Left ventricular mass in offspring of hypertensive parents: does it predict the future?

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Citation


Abstract

OBJECTIVE: to determine effect of parental hypertension on left ventricular mass (LVM) in normotensive offspring.

PARTICIPANTS AND METHODS: Echocardiography was performed in 45 normotensive offspring of hypertensive parent. These subjects were compared with 55 normotensive offspring of normotensive parents. RESULTS: 100 subjects were studied. They were divided into 2 groups, Hypertensive parental group and non Hypertensive parental group. Subjects of both the group were weight, height, body surface area (BSA) and body mass index (BMI) matched. While systolic blood pressure, diastolic blood pressure, mean arterial pressure and left ventricular mass (LVM) were higher in the hypertensive parental group as compared to non hypertensive parental group. The left ventricular mass index (LVMI) was higher in the study group and the difference was statistically significant. On correlation of anthropometric measurements and blood pressure with LV mass, in the non hypertensive parental group LVM had significant correlation with Weight (r=0.45, p=0.000, S) and BMI (r=0.41, p=0.002). While in hypertensive parental group LVM had significant correlation with Weight (r=0.38, p=0.008, S), BMI (r=0.35, p=0.016, S), BSA (r=0.36, p=0.013, S), SBP (r=0.36, p=0.014, S) and MAP (r=0.29, p=0.046, S). CONCLUSION: LV mass depends on variables like weight, BMI, BSA, SBP and MAP. There is a genetic predisposition to increased LV mass in children with parental hypertension and it could be more significant in the transmission of genetic susceptibility to increased LVM. The results highlight the need for further study in this direction.

INTRODUCTION

Left ventricular hypertrophy (LVH), defined either by electrocardiogram or echocardiography, is a potent independent risk factor for coronary heart disease (CHD) roughly doubling the risk of cardiovascular death in both men and women. LVH is the most powerful of any of the traditional risk factors in predicting not only death or myocardial infarction (MI) but also stroke, heart failure (HF), and other cardiovascular (CV) endpoints.

Longitudinal epidemiological studies have shown the utility of different traditional risk factors measured from childhood to adulthood in predicting sub clinical CV changes in adults. Among the sub clinical measures, left ventricular mass (LVM), assessed by 2D M-mode echocardiography, is recognized as an important and powerful predictor of CV morbidity and mortality, independent of other traditional risk factors.

Estimation of left ventricular mass by Echocardiography offers PROGNOSTIC INFORMATION beyond that provided by the evaluation of traditional cardiovascular risk factors. An increase in LVM predicts a higher incidence of clinical events including death, attributable to cardiovascular diseases.

The study was therefore carried out to detect the prevalence of major cardiovascular risk factor i.e. LVM in a healthy young population before the occurrence of actual manifestations of clinical CAD and CHF and to identify candidates who might require early intervention to reduce their risk of future cardiovascular events.

PARTICIPANTS AND METHODS

This study was conducted in department of Medicine in Jawaharlal Nehru Medical College and associated A.V.B.R. Hospital of DMIMS university, Wardha. 100 randomly selected, apparently healthy, normotensive male students in the age group of 19 to 25 years were studied. Definite exclusion criteria was established as follows.
EXCLUSION CRITERIA

- Persistent blood pressure ≥ 140/90 mm Hg.
- Current use of cardio active drug.
- Any cardiopulmonary disease.
- Any organic murmur.
- Any renal lump or renal bruit.
- Echocardiography showing evidence of any structural heart disease.
- Poor echocardiographic window

The subjects were examined for,

1. Weight in kilograms (kg)
2. Height in meters (m)
3. Blood pressure in mmHg

Body mass index (BMI) was calculated by the Quetelet formula

\[ \text{BMI} = \frac{\text{Weight (kg)}}{\text{Height (m)}^2} \]

Body surface area was calculated by the Mosteller formula.

\[ \text{Body surface area (m²)}: \sqrt{\frac{\text{Ht(cm) \times Wt(kg)}}{3600}} \]

Echocardiography of the subjects was done after a resting period of 3-4 minutes.

LVM was estimated by the modified cubed formula of Devereux and Reichek using measurements obtained in accordance with the ‘Penn’ convention.

\[ \text{LVM (gm)} = 1.04 ([\text{LVID} + \text{VST} + \text{PWT}]^3 - (\text{LVID})^3) - 13.6 \]

STATISTICAL ANALYSIS

Statistical analysis was performed using

- Descriptive statistics.
- Z test.
- Pearson’s correlation coefficient.
- p-value was tested at 5% level of significance
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**Figure 3**
Table 2: Comparison of adjusted LVM in the subjects based on parental history of hypertension

