Chronic Pleural Effusion following Coronary Artery Revascularization with the Internal Mammary Artery*

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Two patients developed chronic symptomatic pleural effusions following coronary artery revascularization with the IMA. Both patients had evidence of left ventricular dysfunction and pleural effusions which only involved the side corresponding to the harvested IMA. (Chest 1990; 97:750-51)

Recently, delayed pleuropulmonary complications following coronary artery revascularization with the IMA conduit have been described. The development of chronic symptomatic pleural effusion following coronary artery bypass with the IMA is not reported. Two patients with this complication are described.

CASE REPORTS

Case 1

This 52-year-old man had a history significant for an acute anteroseptal myocardial infarction 20 months earlier. Cardiac catheterization showed severe three-vessel disease. Left ventricular angiography showed severe hypokinesia of the anterior apical wall and the posterior basilar wall, and the ejection fraction was 20 percent. Coronary artery revascularization was performed with a left IMA graft to the LAD and four additional saphenous vein grafts. The left pleural cavity was opened to facilitate the harvesting and anastomosis of the left IMA to the LAD. Pleural and mediastinal tubes remained in place for the first 24 hours after surgery.

Four days after surgery, a chest roentgenogram showed a large left-sided pleural effusion which was new compared to the preoperative chest roentgenograms. Thoracocentesis revealed an exudate without evidence of malignancy or infection. The patient's congestive heart failure was treated after surgery with furosemide, digoxin, and enalapril. Chest roentgenograms taken at intervals showed persistence of the left-sided pleural effusion over 18 months. A repeat thoracocentesis and closed pleural biopsy with an Abrams' needle were performed.

The pleural fluid was hazy and straw-colored, with a glucose level of 94 mg/dl, protein level of 4.0 g/dl (serum protein level, 6.8 g/dl), and LDH level of 149 units/L (serum LDH, 192 units/L). There were 920 RBCs and 633 WBCs per cubic millimeter, with a differential of 3 percent neutrophils, 17 percent lymphocytes, 48 percent histiocytes, and 32 percent mesothelial cells. Cultures and stains of the pleural fluid and biopsy were negative for infection or malignancy; however, pleural fibrosis and chronic inflammation were present on the biopsy. The left-sided pleural effusion reaccumulated after thoracocentesis (Fig 1) and persisted during medical therapy for the patient's congestive heart failure, which otherwise was compensated.

Case 2

This 76-year-old man had developed unstable angina 12 months earlier and had undergone cardiac catheterization, which revealed three-vessel disease and an ejection fraction of 65 percent. Coronary artery revascularization was performed utilizing a left IMA graft to the LAD. A left pleurotomy was performed to allow a more proximal anastomosis of the IMA conduit to the LAD. A left pleural chest tube and mediastinal tube were placed for drainage.

After surgery, prior to discharge from the hospital, the patient had evidence of a large left-sided pleural effusion that was new from preoperative chest roentgenograms and of congestive heart failure. He was treated with digoxin, furosemide, and nifedipine. Thoracocentesis with removal of most of the pleural fluid showed an exudative effusion with 56,800 RBCs and 500 WBCs per cubic millimeter, with 4 percent neutrophils, 42 percent lymphocytes, 34 percent histiocytes, 17 percent eosinophils, and 3 percent basophils. Cytologic findings were negative for malignancy, and there was no evidence of infection. The patient had no symptoms to suggest the postpericardiotomy syndrome. Seven months after bypass, a MUGA scan showed an ejection fraction of 40 percent and marked septal and apical hypokinesia. A chest roentgenogram showed persistence of the left-sided pleural effusion. Repeat thoracocentesis showed an exudative effusion without evidence of malignancy or infection. At 12 months after bypass, the left-sided pleural effusion persists with the patient receiving medical therapy similar to the first case.

