P-wave and QT interval dispersion analysis in children with Eisenmenger syndrome

Eisenmenger sendromlu çocuklarda P dalgası ve QT intervali dispersiyon analizi

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ABSTRACT

Objectives: P-wave and QT dispersion are increased and associated with atrial and ventricular arrhythmia and an increase in sudden death in a variety of diseases. This study aimed to investigate P-wave and QT dispersion in children with Eisenmenger syndrome (ES).

Study design: The study group included 27 children (15 females, 12 males) with both congenital heart disease (CHD) and ES. The control group consisted of 30 children with CHD without pulmonary arterial hypertension. Electrocardiographic records were used to determine P-wave, QT, and corrected QT (QTc) dispersions. 24-hour (h) rhythm Holter was fitted in all patients. Atrial volumes, ventricular dimensions and tricuspid annular plane systolic excursion (TAPSE) were measured by echocardiography.

Results: There was no difference between groups with regard to age, sex, weight, and body surface area (p>0.05). Right atrial volume was significantly larger in the ES group than in the control group. P-wave, QT and QTc dispersions were higher in the patients with ES (50.10±11.12 *vs.* 26.32±8.90, p<0.001; 57.40±24.21 *vs.* 38.20±8.92 ms, p<0.001; and 78.20±16.02 *vs.* 56.52±13.92 ms, p<0.001, respectively). Ventricular and supraventricular ectopy were significantly more frequent in the ES group. Four patients (14.8%) in the study group had tachyarrhythmias during 24-h Holter monitoring.

Conclusion: In our study, P-wave and QT dispersion were found to be greater in children with ES than in the healthy control subjects.

ÖZET

Amaç: P dalgası ve QT dispersiyonu artışı atriyum ve ventrikül aritmileri ve çeşitli hastalıklarda ani ölüm riski artışıyla ilişkili bulunmuştur. Bu çalışmada, Eisenmenger sendromu (ES) olan çocuklarda P dalgası ve QT dispersiyonu araştırılması amaçlanmıştır.

Çalışma planı: Çalışma grubu doğumsal kalp hastalığı (DKH) ve ES bulunan 27 çocuk hastadan (15 kız, 12 erkek), kontrol grubu ise DKH olup pulmoner hipertansiyonu olmayan 30 çocuk hastadan oluşturuldu. Elektrokardiyografik kayıtlardan P dalga, QT ve düzeltilmiş QT (QTc) ölçüldü. Tüm hastalara 24 saatlik ritm Holter takıldı. Ekokardiyografi ile atriyum hacimleri, ventrikül boyutları ve triküspit kapak anülüs düzlemi sistolik hareketi (TAPSE) ölçüldü.

Bulgular: Her iki grup arasında yaş, cinsiyet, kilo ve vücut yüzey alanı arasında anlamlı fark yoktu (p>0.05). Çalışma grubunda sağ atriyum hacmi kontrol grubuna göre belirgin olarak daha genişti. ES'li çocuklarda P dalga, QT ve QTc dispersiyon süresi anlamlı olarak daha yüksek saptandı (sırasıyla, 50.10±11.12 ve 26.32±8.90, p<0.001; 57.40±24.21 ve 38.20±8.92 ms, p<0.001; 78.20±16.02 ve 56.52±13.92 ms, p<0.001). ES'li hastalarda anlamlı olarak daha sık ventriküler ve supraventriküler atım vardı. Yirmi dört saatlik ritm Holter izleminde çalışma grubundaki dört hastada (%14.8) taşiaritmi izlendi.

Sonuç: Çalışmamızda, ES'li çocuklarda P dalgası ve QT dispersiyonu sağlıklı kontrol grubu çocuklarına göre yüksek saptanmıştır.

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Eisenmenger syndrome (ES) occurs in patients with large, congenital cardiac or surgically created extracardiac left-to-right shunts. Development of the syndrome represents a point at which pulmonary hypertension is irreversible and is an indication that the cardiac lesion is likely inoperable.[1,2] ES affects multiple organ systems, including the hematologic, skeletal, renal, and neurologic systems, causing significant morbidity and mortality. Findings show QT dispersion to be an important electrophysiologic marker that exhibits a tendency for ventricular arrhythmia and even for sudden cardiac death.[3,4]

This study aimed to investigate P-wave, QT and corrected QT (QTc) dispersion in children with ES and to compare results in children with congenital heart disease (CHD) without pulmonary arterial hypertension (PAH).

