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Echocardiographic Assessment of Epicardial Adipose Tissue in Obese Children and Its Relation to Clinical Parameters of the Metabolic Syndrome

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Background: The aim of the study was to evaluate cardiac function and epicardial adipose tissue (EAT) of obese children with echocardiography and to study the relationship of EAT to other echocardiographic findings and to clinical parameters of the metabolic syndrome (MS) in children. Patients and Methods: This study included 74 obese children and adolescents from the patient sample attending the obesity clinic of the National Research Center. Metabolic syndrome was determined according to the International Obesity Task Force (IOTF) 2007 criteria. 40 lean children were included in the study as a control group. All children were subjected to clinical assessment including standing height, body weight, Body Mass Index (BMI), waist circumference (WC), hip circumference (HC), systolic blood pressure (SBP), and diastolic blood pressure (DBP) plus an echocardiographic examination with measurement of EAT thickness and biochemical parameters (fasting glucose, insulin, HOMA index, total cholesterol, triglycerides, HDL-C, and LDL-C). Results: BMI, SDS BMI, WC, HC, and HOMA index were significantly higher in obese compared to lean children ($p = 0.001$). Left atrial (LA) diameter, septal posterior wall thickness, relative wall thickness, and left ventricular mass (LVM) were increased in the obese compared to the non-obese group while LV systolic and diastolic functions did not differ in obese versus lean children ($p > 0.05$). Patients had significantly thicker EAT compared to controls ($p = 0.01$). There was a significant correlation between EAT thickness in comparison to BMI, WC, HC, LA diameter, LVM, HOMA, triglycerides, and LDL-C. No significant difference in EAT thickness between obese patients with or without metabolic syndrome was found. Conclusion: Assessment of EAT thickness in routine echocardiographic examinations might be a feasible and reliable method for the evaluation of obesity and its related cardiovascular risks during childhood. There is no significant association between EAT thickness and metabolic syndrome in obese children. ***J Clin Basic Cardiol 2011; 14 (online): 7–11.***

Key words: *childhood obesity, epicardial adipose tissue, metabolic syndrome*

The prevalence of overweight and obesity in children is increasing worldwide at an alarming rate in both developing and developed countries [1]. The prevalence is approximately 16 % for children in the United States [2]. These dramatic changes make obesity a major public health issue [3].

It is well established that obesity is a strong risk factor for cardiovascular morbidity and mortality. Studies in adults using echocardiography, catheterization, and necropsy examinations have shown relations between morbid obesity, structural alterations of the heart, and systolic function [4]. However, the relation between obesity and cardiac function in children is not so well documented and conflicting results have been reported [5]. Visceral fat is a far better indicator of left ventricular mass (LVM) than the Body Mass Index (BMI). Epicardial adipose tissue (EAT), a part of visceral fat, is situated on the free walls of the right ventricle, left ventricular apex, atrium, and coronary vessels [6]. Epicardial adipose tissue, an index of cardiac visceral adiposity, could have a functional and mechanical role in obesity-related LV abnormalities [7]. Epicardial adipose tissue might function as a lipid storage depot, as an endocrine organ secreting hormones, and as an inflammatory tissue secreting cytokines and chemokines, so it could play a role in the pathogenesis of cardiomyopathy and coronary atherosclerosis [8].

In addition, obesity is associated with a heterogeneity of metabolic abnormalities, eg, dyslipidemia, insulin resistance, hyperglycemia, and hypertension [9, 10], that may provide a plausible biologic link between obesity and the increased risk of cardiovascular morbidity and mortality. The clustering of these risk factors for cardiovascular disease is referred to as metabolic syndrome [11]. Epicardial adipose tissue is closely related to insulin resistance and dyslipidemia and producing a much greater risk for metabolic syndrome and cardiovascular disease [12].

The aim of this study was to evaluate cardiac function and EAT of obese children with echocardiography and to study the relationship of EAT with other echocardiographic findings and clinical parameters of the metabolic syndrome in children.

