



Evaluation of Cardiac Functions in Obese Adolescents with Echocardiography

Obes Adolesanlarda Ventrikül Geometrisi ve Kardiyak Fonksiyonların Ekokardiyografi ile Değerlendirilmesi

Büşra Türk¹, Hakan Altın², Murat Tutuç¹

¹Department of Pediatrics, Bursa City Hospital, Bursa, Turkey

²Division of Pediatric Cardiology, Department of Pediatrics, Bursa City Hospital, Bursa, Turkey

ABSTRACT

Aim: Obesity is a serious condition associated with increased blood volume and left ventricular hypertrophy. While diastolic dysfunction is more prominent during childhood, systolic dysfunction is more commonly observed in adulthood. The aim of this study is to investigate the relationship between body mass index (BMI) increase and early structural changes in the heart, systolic/diastolic functions, and vascular intima in obese adolescents.

Material and Method: The study included 80 obese and 60 normal-weight adolescents aged between 12-18 years. The body mass index of all participants was calculated, and their systolic and diastolic blood pressures were measured. Lipid profile and insulin resistance assessments were conducted for the obese adolescents, and waist/hip ratio was calculated. M-mode echocardiography, pulsed Doppler, and tissue Doppler echocardiography were performed on all subjects to evaluate cardiac functions.

Results: The results showed that systolic and diastolic blood pressure were higher in obese adolescents compared to the control group. Left ventricular mass index (LVMI), epicardial adipose tissue (EAT), and carotid intima-media thickness (CIMT) were also found to be higher in the obese group. Additionally, a significant decrease was detected in markers of diastolic dysfunction. No systolic dysfunction was observed; however, the left ventricular Tei index (LVTEI), which reflects both systolic and diastolic dysfunction, was significantly higher in the obese group.

Conclusion: This study demonstrates that cardiac dysfunction can be detected in obese adolescents before symptoms appear, providing an opportunity for early intervention.

Keywords: Obesity, adolescent, echocardiography diastolic dysfunction

ÖZ

Amaç: Obezite, artmış kan hacmi ve sol ventrikül hipertrofisi ile ilişkili ciddi bir durum olup, çocukluk çağında özellikle diyastolik disfonksiyon belirgin iken, yetişkinlikte daha yaygın olarak sistolik disfonksiyon gözlenir. Bu çalışmanın amacı, obes adolesanlarda vücut kitle indeksi artışı ile kalpteki erken yapısal değişiklikler, sistolik/diyastolik fonksiyonlar ve damar intiması arasındaki ilişkiyi incelemektir.

Gereç ve Yöntem: Çalışmaya 12-18 yaş arasındaki 80 obes ve 60 normal kilolu sağlıklı adolesan dahil edilmiştir. Tüm katılımcıların vücut kitle indeksi hesaplanmış ve sistolik ile diyastolik kan basınçları ölçülmüştür. Obes adolesanların lipid profili ve insülin direnci değerlendirilmesi yapılmış olup bel/kalça oranı hesaplanmıştır. Tüm katılımcılara kardiyak fonksiyonları değerlendirmek amacıyla M-mode ekokardiyografi, pulsed Doppler ve doku Doppler ekokardiyografi değerlendirilmesi yapılmıştır.

Bulgular: Sonuçlar, obes adolesanlarda sistolik ve diyastolik kan basıncının kontrol grubuna göre daha yüksek olduğunu göstermiştir. Sol ventrikül kitle indeksi, epikardiyal yağ dokusu ve karotis intima-media kalınlığı da obes grupta daha yüksek bulunmuştur. Ayrıca diyastolik disfonksiyon belirteçlerinde anlamlı bir azalma tespit edilmiştir. Sistolik disfonksiyon gözlenmemiştir; ancak hem sistolik hem diyastolik disfonksiyonu yansıtan sol ventrikül Tei indeksi obes grupta anlamlı derecede yüksek bulunmuştur.

Sonuç: Bu çalışma, obes adolesanlarda semptomlar ortaya çıkmadan önce kardiyak disfonksiyonun tespit edilebileceğini göstererek erken müdahale için bir fırsat sunmaktadır.

