Case Report

Chronic necrotizing pulmonary aspergillosis

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Chronic necrotizing pulmonary aspergillosis is not common and usually involves mildly immunosuppressed patients. We present a case of a 58-year-old man with a history of mining-related pneumoconiosis and corticosteroid therapy who developed bilateral pulmonary infiltrates and subsequent cavitation. The patient was treated at first as having community-acquired pneumonia and was only belatedly diagnosed as suffering from aspergillosis after *Aspergillus fumigatus* precipitins appeared in blood and the same fungus grew from bronchoalveolar lavage fluid. A transthoracic needle biopsy revealed fungal filaments present in material extracted from a pulmonary lesion that was visible on scans. Treatment with amphotericin B, begun at the time that aspergillosis was diagnosed, proved to be ineffective, as was a later change to amphotericin B lipid complex. The diagnosis was confirmed at necropsy.

**Keywords** *Aspergillus fumigatus*, chronic necrotizing pulmonary aspergillosis

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**Case report**

A 58-year-old man with a history of coal worker’s pneumoconiosis and asthma had been treated with inhaled corticosteroids and β₂ agonists. He complained of fever, dyspnea, cough and blood-streaked sputum for 5 days and was admitted to our hospital on 21 February 2002. A chest radiograph showed a consolidation with air bronchogram in the right middle lobe. He was diagnosed with community-acquired pneumonia and received clarithromycin. The clinical course was satisfactory and the consolidation disappeared. Five days later in the hospital, he ran a high fever, dyspnea and cough with brown sputum and hemoptysis.

A physical examination revealed tachypnea, a cardiac murmur and expiratory wheezing in both lungs. He did not have peripheral edema.

Laboratory findings were 12,800 leukocytes per mm³, (neutrophils 77%) and biochemistry results were normal. Arterial blood gas determinations showed pO₂ 57 mmHg, pCO₂ 40, pH 7.5.

A chest radiograph revealed a bilateral infiltrate in the right upper lobe and in the apical segment of left lower lobe (Fig. 1) resulting in a bilateral mass with subsequent cavitation after 2 weeks.

Computerized tomography of the thorax confirmed the presence of a cavitated lesion 7 × 10 cm in diameter with an irregular wall and an air-fluid level in the right upper lobe (Fig. 2), as well as a similar lesion in the apical segment of the left lower lobe. Enlarged right hilar and paratracheal lymph nodes were observed.

Blood cultures were negative as were *Aspergillus* precipitins but one month later precipitins became positive for *Aspergillus fumigatus* and remained negative for *Aspergillus niger*, *A. nidulans*, *A. terreus* and *A. flavus*. Sputum cultures yielded *A. fumigatus*.

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A bronchoscopy was performed. Acid-fast bacilli, *Pneumocystis carinii*, intranuclear inclusions and malignant cells were not seen in the cytological examination. *A. fumigatus* was isolated from bronchoalveolar lavage fluid. Macroscopically the fungal colony on Sabouraud’s glucose agar had a blue-green surface and a white to tan reverse. The colony reached 4.5 cm in diameter after 7 days growth on Czapek–Dox agar at 25°C. The isolate grew well at 45°C. Microscopically, it was characterized by green, echinulate conidia, 2.5–3.0 μm in diameter, produced in basipetal chains from greenish phialides, 6–8 μm long by 2–3 μm wide. Sclerotia, cleistothecia and hülle cells were not observed.

The result of a transthoracic fine-needle aspiration was necrosis, inflammation and hyphae that suggested *Aspergillus*.

The patient was receiving corticosteroids at the time the infection developed. He was treated with amphotericin B but it was not started promptly: it was only three weeks after he had begun to run a fever and after bilateral infiltrates had been observed in a chest radiograph that this treatment began. After a cumulative dosage of 400 mg, serum creatinine increased, therapy was changed to amphotericin B lipid complex; the cumulative dosage was 1450 mg. Three months after the admission he died, on 20 May 2002. The diagnosis of chronic necrotizing pulmonary aspergillosis was supported by histologic appearance of the fungus in a specimen obtained at necropsy, compatible with *Aspergillus* species (Fig. 3).

**Discussion**

Chronic necrotizing pulmonary aspergillosis (CNPA) or semi-invasive aspergillosis was described by Gefter et al. [1] in 1981 and Binder et al. [2]. It is less frequently seen than invasive pulmonary aspergillosis and affects patients with altered local defenses as a result of underlying lung diseases (e.g. pneumoconiosis, previous pulmonary tuberculosis, chronic obstructive lung disease). It may also occur in mild systemically immunosuppressed patients with diabetes, alcoholism, collagen disease, poor nutrition or low-dose corticosteroid therapy [3].

The clinical course of CNPA is slow, featuring fever, weight loss, cough, sputum production and occasional hemoptysis. The most frequent radiologic findings are infiltration with air in the bronchogram, pleural thickening and subsequent cavitation in the upper lung lobes [4].

Bilateral cavitation as seen in our case is extremely rare. A prompt recognition of radiographic features consistent with CPNA is useful for early diagnosis [5]. Pathologically, CNPA is characterized by necrosis of lung tissue, acute or chronic inflammation of the cavity wall, and presence of hyphae suggestive of *Aspergillus* [6].

The diagnosis is often difficult and requires a demonstration of *Aspergillus* in lung biopsy. Clinical criteria are also accepted. A high index of suspicion is necessary, since the prognosis depends on the promptness of treatment and on the nature of the underlying lung disease. The mere isolation of *Aspergillus* from respiratory tract should not, by itself, be considered to indicate colonization [7].
There is no well-established treatment for CNPA. Amphotericin B, itraconazole, voriconazole, caspofungin and surgery are useful [7–10]. We decided to give our patient amphotericin B but he was also being treated at that moment with intravenous methylprednisolone for dyspnea as a result of his history of asthma. The immunosuppression caused by corticosteroids and the delay in initiation of amphotericin B could explain the failure of treatment.

References