Dynamic Left Ventricular Outflow Tract Obstruction Complicating Bilateral Lung Transplantation

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Dynamic left ventricular outflow tract obstruction (DLVOTO) has been observed in a variety of clinical circumstances but not previously reported in the setting of orthotopic lung transplantation. Diagnosis and effective management of this adverse event were facilitated by transesophageal echocardiography (TEE).

Case Report

A 57-yr-old female underwent sequential bilateral lung transplantation for intractable respiratory failure resulting from idiopathic pulmonary fibrosis. Her medical history was significant for polymyositis and hypertension that were treated with prednisone 10 mg daily and enalapril 20 mg twice daily. Her pulmonary hemodynamic status was essentially normal preoperatively with pulmonary artery pressures (PAP) of 23/10 mm Hg (mean, 15 mm Hg) and pulmonary vascular resistance of 2.2 mm Wood units (normal range, 1.5–2.5).

Anesthesia was induced and a double-lumen endobronchial tube was placed for lung isolation. The intent was to perform the procedure without the aid of cardiopulmonary bypass (CPB), and the patient tolerated deflation of the right lung with minor hypotension responding to norepinephrine. Subsequent deflation of the left lung resulted in an increase of the PAP and increasing central venous pressure (Table 1). When the right lung was again deflated to begin implantation, profound pulmonary hypertension resulted and norepinephrine and nitric oxide (NO) were commenced (Table 1). Clamping of the right main pulmonary artery resulted in severe systemic hypotension (Fig. 1) and CPB was instituted.

At the completion of the lung implantations, an attempt at weaning from CPB resulted in severe pulmonary hypertension and systemic hypotension. Epinephrine, dopamine, nitroglycerine, and inhaled NO were administered. After a further failed attempt at separation, an initial loading dose of milrinone 50 μg/kg was administered and an infusion commenced with no improvement (Table 1, F).

TEE revealed mild hypocontractility of the right ventricle with a hypercontractile left ventricle. Marked systolic anterior motion (SAM) of the anterior mitral valve leaflet was present (Fig. 1) with DLVOTO and severe mitral regurgitation (MR), which persisted throughout systole. Left ventricular hypertrophy (LVH) with wall thickness of 16 mm was present without evidence of asymmetric septal hypertrophy.

Norepinephrine and subsequently phenylephrine were substituted (for milrinone), and packed red cells and pentastarch were administered. SAM improved, MR was reduced to moderate, and it was possible to wean from CPB (Table 1, G). Two and a half hours after separation from CPB vasopressors were discontinued (Table 1, H). Hypotension later recurred and vasopressin 6 U/h was commenced. Repeat TEE at this time confirmed the presence of SAM with a left ventricular outflow tract (LVOT) gradient of 31 mm Hg associated with severe MR.

Discussion

DLVOTO is a cause of sudden death in hypertrophic cardiomyopathy. It has been described complicating myocardial infarction (1), cardiac tamponade (2), atrial fibrillation (3), mitral valve repair (4), and during dobutamine stress echocardiography (5). Flow acceleration in the LVOT causing a local reduction in pressure from the Venturi effect has been considered to be the cause of SAM, although recent work (6) demonstrates that SAM can develop early in systole when LVOT velocities are relatively low and emphasizes the role of flow drag. Contributing factors to development of SAM probably include reduced end-diastolic left ventricular (LV) dimensions because of hypovolemia or LVH and increased ejection velocities resulting from enhanced contractile state or peripheral vasodilation. Variations in mitral valve anatomy may play a role with anterior displacement of the coaptation line, redundant valvular tissue, and lax chordae possibly contributing (6).

The spectrum of abnormalities ranges from mild chordal SAM without significant outflow gradient to high-grade obstruction resulting in shock. The associated MR may range from minimal and/or transient to severe.

The initial interpretation of events in this patient was that pulmonary arterial hypertension with lung...
deflation was because of a further increase in resistance in a pathologic pulmonary vasculature and systemic hypotension as a result of acute right ventricular failure. There was no response to measures aimed at supporting right ventricular function and pharmaco-logically relieving pulmonary hypertension. The same situation persisted at initial attempts at separation from CPB.

Assessment with TEE demonstrated that the pulmonary hypertension was secondary to severe mitral regurgitation and hypotension resulting from DLVOTO. Although the right ventricle appeared mildly hypokinetic, it was not dilated and septal shift was not present. No unusual features of mitral valve anatomy were present.

Maneuvers that may reduce DLVOTO include intravascular volume expansion and heart rate reduction which increase LV end diastolic volume as well as vasoconstriction and reduction of inotropic state to reduce LVOT ejection velocities. The substitution of vasoconstrictor drugs for inotropes and volume expansion greatly reduced the hemodynamic impact of the obstruction. Inhaled NO appeared to produce no measurable benefit. The subsequent addition of β blockers in the intensive care unit was considered beneficial although we did not observe a clear relationship between timing of administration and hemodynamic changes.

TEE was invaluable in resolving the cause of hemodynamic instability and resulted in a major change in management and successful outcome.

Table 1. Hemodynamic Variables and Vasoactive Drug Administration

<table>
<thead>
<tr>
<th>Variable</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
<th>G</th>
<th>H</th>
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<tr>
<td>Arterial pressure (mm Hg)</td>
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<td>90/60</td>
<td>100/60</td>
<td>80/40</td>
<td>60/–</td>
<td>100/70</td>
<td>130/70</td>
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<td>40/28</td>
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<td>71/37</td>
<td>70/32</td>
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<td>43</td>
<td>24</td>
<td>22</td>
<td>21</td>
<td>22</td>
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<td>0</td>
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<td>0</td>
<td>0</td>
<td>5</td>
<td>3</td>
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<td>0</td>
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<td>0</td>
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<td>50</td>
<td>40</td>
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A = at induction of anesthesia; B = at right lung dissection; C = at left lung dissection; D = at right lung deflation, first attempt; E = at clamping of right pulmonary artery; F = at initial attempt at weaning from cardiopulmonary bypass (CPB); G = off CPB; H = at 2 h post-CPB.

Figure 1. The anterior mitral valve leaflet (arrow) seen obstructing the left ventricular outflow tract in systole in the midesophageal atrioventricular long axis view.

References