Early Administration of Extracorporeal Life Support for Near Fatal Asthma

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Extracorporeal life support refers to an invasive technique whereby the patient’s blood is circulated extracorporeally through a membrane lung, which enables oxygenation of the blood and removal of CO2. Its use has been described in a variety of patients with severe cardiopulmonary insufficiency, but there are only a few case reports [1–5] describing its role in status asthmaticus (Table 1).

We present the case of a young woman with near-fatal status asthmaticus not relieved by conventional treatment, in whom early administration of extracorporeal membrane oxygenator resulted in a good outcome.

Patient Description

A 19 year old girl was admitted to the emergency department due to the sudden onset of dyspnea, cough and chest tightness. On admission the patient was stuporous and in severe respiratory distress. She was therefore intubated, ventilated and transferred to the intensive care unit.

Physical examination revealed limited air movement and subcutaneous emphysema around the neck and upper chest. Laboratory data including chemistry and complete blood count were within normal limits. Chest radiography demonstrated bilateral hyperinflated lungs, minimal subcutaneous emphysema and mild pneumomediastinum.

Initial ventilator settings were pressure-controlled ventilation at a rate of 8/minute, positive end-expiratory pressure of 5–10 cmH2O and inspiratory/expiratory ratio (I/E ratio) of 1.5. Inspired fraction of oxygen was initially 0.5–0.7. Ventilation with low tidal volumes of 350 ml (5 ml/kg) required inspiratory pressures of up to 60 cmH2O. An arterial blood gas showed pH 7.21, PaCO2 99 mmHg, and PaO2 477 mmHg. The patient was given repeated inhalations with bronchodilators, high dose corticosteroids, intravenous salbutamol and intravenous aminophylline. Subsequent arterial blood gas demonstrated worsening of respiratory acidosis and hypercapnia (pH 7.0, PaCO2 120 mmHg, PaO2 77 mmHg on an FIO2 of 0.6). Magnesium sulphate and sodium bicarbonate infusions were started. Despite this protocol, PaCO2 remained around 120 mmHg with pH around 7.00. The patient was started on isoflurane 2% anesthesia and ventilation on oxygen: helium mixture using a Siemens Servo 900C, but the clinical situation did not improve.

None of these treatments was able to provide adequate oxygenation and ventilation. Both resistance and compliance were poor, and blood pressure intermittently decreased to 80/40 mmHg. Tidal volumes remained around 320 ml (peak inspiratory pressure 70 cmH2O). A decision was made to employ extracorporeal lung assistance about 8 hours after admission to the intensive care unit. A polyurethane catheter was inserted into the right atrium via the right femoral vein for blood drainage using the percutaneous Seldinger technique.

FIO2= inspired fraction of oxygen

References


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Table 1. Case reports of extracorporeal life assistance in severe asthma

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<tbody>
<tr>
<td>Gender</td>
<td>Female</td>
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<td>Male</td>
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<tr>
<td>Age (yrs)</td>
<td>32</td>
<td>62</td>
<td>23</td>
<td>23</td>
<td>18</td>
<td>39</td>
<td>60</td>
<td>19</td>
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<td>Asthma status</td>
<td>Severe, frequent</td>
<td>No prior use of steroids</td>
<td>Mild to moderate asthma</td>
<td>Severe, frequent</td>
<td>Severe</td>
<td>Mild, infrequent</td>
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<tr>
<td>Clinical presentation</td>
<td>Comatose, cyanotic</td>
<td>Comatose</td>
<td>Stuporous</td>
<td>Cyanotic, unconscious</td>
<td>Cyanotic, severely dyspnic</td>
<td>Coma</td>
<td>Cyanosis, stupor</td>
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<tr>
<td>ABG (mmHg)</td>
<td>PaO₂ 22</td>
<td>PaCO₂ 395 (on 100% O₂)</td>
<td>PaCO₂ 153</td>
<td>PaCO₂ 50 (on 100% O₂)</td>
<td>PaCO₂ 221</td>
<td>PaCO₂ 80</td>
<td>PaCO₂ 119</td>
<td>O₂ Sat 86%</td>
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<tr>
<td></td>
<td>PaCO₂ 77</td>
<td>PaCO₂ 63.8 pH 7.168</td>
<td>PaO₂ 101 pH 6.86</td>
<td>PaO₂ 70 pH 7.02</td>
<td>PaO₂ 429 pH 6.87</td>
<td>PaO₂ 90 pH 7.10</td>
<td>PaO₂ 412 pH 7.02</td>
<td>PaCO₂ 120 pH 7.00</td>
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<tr>
<td>Treatment</td>
<td>Epinephrine, aminophylline, steroids, beta agonists, halothane</td>
<td>Aminophylline, steroids, beta agonists, high dose benzodiazepine and muscle relaxants</td>
<td>Epinephrine, beta2 agonists, steroids, aminophylline bicarbonate</td>
<td>Inhalation anesthesia, bronchodilators, steroids</td>
<td>Procedural anesthesia, IV isotropic, aminophylline, steroids, isoflurane anesthesia</td>
<td>Inhalation, SC</td>
<td>Inhalation, IV steroid, aminophylline, halothane, ketamine, isoflurane,</td>
<td>Inhalation, IV beta agonists, steroids, aminophylline, epinephrine,</td>
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<td>Pre-ECMO ventilation (Vt= tidal volume)</td>
<td>FIO₂ 1</td>
<td>PIP 65 cmH₂O Vt 600 ml</td>
<td>PIP 65 cmH₂O Vt 12 mL/kg PE ratio 1:2</td>
<td>PIP 70 cmH₂O Vt 500 ml</td>
<td>PIP 70 cmH₂O Vt 8 mL/kg RR 20/min</td>
<td>FIO₂ 1</td>
<td>PIP 150-170 cmH₂O I:E ratio 1:3</td>
<td>FIO₂ 1</td>
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<td>Complications (on standard treatment)</td>
<td>Pneumothorax, atelectasis (thick secretions), cardiac, arrhythmias, hypotension</td>
<td>None</td>
<td>Subcutaneous emphysema</td>
<td>Massive subcutaneous emphysema, oliguria</td>
<td>Severe subcutaneous and mediastinal emphysema, hypotension</td>
<td>Atelectasis, hypotension, hypoxemia, MRSA infection</td>
<td>Subcutaneous mediastinal &amp; abdominal emphysema, hypotension</td>
<td>Subcutaneous emphysema</td>
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<tr>
<td>ECMO type</td>
<td>Veno-arterial</td>
<td>Venovenous</td>
<td>Portable venovenous</td>
<td>Portable venovenous</td>
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<td>Timing of ECMO</td>
<td>8th day</td>
<td>After 2 days</td>
<td>12 hours after intubation</td>
<td>After 15 hours</td>
<td>After 48 hours</td>
<td>After 4 days</td>
<td>?</td>
<td>8 hours after admission</td>
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<tr>
<td>Post-ECMO complications</td>
<td>Massive intrapulmonary hemorrhage, empyema</td>
<td>None</td>
<td>None</td>
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<td>ECMO results</td>
<td>Improved hemodynamics, improved ventilation, discharged on 57th day</td>
<td>Immediate disappearance of wheezing, improved ventilation, normalization of PaCO₂ and pH ICU discharge after 5 days</td>
<td>Patient extubated 36 hr after ECMO initiated</td>
<td>Improved ventilation, dramatic improvement in urine output, discharged after 15 days</td>
<td>Improved lung compliance, mucus plugs removed, improved ventilation, discharged after 15 days</td>
<td>Improved compliance, gas exchange and clearance of secretions, discharged 12 days after intubation</td>
<td>Improved compliance, discharged after 9 days after ECLS disconnected</td>
<td>Improved hypercapnea &amp; hypotension Discharged 2 days after ECLS disconnected</td>
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nique, and another catheter was inserted into the right atrium via the other femoral vein for blood return (venovenous ECMO). PaCO₂ was maintained at about 50 mmHg. During the ECMO, blood flow was maintained at about 3 L/minute. This enabled oxygenation to be maintained even at dramatically reduced mechanical ventilation parameters. The patient was maintained with a mechanical respiratory rate of 4–6 breaths per minute, pressure controlled ventilation with PEEP 10 cmH₂O, and inspiratory pressure 15–25 cmH₂O. These parameters led to a tidal volume of 250–350 ml, which slowly increased over the next 2 days.

