CASE REPORT

Unusual presentation of disseminated *Nocardia abscessus* infection in a patient with AIDS

Jacqueline Sherbuk,¹ Danielle Saly,¹ Lydia Barakat,² Onyema Ogbuagu²

¹Yale-New Haven Hospital, New Haven, Connecticut, USA
²Section of Infectious Diseases, Yale University School of Medicine, New Haven, Connecticut, USA

Correspondence to
 Dr Onyema Ogbuagu,
 onyema.ogbuagu@yale.edu

Jacqueline Sherbuk and
 Danielle Saly are co-first
 authors of this article.

Accepted 5 July 2016

SUMMARY

A 40-year-old man with AIDS presented with symptoms of a chronic cough, subacute headache, generalised weakness with falls, urinary and faecal incontinence, and acute onset subcutaneous nodules. A chest CT scan showed multiple cavitary and nodular pulmonary infiltrates. MRI of his brain and spinal cord revealed innumerable ring-enhancing lesions. Pathological examination of the purulent material obtained from his subcutaneous lesions, as well as transbronchial tissue specimens obtained by biopsy, revealed beaded and branching Gram-positive rods, subsequently identified by 16S RNA sequencing to be *Nocardia abscessus* species. We observed an excellent therapeutic response to a combination antimicrobial therapy with resolution of the subcutaneous, pulmonary and central nervous system (CNS) lesions. Infections caused by *N. abscessus* are rare and typically occur in immunocompromised patients. In this article, we will review the presentation, diagnosis and treatment of *N. abscessus* infection.

BACKGROUND

In spite of the availability and widespread use of antiretroviral therapy (ART) in the USA, opportunistic infections (OIs) continue to exact a toll on individuals with AIDS.¹ OIs typically occur in individuals who present late in the course of their HIV disease or adhere poorly to ART with resultant low CD4 cell counts. Otherwise, individuals with medical conditions such as diabetes mellitus and those who use medications that suppress the immune system, including steroids, and transplant recipients, are similarly vulnerable to OIs.

The prevalence of CNS OIs among individuals with HIV infection has decreased more than 10-fold following the availability of potent ART, but the CNS infections that occur are associated with significant morbidity and mortality.² In individuals with advanced HIV disease, the most common CNS OIs include cerebral toxoplasmosis, primary CNS lymphoma, and those associated with *Cryptococcus*, *Cytomegalovirus* (CMV), John Cunningham (JC) virus, and *Mycobacterium tuberculosis* infections. Rarely, infections with *Nocardia*, *Histoplasma* and *Aspergillus* spp. may occur. Although the clinical syndromes associated with these organisms may be similar, differentiating between them is critically important as it affects the timely initiation of appropriate management that determines patient outcomes. This case highlights the presentation and management of *Nocardia* infection, a disease which is uncommon among HIV-infected patients, partly due to the widespread

use of sulfa-containing antibiotics for prophylaxis against OIs that have activity against the pathogen. In this article, we present a case of disseminated nocardiosis involving the CNS, lungs, and skin.

CASE PRESENTATION

A 40-year-old man, recently diagnosed with AIDS, was transferred to our facility from another hospital with a 2-month history of headache, persistent altered mental status, generalised weakness and with bowel and bladder incontinence. Three months preceding the transfer, he had presented to the same hospital with a productive cough and was treated with multiple courses of antibiotics for community-acquired pneumonia. A month later, when he developed the neurological symptoms, an MRI of the brain and spinal cord, performed to evaluate his symptoms, revealed multiple ring-enhancing lesions. No cerebrospinal fluid (CSF) was obtained at the time because of cerebral oedema associated with his brain lesions. An HIV test was performed and returned positive. His CD4 count was 21 cells/mm³ and HIV viral load was 74 368 copies/mL. He was started on empirical treatment for CNS toxoplasmosis with sulfadiazine, pyrimethamine and leucovorin. Intravenous (IV) dexamethasone was initiated for cerebral oedema. Since there was no improvement in his neurological status despite the aforementioned treatments, he was transferred to our facility for further management.

