



roentgenogram of the month

A 48-Year-Old Smoker With Cough and Weight Loss*

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A 48-year-old male smoker was referred for the evaluation of symptoms of cough, weight-loss, and chest radiograph abnormality. The patient had a medical history significant for chronic bronchitis and had been seen by his primary care physician 2

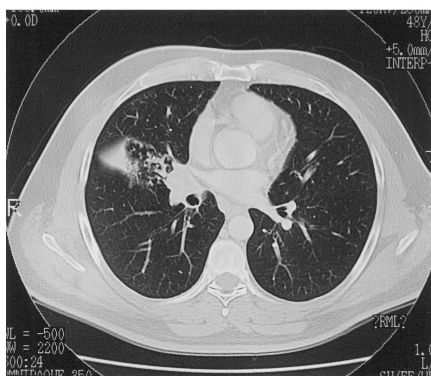


FIGURE 1. Chest radiograph (top) and CT scan (bottom) demonstrating right middle-lobe infiltrate and possible hilar mass.

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months previously for worsening nonproductive cough without fever or shortness of breath. He was treated with erythromycin after chest radiography demonstrated a right middle-lobe infiltrate, while the results of a sputum culture revealed "normal flora." The patient's cough continued over the following month, complicated by gradually worsening dyspnea on exertion and weight loss, and he returned to the clinic. On examination, the patient was afebrile and had normal room air oximetry findings, but had persistent bronchial breath sounds in the right mid-chest. The results of serum chemistry tests and CBC were in the normal range. A repeat chest radiograph and a chest CT scan were performed (Fig 1). Fiberoptic bronchoscopy revealed an erythematous, narrowed right middle-lobe orifice with extruding tan purulence. The results of an endobronchial biopsy demonstrated only inflammatory changes, while the results of an acid-fast bacillus stain of endobronchial washings were negative. A Papanicolaou stain of the washings is shown in Figure 2.

What is the diagnosis?

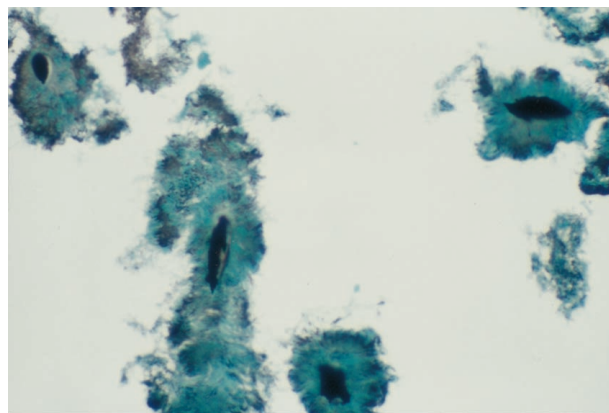


FIGURE 2. Papanicolaou stain of endobronchial washings.

Diagnosis: Thoracic actinomycosis

Initially described by Bollinger in the 1870s as “wooden tongue” disease, a suppurative swelling of the jaw seen in cattle, clinical disease caused by *Actinomyces* species has been recognized for over a century. Human illness caused by *Actinomyces* species was first documented in 1891, but it was not until the 1950s that *Actinomyces* species were recognized to be bacteria rather than fungi, as originally believed.¹ Since that time, several organisms causing illness in humans have been identified within the genus *Actinomyces*, including *Actinomyces israelii* (the most common), *A naeslundii*, *A viscosus*, *A odontolyticus*, *A meyeri*, and *A gerencseriae*.² *Actinomyces* species are of low human pathogenicity and are, in fact, commensal to the oropharynx, GI tract, and female genital tract, having no external environmental reservoir.² Typically characterized by chronic, localized, suppurative lesions with abscess formation, tissue fibrosis, and draining sinuses, actinomycosis tends to spread slowly to contiguous structures without respecting tissue planes and without regional lymphadenopathy. Affected tissues become indurated and produce pus containing 1 to 2 mm of yellow-brown granules, described as “sulfur granules,” composed of *Actinomyces* colonies that can be partially calcified.¹ Prominent sites of infection include the jaw, lung, ileocecal junction, and pelvis. There is hematogenous spread with metastatic disease, usually to the CNS or liver, in < 10% of cases.³

Thoracic disease accounts for 15 to 50% of actinomycosis cases.⁴ It has many similarities to lung abscess and other anaerobic infections. Typically having a long-term course with symptoms that include nonproductive cough, dyspnea, chest pain, hemoptysis, weight loss, and low-grade fever, actinomycosis also may present as a lung mass or even as superior vena cava syndrome. Those persons affected tend to be in the fourth to seventh decade of life and may manifest some degree of abnormal host defenses, with chronic bronchitis, bronchiectasis, and emphysema commonly appearing as comorbidities.⁴ Interestingly, thoracic disease is rarely associated with obvious cervicofacial foci of actinomycosis. While it is often associated with dental caries, in one series 50% of patients were in fact edentulous.⁵ Pulmonary disease is believed to be secondary to aspiration of oropharyngeal or gastric material in the vast majority of cases and, not surprisingly, tends to involve the lower segments, particularly of the right lung.¹ After initially producing pneumonia, infection may spread across the lung fissures and produce cavitation, pleural thickening, and empyema. Chronic airspace infection may lead to lung

fibrosis, while endobronchial obstruction may occur due to airway edema, compression, or formation of an endobronchial mass with or without an initiating foreign body. On a radiograph, actinomycosis may easily be mistaken for tuberculosis, cancer, or non-specific pneumonia.² Disease may also include chest wall invasion, with bony erosion, and draining cutaneous fistulas.

