [2,3] Raised LVM is also very important and can be used as a sole marker for clinical outcomes. [4] For instance, Mejis[4] et al showed an increase in cardiovascular morbidity associated with increased LVM, even when there is no hypertrophy. It has also been reported that for each increase of 50 g/m of LVM indexed to height, the mortality rates increase by 150%. This even rises to more than 400 percent in children with hypertension. De Simone and co-workers also noted that LVH regression in a child with a prior LVH or cardiac disease reveals a better and a favourable prognostic marker. [5]

On the other hand, Amin et al [6] noted LVH among children with obstructive sleep apnoea where a prevalence rate of 39% was documented. Furthermore, Metwalley et al, [7] in Egypt, noted significant changes in left ventricular structure and function among children with newly diagnosed graves' disease.

Similarly, Ngabea et al [8] noted an increased prevalence of LVH among Obese children. They noted that obesity has been linked with increases in the left ventricular thickness and left ventricular mass (LVM), with documented prevalence of LVH of 32.4%.

Left ventricular hypertrophy (LVH) is an important tool in assessing children with cardiovascular disease. [9] Although there is a paucity of data on LVH in healthy children, there are several studies on the prevalence of LVH in children with chronic disease. For instance, Duru et al [10] noted left ventricular hypertrophy in 75.9% of children with HIV/AIDS. Similarly, Ige et al [11] noted increased LVM and diastolic dysfunction in children with HIV compared with controls. The finding by Ige et al [11] was also buttressed by Chinawa et al [12] who documented the prevalence of LVH among Nigerian children with sickle cell anaemia with normal BMI, as 37.5%.

Although LVH has shown prognostic importance in adults, its role in the management and prognosis of children with cardiovascular diseases is even more significant. [13] The diagnosis of LVH may show some innovations in clinical management for children with congenital heart disease and other cardiac anomalies. [14] There have been several controversies in the criteria used for the diagnosis and definition of LVH for children. [13] The exact LVM cut-off may be challenging because LVM depends strongly on body size. [13] In the adult population, several criteria were used to index LVM to body size (left ventricular mass index (LVMI)). [13] Notwithstanding, LVM indexed to body surface area remains the standard method in children. [15,16] Some studies have used a scale where the body size variable is raised to a certain power. [15] Indexing LVM to height, though very useful in adults, cannot be said in children where LV mass is indexed to body surface area. [1]

This study introduced the estimation of LVM to body surface area, and this will eliminate the error generated when LVM alone is used to assess cardiovascular morbidity by echocardiography. This work is therefore aimed at eliciting the prevalence of LVH among apparently healthy children in South-East Nigeria. It also elicits the correlation between LVMI with age and gender.

Methodology

Study area and Study population: This study was conducted in two private hospitals namely, Triple Care hospital and Blessed children hospital Enugu, in Enugu metropolis involving a total of 218 apparently healthy children. These private hospitals provide Paediatric cardiology specialized services in the management of children with heart diseases and review children who came for routine follow-up and who are apparently well after treatment for common paediatric illnesses such as malaria and acute respiratory tract disease. A thorough cardiovascular examination was done for six hundred and seventy-one children seen over the study period. Those that had a clinical murmur, signs and symptoms of cardiac disease were

further screened using echocardiography to exclude any heart disease. Out of the 671 children that were screened for cardiac disease, two hundred and eighteen children who had no echocardiographic diagnosis of shunt defect, valvar regurgitation or other lesions such as pericardial effusion, cardiomyopathy or abnormal aortic arch, were then selected from the 671 children and recruited into the study.

Participant Selection: Apparently healthy children aged 1 month to 19 years who had normal clinical history, normal physical examination without any known systemic illness and normal echocardiographic findings were included in the study. Children with any form of congenital heart disease, any acquired heart disease, or any other chronic or systemic illness, those with a clinical feature of a syndromic disease or those with no congenital heart disease but had pulmonary or systemic hypertension were excluded from the study. The two hundred and eighteen apparently healthy children who had normal echocardiography after echocardiographic screening for suspected/incidental murmurs and who fulfilled the inclusion criteria were then selected from the 671 children and recruited consecutively into the study. Information on this study and its contents were explained to the participants.

Study design and sampling: This study was a descriptive cross-sectional study conducted among healthy children who attended two private health institutions in Enugu metropolis.

