Mycobacterium goodii: An Emerging Nosocomial Pathogen
A Case Report and Review of the Literature

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Abstract: *Mycobacterium goodii*, a rapidly growing nontuberculous mycobacterium, is an emerging pathogen in nosocomial infections. Its inherent resistance patterns make it a challenging organism to treat, and delays in identification can lead to poor outcomes. We present a case of cardiac device pocket infection with *M. goodii*, complicated by both antibiotic resistance and drug reactions that highlight the challenges faced by clinicians trying to eradicate these infections. We also present a brief review of the English literature surrounding this disease, including a table of all reported cases of *M. goodii* infections and their outcomes to act as guide for clinicians formulating treatment plans for these infections. A clear understanding of diagnostic methods and treatment caveats is essential to curing infections caused by these organisms.

Key Words: cardiac, device, infection, mycobacterium, nontuberculous, nosocomial, sulfamethoxazole/trimethoprim

(*Infect Dis Clin Pract* 2017;25: 62–65)

Mycobacterium goodii, a rapidly growing nontuberculous mycobacterium (NTM), is a Gram-positive, acid, and alcohol fast bacillus. It is related to *Mycobacterium smegmatis*; however, in 1999, Brown et al[1] reclassified it as its own species based on gene sequencing and antimicrobial resistance data. Since then, it has been identified in several infections, most of which are nosocomial. We present the third cardiac device pocket infection with *M. goodii*.

CASE REPORT

A 77-year-old man with a medical history of heart failure with reduced ejection fraction and chronic kidney disease was admitted for elective placement of an automated implantable cardiac defibrillator (AICD). He underwent placement of the AICD in the cardiac catheterization laboratory without complication. He was discharged the same day.

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The patient returned 4 weeks later with pus draining from the incision site. He reported erythema and tenderness had initially developed approximately 2 weeks after the procedure. He denied fevers or chills and had no other systemic manifestations of infection.

In the clinic, a 2×2-cm fluctuant mass was noted over the superior edge of the healed surgical incision. The patient otherwise had normal vital signs and an unremarkable physical examination, including lack of thromboembolic phenomena. His laboratory results were notable for a white blood cell count of 10.6×10^2 with a normal differential and a creatinine level of 2.03 mg/dL (his baseline). Two sets of blood cultures (4 bottles) were drawn and were negative for any bacterial growth. Pus was expressed from the AICD pocket and sent for culture. The patient was started on cephalexin 500 mg by mouth twice daily.

Later that week, the patient underwent complete AICD removal with pocket debridement and washout. Tissue from the pocket, a wound swab, and AICD leads were all sent for routine bacterial cultures. The patient was treated empirically with vancomycin 15 mg/kg intravenous (IV) every 12 hours and piperacillin/tazobactam 3.375 g IV every 12 hours (both renally adjusted). A wound vacuum was placed over the pacemaker pocket with excellent clinical response. The patient underwent a transesophageal echocardiogram, which did not show any evidence of endocarditis.

Four days into incubation, all tissue cultures grew Gram-positive rods. The patient's antibiotics were narrowed to vancomycin IV monotherapy, and the cultures were sent to the New Mexico State Laboratory for further identification. The organism was identified as a rapidly growing NTM. Cultures were then sent to Advanced Diagnostic Laboratories, National Jewish Health in Denver, Colo, for identification. The isolate was grown on a Lowenstein-Jensen slant and identified as *M. goodii* by RNA polymerase Beta subunit (rpoB) gene sequencing.

The patient's antibiotic coverage was changed from vancomycin to sulfamethoxazole/trimethoprim one double strength tablet by mouth twice daily and doxycycline 100 mg by mouth twice daily. Unfortunately, 4 days after treatment with this regimen, he developed worsening renal dysfunction with a creatinine level increase to 4 mg/dL and a serum urea nitrogen level of 40 mg/dL. Sulfamethoxazole/trimethoprim was stopped, and the patient was continued on doxycycline with the addition of both ethambutol 900 mg by mouth daily and ciprofloxacin 500 mg by mouth daily (renally adjusted). Two weeks later, ciprofloxacin was discontinued due to concerns for QTC prolongation in the setting of recent AICD removal. At this point, antimicrobial susceptibilities were available, and the patient's isolate was found to be sensitive to ampicillin-sulbactam. The patient was switched to a regimen of amoxicillin-clavulanate 875 mg by mouth twice daily and doxycycline 100 mg by mouth twice daily to complete a 4-month course.

