Resumption of dormant accessory pathway conduction with adenosine administration: a simple intervention to ensure successful accessory pathway ablation

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A 19-year-old man with documented supraventricular tachycardia was referred for a catheter ablation procedure. Atrioventricular reentrant tachycardia utilizing left posteroseptal accessory pathway (AP). After successful radiofrequency ablation, there was no evidence of (AP) conduction during a 1 h waiting period. However, administration of adenosine resulted in transient resumption of the AP conduction. We discuss the mechanism of action and the role of adenosine in unmasking dormant conduction.

A 19-year-old with supraventricular tachycardia was referred for an electrophysiology study (EPS). Delta waves were absent on all pre-procedural electrocardiograms. The HV interval was 36 ms and ventriculoatrial (VA) conduction non-decremental. Orthodromic atrioventricular reentrant tachycardia utilizing a left-septal accessory pathway (AP) was diagnosed and radiofrequency (RF) energy delivered at

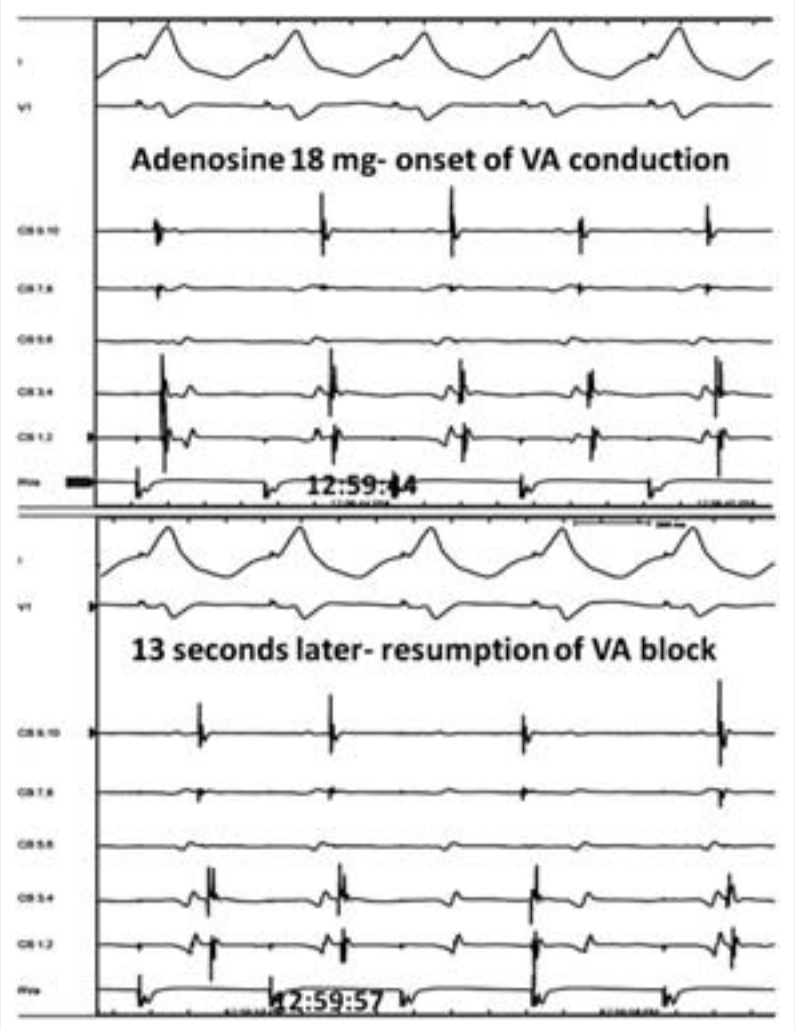

Figure 1 Effect of Adenosine on VA conduction. (*A*) Administration of Adenosine during ventricular pacing resulted in temporary retrograde AP conduction. (*B*) With Adenosine washout, VA block resumed.

the site of earliest retrograde atrial activity during ventricular pacing. Loss of VA conduction occurred during ablation and was maintained for 1 h post-ablation. However, administration of intravenous adenosine (18 mg) during ventricular pacing resulted in transient (12 s) resumption of VA conduction (*Figure 1*). As VA conduction was absent at various pacing rates and adenosine inhibits atrioventricular (AV) nodal conduction, our findings were consistent with transient retrograde AP conduction. As VA conduction block persisted for 1 h post-ablation, the procedure was concluded. Unfortunately, the patient experienced post-procedure arrhythmia recurrence. A repeat EPS confirmed recurrent left-septal AP conduction in a similar location to that ablated.

This case highlights the potential utility of adenosine in unmasking dormant retrograde AP conduction post-ablation and in predicting long-term recurrence after a presumed successful AP ablation procedure. In this case, adenosine's effect was not mediated by its action on the AV node as VA conduction block was present prior to its administration. Rather, we hypothesize that adenosine likely had a direct effect on the injured tissue at the atrial insertion of the AP.

Cardiac tissue damaged by RF energy may lose excitability due to heat-induced inactivation of channels and cell membrane leakage resulting in a less negative resting membrane potential.[1] This change results in inactivation of I_{Na} channels with subsequent loss of tissue excitability.[1] Adenosine administration can facilitate hyper-polarization of injured cardiac tissue thereby restoring tissue excitability in injured, but still viable cardiac tissue.[1] As this effect is not observed in non-viable cardiac tissue, intra-procedural administration of adenosine may be valuable in identifying injured cardiac tissue, which may recover thereby resulting in future arrhythmia recurrence. As adenosine affects atrial and not ventricular tissue, this manoeuvre may be valuable in ablation targeting the atrial insertion sites.

While others have reported the value of adenosine in AP ablation procedures, it has always been in the setting of manifest pre-excitation and/or ongoing VA conduction post-ablation.[2,3] We demonstrate adenosine's utility in the setting of VA conduction block to assess for dormant AP conduction post-ablation. As the proposed mechanism is speculative, this manoeuvre should be reserved to cases with uncertainty.

Conflict of interest: none declared.

References

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