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ARAŞTIRMA / RESEARCH

Relationship between stroke severity and repolarization parameters in patients with acute ischemic stroke

Akut iskemik inmeli hastalarda repolarizasyon parametreleri ile inme şiddeti arasındaki ilişki

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Abstract

Purpose: The aim of this study was to investigate the relationship between repolarization parameters and National Institutes of Health Stroke Scale (NIHSS) score in patients with acute ischemic stroke.

Materials and Methods: The study comprised 97 patients (males, 42; females, 55; 65 ± 16 years) with acute ischemic stroke. 17 patients were excluded. Patients were divided into two groups based on the calculated NIHSS score (Group 1, NIHSS score < 16; Group 2, NIHSS score ≥ 16). Demographic, clinical, and laboratory data for all patients were collected. A 12-lead resting ECG was recorded at admission to the neurology care unit in patients with acute ischemic stroke and were manually measured with a ruler. QTc, QTd, QTcd, Tpe, Tpe/QT parameters were measured.

Results: There were no significant differences among demographic parameters of patients. We found that QTc, QTd, QTcd, Tpe, Tpe/QT parameters were significantly higher in Group 2 than Group 1 patients.

Conclusion: In this study, we found that acute ischemic stroke has been shown to cause changes in repolarization parameters. Repolarization parameters are associated with stroke severity on admission in patients with acute ischemic stroke. Increased dispersion of repolarization parameters make independent contributions to the risk of arrhythmic cardiac death in patients with acute ischemic stroke. We suggested that especially severe ischemic stroke patients closely using cardiac monitoring during the first 24 h.

Key words:. Repolarization parameters, stroke severity, stroke.

Öz

Amaç: Bu çalışmanın amacı, akut iskemik inmeli hastalarda NIHSS ile repolarizasyon parametreleri arasındaki ilişkiyi araştırmak.

Gereç ve Yöntem: Çalışmaya akut iskemik inme geçiren 97 hasta (erkek,42; kadın,55; yaş, 65 ± 16) alındı. 17 hasta çalışmadan çıkarıldı. NIHSS skoruna göre hastalar iki gruba ayrıldı (Grup 1, NIHSS skor <16; Grup 2, NIHSS skoru ≥ 16). Bütün hastalardan demografik, klinik ve laboratuvar verileri toplandı. Hastaların 12 derivasyonlu EKG'leri nöroloji yoğun bakım ünitesine yatırıldıktan hemen sonra çekildi. Ölçümler cetvelle yapıldı. QTc, QTd, QTcd, Tpe, Tpe/QT parametreleri ölçüldü.

Bulgular: Hastaların demografik özellikleri açısından gruplar arasında anlamlı farklılık saptanmadı. Hastaların QTc, QTd, QTcd, Tpe, Tpe/QT parametreleri, Grup 2 hastalarında, Grup 1 hastalarına göre anlamlı derecede daha yüksek bulundu.

Sonuç: Yapmış olduğumuz bu çalışmada, akut iskemik inmeli hastalarda repolarziasyon parametrelerinin inme şiddeti ile ilişkili olduğu sonucuna varılabilir. Bu çalışmada, akut iskemik inmenin repolarizasyon parametrelerinde değişikliğe neden olduğunu bulduk. Repolarizasyon parametreleri, akut iskemik inmeli hastaların yatış esnasındaki inme şiddeti ile ilişkilidir. Repolarizasyon parametrelerinin dispersiyonundaki artış, akut iskemik inmeli hastalarda aritmik kardiyak ölüm riskine bağımsız bir şekilde katkıda bulunmaktadır. Bu nedenle, özellikle ciddi iskemik inmeli hastaların, ilk 24 saatte yakın kardiyak monitorizasyonunu önermekteyiz.

Anahtar kelimeler: Repolarizasyon parametreleri, inme şiddeti, inme.

