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MYOCARDIAL IRRITATION FROM A LEFT VENTRICULAR ASSIST DEVICE RESULTING IN REFRACTORY VENTRICULAR TACHYCARDIA

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☐ **Abstract—Background: Due to an increasing prevalence of heart failure but a steady rate of heart transplantation, the number of left ventricular assist devices (LVADs) implanted is growing. These patients present to emergency departments (EDs) with a variety of complications from their implanted device as well as their baseline cardiomyopathy. One-third of patients will present with a dysrhythmia, the most common of which is ventricular tachycardia. Case Report: A 77-year-old man with nonischemic cardiomyopathy and HeartMate II LVAD presented with sustained ventricular tachycardia and 43 automatic implantable cardioverter-defibrillator (AICD) discharges. Due to left ventricular remodeling, ongoing diuresis, and positioning of his LVAD inflow cannula against his interventricular septum, a likely dysrhythmogenic foci, he quickly decompensated with sedation while in the ED. Why Should an Emergency Physician Be Aware of This?: Refractory ventricular tachycardia is a common dysrhythmia for LVAD patients and may lead to full cardiopulmonary arrest. Common strategies such as chest compressions are used only in limited scenarios, but medical management is possible. This should focus on resolution of the dysrhythmia and identification of the etiology, including possible mechanical compromise.** © **2018 Elsevier Inc. All rights reserved.**

☐ **Keywords—heart-assist devices/adverse effects; cardiovascular agents/therapeutic use; heart failure/surgery; critical care/methods; ventricular dysfunction; ventricular tachycardia**

INTRODUCTION

This case describes the emergent presentation and eventual cardiopulmonary arrest of a patient with a left ventricular assist device (LVAD) in refractory ventricular tachycardia. These complex heart failure patients are presenting more commonly to emergency departments (EDs), and providers must be aware of key strategies for evaluation and resuscitation. Despite receiving 43 internal defibrillator discharges, this patient's dysrhythmia did not resolve, and he rapidly deteriorated with sedation and amiodarone administration, requiring emergent external defibrillation. Through the adjustment of his LVAD speed, appropriate fluid resuscitation, and antidysrhythmic medications, the patient was stabilized. Imaging revealed that mechanical compromise and myocardial irritation due to malposition of the LVAD inflow cannula created this refractory dysrhythmia. This case emphasizes the importance of ED familiarity with the unique pathology of these patients, multidisciplinary care, and critical management steps for refractory ventricular tachycardia in LVAD patients.

CASE REPORT

A 77-year-old man with nonischemic cardiomyopathy and a HeartMate II LVAD (Thoratec Corporation, Pleasanton, CA) placed in 2014 presented to the ED with

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refractory ventricular tachycardia. During the 30-min critical care ambulance ride from his home to the LVAD center, he received 100 μg of fentanyl and 4 mg of midazolam due to extreme discomfort and numerous automatic implantable cardioverter-defibrillator (AICD) discharges. Ten minutes prior to arrival at our academic hospital ED, the patient acutely decompensated, with more frequent AICD discharges and sustained, symptomatic, ventricular tachycardia; however, on arrival at the hospital the patient was oriented and speaking in complete sentences. The critical care transport nurse reported that the patient had been in and out of rapid ventricular tachycardia, which would only briefly convert after an AICD discharge.

