

Case Report

Pulmonary Actinomycosis With Bronchiectasis:A Rare Case Report

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Abstract: 54 year old male chronic alcoholic, diabetic patient presented with massive hemoptysis. He had cough with sputum and fever for the past one week period. Examination showed marfanoid habitus and digital clubbing. Respiratory system showed asthenic chest with findings of right lower lobe bronchiectasis. No hepatic flap was there. Chest X ray showed right lower lobe bronchiectasis with bronchopneumonia but no rib erosion or osteomyelitis were there. HRCT Thorax revealed right lower lobe bronchiectasis without rib lesions or pleural effusion. Due to massive hemoptysis and localised disease patient undergone right lower lobectomy. Lobectomy specimen on histopathology revealed actinomycosis with sulphur granules and bronchiectasis. Postoperatively patient received intravenous penicillin against complications in lung actinomycosis including bronchopleural fistula and relapse.

Keywords: Pulmonary actinomycosis, bronchiectasis, lobectomy.

INTRODUCTION

Pulmonary actinomycosis is a difficult condition to diagnose. Even among experienced physicians, sometimes despite pointers to the disease, delayed diagnosis or misdiagnosis as tuberculosis, lung abscess or lung cancer is common [1]. An increased awareness of the infection may expedite diagnosis and prevent undesirable complications, including unwarranted surgery. A higher incidence of pulmonary actinomycosis has also been reported in patients with underlying respiratory disorders such as bronchiectasis, emphysema, chronic bronchitis and in alcoholics[2,3]. The pulmonary form of actinomycosis constitutes 15% of the total burden of disease, although estimates of up to 50% have been reported.[1,4,5,6]

CASE REPORT

54 Year old male diabetic, chronic alcoholic with hepatic cirrhosis presented with massive hemoptysis . He had cough with yellowish expectoration and right sided chest pain and fever for past one week. He was on treatment for type 2 diabetes mellitus with oral hypoglycemic agents. Patient also had loss of appetite. On examination patient had marfanoid habitus and he was ill nourished with finger clubbing. Tachypnoea and tachycardia was there with blood pressure of 124/ 76 mm Hg in right arm in supine position. Respiratory system examination revealed findings of right lower lobe bronchiectasis. Abdomen examination showed no hepatomegaly, no splenomegaly or ascites but with normal bowel sounds . On cardiovascular system examination JVP was not found elevated; heart sounds normal and there was no murmur. Laboratory evaluation revealed hemoglobin of 11.6 gm per dL with total WBC count of 8200 cells per cmm . Differential count showed 81% polymorphs and 11% lymphocytes with ESR 72 mm in the first

hour. Platelets count was 106000 per cmm. PCV was 34% . LFT showed bilirubin of 1 mg per dL, total protein 5.4 gm per dL with albumin 2.3 gm per dL and globulin 3.1 gm per dL.

ALT was 49 IU/L with AST of 61 IU/L and alkaline phosphatase of 109 IU/L . Sodium was 136 mmol/L and potassium was 5.1 mmol/L .Bleeding time was 2 minutes 45 seconds with clotting time of 10 minutes. Pulmonary function test report was mild obstruction with restriction. Echocardiography revealed good LV function. Chest X- ray showed right lower lobe bronchiectasis . USS abdomen report was cirrhosis liver with portal hypertension and mild splenomegaly. HRCT thorax showed right lower lobe bronchiectasis with surrounding consolidation and no lymphadenopathy nor pleural effusion. HIV & HBsAg were negative. Lobectomy specimen histopathology examination showed features suggestive of pulmonary actinomycosis with sulphur granules and bronchiectasis surrounding lung showing pulmonary oedema, hemorrhage and hemosiderosis. Sputum AFB was negative. Sputum gram stain result was mixed pharyngeal flora. Sputum culture was sterile. Due to massive hemoptysis and localised nature of bronchiectasis thorocotomy done with right lower lobectomy. Post operatively patient received parenteral penicillin for 6 weeks followed by 6 months oral penicillin to prevent complications including the relapse and broncho pleural fistula. Follow up period was uneventful.

