CASE REPORT

NOCARDIA BEIJINGENSIS BRAIN ABSCESS IN AN HIV INFECTED PATIENT: A FIRST CASE REPORT AND LITERATURE REVIEW

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Abstract. We report here brain abscesses caused by Nocardia beijingensis in a 59-year-old-Thai male with human immunodeficiency virus (HIV) infection presenting with progressive right sided hemiparesis. A computed tomography scan of the brain showed multiple brain abscesses. A stereotactic brain biopsy and 16S rRNA sequencing showed Nocardia beijingensis. The patient was treated with trimethoprim-sulfamethoxazole and recovered completely. As far as we are aware, this is the first reported case of a brain abscess in an HIV infected patient due to Nocardia beijingensis.

Keywords: Nocardia beijingensis, brain abscesses, HIV patient

INTRODUCTION

Nocardia species are pathogenic organisms that cause nocardiosis in humans, especially those with defects in cellular mediated immunity such as human immunodeficiency virus (HIV) infection, organ transplants patients and patients receiving immunosuppressive agents. As far as we know, there are no reports of Nocardia beijingensis causing a brain abscess in an HIV patient. We report here such a case and review the literature.

CASE REPORT

A 59-year-old Thai male presented to Siriraj Hospital, Bangkok, Thailand with generalized headache and progressive weakness of his right arm and leg for 2 months. He denied any other health problems but did complain of a productive cough for 8 months prior to presentation. He denied chest pain, fever or weight loss. He went to a province hospital first who performed a chest X-ray that showed a reticulonodular infiltration in both lungs. A sputum Gram stain was unrevealing and an acid-fast stain was negative. He was diagnosed as having smear negative pulmonary tuberculosis and was treated with antituberculous agents (isoniazid, rifampicin, pyrazinamide and ethambutol) for 2 months without clinical improvement. After that he was lost to follow-up.
Two months prior to presentation to our hospital the patient developed right arm weakness and a generalized headache. He denied having fever, blurred vision, nausea and vomiting but continued to have a productive cough. He was admitted to the province hospital and computed tomography (CT) of the brain was done which showed only vasogenic cerebral edema in the left high parietal white matter. There was no mass or hydrocephalus seen on CT. He was diagnosed as having a metastatic brain tumor of unknown origin and treated with oral prednisolone (60 mg/day) without clinical improvement.

Two weeks prior to admission to our hospital the patient developed progressive right sided hemiparesis. He could not walk without assistance. He continued to have a generalized headache. He also had low grade fever and significant weight loss. Two hours prior to admission he developed generalized tonic clonic seizures which persisted for 3 minutes and was admitted to our hospital.

The patient denied having a history of any underlying diseases. He had worked as a plumber for 30 years. He had a history of multiple sex partner without condom use. He had a 30 pack-year smoking history and drank alcohol daily. He denied any intravenous drug use or any animal contacts. He denied any herbal or over-the-counter drug use. There was no family history of chronic lung disease or malignancies.

Physical examination revealed his temperature of 38°C, he had a pulse rate of 90/min, a blood pressure of 100/60 mmHg, a respiratory rate 22/min and an oxygen saturation of 96% on room air. His BMI was 21 kg/m². Oral examination showed whitish plaque on his tongue and multiple dental caries. He did not have any skin rashes. On lung auscultation there were fine crepitations in both lower lungs. He had no lymphadenopathy or hepatosplenomegaly. On neurological examination, he was fully conscious, he had right hemiparesis with hyperreflexia and upper motor neuron right facial nerve palsy. The rest of the neurological examination was unremarkable.

On laboratory testing he had a hemoglobin of 9.1 g/dl, a white blood cell count of 9,960/mm³ and a platelet count of 199,000/mm³. His creatinine level was 0.83 mg/dl, his aspartate aminotransferase (AST) level was 66 U/l, his alanine aminotransferase (ALT) level was 104 U/l, his albumin level was 2.9 g/dl and his globulin level was 4.8 g/dl.