There was a question about whether to treat the patient for endocarditis versus a pocket infection because the AICD leads grew *M. goodii*. On discussion with the laboratory and the operating surgeons, the entire lead was cultured, including the section of the leads attached to the battery within the infected pocket, making a pure ventricular tip lead infection impossible to determine.

TABLE 1. Clinical Infections With *Mycobacterium goodii*

Reference	Age, y	Sex	Comorbid Conditions	Infection Type	Identification Method	Surgical Therapy	Antimicrobial Therapy	Duration of Therapy	Outcome
Freidman et al,[14] 2000	60	M	Hypertension, diabetes, monoclonal gammopathy	Olecranon bursitis 5 wk after bursa injection	DNA polymerase chain reaction	Bursectomy	Doxycycline 100 mg PO BID, ciprofloxacin 500 mg PO BID	6 wk	Clinical cure
Ferguson et al,[5] 2004	65	M	Unknown	Prosthetic hip septic arthritis 4 wk after hip replacement	65-kd heat shock protein gene polymerase	Unknown	Unknown	Unknown	Clinical cure
Sohail and Smilack,[15] 2004	64	M	None	Inguinal hernia mesh infection and intra-abdominal wall abscess 2 d after hernia repair	65-kd heat shock protein gene polymerase	Mesh removal, abscess drainage, wound debridement	SMZ/TMP (dose not specified)	4+ wk	Minimal discharge after 4 wk, SMZ/TMP resumed briefly followed by clinical cure
Ferguson et al,[5] 2004	75	F	Unknown	Prosthetic knee septic arthritis 2 wk after total knee replacement	65-kd heat shock protein gene polymerase	Unknown	Unknown	Unknown	Clinical cure
Spencer et al,[11] 2005	67	M	Unknown	Postcataract endophthalmitis 4 wk after lens replacement	DNA sequencing	Vitrectomy twice	IV amikacin twice (dose unspecified)	~8 mo	Clinical cure
Buijtels et al,[16] 2005	66	M	None	Pneumonia	16s rRNA gene sequencing	Pleurocentesis	Augmentin (dose unspecified)	Unknown	Unknown
Chrissoheris et al,[13] 2008	85	M	Sick sinus syndrome	Pacemaker pocket infection 2 d after insertion	Unknown	Pacemaker removal	SMZ/TMP 1 tablet DS PO BID	8 wk	Clinical cure
Tompkins et al,[6] 2008	63	F	Diabetes	Prosthetic knee septic arthritis 5 mo after total knee replacement	Unknown	Joint washout with retained hardware	Doxycycline 100 mg PO BID and moxifloxacin 400 mg PO daily	36 wk followed by life-long suppression with doxycycline 100 mg PO BID	Clinical healing
Ahmad et al,[17] 2009	44	M	Alcohol abuse	Prosthetic knee septic arthritis ~4 wk after total knee replacement	DNA sequencing	Total removal of hardware	Minocycline 100 mg PO daily, ciprofloxacin 500 mg PO BID	24 wk	Clinical cure
Marchandin et al,[7] 2009	23	M	Congenital heart disease	Pacemaker pocket infection 8 d after insertion	16s rRNA gene sequencing	Washout and debridement, pacemaker retained	Ofloxacin 200 mg IV BID and amikacin 1 g IV daily followed by doxycycline 100 mg PO BID	IV antibiotics for 4 wk, PO antibiotics for 16 wk	Clinical cure

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TABLE 1. (Continued)

Reference	Age, y	Sex	Comorbid Conditions	Infection Type	Identification Method	Surgical Therapy	Antimicrobial Therapy	Duration of Therapy	Outcome
Martínez-González et al,[18] 2011	15	F	Achalasia	Pneumonia	Reverse hybridization assay	NA	Ciprofloxacin 500 mg PO BID and doxycycline 100 mg PO BID	52 wk	Clinical cure
Jönsson et al,[12] 2012	76	M	Ischemic heart disease	Prosthetic valve endocarditis 4 mo after valve replacement	16s rDNA sequencing	Removal of prosthetic valve	Meropenem and ampicillin, initially; finally, tigecycline and ciprofloxacin IV for 4 wk, followed by doxycycline and ciprofloxacin PO for 11 wk (doses unavailable)	15 wk	Clinical cure
Salas et al, 2015 (present case)	77	M	Ischemic cardiomyopathy, chronic kidney disease	AICD pocket infection 2 wk after placement of debrillator	rpoB gene sequencing	Removal of cardiac device and wound debridement	SMZ/TMP and doxycycline initially; finally treated with amoxicillin-clavulanate 875 mg by PO BID and doxycycline 100 mg PO BID	16 wk	Clinical cure

M, male; F, female; DS, double strength; PO, per os; rpoB, RNA polymerase Beta subunit; BID, bid in die (twice daily); SMZ/TMP, sulfamethoxazole/trimethoprim.

