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CASE REPORT

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

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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.

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Abbreviations: CRP, C-reactive protein; CT, computed tomography; ESR, erythrocyte sedimentation rate; FDG, ^{18}F-fluorodeoxyglucose; HIV, human immunodeficiency virus; MALDI-TOF, matrix-assisted laser desorption/ionization time-of-flight; PCP, primary care physician; PET-CT, positron-emission-tomography CT scan TMP-SMX, trimethoprim- sulfamethoxazole; VATS, video-assisted thoracoscopic surgery; WBC, white blood cell count.

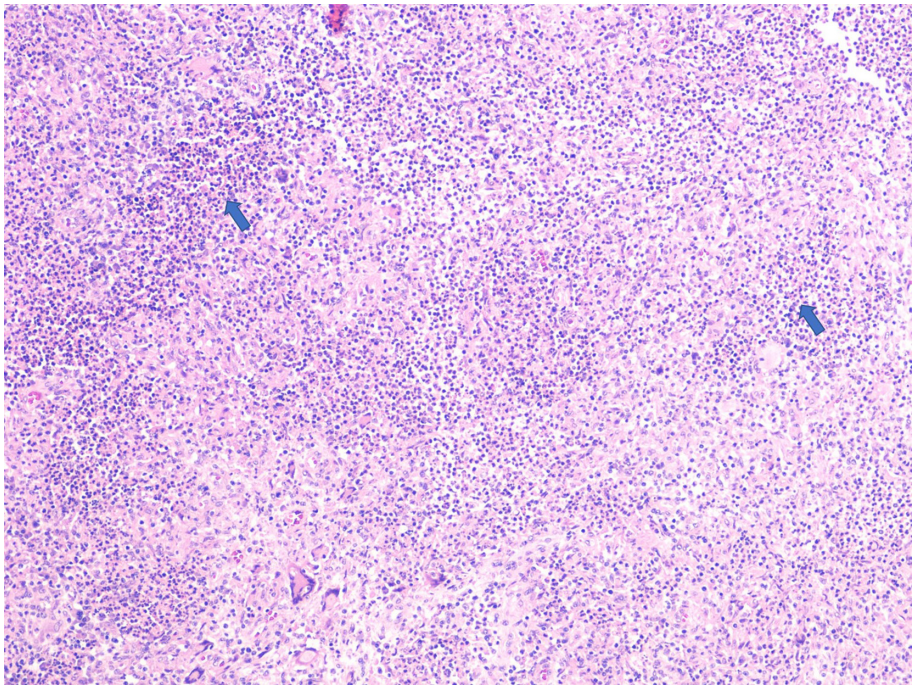

Fig. 1. Haematoxylin and eosin-stained cross-section of the resected lesion (100× magnification) showing a diffuse background of suppurative non-necrotizing granulomatous inflammation (blue arrows).

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 ^{18}F-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-Ɣ 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.

