Unusual presentation of *Nocardia abscessus* infection in an immunocompetent patient

Akshay Khatri¹*, Michael J. Esposito² and Robin Koshy³

**Abstract**

**Introduction.** *Nocardia* infections are being increasingly reported in both immunocompetent and immunocompromised patients. We describe a case of *Nocardia abscessus* infection with an atypical presentation in an immunocompetent patient.

**Case Presentation.** A previously healthy 47-year-old gentleman presented with hiccups and paroxysmal spasms. Imaging revealed a pulmonary nodule, for which he underwent surgical resection. Pathologic evaluation demonstrated evidence of local inflammation, with growth of *Nocardia abscessus* on tissue cultures.

**Conclusion.** *Nocardia abscessus* may have atypical presentations in immunocompetent patients. Further research is needed to understand the factors leading to *Nocardia* infections in immunocompetent patients.

**INTRODUCTION**

The genus *Nocardia* contains Gram-positive, partially acid-fast, aerobic, catalase-positive, non-motile branching rod-shaped bacteria [1]. They have been isolated from multiple environmental sources – soil, rotting vegetation, freshwater and saltwater [2]. They are seen to cause acute granulomatous inflammation in both animals and humans [1, 2].

*Nocardia* infections are common in those with underlying conditions (cancer, diabetes mellitus, chronic obstructive pulmonary disease) and congenital/acquired immune-deficiency (corticosteroid therapy, human immunodeficiency virus [HIV] infection, autoimmune disease, IgG deficiency) [2–6]. They can have diverse clinical presentations – pulmonary, cutaneous, neurologic, cardiac, ophthalmologic and disseminated manifestations [1, 2].

*Nocardia* species are differentiated using different biochemical and molecular testing modalities (including 16S rRNA gene sequencing) [2, 4, 7, 8]. Members of *Nocardia asteroides* complex and *Nocardia nova* complex are responsible for the majority of human infections [1, 2]. *Nocardia abscessus* was first characterized as a distinct species in 2000 and found to be associated with human disease [7].

In this case report, we describe an unusual presentation of *Nocardia abscessus* infection in an immunocompetent patient. We also review the literature related to prior well-reported *Nocardia abscessus* infections.

**CASE REPORT**

A 47-year-old male initially presented to the office of his primary care physician (PCP) with a 2 week history of acute-onset, episodic hiccups and paroxysmal spasms. The symptoms were relieved with meals and assuming a supine posture. Review of systems was otherwise negative. Use of metoclopramide and chlorpromazine provided minimal symptom relief.
He had no significant past medical or surgical history. Family history was remarkable for lung cancer, cardiac disorders and kidney disorders in grandparents (details not known). He resided with his family in Connecticut. He worked as a construction worker and reported inhalational exposure to dust and chemicals at his workplace. He was a former smoker (0.5 packs/day for 15 years, quit 5 years prior to symptoms). He reported ongoing occasional alcohol use and denied recreational drug use.

His physical examination was noted to be unremarkable. Laboratory evaluation revealed white blood cell count (WBC) 5460 K µl⁻¹ (N:3.80–10.50) with normal differential, haemoglobin 14.6 g dl⁻¹ (N: 13.0–17.0) and platelets 297 K µl⁻¹ (150–400). He had unremarkable renal, metabolic and hepatic laboratory test results. Abdominal ultrasound did not reveal any acute pathology. Chest X-ray showed a vague density in the lingular lobe of the left lung. Computed tomography (CT) scan of the chest revealed a 2.1×2.6×5.4 cm soft tissue nodular density with serpiginous internal enhancement (likely pulmonary vascular formation).

He was referred to Cardio-Thoracic Surgery for evaluation of the lung nodule. Whole-body positron-emission-tomography CT scan (PET-CT) using radiolabeled ¹⁸F-fluorodeoxyglucose (FDG) revealed an FDG-avid 3.1×2.0 cm lingular mass with intra-lesional fat. The intensity of metabolism of the lesion was concerning for malignancy, so the patient consented to surgical intervention. He underwent flexible bronchoscopy, video-assisted thoracoscopic surgery (VATS) and wedge resection of the left upper lobe. The lingular lesion was noted in the location corresponding to the CT scans, with no intra-operative evidence of malignancy. Wedge resection of the lingula was performed and sent for pathologic evaluation.

