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A Case of Pulmonary Nocardiosis: Mimicking the Mimicker

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Introduction

Nocardia is a gram-positive, weakly acid-fast, filamentous bacteria belonging to Actinomycetes. Species of Nocardia genus are aerobic organisms. They are ubiquitous in environment and are present as saprophytic organisms in fresh and salt water, soil, dust and decaying vegetation.[1] Currently about 30 species of Nocardia have been described which can cause disease in humans. Among them, Nocardia asteroides is responsible for about 70% of the infections caused by these organisms. Other human pathogens include *N. nova complex, N. abscessus, N. transvalensis complex, N. farcinica and N. brasiliensis*[2].

Nocardiosis primarily effects lungs (75–80%) and the other organs that can be effected are brain, skin and rarely kidney and liver[3]. In the lungs it may produce disease ranging from acute fulminating to chronic disease. In skin and other viscera, it produces chronic suppuration, sinuses, and abscess formation. The cerebral tissue is more susceptible to Nocardia species, and the brain is most likely to be the target organ in hematogenous dissemination consequent to pulmonary nocardiosis[3].

Nocardiosis occurs most commonly in immunocompromised patients such as patients with lymphoreticular malignancy, acquired immune deficiency syndrome, patients with organ transplant, and those receiving high-dose corticosteroids[4,5]. Suppression of cellular immunity appears to play a key role in the establishment of *Nocardia* infection[6]. Although it is usually seen in immunocompromised hosts, isolated cases have been reported in immunocompetent hosts too, who donot have any definable predisposing condition.[7] It can affect immunocompetent host by impairing bronchial defences by damaging ciliated epithelial cells especially in COPD and bronchiectasis patients[8].

Pulmonary nocardiosis (PN) is an infrequent but severe infection. It is usually acquired by the direct inhalation of *Nocardia* species from contaminated soil. Person to person transmission is rare.[9] The clinical presentation of pulmonary nocardiosis is quite variable and nonspecific. It can present as endobronchial masses, localized or diffuse pneumonias, cavitation, abscess formation, pleural effusions, or empyemas.[10] The chest radiographic manifestations are pleomorphic. Consolidations, interstitial pattern, cavities or nodules can be seen. Upper lobes are more commonly involved.[11]

When observed microscopically, either in Gram-stained smears of clinical specimens or cultures, *Nocardia* organisms are branching, beaded,

filamentous, gram-positive bacteria. In acid fast staining with 20% H_2SO_4, they are seen as short and stout acid fast bacilli.[11] If sputum examinations do not yield the diagnosis in a suspected case and the diagnosis cannot be made easily from lesions elsewhere in the body, more invasive diagnostic procedures like bronchoscopy, needle aspiration, and open lung biopsy should be performed.[11]

Nocardiosis has a variable prognosis, depending on the site of infection, extent of infection, and underlying host factors. Ninety percent of pleuropulmonary infections can be cured with appropriate therapy. Sulfonamides are first-line antimicrobial therapy for nocardiosis. Cotrimox-azole alone is highly effective against majority of isolates of *Nocardia spp* .But sometimes combination antimicrobial therapy with amikacin, imipenam or ceftriaxone is required. Therapy must be prolonged to prevent relapses. The duration of treatment for nocardiosis depends on disease site. For pulmonary involvement, therapy is usually continued for 6 to 12 months.[3]

Because of its non specific clinical and radiological features, and the microbiological diagnosis is often difficult, pulmonary nocardiosis mimics other lung diseases like lung carcinoma, lung abscess, pulmonary tuberculosis, mycotic infection. The present case highlights the need for suspicion of pulmonary nocardiosis when clinical and radiological picture mimics PTB but sputum is negative for tubercle bacilli either in the direct smears, PCR technique or cultures.

Case Report

A 42-year-old male presented with fever, productive cough with episodes of hemoptysis and breathlessness from one week and altered sensorium and tremors since 3 days. He was a chronic alcoholic, with history of binge drinking from 1 month and stopped since 3 days. He did not have any other significant past, personal or drug history.