Two days after admission to the ICU, the patient was weaned off ECMO. At this time, ventilation was improved, although there was still a significant hypercarbia (PaCO₂ 73 mmHg, pH 7.34). The ventilatory parameters were peak inspiratory pressure 30–40 cmH₂O, tidal volume 500–600 ml, FiO₂ 0.3, PEEP 8 cmH₂O. The patient was stable hemodynamically without need for inotropic support. There were no hemorrhagic, pulmonary or hematologic complications.

The patient was extubated a week after admission. She was discharged home from a rehabilitation ward 60 days after her first admission to hospital. Five months later she was doing well and her asthma was clinically stable.

**Comment**

Severe asthma crisis should be treated aggressively before it exacerbates. Optimal treatment includes nebulized or intravenous beta-2 agonists, subcutaneous epinephrine, nebulized anticholinergics, intravenous corticosteroids, and sometimes intravenous aminophylline and magnesium sulphate. In refractory asthma, patients can be supported by bronchodilating inhalational anaesthetics (isoflurane, halothane) or by oxygen and helium mixture (heliox), which decreases airway resistance. Mechanical ventilation is an effective tool to correct hypoxemia and hypercapnea, but prolonged positive pressure ventilation with high end-inspiratory pressures causes barotrauma and volutrauma and can result in a permanent deterioration of pulmonary function. Although we applied the maximal supportive care in this patient, lung function continued to deteriorate severely, the patient was progressively more hypoxemic and hypercarbic, and appearance of barotrauma was noted. For these reasons, venovenous ECMO was implemented 8 hours after her admission to the ICU. This was done before major complications of barotrauma (pneumothorax) or other complications (arrhythmias, resistant hypotension) supervened.

Clinicians should remember that ECMO, although costly and invasive, can be a last resort treatment in carefully selected patients, shifting them from decompensated to compensated status asthmaticus. Clearly, the use of ECMO can only be regarded as a bridge to recovery of lung function, while allowing the lungs some ‘rest’ from aggressive and damaging mechanical ventilation.

The complications of ECMO are considerable. This is a highly invasive technique with associated morbidity and mortality. Application of ECMO should therefore only be performed by clinicians experienced in its use and with dealing with the possible complications.

**References**


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**Civilization is a movement and not a condition, a voyage and not a harbour**

A.J. Toynbee (1889-1975, British historian)

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**Capsule**

**Tumor radiotherapy**

Nearly half of all cancer patients are treated with radiation therapy. The magnitude of the tumor response to ionizing radiation is thought to be determined primarily by the death rate of tumor stem cells. Garda-Barros et al. show instead that endothelial cells within the tumor play a major role in determining radiation sensitivity. Murine tumors became radiation-resistant when their endothelial cells were made resistant to ionizing radiation by genetic inactivation of acid sphingomyelinase, an enzyme required for endothelial cell apoptosis. These results suggest that optimal targeting of tumors by radiotherapy may have to take into account endothelial responses.

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