Acute right ventricular failure complicating catheter ablation for right ventricular tachycardia

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Introduction
Catheter ablation is an important treatment modality for management of ventricular arrhythmias in the setting of implantable cardioverter-defibrillator (ICD) shocks and ventricular tachycardia (VT) storm. The frequency of patients with nonischemic cardiomyopathy requiring ablation for ventricular arrhythmias is increasing.1 Arhythmias of right ventricular (RV) origin are less common than those of left ventricular (LV) origin. Arrhythmogenic RV cardiomyopathy (ARVC), congenital heart disease, idiopathic dilated cardiomyopathy, valvular heart disease, and cardiac sarcoidosis are common causes of structural RV disease that manifest with VT. These diseases often mimic each other; differentiation is important for optimal management of these conditions.2 RV function is often difficult to define clearly but has major impact on outcomes from VT ablation.3

Periprocedural mechanical ventricular support is a consideration in VT ablations for patients with hemodynamic instability. However, most ventricular assist is designed for LV support. We present a case with predominantly RV involvement in a presumed ARVC-type cardiomyopathy, where mechanical acute RV failure following ablation culminated in a fatal outcome.

Case report
A 40-year-old morbidly obese Caucasian man originally presented a year previously with lightheadedness and was found to have recurrent VT with ICD therapies with shocks. He presented under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

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of >10 gms, and power delivery of 30–40 W to achieve a >10-ohm impedance drop.

RV biopsy showed moderate myocyte hypertrophy and diffuse intracellular vacuolization. Periodic acid–Schiff and periodic acid–Schiff–diastase stains were negative for glycogen. There was no evidence of active myocarditis, granulomatous inflammation, acute or recent infarction, amyloid heart disease, iron deposition, or fatty infiltration.

Following the ablation procedure, he was taken to the coronary care unit, still intubated. In the ensuing 2–3 hours, he demonstrated evidence of progressive cardiogenic shock. Echocardiography showed RV dilatation and ballooning. Venoarterial extracorporeal membrane oxygenation (VA ECMO) was instituted with immediate reversal of hemodynamic stability. He was on therapeutic heparin for ECMO. He was assessed by the advanced heart failure service and deemed not to be a candidate for long-term advanced heart failure therapies owing to morbid obesity.

Three days later, he was weaned off of mechanical support but continued to require high-dose ionotropic support. VT recurred with inadequate response to intravenous amiodarone. ECMO was preemptively re instituted to provide hemodynamic support for a repeat ablation. Two VT morphologies similar to the initial VTs were induced (VT1: left bundle left superior axis, CL 400 ms; VT2: right bundle basal left inferior axis, CL 440 ms). He was rendered noninducible by ablation in the high septal region endocardially. A total of 51 lesions were delivered with total radiofrequency ablation time of 2069 seconds. He subsequently stabilized, and was weaned off the mechanical support 5 days later. However, he developed cardiogenic shock soon after and an emergent right-sided TandemHeart percutaneous ventricular assist device was implanted, but he was unable to recover from severe circulatory collapse and died within 24 hours of ECMO decannulation.

Discussion

The present case highlights the importance of RV function in VT ablation. Despite adequate LV systolic function and functional class of NYHA II, acute or chronic RV decompensation in this patient resulted in cardiogenic shock. Although ECMO was successful in RV decompression and acute reversal of shock, the absence of longer-term durable RV support and contraindications to heart transplant resulted in a poor outcome. Mapping and ablation for sustained monomorphic VT is typically performed using activation and entrainment mapping when the arrhythmia is hemodynamically stable. In the present case, induced VT was slow and hemodynamically tolerated acutely. However, progressive RV stunning, prolonged anesthesia, and fluid load during the procedure likely led to RV decompensation and shock. In addition, ablation in the outflow tract region of the RV extending to the tricuspid annulus (Figure 3) could have resulted in conduction block or delay in a corridor of viable tissue, leading to transient RV electrical isolation and mechanical failure.

The prognostic significance of RV dysfunction on mortality outcomes in 320 patients with heart failure and LVEF < 0.4 who underwent VT ablation over a 9-year study period has been previously reported from our institution. Mortality was 2-fold higher (61% at 2 years) in patients with at least moderate RV dysfunction and tricuspid regurgitation with estimated pulmonary arterial pressure over 45 mm Hg. Options for mechanical support during ablation for scar-related VT include the Impella (Abiomed Inc, Danvers, MA), the TandemHeart system (Cardiac Assist, Inc, Pittsburgh, PA), the ECMO system, and intra-aortic balloon pump. All of these systems except ECMO provide only LV support, and therefore they are contraindicated or limited in patients with severe RV dysfunction. VA ECMO has a role in mechanical support for patients with severe RV failure, by offloading the right ventricle and bypassing the pulmonary vasculature. There is growing experience with using

![Figure 1](resting electrocardiogram prior to ventricular tachycardia ablation. Late ventricular activation is seen in the precordial leads and limb leads (black arrows).)

![Figure 2](traces of induced ventricular tachycardias (VT). VT 1: left bundle left superior axis. VT2: left bundle right inferior axis (see text for details).)
VA ECMO for temporary support in patients with advanced pulmonary hypertension and severe pulmonary embolism.4 RV assist devices are also available for temporary support or as a bridge to transplant, but not as destination therapy. Investigational right-sided TandemHeart and Impella systems are available at some centers, but these render the right ventricle less accessible to ablation. To date, none of these have been studied for potential use in supporting catheter ablation in patients with severe RV failure.

As most of the current modalities provide support only for the left ventricle, isolated RV failure presents a major challenge. In addition, identification of the patient who is likely to develop RV decompensation is not easy. Factors that can predict RV decompensation are more severe symptoms of heart failure on admission (NYHA III/IV), lower systolic blood pressure, dependence on long-term diuretics, and renal impairment. Echocardiographic features predictive of RV dysfunction include lower LVEF, at least moderate mitral regurgitation, or tricuspid regurgitation and evidence for pulmonary hypertension. However, in primary RV failure, pulmonary artery systolic pressure can be low and a sign of severe RV dysfunction. Several studies have suggested prediction models for RV failure based on renal impairment, hepatic impairment, use of vasopressors, ventilated status, and high central venous pressure.5–7 There has only been 1 small independent validation study that compared the different scores available, and it concluded that no single score could independently predict RV failure.8

**Conclusion**

In cases of primary RV disease with extensive RV scarring, such as in the present case, VT ablation can result in acute hemodynamic decompensation. Options for long-term RV hemodynamic support are very limited, as VA ECMO can only be instituted for a short term. Thus, in patients with severe RV dysfunction, strategies for bail-out in case of acute postprocedure hemodynamic decompensation should be discussed prior to VT ablation.

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