Surgical right pulmonary artery to left atrial shunt for severe pulmonary hypertension and bridge to lung transplantation

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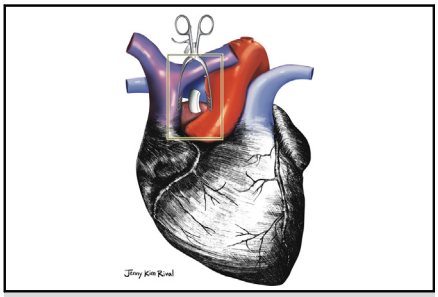

Surgical right pulmonary artery to left atrial shunt using 10-mm woven graft.

CENTRAL MESSAGE

We describe the placement of a surgical shunt from the pulmonary artery to the left atrium to alleviate severe pulmonary hypertension with right ventricular failure and bridge to lung transplantation.

Despite significant advancements in medical management for pulmonary hypertension, 5-year survival is approximately 38.1%.[1] Current guidelines recommend lung transplantation for patients with rapidly progressive pulmonary hypertension and inadequate response to medical management.[2] Bridging to transplant with extracorporeal membrane oxygenation (ECMO) may be required when there is worsening of cardiopulmonary status.

Here, we report a case of a 38-year-old female patient with a medical history of atrial septal defect (ASD) who, over the course of her pregnancy, developed exercise intolerance and cyanosis. This case report was exempt from the Baylor College of Medicine institutional review board review; the patient provided written informed consent to allow the case to be reported. After delivery, she developed right ventricular (RV) failure requiring venoarterial (VA) ECMO. She was transferred to our hospital 10 days' postpartum with femoral VA-ECMO with an ASD occluder that had been placed for persistent hypoxia. She had suprasystemic pulmonary hypertension that was thought to be due to Eisenmenger syndrome resulting from the unclosed ASD. The patient was unable to be weaned from ECMO and required a bridging strategy to bilateral lung transplant versus heart–lung transplant. Ambulatory VA-ECMO strategies were not ideal, given her small peripheral vasculature. Veno-venous (VV) ECMO with balloon atrial septostomy was also not possible due to the ASD occluder.

Transplant work-up was initiated, and the patient was found to have elevated panel-reactive antibodies (58%). Due to the sensitization, she required improved mobility to tolerate an anticipated prolonged period of waiting for transplant. We devised a strategy based on the description of Khouqeer,[3] whereby a shunt was created between the pulmonary artery and dome of the left atrium for right ventricular decompression. For additional information, see the Appendix E1.

TECHNIQUE

A median sternotomy was performed. Following systemic heparinization, cardiopulmonary bypass was initiated through existing femoral venous and arterial cannulas. Cardioplegic arrest was attained using del Nido solution. The right main pulmonary artery was mobilized from the underside of the ascending aorta. A 1.5-cm atriotomy was created at the dome of the left atrium, and a mirror incision was made on the right main pulmonary artery. A 10-mm woven graft approximately 3 cm in length was then anastomosed to the left atrium and right pulmonary artery with running sutures (Figure 1).

Following resumption of cardiac function and initiation of inotropic support, the patient was weaned to partial flows on cardiopulmonary bypass to maintain oxygen saturations. A 28-French bicaval dual-lumen cannula was then inserted percutaneously through the right internal jugular vein for VV-ECMO. With the VV-ECMO in place and flowing, we

