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Co-Infection of Pulmonary Actinomycosis and Pulmonary Tuberculosis: A Case Report

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ABSTRACT

Pulmonary actinomycosis is a rare infection caused by bacteria of the Actinomycetaceae family. Pulmonary actinomycosis itself is uncommon, and its co-occurrence with tuberculosis is even rarer. We report a case of a 61-year-old female patient diagnosed with concurrent pulmonary actinomycosis and pulmonary tuberculosis. High-resolution computed tomography, histopathological examination, and GeneXpert testing of the bronchoalveolar lavage sample allowed us to confirm the diagnosis of co-infection. To our knowledge, this is the first reported case of concomitant pulmonary actinomycosis and pulmonary tuberculosis in Mongolia.

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List of Abbreviations

BAL: Bronchoalveolar Lavage
HRCT: High Resolution Computed Tomography
HRZE: Isoniazid, Rifampicin, Pyrazinamide, Ethambutol
MTB: *Mycobacterium Tuberculosis*
PA: Posterior-Anterior
PET/CT: Positron Emission Tomography and Computed Tomography
PTB: Pulmonary Tuberculosis

Introduction

Actinomycosis is an infectious disease caused by gram-positive anaerobic bacteria of the Actinomycetaceae family (genus Actinomyces), also known as "ray fungi." It is a rare, chronic, slowly progressive granulomatous disease [1,2]. *Actinomyces species* are found in soil and in the microflora of healthy people, particularly as part of the microflora of the oral cavity, colon, and upper respiratory tract [3, 4]. Under conditions of impaired immunity, these organisms can cause the infectious disease actinomycosis. Pulmonary actinomycosis is a rare condition, with an estimated incidence of approximately 1 case per 3,000,000 people per year [5]. Risk factors for pulmonary actinomycosis include aspiration, underlying lung disease, poor oropharyngeal hygiene, and weakened immunity [6]. Clinical manifestations may include cough, sputum production, hemoptysis, pulmonary hemorrhage, chest pain, dyspnea, fever, weight loss, malaise, and night sweats [6,7]. The initial correct diagnosis rate of pulmonary actinomycosis is 4%, and it is commonly misdiagnosed as lung cancer [2,7-9]. Because its clinical presentation is often non-specific, pulmonary actinomycosis remains difficult to distinguish from other chronic or infectious pulmonary diseases, making diagnosis challenging [7,9]. Increasing knowledge and understanding of this infection may allow earlier diagnosis when a long-lasting, non-resolving pulmonary opacity is found, and may therefore help prevent unnecessary surgery. We present a rare case of coexisting pulmonary tuberculosis with pulmonary actinomycosis in an immunocompetent adult patient.

Case Presentation

The patient is a 61-year-old woman who complained of a severe paroxysmal cough, thick green sputum, and high-grade fever reaching 39.0-40.0°C, and pain on the right side of her chest. The cough had started 3–4 months earlier, and she had previously consulted a family physician and a pulmonologist and had received medications; however, she did not know the name of the medication she had taken. Over the past 4 days, her symptoms worsened with increased cough and the onset of fever, prompting her to present to the emergency department of Brilliant Hospital, Ulaanbaatar, Mongolia, where she was subsequently admitted. Her past medical history was significant for type 2 diabetes mellitus diagnosed in 2005, for which she has been taking Galvus Met at a dose of

50/1000 mg daily. She was diagnosed with arterial hypertension in 2006, but has not been on regular antihypertensive therapy. There is no significant family history of hereditary diseases. She does not consume alcohol or tobacco.

Physical examination revealed diminished breath sounds with crackles in both lungs. Heart sounds were clear with regular rhythm. Capillary refill time was 2 seconds. Blood pressure was 115/89 mmHg, heart rate 103 beats per minute, respiratory rate 18 breaths per minute, and oxygen saturation (SpO_2) was 95% on room air.

