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SHORT COMMUNICATION

Veno-veno-arterial ECMO support for acute myocarditis combined with ARDS: a case report

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ABSTRACT

Background: In patients who developed a combined situation of severe acute respiratory distress syndrome with refractory hypoxemia and acute cardiac failure with circulatory collapse, traditional veno-venous or veno-arterial extracorporeal membrane oxygenation approach alone may not be sufficient enough to maintain both an acceptable range of gas exchange and a hemodynamic stability.
Case report: A 27-year-old male patient was suffering from severe acute respiratory distress syndrome caused by community-acquired pneumonia and acute myocarditis with circulatory shock. After mechnical ventilation for respiratory support, he was in a persistently refractory shock state. Veno-veno-arterial mode of extracorporeal membrane oxygenation was thus applied to provide both respiratory and circulatory support simultaneously, with good success.
Discussion: Modifying to a veno-veno-arterial mode can be another alternative strategy in a combined situation of refractory respiratory and cardiac failure, thus providing not only respiratory support but also circulatory support. In veno-veno-arterial mode, the returning circuit from the pump was divided with a Y connector into 2 reinfusion circuits; each reinfusion circuit was connected to the contralateral side femoral vein and artery, respectively. The distribution of reinfusion flow was adjusted depending on the patient's cardiopulmonary status.
Conclusions: Although there is no consensus about the veno-veno-arterial mode of extracorporeal membrane oxygenation, this combined mode can be helpful in patients with acute refractory respiratory and cardiac failure, as shown in the present case. We need further experience and improvements in the circuit system used in the veno-veno-arterial mode of ECMO.

Keywords: Acute myocarditis, Acute respiratory distress syndrome, Extracorporeal membrane oxygenation

Introduction

ECMO (extracorporeal membrane oxygenation) has been useful mechanical devices to temporarily support heart or lung function during cardiopulmonary failure, leading to organ recovery or replacement (1, 2). Currently ECMO is indicated in patients with ARDS, who have severe hypoxia, uncompensated hypercapnia, and the presence of excessively high end-inspiratory plateau pressure, despite the best accepted standard of conventional ventilator care (3). In this setting, the veno-venous mode of ECMO is recommended, unless cardiac function is severely depressed. On the other hand, ECMO has been used as an external circulatory assisted device for temporary support of cardiac function in patients with acute myocarditis or cardiogenic shock (4, 5). In this situation, the veno-arterial mode is used, unless a severe hypoxic respiratory failure is also present.

In patients with combined severe ARDS and severe cardiogenic shock, instead of using veno-venous or veno-arterial mode alone, the use of the veno-veno-arterial mode of ECMO has been reported with a good success (6, 7). In this report, we described our experience of the veno-veno-arterial mode of ECMO in a patient with severe acute cardiopulmonary failure.

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Case report

A 27-year old male presented in the emergency room with a gradual worsening of dyspnea. He had been well until 3 weeks before admission, when he developed a cough. One week before admission he started feeling atypical chest pain. He had no previous medical history. In the emergency room, while he was breathing spontaneously with 10 L/min of oxygen via face mask with a reservoir bag, analysis of

