Case Report

Severe community-acquired pneumococcal pneumonia (CAP) – a potentially fatal illness

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Two cases of severe community-acquired pneumococcal pneumonia (CAP) admitted to our hospital within 24 h are described, both in young males. These two cases illustrate the usefulness of the British Thoracic Society severity criteria and serve to emphasize the importance of early recognition of adverse physiology in critically ill patients. We should not lose sight of the continued impact of pneumonia in this climate of widespread fear about severe acute respiratory syndrome (SARS).

Introduction

In recent months, both the British Medical Journal (Zambon & Nicholson, 2003) and the New England Journal of Medicine (Drazen, 2003; Tsang et al., 2003) have commented extensively, quite rightly in our opinion, on the new severe acute respiratory syndrome (SARS). Epidemiologically, virologically, virologically and clinically, this is a fascinating disorder, attracting the attention of the World Health Organization (WHO, 2003). However, whilst one of the authors (M. G. Kelly) was recently visiting Singapore, he was struck by the high profile engendered by this disease in the media and by the use of facemasks by the general public, particularly in Changi airport. In contrast, countless numbers of cases of community-acquired pneumococcal pneumonia (CAP) often go by unnoticed and unpublicized. Indeed, these too may be severe, life-threatening or even fatal (British Thoracic Society Standards of Care Committee, 2001).

In our hospital, we have recently encountered a number of cases of CAP in young, fit males aged 20–45. Two particular cases, occurring within 24 h of each other, serve to remind us of the continuing significance of this condition, the usefulness of the British Thoracic Society severity score (the CURB score) and, particularly, the role of pneumococcal infection (British Thoracic Society Standards of Care Committee, 2001; Lim et al., 2001).

Case 1

A healthy 22-year-old male welder, who had smoked 20 cigarettes per day for 5 years, was admitted with a 4 day flu-like illness, characterized by dry cough, anorexia and anergia followed 24 h prior to admission by fever and rigors. Clinical examination revealed a relatively undistressed male with a tachycardia (120 beats min⁻¹), tachypnoea (40 breaths min⁻¹), hypotension (80/40 mmHg), fever (39.4 °C) and desaturation (O₂ saturation 85 % breathing room air). He was clammy, sweaty and vasodilated and had dullness with coarse inspiratory crackles at the left base. Chest radiograph confirmed extensive left lower lobe consolidation. Electrocardiograph confirmed sinus tachycardia. Arterial blood gas breathing room air revealed a mixed pattern of respiratory failure [pH 7.31 (normal range 7.35–7.45), pO₂ 4.09 kPa (10–13), pCO₂ 6.0 kPa (4.5–6.0), bicarbonate 26 mmol l⁻¹ (22–28), base excess +1.6 (+1.0 to +2.0)] with a mixed acidosis. Haematological investigation revealed leucopenia (white cell count 2.4 with neutrophils 2.0 × 10⁹ cells l⁻¹). Biochemistry revealed acute renal failure [urea 30.3 mmol l⁻¹ (normal range 3–7), creatinine 342 µmol l⁻¹ (normal range 30–110)]. C reactive protein (CRP) was elevated, at 246 mg l⁻¹ (normal range < 5). Day 1 blood cultures grew penicillin-sensitive (but macrolide-resistant) pneumococcus. Aggressive colloid and crystalloid resuscitation corrected the hypovolaemia, with good improvement in renal function. Intravenous coamoxiclav and oral erythromycin were instituted. Despite high-flow oxygen, ward-based continuous positive airway pressure respiratory support and a change of antibiotics, this patient required intensive care unit admission and ventilation 48 h after admission. He required prolonged invasive ventilation (25 days) and, eventually, a tracheostomy for adult respiratory distress syndrome (ARDS). After 2 weeks in intensive care, the patient was discharged to the ward, where he rehabilitated rapidly to recover from his critical illness polyneuropathy and was eventually discharged home. At review 6 weeks later, he was well and radiological changes had resolved.
Case 2
Twenty-four hours after the first admission, a 22-year-old male alcoholic, a smoker with a history of sociopathic personality traits and evidence of substance misuse, was admitted in a toxic confusional state. A corroborative history suggested a recent dry cough. He had been ‘living rough’. On examination, he was unkempt, cachexic, confused in time and place, aggressive and clinically dehydrated. He had a tachycardia (120) and tachypnoea (24) but was afebrile and normotensive. Oxygen saturations breathing room air were 92 %. Glasgow Coma Scale was 13/15. Pupils were widely dilated, equal and reactive. Extensive bronchial breath sounds were heard over the right hemithorax, but only a few coarse inspiratory crackles. Chest radiograph revealed right lower lobe consolidation and pleural effusion with lingual and left lower lobe consolidation. Arterial blood gas measurement breathing room air confirmed type-1 respiratory failure with respiratory alkalosis [pH 7.478 (normal range 7.35–7.45), Po2 886 kPa (10–13), PCO2 4.24 kPa (4.5–6.0), bicarbonate 24 (22–28), base excess +0.4 (−2.0 to +2.0)]. Sodium was 118 mmol l$^{-1}$, but urea and creatinine were normal. White cell count was normal and CRP was 100 mg l$^{-1}$. Treatment was instituted with both intravenous coamoxiclav and clarithromycin. Crystalloid infusion was commenced and oxygen administered. This patient required one-to-one ratio nursing and boluses of short-acting benzodiazepines to manage his illness initially. He improved well, and the purulent, blood-stained sputum he was expectorating cultured penicillin-sensitive pneumococcus. He developed a complicated right parapneumonic effusion that has failed to resolve, despite intercostal ultrasound-guided insertion of a small-bore drain, and the patient has been referred to thoracic surgeons for further management of this complication, as he has a restrictive lung defect as a consequence with functional impairment.

Discussion
Case 1 had a CURB score of 3/4 [raised urea (>7.0 mmol l$^{-1}$) and respiratory rate (>30 breaths min$^{-1}$) and low blood pressure (systolic ≤90 and/or diastolic ≤60 mmHg)] and case 2 had a score of 1/4 (confusion). Increasing CURB score has been demonstrated to be associated with increasingly adverse outcome, particularly death (British Thoracic Society Standards of Care Committee, 2001; Lim et al., 2001). The clinical course of each patient’s illness reflected these scores, with case 1 having a more critical course.

Although rare, previously well young adults do die from CAP. Simpson et al. (2000) found that 5 % of all cases of CAP in young adults aged 15–44 years in England and Wales involved just such patients and that Streptococcus pneumoniae was the commonest identifiable causative organism. In-hospital management of cases was felt to be generally adequate. Awareness of the potential for critical illness in this population and prompt antimicrobial therapy were felt to be key issues in improving outcome.

Overall mortality from hospitalized CAP has been demonstrated to range between 4 and 14 % (British Thoracic Society Standards of Care Committee, 2001; Lim et al., 2001; Hug & Rossi, 2001). This is comparable with SARS (Drazen, 2003; Tsang et al., 2003) but, of course, SARS has been demonstrated to be highly infective to close contacts, particularly healthcare workers (Drazen, 2003; Tsang et al., 2003; Zambon & Nicholson, 2003). Nonetheless, in this time of increased alertness towards unusual respiratory pathogens, it is important that the more usual presentations of CAP are still recognized. These can cause severe respiratory illness, causing significant morbidity and mortality, yet respond well if recognized and treated promptly. Indeed, early recognition of severely ill patients and their consequent deterioration is often poor (Buist et al., 2002), yet objective triggers such as severity scores may be important in triggering a response. These cases demonstrate such points well.

References