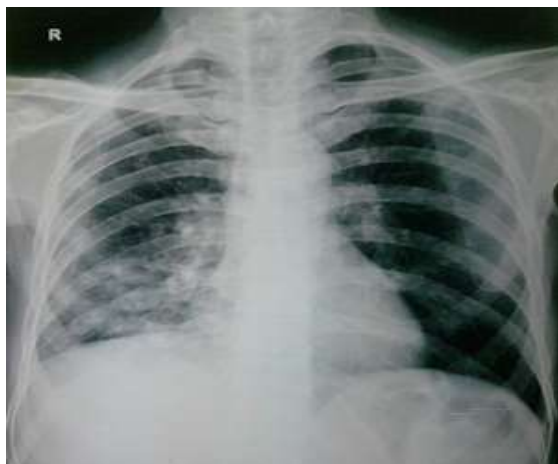


Figure 1: CXR PA showing right lower lobe bronchiectasis

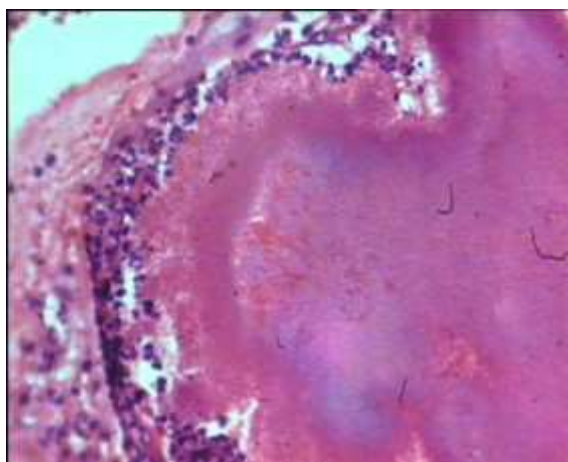


Figure 2: H&E stain of lobectomy specimen showing the ray like arrangement and sulphur granules

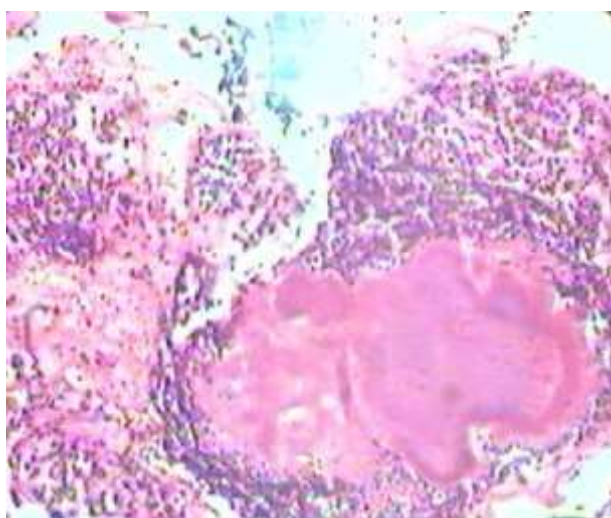


Figure 3 : H&E staining showing lung actinomycosis

DISCUSSION

Actinomyces spp. are higher prokaryotic bacteria belonging to the family Actinomyceataceae. When they were first described in the early 19th Century, they were misclassified as fungi [7]. The word "actinomycosis" was derived from the Greek terms *aktino*, which refers to the radiating appearance of a sulphur granule, and *mykos*, which labels the condition a mycotic disease. Pulmonary actinomycosis is a difficult condition to diagnose. Even among experienced physicians sometimes despite pointers to the disease delayed diagnosis or misdiagnosis as tuberculosis, lung abscess or lung cancer is common. An increased awareness of the infection may expedite diagnosis and prevent undesirable complications, including unwarranted surgery, in patients under investigation for persistent pulmonary shadowing. The pulmonary form of actinomycosis constitutes 15% of the total burden of disease, although estimates of up to 50% have been reported. Pulmonary actinomycosis is having peak incidence in the 4th and 5th decades [3,8]. The incidence of infection is two to four times greater in males compared with females [8]. A higher incidence of pulmonary actinomycosis has also been reported in patients with underlying respiratory disorders, such as emphysema, chronic bronchitis and bronchiectasis, and in alcoholics. Members of the genus *Actinomyces* are Gram-positive, non spore-forming, predominantly anaerobic prokaryotic bacteria belonging to the family Actinomyceataceae. They are bacteria rather than fungi.

Six of these are thought to be pathogenic in humans, including *A. israelii*, *A. naeslundii*, *A. odontolyticus*, *A. viscosus*, *A. meyeri* and *A. gerencseriae*. *A. israelii* is the organism most commonly incriminated in human disease. Culture requires brain/heart enriched agar and the organisms grow best at a temperature of 37°C in an atmosphere of 6–10% ambient carbon dioxide. Characteristically, colonies of *Actinomyces* appear as "molar-tooth" or "bread-crumbs" colonies in broth media after 3–7 days of incubation. Most of the literature classifies the tissue response as granulomatous or "granulomatoid-like", although giant cells and granulomata are rarely seen [21]. Sulphur granules are the pathological hallmark of the disease. These are round or oval basophilic masses with a radiating arrangement of eosinophilic clubs on the surface; they sometimes can be seen even with a magnifying glass. The name "sulphur granule" has its origin in the small nodules resembling elemental sulphur that were commonly used in pharmaceuticals in the 19th century [9]. Pulmonary actinomycosis probably results from aspiration of oropharyngeal or gastrointestinal secretions into the respiratory tract [8]. Poor oral hygiene and associated dental disease may increase the risk^[1]. Support for aspiration as a risk factor comes from reports of a higher prevalence of alcoholism in patients with the pulmonary form of the disease and from the basilar predominance of the disease radiologically [10].