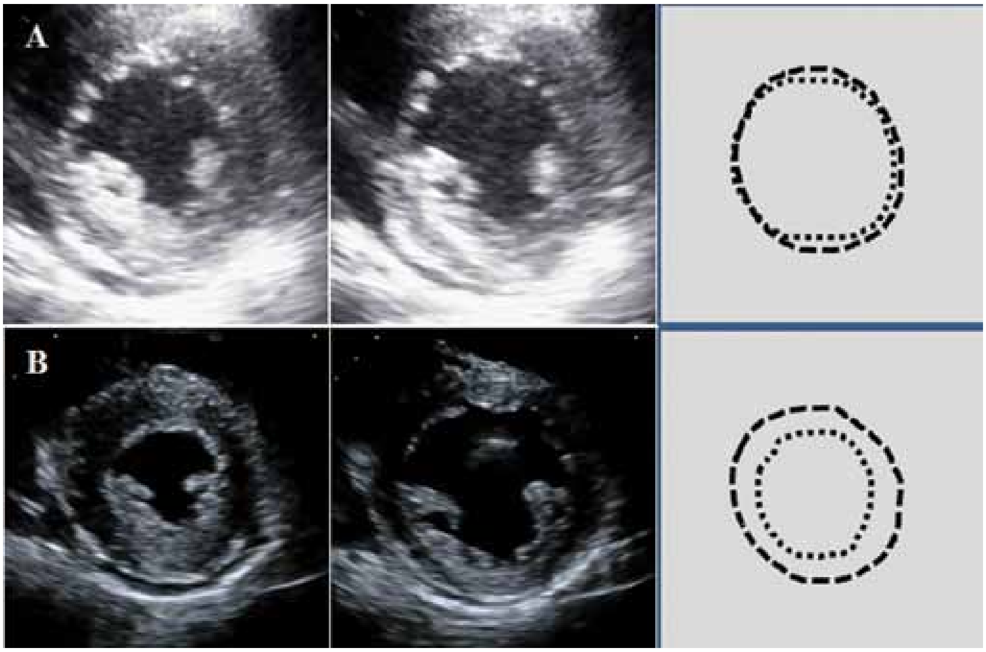

Fig. 1 - Serial transthoracic, parasternal, short-axis views of echocardiograms. (**A**) Echocardiographic **(left columns)** and schematic representation of left ventricular endocardial borders **(rights columns)** during diastole and systole on presentation; evidence of transient apical and midventricular wall dilatation of the left ventricle (LV) accompanied by global hypoakinesia. (**B**) At recovery, 8 days after presentation, transthoracic, parasternal, short-axis views at recovery showing normal LV inner dimensions and contractility; normalization of geometry and wall motion of LV.

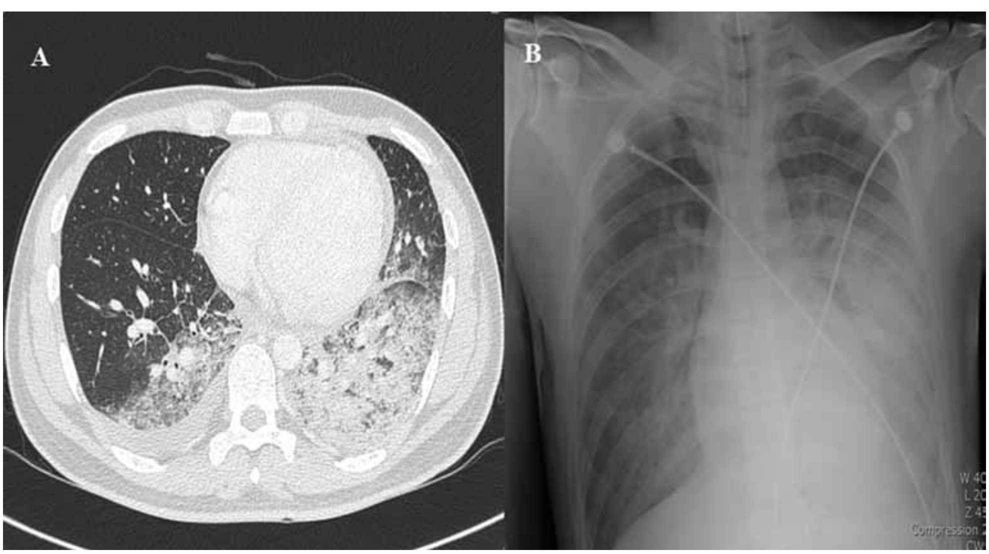

Fig. 2 - (**A**) Computed tomography of chest shows bilateral, dense, dependent consolidation, with areas of ground-glass opacification. (**B**) Chest radiograph on admission shows bilateral infiltrates. There is bilateral consolidation and a left pleural effusion.

arterial blood gases revealed that PaO_2 was 50 mmHg, $PaCO_2$ 32 mmHg, pH 7.35. He was intubated orotracheally and placed on mechanical ventilation. His blood pressure was 85/55 mmHg, pulse rate 120 beats/min, body temperature 37.3°C. The initial ECG showed nonspecific ST elevation with T-wave inversion in precordial leads. In transthoracic echocardiography (TTE), there was a severe global systolic dysfunction without regional wall motion abnormality of the left ventricle (LV) (Fig. 1A), strongly suggesting the presence of myocarditis. Troponin-I was 112.20 pg/mL and N-terminal pro-BNP was 10,026 pg/mL. Chest CT and plain chest radiographic findings were bilateral, predominantly peripheral, asymmetrical consolidation with a small amount of bilateral pleural effusion, and cardiomegaly, consistent with ARDS and acute myocarditis (Figs. 2A and B). It was decided not to proceed with a coronary angiography, because not only was the patient's condition too unstable for the coronary angiography to be performed, but the diagnosis of acute myocarditis was also so highly likely that a coronary angiography was not considered to be absolutely necessary to make a diagnosis at that point.

In the ICU, he developed severe hypoxemia in spite of maximal mechanical ventilator care with high PEEP and FiO_2 and even with inhaled nitric oxide. Also, the hemodynamic status was getting gradually worse even with the use of escalating doses of inotropic and vasopressors. For this refractory hypoxemia and cardiogenic shock, we decided to use veno-veno-arterial ECMO to provide both respiratory and cardiac support simultaneously. ECMO was initiated from right femoral vein cannulation for drainage route and reinfusion to the

