Delayed Pleuropulmonary Complications following Coronary Artery Revascularization with the Internal Mammary Artery

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We have seen four cases of delayed postoperative pleuropulmonary complications associated with use of the internal mammary artery (IMA) conduit. In each case the left IMA was used as a bypass conduit to the left anterior descending (LAD) coronary artery. In two of the instances the complications were life-threatening to the patients. Each patient was left with symptomatic residual roentgenographic changes. The IMA is becoming the graft of choice for coronary artery revascularization. The potential for delayed pleuropulmonary complications associated with use of this graft is not well recognized.

The internal mammary artery (IMA) conduit is becoming the graft of choice for coronary artery revascularization due to its superior patency rates over saphenous vein grafts and improved patient survival with this conduit. Over a six-month period we have seen four cases of delayed pleuropulmonary complications associated with use of the left IMA conduit. As internists and pulmonary specialists we were the first to evaluate these patients with their new respiratory complaints. The relationship of the pleuropulmonary complications to the original bypass surgery was often not appreciated early in the evaluation of these patients.

Case Reports

Case 1

This 58-year-old man had a history significant for a myocardial infarction 13 years earlier. Complete heart block developed requiring implantation of a Medtronic VVI pacemaker. Two weeks following this procedure, unstable angina occurred requiring intravenous (IV) nitroglycerin for control. Emergent cardiac catheterization revealed an 80 percent stenosis of the proximal LAD artery and a 90 percent stenosis of the mid-portion of the LAD artery. Two obtuse marginal branches were occluded, and the right coronary artery (RCA) was proximally occluded and filled retrograde through collaterals. There was also a 70 percent stenosis of the posterior descending artery (PDA). A multiple-gated acquisition (MUGA) imaging study showed an ejection fraction of 20 percent. Coronary artery revascularization was done using saphenous vein grafts to the PDA and to the proximal LAD. The left IMA was grafted to the mid-portion of the LAD. The left pleural cavity was opened to facilitate harvesting of the left IMA.

The patient did well postoperatively. Chest roentgenogram prior to discharge was normal. Five weeks postoperatively the patient complained of increasing shortness of breath and left-sided chest heaviness. A chest roentgenogram (Fig 1) showed a large left pleural effusion. Thoracocentesis of the left effusion yielded 2,200 ml of a dark amber-colored fluid. The characteristics of the pleural fluid are presented in Table 1. No evidence of infection or malignancy was found. The patient's symptoms resolved after the thoracocentesis, and he was discharged. A chest roentgenogram at six weeks following the thoracocentesis showed only a small residual left pleural effusion.

Case 2

This 68-year-old man had a history significant for angina refractory to medical therapy. He underwent quadruple coronary artery bypass to include an IMA graft to the LAD coronary artery. A left pleurotomy was done at the time of coronary revascularization. The patient did well until four months after the bypass procedure, when he developed a cough with dyspnea on exertion.

A chest roentgenogram (Fig 1) revealed a large left pleural effusion that had not been present at discharge from his bypass procedure. Thoracocentesis showed a grossly bloody effusion that was an exudate (Table 1). Results of pleural fluid cytologic study, bacterial cultures, tuberculous and fungal cultures, and antinuclear antibody (ANA) were all negative. Spirometric examination results revealed a new restrictive defect with a FVC of 1.91 L (predicted, 4.21 L) and an FEV, of 1.76 L (predicted, 3.09 L). Computed tomography of the chest showed a large loculated left pleural effusion (Fig 2). Closed pleural biopsy examination was done, revealing no evidence of malignancy or infection. Closed-tube thoracostomy was done under ultrasound guidance, and 400 ml of serosanguinous fluid was removed; however, the left lung remained entrapped without full expansion.

The patient underwent thoracotomy and pleural decortication, which removed the loculated pleural effusion. This allowed nearly complete reexpansion of the left lung. The pathologic specimen showed no evidence of tumor, infection, or granulomatous disease. Postoperatively the patient had clinical and spirometric improvement, with an FVC of 2.63 L. A chest roentgenogram four weeks postoperatively showed a reexpanded left lung with postsurgical

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Changes.

