

Malignant atrioventricular nodal reentry tachycardia resulting in cardiac arrest



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Introduction

Atrioventricular nodal reentry tachycardia (AVNRT) is the most common mechanism of supraventricular tachycardia (SVT) in adults and accounts for close to 60% of cases.¹ The prevalence of AVNRT appears to be higher among patients with implantable cardioverter-defibrillators (ICDs) when compared to the general population.² The development of AVNRT may cause symptomatic palpitations, inappropriate shocks, and, rarely, syncope. Though it is certainly important to recognize and treat SVT in ICD recipients, particularly to avoid inappropriate shocks, most SVTs are considered benign, especially when compared to more malignant arrhythmias like ventricular tachycardia (VT) and ventricular fibrillation (VF). We present a case of “malignant AVNRT” in a patient who presented with an out-of-hospital cardiac arrest.

Case report

A 34-year-old man with nonischemic dilated cardiomyopathy underwent single-chamber ICD implantation in January 2016 for primary prevention of sudden cardiac death. His left ventricular (LV) ejection fraction (EF) was 25% at the time of implant and defibrillation threshold testing was not performed. He had no history of prior ICD discharges or SVT. He suffered a witnessed cardiac arrest at home and bystander CPR was performed. When the rescue squad arrived, the initial pulseless rhythm was VF. He was successfully defibrillated externally and intubated in the field. His post-arrest rhythm was normal sinus with no evidence of pre-excitation. A review of stored electrograms (EGMs) revealed a regular tachyarrhythmia with cycle length (CL) of 281 ms as the initial rhythm, which degenerated into VF (Figure 1). The near-field and far-field EGMs of the regular tachyarrhythmia resembled the EGMs recorded during sinus rhythm

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KEY TEACHING POINTS

- The prevalence of atrioventricular nodal reentry tachycardia seems to be higher among patients with implantable cardioverter-defibrillators when compared to the general population.
- Supraventricular tachycardia is an important cause for inappropriate shocks, but in patients with severe cardiomyopathy it can take a malignant form, resulting in cardiac arrest.
- The finding that the electrograms of the initial tachycardia matched those recorded during normal sinus rhythm was the clue that the tachyarrhythmia was supraventricular in origin.

(Figure 2), consistent with a supraventricular origin. Six successive internal ICD discharges at 36–41 joules failed to terminate VF. The external shock by the rescue squad was successful. The patient had no neurologic sequelae from the arrest and was extubated but remained on inotropes to treat cardiogenic shock. An echocardiogram revealed severe global LV dysfunction with an EF of 15% and an LV thrombus. On telemetry, episodes of short RP-interval SVT were recorded at a similar CL as the clinical tachycardia, consistent with slow-fast AVNRT. An electrophysiology study (EPS) was performed, confirming the diagnosis of slow-fast AVNRT, and radiofrequency catheter ablation of the slow pathway was performed successfully (Figure 3). Owing to refractory cardiogenic shock, the patient underwent an expedited transplant work-up.

Discussion

Patients with nonischemic cardiomyopathy are at high risk of developing malignant arrhythmias, like VT and VF, and therefore benefit from the implantation of ICDs.³ Although SVT is an important cause of inappropriate shocks,⁴ it is usually not associated with the development of ventricular arrhythmias except in rare situations. For example, patients

