

Case Report

Impairment of Venous Drainage on Extracorporeal Membrane Oxygenation Secondary to Air Trapping in Acute Asphyxial Asthma

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Abstract: The inability to adequately support a patient on extracorporeal membrane oxygenation (ECMO) due to impaired drainage is not an uncommon occurrence during support. Typically, the causes include hypovolemia, kinks in the circuit, cannula malposition, or inadequate cannula size. In this report we present an uncommon etiology of this problem. A 3-year-old female presented to our hospital in status asthmaticus and pulseless electrical activity (PEA). This was a result of dynamic hyperinflation of the lungs causing physical obstruction of venous return to the heart.

Upon initiating venoarterial (VA) ECMO, we experienced inadequate drainage that did not improve despite multiple interventions. This resolved with the addition of an inhaled anesthetic gas to treat this patient's severe bronchospasm. This case illustrates the importance of considering a patient's physiology or disease state and how that may affect the mechanics of ECMO support. **Keywords:** extracorporeal membrane oxygenation, ECMO, air trapping, venous obstruction, asthma, status asthmaticus, inhalational anesthetics. *JECT. 2015;47:109–112*

Acute severe asphyxial asthma is a life-threatening event caused by the rapid onset of an asthmatic episode that responds poorly to conventional therapy (1,2). Although less frequent than most asthma exacerbations, these episodes often are so serious that they may require mechanical ventilation or even extracorporeal membrane oxygenation (ECMO) for support (3–5). During these episodes, bronchospasm may be so severe that venous return to the heart is impaired. This tamponade-like physiology leads to impaired cardiac filling, decreased cardiac output, obstructive shock, and potentially, cardiac arrest, if left untreated (2,3,6). Mechanical ventilation may improve oxygenation, ventilation, and work of breathing. However, this modality may increase the obstructive component due to increased air trapping exacerbated with bronchospasm (3,4,6). In fact, mechanical ventilation carries the risk of exacerbating this condition due to worsening of air trapping, acceleration

of the tamponade physiology, and possible subsequent cardiac arrest (1–3,6–8).

Inhaled volatile anesthetic gas has been suggested as a treatment for patients with acute severe asphyxial asthma (9). Case reports and case series have shown that inhaled anesthetic gases improve the partial pressure of arterial carbon dioxide (pCO₂), arterial pH, and ventilator pressures (9,10). However, its use can be complicated by the need for specialized equipment and expertise in both delivery and scavenging of the gas. In addition, inhaled anesthetic gases are well known to cause cardiovascular depression and hypotension (9,10).

ECMO has also been suggested as a method of support for acute, severe, reversible respiratory failure secondary to status asthmaticus (3,4). ECMO allows for gas exchange while potentially avoiding ventilator-induced lung injury (3).

In this report, we detail the events of a patient who presented with acute severe asphyxial asthma who, despite multiple medical interventions, developed respiratory acidosis with a rising lactate and was felt to be moribund. She was placed on venoarterial (VA) ECMO. The patient subsequently required inhaled anesthetic gas to relieve the bronchospasm that was exacerbating the dynamic

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hyperinflation of the lungs resulting in mechanical compression of the heart inhibiting venous drainage.

DESCRIPTION

A 3-year-old 15-kg female presented to an outside facility with a severe asthmatic episode. The patient had a history of poorly controlled asthma despite being treated with montelukast and albuterol nebulizations on a chronic basis. The patient's family reported that in the previous evening the child developed severe wheezing while outside with her siblings as the father mowed the lawn. The patient received several albuterol nebulizations that evening with minimal relief of symptoms and was brought to an outside emergency room in severe respiratory distress.

Upon arrival to the emergency room, the patient's pulse oximetry was 60%, but increased to 90% with the administration of 100% oxygen by face mask and continuous aerosol therapy. A left antecubital peripheral intravenous line was placed and she was given 2 mg/kg of intravenous (IV) methylprednisolone, .01 mL/kg of 1:1000 epinephrine subcutaneously, and a 20 mL/kg normal saline bolus. At this time the decision was made to air transport the patient to our facility for potential ECMO support. Before transport the patient was intubated with a 5.0 uncuffed endotracheal tube (ETT) and ventilated with 100% FiO₂, positive end-expiratory pressure (PEEP) of 5 mmHg, tidal volume of 10 mL/kg, inspiratory time of .8 seconds, and respiratory rate of 25 breaths per minute.

