Cardiovascular device infections due to rapidly growing Mycobacteria: A review of cases at a tertiary care hospital

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ABSTRACT

Cardiovascular device infection due to rapidly growing mycobacteria (RGM) is rarely encountered in clinical practice. Due to the increasing number of indications and use of cardiovascular devices in an aging population, optimized management of these infections is of great importance. We report seven cases of RGM cardiovascular device infection. Three patients had left-ventricular assist device (LVAD) infections; two patients had cardiovascular implantable device (CIED) infections; and one had an aortic vascular stent infection. Specific cardiac valvular infection was not detected among any of the patients. All patients had a high number of comorbidities which limited some patients from receiving optimal combination antimicrobial therapy. The prognosis of cardiovascular device infections with RGM is guarded with only four patients still alive; however, the treatment approach for each patient varied considerably and often based on concurrent medical conditions, overall adjustments to goals of care, and specific patient preferences. Further analysis of cardiovascular device infections with RGM is warranted to establish a more systematic approach in successful management.

1. Introduction

RGM are opportunistic pathogens that are ubiquitous in nature [1,2]. They are tolerant to extreme pH, temperature conditions, and resistant to chlorine and aldehyde-based disinfectants [3,4]. Nosocomial outbreaks of invasive Mycobacterium abscessus complex (MABC) infection in postsurgical cardiac patients have been reported, with a common point source being hospital plumbing systems [5,6]. Although RGM pathogenesis is largely unknown, biofilm formation appears to be a significant factor in bacterial survival [4,7]. Common infections caused by RGM include respiratory, skin and soft tissues, and intravascular catheter infections [7]. Although invasive infections with RGM are reported among both immunosuppressed and immunocompetent hosts, a compromised immune system and intravascular catheters constitute major risk factors [8]. Cardiovascular (CV) device infections with RGM are rare, and current literature is limited to only case reports [9–16]. In this report, we present seven cases of RGM CV device infection and describe treatment approaches and outcomes in order to provide more data on strategies to manage these challenging situations.

2. Methods

We retrospectively reviewed all the RGM isolates from any sterile sources that were obtained from patients seen at Mayo Clinic in Minnesota, Florida and Arizona from November 2011 through March 2021. We included only adult (≥18 years old) patients with CV device infections. The term “CV device infection” included infections of CV implantable electronic device (CIED), left ventricular assist device (LVAD) and prosthetic vascular graft/stents.

Definitions for LVAD driveline, pump, cannula, and pocket infection were adapted from a consensus guideline suggested by The International Society for Heart and Lungs Transplantation (ISHLT) [17]. Mayo Clinic CIED infection classification criteria were implemented to define and categorize CIED infections [18].

Mycobacterial growth on solid media or broth suspension was identified using macroscopic and microscopic morphology, matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS), or 500 bp 16S rDNA gene sequencing as appropriate. The broth microdilution technique was used to perform antimicrobial susceptibility testing according to CLSI guidelines [19]. Institutional
Review Board approval was obtained for this study.

