

# [ CASE REPORT ]

# Lung and Cerebral Nocardiosis Caused by *Nocardia elegans* in a Lung Transplant Recipient: A Case Report and Literature Review

Keitaro Omori<sup>1,2</sup>, Hiroki Kitagawa<sup>1,3</sup>, Rie Nagaoka<sup>4,5</sup>, Yasuhiko Naka<sup>2</sup>, Kazuma Kawamoto<sup>2</sup>, Yasushi Horimasu<sup>2</sup>, Toshihito Nomura<sup>1</sup>, Norifumi Shigemoto<sup>1,3,6</sup>, Takashi Yaguchi<sup>7</sup>, Noboru Hattori<sup>2</sup> and Hiroki Ohge<sup>1</sup>

#### **Abstract:**

Patients after lung transplantation are at risk for *Nocardia* infections. We herein report a case of lung and cerebral nocardiosis caused by *Nocardia elegans*, a rare species of *Nocardia*, in a lung transplant recipient. Antibiotic therapy, including sulfamethoxazole-trimethoprim (ST), and brain abscess drainage improved symptoms and imaging findings. A literature review of *N. elegans* infections showed that 12 of 14 cases (85.7%) were reported from East Asia, particularly Japan (9 cases, 64.2%). The lungs were the predominant site (12/14 cases, 85.7%), and most of the cases were susceptible to ST (9/10 cases, 90%).

Key words: lung and cerebral nocardiosis, *Nocardia elegans*, lung transplant, mass spectrometry, brain abscess

(Intern Med 62: 431-437, 2023) (DOI: 10.2169/internalmedicine.9813-22)

# Introduction

Nocardiosis is an opportunistic infection typically found in immunocompromised patients. An impaired cell-mediated immunity, such as in solid organ transplant recipients receiving corticosteroids and immunosuppressive agents, leads to the development of nocardiosis. Lungs are the most frequently involved organs, followed by skin and soft tissue, and the central nervous system, although it sometimes occurs as disseminated disease (1).

*Nocardia* species are Gram-positive, filamentous bacteria that are ubiquitous in soil, water, and dust. More than 100 species of *Nocardia* have been identified with the advancement of molecular identification methods (2). Of these, approximately 40 species are known to cause infections in hu-

mans. *Nocardia nova*, *N. brasiliensis*, *N. cyriacigeorgica*, *N. farcinica*, *N. abscessus*, and *N. asteroides* are the most frequently reported species (2), and reported cases of infection with *N. elegans* are limited.

In patients with lung transplantation, infections caused by *Nocardia* species, such as *N. farcinica*, *N. nova*, and *N. asteroids*, have been reported (3-7). However, to our knowledge, there have been no reports of *N. elegans* infection in lung transplant recipients. We herein report a case of lung and cerebral nocardiosis caused by *N. elegans* in a lung transplant recipient along with its clinical microbiological characteristics based on a literature review.

## **Case Report**

A 53-year-old man underwent right-sided lung transplan-

Received: March 21, 2022; Accepted: May 30, 2022; Advance Publication by J-STAGE: July 14, 2022 Correspondence to Dr. Keitaro Omori, a722@hiroshima-u.ac.jp

<sup>&</sup>lt;sup>1</sup>Department of Infectious Diseases, Hiroshima University Hospital, Japan, <sup>2</sup>Department of Molecular and Internal Medicine, Graduate School of Biomedical and Health Sciences, Hiroshima University, Japan, <sup>3</sup>Department of Surgery, Graduate School of Biomedical and Health Sciences, Hiroshima University, Japan, <sup>4</sup>Section of Clinical Laboratory, Department of Clinical Support, Hiroshima University Hospital, Japan, <sup>5</sup>Division of Clinical Laboratory Medicine, Hiroshima University Hospital, Japan, <sup>6</sup>Translational Research Center, Hiroshima University, Japan and <sup>7</sup>Medical Mycology Research Center, Chiba University, Japan



