Original Article

Evaluation of the Heart Rate Variability in Cardiogenic Vertigo Patients

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Abstract

Background: The detection of documented cardiac arrhythmias during vertigo attacks and vertigo resolution through the treatment of arrhythmias is defined as cardiogenic vertigo (CV). **Aim and Objective:** We evaluate the impact of arrhythmias on vertigo and assess the types of arrhythmias and heart rate variability using 24-h ambulatory electrocardiogram (Holter) monitoring. **Patients and Methods:** The study included 70 patients with CV. Demographic, clinical, and laboratory data were analyzed. Holter monitoring and echocardiography were conducted, and the results were analyzed. **Results:** The average age of the patient group was 46.7 ± 8.6 years, and the number of female patients (65.7%) was almost twice that of the number of male patients. Clinical features, such as thyroid dysfunction (4.2%), diabetes mellitus (22%), dyslipidemia (27.1%), hypertension (34%), iron deficiency anemia (5.7%), and smoking status (21.4%), were identified. Vertigo manifested in the presence of syncopal episodes in 18 (25.7%) patients, and 47 (67.1%) patients presented with spinning during the episodes. Most of the vertigo attacks were induced by standing in 27 patients (38.6%) and lasted for only a few seconds in 52 (74.2%) patients. In terms of the Holter monitoring records, tachycardia-bradycardia was the syndrome that was observed the most. The computed average low-to-high frequency ratio was 2.24 ± 1.6. **Conclusion:** This study showed that arrhythmias could increase the risk of vertigo attacks in patients with no known history of heart disease. The patients had significant autonomic nervous system dysfunction in favor of the sympathetic system.

Keywords: Arrhythmia, heart rate variability, Holter monitoring, syncope, vertigo

Introduction

Vertigo is the most typical cause of dizziness brought on by cardiovascular diseases.[1] Vertigo can sometimes be accompanied by syncope. Cardiovascular disorders are the second most frequent cause of syncope.[2] Since cardiac syncope causes morbidity and mortality, the underlying causes need to be treated.[3]

If vertigo attacks are accompanied by syncope, diagnosing cardiogenic vertigo (CV) can be difficult.[4] Short recurrent rotatory vertigo may be the first presentation of arrhythmia.[5] When this condition is accompanied by syncope, serious problems can occur, ranging from trauma to cardiac death.

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In this study, we assessed the clinical characteristics of patients with CV caused by arrhythmias and evaluate the types of arrhythmias and heart rate variability (HRV) using Holter monitoring.

Patients and Methods

Selection of participants

The study was conducted retrospectively between 2018 and 2022. The study comprised patients who were referred to cardiology with dizzy complaints by otolaryngologists, neurologists, and psychiatrists. Recurrent episodes of spontaneous reversion or nonreturning vertigo, onset

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over 18 years of age, vertigo duration <1 min, documentation of heart diseases during an attack of vertigo, response to appropriate treatments for heart disease, and inability to be better explained by another diagnosis were defined as diagnostic criteria of CV according to the classification of the Bárány Society (2015). Patients with valvular heart disease, coronary artery disease, cardiomyopathy, acute coronary syndrome, using beta-blockers due to arrhythmia, hypertension and diabetes patients who are unstable despite drug use, no vertigo attack during rhythm recording, and whose data could not be accessed were excluded from the study. Seventy patients who had been diagnosed with CV were included in the study.

Study protocol

Routine blood tests were performed, along with echocardiography using a Philips brand ultrasonography device (Model HD7 X E). The patients were also evaluated by a cardiologist who was not aware of the study by performing a 24-h standard 3-channel (leads V1, V2, and V5) Holter electrocardiogram (ECG). After manually adjusting the intervals of consecutive R waves, all recordings were analyzed using a Century 2000/3000 hV Package System. According to the North American Society of Pacing and Electrophysiology, the recordings were taken as the mean of five different 5-min periods.[6] Low frequency (LF) (0.04–0.15 Hz) and high frequency (HF) (0.16–0.4 Hz) components were defined as the power between 0.003 MHz and 0.40 Hz on the heart rate spectrum.

