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Case Presentation

Role of Veno-arterial Extracorporeal Membrane Oxygenation in Left Ventricular Conditioning after Lung Transplantation

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Abstract

End-stage pulmonary hypertension alters intracardiac pressures, leading to distention and failure of the right ventricle, leftward shifting of the intraventricular septum, and, thus, underfilling of the left ventricle (LV). Following the resolution of severely elevated pulmonary vascular resistance with bilateral lung transplantation, the LV is exposed to relatively high filling pressures from a potentially hypertrophic right ventricle pushing blood through normalized pulmonary vascular resistance. Veno-arterial extracorporeal membrane oxygenation (V-A ECMO) may be a valuable tool to provide a more gradual exposure of the LV to the newly available preload in the immediate postoperative phase of transplantation, thereby reducing the likelihood of primary graft dysfunction developing from the LV diastolic dysfunction. This paper presents a case in which V-A ECMO was initiated during cardiac arrest in a patient with advanced pulmonary hypertension and right ventricular failure and maintained for two days for postoperative patient stability and cardiac conditioning. The discussion includes data from transplant programs using this method to reduce the need for dual organ transplantation and postoperative primary graft dysfunction in the allograft.

Keywords: lung transplantation, diastolic dysfunction, right ventricular failure, veno-arterial extracorporeal membrane oxygenation, pulmonary hypertension, left ventricle remodelling

Background

Idiopathic pulmonary artery hypertension lung transplant patients have the highest 1-year mortality (about 25%). Still, they also have the second-best long-term survival rate compared to transplant recipients with other pulmonary diseases.[1] One factor contributing to this statistic is primary graft dysfunction (PGD) caused by a small and unconditioned left ventricle (LV) prone to developing diastolic dysfunction when exposed to a normal or relatively high pre-load after transplantation. These patients may benefit from veno-arterial extracorporeal membrane oxygenation (V-A ECMO) bridging peri-operatively to allow the LV time to condition to new filling pressures, thereby reducing the incidence of PGD in this population of transplant recipients.

Case Report

Patient History and Presentation

The patient, in this case, is a 46-year-old female who presents to the emergency department with four days of worsening productive cough, shortness of breath, and abdominal pain. She has a past medical history significant for idiopathic pulmonary artery hypertension diagnosed in 2019, hypothyroidism, and type 2 diabetes. She complied with her home medication regimen of dual pulmonary artery hypertension therapy (bosentan and sildenafil) and optimized heart failure guideline-directed medical therapy (metoprolol succinate, spironolactone, and empagliflozin).

In the emergency department, she developed acute hypoxia with perioral cyanosis and cool extremities. Her arterial blood gas showed a compensated metabolic acidosis with a partial pressure of oxygen of 166 mm Hg and a lactate of 6.6 mmol/L, mild transaminitis, as well as elevated brain natriuretic peptide and troponin T levels. Her chest x-ray revealed an enlarged cardiac silhouette and right lower lobe consolidation.

Diagnosis and Intervention

Her initial transthoracic echocardiogram (Figure 1) showed a severely enlarged right ventricle causing a small LV internal diameter, moderate pericardial effusion without tamponade, and abnormal septal wall motion. The parasternal short view showed a D-sign during diastole and systole. Per the report, there was no LV hypertrophy, and the diastolic function of the LV was difficult to determine. Her right atrium was severely dilated, and her tricuspid valve was reported as severely regurgitant with a dilated inferior vena cava and mild pulmonic valve regurgitation. Her initial right heart catheterization measured a pulmonary artery mean pressure of 59 mm Hg, pulmonary artery occlusive pressure of 9 mm Hg, right atrial pressure of 19 mm Hg, a Fick cardiac index of 2.17 L/min, and a pulmonary artery saturation of 55%. Following the procedure, she was initiated on intravenous (IV) treprostinil and dopamine with aggressive diuresis.

