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Original Research Article

Electrocardiographic ventricular repolarization markers associated with obstructive coronary atherosclerosis in patients with coronary artery disease

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ABSTRACT

Background: In patients with coronary artery disease (CAD), myocardial ischemia is manifested by an increase in the instability of cardiomyocytes which is potentially characterized by QT interval and T_{peak} - T_{end} interval duration. This highlights the relevance of this study, which was aimed at the evaluation of electrocardiographic ventricular repolarization parameters associated with obstructive coronary atherosclerosis in patients with CAD.
Methods: The study included 63 patients with CAD and stable angina class I-III, referred for coronary angiography. 26 (41%) patients didn't have hemodynamically significant CAD (stenosis <50%) (group 1), and 37 (59%) patients had significant CAD (stenosis ≥50%) (group 2). At admission, a resting standard 12-lead ECG was recorded, taken at a paper speed of 50 mm/sec. Statistical analysis was performed using the STATISTICA 12.0.
Results: Patients of both groups were comparable in age, prevalence of hypertension, obesity, diabetes mellitus, and atrial fibrillation (p>0.05). Patients of group 2 were predominantly male (p=0.04) and more often suffered from myocardial infarction (p=0.03) than patients of group 1. QT interval duration was greater in patients of group 2 compared with group 1 (p=0.049), as well as T_{peak} - T_{end} interval duration (p=0.019).
Conclusions: Patients with significant obstructive CAD had greater values of QT interval and T_{peak} - T_{end} interval (p<0.05) in comparison with patients without coronary atherosclerosis. These parameters may be further used as potential ECG predictors of CAD and assist in risk stratification before coronary angiography.

Keywords: Atherosclerosis, Coronary artery disease, QT interval, repolarization, T_{peak} - T_{end} interval

INTRODUCTION

Cardiovascular diseases are currently the leading cause of death and disability in the developed world.[1]

The main causes of death from cardiovascular diseases (CVD) are the progression of chronic heart failure (about half of all deaths) and sudden cardiac death (SCD) (the other half).[2-4]

The dominant cause of sudden cardiac death (SCD) is malignant ventricular arrhythmias: 75-80% of cases of life-threatening arrhythmias are of coronary origin due to coronary artery disease (CHD) or acute myocardial infarction. The remaining causes (20-25% of cases) are associated with non-coronary pathology, such as dilated cardiomyopathy, arrhythmogenic dysplasia of the right ventricle, hypertrophic cardiomyopathy, non-compact cardiomyopathy, and ion channelopathy.[2]

Acute myocardial infarction is the main cause of sudden cardiac death (SCD) in people over 40-45 years of age, and in people under 40 years of age, who make up the most active social stratum of the population. On the contrary, the main causes of SCD are structural non-coronary heart disease and channelopathy.[2,3,5]

Electrocardiography (ECG) remains one of the most widely available methods for assessing the state of the cardiovascular system. In patients with coronary artery disease (CAD), myocardial ischemia is manifested not only by depression of the ST segment on the ECG, but also by an increase in the instability of cardiomyocytes in the phase of ventricular repolarization, which is potentially characterized by such parameters as QT interval duration and T_{peak} - T_{end} interval duration.

Aim of the study

The aim of the study was to evaluate electrocardiographic ventricular repolarization parameters associated with obstructive coronary atherosclerosis in patients with CAD.

METHODS

Study design and patient selection

The retrospective observational study included 63 patients with CAD and functional class I-III stable angina, referred to the Grodno Clinical Cardiological Center from June 2023 to December 2023 for diagnostic coronary angiography to verify the diagnosis of CAD.

Inclusion criteria

The inclusion criteria were patients above 18 years of age of either gender presenting with typical anginal pain or likely cardiac (pressure, tightness, squeezing, heaviness, or burning) chest pain and diagnosed with functional class I-III stable angina. All patients underwent coronary angiography and the findings were noted and analyzed.

