Brugada Syndrome Combined with Monomorphic Ventricular Tachycardia and Atrioventricular Nodal Reentrant Tachycardia

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Abstract

A 41-year-old man developed sustained monomorphic ventricular tachycardia (VT) with a left bundle branch block and inferior axis pattern during treadmill exercise concomitantly with unmasking of the typical Brugada electrocardiography (ECG) pattern. The typical ECG phenotype was provoked by a class IC drug. VT was not inducible with programmed electrical stimulation, but premature ventricular beat and non-sustained VT with the same morphology increased in frequency with isoproterenol treatment. Additionally, atrioventricular nodal reentrant tachycardia (AVNRT) was induced by electrical stimulation and VT and AVNRT were treated by radiofrequency catheter ablation.

Key words: Brugada syndrome, monomorphic ventricular tachycardia, exercise, pilsicainide, atrioventricular nodal reentry

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Introduction

Brugada syndrome (BrS) is a serious disease resulting from ventricular fibrillation (VF) occurring without evidence of gross structural heart disease (1). The typical Brugada electrocardiography (ECG) pattern may be unmasked by exercise, typically in the post-exercise recovery phase (2). Although seen infrequently, monomorphic ventricular tachycardia (VT) may combine with BrS (3-9) and may be induced by exercise concomitant to the induction of the typical ECG pattern (7).

We recently treated a patient with BrS who had typical Brugada ECG patterns and monomorphic sustained VT induced during peak exercise. Additionally atrioventricular nodal reentrant tachycardia (AVNRT) was induced by programmed electrical stimulation. VT and AVNRT were successfully ablated by the radiofrequency (RF) catheter technique. We herein report an unusual case of BrS with some discussion.

Case Report

The patient was a 41-year-old man with a past history that was non-contributory until incidences of sudden palpitations 2-3 times a year for the previous 2 years. He had no previous history of syncope or family history of sudden cardiac death. The palpitations appeared during the day and spontaneously terminated within minutes. He underwent an exercise test in another hospital and had a baseline ECG that was nearly normal; however, peak exercise induced monomorphic VT with a left bundle branch block (LBBB) and inferior axis morphology. VT terminated spontaneously, and the patient was transferred to our hospital in December 2012.

On admission, the patient was 167 cm in height and weighed 56 kg. His blood pressure and heart rate were 112/62 mmHg and 72 beats/min, respectively. The physical examination was non-contributory, and the complete blood cell counts, blood chemistry, serological tests and analysis of ar-

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Figure 1. Induction of monomorphic sustained VT during exercise. The baseline ECG (left) shows a slight deviation of the frontal electrical axis to the right and ST segment elevation in the precordial leads, which are not typical for Brugada syndrome. The exercise stress test was performed according to Bruce's protocol, and monomorphic VT with a left bundle branch block and inferior axis pattern was induced (middle). VT terminated spontaneously after the cessation of exercise. Coved-type ST-T change is evident in leads V₁ and V₂ (right). The J-point and ST elevation gradually returned to the baseline level during post-exercise recovery.

Figure 2. Provocation of the Brugada ECG pattern by pilsicainide. The baseline ECG was nearly normal (left), and administration of pilsicainide induced the typical pattern seen in patients with Brugada syndrome (right).

terial blood gas were normal. The ECG on admission showed a normal sinus rhythm with normal PR and QT in-

tervals. Echocardiography showed normal cardiac chambers and function with a left ventricular ejection fraction of 60%.

ECG during exercise and drug testing

The patient's baseline ECG was not typical for BrS, but VT with LBBB and inferior axis at a rate of 176 beats/min was induced during exercise and spontaneously terminated. Immediately prior to and after VT, coved type ST segment elevation was observed and returned to baseline during the post-exercise recovery phase (Fig. 1).

We then attempted a provocation test for BrS and pilsicainide (50 mg i.v. for 10 minutes), a class IC drug, induced a typical Brugada ECG pattern (Fig. 2).

Catheterization and electrophysiology study

After informed consent was obtained from the patient, a cardiac catheterization and electrophysiology study were undertaken. The catheterization showed normal cardiac anatomy and function as well as normal coronary arteries. The induction of monomorphic VT was attempted by programmed stimulation giving up to 2 extrastimuli at two basic paced cycle lengths: 600 ms and 400 ms from the apex and the outflow tract of the right ventricle. When VT was not inducible, isoproterenol was given to increase the sinus rate by 20% and the electrical stimulation was repeated. Pre-