Previous presentation of pulmonary actinomycosis with prominent chest pain and cutaneous fistulas discharging sulphur granules has changed with time in line with the decrease in the disease prevalence [11,12]. The commonest presentation is probably now as a shadow on a chest radiograph. Marked weight loss, malaise and high fever may be more suggestive of disseminated disease [10,13]. Typical respiratory symptoms of patients with thoracic actinomycosis are cough in 84%, sputum in 74 %, chest pain in 68 %, dyspnoea in 47 %, haemoptysis in 31 %, localised chest-wall swelling in 10%. Systemic symptoms are weight loss(53%) malaise (42%),night sweats (32%),fever (21%) [14].

Physical signs are equally nonspecific, except in advanced, untreated disease, when sinuses and fistulae may then give the diagnosis away. The findings are occasionally those of the associated complications, such as pleural effusion or empyema. Plain chest radiograph findings in actinomycosis are nonspecific. A nonsegmental pneumonia, usually in the lower zones, tends to occur peripherally crossing fissures. The disease usually shows a peripheral and lower lobe predominance, probably reflecting the role of aspiration in its pathogenesis. The CT is probably more helpful than the plain radiograph, particularly if performed with a bone window display, which gives a better delineation of minimal bony change, such as early rib erosion and osteomyelitis. Fiberoptic bronchoscopy is usually not diagnostic in pulmonary actinomycosis unless there is clear endobronchial disease on which biopsy can be performed [15].

Traditionally, excisional biopsy was the definitive diagnostic procedure [16]. In general, an attempt at establishing diagnosis by percutaneous biopsy with fine needle aspiration or core biopsy is now made before "blind" thoracotomy[17]. The rationale for the use of penicillin in actinomycosis is based more on extensive successful clinical experience over the last 50 yrs than on randomised control trials [1] . The main principle of treatment is the use of high-dose intravenous penicillin for a long duration of treatment. Although treatment has to be tailored to the individual, generally 18–24 million units of penicillin per day are given for 2–6 weeks followed by oral therapy with penicillin V (or amoxicillin) for 6–12 months. In general, the thoracic form appears to require longer treatment courses compared to the other commoner forms . Tetracyclines are the alternative especially for penicillin-allergic patients. In pregnant, penicillin-sensitive patients, erythromycin is a safe alternative. Commonly used antibiotics in the treatment of actinomycosis are a) efficacious drugs like penicillin (MIC range ≤ 0.25 – 0.5 mg per ml), tetracycline (doxycycline) (MIC range 0.5 – 0.8 mg per ml), erythromycin (MIC range ≤ 0.25 – 1 mg per ml) clindamycin (MIC ≤ 0.25 – 0.5 mg per ml) and b) drugs with probable efficacy which includes imipenem (MIC

0.125 mg per ml,ceftriaxone (MIC <0.06 – 2 mg per ml).Presumably, the avascularity and induration of infected areas account for the need for prolonged treatment and undoubtedly longer course minimise the risk of relapses, a clinical hallmark of the infection. Response to treatment should be monitored radiologically with plain radiographs and/or CT. Diminution in the shadowing on a chest radiograph is expected within 4 weeks. Evidence shows that this standard treatment approach applies to people who are immunocompromised for one reason or another [18]. When surgery has been the initial treatment, even if histology suggests complete resection, it still needs to be followed by prolonged antibiotic therapy, as surgery alone is usually not curative [19,20]. Inadequate antibiotic therapy postoperatively may result in complications such as bronchopleural fistulas and empyema.

The prognosis of the pulmonary form of actinomycosis may be less favourable compared with the other commoner forms, such as cervicofacial and abdominal disease^[10] . This may be related to the greater incidence of disseminated disease in the thoracic form and may also be a reflection of late diagnosis in this condition. However, when the infection is recognised early and proper treatment is given, the condition has an excellent prognosis with a very low mortality. Every respiratory physician should be familiar with this important differential in any patient with long-standing pulmonary infiltrates to prevent unnecessary morbidity or even unwarranted surgery.

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