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Original Article

Obesity and left ventricular mass in children

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Abstract

Background Obesity has negative effects on cardiac function during growth leading to increased heart size and mass, as a result of higher stroke volume and cardiac output.

Objective To assess for a relationship between obesity and left ventricular mass (LVM) in children, as well as to assess for a correlation between the duration of obesity and LVM.

Methods This cross-sectional study was conducted from October 2011 until February 2012 in Medan and included 30 obese and 30 normal weight children, aged 6 to 13 years. All subjects underwent complete echocardiography examinations to assess LVM and other left ventricular parameters. The Devereux formula was used to measure LVM.

Results During the study, 65 children underwent echocardiography, but 5 were subsequently excluded. The left ventricular dimensions in the obese group were significantly higher compared to normal weight group with regards to interventricular septum at end diastole (IVSd), interventricular septum at end systole (IVSS), left ventricular internal diameter at end diastole (LVIDd), left ventricular internal diameter at end systole (LVIDs), left ventricular posterior wall thickness at end diastole (LVPWd), left ventricular mass (LVM), and left ventricular mass index (LVMI) (P=0.0001). Duration of obesity and LVM had a moderate, positive correlation (r=0.407).

Conclusion There is significantly higher LVM in the obese group than in the normal weight group. The duration of obesity had a moderate, positive correlation to LVM. **[Paediatr Indones. 2015;55:224-9]**.

Keywords: *obesity, children, left ventricular mass*

Obesity has become a worldwide health problem. The WHO stated that obesity was a global epidemic that must be solved.[1] The prevalence of overweight and obesity in children is increasing in both developing and developed countries.[2] Of the 155 milion overweight children worldwide, 30 to 45 million are obese.[3] Obesity is a leading cause of morbidity and mortality.[4] The condition of being overweight during childhood and adolescence is likely to impact future cardiovascular health.[5] Atherosclerotic lesions start to develop in early childhood and progress into irreversible lesions in adolescence and adulthood.[6] There is substantial evidence that the atherosclerotic process leading to coronary heart disease begins in childhood and adolescence. Obesity during childhood is associated with an increase in cardiovascular mortality in adult life.[7]

Studies in adults using echocardiography, catheterization, and necropsy examination have

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shown relationships between morbid obesity, structural alterations of the heart, and systolic function, which may lead to a clinical syndrome known as "obese cardiomyopathy". However, the relationship between obesity and cardiac structure as well as function in children has been less well documented and conflicting results have been reported.[2] Body mass index (weight in kilograms/height^2 in meters) is frequently used as a surrogate measure of obesity in children and adults.[8,9] Increased body mass index (BMI) is an independent risk factor for the development of elevated blood pressure, metabolic syndrome, abnormal vascular wall thickness, endothelial dysfunction, and left ventricular hypertrophy.[3,10] The increase in left ventricular mass (LVM) related to obesity is probably more than a mere physiologic adaptation.[11]

The aim of this study was to assess for a relationship between obesity and LVM in children, as well as to assess for a correlation between duration of obesity and LVM.

Methods

A cross-sectional study was conducted from October 2011 until February 2012 in Medan, involving 30 obese and 30 normal weight children, aged 6 to13 years. Subjects were primary and junior high school students, collected by consecutive sampling. Both groups underwent complete echocardiography examinations to assess LVM and other left ventricular parameters. The Devereux formula was used to measure LVM.[12]

Data were obtained from physical examinations, echocardiography examinations, parental interviews and questionnaires. Subjects were divided into 2 groups based on nutritional status. Children with BMI ≥95th percentile were categorized as obese and children with BMI ≥5th percentile and < 85th percentile were categorized as normoweight, based on the BMI index for age and sex from *the Centers for Disease Control, 2000*.[13] Both groups underwent complete echocardiography examinations (2D, M-Mode and Doppler) using a *Toshiba Nemio 3.0* machine. Echocardiography was performed by a pediatric cardiologist at Bunda Thamrin Hospital, Medan to assess LVM and other left ventricular dimensions, such as interventricular septum at end diastole (IVSd), interventricular septum at end systole (IVSS), left ventricular internal diameter at end diastole (LVIDd), left ventricular internal diameter at end systole (LVIDs), left ventricular posterior wall thickness at end diastole (LVPWd), left ventricular mass (LVM), and left ventricular mass index (LVMI). Left venricular mass index calculated as lft ventricular mass (LV) in grams divided by height in meters to the 2.7th power (g/m$^{2.7}$).[14]

We analyzed data using *SPSS version 17* software. Student's T-test was used to assess for a relationship between obesity or normoweight and LVM. Regression linear analysis was used to assess for a correlation between the duration of obesity and LVM. The significance level was accepted as $P<0.05$ and 95% confidence interval (CI). This study was approved by the Ethics Committee of the University of North Sumatera Medical School.

Results

During the study, 65 children underwent echocardiography (32 obese and 33 normal weight children). Of the 32 obese children, 1 child had an innocent murmur grade II/6 on physical exam and 2 children were excluded due to mild pulmonary regurgitation. Of the 33 normal weight children, 3 were excluded due to mild pulmonary regurgitation (1 child), mild-moderate pulmonary regurgitation (1 child), and suspected atrial septal defect (1 child). Hence, there were 30 obese and 30 normal weight subjects, matched for age and gender.

