SAS Journal of Medicine ISSN 2454-5112
SAS J. Med., Volume-2; Issue-4 (Jul-Aug, 2016); p-79-83
Available online at http://sassociety.com/sasjm/

Case Report

Pleuroplumonary Nocardiosis in an Immunocompetent Host

H.K. Sethy[1], B. Meher[2], S. Pradhan[3], M. Patro[4]
[1]Associate Prof. Dept. of pulmonary Medicine, SCB medical College, Cuttack, Odisha-753007.
[2,4]P.G, Dept. of pulmonary Medicine, SCB MCH, Odisha-753007
[3]M.D, Dept. of pulmonary Medicine, SCB MCH, Odisha-753007

***Corresponding author**
H.K. Sethy
Email: sethy.drhemantakumar@gmail.com

Abstract: *Nocardia* species are found in soil rich in organic matter and in healthy gingiva, as well as periodontal pockets as oral microflora. Some species are pathogenic with low virulence, hence cause opportunistic infection most frequently in immunocompromised hosts, though occasionally in immunocompetent individuals. Pleuropulmonary nocardiosis is a rare occurrence. We report such a case in a 72yrs old mason.
Keywords: Nocardiosis, opportunistic, pleuropulmonary, elderly.

INTRODUCTION

Nocardia cells exist ubiquitously in soil, organic matter, or water [1] and help in decaying organic matter. As an oral microflora, they are found in normal gingiva and periodontal pockets along with other species such as Actinomyces, Arthromyces and Streptomyces [2]. Among 85 species till identified, the human disease 'nocardiosis' is associated with *N. asteroides, N. brasiliensis, N. otitidiscaviarum, N. farcinica, N. abscessus, N. nova, N. transvalensis, N. pseudobrasiliensis* and N. *africana* [3]. *Nocardia asteroides* is the predominant human pathogen followed by *N. brasiliensis, Nocardia farcinica* and *Nocardia nova*. When pulmonary infection is usually caused by *N. asteroides*, the skin infection is commonly associated with *N. brasiliensis* [4]. The organisms are gram-positive, branching filamentous, weakly acid – fast, strictly aerobic, slow growing bacilli which may fragment into bacillary and coccoid elements. The disease is most common in men than women (2:1) without apparent racial predilection. All ages from infants to old are susceptible. The mean age at diagnosis is fourth decade of life. The disease affects immunocompromised hosts especially with impaired T-cell–mediated immunity [4], but can affect individuals without detectable defficiency of humeral/ cell mediated immunity. The usual risk factors include malignancy, chronic lung disease, chronic granulomatous diseases, humeral defects, diabetes mellitus or immunosuppression of any cause [4]. The estimated incidence in United states is 500-1000 cases per year [5]. Transmission from animal-to-human and human-to-human has not been reported [6]. They usually spread by inhalation of dusts, whereas direct inoculation through puncture wound/abrasions is unusual [7].

CASE REPORT

A 72yrs old male mason, non-smoker, non-alcoholic presented with irregular low grade fever, right chest pain and weight loss over last 3months with history of 30 yrs'dust exposure admitted to department of pulmonary medicine, SCB medical college cuttack, Odissa . He had no history of asthma, COPD, DM or rheumatlogical disorders or prolonged treatment history of immunosuppressants or recent trauma.

At the time of hospitalization, the patient was conscious, well oriented, afebrile and had PR 72/min, RR 18/min, BP 120/80 mmHg, SpO2 96% with 1x1.5cm firm, non-tender right scalene node without dyspnea, clubbing, cyanosis, pallor and external bodily injury. On chest examination, he revealed decreased VF, percussion note and breath sound on right infrascapular region. FNAC of the node revealed non-specific hyperplasia. CXR PA view revealed encysted pleural effusion. There was periphreal blood leukocytosis (TLC 13,300/cmm) and neutrophilia (86%). Blood sugar, urea, creatinine, sodium and potassium were in normal range. Serology for HIV, HBV or HVC was negative. ECG and echocardiogram were normal. Sputum AFB staining was negative but sputum aerobic culture revealed Enterobacter species sensitive to Amikacin and levofloxacin. Ultrasonography confirmed pleural effusion without any abdominal or pelvic abnormalities. Thoracocentesis revealed serous pleural fluid, which on analysis showed protein 4.5gm/L, sugar 98mg%, ADA 57 IU/ml, LDH 548 IU/dl, and predominant lymphocytes (70%) without

