Extracorporeal Membrane Oxygenation as Acute Rescue Therapy for Negative Pressure Pulmonary Edema in the Post Anesthesia Care Unit: A Case Report

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Author Contribution:

Katrina Augustin: This author helped with case selection, data collection, primary author of manuscript, and obtaining informed consent from the patient.

Christina Creel-Bulos: This author helped with case selection and editing the manuscript.

Gaurav Budhrani: This author helped with case data collection.

Casey Miller: This author helped with case selection and data collection.

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Abstract

Negative pressure pulmonary edema (NPPE) may result in respiratory failure refractory to conventional management strategies. Venovenous extracorporeal membrane oxygenation (VV ECMO) can serve as a rescue therapy in cases of severe respiratory failure. Rapid initiation of VV ECMO can decrease morbidity and mortality while facilitating early liberation from mechanical ventilation and promoting early rehabilitation. We describe the successful utilization of VV ECMO as rescue therapy for severe NPPE-induced hypoxic respiratory failure and peri-arrest state in the post-anesthesia care unit (PACU) in a patient with post-extubation airway obstruction after undergoing patellar tendon repair.

Glossary of Terms:

Negative Pressure pulmonary edema: NPPE

Veno-venous extracorporeal membrane oxygenation: VV ECMO

Post-anesthesia care unit: PACU

Systemic anticoagulation: SA

Membrane pressure gradient: dP

Chest radiograph: CXR

Oxygen delivery: DO2

Introduction

Negative Pressure pulmonary edema (NPPE), also known as postobstructive pulmonary edema, is a rare life-threatening etiology of acute respiratory failure. Classically NPPE develops as a result of upper airway obstruction from post-extubation laryngospasm after surgery, upper airway infections, or tumors[1].

Conventional treatment of severe NPPE is focused on relieving the upper airway obstruction, positive pressure ventilation, and medication therapy including diuretics, with resolution of most cases. Case reports have described the need for delayed veno-venous extracorporeal membrane oxygenation (VV ECMO) in cases of refractory hypoxia not responding to initial therapies [2, 3, 4, 5]. However, the use of VV ECMO as acute rescue therapy in the post anesthesia care unit (PACU) for NPPE-induced severe hypoxic respiratory failure, shock, and peri-arrest physiology is yet to be described in the literature.

We describe the utilization of VV ECMO in the PACU as a rescue therapy for severe NPPE induced hypoxic respiratory failure and profound shock in a patient with post extubation airway obstruction after undergoing elective patellar tendon repair. Additionally, we discuss the challenges of systemic anticoagulation (SA) in the immediate postoperative period, early mechanical ventilation liberation facilitated by VV ECMO support, and the consideration of VV ECMO as part of the difficult airway management algorithm [6].

This manuscript adheres to the Consensus-based Clinical Case Reporting Guidelines [7]. Written Health Insurance Portability and Accountability Act authorization has been obtained for the publication of this case report

Case Description

A 29-year-old ASA II male with history of post-traumatic stress disorder (PTSD) underwent elective open patellar tendon repair under general anesthesia at an outside hospital. The case was uneventful and the patient was extubated prior to transfer to the PACU. On arrival to the PACU, he was noted to be apneic with concern for airway obstruction. Bag mask ventilation and narcan was administered. Despite these interventions the patient developed worsening respiratory distress, profound hypoxemia (SpO2 <85%), and copious pulmonary edema. Reintubation required several attempts due to regurgitation of profuse pulmonary edema. The patient was given 120 mg of furosemide for diuresis. Multiple modes of ventilation, including inverse ratio ventilation, were attempted without success and hypoxemia persisted. Ventilator settings were maximized on a volume targeted pressure-controlled mode with PEEP of 24 cmH2O and 100% FIO2 with a prolonged inspiratory time.