Patients and Methods

Study Population

This study included 74 obese children and adolescents (29 males and 45 females) from the patient population attending the obesity clinic of the National Research Center (NRC). Exclusion criteria were a significant concomitant illness, medication known to affect cardiac function, antihypertensive drugs, hormone replacement therapy, and obvious clinical signs of cardiac disease. The metabolic syndrome was determined according to the International Obesity Task Force (IOTF) 2007 criteria [13]. 40 age- and sex-matched lean children (16 males and 24 females), who were relatives of the medical staff of the NRC, were included in the study as a control group. Parental consent for all children was obtained using the form approved by the Ethics Committee of the National Research Center. All children included in the study were subjected to the assessments listed below.

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Clinical Assessment

Standing height (cm) was measured to the nearest 0.1 cm with a Harpenden fixed stadiometer. Body weight (kg) was measured on a SECA balance scale to the nearest 0.1 kg with children dressed in a light T-shirt and shorts. BMI was calculated using the formula body weight (kg)/body height^2 (m) [14] and BMI values were converted to SD scores (BMI-SDS) adjusted for age and sex using the standard growth curves for Egyptian children and adolescents [15]. Waist circumference (WC) was measured midpoint between the lowest rib and the iliac crest, whereas hip circumference (HC) was taken at the widest part over the trochanters. Systolic (SBP) and diastolic blood pressures (DBP) were measured with a standard clinical sphygmomanometer using a stethoscope placed over the brachial artery pulse. The cuff used was appropriate for the size of a child's upper right arm.

Echocardiographic Examination

Echocardiographic studies were performed with a Vivid3 Expert (Norway) using 3- and 7-MHz transducers. All subjects were studies without sedation while they were lying quietly in the supine position. Triplicate measurements of all variables were made off line by one observer who was blinded to the patient's clinical details.

Chamber dimensions and wall thicknesses were obtained by 2-dimensionally guided M mode according to the recommendations of the American Society of Echocardiography [16]. The LV systolic functions (ejection fraction [EF] and fractional shortening [FS]) were evaluated using the following M mode echocardiographic parameters: interventricular septum systolic and diastolic thicknesses (IVSs and IVSd), LV end-systolic and end-diastolic dimensions (LVEsD and LVEdD), and LV posterior wall systolic and diastolic thicknesses (LVPWs and LVPWd). LV mass and relative posterior wall thickness (RWT) were calculated according to the formulae of Devereux et al [17]. LV diastolic functions were evaluated by measuring peak early diastolic filling velocity (E), peak late diastolic filling velocity (A), E/A ratio, and deceleration time (DT) [18].

Epicardial adipose tissue was measured using 2-dimensional echocardiography as an echo-free space over the pericardial layers and its thickness was measured on the free wall of the right ventricle, perpendicular to the wall from parasternal long and short axis views at end-diastole. The measurement was done for 3 cardiac cycles.

Biochemical Parameters

- Fasting blood samples were drawn from participants after 12-h fasting.
- Fasting serum glucose was measured enzymatically with an automated analyzer (Olympus Au 400).
- Fasting serum insulin was measured using the chemiluminescent technique (DPC kit on Immulite 1000). The estimate of insulin resistance was calculated by a homeostasis model assessment (HOMA) index.
- Total cholesterol and triglycerides were measured with enzymatic colorimetric tests on Olympus Au 400.
- HDL-C was measured using the precipitation method with phosphotungstic acid (centronic GmbH).
- LDL-C was calculated.

Statistical Methods

The Statistical Package for Social Science (SPSS) program version 9 was used for data analysis. All data were expressed as mean ± SD. Student's t-test for quantitative independent variables was used for analysis of difference between 2 groups. Pearson's bivariate correlation was used. Analysis of 2 independent variables was done using a non-parametric test (Mann Whitney U-test). In all tests, p-value was considered significant if < 0.05.