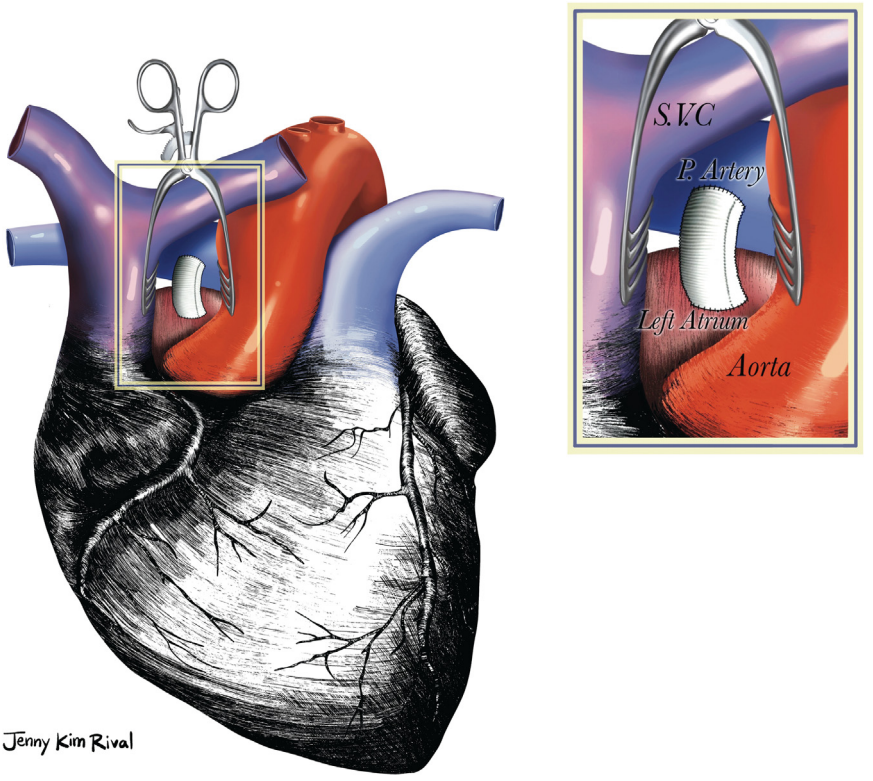

FIGURE 1. Surgical right pulmonary artery-to-left atrial shunt using 10-mm woven graft. Illustration by Jenny K. Rival. *SVC*, Superior vena cava.

were able to fully separate from cardiopulmonary bypass with sufficient arterial oxygenation saturations. The sternum was then reclosed in standard fashion and the femoral vessels were repaired following decannulation. Anticoagulation was initiated with bivalirudin on postoperative day 3.

Subsequent transthoracic echocardiography showed improvement in RV function as well as reduction of RV dilation and persistent flow through the created shunt (Figure 2). Right heart catheterization demonstrated a cardiac index of 3.6 L/min/m^2 without inotropic support.

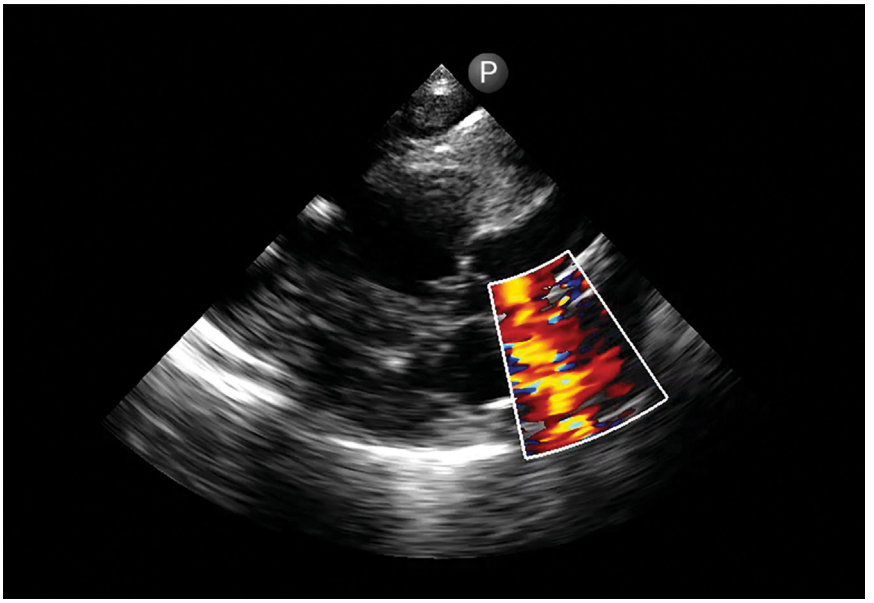

FIGURE 2. Transthoracic echocardiography demonstrated right-to-left shunt flow from the right pulmonary artery to the left atrium.

