Percutaneous Stenting of Bilateral Pulmonary Artery Stenosis Caused by Malignant Extrinsic Compression*

Christian Fierro-Benoy, MD; Hernes Velasquez, MD; Juan Pablo Zambrano, MD; Mustafa Ridha, MD; Kenneth Kessler, MD; and Alan Schob, MD

Percutaneous stenting of the pulmonary arteries (PAs) represents a potential option in cases of PA compression due to a variety of conditions. We present the first reported case of successful bilateral percutaneous stenting of the PAs in a patient with non-small cell lung cancer and severe right ventricular hypertension due to mediastinal lymphadenopathy compressing both PAs. Although the natural course of the disease was not altered, the patient had significant symptomatic relief without adverse effects. Additionally, there was objective evidence of improvement. This case suggests that endovascular stenting is a feasible palliative management option in patients with right ventricular failure due to malignant extrinsic compression of the PAs.

(CHEST 2002; 122:1478–1480)

Key words: lymphadenopathy; malignant stenosis; pulmonary artery; pulmonary hypertension; stenting

Abbreviation: PA = pulmonary artery

Non-small cell lung cancer may present as or be complicated by a variety of cardiovascular symptoms and signs, including those of pulmonary hypertension and right heart failure related to pulmonary artery (PA) compression. The treatment of PA compression has relied on the effects of surgery, radiation, or chemotherapy, the results of which may be delayed and unpredictable. Surgery may be impossible when the tumor involves the main PA.

Percutaneous stenting of the PAs represents a potential option in these cases and has been described in the literature for the treatment of congenital heart disease,2–4 strictures secondary to lung transplantation,5 pulmonary embolism,6 fibrosing mediastinitis,7 and unilateral malignant PA stenosis.8–10

We present the first reported case of successful bilateral percutaneous stenting of the PAs in a patient with lung adenocarcinoma and severe right ventricular hypertension due to mediastinal lymphadenopathy compressing both PAs.

CASE REPORT

The patient was a 53-year-old white male with an 80-pack-year tobacco history in whom stage IV non-small cell lung cancer had been diagnosed 13 months prior to hospital admission.

The patient had received weekly chemotherapy for the past year without radiation therapy. For the last 3 months, he had complained of worsening malaise, easy fatigability, chest pain, dyspnea on exertion, and edema. The findings of a follow-up chest CT scan prior to hospital admission revealed improvement in his initial left upper lobe nodule but the presence of a significant pericardial effusion. Although the presence of mediastinal disease was noted, involvement of the PAs was not initially appreciated. Pertinent physical findings on hospital admission were BP of 130/80 mm Hg with no significant pulsum paradoxus, a pulse of 72 beats/min, and a respiratory rate of 16 breaths/min. The patient was not in acute distress. His neck veins were distended to the angle of the jaw. Heart sounds were distant with a prominent P2 and a 3/6 holosystolic murmur at the left lower sternal border that increased with inspiration. No rubs were heard. Breath sounds were normal. There was hepatomegaly with a smooth, nonpulsatile surface. There was 3+ pitting edema up to the thighs. A transthoracic echocardiogram showed a large pericardial effusion without signs of tamponade. The right-sided chambers were enlarged. There was marked tricuspid regurgitation. A continuous-wave Doppler echocardiogram suggested the presence of severe pulmonary hypertension with an estimated PA systolic BP of 95 mm Hg.

Right heart catheterization and pericardiocentesis were performed for diagnostic purposes. Gradients of 47 and 83 mm Hg, respectively, were noted across the right and left main PAs (Table 1). Quantitative pulmonary angiography demonstrated 73% stenosis of the right PA (reference vessel diameter, 15 mm; minimal lumen diameter, 4 mm) and 80% stenosis of the left PA (reference vessel diameter, 10 mm; minimal lumen diameter, 2 mm). The main PA was moderately dilated, but the results of a distal pulmonary angiogram were normal.

Cytology of the pericardial fluid subsequently revealed adenocarcinoma. A review of a prior chest CT scan confirmed the presence of extrinsic bilateral PA compression due to extensive mediastinal adenopathy (Fig 1).

Despite pericardiocentesis, the patient’s symptoms persisted. After a multidisciplinary review of possible therapeutic options, bilateral PA stenting was performed to unload the right ventricle and to provide symptom relief. The day prior to undergoing angioplasty, the patient received 300 mg clopidogrel. Using the right femoral vein, an 8F hockey-stick catheter with sideholes (Cordis; Miami, FL) was advanced to the left PA. A balloon (Powerflex Plus 6 mm × 30 mm; Cordis) was used to predilate the lesion. A Corinthian stent (8 mm/29 mm; Cordis) then was deployed at 10 atm, achieving a reduction in the stenotic area from 80 to 30% (poststent diameter, 7 mm), no residual gradient, and excellent distal flow (Fig 2, 3). The guiding catheter was exchanged for an 8F multipurpose catheter with sideholes (Cordis), which was advanced to the right PA. The 6 mm/30 mm balloon (Powerflex Plus; Cordis) was used to predilate the