PATIENTS AND METHODS

This retrospective study was performed with two groups of children who had been followed in our Pediatric Cardiology Department. The study group included 27 children with CHD whose pulmonary vascular resistance (PVR) was calculated to be greater than 12 Wood units/m², obtained during cardiac catheterization, and who were diagnosed with ES. The control group consisted of 30 children without PAH.

The study was approved by the local ethical committee.

Patients

Twenty-seven patients (15 females, 12 males) who were diagnosed with ES (study group) between June 2004 and October 2012 in our Pediatric Cardiology Department were included in this cross-sectional case-controlled study. The control group consisted of 30 children with CHD but no PAH. The study exclusion criteria were: known risk factors causing prolongation of QT interval, such as incomplete or complete bundle branch block, certain drugs, dietary deficiencies, metabolic disturbances, possible other familial diseases, long QT syndrome, and a history of previous cardiac surgery.

Clinical and hemodynamic records

Ages at the time of diagnosis of ES were obtained from the medical records of the patients. Cardiac catheterization and angiography records of the patients with ES were reviewed. Pulmonary flow (Qp) and systemic flow (Qs) were calculated using the Fick formula. PVR and systemic vascular resistance (SVR) were calculated using the following formulas: Rp = mean pulmonary artery pressure-mean left atrium (LA) pressure / Qp, and Rs = mean aortic pressure - mean right atrium (RA) pressure / Qs. In addition, left-to-right shunt, right-to-left shunt, LA pressure, RA pressure, mean pulmonary arterial pressure, and mean aortic pressure recordings were also evaluated. If LA pressure was not available, then left ventricular end-diastolic pressure or pulmonary capillary wedge pressure was used. Functional class was assessed by the World Health Organization (WHO) classification.

Abbreviations:

ASD	*Atrial septal defect*
CHD	*Congenital heart disease*
ECG	*Electrocardiography*
ES	*Eisenmenger syndrome*
PAH	*Pulmonary arterial hypertension*
PDA	*Patent ductus arteriosus*
PVR	*Pulmonary vascular resistance*
QTc	*QT and corrected*
SVT	*Supraventricular tachycardia*
TAPSE	*Tricuspid annular plane systolic excursion*
VSD	*Ventricular septal defect*
VT	*Ventricular tachycardia*

Electrocardiography (ECG) and 24-hour (h) rhythm holter monitoring

All ECGs at the time of diagnosis of ES were analyzed from the medical records of the patients. We examined 12-lead ECG recordings at a speed of 25 mm/s and an amplitude of 1 mV/cm with the patient lying down after 5 or 10 minutes (min) of rest in a silent room. During the ECG recording, all the children were in sinus rhythm. The high-resolution computer software program (Adobe Photoshop CS2) was used for evaluation of ECG results by a single observer. The beginning of the P-wave was considered as the joint between the isoelectric line and first prominent upward or downward incline of the trace. The return of the trace to the isoelectric line was defined to be the end of the P-wave. The difference between the longest and shortest P-waves was defined as P-wave dispersion. The QRS interval was measured from the start of the Q-wave or in absence of the Q-wave, from the start of the R-wave to the end of S, that is, to its return to the isoelectric line. The measurement of the QT interval was started from the onset of the QRS complex until the end of the T-wave. A discrete U-wave after T-wave was excluded from measurement. In case of fusion of two waves, the U component was included. The biphasic T-wave was defined as the T-wave with equal and opposite amplitude. When this occurred,

the point at which the last part of the wave crossed the isoelectric line was taken into account. QT and QTc dispersion were calculated as the difference between the maximum and minimum QT and QTc durations. The QTc duration was calculated using Bazett's formula.[5] The 24-h rhythm Holter was fitted (DMS 300-7 Holter recorder; DMS Inc.; New York, NY, USA), and the recordings were evaluated.

