Bronchoscopic findings in a case of bronchopulmonary histoplasmosis

Barry Kneale, Charles Turton

Abstract
At bronchoscopic examination extensive areas of fibrinous slough covering bronchial mucosal inflammation and ulceration were seen in a case of progressive diffuse bronchopulmonary histoplasmosis. Rigid bronchoscopy was needed to obtain sufficient biopsy material for specific histological diagnosis.

Keywords: bronchoscopy, histoplasmosis.

Pulmonary histoplasmosis is uncommon in the UK and is very rarely progressive in those with intact host defence mechanisms. We describe a case of bronchopulmonary histoplasmosis which progressed to respiratory failure with florid bronchoscopic appearances unfamiliar to three experienced consultant bronchoscopists.

Case report
A 50 year old white patient presented with increasing breathlessness, cough producing mucoid sputum, and weight loss following an influenza-like illness one month before. He was a smoker but had suffered no previous chest illnesses. He had not been in contact with tuberculosis and had had no known contact with bird or bat excrement. Two years earlier he had holidayed in the Galapagos Islands but he had not travelled to the USA. He was homosexual but had been HIV antibody negative two years earlier. Initially there were no particular physical signs. The chest radiograph showed bilateral ill defined and fine nodular infiltration in the upper zone. Routine haematological and biochemical screening was non-contributory. The sputum was negative for acid fast bacilli and the tuberculin skin test was non-reactive. HIV antibodies were negative, immunoglobulins and lymphocyte subsets normal, and a bone marrow examination unremarkable. The FEV₁/FVC was 1.9/2.7 (predicted 3.4/4.4), peak expiratory flow rate (PEFR) 380/min (predicted 560), PaO₂ 12.8 kPa, and PaCO₂ 5.7 kPa breathing room air. The bronchoscopic appearances are shown in the figure. Special stains of bronchial biopsies showed the typical yeast forms of *Histoplasma capsulatum*. Serological tests for *Histoplasma* were positive both by immunodiffusion and complement fixation.

Treatment was commenced with intravenous amphotericin. He became more breathless with PEFR falling to 150 l/min, PaO₂ to 6.5 kPa, and PaCO₂ to 3.8 kPa (breathing air). There was a dramatic response to prednisolone 60 mg daily and ketoconazole 400 mg daily. Amphotericin was continued for eight weeks and ketoconazole for nine months. Bronchoscopy was repeated after 12 months and revealed that the slough and ulceration had resolved leaving minor bronchitic changes and stenosis of the upper lobe airways, particularly on the left side where the airway distal to the lingula bronchus was narrowed to the size of a pinhole by granulation tissue. There was no histological or microbiological evidence of persisting *Histoplasma* infection. After two years the patient is well generally but breathless on exertion (MRC grade 1). The chest radiograph shows persistent infiltration in the left upper zone, with computed tomographic evidence of fibrosis and bronchiectasis of the left upper lobe.

Bronchoscopic appearances of (A) the main carina and (B) the right main bronchus showing very extensive plaques of haeck fibrinous slough with underlying severe bronchial inflammation and ulceration. Both upper lobe bronchi were narrowed concentrically, particularly on the left distal to the lingula branch.
Discussion
The patient was exposed to iguana when on vacation in the Galapagos Islands but a two year incubation period seems unlikely. The holiday is probably irrelevant and the case should be considered as a sporadic infection. Histoplasmosis is rare in the UK. Even in the endemic regions of the great river valleys of the USA progressive disease or persistent radiographic abnormality is unusual. Disseminated disease may be an early feature of altered immunity and has been reported as the presenting feature of AIDS.1 There was no evidence of impaired host defence mechanisms in this case. The most striking feature of a remarkable case was the dramatic bronchoscopic appearances. Three consultants experienced in bronchoscopy had seen nothing like them before. Amyloidosis and diffuse lymphoma were considered but the appearances were not typical of either. Biopsy samples taken at fibroptic bronchoscopy, though apparently generous, yielded fibrinous slough only. Rigid bronchoscopy was necessary to obtain sufficiently deep biopsies for histological diagnosis. A year after presentation the visible changes were of non-specific bronchial inflammation, but with stenosis of the upper lobe bronchi similar to the appearances seen occasionally in tuberculosis or sarcoidosis. In a review of bronchoscopy in the diagnosis of pulmonary histoplasmosis2 the macroscopic appearances described were mild and non-specific, “friable mucosa” and “endobronchial nodularity” being reported in one case each.

When bizarre bronchoscopic appearances are encountered rigid bronchoscopy may be necessary to obtain bronchial biopsies of adequate size to rule out specific infections.


A case of Acinetobacter calcoaceticus pneumonia

Hayati Bilgiç, E Serdar Akin, Yücel Taşan, Kudret Ekiz, Olağ Seber

Abstract
A case of community acquired pneumonia with Acinetobacter calcoaceticus is presented. Acinetobacter must be considered in the differential diagnosis of Gram negative coccobacillary pneumonia.

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Keywords: Acinetobacter infection, pneumonia.

Acinetobacter has been isolated from numerous environmental and human sources. Up to 25% of healthy adults exhibit cutaneous colonisation and 7% have transient pharyngeal colonisation.1-5 It is the most common Gram negative organism persistently carried on the skin of hospital personnel and was found to colonise 45% of in-patient tracheostomy sites.1-7 Acinetobacter has been described as the causative agent of supplicative infections in many organ systems.1-7 Usually acknowledged to be opportunistic in patients with altered host defences, Acinetobacter infections have been reported in otherwise healthy hosts.2-7

Case report
A 20 year old white man was admitted to our clinic in July 1990 complaining of shortness of breath, fever, cough, haemoptysis, and vomiting. He had smoked 20 cigarettes/day for five years. He had no history of alcohol consumption, drug abuse or systemic illness that could have predisposed him to opportunistic infection. On physical examination he was comatose with a fever of 39-7°C, heart rate of 140 beats/min, blood pressure of 110/50 mm Hg, and a respiratory rate of 26/min with shallow respiration and fine bibasilar rales. Chest radiography showed extensive bilateral infiltrates with areas of consolidation involving most of the right lung. The patient was hypoxaemic (PaO2 7-1 kPa breathing air). Urine analysis was normal. Cultures of blood urine, and cerebrospinal fluid were sterile.

Gram stains of the initial throat culture and transtracheal aspirates were misinterpreted as Staphylococcus aureus. Sputum culture grew Gram negative bacilli, Klebsiella pneumoniae. Transtracheal aspirates were cultured for anaerobes, mycoplasma, and Acinetobacter.

Initially the patient was treated with intravenous benzylpenicillin (10 million units six hourly) and cephalothin sodium (1 g 12 hourly). After two days of treatment radiological progression was seen. On the third day Acinetobacter was identified in cultures of the transtracheal aspirate. The case was accepted as Acinetobacter pneumonia because Kudret only grew in the first sputum specimen and not in the subsequent sputum cultures nor in the transtracheal aspirate. In addition, Acinetobacter was isolated from the transtracheal aspirate which is a more specific and sensitive sample than sputum culture. Antibiotics were changed to intravenous ceftriaxone 2 g 12 hourly and amikacin 500 mg 12 hourly (according to the result of antibiotic resistance tests in vitro) and the patient subsequently made a full recovery in 10 days.

Discussion
Community acquired Acinetobacter pulmonary infections generally occur in patients with decreased host defences, including alcoholics,
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B Kneale and C Turton

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