A cavitary lesion in the lung crossing the fissure

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CASE SUMMARY

A 60-year-old dravidian male hailing from Chamarajanagar, Karnataka, agriculturist by occupation, who smoked cannabis five-five times everyday for the last 30 years was admitted to our emergency department (ED) with history of cough and breathlessness for a month. Breathlessness was of acute onset and the patient could not walk for more than 100 m. Productive sputum was purulent in nature and was less than 50 ml/day. Two courses of antibiotics (ciprofloxacin and azithromycin) received from his family practitioner for five days each did not give any relief to his symptoms. He was under treatment for COPD with steroid bursts for three-five days (prednisolone 5 mg) five-six times per year and bronchodilators during acute exacerbations since three years. He used to smoke around five beedis (hand rolled tobacco leaves) every day and consumed alcohol socially for the last 30 years.

Physical examination

At the time of presentation to the ED, he was cyanosed and had bilateral pitting pedal edema with an elevated JVP (5 cm) with prominent "a" and "v" waves. Vital signs revealed tachypnea and tachycardia with a blood pressure of 110/60 mm Hg.

Respiratory system examination revealed a barrel shaped chest with kyphosis. Hyper resonant note was heard on percussion all over the lung fields with obliteration of cardiac and liver dullness. Bilateral rhonchi and basal crepitations were heard. Heart sounds were muffled with a short systolic murmur in the tricuspid area. Per abdominal examination revealed a non-tender liver palpable just below the costal margin.

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Laboratory findings

The CBC count showed hemoglobin of 12.6 g/dl, leucocytosis with shift to left (14000 cells / micro liter, with 89% neutrophils) and an elevated ESR of 80 mm at 1 h. Blood chemistry analysis showed a BUN of 62 mg/dl, a creatinine level of 1.4 mg/dl, and a random blood sugar of 166 mg/dl. Liver function tests and serum electrolytes were within normal limits and he was seronegative for human immunodeficiency virus. ECG showed multifocal atrial tachycardia. Pulmonary function testing revealed a post bronchodilator FEV_1 of 52% with insignificant reversibility. Arterial blood gas measurement revealed a PaO_2 of 56 mm Hg and combined respiratory and metabolic acidosis. Chest radiograph revealed a cavitating infiltrate in the right lung crossing the minor fissure and a consolidation in the left lung [Figure 1]. Grams staining study of the sputum for three successive days revealed plenty of pus cells and all three samples were negative for acid fast bacillus by conventional ZN staining. Modified acid fast staining using 1% sulfuric acid as the decolorizer revealed branching acid-fast bacilli irregularly stained consistently in all three samples [Figure 2]. Sputum was

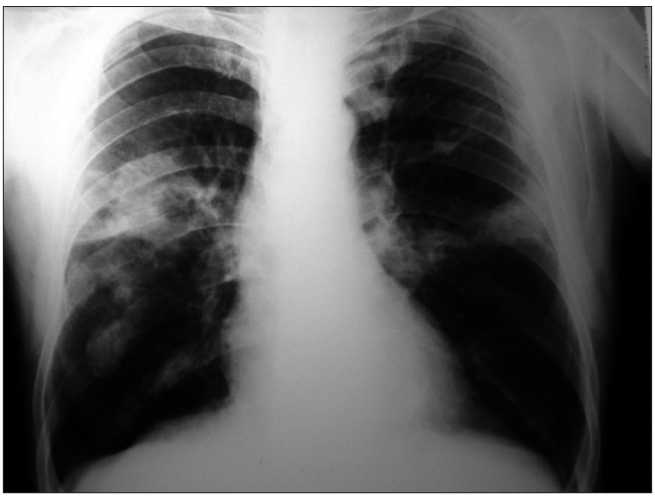

Figure 1: Chest X-ray PA view showing a consolidation in the right upper lobe with cavitation, infiltrating through the minor fissure. A patchy consolidation is also noted in the left mid zone

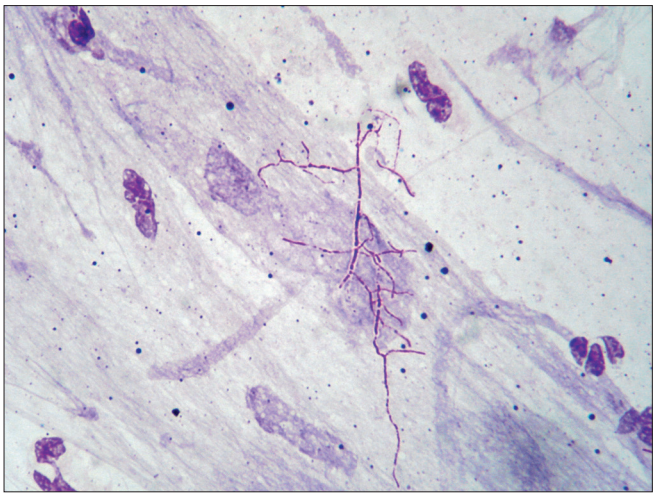

Figure 2: Weak acid staining (1%) by modified ZN staining revealed branching acid fast bacilli (Magnification, ×1000)

cultured on 5% sheep blood agar and LJ medium. On blood agar, the growth (white, dry friable colony) was seen after 72 h. On Sabourauds Dextrose agar without antibiotics medium dry, cream colored colonies were seen after 48 h.

Patient was admitted to the hospital and treated with oral cotrimoxazole (trimethoprim 20 mg/kg/day) along with treatment for his COPD and cor pulmonale. Patient significantly improved and was discharged after two weeks. Patient continued follow-up once a week until four weeks when he showed a near complete resolution of the pulmonary opacity. He tolerated the drug well and was asked to continue treatment with oral cotrimoxazole for six months, but was lost to follow-up after six weeks.