After titrating up to 21 ng/kg/min of IV treprostinil and 2 mcg/kg/min of dopamine, she underwent a second right heart catheterization, which showed refractory disease with a pulmonary artery mean pressure of 63 mmHg, pulmonary artery occlusive pressure of 10 mmHg, a right atrial pressure of 21 mmHg, and a Fick cardiac index of 2.39 L/min. At this time, the lung transplant team was consulted for evaluation, and five days later, she arrested and required intubation, V-A ECMO cannulation, and high-dose vasopressors. Her transthoracic echocardiogram following this event showed decreased LV function with an ejection fraction of 52%, and the right ventricle was decompressed by V-A ECMO, revealing some right ventricular hypertrophy.

Outcome

About a month after the arrest, she received a bilateral lung transplant and a tricuspid valve repair. She came out of the operating room on V-A ECMO. She required a massive transfusion for hemorrhagic shock postoperatively, leading to persistently elevated LV filling pressures despite diuresis. To preserve her pulmonary artery anastomoses, her pulmonary artery diastolic pressure was used as a surrogate for a pulmonary artery occlusive pressure. It ranged from 10 mm Hg immediately postoperatively to 30 mm Hg after her transfusions. She developed grade 3 PGD, which slowly resolved with aggressive diuresis and intermittent, pharmacologic afterload reduction. Two days following her transplant, she was transitioned from V-A to veno-venous (V-V) ECMO with only minimal vasopressor support. After changing cannulation from V-A to V-V, her transthoracic echocardiogram (Figure 2) reported a hyperdynamic LV with an ejection fraction of 79%, indeterminate LV diastolic function, and an enlarged, hypertrophic right ventricle which was no longer compressing the LV.

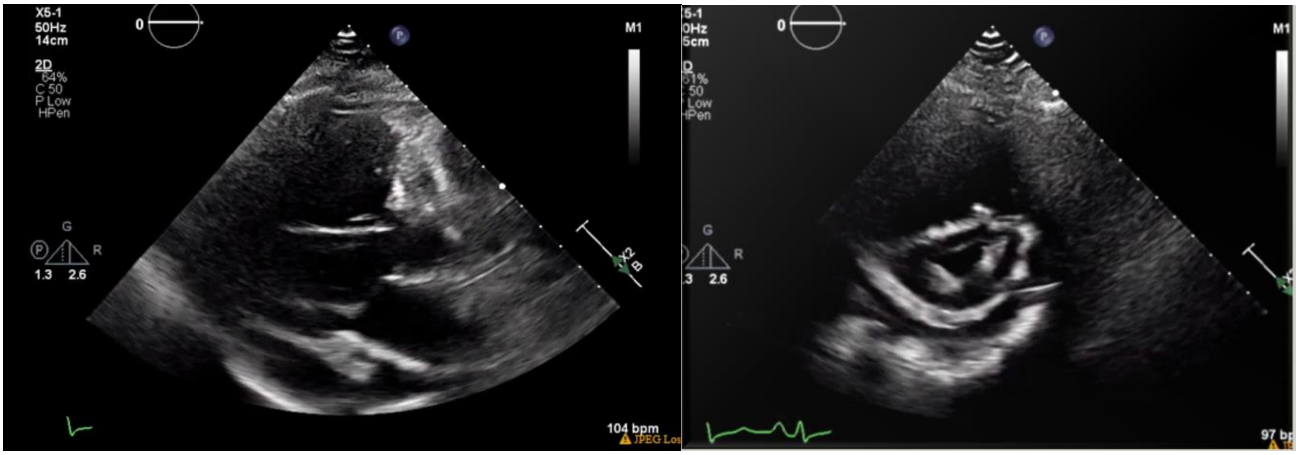

Figure 1. Pre-transplant transthoracic echocardiogram. Pre-transplant parasternal long axis and short axis views show a distended right ventricle and D-sign. Reported values: left ventricle ejection fraction 73%, left ventricle internal diameter during diastole 2.74 cm.

