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Assessment of palpitation complaints in patients with benign paroxysmal positional vertigo

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Abstract. – OBJECTIVE: The detection of cardiac arrhythmias during vertigo attacks along with the resolution of vertigo by treating the arrhythmias is defined as cardiac vertigo. The cause of palpitations that develop following attacks of non-cardiac vertigo is unknown. We aimed to investigate the effect of benign paroxysmal positional vertigo (BPPV) on the cardiac conduction system and evaluate patients' palpitation complaints using 24-hour ambulatory ECG (Holter) monitoring.

PATIENTS AND METHODS: Sixty-five patients with BPPV and forty-eight healthy controls were included in the study. ECG was performed on all participants, and ECG parameters were measured. Holter monitoring was performed, and the recordings were analyzed. Evaluation of the time-domain heart rate variability (HRV) results was done.

RESULTS: The average age of the vertigo group was 49.1 ± 7.8 years. In terms of demographic or laboratory data, there was no statistically significant difference between the groups ($p>0.05$). The study group's ECG parameters, including Pd, TpTe, and TpTec, were longer, and the TpTe/QT and TpTe/QTc ratios were higher ($p<0.05$). Holter monitoring detected abnormal rhythms in 41 (63%) of the study group patients and three (6.2%) of the control group patients. The SDNN, LF, and LF/HF values, which are known as HRV markers, were significantly different in the vertigo group compared to the control group ($p<0.001$).

CONCLUSIONS: This study revealed that the risk of arrhythmias increased following attacks of BPPV in patients without a known history of cardiac vertigo.

Key Words:
Arrhythmia, QRS-T angle, Holter monitoring, Heart rate variability, Benign paroxysmal positional vertigo.

Introduction

The most common cause of vertigo in individuals admitted to the hospital with dizziness is BPPV[1]. While it occurs spontaneously in individuals between 50 and 70 years old, it develops after head trauma in younger people[2]. It is characterized by spontaneous resolution after a period of time, short-lasting attacks, and symptoms triggered by head movements[3].

In clinical practice, we typically encounter patients with vertigo with symptoms of palpitations and dizziness. Some patients complain of dizziness or vertigo after palpitations. Dizziness/vertigo develops due to cardiac arrhythmias[4]. On the other hand, some patients complain of palpitations after dizziness. It is not known whether the risk of arrhythmia develops during or after an attack of BPPV. ECG is a straightforward method for detecting cardiac arrhythmias. For these patients, ECG parameters can be used as non-invasive markers to predict arrhythmia risk[5]. Holter monitoring is the simplest method for excluding cardiac vertigo from arrhythmias caused by a BPPV attack.

This study aimed to investigate the influence of BPPV on the cardiac conduction system using ECG parameters and to assess the efficacy of ECG parameters in estimating arrhythmias using Holter recording.

Patients and Methods

Study Design and Subjects

The research data set consisted of patients diagnosed with vestibular disorders between 2018 and 2021 based on the Bárány Society's classification criteria (2015). Seventy patients over the age of 18 years who had sinus rhythm and Holter monitoring after diagnosis were enrolled. Five patients were omitted due to a lack of data. In all, 50 patients comprised the control group. Two participants in the control group who had vertigo were excluded from the research. Patients with

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structural heart disease, a history of arrhythmia, kidney disease, or electrolyte imbalance, and healthy volunteers in the control group with complaints and symptoms of dizziness were excluded from the study.

Study Protocol

A 12-lead ECG was performed with an electrocardiograph (model ECG-1350K, Kohden-Nihon Corporation) at a rate of 25 mm/s and 10 mm/mV amplitude. A scanner was used to computerize the existing ECGs. Two cardiologists calculated TpTe time under 400x magnification and analyzed it. A significant and almost perfect agreement was found between the analyses of the two cardiologists (κ=0.861). The Bazett formula was used to correct the QT interval and TpTe time for heart rate. These data were acquired directly since QRS, T axis, QT interval, and QTc interval were calculated automatically in the ECG analysis. Utilizing the difference between the QRS and T axis, the frontal QRS-T angle was computed. The P dispersion was calculated by minus the smallest P wave duration from the highest P wave duration[6]. Similarly, QT and QTc dispersions were calculated.