Laboratory investigations at the time of admission showed leukocytosis of $28x10^9$/L (normal range: 4-$10x10^9$/L), neutrophilia of $26.9x10^9$/L (normal range: 2-$7x10^9$/L), and lymphopenia of $0.65x10^9$/L (normal range: 0.8-$4x10^9$/L), elevated blood glucose of 25.9 mmol/L (normal range: 4-6 mmol/L), impaired renal function with serum creatinine of 119.7 mmol/L (normal range: 59-104 mmol/L), urea of 9.6 mmol/L (normal range: 0-8.3 mmol/L), and hyponatremia of 125 mmol/L (normal range: 125-155 mmol/L).

Sputum smear microscopy was negative for *Mycobacterium tuberculosis* (MTB). GeneXpert /MTB assay of the bronchoalveolar lavage sample was positive, and *Mycobacterium tuberculosis* was sensitive to rifampicin. Sputum culture was positive for *Staphylococcus aureus*.

Based on the abnormalities observed on chest X-ray (Figure 1) and high-resolution computed tomography (Figure 2), bronchoscopy was performed (Figure 3), and a biopsy was taken. Histopathological examination confirmed the diagnosis of actinomycosis (Figure 4), which demonstrated a dense fibro-inflammatory lesion containing a large basophilic bacterial colony with radiating filamentous architecture (sulfur granule), surrounded by chronic inflammatory cells and fibrosis. The morphological appearance was highly suggestive of pulmonary actinomycosis. No evidence of malignant cells was identified in the examined section.

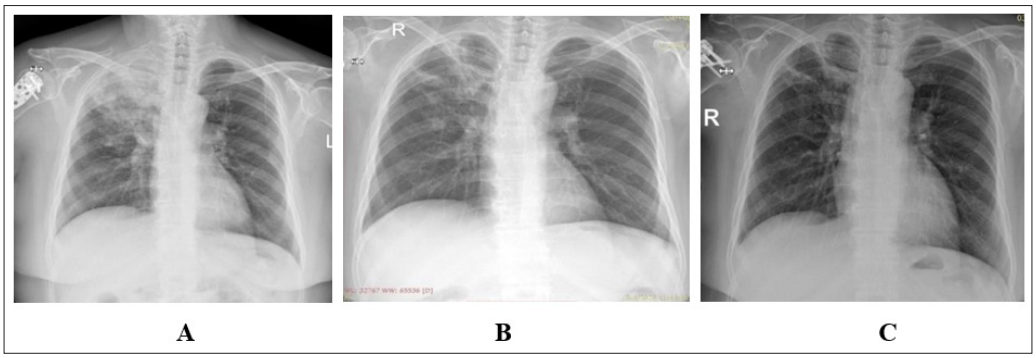

Figure 1: PA Chest Radiograph. (A) Base radiograph showing patchy, air-space dense consolidation in the right upper lobe; (B) One month and (C) Two months after antibiotic therapy, demonstrating resolution of the consolidation with residual parenchymal scarring evident.