Exclusion criteria

The exclusion criteria from the study were acute complication of coronary atherosclerosis (MI or unstable angina) suffered less than 12 months ago; chronic heart failure with reduced left ventricular ejection fraction (<40%); active inflammatory process of any localization of infectious, autoimmune or other etiology; oncological diseases; pregnancy or lactation; or patient's refusal to participate in the study.

Patients received the necessary medications before hospitalization and during their stay in the hospital in accordance with their diagnosis and clinical condition. All patients received lipid-lowering therapy [atorvastatin (44%) or rosuvastatin (56%)] at doses of 10 or 20 mg for at least 3 months before inclusion in the study. Clinical and demographic data, the results of basic laboratory tests, medical history of comorbidities were collected for all patients from the hospital electronic database (4D client).

Instrumental and laboratory assessment

Patients included in the study underwent instrumental and laboratory research methods. At admission, a resting standard 12-lead ECG was recorded. ECGs were taken at a paper speed of 50 mm/s and calibration of 10 mm/mV. Determination of the duration of the waves and intervals was carried out manually using 12 standard ECG leads, with a record of at least five complete cardiac cycles. The definition of the end of the T wave was carried out using the slope method, at the intersection of the baseline with a tangent drawn from the top of the T wave along its descending part. Calculation of the QTc interval was carried out according to the Bazett formula.

All patients underwent coronary angiography according to the Judkins method (1967) in an x-ray operating room using Philips Azurion 7 and GE Innova 3100 IQ angiographic units. The GE Innova 3100 IQ system's computer program was used for quantitative assessment of stenoses.

Statistical methods

Statistical analysis was performed using the STATISTICA 12.0 computer software. Data distribution was assessed for normality via histogram analysis. Sets of quantitative indicators whose distribution deviated from normal were described using median (Me) values and lower and upper quartiles (Q1; Q3). Nominal data were described using absolute values and percentages. Given that most quantitative variables were not normally distributed, non-parametric methods were applied. Differences between two independent groups were evaluated by Mann-Whitney test, alongside a p value of less than 0.05 considered statistically significant. To compare the diagnostic value of indicators that demonstrated statistically significant differences between groups, ROC analysis was used by constructing characteristic curves of the dependence of sensitivity and specificity of the studied features.

Ethical statement

The study was performed in accordance with Good Clinical Practice standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion in the study.

RESULTS

According to the results of coronary angiography, 26 (41%) patients had no hemodynamically significant coronary artery disease (coronary stenosis <50%) (group 1), and 37 (59%) patients had significant coronary artery disease (stenosis >50%) (group 2).

Clinical characteristics of the patients are presented in Table 1.

Table 1: Clinical characteristics of patients.

Parameter	Criteria	Group 1 (n=26)	Group 2 (n=37)	P value
Male gender, N (%)		11 (42)	25 (68)	0.04
Age, years (M±SD)		61.8±9.2	62.9±7.3	>0.05
Hypertension N (%)	No hypertension	2 (7.7)	1 (2.7)	>0.05
	Stage 1	6 (23.1)	6 (16.2)	>0.05
	Stage 2	18 (69.2)	27 (72.9)	>0.05
	Stage 3	0 (0)	2 (5.4)	>0.05
Myocardial infarction history, N (%)		4 (15.4)	13 (35.1)	0.03
Diabetes mellitus, N (%)		11 (42.3)	19 (51.4)	>0.05
Atrial fibrillation, N (%)		5 (19.2)	7 (18.9)	>0.05
Frequent PVCs, N (%)		13 (50)	14 (37.8)	>0.05
Heart failure NYHA Class, N (%)	Class I	3 (11.5)	1 (2.7)	>0.05
	Class II	20 (76.9)	31 (83.8)	>0.05
	Class III	3 (11.5)	4 (10.8)	>0.05
	Class IV	0 (0)	1 (2.7)	>0.05

Note: M±SD- mean value±standard deviation; PVCs- premature ventricular contractions; NYHA- New York Heart Association

Table 2: Laboratory parameters of patients.