On rooming, the patient was placed on the monitor and external defibrillator. Initial vital signs showed a heart rate of 268 beats/min, oxygen saturation of 91% on a nonrebreather, and the patient was breathing 22 times per minute. He reported chest pain and dizziness. Due to the continuous flow of an LVAD pump, automatic blood pressure readings were inaccurate; mean arterial pressure (MAP) was obtained at 36 mm Hg using a Doppler and manual pneumatic cuff (goal MAP is 60–90 mm Hg) (1). His LVAD speed was set at 9000 rpm and the driveline site was clean, dry, and intact. Frequent low-flow and pulsatility index events were noted on device interrogation. Intravenous access was obtained, and fluids initiated. In the first 5 min while the patient was in the ED, he received a shock from his AICD approximately every 30–60 s and the rhythm strip showed persistent ventricular tachycardia (VT) ranging between 240 and 290 beats/min. Both the patient's worsening symptoms and his hypotension indicated the need for defibrillation of this now unstable dysrhythmia. In transport, the patient had received boluses of 100 μg of fentanyl and 4 mg of midazolam and tolerated it well. In preparation for external defibrillation, 300 mg of amiodarone i.v. was started, to run over 30 min, and an additional 50 μg of fentanyl and 2 mg of midazolam were given. Shortly after receiving these medications the patient suffered a cardiorespiratory arrest. A blood pressure was unattainable. Fluid resuscitation was accelerated by opening the bags to gravity and 0.4 mg of naloxone was administered with no response. The patient was emergently intubated and 1 mg of intravenous epinephrine was administered. Chest compressions were not initiated given his LVAD status and the unlikely event of pump malfunction being the etiology of his arrest. The patient, who remained in VT, was externally defibrillated with 300 J and converted to a narrow complex bradycardia. The patient's heart rate improved with 0.5 mg of atropine, however, MAP post cardioversion remained around 30 mm Hg and did not improve with 3 L of normal saline. As they had been prenotified of the patient's arrival, the multidisciplinary LVAD team, including Cardiology and Cardiothoracic Surgery, was at the bedside within 5 min of decompensation. Norepinephrine, epinephrine, and dobutamine drips were initiated by peripheral i.v. and a central line was placed. An arterial line was also placed for more accurate and timely blood pressure monitoring. This was very useful for both active monitoring and to decrease the substantial nursing load of taking multiple manual Doppler blood pressure measurements. With the arrival of the LVAD team, his VAD speed was turned down from 9000 to 8800 rpm to allow for more left ventricular filling and improved cardiac output. Subsequent MAPs ranged from 60–85 mm Hg. Bedside echocardiogram showed a stunned left ventricle (LV) and right ventricle (RV) with limited motion.

The patient was resuscitated in the ED for approximately 1 h prior to being transferred to the cardiac intensive care unit. Initial interrogation of his AICD showed 43 attempted defibrillations of 30–40 J over a period of 4 h, with multiple LVAD alarms for low flow. Review of the patient's chart showed that he had been seen multiple times in the ED and cardiology clinic over the preceding months for elevated MAP readings. Additionally, his antihypertensive medications had been increased 2 weeks prior to this event.

In the cardiac intensive care unit, fluid resuscitation was continued and the patient was weaned from dobutamine, epinephrine, and norepinephrine. Amiodarone drip was continued and over the next 2 days he received three 150-mg boluses of amiodarone for additional episodes of VT. One episode was also treated with 100 mg of i.v. lidocaine with almost immediate resolution of the dysrhythmia and no associated hypotension. Computed tomography (CT) of the chest demonstrated mechanical contact of the left ventricular septal wall and the LVAD inflow cannula; a potential nidus for ectopy (Figure 1). LVAD speed was further decreased to 8200 rpm and the patient's blood pressure medications altered to target a MAP of 80 mm Hg, theoretically increasing LV distension and filling. After adjustments, VT would recur with Valsalva maneuvers, but spontaneously resolve. He was discharged to home after 10 days of hospitalization, neurologically intact. He was not discharged on antidysrhythmic medications outside of his baseline heart failure regimen, which included a beta-blocker.