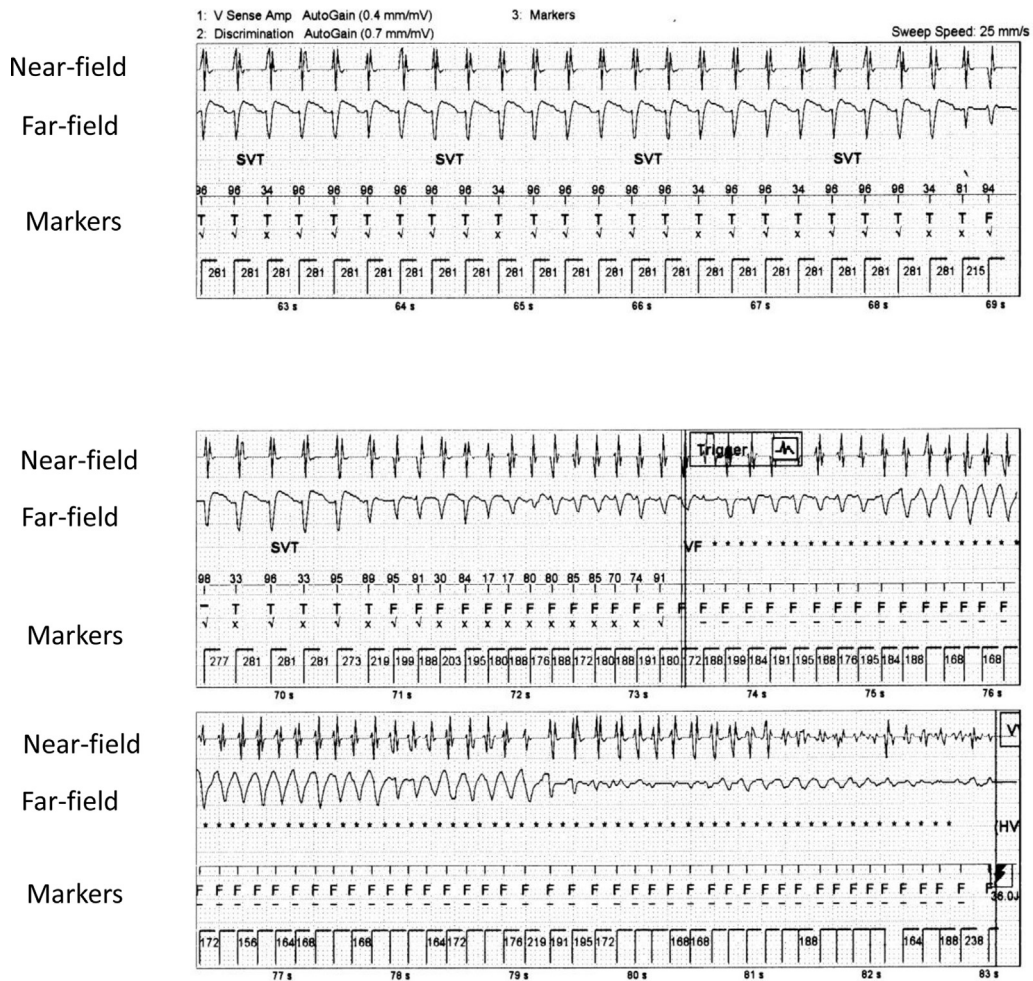


Figure 1 Stored electrograms of the initial tachyarrhythmia. Stored near-field and far-field implantable cardioverter-defibrillator (ICD) electrograms showing a regular stable tachyarrhythmia with cycle length = 281 ms degenerating into ventricular fibrillation (VF). The black dots are indicative of the ICD charging. SVT = supraventricular tachycardia.

with preexcitation syndromes are at risk for sudden cardiac death owing to degeneration of orthodromic SVT to atrial fibrillation, with subsequent rapid conduction to the ventricle.⁵

The link between SVT and VT/VF was described previously in a case report involving a patient admitted to the hospital after

suffering a cardiac arrest.⁶ While being monitored on telemetry, he developed SVT, which degenerated into VF. This patient was eventually found to have a chronic total occlusion of the right coronary artery. He underwent EPS and successful slow pathway ablation. He also underwent ICD placement and had no further events 9 months after ablation.

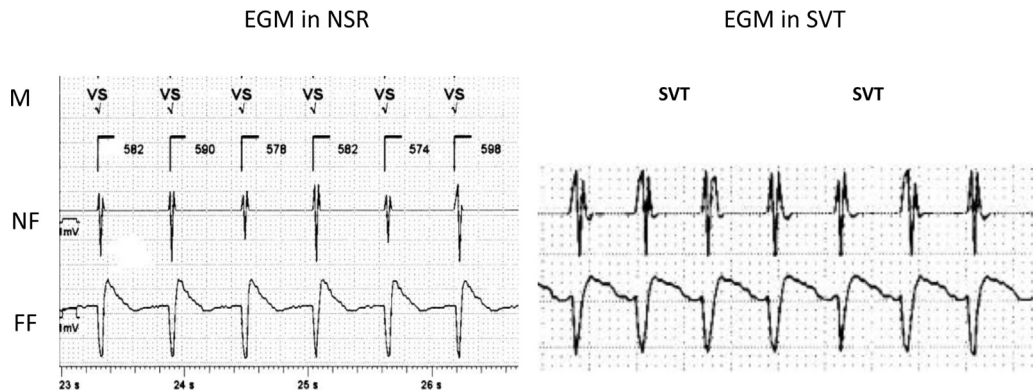


Figure 2 Comparison of the electrograms in normal sinus rhythm (NSR) and tachyarrhythmia. Near-field and far-field stored electrogram recordings of the initial tachyarrhythmia and sinus rhythm. EGM = electrogram; FF = far-field; M = markers; NF = near-field; SVT = supraventricular tachycardia; VS = ventricular sense.



Figure 3 Intracardiac recordings of induced typical atrioventricular nodal reentry tachycardia (AVNRT). Intracardiac electrograms during electrophysiology study of induced supraventricular tachycardia with a ventricle-to-atrium time of 59 ms, consistent with typical slow-fast AVNRT. A = atrial electrogram in His catheter recording; CS = coronary sinus; H = His bundle deflection; HIS d = HIS distal; HIS m = HIS mid; HIS p = HIS proximal; HRA = high right atrium; RVa d = right ventricular apex distal.

In 1 study of out-of-hospital cardiac arrest, 13 of 290 patients (4.5%) with VF arrest had documented or strong presumptive evidence of SVT degenerating to VF.⁷ Three of the 13 were noted to have typical slow-fast AVNRT. These patients had higher rates of associated cardiac disease and became severely hypotensive upon induction of AVNRT during EPS.

Although our case is not the first to document AVNRT causing VF, it is the first to capture AVNRT degenerating into VF on ICD stored EGMs. The finding that the EGMs of the initial tachyarrhythmia matched those recorded during sinus rhythm was the clue that the tachyarrhythmia was supraventricular in origin. In fact, morphology discriminators in the ICD correctly identified the initial tachyarrhythmia as SVT. The recurrent nature of the SVT, as recorded on telemetry, prompted us to proceed with EPS. Because of the patient's cardiogenic shock, the EPS and catheter ablation were performed under general anesthesia for patient safety. This is why, we believe, the CL of the induced AVNRT was much slower than the clinical AVNRT. Induction of AVNRT during EPS resulted in hypotension (systolic blood pressure 80–90 mm Hg), requiring administration of phenylephrine.

There are 2 possible mechanisms to explain why AVNRT led to VF. First, the short CL of the SVT was akin to “burst pacing” the ventricle at a fast rate. In some instances, this by

itself can lead to the development of VF. Second, fast SVT could have led to hypotension and, in the setting of severe LV dysfunction, resulted in the development of VF.

Conclusion

In patients with severe cardiomyopathy, SVT can take a malignant form, resulting in appropriate ICD shocks and cardiac arrest.

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