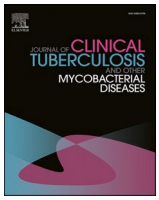
Journal of Clinical Tuberculosis and Other Mycobacterial Diseases 26 (2022) 100296

Contents lists available at ScienceDirect

Journal of Clinical Tuberculosis and Other Mycobacterial Diseases

journal homepage: www.elsevier.com/locate/jctube



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

Isin Yagmur Comba [a,*], Supavit Chesdachai [a], Hussam Tabaja [a], Maryam Mahmood [a], Sharon Deml [b], Nancy L. Wengenack [b], John W. Wilson [a]

[a] Division of Infectious Diseases, Department of Medicine, Mayo Clinic, Rochester, MN, USA
[b] Division of Clinical Microbiology, Department of Laboratory Medicine, Mayo Clinic, Rochester, MN, USA

ARTICLE INFO

Keywords:
Cardiovascular device infection
Rapidly growing mycobacteria
Left ventricular assist device
Case series

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 microscopicmorphology, 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

* Corresponding author at: Division of Infectious Diseases, Department of Medicine, 200 First Street SW, Rochester, MN 55905, USA.
E-mail address: Comba.Isin@mayo.edu (I.Y. Comba).

https://doi.org/10.1016/j.jctube.2022.100296

Available online 7 January 2022

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 subsequqent *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. The therpay 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 theapy 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

Table 1
Overview of Patient Population with Cardiovascular Device Infection due to Rapidly Growing Mycobacteria.

Case	Age (y)/ sex	Relevant comorbidities	Diagnosis	Isolated species	Blood culture	Removal of device	Antimycobacterial therapy, d	Adverse reaction	Outcome
1	38/ M	Idiopathic DCM, PEA s/p AICD placement, CHF s/p HeartMate II implantation as destination therapy due to noncompliance, CRBSI with *Mycobacterium bolletii* 19-months after the device implantation	LVAD driveline and pump infection	*Mycobacterium abscessus* subsp. *bolletii*	Positive	No	LZD 30 d, MIN 38 d, FOX 157 d, AZM 196 d, CLR 77 d, AMK 42 d, TGC46 d, CLO 60 d	Amikacin-induced ototoxicity and nephrotoxicity, linezolid induced pancytopenia with GI bleeding Clarithromycin GI intolerance	Unknown[1]
2	79/ M	Non-ischemic DCM, AF, CKD stage IV, VT s/p AICD, CHF s/p HeartMate II implant as a destination therapy, OSA (not treated), recurrent GI bleeding due to AVM	LVAD driveline infection	*Mycobacterium abscessus* complex	Negative	No	None	None	Died
3	84/ M	Ischemic CHF s/p HeartMate II LVAD implantation as a destination therapy, hypertension, lumbar spondylosis	LVAD driveline infection[2]	*Mycobacterium chelonae*	Negative	No	AZM 90 d	None	Alive
4	70/F	Aortic stenosis and regurgitation s/p minimally invasive AVR, HTN, hypothyroidism	Infection of retained epicardial pacing wires	*Mycobacterium abscessus* complex	Negative	Yes	None	None	Alive
5	44/F	Tetralogy of Fallot s/p left Blalock-Taussig shunt, VSD repair, pulmonary valvotomy and bioprosthetic pulmonary valve replacement	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	Hodgkin's lymphoma s/p Rtx to mediastinum, AS s/p mechanical AVR, sick sinus syndrome s/p PPM	Pacemaker pocket infection	*Mycobacterium abscessus* complex	Negative	Yes	AZM 180 d	None	Died
7	60/F	AAA s/p stent placement in 2014 with revision in one later, HTN, HLP	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; AVM arteriovenous malformation; AVR aortic valve replacement; CHF congestive heart failure; CKD chronic kidney disease; CRBSI catheter-related bloodstream infection; DCM dilated cardiomyopathy; GIB 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.

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 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 and was entirely covered by granulation tissue. Despite using fluoroscopic guidance, the fourth wire could not be initially located 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 debridement 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 pulmonary 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 purulence; therefore, right-sided pacemaker reimplantation was performed during the same surgery (single staged procedure). Cultures from the pacemaker pocket grew *Corynebacterium accolens* 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 supraceliac aorta to abdominal vessels. She continued on lifelong chronic suppression for

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. Bollettii)*													
Isolate 1.1	<8, S	32, I	<0.5, S	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	<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	>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 doxyxycline.

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 co-morbidities and often adjusted overall goals of care with a more palliative focus. Patient intolerance to select combinations of antimicrobial therpay directed against RGM was a common occurrence and often complicating factor in the continuation of therpay.