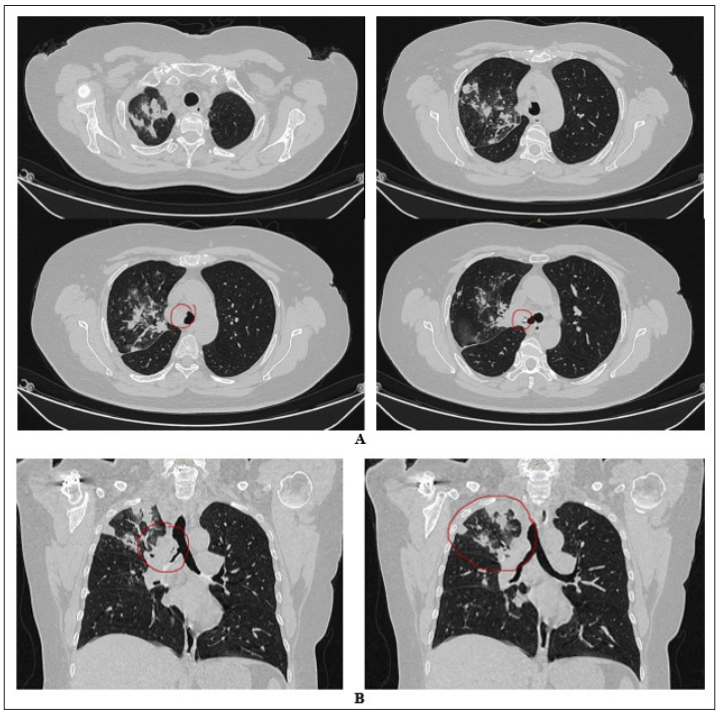

Figure 2: HRCT of the Chest. (A) Axial lung-window image and (B) Coronal images reveal poorly defined, irregularly marginated pulmonary nodules and a poorly defined subpleural mass with adjacent pleural thickening in the right upper lobe. There are surrounding areas of patchy ground-glass attenuation, interlobular septal thickening, and peripheral bronchiectasis.

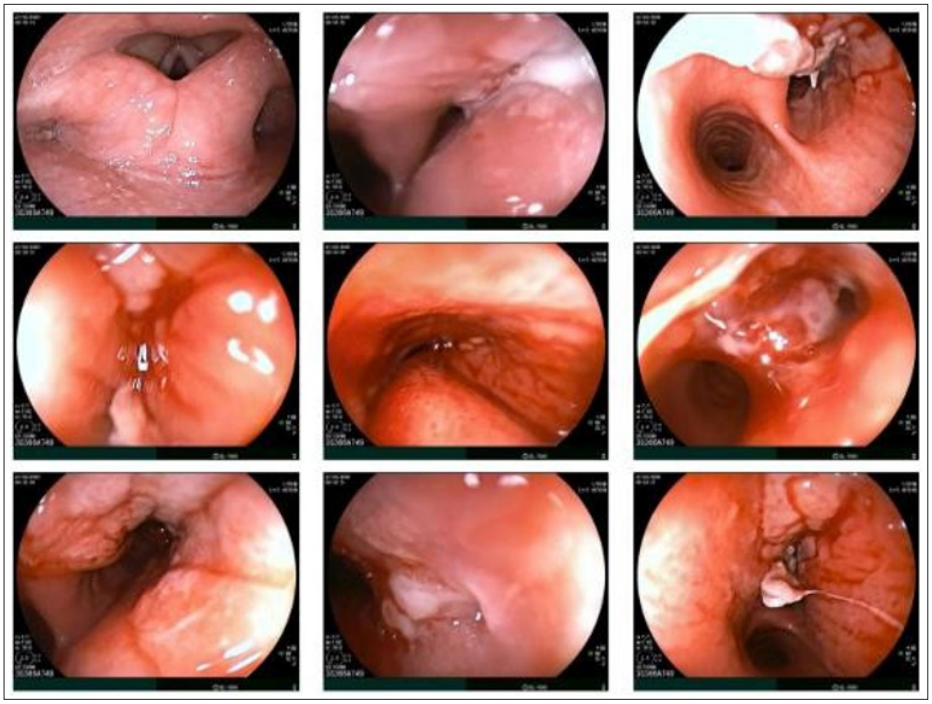

Figure 3: Bronchoscopy revealed an ulcerative, excavated mass-like lesion involving the lower trachea and right main bronchus with thick secretions. Biopsy specimens were obtained.