Anahtar Kelimeler: Obezite, adolesan, ekokardiyografi, diyastolik disfonksiyon

Corresponding Author: Hakan Altın

Address: Bursa City Hospital, Department of Pediatrics, Division of Pediatric Cardiology; Bursa, Turkey

E-mail: dr.hakanaltin@hotmail.com

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INTRODUCTION

Obesity is a serious public health issue, attracting attention with its increasing prevalence in childhood and adolescence in recent years. According to data from the World Health Organization (WHO), in 2022, 8% of children and adolescents (160 million young people) were living with obesity (1). The World Obesity Federation predicts that by 2025, 206 million individuals aged 5-19, and by 2030, 254 million children and adolescents will be living with obesity (2). Obesity has significant effects on the cardiovascular system, leading to various complications later in life. WHO defines obesity as excessive fat accumulation that poses a threat to health (3). The adverse effects of obesity on the metabolic and cardiovascular systems can lead to severe long-term health issues. Cardiovascular complications associated with obesity include hypertension, dyslipidemia, and insulin resistance, which are indicators of metabolic syndrome (4). Individuals who are obese in childhood have an increased risk of developing cardiovascular diseases in adulthood (5). Additionally, obesity can impair myocardial functions, leading to severe complications like heart failure. With an increase in body fat tissue, obesity causes significant changes in the cardiovascular system. Increased fat tissue raises circulating blood volume, resulting in eccentric hypertrophy of the left ventricle. Obesity is regarded as a condition of chronic volume overload, as the heart must pump blood through the extensive and relatively low-resistance adipose tissue reservoir (6,7). While childhood obesity often leads to diastolic dysfunction, systolic dysfunction is seen in adulthood. Therefore, identifying subclinical cardiac changes before systolic functions deteriorate is essential. An increase in epicardial adipose tissue (EAT) in obese adolescents may also play a role in the pathogenesis of atherosclerosis and cardiomyopathy (8). Additionally, ultrasonographic measurement of carotid intima-media thickness (CIMT) is considered a valid indicator for assessing atherosclerotic changes at the subclinical stage, providing an assessment of cardiovascular risk (9,10,11). The aim of this study was to determine the differences in left ventricular muscle index, systolic and diastolic functions, and carotid intima-media thickness between obese adolescents and those with normal body mass index. If abnormalities in the left ventricular diastolic and systolic functions can be detected early, it may offer an opportunity to intervene before heart failure develops.

MATERIAL AND METHOD

Between February 2023 and December 2023, 80 children aged 12-18, diagnosed with exogenous obesity, were referred to the pediatric cardiology outpatient clinic and evaluated in terms of cardiology. These children, who were identified based on their medical history, physical examination, and test results, formed the study group. Additionally, 60 healthy children aged 12-18, who attended the clinic for routine control, general screening

before school or dorm registration, and who had no active complaints or chronic illnesses, were included in the control group. Body mass index (BMI) percentile curves determined for Turkish children according to age and gender were used. BMI below the 85th percentiles was defined as normal weight, and a BMI above the 95th percentile was defined as obesity. Exclusion criteria included acquired or congenital heart disease (e.g., ASD, VSD, PDA, mitral/aortic insufficiency or stenosis), chronic systemic illness, consumption of nutrients or medications that could lead to hypertrophy of cardiac muscles (e.g., corticosteroids, addictive stimulants, testosterone, growth hormone, high-protein supplements, etc.), hypertension due to non-obesity-related causes, endocrine/genetic diseases, and lack of parental consent. Approval for the study was obtained from our hospital's Clinical Research Ethics Committee (Date:01/02/2023, Decision no:2023-3/3).

