

Massive myocardial edema and inflow cannula obstruction due to epicardial surgical ventricular tachycardia cryoablation at time of left ventricular assist device implantation



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Introduction

Ventricular tachycardia (VT) is a common comorbidity in patients with end-stage heart failure. For those that require left ventricular assist device (LVAD) support, the occurrence of VT post LVAD implantation portends a significantly worse prognosis—in terms of both morbidity and mortality. Owing to the technical difficulties of performing VT ablation after LVAD implantation, concurrent surgical ablation at time of surgery has been proposed as a strategy employed to reduce long-term arrhythmia burden. We report a case of hemodynamically significant LVAD inflow obstruction after surgical VT ablation performed simultaneously with LVAD insertion and the successful use of stellate ganglion blockade (SGB) to treat post-LVAD arrhythmias.

Case report

A 61-year-old man with nonischemic idiopathic cardiomyopathy was admitted with acute decompensated heart failure. Over the 2 previous years he had experienced multiple appropriate implantable cardioverter-defibrillator (ICD) shocks for VTs that were refractory to medical therapy and underwent 4 endocardial radiofrequency ablation procedures over an 18-

month period of time. During his most recent radiofrequency ablation, 3 distinct VT morphologies were identified and mapped to the septum. Prolonged periods of VT during the procedure were not hemodynamically tolerated, but all 3 areas were successfully ablated. At the conclusion of the case the patient was only inducible for a nonclinical VT right bundle right superior with an inferolateral, basal epicardial exit site. Postprocedural noninvasive programmed stimulation was performed and showed the same VT, which terminated with antitachycardia pacing.

Owing to the severity of his heart failure symptoms, he underwent an expedited advanced therapies evaluation and was declined for primary transplantation owing to comorbidities. He was unable to tolerate intravenous inotropic therapy (milrinone) owing to increase in VT burden. Therefore, the decision was made to proceed to LVAD implantation as destination therapy. A multidisciplinary meeting between the surgical, heart failure, and electrophysiology teams was convened, and given his arrhythmia history the decision was made to attempt intraoperative VT ablation at the time of his LVAD implantation with a goal to reduce the burden of VT postoperatively.

Surgical VT ablation was performed with a CryoFlex catheter (Medtronic, Minneapolis, MN) targeting the epicardium while on cardiopulmonary bypass. A total of 4 applications for 2 minutes were delivered targeting the remaining inducible VT from the basal lateral left ventricle and additional lesions directed toward the inferior wall where multiple VTs had been targeted from endocardial ablation (Figure 1a and b). No mapping was performed intraoperatively. Following this, a HeartWare HVAD (Medtronic, Minneapolis, MN) was implanted with successful weaning off bypass at a final speed of 2700 rpm and VAD flows of 3 L/min. Intraoperative transesophageal echocardiography (TEE) at the conclusion of the case showed low normal left ventricular size with normal wall thickness.

KEYWORDS Ablation; Complication; Heart failure; LVAD; Stellate ganglion block; Ventricular tachycardia
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KEY TEACHING POINTS

- Concomitant surgical ablation at the time of left ventricular assist device (LVAD) implantation remains in its nascent phase, and careful preoperative assessment and periprocedural management is needed.
- Extensive ablation of partially scarred myocardium combined with acute left ventricle (LV) unloading that occurs at the time of LVAD implantation can lead to edema that causes mechanically and hemodynamically significant changes in the LV cavity.
- Stellate ganglion block can be used in selected LVAD patients and has the potential to be a valuable adjunct therapy for ventricular tachycardia in unstable patients.

The patient was returned to the cardiothoracic intensive care unit, where after an initial period of stability he was noted to have low VAD flows (below 2 L/min) with very low flow variability. Pericardial tamponade was excluded, and lactate dehydrogenase, LVAD power, and bilirubin were normal, thus excluding acute LVAD thrombosis. Attempted alterations to LVAD speed and changing intravenous hemodynamic support were unsuccessful. The decision was made to return to the operating room to assess for acute outflow graft obstruction. At the beginning of the case, the intraoperative TEE showed significant thickening of the lateral wall of the left ventricle causing partial

LVAD inflow cannula obstruction (Figure 1c and d). This was considered most likely due to edema, given the echocardiographic appearance and the rapidity of the change from the initial postimplant TEE.