Table 1. Clinical characteristics of obese and non-obese children

Characteristic	Obese	Non-obese	p
n	74	40	–
Age (yr)	11.28 ± 3.71	11.45 ± 3.44	0.81
Body Mass Index (kg/m^2)	29.68 ± 5.76	18.83 ± 4.34	0.001
SDS BMI	2.17 ± 1.35	0.87 ± 0.53	0.001
Systolic blood pressure (mmHg)	104.64 ± 11.78	105.0 ± 8.51	0.88
Diastolic blood pressure (mmHg)	68.35 ± 9.29	69.67 ± 6.01	0.48
Waist circumference (cm)	88.28 ± 14.12	66.36 ± 15.34	0.001
Hip circumference (cm)	100.55 ± 16.7	80.03 ± 16.85	0.001
Waist circumference/ hip circumference	0.871 ± 0.069	0.825 ± 0.076	0.006
Fasting glucose (mg/dl)	88.8 ± 12.06	79.8 ± 9.3	0.001
Fasting insulin (mclu/ml)	7.16 ± 3.83	4.02 ± 2.13	0.001
HOMA index	1.41 ± 0.76	0.93 ± 0.5	0.003
Total cholesterol (mg/dl)	170.3 ± 34.4	167.13 ± 20.04	0.64
Triglycerides (mg/dl)	106.59 ± 42.8	86.07 ± 18.78	0.01
HDL cholesterol (mg/dl)	41.61 ± 8.9	41.13 ± 6.12	0.79
LDL cholesterol (mg/dl)	110.83 ± 29.4	105.87 ± 17.5	0.39

p significant if < 0.05

Table 2. Comparison of echocardiographic findings between obese and non-obese children

Characteristic	Obese	Non-obese	p
n	74	40	–
LA diameter (mm)	25.34 ± 4.84	20.75 3.24	0.001
IVSd (mm)	8.88 ± 2.28	7.3 ± 0.98	0.001
LVEdD (mm)	44.0 ± 7.9	39.56 ± 5.16	0.002
LVPW d (mm)	9.0 ± 1.9	7.1 ± 1.16	0.001
IVSs (mm)	10.92 ± 2.57	9.46 ± 1.34	0.001
LVEsD (mm)	28.86 ± 5.78	24.73 ± 3.65	0.001
LVPWs (mm)	11.95 ± 3.13	9.69 ± 1.88	0.001
EF (%)	67.89 ± 2.38	66.89 ± 3.93	0.09
FS (%)	34.5 ± 4.4	35.96 ± 3.74	0.07
RWT (mm)	0.41 ± 0.1	0.36 ± 0.05	0.008
LVM (g)	122.55 ± 55.38	77.34 ± 22.04	0.001
EAT thickness (mm)	7.17 ± 2.72	2.04 ± 0.63	0.001
Mitral E wave (m/s)	0.94 ± 0.17	0.9 ± 0.12	0.1
Mitral A wave (m/s)	0.58 ± 0.12	0.61 ± 0.16	0.4
Mitral E/A	1.63 ± 0.31	1.63 ± 0.27	0.9
Deceleration time (ms)	187.82 ± 43.29	188.58 ± 38.39	0.9

EAT: epicardial adipose tissue; EF: ejection fraction; FS: fractional shortening; IVSd: interventricular septum diastolic thickness; IVSs: interventricular septum systolic thickness; LA: left atrial; LVEDs: left ventricular end diastolic dimension; LVEsD: left ventricular end systolic dimension; LVM: left ventricular mass; LVPWd: left ventricular posterior wall diastolic thickness; LVPWs: left ventricular posterior wall systolic thickness; RWT: relative wall thickness. p significant if < 0.05.

Results

The comparison results of the baseline characteristics of obese and non-obese children are reported in Table 1. BMI, SDS BMI, WC, and HC were significantly higher in obese compared to non-obese children ($p = 0.001$), while age,

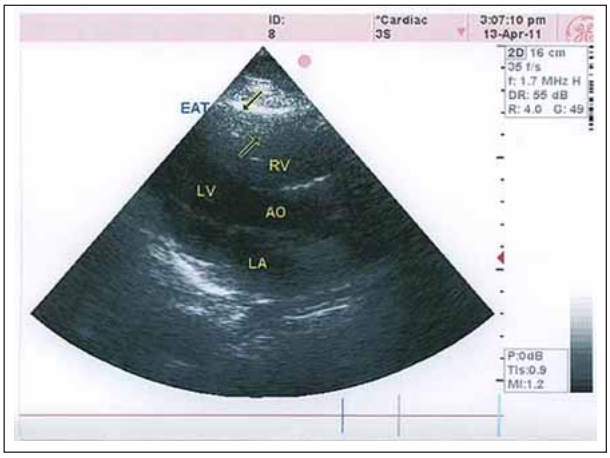

Figure 1. Transthoracic echocardiography showing a large area of epicardial adipose tissue (EAT) on the free wall of the right ventricle (arrows).

