

A case of near fatal asthma: The role of ECMO as rescue therapy

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Abstract:

We report a case of an adolescent with near fatal asthma (NFA). He presented with severe hypoxemia and life-threatening acidemia, who failed to respond to conventional therapy. His hospital course was complicated by barotrauma and hemodynamic instability. Early introduction of extracorporeal membrane oxygenation (ECMO) led to dramatic improvement in gas exchange and lung mechanics. This case illustrates the important role of ECMO as salvage therapy in NFA.

Key words:

ECMO, near fatal asthma, rescue therapy

Near fatal asthma (NFA) is a serious medical condition that often results in profound hypoxemia, hypercapnia, and altered mental status.^[1] Patient with NFA breathe at high lung volumes leading to increased mechanical load and elastic work of breathing.^[2] Mechanical ventilation is often required to manage asthmatic patients who deteriorate despite aggressive management; however, it can have deleterious effects due to worsening dynamic hyperinflation and increase intrathoracic pressure. The reported mortality rate in patients who require ventilator assistance is 8%.^[3] ECMO is an alternative method of cardio-pulmonary support in which oxygen is added and carbon dioxide (CO₂) is removed through an extracorporeal membrane using arteriovenous or veno-venous cannulation.^[4] The goal of this therapy is to minimize ventilator-induced lung injury and allow ample time for the lung inflammatory process to subside. Its use in severe asthma is limited to case reports or a case series.^[5,6] In this case, we report a patient with NFA who had hypoxemia that was refractory to conventional therapeutic modalities and was managed by ECMO.

decreased breath sounds and bilateral wheeze. Chest X-ray was normal. Complete blood count (CBC) and other blood tests were normal.

He was started on IV fluids; nebulizations with Salbutamol, Ipratropium Bromide, Budenoside, and Oxygen; intravenous (IV) Magnesium Sulphate, antibiotics, and high-dose IV steroids. He was also placed on Bi-Level Positive Airway Pressure to reduce the work of breathing (IPAP14 and EPAP 8, FiO₂ of 70%). However, clinically unchanged, with repeat ABGs pH 7.20, pCO₂ 44 mmHg, pO₂ 70 mmHg, HCO₃ 18 mEq/L, and his serum lactate level was 3.1 mmol/L. Therefore, he was intubated and transferred to the intensive care unit (ICU).

The patient was started on assist control mode, with respiratory rate of 18, tidal volume 5ml/kg, FiO₂ 100%, I:E ratio of 1:4, and positive end-expiratory pressure (PEEP) 5cmH₂O. He was sedated with propofol, midazolam, fentanyl, IV muscle relaxant (cis-atracurium), norepinephrine 10 mcg/min, and salbutamol infusion at 5µg/min. Despite ventilator manipulations to decrease minute ventilation and allow permissive hypercapnia, his peak and plateau airway pressure were persistently high at 57 and 40 cmH₂O, respectively. Sodium bicarbonate infusion was started due to severe concomitant metabolic acidosis. About 8 hours later, he developed surgical emphysema and bilateral chest tubes were placed prophylactically [Figure 1]. Bronchoscopy and bronchoalveolar lavage were performed to remove any mucus plugs; however, none were seen.

Despite being on these measures, his respiratory status continued to deteriorate, so the decision

Case Report

A 15-year-old boy known to have asthma presented to the emergency room with one day history of cough and shortness of breath (SOB). On clinical examination, he was diaphoretic, tachypneic, and using his accessory muscles, blood pressure (BP) 120/70 mmHg, pulse 110/min, RR 42/min, temperature 37°C, and oxygen saturation was 92% on FiO₂ of 6L/min. His initial arterial blood gas analysis (ABGs) was pH 7.35, PCO₂ 37 mmHg, PO₂ 64 mmHg, HCO₃ 25 mEq/L. Chest examination revealed

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was made to start him on ECMO, using the left femoral veno-venous route with heparin infusion and monitoring of activated clotting time (150-200 s). His ABGs improved over the next 2 hours [Table 1]. Ventilator settings were weaned to assist control mode, tidal volume 6 ml/kg, rate 14/min, FiO₂ 40%, and PEEP 5 cmH₂O. ECMO was continued for 72 hours; meanwhile, the patient was kept sedated and ventilated; continued on antibiotics, nebulization, steroids, and IV salbutamol, along with heparin infusion, norepinephrine, and cis-atracurium. After 3 days, ECMO was discontinued with no consequences. On the fifth day, he was extubated and transferred to the ward and was discharged home on the sixteenth day after admission to be followed up in the pulmonary clinic.

