

**Pedicle Flap Coverage for Infected Ventricular Assist Device Augmented with Dissolving
Antibiotic Beads: Creation of an Antibacterial Pocket**

Sasha Still MD¹, Rene P. Myers MD², Jose Tallaj MD³, Salpy Pamboukian MD³, William
Holman MD¹, James Davies MD¹, Charles W Hoopes MD¹, Erik Orozco-Hernandez MD¹

Institutions and affiliations:

University of Alabama at Birmingham, Birmingham, Alabama

¹ Department of Surgery, Division of Cardiothoracic Surgery

² Department of Surgery, Division of Plastic Surgery

³ Department of Medicine, Division of Cardiovascular Disease

Classification: Case report

Word Count: 1486

Running Head: Salvaging infected LVAD with antibacterial pocket

Keywords: VAD associated infection, LVAD, flap coverage, antibiotic beads, LVAD coverage,
left ventricular assist device

Financial Disclosure: The authors have no financial disclosures to report nor any affiliations with
industry products.

Conflict of interest: Authors have no potential conflicts of interest to disclose.

Funding source: No external funding was provided for this manuscript.

Corresponding Author:

Erik Orozco-Hernandez, MD

Division of Cardiothoracic Surgery, Department of Surgery, University of Alabama at
Birmingham, Birmingham, AL.

619 19th St S, Birmingham, AL 35249

Email: eorozcohernandez@uabmc.edu

Abstract

Infectious complications following left ventricular assist device implantation can carry significant morbidity and mortality. The main tenet of treatment is source control which entails local wound care, intravenous antimicrobial therapy, surgical debridement, and at times, soft tissue flap coverage. The mode of therapy depends on the severity, etiology, and location of infection as well as the clinical status of the patient. We describe a case of a 46 year-old male who underwent left ventricular assist device placement complicated by pump thrombosis, recurrent infection, and hardware exposure who was successfully treated with a novel method of staged, soft tissue reconstruction.

Introduction

Ventricular assist device (VAD) implantation is an established treatment for end stage heart failure refractory to medical therapy with >25 000 performed to date.¹ More than 2000 devices are placed annually and the majority are implanted for destination therapy.² Although VAD use is associated with improved functional status and quality of life, device related adverse events including hemorrhage, thrombosis, device failure, stroke, and infection can confer significant morbidity and mortality. Infection is a common complication of VAD placement and independent predictor of mortality.³ Infection rates range from 6-52%.^{1,4} According to the 9th annual INTERMACS report, at 48 months post-implantation approximately 50% of patients with experience a pump-related infection regardless of pump flow type.¹ The majority of infections are related to the device's driveline which is tunneled through the skin and often serving as a bacterial conduit.

Treatment of an VAD associated infection begins with antimicrobial therapy which is tailored to culture data and infection severity ranging from superficial cellulitis to deep abscess involving one or more device components. Operative debridement is often necessary to achieve source control and may leave the patient with exposed hardware, thus predisposing the patient to recurrent infection and/or bacterial colonization. To curtail the risk of recurrence, a variety of flap procedures have been utilized to protect the device after eradication of infection. A rectus abdominis myocutaneous flap is the most commonly used in this scenario.⁵⁻⁷ We obtain Plastic Surgery consultation in all cases requiring advanced reconstructive efforts. When rectus flap coverage is anticipated, the patient will undergo Computed Tomography Angiography (CTA) of the chest and abdomen to evaluate anatomy, driveline course, as well as to ensure patency of the superior epigastric arteries. We report a case of a 46 year-old male with a Heartware HVAD (Medtronic, Minneapolis, MN) who developed recurrent infection with hardware exposure and was treated successfully with a rectus abdominus flap and dissolving antibiotic beads.

Case Presentation

A 46 year-old Caucasian male with history of diabetes mellitus, hypertension, chronic kidney disease, morbid obesity, and stage D non-ischemic cardiomyopathy underwent implantation of Heartware HVAD (Medtronic, Minneapolis, MN) as destination therapy in October 2016. His postoperative course was routine. In February 2019 he developed pump thrombosis requiring pump exchange. At follow up, sanguinopurulent was draining from the incision. Computed tomography (CT) of the chest demonstrated inflammatory stranding with scattered foci of air concerning for infection. He was admitted for operative wound exploration and antibiotics. There was purulence around driveline and a sizable amount of residual clot within

the thoracotomy wound. Cultures were obtained from both sites. The wound was irrigated with Dakin's solution, partially closed, and a negative pressure wound vacuum device (KCI, San Antonio, Texas) placed. Intraoperative wound cultures grew oxacillin resistant *Staphylococcus Epidermidis* and methicillin resistant *Staphylococcus Aureus* (MRSA). Blood cultures were positive for oxacillin resistant *S epidermidis* alone. The patient underwent three additional washouts at which no signs of infection were noted. He was discharged with wound vac therapy, IV vancomycin (4-week course) and suppressive doxycycline.

Three months later he presented to clinic with left chest swelling. Repeat CT chest showed a left anterior chest fluid collection concerning for abscess (fig. 1). He was taken to operating room for repeat incision and drainage. Wound cultures were positive for MRSA. The recurrent MRSA infection refractory to antibiotic therapy and partial exposure of the pump (fig. 2) prompted plastic surgery consultation for soft tissue coverage. A left turnover rectus abdominis muscular flap (fig. 3), dissolving vancomycin bead implantation, and wound VAC placement was performed. During the case it was clear that his inferior epigastric artery was dominant over the superior epigastric artery, which was then clamped for 20 minutes to ensure the superior epigastric artery was capable of perfusing the muscle flap. The muscle was then inset with sutures and wrapped around the device. The remaining dead space was managed with composite calcium sulfate dissolving beads mixed with vancomycin powder. A week later the wound was noted to be healthy, granulating, and without evidence of infection and the patient underwent partial-thickness skin graft (fig. 4). He progressed well and eventually was transitioned to IV ceftaroline and discharged home with a 4-week course. He has had no ongoing wound issues or recurrent infection at 6 month follow up.

