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Research Article

A Rare Cause of Cardiac Implantable Electronic Device Infection: *Mycobacterium Chelonae* Bacteremia with Lead Vegetation

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LEARNING OBJECTIVES

1. To recognize nontuberculous mycobacteria as a rare but important cause of cardiac of cardiac implantable electronic device infections, particularly in immune compromised patients
2. To illustrate the role of leadless pacemaker implantation as a viable peacemaking strategy following device extraction in select high risk patients
3. To emphasize the role of transesophageal echocardiography in diagnosing cardiac implantable electronic device infections when bacteremia persists or presents atypically.

CASE PRESENTATION

A 75-year-old man with a past medical history significant for hypertension, hyperlipidemia, type 2 diabetes mellitus, chronic kidney disease stage 4, peripheral arterial disease, coronary artery disease status post coronary artery bypass grafting, heart failure with reduced ejection fraction, pulmonary hypertension, permanent atrial fibrillation on apixaban, and sick sinus syndrome status post dual-chamber permanent pacemaker implantation with subsequent upgrade to a cardiac resynchronization therapy defibrillator (CRT-D) presented to the emergency department with several days of confusion, lethargy, productive cough, and shortness of breath. The patient reported recent treatment of two gout flares with intravenous and oral corticosteroids.

On initial presentation the patient was hypotensive and febrile. Initial laboratory results demonstrated a normal white blood cell count, lactic acidosis, acute kidney injury on chronic kidney disease, and anemia of chronic disease. Notable findings on physical exam included necrotic-appearing wounds of the fingers bilaterally, a cellulitic appearing rash involving the right lower extremity, decreased bibasilar breath sounds, and an irregular heart rhythm. The patient was admitted to the intensive care unit for vasopressor support and treatment of septic shock. Blood cultures later grew methicillin-resistant *Staphylococcus aureus* (MRSA). Broad-spectrum intravenous antibiotic therapy with daptomycin and meropenem was initiated [1].

Transthoracic echocardiography (TTE) revealed severe left atrial enlargement, severe inferior and moderate inferolateral hypokinesis, hypokinesis of the apical, apical septal, and apical anterior segments, and moderately reduced left ventricular systolic function with an estimated ejection fraction of 35–40%. Estimated pulmonary artery systolic pressure was significantly elevated and right ventricular systolic function was at the lower limits of normal. Due to concern for infective endocarditis and possible cardiac device infection, transesophageal echocardiography (TEE) was performed and demonstrated a small mobile echodensity seeding the right atrial lead.

The patient underwent laser lead extraction of the CRT-D system, followed by implantation of a leadless pacemaker. Subsequent blood cultures revealed acid-fast bacilli, prompting modification of antimicrobial therapy. Meropenem was replaced with imipenem, and amikacin and linezolid were added. Final speciation identified *Mycobacterium chelonae*, after which linezolid was discontinued and clarithromycin was initiated. Lead cultures confirmed the diagnosis of mycobacterium chelonae bacteremia with lead vegetation [2].

Computed tomography of the chest without contrast demonstrated moderate bilateral pleural effusions, with associated bilateral lower lobe and lingula consolidations and collapse. The pleural effusions of the right lung appeared to be loculated. Serial sputum cultures were negative for acid-fast bacilli. Diagnostic thoracentesis was performed, from and identified a transudative effusion. Cultures did not identify microorganisms.

Magnetic resonance imaging and radiographs of the extremities showed no evidence of osteomyelitis, and vascular studies revealed no major arterial occlusions. The patient remained on systemic anticoagulation and no surgical interventions were performed.

Renal dysfunction was present on admission and progressively worsened during the hospital course. He developed refractory hypervolemia and persistent metabolic acidosis, ultimately requiring oxygen supplementation with high flow nasal cannula and initiation of daily renal replacement therapy.

INTRODUCTION

Cardiac implantable electronic device (CIED) infections are a serious complication associated with substantial morbidity and mortality, particularly in patients with advanced comorbidities and heart failure. The majority of CIED infections are caused by gram-positive organisms, most commonly *Staphylococcus aureus* and coagulase-negative staphylococci. In contrast, nontuberculous mycobacteria (NTM) represent an uncommon but increasingly recognized cause of invasive infections, particularly among immunocompromised hosts.

