Ventricular Tachycardia during exercise following myocardial infarction: a scar-related mechanism?

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Under psychological stress, a 61-year-old man fainted and quickly regained consciousness, but with a slight head injury. He did not complain of symptoms when he exercised, but was not really physically active. Fifteen years ago, he had a myocardial infarction, then two years later, was diagnosed with congestive heart failure. Three years ago, he underwent a coronary angiography procedure. It showed occlusion of the left descending coronary artery and mild stenosis of the right coronary artery. Stress myocardial scintigraphy showed no ischemic areas requiring a revascularization procedure.

Baseline electrocardiogram (ECG) showed first-degree atrioventricular block and incomplete left bundle branch block. Severe dilation of the left ventricle was observed on the echocardiogram (end-diastolic diameter = 87 mm, ejection fraction = 0.24).

In order to rule out further damage to the coronary arteries, an exercise test was performed using a 30W/3min-step protocol. At the 60W step, the ECG showed sinus tachycardia with QRS widening from 120 to 160 msec (fig. 1A-1B). Although the test was stopped, a wide QRS tachycardia occurred. The patient briefly fainted, with complete regaining of consciousness in the supine position. The tachycardia rate was 155 bpm, with a left bundle branch block pattern and left-axis deviation (fig. 1C).

Atrioventricular dissociation was likely to be present on lead V1, consistent with ventricular tachycardia. What could be the mechanism responsible for this tachycardia ?

Electrophysiological study

Given the good hemodynamic tolerance, we performed an electrophysiological study. Two catheters were introduced through the right femoral vein. A 5F-hexapolar electrode catheter was positioned in the atrioventricular junction to record the His bundle potential. The potential of the right bundle branch could not be recorded using the distal dipole of the same catheter. Another 5F-bipolar catheter was positioned in the right ventricle. Intracardiac recordings confirmed complete atrioventricular dissociation. The His bundle electrogram showed a deflection between the ventriculograms. We stopped the tachycardia by pacing at a driven cycle length of 320 ms, but the tachycardia recurred after two sinus beats, with ventricular instability at the beginning of the cycle (fig. 2). Spontaneous variations in the V-V intervals were preceded by similar changes in the H-H intervals, leading to the diagnosis of bundle branch reentry. We stopped the ventricular tachycardia with rapid pacing. During sinus rhythm, the HV interval was found to be identical to the one measured during ventricular tachycardia (105 ms). Atrial pacing obtained an AV nodal Wenckebach phenomenon at a cycle length of 690 ms and did not induced ventricular tachycardia. Two extrastimuli delivered to the right ventricular apex easily induced clinical tachycardia. Due to the dysfunction of the left ventricle, we implanted an cardioverter defibrillator. Six ventricular tachycardia episodes occurred two months later during light exercise. Four episodes were short-lived, while one episode required cardioversion due to failure of antitachycardia pacing. The rate of the ventricular tachycardia was the same as for the

bundle branch reentry tachycardia. Therefore, we proceeded to ablate the right bundle branch. The patient remained asymptomatic in the following six months.

Discussion

Sustained bundle branch reentry has been reported as a mechanism of clinical ventricular tachycardia (1,2), typically occurring in patients with left ventricular dilation. The most common underlying cardiac disease is dilated cardiomyopathy, but this arrhythmia can be associated with other abnormalities such as valve disease, conduction system disorders, coronary artery disease, myotonic dystrophy or Becker muscular dystrophy (3). Ablation of the right bundle branch has been shown to be the treatment of choice (4-7).

In the case of ventricular tachycardia following a myocardial infarction, a scar-related mechanism is the rule. Although bundle branch reentry is not the most common mechanism of ventricular tachycardia occurring in patients with ischemic heart disease, it has been observed in patient with left ventricular dilation.

During electrophysiologic study, bundle branch reentry is usually induced by programmed ventricular pacing. This is the first report of ventricular tachycardia related to bundle branch reentry, initiated by rapid sinus rhythm during exercise.

One could assume that the exercise test, by increasing the sinus rate, was responsible for the complete anterograde left bundle branch block. This unidirectional block allowed bundle branch reentry, with depolarization of the right bundle branch, followed by retrograde conduction through the left bundle branch. In this case, rapid pacing of the atrium failed to induce clinical tachycardia. The Wenckebach phenomenon occurred at a slower rate at rest than during exercise, which explains the inability to induce complete anterograde left bundle branch block, which was necessary to induce bundle branch reentry. The reentry could have been induced by rapid pacing during isoproterenol infusion, which was not used during the electrophysiological study for safety reasons.

Complete bundle branch block is common in ECG in patients with bundle branch reentrant (BBR) tachycardia. This pattern does not imply a complete unidirectional anterograde block through the left bundle branch or is related to slow conduction. Therefore, bundle branch reentry cannot occur during sinus rhythm and can only be induced by premature ventricular beats. Moreover, right bundle branch ablation can be performed with a low risk for complete atrioventricular block. In the absence of complete bundle branch block on ECG, the role of functional His-Purkinje system abnormalities has been emphasized (8).

Touboul and co-workers (2) reported BBR induction by rapid atrial pacing. In their report, right bundle branch block (RBBB) occurred when the cycle was shortened to 320 ms. After the pacing stopped, a tachycardia started and presented the same RBBB pattern. Mizusawa et al. (9) confirmed that induction of BBR with a RBBB pattern is not uncommon using atrial pacing, which has also been shown to be a useful tool for assessing the diagnosis of BBR. Merino et al. (10) used atropine and isoproterenol to enhance AV anterograde conduction, allowing transient entrainment with orthodromically concealed fusion, which has not been observed in pure myocardial ventricular tachycardia. It should be noted that isoproterenol infusion may be hazardous in these patients with severe coronary artery disease and left ventricular dysfunction.

