Left Ventricular Mass and Geometry in Obese Children

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Abstract: This study was to evaluate the left ventricular mass index (LVMI) and geometry in obese children. Forty-nine obese children, median age of 9.7 (range 3.4-15.4 years), underwent echocardiography to assess LVMI and Relative Wall Thickness (RWT). LV geometry was categorized as normal, concentric remodeling, eccentric hypertrophy and concentric hypertrophy. Mean weight was 61.8±22.0 kg, BMI 29.9±5.8 kg·m⁻², LVMI 38.3±8.9 g·m⁻², and percentage of actual weight to ideal body weight-for-height (IBW %) 172.4±28.3%. Twenty-one children had mild to moderate obesity (group 1) and 28 had severe obesity (group 2). Twenty-six children had normal LV geometry, 2 had concentric remodeling, 15 had eccentric hypertrophy and 6 had concentric hypertrophy. LVMI and abnormal geometry in group 2 were significantly greater than in group 1 (40.7±8.8 vs 35.1±8.1 g·m⁻², 60.7 vs 28.6%, p = 0.03). Thirty-seven children (75.5%) had normotensive whereas 12 (24.5%) had systemic hypertension. The LVMI and abnormal geometry were not significantly different in both groups (37.6±9.7 vs 40.4±5.7 g·m⁻², 40.5 vs 60.7%). Left ventricular mass and abnormal LV geometry were increased in obese children especially in severe obesity. These may increase cardiovascular risk in the future. Weight control to decrease the severity of obesity should be recommended.

Key words: Body mass index, echocardiography, left ventricular hypertrophy, obesity, pediatrics

INTRODUCTION

The prevalence of obesity in children and adolescents is increasing (Freedman et al., 1997; Ogden et al., 2006). In 2003-2004, 17.1% of US children and adolescents (age 2-19 years) were overweight. The prevalence of overweight female and male children and adolescents increased from 13.8 and 14.0% in 1999-2000 to 16.0 and 18.2% in 2003-2004, respectively (Ogden et al., 2006). Overweight and obese adolescents often maintain overweight and obesity in adulthood (Guo et al., 1994; Guo et al., 2002). Obesity in children has affected many systems causing diseases such as systemic hypertension, diabetes mellitus, hyperlipidemia and obstructive sleep apnea. Increased left ventricular mass (LVM) has been reported in overweight children and adolescents and strongly associated with lean body mass and systolic blood pressure (Daniels et al., 1995a). Earlier report revealed that obese children had increased LVM and cardiac output (Humphries et al., 2002). The left ventricular hypertrophy (LVH) and geometry can predict an increased incidence of cardiovascular events especially eccentric LVH which is consistently associated with markedly increased cardiovascular risk (Koren et al., 1991). De Simone et al. (1997) demonstrated that the risk of LVH was significantly higher in children with a high casual blood pressure, compared with children with normal blood pressures, independent of the effect of obesity and they recommended an aggressive
approach to prevention and treatment of obesity in pediatric patients with hypertension to reduce the future cardiovascular morbidity in these children. The aim of this study was to evaluate left ventricular mass index (LVMI) and left ventricular (LV) geometry in young obese children.

**MATERIALS AND METHODS**

**Study Population**

Between August 2004 and April 2006, 49 obese children attending at Nutrition Clinic, Division of Nutrition, Department of Pediatrics, Faculty of Medicine, Ramathibodi Hospital with median age of 9.7 years (range 3.4-15.4 years) underwent history and physical examination, anthropometric assessment (body weight, height, waist and hip circumference and body mass index) and echocardiography. Blood pressure greater than 95th percentile for age was considered to have systemic hypertension. The Body Mass Index (BMI) was calculated by using the formula:

\[
 \text{BMI} = \frac{\text{weight (kg)}}{\text{height (m)}^2}
\]

The percentage of actual weight to ideal body weight for height (IBW %) was calculated as the actual weight divided by the ideal body weight for height at the 50th percentile of reference and times 100. Obesity is defined as a BMI-age-and sex-specific cut off points (Cole et al., 2000). The degree of obesity is defined as followings: IBW% <140%, mild; 141-160%, moderate and >160%, severe obesity. The parents of the patients were informed about the risk and benefit of the study.

