

Recurrent Supraventricular Tachycardia in a Patient with Heart Transplant

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Identification

Female patient, 58 years old.

Clinical history

The patient had a history of acute myocardial infarction in 1992; after the event, she developed heart failure with reduced ejection fraction (35%), New York Heart Association (NYHA) functional class II. She reported no other comorbidities and denied smoking and alcoholism. She was being treated with carvedilol 12.5 mg twice daily, enalapril 10 mg twice daily, simvastatin 20 mg, acetylsalicylic acid 100 mg, furosemide 40 mg, and spironolactone 25 mg once daily. In April 2015, she was hospitalized for an episode of monomorphic ventricular tachycardia (VT) with a heart rate (HR) of 170 bpm, blood pressure of 93/52 mmHg, and persistent chest pain, characterizing unstable VT. She underwent synchronized electrical cardioversion at a 200 J charge, with reversion to sinus rhythm. During the same hospitalization, she was started on amiodarone and underwent implantation of a single-chamber cardioverter-defibrillator.

During outpatient follow-up after cardioverter-defibrillator implantation, she presented recurrent episodes of slow VT, with HR of 100 to 110 bpm (Figure 1). The VT episodes were interrupted by antitachycardia cardioverter-defibrillator pacing. In April 2016, she was referred for VT ablation. During the procedure, an extensive area of fibrosis was identified in the apical region of the left ventricle. During ventricular pacing, the induction of 3 different VT morphologies was observed, with HR of 110 bpm, without hemodynamic instability. Radiofrequency ablation was performed based on entrainment and pace mapping. No attempt was made to induce VT at the end of the procedure.

After ablation, while using carvedilol 12.5 mg twice daily and amiodarone 200 mg once daily, the patient continued

to experience recurrent episodes of slow VT, interrupted by antitachycardia pacing. She progressed with frequent hospitalizations due to decompensated heart failure, NYHA functional class III to IV, and recurrent episodes of VT, and she was listed for heart transplantation.

She received heart transplantation in April 2017 from a 41-year-old female donor. During the procedure, she required mechanical mitral valve replacement due to severe acute mitral regurgitation due to valve tethering, possibly caused by ischemic injury. The cardioverter-defibrillator leads were removed during transplantation, and the generator was maintained. During the postoperative period, she presented with primary graft dysfunction, transient atrioventricular block, sepsis due to *Klebsiella pneumoniae*, and cardiac tamponade due to pericardial effusion requiring drainage and pericardiectomy. She required implantation of new atrial and ventricular pacemaker leads due to chronotropic incompetence. The cardioverter-defibrillator generator was preserved, functioning exclusively as a pacemaker. The patient was discharged after 25 days of hospitalization. The discharge echocardiogram showed preserved ejection fraction (66%) and good functioning of the mechanical mitral valve prosthesis, with no relevant abnormalities.

The patient continued outpatient follow-up with favorable progress. During this period, she was monitored by the electrophysiology team, as the cardioverter-defibrillator remained implanted, functioning as a pacemaker, with the arrhythmia monitoring function deactivated. In October 2024, 7 years after the transplant, during elective hospitalization for orthopedic surgery due to a rotator cuff tear, she presented an episode of tachycardia with a wide QRS complex and a heart rate of 150 bpm (Figure 2). The QRS morphology was similar to her electrocardiogram in sinus rhythm (Figure 3). Analyzing the QRS morphology according to the Brugada criteria, the patient's QRS would be compatible with VT based on the morphological criteria for right bundle branch block (due to the qR pattern in V1). During the arrhythmia episode, she reported only anxiety, without palpitations, angina, dyspnea, or other complaints. Given the patient's native QRS morphology and the suspected diagnosis of paroxysmal supraventricular tachycardia, 6 mg of adenosine was administered intravenously, with immediate reversal of the tachycardia. The carvedilol dose was increased from 12.5 mg to 25 mg twice daily after the episode.