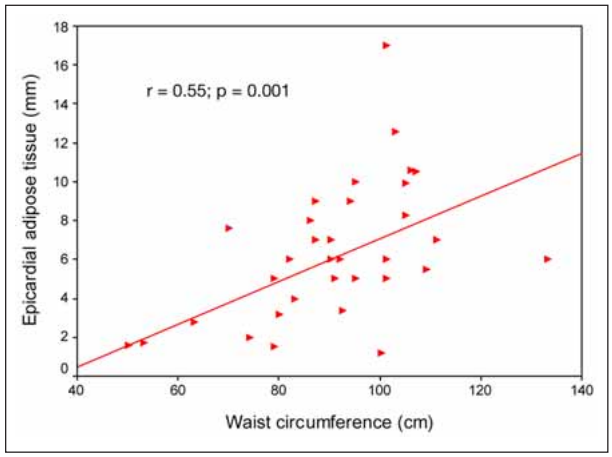

Figure 2. Relation of epicardial adipose tissue thickness to waist circumference.

Table 3. Correlation between epicardial adipose tissue (EAT) and other findings in obese children.

Variable	EAT thickness	
	r	p
BMI (kg/m^2)	0.77	0.001
WC (cm)	0.55	0.001
HC (cm)	0.62	0.001
LA diameter (mm)	0.66	0.001
RWT (mm)	0.22	0.08
LVM (g)	0.43	0.001
Mitral E/A	0.03	0.77
Fasting glucose (mg/dl)	0.01	0.9
Fasting insulin (mclu/ml)	0.28	0.05
HOMA index	0.34	0.02
TC (mg/dl)	0.26	0.06
Triglycerides (mg/dl)	0.32	0.02
HDL cholesterol (mg/dl)	0.11	0.4
LDL cholesterol (mg/dl)	0.29	0.04

BMI: Body Mass Index; WC: waist circumference; HC: hip circumference; LA: left atrial; RWT: relative wall thickness; LVM: left ventricular mass; TC: total cholesterol
P significant if < 0.05.

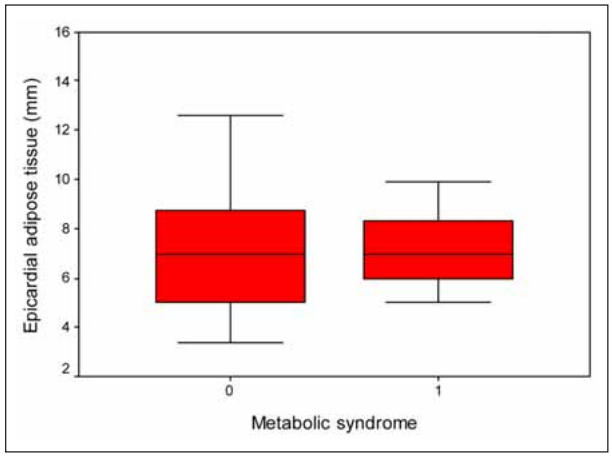

Figure 3. Epicardial adipose tissue thickness in obese children with (1) and without (0) metabolic syndrome (p > 0.05).

SBP, and DBP were similar with no significant difference (p > 0.05). Also, obese children had significantly higher fasting blood glucose, fasting serum insulin levels, HOMA, and triglycerides compared to lean children (p = 0.001), while no significant difference between the obese and non-obese groups regarding total cholesterol, LDL-C, and HDL-C levels was found (p > 0.05).