Given that the patient had no major or minor Duke criteria, was never septic, and had both a negative transesophageal echocardiogram and negative blood cultures, he was treated for an isolated pocket infection. His wounds are currently completely healed, and his inflammatory markers are down trending.

We present the third case of *M. goodii* cardiac device pocket infection reported in the English literature.

DISCUSSION

Infections, particularly nosocomial, with NTM are on the rise.[2] These bacteria, though indolent, are resistant to many forms of decontamination and sterilization, with postsurgical outbreak reports in South America and the United States.[3–5] *Mycobacterium goodii* presents a unique challenge due to its particular resistance pattern. This case highlights the diagnostic and treatment challenges of this particular NTM organism. We will summarize the current literature about diagnosing and treating *M. goodii*.

Mycobacterium goodii is a rapidly growing NTM that was differentiated from *M. smegmatis* in 1999 by Brown et al. These small Gram-positive rods grow in 2 to 4 days on most media (blood agar, chocolate agar, trypticase soy agar, Middlebrook 7H10 or 7H11 agar, and Lowenstein-Jensen agar).[1]

Treatment of *M. goodii* can be complex. All *M. goodii* infections associated with surgical intervention or implants in the literature required adequate surgical debridement and removal of contaminated material for clinical cure, with the exception of 2 cases.[6,7] Treatment subsequently requires prolonged appropriate antibiotic therapy.[2] Because of delays in diagnosis, empiric therapy is often begun for rapidly growing NTM, usually clarithromycin and rifampin. Unfortunately, *M. goodii* is inherently resistant to these medications due to 2 factors: overexpression of the *Wag31* gene and the presence of the *erm* gene.[8] Overexpression of the *Wag31* gene results in increased thickness of the peptidoglycan layer leading to decreased permeability of lipophilic drugs like rifampin.[9] The *erm* gene causes irregularities at the ribosomal binding site for macrolides, causing clarithromycin resistance.[8,10]

Treatment should be guided by antimicrobial susceptibility testing. The most commonly used drugs to treat this organism are sulfamethoxazole/trimethoprim and ethambutol, followed by doxycycline and ciprofloxacin, depending on susceptibilities.[1,2] For more serious infections, amikacin and meropenem have been reported.[11,12] When tolerated, sulfamethoxazole/trimethoprim has the most evidence for treatment success; however, allergies and renal toxicity, as seen in our case, limit its use (Table 1). Combination therapy is often used, but monotherapy, particularly with sulfamethoxazole/trimethoprim, has been reported (Table 1). Table 1 lists all of the cases of *M. goodii* reported in the English literature as well as the medications and durations of therapy that have been used. Please note that Brown et al investigated 28 isolates that were later classified as *M. goodii* using 16s RNA sequencing and DNA-DNA hybridization. The infections listed were predominantly posttraumatic wounds and bone infections, nosocomial infections, and pulmonary disease, including 3 cases with lipoid pneumonia. These clinical isolates are not included in Table 1 due to lack of clinical data surrounding the infections.

Treatment of *M. goodii* usually is prolonged and depends on the clinical syndrome[10] ranging from 4 weeks to 12 months (Table 1). In the case of cardiac device infections, there is no clear evidence as to when a new device can be implanted. In one case report, successful reimplantation occurred within 2 weeks of removal of the infected device.[13] In our patient, reimplantation was delayed until after completion of therapy (16 weeks).

In conclusion, *M. goodii* is a rare but challenging pathogen, most often associated with nosocomial infection. No specific host

risk factors have been reported. Identification of the organism requires gene sequencing to differentiate it from other rapidly growing NTM, which is essential given its unique resistance pattern. Treatment includes adequate surgical debridement, removal of contaminated exogenous material, and the use of 1 to 2 antimicrobial agents, usually including sulfamethoxazole/trimethoprim, guided by susceptibility testing. Duration of therapy is determined by the clinical syndrome.[10] Physicians need to be aware of the challenges to treating NTM infections as the incidence is increasing, and failed recognition can lead to poor outcomes.

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