

Aspergillus tracheobronchitis, bronchopleural fistula and empyema after lobectomy for aspergilloma



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ABSTRACT

Aspergillus tracheobronchitis and *Aspergillus empyema* are two rare manifestations of *Aspergillus* infection. This case report presents a patient with chronic obstructive pulmonary disease who developed a pseudomembranous *Aspergillus tracheobronchitis*, bronchopleural fistula and empyema 16 months after lobectomy for an aspergilloma. Bronchoscopy proved to be important for assessment of severity. Combined systemic anti-fungal treatment (voriconazole) and open window thoracostomy were used to successfully treat the patient.

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1. Introduction

Aspergillus infection, and especially invasive pulmonary aspergillosis, is associated with high morbidity and mortality [1].

Aspergillus tracheobronchitis (ATB) is thought to be a rare manifestation, occurring in less than 7% of patients with an *Aspergillus* infection [2], and primarily affecting lung and other solid organ (e.g. heart or kidney) transplant recipients [3], patients with malignancies (primarily of hematological and pulmonary origin), and immune deficiencies like AIDS [4]. ATB can also be found in patients with chronic obstructive pulmonary disease (COPD) [5], with post-tuberculosis tracheal or bronchial stenosis [2,6], at the level of a bronchial stump after lobectomy [7], or in association with hepatic disease (e.g. cirrhosis) [2], septic shock and ARDS [8], or diabetes mellitus [9,10]. Risk factors for developing ATB include steroid and broad-spectrum antibiotic use, neutropenia, and alcoholism [11].

A review of 121 cases by Karnak et al. showed that *Aspergillus fumigatus* was identified in 63% of *Aspergillus tracheobronchitis* infections. *Aspergillus flavus*, *Aspergillus niger*, and *Aspergillus nidulans* were cultured in 4.1%, 3.3%, and 1.7%, respectively, whereas in 28% of the cases data regarding the species were not available [12]. Diagnosis of ATB may be delayed because of its insidious onset, non-specific signs and symptoms, and scarcity of radiographic abnormalities [13]. Therefore bronchoscopic evaluation should be performed in patients with high risk profile and respiratory symptoms (e.g. dyspnea,

hemoptysis, persistent cough, fever, wheezing or stridor), especially when sputum cultures show growth of *Aspergillus* species in these high-risk patients.

2. Case

A 67-year-old man underwent a right upper lobectomy and systemic treatment (voriconazole) for lung aspergilloma complicated by an invasive aspergillosis. Sixteen months after lobectomy the patient complained of a chronic cough and weight loss. A bronchopleural fistula from the right main stem bronchus to the pleural cavity was seen on a computed tomography (CT) scan (day 0).

Bronchoscopy showed an inflammatory mucosa of the trachea and bronchial tree with patchy white pseudomembranes (Fig. 1, left panel) (day 4). These white pseudomembranes were very adhesive to the mucosa and showed signs of superficial bleeding when aspiration was performed. The orifice of the bronchopleural fistula was visualized in the right main stem bronchus. Pseudomembranes covered the mucosa of the fistula and the bronchus intermedius (Fig. 1, right panel). When entering the fistula opening, a cavity filled with pus and walls covered with green-gray debris and white crystals appeared. Bronchial aspirates of the trachea and pleural cavity were sent for microbiological examination. *Escherichia coli* was found in the samples of the pleural cavity and treated by Amoxicilline-Clavulanic acid (day 5). Although the cultures for *A. fumigatus* came back negative, invasive aspergillosis was the most likely diagnosis because of the highly suspect appearance of the lesions, and an elevated *Aspergillus* antigen

