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References


Community-acquired bacteraemic Acinetobacter pneumonia with survival

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Summary: A 65 year old man was admitted with segmental consolidation of the left upper lobe after having stayed in a hotel for 2 days. He deteriorated rapidly on conventional antibiotic therapy and required ventilatory support. Acinetobacter calcoaceticus var. anitatus was grown from the sputum and blood cultures, which was treated with a combination of anti-pseudomonal agent, aminoglycoside and cotrimoxazole. He made a slow but remarkable recovery from the pneumonia. Acinetobacter is a rare potentially fatal cause of community-acquired pneumonia.

Introduction

Acinetobacter calcoaceticus is an aerobic pleomorphic, encapsulated Gram-negative predominantly coccobacillus or diplococcus, which may be confused with Neisseria or Haemophilus. It is widely distributed in water and soil and may be frequently
found as a commensal in healthy people. Usually regarded as non-pathogenic, it may produce a wide variety of serious hospital-acquired infections. Of these, pneumonia is by far the commonest and carries high mortality. It predominantly occurs in those patients who are on mechanical ventilation or with tracheostomy, in intensive care units and those who have been on antibiotics. However, community-acquired pneumonia due to \textit{Acinetobacter} is rare and only a small number of cases have been reported in the literature. As far as we know this is the first case of community-acquired \textit{Acinetobacter} pneumonia to be reported from the Middle East.

**Case history**

A 65 year old Kuwaiti businessman was admitted in September 1989 with a 24 hour history of feeling suddenly unwell with fever, chills and rigor, difficulty in breathing and left pleuritic pain. He had spent 2 days in a hotel in Bahrain and had become ill soon after returning home. He was a heavy smoker and drank alcohol regularly in moderate amount.

On admission, he was dyspnocic at rest, febrile (38°C) and toxic; pulse rate was 100/minute and blood pressure 140/100 mmHg. There were inspiratory crackles in the left upper zone and axillary area, and generalized inspiratory wheezes. Chest X-ray on admission revealed consolidation of the posterior segment of the left upper lobe. Initially there was leucocytosis of 34,000 x 10³/l with polymorphonuclear shift to the left. The sputum appeared pinkish/blood stained. Initial direct smear of the sputum was suggestive of Gram-positive diplococcus, so he was started on high dose crystalline penicillin and given bronchodilators including nebulized salbutamol and aminophylline infusion. Arterial blood gases on room air showed a \textit{PaO₂} of 10.5 kPa, \textit{PaCO₂} of 5.5 kPa and pH of 7.34. Over the next 24 hours he deteriorated and became tachypnoic, cyanosed and developed septicemic shock, and oliguria with rise in serum urea to 20.3 mmol/l and creatinine to 168 µmol/l. Arterial blood gases on 35% \textit{O₂} showed \textit{PaO₂} of 7.1 kPa \textit{PaCO₂} of 9.3 kPa and pH of 7.17.

He was ventilated and resuscitated with intravenous (i.v.) colloids, crystalloids and inotropes. He was also given 1 g of methylprednisolone i.v. and gentamycin 80 mg i.v.

The next day (that is, 48 hours after admission) both blood and sputum cultures showed heavy growth of \textit{Acinetobacter calcoaceticus var. anitratus} which was initially identified by API 20E and confirmed by API 20NE (La Balme Les Grottes, France). It was sensitive to carbenicillin, tetracycline, cotrimoxazole, gentamicin, amikacin and piperacillin and resistant to ampicillin, cefuroxime, and moderately resistant to cefotaxime. Penicillin and gentamycin were discontinued, and piperacillin i.v. 3 g 6 hourly and amikacin i.v. 500 mg 12 hourly were commenced. By this time there was further consolidation involving the whole of the left upper lobe and evidence of moderate degree of disseminated intravascular coagulation. He recovered from the renal shutdown within 36 hours and made a steady improvement and was extubated after 7 days of artificial ventilation. The pneumatic shadow on the X-ray showed almost complete resolution during this period.

