Successful radiofrequency ablation of junctional ectopic tachycardia in an adult patient

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Introduction
Junctional ectopic tachycardia (JET) is an electrophysiological phenomenon well described in pediatric patients, sometimes following surgical correction of congenital heart lesions.1–3 It characteristically presents with a rapid narrow complex tachycardia and atrioventricular (AV) dissociation, and is often refractory to antiarrhythmic therapy.1–3 JET is rare in adult patients, and it is associated with poor outcomes, including heart failure and complications of tachyarrhythmia. Medical therapy for JET often includes multiple antiarrhythmic agents and attempts at ablation in this region may result in AV block requiring permanent pacemaker insertion.

Case report
A 45-year-old woman presented to a walk-in clinic with a 3-day history of malaise and symptoms of an upper respiratory tract infection. She reported use of an over-the-counter remedy containing ibuprofen and pseudoephedrine during her illness. On initial assessment, her temperature was 38.5°C, with hypotension and a heart rate of 250 beats per minute (bpm). Vagal maneuvers and escalating doses of intravenous (IV) adenosine failed to affect the tachycardia.

At a community hospital, an electrocardiogram (ECG) showed a narrow QRS complex tachycardia at rates of up to 280 bpm (Figure 1A). She required several liters of crystalloid to augment her blood pressure. After IV diltiazem failed to provide a sustained reduction in heart rate, the patient was sedated and had 6 unsuccessful attempts at electrical cardioversion, with no interruption of her tachycardia. Over the next few hours, control of her tachycardia was attempted with IV esmolol, digoxin, magnesium sulfate, and a loading dose of 150 mg amiodarone. Owing to increasing oxygen requirements and pulmonary edema, the patient was diuresed with IV furosemide and transfer was arranged to a tertiary hospital cardiac intensive care unit for further support and management.

In the cardiac intensive care unit, the patient received a second loading dose of IV amiodarone and had 3 additional unsuccessful attempts at electrical cardioversion. After IV boluses of lidocaine and procainamide provided only transient reductions in heart rate, and with worsening respiratory status, the patient was placed under general anesthetic, intubated, and cooled to a target temperature of 35.5°C. On infusions of esmolol, lidocaine, and procainamide her heart rate slowed to 130 bpm and ECG demonstrated AV dissociation (Figure 1B). Her heart rate gradually increased over the next 12 hours and she required an infusion of vasopressin, but struggled to maintain a mean arterial pressure of 50 mm Hg.

The following morning, the patient was taken to the cardiac electrophysiology laboratory and anes-thetic in-suffusions were discontinued at the start of the case. Multipolar catheters were placed in the coronary sinus, His bundle region, and right ventricular (RV) apex. The initial tachycardia cycle length was 504 ms, with clear AV dissociation apparent (Figure 2A). His potentials preceded each ventricular activation and there was no evidence of split His signals. The HV interval in tachycardia or with atrial pacing was 59 ms. The tachycardia was almost incessant and reinitiation was spontaneous, without requirement for critical AH or AV delay. Scanning diastole with premature ventricular contractions from the RV catheter had no effect on the tachycardia cycle length, nor did atrial beats timed to septal refractoriness (Figure 2A). Occasionally, sinus P waves early in the diastolic interval would advance the immediate His potential, with subsequent continuation of the tachycardia. Ventricular overdrive pacing from the RV apical catheter demonstrated retrograde His activation (without conduction to the atrium) and on cessation of pacing there was a VHHV response (Figure 2B). Findings were in keeping with JET (see Discussion). Using an electroanatomic mapping system (Velocity, St Jude Medical) a limited geometry of the basal RV septal region was created and timing points were collected during the tachycardia for local sharp potentials relative to ventricular activation on the RV apical catheter (Figure 3).
The site with earliest local potentials was in the mid-septal region close to the plane of the tricuspid annulus, and catheter pressure here would reproducibly terminate the tachycardia. Cryoablation was considered but owing to patient instability and the fact that the site appeared to be more inferior than the compact AV node region, it was decided to proceed with cautious radiofrequency (RF) ablation. Initial lesions were delivered inferior to the site of earliest activation (Figure 3C), with power increased from 10 to 20 W, with no effect on the tachycardia. Ablation at the site where catheter pressure affected the rhythm terminated the tachycardia after 26 seconds, when the power reached 20 W. Slower junctional beats were seen during the remainder of the lesion. Two insurance lesions were delivered, 1 of which was terminated owing to transient AH prolongation. There was no return of the tachycardia during a 45-minute wait, including with administration of isoproterenol. A temporary atrial pacing wire was left in place owing to profound sinus node suppression (presumed secondary to the antiarrhythmic medication), and this was no longer required after 24 hours.

