CASE REPORT

Extracorporeal membrane oxygenation (ECMO) for near-fatal asthma refractory to conventional ventilation

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SUMMARY

We describe a case of near-fatal asthma, treated successfully by initiation of extracorporeal membrane oxygenation (ECMO). A 29-year-old woman, known asthmatic on steroid inhalers, inhaled/nebulised bronchodilators, long-term oral prednisolone, theophylline and montelukast, presented with acute shortness of breath. She deteriorated following initial treatment with nebulised bronchodilators and magnesium sulfate requiring intubation and mechanical ventilation. Severe bronchospasm ensued following mechanical ventilation and peak airway pressures remained at 55 cm H₂O with intrinsic positive end expiratory pressure (PEEP) of 14 cm H₂O. Despite treatment with sedation, paralysis, intravenous salbutamol and inhaled sevoflurane, her condition deteriorated. She was commenced on mobile ECMO by the retrieval team. While on ECMO, her CO₂ normalised within 48 hours. She was extubated within 72 hours of initiating ECMO and was discharged to the ward next day. We reiterate that ECMO should be considered sooner for status asthmatics not responding to maximal

pharmacological therapy and ventilatory support to prevent ongoing lung injury and mortality.

BACKGROUND

Approximately 2%–4% of all patients hospitalised for acute asthma develop respiratory failure and require invasive mechanical ventilation.^{1 2} Seven per cent of mechanically ventilated patients with asthma will not survive.¹ Status asthmaticus has been recognised as an indication for extracorporeal membrane oxygenation (ECMO) in adult respiratory failure and appears to be associated with greater survival than other indications for ECMO. Compared with 50.8 % of non-asthmatics on ECMO, 83.3% of asthmatics survived to hospital discharge.³ Although ECMO has been widely used in cardiogenic circulatory shock and respiratory insufficiency secondary to severe acute respiratory distress syndrome, its use in asthma is limited and only a few case reports exist to support the use of ECMO. We present a case of a 29-year-old woman presenting with near-fatal asthma whose condition deteriorated despite all possible treatments including mechanical ventilation and eventually got better dramatically while on ECMO.

CASE PRESENTATION

A 29-year-old woman presented to the emergency department via ambulance following an emergency call for increasing shortness of breath. The patient was a known asthmatic on inhalers, home nebulisers, long-term prednisolone (30 mg), theophylline and montelukast. There was history of four previous critical care admissions, none of which had ever required invasive ventilation. Her medical history included migraine and previous intravenous drug abuse. Recent history from accompanying partner suggested she has been treated in the community for a 'chest infection' with oral antibiotics. She was cyanosed when seen by paramedical staff and was given nebulised salbutamol and epinephrine in the ambulance. On arrival in the emergency department, the patient was screaming that she cannot breathe. Her respiratory rate was 34 and she was saturating 93% on air. She was able to complete short sentences. Clinical examination of the chest revealed bilateral diffuse wheeze with poor air entry.

Figure 1 Portable, supine anteroposterior chest X-ray demonstrating hyperinflated lungs with generally coarsened bronchovascular markings. No consolidation or pneumothorax seen. Tip of endotracheal tube seen 5.5 cm above carina.

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Table 1 Serial arterial blood gases at admission and after initial treatment

	Emergency department admission	After initial treatment	Critical Care Unit admission on ventilator	After 4 hours of intubation and ventilation	Deterioration on maximal therapy
Time	16:13	17:05	20:10	23:46	03:49
FiO ₂	60%	100%	60%	60%	100%
pH	7.12	6.9	7.08	7.15	6.91
pO ₂ (kPa)	6.18(venous)	21.3	26.9	17.9	11.0
pCO ₂ (kPa)	14.1	31.7	16.1	10.05	22.9
HCO ₃ (mmol/L)	22.8	14.6	22.2	20.8	18.5
Lactate (mmol/L)	0.9	2.3	0.4	1.2	2.0

INVESTIGATIONS

Initial blood gas revealed hypercapnia with respiratory acidosis. Full blood count, urea and electrolytes were unremarkable. Chest X-ray (figure 1) revealed hyperinflation but no consolidation or pneumothorax. Serial arterial blood gases at admission and after initial treatment showed deterioration despite maximal medical and ventilatory therapy (table 1).

