Occurrence of Potentially Lethal Arrhythmia due to Sudden Exposure of an Overt Accessory Pathway 8 Years After Catheter Ablation of a Concealed Accessory Pathway

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Summary
Although the efficacy of catheter ablation of the accessory pathway (AP) has been established, there are subgroups of APs with an intermittent conduction property, which is sometimes difficult to diagnose accurately. A 57-year-old man with a history of catheter ablation was referred to our clinic due to frequent faintness. He had undergone concealed AP ablation 8 years previously and bilateral circumferential pulmonary vein isolation (CPVI) 6 years previously. During regular electrocardiogram monitoring, it was suggested that irregular wide QRS tachycardia, which was considered to be atrial fibrillation with antegrade AP conduction, was the cause of the present symptoms. In the present electrophysiological study, we noticed a residual antegrade AP in the left lateral wall that was not observed during the previous session. We achieved abolition of overt accessory conduction, bilateral CPVI, and superior vena cava isolation with several radiofrequency applications without any recurrence. We also confirmed the absence of dormant conduction in the AP and the left atrium-PV connection with 20 mg adenosine triphosphate. This case demonstrated the possibility of sudden exposure of overt AP conduction late after catheter ablation of the concealed AP and the importance of confirming the absence of dormant conduction by means of adenosine triphosphate, which has the potential benefit of revealing latent AP conduction.

Key words: Intermittent, Atrial fibrillation, Circumferential pulmonary vein isolation, Electroanatomical mapping

A 57-year-old man who experienced frequent faintness and presyncope during exertion and had a recent episode of syncope was referred to our clinic. The frequency of these symptoms increased over several months. A 12-lead electrocardiogram (ECG) demonstrated AF, complete right bundle branch block, and normal axis deviation. An echocardiogram revealed no structural heart disease except for mild dilatation of the left atrium (LA). He had a history of two catheter ablations performed at another institute. The first session was performed 8 years previously because of frequent palpitations. Initially, ventricular pre-excitation was not observed during sinus rhythm. Right ventricular (RV) pacing demonstrated the earliest atrial activation site in the left lateral wall without decremental properties with an upper rate of 1:1 conduction of 200 bpm. Atrial extra stimulation with a basic cycle length of 600 ms revealed decremental properties and an atrioventricular (AV) nodal effective refractory period (AVNERP) < 250 ms; that is, the atrial effective refractory period was 250 ms. The jump-up phenomenon, gap phenomenon, and supernormal conduction were observed. Adenosine

Case Report
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Figure 1.  A: Elimination of AP conduction was achieved by RFA at the mitral annulus in the four o’clock position (white arrowhead). B: A 12-lead ECG revealed different QRS morphology when atrial ectopy was occurring (asterisks). HRA indicates high right atrium; LLRA, low lateral right atrium; HIS, his bundle; RV, right ventricle; CS, coronary sinus; Abl, ablation catheter; and LAO, left anterior oblique.

Triphosphate and isoproterenol were not administrated during the electrophysiological study. Atrial extra stimuli reproducibly induced tachycardia with an atrial activation sequence equivalent to that during RV pacing. Furthermore, the single RV stimuli during HIS bundle excitation reset the tachycardia. On the basis of these findings, the patient’s tachycardia was diagnosed as orthodromic re-entrant tachycardia via retrograde conduction in a lateral AP. An ablation catheter was delivered to the left ventricle via the aortic root. The retrograde AP was successfully blocked 3 seconds after RFA with a temperature of 55°C and a maximum power of 50 W using a nonirrigated-tipped catheter at the mitral annulus in the four o’clock position, followed by 120 seconds of RFA (Figure 1A), without any recurrence. Approximately 1 year after the procedure, a 12-lead ECG incidentally documented premature atrial ectopy and subsequent QRS complex that had morphology different from that during sinus rhythm. However, this finding was not recognized by the physician then (Figure 1B). The second session, which consisted of circumferential pulmonary vein isolation (CPVI) and cavo-tricuspid isthmus linear ablation, targeting drug-refractory paroxysmal AF and common atrial flutter, was successfully completed 2 years after the first session. During this session, surgeons only confirmed the absence of retrograde left lateral AP, but they intended to reveal latent AP conduction because the existence of the second QRS morphology had not been recognized then. After the procedure, the patient sometimes experienced palpitations but did not have faintness or syncope for approximately 6 years.

