First case of *Nocardia amamiensis* pulmonary infection in Mexico

A. Martinez-Gamboa[1], **M. E. Cervera-Hernandez**[1], **P. Torres-Gonzalez**[1], **A. Rangel-Cordero**[1], **A. Ponce-de-Leon**[1] and **J. Sifuentes-Osornio**[2]

1) *Department of Infectious Diseases, Laboratory of Clinical Microbiology and* 2) *Department of Medicine, Instituto Nacional de Ciencias Médicas y Nutrición Salvador Zubirán, Mexico City, Mexico*

Abstract

We report a case of *Nocardia amamiensis* pulmonary infection in a 43-year-old immunocompromised woman. The patient was treated with imipenem/cilastatin and trimethoprim/sulfamethoxazole and had a favourable outcome. It is important that laboratories perform species identification to understand the epidemiology and susceptibility patterns of the different *Nocardia* spp.

Keywords: Antimicrobial susceptibility, immunocompromised, Mexico, *Nocardia amamiensis*, pulmonary nocardiosis
Original Submission: 5 May 2016; **Revised Submission:** 24 November 2016; **Accepted:** 1 December 2016
Article published online: 9 December 2016

Corresponding author: J. Sifuentes-Osornio, Vasco de Quiroga 15, Colonia Belisario Dominguez Seccion XVI, Tlalpan 14080, Mexico City, Mexico
E-mail: sifuentesosornio@gmail.com

In 2007, a novel species, *Nocardia amamiensis,* was identified from the soil in a sugarcane field in Japan [1]. To date, there have been three reported cases (two ocular and one pulmonary) of *N. amamiensis* infection [2,3]. Here we report a pulmonary infection in an immunocompromised patient.

In 2014, a 43-year-old woman from Oaxaca, Mexico, sought care at the emergency department with recent onset shortness of breath; she had experienced fever, weight loss, and productive cough over the last 3 months. Written informed consent was obtained from the patient for publication of this case report. Her medical history included primary glomerulonephritis treated with 32.5 mg per day of prednisone and tacrolimus for the previous 5 months. During the initial evaluation, she had diminished breath sounds over the right lung base and tachycardia. Contrast-enhanced computed tomography of the chest revealed multiple cavitary lesions in the right lower lung lobe. Laboratory studies revealed hemoglobin of 8.2 g/dL and 13 800/μL white blood cells. Empiric treatment with intravenous clindamycin and ceftriaxone was initiated. We performed a bronchoalveolar lavage, and the sample was inoculated in soy agar with 5% sheep blood plates and incubated under aerobic conditions at 35°C. After 6 days' incubation, we observed the growth of chalky irregular grey colonies in the culture media which were positive for the modified Kinyoun acid-fast staining. We performed conventional biochemical tests (Table 1) along with amplification and nucleotide sequencing of the partial 16S rRNA, as well as *secA1* genes. The sequences were compared with those available at the National Center for Biotechnology Information database (http://www.ncbi.nlm.nih.gov/). The interpretation was performed according to the Clinical and Laboratory Standards Institute's (CLSI) statement [4]. The nucleotide sequences showed 100 and 99.6% matches with *N. amamiensis* respectively. We also performed susceptibility testing for amoxicillin/clavulanate, moxifloxacin, imipenem and amikacin according to the CLSI's statement [5] (Table 1).

On day 9 of admission, the patient developed acute respiratory distress syndrome that required mechanical ventilatory support. She was transferred to the intensive care unit (ICU), where she spent 1 month with a fluctuating clinical course. Upon *Nocardia* spp. identification, ceftriaxone and clindamycin were discontinued, and she initiated therapy with imipenem/cilastatin and trimethoprim/sulfamethoxazole. Brain magnetic resonance imaging scan was negative for central nervous system dissemination, and prednisone was tapered down over the course of hospitalization. After weaning from mechanical ventilation, she was discharged from the ICU and completed 36 days of imipenem/cilastatin and trimethoprim/sulfamethoxazole.

