Ventricular fibrillation in loop recorder memories in a patient with early repolarization syndrome

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We report the first documentation of spontaneous ventricular fibrillation by a loop recorder in a patient with an ECG pattern of early repolarization (ER) in the inferior leads and presenting with syncope.

A 14-year-old boy was referred to our institution after four successive syncopes over the last few years. He did not present with any noticeable medical history or with family history of unexplained ventricular arrhythmias, syncope, or sudden cardiac arrest. Syncope always occurred post-exertion and appeared typically situational, with clinical feature clearly evoking vasovagal syncopes for three of them, but associated with seizures and cyanosis for the latter one. Resting ECG was considered unremarkable, with normal QT duration, except for an early repolarization (ER) in the inferior leads, with slurring of the QRS and an elevated J point together with horizontal ST segment and negative T wave in lead III (Figure 1). Although ER may be frequently observed in young patients, particularly in those engaged in sporting activities,1 the pattern observed in this patient was somewhat atypical and may correspond to what may be now considered as a marker for an increased risk of malignant ventricular arrhythmic events.2

Complete investigation remained unremarkable (echocardiography, cardiac magnetic resonance imaging, signal averaged ECG, provocative test with ajmaline, no conduction disturbances, no pre-excitation, no inducible supraventricular, or ventricular arrhythmia) except for Holter recordings and treadmill, which revealed monomorphic ventricular premature beats coming from the left ventricular outflow tract with long coupling interval and eliminated during exertion or isoproterenol infusion (therefore excluding catecholaminergic polymorphic ventricular tachycardia). Head-up tilt test induced a typical vasovagal syncope and the initial diagnosis was therefore that of vasovagal faints.

However, a few months later, the patient presented with a new syncope after exertion and also associated with seizures and cyanosis. Neurological investigations then established the diagnosis of idiopathic epilepsy, made on an abnormal electroencephalogram, and the patient was then treated with levetiracetam without any recurrence over a follow-up of 1 year.

However, because of the ER pattern, which was recently described as a potential marker for sudden cardiac death,2,3 especially with the present ECG features, an internal loop recorder (Medtronic™Reveal DX) was implanted.

Figure 1 Upper: 12 lead ECG showing the early repolarization pattern in the inferior leads (arrows) and the fractionated QRS with negative T wave in lead III (magnified view on the right side: the fractionation of the QRS was interpreted as a giant J wave). Lower: non-sustained ventricular fibrillation as recorded by the loop recorder (VS, ventricular sensed event by the device; FS, ventricular fibrillation sensing by the device, vertical values representing the instantaneous cycle length durations between successive ventricular events).
At the first follow-up consultation, interrogation of the device revealed an asymptomatic episode of non-sustained ventricular fibrillation (VF) lasting 5 s occurring during recreational exertion, while the patient was still on levetiracetam, and preceded by an increase in heart rate and ventricular bigeminy. The patient was then treated by quinine before receiving an implantable cardioverter defibrillator (ICD).

Although idiopathic VF episodes have been documented using loop recorders, to the best of our knowledge, this is the first reported documentation of spontaneous VF by a loop recorder in a patient implanted for syncopeces associated with an ECG pattern of ER. This case highlights the need for careful and long-term monitoring for such patients, particularly in case of syncope of unknown origin and/or familial case(s) of unexplained sudden cardiac death. The ECG pattern presented here is an additional reason for such a strict monitoring, while ICD implantation as a first choice therapy for ER cannot be currently recommended. The prevalence of ER pattern is high in the normal population and risk stratification in this setting remains problematic. Recently, the presence of horizontal or descending ST segment in the inferior or lateral leads has been shown to be associated with arrhythmic death in patients with ER, while fragmented QRS complexes have been correlated to malignant ventricular arrhythmic events in patients with early repolarization.

Conflict of interest: none declared.

References

CASE REPORT

A modified subcutaneous implantable cardioverter–defibrillator implant in a patient with a previous left ventricular epicardial defibrillation patch

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We describe a case of subcutaneous implantable cardioverter–defibrillator (ICD) implant in a patient with an existing epicardial defibrillation patch. Potential issues with shock vector shielding were overcome by a modification of the generator implant site and poor sensing were successfully managed by programming a sensing vector which excluded the generator.

Case

A 46-year-old woman with arrhythmogenic right ventricular (RV) cardiomyopathy and prior ventricular fibrillation (VF) arrest underwent implantation of a left pectoral endocardial implantable cardioverter–defibrillator (ICD) in 1998. This was complicated by device-related infection requiring system extraction. Surgical implantation of an abdominal ICD with epicardial atrial and right ventricular pace/sense leads and a single left ventricular (LV) epicardial defibrillation patch to the lateral wall of the LV was performed [Guidant CPI (UK) A76]. Subsequently, a fracture of the RV lead occurred, which was capped, and an endocardial RV pace/sense lead was placed via the right subclavian vein and tunneled to the abdominal generator. Further ICD generator changes occurred in 2004 and 2010. A routine ICD check in 2011 demonstrated a significant increase in defibrillation patch impedance from 68 to >200 Ω and electrical noise on the ‘far-field’ patch-can electrogram during provocation testing. Impedance trends demonstrated the rise had occurred 1 month after the last generator change. These indicated a conductor break in the ICD shock circuit. Surgical exploration found no visible fault with the lead connections and the leads were capped and generator was removed.

As a consequence of previous endocardial device infection and absence of a pacing indication, we elected to implant a subcutaneous ICD (S-ICD) (Cameron Health SQ RX 1010) with a tunneled S-ICD lead (Cameron Health QTRAK 3010) (Figure 1). This device has three available sensing vectors: ‘Primary’ vector from the proximal parasternal sensing electrode to device, ‘Secondary’ vector from the distal parasternal sensing electrode to device, and ‘Alternate’ vector from distal to proximal parasternal electrode (‘cold-can’). A pre-procedure sensing check using a patient screening tool (Cameron Health) was satisfactory in all vectors. There was a concern regarding electrical shielding of the S-ICD shock vector by the existing epicardial defibrillation patch. We therefore opted to position the generator in a lower-than-customary left lateral position (eighth intercostal space mid-axillary line) for the shock vector to avoid

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