Gross examination of the specimen reported a 2.2×2.0×1.0 cm firm, tan, ill-defined lesion abutting the pleura. Histological examination revealed suppurative non-necrotizing granulomatous inflammation (Fig. 1), with epithelioid histiocytes, giant cells and micro-abscess formation (Fig. 2) and signs of chronic interstitial pneumonitis. Tissue cultures from lung nodule grew *Nocardia* species, that were identified as *Nocardia abscessus* by matrix-assisted laser desorption/ionization time-of-flight (MALDI-TOF) mass spectrometry (Bruker Corporation, Billerica, MA). No growth was noted in fungal and mycobacterial cultures.

Infectious Diseases was consulted for potential antimicrobial therapy. Further laboratory evaluation revealed erythrocyte sedimentation rate (ESR) 18 mm h⁻¹ (N:0–15 mm h⁻¹), C-reactive protein (CRP) <0.10 mg dl⁻¹ (N:0–0.40 mg dl⁻¹), negative HIV fourth-generation testing, negative interferon-γ release assay (QuantiFERON TB-Gold) testing, angiotensin converting enzyme (ACE) level 33 U l⁻¹ (N:14–82 U l⁻¹). T-cell subset testing revealed CD4 495 µl⁻¹ (N:489–1457 µl⁻¹), CD8 329 µl⁻¹ (N:142–740 µl⁻¹), CD4/CD8 ratio 1.50 (N:0.90–3.60). Immunoglobulin panel testing was unremarkable – serum IgA 230 mg dl⁻¹ (N:84–499 mg dl⁻¹), serum IgM 197 mg dl⁻¹ (N:35–242 mg dl⁻¹), serum IgG 935 mg dl⁻¹ (610–1660 mg dl⁻¹) and kappa-lambda free-light-chain ratio 1.51 (N:0.26–1.65). He underwent outpatient pulmonary function testing – it revealed normal spirometry, normal lung volumes, normal flow rates and normal diffusing capacity of lungs for carbon monoxide.
Antimicrobial susceptibility testing was performed at the reference laboratory (National Jewish Health Advanced Diagnostic Laboratories, Denver, CO) using broth microdilution techniques on the isolate and interpreted using Clinical and Laboratory Standards Institute guidelines [9]. On antimicrobial susceptibility testing, the isolate was noted to be sensitive to trimethoprim-sulfamethoxazole (TMP-SMX), linezolid, amikacin and tobramycin. He was prescribed TMP-SMX 800 mg-160mg (Bactrim DS) one tablet twice daily for 3 months. He self-discontinued this therapy after 5 weeks, due to symptoms of nausea and light-headedness. A repeat CT Chest performed 6 months after surgery showed stable post-surgical changes without evidence of new lesions. After discussion with the patient, it was decided to monitor him off antibiotic therapy. He continues to do well, without recurrence of symptoms.

DISCUSSION
In the past, diagnosis of *Nocardia* infections were challenging due to several factors – the time between symptom onset and microbiologic diagnosis, the time required for growth in cultures and the occasional co-isolation with other pathogens in the same specimen [5, 10]. However, the increasing reporting of *Nocardia* infections over the years can be attributed to increased organ transplantation (with concurrent use of immunosuppressive therapies), as well as better diagnostic modalities [5].

In comparison to other *Nocardia* species, *N. abscessus* infections have been less commonly reported in the literature [3–8, 10–26] (Table 1). *N. abscessus* infections have been seen to occur mostly in adults, in both immunocompetent and immunocompromised patients. Successful therapy of *N. abscessus* infections have been seen to involve combined antimicrobial and surgical techniques. The crude mortality rate for *N. abscessus* and *N. farcinica* infections (78.5%, relative risk of 3.89) is reported to be higher than other *Nocardia* species [6] – thus establishing a species-specific diagnosis and management plan is essential.

TMP-SMX has been used as standard therapy for *Nocardia* infection [2]. However, as the antibiotic sensitivities vary according to the species and geographic location, antimicrobial susceptibility testing should be performed [2]. Treatment duration depends upon the location and extent of disease [2]. *N. abscessus* is susceptible to ampicillin, amoxicillin-clavulanate, ceftriaxone, linezolid, amikacin; and resistant to ciprofloxacin and clarithromycin. Some species have resistance to imipenem [2]. It is important to note that breakthrough *Nocardia* infections can occur in patients receiving prophylactic lower-dose TMP-SMX [14].