Ethical statement

The study protocol was authorized by the Local Ethics Committee (Gazi Yasargil Training and Research Hospital; no. 2022-109, June 11, 2022), and it followed the Declaration of Helsinki's ethical guidelines for human testing (2013).

Statistical methods

All analyses were carried out using the SPSS program (version 24.0, Chicago, IL, USA). According to the data distribution, the initial continuous variables are expressed as mean ± standard deviation (SD) or median (interquartile range). Frequencies and percentages are used to express categorical variables. The Kolmogorov–Smirnov or Shapiro–Wilk tests were used to determine whether the variables' distributions were normal. To compare continuous variables, the Student's *t*-test or the Mann–Whitney *U* tests were employed, and to compare nominal variables, the Chi-squared or Fisher's exact tests were used. The level of significance was set at $P < 0.05$.

Results

The average age of the patient group was 46.7 ± 8.6 years, with nearly twice as many female patients (65.7%) as male patients. Clinical features, such as thyroid dysfunction (4.2%), diabetes mellitus (22%), dyslipidemia (27.1%), hypertension (34%), iron deficiency anemia (5.7%), and smoking status (21.4%) were identified. The mean values for left atrium diameter 3.64 ± 0.19 cm, left ventricle diastolic diameter 4.89 ± 0.31 cm, left ventricle systolic diameter 2.53 ± 0.40 cm, and ejection fraction 61.8% ±5.5% were measured during the echocardiographic examinations. Measurements were also taken for hemoglobin 13.3 ± 2.4 g/dl, glucose 105.1 ± 34.2 mg/dl, fe 64.8 ± 28.7 ml/ng, thyroid-stimulating hormone 2.39 ± 1.1 mIU/L, free triiodothyronine 3 2.49 ± 1.0 pg/ml, low-density lipoprotein 112.0 ± 30.9 mg/dl, and high-density lipoprotein 40.3 ± 8.5 mg/dl [Table 1]. Vertigo occurred without syncopal attacks in 18 (25.7%) patients. The duration of the symptoms of patients with recurrent isolated vertigo ranged from days (22.9%) to years (8.6%), and 47 (67.1%) patients presented with spinning during the episodes. The majority of the vertigo attacks were induced by standing (*n* = 27; 38.6%), fatigue (*n* = 16; 22.9%), walking (*n* = 16; 22.9%), and eating (*n* = 11; 15.7%). Palpitations (*n* = 48; 68.6%), arm twitching (*n* = 11; 15.7%), and dyspnea (*n* = 11; 15.7%) were found to be an associated symptoms [Table 2]. Figure 1 shows the Holter-ECG findings in patients with vertigo. For the HRV, SD of the NN (R-R) intervals (SDNN) 103.4 ± 46.7 ms, root mean square of the successive differences (RMSSD) 33.7 ± 23.5 ms, the proportion of NN50 divided by the total number of NN (R-R) intervals (pNN50) 10.7% ±8.6%, LF 67.3 ± 17.5 nu, HF 31.7 ± 9.2 nu, and LF/HF 2.24 ± 1.6 were measured [Table 3].

Table 1: Clinical characteristics, echocardiography, and laboratory test of vertigo patients

Parameters	Case (*n*=70), *n* (%)
Age	46.7±8.6
Sex (female)	46 (65.7)
Thyroid dysfunction	3 (4.2)
Diabetes mellitus	16 (22)
DL	19 (27.1)
Hypertension	24 (34)
Iron deficiency anemia	4 (5.7)
Smoking	15 (21.4)
Echocardıography	
LAD (cm)	3.64±0.19
LVDD (cm)	4.89±0.31
LVSD (cm)	2.53±0.40
EF (%)	61.8±5.5
Laboratory	
Hemoglobin (g/dl)	13.3±2.4
Glucose (mg/dl)	105.1±34.2
Fe (ml/ng)	64.8±28.7
TSH (mIU/l)	2.39±1.1
fT3 (pg/ml)	2.49±1.0
LDL (mg/dl)	112.0±30.9
HDL (mg/dl)	40.3±8.5

Values are presented as mean±SD as appropriate. DL: Dyslipidemia, LAD: Left atrium diameter, LVDD: Left ventricle diastolic diameter, LVSD: Left ventricle systolic diameter, EF: Ejection fraction, TSH: Thyroid-stimulating hormone, fT3: Free triiodothyronine, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, SD: Standard deviation