DISCUSSION

LVADs are becoming a more frequent treatment option for patients with heart failure from ischemic and nonischemic etiologies. Traditionally, these devices have been used for temporary support, serving as a bridge to transplantation. However, due to an increasing prevalence of heart failure and no parallel increase in transplant availability, LVAD implementation and duration of

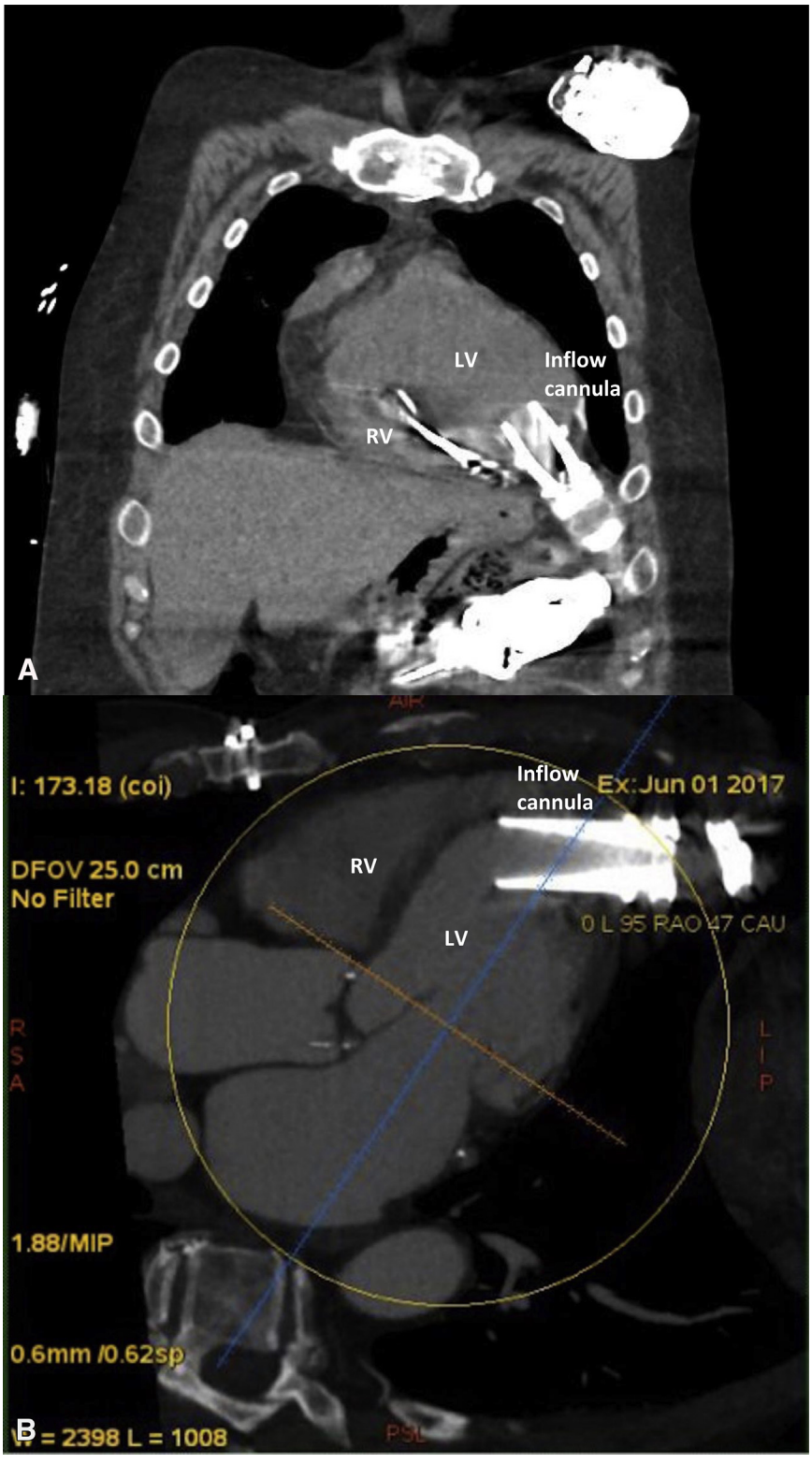

Figure 1. Cardiac computed tomography showing an appropriately positioned left ventricular assist device inflow cannula (A) compared with the malposition of the inflow cannula against the interventricular septum in the case patient (B). RV = right ventricle; LV = left ventricle.

therapy have also increased (2,3). LVADs are now both a bridging and a destination therapy and can improve survival time, quality of life, and functional status for patients with severe disability and symptoms while at rest (New York Heart Association Class IIIB or IV) (4).