http://dx.doi.org/10.1016/j.nmni.2016.12.005

TABLE 1. Microbiologic investigations and antimicrobial susceptibility pattern

Biochemical test	Result
Urease	+
Hydrolysis of:	
Casein	−
Tyrosine	−
Xanthine	−
Hypoxanthine	−
Esculin	−
Utilization of citrate	−
Nitrate reduction	+
Growth at 45°C	−
Growth with lysozyme	+
Antimicrobial susceptibility	MIC (mg/L)
TMP/SMX	0.062/1.1875
Amoxicillin/clavulanate	4/2
Moxifloxacin	>8
Imipenem	0.5
Amikacin	0.25
Gene	% Similarity
16S rRNA	100
secA1	99.6

MIC, minimum inhibitory concentration; TMP/SMX, trimethoprim/sulfamethoxazole.

The patient was discharged from the hospital and sent home with trimethoprim/sulfamethoxazole prophylaxis. No relapse episodes were documented after 20 months of follow-up.

Nocardia spp. are branching, Gram-positive rods that occasionally cause infection in humans. Over two-thirds of the patients have depressed cell-mediated immunity [6,7]. *Nocardia* spp. are found in the environment, and inhalation is the primary route of contagion. Pulmonary nocardiosis is thus the most frequent clinical presentation [8]. The central nervous system is the preferred extrapulmonary site of dissemination, and imaging should be performed of immunocompromised patients [5].

The genus *Nocardia* comprises more than 86 species and will continue to expand as DNA sequencing progresses. Each species may display different antibiotic susceptibility patterns and levels of pathogenicity [9]. Species-level identification is not always possible by biochemical tests, and gene sequencing may help clarify the proper treatment.

N. amamiensis is a newly recognized cause of pulmonary infection. The clinical features resemble those of the rest of the *Nocardia* genus. However, to understand the epidemiology and susceptibility patterns of the different species of *Nocardia,* it is important for laboratories to identify them by promptly using DNA target sequencing.

Conflict of Interest

None declared.

References

[1] Yamamura H, Tamura T, Sakiyama Y, Harayama S. *Nocardia amamiensis* sp. nov., isolated from a sugar-cane field in Japan. Int J Syst Evol Microbiol 2007;57(Pt 7):1599–602.
[2] Reddy AK, Garg P, Kaur I. Spectrum and clinicomicrobiological profile of *Nocardia keratitis* caused by rare species of *Nocardia* identified by 16S rRNA gene sequencing. Eye (Lond) 2010;24:1259–62.
[3] Rudramurthy SM, Honnavar P, Kaur H, Samanta P, Ray P, Ghosh A, et al. Molecular identification of clinical *Nocardia* isolates from India. J Med Microbiol 2015;64:1216–25.
[4] Petti CA; Clinical and Laboratory Standards Institute. Interpretive criteria for identification of bacteria and fungi by DNA target sequencing: approved guideline. Wayne, PA: Clinical and Laboratory Standards Institute; 2008.
[5] Clinical and Laboratory Standards Institute. Susceptibility testing of mycobacteria, Nocardiae, and other aerobic actinomycetes; approved standard—second edition. CLSI document M24–A2. Wayne, PA: Clinical and Laboratory Standards Insititute; 2011.
[6] Wilson JW. Nocardiosis: updates and clinical overview. Mayo Clin Proc 2012;87:403–7.
[7] Marquez-Diaz F, Soto-Ramirez LE, Sifuentes-Osornio J. Nocardiasis in patients with HIV infection. AIDS Patient Care STDs 1998;12:825–32.
[8] Beaman BL, Beaman L. *Nocardia* species: host–parasite relationships. Clin Microbiol Rev 1994;7:213–64.
[9] McTaggart LR, Richardson SE, Witkowska M, Zhang SX. Phylogeny and identification of *Nocardia* species on the basis of multilocus sequence analysis. J Clin Microbiol 2010;48:4525–33.