

Increased P-Wave Duration and P-Wave Dispersion in Patients With Aortic Stenosis

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AORT DARLIKLIL HASTALARDA ARTMIŞ P-DALGA SÜRESİ VE P-DALGA DİSPERSİYONU

ÖZET

Maksimum ve minimum P-dalga süreleri arasındaki fark olarak tanımlanan P-dalga dispersiyonu (PDD)'nin paroksizmal atriyal fibrilasyonu (AF) öngörmede kullanışlı olduğu bildirilmiştir. AF, aort darlıklı hastalarda en sık aritmidir ve klinik bozulma için önemli bir prognostik göstergedir. Bu çalışmada amaç aort darlıklı hastalarda P-dalga dispersiyonunu değerlendirmektir. Çalışma popülasyonu iki grup içeriyordu. Grup I dejeneratif aort darlıklı 98 hasta (76 erkek, 22 kadın, yaş 63 ± 8 yıl) ve grup II herhangi bir kardiyovasküler hastalığı bulunmayan yaş ve cinsiyet açısından birbirine eşleştirilmiş 98 sağlıklı birey içeriyordu. Çalışmaya dahil edilen tüm bireylerin 12-derivasyonu elektrokardiyogramı çekildi. Yüzeysel elektrokardiyogramın tüm derivasyonlarında P-dalga süresi ölçüldü. Maksimum ve minimum P-dalga süresi arasındaki fark PDD olarak tanımlandı. Tüm hastalar ve kontrol bireyleri sol atrium çapı, sol ventrikül ejeksiyon fraksiyonu, sol ventrikül duvar kalınlıkları, maksimum ve ortalama aort gradiyenti ölçümü için ekokardiografi ile değerlendirildi. Ayrıca hastalar dokümente paroksizmal AF varlığı açısından değerlendirildi. Grup I'e ait maksimum P-dalga süresi (126 ms) ve PDD grup II'den anlamlı derecede daha yüksek bulundu ($p < 0.0001$). Buna ek olarak, paroksizmal AF'u bulunan hastalar (130 ms) bulunmayanlara (121 ms) göre anlamlı derecede daha yüksek maksimum P-dalga süresine ve PDD'na sahipti ($p < 0.001$). Minimum P-dalga süresi açısından iki grup arasında anlamlı fark yoktu ($p > 0.05$). Ekokardiografik değişkenlerle PDD'u arasında anlamlı korelasyon yoktu. Sonuç olarak, artmış paroksizmal AF riskini gösteren PDD'u aort darlıklı hastalarda, aort darlığı bulunmayan hastalardan anlamlı derecede daha yüksek bulundu. Ağır aort darlıklı hastalarda paroksizmal AF'u öngördürmede PDD'nun klinik kullanılabilirliğinin daha ileri değerlendirilmesi için daha uzun dönem prospektif çalışmalara ihtiyaç vardır. *Türk Kardiyol Dern Arş 2002; 30: 758-762*

Anahtar kelimeler: Aort darlığı, P-dalga dispersiyonu, atriyal fibrilasyon

P wave dispersion (PWD), defined as the difference between maximum and minimum P wave duration, is a new electrocardiographic marker that has been associated with inhomogeneous and discontinuous propagation of sinus impulses (1,2). The correlation between the presence of interatrial and intraatrial conduction abnormalities and the induction of paroxysmal atrial fibrillation (AF) has been well documented (3,4). In addition, prolonged P wave duration and increased PWD are commonly found in patients with a history of paroxysmal AF (2,5,6). AF, whether chronic or paroxysmal, is the most common sustained arrhythmia encountered in the clinical practice that produces substantial excess cardiovascular morbidity and mortality (7,8). The estimation of the probability of a patient developing frequent AF paroxysms might guide the clinician in the management of paroxysmal AF. Aortic stenosis (AS) is the most common cause of left ventricular outflow tract obstruction (9). AF is the most common arrhythmia seen in patients with severe AS (10). Furthermore, it is an important prognostic indicator for clinical deterioration in patients with AS (9).

The aim of the present study was to investigate a new electrocardiographic marker, PWD, in patients with severe AS. To our knowledge, this is the first study on the effects of AS on PWD.