During air transport, the patient experienced deterioration with saturations decreasing to 80%, therefore, the transport team began to manually ventilate the patient using an appropriate-sized self-inflating bag with 100% oxygen. Despite further treatment with .3 mL of 1:1000 epinephrine given subcutaneously and 2.5 mg of albuterol through the ETT, the patient's saturations continued to decline to 60%. Just before landing at our facility, the transport team noted that the patient was in pulseless electrical activity (PEA) and began cardiopulmonary resuscitation (CPR). The transport team was met by our pediatric critical care team and pediatric intensive care unit (PICU) physician who upon assessment noted a lack of chest rise during ventilation. The ETT was removed and the patient's chest was manually decompressed due to concerns about air trapping causing cardiac tamponade. After the chest was decompressed, the patient was manually ventilated via bag-valve mask with 100% oxygen. Chest compressions were started and performed throughout the transport of the patient to the PICU. It was noted that the patient had fixed and dilated pupils and was not spontaneously moving during transport to the PICU. Upon arrival to the PICU, the patient was found to be in sinus tachycardia, with palpable carotid pulses. The patient was intubated with a 5.0 cuffed ETT

and continued to be manually ventilated. A triple-lumen central line was placed in the right femoral vein and a right radial arterial line was also placed in the patient. The patient was placed on an anesthesia ventilator (Datex Ohmeda Aestiva 3000; GE Healthcare, Wauwatosa, WI) and ventilated on 100% FiO₂, PEEP of 4 cm H₂O, respiratory rate of 20 breaths per minute, and tidal volume of 9 mL/kg, which resulted in a peak inspiratory pressure (PIP) of 51 cm H₂O. Isoflurane was delivered to the patient at 1.5% (Isotec 5, GE Healthcare, Wauwatosa, WI). The patient's PIP decreased to 40 cm H₂O, however, the patient had a persistent hypercapnia with a pCO₂ of 101 mmHg and a rising lactate from 4.7 mEq/L to 5.5 mEq/L. Because of these worsening values the decision was made to place the patient on VA ECMO. The patient was disconnected from the anesthesia ventilator and placed on a Servo-I[®] (Maquet, Rastatt, Germany) with the same previous settings. The ECMO circuit consisted of 3/8 inch Bioline[®] tubing (Maquet Cardiopulmonary AG, Hirrlingen, Germany), a Rotaflow[®] centrifugal pump (Maquet Cardiopulmonary AG), and a Quadrox D[®] oxygenator (Maquet Cardiopulmonary AG).

The patient was cannulated with a 14-fr Bio-Medicus[®] arterial cannula (Medtronic, Minneapolis, MN) in the right carotid and a 14-fr Bio-Medicus[®] venous cannula (Medtronic, Minneapolis, MN) in the right internal jugular. Upon initiation of ECMO, the maximum flow achieved was 50 mL/kg/min despite increasing revolutions per minute (RPMs) with a high negative pressure on the venous drainage line (−32 mmHg). The ECMO circuit and venous cannula were checked for kinks or obstructions and none were noted. Various maneuvers were done in an attempt to improve venous drainage, 5 mL/kg of packed red blood cells (pRBCs) were given and the surgeon repositioned the venous cannula, neither of which improved venous drainage. Therefore, the venous cannula was removed and replaced with a 15-fr Bio-Medicus[®] arterial cannula (Medtronic, Minneapolis, MN) without any improvement of venous drainage.

The patient was placed back on the anesthesia ventilator, again with the previous settings, as it was felt the inadequate venous drainage was a direct result of the patient's severe bronchospasm, hyperinflation, and high intrathoracic pressure causing tamponade-like physiology. Once the inhaled anesthetic gas was delivered an ECMO flow of 85 mL/kg/min was achieved with a venous line pressure of −11mmHg. Over the next 2 hours ECMO flows improved to 100 mL/kg/min without further interventions. Chest x-ray and echocardiogram post cannulation showed appropriate cannula placement and no evidence of pneumothorax or other cause of impaired venous return. Throughout the process of assessment to placing the child on ECMO, arterial blood gases were obtained. The results from these are presented in Table 1.

Table 1. Arterial blood gases obtained during assessment through stabilization on ECMO with isoflurane delivery.

Event	pH	pCO ₂ (mmHg)	pO ₂ (mmHg)	Lactate (mEq/L)	Mode of Ventilation
Arrival at Hospital	6.8	150	76	4.7	Manual ventilation using handbag
Isoflurane Trial	7.17	101	475	5.5	Anesthesia ventilator
On ECMO	7.24	66	297	6.0	Ventilator
Patient on ECMO with isoflurane	7.41	37	268	1.6	Anesthesia ventilator

ECMO, extracorporeal membrane oxygenation.

Because of the period of PEA, the patient experienced she was cooled to a core body temperature of 33°C–34°C for 72 hours. The decision was made to keep the patient on 2% isoflurane throughout the ECMO support period. During that time she required no additional IV analgesia, sedation, or neuromuscular paralysis. The patient was slowly rewarmed on ECMO day 4 over a 12-hour period.

