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*Original Article*

# Evaluation of Tp-Te interval and Tp-Te/QTc ratio in patients with coronary artery ectasia

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**Abstract:** Aim: Coronary artery ectasia (CAE) is commonly defined as local or generalized dilatation of a coronary vessel up to 1.5 times the diameter of an adjacent vessel. Tp-Te interval and Tp-Te/QT ratio have emerged as novel electrocardiographic markers of increased dispersion of ventricular repolarization. The aim of this study was to evaluate ventricular repolarization by using Tp-Te interval and Tp-Te/QT ratio in patients with CAE. Materials and methods: Patients' records were retrospectively analyzed. Electrocardiogram of 28 patients, who were diagnosed as CAE were obtained and scanned. T wave peak to end interval, QT and corrected QT intervals and some other ECG intervals were measured. Electrocardiograms of age and sex matched 22 control individuals were also analyzed for comparison. Patients with critical coronary stenosis, moderate or severe valve disease, left and/or right heart failure, left and/or right ventricle hypertrophy, atrial fibrillation, moderate or severely abnormal electrolytes, right or left bundle block or patients who got pacemaker or ICD implanted and who undergo hemodialyses were excluded. Results: Baseline characteristics and QT, QTc intervals were similar in both groups. Tp-Te (97.71 ± 8.7 vs 85.23 ± 7.1; $p < 0.001$) and Tp-Te/QT (0.22 ± 0.0 vs 0.20 ± 0.0; $p < 0.001$) were significantly worse in CAE group. Conclusions: T wave peak to end interval is a measure of transmural dispersion of repolarization in the left ventricle and accepted as a surrogate for increased ventricular arrhythmogenesis risk. Tp-Te and Tp-Te/QT are relatively new markers which also indicate repolarization defects. Our results show that CAE patients significantly higher values of Tp-Te and Tp-Te/QT than controls. These measurements may indicate increased arrhythmogenesis risk for individuals with CAE.

**Keywords:** Coronary ectasia, Tp-Te interval, Tp-Te/QT ratio

## Introduction

Coronary artery ectasia (CAE), which is generally, defined as distension of the part of a coronary vessel up to 1.5 times the diameter of a close vessel. It is a rare coronary anomaly, and considered to be congenital or acquired [1, 2]. Isolated CAE comprises a small portion of the total of CAE cases with an incidence of 0.1-0.79% [3]. CAE is thought to be a variant of the coronary atherosclerosis [4].

Myocardial repolarization is associated with susceptibility to ventricular tachyarrhythmias, usually in the form of torsades de pointes, which can degenerate into life-threatening arrhythmias such as ventricular fibrillation [5]. It can be assessed with QT interval (QT), QT dispersion, and transmural dispersion of repolarization. Tp-Te, which is the interval between the peak and the end of T wave on electrocardiogram (ECG), is accepted as an index of transmural dispersion of ventricular repolarization. Tp-Te/QT ratio and Tp-Te/QTc ratio are also used as a new electrocardiographic index of ventricular arrhythmogenesis [6-8].

Even though ventricular repolarization was already evaluated by using T wave and QT interval measurements in patients with CAE [1] the novel myocardial repolarization indexes Tp-Te interval and Tp-Te/QTc ratio, is not studied in these patients before.

In our study, we aimed to assess ventricular repolarization in patients with CAE by using the Tp-Te interval and Tp-Te/QTc ratio.

## Materials and methods

Retrospective data of patients who underwent coronary angiography between January 2013

and February 2013 had been analyzed. Twenty eight patients with CAE (mean age 51.1 ± 7.1 years) and 22 patients with normal coronary anatomy (mean age 49.5 ± 9.4 years) were enrolled as study and control groups. Permission from local Ethical Committee was obtained (authorization code: 2013/1098). Patients with left ventricular dysfunction, echocardiographically proven AF, bundle-branch block, evidence of any other intraventricular conduction defect, or electrolyte abnormalities left ventricular (LV) hypertrophy, hyperthyroidism, chronic obstructive pulmonary disease, ventricular preexcitation, and atrioventricular conduction abnormalities and those on medications known to alter cardiac conduction (antiarrhythmic drugs, digitalis, β-blocker, or calcium-channel blocker medication) were excluded from the study.

*Electrocardiography*

For analysis of the electrocardiographic parameters, lead II, recorded at a paper speed of 50 mm/s (Nihon Kohden®, Tokyo, Japan) at rest in the supine position, was used. All ECGs were scanned. T wave peak to end interval, QT and corrected QT intervals and some other ECG intervals were measured by an engineer with a computer program. By using a ruler, vernier caliper or any other manual measuring tool, getting measurements off from ECG papers could be either inaccurate or slow. Therefore, ECG papers were scanned and this made gathering measurements possible in digital environment. These measurements are done by a program which is generated with MATLAB® (MathWorks, Natick, Massachusetts, U.S.A.) codes that written by an engineer. These codes are based on image manipulation principles.

