C A S E R E P O R T

UDC: 616.24-079.4::[616.24-02::579.87
DOI: https://doi.org/10.2298/VSP251104016Z

Pulmonary actinomycosis – a diagnostic dilemma

Aktinomikoza pluća – dijagnostička dilema

Vladan Živković*, Sanja Šarac*†, Svetlana Popović‡, Miloš Zarić§, Saša Ristić§, Vanja Kostovski‖, Jasna Pešić¶, Bojan Nikolić¶, Bojan Rakonjac, Jelena Vuković*†**

Military Medical Academy, *Clinic for Pulmonology, ‡Clinic for Infectious and Tropical Diseases, ‖Clinic for Thoracic Surgery, §Institute of Pathology and Forensic Medicine, ¶Institute of Radiology, **Institute of Microbiology, Belgrade, Serbia; †University of Defence, Faculty of Medicine of the Military Medical Academy, Belgrade, Serbia

Abstract

Introduction. Pulmonary actinomycosis is a chronic inflammatory infectious disease caused by anaerobic and/or microaerophilic bacteria of the genus *Actinomyces* spp. It causes non-specific symptoms in the patient and gives an atypical clinical and radiographic presentation. It is extremely rare and is characterized by the formation of abscesses and fistulas in the lungs or local fibrosis. In the differential diagnosis, diseases and pathological conditions from tuberculosis to neoplastic processes in the lungs must be considered. **Case report.** We present a 39-year-old patient with an infiltrative lesion in the apex of the right lung and non-specific symptoms. In this patient, actinomycosis was pathohistologically proven after surgical intervention. Within a period of just over a month, a multi-detector computed tomography of the chest and bronchoscopy with transbronchial biopsy and bronchial swab were performed twice. Initially, pulmonary actinomycosis was not suspected. After an atypical resection of the tumor mass in the apex of the right lung and histopathologically proven infection, the patient was treated with antibiotic therapy for 6 months. Further examination was carried out to prove a potential primary or secondary immunodeficiency. **Conclusion.** Patients with non-specific changes in the lung parenchyma should also be suspected of having actinomycosis, and after diagnosis, it is necessary to supplement the examination to confirm or exclude immunodeficiency.

Keywords:
actinomyces; actinomycosis; biopsy; bronchoscopy; diagnosis; multidetector computed tomography; thoracoscopy.

Apstrakt

Uvod. Aktinomikoza pluća je hronična zapaljenska infektivna bolest koju izaziva anaerobna i/ili mikroaerofilna bakterija iz roda *Actinomyces* spp. Izaziva nespecifične simptome kod bolesnika i daje atipičnu kliničku i radiografsku sliku. Veoma je retka i karakteriše je stvaranje apscesa i fistula u plućima, ili lokalna fibroza. U diferencijalnoj dijagnozi moraju se razmotriti bolesti i patološka stanja od tuberkuloze pluća do neoplastičnog procesa u plućima. **Prikaz bolesnika.** Predstavljamo 39-godišnjeg bolesnika sa infiltrativnom promenom u gornjem delu desnog plućnog krila i nespecifičnim simptomima. Kod ovog bolesnika, aktinomikoza je patohistološki dokazana posle hirurške intervencije. U periodu od nešto više od mesec dana, dva puta su urađene multidetektorska kompjuterizovana tomografija grudnog koša i bronhoskopija sa transbronhijalnom biopsijom i bronhijalnim brisom. U početku se nije sumnjalo na aktinomikozu pluća. Nakon atipične resekcije tumorske mase u gornjem delu desnog plućnog krila i histopatološki dokazane infekcije, bolesnik je lečen antibiotskom terapijom u trajanju od 6 meseci. Sprovedena su dalja ispitivanja da bi se potvrdila moguća primarna ili sekundarna imunodeficijencija. **Zaključak.** Kod bolesnika sa nespecifičnim promenama u plućnom parenhimu treba sumnjati i na aktinomikozu, a nakon dijagnoze potrebno je dopuniti ispitivanje kako bi se potvrdila ili isključila imunodeficijencija.

Ključne reči:
actinomyces; aktinomikoza; biopsija; bronhoskopija; dijagnoza; tomografija, kompjuterizovana, multidetektorska; torakoskopija.

Introduction

Actinomyces are opportunistic Gram-positive bacteria with a filamentous morphology, characterized by slow growth and the ability to cause infection. They are usually found in the oral cavity, in dental caries, and in the tonsils. Pulmonary actinomycosis (PA) is a chronic, purulent, granulomatous disease caused by the bacteria from the

Correspondence to: Sanja Šarac, Military Medical Academy, Clinic for Pulmonology, Crnotravska 17, 11 000 Belgrade, Serbia. E-mail: sanja.sarac9@gmail.com

Actinomyces spp., of which *Actinomyces (A.) graevenitzii*, which is an anaerobic or microaerophilic bacterium, is the most causative agent [1, 2].

The largest number of infections occurs due to poor oral hygiene and inhalation of actinomycetes-containing secretions. Infections can also spread through the blood or through abdominal lesions. The most common infections are cervicofacial or abdominopelvic, while lung actinomycosis accounts for about 15% of all actinomycosis. The ratio of men to women is 3 : 1. The clinical and radiographic presentation of PA is non-specific and can lead to confusion, misdiagnosis, and wrong treatment. PA can resemble a tumor or tuberculosis in the lungs [3].

