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A Rare Presentation of Pulmonary Actinomycosis Mimicking Lung Metastasis

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Abstract:
Pulmonary actinomycosis is a rare, chronic granulomatous infection caused by Actinomyces *species. Its clinical and radiological features often mimic lung malignancy, leading to diagnostic challenges.*
Keywords: *Pulmonary actinomycosis, lung nodules, Actinomyces, lung cancer mimicry*

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I. Introduction:

Actinomycosis is a rare, chronic granulomatous infection caused by the bacterial species *Actinomyces*, wich normally inhabits the human oral cavity , digestive tract and genital tract [1] .

Actinomyces are filamentous gram-positive facultative anaerobic bacilli. More than 30 Actinomyces species have been identified, and the majority of infections are caused by Actinomyces israelii and Actinomyces genencseriae [2].

There are four primary subtypes of actinomycosis infection: Cervicofacial (60 %), abdominal (20 %), pulmonary (15 %) and pelvic (5 %). This review focuses on the pathology of pulmonary actinomycosis [1] [3] . This review focuses on the pathology of pulmonary actinomycosis.

Pulmonary actinomycosis poses a rare yet significant diagnostic challenge due to its varied presentation, ranging from resembling bronchogenic carcinoma to mimicking tuberculosis-like pneumonitis [4] . Occasionally, it manifests with either massive or recurrent hemoptysis [5] [6].

We present a case of pulmonary actinomycosis mimicking lung cancer, initially presenting with dyspnea and cough with radiological deterioration but ultimately responded to appropriate antibiotic therapy.

Case presentation:

A 30-year-old female patient, with no pathological history, was admitted to the department of pulmonary medicine of our hospital with a 5-month history of progressively worsening dyspnea, becoming grade 4 of mMRC, associated with cough and purulent foul-smelling sputum, 2 episodes of mild hemoptysis, and weight loss estimated at 13 kg, night sweats, and anorexia.

Clinical examination revealed tachypnea (26 beats/min), fever (38–39 °C), and tachycardia (102 beats/min). Blood pressure (110/80 mmHg) and oxygen saturation (99%) were measured while breathing ambient air. Oral examination revealed poor hygiene, and physical examination revealed left condensation.

Laboratory testing revealed a white blood cell count of 13,500/mm3 with predominantly polynuclear neutrophils (12,000 elements/mm3), a hemoglobin level of 6.3 g/dl, a platelet count of 296×109/l, a C-reactive protein level of 170.00 mmol/l, and hyponatremia at 126 mEq/l. The results of the Human immunodeficiency virus (HIV) serology test were negative. Sputum cultures tested negative for bacteria and mycobacteria.
Quantification of the mycobacterium tuberculosis (TB) DNA yielded a negative test result.

The chest CT scan showed a left basal consolidation with a Left pleural effusion (figure 1), for which the patient underwent a first CT-guided biopsy , which yielded negative for tuberculosis and malignant cells on cytology but revealed chronic organizing inflammation.

The patient had a cough with hemoptysis and evidence of inflammation, which suggested the presence of lung infection, received intravenous antibiotic therapy Cephalosporin + Metronidazole) but the cough and other symptoms persisted.

Given the patient's increasing clinical condition, a second chest CT scan was performed, revealing radiological deterioration with the appearance of multiple bilateral pulmonary nodules, The largest ones are located in the right apical area and measure 13*22 mm, and the left postero-basal area, measuring 28*30 mm (figure 2), resembling a "lacher de ballons" pattern suggestive of pulmonary metastases. Therefore, a second CT-guided biopsy was performed, revealing the presence of actinomycotic filaments.

Thus, a final diagnosis of pulmonary actinomycosis was made

Medical treatment was initiated with 1 g of amoxicillin-clavulanic acid intravenously every six hours for two weeks, with a switch to oral administration for 1 month, followed by oral amoxicillin at a dosage of 2 g/day, with good clinical improvement. It was recommended that the patient be treated with antibiotics for an additional three months after discharge from the hospital.

After two weeks of antibiotic therapy, the cough significantly improved, and the patient's temperature returned to normal. Therefore, we repeated the CT scan and found radiographic resolution by the remarkable reduction in the size of the nodules, 7 mm for the right apical nodule, and 8 mm for the left postero-basal (figure 3).

