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Case Report

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Multiple Nocardial Brain Abscesses in a Renal Transplant Recipient: A Case Report

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ABSTRACT

Nocardia is a rare but life-threatening opportunistic infection, especially in immunocompromised patients, including renal transplant recipients. It can cause intracranial abscess which has a mortality of more than 30%. So, when a renal transplant recipient presents with central nervous system involvement, nocardial brain abscess must be considered in the differential diagnosis. Here, we report a such case with multiple nocardial brain abscesses. This case highlights the importance of early diagnosis and treatment to get a good clinical outcome.

Keywords
Brain abscess, nocardia, renal transplant

INTRODUCTION

One of the most dreaded complications of organ transplantation is infection.[1] In the setting of immunosuppression, the rate of infection is drastically increased. Nocardiosis is a rare opportunistic infection that is associated with severe complications in kidney transplant recipients receiving immunosuppressive therapy.[2] Here, we are reporting a case of a multiple Nocardial brain abscesses in a renal transplant recipient.

CASE PRESENTATION

A 60-year-old male, a renal transplant recipient was admitted to our hospital with a history of abnormal bilateral facial twitching and inability to speak for 15 days and gradual weakness of right upper limb with facial deviation to left side for 2 days. He had undergone live non-related renal transplant (LNRRTx) for end-stage renal disease secondary to hypertension 12 months back. His brother was the donor with HLA mismatch of 0/6. The patient had not received any induction immunosuppression. He was under maintenance immunosuppression of tacrolimus, mycophenolate mofetil, and corticosteroid. He had also

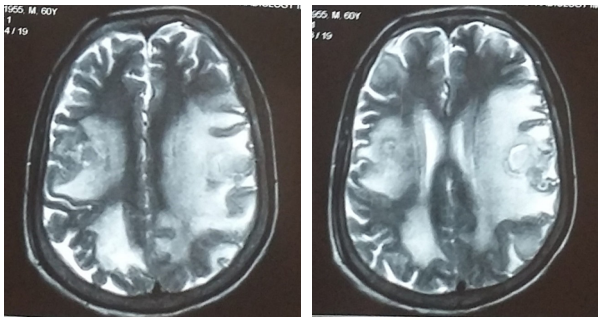

Figure 1. T2 weighted MR images of the brain at the level of lateral ventricles show multiple high signal intensity lesions with isointense thin wall in both frontal and left parietal lobes. Marked hyperintensity in surrounding brain parenchyma, predominantly in white matter represents perilesional edema.

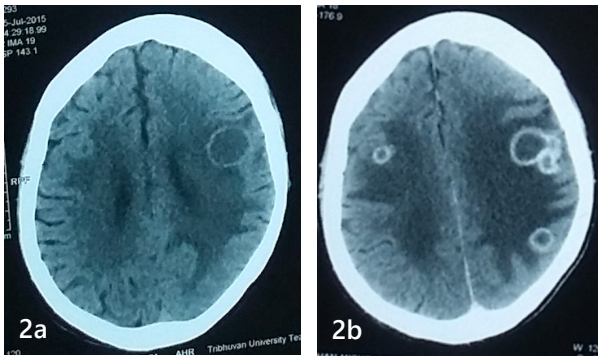

Figure 2a. Non-contrast computed tomography (CT) scans of head at the level of body of lateral ventricles and supraventricular level showing hypodense round lesion with a thin isodense peripheral wall in left frontal lobe. Figure 2b. Contrast CT image showing peripheral wall enhancement of left frontal lobe lesion. Multiple other ring enhancing lesions are also seen in bilateral frontal lobes and left parietal lobe. Marked surrounding hypodensity is seen suggestive of perilesional edema.

received trimethoprim/sulfamethoxazole for 1 year and valganciclovir for 3 months as prophylaxis.

He gave history of intermittent abnormal facial twitchings for 15 days associated with inability to speak. He also complained of gradual weakness the right upper limb, distal more than the proximal associated with facial deviation to the left side. It was accompanied by drooling of saliva from the same side along with history of regurgitation. However, he did not give history of fever, loss of consciousness, abnormal movement of the whole body. He was under anti-tubercular treatment with isoniazid, rifampicin and pyrazinamide for last 5 months for sputum positive pulmonary tuberculosis.

