CASE REPORT

Disseminated Nocardiosis in a Patient with Steroid Dependent Nephrotic Syndrome

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Abstract

Aim: We aim to describe an unusual case of disseminated nocardiosis in a patient with steroid-dependent nephrotic syndrome and its successful management with medical therapy alone.

Background: Nocardia infection is uncommon in clinical practice, with most cases occurring as the result of opportunistic infection in immunocompromised patients. Here, we report a case of disseminated nocardiosis with brain abscesses in a patient with nephrotic syndrome.

Case description: We report a middle-aged female with steroid-dependent nephrotic syndrome with disseminated nocardiosis. The patient was managed with imipenem/cilastatin, oral trimethoprim/sulphamethoxazole (TMP/SMX), and amikacin for 4 weeks followed by dual therapy with co-amoxiclav and TMP/SMX for 6 months. The patient had both clinical and radiological recovery.

Clinical significance: The present case indicates the risk of life-threatening infection in patients receiving steroids and the need for prophylactic therapy to prevent serious infections during the course of steroid therapy.

Keywords: Brain abcess, Nephrotic syndrome, Nocardiosis.

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Background

Disseminated nocardiosis is a rare but serious infection seen mostly in immunocompromised individuals. Here we describe an interesting case of nephrotic syndrome who developed disseminated nocardiosis involving the lungs and central nervous system (CNS). The patient was successfully managed with triple-drug therapy. This case illustrates the importance of suspecting these unusual infections and their effective management.

Case Description

A 54-year-old female presented with fever, night sweats, productive cough, and shortness of breath for 3 days. Two years back, she was diagnosed as a case of steroid-dependent nephrotic syndrome (minimal change disease) and had frequent relapses managed with oral steroids and calcineurin inhibitors. Her current medications included prednisolone 60 mg daily and diuretics for the last 2 months. Laboratory evaluation revealed leucocytosis (15.6 x10^9/L with 90% polymorphs), raised procalcitonin 8.7 ng/mL, and C reactive protein 279 mg/L. Serum creatinine was 0.6 mg/dL, serum albumin 3.5 gmm/dL, and 24-hour urine protein of 1.6 gmm/day suggestive of partial remission. Computed tomography of the chest showed bilateral diffuse patchy areas of consolidation with interlobular septal thickening and right side pleural effusion. Modified Ziehl–Neelsen (ZN) stain of endotracheal aspirate and bronchoalveolar lavage revealed acid-fast branching filamentous bacilli suggestive of *Nocardia* and culture on blood agar incubated at 37°C grew dry cream-colored colonies identified as *Nocardia otitidiscaviarum* by matrix-assisted laser desorption/ionization-time of flight (Figs 1A and B). Fungal culture and GeneXpert were negative. She was intubated and required ventilatory support for 5 days. The patient was started on intravenous imipenem/cilastatin, oral TMP/SMX, and amikacin. Steroids were rapidly tapered by 10 mg/week and stopped. Five days later, the patient developed two episodes of seizures, and a brain MRI (Fig. 2) showed multiple ring-enhancing lesions in the bilateral parietal, occipital lobes, and the lateral ventricle. Cerebrospinal fluid (CSF) analysis revealed a total cell count of 190 with 90% polymorphs, protein 128 mg/dL, and normal sugar. CSF bacterial, fungal cultures, and acid fast bacilli (AFB) staining did not demonstrate any organism. Gradually the patient improved and was extubated and discharged after the completion of 4 weeks of intravenous antibiotics. We plan to give dual therapy with co-amoxiclav and TMP/SMX for 6 months followed by 1-year of TMP/SMX prophylaxis.

Discussion

Disseminated nocardiosis is a rare, life-threatening infection usually seen in immunocompromised patients.[1] It is defined as two noncontiguous sites of involvement that may or may not involve lungs.[2] The lungs are the primary site of infection in two-thirds of cases and it has a special tropism for neural tissue. An earlier study from our center reported a wide spectrum of *Nocardia* species among Indian isolates causing human infection.[3] In an appropriate clinical setting, a presumptive diagnosis of nocardiosis can be made if partially acid-fast filamentous branching rods are