Table 2: Characteristics of vertigo patients

Vertigo	Case (n=70), n (%)
Spinning nature	47 (67.1)
Syncope	18 (25.7)
Frequency	
Day	16 (22.9)
Week	42 (60)
Month	6 (8.6)
Year	6 (8.6)
Duration	
3-5 s	27 (38.6)
5-10 s	25 (35.7)
>10 s	9 (12.9)
A few minute	9 (12.9)
Trigger factors	
Eating	11 (15.7)
Standing	27 (38.6)
Fatigue	16 (22.9)
Walking	16 (22.9)
Accompanying symptoms	
Palpitation	48 (68.6)
Arm twitching	11 (15.7)
Dyspnea	11 (15.7)

Table 3: Heart rate variability of vertigo patients

	Case (n=70)
SDNN (ms)	103.4±46.7
RMSSD (ms)	33.7±23.5
pNN50 (%)	10.7±8.6
LF (Nu)	67.3±17.5
HF (Nu)	31.7±9.2
LF/HF	2.24±1.6

Values are expressed as mean±SD as appropriate. SD: Standard deviation, SDNN: SD 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

Discussion

This study revealed that patients suffering from arrhythmias can develop vertigo, which is generally induced by standing. In addition, the patients had significant autonomic nervous system dysfunction in favor of the sympathetic system.

Vertigo occurs at a rate of 2.4% throughout life and accounts for 17.1% of all complaints of dizziness in the general population but nearly half of the complaints of dizziness in the elderly.[7,8] It develops mainly after arrhythmia in younger people.[9] The number of patients without cardiovascular disease who complained of palpitations during vertigo attacks is not small. The importance of early CV diagnosis in decreasing morbidity and mortality from arrhythmia complications such as cardiac arrest, syncope, and embolic stroke is crucial.[10]

When structural heart diseases are excluded, problems such as underlying cardiac conduction system diseases, diabetes,

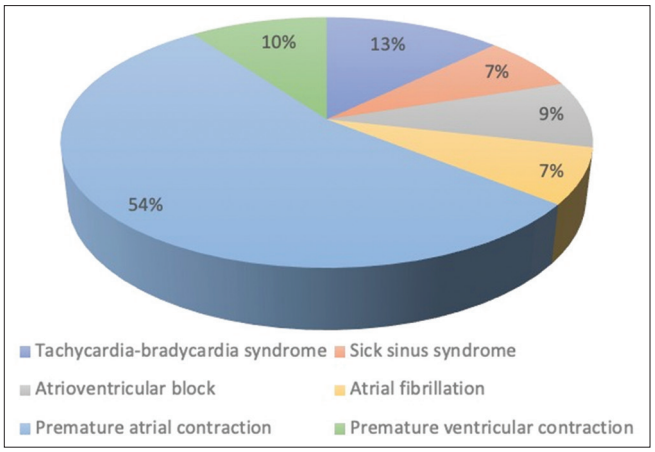

Figure 1: Holter ECG findings in patients with vertigo. ECG = Electrocardiogram

thyroid dysfunction, and iron deficiency may predispose the patient to arrhythmia. When the heart does not provide enough cardiac output, the brain suffers from hypoperfusion and malfunctions, resulting in syncope. In bradyarrhythmias, the heart beats too slowly to generate enough flow, resulting in cerebral hypoperfusion, whereas tachyarrhythmias cause the heart to beat too fast, resulting in inadequate ventricular filling and poor cardiac output.[11] We can expect isolated vertigo without loss of consciousness as a result of the temporary or moderate reduction in brain perfusion induced by arrhythmias.[12] CV can develop in isolation or with syncope, depending on a variety of factors including the duration or severity of brain hypoperfusion, individual differences in the cerebral vasculature, and individual vulnerability to decreased perfusion.

The attacks were generally spontaneous, and vertigo could be also provoked by standing, fatigue, walking, or eating. Other cardiac or neurological symptoms, such as palpitation, arms twitching, and dyspnea, are frequently associated with CV.[13] Remarkably, CV lasted only a few seconds or minutes, meeting the diagnostic criteria for vestibular paroxysmia.[14] In our study, most of the vertigo attacks were induced by standing in 27 patients (38.6%) and lasted for only a few seconds in 52 (74.2%) patients.