<table>
<thead>
<tr>
<th>Adjusted LVM</th>
<th>Total population (n=100)</th>
<th>Negative parental history of HTN (n=55)</th>
<th>Positive parental history of HTN (n=45)</th>
<th>z-value</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVM/Height (g/m²)</td>
<td>82.64±24.42</td>
<td>73.59±23.89</td>
<td>92.36±21.07</td>
<td>4.17</td>
<td>0.000</td>
</tr>
<tr>
<td>LVM/BSA (g/m²²)</td>
<td>77.84±21.33</td>
<td>70.70±20.87</td>
<td>85.57±18.64</td>
<td>4.01</td>
<td>0.000</td>
</tr>
<tr>
<td>LVM/BMI (g/m²)</td>
<td>6.12±1.71</td>
<td>5.61±1.62</td>
<td>6.73±1.61</td>
<td>3.41</td>
<td>0.001</td>
</tr>
<tr>
<td>LVM/IBI (g/m²/m²²)</td>
<td>32.68±9.97</td>
<td>29.28±9.47</td>
<td>35.82±9.04</td>
<td>4.05</td>
<td>0.000</td>
</tr>
</tbody>
</table>

**Figure 4**
Graph 2: Comparison of adjusted LVM in the subjects based on parental history of hypertension

**Figure 6**
Graph 3: Correlation of anthropometric measurements and blood pressure with LVM.

**Figure 5**
Table 3: Correlation of anthropometric measurements and blood pressure with LVM.

<table>
<thead>
<tr>
<th>Observed parameters</th>
<th>Total population (n=100)</th>
<th>Pearson Correlation coefficient</th>
<th>R²</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>0.46</td>
<td>0.211</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>Height</td>
<td>0.08</td>
<td>0.006</td>
<td>0.405</td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>0.41</td>
<td>0.168</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>BSA</td>
<td>0.44</td>
<td>0.193</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>0.36</td>
<td>0.152</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>0.33</td>
<td>0.108</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>MAP</td>
<td>0.31</td>
<td>0.0941</td>
<td>0.024</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 7**
Table 4: Correlation of anthropometric measurements and blood pressure with LVM according to parental history of hypertension.
RESULTS

100 patients were studied. They were divided into 2 groups-

1. Hypertensive parental group- subjects with h/o parental hypertension (n=45)

2. Non hypertensive parental group- subjects with no h/o parental hypertension (n=55).

In all subjects anthropometric measurements i.e. weight, height, body surface area (BSA) and body mass index (BMI) did not differ significantly in the two groups. While systolic blood pressure, diastolic blood pressure and mean arterial pressure were higher in the hypertensive parental group as compared to non hypertensive parental group. Left ventricular mass (LVM) was higher in hypertensive parental group as compared to non hypertensive parental group.

LVM adjusted for anthropometric measurements allows comparison between subjects with different height, BSA, BMI and negates any differences which occur on account of these variables. Even after adjustment for these variables, LVM was found to be higher in hypertensive parental group as compared to non hypertensive parental group. On correlation of anthropometric measurements and blood pressure with LV mass, in the non hypertensive parental group LVM had significant correlation with Weight (r=0.45, p=0.000, S) and BMI (r=0.41, p=0.002). While in hypertensive parental group LVM had significant correlation with Weight (r=0.38, p=0.008, S), BMI (r=0.35, p=0.016, S) BSA (r=0.36, p=0.013, S), SBP (r=0.36, p=0.014, S) and MAP (r=0.29, p=0.046, S).

DISCUSSION

Left ventricular hypertrophy has been established as an independent risk factor for the development of cardiovascular morbidity and mortality1,2. Therefore, understanding the determinants of left ventricular mass in children and adolescents has become increasingly important. It has been suggested that factors, such as blood pressure, body growth and ponderosity may have an effect on left ventricular mass. However, the extent to which these other factors have an impact on left ventricular mass has been controversial2.

In our study Weight, height, BMI3,4 and BSA5 did not differ significantly in the two groups. Thus the subjects in the two groups were matched for anthropometric measurements.

SBP6,7,8,9 and DBP8,9,10 were significantly higher in the hypertensive parental group as compared to non hypertensive parental group suggesting that offspring of hypertensive parents are more likely to develop hypertension in future.

Unadjusted LVM was significantly higher in hypertensive parental group as compared to non hypertensive parental group5,8,11,12.

Even when LVM was adjusted for height, BSA and BMI to negate any differences which can occur because of these variables, LV mass was found to be significantly higher in hypertensive parental group.

Weight and BMI had significant positive correlation with LV mass irrespective of parental history of hypertension, as weight and BMI increased LV mass also increased.
BSA, SBP and MAP had significant positive correlation with LV mass in hypertensive parental group; same was not true for non hypertensive parental group.

CONCLUSION

LV mass depends on variables like weight, BMI, BSA, SBP and MAP. There is a genetic predisposition to increased LV mass in children with parental hypertension and it could be more significant in the transmission of genetic susceptibility to increased LVM. The results highlight the need for further study in this direction.

STRENGTH & LIMITATIONS

The greatest strength of the study was that the participants in both the group were matched for anthropometric characteristics (weight, height, BMI and BSA).

This has been a single contact study, hence for further validation, the high risk subjects should be followed up for regular, progressive monitoring of LV mass, and development of hypertension.

References

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