CLINICAL VIGNETTE

Evaluation of a complex lung mass

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Case Presentation

A 46-year-old male without significant past medical history presents with 2 months of persistent dry cough associated with fatigue, intermittent night sweats, and an approximate 20 pound weight loss. The patient initially developed intermittent fever and dry cough that resolved within 1 week. These symptoms were initially attributed to an upper respiratory tract infection. He denied ongoing fevers, chest pain, shortness of breath, or hemoptysis.

The patient is a lifelong non-smoker and has no history of alcohol or drug use. He is a university professor who grew up in the U.S. and previously worked in the U.K. He traveled to developing nations for academic conferences, but he never resided in an endemic tuberculosis region and denied any contact with individuals suspected of having tuberculosis. The patient does not have any pets and denied recent travel to caves, estuaries, or local deserts.

Physical exam was notable for a thin, cachectic male (BMI 18), without other remarkable findings. There was no cervical, supraclavicular, or axillary adenopathy. The lung exam was normal.

Laboratory evaluation was notable for anemia (hemoglobin 9.7 g/dL) and thrombocytosis (platelet count 530 x 10^3). Blood bacterial and fungal cultures, as well microbiologic and auto-immune serologies, were obtained.

Chest X-ray revealed a large 9 cm opacity overlying the posterior right upper lobe (Image 1).

Chest CT showed complex solid and cystic mass-like consolidation within the right upper lobe with peribronchial and peribronchiolar thickening of the adjacent and subtending airways with the mass abutting the apical posterior pleura, deforming the adjacent superior right major fissure and extending into the right lower lobe. There was associated interlobar lymphadenopathy (Image 2). Additional imaging, including CT of the brain and neck, were normal.

The patient was initially admitted to airborne isolation and ruled out for infectious tuberculosis based on 3 sputum samples that were negative for acid fast bacilli (AFB). After ruling out infectious tuberculosis, he underwent CT guided biopsy based on the location of the complex mass with the final pathology showing acute and chronic inflamed granulation and fibrous tissue with special stains (AFB, GMS, Gram) negative for organisms.

Because of ongoing concern for malignancy and potential endobronchial pathology, the patient underwent video bronchoscopy with bronchoalveolar lavage (BAL) and transbronchial biopsy. There were no endobronchial lesions, and BAL fluid cytology showed neutrophil predominant acute inflammation, which was negative for malignant cells. The trans-bronchial biopsies showed acute and chronic inflamed granulation and scar tissue in an organizing pneumonia pattern. Special stains (AFB, GMS, CMV) were again negative for organisms.

All microbiology tests were negative except for one anaerobic culture obtained via CT guided biopsy, which grew Streptococcus anginosus group (also known as S. milleri group), and the patient was started on antibiotics (ertapenem). Additional studies including fungal serologies, viral PCR, and HIV antibody tests were all negative. Serum immunoglobulins were within normal limits, and MTb quantiferon gold was indeterminate. Thoracic surgery consultation was obtained for consideration for surgical biopsy.

Thoracic surgery felt that surgical resection would be complex based on the location of the mass and may require right upper lobectomy and possibly pneumonectomy.

Given the positive culture and clinical presentation, the working diagnosis was atypical presentation of lung abscess with primary bronchogenic carcinoma being less likely. The patient was discharged home for at least 4 weeks of intravenous ertapenem therapy.

CT chest after 2 weeks of antibiotic treatment showed near complete resolution of consolidation within the right upper lobe, largely replaced by cavitation. There was also interval decrease in the reactive adenopathy (Image 3). Follow-up chest X-ray performed 15 months after initial presentation showed a persistent right apical cavity with pleural and parenchymal scarring but no new masses (Image 4).

Discussion

In the modern antibiotic era, lung abscess is most commonly a complication of aspiration pneumonia in individuals prone to aspiration due to impaired consciousness. So called “secondary” lung abscesses may arise in the context of pre-

existing conditions such as bronchogenic carcinoma or immunocompromised states. Most lung abscesses are caused by anaerobic bacteria that reside in the oral cavity and gingival crevices that are then aspirated into the lung causing pneumonitis and eventual tissue necrosis.[1] Other bacteria can produce lung abscesses, notably Staphylococcus aureus and Klebsiella pneumonia; they usually present acutely with fulminant disease, as well as more indolent pathogens such as Nocardia and Actinomyces, that may develop in immunocompromised individuals.

The only identified pathogen in this case, S. Anginosus group, is facultative anaerobes that are part of the normal flora of the oral cavity and gastrointestinal tract. They are known for the ability to be pathogenic and cause abscess formation especially in individuals with dental and periodontal disease.[2] In addition, S. anginosus infections are able to cross tissue planes in the lung, including the interlobar fissure, diaphragm, and chest wall.[3]

Though a clear history of an aspiration event, systemic infection or oral/periodontal disease was not evident in this case, an aspiration event was likely the initial insult. Fiberoptic bronchoscopy was indicated to exclude endobronchial lesions, including bronchogenic carcinoma and foreign body aspiration, especially in this case with atypical presentation.[4]

Antibiotics are the current standard of care for the treatment of the uncomplicated lung abscess. Clindamycin, beta lactams with beta-lactamase inhibitors, or carbapenems are the regimens of choice. Duration of antibiotic treatment remains controversial but typically is on the order of weeks, based on clinical response. The vast majority of patients with primary lung abscess are cured with antibiotic therapy alone.

