CASE REPORT

Nocardia causing chronic suppurative otitis media and cortical venous thrombosis

Kiran Chawla, Anusha Gopinathan, Chandrashekar Udyawara Kudru[1], Shivashankara Kaniyoor Nagiri[1]

Departments of Microbiology and [1]Internal Medicine, Kasturba Medical College, Manipal, Karnataka, India

ABSTRACT

Nocardia is known to cause myriad of infections like pulmonary, cutaneous or disseminated in immunocompromised persons. We report a rare case of *Nocardia asteroides complex* causing chronic suppurative otitis media (CSOM) in a patient with idiopathic thrombocytopenic purpura (ITP), and then leading to cortical venous thrombosis. The patient was treated successfully and discharged till follow-up.

Key words: Cortical venous thrombosis, CSOM, *Nocardia asteroides*

INTRODUCTION

Nocardia is an aerobic, gram positive and branching filamentous bacterium causing infections mainly in immunocompromised individuals.[1] Though, pulmonary nocardiosis being the most common manifestation of nocardial disease, other sites of nocardial dissemination include skin, subcutaneous tissues, and the central nervous system.[2] There are scant reports of nocardia causing chronic suppurative otitis media (CSOM). We report a rare case of *Nocardia asteroides complex* causing chronic suppurative otitis media (CSOM) in a patient with idiopathic thrombocytopenic purpura (ITP) and then leading to cortical venous thrombosis (CVT).

CASE REPORT

A 22-year-old female, who was a known case of idiopathic thrombocytopenic purpura (ITP) on corticosteroids with history of splenectomy, presented with intermittent low-grade fever, bilateral ear pain and right ear discharge for the past 3 days at Kasturba Hospital, Manipal, on 16th August 2013. She had bilateral loss of hearing and tinnitus. There was no history of vertigo. She had a past history of intermittent, watery, odourless ear discharge for the last 3 years for which no treatment was obtained.

Access this article online

Quick Response Code:

Website: www.jacmjournal.org

DOI: 10.4103/0972-1282.134461

General physical examination was not significant except for the presence of tachycardia with a pulse rate of 96/min. Examination of the right ear revealed tragal tenderness and purulent discharge in the external auditory canal with skeletonisation of the handle of malleus. Left ear examination showed Grade 1 retraction and congestion of the tympanic membrane. A preliminary diagnosis of chronic suppurative otitis media (CSOM) was performed, and the patient was started empirically on Ciprofloxacin ear drops for the right ear (thrice daily for 7 days).

Culture of the discharge from right ear yielded growth of Nocardia after 48 hours on sheep blood agar [Figure 1]. The isolate showed hydrolysis of urea due to production

Figure 1: Growth of *Nocardiaa steroides complex* on sheep blood agar at 48 hours

Address for correspondence: Dr. Kiran Chawla, E-mail: arunkiranchawla@yahoo.com

of urease enzyme, but hydrolysis of casein, aesculin, hypoxanthine and xanthine was negative. The isolate failed to grow at 45°C. Based on these biochemical tests, the isolate was speciated as *Nocardia asteroides complex*.[1] The isolate showed sensitivity to Amikacin, Gentamicin, Trimethoprim-sulfamethoxazole, Ciprofloxacin, Linezolid and resistance to Amoxicillin-clavulanate, Ceftriaxone, Tetracycline and Meropenem. The patient was started on a specific therapy with Trimethoprim-sulfamethoxazole twice daily for 10 days. She was discharged after observing clinical improvement.

The patient again presented to the hospital after one week of discharge with intermittent fever associated with chills and rigors, four episodes of non-projectile, non-bilious vomiting per day and right post auricular and temporal headache for the past 3 days. There was no history of seizures, blurring of vision, loss of consciousness or focal neurological deficits. Her vitals and routine blood tests were normal. Examination of the right ear revealed subtotal perforation of the tympanic membrane and wet middle ear mucosa. Left ear appeared to be normal on examination. Culture of the swab of the right middle ear mucosa grew *Candida spp.* which was considered to be a colonizer. Blood culture was sterile after 7 days of incubation.