However, 2 days later he again became pyrexial and dyspnocic with productive cough of mucopurulent phlegm. Radiologically he had developed recurrence of consolidation of the left upper lobe and the white cell count had gone up to 27.7 x 10³/l while he was still on a combination of piperacillin and amikacin for 9 days. Gram-staining and culture of the sputum showed no significant organism. At this stage he was started on cotrimoxazole infusion 10 ml twice daily (800 mg of sulphamethoxazole and 160 mg of trimethoprim/10 ml) along with continuation of amikacin (piperacillin was discontinued). Twenty-four hours after starting cotrimoxazole, he was noticed to be confused and had myalgia and diarrhoea. The left upper lobe looked more dense on chest X-ray. Erythromycin infusion 1 g 6 hourly was commenced to cover any possible \textit{Legionella} infection. Fiberoptic bronchoscopy was performed which showed no abnormality. Subsequent bronchial lavage and blood cultures did not reveal any bacterial growth. Computerized tomography of the chest did not show any abnormality other than dense consolidation of the left upper lobe. He improved slowly with temperature returning to normal after 5 days on cotrimoxazole and erythromycin, which were given for a total period of 2 weeks along with amikacin.

Further investigations revealed no antibodies to human immune deficiency virus (HIV), \textit{Legionella pneumophila} and \textit{Mycoplasma}. He was discharged home 2 months after admission. The left upper lobe shadowing gradually resolved over the next 6 months. A chest X-ray in May 1990 (that is, 8 months later) showed only a residual shadowing of the left upper lobe.

**Discussion**

\textit{Acinetobacter} pneumonia is an uncommon but important cause of community-acquired pneumonia. Cordes et al.\textsuperscript{2} in their review have cited 18 cases of non-nosocomial pneumonias which were reported mainly from the United States between 1955 and 1979 (including cases described by Rudin...
et al.\textsuperscript{4}). Since then there have been a further 23 cases\textsuperscript{3,5–14} (including this report) of community-acquired \textit{Acinetobacter} pneumonia described in the literature and the majority of these have been reported from developing countries.\textsuperscript{10,12,13} The major risk factors among these patients seem to be heavy cigarette smoking with chronic lung disease, alcoholism, diabetes mellitus and impaired immunity. The three cases reported by Cordes \textit{et al.} occurred in an iron foundry thus suggesting chronic exposure to industrial dust may be an environmental factor in the development of infection due to \textit{Acinetobacter}.\textsuperscript{2} It is suggested that like \textit{Legionella pneumophila}, \textit{Acinetobacter} is also capable of adapting to an aqueous environment and of being dispersed in industrial aerosols created by compressed air, or humidification systems and causing infection in susceptible individuals.\textsuperscript{3} An outbreak of hospital-acquired pneumonia has been proved to be due to contamination of aerosolizing fluid with \textit{Acinetobacter}.\textsuperscript{15} It is thus possible that the cooling system in the hotel where the patients stayed may have been the source of infection in this particularly susceptible individual.

\textit{Acinetobacter} pneumonia occurs predominantly in men, usually above the age of 50 years (range 18–80 years). The usual presentation of the illness is acute high fever, dyspnoea, pleuritic pain and productive cough; the sputum may be rusty or frankly blood stained. The patient may be shocked at the time of admission or will do so rapidly if no appropriate measures are taken. White cell responses vary from marked leucocytosis to leucopenia. The chest X-ray usually shows lobar consolidation in the majority of cases and diffuse bronchopneumonia may be seen at the onset and sometimes lobar infiltrate may progress to diffuse bilateral infiltration in fulminant cases.\textsuperscript{3,4,10,14} Unilateral or bilateral pleural effusions, often large, may occur with initial consolidation in half of the patients. Rarely an empyema or an abscess and multiple cavities may also complicate the initial infection.\textsuperscript{4,8,9,12} Blood cultures are positive in the majority of cases (\textgreater{}70\%) of community-acquired \textit{Acinetobacter} pneumonias in contrast to nosocomial infections where bacteraemia is not a characteristic feature.\textsuperscript{15} The diagnosis is also established by culture of the organisms from pleural fluid, pulmonary aspirate and sputum.\textsuperscript{10,12} Gram-staining of the sputum may be misinterpreted occasionally for \textit{Streptococcus} or \textit{Staphylococcus} as the organisms tend to resist alcohol decolorization,\textsuperscript{1,8,9} thus leading to delay in the diagnosis and institution of appropriate therapy.