Anthropometric and Biochemical Measurements

To determine anthropometric parameters, height, weight, hip, and waist circumference measurements were taken from the children. BMI was calculated by dividing body weight in kilograms by the square of height in meters (12). Hip circumference was measured at the broadest point of the hips, at the level of the greater trochanter, with the legs close together in centimeters (cm). Waist circumference was measured while the child was standing, using a non-flexible tape measure from the midpoint between the arcus costae and spina iliaca anterior superior in cm. Patients were rested for at least 20 minutes before blood pressure measurement. Blood pressure measurements for the entire group were taken three times by the same researcher using a suitable cuff size for the patient's right arm with a sphygmomanometer, and the average of these measurements was calculated for evaluation. In the obese group, laboratory test results (fasting glucose, fasting insulin, lipid profile, aspartate amino transferase (AST), alanine amino transferase (ALT), thyroid-stimulating hormone (TSH), thyroxine (T4), triglycerides, total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), hemoglobin A1c (HbA1c)) from the last three months were obtained from the Pediatric Endocrinology Clinic data. No laboratory tests were conducted in the control group. Homeostasis Model Assessment Insulin Resistance (HOMA-IR) was calculated using the formula: $\text{FBG (mg/dl)} \times \text{fasting insulin } (\mu\text{U/ml}) / 405$. HOMA-IR value is the most widely used and reliable method to determine insulin resistance in children, and HOMA-IR ≥ 3.16 in adolescents is considered insulin resistance (13).

Echocardiography

Echocardiography was performed by the same pediatric cardiologist using the Vivid S60N (GE Vingmed Ultrasound AS Strandpromenaden 45, 3191 Horten, NORWAY) device and a 3ScRs probe. All measurements were taken

during three consecutive cardiac cycles and averaged. Standard 2D, M-mode, and Doppler measurements were performed according to the guidelines of the American Society of Echocardiography.

Two-Dimensional and M-Mode Echocardiography Measurements

Left ventricular M-mode measurements were obtained from a parasternal long-axis view. Interventricular septal end-diastolic dimension (IVSd), interventricular septal end-systolic dimension (IVSs), left ventricular end-diastolic dimension (LVEdD), left ventricular end-systolic dimension (LVEsD), left ventricular posterior wall end-diastolic dimension (LVPWd), left ventricular posterior wall end-systolic dimension (LVPWs), and left atrial dimension (LAd) were measured. Ejection fraction (EF) was calculated using the Simpson method. Left ventricular mass (LVM) was calculated using the Devereux formula: $0.8\{1.04[(LVIDd + IVSd + LVPWd)^3 - LVIDd^3]\} + 0.6$. Left ventricular mass index (LVMI) was calculated by dividing LVM in grams by body surface area (BSA). Epicardial adipose tissue (EAT) was defined as an echo-free space within the pericardial layers in two-dimensional echocardiography and was measured perpendicularly on the free wall of the right ventricle at the end of diastole from a parasternal long-axis view (14-16). Measurement of carotid intima media thickness (CIMT) of carotid bifurcation was performed by the pediatric cardiologist using the Vivid S60N, GE, Horten, Norway echocardiography device with linear probe. Measurements were taken with the patient lying supine, with the head slightly extended and turned to the side opposite the artery being evaluated.

Conventional Diastolic Parameters

Ventricular diastolic flow rates were determined by placing the sample volume probe at the tip of the tricuspid and mitral leaflets in an apical four-chamber view using pulsed Doppler. The peak early diastolic filling velocity (E), the peak late diastolic filling velocity (A), and the E/A ratio, indicating diastolic function status, were obtained.

Tissue Doppler Echocardiography

Tissue Doppler imaging was performed by placing the sample volume at the mitral annulus on the posterior wall of the left ventricle, the right ventricular anterior wall at the tricuspid annulus, and the basal part of the interventricular septum in apical four-chamber views. Peak systolic flow of the left ventricle (LVSm), peak early diastolic myocardial velocity (E'), and peak late diastolic myocardial velocity (A') were measured. The isovolumic contraction time (IVCT) was measured from the end of the A' wave to the beginning of the Sm wave, and the isovolumic relaxation time (IVRT) was measured from the end of the Sm wave to the beginning of the E' wave. The myocardial performance index (LVTEI) was calculated by dividing the sum of IVCT and IVRT by the ejection time (ET) (17,18).

