

Transient J Wave after Resuscitation from Ventricular Fibrillation

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ABSTRACT

Although early repolarization is a common benign finding, it is sometimes associated with idiopathic ventricular fibrillation, turning necessary the identification of the two possibilities. J wave can be a sign, a syndrome, or it can be associated with idiopathic ventricular fibrillation. Its dynamics and overlapping complicate its diagnosis.

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Key words >

Ventricular fibrillation; Sudden cardiac death; Cardiopulmonary resuscitation

Abbreviations >

AECG Electrocardiography
VF Ventricular fibrillation

SD Sudden death
ER Early repolarization

INTRODUCTION

Idiopathic ventricular fibrillation (VF) is a cause of sudden cardiac death (SD) in patients without documented structural heart disease (excluding known channelopathies), which has been associated with J wave and short QT interval. (1-3)

Early repolarization (ER) or J wave is a diverse ECG pattern, featuring a hump or notch between the end of the QRS complex and the beginning of the ST segment, or featuring end QRS slurring with or without ST-segment elevation. The location, width and height may vary among patients. There is a tendency for the J wave to be accompanied by a short QT interval. (1-3)

J wave occurs in 2-5% of the population (more likely in males, athletes and young subjects). It was considered a common and innocent finding, but it has recently been associated with syncope and SD. It is very important to differentiate between benign and arrhythmogenic findings, and to know how to proceed with asymptomatic patients. (1-3)

CASE REPORT

This is the case of a 38-year-old woman, with untreated mild hypertension and no family history of SD. A week before the episode, she consulted for palpitations (isolated, coupled, not too premature ventricular extrasystoles, presenting different shapes and with left bundle branch block morphology), and with no other abnormality (Figure 1)

Sudden death occurred at 7 a.m. and the patient received immediate cardiopulmonary resuscitation at a local health center near her home. Ventricular fibrillation was detected, which reversed with external electric shock (300 joules), external cardiac massage and other routine procedures. Calcium was not administered, and there were no neurological sequelae. The ECG tracing taken 10 minutes after the event (Figure 2) showed sinus rhythm, prominent J wave (4 mm magnitude) in DII, DIII and aVF (decreasing during the first hour after the episode and disappearing five days later), and short QT interval (not appearing afterwards). Couplet and triplet ventricular extrasystoles persisted (Figures 2 & 3). The 24-hour Holter showed coupled extrasystoles (now with short interval), of different shapes, without J wave or short QT interval.

Physical examination and blood tests (electrolytes, enzymes, acid-base equilibrium) were normal, and cardiac tests ruled out structural cardiomyopathy: telecardiogram, echocardiogram, and stress test were normal. Coronary artery disease was not considered as the patient presented normal coronary angiography, no ischemic-necrotic changes in ECG and no abnormal enzymatic values.

Other causes of the ER pattern were also ruled out: hypothermia, hypercalcaemia, brain damage (neurological assessment, electroencephalogram and computed tomography were normal), vasospasm angina (no angina and ECG abnormalities lasted more than 30 minutes), and Brugada syndrome (negative IV

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Fig. 1. Baseline tracing at 25 mm/s, 1 cm = 1 mV. ECG recorded a week before ventricular fibrillation and resuscitation from sudden death. Isodiphasic, flat T wave, in aVF. Coupled ventricular extrasystoles, not too premature, presenting different shapes in the same lead, with left bundle branch block morphology. Neither J wave nor short QT interval is observed.



Fig. 2. Tracings at 25 mm/s, 1 cm = 1 mV. Episode of ventricular fibrillation and ECG at 10 minutes, an hour, and five days after the event. Strips, from top to bottom show: first - A) Ventricular fibrillation causing sudden death; second - B) Ten minutes after the event and the external electric shock, sinus rhythm with prominent J wave (4 mm magnitude) in DII, DIII and aVF (which did not exist), and short QT interval of 280 ms; third - C) An hour later, short J wave, ventricular extrasystoles of up to seven complexes, not too premature, and with different shapes in the same lead. An episode of self-limiting torsade de pointes and normal QT interval is observed; fourth D) Five days later, coupled ventricular extrasystoles, with different shapes in the same lead. There is sinus tachycardia; neither J wave nor short QT interval is observed.

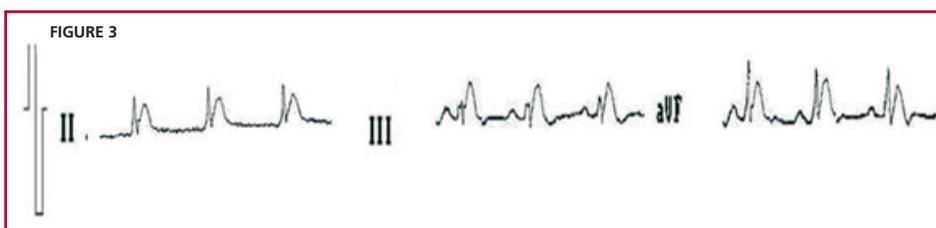


Fig. 3. Tracing at 25 mm/s, 1 cm = 1 mV, recorded several minutes after ventricular fibrillation. Detail of leads showing prominent J wave after the episode of ventricular fibrillation.

procainamide test). A cardioverter defibrillator was implanted. Electrical stimulation was performed during device implant in order to program it and test the efficacy of shocks.

Quinidine was prescribed and then discontinued due to adverse gastrointestinal effects. There has been no relapse at one-year follow-up.