Case 3
The patient was a 60-year-old man with a history significant for a myocardial infarction ten years earlier. He had acute onset of chest pain and, subsequently, a nontransmural inferolateral myocardial infarction. An emergent cardiac catheterization was done, revealing a 99 percent LAD stenosis, a high-grade stenosis of the second obtuse marginal branch, and a 50 percent stenosis of the RCA. Left ventricular wall function appeared normal. Coronary bypass surgery was urgently done, utilizing the left IMA as a graft to the LAD. A sequential saphenous vein graft was anastomosed to the obtuse marginal branch, terminating on the PDA. To facilitate use of the left IMA, a left pleuropericardial incision was made.

The patient had a postoperative chest x-ray film that showed no evidence of pleural effusion. Eight weeks after surgery, he was walking two miles daily. Three months after surgery, the patient noted increasing dyspnea on exertion. A chest roentgenogram (Fig 3) showed a new large left pleural effusion. Thoracocentesis yielded 1,400 ml of a hemorrhagic fluid (Table 1). This resulted in marked symptomatic improvement. Analysis of the pleural fluid showed no evidence of malignancy or infection.

Three weeks after the thoracocentesis, the patient had a recurrence of the dyspnea on exertion. Chest roentgenogram showed reaccumulation of the left pleural effusion to its original level. Thoracocentesis yielded 2,200 ml of serosanguinous fluid (Table 1). The patient again improved symptomatically. The patient has remained symptom free at three months following his second thoracocentesis. Chest roentgenogram at that time showed pleural thickening and blunting of the left costophrenic angle.

Case 4
The patient was a 75-year-old man with a history significant for an inferior myocardial infarction 26 years earlier and stable angina pectoris. He had unstable angina requiring admission to the coronary care unit and IV nitroglycerin for control of his chest pain. A myocardial infarction was ruled out using serial ECGs and cardiac isoenzyme determinations. Owing to the patient's unstable angina, an urgent cardiac catheterization was performed. This revealed a 90 percent occlusion of the LAD artery, a 100 percent occlusion of the circumflex artery, and a 100 percent occlusion of the RCA. The left ventricular ejection fraction was 60 percent. Coronary artery revascularization was performed, using the left IMA as a conduit to the LAD artery. A left pleurotomy was done to facilitate exposure. Single saphenous vein grafts were anastomosed to the first diagonal branch and the PDA. A sequential saphenous vein graft to two obtuse marginal branches was also placed.

The patient's immediate postoperative course was complicated by ventricular ectopy requiring antiarrhythmic therapy. Subcutaneous emphysema developed after removal of the chest tube. This resolved spontaneously. Twenty-one days after surgery, the patient began to develop increasing respiratory distress. This progressed over several hours, with increased dyspnea and respiratory rate and labored breathing. Acute respiratory acidosis developed. A chest roentgenogram showed a new large left pleural effusion (Fig 3). As the patient was being intubated, ventricular fibrillation developed. The patient was intubated and had conversion to a sinus rhythm by electric defibrillation.

The patient was slowly weaned off the ventilator over ten days. His chest tube was removed. A thoracocentesis yielded 1,900 ml of serosanguinous fluid (Table 1). The pleural fluid showed no evidence of malignancy or infection. Following the thoracocentesis, increasing

**Table 1—Pleural Fluid Characteristics**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Protein, g/dl</th>
<th>LDH, units/L</th>
<th>Glucose, mg/dl</th>
<th>WBCs, cu mm</th>
<th>RBCs, cu mm</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>3.7</td>
<td>213</td>
<td>93</td>
<td>2,900</td>
<td>7,250</td>
</tr>
<tr>
<td>2</td>
<td>5.5</td>
<td>1,501</td>
<td>82</td>
<td>622</td>
<td>1,200,000</td>
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<tr>
<td>3</td>
<td>4.2</td>
<td>197</td>
<td>128/</td>
<td>439/</td>
<td>74,250/</td>
</tr>
<tr>
<td>3</td>
<td>3.8</td>
<td>129</td>
<td>500</td>
<td>32,250</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>19</td>
<td>—</td>
<td>123</td>
<td>111</td>
<td>235,000</td>
</tr>
</tbody>
</table>

