A call for QT interval measurement and its correction in acute myocardial ischemia

Un llamado a la medición del intervalo QT y su corrección en la isquemia miocárdica aguda

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To the Editor:

The electrocardiogram (ECG) of twelve leads is both available and easily performed at the bedside of the patient, when an acute ischemic heart disease is suspected. These characteristics have made it easier for several investigators to evaluate the corrected QT interval as an independent risk marker in non ST-segment elevation acute coronary syndrome (NSTE-ACS)[1].

In the issue 5 (1) of this journal, Rodríguez González *et al.*[2], investigated the association between prolonged corrected QT interval, during ST segment elevation acute coronary syndrome (STE-ACS) with the presence of ventricular arrhythmias and occurrence of a new acute episode of coronary ischemia. In the same issue of the journal, Elizundia *et al.*[3], show a ECG with prolonged QT, preceding the current of injury in a patient with STE-ACS.

In studies with both NSTE-ACS and STE-ACS[1,2] the corrected QT interval has been described as a risk marker in the populations studied. The poor inclusion of patients in these studies limits the possibility of considering prolonged corrected QT interval in acute cardiac ischemia, as a variable to predict risk.

Cruz Elizundia *et al.*[4], summarized the meaning and mechanisms of prolonged QT interval in acute myocardial ischemia, by mentioning that M cells, located in the mid myocardium, show a significantly longer action potential duration in the epicardium and endocardium, and coincides with the end of the T wave of ventricular repolarization, hence there is electrotonic coupling with the adjacent layers. After an injury, as occurs in myocardial infarction, the decoupling of M cells with adjacent cell layers eliminates these electrotonic influences and allows the expression of the intrinsic properties of M cells, which show themselves in the surface ECG as QT prolongation.

This expression of the intrinsic properties of M cells depends, of course, on the shortening of the action potential of adjacent cells that received the ischemic damage resulting from electrolytic changes in both intracellular and extracellular medium. The latter has been described in the electrophysiological changes occurring during acute myocardial ischemia[5,6]. These changes will occur in the region of the coronary artery responsible for the ischemia, which gives great heterogeneity to myocardial electrophysiology at that moment, and is expressed not only with the corrected QT but also with increments in the corrected QT interval dispersion, as demonstrated by Rodríguez González *et al.*[2].

In the presence of precordial chest pain, the doctor will always evaluate the ST segment which has been historically observed in suspected acute coronary ischemia. However, if the electrophysiological changes that occur during acute myocardial ischemia, which are responsible for the prolongation of ventricular repolarization (QT), are understood, then it is necessary to convene the medical community to design studies to validate the corrected QT interval prolongation as an independent risk marker in NSTE-ACS and STE-ACS.

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Diabetes mellitus and its relation to cardiovascular disease

La diabetes mellitus y su relación con las enfermedades cardiovasculares

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To the Editor:

According to the World Health Organization, cardiovascular disease is a major public health problem in the world, and the leading cause of mortality as it causes 17 million deaths per year, accounting for half of all deaths in the United States and other developed and developing countries[1].

Although estimates of life expectancy reflect how old a person expects to live, they do not specify the "expected" health status during life, given the mortality rates of certain environments. Mortality statistics by themselves are not enough to describe and compare the health status of different populations because they underestimate the serious health problems caused by chronic diseases.

The number of cardiovascular events in a year is much higher in patients who have already developed one or more forms of atherosclerotic disease (cerebral, coronary or peripheral artery disease) than in those with a high risk profile but who have not developed any disease expression[2,3].