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CASE REPORT

Endobronchial actinomycosis

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Endobronchial actinomycosis. K. Dalhoff, S. Wallner, C. Finck, S. Gatermann, K.J. Wießmann. ©ERS Journals Ltd 1994.

ABSTRACT: Endobronchial actinomycosis was found to be the cause of right-sided atelectasis and haemoptysis in a 57 year old man without predisposing conditions.

Fibreoptic bronchoscopy revealed occlusion of the intermediate bronchus by yellow-white masses. The diagnosis was confirmed histologically and by positive *Actinomyces* culture from bioptic material.

Prolonged antibiotic treatment resulted in complete recovery, without need for surgical resection.

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Actinomycosis is a chronic suppurative infection, which infiltrates mucosa-associated tissues [1]. *Actinomyces* spp. are gram-positive, anaerobic micro-organisms, belonging to the resident flora of the oropharynx and gastrointestinal tract and found in 30–50% of normal saliva specimens [2]. Interruption of the mucosal barrier by local inflammation, traumatic or surgical injury is regarded as a predisposing factor for tissue infection [2]. Most cases of clinical actinomycosis are of polymicrobial aetiology, since the invasiveness of *Actinomyces* spp. is greatly enhanced by synergistic interaction with "associates", such as Staphylococci, Streptococci and other anaerobic bacteria [1, 2].

Cervicofacial infection is the most frequent manifestation, and thoracic actinomycosis accounts for approximately 20% of cases. Abdominal and pelvic manifestations are less frequently observed [3, 4]. Pulmonary actinomycosis is acquired mainly through aspiration of the organism from the oropharynx, although infection *via* inhalation, haematogenous dissemination and direct extension from adjacent tissues may occur [1]. The classical presentation of this disease consists of thoracic mass lesions spreading unimpeded by anatomical barriers, rib destruction and pleural empyema; but nonspecific symptoms suggesting malignancy or tuberculosis have been more common in recent series [5]. Thus, the correct diagnosis is frequently missed, until surgical resection is performed because of presumed bronchogenic carcinoma [6, 7].

We observed an unusual presentation of thoracic actinomycosis, with haemoptysis and an endobronchial tumour causing right-sided atelectasis. The aetiological diagnosis was confirmed by fibreoptic bronchoscopy.

Case history

A 57 year old nonsmoking man was admitted to the hospital because of bradyarrhythmia with dyspnoea, faintness and confusion. He had a 2 year history of right-sided thoracic pain and haemoptysis, without generalized symptoms or body weight loss. There was no history of recurrent infection or other evidence of immunodeficiency; and alcohol or drug abuse was denied. Temperature at admission was 36.8°C. On physical examination, the dental status was deficient without mucosal inflammation in the oral cavity, heart rate was 42 beats·min^{-1}, and blood pressure 130/85 mmHg. Lung auscultation revealed diminished breath sounds at the right lower base.

Laboratory tests revealed an elevated erythrocyte sedimentation rate (ESR) of 21 mm in the first hour, a C-reactive protein of 6 mg·l^{-1}, and normal white and red cell count. Differential cell count disclosed the following values: neutrophils 52%; lymphocytes 34%; monocytes 7%; eosinophils 1%. Lymphocyte subsets were in the normal range including the absolute

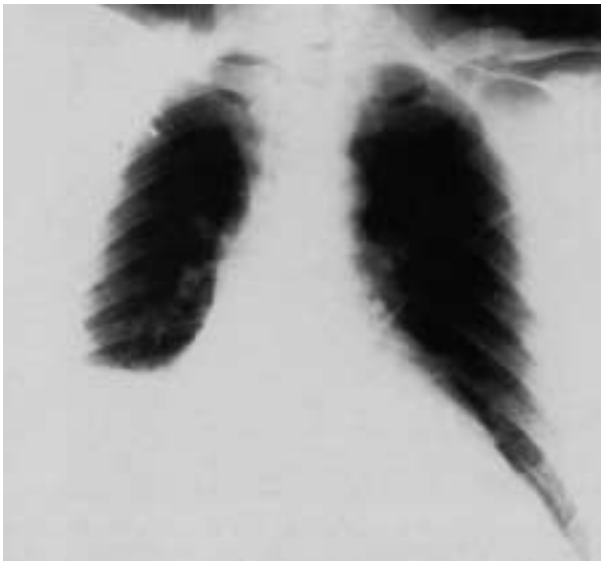

Fig. 1. – Posteroanterior chest radiograph at admission, showing atelectasis of the right lower lobe with ipsilateral tracheal deviation.

CD4-lymphocyte count, and the biochemical profile, including quantitative immunoglobulins, was normal. Human immunodeficiency virus (HIV)-serology and tuberculin skin-testing were negative.

Chest radiograph showed atelectasis of the right lower lobe, without a clearly distinguishable mass lesion (fig. 1).