Our patient had an atypical presentation of nocardiosis – *N. abscessus* growing in the lingular lobe likely caused pressure on the left phrenic nerve, resulting in hiccoughs and spasms. As noted in another case, he was at risk of acquiring *Nocardia* from his workplace (environmental dust exposure) and potentially due to his history of smoking [26]. Our evaluation did not reveal any underlying co-morbidities or immuno-suppressive conditions. Due to the ubiquitous environmental presence of *Nocardia*, it

Fig. 2. Haematoxylin and eosin-stained cross-section of the resected lesion (400× magnification), showing epithelioid histiocytes (red arrow), giant cells (yellow arrow) and microabscess formation (green arrow).
### Table 1. Prior documented cases of *N. abscessus* infections

| Sr. no. | Patient age/sex | Past medical history | Immune status | Clinical features | Clinical presentation | Relevant labs | Treatment | Reference |
|---------|-----------------|----------------------|---------------|-------------------|----------------------|--------------|-----------|-----------|
| 1       | 47 /M           | None                 | IMM           | Hiccoughs         | Lung mass            | WBC 5,460 K µl⁻¹ (N:3.80–10.50) ESR 10 mm h⁻¹ (N:0–15 mm/h) CRP <0.1 mg dl⁻¹ (N:0–0.4 mg dl⁻¹) | 1. Surgical excision 2. IV TMP-SMX | Current case |
| 2       | 24 /M           | None                 | IMM           | Post-traumatic infected swelling | Cutaneous (co-infection with *Blastomyces dermatitidis*) | NA | Antibiotics (details not known) | [10] |
| 3       | 60 /M           | None                 | IMM           | Temporal headaches | Brain abscess with intracranial internal carotid artery aneurysm | WBC 6×10³ /mm³ ESR 18 mm h⁻¹ (N:0–15 mm/hr) CRP <0.1 mg dl⁻¹ (N:0–0.4 mg dl⁻¹) | 1. Stereotactic aspiration of abscess 2. Infected aneurysm resection 3. Antibiotics a. IV CTX×4 weeks b. IV CTX×high dose TMP-SMX×6 weeks | [3] |
| 4       | 75 /F           | Seronegative RA (on immunosuppressive therapy) | ICS           | Subjective memory complaints | Disseminated (cutaneous, cerebral, pulmonary) | (a) Initial WBC 8.9×10⁸ /mm³ ESR 59 mm/hr CRP 12.7 mg dl⁻¹ (b) 1 week after admission: WBC 1.1×10⁹ /mm³ ESR 43 mm/hr CRP 23.3 mg dl⁻¹ | Antibiotics: a. IV TMP-SMX×1IVCTX×1 month b. IV LIN×IV MIER | [11] |
| 5       | 40 /M           | HIV/AIDS             | ICS           | Headache | Disseminated (cutaneous, cerebral, pulmonary) | CD4 +21 cells mm⁻³ HIV viral load 74,368 copies ml⁻¹ | Antibiotics: a. IV MIER+PO LIN×PO TMP-SMX×4 weeks b. IV CTX×PO DOX×PO TMP-SMX×6 months c. TMP-SMX PO×15 months | [12] |
| 6       | 33 /M           | HIV/AIDS             | ICS           | Fever | Disseminated (cutaneous, cerebral, pulmonary) | WBC 6×10⁹ /mm³ (Neutrophils 99%, lymphocytes: 2%) ESR 46 mm h⁻¹ CD4 +11 cells mm⁻³ | Antibiotics: a. TMP-SMX×CAPREOFLO×1 month b. IV CTX+TMP-SMX×3 weeks c. TMP-SMX PO | [13] |
| 7       | 50 /F           | Acute myeloid leukemia (S/P bone marrow transplant) | ICS           | Fever | Disseminated (cutaneous, cerebral, pulmonary, hepatic, lymph node) | NA | Antibiotics: a. PO TMP-SMX b. PO MBL×3 weeks c. PO TMP-SMX | [14] |
| 8       | 49 /M           | Metastatic squamous cell cancer of lung (S/P chemotherapy and cerebral irradiation) | ICS           | Soft tissue swelling | Subcutaneous (soft tissue abscess) | NA | Incision, drainage and antibiotic lavages | [4] |