On initial examination, he appeared to be cachectic and was oriented to person only but not to place nor time. He was afebrile, heart rate was 88 bpm, blood pressure was 100/65 mm Hg and oxygen saturation was 96% on room air. Examination of his oropharynx revealed thrush. Cardiac, pulmonary and abdominal examinations were normal. Skin examination was notable for multiple, tender, erythematous 1–2 cm subcutaneous nodules located on his extremities and chest, some with active purulent drainage (figure 1A). Neurological examination revealed ataxia and weakness of his left lower and right upper extremities. His sensory examination was normal.

INVESTIGATIONS

Initial imaging studies at our facility included a brain MRI, which revealed innumerable ring-enhancing lesions (abscesses) scattered throughout the supratentorial and infratentorial compartments bilaterally with varying degrees of mild perilesional oedema but without focal mass effect (figure 2A). A total spine MRI showed fewer ring-enhancing lesions that were similar in appearance to those

CrossMark

To cite: Sherbuk J, Saly D, Barakat L, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2016-215649

Figure 1 (A) Picture showing tender, erythematous subcutaneous nodules located on patient's proximal right lower extremity. (B) CT scan of chest (axial view) revealing bilateral cavitary lung lesions. The cavitary lesion in the right lower lobe has an air-fluid level and extends into the mediastinum. (C) Gram stain of purulent secretion obtained by biopsy of a subcutaneous nodule revealing beaded and branching filamentous Gram-positive bacteria.

that were noted on the brain MRI. There was no cord compression. A chest radiograph revealed a round consolidation along the right heart border as well as a small nodular opacity in the left retrocardiac region. A CT scan of the chest revealed multiple small lung nodules and cavitary lesions in lungs with the largest cavity located in the right lower lobe measuring 3×4×6 cm with an air-fluid level and subcarinal mediastinal extension. There was no pleural effusion and only a few subcentimetre mediastinal lymph nodes were observed (figure 1B). On the basis of these findings, an extensive workup was initiated, focusing on the differential diagnoses of an immunocompromised

individual presenting with altered mental status, focal neurological deficits, multiple ring-enhancing lesions in the brain and spinal cord, skin nodules and cavitary lung lesions.

Given his neurological symptoms and abnormal CNS imaging, a lumbar puncture was performed. CSF analysis revealed protein 109 mg/dL, glucose 48 mg/dL, no red blood cells and 7 nucleated white cell counts per microlitre comprising 94% lymphocytes and 6% monocytes. Utilising a real-time Taqman PCR assay validated for use in our virology laboratory (not approved by the US Food and Drug administration), CSF tested positive for CMV with an estimated viral load of 26 160

Figure 2 (A) MRI brain (axial view) with IV contrast at the time of presentation revealing multiple ring-enhancing lesions. (B) A CT scan of head with contrast (axial view) 2 weeks into the treatment showing marked decrease in visible lesions seen. (C) A CT scan of head with contrast (axial view) 3 months into the treatment showing complete resolution of lesions.