Factors associated with the development of PA include inadequate oral hygiene, periodontal inflammation, dental pathology, recent dental procedures, mucosal or thoracic trauma, aspiration events, states of impaired immunity, as well as previously existing actinomycosis at local or distant sites [4].

Two studies have shown that PA is accurately diagnosed in fewer than 4–7% of patients. Additionally, reports indicate that up to 25% of cases are initially misclassified as malignant disease [4, 5].

We present a patient whose actinomycosis in the lungs was confirmed after an atypical resection of the upper lobe of the right lung, and after non-specific findings on two bronchoscopies. After surgery, the treatment was continued with antibiotics.

Case report

A 39-year-old male patient presented to the emergency department with a cough lasting for the past 3 months and stabbing pain in the right scapular region. The pain worsened during coughing. He denied hemoptysis and reported occasional expectoration of greenish sputum. Vital parameters were stable: arterial blood pressure was 120/80 mmHg, pulse was 89 beats *per* min, and there were no changes on the electrocardiogram. Chest X-ray revealed an infiltrative lesion in the apex of the right lung. Laboratory tests showed the following: white blood cells 12.2 × 10^9/L [reference range (RR) 4.0–10.0 ×10^9/L] with a predominance of neutrophils, platelets 434 ×10^9/L (RR 150–400 ×10^9/L), C-reactive protein 41 mg/L (RR < 5 mg/L), and the rest was within the reference value limits. As there were no indications for emergency admission, the patient was discharged with cefixime and analgesic antipyretics as needed, and an outpatient contrast-enhanced multi-detector computed tomography (MDCT) of the chest was requested.

Chest MDCT showed an infiltrative lesion in the apex of the right lung, measuring 32 × 46 × 78 mm, with involvement of the visceral pleura, and surrounded by bullae and emphysema, along with a right paratracheal (4R) lymph node measuring 14 × 15 mm. An outpatient bronchoscopy with transbronchial biopsy (TBB) was performed 7 days after the initial presentation to the emergency center–endobronchial ultrasound radial probe with TBB, where the endoscopic findings were normal, and the histopathological (HP) findings were in favor of acute inflammation, with no signs of a specific inflammatory process and without any tumor infiltration.

As the patient's weakness, sweating, cough, and pain in the upper right hemithorax persisted, he returned to the emergency center 20 days after the initial examination. Vital parameters were stable again, and auscultatory findings were normal. The patient was given opioid analgesics and co-analgesics along with oral corticosteroids and gastroprotection therapy; a neoplastic process in the apex of the right lung was suspected. The day after the second examination at the emergency center, a repeated bronchoscopy was scheduled, and 7 days later, the patient was admitted to the hospital. Bronchoscopy again yielded non-specific HP findings–a mixed inflammatory infiltrate with a predominance of neutrophils and no evidence of malignant cells–from bronchial swabs and aspirates. HP analysis showed that the lung tissue was without neoplasm or specific inflammation. TBB was performed under X-ray control through the bronchus of the apical segment of the right lung, and bronchial swabs and aspirates were obtained for cytology, bacteriological analysis, acid-fast bacilli/Lowenstein testing.

Following the negative results of the above-mentioned tests, the patient was admitted for inpatient evaluation in a significantly improved general condition, with pain fully controlled by opioids. He was not receiving treatment for any chronic conditions, denied drug and food allergies, and reported no history of serious illnesses, injuries, or surgeries. He was in good general condition, self-motivated, physically active, and able to work. The patient reported undergoing dental prosthetic procedures on both jaws in 2015 (the upper jaw), and, in August 2022, a tooth root extraction in the lower jaw followed by placement of a dental bridge. He also reported a smoking history of approximately 20 pack-years.

During the hospital treatment, a second chest MDCT was performed, revealing a heterogeneous lesion in the apical segment of the right lung measuring 42 × 70 × 65 mm. The lesion appeared partly as consolidation and partly as an infiltrative lesion, surrounded by smaller inflammatory zones. High subpleural zones of hypodensity were present, possibly corresponding to zones of necrosis; some of them contained gas inclusions, for which it is not possible to say with certainty whether it was the formation of an abscess or zones of necrosis around subpleural bullae. Minimal right-sided pleural effusion was also observed. Right bronchopulmonary and paratracheal lymph nodes measuring up to 15 mm were noted (Figures 1–6).

