# Increased aortic pulse wave velocity in obese children

## Obez çocuklarda artmış aortik nabız dalga hızı

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**ABSTRACT**

***Objectives:*** Obesity may start in childhood and obese children are more likely to grow up to be obese adults. Atherosclerosis is one of the most important complications of obesity. Pulse wave velocity (PWV), a noninvasive measure of arterial stiffness, is accepted to be an indicator of subclinical atherosclerosis. The aim of the study was to determine PWV in obese children.

***Study design:*** The study included 30 obese (12 boys, 18 girls; mean age 13±2 years) and 30 lean children (13 boys, 17 girls; mean age 12.5±1.7 years). Weight and height were measured and obesity was defined as body mass index (BMI) of greater than the 95th percentile for age. All the subjects underwent echocardiographic evaluation and blood samples were obtained. Pulse-wave velocity was calculated using the following equation: PWV (m/sec) = height-based aortic length (cm) / (100 x transit time [sec]). The latter was measured as the difference in the time of onset of two flows at the diaphragm and the aortic valve.

***Results:*** Obese subjects had significantly higher blood pressure levels compared to the control group (p<0.001). The two groups were similar with respect to fasting glucose, hemoglobin, serum creatinine, and lipid levels. Among echocardiographic parameters, left ventricular end-diastolic dimension, interventricular septum thickness, posterior wall thickness, left ventricular mass index, left atrium dimension, and aortic root dimension were significantly increased in obese subjects compared to controls (p<0.01). Obese children had significantly higher PWV values than the controls (4.0±0.8 *vs.* 3.3±0.7 m/sec, p<0.001). A positive significant correlation was found between PWV and BMI (r=0.391, p=0.002).

***Conclusion:*** Our findings show that aortic PWV is increased in obese children, suggesting that obesity may cause subclinical atherosclerosis even at early ages.

**ÖZET**

***Amaç:*** Obezite çocukluk çağında başlayabilir ve obez çocukların büyüdüklerinde de obez erişkin olmaları olasılığı fazladır. Ateroskleroz bu hastalığın önemli komplikasyonlarından biridir. Aort sertliğinin invaziv olmayan bir ölçüm yöntemi olan nabız dalga hızı (NDH) subklinik aterosklerozun bir göstergesi olarak kabul edilmektedir. Bu çalışmada, obez çocuklarda NDH değerlendirildi.

***Çalışma planı:*** Çalışmaya 30 obez çocuk (12 erkek, 18 kız; ort. yaş 13±2) ve normal kilolu 30 çocuk (13 erkek, 17 kız; ort. yaş 12.5±1.7) alındı. Katılımcılarda ağırlık ve boy ölçüldü ve beden kütle indeksinin (BKİ) yaşa göre 95. persentilden büyük olması obezite olarak kabul edildi. Tüm çocuklar ekokardiyografi ile incelendi ve rutin kan tetkikleri için kan örnekleri alındı. Nabız dalga hızı şu formülle hesaplandı: NDH (m/sn)=boy temelli aort uzunluğu (cm) / (100 x akım geçiş süresi [sn]). Akım geçiş süresi, diyafram ve aort kapağındaki akımların başlangıç sürelerinin farkı olarak alındı.

***Bulgular:*** Kontrol grubuyla karşılaştırıldığında, obez çocukların kan basınçları daha yüksek bulunurken (p<0.001), kan değerleri (açlık glukozu, hemoglobin, serum kreatinin ve lipit düzeyleri) anlamlı farklılık göstermedi. Ekokardiyografik parametreler içinde, sol ventrikül diyastol sonu çapı, interventriküler septum kalınlığı, arka duvar kalınlığı, sol ventrikül kütle indeksi, sol atriyum çapı ve aort kökü çapı obez grupta anlamlı derecede daha yüksek değerlerdeydi (p<0.01). Obez çocuklarda NDH değerleri normal kilolu çocuklara göre artmış bulundu (4.0±0.8 ve 3.3±0.7 m/sn, p<0.001). Nabız dalga hızı, BKİ ile anlamlı pozitif ilişki gösterdi (r=0.391, p=0.002).

***Sonuç:*** Bulgularımız, aorttaki NDH'nin obez çocuklarda arttığını ve obezitenin erken yaşlarda bile subklinik ateroskleroza neden olabileceğini göstermektedir.

