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Case reports

An unusual form of slow pathway ablation in atypical slow–slow atrioventricular nodal reentrant tachycardia: A case report

Forma peculiar de ablación de la vía lenta en una taquicardia por reentrada intranodal atípica variante lenta-lenta. A propósito de un caso

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ABSTRACT

Atrioventricular nodal reentrant tachycardia is the most common cause of paroxysmal supraventricular tachycardia syndrome and occurs predominantly in females. The current recommended classification divides this arrhythmia into two forms according to the tachycardia circuit: a typical form and a rare atypical form. We present the case of a 41-year-old woman with a history of recurrent palpitations. Diagnostic studies performed to document arrhythmias yielded negative results; therefore, an electrophysiological study was undertaken, confirming atypical slow-slow variant AVNRT. Subsequently, slow pathway ablation was performed, guided by the earliest atrial electrogram recordings obtained during tachycardia, an appropriate approach recommended for atypical variants of atrioventricular nodal reentrant tachycardia.

Keywords: Intranodal reentrant tachycardia; Atypical variant; Slow pathway; Ablation

RESUMEN

La taquicardia por reentrada intranodal representa la etiología más frecuente del síndrome de taquicardia paroxística supraventricular, con un predominio notable de su presentación en el sexo femenino. La clasificación actual recomendada subdivide esta arritmia en dos tipos según la forma del circuito de la taquicardia: una típica y una atípica poco frecuente. Se presenta el caso de una paciente del sexo femenino, de 41 años de edad con historia de palpitaciones recurrentes. Los resultados de los estudios realizados para documentar arritmias fueron negativos, por lo que se realizó un estudio electrofisiológico que confirmó una taquicardia por reentrada intranodal atípica variante lenta-lenta. Ante esto se efectuó la ablación de la vía lenta guiada por registros de electrogramas atriales más precoces en taquicardia, un abordaje idóneo recomendado para las variantes atípicas de la taquicardia por reentrada intranodal.

Palabras clave: Taquicardia por reentrada intranodal; Variante atípica; Vía lenta; Ablación

INTRODUCCIÓN

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common cause of paroxysmal supraventricular tachycardia. It exhibits a marked female predominance, with a female-to-male ratio of approximately 2:1. AVNRT typically occurs in the absence of structural heart disease.[1]

From a clinical perspective, AVNRT is characterized by episodes of rapid, regular tachycardia with abrupt onset and termination. Patients commonly report sudden palpitations and, in some cases, prominent neck pulsations resulting from simultaneous atrial and ventricular contraction, a finding classically referred to as the "frog sign." Although suggestive of AVNRT, this sign is not pathognomonic.[2]

The arrhythmogenic substrate of AVNRT is dual AV nodal physiology, consisting of functionally distinct pathways with different conduction velocities and refractory properties within the atrioventricular node.[3]

The most widely accepted classification currently divides AVNRT into two subtypes: a typical form, in which the reentrant circuit involves slow pathway antegrade conduction and fast pathway retrograde conduction; and an atypical form, which is further subdivided into fast-slow (antegrade conduction proceeds through the fast pathway and retrograde conduction through the slow pathway) and slow-slow variant.[4]

The atypical form accounts for only 6% of cases[5], and may coexist with the typical form in a subset of patients.[6]

The aim of this presentation is to describe a distinctive method for slow-pathway ablation in atypical AVNRT.

CASO REPORT

A 41-year-old female patient with a history of systemic high blood pressure treated with enalapril 10 mg twice daily presented with a history of palpitations of abrupt onset and termination for more than one year. Physical examination was unremarkable from a cardiovascular standpoint. She was evaluated in the outpatient arrhythmia clinic, where a 12-lead electrocardiogram and transthoracic echocardiogram were performed, both yielding normal findings. Twenty-four-hour Holter monitoring was performed on two separate occasions and showed no abnormalities. Given the persistence of recurrent and clinically significant symptoms despite the absence of electrocardiographic documentation, an invasive electrophysiological study was performed to elucidate the underlying arrhythmia mechanism.

Electrophysiological study:

After conscious sedation with propofol, vascular access was obtained using a modified Seldinger technique. Three venous sheaths were inserted: one 8-F sheath through the right femoral vein and two sheaths (6-F and 7-F) through the left femoral vein. Three Biotronik diagnostic catheters were subsequently positioned: a deflectable decapolar catheter in the coronary sinus, a quadripolar catheter at the His bundle region, and a bipolar catheter at the right ventricular apex.

