

Pulmonary Nocardiosis in Immunocompetent Female

Nandini Chatterjee^{1,*}, Mohua Bose², Suvajit Das³, Sudipta Kumar Dey⁴,
Kaushik Mukhopadhyay⁵, Chandan Chatterjee⁶

¹Associate Professor, Dept. of Medicine, Murshidabad Medical College

²Associate Professor, Dept. of Microbiology, Murshidabad Medical College

^{3,4}Assistant Professor, Dept. of Pharmacology, ESI-PGIMSR MC

⁵Tutor, Pharmacology, ESI-PGIMSR MC

⁶Associate Professor, Dept. of Pharmacology, ESI-PGIMSR MC

***Corresponding Author:**

E-mail: rivuc2006@gmail.com

ABSTRACT

Pulmonary nocardiosis, though a well recognized entity, is often missed due to its clinicoradiological similarities with tuberculosis. We present the case of an immunocompetent young girl of eighteen years with fever cough and weight loss, who was later diagnosed to be suffering from Pulmonary nocardiosis.

Key words: Nocardiosis, Lung, Immunity, Fever, Hemoptysis

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INTRODUCTION

The frequency of Pulmonary nocardiosis reported in literature is increasing due to the growing number of immunosuppressed patients, improved isolation techniques and increased clinico microbiological awareness. The diagnosis is delayed due to nonspecific clinicoradiological presentation and difficulty in cultivating the bacteria.^[1]

CASE REPORT

Our patient presented to us with history of recurrent episodes of fever for the last four years. She had been treated with myriads of antibiotics along with two courses of antitubercular drugs. Fever temporarily subsided with every therapeutic misadventure only to reappear with a vengeance after few weeks. This time she had high rise of temperature with productive cough and mild hemoptysis.

O/E She had mild pallor and tachycardia. Respiratory system examination revealed a zone of diminished vesicular breath sounds and scattered crepitations in the left lower part of chest. Examination of lymph nodes, skin, gastrointestinal system, cardiovascular system, and nervous system was noncontributory.

Investigations portrayed a TC-19,000/ cu mm N-80%, L 15% E-3%, B-2% Hb-9.5 gm/dl. Blood biochemistry was unremarkable. HIV screening was

non reactive. ANF was also negative. The Chest X ray revealed multiple well circumscribed, patchy areas of consolidation in both lung fields. USG of the abdomen showed mild hepatomegaly. Blood culture showed no growth. Sputum was sent for AFB stain and culture which came out to be negative too. This prompted us to go for a CT scan of thorax that proclaimed irregular opacities with a tendency to coalesce in both lung fields along with hilar lymphadenopathy and focal bronchial dilatation. CT guided FNAC from lung lesion showed necrotizing inflammation and gram positive beaded organisms with branching filaments. Routine phenotypic test results were characteristic of Nocardia asteroides complex. A CT scan of brain was done to exclude CNS involvement, however it was normal.

The patient was put on linezolid and cotrimoxazol combination for fifteen days, then cotrimoxazol was continued for six months. Her fever subsided within six days and she is doing well till date.



Fig. 1: Chest X ray revealed well circumscribed, patchy areas of consolidation in left lung field.

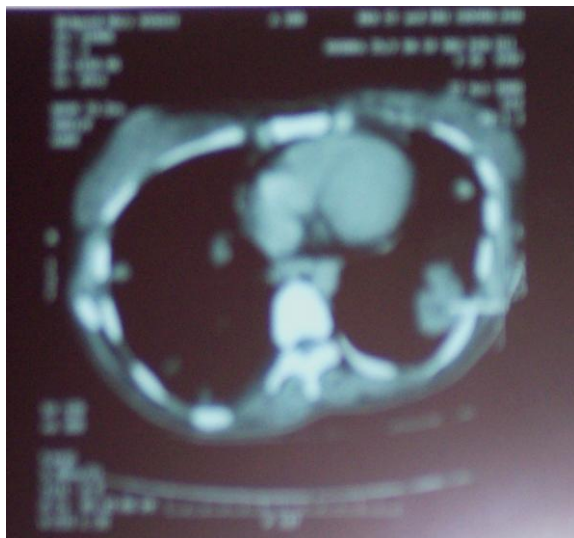


Fig. 2: CT scan of thorax shows irregular opacities in both lung fields with a tendency to coalesce and hilar lymphadenopathy.

