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Case Conference

Perioperative Management of a Large Idiopathic Pulmonary Artery Aneurysm Without Pulmonary Arterial Hypertension

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PULMONARY ARTERY (PA) aneurysm is a rare finding that occurs in only 0.007% of the general population.[1] The diagnosis most often is established when the main PA diameter exceeds the upper limit (2.9 cm) of normal,[2] but a vessel diameter >4.0 cm also may be used to define pathological enlargement.[3] Identification of PA aneurysms as "large" (5-8 cm in diameter) and "giant" (>8 cm) also has been proposed.[4] The presence or absence of PA hypertension is further used to classify PA aneurysms.[5,6] Congenital heart disease,[6–9] infection,[1,10–13] connective tissue disorders,[6,14] Behçet vasculitis,[15,16] malignancy,[17] chronic pulmonary embolism,[2] and idiopathic causes[18,19] may be responsible for the development of a PA aneurysm when PA hypertension is absent.[6,20] In the current report, the authors describe their management of a 64-year-old, 116 kg, 187 cm man with a known idiopathic PA aneurysm that had enlarged substantially during the preceding 6 months of surveillance. The anesthetic implications and surgical treatment of PA aneurysms also are discussed.

Case:*

The patient's PA aneurysm was first identified 5 years before the current admission when he underwent thoracic computed tomography (CT) as part of an evaluation for pulmonary embolism after elective orthopedic surgery. Serial CT scans documented gradual but progressive growth of the aneurysm from its original size of 5.0 cm to 6.2 cm over the following 51 months, but more rapid aneurysmal dilation to 6.7 cm (Fig 1) was noted during the most recent 6-month period of surveillance concomitant with worsening pulmonic valve insufficiency, as documented with transthoracic echocardiography. Surgical treatment was recommended. The patient denied experiencing cardiac and respiratory symptoms including syncope, dizziness, chest pain or pressure, palpitations, hoarseness, cough, orthopnea, and shortness of breath at

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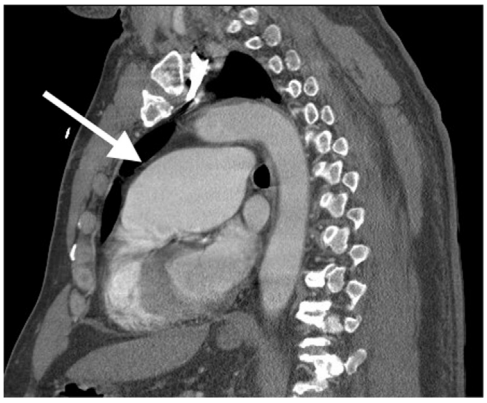

Fig 1. Sagittal computed tomography scan showing large pulmonary artery aneurysm (*white arrow*).

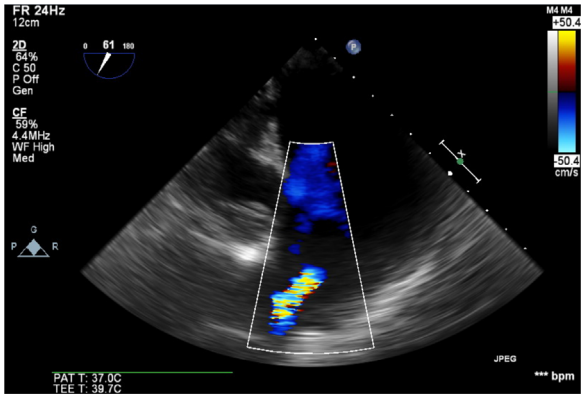

Fig 3. Upper esophageal aortic arch short-axis color Doppler transesophageal echocardiographic view showing moderate pulmonic valve insufficiency.

rest or with mild-to-moderate exertion. The remainder of the review of systems was noncontributory. The patient's medical history was notable for hypertension, hyperlipidemia, pulmonary embolism, chronic tobacco use, alcohol and cocaine abuse, and abdominal aortic and iliac aneurysms. He denied recent tobacco use and substance abuse. The patient also denied having a family history of aneurysmal disease and connective tissue disorders. His physical examination was unremarkable. Laboratory analysis failed to show occult infection nor did it provide evidence of rheumatologic or collagen vascular disease. Right heart cardiac catheterization revealed normal right ventricular (RV) and PA pressures (28/12 mmHg and 25/17 mmHg, respectively). RV systolic function (serial transthoracic echocardiography) and cardiac output (5.6 L/min; thermodilution) were normal. Coronary angiography showed no hemodynamically significant coronary artery stenoses. Magnetic resonance imaging confirmed the CT and echocardiography findings.

