

Pulmonary nocardiosis mimicking tuberculosis

Mahajan V¹, Kashyap A²

¹Dr Vineet Mahajan
Assistant professor, Department of
Chest & TB
Punjab Institute of Medical
Sciences
Jalandhar, Punjab, India
drvineetmahajan14@yahoo.co.in
²Dr Anil Kashyap
Associate Professor, Department of
Pulmonary Medicine
Dayanand Medical College &
Hospital (DMC&H)
Ludhiana, Punjab, India
anildoc11@gmail.comj

Received: 27-10-2014

Revised: 12-11-2014

Accepted: 19-11-2014

Correspondence to:

Dr. Vineet Mahajan
drvineetmahajan14@yahoo.co.in
09888857733, 01812464674

ABSTRACT

Pulmonary nocardiosis is an infrequent and severe infection due to Nocardia species, microorganisms that may behave both as opportunists and as primary pathogens. Diagnosis of pulmonary nocardiosis is frequently delayed and a high level of suspicion is required in patients with underlying diseases or chronic corticosteroid therapy. Hereby we are presenting a case of pulmonary nocardiosis mimicking tuberculosis in an immunocompromised patient.

Key words: Nocardia, tuberculosis, immunosuppression, COPD, pneumonitis

Introduction

Pulmonary nocardiosis is an acute or suppurative chronic disease occurring in immunocompromised patients mimicking tuberculosis, mycotic infection or malignancy. It is caused by aerobic actinomycetes which usually originate from soil. Clinical manifestations include inflammatory endobronchial masses or localized or diffuse pneumonias, which may be accompanied by cavitation, abscess formation, pleural effusions, or empyemas. Sulfonamides are first-line antimicrobial therapy for nocardiosis.

Case Report

A 37 years old male was admitted to our department with complaints of productive cough, progressive exertional dyspnoea,

high grade fever from last 7 days. On detailed evaluation he had a long history of intermittent breathlessness, cough, and wheeze since childhood. Initially he used to have 2-3 attacks of dyspnoea but since one year he was getting the attacks more frequently and taking oral corticosteroids for symptomatic relief only.

On physical examination, the patient was febrile with a pulse rate of 124/min, respiratory rate of 28/min and a blood pressure of 136/72 mmHg. Chest examination was unremarkable on inspection, palpation and percussion. On auscultation bilateral crepitations and rhonchi were audible.

Routine investigations showed; Hb - 13 gm%, TLC - 13900/mm³, DLC - neutophils 75%, lymphocyte 20%,

eosinophil 5%. Liver function tests, renal function tests and serum electrolytes came out to be normal. Arterial blood gas analysis showed type one respiratory failure. Chest x ray showed bilateral fluffy infiltrates more concentrated in lower zones (Fig.1). High Resolution CT scan showed bilateral mucoid shadows with bronchiectatic changes in bilateral basal segments, paracardiac and parahilar regions (Fig.2).



Fig.1 Bilateral fluffy infiltrates

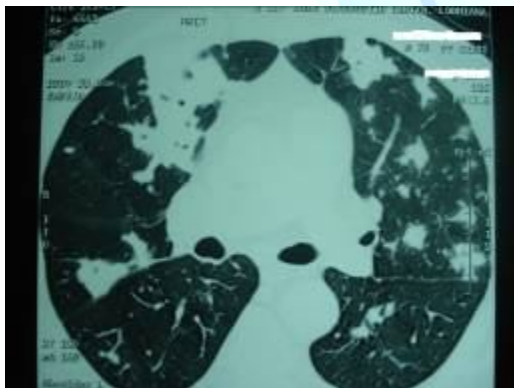


Fig.2 Bilateral mucoid shadows with bronchiectatic changes

Sputum samples for acid-fast bacilli (mycobacterium) were found to be negative on three consecutive days but came out to be positive for nocardia (weakly acid fast). Tests for HIV, HBsAg and HCV were negative. He was started in cotrimoxazole

(2 double strength tablets twice a day) , amikacin (1 gram intravenous once a day) and ceftriaxone (2 gram intravenous twice a day). Brocho alveolar lavage done after patient got stabilized came out to be positive for Nocardia. BAL fluid culture showed growth of nocardia asteroides sensitive to gentamicin, linezolid, cotrimoxazole, amikacin, vancomycin, ceftriaxone and imipenem. Antibiotics were continued for 2 weeks in intravenous form and 4 weeks thereafter as oral cotrimoxazole after confirmation of sensitivity and patients improved clinically.