**Echocardiography Study**

Echocardiography was performed in all children using Hewlett Packard Sonos 4500. Left ventricular end-diastolic dimension (LVEDD), left ventricular end-systolic dimension (LVESD), interventricular septal thickness and posterior wall thickness were measured through 2-dimensional guided M-mode echocardiography using parasternal short axis view at the level of papillary muscles. Left ventricular mass (LVM) was calculated by the formula described by Devereux and Reichek (1977). LV mass index (LVMI) was obtained by dividing LV mass by height$^2$ to normalize and linearize the relations between LVM and height (De Simone et al., 1992). The percentile of LVMI was obtained, based on normal value (De Simone et al., 1992). LVMI was used to evaluate left ventricular hypertrophy (LVH) adjusted to body size as described elsewhere (De Simone et al., 1992). LVH is diagnosed when LVMI is over the 95th percentile for healthy children and adolescents (38.6 g m$^{-2.7}$) (Daniels et al., 1995). Relative Wall Thickness (RWT) was measured at end diastole as the ratio of the sum of posterior wall thickness and septal thickness over LVEDD. The sex-specific 95th percentile for LVMI from normal children and adolescents was used as one cut-point. A RWT of 0.41 was used, which represents the 95th percentile for relative wall thickness for normal children and adolescents. This value was also used by Ganau et al. (1992) for partitioning by relative wall thickness in adults. Cutoff levels for LV mass and RWT were created to evaluate LV geometry. From these values LV can be categorized into four categories: normal, concentric remodeling, eccentric hypertrophy and concentric hypertrophy. Children with normal geometry had LV mass and RWT below the 95th percentile. Concentric remodeling was defined as normal LVMI but elevated RWT, eccentric hypertrophy was defined as elevated LVMI with normal RWT and concentric LV hypertrophy was defined as both LVMI and RWT greater than the 95th percentile.

The cutoff level used to define the most severe LVH was a LVMI of 51 g m$^{-2.7}$. This value represents approximately the 97.5th percentile for LVMI in adults.
Left Ventricular Systolic Function

Left ventricular ejection fraction (LVEF) and left ventricular fractional shortening (LVFS) were calculated by Wilson et al. (1981) formula. The normal values for LVEF and LVFS are >55% and >25%, respectively.

Left Ventricular Dimension

The predicted LVEDD was calculated using equation by Henry et al. (1980):

\[
\text{Predicted LVEDD} = [45.3 \times (\text{body surface area (m}^2) \times 0.03 \times \text{age (years)}] - 7.2.
\]

The ratio of left ventricular diastolic dimension to predicted LVEDD expressed in percentage (LVEDD %) was calculated using the following formula:

\[
\text{LVEDD} \% = \left( \frac{\text{Measured LVEDD}}{\text{Predicted LVEDD}} \right) \times 100.
\]

The normal value for LVEDD% is < 112%. When the% LVEDD is >117%, there is left ventricular enlargement.

Statistical Analysis

Statistical analyses were performed using SPSS 13.0 for window soft ware. Data were presented as Mean ± Standard Deviation (SD) for continuous variables and as proportions for categorical variables. The unpaired Student t-test was used to compare numeric variables between groups. Mann-Whitney U-test was used to compare nonparametric variables between groups. Correlations between numeric variables were determined by Spearman correlation analysis. A p-value <0.05 was considered to be statistically significant.

