# A case report of a rare complication of an iatrogenic ventricular septal defect secondary to radiofrequency ablation

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## Introduction

Ablation is a common management technique for pharmacologically resistant ventricular tachycardias that is relatively safe for patients. However, up to 88% of patients who receive ablation will experience recurrence of their ventricular tachycardia and a proportion of these will require repeat ablation, putting them at higher risk of pericardial effusion and venous thrombosis.[1] In this case report, we describe a patient who suffered from delayed-onset ventricular septal defect formation following repeated ablations of the ventricular septum for ventricular tachycardias. To our knowledge, this is only the second case of its kind reported; however, this may present a previously unrecognized complication of the procedure.

**KEY TEACHING POINTS**

- Repeated/aggressive radiofrequency ablation has the potential to create delayed necrosis in myocardial tissue that may be sufficient to cause iatrogenic ventricular septal defects.
- Patients with underlying structural abnormalities may be more susceptible to radiofrequency ablation–induced tissue necrosis.
- Novel ablation strategies may be used in high-risk patients in an effort to avoid iatrogenic injury.

## Case report

The patient first presented at age 43 in 2002 with sustained ventricular tachycardia. Investigations revealed a nonischemic cardiomyopathy with an inferobasal aneurysm of the left ventricle abutting the mitral annulus and extending through the septum to the inferior portion of the right ventricle on echocardiography and ventriculograms, though preserved left ventricular function. Magnetic resonance imaging did not detect features of arrhythmogenic cardiomyopathy. At initial electrophysiological assessment, multiple ventricular tachycardia morphologies were induced, with 1 example shown in Figure 1.

Medical therapy failed to control the ventricular tachycardia.

Initial ablation was performed in 2003, with the multiple ventricular tachycardias arising in the aneurysmal portion of the septum, with exit points on both the right and left side of the septum. Radiofrequency ablation was performed on the left side of the septum without success. Subsequently a dual-chamber defibrillator was implanted.

In 2006 she underwent a second ablation attempt because of poor rhythm control with a substrate-based approach, with multiple radiofrequency ablations placed on both the left and right ventricular side of the septum, using an irrigated catheter and up to 50 W, though the ventricular tachycardia was not abolished.

In 2012 she underwent a third ablation attempt, with extensive irrigated radiofrequency endocardial ablation up to 50 W on both sides of the septum and adjacent epicardial ablation performed. This reduced ventricular tachycardia frequency and ventricular tachycardia rates to 96–100 beats per minute.

Later that year a further attempted ablation was aborted owing to inadvertent right ventricular perforation during epicardial access. Subsequently in late 2012 she underwent a final ablation. Extensive irrigated radiofrequency ablations up to 50 W were placed in the previously targeted area on the endocardial left ventricular septal area (Figure 2), and ventricular tachycardia was rendered noninducible. No steam pops were recorded in any of the ablation procedures that the patient received.

However, 4 months after her last ablation, she presented with sudden onset of dyspnea and was found to have a