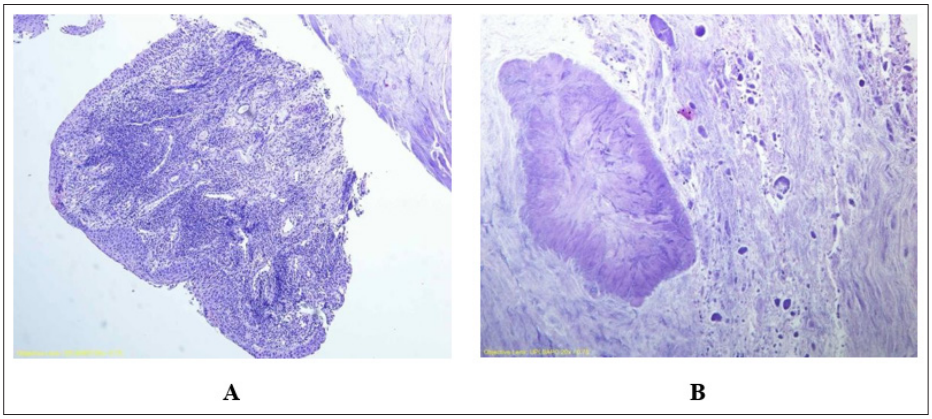

Figure 4: Histopathological Examination of the Endobronchial Biopsy Specimen. Hematoxylin and eosin (H&E)-stained sections of biopsy specimens obtained from the trachea and right upper lobe bronchus (original magnification x100), showing (A) dense chronic lymphoplasmacytic inflammation with fibrosis; (B) the characteristic sulfur granule (Actinomyces colony) with surrounding chronic inflammatory reaction, consistent with pulmonary actinomycosis.

The histopathological findings were consistent with pulmonary actinomycosis, while positive microbiological testing for MBT confirmed the coexistence of both infections.

Treatment

The patient is receiving anti-tuberculosis therapy according to the 2HRZE/4HR regimen (H - isoniazid, R - rifampicin, Z - pyrazinamide, E - ethambutol). HRZE is administered at a dose adjusted to body weight, with a total of four tablets daily, along with hepatoprotective agents and pyridoxine. At the same time, penicillin G treatment, followed by amoxicillin, was continued.

Outcome and Follow-up

As the treatment was effective and the patient's condition improved, she was discharged from the hospital on April 29, 2026, to continue treatment as an outpatient under the supervision of the tuberculosis dispensary.

Discussion

This case report describes a rare case of concomitant pulmonary actinomycosis and PTB. The diagnosis was confirmed by histopathological examination of specimens obtained via bronchoscopic endobronchial biopsy, together with a positive GeneXpert assay performed on the bronchoalveolar lavage (BAL) sample. It aims to compare the clinical features, diagnostic process, CT imaging, and therapeutic management of this case with those reported in previously published studies.

PTB is one of the leading infectious causes of morbidity and mortality worldwide, particularly in low-and middle-income countries. The estimated tuberculosis incidence in Mongolia was about 3.4 times higher than both the global and Western Pacific regional averages [10].

In contrast, pulmonary actinomycosis is an uncommon chronic bacterial infection caused by anaerobic or microaerophilic Gram-positive filamentous bacteria of the genus Actinomyces, most commonly A. israelii. However, other species such as A. odontolyticus, A. meyeri, A. naeslundii, and A. viscosus may also be implicated [4]. Actinomycosis is a chronic infectious disease that clinically and histopathologically resembles a fungal infection; however, it is actually caused by Gram-positive, branching filamentous bacteria. Because of their morphological characteristics, indolent clinical course, chronic inflammatory response, and propensity for deep tissue invasion, these organisms have been described as "bacteria that masquerade as fungi" [2].

Actinomycosis has become increasingly uncommon following the widespread use of antibiotics and improvements in oral hygiene [11]. Pulmonary actinomycosis accounts for approximately 15-20% of all actinomycosis cases and less than 0.2% of pulmonary infections [12-15]. Although each disease has been extensively studied individually, the coexistence of PTB and pulmonary actinomycosis is extremely rare. Only a limited number of individual case reports or case series indicate the condition is probably underdiagnosed rather than truly rare [12]. The true incidence is unknown [5].

