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Management of Pulmonary Hemorrhage After Pulmonary Endarterectomy With Venovenous Extracorporeal Membrane Oxygenation Without Systemic Anticoagulation

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PULMONARY ENDARTERECTOMY (PEA) is the most appropriate treatment for chronic thromboembolic pulmonary hypertension (CTEPH) and can be curative. The success of PEA depends upon complete endarterectomy of the intima, chronic organized thromboembolic material, and part of the media of the affected pulmonary arteries.[1] Despite the technically challenging nature and the increasing frequency of this surgery, pulmonary hemorrhage remains a rare occurrence after PEA, with a reported incidence of 0.5% to 2%.[2,3] While the exact mechanism of pulmonary hemorrhage after PEA is often unknown, surgical trauma resulting in the disruption of the arterial adventitia and/or increased capillary permeability following restored perfusion as a form of severe acute lung injury are both plausible mechanisms.[4] The diagnosis of pulmonary hemorrhage usually is made at the conclusion of cardiopulmonary bypass (CPB), when ventilation and cardiac ejection have resumed. Initial management focuses on the maintenance of hemodynamics and gas exchange. Previously reported management options include the application of positive airway pressure, reversal of anticoagulation/coagulopathies, topical vasconstrictors, lung isolation, vascular balloon occlusion, embolization, and/or pulmonary resection;[2] however, pulmonary hemorrhage typically is managed initially with positive airway pressure, reversal of coagulopathies, and lung isolation.

For the past two decades, nonsurgical pulmonary hemorrhage has been managed with various forms of extracorporeal life support (ECLS), yet the use of ECLS in postsurgical pulmonary hemorrhage is a relatively recent development;[5–7] however, ECLS after PEA has been associated with significant morbidity/mortality and massive pulmonary hemorrhage in the setting of anticoagulation with heparin has been identified as a major etiology.[8] To the authors' knowledge, this is the first reported case of pulmonary hemorrhage after PEA being managed in the operating room and intensive care unit (ICU) with venovenous extracorporeal membrane oxygenation (vv-ECMO) after the intraoperative reversal of heparin and without postoperative systemic anticoagulation.

CASE PRESENTATION

A 44-year-old, 180-cm, 78-kg male was admitted from an outside hospital with a history of CTEPH and recent worsening of functional status. His medical history included multiple deep venous thromboses and pulmonary emboli despite anticoagulation, remote gastrointestinal bleeding, former 39-pack-a-year smoker, chronic obstructive pulmonary disease (COPD) requiring 3 L/min of home oxygen therapy via nasal cannulae, pulmonary hypertension, tricuspid regurgitation, and chronic kidney disease (baseline creatinine 1.5 mg/dL). Past surgical history was limited to a tonsillectomy. He initially was admitted to an outside hospital for a presumed COPD exacerbation. During that admission his functional status improved with diuresis (total weight loss approximately 12 kg) and he was diagnosed via transesophageal echocardiography (TEE) with right ventricular failure (Fig 1) and bilateral pulmonary emboli. Right-heart catheterization revealed a right ventricle (RV) pressure of 40/9 mmHg and pulmonary artery (PA) pressure of 50/30 mmHg (systemic pressure at assessment 101/60 mmHg). His preoperative medications included therapeutic enoxaparin, furosemide, aldactone, albuterol, tiotropium bromide, and fluticasone/salmeterol.

Initial examination of the patient identified bulging neck veins, jugular venous distention to the mandible, crackles on auscultation at the bases, a 3/6 holosystolic murmur at the left lower sternal border, and trace lower extremity edema. Transthoracic echocardiography (TTE) identified a severely enlarged RV with depressed systolic function, paradoxical ventricular septal motion, severe tricuspid regurgitation, and moderate pulmonary hypertension. Lung ventilation/perfusion scans identified multiple segmental perfusion/ventilation defects throughout both lungs consistent with bilateral pulmonary thromboembolic disease, which was confirmed on pulmonary angiography. After an inferior vena cava (IVC) filter was inserted, left-heart catheterization identified a left ventricle (LV) pressure of 101/7 mmHg and no evidence of obstructive coronary artery disease. He then was scheduled for PEA.

