CASE REPORT

A narrow QRS tachycardia and cannon A waves: What is the mechanism?

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Regular narrow QRS tachycardia, particularly if well-tolerated, is usually considered a “benign” arrhythmia of a supraventricular origin. This case concerns an 82-year-old male with ischemic heart disease who presented with recurrent episodes of a narrow QRS tachycardia that was initially diagnosed and treated as atrial tachyarrhythmia. However, careful physical examination and ECG analysis established the correct diagnosis, and the patient was managed appropriately. Remarkably, the observation of irregular cannon A waves, and Lewis lead recording, confirmed atrioventricular dissociation during tachycardia and indicated its underlying mechanism.

KEYWORDS

atrioventricular dissociation, cannon A waves, Lewis lead, ventricular tachycardia

1 | CASE PRESENTATION

An 82-year-old man with ischemic heart disease was referred to our center for catheter ablation of recurrent episodes of atrial flutter/tachycardia. The patient had a history of remote myocardial infarction with multivessel coronary disease treated by surgical revascularization. During the successive follow-up, the patient maintained a preserved left ventricular function and remained asymptomatic until a few months ago when he started to present recurrent palpitations. Consequently, he was admitted to another center with electrocardiographic recordings of a narrow QRS tachycardia (NQT) that was diagnosed and treated as atrial flutter. Pharmacological cardioversion failed to interrupt the arrhythmia requiring electrical cardioversion, but early recurrence was observed. A coronary angiogram revealed stable coronaropathy with patent coronary bypass grafts. The patient was discharged on beta-blocker therapy, and catheter ablation was planned since he refused long-term amiodarone therapy.

On admission to our center, the patient was symptomatic for palpitations, but he denied chest pain or dyspnea. Physical examination revealed a blood pressure of 110/80 mmHg, heart rate of 170 bpm, and the aspect of the jugular veins is shown in the Movie S1. Twelve-lead ECG showed a regular, relatively NQT (QRS width = 110–120 ms) at 170 bpm (Figure 1). Twelve milligrams of intravenous adenosine was administered without any effect on tachycardia. Twelve-lead ECG recording during sinus rhythm (SR) is presented in Figure 2. Based on these available data, what is the most likely mechanism of this NQT?

2 | DISCUSSION

The patient presented with a well-tolerated, regular NQT suggesting a supraventricular origin. P waves were not clearly identifiable, but flutter-like waves or 2:1 atrioventricular (AV) conduction was initially suspected in the inferior leads. However, several clinical and electrocardiographic features argued against this hypothesis as the underlying mechanism. First of all, QRS complex during tachycardia differed than that during SR with a shorter duration (90 ms), higher amplitudes, slower onset, and earlier R/S transition in the precordial leads. Importantly, during tachycardia, QRS onset was slower than its offset with initial QRS slurring indicating early activation through the working myocardium rather than the Purkinje fibers. Supraventricular rhythms, with or without aberrancy, usually show rapid QRS onset due to fast activation through the His-Purkinje system. Moreover, the patient age, beta-blocker therapy, and the presence of 1st degree AV block during SR suggested reduced conduction capabilities over the AV node, making unlikely the hypothesis of a supraventricular tachycardia with high ventricular response (170–175 bpm). Notably, the unresponsiveness to intravenous adenosine, either termination or induction of a transient AV block, makes supraventricular tachycardia unlikely. These electrocardiographic features suggested a ventricular origin of the tachycardia and the patient clinical background with previous myocardial infarction provided an appropriate substrate for scar-related ventricular tachycardia (VT). Intravenous adenosine usually terminates idiopathic outflow VT (trigger activity), but it has no effect on postinfarction VT (macro-reentry).