3. Results

3.1. Overview of cases

3.1.1. Case 1

A 38-year-old male underwent implantation of a HeartMate-II™ LVAD (Thoratec Corporation, Pleasanton, California) for dilated cardiomyopathy (DCM). Nineteen months later, he developed a methicillin-susceptible Staphylococcus aureus (MSSA) driveline infection and subsequent Mycobacterium abscessus subspecies (subsp.) bolletii catheter-related bloodstream infection (CRBSI), which prompted catheter removal. The M. abscessus subsp. bolletii CRBSI was treated with combination antimicrobial therapy, including amikacin, cefoxitin, and clarithromycin based on initial susceptibilities (Table 2, isolate 1.1). The patient initially defervesced and repeat blood cultures were negative. He subsequently developed amikacin-induced ototoxicity, and nephrotoxicity, and clarithromycin-related GI intolerance. Therefore, amikacin was stopped on day 42 of therapy, and clarithromycin was switched to azithromycin on day 77 while cefoxitin was continued. Five months after the initial CRBSI, the patient again developed MABC bacteremia and LVAD infection was suspected. It was determined that the patient was noncompliant with his antimicrobial program. Therapy was changed to minocycline, azithromycin, and linezolid (Table 2, isolate 1.2). Due to linezolid induced pancytopenia, the treatment was further modified to clofazimine, azithromycin, and minocycline. The patient was deemed not a candidate for a heart transplant due to persistent mycobacterial infection and a high risk of transplant-associated mortality. A pump exchange of LVAD was considered, but the final plan was to pursue only medical management due to a high risk of recurrence. He continued to have interruptions in antimicrobial therapy due to non-compliance, which resulted in a third episode of MABC bacteremia. An abdominal ultrasound revealed a small amount of fluid collection around the LVAD wires that was felt to be infected. Eventually, the patient opted for palliative care and was lost to follow-up.

3.1.2. Case 2

A 79-year-old male presented with bleeding and serous fluid discharge from his LVAD driveline exit site after falling into nearby bushes while trying to stand up from his wheelchair. The incident occurred six years after HeartMate II™ LVAD implantation as a

| Case | Age (y) | Sex | Diagnosis | Isolated species | Blood culture | Removal of device | Antimycobacterial therapy, d | Adverse reaction | Outcome |
|------|---------|-----|-----------|-----------------|---------------|-----------------|---------------------------|-----------------|---------|
| 1    | 38/M    |     | LVAD driveline and pump infection | Mycobacterium abscessus subsp. bolletii | Positive | No | AMK 78 d; MXF 162 d, LZD 30 d, MIN 38 d, FOX 157 d, AZM 196 d, CLR 77 d, AMK 42 d, TGC 46 d, CLO 60 d | Amikacin-induced ototoxicity and nephrotoxicity, linezolid induced pancytopenia with GI bleeding | Died |
| 2    | 79/F    |     | LVAD driveline infection | Mycobacterium abscessus complex | Negative | No | None | None | Alive |
| 3    | 84/F    |     | LVAD driveline infection | Mycobacterium chelonae | Negative | No | AZM 90 d | None | Alive |
| 4    | 70/M    |     | Infection of retained epicardial pacing wires | Mycobacterium abscessus complex | Negative | Yes | None | None | Alive |
| 5    | 44/F    |     | AICD infection | Mycobacterium fortuitum complex | Positive | Yes | CLR 10 d, IPM 196 d, AMK 78 d, MXF 162 d, TGC 77 d, LVX 24 d | Amikacin-induced ototoxicity, moxifloxacin-induced GI intolerance | Alive |
| 6    | 67/M    |     | Pacemaker pocket infection | Mycobacterium abscessus complex | Negative | Yes | AZM 180 d | None | Died |
| 7    | 60/M    |     | Aortic vascular stent infection | Mycobacterium abscessus subsp. massiliense | Negative | Yes | CLR 180 d, AZM lifelong | None | Alive |

1 = Patient opted for palliative care; outcome is unknown due to lost to follow up. AMK amikacin; AZM azithromycin; CLO clofazimine; CLR clarithromycin; FOX cefoxitin; IPM imipenem; LVX levofloxacin; LZD linezolid; MFX moxifloxacin; MIN minocycline; TGC tigecycline.

AAA abdominal aortic aneurysm; AF atrial fibrillation; AICD automatic implantable intracardiac defibrillator; AS aortic stenosis; AVR aortic valve replacement; CHF congestive heart failure; CKD chronic kidney disease; CRBSI catheter-related bloodstream infection; DCM dilated cardiomyopathy; GI gastrointestinal bleeding; HTN hypertension; HLP hyperlipidemia; OSA obstructive sleep apnea; PEA pulseless electrical activity; PPM permanent pacemaker; Rtx radiotherapy; VSD ventricular septal defect; VT ventricular tachycardia.