Chest radiography showed interstitial infiltrates in both lungs. A fourth generation human immunodeficiency virus (HIV) antibody assay was positive. His absolute CD4 count was 65 cells/mm³ (6.24%), hepatitis B surface antigen, anti-hepatitis C virus and venereal disease research laboratory (VDRL) and cryptococcal antigen were all negative. Hemoculture for bacteria, mycobacteria and fungus were also negative.

A CT scan of the brain with contrast (Fig 1) showed multiple rim enhancing lesions in the subcortical bilateral frontal regions, left frontal white matter with perilesional vasogenic edema and mild hydrocephalus. The patient’s serum toxoplasma IgG test was negative. Stereotactic needle biopsy of the brain lesion was performed and seven milliliters of pus was obtained. Direct microbiological examination (Gram stain, acid-fast stain, modified acid-fast stain and Wright stain) and pus culture for bacteria, mycobacteria and fungus were all negative. However, molecular detection using 16S ribosomal
RNA sequencing revealed *Nocardia beijingensis*.

A CT scan of the chest (Fig 2) revealed two rim enhancing extrapulmonary lesions in the posterior right chest with adjacent right sixth rib destruction. There was traction bronchiectasis, centrilobular nodules in both lungs and multiple mediastinal lymph node enlargement. Sputum culture for bacteria, mycobacteria and fungus were all negative. An ultrasound guided needle biopsy of the lung was obtained but there was not adequate specimen for culture. The final diagnoses were acquired immune deficiency syndrome (AIDS) and nocardial brain abscesses with suspected pulmonary nocardiosis.

The patient was treated with intravenously trimethoprim-sulfamethoxazole (15 mg/kg/day) for two weeks then the strength in his right upper and lower extremities improved significantly. There
was also significant improvement in his pulmonary symptoms. A followed-up CT of the brain was done two weeks later showing a marked decreased in the sizes of the brain abscesses (Fig 3). Oral trimethoprim-sulfamethoxazole was continued as outpatient therapy for 12 months. Antiretroviral agents were given one month after initiating antibiotic therapy. A follow-up CT of the brain and the chest 12 months later showed complete resolution of the lesions.

DISCUSSION

A nocardial brain abscess is a presentation of nocardiosis. One-third of patients with a nocardial brain abscess have focal neurological deficits or seizure (Mamelak et al, 1994). Most nocardial brain abscesses are located in the supratentorial region. Nocardial abscesses may be single (54%) or multiple (38%) (Mamelak et al, 1994; Tang et al, 2014). The most common nocardial pathogenic species is Nocardia asteroides complex (Kageyama et al, 2004). Gene sequencing has become an important test for distinguishing nocardial species which have different drug susceptibilities (Kageyama et al, 2004). Several new Nocardia species have been isolated and reported from the clinical specimen including Nocardia beijingensis (Kageyama et al, 2004). However, there are no published cases of Nocardia beijingensis causing a central nervous system infection.

Nocardia beijingensis was first isolated from soil in China in 2001 (Wang et al, 2001). The first infectious case series was reported by Kageyama in 2004 who reported specimens from pus, blood, pericardial fluid and respiratory secretions from Thailand and Japan (Kageyama et al, 2004). The Table summarizes the published reports of infections caused by Nocardia beijingensis. Most of the cases were from Asia and were pulmonary or cutaneous nocardiosis. All the cases were diagnosed by culture and the species were indentified using 16S rRNA sequencing.

Species identification is important because it guides empirical antimicrobial susceptibility. Many Nocardia sp including Nocardia beijingensis have been reported to be susceptible to trimethoprim-sulfamethoxazole, which is standard treatment for nocardiosis (Wang et al, 2001);
## Table

Published reports of *Nocardia beijingensis* infections.

