A 7-year-old boy with mycoplasmal infection requiring extracorporeal membrane oxygenation

Abstract A 7-year-old boy with Down syndrome developed severe acute respiratory distress syndrome after a respiratory infection with *Mycoplasma pneumoniae* with an unusually high agglutination titre (1:10240). Initially, mechanical ventilation and nitric oxide inhalation were used, but these did not improve the alveolar-arterial oxygen gradient. Extracorporeal membrane oxygenation for 152 h improved the lung condition. Conclusion: our case suggests that *Mycoplasma pneumoniae* should be considered as an aetiological agent in acute respiratory distress syndrome. Extracorporeal membrane oxygenation might have a valuable role in the management.

Keywords Acute respiratory distress syndrome · Extracorporeal membrane oxygenation · *Mycoplasma pneumoniae* infection

Abbreviations AaDO2 alveolar-arterial oxygen gradient · ARDS acute respiratory distress syndrome · ECMO extracorporeal membrane oxygenation · iNO inhaled nitric oxide

Introduction

*Mycoplasma pneumoniae* is a major cause of respiratory infections in school-aged children and young adults. However, since *M. pneumoniae* is sensitive to antibiotics (macrolides and tetracyclines), this illness is usually mild and fatal infections are rare [4]. Acute respiratory distress syndrome (ARDS) is characterised by capillary permeability resulting in pulmonary oedema. As a self-perpetuating state of inflammatory derangement, ARDS manifests clinically as a rapid development of radiographic infiltrates, severe hypoxaemia and reduced lung compliance [3]. ARDS is caused by a variety of medical and surgical illnesses, but ARDS caused by mycoplasmal infection is very rare. To the best of our knowledge, Van Bever et al. [8] reported the only case of *M. pneumoniae* infection that progressed to ARDS in children. Their patient was mechanically ventilated for 17 days, but could be discharged with interstitial lung fibrosis after 36 days of hospitalisation. Our case appears to have been more severe, but was rescued without any sequelae by using extracorporeal membrane oxygenation (ECMO).

Case report

A 7-year-old boy with Down syndrome was referred to the University Hospital because of progressive respiratory distress. Seventeen days before admission, the patient developed cough and fever. Ten days before admission, the general practitioner diagnosed bacterial pneumonia. The patient was then admitted to a regional hospital and cefotiam hydrochloride (100 mg/kg per day) was administered intravenously. His chest X-ray film in a regional hospital showed unilateral (left side) infiltrates and atelectasis of the left upper lobe. Seven days before admission, he was intubated because of tachypnoea, dyspnoea, and hypoxia. When he was intubated, his chest X-ray film showed bilateral diffuse infiltrates. On the same day, bilateral intrathoracic drainage was performed because of pneumothorax. Just before developing pneumothorax, artificial ventilation was performed at zero PEEP with spontaneous respiration. Due to the persistence of these symptoms, the boy was referred to another general hospital, where mechanical ventilation was continued and antibiotic medication (minocycline hydrochloride, cefotaxime sodium) and steroid pulse treatment (methylprednisolone sodium succinate 30 mg/kg per day × 2 days) were administered (Fig. 1). Since his condition did not improve, he was brought to our hospital. In the first blood test upon admission to our hospital, the leukocyte count was 13.2×10⁹/l and slight anaemia...
(Hb 9.0 g/dl) was observed. Serum CRP was high (11.3 mg/dl) and blood culture was negative. Serum concentrations of IgG, IgM and IgA were 1160, 539, and 173 mg/dl, respectively. Serum ferritin level was 265 ng/ml. The passive agglutination titre for *M. pneumoniae* was unusually high (1:10240), and a cold agglutinin test was also positive (1:1024). The chest X-ray film showed diffuse bilateral infiltrates and slight pleural effusion (Fig. 2). A cardiac ultrasound test showed slight pericardial effusion, whereas pulmonary hypertension was not observed. The ejection fraction of the left ventricle was normal (0.73). A blood gas analysis showed that the alveolar-arterial oxygen gradient (AaDO₂) was extremely high (595 mm Hg) and the ratio of arterial oxygen tension to inspired oxygen fraction (PaO₂/FiO₂) was extremely low (50 mm Hg). Initially, inhaled nitric oxide (iNO) treatment was administered immediately with supportive care. However, AaDO₂ after 6 h of iNO treatment fell only to 471 mm Hg (PaO₂/FiO₂ = 90 mm Hg), which was not satisfactory (Fig. 1). Finally, venovenous ECMO, where desaturated blood was drained from the right atrium and oxygenated blood was returned through the right femoral vein, was used. Immediately after introducing ECMO, a significant improvement in gas exchange was observed (Fig. 1). Once the patient was placed on ECMO, mild mechanical ventilation was used to avoid any further barotrauma or oxygen toxicity to the lung. He was maintained at a low dose of O₂ (FiO₂ 0.4), with a PIP of 25 cm H₂O and PEEP of 12 cm H₂O (Fig. 1). Respiratory physical therapy was actively performed, including use of the prone position, to improve or prevent atelectasis. CRP began to fall beginning the 3rd day after hospitalisation, and a chest radiograph showed the resolution of opacity. ECMO was performed without any problems, and was discontinued at 152 h, after checking the patient’s condition under usual mechanical ventilation. Artificial ventilation was performed for 17 days. There was no need for oxygen medication before leaving the hospital. Immunological data were virtually normal almost 6 months later (IgG, IgM and IgA 1430, 195, and 176 mg/dl respectively, complement C3 and C4 72 and 42 mg/dl). Neurological and lung sequelae were not observed.

### Discussion

The boy described here had an unusual clinical course. Before the present illness, he was healthy and had no immunological problems. Despite antibiotic treatment, his respiratory state gradually worsened. The diagnosis of ARDS was based on severe hypoxaemia and the presence of diffuse infiltrates on the chest X-ray film. Since he did not show heart failure or pulmonary hypertension, a diagnosis of ARDS was made according to the diagnostic standards of acute lung injury and ARDS [3]. Despite recent advances in supportive care, a standard method has not yet been established to improve the prognosis of ARDS [2]. Meduri et al. [5] and Radisic et al. [6] reported that the use of steroids could potentially be effective in ARDS; however, in our case, the patient’s condition did not improve with steroid pulse treatment. ARDS in our case may have been too severe for conventional steroid pulse treatment or the dosage...