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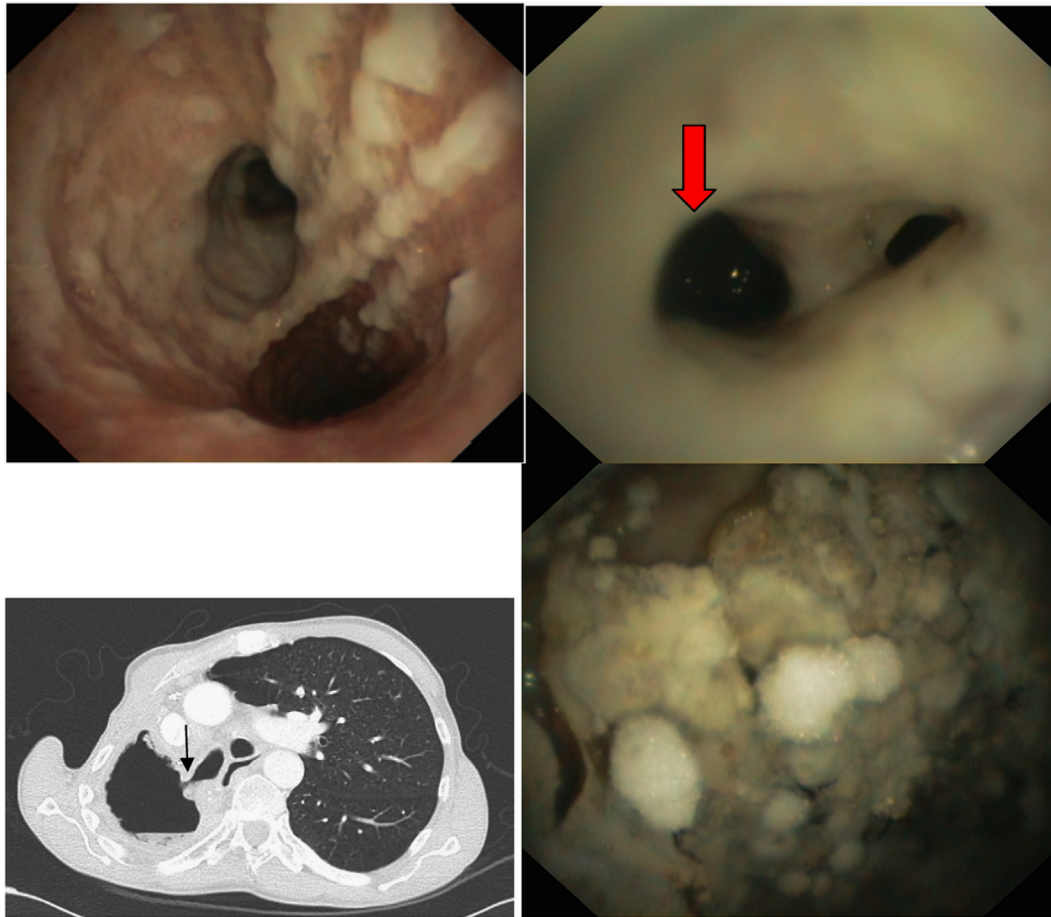


Fig. 1. Left upper panel: Aspergillus tracheobronchitis with white pseudomembranes; Right upper panel: fistula between right main stem bronchus and pleural cavity (red arrow), stenosis of bronchus intermedius due to pseudomembranes. Left lower panel: CT-scan shows bronchopleural fistula (black arrow) and post-lobectomy cavity with air-fluid level and irregular walls. Right lower panel: post-lobectomy cavity with pus and white crystals.

level with optical density (OD) index of 8,14 (reference value: OD index < 0.5) (day 0).

Because of the poor nutritional status of the patient due to COPD and extent of the infected cavity, an open window thoracostomy was performed (day 10). The pleural cavity was cleaned and green-grey substance and white crystals were removed. Biopsies taken from the walls of the pleural cavity and fistula were sent for microbiological examination and cultures came back positive for *A. fumigatus*. Voriconazole 250 mg b.i.d. was started (day 4) and antiseptic packages in the pleural cavity were changed regularly. The voriconazole level was 4.7 mg/l at day 8. Trachea, fistula orifice and bronchial tree had almost normal mucosa when bronchoscopy was performed after surgery (day 31), and the Aspergillus antigen level had a lower OD index of 0.83 (day 33). Voriconazole was stopped after 7.5 months of treatment (day 232). Although a therapeutic level at day 150 (3,5 mg/l), dose modification had to be made to 300 mg b.i.d. (day 172) because of subtherapeutic drug level (< 0.2 mg/l). Antigen levels had been normal during the last 3 months of the treatment (OD index 0.44; 0.34; 0.24; 0.011 at day 140; 172; 213; and 235 respectively).

3. Discussion

Postlobectomy or postpneumonectomy empyema is a known and feared surgical complication. In this case *A. fumigatus* was found as one of the causative agents of empyema formation. A pseudomembranous ATB and bronchial stump aspergillosis were

thought to be the primary event, leading to a fistula, followed by a pleural cavity infection. In this hypothesis the patient was re-exposed to an environmental source of Aspergillus after being successfully treated for an aspergilloma. A second hypothesis suggests an existing Aspergillus colonization of the pleural cavity, in other words an unsuccessfully treated aspergilloma 16 months before. This could have led to a bronchopleural fistula and colonization of the trachea.

In 1970, Young et al. [7] reviewed 98 cases of histologically documented invasive aspergillosis, of which eight had ATB. In five patients, the infection seemed to be limited to the tracheobronchial tree, without signs of parenchymal involvement. However, invasive forms of Aspergillus tracheobronchitis had not been fully described until 1991 by Kramer et al. [3].

Kramer et al. proposed a classification of ATB in three entities: invasive, saprophytic and allergic ATB. In their patients they observed three morphologic subtypes of invasive ATB: tracheobronchitis, ulcerative tracheobronchitis, and pseudomembranous tracheobronchitis.

An impairment of the local natural antifungal defense mechanisms that significantly increases the risk of airway colonization was suggested [3,9]. The hypothesis that local factors play a role in the development of ATB, was also seen in other studies. Wu et al. included nineteen patients with ATB in a retrospective analysis, and observed that malignancy was the most common underlying disease (73.7%). Tracheobronchial tuberculosis and traumatic tracheostenosis were seen in two (10.5%) and one (5.3%) patient, respectively [6]. Noticeably, seven patients (36.8%) had underlying

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