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INTRODUCTION

Acute stroke is an important cause of morbidity and mortality[1], and cardiovascular complications are common after an acute stroke[2]. Myocardial injury is an important complication of acute cerebrovascular events. Autonomic nervous system dysregulation after acute cerebrovascular events possibly cause sympathetic activation resulting in hypercontraction of cardiac myocytes and subsequent myocardial injury. Neurogenic stress cardiomyopathy (NSC) is a condition of acute myocardial systolic dysfunction that can be observed after acute cerebrovascular events, such as acute ischemic or hemorrhagic stroke, cranial trauma, subarachnoid hemorrhage, and seizures[3]. Often, NSC can be very similar to myocardial infarction, presenting with ischemic ECG changes, including ST segment and T wave deviations, reduced ejection fraction, elevated troponin levels, and ventricular wall motion abnormalities. However, there is no significant obstruction of the coronary arteries in patients with NSC. Acute heart failure due to NSC is an important cause of lethal ventricular arrhythmias and mortality[4]. Ventricular repolarization abnormalities play an important role in the occurrence of arrhythmia[5]. QT dispersion, a marker of repolarization homogeneity, is considered a predictor of sudden cardiac death and mortality in patients with acute ischemic stroke. Despite some controversial data about the positive predictive value of increased QT dispersion, this ECG marker appears to be a powerful tool for risk stratification in patients with impaired left ventricular function after acute ischemic stroke.

The T wave is generated by myocardial voltage gradients during the repolarization phase of cardiomyocyte action potentials. QT interval is a measure of repolarization duration, but may not reveal other changes during the repolarization process. T-wave peak to T-wave end (TPE) interval measures terminal repolarization, and has experimentally been linked to arrhythmogenic repolarization dispersion in the myocardium[6]. In this study, we aimed to investigate the relationship between repolarization parameters and National Institutes of Health Stroke Scale (NIHSS) score in patients with acute ischemic stroke.

MATERIALS AND METHODS

We prospectively studied consecutive 97 adult patients (males, 42; females, 55; mean age, 65±16 years; range 41–80 years) with acute ischemic stroke (≤24 hours of symptom onset) admitted to the neurology care unit, between October 2016 and December 2017. 17 patients were excluded. Demographic and baseline clinical data, including neurological deficit severity assesment with NIHSS on admission to the neurology care unit were recorded. Patient clinical data, history of cardiovascular risk factors, and stroke onset were determined, and neurologic examination was conducted at the time of admission.

The diagnosis was made on the basis of neurologic examination and cranial imaging within 24 hours of symptom onset. Patients with a well-defined time of ischemic stroke symptom onset were included in the study and those with any previous history of cerebrovascular disease or transient ischemic attack, cerebral hemorrhage, documented atrial fibrillation, coronary heart disease, congestive heart failure, serious valvular heart disease, congenital heart disease, chronic obstructive pulmonary disease, chronic renal failure were excluded. In addition, patients in whom a proper position could not be obtained during echocardiography and those with poor echocardiographic image quality, were excluded. Seventeen patients were excluded because of previous history of cerebrovascular disease (n=2), documented atrial fibrillation (n=4), congestive heart failure (n=3), coronary heart disease (n=5), serious valvular heart disease (n= 2), a poor echocardiographic image quality (n=1). Baseline stroke severity was assessed using the NIHSS score[7].

All patients underwent immediate computed tomography after being admitted to emergency care unit. Troponin levels were measured and electrocardiogram (ECG) was recorded after admission to the neurology care unit. Echocardiography was performed within the first 48 hours of admission to the neurology care unit. The NIHSS evaluation and echocardiographic examination were conducted by blinded investigators (U.O and O.O, respectively). The study was approved by the Ethics Committee of Diyarbakir Gazi Yasargil Education and Research Hospital (Date:09 September 2016, Ethics committee number:57), and informed consent was obtained. The study was conducted in accordance with the the principles of the Declaration of Helsinki.

