Left Ventricular Mass – Forerunner of Future Cardiovascular Morbidity in Young Healthy Population?

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Citation

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Abstract

Objective: The aim of the study was to compare left ventricular mass (LVM) by M-Mode echocardiography between normotensive young adult offspring of hypertensive and normotensive parents.Methods: One hundred randomly selected, apparently healthy, normotensive, male medical students in the age group of 19 to 25 years were studied by M-mode echocardiography. Fifty students with parental history of hypertension belonged to study group, and rest of the fifty without such history constituted the control group. They were examined for anthropometric characteristics, blood pressure, Body mass index (BMI), then M-mode echocardiograms were obtained.Data was statistically analysed by, t-test, simple linear regression technique, multiple regression analysis and $p < 0.05$ was considered statistically significant.Observation: Weight and BMI were significantly higher in the study group. In the echo characteristics, significant increase in Ventricular septal thickness(VST) by 15%, Posterior wall thickness (PWT) by 8% was observed in the study group. In the calculated echo characteristics of the two groups unadjusted LVM was significantly higher,(22%), $p<.0001$, in the study group. LVM adjusted for height was 20% higher and LVM adjusted for BMI was 14% higher in the study group,($p=0.001$). LVM indexed for height was also found to be significantly higher in the study group, (18%), $p=0.0002$.Conclusion: We concluded that mean LVM of normotensive offspring of hypertensive parents(144±37.95 gm) was significantly higher than the mean LVM of normotensive offspring of normotensive parents(118±17.41 gm). VST and PWT were greater in offspring of hypertensive parents. Anthropometic characteristics, BMI and BSA(Body surface area) had significant positive effect on LVM.

INTRODUCTION

Cardiovascular diseases will be the number one cause of deaths by 2020, accounting for 36.3% of all deaths in the world. In India, cardiovascular diseases account for 24% of total deaths of which 50% are due to coronary artery disease. [1] Left ventricular hypertrophy(LVH), a consistent feature of hypertension is now recognised to be an independent risk factor for subsequent cardiovascular mortality and morbidity. The Framingham Heart Study has implicated LVH as the most important predictor of future cardiovascular disease showing the presence of high risk of cardiovascular morbidity and mortality in people with electrocardiographic and echocardiographic evidence of LVH. [2]

The American College of Cardiology's Bethesda Conference has given LVH a category 1 risk factor for cardiovascular diseases. [1] In today's medicine the advent of echo, has revolutionized the sensitivity and specificity of detecting LVM. [3] The regression of LVH has now shown to reduce rates of subsequent cardiovascular events by 50% to 75%. [4,5] There is increased incidence of LVH in hypertension. The Framingham Heart Study [6] and the Tecumesh Offspring Study [7] have concluded that heredity can explain a definite proportion of variance of LVM. Therefore there are increased chances of getting increased LVM in offspring of hypertensive parents. A limited echo(only 2-dimentional views and a brief M-mode scan) can be a cost effective procedure to evaluate offsprings of hypertensive parents for LVH because of high pretest probability. Detection of LVM in these offspring could identify not only future hypertensives but also a population at high risk of future cardiovascular morbidity and mortality. This study was therefore carried out to detect the prevalence of a major cardiovascular risk factor(LVH) in a hypertension prone healthy normotensive population before actual manifestations of clinical hypertension, and identify candidates who might require early intervention to reduce their future cardiovascular events.

AIMS AND OBJECTIVES

To determine LVM of normotensive young adult offspring of normotensive and hypertensive parents.

To identify a subset of young healthy population who might require early intervention to reduce their risk of future cardiovascular events.

MATERIAL AND METHOD

This study was carried out in Acharya Vinoba Bhave Rural Hospital of Jawaharlal Nehru Medical College, Sawangi (Meghe), Wardha (M.S.) over a period of 2 years.

SUBJECTS

One hundred randomly selected, apparently healthy, normotensive, male medical students in the age group of 19 to 25 years were studied by M-mode echocardiography.

