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Refractory supraventricular tachycardia from a coronary sinus diverticulum leading to cardiogenic shock: a challenging ablation case

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Abstract

A 58-year-old male with a past medical history of radiofrequency ablation of a concealed posteroseptal accessory pathway presented to the emergency department with palpitations and acute heart failure. The electrocardiogram showed narrow QRS tachycardia with a heart rate of 170 bpm. Despite boluses of adenosine and amiodarone perfusion, the patient experienced recurrent supraventricular tachycardia (SVT), leading to cardiogenic shock. Extracorporeal membrane oxygenation was initiated for hemodynamic support. Electrophysiologic study identified an accessory pathway in a coronary sinus diverticulum. Successful radiofrequency ablation was performed, resulting in clinical and left ventricular ejection fraction improvement. This case highlights the challenges in managing incessant SVT with cardiogenic shock and complex ablation procedures.

Key words: incessant supraventricular tachycardia, cardiogenic shock, extracorporeal membrane oxygenation, coronary sinus diverticulum.

Introduction

Arrhythmia-induced cardiomyopathy is a common cause of left ventricular (LV) systolic dysfunction, including tachycardia-induced cardiomyopathy (TIC) [1]. Supraventricular arrhythmias, particularly atrial fibrillation/flutter with rapid ventricular response, are the most frequent contributors and can be reversible with appropriate tachycardia management. Less common arrhythmias, like atrioventricular reentrant tachycardia, may also cause TIC [2,3].

Case Report

A 58-year-old male presented to the emergency department with worsening sustained palpitations and shortness of breath over the past few weeks. A recent Holter monitor showed supraventricular tachycardia (SVT) with a heart rate of 190 bpm lasting 13 hours. His medical history included SVT, for which he underwent an electrophysiological study (EPS) 12 years earlier with successful radiofrequency ablation of a concealed posteroseptal accessory pathway. On physical examination, he had regular tachycardia (170 bpm), a blood pressure of 130/80 mmHg and signs of acute heart failure. The electrocardiogram (ECG) showed narrow QRS tachycardia at 170 bpm and a long RP interval (Figure 1). After administering adenosine (6+12+18 mg), the tachycardia was terminated with a short period of auriculo-ventricular block followed by the restoration of sinus rhythm (SR) (Figure 2). The ECG in SR didn't show pre-excitation. However, the patient experienced multiple recurrences of SVT, requiring repeated boluses of adenosine. Due to this, amiodarone infusion was initiated.

Blood tests revealed no anemia (hemoglobin = 13.9 g/dL), normal thyroid and renal function as well as ionogram (namely potassium, sodium, magnesium, calcium), mildly elevated T-troponin levels with a plateau, and a pro-BNP level of 2477 pg/mL (cut-off <120 pg/mL). Echocardiogram revealed a non-dilated LV with global hypokinesis, severe LV dysfunction and severe functional mitral regurgitation. Non dilated right ventricule (RV) with depression of systolic function. No pericardial effusion.

A computed tomography pulmonary angiogram excluded pulmonary embolism and showed bilateral pleural effusion. Despite amiodarone infusion, the patient maintained recurrent and sustained SVT episodes, requiring repeated adenosine boluses. His condition deteriorated into cardiogenic shock with hypotension, hyperlactacidemia, tachypnoea, desaturation and olygoanuria. He initiated non-invasive ventilation, inotropic (dobutamine) and vasopressor (noradrenaline) support. Despite treatment, SVT persisted, leading to progressive multi-organ failure and severe biventricular dysfunction being referred to extracorporeal membrane oxygenation (ECMO). A venoarterial ECMO system was inserted via left femoral artery and vein, with a flow rate of 2,8 L/min. After the initiation of ECMO, there was clinical improvement, allowing for the withdrawal of vasopressor support. However, recurrent SVT

episodes persisted and echocardiogram showed persistent moderate LV dysfunction and moderate to severe mitral regurgitation.