Discussion

Nocardiosis, caused by gram-positive, aerobic actinomycetes is a rare and serious infection mainly affecting immunocompromised with approximately 65% of cases occurring in individuals with some compromise of host defense systems. [1] Human disease is most commonly caused by *Nocardia asteroides* but *N. brasiliensis*, [2] *N. farcinica* and *N. transvalensis* [3] have also been described rarely. Being a saprophyte, the organism causes pulmonary infection by entering via the respiratory tract and person-to-person transmission is rare. [4]

The manifestations of nocardiosis can be solely pulmonary (75–80%), cutaneous or neurological (44%) but virtually any organ system may be involved. [5] When there is involvement of two or more noncontiguous organs, with or without CNS involvement, the disease is said to be disseminated. The incidence of disseminated disease is 25–40%. [6] The risk of pulmonary and disseminated disease is greater among persons with deficient cell-mediated immunity especially that associated with lymphoma, transplantation, [7] glucocorticoid therapy or AIDS.

Nocardiosis has also been reported to complicate pulmonary alveolar proteinosis and rarely sarcoidosis.^[8]

Clinical features are relatively nonspecific with a chronic course in 70% before diagnosis.^[9] Pulmonary nocardiosis tends to present typically as a subacute pneumonia, the symptoms appearing over the course of several days or weeks. Dyspnea, pleuritic pain and hemoptysis are less common.^[10] Radiologically, it may present as consolidation, well – circumscribed nodules, large, multiloculated abscesses or cavities.^[11] The major differentials are pneumonia, tuberculosis, bronchogenic carcinoma or lung abscess. Pulmonary nodules with multiple cavitations can easily mimic actinomycosis, septic emboli, metastasis, sarcoidosis, wegener’s granulomatosis or other fungal infections.^[12] Pulmonary nocardiosis mimics pulmonary tuberculosis in clinical symptoms and radiological characteristics, and it is often wrongly treated with anti-tuberculosis drugs.^[13] A classic radiographic picture of tuberculosis that is unresponsive to medication should raise the suspicion of Nocardia infection. In patients with AIDS having superior bilateral infiltrates, pulmonary nocardiosis should be taken into account because, in AIDS patients, pulmonary tuberculosis does not normally show cavitation or upper lobe lesions.

The diagnosis should always be based on isolation of organism in respiratory secretions which can be sputum or bronchial washings. The invasive diagnostic techniques are used when sputum fails to prove the diagnosis. The organism grows relatively slowly and colonies may take 2 to 4 weeks to appear or to take their characteristic appearance.

Cultures should be maintained for at least 3 weeks before being discarded as negative. The microbiology laboratory should be informed of the suspicion since the incubation time of the cultures must be prolonged, and the laboratory should also be advised to use concentration and decontamination techniques with the sample.^[14] Molecular techniques can also be used for diagnosis like 16s rRNA sequence analysis, Restriction Fragment Length Polymorphism (RFLP) and Polymerase Chain Reaction (PCR) but in reference laboratories only.^[15] To rule out dissemination in patients with nocardial pneumonia, a careful history should be obtained and a thorough physical examination performed. CT or MRI of the head, with and without contrast should be undertaken if signs and symptoms suggest brain involvement.

Antibiograms should be obtained in the samples isolating nocardia species because of the need for multiple antibiotics in severe infections and variable susceptibility of these organisms to different antibiotics.

Management includes antimicrobial therapy in all cases with surgical drainage wherever needed. Cotrimoxazole is the drug of choice which can be given alone or in combination with other drugs like imipenem, amikacin, third-generation cephalosporins or minocycline in serious cases. Experimental studies using combination of antimicrobial have demonstrated synergy in vitro also supported by clinical observation with imipenem-amikacin, imipenem-cefotaxime and amikacin-cefotaxime.^[16] The duration of therapy should be at least 6 months in localized disease and one year or more in

disseminated form depending upon severity of infection and the host immune status.