Two-dimensional echocardiography

All patients underwent comprehensive transthoracic

echocardiography examinations, which were conducted according to the guidelines of the American Society of Echocardiography. An ultrasound system (Philips EPIQ 7C, Philips Healthcare, Andover, MA, USA) equipped with a multifrequency transducer (3–8 MHz) and tissue harmonic imaging capability was used. Single-lead electrocardiogram was continuously recorded. Patients were kept in the left lateral decubitus position. Images were obtained from the parasternal long- and short-axes, apical two- and four-chamber, and long-axis views. All echocardiograms included at least three cardiac cycles and were digitally stored for offline analysis. In addition to LVEF, end-systolic and end-diastolic volumes were measured from the apical two- and four-chamber views, using the standard biplane Simpson's technique. Interventricular septum, posterior wall thickness, LV end-diastolic diameter, and left atrial antero-posterior diameter were measured from a parasternal long-axis view[8]. Echocardiographic parameters were calculated by qualified physician (O.O.), and echocardiographic examination was conducted by an investigator who was blinded to the patients' clinical information.

Analysis of QT, TPE interval

All standard 12-lead ECGs were recorded at 25 mm/s speed and 10 mm/mv gain with Nihon Kohden ECG-9132K electrocardiograph (Nihon Kohden Corporation, Tokyo, Japan). A 12-lead resting ECG was recorded at admission in the coronary care unit for patients with an anterior AMI at the first six hours prior to the thrombolysis and then were manually measured with a ruler. All ECGs, were manually analyzed by 1 experienced cardiologist (Onder Ozturk) who was unaware of the clinical data. The QT interval was measured from the beginning of the QRS to the end of the T-wave. The end of the T-wave was defined as the point of return to the isoelectric line9. In cases where the T-wave was interrupted by a U-wave, the end of the T-wave was defined as the nadir between the T- and U-waves. In instances where the T-wave could not be reliably determined due to extremely low voltage (<0.1 mV), measurement of QT interval was not established and consequently these leads were excluded from analysis. In order to exclude the effects of the heart rate (HR) on the QT interval, the QT interval was corrected according to the Bazett formula (QTc=QT/square root of RR interval). QTd was defined as the difference between the maximum and minimum QT intervals. TPE was measured with a ruler from the peak of the T-wave to its end; TPE was corrected for heart rate[9]. The criteria to determine the endpoint of the T-wave were similar to the aforementioned criteria considered for the QT measurement.

Assesment of stroke severity

According to the updated definition of stroke in the American Heart Association/American Stroke Association guidelines, ischemic stroke is diagnosed based on the combination of symptoms and/or signs of typical neurological dysfunction and imaging evidence of central nervous system infarction. Therefore, ischemic stroke is defined as a neurological dysfunction episode caused by focal cerebral, spinal, or retinal infarction on imaging[10].

NIHSS is a simple, valid, and realiable systematic assessment tool that measures acute stroke-related neurologic deficit[7]. The NIHSS score is very important scale for clinical assessment as it enables determination of appropriate treatment, prediction of lesion size, measurement of stroke severity, and prediction of patient outcome in patients with acute ischemic stroke. The NIHSS comprises 11 different elements evaluating specific ability. Each ability is scored between 0 and 4, where 0 corresponds to normal functioning and 4 corresponds to complete impairment. A patient's NIHSS score is calculated by adding the score for each element of the scale; 42 is the highest score possible. A higher NIHSS score corresponds to greater impairment of cerebral function in a stroke patient.

The higher the NIHSS score, the higher the impairment of a stroke patient. According to NIHSS score, there are five stroke severity groups: NIHSS =0 (no stroke), NIHSS=1-4 (minor stroke), NIHSS=5-15 (moderate stroke), NIHSS=16-20 (moderate to severe stroke), NIHSS=21-42 (severe stroke). A baseline NIHSS score greater than 16 indicates a strong probability of patient disability and death[7].Stroke severity at admission to the neurology care unit was assessed by the NIHSS score by a neurologist (U.O). Patients were categorized into two groups; Group 1 comprised of patients with non-severe stroke (NIHSS<16; n=58), whereas Group 2 comprised of patients with severe stroke (NIHSS≥16; n=22).