malignant cells. Basing on sputum culture and pleural fluid characterstics, antibiotics and CAT I DOTs were started. After 6wks, AFB culture of plural fluid came out negative. But after 2months of IP CAT I DOTS, the patient returned with same fever without radiological improvement. HRCT thorax revealed patchy consolidation on RML medial and RLL posterior basal segments with enlarged pretracheal lymph nodes. FOB showed hypertrophic, hypersecretory mucosa in right lung but atrophic mucosa in left lung without any intraluminal growths. Bronchoalveolar lavage fluid (BALF) cytology from right side revealed plenty of polymorphs, cystic macrophages, plenty of pus cells and plenty of branched filamentous bacteria on gram staining. Subsequent Modified Z-N staining of BALF and pleural fluid suggested nocardia species. After administration of oral co-trimoxazole as a first line drug, patient became afebrile and showed clinicoradiological improvement after one month follow up.

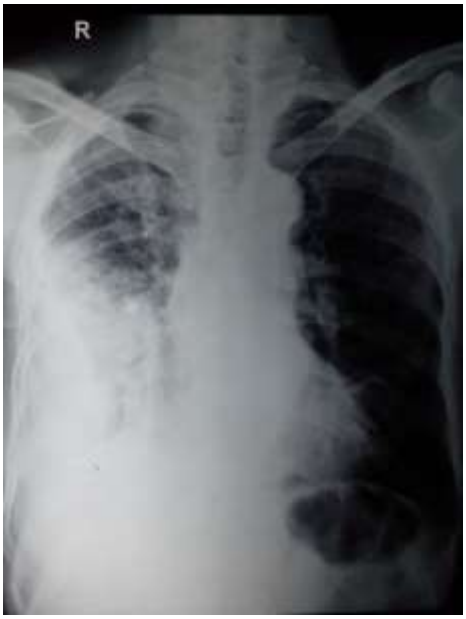

Fig-1: CXR PA view showing encysted pleural effusion.

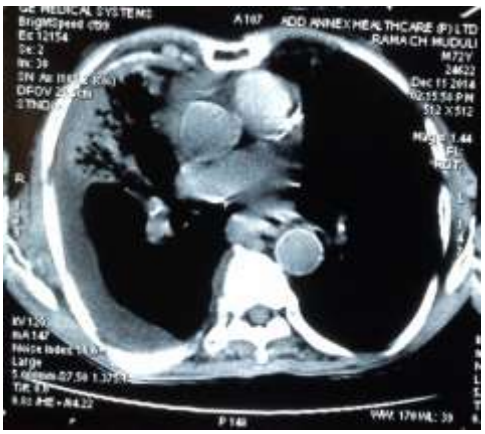

Fig-2: CT Axial view showing pulmonary infiltration with right pleural effusion and pleural calcification.

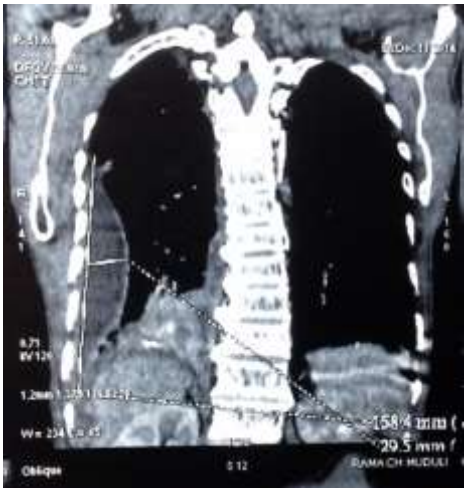

Fig-3: CT Coronal view showing encysted pleural effusion with pleural thickening.

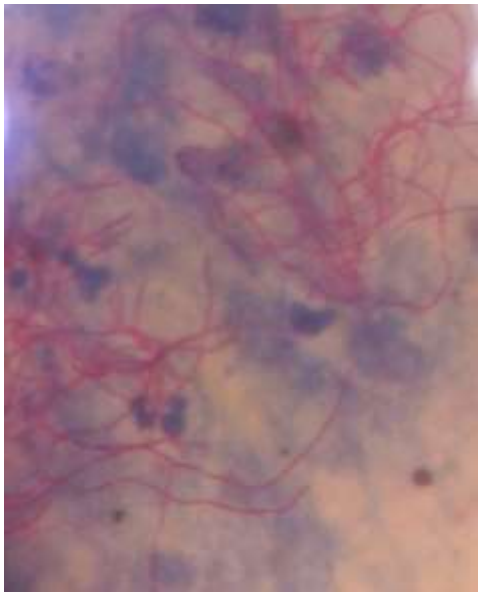

Fig-4: Photomicrograph showing Nocardia spp. as branching filaments in BAL fluid on 1% ZN staining.

