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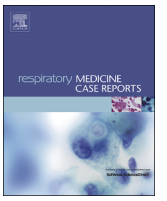

Case Report

Circulatory support with triple cannulation V-PaA ECMO in a patient with acute right ventricular failure and refractory hypoxemia secondary to diffuse alveolar hemorrhage: A case report

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ABSTRACT

A 48-year-old woman presented to the emergency department with a one-week history of progressive dyspnea. During her hospitalization, the diagnosis of diffuse alveolar hemorrhage was made. She subsequently developed respiratory failure and acute right ventricular failure. Despite medical treatment, she continued to experience distributive shock due to a generalized inflammatory response. Circulatory support with ECMO was needed. We opted for triple cannulation to manage the multiorgan failure as a bridge to recovery. We describe our experience with an uncommon cannulation technique: veno-pulmonary-arterial cannulation, which enabled us to address cardiogenic shock, refractory hypoxemia, and distributive shock, leading to the successful recovery of the patient.

1. Introduction

Extracorporeal membrane oxygenation (ECMO) has become an invaluable tool for managing severe cases of lung and heart failure, whether isolated or combined. Percutaneous ECMO circuits typically employ a dual cannulation approach, either veno-venous or veno-arterial configuration. A novel and advanced technique involves triple cannulation, where three large vessels are cannulated, resulting in veno-veno-arterial or veno-arterio-venous cannulation. Additionally, both veno-venous and veno-arterio-venous cannulation can be further refined into veno-pulmonary-arterial or veno-arterial-pulmonary arterial cannulation, respectively.

We describe our experience with a patient who required ECMO as bridge to recovery for right ventricular (RV) acute failure and hypoxemic respiratory failure due to diffuse alveolar hemorrhage (DAH) associated with Granulomatosis polyangiitis, despite the medical treatment the patient persisted with distributive shock due to the generalized inflammatory response so a triple cannulation was needed to manage the cardiogenic shock, the refractory hypoxemia and the distributive shock with a successful recovery of the patient.

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2. Case

We present the case of a 48-year-old woman with no significant medical history who presented to the emergency department with a one-week history of asthenia, adynamia, intermittent fever, and progressive dyspnea. She was evaluated by a family doctor who made the diagnosis of community-acquired pneumonia and started treatment with ceftriaxone 1g BID; due to worsening the dyspnea, she was referred to our hospital. Upon her arrival at the Emergency Department, a physical examination showed a heart rate of 137 bpm, respiratory rate of 40 rpm, blood pressure of 80/45 mmHg, oxygen saturation of 60 % at room air, cold skin, distended jugular veins, and bilateral generalized pulmonary rales. Emergency orotracheal intubation was performed, and a chest CT was obtained (Fig. 1), documenting a diffuse infiltrative opacification pattern.

Laboratory tests revealed anemia (8.2 g/dL), leukocytes (10.09/mm3), platelets (288,000/mm3), creatinine (5.7 mg/dL), elevated lactate dehydrogenase (716 U/L), and aspartate aminotransferase (67 U/L), N-terminal pro-B-type natriuretic peptide (NT-proBNP) was reported at 29,827 pg/mL, high-sensitivity troponin at 89 ng/mL, and C-reactive protein (CRP) at 300 mg/L.

The initial transthoracic echocardiogram (TTE) revealed dilation of right chambers, severe right ventricular systolic dysfunction, right ventricular fractional shortening (RVFS) of 11 %, tricuspid annular plane systolic excursion (TAPSE) of 11 mm, tricuspid regurgitation 2.9 m/seg, left ventricular ejection fraction (LVEF) of 45 % and an estimated cardiac index of 1.06 (Fig. 2).

Due to hemodynamic instability, dual vasopressors (norepinephrine and vasopressin) and inotropic therapy (levosimendan) at high doses were required. However, refractory cardiogenic shock persisted, and refractory hypoxemia despite optimal mechanical ventilatory support. The patient developed renal dysfunction requiring continuous renal replacement therapy.