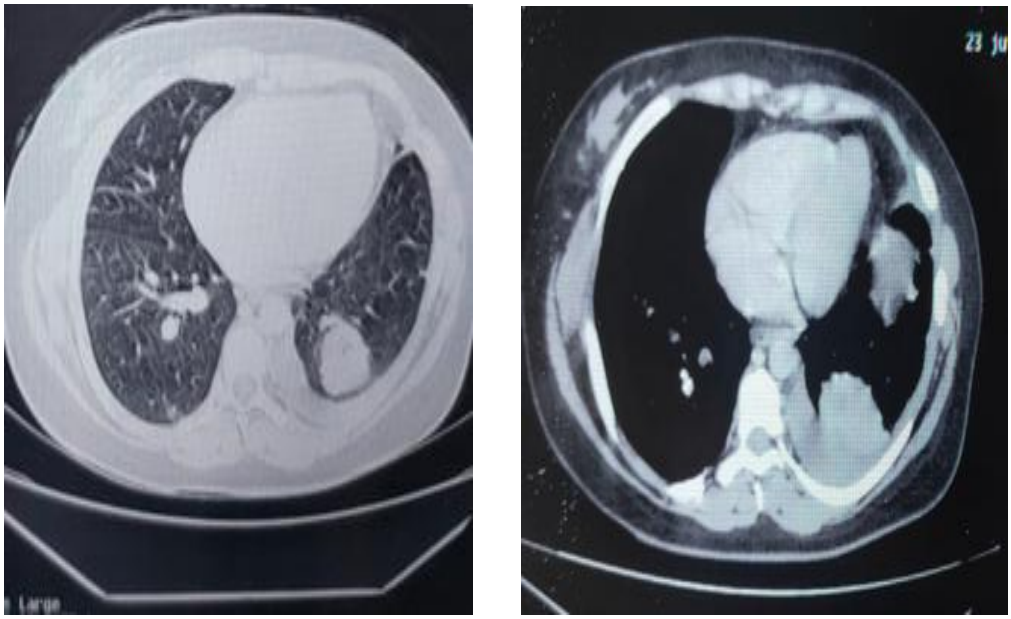
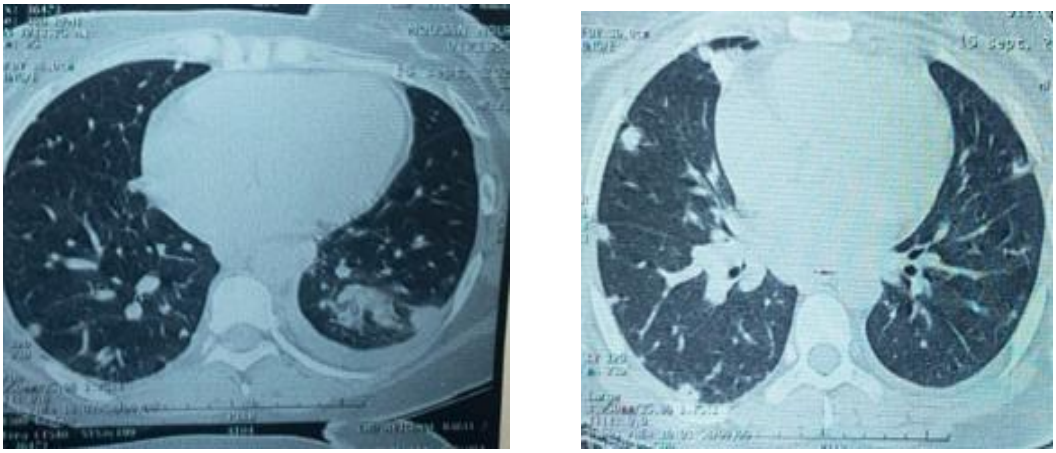


Figure 1 : CT scan revealing a left basal consolidation with a Left pleural effusion



