Incessant atrioventricular nodal reentrant tachycardia resulting in tachycardia-induced cardiomyopathy and catastrophic embolization of left ventricular thrombus

Roy Taoutel, MD,* Fares Sukhon, MD,* Steven Liskov, MD,† Bahij Kreidieh, MD,†‡ Sheetal Chandhok, MD§

*From the *Lankenau Medical Center, Wynnewood, Pennsylvania, †Lankenau Heart Institute, Wynnewood, Pennsylvania, ‡Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, and §Bryn Mawr Medical Specialists Association, Bryn Mawr, Pennsylvania.*

Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is a common paroxysmal supraventricular tachycardia (SVT) featuring repetitive salvos with spontaneous termination. Few reports exist demonstrating incessant AVNRT with chronically uncontrolled ventricular rates and tachycardia-induced cardiomyopathy (TIC).[1,2] Persistent cases of incessant AVNRT resulting in TIC and intracavitary thrombus are exceedingly rare.[2] We report a case of incessant slow/fast AVNRT associated with TIC and catastrophic embolization of left ventricular (LV) thrombus treated with acute radiofrequency slow pathway modification.

KEY TEACHING POINTS

- Incessant atrioventricular nodal reentrant tachycardia associated with tachycardia-induced cardiomyopathy is exceedingly rare.
- Tachycardia-induced cardiomyopathy may be associated with intracavitary thrombus and systemic embolization.
- Further research is needed to delineate the pathophysiology of tachycardia-induced cardiomyopathy and cardioembolism risk in supraventricular tachycardias other than atrial flutter and atrial fibrillation.

Case report

A 66-year-old male patient with no cardiovascular history was referred to our cardiology practice for palpitations lasting 2 weeks. The patient denied syncope, presyncope, chest pain, or dyspnea. He denied a history of cardiovascular disease and had never undergone cardiac imaging or functional testing. Vital signs included resting heart rate 133 beats per minute (bpm), blood pressure 138/98, and oxygen saturation 100% on room air. Physical examination revealed an obese male with decreased breath sounds at lung bases and bilateral lower extremity edema. Twelve-lead electrocardiogram revealed regular narrow complex tachycardia with cycle length 476 ms (126 bpm), incomplete right bundle branch block, and indistinct atrial activation (Figure 1). Bedside vagal maneuvers failed to perturb the arrhythmia. The patient was monitored appropriately and treated with intravenous (IV) adenosine in escalating doses of 6 mg, 9 mg, and 27 mg with brief sinus rhythm, multiple premature atrial depolarizations, and immediate reinitiation of SVT. He was subsequently treated with IV diltiazem, IV metoprolol, and 300 mg oral flecainide without durable sinus rhythm. The patient refused Emergency Department transfer, ultimately presenting 24 hours later with progressive heart failure symptoms. Upright chest radiograph revealed pulmonary vascular congestion. Brain natriuretic peptide concentration was normal at 46 pg/mL (normal <100 pg/mL), in keeping with elevated body mass index (36.9 kg/m^2), and cardiac troponin-I was normal at 0.03 ng/mL (normal <0.05 ng/mL). Transthoracic echocardiogram (TTE) revealed biventricular dilatation with LV end-diastolic diameter 7.1 cm (normal <5.8 cm), biatrial dilatation with left atrial volume indexed to body surface area 2.5 m^2 of 39 cm^2 (normal <35 cm^2) and severely reduced LV ejection fraction 20%, and no significant valvular pathology. The study also revealed a large, spherical, pedunculated thrombus measuring 2.4 × 1.6 cm adherent to the LV apex (Figure 2). Within 24 hours, the patient reported acute numbness and weakness

KEYWORDS Atrioventricular nodal reentrant tachycardia; Supraventricular tachycardia; Tachycardia-induced cardiomyopathy; Left ventricular thrombus; Acute limb ischemia; Arterial thromboembolism
(Heart Rhythm Case Reports 2022;■:1–4)