The ECMO team evaluated the patient, and the hospital survival rate was calculated at 42 % based on the "Survival after Veno-Arterial ECMO Score" (SAVE-Score). Circulatory support was initiated using veno-arterial extracorporeal membrane oxygenation (VA-ECMO) as a bridge to recovery. Peripheral cannulation was performed in the right femoral artery and vein. Due to the right ventricular dysfunction, a second return cannula was placed by right jugular cannulation and placed into the Pulmonary Artery (Fig. 3, Fig. 4). The flow was titrated to 4.5 L/min. After the initiation of circulatory support, the patient's condition improved with hemodynamic stabilization. Vasopressors were discontinued within the next 12 hours, and immediate improvement in oxygen saturation was documented.

Once the patient was stabilized and due to the pulmonary, hematologic, and renal manifestations, a Pulmonary-renal syndrome was suspected. An immunological panel was requested, which showed elevated antinuclear antibodies titers (1:160), hypocomple-

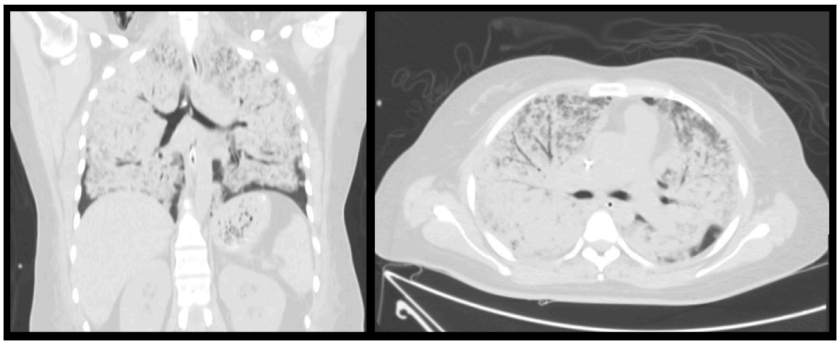

Fig. 1. Chest CT axial and coronal axis. Showed a diffuse infiltrative opacification pattern compatible with diffuse alveolar hemorrhage.

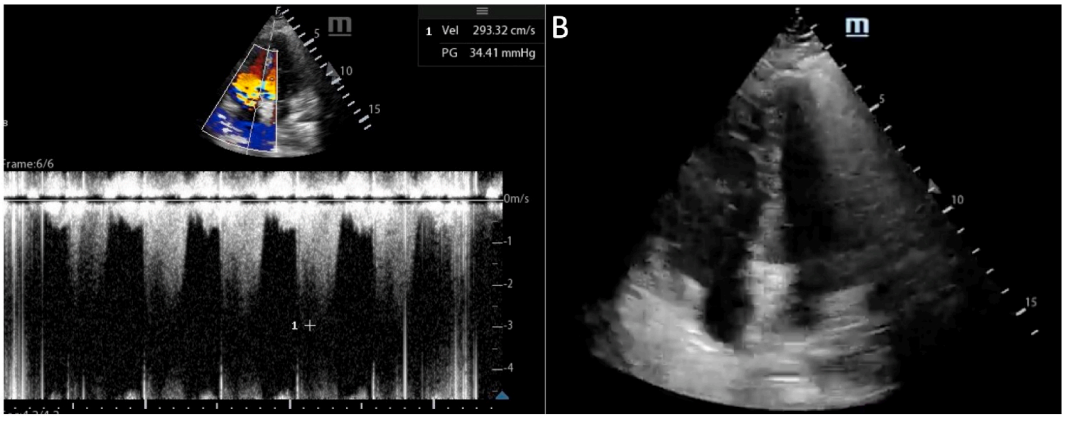

Fig. 2. Initial Transthoratic echocardiogram obtained with the patient in prone position. A) 4 Chambers view showing the Tricuspid regurgitation of 2.9 m/seg measured with CW. B) 4 chamber view showing the right ventricular dilation, systolic RV disfunction was assessed.