Figure 3. Induction of atrioventricular nodal reentrant tachycardia (AVNRT) and mapping of the origin of ventricular tachycardia (VT). (A) By rapid pacing at the right ventricle at a cycle length of 280 ms, narrow QRS complex tachycardia was repeatedly induced. The HA interval and AH interval were 65 ms and 180 ms, respectively. Tachycardia became non-inducible after catheter ablation at the earliest activation site of the atrium near the coronary sinus os. (B) The ablation site of the premature ventricular beat (PVB) showing identical QRS morphology to sustained VT is depicted. Radiofrequency currents were delivered to the earliest PVB site of activation, and PVB and non-sustained VT disappeared after ablation.

mature ventricular beat (PVB) and VT with LBBB and inferior axis occurred sporadically; however, they were not inducible with programmed ventricular stimulation. After isoproterenol administration, PVB and non-sustained VT were increased in frequency, and AVNRT was reproducibly induced by rapid pacing from the right ventricle as follows: cycle length, 260 ms; AH interval, 65 ms; and HA interval, 180 ms (Fig. 3A). For the induction of VF, up to three extrastimuli with coupling intervals ≥ 180 ms were given from the apex of the right ventricle (260 ms, 200 ms and 200 ms) following a basic paced cycle length of 400 ms, and VF was induced and required direct current (DC) shock delivery for termination.

RF catheter ablation was attempted at the origin of the PVB mapped to the outflow tract of the right ventricle (Fig. 3B). PVB and non-sustained VT disappeared soon after ablation. The atrial site of the slow pathway was mapped to the orifice of the coronary sinus, to which the RF delivery was able to ablate the retrograde conduction and AVNRT became non-inducible.

The post-ablation course was uneventful, and the frequency of PVB and non-sustained VT declined from 16.0%

to 0.26% of the total heart beats per day after ablation. VT became non-inducible by exercise, but the patient received an implantable cardioverter-defibrillator (ICD) for possible VF. He has currently experienced no arrhythmic events at the time of the writing of this article.

Discussion

The patient developed palpitation due to sustained VT which was inducible during exercise concomitantly with the induction of the typical Brugada ECG pattern. Pilsicainide administration provoked the typical Brugada ECG pattern. VT was not inducible by programmed electrical stimulation, but AVNRT was induced. PVB and non-sustained VT with the same morphology as VT increased in frequency with isoproterenol treatment. After the endocardial mapping, VT and AVNRT were ablated by RF catheter ablation. Because of induced VF during the electrophysiology study, the patient was treated with ICD. He is currently being followed at the outpatient clinic and has not had any arrhythmic events to date.

In BrS, polymorphic VT is more common than monomorphic VT and may lead to VF (1). Although infrequent, there are case reports of BrS combined with sustained monomorphic VT (3-17). Sustained monomorphic VT may be induced by exercise (8, 11, 14, 15, 17), by antiarrhythmic drugs alone (6, 16) or by antiarrhythmic drugs with electrical stimulation (4, 10, 16). In these cases, VT was always associated with provocation of the typical Brugada ECG pattern, and a close linkage was suggested between initiation of VT or PVB and the unmasking of the typical Brugada ECG pattern.

It is known that the typical Brugada ECG pattern may be induced in the post-exercise recovery phase, and it is considered due to the withdrawal of sympathetic activities and the enhancement of vagal activities. Changes of the autonomic nervous activities would augment the J-point elevation in BrS and may precipitate phase 2 reentry (18). The occurrence of PVB in association with the Brugada ECG pattern is common during drug testing for the provocation of BrS (19, 20). However, our patient was unusual in that he exhibited the typical Brugada ECG pattern and the induction of monomorphic VT during peak exercise rather than during the post-exercise recovery phase. In patients with mutations in the cardiac sodium channel, heart rate may play a more important role in developing the typical Brugada ECG pattern than increased parasympathetic activities (21, 22). However, due to lack of genetic screening, this could not be determined for our patient.

VT with reentrant mechanism may be rarely proved in BrS (17), but the mechanism of most sustained VT originating from the outflow tract of the right ventricle seems to be non-reentrant. Such non-reentrant VT is not typically induced due to programmed electrical stimulation but may appear spontaneously or aggravated by exercise, an increase in sympathetic nervous activity or administration of isoprotere-

nol. In the present case, VT was not inducible by electrical stimulation, but PVB and non-sustained VT with the same morphology as VT increased after isoproterenol treatment. After RF catheter ablation, PVB and non-sustained VT decreased and VT became non-inducible on exercise.

In addition to monomorphic VT, an association of supraventricular arrhythmias with BrS has been reported (23). These arrhythmias may share a common arrhythmogenic substrate with BrS, but further studies are necessary to establish a relationship.

Summary

In our patient, exercise unmasked the typical Brugada ECG pattern and induced VT with LBBB and inferior axis. Pilsicainide was diagnostic in provoking the typical Brugada ECG pattern. The patient also had AVNRT. VT and AVNRT were successfully ablated, and the patient was treated with ICD therapy for possible VF.

The authors state that they have no Conflict of Interest (COI).

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