Implantable Defibrillator Pocket Infection and Bacteremia Caused by *Nocardia nova* Complex Isolate

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***Nocardia* spp. are common environmental organisms that, to our knowledge, have never been implicated as causing an implantable defibrillator or pacemaker infection. We describe a 70-year-old male with a recent implantable cardiac defibrillator revision and subsequent device infection and bacteremia caused by a *Nocardia nova* complex isolate.**

CASE REPORT

A 70-year-old-male with long-standing diabetes, ischemic cardiomyopathy, and inducible ventricular tachycardia presented with inappropriate shocks due to fracture of his implantable cardiac defibrillator (ICD) lead 3 years postimplantation. He underwent a procedure in which his fractured lead was abandoned and a new lead and pulse generator were implanted.

He tolerated the replacement procedure well and approximately 2 weeks later noticed swelling around the site. He presented to a surgeon, who noted swelling around the site and aspirated serosanguinous fluid from the pocket. A routine aerobic culture of this fluid was negative. The swelling continued, and 1 week later the patient returned to the surgeon, who again aspirated the site. He returned for a third visit 1 week later with additional symptoms, including swelling, erythema, tenderness, fevers, and chills. The pocket was again aspirated, and the patient was transferred to our institution for further evaluation.

Upon arrival at Geisinger Medical Center, the patient had a temperature of 38.4°C, heart rate of 79/min, respirations of 16/min, and an admission blood pressure of 114/70. Two routine peripheral blood cultures were collected, and vancomycin was initiated for suspected defibrillator pocket infection. Aerobic cultures from the second and third aspiration as well as from the admission blood cultures grew a pure culture of a beaded, branching gram-positive bacillus identified as a *Nocardia nova* complex isolate. Vancomycin was discontinued, and treatment was begun with trimethoprim-sulfamethoxazole at a dose of 8 mg/kg of body weight/day of trimethoprim divided twice daily. A transesophageal echocardiograph showed no evidence of endocarditis. The newly implanted ICD system and previously abandoned lead were removed to optimally treat the patient's infection. The pocket had 10 ml of purulent bloody fluid as well as necrotic tissue removed during reimplantation. The patient completed 6 weeks of oral antibiotic treatment and had his defibrillator replaced approximately 2 months later. There has been no recurrence to date.

Microbiology. The first culture of the defibrillator pocket from this patient was negative, but three subsequent pacer pocket cultures grew pure cultures of a branching, beaded gram-positive bacillus. Blood cultures performed with the BacT/ALERT blood culture system (bioMérieux, Durham, N.C.) were positive in the aerobic FA bottles from two blood culture sets at 2.6 and 2.7 days of incubation. Initial growth of the defibrillator pocket specimens on blood agar plates (Remel, Lenexa, Kans.) occurred within 2 days of inoculation and showed white opaque and dry colonies, which became chalky and orange with prolonged incubation. Casein, tyrosine, and xanthine hydrolysis agars were all negative. The organism was lysozyme resistant and modified acid fast positive. Disk diffusion susceptibility testing (11) was performed with the following results: cefamandole, 44 mm, susceptible; erythromycin, 40 mm, susceptible; and tobramycin, 14 mm, resistant. The isolate was presumptively identified as *N. nova* (11). This identification was confirmed by the Mycobacteria/Nocardia Research Laboratory at the University of Texas Health Science Laboratory at Tyler, by PCR restriction enzyme analysis (9).

Discussion. Based upon the antibiogram profile (9) and a molecular method (11), this patient's isolate was identified as *N. nova*. Historically, *N. nova* was initially differentiated from other members of the *Nocardia asteroides* complex by DNA homology and antibiograms (9, 10, 11). Subsequent to our identification, Conville et al. (3), utilizing a restriction fragment length polymorphism technique applied to an amplified portion of the 16S rRNA gene of *Nocardia* isolates, reported that the *N. nova* complex includes four distinct species. Consequently, it is more appropriate to refer to our isolate as a member of the *N. nova* complex.

Nocardia infections in humans are uncommon, with a reported incidence in the United States of 500 to 1,000 new cases per year (2) although, since this is not a reportable organism, the true incidence is likely underestimated. Most *Nocardia* infections have been reported to be caused by *N. asteroides* and are pulmonary, central nervous system related, or cutaneous in nature (1). Given the recent changes in *Nocardia* taxonomy resulting from the newer molecular techniques (3, 9), it is reasonable to assume that some of the reports of *N. asteroides* infections that were based upon phenotypic identification

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methods were likely caused by other *Nocardia* species. Therefore, the true incidence of infections due to isolates of *N. nova* complex is unknown.