DISCUSSION

Congestive heart failure is probably the most common cause of pleural effusions. Of patients with pleural effusions secondary to congestive heart failure, 88 percent have bilateral effusions, 8 percent have right-sided effusions, and only 4 percent have left-sided effusions. The effusions are typically transudates unless prior therapy with diuretics occurred, resulting in concentration of the pleural fluid protein. Other causes of pleural effusions associated with cardiac disease include constrictive pericarditis, pulmonary embolism, postpericardiotomy syndrome, postmyocardial infarction syndrome, and chylothorax following cardiothoracic procedures. Recently, delayed pleuropulmonary complications following coronary artery bypass with the IMA graft have been described.

The delayed pleuropulmonary complications described

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Figure 1. Chest roentgenogram showing chronic left-sided pleural effusion one week after thoracocentesis and 18 months following surgery (case 1).
presented between three weeks and four months from the time of surgery and included formation of a pleural effusion, fibrothorax, and respiratory failure. None of the patients had evidence of the postpericardiotomy syndrome, and all had resolution of their complications with appropriate medical and surgical therapy. The two cases presented in this report differ in that their symptomatic pleural effusions occurred shortly following surgery and persisted despite aggressive medical therapy over 20 and 12 months of follow-up, respectively. Their surgical procedures to include the pleurotomies and use of pleural and mediastinal tubes did not differ technically from the patients with the delayed pleuropulmonary complications. The two patients described in this article were similar to one another in that the pleural effusions corresponded to the side of the pleurotomy, both had evidence of significant left ventricular dysfunction, and neither had evidence of pleural disease or pleural effusion prior to their bypass surgery.

The temporal relationship of the development of the chronic pleural effusions in these patients to their bypass surgery suggests a causal relationship. The combination of a pleural defect allowing inadequate pleural fluid drainage possibly due to pleural fibrosis or a denuded chest wall requiring repleuralization and the increased pulmonary hydrostatic pressures resulting from left ventricular dysfunction probably contributed to the formation and persistence of these pleural effusions. Their exudative nature may be a result of the pleural injury, concomitant diuretic therapy, or blood or air in the pleural space.16

The association of these pleuropulmonary complications with the prior bypass surgery is often not recognized. The diagnosis of delayed pleuropulmonary complications or persistent pleural effusion due to prior pleurotomy should be one of exclusion and made only after thorough evaluation to exclude other diagnoses. Evidence of pleural fibrosis on biopsy may suggest this diagnosis. Management of the patient's congestive heart failure and periodic thoracocentesis are warranted; however, more aggressive measures to include pleurodesis may be necessary, depending on the clinical situation. The increasing use of the IMA graft with associated pleurotomy will probably increase the number of patients presenting with similar complications to their primary care physicians.

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Cholecystopleural Fistula with Cholelithiasis Presenting as a Right Pleural Effusion*

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At autopsy, multiple gallstones were recovered from the right pleural space of an elderly patient who presented with a massive right pleural effusion and septic shock. The mechanisms of gallstone migration and fistula formation between the gallbladder and right pleural space are described. Despite atypical presentations, gallbladder disease remains an important differential consideration of right pleural effusion in the elderly. (Chest 1990; 97:751-52)

Fistulae from the biliary tree to the pleura are rare and result mainly from four causes. These include thoracoabdominal trauma, parasitic liver disease, suppurative biliary tract obstruction, and percutaneous biliary drainage.1 One of the more common causes of subhepatic abscess formation is perforation of the gallbladder. These abscesses can potentially extend into the pleural space or directly into the lung forming biliopleural and bronchohilar fistulae.2 We report the first case of biliopleural fistula with gallstones and massive effusion in the right pleural space.

CASE REPORT

A 79-year-old woman presented to the emergency room with progressive shortness of breath over four days without fever, chills, sputum production, or antecedent upper respiratory infection. A vague complaint of suprapubic pain was elicited. Severe degenerative joint disease with a remote history of polio had confined her to bed for the previous year. She had a 50 pack-year smoking history.

Pertinent physical findings included an obese woman with a blood pressure of 160 mm Hg systolic by Doppler, pulse 130, and temperature of 37.5°C. The right hemithorax was dull to percussion and no breath sounds were appreciated. Deep palpation of the abdomen produced suprapubic pain, but no masses were felt. The initial hemoglobin value was 11.3 g/dl with a white blood cell count of 19,500/cu mm with 79 percent neutrophils, 7 percent band

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