

# Incessant atrioventricular nodal reentrant tachycardia with tachycardia-induced cardiomyopathy, biventricular thrombosis, and pulmonary emboli



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## Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common regular supraventricular arrhythmia. It typically presents with rapid and regular palpitations of abrupt onset in young adults, with a female preponderance. In patients without structural heart disease, AVNRT is largely considered to have a benign course and is rarely associated with disabling symptoms, such as syncope.<sup>1</sup> Herein, we report the case of an otherwise healthy young woman with a catastrophic presentation of AVNRT.

## Case report

A 26-year-old female, with no significant medical history, presented emergency department of the local hospital with a 2-week history of exertional dyspnea and fatigue. She denied palpitations, chest pain, orthopnea, paroxysmal nocturnal dyspnea, dizziness, and syncope. Review of systems and family history were unremarkable. Her 12-lead electrocardiogram at presentation, shown in [Figure 1](#), depicts a rapid and regular supraventricular tachycardia at a rate of 180 beats/min, with a short RP interval (ie, RP interval < PR interval). Intravenous adenosine boluses transiently interrupted tachycardia, albeit with the tachyarrhythmia recurring after a few sinus beats. Intravenous boluses of metoprolol before adenosine injection were likewise ineffective in preventing early recurrences. The patient was then transferred to our institution for further management of the incessant tachycardia, where an intravenous procainamide

infusion successfully terminated the arrhythmia and produced lasting sinus rhythm.

Upon workup, echocardiography revealed biventricular dysfunction with thrombus in the right ventricle. On cardiac magnetic resonance imaging, shown in [Figure 2A](#), biventricular thrombi were identified, with a left ventricular ejection fraction of 27% and a right ventricular ejection fraction of 29%. Computed tomography pulmonary angiography, displayed in [Figures 2B–2D](#), revealed bilateral pulmonary emboli with associated pulmonary infarction. Coronary angiography was normal. Hematological workup for an inherited hypercoagulable condition was negative, including antinuclear antibodies, lupus anticoagulants, antiphospholipid antibodies, fibrinogen, antithrombin III, protein C, protein S, factor V Leiden, homocysteine, and prothrombin G20210A.

Standard heart failure and anticoagulation therapies were initiated. An electrophysiology study was deferred in the context of ventricular thrombosis. Amiodarone was administered and complicated by amiodarone-induced hypothyroidism, which required thyroid replacement therapy. At 1 month of follow-up, repeat echocardiography showed resolution of the intracardiac thrombi with improvement in left ventricular function (ejection fraction 48%). An electrophysiology study was performed under conscious sedation. There was no evidence of an accessory pathway and no retrograde conduction at baseline. During isoproterenol infusion (1  $\mu\text{g}/\text{min}$ ), typical slow-fast AVNRT was reproducibly inducible with 2 atrial extrastimuli ([Figure 3](#)). The slow pathway was eliminated with cryoablation by means of a 6-mm electrode tip catheter (Freezor Xtra, Medtronic CryoCath LP, Montreal, Quebec, Canada). Amiodarone and  $\beta$ -blockers were discontinued postprocedure. Biventricular size and function had completely normalized at 6 months of follow-up, prompting gradual weaning of her angiotensin-converting enzyme inhibitor. She remains free of arrhythmia recurrence 9 months postablation.

**KEYWORDS** Atrioventricular nodal reentrant tachycardia; Tachycardia-induced cardiomyopathy; Intracardiac thrombus; Pulmonary emboli (*Heart Rhythm Case Reports* 2016;2:142–145)

Dr Khairy is supported by a Canada Research Chair in Electrophysiology and Adult Congenital Heart Disease. **Address reprint requests and correspondence:** Dr Paul Khairy, Electrophysiology Service, Montreal Heart Institute, 5000 Belanger St E, Montreal, Quebec, Canada, HIT 1C8. E-mail address: paul.khairy@umontreal.ca.

