Independent Lung Ventilation for the Management of Unilateral Reperfusion Pulmonary Edema After Pulmonary Thrombendarterectomy: A Case Report

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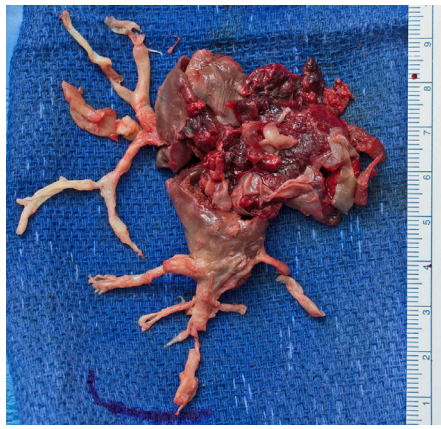
Independent lung ventilation (ILV) has been successfully used in the management of unilateral lung pathology to improve hypoxemia refractory to conventional mechanical ventilation.[1–3] ILV via double lumen endotracheal tube (DLT) allows tailoring an independent ventilatory strategy for each lung. It has proved useful in cases of asymmetric lung pathology, including pulmonary contusion, bronchopleural fistula, unilateral pneumonia, single lung transplantation, and reexpansion pulmonary edema.[1] We report a case in which ILV was successfully used in the perioperative setting as a rescue strategy for the management of unilateral reperfusion pulmonary edema (RPE) after pulmonary thrombendarterectomy (PTE).



Pulmonary arterial tree cast from right pulmonary thrombendarterectomy.

Central Message

Independent lung ventilation successfully corrected refractory hypoxemia due to unilateral reperfusion edema after pulmonary thrombendarterectomy.

CLINICAL SUMMARY

A 78-year-old male with progressive functional decline secondary to chronic thromboembolic pulmonary hypertension was scheduled to undergo PTE. Preoperative imaging showed asymmetric disease burden, mostly affecting the right lung (Fig. 1A). PTE was performed via median sternotomy under deep hypothermic circulatory arrest. The right pulmonary artery was endarterectomized as shown by surgical specimen (Fig. 1B). The left main pulmonary artery was patent up to segmental branches where webbing was noted, but in the absence of gross thrombus, endarterectomy was not feasible.

After weaning from cardiopulmonary bypass, the patient experienced progressive hypoxemia with saturation by pulse oximetry to the 70s and nadir measured PaO_2 of 57 mm Hg. The left lung was massively hyperinflated, while the right lung was collapsed and motionless. Bronchoscopy revealed blood-tinged frothy secretions originating mostly from the right side. Hypoxemia was refractory to recruitment maneuvers, adjustments in positive end expiratory pressure (PEEP), inhaled nitric oxide, suctioning, and lower inspiratory pressures.

In order to stabilize the patient until the initiation of venovenous ECMO, a trial of ILV was attempted so as to isolate and recruit the right lung without risking volutrauma to the left lung. A left DLT was placed over an airway exchange catheter, and this was unintentionally advanced into the right main bronchus. Correcting this was deemed unnecessary given good anatomic and functional lung isolation, albeit risking inadequate expansion of the right upper lobe. Volume control mode with a tidal volume (Vt) of 200 ml and PEEP 5 cm H_2O resulted in peak airway pressures ~50 cm H_2O for the right lung. Continuous positive airway pressure (5 cm H_2O) was applied separately to the left lung. This configuration resulted in rapid improvement of

