Cryoablation during left ventricular assist device implantation: A case report

Ignoramus et Ignorabimus, Sapere Aude

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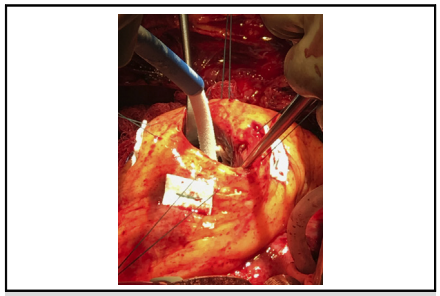

Arrested heart endocardial cryoablation through an apical ventriculotomy before LVAD.

CENTRAL MESSAGE

Endomyocardial cryoablation during an LVAD implant is an option for patients with end-stage heart failure who have refractory VT. More studies are needed to evaluate this strategy.

See Commentaries on pages 58 and 60.

Ignoramus et ignorabimus, sapere aude (We ignore and we'll ignore, dare yourself to know). A left ventricular assist device (LVAD) is an effective treatment modality for select patients with advanced heart failure (AHF). Ventricular arrhythmias (VAs) are common in the preoperative period with an incidence ranging from 22% to 52%.[1] Although patients can tolerate VA in the short-term after LVAD implantation, VAs are associated with increased morbidity, mortality, and length of stay.[2,3] There is no standardized strategy for managing these patients.

Case reports of cryoablation during LVAD implant surgery have controversial results.[3-5] This procedure is based on the premise that the epicardium and endocardial surface can be visualized. We present a patient with AHF with nonischemic cardiomyopathy and ventricular tachycardia (VT), refractory to conventional therapy, who received a HeartMate 3 (Abbott, Chicago, Ill) LVAD and simultaneous endomyocardial cryoablation.

CASE REPORT

A 71-year-old white man with AHF, biventricular implantable cardioverter defibrillator, and recurrent monomorphic VT with 2 radiofrequency ablations within 12 months was referred for advanced therapy consideration. A nuclear stress test demonstrated significant inferoapical scaring. Previous electrophysiologic mapping suggested scar-mediated VT involving the basal wall of the left ventricle (LV). Despite ablation and medical therapy, he had multiple readmissions decompensated with VT.

With the patient's declining functional status, home intravenous dobutamine was initiated. This improved his functional status, but antitachycardia pacing episodes and implantable cardioverter defibrillator shocks leading to hospital admissions persisted.

Echocardiography revealed a dilated LV, ejection fraction less than 20%, and decreased right ventricular function. Right heart catheterization revealed a right atrial pressure of 19 mm Hg, right ventricle pressure of 50/13 mm Hg, pulmonary artery pressure of 50/30 mm Hg, wedge pressure of 35 mm Hg, and cardiac index of 1.37. Creatinine was 1.7 mg/dL. An intra-aortic balloon pump was placed as a bridge to advance therapy candidacy. In 1 week, hemodynamics improved with inotropes, intra-aortic balloon pump, amiodarone, sotalol, lidocaine, and diuretics. VT decreased, and renal function normalized.

The patient was categorized as INTERMACS 2, and he underwent HeartMate 3 implant. On full cardiopulmonary bypass, the patient's aorta was crossclamped, and cardioplegic solution was administered. Apical ventriculotomy was performed with the coring device. We performed endocardial cryoablation on the arrested heart so visualization could be maximized, which allows detailed,

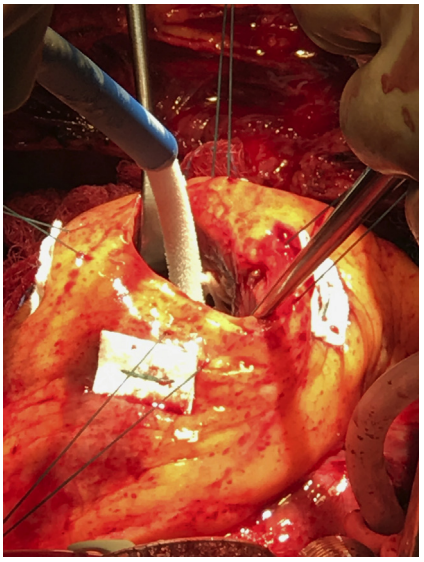

FIGURE 1. Arrested heart endocardial cryoablation through an apical ventriculotomy before LVAD implantation.

precise, complete and constant myocardial contact (Figures 1 and 2). To prevent reentry circuits, we created a surrounding ablation tract to fixed anatomic sites, that is, the mitral valve. No intraoperative or postoperative electrophysiologic mapping was performed.

