Epicardial Catheter Ablation Through Subxiphoid Surgical Approach in a Patient With Implanted Left Ventricular Assist Device and Cannula-Related Ventricular Tachycardia

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A 70-year-old man with nonischemic dilated cardiomyopathy initially diagnosed in 2001 and history of cardiac resynchronization therapy-defibrillator implant was admitted to our institution for advanced heart failure therapies. He required hospitalization in December 2013 for marked volume overload, at which time cardiac imaging showed his left ventricular (LV) ejection fraction to be 10% to 15% and his LV end-diastolic dimension 7.9 cm. The patient underwent implant of a Heartmate II LV assist device (LVAD; Thoratec, Pleasanton, CA) with concurrent mitral valve ring repair, tricuspid valve ring repair, and aortic valve repair. However, on postoperative day 9, he developed repetitive monomorphic ventricular tachycardia (VT) with rate 141 bpm (Figure 1). During VT, a decrease in pulsatility index was noted on LVAD interrogation. A transesophageal echocardiogram demonstrated excellent cannula position, and no evidence of LVAD-cannula–induced suction events was observed. The VT was unresponsive to antitachycardia pacing, and despite treatment with amiodarone, lidocaine, and esmolol infusions, VT remained incessant.

An initial catheter ablation was performed using an endocardial approach. A 3-dimensional electroanatomic map (CARTO; Biosense Webster, Diamond Bar, CA) of the left and right ventricles with a 3.5-mm open-irrigated catheter (Thermocool SF; Biosense Webster) demonstrated a small area of low voltage, defined according to bipolar electrogram amplitude <1.5 mV, mainly near the LVAD inflow cannula (Figure 2A). Activation mapping during VT demonstrated earliest endocardial site of activation at the apical septum in both the LV and the right ventricle, in close proximity to the LVAD inflow cannula. However, these areas were only 26 ms early compared with the surface QRS. Several ablation lesions, with maximum power of 35 W, maximum duration of 180 s, were delivered in the LV with no effect on the VT. Ablation on the right ventricular side of the apical septum at an electrogram of 37 ms pre-QRS (35 W; 240 s) resulted in VT termination. However, within several hours after the initial catheter ablation, VT recurred and again became incessant, although slightly slower than prior to the procedure (=125 bpm).

The next day, a second ablation procedure was performed with the purpose of epicardial mapping and ablation of the incessant apical VT. General anesthesia was induced, and intravenous heparin infusion was discontinued immediately before reopening of the lower edge of the sternotomy incision by 2.5 inches in the subxiphoid area. The pericardium was reopened, and blunt dissection was used to ensure access to the epicardial surface around the inflow cannula. An 8.5 French deflectable sheath (Agilis, St. Jude Medical) was used to direct a 3.5-mm open-irrigated RF catheter (Thermocool SF). Activation mapping during VT demonstrated electrograms that were 97 ms pre-QRS, much earlier when compared with the endocardial electrograms. Radiofrequency energy was delivered on the epicardial surface near the LVAD inflow cannula, with power titration to a maximum of 30 W for 60 to 210 s. VT slowed and terminated during radiofrequency energy delivery, and further ablation was performed around the inflow cannula at sites of late potentials (Figures 2B and 3). During voltage mapping of the epicardial surface, a more extensive scar burden was noted, especially around the LVAD inflow cannula. After radiofrequency delivery, VT was no longer inducible with programmed stimulation with triple extrastimuli from the right ventricular apex. The pericardium and the soft tissue were surgically closed, and the patient was extubated shortly after return to the intensive care unit. The time required to obtain subxiphoid access was 8 minutes, incision closure time was 11 minutes, and total procedural time from percutaneous access to removal of femoral sheaths was 2 hours 20 minutes. Amiodarone and mexiletine were continued, and the patient remained hospitalized for further diuresis and postoperative recovery. Nineteen days after the epicardial VT ablation, the patient was discharged home with no evidence of VT recurrence. During 6 months of follow-up, the patient has remained free of sustained VT, while prescribed oral amiodarone and mexiletine, and is now awaiting orthotopic heart transplantation.

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Discussion

Ventricular arrhythmias after LVAD implant are common and often amenable to treatment with catheter ablation. In patients with ischemic cardiomyopathy, re-entrant VT can often be ablated endocardially near areas of electric scar associated with previous myocardial infarction. However, in nonischemic cardiomyopathy, epicardial distribution of scar and sites of origin for VT is comparatively more frequent. Here, we report a case of VT in a patient with implanted LVAD that was ultimately ablated on the epicardial surface near the inflow cannula. Although the role of pre-existing epicardial substrate associated with his cardiomyopathy cannot be denied, we suspect that the additional scar created by the LVAD implant was an important factor for the development of re-entrant VT.

Although the presence of the LVAD allows for VT to be better tolerated hemodynamically and thus for the critical isthmus to be mapped more precisely during ablation procedures, important procedural challenges arise in the case of epicardial VT in the patient with LVAD. The use of a limited anterior thoracotomy and a surgical ablation tool to treat epicardial VT in a patient with an implanted LVAD has previously been reported. The acquired VT in our patient required percutaneous subxiphoid window access to allow for catheter mapping and ablation. In this case, the subxiphoid window performed by cardiac surgeons reduced the risks inherent in accessing the pericardial space and allowed for wide access around the inflow cannula. In conclusion, epicardial cannula-related VT can be ablated safely in the setting of implanted LVAD.

Disclosures

None.

References


Key Words: catheter ablation ■ epicardial mapping ■ tachycardia, ventricular

Figure 1. ECG of ventricular tachycardia, consistent with apical site of origin.

Figure 2. A, Left anterior oblique view of endocardial right ventricular (RV) and left ventricular (LV) voltage map, showing small area of low-amplitude signal <1.5 mV at the LV apex represented by red shading. B, Left anterior oblique view of epicardial LV voltage map, showing more extensive low-voltage area at the apex. Blue dots represent sites of ventricular tachycardia termination during ablation, red dots represent additional ablation sites. AP indicates anteroposterior; INF, inferior; LAO, left ventricular outflow; LAT, lateral; LL, left lateral; PA, posterolateral; RAO, right ventricular outflow; RL, right lateral; and SUP, superior.

Figure 3. Anterior-posterior fluoroscopy image with ablation catheter (Abi) position in the pericardial space lateral to the left ventricular assist device inflow cannula at ventricular tachycardia termination site.
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