viral copies/mL (4.4 logs). Otherwise, CSF venereal diseases research laboratory (VDRL) and cryptococcal antigen testing returned negative as well as PCR tests for toxoplasmosis, *herpes simplex* virus (HSV), *varicella zoster* virus, *Epstein-Barr* virus (EBV) and *JC* virus. CSF bacterial and acid-fast bacilli cultures showed no growth. A punch biopsy of a 1.5 cm nodule on the patient's right anterior shin was performed with Gram stain of pus obtained from the lesion revealing beaded and branching filamentous rods concerning for *Actinomyces* or *Nocardia* (figure 1C). Serum *Toxoplasma* IgG and PCR, cryptococcal antigen, HSV and EBV viral loads tests were not detected. Serum CMV IgG and IgM tests returned negative. Urine *Histoplasma* antigen, serum anticysticercal antibody and *Aspergillus* galactomannan index tests were also negative. Fungal cultures of blood, pus from skin lesions and lung tissue specimens showed no growth. Ophthalmological examination revealed retinitis in the left eye with classic features of CMV disease. A bronchoscopy was performed and included aspiration of fluid from the largest cavitary lesion and a trans-bronchial biopsy. GeneXpert testing on the bronchoscopy specimen for the detection *M. tuberculosis* was negative. However, a Gram stain of the specimen showed 2 + beaded and branching Gram-positive rods; and subsequent 16S ribosomal RNA sequencing led to the identification of the organism as *Nocardia abscessus*.

DIFFERENTIAL DIAGNOSIS

The differential diagnoses of ring-enhancing CNS lesions in an individual with AIDS are quite broad but typically consist of infectious/inflammatory or neoplastic disorders.³ In the USA, the most common aetiologies are toxoplasmosis, bacterial brain abscesses and primary central nervous system lymphoma. Cerebral toxoplasmosis was unlikely as our patient had already failed prolonged targeted treatment, and CSF and serum studies were negative. We considered it unusual for primary CNS lymphoma to present innumerable ring-enhancing lesions located in the brain and spinal cord. Therefore, we focused on the differential diagnoses of pyogenic CNS lesions that include infections by *Nocardia*, *Listeria* and other pyogenic bacteria, *Aspergillus*, *Histoplasma*, *Cryptococcus* and *M. tuberculosis*. Except for *Nocardia*, all of these were screened for but not identified in our patient. CMV infection typically presents radiologically as a micronodular encephalitis or ventriculoencephalitis rather than with multiple ring-enhancing lesions.³ Thus CMV infection alone could not have accounted for our patient's entire clinical syndrome.

TREATMENT

While antimicrobial sensitivity testing was in progress, empirical treatment for *N. abscessus* was initiated with IV meropenem (2 g every 8 hours) and high-dose oral trimethoprim (20 mg/kg)/sulfamethoxazole (TMP/SMX) administered in divided doses three times daily based on the organism's known susceptibility profile. Oral linezolid (600 mg every 12 hours) was later added as a third agent to optimise therapy given its good CSF penetration. When the patient was stable enough to be discharged to a rehabilitation facility 4 weeks after his admission to our hospital, the antibiotic regimen was transitioned to IV ceftriaxone 2 g daily, administered through a peripherally inserted central catheter, oral doxycycline 100 mg two times per day and TMP/SMX. This was based on the organism's antimicrobial susceptibility profile determined by broth microdilution (Department of Microbiology Research, Mycobacteria/*Nocardia* Laboratory, University of Texas Health Center at Tyler) which revealed susceptibility to TMP/SMX, linezolid, imipenem, amoxicillin–

clavulanic acid, amikacin, ceftriaxone, doxycycline, minocycline, gentamicin, tobramycin and clarithromycin. His strain of *N. abscessus* was resistant to kanamycin, ciprofloxacin and moxifloxacin.

During his hospitalisation, he was concomitantly treated for CMV retinitis with ganciclovir induction therapy (5 mg/kg every 12 hours) for 3 weeks followed by maintenance therapy and secondary prophylaxis with valganciclovir (900 mg daily) which was to be continued until he had a sustained improvement in his CD4 count. Oral azithromycin 1200 mg dosed weekly was instituted for prophylaxis against *Mycobacterium avium-intracellulare*. The patient was initiated on dolutegravir 50 mg, tenofovir 300 mg and emtricitabine 200 mg, all taken once daily orally, for the treatment of his HIV disease 1 week into his admission.