Parameters	Group 1 (n=26) Me (Q1; Q3)	Group 2 (n=37) Me (Q1; Q3)	P value
RBC, 10^{12}/l	4.8 (4.4; 5.1)	4.37 (4; 4.8)	0.006
Hemoglobin, gm/l	146.5 (131; 157)	136 (121.5; 144.5)	0.028
WBC, 10^9/l	5.9 (4.7; 6.9)	7.3 (5.8; 8.7)	0.017
Platelets, 10^9/l	231.6 (202.5; 235)	230.9 (188.5; 266)	>0.05
Urea, mmol/l	5.74(4.81; 6.28)	6.51(5.25; 7.25)	>0.05
Creatinine, µmol/l	88.4 (73.9; 99)	101.1 (83.3; 111.8)	0.049
eGFR, ml/min/1.73m^2	64.5 (55; 76)	62.2 (52.3; 73.5)	>0.05
Total Cholesterol, mmol/l	4.57 (3.22; 5.3)	5.59 (4.82; 6.48)	0.017
Triglycerides, gm/l	1.77 (1.28;2.11)	1.89 (1.08; 2.3)	>0.05
Glucose, mmol/l	6.28 (5.6;6.5)	6.54 (5.39; 6.86)	>0.05
Sodium, mEq/l	138.3 (141; 145)	143.2 (141; 146)	>0.05
Potassium, mEq/l	4.5 (4.2; 4.7)	4.51 (4.18; 4.72)	>0.05
D-dimer, ng/ml	284.5 (224; 319)	416 (220; 382)	>0.05
NT-proBNP, pg/ml	241 (33.9; 390)	1029 (308; 1849)	0.034

Note: Me- median value; Q1- lower quartile; Q3- upper quartile; RBC- red blood cells; WBC- white blood cells; eGFR- estimated glomerular filtration rate; NT-proBNP- N-terminal pro-B-type natriuretic peptide.

Table 3: Electrocardiographic parameters of patients.

Parameter	Group 1 (n=26) Me (Q1; Q3)	Group 2 (n=37) Me (Q1; Q3)	P value
Heart rate, bpm	65 (58; 72)	62 (56; 69)	>0.05
P wave duration, ms	96 (80; 115)	95 (80; 120)	>0.05
PQ interval duration, ms	169(140; 180)	170 (140; 180)	>0.05
QRS complex duration, ms	81 (70; 90)	77 (70; 90)	>0.05
Corrected QT interval duration (Bazett), ms	384 (360; 400)	405 (380; 420)	0.049
T_{peak}-T_{end} interval duration, ms	86 (80; 100)	98 (80; 110)	0.019
T_{peak}-T_{end} / QT ratio	0.22 (0.19; 0.26)	0.24 (0.21; 0.26)	>0.05

Note: Me- median value; Q1- lower quartile; Q3- upper quartile.

Patients of both groups were comparable in age, prevalence of hypertension, obesity, diabetes mellitus, and atrial fibrillation (p>0.05). However, patients of group 2 were predominantly male (68% versus 42%, p=0.04) and had a higher prevalence of prior myocardial infarction (MI) (35% versus 15%, p=0.03) than patients of group 1. Heart failure severity was similar between groups (NYHA class II in the majority of patients in both groups).

Laboratory parameters of patients are presented in Table 2.

In the complete blood count, patients had significant differences in levels of RBC (p=0.006), WBC (p=0.017) and haemoglobin (0.028). In the biochemical blood test, patients of group 2 had higher levels of creatinine and total cholesterol (p<0.05). However, no significant intergroup differences were observed in values of glucose, triglycerides, sodium, and potassium (p>0.05). Importantly, patients with obstructive CAD had significantly higher NT-proBNP levels [1029 (308; 1849) versus 241 (33.9; 390) pg/ml, p=0.034].

