

Early Repolarization Pattern at High Risk for Sudden Cardiac Death Unmasked by Exercise Test

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Introduction

Early repolarization is characterized by elevation of the QRS-ST junction (J point) in leads other than V1 through V3 on 12-lead electrocardiography [1]. It was thought to be an innocent ECG finding [2,3]. However, lately this pattern was suspected as a possible predictor for serious ventricular arrhythmias [4,5].

Case Report

A 32 years old male patient complained of palpitation and recurrent pre-syncope for 3 years.

The patient was investigated 6 months previously at another place for the same complaint. He underwent an electrophysiological study that was reported to be normal. The patient was treated by bisoprolol. The patient continued to have symptoms. He was referred to our center for further evaluation.

The clinical examinations as well as routine laboratory investigations were normal. Electrolytes as well as thyroid function tests were all normal.

The ECG showed normal sinus rhythm at a rate of 50 beats per minute (b/min) with some premature ventricular contractions (PVCs). The P-R and corrected QT (QTc) intervals were normal (QTc interval: 395 ms). There was no intraventricular conduction abnormality, pre-excitation or Brugada pattern. However there was ST elevation of around 1 mm with J wave mainly in the inferolateral leads (J-point elevation and notching in the terminal portion of the QRS complex) (Figure 1).

The patient's echocardiography was normal. A 24 hour ambulatory ECG recording revealed basic sinus rhythm with average heart rate between 50 and 70 with infrequent PVCs and few couplets. An exercise stress ECG was performed. The patient's resting ECG showed frequent PVCs with no other changes apart from inferolateral early repolarization (Figure 2). The patient was exercised according to Bruce protocol for 13 minutes, achieving a heart rate of 184 bpm and



Figure 1: Baseline 12 lead Electrocardiogram of the patient ST elevation of around 1 mm with J wave mainly in the inferolateral leads.

disappearance of the PVCs. Blood pressure increased from 130/80 mmHg to 140/80 mmHg. He was free of symptoms during the test. The test was terminated because of achievement of target heart rate response and absence of ST-T changes and arrhythmias. During the 1st minute of the recovery period when the heart rate was 153 b/min, while the QTc interval was within normal (443 msec), the J point in the inferior leads showed exaggerated elevation ≥ 2 mm and without any preceding symptoms, the patient developed a very early R on T PVC (Figure 2) that initiated ventricular fibrillation (VF) (Figure 3). This was immediately treated by defibrillation with 200 Joules biphasic shock. Later, Coronary angiography showed normal anatomy.

The patient was diagnosed as having idiopathic exercise induced VF and had an implantable cardioverter defibrillator (ICD) implanted.

Discussion

Ventricular fibrillation triggered by exercise test in patients with otherwise normal heart is very rare [6]. Our patient had evidence of early repolarization on his resting ECG and had VF immediately after exercise test. It occurred very early in the recovery period, it can't be explained by high vagal tone during recovery as the heart rate of 150/min and the absence of any vagal symptoms as nausea or vomiting

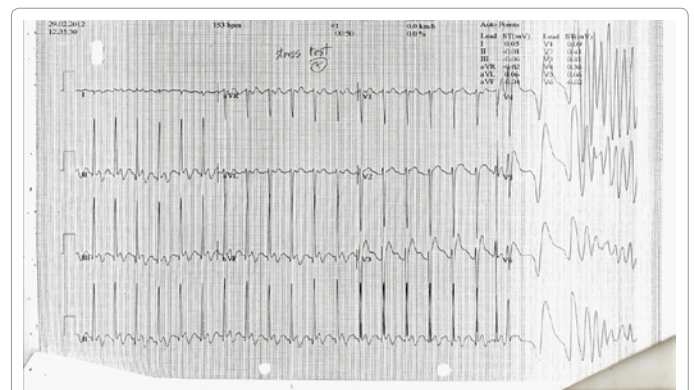


Figure 2: Ventricular fibrillation initiation by an R on T early ventricular ectopic beat during early recovery phase of the exercise test, this was terminated by a DC shock.

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Figure 3: ECG recording of Ventricular fibrillation recording just before its termination by Defibrillation (lead V2 was accidentally disconnected during patient arrest).

is against this assumption. The VF (Figure 3) was not preceded by bidirectional ventricular tachycardia (VT), therefore making catecholaminergic polymorphic VT unlikely, also it was not preceded by abnormal QTc or Brugada pattern. There was no other clinical or laboratory abnormality to predispose the patient to sudden cardiac death and the ECG showed exaggeration in the early repolarization just before the VF. This is the same phenomenon that Haïssaguerre has described before [4]. The exercise stress test unmasked our patient vulnerability for sudden cardiac death.

Although the prevalence of repolarization abnormalities in patients resuscitated from VF with no evident structural heart disease is higher than expected, these abnormalities are also common in young, healthy individuals. The sensitivity, specificity, and predictive accuracy of this finding are not clear [7]. The risk of cardiac arrest in asymptomatic patients with early repolarization is likely very low. It was reported by Wellens [3], that approximately 2-5% of the population demonstrates early repolarization on electrocardiography; this population mostly consists of men, young adults, athletes, and people of African American heritage.

A study by Haïssaguerre et al. [4] evaluated the clinical association between early repolarization in the inferolateral leads and idiopathic ventricular arrhythmias leading to syncope and/or sudden cardiac death. A case-control study involving 206 case subjects with a prior history of idiopathic ventricular fibrillation (IVF) showed that early repolarization occurred statistically more frequently in the subjects with IVF than the control subjects (31% vs. 5%, $P < 0.001$). Early repolarization pattern were more likely to be seen amongst male patients with lower baseline heart rates [4].

Tikkanen et al. [8] investigated the long-term outcome associated with early repolarization on the electrocardiogram. This evaluation was an extension of the data described by Haïssaguerre et al. Tikkanen emphasized the prognostic significance of the J point pattern in the inferolateral leads which were hypothesized to be more arrhythmogenic than the more commonly studied anterior precordial leads (leads V1 through V3). Based on their findings, early repolarization in the inferior leads appeared to be a strong predictor of death from cardiac causes or from arrhythmia than J-point elevation in the lateral leads. Thus, the pattern of early repolarization (ER) associated with highest risk seems to be global or inferolateral early repolarization with prominent J waves on the resting ECG [9]. In addition to the location of the early repolarization pattern, the amplitude of the J-point elevation had great prognostic value. There was a significantly higher risk of death from cardiac causes among subjects with a markedly elevated J point (> 0.2 mV) than among those with a more moderate elevation (≥ 0.1 mV) [8].

The mechanisms of ER-induced arrhythmias are incompletely understood. One possible scenario is that If ER is accelerated in certain myocardial cells, phase 1 notch of the action potential increases and/or all-or-none repolarization can occur, producing large voltage gradients. These voltage gradients can initiate arrhythmogenesis, either by propagation of the AP dome ("phase 2 re-entry") or via boundary currents analogous to "injury currents" in acute myocardial infarction [10] that raise ER cells to threshold and induce spontaneous activity.

Our patient was a young non athletic male who had history of recurrent dizziness and pre-syncope and an early repolarization pattern with inferolateral leads J point elevation more than 1 mm. The only test that unmasked the patient vulnerability to VF and sudden cardiac death was the exercise stress test. This test was not mentioned in previous studies in the assessment of these patients. We propose including this test in the evaluation of symptomatic patients with early repolarization or assessing its value in larger studies.

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