*Streptococcus pneumoniae* was grown on sputum cultures and this was treated with IV antibiotics. The patient was discharged 9 days later, and there was no recurrence of tachycardia during her hospital stay or in 9 months of follow-up. Of note, she had had 2 previous medical contacts with unstable tachycardia, 1 of which resulted in a prolonged admission to an intensive care unit. Although these presentations were in another country and detailed medical records were not available to us, the patient’s arrhythmia had been described to her as a supraventricular tachycardia and resolved during supportive medical treatment and administration of beta blockade.

**Discussion**

The differential diagnosis of a VA-dissociated tachycardia with more ventricular than atrial beats includes the

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**KEY TEACHING POINTS**

- Junctional ectopic tachycardia is rare in adults but can present with an incessant tachycardia, resistant to usual therapies.
- The dysrhythmia can result in a narrow QRS complex rhythm on electrocardiogram, with ventriculoatral dissociation. The differential diagnosis for this rhythm includes atrioventricular (AV) node reentry tachycardia and orthodromic nodoventricular or nodofascicular reentry tachycardia.
- In the electrophysiology laboratory, maneuvers including the response to ventricular extrastimuli and overdrive pacing can be useful to make the diagnosis.
- Ablation can be considered in cases where drug control is not successful, and can be aided by electroanatomic mapping. Cryoablation should be considered, given the risk of inadvertent AV block, although successful radiofrequency ablation has also been reported.

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**Figure 1**

A: Twelve-lead electrocardiogram showing a narrow QRS complex tachycardia at 280 beats/min with a rightward cardiac axis. Atrial activity is hard to discern, but with pharmacologic slowing of the ventricular rate to 180 beats/min (B, lead II rhythm strip), dissociated sinus P waves were apparent (arrows).
following: (1) AV nodal reentrant tachycardia with block in an upper common pathway, (2) nodoventricular or nodofascicular reentrant circuits, (3) JET, and (4) ventricular tachycardia (VT), including idiopathic (fascicular) VT and bundle branch reentrant VT. Other potential circuits such as intrahisian reentry owing to longitudinal dissociation of the His bundle have been proposed but never, to our knowledge, reported. The latter would be expected to show split His electrograms, which were not seen in our case. In the case presented here, the narrow QRS complex with evidence of orthodromic activation of the His-Purkinje system rules out myocardial VT and bundle branch reentry VT.

AV node reentry tachycardia with block in an upper common pathway seems unlikely for a number of reasons. Firstly, initiation of tachycardia did not require a critical AH delay. Secondly, no evidence of AV node slow pathway conduction was seen at other times during the case. Thirdly, the tachycardia did not terminate with adenosine or DC cardioversion. Fourthly, while intermittent VA block is occasionally seen during AV nodal reentrant tachycardia, persistent complete VA dissociation would be very unusual.

The lack of response to scanning diastole with premature ventricular contractions makes orthodromic nodoventricular or nodofascicular reentry less likely, but these circuits can be ruled out by the response to overdrive ventricular pacing shown in Figure 2B. If either circuit was present then entrainment from the ventricle should demonstrate orthodromic capture of the His potential (since the upper turnaround point of the circuit would be in the AV node) and on cessation of pacing a VHV response would be expected. In Figure 2B, retrograde His potentials are clearly seen, preceding local ventricular activation at the basal RV septum (and implying that the pacing site was most likely close to an arborization of the right bundle). The last entrained His potential is the one immediately following the pacing stimulus and on cessation of pacing the response is VHHV. It is fortuitous that retrograde His potentials were clearly recorded in this case, because the response to overdrive pacing would be harder to interpret in their absence.