Statistical Analysis

In this study, the conformity of continuous variables to a normal distribution was examined using the Shapiro-Wilk test. Continuous variables were expressed as mean and standard deviation values if they conformed to a normal distribution, and as median (minimum-maximum) values if they did not conform, while categorical variables were reported with frequency and the relevant percentage values. Comparisons between study groups for continuous variables were performed using the independent samples t-test if the data conformed to a normal distribution, and the Mann-Whitney U test if they did not. For categorical variables, comparisons between study groups were made using the Chi-square and Fisher's exact Chi-square tests. The relationships between continuous and discrete variables were examined by correlation analysis, and the relationships between variables were reported with the Spearman correlation coefficient. The analyses of the study were conducted using the SPSS (IBM Corp. Released 2015. IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp.) program, and a type I error rate of 5% was accepted for statistical comparisons. A two-tailed $p < 0.05$ was considered statistically significant.

RESULTS

There was no significant difference between the groups participating in the study in terms of age and gender ($p: 0.312$). However, in the obese group, systolic and diastolic blood pressure values were significantly higher than those in the control group ($p < 0.001$, $p < 0.001$). BMI was found to be significantly higher in the obese group ($p < 0.001$). A comparison of demographic data, anthropometric measurements, and blood pressure values of obese patients and children in the control group is presented in **Table 1**.

Table 1. Comparison of demographic data, anthropometric measurements, and blood pressure values of obese patients and children in the control group

	Control Group (n=60)	Obese Group (n=80)	p value
Age (Years)	14 (12-17)	15 (12-17)	0.312 ^a
Gender			0.921 ^b
Female	25 (41.70%)	34 (42.50%)	
Male	35 (58.30%)	46 (57.50%)	
Body Weight (kg)	61.62±10.03	101.93±13.70	<0.001 ^c
Height (cm)	1.67 (1.52-1.85)	1.68 (1.49-1.90)	0.300 ^a
BMI (kg/m ²)	21.78 (17.93-25.71)	34.57 (30.41-46.17)	<0.001 ^a
BMIstds	0.28 (-1.53-0.99)	2.99 (2.03-4.35)	<0.001 ^a
SBP (mmHg)	120 (106-135)	130.50 (111-164)	<0.001 ^a
SBPstds	0.84 (-0.10-2.05)	1.55 (-0.33-2.33)	<0.001 ^a
DBP (mmHg)	67 (50-82)	74 (55-110)	<0.001 ^a
DBPstds	0.14 (-1.28-1.48)	0.83 (-0.95-2.33)	<0.001 ^a

Data are reported as median (minimum-maximum), mean ± standard deviation, and n (%). a: Mann-Whitney U Test, b: Chi-square Test, c: Independent Samples t-test. BMI: Body Mass Index, BMIstds: Body Mass Index standard deviation score, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, DBPstds: Diastolic blood pressure standard deviation score.



In the obese group, the HOMA-IR value was also found to be high, indicating insulin resistance. The prevalence of elevated HOMA-IR levels was 94.10% in females and 82.60% in males. Among the obese adolescents in the study, 50% showed decreased HDL levels, and 10% had elevated triglyceride levels. The descriptive statistics of biochemical parameters for the obese patient group are shown in **Table 2**.

Table 2. Descriptive statistics of biochemical parameters in the obese patient group

Biochemical Parameter	Obese Group (n=80)	Reference Ranges (Min-Max)
Fasting Glucose (mg/dL)	90.38 (70-110)	70-100
AST (U/L)	21.14 (10-35)	0-32
ALT (U/L)	27.50 (15-40)	0-33
Fasting Insulin (uIU/ml)	28.07 (15-50)	2.6-24.9
HOMA-IR	6.31 (3-10)	0-3.16
HbA1C (%)	5.24 (4-6.50)	3.5-5.6
Cholesterol (mg/dL)	159.19 (130-190)	3-200
LDL (mg/dL)	87.05 (60-110)	0-130
HDL (mg/dL)	45.35 ± 10.45	45-65
Triglycerides (mg/dL)	130.81 (80-200)	0-200
Tsh (mIU/L)	2.15 (1-3.50)	1.13-5.34
Free T4 (ng/dL)	1.16 (0.90-1.50)	1-1.53

Data are reported as median (minimum-maximum) and mean ± standard deviation. Min: Minimum, Max: Maximum, AST: Aspartate amino transferase, ALT: Alanine amino transferase, HOMA-IR: Homeostasis Model Assessment Insulin Resistance, HbA1c: Hemoglobin A1c, LDL: Low-density lipoprotein, HDL: High density lipoprotein, Tsh: Thyroid-stimulating hormone, T4: Thyroxine.

