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Original Article

Relationship between obesity and left ventricular hypertrophy in children

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Abstract

Background Obesity is a chronic metabolic disorder associated with cardiovascular disease (CVD) increasing morbidity-mortality rates. It is apparent that a variety of adaptations/alterations in cardiac structure and function occurs as excessive adipose tissue accumulates. This leads to a decrease in diastolic compliance, eventually resulting in an increase in left ventricular filling pressure and left ventricular enlargement.

Objective To evaluate left ventricular hypertrophy (LVH) among obese using electrocardiographic (ECG) criteria.

Methods A cross-sectional study was conducted on 74 children aged 10-15 years from February 2009 to October 2009. The subjects were divided into obese and control groups. Physical examination and standard 12 lead electrocardiography (ECG) were done in both groups.

Results Of 37 obese children, LVH were featured in 3 subjects, while in control group, only 1 child had LVH (P= 0.304). We found that mean RV6 in obese and control group were 9.8446 (SD 3.5854) and 11.9662 (SD 3.2857), respectively (P= 0.005). As an additional findings, we found that birth weight was related to obesity in children.

Conclusion There is no relation between obesity and left ventricular using ECG criteria in obese children aged 10–15 years. **[Paediatr Indones. 2010;50:331-5]**.

Keywords: *left ventricular hypertrophy, electrocardiography*

Obesity, which becoming a global epidemic, is an independent risk factor for cardiovascular diseases such as arterial hypertension, congestive heart failure, and ischaemic heart disease, and has been proposed as a risk factor for ventricular arrhythmias and sudden death.[1,2] Obesity is a chronic metabolic disorder associated with cardiovascular disease (CVD) and increases morbidity and mortality rates. It is apparent that a variety of adaptations/alterations in cardiac structure and function occurs as excessive adipose tissue accumulates, even in the absence of systemic hypertension or underlying organic heart disease. To meet increased metabolic needs, circulating blood volume, plasma volume, and cardiac output all increase. The increase in blood volume in turn increases venous return to the right and the left ventricles, eventually producing dilatation of these cardiac cavities, increasing wall tension. This leads to left ventricular hypertrophy (LVH), which is accompanied by a decrease in diastolic chamber compliance, eventually resulting in an increase in left ventricular filling pressure and left ventricular enlargement.[3,4]

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Left ventricular hypertrophy takes weeks even months to years to develop. It has been proposed that a cardiac renin-angiotensin system and angiotensin converting enzyme activity may be an important determinant of the hypertrophic response. There are two predominant types of hypertrophy, concentric and dilatation. In concentric type, wall thickness is increased relatively to cavity dimensions, so the enlargement happens to be eccentric. While dilatation means there is an increase in muscle mass so that ratio between wall thickness and ventricular cavity size remains relatively constant.[5,6] Like physical evaluation, the ECG is influenced by morphological changes induced by obesity, such as (1) displacement of the heart by an elevated diaphragm in the supine position, (2) increased cardiac workload with associated cardiac hypertrophy, (3) increased distance between the heart and the recording electrodes induced by the accumulation of adipose tissue in the subcutaneous tissue of the chest wall (and possibly increased epicardial fat), and (4) the potential associated chronic lung disease secondary to the sleep apnea/ hypoventilation syndrome.[7-9] The ECG is a useful but imperfect tool for detecting LVH. The utility of the ECG related to its being relatively inexpensive and widely available, but the sensitivity or specificity depending upon which of the many proposed sets of criteria are applied. However, the ECG may be used in poor resource countries where echocardiography is unavailable or too expensive.[10-12]

Early detection and treatment of LVH in obese children may prevent cardiovascular risk later in adult. The aim of this study was to evaluate the prevalence of obesity in left ventricular hypertrophy using ECG criteria.

Methods

This cross-sectional study was conducted at five junior high schools in Manado between February to October 2009. Ethical approval was obtained from the Medical Ethics Committee, Medical School, Sam Ratulangi University. We included 10–15 year old children with obesity and employed healthy-normal weight children as control. We excluded children with clinical signs of cardiac dysfunction, congenital heart diseases, metabolic disorders and left bundle branch block (LBBB). Informed consent was obtained from their parents or caretakers.

Physical examination was following information including gender, age at the time of diagnosis, weight, height, calculated body mass index (BMI) and birth weight. The children were categorized obese if BMI was 95th percentile according to the WHO and Center for Disease Control and Prevention (CDC) 2000.[13-15]

A resting 12 lead ECG was obtained from each subject on admission according to standard procedure, then evaluated by author. We used Cardimax FX/2111 for electrocardiograhic examination. Every ECG data was examined by two observers. ECG criteria for LVH used standard pediatric criteria for LVH and Sokolow-Lyon criteria ((S in V1+ R in V5 or V6) > 35 mm).9 The standard pediatric criteria for LVH were based on one of the following ECG criteria, included high R wave in V6, changes in T wave in V5 and V6, R in V5 and V6 higher than maximum, S in V1 higher than maximum, R/S ratio in V1 less than minimum, deep Q wave in V5 and V6, and VAT left precordium higher than maximum.[8]

All analyses were performed using the statistic`program SPSS version 17. Data were presented as mean (SD). T test were used to assess the differences between the variables in patients with obese and control subjects. Differences were considered significant at a probability value of $P < 0.05$.