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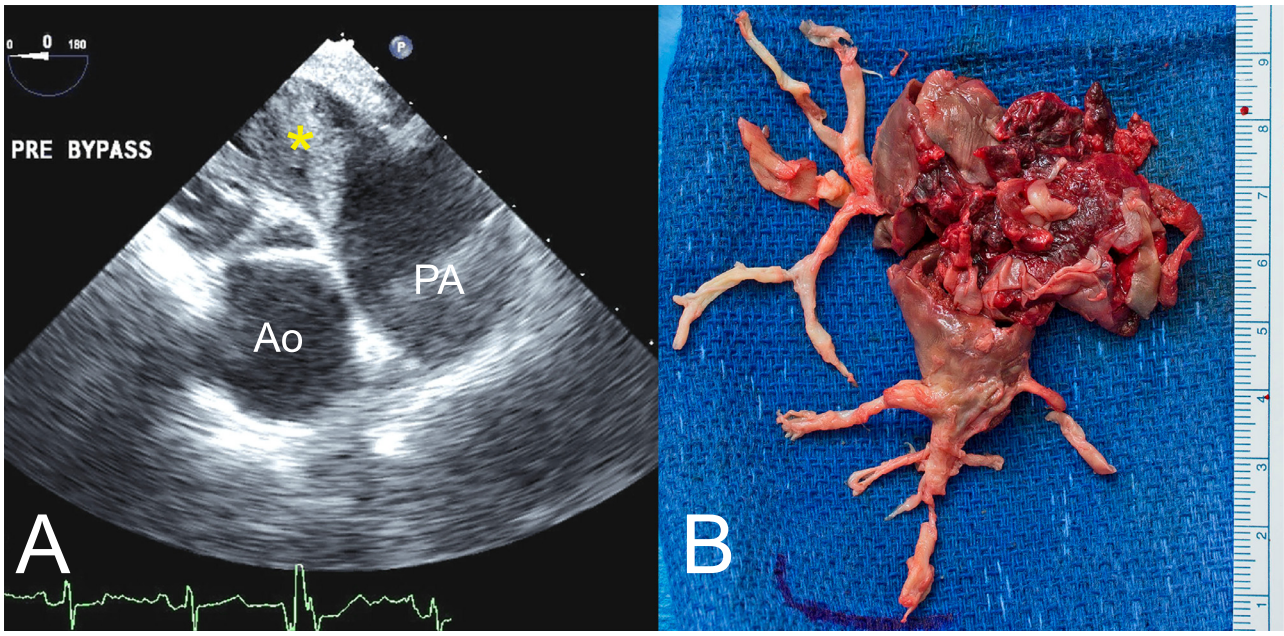


Figure 1. Intraoperative transesophageal echocardiogram showing almost complete occlusion of the right main pulmonary artery (A). Surgical specimen showing cast of pulmonary arterial tree (B). Aorta (Ao), pulmonary artery (PA), thrombus (*).

oxygenation (PaO_2 198 mm Hg). Low minute ventilation resulted in respiratory acidosis ($PaCO_2$ 75 mm Hg) but this was predicted to resolve with initiation of ventilation to the left lung. The chest was left open.

Upon arrival to the ICU institution of ILV was completed with use of 2 ventilators. The right lung was ventilated with a Vt of 250 mL, PEEP 20 cm H_2O, and resultant plateau pressure of 30 cm H_2O. Inhaled nitric oxide was continued to the left lung which was ventilated with a Vt of 200 ml and PEEP 5 cm H_2O. The ECMO team was on standby for several hours, but cannulation was deemed unnecessary after a period of stability. ILV was maintained for 24 hours after

