Effect of overweight on P-wave and QT dispersions in childhood

Çocuklukta fazla kilonun P dalgası ve QT dispersiyonları üzerine etkisi

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ABSTRACT

Objectives: The effects of obesity on atrial conduction and ventricular repolarization have been studied in detail, but these parameters have not been well documented in overweight children. The aim of our study was to investigate the effects of overweight on atrial conduction and ventricular repolarization in children by using P-wave dispersion (Pw-d) and QT dispersion (QT-d) analyses.

Study design: Sixty-seven overweight children and 70 children within normal limits were included in this cross-sectional prospective controlled study. All subjects underwent electrocardiographic and anthropometric evaluation, and blood samples were obtained. Pw-d and QT-d were investigated between two groups.

Results: Homeostatic model assessment of insulin resistance levels were higher in the overweight group (2.9±1.2 *vs.* 1.1±0.8, p=0.001). No statistically significant differences were found in Pw-d and QT-d when the groups were compared. The following findings were recorded for the overweight and control groups, respectively: mean RR interval (635±42 msec *vs.* 645±45 msec, p=0.867), Pw-d [30 (10-55) msec *vs.* 27.5 (15-50) msec, p=0.441] and QT-d (30 (15-55) msec *vs.* 22.5 (10-60) msec, p=0.476). In addition, Pw-d and QT-d were not correlated with the levels of insulin or body mass index.

Conclusion: There was no significant difference in atrial conduction or ventricular repolarization features between overweight children and normal-weight children.

ÖZET

Amaç: Şişmanlığın atriyum iletisi ve ventrikül repolarizasyonu dağılımı üzerine etkileri iyi araştırılmıştır. Ancak fazla kilolu çocuklarda bu parametreler yeteri kadar incelenmemiştir. Bu çalışmanın amacı çocuklarda fazla kilonun atriyum iletisi ve ventrikül repolarizasyonu dağılımı üzerine etkilerini, P dalga (Pw-d) ve QT (QT-d) dispersiyonunu hesaplayarak araştırmaktı.

Çalışma planı: Fazla kilolu 67 ve normal kilolu 70 çocuk kesitsel ve ileriye dönük kontrollü bir çalışma olan bu araştırmaya dahil edildi. Tüm çocuklarda elektrokardiyografik ve antropometrik inceleme yapıldı ve rutin kan tetkikleri için kan örnekleri toplandı. Pw-d ve QT-d her iki grupta araştırıldı.

Bulgular: Homeostatik modelle değerlendirilen insülin direnci düzeyleri fazla kilolu grupta daha yüksekti (2.9±1.2 ve 1.1±0.8, p=0.001). Her iki grup karşılaştırıldığında Pw-d ve QT-d arasında istatistiksel farklılık yoktu. Fazla kilolu ve normal kilolu grupta sırası ile şu bulgular saptandı: Ortalama RR aralığı (635±42 msn ve 645±45 msn, p=0.867), Pw-d [30 (10-55) msn ve 27.5 (15-50) msn, p=0.441], QT-d [30 (15-55) msn ve 22.5 (10-60) msn, p=0.476]. Ayrıca Pw-d ve QT-d hem serum insülin düzeyleri hem de beden kütle indeksi değerleri ile ilişkili değildi.

Sonuç: Fazla kilolu çocuklarda hem atriyum iletisi, hem de ventrikül repolarizasyonu dağılım özellikleri normal kilolu çocuklar ile karşılaştırıldığında istatistiksel olarak farklı değildi.

