Early Repolarization and Sudden Cardiac Death Due to an Acute Coronary Event

Tikkanen et al. propose a significant and independent association between inferior/lateral ECG early repolarization (ER) patterns and sudden cardiac death (SCD) during an acute coronary event—an observation mainly based on the inclusion of ER pattern in multivariate predictive models for the occurrence of SCD, even after adjustment for several variables with usual prognostic value.

Recent/new investigation has claimed a role for ER in the pathogenesis of idiopathic ventricular fibrillation or cardiac death in the community-based general population and in patients with ischemic or nonischemic chronic heart failure. ER (in particular, notching in the inferior leads) has also been shown to increase the risk of life-threatening ventricular arrhythmias in patients with coronary artery disease, even after adjustment for left ventricular ejection fraction.

Now, Tikkanen et al. add invaluable data to their already renowned contribution to this topic. However, their findings justify a few considerations:

1. Significant differences were found between cases and controls, suggesting potential biases. In fact, control patients were more often medicated with statins, β-blockers, and angiotensin-converting-enzyme inhibitors/angiotensin II receptor antagonists, which are known to reverse remodeling processes and reduce mortality and SCD. Also, it is likely that antithrombotic therapies and revascularization procedures reduced overall mortality in the control group. A reliable comparison would only be possible if the control group had not been treated during the acute event (ie, without hospital care).

2. Some studies have suggested that fragmented QRSs and ER patterns may actually represent delayed depolarization of those segments with heterogeneous scar tissue, irrespective of its cause.

3. Remote myocardial scarring constitutes a potential anatomic and functional substrate for reentrant ventricular arrhythmias and thus may associate with a poorer prognosis. Although prior myocardial infarction was more common in the control group, the extent of myocardial fibrosis and overall left ventricular function are unknown and Q waves were similarly prevalent between groups. Accordingly, contrary to the authors’ statement, peri-infarction block caused by inhomogeneous remote scar tissue and translated as inferior ER pattern may still occur.

4. Information on the prevalence of ER patterns in anterior precordial leads is not provided. As this particular pattern is still considered a mostly benign one (not refuted by any study to date, to our knowledge), a potential (coincidental?) association between its isolated occurrence and SCD would challenge the main findings of this study.

5. Assuming that ER pattern may also represent transmural dispersion of repolarization, it could be additive to the electrophysiological mechanisms potentially involved in the highly arrhythmogenic early ST-segment elevation myocardial infarction period, potentiating the risk for phase 2 reentry and polymorphic ventricular tachycardia/ventricular fibrillation. This could explain a potentially higher risk of SCD in patients with ER patterns and acute ischemia. However, the authors did not explain why the presence of ST-segment elevation removed any potential malignant role of ER pattern in inferior/lateral leads. Is the malignant nature of ER dependent on the absence of an ST-segment elevation?

6. The authors could provide the coefficient of determination R² and the area under the curve for the prediction of SCD of their multivariate predictive models so that readers would know how well future outcomes might be predicted and the potential gain in discriminative performance with the addition of variable ER pattern.

In conclusion, ER in inferior/lateral leads may increase vulnerability to fatal arrhythmias during acute coronary events. Further studies are necessary to confirm this concept and before more aggressive primary prevention strategies are recommended.

Disclosures

None.

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