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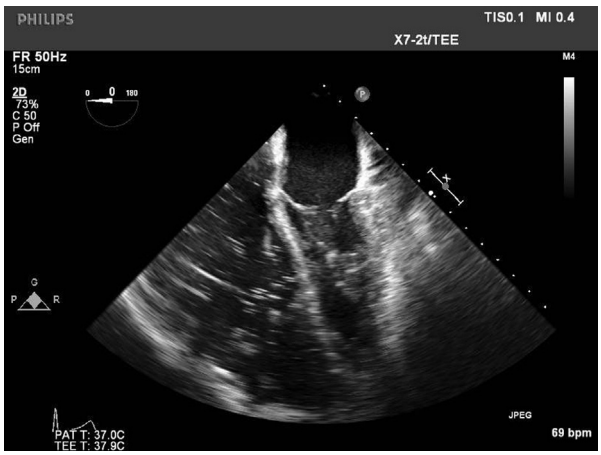

Fig 1. Transesophageal echocardiography, mid-esophageal four-chamber still image demonstrating right ventricular (RV) dilation and paradoxical ventricular septal motion characteristic of RV failure. Artifact from intravenous injection is seen in the RV.

After stable intravenous induction with midazolam (2 mg)/fentanyl (500 μg)/etomidate (20 mg)/rocuronium (100 mg) and initiation of CPB, PEA was completed with one 20-minute period of deep hypothermic circulatory arrest (DHCA) per side and a tricuspid valve repair utilizing a 34-mm annuloplasty ring was performed during rewarming.[9] Upon initial separation from CPB with intravenous dopamine (5 μg/kg/min), blood was noted in the endotracheal tube (ETT). Therefore, CPB was resumed and the PA vent was placed back on suction. Initial fiberoptic bronchoscopy (FOB) identified a frothy, bright red bleed originating from the right lower lobe. After the 8 mm Mallinckrodt ETT (Covidien, Mansfield, MA) was exchanged for a 9 mm ETT, a 9-French Uniblocker (LMA North America, San Diego, CA) was inserted into the right bronchus intermedius; however, when the PA vent suction was decreased and the heart allowed to eject, additional bleeding was noted from the right upper lobe. Therefore, the bronchial blocker was withdrawn into the right mainstem bronchus (Fig 2). After proper lung isolation was confirmed and therapeutic bronchoscopy was completed to facilitate gas exchange in the patient's patent left lung, separation from CPB once again was attempted. This was accomplished with intravenous epinephrine (0.1 μg/kg/min), intravenous dopamine (5 μg/kg/min), and hyperventilation (volume control mode, respiratory rate [RR] 15 breaths/min, tidal volume [TV] 500 mL, F_IO_2 100%, and positive end-expiratory pressure [PEEP] 15 cmH_2O). Heparin was reversed with intravenous protamine (350 mg) and coagulopathies were corrected with two units of fresh frozen plasma (FFP) and two units of platelets (PLT). A post-protamine activated coagulation time (ACT) of 131 seconds (baseline 125 seconds) was obtained and a normal thromboelastogram (TEG) confirmed the correction of coagulopathies. Three units of packed red blood cells (pRBC) were administered concurrently with the FFP based on the hematocrit and the need for volume expansion.