After excluding myocardial infarction and significant coronary heart disease, fibreoptic bronchoscopy was performed under the presumptive diagnosis of bronchogenic carcinoma or tuberculosis.

Bronchoscopy showed subtotal occlusion of the right intermediate bronchus by a yellow-white mass, which was surrounded by severely inflamed and oedematous bronchial mucosa (fig. 2a). After biopsy and removal of the stony hard material, profuse bleeding occurred. Control examinations demonstrated multiple fragments of the endobronchial mass and white spots in the inflamed mucosa (fig. 2b); however, no endobronchial tumour was visible in the reopened segments of the right middle and lower lobe.

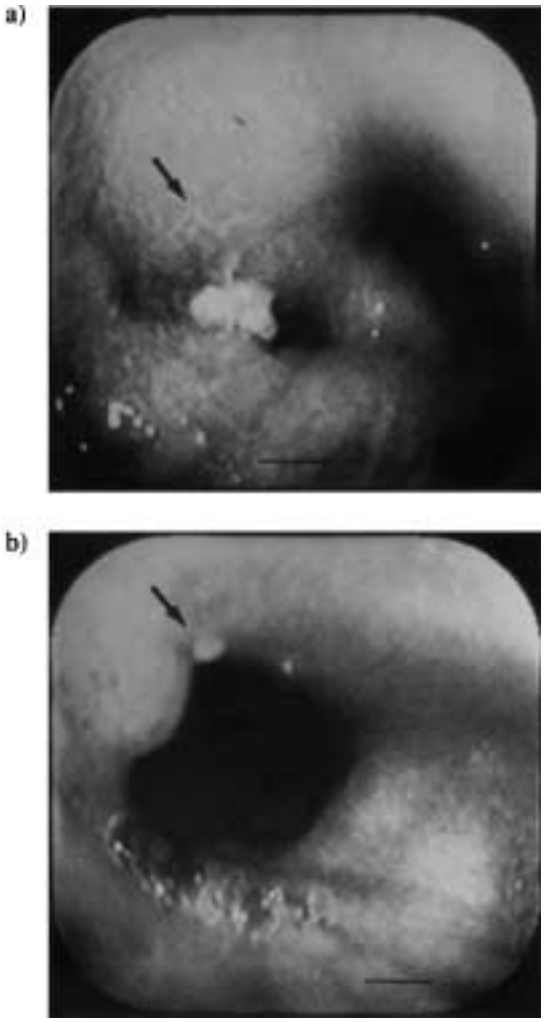

Fig. 2. – a) Subtotal occlusion of the intermediate bronchus by stony hard, yellow-white material (arrow) and marked oedema of surrounding bronchial mucosa. (Bar=4 mm). b) Endoscopic aspect of the intermediate bronchus after removal of endobronchial masses. White spots in inflamed bronchial mucosa (arrow). (Bar=4 mm).

Microscopic examination of specimens obtained by bronchoalveolar lavage (BAL) and bronchial suction showed neutrophilic inflammation (BAL differential: 70% alveolar macrophages, 23% neutrophils, 1.5% eosinophils and 5.5% lymphocytes). Quantitative cultures (including anaerobic cultures) of BAL yielded 10^5 colony forming units (cfu)·ml^{-1} mixed throat flora. Fungal and mycobacterial smears were negative.

Histological examination of the bronchial mucosa revealed large colonies of *Actinomyces* spp. ("sulphur granules") which were surrounded by necrotic masses and inflammatory cells (fig. 3). These infiltrates consisted of neutrophils, macrophages and some giant cells of the foreign body type.

Bioptic material was cultured as follows. Stony hard material obtained by fibreoptic bronchoscopy was washed twice with brain heart infusion (BHI) (Merck, Darmstadt, FRG), to remove loosely adhering microorganisms, and ground in 3 ml of BHI in an ultraturrax device. Samples of the broth were streaked onto various media and incubated aerobically and anaerobically for seven days. Identification of bacteria was performed according to standard conditions [8]. After the incubation period, *Actinomyces* spp. were identified. Further classification was not carried out due to insufficient growth on repeated subcultures.

Clinical course and outcome

Before receiving the final results of the morphological and microbiological work-up, combination chemotherapy for suspected poststenotic infection was instituted with cefotaxime (3×2 g·day^{-1}) and clindamycin (3×600 mg·day^{-1} *i.v.*). After becoming aware of the aetiology we re-evaluated the patient four weeks later. Since the clinical condition did not improve and since endoscopy revealed severe, persisting bronchial inflammation, the treatment was changed to a more pathogen-directed regimen with amoxycillin/clavulanate (3×1.2 g·day^{-1} *i.v.*) for another four weeks, resulting in complete clinical, radiological and endoscopic cure, including sterile cultures of bronchial mucosa. Lung function tests at the end of treatment revealed an increase in vital capacity (VC) (107 *vs* 95% pretreatment level), forced

