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Evaluation of ventricular repolarization parameters during migraine attacks

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ABSTRACT

Aims: Migraine is a chronic neurovascular disorder characterized by intermittent episodes of severe headache. Abnormalities in the autonomic nervous system (sympathetic and parasympathetic nervous systems) have been detected during migraine-free periods in patients with migraine. In these patients, disrupted autonomic innervations of the heart and coronary arteries may lead to electrocardiographic changes during a migraine attack. T-wave peak-to-end interval (Tp-e interval) and Tp-e/QT ratio are relatively new markers of ventricular arrhythmogenesis and repolarization heterogeneity. In the present observational study, we investigated the changes in ventricular repolarization during migraine attacks and attack-free periods by performing 12-lead electrocardiography (ECG).

Methods: This study included 63 patients (54 [86%] women; mean age: 33.3 ± 9.9 years) with migraine. The QT and corrected QT (QTc) intervals, Tp-e interval, and Tp-e/QT ratio of the patients during migraine attacks and attack-free periods were measured by performing 12-lead ECG.

Results: The QT and QTc intervals, Tp-e interval, and Tp-e/QT ratio were higher during migraine attacks than during attack-free periods ($P < 0.001$ for all).

Conclusion: These results indicate that migraine attacks are associated with an increase in ventricular repolarization parameters compared with attack-free periods possibly because of the dysregulation of the autonomic nervous system.

Introduction

Migraine is a neurological disorder characterized by recurrent episodes of severe headache that affect various parts of the body, including the circulatory tract, gastrointestinal tract, and autonomic nervous system (ANS) [1].ANS dysregulation is suggested to be involved in the pathophysiology of migraine because autonomic symptoms such as nausea, vomiting, headache, and photophobia are common during acute migraine attacks. The symptoms associated with ANS dysregulation may be caused by an imbalance between the sympathetic and parasympathetic nervous systems [2] that may induce some changes in ventricular repolarization during migraine attacks in patients with migraine [3].Some studies have reported prolonged PR and corrected QT (QTc) intervals during acute migraine attacks [4]. Recent studies have defined some electrocardiographic parameters for assessing the elevated risk of ventricular arrhythmias in patients with acute migraine; however, it is unclear as to which of these parameters are suitable for these patients.

T-wave is one of the most important components of ventricular repolarization on an electrocardiogram. Increased transmural dispersion of ventricular repolarization in the normal heart is associated with cardiac arrhythmias. Recent studies have reported that some ventricular repolarization markers, such as QT and QTc intervals, T-wave peak-to-end (Tp-e) interval, and Tp-e/QT ratio, are useful for predicting life-threatening cardiac arrhythmias in patients with various clinical disorders but without a structural heart disease [5]. Some studies have also reported that increased Tp-e interval and Tp-e/QTc ratio are associated

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an elevated risk of malignant ventricular arrhythmias [5–8]. Any increase in the Tp-e interval increases the risk of malignant ventricular arrhythmias; moreover, this new parameter shows increased correlation with the distribution of ventricular repolarization compared with the QT and OTc intervals [9,10].

Cardiac arrhythmias have been reported in several patients with migraine [11,12]. However, data concerning the changes in the Tp-e interval and Tp-e/QT ratio during migraine attacks are limited. In addition, limited information is available on the increase in QT and QTc intervals during migraine attacks compared with that during attack-free periods. We hypothesized that the Tp-e interval and Tp-e/QT ratio increased in patients with migraine because of ANS dysregulation. Thus, the present study investigated the increase in the Tp-e interval and Tp-e/QT ratio during migraine attacks.

Methods

Study design

This is a cross-sectional and observational study.