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Excess body mass index (BMI) is associated with metabolic disorders, cardiovascular diseases and diabetes mellitus, as well as psychiatric problems. Obesity prevalence in children is gradually increasing. Both overweight and obesity in children and adolescents are regarded as an extremely high risk for the development of atherosclerosis and cardiovascular complications in adulthood.[1-3]

Obesity in adults is a risk factor for atrial fibrillation;[4] however, there has been no study that proves that obesity causes atrial fibrillation in children. P-wave dispersion (Pw-d) is known as a non-homogeneous and interrupted conduction of sinusal impulses both intra- and interatrially, and a Pw-d value of >40 msec has been accepted as a marker for risk of atrial arrhythmias.[5,6]

There is a linear relationship between BMI and the QT corrected (QTc) interval.[7] Nigro et al.[8] put forward that heterogeneous ventricular repolarization and increased QT dispersion (QT-d) values are present in obese children. QT-d decreases significantly in obese subjects after weight loss, as does Pw-d.[9,10] Although QT-d has been accepted as reflecting the physiological variability of regional ventricular repolarization, a QT-d value of >40 msec was identified as facilitating ventricular arrhythmias.[11]

The normal values for P-wave indices and QT derivatives and the effect of morbid obesity combined with age on the QT derivatives are well established in children,[8,12-14] but the effects of the degree of weight on the P-wave indices and QT derivatives have not been studied in detail in overweight children. The aim of this study was to compare the Pw-d and QT-d between normal-weight and overweight children.

PATIENTS AND METHODS

Study design

This study is a cross-sectional, controlled study.

Study population and protocol

The study was conducted in our cardiology and pediatric clinics from June 2010 to June 2011. One hundred and thirty-seven consecutive subjects were studied, categorized into two groups. The first group was composed of 67 overweight children between 73 and 179 months of age, all of whom were overweight (BMI ≥25 and ≤29.9 kg/m^2). The second group was composed of 70 children between 72 and 177 months of age, all of whom were normal weight (BMI ≥18.6 and ≤24.9 kg/m^2); they served as the control group. For the overweight group, we recruited 38 female and 29 male children with a BMI between 25.02 and 29.93 kg/m^2 (mean BMI: 27.17±1.08 kg/m^2). For the control group, 30 females and 40 males with a BMI between 18.7 kg/m^2 and 24.81 kg/m^2 (mean BMI: 21.2±1.3 kg/m) were enrolled. Baseline characteristics such as anthropometric measurements, clinical features, and biochemical and hormonal parameters of the overweight and control groups are shown in Table 1.

Abbreviations:

BMI	*Body mass index*
ECG	*Electrocardiography*
HDL	*High-density lipoprotein*
IVSd	*Diastolic interventricular septum thickness*
LAd	*Left atrial dimension in early diastole*
LDL	*Low-density lipoprotein*
LVEF	*Left ventricle ejection fraction*
LVIDd	*Left ventricular end-diastolic internal diameter*
LVIDs	*Left ventricular end-systolic internal diameter*
LVPWd	*Diastolic posterior wall thickness*
TC	*Total cholesterol*
TG	*Triglyceride*
QTc	*QT corrected*
QTd	*QT dispersion*

The inclusion criteria were children with BMI ≥25 and BMI ≤29.9 kg/m^2 for the overweight group and children with BMI ≤24.9 kg/m^2 for the control group. The following exclusion criteria were defined for the two groups: children with BMI ≤18.5 kg/m^2; presence of atrial fibrillation or flutter; bundle branch or atrioventricular block; the presence of abnormal QTc (>450 msn or <330 msn); moderate-severe valvular disease; infection; the presence of left ventricle ejection fraction (LVEF) <45%; electrolyte disorders; history of heart disease, hypertension, diabetes mellitus, or any systemic disease; and those on medication known to affect electrocardiographic (ECG) parameters, such as beta-blocker, erythromycin or pseudoephedrine.

All subjects underwent routine clinical and laboratory examinations, including anthropometric measurements, 12-lead ECG, transthoracic echocardiography, and routine blood tests. We analyzed ECG and echocardiographic examinations on the same day. Blood samples were obtained the following day.

The procedure and the study protocol were approved by the bioethics committee of our institution.