Table 2 shows the echocardiographic findings in obese and non-obese children. Left atrial diameter, septal, posterior wall thickness, RWT, LVEsD, LVEdD, and LVM of obese children were significantly increased compared to the non-obese control group (p = 0.001), while EF and FS of the left ventricle did not differ significantly in obese versus lean children (p > 0.05). Obese children had a significantly thicker EAT compared to the control group (p = 0.001; Figure 1).

Table 2 also summarizes the diastolic function of the left ventricle (mitral E wave, A wave, E/A ratio, and deceleration time) for obese and non-obese children. Doppler measurement did not differ significantly between the groups (p > 0.05).

A significant correlation was revealed between EAT thickness in comparison to BMI, WC, HC, LA diameter, LVM, HOMA, triglycerides, and LDL-C. However, EAT thickness did not correlate with RWT and mitral E/A, fasting glucose, fasting insulin, total cholesterol, and HDL-C (Table 3).

A significant correlation between EAT and WC (p = 0.001; r = 0.55) suggests that EAT is a good indicator of visceral fat (Figure 2).

Nine obese children fulfilled the criteria for the MS. We did not find a significant difference in EAT thickness between obese patients with or without metabolic syndrome (p = 0.75; Figure 3).

Discussion

Obesity is an important risk factor for atherosclerotic cardiovascular disease; it has been shown that obesity is associated with the development of early myocardial and coronary artery changes in children. Risk factor control in young people might retard the progression of atherosclerosis [19]. Therefore, it is very important to diagnose obesity and to examine its complications during childhood.

In this study, there was a significant increase in the LVM, RWT, septal and posterior wall thicknesses in obese compared to lean children (p = 0.001).

Several studies [6, 20–22] have reported the effects of obesity on echocardigraphic findings in children. The authors found that obesity was associated with a higher prevalence of LV hypertrophy and increased LVM in children. In addition to the echocardiographic studies, in a study using cardiovascular magnetic resonance Fribery et al [23] demonstrated increased LVM in obese adolescents and determined that the increase in LVM correlated mainly with BMI.

Also, Ippisch et al [24] found that elevated LVM, LV hypertrophy, and cardiac workload improved significantly following surgically induced weight loss in morbidly obese adolescents. They suggested that weight loss interventions during adolescence might be more likely to change cardiac parameters than similar interventions in adults who have been exposed to the long-term effects of obesity.

In the current study, assessment of systolic function has shown normal results and no patient presented with subclinical systolic dysfunction. Similarly, most echocardiographic studies that assessed systolic function in obese subjects showed normal results [25–27]. Chinali et al [28] and Pascual et al [29] reported that a reduction of indices of systolic function was only found in patients with a considerable degree of obesity, suggesting that left ventricular function is affected late in the course of obesity.

In the present study, the patients' diastolic functions were normal and not significantly different from those of the lean children. The Doppler method of measuring indices of left ventricular filling has been shown to be of great value in assessing diastolic function. However, when volume overload is present, as it is in obesity, normal values may result as the increase in left atrial pressure caused by intravascular volume and can mask alterations observed in the early phases of abnormal diastolic relaxation [1].

Few studies [20, 22, 30–32] have specifically investigated the impact of obesity on diastolic function in children. In contrast to most results, Harada et al [33] found altered transmitral and pulmonary venous velocities in 21 obese children using pulse wave Doppler, suggesting a reduction in early diastolic filling. Recently, tissue Doppler echocardiography (TDE) has been used to define regional myocardial dysfunction more accurately; the data obtained by this technique can be an early sign of future diastolic dysfunction [34]. Mehta et al [35] used TDE to measure the diastolic function of obese children who had normal conventional echocardiographic examinations and compared the results with those of normal children. They found that TDE revealed impaired diastolic function in obese subjects. Other studies using TDE to assess the effects of childhood obesity on diastolic function have reported similar findings [20, 22, 36, 37].

The relation between obesity and diastolic filling is not well understood. Although the association of myocardial fatty infiltration with obesity is well recognized, it is not a prominent finding at autopsy. However, altered LV relaxation could be related to early alterations in the myocardial collagen-to-muscle ratio that are secondary to insulin resistance and commonly seen in obese patients [8].

Our study found an increased LA diameter in obese children. Levent et al [30] and Yu et al [37] reported the same finding in their studies.

