COVID-19 Patient Bridged to Recovery with Veno-Venous Extracorporeal Membrane Oxygenation

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Abstract

Background: In severe cases, the COVID-19 viral pathogen produces hypoxic respiratory failure unable to be adequately supported by mechanical ventilation. The role of extracorporeal membrane oxygenation (ECMO) remains unknown, with the few publications to date lacking detailed patient information or management algorithms all while reporting excessive mortality. Methods: Case report from a prospectively maintained institutional ECMO database for COVID-19. Results: We describe veno-venous (VV) ECMO in a COVID-19 positive woman with hypoxic respiratory dysfunction failing mechanical ventilation support while prone and receiving inhaled pulmonary vasodilator therapy. After nine days of complex management secondary to her hyperdynamic circulation, ECMO support was successfully weaned to supine mechanical ventilation and the patient was ultimately discharged from the hospital. Conclusions: With proper patient selection and careful attention to hemodynamic management, ECMO remains a reasonable treatment option for COVID-19 patients.

Introduction

Beginning December 2019, the COVID-19 pandemic has spread globally now with over three million cases worldwide[1]. While many patients are adequately supported by mechanical ventilation, there exists no consensus for ECMO, with some concern it may worsen the illness[2]. Despite anecdotal evidence, data is limited and patient details remain sparse[3]. We describe our experience recovering a COVID positive patient with VV ECMO and the unique difficulties of the clinical course.

Methods

This is a case report of one patient with data extracted from a prospective institutional database focused on ECMO for COVID-19 patients. IRB approval was obtained from the Brigham and Women's hospital and individual consent was not deemed necessary.

Results

Our patient is a 49 year-old woman with obesity (BMI 39) and hypertension who developed cough, sore throat, and fever progressing to severe dyspnea. She presented to the emergency room with a resting oxygen saturation of 75% (room air) improving to 88% via non-rebreather. Chest radiograph revealed bilateral infiltrates, attempts to obtain an arterial blood gas (ABG) were unsuccessful due to clotted samples.

Her dyspnea worsened prompting intubation and mechanical ventilation support, with a tidal volume of 6 ml/kg (ideal body weight), PEEP 18cmH_2O, and FiO_2 100% yielding an arterial partial pressure of oxygen (PAO2) 134mmHg with plateau pressure 30cmH_2O. Echocardiogram revealed normal cardiac function, renal and liver function were without abnormality, and intravenous heparin was started (PTT 60-80) for a D-dimer greater than 4000ng/ml and fibrinogen above assay. She was paralyzed, treated with inhaled nitric oxide, and underwent prone positioning.

Due to persistent hypoxia she was ultimately initiated on VV ECMO. Ultrasound was used to access the right femoral vein (RFV) and right internal jugular vein (RIJ). A 10,000 unit bolus of intravenous heparin was administered followed by insertion of a 25Fr multistage cannula in the RFV and a 17Fr return cannula in the RIJ. Flows ranging from 4.5-5.0 liters/minute were achieved at pump RPMs of 3700. Despite excellent circuit oxygenation (confirmed with post-membrane oxygenator ABG) and ECMO optimization, the patient required an FiO2 of 70% to maintain a PAO2 > 60mmHg.

She remained febrile and tachycardic with an estimated cardiac output(CO) of 9.8L/min (via Fick equation). We hypothesized that her elevated CO was not required to maintain adequate oxygen delivery (DO2), as her estimated basal output was 5.5-6L, but instead provoked by the infection. In an effort to increase the fraction of her CO entrained into the circuit we initiated an esmolol infusion (50mcg/kg/min) titrated to a pulse of 60-70 bpm. Phenylephrine (50 mcg/min) and vasopressin (0.04 units/min) infusions were started to maintain a mean arterial pressure >65mmHg. These interventions enabled decreasing the FiO_2 on the ventilator to 50%, PEEP 16cmH2O (per lung protective ventilation protocol) and achieved a PAO2>80mmHg with low tidal volumes.

After nine days on ECMO, compliance measured on the ventilator showed mild improvement (12-20ml/cmH_20) and a trial off ECMO maintained PAO2>100 on 60%FIO2 and PEEP 16cmH2O. In the setting of persistent bleeding at her cannulation sites and ability to be maintained on non-injurious ventilator support we decannulated. The patient improved on supine ventilation and was ultimately extubated and discharged from the hospital.

