Duodromic tachycardia: A case report of a rare presentation of wide complex supraventricular tachycardia

Andrew Headrick, MD, MPH,* Claire Newlon, MD,[†] Susan P. Etheridge, MD, FHRS,*
S. Yukiko Asaki, MD, FHRS,* Thomas Pilcher, MD, FHRS,* Mary Niu, MD, MS*

From the *Department of Pediatrics, Division of Cardiology, University of Utah and Primary Children's Hospital, Salt Lake City, Utah, and [†]Department of Pediatrics, Division of Pediatric Cardiology, UCLA David Geffen School of Medicine, Los Angeles, California.

Introduction

Atrioventricular reentrant tachycardia (AVRT) requires an antegrade and retrograde limb to facilitate tachycardia. In most cases, this involves the atrioventricular (AV) node, which commonly serves as the antegrade limb, but can participate in either limb. Less commonly, reentry uses 2 separate accessory pathways (APs) with distinct conductive properties, without participation from the AV node.

Case report

A 13-year-old previously healthy male patient presented to the emergency department for abrupt onset of palpitations at rest. His Apple Watch® recorded a heart rate over 200 beats per minute. He was alert, warm, and well perfused. An electrocardiogram (ECG) demonstrated wide complex tachycardia (WCT) with right bundle branch block (RBBB) morphology and superior axis (Figure 1A). On the 12-lead rhythm strip, there was subtle variation in the QRS duration and morphology (Figure 1B).

Adenosine 6 mg, followed by 12 mg, was given via an antecubital intravenous (IV) insertion, with no effect on rhythm or ventriculoatrial (VA) conduction. Verapamil 5 mg IV was given, also without effect. The patient was sedated, and synchronized conversion was attempted with 50 joules of energy, which did not terminate the tachycardia.

He was transferred to the cardiac intentive care unit, where an echocardiogram demonstrated normal intracardiac anatomy and normal biventricular systolic function. Amiodarone IV was administered (150 mg IV slow bolus followed by continuous 5 mcg/kg/min infusion). This slowed but did

KEY TEACHING POINTS

- A presenting wide complex tachycardia carries a broad differential, and an organized approach can elucidate the mechanism.
- Subtle electrocardiographic changes following adenosine and cardioversion may provide evidence for rare and unique reentry mechanisms.
- Electrophysiology study maneuvers can be used to differentiate complex reentrant arrhythmia mechanisms.

not terminate the tachycardia. Although the patient remained alert and responsive, there was an interval decrease in peripheral perfusion. Synchronized cardioversion (50 J) was repeated, with termination of his arrhythmia and transition to sinus rhythm with ventricular pre-excitation. The QRS in pre-excited sinus rhythm had a left bundle branch block (LBBB) morphology and was distinctly different from that in WCT (Figure 1A–1C). The presence of 2 distinct QRS morphologies in sinus rhythm vs WCT was indicative of 2 separate APs with discrete antegrade conduction properties, LBBB morphology in sinus rhythm and RBBB morphology in WCT. Failure of adenosine to terminate the WCT suggested that the AV node was not a requisite part of reentry. Rather, the reentry mechanism used 2 separate APs for each limb of AVRT, specifically a duodromic tachycardia.

