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A thoracic mass infiltrating the chest wall

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Abstract. – A case of thoracic mass infiltrating the chest wall mimicking a pulmonary invasive neoplasm is reported. Differential diagnosis and characteristic radiological and histological imaging had a decisive role in the case management. Actinomycosis is caused by a gram-positive, filamentous, microaerophilic bacteria. About 15% of the infections caused by Actinomyces involve the thorax. If not promptly diagnosed and treated thoracic actinomycosis may determine contiguous and systemic involvement. Actinomycosis is an anaerobic-to-microaerophilic bacteria and direct identification and isolation are difficult to obtain. In depth discussion diagnostic and therapeutic features are described in this report.

Key Words:

Actinomycosis, Thoracic, Pulmonary, Infection.

Case History

A 54 years old man, current smoker of 20 cigarettes per day for 30 years (30 pack/yrs), with a medical history of chronic obstructive pulmonary disease (COPD), hypertension, diabetes mellitus, and hypercholesterolemia, was admitted to our Respiratory Disease Unit because of poorly productive cough in the last two months, chest pain, lack of appetite, and weight loss (5 kg in 3 months), without dyspnoea and fever. Patient reported pyorrhea in the last 2 years with no recent surgery.

Investigations showed a neutrophil leukocytosis (total white cells count 12.41 · $10^3/\mu$l), with anaemia (Hb 9,20 g/dl, RBC 3,65 million/μl), thrombocytosis (PLT 833 · $10^3/\mu$l), increased erythrocyte sedimentation rate (ESR) (112 mm/h), and protein electrophoresis with hypoalbuminemia, increased alfa-2 proteins and gamma band.

Chest X-ray showed a pulmonary infiltrate in the left lower lobe with an accentuation of ipsilateral hilum (Figure 1).

Chest Computed Tomography (CT) revealed an extensive infiltrate in the apical segment of left lower lobe that extends to the hilum. The infiltrate adheres to the pleura in the posterior-medial side, whereas an irregular streak is connected to the pleura, and the mass infiltrates the chest wall in the left paravertebral region. Left pulmonary artery is in close contact with the infiltrate. Hilar adenopathy is observable.

Thoracic palpation revealed a scarcely movable and painful subcutaneous swelling in the left paravertebral area (3 cm).

Fiberoptic bronchoscopy showed a thickened mucosa in the apical segment of the left lower lobe. Chronic inflammatory infiltration was observed in biopsies and bronchoalveolar lavage (BAL).

CT scanning and histological exam (Gram stain) by fine needle aspiration biopsy (FNAB) is observable in the Figures 2 and 3.

Histological exam (Gram stain) showed the presence of chronic inflammatory cells, connective-tissue proliferation, and the pathognomonic sulfure granule. The exam is diagnostic for an invasive thoracic actinomycosis.

Our patient started an i.v. penicillin for 6 weeks followed by oral penicillin for 4 months. Chest X-ray after 6 months showed the resolution of pulmonary infiltrate (Figure 4).

Discussion

Actinomycosis is a chronic granulomatous disease caused by a gram-positive, non acid-fast, filamentous, microaerophilic bacteria. About 15% of the infections caused by *Actinomyces* involve the thorax. Actinomycosis most likely results from the aspiration of organisms from the

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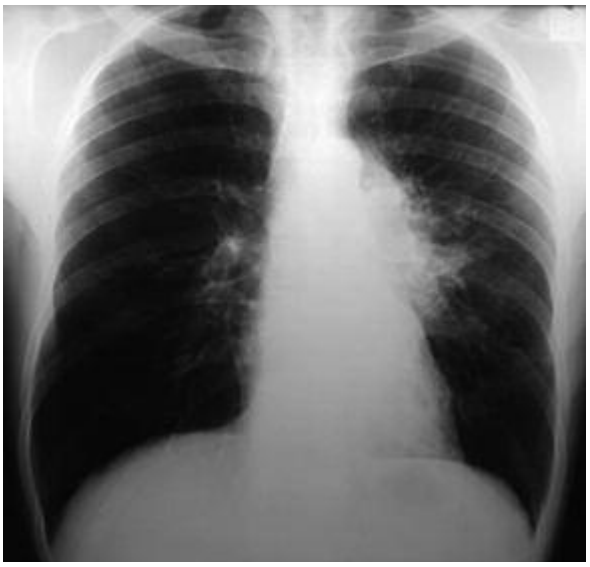

Figure 1. Chest X-ray: pulmonary infiltrate in the left lung and prominence of ipsilateral hilum.

oropharyngeal cavity or bronchial aspiration from the gastrointestinal (GI) tract. In fact, actinomycetes are conspicuous among the normal flora of the oral cavity and in the lower GI tract, less frequent in the female genital tract[1-2]. Symptoms and signs of thoracic actinomycosis include chest pain, dry or productive cough, hemoptysis, fever, weight loss, and shortness of breath[3].