On the 5th day of admission, it was decided to perform an EPS with ECMO support. During programmed ventricular stimulation, retrograde conduction was observed with a fixed ventriculoatrial interval and orthdromic atrioventricular reentrant tachycardia with concentric atrial activation was induced. A 3-D activation mapping using CARTO 3 System (Biosense Webster, Inc, Diamond Bar, CA) was performed, revealing the earliest activation site anterior to the ostium of the coronary sinus (CS), where a diverticulum was identified (Figure 3). Radiofrequency energy was applied around the diverticulum neck. At the end of the procedure, the tachycardia was no longer inducible by programmed stimulation.

After APS, echocardiogram showed improvement with mild LV dysfunction and mild to moderate mitral regurgitation. Amiodarone was suspended, there was no further SVT recurrences and ECMO support was withdrawn the following day. He remained haemodynamically stable in sustained SR. Echocardiogram at discharge showed: Mildly reduced LV ejection fraction (49%), normal RV systolic function and mild to moderate mitral regurgitation.

Discussion

This is a rare presentation of an incessant SVT, with poor response to medical therapy and challenging ablation, leading to TIC and cardiogenic shock. The recovery of LV ejection fraction after arrhythmia ablation confirmed our diagnosis.

The presentation with cardiogenic shock is exceptionally rare in the context of SVT. International guidelines do not provide specific recommendations for managing hemodynamically unstable patients or those in cardiogenic shock with SVT that is unresponsive to medical therapy and cardioversion, nor do they offer guidance on the optimal LV assist device for these situations [4,5]. In this setting, definitive SVT treatment is required to regain hemodynamic stability, so ablation should be considered [5].

In our case, the use of ECMO allowed hemodynamic support and EPS was performed with ablation of concealed accessory pathway.

A CS diverticulum is an anatomical variant that can contain accessory pathways connecting the ventricle and atria, difficult to ablate endocardially outside the diverticulum [6]. CS angiography can be considered, therefore, prior to catheter ablation of posteroseptal APs, especially when endocardial signals are not optimal [6].

CS diverticulum-associated accessory pathways are uncommon and the success rate of ablation is lower [7].

Failure to recognize a diverticulum can lead to unsuccessful ablation. In this case, the recurrence after a previous ablation and the posteroseptal location near coronary sinus raise the suspicion of a coronary sinus diverticulum.

Conclusions

This case underlines the complexity of managing a arrhythmia-induced cardiomyopathy, particularly in cases with anatomical variants and severe hemodynamic compromise and provides valuable insights into an under-recognized arrhythmia mechanism and management strategy.

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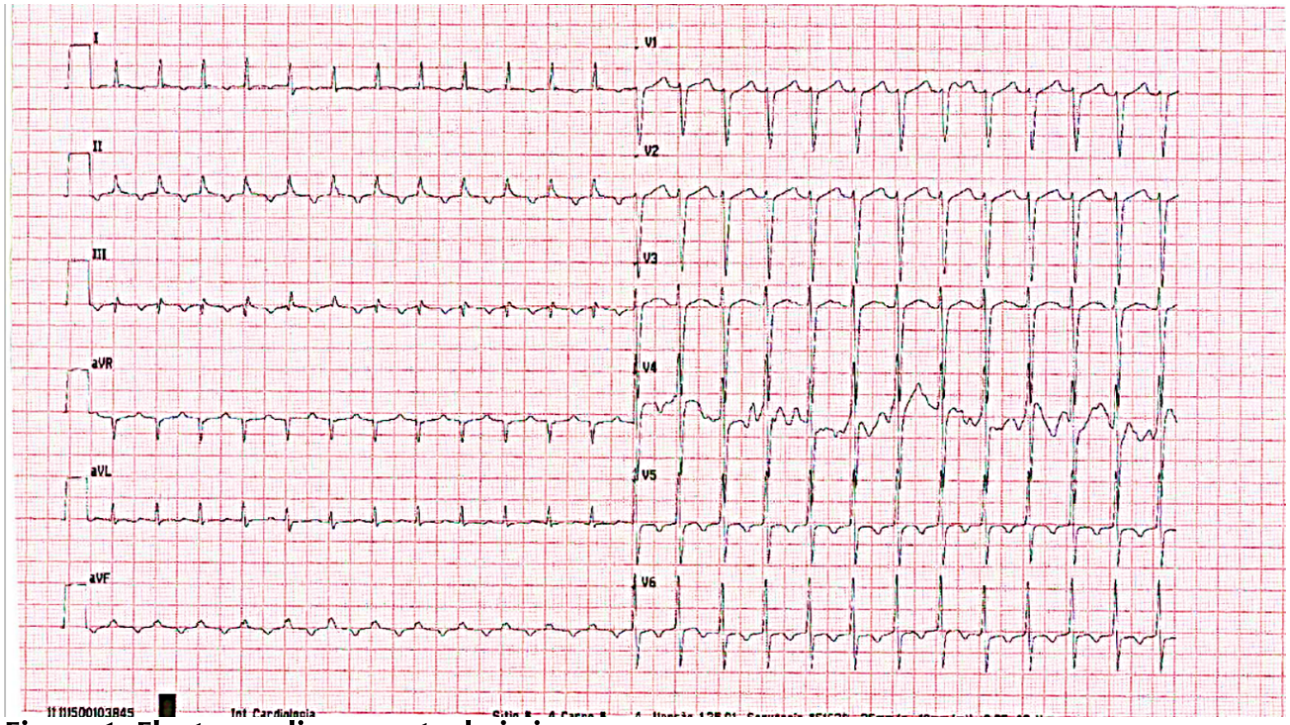

