



# Unusual Indication for Extracorporeal Membrane Oxygenation Immediately After Successful Sequential Bilateral Lung Transplantation: A Case Report

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## ABSTRACT

**Background.** Ischemia-reperfusion injury-induced primary graft dysfunction after lung transplantation is a major cause of early morbidity and mortality.

**Case Report.** We report an unusual case of primary graft dysfunction grade III following uneventful off-pump bilateral sequential lung transplantation caused by paradoxical left ventricular failure due to systolic anterior motion of the mitral valve-induced left ventricular outflow tract obstruction. Cardiac failure was precipitated by profound dehydration and administration of high doses of vasopressin and norepinephrine. Immediate connection to extracorporeal membrane oxygenation treated the graft failure-associated respiratory-pulmonary hypoxia and reversed the cardiogenic shock syndrome.

**Conclusions.** Hypovolemia together with a hyperdynamic state resulting from catecholamine administration may result in the development of dynamic left ventricular outflow tract obstruction even if baseline cardiac evaluation is unremarkable. Early detection and intensive efforts to reverse the underlying conditions including cessation of catecholamine therapy and correction of hypovolemia are essential.

**I**SCHEMIA-REPERFUSION injury-induced primary graft dysfunction (PGD) after lung transplantation (LTx) is a major cause of early morbidity and mortality; it is characterized by pulmonary edema with diffuse alveolar damage manifesting clinically as progressive hypoxemia with radiographic pulmonary infiltrates [1]. We present an unusual case of PGD grade III following uneventful off-pump bilateral sequential LTx.

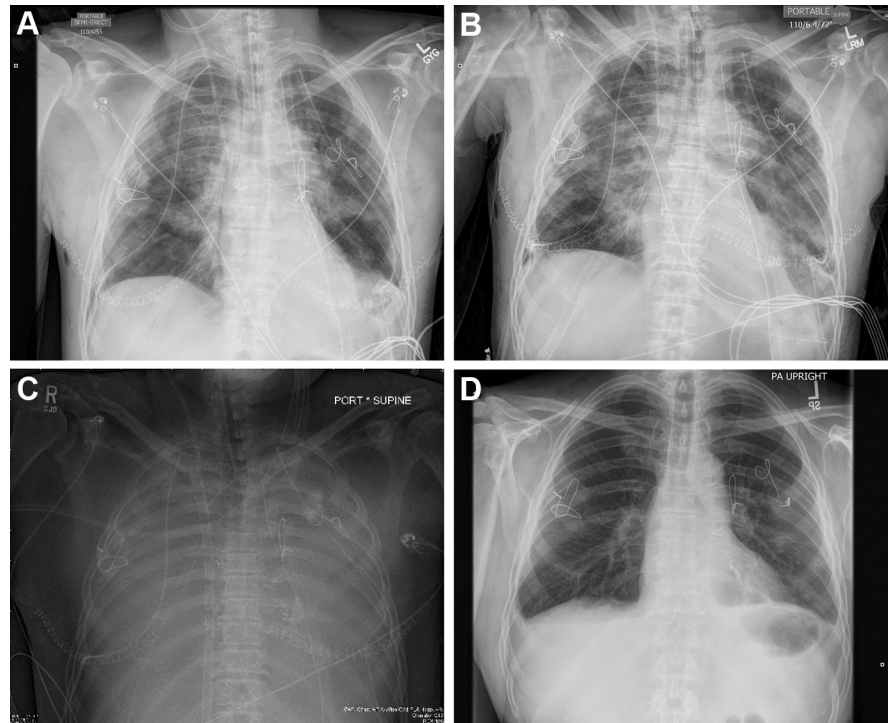
## CASE REPORT

A 53-year-old man with idiopathic pulmonary fibrosis and moderate secondary pulmonary arterial hypertension underwent urgent off-pump bilateral sequential LTx performed as described previously [2]. Following reperfusion, initial cardiac and pulmonary functions were good. The patient was transferred to intensive care on norepinephrine (0.08 µg/kg/min) and vasopressin (0.8 units/min) with very restrictive intravenous (iv) fluid maintenance according to our center's specific LTx protocol.

