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CASE REPORT

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COVID-19 patient bridged to recovery with veno-venous extracorporeal membrane oxygenation

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Abstract

Background: In severe cases, the coronavirus disease 2019 (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 9 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 patients with COVID-19.

KEYWORDS
cardiovascular pathology, perfusion

1 | INTRODUCTION

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

2 | METHODS

This is a case report of one patient with data extracted from a prospective institutional database focused on ECMO for patients with COVID-19. Institutional board review approval was obtained from the Brigham and Women's Hospital and individual consent was deemed unnecessary.

3 | RESULTS

Our patient is a 49-year-old woman with obesity (body mass index: 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 ventilator support, with a tidal volume of 6 mL/kg, positive end-expiratory pressure (PEEP) 18 cm H_2O, and fraction of inspired oxygen (FiO_2) 100% yielding an arterial partial pressure of oxygen (PAO_2) 134mm Hg with plateau pressure of 30 cm H_2O. Echocardiogram revealed normal cardiac function, renal and liver function were without abnormality, and intravenous heparin was started for a D-dimer greater than 4000 ng/mL and fibrinogen above assay. Adjunctive treatments included neuromuscular blockade, inhaled nitric oxide, and 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 25-Fr multistage cannula in the RFV and a 17-Fr return cannula in the RIJ. Flows ranging from 4.5 to 5.0 L/min were achieved at 3700 pump rpm. Despite excellent circuit oxygenation (confirmed with postmembrane oxygenator ABG) and ECMO optimization, the patient required an FiO_2 of 70% to maintain a PAO_2 > 60 mm Hg.

She remained febrile and tachycardic with an estimated cardiac output (CO) of 9.8 L/min (via Fick equation). We hypothesized that her elevated CO was not required to maintain adequate oxygen delivery (DO_2), as her estimated basal output was 5.5 to 6 L, but instead provoked by infection. To increase the fraction of her CO entrained into the circuit, we initiated an esmolol infusion (50 mcg kg^{-1} min^{-1}) titrated to a pulse of 60 to 70. Phenylephrine (50 mcg/min) and vasopressin (0.04 units/min) infusions were started to maintain a mean arterial pressure of more than 65 mm Hg. These interventions enabled decreasing the FiO_2 on the ventilator to 50%, PEEP 16 cm H_2O (lung protective protocol), and achieved a PAO_2 > 80mm Hg with low tidal volumes.

After 9 days on ECMO, compliance measured on the ventilator showed improvement (12-20 mL/cm H_2O) and a trial off ECMO maintained PAO_2 > 100 on 60% FiO_2 and PEEP 16 cm H_2O. In the setting of bleeding at her cannulation sites and ability to be maintained on noninjurious ventilator support we decannulated. The patient improved on supine ventilation, was extubated, and discharged from the hospital.

4 | CONCLUSIONS

Here we describe our approach to a patient with COVID-19 who failed medical management and required VV ECMO. Currently, there are no guidelines available, therefore we have formulated an algorithm for early identification of patients with COVID-19 requiring ECMO and devised management strategies to navigate their course (Figure 1). We have included information on patient selection and contraindications including advanced age, malignancy, and profound

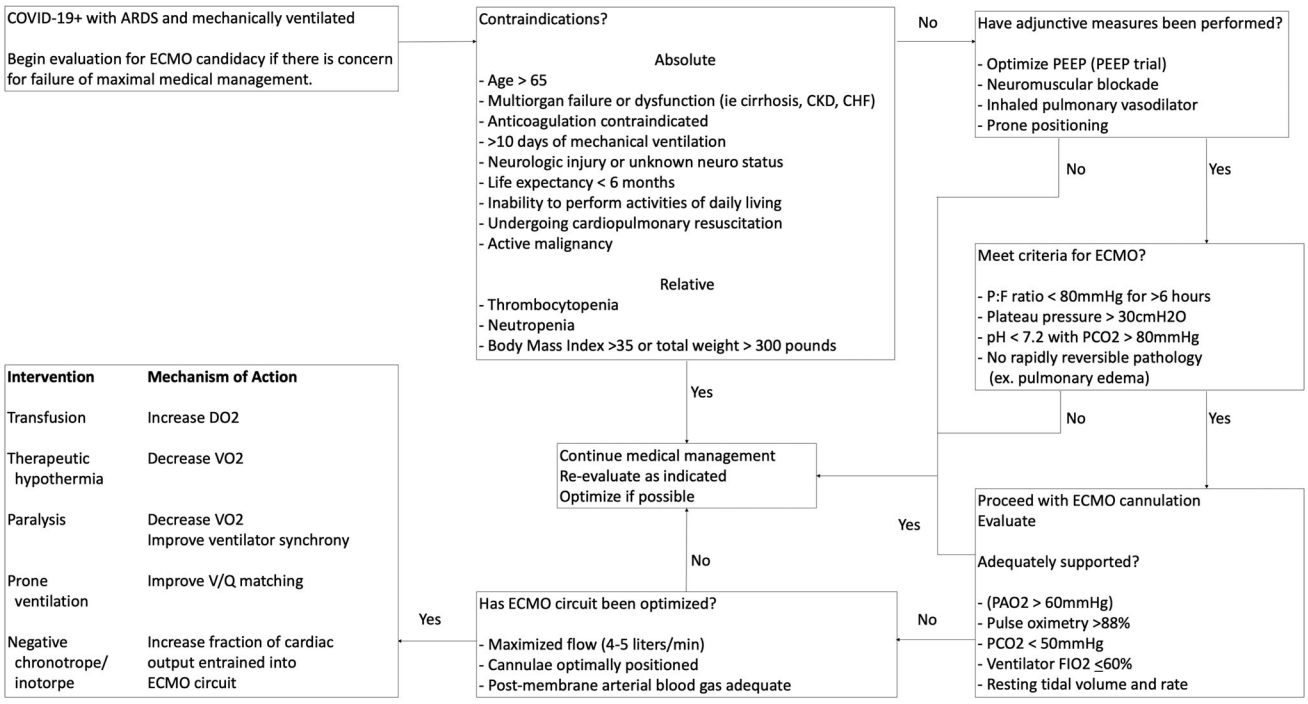

FIGURE 1 Patient selection, evaluation, and treatment strategies. ARDS, acute respiratory distress syndrome; CHF, congestive heart failure; CKD, chronic kidney disease; DO_2, oxygen delivery; ECMO, extracorporeal membrane oxygenation; PAO_2, partial pressure of oxygen; PCO_2, partial pressure of carbon dioxide; PEEP, positive end-expiratory pressure; VO_2, oxygen consumption; V/Q, ventilation/perfusion

neurologic injury. 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 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 PAO_2 of 60 mm Hg.[4] Our patient responded well to the combination of short acting β-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 venoarterial configuration should be considered on a case-by-case basis, and invasive hemodynamic monitoring and frequent bedside echocardiography are useful. Approaches to the management of persistent hypoxia while on ECMO support are detailed in Figure 1.

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 aerosolization, we deferred tracheostomy which varies from our usual practice of early tracheostomy and reduction in sedation. Given these changes, patients with COVID-19 are at risk for deconditioning and ventilator-associated pneumonia, which may further complicate the ability to wean ECMO.

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

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.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

AUTHOR CONTRIBUTION

All authors contributed to the concept, design, drafting, revision and approval of the article.

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