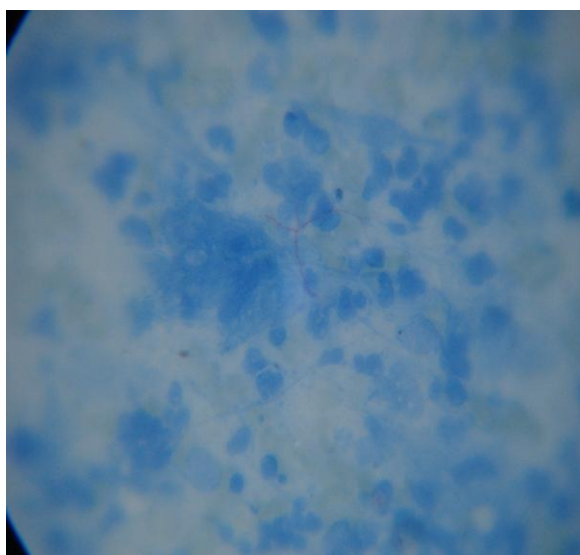


Fig. 3: High power field ($\times 400$) shows organisms with branching filaments in a background of necrotizing inflammation (modified acid fast staining).

DISCUSSION

Pulmonary nocardiosis is usually an opportunistic infection in patients of impaired cell mediated immunity. However, involvement in immunocompetent patients is not unheard of. Prevalence of pulmonary nocardiosis was quoted to be 1.4% in a large Indian study with 1016 patients.^[2] Another indicator study proclaimed prevalence to be 1.9%. All their patients were immunocompetent.^[3] *Nocardia* species are aerobic, gram positive, often beaded bacilli with branching filaments. They are found in soil and water and infection is transmitted by inhalation or traumatic inoculation. In a retrospective study from Thailand with 70 cases it was found to be

mostly prevalent among males. Mean age was 39.7 ± 14.9 . Underlying immunosuppression was found in 80% cases, of which HIV infection was foremost.^[4]

The most common clinical form of nocardiosis infection is lower respiratory tract infection in COPD patients.^[5] The clinical manifestations are not distinctive and mimic tuberculosis. Patients present with fever, weightloss, chest pain and cough, Even pleural effusions, empyema and mediastinitis have been documented. Infection may spread to the skin, kidney and brain. CNS dissemination is found in 30%. Mortality appears to correlate with causative species and the site of infection. It can be as high as 50% in patients with disseminated diseases.^[6] Diagnosis is by smear and culture of sputum or exudates. Multiple specimens are to be sent as simultaneous smear and cultures are positive in only a third of the cases. It is extremely difficult to make precise species level determinations as most *Nocardia* isolates fail to react in many of the standard biochemical reactions utilized in clinical microbiological laboratories. Recently nocardia species identification and taxonomy have been improved by molecular methods such as 16 Sr RNA and hsp 65 gene sequence analysis.^[7] Having said that, it is also to be stressed that such methods are well beyond the scope of our institution.

Therapy classically described is Co-Trimoxazole for six months. However resistance to the drug is increasing. A 5 yr studies with 15 patients showed 80% sensitivity to Co-Trimoxazole.^[8] Commonest consensus is combination therapy of cotrimoxazole with other agents like linezolid, amikacin, imipenam -cilastatin in the acute phase, then maintenance with Co-Trimoxazole for 6 months.

Our case was a lesson for us as she was a teenaged female, immunocompetent, with no predisposing risk factor whatsoever. This fact underscores the point that nocardiosis deserves greater attention in the differential diagnosis of bronchopulmonary diseases.

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