ACS Case Reviews in Surgery
Vol. 1, No. 2

Use of Extracorporeal Membrane Oxygenation (ECMO) for Refractory Hypercarbic Respiratory Failure Due to a Severe Inhalation Burn Injury

AUTHORS:
Spanier MJ, MD[a], Jara CB[b], Carter DW[c], Groom RC[b], Rappold JF[c]

CORRESPONDENCE AUTHOR:
Matthew Spanier, MD
Department of Emergency Medicine
22 Bramhall Street
Portland, ME, 04102
(207) 662-7050
mspanier@mmc.org

AUTHOR AFFILIATIONS:
a. Maine Medical Center
Department of Emergency Medicine
Portland, ME, 04102

b. Maine Medical Center
Department of Cardiovascular Perfusion
Portland, ME, 04102

c. Maine Medical Center
Department of Surgery
Portland, ME, 04102

Background	While extracorporeal membrane oxygenation (ECMO) has been utilized for reversible cardiac dysfunction and hypoxemic respiratory failure, there is limited reported use for hypercarbic respiratory failure due to inhalational burn injuries.
Summary	We present a 24-year-old burn patient who presented with 37% total body surface area (TBSA) burns and severe (grade 4) inhalational injury resulting in profound hypercarbic respiratory failure. Her respiratory failure was unresponsive to standard therapies and mechanical ventilator manipulation. She was placed on veno-venous (VV) ECMO with rapid correction of hypercarbia and remained on the circuit through a tenuous hospital course that included a severe lactic acidosis due to burn sepsis and hemorrhagic shock requiring massive transfusion.
Conclusion	We believe this represents a unique application of VV ECMO in treating severe burn injury. The ECMO circuit rapidly corrected the patient's hypercarbia while allowing for lung protective ventilation, serving as a bridge until return of adequate pulmonary function. This case illustrates that severe inhalational injuries and associated respiratory failure are potentially reversible if gas exchange is supported by ECMO.
Keywords	ECMO, burn, inhalational injury, hypercarbic respiratory failure

Case Description

Extracorporeal membrane oxygenation (ECMO) has traditionally been used to support cardiac function and as a bridge for reversible hypoxemic respiratory failure. Limited reports exist applying the therapy to hypoxemic respiratory failure due to burn and smoke inhalation injury, the most comprehensive of which has revealed no survival benefit.[1] While ECMO has been reported in limited cases for CO_2 exchange in medical patients with respiratory failure, as a modality for respiratory support it has primarily been applied in the treatment of refractory hypoxemia in diseases such as acute respiratory distress syndrome.[2, 3] In this case report, we will summarize the use of veno-venous (VV) ECMO for respiratory support in a young patient with profound hypercarbic respiratory failure due to an inhalational injury as the indication for ECMO therapy. We believe this to be a unique application of ECMO, as the patient's hypoxemic respiratory failure was corrected with standard manipulation of ventilator settings. An additional point of interest in this case is the development of a severe metabolic acidosis and hemorrhage requiring a massive blood product transfusion while on the ECMO circuit.

To Cite: Spanier MJ, Jara CB, Carter DW, Groom RC, Rappold JF. Use of Extracorporeal Membrane Oxygenation (ECMO) for Refractory Hypercarbic Respiratory Failure Due to a Severe Inhalation Burn Injury. *ACS Case Reviews in Surgery.* 2017;1(2):12-15.

Spanier MJ, MD[a], Jara CB[b], Carter DW[c], Groom RC[b], Rappold JF[c]

The patient, a healthy 24-year-old female, was admitted to our surgical intensive care unit (ICU) after being extricated from a mobile home fire. She was initially stabilized at an outstate critical access emergency department where the patient underwent rapid sequence intubation and was fluid resuscitated. Upon arrival, we calculated her to have 37 percent TBSA burns (partial thickness burns to the face, anterior neck, ears, and full thickness burns circumferentially on the bilateral upper extremities and scattered across the back and shoulders). A right upper extremity escharotomy was performed. Bronchoscopy revealed copious carbonaceous deposits, mucosal sloughing, and necrosis consistent with severe (grade 4) inhalational injury (Figure 1).

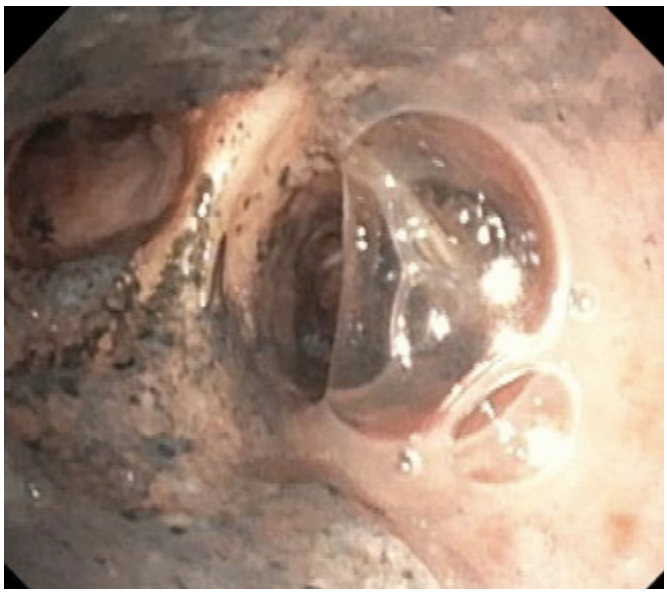

Figure 1. Repeat bronchoscopy the day following admission reveals carbonaceous deposits at the carina.