Echocardiography

All echocardiography examinations were performed with a commercially available echocardiographic machine, the Vivid S6 (Vingmed–General Electric), equipped with 4- and 7-megahertz transducers. Echocardiography was performed in a silent room, with the patient lying on the left side, calm and breathing comfortably. Measurements were performed through the parasternal window; long-axis and short-axis, apical window; 4- and 5-chamber views. PAH was defined as tricuspid regurgitant jet velocity ≥2.7 m/s by echocardiography. The LA volume was measured by single-plane area-length algorithm from apical 2-chamber and 4-chamber views and from their combination by means of Simpson's rule. RA volume was measured from the 4-chamber view in a manner similar to the LA volume. The tricuspid annular plane systolic excursion (TAPSE) value was measured during right ventricular systole in the right ventricle and tricuspid valve intersection point through the apical 4-chamber images of M-mode echocardiography.

Statistical analysis

Descriptive statistics for continuous variables (characteristics) are presented as mean, standard deviation, and minimum and maximum values as well as count and percent for categorical variables. Student's t test was performed to compare means of the patient and control groups for continuous variables. Non-normally distributed continuous variables were compared with Mann-Whitney U-test. Pearson correlation coefficient was used to determine linear relationships among these variables. In addition, chi-square test was used to indicate linear relationships among the categorical variables. Statistical significance levels were considered as 5%, and the Statistical Package for the Social Sciences (SPSS) 16.0 computer program (SPSS, Chicago, IL, USA) was used for the statistical analysis.

RESULTS

The mean age of study group was 11.32±3.82 (5-17) years and the mean age of the control group was 10.73±3.05 (3-15) years ($p>0.05$). There was no difference in the comparison of groups with regard to age, sex, weight, and body surface area ($p>0.05$). We found a statistically significant difference between the two groups in terms of oxygen saturation ($p<0.001$). Echocardiographic examination of the study group showed that 20 patients (74%) had ventricular septal defect (VSD), 12 patients (44.4%) - atrial septal defect (ASD), 7 patients (25.9%) - patent ductus arteriosus (PDA), 4 patients (14.8%) - complete atrioventricular septal defect, 6 patients (22.2%) - mild mitral regurgitation, 3 patients (11.1%) - mild aortic regurgitation, and 8 patients (29.6%) moderate-to-significant tricuspid regurgitation. In the control group, 18 patients (60%) had ASD, 6 patients (20%) - VSD, and 6 patients (20%) - PDA. There was no clinical significance of CHDs in the control group. Valve regurgitation was not found in the control group. Although RA volume was significantly larger in the study group, no difference was determined between the study and control groups regarding left/right end-diastolic diameter (LVEDD, RVEDD) and LA volume values. TAPSE levels in the study group were significantly lower than those in the control group ($p<0.001$). Demographic, clinical and echocardiographic characteristics of the subjects are shown in Table 1. Functional and hemodynamic characteristics of patients with ES are shown in Table 2. Mean pulmonary arterial pressure and mean aortic pressure were found to be 69.07±13.3 mmHg and 70.6±10.6 mmHg, respectively. Mean PVR and mean pulmonary-to-systemic resistance ratio of the patients were 17.8±3.4 U/m² and 1.06±0.19, respectively.

The ECG and Holter characteristics are presented in Table 3. Although P-wave dispersion and P maximum were significantly higher in the study group (50.10±11.12 *vs.* 26.32±8.90, $p<0.001$; 95.42±20.32 *vs.* 72.71±9.91 ms, $p<0.001$, respectively), no difference was determined between P minimum values of the study group and the control group (43.11±16.62 *vs.* 45.90±8.65 ms, respectively; $p>0.05$). There was a positive correlation between P-wave dispersion and RA dilatation ($p<0.001$, $r=0.760$). The minimum QT durations were found to be similar in the two groups

Table 1. Comparison of the demographic, clinical and echocardiographic characteristics of the groups

	Study group		Control group		*p*
Gender (Female/Male)	15/12		17/13		NS
Age (years)	11.32±3.82 (5-17)		10.73±3.05 (3.0-15)		NS
Weight (kg)	29.2±12.1 (10-49)		25.2±9.9 (9-45)		NS
Body surface area (m²)	0.95±0.31		0.94±0.14		NS
O_2 saturation (%)	82 (74-92)		95.5 (93-99)		**<0.001**
Type of congenital heart defect, n (%)	VSD	8 (29.6)	VSD	6 (20)	
	VSD+ASD	8 (29.6)	ASD	18 (60)	
	VSD+ASD+PDA	4 (14.8)	PDA	6 (20)	
	PDA	3 (11.1)			
	CAVSD	4 (14.8)			
RVEDD (cm)	3.55±1.20		3.3±1.08		NS
LVEDD (cm)	4.02±1.15		3.94±1.04		NS
TAPSE	14.8±1.72		21.7±3.07		**<0.001**
Left atrium volume (mL/m²)	27.22±5.35		26.15±4.05		NS
Right atrium volume (mL/m²)	31.62±7.24		23.91±6.52		**<0.001**