Table 1—Pressures Before and After Stenting

<table>
<thead>
<tr>
<th>Location</th>
<th>Prestent BP, mm Hg</th>
<th>Poststent BP, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>103/22</td>
<td>45/15</td>
</tr>
<tr>
<td>Pulmonary trunk</td>
<td>98/20</td>
<td>45/15</td>
</tr>
<tr>
<td>Proximal right PA</td>
<td>98/20</td>
<td>20/13</td>
</tr>
<tr>
<td>Proximal left PA</td>
<td>98/20</td>
<td>45/15</td>
</tr>
<tr>
<td>Distal right PA</td>
<td>25</td>
<td>15</td>
</tr>
<tr>
<td>Distal left PA</td>
<td>15</td>
<td>12</td>
</tr>
</tbody>
</table>

*Values given as mean.
lesion. A Corinthian 8 mm/29 mm stent (Cordis) then was deployed and subsequently was postdilated with a 10 mm/20 mm balloon (Powerflex Plus; Cordis) up to 9 atm. The final image revealed a 40% residual stenosis (poststent diameter, 9 mm), a residual gradient of 25 mm Hg, and excellent distal flow.

The patient tolerated the procedure well and had an uneventful recovery. Therapy with clopidogrel, 75 mg/d, was continued. On a follow-up visit to the clinic, the patient reported marked amelioration of his symptoms, resolution of edema, improved appetite, and significant improvement in activity tolerance. An echocardiogram that had been performed 4 weeks postprocedure showed a marked decrease in the degree of tricuspid regurgitation with an estimated PA systolic BP of 42 mm Hg (prior to the procedure, 98 mm Hg). Clinical follow-up at 4 months showed sustained improvement in cardiovascular symptoms.

**DISCUSSION**

Extrinsic compression of the main and branch PAs as a cause of right ventricular hypertension and leading to right ventricular failure is rare and can be missed easily. This case is unusual in that the severe pulmonary hypertension was due to bilateral extrinsic compression. This is the first report to demonstrate that this manifestation of malignant pulmonary disease can be ameliorated by bilateral PA stent placement. When clinically suspected, the diagnosis of this entity can be made by contrast CT scan or MRI. Right heart catheterization with pulmonary angiography can be used to confirm the diagnosis. The use of a stent endoprosthesis in the PAs has been described in a variety of clinical situations.2,5–11 The main objective has been to avoid vessel recoil, to support the vascular wall in cases of extrinsic compression,12 and to prevent and treat intimal dissection. The largest experience with stent endoprostheses in the PAs has been seen in children and young adults in whom stents have been used to relieve postsurgical and isolated proximal and distal PA stenosis. Early and late (up to 5 years of follow-up) clinical results have been excellent.2–4, 13 Other reports have described the use of stents to treat unilateral PA stenosis due to malignancy, fibrosing mediastinitis, and bronchial carcinoma with excellent proce-

![Figure 1. CT scan showing severe right and left PA stenosis.](image1)

![Figure 2. PA angiograms showing preprocedural and postprocedural results.](image2)
dural success. While clinical improvement has been uniformly noted, it often has been tempered by the underlying disease processes.7–10

Complications that have been reported in the literature have included ventricular arrhythmias, compromise of arterial sidebranches,9 intimal flaps and arterial rupture,3 misplacement and migration of stents, and pulmonary edema.2,4

In our patient, who presented with right-sided heart failure due to bilateral extrinsic compression of the PAs caused by extensive mediastinal lymphadenopathy due to adenocarcinoma of the lung, there were limited therapeutic options. The severe pulmonary hypertension appeared to represent an immediate threat to both the quality and quantity of this patient’s life. The angiographic appearance of the compressive lesions appeared to be amenable to stent placement. The patient was treated successfully with the placement, percutaneously, of bilateral PA stents. Although the natural course of the disease was not altered, he had significant symptomatic relief without adverse effects. Additionally, there was objective evidence of improvement.

In conclusion, this case suggests that in patients with mediastinal disease and pulmonary hypertension, evaluation should include careful examination of the mediastinum by CT scanning or MRI for possible PA compromise. Endovascular stenting is a feasible palliative management option in patients with right ventricular failure due to malignant extrinsic compression of the PAs.

REFERENCES


Pulmonary Alveolar Proteinosis*

Treatment by Bronchofiberscopic Lobar Lavage

Shih-Lung Cheng, MD; Hon-Tai Chang, MD; Hon-Ping Lau, MD; Li-Na Lee, MD, PhD; and Pan-Chyr Yang, MD, PhD, FCCP

Figure 3. PA angiograms showing preprocedural and postprocedural results.