FIGURE 2. Intraoperative photograph of patient 3 showing the exposure of the pseudoaneurysm (arrowhead) at the inferior surface of the heart. The pseudoaneurysm was resected successfully, and the defect was covered by Teflon patches.

**DISCUSSION**

Although the estimated incidence of ventricular free-wall rupture following AMI is 25,000 cases per year in the United States alone, a recent review listed only 90 cases of subacute rupture in which the patients survived to hospital discharge.\(^5\) All survivors had successful operative repair of the rupture. To our knowledge, the longest interval between subacute rupture and corrective surgery was 54 days in a case described by Raitt et al.,\(^5\) but the authors did not state the length of survival, and there is general agreement that long-term survival can be achieved only by emergency surgery.

We report on three well-documented cases of subacute ventricular free wall rupture in which patients survived for years without emergency surgery. All our patients were hemodynamically impaired at the time of rupture and at first had nonencapsulated pericardial effusions. This argues against the occurrence of myocardial rupture into a space already confined by epicardium and pericardial adhesions which may go unnoticed until rupture of the pseudoaneurysm causes sudden death.\(^6\) The pseudoaneurysm in patient 3 must have developed either very gradually or at some time later than 3 weeks after AMI, since it was not seen by echocardiography or MRI of the chest during the first hospitalization but was readily visible at contrast ventriculography 3 months after the AMI.

To place our three cases in context, we reviewed the charts of patients with AMI treated in our ICU between the years 1986 and 1994 who developed myocardial free-wall rupture. Among 2,862 patients treated for AMI, 107 patients had rupture of the myocardial free wall documented by autopsy in 104 cases and by echocardiography or delayed operation in the 3 cases previously described. In 29 patients, the clinical course of rupture was subacute, arbitrarily defined as survival for 1 h or more from the time of abruptly developed hemodynamic compromise. Since emergency surgical repair of ruptured myocardium was not available in our hospital during the last 8 years, all patients were treated initially only with hemodynamic support. Only 3 of 29 (10%) patients survived to hospital discharge. Accordingly, survival in our patients was much lower than in patients who had undergone emergency surgical repair and survived long term in about 50%.\(^3\)

In conclusion, our report indicates that in a small subset of patients with subacute ventricular free wall rupture the course is more benign than was previously thought possible. As long as no criteria exist to identify those patients in advance, however, prompt surgical repair remains the preferred treatment.

**REFERENCES**


**Treatment of Left Pneumonectomy Syndrome With an Expandable Endobronchial Prosthesis**

Francis C. Cordova, MD; John M. Travaline, MD; Gerald M. O’Brien, MD; David S. Ball, DO; and Michael Lippmann, MD

Postpneumonectomy syndrome has only been described after a right pneumonectomy except in cases of congenital mediastinal anomalies or right-sided aortic arch. Placement of Silastic prostheses into the empty

*From the Division of Pulmonary and Critical Care Medicine, Department of Medicine, and the Division of Interventional Radiology, Department of Diagnostic Imaging, Temple University School of Medicine/Albert Einstein Medical Center, Philadelphia. Reprint requests: Dr. Travaline, Division of Pulmonary and Critical Care Medicine, Temple University School of Medicine, 3401 N. Broad Street, Philadelphia, PA 19140*
hemithorax is the preferred surgical treatment; how-
ever, other nonsurgical options exist. Herein, we report a case of left postpneumonectomy syndrome in an adult who was successfully treated with the placement of an endobronchial stent.

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Key words: endobronchial stent; postpneumonectomy syndrome

Postpneumonectomy syndrome is a rare complication characterized by extrinsic compression of the mainstem bronchus between the pulmonary artery anteriorly, and the aorta and the thoracic vertebrae posteriorly, due to herniation of mediastinal structures into the empty hemithorax. The syndrome usually occurs after right pneumonectomy but has been described after left pneumonectomy in the presence of right-sided aortic arch or congenital mediastinal anomalies.1 In this report, we describe a case of left postpneumonectomy syndrome in an adult and its successful management with an endobronchial stent.