A comprehensive electrophysiological protocol was performed according to the institutional approach for patients with undocumented symptomatic tachyarrhythmias. The most relevant findings were as follows:

- No evidence of antegrade or retrograde conduction over an accessory pathway was identified.
- Retrograde conduction during pacing from the right ventricular apex occurred via the AV node and was concentric.
- Incremental atrial pacing and programmed atrial stimulation with up to three extrastimuli delivered from the coronary sinus catheter failed to induce tachycardia and did not demonstrate evidence of antegrade dual AV nodal physiology.
- Programmed ventricular stimulation induced a regular tachycardia with a fixed, long ventriculoatrial interval (**Figures 1 and 2**).
- Electrophysiological findings were consistent with atypical AVNRT of the slow-slow subtype.
- Other arrhythmias were excluded using standard diagnostic pacing maneuvers.

Subsequently, via the right femoral venous access, a 4-mm tip deflectable Biotronik ablation catheter was advanced, and slow pathway mapping and ablation were performed.

Notably, electroanatomic mapping and ablation guided by slow pathway potentials recorded during sinus rhythm within the region of the triangle of Koch were unsuccessful. Although junctional rhythm was consistently observed during radiofrequency energy delivery, AVNRT remained readily inducible during post-ablation testing. Consequently, a strategy based on electroanatomical mapping during tachycardia was adopted, targeting the site of earliest atrial activation.

Using this approach, radiofrequency energy delivery (60°C, 60 W) resulted in immediate tachycardia termination, followed by the appearance of a stable junctional rhythm. Following ablation (total radiofrequency application

time: 120 seconds), the baseline induction protocol was repeated during isuprel infusion, and the tachycardia was no longer inducible.
The procedure was completed without complications. The final diagnosis was atypical slow-slow AVNRT successfully treated by slow pathway ablation. During 12 months of follow-up, the patient remained free of arrhythmia recurrence.