| 9       | 54 /M           | Metastatic angiosarcoma (S/P chemotherapy) | ICS           | Febrile | Pulmonary with empyema thoracis | WBC 16.4×10³ /µl Urea nitrogen 190 mg dl⁻¹ (N:10–30) CRP 93 mg dl⁻¹ (N:0–8 mg dl⁻¹) | 1. Debridement 2. Chest tube insertion 3. Antibiotics a. CTX b. IMI-CIL | [15] |
| 10      | 37 /M           | None                 | IMM           | Headache | Brain abscess | WBC 15,500 µl⁻¹ ESR 10 mm h⁻¹ CRP 5 mg dl⁻¹ | 1. Surgical evacuation 2. Antibiotics a. IV CTX×IV TMP-SMX×IV LIN×5 weeks b. PO TMP-SMX×8 weeks | [16] |
| 11      | 64 /M           | Primary biliary cirrhosis and autoimmune hepatitis (S/P orthotopic liver transplant) Diabetes mellitus Chronic kidney disease | ICS           | Recurrent skin lesion | Cutaneous | NA | Antibiotics: a. Levofloxacin+PIP-TAZ b. PIP-TAZ c. CTX+TMP-SMX | [17] |
| 12      | 39 /M           | None                 | IMM           | Fever | Disseminated (cerebral, pulmonary) | NA | 1MD×CTX×Amikacin | [18] |
| Sr. no. | Patient age/sex | Past medical history | Immune status | Clinical features | Clinical presentation | Relevant labs | Treatment | Reference |
|---------|-----------------|----------------------|---------------|-------------------|----------------------|---------------|------------|-----------|
| 13      | 73 /M           | na                   | na            | Recurrent rashes   | Cardiac              | na            | na         | [19]      |
| 14      | 74 /M           | Hypertension         | IMM           | Headache          | Cerebral             | WBC 10000/μm³, CD4+ 108/μm³, CD4+/CD8+ ratio 0.5, Total Immunoglobulin (IgG) and IgG subclasses 1/2/3/4 low | 1. Aspiration of abscess 2. Excision of brain lesion 3. Antibiotics a. Cefotaxime×1 week b. CTX + Metronidazole + Steroids ×2 weeks c. CTX + DOX + TMP-SMX ×2 months d. IMI + DOX ×10 months e. DOX ×3 months | [20]      |
| 15      | 45 /F           | Marfan’s syndrome with aortic bioprosthesis | IMM | Hand abscess | Cutaneous (co-infection with methicillin-resistant coagulase-negative Staphylococcus) | na | LIN×1 month | [5]       |
| 16      | na              | na                   | na            | Cerebral          | na                   | na            | 1. Abscess aspiration 2. Antibiotics a. MER + LIN b. TMP-SMX | [21]      |
| 17      | na              | na                   | na            | Pulmonary         | na                   | na            | Antibiotics: a. IMI + TMP-SMX b. IMI + Levofloxacin c. TMP-SMX | [21]      |
| 18      | na              | na                   | na            | Pulmonary         | na                   | na            | Antibiotics: a. IMI + TMP-SMX b. CTX + TMP-SMX c. TMP-SMX | [21]      |
| 19      | na              | na                   | na            | Pulmonary         | na                   | na            | Antibiotics: a. TMP-SMX b. TMP-SMX + MER c. Levofloxacin | [21]      |
| 20      | na              | na                   | na            | Disseminated (Neurologic, Pulmonary) | na | na | 1. Brain abscess aspiration 2. Implantation of Ommaya reservoir 3. Antibiotics a. CTX + TMP-SMX b. CTX + TMP-SMX + Intrathecal Amikacin c. Metoclopramide | [21]      |
| 21      | 65 /M           | COPD                 | IMM           | Pulmonary         | na                   | na            | TMP-SMX | [6]       |
| 22      | 65 /M           | HBV COPD             | ICS           | Pulmonary         | na                   | na            | na         | [6]       |