Funding Sources: The authors have no funding sources to disclose. Disclosures: Drs Toutel, Sukhon, Liskov, Kreidieh, and Chandhok have no relevant disclosures. **Address reprint requests and correspondence:** Dr Sheetal Chandhok, Bryn Mawr Medical Specialists Association, 825 Old Lancaster Rd, Suite 400, Bryn Mawr, PA, 19010. E-mail address: chandhokmd@yahoo.com.

https://doi.org/10.1016/j.hrcr.2022.11.015

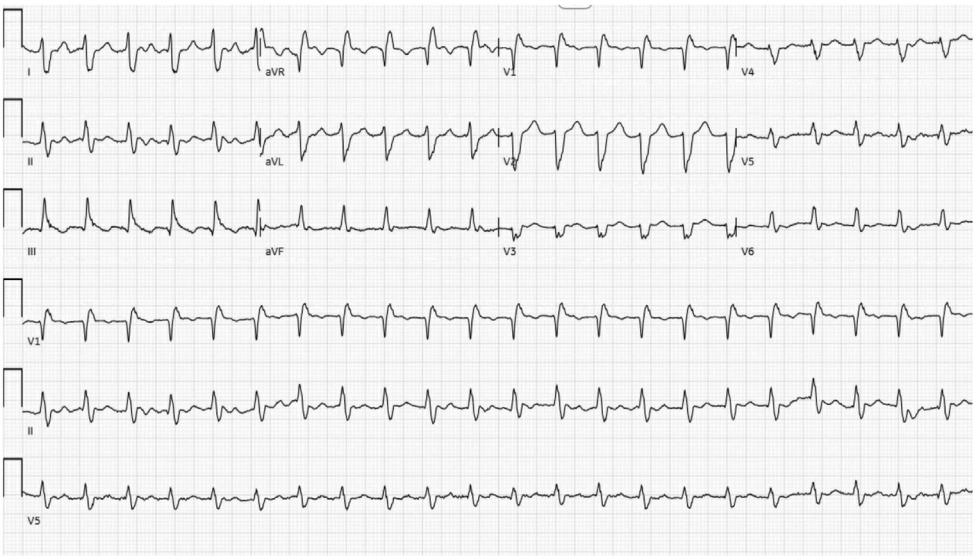

Figure 1 Narrow complex tachycardia at 476 ms (126 beats/min) with rate-related right bundle branch delay. Atrial activation is obscured by the QRS complex. The most likely diagnosis is typical slow-fast atrioventricular nodal reentrant tachycardia and sinus tachycardia or atrial tachycardia with prolonged atrioventricular conduction. Orthodromic atrioventricular tachycardia is less likely in the setting of closely coupled retrograde atrial activation.

of his right lower extremity concerning for acute limb ischemia, confirmed by computed tomography angiogram demonstrating acute thromboembolism to the right popliteal artery (Figure 2). Fortunately, the patient's symptoms resolved following initiation of therapeutic anticoagulation with IV heparin. Repeat TTE revealed complete resolution of previously visualized LV thrombus. The patient underwent coronary angiography for further characterization of his undifferentiated severe cardiomyopathy. The study revealed single-vessel borderline obstructive coronary artery disease confined to the mid-left anterior descending coronary artery, for which he received 1 drug-eluting stent. This stenosis was not considered the culprit for the patient's severe global systolic dysfunction.

The patient underwent electrophysiology study demonstrating baseline SVT with tachycardia cycle length 420 ms and septal ventriculoatrial time 16 ms (Figure 3). Right ventricular (RV) pacing accelerated the tachycardia to the paced cycle length and revealed concentric retrograde atrial activation with V-A-His response excluding atrial tachycardia, consistent with slow/fast AVNRT. RV para-Hisian pacing revealed a nodal response and His-refractory ventricular pacing did not advance the subsequent atrial signal, indicating the absence of an atrioventricular bypass tract. The His-ventricular interval and PR interval in sinus rhythm were 52 ms and 174 ms, respectively. Typical slow/fast AVNRT was diagnosed. Slow pathway modification was performed in sinus rhythm with 12 ablation lesions over 9 minutes and 50 seconds targeting 55°C in a power-titration method. Following a waiting period, AVNRT was no longer inducible with programmed atrial extrastimulation on 5 mcg/min isoproterenol. Slow pathway modification was considered acutely successful and there were no complications.