Study Instrument

Given the clinical relevance of LVM, it is crucial to use a reliable and reproducible method for its estimation. Echocardiography was used in this study for estimation of LVM. The M-mode images were derived from the 2-dimensional images. The end-diastolic linear measurements of the LV inferior-lateral wall thickness, and LV internal diameter both in systole and diastole, interventricular septum (IVSd), both in systole and diastole were elicited. The LV images were acquired at end-diastole with the ultrasound beam directed perpendicular to the LV long axis at the level of the leaflet tips of the mitral valve using a parasternal short-axis view. The cursor was placed centrally bifurcating through the RV and the LV via the mitral valve. The readings of the LV function including LV mass were displayed.

A low dose mild sedative, rectal Diazepam (0.3mg/kg/dose) was given to those who were uncooperative. This was explained to the parents and caregivers of the participants. They were also told about the purpose of the mild sedative, the dosage and possible complications if any. All the echocardiographic images were viewed in the left lateral decubitus position. All the studies were done by the same investigator, so inter-observer variability testing was not necessary. The M-mode that had poor tracing was repeated twice and the best score for each patient was documented. The investigator had a one-year training in paediatric cardiology with a bias in paediatric echocardiography in Mumbai, India. Left ventricular hypertrophy was defined as LVMI i.e. (LVM/surface area) above the 95th percentile. [18,19] Anthropometric measurements were also documented. Weight was measured in kilograms using a bathroom scale, for age above 2 years and bassinet for those below 2 years, while height or supine length (for those less than 2 years) and stadiometer for those above 2 years was measured in centimeters. The research was done within 6 years from 2018 to 2023.

Data analysis

Data entry and analysis were done using IBM SPSS statistical software version 25. Categorical variables were summarized using frequencies and proportions while continuous variables were summarized using mean and standard deviation. The median and interquartile range were reported when the data was skewed. Chi square test of statistical significance was used to compare the difference in ventricular hypertrophy with gender and age. Correlation analysis was used to determine the strength of the linear relationship between left ventricular mass and anthropometry. Mann Whitney U test was used to compare the left ventricular mass by gender, while Kruskal Wallis test was used to compare the left ventricular mass index

by age group. The level of statistical significance was determined by a p value of 0.05. The left ventricular mass index was determined by dividing the left ventricular mass by the surface area.

Ethical approval and Consent to Participate

This was sought from the research and ethics committee of the University of Nigeria, Ituku-Ozalla Campus while verbal informed consent was obtained from the mothers who brought their children to the health centers. Informed consent was sought from a parent and/or legal guardian for study participation. Assent was taken from children over 7 years and consent was taken from those patients who had attained the age of majority.

Results

Table 1: Demographic characteristics of the respondents and prevalence of left ventricular hypertrophy

Variable	Frequency (n=218)	Percent (%)
Age of respondents in years		
Minimum	1.2 months	
Maximum	19 Yrs	
Mean±(SD)	5.6±5.7 Yrs	
Median (IQR)	4.0 (7.0) Yrs	
Age of respondents in groups		
<5 years	115	52.8
5-9 years	42	19.3
10-19 years	61	28.0
Gender		
Male	119	54.6
Female	99	45.4
Prevalence of left ventricular hypertrophy (All respondents)		
Yes	8	3.7
No	210	96.3
Left ventricular hypertrophy (LV mass indexed to weight) (All respondents)		
Yes	8	3.7
No	210	96.3

Table 1 shows the socio-demographic characteristics of the respondents and prevalence of left ventricular hypertrophy. The median age of the respondents was 4.0 years, (IQR=7.0 years). The highest proportion of the respondents, 115 (52.8%) were less than five years.

There were more of male respondents/participants than females with M: F ratio of 1.2:1. The prevalence of left ventricular hypertrophy among the respondents was 5.0%. The prevalence of left ventricular hypertrophy (LV mass indexed to weight) among the respondents was 3.7%.

Table 2: Differences in ventricular hypertrophy with gender and age

Variable	Left ventricular hypertrophy (n=218)		χ^2	p value
	Yes N (%)	No N (%)		
Gender				
Male	2 (1.7)	117 (98.3)	2.933	0.087
Female	6 (6.1)	93 (93.9)		
Age of respondents in groups				
<5 years	2 (1.7)	113 (98.3)	2.793	0.247
5-9 years	2 (4.8)	40 (95.2)		
10-19 years	4 (6.6)	57 (93.4)		

Table 2 shows the factors associated with left ventricular hypertrophy. A higher proportion of female respondents, 6.1% had left ventricular hypertrophy when compared to the males, 1.7% but the difference in proportions was not found to be statistically significant, ($\chi^2=2.933$, p=0.087).