Discussion

Mechanical circulatory support with VAD is a cornerstone therapy for patients with end stage heart failure, either as a bridge to transplant or destination therapy. VAD-associated infections can be associated with high mortality and morbidity. At one year, the incidence of infection ranges between 25 to 28%, while at two years it approaches 50% regardless of INTERMACS profile.² During the first 3 months after VAD implantation, infection is the second most common complication while after 3 months it is the most common.²

The International Society for Heart and Lung Transplantation established a classification scheme for infection following VAD implantation and stratifies patients into three groups: VAD-specific infections, VAD-related infections, and non-VAD infections.⁷ VAD-specific infections involve components of the device such as pump, pocket cannula and / or driveline. VAD-related infections occur in patients with and without MCS but more frequently among those with a device (e.g. mediastinitis, endocarditis). Non-VAD infections are unrelated completely to the VAD itself (e.g. urinary tract infection). Hannan et al. went on to propose more comprehensive definitions of these categories based on a variety of microbiologic, histopathologic, radiologic, and clinical data.⁸ Based on these proposed schema, our patient meets criteria for both a deep VAD-specific infection of the percutaneous driveline and a deep VAD-specific infection of the pump pocket.

Deep infections involving device components present a unique challenge. They require surgical debridement(s) with or without negative pressure wound vac therapy in combination with aggressive intravenous antibiotic therapy, and when severe and persistent, may progress to device removal, exchange, or cardiac transplantation. Further challenge arises when debridement leaves hardware exposed. In this case, soft tissue coverage is often necessary and can be accomplished

by various flap procedures utilizing local soft tissue, rectus abdominus muscle, or omentum.⁵ Polymethylmethacrylate or calcium sulfate antibiotic beads have also been used for local control with success.⁶ Calcium sulfate beads are dissolvable and can be placed beneath the muscle flap without need for exchange, creating an antibacterial pocket which provides coverage and longer term antibiosis.

At our institution, deep VAD-specific infections are managed with aggressive operative debridement of non-viable or infected tissue, IV antibiotics, and negative pressure wound vac therapy with sponge change three times per week. When intraoperative cultures are negative we obtain plastic surgery consultation for soft tissue coverage. Typically, the rectus muscle is utilized if the superior epigastric artery is patent, otherwise omentum is chosen. In all cases we aim to salvage the device if possible.

Conclusions

VADs are an important adjunct in the treatment of patients with end-stage heart failure and their complications can be complex and cumbersome. VAD-associated infections can lead to significant patient morbidity and mortality. Individualized and aggressive early treatment of infection may improve long-term outcomes. In the short term, multidisciplinary methods lead to successful results.

Figures

Figure 1. CT chest demonstrating left chest deep pocket abscess.

Figure 2. Exposure of the driveline and hardware after debridement

Figure 3. Insetting sutures in place and exposed rectus abdominis muscle through paramedian incision.

Figure 4. Wound and muscle flap after skin grafting and subsequent negative pressure dressing removal.

Citations

- [1] Kormos RL, Cowger J, Pagani FD, et al. The Society of Thoracic Surgeons Intermacs database annual report: Evolving indications, outcomes, and scientific partnerships. *J Heart Lung Transplant*. 2019;38(2):114–126. doi:10.1016/j.healun.2018.11.013
- [2] Kirklin JK, Pagani FD, Kormos RL, et al. Eighth annual INTERMACS report: Special focus on framing the impact of adverse events. *J Heart Lung Transplant*. 2017;36(10):1080–1086. doi:10.1016/j.healun.2017.07.005
- [3] Han JJ, Acker MA, Atluri P. Left Ventricular Assist Devices. *Circulation*. 2018;138(24):2841–2851. doi:10.1161/CIRCULATIONAHA.118.035566
- [4] Topkara VK, Kondareddy S, Malik F, et al. Infectious complications in patients with left ventricular assist device: etiology and outcomes in the continuous-flow era. *Ann Thorac Surg*. 2010;90(4):1270–1277. doi:10.1016/j.athoracsur.2010.04.093
- [5] Jacoby A, Stranix JT, Cohen O, Louie E, Balsam LB, Levine JP. Flap coverage for the treatment of exposed left ventricular assist device (LVAD) hardware and intractable LVAD infections. *J Card Surg*. 2017;32(11):732–737. doi:10.1111/jocs.13230
- [6] Kretlow JD, Brown RH, Wolfswinkel EM, et al. Salvage of infected left ventricular assist device with antibiotic beads. *Plast Reconstr Surg*. 2014;133(1):28e–38e. doi:10.1097/01.prs.0000436837.03819.3f

[7] Kusne S, Mooney M, Danziger-Isakov L, et al. An ISHLT consensus document for prevention and management strategies for mechanical circulatory support infection. *J Heart Lung Transplant*. 2017;36(10):1137–1153. doi:10.1016/j.healun.2017.06.007

[8] Hannan MM, Husain S, Mattner F, et al. Working formulation for the standardization of definitions of infections in patients using ventricular assist devices. *J Heart Lung Transplant*. 2011;30(4):375–384. doi:10.1016/j.healun.2011.01.717