Fluid resuscitation was initiated per our institutional protocol using a modified Parkland formula. The patient was started on vasopressin due to intermittent hypotension on arrival.

As illustrated in Table 1, the patient presented with a primary respiratory and secondary metabolic acidosis.

The patient's respiratory acidosis remained refractory to ventilator manipulation, including assist control ventilation with a tidal volume (T_v) of 500 ml (8ml/kg adjusted for ideal body weight [IBW]) and a rate of 30 respirations/min as well as a trial of pressure control ventilation. We pursued several adjunct therapies: administration of the renally excreted buffer tris-hydroxymethyl aminomethane (THAM) in order to decrease the ventilation requirement to compensate for the patient's acidosis; nebulized heparin, albuterol, and n-acetylcysteine to facilitate mucolysis, bronchodilation, and to inhibit fibrin clot formation within the airways; and a single dose of vecuronium (after which we assessed clinically for improved ventilation without twitch monitoring). Of note, oxygenation remained stable on 85–100 percent FiO_2 and a positive end expiratory pressure (PEEP) of 10 cm H_2O.

Approaching 24 hours postinjury, the patient's ventilatory status worsened and the decision was made to pursue an aggressive alternative strategy with VV ECMO given the patient's young age and lack of other immediate life threatening injury. An inflow tract for the ECMO circuit was established with a 25 french multi-stage venous catheter inserted through the right femoral vein into the inferior vena cava at the levels of the diaphragm. A second venous catheter (17 French) was placed in the superior vena cava at the level of right atrium under ultrasound guidance via the right internal jugular vein to serve as an outflow tract. We then initiated VV ECMO with a Cardiohelp™ ECMO console and an HLS Bioline Advanced 5.0 circuit at an initial flow rate of 2.6 L/min (LPM) and a sweep gas of 6 LPM. The patient's respiratory acidosis resolved approximately one hour after initiation of ECMO therapy (Table 1). At this time, the patient was placed on lung protective ventilator settings (V_t 300 cc [5 cc/kg IBW], PEEP 10 cm H_2O, FiO_2 80%). Throughout the duration of ECMO therapy, sweep gas rates were adjusted to maintain a normal pCO2 (35–45 mmHg) as measured by arterial blood gas (ABG). The patient was anti-coagulated on a heparin infusion per institutional policy.

Day	1				2				3				4	
Time	3:40	10:00	14:55	20:50	4:55	12:30	16:15	22:15	3:15	11:21	17:00	20:35	11:20	17:04
ABG														
pH	6.91	6.99	7.24	7.31	7.19	7.16	7.15	7.11	7.18	7.35	7.43	7.48	7.34	7.37
pCO2	109	104	43	35	37	35	35	31	36	29	28	32	47	40
Bicarbonate	21	24	18	17	13	12	12	9	13	16	18	23	25	23
pO2	220	129	277	94	102	240	125	132	168	161	177	202	122	143
Base Excess	-17	-13	-13	-8	-15	-15	-16	-19	-14	-9	-5	1	-1	-2
Time					6:30	8:43	16:15	19:40	4:05	11:15	17:00	23:05	11:20	20:25
Lactate (mmol/L)					9	8.9	7.5	8	15.7	14.3	10.8	7.3	4.7	3

Table 1. Arterial blood gas (ABG) and lactate measurement by hospital day. VV-ECMO therapy was initiated at approximately 13:00 hours on hospital day one. The patient underwent excision of bilateral full thickness arm burn at approximately 13:00 hours on hospital day three.

The following 24 hours were notable for anuric acute renal failure with hyperkalemia requiring continuous renal replacement therapy and the development of a profound metabolic (lactic) acidosis. Multiple possible etiologies for her worsening acidosis were considered and treated: sodium thiosulfate for recurrent cyanide toxicity (hydroxocobalamin was administered on scene); an unremarkable exploratory bedside laparotomy; cefepime and vancomycin administration for sepsis; and a left upper extremity and hand escharotomy for increasing compartment pressure. The patient's metabolic acidosis continued to worsen despite our interventions, and by the end of her second hospital day, the patient required vasopressor support with norepinephrine, dobutamine, and vasopressin. Resuscitative fluid was changed to fresh frozen plasma (FFP) and sodium bicarbonate.