After cryoablation, the aortic clamp was released and LVAD implantation was completed. After cardiopulmonary bypass weaning, intraoperative transesophageal echocardiogram showed right ventricular dilatation with poor LVAD filling. Right ventricular mechanical support with the CentriMag (Abbott) was initiated via percutaneous venous femoral access and direct pulmonary artery cannulation. Three days later, this was transitioned to a ProtekDuo cannula (TandemLife, Pittsburgh, Pa) via the right internal jugular vein. The patient had a VT episode in the early postoperative period that was treated successfully with defibrillation and intravenous lidocaine and amiodarone.

Ventricular ectopy decreased during the first 72 hours after surgery. Right ventricular support was removed after 11 days. His subsequent hospital stay was notable for ileus, urinary retention, and physical deconditioning. He was discharged to inpatient rehabilitation. Five months after implant, he remains in New York Heart Association Class I, without VT or implantable cardioverter defibrillator shocks.

DISCUSSION

Dor and colleagues[6] reported cryoablation and LV reconstruction in 1994 with optimal results. Other groups, following myocardial scar resection principles and endomyocardial and epicardial cryoablation, achieved arrhythmia resolution with ventricular reconstruction.[7,8] Even when myocardial scar is not resected, ventricular decompression in conjunction with cryoablation may be effective.

To our knowledge, this case is the first in which endomyocardial cryoablation was performed before HeartMate 3 implant. Criteria to select patients who may benefit from this procedure are not known, but it is well established that risk of VT recurrence is highest in those with VT before LVAD implant.[9] Reports describe this approach with the HeartMate II (Abbott) LVAD implantation.[3-5] The patient presented could represent a select case in whom ablation provides an additional tool for arrhythmia reduction, similar to previous studies. One potential complication is LVAD thrombosis after endomyocardial cryoablation. As a result, some authors advise limiting to epicardial surface ablations.[5]

Right ventricular failure developed in our patient, which was not unexpected because he exhibited preoperative risk factors for this complication. In addition, because of

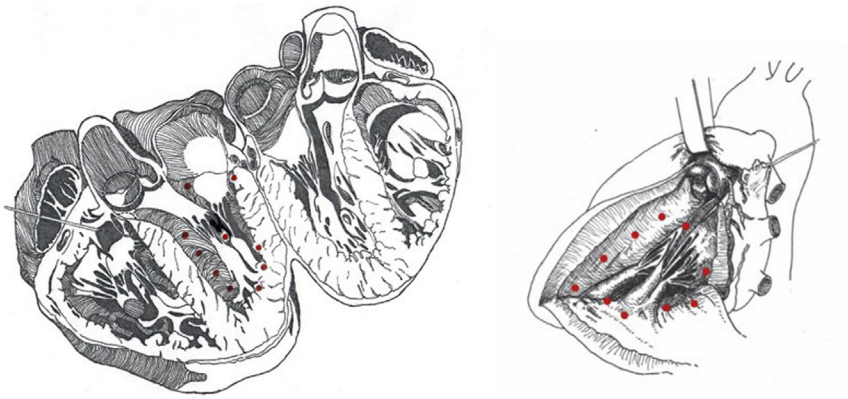

FIGURE 2. The probe was applied to the papillary muscles base, septum, and over the basal and inferior LV surface by direct visualization. There were 10 points ablated homogeneously. Each arrhythmogenic substrate was ablated at −70° C for 2 minutes without repetition. The *points* and *lines* were based on previous electrophysiologic mapping that showed basal to mid inferoseptal substrate. *Points* are shown above in *red*.

interventricular interdependence, there is a possibility that the septal cryoablation extent may have contributed.

There is the theoretical possibility that our patient's VT burden would be alleviated by adequate LV unloading alone, obviating the need for cryoablation. However, we do not believe our patient's VT was purely hemodynamically mediated. Although ectopy improved preoperatively, it did not completely resolve.

CONCLUSIONS

Cryoablation during LVAD implantation as a combined procedure is a feasible approach to manage refractory VT in patients with AHF. Additional studies to refine patient selection are needed.

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