The mean age in both groups was 9.6 years, with mostly male subjects (66.7%). The mean weights were 54.3 kg in the obese group and 31.9 kg in the normal weight group. The mean BMIs were 26.4kg/m^2 in the obese group and 17.6 kg/m^2 in the normal weight group. The mean duration of obesity was 4.3 years and 56.7% of subjects in the obese group had fathers with histories of obesity (**Table 1**).

Table 2 shows the significant differences in left ventricular dimension between the obese and normal weight groups with regards to IVSd, IVSs, LVIDd, LVIDs, LVPWd, LVM and LVMI ($P=0.0001$). However, the LVPWS were not significantly different between groups.

Table 3 and **Figure 1** show that duration of obesity and LVM had a moderately positive correlation

Table 1. Characteristics of subjects

Characteristics	Normal weight (n = 30)	Obese (n = 30)
Mean age (SD), years	9.6 (1.58)	9.6 (1.58)
Gender, n		
Male	20	20
Female	10	10
Mean weight (SD), kg	31.9 (8.58)	54.3 (11.03)
Mean height (SD), cm	133.4 (13.19)	142.9 (11.08)
Mean body mass index (SD), kg/m^2	17.6 (1.26)	26.4 (3.55)
Body mass index of age percentile, n		
50 – 74	23	0
75	1	0
76 – 84	6	0
96 – 97	0	6
> 97	0	24
Mean blood pressure (SD), mmHg		
Systolic	93.0 (8.36)	121.6 (8.74)
Diastolic	61.6 (8.74)	76.6 (6.60)
Mean heart rate (SD), bpm	88.8 (6.45)	93.7 (9.30)
Mean duration of obesity (SD), years	-	4.3 (1.72)
History of obesity, n		
Father only	0	17
Mother only	0	5
Father and mother	0	2
None	0	6

Table 2. Left ventricular dimensions in the obese and normal weight groups

Left ventricular dimensions, mean (SD)	Normal weight (n = 30)	Obese (n = 30)	95% CI of differences	P value
IVSd, cm	0.7 (0.15)	0.9 (0.15)	0.144 to 0.266	0.0001
IVSs, cm	0.8(0.17)	1.1 (0.16)	0.097 to 0.271	0.0001
LVIDd, cm	3.7(0.30)	2.5 (0.37)	-1.501 to -1.102	0.0001
LVIDs, cm	2.3(0.26)	3.2 (1.0)	0.430 to 1.182	0.0001
LVPWd, cm	0.6 (0.11)	1.7 (0.97)	0.676 to 1.405	0.0001
LVPWs, cm	0.7 (0.19)	0.8 (0.22)	-0.026 to 0.192	0.130
LVM, g	67.7 (17.60)	104.7 (21.46)	28.877 to 45.156	0.0001
LVMI, g/m$^{2.7}$	31.4 (5.64)	69.9 (39.90)	23.865 to 53.144	0.0001

IVSd: interventricular septal thickness at end-diastole, IVSs: Interventricular septal thickness at end-systole, LVIDd: left ventricular internal dimension at end-diastole, LVIDs: left ventricular internal dimension at end-systole, LVPWd: left ventricular posterior wall thickness at end-diastole, LVPWs: left ventricular posterior wall thickness at end-systole, LVM: left ventricular mass, LVMI: left ventricular mass index.

Table 3. Duration of obesity on LVM

Variable	r	R^2	Equation of line	P value
Duration of obesity	0.407	0.166	LVM = 82.924 + 5.069 (duration of obesity)	0.025

(r=0.407). The determination coefficient was 0.166, indicating that the regression equation obtained can explain 16.6% variation LVM or equation of line unfavourable explain variable LVM.

Discussion

Obesity is influenced by different factors, including genetics, metabolism, behavior, culture, and environ-

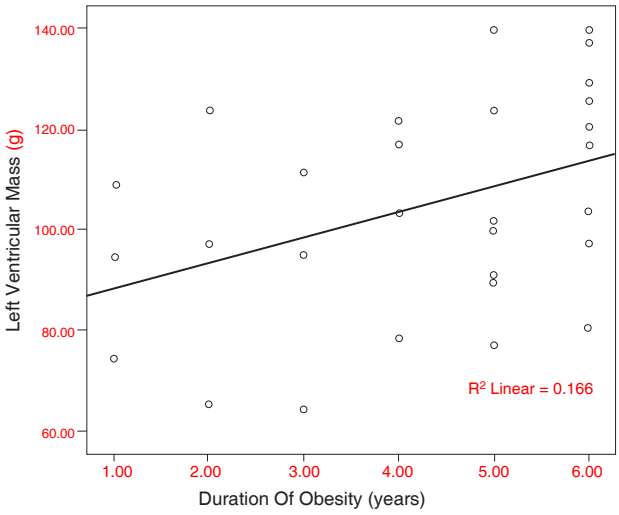

Figure 1. Correlation between duration of obesity and LVM

ment.[7,14] In our obese subjects, family history of obesity was observed in 56.7% of subjects' fathers, 16.7% of subjects' mothers, and 6.7% of both parents. Twenty percent of subjects had no parental history of obesity. None of the normal weight subjects had obese parents. Male obese subjects outnumbered females (66.7% vs. 33.3%, respectively).