Contrast-enhanced computed tomography (CECT) and magnetic resonance imaging (MRI) of the brain showed features suggestive of CVT involving the right transverse and sigmoid sinus. [Figure 2] CSOM was considered to be the cause of the cortical venous thrombosis as the workup to rule out non-infective causes was negative. She was started on a therapy with Amikacin (500 mg once daily) for 14 days and Linezolid (600 mg twice daily) for 10 days. She was discharged from the hospital after switching to oral Trimethoprim-sulfamethoxazole for 3 months and was advised follow-up after 30 days.

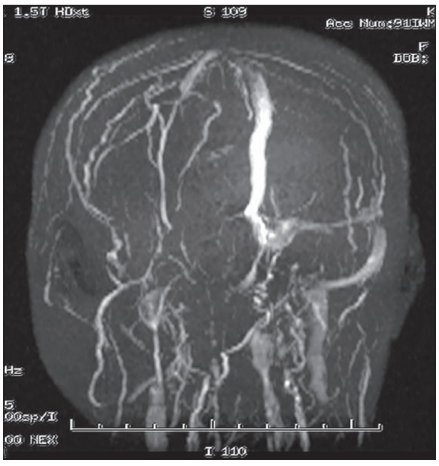

Figure 2: MRI showing right transverse and sigmoid sinus thrombosis up to the confluence and loss of flow related enhancement

DISCUSSION

Nocardiosis is seen in patients with immunosuppression, especially with deficient cell-mediated immunity.[1] Nocardial infection in ITP and post-splenectomy cases has been reported previously in literature.[2,3] A few case reports have documented the association of *Nocardia* spp. and CSOM.[4]

The present case had undergone splenectomy due to uncontrolled vaginal bleeding and hematuria due to ITP and was on steroid treatment. She was diadgnosed to have CSOM caused by *Nocardia asteroides complex* during the first hospital admission. Later she was readmitted within a week with development of CVT involving the transverse and sigmoid sinus.

CVT though potentially treatable, is a serious condition with mortality of about 10%. It often affects young-to-middle-age persons especially females. The infective causes due to middle ear, facial skin infections and penetrating head trauma account for <10% cases. Development of CVT in this case may be due to long, protracted infection of otitits media over a period of 3 years. The sigmoid sinus is prone to thrombosis in patients with otitis media. This complication was seen more so in pre-antibiotic era, but with the advent of antibiotics it has become a rare.[5]

In this patient, nocardia was found to be causing CSOM. Recrudescence of the infection by the same organism was thought as a probable cause leading to the later complication, as the patient reported within a week and other causes excluded. The most common treatment for nocardiosis is trimethoprim-sulfamethoxazole though cases have also been successfully treated with minocycline, fluoroquinolones, aminoglycosides, cephalosporins, carbapenems and linezolid.[1] Our case was treated successfully as per sensitivity report. The subsequent follow-up of the patient showed complete clinical improvement. The present case highlights the virulent potential of nocardia in causing CSOM as well as CVT, and stresses the importance of early diagnosis and prolonged treatment of such cases so as to prevent complication.

REFERENCES

1. Brown-Elliott BA, Brown JM, Conville PS, Wallace RJ Jr. Clinical and laboratory features of the Nocardiaspp. based on current molecular taxonomy. Clin Microbiol Rev 2006;19:259-82.

2. Lai CC, Lee LN, Teng LJ, Wu MS, Tsai JC, Hsueh PR. Disseminated Nocardia farcinica infection in a uraemia patient with idiopathic thrombocytopenia purpura receiving steroid therapy. J Med Microbiol 2005;54:1107-10.
3. Abdi EA, Ding JC, Cooper IA. Nocardia infection in splenectomized patients: Case reports and a review of the literature. Postgrad Med J 1987;63:455-8.
4. Kumar TS, Scott JX, Viswanathan S, Agarwal I, Raj PM, Lalitha MK. Cervicofacial nocardiosis in an immunocompetent child. Acta Paediatr 2005;94:1342-3.
5. Guenther G, Arauz A. Cerebral venous thrombosis: A diagnostic and treatment update. Neurología 2011;26:488-98.

How to cite this article: Chawla K, Gopinathan A, Kudru CU, Nagiri SK. Nocardia causing chronic suppurative otitis media and cortical venous thrombosis. J Acad Clin Microbiol 2014;16:27-9.

Source of Support: Nil. **Conflict of Interest:** None declared.