Figure 1. Electrocardiogram at admission.

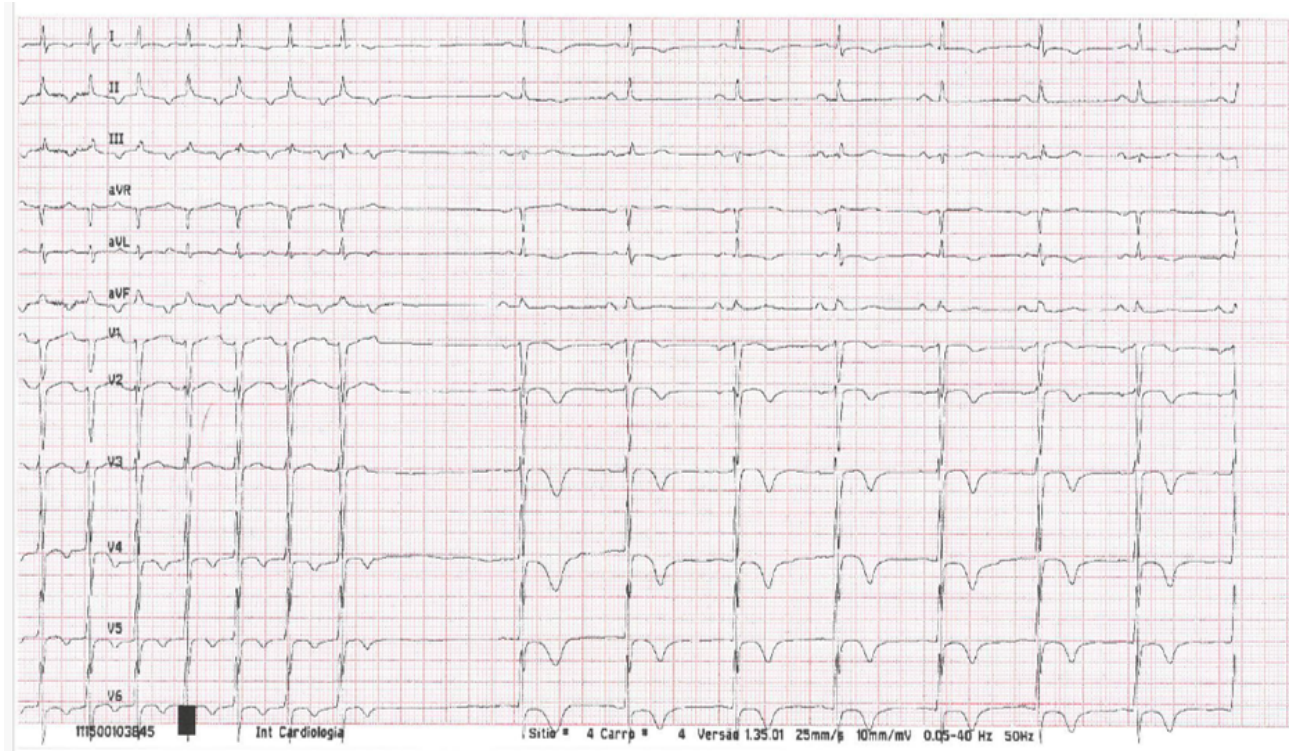

Figure 2. Electrocardiogram after adenosine bolus.

