

Beyond the Classical Approach: Successful Sequential Ablation of Fast-Slow and Slow-Fast Atrioventricular Nodal Reentrant Tachycardia. An Illustrative Case

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ABSTRACT

Atrioventricular nodal reentrant tachycardia (AVNRT) is a frequent mechanism of regular narrow-QRS supraventricular tachycardia and is usually supported by dual atrioventricular nodal physiology. Typical slow-fast AVNRT is the most common pattern, whereas atypical fast-slow forms are less frequent and may require a more detailed electrophysiological evaluation. This case describes a 72-year-old woman with long-standing palpitations that became progressively more frequent and symptomatic despite antiarrhythmic treatment. A 12-lead electrocardiogram documented a narrow-QRS supraventricular tachycardia at 172 beats/min with RP interval longer than PR interval, and structural heart disease was excluded by transthoracic echocardiography. During the electrophysiological study, a spontaneous clinical tachycardia with a cycle length of 330 ms and ventriculoatrial interval of 198 ms was observed. Ventricular pacing maneuvers excluded an accessory pathway and supported the diagnosis of atypical fast-slow AVNRT. Radiofrequency ablation close to the coronary sinus ostium terminated the tachycardia. Subsequently, atrial stimulation induced a second tachycardia with a cycle length of 430 ms and simultaneous atrioventricular activation, compatible with typical slow-fast AVNRT. A second target located 6 mm from the initial ablation site was identified and treated with radiofrequency energy, resulting in noninducibility without atrioventricular conduction compromise. This case highlights the diagnostic and therapeutic relevance of recognizing coexisting AVNRT mechanisms and supports individualized sequential ablation when immediate recurrence or a different inducible circuit is demonstrated.

KEYWORDS: Atrioventricular nodal reentrant tachycardia; atypical AVNRT; slow pathway ablation; fast-slow tachycardia; electrophysiological study; radiofrequency ablation.

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I. INTRODUCTION

The atrioventricular node modulates impulse transmission from the atria to the ventricles, providing physiological delay that supports ventricular filling and limiting excessive ventricular activation during rapid atrial rhythms. The endocardial landmark most frequently used in electrophysiological procedures is Koch's triangle, bordered by the septal leaflet of the tricuspid valve, the tendon of

Todaro, and the ostium of the coronary sinus. This region contains the compact atrioventricular node, the His bundle area, and the anatomic extensions involved in AVNRT.

AVNRT is classically explained by dual atrioventricular nodal physiology. In typical slow-fast AVNRT, antegrade conduction usually proceeds through the slow pathway and retrograde conduction through the fast pathway. In atypical fast-slow or slow-slow AVNRT, the relative direction and

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activation sequence differ, producing diagnostic challenges, particularly when the RP interval is long or when atrial activation during tachycardia is eccentric. Contemporary literature emphasizes that the circuit is more complex than a single fixed pathway and may involve right inferior, left inferior, superior, or multiple slow-pathway extensions. Catheter ablation is considered a curative treatment for symptomatic AVNRT and is generally directed to slow pathway modification. Nevertheless, atypical AVNRT has a lower prevalence than typical AVNRT and may require individualized mapping, differential pacing maneuvers, and targeted ablation. In this context, the present case is relevant because two different AVNRT mechanisms were documented and eliminated during the same procedure.

II. CASE REPORT

A 72-year-old woman from San Luis Potosi, Mexico, was referred for electrophysiological evaluation because of recurrent symptomatic palpitations. Her medical history included systemic arterial hypertension and type 2 diabetes mellitus of 30 years of evolution, as well as hypothyroidism diagnosed 10 years earlier, all under pharmacological treatment. There were no relevant hereditary cardiovascular antecedents.

The patient reported sporadic palpitations since approximately 20 years of age. Initially, episodes were self-limited, without associated symptoms, lasted 1 to 5 minutes, and occurred every 3 to 4 years. During the previous 4 years, episodes became more frequent and intense, occurring once or twice per month and associated with chest pain and blurred vision. Pharmacological therapy with propafenone and metoprolol was initiated by an external cardiologist; however, the symptoms persisted and progressed, requiring pharmacological cardioversion in the emergency department of the referring hospital.

At the electrophysiology outpatient clinic, a 12-lead electrocardiogram documented a regular narrow-QRS supraventricular tachycardia without QRS alternans, with a ventricular rate of 172 beats/min and RP interval longer than PR interval. Baseline electrocardiogram in sinus rhythm showed right bundle branch block and PR interval of 210 ms. Transthoracic echocardiography ruled out structural heart disease. Based on these findings, atypical fast-slow AVNRT was suspected, and an elective electrophysiological study with electroanatomic mapping and catheter ablation was proposed. The patient accepted the procedure after informed consent.

III. ELECTROPHYSIOLOGICAL STUDY AND ABLATION

The procedure was performed on July 31, 2024, in the hemodynamics/electrophysiology laboratory under sedation. After asepsis and antisepsis of the right femoral region, two femoral venous punctures were obtained and two 6 Fr

introducers were placed. A decapolar catheter was advanced to the coronary sinus, and a deflectable quadripolar catheter was positioned at the His/right ventricular region.

