

T wave peak to T wave end interval is prolonged in patients with atrioventricular nodal reentry.

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Abstract

Atrioventricular nodal reentry tachycardia (AVNRT) is known to be associated with increased risk of susceptibility for ventricular arrhythmias and sudden death. Studies indicate that prolongation of the interval between the peak and end of the T wave (T_{peak} to T_{end}, T_{p-e}) on the 12-lead ECG is a marker of ventricular arrhythmogenesis. The aim of this study was to assess ventricular repolarization in patients with AVNRT by using T_{p-e} interval, T_{p-e}/QT ratio, and T_{p-e}/QT_c ratio. Patient records of Bursa Postdoctorate Training and Research Hospital were retrospectively analyzed. Electrocardiogram of 62 patients, who were diagnosed with AVNRT by electrophysiological study, were obtained and scanned. T wave peak to end interval, QT and corrected QT intervals and some other ECG intervals were measured. Electrocardiograms by age and sex were matched with 34 healthy individuals who were compared as controls. Patients with critical coronary stenosis, moderate or severe valvular disease, left and/or right heart failure, left and/or right ventricle hypertrophy, atrial fibrillation, right or left bundle block or patients who got pacemaker or ICD implanted were excluded from this study. Both groups did not differ in patient demography. Mean values for AVNRT patients and control group were: QT (362.7±43.7 vs 364.1± 34.1), QT_c (417.8±43.2 vs 438.3±44.2) and T_{p-e} interval (83.5±16.6 vs 77.0±9.7). T_{p-e}/QT and T_{p-e}/QT_c ratio were also higher in the AVNRT group. 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 risk of ventricular arrhythmogenesis. T_{p-e}/QT and T_{p-e}/QT_c are relatively new markers; which also indicate repolarization defects. Our results show that T_{p-e} (p=0.018), T_{p-e}/QT (0.23 vs 0.21; p=0.008) and T_{p-e}/QT_c (0.20 vs 0.17; p=0.000) were significantly higher in AVNRT group; which correspond to previous studies about strong relationship between AVNRT and ventricular arrhythmia.

Keywords: electrocardiogram (ECG), nodal reentry, tachycardia

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Introduction

Atrioventricular nodal reentry tachycardia (AVNRT) is the most common form of regular supraventricular tachycardia (SVT) [1]. AVNRT is known to be associated with higher susceptibility for ventricular arrhythmias than healthy individuals [2, 3]. Sudden cardiac death due to ventricular fibrillation is a rare, but serious complication of nodal reentry. Underlying mechanisms of sudden cardiac death due to these arrhythmias are unclear yet. Recent studies indicate that prolongation of the interval between the peak and end of the T wave (T_{peak} to end,

T_{p-e}) on the 12-lead ECG is a marker of ventricular arrhythmogenesis [4, 5, 6]. Prolongation of this interval represents a period of potential vulnerability to re-entrant ventricular arrhythmias. Prolonged T_{p-e} has been associated with increased risk of mortality in the congenital and acquired long QT syndromes [7], hypertrophic cardiomyopathy [8] and also in patients undergoing primary PCI for myocardial infarction [9]. However, there is no information available assessing the T_{p-e} in patients with AVNRT. The aim of this study was to assess ventricular repolarization in patients with AVNRT by using T_{p-e} interval, T_{p-e}/QT ratio, and T_{p-e}/QT_c ratio.

Methods

Subjects of study

Patient records of Bursa Postdoctorate Training and Research Hospital were retrospectively analyzed. ECG of 62 patients, who were later diagnosed as AVNRT by electrophysiological study, were obtained and scanned. ECGs by age and sex were matched with 34 healthy individuals, who did not have AVNRT or any supraventricular tachycardia. They were used as controls. Patients with critical coronary stenosis, moderate or severe valvular disease, left and/or right heart failure, hypertension of any stage, left and/or right ventricle hypertrophy, atrial fibrillation, right or left bundle block or patients who got pacemaker or cardioverter/defibrillator implanted were excluded from the study.

Measurement of Tp-e, QT and QRS Intervals from the 12-Lead ECG:

All ECGs were scanned. The Tp-e interval was defined as the interval from the peak of T wave to the end of T wave. Measurements of Tp-e intervals were performed from precordial leads [10]. T wave peak to end interval, QT and corrected QT intervals were measured by an engineer with a computer program involving the MATLAB (MathWorks, Natick U.S.A.) since using a ruler, vernier caliper or any other manual measuring tool to get measurements from ECG papers could be either inaccurate or slow. 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 the 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 that is 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. 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: $QTc \text{ (milliseconds)} = QT \text{ measured} / \sqrt{RR} \text{ (seconds)}$.

