

Successful catheter ablation of hemodynamically significant ventricular tachycardia in a patient with biventricular assist device support



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Introduction

Ventricular assist devices (VADs) are playing a large role in the management of end-stage heart failure. Approximately 35% of patients with a VAD have ventricular arrhythmias within the first month of implantation.¹ Electrophysiologic ablations in the setting of a VAD are limited to left VADs (LVADs). To our knowledge, we describe the first report of ablation of a hemodynamically significant ventricular tachycardia in a patient with a biventricular assist device (BiVAD).

Case report

A 63-year-old female with a history of nonischemic cardiomyopathy underwent implantation of a HeartWare LVAD (Framingham, MA) in June 2013, but postoperatively developed severe right ventricular (RV) heart failure and underwent HeartWare RVAD placement. In February 2014, the patient presented with recurrent presyncope due to sustained ventricular tachycardia (VT). Despite the use of multiple antiarrhythmic drugs, multiple cardioversions, and changes in RV and left ventricular inlet flows, the patient continued to have recurrent, sustained VT, leading to hemodynamic compromise. The sustained VT caused a decrease in mean arterial blood pressure by roughly 10 mm Hg and decreased BiVAD cannulae flows.

Because of sustained and symptomatic VT despite biventricular support, the patient was taken for electrophysiologic study and VT ablation. The clinical VT had a cycle length of 300 milliseconds, with axis and precordial

transition suggestive of an inferoapical exit site (Figure 1). Extensive mapping was performed in the right ventricle. Overdrive pacing in the right ventricle at presystolic sites near the RV inlet cannula led to good pace maps of the VT with a postpacing interval minus tachycardia cycle length of 14 milliseconds (Figure 2A). Neither broad activation mapping to suggest a macroreentrant circuit nor maneuvers to demonstrate progressive fusion were performed, so it is unclear if the VT mechanism was focal or reentrant. Catheter ablation was performed at this site using a ThermoCool SF catheter (Biosense Webster, Diamond Bar, CA) at 35 W with a maximum temperature of 40°C. Twenty-five seconds into ablation, the VT terminated (Figure 2B). Further ablation was performed in this region, toward the RV inlet cannula (Figure 3). VT was noninducible despite triple extrastimuli and changes in RV inlet flow (previously this method had reproducibly induced the clinical VT). Mapping of the left ventricle was not performed, as the arrhythmia was not inducible after termination. Afterward, administration of the antiarrhythmic agents lidocaine, amiodarone, and propranolol was discontinued. At similar pump speeds, the RVAD pump flow increased from 4.4 L/min to 5 L/min and the LVAD pump flow increased from 3.5 L/min to 5 L/min postablation. The patient was symptom free in subsequent follow-up clinic visits, and she underwent successful heart transplantation in June 2014. Direct visualization of the explanted heart showed extensive scarring within the right ventricle in the region of successful ablation.

Discussion

Ventricular arrhythmias are known to occur after LVAD implantation.² Although initial reports suggest that ventricular arrhythmias may be tolerated in patients with LVADs,³ prolonged RV failure, thrombus formation, or decreased flows via the LVAD may lead to undesired consequences. Further, recent data have suggested that ventricular arrhythmias post-LVAD implantation in this patient population may be associated with increased mortality.⁴ Given these considerations, catheter ablation for patients with LVAD has been reported.^{1,5}

KEYWORDS Ventricular tachycardia; Catheter ablation; Biventricular assist device; Ventricular assist device

ABBREVIATIONS Abl = ablation catheter; BiVAD = biventricular assist device; CS = coronary sinus catheter; LVAD = left ventricular assist device; LVIC = left ventricular inlet cannula; RV = right ventricular; RVAD = right ventricular assist device; RVIC = right ventricular inlet cannula; VT = Ventricular tachycardia (Heart Rhythm Case Reports 2015;1:209–212)

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KEY TEACHING POINTS

- Patients with left ventricular assist devices can develop hemodynamically significant ventricular tachycardia.
- Catheter ablation can be safely performed in patients with ventricular tachycardia and ventricular assist devices.
- Ventricular tachycardia may cause hemodynamic sequelae even in patients with biventricular assist devices.