Pulmonary actinomycosis usually develops following aspiration of oropharyngeal secretions containing *Actinomyces* species. *Actinomyces* are commensal bacteria that constitute part of the normal flora of the oral cavity, pharynx, gastrointestinal tract, and female genital tract. Disruption of the mucosal barrier allows these organisms to penetrate the underlying tissue, resulting in infection [13,11].

Actinomycosis can occur not only in immunocompromised individuals but also in immunocompetent hosts [6]. Aspiration of oral secretions, disruption of the mucosal barrier, tissue hypoxia, and a chronic inflammatory microenvironment are recognized risk factors that increase susceptibility to actinomycosis [12].

A milestone in understanding actinomycosis pathogenesis was the work of Holm, who demonstrated that human actinomycosis is rarely caused by *Actinomyces* alone [13]. Instead, infection develops through synergistic interactions between *Actinomyces* and numerous accompanying microorganisms ("companion microbes"), including anaerobic and facultative bacteria inhabiting the oral cavity. Holm proposed that these accompanying bacteria reduce tissue oxygen tension, impair host defense mechanisms, and facilitate invasion of *Actinomyces* into deeper tissues. This concept fundamentally changed the understanding of actinomycosis from a single-pathogen disease to a polymicrobial infection and remains widely accepted today.

Common co-pathogens associated with pulmonary actinomycosis include *Actinobacillus actinomycetemcomitans*, *Streptococcus spp*., and *Haemophilus spp* [12].

The patient in our case had a co-infection with MTB and Staphylococcus aureus. Both pathogens have the potential to induce inflammation and structural destruction of the lung parenchyma.

This concept is particularly relevant in pulmonary tuberculosis. Several mechanisms may explain coexistence with tuberculosis. On the one hand, tuberculosis produces chronic cavitary lesions suitable for anaerobic colonization, impairs local pulmonary immune defenses, promotes bronchiectatic changes that lead to secretion retention, long-term inflammation, and fibrosis, and previous antituberculosis treatment may alter pulmonary microbiota, facilitating opportunistic colonization [14,13]. On the other hand, chronic actinomycotic infection induces persistent inflammation, tissue necrosis, and fibrosis, potentially impairing local host defenses and facilitating *Mycobacterium tuberculosis* infection [14]. Our patient`s clinical characteristics are consistent with some of these recognized risk factors for pulmonary actinomycosis. For instance, active tuberculosis, diabetes, and advanced age.

Therefore, tuberculosis may act not only as a differential diagnosis but also as a biological facilitator of pulmonary actinomycosis [12,13].

A hallmark of actinomycosis is its indolent, progressively invasive spread into contiguous tissue, crossing normal anatomical boundaries. This process can result in granulomatous inflammation, fibrosis, abscess formation, cavitary lesions, and fistula formation [4].

Pulmonary actinomycosis remains an important diagnostic challenge because it frequently mimics PTB, lung cancer, fungal infections, or chronic lung abscesses [5,6]. It has long been recognized as "the great masquerader".

One of the greatest clinical challenges is the remarkable similarity between pulmonary actinomycosis and PTB. Both diseases commonly present with chronic cough, sputum production, fever, night sweats, weight loss, chest pain, fatigue, dyspnea, and hemoptysis [4,5,7,15]. These symptoms overlap almost completely with PTB, making clinical differentiation difficult. Symptoms usually persist for weeks to months before diagnosis.

Although actinomycosis may affect virtually any organ system, it most commonly presents in the following clinical forms [1,11]. These include:

- Cervicofacial actinomycosis – the most common form (accounting for 50-60% of cases), characterized by chronic inflammation, abscess formation, and draining sinus tracts (fistulas), and is referred to as "lumpy jaw".
- Thoracic actinomycosis accounts for 15-20% of cases and may involve the lungs, pleura, and chest wall, often mimicking PTB or lung cancer.
- Abdominal actinomycosis accounts for approximately 20% of all cases of actinomycosis and is most frequently associated with appendicitis, intestinal perforation, or previous abdominal surgery.
- Pelvic actinomycosis is strongly associated with prolonged intrauterine device use.

