

# Endovascular Management of Early Lung Transplant–Related Anastomotic Pulmonary Artery Stenosis

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## ABSTRACT

**Purpose:** To report the safety and short-term efficacy of endovascular interventions for symptomatic lung transplant–related anastomotic pulmonary artery stenosis (PAS).

**Materials and Methods:** From February 2008 to December 2011, 354 lung transplants were performed. Pulmonary arteriography was performed in 19 patients (63% men; age, 57 y  $\pm$  21, mean  $\pm$  SD; seven double-lung transplants) because of respiratory decompensation (mean 6.7 mo after transplant). Seven arteriograms were normal, and 12 showed significant PAS. One patient (5%) underwent angioplasty alone, and 11 patients (57%) underwent stent placement.

**Results:** All patients underwent general anesthesia, and femoral access was used for the intervention. Technical success was 100% in the 12 patients treated. Symptoms improved in all patients who underwent intervention, with resolution in 11 of 12 (92%). There were no major or minor complications. Three patients (16%) had recurrent symptoms after discharge secondary to chronic rejection or pneumonia. Two patients died as a result of sepsis and multiorgan failure at 2 days and 14 days, respectively, after undergoing only pulmonary arteriography. In-stent stenosis occurred in 1 (9%) patient who required additional stent placement. During a mean follow-up period of 11 months, the remaining stents were patent, and the patients were asymptomatic.

**Conclusions:** Endovascular stent placement provides an alternative to open repair for transplant-related anastomotic PAS. It has low mortality and morbidity rates, and it has shown excellent short-term functional and anatomic outcomes.

## ABBREVIATIONS

PA = pulmonary artery, PAS = pulmonary artery stenosis

Anastomotic pulmonary artery stenosis (PAS) after lung transplantation was defined by Hausmann et al in 1992 (1) as an anastomotic diameter of < 75% that of the neighboring vessels. Anastomotic PAS is relatively rare with reports in the literature of < 2% (2–5). Causes of anastomotic PAS include excessive length of the donor and recipient segments, distortion of the anastomosis because of inadequate donor length, technical

anastomotic narrowing, twisting of the anastomosis, and intraluminal thrombus formation (1,4). Clinical manifestations include nonproductive cough, dyspnea, and fatigue with persistent pulmonary hypertension that can lead to pulmonary edema, effusion, hypoxemia, and ventilator dependence (2–5). The prognosis of lung transplant recipients who develop PAS is very poor; Clark et al (4) reported death in five patients with PAS within 15 days after single and bilateral lung transplantation. However, changes in surgical techniques continue to reduce the incidence of PAS. Open interventions have been performed to salvage the organ and the patient but carry a significant risk to both (4,6). To date, there have been few case reports of endovascular intervention early after lung transplantation. However, these reports demonstrated that percutaneous angioplasty with or without stent placement appears to be safe and efficacious for treatment of PAS (3,7–10). The purpose of this study is to report the contemporary

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None of the authors have identified a conflict of interest.

From the SIR 2011 Annual Meeting.

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*J Vasc Interv Radiol* 2015; 26:878–882

<http://dx.doi.org/10.1016/j.jvir.2015.02.017>

results of endovascular intervention for symptomatic lung transplant–related anastomotic PAS in a relatively large series of patients at a single institution.

## MATERIALS AND METHODS

The Houston Methodist Hospital institutional review board approved this study. A retrospective analysis was performed of the records of all patients who underwent endovascular interventions for lung transplant–related anastomotic PAS at our institution between February 2008 and December 2011. During this period, 354 patients underwent either single-lung or double-lung transplantation. Pulmonary arteriography was performed in 19 of these patients (63% men; age,  $57 \text{ y} \pm 21$ , mean  $\pm$  SD; seven double-lung transplants). All patients had presented with worsening respiratory status and pulmonary decompensation. Patient demographics and medical pulmonary and nonpulmonary comorbidities are listed in the **Table**. After pulmonary angiography, 12 of 19 patients required intervention. The indications for lung transplantation in these 12 patients were idiopathic pulmonary fibrosis in eight patients (67%), chronic obstructive pulmonary disease in three patients (25%), and cystic fibrosis in one patient (8%). Among the patients who did not undergo intervention ( $n = 7$  patients), the indications for lung transplantation were idiopathic pulmonary fibrosis ( $n = 5$  patients;

