

ARAŞTIRMA

Association between retinopathy and QT and P wave dispersion prolongation in hypertensive subjects

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Abstract

Objective: This study was designed to evaluate the relationship between prolonged P wave dispersion and development of retinopathy in hypertensive patients.

Methods: This case-controlled analytical study included hypertensive patients and healthy individuals. Hypertensive patients who presented to the Cardiology and Family Medicine outpatient clinics were investigated by clinical examinations, history, fundus examinations, electrocardiography (ECG), and biochemical studies. Healthy individuals comprised the control group. The hypertensive patients were classified into two groups as those with retinopathy and those without. The values of the ECG parameters, QT dispersions, and P wave dispersions of the control and the patient groups were investigated and compared.

Results: Forty-nine patients (21 male and 28 female) were included in the study. Retinopathy was not detected in twenty-five patients. Twenty-four of the patients had retinopathy. The P wave dispersions of the patient group were significantly longer than those of the control group (34 ± 10 and 25 ± 7.4 msec). There was no statistically significant difference in P wave dispersion between hypertensive patients with retinopathy and without retinopathy (37.3 ± 12.5 and

31.3 ± 7.5 msec). In male patients, those with retinopathy had significantly longer P wave dispersions than those without (41.6 ± 14.4 and 27.8 ± 6.7 msec).

Conclusion: Hypertensive patients have prolonged P wave dispersion. In these patients, prolonged P wave dispersion is associated with retinopathy development, particularly in males. P wave dispersion of hypertensive patients should be routinely studied, and those with higher values, particularly patients with retinopathy, should be followed-up for possible paroxysmal AF development.

Key Words: Hypertension, retinopathy, P wave dispersion

Hipertansif kişilerde QT ve P dalga dispersiyonu uzaması ve retinopati arasındaki ilişki

Özet

Amaç: Bu çalışma, hipertansif hastalarda retinopati gelişimi ve P dalgası dispersiyonu uzaması arasındaki ilişki değerlendirmek için tasarlanmıştır.

Metod: Bu çalışma hipertansif hasta ve sağlıklı kişileri içeren vaka kontrollü analitik bir çalışmadır. Kardiyoloji ve Aile hekimliği polikliniğine başvuran hipertansif hastalarda; klinik muayene, tıbbi tarihçe, fundus muayenesi, elektrokardiyografi ve biyokimyasal çalışma yapılmıştır. Kontrol grubu sağlıklı kişilerden oluşmaktadır. Hipertansif hastalar retinopati mevcut olan ve olmayan olmak üzere iki gruba

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ayrılmıştır. Hasta ve kontrol grubunda elektrokardiografi parametreleri, QT dispersiyonu ve P dispersiyonu değerleri ölçülmüş ve karşılaştırılmıştır.

Bulgular: Kırk dokuz hastayı içeren bir çalışmadır. Yirmibeş hastada retinopati saptanmadı. Yimidört hastada retinopati mevcuttu. P dalga dispersiyonu hasta grubunda kontrol grubundan anlamlı olarak daha uzun saptanmıştır (34 ± 10 and 25 ± 7.4 ms). Retinopatili ve retinopatisiz hipertansif hastalar arasında P dalga dispersiyonu açısından istatistiki anlamlı farklılık saptanmadı (37.3 ± 12.5 and 31.3 ± 7.5 ms). Retinopatili erkek hastalarda P dalga dispersiyonu retinopatisiz hastalardan anlamlı olarak daha uzundur (41.6 ± 14.4 and 27.8 ± 6.7 ms).

Sonuç: Hipertansif hastalarda P dalga dispersiyonu uzamıştır. Özellikle erkek hastalarda uzamış P dalga dispersiyonu retinopati gelişimi ile ilişkilidir. P dalga dispersiyonu hipertansif hastalarda rutin olarak çalışılmalı ve özellikle retinopati saptanan yüksek değerlerdeki hastalar paroksizmal atrial fibrilasyon olasılığı açısından takip edilmelidir.

