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SHORT COMMUNICATION

# *Nocardia abscessus* brain abscess in an immunocompetent host

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## KEYWORDS

Nocardia;  
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*Nocardia abscessus*

**Summary** *Nocardia* brain abscesses typically occur in immunocompromised patients. Most cases of nocardiosis are caused by the *Nocardia asteroides* complex and *Nocardia brasiliensis*. Here, we present a patient with a *Nocardia abscessus* brain abscess. The diagnosis was confirmed by DNA sequencing, and the organism was susceptible to linezolid, clarithromycin, ceftriaxone, imipenem, tobramycin, amikacin, minocycline and sulfamethoxazole. The patient was successfully treated medically in combination with surgical excision.

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## Introduction

*Nocardia* brain abscess is rare and accounts for 1–2% of all cerebral abscesses [1]. However, brain abscess is the most common secondary infection due to *Nocardia*. Many cases occur in immunocompromised patients, half of the cases have been reported to have no identifiable risk factors [2]. Most cases of nocardiosis are caused by the *N. asteroides* complex and *N. brasiliensis*. The

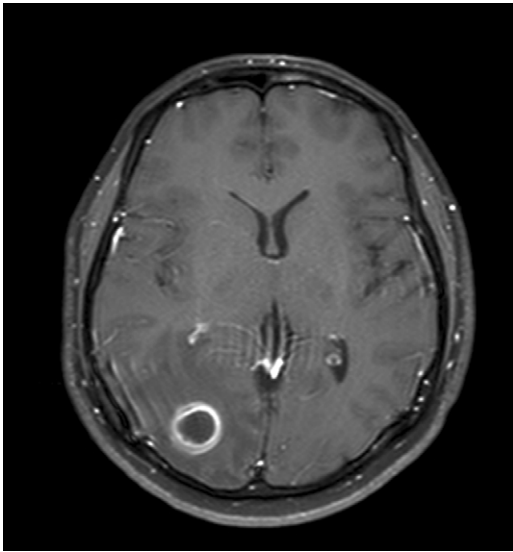
*N. asteroides* complex includes *N. asteroides*, *N. brasiliensis*, *N. otitidiscaviarum*, *N. transvalensis*, *N. farcinica* and *N. nova* [3]. *N. abscessus*, previously known as *N. asteroides* type 1, has been recently reported to cause disease in humans [4]. Thus, *N. abscessus* data may be buried within this broader designation in earlier studies. Here, we present a case of *N. abscessus* brain infection in an immunocompetent patient.

## Case presentation

The patient was a 37-year-old Saudi gentleman who was admitted with a three- to four-week history

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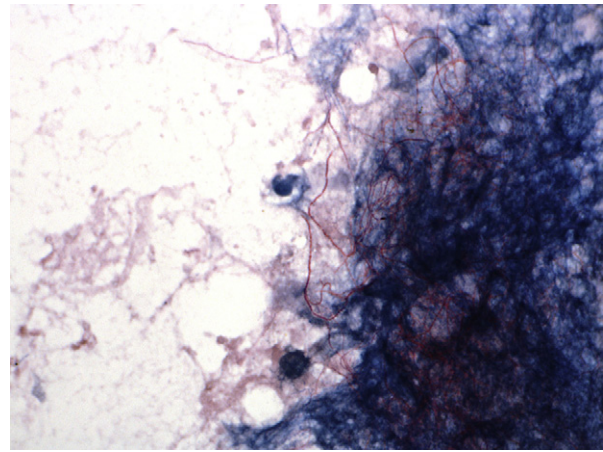


**Figure 1** Magnetic resonance imaging (MRI) of the brain showing an intra-axial, right occipital ring, enhancing lesion surrounded by extensive vasogenic edema. There was a mild adjacent mass effect on the right lateral ventricle with a preserved vascular flow void.

of headache, which gradually increased in severity, associated with some dizziness and blurred vision. The patient did not report any fever or chills, and he did not notice any weakness or numbness in the extremities. The patient was initially observed in an outside hospital where he underwent CT and magnetic resonance imaging (MRI), which showed a right occipito-parietal space-occupying lesion with edema that was more compatible with abscess than glioblastoma multiforme. The patient had no history of travel and no significant past medical history. On examination, the patient was afebrile, alert, and oriented to time, place, and person. His lungs were clear to auscultation, and heart sounds were normal. His abdomen was soft and lax and had no tenderness. He had no cranial nerve or motor deficits.

Laboratory data revealed an ESR of 10 mm/h, C-reactive protein 0.5 mg/dl, BUN 17 mg/dl, creatinine 0.9 mg/dl, white blood cell count 15,200/microliter, and hemoglobin 14.4 g/dl. The patient had a normal neutrophil function test, lymphocyte count and normal immunoglobulin levels. He was negative for human immunodeficiency virus.

MRI revealed an intra-axial, the right occipital, 2.3 cm enhancing lesion surrounded by extensive vasogenic edema, with imaging and spectroscopic features most consistent with a brain abscess (Fig. 1). There was a mild adjacent mass effect on the right lateral ventricle, with a preserved vascular flow void.



**Figure 2** Modified Ziehl-Neelsen smear revealed acid-fast branching filaments resembling *Nocardia* spp.

The patient underwent a right occipital craniotomy for gross total resection of the brain lesion using a neuronavigation system. Intra-operatively, there was a hardened vascular capsule containing purulent material. The lesion was resected, and the aspirated material was sent for Gram staining. The stain revealed moderate WBCs, and no organism was identified. Brain tissue showed central acute and chronic inflammation, with reactive changes consistent with an abscess. The patient was started on intravenous vancomycin (500 mg IV q6 hrs), metronidazole (500 mg q8 hrs) and ceftriaxone (2 g q12 hrs) empirically. On the following day, the AFB smear revealed branching filaments resembling *Nocardia* spp. (Fig. 2). The organism initially grew in AFB culture, and it was then sent for identification to the Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, MA, USA. Antibiotics were adjusted to include trimethoprim-sulfamethoxazole (400 mg trimethoprim IV q8 hrs), ceftriaxone (2 g IV q12 hrs) and linezolid (600 mg IV q12 hrs) for 5 weeks. He had an uneventful hospital stay, and he was discharged to continue oral TMP-SMX for an additional 8 weeks. At the time of discharge, the final identification and susceptibility testing became available. He did well with no recurrence one year after the initial presentation. Repeat MRI at that time showed only post-operative changes and parenchymal encephalomalacia of the right occipital lobe with no recurrence.

The cultured organism was identified by DNA sequencing at the Department of Laboratory Medicine and Pathology, Mayo Clinic, Rochester, MA, USA. The organism was identified as *N. abscessus*. Susceptibility testing revealed that the organism was susceptible to linezolid

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