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Case Report

Wide QRS tachycardia associated with multiple accessory pathways in a patient with Wolff–Parkinson–White syndrome

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ABSTRACT

The electrocardiogram of a 14-year-old boy with recurrent palpitation showed a wide QRS regular tachycardia with a right bundle branch block and right-axis deviation of 226 beats per minute. Verapamil infusion terminated the tachycardia after a few minutes. Electrophysiological study revealed that this tachycardia was considered as a reentrant tachycardia associated with the anterograde left posterior accessory pathway (AP) and retrograde right septal AP. Radiofrequency application was performed and eliminated both APs, and there was no recurrence of wide QRS tachycardia.

<**Learning objective:** Wide QRS tachycardia in young patients with no organic heart disease includes an uncommon supraventricular tachycardia. Wide QRS tachycardia utilizing different dual accessory pathways (APs) has rarely been reported. The refractory periods of the APs were shorter than that of atrioventricular node, and the shortest refractory period on anterograde and retrograde conduction was recorded at the left posterior AP and the right septal AP respectively. These findings were to be felt most consistent with the mechanism of maintaining an atrioventricular reentrant tachycardia with multiple APs.>

Introduction

Atrioventricular reentrant tachycardia (AVRT) is a paroxysmal supraventricular tachycardia (SVT) with an electrically defined circuit that consists of two distinct pathways, the normal AV conduction system and an atrioventricular accessory pathway (AP). A paroxysmal SVT utilizing multiple APs was rarely reported, however, no detailed mechanism was shown. We hereby report a case of paroxysmal SVT associated with the left posterior and the right septal AP.

Case report

A 14-year-old boy with recurrent palpitation and pre-syncope during exercise was referred to the emergency room. The electrocardiogram (ECG) showed a wide QRS regular tachycardia with a right bundle branch block and right axis deviation of 226 beats per minute (Fig. 1A). Intravenous 20 mg adenosine administration could not terminate the tachycardia, while 5 mg verapamil infusion could prolong the RR interval and then terminate with the disappearance of the P' wave after a few minutes. The prolongation of the RR interval was mainly caused by the prolongation of the RP' interval but not P'R one. The ECG during sinus rhythm showed a delta wave: a negative delta wave in V1 and abrupt transition to R > S in V2, a negative delta wave in III and aVF but upright in II, which indicated the right septal AP (Fig. 1B) [1]. However, the morphology was completely different from that during the tachycardia. Although the left ventricular wall motion was slightly reduced, no organic heart disease could be detected by any imaging modalities.

Electrophysiological study was performed to assess the mechanism of the wide QRS tachycardia under conscious sedation. We performed the mapping along tricuspid annulus during a right ventricular apical (RVA) pacing of 90 ppm. The earliest atrial potential was recorded at the right septum near the His region. After an intravenous 20 mg adenosine administration, the atrial potential at the His region was delayed but earliest atrial potential at the right septum could not be changed. During high right atrium (HRA) burst pacing of 90–200 ppm, the earliest ventricular activation was recorded at the right septum where the earliest atrial activation could be recorded during RVA pacing (Fig. 1C).