In March 2025, the patient was hospitalized for dengue fever, in the context of immunosuppression,

Keywords

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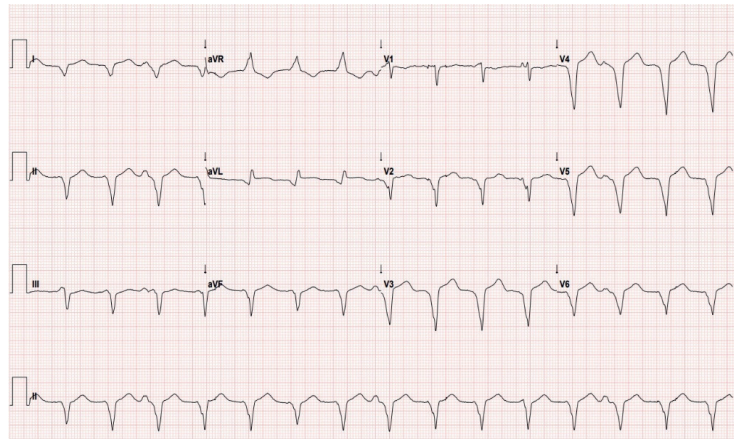


Figure 1 – Electrocardiogram during outpatient follow-up after implantation of a cardioverter-defibrillator, showing an episode of slow ventricular tachycardia.

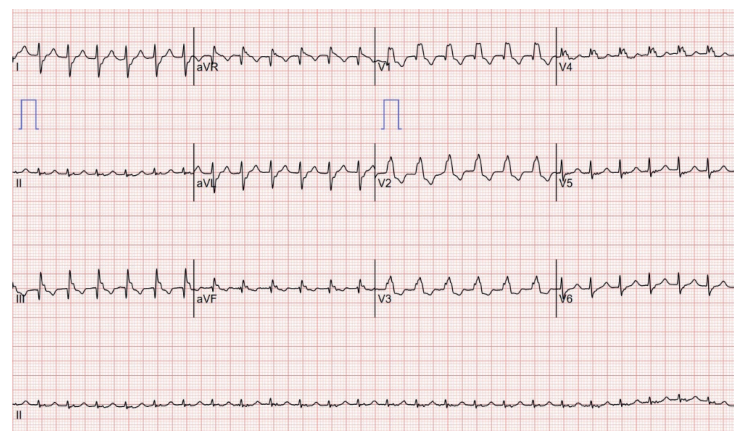


Figure 2 – Electrocardiogram performed during elective hospitalization in October 2024, showing an episode of tachycardia with wide QRS complex.

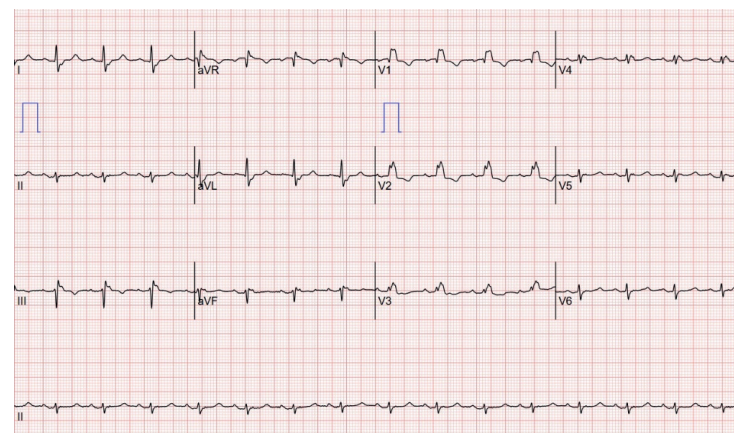


Figure 3 – Electrocardiogram in sinus rhythm.

