Masquerade: An unusual accessory pathway with ventricular insertion at the right–left sinus of Valsalva mimicking outflow tract ventricular tachycardia

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Introduction

Although rare, an accessory pathway (AP) can have a ventricular insertion in the region of the aortic sinuses of Valsalva.[1–4] This can be difficult to diagnose and may resemble outflow tract ventricular tachycardia (VT).[4] Despite the existence of various algorithms that can differentiate wide-complex supraventricular tachycardia (SVT) from VT, these conditions may be impossible to differentiate based on surface QRS morphology alone.[5,6]

Case report

A 31-year-old man with no medical issues and a structurally normal heart by echocardiography and cardiac magnetic resonance imaging presented with symptomatic, wide-complex tachycardia at 223 bpm (Figure 1A). Ventricular activation was consistent with outflow tract VT, with a QS complex in leads I, aVR, and aVL, as well as V_1 and V_2. The QRS was completely positive in leads II, III, and aVF, as well as V_3–V_6. In addition, electrical alternans (alternating QRS amplitudes) can be seen in both SVT and VT. The transition in lead V_3 with absence of an r wave in V_2 is suggestive of a left-sided outflow tract origin, possibly the right–left sinus of Valsalva with the QS in V_1.[7,8] While the patient was in the emergency room, administration of 12 mg of adenosine intravenously terminated the arrhythmia to sinus tachycardia, with a narrow QRS at 130 bpm.

Because of the patient's young age, structurally normal heart, and adenosine-responsive arrhythmia, an electrophysiological (EP) study was performed. During catheter placement, a premature atrial contraction (PAC) couplet resulted in similar QRS complexes as the wide-complex tachycardia and were reproducible (Figure 1B). The variable His-ventricle interval with these PACs was not consistent with aberrant conduction and was suggestive of an unusual AP ventricular insertion. Although a coincidental premature ventricular contraction couplet could mimic this tracing, pre-excitation was reproducible with atrial extrastimulus pacing and atrial overdrive pacing, consistent with an AP.

Continuation of the EP study demonstrated atrioventricular (AV) block at 320 ms. With rapid atrial pacing, antidromic tachycardia was inducible with a cycle length of 350 ms. A single PAC was inserted, which reset the tachycardia and pulled in the ventricle reproducibly, ruling out VT. Retrograde conduction was concentric and decremental, and retrograde dual AV nodal physiology (V-A jump) also was present. The AV interval was assessed at various atrial pacing cycle lengths during maximum pre-excitation. It was found to be slowly conducting but had a fixed AV time (nondecremental).

After the EP study, mapping for an AP potential was performed in the region of the ventricular insertion in the outflow tracts. Mapping (during atrial pacing with maximal pre-excitation) was performed in the right ventricular outflow tract, distal coronary sinus, and aortic sinuses of

KEY TEACHING POINTS

- Although numerous criteria/algorithms exist for differentiating wide-complex supraventricular tachycardia from ventricular tachycardia, they do not distinguish antidromic tachycardia from ventricular tachycardia.
- Antidromic tachycardia with a ventricular insertion of the accessory pathway in the outflow tract can mimic ventricular tachycardia.
- Maneuvers should be performed to carefully elucidate the etiology of candidate accessory pathway potentials. In the outflow tracts, valve closure artifact should be excluded.

KEYWORDS Accessory pathway; Outflow tract; Pathway potential; Ventricular tachycardia; Wolff-Parkinson White syndrome
(Heart Rhythm Case Reports 2022;8:752–755)

Funding Sources: None. Disclosures: Dr Sheldon reports compensation for services (speaking/honoraria) from Abbott, Boston Scientific, and Medtronic; and consulting fees from Biosense Webster. All other authors report no conflicts of interest. **Address reprint requests and correspondence:** Dr Seth Sheldon, Department of Cardiovascular Medicine, The University of Kansas Health System, 4000 Cambridge St, Kansas City, KS 66160. E-mail address: ssheldon@kumc.edu.