Table 1: Overview of Patient Population with Cardiovascular Device Infection due to Rapidly Growing Mycobacteria.
destination therapy for idiopathic DCM. He had multiple other comorbidities, as described in Table 1. After the fall, the patient had persistent fluid discharge and pain from the LVAD driveline site. Multiple swab cultures from drainage fluid grew MABC. He opted for dismissal to home with hospice care. No additional diagnostic work-up or antimicrobial treatment was pursued, and the patient passed away five days later.

3.1.3. Case 3
An 84-year-old male initially presented with brown discharge from LVAD driveline exit site associated with redness 33 months after the implantation of HeartMate-II™ LVAD. Position emission tomography/computed tomography (PET/CT) scan revealed an fluorodeoxyglucose (FDG)-avid soft tissue thickening in the subcutaneous fat along the exit site tract. Bacterial cultures from the exit line drainage grew M. chelonae. In accordance to patient’s wishes, and due to concerns for drug toxicity and multiple medical co-morbidities, he was started on azithromycin monotherapy. Three months into the therapy, patient self-terminated azithromycin and opted for observation with periodic clinic visits and CT scans. Interestingly, he remained asymptomatic during 2 years of monitoring.

3.1.4. Case 4
A 70-year-old female underwent minimally invasive aortic valve replacement via right anterior thoracotomy approach for aortic stenosis. Two months after the surgery, she presented with a dehiscence of the surgical incision. Swab cultures from the drainage grew MABC. She underwent incision and drainage with removal of three out of four pacing wires. One of the atrial wires had retracted into the chest wall underwnt underwent incision and drainage with removal of three out of four replacement via right anterior thoracotomy approach for aortic stenosis. Three months later, she had an event which was later changed to azithromycin because of nausea, for at least one days after the initial wound exploration, the wound developed a sinus tract which extended into the pleural space. Upon entering the pleural space, the remnant 4th temporary pacing wire was identified and therefore not removed. Intraoperative right chest tissue cultures grew - MABC (Table 2, case 4). The patient declined antimicrobial treatment and opted for outpatient observation and localized wound care. Twenty-one days after the initial wound exploration, the wound developed a sinus tract. Subsequently, the patient underwent a resection of the sinus tract which extended into the pleural space. Upon entering the pleural space, the remnant 4th temporary pacing wire was identified and removed entirely. Over the next two years from further tissue debride-ment and removal of the 4th wire, the patient’s chest wall wound eventually completely healed without antimicrobial therapy.

3.1.5. Case 5
A 44-year-old female with history of Tetralogy of Fallot underwent bioprosthetic pulmonic valve replacement and automatic implantable cardioverter defibrillator (AICD) implantation for progressive right ventricular enlargement due to pulmonary regurgitation and symptomatic ventricular arrhythmias in 2015. In 2018, she presented with left shoulder pain, nausea and vomiting. Blood cultures were positive for M. fortuitum complex (Table 2, case 5). Transesophageal echocardiogram demonstrated linear strands attached to the ICD leads. There was no evidence of valvular involvement. A combination regimen with amikacin, imipenem, and moxifloxacin was started empirically. The patient underwent complete AICD extraction (generator and wire leads) along with capsulectomy, and cultures from the AICD pocket grew M. fortuitum complex. Repeat blood cultures were negative. The patient continued combination therapy (Table 1) that also included tigecycline and linezolid at different time intervals, based on antimicrobial susceptibilities and tolerance. Reimplantation of a new right-sided AICD was performed six months after the extraction. The following day, antibiotics were discontinued. She was followed up with monthly mycobacterial blood cultures for three months. The patient remains free of infection two years after complete AICD extraction and delayed reimplantation.

