
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 cm H₂O 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 147 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 hypercapnea (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 tech-

FIO₂= inspired fraction of oxygen

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

Case report	Ann Thorac Surg, 1981 [2]	Intern Care Med, 1988 [5]	Chest, 1993 [1]	Ann Thorac Surg, 1996 [3]	Am J Emerg Med, 1997 [4]-Case 1	Am J Emerg Med, 1997[4]-Case 2	Am J Emerg Med, 1997[4]-Case 3	Our patient
Gender	Female	Female	Female	Male	Female	Female	Female	Female
Age (yrs)	32	62	23	23	18	39	60	19
Asthma status	Severe, frequent		No prior use of steroids		Mild to moderate asthma	Severe, frequent	Severe	Mild, infrequent
Clinical presentation	Comatose, cyanotic	Comatose	Stuporous	Cyanotic, unconscious	Cyanotic, severely dyspneic	Dyspneic, cyanotic, comatose	Coma	Cyanosis, stupor
ABG (mmHg)	PaO ₂ 22 PaCO ₂ 77 pH 7.07	PaO ₂ 395 (on 100% O ₂) PaCO ₂ 63.8 pH 7.168	PaCO ₂ 153 PaO ₂ 101 pH 6.86	PaCO ₂ 100 PaO ₂ 50 (on 100% O ₂) pH 7.02	PaCO ₂ 221 PaO ₂ 429 pH 6.87	PaCO ₂ 80 PaO ₂ 92 pH 7.10 Sat 70%	PaCO ₂ 119 PaO ₂ 412 pH 7.02	O ₂ Sat 86% PaCO ₂ 120 pH 7.00
Treatment	Epinephrine, aminophylline, steroids, beta agonists, halothane	Aminophylline, steroids, beta agonists, high dose benzodiazepine and muscle relaxants	Epinephrine, beta2 agonists, steroids, aminophylline bicarbonate	Inhalation anesthesia, bronchodilators, steroids	Procaterol inhalation, IV isoproterenol, aminophylline, steroids, isoflurane anesthesia	O ₂ , salbutamol inhalation, IV hydrocort.+ aminophylline, Isoflurane, ketamine	Inhalations, SC Epinephrine, IV steroids+ aminophylline, halothane	Inhaled+ IV beta agonists, steroids, epinephrine, aminophylline, MgSo ₄ , ketamine+ isoflurane, He-O ₂ mixture
Pre-ECMO ventilation (Vt= tidal volume)	FiO ₂ 1 PIP 55 cmH ₂ O Vt 600 ml	PIP 65 mH ₂ O Vt 12 ml/kg I/E ratio 1:2	PIP 80 mH ₂ O Vt 500 ml	PIP 70 cmH ₂ O Vt 8 ml/kg RR 20/min	FiO ₂ 1 PIP 30–40 cmH ₂ O I:E ratio 1/3 Vt 150–170 ml	FiO ₂ 1 PIP 50–60 cmH ₂ O I:E ratio 1/3	FiO ₂ 1 PIP 50–60 cmH ₂ O Vt 250–300 ml	PIP 60–70 cmH ₂ O FiO ₂ 1 Vt 350 ml I:E ratio 1/4
Complications (on standard treatment)	Pneumothorax, atelectasis (thick secretions), cardiac arrhythmias, hypotension	None	Subcutaneous emphysema	Massive subcutaneous emphysema, oliguria	Severe subcutaneous and mediastinal emphysema, hypotension	Atelectasis, hypotension, hypoxemia, MRSA infection	Subcutaneous mediastinal & abdominal emphysema hypotension	Subcutaneous emphysema
ECMO type	Veno-arterial	Venovenous	Portable venovenous	Portable venovenous	Venovenous	Veno-arterial	Venovenous	Venovenous
Timing of ECMO	8th day	After 2 days	12 hours after intubation	After 15 hours	After 48 hours	After 4 days	?	8 hours after admission
Post-ECMO complications	Massive intrapulmonary hemorrhage, empyema	None	None	None	None	None	Subcutaneous hematoma	None
ECMO results	Improved hemodynamics, improved ventilation, discharged on 57th day	Immediate disappearance of wheezing, Improved ventilation, normalization of pCO ₂ and pH. ICU discharge after 5 days.	Patient extubated 36 hr after ECMO initiated	Improved ventilation, dramatic improvement in urine output, discharged after 15 days	Improved lung compliance, mucus plugs removed, improved ventilation, discharged 12 days after intubation	Improved compliance, gas exchange and clearance of secretions. Discharged 9 days after ECLS discontinued	Improved hypercapnea & hypotension Discharged 2 days after ECLS disconnected	Improved hypercarbia and ventilation Discharge from ICU 5 days after ECLS disconnected

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 0.5, 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 anesthetics (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 asth-

maticus. 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.

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