The above findings strongly support a diagnosis of a focal JET. This also fits the clinical picture, with an almost incessant tachycardia unresponsive to vagal maneuvers, adenosine, multiple antiarrhythmic drugs, and DC cardioversion. The fact that it occurred in the presence of fever presumed secondary to a community-acquired pneumonia is also in keeping with this type of tachycardia, although it is extremely rare to see JET in an otherwise healthy adult patient with no history of congenital heart disease or cardiac surgery. The response to catheter pressure in a localized region and the site of successful ablation implies that the focus was in the inferior portion of the distal AV node and, indeed, slower junctional beats were seen during RF application. The site of successful ablation matches some other reports of this dysrhythmia.2–4

Figure 2 A: Surface electrocardiogram (ECG) and intracardiac electrograms during tachycardia with a cycle length (CL) of 504 ms. Catheters were placed at the His bundle region (His), coronary sinus (CS), and right ventricular apex (RV). Atrioventricular dissociation is apparent, with independent atrial activity visible on the surface ECG and on the CS catheter. Premature ventricular beats timed to His refractoriness (arrow) had no effect on the tachycardia CL, nor did paced or spontaneous (asterisk) atrial beats occurring at the time of septal refractoriness. B: Ventricular overdrive pacing during tachycardia with a CL of 480 ms. His activation (H) was accelerated to the pacing CL (460 ms), without conduction to the atrium. On cessation of pacing there was a VHHV response.
Prior to ablation, the patient had a declining clinical course, with profound hypotension and evidence of hypoperfusion, presumably secondary to her tachycardia, the multiple vasoactive agents used to try to control it, and possibly some vasodilatation owing to sepsis. Control of pediatric and postoperative JET has been attempted with a large number of antiarrhythmic agents and therapeutic hypothermia, with variable success. Often with supportive care the dysrhythmia will resolve, with ablation or even extracorporeal membrane oxygenation used in intractable cases. Given the location of the focus and risk of complete heart block, cryoablation should be considered, although successful RF ablation with preservation of AV conduction has also been reported.

We hope to raise awareness of this potentially life-threatening entity, which may occur in predisposed individuals during times of physiological stress or pharmacologic stimulation.
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References
1. Haas NA, Plumpton K, Justo R, Jalali H, Pohlner P. Postoperative junctional ectopic tachycardia (JET). Z Kardiol 2004;93:371–380.
2. Law IH, Von Bergen NH, Gingerich JC, Saarel EV, Fischbach PS, Dick M II. Transcatheter cryothermal ablation of junctional ectopic tachycardia in the normal heart. Heart Rhythm 2006;3:903–907.
3. Pierick AN, Law IH, Muldonado JR, Von Bergen NH. Junctional ectopic tachycardia localization and procedural approach using cryoablation. Pacing Clin Electrophysiol 2017;40:655–660.
4. Hamdan M, Van Hare GF, Fisher W, Gonzalez R, Dorostkar P, Lee R, Lesh M, Saxon L, Kalman J, Scheinman M. Selective catheter ablation of the tachycardia focus in patients with nonreentrant junctional tachycardia. Am J Cardiol 1996;78:1292–1297.
5. Dyamenahalli U, Tuzcu V, Fontenot E, Papagiannis J, Jaquiss RDB, Bhutta A, Morrow WR, Erickson CC, Imanura M, Prodhan P. Extracorporeal membrane oxygenation support for intractable primary arrhythmias and complete congenital heart block in newborns and infants: short-term and medium-term outcomes. Pediatr Crit Care Med 2012;13:47–52.
6. Han FT. Incessant palpitations and narrow complex tachycardia. Card Electrophysiol Clin 2016;8:61–65.