| 23      | 77 /M           | COPD                 | ICS           | Pulmonary         | na                   | na            | TMP-SMX | [6]       |
| 24      | 76 /M           | COPD                 | ICS           | Pulmonary         | na                   | na            | None      | [6]       |
| 25      | 69 /M           | COPD                 | IMM           | Pulmonary         | na                   | na            | TMP-SMX | [6]       |
| 26      | 83 /F           | COPD                 | IMM           | Pulmonary         | na                   | na            | Levofloxacin | [6]      |
| 27      | 56 /M           | na                   | na            | Prosthetic knee joint abscess | Endoprosthetic infection | na | na | | [7]       |
| 28      | 48 /F           | na                   | na            | Pain, redness, swelling from eye | Ocular (keratitis) | na | a. Topical Amikacin b. PO TMP-SMX ×6 weeks | [22]      |
| Sr. no. | Patient age/sex | Past medical history | Immune status | Clinical features | Clinical presentation | Relevant labs | Treatment | Reference |
|-------|-----------------|----------------------|---------------|------------------|----------------------|--------------|------------|----------|
| 29    | 20 /M           | None                 | IMM          | Pain, redness, decreased vision in eye | Ocular (keratitis) | NA          | a. Topical Moxifloxacin  
b. Topical TMP-SMX | [23]     |
| 30    | 56 /M           | Systemic lupus erythematosus | ICS          | NA               | Pulmonary             | NA          | NA         | [8]      |
| 31    | 62 /M           | NA                   | NA           | NA               | Brain abscess         | NA          | NA         | [8]      |
| 32    | 69 /M           | RA                   | ICS          | NA               | Pulmonary†             | NA          | NA         | [8]      |
| 33    | 42 /M           | HBV                  | ICS          | NA               | N.a.n.               | NA          | NA         | [8]      |
| 34    | 84 /M           | Lung cancer          | ICS          | NA               | N.a.n.               | NA          | NA         | [8]      |
| 35    | 56 /M           | Complete knee endoprosthesis | IMM         | NA               | Joint abscess         | NA          | NA         | [8]      |
| 36    | 7 /F            | Idiopathic pulmonary hemosiderosis | CS therapy | Cough  
Paravertebral x 20 days | Pulmonary | WBC 20.6×10^3/µl (N=4–10×10^3/µl)  
IgG 5.40 g/l  
IgA 0.81 g/l  
IgM 1.87 g/l | LIN x 3 weeks | [24]     |
| 37    | 54 /M           | Atypical anti-glomerular basement membrane glomerulonephritis (S/P plasmapheresis, IVCS, cyclophosphamide) | CS therapy | Fever  
Acute stabbing right chest pain  
Fatigue  
Gross hematuria | Pulmonary | WBC 5.3×10^3/µl (N=4.3–10.3×10^3/µl)  
Neutrophil count 4.87×10²/µl (N=2.1–6.1×10²/µl),  
ESR 1.3mm/hr  
CRP 7.78 mg/dl (0–0.8 mg/dl),  
Procalcitonin 0.4 ng/ml (0–0.1 ng/ml),  
Blood urea nitrogen (BUN) 128 mg/dl (6–20 mg/dl),  
Creatinine 5.99 mg/dl (0.67–1.17 mg/dl) | Antibiotics:  
a. IV TMP-SMX +IMI CIL x 1 month  
b. PO TMP-SMX x minimum 6 months | [25]     |
| 38    | 40 /M           | Active smoker         | ICS²         | Headache  
Subacute left brachiofacial deficit | Brain abscess | High anti-GM-CSF autoantibody titre in serum  
(Previously undiagnosed) | 1. Cerebral abscess drainage  
2. Antibiotics:  
a. IV MER (x 6 weeks)+high dose PO TMP-SMX x 5 weeks  
b. High dose PO TMP-SMX x 12 months  
c. PO TMP-SMX (ongoing) | [26]     |