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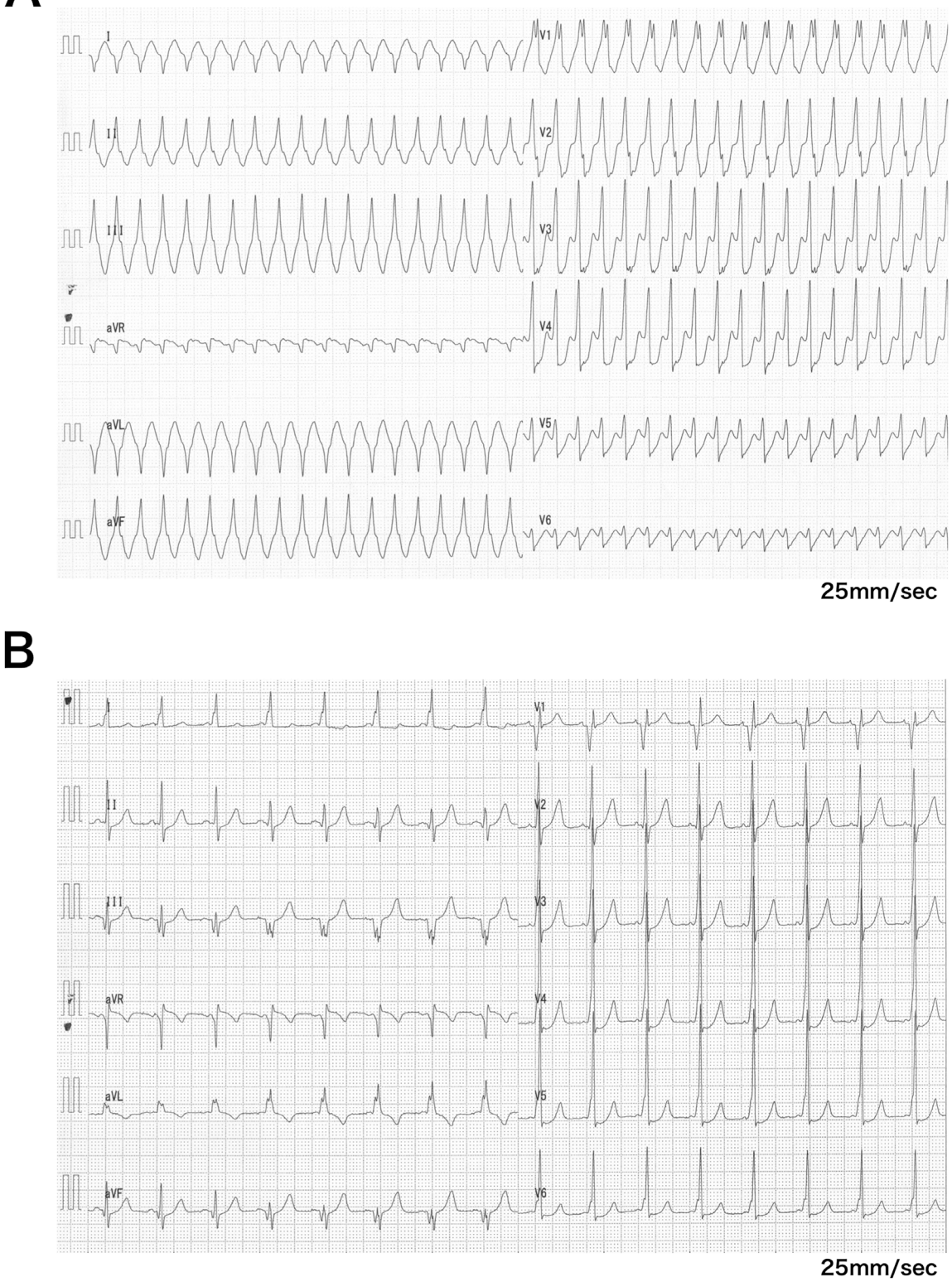

Fig. 1. (A) 12-lead ECG showed a wide QRS regular tachycardia of a RBBB with RAD at 226 beats per minute. (B) 12-lead ECG during sinus rhythm after verapamil infusion showed the delta wave. Note the negative delta wave in V1 and abrupt transition to R > S in V2. In addition, the delta waves were negative in III and aVF but upright in II, which indicated the right septal AP. (C) Intracardiac electrograms (the left panel) and 12-lead ECG (the right panel) during HRA burst pacing of 200 ppm. The dotted line indicated that the earliest ventricular activation recorded by the ablation catheter which was placed at the right septum. (D) Intracardiac electrograms (the left panel) and 12-lead ECG (the right panel) during HRA burst pacing of 210 ppm. The dotted line indicated that the earliest ventricular activation recorded at the left posterior wall. The solid line indicated the ventricular activation which activated from CS distal to CS proximal site. Of note, the QRS morphology was identical to that of the tachycardia.
ABL, ablation catheter; AP, accessory pathway; CS, coronary sinus; CSd, coronary sinus distal; ECG, electrocardiogram; HRA, high right atrium/atrial; RAD, right axis deviation; RBBB, right bundle branch block; RV, right ventricle/ventricular.