Fifty students belonging to the study group had history of parental hypertension (single or both parent) whereas the other fifty did not have any history of parental hypertension (control group). The study and control groups were matched for sex and age to minimise the effect of these variables in the calculation of LVM.

Subjects were excluded if they met any of the following criteria

- Blood Pressure > 140/90 mmHg.
- Current use of cardioactive drugs.
- Any cardiopulmonary disease.
- Systolic murmur of grade 3 or more.
- Any diastolic murmur.
- Any renal lump or renal bruit
- Echo showing evidence of any structural heart disease.
- Poor echocardiographic window.

METHODS

The subjects were examined for

- Weight in kilograms (kg)
- Height in meters (m)
- Blood pressure in mm of Hg

Blood pressure was measured in the right arm in sitting position by a mercury sphygmomanometer usuing a standard size cuff with the arm supported and positioned at the level of the heart, after a resting period of 5 minutes. The first and fifth Korotkoff sounds were taken as systolic blood pressure (SBP) and diastolic blood pressure (DBP) respectively.

Body mass index (BMI) was calculated by the Quetelet formula, BMI = Weight (kg) / Height (m^2). Body surface area (BSA) was derived from a standard nomogram. [8]

ECHOCARDIOGRAPHIC METHOD

M-mode Echocardiograms were obtained on a Toshiba CorevisionPro USG machine, Model SSA-350A using a cardiac phased array transducer PSF-25LT at 3 MHz.

Echo of the subjects was done in the partial left lateral decubitus position after a resting period of 3 - 4 minutes.

A 2-D guided M-mode recording of the left ventricle was obtained in the parasternal long axis view with the ultrasound beam passing through the left ventricle just beyond the tips of the mitral valve leaflets [9] and measurements of wall thickness and chamber diameter were made in diastole in accordance with methods outlined by ASE. [10] The mean value of LVID, VST and PWT were taken from three consecutive beats. LVM was estimated by the modified cubed formula of Devereux & Reichek using measurements obtained in accordance with the 'Penn' convention.

$$LVM\ (gm) = 1.04[(LVID \pm VST \pm PWT)^3 - (LVID)^3] - 13.6$$

All the data was entered into the proforma.

LVM was adjusted for height, BMI, BSA and height [2 7].

STATISTICAL ANALYSIS

Statistical analysis was carried out with the help of computer software. Mean and 1 Standard deviation (S.D.) of all variables in both groups was calculated. The student 't' test for unpaired observations was used to compare data in the two groups. Simple Linear regression technique was used to test correlation of LVM (dependent variable) with age, height, weight, BMI, BSA, SBP and DBP (independent variables) in both groups. The significance of values was tested using standard tables. Multiple regression analysis was carried out to test the combined effect of the independent variables on LVM in both groups, its significance was tested with the F test. $p<0.05$ (2-tailed) was considered statistically significant.

Figure 1

Table No. 1 : Comparison of Calculated Echocardiographic Characteristics in Control and Study Group.

Calculated Echocardiographic Characteristics	Control group Mean ± S-D. (n = 50)	Sturdy Group Mean ± S-D. (n = 50)	P
Unadjusted LVM (gm)	118.21 ± 17.41	144.55 ± 37.95	<0.0001
LVM / Height (gm/m)	69.94 ± 9.29	83.98 ± 21.04	<0.0001
LVM / BMI (gm m^2/kg)	5.7 ± 0.84	6.51 ± 1.36	0.001
LVM/BSA or LVMI (gm/m^2)	65.84 ± 7.75	77.17±17.49	0.0001
LVM/Height$^{2.7}$ (gm/$m^{2.7}$)	28.36 ± 4.21	33.41 ± 8.19	0.0002
VST/PWT	0.97 ± 0.15	1.02 ± 0.17	0.127