**KEY TEACHING POINTS**

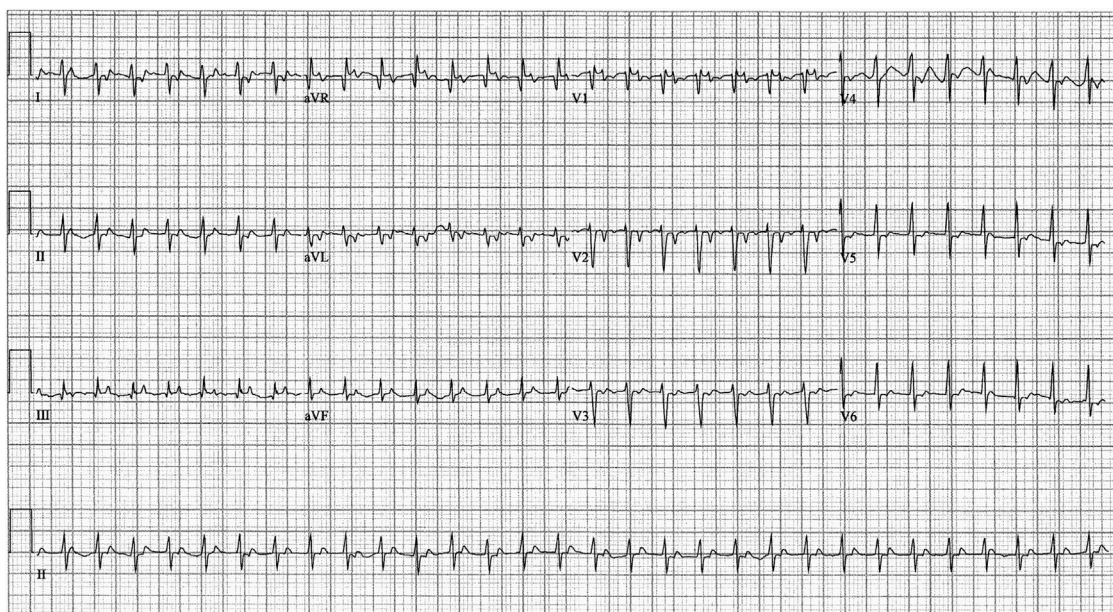
- Although atrioventricular nodal reentrant tachycardia is generally considered a benign arrhythmia in otherwise healthy individuals, nonspecific symptoms may lead to late presentation with untoward consequences.
- Long-standing incessant atrioventricular nodal reentrant tachycardia can provoke tachycardia-induced cardiomyopathy, characterized by potentially reversible ventricular dilation and dysfunction.
- This case report extends the spectrum of potential major complications associated with atrioventricular nodal reentrant tachycardia to include severe biventricular dysfunction, biventricular thrombosis, and pulmonary emboli with pulmonary infarction.

**Discussion**

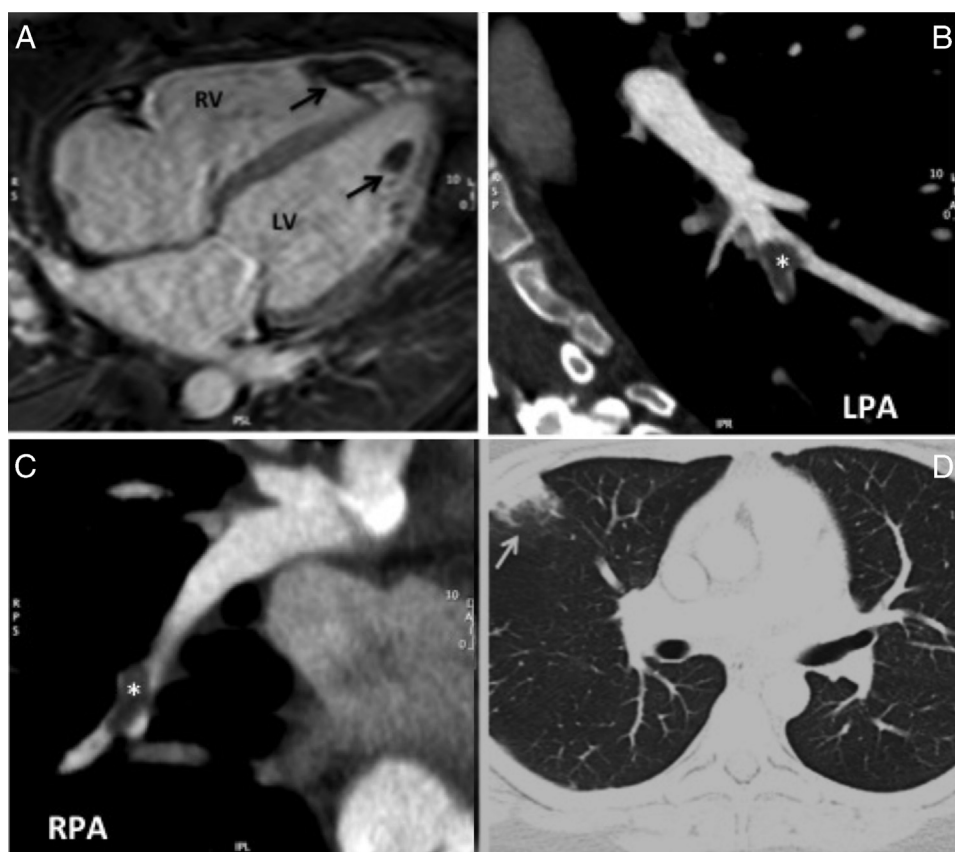
Tachycardia-induced cardiomyopathy is characterized by reversible ventricular dilation and systolic dysfunction due to sustained or frequent supraventricular or ventricular arrhythmias. It has primarily been described in the context of atrial fibrillation, atrial flutter, focal atrial tachyarrhythmias, permanent junctional reciprocating tachycardia, and frequent ventricular ectopy.<sup>2</sup> The literature on the

