

## Case reports

# Extracorporeal Membrane Oxygenation for Legionnaires Disease: A Case Report

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### ABSTRACT

*A case of Legionella pneumonia, severe adult respiratory distress syndrome (ARDS) and multiple organ failure is described in a patient who required extracorporeal membrane oxygenation (ECMO) prior to transfer to a hospital with ECMO facilities. She eventually made a good recovery, highlighting the potential benefits of ECMO in patients with severe and refractory ARDS. (Critical Care and Resuscitation 2002; 4: 28-30)*

**Key words:** Legionella pneumonia, ARDS, ECMO, multiple organ failure

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Legionnaires disease is a common cause of community acquired pneumonia and can be severe with multi-system involvement. We describe a case of a young female with Legionella infection resulting in multiple organ failure and severe ARDS, which required ECMO prior to transfer to a hospital with ECMO facilities.

### CASE REPORT

A 32 year-old female presented to hospital in acute respiratory distress, with a four-day history of fever, myalgia, non-productive cough and breathlessness. She worked as a masseuse at a local sports club and was a moderate smoker with a past history of mild asthma and iron deficiency anaemia.

On examination she was sweaty and mildly confused. She had a temperature of 39.9°C, respiratory rate of 50 breaths per minute and oxygen saturation of 79%, breathing oxygen at 4 litres per minute. She was in sinus rhythm at a rate of 160 beats per minute and had a blood pressure of 130/80 mmHg. Chest examination revealed bi-basal inspiratory crackles. Arterial blood gases while breathing oxygen at 15 litres per minute were, pO<sub>2</sub> 60 mmHg, pCO<sub>2</sub> 23 mmHg, pH 7.50, HCO<sub>3</sub> 18 mmol/L

and SO<sub>2</sub> 94%. Her chest X-ray showed bi-basal consolidation, which extended into the mid-zones. A provisional diagnosis of severe community-acquired pneumonia was made and intravenous erythromycin 1 g 6-hourly and cefotaxime 2 g 12-hourly were commenced.

In the emergency department her respiratory function deteriorated. She required intubation and mechanical ventilation and was transferred to the intensive care unit where she developed progressive and severe hypoxia (p<sub>a</sub>O<sub>2</sub> 49 mmHg), despite an F<sub>I</sub>O<sub>2</sub> 100%, positive end expiratory pressure (PEEP) of 20 cm H<sub>2</sub>O, pressure control ventilation, sedation and muscular relaxants.

Fourteen hours after her admission, a pulmonary artery balloon floatation catheter was inserted with haemodynamic studies revealing a high cardiac output (9.66 L/min), high pulmonary artery occlusion pressure (27 mmHg) and low systemic vascular resistance (497 dynes/cm<sup>5</sup>). A noradrenaline infusion at 2 - 8 µg/min, frusemide infusion varying from 2 - 7 mg/hr and nasogastric feeding were commenced. As her core temperature ranged from 38 - 40°C, paracetamol, surface cool-

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ing and reduction of inspired gas temperature were used to reduce her pyrexia.

As the legionella urinary antigen test was positive (the legionella infection was also confirmed later by paired serology as *Legionella pneumophila* serogroup 1) the cefotaxime was discontinued on the third day of her admission and intravenous ciprofloxacin 300 mg 12-hourly and rifampicin 600 mg daily were commenced. Prone position ventilation for periods of 4 hours was used to improve arterial oxygenation,<sup>1-2</sup> with the arterial pO<sub>2</sub> on 100% oxygen increasing from 58 mmHg to 82 mmHg when she was in the prone position.

From the 4<sup>th</sup> to the 6<sup>th</sup> post admission days, as she remained in severe respiratory failure (her lowest p<sub>a</sub>O<sub>2</sub> was 48 mmHg on 100% oxygen), dexamethasone (10 mg 6-hourly) was commenced.<sup>3</sup> By the 6<sup>th</sup> post admission day she was anuric. There was evidence of rhabdomyolysis (plasma creatine kinase 3,809 IU/L), disseminated intra-vascular coagulation (INR 2.6, platelet count 57 x10<sup>9</sup>/L, FDP's 1200 ng/L, D-dimer > 2000 ng/L), ischaemia of fingers and toes, hypernatraemia (plasma sodium 165 mmol/L) and worsening hyperthermia (core temperature ranged from 41 - 42°C).

Her ARDS was unresponsive to conventional therapy, including pressure control and inverse ratio ventilation modes (using low tidal volumes), prone ventilation, high PEEP, transtracheal oxygen insufflation and aggressive fluid restriction.<sup>4</sup> Nitric oxide therapy was unavailable. On 6<sup>th</sup> post admission day, the decision was made to treat the patient with ECMO. As the patient was too unstable to transfer to a centre with ECMO facilities, the necessary expertise and equipment were provided by a private company (Perfusion Services, Melbourne, Australia).