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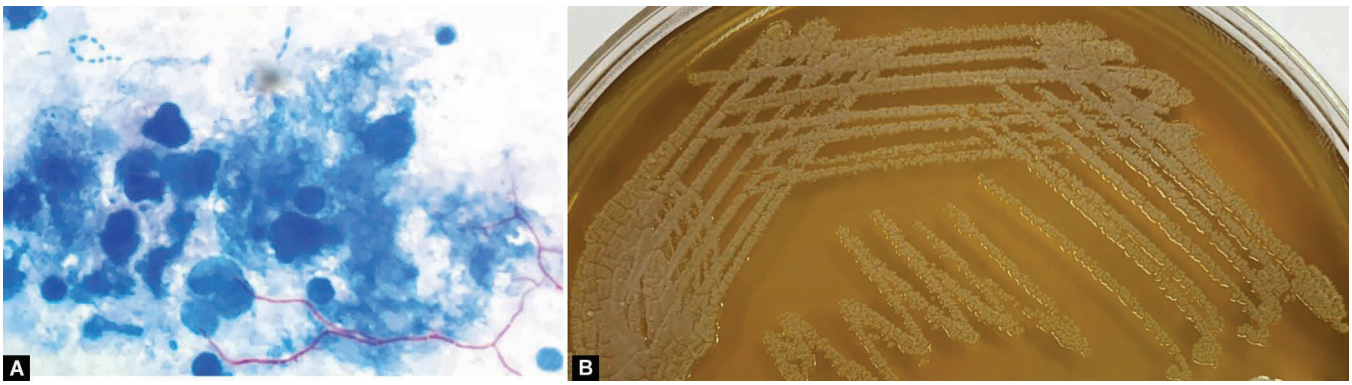

Figs 1A and B: (A) Modified Ziehl–Neelsen (ZN) stain of endotracheal aspirate showing acid-fast branching filamentous acid-fast bacilli suggestive of *Nocardia*; (B) Culture on blood agar showing dry cream coloured colonies identified as *Nocardia otitidiscaviarum* by matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF)

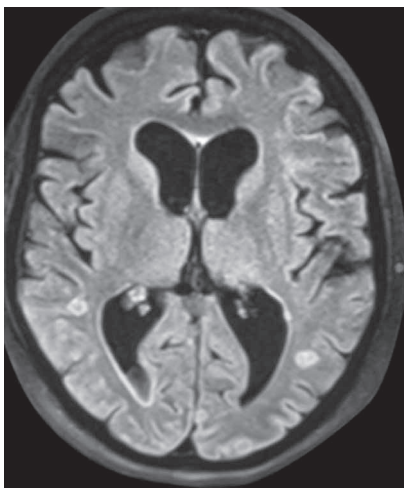

Fig. 2: Magnetic resonance imaging of brain (fluid-attenuated inversion recovery) showing multiple ring-enhancing lesions in both cerebral, cerebellar hemispheres, and lateral ventricle likely abscesses

seen in clinical specimens stained with modified ZN stain. All immunocompromised patients with pulmonary nocardiosis, even those without symptoms of CNS disease warrant brain imaging. The Hallmark of CNS nocardiosis is the formation of parenchymal abscesses that can occur in any region of the brain with CSF findings characteristic of bacterial meningitis.[4] Treatment in disseminated life-threatening CNS infection is usually a combination of oral TMP/SMX, intravenous amikacin, and imipenem/cilastin.[5] Patients who respond to initial therapy are switched to dual oral therapy after 4–6 weeks consisting of TMP/SMX and amoxicillin/clavulanate. A total of 6–12 months of therapy is advised in patients with CNS spread.[6] In immunocompromised patients, sometimes lifelong suppressive therapy is advised. We plan to give dual oral therapy for 6 months and repeat brain imaging after 3 months.

Trimethoprim/sulphamethoxazole prophylaxis is effective against opportunistic pathogens such as *Nocardia*, however, it has been shown that 10.4% of patients receiving TMP/SMX prophylaxis were infected by *Nocardia*.[7] Therefore, early detection of any sign of infection in patients undergoing systemic steroid treatment and aggressive treatment is the key to successful recovery.

Clinical Significance

- Clinicians should be aware of the possibility of disseminated *Nocardia* infection in immunosuppressed patients.
- The early detection and prolonged therapy with properly selected antibiotics may improve the prognosis of this life-threatening infection.

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