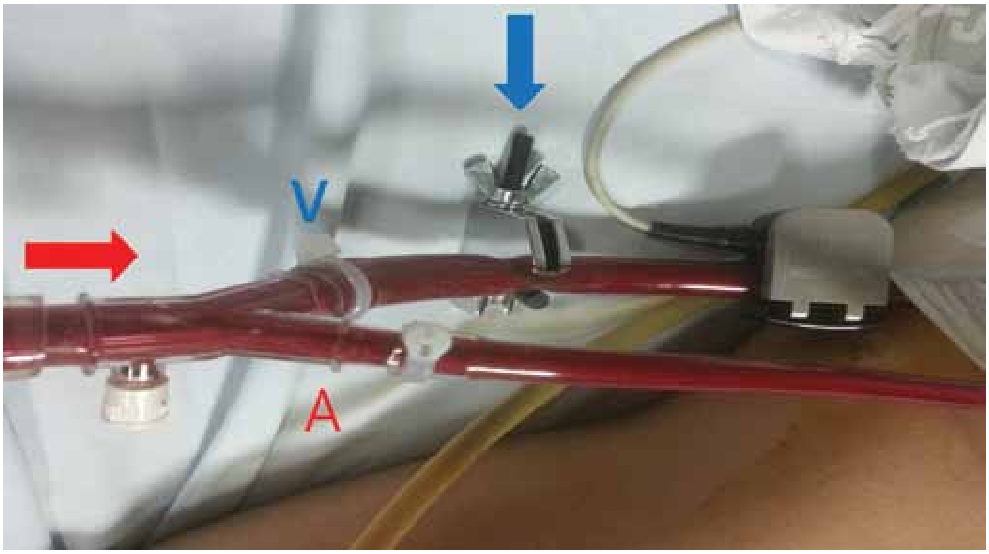

Fig. 3 - Distribution of reinfusion blood flow. (**Red arrow**) indicates flow direction. (**Blue arrow**) indicates clamp modulator. (**V**) venous reinfusion cannula, (**A**) arterial reinfusion cannula.

left femoral artery and vein. On ICU day 1, the initial ECMO setting consisted of the ratio of the total flow/arterial flow of 3.8/0.8 L/min. We maintained this setting for 2 full days. On the ICU day 3, repeated TTE showed further decreased cardiac contractility. For more cardiac support, we increased arterial reinfusion flow up to 2.2 L/min by clamping the venous reinfusion cannula (Fig. 3).

On ICU day 5, the patient was able to maintain mean arterial pressure around 70 mmHg without inotropics and he was able to breath spontaneously on pressure support mode with SpO_2 running around 97%. The ECMO setting was decreased to a total/arterial flow ratio of 3.5/0.7 L/min. On ICU day 7, the ECMO was weaned off without any complication. On ICU day 8, the patient was weaned off the ventilator and his trachea was extubated. There were no complications related to this new modified mode of ECMO. The follow-up TTE showed a significant improvement in LV systolic function (Fig. 1B). The patient recovered fully without any functional or neurological deficit and was transferred to the general ward on ICU day 8. He was discharged from hospital on day 14.

Discussion

Sometimes myocarditis can present as an acute fulminant disease with cardiovascular collapse. The acute fulminant form of myocarditis has been shown to have a better overall prognosis, but mechanical circulatory support is often needed to bridge patients to myocardial recovery (4). Previous studies have shown that the clinical outcome and long-term prognosis in patients with fulminant myocarditis supported by ECMO is favorable, similar to that observed in patients with nonfulminant myocarditis (8, 9). As such, an aggressive hemodynamic support is warranted for patients with fulminant myocarditis. If patients with fulminant myocarditis develop severe refractory hypoxemia owing to ARDS, however, the situation becomes a lot more complicated. Since the veno-arterial mode of ECMO is not able to provide adequate oxygenation supply, particularly in the brain and heart (6), in this situation the veno-venous mode is needed in addition to the veno-arterial mode of ECMO.

Combining veno-veno and veno-arterial ECMO by using the ECMO pump is technically complex, cost-ineffective and will likely encounter difficulties in cannulation. A single ECMO pump with veno-veno-arterial mode would be feasible in providing both the adequate oxygenation supply and cardiac support. However, the veno-veno-arterial mode of a single ECMO pump has difficulty with regulating the distribution of flow between the arterial and venous reinfusion circuits. When the reinfusion flow was divided into arterial and venous parts via a Y-connector, more blood flow tended toward the venous reinfusion circuit because of a relatively lower pressure and a larger cannula size in the venous side. In the present case, flow distribution was controlled by clamping the venous reinfusion circuit. There might be a risk of mechanical hemolysis caused by turbulent flow generated by the abrupt narrowing of the cannula lumen on clamping site, but in this case such an adverse effect was not observed.

Conclusions

The veno-veno-arterial mode of ECMO is technically feasible and a useful rescue strategy in treatment for concurrent cardiac and respiratory failure. Severe ARDS and cardiac dysfunction commonly occur simultaneously in the context of severe sepsis. Application of the veno-veno-arterial mode of ECMO can be considered in these complicated cases. But the benefits and disadvantages of the different modes should be evaluated in future trials. We also need further experience and improvements in the circuit system used in the veno-veno-arterial mode of ECMO.

Disclosures

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Conflict of interest: None of the authors have any conflicts of interest associated with this report.

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