On ECMO day 11, the ventilator settings were an FiO₂ of 50%, rate of 18 breaths per minute, PEEP of 5 cm H₂O, and a tidal volume of 8 mL/kg, which resulted in a PIP of 32 cm H₂O. The patient's chest x-ray showed good aeration and the intensive care physician felt that her bronchospasm was under control and that the patient could be weaned from isoflurane and ECMO. The patient was transitioned to a Servo-I® (Maquet, Rastatt, Germany) with an FiO₂ of 60%, rate of 13 breaths per minute, PEEP of 7 cm H₂O, and a tidal volume of 7 mL/kg, which resulted in a PIP of 25 cm H₂O. ECMO circuit flow was slowly reduced from 100 mL/Kg/min to 30 mL/kg/min over 12 hours. The circuit was then clamped off to the patient while maintaining flow through an arterial to venous bridge within the ECMO circuit. After 10 minutes, a patient arterial blood gas was obtained, which had a pH of 7.24, pCO₂ of 67 mmHg, a pO₂ of 73 mmHg, and a lactate of .5 mEq/L. The patient was then separated from ECMO without issue and a post-ECMO blood gas had a pH of 7.32, pCO₂ of 63 mmHg, a pO₂ of 90 mmHg, and a lactate of .8 mEq/L. On day 13 the patient extubated and was placed on Vapotherm® 2000i (Vapotherm, Exeter, NH) of 10 L/min with an FiO₂ of 80%. The patient's neurologic status was closely monitored due to the unknown amount of time the patient was in PEA before receiving CPR. Pediatric neurology performed an electroencephalography (EEG) study on ECMO day 7 and no seizure activity was noted. At the time of discharge (PICU day 16), she was alert and appropriately responsive to verbal commands. She did have some right-sided weakness and therefore she was transferred to an outside facility for intensive rehabilitation services.

COMMENT

Impaired venous drainage is common on ECMO, but this case demonstrates an uncommon etiology to this common

problem. Although the patient required VA ECMO support, her condition was a result of severe hypercapnia, and cardiopulmonary failure caused by central venous obstruction and impaired venous return from severe asphyxial asthma. This venous obstruction was so significant that immediately after ECMO cannulation we were not able to obtain sufficient venous drainage to adequately support this patient on ECMO. Physiologically similar and slightly more common examples of this type of venous obstruction can be seen with tension pneumothorax and pericardial tamponade.

Air trapping causing significant venous obstruction has been reported in the literature. Adhiyaman et al. reviewed the delayed return of spontaneous circulation (ROSC) after CPR has been terminated. They report that one potential explanation for delayed ROSC was that rapid manual ventilation without adequate time for exhalation during CPR could result in significant air trapping, which could lead to diminished venous return and low cardiac output (8). The authors go on to state, the event would present similar to that of a pericardial tamponade in which circulation can only be restored if the obstruction is removed (8). Our

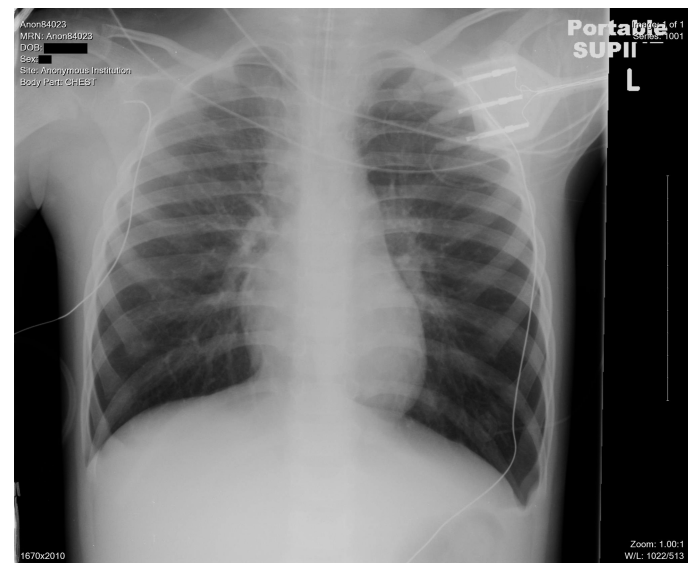


Figure 1. Chest x-ray before extracorporeal membrane oxygenation (ECMO) cannulation. Showing moderate hyperinflation of the lower lobes, with left greater than right. Mild right upper lobe atelectasis.

case seems to support that theory as our patient exhibited ROSC once manual chest decompression was performed. It is very likely that our patient's obstructive airway disease played a role in contributing to the dynamic hyperinflation of the lungs during mechanical ventilation. Even after manual chest decompression before ECMO, a chest x-ray showed moderate hyperinflation of the patient's lungs (Figure 1). Ten hours after ECMO initiation, another chest x-ray was taken and showed increased atelectasis, but the lungs were no longer hyperinflated (Figure 2). In this case, utilizing ECMO to support the patient allowed us to use lower ventilator setting with longer exhalation times, limit hyperinflation, and reduce the risk of ongoing barotrauma.