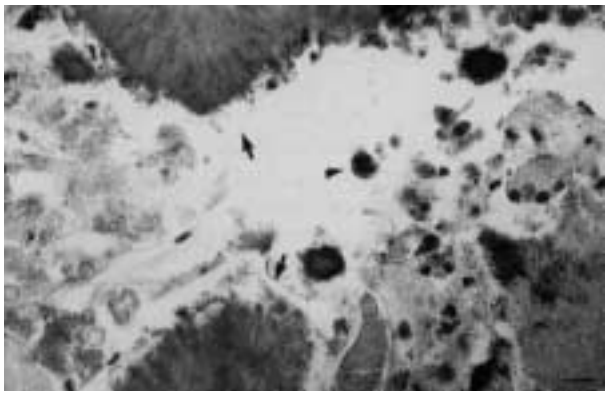

Fig. 3. – Periodic-acid-Schiff (PAS) -stained histological slide, showing *Actinomyces* colonies (arrows) surrounded by inflammatory cells, including a few giant cells (arrowhead). (Bar =15 µm).

expiratory volume in one second (FEV_1) (104 *vs* 88%) and arterial oxygen tension (Pa_{O_2}) (9.05 *vs* 7.63 kPa). Computed tomography of the thorax did not show any remaining abnormalities in the lung parenchyma. No clinical symptoms were observed during nine months of follow-up.

Discussion

Endobronchial actinomycosis is a rare cause of haemoptysis and a tumour occluding the large bronchi. To our knowledge, only four reports have been published in the last decades [9–12]. Since the onset of disease is mostly insidious, and signs of infection are frequently missing, the suspected diagnosis is usually bronchogenic carcinoma [5, 11].

The clinical characteristics of our case were similar to previous reports [2, 5–7, 13] regarding the absence of severe underlying diseases and the nonspecific presentation without fever or purulent sputum. Haemoptysis has been described in 22–53% of cases, thoracic pain in 35–68%, mainly in patients with pleural involvement, which was not documented in our case. A delayed diagnosis up to 44 months [7] from the beginning of symptoms is reported by all authors. As in our case, the correct diagnosis is nearly always missed on hospital admission.

Whereas in the past most cases of thoracic actinomycosis were diagnosed by thoracotomy [6, 7], fibreoptic evaluation offers the chance of a minimal invasive diagnostic and therapeutic management. Since the diagnosis may elude routine microscopy and culture, special requirements for successful *Actinomyces* culture are needed, which include avoidance of contamination, anaerobic conditions, and prolonged culturing [1]. Physiological saline, which is commonly used for BAL, inhibits the growth of pathogenic *Actinomyces* spp. [3]. Thus, in our patient, only the cultures of bioptic material, but not BAL and bronchial secretions, yielded the organism.

Little is known about predisposing host conditions and the role of the immune system in actinomycosis [1]. Poor dental hygiene, as found in our patient, seems to be a disease-promoting factor. Opportunistic infections in immunocompromised patients are rarely observed [12]. The inflammatory host response may play an additive role in the pathogenesis of clinical actinomycosis. A special feature in this context is the deposition of calcium/phosphate complexes in inflamed tissue by phosphatases of activated host cells [1], leading to the characteristic, stony hard granulation tissue.

The treatment of choice for infection with *Actinomyces* spp. is penicillin. Since the micro-organisms of the associate flora are not always susceptible to penicillin G, but may have an additional pathogenic role, some investigators favour aminopenicillins +/- clavulanic acid [2]. Clindamycin, tetracyclines and erythromycin are alternative drugs in the case of penicillin allergy [1]. Our patient, who was first treated with a combination of clindamycin and cefotaxime, showed improvement only after the treatment was changed to amoxycillin/ clavulanate. However, no definite conclusions can be drawn from this observation, since *in vitro* sensitivity data were not available, and a prolonged treatment period may be required for assessment of the therapeutic effect of any drug. In general, a treatment course of 3–12 months is recommended for pulmonary actinomycosis, considering the difficult penetration of antibiotics in areas of dense fibrosis [1]. Since no parenchymal involvement was observed in our case and since most of the infectious material could be removed fibreoptically, antibiotics were withdrawn after 2 months of therapy, without relapse during an observation period of 9 months. High cure rates of actinomycosis on appropriate medical treatment have been reported in all recent series, with a mortality of only 1 out of 48 cases [6, 7, 11, 13].

In conclusion, endobronchial actinomycosis is a rare cause of haemoptysis as well as endobronchial masses and atelectasis. A presumptive clinical diagnosis should be attempted, since special requirements for successful culturing are needed. Fibreoptic bronchoscopy is a useful tool for diagnosis and may avoid surgical procedures. In most cases, a pathogen-directed course of antibiotic therapy is effective and results in microbiological and clinical cure.

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