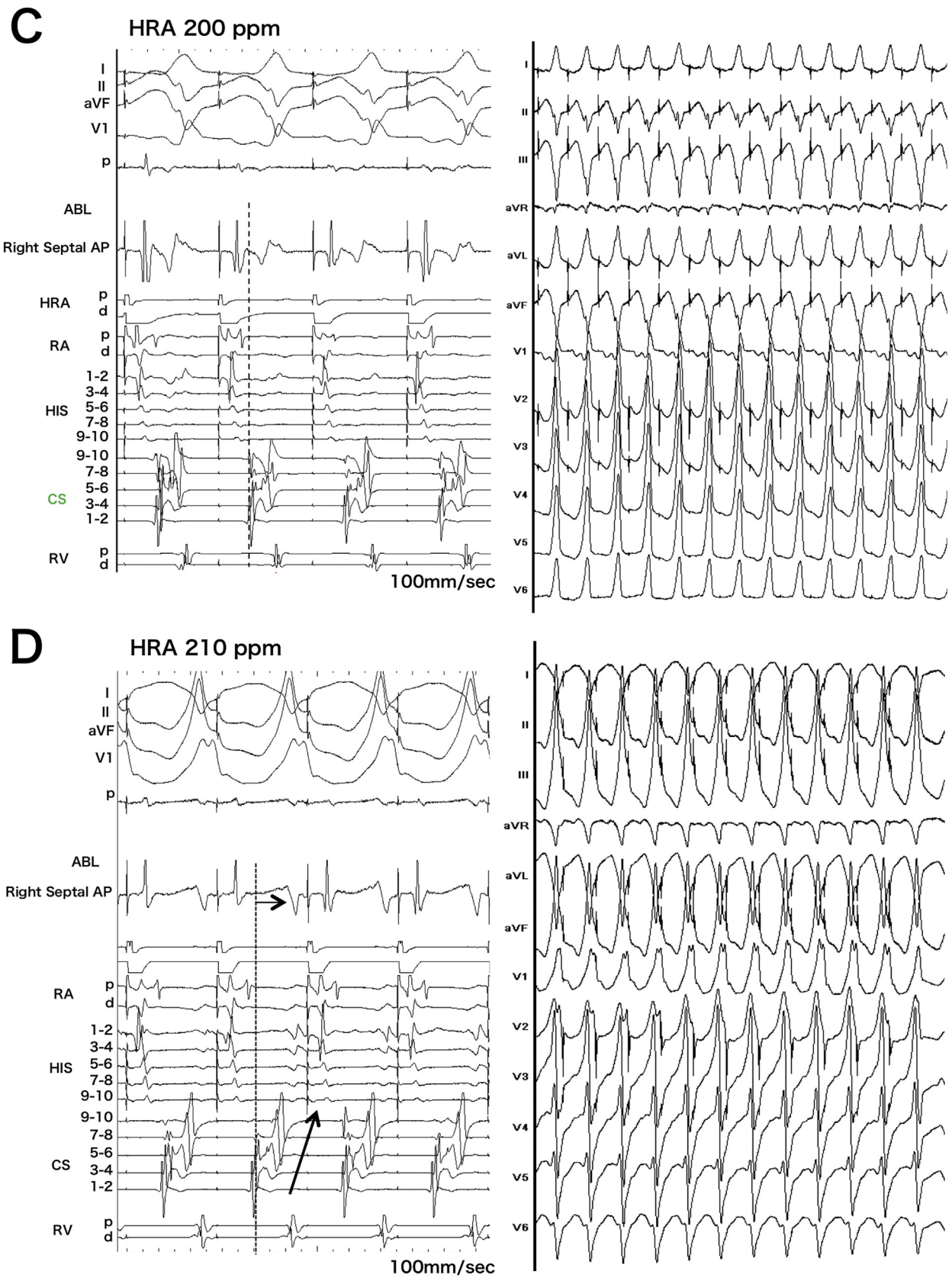

Fig. 1. *(Continued).*

Furthermore, the earliest ventricular activation abruptly changed to the left posterior wall by HRA burst pacing of 210 ppm. Of interest, the QRS morphology was identical to that during the tachycardia (Fig. 1D). The effective refractory period (ERP) of anterograde conduction including the left posterior AP, right septal AP, and fast pathway was 250 ms, 260 ms, and 330 ms, respectively, while that of retrograde conduction was 290 ms, 270 ms, and 350 ms, respectively. Tachycardia could be induced by a burst pacing from HRA under isoproterenol infusion. The earliest atrial potential was recorded by the ablation catheter which was located at the right septum close to the coronary sinus (CS), while the earliest ventricular potential was recorded at the posterior wall of the left ventricle. Electrograms recorded by conventionally placed catheters (CS, His, and RVA) could completely fulfill the tachycardia cycle length (TCL). Although entrainment pacing from the right ventricle was attempted, adequate entrainment could not be achieved due to the extremely high-rate tachycardia which was relatively hemodynamically deteriorated. The pacing from the right atrial septum where the earliest atrial potential was recorded could capture the right atrial signal demonstrating the concealed entrainment and the post-pacing interval-TCL was measured as only 16 ms, which indicated that the re-entrant circuit could include the right atrial septum (Fig. 2A and B). Furthermore, the VA interval after a cessation of the last entrainment pacing was identical to that during tachycardia, which indicated the VA linking. This indicated that the mechanism of the tachycardia was likely AVRT or atrioventricular nodal reentry tachycardia, but not AT. As for the retrograde conduction, the ERP of the retrograde conduction over the fast pathway was longer than the TCL. Therefore, retrograde fast pathway could be blocked during the tachycardia. Furthermore, the atrial activation sequence during the tachycardia was identical to that after an intravenous 20 mg adenosine administration during RVA pacing and program stimulation from RVA of 400/350 ms (retrograde fast pathway ERP). Therefore, the retrograde conduction was considered as the AP but not the fast pathway. Intravenous 20 mg adenosine administration could not terminate the tachycardia and no electrograms which were recorded by all diagnostic catheters