Despite maximal medical optimization he had severe refractory hypoxia with subsequent circulatory shock. The patient required high dose vasopressor support including norepinephrine, epinephrine, and vasopressin infusions, along with bolus dosing of vasopressors to maintain adequate blood pressure. Given this, the ECMO team was consulted for emergent VV ECMO cannulation. The patient was deemed a suitable candidate for ECMO cannulation. Given the hemodynamic instability the decision was made to initiate ECMO therapy in the PACU. A right femoral 25 french multistage venous access cannula and a 22 french right internal jugular venous return cannula were placed with subsequent ECMO initiation via a Cardiohelp system. ECMO flow was set to 5 liters at 3460 RPMS and a sweep gas of 8 liters. Prior to leaving the PACU, the HLS circuit was noted to have a rapidly rising membrane pressure gradient (dP), from 20 mmHg to 80 mmHg, necessitating a circuit exchange due to concern for oxygenator thrombosis. The patient received 5000 units of heparin at the time of cannulation and an additional 5000 units followed by a heparin infusion at the time of oxygenator exchange. Subsequent dP maintained within acceptable range of 20-30 mmHg.

The patient was then transferred to the intensive care unit at our institution (Table 1), where he was transitioned to pressure control ventilation with PEEP of 15 cmH2O and inspiratory pressures of 16 cmH2O. Despite these settings, tidal volumes of only 20 ml to 70 ml could be achieved due to the poor pulmonary compliance. Initial chest radiograph (CXR) demonstrated complete opacification of bilateral lung fields (Figure 1). Within 24 hours of cannulation the patient demonstrated a rapid improvement in his oxygenation (Table 2). Vasopressor requirements dramatically declined and he was weaned off all vasopressor support. Transthoracic echocardiogram revealed hyperdynamic left ventricular function, ejection fraction >70%, and normal right ventricular function. The acute kidney injury present on admission rapidly resolved, and he required diuresis for the first 48 hours. Forty-eight hours post cannulation, he was able to be extubated with ongoing support via VV ECMO despite his CXR demonstrating persistent extensive bilateral airspace opacities (Figure 2). Anticoagulation for the ECMO circuit was maintained with heparin infusion targeting aPTT goal of 40 seconds. A small amount of bleeding from the cannulation site occurred but resolved with suturing. No other hemorrhagic complications were experienced including at the operative site.

On day 5, he tolerated a sweep trial and was subsequently liberated from ECMO. He briefly required oxygen via nasal cannula but was weaned to room air within the next 48 hours with drastic improvements in his CXR (Figure 3). He was transferred to the floor on day 7. On day 8, he was discharged home-neurologically intact without a supplemental oxygen requirement (Table 1).

Discussion

This case illustrates the feasibility of early initiation of ECMO support in the perioperative period. In this case, ECMO therapy was initiated while the patient was in extremis with profound hemodynamic instability and shock secondary to severe hypoxemia. In the presence of severe hypoxemia, an inability to match the high myocardial oxygen demand leads to hypotension and arrest if the cycle is not interrupted. Rapid

institution of VV ECMO in the PACU allowed our team to interrupt this vicious cycle preventing cardiac arrest and anoxic brain injury.

When initiating VV ECMO in the immediate postoperative period, the need for systemic anticoagulation (SA) poses a myriad of challenges. SA is generally required during ECMO therapies as blood exposed to foreign materials triggers the inflammatory response/coagulation cascade leading to prothrombotic state [9, 10]The risks of thromboembolic events such as circuit thrombosis, oxygenator failure, and venous thromboembolism must be weighed against the potential risks of major bleeding with SA in the postoperative period. With heparin bonded extracorporeal tubing and newer generation centrifugal pumps there has been increased interest in minimizing SA in these patients and literature supports the feasibility of an anticoagulation free approach with no increased risk of thrombosis [11, 12]. Unfortunately, our patient had a rapidly rising dP immediately post cannulation, raising concern for oxygenator thrombosis necessitating a circuit exchange. Given the concerns for a prothrombotic state, SA with heparin infusion was initiated and maintained until decannulation. The patient had no further significant oxygenator thrombosis with stable dPs and no post decannulation venous thromboembolism. Additionally, the patient had no hemorrhagic complications despite his recent orthopedic surgery.

Severe NPPE can significantly impair lung compliance making maintenance of lung protective ventilation while promoting adequate gas exchange a considerable challenge.