With more devices present in the community, emergency physicians must be aware of commonly encountered complications. These include hemorrhagic complications such as gastrointestinal bleeding and intracranial hemorrhage, thrombotic complications such as stroke and mesenteric ischemia, and infectious complications, especially associated with the driveline of the device. In addition, patients with LVADs are particularly susceptible to ventricular dysrhythmias, with one meta-analysis identifying the prevalence of ventricular dysrhythmias among LVAD patients as 29%, with 85% of those dysrhythmias being sustained VT (5). This case provides the ideal opportunity to review pertinent LVAD physiology, discuss resuscitation techniques for acutely decompensated patients, and provide an up-to-date overview of antidysrhythmic medications for the termination of ventricular dysrhythmias.

The HeartMate II, a common LVAD currently in use, is an implanted continuous flow pump with an inflow cannula in the apex of the LV and outflow cannula in the proximal aorta. The pump is connected to the external controller via a driveline and powered by an external battery pack. The LVAD device produces a hum when the motor is functioning, which can be auscultated over the precordium. Emergencies can be divided into two primary categories: either the LVAD is working or the LVAD is not working. If there is no audible hum, the LVAD is not working and all connections should be checked and the battery replaced or connected to a wall power source. If the LVAD is working, assess for shock. As mentioned above, LVADs improve the functional status of patients with advanced heart failure, however, their heart failure is not reversed and LVAD patients can still suffer from fluid imbalance. Suction events occur when there is insufficient preload to the LV. This is easier to recognize in the setting of hypovolemia, where the left ventricular preload is inadequate, causing the inflow cannula to become blocked by the heart itself. Fluid resuscitation and further evaluation of the underlying etiology for the suction event is indicated in this situation. However, hypervolemia or other causes of right heart failure, when the RV is insufficiently providing preload to the LV, can also cause suction events (6). In the case reported above, the underlying etiology for suction events was actually left ventricular remodeling and subsequent inflow cannula mispositioning. Point-of-care ultrasound can be a useful tool in assessing ventricular size as well as function and overall fluid status.

Opinions about the appropriateness of chest compressions in LVAD patients differ between clinical situations and provider preferences. At our institution, chest compressions are discouraged if the underlying etiology for the cardiac arrest can be reversed and if the device is considered to be functioning properly. Conventional teaching suggests that chest compressions can result in displacement of the inflow and outflow cannulas, leading to catastrophic hemorrhage in these anticoagulated patients. Two studies with small cohorts of patients have recently shown that this fear may be exaggerated and that chest compressions may be safer than previously thought (7,8). Some algorithms for the treatment of unresponsive patients with no discernable MAP and other signs of hypoperfusion, such as delayed capillary refill, suggest performing compressions if there is no hum (9). Regardless, antidysrhythmic and vasoactive medications, fluid resuscitation, defibrillation, and LVAD setting adjustments should be pursued. For the patient in cardiopulmonary arrest, we recommend Advanced Cardiac Life Support guidelines for the administration of epinephrine and amiodarone. If the patient's MAP is detectable but low, norepinephrine is an acceptable choice as a first-line pressor agent, providing some assistance with cardiac contractility but primarily increasing systemic vascular resistance and thus improving cerebral blood flow. Dobutamine and epinephrine are suitable second-line agents if the patient requires additional circulatory support. This decision should be guided by clinician examination and bedside ultrasound to assess fluid status and cardiac contractility. Adjustments to the LVAD settings, as was done in this case, ideally should be attempted only under the direction of the patient's LVAD team, as they can lead to further decompensation, if physiology is miscalculated, and a higher risk of pump thrombosis.

