

Acquired tracheomalacia due to aortic aneurysm managed with venopulmonary extracorporeal membrane oxygenation for perioperative support

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ABSTRACT

Extracorporeal membrane oxygenation (ECMO) has diverse applications. In the present report, we have described a case of tracheomalacia from a thoracic aortic aneurysm causing respiratory failure. Total arch replacement with reverse frozen elephant trunk grafting was performed. Perioperative ECMO support was accomplished with venopulmonary artery ECMO. This strategy allowed for preoperative oxygenation support, venous drainage during cardiopulmonary bypass, and postoperative support without cannula exchanges. Our patient required ECMO support for 12 days postoperatively. We have illustrated a unique case of acquired tracheomalacia but also an ECMO cannulation strategy allowing for preoperative oxygenation, seamless transition to cardiopulmonary bypass, and postoperative support. (*J Vasc Surg Cases Innov Tech* 2021;7:737-40.)

Keywords: Extracorporeal membrane oxygenation; Tracheomalacia; Veno-venous

Extracorporeal membrane oxygenation (ECMO) has demonstrated benefit for patients with cardiac and/or pulmonary failure.¹ In those with respiratory failure, ECMO supports oxygenation until respiratory function has recovered.² The role of ECMO has since expanded to a variety of other indications, including airway surgery and obstruction.³ However, ECMO is also associated with complications, including Harlequin syndrome, a differential oxygenation between the upper and lower body regions as a result of competing left ventricular function in venoarterial (V-A) ECMO.⁴ Nonetheless, ECMO is effective in providing support and its role has been expanding within the perioperative setting. In the present report, we have described the repair of a chronic thoracic aortic dissection causing acquired tracheomalacia, airway obstruction, and respiratory failure using the TandemLife Protek Duo (TPD; LivaNova, London, UK) as perioperative ECMO support. Our patient provided written informed consent for the report of her case and imaging studies.

CASE REPORT

A 61-year-old woman with a history of asthma, chronic obstructive pulmonary disease, mild pulmonary hypertension, and mechanical aortic valve replacement, patent foramen ovale closure, and Maze procedure had developed an acute type A aortic dissection requiring redo sternotomy and ascending aorta replacement. She had subsequently developed aneurysmal degeneration of her aortic arch and descending aorta in the succeeding 9 years, prompting a staged total aortic arch replacement. She had initially presented to our institution for left carotid artery to subclavian artery bypass as the first stage of her repair. However, the procedure was aborted owing to severe bronchospasms on induction that were unresponsive to medical management. She required endotracheal intubation and medical bronchospasm therapy. Two months later, she returned for the surgery. After anesthesia induction, endotracheal intubation was successful but difficultly with oxygenation and ventilation ensued. The peak airway pressure was 40 mm Hg, the tidal volume was 20 mL, with minimal end-tidal carbon dioxide, no breath sounds, and diffuse wheezing. Because of hypotension and bradycardia, bifemoral V-A ECMO was established. Cerebral near-infrared spectrometry revealed a venous oxygen saturation of 30%. The patient had blue facies suggestive of Harlequin syndrome. Repeated attempts at mechanical ventilation were unsuccessful, and hand ventilation became increasingly difficult. Bedside bronchoscopy demonstrated complete collapse of her airway, raising concern for acquired tracheomalacia in the setting of chronic tracheal compression. The results from a retrospective review of the preoperative imaging studies (Fig 1) were consistent with this finding. Given the inability to provide adequate oxygenation via mechanical ventilation coupled with normal cardiac function, we suspected that deoxygenated blood was competing with the peripheral V-A ECMO arterial perfusion resulting in Harlequin syndrome. Thus, we decided to convert from V-A ECMO to venopulmonary artery (V-Pa)