The patient was taken to the operating room for repair of the PA aneurysm. Arterial and central venous catheters were inserted using local anesthesia (1% lidocaine) in the presence of conscious sedation (midazolam and fentanyl). Anesthesia was induced using intravenous fentanyl (10 μg/kg), propofol (1 mg/kg), and rocuronium (1 mg/kg) and was maintained using inhaled isoflurane (end-tidal concentration of 0.5%-1.0%) in an air-oxygen mixture, fentanyl (1 to 2 μg/kg /h), and intermittent doses of rocuronium (0.05 mg/kg) titrated to effect using neuromuscular monitoring. Inspired oxygen concentration and positive-pressure ventilation were adjusted during surgery to avoid hypoxemia and hypercarbia (as confirmed with arterial blood gas analysis) and thereby prevent PA vasoconstriction, increases in pulmonary vascular resistance, and elevations in pressure within the aneurysm. Transesophageal echocardiography (TEE) demonstrated the PA aneurysm (Fig 2), which extended into the proximal right and left PA branches. Moderate central pulmonic valve insufficiency also was observed (Fig 3), but the structure of the valve leaflets and their motion were normal (not shown). Thus, the cause of the pulmonic valve insufficiency was presumed to be annular dilation. The aneurysm was immediately visible when the pericardium was opened (Fig 4, *white arrow*). The bulk of the aneurysm wall was directed toward the left PA. During cardiopulmonary bypass, a longitudinal arteriotomy was made in the PA and a normal pulmonic valve was encountered. The valve was repaired with commissural plication (Fig 5). A large elliptical section of the aneurysm then was resected and the reduction arteriotomy was closed in 2 layers over a #18 Hegar dilator to prevent stenosis of the main PA and its proximal branches (Fig 6). After the patient was rewarmed and his heart was deaired, the patient was weaned from cardiopulmonary bypass without difficulty using

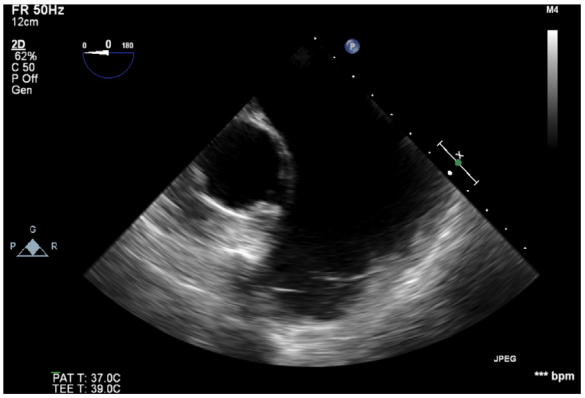

Fig 2. Midesophageal ascending aortic short-axis transesophageal echocardiographic view showing pulmonary artery aneurysm.

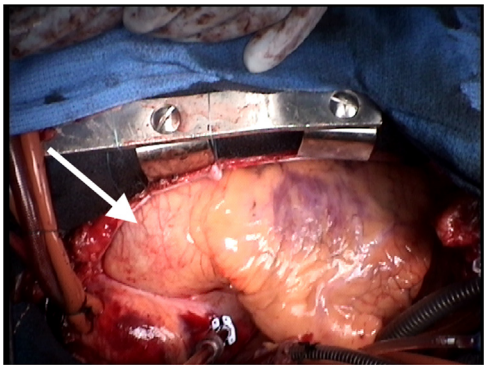

Fig 4. Intraoperative photograph showing giant pulmonary artery aneurysm (*white arrow*).

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