Novi pokazatelji aritmogenog rizika (Tp-e interval; Tp-e/QT omjer): elektrofiziološke postavke i moguća primjena

Novel indicators of arrhythmogenic risk (Tp-e interval; Tp-e/QT ratio): electrophysiological bases and possible application

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U više od 90% iznenadno umrlih srčanom smrću (iznenadna srčana smrt, ISS) nalazi se koronarna bolest srca (KBS) sa suženjem lumena koronarne arterije > 75%. Uzrok ISS – nerijetko prve manifestacije KBS – su, u pravilu, maligni poremećaji srčanog ritma (ventrikulska tahikardija i ventrikulska fibrilacija). Istraživanja aritmogeneze u cilju predviđanja, a time i sprječavanja, možebitnih ISS u bolesnika s KBS su do prije 10-ak godina bila usmjerena na promjene QT i QTc (korigiranog QT) intervala te QT i QTc (korigirane QT) disperzije.[1-3] U tom je slijedu produženi QT interval prihvaćen kao pokazatelj produženog vremena repolarizacije, a povećana QT disperzija smatrana odrazom prostornih razlika repolarizacije miokarda klijetki. Posljednjih su godina istraživanja ventrikulske aritmogeneze sve više usmjerena na dva novija elektrokardiografska (EKG) pokazatelja: Tp-e (engl. *T peak-to-end*) interval i Tp-e/QT omjer. Prema dosadašnjim rezultatima, ovi EKG pokazatelji su odraz heterogenosti transmuralne repolarizacije, odnosno razlike napona između pojedinih slojeva istog segmenta stijenke lijeve klijetke, a njihove su promjene prihvaćene kao obećavajući pokazatelj aritmogenog potencijala u bolesnika s nizom srčanožilnih bolesti, poput produljenog (kongenitalnog ili stečenog) QT sindroma, kratkog QT sindroma, Brugada sindroma, hipertrofijske kardiomiopatije, akutnog koronarnog sindroma te kronične stabilne KBS. U izlaganju će biti iznesene osnovne spoznaje o elektrofiziološkoj podlozi Tp-e intervala i Tp-e/QT omjera s osobitim osvrtom na njihove promjene u bolesnika sa stabilnom KBS u kojih revaskularizacija prethodno ishemičnog miokarda normalizira naporom potaknute promjene repolarizacije, sugerirajući smanjenje aritmogenog potencijala.

More than 90% of sudden cardiac deaths (SCD) is caused by coronary artery disease (CAD) with arterial stenosis greater than 75%. The pathophysiology bases of SCD - often the first manifestation of CAD – are malignant arrhythmias (ventricular tachycardia and ventricular fibrillation). Until 10 years ago, the investigation of ventricular arrhythmogenesis, in order to predict the arrhythmogenic potential and prevent SCD, was focused to QT and QTc (corrected QT intervals) and QT and QTc (corrected QT) dispersion (QTd, QTcd).[1-3] In this context, the prolonged QT interval was accepted as an indicator of extended repolarization time, and increased QT dispersion was considered a reflection of spatial differences in myocardial repolarization. Recently, research of ventricular arrhythmogenesis has been increasingly focused on two newer electrocardiographic (ECG) indicators: Tp-e (T peak-to-end) interval and Tp-e/QT ratio. According to the latest findings, these ECG indicators are the reflection of transmural heterogenicity of repolarization, i.e. the differences in voltage between the individual layers of the same segment of the ventricle wall. Their changes are accepted as a promising indicator of arrhythmogenic potential in patients with several cardiovascular diseases, such as prolonged (congenital or acquired) QT syndrome, short QT syndrome, Brugada syndrome, hypertrophic cardiomyopathy, acute coronary syndrome and chronic stable CAD. This presentation will provide basic insights into the electrophysiological background of the Tp-e interval and the Tp-e/QT ratio with particular attention to their changes in CAD patients in which restoration of blood supply normalizes exercise-induced repolarization abnormalities, suggesting that revascularization of a previously ischemic myocardium lowers its arrhythmogenic potential.

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