Effect of Cigarette Smoking on P Wawe Dispersion

SİGARA İÇİLMESİNİN P DALGA DİSPERSİYONUNA ETKİSİ

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Özet

Amaç: Sigara içilmesinin atriyoventriküler nodal iletiyi hızlandırdığı, atriyal fibrilasyonla birlikte çeşitli supraventriküler aritmilere neden olduğu gösterilmiştir. Çalışmamızın amacı 12 derivasyonlu yüzey elektrokardiyogramından kolayca elde edilen ve atriyal fibrilasyon gelişme riskini belirlemede kullanılan P dalga dispersiyonunun sigara içimi ile olan ilişkisini araştırmaktır.

Gereç ve Yöntemler: Çalışmaya sigara içen 48 olgu (30 erkek, 18 kadın, yaş ortalaması 47±13), hiç sigara içmemiş 29 olgu (17 erkek, 12 kadın yaş ortalaması 49±11) alındı Sigara içen ve içmeyen grubun P dalga süreleri, klinik ve ekokardiyografik bulguları karşılaştırıldı. Ayrıca sigara içenler, sigara içim sürelerine göre 3 gruba ayrılarak P dalga süreleri karşılaştırıldı.

Bulgular: P maksimum süresi ve P dispersiyonu; sigara içen grupta 106±11 ms ve 40±9 ms, sigara içmeyen grupta ise 93±9 ms ve 28±6 ms olup iki grup arasında anlamlı derecede farklıydı ($p<0.01$). Ayrıca sigara içenler içim süresine göre üç gruba ayrıldığında P maksimum süresi ve P dispersiyonu anlamlı derecede farklı olup sigara içim süreleri arttıkça P maksimum süresi ve P dispersiyonu artmaktaydı ($p<0.05$).

Sonuç: Sigara içenlerde nikotinin sempatik aktiviteyi artttırıcı etkisiyle P maksimum süresi ve P dispersiyonunun belirgin olarak arttığı, özellikle atriyal fibrilasyon riski yüksek hastalarda bu bulgunun pratik kullanımının daha geniş çalışmalarla araştırılması, bu konu ile ilgili ileride yapılacak çalışmalarda bu sonucun gözönüne alınması gerektiği söylenebilir.

Anahtar Kelimeler: P dalga dispersiyonu, sigara

Abstract

Objective: Cigarette smoking has been shown to accelerate atrioventricular node conduction and it could contribute to supraventricular arrhythmias, including atrial fibrillation (AF). The purpose of the present study is to investigate the effects of smoking on P wave dispersion which is reported to predict AF risk calculated on a 12 lead surface ECG.

Material and Methods: Fourty eight smokers (30 male and 18 female with a mean age of 47±13), and 29 subjects who had never smoked (17 male, 12 female with a mean age of 49±11) were included in to the study. The P wave durations, clinical and echocardiographic variables were compared between smokers and non-smokers. Smokers were also grouped into three subgroups according to the duration of smoking history and P wave durations were compared.

Results: Maximum P wave duration and P wave dispersion times were found significantly different ($p<0.01$, 106±11 ms versus 93±9 ms for P maximum and 40±9 ms versus 28±6 for P dispersion in smokers and non-smokers, respectively). The subgroups of smokers were compared. P maximum and P dispersion values were significantly different ($p<0.05$). The duration of P maximum and P dispersion increased as the duration of smoking history increases.

Conclusion: As a result, it can be said that p maximum and p dispersion is increased in smokers and this is an effect of nicotine which increases sympathetic activity and practical usage of this finding should research with larger studies, in patients,especially have high atrial fibrillation risk, this result should be taken into account in studies which are related this subject will be done future

Key Words: P wave dispersion, smoking

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Smoking is known to be a major risk factor for ischemic heart disease. Adverce effects on the cardiovascular system are related to the toxic substances, especially nicotine. Nicotine has been shown to increase the plasma catecholamine levels, heart rate, blood pressure and can cause sudden death by triggering atrial and ventricular arrythmias.[1-3]

Atrial fibrillation (AF) is the most common chronic arrhythmia encountered in the population and a predictor of death due to cardiovascular reasons. Several non-invasive electrocardiographic parameters were used to predict AF in high-risk patients. P wave dispersion was found to be related with the intraatrial and interatrial non-homogeneous depolarization and blocked conduction of the sinus node stimulus, providing the possibility to predict AF risk with the help of 12 lead ECG.[4,5]

The purpose of the present study is to investigate the effects of smoking on P wave dispersion which is an easy way to predict AF risk calculated from a 12 lead surface ECG.

