Supraventricular arrhythmia with discordant electrocardiographic features: What is the arrhythmia mechanism?

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Abstract
Junctional and AV nodal reentrant tachycardia share common electrocardiographic features, but they differ in their management and outcomes after catheter ablation. This case concerns a 60-year-old female who presented with recurrent episodes of a relatively slow, regular supraventricular arrhythmia. Electrocardiographic features of the arrhythmia were discordant regarding its underlying mechanism. However, careful analysis of 12-lead electrocardiograms, with focus on the effect of spontaneous premature beats, pointed out the arrhythmia etiology. Electrophysiological study and pacing maneuvers defined the arrhythmic substrate that was successfully treated by catheter ablation.

1 | CASE PRESENTATION

A 60-year-old female with recurrent episodes of paroxysmal palpitations was referred to our center for evaluation. She had no history of ventricular preexcitation or structural heart disease. During symptomatic episodes, 12-lead ECG documented regular supraventricular arrhythmia at a relatively slow rate of 90–100 bpm (Figure 1). Pharmacological therapy, including beta-blockers and flecainide, had no clinical benefit. On admission, she was symptomatic for asthenia and neck pulsations, and her 12-lead ECG showed a regular narrow QRS rhythm (Figure 2). Based on these available data, what is the most likely mechanism of this arrhythmia?

2 | DISCUSSION

Figure 1 showed a regular supraventricular arrhythmia at a relatively slow rate of 95 bpm, with evidence of isorhythmic AV dissociation (the filled circles indicate sinus P waves) suggesting a focal junctional rhythm.

However, on admission, a fortunate 12-lead ECG (Figure 2), documented a regular supraventricular rhythm at a similar rate without clearly visible P waves while an interesting phenomenon was observed. A spontaneous premature ventricular beat (PVB) recorded on the chest leads (the asterisk, Figure 2), terminated the arrhythmia and restored sinus rhythm with a normal PR interval.

Isorhythmic AV dissociation during the ongoing arrhythmia (Figure 1) indicates an infra-atrial origin, and is against the diagnosis of atrial tachycardia or AV reentry via a concealed accessory pathway, with the rare exception of a nodo-ventricular/fascicular tract.

Furthermore, undetectable P waves (Figure 2) reflect a very short RP interval (P hidden within QRS) favoring AV nodal reentrant, or junctional, arrhythmia. The hypothesis of atrial rhythm at 95 bpm conducted with a very long PR interval (>500 ms), and P waves hidden within the preceding QRS is unlikely since the PR interval during sinus rhythm at similar rates was noted to be within normal limits and no evidence of first degree AV block.

Importantly, arrhythmia termination with a single premature beat (Figure 2, chest leads) favors reentry (e.g., AV nodal reentry) rather than automaticity or trigger activity (e.g., junctional rhythm) as the underlying mechanism (Gaztañaga, Marchlinski, & Betensky, 2012). The above-mentioned electrocardiographic features indicate the diagnosis of AV nodal reentry at unusually slow rates (<100 bpm) with phases of isorhythmic AV dissociation or competition with sinus rhythm.
**FIGURE 1** 12-lead ECG showing regular supraventricular arrhythmia with evidence of isorhythmic AV dissociation (gray circles indicate sinus P waves)

**FIGURE 2** 12-lead ECG showing regular supraventricular rhythm terminated by a single premature ventricular beat (the asterisk)
An electrophysiological study was performed, and typical AV nodal reentrant tachycardia with a medium rate of 100–120 bpm was reproducibly induced during Isoproterenol infusion. Pacing maneuvers confirmed the diagnosis and excluded other arrhythmia circuits. Notably, a premature His-refractory atrial extrastimulus terminated the arrhythmia (Figure 3), making junctional tachycardia unlikely as a mechanism (Padanilam et al., 2008). Standard slow pathway ablation provoked prolonged phases of junctional rhythm and rendered the arrhythmia not inducible anymore. The patient remained asymptomatic, off antiarrhythmic therapy, during the subsequent 6-months follow-up period.

In this case, careful analysis of electrocardiographic features during the arrhythmia, with a focus on its termination by a spontaneous PVB, pointed out the correct diagnosis of typical AV nodal reentry with an extremely slow rate.

CONFLICT OF INTERESTS

None.

FIGURE 3  Surface ECG and intracardiac recordings showing tachycardia termination by applying a single, His-refractory, atrial extrastimulus (S). CS: coronary sinus; d: distal; H: His potential; p: proximal; S: stimulus

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REFERENCES