An electrophysiological study (EPS) was performed the following day. The presenting rhythm was pre-excited sinus rhythm with LBBB morphology, with the earliest site of ventricular activation localizing to the His channels. With decremental atrial pacing, antegrade conduction changed at 290 ms from LBBB to RBBB pre-excitation pattern (Figure 2A). While the AV interval on the coronary sinus (CS) channel remained unchanged at 50 ms, the AV interval
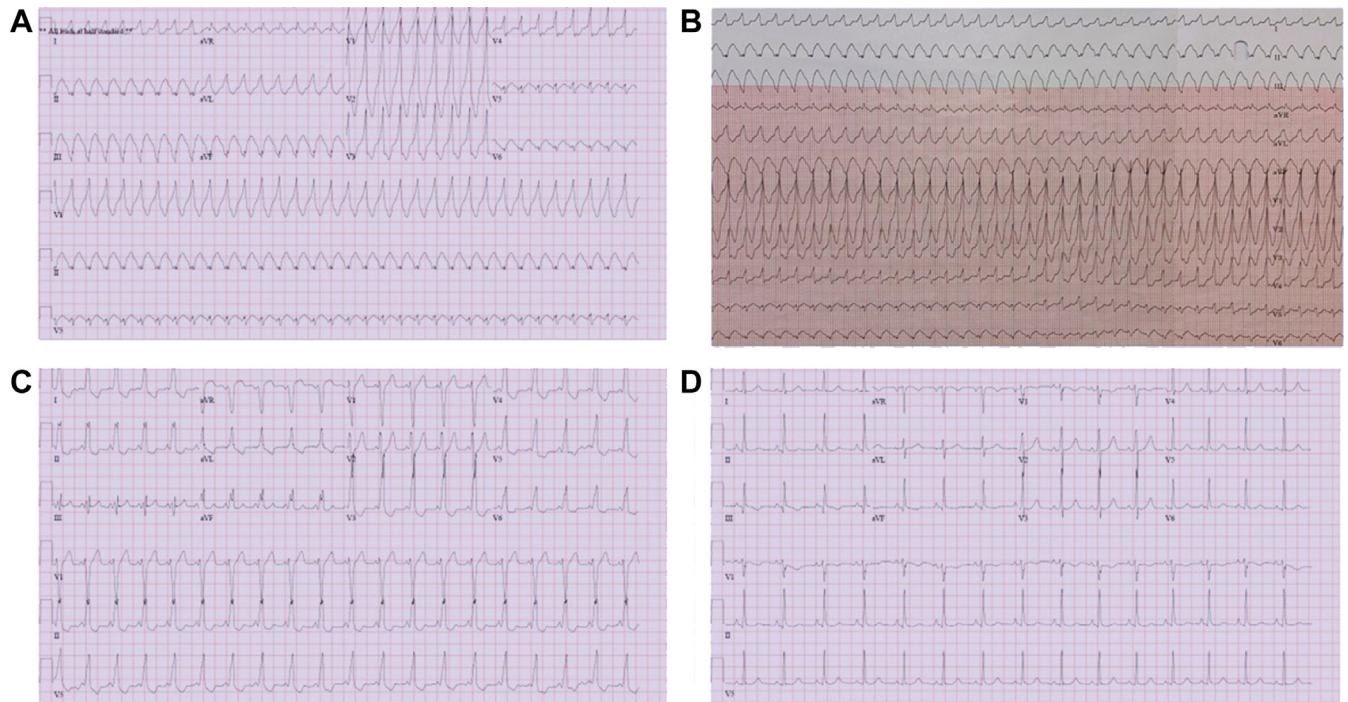
Figure 1   **A:** Presenting electrocardiogram (ECG) with a wide complex tachycardia with a right bundle branch block pattern and superior axis. **B:** Twelve-lead rhythm strip with subtle variation in the QRS duration and morphology in the precordial leads. **C:** Ventricular pre-excitation in sinus rhythm following cardioversion has a left bundle branch block morphology, distinctly different from panel A. **D:** Postablation ECG in sinus rhythm with no pre-excitation.

on the His channel increased from 58 to 138 ms. These findings were consistent with antegrade block through the right-sided AP. The left-sided AP block cycle length was 210 ms. No antegrade nodal beats were observed at this point. With atrial extrastimulus pacing, differences in antegrade conduction were again seen: the right AP effective refractory period (ERP) occurred at a longer coupling interval of 600/340 with the change in antegrade conduction corresponding with a change in pre-excitation (Figure 2B). Antegrade conduction over the left-sided AP remained nondecremental until it blocked at 600/280 ms.

Differences in retrograde conduction over the 2 APs were unmasked with decremental right (para-Hisian) ventricular and extrastimulus pacing. The left AP VA conduction was more brisk, conducting at shorter cycle lengths and coupling intervals. As such, during decremental pacing, the right retrograde AP block cycle length at 290 ms was associated with VA separation on the HRA channels (Figure 2C) while the local VA conduction on the distal CS—corresponding to retrograde conduction over the left AP—remained unchanged. Similar changes were unmasked at the right AP ERP of 400/270 ms (Figure 2D). Importantly, ventricular extrastimulus pacing elicited reentry beats that unmasked antegrade conduction through the AV node (Figure 2D).