Unfortunately, the patient did not tolerate weaning of VV-ECMO due to hypoxia. The patient was ultimately listed for a bilateral lung transplant, given the improvement of RV function. After 59 days from shunt creation, a bilateral lung transplant was performed via sequential thoracotomies. Due to concerns for clot formation in the shunt, we ligated the shunt with titanium clips. VV-ECMO was decannulated the following day. The patient was discharged home 2 months after transplant and reports good quality of life on subsequent follow-up.

DISCUSSION

Our approach was invasive but achieved important objectives. First, it provided an effective method to reduce the RV strain and improve RV performance, thereby avoiding the need for combined heart–lung transplant. Second, it allowed us to bridge the patient to bilateral lung transplant with VV-ECMO through the right internal jugular vein, a configuration that facilitates ambulation and physical therapy.[4]

In this case, pulmonary pressures were reduced from 95/35 mm Hg preshunt to 57/13 mm Hg pretransplant. Conceptually, right-to-left shunting distal to the RV should decrease systolic and diastolic pulmonary pressures and reduce RV work more effectively than shunting at the atrial level. No deleterious effects were noted due to the shunt in regard to atrial dilatation or arrythmias. Alternative strategies include a reversed Potts shunt; however, this would not have provided the same extent of RV unloading. In the future, use of a ringed Gore-Tex graft would be a better conduit for shunt creation, as it would have resisted compression and adhesion formation better than a woven graft. Future studies are needed to study the effectiveness of this strategy in patients with severe pulmonary hypertension.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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APPENDIX E1

Detailed Patient History

Our patient was a 38-year-old female gravida 1, para 1, with a history of atrial septal defect (ASD) since childhood. She was told she had a murmur throughout childhood but never had any exercise limitation (she was on the school swim team and very active growing up). At some point, she was told the ASD had "closed" spontaneously in her early 30s. During her pregnancy, she had extreme fatigue, breathlessness with minimal exertion or speaking, and cyanotic fingertips with exertion, which improved with rest.

No other medical history or surgical history was noted. Medication use included an oral contraceptive. She denied tobacco use or significant alcohol intake.

She was admitted in labor and had a vaginal delivery of a healthy baby girl, shortly after which (within 15 minutes), the patient went into acute hypoxic respiratory failure requiring emergent intubation. Despite positive end-expiratory pressure 18 and fraction of inspired oxygen 100%, she remained hypoxic and required inhaled nitric oxide at 40 ppm. Stat transthoracic echocardiogram demonstrated 13-mm ASD with right-to-left shunting, dilated right ventricle, and pulmonary artery. Computed tomography for pulmonary embolism was negative for pulmonary embolism. During this time, she was in profound metabolic acidosis and shock on escalating vasoactive agents. The decision was made to cannulate with peripheral venoarterial extracorporeal membrane oxygenation. Five days later, her ASD was repaired via percutaneous closure due to persisting hypoxia. Right heart catheterization at that time showed pulmonary capillary wedge pressure 7, pulmonary artery (PA) pressure 66/23, right ventricle 55-0-4, right atrium 2, cardiac output 3.4, cardiac index 2.5.

On arrival at our institution, venoarterial extracorporeal membrane oxygenation was as follows: sweep 0.5, flow 3.48, speed 3250 rpm. PA pressure 100/39, central venous pressure 13, cardiac output 3.7, cardiac index 2.7. Infusions: milrinone + norepinephrine + heparin + inhaled nitric oxide 40 PPM. Transthoracic echo: All left ventricular (LV) segments contracted normally. LV ejection fraction was normal (>60%). Small LV cavity. Interventricular septum was flattened in systole c/w severely elevated right ventricular systolic pressure. Right ventricular chamber size was severely enlarged. Global right ventricular systolic function was depressed. Tricuspid annular plane systolic excursion was 0.8 cm. Moderate-to-severe tricuspid regurgitation was observed. Estimated peak systolic PA pressure was 120 to 125 mm Hg (severe pulmonary hypertension). The estimated right atrium pressure by inferior vena cava dynamics was >20 mm Hg.