Standard 24-hour, three-leads (leads V1, V2, and V5) Holter ECG (Northeast Monitoring, MA) recordings were used to evaluate the patients. After manually adjusting R-R intervals, the records were analyzed using a Century 2000/3000 HRV system (Biomedical Systems, St. Louis, MO, USA). Throughout the recordings, the patients remained in a supine position and had fasted overnight. Intervals between normal and ectopic beats and between ectopic beats, as well as intervals assessed incorrectly due to artifacts, were eliminated from the analysis. The time-domain analysis examined the mean heart rate, the root means square of successive differences (RMSSD), the standard deviation of R-R intervals (SDNN), and the number of R-R intervals deviating by more than 50 ms from adjacent intervals divided by the total number of R-R intervals (pNN50). Applying the fast Fourier transform method, spectral measurements were obtained. Following the recommendations of the North American Society of Pacing and Electrophysiology and the Task Force of the European Society of Cardiology, the recordings were obtained at an average of five distinct five-minute intervals[7]. The total power was categorized as high frequency (HF) and low frequency (LF) components.

Statistical Analysis

All analyses were performed with the SPSS program version 24.0 (IBM, Armonk, NY, USA). The initial continuous variables were expressed as mean±standard deviation or median (interquartile range) according to the given data. Continuous variables were compared using Student's *t*-test or the Mann-Whitney U test. Categorical variables were presented as frequencies and percentages. Nominal variables were compared using the chi-squared test or Fisher's exact test. The statistical significance was defined as *p*-values <0.05.

Results

The average age of study individuals was 49.1 ± 7.8 years and the female patient population (63.1%) was nearly twice as large as the male population. The mean age of the control group was 48.5 ± 7.2 years. In terms of hypertension (0.38), diabetes mellitus (0.527), smoking (0.442), heart rate (0.07), systolic blood pressure (0.313), dyslipidemia (0.531), and ejection fraction (0.890), there was no significant difference between the groups. Regarding laboratory tests, there were no significant differences between the groups ($p>0.05$) (Table I). Pd, TpTe, and TpTec were longer, and TpTe/QT and TpTe/QTc ratios and frontal QRS-T angle were higher in the study group compared to the control group ($p<0.05$). Pmax, Pmin, QTd, and QTc values did not differ significantly between the groups (Table II). Holter monitoring of the vertigo patients revealed an abnormal ECG in 41 (63%) patients, while abnormal ECG findings were found in only three (6.2%) patients in the control group. The distribution of abnormal rhythms in the vertigo group was shown in Figure 1. In the vertigo group, SDNN was lower and LF and LF/HF were significantly higher than in the control group ($p<0.05$). There were no significant differences in pNN50 ($p=0.480$), RMSSD ($p=0.522$), or HF ($p=0.346$) between the groups (Table III).

Discussion

This study showed that the durations of Pd, TpTe, and TpTec were longer and TpTe/QT, and TpTe/QTc ratios and frontal QRS-T angle were higher in BPPV patients than in healthy controls, and ECG parameters had high predictive

Table I. Clinical characteristics and laboratory parameters of patients.