Image manipulation method could be divided into three subdivisions: image processing, image analysis and image understanding. Image analysis is the technique that should be used to gather measurement data from ECG. Running the written code imports the image file first and then, by choice, allows user to pick points that need to be picked to get measurements or generates a matrix that consists of a dedicated numeric value of each pixel's color. Creating a matrix gives user the flexibility of using functions which predefined by program. In spite of this, hand picking is easier and has a simple interface especially for beginner level users. Algorithms are developed and used to get excellent measurements in order to tolerate differences: such are tilting during scanning process, different scanning resolutions and using different ECG.

The QT interval was defined as extending from the beginning of the QRS complex to where T waves descend onto the isoelectric baseline [9]. When a U wave interrupted the T wave before returning to baseline, the QT interval was measured to the nadir of the curve between the T and U waves. The QTc interval was calculated using the Bazett formula:

$$QTc\ (ms)=QT\ measured/\sqrt{RR}\ (sec).$$

Extended QTc interval was defined as a duration of > 440 ms. The QT dispersion (QTd) value was determined as the difference between the longest and shortest QT intervals observed from the 12 ECG leads [10]. The Tp-Te interval was defined as the interval from the peak of T wave to the end of T wave. Measurements of Tp-Te interval were performed from precordial leads [11]. Tp-Te/QT ratio was calculated from these measurements.

*Echocardiography*

A Vivid 7 pro echocardiographic unit® (GE, Norway) with 3.5 MHz probe was used. Echocardiographic study was performed in left lateral decubitus position. According to the recommendation of the American Echocardiography Association, left ventricular end-diastolic (LVEDD) and left ventricular end-systolic dimensions (LVESD) were measured [12]. We used the Teichholz method to determine left ventricular ejection fraction [13].

*Coronary angiography*

Selective coronary angiography was performed by the Judkins or Sones technique without using of nitroglycerin. Coronary angiograms were analyzed by two experienced observers. CAE was defined as an enlargement of the vessel's lumen above 1.5 times that of an adjacent normal artery or normal parts of the same vessel [14].

*Statistical analysis*

SSPS® version 16.0 was used for statistical analyses. We expressed variables as mean values with standard deviations. Mean values of continuous variables were compared between groups using the Student's t test or Mann–

**Table 1.** Demographic and clinical characteristics of the compared groups

| Parameters | Patients | Controls | *p* value |
|---|---|---|---|
| Mean age (years) | 56.4 ± 12 | 35.6 ± 11 | < 0.001 |
| Male gender [No. (%)] | 12 (42%) | 11 (50%) | NS |
| Smoking (%) | 6 (21%) | 6 (27%) | NS |
| Hypertension (%) | 1 (0.3%) | 14 (60%) | < 0.001 |
| Diabetes Mellitus (%) | 2 (0.7%) | 0 (0%) | NS |
| Total cholesterol (mg/dl) | 180.6 ± 32.1 | 177.09 ± 58.7 | NS |
| Triglyceride (mg/dl) | 165.3 ± 75.5 | 118 ± 49.1 | 0.016 |
| High density lipoprotein (mg/dl) | 47.3 ± 12.7 | 50.0 ± 12.8 | NS |
| Low density lipoprotein (mg/dl) | 99.9 ± 28.0 | 106.8 ± 27.4 | NS |

BMI, body mass index; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; AST, aspartate amino transferase; ALT, alanine amino transferase. Data are presented as means ± SD; NS: Not significant.

**Table 2.** Echocardiographic and electrocardiographic parameters between the patient group with the control group

| Parameters | Patients | Controls | *p* value |
|---|---|---|---|
| LVEDD (mm) | 45 ± 8 | 43 ± 10 | NS |
| LVESD (mm) | 28 ± 5 | 26 ± 6 | NS |
| LVEF (%) | 62.9 ± 4.4 | 65.9 ± 5.1 | 0.03 |
| QT max | 383.43 ± 30 | 375.91 ± 23 | NS |
| QTc max | 421.32 ± 26 | 409.95 ± 18 | NS |
| TP-Te | 97.71 ± 8.7 | 85.23 ± 7.1 | < 0.001 |
| TP-Te/QT ratio | 0.22 ± 0.0 | 0.20 ± 0.0 | < 0.001 |
| QT disp | 26.89 ± 9.9 | 24.41 ± 6.5 | NS |

LVEDD, left ventricle end-diastolic diameter; LVESD, left ventricle end-systolic diameter; IVS, interventricular septum; LVEF, left ventricle ejection fraction; QTc, corrected QT; QTd, QT dispersion. Data are presented as means ± SD. NS: Not significant.

Whitney U test, according to whether normally distributed or not, as tested by the Kolmogorov–Smirnov test. The chi-square test was used to assess differences between categorical variables. We accepted $p < 0.05$ value as statistically significant for our analyses in our study.

## Results

Baseline characteristics and demographic properties of both groups were similar (**Table 1**), except age, serum triglyceride and coexistence of hypertension. However, we think that serum triglyceride is not an important issue and would not affect our measurements on ECG. As it is easily noticed, groups also differ in amount of hypertensive patients. An explanation for this may be that; control group subjects were selected from patients who admit to outpatient clinic with a history of negative coronary angiography for CAE. Probably, these patients were mostly hypertensive patients. On the other hand, hypertension would be a challenging handicap if there was left ventricle hypertrophy, but there isn't (**Table 1**).