In echocardiographic measurements, LVMI was found to be significantly higher in the obese group ($p < 0.001$). Additionally, EAT and CIMT measurements were significantly higher in the obese group compared to the control group ($p < 0.001$, $p < 0.001$). No differences were observed between the control and obese groups regarding EF and fractional shortening (FS) measurements, which reflect systolic functions ($p: 0.175$, $p: 0.106$). The results of conventional echocardiography, EAT, and CIMT measurements for the obese and control groups are presented in **Table 3**.

In the obese group, positive significant correlations were found between BMI SDS and CIMT, EAT, E/E', and LVTEI ($r_s = 0.61$ - $p < 0.001$, $r_s = 0.58$ - $p < 0.001$, $r_s = 0.52$ - $p < 0.001$, $r_s = 0.64$ - $p < 0.001$), while negative significant correlations were found between BMI SDS and E/A, E', and E'/A' ($r_s = -0.76$ - $p < 0.001$, $r_s = -0.63$ - $p < 0.001$, $r_s = -0.77$ - $p < 0.001$) (**Table 5**). A positive correlation was also found between waist/hip ratio and CIMT ($r_s = 0.30$; $p: 0.007$), EAT ($r_s = 0.26$; $p: 0.021$), LVMI ($r_s = 0.24$; $p: 0.029$), and LVTEI ($r_s = 0.26$; $p: 0.020$), while negative correlations were observed between waist/hip ratio and E/A ($r_s = -0.27$; $p: 0.017$) and E'/A' ($r_s = -0.26$; $p: 0.019$). **Table 4** shows the correlation analysis between waist circumference, waist/hip ratio, and cardiac measurements in the obese group.

Table 3. Comparison of conventional echocardiography, pulsed, and tissue Doppler echocardiography findings, and EAT and CIMT measurements in obese and control groups

	Control Group (n=60)	Obese Group (n=80)	pa
IVSd (mm)	6.25(4.50-8)	8.23(5-11)	<0.001
IVSs (mm)	11.25(8-14)	13.28(10-16)	<0.001
LVEdD (mm)	37.32(30-45)	41.98(35-50)	<0.001
LVEsD (mm)	23.07(18-28)	25.67(20-30)	<0.001
LVPWd (mm)	6.12(4.50-8)	8.03(5-11)	<0.001
LVPWs (mm)	10.87(8-13.50)	13.63(10-16)	<0.001
LVM (gr)	61.79(40-80)	105.88(70-140)	<0.001
LVMI (gr/m ²)	34.03(20-50)	50.86(30-65)	<0.001
EF (%)	70.42(65-75)	69.86(65-75)	0.175
CIMT(μ)	470.17(400-600)	690.94(500-900)	<0.001
EAT (mm)	3.03(2-4)	5(4-7)	<0.001
E (m/sn)	92(87-103)	96(81-111)	0.850
A (m/sn)	58(55-70)	64(51-86)	0.002
E/A	1.58(1.31-1.78)	1.47(1-1.96)	0.037
E'(cm/s)	14(11-16)	14(9-16)	0.295
A'(cm/s)	8.75 (6.88-10)	8.99(7.14-11.54)	0.034
E'/A'	1.60(1.30-1.80)	1.45(1.10-1.90)	0.002
E/E'	6.71(6-8)	6.89(5.80-9.10)	0.009
LVTEI	37(35-41)	39(36-45)	<0.001

Data are reported as median (minimum-maximum). a: Mann-Whitney U Test. IVSD: Interventricular septal end-diastolic dimension, IVSS: Interventricular septal end-systolic dimension, LVEdD: Left ventricular end-diastolic dimension, LVEsD: Left ventricular end-systolic dimension, LVPWd: Left ventricular posterior wall end-diastolic dimension, LVPWs: Left ventricular posterior wall end-systolic dimension, LVM: Left ventricular mass, LVMI: Left ventricular mass Index, EF: Ejection fraction, CIMT: Carotid intima media thickness, EAT: Epicardial adipose tissue, E: The peak early diastolic filling velocity, A: The peak late diastolic filling velocity, E/A: Peak velocity ratio, E': Peak early diastolic myocardial velocity, A': Peak late diastolic myocardial velocity, E'/A': Mean peak diastolic mitral annular velocity ratio, E/E': Early entry, early diastolic peak velocity ratio, LVTEI: Myocardial performance index.

