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Short communication

# Ischemia with marked ST elevation or J-wave syndrome?

Ricardo Cardona-Guarache, MD, MPH *, Vasanth Vedantham, MD PhD, Melvin M. Scheinman, MD

*Division of Cardiology, University of California San Francisco, San Francisco, CA, United States of America*

## Case presentation

A 55-year-old male with no prior history of heart disease presented to the emergency department of an outside hospital after an episode of bradycardic cardiac arrest. Rhythm strips of the event were unobtainable. After resuscitation, a 12 lead ECG was obtained (Fig. 1A), and a transthoracic echocardiogram, a coronary angiogram, and a cardiac MRI were performed, which were all normal. He had no family history of sudden cardiac death. A dual-chamber pacemaker was implanted, and he was then referred to our facility due to episodes of ventricular tachycardia (VT).

In our institution, we upgraded his pacemaker to an implantable cardioverter defibrillator (ICD), and we performed genetic testing, which showed no pathogenic mutations in an extensive panel including those associated with J-wave syndrome. The patient had spontaneous ST-segment elevations (Fig. 1B) that preceded recurrent episodes

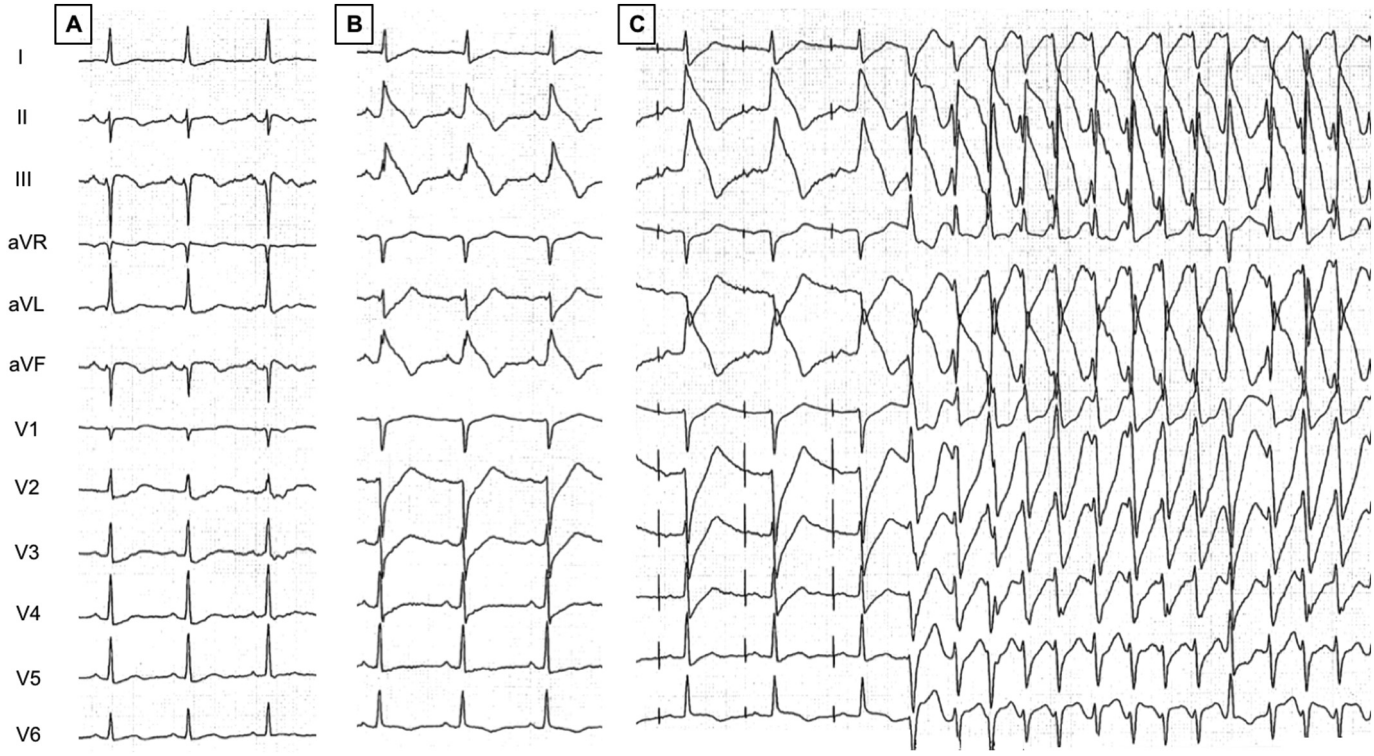


**Fig. 1.** 12-lead ECG of (a) baseline, (b) spontaneous ST-segment elevations that preceded ventricular tachycardia, and (c) ventricular tachycardia with a right bundle branch block morphology and right-inferior axis.

* Corresponding author at: University of California San Francisco, 500 Parnassus Ave, MUE 434, San Francisco, CA 94143-1354, United States of America.
*E-mail address:* ricardocardona08@gmail.com (R. Cardona-Guarache).