On examination, the patient was disoriented to time, place and person. His general physical examination showed a right solitary posterior cervical lymph node and his Glasgow Coma Scale (GCS) was 10/15. His neurological examination showed right sided upper motor neuron facial nerve palsy with absent gag reflex. Tone was decreased on the right side and his Babinski's reflex was positive on the same side.

His laboratory investigations showed hemoglobin of 10 gm%, total leucocyte count of 7850 cells/mm^3, blood urea of 11.1 mmol/L and creatinine of 101 micromol/L. Both magnetic resonance imaging (MRI) and computed tomography (CT) scans of head showed multiple ill-marginated lesions in both hemispheres with marked perilesional edema suggestive of a granulomatous disorder (Fig. 1,2A,2B). A contrast enhanced CT scan of the chest and abdomen was done which showed multiple nodular opacities of varying sizes in bilateral lung fields with mediastinal and retroperitoneal lymphadenopathy and hyperdense areas with enhancing wall in the subcutaneous and muscular planes in the abdomen and pelvis. Since the patient was already under anti-tubercular treatment and not responding, we sent sputum for acid-fast bacilli (AFB) culture and sensitivity.

Further, bronchoscopy with bronchoalveolar lavage was done which showed modified long slender branching AFB suggestive of Nocardia. His sputum acid fast staining also showed long slender AFB suggestive of Nocardia.

We started the patient on Trimethoprim/Sulfamethoxazole. After a week, the patient's general condition improved, his GCS became 15/15 and the facial weakness improved. On the day of discharge, the weakness had improved to a point that he could walk on his own.

DISCUSSION

Nocardiosis is a rare disorder caused by the bacteria called Nocardia, which tends to primarily affect the lungs, brain and the skin. It occurs primarily in individuals with a weakened immune system.[3] It may also involve kidneys, joints, heart, eyes, and bones. Nocardia is found in soil. It can be acquired from the environment through inhalation or via the contamination of a wound with soil that contains Nocardia. About 90% of such infections are caused by Nocardia (N) asteroides and the remaining 10% are caused by N. brasiliensis and N. caviae.[4,5]

The patients who receive steroids and/or chemotherapy are highly susceptible to this infection. There is increased incidence of nocardiosis in patients suffering from bronchial asthma, malignancy or chronic pulmonary granulomatous conditions.[6] In organ transplant recipients, the non-specific and subtle clinical presentation delays a definite diagnosis and institution of prompt treatment, thereby often resulting in an increased morbidity and mortality.[7]

The clinical manifestations of nocardiosis are extremely variable.[8] It usually presents as a respiratory illness with fever, malaise and unproductive cough. Lung lesions may present as diffuse fibrino-purulent pneumonia, resembling bacterial processes, or as single/multiple abscess cavities.[9] Patients can develop skin abscesses and joint infections. In about 20% of patients, infection may spread to the brain and manifest as well-defined localized abscesses.[10] Rarely, the cerebral lesions may be in the form of meningoencephalitis resulting in a more chronic clinical picture with headache, confusion and lethargy without well-defined localizing signs. Overall, the mortality rate for Nocardial abscesses (31%) is high as compared to that for other bacterial brain abscesses (10%).[11]

Our patient had the involvement of the central nervous system. The Nocardia species have a special tropism for the neural tissue and the most common site for their dissemination is the brain. The dissemination occurs by a hematogenous spread. The prognosis depends on the rapidity with which the diagnosis is established. The treatment of choice is Cotrimoxazole. Carbapenems are other efficacious agents. Synergy may exist between imipenem and cotrimoxazole, and imipenem and cefotaxime, thus enhancing the efficacy of the treatment.[12]

CONCLUSION

Primary nocardial cerebral abscess is rare in renal transplant recipients. But when central nervous system findings are observed in such patients, nocardial brain abscess must be considered in the differential diagnosis. With appropriate and urgent imaging modalities and treatment, good clinical outcome can be assured.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

CONFLICT OF INTEREST

None declared.

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