Traditionally, with BiVAD support, ventricular arrhythmias are generally tolerated given both RV and left ventricular support. Refaat et al⁶ reported no difference in survival to transplant in BiVAD patients with an implantable cardioverter-defibrillator. We, however, report a case of symptomatic, hemodynamically significant VT despite BiVAD support. Continuous flow VADs are known to cause

“septal push,” which can induce VT from the inlet cannula as it creates a suction effect against the myocardium. In our patient, despite multiple changes in flow rates, VT persisted. The patient’s symptoms potentially were from lack of pulsatile flow in VT, as pulsatility was restored after ablation, with improved BiVAD cannulae flows. Given the patient’s symptoms and that no donor heart was available, the decision was made to pursue catheter ablation.

The successful site of ablation in this patient was near the RV inlet cannula within the right ventricle. Although reentrant VT can occur around an inlet cannula, intrinsic myocardial scar-related VT has been the predominant mechanism reported in LVAD patients.^{1,5} Entrainment maneuvers were not performed around the various cannulae to elucidate if this VT was focal or a reentrant one involving the RV, LV, or both inlet cannulae. This patient had an underlying nonischemic cardiomyopathy and electrocardiogram features suggestive of an epicardial exit.⁷ Further, unipolar RV voltage mapping revealed a large area of low voltage (<5.5 mV) in this region, also suggestive of epicardial scar.⁸ This finding is potentially why there was a delayed termination during ablation.

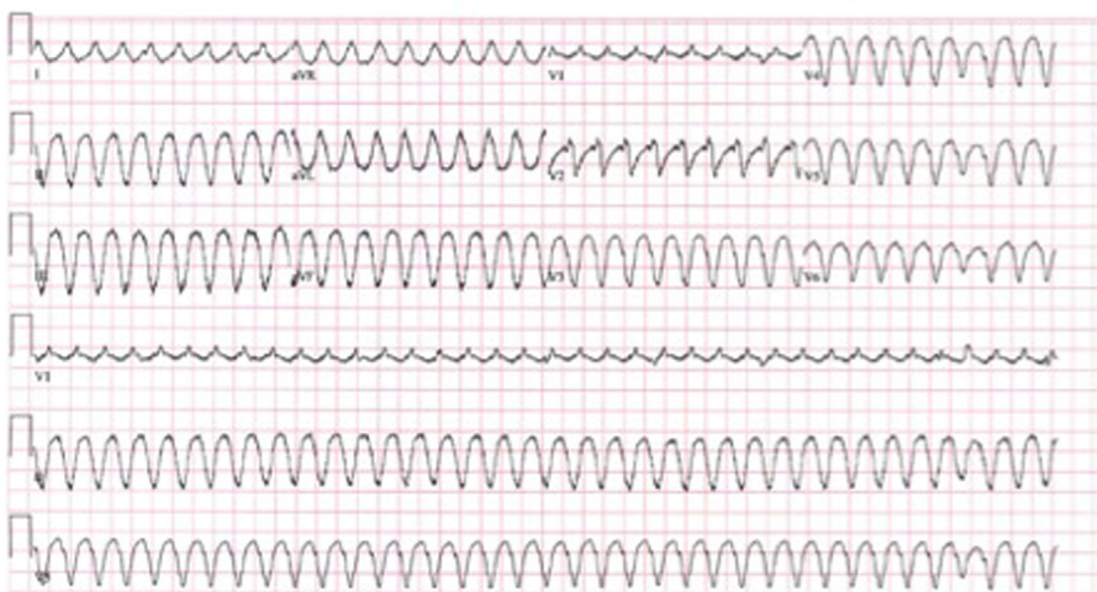


Figure 1 A 12-lead electrocardiogram of clinical ventricular tachycardia.

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