Statistical analysis

Statistical analysis was conducted with the SPSS statistical package (Version 12.0; SPSS Inc., Chicago,

IL,USA). All baseline parameters were analyzed. Continuous variables are expressed as mean±SD, and categorical variables are expressed as percentages. Intra-observer variability was calculated as the absolute difference between the two measurements as a percentage of their mean. Mann–Whitney U test and Chi-square test were used for comparison of data as appropriate. p values <0.05 were considered statistically significant. Pearson's correlation was used to determine the relationship between LV MPI and other echocardiographic parameters. The Pearson's or Spearman's correlation was used for assessing correlations between variables.

Table 1. Clinical characteristics of patients.

Variables	Group 1 (NIHSS score<16) n=58	Group 2 (NIHSS score≥l6) n=22	p Value
Age (years)	65.4±12.7	69.8±17.7	0.619
Gender (F/M), n	32 / 26	13 / 9	0.718
Hypertension, n %	28 (48%)	13 (59%)	0.062
SBP (mmHg)	143.5±15.2	159.8±18.5	0.043
DBP (mmHg)	76.4±8.6	84.3±12.7	0.048
Heart Rate (bpm)	92.3±14.7	115±16.5	0.027
Diabetes Mellitus, n %	14 (24%)	6 (29%)	0.091
Smoking, n %	7 (12%)	4 (16%)	0.076
Dyslipidemia, n %	9 (15%)	8 (36%)	0.043
Troponin (ng/L)	6.143	14.705	0.037
HbA1c (%)	6.24±1.43	9.56±1.53	0.009
Glucose (mg/dl)	143.5±37.4	192.7±48.5	0.026
Creatinine (mg/dL)	1.3±0.4	1.8±0.6	0.023
LDL cholesterol (mg/dL)	103.3±27.1	128.4±39.5	0.008
HDL cholesterol (mg/dL)	42.1±12.3	39.4±10	0.384

*F:Female, † M:Male, ‡ SBP: Systolic Blood Pressure, § DBP: Diastolic Blood Pressure, || LDL: Low Density Lipoprotein, ¶ HDL: High Density Lipoprotein.

Table 2. Echocardiographic parameters of patients.

Variables	Group 1 (NIHSS score<16) n=58	Group 2 (NIHSS score≥l6) n=22	p Value
LV septal thickness, mm	11.2±1.8	12.7±1.7	0.027
LVDd (mm)	51.2±6.3	54.6±6.8	0.348
LV posterior Wall thickness, mm	10.8±1.5	11.9±1.6	0.034
LVDs (mm)	40.5±4.2	43.2±5.8	0.241
LVEDV (mL)	86.0±17.7	95.4±24.3	0.192
LVESV (mL)	41.1±12.4	44.7±14.2	0.246
LAD (mm)	39.5±4.3	42.4±4.6	0.624
RAD (mm)	32.6±3.4	34.7±3.7	0.590
RVDd (mm)	28.1±2.5	30.7 ± 2.8	0.369
LVEF (%)	59.2±5.6	51.4±6.3	0.024
E/e'	8.7±2.9	10.4 ±3.6	0.017

* NIHSS: National Institutes of Health Stroke Scale, † LV: Left Ventricle, ‡ LVDd: Left ventricular diastolic diameter, § LVDs: Left ventricular systolic diameter, || LVEDV: Left ventricular end-diastolic volume, ¶ LVESV: Left ventricular end-systolic volume, ** LAD: Left atrial diameter, †† RAD: Right atrial diameter, ‡‡ RVDd: Right ventricular diastolic diameter, *** LVEF: Left ventricular ejection fraction.

