

Is Arterial Dysfunction Evident in Obese Pediatric Patients?

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Obesity is one of the most rapidly growing health problems in the world, not only for adults but also for children. The prevalence of childhood obesity in the United States has more than doubled (from 7% to 15.3%) between 1975 and 2000. Childhood obesity is associated with left ventricular hypertrophy, a major risk factor for coronary artery disease. However, a direct association between childhood obesity and vascular disease has not yet been demonstrated. The purpose of this study was to determine if arterial disease is present in the pediatric obese population and if so, explore its determinants. Twenty-one obese children (17 females, 4 males; 11.69 yr +/- 2.62; 10 African American, 11 Caucasian) with a body mass index (BMI) > 95th percentile recruited from a weight control clinic underwent echocardiography and vascular ultrasound. Measurements included indices of cardiac function [ejection time, heart rate, shortening fraction, wall stress, velocity of circumferential fiber shortening (VCF), and contractility (difference between measured and predicted VCF)] and cardiac geometry [left ventricular (LV) posterior wall thickness at end-systole (h_s) and end-diastole, interventricular septal thickness, LV end-diastolic dimension, left atrial diameter, and indexed LV mass]. Vascular structure was assessed by carotid artery intima medial thickness (CA IMT), which correlates well with the development of carotid artery atherosclerotic changes. Vascular function was evaluated by brachial artery (BA) reactivity (a marker of vascular endothelial function). Data were compared to age-matched control patients using the Student t test. Multiple regression analysis was performed to demonstrate predictors of vascular disease. The obese patients had significantly increased weight (76 kg +/- 20 vs. 44 kg +/- 17; $p < 0.0001$), BMI (32 kg/m² +/- 6 vs. 19 kg/m² +/- 4; $p < 0.0001$), and systolic blood pressure (115 mmHg +/- 15 vs. 104 mmHg +/- 11; $p = 0.0146$). The obese patients also demonstrated significant differences in cardiac geometry and function, including increased end-diastolic wall and septal thicknesses (0.875 cm +/- 0.228 vs. 0.650 cm +/- 0.108, $p = 0.0003$; 0.859 cm +/- 0.210 vs. 0.652 cm +/- 0.084; $p = 0.0003$), left atrial dimension (3.18 cm +/- 0.43 vs. 2.39 cm +/- 0.45; $p < 0.0001$), and indexed LV mass (35 +/- 16 vs. 23 +/- 5; $p < 0.0001$). Obese patients tended to have a higher percent change in BA reactivity [(BA diameter 60 sec post-deflation - BA diameter at rest) / BA diameter at rest], but this difference did not achieve statistical significance (0.05 +/- 0.09 vs. 0.02 +/- 0.09; $p = 0.2$). Although no differences were found in BA diameter, CA IMT was significantly increased in the obese patients (0.0455 mm +/- 0.0086 vs. 0.0376 mm +/- 0.0042; $p = 0.0009$). Multiple regression analysis determined that CA IMT correlated with African American race, increased weight, increased h_s , and increased heart rate. Obesity has previously been proven a significant factor in cardiac disease but this study shows that obesity is also an important factor in vascular disease. The vascular disease being seen in obese pediatric patients probably develops prior to, and therefore may be a causative agent in the development of LV hypertrophy. These vascular problems are directly related to race and weight and associated with increased myocardial thickness. Cardiac and vascular changes are being observed in obese children at an earlier age; this is an indication of the need for earlier intervention and treatment of obesity.