After ED stabilization by a multidisciplinary team, our patient underwent formal transthoracic echocardiography (TTE) (Figure 2), which identified significant LV remodeling and a reduced LV end-diastolic dimension from 7.6 cm (pre-LVAD implantation) to 4.8 cm. This resulted in a marked reduction in end-diastolic volume and orientation of the inflow cannula toward the intraventricular septum. In general, echocardiography is a useful screen in decompensated LVAD patients and can detect pathologies such as pericardial effusion, aortic dissection, incomplete filling, worsening heart failure, intracardiac thrombus, or fusion of the aortic valve. Off-axis views are often necessary due to acoustic shadowing from the LVAD itself, and this may not be within the standard practice of an emergency physician. Due to the continuous flow of many of these devices, paradoxical movements may occur, and findings are best interpreted by a provider with LVAD expertise.

Although our patient's TTE hinted at the likely cause of his dysrhythmia, a cardiac CT scan identified malposition of the inflow cannula against the septal wall and significant remodeling of the myocardium, likely from irritation. This was the likely dysrhythmogenic focus and resulted in decreased flow through the cannula. When available, cardiac CT is important in evaluating cannula position and possible mechanical reasons for LVAD failure and alarming (10).

During his acute presentation, our patient experienced an exceptional 43 total AICD discharges for monomorphic ventricular tachycardia. The critical care transport team appropriately chose not to externally defibrillate the patient given that he was conscious, alert, oriented, and otherwise asymptomatic. By the time our patient arrived in the ED, he was symptomatic and hypotensive with an accelerating tachycardia. The monitor clearly showed that the patient was in continuous VT, without even a temporary