CASE REPORT

A 34-year-old woman with sarcoidosis was admitted for severe respiratory distress. Four years prior to presentation, she required a left pneumonectomy for massive hemoptysis secondary to an aspergilloma. One year prior to presentation to our institution, she began to experience exertional dyspnea and wheezing. These symptoms progressed, and treatment with bronchodilators, corticosteroids, and theophylline provided little improvement. She had an FVC of 1.77 L (56% of predicted), an FEV₁ of 1.03 L (41% of predicted), and an FEV₁/FVC ratio of 51%

On presentation, she was in severe respiratory distress with a respiratory rate 40 breaths per minute, perspiration, and cool clammy skin. She had suprasternal and substernal retractions, accessory muscle recruitment, and bilateral wheezing. Pulse oximetry showed 92% oxygen saturation while breathing ambient air.

Despite intensive treatment with intravenous methylprednisolone, albuterol, and supplemental oxygen, her oxygen saturation fell to 72%, and she was intubated and placed on mechanical ventilation. Because of high peak airway pressures (60 to 65 cm H₂O), she was sedated and paralyzed to optimize her ventilation. A chest x-ray film showed leftward deviation of the trachea and mediastinum. The right lung was hyperinflated and partially herniated into the left hemithorax. Pleural thickening and interstitial infiltrates were noted. Despite intensive bronchodilator and high-dose corticosteroid therapy, she was unable to be weaned from mechanical ventilation.

To evaluate the possibility of major airway compromise, flexible fiberoptic bronchoscopy was performed and revealed elongated narrowing of the distal trachea and right mainstem bronchus. A CT scan of the chest showed compression of the right mainstem bronchus between the mediastinum and thoracic vertebrae (Fig 1).

To restore airway patency, a stent (Wallstent; Schneider; Minneapolis), 12×40 mm, was positioned in the right mainstem bronchus. Because of the sharp angulation of the right mainstem bronchus with the trachea, and the necessary selection of a 12-mm diameter stent to relieve the right mainstem bronchus obstruction, the proximal one third of the stent was situated in the distal trachea. Immediate reinspection of the airway with a flexible bronchoscope after the stent was deployed showed patency of the right mainstem bronchus but narrowing of the intermediate bronchus secondary to bronchomalacia. For this obstruction, a second stent measuring 10×20 mm was positioned to overlap the first stent and thereby relieve the obstruction. Although the placement of the second stent traversed the right upper lobe bronchus, this was unavoidable in order to ensure adequate airway patency to the right middle and right lower lobes. However, although the stent passed in front of the right upper lobe bronchus, access to this lobe was easily gained with the bronchoscope by following the lumen on the outside of the first stent (Fig 2).

Immediately after the placement of the stents, the patient’s peak inspiratory pressure decreased to 15 to 20 cm H₂O. She was suc-

**Figure 1.** CT scan of the chest showing compression of the right mainstem bronchus immediately below the carina (arrow) between the vascular structures anteriorly and the thoracic vertebrae posteriorly. An incidentally found pleural-based hyperdensity and loculated pneumothorax are also seen on this section. These abnormalities were of no clinical significance and were chronic in nature.

**Figure 2.** Endoscopic view demonstrating the proximal extension of the stent into the distal trachea and the small lumen leading to the right upper lobe bronchus.
successfally weaned from mechanical ventilation and was extubated within 24 h. Three weeks later, repeat spirometry showed improvement in the degree of airflow obstruction (FEV1/FVC ratio, 74%). Another CT scan of the chest (Fig 3) showed the airway improvement after the stent was put in place. She continued to do well after a 12-month follow-up period.