The patient was discharged to a nursing home.
respiratory insufficiency developed. Respiratory acidosis recurred despite aggressive support. The patient elected reintubation and mechanical ventilation. Over the next 48 hours, the pleural effusion reaccumulated to its original level, and thoracocentesis was repeated. Over five days, the patient was weaned off the ventilator. The patient’s condition progressively improved, and at four months following the bypass procedure the left pleural effusion has not reaccumulated.

**DISCUSSION**

Our four patients represent examples of delayed pleuropulmonary complications following the use of the IMA conduit for coronary revascularization. The onset of the presenting symptoms from the time of surgery ranged from three weeks to four months. In two of the cases the complications were potentially life-threatening. The range of pleuropulmonary complications in our cases included pleural effusion formation (recurrent in two patients), pleural fibrosis, and respiratory insufficiency. The association of the pleuropulmonary complications with the bypass procedures was not immediately evident at presentation in our patients, especially when symptoms occurred later than one month following the time of bypass surgery. All four of these cases involved use of the left IMA as a conduit to the left anterior descending coronary artery through an intrapleural route. Pleurotomy has been advocated in the literature on thoracic surgery in this setting, because it provides a more direct intrapleural course for the IMA pedicle to the heart, avoiding compression of the conduit from lung expansion.

There are several reasons to expect an increase in pleuropulmonary complications when using the IMA conduit. These include opening the pleural sac with placement of pleural drainage tubes and the duration of time these tubes are left in place postoperatively; possible injury to the phrenic nerve; and trauma to the chest wall during harvesting of the IMA pedicle creating a denuded surface area requiring repleuralization. In our patients the left pleural tubes were left in place for two to four days postoperatively until pleural drainage was felt to be minimal by the thoracic surgeon.

In all of our cases pleural effusion formation was the major factor contributing to pleuropulmonary complications. Olearchyk et al\(^8\) in their series examined 833 patients who underwent IMA grafting alone or in conjunction with use of saphenous vein grafts. Mediastinal and pleural tube drainage after IMA dissection without pleurotomy was \(849 \pm 523\) ml. This was significantly less (\(p<0.05\)) than the mediastinal and pleural tube drainage found when the pleura was opened, which was \(1,187 \pm 683\) ml. This study suggested that pleurotomy itself could interfere with the lymphatic clearance of pleural fluid or contribute to its production.

The effects of pleurotomy may extend beyond increasing total mediastinal and pleural tube drainage in the postoperative period. Burgess et al\(^9\) examined 32 patients undergoing elective coronary artery bypass surgery. They showed that pleurotomy led to lower postoperative oxygen tensions and higher shunt fractions in bypass patients irrespective of the type of conduit used. These consequences of pleurotomy were thought to result from changes in pleural surface pressure allowing lung retraction, which has also been shown to increase total flow resistance of the airways, decrease total static compliance of the lung, and increase the alveolar-arterial oxygen tension gradient in patients undergoing coronary artery bypass.\(^10\)

In our four patients the left pleurotomy contributed to the occurrence of their pleuropulmonary complications. The pleural effusions in all cases were exudates, and in at least the first case may have been exacerbated by underlying congestive heart failure.
Early treatment of these pleural effusions with needle or catheter drainage is justified for symptomatic improvement and may minimize development of later complications such as pleural fibrosis or respiratory insufficiency, as seen in two of our cases. Management of congestive heart failure may also decrease the incidence of these complications. More aggressive treatment of the pleural effusions may be necessary if they reaccumulate or appear to be loculated. Recognizing the potential for delayed pleuropulmonary complications following use of the IMA conduit is important, especially in the elderly patient or the patient with compromised cardiopulmonary function.

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