SUPRAVENTRICULAR TACHYCARDIA

COMPLEX CASE STUDY

Long RP Tachycardia: Diagnostic and Therapeutic Challenges: A Patient with Two Long RP Tachycardias

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ABSTRACT. A 30-year-old woman with a structurally normal heart and aborted cardiac arrest in the setting of exertion underwent a diagnostic electrophysiologic study. Two long RP narrow QRS tachycardias were induced during the study. The tachycardia characteristics and the results after ventricular entrainment showed a subtle difference between the two tachycardias. Allowing for proper diagnosis and treatment of each tachycardia, no supraventricular tachycardia could be induced after ablation at two separate sites. This is a rare and challenging case in which two long RP tachycardias with different mechanisms were diagnosed and treated in the same patient. To the best of our knowledge, this is the first report of this finding.

KEYWORDS. atypical atrioventricular nodal re-entrant tachycardia, decremental accessory pathway, long RP tachycardia, supraventricular tachycardia.

Case presentation

A 30-year-old woman with a structurally normal heart and aborted cardiac arrest in the setting of severe exertion underwent a diagnostic electrophysiologic study. Baseline sinus cycle length, AH interval, HV interval, QRS duration, QT interval, and QTc were 1185, 78, 48, 91, 427, and 392 ms, respectively. QRS morphology was normal with no pre-excitation. Both atrial pacing and ventricular pacing showed decremental atroventricular nodal conduction, and no jump was observed. Para-Hisian pacing demonstrated a typical nodal response. No ventricular tachyarhythmia was induced in response to programmed ventricular pacing at two drive cycle lengths and triple extrastimuli on or off isoproterenol.

A long RP narrow QRS complex tachycardia (Tach-1) was readily induced by atrial extrastimuli during isoproterenol infusion (Figure 1a). The tachycardia cycle length (TCL), AH interval, HV interval, and VA interval were 318, 75, 46, and 239 ms, respectively. It was found that the AA interval defined the HH interval when the tachycardia was slightly irregular (Figure 1b), and His synchronous premature ventricular contraction (PVC) did not affect the AA interval. The tachycardia terminated spontaneously with VA conduction block, and was also terminated by an early premature atrial contraction (PAC). The results of entrainment with ventricular pacing at 300 ms are shown (Figure 2ab). The postspacing interval (PPI) compared to TCL (PPI-TCL) after ventricular entrainment was 165 ms, and the difference between the stimulus-atrial (Stim-A) interval and VA interval (Stim-A–VA) was 162 ms. At other times, ventricular overdrive pacing during Tach-1 is shown (Figure 3).

Discussion

Differential diagnosis and catheter ablation

This patient presented with two long RP supraventricular tachycardias (SVTs). The differential diagnosis for these SVTs includes atrial tachycardia (AT), atypical atrioventricular nodal re-entrant tachycardia (AVNRT), orthodromic reciprocating tachycardia (ORT) using a
slowly conducting and decremental accessory pathway (AP), and ORT using a concealed nodo-fascicular/ventricular AP (NFRT). In this case, with the finding that the AA interval defined HH interval and that there was no jump in AH in response to programmed atrial stimulation, both of these observations were compatible with AT. However, AT was excluded with the observed VAV (pseudo-VAAV) response to right ventricular entrainment (Figure 2a) as well as tachycardia termination with VA block. The long PPI–TCL and the long Stim–VA after ventricular entrainment were both consistent with AVNRT. In addition, the third unfused beat advanced the next atrial activation during ventricular entrainment (Figure 2b). ORT was excluded with the observed atrioventricular (AV) dissociation during ventricular pacing (Figure 3). Taken together, the features of Tach-1 were most consistent with atypical AVNRT, and the initial RF was delivered to the slow pathway region.