The left AP was the first ablation target owing to the following rationale: First, since antegrade and retrograde conduction were exclusively over the left AP at shorter cycle lengths, we could exclude right AP conduction by pacing at shorter cycle lengths. Second, the local activation on the CS

catheter was consistent even when retrograde conduction was fused over the right AP and left AP. As such, separation in VA timing in the distal CS could serve as a marker for ablation success. Finally, we could reliably promote retrograde conduction over the left AP with retrograde left ventricular (LV) pacing, even at slower cycle lengths (Figure 3A). Ultimately, the left AP was successfully ablated using an irrigated radiofrequency (RF) contact force catheter in an SL1 (long) sheath through a transseptal approach. At 3 o'clock on the mitral valve annulus (Figure 3D), there was loss of retrograde left AP conduction with 3.2 seconds of RF energy.

Mapping of the right AP was performed in pre-excited sinus rhythm and para-Hisian pacing (Figure 3B and 3C). Because the earliest atrial and ventricular activation localized to 12 o'clock on the tricuspid valve annulus (Figure 3E), RF ablation was performed in sinus rhythm to monitor antegrade conduction through the AV node. RF application resulted in loss of pre-excitation in 2.6 seconds. However, with ventricular pacing there was persistent VA conduction with and without adenosine administration. With additional mapping (via ventricular pacing), early atrial signals were identified just lateral to the initial lesion set. These findings delineated an oblique AP course with the ventricular insertion slightly more lateral to the atrial insertion. An SR1 (long) sheath was used to optimize catheter stability and contact. RF application resulted in retrograde AP block in 2 seconds (Figure 3C) and subsequent AV and VA block with adenosine. Following ablation, AV nodal conduction was normal (Figure 1D).
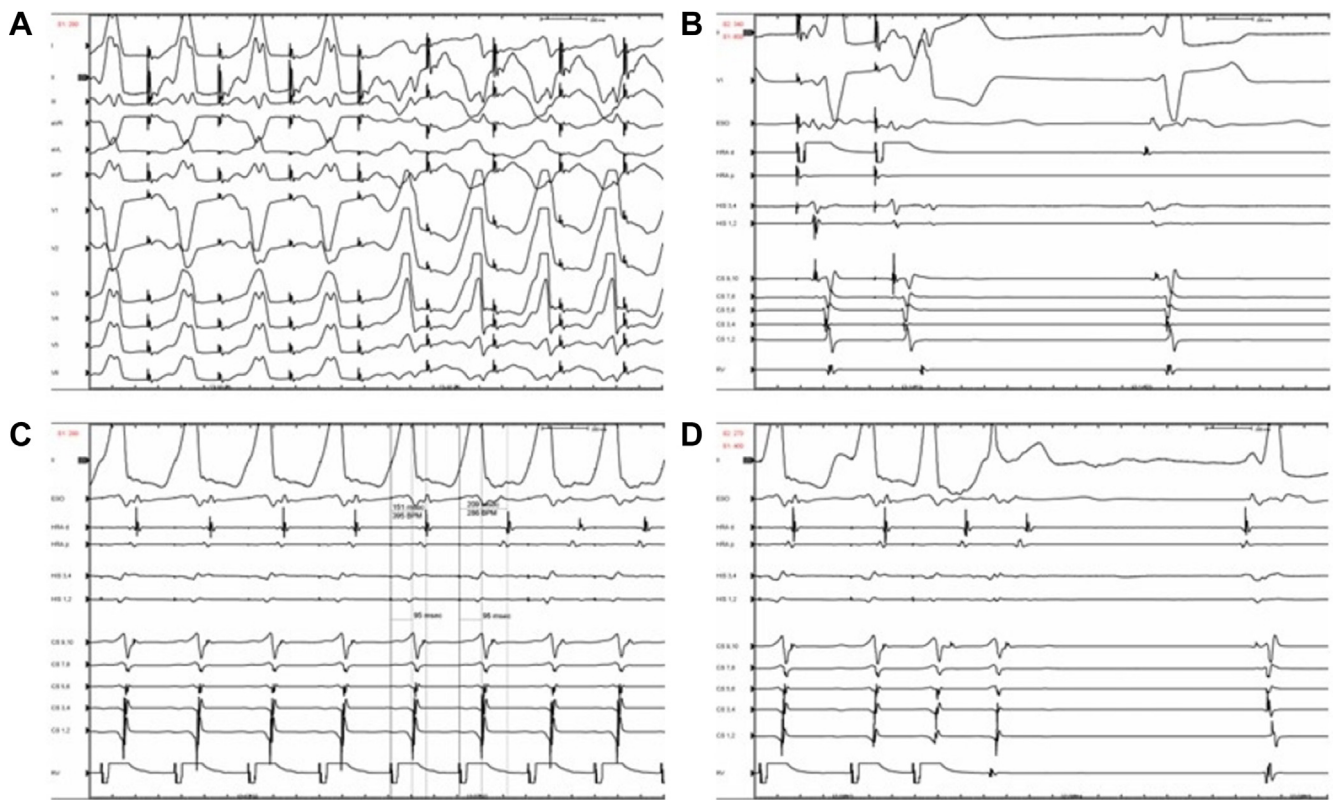
Figure 2   Differences in conduction unmasked with atrial and ventricular pacing maneuvers. **A:** During decremental atrial pacing from the high right atrium (HRA), at 290 ms pre-excitation changes from left bundle branch block (LBBB) to right bundle branch block (RBBB) morphology on the surface leads. **B:** The right accessory pathway (AP) effective refractory period occurred at 600/340 ms and corresponds to a change in pre-excitation from LBBB to RBBB. **C:** During para-Hisian ventricular decremental pacing, the local ventriculoatrial (VA) conduction on the HRA channel lengthens at 290 ms while the VA conduction on the coronary sinus (CS) channels remain unchanged. **D:** Para-Hisian ventricular extrastimulus pacing at 400/270 results in retrograde block over the right AP and exclusive retrograde conduction over the left AP; this is followed by antegrade conduction through the atrioventricular node.
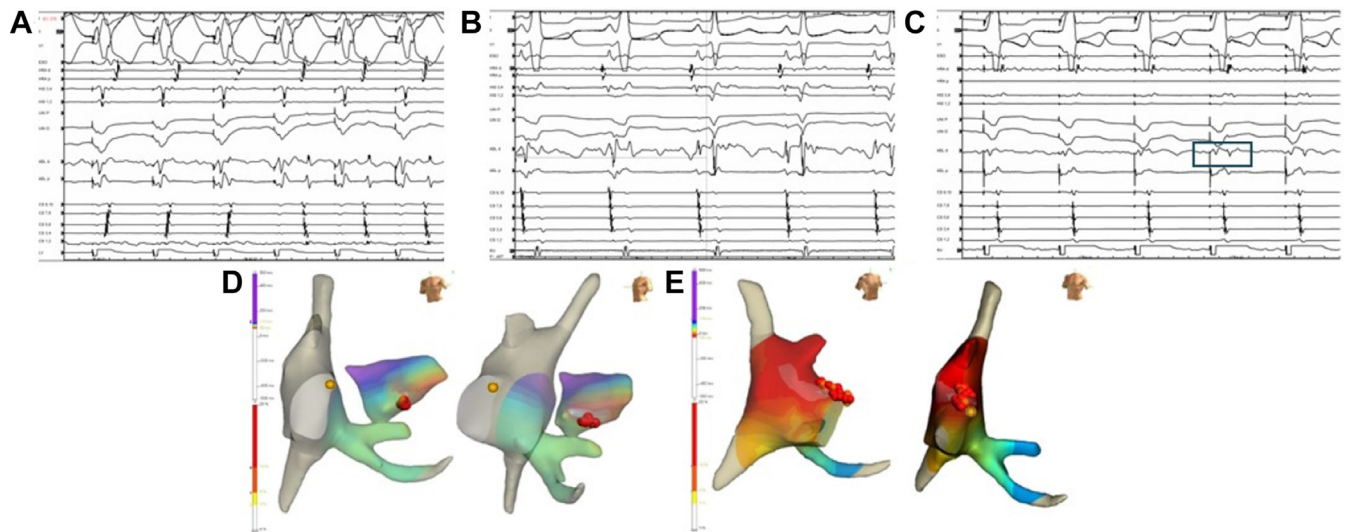