RESULTS AND DISCUSSION

Forty-nine children had mean weight 61.8±22.0 kg, BMI 29.9±5.8 kg m⁻² and percentage of actual weight to ideal body weight for height (IBW%) 172.4±28.3%. According to grading severity using IBW%; 3, 18 and 28 children had mild, moderate and severe obesity, respectively. Cardiac dimension determined by LVEDD% was 98.4±7.8%. None had LVEDD% >117%. LV systolic function determined by the LVFS and LVEF were 37.5±4.8 and 67.7±5.9%, respectively. Hence, all children had normal LV dimension and systolic function. Overall, mean LVM, LVMI and RWT were 100.4±37.7 g, 38.3±8.9 g m⁻² and 0.34±0.06, respectively. Twenty children (40.8%) had LVH (LVMI >38.6 g m⁻²). Three of these children had LVMI >51 g m⁻². Of these 3 children, two of them had severe obesity (IBW% = 169 and 215%) and one had moderate obesity (IBW% = 156%), however, none of these 3 children had systemic hypertension. Eight children (16.3%) had RWT >0.41. By LVMI and RWT, LV geometry was categorized into four groups: normal (n = 26), concentric remodeling (n = 2), eccentric hypertrophy (n = 15) and concentric hypertrophy (n = 6). Figure 1a demonstrated LV geometry according to severity of obesity, mild to moderate vs severe obesity. Thirty-seven children (75.5%) had normotension whereas 12 (24.5%) had systemic hypertension. Figure 1b demonstrated LV geometry according to systemic blood pressure, normotension vs systemic hypertension. Characteristics including age, sex, body weight, BMI, IBW%, blood pressure, LVMI and LV geometry were compared in normotensive and hypertensive obese children (Table 1). Mean age in hypertensive group was significantly greater than in normotensive group (11.5±2.4 vs 9.0±2.4 years). Body weight, BMI and IBW% were significantly higher in hypertensive group than in normotensive group (Table 1). LVMI and abnormal LV geometry were not significantly different in
Fig. 1: Distribution of left ventricular geometry according to severity of obesity in this cohort study (a) and according to systemic blood pressure in this cohort study (b).

Table 1: Demonstration of characteristics, blood pressure, left ventricular mass index, and abnormal left ventricular geometry in normotensive and hypertensive obese children

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Normotensive (n = 37)</th>
<th>Hypertensive (n = 12)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>9.4±2.4</td>
<td>11.5±2.4</td>
<td>0.005</td>
</tr>
<tr>
<td>Female/Male ratio</td>
<td>17/9</td>
<td>20/17</td>
<td>0.22</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>54.8±16.9</td>
<td>83.3±22.5</td>
<td>0.001</td>
</tr>
<tr>
<td>BMI (kg m⁻²)</td>
<td>27.9±4.3</td>
<td>36.2±5.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>IBW%</td>
<td>163.3±22.2</td>
<td>200.6±26.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>107.5±7.6</td>
<td>133.8±9.1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>65.4±8.5</td>
<td>74.1±11.6</td>
<td>0.03</td>
</tr>
<tr>
<td>LV mass index (g m⁻²)</td>
<td>37.6±9.7</td>
<td>40.4±5.6</td>
<td>0.23</td>
</tr>
<tr>
<td>Abnormal LV geometry</td>
<td>15 (40.5%)</td>
<td>8 (66.7%)</td>
<td>0.18</td>
</tr>
</tbody>
</table>


Both groups. However, there were 8 of 12 (66.7%) in hypertensive group had LVMi >38.6 g m⁻² compared to 13 of 37 (35.1%) in normotensive group. We also performed the subgroup analysis by severity into group 1: mild to moderate obesity (n = 21) and group 2: severe obesity (n = 28).
Table 2: Demonstration of characteristics, blood pressure, left ventricular mass index and abnormal left ventricular geometry in different severity of obese children

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Mild to moderate (n = 21)</th>
<th>Severe (n = 28)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>9.7±2.2</td>
<td>9.5±3.0</td>
<td>0.78</td>
</tr>
<tr>
<td>Female/male ratio</td>
<td>12/9</td>
<td>12/16</td>
<td>0.35</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>53.2±15.8</td>
<td>68.3±24.0</td>
<td>0.01</td>
</tr>
<tr>
<td>BMI (kg m⁻²)</td>
<td>25.7±2.4</td>
<td>33.1±5.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>IBW%</td>
<td>147.3±7.1</td>
<td>191.3±22.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>106.5±8.3</td>
<td>119.5±14.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>63.6±7.2</td>
<td>74.5±10.8</td>
<td>0.01</td>
</tr>
<tr>
<td>LV mass index (g m⁻²)</td>
<td>35.1±8.1</td>
<td>40.7±8.8</td>
<td>0.03</td>
</tr>
<tr>
<td>Abnormal LV geometry</td>
<td>6 (28.6%)</td>
<td>17 (60.7%)</td>
<td>0.03</td>
</tr>
</tbody>
</table>


Characteristics including age, sex, body weight, BMI, IBW%, blood pressure, LVMI and LV geometry were compared in both groups (Table 2). LVMI, blood pressure and abnormal LV geometry were significantly higher in group 2 than in group 1 (Table 2).

