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Case Report

Interesting electrophysiological findings in a patient with coexistence of atrial tachycardia originating from coronary sinus and slow-fast atrioventricular nodal reentrant tachycardia

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ABSTRACT

Slow-fast atrioventricular nodal tachycardia (AVNRT) has various electrophysiological aspects due to atrioventricular (AV) nodal physiology. In addition, concomitantly another form of arrhythmia with AVNRT, especially atrial tachycardia (AT), was an infrequent arrhythmia. A 38-year-old female with narrow QRS tachycardia underwent electrophysiological study due to frequent faintness. The electrophysiological study disclosed the coexistence of AT originating from coronary sinus (CS) with slow-fast AVNRT. We easily diagnosed AT originating from CS and terminated with several radiofrequency ablations (RFA) around CS. The diagnosis of slow-fast AVNRT, however, was somewhat difficult due to the following findings: (1) small amount of adenosine triphosphate (ATP) could terminate slow-fast AVNRT reproducibly; (2) we could provoke slow-fast AVNRT only by RV pacing with isoproterenol infusion. With other electrophysiological findings, we diagnosed slow-fast AVNRT. Radiofrequency energy was delivered initially in the posteroseptal region, followed by inside CS, and finally in the middle septal region, which completed the slow pathway ablation. After the procedure, we could never provoke these arrhythmias.

<**Learning objective:** Coexistence of focal AT originating from CS with slow-fast AVNRT is a rare phenomenon. Furthermore, slow-fast AVNRT could show unusual characteristic as following: (1) small amount of ATP terminates slow-fast AVNRT; (2) atrial pacing never provoked slow-fast AVNRT with isoproterenol infusion whereas ventricular pacing did, which depends on the physiological characteristic of the dual AV nodal pathway. Accordingly, we should precisely assess the obtained electrophysiological findings.>

Introduction

Coronary sinus (CS) is an uncommon site of origin for focal atrial tachycardia (AT), which was reported to be 6.7% of focal AT [1]. The success rate of radiofrequency ablation (RFA) was high at the point where the discrete potential was recorded [2]. On the contrary, slow-fast atrioventricular nodal tachycardia (AVNRT) is a well-known cause of paroxysmal supraventricular tachycardia (PSVT). However, in some cases, its diagnosis is difficult due to the electrophysiological characteristics. In our case, anterior slow pathway had high sensitivity to a small dose of adenosine triphosphate (ATP) and, moreover, we could not provoke slow-fast AVNRT by atrial pacing with or without isoproterenol infusion, which nearly misled us. Here, we describe the experience of coexistence of AT originating from CS with slow-fast AVNRT, besides their interesting electrophysiological findings.

Case report

A 38-year-old female was referred to our clinic with a complaint of repetitive faintness and palpitation. Ambulatory electrocardiogram (ECG) documented narrow QRS tachycardia with 1:1 atrioventricular (AV) conduction. There was no 12-lead ECG during the tachycardia. Echocardiography elucidated no

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structural heart disease. Electrophysiological study and subsequent catheter ablation were performed with the setting of multipolar electrode catheter in the high right atrium, His bundle, CS, and right ventricle (RV). Electroanatomical mapping was performed with 3-D electroanatomical mapping system (Ensite Velocity™, St. Jude Medical. Co., Ltd., St Paul, MN, USA) with a mapping catheter (Ensite Array™, St. Jude Medical. Co., Ltd.). RFA was performed with a 4-mm-tip temperature-controlled non-irrigated catheter. Initially, atrial overdrive pacing provoked a narrow QRS tachycardia (hereafter, SVT1). 12-Lead ECG showed long RP tachycardia with P-wave negative deflection in inferior leads (Fig. 1, upper panel). Tachycardia cycle length (TCL) was 420 ms with early activation site in CS ostium (Fig. 2A). RV overdrive pacing showed 1:1 ventriculoatrial (VA) conduction limited to a maximum of 100 bpm. The atrial activation sequence in electrode catheter during SVT1 was different from during RV pacing. Isopotential map showed a breakout site in the CS. Virtual unipolar electrogram (VUE) showed rS pattern, which represented that earlier atrial site was located inside CS. (Fig. 2C). In addition, return cycle after entrainment pacing from CS ostium was not identical with from His bundle area—VA linking was not observed (Fig. 3A and B). These findings were consistent with focal AT originating from the CS. SVT1 was sustained and accelerated with isoproterenol administration—TCL became 310 ms (Fig. 2B). Radiofrequency energy was delivered initially in CS ostium, targeting temperature of 55° and a power of 30 W. About 10 times of RFA around the CS was required to eliminate SVT1 where the discrete potential preceding the P-wave onset was recorded in distal ablation catheter (Fig. 2B), which implied the substrate for SVT1 expanded to inside CS (Fig. 2D).