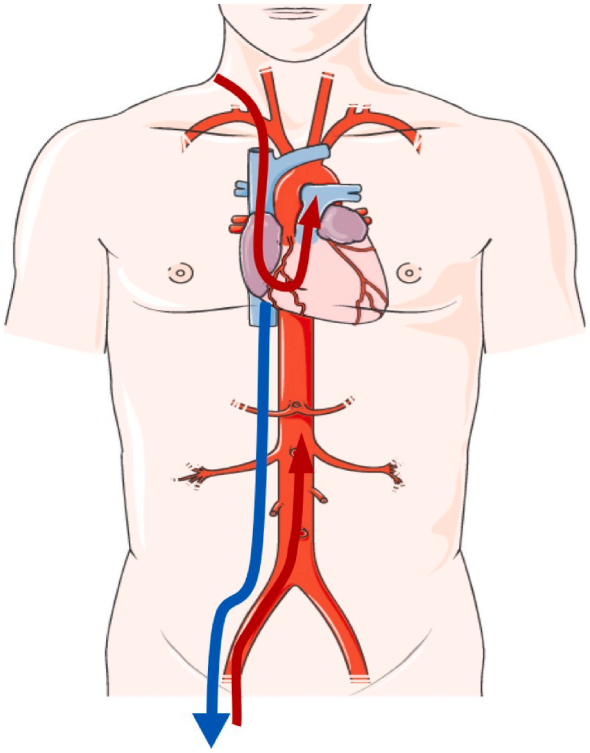

Fig. 3. V-PaA ECMO configuration. Blue arrow represents the blood flow through the extraction cannula extracting from the inferior cavoatrial joint and the Red arrows represents the return cannulas, the first one placed in the aorta and the second one in the pulmonary artery. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

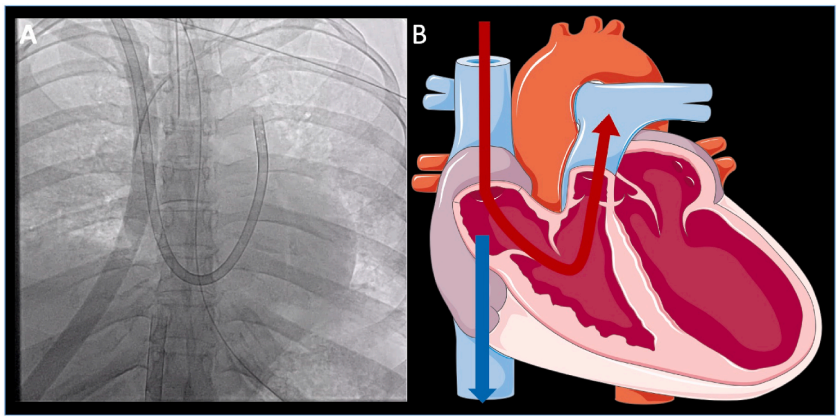

Fig. 4. V–Pa ECMO configuration detail. A) Cardiac fluoroscopy showing the extraction cannula placed in the inferior cavoatrial joint and the return cannula placed in the pulmonary artery. B) V–Pa ECMO represented in a cardiac diagram showing the extraction cannula (Blue) and the return cannula (Red). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

mentemia (C3 77mg/dL and C4 8.5 mg/dL), and positive PR3-antineutrophilic cytoplasmic antibodies (c-ANCA). Clinical and biochemical criteria for the diagnosis of Granulomatosis with polyangiitis were fulfilled, leading to immunosuppressive treatment with methylprednisolone pulses, plasma exchange, immunoglobulins, and anticoagulation.

Following the initiation of immunosuppressive treatment, a favorable renal and pulmonary response was achieved. On the fourth day of V-PaA ECMO support, adequate echocardiographic criteria for weaning from circulatory support were achieved, with LVEF of 50 %, ITV of 20 cm, lateral mitral S′ of 8 cm/s, TAPSE of 15 mm, right ventricular fractional shortening of 30 %, even though the pulmonary support was still needed, It was decided to remove the return cannula (Right Femoral artery) and due to the recovery of the right ventricular function, the second return canula (Right yugular vein) was pulled out of the pulmonary artery and placed in the superior cavoatrial junction turning the configuration into a V–V ECMO. The patient was successfully extubated on the fifth day of

ECMO support and continued until the pulmonary recovery criteria were achieved. V–V ECMO was stopped on the 20th day of circulatory support.

Within a few days, the patient was transferred in stable condition to the internal medicine department to continue immunosuppressive therapy and pulmonary rehabilitation.

3. Discussion

Acute RV failure secondary to acutely increased right ventricular afterload is a life-threatening condition that may arise in different clinical settings. Patients at risk of developing this condition usually present with an acute pulmonary disease [1].