Possible causal factors for LA enlargement include increased preload, increased LV mass, and LV diastolic dysfunction. At present, it is not clear whether LA dilatation in obese children is an independent risk factor of cardiovascular disease, as it is in adults [6].

Our patients had significantly thicker EAT compared to lean children (7.17 ± 2.72 vs 2.04 ± 0.63; p = 0.001); in agreement with our results, Ozdmir et al [6] found that mean EAT thickness was 6.99 ± 1.45 vs 1.53 ± 0.26 (p = 0.001).

A study by Djaberi et al [38] recently demonstrated a significant relation between EAT thickness and the presence of coronary atherosclerosis.

Until now, MRI has been accepted as the gold standard for measuring EAT thickness. In 2003, Iacobellis et al [39] first reported the development of echocardiographic measurement of EAT. Their study suggests that echocardiographic EAT might be a simple and practical measure of this tissue in clinical practice and research. Epicardial and intraabdominal fat both originate from brown fat tissue during embryogenesis and subsequently differentiate into white adipose tissue [19]. It has been demonstrated that EAT has endocrine and inflammatory activities, as do other adipose tissue depots [8].

In this study, there was a significant correlation between EAT with LVM and LA. This result is similar to Ozdemir et al [6], who demonstrated that echocardigraphic measurements of EAT were significantly correlated with LVM and LA enlargement but did not find any correlation between EAT and diastolic filling in obese children and reported that echocardiographic assessment of EAT could be an easy method to identify obese children at high cardiovascular risk.

In the current study, there was a significant correlation between EAT and WC. This is in agreement with Ozdemir et al [6] and Iacobellis et al [39], who found that WC is widely accepted as a good predictor of intraabdominal fat mass and the strongest determinant of EAT thickness.

Our patients showed a significant correlation of EAT with insulin resistance. Metabolic syndrome is a cluster of risk factors associated with an increased risk of life-threatening metabolic and cardiovascular complications of obesity [12]. Ahn et al [40] showed that average epicardial fat thickness was higher in patients with unstable ischemic heart disease. This finding can be due to the fact that epicardial fat can be a source of inflammation which might increase the risk of cardiac ischemia. Mazurek et al [41] compared epicardial fat and subcutaneous fat from patients undergoing bypass surgery and found that epicardial fat produces numerous pro-inflammatory cytokines including IL-16, IL-6, soluble IL-6 receptor, and TNF-α. The levels of these cytokines were much higher in epicardial fat than in subcutaneous fat and the authors therefore suggested that TNF-α increases lipolysis and by releasing free fatty acids induces insulin resistance of the peripheral tissues.

In our study, there was no significant difference in EAT thickness between obese children with and without metabolic syndrome ($p > 0.05$). There was a comparable study in children by Mazur et al [12] who found the same results, but in adults Okyay et al [42] showed a close relationship between EAT thickness and MS. Ahn et al [40] demonstrated that patients with MS had thicker EAT than those without. Thickness increased with an rising number of components of MS. In another study [43], adults with insulin resistance had higher EAT thickness independent of the BMI. This discrepancy between the results obtained in adults and in children may be due to the difference in metabolic activity of EAT in younger subjects. Also, the time of exposure to obesity, which is shorter in children compared to adults, may not be long enough to promote the process of chronic inflammation. Therefore, the correlation between EAT, insulin resistance, and metabolic syndrome is relatively low [12].

Our study's limitation is the lack of pubertal status assessment in all children examined as a number of metabolic and endocrine changes occurs in that particular phase of growth. The sample size is also too small to study data separately for each gender.

Conclusion

Assessment of EAT thickness in routine echocardiographic examinations might be a feasible and reliable method for the evaluation of obesity and its related cardiovascular risks during childhood.

Although there is a significant correlation between EAT thickness with BMI, WC, HOMA, triglycerides, and LDL-C, there is lack of relationship of EAT thickness with MS in obese children.

Longitudinal studies on the role of EAT in the stimulation of different metabolic parameters in adolescents and increase in the risk of coronary artery disease in adults may unravel the mechanisms which promote the development of these common obesity complications.

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