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ORIGINAL ARTICLE

Prognostic value of T peak-to-end interval for risk stratification after acute myocardial infarction☆

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KEYWORDS

Tp-Te interval;
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Sudden death;
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Abstract *Aim:* Fatal arrhythmia is the main cause of sudden death in patients with acute myocardial infarction either during hospital admission or in post-discharge period. Our aim is to identify groups at high risk of arrhythmic mortality by using a simple bed-side test in electrocardiogram.

Background: Trans-mural dispersion of repolarization (TDR) in patients with ST elevation myocardial infarction is the main trigger of arrhythmias. The potential value of measuring the interval between the peak and end of the T wave (Tpeak-Tend, Tp-Te) as an index of spatial dispersion of repolarization is a parameter thought to be capable of reflecting dispersion of repolarization and thus may be prognostic tool for detection of arrhythmic risk. Little is known about its use for identifying risk of arrhythmias in acute myocardial infarction and this must be approached with great caution and require careful validation.

Methods: A prospective analysis of data from 564 patients admitted to our CCU by acute myocardial infarction along a period of two years from January 2012 to December 2013 was done. After exclusion of valvular, congenital lesions, HOCM, IDCM, pericardial diseases, accessory pathway, any Bundle branch block, metabolic disorders and re-perfusion arrhythmia. Analysis of TpTe interval and its dispersion were done for all patients and a Holter-24 h was performed after one month. Patients were then classified into two groups based on Lown grading score for arrhythmia:

Abbreviations: AP, action potential; ECG, electrocardiogram; STEMI, ST elevation myocardial infarction; TDR, transmural dispersion of repolarization; Tp-Te, Tpeak-Tend interval; VF, ventricular fibrillation; VT, ventricular tachycardia; EAD, early after depolarization; LAS, low amplitude signals; AMI, acute myocardial infarction

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group (I) (441 patients) with no or minimal arrhythmias (Lown score < 3), and group (II) (123) with high grade arrhythmias (Lown score $\geqslant 3$). In-hospital predischarge echocardiography was done for all patients to evaluate left ventricular functions and presence of myocardial aneurysm. Signal average ECG was done to detect low amplitude signals (LAS). Pre-discharge coronary angiography was done for all patients.

Results: Statistical analysis of the results revealed that, group (II) patients carry a significantly higher number of obese, diabetic, and hypertensive patients. Most of patients in this group were smokers, having higher creatinine levels, and exposed previously to cerebral insults in significantly higher values than group (I). Also, group (II) patients need significantly higher doses of diuretic and ACEIs than group (I). The percentage of anterior wall infarction is significantly higher in group (II), with higher inferior wall affection in group (I). TpTe interval and dispersion between both groups revealed that, a higher TpTe interval was found in group (II) than group (I) and this was linked to occurrence of sudden death or malignant VT and deterioration in Lv functions than in group (I). Also, patients in group (II) exposed to re-infarction and cardiogenic shock in statistically significant values ($P < 0.01$) than group (I).

Conclusion: TpTe was significantly and independently associated with increased odds of SCD and is linked to deterioration of Lv functions and myocardial aneurysms. It's highly correlated to presence of LAS and associate with severity of coronary lesions. Patients with prolonged TpTe intervals and dispersions were likely to develop fatal arrhythmias.

1. Introduction

Amplification of spatial dispersion of repolarization, particularly transmural dispersion of repolarization (TDR), within the ventricular myocardium has been suggested to underlie arrhythmogenesis in different cardiac diseases, such as the Brugada, short QT, long QT syndromes, and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) [1].

Numerous studies [2,3] have highlighted the heterogeneity that exists among the cells that comprise ventricular myocardium, demonstrating unique electrophysiologic and pharmacologic profiles for epicardial, endocardial, and M cells in a number of species, including man. Differences in the time-course of repolarization of these three predominant ventricular myocardial cell types contribute prominently to the inscription of the electrocardiographic T wave recorded in the precordial leads V5.

These differences in action potential (AP) morphology lead to the development of opposing voltage gradients on either side of the M region, which contribute to inscription of the T wave, especially those inscribed in the precordial leads (Fig. 1). The interplay between these opposing transmural forces determines the height and width of the T wave and the extent to which the T wave may be interrupted, resulting in a bifid or notched appearance [4].

Recent studies have identified TDR and other forms of spatial dispersion of repolarization as the principal substrate and early afterdepolarization (EAD)-induced extrasystoles as the most common trigger for the development of lethal arrhythmias [5].

During bradycardia or because of a repolarization-prolonging insult, the action potential of the M cells is more vulnerable to prolongation compared with the other 2 cell types [6], likely because of larger late-sodium and sodium/calcium exchange currents and a weaker, slowly activating delayed rectifier current [7]. TpTe 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 are vulnerable to the occurrence of early afterdepolarizations [8,9].

One of the challenges ahead is to identify a means to quantitate spatial dispersion of repolarization and TDR non-invasively. In this study, we discuss the potential value of the interval between the peak and end of the T wave (Tpeak-Tend,

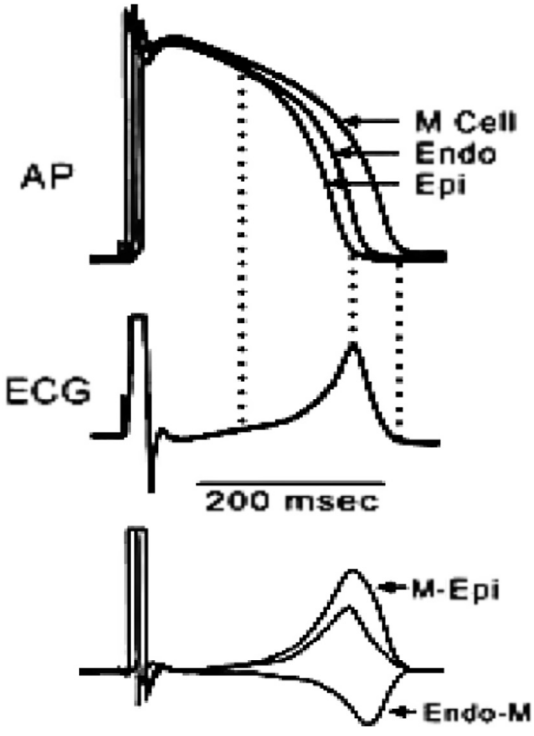

Figure 1 Voltage gradients on either side of the M region are responsible for inscription of the electrocardiographic T wave.