Discussion

The severe derangement in gas exchange of this patient was managed beyond the current clinical practice guidelines. He had persistent extreme respiratory embarrassment and ventilatory failure despite being on maximum conventional therapy including

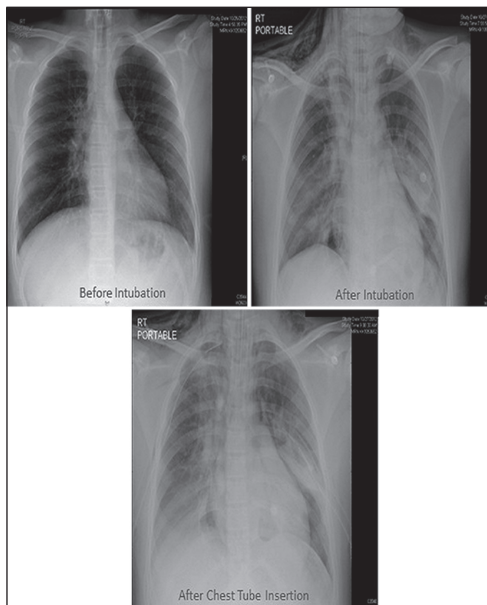


Figure 1: Comparison of chest x-rays on admission before and after tube insertion

mechanical ventilation. He also sustained both barotrauma and hemodynamic instability requiring vasopressors. The mechanism of hypoxemia and hypercapnia in acute asthmatic attack is mainly due to low ventilation perfusion, mismatch, shunting, and hypoventilation.^[2,7] The application of ECMO in this case has provided a bypass where extra-corporeal blood flow through a membrane has achieved adequate oxygenation and CO₂ removal. This was necessary to rest the lung, until the inflammation causing his bronchospasm has subsided.

ECMO has been used primarily to treat respiratory failure due to acute lung injury (ALI) that failed to respond to maximal medical therapy and its use in severe asthma is unusual.^[5,7,8] In a recent report of acute respiratory failure due to influenza A (H1N1) epidemic in 2009, applying ECMO to these patient was associated with favorable results.^[9]

In a retrospective cohort study, using life support organization (ELSO), Mikkelsen *et al.*, found that among 1,257 patients treated with ECMO, 24 (1.9%) were asthmatics of whom 20 (83.3%) survived to hospital discharge, compared to 1,233 (50.8%) non-asthmatics.^[10] The success of ECMO in asthma was likely due to the natural reversibility of the airflow obstruction in asthma. This is in contrast to the patient with diffuse alveolar damage due to ALL, where the recovery is usually slow.^[10] Moreover, by virtue of its mechanism of action, applying ECMO allows reduction of both the tidal volume and minute ventilation, which subsequently decrease the dynamic hyperinflation. These further reduce the development of barotraumas and hemodynamic instability particularly with persistent hypoxemia and acidemia, and who appear to have extremely severe disease.

ECMO complications can arise during catheter insertion or during patient's management. It is vital that operators are aware of these complications, to allow taking swift action to appropriately remedy the situation. The other common complication is bleeding, either due to fatal vascular perforation during cannulation or secondary to heparin infusion or platelets dysfunction.^[4,8] Cerebral hemorrhage or infarction occurs in approximately 10–15% in patient with acute respiratory distress syndrome (ARDS). Systemic thromboembolism due to thrombus formation within the extracorporeal circuit is an infrequent but important complication.^[4]

Table 1: Ventilator parameters and arterial blood gas analysis

	On admission	Immediately before ECMO*	Immediately after ECMO*	Before weaning from ECMO*	After weaning from ECMO*
Ventilator parameters					
PEEP (cmH ₂ O)	5	5	5	5	5
PEEPi (cmH ₂ O)	12	15	12	10	7
P plateau (cmH ₂ O)	40	50	35	23	22
FiO ₂ (%)	100	100	80	40	50
Arterial blood gas					
pH	7.25	6.92	7.38	7.40	7.40
PaCO ₂ (mmHg)	48	92	35	45	45
PaO ₂ (mmHg)	62	76	61	70	62
HCO ₃ (mmol/L)	20	14	21	26	27
SaO ₂ (%)	90	95	92	95	93

PEEP = Positive end expiratory pressure, PEEPi = Intrinsic positive end expiratory pressure, P plateau = Plateau pressure at end of inspiration, FiO₂ = Fraction of inspired oxygen, PaO₂ = Partial pressure of oxygen, PCO₂ = Partial pressure of carbon dioxide, SaO₂ = Oxygen saturation, *ECMO = Extracorporeal membrane oxygenation

Conclusion

Successful management of patient with NFA in ICU requires high level of expertise and intensive support. Aggressive therapy, including careful manipulation of mechanical ventilation, and possible use of ECMO in the proper setting can be life-saving.

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