On examination, he was conscious but drowsy. He was febrile with body temperature of 102^0F, pulse rate was 124beats/min, respiratory rate was 28 cycles/min and Blood pressure was 114/70mmHg. His O2 saturation was 88%. On systemic examinations, fine crepitations were heard in all the areas of right lung. Other systems examination was unremarkable.

His blood investigations revealed TLC-17,250cell/mm3. Other investigations (Hb, platelet count, RBS, RFT, LFT, FBS, PPBS, HbA1C) were normal. VCTC was negative. Chest x-ray showed non-homogenous opacities, infiltrations and two thin walled cavities in right lung upper and middle zone. (fig.1)

Differential diagnosis of community acquired pneumonia or pulmonary tuberculosis in alcohol withdrawl state was made and patient was started on Inj.Linezolid(600mg bid), Inj.meropenem (1g tid) and bronchodilators. 4hrs after admission, patient became tachypneic and his oxygen saturation was not maintained on oxygen face mask. Then patient was intubated and kept on ventilator. Despite broad-spectrum antimicrobial therapy, patient's condition was deteriorating.

ATT was planned to start but several sputum samples collected and tested for the presence of acid-fast bacilli, were negative. Sputum for GeneXpert was also negative. Sputum Gram stain revealed Gram positive thin branching filaments (figure 2). Modified Ziehl-Neelsen staining showed branching Acid fast bacilli consistent with the morphology of *Nocardia* species(Figure 3). Culture and sensitivity of tracheal aspirate identified the organism as Nocardia. Tablet Cotrimoxazole (160/800mg DS tablets bd) was added to meropenem. Patient improved clinically and radiologically and was discharged after 15 days and advised to continue Cotrimoxazole for 9 months. Patient is coming for follow up and is doing well.

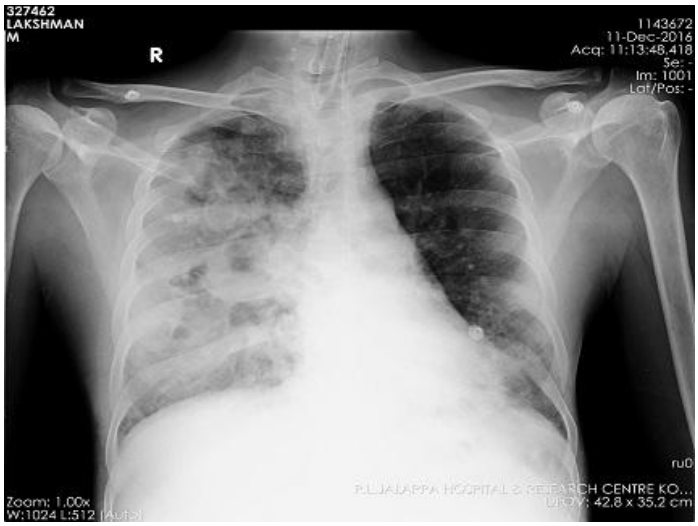

Figure 1: Chest x-ray showed non-homogenous opacities in right lung, upper, middle and lower zones and two thin walled cavities in right lung middle and lower zones.

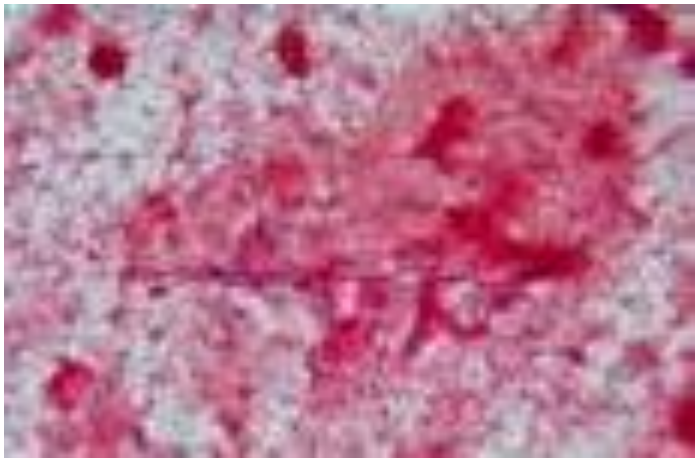

Figure 2: Sputum Gram stain revealed Gram positive branching filaments.