Postoperative chest x-rays documented initial mild ischemia-reperfusion injury (Fig 1A), which worsened rapidly over the next 24 hours (Fig 1B, 1C). Increasing oxygen requirements to 100% fraction of inspired oxygen and end-expiratory pressure ventilator settings to

15 mm Hg did not attenuate the respiratory-pulmonary hypoxic state (O<sub>2</sub> saturation decreased to 85%–89%, arterial partial pressure of O<sub>2</sub> to fraction of inspired oxygen ratio to 60 mm Hg, pH 7.124). Despite aggressive volume resuscitation and additional inotropic support (epinephrine 0.15 µg/kg/min), the cardiac index remained <1.5 L/[min·m<sup>2</sup>]. Immediate trans-thoracic echocardiography (TTE) demonstrated severe mitral valve regurgitation (MR). Grade III injury score was used for diagnosis of severe post-transplantation reperfusion injury and PGD associated with metabolic acidosis, systemic hypoperfusion, and uncontrollable left ventricular failure. Persistent hemodynamic and respiratory instability required further resuscitation and emergency cannulation for veno-arterial extracorporeal membrane oxygenation (ECMO). Intraoperative trans-esophageal echocardiography (TEE) confirmed severe MR but also documented near total left ventricular outflow tract (LVOT) obstruction by the very elongated anterior mitral valve leaflet (3.2 cm; Fig 2A). After initiation of ECMO support with 4.0–4.5 L/min, rehydration and discontinuation of pressors, systolic anterior motion (SAM) disappeared and MR immediately improved.

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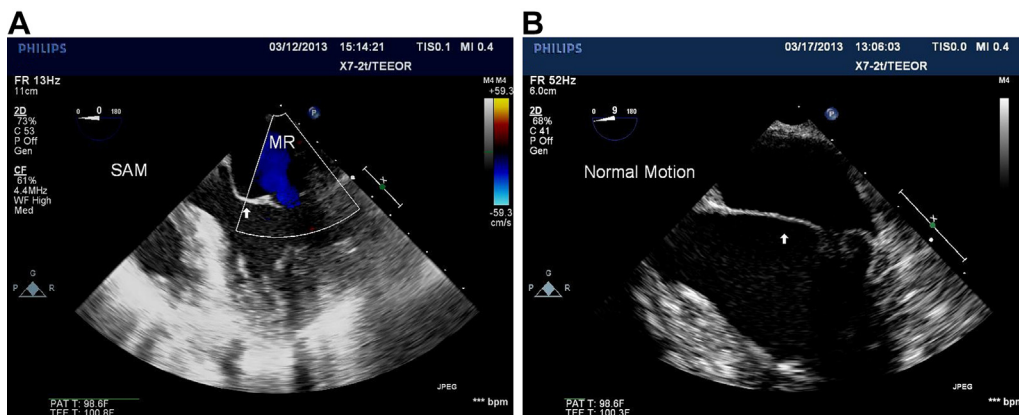
**Fig 1.** Chest radiographs. **(A)** Immediately after bilateral sequential LTx: mild pulmonary edema. **(B)** Eight hours postoperatively: worsening pulmonary edema, grade III ischemia-reperfusion injury. **(C)** Postoperative day 2: severe bilateral pulmonary edema while on veno-arterial extracorporeal membrane oxygenation. **(D)** Four weeks post-surgery with complete resolution of pulmonary edema and lung injury.

Within 12 hours, lactate level normalized and hypoxia and cardiopulmonary dysfunction improved. Cardiopulmonary recovery was confirmed on day 6 (Fig 2B), and ECMO was discontinued. Respiratory-pulmonary function improved further after a tracheostomy and was normalized 4 weeks post-surgery (Fig 1D).

**DISCUSSION**

Although the majority of lung transplantation centers apply a perioperative dehydration regimen to protect newly grafted donor lungs and to prevent severe ischemia-reperfusion

injury, our case of transient heart failure was paradoxically induced by extreme dehydration and catecholamine therapy precipitating intraventricular obstruction and significant MR, and thereby inducing an acute decrease in cardiac output along with hemodynamic impairment. Classically, cardiogenic shock is characterized by increased pulmonary capillary wedge pressure (PCWP) and decreased cardiac output, whereas hypovolemic shock includes low PCWP and reduced cardiac output. Due to different management strategies, distinguishing between cardiogenic and hypovolemic



**Fig 2.** **(A)** Demonstration of systolic anterior motion (SAM, see arrow) resulting in partial obstruction of the LVOT with MR. **(B)** Normal position and coaptation of the anterior and posterior leaflets of the mitral valve during systole while on veno-arterial ECMO and weaned off catecholamines and vasopressors. Arrow, anterior leaflet of the mitral valve during late systole.

shock is critical. In the present case, pulmonary infiltrates were notable and were considered a consequence of elevated PCWP due to increased left arterial pressure, which resulted from intracardiac dynamic obstruction and significant MR. Therefore, the cause of the patient's rapid deterioration was cardiogenic rather than graft-related, and early recognition of the unexpected hemodynamic feature was essential for appropriate treatment.

In conclusion, the present case serves as a reminder that hypovolemia together with a hyperdynamic state resulting from catecholamine administration may result in the development of dynamic LVOT obstruction even if baseline

cardiac evaluation is unremarkable. Early detection and intensive efforts to reverse the underlying conditions, including cessation of catecholamine therapy and correction of hypovolemia, are essential.

#### REFERENCES

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