OUTCOME AND FOLLOW-UP

After 2 weeks of antibiotic therapy for *N. abscessus* infection, the patient showed a marked improvement in his mental status and was eventually discharged to a skilled nursing facility for physical rehabilitation. A CT scan of his head at the time showed a marked decrease in the size of the previously described brain abscesses (figure 2B). Three months following his discharge from our facility, the patient returned to work full time and a repeat CT scan of the head showed interval resolution of the brain lesions (figure 2C). His CD4 count had also improved to 42 cells/mm³ and HIV viral load had declined to 36 copies/mL. At 6 months, doxycycline and ceftriaxone were discontinued. He remained on TMP/SMX monotherapy for his disseminated *N. abscessus* infection for a total of 15 months, at which time a brain and total spine MRI confirmed complete resolution of ring-enhancing lesions. Thereafter, his dose of TMP/SMX was decreased to a prophylactic dose of 160/800 mg, taken once daily orally, and he remained on azithromycin and valganciclovir for prophylaxis against *M. avium-intracellulare* and CMV, respectively. His antibiotic prophylaxis would be discontinued when his CD4 counts are stably improved; however, due to potential overlapping toxicities of cytopenias by TMP/SMX and valganciclovir, his CD4 cell count trajectory is being monitored closely.

DISCUSSION

In patients with AIDS, it is not unusual for multiple infections to occur at the same time in the same individual. In one case series, 15% of patients with CNS OIs had multiple infections.² Invasive diagnostic testing is frequently required as it has been reported that in up to 20% of cases, biopsies of brain lesions reveal a different disease process than the leading diagnosis prior to the test.³ As we isolated *Nocardia* from lung tissue and pus from skin lesions of our patient, and since it is well known that the organism has a predilection for the CNS and the radiological appearance of the lesions were compatible with infection by the organism, it was unnecessary to perform a brain biopsy. Moreover, his response to treatment targeting *Nocardia* essentially confirmed our presumed diagnosis. It is pertinent to mention at this point that the lack of response to treatment for community-acquired pneumonia prior to his presentation to our facility should have prompted an aggressive workup for other chronic and/or atypical infections and an earlier accurate diagnosis may have prevented dissemination of his disease from the initial lung foci. It is also likely that the use of steroids for his brain oedema, especially in the absence of appropriate therapy for his infection, may have contributed to his disease progression. We did not continue his steroid treatment at our facility.

It is important to note that our patient's CSF tested positive for CMV and ocular examination revealed CMV retinitis. We elected to perform a lumbar puncture, which was deferred at the referring hospital, as the amount of oedema around his brain lesions was mild with no mass effect (probably affected by steroids he had received prior to his admission at our facility) and the risk of adverse events, including herniation or obstructive hydrocephalus, was deemed to be small and the potential benefit of a diagnostic yield of CSF analysis was thought to be crucial to managing him appropriately. On the basis of the CSF and ocular findings, he was treated for CMV retinitis with ganciclovir. While his altered mental status may in part be attributed to CMV, given the extent of *Nocardia* lesions, as well as the lack of typical radiological CMV CNS disease features, we thought that his clinical presentation is best attributed to disseminated *Nocardia*. It is also noteworthy that CMV coinfection manifesting as reactivation disease (such as confirmed retinitis in our patient) frequently occurs in patients with advanced HIV disease who may have other OIs and has been identified as a poor prognostic marker.⁴

Nocardia organisms are ubiquitous in the environment and may be found in soil, water sources and decaying organic matter.⁵ Inhalation is the primary route of human exposure; therefore, pulmonary disease is the most common presentation. Cutaneous inoculation or haematogenous dissemination may occur resulting in skin and soft tissue infections typically presenting as cellulitis, pustules, frank abscesses or subcutaneous nodules. Haematogenous dissemination may also result in CNS disease.⁶ Our patient was an avid gardener, a hobby which may have led to his exposure to the organism.

