Association of early repolarization with long-term mortality and major adverse cardiac events in patients with ST-segment elevation myocardial infarction

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Early repolarization syndrome
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Early repolarization (ER) is a common electrocardiographic (ECG) finding manifested as elevated QRS-ST junction or J-point, that is either notched or slurred. Historically, ER was considered to be benign and was common in healthy and young individuals who had no structural heart disease. However, several studies have suggested an association between the presence of ER and fatal arrhythmic events.

In 1993, Aizawa et al. [1] reported the first case of idiopathic ventricular fibrillation (VF) with prominent ER on 12-lead ECG, which showed great variation. In this report, prominent ER was associated with the occurrence of spontaneous VF. Several papers have since reported cases of idiopathic VF associated with ER [2]. In an experimental study, Gussak and Antzelevitch reported the significance of ER as an arrhythmogenic substrate [3]. They reported that the depression of the epicardial action potential plateau caused the augmentation of the transmural voltage gradient, which is responsible for the development of ER with ST-segment or J-point elevation. This proposed mechanism is similar to that in Brugada syndrome.

In 2008, Haïssaguerre et al. were the first to report an association between a history of idiopathic VF and the presence of ER in a multicenter, case–control study [4]. In this study, 206 case subjects with idiopathic VF who had a history of resuscitation after cardiac arrest and 412 subjects without apparent heart disease were evaluated. The authors found that the prevalence of ER was significantly higher in patients with idiopathic VF than that in control subjects. In addition, during a follow-up period of 61 months, the incidence of VF episodes recorded with an implantable cardioverter defibrillator was significantly higher in VF patients with ER than that in those without ER. Using a large population-based study including 10,864 middle-aged subjects, Tikkanen et al. showed that the presence of ER in the inferior leads is associated with an increased risk of death from cardiac causes in the general population [5]. Because Brugada syndrome and ER syndrome showed several similar characteristics, Antzelevitch et al. proposed the term J-wave syndrome, which collectively describes Brugada syndrome and ER syndrome associated with inherited arrhythmia substrate and ischemic heart disease-related VF associated with acquired arrhythmia substrate [6]. They reported that the anatomical locations responsible for main electrophysiological manifestations are the right ventricle (RV) in Brugada syndrome and the left ventricle (LV) in ER syndrome. In previous studies, we reported that the location associated with ECG manifestation and arrhythmia substrate in Brugada syndrome is the epicardial myocardium of the RV outflow tract [7,8]. We introduced an electrical guidewire into the conus branch of the right coronary artery for RV epicardial mapping. Using a similar method, we first reported the case of idiopathic VF with inferior and lateral ER in which LV epicardial electrogram was recorded (Fig. 1) [9]. We could record prominent J waves and potentials after the QRS complex in the lateral LV epicardium, but not in the endocardium of the opposite area. These features were accentuated with pilsicainide administration but diminished with constant atrial pacing and isoproterenol administration. The epicardial J wave coincided with ER on ECG. VF was induced with programmed stimulation but only in the lateral LV epicardium. Thus, LV epicardial myocardium might be associated with arrhythmogenesis in this ER syndrome patient.

The significance of ER in ischemic heart disease patients was also reported in several papers. Patel et al. showed that, especially in the inferior leads with notched type, is associated with an increased risk of fatal ventricular arrhythmias irrespective of LV ejection fraction in patients with coronary artery disease and prior myocardial infarction [10]. Naruse et al. evaluated 220 consecutive patients with ECG recording before the development of acute myocardial infarction (AMI), and found ER as a predictor of VF occurrence in the early phase of AMI [11].

In this issue of the Journal, Özcan et al. provide new clinical information about the significance of ER in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous intervention (PCI) [12]. They evaluated 521 consecutive patients with acute STEMI who underwent primary PCI and examined the ECG recorded during the initial diagnosis. ECG analysis showed that ER was present in 61 patients (11.7%) with the following types: notched (14 patients), slurred (10 patients), and only J-point elevation (37 patients). During a follow-up period of 21.1 ± 10.2 months, major adverse cardiac events (MACE) were observed in 85 patients, death was observed in 26 patients, and
were not recorded within the endocardium at the opposite area. The J wave and the potential after the QRS complex (arrow) and potential after the QRS complex (arrow head) were observed from the left lateral coronary vein. The J wave and bipolar (Epi 3–4, Endo 2–3) electrograms at lateral coronary vein of the LV epicardium and at the opposite area within the endocardium. Prominent J (Epi 4, Endo 2) and bipolar (Epi 3–4, Endo 2–3) electrograms at lateral coronary vein of the LV epicardium and at the opposite area within the endocardium. Prominent J wave (arrow) and potential after the QRS complex (arrow head) were observed from the left lateral coronary vein. The J wave and the potential after the QRS complex were not recorded within the endocardium at the opposite area.

Fig. 1. (A) Electrocardiogram in a 42-year-old male patient with inferior and lateral early repolarization (arrow) and an episode of ventricular fibrillation. (B) Electrograms at epicardium (Epi) and endocardium (Endo) of left ventricle (LV). Unipolar (Epi 4, Endo 2) and bipolar (Epi 3–4, Endo 2–3) electrograms at lateral coronary vein of the LV epicardium and at the opposite area within the endocardium. Prominent J wave (arrow) and potential after the QRS complex (arrow head) were observed from the left lateral coronary vein. The J wave and the potential after the QRS complex were not recorded within the endocardium at the opposite area.

Modified from Nakagawa et al. [9] with permission.

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References


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