The post-pacing interval following entrainment by right ventricular apex stimulation has been proposed to suggest BBR but should not be considered without further criteria (11).

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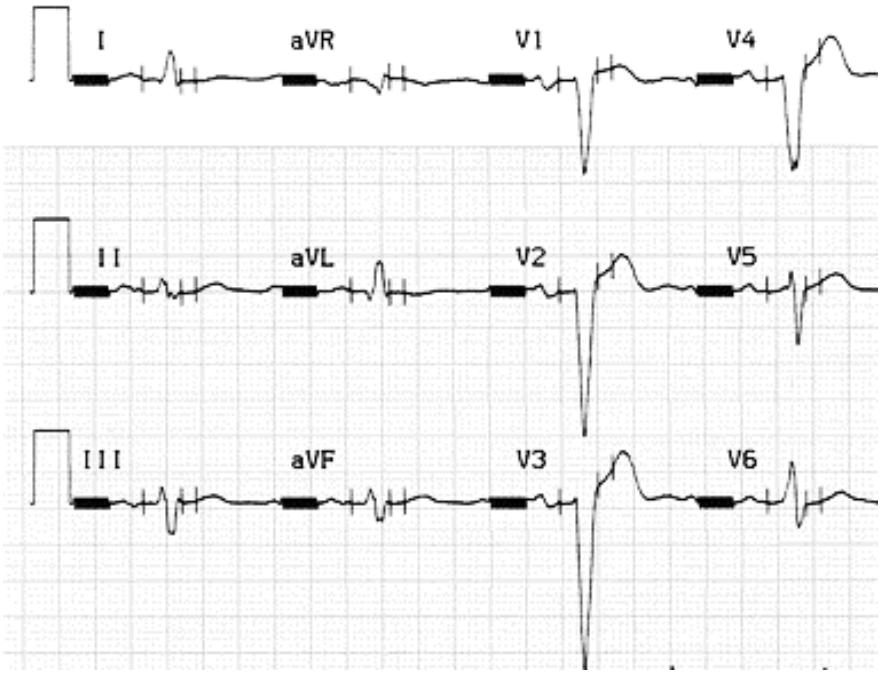
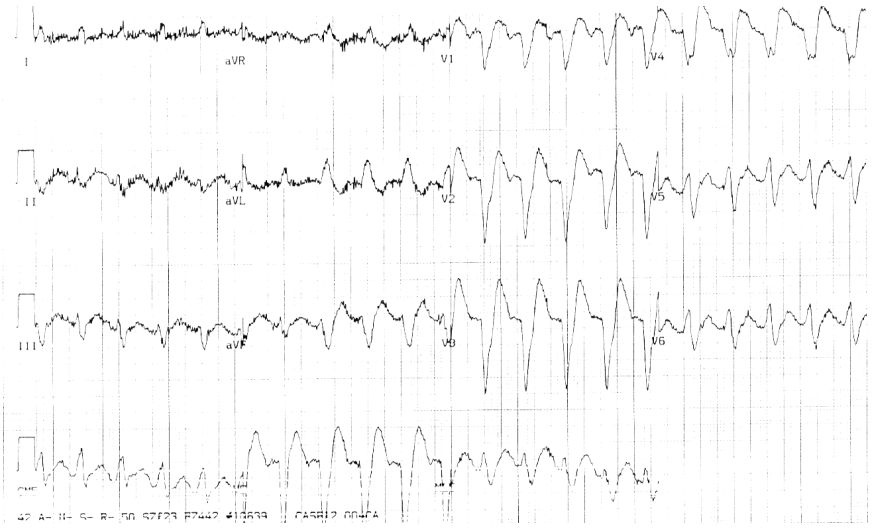


Figure 1: A- resting ECG: QRS width = 130 ms, HR = 71 bpm; B- QRS widening during exercise (QRS width = 190 ms, HR = 123 bpm); C- wide QRS tachycardia immediately after QRS widening.

A



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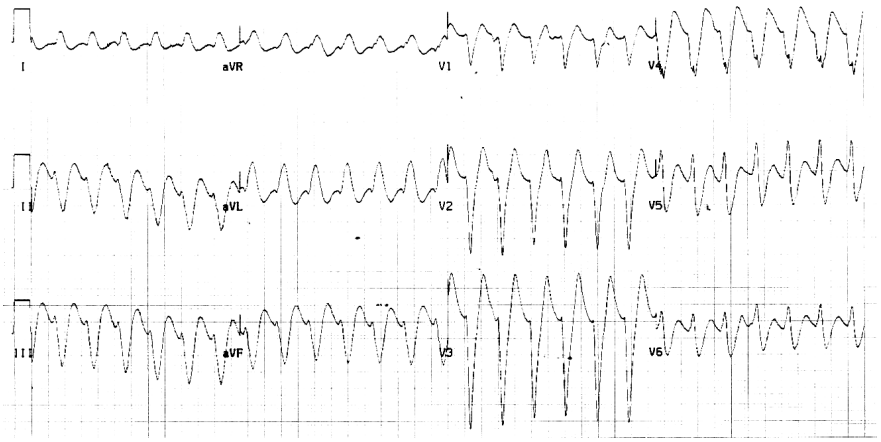

C

Figure 2: His bundle recording at the re-occurrence of tachycardia: spontaneous variations in V-V intervals preceded by similar changes in H-H intervals, with atrioventricular dissociation. HBE = His bundle electrode. RVA = right ventricular apex.