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CORRESPONDENCE

Venoarterial extracorporeal membrane oxygenation support for two patients after pulmonary thromboendarterectomy

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To the Editor,

Most patients with chronic thromboembolic pulmonary hypertension (CTPH) can benefit from the pulmonary thromboendarterectomy (PTE) irrespective of the severity of pulmonary hypertension, right-heart failure, and hypoxia preoperatively.[1-3] Some patients cannot be weaned from cardiopulmonary bypass (CPB) after PTE secondary to reperfusion injury and massive pulmonary hemorrhage, which are the main causes of in-hospital deaths. Extracorporeal membrane oxygenation (ECMO) may be utilized as a treatment for such critically ill patients, providing basic life support and time for recovery. Recent reports are now suggesting support ECMO using the venoarterial mode (VA ECMO). Here we report two successful cases of VA ECMO in two patients who were also critically ill after PTE. The patients gave their written consent for the publication of this article.

In the first case, a 49-yr-old female presented with recurrent syncope and shortness of breath lasting for one month after being immobilized for two months due to a fracture of the left patella. Transthoracic echocardiography (2D TTE) showed an enlarged right ventricle, a normal-sized left ventricle, and right ventricular systolic pressure (RVSP) of 87 mmHg. Cardiac magnetic resonance imaging showed considerable thrombus extending from the main pulmonary artery to the segmental pulmonary arteries. Right-heart catheterization indicated severe pulmonary arterial hypertension with a systolic pulmonary arterial pressure of 86/37 mmHg (mean, 50 mmHg). Thromboendarterectomy was performed under ten minutes of deep hypothermic circulatory arrest (DHCA). The aortic cross-clamping time was 65 min. The pulmonary arterial pressure on weaning from CPB was equal to the pressure of the systemic circulation, while the volume of blood drainage from the tracheal catheter was greater than 400 mL, hence, the patient was continued on CPB. Bleeding was controlled during CPB; however, the patient could not be weaned from it even after an intervening period. Thus, VA ECMO was established through the femoral artery and vein, respectively, in the operating room to wean her from CPB. The VA ECMO flow rate was maintained at $\sim$2.5 L·min^{-1}, pulmonary arterial pressure decreased to 53/40 mmHg, and the pulmonary hemorrhage was controlled. Heparin was not used after surgery. The total duration of ECMO support was 20 hr. Twenty-six units of packed red blood cells, 2,000 mL of blood plasma, and 1 unit of platelets were transfused postoperatively. The mean pulmonary pressure was $\sim$50 mmHg. The ECMO was discontinued when bleeding from the tracheal catheter had stopped and the vital signs were stabilized. The patient was discharged from hospital 20 days after surgery. At the time of hospital discharge, the 2D TTE RVSP was 55 mmHg.

In the second case, a 19-yr-old male was immobilized for one month due to a fracture of the right ankle. Four months after the fracture, he began to complain of shortness of breath, palpitations, and fatigue. Two-dimensional TTE showed an enlarged right ventricle, and the RVSP was 88 mmHg. A computed tomography pulmonary angiography showed involvement of the main branch of the left and right pulmonary arteries as well as parts of the pulmonary lobar and segmental branches, more extensive on the left side. Right-heart catheterization showed a pulmonary artery pressure of 96/34 mmHg (mean, 54 mmHg), and the pulmonary vascular resistance was 968 dyne·sec^{-1}·cm^{-5}

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(12.1 Wood units). The PTE procedure was carried out under DHCA deep hypothermia lasting 33 min (aortic cross-clamping time was 118 min). The pulmonary pressure was higher than the aortic pressure on the initial attempt to wean from CPB, the arterial saturation declined, and the $PaCO_2$ was 78 mmHg. The VA ECMO was established through the femoral vessels, and the patient's circulatory and respiratory status was maintained stable with a flow rate of 2.7 $L{\cdot}min^{-1}$. The pulmonary arterial pressure decreased to 75/34 mmHg (mean, 50 mmHg). Throughout the ECMO support, activated clotting time was maintained from 180 sec to 220 sec, and ventilatory settings were reduced to a minimum. During ECMO, the patient was transfused with 4 units of packed red blood cells, 200 mL of blood plasma, and 3 units of platelets. After 80 hr of ECMO support, the patient was weaned successfully from ECMO. He was discharged from hospital 30 days after surgery with a 2D TTE measured RVSP of 57 mmHg.

Thromboendarterectomy has become the major treatment for CTPH. While the related surgical techniques and postoperative care have improved, perioperative mortality remains high due to hemorrhage and reperfusion injury. Hemorrhage is multifactorial, as a result of enhanced permeability of the vasculature and persistent hypertension.[4] Hemorrhage can also result from pulmonary artery rupture. Reperfusion injury of the lung usually occurs within the first 48 hr after the surgical procedure. It is characterized by non-specific reperfusion injury of the alveoli, pulmonary edema, hypercarbia, and hypoxemia. Some patients with reperfusion lung injury do not improve with mechanical ventilation. Extracorporeal membrane oxygenation has been shown to improve survival in a critically ill subset of patients. Pretorius *et al.*[5] described the novel use of ECMO to control life-threatening pulmonary hemorrhage. Thistlethwaite *et al.*[3] reported that 20 of 1,800 patients who underwent pulmonary endarterectomy required ECLS support. These studies showed that venovenous ECMO (VV ECMO) may be used to salvage patients with fulminant reperfusion edema with a 30.0% survival rate. Berman *et al.*[6] reported seven cases of VA ECMO support where four patients survived to decannulation.

The two patients we describe were supported by VA ECMO after PTE due to life-threatening pulmonary hemorrhage or cardiopulmonary failure, and both patients survived to hospital discharge. A VA ECMO strategy for these types of patients may be beneficial for three reasons. First, a VA strategy constructs a new circuit that enables venous blood flow to bypass the pulmonary artery, thus reducing pulmonary pressure and diverting blood away from injured vasculature. Second, a VA strategy can alleviate the adverse impact of right-heart failure on the circulation, and it improves the blood supply for the whole body. Third, a VA strategy can provide efficient gas exchange and reduce ventilator-induced lung injury simultaneously due to the protective ventilation strategy. This protocol provides time for repair of pulmonary arterial injury, lowers pulmonary vascular resistance, and improves right and left ventricular diastolic function. Compared with a VA strategy, a VV strategy can provide only respiratory support; it cannot offload the pulmonary vasculature or the probability of massive hemorrhage. In conclusion, ECMO can be an alternative therapy for critically ill patients after PTE. A VA strategy can effectively alleviate massive hemorrhage, reperfusion injury, and residual pulmonary hypertension.

Competing interests None declared.

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