METHODS

Study population

This prospective study consisted of two groups: Group I consisted of 98 patients with degenerative AS (76 men, 22 women; aged 63 ± 8 years) and group II consisted of 98 age and sex matched subjects (76 men, 22 women; aged 62 ± 6 years) who underwent coronary angiography in our cardiology clinic and considered as healthy subjects without any cardiovascular disease. All patients underwent selective coronary angiography. At the time of electrocardiographic recording, all subjects were in sinus rhythm, and none of

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them were taking any type of antiarrhythmic agent. Patients who had coronary artery disease, hypertension, moderate to severe aortic regurgitation, more than mild mitral regurgitation, mitral stenosis, diabetes mellitus, hyperthyroidism, pericardial effusion, chronic obstructive pulmonary disease, ventricular preexcitation, atrioventricular conduction abnormalities, or abnormal serum electrolytes were excluded from the study.

Electrocardiographic measurement

Twelve-lead electrocardiogram was recorded for each subject at a rate of 50 mm/s in the supine position by a 6-channel recorder (Hewlett Packard page writer, Model M 1772A, USA). Electrocardiograms were coded and all annotations were masked. The measurements of the P wave duration were performed manually by two of the investigators without knowledge of the clinical status of the patients and controls. To improve accuracy, measurements were performed with calipers and magnifying lens for defining the electrocardiographic deflection. P wave duration was measured from the onset to the offset of the P wave. The onset and offset of the P wave were defined as the junction between the P wave pattern and isoelectric line. After completion of the measurements, all electrocardiograms were decoded. PWD was defined as the difference between maximum and minimum P wave duration (2). Intraobserver and interobserver coefficients of variation were found to be 4.1% and 4.4% for PWD, respectively. All patients were evaluated for the presence of documented paroxysmal AF on a 12-lead electrocardiogram during their past medical history. Electrocardiographic diagnosis of paroxysmal AF was made according to Bellel's definition (11). Paroxysmal AF was diagnosed in a patient with a history of recurrent episodes of AF lasting >2 minutes and < 7 days.

Echocardiographic evaluation

Transthoracic echocardiographic examination was performed for each subject. Maximum and mean aortic gradients were calculated by Doppler studies. Color flow Doppler was used to detect the presence of mitral and aortic regurgitation. Left atrial diameter, left ventricular end diastolic and end systolic diameters, left ventricular ejection fraction, and left ventricular wall thicknesses were measured by M-mode echocardiography. Pulmonary artery systolic pressure was calculated by the help of continuous wave Doppler studies using the Bernoulli equation.

Statistical analysis

All numeric variables were expressed as mean±sd and categorical variables were expressed as percentage. Statistical analysis was performed using unpaired t test, Mann-Whitney U test and Chi-square test where appropriate, and Pearson correlation test was used to determine the correlation between PWD and echocardiographic variables in pati-

ents with AS. A p value < 0.05 was considered statistically significant.

RESULTS

There was no statistically significant difference between 2 groups in respect to age and gender (P>0.05). Eight patients (8%) were diagnosed as having documented paroxysmal AF with the evaluation of their past medical history. Echocardiographic variables were similar in patients with and without paroxysmal AF (table 1). Maximum P wave duration and PWD of group I were found to be significantly higher than those of group II (p<0.0001, table 2). However, there was no statistically significant difference between group I and group II regarding minimum P wave duration (p>0.05, table 2). Patients with AS and paroxysmal AF had higher maximum P wave duration and PWD than those without paroxysmal AF (p<0.001, table 3). There was no statistically significant difference between the values of minimum P wave duration in patients with and without paroxysmal AF (p>0.05, table 3). We found no significant correlation between PWD and echocardiographic variables including left atrial diameter, maximum and mean aortic gradients, left ventricular posterior wall and interventricular septal thicknesses and left ventricular ejection fraction (p>0.05 for all).

DISCUSSION

In this study, we found that patients with AS have longer maximum P wave duration and higher PWD than healthy control subjects. Besides, patients with AS and paroxysmal AF have higher values of maxi-

Table 1. Echocardiographic variables of patients with and without paroxysmal atrial fibrillation

Variable	Patients with paroxysmal AF (n=8)	Patients without paroxysmal AF (n=90)	p
Maximum aortic gradient (mmHg)	82±7	78±6	NS
Mean aortic gradient (mmHg)	43±6	42±7	NS
Thickness of interventricular septum (cm)	1.43±0.12	1.38±0.14	NS
Thickness of posterior wall (cm)	1.34±0.14	1.32±0.09	NS
Left atrial diameter (cm)	3.8±0.52	3.8±0.65	NS
Left ventricular ejection fraction (%)	66±5	66±6	NS

AF: Atrial fibrillation, NS: Nonsignificant

Table 2. Electrocardiographic variables in patients with aortic stenosis and in healthy control subjects

Variable	Control subjects	Patient with aortic stenosis	p
P minimum (ms)	76±8	78±7	NS
P maximum (ms)	108±7	126±8	<0.0001
P-wave dispersion (ms)	32±5	48±5	<0.0001

