RARE PRESENTATION OF DISSEMINATED NOCARDIA AS SIADH IN RENAL TRANSPLANT
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INTRODUCTION

Nocardia (N.) species are aerobic, partially acid-fast, branching gram positive bacilli. Nocardiosis is an opportunistic infection with estimated incidence < 5% among renal transplant recipients. Nocardiosis infections typically start in the lungs, can disseminate to CNS and as such have mortality up to 80%. Calcineurin inhibitors (ex. Tacrolimus) and corticosteroids, increase the risk of nocardiosis. Most commonly isolated Nocardia species in renal transplant patients with nocardiosis are N. Farcinica and Asteroides with Farcinica typically implicated in disseminated CNS infection. Here we present a case of cerebral abscess in a renal transplant patient with N. Araoensis and N. Bejingensis. Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) can be the initial sign of CNS disease progression.

CASE PRESENTATION

HPI
A 31-year-old male with ESRD due to HTN and NSAID use, s/p transplantation with several days duration of diffuse headaches associated with vomiting and left upper extremity jerking.

Medications:
Myкопенолат 540 mg BID
Tacrolimus 2 mg BID
Prednisone 10 mg QD

Physical Exam:
Vitals: T 98.3º F, BP 172/96 mmHg, HR 60 bpm, SpO2 = 100%
Lungs: rhonchi at the right lung base
General: not in acute distress
Neuro: alert, CN II – XII intact, motor strength grossly intact
Labs:

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<th>1st image (left) – resolution of hydropneumothorax and pleural effusion</th>
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<td>2nd image (right) – Intracranial abscess on T1 weighted post contrast</td>
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Hospital Course:
The patient underwent a stereotactic left frontal craniotomy with resection of the abscess. Biopsy showed N. Araoensis and N. Bejingensis. The patient did not have orthostatic hypotension.

Outcome:
One month later – patient clinically improved, no further seizures, resumed Tacrolimus 1 mg am and 2 mg pm Repeat CT head – improvement in vasogenic edema; Repeat CT chest – resolution of hydropneumothorax and effusion. Renal transplant recipients are at higher risk of developing N. arnaoensis infections. The patient was transitioned to Bactrim DS 2 tablets, 3 times per day. This regimen was continued for 4 weeks. Mycophenolate and Tacrolimus were held, and only Prednisone 10 mg daily was continued.

RESULTS

Hospital Course:
The patient underwent a stereotactic left frontal craniotomy with resection of the abscess. Biopsy showed N. Araoensis and N. Bejingensis. (Figure 3). The patient had a right pigtal catheter placement, and the same organisms were isolated from pleural fluid.

SIADH was diagnosed based on history, blood work and urine studies.

The patient was treated with Imipenem 500 mg IV every 6 hours and Bactrim DS 2 tablets, 3 times per day. This regimen was continued for 4 weeks. Mycophenolate and Tacrolimus were held, and only Prednisone 10 mg daily was continued.

SIADH was diagnosed based on history, blood work and urine studies. The patient did not have orthostatic hypotension.

Outcome:
One month later – patient clinically improved, no further seizures, resumed Tacrolimus 1 mg am and 2 mg pm Repeat CT head – improvement in vasogenic edema; Repeat CT chest – resolution of hydropneumothorax and effusion (figure 4) and resolution of SIADH. TMP-SMX and Imipenem were stopped due to hyperkalemia and myelosuppression.

Antibiotics were changed to Linezolid 600mg BID and Ceftriaxone 2gm Q 12 x 2 weeks. The patient was transitioned to Bactrim DS once K+ and CBC stabilized.

DISCUSSION

Cerebral nocardiosis is a life-threatening opportunistic infection. It is often misdiagnosed because of its rarity and nonspecific clinical signs. The organism requires multiple days to grow in culture resulting in delayed detection and treatment. A high index of clinical suspicion is the key to early diagnosis. Nocardia should be included in the differential diagnosis when evaluating any patient with risk factors for immunosuppression and a possible opportunistic infection.

Early identification of hyponatremia due to SIADH should alert clinicians to the possibility of Nocardial brain dissemination. Hypoosmolarity due to cerebral salt wasting can be differentiated from SIADH by the presence of hypovolemia and positive orthostatic vital.

Treatment of cerebral nocardiosis involves total surgical excision of the brain abscess, tapering down immunosuppressive regimen and early initiation of antibiotics. TMP-SMX in combination with Imipenem remain the first-line antimicrobials in the treatment of Nocardia infection. Bone marrow toxicity and electrolyte abnormalities may necessitate a temporary change in antimicrobial regimen depending on susceptibility pattern. Prolonged therapy up to 12 months may be needed to prevent relapse.

REFERENCES


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