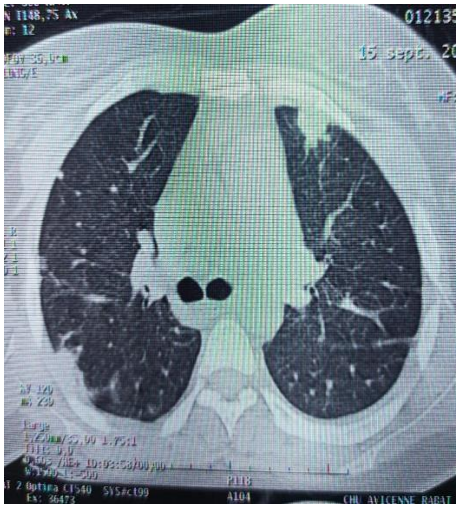
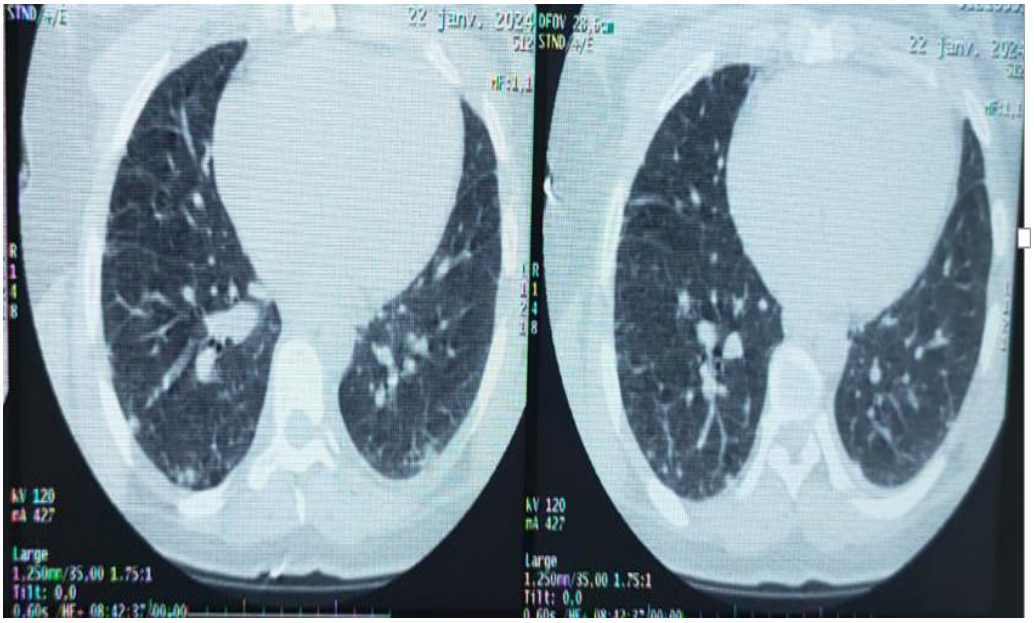


Figure 2: Second CT scan showing the appearance of multiple bilateral nodules pulmonary nodules The largest ones are located in the right apical area and measure 13*22mm , and the left postero-basal area , measuring 28*30 mm



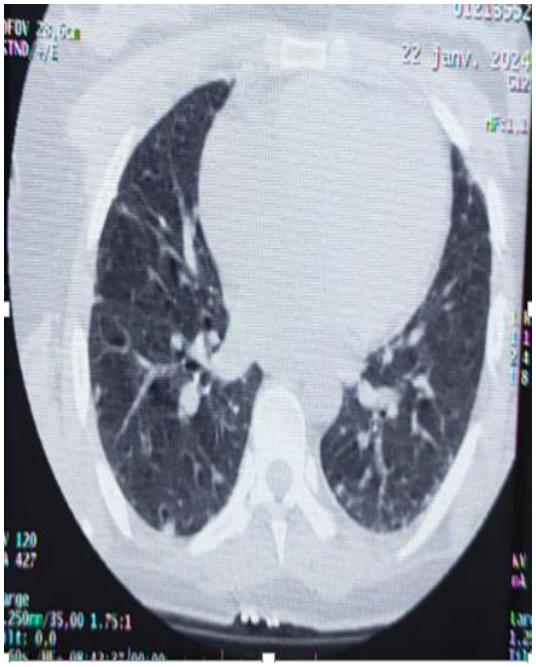


Figure 3 : CT scan 1 month after Antimicrobial therapy showing the reduction in the size of the nodules , 7 mm for the right apical nodule , and 8 mm for the left postero-basal