**Figure 1.** Images of chest computed tomography (CT) and brain magnetic resonance imaging (MRI) of the patient. Chest CT images showing infiltration shadows of the upper lobe of the left lung and the lower lobe of the right lung on admission (A, D). These infiltration shadows showed improvement on day 14 of treatment (B, E) and had almost disappeared after three months of treatment (C, F). Brain MRI showing a 2-cm enhancing lesion with perilesional edema in the left temporal occipital lobe on admission (G, J). This lesion was enlarged on day 14 of treatment (H, K). However, it had improved markedly at three months following abscess drainage and antimicrobial therapy (I, L). (G-I) T2-weighted and (J-L) fluid-attenuated inversion recovery (FLAIR).

tation for interstitial pneumonia 13 months prior to presentation, and was administered prednisolone (10 mg/day), tacrolimus (0.4 mg/day), mycophenolate mofetil (500 mg/ day), and prophylactic sulfamethoxazole-trimethoprim (ST, 400 mg sulfamethoxazole, 80 mg trimethoprim). He had a low-grade fever for 40 days, cough, and sputum before admission. Because treatment with garenoxacin did not improve his symptoms, he was admitted to our hospital.

Chest computed tomography (CT) showed new infiltrative shadows in the upper lobe of the left lung and partially in the lower lobe of the right lung (Fig. 1A, D). Gram staining of bronchial lavage fluid (BLF) retrieved from the left S1+2 revealed Gram-positive branched filamentous bacteria, and *Nocardia* species were cultured on the fourth day on blood agar and chocolate agar under aerobic conditions (Fig. 2). A mycobacterial culture was negative. Although he had no abnormal neurologic findings, magnetic resonance imaging (MRI) of the brain revealed a 2-cm brain abscess in the left temporal occipital lobe (Fig. 1G, J), and he was diagnosed with lung and cerebral nocardiosis. Antimicrobial therapy with ST (15 mg/kg, as trimethoprim) and meropenem (MEPM, 6 g/day) was initiated. Given concerns of allograft rejection, the dose of immunosuppressive drugs was not reduced.



**Figure 2.** Microbiological images of *Nocardia elegans* isolated from bronchial lavage fluid. (A) Gram staining revealed beaded and branched filamentous gram-positive rods. Dry, white colonies on a blood-agar plate after four days (B) and six days (C) of incubation under aerobic conditions.

Lung lesions and his symptoms were apparently improved (Fig. 1B, E), whereas the brain abscess was enlarged on brain MRI on day 14 of treatment (Fig. 1H, K). Therefore, fine-needle abscess drainage was performed on day 15, and the abscess was gradually absorbed and improved thereafter. *Nocardia* species were also cultured from brain abscesses.

The isolates from the BLF were identified as N. nova by matrix-assisted laser desorption ionization time-of-flight mass spectrometry (MALDI-TOF-MS; BD MALDI Biotyper Sirius system; Becton, Dickinson, Franklin Lakes, USA) using the MBT Compass 4.1 with reference database MBT Compass library: Ver.9.0.0.0 (8468MSPs, Bruker Daltonik GmbH, Bremen, Germany) with a score value of 2.07-2.18. However, subsequent 16S rRNA gene sequencing identified the isolates as N. elegans, showing 100% homology of 1,462 base pairs to the N. elegans type strain IMMIB N-402 (Sequence ID: NR 042353). In contrast, the sequencing showed 98.1% homology to the N. nova strain JCM 6044 (Sequence ID: NR\_041858). The strain isolated from BLF was preserved as IFM 12243 at the Medical Mycology Research Center, Chiba University, a reference center through the National Bio-Resource Project of Japan.