Repeat inpatient echocardiogram on heparin after ablation revealed improvement of LV ejection fraction to 35% with complete resolution of intracavitary thrombus. The patient was discharged on therapeutic warfarin according to international normalized ratio goal 2.0–3.0. Outpatient follow-up at 3 months revealed no clinical evidence of recurrent AVNRT, and TTE demonstrated normalization of LV systolic function. The patient reported minimal residual lower extremity weakness.

Discussion

Paroxysmal supraventricular tachycardias such as AVNRT feature abrupt episodes of tachycardia with spontaneous termination. Repetitive and persistent forms of AVNRT with chronically uncontrolled ventricular rates are rare, while incessant AVNRT resulting in TIC is case-reportable.[1,2] This case features progressive heart failure symptoms associated with onset of symptomatic incessant typical slow/fast AVNRT with ventricular rates approximately 130 bpm and resolution of symptoms and systolic dysfunction with treatment of his arrhythmia. The magnitude and noncoronary distribution of systolic dysfunction are out of proportion to the patient's single-vessel obstructive disease, and systolic function normalized following ablation, implicating uncontrolled tachycardia as the pathway for his cardiomyopathy.

Incessant AVNRT associated with TIC has been rarely reported in the literature.[2] In a single-center retrospective review over 8 years, Kawamura and colleagues[1] report 8 cases of incessant AVNRT with almost all patients demonstrating atypical fast/slow AVNRT and significantly lower LV ejection fractions (49% ± 12% vs 60% ± 8%, $P = .003$). A separate case report of slow/fast AVNRT associated with TIC featured LV intracavitary thrombus associated with systemic embolization.[2] The authors report incessant AVNRT associated with severe biventricular dysfunction, RV and LV intracavitary thrombi, and acute pulmonary

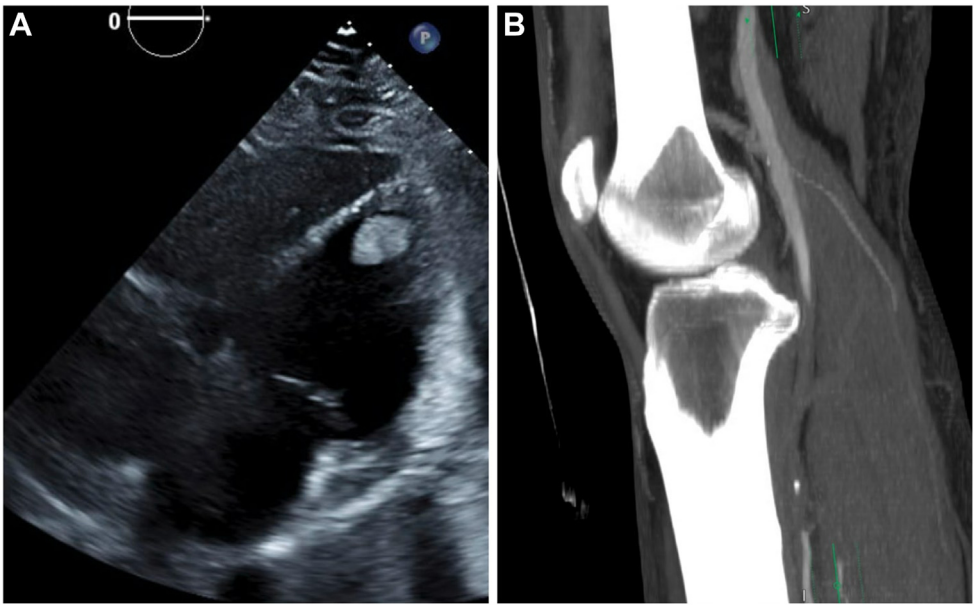

Figure 2 **A:** Transthoracic echocardiogram apical 4-chamber view demonstrating biventricular cavity dilation, biatrial enlargement, and a large, spherical, mobile thrombus measuring 2.4 × 1.6 cm seen adherent to the left ventricular apex. **B:** Multiplanar reconstruction of right lower extremity computed tomographic angiography demonstrated acute embolic occlusion of the distal right femoral artery.