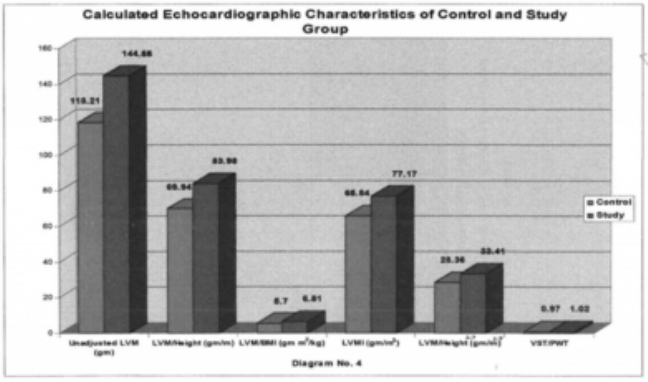


Figure 2

Table No. 2 Distribution of unadjusted and adjusted LV mass in the present study.

Method of indexation	Mean ± SJX .
Unadjusted LVM (gm)	130.9 ± 32.13
LVM / Height (gm/m)	76.72 ±17.61
LVM /BMI (gm m^2/kg)	6.1 ± 1.19
LVM /BSA or LVMI (gm/m^2)	71.34±14.61
LVM /height $^{2.7}$ (gm/m $^{2.7}$)	30.88 ± 6.98

Figure 3

Table No. 3 : Correlation and Predictive value of Observed Clinical Parameters for unadjusted LVM in Study Group

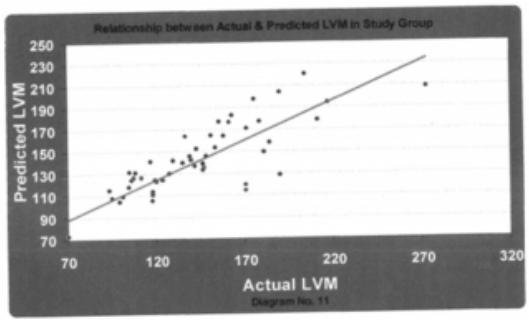
Observed Clinical parameters	Pearson correlation coefficient	R^2	p
Age	0.07	0.005	0.619
Weight	0.7	0.489	<0.0001
Height	0.37	0.139	0.008
BMI	0.64	0.413	0.0001
BSA	0.58	0.334	<0.0001
SBP	0.33	0.108	0.02
DBP	0.4	0.16	0.004
MAP	0.42	0.178	0.002

Multiple R2 - 0.6450

Adjusted R2 - 0.0.5757

F Statistic - 9.3097 (p < 0.05)

Diagram No. 3 shows the relationship between actual LVM and predicted LVM (by multiple regression) in the study population.



DISCUSSION

The present study compared the echocardiographic LVM characteristics of 50 normotensive young adult offsprings of hypertensive parents with 50 normotensive offsprings of normotensive parents.

In the observed clinical characteristics weight was significantly higher(6.7%) in the study group,(p=0.029). Systolic blood pressure (SBP) was also significantly higher in the study group,(4%, p=0.0009).Some other studies also had the same findings. [1112] In the calculated clinical characteristics between the two groups BMI was 5% higher in the study group than the control group,(p=0.037) like one study. [13]

As far as the echo characteristics are concerned we found that VST was significantly increased (15%) in the study group (p<0.001) similar to some studies. [131415] PWT in our study was also significantly increased(8%) in the study group as compared to the controls(p=0.003) similar to some studies. [1415] LVID did not differ significantly between the two groups.

In the calculated echo characteristics (Table 1, graph 1), of the two groups in our study unadjusted LVM was significantly (22%) higher in the study group than the control group,($p<0.001$) like the observations of other studies. [14] This may be due to the significant increase in the SBP in the study subjects. LVMI (Left ventricular mass index) was also significantly higher in the study population in our study similar to other studies. [15][16][17] .