https://doi.org/10.1016/j.hrcr.2022.08.005

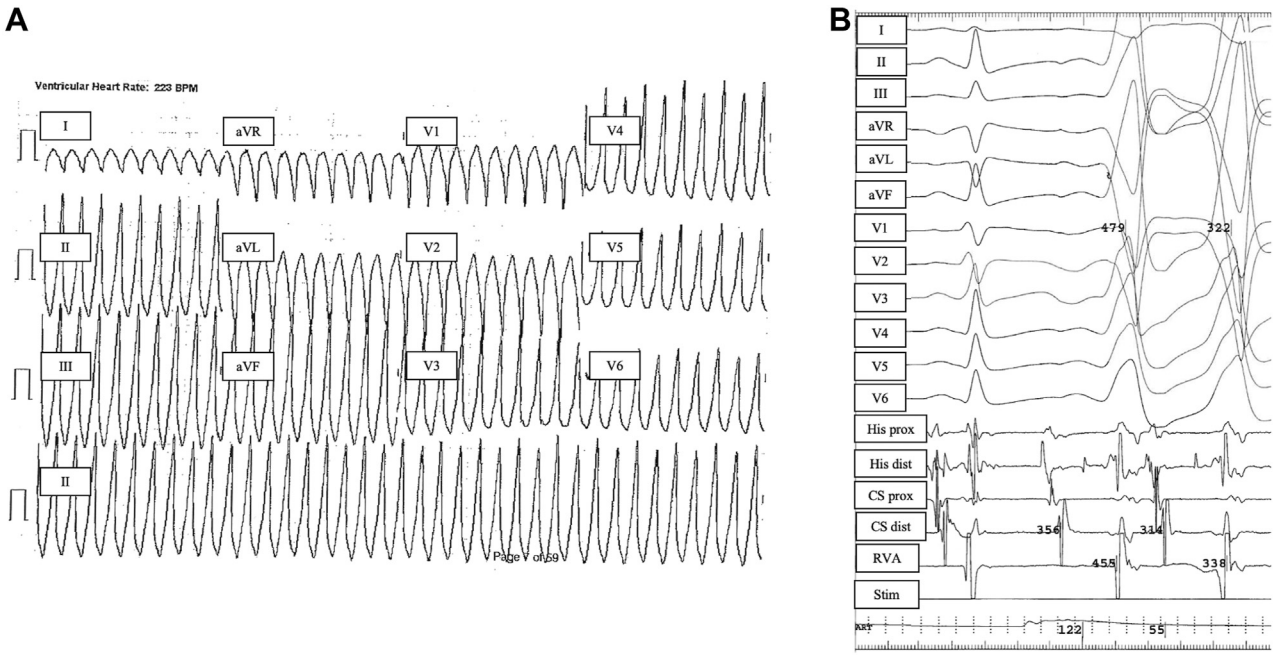

Figure 1 Twelve-lead electrocardiogram (ECG) of tachycardia and surface/intracardiac tracings from electrophysiological (EP) study. **A:** Twelve-lead ECG recorded in the emergency room at the time of patient presentation demonstrating outflow tract ventricular activation, potentially consistent with outflow tract ventricular tachycardia. Electrical alternans also is present. **B:** Surface and intracardiac tracings at the time of EP study showing a sinus beat on the left and response to a premature atrial couplet on the right. Ventricular activation following the premature atrial couplet is consistent with outflow tract origin. The His signal on the His distal electrogram is on time with ventricular activation, ruling out aberrant conduction. CS = coronary sinus; Dist = distal; Prox = proximal; RVA = right ventricular apex; Stim = stimulation channel.

Valsalva using a ThermoCool RMT Catheter (Biosense Webster; Irvine, CA) with Stereotaxis (St. Louis, MO) guidance. Ventricular activation was only 10–15 ms and 12 ms presystolic in the right ventricular outflow tract and distal coronary sinus, respectively. During mapping in the aortic sinuses of Valsalva, a candidate AP potential was seen (Figure 2A). Maneuvers were performed to assess the nature of this candidate potential, including pacing at a slower cycle length and assessment during sinus rhythm. As shown in Figure 2B, the candidate potential was seen at the end of the T wave and the timing from the candidate potential to the QRS during pre-excitation varied, consistent with aortic valve closure artifact.

Eventually, an AP potential was identified (Figure 2C) that was 82 ms presystolic and located at the right–left aortic sinus of Valsalva near the commissure (Figure 3). Pacing maneuvers were consistent with an AP potential. Pacing was performed at the site at 10 mA/2 ms, with an 11/12 lead match to ventricular pre-excitation (90% PASO®; https://www.jnjmedtech.com/en-US/product/paso-module). Retrograde atrial activation remained concentric with pacing from this site. Ablation performed at 40–45 W during atrial pacing with pre-excitation eliminated pathway conduction in 3.8 seconds. We waited >60 minutes and observed no return of pathway conduction. Isoproterenol 4 μg/min was administered to confirm no other atrial arrhythmias and intact AV nodal conduction. Parahisian pacing response was nodal. As of 3-year follow-up, the patient has not experienced further episodes of AV re-entrant tachycardia.

Discussion

The initial 12-lead electrocardiogram was consistent with structurally normal VT from the outflow tract. Adenosine responsiveness has been described with outflow tract VT and is not diagnostic of an AV node–dependent arrhythmia.[9] Furthermore, there was no evidence of pre-excitation or an abnormally short P-R interval on electrocardiogram recorded in sinus rhythm suggesting an AP. Thus, the decision was made to induce VT during EP study.

