

Childhood Obesity

Impact on Cardiac Geometry and Function



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ABSTRACT

OBJECTIVES The aim of our study was to assess geometric and functional changes of the heart in obese compared with nonobese children and adolescents.

BACKGROUND Obesity in children and adolescents has increased over the past decades and is considered a strong risk factor for future cardiovascular morbidity and mortality. Obesity has been associated with myocardial structural alterations that may influence cardiac mechanics.

METHODS We prospectively recruited 61 obese (13.5 ± 2.7 years of age, 46% male sex, SD score body mass index, 2.52 ± 0.60) and 40 nonobese (14.1 ± 2.8 years of age, 50% male sex, SD score body mass index, -0.33 ± 0.83) consecutive, nonselected Caucasian children and adolescents. A standardized 2-dimensional (2D) echocardiography and 2D speckle-tracking analysis was performed in all children. Furthermore, blood chemistry including lipid and glucose metabolism was assessed in all children.

RESULTS Compared with nonobese children, blood pressure, low-density lipoprotein cholesterol, and parameters of glucose metabolism were significantly increased in obese children, whereas high-density lipoprotein cholesterol was significantly lower. Compared with nonobese children, obese children were characterized by enlarged left- and right-sided cardiac chambers, thicker left ventricular walls, and, consequently, increased left ventricular mass. Despite a comparable left ventricular ejection fraction, decreased tissue Doppler-derived peak systolic velocity and regional basoseptal strain were found in obese children compared with nonobese children. Beyond that, 2D speckle tracking-derived longitudinal (-18.2 ± 2.0 vs. -20.5 ± 2.3 , $p < 0.001$) and circumferential (-17.0 ± 2.7 vs. -19.5 ± 2.9 , $p < 0.001$) strain of the left ventricle was reduced in obese children compared with nonobese children. Diastolic function was also impaired in obese compared with nonobese children. Both longitudinal strain and circumferential strain were independently associated with obesity.

CONCLUSIONS The results of this study demonstrate that childhood obesity is associated with significant changes in myocardial geometry and function, indicating an early onset of potentially unfavorable alterations in the myocardium. (J Am Coll Cardiol Img 2014;7:1198-205) © 2014 by the American College of Cardiology Foundation.

In the 1980s and 1990s, the prevalence of obesity increased dramatically in children and adolescents (1,2) and has plateaued in the past years at a disturbingly high level (3,4) to become a major health problem. Obesity has been associated with heart failure (5), left ventricular (LV) dilation, increased LV wall stress, and compensatory LV hypertrophy in adults (6). However, it has become clear

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that many of these abnormalities may already occur in childhood and adolescence (1,7-11).

Nevertheless, the association between isolated obesity and cardiovascular risk has been difficult to assess because obesity itself is highly associated with cardiovascular risk factors such as arterial hypertension, impaired glucose tolerance, diabetes mellitus, and dyslipidemias. Hence, the results have been controversial with regard to the role of obesity per se (12-15).

For this reason, children are likely to be the ideal candidates for providing insight into the myocardial changes related to obesity because they are suggested to be free of other cardiovascular risk factors in different echocardiographic studies (7,8,10). However, numerous studies have shown that a high body mass index (BMI) or other measures of obesity in children and adolescents are associated with adverse levels of lipids/lipoproteins and blood pressure and are related to insulin resistance (16,17). The value of the aforementioned echocardiographic studies is limited due to the lack of metabolic data in the control group (7,8,10,18) and the sole use of tissue Doppler imaging (TDI) for LV deformation analysis (7).

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The aim of our study, therefore, was to assess geometric and functional alterations in obese compared with nonobese children and adolescents when blood pressure, glucose metabolism, and lipid levels were accounted for in both groups.

METHODS

STUDY POPULATION. We prospectively recruited 61 overweight and obese and 40 nonobese consecutive Caucasian children and adolescents from the previously described Leipzig Atherobesity Childhood cohort (19). The children and adolescents were 8 to 21 years of age, free of known diseases, and not taking any medication. All subjects were characterized using anthropometric parameters, measures of glucose and insulin metabolism, and cardiovascular risk, as described recently (19,20). To determine insulin resistance, homeostatic model assessment insulin resistance (HOMA-IR) was calculated. The HOMA model (21) is a structural computer model of the glucose insulin feedback system in the homeostatic (overnight-fasted) state calculated from fasting plasma glucose and insulin concentrations.

The BMI was standardized to age- and sex-specific percentiles applying German reference data (22) and is given as the BMI standard deviation score (BMI-SDS), which is a measure of relative weight adjusted

for child age and sex. Cutoffs of 1.28 SD score (90th percentile) and 1.88 SD score (97th percentile) were defined as overweight and obese, respectively. Written informed consent was obtained from all parents and from children older than 12 years of age. The study was approved by the local ethics committee (registration no. 029-2006).

ECHOCARDIOGRAPHY. Every subject underwent a standardized 2-dimensional (2D) echocardiography examination using a commercial ultrasound system (Vivid 7, GE Health Medical, Milwaukee, Wisconsin). All images were recorded using harmonic imaging and stored digitally for analysis. 2D images were recorded using a temporal resolution of at least 60 frames/s; TDI frames were recorded at a rate of >100 frames/s. All data were read and analyzed by investigators who were blinded to conditions.

Chamber quantification and calculation of parameters of global LV systolic function followed current standards and, if applicable, were also indexed to body height raised to a power of 2.7 (23). Furthermore, *z*-scores of LV dimensions and LV mass were calculated on the basis of normal values of M-mode measurements (24,25). Of note, 7 obese children could not be included in the *z*-score analysis for interventricular septum diastolic (IVSd), posterior wall diastolic (PwD), left ventricular end-diastolic diameter (LVEDD), and left ventricular end-systolic diameter (LVESD) due to a body surface area >2.25 cm². For those children, the *z*-score is not validated according to national reference values (25). Deformation analysis of the left ventricle using TDI and 2D speckle-tracking echocardiography (2D-STE) was performed using EchoPac PC software version 113 (GE Health Medical) and according to a recent consensus statement (26). Analysis of diastolic function was also performed according to current guidelines (27).

For correlation analysis, endothelial function was evaluated by measuring the reactive hyperemia index using the EndoPat-Device (Itamar Medical Ltd., Caesarea, Israel), and intima media thickness of the left and right common carotid arteries were recorded.

STATISTICAL ANALYSIS. Statistical analysis was carried out using SPSS version 20 (IBM, Chicago, Illinois). Quantitative data are expressed as mean ± SD and qualitative data as absolute number (%). Comparisons between the groups were made with the Pearson chi-square test for categorical variables and 2-tailed Student *t* test for continuous variables. When continuous variables were not normally distributed

ABBREVIATIONS AND ACRONYMS

2D	= 2-dimensional
2D-STE	= 2-dimensional speckle-tracking echocardiography
3D	= 3-dimensional
BMI-SDS	= body mass index standard deviation score
CI	= confidence interval
HDL	= high-density lipoprotein
HOMA-IR	= homeostatic model assessment insulin resistance
ICC	= intraclass correlation
IVSd	= interventricular septal thickness at diastole
LA	= left atrial
LV	= left ventricular
LVEDD	= left ventricular end-diastolic diameter
PwD	= posterior wall at diastole
TDI	= tissue Doppler imaging

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