**Actinomyces turicensis Bacteremia Presenting with Empyema of Lung: A Case Report**

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**Abstract**

**Background:** Bacteremia due to *Actinomyces turicensis* is very rare. Of the limited number of reported cases of *A. turicensis* bacteremia, none had pulmonary involvement. *Actinomyces* require anaerobic conditions and positive cultures may not show up within 10 days, making its diagnosis very difficult. In addition, it can be a challenge to treat, requiring multiple debridement and long courses of IV and oral antibiotics. **Case presentation:** A 72-year-old male former shipyard and coalmine worker with history of asbestos exposure, 40 pack-year history of cigarette smoking, emphysema, peripheral vascular disease, chronic kidney disease, and prior history of melanoma was admitted after he developed hypoxia following a Port-a-Cath placement. He was recently diagnosed with right lower lobe pleural-based stage 3B squamous cell carcinoma of the right lung. On admission, he was afebrile, but tachycardic and tachypneic. Laboratory findings were consistent with anemia, and leukocytosis which worsened the next day, and repeated chest X-ray showed enlarging pleural effusion. Thoracentesis was performed and fluid analysis was consistent with empyema. Antibiotics were transitioned to (Ampicillin and Sulbactam). Chest tube with tissue plasminogen activator (t-PA) was placed for loculated pleural effusion. On day two, blood cultures came back positive for *Actinomyces turicensis* and *Rothia mucilaginosa*. **Conclusions:** Recognized clinical risk factors for *Actinomyces* infection should form part of the clinical assessment to establish the significance of *Actinomyces turicensis* isolated from blood cultures.

**Keywords:** *Actinomyces turicensis*; Bacteremia; Carcinoma; Empyema; Leukocytosis

**Introduction**

*Actinomyces* are a species of slow growing, microaerophilic gram-positive rods characterized by filamentous branching growth patterns. They constitute a part of the commensal flora in the oropharynx, gastrointestinal tract, and urogenital tract [1]. They rarely cause disease in healthy individuals; however, they are opportunists. They require a disruption in the mucosal barrier to allow displacement of bacteria [2]. Sometimes, this inoculation can produce a slowly progressive granulomatous disease known as actinomycosis, which is characterized by abscess formation and draining sinus tracts that can penetrate surrounding tissues. *Actinomyces* require anaerobic conditions and positive cultures may not show up within 10 days, making its diagnosis very difficult. In addition, it can be a challenge to treat. Often patients require multiple debridement and long courses of intravenous (IV) and oral antibiotics [3]. The most commonly affected site is the cervicofacial area and is usually associated with poor dental hygiene, tooth extraction, or trauma. However, *Actinomyces* species can cause ocular infections, urinary tract infections, genital infections, intrauterine contraceptive device infections, intraabdominal infections, endocarditis, pericarditis, pulmonary infections, central nervous system infections and bacteremia [4]. Although *Actinomyces turicensis* is a relatively common species in humans, cases of *actinomyces* bacteremia are rare and present only in 3.8% examinations (3/80 strains) [5]. Furthermore, based on literature review performed by Hagiya et al. [6], of all the reported cases of *A. turicensis*, none included pulmonary involvement. We describe a case of a patient with non-small cell lung carcinoma presenting with febrile illness and shortness of breath and who was found to have *actinomyces* bacteremia that had a clinical course complicated with recurrent empyema.

**Case presentation**

A 72-year-old male, former shipyard and coalmine worker with 40 pack-year history of cigarette smoking, emphysema, chronic kidney disease, prior history of melanoma and peripheral vascular disease was admitted after he developed hypoxia following Port-a-Cath placement. He was recently diagnosed with stage 3 squamous cell carcinoma of the right lung. On admission, he was afebrile (98.3°F), tachycardic at 108 beats per minute, and tachypneic. Laboratory findings were consistent with anemia (8.9 gm of...
hemoglobin per deciliter (dL) of blood), leukocytosis ($25.0 \times 10^9$/L) with a neutrophilic predominance. Computed Tomography (CT) scan of the chest for Pulmonary Embolus study protocol showed right pleural effusion with atelectasis and infiltrates in the right middle and lower lobes (Figures 1 and 2).

He was started empirically on IV cefepime and vancomycin. The following day, his leukocytosis worsened ($30.6 \times 10^9$/L) and the repeated chest X-rays showed enlarging pleural effusion. Thoracentesis was performed and 1100 cc of cloudy green fluid were removed. Fluid analysis showed WBC count of 9222, glucose less than 10 mg/dl, Lactate dehydrogenase (LDH) of 2140 and pH of 7.5 consistent with empyema. Pleural fluid cultures remained negative. Antibiotics were transitioned to IV Unasyn (Ampicillin and Sulbactam).

Figure 1: Lung window. Loculated Right Pleural effusion with consolidation and atelectasis.