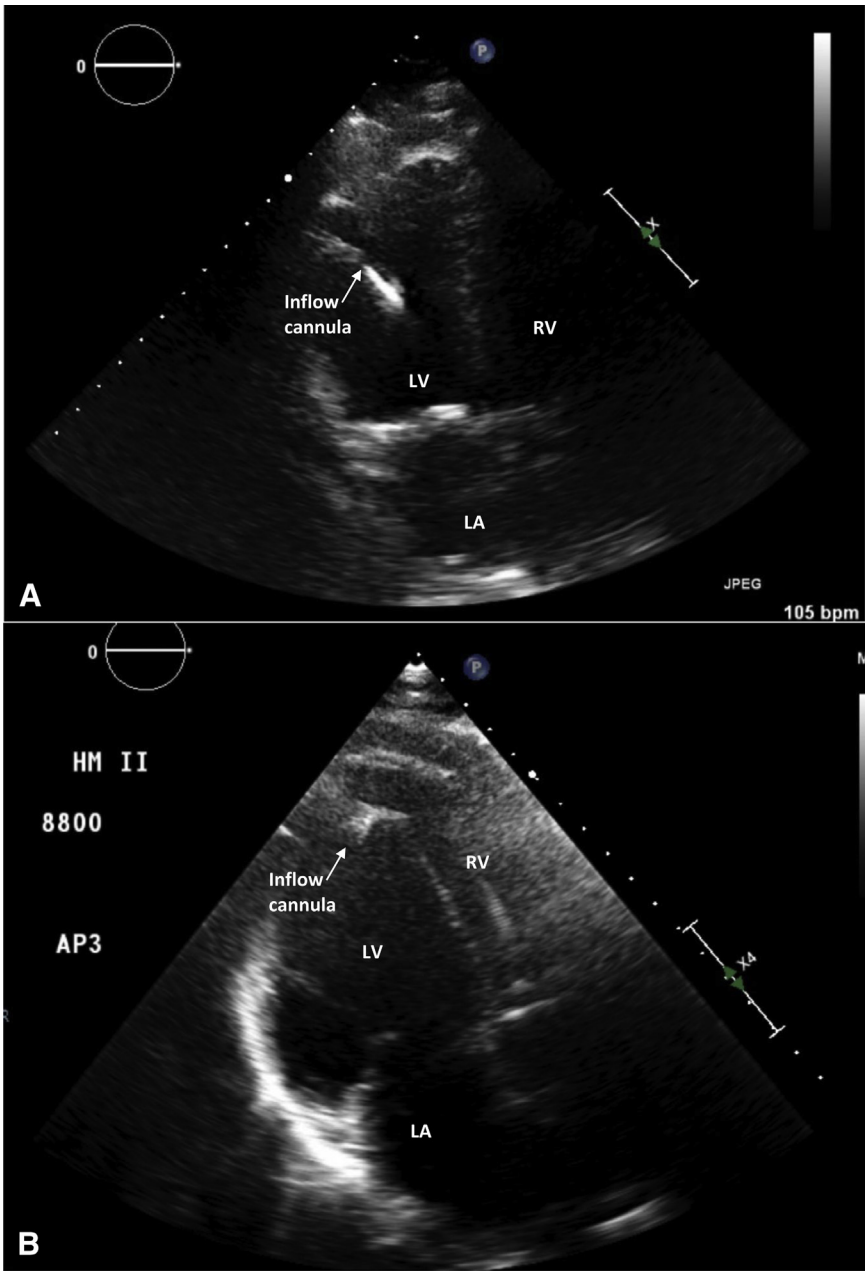

Figure 2. Transthoracic echocardiogram of the case patient post left ventricular assist device placement showing appropriate placement of inflow cannula (A) compared with immediately post ventricular tachycardia event with orientation of cannula toward the intraventricular septum (B). RV = right ventricle; LV = left ventricle; LA = left atrium.

reprieve after each AICD discharge. Although his LVAD was able to maintain a low blood pressure and some cerebral perfusion, the dysrhythmia seemed to be compromising his full cardiac output. Imaging also made clear the negative feedback loop between the malposition of the inflow cannula and the dysrhythmia: the cannula caused the dysrhythmia, which then further displaced the cannula and additionally compromised flow. This may not be the case for all LVAD patients, some of which are particularly dependent on the LVAD for cardiac output and can remain hemodynamically stable with what would otherwise be a terminal cardiac rhythm. Mechanisms of ventricular dysrhythmias are variable, and include suction events, inflow cannula malfunction, existing or developing myocardial scarring, variations in contractility, and new myocardial irritation. The most common risk factor for ventricular dysrhythmia post LVAD placement was an episode of ventricular dysrhythmia prior to LVAD placement (5). Another analysis identified both antecedent ventricular dysrhythmia and antecedent atrial fibrillation as risk factors (11). Whereas internal defibrillation transfers approximately 30–40 J of energy, an external defibrillator will deliver 200–300 J. Although, this is through the added impedance of the chest wall, there are no good data about the exact amount of energy that reaches the myocardium, and it likely varies widely given the patient's body habitus. When an AICD is unsuccessful it may also be due to lead displacement or dysfunction. External defibrillation should be attempted in all cases of unresolved, unstable dysrhythmia. In preparation for external defibrillation in the ED, the patient's AICD was deactivated with a magnet and he was given small doses of both midazolam for anxiolysis and fentanyl for pain control. Discussion among the ED providers and Cardiology led us to treat the patient using full-code doses of antidysrhythmics. Therefore, he received concurrent doses of midazolam, fentanyl, and amiodarone. Amiodarone has known vasodilatory effects as a result of its calcium channel and alpha-receptor blockade, in addition to its desired class I (sodium channel), class II (beta-blockade), and class III (potassium outflow) effects (12). Immediately after the administration of those three medications, the patient became unresponsive and suffered a cardiorespiratory arrest, requiring intubation. It is unclear which medication, individually or in combination, resulted in decreased cerebrovascular flow in that moment, but each of those medications could cause venodilation and thus, decreased RV filling, resulting in decreased LV filling and LVAD output. A combined strategy including successful defibrillation, fluid resuscitation to increase preload, antidysrhythmic medications, and decreasing his LVAD speed to increase LV filling, led to long-term stabilization and allowed the patient to be discharged home with a good neurologic outcome. All of these interventions focused on increasing the LV chamber size to move the inflow cannula off the septal wall and improve flow into the LVAD, thus improving overall cardiac output.