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Childhood obesity is a public health problem all over the world.[1] There has been a growing trend for becoming more obese in all ages as well as in children.[2,3] Obese children are likely to become obese adults and the associated morbidities can be expected to result in higher rates of hospitalizations, interventions, and premature deaths.[4] It is already well known that obesity is tightly linked to coronary artery disease and accepted as a major risk factor for developing coronary artery disease.[5,6] Although clinical complications of coronary heart disease mainly occur in middle ages, recent studies indicate that atherosclerotic process starts in childhood.[7] Fatty streaks first appear in the aortic intima at three years of age, and in the coronary arteries during the adolescence period.[8]

Pulse-wave velocity is the distance traveled by the wave divided by the time for the wave to travel that distance. It is a measure of arterial stiffness and has a strong correlation with cardiovascular events and all-cause mortality.[9,10] It has been recognized by the European Society of Hypertension as integral to the diagnosis and treatment of hypertension.[11] Several studies have shown that PWV is correlated with cardiovascular events also in children and adolescents and increases with age in both sexes.[12,13] Although the influence of obesity on PWV has been documented in adults, there is limited and conflicting information in childhood population.[13-18]

The aim of the present study was to assess PWV in obese children in comparison with lean controls.

## PATIENTS AND METHODS

### Patients

A case-control study was conducted between October 2010 and February 2011 in 30 obese schoolchildren and adolescents aged 10 to 16 years, who were referred from our primary health care pediatrician to our pediatric outpatient clinic. Selection was made consequently and patients presenting dysmorphic syndromes or with endocrine disorders were excluded. Those with structural heart disease or atrial fibrillation/flutter were also excluded from the study. A control group of 30 age- and gender-matched children who did not have obesity and dyslipidemia was also included in the study. No patients were on any cardiovascular medication and all were nonsmokers. Children were included in the study after informed consent of a guardian was given. The study protocol was approved by the hospital ethics committee.

### Clinical evaluation

**Abbreviations:**
*BMI* *Body mass index*
*PWV* *Pulse-wave velocity*

Family history of cardiovascular risks, personal history, and growth curve assessment were recorded for each patient. Anthropometric measurements (weight and height) were made using a standardized technique and obesity was defined as a body mass index greater than the 95th percentile for age, based on gender- and population-specific data.[19] Body mass index was calculated as weight in kilograms divided by the square of height in meters. Blood pressure was measured in the right arm of a relaxed, seated child, using correct cuff size and after a few minutes of rest in a calm room. Systolic and diastolic blood pressures were measured twice according to the recommendations of the guidelines.[20]

### Biochemical analysis

Blood samples for glucose, triglyceride, total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol levels were obtained after 12 hours of fasting. Glucose levels were measured with the glucose oxidase method. Plasma concentrations of total cholesterol and triglyceride were measured via routine enzymatic methods. High-density lipoprotein cholesterol was measured using a homogeneous colorimetric method. Low-density lipoprotein cholesterol concentration was calculated using the Friedewald formula.[21]

### Echocardiography and PWV measurement

All participants underwent PWV measurements using a conventional echocardiography device (Philips EnVisor C, Bothell, Washington, USA) and a 2.5 MHz transducer. Thoracic aortic length was calculated using a linear regression equation based on the height of the subject [thoracic aortic length (cm)= 1.7 cm+0.1 height (cm)].[22] Transit time was defined as the difference in the time of onset of two flows at the diaphragm and the aortic valve, measured by pulsed Doppler using the electrocardiogram as a time reference. Pulsed Doppler recordings were performed at a sweep speed of 100 m/sec, with the subject in normal sinus rhythm. Pulsed Doppler recordings of the ascending aorta were obtained from the apical view with the sample volume placed at the level of the valve leaflet tips. Pulsed Doppler recordings of the descending aorta were obtained from the subcostal sagittal view with the sample volume placed in the center of the aorta at the level of the diaphragm. The time from the onset of the QRS complex to the foot of the aortic waveform was measured at each location. Pulse-wave velocity was calculated using the

