Case Report

Nocardia thailandica Brain Abscess in an Immunocompromised Patient

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Objectives. Successful treatment for Nocardia thailandica is not well elucidated in the literature. To the best of our knowledge, N. thailandica has not yet been described in the medical literature to cause central nervous system (CNS) infection from brain abscess. We report the case of an immunocompromised patient who underwent successful treatment to treat his brain abscess caused by N. thailandica. Methods. After failing medical therapy, the patient underwent a craniotomy, and tissue was sent for culture. Upon identification by 16S rDNA sequencing, the organism causing infection was identified to be N. thailandica. Results. Based on susceptibilities, the patient was treated with IV ceftriaxone 2 grams daily for five months. The patient demonstrated clinical and radiological improvement which persisted to 7 months after initiation of therapy. Conclusions. To the best of our knowledge, this is the first documented case of a brain abscess due to N. thailandica which was successfully treated. Due to the location of the infection, ceftriaxone was chosen because of optimal CNS penetration. Ceftriaxone monotherapy demonstrated clinical and radiographic treatment success resulting in the successful treatment of this infection.

1. Introduction

Nocardia spp. are responsible for both localized infections, such as pneumonia, and disseminated infections which can occur in the central nervous system (CNS). Immunosuppression is a known risk factor for nocardia infection. Nocardia thailandica is a rare species which has only been documented to cause infections in humans four times in the medical literature since its original classification in 2004. Here, we report the first documented case of a brain abscess due to N. thailandica which was successfully treated with ceftriaxone monotherapy.

2. Case Presentation

A 44-year-old immunocompromised male presented to an outside hospital with new-onset seizures and altered mental status. Relevant past medical history included pure red cell aplasia, concomitant autoimmune hemolytic anemia, and T-cell large granular lymphocytic (LGL) leukemia for which he received chemotherapy with cladribine two years prior to presentation. A CT of the brain revealed a small ring-enhancing lesion (measuring 0.9 (AP) × 0.9 (TR) × 0.9 (CC) cm) in the right parietal lobe with surrounding edema. A lumbar puncture was performed which revealed 11 WBC
granulation tissue, which contains a dense polymorphous inflammatory infiltrate composed of neutrophils, macrophages, lymphocytes, and plasma cells. A capsule with embedded fibroblasts and capillaries separates the abscess from the adjacent reactive brain. Cultures from this procedure revealed branching Gram-positive rods on Gram stain.

Given the concern for *Nocardia*, the patient was initiated on ceftriaxone and sulfamethoxazole/trimethoprim (TMP/SMX), in addition to metronidazole and amphotericin B. Once the patient was clinically stable, the patient was transferred back to the outside institution for further care. At the outside institution, ceftriaxone was discontinued and meropenem was initiated. Over the next two days, the patient developed severe headaches and profound left-sided weakness. A repeat CT of the brain revealed an interval increase in vasogenic edema involving the right frontoparietal, occipital, and temporal regions with the development of 5 mm midline shift. Given worsening of his clinical status and imaging, he was transferred back to our institution. The day following the transfer, the Gram-positive rods on Gram stain were identified to be *Nocardia thailandica* by 16S rDNA sequencing with a 99.8% match to the *N. thailandica* type strain IFM 10145.

Upon definitive identification, ceftriaxone 2 g IV daily was initiated, TMP/SMX was continued on a dose of 15 mg/kg/day of the trimethoprim component, and meropenem was discontinued. Metronidazole and amphotericin were not continued given the findings. Two weeks after the identification of *N. thailandica*, susceptibility testing at the University of Texas revealed susceptibility to ceftriaxone. Given the susceptibility results and the development of significant hyperkalemia, the decision was made to discontinue TMP/SMX after 19 total days of therapy.

Following the optimization of the antimicrobial regimen, the patient improved both clinically and radiographically. He was discharged from the hospital 63 days from the brain biopsy that revealed *N. thailandica*. The patient completed a total of five and a half months of ceftriaxone monotherapy.