Tach-2 was recognized by both a change in rate and more negative P waves in inferior leads and V1 lead (Figure 4b), which suggested the earliest atrial activation for Tach-2 was at the inferior tricuspid valve (TV) annulus. VAV (pseudo-VAAV) response to ventricular entrainment was also seen and excluded AT (Figure 5a). In general, the long PPI–TCL and the long Stim–VA difference seemed to be consistent with AVNRT in spite of the previous slow pathway modification. The long PPI–TCL difference suggested that right ventricular apical pacing was far outside the tachycardia circuit, and the long Stim–VA timing suggested that there was a change in VA activation sequence. These results are typically seen in the cases with AVNRT and not in the cases with ORT using a septal AP. However, it is reported that the same result could be seen in patients with ORT using a free wall AP or slowly conducting AP. In contrast, the third paced beat is fused and
advanced the next atrial activation at the beginning of ventricular pacing during Tach-2, which excluded AVNRT and proved the presence of an AP (Figure 5b). Furthermore, AV dissociation was not seen in Tach-2. A PVC delivered when the His was committed did not advance the next atrial activation for Tach-1, which argues against the presence of right-sided NFRT. Taken together, the features in Tach-2 were most consistent with ORT using a slowly conducting and decremental AP. In this case, Tach-2 was present after slow pathway modification, and it suggested that slow pathway modification allowed expression of the concealed AP.6

Using a three-dimensional electroanatomic mapping system during Tach-2, the earliest activation was mapped to the inferior TV annulus (Figure 6). RF was delivered in this site and terminated the tachycardia. SVT could no longer be induced after ablation.

Methods for differentiating long RP tachycardias

Many diagnostic maneuvers have been developed for distinguishing SVTs. However, all of them have some limitations, and the diagnosis of long RP tachycardia may be challenging.

Resetting the tachycardia by delivery of His-refractory PVC is one of the most useful maneuvers to identify the presence of an AP.7,8 However, this maneuver cannot determine a participatory versus bystander role during tachycardia, and there is frequently an absence of resetting following only a single ventricular stimulus with left-sided or decremental APs. Hirao et al found that para-Hisian pacing is useful for differentiating retrograde conduction over a robust septal AP compared with AV nodal conduction, but this maneuver will not distinguish nodal conduction from that via a decremental AP.9 Ormaetxe et al showed that the presence of ventricular fusion with resetting or entrainment of tachycardia occurred in all patients with ORT using a septal AP, but not in any cases of AVNRT.10 It is also reported that perturbation of the tachycardia in the transition zone (progressive paced QRS fusion) or within one fully paced complex during ventricular entrainment suggests a diagnosis of ORT,11,12 which was present in Tach-2 and proved the presence of an AP.

Michaud et al found that Stim-A–VA interval > 85 ms and PPI–TCL > 115 ms are useful in distinguishing atypical AVNRT from ORT using a septal AP.3 The ability of these criteria was supported and developed by many other studies.4,13 However, Bennett et al reported that these criteria cannot be applied to patients with slowly conducting concealed septal APs.5 Man et al reported...
that a delta AH (AH\text{atrial entrainment} – AH\text{SVT}) interval >40 ms was associated with atypical AVNRT, but 2 out of 12 atypical AVNRTs had a delta AH interval <40 ms in the same study.\textsuperscript{14} Miller et al found that delta HA (HA\text{ventricular entrainment} – HA\text{SVT}) interval >-10 ms was an excellent cutoff value for distinguishing atypical AVNRT from ORT.\textsuperscript{15} However, retrograde His potentials must be evident for this maneuver, and this is often not possible to establish during SVT. In fact, we were not able to recognize retrograde His potentials in this case. Martínez-Alday et al found that a difference in VA intervals >10 ms by RV pacing between the apical and posterobasal locations distinguished concealed posteroseptal pathways from AV node pathways, but this criterion cannot be applied to anteroseptal AP.\textsuperscript{16}

In summary, we present an unusual case of a patient with long RP tachycardia due to both atypical AVNRT and ORT using a decremental right-sided AP. After slow pathway modification followed by ablation of a right-sided AP, no SVTs were inducible. The diagnoses of both Tach-1 and Tach-2 were made without a catheter in the coronary sinus, and it was placed just before mapping and ablation for Tach-2 to confirm atrial activation, recognize anatomic location, and be used as a reference. We emphasize that the combination of the various diagnostic maneuvers was crucial for proper diagnosis and treatment. Although the relationship of the induced SVT and aborted sudden death for our patient remains uncertain, there are prior reports of aborted sudden death due to SVT.\textsuperscript{17} After follow-up of 10 months, no arrhythmias have been recorded from interrogation of her implantable cardioverter-defibrillator.

**References**


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