Key: AIDS: acquired immunodeficiency syndrome; AMS: altered mental status; anti-GM-CSF: anti-granulocyte-macrophage colony-stimulating factor; CD4+: CD4+ lymphocyte count; COPD: chronic obstructive pulmonary disease; CRP: C-reactive protein; CS: corticosteroid; CTX: Ceftriaxone; DOX: Doxycycline; ESR: erythrocyte sedimentation rate; F: female; HIV: human immunodeficiency virus; ICS: immune compromised patient; IMI: Imipenem; IMI-CL: Imipenem-GA state; IMM: immune competent patient; IV: intravenous; LIN: lincomycin; M: male; MER: Meropenem; NA: not available; PIP-TAZ: Piperacillin- tazobactam; PO: oral; RA: rheumatoid arthritis; TMP-SMX: trimethoprim-sulfamethoxazole; S/P: status post (after treatment with); WBC: white blood cell count.

²The authors in this study considered this patient to be immune compromised due to the presence of anti-GM-CSF (granulocyte-macrophage colony-stimulating factor) autoantibodies that were detected at time of Nocardia infection diagnosis.
may be difficult to distinguish colonization versus true infection – particularly in sputum/skin specimens [21]. Our suspicion for colonization was low, given the isolation of *N. abscessus* in an operative specimen and the findings of necrotizing granulomas on histology. It is unclear as to why he developed nocardiosis – further research is needed to investigate the pathophysiologic mechanisms and risk factors of *Nocardi*a infections in immunocompetent patients.

*N. abscessus* may have atypical presentations in immunocompetent patients and require combined medical and surgical interventions to achieve optimal outcomes. Further research is needed to understand the factors leading to *Nocardi*a infections in immunocompetent patients.

**Funding information**
This work received no specific grant from any funding agency.

**Acknowledgements**
The authors would like to gratefully acknowledge the contributions of late Dr Tawfiqul Bhuiya, our colleague in the Department of Pathology, for his initial pathologic review of the case.

**Author contributions**
A.K. and R.K. were involved in conceptualization, writing of original draft and review and editing of subsequent drafts. M.E. assisted with acquisition and initial pathologic review of the case. The authors would like to gratefully acknowledge the contributions of late Dr Tawfiqul Bhuiya, our colleague in the Department of Pathology, for his interpretation of histologic slides. All authors reviewed the final manuscript prior to submission.

**Conflicts of interest**
The authors declare that there are no conflicts of interest.

**Ethical statement**
The research met our institutional definition of a case report (a medical chart review of three or fewer patients), and thus institutional research board review was not needed. Written informed consent was obtained from the patient.