There was fair correlation between LVMI and IBW% (r = 0.31, p = 0.01) but no correlation between LVMI and systemic hypertension (r = 0.17, p = 0.1).

Obesity in adolescents has been associated with increased values of LVM and high prevalence of left ventricular hypertrophy (Chiniali et al., 2006). Echocardiography performed on asymptomatic severely obese adult patients can detect alterations in the cardiac structure including enlarged left chambers (42.9%), diastolic dysfunction (54.6%) and left ventricular hypertrophy (82.1%), leading to obesity cardiomyopathy, the development of heart failure, arrhythmias and sudden death (Rocha et al., 2007). One study reported the autopsies of 210 children aged 5-15 years who suffered a violent death in Finland and found that the ponderous index was a significant predictor for heart weight and the presence of coronary intimal fatty streaks (Kortelainen et al., 1997). Interestingly, coronary fatty streaks were not found at all in the leanest individuals (Kortelainen et al., 1997). The cardiac adaptation to obesity in adults consists of left ventricular dilatation and hypertrophy regardless of the level of blood pressure (Messerli, 1983). In present study there was left ventricular hypertrophy in obese children but not dilatation associated with obesity. This may be explained by the early onset of detection in children which dilatation may not develop yet.

The present study has been studied in younger children with median age of 9.7 years, range from 3.4 to 15.4 years. LV dimension and systolic function were normal in all of these obese children. Present findings demonstrated that, even in the younger age group, there was the high prevalence of LVH (40.8%). Interestingly, 3 of 49 (6.1%) with normotension had severe LVH (LVMI of 51 g m⁻²²) which has previously been shown to be associated with a fourfold increase in risk for cardiovascular morbidity in adults (De Simone et al., 1995). Abnormal LV geometry has been demonstrated in 23 obese children (46.9%). Moreover, concentric LV hypertrophy with associated with increased cardiovascular morbidity in adults 11 has been demonstrated in 6 children. The LVH has been shown to be an independent risk factor for cardiovascular morbidity and mortality in adult population (Levy et al., 1990). Importantly, LVH may start early even in the younger age group as shown in this study. Factors associated with LVH include obesity, high blood pressure and dietary sodium intake (Daniels et al., 1995b). In this study, the severity of obesity was an important factor in developing LVH and abnormal LV geometry in obese children. Mean LVMI in severe obese children was significantly greater than in mild to moderate obese children (40.7±8.8 vs 35.1±8.1 g m⁻²², p = 0.03) and abnormal LV geometry was demonstrated more in severe obese children than in mild to moderate obese children (60.7 vs 28.6%). There also was fair correlation between %IBW (which indicated severity of obesity) and LVMI (which indicated LVH).
Only 12 obese children had high blood pressure. Although, mean LVMI in hypertensive group and abnormal LV geometry were not significantly different compared to normotensive group (40.4±5.6 vs 37.6±9.7 g m⁻², p = 0.23 and 66.7 vs 40.5%) and no correlation between systolic blood pressure and LVMI (r = 0.17) but systemic hypertension may be a factor in developing LVH in obese children according to 8 of 12 hypertensive obese children had LVH (LVMI >38.5 g m⁻²) compared to 13 of 37 in normotensive obese children. This may be explained by the small number of hypertensive obese children in our study.

Both LVH and abnormal LV geometry may therefore be affected not only by severity of obesity, but also by other factors such as systemic hypertension, hypercholesterolemia and obesity itself. Whether the findings of LVH and abnormal LV geometry especially LV concentric hypertrophy in younger obese children have any effect on cardiovascular morbidity and mortality when these children grow up to be adult or not is interesting and needed to have a long-term follow-up. However, there was evidence that cardiac adaptation change can occur even in these younger obese children.

CONCLUSION

The left ventricular mass and abnormal LV geometry were increased in obese children especially in severe obesity. These may increase cardiovascular risk in the future. Weight control to decrease the severity of obesity should be recommended.

ACKNOWLEDGMENTS

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REFERENCES