RESULTS

Baseline characteristics of patients are summarized in Table 1. Clinical characteristics of groups were similar with respect to age, gender, hypertension, diabetes, smoking (p>0.05). Systolic blood pressure (BP), diastolic BP, heart rate, dyslipidemia, and troponin levels in Group 2 patients were signficantly higher than Group 1 patients (p<0.05). Serum hemoglobin HbA1c and low density lipoprotein cholesterol levels were significantly higher in Group 2 patients than Group 1 patients (p<0.05).

Echocardiographic findings are summarized in Table 2. LV wall thickness and E/e' values were

significantly higher in Group 2 patients than in Group 1 patients ($p < 0.05$). LVEF were significantly higher in Group 1 patients having lower NIHSS scores than in Group 2 patients having higher NIHSS. Electrocardiographic findings are summarized in Table 3. Repolarization parameters were significantly higher in Group 2 patients than in Group 1 patients ($p < 0.05$).

Correlation analysis performed to investigate the relationship between NIHSS score and echocardiographic parameters, showed a negative correlation between the NIHSS score and LVEF . In addition, there was a positive correlation between absolute value of the NIHSS score and replarization parameters, age, heart rate and E/e'. (Table 4).

Table 3. Electrocardiographic parameters of patients.

Variables	Group 1 (NIHSS score<16) n=58	Group 2 (NIHSS score≥l6) n=22	p Value
QTc (ms)	486±54.3	538±74.1	<0.05
QTd (ms)	63.8±3.6	89.3±2.5	<0.05
QTcd (ms)	66.5±3.7	92.5±2.8	<0.05
Tpe (ms)	66±328	95±34	<0.05
Tpe / QT	0.13±0.023	0.17±0.029	<0.05

*Tpe: T-wave peak to T-wave end

Table 4. Correlation between NIHSS score and clinical parameters in patients with acute ischemic stroke.

Parameters	Pearson's correlation coefficient (r value)	p Value
LVEF	-0.314	0.038
E/e'	0.217	0.026
Age	0.320	0.042
Heart rate	0.419	0.023
QTc (ms)	0.294	0.045
QTd (ms)	0.257	0.042
QTcd (ms)	0.261	0.039
Tpe (ms)	0.389	0.037
Tpe / QT	0.327	0.029

* NIHSS: National Institutes of Health Stroke Scale, † Tpe: T-wave peak to T-wave end, ‡ LVEF: Left ventricular ejection fraction.

DISCUSSION

Ischemic stroke and heart failure are important causes of morbidity and mortality. It has been previously reported that heart failure is associated with an increased risk of ischemic stroke. Heart failure is also reportedly associated with a poor prognosis in patients with stroke[10,11]. In addition, LV dysfunction occurs frequently after cerebrovascular events[12-14]. LV systolic and diastolic dysfunctions result from complex and interrelated alterations in cardiac function and structure, peripheral vasculature, and neurohumoral regulation. Neurogenic stunned myocardium (NSM) is defined as myocardial injury and dysfunction of the autonomic nervous system, that occurs after cerebrovascular events. Previous studies have suggested that severe neurologic injury, female gender, and elevated plasma troponin and brain natriuretic peptide levels are independent predictors of NSM[15,16]. The effect of ischemic stroke severity on LV repolarization parameters is not very well known, and there are only a few studies investigating this relationship[17,18]

