



Case Report

Mycobacterium chelonae, an ‘atypical’ cause of an LVAD driveline infection



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ABSTRACT

We describe the first patient with a left ventricular assist device (LVAD) driveline infection caused by *Mycobacterium chelonae* presenting with persistent infection despite conventional antibiotics. Treatment was successful with surgical debridement, driveline exit relocation, and a 4-month period of antibiotics. In the case of a culture-negative LVAD driveline infection, non-tuberculous mycobacteria should be considered. This case illustrates that multidisciplinary collaboration is essential in providing optimal care for LVAD patients.

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Introduction

In a selected group of patients with end-stage heart failure, a left ventricular assist device (LVAD) is implanted. The number of LVAD implantations is rising rapidly (de By et al., 2018; Kormos et al., 2019). One of the most common complications is a driveline infection. The incidence of LVAD-associated infection (including driveline infection) is around 28% in the first 12 months post-implantation (de By et al., 2018; Kormos et al., 2019). Early diagnosis and appropriate treatment is important in order to prevent an ascending infection, including LVAD pump infections, which are associated with increased morbidity and mortality (de By et al., 2018; Kormos et al., 2019; Simeon et al., 2017). This report describes the case of a patient with a rare cause of an LVAD driveline infection.

Case report

A 56-year-old Caucasian male with a history of ischemic cardiomyopathy, diabetes, and an LVAD (HeartMate II, Abbott)

since 2015 as a bridge to transplant, presented at the outpatient clinic with pain and a purulent discharge at the driveline exit 3.5 years post-LVAD implantation. There was no fever or redness, and his C-reactive protein (CRP) level was low at 0.6 mg/l. The patient was treated with oral levofloxacin 500 mg twice daily after a bacterial culture of the wound fluid was obtained. As a consequence, he was deemed not eligible for heart transplantation at that time.

Two weeks later, the patient returned to the outpatient clinic with a persistent purulent discharge from the driveline exit without systemic symptoms and with negative bacterial cultures. The patient was admitted and treated with intravenous antibiotics. Bacterial, mycobacterial, and fungal cultures were obtained and incubated for a prolonged period of time (14 days) for the identification of potential fastidious and/or slowly growing microorganisms. An ultrasound was performed, demonstrating a fluid collection around the driveline over a length of 15 cm. Subsequently, a positron emission tomography-computed tomography (PET-CT) scan was performed to evaluate the extent of the infection (Akin et al., 2018) and showed uptake in the subcutaneous tissue surrounding the LVAD driveline compatible with the ultrasound findings (Figure 1A). Since regular microbial cultures remained negative, the patient was discharged without antibiotics.

After 6 days, routine bacterial cultures became positive (Figure 1B). Matrix-assisted laser desorption/ionization (MALDI) identified *Mycobacterium chelonae*, and acid-fast staining showed the

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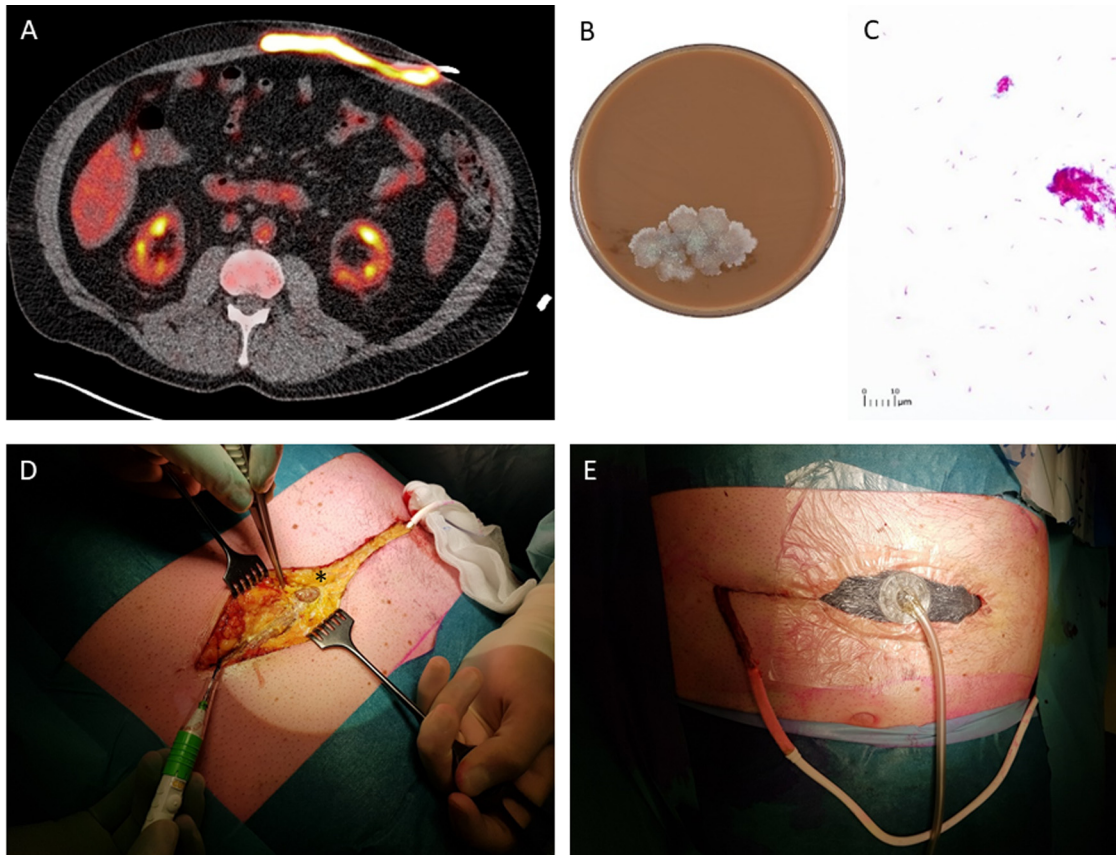