Table 4. Correlation analysis of BMI SDS and waist/hip ratio with ventricular function parameters measured by echocardiography in the obese and control groups

n=80	BMISds		Waist/Hip Ratio	
	rs	p-value	rs	p-value
CIMT	0.61	<0.001	0.30	0.007
EAT	0.58	<0.001	0.26	0.021
LVMI	0.10	0.398	0.24	0.029
E/A	-0.76	<0.001	-0.27	0.017
E'/A'	-0.77	<0.001	-0.26	0.019
E/E'	0.52	<0.001	0.07	0.538
LVTEI	0.64	<0.001	0.26	0.020

rs: Spearman Correlation Coefficient, CIMT: Carotid intima media thickness, EAT: Epicardial adipose tissue, LVMI: Left ventricular mass Index, E/A: Peak velocity ratio, E'/A': Mean peak diastolic mitral annular velocity ratio, E/E': Early entry, early diastolic peak velocity ratio, LVTEI: Myocardial performance index.

Graphs of correlation analyses between BMI SDS and ventricular function parameters measured by echocardiography in obese and control groups are shown in **Figure 1**.

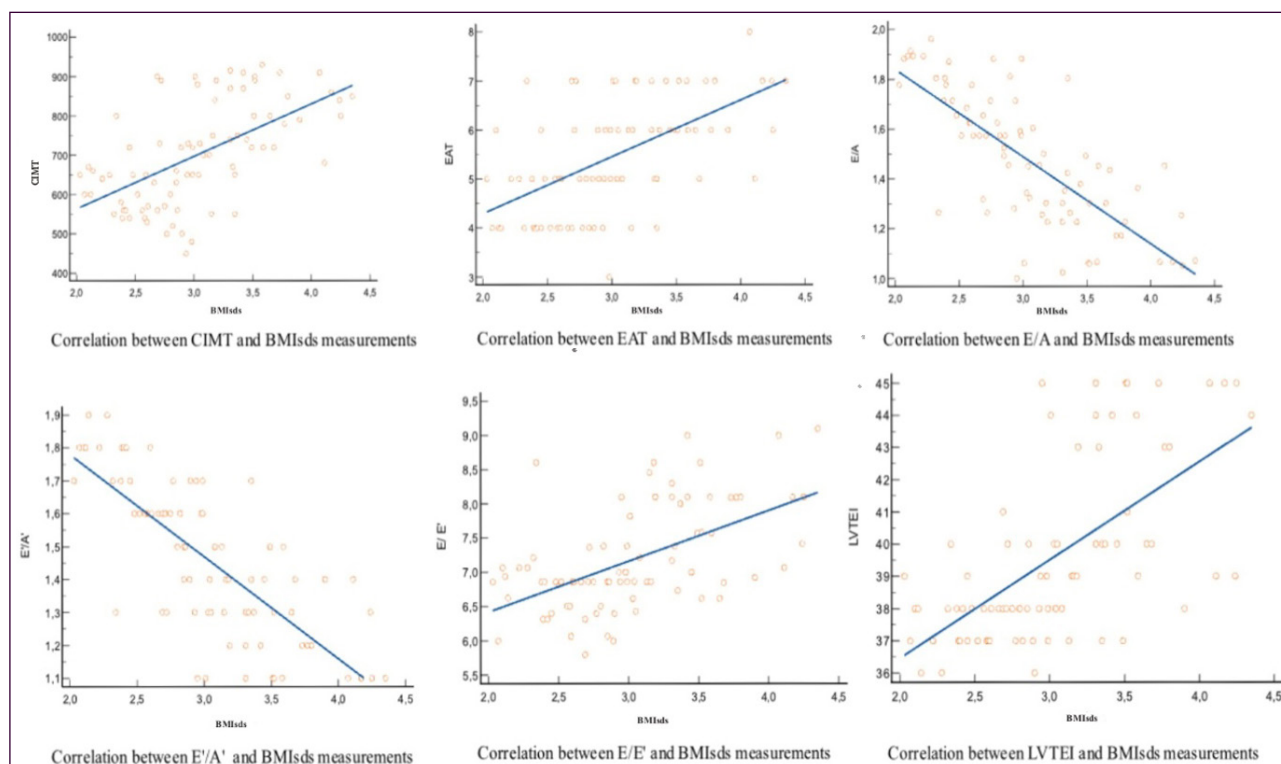


Figure 1.

DISCUSSION

The prevalence of obesity among children and adolescents is increasing worldwide (17). In a cohort study conducted by Lindberg et al., using the Swedish Childhood Obesity Treatment Register (BORIS), it was shown that individuals with childhood obesity had a threefold higher risk of all-cause mortality in early adulthood compared to a community-based comparison group (19). When evaluating the systolic and diastolic blood pressures of the obese and control groups in our study, the blood pressures in the obese group were found to be higher than in the control group ($p < 0.001$). Similarly, in the study by Skinner et al., a significant relationship between increased BMI and high systolic and diastolic blood pressure was demonstrated (20).

Waist circumference is one of the indicators of cardiovascular risk factors in children, and increased waist circumference in childhood obesity has been shown to be predictive of metabolic syndrome. Studies have demonstrated a correlation between waist circumference and BMI in children (21,22). In our study, a weak correlation was found between BMIstds and waist circumference in the obese group ($r_s = 0.24$; $p < 0.032$). Insulin resistance, a condition requiring higher insulin levels to maintain normal blood glucose and biological responses, is considered a common cause of cardiovascular and metabolic complications. Hyperinsulinemia and insulin resistance, which are also closely related to abdominal obesity, may induce