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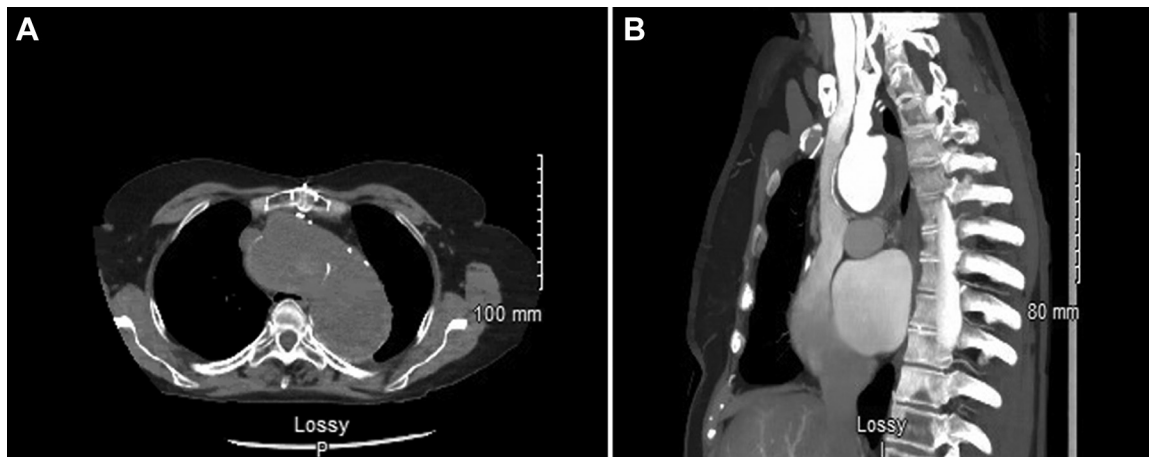


Fig 1. Preoperative cross-sectional computed tomography images depicting the ascending aortic aneurysm with significant compression of the proximal trachea due to a local mass effect. **A,** Axial view. **B,** Sagittal view.