Holter monitoring is the most basic method for determining whether a particular type of arrhythmia causes vertigo. In our study, 54% premature atrial contraction, 10% premature ventricular contraction, 7% sick sinus syndrome, 9% atrioventricular block, 13% tachycardia-bradycardia syndrome, and 7% atrial fibrillation were observed. After COVID-19 infection, premature atrial contraction arrhythmias may occur as a consequence of autonomic dysfunction caused by excessive catecholamine release. Malayala *et al*.[15] reported that patients with COVID-19 developed vertigo due to arrhythmia a few weeks after treatment.

Sympathoneural dysfunctions are frequent in patients with posttreatment vertigo. Residual vertigo could be caused

by sympathoneural autonomic dysfunction.[16] HRV can be used to detect sympathetic-parasympathetic balance in the autonomic nervous system.[17] It is shown that HF, RMSSD, and pNN50 are parasympathetic activity markers, whereas LF and SDNN are sympathetic activity markers.[18] Hayano *et al*.[19] reported that fatal arrhythmias developed in patients with SDNN <50 ms. SDNN <70 ms in patients undergoing percutaneous coronary intervention was associated with premature contractions.[20] In some studies, RMSSD values of more than 28 ms and pNN50 values of more than 5.5% were associated with bradycardia.[21,22] Increased cardiac sympathetic activity was reported to be associated with high LF and LF/HF ratios.[23] In our study, tachycardia-bradycardia syndrome and high LF/HF ratio were observed.

Variations in HRV time (pNN50, RMSSD, SDNN) and frequency (LF, HF) domain indices have frequently been seen in chronic diseases, such as diabetes and hypertension, and have been linked to cardiac autonomic dysfunction.[24] A reduction in HRV is a measure of decreased parasympathetic and increased sympathetic tone and has long been considered to be having a detrimental effect on the prognosis of cardiovascular disease.[25] Sympathetic overactivation and autonomous imbalance play important roles in the pathogenesis of hypertension.[26] Abnormal HRV was demonstrated in hypertensive patients in previous studies.[27] Sheng *et al*.[28] found that the LF/HF ratio was higher in white-coat patients than in sustained hypertension patients. Menezes Ada *et al*.[29] reported that LF power decreased and LF/HF increased before treatment and regressed to normal after treatment. After 12 months of anti-hypertensive medication, Maciorowska *et al*.[30] observed a considerable improvement in SDNN, pNN50, and RMSDD. On the other hand, excessive autonomic fluctuation, represented by higher HRV in patients with hypertension, was associated with an increased risk of atrial fibrillation.[31] It was observed at a low rate (7%) in our study. Controlling hypertension has a beneficial effect on HRV and could prevent arrhythmias.

In a meta-analysis, patients with diabetes were found to have considerably lower HRV values.[32] Patients who were exercising and under the control of medications demonstrated higher HRV values.[21] Agashe and Petak.[33] reported that autonomic dysfunction in diabetes patients can be determined more easily by calculating the frequency domains of HRV. Martinez and Okoshi.[34] did not include healthy controls in their study. They suggested that there would be no significant differences in terms of HRV time domain between the patients under treatment and the healthy controls. Patients' frequency domain of HRV was only calculated. As a result, they defined an LF/HF ratio >1 as a sympathetic system dysfunction. In our study, this ratio was found to be higher.

Study limitations

Our study was retrospective and relatively small. Holter monitoring was only 24-h. In the future, an implantable loop recorder could be used to identify whether there are different rhythms during vertigo attacks. Electrophysiological studies were not conducted. Cardiac magnetic resonance imaging was not performed. In addition, the absence of control patients is one of the limitations of this study.

Conclusion

This study showed that arrhythmias could increase the risk of vertigo attacks in patients with no known history of heart disease. Standing-induced attacks were the most and lasted only a few seconds. The patients had significant autonomic nervous system dysfunction in favor of the sympathetic system. Additional studies are needed to determine the role of autonomic nerve dysfunction as a prognostic indicator in patients with CV.

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Conflicts of interest

There are no conflicts of interest.

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