The patient's oxygenation/ventilation progressively deteriorated over the next 30 minutes during chest closure. Despite aggressive ventilation settings (pressure control mode, RR 16 breaths/min, TV 450 mL, F_IO_2 100%, PEEP 20 cmH_2O), oxygenation/ventilation on one lung were inadequate after the chest was closed and resulted in an arterial pH of 7.0, pCO_2 >90 mmHg, pO_2 49 mmHg, HCO3 23 mmol/L, base deficit -6 mmol/L, and SpO_2 84%. Inadequate oxygenation/ventilation resulted in cardiac arrest that required two doses of intravenous epinephrine (1 mg), one ampule of intravenous sodium bicarbonate, and one minute of closed-chest compressions to restore the systemic blood pressure to 130/60 mmHg. Given that gas exchange was thought to be the principal problem and lung isolation still was required, the decision was made to initiate vv-ECMO. All infusions were transferred from the right internal jugular (RIJ) pulmonary artery catheter (PAC) to an existing left subclavian vein triple-lumen catheter. After the PAC was removed, a wire was passed through the existing 9-French introducer and the introducer subsequently was removed. With the wire in the RIJ and the distal end visualized in the IVC via TEE, a 27-French Avalon Elite Bi-Caval Dual-Lumen Catheter (Avalon Laboratories, Rancho Dominguez, CA) was inserted following serial dilations (Fig 3).[10–12] Given the concern for continued pulmonary hemorrhage, vv-ECMO at a flow of 3.5 L/min was instituted via the Avalon Elite without systemic anticoagulation. Once arterial blood gases confirmed a trend toward normalization, the patient was transferred to the ICU on infusions of epinephrine (0.15 μg/kg/min), dopamine (5 μg/kg/min), and propofol (35 μg/kg/min) with stable vital signs. Arterial blood gas analysis obtained prior to leaving the operating room revealed a pH of 7.29, pCO_2 37 mmHg, pO_2 95 mmHg, HCO3 18 mmol/L, base deficit -8.5 mmol/L, and SpO2 97% on a F_IO_2 1.0.

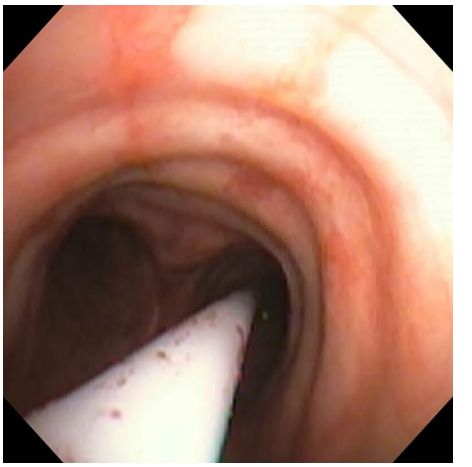

Fig 2. 9-French Uniblocker bronchial blocker (deflated), positioned in the right mainstem bronchus.

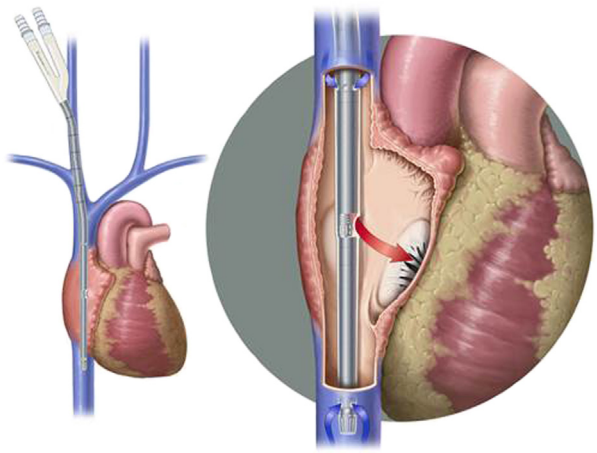

Fig 3. Artist representation of the Avalon Elite Bi-Caval Dual-Lumen Catheter in situ, inflow cannulae located in the superior/inferior vena cava and outflow is directed toward the tricuspid valve. Copied from Mazzeffi M: Transesophageal echocardiographic guided placement of a right internal jugular dual-lumen venovenous extracorporeal membrane oxygenation (ECMO) catheter. *J Cardiothorac Vasc Anesth* 27:4:e46-47, 2013.