After brain abscess drainage, the patient was treated with amikacin (AMK) instead of ST until ST susceptibility testing was completed, which was performed at Chiba University. Antimicrobial susceptibility testing using the microdilution method showed that the strain was susceptible to ST and ceftriaxone (CTRX) (Table 1); therefore, the treatment was changed to ST+CTRX 4 g/day. Intravenous therapy was continued for a total of 7 weeks. During this time, ST was reduced to 10 mg/kg of trimethoprim due to hyponatremia and anorexia. The patient was discharged after transition to ST monotherapy.

CT and MRI after 3 months of treatment showed marked improvement in both the pulmonary and brain lesions (Fig. 1C, F, I, L). Treatment with ST will be continued for one year, the standard duration for brain abscess treatment in immunosuppressed patients.

# Discussion

Nocardiosis is a rare and invasive opportunistic infection in immunocompromised patients. Patients after solid organ transplantation are at risk for Nocardia infections due to the use of immunosuppressive agents (3). It has been previously reported that 1.0-3.5% of lung transplant recipients develop nocardiosis (3-7). In addition, lung transplantation was suggested to have a higher risk of nocardiosis than other solid organ transplantations. Peleg et al. reported that the frequency of nocardiosis in all patients after solid organ transplantation was 0.6%, with rates of 3.5%, 2.5%, 1.3%, 0.2%, and 0.1% reported in lung, heart, small intestine, kidney, and liver transplantation, respectively; lung transplantation patients had the highest frequency of nocardiosis (7). A report from Duke University showed that among the 37 patients who developed nocardiosis after solid organ transplantation, patients with lung and heart transplantation accounted for 40.5% each (8).

Several mechanisms underlying the increased susceptibility of patients with lung transplantation to *Nocardia* have been proposed (3, 4, 9). First, the transplanted lung is exposed to the external environment via the airway and is prone to inhalation of airborne microorganisms. Second, the transplanted lungs have reduced airway clearance function due to denervation. Third, immunosuppressive agents have been used for a long time to treat pre-existing lung diseases, such as interstitial lung disease. In a previous study of nocardiosis after lung transplants, most cases were caused by *N. farcinica*, *N. nova*, and *N. asteroides* (3-7). To our knowledge, this is the first documented case of *N. elegans* infection after lung transplantation.

Following advances in the taxonomy of Nocardia species

Antibiotics	MIC (µg/mL)	Interpretive categories and MIC breakpoints (µg/mL)*			
		Susceptible	Intermediate	Resistant	
Amikacin	≤0.5	≤8	-	≥16	
Amoxicillin-clavulanic acid	16/8	≤8/4	16/8	≥32/16	
Ceftriaxone	≤2	≤8	16-32	≥64	
Ciprofloxacin	>4	≤1	2	≥4	
Clarithromycin	≤0.25	≤2	4	≥8	
Imipenem	≤0.5	≤4	8	≥16	
Linezolid	≤1	≤8	-	-	
Minocycline	2	≤1	2-4	≥8	
Sulfamethoxazole-trimethoprim	≤9.5/0.5	≤38/2	-	≥76/4	
Tobramycin	>16	≤4	8	≥16	
Cefotaxime	≤2	≤8	16-32	≥64	
Cefepime	≤0.5	≤8	16	≥32	
Doxycycline	4	≤1	2-4	≥8	
Gentamicin	8				
Ampicillin	0.5				
Erythromycin	0.5				

 Table 1. Antimicrobial Susceptibilities of the Isolated Nocardia elegans.

MIC: minimum inhibitory concentration

\*These data are based on CLSI M24-A.

in recent years, N. elegans was first identified in Germany in 2005 (10) and classified as a N. nova complex, alongside N. africana, N. aobensis, N. kruczakiae, N. nova, and N. veterana (11). Reported cases of infection with N. elegans are limited, with just 13 reported to date (Table 2) (12-23). Little is known about the clinical microbiological characteristics of N. elegans infections. Therefore, we reviewed previous reports as well as the present case. Twelve of the 14 total cases (85.7%) were reported from East Asia, of which 9 (64.2%) were reported from Japan, followed by 1 (7.1%) each from South Korea, China, and Taiwan. Outside Asia, one case each was reported in Spain and Germany. Although the factors responsible for these regional differences remain to be determined, N. elegans infection appears to be more prevalent in East Asia, especially in Japan, than in other regions. Previous studies have shown that the distribution of Nocardia species varies according to the region (15, 24), probably due to climatic and geographical diversity.