Images

Image 1. Initial Chest X-ray.

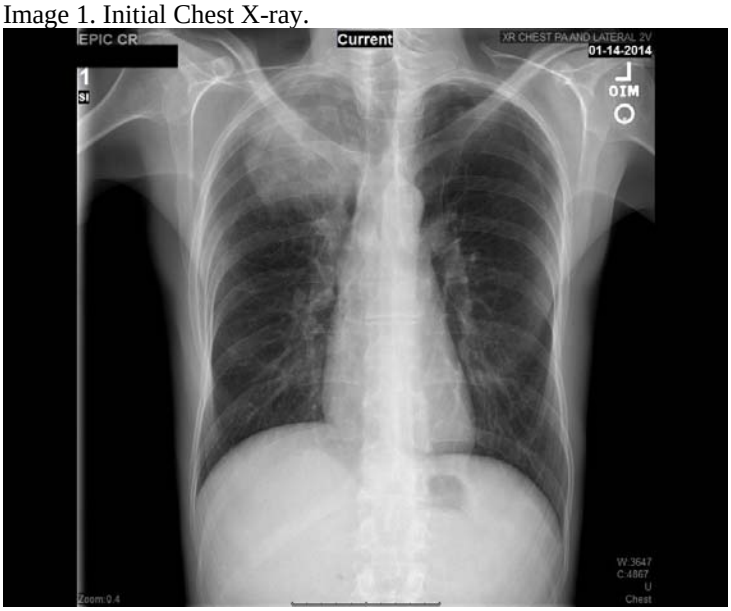


Image 2. Initial Chest CT.

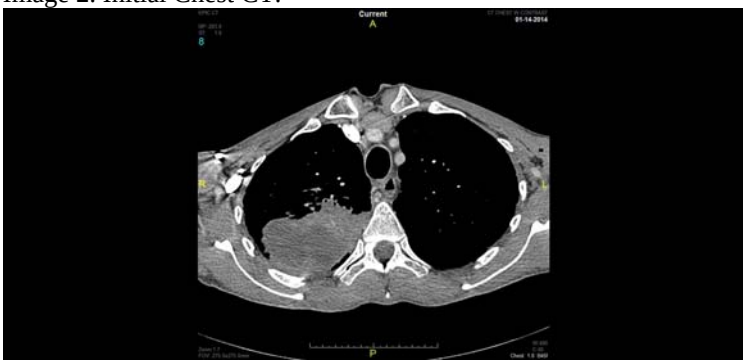
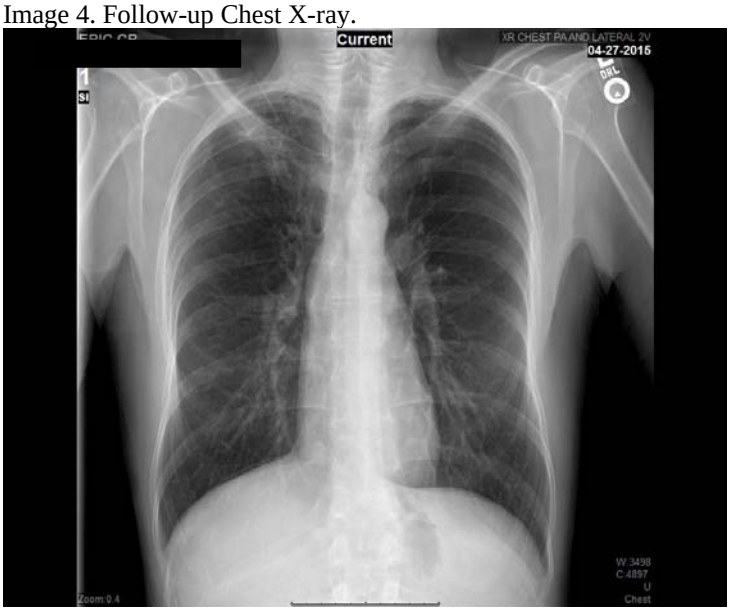


Image 3. Follow-up Chest CT.

Image 4. Follow-up Chest X-ray.



REFERENCES

1. **Chung G, Goetz MB**. Anaerobic Infections of the Lung. *Curr Infect Dis Rep*. 2000 Jun;2(3):238-244. PubMed PMID: 11095862.
2. **Porta G, Rodríguez-Carballeira M, Gómez L, Salavert M, Freixas N, Xercavins M, Garau J**. Thoracic infection caused by Streptococcus milleri. *Eur Respir J*. 1998 Aug;12(2):357-62. PubMed PMID: 9727785.
3. **Sunwoo BY, Miller WT Jr**. Streptococcus anginosus infections: crossing tissue planes. *Chest*. 2014 Oct;146(4):e121-5. doi: 10.1378/chest.13-2791. PubMed PMID:25288003.

4. **Sosenko A, Glassroth J**. Fiberoptic bronchoscopy in the evaluation of lung abscesses. *Chest*. 1985 Apr;87(4):489-94. PubMed PMID: 3979136.

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