ATRIAL FIBRILLATION

COMPLEX CASE STUDY

Recurrent Arrhythmias after Pulmonary Vein Isolation

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ABSTRACT. Evaluation of recurrent arrhythmias after catheter ablation of atrial fibrillation is challenging and requires specialized knowledge of the mechanisms of atrial fibrillation, the substrate for re-entrant arrhythmias after left atrial ablation, and an understanding of arrhythmias which can trigger or mimic atrial fibrillation. We present the case of a middle-aged female who continued to have symptoms of palpitations, dizziness, and shortness of breath after undergoing a catheter-based pulmonary vein antral isolation. Event monitoring and 12-lead electrocardiogram were reported as showing predominantly junctional rhythm along with repetitive bouts of a supraventricular tachycardia (SVT) and periods of atrial fibrillation. Electrophysiologic testing demonstrated the spontaneous occurrence of tachycardia matching the clinical arrhythmias during isoproterenol infusion. The tachycardia was diagnosed as repetitive non-re-entrant atrioventricular nodal tachycardia due to short periods of anterograde double firing and long episodes of persistent slow pathway conduction which mimicked a junctional rhythm. A simple slow pathway modification eliminated the rhythm abnormalities and associated symptoms.

KEYWORDS. atrial fibrillation, double atrioventricular node fire, junctional rhythm, non-re-entrant atrioventricular nodal tachycardia.

Case presentation

A 57-year-old female was referred to our clinic for continued palpitations, dizziness, and shortness of breath after undergoing prior pulmonary vein antral isolation for treatment of atrial fibrillation. She had been treated with dronedarone post ablation without control of her symptoms. Her electrocardiogram (ECG) from the clinic visit is shown in Figure 1 and was reported as demonstrating junctional rhythm with short runs of a supraventricular tachycardia (SVT). She underwent event monitoring, which was reported as demonstrating predominantly a junctional rhythm with short runs of a supraventricular tachycardia (SVT) and periods of atrial fibrillation (Figure 2). Given the failure of antiarrhythmic medications to control her arrhythmia symptoms, the patient agreed to undergo repeat electrophysiologic testing and possible ablation. Quadripolar catheters were placed in the right ventricle and His bundle recording positions, and a 20 pole catheter was placed along the lateral wall of the right atrium and into the coronary sinus. The baseline study demonstrated normal sinus rhythm, normal sinus node function, dual AV node physiology, and no VA conduction. The dual AV node physiology was demonstrated by finding an anterograde double fire with atrial extra-stimulation. The baseline AV block cycle length was 400 ms and all conduction was via the fast pathway prior to AV block. After infusion of isoproterenol at 2 μg/min, the rhythm abnormalities in Figure 3 emerged spontaneously. These arrhythmias mimicked the clinical arrhythmias seen on the 12-lead ECG and event monitor. The arrhythmia noted on the last three beats of the tracing shown in Figure 3 became nearly incessant. At the start of this tracing the patient was in sinus rhythm at a cycle length of 620 ms with 11 conduction to the ventricle with a short AV delay (first two QRS complexes). When the atrial cycle length shortened to 585 ms, the patient developed a brief period of a 12 A:V relationship consistent with AV nodal double
fires (atrial complexes 3–5, QRS complexes 3–8) and at the end of the tracing (last three QRS complexes) the patient was in a tachycardia of variable cycle length with a 1:1 AV relationship and a short VA time (<70 ms). The tachycardia appears to have been initiated with a long AH conduction time due to conduction down the slow pathway after the atrial electrogram labeled (#) at the termination of the anterograde double fires. The differential diagnosis of this final rhythm in Figure 3 includes junctional tachycardia, AV node re-entry, and either atrial or sinus tachycardia with a long anterograde conduction time. The very short VA time and VA dissociation with ventricular pacing excluded AV re-entry as a possibility. Although the AV and VA timing and the initiation with a long AV conduction time are consistent with atrioventricular nodal re-entry tachycardia (AVNRT), the fact that the activation sequence was the same as sinus rhythm with the earliest atrial activation in the high right atrium excludes AVNRT. The lack of change in the activation sequence from sinus rhythm also effectively excludes an atrial tachycardia. This leaves either junctional tachycardia with isorhythmic AV dissociation or sinus tachycardia with 1:1 conduction to the ventricle via the slow pathway as the remaining diagnostic possibilities. Close inspection of the later portion of the tracing (last three QRS complexes) demonstrates that the V-V cycle length was driven by the A-A cycle length, making sinus tachycardia with 1:1 AV conduction via the slow pathway the correct diagnosis. Figure 4 shows the response to atrial overdrive pacing at a cycle length of 400 ms. Fast pathway conduction is seen on the first and third atrial paced beats where anterograde double fires occur and conduction to the ventricle is exclusively via the fast pathway for the last four atrial paced beats seen in the tracing. This response to atrial overdrive pacing demonstrated that the mechanism for the periods of exclusive slow pathway conduction was due to fast pathway refractoriness as a result of retrograde penetration from the slow pathway and not due to rate dependent block of the fast pathway. Radiofrequency ablation of the slow pathway eliminated the patients arrhythmias and associated symptoms.