The LVAD speed was reduced to allow for increased cavity size, resulting in improved LVAD variability and flows. The combination of septal deformation and second chest opening led to worsening right ventricular function, and an Impella RP (Abiomed Inc, Danvers, MA) was inserted for temporary right ventricular support. The patient was left on this biventricular support platform for the following 7 days, over which time there was progressive improvement of ventricular edema. The Impella RP was removed on postoperative day 7, with complete resolution of ventricular edema by postoperative day 14.

After the removal of the Impella RP, there was an increased need for inotropic support, which in turn led to the development of slow monomorphic VT, which was different from the targeted clinical VT (Figure 2), which was hemodynamically significant. Given the recovering left ventricle, an emergent ablation was deemed too high risk, and the decision was made to pursue a percutaneous bilateral SGB in an effort to bridge the patient to clinical stability in the form of reduced proarrhythmic inotrope doses, appropriate loading of antiarrhythmic medications, and resolution of myocardial edema. This was performed at the bedside and resulted in termination of VT. Cardiac sympathectomy was considered; however, owing to the recent surgery, anticoagulation, and the resolution of VT it seemed to be a greater-than-usual risk and was not pursued further.

The patient had a prolonged intensive care course with a respiratory wean but was eventually successfully discharged to a rehabilitation facility 6 weeks post implant.

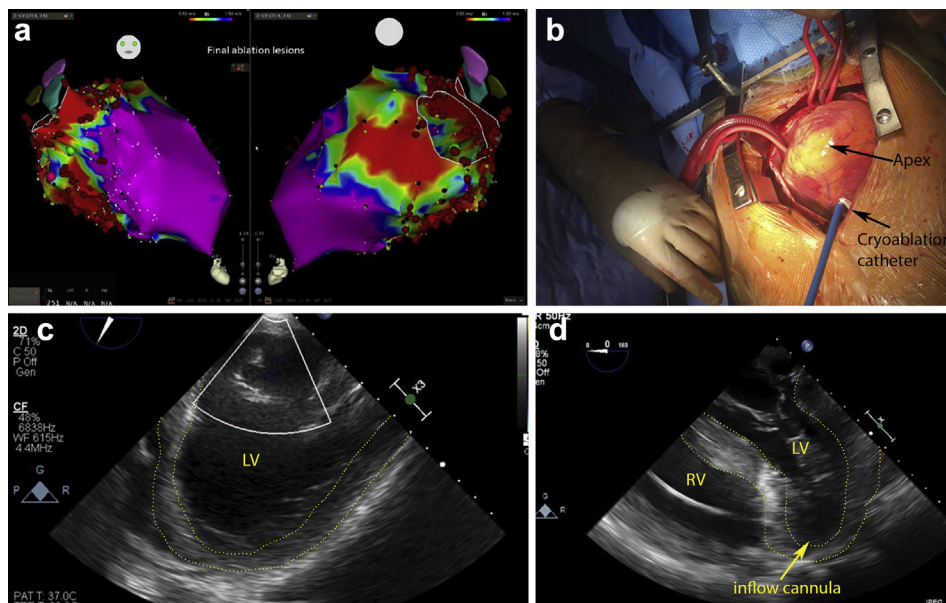


Figure 1 a: CARTO (Biosense Webster Inc, Diamond Bar, CA) mapping of arrhythmia. b: Intraoperative cryoablation. c: Intraoperative transesophageal echocardiography (TEE) showing left ventricle (LV) size. d: Postoperative TEE showing reduced LV size with increased wall thickness. RV = right ventricle.

