Bronchoscopy findings in invasive pulmonary aspergillosis

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ABSTRACT Two patients with invasive aspergillosis had unusual endobronchial appearances at fibreoptic bronchoscopy. Diagnosis was achieved by endobronchial biopsy.

Fibreoptic bronchoscopy is used to diagnose infections in immunocompromised patients. The samples taken are usually bronchial washings and brushings, bronchoalveolar lavage fluid, or transbronchial biopsy material. Diagnosis of invasive aspergillosis is rarely achieved by biopsy of visualised bronchial lesions.1

We report two patients with invasive aspergillosis whose bronchial tree showed particular features that led to diagnosis of the infection.

Case reports

PATIENT 1

A 48 year old retired quarryman, a smoker, was treated for pulmonary tuberculosis in 1981 and presented in 1982 with malaise and polyarthritis. Subluxation and joint destruction were present in both hands. Antinuclear antibodies were present at a titre of 1:10244 with a homogeneous pattern and antibodies to double stranded DNA at a titre of 1:320, and the lupus erythematosus cell test gave a positive result. Rheumatoid factor and extractable nuclear antigens were undetectable; other criteria for the diagnosis of systemic lupus erythematosus were not met. His condition improved after treatment with prednisone.

In 1986 he presented with progressive weakness, anorexia, weight loss, fever, and haemoptysis. He had received treatment with chloroquine alone for the previous 12 months. The white blood cell count was 9.06×10^9 /l with 73% neutrophils. Antinuclear antibodies were present at a titre of 1:1280; antibodies to double stranded DNA were not detected. The chest radiograph showed an irregular nodular infiltrate in the right upper lobe, segmental consolidation in the left upper lobe, and slight paratracheal lymphadenopathy (fig 1). Fibreoptic bronchoscopy showed brownish, jelly like, cone shaped excrescences in the distal trachea (fig 2a) and in the openings of both upper lobe bronchi. Biopsy of this material showed granulation tissue and septate hyphae. Aspergillus fumigatus was cultured; anaerobes, aerobes, and mycobacteria were not isolated. Serum aspergillus precipitins were

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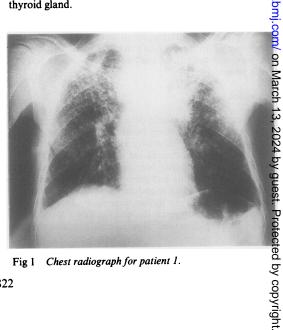
Thorax: first published as 10.1136/thk as 10.1136/t of 1700 mg, but neurological, ophthalmological, and cardia complications ensued and the patient died.

Necropsy showed a left upper lobe, necrotic, aspergilluso pneumonia affecting also the mediastinum. Mediastina lymph nodes were discharging into the trachea and bronchus. Fungal invasion of the heart, aorta, kidneys, spleen, and central nervous system was seen. Scattered coniotic micronodules in the lung and a large embolus in the right pulmonary artery were also found.

PATIENT 2

A 26 year old man receiving chemotherapy for lymphoblastic leukaemia was admitted because of fever and a segmental infiltrate in the left upper lobe. The granulocyte count was 0.31×10^9 /l. After seven days' treatment with ticarcillin and tobramycin, the shadowing on the chest radiograph hat increased, though there was no cavitation. His fever persistes and amphotericin B was started. The granulocyte count was 3.2×10^{9} /l and the platelet count 165 × 10°/l. At fibreoption bronchoscopy an eroded bronchial wall was seen at the left. superior division (fig 2b) and at the lingular bronchuQ Through one of the ulcers the bronchoscope tip entered and empty, black walled cavity. Biopsy of the wall showed septate hyphae and Aspergillus fumigatus was cultured. The patient died 36 hours later from a massive haemoptysis.

At necropsy aspergillus necrotising pneumonia was found in the left upper lobe with spread to the heart, kidneys, and thyroid gland.



Chest radiograph for patient 1. Fig 1



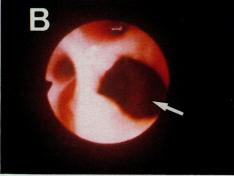


Fig 2 (a) Polypoid lesions in the trachea of patient 1. (b) Ulcer of the bronchial wall and necrotic cavity (arrow). On the left is the opening of the B1 and B2 bronchi and at the top the B3 bronchus.

Discussion

In invasive aspergillosis the bronchi are less frequently colonised than the parenchyma² and bronchoscopic findings have been reported infrequently. The first patient had endobronchial lesions resembling perforation from contiguous lymph nodes, which was later confirmed. Segmental mucosal lesions of the trachea with paratracheal soft tissue

inflammation and no mediastinal lymphadenopathy have been found by computed tomography.³ Two other cases presented with large airways obstruction by fungal plugs but mediastinal invasion was not documented.⁴

Fatal haemoptypsis, as occurred in the second patient, has been reported in invasive aspergillosis. Lung necrosis may develop in patients with pulmonary aspergillosis recovering from granulocytopenia induced by chemotherapy, 5 as in our case. This happened also in a patient with a bronchial ulcer and a broncho-oesophageal fistula. 6 We were able to visualise a pulmonary cavity, which could represent an early stage of a "mycotic lung sequestrum." This bronchial lesion suggests the need to consider surgical resection, as proposed by Kibbler et al. 7

The bronchial biopsy specimens from our two patients showed tissue invasion by fungus but no colonisation of blood vessels, as reported by Chung et al,⁸ who used transbronchial biopsy. The particular endoscopic findings in our cases led us to start treatment with amphotericin B and invasive aspergillosis was confirmed at necropsy.

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