Veno-venous ECMO was instituted using a 19 French reinforced percutaneous cannula inserted into the right femoral vein and advanced to the junction of the right atrium and inferior vena cava (used as the take-off line into the oxygenator). A second cannula was inserted into the right internal jugular vein and advanced into the right atrium (used as the return line after oxygenation). Bypass began with flow rates varying between 3 to 5 L/min with systemic anti-coagulation being achieved with intravenous heparin.

With the institution of ECMO her ventilatory requirements decreased and her oxygenation improved dramatically. Arterial blood gases after 10 minutes on bypass (F<sub>I</sub>O<sub>2</sub> 100%) were: pO<sub>2</sub> 330 mmHg (increasing from 64 mmHg), pCO<sub>2</sub> 36 mmHg, pH 7.52, HCO<sub>3</sub> 29 mmol/L. Continuous veno-venous haemofiltration (CVVH) was also used to treat her anuric renal failure, which was achieved by creating a shunt within the extracorporeal circuit with a blood flow rate of 200 mL/min.

On the 7<sup>th</sup> post admission day her severe cardiac and respiratory failure had stabilised sufficiently for her to be transferred to the nearest hospital with on-site ECMO facilities. ECMO was maintained during the 12 km transfer and was continued for a further 4 days. Her coagulopathy and rhabdomyolysis (her peak plasma creatine kinase was 52 000 IU/L) gradually improved. On the 15<sup>th</sup> day a surgical tracheostomy was inserted and the inotropic agents were discontinued.

She continued to improve slowly over the next eight weeks. By the 22<sup>nd</sup> day, her renal function improved and CVVH was discontinued. Ventilatory weaning began and by the 29<sup>th</sup> day her trachea was decannulated. She was discharged to a general ward the next day and transferred to a rehabilitation unit on the 45<sup>th</sup> day, with ongoing issues of generalised weakness, due to a myopathy/polyneuropathy of 'critical illness' and distal gangrene of her toes. She was discharged home a month later, 72 days after admission, and is now living independently at home.

## DISCUSSION

Legionnaires disease is caused by the intracellular bacterium *Legionella* and is the third commonest microbial cause of community-acquired pneumonia (3 to 15 %), after *Streptococcus pneumoniae* and *Haemophilus influenzae*.<sup>5</sup> The majority of cases are caused by *Legionella pneumophila*, which has 14 serogroups. Serogroup 1 accounts for more than 80% of the reported cases caused by *L. pneumophila*.<sup>5</sup> Common risk factors include cigarette smoking, chronic lung disease, advanced age and immunosuppression. Legionella infection can be severe, causing multiple organ failure and severe ARDS, and has a reported mortality rate varying between 10 - 20%.<sup>6-7</sup>

Acute respiratory distress syndrome is characterised by refractory hypoxaemia (p<sub>a</sub>O<sub>2</sub>:F<sub>I</sub>O<sub>2</sub> ratio < 200) and bilateral lung infiltrates caused by non-cardiogenic pulmonary oedema, and has a reported mortality of 50 - 60%.<sup>3</sup> Ventilatory strategies are aimed at maintaining oxygenation, recruiting collapsed lung units and minimising lung over-inflation, by pressure controlled ventilation, low tidal volumes, and high levels of PEEP. Fluid restriction, diuretic therapy and attention to fluid balance may assist by reducing extra-vascular lung water.

In refractory cases of ARDS, additional strategies include prone ventilation and inhaled nitric oxide. Ventilation in the prone position is thought to recruit lung units in previously dependent lung segments and may dramatically improve oxygenation.<sup>1-2</sup> Inhaled nitric oxide results in pulmonary vasodilatation (and possibly bronchodilatation), thereby reducing pulmonary hypertension and correcting ventilation/perfusion mismatch.

Nitric oxide is superior to intravenous agents (such as prostacyclin), which significantly reduce systemic arterial pressure and increase intrapulmonary shunting.<sup>8</sup>

If the above measures are unsuccessful, extracorporeal life support is a therapeutic option.<sup>4</sup> ECMO reverses hypoxaemia by exposing blood to oxygen across a semi-permeable membrane. The technique is associated with improved outcome in neonates with severe RDS, but the role of ECMO in adults with ARDS is less clear. An early randomised controlled study of veno-arterial ECMO and a more recent study of veno-venous extracorporeal CO<sub>2</sub> removal showed no benefit compared with conventional therapies.<sup>9-10</sup> Whilst uncontrolled reports of veno-venous ECMO suggest there may be a benefit in selected patients, a randomised controlled study is still awaited.<sup>11</sup> The transportation of a patient receiving extracorporeal life support is a considerable logistical exercise and significant complications with such patients have been reported.<sup>12</sup> Nevertheless, there are reports of successful outcomes of patients transferred on ECMO, both locally and internationally.<sup>12-14</sup>

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