Material and Methods

Study patients: Fourty eight smokers (30 male and 18 female with a mean age of 47±13), and 29 caseswho had never smoked (17 male, 12 female with a mean age of 49±11) included to the study. The P wave durations, clinical and echocardiographic variables were compared between smokers and non-smokers. Smokers were also grouped into three subgroups according to the duration of smoking history as group I (0-15 pack/year), group II (16-30 pack/year) and group III (>30 pack/year) and P wave durations were compared

The exclusion criteria were ischemic heart disease, hypertension, diabetes, chronic obstructive pulmonary disease, chronic renal disease, cardiomyopathies, thyroid function disturbances, electrolyte imbalance, branch blocks and arrhythmias on the ECG, alcohol consumption and any drug use that is known to interfere with atrial conduction.

Diabetes was defined as hyperglycemia requiring previous or ongoing pharmacological therapy. Hypertension was defined as either a systolic or diastolic elevation of blood pressure (>140/90 mm Hg) or ongoing antihypertensive pharmacological therapy. In addition, if segmenter wall motion defect was detected on echocardiography and/or if symptoms of ischemic heart disease were present, other noninvasive tests were undertaken and patients with one or more coronaries with more than %50 stenosis were defined as ischemic heart disease in coronary angiography.

Transthoracic echocardiography and electrocardiography: One experienced echocardiographer assessed echocardiography studies. All the subjects were screened by transtoracic echocardiography (Vivid 3, General Electric Vingmed Ultrasound, Israel) and their left ventricular ejection fraction, left atrial diameter and left atrial volume were recorded. 12 lead surface ECG's were recorded in each subject with sinus rhythm and normal ECG; in 50 mm/s rate and 1mV/cm amplitude. P wave dispersion measurements were performed with a high resolution monitor after the ECG's were scanned and stored to digital format as jpeg files in 1200 dot per inch (dpi) scale.[6] Adobe-photoshop software (Adobe systems inc.,USA) was used in reading and calculating the P wave morphology. The images were enlarged to 100% of the print size. The starting point of P wave was referred as the positive deflection crossing the isoelectric line and the end-point was referred as the end of the deflection crossing the isoelectric line. The patients were excluded if these points were not clear. The measurements were done in at least eight leads. The P wave dispersion is calculated by subtracting the minimum P wave duration time from the maximum duration.

Statistical analysis: Statistical analyses were performed by statistical program for social sciences (SPSS) software. Qualitative data were expressed as % and analysed by chi square test. Quantitative data were analysed with student's t test and expressed as mean ± SD. The data compared with ANOVA test in more than two groups. P value <0,05 was considered significant.

Results

The demographic, echocardiographic and electrocardiographic data were compared between the smokers and non-smokers. The results were expressed in Table 1. Maximum P wave duration and P wave dispersion were found significantly different between groups (106±11 ms versus 93±9 ms for P maximum and 40±9 ms versus 28±6 for P dispersion in smokers and non-smokers, respectively).

Table 1. The comparison of clinical, echocardiographic and electrocardiographic data between smokers and non-smokers

Variables		SMOKERS	CONTROL GROUP (NON-SMOKERS)	P value
Age		47±13	49±11	>0.05
Gender male/female		30/18	17/12	>0.05
Left atrial diameter	mm	36±7	35±6	>0.05
Left atrial volume	ml	46±21	45±12	>0.05
LV EF *	(%)	63±7	63±7	>0.05
P maximum	ms	106±11	93±9	**<0.01**
P minimum	ms	64±9	65±8	>0.05
P dispersion	ms	40±9	28±6	**<0.01**

*LV EF : Left ventricular ejection fraction

Table 2. The comparison of clinical, echocardiographic and electrocardiographic data of smokers who were grouped according to the duration of smoking history