Conclusions

Here we describe our approach to a COVID-19 patient who failed medical management and ultimately required VV ECMO. Currently there are no specific guidelines available, therefore we have formulated an algorithm for early identification of COVID-19 patients requiring ECMO and devised specific management strategies to navigate their course (Figure1). This case had several challenging aspects including hyperdynamic cardiac function and coagulopathy.

COVID-19 has been associated with a hyperinflammatory state secondary to cytokine storm, manifested by elevated inflammatory markers, vasodilatory shock, and increased CO. This high output state can be difficult to manage on ECMO due to inadequate entrainment of CO into the circuit. Previous studies reported that extracorporeal capture of at least 60% of the native CO is essential for a saturation of 90% or Pa02 of 60mmHg[4]. Our patient responded well to the combination of short acting beta blockers and vasoconstrictors, however careful hemodynamic monitoring must be maintained due to concern for cardiac dysfunction from sepsis or COVID-19 related cardiomyopathy[5]. A plan to convert to a veno-arterial configuration should be considered on a case by case basis, and invasive hemodynamic monitoring and frequent bedside echocardiography are useful adjuncts. Approaches to the management of persistent hypoxia while on ECMO support are detailed in Figure1.

We anticipate that weaning of VV ECMO support in the COVID-19 cohort will be challenging given the variable evolution of lung disease we have observed in our non-ECMO cases manifesting with severe hypoxic failure. Due to risks of aersolization, we deferred tracheostomy which varies from our usual practice of early tracheostomy and reduction in sedation. Given these changes, COVID-19 patients are at risk for deconditioning and ventilator-associated pneumonia which may further complicate the ability to wean ECMO.

Given reports of thrombosis in COVID-19 patients[6] we began a heparin infusion on return of abnormal laboratory values in addition to a larger bolus of heparin before cannulation to avoid these complications. We experienced no issues with clot formation in the cannula or circuit but did experience persistent bleeding at the cannulation sites prompting a trial off ECMO. While thrombosis remains a risk, bleeding complications are significant, therefore we advise careful monitoring of coagulation studies and ECMO circuitry in this cohort.

To date outcomes using ECMO with COVID-19 remain poor with few details on the specifics of patient

characteristics, acceptance criteria and management. Henry et al published the first pooled series of patients yielding a combined ECMO mortality of 94%[3], hypothesizing the immunologic consequences of ECMO lead to worse outcomes[2]. Li et al published a series of COVID-19 ECMO patients (n=8) with 50% mortality[7].

In summary we were able to recover one COVID-19 patient with VV ECMO. With careful patient selection, mechanical support is a reasonable treatment strategy.

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References

1. https://covid19.who.int/
2. Henry BM. COVID-19, ECMO, and lymphopenia: a word of caution. Lancet Respir Med. 2020;8(4):e24.
3. Henry BM, Lippi G. Poor survival with extracorporeal membrane oxygenation in acute respiratory distress syndrome (ARDS) due to coronavirus disease 2019 (COVID-19): Pooled analysis of early reports. J Crit Care. 2020;58:27-28.
4. Schmidt M, Tachon G, Devilliers C, et al. Blood oxygenation and decarboxylation determinants during venovenous ECMO for respiratory failure in adults. Intensive Care Med. 2013;39(5):838-46.
5. Bonow RO, Fonarow GC, O'gara PT, Yancy CW. Association of Coronavirus Disease 2019 (COVID-19) With Myocardial Injury and Mortality. JAMA Cardiol. 2020
6. Klok FA, Kruip MJHA, Van der meer NJM, et al. Incidence of thrombotic complications in critically ill ICU patients with COVID-19. Thromb Res. 2020
7. Li X, Guo Z, Li B, et al. Extracorporeal Membrane Oxygenation for Coronavirus Disease 2019 in Shanghai, China. ASAIO J. 2020

Figure1 : Patient selection, evaluation, and treatment strategies. ARDS= acute respiratory distress syndrome, CKD=chronic kidney disease, CHF=congestive heart failure, PEEP=positive end expiratory pressure, ECMO=extracorporeal membrane oxygenation, PAO2=partial pressure of oxygen , PCO2=partial pressure of carbon dioxide, DO2 = oxygen delivery. VO2 = oxygen consumption. V/Q = ventilation/perfusion.