Table 3: Correlation of Left ventricular mass and Anthropometry

Variable	Sample size (n=218)	Pearson correlation (r)	p value
Correlation of left ventricular mass and			
Surface area	n=218	0.751	<0.001
Weight		0.755	<0.001
Height		0.126	0.238
BMI		0.34	0.004
LVIDd		0.738	<0.001
LVPWd		0.743	<0.001
IVSs		0.787	<0.001
LVIDs		0.673	<0.001
LVPWs		0.440	<0.001
EF		-0.072	0.291
FS		0.044	0.516

IVSd, interventricular septum diameter in diastole; **LVIDd**, left ventricular internal diameter in diastole; **LVPWd**, left ventricular posterior wall diameter in diastole; **IVSs**, interventricular septum diameter in systole; **LVIDs**, left ventricular internal diameter in systole; **EF**, ejection fraction; **FS**, fractionating shortening; **LVM**, left ventricular mass; **VSD**, ventricular septal defect.

Table 3 shows the correlation of left ventricular mass with other variables. There was a strong positive correlation between left ventricular mass and surface area, increases in left ventricular mass correlated with increases in surface area and this was found to be statistically significant, (n=218, r=0.751, p<0.001). There was a very strong positive correlation between left ventricular mass and weight, increases in left ventricular mass correlated with increases in weight and this was found to be statistically significant, (n=218, r=0.755, p<0.001). There was a very strong positive correlation between left ventricular mass and BMI, increases in left ventricular mass correlated with increases in BMI and this was found to be statistically significant, (n=218, r=0.34, p=0.004). There was a positive correlation between left ventricular mass and height, increases in left ventricular mass correlated with increases in height and this was found to be statistically significant, (n=218, r=0.126, p=0.238).

Table 4 shows the comparison of left ventricular mass index with gender. The mean left ventricular mass index for the males, 138.4 (95%CI=125.3-151.6) was comparable to that of females, 141.0 (95%CI=120.2-161.9) (p=0.830). The mean left ventricular mass for the males, 128.8 (95%CI= 107.3- 150.4) was higher than that of females, 103.8 (95%CI= 84.8-122.7) but the mean difference was not found to be statistically significant, (p=0.091). The mean left ventricular mass index was highest among respondents who were 10-19 years, 163.5.1±78.8 and least among those who were less than 5 years old, 114.6.6±71.9 and the mean difference was found to be statistically significant, (Kruskal Wallis=10.780, p<0.001).

Table 4: Comparison of left ventricular mass index by gender and age group

Variable	Male (n=119)	Female (n=99)	Difference*	p-value
Left ventricular mass index (g/m²)				
Mean	138.4	141.0	2.6	0.831
95%CI	125.3- 151.6	120.2- 161.8		
Left ventricular mass (grams)				
Mean	128.8	103.8	25.1	0.091
95%CI	107.3- 150.4	84.8- 122.7		
Weight (kg)				
Mean	24.4	20.1	4.4	0.125
95% CI	20.6- 28.3	16.0- 24.2		
Surface area (m²)				
Mean	0.8	0.7	0.1	0.108
95%CI	0.7-0.9	0.6-0.8		
Variable	Sample size (n)	Left ventricular mass index (Mean±SD)	Kruskal p-value Wallis	
Age of respondents in years				
<5 years	(n=115)	114.6±71.9	10.780 <0.001	
5-9 years	(n=42)	172.9±115.5		
10-19 years	(n=61)	163.5±78.8		

*Mann Whitney U; F=Kruskal Wallis test

Table 5: Predictors of Left ventricular mass

Variable	Unstandardized coefficients		t	p value	95% CI for B	
	B	Std error			Lower	Upper
Constant	137.509	13.503	10.184	<0.001	110.891	164.127
Age in years	12.318	2.200	5.599	<0.001	7.981	16.655
Surface area	-80.198	42.632	-1.881	0.061	-164.239	3.842
Weight	-0.333	1.088	-0.306	0.760	-2.479	1.812
Gender (Male)	5.420	11.361	0.477	0.634	-16.975	27.816

Adjusted R square=0.141, F=9.799, p<0.001

Table 5 shows the predictors of left ventricular mass with an adjusted R square of 0.141, the model predicts that 14.1% of the variability in left ventricular mass is explained by the age of the respondents in years, surface area, weight, and gender. For one-unit change in age, ventricular mass increases by 12.318 and this was found to be statistically significant, (B= 12.318, 95%CI: 7.981- 16.655).