Anahtar Kelimeler: Hipertansiyon, retinopati, P dalga dispersiyonu

Introduction

Is retinopathy associated with a cardiac pathology? A study on diabetic patients has shown retinopathy, especially the severe form, to be accompanied by restricted coronary flow reserve (1). Another study has revealed that increased heart beat rate was associated with diabetic retinopathy (2). Retinopathy is associated with impaired left ventricular systolic-diastolic functions in noninsulin-dependent diabetic patients (3,4). There are also studies claiming an association between serious left ventricle hypertrophy and diabetic retinopathy (5). The development of left ventricle hypertrophy and a simultaneous decrease in the heart beat rate have been striking (6). In left ventricle hypertrophy, normalizing of hypertrophy with treatment has decreased the risk of arrhythmia in hypertensive patients, and supraventricular and ventricular tachycardia have been detected through holter ECG (7,8). In patients with increased left ventricle mass, risky ventricular arrhythmia is more frequent as a result of prolonged QT dispersion (9). Left ventricle hypertrophy has been shown to be associated with prolonged QT dispersion (10).

Furthermore, there are studies on the association of prolonged P dispersion with paroxysmal atrial fibrillation in hypertensive patients (11). A value of P wave dispersion of up to 29 ms is considered normal.

Hypertension may have long-term complications, such as retinopathy and nephropathy because it has common clinical effects on the retina. All the changes are due to the direct effect of blood pressure on the vessel.

The literature review reveals an association of diabetic retinopathy with cardiac changes and the cardiac effects of hypertension. The present study aimed to investigate the effects of hypertension on electrocardiography, the changes in QT and P dispersion, the effects of hypertensive retinopathy on these changes as a novel aspect, and the effects of various electrocardiographic parameters of hypertension on each other, QT, and P dispersions in association with retinopathy.

Materials and methods

This was a cross-sectional, observational, and case-controlled analytical study. The patients were randomly selected as those with retinopathy and those without among the hypertensive patients who presented to the cardiology and family medicine outpatient clinics of the hospital. The hypertensive patients (higher than 140 mmHg systolic or 90 mmHg diastolic values with repeated measurement of blood pressure) with no accompanying disorder or drug use that could affect ECG findings and no electrolyte disturbance were included in the study. Diastolic or systolic murmur, electrolyte disturbance, valvular heart disease, and use of beta blockers or antiarrhythmic drugs that affect the cardiac conduction system comprised the exclusion criteria. Healthy individuals with no hypertension or any other disease and with normal values of biochemical parameters comprised the control group.

All the patients and the control subjects underwent evaluations of history, physical examination, biochemical analyses (including the electrolytes), and electrocardiography (ECG). A fundus examination was performed in all in the ophthalmology clinic. The study was carried out by specialists from the Family Medicine, Cardiology, and Ophthalmology clinics. Two double-blinded independent cardiologists evaluated the ECG findings, the ophthalmologist who performed the mydriatic fundus examinations was blinded to the disease conditions and ECG evaluations, and the cardiologists were blinded to the ophthalmologic evaluations of the groups. The findings and the laboratory values of the patients were followed up at various times.

The duration of the disease and any drug intake were recorded. The biochemical parameters, electrolyte disturbances, and lipid parameters of

the patients were evaluated. The findings of the fundus examination were graded as follows: Normal, Grade-1, Grade-2, Grade-3, and Grade-4. In grade 1 and 2 retinopathy, arteriolar vasoconstriction occurs to prevent hyper perfusion and intracapillary hypertension, which leads to increased pressure in arteriolar and capillary circulation, endothelial cell damage, obliteration, and rupture in these vessels (12). Flame shaped hemorrhages, cotton wool spots, and yellow-white exudates are observed in grade 3 retinopathy. In grade 4, papil edemas also accompany these changes (13). The hypertensive patients were divided into two groups as those without retinopathy (Group 1) and those with retinopathy (Group 2). For all the patients and the control subjects, concurrent 12-derivation ECG was performed at 25 mm/sec. While they were lying down (with a Nihon-Kohden trade-

mark Electrocardiography device). While they were allowed to breathe at a normal rate during ECG, they were told not to move or speak. PR segment distances; QRS times and QT intervals; SV1+RV5 amplitudes were recorded.

