Adenosine Facilitates Transient Resumption of Accessory Pathway Conduction

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ABSTRACT. Adenosine is frequently used to induce atrioventricular nodal block to confirm successful ablation of accessory pathways and also to unmask dormant pulmonary vein conduction. We report an interesting case of an accessory pathway during which ventriculoatrial (VA) conduction through the pathway only recovered following adenosine administration. This finding was used to facilitate successful ablation of the accessory pathway. This observation may serve as a helpful maneuver in cases where mechanical trauma renders a pathway unmappable or to confirm successful radiofrequency ablation in the absence of VA conduction.

KEYWORDS. accessory pathway, adenosine, catheter ablation.

Introduction

Adenosine has been widely used in the electrophysiology (EP) laboratory to identify transient reconnection (dormant conduction) between pulmonary veins (PVs) and the left atrium after PV isolation.1 Moreover, intravenous (IV) adenosine has also been used as a diagnostic strategy to confirm the elimination of anterograde and retrograde conduction over an ablated accessory pathway (AP) by prolonging atrioventricular (AV) nodal conduction time and/or causing transient AV block favoring conduction over the AP.2,3 Alternatively, another possibility that has not been well described is that IV adenosine may enhance conduction over an AP, which would explain why after adenosine AP VA conduction time tends to be shorter than AV node VA conduction time. We report an interesting case where accessory pathway VA conduction was inhibited by mechanical trauma. Resumption of conduction through the pathway was only seen following adenosine administration.

Case history

A 62-year-old man with a past medical history of non-ischemic cardiomyopathy and a baseline left bundle branch block branch block was brought to the electrophysiology (EP) laboratory for assessment of palpitations and a sustained wide complex tachycardia (left bundle branch block (LBBB) morphology) at a rate of 190 bpm. A standard EP study was performed with multipolar catheters positioned in the right ventricle (RV), coronary sinus (CS), and the His bundle. The baseline findings were RR, 790 ms; PR, 185 ms; QRS, 170 ms; QT, 422 ms; AH, 100 ms; and HV, 59 ms. Programmed stimulation was performed from the CS and RV apex. Retrograde conduction was found to be concentric (CS proximal earlier than CS distal) and non-decremental.

With ventricular pacing using a single extrastimulus, wide complex tachycardia with an identical LBBB morphology to baseline was induced (cycle length 390 ms). The VA time during tachycardia was 92 ms (Figure 1). The earliest atrial activation during tachycardia was found at the proximal His. Ventricular overdrive pacing during tachycardia revealed a V–A–V response with a PPI–TCL of 66 ms and SA–VA of 40 ms. A premature ventricular extrastimulus delivered during the His refractory period repeatedly either advanced the subsequent A or terminated tachycardia without reaching the A (Figure 1c). The tachycardia also terminated spontaneously in the retrograde limb (AP) on several occasions (Figure 1d).
While mapping the earliest A on the RA septum during tachycardia, there was loss of VA conduction, consistent with mechanical trauma to a parahisian accessory pathway. Although intravenous isoproterenol did not restore VA conduction, infusion of 12 mg of IV adenosine resulted in transient recovery of VA conduction (Figure 2). Only non-sustained atrioventricular reciprocating tachycardia (AVRT) could be induced after adenosine injection due to its transient effect. This was a reproducible phenomenon: transient resumption of VA conduction via the AP despite VA dissociation during RV pacing lasting for approximately 10–15 s after each adenosine bolus.

Subsequently, the patient was brought back 1 month later for repeat EP study. The earliest retrograde atrial activation during ventricular pacing was mapped and once again, mechanical trauma caused loss of VA conduction. Multiple boluses of adenosine were utilized to facilitate transient pathway recovery and allow successful mapping of the earliest atrial electrogram during ventricular pacing. Mapping was assisted by a three-dimensional electroanatomical mapping system (CARTO, Biosense-Webster, Diamond Bar, CA). The ablation catheter electrogram at the site of earliest activation had a large His deflection (Figure 3). Therefore, radiofrequency ablation was performed during sinus rhythm to allow monitoring of anterograde AV nodal conduction. Initial applications were performed here and more “ventricular” from this location to try to minimize the likelihood of AV nodal injury. After each radiofrequency (RF) application, the persistence of pathway conduction was checked during ventricular pacing after adenosine administration. Using this strategy, ablation of the parahisian accessory pathway was successful while maintaining intact anterograde AV nodal conduction.