The majority of *N. elegans* cases were immunocompromised (10/12 cases, 83.3%). The lungs were the most common site (12/14 cases, 85.7%), followed by the central nervous system, skin and soft tissue (2/14 cases, 14.2%), and disseminated infection (4/14 cases, 28.5%). Antimicrobial susceptibility patterns were highly variable among different *Nocardia* species, and some species showed resistance to ST. Therefore, it is important to know the susceptibility profile of each *Nocardia* species. Most of the *N. elegans* cases for which antimicrobial susceptibility was described were susceptible to ST (9/10 cases, 90%). Imipenem (6/6, 100%), AMK (4/4, 100%), and clarithromycin (CAM, 5/5, 100%) were also effective against isolated *N. elegans*, while amoxicillin/clavulanate (AMPC/CVA, 1/6, 16.6%) and ciprofloxacin (0/6, 0%) were often ineffective. Previous *N. elegans* cases were treated with a combination therapy of ST and MEPM, imipenem/cilastatin, AMK, minocycline, or monotherapy with ST or CAM. The lung and skin lesions improved with antimicrobial therapy alone. However, it should be noted that surgical treatment was required in cases of endophthalmitis and brain abscess (19).

The patient did not improve on treatment with garenoxacin prior to admission. Clinical experience with quinolones in nocardiosis is limited (1). The susceptibility to quinolones differs widely among species of *Nocardia*, with about half of *N. farcinica* cases being susceptible, while almost all *N. cyriacigeorgica* and *N. nova* complex cases are resistant (25). As mentioned above, all six cases of *N. elegans* were resistant to ciprofloxacin. This suggests that quinolones are not suitable for the treatment of *N. elegans*.

The present patient suffered from a brain abscess. Although it is unclear which *Nocardia* species are more likely to cause brain abscesses, *N. asteroides*, *N. farcinica*, *N. cyriacigeorgica*, and *N. abscessus* have been commonly reported as causative species. (26, 27). The affinity to the central nervous system and virulence may influence the likelihood of brain abscesses. Two out of 12 case reports of *N. elegans* also showed brain abscesses without symptoms; therefore, a brain examination should be performed even in the absence of symptoms.

Molecular methods, such as 16S rRNA gene sequencing, are conventional approaches for the identification of *Nocardia* species. All previous *N. elegans* cases were confirmed by 16S rRNA gene sequencing. Recently, an increasing number of reports have demonstrated that MALDI-TOF MS is a rapid and simple technique for the identification of *Nocardia* species (28, 29). It was reported that *N. elegans* can also be identified by MALDI-TOF MS (18). However, the