**Table 1. Demographic and clinical characteristics and echocardiographic findings of the study groups**

| | Obese subjects (n=30) | | | Lean subjects (n=30) | | | *p* |
|---|---|---|---|---|---|---|---|
| | n | % | Mean±SD/ Median ($Q_1$-$Q_3$) | n | % | Mean±SD/ Median ($Q_1$-$Q_3$) | |
| Age (years) | | | 13.2±2.0 | | | 12.5±1.7 | 0.193 |
| Sex | | | | | | | 0.793 |
| Male | 12 | 40.0 | | 13 | 43.3 | | |
| Female | 18 | 60.0 | | 17 | 56.7 | | |
| Body mass index (kg/m$^2$) | | | 26.0 (25.5-27.7) | | | 17.4 (16.5-20.1) | **<0.001** |
| Systolic blood pressure (mmHg) | | | 110 (100-110) | | | 90 (90-95) | **<0.001** |
| Diastolic blood pressure (mmHg) | | | 70 (65-70) | | | 60 (60-65) | **<0.001** |
| Fasting glucose (mg/dl) | | | 87.4±9.0 | | | 86.8±10.6 | 0.827 |
| Hemoglobin (g/dl) | | | 12.2±1.0 | | | 12.4±0.9 | 0.697 |
| Creatinine (mg/dl) | | | 0.5±0.1 | | | 0.5±0.1 | 0.535 |
| Total cholesterol (mg/dl) | | | 165.4±30.3 | | | 159.6±18.2 | 0.629 |
| LDL cholesterol (mg/dl) | | | 101.6±25.0 | | | 91.3±12.8 | 0.122 |
| HDL cholesterol (mg/dl) | | | 49.6±11.2 | | | 56.4±13.2 | 0.156 |
| Triglyceride (mg/dl) | | | 144.5±81.9 | | | 91.6±23.3 | 0.099 |
| Echocardiographic data | | | | | | | |
| Left ventricle | | | | | | | |
| Ejection fraction (%) | | | 68.2±5.9 | | | 67.8±5.0 | 0.814 |
| End-diastolic dimension (mm) | | | 40.9±4.7 | | | 37.4±3.3 | **0.002** |
| End-systolic dimension (mm) | | | 25.2±2.9 | | | 24.7±2.4 | 0.464 |
| Interventricular septum thickness (mm) | | | 9.0 (9.0-10.0) | | | 8.0 (7.0-9.0) | **<0.001** |
| Posterior wall thickness (mm) | | | 8.3±1.0 | | | 6.9±1.0 | **<0.001** |
| Mass index (g/m$^2$) | | | 73.4±9.5 | | | 65.8±10.5 | **0.004** |
| Aortic root dimension (mm) | | | 22.6±2.5 | | | 20.3±2.2 | **<0.001** |
| Left atrium dimension (mm) | | | 30.5±3.6 | | | 27.1±2.8 | **<0.001** |
| Pulse wave velocity (m/sec) | | | 4.0±0.8 | | | 3.3±0.7 | **<0.001** |

following equation: PWV (m/sec) = height-based aortic length (cm) / (100 x transit time [sec]). Subjects were refrained from taking caffeine-containing beverages at least three hours before the examination. Measurements were made in the supine position after at least 10 minutes rest in a calm room with a temperature of 22 °C. Measurements were performed individually by two different physicians and the mean of these measurements was used for analysis of Doppler data. Interobserver variability was also assessed.

### Statistical analysis

Following checking of the variables by the Kolmogorov-Smirnov normality test, the independent two-sample t-test was used to compare normally distributed variables, and Mann-Whitney U-test was used to compare non-normally distributed variables. Normally distributed continuous data were expressed as mean ± standard deviation, and non-normally distributed continuous variables were presented as median and interquartile range. Categorical data were expressed as count and percentages and were compared using the chi-square test. Correlations were sought using the Spearman's test. Interobserver variability was evaluated by the t-test. P values below 0.05 were considered statistically significant. Statistical analyses were performed using a commercial software (IBM SPSS Statistics 19).

## RESULTS

Demographic and clinical characteristics and echocardiographic findings of the two groups are shown in Table 1. Obese subjects had significantly higher BMI and higher blood pressure levels compared to the con-

trol group (p<0.001). There were no significant differences with respect to fasting glucose, hemoglobin, serum creatinine levels, and lipid parameters between the two groups.

