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Mycobacterium Fortuitum Device Infection with Subsequent Endocarditis

Paurush Shah, MD
Thomas Jefferson University

Alec Vishnevsky, MD
Thomas Jefferson University

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Case

A 78-year-old female with a past medical history of mild dementia, hypertension, diabetes, coronary artery disease status post automatic implantable cardioverter defibrillator (AICD) for congestive heart failure presented with suspected bacterial endocarditis and AICD lead infection from an outside hospital (OSH). The patient initially presented to the OSH with chest wall tenderness, fevers up to 101° F, chills, decreased appetite, weakness and weight loss. She was diagnosed with a non-ST segment myocardial infarction based on elevated troponin levels without electrocardiogram changes. At the OSH, blood acid fast bacillus (AFB) cultures were checked after routine blood cultures and fungal cultures were negative. Three separate blood cultures grew out *Mycobacterium fortuitum* over 3 different time intervals at the OSH. The patient was started on trimethoprim/sulfamethoxazole 160mg/800mg orally three times a day and ciprofloxacin 500 mg orally twice a day. It was decided that definitive treatment for her persistent bacteremia and suspected endocarditis would require removal of the device and leads. The patient was transferred to Thomas Jefferson University Hospital (TJUH) for AICD lead removal and temporary pacemaker placement.

On admission to TJUH, the patient was afebrile with a normal heart and respiratory rate and a blood pressure of 102/78 mm Hg. She was awake and alert, but oriented only to person and place due to mild underlying dementia. Cardiac exam did not reveal any murmurs, rubs, or gallops, but the patient did have mild tenderness upon palpation of the pacer pocket site. There was no overlying erythema or exudates from the site. Her pulmonary, abdominal, and musculoskeletal exams were all grossly normal.

Initial laboratory values revealed a white blood cell count of 4.4/μL. A basic metabolic panel revealed a mildly elevated creatinine of 1.6 mg/d with no other major abnormalities. Troponins were negative at the time of admission.

Blood cultures for AFB were again positive upon admission to TJUH, and intravenous (IV) amikacin and imepenem were initiated. The patient then underwent removal of the AICD leads. An echocardiogram was done at this time which revealed a 0.8 cm heterogenous mass on the aortic side of the left coronary cusp consistent with vegetation versus complex fibroelastoma, and a 0.8 cm mobile echodensity on the tricuspid valve consistent with vegetation.

After removal of her AICD, her blood cultures cleared, but the patient remained hospitalized for 4 weeks given the need for IV antibiotics and temporary pacing prior to reimplantation of her AICD. An AICD was reimplanted one month later without complications. The patient was then transferred to a skilled nursing facility to continue IV antibiotics for an additional month.

Discussion

There are currently approximately 3 million people with pacemakers implanted worldwide. Despite obvious benefits conferred by these devices, infection of the leads and device and subsequent endocarditis remains a common complication. The incidence of device related infections has more than doubled since 1990, the majority of which are caused by Staphylococcus and Streptococcus species. Rapidly Growing Mycobacterium (RGM), so called because they grow in 3-4 days as compared to other mycobacteria which can takes weeks to grow on culture media, are a rare cause of pacemaker infections that carry significant morbidity and mortality. The most common of these, *Mycobacterium fortuitum*, is ubiquitous in the environment and has been isolated from fresh water, salt water, soil, and in hospitals. This RGM can grow in water systems and distilled water, and is resistant to sterilizers, antiseptics, and other standard disinfectants. The spectrum of disease caused by *M. fortuitum* ranges from surgical wound infections and dermal abscesses to osteomyelitis, septic arthritis, pericarditis, and device infections. Of note, pulmonary disease caused by *M. fortuitum* infection is rare except in patients with esophageal achalasia, lipoid pneumonia, or other diseases characterized by chronic vomiting and aspiration. A review of the literature reveals that the most common presenting complaints of device infections with *M. fortuitum* include wound discharge, fever, and pain at the generator site. Mycobacteria in gram stained specimens may appear as refractile, gram positive or gram neutral bacilli, and misidentification as *Nocardia spp.* is common.

Although previously rare, *M. fortuitum* device infection rate has been on the rise, with over 50% of the cases arising since 2007. Such infections commonly arise within the first 6 months of device implantation, thus suggesting a nosocomial source. RGM outbreaks have previously been traced to contaminated tap water used to make cardioplegia solution or contaminated aqueous solutions used to mark incision sites. As with all device infections, definitive treatment involves removal of the device and treatment with antibiotics for 4-6 weeks. According to the American Thoracic Society Guidelines, *M. fortuitum* isolates are usually susceptible to multiple oral antimicrobial agents, including macrolides, quinolones, doxycycline, sulfonamides, and beta-lactam antibiotics including imipenem. Although specific antibiotic choice depends on susceptibility testing, combination therapy is usually preferred to monotherapy. Empiric therapy with clarithromycin and a fluoroquinolone is a reasonable first step, although mounting evidence suggests

increasing resistance to macrolides. In some case series linezolid has been used additionally in the first 2 weeks of treatment.

Our patient's course was complicated by need for multiple antibiotic regimens. The patient failed the initial regimen of trimethoprim/sulfamethoxazole and ciprofloxacin, and was subsequently started on imipenem and amikacin. However, the patient developed thrombocytopenia as a side effect of imipenem. Final regimen on discharge included tigecycline, amikacin, and trimethoprim/sulfamethoxazole.

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