MRI of the brain obtained four months after initiation of therapy revealed a stable associated mass effect and surrounding edema. A subsequent MRI obtained 7 months from the initiation of therapy revealed slightly decreased interval size of the surgical cavity and no significant interval change in vasogenic edema/gliosis. The patient did not experience any additional seizures or neurologic deficits after starting ceftriaxone therapy.

### 3. Discussion

*Nocardia* is a genus of the aerobic actinomycetes family, a large and diverse group of Gram-positive bacteria. Accurate and rapid identification of *Nocardia* spp. is critical to optimize empiric antimicrobial therapy. Routine identification of *Nocardia* to the species level is a time-consuming process. Furthermore, these phenotypic tests may be inconclusive and difficult to interpret, resulting in limitations in the identification of *Nocardia* species [1, 2]. Additionally, the genus *Nocardia* has undergone substantial taxonomic
Optimal therapy for *Nocardia* spp. has not been well established [1, 8]. Considerations for selecting therapy should be based on species of *Nocardia* identified, site, and severity of infection. Combination therapy against *Nocardia* spp. has been thought to provide enhanced activity and is recommended for initial treatment for most forms of nocardiosis. Single-drug therapy may be sufficient after species identification, and antimicrobial drug susceptibility information can be confirmed [8]. As no randomized controlled trials provide guidance on optimal treatment, this recommendation is largely based on clinical experience.

Patients with CNS nocardiosis may have increased mortality; therefore, combination therapy is often strongly recommended [9]. Ceftriaxone, meropenem/imipenem, sulfonamides, linezolid, and amikacin are often options for the treatment of nocardiosis. However, based on drug susceptibility testing at the species level, there is a wide range of variation in coverage [10, 11]. Table 1 reveals antibiotic susceptibility data of *Nocardia thailandica* from the literature and our case.

Of the four strains of *N. thailandica* with full susceptibility data reported, all were susceptible to ceftriaxone and carbapenems. Of the most common susceptible agents, ceftriaxone and TMP/SMX have the most optimal blood-brain barrier penetration and may be ideal for the treatment of CNS nocardiosis [12]. Due to poor penetration into the CNS and an unfavorable toxicity profile with prolonged administration, amikacin would be a suboptimal option. Linezolid, despite good CNS penetration, has significant adverse effects associated with prolonged use, such as thrombocytopenia, peripheral neuropathy, and optic neuropathy, the latter two of which are irreversible. Imipenem also penetrates the CNS well; however, it has well-known toxicity of seizures limiting its use. Moxifloxacin reaches high CSF concentrations and is active against selected *Nocardia* spp.; however, it has extremely limited human data [10, 12]. Meropenem, while certainly an option for CNS nocardiosis, may prove to be overly broad and increase the risk of selecting resistant organisms. Additionally, the increased dosing frequency of meropenem would also prove to be a limitation of outpatient treatment as compared to the dosing regimen selected for our patient, once-daily dosing of ceftriaxone. In summary, ceftriaxone and TMP/SMX would be potentially ideal agents for the treatment of CNS nocardiosis given their coverage for *Nocardia thailandica* and good penetration into the CNS.

### Table 1: Susceptibility profile of *Nocardia thailandica* based on the existing published literature.

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AMK: amikacin; AMC: amoxicillin-clavulanic acid; AZI: azithromycin; FEP: cefepime; CRO: ceftriaxone; CIP: ciprofloxacin; CLR: clarithromycin; DOX: doxycycline; GAT: gatifloxacin; IPM: imipenem; LZD: linezolid; MIN: minocycline; MXF: moxifloxacin; TOB: tobramycin; SXT: trimethoprim-sulfamethoxazole. S: susceptible; I: intermediate; R: resistant; *1/2 strains were susceptible and the other resistant.
clinical and radiographic treatment success resulting in the successful treatment of this infection.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References