DISCUSSION

We present a female patient with idiopathic VF as the cause of SD, associated with transient electrical signs (ER and short QT interval) related to malignant ventricular arrhythmias coexisting with primary VF. These signs may be prognostic markers of relapses and electrical storm or they may be associated with fibrillation. Although it cannot be confirmed that ER caused VF; its occurrence after VF is unexpected (not attributable to the arrest, as there was immediate recovery and there were no electrolyte changes or acid-base imbalance). Moreover, the finding persisted but was of smaller magnitude and afterwards disappeared.

We followed the National Registry of Sudden Death (2000-2010) in subjects with no documented structural heart disease (long QT, short QT and Brugada syndromes, idiopathic VF, and others). This is our first case of idiopathic VF with J wave and short QT.

ECG immediately prior to SD and to cardioversion is not available, which would have been important because of the changing nature of this phenomenon.

Ventricular extrasystoles, a week prior to the episode may have triggered the malignant arrhythmia, and they also occurred later, with repetitive and different shapes. Holter monitoring revealed extrasystoles with short coupling intervals, possibly triggering idiopathic VF and ER. We have not performed ablation in these patients, which is another therapeutic option. Although the relationship between extrasystole frequency and complexity and the risk of arrhythmia is not always a predictor, we believe this was the case in our patient. The degree of premature ventricular extrasystoles was low; however, a subsequent Holter revealed they had short coupling intervals without eliciting VF, indicative of their variability.

The J wave is a deflection immediately following the QRS, appearing as a ST segment elevation equal to or greater than 0.1 mV, 1 mm, with a dome or hump morphology in the same direction as the R wave, though it may also be partially concealed in it, appearing as a J point elevation or ST segment elevation, marking the end of depolarization (QRS) and the beginning of repolarization (J, ST, T, U). (2-5) It appears in precordial inferior, lateral leads (V4-V6) or in several leads (V1-V3 leads should be excluded because it might correspond to Brugada syndrome, arrhythmogenic right ventricular dysplasia, or use of antiarrhythmic agents). (2)

Configuration, extent, amplitude, localization and

evolution of the J wave are studied in baseline ECG. The J wave is variable, difficult to record at the onset of the arrhythmia, when major changes generally occur and can be lost within depolarization. The QT interval does not increase with decreased heart rate. (3, 5)

Since resuscitation from SD episodes is low, it would be very important to identify patients at risk, and distinguish "innocent" (most patients) from arrhythmogenic J waves (risk marker for malignant ventricular arrhythmias, SD, recurrence and electrical storm). (3, 5-7)

J wave syndromes are a spectrum of phenotypic expressions (ER, Brugada syndrome, idiopathic VF, sudden unexplained nocturnal death, among the inherited causes), with some acquired varieties. They emphasize the epicardial action potential notch in different cardiac regions, and give rise to the prominent J wave and the substrate for phase 2 reentry which triggers arrhythmias. Genetic evidences of idiopathic VF and J wave have been characterized. (4, 7)

Our patient had type 2 ER, inferior or inferolateral, with higher risk for arrhythmias and electrical storm. (4)

The Haissaguerre syndrome, observed in one out of three patients, is the idiopathic VF with J wave in inferolateral leads with a propensity to shorter QT, arrhythmias, and electrical storm. (5)

We do not know whether the J wave existed before the VF episode; it was not present a week before the event, and its dynamic behavior hampers the diagnosis as there are no records closer to the event. It usually precedes and ends the arrhythmic episode, or it recurs. (2) In this case, it was a transient, primary electrophysiological phenomenon, with progressive reduction and disappearance of J waves, with a SD episode at its onset and prior ventricular extrasystoles with early coupling interval, which could have triggered the VF. There was a transient short QT and a brief J-Tpeak interval (100 ms) possibly linked to the ER (both involve transmural dispersion of repolarization). (8, 9)

There may be overlapping of primary arrhythmogenic entities: ER, short QT interval and idiopathic VF. Was the ER associated with idiopathic VF or did both of them and the short QT interval coexist? Is it a subpopulation of SD, a sign, a syndrome, an association? In idiopathic VF, it may represent greater malignancy rather than a different model. (7)

Sorgente presented a Brugada syndrome with J wave disappearance after an electrical shock; ours was the reverse case, with no convincing explanation. (8)

The role of electrical stimulation to determine risk and therapy is debatable, particularly in asymptomatic subjects, (1, 5) and the outcomes do not change the conduct.

An implantable cardioverter defibrillator, indicated in syncope and SD, was used and quinidine, which homogenizes transmural dispersion, blocks Ito

current, avoids relapses, electrical storm and supra-ventricular arrhythmias that could be misinterpreted by the device, was administered. (10)

What to do with ER subjects recovered from SD is clearly stated, but what approach to follow with asymptomatic patients is a complex decision.

RESUMEN

Onda J transitoria después de reanimación por una fibrilación ventricular

La repolarización precoz es un hallazgo electrocardiográfico benigno común, en ocasiones asociado a la fibrilación ventricular idiopática. Se presenta una mujer de 38 años sin cardiopatía estructural ni historia familiar de muerte súbita cardiaca, reanimada de episodio de fibrilación ventricular. Una semana antes tuvo extrasístoles ventriculares. Se detectaron la elevación del punto J, después del choque eléctrico, que disminuyó hasta desaparecer, y un intervalo QT corto transitorio. Se implantó un cardioversor-desfibrilador automático y se administró quinidina. Su evolución ha sido satisfactoria, sin recidivas. La repolarización precoz podría ser un signo, un síndrome o un marcador asociado a la fibrilación ventricular idiopática.

Palabras clave > fibrilación ventricular, muerte súbita cardiaca, Resucitación cardiopulmonar

Conflicts of interest:
None declared.

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