QT_d and QT_{cd} reflect the variation between the durations of QT and QT_c. Increased QT_d and QT_{cd}, which are thought to indicate local heterogeneity in myocardial repolarization, have been shown to cause severe ventricular arrhythmias and sudden heart death[19]. There is evidence of an interaction between the central nervous system and the cardiovascular system during acute cerebrovascular disease[20,21]. Previous studies have reported a significant association between acute ischemic stroke and baseline QT_d and QT_{cd}[18,22]. The effect of acute ischemic stroke on QT_d are thought to be due to neural myocardial stunning, changes in autonomic

nervous system, catecholamine mediated injuries and decreased heart rate variability, reduced parasympathetic control[23]. Lazar et al found that a positive relationship between baseline QT_d and NIHSS and modified ranking scores[24]. In this study, we found that, QTc, QTd, QTcd parameters are positively correlated with stroke severity. Emektar et al found that Tpe and Tpe/QTc parameters were significantly higher in acute ischemic stroke patients than control groups[25]. In our study, we found that Tpe and Tpe/QT parameters are positively correlated with stroke severity. Hypertension, hyperlipidemia and Diabetes Mellitus are important risk factors for atherosclerotic cerebrovascular disease. We found that, blood pressure at admission is significantly higher in severe ischemic stroke patients. However, Bonardo P et al found that, young patients with acute ischemic stroke, large infarct volume was not associated with high blood pressure at admission[26]. In our study, we found that LDL cholesterol and HbA1c were signficantly higher in patients with higher NIHSS scores than in those with lower NIHSS scores. Hendrix P et al. found that diabetes mellitus history is an important predictor of stroke severity[27].

Acute stroke is characterized by profound autonomic dysregulation, including alterations in the autonomic reflex pathways, central autonomic neuroanatomical sites, and hormonal factors. Stroke-related sympathetic activation is high in patients with higher NIHSS score. Irrespective of prior cardiovascular status, an acute phase of stroke markedly influences systemic BP, heart rate, LV function, and biochemical parameters (Glucose, troponin, creatinin)14. In this study, we found that troponin levels were significantly higher in severe ischemic stroke patients. Chang et al observed that cardiac biomarkers, particularly serum troponin levels, are associated with acute large vessel occlusion in patients with ischemic stroke[28]. Lindsberg et al observed elevated blood glucose is common in the early phase of stroke. In our study blood glucose levels were significantly higher in severe stroke patients on admission. Although up to one-third of severe acute ischemic stroke patients have diagnosed diabetes, probably a major proportion of patients have stress hyperglycemia mediated partly by the release of cortisol and norepinephrine[29]. In our study we found that E/é value was significantly higher in severe stroke patients. Ryu WS et al. suggested that E/e' ratios were associated with arterial occlusion in AF-related stroke and may play a role in identifying patients at high risk of severe stroke[30]. In this study we found that creatinine levels were significantly higher in severe stroke patients. Mostofsky E et al suggesting that, shared risk factors underlying vascular diseases including age, diabetes mellitus, hypertension, left ventricular hypertrophy may represent a unique vascular pathogenesis resulting from reduced renal clearence. Renal function predicts survival in patients with acute ischemic stroke[31].

Our study has several limitations. Although we excluded patients with major cardiac history, it is possible that chronic heart failure was missed. However, we excluded patients with echocardiographic parameters that are compatible with chronic heart failure, such as segmental dyskinesis, dilated or hypertrophic cardiomyopathy, or severe valvular disease. Moreover, elevation of cardiac troponin levels over time is in accordance with the current definition of stress cardiomyopathy. Therefore, we suggest that stress cardiomyopathy features should be studied in patients with acute ischemic stroke and without chronic asymptomatic heart failure.

In this study, we found that acute ischemic stroke has been shown to cause changes in repolarization parameters, including prolonged QTc, QTd, QTcd, Tpe, Tpe/QT. Repolarization parameters are associated with stroke severity on admission in patients with acute ischemic stroke. Increasing dispersion of repolarization parameters are associated with an elevated likelihood of arrhythmic cardiac death. Increased dispersion of repolarization parameters make independent contributions to the risk of arrhythmic cardiac death in patients with acute ischemic stroke. Therefore, we suggested that especially severe ischemic stroke patients closely using cardiac monitoring during the first 24 h.

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