In this case, the patient developed acute RV failure secondary to DHA, this is a severe pulmonary manifestation of ANCA associated vasculitis. It's occurrence is a factor of poor prognosis with a mortality of up to 10–25 % [2] and has a similar pathophysiology with ARDS; Due to the systemic inmune response, the RV failure was worsened by vascular pulmonary alterations, hypoxia, hypercapnia, and the effects of mechanical ventilation [3]. All of these factors raised the RV afterload and the systemic inflammation caused a generalized vasoplegia leading to a distributive shock profile diminishing even more the systemic perfusion.

The management of combined respiratory and right ventricular failure is highly complex due to the interdependency of both organ systems. If decompensation occurs under conventional treatment, several options for mechanical cardiorespiratory support can be chosen: VA ECMO, VV ECMO, or VAV ECMO [4]. However, an individualized decision should always be made based on the risk-benefit ratio of each device.

According to current guidelines, short-term circulatory support should be considered in patients with cardiogenic shock as bridge to recovery [5].

The most usual ECMO setup involves dual-cannulation, specifically veno-venous (VV) or veno-arterial (VA) cannulation using two large cannulae. VV-ECMO draws deoxygenated blood from the right atrium, passes it through the ECMO system for oxygenation and decarboxylation, and then returns it to the right atrium. Essentially functioning as an external lung, VV-ECMO is typically employed in patients with severe acute respiratory distress syndrome (ARDS). In contrast, VA-ECMO withdraws blood from the right atrium and returns it, after processing through the ECMO device, to the patient's arterial system, often through the femoral artery towards the aorta. VA-ECMO creates a significant extracorporeal right-to-left shunt and primarily offers hemodynamic support, while its impact on oxygenation varies depending on factors such as cannulation sites, the patient's cardiac output, and respiratory function [6].

V-PaA ECMO is a special variant of VAV cannulation; While VAV-ECMO combines the characteristics of VV and VA ECMO, VAPa ECMO aims to provide additional support for right heart failure during VAV-ECMO. To achieve this, the venous cannula returning blood is directed through the tricuspid valve, the right ventricle, and the pulmonary valve to reach the pulmonary artery. This procedure requires guidance via angiography or transesophageal echocardiography, and a flexible 17 French cannula, similar to VPa cannulation, must be used for this purpose. The smaller inner diameter of this cannula inherently affects the flow balance of both returning cannulas, which is further adjusted by a clamp and monitored by a flow sensor, as in VAV-ECMO. Like VA and VPa cannulation, it's recommended to position the tip of the draining cannula in the mid right atrium to ensure uniform drainage of blood from both the upper and lower body.

Once the circulatory support was initiated, the patient maintained adequate perfusion and reversed multi-organ failure while recovering ventricular function, and due to the change of the ECMO configuration we were able to stop the cardiac support and continue the pulmonary support until we reach a favorable clinical response.

This is an exceptional case of success in which V-PaA-ECMO was employed as a bridge-to-recovery therapy in a patient with refractory hypoxemic respiratory failure and RV acute failure due to DHA associated with Granulomatosis polyangiitis.

The case highlights that timely initiation of circulatory support is essential to maintain tissue perfusion while ventricular function recovers, we used a triple cannulation because that was the optimal ECMO configuration at the beginning, when we documented an improvement of the cardiac function with a control of the inflammatory response we were able to change the configuration to maintain a pulmonary support until the immunosuppressive treatment lead to the pulmonary recovery.

CRediT authorship contribution statement

Uriel Encarnación-Martínez: Writing – original draft, Conceptualization. **Abraham Torres-Pulido:** Investigation. **Emmanuel Adrián Lazcano-Díaz:** Writing – original draft. **Daniel Manzur-Sandoval:** Writing – original draft. **Luis Augusto Baeza-Herrera:** Visualization. **Francisco Javier González-Ruiz:** Writing – review & editing. **Gian Manuel Jiménez-Rodríguez:** Investigation. **Gustavo Rojas-Velasco:** Writing – review & editing.

Declaration of competing interest

The authors declare they have no known competing financial interest or personal relationships that could have appeared to influence the work reported in this paper.

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