In our case, the patient was diagnosed with thoracic actinomycosis, without evidence of extrapulmonary involvement.

CT findings are not specific and include consolidation, pulmonary mass, cavitary lesion, tree-in-bud appearance, nodules, fibrosis, bronchiectasis, pleural effusion, and occasionally chest wall invasion [4,6,15,16]. Radiological overlap between pulmonary actinomycosis and tuberculosis is considerable. Kim TS et al. analyzed the CT imaging characteristics of thoracic actinomycosis in relation to the underlying histopathological findings. Characteristic but nonspecific CT features favoring actinomycosis include: segmental consolidation crossing fissures, low-attenuation areas within consolidation, peripheral enhancement, adjacent pleural thickening, and slow progression despite antibiotic therapy [17, 18]. (Table 1)

Table 1: Radiologic–Pathologic Correlation of CT Findings in Thoracic Actinomycosis [17,18]

CT findings	General characteristics	Histopathology
Segmental airspace consolidation	Typically localized to a pulmonary segment or lobe: • features of chronic inflammation • persist despite antibiotic therapy and does not completely resolve (often mimicking lung cancer or pulmonary tuberculosis)	Chronic inflammatory infiltration
Low-attenuation area	A low-attenuation (hypodense) area within the central portion of the consolidation (represents necrosis, indicating suppurative inflammation/abscess formation)	Necrosis + abscess
Cavitation	Pulmonary cavitation results from central necrosis of the infiltrate associated with suppurative inflammation	Liquefactive necrosis
Pleural thickening	Pleural thickening and pleural reaction adjacent to the infiltrative lesion (consistent with the characteristic propensity of Actinomyces to invade across tissue planes)	Pleural inflammation
Chest wall invasion	In some cases, inflammation may extend to: - The pleura - The chest wall (mimicking a tumor)	Contiguous tissue invasion
Endobronchial actinomycosis • proximal calcification • distal obstructive pneumonia • atelectasis	Some patients presented with a calcified broncholith associated with peribronchial Actinomyces infection.	Broncholith + actinomyces colony

It frequently exhibits intense uptake on PET/CT imaging, making it difficult to distinguish from lung cancer [12]. However, these imaging findings substantially overlap with PTB and lung malignancy, making histopathological consolidation essential for definitive diagnosis [4,8,18].

To differentiate PTB from lung cancer, we planned to perform bronchoscopic biopsy with histopathological evaluation and to examine sputum and bronchoalveolar lavage (BAL) fluid for acid-fast bacilli (AFB). Bronchoscopy revealed an ulcerative, excavated mass-like lesion involving the trachea and right main bronchus with thick secretions. Histopathological examination of the bronchial biopsy demonstrated sulfur granules together with filamentous Gram-positive organisms. In addition, GeneXpert analysis of BAL fluid was positive for *Mycobacterium tuberculosis*, confirming the coexistence of PTB and actinomycosis.

A concomitant infection was confirmed, while no evidence suggestive of malignancy was detected. Histopathological examination of tissue specimens remains the gold standard for definitive diagnosis [1,4,6,7].

Since *Actinomyces species* are slow-growing anaerobic bacteria, the sensitivity of culture is relatively low [1,4,6]. Moreover, because sputum specimens may be contaminated with normal oral flora, sputum culture alone is inadequate for the definitive diagnosis of actinomycosis [4]. In our case, bronchoscopy with BAL and endobronchial biopsy played a critical role because imaging findings mimicked malignancy or tuberculosis.

Comparison with previously published reports demonstrates several similarities. Most reported patients were middle-aged men presenting with chronic respiratory and general symptoms and cavitary or massive pulmonary lesions.