Pulmonary function tests were performed. Spirometry showed a forced expiratory volume in one second (FEV1) of 3,330 mL or 86.3% (RR ≥ 80% of predicted) and a forced vital capacity (FVC) of 4,650 mL or 99.6% (RR ≥ 80% of predicted). The FEV1/FVC ratio was 71.66% (RR ≥ 70%). Maximal expiratory flow at 75% was 73.8% (RR ≥ 65% of predicted), maximal expiratory flow at 50% was 57.4% (RR ≥ 65% of predicted), and maximal expiratory flow at 25% was 47.3% (RR ≥ 60% of predicted). Diffusion capacity for carbon monoxide (DLCO) was 71.2% (RR 80–120% of

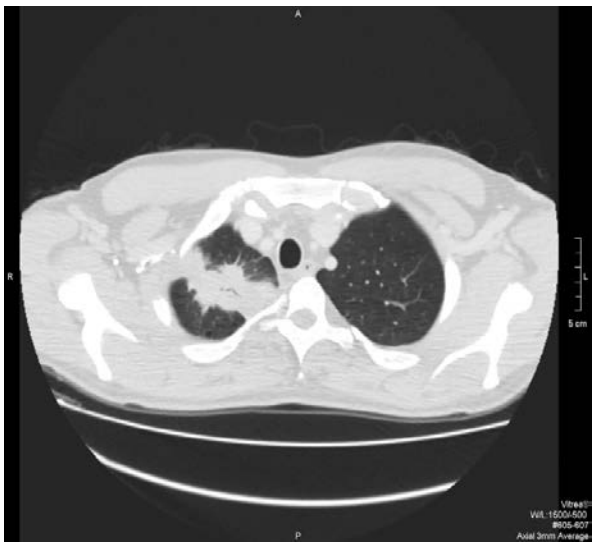

Fig. 1 – Chest multi-detector computed tomography: infiltrative/consolidative lesion in the apex of the right lung characterized as infiltrative (axial section).

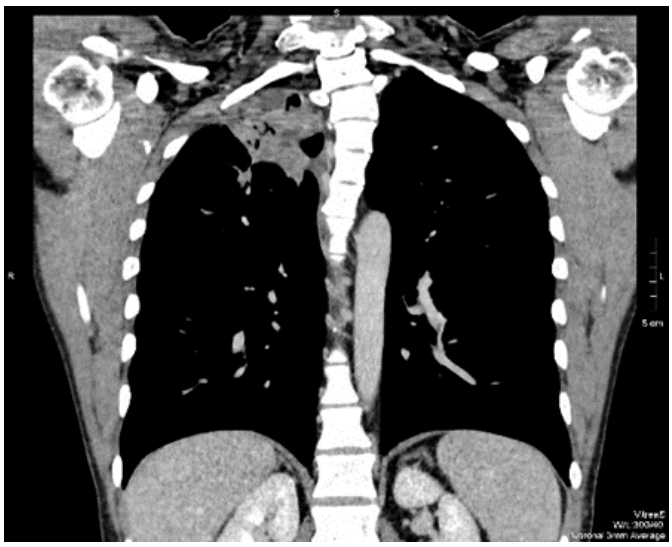

Fig. 2 – Chest multi-detector computed tomography: above lesion visible areas of necrosis (coronal section).

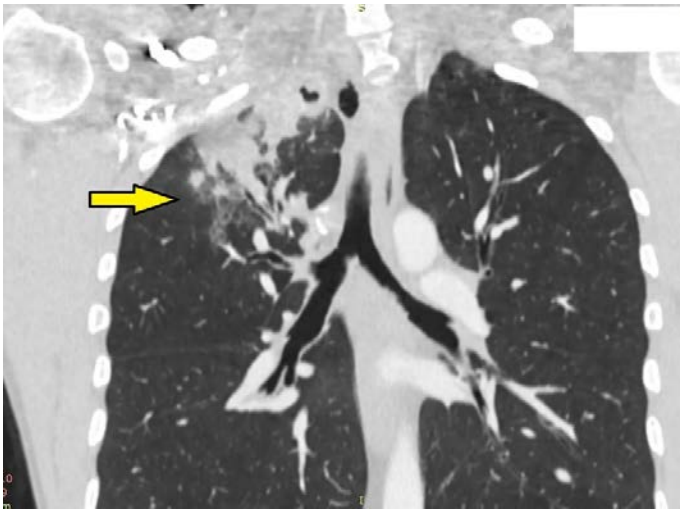

Fig. 3 – Chest multi-detector computed tomography: adjacent zones of patchy consolidation and pneumonitis.

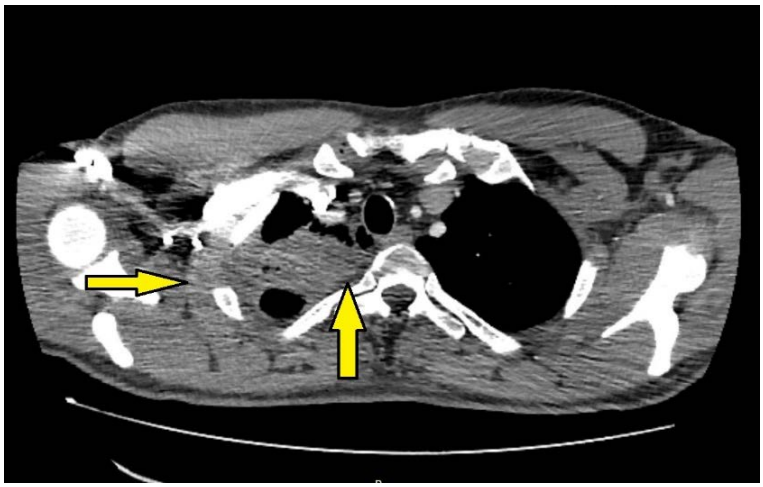

Fig. 4 – Chest multi detector computed tomography (axial section) demonstrating areas of demarcation between pulmonary parenchymal changes and the adjacent pleura (arrows).