Among echocardiographic findings, only left ventricular ejection fraction and end-systolic dimension were similar in the two groups (Table 1). The remaining echocardiographic parameters (left ventricular end-diastolic dimension, interventricular septum thickness, posterior wall thickness, left ventricular mass index, left atrium dimension, and aortic root dimension) were all significantly higher in obese subjects compared to controls (p<0.01).

Pulse-wave velocity measurements yielded significantly higher values in the obese group (p<0.001). In correlation analysis, PWV was positively correlated with BMI (r=0.391, p=0.002) (Fig. 1). Interobserver variability did not show any significant difference (p>0.05).

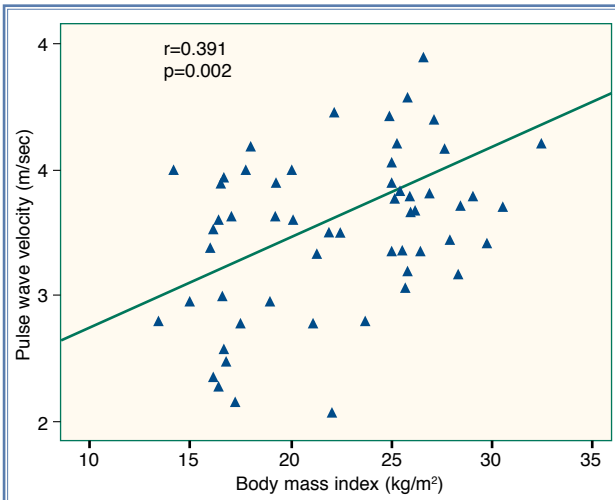


**Figure 1.** Correlation between pulse wave velocity and body mass index.

## DISCUSSION

The main finding of the present study is that aortic PWV is increased in obese children.

Obesity may start in childhood and continue until adulthood. Obese children are more likely to grow up as obese adults.[23] Severely obese children may have complications such as diabetes, hypercholesterolemia, hypertension, and atherosclerosis.[16] Considering the fact that cardiovascular diseases are the primary cause of mortality all over the world, it is very important to detect atherosclerosis at an early phase and prevent its progression to clinical events.

There are several noninvasive techniques, mostly based on ultrasound, to detect vascular injury in the subclinical phase. The most frequently used methods are quantification of flow-mediated dilatation of the brachial artery and measurement of intima-media thickness of the carotid artery.[24,25] Reduced flow-mediated dilatation and increased intima-media thickness have been found to be related with cardiovascular events in adults as well as in children.[24-26] Obesity has been shown to be highly associated with both in several studies.[27-30]

Pulse-wave velocity, an index of arterial stiffness, is a useful method for evaluating the severity of atherosclerosis.[9,10] Several studies have shown a close correlation between PWV and adult obesity.[18,31-33] It has been concluded that obesity stiffens the aortic wall in adults, but conflicting results exist in childhood population. Sakuragi et al.[15] found that BMI and body fat showed correlations with increased PWV in prepubescent children. Similarly, Urbina et al.[17] demonstrated increased arterial stiffness in young obese individuals. Pandit et al.[34] found that obese and overweight children had significantly increased stiffness, PWV, elastic modulus, and blood pressure compared to normal counterparts. However, several studies reported conflicting results. Dangardt et al.[14] who found decreased PWV in obese children hypothesized that decreased PWV in obese versus lean subjects might reflect general vasodilatation. Zebekakis et al.[18] reported that PWV did not increase with higher BMI in children over 10 years of age. Niboshi et al.[13] did not find any correlation between obesity and PWV in Japanese children. Lee et al.[16] showed that weight loss associated with a short-term exercise program did not affect PWV, despite significant decreases in blood pressure, waist circumference, and cholesterol levels. These conflicting results may be partially explained by the differences in patient populations (age, gender, sample size, and duration of obesity) and the heterogeneity of the techniques used. In our study, aortic PWV was significantly higher in obese children compared with lean controls. Yet, conflicting results in the literature prevent from drawing decisive conclusions as to whether aortic stiffness increases in obese children, requiring longitudinal studies with large samples.