The ED provider has several anti dysrhythmic options for sustained ventricular tachycardia, including amiodarone, lidocaine, sotalol, procainamide, and electricity. LVAD patients are often excluded from studies comparing anti dysrhythmic agents, and like our patient, many of these patients will remain conscious during periods of sustained VT and do not fit neatly into "stable" or "unstable" categories. Data from prehospital literature have not shown superiority of one agent for patients with ventricular fibrillation or pulseless VT as measured by survival or favorable neurologic outcome (13,14). There was some very mild benefit of amiodarone over lidocaine in witnessed out-of-hospital arrests with good bystander cardiopulmonary resuscitation (14). It is possible that this result could be extrapolated to LVAD patients because the device provides some effective circulation in the absence of an organized rhythm, similar to effective cardiopulmonary resuscitation. In addition, although amiodarone is an indiscriminate antidysrhythmic, with effects across all classes, other agents may be more targeted to VT. Although all of these agents can cause hypotension, lidocaine would, in theory, have similar class I effects, targeting monomorphic VT without the vasodilatory and beta-blockade effects of amiodarone that may have reduced our patient's preload. While hospitalized, our patient received lidocaine for one episode of VT, which did immediately resolve. However, when examined in a larger sample of patients, lidocaine has shown poor rates of cardioversion (15). Research has also shown that procainamide may be more successful when converting VT, with some studies suggesting cardioversion rates of 50–80%, as opposed to 30–40% with amiodarone and fewer adverse events (16). Sotalol, with a very unique mechanism of action including both class II (beta-blockade) and class III (potassium outflow) effects—theoretically both slowing the intrinsic rate and limiting alternative conduction pathways by prolonging the refractory period—has also proven effective and, when compared with lidocaine in patients with refractory ventricular dysrhythmia, showed similar rates of dysrhythmia termination and survival to hospital discharge (17).

Upon review of available literature, one case report described patients who required surgical repositioning of their inflow catheter to resolve ventricular dysrhythmias long term (18). Other case reports describe radiofrequency ablation of dysrhythmogenic foci in both the immediate postoperative and emergency settings (19,20). Our patient did experience continued episodes of asymptomatic ventricular dysrhythmia, especially during Valsalva maneuvers. This increase in intrathoracic pressure and thus, decrease in LV filling, has been identified as a cause of intermittent asymptomatic ventricular dysrhythmia in

other patients with LVADs (21). On TTE, this patient also had paradoxical movement of the intraventricular septum toward the inflow cannula with Valsalva maneuvers or deep breathing, further exacerbating flow through the inflow cannula and causing acute reductions in inflow velocity. Repeat echocardiogram 2 weeks after discharge showed an improved LV end-diastolic dimension of 6 cm (4.8 cm at the time of the event) and increased space between the inflow cannula and the septum. Surgical repositioning was not considered in this case due to the high risk of the procedure and relatively successful medical management.

As the number of patients with LVADs continues to increase, opportunities for further study will hopefully guide clinical recommendations regarding antidysrhythmic medications for the treatment of ventricular dysrhythmias in LVAD patients. In addition, it is important for the ED provider to be aware of the basic physiology of these patients, as well as the major complications that can arise, including mechanical causes for ventricular dysrhythmias. Currently, the algorithm for managing ventricular dysrhythmias in LVAD patients is the same as with non-LVAD patients, calling for antidysrhythmic medications in the stable patient and electrocardioversion if the patient becomes unstable. There are also additional studies and techniques that can be helpful to adjust LV filling and output. For this reason, multidisciplinary support is essential in caring for these complicated patients, and EDs should have defined protocols on how to access assistance when needed.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Refractory ventricular tachycardia is a common dysrhythmia for LVAD patients and may lead to full cardiopulmonary arrest. Common strategies such as chest compressions should be used judiciously and under certain clinical situations, but medical management is possible and should focus on resolution of the dysrhythmia and identification of the etiology, including possible mechanical compromise. Appropriate LVAD adjustment and fluid resuscitation is almost always necessary, and relevant literature suggests that although amiodarone may be effective, procainamide, lidocaine, and sotalol may result in equal or improved rates of cardioversion, with a reduction in side effects.

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