Once the EP study demonstrated the presence of an AP with antidromic tachycardia, we mapped for the ventricular insertion and searched for a possible AP potential, as mapping of the AP is ideally accomplished with identification of a pathway potential during fully pre-excited complexes.[10] A unique challenge during AP potential mapping for APs with insertion into the aortic sinuses of Valsalva is aortic valve closure artifact, which is observed in up to one-third of cases during mapping within the aortic cusps[11] and could be mistaken as an AP potential. Fortunately, similar to distinguishing aortic valve closure artifact from early ventricular activation with sinus of Valsalva origin premature ventricular contractions/VT, careful assessment of the potential at different rates and with pacing maneuvers can differentiate aortic valve closure artifact from an AP potential, as demonstrated in this case.

Although ablation of anteroseptal APs occasionally has been performed from the adjacent noncoronary aortic sinus of Valsalva,[12–14] APs with insertion directly into the aortic sinuses of Valsalva are rare. At least 4 cases of APs ablated successfully in the left aortic sinus of Valsalva have been

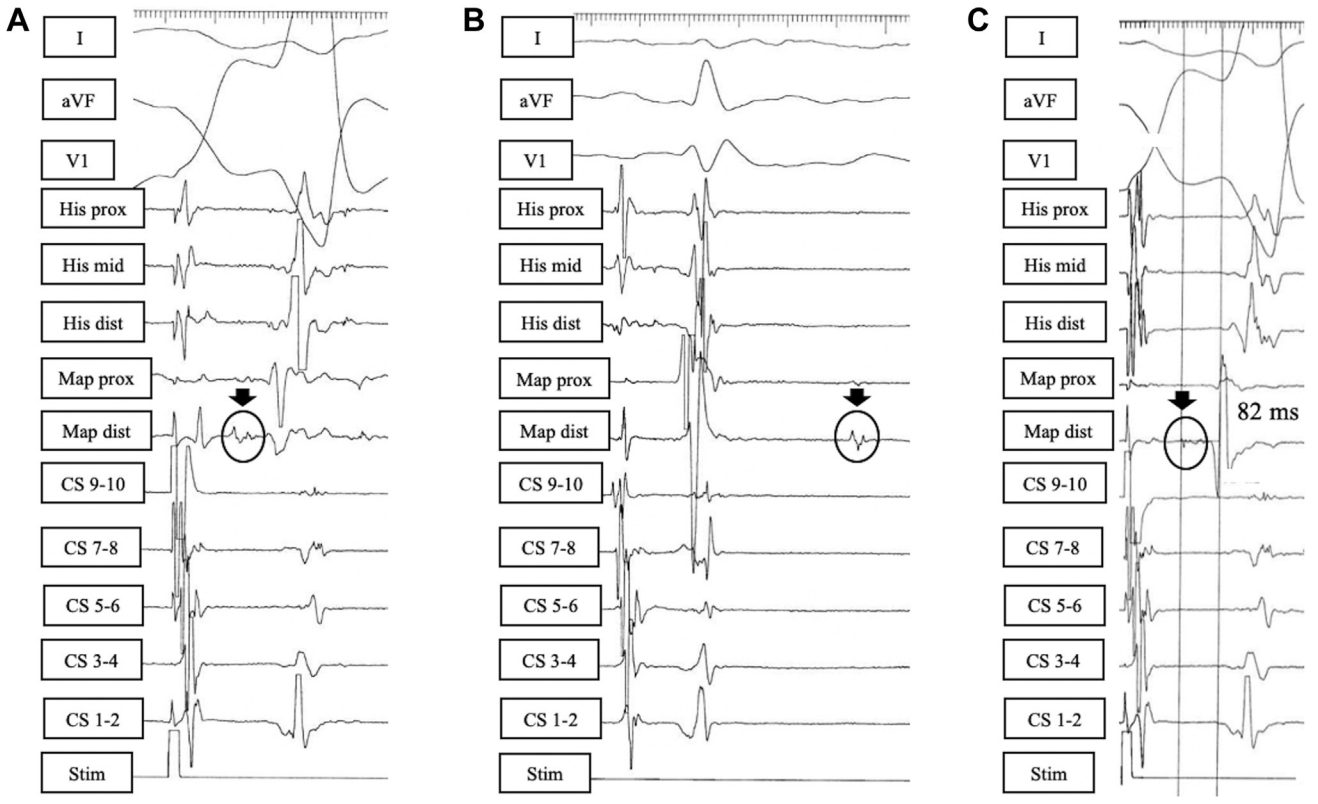

Figure 2 Candidate accessory pathway potentials. **A:** Surface and intracardiac tracings during atrial pacing from the proximal coronary sinus. The potential *(circle)* occurs between atrial and ventricular activation. **B:** Assessment of the candidate potential during sinus rhythm demonstrates a similar potential at end-systole timing with aortic valve closure at the end of the T wave. **C:** Candidate potential at the right–left aortic sinus of Valsalva. This potential had a consistent pre-QRS timing at various pacing rates and was not present at the end of the T wave, consistent with an accessory pathway potential. Map = ablation signal; other abbreviations as in Figure 1.