Discussion

Left ventricular hypertrophy is a very crucial index used in the diagnosis of children with cardiovascular disease. This study aimed at eliciting the prevalence of LVH among children in Southeast Nigeria. This study showed the prevalence of left ventricular hypertrophy among apparently healthy children as 5.0%. Movahed [17] et al noted a lower prevalence of 0.2 % in their study which was among healthy children. In addition, Chinali [18] and colleagues recorded a prevalence rate of 3.5 % while Woodiwiss [19] and Falkner [20] et al documented prevalence rates of 12% and 9.1% respectively in their reportage. These differences in prevalence rates may be explained by race and geographical location as well as sample size, as the sample size used by most of the cited studies above was smaller than our sample size. This study showed an increase in the prevalence of LVH with age. Studies have shown a varying correlation between left ventricular hypertrophy and age. [21-27] Cuspidi [22] et al noted that increasing LVH with age were both associated with a greater prevalence of concentric left ventricular morphology with attendant extra-cardiac organ damage.

There was no statistical difference in the mean LV mass index of males and their female counterpart. The finding above was corroborated with Va Gerdts [28] et al who noted that LVH did not differ between gender. They however noted that females with obesity and diabetes have higher odds of having LVH than their male folks. However, studies have shown that in certain cardiac diseases, LVH becomes a stronger risk factor for heart failure in females than in males. [27-31] Left ventricular mass was noted to be higher in males compared to females in this study.

This study showed a steady increase in LVM with weight. Furthermore, it is interesting to note in this study that when LVM was indexed to weight, the prevalence of LVH was similar to when LV mass was indexed to body surface area. Foster [26] et al among 231 children at risk for LVH noted that LV mass was higher using the LV mass index-for-age percentile method than the LV mass-for-weight percentile method. They noted that LVH was more likely to be diagnosed among overweight children than in children with normal weight. It is documented that the new LV mass reference centiles have shown LV mass relative to weight as the strongest determinant of LV mass and LVH. These findings of LVM versus weight driven LVH can help in screening children with cardiovascular risk. This implies that nutritional rehabilitation among obese children can prevent LV hypertrophy through weight control in overweight children. For instance, in a

study by Devereux [32] et al among children aged 7-18 years, a strong correlation was noted between LVM, LVH and weight. [32-34] Besides, it is shown in other studies that overweight or obesity is a strong independent predictor of LVH in children. [35,36] There is a link between being overweight and LVH. Abel [37] et al noted that increased tissue adipocytes in overweight children could lead to hyper-metabolic states and increased cardiac output. [37-41]

It is evident in this study that a strong positive correlation exists between left ventricular mass and surface area and weight. It is pertinent to note that of all the variables, LVM has the strongest correlation to body surface area. Thus, body surface area, when used in conjunction with other clinically accepted evaluations, is a useful tool for estimating normal cardiac structure and function.

Indexation of LVM to body surface area remains an independent predictor of sudden cardiac death and could help predict sudden cardiac death beyond the usual cardiovascular risk factors. [42,43] Reinier [44] et al in a case-control study noted that LVM indexed to body surface area (BSA) may have a positive effect in the reduction of sudden cardiac death despite normal left ventricular function of the individual. Granted that LV mass is commonly indexed to body surface area (BSA). However, its strong association with weight as seen in this study will support its use in obese children. The use of LVM indexed to weight will help ascertain the actual prevalence of LVH among them when compared with those with lean body weight. [45, 46]

Though this study showed a weak association between LVM and height, some studies have noted that even indexing LV mass/height to the power of 2.7 does not adequately normalize LV mass for height in children which makes it inaccurate to diagnose LVH. [47]

In general, there is no concrete agreement in the literature about the anthropometric parameter that showed the best correlation with left ventricular mass. [48] Currently, no significant differences were observed when echocardiographic measurements were correlated with BSA, weight, and height. [49]

Limitations of the study

A community-based design with a large cohort of participants may yield a better outcome in establishing a population-based prevalence study and baseline information in healthy children.

Conclusion

The mean values of LVM indexed to height, BMI, surface area, and weight and compared with gender were elicited in this study. These mean normative values could be a guide for the cardiothoracic surgeon and paediatric cardiologist in some clinical decision making. This study also showed that LVH though rare is not uncommon in apparently healthy children.

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