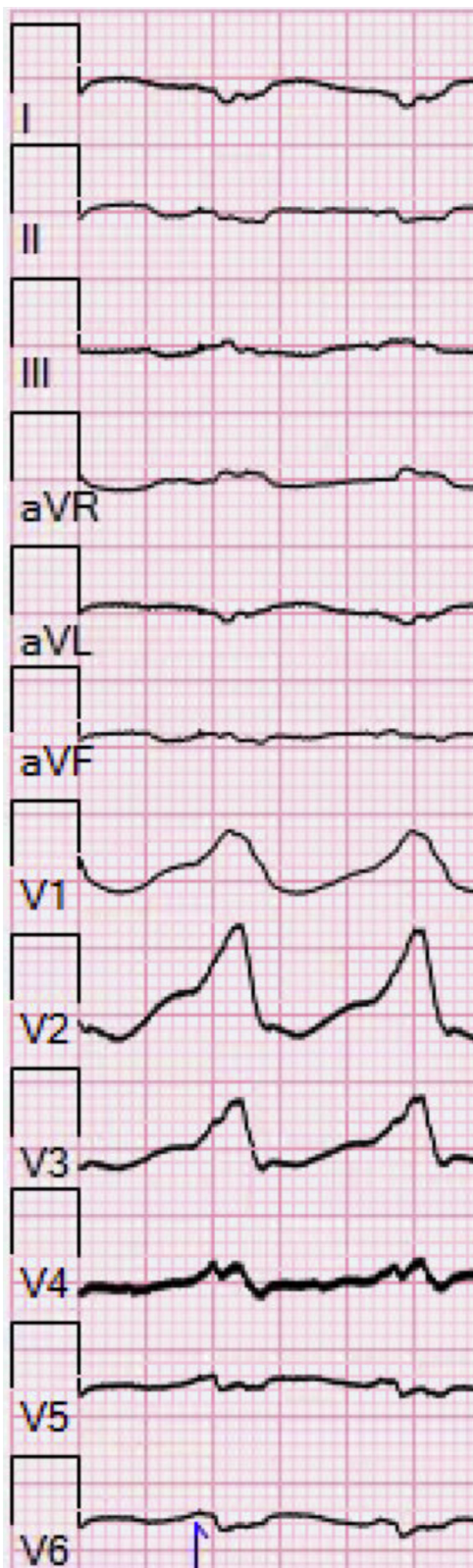


Figure 2 Post Impella (Abiomed Inc, Danvers, MA) removal: slow monomorphic ventricular tachycardia (right bundle right inferior, cycle length 560 ms).

Discussion

Ventricular arrhythmias in the LVAD population are a unique management dilemma. VT itself is not only a common complication but is also a poor prognostic indicator notable for increased adverse events (including stroke and heart failure hospitalizations) and mortality. Studies have shown that preimplant VT is one of the strongest predictors of postimplant VT.¹

Currently there is no consensus on the optimal management strategy in this patient population. In patients who have preimplant VT, it has been noted that antiarrhythmic therapy has no survival advantage over ICD therapy—mostly owing to the fact the VT is often better tolerated in the LVAD population as the LVAD maintains circulatory support. The larger problem arises in that ICD therapy in an LVAD patient is often fraught—the rates of inappropriate therapy are high, and the majority of therapy is delivered while the patient is conscious and is highly unpleasant, with significant psychological trauma. There is also the paradox of increased morbidity of ICD shock therapies in themselves.²

VT ablation in the LVAD population

A recent meta-analysis has been published that looks at the use and efficacy of VT ablation after LVAD implantation.³ This study included 18 studies and a total of 110 patients and showed a reasonable procedural success—with noninducible clinical VT achieved in 77.9% of patients and a low complication rate (9.4%). These studies have showed that there was a significant reduction in ICD therapy (57.1% vs 23.8%), albeit with a very high rate of VT recurrence (43.6%). One of the hypothesized reasons for this is that there are significant technical challenges to ablation procedures in LVAD patients. The aortic valve may be surgically closed at the time of LVAD implantation, limiting access to the basal septum, and percutaneous epicardial VT ablation is generally not possible owing to an inability to access the pericardium.

This has led a number of centers to propose the practice of concomitant VT ablation at the time of implantation. This has the benefit of allowing for open exposure of the entire epicardial surface, as well as access to the endocardium from the apex (via the ventriculotomy made to insert the LVAD inflow cannula), and potentially the ability to tolerate more aggressive mapping/prolonged periods of VT while the patient is on full-flow cardiopulmonary bypass. Although there are a number of anecdotal reports of this approach, there are small case series that have been published.⁴⁻⁷

The first looked at 7 patients who underwent a combination of endocardial and epicardial cryoablation corresponding to areas of interest as defined by previous electrophysiologic mapping⁴—similar to the patient in our case. It compared these patients to 7 control patients (who did not have ablation procedures) and demonstrated there was no recurrence of VT, compared to 4 in the control group.