Clinical, biochemical and echocardiographic measurements

Echocardiographic data were obtained using a com-

Table 1. Anthropometric and clinical features, and biochemical and hormonal parameters of the overweight and control groups

	Overweight group (n=67)			Control group (n=70)			*p**
	n	%	Mean±SD	n	%	Mean±SD	
Age (month)			118.7±23.9			114.2±26.2	0.231
Male	29	43.2		30	42.9		0.238
Female	38	56.8		40	57.1		0.441
Weight (kg)	60.4	38-82		31	15.8-55		**<0.001**
Height (cm)	139	112-179		135	109-174		0.098
Waist circumference (cm)	82	54-102		61	43-86		**<0.001**
Hip circumference (cm)	92.6	60-106		69	52.5-89		**<0.001**
Body mass index (kg/m^2)			27.17±1.08			21.2±1.3	**<0.001**
Systolic blood pressure (mmHg)			118.8±11.2			102.6±7.7	**<0.001**
Diastolic blood pressure (mmHg)			77.2±7.3			66.9±10.1	**<0.001**
Fasting glucose (mg/dl)			83.1±14.1			82.6±11.8	0.848
Total cholesterol (mg/dl)			166.9±28.4			159.8±26.2	0.199
Triglyceride (mg/dl)			98.6±50.9			80±56.8	0.089
LDL-C (mg/dl)			104.2±27.5			96.9±25.2	0.182
HDL (mg/dl)			43.1±7.4			49.2±11.4	**0.004**
Fasting insulin (μIU/ml)			14.2±7.1			4.55±4.7	**0.001**
HOMA			2.9±1.2			1.1±0.8	**0.001**
Hemoglobin (mg/dl)			13.02±0.79			13.01±0.85	0.440

Data are presented as mean ± SD and median (interquartile range). HDL: High-density lipoprotein; LDL: Low-density lipoprotein. *Chi-square, Mann-Whitney U test and Student's t test. Homeostatic model assessment (HOMA) index = (Fasting glucose X fasting insulin concentration X 0.0555) / 22.5.

mercial ultrasound machine with a cardiac probe (2.5-3.5 MHz) (Esaote, My Lab 50, Florence, Italy), and the following echocardiographic parameters were measured: left atrial dimension in early diastole (LAd), left ventricular end-diastolic internal diameter (LVIDd), left ventricular end-systolic internal diameter (LVIDs), diastolic interventricular septum thickness (IVSd), diastolic posterior wall thickness (LVPWd), and LVEF. Parameters were obtained in standard parasternal long-axis and short-axis views, as well as apical two-, four-, and five-chamber views. LVEF estimations were performed by two cardiologists with the modified Simpson's method.[15]

Anthropometric measurements were obtained from all subjects and were then used to calculate BMI. For the measurement of the levels of fasting glucose, total cholesterol (TC), triglyceride (TG), and high-density lipoprotein (HDL) were obtained from their fasting (≥10 hours) blood samples, and then their levels of low-density lipoprotein (LDL) were calculated using the Friedewald equation: LDL-C = TC (HDL-C [TG/5]).[16]

The quantitative analysis of insulin in serum samples was detected by using the Cobas e 601 kit (detection range: 0.2 mIU/ml - 1000 mIU/ml), which was obtained from Roche Diagnostics, Mannheim, Germany.

Measurement of P-wave indices and QT derivatives on 12-lead ECG

ECG recordings were taken with a standard 12-channel surface ECG at 50 mm/s speed and amplitude of 10 mm/mV (AT-2 plus Schiller™, Baar, Switzerland), in the same quiet room, with the subjects in supine positions, at the same time interval (09:00-12:00), in order to avoid diurnal variations. The 12-lead were simultaneously recorded, after which all ECG data

were calculated by two cardiologists unaware of the group assignments. Each ECG parameter was measured manually three times, and the average values were accepted. We measured the following parameters: P-wave maximum, P-wave minimum, QT minimum, and QT maximum, respectively, and the Pw-d and QT-d were then calculated.

P-wave duration was measured from the point of the visible upward or downward slope of P-waveforms to the point at which P-waveforms returned to the isoelectric line. The difference between the longest and shortest P-wave measurement was accepted as the Pw-d. QT interval was obtained from the beginning of the QRS complex to the point at which the steepest T-wave down-slope crossed the isoelectric line. The difference between the longest and the shortest QT interval in each of the 12-lead electrocardiograms was calculated for the value of the QT-d.