Inhalational anesthetic gases have been used to treat the severe bronchospasms that can occur with status asthmaticus (9,10). By exerting its bronchodilation effects, inhaled anesthetic gases may greatly benefit patients who are exhibiting the effects of air trapping due to significant obstructive airway disease. However, the use of inhaled anesthetic

gas is often associated with hypotension and myocardial depression in a dose-dependent fashion (10). Therefore, the combined therapies of VA ECMO and inhaled anesthetic gases may be necessary to adequately support these extremely critical patients, especially if cardiac function is of concern.

ECMO support in status asthmaticus allows clinicians to avoid ventilator-induced lung injury and support the patient until improvement of the severe bronchospasm occurs (3,4). ECMO is a highly successful and important support mechanism whose success is predicated on accurate diagnosis of disease processes and subsequent treatment while providing organ support to allow that treatment to be successful. Vigilance in obtaining accurate diagnoses must be taken both before going on ECMO, and while support is being provided through continual reevaluation. Diagnosing ECMO-related issues must be put in context with patient disease processes before a satisfactory resolution can be obtained.

REFERENCES

1. Higgins JC. The 'Crashing Asthmatic.' *Am Fam Physician*. 2003;67:997-1004.
2. Bohn D, Kissoon N. Acute asthma. *Pediatr Crit Care Med*. 2001;2:151-63.
3. Mikkelsen ME, Pugh ME, Hansen-Flaschen JH, Woo YJ, Sager JS. Emergency extracorporeal life support for asphyxic status asthmaticus. *Respir Care*. 2007;52:1525-9.
4. Ju MH, Park J-J, Jhang WK, Park SJ, Shin HJ. Extracorporeal membrane oxygenation support in a patient with status asthmaticus. *Korean J Thorac Cardiovasc Surg*. 2012;45:186-8.
5. Chan-Dominy A, Anders M, Horton S, et al. Extracorporeal membrane modality conversions. *Perfusion*. 2015;30:292-4.
6. Duke GJ. Cardiovascular effects of mechanical ventilation. *Crit Care Resusc*. 1999;1:388-99.
7. Duff JP, Joffe AR, Sevcik W, deCaen A. Autoresuscitation after pediatric cardiac arrest: Is hyperventilation a cause? *Pediatr Emerg Care*. 2011;27:208-9.
8. Adhiyama V, Adhiyaman S, Sundaram R. The Lazarus Phenomenon. *J R Soc Med*. 2007;100:552-557.
9. Char DS, Ibsen LM, Ramamoorthy C, Bratton L. Volatile anesthetic rescue in children with acute asthma: Innovative but costly or just costly. *Pediatr Crit Care Med*. 2013;14:343-50.
10. Shankar V, Churchwell KB, Deshpande JK. Isoflurane therapy for severe refractory status asthmaticus in children. *Intensive Care Med*. 2006;32:927-33.

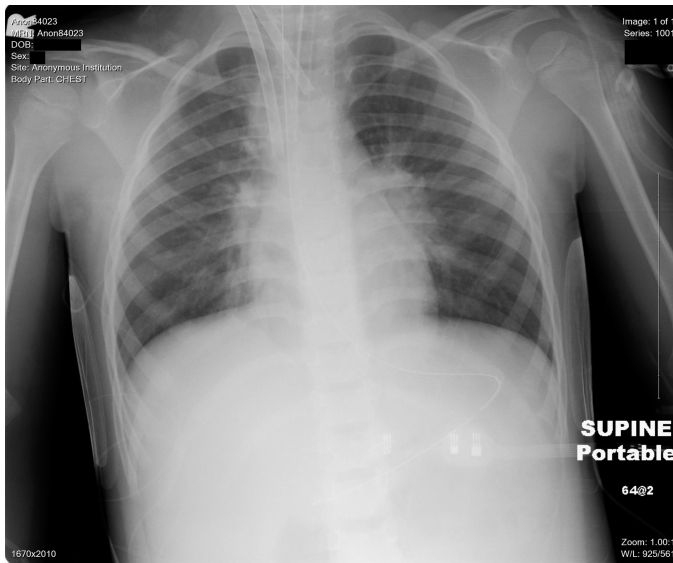


Figure 2. Chest x-ray 10 hours after extracorporeal membrane oxygenation (ECMO) initiation. Normal inflation of the lungs. Mild atelectasis in right upper and lower lobes.