Parameters	Study group, n = 65	Control group, n = 48	*p*-value
Age (Years)	49.1 ± 7.8	48.5 ± 7.2	0.676
Gender, female, n (%)	41 (63.1)	29 (60.4)	0.773
SBP (mmHg)	125.3 ± 13.6	122.4 ± 16.8	0.313
Heart Rate (beat/min.)	95.4 ± 14.3	90.1 ± 16.0	0.070
Hypertension, n (%)	47 (72.3)	31 (64.6)	0.380
Diabetes mellitus, n (%)	13 (20)	12 (25)	0.527
DL, n (%)	24 (36.9)	15 (31.3)	0.531
Smoking, n (%)	10 (15.4)	5 (10.4)	0.442
White blood cell count ($\times 10^3$ µL)	9.9 ± 3.8	9.3 ± 2.7	0.323
Hemoglobin (g/dl)	13.7 ± 2.2	13.2 ± 2.0	0.230
ALT (U/L)	18 (14.5)	16.5 (16)	0.235
AST (U/L)	20 (9.5)	18 (9.5)	0.533
CRP (mg/L)	0.4 (0.4)	0.32 (0.3)	0.539
Glucose (mg/dl)	87.0 ± 19.5	87.9 ± 20.4	0.808
Creatine (mg/dl)	0.98 ± 0.31	0.94 ± 0.19	0.430
Total cholesterol (mg/dl)	179.8 ± 47.1	175.1 ± 32.3	0.554
Triglyserides (mg/dl)	158.9 ± 69.0	151.7 ± 55.6	0.557
LDL (mg/dl)	112.0 ± 30.9	103.6 ± 28.5	0.145
HDL (mg/dl)	40.3 ± 8.5	39.9 ± 8.3	0.821
EF %	59.4 ± 3.5	59.2 ± 2.8	0.890

Data are expressed as appropriate as mean ± SD and median [interquartile range]. SBP: Systolic blood pressure, DL: dyslipidemia, ALT: alanine aminotransferase, AST: aspartate aminotransferase, CRP: C-reactive protein LDL: low-density lipoprotein, HDL: high-density lipoprotein, EF: Ejection fraction.

power for arrhythmia on Holter monitoring. Additionally, patients with BPPV had significant autonomic nervous system dysfunction in favor of the sympathetic system compared to the control group.

Cardiogenic vertigo is frequently observed as a complication of cardiovascular diseases, especially in patients with rhythm disorders[8]. The number of patients without cardiovascular disease who complained of palpitations during or after a BPPV attack was not small. Using non-invasive methods, the development of arrhythmia can be predicted.

Paroxysmal atrial fibrillation (PAF) can be predicted independently by Yavuzkır et al[10] in patients with RA, Ahlehoff et al[11] in psoriasis, and Dogan et al[9] in patients with Behcet's disease reported that the Pd owing to inflammation was lengthened, and the risk of atrial fibrillation was raised[9-11]. Scholars[12] have shown that with

Table II. Comparison of ECG parameters between groups.

ECG parameters	Study group, n = 65	Control group, n = 48	*p*-value
Pmax (ms)	120.6 ± 6.5	118.7 ± 7.9	0.152
Pmin (ms)	72.4 ± 8.7	73.3 ± 11.3	0.662
Pd (ms)	51.1 ± 7.6	45.8 ± 11.6	0.014
QTd (ms)	53.2 ± 5.9	51.0 ± 7.5	0.083
QTcd (ms)	67.9 ± 8.1	65.2 ± 9.4	0.104
TpTe (ms)	75.6 ± 18.1	65.8 ± 14.4	< 0.001
TpTec (ms)	81.3 ± 3.1	75.5 ± 2.8	< 0.001
TpTe/QT	0.2 ± 0.04	0.18 ± 0.02	< 0.001
TpTe/QTc	0.19 ± 0.05	0.17 ± 0.03	< 0.001
Heart rate (beats/min)	72 ± 5	74 ± 3	0.590
Frontal QRS-T angle	48 ± 17	38 ± 11	< 0.001
Abnormal ECG rhythm, n (%)	41 (63)	3 (6.2)	< 0.001

Values are presented as mean ± SD and median [interquartile range]. Pmax: P-wave maximum duration, Pmin: P-wave minimum duration, Pd: P-wave dispersion, QTd: QT dispersion, QTcd: Corrected QT dispersion, TpTe: T-peak to T-end, TpTec: Corrected TpTe.