There were no major procedural or postoperative complications noted in this study.

A second study looked at 5 patients who had previously failed endocardial ablation or had clinical epicardial VT, and then subsequently underwent epicardial radiofrequency ablation at the time of LVAD implant.⁵ There was procedural success in 60% of patients, with 1 patient having significant mediastinal bleeding as a complication.

The third study was of 2 patients who underwent endocardial and epicardial cryoablation.⁶ Both had successful ablation procedures but were complicated by LVAD thrombosis post implant, although this may have been related to the LVAD device type, which has been shown to predispose patients to a significantly higher risk of VAD thrombosis than newer-generation LVADs.

The final study⁷ surgically examined epicardial mapping and ablation at the time of VAD implantation. This multicenter prospective cohort study involved 36 patients, of which 15 were included in the final analysis (11 were excluded as part of an early-phase/learning cohort, and a further 11 did not have analyzable maps). This study showed that adding mapping to the operation is quick, adding a median of 11.8 minutes of mapping time; is safe; and potentially reduces the overall arrhythmia burden.

Our case is the first reported of significant acute myocardial edema in the setting of a peri-implant ablation procedure. This is most likely owing to ablation being performed in patchy nonischemic substrate interdigitated with viable myocardium and normal coronary perfusion. The net result was significantly more myocardial edema generated than expected, which in turn was exacerbated by a smaller-than-usual left ventricular cavity and acute left ventricular unloading. One differential diagnosis for this presentation is injury or edema to the coronary arteries or branches of the great cardiac vein owing to the anatomical location in relation to the ablation. The definitive diagnosis would be via coronary angiogram with levophase ventriculography, which unfortunately is not possible in the setting of an LVAD because of the rapid emptying of the left ventricle through the LVAD itself, but selective imaging of the coronary arteries and coronary sinus could be considered if the patient is clinically stable.

Stellate ganglion block

Previous studies have shown that increased sympathetic activation increases the risk of ventricular arrhythmias. Blocking sympathetic activation—either through surgery (sympathetic denervation, sympathetic ganglion resection) or pharmacologically (SGB, thoracic epidural anesthesia, or aggressive beta-blockade)—has been used to help treat arrhythmias that persist despite medical therapy. Although it is nondefinitive, its relative ease and noninvasive nature has meant that percutaneous SGB is being turned to as an option in recurrent VT. A recent review⁸ looked at the outcomes of 38 patients who underwent SGB for electric storm and showed that there is significant decrease in arrhythmia

burden (12.40 ± 8.80 episodes/day vs 1.04 ± 2.12 episodes/day; $P < .001$) and number of shocks (10.00 ± 9.10 shocks/day vs 0.05 ± 0.22 shocks/day; $P < .01$). Thus, SGB is a particularly promising option in the LVAD population, where excessive sympathetic activity comes from the need for inotropic support agents during the early postoperative period. In some cases, titration or alternative agents may be available, but often this is not enough to ameliorate the effects in patients who have a low threshold for VT. In cases such as this, SGB can be a useful option as a bridge to either resolution of VT from a low cardiac output state or more definitive therapy once clinically stable. Previously there have been concerns about the need to cease anticoagulation to perform this procedure, but our case adds to the literature^{9,10} that demonstrates it can be effectively and safely performed at the bedside without cessation of therapy.

Finally, if there is temporary success with SGB followed by recurrence of VT, then a sympathectomy should be considered, although this would be high risk in this patient population.

Conclusions

As LVAD technology continues to improve, and its uptake continues to grow, so too will the push for multidisciplinary approaches for VT in the LVAD population. We describe a case of surgical VT ablation at the time of LVAD implantation with 2 salient learning points. First, this is the first published case of severe myocardial edema occurring in the setting of extensive surgical ablation resulting in decreased left ventricular chamber size and recurrent VAD inflow obstruction. This required a unique combination of right ventricle support and reduction in LVAD speeds to provide adequate support while edema resolved, and we propose that as the best option in this situation. Secondly, we demonstrate the successful use of adjunct SGB to terminate ongoing VT in an LVAD patient. This therapy allowed bridging to a time when a definitive ablation is available and adds to the growing literature in this area.

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