A 38-year-old man diagnosed with COVID-19 was hospitalized for 20 days and received treatment with levofloxacin, lopinavir/ritonavir, tocilizumab, and steroids; however, he did not show clinical signs of improvement and was transferred to our medical center.

At the emergency department, he presented with respiratory failure. On examination, his temperature was 98.6°F (37.0°C); blood pressure, 110/60 mm Hg; heart rate, 115 beats per minute; respiratory rate, 35 breaths per minute; and oxygen saturation, 75% while wearing a nonrebreather mask at 10 L/minute. Physical examination showed bilateral coarse crackles in the lower lungs field. The remainder of the physical examination was normal. Endotracheal intubation was performed successfully.

Laboratory test showed leukocytosis, 14,700/µL; pH, 6.96; pCO2, 198.3 mm Hg; pO2, 61 mm Hg; Sat O2, 70.3%; HCO3, 28.6 mmol/L; base excess, –2.5 mmol/L; lactate, 3.52 mmol/L; D-dimer level, 183 ng/mL; N-terminal pro B-type natriuretic peptide, 941 pg/mL; and hs-troponin I, 43.8 ng/mL.

The electrocardiogram showed sinus tachycardia with right axis deviation. Computed tomography of the chest revealed multiple bilateral ground glass opacities associated with consolidations with superimposed irregular septa (Figure 1).

The patient presented with refractory severe hypoxemia and hypercapnia after 12 hours of mechanical ventilation; therefore, it was decided to initiate treatment with veno-venous (VV) extracorporeal membrane oxygenation (ECMO).

During placement, transesophageal echocardiogram showed right cavitary dilatation and right ventricle dysfunction with akinesia of the right ventricle mid free wall. Right ventricle fractional area change (FAC) was measured at 25%, tricuspid annular plane systolic excursion (TAPSE) at 10 mm, right ventricle (RV) outflow tract velocity-time integral (RVOT VTI) at 4.9 cm (Figure 2, Video 1, Table 1), without tricuspid regurgitation. The diagnosis of RV failure was made, and the configuration was switched from VV-ECMO to venoarteriovenous (VAV) ECMO (Figure 3).

In the following 3 days, the patient showed progressive decrease in hs-troponin I and N-terminal pro B-type natriuretic peptide. A second transesophageal echocardiogram was performed showing normal size of right cavities and normal RV function. The FAC was now measured at 52%; TAPSE at 22 mm; RVOT VTI at 23 cm (Figure 4, Video 3, Table 1), without tricuspid regurgitation; right atrial volume index at 28 mL/m², and left ventricular ejection fraction at 71%, which was normal. Estimated pulmonary artery systolic pressure decreased from 50 to 30 mm Hg, and the tricuspid regurgitation disappeared without observation of any intrinsic abnormalities in the tricuspid valvular apparatus and secondary to pressure overload. Consequently, the medical team decided to change to VV-ECMO configuration 72 hours after the start of treatment.

Tracheostomy was performed on day 14, replacement of the oxygenator was performed on day 21, and ECMO weaning was started on day 30, once the lungs showed signs of improving compliance and better gas exchange. The VV-ECMO was withdrawn on day 75. Mechanical ventilation was weaned 6 days after ECMO removal, and he was discharged from the hospital 10 days later.

DISCUSSION

For patients who require ECMO assistance, configurations are classified according to the vessels by which the blood is withdrawn from or returned to the body. Veno-arterial (VA)-ECMO draws blood from a central vein and reinfuses it back into a major artery. It is used primarily in patients with right- or left-sided heart failure as a bridge to
Figure 1  Computed tomography shows multiple bilateral and ground glass opacities associated with consolidations with superimposed irregular septa.

Figure 2  Transesophageal echocardiogram shows (A) right ventricle dilatation and FAC of 25%, (B) RVOT VTI of 4.9 cm, and (C) TAPSE of 10 mm.
ARDS. VV-ECMO is usually employed in severe respiratory failure, with COVID-19-associated acute respiratory distress syndrome (ARDS). COVID-19 recommends the use of VV-ECMO in eligible patients [1].

