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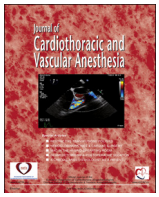

Case Conference

Systolic Anterior Motion of the Mitral Valve With Left Ventricular Outflow Tract Obstruction: A Rare Cause of Hypotension After Lung Transplantation

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Key Words: mitral valve; systolic anterior motion; lung transplantation; hemodynamic instability; left ventricular outflow tract obstruction

BILATERAL LUNG TRANSPLANTATION (BLT) is one of the most complex procedures anesthesiologists face. In fact, in certain populations early (1-month) mortality is greater than 20%.[1] The patient's baseline respiratory compromise makes ventilation and oxygenation challenging, especially with the need for one-lung ventilation. Furthermore, patients are prone to hemodynamic instability from both their underlying cardiopulmonary disease and the surgical manipulation that occurs throughout the procedure. Critical decision-making, such as the need for cardiopulmonary bypass (CPB), must be made jointly by the surgical and anesthetic teams. Even after transplantation is complete, hypotension frequently is encountered. In this case conference, the common causes of hemodynamic instability during lung transplantation, and the relevant transesophageal echocardiographic (TEE) findings during BLT are reviewed. In addition, a rare cause of hypotension that was encountered in a patient due to systolic anterior motion (SAM) of the mitral valve with left ventricular outflow tract (LVOT) obstruction is examined.

Case Report[*]

A 63-year-old female (height 160 cm, weight 57 kg) with a history of interstitial lung disease secondary to interstitial pneumonia with autoimmune features (high Sjögren's-syndrome-related antigen A) and secondary pulmonary hypertension presented to the authors' institution for BLT. At baseline, she required 2 L/min of oxygen per nasal cannula, which increased to 4-to-6 L/min with activity. She had a medical history significant for hypertension, Sjögren syndrome, and numerous antibodies, which made finding a suitable organ difficult (transplantation list time was 4.5 years). Her surgical history was significant for a right video-assisted thoracoscopic surgery 10 years prior for exploration and lung biopsy.

She underwent cardiac catheterization 6 months before transplantation, for which pulmonary artery pressure of 40/15 mmHg, mean pulmonary artery pressure of 21 mmHg, and pulmonary vascular resistance (PVR) of 1.77 Wood units were noted. She completed a 6-minute walk test (on 4 L/min of oxygen), with a distance of 822 feet and oxygen saturation nadir of 87% (baseline 95%). Pulmonary function testing performed 7 weeks before transplantation revealed a total lung capacity of 2.19 L (47% predicted), forced vital capacity of 0.89 L (31% predicted), and a first second of forced exhaled volume of 0.76 L (36% predicted).

Her medications at the time of surgery included atovaquone (1,500 mg daily); triamterene-hydrochlorothiazide (37.5/25 mg daily); and cholecalciferol. On the day of surgery, the patient noted a recent upper respiratory infection and complained of rhinorrhea, a cough, and shortness of breath at rest. Physical examination was significant for nasal congestion yet clear lung auscultation bilaterally. Laboratory results revealed a sodium of 136 mmol/L, a potassium of 2.8 mmol/L, a creatinine of 0.7 mg/dL, a hemoglobin of 16.5 g/dL, a platelet count of

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397,000/uL, and an international normalized ratio of 1.0. The most recent transthoracic echocardiogram (TTE) had been performed 8 months prior and showed a flattened ventricular septum consistent with right ventricular (RV) pressure overload (at least 65 mmHg), a mildly dilated RV, mild-to-moderately reduced RV systolic performance, mild left ventricular (LV) diastolic dysfunction without LV hypertrophy or dilation, and an LV ejection fraction (LVEF) of 58%. Mild mitral regurgitation (MR) was present without SAM of the mitral valve. Due to the patient's significant sensitization and high (97%) panel of reactive antibodies, she underwent plasmapheresis in the intensive care unit (ICU) before proceeding to the operating room.

In the operating room, after intravenous access, a right brachial arterial line (attempts at radial access were unsuccessful) was inserted and was followed by an uneventful intravenous induction of general anesthesia with 6 mg of midazolam, 500 μg of fentanyl, and 100 mg of succinylcholine. A left-sided, 37-Fr, double-lumen endotracheal tube was inserted after 2 attempts, 1 by a resident and 1 by the attending anesthesiologist (note: a grade-1 view was obtained, yet there was initial difficulty passing the tube into the trachea). Before central venous catheter and pulmonary artery catheter insertion, a TEE probe was inserted for ultrasound guidance of the catheter insertions. A 9-Fr, double-lumen introducer was inserted into the left subclavian vein, into which a continuous cardiac output pulmonary artery catheter was floated. The left subclavian site was chosen to allow for venovenous extracorporeal membrane oxygenation (V-V ECMO) cannulation (via an Avalon Elite catheter; Maquet, Rastatt, Germany) into the right internal jugular vein, as requested by the surgeon. A complete TEE examination then was performed on which a mildly dilated RV with mildly reduced function, no septal flattening or hypertrophy, a low-normal LVEF, and mild MR without SAM of the mitral valve were noted.