TREATMENT

She was initially treated with nebulised salbutamol in repeated doses with a total of 15 mg, ipratropium of 0.5 mg, intravenous hydrocortisone 100mg and intravenous magnesium sulfate 2gm. After commencement of initial treatment, the patient had a rapid deterioration with CO₂ rising to 31.2 kPa and pH falling to 6.9. She became deeply cyanosed with very poor respiratory effort and bag valve mask ventilation was undertaken. A rapid sequence induction was undertaken using ketamine and rocuronium followed by an uneventful grade 1 intubation. Severe bronchospasm ensued following intubation and positive pressure ventilation. Following administration of a total of 350 µg diluted epinephrine in small increments and two 250 µg boluses of intravenous salbutamol, the patient received sufficient tidal volumes and oxygenation to enable transfer to the critical care unit. On arrival in the critical care unit, an initial arterial blood gas revealed pH 7.05, PaCO₂ 16.1 kPa and PaO₂ 26.9 kPa with ventilator settings of FiO₂ 60%, tidal volumes 300 mL, positive end expiratory pressure (PEEP) 0 and I:E ratio of 1:4. Treatment on arrival in the critical care unit consisted of intravenous propofol and alfentanil for sedation and cisatracurium to ensure ongoing paralysis. Regular salbutamol and ipratropium nebulisation was undertaken and the patient was commenced on intravenous ketamine and salbutamol infusions; however, despite all these measures, peak airway pressures remained at 55 cm H₂O with intrinsic PEEP of 14 cm H₂O. There was initial improvement in gas exchange to pH 7.14, PCO₂ 10.5 kPa, PO₂ 17.9 kPa on the above ventilator settings but with a persistent high peak airway pressure and high intrinsic levels of PEEP. Over the course of the evening, the ketamine infusion was increased to the maximum rate (45 µg/kg/min) along with continued intravenous and nebulised salbutamol, hence receiving further 15 mg nebulised salbutamol, 12.3 mg intravenous salbutamol and 0.5 mg ipratropium while intubated and ventilated. The patient remained with full neuromuscular blockade with a train-of-four count of zero. Despite this, airway pressures remained high and gas exchange worsened; on above settings and FiO₂ of 100%, her blood gases showed pH 6.8, PaCO₂ 22.9 kPa and PaO₂ 11 kPa. The patient was switched to a Maquet anaesthesia machine with the previous settings and 8% inhaled sevoflurane was added, which made minimal difference to the situation. Due to her high intrinsic PEEP and inability to mechanically ventilate successfully with

rising CO₂, she was referred to ECMO centre for initiation of ECMO. The patient was retrieved from the Royal Stoke University Hospital and commenced on ECMO during transfer.

OUTCOME AND FOLLOW-UP

Patient received venovenous ECMO for 48 hours and was extubated at 72 hours. Intense medical treatment continued during ECMO. Trial off ECMO was successful on the second day and ECMO cannula was removed on the second day with endotracheal extubation on the third day. She was discharged back to the referring hospital on the fourth day with 2 L of oxygen via nasal cannula. There were no haemorrhagic or neurological complications. She was later discharged home with planned follow-up as outpatient.

DISCUSSION

Extracorporeal membrane oxygenation is a technological intervention that provides respiratory and circulatory support allowing gas exchange to occur outside the body.⁴ It is a modified adaptation of conventional cardiopulmonary bypass techniques developed for use during cardiac surgery. The use of ECMO in patients with severe acute respiratory failure first took place in the 1970s in California⁵ and was introduced in the UK in 1989. The first case reported to be successfully treated with ECMO was published in 1981.⁶ Status asthmaticus has been recognised as an indication for ECMO in adult respiratory failure and appears to be associated with greater survival than other indications for ECMO. Compared with 50.8 % of non-asthmatics on ECMO, 83.3% of asthmatics survived to hospital discharge.⁵ Although ECMO has been widely used in cardiogenic circulatory shock and severe respiratory insufficiency, its use in asthma is limited and only a few case reports exist to support the use of ECMO. The multicentre trial of conventional ventilatory support vs extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR) found that transfer to an ECMO centre reduced

Learning points

- Difficult asthma may present with acute exacerbation leading to profound hypercapnia refractory to pharmacological treatment and mechanical ventilation.
- Near-fatal asthma not responding to maximal pharmacological therapy and full ventilatory support should be considered for extracorporeal membrane oxygenation (ECMO) to prevent lung injury and reduce patient mortality.
- Our case reiterates the fact that early treatment with ECMO in status asthmaticus refractory to mechanical ventilation is associated with excellent outcome without any complications.

the chance of death or severe disability at 6 months in adults with advanced respiratory failure; however, this was primarily aimed at patients with acute respiratory distress syndrome.⁷ Our case reiterates the fact that early treatment with ECMO in status asthmaticus refractory to mechanical ventilation is associated with excellent outcome without any complications.

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