CorSalud 2013 Jan-Mar;5(1):130-132

CorSalud

Cuban Society of Cardiology

Letter to the Editor

Cardiocentro ERNESTO CHE GUEVARA SANTA CLARA CUBA

Significance and mechanisms of a prolonged QT interval in acute myocardial ischemia

Significado y mecanismos de un intervalo QT prolongado en la isquemia miocárdica aguda

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ARTICLE INFORMATION

Received: July 01, 2012
Accepted: August 22, 2012

Key words: QT interval, acute coronary syndrome, acute myocardial infarction
Palabras Clave: Intervalo QT, síndrome coronario agudo, infarto agudo de miocardio

To the Editor:

The electrocardiographic evolution in a patient with ST segment elevation acute myocardial infarction is reported in this issue[1]. Preceding the current of injury (ST elevation), this patient experienced a huge QT prolongation with T wave inversion in multiple leads of the electrocardiogram (ECG). For this reason, it is important to comment, briefly, this interesting association.

The QT interval is determined on the ECG from the start of the QRS complex to the point where the T wave (or U wave if present) returns to the isoelectric line. Therefore, it includes the duration of ventricular depolarization and repolarization, and corresponds to the action potential duration.

Clinical significance

The QT prolongation in coronary ischemia is a well-known sign, which has become part of the parameters to be considered in the calculation of the ischemic risk in acute coronary syndrome[2,3]. In addition, it is known that the prolongation of this interval, after an acute myocardial infarction with Q wave, is associated with a significantly increased risk of sudden death[4]. Then, it is important to emphasize that although the prolonged QT interval is a risk marker of ventricular arrhythmias, which increases even further in ischemic myocardium, some authors consider it as a predictor of ischemic risk, and not of arrhythmic risk, in patients with acute coronary syndrome without ST elevation, because it is a marker of advanced coronary disease, or of the seve-

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rity of underlying myocardial ischemia[5,6].

Electrophysiological mechanisms

The mechanisms responsible for QT prolongation in patients with acute infarctions are controversial, and are probably related to the electrical heterogeneity of the ventricular myocardium, which consists of three types of cells that have variable electrophysiological properties. The M cells, located in the mid-myocardium, have an action potential duration significantly longer than in the epicardium and endocardium, and it coincide with the end of the T wave[7]. In the absence of injury, the electrotonic coupling of M cells with the adjacent cell layers minimizes these inherent differences of the action potential[8]. However, after an injury, as occurs in acute myocardial infarction, the decoupling M cells from the adjacent cell layers eliminates these electrotonic influences and allows the expression of the intrinsic properties of the M cells, which are manifested in the surface ECG as QT prolongation[9].

There are other mechanisms that are thought to cause the prolongation of the QT interval during an acute myocardial ischemia. They are: the reduction of temperature in the epicardium[10], impedance changes[11], acidosis[12-14], and it has also been observed that there is an inward current of sodium preceding a potassium efflux during ischemia. During the first minutes of sudden reduction in blood flow, the activation of the inward current of sodium prolongs the action potential duration, and the gain of sodium is the trigger of the net potassium loss[15].

The lysophosphatidylcholine, a product of the catabolism of phospholipids induced by ischemia, has been linked to alterations in the kinetics of sodium channels, resulting in a no inactivation of this ion current and, and therefore, in the prolongation of repolarization[16-18].

The authors of this letter did not want to omit the explanation of these electrocardiographic findings reported in the section Images in Cardiology[1], as most of the texts that are available to many doctors do not emphasize this association, which may warn us of a major problem.

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