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 fibreoptic bronchoscopy, though apparently generous, yielded fibrinous slough only. Rigid bronchoscopy was necessary to obtain sufficiently deep biopsies for histological diagosis. 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 histoplasmosis[2] 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.

1 Salzman SH, Smith RL, Aranda CP. Histoplasmosis in patients at risk for the acquired immunodeficiency syndrome in a non-endemic setting. *Chest* 1988;**93**:916–21.
2 Prechter GC, Prakash UBS. Bronchoscopy in the diagosis of pulmonary histoplasmosis. *Chest* 1989;**95**:1033–6.

A case of *Acinetobacter calcoaceticus* pneumonia

Hayati Bilgiç, E Serdar Akin, Yücel Taşan, Kudret Ekiz, Olgaç 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.

(*Thorax* 1995;**50**:315–316)

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.[1-6] It is the most common Gram negative organism persistently carried on the skin of hospital personnel and was found to colonise 45% of inpatient tracheostomy sites.[1-4 7 8] *Acinetobacter* has been described as the causative agent of suppurative infections in many organ systems.[1-7 9] Usually acknowledged to be opportunistic in patients with altered host defences, *Acinetobacter* infections have been reported in otherwise healthy hosts.[2 7]

Department of Chest Disease, Gülhane Military Medical Academy, Ankara, Turkey
H Bilgiç
E S Akin
Y Taşan
K Ekiz
O Seber

Reprint requests to:
Dr E S Akin, Gata Gogus Hastaliklari ABD, Ögretion Uyesi, Etlik, Ankara, Turkey.

Received 13 April 1993
Returned to authors 28 May 1993
Revised version received 20 December 1993
Accepted for publication 5 January 1994

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 (Pao_2 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 *Klebsiella* 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,

tobacco abusers, patients with renal failure, and those with underlying pulmonary disease. Nosocomial *Acinetobacter* pneumonias are often associated with endotracheal intubation or tracheostomy, prior antibiotic therapy, residence in an intensive care unit, or recent surgery.[2 3 5 9] Nosocomial spread in the intensive care unit has been attributed to ventilatory equipment and to colonised personnel.[2]

Community acquired *Acinetobacter* pneumonia is a fulminant disease. More than half the patients develop hypotension and shock within 24 hours, and nearly all are profoundly hypoxaemic. The chest radiograph may reveal either lobar consolidation or bronchopneumonia, but progression to diffuse, bilateral involvement often occurs rapidly. Pleural effusions occur in half and many of these are purulent.[10]

Mortality from community acquired *Acinetobacter* pneumonia is close to 50%, with death often occurring in the first few days of the illness.[10] Secondary bacteraemia, septic shock, and empyema are associated with a poor prognosis.[2 4 10]

In two of three cases of *Acinetobacter* pneumonia in foundry workers, iron particles and mixed dust pneumoconiosis were found in the lungs at postmortem examination. This suggested that chronic exposure to such particles may increase the susceptibility to infection by this organism.[3]

In vitro susceptibility testing generally reveals that there is no antimicrobial agent to which *Acinetobacter* is uniformly sensitive. However good responses are recorded to the combination of imipenem and perfloxacin.[7] Many mild to moderately severe infections will respond to (effective) single antibiotic treatment. However, in serious infections treatment should be based on the result of susceptibility testing and the contribution of a β-lactam (piperacillin, ticarcillin, ceftazidime) and an aminoglycoside.[1 2] As with other Gram negative bacillary pneumonias, serious pulmonary infection should be treated for at least three weeks.[10]

Because of the acute onset of symptoms, extensive and progressive bilateral infiltrates, occurrence in the summer, and the dramatic clinical presentation, we interpreted our case as one of a community acquired pneumonia. Misinterpretation of the initial Gram stain of the transtracheal aspirate as *Staphylococcus* caused a delay in starting correct treatment. In spite of this, no therapeutic advantages for third generation cephalosporins have been reported. We achieved a rapid clinical improvement with an aminoglycoside (amikacin) and a third generation cephalosporin (ceftriaxone), chosen on the results of susceptibility testing.

The value of transtracheal aspiration in isolating the causative organism when clinical findings are not consistent with pathogens isolated from sputum cultures in an immunocompromised host or in those unable to expectorate is emphasised.

1 Berk SL, McCabe WR. Meningitis caused by *Acinetobacter calcoaceticus* var. *anitratus*: a specific hazard in neurosurgical patients. *Arch Neurol* 1981;**38**:95–8.
2 Allen DM, Harman BJ. Acinetobacter species. In: Donowitz GR, Mandell GL, Douglas RG, Bennett JE, eds. *Principles and practice of infectious disease*. New York: Churchill Livingstone, 1990:1696–700.
3 Cordes LG, Brink EW, Checko PJ, Lentnek A, Lyons RW, Hayes PS, *et al.* A cluster of *Acinetobacter* pneumonia in foundry workers. *Ann Intern Med* 1981;**95**:688–93.
4 Smego RA. Endemic nosocomial *Acinetobacter calcoaceticus* bacteremia: clinical significance, treatment and prognosis. *Arch Intern Med* 1985;**154**:2174–9.
5 Smith PW, Massanari M. Room humidifiers as a source of *Acinetobacter* infections. *JAMA* 1977;**237**:795–7.
6 Berk SL, McCabe WR. Meningitis caused by gram-negative bacilli. *Ann Intern Med* 1980;**93**:253–60.
7 Ygout JF, Housset B, Derenne JP, Daguet GL. Hospital acquired *Acinetobacter baumanii* pneumonitis. *Lancet* 1987; i:802.
8 Wallace RJ, Awe RJ, Martin RR. Bacteremic *Acinetobacter* pneumonia with survival: case report. *Am Rev Respir Dis* 1976;**113**:695–9.
9 Goodhard GL, Abrutyn E, Watson R, Root RK, Egert J. Community-acquired *Acinetobacter calcoaceticus* var *anitratus* pneumonia. *JAMA* 1977;**238**:1516–8.
10 Johnson CC, Finegold SM. Pyrogenic bacterial pneumonia lung abscess and empyema. In: Murray JF, Nadel JA, eds. *Textbook of respiratory medicine*. Philadelphia and London: WB Saunders, 1988:816–27.