Study population

This cross-sectional study included 63 patients (54 [86%] women; mean age: 33.3 ± 9.9 years) with migraine and without any cardiovascular involvement who were diagnosed according to the International Classification of Headache Disorders criteria [13] by an experienced physician between July 2016 to June 2017 at the outpatient headache clinic of the Department of Neurology. Patients with diabetes mellitus, hypertension, congestive heart failure, smoking habit, coronary artery disease, valvular heart disease, history of myocardial infarction, hyperthyroidism, hypothyroidism, atrial fibrillation, chronic kidney disease, chronic obstructive pulmonary disease, bundle branch block, atrioventricular block, and malignancy and who took migraine-specific medication during migraine attacks were excluded from the study. All the patients had a normal sinus rhythm and did not take medications, such as antiarrhythmics, tricyclic antidepressants, and antipsychotics. Each patient underwent 12-lead electrocardiography (ECG) within the first 30 min after emergency department admission and was monitored in the emergency department until the elapse of a migraine attack-free period. Electrocardiograms of the patients in the attack-free period were obtained at least 24 h after a migraine attack. The patients were asked to not consume alcohol or caffeinated beverages for 3 h before undergoing ECG. Blood glucose, sodium, potassium, magnesium, and calcium levels of the patients were determined upon their admission into the emergency department. The study protocol was approved by the institutional ethics committee, and informed written consent was obtained from each patient.

Electrocardiographic and echocardiographic examinations

The 12-lead ECG examination (10 mm/mV and 25 mm/s; Cardiofax V; Nihon Kohden Corp., Tokyo, Japan) was performed during an acute migraine attack (within the first hour of migraine onset) and an attack-free baseline period (at least 24 h after the attack) by resting the patients in the supine position. The obtained electrocardiograms were transferred to a computer by using a scanner and were examined at ×400% magnification by using Adobe Photoshop CS2 software (Adobe Systems Inc., San Jose, California, USA). The Tp-e and QT intervals were measured on the computer by two experienced cardiologists who were blinded to the clinical data of each patient.

QT and R-R intervals were obtained from leads V2 and V5. The QT interval was defined as the time from the start of QRS to a point at which the T-wave returned to an isoelectric line. The R-R interval was measured as an average of three complexes and was used to calculate the heart rate and QTc interval with Bazett's formula. Patients with U-waves and low-amplitude T-waves in their electrocardiograms were excluded from the study.

Although the Tp-e interval can be measured using both tail and tangent methods, the tail method is a better predictor of mortality than the tangent method [14]. Therefore, was used the tail method in the present study. This method defines the Tp-e interval as the time from T-wave peak to T-wave end to a point where it reaches the isoelectric line [15]. The Tp-e interval was measured from the leads V2 and V5, and the Tp-e/QT ratio was calculated from these measurements. All the measurements were made by two independent cardiologists by averaging three consecutive beats. For the Tp-e interval measured from the leads V2 and V5, intraobserver variability was 3.4% and 3.8%, respectively, and interobserver variability was 2.5% and 2.9%, respectively.

All echocardiographic examinations were performed using a 2.5–3.5 MHz transducer (General Electric Vivid S5, Milwaukee, WI, USA) by resting the patients in the left decubitus position. Two-dimensional and pulsed Doppler measurements were obtained using the criteria defined by the American Society of Echocardiography [16]. Left ventricular ejection fraction (LVEF) was assessed using Simpson's method.

Statistical analysis

All statistical analyses were performed using SPSS software (SPSS 18.0 for Windows Inc., Chicago, IL, USA). Categorical variables are expressed as n (%), and continuous variables are expressed as mean ± standard deviation. Eligibility of data for normal distribution was evaluated using Kolmogorov–Smirnov test. Relative differences between acute migraine attacks and attack-free periods for each electrocardiographic parameter were evaluated using a paired-sample *t*-test. Differences between the categorical variables were compared using chi-square test. $P < 0.05$ was considered statistically significant.