thromboembolism. TIC itself is rarely associated with LV intracavitary thrombus.[2,3] In vivo models of LV ultrastructural changes in the setting of incessant SVT have demonstrated abnormal collagen fibril architecture in cases of significant systolic dysfunction.[4] The development of intracavitary thrombus represents an interaction between severe systolic heart failure and the relatively hypercoagulable state of chronic heart failure, and is associated with elevated risk of stroke and systemic arterial embolization.[5] Our case represents the first report of TIC associated with SVT complicated by intracavitary thrombus and systemic arterial embolization.

Tachycardia-induced cardiomyopathy is associated with SVT other than AVNRT. Incessant focal atrial tachycardia

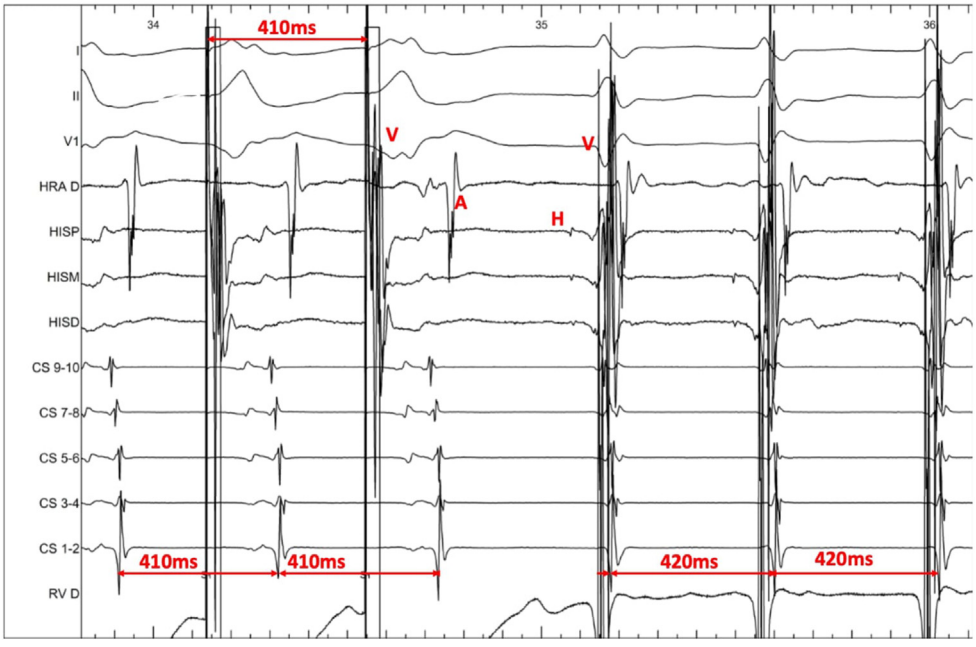

Figure 3 Electrophysiology study including surface electrodes, high-right atrial catheter (HRA D), His catheter (HISP, HISM, HISD), coronary sinus catheter (CS 1–10), and right ventricular catheter (RV D). Ventricular pacing at 410 ms demonstrates entrainment of the supraventricular tachycardia (SVT) with concentric retrograde atrial activation. The response to termination of overdrive ventricular pacing is V-A-His and resumption of SVT at tachycardia cycle length 420 ms, ruling out atrial tachycardia. Simultaneous atrial and ventricular activation seen on resumption of tachycardia further supports a diagnosis of typical slow-fast atrioventricular nodal reentrant tachycardia.

is associated with TIC in approximately 10% patients, most often at shorter tachycardia cycle lengths compared to paroxysmal variants, and is reversible with radiofrequency ablation.[6] Junctional reciprocating tachycardia is associated with TIC in pediatric and adult patients in multiple case series.[7,8] Atrial flutter, if associated with uncontrolled ventricular response, may be associated with TIC and recovery after ablation in up to 8% of patients.[9] Cardiomyopathy associated with atrial fibrillation may represent a complex interplay between uncontrolled ventricular rates and irregular ventricular rhythm.[10]