Data are shown as mean±standard deviation for normally distributed variables; variables without normal distribution are shown as median, minimum, and maximum. ASD: Atrial septal defect; CAVSD: Complete atrioventricular septal defect; LVDD: Left ventricular end-diastolic diameter; NS: Non-significant; PDA: Patent ductus arteriosus; RVEDD: Right ventricular end-diastolic diameter; TAPSE: Tricuspid annular plane systolic excursion; VSD: Ventricular septal defect.

(p>0.05). The duration of QT dispersion was found to be significantly longer in the ES group (57.40±24.21) than in the control group (38.20±8.92 ms; p<0.001). QTc dispersion duration was found to be significantly increased in the ES group compared with the control group (p<0.001). The durations and dispersions of QT and QTc are presented in Table 3. In order to investigate the tendency towards arrhythmias, we evaluated the relationship between QT dispersion and the catheter angiographic data of the patients with ES. There was a positive correlation between QT dispersion and PVR/SVR ratio (p=0.024, r=0.503). Twenty-four-

Table 2. Functional and hemodynamic characteristics of the patients with Eisenmenger syndrome associated with congenital heart disease

	n	%	Mean±SD
World Health Organization functional class			
I	2	7.4	
II	13	48.1	
III	11	40.7	
IV	1	3.7	
Six minute walk distance (m) at diagnosis, (n)			395.2±67.04
Mean right atrial pressure (mmHg)			7.4±1.8
Mean left atrial pressure (mm Hg)			7.5±2.4
Mean pulmonary arterial pressure (mmHg)			69.07±13.3
Mean arterial pressure (mmHg)			70.6±10.6
Pulmonary vascular resistance (Wood units/m²)			17.8±3.4
Pulmonary vascular resistance/Systemic vascular resistance ratio			1.06±0.19

All data are presented as means±SD unless otherwise stated. SD: Standard deviation.

Table 3. Comparison of the electrocardiographic and 24-h rhythm Holter parameters of the groups

	Study group		Control group		*p*
	n	Mean±SD	n	Mean±SD	
Minimum P-wave duration (ms)		43.11±16.62		45.90±8.65	NS
Maximum P-wave duration (ms)		95.42±20.32		72.71±9.91	**<0.001**
P-wave dispersion (ms)		50.10±11.12		26.32±8.90	**<0.001**
QRS duration (ms)		92.8±12.0		71.48±7.18	**<0.001**
QT minimum, ms (range)	276 (256-304)		280 (260-324)		NS
QT maximum, ms (range)	332 (260-424)		316 (304-364)		**0.04**
QT dispersion, ms		57.40±24.21		38.20±8.92	**<0.001**
QTc minimum, ms (range)	360.0 (324-464)		356 (340-404)		NS
QTc maximum, ms (range)	442 (400-496)		420 (380-444)		**0.04**
QTc dispersion, ms		78.20±16.02		56.52±13.92	**<0.001**
Average heart rate (beats/min)		90±9.7		83±7.2	**<0.01**
SVT (n)	2		0		
Atrial flutter (n)	1		0		
Ventricular tachycardia (n)	1		0		
SVEs, n (range)	1422 (82-12354)		108 (8-1200)		**<0.001**
VEs, n (range)	108 (10-6442)		102 (0-632)		**<0.01**

Data are shown as mean ± standard deviation for normally distributed variables; variables without normal distribution are shown as median, minimum, and maximum. NS: Non-significant; SVEs: Supraventricular extrasystoles; SVT: Supraventricular tachycardia; VEs: Ventricular extrasystoles.

hour rhythm Holter monitoring of the study group showed that 2 patients (7.4%) had non-sustained supraventricular tachycardia (SVT), 1 patient (3.7%) - atrial flutter, and 1 patient (3.7%) - non-sustained ventricular tachycardia (VT). Similarly, both supraventricular and ventricular ectopic beats were significantly higher in the study group during 24-h Holter monitoring ($p<0.001$) (Table 3). Tachyarrhythmia was not found in the control group.