Nocardia is thought to cause 1–2% of all brain abscesses.⁷ Most cases occur in immunocompromised individuals, but some studies suggest that in up to 50% of cases, there is no identifiable risk factor.⁵ Disseminated *Nocardia* infection typically first manifests symptomatically in the lungs, after which about half of the infected hosts will develop CNS infection that may progress silently for months to years before clinical symptoms and/or signs develop.^{5–8} While isolated CNS disease has been reported, it is likely that the primary site of infection had resolved at the time of diagnosis.⁵ As in our patient, nocardiosis can also involve the spinal cord.⁶ Unlike other forms of the disease, CNS infection is unlikely to resolve without treatment.⁹

HIV-infected individuals are particularly susceptible to *Nocardia* infections as immune responses to the pathogen are primarily T cell mediated.⁶ This is supported by the observation that disseminated nocardiosis is typically seen in HIV-infected individuals when CD4 counts are below 250 cells/mm³ and, occasionally, may be the initial presentation of HIV infection.^{10–12} However, the overall incidence in patients with AIDS remains low, which may be in part due to the routine use of TMP/SMX for OI prophylaxis which has activity against *Nocardia* species.¹⁰

Nocardia is typically identified in the laboratory by a combination of its morphological appearance on Gram stain, unique biochemical properties and antimicrobial drug susceptibility patterns. Identification of individual species is challenging due to their relatedness as they share similar microbiological and biochemical characteristics. However, in the past two decades, the availability of newer genetic and molecular-based laboratory techniques has made the determination of *Nocardia* species possible and reliable. Current molecular methods for the detection of *Nocardia* species include 16S rRNA gene sequencing, heat shock protein 65 (hsp65) gene sequencing and, more recently, mass spectrometry analysis of bacterial cell-wall proteins.¹³ Our laboratory used 16S rRNA sequencing to determine that

our patient was infected with *N. abscessus*, a species first characterised in 2000.¹⁴ Prior to this time, *N. abscessus* was classified within the '*N. asteroides* species complex'. Recent literature has reported *N. abscessus* to be the pathological species in 2–22% of nocardiosis cases.^{15–19}

Trimethoprim-sulfamethoxazole is considered the drug of choice for the treatment of nocardiosis.¹⁰ Other drugs with broad activity against *Nocardia* species include imipenem, amikacin and linezolid.^{20–21} However, antimicrobial susceptibility patterns differ widely among *Nocardia* species.^{10–22} Multiple small studies have compared the susceptibility profile of *N. abscessus* to other *Nocardia* species.^{16–19–21–23–24} These have shown that *N. abscessus* is usually sensitive to TMP/SMX,^{19–21} though resistant isolates of *N. abscessus* have been reported.²⁵ In multiple published antimicrobial susceptibility surveys, *N. abscessus* was found to be sensitive to ceftriaxone, tobramycin, linezolid and amikacin.^{16–19–21–23–24} Importantly, *N. abscessus* has high MICs for imipenem (8–32 µg/mL), making it one of a few *Nocardia* species resistant to imipenem.²⁶ Our patient's *Nocardia* strain had antimicrobial sensitivity patterns similar to what is described in the published literature but retained sensitivity to imipenem.

Current antibiotic recommendations for the empirical treatment of nocardiosis include TMP/SMX, linezolid or amikacin as first-line agents and then tailoring the treatment based on the species and its susceptibilities.^{10–20–27} Three-drug regimens are generally advised for those with severe disease. In such cases, where compatible with sensitivity profiles, treatment regimens typically include TMP/SMX, amikacin and either a carbapenem or a third-generation cephalosporin.²⁸ Unfortunately, while TMP/SMX remains a useful agent, its utility is limited by sulphonamide intolerance which occurs in up to 55% of patients with AIDS.²⁹