NS: Nonsignificant, P minimum: Minimum P-wave duration, P maximum: Maximum P-wave duration

Table 3. Electrocardiographic variables in patients with and without paroxysmal atrial fibrillation

Variable	Paroxysmal AF (-)	Paroxysmal AF (+)	p
P minimum (ms)	78±8	78±7	NS
P maximum (ms)	121±8	130±8	<0.001
P-wave dispersion (ms)	43±5	52±5	<0.001

AF: Atrial fibrillation, NS: Nonsignificant, P minimum: Minimum P-wave duration, P maximum: Maximum P-wave duration

mum P wave duration and PWD than those without paroxysmal AF. An interesting finding of this study is that there is no significant correlation between PWD and echocardiographic variables.

PWD is a new electrocardiographic marker that has been associated with the inhomogeneous and discontinuous propagation of sinus impulses (1,2). Prolongation of intraatrial and interatrial conduction time and inhomogeneous propagation of sinus impulses are well known electrophysiologic characteristics in patients with paroxysmal AF (1,2). Moreover, the correlation between the presence of intraatrial conduction abnormalities and the induction of paroxysmal AF has been well documented (3,12). This electrophysiologic characteristic results in increased PWD on electrocardiographic measurements. Therefore, PWD can be used to separate patients with a high risk of AF during sinus rhythm (12).

Degenerative calcific AS is now the most common cause of left ventricular outflow tract obstruction in adults (9). AF is the most common arrhythmia and an important prognostic indicator for clinical deterioration in patients with aortic stenosis (9,10,13). AF in a patient with severe AS results in loss of the atrial contribution to left ventricular filling, which causes a precipitous decline in cardiac output and aggravati-

on of symptoms (9). The estimation of the probability of a patient developing frequent AF paroxysms might guide the clinician in the management of paroxysmal AF.

P wave duration and PWD have been reported to be influenced by the autonomic tone, which induces changes in the velocity of impulse propagation (14). Furthermore, AF, either intermittent or chronic, may be influenced by autonomic activity (15). Ramirez-Gil et al (16) have shown increased sympathetic activity in patients with AS. In addition, Somsen et al (17) have reported that in a condition of cardiac pressure or volume overload, sympathetic activity is enhanced. Furthermore, Tükek et al (18) have reported that increased sympathetic activity causes a significant increase in PWD. As a result of this findings, we can suggest that increased sympathetic activity may be the underlying cause of higher PWD in patients with severe AS.

The elevation of left ventricular end-diastolic pressure, which is characteristic of severe AS, often reflects diminished compliance of the hypertrophied left ventricular wall (19,20). Increased left ventricular end-diastolic pressure in patients with severe aortic stenosis causes elevation of the left atrial pressure. As in all cardiac chambers, elevated intracavitary pressure may results in hypertrophy of that cavity. The development of myocardial fibrosis has been shown in hypertensive patients (21). Therefore, AS may alter the anatomic structures and electrical activity of the atrium as in hypertensive patients (22). Myocardial fibrosis may be responsible of heterogeneity of structural and electrophysiologic properties of the atrial myocardium. Consequently, propagation of sinus impulses may become inhomogeneous and discontinuous, resulting in prolonged P-wave duration and increased PWD.

We found no significant correlation between PWD and echocardiographic variables (left atrial diameter, left ventricular ejection fraction, thickness of interventricular septum and posterior wall, maximum and mean aortic gradients). In addition, patients with and without paroxysmal AF had similar echocardiographic variables (table 1). Previous authors have suggested that left atrial dimension (23-26), left ventricular dysfunction (23), and presence of organic heart di-

sease (27) could predict paroxysmal AF. However, our patients had a preserved left ventricular function, and a more or less normal mean left atrial maximal diameter. Recently, we have reported that PWD in patients with mitral stenosis is not related with left atrial diameter and the decrease in PWD after PMBV is not correlated with the improvement in left atrial diameter, mitral valve area, mean mitral gradient and left atrial pressure (28). Although some authors (18,26) reported that left atrial diameter is a significant predictor of AF episodes, some others (1,29,30) have reported that left atrial maximal diameter is not a significant predictor of paroxysmal AF.

In conclusion, PWD, indicating increased risk for paroxysmal AF, is significantly higher in patients with AS than in healthy control subjects. In addition, PWD in patients with AS associated with paroxysmal AF is significantly higher than those without paroxysmal AF. Further assessment of the clinical utility of PWD for the prediction of paroxysmal AF in patients with severe AS will require longer prospective studies.

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Düzelme

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