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 thepray 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 comperative 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.

References

[1] Runyon EH. Identification of mycobacterial pathogens utilizing colony characteristics. Am J Clin Pathol 1970;54(4):578–86.
[2] Falkinham JO. Environmental sources of nontuberculous mycobacteria. Clin Chest Med 2015;36(1):35–41.
[3] De Groote MA, Gibbs S, de Moura VCN, Burgess W, Richardson K, Kasperbauer S, et al. Analysis of a panel of rapidly growing mycobacteria for resistance to aldehyde-based disinfectants. Am J Infect Control 2014;42(8):932–4.
[4] Carson LA, Petersen NJ, Favero MS, Aguero SM. Growth characteristics of atypical mycobacteria in water and their comparative resistance to disinfectants. Appl Environ Microbiol 1978;36(6):839–46.
[5] Baker AW, Lewis SS, Alexander BD, et al. Two-phase hospital-associated outbreak of Mycobacterium abscessus: investigation and mitigation. Clin Infect Dis 2017; 64: 902–11.
[6] Baker AW, Maziarz EK, Lewis SS, et al. Invasive Mycobacterium abscessus Complex Infection after Cardiac Surgery: Epidemiology, Management, and Clinical Outcomes. Clin Infect Dis 2020.
[7] Brown-Elliott BA, Philley JV: Rapidly Growing Mycobacteria. Microbiol Spectr 2017, 5(1).
[8] Han XY, Dé I, Jacobson KL. Rapidly growing mycobacteria: clinical and microbiologic studies of 115 cases. Am J Clin Pathol 2007;128(4):612–21.
[9] Jain H, Gada K, Yadava SK, Paolino K, Eranki A. Cardiovascular implantable electronic device infection with rapidly growing mycobacteria. Proc (Bayl Univ Med Cent) 2019;32(3):390–1.
[10] Al-Ghamdi B, Widaa HE, Shahid MA, Aladmawi M, Alotaibi J, Sanei AA, et al. Cardiac implantable electronic device infection due to Mycobacterium species: a case report and review of the literature. BMC Res Notes 2016;9(1). https://doi.org/10.1186/s13104-016-2221-1.
[11] Zhu J, Yang Q, Pan J, Shi H, Jin B, Chen Q. Cardiac resynchronization therapy-defibrillator pocket infection caused by Mycobacterium fortuitum: a case report and review of the literature. BMC Cardiovasc Disord 2019;19(1):53.
[12] Orellana-Barrios M, Sotello Aviles DA, Oyenuga O, Nugent K. Implantable cardiac defibrillator infections: the emerging importance of Mycobacterium fortuitum. BMJ Case Rep 2017, 2017..
[13] Schlossberg D, Aaron T. Aortitis caused by Mycobacterium fortuitum. Arch Intern Med 1991;151(5):1010–1.
[14] Cutay A, Horowitz H, Pooley R, Van Horn K, Wormser G. Infection of epicardial pacemaker wires due to Mycobacterium abscessus. Clin Infect Dis 1998;26(2): 520–1.
[15] Marion MD, Swanson MK, Spellman J, Spieth ME. Femoropopliteal prosthetic bypass graft infection due to Mycobacterium abscessus localized by FDG-PET/CT scan. J Vasc Surg 2009;50(4):907–9.
[16] Vail G, Kohler R, Steiner F, Donepudi R. Successful treatment of Mycobacterium fortuitum prosthetic valve endocarditis: case report. Clin Infect Dis 2000;30(3): 629–30.
[17] Hannan MM, Husain S, Mattner F, Danziger-Isakov L, Drew RJ, Corey GR, et al. Working formulation for the standardization of definitions of infections in patients using ventricular assist devices. J Heart Lung Transplant 2011;30(4):375–84.
[18] DeSimone DC, Sohail MR, Kraft CS. Approach to Diagnosis of Cardiovascular Implantable-Electronic-Device Infection. J Clin Microbiol 2018;56(7). https://doi.org/10.1128/JCM.01683-17.
[19] CLSI. *Susceptibility Testing of Mycobacteria,* Nocardia *spp., and Other Aerobic Actinomycetes.* 3rd ed. CLSI standard M24. Wayne, PA: Clinical and Laboratory Standards Institute; 2018.
[20] Siu C-W, Cheng L-C, Woo PCY, Lau C-P, Tse H-F. A patient with relapsing pacemaker infection due to "Gram-positive bacilli". Int J Cardiol 2007;114(2): E40–1.