Reference	Country	Age/ Sex	Underlying condition	Immuno- suppressive agents	Sites of infection	Susceptibil- ity of ST	Antibiotics	Surgery	Outcome
(9)	Germany	-	-	-	Lung	-	-	-	-
(11)	Japan	46/F	-	-	Bronchitis	-	-	-	-
(12)	Spain	26/M	Cystic fibrosis	None	Lung	-	MEPM+TOB	None	-
(13)	Korea	39/M	Kidney transplantation	mPSL, CyA, MMF	Lung	S	ST+AMK; IPM/ CS+AMK; AMPC/ CVA	None	Improved
(14)	Taiwan	51/M	Dermatomyositis	-	Lung	S	ST	None	Improved
(15)	Japan	66/F	None	None	Ankle joint	S	-	Bursectomy	-
(16)	Japan	69/M	Systemic lupus erythematosus	PSL	Lung, brain	S	MEPM+AMK+ST; ST+CAM	None	Improved
(17)	Japan	73/M	Still disease	PSL, CyA	Lung	R	IPM+AMK	None	Improved
(18)	Japan	73/M	Rheumatoid arthritis	PSL, TAC	Lung, endophthal- mitis, skin	S	ST; MINO+IPM/ CS: MINO; CAM	Ophthalmectomy	Improved
(19)	China	62/F	Diabetes mellitus	None	Skin, lung	-	PC+CFPM+ST; DOXY+PC+ST	None	Improved
(20)	Japan	57/F	Systemic lupus erythematosus	Steroid	Lung	S	ST	None	Improved
(21)	Japan	34/M	Acute leukemia, HSCT, GVHD	PSL, CyA	Lung	S	DRPM+ST; DRPM; CAM	None	Improved
(22)	Japan	69/F	Renal transplantation	PSL, TAC, MMF	Lung	S	ABPC/SBT; ST	None	Improved
Present case	Japan	53/M	Lung transplantation	PSL, TAC, MMF	Lung, brain	S	MEPM+ST; CTRX+ST; ST	Brain drainage	Improved

#### Table 2. Clinical and Microbiological Characteristics of Reported Nocardia elegans Infections.

ABPC/SBT: ampicillin/sulbactam, AMK: amikacin, AMPC/CVA: amoxicillin/clavulanate, CAM: clarithromycin, CFPM: cefepime, CTRX: ceftriaxone, CyA: cyclosporine A, DOXY: doxycycline, DRPM: doripenem, F: female, GVHD: graft versus host disease, HSCT: hematopoietic stem cell transplant, IPM/CS: imipenem/cilastatin, M: male, MEPM: meropenem, MINO: minocycline, MMF: mycophenolate mofetil, mPSL: methylprednisolone, PC: penicillin, PSL: prednisolone, R: resistant, S: susceptible, ST: sulfamethoxazole-trimethoprim, TAC: tacrolimus, TOB: tobramycin



**Figure 3.** Clinical course of the patient. Clinical symptoms improved with treatment with ST and MEPM. However, drainage was necessary for the enlarged brain abscess. Ultimately, the patient was treated with ST+CTRX followed by ST monotherapy according to susceptibility. AMK: amikacin, CRP: C-reactive protein, CT: computed tomography, CTRX: ceftriaxone, FLAIR: fluid-attenuated inversion recovery, MEPM: meropenem, MRI: magnetic resonance imaging, ST: sulfamethoxazole-trimethoprim. Doses of ST are presented in equivalents of trimethoprim.

isolate in our case was identified as N. nova by MALDI-TOF MS with a score value of 2.07-2.18 but was identified as N. elegans by 16S rRNA gene sequencing. N. elegans was the eighth candidate, with a lower score of 1.88 on MADLI-TOF MS. Tajima et al. also reported that the N. elegans isolate identified by 16S rRNA gene sequencing had a MALDI-TOF MS score of 1.84 for N. nova and 1.61 for N. elegans (22). Regarding the accuracy of species identification by MALDI-TOF MS, 76% of the 312 Nocardia strains were able to be accurately identified at the species level, but 14% could only be identified at the complex level, and 7% could not be identified. Among the N. nova complex, N. veterana can be identified at the species level, but N. nova and N. elegans can only be identified at the complex level (28). Another report showed that, according to a multilocus sequence analysis, N. elegans and N. nova are highly similar phylogenetically, implying that it may be difficult to discriminate between them using MALDI-TOF MS (11). In addition, the reference database MBT Compass library: Ver.9.0.0.0 (8468MSPs) used in this study included only one strain of N. elegans. These facts suggest that N. elegans may be underdiagnosed using MALDI-TOF MS. Increasing the number of reference strains in the MALDI-TOF MS database will lead to the more accurate identification of N. elegans.