II. Discussion:

Actinomyces species, founds as commensal bacteria in our gastrointestinal and urogenital polymicrobial flora[4], are the cause of actinomycosis, a rare, and persistent granulomatous, gram-positive microaerophilic anaerobic bacterial infection. However, certain species become pathogenic when mucosal barriers break down and they are introduced into new tissue [1] [2].

It commonly affects young- to middle-aged population with a male predisposition [7] [8] , It is characterized by slow invasion of body tissue and incidence has decreased because of advent of antimicrobial agents and better oral hygiene [9]. Our patient was a young woman aged 33 suffering from pulmonary Actinomycosis.

The clinical forms of Actinomycosis involve cervicofacial, thoracic, abdominal, and pelvic regions. Pulmonary Actinomycosis is the third most common type of *Actinomycosis (15%).*

The three main causes of pulmonary actinomycosis include inhaling particles from the oropharyngeal region, aspirating gastrointestinal secretions, and secondary spreading from the cervicofacial region or other distant sites. [3] [5] [10]. Poor oral hygiene, gingivitis, dental illness, recent dental surgery, trauma, aspiration, compromised immune system, and prior local or distant actinomycosis infections are risk factors that might lead to the development of pulmonary actinomycosis [4] [5] [11]. Our patient had a very poor oral hygiene and dental disease with severe caries.

small case series have reported a higher incidence of pulmonary actinomycosis in patients with pre-existing chronic respiratory conditions, including chronic bronchitis, emphysema, and bronchiectasis [12] [13].

Even with regard to immunodeficiency due to human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome(AIDS), only a few cases of actinomycosis infection have been reported [14].

Clinical symptomatology is nonspecific, making its diagnosis difficult, often leading to misdiagnosis of malignancy rather than an infective disease [2].In our patient as well, the initial CT scan made us think for malignancy.

As the disease progresses, it causes dense consolidation and abscesses. The patient may present with cough, sputum production, hemoptysis, chest pain, dyspnea, fever, weight loss, malaise, night sweats and localised chest wall swelling [15] [16][17] , non-specific findings include anemia, leukocytosis, raised C-reactive protein (CRP), and raised erythrocyte sedimentation rate (ESR).

Our patient had suffered from cough, sputum production, hemoptysis, dyspnea, fever, an important weight loss, and night sweats with an elevated white blood cell count and CRP inflammation.

Examination findings are usually nonspecific with signs indicating a chest infection, such as dullness to percussion and course crepitus on auscultation [15][17]. The disease usually predominates in peripheral and lower lung lobes, probably reflecting the role of aspiration in its pathogenesis [15][17].

Clinical suspicion of pulmonary actinomycosis should be raised in the presence of cervicofacial infection presenting with a nodular jaw, evidence of chest wall sinus tracts, yellow sulphur granules and refractory infection to short-term antibiotics.

It is a challenge to distinguish pulmonary actinomycosis from benign infections or malignancy on imaging, as findings are generally non-specific. Therefore, imaging cannot diagnose pulmonary actinomycosis, but it can assess the location and extent of the disease for biopsy and assess response to treatment.

Nodules or densities can be seen on the chest x-ray, which are often difficult to distinguish from non-segmental pneumonia [5]. These consolidations generally occur in the peripheral lower lobes and can be seen to cross fissures [1] [18] [19].

Major findings reported on Computed Tomography (CT) of the chest include consolidation, mediastinal or hilar lymphadenopathy, atelectasis, cavitation, ground-glass opacity, and infrequently pleural effusion [19] [20] [21]. Pleural thickening and pleural effusion occur in 15–50% of cases, while empyema is associated with approximately 9–15% of cases of pulmonary actinomycosis [22]. In rare cases, a CT chest will find pericardial effusion secondary to pericardial involvement [21].