Electrocardiographic parameters of patients are presented in Table 3.

According to the results of ECG, patients of both groups did not have significant differences in heart rate [65 (58; 72) versus 62 (56; 69) bpm, p>0.05], duration of P wave [96 (80; 115) ms versus 95 (80; 120) ms, p>0.05], and QRS complex [81 (70; 90) ms versus 77 (70; 90) ms, p>0.05]. However, QTc interval duration was longer in patients of group 2 compared with group 1 [405 (380; 420) ms versus 384 (360; 400) ms, p=0.049]. Moreover, patients of group 2 had higher T_{peak} - T_{end} interval duration than patients of group 1 [98 (80; 110) ms versus 86 (80; 100) ms, p=0.019]. However, patients of groups 1 and 2 didn't have significant differences in T_{peak} - T_{end} /QT ratio [0.22 (0.19; 0.26) versus 0.24 (0.21; 0.26), p>0.05].

There were no significant differences observed in the prevalence of ST segment depression (>0.5 mm) prevalence, [9 patients (34.6%) versus 17 patients (45.9%), p=0.262]. Average ST dispersion was 1 (0.05; 1) mm in group 1 and 1.2 (1; 1.5) mm in group 2, (p=0.433).

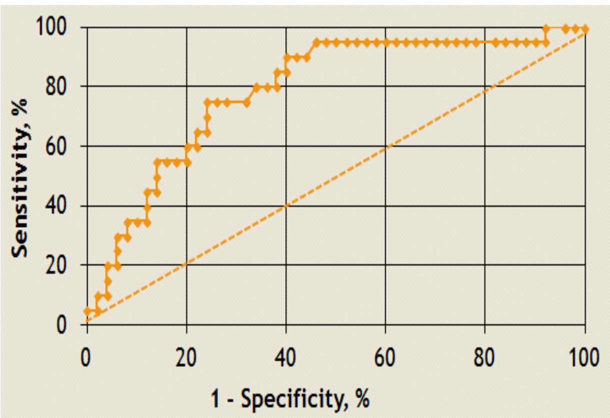

Figure 1: ROC curve for the development of obstructive coronary atherosclerosis depending on the T_{peak}-T_{end} interval value.

A univariate ROC analysis, showed that a T_{peak}-T_{end} interval value of more than 90 ms was associated with a high probability of developing obstructive coronary artery disease, demonstrating high sensitivity (96.15%), but rather low specificity (54.33%). The area under the ROC curve was 0.79 (95% CI 0.68-0.91), p=0.006 (Figure 1).

DISCUSSION

Currently, there are more than three dozen different electrocardiographic markers of electrical instability of the myocardium.[6] Thus, the duration and heterogeneity of the repolarization process reflect such indicators as the duration and variance of the QT interval, the duration and variance of the JT interval, the alternation of the T wave, the duration of the T_{peak} - T_{end} interval and its variance, as well as the ratio of T_{peak} - T_{end}/QT intervals. Markers that reflect the features of myocardial depolarization include the duration and fragmentation of the QRS complex, as well as the magnitude of the QRS-T spatial angle.[6-13]

A separate group of electrocardiographic indicators characterize the balance between ventricular depolarization and repolarization, which include the cardioelectrophysiological balance index (CAB), defined as the duration of the QT interval divided by the duration of the QRS complex, and the corrected CAB index, which is calculated using the duration of the corrected QT interval (QTc).[13-15]

The QT interval is defined as the time from the start of the Q wave to the end of the T wave, representing depolarization and subsequent repolarization of the ventricular myocardium. On a standard ECG the onset of the QRS complex is usually easy identifiable, in contrast to the end of the T wave, which is affected by its morphology, amplitude and presence of the U wave, which represents Purkinje fibers repolarization. The end of the T wave can be measured manually or automatically with the help of threshold method (see Figure 2a), slope method (see Figure 2b) and their variations or novel approach proposed by Hunt. The latter is based on the axiomatic principle that the T wave end point is the first point of intersection of the T wave with a superimposed inverted image of itself, so the T wave becomes a template which measures itself (see Figure 2c).