Although *N. elegans* is considered a rare causative species, whether or not it is also rare in the environment, whether it is less likely to cause infection in humans, or whether it is underestimated due to difficulty in identification in the clinical setting remains unclear. Further studies will be required to evaluate the prevalence of *N. elegans* in strains identified as *N. nova* by MALDI-TOF MS.

In conclusion, patients after solid organ transplantation are at risk of contracting *Nocardia* infections, and lung transplantation was suggested to carry a markedly higher risk of nocardiosis than other solid organ transplantations. We encountered a lung transplant recipient with lung and cerebral nocardiosis caused by *N. elegans*. Although *N. elegans* is a rare causative microorganism, it is important to understand the clinical microbiological characteristics of each *Nocardia* species.

#### The authors state that they have no Conflict of Interest (COI).

### References

- Wilson JW. Nocardiosis: updates and clinical overview. Mayo Clin Proc 87: 403-407, 2012.
- Restrepo A, Clark NM; Infectious Diseases Community of Practice of the American Society of Transplantation. Nocardia infections in solid organ transplantation: guidelines from the Infectious Diseases Community of Practice of the American Society of Transplantation. Clin Transplant 33: e13509, 2019.
- Husain S, McCurry K, Dauber J, Singh N, Kusne S. Nocardia infection in lung transplant recipients. J Heart Lung Transplant 21: 354-359, 2002.
- 4. Goodlet KJ, Tokman S, Nasar A, Cherrier L, Walia R, Nailor MD.

Nocardia prophylaxis, treatment, and outcomes of infection in lung transplant recipients: a matched case-control study. Transpl Infect Dis **23**: e13478, 2021.

- Khan BA, Duncan M, Reynolds J, Wilkes DS. Nocardia infection in lung transplant recipients. Clin Transplant 22: 562-566, 2008.
- **6.** Poonyagariyagorn HK, Gershman A, Avery R, et al. Challenges in the diagnosis and management of *Nocardia* infections in lung transplant recipients. Transpl Infect Dis **10**: 403-408, 2008.
- Peleg AY, Husain S, Qureshi ZA, et al. Risk factors, clinical characteristics, and outcome of *Nocardia* infection in organ transplant recipients: a matched case-control study. Clin Infect Dis 44: 1307-1314, 2007.
- Hemmersbach-Miller M, Stout JE, Woodworth MH, Cox GM, Saullo JL. *Nocardia* infections in the transplanted host. Transpl Infect Dis 20: e12902, 2018.
- Okamoto K, Santos CAQ. Management and prophylaxis of bacterial and mycobacterial infections among lung transplant recipients. Ann Transl Med 8: 413, 2020.
- Yassin AF, Brenner S. *Nocardia elegans* sp. nov., a member of the *Nocardia vaccinii* clade isolated from sputum. Int J Syst Evol Microbiol 55: 1505-1509, 2005.
- Tamura T, Ohji S, Ichikawa N, et al. Reclassification of *Nocardia* species based on whole genome sequence and associated phenotypic data. J Antibiot (Tokyo) **71**: 633-641, 2018.
- 12. Watanabe K, Shinagawa M, Amishima M, et al. First clinical isolates of *Nocardia carnea*, *Nocardia elegans*, *Nocardia paucivorans*, *Nocardia puris* and *Nocardia takedensis* in Japan. Nihon Ishinkin Gakkai Zasshi (Jpn J Med Mycol) 47: 85-89, 2006.
- Barrio MI, Martínez MC, Prados C, Girón RM, Maiz L, Martínez MT; Grupo de Fibrosis Quística de Neumomadrid. Isolation of *Nocardia* species in patients with cystic fibrosis. Arch Bronconeumol 44: 109-112, 2008.
- 14. Park KH, Ko SY, Oh R, et al. A case of lung abscess caused by *Nocardia elegans* in a kidney transplantation recipient. Infect Chemother 40: 116-120, 2008 (in Korean).
- 15. Liu WL, Lai CC, Ko WC, et al. Clinical and microbiological characteristics of infections caused by various *Nocardia* species in Taiwan: a multicenter study from 1998 to 2010. Eur J Clin Microbiol Infect Dis 30: 1341-1347, 2011.
- Masaki T, Ohkusu K, Ezaki T, Miyamoto H. *Nocardia elegans* infection involving purulent arthritis in humans. J Infect Chemother 18: 386-389, 2012.
- 17. Ueda Y, Yamamoto K, Watanabe K, Yamashita H, Ohmagari N, Mimori A. [Obstructive pneumonia and brain abscess due to *No-cardia elegans* in a patient with systemic lupus erythematosus]. Kansenshogaku Zasshi (J Jpn Assoc Infect Dis) 88: 282-287, 2014 (Japanese).
- 18. Ooi Y, Shiba H, Nagai K, et al. Lung *Nocardia elegans* infection diagnosed on matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF MS). Intern Med 53: 2111-2113, 2014.
- 19. Nakamura I, Nagakura T, Fujita H, Fukusima S, Gonoi T. Nocardia elegans infection: a case report and literature review. Int J Infect Dis 54: 15-17, 2017.
- 20. You Y, Chen W, Zhong B, Song Z, Yang X. Disseminated nocardiosis caused by *Nocardia elegans*: a case report and review of the literature. Infection 46: 705-710, 2018.
- Kobashi Y, Kittaka M, Shirai R, Kato S, Oka M. Clinical analysis of pulmonary nocardiosis in a tertiary hospital. Am J Infect Dis 14: 51-56, 2018.
- 22. Tajima K, Okuyama S, Terada T, et al. Clarithromycin as an alternative and prophylactic agent in a hematopoietic stem cell transplantation patient. Am J Case Rep 22: e931731, 2021.
- 23. Watanabe C, Kimizuka Y, Fujikura Y, et al. Mixed infection of Cytomegalovirus and pulmonary nocardiosis caused by *Nocardia elegans* diagnosed using nanopore sequencing technology: a case re-