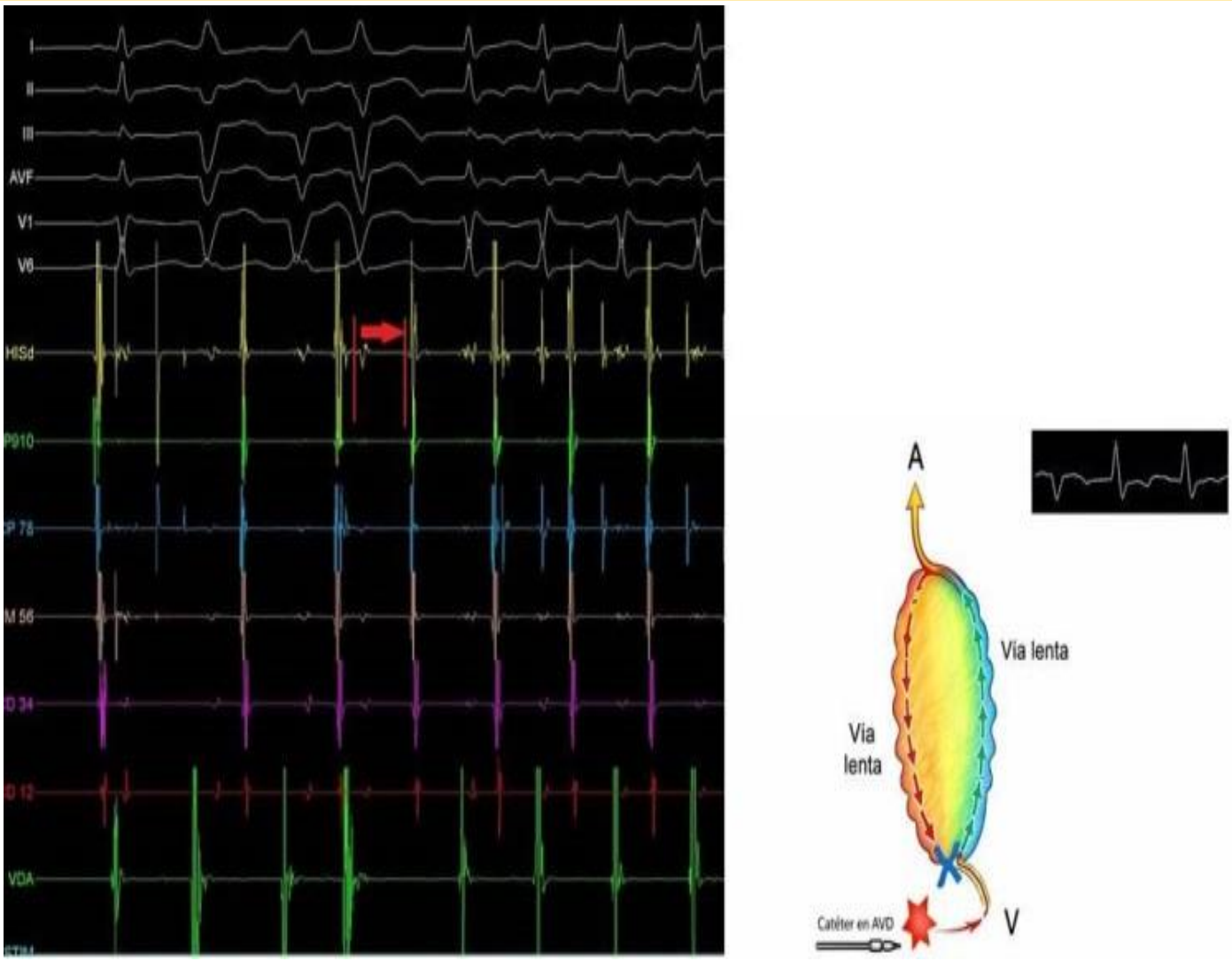

Figure 1- AVNRT **i**nduction
Legend: Left (from top to bottom): surface ECG recording, Hisd (highlighted in yellow, corresponding to electrograms recorded with a catheter positioned at the His bundle region), electrograms recorded from a decapolar catheter positioned in the coronary sinus from proximal to distal (poles 9-10 to 1-2), and RVA (electrograms recorded with a catheter positioned at the right ventricular apex). The figure shows three paced ventricular beats, and after a ventricular extrastimulus at 300 ms, a retrograde conduction pathway shift occurs with an atrial echo beat (red arrow), initiating atrioventricular nodal reentrant tachycardia (AVNRT). Right (*): substrate of slow-slow AVNRT. After a ventricular extrastimulus delivered from the right ventricular apex (red star), the wavefront propagates toward the atrioventricular junction, encounters block in one of the slow pathways (blue X), and ascends retrogradely through an alternative slow pathway (green arrows), resulting in atrial activation and subsequent antegrade conduction down the initially retrogradely blocked slow pathway (red arrows), thereby initiating slow-slow AVNRT. The surface ECG corresponding to the events described above is shown in the upper right corner of the image.
(*) Hand-drawn schematic edited using artificial intelligence (Copilot).