Unfortunately, misdiagnosis and delay in diagnosis are extremely common in *Actinomyces* infection. In the series of 181 cases by Brown,³ actinomycosis was diagnosed just 19 times on admission, with the majority of patients having a delay in diagnosis ranging from 1 month to 2 years. In addition to the aforementioned clinical and radiographic mimicry, *Actinomyces* species are notoriously slow to grow in culture, and overgrowth by accompanying bacteria is a major problem.¹ Thus, the laboratory must be notified of the suspicion of *Actinomyces* infection to increase the odds of a successful culture. When collecting and examining a specimen, granules should be sought and, if present, should be crushed between two slides for Gram's stain.¹ The round or doughnut-shaped granules, which are 1 to 2 mm in size, may appear to be basophilic or amphophilic, with eosinophilic clubs on the surface. A central core of branching, thin (< 1 μ m), Gram-positive filamentous rods⁶ strongly supports the diagnosis of actinomycosis and may allow diagnosis despite an unsuccessful culture.^{1,2} Granules are not, however, invariable or pathognomic, as occurs in *Nocardia* and other chronic infections.²

Two goals govern the treatment of *Actinomyces* infections: the prevention of occult metastatic disease and the prevention of relapse.⁴ Therefore, early and prolonged (6- to 12-month) antimicrobial therapy is essential.² Antibiotics for this organism include penicillin, chloramphenicol, tetracyclines, erythromycin, clindamycin, imipenem, streptomycin, or a cephalosporin. Fluoroquinolones, aztreonam, fosfomycin, and aminoglycosides have poor activity against these organisms and should not be used.² Treatment of secondary organisms identified concurrently with *Actinomyces* species as part of a polymicrobial flora is not required.² Therapy for severe infections of the chest, abdomen, pelvis, and CNS may require a combined medical/surgical approach, with resection of necrotic tissue, excision of sinus tracts, drainage of empyemas or abscesses, and curettage of infected bone. While the prognosis of this disease prior to the antibiotic era was poor, with 75 to 100% mortality rate, current therapy with antibiotics and surgical resection, when indicated, has improved outcomes for actinomycosis tremendously,⁴ as our patient demonstrates (Fig 3). However, the morbidity and mortality rates surrounding

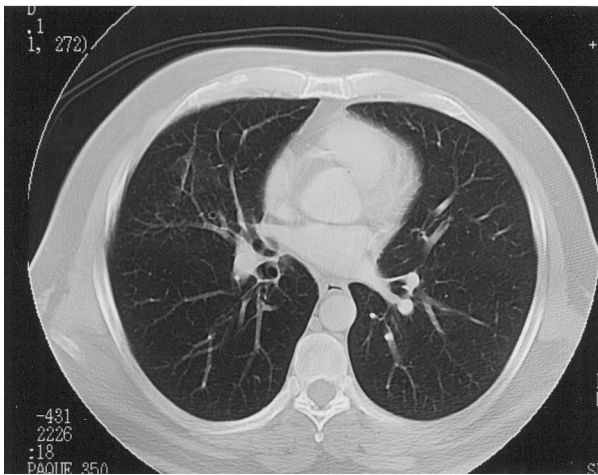
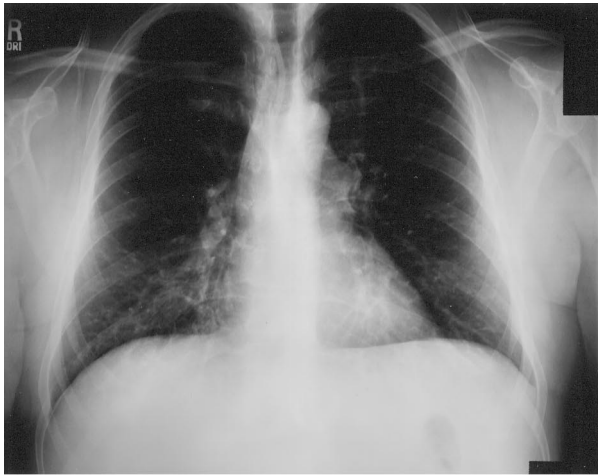


FIGURE 3. Chest radiograph (*top*) and CT (*bottom*) demonstrating resolution of right middle-lobe and hilar changes following 3 months of antibiotic therapy.

misdiagnosis of this illness continue to be substantial and merit constant vigilance.

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