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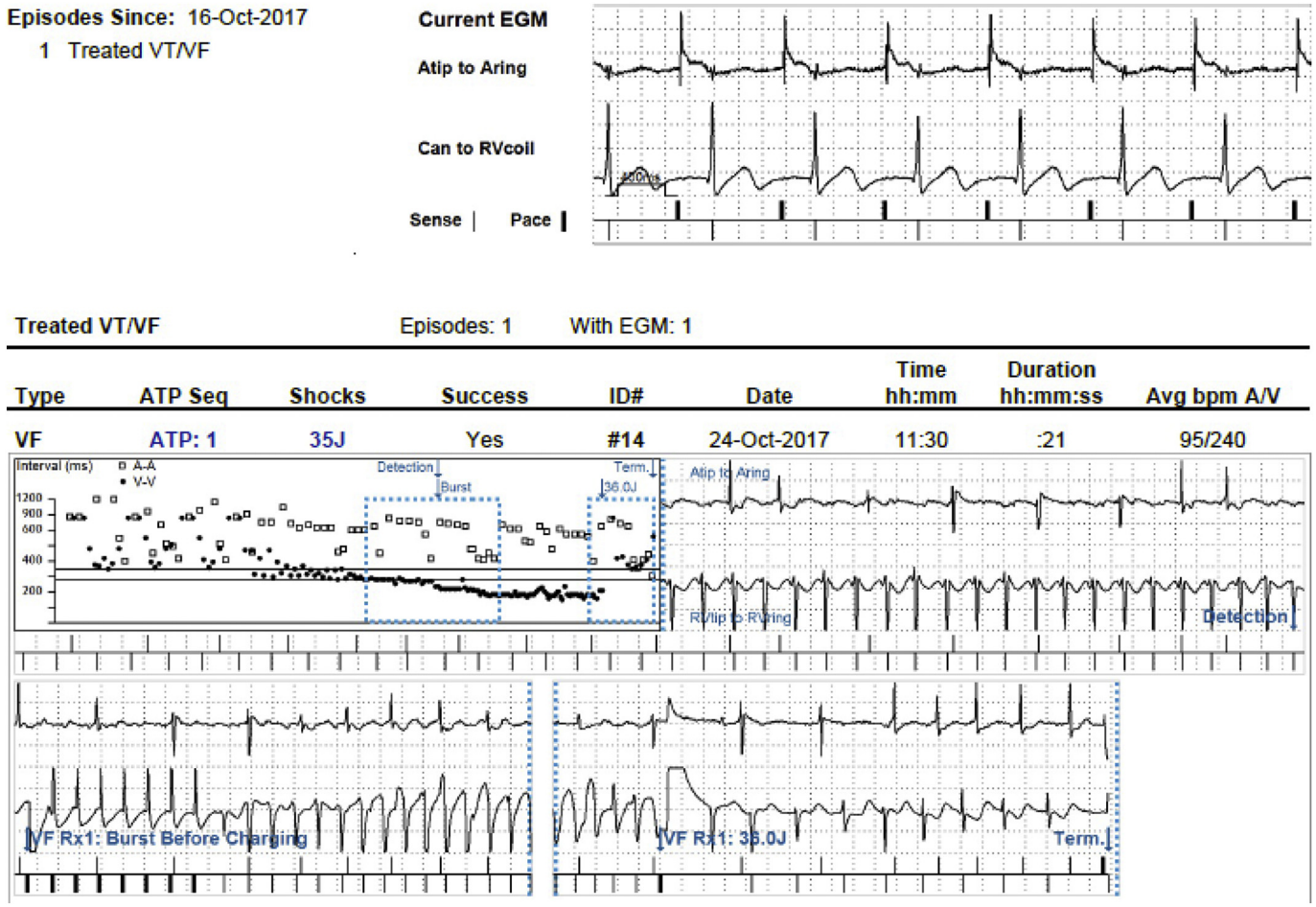


**Fig. 2.** Interrogation of implantable cardioverter-defibrillator.

monomorphic VT. The VT was relatively narrow and had a right bundle branch block morphology with a right-inferior axis, suggestive of a fascicular tachycardia from the left anterior fascicle (Fig. 1C). The arrhythmia often terminated spontaneously with a resolution of the J-point elevations observed in the inferior leads. However, on one occasion the VT deteriorated into VF resulting in an ICD discharge (Fig. 2). We administered sublingual nitroglycerin, which was ineffective, and performed a coronary angiogram with acetylcholine challenge that was negative. Procainamide challenge was negative for Brugada pattern.

The patient's downsloping ST-segment elevations in inferior leads were consistent with J-wave syndrome (i.e., early repolarization syndrome). Likely, the initial presentation of cardiac arrest was triggered by bradycardia because at slower heart rates there is a more significant contribution of the $I_{to}$ and $I_{KATP}$ channels to the slope of the "spike and dome" of the action potential (and less contribution of the sodium and calcium channels). Based on experimental models, Gussak and Antzelevitch first proposed the arrhythmogenic potential of early repolarization syndrome [1]. Later, Haïssaguerre et al. observed an association between patients with sudden cardiac arrest from arrhythmia and early repolarization and without structural heart disease [2]. Most recently, Riera et al. showed similar findings in another patient [3].

Quinidine reduces repolarization abnormalities via its $I_{to}$-blocking properties [4]. We initiated treatment with quinidine gluconate 325 mg orally three times a day that resulted in a prompt reduction of episodes of VT. VT episodes were controlled entirely at a dose of 650 mg orally three times a day. He was discharged on quinidine gluconate with normalization of his ECG and no recurrence of VT after an 18-month follow-up. There is limited data showing that quinidine is successful in decreasing VF episodes in patients with J-wave syndrome [5]. Monomorphic VT involving the fascicular system has not been reported in J-wave syndrome. Our case is unique in that we show that the J-wave syndrome may result in monomorphic VT responsive to quinidine.

## Disclosures

The authors have no conflicts of interest to disclose relevant to this manuscript.

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