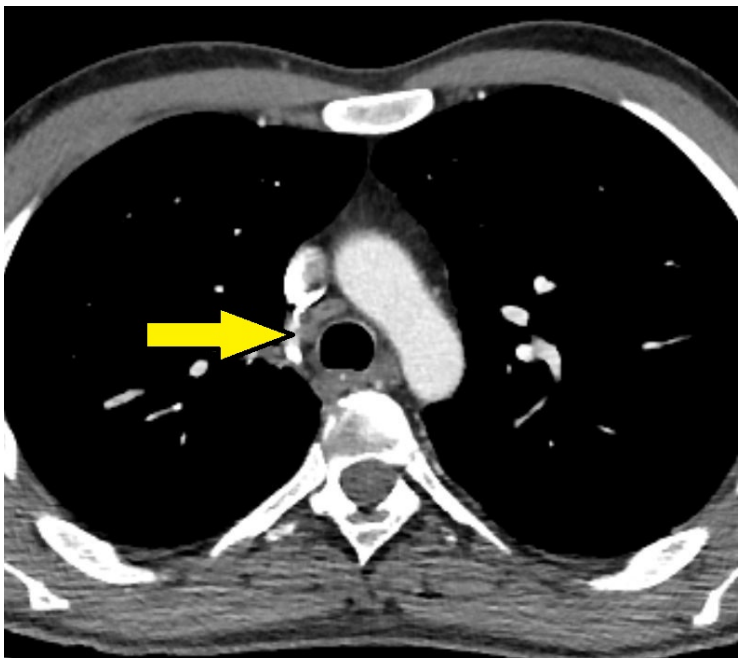

Fig. 5 – Chest multi-detector computed tomography: enlarged right lower paratracheal (station 4R) lymph node (axial section).

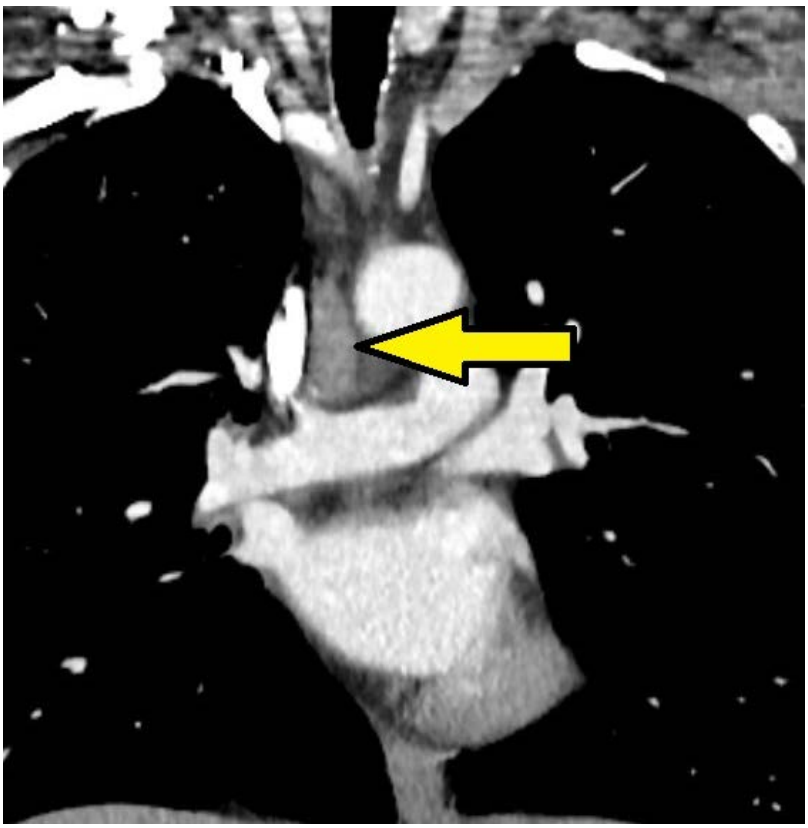

Fig. 6 – Chest multi-detector computed tomography: enlarged right lower paratracheal (station 4R) lymph node (coronal section).

predicted). Diffusing capacity divided by the alveolar volume (DLCO/VA) was 72.3% (RR 80–120% of predicted). Laboratory tests showed mild leukocytosis and slightly elevated inflammatory markers, while other parameters were within reference ranges.

During hospital treatment, the patient was treated with a parenteral antibiotic (ceftriaxone), and therapy was continued with tapentadol (sustained release). Throughout the hospital treatment, the patient was afebrile, and the pain syndrome was no longer present.

A thoracic surgeon was consulted, and the patient was prepared for a diagnostic and therapeutic thoracic surgical procedure due to the suspicion of a neoplastic process of the right lung.

A month after the initial examination in the emergency center, video-assisted thoracoscopy (VATS) and atypical resection of the upper lobe of the right lung were performed. Operative findings revealed that the apical segment of the upper lobe was attached to the mediastinal and costal pleura. The apical segment was almost entirely firm in consistency, more by type of consolidation. A biopsy was performed on the altered part of the upper lobe. The tissue was sent for *ex tempore* examination, which indicated a benign finding (inflammation). Athesiolysis of the upper lobe was performed with a sharp blunt preparation. Then, an atypical resection of the upper lobe with a change was performed with the help of endoscopic mechanical staplers.