ECMO provides hemodynamic and respiratory support at the same time. V AV-ECMO draws blood from a central vein and reinfuses it back into a different central vein in addition to a major artery. V AV-ECMO allows a combination of support from V A- and VV-ECMO. VV-ECMO helps in recovery, other ventricular assist devices, or heart transplantation. VV-ECMO draws blood from a central vein and infuses it back into another central venous location. It is employed in severe respiratory failure as a bridge to recovery and to lung transplantation. VAV-ECMO allows a combination of support from VA- and VV-ECMO. VV-ECMO draws blood from a central vein and reinfuses it back into a different central vein in addition to a major artery. VAV-ECMO provides hemodynamic and respiratory support at the same time.

The World Health Organisation guidelines for management of COVID-19 recommend the use of VV-ECMO in eligible patients with COVID-19-associated acute respiratory distress syndrome (ARDS). VV-ECMO is usually employed in severe respiratory failure, demonstrating satisfactory results in bridging to recovery and/or to lung transplantation. However, in patients with COVID-19, the association of ARDS and RV failure is highly prevalent; hence in this scenario, it is feasible to switch strategy to VAV-ECMO. Stöhr et al. suggest that VAV-ECMO confers greater benefit in comparison with VV-ECMO in patients with ARDS.

The incidence of RV dysfunction in ARDS is 10%-25% and is associated with greater mortality; thus assessing rapid and efficient recovery of RV function could be of paramount importance in this clinical context.

In our clinical case, we decided to switch from VV-ECMO to VAV-ECMO due to the presence of refractory hypoxemia and new-onset RV failure. The etiology of RV dysfunction is a common finding in patients with COVID-19 [5,7]. While no single causative factor has been elucidated, there are several potential etiologic factors that have been shown to increase RV afterload and impair RV contractility. In this case, RV dysfunction probably was multifactorial (thrombosis, vascular alterations, hypoxia, hypercapnia, effects of mechanical ventilation, direct myocardial injury).

During placement of VV-ECMO, the diagnosis of RV dysfunction was based on the presence of RV enlargement and contractility impairment; therefore, it was decided to switch to VAV-ECMO. In our clinical case, improvement in RV function demonstrated by transesophageal echocardiogram was clearly evident. After cannulation, the patient presented hemodynamic improvement, RV structural improvements, and increase in partial pressure of oxygen (Table 2).

With VAV-ECMO, arterialized blood flow is divided using a Y connector with one cannula returning blood toward the central aorta and one returning blood to the right atrium. The flow in both cannulas depends on cannula diameters and is balanced by using an adjustable clamp and monitored by a flow sensor. The demand for arterialized blood flow on each return cannula varies from patient to patient and over time. In this case, the divided return flow was controlled by partially clamping the return cannula. The arterial return flow was maintained at about 30%-40% of the cardiac output, and the venous return flow was maintained at about 60%-70% of the cardiac output.

During VAV-ECMO support, repetitive echocardiography and continuous oxygenation surveillance were mandatory to assess tissue oxygenation and RV filling and function. The VAV modality may allow for the preservation of RV preload while concomitantly improving overall oxygenation and eliminating differential hypoxemia. This phenomenon has been described in patients with severe respiratory failure connected to VA-ECMO and consists of hypoxemia in the upper body with normal oxygen saturation in the lower body (the patient’s head appears blue, whereas the lower extremities appear pink). A typical VA-ECMO flow rate can result in desaturated blood from the left ventricle perfusing the aortic arch and fully saturated infusion blood perfusing the lower body, secondary to high afterload (physiological obstruction) with normal or recovered left ventricular systolic function.

According to the Extracorporeal Life Support Organization registry, the modality of support in COVID-19 has been VV-ECMO (94%), VA-ECMO (4%), and VAV-ECMO (0.9%), which correlates with the presence of ARDS in COVID-19.

**CONCLUSION**

Early detection and treatment of RV dysfunction may lead to a decrease in mortality and improve patient outcomes in COVID-19. VAV-ECMO appears to be a viable mode of treatment for patients with COVID-19 with ARDS and RV failure. Echocardiography plays an important role in early diagnosis and treatment in these patients.
In response to the COVID-19 global health crisis, the American Society of Echocardiography Foundation, with the help of our generous donors, raised more than $5,000 for COVID-19-related aid in May 2020. The American Society of Echocardiography Foundation is proud to cover the processing fee for this report from these funds.

**SPECIAL NOTE**

In response to the COVID-19 global health crisis, the American Society of Echocardiography Foundation, with the help of our generous donors, raised more than $5,000 for COVID-19-related aid in May 2020. The

**SUPPLEMENTARY DATA**

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2021.02.004.

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