Before incision, the patient became hypotensive, and a vasopressin infusion was started. A 20-Fr Avalon Elite cannula was inserted electively by the surgeon, and V-V ECMO was initiated. After bilateral thoracotomies and transverse sternotomy (clamshell incision), ventilation to the right lung was stopped. After clamping the right main pulmonary artery, the patient experienced hypotension, with mean arterial pressure

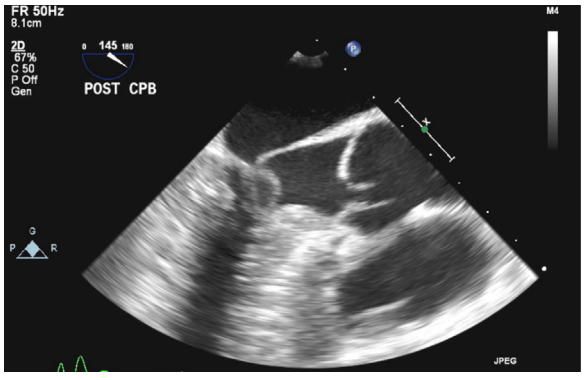

Fig 1. Postoperative 2D midesophageal long-axis view revealing SAM of the mitral valve.

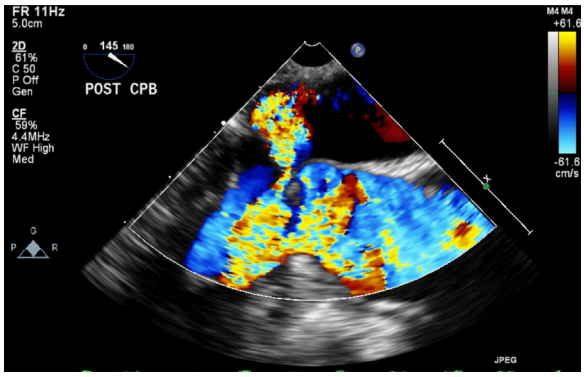

Fig 2. Postoperative 2D midesophageal long-axis color view revealing SAM of the mitral valve. Note the new mitral regurgitation and turbulent flow through the LVOT.

(MAP) around 40 mmHg. Thus, an epinephrine infusion was initiated and a 500 mL bolus of normal saline was administered. Hypotension continued despite these interventions, so epinephrine bolus doses were administered as needed. TEE at this time showed no changes: RV function was maintained, LV end-diastolic volume was adequate, LVEF was low-normal, and SAM of the mitral valve still was not noted. ST depressions then were observed with the continued vasoplegia despite the administration of vasopressin and epinephrine. Hydrocortisone also was given per the surgeon's request to rule out possible adrenal insufficiency. In addition, the intravenous immunoglobulin therapy infusion (started before surgery) was stopped at the surgeon's request and the epinephrine infusion was switched to norepinephrine due to a heart rate increase >100 beats/min. It should be noted that from a surgical standpoint, the right lung resection, right donor lung implantation, and right donor lung reperfusion were without issue.

Hemodynamics improved temporarily after reperfusion of the right lung, but there was significant hypotension as dissection of the left lung began. The decision then was made to use "standard" CPB to facilitate left pneumonectomy and implantation. Initiation of CPB was via the right atrium/ascending aorta and was uncomplicated, and the left donor lung was anastomosed successfully. After discontinuation of CPB (124 min), the patient again became hypotensive, with MAP around 50 mmHg and tachycardia despite a vasopressin infusion. TEE examination at this time revealed a mildly underfilled, hyperdynamic LV with minimal intracardiac air and no signs of anastomotic or RV failure. The hypotension worsened when sinus tachycardia converted to a supraventricular tachycardia (SVT) and the heart rate increased to 140 beats/min. TEE then revealed SAM of the mitral valve, with LVOT obstruction seen on both 2-dimensional (2D) (Fig 1) and color imaging (turbulent flow was noted) (Fig 2). Moreover, continuous-wave Doppler across the LVOT showed a late-peaking, dagger-like waveform. Norepinephrine and calcium chloride were used to temporarily increase blood pressure, magnesium was administered to combat the SVT, and blood products were given for volume resuscitation. Her blood pressure eventually stabilized (MAP around 60 mmHg)

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