Adult respiratory distress syndrome due to mycoplasma pneumonia

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Summary
A previously fit 48-year-old man was admitted with an acute respiratory failure due to mycoplasma pneumonia that was confirmed by raised mycoplasma titre on complement fixation test. It was also associated with disseminated intravascular coagulopathy. The patient made a full recovery but required intermittent positive pressure ventilation.

Keywords: mycoplasma, pneumonia, adult respiratory distress syndrome

Mycoplasma pneumonia is caused by Mycoplasma pneumoniae. The infection is characterised by fever, pharyngitis, cough and often multilobular infiltrate. The X-ray signs are more extensive than indicated by physical examination. Mycoplasma pneumoniae can rapidly haemolysse guineapig red cells at a low temperature with production of anti-I antibody. Therefore, the infection is also called cold-agglutinin-positive pneumonia. Mycoplasma pneumonia commonly affects people with normal lungs but invasive disease is uncommon. We describe a case of invasive mycoplasma pneumonia with respiratory distress syndrome in a patient with previously healthy lungs.

Case history
A healthy 48-year-old man who smoked 40 cigarettes per day presented with a seven-day history of fever, progressive dyspnoea, cough with muco-purulent sputum and streaky haemoptysis. He had no previous history of respiratory illness. Despite treatment with amoxycillin by his general practitioner he continued to deteriorate, necessitating hospitalisation. On admission he was pyrexial (38.5°C), centrally cyanosed, and tachypnoeic, with a respiratory rate of 46/min, pulse 120 beats/min and blood pressure 110/60 mmHg. Chest examination revealed extensive crackles throughout both lung fields. His chest X-ray showed extensive bilateral alveolar shadowing consistent with bronchopneumonia (figure).

Investigations revealed: white cell count 26.3 x 10⁹/l (neutrophils 22.7 x 10⁹/l), haemoglobin 15.3 g/dl, platelets 336 x 10⁹/l, sodium 129 mmol/l, potassium 4.2 mmol/l, urea 7.5 mmol/l, blood gases on 28% oxygen, PaO₂ 3.6 kPa, PaCO₂ 4.2 kPa, pH 7.4 and HCO₃⁻ 19.5 mmol/l. Subsequent tests showed disseminated intravascular coagulopathy (box 1), which was confirmed by clotting screening, ie, International Normalised Ratio 1:1.1, activated partial thromboplastin time 70.4 s, (reference range 30–40 s), thrombin time 150 s (9–13 s), D-dimer 8.0 µg/ml (normal <0.5 µg/ml), fibrinogen 7.1 g/l (2.2–4.8 g/l). The complement fixation test showed a rise in mycoplasma titre from 10 to >5120. The Direct Coombs test became positive due to the presence of anti-I (titre 1/60 at 18°C) and was associated with mild haemolysis.

The clinical, radiological and arterial blood gas features were consistent with adult respiratory distress syndrome (box 2). The patient was transferred to the intensive therapy unit and ventilated with intermittent positive pressure ventilation. He received parenteral erythromycin (500 mg qid) and cefotaxime (1 g bid). An initial inspired oxygen concentration (FiO₂) of 100% was associated with an arterial oxygen saturation varying between 86% and 92%. Improvement in the patient’s condition

Diagnostic criteria for disseminated intravascular coagulopathy

- raised level of fibrinogen degradation product (commonly assayed as D-dimer) in presence of significantly prolonged thrombin time
- prolonged clotting time, prothrombin time, activated partial thromboplastin time
- short clot lysis time

Box 1
resulted in a better oxygenation with a reduced inspired oxygen concentration. He was ventilated for eight days and discharged after six weeks following full recovery.

Discussion

Adult respiratory distress syndrome due to Mycoplasma infection is rare in a patient with previously healthy lungs. This syndrome occurs in association with direct and indirect lung insults, varying from fulminating bacterial infections and major traumas to neurogenic, haematological and metabolic disorders (box 3). It is asymptomatic in 25% of cases; in 75% of cases the lower respiratory tract is involved but only a small proportion of cases are fatal.

Adult respiratory distress syndrome has rarely been reported in healthy individuals. The prognosis of patients with adult respiratory distress syndrome remains poor with over 50% mortality. However, with effective ventilatory support most patients now die from sepsis or organ (e.g., kidney) failure in addition to respiratory failure. In a study by the British Thoracic Society and the Public Health Laboratory Service, of community-acquired pneumonia, out of a total of 81 deaths only four were due to mycoplasma pneumonia, none of whom received appropriate antibiotics before hospitalisation.

Respiratory infection due to mycoplasma is common in children and young adults but invasive disease is more common in patients who are immunosuppressed. Mycoplasma is an important cause of community-acquired pneumonia and can be diagnosed throughout the year. An epidemic of mycoplasma pneumonia occurs every four years and 1994 was expected to be an epidemic year. The epidemic may last up to two years and may account for approximately 20% of community-acquired pneumonias in all age groups.

Our case emphasises that, even in healthy individuals, the infection can progress rapidly, particularly if inappropriate antibiotics are administered at the onset of the illness. This reaffirms the view that one should adhere to the British Thoracic Society guidelines for antibiotic use in severe pneumonia (box 4), particularly during epidemic years.

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