DISCUSSION

Pulmonary arterial hypertension (PAH) is commonly associated with CHD and can develop earlier or later in life depending on the size and location of the underlying defect. Patients with PAH-CHD are at risk of developing right-to-left cardiac shunting, that is, ES. Arrhythmias are an increasingly common problem in patients with ES due to the morphological changes in the right cardiac chambers and the modulation of autonomic activity.[6] There are few published studies about dispersion analysis in children with PAH or ES.[7,8]

We investigated dispersion analysis in children with ES in comparison with those of age- and gender-matched CHD patients without PAH. In the current study, P-wave, QT and QTc dispersion were detected to be significantly greater in patients with ES than in the control subjects. In addition, we found that increased dispersion of repolarization was positively correlated with the PVR/SVR ratio.

Prolonged P-wave duration and increased P-wave dispersion are reported to carry an increased risk for atrial flutter or fibrillation.[9,10] Many studies[7,11-13] have shown that many diseases, such as PAH, bronchial asthma, diabetes mellitus, and acute rheumatic fever, in which the heart may be affected, exhibit a significantly longer P-wave duration. Large prospective clinical trials have shown that chronic atrial dilatation is an important and independent risk factor for the development of atrial fibrillation.[14] In our study, the RA was more enlarged in the study group than in the control group, and 2 patients had SVT and 1 had atrial flutter.

Some studies[8,15,16] have shown that QT disper-

sion can be a marker of increased risk for ventricular arrhythmia and even sudden cardiac death. It would be reasonable to suspect that sudden deaths in the ES population could be arrhythmic in origin. Therefore, evaluation of arrhythmias in patients with ES is of crucial importance. It has been proposed as a non-invasive electrocardiographic parameter that might predict an increased risk of malignant arrhythmias.[17] Although P-wave, QT and QTc dispersion are not an alternative to invasive methods of electrophysiology studies, they are useful and simple parameters for evaluating the tendency to arrhythmias in patients with ES. The normal range for QT and QTc dispersion are 40-50 ms and 10-44 ms, respectively.[18,19] The risk for serious ventricular arrhythmias or sudden death has been observed in subjects with QT dispersion greater than 65 ms. Similarly, QT dispersion >40 ms was found to have 88% sensitivity and 57% specificity for prediction of inducibility of sustained VT during an electrophysiology study.[20] In our study, QT and QTc dispersion were found as 57.40±24.21 and 78.20±16.02 ms, respectively, and 1 patient had VT. Semizel et al.[8] found that prolongation in QT and QTc dispersion may indicate an increased risk of cardiac arrhythmias, and increased QT dispersion was positively correlated with pulmonary-to-systemic resistance ratio in patients with ES. Similarly, QT and QTc dispersion were found to be higher in ES patients than in the controls and increased QT dispersion was positively correlated with PVR/SVR ratio in our study. This might indicate an increased risk of ventricular arrhythmias in a patient with high pulmonary resistance.

Limitations of the study

The limitations of this study include its retrospective design and the relatively small number of patients. The patients without PAH were determined by echocardiography alone, and right heart catheterization was deemed to be unethical in these patients. Further studies with larger sample sizes and longer follow-up periods could provide additional information.

In conclusion, the P-wave, QT and QTc dispersion were found to be greater in children with ES than in the healthy control subjects. Physicians should pay close attention to possible atrial and ventricular arrhythmias during the clinical follow-up assessment and treatment of these patients. The value of these abnormalities in the prediction of arrhythmia in patients with ES should be validated in further prospective studies.

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Ethical standards

The research does not involve human and/or animal experimentation.

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Key words: Arrhythmias, cardiac/physiopathology; heart catheterization; child; echocardiography; Eisenmenger complex/complications; electrocardiography; hypertension, pulmonary.

Anahtar sözcükler: Aritmi, kardiyak/fizyopatoloji; kalp kateterizasyonu; çocuk; ekokardiyografi; Eisenmenger kompleksi/komplikasyonlar; elektrakardiyografi; hipertansiyon, pulmoner.