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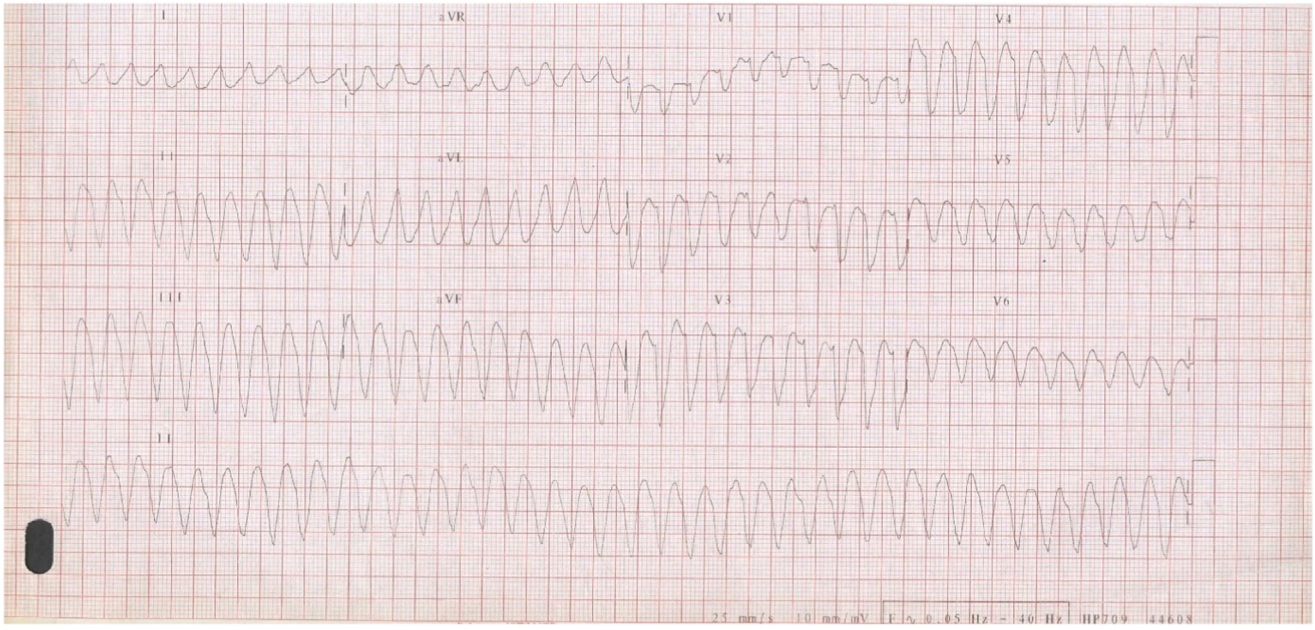

**Figure 1** Twelve-lead electrocardiogram of 1 morphological example of the patient's ventricular tachycardia.

ventricular septal defect (Figure 3) and severe tricuspid regurgitation. She developed progressive heart failure and proceeded to surgical repair.

At surgery, the patient was found to have a basal ventricular septal defect adjacent to the mitral and tricuspid valves. The edges were fragile, with necrotic tissue. The initial defect was 15 mm, but following debridement it was more than 20 mm in diameter. The defect was closed with a polyethylene terephthalate patch. In addition, the posterior tricuspid chord was thickened and the valve repaired with a De Vega annuloplasty, and part of the right atrium resected to reduce its size.

During 10 years of follow-up, ventricular tachycardia has not recurred. However, a small residual ventricular septal defect is present and she has developed moderately impaired left ventricular function and paroxysmal atrial fibrillation.

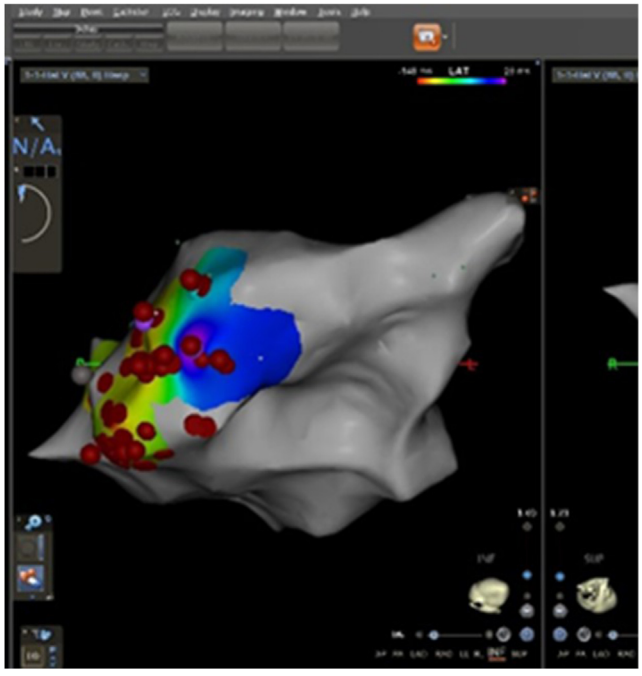

**Figure 2** CARTO (Biosense Webster, Irvine, CA) 3-dimensional activation map of ventricular tachycardia, inferior view of left ventricle, with earliest activation in red, and ablation points as red spheres.