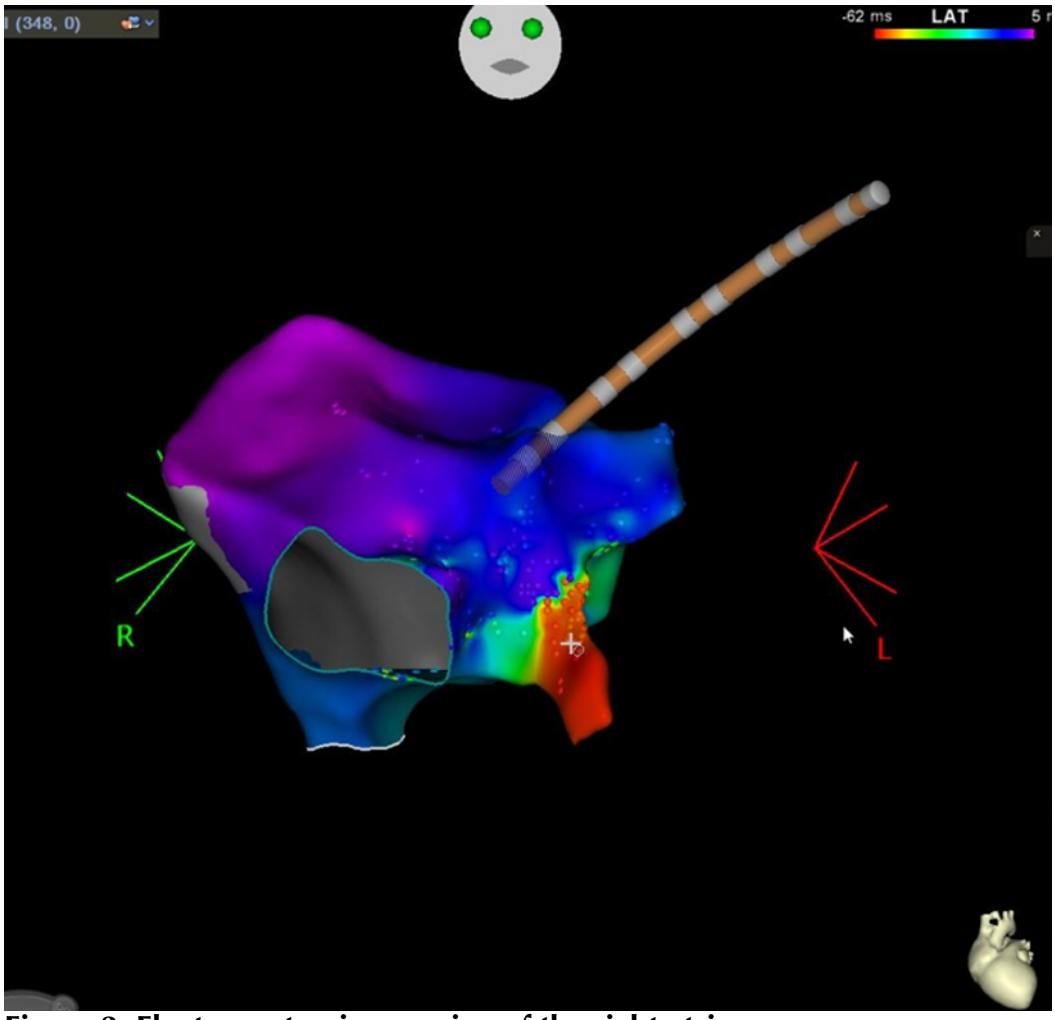

Figure 3. Electroanatomic mapping of the right atrium.