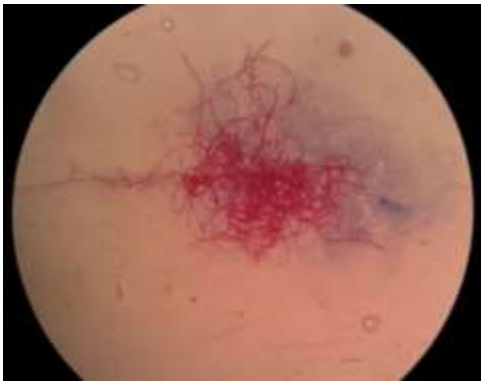

Fig-5: Photomicrograph showing Nocardia spp. in hyphal form in Pleural fluid on 1% ZN staining.

DISCUSSION

Nocardiosis manifests in Pulmonary, Cutaneous and Disseminated forms. Among these, pulmonary nocardiosis is rare but more common than other two forms. It usually affects immunocompromised/occasionally immunocompetent hosts, whereas cutaneous nocardiosis is usually seen in immunocompetent individuals [8]. Disseminated nocardiosis occurs by spread from cutaneous /pulmonary origin due to defective T-cell-mediated immunity and due to effect of enzymes possessed by bacteria.

The clinical and radiological findings in nocardiosis are non-specific. Pulmonary nocardiosis usually presents with subacute onset, but may manifest as an acute or chronic pneumonitis. It produces an indolent progressive pneumonia, may run a chronic course and show a granulomatous reaction mimicking PTB in immunocompetent subjects [9]. In immunocompromised hosts, the lesions develop rapidly causing an acute pyogenic necrotizing pneumonia. The constitutional symptoms are weight loss, malaise, night sweats and fever (low grade to spiking with chills). Pulmonary symptoms like cough, copious purulent/bloody sputum, pleuritic chest pain and dyspnea are more common in at least 40%-75% of patients with disseminated nocardiosis. In 25% cases, pulmonary focus is subclinical/ healed/can not be detected at time of presentation. In 25% cases, CNS is involved.

In immunocompromised hosts, pulmonary infection results in abscess formation, rarely granulomas and usually haematogenous or lymphatic dissemination to the skin or central nervous system [4]. Clinical manifestations of established pulmonary infection are endobronchial inflammatory masses, pneumonia, lung abscess, cavitary disease, effusion and empyema.

Radiologic features are multiple pulmonary infiltrates, solitary pulmonary nodule, lung abscess and pleural involvement resulting in pleural thickening, pleural effusions and empyema [10]. The parenchymal infiltration mimicks tuberculosis or bronchoalveolar cell carcinoma. About 75 % of patients with nocardiosis have lung involvement [11], as many as 50 % of patients with pulmonary nocardiosis have a pleural effusion [12, 13] and 25% cases may be associated empyema [14]. The pleural fluid is exudative, ranging from serous fluid to frank pus. Nocardiosis has a variable prognosis, depending on the site of infection, extent of infection, and underlying host factors [15].

Diagnosis is made by modified Z-N staining using 1% sulphuric acid and culture (5-21days) of sputum, BAL, PF specimens and tissue biopsy. Gram's stain of clinical specimens is useful for early presumptive diagnosis of nocardiosis. Nocardia is an aerobic, delicate filament(<2µm) with pronounced branching as against anaerobic, slightly thicker, straight and less branching filaments of actinoomyces needing culture for species differentiation [7] and hydrolysis agar test adding casein, tyrosine, xanthine for subspecies differentiation followed by DST. Early diagnosis is not easy because of its slow growth rate and overgrowth of other organisms, but can be possible by availability of molecular identification facilities (16S RNA gene) [16]. In the Indian literature, there are few isolated case reports of Nocardia asteroides causing hydropneumothorax [17], empyema [18] and pyopneumothorax [19]. The mortality exceeds 80% in brain involvement and in other forms; mortality is 50%, even with appropriate therapy. The disease requires at least 6 months of treatment, preferably with trimethoprim/sulfamethoxazole or high doses of sulfonamides. In patients who do not respond to sulfonamide treatment, the drugs like ampicillin, erythromycin, or minocycline, may be added. The combination of sulfonamide, ceftriaxone, and amikacin has also shown promise [7]. Surgical treatment includes drainage of abscesses and excision of necrotic tissue.

