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Case Report of Nocardial Brain Abscess in a Patient on Steroids

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Abstract Nocardial brain abscesses are a rare cause of cerebral abscesses that usually arise in immunocompromised patients and are associated with high morbidity and mortality. We present the case of a 57-year old male who presented with fever, progressive vision loss and disorientation in the emergency room while on treatment for community acquired pneumonia. He has been using steroids for 20 years and has uncontrolled diabetes mellitus, and secondary adrenal insufficiency as a result. Chest computed tomography (CT) showed consolidation in left posterior-inferior lobe. Blood and cerebrospinal fluid (CSF) cultures were negative. Magnetic resonance imaging (MRI) of the head revealed multiple small dispersed lesions with peripheral ring enhancement after contrast administration, concerning for abscesses. Pus was sampled from a superficial occipital abscess and content grew Nocardia sensitive to trimethoprim/sulfamethoxazole (TMP/SMX) and imipenem so the patient was switched to targeted therapy. The following days he developed new motor deficits and became unresponsive to verbal stimulus. Another MRI revealed enlargement of the existing lesions and new lesions with signal restriction on diffusion (DWI) in the levels of the midbrain and pons. The patient developed respiratory insufficiency and passed away regarding of ongoing supportive treatment.

Keywords: *immunocompromised, brain abscess, targeted therapy*

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1. Introduction

Nocardia species are gram-positive, aerobic, branching filamentous bacteria belonging to *Actinomycetales*. They spread to the brain hematogenously or by inhalation and account for ~2% of cerebral abscesses. [1,2] Nocardiosis usually arises in immunocompromised patients (deficient cell-mediated immunity, including organ transplants, leukemia, diabetes mellitus, alcoholism, underlying malignancy, human immunodeficiency virus, long-term use of steroid and autoimmune disease) [3] but may appear in otherwise healthy individuals and is associated with high morbidity and mortality.

2. Case Description

We report the case of a 57-year old male who was discharged 3 weeks ago from another hospital, where he was treated for community acquired pneumonia with ceftriaxone and vancomycin for two weeks. The patient was doing fine, until the past 5 days when he reports progressive vision loss and fluctuations in conscience with episodes of drowsiness and sleepiness during the last week.

He has been using steroids (methylprednisolone 16 mg/day) for 20 years which were prescribed for tonsillitis but he never quit ever since. He also has diabetes mellitus type 2 and has been on treatment with insulins for ten years.

At current admission the patient was febrile with a body temperature of 38°C. He manifested temporo-spatial disorientation and visual field restriction. No other focal neurological signs were found on examination. Uncontrolled blood sugars, and secondary adrenal insufficiency due to steroid use were found so Insulin doses were adjusted accordingly, methylprednisolone was stopped and hydrocortisone (20 mg, twice per day) was started.

Leukocytosis was present on complete blood count and inflammatory markers (ESR, CRP) were elevated. Blood and cerebrospinal fluid cultures didn't grow any organisms and serum immunoglobulins for Toxoplasma were negative. He tested negative for both HIV and tuberculosis (Mantoux test and gamma-INF).

Chest CT (Figure 1a), three weeks after empiric treatment for community acquired pneumonia, showed consolidation in left posterior-inferior lobe (previous CT

showed larger areas of consolidation and pulmonary bullae). MRI of the head (Figure 1b, Figure 1c and Figure 1d) revealed multiple small dispersed lesions with peripheral ring enhancement after contrast administration, suggestive of abscesses. Pus was sampled from a superficial occipital abscess as illustrated on Figure 1c.

The content of the occipital abscess was viscous, whitish and odorless. On pathology foci of acute inflammatory infiltrate containing polymorphs-brain abscesses and lymphocytic infiltrate in the meninges were seen, without any evidence of granulomas. Based on microbiology which concluded *Nocardia*, sensitive to trimethoprim-sulfamethoxazole (TMP/SMX) and imipenem, targeted therapy was started.

In the following days the patient became less responsive to verbal stimuli and developed new motor deficits. MRI on the 5th day post-operatively revealed enlargement of the existing lesions and new lesions in the brainstem, particularly in the midbrain and pons, which showed signal restriction in DWI (Figure 1e). The patient developed respiratory insufficiency and passed away two days later despite of ongoing supportive treatment.

