Successful treatment of tachycardia-induced cardiomyopathy secondary to dual atrioventricular nodal nonreentrant tachycardia using cryoablation

Harold Rivner, MD,[*] Chris Healy, MD,[†] Raul D. Mitrani, MD, FHRS[†]

From the [*]Department of Medicine, University of Miami Miller School of Medicine and Jackson Memorial Hospital, Miami, Florida, and [†]Cardiovascular Division, Department of Medicine, University of Miami Miller School of Medicine, and Jackson Memorial Hospital, Miami, Florida.

Introduction

Dual atrioventricular nodal nonreentrant tachycardia (DAVNNT) is a rare tachycardia in which dual AV nodal physiology allows for a single sinus complex to conduct down both the fast and slow AV nodal pathways, resulting in 2 ventricular depolarizations. A recent review of the literature surrounding DAVNNT identified only 68 published cases.[1] All ablations have been performed via radiofrequency ablation (RFA), with the exception of 1 cryoablation, which was unsuccessful and required further RFA.[1,2] Here, we present a case of DAVNNT successfully treated with cryoablation with elimination of tachycardia and improvement of tachycardia-induced cardiomyopathy. To our knowledge, this is the first successful example of cryoablation for the treatment of DAVNNT.

Case report

A 40-year-old man without any significant past medical history was found to have an abnormal electrocardiogram (ECG) during a pre-employment physical examination. The patient had noted a several-week history of progressive shortness of breath and fatigue. The ECG was interpreted as fascicular ventricular tachycardia (VT). Echocardiogram revealed dilated cardiomyopathy with an ejection fraction of 15%. The patient underwent electrophysiology study and ablation and was diagnosed with a parahisian VT. During that procedure, an area distal to the His bundle on the right ventricular septum was ablated using radiofrequency energy, resulting in complete right bundle branch block (RBBB). The tachycardia remained nearly incessant.

KEYWORDS DAVNNT; Dual atrioventricular nodal nonreentrant tachycardia; Dual atrioventricular nodal physiology; Cryoablation; Tachyarrhythmia; Cardiomyopathy (Heart Rhythm Case Reports 2016;0:1–6)

Address reprint requests and correspondence: Dr Raul D. Mitrani, Cardiovascular Division, Department of Medicine, University of Miami Miller School of Medicine and Jackson Memorial Hospital, 4062 UMH – East Building, Miami, FL33136. E-mail address: RMitrani@med.miami.edu.

The patient was then referred to our institution to be evaluated for focal cryoablation to target what was thought to be a parahisian VT and for defibrillator implant if his cardiomyopathy did not improve. After transfer, the patient's ECG, as shown in Figure 1A, was initially interpreted as sinus rhythm with fascicular bigeminy. Telemetry recordings were interpreted as ventricular (fascicular) tachycardia owing to the presence of more QRS complexes than P waves (Figure 2).

During repeat electrophysiology study, no ventricular tachyarrhythmias were inducible. However, the intracardiac electrograms confirmed sinus rhythm with conduction down both the fast and slow AV nodal pathways (Figure 3A). The coupling interval of the second His electrogram to the previous A remained constant when the patient was in sinus rhythm. Atrial overdrive pacing at a rate only slightly faster than the baseline sinus rate resulted in 1:1 AV conduction (down only the fast AV nodal pathway) and a slowing of the ventricular rate. There were extreme differences in the conduction velocities of the fast and slow AV nodal pathways: baseline AH interval of the fast pathway was 148 msec and baseline AH interval of the slow pathway was 535 msec. Demonstration of an AH jump by programmed stimulation was not possible, as none of the cycle lengths chosen for the drive train resulted in consistent conduction down only the fast pathway. There was no ventriculoatrial conduction. These findings were highly suggestive of DAVNNT as the mechanism of the tachycardia.