Variables		Group I (0-15 pack/years)	Group II (16-30 pack/years)	Group III (>30 pack/years)	P value
Age		44±14	45±12	51±10	>0.05
Gender male/female		10/8	11/6	9/4	>0.05
Left atrial diameter	mm	33±7	37±9	38±6	>0.05
Left atrial volume	ml	43±18	48±14	49±22	<0.05
LV EF *	(%)	66±8	63±7	61±9	>0.05
P maximum	ms	96±12	103±10	111±7	**<0.05**
P minimum	ms	63±9	65±10	65±7	>0.05
P dispersion	ms	34±8	39±7	45±12	**<0.05**

*LV EF : Left ventricular ejection fraction

The subgroups of smokers were compared. P maximum and P dispersion values were significantly different (p:<0,05 Table 2). The duration of P maximum and P dispersion increased as the time of smoking history increases.

Discussion

Smoking is associated with an increased risk of acute cardiovascular events, including acute myocardial infarction, sudden death and stroke.[7] Mechanisms by which cigarette smoking is likely to contribute to acute vascular events include 1) induction of a hypercoagulable state; 2) increased myocardial work; 3) carbon monoxide–mediated reduced oxygen-carrying capacity of the blood; 4) coronary vasoconstriction 5) catecholamine release.[8]

Nicotine; the active agent in tobacco, is the main constituent of tobacco smoke responsible for the elevated risk of the cardiovascular disease and sudden coronary death associated with smoking, presumably by provoking cardiac arrhythmias.[9]

Cigarette smoking has been shown to accelerate atrioventricular node conduction, which could contribute to supraventricular arrhythmias, including atrial fibrillation.[10,11] The clinical consequences of AF are diverse. The loss of atrial contraction may significantly depress cardiac output, especially in patients with a noncompliant ventricle, and this may lead to congestive heart failure. Other sequelae include an increased risk of embolic events and stroke, angina pectoris, increased total and cardiac, impaired left ventricular systolic function, and tachycardia-induced cardiomyopathy. [12]

As it is clear that AF is a cause of cardiac morbidity and mortality, it is a primary target to identify patients with high AF occurrence risk and to prevent this arrhythmia. Several non-invasive electrocardiographic parameters are used extensively in order to identify the high-risk group for AF. It is expressed that P wave dispersion is related to the inter and intraatrial nonhomogenious conduction of the sinus node impulse, providing the possibility to predict AF risk via 12 derivation surface ECG.[4,5]

It is clear that a substantial number of patients with high AF risk are also smokers and that smoking contributes to the occurrence of AF. P wave dispersion was shown to increase in patients with ischemic heart disease and hypertension.[13,14] The issue of whether increase in P dispersion was due to the disease effect or smoking was not studied before. In our study, we investigated the independent effect of smoking on P wave dispersion by excluding patients with any condition which is known to increase the risk of AF and comparing healthy smokers and healthy nonsmokers as the control group.

In a recent paper, in which the clinical factors that increase the P wave duration were studied, only age was found to be correlated.[15] In our study, however, clinical factors (age, gender) were not significantly different between groups ($p>0,05$).

Although there are studies concluding that increase in left atrial diameter and decrease in left ventricular ejection fraction can increase p wave duration [16,17] there are also conflicting investigations.[4,18] In our study, although there was not a statistical difference in left atrial diamater, left atrial volume and left ventricular ejection fraction between two groups, P dispersion was increased in one group. This points out that these are not the only factors with an effect on P wave dispersion. Tükek et al.[19] demonstrated that the increase in sympathomimetic activity can increase in p wave durations. Nicotine in the tobacco, increases sympathomimetic activity, action potentials and depolarization membrane potentials possibly causing the increase in P wave dispersion.[9]

Another interesting finding in the present study, is the increase in P wave dispersion with the increase in the duration of smoking history ($p<0,05$).

As a result, it can be said that P maximum. and p dispersion is increased in smokers and this is an effect of nicotine which increases sympathetic activity which possible results in an increase in action potantial and depolarization membrane potential and, in addition, coronary vasoconstriction and increased myocardial work. Smoking can independently increase the risk of AF, [10,11] especially in the high risk group; and this should be taken into account in future studies.

Abbrevations: AF: Atrial Fibrillation.

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