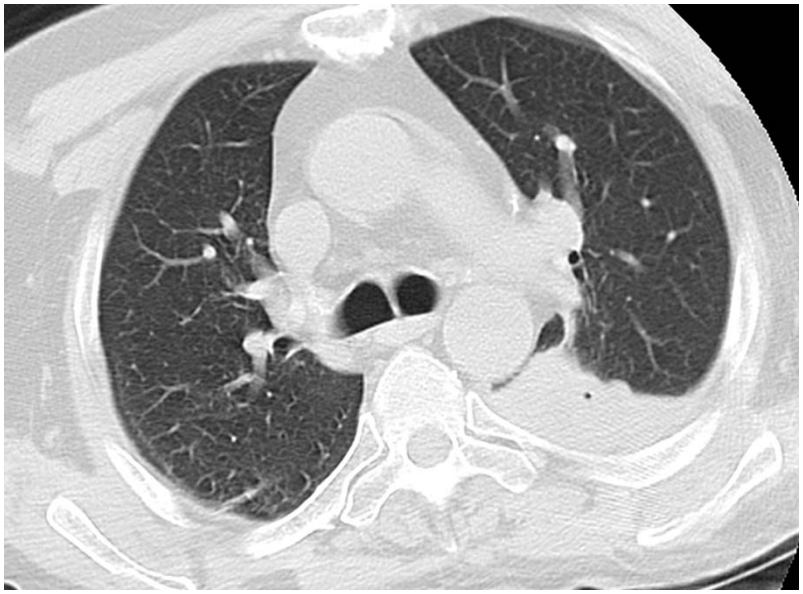

Figure 1a. Chest CT showing pulmonary consolidation in the left posterior-inferior lobe

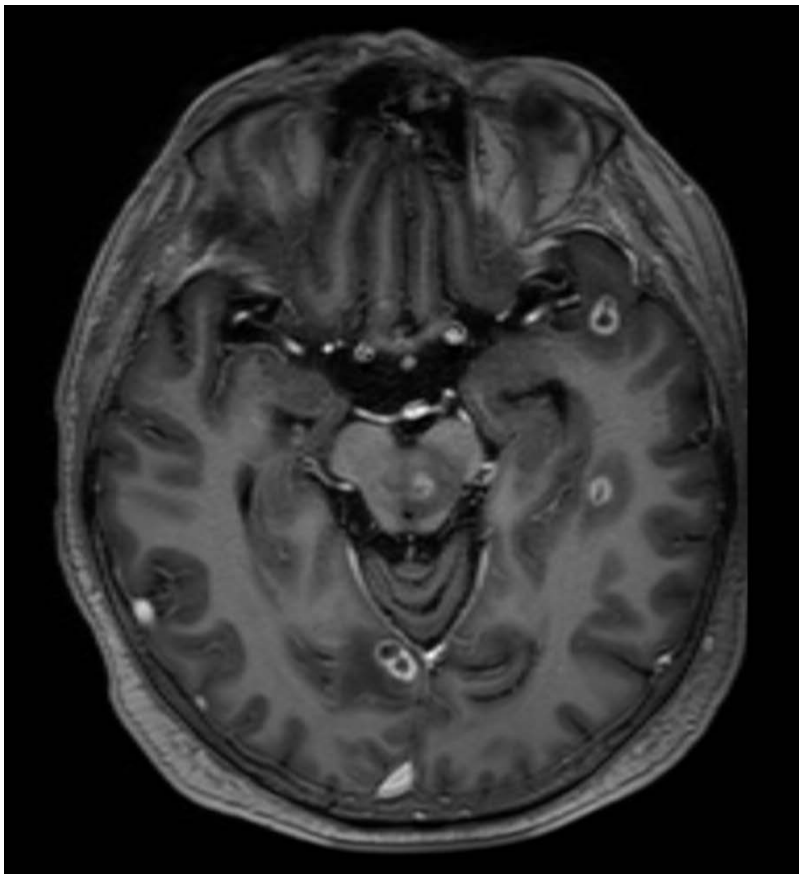

Figure 1b. Multiple dispersed ring enhancing lesions - T1 weighted contrast enhanced image