reported, 2 that were anterograde only[3,4] and 2 that were bidirectional.[1,2] These pathways can be adenosine-sensitive by report.[4] We report a unique and, to our knowledge, unreported slowly conducting, nondecremental, anterograde-only AP with insertion into the right–left aortic sinus of Valsalva. This site is anatomically distant from the septum. Whether

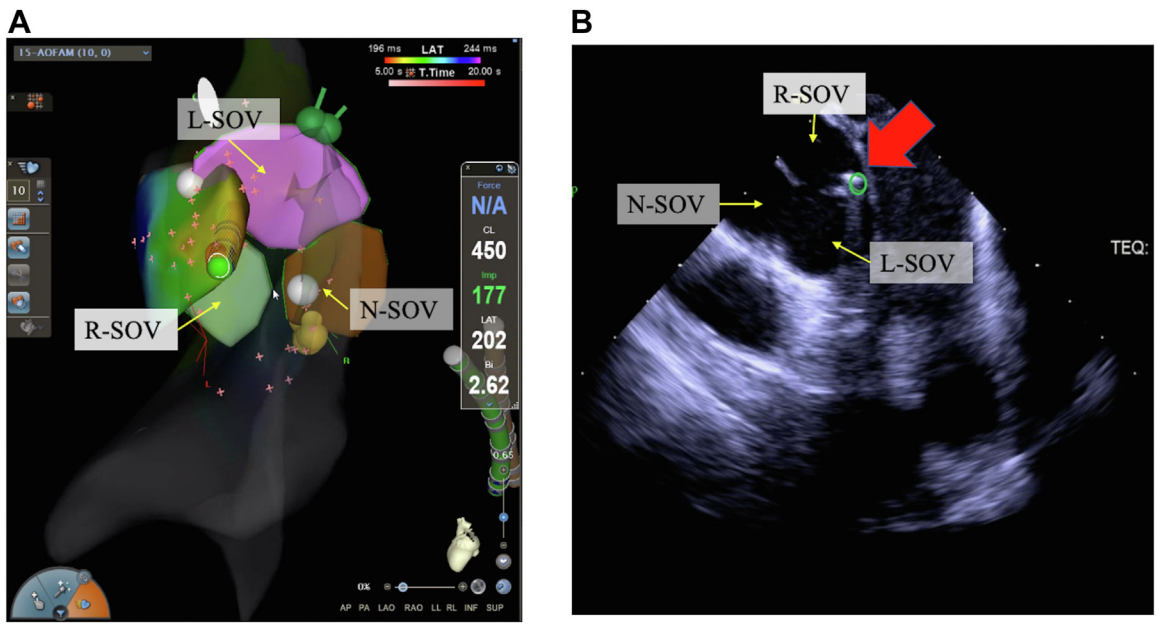

Figure 3 Activation Map and Intracardiac Echocardiogram Images. **A:** Activation map in the region of the right–left aortic sinus of Valsalva. The labeled aortic cusps were drawn from the CARTO Sound module. **B:** Ablation catheter (*red arrow* with tip in *green circle*) at the right–left aortic sinus of Valsalva at the successful site for ablation. The aortic valve cusps are labeled. L-SOV = left aortic sinus of Valsalva; N-SOV = noncoronary aortic sinus of Valsalva; R-SOV = right aortic sinus of Valsalva.

the length of the AP contributes to the slow conduction is uncertain. Unfortunately, we could not map the atrial insertion site due to the presence of anterograde-only conduction. If AP was bidirectional, it would have been interesting to see whether it inserted into either the right or left atrial appendage.

Conclusion

We report a case of wide-complex tachycardia due to an anterograde-only, slowly conducting AP with an AP potential and ventricular insertion in the region of the right–left aortic sinus of Valsalva mimicking VT. AP insertion into the aortic sinus of Valsalva should be considered in the differential diagnosis of wide-complex tachycardia resembling outflow tract VT. When mapping for an AP potential in this region, careful analysis of the candidate potential with diagnostic maneuvers may be necessary to exclude aortic valve closure artifact.

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