## Discussion

Iatrogenic ventricular perforations are rare, typically 1% of ablations.[2] However, it is highly unusual for iatrogenic ventricular septal perforation to be reported post ablation. Schönbauer and colleagues[3] report a case of a patient who experienced intra-ablation steam pop and presented to hospital 2 weeks later with a ventricular septal perforation. Michaelis and colleagues[4] have reported a case of a 78-year-old woman who presented 1 week post ablation with a ventricular sepal perforation with the semi-lunar appearance typical of the steam pop phenomenon; however, no steam pops were recorded during the procedure.

To our knowledge, there is only 1 other case of a person presenting with a delayed ventricular septal perforation secondary to ablation and without recorded steam pops or features suggestive of one during or after the procedure. This case involved a 72-year-old woman with cardiac sarcoidosis.[5] Following 2 ablations for ventricular tachycardia, the patient presented with a left-to-right ventricular shunt in her basal septum, 1 year post procedure. The patient had thinning of the basal interventricular septum secondary to cardiac sarcoidosis, a trait shared with our patient (although our patient did not have any features of cardiac sarcoidosis). This raises the possibility that preprocedural septal thinning may

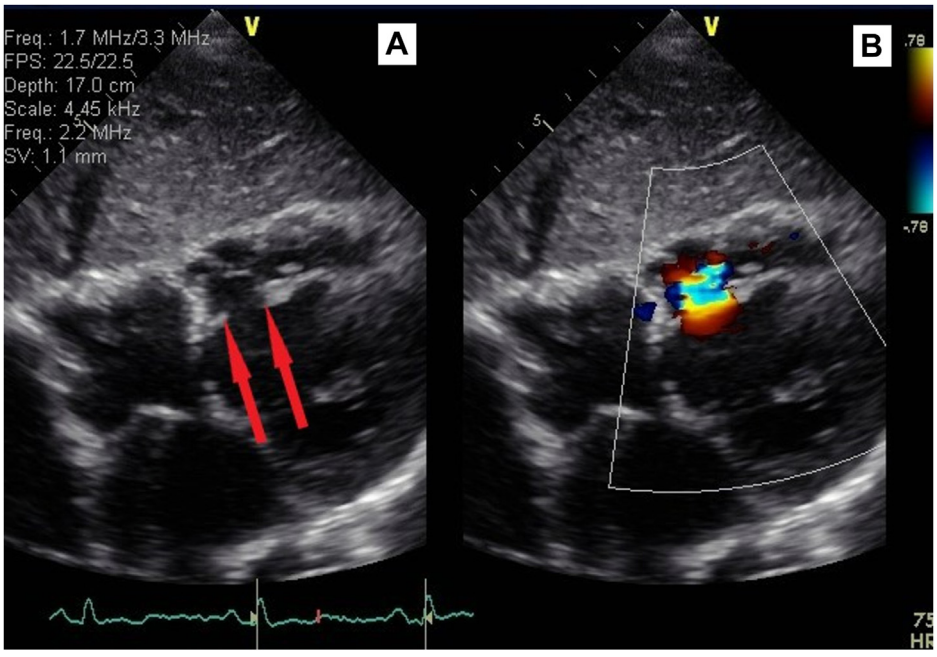


**Figure 3** Transthoracic echocardiogram. **A:** Subxiphoid view showing a large ventricular septal perforation wherein the red arrows mark edges. **B:** Color Doppler shows flow through the defect.

be a previously unrecognized risk factor for delayed formation of ventricular septal perforation following ablation procedures, particularly in the case of repeat ablations resulting in progressive myocardial fragility and increased delayed perforation risk.

In our case, the likely critical tachycardia circuit was deep in the septum, and initial attempts at ablation were unsuccessful. Following repeated ablations the ventricular tachycardia was abolished, but likely resulted in full-thickness necrosis of the septum that subsequently resulted in a large ventricular septal defect that necessitated surgical repair.

In future, for patients with deep septal tachycardias and/or underlying risk factors such as thinning of cardiac muscle in areas being ablated, alternative ablation strategies may reduce the risk of complications. Neira and colleagues[6] have postulated that bipolar septal ablation may both be more effective and reduce the risk of collateral damage.

We present this case to highlight this rare complication of radiofrequency ablation for ventricular tachycardia.

## Acknowledgments

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