Utilization of VV ECMO not only allows for lung protective ventilation, but also facilitates early extubation with gas exchange supported by ECMO [13]. Additionally, early liberation from mechanical ventilation and associated sedation can minimize risk of delirium, critical illness myopathy/polyneuropathy, diaphragmatic dysfunction, and ventilator associated pneumonia [14]. Early extubation also facilitates patient interaction which is often an under-appreciated benefit that can not only minimize patient anxiety and discomfort but also facilitate better care as patients can relay their symptoms to better inform care [14]. This case demonstrated the ability to successfully liberate our patient from the ventilator within 36 hours of the initial insult while supporting him on VV ECMO till he had recovered gas exchange. Early extubation facilitated removal of sedation, minimized deconditioning in the setting of his patellar tendon repair, and allowed for enhanced patient interaction which was especially important to this patient in the setting of his history of PTSD.

The profuse pulmonary edema in this case created a difficult airway for reintubation. The difficult airway algorithm historically relied on an invasive emergency cricothyrotomy as a last resort for a difficult airway if intubation or other airway adjuvants failed[6]. In the 2022 update of the American Society of Anesthesiologists Practice Guidelines for Management of the Difficult Airway, ECMO is now considered a vital part of the algorithm for management of the difficult airway. The guidelines reinforce the importance of minimizing time where a patient cannot be ventilated or oxygenated [6]. Research has shown that anoxic brain damage can occur within minutes without oxygen and death shortly after, making early intervention imperative [15]. Rapid institution of VV ECMO, as in this case, can be lifesaving and is an important resource that should be part of every difficult airway algorithm for a patient that cannot be ventilated or oxygenated.

In cases of severe NPPE with refractory hypoxia, despite conventional treatment with positive pressure ventilation, rapid initiation of VV ECMO in the postoperative is not only feasible but can also decrease morbidity and mortality. VV ECMO facilitates lung protective ventilation as well as early liberation from mechanical ventilation thus minimizing risk of delirium, critical illness myopathy/polyneuropathy, diaphragmatic dysfunction, and ventilator associated pneumonia while promoting early rehabilitation. Early initiation of ECMO therapy should be incorporated into any clinical algorithm when presented with refractory respiratory failure.

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Tables

Table 1 Title: Timeline of Relevant Clinical Events

Day	Events
1	V-V ECMO cannulation (late afternoon) ECMO Circuit Exchange (approximately 30 minutes post cannulation) Transfer to our hospital /Admit to ICU
2	CXR with complete opacification of bilateral lung fields TTE with hyperdynamic EF >70% Transitioned to Bilevel ventilation
3	Liberated from mechanical ventilation
6	Decannulation from V-V ECMO
7	Transfer to floor
8	Discharge to home

Table 1 Caption: Outline of key clinical events during the course of ICU admission and overall 8-day hospitalization.

Table 2 Title: Arterial Blood Gases Throughout Hospitalization

Time	Fraction of Inspired oxygen	ECMO flow (L)/ Sweep gas (L) (100% FIO2 via sweep gas)	pH/pCO2/Pa02
Prior to cannulation	100%	0/0	(SpO2 remained <85%, ABG not available)
Immediate Post cannulation	100%	4/4.5	7.22/64/154

<8 hrs post cannulation	70%	5/8	7.3/51/55
Day 3: post extubation	80%	5.25/3	7.44/44/188
Day 6: post decannulation	80%	0/0	7.49/39/141

Table 2 Caption: Arterial blood gas values throughout hospitalization.

Figure Titles and Legends

Figure 1 Title: Portable chest radiograph from hospital day 1.

Figure 1 Legend: This radiograph demonstrates complete opacification of bilateral lung fields with appropriately positioned ECMO cannulas 8.3 cm apart.

Figure 2 Title: Portable chest radiograph hospital day 3 (less than 48 hours after cannulation)

Figure 2 Legend: This radiograph demonstrates persistent extensive bilateral interstitial/airspace opacities with subsequent removal of endotracheal tube.

Figure 3 Title: Portable chest radiograph from hospital day 7 (day before discharge)

Figure 3 Legend: This radiograph demonstrates interval improvement of pulmonary edema.