Prognostically this infection can be divided into two groups, one receiving immunosuppressive antineoplastic or corticosteroid therapy (mortality 80-100%) and the other group constitutes previously healthy patients and those with untreated underlying disease (mortality 15-20%). The difference results from the extensive pulmonary necrosis (not dissemination) in those receiving immunosuppressants.^[17] Disseminated nocardiosis has a poor prognosis with a mortality rate >85 % in immunocompromised hosts.^[18]

References

1. Frazier A, Rosenow E, Roberts G. Nocardiosis: a review of 25 cases occurring during 24 months. *Mayo Clin Proc* 1975;50:657-63.
2. Wadhwa V, Rai S, Kharbanda P, Kabra S, Gur R, Sharma VK. A fatal pulmonary infection by nocardia brasiliensis. *Indian Journal of Medical Microbiology* 2006;24:63-4.
3. McNeil MM, Brown JM, Georghiou PR, Allworth AM, Blacklock ZM. Infections due to nocardia transvalensis: clinical spectrum and antimicrobial therapy. *Clin Infect Dis* 1992;15:453.
4. Palmer DL, Harvey RL, Wheeler JK. Diagnostic and therapeutic considerations in Nocardia asteroides infection. *Medicine* 1974;53:391-401.
5. Sud S, Buxi TBS, Anand I, Rohatgi A. Case series: nocardiosis of the brain and lungs. *Indian J Radiol Imaging* 2008;18:218-21.
6. Wakhlu A, Agarwal V, Dabadghao S, Prasad KN, Nityanand S. Nocardiosis in patients of chronic idiopathic thrombocytopenic purpura on steroids. *J Assoc Physicians India* 2004;52:501-3.
7. Biglarnia AR, Wadstrom J, Tufveson G, Eriksson BM. Pulmonary nocardiosis with brain abscess in a sensitized kidney transplant recipient with history of repeated graft loss and HLA-antibody depletion treatment – a case report. *Upsala J Med Sci* 2008;113:111-6.
8. Jastrzembki SA, Teirstein AS, Herman SD, Depalo LR, Lento PA. Nocardiosis presenting as an anterior mediastinal mass in a patient with sarcoidosis. *The Mount Sinai Journal of Medicine* 2002;69:350-3.
9. Menéndez R, Cordero PJ, Santos M, Gobernado M, Marco V. Pulmonary infection with Nocardia species: a report of 10 cases and review. *Eur Respir J* 1997; 10:1542-6.
10. Betriu C. Infections of Nocardia. *Enferm Infec Microbiol Clin* 1997;15:154-60.
11. Feigin DS. Nocardiosis of the lung: chest radiographic findings in 21 cases. *Thor Radiol* 1986;159:9-14.
12. Hwang JH, Koh WJ, Suh GY, Chung MP, Kim H, Kwon OJ et al. Pulmonary nocardiosis with multiple cavitory nodules in the immunocompromised HIV- negative patient. *Internal Medicine* 2004;43:852-54.
13. Chopra V, Ahir GC, Chand G, Jain PK. Pulmonary nocardiosis mimicking pulmonary tuberculosis. *Ind J Tub* 2001;48:211-3.
14. Mari B, Monton C, Mariscal D, Lujan M, Sala M, Domingo C. Pulmonary nocardiosis: clinical experience in ten cases. *Respiration* 2001;68:382-8.
15. Agterof MJ, Van der Bruggen T, Tersmette M, Borg EJ, Van den Bosch JMM, Biesma DH. Nocardiosis: a case series and a mini review of clinical and microbiological features. *Netherlands J Med* 2007;65:199-202.
16. Gombert ME, Aulicino TM, du Bouchet L, Silverman GE, Sheinbaum WM. Therapy of

Mahajan et al: Pulmonary nocardiosis

experimental cerebral nocardiosis with imipenem, amikacin, trimethoprim-sulfamethoxazole, and minocycline. *Antimicrob Agents Chemother* 1986; 30:270–3.

17. Presant CA, Serpick AA. Factors affecting survival in Nocardiosis. *Am Rev Respir Dis* 1971;103:882–3.
18. Lederman ER, Crum NF. A case series and focused review of nocardiosis: clinical and microbiologic aspects. *Medicine* 2004;83:300-13.

Cite this article as: Mahajan V, Kashyap A. Pulmonary nocardiosis mimicking tuberculosis. *Int J Med and Dent Sci* 2015; 4(2):874-878.

**Source of Support: Nil
Conflict of Interest: No**