Figure 1. (A) Transverse PET-CT view showing clear ^{18}F -FDG uptake in the subcutaneous tissue surrounding the driveline. (B) Growth of *Mycobacterium chelonae* on chocolate agar. (C) Acid-fast acid staining showing the presence of mycobacteria. (D) Driveline relocation procedure; the asterisk (*) marks the infected part of the LVAD driveline. (E) Negative pressure wound therapy after driveline relocation.

presence of mycobacteria (Figure 1C). Reverse line blot hybridization confirmed the MALDI findings, showing a banding pattern fitting *Mycobacterium chelonae*/*Mycobacterium abscessus* complex/*Mycobacterium immunogenum*. Further molecular identification with partial gene sequencing of the 16S–23S rRNA internal transcribed spacer (ITS) region confirmed *M. chelonae* as the cause of the driveline infection. The patient was readmitted and started on imipenem 500 mg four times daily intravenously and clarithromycin 500 mg twice daily orally, based on susceptibility results. After 2 weeks of adequate antimicrobial treatment, surgical debridement was performed with relocation of the driveline exit to the right side of the abdomen. Negative pressure wound therapy (vacuum-assisted closure) was initiated at the location of the old, infected exit site (Figure 1D, E).

After surgical exploration, imipenem and clarithromycin were continued for one additional week, after which clarithromycin was combined with doxycycline 100 mg twice daily orally. The total duration of antibiotic therapy was 4 months. To date (8.5 months after stopping antibiotics), no infection recurrence has occurred and the patient is considered eligible for heart transplantation again.

Discussion

LVAD-associated infections are serious infections associated with high morbidity and mortality (de By et al., 2018; Kormos et al., 2019; Simeon et al., 2017). LVAD-associated infections can be divided into driveline infections and ascending or pump infections. Early diagnosis is important in order to initiate appropriate antimicrobial therapy with or without surgical debridement to prevent disease progression. Causative pathogens of LVAD-

associated infections are mainly *Staphylococcus aureus* and coagulase-negative staphylococci, which cause approximately 50% of infections, but *Pseudomonas aeruginosa* and *Enterobacteriaceae* infections have also been described (Simeon et al., 2017; Nienaber et al., 2013; Kusne et al., 2017). LVAD infections caused by non-tuberculous mycobacteria (NTM) are extremely rare. Recently, Nunez Breton et al. reported two cases of driveline infection caused by *M. abscessus* (Nunez Breton et al., 2018). *Mycobacterium chimaera* was reported in two patients in the context of a worldwide outbreak of infected heater cooler units (Balsam et al., 2017). Here, we describe the first patient with an LVAD driveline infection caused by *M. chelonae*.

NTM are ubiquitous environmental organisms found in soil, dust, and water. Together with *M. abscessus* complex and *Mycobacterium fortuitum*, *M. chelonae* is the most clinically relevant species within the group of rapidly growing NTM (RGM). RGM are characterized by the ability to grow on solid culture media within seven days, as opposed to the slowly growing NTM, which usually require more than seven days for growth. In general, it is recommended that cultures are obtained in specific liquid media as well as solid media when there is a clinical suspicion of NTM infection (Griffith et al., 2007). Furthermore, it is recommended that low temperature settings are used, as for several NTM, including *M. chelonae*, the optimal growth temperature is 30 °C. Interestingly, in our case, culture of the pus from the exit site on routine bacterial medium was positive on day six, while the liquid and solid cultures on specific mycobacterial culture media remained negative. This might be due to the decontamination procedure, which is routinely performed when non-sterile specimens are prepared for mycobacterial diagnostics. Although this

procedure is important for reducing contamination with other microorganisms, it may hamper the detection of NTM and RGM in particular (Griffith et al., 2007). Therefore, including prolonged incubation on non-specific bacterial culture media might increase the detection rate of NTM, as was the case for our patient. Once a mycobacterial culture is positive, proper species identification is crucial, since susceptibility and optimal treatment regimens differ between different NTM and between different RGM. In general, treatment requires a combination of antibiotic agents based on susceptibility results for at least four months in the case of skin and soft tissue infections (Griffith et al., 2007). Our patient was successfully treated with a 4-month combination antibiotic regimen, surgical debridement, and driveline relocation. Since NTM including RGM have the tendency to form biofilms, removing the foreign material in the case of NTM infections of prosthetic material seems prudent whenever feasible (Sousa et al., 2015).

Measures to prevent the recurrence of NTM infection could not be established, as specific risk factors (e.g., hot tub exposure or sauna visits) were not identified. Furthermore, such exposures are not allowed in this patient population.

In conclusion, in patients with an LVAD driveline infection unresponsive to conventional antibiotic regimens and with negative bacterial cultures on non-selective culture media, NTM and RGM in particular should be considered as a cause. Furthermore, the use of selective culture media including prolonged incubation in non-selective culture media at different temperatures might increase the yield. The combination of prolonged antibiotic therapy and surgical debridement and driveline relocation seems a prudent approach. Nevertheless, a multidisciplinary approach should be used in each case to determine the optimal management of LVAD-associated NTM infections.

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Ethical approval

No ethical approval was needed. Written consent was signed by the patient.

Conflict of interest

All authors declare no conflict of interest.

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