**Discussion**

The presence of a septal VA time of >70 ms, a V–A–V response during tachycardia, ventricular overdrive pacing with a PPI-TCL of <115 ms and SA–VA of <85 ms and repeated termination of the tachycardia with PVCs.
Figure 2: Effect of adenosine of VA conduction during right ventricle (RV) apical pacing despite VA dissociation. (a) VA dissociation caused by manipulation of the His catheter during mapping. (b) The effect of adenosine on anterograde AV conduction causing AV block. Then RV pacing was initiated and VA conduction is seen through the concealed accessory pathway (AP) after the fourth captured ventricular beat. No VA is present when RV pacing is initiated. (c) The transient effect of adenosine on the AP during RV pacing as loss of VA conduction is seen after three initial beats.

Figure 3: The signal during sinus rhythm from ablation catheter at successful ablation site (site of earliest atrial activation during ventricular pacing). Note the His deflection (H). A: atrial electrogram; V: ventricular electrogram.
delivered during His refractoriness without reaching the atrium provided proof of the presence and participation of a concealed septal accessory pathway. However, manipulation of the His catheter caused mechanical trauma on the parahisian accessory pathway leading to loss of VA conduction (VA dissociation at different RV pacing rates). Despite the absence of VA conduction during RV pacing at 600 ms, an 18 mg bolus of IV adenosine was administered in an attempt to cause the AP and/or the atrial insertion of the AP to hyperpolarize and thus reveal dormant conduction analogous to the phenomenon seen in the PVs during an AF ablation procedure.

**Resumption of VA conduction during RV pacing**

The initial dose of adenosine was given during continuous RV pacing and transient resumption of the VA conduction was noted within seconds. Although suggested, the effect of adenosine on the pathway could not be proven. Therefore, a second dose of adenosine was given during sinus rhythm and RV pacing was initiated only after adenosine-induced anterograde AV block (Figure 2). Surprisingly, resumption of VA conduction was seen at adenosine’s peak effect within a few seconds after anterograde AV block making it very unlikely to be AV-node retrograde conduction (adenosine would be expected to cause retrograde block via the AV node rather than facilitate conduction).

**Effect of adenosine**

The cardiac effects of adenosine are primarily attributed to inhibition of automaticity and conduction over the AV node, and to a lesser extent at the SA node. As a result, adenosine is widely used as a diagnostic tool to confirm absence of AP conduction following catheter ablation. In addition, the ability of adenosine to inhibit conduction in accessory pathways, particularly those with decremental conduction properties, has been previously described. Recent publications have proposed a new role for adenosine in unmasking dormant conduction within PVs and superior vena cava (SVC) during ablation and isolation of these structures. One possible mechanism of action may include cellular hyperpolarization, making the cell more amenable to electrical depolarization. In our case, the accessory pathway lost its conduction capacity as a consequence of mechanical trauma. However, adenosine administration transiently restored VA conduction. No information is available on the direct effect of IV adenosine on the anterograde and retrograde effective refractory periods of accessory pathways. One explanation for the observed phenomenon may take into account the direct effect of adenosine on the AP and/or its atrial insertion, similar to the effect exerted on atrial myocardium following pulmonary vein isolation (PVI), i.e., shortening the atrial effective refractory period and cellular hyperpolarization. As anticipated, the effects of adenosine on the AP were transient.

**Conclusion**

This report provides an unusual finding of transient restoration of accessory pathway conduction during adenosine administration. This finding was used to successfully ablate the accessory pathway. This report indicates that administration of IV adenosine may serve as a helpful maneuver in cases where mechanical trauma renders a pathway unmappable or to confirm successful radiofrequency ablation in the absence of VA conduction.

**References**