Statistical Analysis

SPSS 13.0 statistical software was utilized. Independent-samples T test and Pearson's chi-square tests were used for univariate case-control comparisons of continuous and categorical variables for all cases vs. controls, respectively.

Results

Both groups did not differ in patient demography (Table 1). Mean values for AVNRT patients and control group were: QT (362.7±43.7 vs 364.1± 34.1), QTc (417.8±43.2 vs 438.3±44.2) and Tp-e interval (83.5±16.6 vs 77.0±9.7). Tp-e/QT and Tp-e/QTc ratio were also higher in AVNRT group. All of the measurements are listed in table 2.

Table 1. Demographic findings of both groups.

Variable	Patient group (n=62)	Control group (n=34)	P value
Male sex (n, %)	27/62 (43.5%)	10/34 (29.4%)	0.176
Age (years)	48.3±14.4	42.2±13.9	0.091
Hypertension (n, %)	0/62 (0%)	0/34 (0%)	1.000
Diabetes (n, %)	10/62 (16.1%)	6/34 (17.6%)	0.849
Smoker (n, %)	15/62 (24.2%)	7/34 (20.6%)	0.689

Table 2. Electrocardiographic measurements of both groups.

Parameter	Patient group (n=62)	Control group (n=34)	P value
QT interval (msec)	362.7±43.7	364.1± 34.1	0.870
QTc (msec)	417.8±43.2	438.3±44.2	0.030
Tp-e interval (msec)	83.5±16.6	77.0±9.7	0.018
Tp-e/QT	0.23±0.0	0.21±0.0	0.008
Tp-e/QTc	0.20±0.0	0.17±0.0	<0.001

Tp-e: T wave peak to end interval, QTc: corrected QT interval, msn: milliseconds, Data are presented as means ± SD

Discussion

The present study showed that Tp-e interval, Tp-e/QT and Tp-e/QTc ratio were prolonged in patients with AVNRT. 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 risk of ventricular arrhythmogenesis. Tp-e/QT and Tp-e/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 population, such as, patients with hypertrophic cardiomyopathy [11, 12, 13], post-myocardial infarction [9, 11], long QT syndrome [14, 15, 16] inducible ventricular tachycardia [17, 18, 19], end-stage renal disease [20], repaired tetralogy of Fallot [21] or Brugada syndrome [10, 22] Tp-e interval had been found to be more prolonged than control patients.

Underlying mechanism of Tp-e prolongation in ventricular repolarization abnormality was proposed by Antzelevitch and coworkers [6]. The authors describe in their numerous articles that there are three types of identifiable cells in the ventricular 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 [23]. The interval of Tp-e 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 [19, 24, 25]. In suitable conditions, a critical early after-depolarization starts a reentry circuit and maintains it long enough to evolve into polymorphic VT or VF.

As we have mentioned above, Tp-e interval and Tp-e/QT and Tp-e/QTc ratios were validated in various cardiac conditions that may lead to sudden cardiac death. Atrioventricular nodal reentry has been known for its benign course. However, some cases of sudden death are available in the literature [2, 3]. In addition, idiopathic ventricular arrhythmias may coexist with AVNRT [26]. Mechanism of this susceptibility in patients with AVNRT remains unclear. Nigro et al. showed relationship between autonomic function and AVNRT [27]. Thus, alterations in autonomic neural tone may be another reason for the increase of Tp-e interval and Tp-e/QT ratio in patients with AVNRT.

In our study, Tp-e; which is a surrogate for transmural repolarization heterogeneity and an accepted marker for sudden death caused by ventricular arrhythmia, was found to be longer ($p=0.018$), in patients

with AVNRT than healthy controls. What is more, results show that Tp-e/QT (0.23 vs 0.21; $p=0.008$) and Tp-e/QTc (0.20 vs 0.17; $p=0.000$) were significantly higher in AVNRT group.

Conclusion

Our findings put forward that Tp-e, Tp-e/QT and Tp-e/QTc measurements; which were obtained from surface ECG, are significantly high in patients who suffer from AVNRT rather than healthy controls. Keeping the coexistence of AVNRT and ventricular arrhythmias in mind, results raise the question whether there is myocardial repolarization heterogeneity in these patients or not. Since Tp-e prolongation is an accepted marker for sudden death caused by ventricular arrhythmia in some conditions, it is worth investigating for its utility in risk assessment for this group of patients.

Limitations

This is the first study; which aims to relate AVNRT with Tp-e measurements. However, our subject number is quite small in establishing a definite conclusion. A prospective study with long-term follow-up for ventricular arrhythmias would shed more light on this subject. Furthermore, utilization of electro-anatomic mapping systems would contribute to the evaluation of disturbances in ventricular conduction and/or electrical disarrangement. This limitation is also a result of the retrospective nature of our study.

Conflict of interest

Authors do not declare any conflict of interest.

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