<table>
<thead>
<tr>
<th>Country</th>
<th>Case</th>
<th>Diagnosis</th>
<th>Specimen</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thailand</td>
<td>17 cases</td>
<td></td>
<td>Sputum (5) Pus (3) BAL (2) Lung (2) Bronchial washing (2) TTA (1) Pleural fluid (1) Pericardial fluid (1) Blood (1)</td>
<td>Kageyama <em>et al.</em>, 2004.</td>
</tr>
<tr>
<td>Japan</td>
<td>1 cases</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Japan</td>
<td>4 cases were HIV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>China</td>
<td>Female, 11 years Lung abscess</td>
<td>Bronchoalveolar larvage</td>
<td></td>
<td>Chu <em>et al.</em>, 2008.</td>
</tr>
<tr>
<td>Japan</td>
<td>Female, 60 years Pneumonia</td>
<td>Sputum</td>
<td></td>
<td>Takayanagi <em>et al.</em>, 2008.</td>
</tr>
<tr>
<td>Japan</td>
<td>Male, 48 years Post kidney transplant</td>
<td>Pneumonia</td>
<td>Protective specimen brush</td>
<td>Ogawa <em>et al.</em>, 2011.</td>
</tr>
<tr>
<td>Japan</td>
<td>Male, 79 years No U/D Cutaneous</td>
<td>Pus</td>
<td></td>
<td>Ohmori <em>et al.</em>, 2012.</td>
</tr>
<tr>
<td>France</td>
<td>Male, 47 years Chronic sinusitis Lung abscess</td>
<td>HIV</td>
<td>Bronchoalveolar larvage and transbronchial biopsy</td>
<td>Martinaud <em>et al.</em>, 2011.</td>
</tr>
<tr>
<td>England</td>
<td>Male, 42 years No U/D Cutaneous</td>
<td>Skin biopsy</td>
<td></td>
<td>Derancout <em>et al.</em>, 2012.</td>
</tr>
<tr>
<td>Italy</td>
<td>Male, 47 years Post kidney transplant</td>
<td>Cutaneous</td>
<td>Pus</td>
<td>Arunachalarm <em>et al.</em>, 2014.</td>
</tr>
<tr>
<td>USA</td>
<td>Female, 50 years Post kidney-pancreas transplant</td>
<td>Pneumonia</td>
<td>Transbronchial needle aspiration</td>
<td>Aragaki <em>et al.</em>, 2014.</td>
</tr>
<tr>
<td>USA</td>
<td>Male 48, years No U/D Pneumonia</td>
<td>Sputum and lymph node biopsy</td>
<td></td>
<td>Crozier <em>et al.</em>, 2014.</td>
</tr>
<tr>
<td>Italy</td>
<td>Male 75, years No U/D Spondylodiscitis</td>
<td>Open biopsy</td>
<td></td>
<td>Rigotti <em>et al.</em>, 2015.</td>
</tr>
<tr>
<td>Australia</td>
<td>Male 80, years No U/D Subretinal abscess</td>
<td>Subretinal tissue culture</td>
<td></td>
<td>Richards <em>et al.</em>, 2015.</td>
</tr>
<tr>
<td>Thailand</td>
<td>Male 58, years Post-kidney transplant</td>
<td>Psoas abscess</td>
<td>Biopsy</td>
<td>Palavutitotai <em>et al.</em>, 2015.</td>
</tr>
<tr>
<td>Thailand</td>
<td>Male 59, years No U/D Brain abscesses</td>
<td>Stereotactic brain biopsy</td>
<td></td>
<td>Current reported case.</td>
</tr>
</tbody>
</table>

BAL, Bronchoalveolar larvage; TTA, Transthoracic aspiration; U/D, Underlying disease; HIV, Human immunodeficiency virus; SLE, Systemic lupus erythematosus.

however, some species of *Nocardia*, such as *Nocardia farcinica*, are resistant (Kageyama *et al.*, 2004). *Nocardia beijingensis* has also been reported to be susceptible to imipenem, tobramycin and kanamycin (Kageyama *et al.*, 2004). In-patients with disseminated nocardiosis and immunosuppressed patients require treatment with a combination of antibiotics, such as trimethoprim-sulfamethoxazole com-
combined with imipenem.

We report a case of *Nocardia beijingensis* cerebral abscess diagnosed using 16S rRNA sequencing and treated successfully with trimethoprim-sulfamethoxazole. To the best of our knowledge, this is a first reported case.

**REFERENCES**