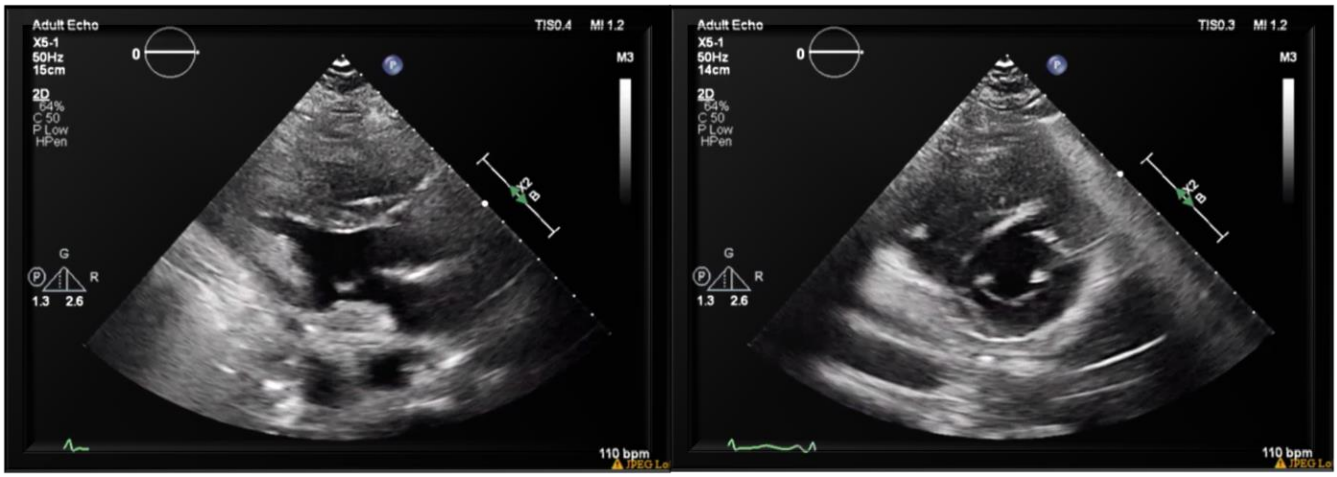

Figure 2. Post-transplant transthoracic echocardiogram. Post-transplant parasternal long axis and short axis views show a decompressed right ventricle and larger left ventricle internal diameter. Reported values: left ventricle ejection fraction 79%, left ventricle internal diameter during diastole 3.11 cm.

Comment

The concept of right ventricular failure in the setting of pulmonary artery hypertension contributing to an unconditioned LV is not new.[2-3] However, treatment approaches to this problem vary from peri-operative V-A ECMO to inotropes with aggressive diuresis. Primary pulmonary artery hypertension comprises 5-6% of lung transplantation nationally, and postoperative ECMO data is not tracked.[1] However, in the center where this case occurred, postoperative ECMO is rarely utilized in pulmonary hypertension lung transplant cases. This patient was particularly vulnerable to PGD because of her peri-operative mechanical ventilation and the large volume of blood administration due to coagulopathies related to IV prostacyclin analogue administration. She remained on V-A ECMO postoperatively, with the understanding that she was at high risk for developing PGD and requiring cardiac support in the immediate postoperative period that may not have been adequately supported with IV inotropes alone.

In 2002, a team in Austria published a prospective study (n=17) in which they found that peri-operative V-A ECMO, instead of cardiopulmonary bypass, reduces postoperative reperfusion injury by reducing forceful blood flow through the graft initially.[4] Their study resulted in more controlled reperfusion, less aggressive ventilation strategies, and improved postoperative hemodynamics.[4] Another prospective study of 23 bilateral lung transplants for patients with severe pulmonary hypertension in Germany looked at V-A ECMO as a bridge for LV conditioning while monitoring left atrial pressures, invasive hemodynamics, and LV diameter for weaning.[5] They reported decreased total ventilator, intensive care unit, and inpatient days with increased 90-day and 1-year survival rates (94%) in their transplant-ECMO group as compared to their other groups.[5]

The physiology of a poorly conditioned LV also exists among the chronic thromboembolic pulmonary hypertension population, as evidenced by a study conducted in the Netherlands, which revealed a reversible reduction in LV free wall mass likely related to atrophic remodeling in the setting of right ventricular failure among patients with chronic thromboembolic pulmonary hypertension (CTEPH).[6] Though the application of V-A ECMO is not without risk of complications, this is an exciting topic and deserves the focus of future research because of its potential to reduce the need for dual heart-lung transplantation and improve the one-year survival of pulmonary hypertension patients undergoing transplantation. The concept also may have application among CTEPH patients undergoing pulmonary thromboendarterectomy.

Acknowledgments

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