association between AVNRT and tachycardia-induced cardiomyopathy is limited to 1 case report of incessant atypical (fast-slow) AVNRT mimicking permanent junctional reciprocating tachycardia<sup>3</sup> and a patient with typical AVNRT, concomitant trifascicular block, intermittent complete heart block, and a single-chamber pacemaker.<sup>4</sup> To our knowledge, this is the first report of typical AVNRT provoking tachycardia-induced cardiomyopathy in an otherwise healthy individual and the first description of AVNRT associated with intracardiac thrombosis and pulmonary emboli.

Although supraventricular tachycardia typically presents with palpitations and shortness of breath, heart rates  $\geq 170$  beats/min are more likely to be associated with disabling symptoms such as syncope and dizziness.<sup>3-6</sup> Since AVNRT is almost always associated with palpitations, is often self-remitting, and rarely goes unrecognized for weeks on end, tachycardia-induced ventricular dilation and dysfunction appear to be exceedingly rare. In our patient, there were no atypical electrophysiological characteristics of the slow pathway to account for the unusual clinical scenario. Rather, the nondescript symptoms combined with an unremitting course likely resulted in a late presentation with dire consequences. Tachycardia-induced cardiomyopathy most commonly presents with heart failure symptoms,<sup>2</sup> which in our patient developed weeks, if not months, after the onset of tachycardia. Fortunately, as in other forms of tachycardia-induced cardiomyopathy, cardiac function recovered upon restoration of a stable sinus rhythm.<sup>7</sup> In general, once diagnosed with tachycardia-induced cardiomyopathy,



**Figure 1** Presenting electrocardiogram. The electrocardiogram shows a narrow complex tachycardia at 180 beats/min with RP interval < PR interval.