During catheter placement, the clinical tachycardia appeared spontaneously. The tachycardia cycle length was 330 ms, with a ventriculoatrial interval of 198 ms. Differential ventricular pacing from the right ventricular apex at 350 ms showed a ventriculoatrial interval during pacing of 308 ms and a post-pacing interval of 618 ms. The VA pacing minus VA tachycardia difference was 110 ms, and the post-pacing interval minus tachycardia cycle length was 284 ms. These findings excluded an accessory pathway and confirmed atypical fast-slow AVNRT.

An HD Grid Advisor mapping catheter was advanced, and electroanatomic mapping of the right atrium was performed, marking the His region and the coronary sinus ostium. A TactiCath ablation catheter was then positioned near the coronary sinus ostium. Radiofrequency energy was delivered at 25 W, resulting in termination of the tachycardia during ablation. Application was continued until 2 minutes of total radiofrequency delivery were completed, without junctional rhythm.

After the first ablation, atrial stimulation from the coronary sinus at a cycle length of 600 ms was performed to confirm procedural success. A new tachycardia was induced with a cycle length of 430 ms and simultaneous atrial-ventricular activation, compatible with typical slow-fast AVNRT. A second slow-conduction zone was identified 6 mm from the site treated for the fast-slow tachycardia. Radiofrequency energy was then applied at 25-30 W, producing tachycardia termination and completing 2 minutes of total energy delivery.

Final programmed stimulation, including anterograde and retrograde stimulation and an anterograde curve from coronary sinus electrodes 7-8 with S1 600 ms and S2 400 ms, showed the persistence of a jump but without echo beats or induction of clinical tachycardia. The procedure was therefore considered successful.

IV. RESULTS

The electrophysiological study confirmed the presence of two inducible AVNRT mechanisms in the same patient: an atypical fast-slow AVNRT that corresponded to the clinical tachycardia and a subsequently inducible typical slow-fast AVNRT. Sequential radiofrequency ablation was successful in eliminating both tachycardias.

At the end of the procedure, no sustained tachycardia or echo beats were induced. Atrioventricular conduction was preserved, and there was no procedural evidence of high-grade atrioventricular block. The case therefore demonstrates acute procedural success through individualized mapping and sequential ablation of two neighboring but distinct nodal substrates.

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V. DISCUSSION

This case illustrates a clinically relevant variation of AVNRT in which the initial electrophysiological diagnosis of atypical fast-slow AVNRT did not fully explain the arrhythmogenic substrate. The immediate induction of a second tachycardia after apparently successful ablation shows that procedural endpoints in complex nodal reentry should not rely only on termination of the presenting tachycardia. Instead, repeated stimulation maneuvers remain necessary to test for alternative nodal circuits.

The finding is consistent with contemporary concepts indicating that AVNRT may involve multiple right- or left-sided inferior extensions of the atrioventricular node rather than a single slow pathway. Although typical AVNRT accounts for most cases, atypical forms are less common and may present long RP intervals, distinct atrial activation patterns, and a higher level of procedural complexity. The present case adds educational value because fast-slow and slow-fast AVNRT were documented sequentially during the same intervention.

From a diagnostic standpoint, ventricular entrainment and post-pacing measurements were essential to exclude a concealed accessory pathway. This distinction was clinically important because long-RP narrow-QRS tachycardias may mimic atrioventricular reentrant tachycardia or atrial tachycardia. In this case, the VA pacing minus VA tachycardia difference and the post-pacing interval supported AVNRT as the mechanism.

From a therapeutic standpoint, the ablation strategy was adapted to the findings obtained during the procedure. The first target, near the coronary sinus ostium, terminated the fast-slow tachycardia. However, the second inducible tachycardia required a separate application 6 mm from the first site. This small spatial distance is clinically relevant because overly extensive ablation near the compact atrioventricular node may increase the risk of conduction injury, whereas insufficient mapping may leave an additional circuit untreated.

The main limitation of this report is the absence of electrogram tracings and electroanatomic mapping images in the source file. For journal submission, representative figures showing the 12-lead tachycardia ECG, intracardiac intervals, activation map, ablation sites, and final noninducibility would strengthen the manuscript.

VI. CONCLUSION

The coexistence of atypical fast-slow and typical slow-fast AVNRT in the same patient is uncommon and represents a diagnostic and therapeutic challenge. This case supports the need for meticulous electrophysiological evaluation, differential pacing maneuvers, electroanatomic mapping, and reassessment after the first apparently successful ablation.

Sequential and targeted radiofrequency ablation of both nodal substrates achieved acute procedural success without compromising atrioventricular conduction. Therefore, in patients with atypical features, recurrence during the same procedure, or unusual induction patterns, multiple nodal pathways should be considered as potential coexisting arrhythmogenic mechanisms.

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DECLARATIONS

Ethics approval and consent to participate: The patient signed informed consent for the electrophysiological study and catheter ablation. Institutional ethical approval should be confirmed according to local requirements for case reports.

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Beyond the Classical Approach: Successful Sequential Ablation of Fast-Slow and Slow-Fast Atrioventricular Nodal Reentrant Tachycardia. An Illustrative Case

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