Obesity may contribute to left ventricular dilatation by increasing left ventricular filling pressure. As a result of obesity-related hypertension, ventricular hypertrophy occurs.[35] In our study, obese children had more dilated and thickened left ventricles. Higher

blood pressure levels in the obese group may account for this dilatation and thickening. Moreover, more dilated left atrium and aortic root in obese subjects may contribute to this pathophysiology.

The main limitation of our study is the small sample size. Because small sample size results in low statistical power for equivalency testing, negative results may be simply due to chance. The lack of a reference method for studying PWV is another limitation. The carotid femoral method by pressure tonometry is the gold standard and the most widely used method of measuring central aortic PWV.[36] In our study, we used conventional echocardiography and pulsed Doppler recordings of the aorta to measure aortic PWV. This method has been validated for measuring aortic stiffness by Jo et al.[22] Unfortunately, we did not evaluate diastolic functions and left atrial functions especially in obese subjects with left atrial dilatation. It may also be necessary to perform anthropometric measurements in addition to BMI.

In conclusion, PWV is increased in obese children. Obesity may cause subclinical atherosclerosis even at early ages and this can be detected noninvasively by conventional echocardiography.

***Conflict-of-interest issues regarding the authorship or article: None declared***

## REFERENCES


1. Lobstein T, Baur L, Uauy R; IASO International Obesity Task Force. Obesity in children and young people: a crisis in public health. Obes Rev 2004;5 Suppl 1:4-104.
2. O'Brien PE, Dixon JB. The extent of the problem of obesity. Am J Surg 2002;184:4S-8S.
3. Onat A. The impact of obesity on cardiovascular diseases in Turkey. [Article in Turkish] Türk Kardiyol Dern Arş 2003;31:279-89.
4. DiPietro L, Mossberg HO, Stunkard AJ. A 40-year history of overweight children in Stockholm: life-time overweight, morbidity, and mortality. Int J Obes Relat Metab Disord 1994;18:585-90.
5. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation 1983;67:968-77.
6. Onat A, Ceyhan K, Sansoy V, Uyarel H, Yazıcı M, Uzunlar B, et al. Indices of abdominal obesity and obesity in Turkish adults: influence on levels of insulin, glucose intolerance, inflammation and on coronary prediction. [Article in Turkish] Türk Kardiyol Dern Arş 2003;31:65-73.
7. Stary HC. Evolution and progression of atherosclerotic lesions in coronary arteries of children and young adults. Arteriosclerosis 1989;9(1 Suppl):I19-32.
8. Ford ES; National Health and Nutrition Examination Survey. C-reactive protein concentration and cardiovascular disease risk factors in children: findings from the National Health and Nutrition Examination Survey 1999-2000. Circulation 2003;108:1053-8.
9. Yıldız M, Gül Ç, Kürüm T. Arterial pulse wave velocity. [Article in Turkish] Türk Kardiyol Dern Arş 2003;31:504-15.
10. Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. Hypertension 2001;37:1236-41.
11. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G, et al. 2007 Guidelines for the management of arterial hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). Eur Heart J 2007;28:1462-536.
12. Collins RT, Somes GW, Alpert BS. Arterial stiffness is increased in American adolescents compared to Japanese counterparts. Pediatr Cardiol 2009;30:794-9.
13. Niboshi A, Hamaoka K, Sakata K, Inoue F. Characteristics of brachial-ankle pulse wave velocity in Japanese children. Eur J Pediatr 2006;165:625-9.
14. Dangardt F, Osika W, Volkmann R, Gan LM, Friberg P. Obese children show increased intimal wall thickness and decreased pulse wave velocity. Clin Physiol Funct Imaging 2008;28:287-93.
15. Sakuragi S, Abhayaratna K, Gravenmaker KJ, O'Reilly C, Srikusalanukul W, Budge MM, et al. Influence of adiposity and physical activity on arterial stiffness in healthy children: the Lifestyle of Our Kids Study. Hypertension 2009;53:611-6.
16. Lee YH, Song YW, Kim HS, Lee SY, Jeong HS, Suh SH, et al. The effects of an exercise program on anthropometric, metabolic, and cardiovascular parameters in obese children. Korean Circ J 2010;40:179-84.
17. Urbina EM, Kimball TR, Khoury PR, Daniels SR, Dolan LM. Increased arterial stiffness is found in adolescents with obesity or obesity-related type 2 diabetes mellitus. J Hypertens 2010;28:1692-8.
18. Zebekakis PE, Nawrot T, Thijs L, Balkestein EJ, van der Heijden-Spek J, Van Bortel LM, et al. Obesity is associated with increased arterial stiffness from adolescence until old age. J Hypertens 2005;23:1839-46.
19. Poskitt EM. Defining childhood obesity: the relative body mass index (BMI). European Childhood Obesity group. Acta Paediatr 1995;84:961-3.
20. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics 2004;114(2 Suppl 4th Report):555-76.
21. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol

in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972;18:499-502.
22. Jo CO, Lande MB, Meagher CC, Wang H, Vermilion RP. A simple method of measuring thoracic aortic pulse wave velocity in children: methods and normal values. J Am Soc Echocardiogr 2010;23:735-40.
23. Steinberger J, Daniels SR, Eckel RH, Hayman L, Lustig RH, McCrindle B, et al. Progress and challenges in metabolic syndrome in children and adolescents: a scientific statement from the American Heart Association Atherosclerosis, Hypertension, and Obesity in the Young Committee of the Council on Cardiovascular Disease in the Young; Council on Cardiovascular Nursing; and Council on Nutrition, Physical Activity, and Metabolism. Circulation 2009;119:628-47.
24. Aggoun Y, Szezepanski I, Bonnet D. Noninvasive assessment of arterial stiffness and risk of atherosclerotic events in children. Pediatr Res 2005;58:173-8.
25. Groner JA, Joshi M, Bauer JA. Pediatric precursors of adult cardiovascular disease: noninvasive assessment of early vascular changes in children and adolescents. Pediatrics 2006;118:1683-91.
26. Tounian P, Aggoun Y, Dubern B, Varille V, Guy-Grand B, Sidi D, et al. Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study. Lancet 2001;358:1400-4.
27. Zhu W, Huang X, He J, Li M, Neubauer H. Arterial intima-media thickening and endothelial dysfunction in obese Chinese children. Eur J Pediatr 2005;164:337-44.
28. Woo KS, Chook P, Yu CW, Sung RY, Qiao M, Leung SS, et al. Overweight in children is associated with arterial endothelial dysfunction and intima-media thickening. Int J Obes Relat Metab Disord 2004;28:852-7.
29. Sorensen KE, Celermajer DS, Georgakopoulos D, Hatcher G, Betteridge DJ, Deanfield JE. Impairment of endothelium-dependent dilation is an early event in children with familial hypercholesterolemia and is related to the lipoprotein(a) level. J Clin Invest 1994;93:50-5.
30. van Merode T, Hick PJ, Hoeks AP, Reneman RS. Noninvasive assessment of artery wall properties in children aged 4-19 years. Pediatr Res 1989;25:94-6.
31. Wildman RP, Mackey RH, Bostom A, Thompson T, Sutton-Tyrrell K. Measures of obesity are associated with vascular stiffness in young and older adults. Hypertension 2003;42:468-73.
32. Rider OJ, Tayal U, Francis JM, Ali MK, Robinson MR, Byrne JP, et al. The effect of obesity and weight loss on aortic pulse wave velocity as assessed by magnetic resonance imaging. Obesity 2010;18:2311-6.
33. Güray Y, Refiker M, Demirkan B, Güray Ü, Boyacı A, Korkmaz Ş. Aortic strain and distensibility in patients with metabolic syndrome. [Article in Turkish] Türk Kardiyol Dern Arş 2010;38:14-9.
34. Pandit D, Kinare A, Chiplonkar S, Khadilkar A, Khadilkar V. Carotid arterial stiffness in overweight and obese Indian children. J Pediatr Endocrinol Metab 2011; 24:97-102.
35. Schunkert H. Obesity and target organ damage: the heart. Int J Obes Relat Metab Disord 2002;26 Suppl 4:S15-20.
36. Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. Eur Heart J 2006;27:2588-605.

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*Anahtar sözcükler:* Ergen; ateroskleroz/etyoloji; ekokardiyografi, Doppler, nabızlı/yöntem; elastisite; obezite/komplikasyon; vasküler direnç.