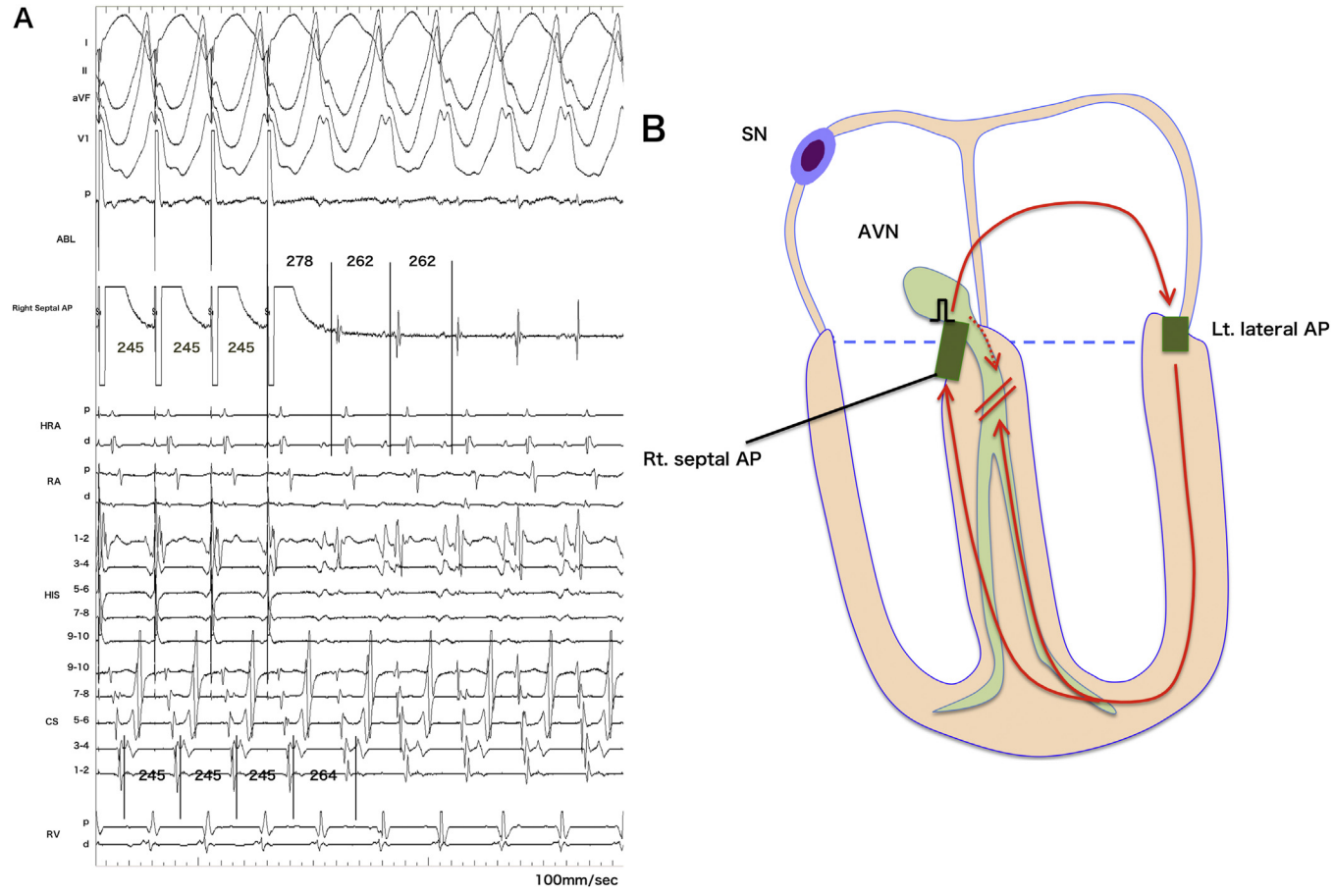

Fig. 2. (A) Entrainment from the right septal AP. The ablation catheter was placed at the right septal AP. This pacing could capture the right atrial signal demonstrating the concealed entrainment and the PPI-TCL was measured as only 16 ms, which indicated that the reentrant circuit could include the right atrial septum. (B) A schema demonstrating tachycardia circuit. The pacing from the right atrial septum where the right accessory pathway could connect to the right atrium could demonstrate the concealed entrainment. Only the right atrial signal around the His recording site near the pacing site could be captured antidromically, however, the His potential could not be activated due to the ERP of AVN. The arrow and dotted one indicate orthodromic and antidromic capture, respectively. (C) Intracardiac electrograms (the left panel), 12-lead ECG (the mid panel), and the fluoroscopic image (the right panel) during successful ablation for the left posterior AP. Success site was shown in the right panel. Immediately after RF application, the earliest ventricular activation abruptly changed from the left posterior wall to the right septum (the left panel). The QRS morphology was also changed (the mid panel). (D) Intracardiac electrograms (the left panel), 12-lead ECG (the mid panel), and the fluoroscopic image (the right panel) during successful ablation for the right septal AP. Success site is shown in the right panel. The AP could be eliminated immediately after RF application (the left panel) and the delta wave disappeared (the mid panel).
ABL, ablation catheter; AP, accessory pathway; CS, coronary sinus; LAO, left anterior oblique; PPI, post pacing interval; RA, right atrium; RAO, right anterior oblique; RF, radiofrequency; RV, right ventricle/ventricular; TCL, tachycardia cycle length.

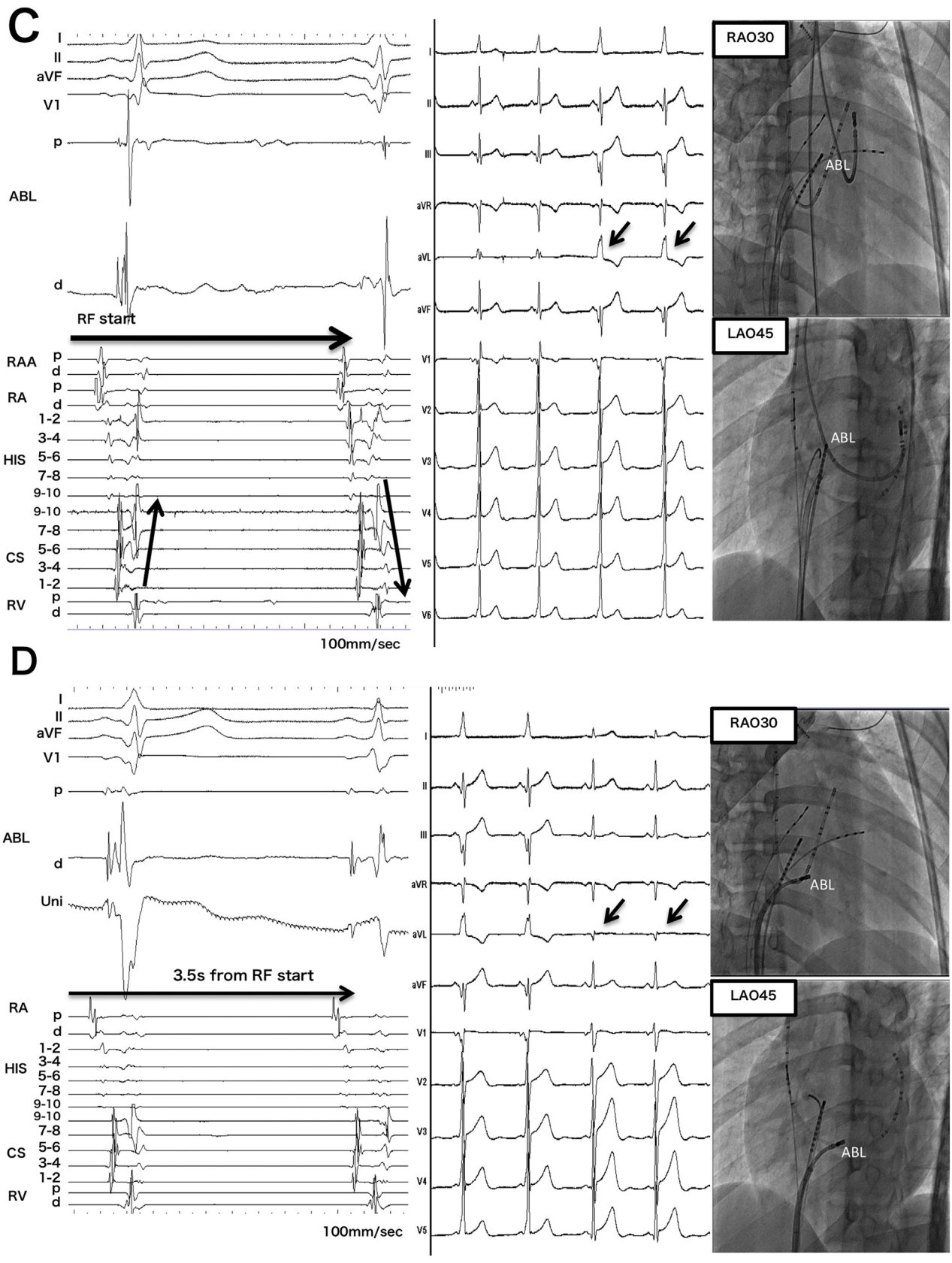

Fig. 2. (*Continued*).