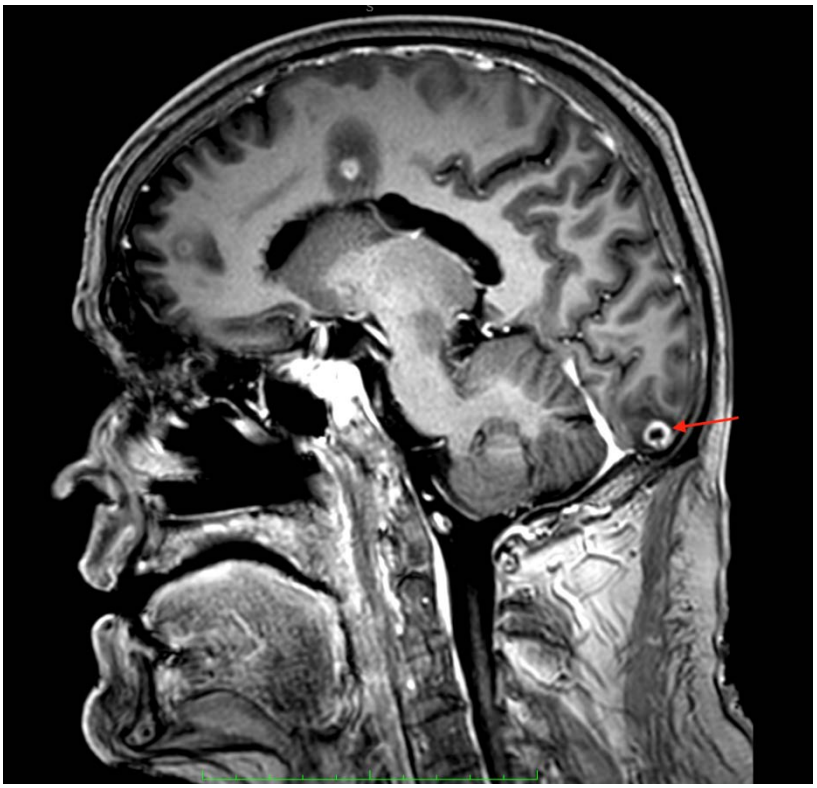

Figure 1c. Pus was sampled and sent for culture from the occipital abscess, marked with a red arrow – Sagittal view of T1 weighted contrast enhanced image

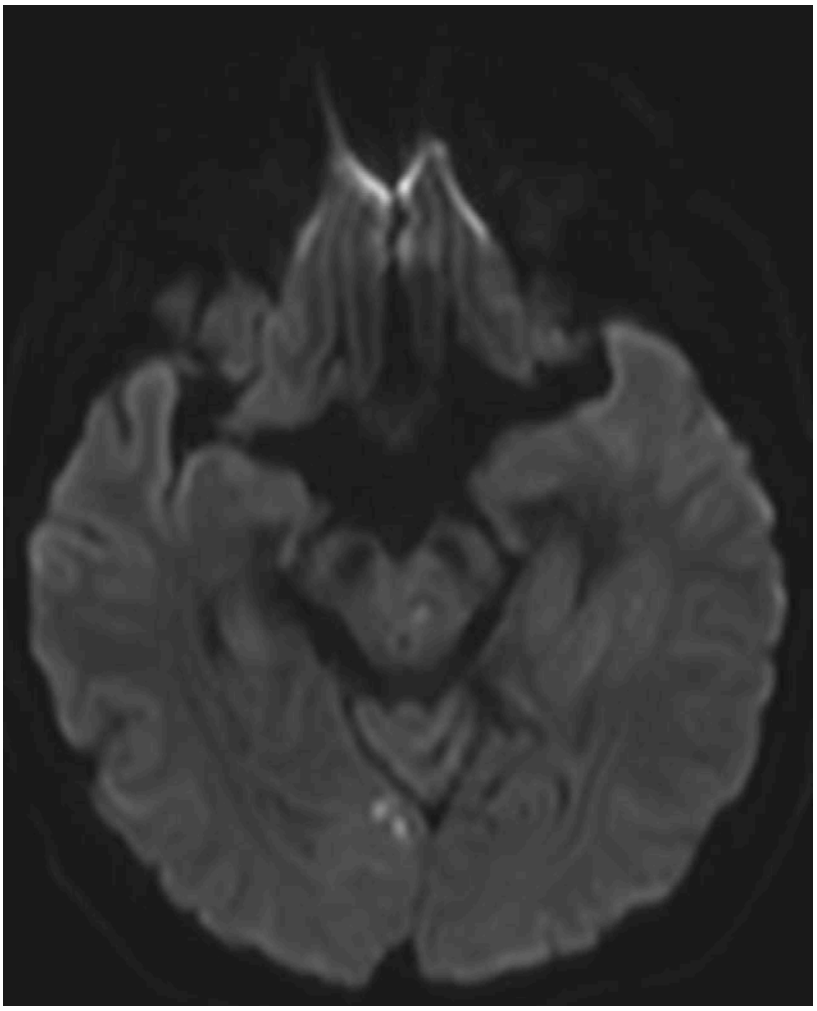

Figure 1d. Small occipital and brainstem lesions with signal restriction on presentation - Diffusion Weighted Image (DWI)