Mycobacterium chelonae is a rapidly growing NTM that most frequently causes skin and soft tissue infections, with bacteremia and endovascular involvement reported far less commonly. CIED infections due to *M. chelonae* are exceedingly rare and pose diagnostic and therapeutic challenges, as they may present with nonspecific systemic manifestations and require specialized microbiologic identification and prolonged antimicrobial therapy [3].

We present a case of *Mycobacterium chelonae* bacteremia complicated by CIED lead infection in a patient presenting with septic shock, multiorgan failure, and cutaneous manifestations following recent corticosteroid exposure. This case highlights the importance of maintaining a broad differential diagnosis for device-related infections, the role of transesophageal echocardiography in identifying lead involvement, and the necessity of complete device extraction with consideration of alternative pacing strategies.

DISCUSSION

This case illustrates several important clinical lessons regarding the diagnosis and management of CIED infections, particularly those caused by atypical organisms. While *Staphylococcus aureus* remains the most common cause of device-related infections, nontuberculous mycobacteria should be considered in patients with persistent hypotension and bacteremia, immunosuppression, or unusual systemic or cutaneous findings [4].

Nontuberculous Mycobacteria and CIED Infection

Rapidly growing NTM, including *M. chelonae*, are environmental organisms capable of causing invasive disease in susceptible hosts. Risk factors include chronic kidney disease, diabetes mellitus, advanced age, indwelling prosthetic material, and exposure to immunosuppressive therapies such as corticosteroids—all of which were present in this patient. Although *M. chelonae* most commonly causes localized skin and soft tissue infection, hematogenous spread with endovascular seeding has been reported and may be underrecognized.

CIED infections caused by NTM are rare, but when present, they often necessitate a high index of suspicion. Standard blood cultures may initially grow more common organisms or be slow to identify acid-fast bacilli, potentially delaying definitive diagnosis and appropriate antimicrobial therapy. In this case, initial treatment was directed toward MRSA bacteremia, with subsequent identification of acid-fast bacilli prompting broadened antimicrobial coverage.

Management and Device Extraction

Current guidelines strongly recommend complete device and lead removal in patients with definite CIED infection, regardless of the causative organism. This recommendation extends to infections caused by atypical pathogens such as NTM, for which antimicrobial therapy alone is insufficient. In the present case, laser lead extraction was successfully performed, followed by implantation of a leadless pacemaker to minimize the risk of reinfection and avoid transvenous hardware in an actively infected patient [5].

The use of leadless pacing systems has emerged as an attractive option in select patients with device infections, particularly those requiring ongoing pacing support. This strategy aligns with evolving approaches to reduce foreign material burden while maintaining hemodynamic stability.

Multisystem Involvement and Clinical Implications

The patient's presentation with septic shock, cutaneous

necrosis, and progressive renal failure highlights the potential severity of disseminated NTM infection. Although imaging did not reveal osteomyelitis or large-vessel occlusion, the presence of peripheral skin findings suggests possible microvascular or immune-mediated involvement, which has been described in disseminated mycobacterial disease.

This case emphasizes the importance of multidisciplinary collaboration involving cardiology, infectious disease, electrophysiology, nephrology in managing complex device-related infections complicated by multiorgan dysfunction.

Diagnostic Role of Echocardiography

Transesophageal echocardiography remains the imaging modality of choice for suspected CIED infection, with superior sensitivity for detecting lead-associated vegetations compared with transthoracic echocardiography. The identification of a mobile echodensity on the right atrial lead in this patient, in the setting of persistent bacteremia and septic shock, supported the diagnosis of device-related infection and guided definitive management [6].

The echocardiographic findings also demonstrated significant underlying structural heart disease, underscoring the complexity of management decisions in patients with advanced cardiomyopathy.

Take-Home Message

Clinicians should maintain a broad differential diagnosis for CIED infections, particularly in immunocompromised patients or those with atypical systemic manifestations. Early use of transesophageal echocardiography, prompt microbiologic evaluation for acid-fast organisms, and adherence to guideline-directed device extraction are essential to optimizing outcomes. Leadless pacing may serve as a valuable alternative in select cases following device removal.

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