To facilitate increased demand for CO_2 removal, blood flow through the ECMO circuit was increased to 3.9 LPM and sweep gas increased to 10 LPM. She tolerated continued down-titration of FiO_2.

By hospital day three, the patient's profound lactic acidosis was thought to be a result of burn sepsis and bilateral upper extremity full-thickness burns were excised. During the following 24 hours, she was weaned from vasopressors and stabilized clinically. Interestingly, in the 24 hours prior to excision of the bilateral upper extremity burns, we noted a mixed venous oxygen saturation (SvO_2) of 79 percent (Hgb 7.2). Following burn excision there was a persistent drop in SvO_2 to 51 percent for approximately 24 hours, despite pRBC transfusion to a Hgb of 8.8. The patient had normal cardiac function during this time as assessed by transthoracic echocardiography.

Further hospital course was significant for excision and grafting of full thickness back burns on hospital day seven which was complicated by significant blood loss (EBL 1.5 L) and continued hemorrhage in the ICU. This required activation of our institution's massive transfusion protocol (MTP) and administration of 33 units of packed red blood cells, 32 units of FFP, and eight units of platelets. The patient's heparin infusion had been discontinued intraoperatively once she developed significant hemorrhage. During the MTP, the patient remained on the ECMO circuit and it was challenging to maintain adequate preload in the setting of hemorrhagic shock.

The following day (hospital day eight) the patient stabilized. She was removed from the ECMO circuit and placed on a standard mechanical ventilatory mode. The patient thereafter continued to clinically improve and was weaned from hemodialysis and mechanical ventilation. After several additional operations to excise and graft her burns, the patient was discharged from the hospital 66 days after admission.

Discussion

Recent case series and a meta-analysis have reported limited but encouraging results on the use of ECMO for inhalational burn injury associated with hypoxemic respiratory failure.[4,5] Both VV and veno-arterial ECMO have been successfully applied in adult and pediatric burn patients with acute respiratory failure.[5,6] However, even the landmark CESAR trial, which revealed a survival benefit from ECMO therapy in medical patients, enrolled relatively few participants with uncompensated hypercapnia.[3]

Our case is unique in that it presents the application of VV ECMO to correct hypercarbic respiratory failure after a severe inhalational injury. This represents a poorly studied indication for ECMO therapy in burn patients. The ECMO circuit facilitated placing the patient on a lung protective ventilation strategy that, along with aggressive pulmonary hygiene, resulted in a return of pulmonary function within seven days. Although we present evidence limited to a single case, further development and trials of ECMO for profound acute hypercarbic respiratory failure with or without hypoxia may show it to be a valuable bridge therapy in burn patients.

We believe this case also illustrates the phenomenon of cytopathic hypoxia[7] in the setting of burn sepsis. The patient experienced a worsening lactic acidosis and vasopressor requirement in the setting of a normal SvO2. Following burn excision, there was a substantial drop in the SvO2 despite correction of the patient's anemia, improving lactic acidosis, normal cardiac function, and decreasing vasopressor requirement. We hypothesize this may have been due to the return of mitochondrial function and normal aerobic metabolism.

Conclusion

Extracorporeal membrane oxygenation has evidence supporting its use in hypoxemic respiratory failure; however, most published experience has been in medical and burn patients with hypoxemia as the indication for therapy. This case illustrates that VV ECMO may be a valuable bridge therapy in the treatment of acute hypercarbic respiratory failure resulting from inhalational burn injuries.

Lessons Learned

Severe inhalational burn injuries are reversible if pulmonary function can be supported. VV ECMO may be a valuable therapy in the treatment of both hypoxemic and hypercarbic respiratory failure following inhalational burn injuries.

References

1. Asmussen S, Maybauer D, Fraser J, et al. Extracorpeal membrane oxygenation in burn and smoke inhalation injury. *Burns* 2013;39:429-435.
2. Gattinoni L, Pesenti A, Mascheroni D, et al. Low-frequency positive-pressure ventilation with extracorporeal CO2 removal in severe acute respiratory failure. *JAMA* 1986;256:881-886
3. Peek G, Mugford M, Tiruvoipati R, et al. Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. *Lancet* 2009;374:1351-1363.
4. Walker P, Buehner M, Wood L, et al. Diagnosis and management of inhalation injury: an updated review. *Crit Care* 2015;19:351
5. Hsu P, Tsai Y, Lin C, et al. Benefit of extracorporeal membrane oxygenation in major burns after stun grenade explosion: Experience from a single military medical center. *Burns* 2017;43:674-680
6. Askegard-Giesmann J, Besner G, Fabia R, et al. Extracorporeal membrane oxygenation as a lifesaving modality in the treatment of pediatric patients with burns and respiratory failure. *J Pediatr Surg* 2010;45:1330-1335
7. Loiacono LA, Shaprio DS. Detection of Hypoxia at the Cellular Level. *Crit Care Clin* 2010;26:409-421.