myocardial hypertrophy by growth-stimulating effect of insulin or increasing blood volume. Bjelakovic et al. showed a positive correlation between HOMA-IR and LVMI (23). The study by Skinner et al. demonstrated the association between obesity and high HOMA-IR values (20). Similarly, Thota et al. showed that obesity was significantly associated with higher fasting insulin and HOMA-IR values in adolescents (24). In our study, the median HOMA-IR value for obese adolescents was 6.31, with elevated HOMA-IR levels observed in 94.10% of obese females and 82.60% of obese males. Dyslipidemia associated with obesity can be characterized by hypertriglyceridemia, reduced HDL levels, and normal or slightly elevated LDL levels. Gómez-Díaz et al. found a 26% prevalence of hypertriglyceridemia and a 67% reduction in HDL levels among obese adolescents (25). In our study, 10% of patients in the obese group had hypertriglyceridemia, and 8% had slightly elevated LDL levels, while 50% had reduced HDL cholesterol levels.

The relationship between obesity in childhood and adolescence and the development of hemodynamic, thrombotic, and inflammatory disorders, such as atherosclerosis in later life, is well-supported in the literature (26). The first subclinical sign of atherosclerosis is an increase in arterial intima-media thickness. There is evidence that children with diabetes, hypercholesterolemia, and obesity have higher CIMT than healthy controls. In a study by Simsek et al., increased CIMT was observed in obese children compared to those of normal weight (27). Similarly, a study by Freedman et



al. demonstrated a relationship between CIMT and BMI (28). In our study, the median CIMT measurement in the obese group was 690.94 μ , while it was 470.17 μ in the control group ($p < 0.001$). A positive significant correlation was found between BMIs and CIMT ($r_s = 0.61$, $p < 0.001$).