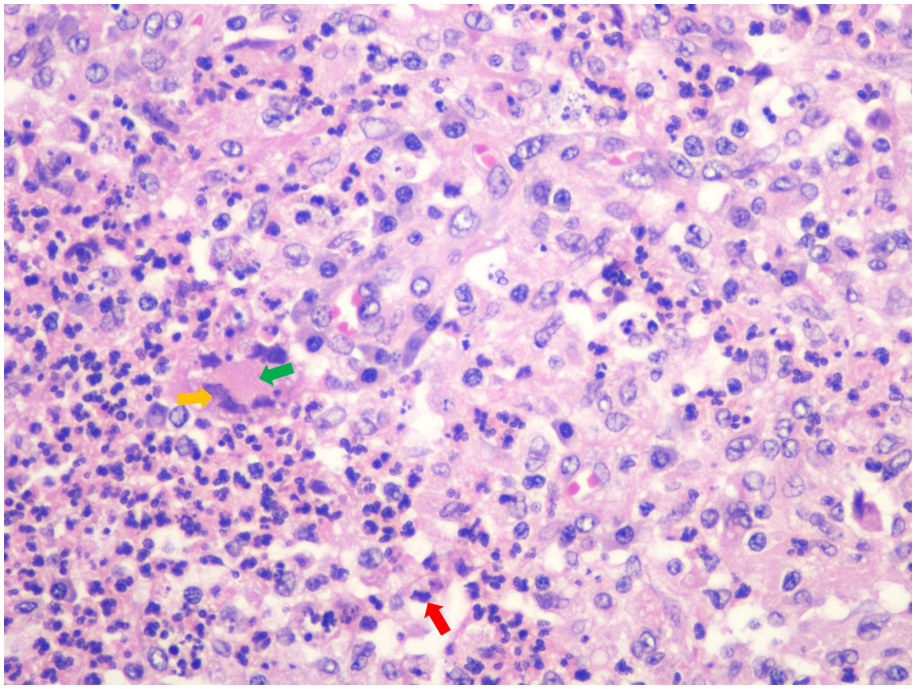

Fig. 2. Haematoxylin and eosin-stained cross-section of the resected lesion (400× magnification), showing epithelioid histiocytes (red arrow), giant cells (yellow arrow) and microabscess formation (green arrow).

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

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 18 mm h^{-1} (N:0–15 mm/hr) CRP <0.1 mg dl^{-1} (N:0–0.4 mg dl^{-1})	1. Surgical excision 2. PO TMP-SMX	Current case
2	24/M	None	IMM	Post-traumatic infected swelling	Cutaneous (co-infection with *Pseudallescheria boydii*)	NA	Antibiotics (details not known)	[10]
3	60/M	None	IMM	Temporal headaches Fatigue Memory loss Behavioural abnormalities ×2–3 weeks	Brain abscess with intra-cranial internal carotid artery aneurysm	WBC 4×10^9 l^{-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 Asthenia Depression Worsening AMS ×2 months	Disseminated (cutaneous, cerebral, pulmonary, hepatic, pancreatic)	(a) Initial: WBC 8.39×10^3/mm^3 ESR 59 mm/hr CRP 12.7 mg dl^{-1} (b) 1 week after admission: WBC 11.44×10^3/mm^3 ESR 83 mm/hr CRP 23.3 mg dl^{-1}	Antibiotics: a. IV TMP-SMX +IV CTX×1 month b. IV LIN+IV MER	[11]
5	40/M	HIV/AIDS	ICS	Headache Persistent AMS Generalized weakness Bowel/bladder incontinence ×2 months	Disseminated (cutaneous, cerebral, pulmonary)	CD4 +21 cells mm^{-3} HIV viral load 74368 copies ml^{-1}	Antibiotics: a. IV MER +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 Asthenia, malaise Weight loss Skin lesions ×2 months	Disseminated (cutaneous, cerebral, pulmonary)	WBC 6900 cells mm^{-3} (Neutrophils: 59%; Lymphocytes: 28%) ESR 46 mm h^{-1} CD4 +11 cells mm^{-3}	Antibiotics: a. TMP-SMX +Ciprofloxacin×1 month b. IV CTX +TMP SMX×2.5 months c. TMP-SMX PO	[13]
7	50/F	Acute myeloid leukaemia (S/P bone marrow transplant)	ICS	Fever Cough Fatigue Skin nodules	Disseminated (cutaneous, cerebral, pulmonary, hepatic, lymph node)	NA	Antibiotics: a. PO TMP-SMX b. IV IMI-CIL +TMP SMX×3 weeks c. PO TMP-SMX	[14]
8	49/M	Metastatic squamous cell cancer of lung (S/P chemotherapy and cerebral radiation)	ICS	Soft tissue swelling	Subcutaneous (soft tissue abscess)	NA	Incision, drainage and antiseptic lavages	[4]
9	54/M	Metastatic angiosarcoma (S/P chemotherapy)	ICS	Fever Chest pain Mild respiratory distress Purulent discharge from surgical site ×1 week	Pulmonary with empyema thoracis	WBC 16.49×10^9 l^{-1} Urea nitrogen 190 mg l^{-1} Creatinine 5.8 mg l^{-1} CRP 93 mg l^{-1} (N:<8 mg l^{-1})	1. Debridement 2. Chest tube insertion 3. Antibiotics: a. CTX b. IMI-CIL	[15]
10	37/M	None	IMM	Headache Dizziness Blurred vision ×3–4 weeks	Brain abscess	WBC 15200 μl^{-1} ESR 10 mm h^{-1} CRP 0.5 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	59/M	None	IMM	Fever Right hemihypoesthesia AMS	Disseminated (cerebral, pulmonary)	NA	IMI+CTX+Amikacin	[18]