The definitive HP finding was chronic bronchopneumonia in acute exacerbation with suppurative abscess formation and incomplete organization, pulmonary actinomycosis, perifocal cholesterol pneumonitis, fibrosis of the visceral pleura, and recent intraalveolar hemorrhage (Figures 7–11).

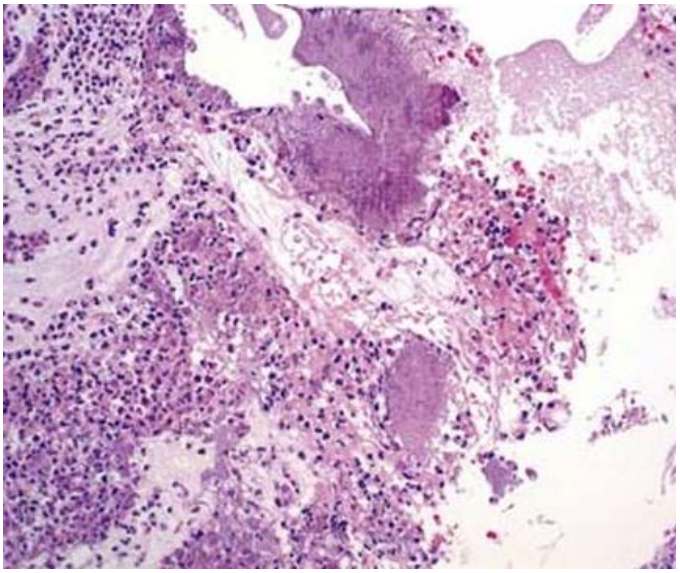

Fig. 7 – Histological examination of the sample lung parenchyma: inflammatory cellular lymphoplasmacytic infiltration and colonies of *Actinomyces* spp. surrounded by necrotic tissue and inflammatory cells (hematoxylin-eosin staining, ×10).

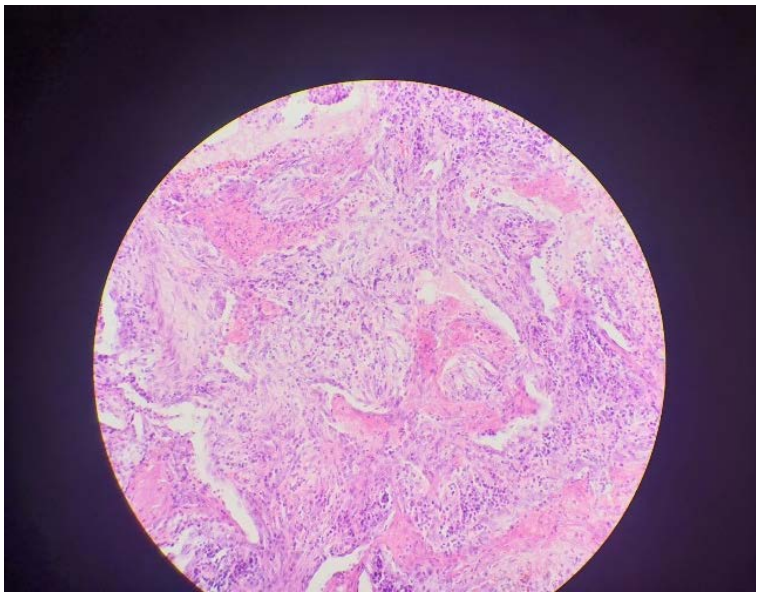

Fig. 8 – Masson bodies (hematoxylin-eosin staining, ×4).

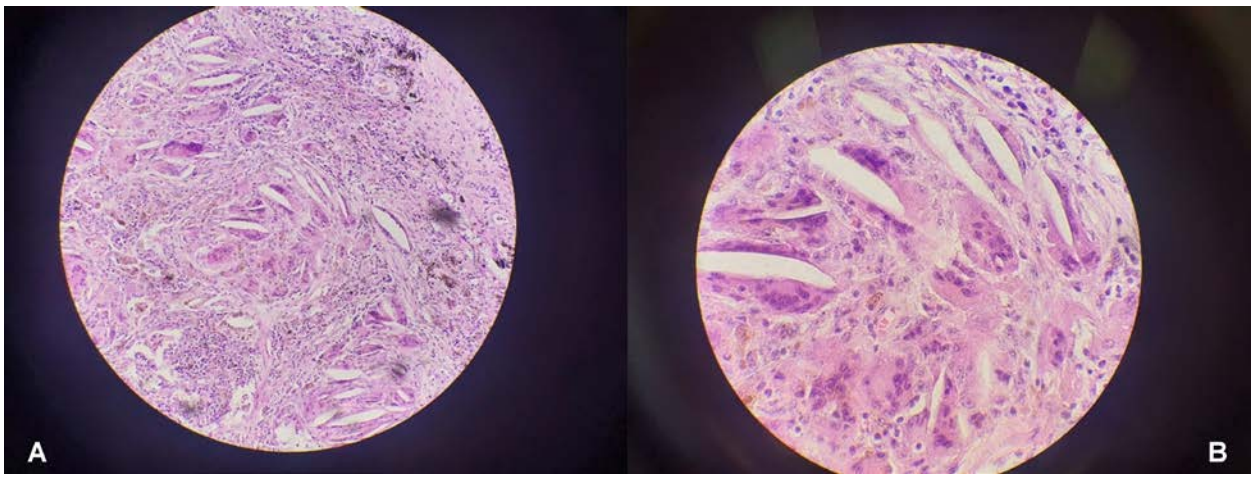

Fig. 9 – Cholesterol granulomas: hematoxylin-eosin staining magnification, ×10 (A) and ×20 (B).