Epicardial adipose tissue (EAT) accumulates around the heart and coronary vessels. EAT is located between the outer edge of the myocardium and the visceral pericardium, and it is distinct from pericardial adipose tissue located outside the visceral pericardium (8). EAT plays a role in diastolic dysfunction and may exert a direct compressive force on the myocardium. Coronary arteries are embedded within EAT, and increased EAT thickness is associated with impaired coronary blood flow reserve due to the perivascular compressive effect (29,30). In a study by Yang et al., a strong correlation between BMIs and increased EAT was demonstrated. Similarly, Alp et al. showed that EAT thickness was increased in obese individuals (8). In our study, even though the adolescents were obese, they were otherwise healthy. This controlled for the effects of additional comorbidities or chronic diseases. Our results also showed elevated EAT in the obese group ($p < 0.001$). In the obese group, the median EAT was 5 mm, while in the control group, it was 3.03 mm.

Obesity is associated with increased fat tissue, increased blood volume, and, consequently, an increase in preload returning to the heart. Left ventricular mass index (LVMI), which reflects the thickening of the left ventricle to compensate for the increased afterload caused by vascular changes affecting arterial stiffness and resistance, is a related parameter. In a study by Brady et al., LVMI was shown to be associated with BMI SDS independently of hypertension (31). In another study by Porcar-Almela et al., higher LVMI was observed in obese individuals compared to the control group (32). Similarly, Bartkowiak et al. found that obese children had higher LVMI (33). In our study, LVMI was also found to be elevated in obese individuals ($p < 0.001$). The median LVMI was 50.86 g/m^2 in obese adolescents, whereas it was 34.03 g/m^2 in healthy adolescents.

Obesity beginning in childhood should be considered a risk factor not only for cardiovascular diseases in adulthood but also as a factor in subclinical cardiac dysfunction during childhood. In a study by Alp et al., increased E/E', decreased E/A, and E'/A' ratios were reported, indicating reduced diastolic function in obese children (8). Similarly, Porcar-Almela et al. demonstrated increased E/E' and decreased E/A ratios in obese children (32). In the study by Mangner et al., elevated E/E' ratios were also shown in obese individuals (34). In our study, the E/A ratio, detected by standard Doppler as a parameter showing diastolic function, was lower in obese individuals compared to the control group ($p: 0.037$). The E'/A' ratio obtained through tissue Doppler was also significantly lower in obese individuals compared to normal-weight adolescents

($p: 0.002$). Additionally, the E/E' measurement was found to be increased in the obese group compared to the control group ($p: 0.009$). This indicates that the adverse effects of obesity are already present during adolescence.

In childhood obesity, systolic dysfunction can be predicted by EF, FS, and tissue Doppler parameters. In our study, while abnormal diastolic parameters were detected in obese adolescents in both conventional and tissue Doppler measurements, systolic functions were preserved in both groups. There was no difference between the groups in terms of fractional shortening (FS) (%) ($p: 0.106$), which is a variable parameter related to load. Similarly, there was no difference between the groups in terms of EF measurement ($p: 0.175$). Studies report that LVTEI, which reflects both systolic and diastolic function of the heart, is impaired in obese individuals. This may indicate that overt diastolic dysfunction is accompanied by subclinical systolic dysfunction in obesity (35,36,37). Similarly, in our study, LVTEI was found to be higher in the obese group compared to the control group and was correlated with BMI SDS ($r_s = 0.64$, $p < 0.001$).

Limitations of Our Study

Since no laboratory tests were performed on the control group, no comparisons between groups could be made in this area. Additionally, non-alcoholic steatohepatitis (NASH) and its effects on the cardiovascular system could not be studied due to the lack of ultrasound screening for liver steatosis. Due to the limited number of obese patients, we did not classify obesity within its own subcategories. Since this was a cross-sectional study, we were unable to detect possible cardiac changes in patients following multidisciplinary treatments by dietitians, endocrinologists, and nephrologists..

CONCLUSION

Our study identified diastolic dysfunction in asymptomatic obese children in terms of cardiovascular function. We observed that systolic dysfunction had not yet reached a level detectable by conventional methods. We believe these pathological findings may provide an opportunity for early intervention in the subclinical phase before clinical symptoms develop.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Bursa City Hospital Clinical Researches Ethics Committee (Date: 02.02.2023, Decision No: 2023-3/3).

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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