**DISCUSSION**

The differential diagnosis of progressive shortness of breath after a pneumonectomy should include postpneumonectomy syndrome. The syndrome was originally described after right pneumonectomy but can occur after left pneumonectomy in the presence of congenital mediastinal anomalies and right-sided aortic arch. This case, however, documents pneumonectomy syndrome with no associated congenital mediastinal anomalies or right-sided aortic arch. The most common clinical presentation is progressive exertional dyspnea, and recurrent infection and symptoms can occur as early as a few weeks postpneumonectomy, especially in infants and young children. The exact pathophysiology of pneumonectomy syndrome is not well elucidated, but since this syndrome is more common in children, it is believed that the extreme herniation of the mediastinum and marked hyperinflation of the lung are the result of the elasticity and increased compliance of the developing mediastinum and remaining lung. The herniation then results in extrinsic tracheal or bronchial compression, or both. Early recognition and treatment of this syndrome is important to prevent malacic changes of the airways due to prolonged extrinsic compression.

Management options for this syndrome are limited, and reports of successful treatment are largely anecdotal. Various surgical techniques have been described include division of the aortic arch with interposition of a Dacron graft between the ascending and descending aorta, right pneumonectomy to decrease thoracic volume, and instillation of saline solution and albumin into the empty hemithorax. The preferred surgical procedure, however, is the placement of an expandable Silastic prosthesis within the empty hemithorax in an attempt to return the mediastinum to the midline and relieve the bronchial obstruction. Excellent results have been reported in children with the use of an expandable prosthesis to allow for the future growth of the chest cavity. Recently, Grillo et al reported similar favorable outcomes in five of eight adult patients after mediastinal repositioning with silicone prostheses in 5 months to 6 years of follow-up. However, persistent airway obstruction due to bronchomalacia after surgical intervention remained a significant dilemma with no clear solution. In our patient, because of extensive pleural adhesions and sepsis, surgery was deemed not to be the best treatment option.

Recently, self-expanding bronchial stents have been successfully used in the treatment of tracheal and bronchial obstruction due to malignancy, tuberculosis, congenital stenosis, and postoperative bronchial stenosis and malacia. The Wallstent and Gianturco Z-stent (Cook; Bloomington, Ind) are two types of self-expanding metallic stents which are readily available in the United States. In addition to these self-expanding metallic stents, a variety of Silastic stents such as the Dumon, Hood, and Montgomery tubes are commonly employed in the airway to relieve obstructions. In our patient, who was critically ill, intubated, and receiving mechanical ventilation, it was deemed too risky to attempt rigid bronchoscopy which would have been necessary to deploy any of the Silastic stents.

In general, it is recommended that when these stents are placed in the airway that they maintain contact with the bronchial wall so that over time they embed themselves in the mucosa to help prevent stent migration and promote epithelialization. In our case, however, because of the already distorted airway, and the need to use an appropriately sized stent, the proximal portion of the first stent extended into the distal tracheal lumen and was not able to embed itself into the mucosa. While this departs somewhat from the conventional technique of placing the stent, it was unavoidable in this case. Moreover, in this patient, there were no complications related to its positioning, and the patient continues to do well.

Another issue in this case with regard to the stent placement is that it was necessary to traverse the right upper lobe bronchus in order to provide an adequate airway. The use of the stent for vascular stenoses allows the stent to overlie side branches because in most cases they remain patent. In the airway, this also does not appear to create a problem. In this case, while the stent traversed the right upper lobe bronchus, it was sufficiently away from the wall so as to permit a bronchoscope to pass on the outside of the stent and gain access to the right upper lobe. In some cases, however, the stent may cross over a bronchus more closely, and in such cases it may be necessary to use a Nd-YAG laser to enlarge an area in the side of the stent to facilitate drainage from a bronchus.

The use of airway stents in the treatment of postpneumonectomy syndrome is limited. Dumon reported unsuccessful attempts to relieve bronchial obstruction in two patients with respiratory failure due to postpneumonectomy syndrome using a molded silicone prosthesis.

