

Tp-e interval and Tp-e/QTc ratio: new choices for risk stratification of arrhythmic events in patients with hypertrophic cardiomyopathy

To the Editor,

I read with a great interest the paper entitled "Tp-e interval and Tp-e/QTc ratio as novel surrogate markers for prediction of ventricular arrhythmic events in hypertrophic cardiomyopathy" by Akboğa et al. (1) published in *The Anatolian Journal of Cardiology*. In this novel investigation, the authors have shown that the Tp-e interval and Tp-e/QTc ratio were significantly longer and higher in patients with hypertrophic cardiomyopathy (HCM) than in controls. In addition, multivariate analysis revealed that these markers were associated with a higher risk of ventricular arrhythmic events (OR: 1.060; 95% CI: 1.005–1.117; $p=0.012$ and OR: 1.148; 95% CI: 1.086–1.204; $p=0.049$, respectively).

The electrocardiogram is commonly used for predicting arrhythmogenic risk in clinical practice. Now, the Tp-e interval and Tp-e/QTc ratio have been proposed as markers for predicting malignant ventricular arrhythmias and have been evaluated and recommended as alternatives for risk stratification of sudden cardiac death in patients with several medical conditions.

The Tp-e interval is an index of the transmural dispersion of ventricular repolarization (VR); it reflects the different duration of the action potential in the epicardium, endocardium, and M cells from the heart. These cellular mechanisms are translated to the T wave on surface 12-lead electrocardiogram and allow the determination of an increase in the transmural dispersion of VR through a single measure from the peak or nadir to the end of the T wave. The Tp-e/QTc ratio includes values of the transmural and spatial dispersion of VR. Although it was initially thought that the Tp-e/QTc ratio remains relatively constant between a heart rate of 60 to 100 beats/min, many researchers have recently published good outcomes after the correction of this parameter by the heart rate (2, 3).

Patients with HCM have a predisposition for ventricular arrhythmias and sudden cardiac death. The structural abnormalities in HCM are diverse and generally associated with the severity and extension of the pathophysiological process. Disarray of cardiac fibers, microvascular ischemia, and fibrosis are conditions that predispose patients with HCM to an increase in the dispersion of VR, reentrant arrhythmias, and sudden cardiac death (4).

Current European guidelines propose an algorithm for the risk stratification of sudden cardiac death and suggest the insertion of an implantable cardioverter defibrillator in these patients based on several variables, including age, family history of sudden cardiac death, unexplained syncope, left ventricular outflow

gradient, maximum left ventricular wall thickness, left atrial diameter, and presence of non-sustained ventricular tachycardia during 24–48-h ambulatory electrocardiographic monitoring (5). However, no electrocardiographic marker is used on the basis of the analysis of VR, presumably because of a lack of evidence about its utility. The study by Akboğa et al. (1) may open a new field of investigation on this topic. The electrocardiogram is accessible by most patients. These markers may be obtained, analyzed, and interpreted easily by all physicians without any specific training. These features could represent an incentive to introduce these markers as part of future risk stratification models in patients with HCM. However, for this purpose, it is necessary to continue investigations in this field with prospective studies and with a larger number of patients.

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