P wave duration, one of the derivations concurrently recorded, was measured. The measurement was performed after ECG recordings were transferred to the digital media and enlarged nearly ten times on a high-resolution monitor. This has been reported as one of the best techniques for P wave measurement (14). The beginning of the P wave was determined as the intersection of first deflection and isoelectric line, and the end of P wave was determined as the intersection of the last deflection and isoelectric line. The P wave dispersion was calculated by subtracting

Table 1. Clinical Characteristic of Hypertensive Patients

Characteristic	Control	Hypertensive without Retinopathy	Hypertensive with Retinopathy
Men (N, percent)	15 (58)	11 (44)	10 (42)
Women (N, percent)	11 (42)	14 (56)	14 (58)
Age (Mean, SD)	52.5 ± 9	56 ± 7.7	58.7 ± 10
Duration			
Of disease (Mean ,SD)		6.6± 5.5	6.4 ± 5.7
Medication			
ACE inh. (N, percent)		15 (60)	20 (83)
Diuretic (N, percent)		1 (4)	1 (4.2)
ARB (N, percent)		9 (36)	3 (12.6)

Table 2. Electrocardiographic Features of Subjects

Parameter	Control 26	Hypertensive without Retinopathy 25	Hypertensive with Retinopathy 24	p
Heart Rate	74.8 ± 15	77.8 ± 14.8	73 ± 12.3	NS
PR ms	145.5 ± 27	154.2 ± 30.6	155.5 ± 19.6	NS
QT dispersion ms	32.4 ± 8.8	33.7 ± 10	39.4 ± 15.4	NS
QTc dispersion ms	35 ± 9.5	35.9 ± 11.2	41.4 ± 14.2	NS
S1+R5 mm	196.5 ± 85.2	216 ± 80.9	224.4 ± 84.9	NS
P dispersion	25 ± 7.4	31.3 ± 7.5	37.3 ± 12.5	< 0.001
Heart Rate (beat/minute), P dispersion (ms)				