Laboratory exams usually show anaemia and mild leukocytosis. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are elevated, whereas serum proteins show a typical sight of chronic inflammation (hypoalbuminaemia, increased alfa-2 proteins and gamma band).

Predisposing factors are poor oral hygiene, smoke, alcoholism, immunosuppression, and events with loss of consciousness[2]. Differential diagnosis includes lung abscess, lung cancer, lymphoma, nocardiosis, aspiration pneumonia, tuberculosis.

If not promptly diagnosed and treated thoracic actinomycosis determines pulmonary lesions extending through the chest wall, pleurocutaneous fistula, destruction of ribs contiguous with chronic pulmonary involvement, adjacent lobe involvement, and characteristic destruction of the vertebrae[4].

Actinomycosis is characterized by contiguous spread, suppurative and granulomatous inflammation, formation of multiple abscesses and sinus tracts that may discharge sulfur granules. Hematogenous dissemination to distant organs may occur. The most common clinical forms of actinomycosis are cervicofacial (55%), thoracic (15%), abdominal and pelvic in women (20%), and rarely (10%) in skin, brain and limbs[5,6].

Thoracic actinomycosis is most common in men than in women (M/F 3:1).

Actinomycetes grow slowly in anaerobic-to-microaerophilic conditions, and the most common isolated species is *Actinomyces (A.) israelii*. *A. meyeri, A. viscosus, A. pyogenes, A. odontolyticus, A. naeslundii*, and *A. propionicus* are less recurrent[7].

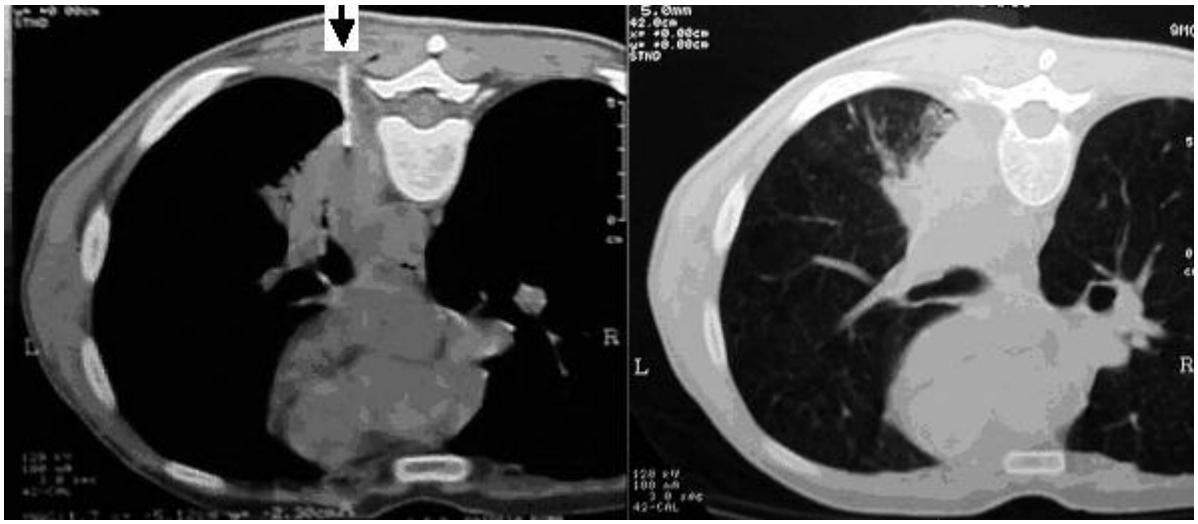

Figure 2. CT scanning during fine needle aspiration biopsy. Arrow points out the needle into the mass.

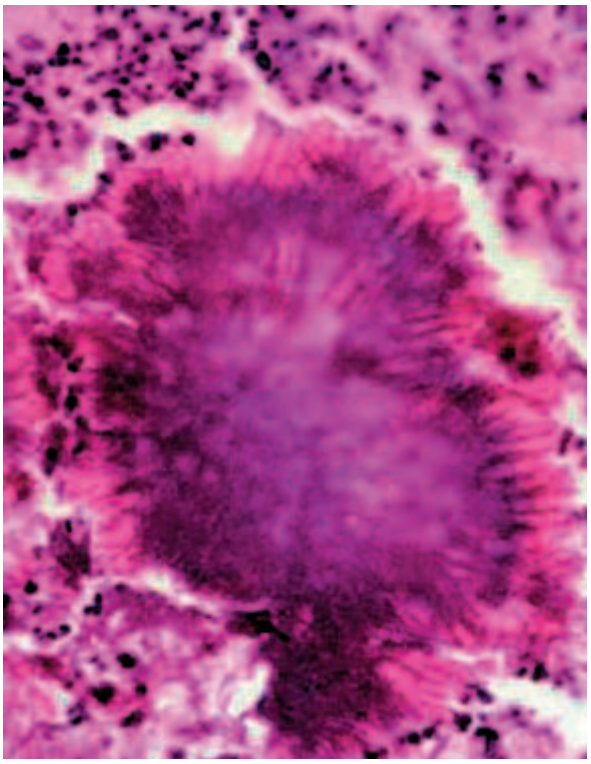

Figure 3. Histological exam with Gram stain. Microcolonies of Actinomyces with radially arranged, intertwined branching filaments, surrounded by granulomatous infiltration. Magnification × 100.