Nocardia species infections are found mostly in immunocompromised hosts. Our patient was a long-standing diabetic but had no other risk factors or evidence of impaired host defense. We believe that this is the first reported case of an *N. nova* complex ICD infection. Several case reports have described prosthetic and native valve endocarditis as well as catheter-related infections caused by *N. asteroides* (4, 5, 6, 7, 8, 12; W. Y. Lui, A. C. Lee, and T. L. Que, Letter, Clin. Infect. Dis. 33:1613-1614, 2001).

Nocardia species are found worldwide and are common components of soil, decaying matter, and aquatic environments. The most common mode of entry in humans is inhalation, although cutaneous disease by direct inoculation of the organism has been reported. Our patient denies any trauma or other injury to the device location after the time of his revision. It is possible that inoculation occurred at the time of reimplantation; however, given the ubiquitous presence of these organisms in the environment, local inoculation after reimplantation is another possibility.

We are unsure why the first aspiration culture yielded a negative result; it may have simply been a sampling issue. It is also possible but, we believe, unlikely that the organism was introduced by the first aspiration procedure.

Nocardia bacteremia is a rare event (6). Our patient had positive blood cultures with no evidence of pulmonary infection and had a transesophageal echocardiogram without evidence of endocarditis. Interestingly, the isolates grew quickly and were detected in both of the aerobic FA bottles used for the blood cultures. The isolate was not recovered from either of the anaerobic FN blood culture bottles.

No prospective randomized trials have been performed to

determine the most effective therapy for nocardiosis. Sulfonamides have long been considered the "gold standard," with trimethoprim-sulfamethoxazole considered the drug of choice. The duration of effective treatment is believed to be at least 6 weeks or until complete resolution of clinical symptoms, followed by a course of reduced therapy for several months.

REFERENCES

1. Beaman, B. L., and L. Beaman. 1994. *Nocardia* species: host-parasite relationships. Clin. Microbiol. Rev. 7:213-264.
2. Beaman, B. L., J. Burnside, B. Edwards, and W. Causey. 1976. Nocardial infections in the United States, 1972-1974. J. Infect. Dis. 134:286-289.
3. Conville, P. S., J. M. Brown, A. G. Steigerwalt, J. W. Lee, D. E. Byrer, V. L. Anderson, S. E. Dorman, S. M. Holland, B. Cahill, K. C. Carroll, and F. G. Witebsky. 2003. *Nocardia veterana* as a pathogen in North American patients. J. Clin. Microbiol. 41:2560-2568.
4. Ertl, G., K. P. Schaal, and K. Kochsieck. 1987. Nocardial endocarditis of an aortic valve prosthesis. Br. Heart J. 57:384-386.
5. Esteban, J., J.-M. Ramos, M. L. Fernandez-Guerrero, and F. Soriano. 1994. Isolation of *Nocardia* sp. from blood cultures in a teaching hospital. Scand. J. Infect. Dis. 26:693-696.
6. Falk, R. H., F. R. Dimock, and J. Sharkey. 1979. Prosthetic valve endocarditis resulting from *Nocardia asteroides*. Br. Heart J. 41:125-127.
7. Kiska, D. L., K. Hicks, and D. J. Pettit. 2002. Identification of medically relevant *Nocardia* species with an abbreviated battery of tests. J. Clin. Microbiol. 40:1346-1351.
8. Kontoyannis, D. P., K. L. Jacobson, E. E. Whimbey, K. V. I. Rolston, and I. I. Raad. 2000. Central venous catheter-associated *Nocardia* bacteremia: an unusual manifestation of nocardiosis. Clin. Infect. Dis. 31:617-618.
9. Steingrube, V. A., B. A. Brown, J. L. Gibson, R. W. Wilson, J. Brown, Z. Blacklock, K. Yost, and S. Locke. 1995. DNA amplification and restriction endonuclease analysis for differentiation of 12 species and taxa of *Nocardia*, including recognition of four new taxa within the *Nocardia asteroides* complex. J. Clin. Microbiol. 33:3096-3101.
10. Wallace, R. J., Jr., M. Tsukamura, B. A. Brown, J. Brown, V. A. Steingrube, Y. Zhang, and D. R. Nash. 1990. Cefotaxime-resistant *Nocardia asteroides* strains are isolates of the controversial species *Nocardia farcinica*. J. Clin. Microbiol. 28:2726-2732.
11. Wallace, R. J., B. A. Brown, M. Tsukamura, J. M. Brown, and G. O. Onyi. 1991. Clinical and laboratory features of *Nocardia nova*. J. Clin. Microbiol. 29:2407-2411.
12. Watson, A., P. French, and M. Wilson. 2001. *Nocardia asteroides* native valve endocarditis. Clin. Infect. Dis. 32:660-661.