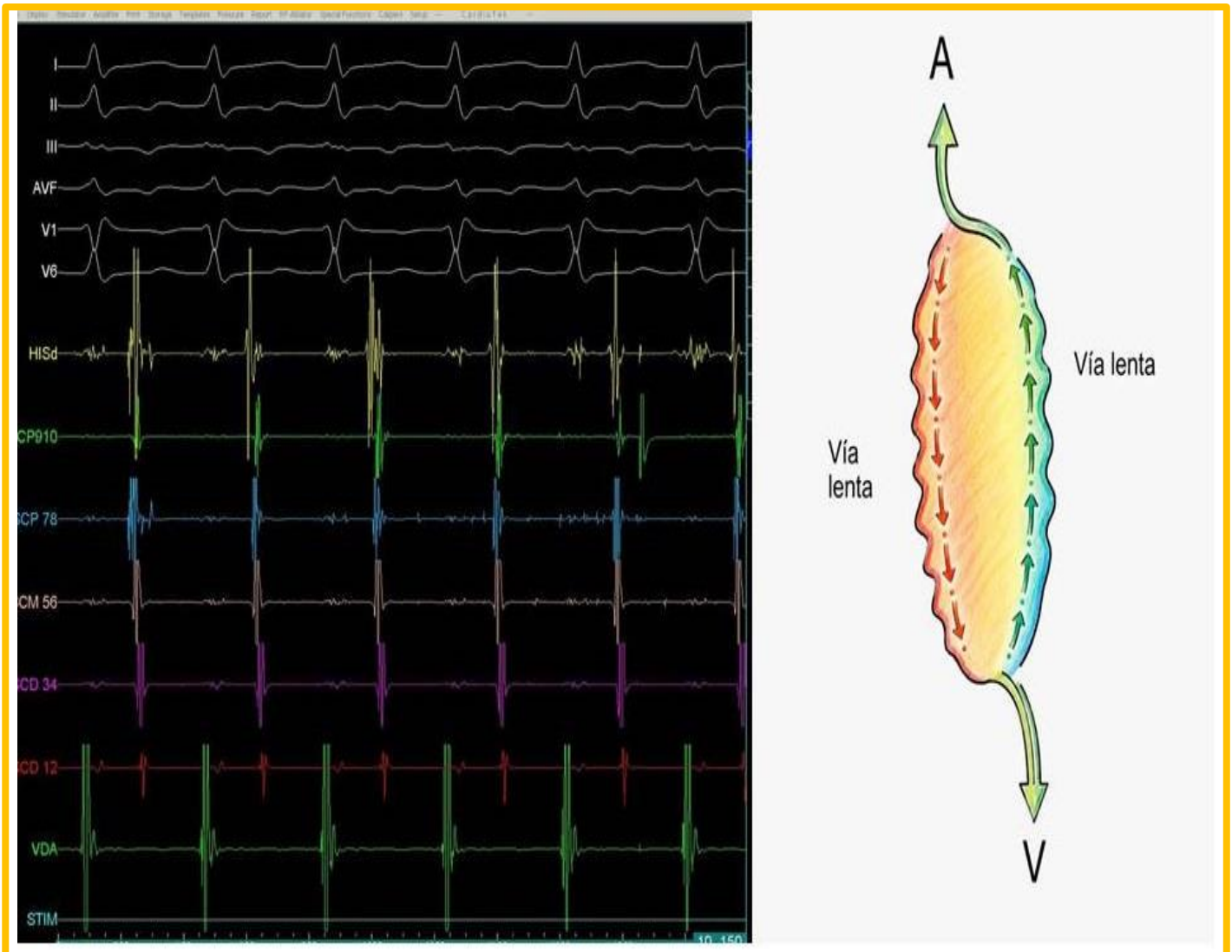

Figure 2- Sustained AVNRT
Legend: Left: Surface ECG (top) and intracardiac electrograms (His, coronary sinus, and right ventricle) during sustained AVNRT, with VA interval of 153 ms, HA interval of 176 ms, AH interval of 205 ms, AH/HA ratio ≥1, and tachycardia cycle length of 381 ms. Right (*): circuit of slow-slow AVNRT variant.
(*) Hand-drawn schematic edited using artificial intelligence (Copilot).

COMMENT:

Although atypical AVNRT is uncommon[5], its distinctive electrophysiological characteristics require a thorough understanding of the diagnostic and therapeutic considerations associated with this variant.
Many of these aspects are illustrated in the table below, which outlines the distinguishing features of this AVNRT.
Aspects of the electrocardiogram and the electrophysiological study are shown.
The findings described above (AH interval >200 ms, HA interval ≥70 ms, AH/HA ratio ≥1, and the earliest atrial activation site during tachycardia at the coronary sinus ostium), together with electrophysiological maneuvers that excluded other arrhythmias (which are beyond the scope of this report), support the diagnosis of the slow–slow variant of atypical AVNRT with certainty in this case.
As an interesting and uncommon observation, this case highlights the utility of electroanatomic mapping and slow pathway ablation during tachycardia.

Table: The principal characteristics of atypical AVNRT**.**

Distinctive Features of Atypical AVNRT	
Electrocardiographic findings	• Visible P waves, typically narrow, negative in the inferior leads and positive in lead V1. • RP interval longer than the PR interval in the fast-slow variant. RP interval equal to or shorter than the PR interval in the slow-slow variant.[7]
Electrophysiological findings	
Induction by atrial stimulation	• Antegrade dual AV nodal physiology is frequently absent or difficult to demonstrate. When induced by atrial pacing, it is usually associated with a slight prolongation of the AH interval.[8]
Induction by ventricular stimulation	• A ventricular extrastimulus may occasionally produce a 1:2 response (retrograde conduction over both the fast and slow pathways), followed by induction of atypical AVNRT. Fixed-cycle ventricular pacing may induce atypical AVNRT when sufficient retrograde nodal delay occurs, particularly during Wenckebach periodicity, with retrograde block in the fast pathway and conduction shift to the slow pathway.[8]
Atrial activation sequence during tachycardia	• The earliest atrial activation site is recorded at the CS ostium or 1-2 cm within the coronary sinus.[8]
AH interval	• Fast-slow variant: <200 ms. • Slow-slow variant: >200 ms.
HA interval	• ≥70 ms
AH/HA ratio	• Fast-slow variant: <1 • Slow-slow variant: ≥ 1[8]
Source: authors' own work based on the literature review	

This approach should not be overlooked in atypical AVNRT variants, considering that the site of earliest atrial activation during tachycardia should correspond to the retrograde exit of the circuit (the slow pathway), which constitutes the ablation target. In our opinion, mapping-guided ablation during tachycardia should be considered a first-line strategy in atypical AVNRT, or alternatively when conventional sinus rhythm-guided slow pathway ablation fails to eliminate inducibility. Furthermore, as proposed by several authors[9], a sequential approach may be advantageous: initial ablation guided by the earliest atrial activation during tachycardia, followed by conventional slow pathway modification during sinus rhythm. Theoretically, this strategy may eliminate conduction through both the right inferior nodal extension and the left inferior nodal extension,[8,9] thereby reducing the likelihood of recurrence across different AVNRT subtypes. Finally, successful management of atypical forms requires recognition that, to date, at least four anatomically distinct slow pathway variants have been described: the right inferior extension of the atrioventricular node (the most common), the left inferior nodal extension, the inferolateral left atrial slow pathway, and the anteroseptal slow pathway.[8]

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