Discussion

With the advent of ablation as an option for treating atrial fibrillation the clinical electrophysiologist has had to become educated as to who is an appropriate candidate to select for a left atrial based ablation as well as how to diagnosis and manage continued arrhythmias post ablation. Numerous studies have demonstrated that in select patients referred for ablation of atrial fibrillation other forms of supraventricular arrhythmias may be found which may either trigger or mimic atrial fibrillation. In these cases, a simpler and less risky ablation can often be performed and thereby avoid a more extensive left atrial
Figure 2: Representative tracings from the cardiac event monitor. The event monitor shows sinus rhythm, possible junctional rhythm, and repetitive short runs of supraventricular tachycardia.
based ablation.\textsuperscript{4,5} In most reports of atrial fibrillation ablation the most common arrhythmia trigger for atrial fibrillation is AV node reentrant tachycardia which can be safely ablated in over 95\% of cases using a slow pathway approach. Ablation of this trigger will control the atrial fibrillation approximately 85\% of the time.\textsuperscript{3} A less common but increasingly recognized arrhythmia which can mimic atrial fibrillation is non-re-entrant AV node tachycardia. The majority of patients with this arrhythmia will present with repetitive or incessant double firing through the AV node which results in an irregular tachycardic rhythm. In a paper which analyzed 49 reported cases of this arrhythmia collected between 1950 and 2011, approximately one-third of these patients were misdiagnosed as having atrial fibrillation.\textsuperscript{6} Recent reports have highlighted that this misdiagnosis has led to inappropriate referrals for atrial fibrillation ablation.\textsuperscript{7,8}

The unique aspect of this case is that although the patient did manifest limited episodes of anterograde double firing mimicking atrial fibrillation, the predominant arrhythmia seen occurred because of long periods of exclusive slow pathway conduction which mimicked a junctional tachycardia. In the previously mentioned collection of reported cases, junctional rhythm or junctional tachycardia was not noted to be a misdiagnosis given to these patients.

In the vast majority of patients with dual AV nodal physiology, anterograde double firing does not occur either because of interactions between the AV nodal pathways or because of a mismatch between the refractory periods of the distal AV node common pathway or His bundle and the difference in conduction times between the fast and slow pathways. When dual AV nodal physiology is present, the fast and slow pathways can interact through two major mechanisms: electronic interaction and direct retrograde penetration of one pathway by the other. In order for a single anterograde double fire to occur the three minimum conditions that have to be met are: 1) dual AV nodal physiology has to be present; 2) the fast pathway impulse cannot penetrate the distal slow pathway and cause anterograde slow pathway block; and 3) the difference in conduction times between the fast and slow pathways has to be greater than the refractory period of the distal AV node common pathway and His bundle. In order for repetitive double firing to continue, another condition
which has to be met is that slow pathway conduction cannot penetrate the distal end of the fast pathway and cause anterograde fast pathway block. In this patient, once anterograde slow pathway conduction became established it extinguished fast pathway conduction, most likely as a result of direct retrograde penetration of the fast pathway. This phenomenon only occurred during a critical window of atrial rates which varied depending on the patient’s adrenergic state. At atrial rates below this critical window only fast pathway conduction was manifest, and anterograde double fires were not seen. This most likely was because at these slower rates the difference in conduction time between the fast and slow pathway was shorter than the refractory period of either the distal common pathway or the His bundle. At rates above the critical window, slow pathway block occurred which eliminated the retrograde penetration of the fast pathway, allowing fast pathway conduction to resume. This last phenomenon is clearly demonstrated in Figure 4. Pacing during this tracing was at a cycle length less than the block cycle length of the slow pathway. On the first and third paced beats, both fast and slow pathway conduction occurred, resulting in anterograde double firing. On the second and fourth paced beats, both fast and slow pathway block occurred; the slow pathway block was the result of pacing below the slow pathway block cycle length and the fast pathway block was because of retrograde penetration from slow pathway conduction from the preceding beat. Continued pacing at this cycle length resulted in persistent slow pathway block which eliminated its modulating effects on the fast pathway and allowed for consistent fast pathway conduction to occur. This case emphasizes the need to be aware of arrhythmias which can either trigger or mimic atrial fibrillation both during the initial evaluation and the post-ablation periods. This is especially true for patients with unusual arrhythmia patterns and those referred for evaluation after an apparently unsuccessful left atrial ablation. This case also highlights the need to perform basic electrophysiologic testing prior to proceeding with empiric left atrial based ablations in patients with paroxysmal atrial fibrillation. Although non-re-entrant AV nodal tachycardia is now increasingly well recognized, it is still a
relatively rare arrhythmia, which may explain why it is often misdiagnosed on surface ECG and telemetry tracings. Since few patients with this arrhythmia have been described in the literature, any additional information regarding clinical and electrophysiologic characteristics should be helpful to assist with appropriate identification and treatment of these patients. This case was unusual in that the patient’s major dysrhythmia mimicked a junctional tachycardia. We could find only two other reported cases in the literature where a patient manifested periods of an apparent junctional rhythm similar to our patient; however, the phenomenon of exclusive slow pathway conduction while pacing at a critical window of atrial rates has been reported in other patients with non-re-entrant AV node tachycardia, indicating that this may be an under-recognized manifestation of this clinical syndrome. Of the two cases, one was referred for pacemaker implant due to apparent diagnosis of intermittent impairment of AV conduction. As was performed in this patient, the literature supports a slow pathway ablation as the most effective therapy for this arrhythmia.

References