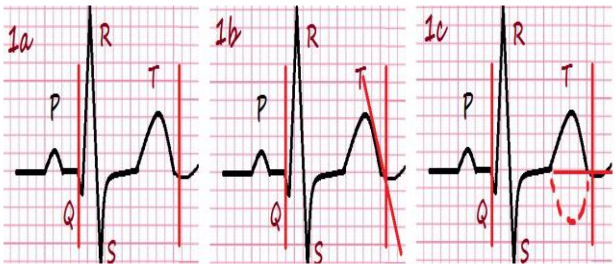

Figure 2: Methods of QT interval measurement: 1a) threshold method; 1b) slope method; 1c) novel-method).

Prolongation of the QTc interval over 450 ms in men and 470 ms in women is associated with the risk of syncope and SCD in patients with both congenital and acquired long QT syndrome.[16-18] In a meta-analysis by Y. Zhang et al prolongation of the corrected QT interval over 450 ms in patients with coronary artery disease, compared to the control group, was associated with an increase in the relative risk of total mortality (OR=1.20, 95% CI 1.15-1.26), cardiovascular mortality (OR=1.29, 95% CI 1.15-1.46) and sudden cardiac death (OR=1.24, 95% CI 0.97-1.60).[19]

The T_{peak} - T_{end} interval seems to be another promising marker of both ischemic and arrhythmic risk, which has been reported as an index of transmural dispersion of repolarization. It is defined as the time difference between the peak and the end of the T-wave, and in the case of negative or biphasic ones it can be measured from the nadir to the end of the T-wave.[20] An increased T_{peak} - T_{end} duration may reflect the period when the epicardium is completely repolarized, but the subendocardial layer (M-cells) remains depolarized, forming an electrical substrate for subsequent depolarization, leading to ventricular arrhythmias. The T_{peak}-T_{end} to QT interval ratio (T_{peak}-T_{end} /QT ratio) is considered less heart rate dependent than T_{peak}-T_{end} itself, because it remains constant despite dynamic heart rate changes.

According to recent studies, an increase in the T_{peak}-T_{end} duration also elevates the risk of life-threatening arrhythmias and, consequently, sudden cardiac death, in patients with Brugada syndrome, cardiac syndrome X and slow coronary flow.[20-22]

However, several studies showed that the duration of the QT interval and T_{peak}-T_{end} are closely related, and prolongation of T_{peak}-T_{end} often reflects a fraction of total QT-interval prolongation. Therefore, the T_{peak}-T_{end} interval cannot be used to distinguish symptomatic patients and should be used only as an additional repolarization marker.[22]

The contradictory nature of the data obtained in experimental and clinical studies does not allow any indicator to be considered universal, which limits the possibility of their independent application and requires an integrated approach.

The choice of formula for calculating the QT interval has not been fully resolved, taking into account the individual nature of the relationship between the QT interval and heart rate, as well as adaptation to the gender and age of patients. Moreover, the use of many promising research methods is difficult due to the presence of persistent atrial fibrillation, low-amplitude or negative T-waves, expansion of the QRS complex, and constant ventricular stimulation.

CONCLUSION

Patients with significant obstructive CAD were predominantly male and more often suffered from prior myocardial infarction. Patients with significant obstructive CAD had higher levels of total cholesterol, creatinine, and NT-proBNP.

These patients also showed significantly longer QT intervals and T_{peak} - T_{end} intervals ($p<0.05$) compared to patients without coronary atherosclerosis. These parameters may serve as potential ECG predictors of coronary artery disease and aid in risk stratification before coronary angiography.

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