3.1.6. Case 6
A 67-year-old male presented to the cardiology clinic for a second opinion regarding management of a non-healing and intermittently draining surgical wound from a pacemaker placement (PPM) 14 months earlier. Physical examination revealed no erythema, warmth or tenderness to gentle palpation over the PPM pocket. He was treated with multiple courses of empiric oral antibiotics without resolution of the drainage. Due to concern for pocket infection, the patient underwent complete extraction of the pacemaker. Intraoperatively, there was no gross purlulence; therefore, right-sided pacemaker reimplantation was performed during the same surgery (single staged procedure). Cultures from the pacemaker pocket grew Corynebacterium acellens and MABC. The patient initially was started on IV tigecycline and clarithromycin, which was later changed to azithromycin because of nausea, for at least three months and later discontinued for unclear reasons. There was no evidence of recurrent infection over the next three years. Unfortunately, the patient passed away due to heart failure complications.

3.1.7. Case 7
A 60-year-old female with history of endovascular stent repair of an abdominal aortic aneurysm presented with lumbar back pain. Two months prior to this presentation, she was hospitalized due to a left psoas abscess with Salmonella sp. for which she had a drain placed. She completed six weeks of levofloxacin and was transitioned to trimethoprim-sulfamethoxazole. Due to suspected aortic stent involvement, she underwent axillo-femoral bypass and excision of the infected aortic stent, polytetrafluoroethylene graft placement. Mycobacterial cultures from both the aortic wall and stent grew one colony of M. abscessus subsp. massiliense. Because of both patient and provider preferences, and based on other co-morbidities, an atypical approach of monotherapy with clarithromycin was prescribed. One month after the excision of the infected stent, her course was further complicated with aortic stump rupture in the setting of Candida albicans fungemia. The patient required multiple operations for further aortic debridement and extra-anatomic bypass with a multi-limbed graft from suprarenal aorta to abdominal vessels. She continued on lifelong chronic suppression for

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**Table 2**

RGM Antimicrobial Resistance Profiles.

| MIC, CLSI interpretation | AMK | FOX | CLO | CLR | IPM | SXT | LZD | MFX | CIP | AZM | MIN | TGC | DOX |
|--------------------------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| **Case 1 (M. Bolellii)** |     |     |     |     |     |     |     |     |     |     |     |     |     |
| Isolate 1.1              | <8, S 32, I | <0.5, S ND | ND | 8, I | ND | 4, S | >4, R ND | <16, S 1, S | <0.25, 8, I |
| Isolate 1.2              | 16, S 128, R <0.5, S 2, S | 64, R >8/152, R 32, R 4, R | >4, R ND | <16, S 8, S 0.5 ND |
| **Case 2 (M. Abscessus complex)** | 16, S 32, I ND | 1, S 16, I >8/152, R 32, R >8, R 4, R ND | <8, R 0.5 >16, R |
| **Case 3 (M. Chelonae)** | 16, S >128, R ND 0.5, S 16, I >8/152, R 16, I 8, R >4, R ND >8, R 0.12 >16, R |
| **Case 4 (M. Abscessus complex)** | 8, S 128, R NR, S >16, R 32, R >8/156, R 32, R >8, R 4, R ND >8, R 1.0 >16, R |
| **Case 5 (M. Fortuitum complex)** | 4, S 128, R 0.25, S 8, R 8, I 4/76, R >32, R 0.25, S 0.25, S ND ND >8, R 0.06 >16, R |
| **Case 6 (M. Abscessus group)** | 16, S 32, I ND 1, S 8, I >8/152, R 32, R 8, R 4, R ND >8, R 0.25 >16, R |
| **Case 7 (M. Massiliense)** | 16, S 32, I ND 1, S 16, I >8/152, R 32, R >8, R 4, R ND >8, R 0.25 >16, R |

AMK amikacin; AZM azithromycin; CLO clofazimine; CLR clarithromycin; FOX cefoxitin; IPM imipenem; LVX levofloxacin; LZD linezolid; MFX moxifloxacin; MIN minocycline; TGC tigecycline; DOX doxycycline; SXT trimethoprim-sulfamethoxazole; NR not reported; ND not done; R resistant; I intermediate; S sensitive; MIC minimum inhibitory concentration.
polymicrobial graft infection with fluconazole, azithromycin and doxycycline.