After all, it is impossible to exclude risk factors that had led to coronary disease. In echocardiography, left ventricle dimensions were similar whereas control group had slightly higher ejection fraction. However, both groups' mean ejection fraction values were in normal range (**Table 2**).

On the other hand, our results on surface ECG are clear and, there are significant differences between both groups in QT, QTc, Tp-Te intervals (**Table 2**). Also, Tp-Te/QT ratio is significantly higher in patients with CAE (**Table 2**).

## Discussion

Atherosclerosis is one of the major confounding factors for ventricular arrhythmogenesis. Coronary artery ectasia is a variation of atherosclerotic process. However, underlying mechanism is not only oxidized lipid accumulation and plaque forming, but also occurrence of deformations in connective tissue which may manifest as lupus or scleroderma [15, 16]. In autopsy series, varying degrees of diffuse hyalinization, local calcification and fibrosis, destructive process in arterial intima and media [17] were detected. Ectatic segments were shown to be associated with spasm [18], micro thrombi [19] and dissections [20] within neighboring arterial segments. Isolated coronary ectasia may cause ischemia even without significant coronary stenosis. Kruger et al. showed exercise-induced ischemia in patients with isolated CAE by coronary sinus lactate work-up and ergometry [21]. There are also other factors such as coronary slow-flow [22] that may contribute in ischemia. Epicardial and microvascular perfusion failure was also shown by other authors [23] in patients with CAE. Tissue Doppler findings of two echo trials support this microvascular perfusion failure with increased occurrence of left

ventricular diastolic dysfunction in a patient group with CAE [24, 25].

T wave peak to end interval is a measure of transmural dispersion of repolarization in the left ventricle and accepted as a surrogate for increased ventricular arrhythmogenesis risk. Tp-Te/QT and Tp-Te/QTc are relatively new markers which also indicate repolarization defects. Published studies clearly suggest the applicability of Tp-e/QT ratio as a potentially important index of arrhythmogenesis, both under the conditions of short, normal and long QT interval, as well as in congenital and acquired channelopathies. In various high-risk populations, such as, patients with hypertrophic cardiomyopathy [11, 26], post-myocardial infarction [11, 27], long QT syndrome [27-29] inducible ventricular tachycardia [30-32], end-stage renal disease [33], repaired tetralogy of Fallot [34] or Brugada syndrome [35, 36] Tp-Te interval had been found to be more prolonged than control patients.

Underlying mechanism of Tp-Te prolongation and ventricular repolarization abnormality was proposed by Antzelevitch and coworkers [37]. As far as authors describe in their numerous articles, there are three identifiable types of cells in ventricle myocardium. One type of these cells is the subendocardial M cell (Mid-myocardial) which has larger late sodium and sodium/calcium exchange currents and a weaker slowly activating delayed rectifier current [38]. The interval of Tp-Te corresponds with transmural dispersion of repolarization in the ventricular myocardium, a period during which the epicardium has repolarized and is fully excitable, but the M cells are still in the process of repolarization and vulnerable to the occurrence of early after-depolarizations [32, 39, 40]. In suitable conditions, a critical early after-depolarization start a reentry circuit and maintain it for enough time to evolve into polymorphic VT or VF.

Recently published two manuscripts strengthened the relationship between Tp-Te interval and ventricular arrhythmogenesis. Tatlisu et al. [41] reported that Tp-Te and heart rate corrected Tp-Te are both predictors of both in-hospital and long-term mortality. In their study, they followed up 488 consecutive patients who underwent primary percutaneous coronary intervention for ST segment elevated myocardial infarction. The Tp-Te interval was associated with not only in-hospital ventricular tachycardia/fibrillation, target vessel revascularization, and death but also long-term target vessel revascularization and death.

Hetland et al. [42] assessed whether Tp-Te may serve as a predictor of ventricular arrhythmias in patients with previous MI fulfilling current implantable cardioverter-defibrillator (ICD) indications. Their findings were unsurprising; Tp-Te was longer in ICD patients with recorded ventricular arrhythmias compared with those without. Tp-Te was found to be an independent predictor of ventricular arrhythmias when adjusted for age, EF and QRS duration.

In conclusion, our findings indicate that Tp-Te, Tp-Te/QT and Tp-Te/QTc measurements, which were obtained from surface ECG, are significantly high in patients who have CAE rather than healthy controls. CAE is a rare entity which has similar mortality risk with significant coronary stenosis. Ectasia induced myocardial deformations such as ischemia, inflammation and/or fibrosis may end up with myocardial heterogeneity. It seems that Tp-Te prolongation is a promising marker for ventricular arrhythmogenesis in patients with CAE.

However, our study group is small for reaching definite conclusions. A prospective study with long term follow-up for ventricular arrhythmias and/or sudden death would shed more light on this subject.

## Disclosure of conflict of interest

None.

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