We planned a repeat CPVI targeting drug-refractory paroxysmal AF. Regular monitoring of ECG documented some transient short-running premature atrial contractions followed by irregular wide QRS complexes (Figure 2A) during which the patient reported discomfort, which allowed us to understand the cause of the symptoms. During the electrophysiology study (EPS), sinus rhythm was maintained at the beginning of the procedure. Multielectrode catheters were introduced into the coronary sinus, high right atrium, and RV. Two multielectrode ring catheters were introduced into the LA with a trans-septal puncture. Electroanatomical mapping and subsequent ablation were performed using a three-dimensional electroanatomical mapping system (Carto3®; Biosense Webster, Diamond Bar, CA, USA) with a 3.5-mm open irrigated-tip catheter (Thermocool SmartTouch® SF; Biosense Webster). At first, we conducted RV pacing, which could not reveal VA conduction via the left lateral side but could reveal conduction via the septal side with a decremental property, implying the presence of retrograde AV nodal conduction. Notably, we noticed AV conduction without the decremental property via the left lateral side where the retrograde AP had existed previously, suggesting the presence of antegrade AP. The EPS revealed an effective refractory period (ERP) of AP (APERP) of 270 ms and 1:1 conduction...
during atrial pacing up to 220 bpm. Extra stimuli shorter than 270 ms could not reveal other AV conduction. We performed RFA with a power of 30 W and an upper limit temperature of 42°C at the mitral annulus at the four o’clock position (Figure 2B), where the local ventricular potential preceding the QRS onset was recorded during sinus rhythm, achieving conduction block within 1 second (Figure 3). This was followed by an additional application of 90 seconds. Simultaneous 12-lead ECG demonstrated the disappearance of delta waves in lead III and morphological changes within the QRS complex in precordial leads just after RFA (Figure 2C). Atrial overdrive pacing revealed AV conduction via the septal side with a decremental property. Subsequently, we completed bilateral CPVI and isolation of the superior vena cava (SVC) due to the LA-PV reconnection in all PVs and the presence of SVC potential. After 30 minutes of waiting, we confirmed the absence of both LA-PV dormant conduction and AP conduction by an intravenous ATP injection of 20 mg. Overt AP conduction and atrial fibrillation did not recur during the last 6 months of follow-up.

**Discussion**

In the present case, we demonstrated the sudden exposure of overt AP conduction late after catheter ablation for concealed AP, which had been originally recognized as unidirectional retrograde conduction.

To explain this rare experience, we present the following significant topics: (1) the mechanism of concealed antegrade AP, (2) the anatomical relationship of previous and present AP, and (3) the role of ATP use. First, manifestation of antegrade AP was strongly associated with AV nodal conduction. We speculated that antegrade AP was ignored during the first session due to the fast AV nodal conduction. During the latest session, APERP was demonstrated as 270 ms, and AVNERP was estimated to be longer than 270 ms, which implied impaired intra-atrial or...
AVN conduction. Moreover, atrial ectopy incidentally elucidated the altered QRS morphology 1 year after the first session, suggesting the presence of an intermittent form of AP. Mildly enlarged LA, which was probably associated with AF occurrence, might contribute to intra-atrial conduction disturbance, probably related to this result. Second, we focused on the anatomical relationship of previous and present AP; whether AP was single, broad, or multiple bundles remained unclear. In the series of sessions, both retrograde and antegrade AP were blocked within a few seconds of RFA, which was an unfavorable phenomenon in broad AP and suggested two possibilities: (1) single AP with bidirectional conduction developed into unidirectional conduction after RFA or (2) two closely located APs existed. We considered that the former possibility was more natural and easier to understand than the latter, as previously reported. Third, intravenous ATP has been widely used in confirming the dormant PV-LA conduction after PV isolation and has been considered helpful for confirming the presence of dormant AP conduction and elucidating its location more precisely, resulting in a higher success rate for catheter ablation. Furthermore, it is also accepted that ATP could unmask the entity of intermittent antegrade AP. In the latest session, we utilized ATP injection to confirm the absence of dormant AP conduction, which might contribute to success. We would have revealed the entity of latent AP if ATP use had been attempted during the first session. Isoproterenol, which was also reported to have the potential to unmask latent AP conduction or to shorten the APERP, was not administered during the EPS series.

Antegrade AP, which contributes to ventricular pre-excitation, is sometimes responsible for antidromic re-entrant tachycardia or conduction during AF, resulting in subsequent development of ventricular fibrillation, a life-threatening arrhythmia. The intermittent form of AP has been recognized and this subgroup was considered safer than others. However, it was also reported that there was no difference in ERP frequency < 250 ms, which is known as high-risk AP between persistent AP and intermittent AP. Transient fast antidromic tachycardia episodes via adrenaline-dependent left lateral AP during emotions or exercise were reported in the previous literature. In our case, although EPS revealed an APERP of 270 ms, the patient experienced symptomatic irregular wide QRS tachycardia. These findings suggested that APERP cannot always predict the occurrence of such a potentially lethal arrhythmia. Therefore, intermittent or latent AP conduction is challenging, and we need to accurately detect and manage it to avoid potentially lethal arrhythmia in clinical practice.

This case highlighted the following crucial issues: (1) intra-atrial conduction delay or impaired AV nodal function might affect the increased frequency of the manifestation of AP conduction (in other words, AP conduction could be overlooked under specific conditions) and (2) even intermittent antegrade AP could provide a potential risk for the development of a potentially lethal arrhythmia. This was a meaningful case of overt AP that intermittently appeared late after catheter ablation of the concealed AP. We should always take into account this possibility to prevent the development of potentially lethal arrhythmia.

Figure 3. Intracardiac electrogram revealed the conduction block of the antegrade AP just after RFA. HRA indicates high right atrium; Abld, distal ablation catheter; Ablp, proximal ablation catheter; CSP, proximal coronary sinus; and CSD, distal coronary sinus.
Disclosure

Conflicts of interest: None

References