4. Discussion

CV device infections due to RGM remain a rare entity [9,10]. Current data is limited to case reports although number of cases diagnosed during the past two decades is rising [9-16,20-31]. This could be due to both an increase in the number of CIED implanted in an aging population [32,33], as well as, an increased clinical awareness of extrapulmonary RGM infections. An optimal treatment approach for RGM CV device infections has not yet been established and generally requires device extraction with concurrent and often prolonged combination antimicrobial therapy. Current treatment practice for pulmonary, skin and soft tissue, and catheter RGM infections is based on a number of retrospective case series and published clinical experience [7]. In this report, we describe seven cases with complex RGM CV infections with challenging management. Only two out of seven patients received combination antimicrobial therapy for RGM infection along with device removal. The other patients received more unconventional and simplified therapies, usually because of patient preferences, medical comorbidities and often adjusted overall goals of care with a more palliative focus. Patient intolerance to select combinations of antimicrobial therapy directed against RGM was a common occurrence and often complicating factor in the continuation of therapy.

CIED infections can involve leads and/or generator pocket site (battery and electronics) [18]. Regardless of the degree of involvement, once a segment of the system is infected, sterilization of the implanted device is not feasible and it should be entirely removed [18,33]. Antimicrobial therapy is considered as adjunctive in the management of CIED infection and shouldn’t be delayed. Involvement of the cardiac valves should be assessed as additional surgery may need to be considered. The duration of antimicrobial therapy for CIED infections depends on the extent of infection, the presence of bloodstream infection, and type of causative organism [33]. M. fortuitum is the most common RGM that has been implicated in CIED infections in previous case reports [11,12,28,30]. Cure of the infection and successful reimplantation have been described in previous cases with complete device removal and appropriate antimicrobial therapy; duration of antimicrobial treatment was quite varied, from four weeks to six months in cases without valvular involvement or bacteremia [12,28,30]. Interestingly, none of the patients in our series demonstrated cardiac valvular infection with RGM. Our two cases with CIED infection had reimplantation of the device with no recurrence of the RGM infection; one of which had reimplantation shortly after the extraction, and the other reimplanted six months after. Case four had infection of retained epicardial wire with MABC similar to one prior case report [14].

LVAD infections can occur in 9–48% of patients within 6–8 months from implantation [34]. We found two small case series describing a total of five patients with MABC LVAD infection [31,34]. Four cases had driveline exit site infection and one had pump infection. In the first series, the device was retained in all three patients. They all exhibited intolerance or side effects to the antibiotic therapy, as commonly encountered in our case series, and eventually two opted for comfort care while one patient continued on lifelong suppression [34]. In the second series, one patient had driveline unroofing along with combination antibiotics until his blood and drainage cultures turned negative after which he underwent device removal and heart transplantation [31]. There was no clinical evidence of infection recurrence. The other patient underwent device exchange along with antibiotics and his follow up cultures remained negative [31].

In our cohort, all patients had high number of comorbidities. Among the four out of seven patients still alive, three of them had complete device removal. All three patients that had the device retained, eventually declined antimicrobial therapy and two of them pursued hospice care. We also acknowledge that most of our patients did not receive typical combination antimicrobial therapy as recommended in prior NTM published recommendations [35–37] which often reflected patient wishes and more palliative goals of care. We believe that further descriptive and comparative studies are warranted to establish guidance in management of nontuberculous mycobacteria CV device infection.

5. Conclusion

RGM CV device infections are associated with high morbidity and mortality. All of our patients had a number of medical co-morbidities. Currently there is not consensus treatment approach, but device removal is recommended when possible, and concurrent antimicrobial therapy may need to be individualized for each patient. Further assessment is needed with these complex infections.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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