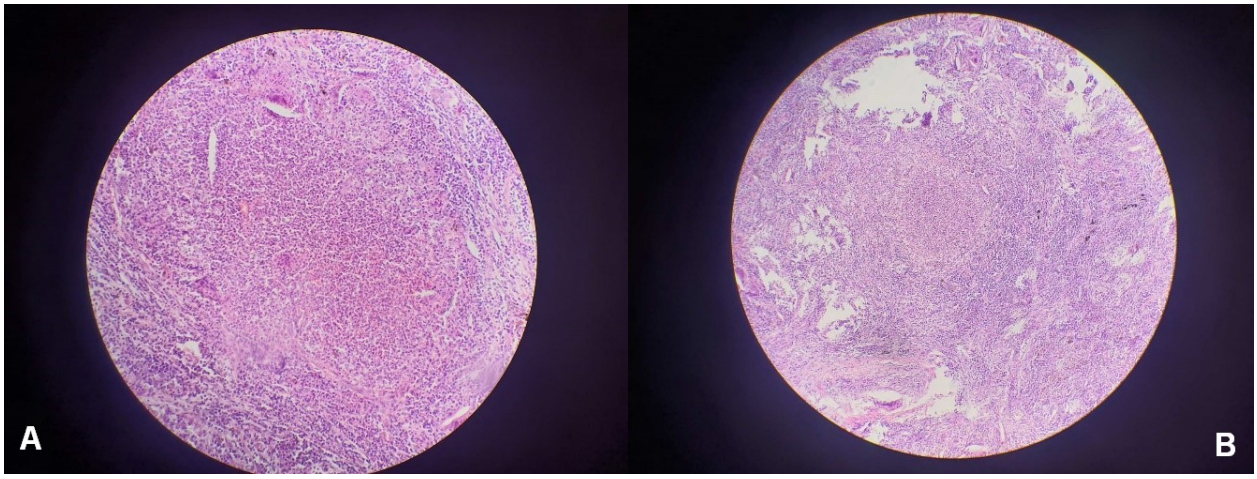

Fig. 10 – Abscessed inflammation: hematoxylin-eosin staining magnification, ×10 (A) and ×4 (B).

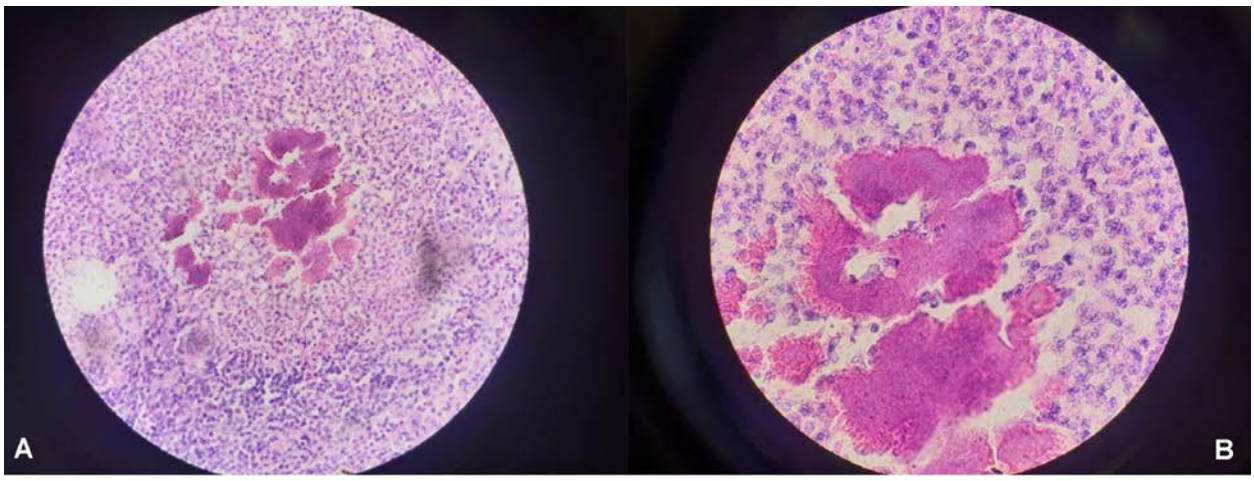

Fig. 11 – Actynomices colonies: hematoxylin-eosin staining magnification, ×40 (A) and ×60 (B).

The postoperative course went well, the chest drain was removed after establishing the aerostasis, and the patient was discharged home for treatment 2 days after the intervention.