FIGURE 1 Twelve-lead ECG of the clinical narrow QRS tachycardia (NQT)

FIGURE 2 Twelve-lead ECG during sinus rhythm (SR) showing 1st degree AV block and a premature ventricular complex

Physical examination of the jugular veins showed irregular cannon A waves (Movie S1) suggesting AV dissociation and indicating VT as the underlying mechanism. To facilitate P waves recognition during tachycardia, Lewis lead was obtained by recording lead I after applying the right arm electrode to the right of the sternum at the 2nd intercostal space and the left arm electrode to the 4th intercostal space (Bakker et al., 2009). This particular lead configuration

revealed distinct P waves showing AV dissociation during tachycardia and establishing VT diagnosis (Figure 3). The differential diagnosis of NQT with VA block/dissociation may include AV nodal tachycardia, junctional tachycardia, and orthodromic AV reentry using a nodo-fascicular/ventricular accessory pathway. However, these arrhythmias typically show a QRS pattern identical to that during SR, and they are adenosine-sensitive excepting automatic junctional

FIGURE 3 Lewis lead during narrow QRS tachycardia (NQT) showing AV dissociation (the arrows indicate P waves)

FIGURE 4 Atrial, ventricular, and shock recordings from the implanted defibrillator showing a ventricular tachycardia (VT) episode that was terminated successfully by anti-tachycardia pacing

tachycardia. Antidromic AV reentry over a nodoventricular accessory pathway may present similar electrocardiographic features with VA block. Nevertheless, the latter tachycardia is extremely rare at this age and with this substrate, and it is usually responsive to intravenous adenosine. Furthermore, there were no electrocardiographic signs of preexcitation (delta waves) during SR despite the prolonged PR interval.

The patient was transferred to the intensive care unit and underwent elective electrical cardioversion after ineffective intravenous amiodarone. Successively, ECG monitoring revealed 1st

degree AV block with episodic 2nd degree AV block (Mobitz 1). Reviewing the patient clinical files "Holter recordings" revealed one documented episode of wide QRS tachycardia at a similar heart rate (not shown). Based on the clinical scenery of sustained VT, probably with multiple forms, in the presence of structural heart disease, the patient underwent uneventful implantation of a dual-chamber defibrillator. During the successive follow-up, the patient had multiple episodes of VT which was successfully terminated by anti-tachycardia pacing (Figure 4). No atrial tachyarrhythmias were observed at the device interrogation. Catheter ablation of the VT

substrate is likely to be planned during the upcoming follow-up since the patient is unwilling to continue chronic amiodarone therapy, and he demonstrates good general conditions and a younger biological age.

This case underlines several important points. First, the initial diagnosis should not be considered as definitive, and reassessment of all available data is prudent. Second, an NQT makes VT diagnosis unlikely but not excluded since VT with a septal origin, or early activation of the specialized conduction system may present as NQT. Moreover, hemodynamic stability during tachycardia should not favor the diagnosis of a supraventricular origin.

Lewis lead recording and the irregular cannon A waves confirmed AV dissociation which is a hallmark for VT diagnosis. Jugular cannon A waves occur when atrial contraction coincides with a closed tricuspid valve. Cannon A waves are encountered during AV block, VT, and in AV nodal reentry (Tung & Healy, 2016). AV nodal reentrant tachycardia may show regular A waves due to simultaneous atrial and ventricular activation. In the presented case, there were *irregular* cannon A waves indicating AV dissociation. Finally, Lewis lead recording is a valuable and simple method to amplify P-wave amplitude facilitating its recognition during tachycardia (Bakker et al., 2009; Lewis, 1931).

DISCLOSURES/CONFLICT OF INTERESTS

None.

REFERENCES

- Bakker, A. L., Nijkerk, G., Groenemeijer, B. E., Waalewijn, R. A., Koomen, E. M., Braam, R. L., Wellens, H. J. (2009). The Lewis lead: Making recognition of P waves easy during wide QRS complex tachycardia. *Circulation*, 119, e592–e593.
- Lewis, T. (1931) Auricular fibrillation. In T. Lewis (Ed.), *Clinical electrocardiography* (5th ed., pp. 87–100). London, UK: Shaw and Sons.
- Tung, M. K., & Healy, S. (2016). Images in clinical medicine. Cannon A waves. *The New England Journal of Medicine*, 374, e4.

SUPPORTING INFORMATION

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