Coexistence of the Permanent Form of Junctional Reciprocating Tachycardia and Atrial Tachycardia

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This case report describes a patient with the permanent form of junctional reciprocating tachycardia coexisting with atrial tachycardia. A detailed electrophysiological study established the diagnosis, and radiofrequency catheter ablation abolished both arrhythmias. (Circ J 2005; 69: 1003 – 1006)

Key Words: Atrial tachycardia; Catheter ablation; Permanent form of junctional reciprocating tachycardia (PJRT); Supraventricular tachycardia

The permanent form of junctional reciprocating tachycardia (PJRT) is a rare supraventricular tachycardia in which retrograde ventriculoatrial (VA) conduction occurs with a decremental property. PJRT is frequently incessant and occasionally tachycardia-induced cardiomyopathy develops. The electrocardiographic feature of PJRT is a narrow QRS tachycardia with a long RP' interval. However, atypical forms of atrioventricular (AV) node reentry tachycardia (AVNRT) and atrial tachycardia (AT) show similar electrocardiographic findings. Therefore, a detailed electrophysiological study (EPS) must be done to establish the diagnosis. We describe a patient with coexisting PJRT and AT.

Case Report

A 31-year-old woman was referred to hospital for recurrent episodes of palpitation, the first attack of which had occurred 1 year ago and since then, the frequency had increased 2 or 3 times a day. She was treated with verapamil, but it did not completely suppress the attacks. A 12-lead electrocardiogram during sinus rhythm did not show a delta wave, but a narrow QRS tachycardia characterized by a negative P wave in leads II, III, and aVF with a long RP' interval was documented during a palpitation attack. An extensive work up, including echocardiographic study, did not reveal any structural heart disease.

She underwent an EPS after giving written informed consent. Two standard quadrupolar electrode catheters were positioned at the high lateral right atrium (HRA) and right ventricular apex (RVA). An octapolar electrode catheter was positioned at the His bundle and a 2.5Fr 16-electrode catheter was positioned in the coronary sinus ostium to eliminated it. Finally, there was residual VA conduction with the same atrial activation sequence as Tachycardia 1 or 2. A single ventricular extrastimulus (RV-St (RV)) resulted in increases in St (RV)-A2, indicating that the retrograde VA conduction during Tachycardia 1 had a decremental property. Based on these observations, Tachycardia 1 was diagnosed as PJRT and radiofrequency catheter ablation (RFCA) performed at the earliest atrial activation site near the coronary sinus ostium was able to terminate it. However, the other narrow QRS tachycardia with a long RP' interval (Tachycardia 2; Fig 1B) was induced by a single extrastimulus from RVA with isoproterenol infusion and the RVA–HRA interval was 208 ms. Tachycardia 2 was unstable, and subsequently changed to another narrow QRS tachycardia (Tachycardia 3; Fig 1C). The RVA–HRA interval during Tachycardia 3 was 108 ms and shorter than that of Tachycardia 1 or 2. A single ventricular extrastimulus could not affect the atrial activation sequence during Tachycardia 3 when the His bundle was refractory. Moreover, burst pacing from the RVA demonstrated AV dissociation without affecting the cycle length of Tachycardia 3 (Fig 4), suggesting that Tachycardia 3 was AT. Detailed endocardial mapping during Tachycardia 3 showed that the earliest site of atrial activation was in the posterolateral right atrium near the tricuspid annulus. A single burst of RF energy immediately changed Tachycardia 3 to Tachycardia 2, which then terminated spontaneously. However, there was residual VA conduction with the same atrial activation sequence as Tachycardia 2, so we performed RFCA at the earliest site via retrograde VA conduction in the midseptum of the right atrium (between the His bundle and coronary sinus ostium) to eliminated it. Finally, there was tachycardia was not induced by any pacing protocols. However, after isoproterenol infusion, a clinically documented narrow QRS tachycardia (Tachycardia 1) was induced by a single extrastimulus from the RVA (Fig 1A). Intracardiac tracing during Tachycardia 1 revealed that the earliest atrial activation was recorded near the coronary sinus ostium and the RVA–HRA interval was 200 ms. A single ventricular extrastimulus, which was introduced from the RVA when the His bundle was refractory, delayed the subsequent atrial activation (“postexcitation” phenomenon) (Fig 2). A retrograde VA conduction time (St (RV)-A2) curve was obtained after a single ventricular extrastimulus during Tachycardia 1 (Fig 3). Shortening of the coupling intervals of the extrastimulus (RV-St (RV)) resulted in increases in St (RV)-A2, indicating that the retrograde VA conduction during Tachycardia 1 had a decremental property.

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Fig 1. Twelve-lead electrocardiograms and intracardiac tracings during Tachycardia 1 (A), Tachycardia 2 (B), and Tachycardia 3 (C). Each narrow QRS tachycardia was characterized by a negative P wave in leads II, III, and aVf. Intracardiac tracings show that the RVA–HRA interval of Tachycardia 1 (200 ms) was similar as that of Tachycardia 2 (208 ms), whereas that of Tachycardia 3 (108 ms) was significantly different. CS 1–2, distal coronary sinus electrogram; CS 15–16, proximal coronary sinus electrogram; His–d, distal His bundle electrogram; HRA–d, distal high right atrium electrogram; HRA–p, proximal high right atrium electrogram; RVA, right ventricular apex electrogram.

Fig 2. Simultaneous recording of ECG and intracardiac bipolar electrograms during Tachycardia 1 show the “postexcitation” phenomenon. A single ventricular extrastimulus, which was introduced from the RVA when the His bundle was refractory, delayed the subsequent atrial activation (“postexcitation” phenomenon). CS 1–2, distal coronary sinus electrogram; CS 15–16, proximal coronary sinus electrogram; His–d, distal His bundle electrogram; His–p, proximal His bundle electrogram; HRA–d, distal high right atrium electrogram; HRA–p, proximal high right atrium electrogram; RVA, right ventricular apex electrogram.
no VA conduction after extrastimuli from the right ventricle, and none of the pacing protocols could induce the tachycardias, even with isoproterenol infusion. She has been clinically free from symptoms of palpitations during 2 years of follow-up.

Discussion

PJRT is usually characterized by incessant forms of supraventricular tachycardia, and occasionally the development of tachycardia-induced cardiomyopathy. However, some reports have identified a paroxysmal type of PJRT, such as in the present patient. The electrocardiographic feature of PJRT is a narrow QRS tachycardia with a long RP interval, but atypical forms of AVNRT and AT sometimes demonstrate similar electrocardiographic findings. In the present patient there were 3 types of narrow QRS tachycardia induced during the EPS. Tachycardia 1 was diagnosed as PJRT and Tachycardia 3 as AT, but Tachycardia 2 was not diagnosed precisely because of its spontaneous change to Tachycardia 3 or termination. However, the residual VA conduction by pacing from RVA showed a decremental property and the atrial activation sequence via the VA conduction was identical to that of Tachycardia 2.
Moreover, variable locations of the accessory pathway of PJRT, including the coronary sinus ostium and posterior mid-septum of the right atrium, have been reported. Therefore, it is more likely that Tachycardia 2 was PJRT or an atypical form of AVNRT than AT.

We performed RFCA targeting the earliest atrial activation site during the tachycardias or via the VA conduction, which resulted in successful ablation of both arrhythmias.

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