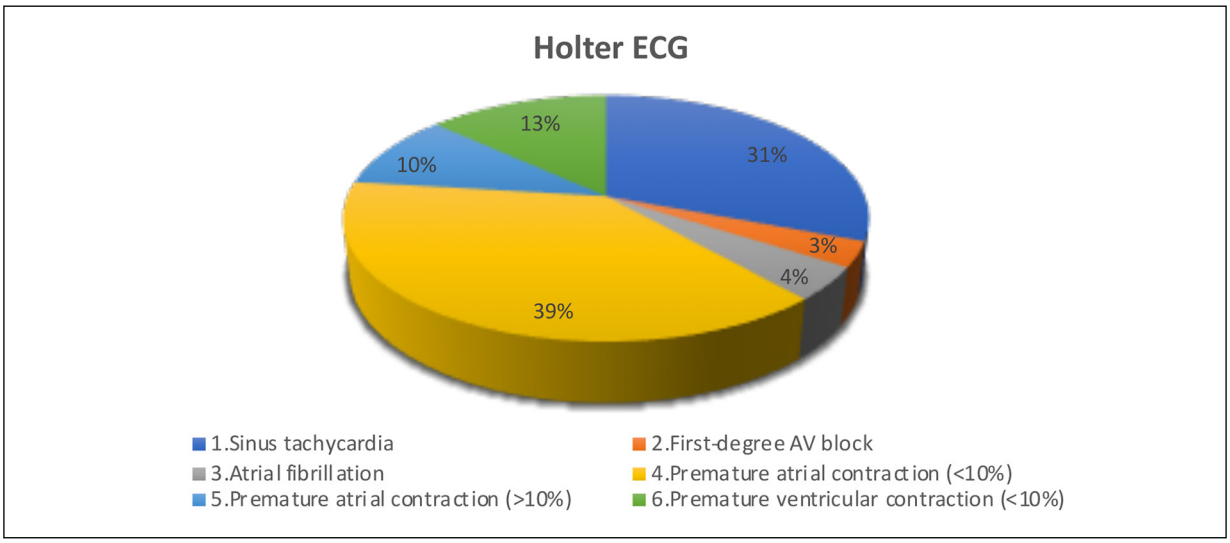

Figure 1. Abnormal rhythms in the vertigo group.

prolonged QTd, ventricular arrhythmias are triggered, and sudden cardiovascular death develops. In rheumatic disorders, such as rheumatoid arthritis, Behcet's disease, systemic sclerosis, and systemic lupus erythematosus, QTd was shown to be lengthy[13]. Electrolyte imbalance, heart failure, myocardial infarction, drug toxicity, anti-arrhythmic drugs, hypertrophic cardiomyopathy, and heart failure are other causes that prolonged QTd[14]. Therefore, these diseases were excluded from this study.

Several ventricular repolarization metrics were also evaluated, including TpTe time, TpTe/QT ratio, and QRS-T angle. These parameters were found to be valuable markers for the distribution of ventricular repolarization. Yıldırım et al reported that TpTe time and TpTe/QT ratio were considerably higher in BPPV patients[15]. Slightly more than 90 ms of TpTec was associated with an almost threefold higher risk of sudden cardiac death[16]. In comparison to electrocardiographic risk indicators such as the QT period, the QRS-T angle has been identified as a powerful and independent risk predictor for cardiac morbidity and mortality[17]. While Hanutkova et al[18] determined the threshold value of the QRS-T angle to be 75-degree, Portland et al[19] calculated approximately 100-degree. In our study, the threshold value of the QRS-T angle was not determined because of the absence of mortality or sudden cardiac death. According to our findings, this parameter indicates that in patients with BPPV, ventricular heterogeneity and the likelihood of catastrophic ventricular arrhythmia may increase.

Holter monitoring is the easiest method for determining if arrhythmia risk arises in patients with prolonged ECG parameters[20]. In Holter mon-

Table III. Heart rate variability parameters of groups.