Statistics

We obtained all statistical data by using PASW® Statistics 18 for Windows (SPSS Inc., Chicago, IL, USA). All continuous variables were checked primarily by using the Kolmogorov-Smirnov test to show their distributions. Student's t test was used when the data fit the normal distribution, and Mann-Whitney U test was performed for data with abnormal distribution. A p value less than 0.05 was considered statistically significant.

The continuous variables are presented as mean±SD for parametric data, or median (interquartile range) for non-parametric data. Spearman rank correlation test was used to test the relationship between atrial conduction and ventricular repolarization parameters and BMI and serum insulin levels.

RESULTS

Baseline characteristics, biochemical analysis and echocardiographic measurements

Age, gender, height, levels of fasting glucose, TC, TG, LDL, and hemoglobin were similar between the two groups. All results are presented in Table 1. The following findings were recorded for the overweight and control groups, respectively: age (118.7±23.9 *vs.* 114.2±26.2 months, p=0.231); male gender (43.2% *vs.* 42.9%, p=0.238); height (142±10.8 *vs.* 134.9±11.9, p=0.242); fasting glucose levels (83.1±14.1 *vs.* 82.6±11.8 mg/dl, p=0.848); TC (166.9±28.4 *vs.* 159.8±26.2 mg/dl, p=0.199),

Table 2. Electrocardiographic and echocardiographic data of the overweight and control groups

	Overweight group (n=67)	Control group (n=70)	*p**
Mean heart rate (msc/min)	635±42	645±45	0.867
P-wave maximum (msc)	80 (60-120)	90 (80-110)	0.295
P-wave minimum (msc)	60 (45-80)	60 (40-80)	0.411
Pw-d (msc)	30 (10-55)	27.5 (15-50)	0.441
QT minimum (msc)	310 (260-350)	305 (290-340)	0.031
QT maximum (msc)	340 (285-370)	330 (310-370)	0.046
QT-d (msc)	30 (15-55)	22.5 (10-60)	0.476
LAd (mm)	25.5±2.2	24.4±1.8	0.083
LVIDd (mm)	37.1±3.5	34.4±2.8	0.234
LVIDs (mm)	22.8±2.0	21.3±3.06	0.178
IVSd (mm)	8.6±0.9	7.6±1.1	0.191
LVPWd (mm)	8.6±0.93	7.6±1.1	0.306
LVEF (%)	67.9±3.5	66.9±2.8	0.425

Data are presented as the median or the mean. * Mann-Whitney U-test and Student's t test. msc: Millisecond; LAd: Left atrial dimension in early diastole; LVIDd: Left ventricular end-diastolic internal diameter; LVIDs: Left ventricular end-systolic internal diameter; IVSd: Diastolic interventricular septum thickness; LVPWd: Diastolic posterior wall thickness; LVEF: Left ventricle ejection fraction; Pw-d: P-wave dispersion; QT-d: QT dispersion; QTc-d: Corrected QT dispersion.

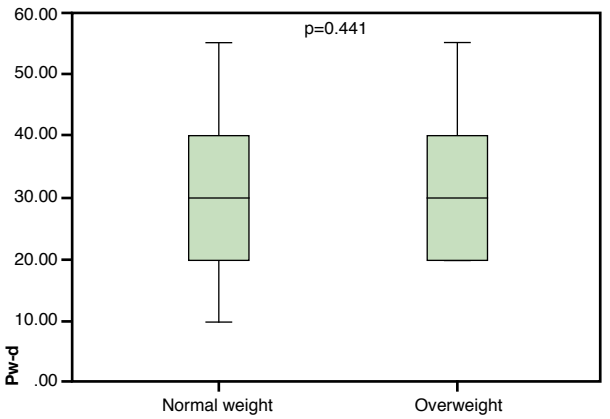

Figure 1. Comparison of P-wave dispersion between the two groups by a Box-and-Whisker plot. The thick horizontal line is the median, the filled box is the interquartile range, and the whiskers are 1.5 times the interquartile range.