Q1. What is your diagnosis?

ANSWER

Pulmonary nocardiosis.

DISCUSSION

Nocardia, a common soil inhabitant, is an uncommon opportunistic infection of the lungs noted in cases of severe immunosuppression.[1] Pulmonary nocardiosis is uncommon in India.[1] However, with an increase in the prevalence of HIV infection, AIDS, organ transplants and use of potent immunosuppressants, we may observe an increase in the number of cases with nocardiosis in the future.[2] To have an established nocardia infection, suppression of the cellular immunity is required.[3] It mimics pulmonary tuberculosis clinically and radiologically.[4] Nocardia asteroides is responsible for about 80% of invasive disease.[5] Respiratory colonization can occur, and in a compromised host it can progress to tissue invasion and dissemination.[5] It has been observed in a patient with cirrhosis and glomerulonephritis on oral steroids.[6] A recent report indicates that nocardia can cause infection in immunocompetent hosts as well.[7] Pulmonary nocardiosis occurs because of direct inhalation of nocardia from the contaminated soil and it is rare to have person to person transmission.[2]

Tashkin *et al*.[8] have demonstrated that habitual smoking of cannabis has deleterious effects on the respiratory and immune systems, including airway inflammation and injury, dysregulation of bronchial epithelium similar to that of bronchogenic carcinoma, and COPD. Immunological effects are mainly noted in the alveolar macrophages with impaired immunostimulatory cytokine production and antimicrobial activities. The major mechanism of suppression of the immune responses is related to the effects of delta9-tetrahydrocannabinol mediated through the cannabinoid receptor on the leucocytes. Morris[9] in an autopsy study of subjects who smoked cannabis demonstrated accelerated pathological changes in the lungs in these subjects as compared to tobacco smokers that caused pulmonary scarring and COPD. A recent study observed that corticosteroid administration and presence of COPD were significantly associated with nocardioisis.[5]

The clinical manifestations of pulmonary nocardiosis are usually sub acute, although rarely it can have an acute presentation in some immunosuppressed patients. The prominent symptom is productive cough with small amounts of thick, purulent sputum. Fever, anorexia, weight loss, and malaise are common. Some patients present with dyspneoa, pleuritic pain, or hemoptysis. Disseminated disease is observed in nearly 50% of cases of pulmonary nocardiosis. Central nervous system is the most common site of disseminated disease, though skin, kidneys, bone or muscle can be involved. Rare presentations include peritonitis, endocarditis of native or prosthetic valves, bacteremia associated with infected central venous catheters and infection of sternotomy wounds.

The most common radiologic abnormality observed in the lungs is homogenous consolidation, usually multilobar, with predilection to upper lobes.[4] Cavitations are common. Nodules and mass lesions can occur.[10] Pleural extension results in effusion, empyema or pleural thickening. In a pre existing cavity, invasion of nocardia causing a 'fungal ball' appearance has been reported.[11] Broncholithiasis can occur with endobronchial nocardiosis.[12] CT scan may delineate the involvement of lungs more clearly than the chest X-ray, especially chest wall extension. There are few diseases that can infiltrate across fissures, which include malignancy, tuberculosis, actinomycosis, nocardiosis and fungal infections, and the presence of this feature on radiography should direct investigations toward these diseases.

Diagnosis in suspected cases of pulmonary nocardiosis is by examination of sputum after Gram and modified acid-fast stains. Sputum smears are often negative although sputum cultures are usually positive. However, the growth may not be apparent for 3 to 21 days. Most *nocardia* are acid-fast if a weak acid (1% sulphuric acid) is used for decolorization (e.g., in the modified Kinyoun, Ziehl-Neelsen, and Fite-Faraco methods). Bronchoscopic aspiration may be necessary to obtain a good specimen. Demonstration of the Gram-positive and acid-fast branching filaments, 1 μm wide and up to 50 μm long, in the clinical material from a patient with pulmonary lesion, should be sufficient for the diagnosis and institution of specific therapy. Culture mediums used for the diagnosis of nocardiosis are sheep blood agar, Sabouraud's dextrose agar and Lowenstein-Jensen (LJ) medium. Nocardiosis should be borne in mind especially in suspected cases of tuberculosis not responding to anti-tubercular therapy and where tubercle bacilli could not be demonstrated either in the direct smears or cultures.[13]

Cotrimoxazole is the drug of choice in nocardiosis, but the organism is also susceptible to amikacin, minocycline, imipenem, linezolid, cefotaxime, ceftriaxone, clindamycin, ampicillin and cycloserine.[14-15] The choice of antibiotics should be guided by drug sensitivity patterns wherever possible. Cotrimoxazole is usually used in the doses of 5-20 mg/kg/day of trimethoprim and 25-100 mg/kg/day of sulfamethaxazole in divided doses. In cases of resistance or hypersensitivity to cotrimoxazole, amikacin is a good alternative at a dose of 15 mg/kg/day. Minocycline is also useful and is prescribed at a dose of 100 to 200 mg twice a day.[16] Therapy must be prolonged and the usual duration of treatment is for 6-12 months to prevent the occurrence of relapses.[17] Factors related to the outcome of the treatment include disseminated disease, species of nocardia and the degree of immunosuppression and corticosteroid use.[18]

CONCLUSION

A high degree of suspicion is required when evaluating pulmonary opacity in patients with immunosuppression. It is likely that a combination of factors such as

COPD, cannabis smoking, tobacco smoking, alcohol consumption and borderline diabetes have contributed to the immunosuppression in this case. More studies are required to assess the impact of smoking regular cannabis on the respiratory and immune systems.

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