Our case was a 72yrs old male mason with prolonged history of dust exposure, presented with pleural effusion and parenchymal infiltration, bronchoscopy showed no endobronchial masses. Both BALF and pleural fluid revealed nocardia species on modified gram staining indicating definite pulmonary nocardiosis rather than contamination and it well responded to co-trimoxazole.

CONCLUSION

Pleuropulmonary nocardiosis may occur in ageing process without obvious immunosuppressive drugs/disease or trauma. Occupational exposure to dusts may be the risk factor for seedling of organisms by inhalation process. Tuberculosis as a differential diagnosis should be excluded as early as possible by CBNAAT if available.

ABREVIATIONS

FOB- Fibreoptic bronchoscopy, BALF- Bronchoalveolar lavage fluid, CBNAAT- Cartridge-Based Nuclic Acid Amplification Test

REFERENCES

1. 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: 1542-1546.
2. Roth, GD, Thurn AN; Continued study of oral nocardia. Journal of dental research, 1962; 41: 1279–92.
3. Zaatreh M, Alabulkarim W; Images in clinical medicine. Disseminated central nervous system nocardiosis. N Eng J Med., 2006; 354: 2802.
4. Beaman BL, Beaman L; Nocardia species: host-parasite relationships. Clin Microbiol Rev., 1994; 7(2):213-64.
5. Centers for Disease Control and Prevention. Available at http://www.cdc.gov/nocardiosis/infection. Accessed: June 7, 2013.
6. Bennett NJ, Domachowske J, Johann-Liang R; Pediatric Nocardiosis, 2007.
7. Nocardiosis (Professional Guide to Diseases (Eighth Edition)) - WrongDiagnosis.com. Retrieved 2007-07-12.
8. Rapini, Ronald P, Bolognia, Jean L, Jorizzo, Joseph L; Dermatology: 2-Volume Set. St. Louis: Mosby, 2007; ISBN 1-4160-2999-0.

9. Apisarnthanarak A, Razavi B, Bailey T; Disseminated Nocardia asteroides presenting as pulmonary non-caseating granulomas in a patient with Waldenstrom macroglobulinemia. Infection. 2002;30(1):38-40.
10. Heffner JE; Pleuropulmonary manifestations of actinomycosis and nocardiosis. Semin Respir Infect, 1988; 3: 352-61.
11. Uttamchandani RB, Daikos GL, Reyes RR, Fischl MA, Dickinson GM; Nocardiosis in 30 patients with advanced human immunodeficiency virus infection: clinical features and outcome. Clin Infect Dis., 19894; 18: 348-353.
12. Kramer MR, Uttamchandani RB; The radiographic appearance of pulmonary nocardiosis associated with AIDS. Chest, 1990; 98: 382-385.
13. Feigin DS; Nocardiosis of the lung: Chest radiographic findings in 21 cases. Radiology, 1986; 159: 9-14.
14. Lerner PI; Nocardia species. In : Mandell GL, Bennett JE, Dolin R, ed. Mandell, Douglas and Bennett's Principles and Practice of Infectious Diseases; 4th edn; Voll II. New York: Churchill Livingstone, 1995: 2273-80.
15. Smego RA Jr, Moeller MB, Gallis HA; Trimethoprim-sulfamethoxazole therapy for Nocardia infections. Arch Intern Med., 1983; 143(4):711-8.
16. Kiska DL, Hicks K, Pettit DJ; Identification of medically relevant Nocardia species with an abbreviated battery of tests. J Clin Microbiol., 2002; 40: 1346-51.
17. Gowrinath K, Das S, Ranjitham M, Sekhar U, Thanasekaraan V; Nocardial hydropneumothorax. Indian J Chest Dis Allied Sci., 2004; 46:51-3. 4.
18. Kulkarni SA, Kulkarni AG; Disseminated nocardiosis. J Assoc Physicians India, 1991; 39:779-80. 5.
19. Gupta E, Dhawan B, Thabah MM, Das BK, Sood S, Kapil A; Nocardia pyopneumothorax in an immunocompetent patient. Indian J Med Res., 2006; 124:363-4.