Decremental Atriofascicular Accessory Pathway with Bidirectional Conduction: Delineation of Atrial and Ventricular Insertion by Radiofrequency Current Application

RAFAEL PEINADO, M.D., JOSE LUIS MERINO, M.D., LEONARDO RAMÍREZ, M.D., and IGNACIO ECHEVERRÍA, M.D.

From Unidad de Arritmias y Electrofisiología, Hospital General Universitario “La Paz,” Universidad Autónoma, Madrid, Spain

Decremental Atriofascicular Pathway with Bidirectional Conduction. A 17-year-old girl with a corrected complex congenital heart disease and recurrent episodes of supraventricular tachycardia was referred for catheter ablation. Electrophysiologic studies revealed the presence of an accessory pathway (AP) with bidirectional conduction and decremental properties. We demonstrated a course parallel to the node-His AV conduction system. Transient abolition of the bidirectional conduction through the AP was obtained by radiofrequency application to the ventricular insertion located in the distal right bundle branch and to the atrial insertion, located in the mid-anterior atrial septum. Radiofrequency application at the low anterior atrial septum, above the His bundle, successfully abolished AP conduction without affecting AV nodal conduction. Demonstration of the course and insertions of the AP, its bidirectional decremental conduction properties, and the association with a complex congenital heart disease are exceptional and interesting findings and raise the possibility of an accessory AV node with a parallel conduction pathway to the right bundle branch. (J Cardiovasc Electrophysiol, Vol. 12, pp. 489-492, April 2001)

supraventricular tachycardia, decremental accessory pathway, bidirectional conduction, preexcitation, radiofrequency ablation

Introduction

Most overt decremental accessory pathways (APs) are right free-wall atriofascicular APs with only anterograde conduction.¹ - ⁶ Although several cases of concealed atriofascicular or nodoventricular AP have been described,⁷,⁸ to the best of our knowledge an AP with decremental and bidirectional conduction has been reported only once before.⁹ We describe an exceptional case of an AP with decremental conduction and decremental properties in which we demonstrated a course parallel to that of the node-His-right bundle branch conduction system.

Case Report

A 17-year-old girl with narrow QRS tachycardias was referred to our unit for radiofrequency (RF) ablation. She was diagnosed shortly after birth with double-outlet right ventricle, d-transposition of the great arteries, interventricular septal defect, and pulmonary stenosis. Corrective surgery was performed before she was 8 years old. The patient presented with episodes of sudden-onset tachycardia resulting in palpitations, faintness, and anxiety when she was age 16 years. An ECG obtained in the emergency room showed a wide, regular QRS tachycardia, right bundle branch block morphology, right superior axis, cycle length 390 msec, and short RP interval (Fig. 1B). Tachycardia was converted to sinus rhythm by vagal maneuvers or adenosine infusion.

After obtaining informed consent, an electrophysiologic study was performed with the patient in the fasting state. Antiarrhythmic drugs were discontinued 4 days before the procedure. At baseline, sinus rhythm alternating with nodal rhythm, without preexcitation, with right bundle branch block QRS morphology, and right superior axis was observed (Fig. 1A). Baseline conduction intervals were AH 70 msec and HV 50 msec. Incremental ventricular pacing demonstrated retrograde VA conduction with decremental properties. Earliest atrial activation was recorded at the His-bundle area. Incremental atrial pacing showed transformation of the QRS morphology from an incomplete right bundle branch block to a left bundle branch block configuration, superior axis, as the pacing cycle length was reduced. This was accompanied by lengthening of the AH and AV intervals and shortening of the HV interval from 50 msec to negative (Fig. 2). The minimum atrial pacing cycle length maintaining 1:1 anterograde conduction through the AP was 270 msec. Conduction through the AP was blocked during rapid atrial pacing and resulted in higher degrees of right bundle branch block and HV interval lengthening to 75 msec. Right ventricular apex electrogram onset coincided with QRS onset during anterograde AP conduction (Fig. 2) but was delayed in the absence of anterograde conduction. Differential pacing from several points of the right atrium and coronary sinus showed the greatest preexcitation and shortest AV interval during preexcitation at the anterior right atrial septum. Orthodromic AV tachycardia with cycle length of 300 msec was reproducibly induced by atrial stimulation (Fig. 1C). The atrial retrograde activation pattern was similar to that observed during ventricular stimulation. Spontaneous development of complete right bundle branch block was observed during orthodromic tachycardia, resulting in a 100-msec prolongation of the VA interval and 90-msec prolongation of the tachycardia cycle length. Entrainment with fusion and tachycardia termination during right ventricular pacing were observed. In addition, the interval from the stimulus artifact to the right atrial electrogram during entrainment pacing from the right ventricular apex was shorter than dur-
ing entrainment pacing from basal or para-Hisian areas of the right ventricle. Infusion of adenosine 20 mg during atrial pacing resulted in disappearance of preexcitation. Infusion of adenosine during orthodromic tachycardia induced atrial fibrillation with different degrees of preexcitation and right bundle branch block.