Consistent with previously published reports, our patient presented with chronic respiratory and general non-specific symptoms, right upper lobe pulmonary nodules, a subpleural mass with adjacent pleural thickening, and a pattern of patchy ground-glass attenuation, interlobular septal thickening, and peripheral bronchiectasis.

In many reports, pulmonary actinomycosis was diagnosed only after surgery because the lesions were initially suspected to represent lung cancer or persistent tuberculosis. In general, reported cases of actinomycosis were diagnosed only after failure of antituberculosis therapy. In our case, both infections were identified during the same diagnostic evaluation – bronchoscopy, allowing timely initiation of appropriate combined treatment.

The largest retrospective study conducted in China included 145 patients with primary pulmonary actinomycosis and comprehensively evaluated the disease`s clinical characteristics, diagnostic methods, treatment strategies, and clinical outcomes [7]. The majority of patients were male, with a male-to-female ratio of 2.7:1. The mean age was 48±12 years, and 60.7% had no underlying comorbidities. Of the 145 patients, only five were diagnosed at their initial presentation. Diagnosis was challenging because the clinical and radiological findings were nonspecific, making it difficult to differentiate the disease from lung cancer and tuberculosis. Overall, 75.9% of patients recovered, 20% had an undetermined treatment outcome, and 4% died.

Management of concomitant PTB and pulmonary actinomycosis requires treatment directed at both pathogens. Standard antituberculosis chemotherapy should be administered according to national or international guidelines, while pulmonary actinomycosis generally responds well to prolonged beta-lactam therapy, typically high-dose intravenous penicillin for 2-6 weeks followed by oral amoxicillin for 6-12 months.

Surgical intervention is reserved for selected patients with persistent hemoptysis, extensive pulmonary destruction, empyema, diagnostic uncertainty, or medical therapy.

Early diagnosis allows appropriate antimicrobial treatment and may avoid unnecessary lung resection that is often performed because of suspected malignancy [1]. Clinical improvement should be followed carefully and monitored for drug interactions. Our patient started therapy with antituberculosis drugs and oral amoxicillin with good results.

Straightness and Limitations

The strengths of this case report include the presentation of CT, endoscopic, and histopathological images, together with a comparison of the findings with previously reported cases, and contribute additional evidence regarding this uncommon clinical entity, highlighting the importance of bronchoscopy in the diagnosis of dual infection, allowing for samples for histopathology and microbiology.

Our case emphasizes several important clinical messages. First, pulmonary actinomycosis should remain in the differential diagnosis of patients with chronic pulmonary infiltrates or mass-like lesions, particularly in tuberculosis-endemic countries. Secondly, histopathological confirmation and multidisciplinary collaboration among pulmonologists, microbiologists, pathologists, thoracic surgeons, and infectious disease specialists are essential for timely diagnosis and optimal management.

However, this report has several limitations. Species-level identification and antimicrobial susceptibility testing of Actinomyces were not available.

Conclusions

In conclusion, concomitant pulmonary actinomycosis and PTB is a rare but clinically important condition that can closely mimic either disease alone. Awareness of this possibility, combined with careful microbiological and histopathological evaluation, is essential for early diagnosis and appropriate treatment. Clinicians should maintain a high index of suspicion, particularly in tuberculosis-endemic settings, when clinical or radiological findings are atypical or when patients respond poorly to conventional antibiotics or anti-tuberculosis therapy.

Ethics Approval

The Institutional Medical Ethics Subcommittee waived the requirement for ethical approval because this report describes a single clinical case.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief upon reasonable request.

Author Contribution

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Dr. T.J. and Kh.P. contributed to patient management, data collection, literature review, manuscript drafting, and figure preparation.

Dr. G.D. contributed to radiological interpretation and manuscript revision. Dr. B.N. conceived and supervised the study.

Dr. I.D. conceived and supervised the study, interpreted the findings, critically revised the manuscript, and approved the final version.

Disclosures

All authors have declared that they have no financial relationships at present with any organizations that might have an interest in the submitted work.

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