Figure 3   **A:** Mapping and ablation of the left accessory pathway (AP) with differential left ventricular (LV) pacing localizes the earliest retrograde activation to 3 o'clock on the lateral mitral valve annulus (**D**). Radiofrequency (RF) application results in retrograde block through the left AP and local ventriculoatrial separation on the ablation channels (**A**). Note that even at longer cycle lengths (500 ms in panel A), LV pacing promotes conduction over the left AP and the earliest atrial signals are localized to the distal coronary sinus (CS). **B:** Mapping and ablation of the right AP in pre-excited sinus rhythm localizes the earliest retrograde activation to 12 o'clock on the tricuspid valve annulus (**E**). **C:** Para-Hisian right ventricle mapping and ablation of the right AP. Following RF application, the VA conduction changes on the distal ablation channel (C, blue box). Red spheres indicate RF lesions, while yellow sphere localizes His.

Discussion

This case illustrates a rare case of duodromic tachycardia that was recognized owing to careful attention to changes in QRS morphology with clinical course and a methodical approach to the EPS. The 2 bidirectional pathways were successfully ablated.

With this case example, we describe a comprehensive approach to WCT in a child. At presentation, the patient was hemodynamically stable, which provided an opportunity to methodically work through the differential diagnosis of WCTs. This includes ventricular tachycardia (VT), orthodromic reentrant tachycardia (AVRT) with aberrancy, antidromic reciprocating tachycardia, Mahaim tachycardia, and atrial tachycardia with pre-excitation or aberrancy.

Although electrocardiographic differentiation of VT from supraventricular tachycardia (SVT) with aberrancy is not always possible, certain ECG features support reentrant SVT over VT. Not only does the ECG allow for assessment of the relationship between atria and ventricles, it also provides information on how the ventricles are activated. First, the presence of VA dissociation excludes AVRT SVT as the mechanism; VA association, however, does not exclude VT. Second, a rapid initial upstroke of the QRS complex (<30 ms) provides information about ventricular activation; a narrower and shorter initial upstroke in the QRS complex indicates ventricular activation utilizing or in proximity to the His-Purkinje system, suggesting SVT with aberrancy.[1,2] A narrow QRS can also occur with VT originating from the His-Purkinje system, such as a fascicular VT; but if the QRS during tachycardia is more narrow than sinus, VT should be suspected.[1,3] Third, precordial lead concordance (either positive or negative) argues in favor of VT, whereas its absence is more suggestive of SVT with aberrancy. Finally, the presence of QRS morphology changes despite a fixed cycle length during WCT is consistent with an underlying mechanism of reentrant SVT with aberrancy.

In this case, there was no evidence of VA dissociation at rest, there was no precordial lead concordance, and the width of initial upstroke of the QRS was not suggestive of activation near the His-Purkinje system. During WCT, the cycle length was fixed in the presence of subtle QRS changes (Figure 1). Collectively, these ECG data argued toward SVT with aberrancy over VT.

For any remaining ambiguity, adenosine can be a useful diagnostic maneuver. Its diagnostic utility lies in its ability to block conduction through the AV node and reveal whether the AV node is necessary for sustaining the arrhythmia. In this instance, adenosine failed to terminate the tachycardia. It did, however, lead to a subtle change in the QRS morphology and duration (Figure 1B) without changing the cycle length. In doing so, it excluded the AV node as a part of the mechanism of reentry even as reentrant SVT remained more probable than VT as the arrhythmia mechanism. The

subsequent failure to respond to verapamil made fascicular VT exceedingly unlikely.

In retrospect, the QRS change with adenosine revealed the antegrade limb of the reentrant arrhythmia at baseline to be a fusion between nodal conduction and aberrant AP conduction, while also excluding the AV node as a vital part of the arrhythmia circuit. This change in QRS morphology with exclusion of AV nodal conduction confirmed that the mechanism must not only be due to a reentry SVT, as inhibition of nodal conduction in VT would not have changed the QRS morphology, but also must be mediated by at least 2 separate accessory pathways. Taken together, these subtle but important findings supported the diagnosis of duodromic tachycardia.