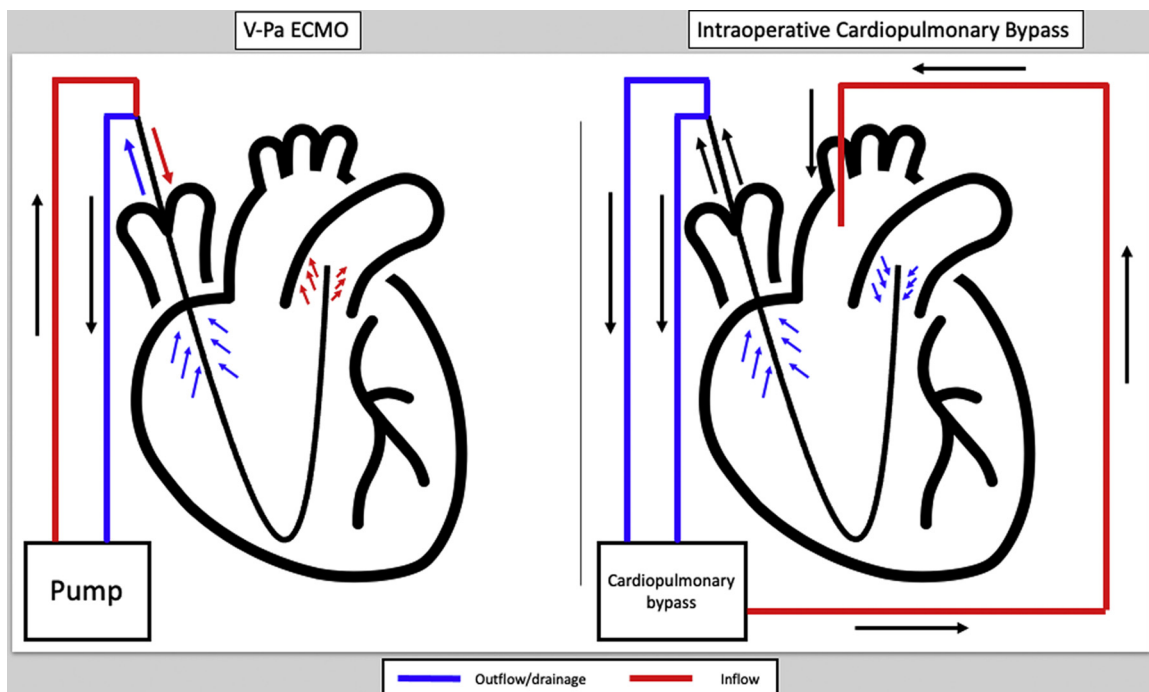


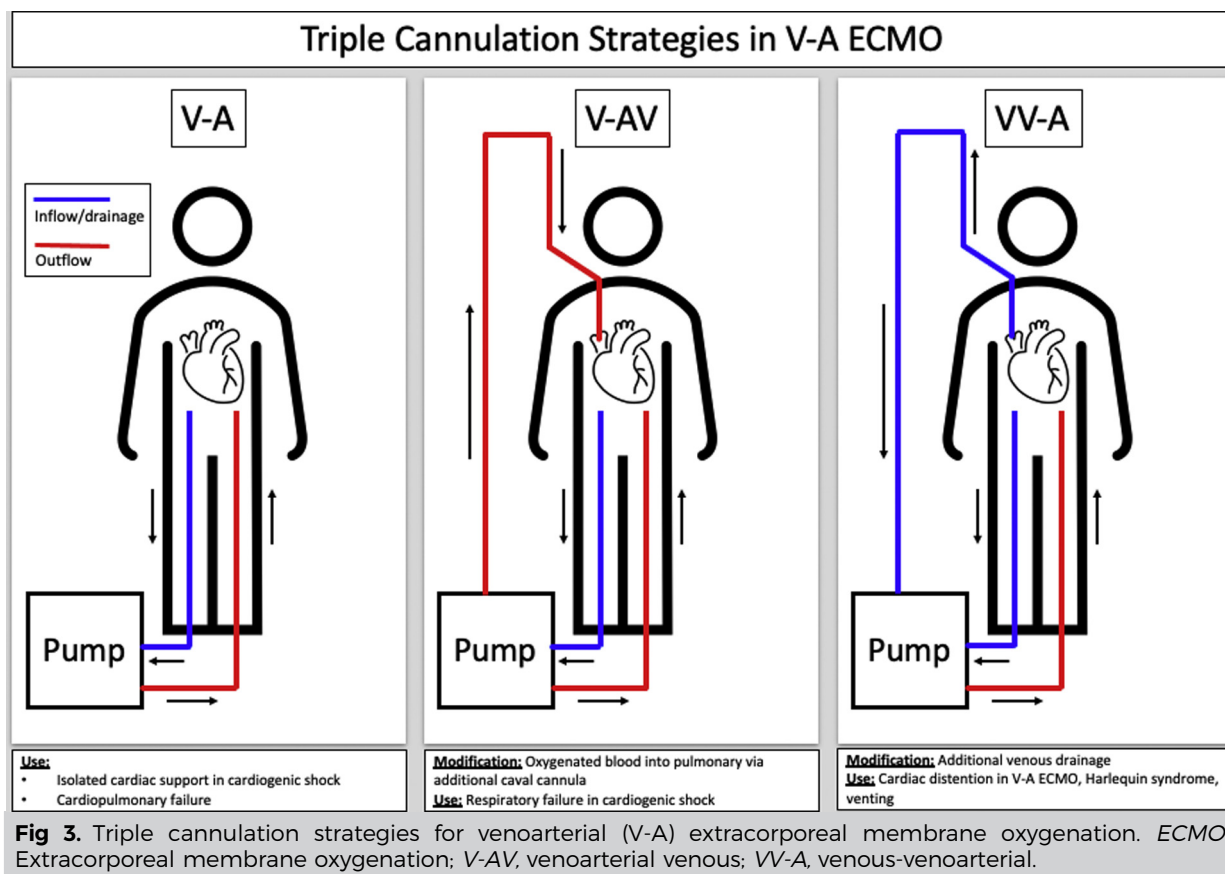
Fig 2. Diagram of the venopulmonary artery (V-Pa) extracorporeal membrane oxygenation (ECMO) with transition to intraoperative cardiopulmonary bypass.

ECMO to provide oxygenation with central outflow, improve venous drainage, and unload the right ventricle (RV), given the patient's likelihood of RV dysfunction in the setting of respiratory failure with comorbid pulmonary hypertension. She was converted to V-Pa ECMO cannulation with a 31F TPD cannula via the right internal jugular vein (IJV; Fig 2). Both femoral cannulas were removed.

The V-Pa ECMO settings were 3300 rpm, flow of 3.3 L/min, sweep gas of 2 L/min, and inspired oxygen fraction at 100%, which provided satisfactory oxygenation without mechanical ventilation. Rigid bronchoscopy was performed, demonstrating near complete tracheal obstruction from 2 cm below the vocal

cords to the carina due to external compression from the aortic aneurysm. Tracheal stenting was attempted but was unsuccessful owing to collapse of the deployed silicone stents. Further attempts at establishing tracheal patency were aborted, and the patient was intubated with an endotracheal tube positioned in the right mainstem bronchus. Definitive repair was planned for the next day to address the aneurysm causing tracheal obstruction.

