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ASYMPTOMATIC SUSTAINED POLYMORPHIC VENTRICULAR TACHYCARDIA IN A PATIENT WITH A LEFT VENTRICULAR ASSIST DEVICE: CASE REPORT AND WHAT THE EMERGENCY PHYSICIAN SHOULD KNOW

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Abstract—Background: Left ventricular assist devices (LVADs) are a viable treatment option for patients with end-stage heart failure. LVADs can improve survival, quality of life, and functional status. The indications for LVAD placement to support left ventricular function are temporary support, a bridge to transplantation, or destination therapy. **Case Report:** A 61-year-old man with past medical history significant for advanced congestive heart failure from ischemic cardiomyopathy, status post LVAD (Heart-Mate II; Thoratec Corporation, Pleasanton, CA) placement 2009 as destination therapy, presented to the Emergency Department (ED) with implantable cardiac defibrillators firing four times that morning. While in the care of Emergency Medical Services, he was in ventricular tachycardia, and they gave him a bolus of amiodarone 150 mg intravenously prior to arrival in the ED. He was reportedly alert and oriented without any chest pain on arrival to the ED, where an electrocardiogram was obtained showing polymorphic ventricular tachycardia. **Why Should an Emergency Physician Be Aware of This?:** Emergency physicians must be familiar with the atypical presentations of potentially lethal dysrhythmias in this patient population. They must also be familiar with the major adverse events after LVAD implantation. These include device malfunction, cardiac dysrhythmias, bleeding, thromboembolism, neurological events, and infection. The causes of device malfunction can include thrombus formation with hemolysis, mechanical failure of the impeller, and driveline lead fractures with electric failure. Although time is critical in the heart failure patient with an LVAD failure or complication, expert

consultation with cardiology or the LVAD specialist should occur when possible. © 2016 Elsevier Inc.

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INTRODUCTION

Congestive heart failure (CHF) has been a challenge for health care providers for many decades. There has been an increasing prevalence, with more than 5.8 million cases in the United States and more than 23 million worldwide (1). Of these cases, it has been estimated that more than 100,000–200,000 have severe, refractory CHF that is not amenable to medical therapy, and this number is expected to increase over the coming years with an aging population and further use of cardiac support devices (1–3). One of these cardiac support devices, the left ventricular assist device (LVAD), is becoming a more popular option as “destination therapy” for end-stage CHF patients, given the limited number of available transplants, increasing comorbid conditions, and technological advances of the implantable devices (4,5).

The use of LVADs has developed from extensive research on practical forms of cardiac support devices,

and the device typically comes in one of three forms: the centrifugal pump, the volume displacement pump, and the axial-flow pump. The centrifugal pump uses a centrifugal force to generate nonpulsatile flow, produced by a conical-shaped rotor conforming to the cone-shaped housing. The volume displacement pump, unlike other devices, produces pulsatile flow by collecting blood within a chamber that is then externally compressed to mimic systole and diastole. The latter of the group, the axial-flow type pumps, includes the device used in our case presentation, the HeartMate II (Thoratec Corporation, Pleasanton, CA). In this axial-flow device, an impeller (a helical curved rotor blade fitted to a closed housing system) continuously draws blood in a nonpulsatile fashion from the inflow orifice (typically in the left ventricle [LV]) to the outflow orifice (typically in the aortic outflow tract). It is this lack of pulsatility that gives patients a benign “pulseless” feeling on physical examination and a continuous “hum” when auscultating the heart. The nonpulsatile flow seems to confer an advantage to long-term outcomes and complications compared to previous pulsatile flow devices, and the HeartMate II device itself, while connected to the heart in the mediastinum, is usually implanted into a preabdominal “pocket” or within the abdomen itself (6–8). The design of the HeartMate II allows the left ventricle to still function normally. This means some blood will exit the LV through the aortic valve secondary to the native heart function. The level of LV function and the compliance of the aorta will determine the pulsatility of the patient.

The indications for LVAD placement to support left ventricular function are generally classified into three categories. The first indication is for temporary support, a common example being a postviral myocarditis where a temporary LVAD is used until the heart recovers its systolic function. The second indication is viewed as a “bridge to transplantation,” where patients who qualify as candidates for transplant need mechanical left ventricular support while awaiting definitive surgical therapy. The third indication, a continuously growing indication, is termed “destination therapy.” This indication is for patients who are poor transplant candidates who have maximized medical management, and this was the indication cited for our patient (4,6).

Some well-known long-term complications post-LVAD implantation and their causes are listed in Table 1. These complications include but are not limited to: thromboembolism, anemia, platelet malfunction, gastrointestinal bleeding, and infection (8–10). An interesting complication increasingly reported in the literature is the development of sustained, malignant dysrhythmias. From a current literature search, it seems that many reported cases demonstrate sustained intervals of ventricular fibrillation (VF) or ventricular

Table 1. Long-term Complications Post LVAD Implantation

Potential Complication	Mechanism
1 – Thromboembolism	Implanted device serves as potential nidus for thrombus formation, requiring patients to be on anticoagulation
2 – Anemia	Chronic mechanical hemolysis from turbulent flow in device
3 – Platelet malfunction	Acquired Von Willebrand factor (VWF) deficiency from shearing of VWF multimers in device turbine
4 – GI bleeding	Nonpulsatile flow predisposes to development of arteriovenous malformations, particularly in the GI mucosa
5 – Infection	Implanted device, along with driveshaft connecting to battery, a potential site to support bacterial growth
6 – Dysrhythmias	Postsurgical scarring after device implantation alters conduction within myocardium

LVAD = left ventricular assist device; GI = gastrointestinal.

tachycardia (VT), with a majority of cases occurring perioperatively (11–16). Here, we present a case of newly documented polymorphic ventricular tachycardia (PVT) in a patient with an implantable cardiac defibrillator (ICD), 3 years post-LVAD placement.

CASE REPORT

A 61-year-old man with past medical history significant for advanced CHF from ischemic cardiomyopathy, status post-LVAD (HeartMate II) placement 2009 as destination therapy, presented to a high-volume tertiary care center with an associated heart hospital. He had a reported chief complaint that his ICD had fired repeatedly that morning, and he had noted an episode of chest pain radiating to his back prior to his ICD firing. He denied syncope or lightheadedness. At that time he called his cardiologist, a member of the LVAD response team, who recommended he come to the hospital via Emergency Medical Services (EMS). The presenting rhythm for EMS was reported VT, and the patient was treated with a bolus of amiodarone 150 mg en route by EMS. He was reportedly alert and oriented without any chest pain on arrival to ED, where electrocardiogram (ECG) (Figure 1) was taken and showed the patient was in PVT.

His past medical history was significant for a recent 11-day hospital stay for exacerbation of his heart failure (discharged 9 days prior to ED visit). During that stay, his inotropic support (dobutamine) was increased to 4 $\mu\text{g}/\text{kg}/\text{h}$ (patient with peripherally inserted central catheter [PICC] line), and he was aggressively diuresed an estimated 12 L. His home torsemide (loop diuretic) was increased to 60 mg twice a day, and other notable

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