In patients with AIDS, a prolonged treatment course is essential for nocardiosis as relapses are common and re-treatment of relapsed disease is often unsuccessful.³⁰ Shorter courses of therapy (<90 days) are associated with higher rates of relapse.²⁷ There are no clinical trials to support optimal or definitive treatment durations for disseminated nocardiosis and there are no established CD4 count thresholds in HIV-infected patients at which therapy can be discontinued without concerns for disease relapse. However, experts recommend at least 6–12 months of therapy for nocardiosis.^{16–30} Owing to their relatively superior CNS penetration, drugs such as TMP/SMX, linezolid, third-

Learning points

- ▶ *Nocardia* is an uncommon but important cause of pulmonary, cutaneous and central nervous system disease in HIV-infected patients and may result in serious morbidity and mortality.
- ▶ Since it may also occur in conjunction with, or mimic other OIs, the diagnosis may be missed and requires a high index of suspicion, especially in HIV-infected individuals with low CD4 counts who are not on TMP/SMX prophylaxis.
- ▶ Recent laboratory techniques now allow for identification of *Nocardia* organisms and their specific species, which is clinically relevant due to their differing antimicrobial susceptibility patterns.
- ▶ The optimal antibiotic treatment duration for disseminated nocardiosis remains unknown and deserves future investigation to decrease the risk of relapse and mortality associated with the infection.

generation cephalosporins and/or carbapenems should be considered for use in patients with CNS disease. In our patient, species identification and subsequent availability of susceptibilities allowed for the treatment regimen to be transitioned to effective and well-tolerated agents with impressive clinical outcomes after a prolonged treatment course. Unfortunately, this is not observed in every case as, in spite of appropriate treatment, the mortality for nocardial brain abscesses remains ~50%.¹⁰