Results

Data from obese and non-obese children were reviewed from 3 junior high schools. Demographic data of obese children and healthy controls are summarized in **Table 1**. Weight, birth weight and body mass index were significantly smaller in control subjects.

There were 37 obese children and 37 healthy-normal weight children as control. Out of 37 obese subjects participating in the study, 20 were males and 17 were females. Most of the subjects were 11 to 12 years of age (11/37). Mean BMI for age were 32.70 (SD 2.135) in obese and 16.19 (SD 1.660) in control

subjects. Mean body weight in obese children was 73.46 (SD 8.881)kg compared to control 33.97 (SD 5.331)kg. Mean body weight at birth was 3537.8 (SD 608.3)g in obese children and 2989.2 (SD 283.4)g in control subjects (P<0.001).

Left ventricular hypertrophy were featured with ECG in 3 obese children and 1 child in control subjects (P 0.304) (**Table 2**). Based on Sokolow - Lyon criteria, in obese children, the lowest SV1 was 1.0 mm and the highest was 17.0 mm with mean SV1 8.72 mm. In obese children group, the lowest RV5 was 7.0 mm and the highest was 25.0 mm with mean RV5 14.9 mm, lowest RV6 was 6.0 mm and the highest was 21.25 mm with mean RV6 11.96 mm. Therefore the lowest SV1 + RV5 or RV6 was 9.75 mm and the highest was 37.5 mm with mean SV1 + RV5 or RV6 23.98 mm. **Table 3** shows that mean RV6 from ECG in obese children was 9.8446 (SD 3.5854) and mean RV6 from ECG in control children was 11.9662 (SD 3.2857) (P 0.005). Phi correlation failed to show significant correlation between obese and non-obese children (rø=0.120 and P=0.304).

Discussion

Our study found obesity is more frequent in children who were born with heavier birth weight. Mean body weight at birth was 3537.8 (SD 608.3) in obese children and 2989.2 (SD 283.4) in control subjects (P <0.001). These indicate that the heavier the birth weight, the more risk to become obese. These results are similar with those found by Danielzik[16], who studied obese children aged 5–7 years (P= 0.005). Maternal obesity and maternal diabetes were possibly factors responsible in this mechanisms.

One subject of control group had LVH based on ECG, but his blood pressure was above average normal limits. Hypertension is the main cause of LVH, it makes myocardium contraction in left ventricular strengthen to balance the increasing pressure. Finally, it becomes ventricular dilatation because of increased circulation resistance.[4,5] The hypertrophy muscle fibers become thicker and shorter so they lose their ability to relax, influencing cardiac output.[17,18] We suggest further examination on this child to make sure the cause of hypertrophy then perform early treatment.

Table 1. Anthropometrics data of obese children and control

Variabel	Control (n=37)		Obesity (n=37)		P*
	Mean	SD	Mean	SD	
Birth weight (g)	2989.2	283.4	3537.8	608.3	<0.001
Height (cm)	144.86	7.285	149.70	7.371	0.006
Weight (kg)	33.97	5.331	73.46	8.815	<0.001
BMI (kg/m^2)	18.19	1.660	32.07	2.135	<0.001

*T test mean in 2 pair group

Tabel 2. LVH featured in ECG ,hj

ECG Featured	Group		Total
	Control	Obese	
Normal	36	34	70
LVH	1	3	4
Total	37	37	74

X^2 = 1.057 db = 1 P = 0.307

Tabel 3. ECG feature using Sokolow-Lyon criteria.

	Control (n=37)		Obese (n=37)		P*
	Mean	SD	Mean	SD	
SV1 (mm)	8.0608	3.6488	8.7230	4.1218	0.234
RV5 (mm)	13.6284	4.9633	14.9730	4.6606	0.117
RV6 (mm)	9.8446	3.5854	11.9662	3.2857	0.005
SV1 + RV5 or RV6 (mm)	21.7432	7.4142	23.9865	6.4410	0.085

*T test mean in 2 pair group

In obese children, the proportion of LVH is higher than that in normal children, but cannot be statistically proven yet in this study.[17] In children, LVH only occurs if the process of obesity already chronic. T test showed significant different in RV6 (P= 0.005). This study showed that RV6 can be used to detect LVH in obese children, but needs further investigation. Increasing LVH is associated with increasing duration and height of complex QRS. Although the ECG has high specificity but it has low sensitivity. In this study we used Sokolow – Lyon criteria for ECG reading that has better sensitivity and specificity, and has international recommendation.[19,20] Study from Domingos,[21] showed that Sokolow – Lyon criteria has sensitivity 40% and specificity 100%.

In fact, the gold standard for measuring LVH was using echocardiography. Echocardiography, cannot be done as a comparison to ECG because the tools is not available in this study. Despite the limitations, ECG is a useful tool for detecting LVH, especially in poor resource countries where echocardiography is unavailable or too expensive.[22] In conclusion, there is no relation between obesity and left ventricular hypertrophy in obese children aged 10–15 years. Heavier birth weight is related to obesity in children.

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