Results

This study included 63 patients (54 women and 9 men; mean age: 33.3 ± 9.9 years). The basic clinical features of the patients are shown in Table 1. Patients had a history of irregular medication for the treatment of migraine attacks. Drugs used by the patients for treating migraine attacks previously are listed in Table 1. In all, 36 (57%) patients were on prophylactic medication for migraine. However, none of the patients had migraine specific medication during the last attack period. Blood pressure of the patients was not different between migraine

Table 1
Baseline characteristics, laboratory and echocardiographic parameters among patients with migraine (n = 63).

Age (mean, years)	33.3 ± 9.9
Women (n, %)	54 (86.4)
BMI (kg/m^2)	24 ± 2.5
Glucose (mg/dl)	87.8 ± 6.5
Sodium (mmol/l)	140 ± 2.2
Potassium (mmol/l)	4.5 ± 0.4
Calcium (mg/dl)	9.6 ± 0.3
Magnesium (mg/dl)	1.9 ± 0.15
Creatinine (mg/dl)	0.68 ± 0.1
IVS (mm)	9.9 ± 1.1
PW (mm)	8.7 ± 0.9
LA (mm)	30.2 ± 1.8
LVSD (mm)	29.3 ± 2.8
LVDD (mm)	44.5 ± 2.5
Ejection fraction (%)	65.3 ± 3.8
Previous medications (n, %)	
Eletriptan	14 (22%)
Sumatriptan	6 (9%)
NSAID	25 (40%)

BMI: Body mass index, IVS: interventricular septum, PW: posterior wall, LA: left atrium, LVSD: left ventricle systolic diameter, LVDD: left ventricle diastolic diameter, NSAID: non-steroidal anti-inflammatory drug.

Table 2
Electrocardiographic data in patients with and without migraine attack.

	Attack-free period	Migraine attack	P
Heart rate (beat/min)	75.3 ± 10.6	78.7 ± 12.6	0.141
Lead V2			
QT (ms)	323.8 ± 18.6	339.9 ± 20.1	<0.001
QTc (ms)	368.7 ± 19.7	391.9 ± 23.8	<0.001
Tp-e (ms)	75.8 ± 8.1	86.5 ± 7.7	<0.001
Tp-e/QT	0.23 ± 0.02	0.25 ± 0.02	<0.001
Lead V5			
QT (ms)	322.0 ± 21.4	337.3 ± 22.2	<0.001
QTc (ms)	366.3 ± 15.5	387.9 ± 20.1	<0.001
Tp-e (ms)	70.5 ± 7.4	79.1 ± 8.6	<0.001
Tp-e/QT	0.21 ± 0.02	0.23 ± 0.02	<0.001

QTc: Corrected QT, Tp-e: T wave peak-to-end interval.
Data are shown as mean ± SD (n = 63).

attacks and attack-free periods. Serum levels of electrolytes, such as sodium, potassium and calcium, which may affect ventricular repolarization, were within normal range. Creatinine; fasting serum glucose; and total, low-density, and high-density cholesterol levels were also in normal range. Echocardiographic examination showed that LVEF, left atrial and ventricular systolic and diastolic diameters, and interventricular septum and posterior wall thicknesses were in normal range.

Electrocardiographic parameters of the patients are shown in Table 2. The values of the QT interval (323.8 ± 18.6 vs. 339.9 ± 20.1 millisecond [ms]), QTc interval (368.7 ± 19.7 vs. 391.9 ± 23.8 ms), and Tp-e interval (75.8 ± 8.1 vs. 86.5 ± 7.7 ms) (Fig. 1) were significantly higher during migraine attacks than during attack-free periods ($P < 0.001$ for all the parameters). However, no significant difference was observed in heart rate between migraine attacks and attack-free periods.

Discussion

This prospective cohort study is the first to show that the Tp-e interval and Tp-e/QT ratio increase during migraine attacks compared with those during attack-free periods. In addition, the present study showed that the QT and QTc intervals were significantly higher during migraine attacks than during attack-free periods.