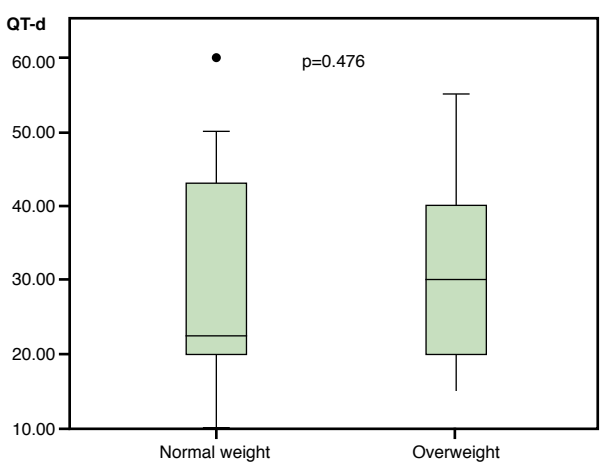

Figure 2. Comparison of QT dispersion between the two groups by a Box-and-Whisker plot.

TG (98.6±50.9 *vs.* 80±56.8 mg/dl, p=0.089), LDL (104.2±27.5 *vs.* 96.9±25.2 mg/dl, p=0.182), and hemoglobin (13.02±0.79 *vs.* 13.01±0.85 mg/dl, p=0.440) (Table 1).

Compared with the healthy control group, overweight children had a higher waist and hip circumference (p<0.001 and p<0.001), higher BMI (27.17±1.08 and 21.2±1.3 kg/m^2; p≤0.001), higher systolic blood pressure (118.8±11.2 and 102.6±7.7 mmHg; p≤0.001), higher diastolic blood pressure (77.2±7.3 and 66.9±10.1 mmHg; p≤0.001), higher levels of insulin serum concentration (14.2±7.1 and 4.55±4.7 µIU/L; p=0.001), higher homeostatic model assessment (HOMA) index values (2.9±1.2 *vs.* 1.1±0.8, p=0.001), and lower HDL (43.1±7.4 and 49.2±11.4 mg/dl; p=0.004), respectively (Table 1). Echocardiographic parameters (LAd, LVIDd, LVIDs, IVSd, LVPWd, and LVEF) were similar in the two groups, as shown in Table 2.

Assessments of heart rate, P-wave and QT rerivatives

There were no statistically significant differences between the overweight and normal- weight groups according to the analysis of the results of mean heart rate, P-wave maximum, P-wave minimum, and the Pw-d (635±42 *vs.* 645±45 msec, p=0.867; 80 [60-120] *vs.* 90 [80-110] msec, p=0.295; 60 [45-80] *vs.* 60 [40-80] msec, p=0.411; and 30 [10-55] *vs.* 27.5 [15-50] msec, p=0.441; respectively) (Table 2, Fig. 1).

Although QT minimum (310 [260-350] *vs.* 305 [290-340] msec, p=0.031) and QT maximum (340 [285-370] *vs.* 330 [310-370], p=0.046) were found to be higher in the overweight group compared to the control group, no significant difference was detected between the QT-d of the two groups (30 [15-55] msec *vs.* 22.5 [10-60] msec, p=0.476) (Table 2, Fig. 2).

The levels of serum insulin and BMI were not cor-

Table 3. Spearman rank correlation analysis according to serum insulin levels and body mass index in the overweight group (n=67)

	Levels of serum insulin		Body mass index	
	r	*p*	r	*p*
Mean heart rate (msc)	0.035	0.724	0.199	0.085
Pw-d (msc)	0.107	0.377	0.141	0.245
QT-d (msc)	0.183	0.129	0.159	0.188

msc: Milliseconds; r; Correlation coefficient; Pw-d: P-wave dispersion; QT-d: QT dispersion.

related with heart rate, QT-d or Pw-d in either group (Table 3).

DISCUSSION

In our study, we investigated whether differentiation existed on the dispersion of atrial conduction and the disparity of ventricular recovery times between overweight and normal-weight children. In the ECG analysis of overweight children, the Pw-d and QT-d durations were similar when compared with those of the control group.