Contributors JS, DS, LB and OO, all provided direct clinical care to the patient and identified this case as worth sharing. All the authors were responsible for conception of the article, literature search, drafting and revision of the article as well as approval of the submitted draft.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Joint United Nations Programme on HIV/AIDS (UNAIDS). Global report: UNAIDS report on the global AIDS epidemic. 2013. http://www.unaids.org/sites/default/files/media_asset/UNAIDS_Global_Report_2013_en_1.pdf (accessed on 31 Mar 2016).
- 2 Tan IL, Smith BR, von Geldern G, et al. HIV-associated opportunistic infections of the CNS. *Lancet Neurol* 2012;11:605–17.
- 3 Skiest DJ. Focal neurological disease in patients with acquired immunodeficiency syndrome. *Clin Infect Dis* 2002;34:103–15.
- 4 Wohl DA, Zeng D, Stewart P, et al. Cytomegalovirus viremia, mortality, and end-organ disease among patients with AIDS receiving potent antiretroviral therapies. *J Acquir Immune Defic Syndr* 2005;38:538–44.
- 5 Lerner PI. Nocardiosis. *Clin Infect Dis* 1996;22:891–903; quiz 904–5.
- 6 Beaman BL, Beaman L. Nocardia species: host–parasite relationships. *Clin Microbiol Rev* 1994;7:213–64.
- 7 Yorke RF, Rouah E. Nocardiosis with brain abscess due to an unusual species, *Nocardia transvalensis*. *Arch Pathol Lab Med* 2003;127:224–6.
- 8 McNeil MM, Brown JM. The medically important aerobic actinomycetes: epidemiology and microbiology. *Clin Microbiol Rev* 1994;7:357–417.
- 9 Horré R, Schumacher G, Marklein G, et al. Mycetoma due to *Pseudallescheria boydii* and co-isolation of *Nocardia abscessus* in a patient injured in road accident. *Med Mycol* 2002;40:525–7.
- 10 Corti ME, Villafañe-Fioti MF. Nocardiosis: a review. *Int J Infect Dis* 2003;7:243–50.
- 11 Pintado V, Gómez-Mampaso E, Cobo J, et al. Nocardial infection in patients infected with the human immunodeficiency virus. *Clin Microbiol Infect* 2003;9:716–20.
- 12 Márquez-Díaz F, Soto-Ramírez LE, Sifuentes-Osorio J. Nocardiasis in patients with HIV infection. *AIDS Patient Care STDS* 1998;12:825–32.
- 13 Sorrell TC, Mitchell DH, Iredell JR, et al. Nocardia species. In: Bennett JE, Dolin R, Blaser MJ, eds. *Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases*. 8th edn: Saunders, 2015:2853–63.e2852.
- 14 Yassin AF, Rainey FA, Mendrock U, et al. *Nocardia abscessus* sp. nov. *Int J Syst Evol Microbiol* 2000;50:1487–93.
- 15 Portolá O, Guitart R, Gómez F, et al. [Epidemiology and clinical manifestations of infection due to Nocardia species in Tarragona, 1997–2008: *Nocardia cyriacigeorgica* is an emerging pathogen]. *Enferm Infect Microbiol Clin* 2009;27:585–8.
- 16 Maraki S, Scoulica E, Nioti E, et al. Nocardial infection in Crete, Greece: review of fifteen cases from 2003 to 2007. *Scand J Infect Dis* 2009;41: R122–7.
- 17 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* 2011;30:1341–7.
- 18 Muñoz J, Mirelis B, Aragón LM, et al. Clinical and microbiological features of nocardiosis 1997–2003. *J Med Microbiol* 2007;56:545–50.
- 19 Larruskain J, Idigoras P, Marimón JM, et al. Susceptibility of 186 *Nocardia* sp. isolates to 20 antimicrobial agents. *Antimicrob Agents Chemother* 2011;55:2995–8.
- 20 Wang HL, Seo YH, LaSala PR, et al. Nocardiosis in 132 patients with cancer: microbiological and clinical analyses. *Am J Clin Pathol* 2014;142:513–23.
- 21 McTaggart LR, Doucet J, Witkowska M, et al. Antimicrobial susceptibility among clinical Nocardia species identified by multilocus sequence analysis. *Antimicrob Agents Chemother* 2015;59:269–75.
- 22 Tripodi MF, Adinolfi LE, Andreana A, et al. Treatment of pulmonary nocardiosis in heart-transplant patients: importance of susceptibility studies. *Clin Transplant* 2001;15:415–20.
- 23 Glupczynski Y, Berhin C, Janssens M, et al. Determination of antimicrobial susceptibility patterns of *Nocardia* spp. from clinical specimens by Etest. *Clin Microbiol Infect* 2006;12:905–12.
- 24 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.
- 25 Daeschlein G, Fetouh Yassin AA, Franke A, et al. Unusual infections: femoral abscess due to *Nocardia abscessus* in a patient suffering from metastatic peripheral bronchial carcinoma and hygienic consequences. *GMS Krankenhhyg Interdisziplin* 2011;6:Doc03.
- 26 Brown-Elliott BA, Brown JM, Conville PS, et al. Clinical and laboratory features of the *Nocardia* spp. based on current molecular taxonomy. *Clin Microbiol Rev* 2006;19:259–82.
- 27 Lai CC, Tan CK, Lin SH, et al. Comparative in vitro activities of nemonoxacin, doripenem, tigecycline and 16 other antimicrobials against *Nocardia brasiliensis*, *Nocardia asteroides* and unusual *Nocardia* species. *J Antimicrob Chemother* 2009;64:73–8.
- 28 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* 2007;44:1307–14.
- 29 Gordin FM, Simon GL, Wofsy CB, et al. Adverse reactions to trimethoprim-sulfamethoxazole in patients with the acquired immunodeficiency syndrome. *Ann Intern Med* 1984;100:495–9.
- 30 Uttamchandani RB, Daikos GL, Reyes RR, et al. Nocardiosis in 30 patients with advanced human immunodeficiency virus infection: clinical features and outcome. *Clin Infect Dis* 1994;18:348–53.

Copyright 2016 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow