A wide QRS complex tachycardia utilizing an atypical accessory pathway in latent Wolff-Parkinson-White syndrome: Manifestation of anterograde conduction during atrial fibrillation without delta waves in sinus rhythm

Toshihiro Nakamura, MD,* Koji Fukuzawa, MD,* Jun Kurose, MD,*
Hideya Suehiro, MD,* Kensuke Matsumoto, MD,† Ken-ichi Hirata, MD, PhD*

*From the *Section of Arrhythmia, Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, Hyogo, Japan, and †Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, Hyogo, Japan.*

Introduction

Atrial fibrillation (AF) may be fatal in patients that have accessory pathways (APs) with fast anterograde conduction. In some individuals, however, conduction across the AP may not be observed during sinus rhythm despite the presence of a pathway capable of rapid anterograde conduction. This unique and interesting pathway was previously reported as a latent AP.[1]

Here, we would like to describe a case in which preexcitation was not visible on the resting electrocardiogram (ECG) and anterograde AP conduction was manifested only during the tachycardia or rapid atrial rates and was diagnosed as at high risk for the development of ventricular fibrillation (VF).

KEY TEACHING POINTS

- Occasionally, conduction across the accessory pathway (AP) may not be observed during sinus rhythm despite the presence of a pathway capable of rapid anterograde conduction, which is called "latent preexcitation."
- The AP in this case exhibited atypical electrophysiological characteristics as compared to conventional APs, such as the atrioventricular accessory pathway or atriofascicular pathway.
- We should be aware of latent Wolff-Parkinson-White syndrome, which has very unique electrophysiological characteristics and may be high risk for developing ventricular fibrillation.

Case report

A 60-year-old woman with palpitations and presyncope was taken to the emergency room. Her blood pressure and pulse rate were 100/83 mm Hg and 174 beats per minute (bpm), respectively. Her consciousness level was awake and alert. The 12-lead ECG exhibited a wide QRS irregular tachycardia (Figure 1A), which was diagnosed as ventricular tachycardia. Then, direct cardioversion was performed in order to restore sinus rhythm. The ECG during sinus rhythm did not exhibit any delta waves (Figure 1B). No structural heart disease could be detected by any imaging modality, including cardiac ultrasound or computed tomography images. No renal failure or electrolyte imbalance could be detected from her laboratory tests. She was transferred to our hospital for further detailed analysis and evaluation.

Although she has had no major illnesses in the past, she was admitted to another hospital 4 years prior to this presentation owing to chest discomfort while working on a farm. According to her memory, she was treated with an automated external defibrillator but she had not fainted. Since the medical records were not present, the details were unclear.

An electrophysiological study (EPS) was performed in a nonsedated state. Two quadripolar electrode catheters were positioned in the right atrial appendage (4F Inquiry, Irvine Biomedical, St. Jude Medical, Irvine, CA) and right ventricle

KEYWORDS Atrial fibrillation; Catheter ablation; Delta wave; Latent preexcitation; Wolff-Parkinson-White syndrome
(Heart Rhythm Case Reports 2019;5:419–423)

The Section of Arrhythmia is supported by an endowment from Medtronic JAPAN and Abbott JAPAN. Dr Hirata chairs the Section, and Dr Fukuzawa belongs to the Section. All authors declare no conflicts of interest for this article. **Address reprint requests and correspondence:** Dr Koji Fukuzawa, Section of Arrhythmia, Division of Cardiovascular Medicine, Department of Internal Medicine, Kobe University Graduate School of Medicine, 650-0017, Kusunoki-Cho 7-5-2, Chuoh-Ku, Kobe, Japan. E-mail address: kfuku@med.kobe-u.ac.jp.

https://doi.org/10.1016/j.hrcr.2019.05.001

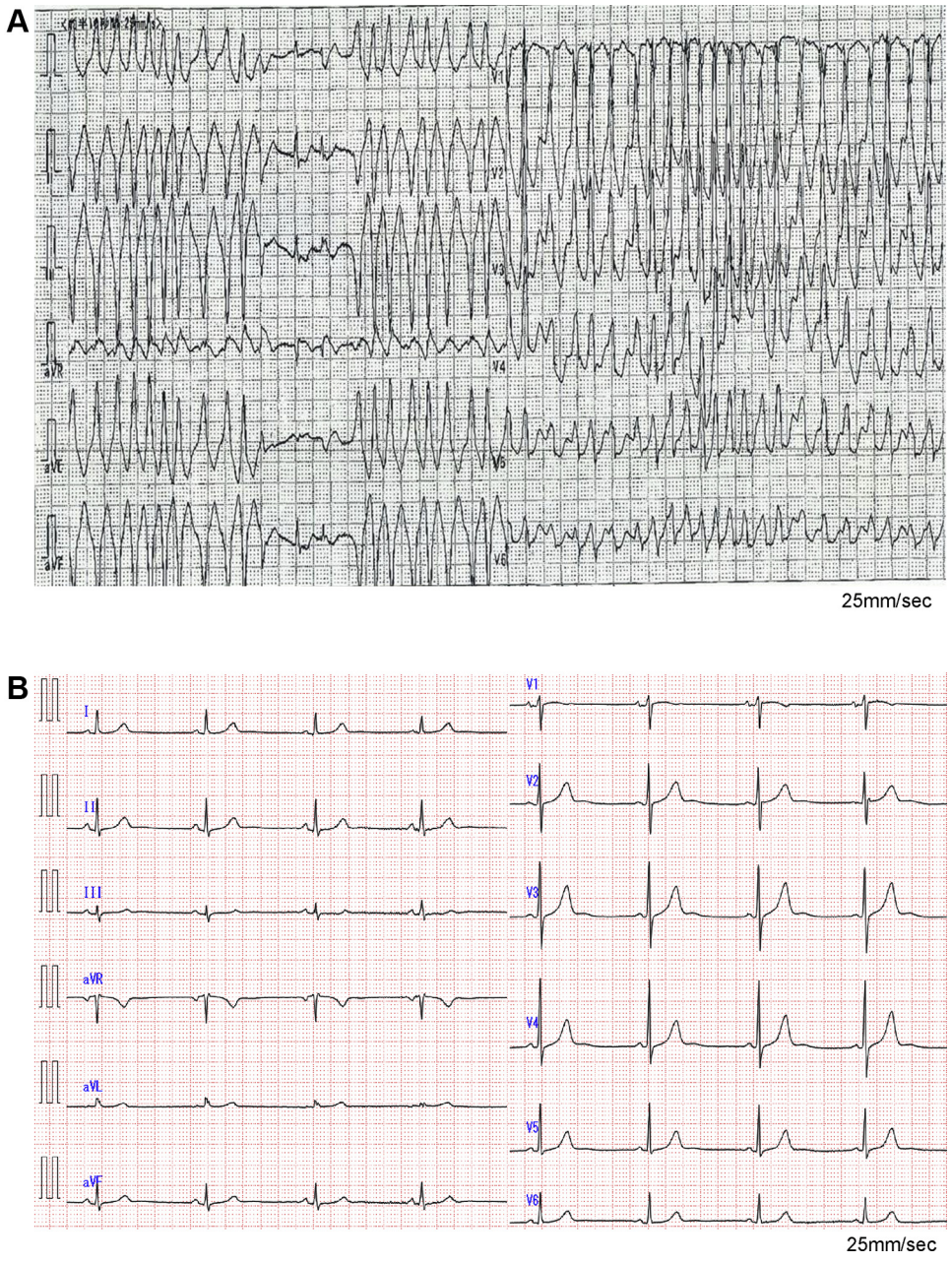

Figure 1 **A:** A 12-lead electrocardiogram (ECG) exhibiting a wide QRS irregular tachycardia suspected to be atrial fibrillation with a rapid ventricular response. **B:** The resting 12-lead ECG during sinus rhythm exhibited no delta waves. The heart rate was 49 beats per minute and PR interval 149 ms.

(4F Woven; Bard Electrophysiology, Lowell, MA). An octapolar catheter (5F Inquiry, IBI, Irvine Biomedical, Irvine, CA) was placed in the His-bundle region. A steerable decapolar electrode catheter was introduced into the coronary sinus and also simultaneously recorded the activity from the high right atrium (6F BeeAT, Japan Lifeline Co, Ltd, Tokyo, Japan). All bipolar electrograms were bandpass filtered between 30 and 500 Hz. At baseline, the heart rate was 62 bpm, and the atrial-His and His-ventricular intervals were 89 ms and 41 ms, respectively. During right ventricular apical (RVA) pacing, the earliest atrial activation was recorded at the ostium of the coronary sinus (CSo) without any decremental conduction properties, which was considered to represent retrograde conduction via an AP. During RVA pacing at a rate of 130 paces per minute (ppm), a V-A-V sequence pattern was observed and a narrow complex tachycardia with 2 different cycle lengths alternating between 420 and 540 ms was induced. The His-ventricular interval of the tachycardia was constant at 40 ms and the earliest atrial activation was recorded at the CSo, and the atrial activation sequence during the tachycardia was identical to that of the retrograde AP conduction during RVA pacing. The reason for the short and long RR cycle lengths was an alternation in the atrial-His intervals, which was 190 ms for the shorter cycles and 310 ms for the longer cycles. Therefore, this tachycardia was considered to be an orthodromic

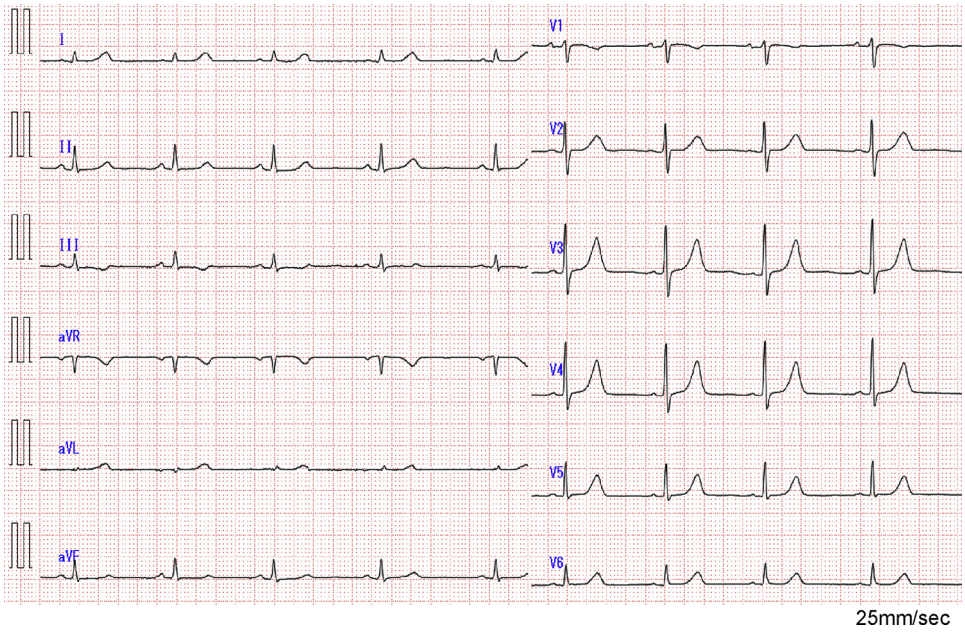

Figure 2 Resting 12-lead electrocardiogram post ablation procedure. The heart rate was 55 beats per minute and PR interval 173 ms. No septal Q waves were recognized.

atrioventricular (AV) reentrant tachycardia associated with anterograde alternating conduction between the fast and slow pathways with retrograde AP conduction. The results of para-Hisian pacing at 150 ppm indicated that the retrograde conduction passed through the AP. Retrograde conduction without any decremental conduction was observed with extrastimuli delivered from the RVA at 500/450–320 ms. With extrastimuli delivered at 500/320–220 ms, the atrial activation site remained the same (CSo), but the atrial activation sequence changed and the retrograde conduction exhibited a decremental property. Therefore, the retrograde AP effective refractory period (ERP) was 500/320 ms and the retrograde AV node ERP was 500/220 ms.

Incremental atrial pacing at a rate of 90 to 130 ppm demonstrated narrow complexes without any preexcitation. However, with a right atrial appendage pacing rate of over 150 ppm the His potential became hidden and preexcitation became obvious on the 12-lead ECG. This anterograde AP conduction was present during atrial pacing at a rate of up to 250 ppm under controlled conditions without an isoproterenol (ISP) infusion. The stimulus-to-delta interval was 196 ms and it was not changed by the pacing rate. It conducted in a 1-to-1 manner up to 310 ppm under an ISP infusion. With programmed atria1 stimuli delivered at a basic cycle length of 500 ms, the anterograde ERP of the AV node was 500/400 ms and that of the AP was 500/220 ms. The administration of 20 mg of intravenous adenosine induced AV nodal block and the manifestation of anterograde AP conduction.

AP mapping was performed with a 7.5F deflectable catheter (TactiCath, Abbott, St. Paul, MN) guided by a 3-dimensional mapping system (EnSite NavX system, St. Jude Medical, St. Paul, MN). Mapping was performed during CSo pacing at 150 ppm in order to unmask the anterograde AP conduction. During the mapping along the tricuspid annulus, a tiny potential was recorded, which was thought to be an AP potential. A radiofrequency current application at that site at 35 watts (temperature limit 40°C, flow rate of 30 mL/minute) transiently eliminated the delta wave. Further detailed mapping was performed, and permanent AP block was achieved at an inferoseptal site of the tricuspid annulus.

Although the 12-lead ECG showed a slight PR interval prolongation, no apparent change was observed as compared to that before the ablation. Importantly, there were no septal Q waves (Figure 2) on the 12-lead ECG before or after the ablation. The patient has been free from any tachycardia recurrence during a follow-up of 6 months.

Discussion

This patient with latent preexcitation was in a high-risk group of Wolff-Parkinson-White (WPW) syndrome, which can lead to VF. Generally, the preexcitation syndromes are classified into 3 types depending on the characteristics of the anterograde conduction: manifest, concealed, or intermittent types of WPW. However, there is another classification, which is called "latent preexcitation." Latent preexcitation is defined ventricular preexcitation that is absent during sinus rhythm but evident during atrial pacing, atrial extrastimuli, or atrial arrhythmias.[1,2] AF with extremely fast anterograde AP conduction may result in VF and be life-threatening. In a previous report, the measurement of shortest preexcited R-R intervals has been used to determine the AP properties. A shortest preexcited R-R interval of 220 to 250 ms and especially that less than 220 ms is more commonly seen in patients with WPW syndrome who

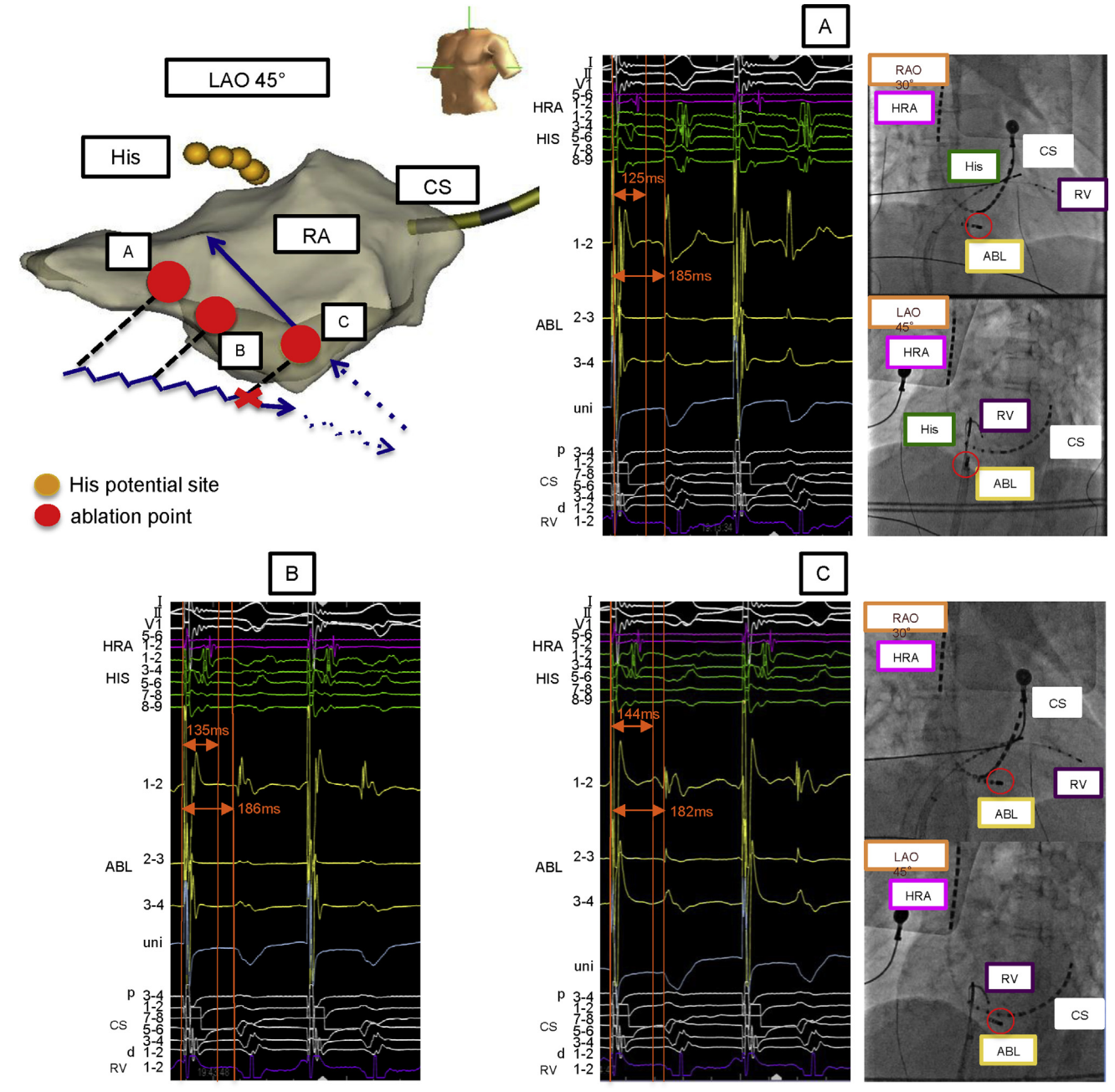

Figure 3 Accessory pathway (AP) mapping was performed during coronary sinus ostium (CSo) pacing at 150 ppm based on the prematurity of the AP potential and ventricular activation potential (V). The location markers recorded by the EnSite NavX system (St. Jude Medical, St. Paul, MN) are shown on the electroanatomical map. Site A is the site where transient block was obtained, and site C is the site of the successful ablation. The stimulus-AP intervals at sites A, B, and C were 125 ms, 135 ms, and 144 ms, respectively. The shortest AP-V interval was recorded at the successful site C. On the electroanatomical map, the *wavy arrow* indicates the slow conduction over the AP. The *straight arrow* indicates the relatively fast conduction over the normal myocardium. The anatomical distance between the AP fiber and each ablation site (*dotted straight lines*) gradually became closer as the ablation catheter came closer to the successful ablation site C. The *solid line* indicates the pathway proven during the electrophysiological study and the *dotted line* indicates the speculated pathway. The catheter position is shown in the fluoroscopic image and each *red circle* indicates the ablation site (sites A and C). ABL = ablation catheter; CS = coronary sinus; CSo = coronary sinus ostium; His = His potential site; HRA = high right atrium; LAO = left anterior oblique; RA = right atrium; RAO = right anterior oblique; RV = right ventricle.

have experienced cardiac arrest.[3–5] In our case, the shortest R-R interval of the clinical tachycardia was obviously less than 220 ms, which indicated that this AP was considered to be at very high risk for sudden death. Robinson and colleagues[1] reported 3 cases of latent preexcitation whose anterograde conduction across a previously undocumented AP was exposed by the development of AF. In 2 of those 3 patients, the shortest R-R interval during AF was less than 250 ms, which indicated that they were regarded as being at high risk for rapid conduction across the AP. However, a recent paper reported that in children with WPW syndrome, a life-threatening event can be the first symptom, and risk stratification using clinical and

EPS-derived data are imperfect and fail to identify all those at risk.[6]

Bogun and colleagues[7] concluded that the presence of septal Q waves on the surface ECG in lead V6 excludes manifest preexcitation. In this case, we could not find any septal Q waves, despite carefully reviewing the preoperative and postoperative resting ECG.

The AP in this case exhibited atypical electrophysiological characteristics as compared to conventional APs, such as AV APs or atriofascicular pathways. The characteristics of the AV AP and atriofascicular pathway were as follows:
AV AP

- with delta waves during anterograde conduction
- without decremental conduction properties
- no effect during an adenosine administration

Atriofascicular pathway

- mainly located on the right atrial free wall
- relatively long conduction time
- with decremental conduction properties
- transient block during an adenosine administration
- with anterograde-only conduction

Moreover, nodal-ventricular and nodal-fascicular fibers also have decremental conduction properties. Therefore, this patient's AP was not classified as any other typical AP.

In principle, if the time taken for the propagation of a sinus impulse to and across the AP is greater than the time taken for that impulse to propagate to the ventricles via the AV node, preexcitation may not be seen. In a previous report, it was observed that latent APs typically were located quite laterally in the heart, making conduction through the AV node more likely, given the shorter distance the impulse had to travel to the AV node rather than the longer distance to the AP.[1] In the present case, however, we could successfully eliminate the AP from the right atrium.

To clarify the localization of this AP, we carefully reviewed the electrophysiological findings during the mapping of the tricuspid annulus. Despite the stimulus-V interval being almost the same anywhere the AP potential was recorded, the stimulus–AP potential interval was longest at the successful site (Figure 3). This finding indicated that the conduction velocity over the AP was very slow and that in the normal ventricular myocardium was relatively fast. Moreover, we presumed that the anatomical distance between the AP fiber and each ablation site became gradually closer as the ablation catheter came closer to the success site, which could explain why the permanent AP block was completed at the successful site. Nevertheless, the AP potential–V interval was still 40 ms even at the success site. Therefore, we could speculate that the ventricular insertion site of the AP was seen as an extension of the successful site. Unfortunately, a detailed evaluation of the atrial insertion site of the AP was not performed.

Despite the slowly conducting AP, an Ebstein anomaly was not detected by any other imaging modality such as echocardiography or computed tomography. Moreover, the preexcitation was not observed during sinus rhythm even though ISP was given during the EPS prior to the ablation, and the AP conduction was not blocked by an administration of adenosine. Therefore, this AP was not identical to the characteristics of the slowly conducting AP as previously reported.

Conclusion

We experienced a case of a wide QRS complex tachycardia in a patient with latent preexcitation syndrome. We must pay attention to this unusual AP capable of anterograde conduction despite the absence of delta waves during sinus rhythm.

Acknowledgments

The authors thank Mr John Martin for his linguistic assistance.

References

1. Robinson K, Rowland E, Krikler DM. Latent pre-excitation: exposure of anterograde accessory pathway conduction during atrial fibrillation. Br Heart J 1988; 59:53–55.
2. Garratt C, Linker N, Griffith M, Ward D, Camm AJ. Comparison of adenosine and verapamil for termination of paroxysmal junctional tachycardia. Am J Cardiol 1989;64:1310–1316.
3. Sharma AD, Yee R, Guiraudon G, Klein GJ. Sensitivity and specificity of invasive and noninvasive testing for risk of sudden death in Wolff-Parkinson-White syndrome. J Am Coll Cardiol 1987;10:373–381.
4. Santinelli V, Radinovic A, Manguso F, et al. The natural history of asymptomatic ventricular pre-excitation a long-term prospective follow-up study of 184 asymptomatic children. J Am Coll Cardiol 2009; 53:275–280.
5. Bromberg BI, Lindsay BD, Cain ME, Cox JL. Impact of clinical history and electrophysiologic characterization of accessory pathways on management strategies to reduce sudden death among children with Wolff-Parkinson-White syndrome. J Am Coll Cardiol 1996;27:690–695.
6. Etheridge SP, Escudero CA, Blaufox AD, et al. Life-threatening event risk in children with Wolff-Parkinson-White syndrome: a multicenter international study. JACC Clin Electrophysiol 2018;4:433–444.
7. Bogun F, Kalusche D, Li YG, Auth-Eisernitz S, Gronefeld G, Hohnloser SH. Septal Q waves in surface electrocardiographic lead V6 exclude minimal ventricular preexcitation. Am J Cardiol 1999;84:101–104.