Overweight is considered as a cardiovascular risk factor among children.[2] It is well known that increased BMI in overweight and obese children associates with higher blood pressures,[17] which may lead to left ventricle and left atrial size changes that may be responsible for the alteration of P-wave and QT measurements. In the present study, overweight children had higher systolic and diastolic blood pressures than normal-weight children, even though in the normotensive range. There were also mildly increased echocardiographic values in terms of LAd, LVDd, LVIDs, IVSd, and LVPWd in the overweight group compared to normal-weight children, but the differences were not statistically significant. Mildly higher blood pressures and increased insulin levels in the overweight children may potentially be responsible for the echocardiographic changes; however, these changes were not found to influence the Pw-d and QT-d in our data, and no significant correlation was found between Pw-d and QT-d and BMI or serum insulin levels.

A moderate increase in heart rate in overweight subjects may represent the changes in cardiac autonomic response, which are also known as a factor in QT interval duration.[18] A small increase in resting heart rate was correlated with BMI in the overweight children in our study. The exact mechanism of a prolonged QT interval in obesity is unknown. Some authors put forward that there is a relationship between cardiac arrhythmias and the high levels of circulating free fatty acids in obese subjects, disturbing the metabolism of myocardial energy.[19,20] Corbi et al.[20] showed that there was a positive correlation between the QTc and the plasma free fatty acid levels, and they also revealed that dietary weight loss produced a significant decrease in the mean QTc interval. Obese subjects have an increased cardiac output, which is calculated using stroke volume multiplied by heart rate, as a result of their higher body oxygen consumption.

Obesity is associated with Pw-d in accordance with enlarged left atrial diameter,[21] but whether there is a relation between BMI and Pw-d has not been well studied. In our study, there were no statistical differences in either left atrial dimension or P-wave indices between the overweight and normal-weight groups. Our findings suggest that overweight in children is not associated with left atrial enlargement and does not play a role in the electrical instability, unlike in obesity models.[22]

There was no heterogeneous ventricular repolarization for the overweight and normal-weight children in the study. QT prolongation represents longer duration of ventricular repolarization, while QT dispersion >40 msec indicates ventricular repolarization heterogeneity, which is associated with the differences in refractoriness in the consecutive phases of the cardiac cycle and related to an increase in vulnerability to arrhythmias.[23] In our study, although QT max and QT min were different between the two groups, there was no statistical difference in the values of QT-d between the overweight and normal-weight groups.

Vardar et al.[24] showed no significant differences in terms of ventricular repolarization between overweight and normal-weight young adults. El Gamal et al.[25] reported a positive correlation between BMI and QT interval in 742 moderately-morbidly obese adult patients. Nigro et al.[8] also found that there was significant ventricular repolarization heterogeneity in obese children when compared with normal-weight healthy children. Our data do not oppose Negro et al.'s or El Gamal et al.'s findings, because their studies dealt with another category, moderately and morbidly obese children.

Our results potentially suggest that BMI values >30 kg/m^2, as well as longer duration, may be required to affect homogeneous ventricular repolarization in children.

Study limitations

We did not classify whether overweight in our participants was central or visceral in spite of our measurement of the waist and hip. Recently, central obesity has been associated more with hyperinsulinemia, insulin resistance, dyslipidemia, and proinflammatory and prothrombotic clinical states.[26] If we studied an-

other group of obese as a third group, adding parameters of P-wave indices and QT derivatives, this would further increase the validity of the study. Finally, the relatively small number of subjects in the study group is the most important limitation of the study.

In conclusion, our study demonstrates that overweight children do not have abnormalities in homogeneous atrial conduction or dispersion of ventricular recovery times or echocardiographic changes. Further studies will be necessary to confirm that our study is applicable to the general population of overweight children.

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

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Key words: Body mass index; body weight; cardiovascular diseases / etiology; children; overweight / physiology.

Anahtar sözcükler: Beden kütle indeksi; vücut ağırlığı; kardiyovasküler hastalıklar/etiyoloji; çocuklar; aşırı kilo / fizyoloji.