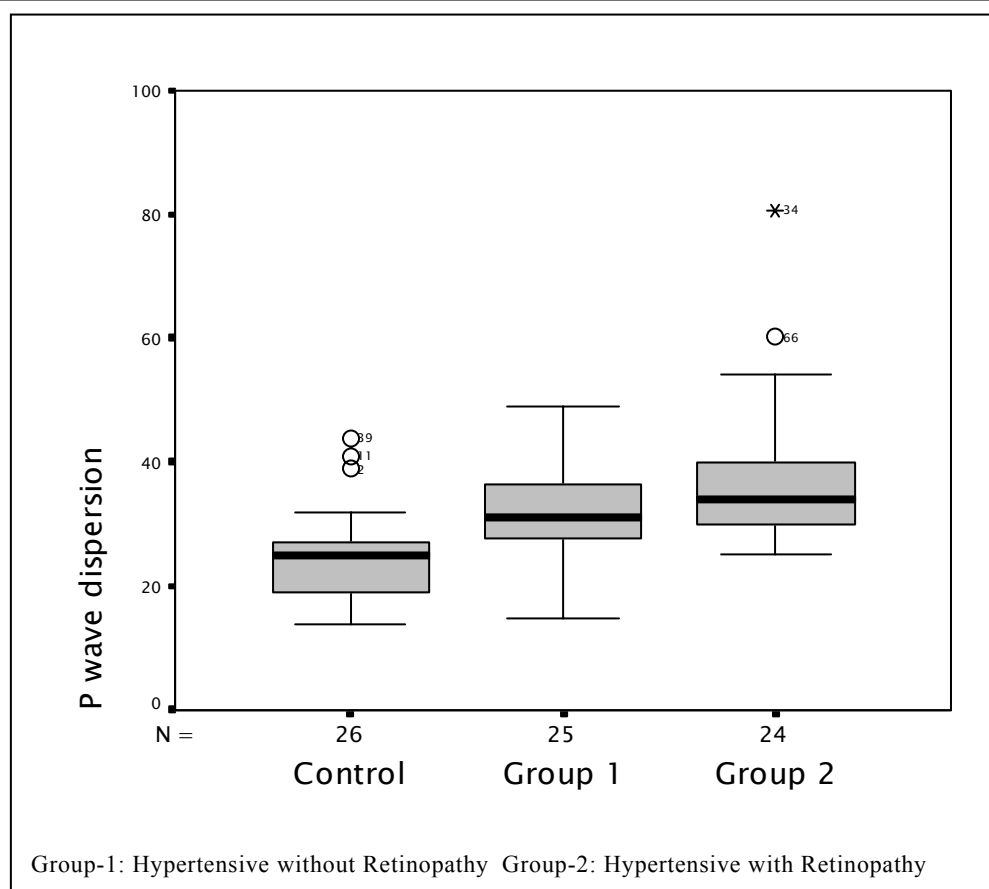


Figure 1. Comparison of P wave dispersion of Groups generally

minimum P wave duration (P minimum) from maximum P wave duration (P maximum) ($P \text{ dispersion} = P \text{ maximum} - P \text{ minimum}$). The P wave was measured for at least ten derivations of each ECG. The QT interval of each derivation was measured from the beginning of the QRS complex until the end of the T wave where the isoelectric line started. When the T wave followed the U wave, the lowest point between T and U waves was taken as a basis. The derivations with vague T wave end-points were excluded from the study. The difference between the shortest and longest QT distances was measured, and QT dispersion was calculated. QT values were corrected through Bazett's formula, taking the effect of heart beat into consideration. Similarly, QT dispersions were calculated based on the corrected formula.

Statistical analyses

The PR segment distances, SV1+RV5 amplitudes, P dispersion, QT and QTc dispersion durations of all groups were analyzed. Initially, the Kruskal-Wallis test was used between the three groups. Differences in the means of continuous measurements were tested by the

Mann-Whitney U test. The correlations among and between the parameters and age were studied through multivariate analyses. A P value of <0.05 was considered statistically significant; all tests were two-tailed. All statistical analyses were performed using SPSS version 11.5 (SPSS, Inc., Chicago, Illinois).

Results

The study involved 60 hypertensive patients, 11 of whom were excluded from the study (3 patients due to β -blocker use, 3 due to diabetes mellitus, 3 due to valvular heart disease, and 2 due to electrolyte disturbance). Forty-nine patients met the inclusion criteria.

Patient characteristics

This study included forty-nine hypertensive patients (21 male and 28 female) and twenty-six healthy individuals (15 male and 11 female). Retinopathy was not detected in twenty-five patients (11 male and 14 female). Twenty-four of the patients had retinopathy (10 male and 14 female). The mean age of the control subjects was 52.5 ± 9 , for the patients without retinopathy it was 56 ± 7.7 , and for the patients with

retinopathy it was 58.7 ± 10 . There were no statistically significant differences between the groups in terms of age or disease duration. (Table 1) Twenty-three of the patients had grade 1 retinopathy, while one had grade 3. Creatinine levels of all the patients were within reference values. The drugs most frequently used for treatment were ACE inhibitors. Other clinical features of the patients are presented in Table 1.

Comparisons of ECG parameters of the groups

In the comparison of the ECG parameters of all three groups, there were no statistically

significant differences except for among P wave dispersions (Kruskal-Wallis $X^2 = 20.6$, $SD = 2$ $P \leq 0.001$) (Table 2). With respect to P wave dispersions, there were significant differences between the control group and Group 1 (hypertensive patients without retinopathy) (means= 25 ± 7.4 and 31.3 ± 7.4 ms Mann-Whitney U $P = 0.002$) and between the control group and Group 2 (hypertensive patients with retinopathy) (means= 25 ± 7.4 and 37.3 ± 12.5 ms Mann-Whitney U $P \leq 0.001$). However, between Groups 1 and 2, no statistically significant differences were found in terms of P wave dispersion (Mann-Whitney U $P = 0.114$) (Fig. 1).