Continued

Table 1. Continued

Sr. no.	Patient age/sex	Past medical history	Immune status	Clinical features	Clinical presentation	Relevant labs	Treatment	Reference
13	73/M	NA	NA	Pericarditis	Cardiac	NA	NA	[19]
14	34/M	Hypertension	IMM	Headache Right hemiparesis	Cerebral	WBC 10600/mm^3 CD4 +108/mm^3 CD4+/CD8 +T cell ratio 0.5 Total Immunoglobulin (Ig)G 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 Lymphangitis Inflammatory lymphadenitis	Cutaneous (co-infection with methicillin-resistant coagulase-negative *Staphylococcus*)	NA	LIN×1 month	[5]
16	NA	NA	NA	NA	Cerebral	NA	1. Abscess aspiration 2. Antibiotics: a. MER +LIN b. TMP-SMX	[21]
17	NA	NA	NA	NA	Pulmonary	NA	Antibiotics: a. IMI+TMP-SMX b. IMI +Levofloxacin c. TMP-SMX	[21]
18	NA	NA	NA	NA	Pulmonary	NA	Antibiotics: a. IMI+TMP-SMX b. CTX +TMP-SMX c. TMP-SMX	[21]
19	NA	NA	NA	NA	Pulmonary	NA	Antibiotics: a. TMP-SMX b. TMP-SMX +MER c. Levofloxacin	[21]
20	NA	NA	NA	NA	Disseminated (Neurologic, Pulmonary)	NA	1. Brain abscess aspiration 2. Implantation of Ommaya reservoir 3. Antibiotics: a. CTX +TMP-SMX b. CTX +TMPSMX+Intrathecal Amikacin c. Minocycline	[21]
21	65/M	COPD	IMM	NA	Pulmonary	NA	TMP-SMX	[6]
22	65/M	HIV COPD Solid tumour	ICS	NA	Pulmonary	NA	NA	[6]
23	77/M	COPD Solid tumour CS therapy	ICS	NA	Pulmonary	NA	TMP-SMX	[6]
24	76/M	COPD CS therapy	ICS	NA	Pulmonary	NA	None	[6]
25	69/M	COPD	IMM	NA	Pulmonary	NA	TMP-SMX	[6]
26	83/F	COPD	IMM	NA	Pulmonary	NA	Levofloxacin	[6]
27	56/M	NA	NA	Prosthetic knee joint abscess	Endoprosthetic infection	NA	NA	[7]
28	48/F	NA	NA	Pain, redness, watering from eye	Ocular (keratitis)	NA	a. Topical Amikacin b. PO TMP-SMX ×5 weeks	[22]

Continued

Table 1. Continued

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	HIV	ICS	NA	Nasal?	NA	NA	[8]
34	84/M	Lung cancer	ICS	NA	Nasal?	NA	NA	[8]
35	56/M	Complete knee endroprosthesis	IMM	NA	Joint abscess	NA	NA	[8]
36	7/F	Idiopathic pulmonary hemosiderosis CS therapy	ICS	Cough Purulent sputum x 20 days	Pulmonary	WBC $20.62\times10^{9}l^{-1}$ (N:4–$10\times10^{9}l^{-1}$) IgG 5.40 g l^{-1} IgA 0.81 g l^{-1} IgM 1.87 g l^{-1}	LIN x 3 weeks	[24]
37	54/M	Atypical anti-glomerular basement membrane glomerulonephritis (S/P plasmapheresis, IV CS, cyclophosphamide) CS therapy	ICS	Fever Acute stabbing right chest pain Fatigue Gross hematuria	Pulmonary	WBC 5.3×10^{3} μl^{-1} (N:4.3–10.3×10^{3} μl^{-1}) Neutrophil count 4.87×10^{3} μl^{-1} (N:2.1–6.1×10^{3} μl^{-1}), ESR 13 mm/hr CRP 7.78 mg dl^{-1} (0–0.8 mg dl^{-1}), procalcitonin 0.54 ng ml^{-1} (0–0.1 ng ml^{-1}), blood urea nitrogen (BUN) 128 mg dl^{-1} (6–20 mg dl^{-1}), creatinine 5.09 mg dl^{-1} (0.67–1.17 mg dl^{-1})	Antibiotics: a. IV TMP-SMX +IMI CIL x 1 month b. PO TMP-SMX x minimum 6 months	[25]
38	40/M	Active smoker	ICS*	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 +T-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-CIL: Imipenem-Cilastatin; IMM: immune-competent patient; IV: intravenous; LIN: linezolid; 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.
*The 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 *Nocardia* 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 *Nocardia* infections in immunocompetent patients.

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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 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.

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