After completing the electrophysiologic study, a 4-mm deflectable-tip electrode ablation catheter was introduced and activation mapping was performed during orthodromic tachycardia. The earliest atrial activation was located in the right aspect of the mid-anterior atrial septum. RF application at this site resulted in tachycardia termination and transient AP bidirectional conduction block on several occasions. However, bidirectional conduction through the AP recurred, and activation mapping of the ventricular insertion was again performed during sinus rhythm and atrial stimulation. The distal insertion site of the AP was located at the distal right bundle branch, where a sharp potential preceded the ventricular electrogram of preexcited and nonpreexcited QRS complexes (Fig. 3). A single RF application in this area induced automatic AP rhythm, abolished preexcitation, and transiently induced right bundle branch block. VA dissociation was observed after the RF pulse. However, bidirectional conduction through the AP resumed shortly afterward. In a second procedure, AP block and distal right bundle branch block were induced mechanically during ablation catheter placement in the right ventricle, without resumption before the end of the procedure 3 hours later. Finally, in a third procedure, activation mapping during orthodromic tachycardia showed early atrial electrograms in the low right atrial septum, near the His-bundle region. RF application in this area permanently abolished AP conduction without affecting AV conduction through the specific conduction system (Fig. 4). The patient is asymptomatic and had no recurrence of tachycardia or preexcitation at 16-month follow-up.

Discussion

It is accepted that, in most cases, overt decremental APs are right-sided APs without retrograde conduction. They cross the right AV ring and insert into the right bundle branch or right ventricle. Concealed atriofascicular or nodofascicular APs are rare. To the best of our knowledge, an AP with decremental conduction and bidirectional conduction has been reported only once before. Our patient could be another example of a decremental AP with bidirectional conduction and unusual location.

The transient and definitive abolition of AP conduction by RF application at three different sites demonstrates the course and the atrial and ventricular insertion of the AP in our patient. In most cases, the atrial insertion is reported in the posterior or posterolateral right AV ring. In our case,
the atrial insertion was located in the mid-anterior interatrial septum, where RF application transiently abolished the AV conduction. The AP coursed from the mid-anterior right atrial septum to the distal apical third of the right bundle branch through the area located immediately above the His bundle. To conclusively prove that the ventricular insertion was located at the right bundle branch, we should have demonstrated reversal of the activation sequence from the distal right bundle to the proximal right bundle to the His direction. Nevertheless, we believe that the distal insertion of the AP was linked to the distal portion of the right bundle branch based on two findings. (1) We recorded a distinct high-frequency potential, with and without preexcitation, at the level of the transient successful catheter ablation in the ventricular insertion. This potential probably represented activation of a distal segment of the right bundle branch. (2) More importantly, we recorded the development of AP block followed by right bundle branch block induced by mechanical trauma during catheter manipulation or RF application.\textsuperscript{2,4,5}

Electrophysiologic evidence supports the hypothesis that an atriofascicular pathway is an ectopic node–His-like AV conduction system,\textsuperscript{10} although a well-documented anatomic-electrophysiologic correlation establishing the substrate for atriofascicular pathways remains elusive. In our case, the course of the AP, the decremental properties of the anterograde conduction, the response to adenosine infusion,
and the development of an automatic AP rhythm during RF applications support this hypothesis. However, an AP with bidirectional conduction and decremental conduction properties in either direction, located in the low anterior septum and with a long course, could not be excluded. Accessory or duplicate AV nodes have been rarely observed. When they are, they are mainly in some congenital heart diseases.\textsuperscript{11,12} The course and insertions of the AP, its bidirectional decremental conduction properties, and the association with a complex congenital heart disease, in this case, raise the possibility of a subsidiary or duplicate AV node with a parallel conduction pathway to the right bundle branch. The relationship of this specific entity to the more conventional atriofascicular pathways is not clear and may be an entirely different anomaly with different embryologic origins. Unfortunately, we cannot firmly conclude either possibility with the data collected in our case. However, accessory nodes usually do not connect with the ventricular musculature or bundle branches, and their participation in AV reentrant tachycardia has not been reported until recently. Gollob et al.\textsuperscript{13} reported the first anatomically and electrophysiologically characterized accessory AV node that connected directly to the ventricle. They found an accessory anterior AV node located in the anterosuperior wall of the right atrium, 0.5 cm anterior to the AV septal junction, in a patient with AV reentry tachycardia. However, in contrast to our case, the node curved to form a direct communication between the right atrium and a wide area anterior to the ventricular summit.

Finally, our patient’s AP was associated with a complex congenital heart disease. Although decremental overt AP has been reported in patients with Ebstein anomaly of the tricuspid valve,\textsuperscript{2,3,5,6} an association between our type of AP and complex congenital heart disease has not been reported previously.

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\textbf{References}