Left atrial myopathy and appendage thrombus associated with SVT is not well reported. Thromboembolism risk associated with incessant AVNRT may be extrapolated from evidence of stroke risk with typical cavotricuspid isthmus–dependent atrial flutter. While evidence is limited to observational studies and few systematic reviews, the risk of thromboembolism with atrial flutter is significant and provided an equal recommendation for anticoagulation as atrial fibrillation in the 2014 American College of Cardiology guidelines.[11,12] Incessant AVNRT is rare and there is no guidance for prophylactic anticoagulation in this population. However, as atrial tachycardia cycle lengths approach that of atrial flutter and uncontrolled ventricular rates provoke cardiomyopathy, consideration must be given to possible increased cardioembolic stroke risk. Future research is needed to demonstrate atrial stunning and stroke risk associated with incessant SVT other than atrial fibrillation and flutter.

Conclusion

Incessant AVNRT associated with uncontrolled ventricular rates and TIC is exceedingly rare. This case features catastrophic embolization of intracavitary thrombus associated with severe systolic dysfunction. There is a paucity of data regarding the impact of incessant SVT on global cardiac contractility and the risk of TIC. This case highlights the potential risks associated with incessant SVT with regard to cardioembolic stroke, the absence of guideline-supported management in prophylactic anticoagulation, and the role of early rhythm control in recovery of myocardial contractility.

References

1. Kawamura M, Scheinman MM, Vedantham V, Marcus GM, Tseng ZH, Badhwar N. Clinical and electrophysiological characteristics of incessant atrioventricular nodal re-entrant tachycardia. JACC Clin Electrophysiol 2016; 2:596–602.
2. Meti N, Mongeon FP, Guerra PG, O'Meara E, Khairy P. Incessant atrioventricular nodal reentrant tachycardia with tachycardia-induced cardiomyopathy, biventricular thrombosis, and pulmonary emboli. HeartRhythm Case Rep 2016; 2:142–145.
3. Nakasuka K, Ito S, Noda T, et al. Resolution of left ventricular thrombus secondary to tachycardia-induced heart failure with rivaroxaban. Case Rep Med 2014; 2014:814524.
4. Spinale FG, Tomita M, Zellner JL, Cook JC, Crawford FA, Zile MR. (1991). Collagen remodeling and changes in LV function during development and recovery from supraventricular tachycardia. Am J Physiol 1991;261(2 Pt 2):H308–H318.
5. Massussi M, Scotti A, Lip GYH, Proietti R. Left ventricular thrombosis: new perspectives on an old problem. Eur Heart J Cardiovasc Pharmacother 2021; 7:158–167.
6. Medi C, Kalman JM, Haqqani H, et al. Tachycardia-mediated cardiomyopathy secondary to focal atrial tachycardia: long-term outcome after catheter ablation. J Am Coll Cardiol 2009;53:1791–1797.
7. Dorostkar PC, Silka MJ, Morady F, et al. Clinical course of persistent junctional reciprocating tachycardia. J Am Coll Cardiol 1999;33:362–375.
8. Vaksmann G, D'Hoinne C, Lucet V, et al. Permanent junctional reciprocating tachycardia in children: a multicenter study on clinical profile and outcome. Heart 2006;92:101–104.
9. Brembilla-Perrot B, Ferreira JP, Manenti V, et al. Predictors and prognostic significance of tachycardiomyopathy: insights from a cohort of 1269 patients undergoing atrial flutter ablation. Eur J Heart Fail 2016;18:394–401.
10. Huizar JF, Ellenbogen KA, Tan AY, et al. Arrhythmia-induced cardiomyopathy: JACC state-of-the-art review. J Am Coll Cardiol 2019;73:2328–2344.
11. Vadmann H, Nielsen PB, Hjortshoj SP, et al. Atrial flutter and thromboembolic risk: a systematic review. Heart 2015;101:1446–1455.
12. January CT, Wann LS, Alpert JS, et al. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology / American Heart Association task force on practice guidelines and the Heart Rhythm Society. Circulation 2014;130:199–267.