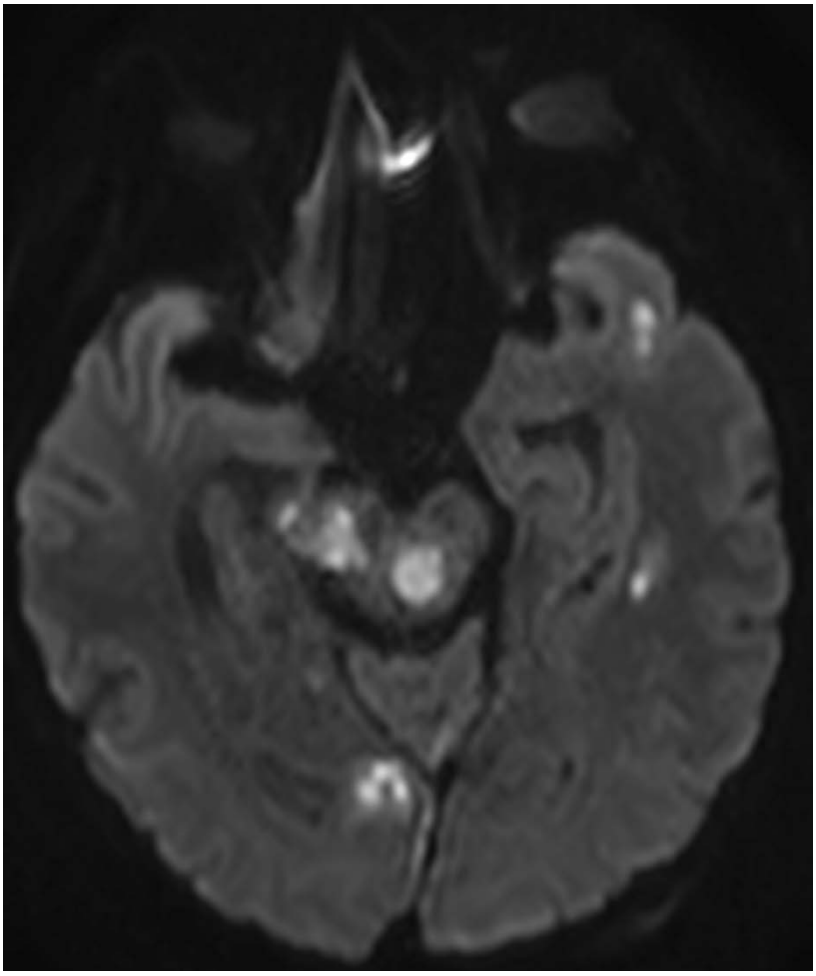

Figure 1e. Follow up MRI revealing enlarged existing lesions and new lesions with signal restriction - Diffusion Weighted Image (DWI)

3. Discussion

Nocardia brain abscess has a mortality rate as high as 20% in immunocompetent patients and 55% in immunocompromised patients, even when they are started on targeted antibiotic therapy. These high rates are attributed to the severity of underlying disease, difficulties in identifying the pathogen, and its inherent resistance to antibiotics, leading to inappropriate or late initiation of therapy. [4,5]

The same thing happened in this patient, who was initially treated for community acquired pneumonia with Ceftriaxone and Vancomycin and while the pneumonia didn't resolve he developed neurological symptoms and presented to our hospital. We recommend being more cautious in immunocompromised patients before making a diagnosis of community acquired pneumonia and always trying to culture before starting empiric antibiotic treatment.

On the other hand, as mentioned above, identifying *Nocardia* can be difficult because this germ grows in a slow fashion and positive rates are relatively low, with colonies requiring at least 48h of incubation, but generally taking even longer. This only prolongs the time from starting targeted antibiotic treatment. In order to isolate *Nocardia*, multiple cerebrospinal fluid specimens may need to be cultured, and the bacteria may be recovered only when direct pus is cultured, as in the present case, when the decision to culture pus from the superficial occipital lesion was made only after one sole blood and cerebrospinal fluid culture came back negative. The decision to undergo surgical biopsy and culture of the pus was made relatively fast in our case because the patient was deteriorating and we felt the need to isolate the microorganism in order to shift from empiric to targeted antibiotic therapy as quickly as possible.

This patient came to our attention only after he presented with neurological symptoms, three weeks after his initial presentation in another hospital with pulmonary findings, so it took about five weeks before the initiation of targeted treatment with TMP/SMX and imipenem. This patient's immunocompromised state added to the relatively late initiation of targeted treatment may have contributed to his prognosis.

4. Conclusions

For pneumonia of unknown cause, especially in immunocompromised patients, vigorous efforts should be made to determine the pathogen as soon as possible, rather than solely starting empiric antibiotic therapy for community acquired pneumonia. High clinical suspicion for Nocardia is needed in patients who also fail treatment for community acquired pneumonia after having been misdiagnosed.

Due to the challenging diagnosis and the culture results, as well as the late presentation in our neurology department, the targeted antibiotic therapy was started at least 5 weeks from his initial presentation with pneumonia,

contributing to his poor prognosis. Early specific antibiotic treatment remains the most important factor to a good prognosis.

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