Migraine is associated with several symptoms that affect the circulatory tract, gastrointestinal tract, and central nervous system. Clinical studies have investigated ANS dysfunction in patients with migraine. However, majority of these studies do not provide clear information on the possible changes in the ANS of these patients. Several studies have reported sympathetic hypofunction [17], sympathetic instability or hyperfunction [18], and/or parasympathetic dysfunction [19] in patients with migraine. The complex autonomic innervations of the heart play an important role in regulating cardiovascular functions. For example, an increase in sympathetic tonus may shorten the QT interval and sinus tachycardia. In contrast, an increase in parasympathetic tonus can lead to sinus bradycardia, atrioventricular block, and T-wave abnormalities, eventually leading to ventricular fibrillation or ventricular tachycardia [20]. In the literature, two separate case reports suggest that migraine attacks could lead to ventricular tachycardia as a clinical evidence of pathophysiological link between migraine and ventricular arrhythmias [11,12].

The effect of the ANS on the heart can be evaluated by assessing heart rate variability (HRV). HRV is a potential indicator of ANS function and is regulated continuously by the ANS through the sinoatrial node [21]. Decreased HRV is an indicator of increased cardiovascular risk and mortality [22]. Vollono et al. [23] and Matei et al. [24] evaluated HRV and reported that individuals with migraine showed an imbalance between the parasympathetic and sympathetic nervous systems. These findings indicate that patients with migraine show an imbalance in the ANS.

Coronary vasospasm, which may develop due to the increased activity of the sympathetic nervous system during a migraine attack, may be another mechanism underlying the electrocardiographic changes in patients with migraine who do not have the risk factors of a coronary artery disease. A coronary vasospasm may induce ischemia distal to the spasmodic segment and may prolong ventricular repolarization. Most electrocardiographic changes in patients with migraine are transient. Moreover, coronary vasospasm has been described previously in patients with migraine [3]. Thus, a coronary vasospasm due to the

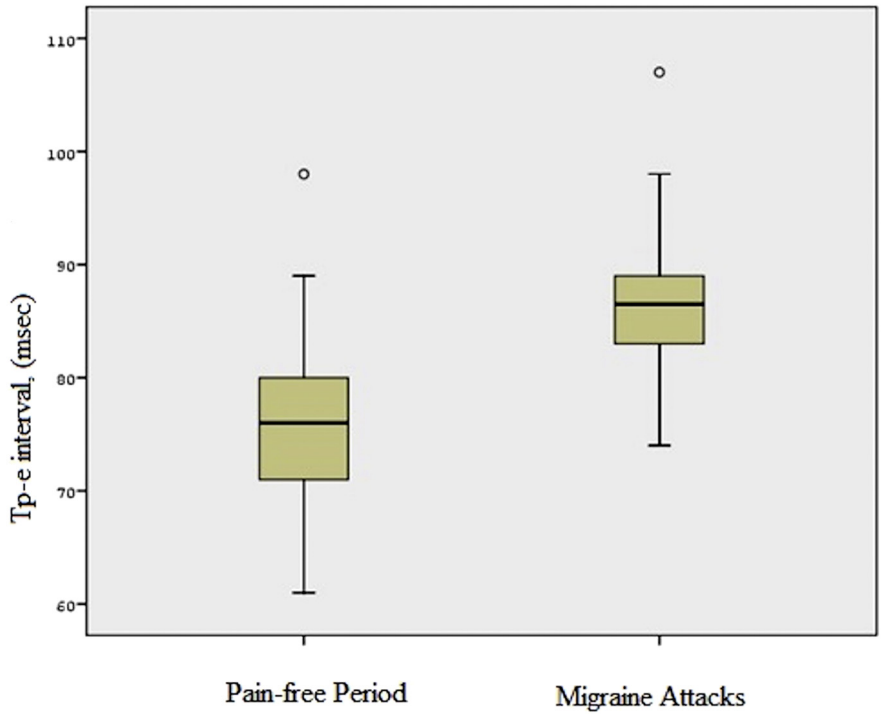

Fig. 1. Box plot of the distribution of mean Tp-e tail (ms) among migraine patients' baseline and attack.

increased activity of the sympathetic nervous system may induce ventricular repolarization disorders in patients with migraine.