Cardioversion is usually effective for terminating reentrant arrhythmias. As such, it is curious that cardioversion initially failed to restore sinus rhythm. A number of explanations are possible. First, although the initial energy was weight appropriate, it may have been insufficient for conversion. Second, following cardioversion, there may have been a few beats of sinus rhythm obscured by artifact before WCT resumed.

After the second cardioversion restored sinus rhythm, the presence of ventricular pre-excitation confirmed the existence of an antegrade-conducting AP, consistent with Wolff-Parkinson-White syndrome. The difference between the QRS morphology in pre-excited sinus rhythm vs that in tachycardia further substantiated the existence and involvement of a second AP in sustaining reentry tachycardia.

In considering the approach for the EPS, APs in Wolff-Parkinson-White can demonstrate complex ventricular insertions, and the presence of multiple APs is well documented in the literature. Typically, these pathways are closely related anatomically; more rarely, the pathways sit on separate valve annuli.[4] This highlights the importance, prior to proceeding with ablation, of elucidating the antegrade and retrograde properties of the respective APs as well as the AV node. A thorough and protocolized assessment and mapping both antegrade and retrograde allows for delineation of these insertion points.[4]

In this case, differential ventricular pacing from the para-Hisian right ventricle and LV was critical for differentiating the discrete conductive properties of each pathway and reliably isolating conduction over the target pathway during respective ablation. Specifically, in the presence of both a right- and left-sided AP, LV pacing is critical to promoting and isolating conduction over the left-sided AP. Additionally, it was equally important to delineate the conductive properties of the AV node and confirm antegrade AV nodal conduction prior to pursuing pathway ablation. Successful ablation of both APs resulted in sinus rhythm without pre-excitation (Figure 1D), and AV and VA block with adenosine administration.

This case demonstrates the necessity of a broad differential in managing WCT, and in particular differentiating

SVT from VT. Moreover, we describe duodromic tachycardia, a rare but potentially difficult-to-control AV reentrant mechanism utilizing a left and right AP as the antegrade and retrograde limbs, respectively, and refractory to adenosine given its mechanistic independence of the AV node.

For macroreentrant tachycardia to manifest utilizing the 2 pathways, independent of the AV node, they must demonstrate different conductive and refractory properties, they must be anatomically separated, and VA conduction via the AV node must not be present to interrupt the circuit.[5,6]

Conclusion

Duodromic tachycardia is a 2 pathway–mediated reentrant tachycardia is an uncommon entity that should be considered in the differential of a regular WCT. Suspicion of this mechanism must remain high, particularly when ECG features and additional diagnostic maneuvers point to a reentrant mechanism independent of the AV node.

References

1. Wellens HJ. Electrophysiology: ventricular tachycardia: diagnosis of broad QRS complex tachycardia. Heart 2001;86:579–585.
2. Pambrun T, El Bouazzaoui R, Combes N, et al. Maximal pre-excitation based algorithm for localization of manifest accessory pathways in adults. JACC Clin Electrophysiol 2018;4:1052–1061.
3. Ban J-E, Chen Y-L, Park H-C, et al. Idiopathic ventricular arrhythmia originating from the para-Hisian area: prevalence, electrocardiographic and electrophysiological characteristics. J Arrhythm 2014;30:48–54.
4. Zachariah JP, Walsh EP, Triedman JK, et al. Multiple accessory pathways in the young: the impact of structural heart disease. Am Heart J 2013;165:87–92.
5. Nakamura T, Kanda G, Sasaki Y, et al. Wide QRS tachycardia associated with multiple accessory pathways in a patient with Wolff-Parkinson-White syndrome. J Cardiol Cases 2019;20:191–196.
6. Yakabe D, Fukuyama Y, Araki M, Nakamura T. Bidirectional atrioventricular reentrant tachycardia using bilateral accessory pathways. J Cardiol Cases 2021; 23:115–118.