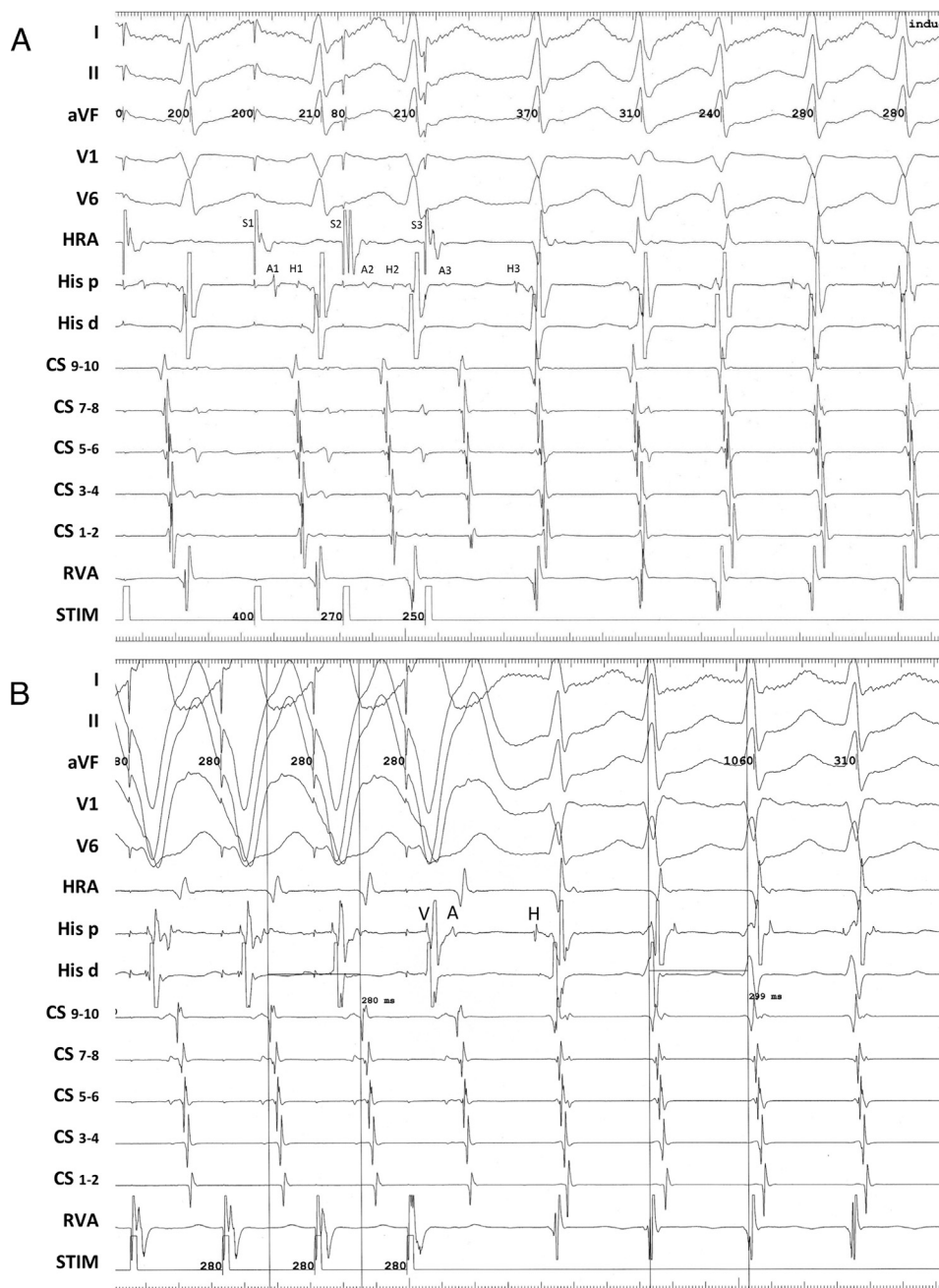
**Figure 2** Ventricular thrombi and pulmonary emboli. **A:** A cardiac magnetic resonance view in a 4-chamber orientation. Early gadolinium enhancement imaging reveals the presence of avascular masses in the right ventricle (RV) and left ventricle (LV), suggestive of thrombi. **B and C:** Computed tomographic images. In panels B and C, multiplanar reconstruction images of the left inferior lobe pulmonary artery (LPA) and interlobar right pulmonary artery (RPA) show sharply defined filling defects (asterisk), consistent with bilateral pulmonary emboli. **D:** A triangular opacity in the right upper lobe along the anterior chest wall (arrow), consistent with a pulmonary infarction.

recurrent arrhythmias may be associated with a rapid decline in ventricular function such that vigilant follow-up is required.

## Conclusion

Although the incidence of disabling symptoms associated with AVNRT is generally low in patients with structurally

normal hearts, unrecognized incessant episodes can present with tachycardia-induced cardiomyopathy. This case highlights the diagnostic challenges associated with nonspecific symptoms related to AVNRT and the potential devastating consequences. It further extends the gamut of reported major complications associated with ANVRT to include severe biventricular dysfunction, biventricular thrombi, and pulmonary emboli with consequent pulmonary infarction.



**Figure 3** Electrophysiology study. Shown are the recordings from surface electrocardiographic leads I, II, aVF, V<sub>1</sub>, and V<sub>6</sub> and intracardiac tracings from the high right atrium (HRA), His bundle proximal (His p) and distal (His d), coronary sinus (CS) proximal (9-10) to distal (1-2), and right ventricular apex (RVA). A stimulation (STIM) channel is also shown. **A:** Two atrial extrastimuli (S2 and S3) delivered from the HRA at coupling intervals of 270 and 250 ms result in prolongation of the A3-H3 interval, followed by sustained supraventricular tachycardia with a septal VA interval of 18 ms. **B:** The tachycardia (cycle length 299 ms) is entrained by ventricular pacing at 280 ms, with 1:1 ventriculoatrial conduction. Upon cessation of ventricular pacing, a V-A-H response is observed, consistent with atrioventricular nodal reentrant tachycardia.

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