	Study group, n = 65	Control group, n=48	*p*-value
SDNN (ms)	102.6 ± 43.2	140.3 ± 18.9	< 0.001
RMSSD (ms)	32.6 ± 22.5	28.7 ± 8.9	0.522
pNN50 (%)	10.6 ± 9.2	8.0 ± 5.3	0.480
LF (nu)	67.6 ± 15.6	53.8 ± 12.4	< 0.001
HF (nu)	27.2 ± 11.2	25.2 ± 7.6	0.346
LF/HF	3.54 ± 1.8	2.24 ± 1.3	0.003

Data are expressed as mean ± SD as appropriate. SDNN: Standard deviation of the NN (R-R) intervals, RMSSD: Root mean square of the successive differences, pNN50: The proportion of NN50 divided by the total number of NN (R-R) intervals, LF: Low frequency, HF: High frequency.

itoring, atrial arrhythmias were high in the BPPV group. Sinus tachycardia (31%), first-degree AV block (3%), atrial fibrillation (4%), premature atrial contraction (49%), and premature ventricular contraction (13%) were observed in the study group.

Patients with vertigo have impaired autonomic nerve function[21]. PAC arrhythmias can develop after BPPV as a result of autonomic dysfunction induced by excessive catecholamine release. Karataylı-Ozgursoy et al reported that Takotsubo cardiomyopathy develops in these patients[22]. Post-treatment anxiety is common in patients with BPPV. This may lead to the development of post-anxiety arrhythmia. However, it should be differentiated from an arrhythmia caused by BPPV due to anxiety[23,24]. After treatment, patients with residual BPPV frequently exhibit sympathoneural dysfunctions. Residual BPPV may be related to autonomic dysfunction of the sympathoneural system.

HRV is a useful indicator of the equilibrium between sympathetic and parasympathetic activity[25]. It is shown that HF, RMSSD, and pNN50 are markers of parasympathetic activity, and LF and SDNN are markers of sympathetic activity[26]. In vertigo diseases, severe studies have shown a significant increase in HRV parameters[27]. In our study, pNN50 and RMSSD increased somewhat, but SDNN decreased significantly in the study group (p=0.480, p=0.522, and p<0.001). We observed a reduction in HF and a significant rise in LF and LF/HF ratio in BPPV patients, indicating that sympathetic regulation of the heart is predominant.

There are some limitations in our study. The study population was relatively small. Holter ECG recording of our device lasted only 24 hours. In the future, an implantable loop recorder could be used. Electrophysiological studies were not conducted. The effects of the therapeutic agents on the cardiac conduction system were not evaluated and rhythm follow-up of the patients was not performed after the treatment.

Conclusions

Our study found that BPPV affects the cardiac conduction system and disturbs the autonomic nervous system in favor of the sympathetic system. However, we observed that prolonged ECG parameters on Holter monitoring posed a risk of arrhythmias post-attack of BPPV in patients without a known history of cardiac vertigo. Therefore, it should be recognized that BPPV increases the risk of arrhythmias. We can prevent the development of cardiac complications with early treatment.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Ethics Approval

The local ethics committee approved the study protocol (Gazi Yaşargil Training and Research Hospital; no. 2022-99, June 10, 2022), and it followed the Declaration of Helsinki's ethical guidelines for human testing (2013).

Availability of Data and Materials

Data are available upon reasonable request to the corresponding author. De-identified data might be available after the consent of all authors and the privacy policy of the Dagkapı State Hospital.

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Authors' Contribution

Conceptualization; Serhat Günlü (SG), Adem Aktan (AA); Data curation; SG, AA; Formal analysis; SG, AA; Funding acquisition; SG, AA; Investigation; SG, AA; Methodology; SG, AA; Project administration; SG, AA; Resources; SG, AA; Software; SG, AA; Supervision; SG, AA; Validation; SG, AA; Visualization; SG, AA; Roles/Writing - original draft; SG, AA; Writing - review & editing; SG, AA.

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