A month after the surgery, the patient was admitted to the Clinic for Infectious and Tropical Diseases, Belgrade, Serbia, for evaluation and initiation of antibiotic therapy for pulmonary actinomycosis. At the aforementioned clinic, the patient was fully examined to identify an immunosuppressive condition that may have contributed to the development of a lung infection. Numerous analyses were performed, including an ultrasound of the abdomen,

pelvis, and scrotum, which was normal, and esophagogastroduodenoscopy, which showed no pathological findings. MDCT of the paranasal cavity revealed polypoid masses in the left nasal passage and *deviatio septi nasi* on the right side. Ultrasound of the axilla was normal, stool testing for occult bleeding was negative, and immunological analyses showed no deviations from normal values.

He was treated with ceftriaxone and clindamycin for 4 weeks and discharged from the hospital with doxycycline for further home treatment (6 months). A follow-up MDCT of the thorax and abdomen was performed; there were no signs of residual or recurrent infection. At the control pulmonology outpatient examination, the patient reported no complaints and was in good general condition. He remained under follow-up by infectious disease and pulmonary specialists for two years with no ongoing pharmacological therapy.

Discussion

PA is a very rare condition, with an estimated incidence of approximately 1 *per* 3,000,000 people a year. It is a chronic, slowly progressive bacterial infection caused by several species belonging to the *Actinomyces* genus. Among the six *Actinomyces* spp. known to be pathogenic in humans, *A. israelii* is the most commonly identified. In pulmonary forms of the disease, however, *A. graevenitzii* is most frequently reported as the causative agent [4].

Actinomycosis is often referred to as the "great imitator" because of its ability to mimic various other conditions. Nevertheless, it can be distinguished from infections caused by other non-acid-fast filamentous bacilli by the presence of sulfur granules, which are considered pathognomonic. Although the infection most commonly involves the oral and cervicofacial areas, it may also affect other anatomical sites, particularly in immunocompromised patients. PA usually develops following aspiration of oropharyngeal or gastrointestinal secretions into the lower airways [6]. Furthermore, this form of the disease is reported more frequently in developing regions than in developed countries [7].

It is assumed in this case and in our patient that, due to inadequate oral hygiene and in the absence of an immunosuppressive state, there was a slow development of PA with the formation of a larger pulmonary infiltrate, one year after the mentioned dental intervention.

Clinical manifestations of PA are very nonspecific. They can be differentially diagnosed from chronic lung infections to neoplastic manifestations [8, 9]. The symptoms that are manifested also have a wide range. The most common symptoms are fever, very often subfebrile, weight loss, productive cough, chest pain, and hemoptysis [4]. Clinical and radiological signs can mimic various lung diseases, from tuberculosis to malignant lung diseases [10]. The diagnosis of PA is established based on histological and microbiological examination of a sample obtained *via* bronchoscopy or after surgical intervention [11].

Indications for surgical intervention are the existence of severe invasive disease, infection of critical areas, as well as the existence of massive refractory hemoptysis. Surgical treatment is indicated in patients with involvement of, or proximity to, the main blood vessels or large airways, as well as in cases of greater involvement of the lung parenchyma. The primary goal of surgical intervention is to reduce the extent of the disease (tumor mass) and prevent life-threatening complications–in this case, involvement of almost the entire right upper lobe, accompanied by a long-standing cough and pain syndrome. The mass in the right upper lobe had the appearance of a neoplastic lesion, which had not previously received HP verification [12].

Since the diagnosis could not be established by repeated TBB, the lesion appeared as tumorous on MDCT, and the patient's symptoms persisted, VATS was performed.

Due to the processing of the sample for HP analysis, subsequent microbiological processing at the Institute of Microbiology, was not possible in this case, which would involve microscopic examination (Gram's staining to observe filamentous, Gram-positive bacteria arranged in characteristic aggregates–the so-called "sulfur granules"), as well as cultivation on Brucella blood agar in anaerobic conditions, with additional identification and differentiation of the most common causative agents of actinomycosis, including *A. israelii*, *A. naeslundii*, *A. odontolyticus*, *A. meyeri*, and *A. graevenitzii*.

Treatment of PA involves the use of antimicrobial therapy with or without surgery. Surgical treatment of PA requires removal of infected tissue, which can be performed by VATS or open surgery. In severe disease, segmentectomy or even lobectomy is sometimes indicated. Surgical treatment must be combined with antimicrobial therapy, because isolated surgical treatment as the only type of treatment is rarely successful [13]. In this case, our patient underwent an atypical resection of the right upper lobe. Once the pathologist called the *ex tempore* as benign, the lung resection was ended as atypical. Antibiotic therapy for mild to moderately severe disease includes oral phenoxymethylpenicillin, 2–4 g in two doses, or amoxicillin, 1.5–3 g daily in three or four doses. In cases where infection by additional pathogens is suspected, the use of amoxicillin with clavulanic acid in a dose of 1g twice a day *per os* is indicated. The duration of treatment should usually be continued for 1–2 months after the cessation of symptoms. The treatment usually lasts 2–6 months in mild forms of the disease. In severe forms of the disease, the use of high doses of parenteral antibiotics, benzylpenicillin or ceftriaxone is required. If there is any doubt about the presence of pathogenic bacteria, the use of piperacillin, tazobactam 4.5 g four times a day is recommended. The duration of intravenous therapy is 2–6 weeks, after which, upon clinical improvement, the patient is transferred to oral therapy. Oral treatment lasts between 6 and 12 months, depending on the severity of the disease and indicators of clinical improvement [14].