Table 3. Electrocardiographic Features of Study population

Parameter	Control	Hypertensive without Retinopathy	Hypertensive with Retinopathy	p
Age				
Men	55.3 ± 9.7	57.3 ± 8.5	58.3 ± 9.4	NS
Women	48.8 ± 7.5	55 ± 7.2	58.9 ± 10.7	NS
Duration of Disease (year)				
Men		7.4 ± 6.7	3.3 ± 2.7	
Women		6 ± 4.6	8.6 ± 6.4	
Heart Rate (beat/minute)				
Men	73 ± 14	74 ± 17.5	68.7 ± 13	NS
Women	77.3 ± 15.6	80.9 ± 12	76 ± 11	NS
PR (ms)				
Men	156.7 ± 22	160.4 ± 25	152.8 ± 18.4	NS
Women	130.4 ± 26.4	149.3 ± 34.4	157.4 ± 21	0.023
QT dispersion (ms)				
Men	33 ± 7.7	34.3 ± 9.5	46.3 ± 21.7	NS
Women	31.5 ± 10.4	33.2 ± 10.9	34.5 ± 5.7	NS
QTc dispersion (ms)				
Men	35.3 ± 7.5	36.3 ± 11	47.3 ± 19.8	NS
Women	34.8 ± 12	35.5 ± 11	37.3 ± 6	NS
P dispersion (ms)				
Men	26.0 ± 7.3	27.8 ± 6.7	41.6 ± 14.4	0.001
Women	23.7 ± 7.7	33.9 ± 7	34.2 ± 10.5	NS

Comparisons of ECG parameters according to gender

While there were no significant differences among the females when a gender-based

comparison was made between the two groups (Group 1 and 2) with regard to P wave dispersions (Mann-Whitney U $P = 0.5$), in males the P wave dispersions of Group 2 were significantly higher than those of Group 1

(means= 41.6 ± 14.4 and 27.8 ± 6.6 ms Mann-Whitney U $P=0.001$). In gender-based comparisons, there were no significant differences in terms of age or disease duration between Group 1 and Group 2 (Table 3). We

performed factorial ANOVA analysis to estimate the possible effects of age and disease duration between the groups among males. We observed significance among the groups in the corrected model (Table 4).

Table 4. Output of Factorial ANOVA for testing between-subjects effects on P wave Dispersion scores among men

Dependent variable: P wave Dispersion					
Source	Type III Sum of Squares	df	Mean Square	F	p
Corrected Model	1150.8	3	383.6	3	0.059
Intercept	113	1	113	0.89	0.36
Retinopathy Group	798.3	1	798.3	6.2	0.023*
Age	133.4	1	133.4	1	0.32
Duration of disease	0.334	1	0.334	0.003	0.96
Total	28167.9	21			
Retinopathy Group: Group 1: Hypertensive without Retinopathy Group 2: Hypertensive with Retinopathy					

Table 5. Output of Factorial ANOVA for testing between-subjects effects on P wave Dispersion scores among subjects older than 50 yrs

Dependent variable: P wave Dispersion					
Source	Type III Sum of Squares	df	Mean Square	F	p
Corrected Model	580.24	3	193.4	1.5	0.226
Intercept	600.4	1	600.4	4.7	0.037
Retinopathy Group	549	1	549	4.4	0.046*
Age	5.6	1	5.6	0.044	0.834
Duration of disease	0.207	1	0.207	0.002	0.97
Total	42574	34			
Retinopathy Group: Group 1: Hypertensive without Retinopathy Group 2: Hypertensive with Retinopathy					

Comparisons of ECG parameters according to age

In paired comparisons of the groups, there were no significant differences in P wave dispersions between Group 1 (hypertensive patients without retinopathy) and Group 2 (hypertensive patients with retinopathy) in either age group (under 50 years of age and over 50). However, we performed factorial ANOVA analysis for estimating the possible effects of age and disease duration between the groups. We observed

significance among Group 1 and Group 2 in the subjects above 50 years old in the corrected model (Table 5).