We suggest that self-expandable metallic stents should be considered an alternative treatment option in the management of adult patients with postpneumonectomy syndrome who are not surgical candidates.
Postcardiac Injury Syndrome*

An Immunologic Pleural Fluid Analysis

Sola Kim, MD, and Steven A. Sahn, MD, FCCP

The postcardiac injury syndrome (PCIS) is characterized by inflammation of the pericardium, pleura, and pulmonary parenchyma following a variety of cardiac injuries. Although it has been clinically recognized for decades, confirmation of the syndrome has been problematic owing to lack of a sufficiently diagnostic test. Previously, we have reported pleural fluid characteristics which help to exclude other diagnoses that may mimic the syndrome. We describe the first immunologic assessment, including antmyocardial antibody testing, of pleural fluid from a patient with PCIS which supports a local immunologic mechanism in the pathogenesis of the syndrome. These results support the important role of pleural fluid analysis in the diagnosis of PCIS.

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Key words: antmyocardial antibody; pleural effusion; postcardiac injury syndrome

The postcardiac injury syndrome (PCIS) is characterized by fever, pericardial inflammation, and pleuropulmonary manifestations days to weeks following a variety of injuries to the myocardium or pericardium. It has been described following myocardial infarction,1 cardiac surgery,2 blunt chest trauma,3 percutaneous left ventricle puncture,4 and implantation of a pacemaker.5 Although PCIS has been a clinically recognized entity for several decades, the lack of a confirmatory diagnostic test renders the diagnosis one of exclusion. Previously, we have identified several pleural fluid characteristics which help to differentiate the diagnosis of the syndrome from other clinical conditions that may appear following cardiac injury.6 Now we describe the first report of antmyocardial antibody (AMA), reduced complement levels, and immune complexes identified in the pleural exudate of a patient with PCIS following coronary artery bypass grafting. We suggest a new utilization of pleural fluid analysis to aid in the diagnosis of PCIS.

CASE REPORT

A 37-year-old man was hospitalized 11 days after coronary artery bypass surgery with several days of dyspnea, low-grade fever, and pleuritic chest pain. His symptoms progressed despite outpatient treatment with a diuretic and a nonsteroidal anti-inflammatory agent. The bypass operation utilized bilateral internal mammary artery grafting during which both pleural spaces were entered. He had no history of preexisting autoimmune disease.

Physical examination showed a temperature of 38.0°C and a respiratory rate of 30 breaths per minute. Jugular venous pressure was not elevated. An S3 gallop was absent. A trace amount of pedal edema was noted. The chest radiograph showed bilateral, moderate, free-flowing pleural effusions. With the patient breathing room air, arterial blood gas values were as follows: pH, 7.45; PaCO2, 41 mm Hg; PaO2, and 72 mm Hg. Other laboratory findings included a WBC count of 16,400/μL and an erythrocyte sedimentation rate of 41 mm/h. Thirteen days postoperatively, thoracentesis was performed on the right effusion, which revealed a serosanguineous fluid with a total protein level of 3.7 g/dL (serum, 5.4 g/dL); lactate dehydrogenase level, 999 IU/L (serum, 218 IU/L); WBC count, 1,092/μL (64% lymphocytes, 24% macrophages, 8% polymorphonuclear leukocytes, 3% eosinophils, 1% basophils); RBC count, 21,306/μL; glucose value, 105 mg/dL (serum, 103 mg/dL); and pH, 7.37. Routine bacteriologic studies were negative for organisms. Ventilation-perfusion lung scan disclosed a low probability for a pulmonary embolus. The echocardiogram showed an ejection fraction greater than 60% and a small pericardial effusion.

AMA was detected in pleural fluid and serum by indirect immunofluorescence staining against monkey heart tissue substrate and striated muscle control specimens.7 The pleural fluid AMA titer was positive at 1:80 dilution, while the serum AMA titer was positive at 1:40 dilution (Table 1). Pleural fluid complement levels were low: C3, 44.7 mg/dL; and C4, less than 8 mg/dL. Serum complement levels were within normal range: C3, 116 mg/dL (normal, 53 to 177 mg/dL); and C4, 21 mg/dL (normal, 15 to 45 mg/dL). When complement levels were adjusted for total protein concentrations (pleural-to-serum C3 ratio divided by pleural-to-serum total protein ratio), the C3 index was 0.56 and the C4 index, less than 0.56. The C1q binding assay detected the presence of immune complexes in the pleural fluid but not in the serum.

Despite treatment with ibuprofen, the patient experienced...