HP etiologically clarified the lesion in the right upper lobe of the lung after it was removed. Our patient had no primary or secondary immunodeficiency, so we concluded that the actinomycosis arose in the context of extensive dental interventions performed in the previous period.

According to Kim et al. [10], out of 94 analyzed cases, 50% of diagnoses were established through surgical biopsy, while bronchoscopic biopsy was successful in 25.5% of cases. Other authors report that the surgical method was twice as effective in establishing a diagnosis compared to bronchoscopy [15].

Conclusion

Actinomycosis of the lung is a very rare disease that can have an atypical clinical and radiological presentation. It very often imitates other diseases, including neoplastic processes in the lungs. In this regard, it requires a multidisciplinary approach, both diagnostic and therapeutic.

Conflict of interest

Authors declare no conflict of interest.

R E F E R E N C E S

1. *Oikonomidis P, Fousekis F, Kotsaftis P, Pilios I, Dimas D, Giannoulis G.* A case of pulmonary actinomycosis presented with endobronchial involvement. Respir Med Case Rep 2019; 28: 100930. DOI: 10.1016/j.rmcr.2019.100930.
2. *Khoshbayan A, Amirmozafari N, Mirkalantari S.* An overview of case reports and case series of pulmonary actinomycosis mimicking lung cancer: a scoping review. Front Med (Lausanne) 2024; 11: 1356390. DOI: 10.3389/fmed.2024.1356390.
3. *Baykal H, Ulger AF, Çelik D, Tanrikulu FB, Tatci E.* Clinical and radiological characteristics of pulmonary actinomycosis mimicking lung malignancy. Rev Assoc Med Bras (1992) 2022; 68(3): 372−6. DOI: 10.1590/1806-9282.20211017.
4. *Zhang N, Liu C, Kang L, Zhou J, Liu W, Qu X.* A Case of Pulmonary Actinomycosis With Concurrent Gastric Adenocarcinoma in an Older Adult. Cureus 2024; 16(5): e60180. DOI: 10.7759/cureus.60180.
5. *Yildiz O, Doganay M.* Actinomycoses and nocardia pulmonary infections. Curr Opin Pulm Med 2006; 12(3): 228−34. DOI: 10.1097/01.mcp.0000219273.57933.48.
6. *Moniruddin A, Begum H, Nahar K.* Actinomycosis: an update. Med Today 2010; 22 (1): 43−7. DOI: 10.3329/medtoday.v22i1.5606.
7. *Allan L, Said H, Shanks AM.* Pulmonary actinomycosis presenting with empyema. BMJ Case Rep 2023; 16(11): e256320. DOI: 10.1136/bcr-2023-256320.
8. *Mabeza GF, Macfarlane J.* Pulmonary actinomycosis. Eur Respir J 2003; 21(3): 545−51. DOI: 10.1183/09031936.03.00089103.
9. *Boot M, Archer J, Ali I.* The diagnosis and management of pulmonary actinomycosis. J Infect Public Health 2023; 16(4): 490−500. DOI: 10.1016/j.jiph.2023.02.004.
10. *Kim SR, Jung LY, Oh IJ, Kim YC, Shin KC, Lee MK,* et al. Pulmonary actinomycosis during the first decade of 21st century: cases of 94 patients. BMC Infect Dis 2013; 13: 216. DOI: 10.1186/1471-2334-13-216.
11. *Suzuki M, Araki K, Matsubayashi S, Kobayashi K, Morino E, Takasaki J,* et al. A case of recurrent hemoptysis caused by pulmonary actinomycosis diagnosed using transbronchial lung biopsy after bronchial artery embolism and a brief review of the literature. Ann Transl Med 2019; 7(5): 108. DOI: 10.21037/atm.2019.02.11.
12. *Wong VK, Turmezei TD, Weston VC.* Actinomycosis. BMJ 2011; 343: d6099. DOI: 10.1136/bmj.d6099.
13. *Song JU, Park HY, Jeon K, Um SW, Kwon OJ, Koh WJ.* Treatment of thoracic actinomycosis: a retrospective analysis of 40 patients. Ann Thorac Med 2010; 5(2): 80−5. DOI: 10.4103/1817-1737.62470.
14. *Yuan Y, Hou Z, Peng D, Xing Z, Wang J, Zhang S.* Pulmonary Actinomyces graevenitzii Infection: Case Report and Review of the Literature. Front Med (Lausanne) 2022; 9: 916817. DOI: 10.3389/fmed.2022.916817.
15. *Ding X, Sun G, Fei G, Zhou X, Zhou L, Wang R.* Pulmonary actinomycosis diagnosed by transbronchoscopic lung biopsy: a case report and literature review. Exp Ther Med 2018; 16(3): 2554–8. DOI: 10.3892/etm.2018.6483.

Received on November 4, 2025
Accepted on January 14, 2026
Online First April 2026