Actinomycosis is characterized by suppurative and granulomatous inflammatory reactions with the presence of sulfur granules. The sulfur granules are nearly pathognomonic for actinomycosis, although similar findings have been reported in infections by *Nocardia*, *Streptomyces*, and *Botryomycosis*. The granules are 0.1-1 mm in diameter and may be observed as yellow particles. Gram stain makes these microcolonies visible as gram-positive, intertwined branching filaments, with radially arranged, peripheral hyphae[7].

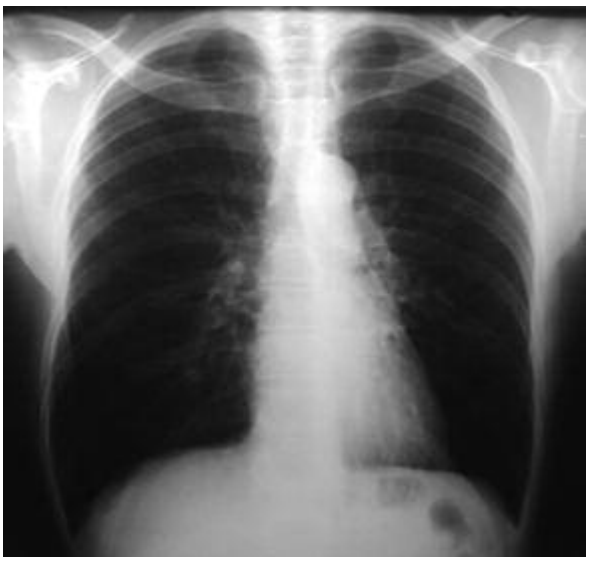

Figure 4. Chest X-ray after 6 months: resolution of pulmonary infiltrate.

Frequently other bacteria, gram-positive and gram-negative, may coexist with actinomyces. Polimicrobic infections are observed in the 65% of actinomycosis cases, remarking the synergic role of co-pathogens for the microaerophilia. In fact, co-pathogens appear to magnify the low pathogenic potential of actinomycetes[8,9].

In our case histological exam was colture-negative, with absence of malignant cells. Sulfur granules appear yellow when observed grossly and are actually colonies of Actinomyces composed of radiating, club-shaped filaments of organisms, surrounded by an area of granulomatous reaction. It is almost certainly diagnostic for an invasive thoracic actinomycosis[7].

Actinomyces is an anaerobic-to-microaerophilic bacteria and direct identification and isolation are difficult to obtain. Acceptable specimen are obtained from draining sinuses, deep needle aspirate, or biopsy specimens, whereas sputum specimens are inappropriate being actinomyces a normal oral commensal[8,9]. An anaerobic transport device to the microbiology laboratory is necessary for most favourable isolation of actinomycetes[7].

Actinomycosis infection in human is well known since the 19th century. In the preantibiotic era such infections were severe and fatal. Currently, the real incidence of actinomycosis is difficult to define because many cases are eradicated by the common use of antibiotics[10].

Antimicrobial therapy is the only treatment required in the majority of actinomycosis, although surgery can be necessary in selected cases (diagnostic procedures and complications such as empyema, abscesses, or hemoptysis).

Actinomycetes have a predictable susceptibility to antibiotics. The regimen of choice is i.v. penicillin for at least 6 weeks, followed by oral penicillin for at least 3 months after symptoms disappear.

Penicillin G is the drug of choice administered over a prolonged period (6 months to 1 year). It interferes with synthesis of cell wall mucopeptide during active multiplication, resulting in bactericidal activity[10]. However, the

treatment duration should be personalized to the patient based on clinical and radiographic response. Therefore, traditional recommendation of i.v. antibiotic therapy for 2 to 6 weeks followed by oral antibiotic therapy for 6 to 12 months is not always necessary for all thoracic actinomycosis patients[11].

Alternatives to penicillins may include cephalosporins, doxycycline, erythromycin, clindamycin, co-trimoxazole, aminoglycosides, and fluoroquinolones.

In conclusion, thoracic masses infiltrating the chest wall are almost all neoplastic diseases, but an accurate clinical exam and a thorough differential diagnosis permit to diagnose and to treat infectious diseases with a good prognosis. Adequate specimens with invasive manoeuvres and suitable isolation with anaerobic transport device are essential in thoracic actinomycosis.

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