CASE REPORT

“Nonsignificant” early repolarization pattern on postresuscitation ECG as a harbinger of impending electrical storm

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Abstract

We report a 55-year-old man who was resuscitated from out-of-hospital cardiac arrest and subsequently developed three episodes of ventricular fibrillation (VF) on the same day. Early repolarization (ER) pattern was not significant (<0.1 mV) on postresuscitation ECG. However, ER pattern became evident (0.25 mV) before the onset of VF and then completely disappeared. The unusual dynamics of ER pattern observed in the present case could be called “masked” ER syndrome.

KEYWORDS

dynamics, Early repolarization; J-point amplitude

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Early repolarization syndrome (ERS) is diagnosed in the presence of J-point elevation ≥ 0.1 mV in ≥ 2 contiguous inferior and/or lateral leads of a standard 12-lead ECG in a patient resuscitated from otherwise unexplained ventricular fibrillation (VF) or polymorphic ventricular tachycardia (Priori et al., 2013).

A previously healthy 55-year-old man, who had no clinical history of syncope and no family history of unexplained sudden death, developed loud snoring followed by agonal respiration during sleep and was found unresponsive by his wife. She called emergency medical service (EMS) and initiated chest compression (at 05:43 a.m.). The EMS team found the patient to be in VF (at 05:50), which was successfully defibrillated (at 05:55). An admission electrocardiogram (ECG) demonstrated sinus rhythm (90 bpm) with small terminal QRS notching with its amplitude <0.1 mV (i.e., nonsignificant ER pattern) in the lateral leads (Figure 1) (06:15). An echocardiogram did not show regional wall motion abnormality. Laboratory evaluation demonstrated normal serum potassium level of 3.6 mEq/L (normal, 3.5–4.8), mild metabolic acidosis of PH 7.3 (normal, 7.35–7.45), elevated lactate of 41 mg/dl (normal, 4.5–14.4), and slight elevation of liver enzymes. A therapeutic hypothermia protocol was initiated immediately afterward. An immediate coronary angiogram revealed normal coronary arteries. The patient was initially diagnosed with “idiopathic” VF and treated with transdermal isosorbide dinitrate

FIGURE 1 (a) Admission 12-lead electrocardiogram. 1-B: Close-up illustration of leads I, II, and V6. Note the “nonsignificant” small QRS notch with its amplitude < 0.1 mV (red arrows)

FIGURE 2 Rhythm strips of leads II and modified V5 obtained at the onset of ventricular fibrillation. Note the prominent QRS notch with its amplified magnitude of 0.25 mV (red arrows) in the both leads

and intravenous magnesium. After admission to cardiac care unit, he developed 3 episodes of VF requiring emergency defibrillation at 10:32, 10:57, and 13:22. Just after the second episode of VF, continuous infusion of nifekalant (a pure potassium channel blocker), isosorbide dinitrate, and potassium chloride was initiated. However, the patient developed the third VF. Rhythm strips of leads II and modified V5 recorded at the onset of the third VF demonstrated prominently amplified QRS notching with its amplitude of 0.25 mV (Figure 2). Twelve-lead ECG obtained at immediately after the third VF episode confirmed the amplified QRS notching and increased distribution of the leads with the ER pattern (Figure 3a). However, ECG recorded at 24 hr after the last VF episode showed an absence of significant ER pattern (i.e., QRS notching with its amplitude <0.1 mV) (Figure 3-B). Moreover, the QRS notching completely disappeared on ECG taken at 48 hr after the last VF episode (Figure 3c). On hospital day 13, ergonovine provocative test for coronary spasm was negative and intravenous pilsicainide (a pure sodium channel blocker) challenge

did not provoke Brugada or ER pattern. Thus, a final diagnosis of ERS was made. The patient received an implantable cardioverter–defibrillator and was discharged from the hospital without neurologic sequelae. The patient has not experienced VF or appropriate ICD therapy for 8 years. Moreover, 12-lead ECG taken every 3–4 months during the follow-up period showed absence of QRS notching.

In our patient, J-point elevation (amplitude of the QRS notching) was not significant (<0.1 mV) on postresuscitation ECG. Moreover, the QRS notching became evident only before and just after VF and then completely disappeared. The unusual dynamics of ER pattern observed in the present case could be called “masked ERS”. Haïssaguerre et al demonstrated an increase of J-point amplitude from 2.6 ± 1 to 4.1 ± 2 mm (mean $\pm$ SD, $n = 18$) before the onset of VF (Haïssaguerre et al., 2008). Moreover, transient ER pattern associated with hypokalemia (Myojo et al., 2012) or myocardial ischemia (Jastrzebski & Kukla, 2009) has been reported, although neither hypokalemia nor ischemia was observed in our patient. Thus, the

FIGURE 3 (a–c) Twelve-lead electrocardiograms obtained immediately after (a), 24 hr after (b), and 48 hr after (c) the last episode of ventricular fibrillation. (a) Note the amplified QRS notch followed by descending ST segment in leads V1–5 and the wide distribution of the notch, which appeared in all leads except for lead III (red arrows). Note the QTc prolongation of 491 ms due to nifekalant (a pure potassium channel blocker). (b) Note the decrease in amplitude of QRS notch (red arrows) and in its distribution range. QTc = 502 ms. (c) Note the absence of early repolarization pattern (dashed arrows) and the shortened QTc after discontinuation of nifekalant (QTc = 476 ms)

amplification of J-point magnitude may portend a higher risk for VF and transient change in J-point amplitude is more like a rule rather than an exception. Besides, it should be noted that some ERS patients might exhibit ER pattern only during arrhythmic period, as in the present case. One possible explanation for the transient ER pattern is the sensitivity of J-point amplitude to plasma catecholamine level, which may rise markedly with cardiac arrest and attempted resuscitation. Clinical studies demonstrated that isoproterenol completely eliminates ER pattern in some ERS patients and therefore suggested the acute use of isoproterenol for suppression of recurrent VF (Haïssaguerre et al., 2008; Nam et al., 2010). Thus, J-point amplitude could decrease to nonsignificant level (<0.1 mV) on periresuscitation ECG in some ERS patients with high sensitivity of ERS-causing cardiac ion channels to catecholamine.

Our case suggests that absence of significant ER pattern on the postresuscitation ECG does not guarantee an exclusion of ERS. Thus, the “masked ERS” may be misdiagnosed as idiopathic VF. Careful monitoring of 12-lead ECG, especially before and after VF, is important to make a correct diagnosis, which can lead to appropriate therapies such as isoproterenol or quinidine (Haïssaguerre et al., 2008; Nam et al., 2010).

CONFLICT OF INTEREST

The authors have declared no conflicts of interest.

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