Another narrow QRS tachycardia (SVT2) with TCL of 380 ms was induced and sustained by RV extra stimulation with isoproterenol infusion. 12-Lead ECG did not show the obvious P-wave (Fig. 1, lower panel). The electrophysiological study revealed the following findings: (1) atrial early activation site during tachycardia was the His bundle area, whose atrial activation sequence was identical with when RV burst pacing (Fig. 3C and D); (2) both AV and VA conduction showed decremental property and ventricular stimulation at refractory time of the His bundle could not affect the tachycardia cycle length; (3) atrial overdrive pacing from CS ostium could demonstrate manifest entrainment; (4) during entrainment study with atrial pacing, the VA interval of the return beat was the same as the VA interval of the tachycardia, which was known as VA linking; (5) the initiation of the tachycardia by RV extra stimulation showed V-A-V sequence pattern; (6) SVT2 could be provoked only by ventricular pacing—atrial program stimulation at basic cycle length of 400 ms or 600 ms could never provoke SVT2, besides the jump up in the atrio-His(AH) interval with or without isoproterenol infusion; (7) we could terminate SVT2 using small amount of ATP (2 mg) with AV

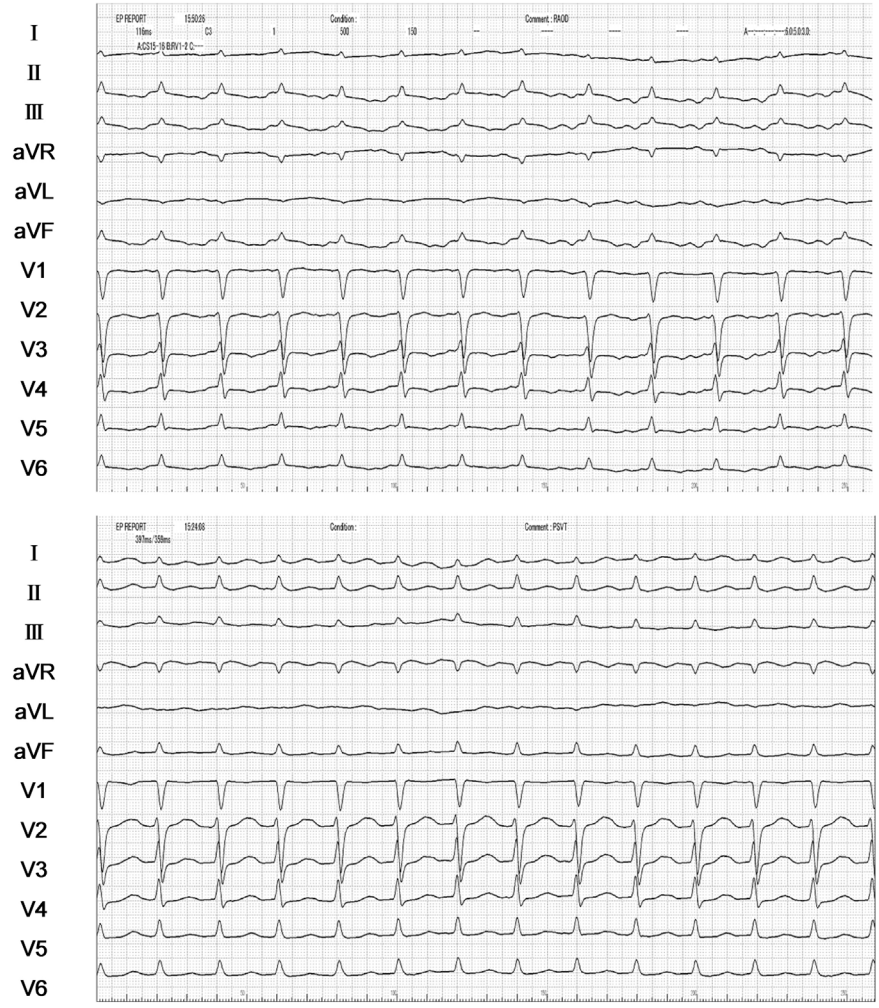

Fig. 1. Upper panel: 12-lead electrocardiogram (ECG) during SVT1 showed long RP tachycardia and P-wave deflection was negative in lead II, III, and aVF. Lower panel: 12-lead ECG during SVT2 showed narrow QRS tachycardia with the hidden P wave in QRS complex.

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