The next day, total arch replacement with reverse frozen elephant trunk grafting was performed. The prior aortic graft was cannulated, and both lumens of the TPD were used as venous outflow connected to the cardiopulmonary bypass



(CPB) circuit (Fig 2). Cold blood del Nido cardioplegia was used, and the patient was cooled to 18°C for deep hypothermic circulatory arrest with antegrade cerebral perfusion via the innominate and left carotid arteries. A Sienna multibranch collared graft (Vascutek, Ltd, Inchinnan, UK) was used for her repair. Once the patient had reached 36.5°C on rewarming, she was transitioned back to V-Pa ECMO using the TPD cannula, and the aorta was decannulated. She required no vasopressor or inotropic support coming off CPB. The total CPB time was 304 minutes, and the total cross-clamp time was 138 minutes. Postoperative V-Pa ECMO settings were 3000 rpm, flow of 3.0 L/min, sweep gas of 2 L/min, and inspired oxygen fraction at 100% with 0.02 µg/kg/min of norepinephrine.

She was extubated on postoperative day 2 and underwent repeat tracheal stenting on postoperative day 8 owing to persistent tracheal obstruction despite aneurysm repair due to her acquired tracheomalacia. She did not tolerate further attempts at weaning from ECMO support and was reintubated on postoperative day 9. A tracheostomy was performed on postoperative day 11. She was decannulated from V-Pa ECMO on postoperative day 12 but continued to require mechanical ventilation. She was discharged to a long-term care facility on postoperative day 30.

DISCUSSION

Acquired tracheomalacia secondary to chronic aortic compression is rare, with one case report previously

describing a patient with syphilitic aneurysm⁵ and no reports of ECMO. The use of ECMO for inadequate cardiopulmonary function has been well-documented to be lifesaving for appropriately selected patients.¹ However, ECMO has been associated with potential complications.⁴ The use of TPD as a V-Pa ECMO strategy might help alleviate these complications.

The TPD is a dual-lumen omnidirectional cannula that is inserted into the main pulmonary artery via the right IJV.^{6,7} It was originally designed as a venovenous (V-V) ECMO cannula. This was achieved through an inflow cannula in the right atrium and the outflow cannula in the pulmonary artery.^{6,7} The in-line oxygenator allows the circuit to assume complete and centralized oxygenation support.⁸ The TPD has the ability to provide right heart support as a temporary right ventricular assist device in those patients with RV failure, either isolated or combined with respiratory failure.⁶ The TPD has the added benefit of central insertion into the IJV, improving patient mobility and decreasing the risk of femoral access complications compared with peripheral ECMO.⁶ Additionally, its location past the right heart decreases the potential for RV distension and recirculation of oxygenated blood seen in high-flow V-V ECMO circuits, which results in decreased oxygen delivery. Alternative cannulation strategies such as triple cannulation are available to

address inadequate oxygenation in patients supported with V-A ECMO (Fig 3). These strategies improve venous drainage (ie, venous-venoarterial) or increase oxygenated outflow into the pulmonary circulation (ie, venoarterial venous). For our patient, V-Pa was preferred over triple cannulation owing to our institution's extensive experience with the TPD cannula for V-V ECMO, the patient's risk of RV dysfunction with concomitant respiratory failure because of her history of pulmonary hypertension, and a desire to limit the number of peripheral cannulation sites owing to their propensity for complications.⁹

We have reported a case of acquired tracheomalacia from external compression of the trachea requiring ECMO. We used the TPD as V-Pa ECMO for prolonged respiratory support. In our experience of nearly 200 cases, we have found several advantages for V-Pa ECMO compared with V-V ECMO. These include the ability to provide RV support, the ease of transition to CPB, the reestablishment of ECMO postoperatively, the ease of insertion, improved patient ambulation, and a decreased risk of limb ischemia. Versatile and durable cannulation strategies should be considered for patients requiring ECMO support with a need for concomitant or multiple operative procedures and might be beneficial in avoiding the complications associated with extended periods of peripheral ECMO cannulation.

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