LVM adjusted for height was 20% higher and LVM adjusted for BMI was 14% higher ($p=0.001$) in the study group. This method of indexation of LVM for BMI has not been used in many of the studies like that of ours. Similarly LVM indexed for height [27] was found to be significantly higher (18%) in the study group, ($p=0.002$). In our study, within the study group, the subjects with single hypertensive parent (SHP, n=43) and those with both hypertensive parents (BHP, n=7) were compared for their echocardiographic LVM characteristics. The unadjusted LVM did not differ significantly in the two groups and all other indexations of LVM in the SHP and BHP groups were statistically insignificant.

In this study the distribution of LVM was calculated in the whole population(n=100). (Table no.2)Unadjusted LVM was 130.9± 32.13 gm, LVM/Height was 76.72±17.61 gm/m,LVM/BMI was 6.1±1.19gm.m ^{2} /kg, LVMI was 71.34±14.61 gm/m, ^{2} and LVM/Height [27] was 30.88±6.98gm/m. [27]

The distribution of LVM in the whole population was then compared with the FraminghamHeart study [18] and another Indian study(Trivedi et al). [19]

The observations of our study were similar to the Indian study suggesting that, Indian values of LVM differs significantly from western values because of great difference in body habitus of Indian population as compared to the western population. We used the Framingham Heart Study criteria, Trivedi et al criteria, and the present study criteria (LVM more than 2 standard deviation above mean of whole population) to detect number of subjects who had LVH in the study and control population. No subjects in the control group were found to have LVH usuing any of these criteria. In the study population, usuing Framingham criteria only 1 subject was found to have LVH. Usuing the Trivedi et al criteria 4 subjects had LVH by adjusting LVM for height and 1 subject was found to have LVH by adjusting LVM for BSA.

In the study group usuing univariate analysis (Table no. 3, Graph no.2), unadjusted LVM had significant positive correlation with weight($r=0.7$,$p<0.0001$),BMI($r=0.64$,$p<0.0001$),BSA($r=0.58$,$p<0.0001$),MAP($=0.42$,$p=0.002$),DBP($r=0.4$,$p=0.004$),height($r=0.37$,$p=0.008$)andSBP($r=0.33$,$p=0.02$). Using multivariate analysis, weight could explain 49%,BMI 41%,BSA 33%, MAP 18%, DBP 16%, SBP 11% and height 14% of change in LVM. All the eight variables together explained 64.5% in unadjusted LVM($R^2=0.645$, $p<0.05$). In our study weight showed excellent positive correlation with LVM in both groups similar to other studies. [20][21][22][23] A likely possible explanation for the association of obesity with LVM may be the volume demand of adipose tissue. We observed that BMI had a statistically strong positive correlation in the control group. BSA also showed excellent positive correlation with unadjusted LVM in both the groups like some other studies. [24][25][26] Resting SBP and DBP had a positive correlation with LVM in the study group. Usuing multivariate analysis, in our study BSA and weight had highest predictive value for LVM in the control group.

CONCLUSIONS

The following conclusions were drawn from our study:

The mean LVM of normotensive offspring of hypertensive parents was significantly higher than the LVM of normotensive offspring of normotensive parents.

VST and PWT were greater in offsprings of hypertensive parents.

Our values of LVM differed significantly from the western values but matched with Indian values(Trivedi et al).

Weight, height, BMI and BSA had significant positive effect on LVM of an individual independent of parental history of hypertension.

In the subjects with parental hypertension, MAP,resting SBP and DBP also had significant positive effect on LVM.

Based on the present study, we would like to recommend that all normotensive young offsprings of hypertensive parents should undergo regular M- mode echocardiographic examinations for detection of LVH. Once detected early, necessary measures should be taken to reduce the risk of future cardiovascular events. However feasibility and cost effectiveness of 2D echo needs to be looked into, after validation of findings through long term prospective studies.

LIMITATION OF THE STUDY

The only limitation of this study was that the observer was not blinded to family history of the subjects.

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