There is clearly an important genetic contribution to BMI and obesity.[15] All available data suggest that 60 - 80% of the observed variance in human body weight can be accounted for by inherited factors.[16] Parental BMI is the most powerful determinant of their offspring's BMI. Inheritance of BMI is thought to arise from a combination of both inherited genes and shared environment, in which both parents contribute equally to the body composition of their offspring.[17] A study in England suggested that the association between maternal BMI and offspring BMI was similar to that between paternal BMI and offspring BMI.[15]

A Brazilian study showed that increased resting heart rate (RHR) was significantly associated with dyslipidemia in obese children and adolescents, and that elevated RHR may be used to screen subjects at increased risk of atherosclerosis development.[18] A high body fat is responsible for releasing inflammatory adipokines into the bloodstream. These adipokines play an important role in the pathogenesis of many chronic diseases as well as sympathetic and parasympathetic changes in children and adolescents, which can result in an increased RHR.[18] We observed that the mean heart rate in the obese group was higher than that of normal weight children (P=0.020), however, we did not assess for dyslipidemia in our subjects.

Cardiovascular involvement, such as hypertension, increased LVM, or cardiac malfunction can be identified in the early stages of obesity.[19] We found that diastolic and systolic blood pressures were higher in obese children than in normal weight children (P=0.0001). Hypertension and obesity are closely linked and often exert a dual burden on the left ventricle, leading to both dilatation and hypertrophy. Changes such as left ventricular hypertrophy, left atrial enlargement and subclinical impairment of left ventricular function are believed to be the precursors to more overt forms of cardiac dysfunction and heart failure later in life. Different combinations of volume and pressure overload associated with obesity and its comorbidities can cause different left ventricular geometry adaptations in both children and adults. These geometric patterns have prognostic implications above that accounted for by increased LVM.[20] We found significant differences in left ventricular dimension at the end of the systolic phase and the end of the diastolic phase with regards to interventricular septal thickness and left ventricular internal dimension, left ventricular posterior wall thickness at end diastole, LVM and LVMI between the two groups (P=0.0001). Nevertheless, left ventricular posterior wall thickness at the end of systole (LVPWs) was not significantly different between groups. This indicates that differences in left ventricle geometry in obese children are related to the filling of the left ventricle. As such, this condition is indicative of myocardium relaxation disorders. Diastolic properties of the left ventricle are probably influenced by multiple factors, including completeness of ventricular relaxation, composition and thickness of the ventricular wall. The completeness of left ventricular relaxation is thought to be an important determinant of diastolic pressure and compliance.[21`]

In our study, LVM was 1.5 times higher and LVMI was 2 times higher in the obese group compared to the normal weight group. These findings are indicative of increased cardiac size and mass in obese children.

Longer duration of severe obesity has been suggested to be the main predisposing factor of

cardiac dysfunction.[22] We found the average duration of obesity to be 4.3 years. The duration of obesity had a moderately positive strong (r=0.407) relationship to LVM. In this study, we took care to exclude children with a history of certain diseases in order to avoid the possibility of heart dimension change due to the consequences of other systemic diseases.

We also excluded 2 obese children with mild pulmonary regurgitation. Three normal weight children were excluded, namely those with mild pulmonary regurgitation, moderate pulmonary regurgitation and an echo drop finding which we suspected to be an ASD. Transesophageal echocardiography (TEE) was suggested to confirm the diagnosis of ASD for this participant. On physical examination, one obese child had an innocent murmur degree II/6. Appoximately half of all children have a detectable murmur when the precordium is auscultated. Yet the incidence of congenital heart disease is only 0.8%. Thus, the problem for primary care givers is to distinguish murmurs related to an underlying heart defect from murmurs created by the normal blood flow within a structurally sound cardiovascular system.[23]

Abnormal cardiac geometry and altered diastolic function in morbidly obese adolescents improve with significant weight loss. This is important because reversal of abnormal cardiac geometry might improve predictors of future cardiovascular morbidity in young people.[24] Another study demonstrated that obese children and adolescents have early significant changes in left ventricular wall dimensions and early diastolic filling compared to non-obese subjects, and these changes were reversible with weight reduction.[25]

Asssessment of LVM in the obese children in our study lacked a program of body weight reduction, so we did not assess LVM reversibility. Another limitation of our study was that we did not assess subjects' birth weight, lipid profile status, or insulin sensitivity. Furthermore, duration of obesity was based parental reporting, hence, may have been subject to recall bias. Further study is needed to assess cardiac dimensions in obese children after a weight reduction program.

In conclusion, there is a significant difference in LVM between obese and normal weight children. The duration of obesity is moderately, positively, and significantly correlated to LVM.

Conflict of interest

None declared.

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