could be changed. This tachycardia was considered as a reentrant tachycardia associated with the anterograde left posterior AP and the retrograde right septal AP. Radiofrequency (RF) current deliveries were applied with a 3.5-mm-irrigated tip ablation catheter (ThermoCool SmartTouch, Biosense Webster, Diamond Bar, CA, USA), with a maximum power and temperature of 30 W and 43 °C. Immediately after RF application, the earliest ventricular activation abruptly changed from the left posterior wall to the right septum. The QRS morphology was also changed (Fig. 2C). The right accessory pathway was successfully ablated immediately after RF application at anterior side of the ostium of CS where the accessory pathway potential was recorded (Fig. 2D). After the elimination of both APs, electrophysiological study could demonstrate anterograde dual pathway and the retrograde fast pathway, which was blocked by the adenosine administration. There has been no recurrence of a wide QRS tachycardia as of the last available follow-up (12 months).

Discussion

AVRT associated with both anterograde and retrograde AP is rare. Ceresnak et al. reported that approximately 10% of the patients with Wolff–Parkinson–White syndrome had multiple APs, in whom only one case with tachycardia associated with anterograde left posterior AP and retrograde right septal AP was reported [2]. The electrophysiological study revealed two distinct APs: the right septal AP and left posterior AP. The QRS morphology of the left posterior AP was identical to that of the tachycardia. Therefore, the possible reentry circuit of the tachycardia was as follows: (1) verapamil sensitive idiopathic VT; (2) AT with a left posterior AP; (3) antidromic AVRT with a left posterior AP [3]. Continuous fractionated potential between ventricular and atrial electrograms could be recorded at the right septal AP where the entrainment with the local atrial capture could clearly demonstrate the concealed one. This strongly suggested that the tachycardia circuit included the right atrium as well as ventricle. Thus, both VT and AT could be completely ruled out. Furthermore, anterograde ERP of the left posterior AP and retrograde ERP of the right septal AP were shorter than any other pathways which would play an important role in perpetuating the tachycardia.

Intravenous verapamil usually has no significant effects on electrophysiological properties of the AP. There were two possible considerations of the anatomic substrate of an AP that exhibited properties similar to those of the AV node: (1) heterogeneous physiologic properties of individual myocardial fibers or longitudinal dissociation of the myocardial fibers of the AP, and (2) the AP containing cells with histologic characteristics of AV nodal cells [4]. Additionally, Tai et al. reported that an AP with AV node-like properties in which AP could be blocked by intravenous verapamil. The possible mechanism was as follows: (1) direct prolongation of conduction velocity and refractoriness of an AP; (2) indirect prolongation of conduction velocity and refractoriness of an AP due to the alternating long and short cycle lengths by verapamil, which lead to development of the Ashman phenomenon in the AP. In the current case, the tachycardia was terminated with gradual prolongation of R–R interval. Therefore, the possible mechanism of termination by verapamil was speculated to be the right septal AP with AV node-like property rather than the Ashman phenomenon. AV node property was characterized as the shallow resting potential due to the less IK1 channel. Therefore, Ca channel blocker such as verapamil was effective to suppress the conduction. Adenosine increased the inward rectifier potassium current IKado and induced the hyperpolarization, which suppressed the depolarization and induced the conduction block [5]. We speculated that this accessary pathway cell might include both less IK1 and IKado channel. Therefore, conduction of accessory pathway could be eliminated by verapamil but not adenosine.

We experienced wide QRS tachycardia associated with multiple APs in a patient with Wolff–Parkinson–White syndrome. A careful electrophysiological study could reveal the two distinct accessary pathways as tachycardia circuit and the precise RF application could eliminate all arrhythmogenic accessory pathways.

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Conflict of interest

Authors declare no conflict of interests for this article.

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