**References**
1. Fatahi-Bafghi M. Nocardiosis from 1888 to 2017. Microb Pathog 2018;114:369–384.
2. Welsh O, Vera-Cabrera L, Salinas-Carmona MC. Current treatment for nocardia infections. Expert Opin Pharmacother 2013;14:2387–2398.
3. Farran Y, Antony S. Nocardia abscessus-related intracranial aneurysm of the internal carotid artery with associated brain abscess: A case report and review of the literature. J Infect Public Health 2016;9:358–361.
4. Daesschelig G, Fetouh Yassin AA, Franke A, Kramer A, Schaal KP. Unusual infections: Femoral abscess due to *Nocardi*a *abscessus* in a patient suffering from metastatic peripheral bronchial carcinoma and hygienic consequences. GMS Krankenhyy Interdiszip 2011;6:Doc03.
5. Hémar V, Danjean MP, Imbert Y, Rispal P. Retrospective analysis of nocardiosis in a general hospital from 1998 to 2017. Med Mal Infect 2018;48:516–525.
6. Muñoz J, Mirelis B, Aragón LM, Gutiérrez N, Sánchez F, et al. Clinical and microbiological features of nocardiosis 1997-2003. J Med Microbiol 2007;56:545–550.
7. Yassin AF, Rainey FA, Mendrock U, Brzezinka H, Schaal KP. *Nocardi*a *abscessus* sp. nov. Int J Syst Evol Microbiol 2000;50 Pt 4:1487–1493.
8. Kageyama A, Yazawa K, Kudo T, Taniguchi H, Nishimura K, et al. First isolates of *Nocardi*a *abscessus* from humans and soil in Japan. Nihon Ishinkin Gakkai Zasshi 2004;45:17–21.
9. CLSI. Performance Standards for Susceptibility Testing of Mycobacteria, Nocardia spp., and Other Actinomycetes. 1st ed. CLSI guidelines M62. Wayne, PA: Clinical and Laboratory Standards Institute; 2018.
10. Horrè R, Schumacher G, Marklein G, Stratmann H, Wardelmann E, et al. Mycetoma due to *Pseudallescheria boydii* and co-isolation of *Nocardi*a *abscessus* in a patient injured in road accident. Med Mycol 2002;40:525–527.
11. Boccardi V, Croce MF, Ruggiero C, Aisa G, Tosti A, et al. Subjective memory complaints and depression as clinical symptoms of disseminated nocardiosis by *Nocardi*a *abscessus*. Geriatr Gerontol Int 2016;16:1167–1168.
12. Sherbuk J, Saly D, Barakat L, Ogbaru O. Unusual presentation of disseminated *Nocardi*a *abscessus* infection in a patient with AIDS. BMJ Case Rep 2016;2016:bcr2016215649.
13. Diego C, Ambrosioni JC, Abel G, Fernando B, Tomás O, et al. Disseminated nocardiosis caused by *Nocardi*a *abscessus* in an HIV-infected patient: first reported case. AIDS 2005;19:1330–1331.
14. Hino Y, Doki N, Seno Y, Sekiya N, Kurosawa S, et al. Disseminated nocardiosis after unrelated bone marrow transplantation. Transpl Infect Dis 2016;18:942–945.
15. Lai CC, Tsai HY, Ruan SY, Liao CH, Hsueh PR. Fatal pneumonia and empyema thoracis caused by imipenem-resistant *Nocardi*a *abscessus* in a cancer patient. J Microbiol Immunol Infect 2015;48:706–708.
16. Al Tawfiq J, Maytan M, Memish ZA. *Nocardi*a *abscessus* brain abscess in an immunocompetent host. J Infect Public Health 2013;6:158–161.
17. Kober P, Gozdowska J, Sawicka M, Ślusowska K, Pacholczyk M, et al. Cutaneous nocardiosis in a liver transplant recipient - case report. Pol Merkur Lekarski 2020;48:108–111.
18. Pytigorskaya N, Brugieres P, Hodel J, Mekontso Dessap A, Gaston A. What is your diagnosis? *Nocardi*a *abscessus* infection. J Neurol 2010;37:192–195.
19. Wellhausen N, Pietzcker T, Kern WV, Essig A, Marre R. Expanded spectrum of *Nocardi*a species causing clinical nocardiosis detected by molecular methods. Int J Med Microbiol 2002;292:277–282.
20. Marchandin H, Eden A, Jean-Pierre R, Reyes J, Jumaa-Blak E, et al. Molecular diagnosis of culture-negative cerebral nocardiosis due to *Nocardi*a *abscessus*. Diagn Microbiol Infect Dis 2006;55:237–240.
21. Mazzaferrri F, Cordioli M, Segato E, Adami I, Maccaraco L, et al. Nocardia infection over 5 years (2011-2015) in an Italian tertiary care hospital. New Microbiol 2018;41:136–140.
22. Reddy AK, Garg P, Kaur I. Spectrum and clinicomicrobiological profile of *Nocardi*a keratitis caused by rare species of *Nocardi*a identified by 16S rRNA gene sequencing. Eye (Lond) 2010;24:1259–1262.
23. Galor A, Hall GS, Procop GW, Tuohy M, Millstein ME, et al. Rapid species determination of *Nocardi*a keratitis using pyrosequencing technology. Am J Ophthalmol 2007;143:182–183.
24. Qin L, Zhang FZ, Yang TY, Tao XF, Tang LF. Pulmonary Nocardia infection in a child with idiopathic pulmonary hemosiderosis. *BMC Pulm Med* 2021;21:182.

25. Ismailov R, Koray N, İnal N, *et al*. Pulmonary nocardiosis caused by nocardia abscessus mimicking pulmonary thromboembolism in a patient with atypical anti-glomerular basement membrane glomerulonephritis. *Tuberk Toraks* 2021;69:237–241.

26. Berthoux C, Mailhe M, Vély F, Gauthier C, Mège J-L, *et al*. Granulocyte macrophage colony-stimulating factor-specific autoantibodies and cerebral nocardia with pulmonary alveolar proteinosis. *Open Forum Infect Dis* 2021;8:ofaa612.

---

**Five reasons to publish your next article with a Microbiology Society journal**

1. The Microbiology Society is a not-for-profit organization.
2. We offer fast and rigorous peer review – average time to first decision is 4–6 weeks.
3. Our journals have a global readership with subscriptions held in research institutions around the world.
4. 80% of our authors rate our submission process as ‘excellent’ or ‘very good’.
5. Your article will be published on an interactive journal platform with advanced metrics.

Find out more and submit your article at microbiologyresearch.org.