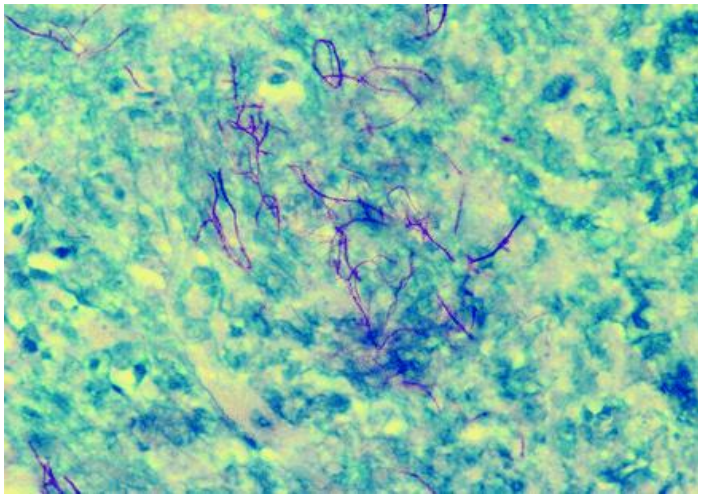

Figure 3: Modified Ziehl Neelson staining showed branching Acid fast bacilli.

Discussion

Nocardia was first isolated from cattle by Nocard in 1888 and a case of human infection of nocardiosis with cerebral abscesses was reported by Eppinger in 1890[11]. Since then, many cases of nocardiosis have been reported which primarily effected lungs. Prevalence of pulmonary nocardiosis was quoted to be 1.4% in a large Indian study with 1016 patients.[12]

Pulmonary nocardiosis can have a acute fulminating or chronic course. In our case, symptoms were present for 1 week before presenting to the hospital and are severe as patient presented with respiratory failure. Disease manifestations of nocardiosis are determined by type of strain, infection site, ability to survive initial neutrophilic leukocyte phagocytic attack and the nature of the immune response. T-cell–mediated immunity is the principal protective immune response to nocardiosis. Therefore, nocardiosis is most problematic in individuals with impaired T-cell–mediated immunity. Our patient was a chronic alcoholic. Though he neither have any definable predisposing condition of immunosupression not not on any immunosup-pressant drugs, chronic alcoholism itself could have lead to impaired cell mediated immunity.

Our patient presented with low grade fever, cough with hemoptysis and breathlessness. Chest radiograph showed non-homogenous opacities in right lung, upper, middle and lower zones and two thin walled cavities in right lung middle and lower zones. Thus our case was mimicking pulmonary tuberculosis both clinically and radiologically. Pulomonary TB itself is called a mimicker as it can mimick many disease processes in lungs like pneumonia, malignany, lung abscess etc. pulmonary nocardiosis can mimick pulmonary TB, the mimicker itself. In countries where tuberculosis is very common, antituberculous drugs are usually started on basis of radiology and clinical symptoms. Similar cases mimicking pulmonary tuberculosis had been reported.[13,14] Pulmonary nocardiosis, though a well recognized entity, is often missed due to its clinicoradiolo-

gical similarities with tuberculosis. Our report emphasizes that a high level of clinical suspicion is required in patients without risk factors. In a patient with pneumonia if the lung infection responds poorly to antimicrobial therapy for community acquired pneumonia, pulmonary nocardiosis should be considered and a careful search for evidence of the organism is necessary.

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