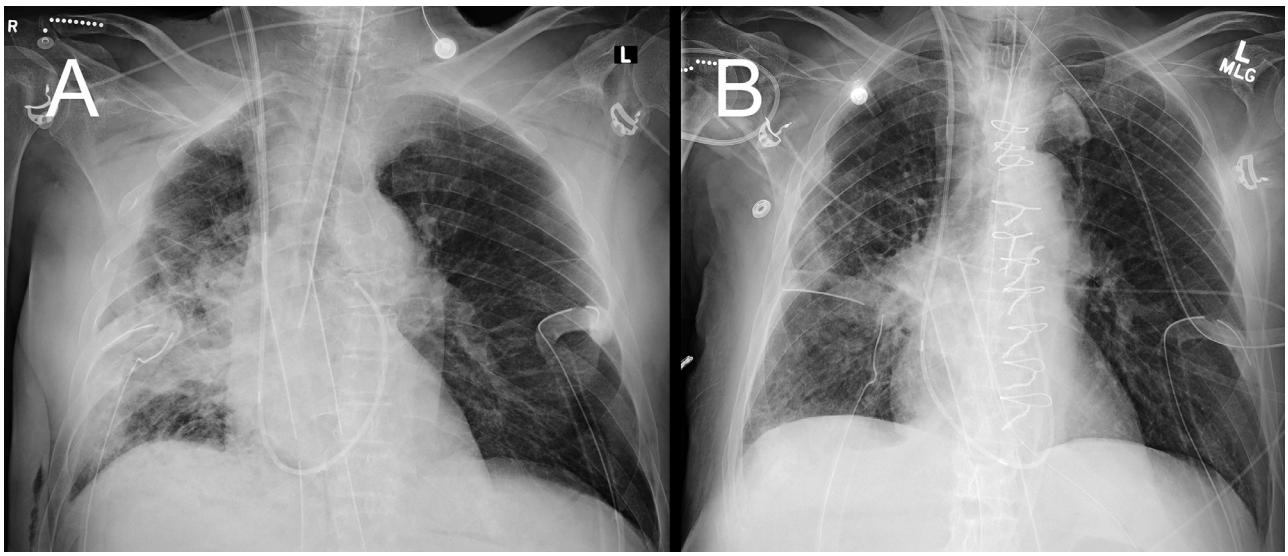


Figure 2. Evolution of right-sided reperfusion pulmonary edema from operative day with open chest (A) to postoperative day 2 with closed chest (B).

which there was significant improvement in hypoxemia and hypercarbia and this allowed for sternal closure. Resolution of RPE occurred within 2 days (Fig. 2), and the patient was discharged on hospital day 44.

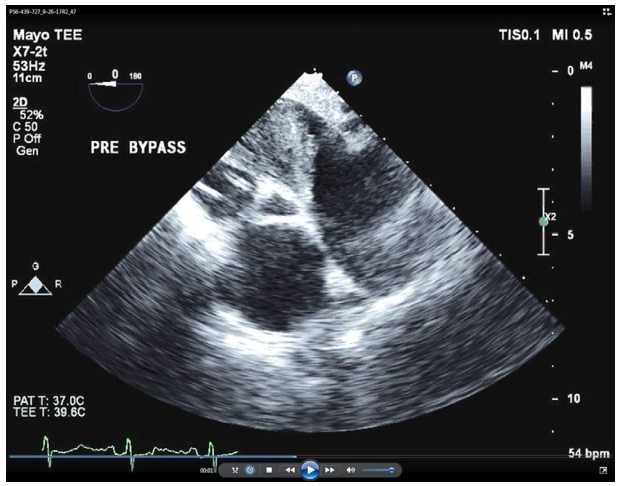
DISCUSSION

RPE is a known complication of PTE occurring in ~20% of cases. In this case, hypoxemia, frothy secretions, and interstitial infiltrates in the recently reperfused right lung were highly suggestive of the diagnosis. Hypoxemia refractory to mechanical ventilatory support occurs in ~1% of cases of RPE after PTE and is most commonly treated with venovenous ECMO.[4] Traditional mechanical ventilation fails to address unilateral RPE. The difference in compliance between lungs causes preferential distribution of Vt to the most compliant lung which may result in hyperinflation, increased capillary resistance, and diversion of perfusion to the noncompliant (less ventilated) lung, with overall worsening of ventilation perfusion (V/Q) mismatch.[2,3] Under anesthesia, V/Q can be worsened by attenuation of hypoxic vasoconstriction by inhaled anesthetics.

ILV using a low Vt and high PEEP on the lung with RPE and a low Vt with usual PEEP on the uninjured lung is a potential rescue strategy for hypoxemia in severe unilateral RPE. We hypothesize that selective delivery of high airway pressures and PEEP to the lung with RPE promoted alveolar recruitment and diverted perfusion toward the unaffected lung improving V/Q matching. A putative role exists for inhaled vasoconstrictor therapy to the reperfused lung to mimic hypoxic pulmonary vasoconstriction. DLT use does demand vigilance as far as monitoring cuff pressures to avoid mucosal ischemia and ensuring correct positioning of the endobronchial cuff. ILV is an alternative mode of rescue for asymmetric RPE that has not previously been reported after PTE.

SUPPLEMENTARY MATERIAL

The following is the supplementary data to this article:



Video 1. Intraoperative transesophageal echocardiogram showing almost complete occlusion of the right main pulmonary artery by thrombus.

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