\textit{Acinetobacter} pneumonia often runs a fulminant course with a high mortality rate (\textgreater{}50\%), especially when the patient presents with bacteraemia or shock. Often it is the inappropriate antibiotics given before the confirmation of the diagnosis that seem to be responsible for the high mortality in majority of the cases. If appropriate antibiotics including an aminoglycoside are given at least for 3 days mortality rate is significantly reduced.\textsuperscript{4,12} Although our patient deteriorated initially, early institution of aminoglycoside seems to have contributed to his survival.

\textit{Acinetobacter calcoaceticus} is usually resistant to ampicillin, erythromycin, first- and second-generation cephalosporins, clindamycin and chloramphenicol. Aminoglycosides are reported to be the most reliable agents (especially tobramycin and kanamycin),\textsuperscript{2} but the organisms may have variable sensitivities.\textsuperscript{16} Third-generation cephalosporins have been shown to have appreciable anti-\textit{Acinetobacter} activity and have been advocated to be one of the preferred antibiotics in the initial management of suspected \textit{Acinetobacter} pneumonia.\textsuperscript{1} In our case the organisms were found to be resistant to both cefuroxime and cefotaxime. Trimethoprim-sulphamethoxazole has the highest degree of anti-\textit{Acinetobacter} activity and is the preferred drug of choice. Anti-pseudomonal penicillins are also effective antibiotics against \textit{Acinetobacter}, especially in combination with aminoglycosides, because of their synergistic effects.\textsuperscript{1,2}

This case highlights occasional occurrence of \textit{Acinetobacter} pneumonia in the community, initial difficulties in the diagnosis and the serious nature of this infection. It should be considered in the differential diagnosis of community-acquired pneumonias in the appropriate clinical setting.

References

Chronic hypothermia and water intoxication associated with a neurodegenerative disease

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Summary: We describe a 71 year old man with a neurodegenerative condition who developed chronic inappropriate antidiuretic hormone secretion and hypothermia resulting in recurrent episodes of impaired consciousness. This combination of abnormalities is attributable to hypothalamic disease and has not to our knowledge been previously reported with clearly documented antidiuretic hormone excess.

Introduction

Chronic hypothermia of neurological origin is a rare condition that has been described in association with a number of different diseases, some of which can also cause the syndrome of inappropriate antidiuretic hormone (ADH) secretion.1–6 We report a case of chronic hypothermia and concurrent hyponatraemia in a patient with a neurodegenerative condition. He had required several previous admissions because of a confusional state due to acute exacerbations of hypothermia and hyponatraemia, and on follow-up remained hypothermic and hyponatraemic with inappropriately high ADH levels.

Case history

A 71 year old man was admitted as an emergency in March 1992 with a 3 day history of increasing confusion and somnolence. He was disabled and lived with his able-bodied brother in a well-heated adapted flat.

He had congenital deformity of both feet with short Achilles tendons. There was a history of progressive neurological impairment, starting with pyramidal weakness in the legs first noted at the age of 44 years. Asymptomatic optic atrophy was also present at that time. Spastic tetraparesis, cerebellar ataxia and a sensorimotor peripheral neuropathy were apparent by the age of 55 years, when nerve conduction studies and biopsy showed chronic peripheral demyelination. A full myelogram was normal, as was cerebrospinal fluid analysis, although electrophoresis was not done. Vitamin B12 was normal and syphilis serology was negative.

There was a family history of congenital foot deformity. No firm diagnosis was made but at that time the condition was felt most likely to be a hereditary spino cerebellar degeneration.

Examination on this admission showed him to be somnolent and cold, with a rectal temperature of


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