Chest tube was placed for loculated pleural effusion and tissue plasminogen activator (t-PA) was instilled via the tube. On day 2, blood cultures came back positive for *Actinomyces turicensis* and *Rothiamucilaginosa*. He was started on IV penicillin G 12 million units every 12 hours for 6 weeks and IV vancomycin for 2 weeks, followed with plans to complete oral penicillin for 6-12 months. Chest tube was removed and he was discharged to a short-term rehabilitation facility. Six days later, he again presented with tachycardia and dyspnea. He was afebrile (99.9°F), but tachycardic (109 beats per minute). Laboratory findings were significant for initial lactate of 0.7 mmol/L, leukocytosis of $11.2 \times 10^9$/L. Vancomycin was empirically added. The next day, he became febrile (102°F). While he was on IV penicillin and vancomycin, his leukocytosis also worsened to 17600. He was empirically converted to Vancomycin and Piperacillin-tazobactam. A chest tube was placed. Pleural analysis was consistent with empyema, with 97980 nucleated cells, glucose less than 10 mg/dl and LDH of 12000. Pleural culture was negative. He continued to clinically improve and chest tube was removed once output decreased. He was set to continue Piperacillin- tazobactam for 2 more weeks and amoxicillin for 6 months.

**Discussion**

The genus *Actinomyces* comprises a group of 42 species and 2 subspecies. *Actinomyces* spp. are present in polymicrobial flora and are often isolated with normal commensals, such as *Aggregatibacter actinomycetemcomitans*, *Eikenella corroden*, *Capnocytophaga*, * fusobacteria*, *Bacteroides*, * staphylococci*, * streptococci*, or *Enterobacteriaceae*. The most common cause of human disease among the *Actinomyces* species is *A. israelii*, however with improved molecular techniques, there have been several newly discovered species [7]. This case report focuses on one species, *A. turicensis* that is becoming an important emerging cause of infection.

Figure 2: Coronal view with soft tissue window. Pleural effusion with parenchymal opacity from atelectasis and consolidation of Right middle and lower lobe.

Pulmonary actinomycosis is the third most common type of actinomycosis. Respiratory tract actinomycosis includes pulmonary, bronchial, and laryngeal actinomycosis [7]. The peak incidence is reported to be in the fourth and fifth decades of life. Males are more often affected than females, with a 3:1 ratio. Pulmonary actinomycosis results mainly from aspiration of oropharyngeal or gastrointestinal secretions, direct or indirect extension from cervicofacial infection to the thorax and hematogenous spread [8]. Individuals with poor oral hygiene, preexisting dental disease, and alcoholism, malignancy, chronic lung disease such as emphysema, chronic bronchitis, and bronchiectasis, and patients with pulmonary sequelae following tuberculosis, are at risk for pulmonary actinomycosis [8]. At early stages of the disease, a focal pulmonary consolidation occurs, and can be surrounded by pulmonary nodules, but there are often no associated physical symptoms at this stage. Progression can lead to a peripheral mass, with or without cavitation. Conversely, pulmonary actinomycosis could be associated with extra pulmonary spread, from the lung to the pleura resulting in empyema or mediastinal actinomycosis. Rarely, it can be associated with abscess formation on the thoracic wall and pus eroding through the chest wall, causing “empyema necessitatis” [4].

After it was first identified in 1995, there have been only 8 cases of A. turicensis bacteremia described in published articles. Although A. turicensis is a relatively common infectious cause, out of a group of 80 people in one study, blood culture examinations were positive in only three cases [6]. Bacteremia is common after invasive dental procedures and after tooth brushing in subjects with gingivitis and periodontitis. The case of our patient is unusual in that there were no direct aspiration events or oral manipulative events that could be behind his bacteremia. He however has several risk factors for pulmonary actinomycosis which could have progressed into a bacteremia, including malignancy and chronic lung disease. Micro aspiration events could be a possible etiology. As the bronchoscopy was introduced into his oropharynx, micro aspirations could have introduced oral flora into his lung, where a lung mass with a large necrotic burden may have acted as a nidus for infection [7]. The slow clinical course may have allowed for development of pneumonia progressing to empyema and bacteremia. Another possibility could be the recent port catheter placement; however, there was no evidence of line infection. The diagnostic challenge is the difficulty in accurately identifying this bacterium and is considered to be a major reason for the small number of reported cases. Moreover, conventional laboratory methods using biochemical profiles may lead to misidentification and molecular analysis may not be readily available at all facilities. Finally, acquiring adequate samples may be difficult as purulence may be minimal and localized in sulfur granule micro colonies deep in the indurated tissue. Ideally, a tissue culture would have to be performed [8].

Penicillin G is the treatment of choice for actinomycosis, although a number of other antimicrobials (ampicillin,
doxycycline, erythromycin, and clindamycin) are active in vitro and have shown clinical effectiveness [3]. It is noteworthy that there are considerable differences in (minimal inhibitory concentrations) MICs among Actinomyces species, with A. turicensis being one of the most resistant. A. turicensis strains may show resistance to clindamycin, tetracyclines (doxycycline and tetracycline), macrolides (clarithromycin and erythromycin), ciprofloxacin, and/or linezolid [4]. High doses of penicillin must be used and therapy prolonged for up to 6 weeks or longer before any response can be seen. The initial treatment course is usually followed with oral penicillin suppressive therapy for 6 to 12 months.

**Conclusions**

We present a rare case of *Actinomyces turicensis* empyema where the pathogen was identified on positive blood cultures. The difficulty in accurately identifying this bacterium is considered to be a major reason for the small number of reported cases. In the correct clinical setting, infection with Actinomyces should be considered and blood cultures may be useful to establish the diagnosis [9].

**References**


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