port. Intern Med 2021.

- Saubolle MA, Sussland D. Nocardiosis: review of clinical and laboratory experience. J Clin Microbiol 41: 4497-4501, 2003.
- 25. Hamdi AM, Fida M, Deml SM, Abu Saleh OM, Wengenack NL. Retrospective analysis of antimicrobial susceptibility profiles of *Nocardia* species from a tertiary hospital and reference laboratory, 2011 to 2017. Antimicrob Agents Chemother **64**: e01868-19, 2020.
- 26. Anagnostou T, Arvanitis M, Kourkoumpetis TK, Desalermos A, Carneiro HA, Mylonakis E. Nocardiosis of the central nervous system: experience from a general hospital and review of 84 cases from the literature. Medicine (Baltimore) 93: 19-32, 2014.
- 27. Corsini Campioli C, Castillo Almeida NE, O'Horo JC, et al. Clinical presentation, management, and outcomes of patients with brain

abscess due to *Nocardia* species. Open Forum Infect Dis 8: ofab 067, 2021.

- 28. Body BA, Beard MA, Slechta ES, et al. Evaluation of the Vitek MS v3.0 matrix-assisted laser desorption ionization-time of flight mass spectrometry system for identification of *Mycobacterium* and *Nocardia* species. J Clin Microbiol 56: e00237-18, 2018.
- 29. Marín M, Ruiz A, Iglesias C, et al. Identification of *Nocardia* species from clinical isolates using MALDI-TOF mass spectrometry. Clin Microbiol Infect 24: 1342.e5-e8, 2018.

The Internal Medicine is an Open Access journal distributed under the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License. To view the details of this license, please visit (https://creativecommons.org/licenses/by-nc-nd/4.0/).

© 2023 The Japanese Society of Internal Medicine Intern Med 62: 431-437, 2023