Early Repolarization: How to Differentiate the “Malignant” from the “Benign” Pattern

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The early repolarization syndrome (ERS) on the electrocardiogram is characterized by J-point elevation, distinct J waves with or without ST-segment elevation, symmetric tall T wave, and slurring of the terminal part of the QRS in inferior limb and lateral precordial leads, but not in the V2 to V5 leads. The new definition of ERS proposed by Haissaguerre et al, centers more on the J wave notch or slur, irrespective of the ST-segment changes, which often helps to avoid overlap with the Brugada syndrome.1-3

The ERS has been considered for over 60 years a benign ECG phenomenon that is found in 1% to 13% of the general population, more commonly seen in young healthy men and athletes. ERS may also coexist or mimic the ECG of other cardiac diseases such as acute myocardial infarction, pericarditis, ventricular aneurysm, hyperkalemia, or hypothermia, cocaine users, patients with hypertrophic obstructive cardiomyopathy and in patients with defects and/or hypertrophy of the interventricular septum.4-6 Practically, the diagnosis of ERS should be made after evaluation of all possible causes, such as coronary artery spasm or subtle forms of arrhythmogenic right ventricular dysplasia. Recent data suggest the KCNJ8 gene may be involved in early repolarization syndrome.7 Experimental data showed that phase 2 reentry can be initiated in ERS and trigger polymorphic ventricular tachycardia (VT) and ventricular fibrillation (VF).8 Clinically, a growing number of recent case reports and case-control studies indicate that ERS patterns may be related to electrophysiological instability and even may be associated with increased risk of idiopathic ventricular fibrillation.4,5 The observation of early repolarization (ER) becomes important in context with syncope or a family history of sudden cardiac death (SCD). The ER pattern may be variable and J waves can be very small during normal sinus rhythm, bigger during increased vagal tone and bradycardia, and still further accentuated following successive extrasystoles, and biggest after compensatory pauses that can cause short-long-short sequences and induce VT/VF.2,10,11 However, overall, considering the high prevalence of an ERS in the general population, the association of ER patterns with the pathogenesis of VF is not certain and thus it is an insensitive sign for increased risk of SCD.

One should consider elevated risk of death from cardiac causes/arrhythmia in a person with ERS pattern if there is:
1. association with unexplained syncope or unexplained family history of SCD,
2. J point or ST-elevation ≥0.1 mV in the inferior leads with horizontal or descending ST segment,
3. prominent and/or transient J-wave augmentation, giant J waves,
4. association of ERS with increased indices of dispersion of repolarization,
5. association of ERS with closely coupled premature beats.

Abbreviations

ER = early repolarization
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SCD = sudden cardiac death
VF = ventricular fibrillation
VT = ventricular tachycardia

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Contrariwise, rapidly ascending ST segment after the J point, which is the dominant ST pattern in healthy athletes, seems to be a benign variant. Furthermore, early repolarization pattern in the anterior precordial leads is considered to have a benign prognosis. At present, patients with ST-segment elevation only (without J waves) or patients with a J wave and rapidly ascending ST-segment could be considered to have favorable prognosis. Otherwise, accurate prediction of the arrhythmic risk is practically impossible in asymptomatic ambulatory subjects with an ERS pattern.

REFERENCES