Inspection of the preprocedure ECG is consistent with conduction of each sinus complex down both the fast and slow AV nodal pathways, resulting in 2 QRS complexes for each P wave (Figure 1A). As the patient had a preexisting RBBB, both QRS complexes conduct with RBBB. However, the second QRS complex has an additional left posterior fascicular block (a long-short of the His-Purkinje bundle), resulting in further aberrancy. Similarly, careful evaluation of the telemetry recording shows evidence of P waves that conduct down the fast AV nodal pathway only, both the fast and slow AV nodal pathways, and the slow AV nodal

http://dx.doi.org/10.1016/j.hrcr.2016.09.005

KEY TEACHING POINTS

- Dual atrioventricular nodal nonreentrant tachycardia (DAVNNT) is a rare tachycardia in which dual AV nodal physiology allows for a sinus beat to conduct down both the slow and fast pathway, creating 2 consecutive double ventricular beats.
- DAVNTT is often confused with other tachyarrhythmias, leading to a delay in correct diagnosis. This delay can lead to ineffective treatments and to errant procedures with possible complications.
- DAVNNT can cause a tachycardia-induced cardiomyopathy, which can be at least partially reversed with ablation.

pathway only (Figure 2). Cryoablation was performed in the area of the slow pathway with elimination of dual AV nodal conduction (Figure 3B).

ECG after the procedure showed sinus rhythm with RBBB without any evidence of dual AV nodal conduction (Figure 1B). Serial echocardiograms showed improvement in the patient's left ventricular ejection fraction to 45%-50% at 1 year. Serial Holter monitoring showed sinus rhythm without any evidence of tachyarrhythmias. The patient remained asymptomatic, and defibrillator implantation was not necessary.

Discussion

DAVNNT is a rare arrhythmia in which dual AV nodal physiology allows a sinus complex to conduct down both the fast and slow AV nodal pathways, with 2 resultant ventricular depolarizations.[3,4] This is represented on the surface ECG as a single P wave followed by 2 QRS complexes. The electrophysiology study is characterized by a wide disparity of AH intervals on the slow and fast pathways, lack of retrograde ventriculoatrial conduction, and the lack of inducible atrioventricular nodal reentrant tachycardia. The diagnosis is not always straightforward, owing to intermittent dual AV nodal conduction, bundle branch block, or intraventricular conduction disturbances.[1] This diagnostic challenge is further compounded by the arrhythmia's rarity, often leading to misdiagnosis and inappropriate treatment. Hence, it is likely that DAVNNT is underdiagnosed. In a review of 68 cases of DAVNNT, 48 of them were initially misdiagnosed,[1] with 4 being mislabeled as ventricular tachycardia, similar to our case.[5–7] Delay in diagnosis can lead to ineffective treatment with antiarrhythmic medications and/or development of a tachycardia-induced cardiomyopathy. A systematic review found 8 prior cases of tachycardia-induced cardiomyopathy with ejection fraction $<45\%$ secondary to DAVNNT; all cases were reversed with appropriate ablation.[8] An additional case of DAVNNT-induced cardiomyopathy was also successfully reversed with RFA.[9] Furthermore, misdiagnosis can lead to errant procedures such as pulmonary vein isolation or implantable cardioverter-defibrillator placement.[8] Our case highlights this issue, as our patient was initially misdiagnosed with fascicular tachycardia and received an ablation procedure that not only failed to target his arrhythmia, but also resulted in an iatrogenic RBBB. In addition, our patient was being considered for implantable cardioverter-defibrillator implantation for his cardiomyopathy. Ultimately, this proved unnecessary after successful treatment of his arrhythmia and the resultant improvement in his ejection fraction.

As per the previously mentioned review, 64 out of 68 cases of DAVNNT have been treated with RFA. Three of the non-RFA cases were treated with antiarrhythmic medications, with variable degrees of success.[1] In the fourth case, cryoablation was attempted but proved unsuccessful, and RFA was performed during the same procedure.[2] In our case, previous RFA for a putative fascicular tachycardia had already caused RBBB. After the correct diagnosis had been made, a decision was made to target the slow pathway using cryoablation in order to minimize the risk of causing complete heart block, considering that prior RFA lesions were delivered to what was thought previously to be a parahisian fascicular tachycardia. To our knowledge, this represents the first successful cryoablation of DAVNNT.