70%), chronic obstructive pulmonary disease ( $n = 1$  patient; 15%), and hypersensitivity pneumonitis ( $n = 1$  patient; 15%). Mean time from transplant to pulmonary arteriography was 6.7 months (range, 2 weeks to 27 months; intervention group 6.4 months vs no intervention group, 6.9 months). The average ischemia time during the initial lung transplant for the subsequent presumed stenotic PA was 226 minutes for patients who underwent interventions and 219 minutes for patients who underwent arteriography alone.

Before being considered for pulmonary angiography, patients underwent an extensive workup by the transplant pulmonary medicine service to exclude more common etiologies of pulmonary decompensation after lung transplantation: infectious, cardiogenic, and allograft rejection. All patients underwent contrast-enhanced computed tomography (CT) of the chest. CT suggested significant PAS, but the artifact secondary to donor recipient vessel size mismatch was often difficult to evaluate and exclude stenosis. Several patients underwent dynamic magnetic resonance angiography in addition to contrast-enhanced CT (**Fig 1**). Our current patient evaluation and treatment algorithm is shown in **Figure 2**.

All procedures were performed in a hybrid operating room suite under general anesthesia; one of two prophylactic antibiotics was administered (ie, intravenous cefazolin or vancomycin). Cardiovascular anesthesiology, thoracic transplant surgery, and cardiopulmonary bypass teams were available for all cases. Under ultrasound guidance, access was obtained to the right common femoral vein, and 6-F  $\times$  10-cm sheath was placed. Using a combination of a Bentson wire (Cook, Inc, Bloomington, Indiana) and a Mont (PA) pigtail catheter (Cook, Inc) or Berenstein catheter (Merit Medical Systems, Inc, South Jordan, Utah), access was gained to the pulmonary circulation. Initially, a main pulmonary arteriogram was performed, and this was followed by a selective pulmonary angiogram on the side of interest. After diagnostic imaging, a Berenstein catheter was advanced across the pulmonary arterial anastomosis using a 260-mm Glidewire (Onset Medical Corporation, Irvine, California). This wire was exchanged for either a 260-mm Amplatz Super Stiff Guide Wire (Boston Scientific, Marlborough, Massachusetts) or a Rosen wire (Cook, Inc), depending on PA tortuosity. A long 10-F  $\times$  100-cm sheath (Cook, Inc) was advanced into the PA of interest. The anastomosis was interrogated further with intravascular ultrasound (Volcano Corporation, San Diego, California) and by pull-back systolic pressure gradients. A dobutamine challenge (infusion of dobutamine beginning at 2.5  $\mu\text{g}/\text{kg}/\text{min}$  escalating until there was a 20% increase in heart rate from baseline) was used selectively in five patients, when the pull-back systolic pressure gradients did not correlate with either angiographic or intravascular ultrasound findings and when taken in context of the

**Table.** Patient Demographics and Medical Pulmonary and Nonpulmonary Comorbidities

Demographics	Value
No. patients	19
Age (y) $\pm$ SD	57 $\pm$ 21
Male patients	13 (68%)
Medical comorbidities	
Pulmonary related	
Idiopathic pulmonary fibrosis	14 (74%)
COPD	6 (34%)
Cystic fibrosis	1 (5%)
Hypersensitivity pneumonitis	1 (5%)
Non–small cell lung cancer	1 (5%)
Pulmonary embolism	1 (5%)
Oxygen dependent	15 (79%)
Tobacco use	6 (34%)
Nonpulmonary related	
Arterial hypertension	7 (37%)
Deep venous thrombosis	7 (37%)
Diabetes	5 (26%)
Hyperlipidemia	6 (34%)
Chronic kidney insufficiency	6 (34%)
Congestive heart failure	2 (10%)
Coronary artery disease	1 (5%)

Note—Values are presented as n (%) or mean  $\pm$  SD (range). COPD = chronic obstructive pulmonary disease.

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