ANS dysfunction may also affect ventricular repolarization in patients with migraine. The action potential of myocardial cells depends on endocardial, epicardial, and mid-myocardial M cells. A change in the repolarization timing of these three cell layers produces T-wave changes in surface electrocardiograms [25,26]. Previous studies have suggested that a dysfunction of the sympathetic and parasympathetic nervous systems leads to cardiac arrhythmias by affecting ventricular repolarization [3]. Prolonged QT and QTc intervals are closely associated with an increased risk of sudden cardiac death in patients without any structural heart disease [27]. Pogacnik et al. [28] performed single-lead ECG and reported the increased incidence of cardiac arrhythmias during migraine attacks compared with that during attack-free periods. At present, only few studies have investigated the changes in ventricular repolarization during migraine attacks [4,29]. Aygun et al. [4] and Duru et al. [29] found that the QT and QTc intervals increased significantly during migraine attacks compared with those during attack-free periods. In the present study, the QT and QTc intervals of all the patients were normal during migraine attacks or attack-free periods. However, similar to the studies by Aygun et al. and Duru et al., we found that the QT and QTc intervals were significantly prolonged during migraine attacks compared with that during attack-free periods.

A recent study has reported that the Tp-e interval is an electrocardiographic parameter used for evaluating ventricular repolarization [30]. An increase in the Tp-e interval and Tp-e/QT ratio can be used to predict ventricular tachyarrhythmias and cardiovascular mortality [9]. However, the Tp-e/QT ratio appears to be a more sensitive arrhythmogenic indicator because it remains constant irrespective of dynamic variations in heart rate and body weight than the QT, QTc and Tp-e intervals [31]. A meta-analysis included 33 studies and 155,856 patients demonstrated that prolonged Tp-e interval is associated with 1.14 times higher risk of VT/VF or sudden cardiac death and this parameter might be a useful risk stratification tool in different diseases and in the general population [32]. Based on these findings, we evaluated the changes in the dispersion of ventricular repolarization in patients with migraine by using new parameters, such as the Tp-e interval and Tp-e/QT ratio. To our knowledge, this is the first study to evaluate the transmural dispersion of ventricular repolarization during migraine attacks. Our results showed that the Tp-e interval and Tp-e/QT ratio were significantly increased during migraine attacks compared with those during attack-free periods.

Study limitations

Our study has some limitations that should be considered. First, our study included a small number of patients. Second, difficulties can arise while evaluating T-wave end in an electrocardiogram because of T-wave variability. To counter this and to minimize its effect on our results, an electrocardiographic derivation that best showed the T-wave end was used for analysis (usually the V2 and V5 derivations). Third, analytical discrepancies among the analyzers exceeding 20 ms resulted in the exclusion of patient data from the study. Fourth, because we did not include other ambulatory Holter measures, such as HRV and heart rate turbulence, we could not obtain data on autonomic imbalance during migraine attacks. Finally, the design of the present study did not allow us to document the presence of ventricular arrhythmias by performing Holter ECG. Therefore, our results should be considered with caution and should be validated in a future study.

Conclusion

The present study is the first to illustrate a connection between migraine attacks and the Tp-e interval and Tp-e/QT ratio in an adult population. Migraine attacks may induce ANS dysfunction, and this may exert a negative effect on total ventricular repolarization. Considering the prognostic significance of the Tp-e interval and Tp-e/QT ratio, it is necessary to closely monitor patients with migraine for adverse cardiovascular outcomes. In addition, because sumatriptan use is common among patients with migraine who are at a risk of ventricular arrhythmias, control ECG should be performed in patients taking triptan group medications. Moreover, additional studies should be performed to evaluate the correlation of increased Tp-e interval and Tp-e/QT ratio with increased ventricular arrhythmia risk in this patient population.

Conflicts of interest

None declared.

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