[21] Hu YL, Bridge B, Wang J, Jovin IS. Mycobacterium fortuitum causing infection of a biventricular pacemaker/implantable cardioverter defibrillator. Int J Mycobacteriol 2012;1(4):221–3.
[22] Giannella M, Valerio M, Franco JA, Marin M, Bouza E, Muñoz P. Pacemaker infection due to Mycobacterium fortuitum: the role of universal 16S rRNA gene PCR and sequencing. Diagn Microbiol Infect Dis 2007;57(3):337–9.
[23] Roest S, Bax HI, Verkaik NJ, Brugts JJ, Constantinescu AA, de Bakker CC, et al. Mycobacterium chelonae, an 'atypical' cause of an LVAD driveline infection. Int J Infect Dis 2020;92:127–9.
[24] Phadke VK, Hirsh DS, Goswami ND. Patient Report and Review of Rapidly Growing Mycobacterial Infection after Cardiac Device Implantation. Emerg Infect Dis 2016; 22(3):389–95.
[25] Marchandin H, Battistella P, Calvet B, Darbas H, Frapier JM, Jean-Pierre H, Parer S, Jumas-Bilak E, Van de Perre P, Godreuil S: Pacemaker surgical site infection caused by Mycobacterium goodii. J Med Microbiol 2009, 58(Pt 4):517-520.
[26] Kessler AT, Kourtis AP. Mycobacterium abscessus as a cause of pacemaker infection. Med Sci Monit 2004;10(10):CS60-62.
[27] Hooda A, Pati PK, John B, George PV, Michael JS: Disseminated Mycobacterium chelonae infection causing pacemaker lead endocarditis in an immunocompetent host. BMJ Case Rep 2014, 2014.
[28] Yoo DK, Hosseini-Moghaddam SM. Pacemaker pocket infection due to Mycobacterium goodii, a rapidly growing mycobacteria. BMJ Case Rep 2017, 2017..
[29] Radigan A, Jevert-Eichorn S. Rare case of pacemaker infection with Mycobacterium abscessus. BMJ Case Rep 2019;12(9):e230100. https://doi.org/10.1136/bcr-2019-230100.
[30] Al Zoubi M, Cheng J, Dontaraju VS, Evans CE, Spier AB. Native valve endocarditis and pacemaker infection with Mycobacterium fortuitum. IDCases 2021;25:e01200. https://doi.org/10.1016/j.idcr.2021.e01200.
[31] Nunez Breton JD, Hernandez G, Simkins J, Chaparro SV: Mycobacterium abscessus Left Ventricle Assist Device.
[32] Greenspon AJ, Patel JD, Lau E, Ochoa JA, Frisch DR, Ho RT, et al. 16-year trends in the infection burden for pacemakers and implantable cardioverter-defibrillators in the United States 1993 to 2008. J Am Coll Cardiol 2011;58(10):1001–6.
[33] Sohail MR, Eby EL, Ryan MP, Gunnarsson C, Wright LA, Greenspon AJ: Incidence,. Treatment Intensity, and Incremental Annual Expenditures for Patients Experiencing a Cardiac Implantable Electronic Device Infection: Evidence From a Large US Payer Database 1-Year Post Implantation. Circ Arrhythm Electrophysiol 2016;9(8).
[34] M et al. "Mycobacterium Abscessus Infection In Left Ventricular Assist Device (LVAD): A Case Series". Cureus , 2021. Cureus, Inc. , doi:10.7759/cureus.15718.
[35] Hernandez, Daley CL, Iaccarino JM, Lange C, Cambau E, Wallace Jr RJ, Andrejak C, Böttger EC, Brozek J, Griffith DE, Guglielmetti L, Huitt GA. Treatment of nontuberculous mycobacterial pulmonary disease: an official ATS/ERS/ESCMID/ IDSA clinical practice guideline. Clinical Infectious Diseases. 2020 Aug 14;71(4): e1-36.
[36] Haworth CS, Banks J, Capstick T, Fisher AJ, Gorsuch T, Laurenson IF, Leitch A, Loebinger MR, Milburn HJ, Nightingale M, Ormerod P. British Thoracic Society guidelines for the management of non-tuberculous mycobacterial pulmonary disease (NTM-PD). Thorax. 2017 Nov 1;72(Suppl 2):ii1-64.
[37] Strnad L, Winthrop KL. Treatment of Mycobacterium abscessus complex. InSeminars in respiratory and critical care medicine 2018 Jun (Vol. 39, No. 03, pp. 362-376). Thieme Medical Publisher.