The usefulness of bronchoscopy is usually limited to endobronchial actinomycosis, where a biopsy can provide a histopathological diagnosis [23]. A bronchoalveolar lavage can be performed and may show non-acid-fast, gram-positive, filamentous bacteria in the culture which points to actinomycosis [24].

Eventually, The gold standard for diagnosing pulmonary actinomycosis is histological examination and bacterial culture of a lung biopsy, obtained by percutaneous biopsy guided by CT scan or by open surgical resection [2] [24] . Isolation and identification of actinomyces in anaerobic culture require 2-3 weeks.

The aim of both biopsy and surgical resection is to ascertain the diagnosis while preserving the remaining lung tissue [24]. On histology, the specimen should reveal inflammatory cellular infiltrates and colonies of filamentous microorganisms with sulfur granules, surrounded by necrotic tissue and inflammatory cells.

Treatment of pulmonary actinomycosis mainly consists of long term use of antimicrobial beta-lactamase agents use over 3–12 months and in some cases requires surgical intervention [2]. The recommended duration and method of treatment for pulmonary actinomycosis depends on the severity of the disease. Mild to moderate disease is classified as disease without local invasion, bone involvement, fistulas/sinuses, and abscesses or
necrotic tissue. Serious disease includes invasive disease, including large purulent disease (dense necrotic abscess,pleural effusion, empyema), fistula/sinus, bone involvement, and infection comp licated by massive hemoptysis.

Indications for surgical intervention include severe and invasive disease, failure of antibiotics therapy, infections in critical spaces, and massive refractory hemoptysis .

Since our patient shows no signs of severity or local invasion, our discussion will focus on mild to moderate disease.

Antimicrobial treatment for mild to moderate disease includes oral phenoxymethylpenicillin (2–4 g per day divided into four daily doses) or amoxicillin (1.5–3 g per day divided into three/four daily dosing). If co-pathogenicity is suspected, oral amoxicillin-clavulanic acid (875–125 mg twice daily) is indicated. Duration should continue for at least one to two months after the resolution of symptoms. This generally amounts to two to six months for treatment of mild disease [5] [25] [26].

When choosing antibiotics, it is important to recognize that no randomized control studies have evaluated antimicrobial regimens for actinomycosis. Alternative antibiotics for patients with penicillin allergy include ceftriaxone, doxycycline, macrolides, and carbapenems . Ineffective agents include aminoglycosides, metronidazole, aztreonam, trimethoprim-sulfamethoxazole, cephalexin, ceftazidime and antifungals [26].

Follow-up imaging is suggested to show the resolution of the disease and ensure no underlying malignancy is present. This can be executed via serial chest x-rays or CT to ensure resolution [5]. Clinical improvement in radiology is generally noted within four weeks of starting antimicrobial therapy [5], [27]. In the case of no consolidation or mass improvement on radiological imaging, a co-existing local malignancy must be investigated. No research indicates the time frame for follow-up and surveillance of recurrent infections. Some studies have described follow-up at three months, six months and one year to assess complete resolution of the infection.

Complications of undiagnosed pulmonary actinomycosis include empyema, endocarditis, pericardial effusion, empyema, and sepsis. These complications are infrequent but present with rapidly progressive dyspnea, oxygen desaturations and hemodynamic collapse [1] [2]. Severe disease requiring surgical intervention has a higher mortality rate than mild pulmonary actinomycosis. With early diagnosis and treatment, patients usually

recover with a 98% success rate [1] [3] [4]. The prognosis of pulmonary actinomycosis is less favorable than cervicofacial, abdominal and pelvic disease due to the rapidly progressive, higher morbidity and mortality-associated complications such as empyema, pericardial effusion, and mediastinal invasions.

III. Conclusion

Pulmonary actinomycosis is a rare with a nonspecific clinical presentation mimicking lung cancer. diagnosis of pulmonary actinomycosis requires a combination of clinical and radiological features with microbiological culture of specimens with identification of Actinomyces species with sulfur granules. Even if actinomycosis is diagnosed, it is important to exclude potential coexisting lung malignancy. This can only be confirmed following symptomatic and radiological resolution with antibiotics regimen, or with clinical improvement on postoperative follow-up after surgical resection of the lung with histopathological substantiation.

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