

An African woman with pulmonary cavities: TB or not TB?

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ABSTRACT

Cavitary lung lesions in patients from developing countries are mostly caused by tuberculosis (TB). However, when TB cannot be confirmed, a primary lung abscess caused by anaerobic bacteria from the mouth should be considered, especially in patients with poor dentition. We present a case of a Sudanese woman with a cavitary lung lesion and severe gingivitis. *Bulleidia extracta* was isolated as a single pathogen from the pulmonary cavity.

KEYWORDS

Anaerobic bacteria, lung abscess, periodontitis

INTRODUCTION

Cavitary lung lesions can be caused by a broad range of necrotising infections and non-infectious diseases. In immigrants from Third World countries tuberculosis (TB) is the most likely cause. If TB cannot be confirmed, other causes should be considered. We present a case of a Sudanese woman with multiple pulmonary cavities negative for *Mycobacterium tuberculosis*. *Bulleidia extracta*, an anaerobic rod found in gingivitis was isolated as the causative agent in a primary lung abscess.

CASE REPORT

A 56-year-old HIV-negative Sudanese woman presented with a persistent productive cough and fever, four

What was known on this topic?

A pulmonary cavity is a gas-filled area of the lung in the centre of a nodule or area of consolidation. It may be detected by plain chest radiography or computed tomography. Cavitary lung lesions are frequent manifestations of a wide variety of infectious and non-infectious processes involving the lung. In patients originating from TB-endemic countries, TB is the most likely cause.

What does this add?

We show, for the first time, that the anaerobic rod *Bulleidia extracta* was isolated as the causative agent in a primary lung abscess. Clinicians should be aware that a cavitary lung lesion in a patient from a TB-endemic country is not always caused by TB. Poor dentition should raise suspicion of a lung abscess caused by oral anaerobic bacteria. The treatment duration should be long enough.

years after coming to the Netherlands. Elsewhere, chest radiography and computed tomography (CT) showed an infiltrate with a cavitary lesion in the left upper lobe. Bronchoscopy was normal, the tuberculin skin test was negative and repeated examinations of sputum and bronchoalveolar lavage were negative for *Mycobacterium tuberculosis* (microscopy, culture, and PCR). CT-guided transthoracic aspirate showed chronic, non-specific inflammation and again, the cultures were

negative. Amoxicillin-clavulanic acid for ten days led to a temporary improvement of the symptoms. Three months later she returned with fever, cough, and haemoptysis. The CT scan showed progression of the cavitary lesion (figure 1). Even though sputum cultures were negative, ciprofloxacin was given for ten days. She did not improve and was referred to our hospital where she reported progressive cough, haemoptysis and weight loss. She reported a tooth extraction two months before the start of her symptoms. In the last six months, her daughter had noticed an extremely putrid smell on her mother's breath. On physical examination she was febrile (38.5 °C) with very poor dentition, periodontitis, and a prominent fetor ex ore. Laboratory investigations showed anaemia and mild leukocytosis. Chest radiography and CT scan showed extensive consolidations in the left upper lobe, with multiple cavities filled with fluid and air. Orthopantomography showed extensive general periodontitis with periapical radiolucencies of multiple teeth (figure 2).

Bronchoscopy showed massive secretion from the left upper lobe, with large amounts of a single species of

gram-positive anaerobic rods cultured. Using 16S PCR analysis this was identified as *Bulleidia extracta*, a pathogen until now only described in periodontitis, and recently in a total hip arthroplasty infection.^{1,3} The patient underwent extraction of the diseased teeth and was treated with 600 mg clindamycin orally three times a day for two months, after which she showed complete clinical and radiological recovery.

DISCUSSION

Primary lung abscesses are usually caused by aspiration of anaerobic bacteria present in the gingival crevices and dental pockets,⁴ sometimes associated with altered consciousness (e.g. alcoholism), dysphagia, oesophageal disease or recent tooth extractions. Patients commonly have poor dentition with periodontitis, resulting in an unusually high load of oral anaerobic organisms. Lung abscesses are rare in edentulous patients, in which case airway obstruction (e.g. bronchogenic carcinoma) should be suspected. Aspiration most frequently occurs in the supine patient and therefore lung abscesses mostly occur in the posterior segment of the right and left upper lobe.

Patients generally present with slowly progressive symptoms of fever, productive cough, malaise and weight loss, and sometimes haemoptysis. Rigors are rare. Many patients and their close contacts complain of a putrid smell on the patient's breath. Physical examination frequently shows gingival crevice disease with dental pockets and other signs of periodontitis; lung auscultation may be abnormal. Anaemia of chronic disease and leucocytosis are usually present. Chest radiography typically shows a cavitary lesion with an air-fluid level, and computed tomography should exclude an associated obstructing endobronchial lesion.

The most frequently isolated anaerobes are *Peptostreptococcus* spp., *Fusobacterium nucleatum* and *Prevotella melaninogenica*.⁵ Cultures usually show multiple anaerobic species, and microaerophilic streptococci and *S. milleri* in mixed infections. In a typical case, therapy may be initiated without microbiological diagnosis. Isolation of the causative pathogens is difficult since sputum or bronchoscopy aspirates are often contaminated by upper airway flora.

Historically penicillin was the treatment of choice for anaerobic lung abscess, but clindamycin is the preferred drug today. More anaerobes including *Prevotella* spp, *Bacteroides* spp (non fragilis) and *Fusobacteria* now produce penicillinase, and two trials demonstrated superiority of clindamycin compared with parenteral penicillin.^{6,7} Metronidazole often leads to failure due to the presence of aerobic and microaerophilic streptococci in mixed infections.

Figure 1. Computed tomography scan showing progression of the cavitary lesion



Figure 2. Orthopantomogram showing extensive general periodontitis with periapical radiolucencies of multiple teeth



No studies have evaluated optimum duration of treatment. Our patient was initially treated with amoxicillin-clavulanic acid for ten days, but patients should probably be treated for 6-8 weeks or more, until chest radiography has markedly improved or even normalised. Surgery is rarely indicated and bronchoscopic drainage should be reserved for patients with an obstructing lesion. Of course, the origin of the lung infection needs causative treatment, in this case complete extraction of the diseased dentition. In conclusion, cavitory lung lesions in a patient from a TB-endemic country are not always caused by TB. Poor dentition should raise suspicion of a lung abscess caused by oral anaerobic bacteria; the treatment duration should be long enough.

DISCLOSURES

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