Discussion

Hypertension is a significant cause of mortality and morbidity due to the target organ damage. The retina is considered to be one of the target organs in the hypertensive disease. Hypertensive retinopathy has long been regarded as a risk indicator for systemic morbidity and mortality. Hypertensive retinopathy signs are associated

with systemic vascular diseases (15). Ophthalmoscopic examination is a simple and important method for determining the grade of vascular damage in essential hypertension.

In this study, the hypertensive patients did not seem to differ significantly from the control subjects in terms of ECG parameters. However, subtle differences may be detected with further studies such as P wave dispersion measurements. Furthermore, it can bring about important changes in structural and molecular structure of atrium. The studies claiming that hypertension leads to prolonged P wave dispersion have also proposed that in patients with essential hypertension P wave length and prolonged P wave dispersion trigger atrial fibrillation development independent of age, left ventricle mass, and left atrial dimension (11).

To the best of our knowledge, this is the first report to evaluate the association between the retinopathy and P wave dispersion in hypertensive patients. In our study, the P wave dispersions of both the hypertensive patients with retinopathy and those without retinopathy were longer than the P wave dispersions of the control group, which was more significant in the patients over 50 years of age.

For maximal P wave, however, there were no differences among the groups. These findings indicate that hypertension is the primary factor in the prolonged P wave dispersion. Among the hypertensive patients, only two patients from each of the two groups had left ventricular hypertrophy in ECG ($RV1+SV5 > 35$ mm). Their cardiac indexes, on the other hand, were within reference values on telecardiography. This suggests that hypertension causes changes to the P wave even before it leads to hypertrophy in the heart. Prolonged QT dispersion in hypertensive patients has been shown to be associated with left ventricle hypertrophy and to cause serious arrhythmia (10). In our study, despite the longer QT dispersions in the study group compared to those of the control group, the difference was not statistically significant. This may be attributed to the relatively low number of patients with left ventricle hypertrophy.

Previous study has been reported that P wave dispersion could be correlated with left atrium diameter, atrial maximal and ejection volume, left ventricular index and diastolic functions in hypertensive cases. P wave dispersion can be used as a non-invasive marker of target organ damage (such as left ventricular diastolic dysfunction) in the hypertension population (16). In our study, the P wave dispersions of hypertensive patients with retinopathy were

longer than the hypertensive patients without retinopathy, which is even longer in males with retinopathy development. We have demonstrated that P wave dispersion increased and was correlated with target organ damage such as retinopathy in hypertensive males.

The importance of hypertensive retinopathy is that it is a non-invasive indicator of poor arterial blood pressure management, the risk of which is more marked in males.

Addo et al. showed that the odds of developing target organ damage was lower in women compared to men. This can be accounted for the susceptibility of males to cardiovascular complications because of their hormonal and constitutional features (17). There is a need to examine the reasons for the poor control of hypertension in this population and particularly in men.

Prolonged P wave dispersion has been shown to trigger atrial fibrillation development in hypertensive patients (14). In our patients, significantly prolonged P wave dispersions may suggest possible paroxysmal atrial fibrillation development. The patients with markedly prolonged P wave dispersions, particularly male patients with retinopathy, should be observed using ECG for 24 hours.

Hypertension alone prolongs P wave dispersion, which increases the risk of paroxysmal atrial fibrillation development. Retinopathy development, particularly in males and at an advanced age, is an additional risk factor.

In conclusion, it can be concluded that P wave dispersion measurements in hypertensive patients and those with higher values should be subjected to retinopathy evaluations.

The following limitations can be seen in this study, we evaluated the retinopathy effects on P wave in hypertensive subjects and did not include patients with atrial arrhythmias. We did not observe any arrhythmias in the study population. So we do not know whether increased values of P wave dispersion predict atrial and/or ventricular arrhythmias in this population.

For this purpose, further large-scale prospective studies are needed to determine if retinopathy have clinical reflections of arrhythmias through increased P wave dispersion. Finally, manual measurement of P wave duration and P dispersion paper-printed ECG might have underpowered the results because it would be more reliable if measured on the high-resolution screen of a digital ECG system.

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