Case Report

warfarin use with prolonged prothrombin time, and associated acute kidney injury. During hospitalization, two new oligosymptomatic episodes of supraventricular tachycardia were recorded, which were reversed with 6 mg adenosine. The patient did not develop severe dengue or any major associated complications. When questioned about previous arrhythmia symptoms in recent years, the patient reported occasional anxiety similar to the tachycardia episodes she experienced in the hospital, but never more intense symptoms that prompted her to seek hospital care. Given this account, coupled with the fact that cardioverter-defibrillator event monitoring remained disabled in the years following transplantation, it was not possible to determine the exact timing of her first episode of paroxysmal supraventricular tachycardia. According to the team's assessment, the patient had already been experiencing rather vague symptoms of arrhythmia (anxiety, nonspecific discomfort), delaying diagnosis, which was eventually made in a timely manner during hospitalization for another reason.

After recovering from dengue, she was referred for ablation of supraventricular tachycardia. During the procedure, the presence of dual nodal physiology was identified, with easy and reproducible induction of typical atrioventricular nodal reentrant tachycardia with a cycle length of 414 ms. Slow-pathway ablation was performed with a 4 mm catheter. After ablation, the patient did not have any new episodes of supraventricular tachycardia.

Atrioventricular nodal reentrant tachycardia in heart transplant recipients

Supraventricular tachycardias are relatively common after heart transplantation. Atrial flutter and atrial fibrillation are the main arrhythmias, occurring in 10% to 25% of cases, whereas atrioventricular nodal reentrant tachycardia is relatively rare, occurring in under 0.5% of patients who undergo transplantation.^{1,2}

A cohort of 729 patients undergoing heart transplantation identified 47 patients with persistent or paroxysmal supraventricular tachycardia,³ 24 of whom underwent electrophysiological study. Among the patients who underwent electrophysiological study, 14 were diagnosed with atrial flutter, 7 with macro-reentrant atrial tachycardia, 1 with atrioventricular nodal reentrant tachycardia, and 2 with accessory pathway atrioventricular reentrant tachycardia.³ In that cohort, the patients with accessory pathway-mediated atrioventricular reentrant tachycardia required ablation within the first month after transplantation, but no mention was made of the timing of the diagnosis of the only case of atrioventricular nodal reentrant tachycardia.³ In a few cases of atrioventricular nodal reentrant tachycardia after heart transplantation reported in the literature, the identification of the arrhythmia and radiofrequency ablation occurred within the first 6 months after transplantation.^{4,5}

The proposed mechanisms for the development of atrioventricular nodal reentrant tachycardia in patients undergoing transplantation include the preservation of dual nodal physiology in the donor heart and alterations in autonomic tone, secondary to cardiac denervation and surgical stress.²

The use of adenosine in heart transplant recipients has been a subject of discussion. These patients may have an exaggerated bradycardic response due to the effect of the drug, which was contraindicated in this context for many years.² More recent studies have demonstrated the safety of the medication at doses up to 12 mg.² Administration of adenosine in increasing doses is currently recommended for the reversal of this arrhythmia, with the initial dose ranging between 1.5 and 3 mg.^{2,6}

The treatment for preventing recurrence of atrioventricular nodal reentrant tachycardia in patients who have undergone transplantation follows the same principles applied to the general population; slow-pathway catheter ablation is the therapy of choice in most cases, with a high success rate and low incidence of complications.⁷ Some case series have demonstrated the safety of performing this procedure with long-term success.^{3,8} Moreover, according to the Third Brazilian Guideline for Heart Transplantation, a history of supraventricular tachycardia does not constitute a contraindication for procurement of a donor heart for transplantation.⁹

Author Contributions

Conception and design of the research: Bernardon LG, Pimentel M; Acquisition of data: Bernardon LG, Schreiber Junior A; Analysis and interpretation of the data and Writing of the manuscript: Bernardon LG; Critical revision of the manuscript for intellectual content: Bernardon LG, Pimentel M, Magalhães AP, Faganello L, Zimmerman L.

Potential conflict of interest

No potential conflict of interest relevant to this article was reported.

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Study association

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Ethics approval and consent to participate

All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

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