Tracheal compression due to thoracic duct injury associated with thoracic vertebral fracture has been reported, but the two cases reported herein seem to be the first reported cases of mediastinal lymphocoele causing large-airway obstruction after esophagectomy.

REFERENCES


Pleural effusion should be added to the differential diagnosis of a lymphocyte-predominant exudative pleural effusion. 

(CHEST 1997; 111:1128-30)

Key words: lung rejection; lung transplantation; lymphocytosis

Pleural effusions are common in the early postoperative period after lung transplantation. New or increased pleural fluid in the 2nd to 6th week after heart-lung transplantation is common in patients with acute lung rejection; however, the characteristics of a pleural effusion associated with acute lung rejection have not been previously described. We report the characteristics of such a pleural effusion that was related to acute lung rejection.

CASE REPORT

A 38-year-old man with severe bullous lung disease secondary to sarcoidosis underwent a right single-lung transplant. He had a negative tuberculin skin test with positive controls prior to transplantation. His postoperative course was complicated by transient pulmonary edema in the allograft, which delayed extubation until the 3rd postoperative day. He also developed colonic distension postoperatively, which slowly resolved with conservative measures by the 14th postoperative day.

As the patient's abdominal distension resolved and he was able to walk farther, he began to notice dyspnea. On the 13th postoperative day, oxygen desaturation with walking was observed and worsened over the next 2 days. Spirometry (Fig 1) revealed a severe restrictive ventilatory defect. A chest radiograph (Fig 2) showed a moderate-sized right pleural effusion. A thoracentesis was performed which revealed a lymphocyte-predominant exudative pleural effusion (Table 1). Stains were negative for bacteria, mycobacteria, and fungi. Thoracoscopic inspection of the right pleural space did not reveal any abnormalities. Bronchoscopy showed an intact, well-healed bronchial anastomosis with a normal airway examination. Histologic examination of transbronchial biopsies showed minimal acute lung rejection (grade A1). Stains were negative for bacteria, mycobacteria, fungi, and cytomegalovirus.

Although the transbronchial biopsy revealed only grade A1 rejection, acute lung rejection was considered as a cause of the patient's dyspnea, pulmonary dysfunction, and right pleural effusion. However, there was concern that there might be underlying infection that would worsen if the patient were to

Pleural Effusion From Acute Lung Rejection*

Marc A. Judson, MD; John R. Handy, MD; and Steven A. Sahn, MD

A single-lung transplant recipient developed an ipsilateral pleural effusion from acute lung rejection 2 weeks after transplantation. The pleural effusion was exudative and contained more than 80% lymphocytes on two separate determinations. Acute lung

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Figure 1. Spirometry pre- and post-treatment for acute lung rejection. Treatment of acute lung rejection initiated on postoperative day 20. Single line=FVC; double line=FEV1.
receive high-dose corticosteroid therapy for the rejection. A decision was made to follow the patient closely until pleural fluid mycobacterial and fungal stains, final bacterial cultures, and initial fungal culture results were obtained; treatment with corticosteroids was planned if the pleural fluid results were negative for organisms or if his condition deteriorated prior to receiving these results. A subsequent thoracentesis (Table 1) on the 20th postoperative day again revealed a lymphocyte-predominant exudative effusion. All initial pleural and bronchoscopic stains and cultures were sterile, and the patient received 1 g of intravenous prednisolone on the 20th postoperative day, followed by 500 mg twice daily for the next 2 days. The patient noted a dramatic improvement in dyspnea within 24 h, and his spirometry test results also improved (Fig 1). Serial chest radiographs showed significant resolution of the right pleural effusion (Fig 3). All the cultures from the bronchoscopic examination and two thoracenteses were negative for mycobacteria and fungi.

**DISCUSSION**

New or increased pleural fluid in the 2nd to 6th week after heart-lung transplantation is common in patients with acute lung rejection. A study of 16 heart-lung transplant recipients demonstrated that new or increased pleural fluid and septal lines had a sensitivity of 68%, a specificity of 90%, and an accuracy of 84% for acute lung rejection. Although the appearance of pleural fluid as a predictor of acute lung rejection has been challenged, the association of effusions with acute lung rejection has been confirmed by others.

Despite controversy concerning an association between pleural effusion and acute lung rejection, pleural fluid characteristics have not been described. The pleural fluid in our patient was a lymphocyte-predominant exudate.

**Table 1—Pleural Fluid Characteristics After Lung Transplantation**

<table>
<thead>
<tr>
<th>Post-operative Day</th>
<th>9*</th>
<th>13</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red blood cells, /mm³</td>
<td>15,495±11,438</td>
<td>17,600</td>
<td>20,900</td>
</tr>
<tr>
<td>Nucleated cells, /mm³</td>
<td>637±208</td>
<td>3,095</td>
<td>8,770</td>
</tr>
<tr>
<td>Lymphocytes, %</td>
<td>33±14</td>
<td>92</td>
<td>98</td>
</tr>
<tr>
<td>Neutrophils, %</td>
<td>51±13</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>Macrophages, %</td>
<td>14±6</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>LDH, IU/L</td>
<td>846±210</td>
<td>767</td>
<td>578</td>
</tr>
<tr>
<td>Total protein, g/dL</td>
<td>1.7±0.3</td>
<td>3.7</td>
<td>4.2</td>
</tr>
<tr>
<td>Cholesterol, mg/dL</td>
<td>ND</td>
<td>ND</td>
<td>16</td>
</tr>
</tbody>
</table>

*Mean±SEM of nine single lung transplant recipients.
1Patient described in this report.
2Upper limits of normal in our laboratory is 618 IU/L.
3ND=Not done.

Although there is no information concerning the characteristics of pleural fluid 2 to 4 weeks after transplantation, we previously examined pleural fluid characteristics during the first 9 postoperative days in 9 lung transplant recipients. The nucleated cell count (637±208/mm³, mean±SEM) and total protein value (1.7±0.3 g/dL) in the pleural fluid of patients on the 9th postoperative day were lower than the two determinations made in our patient. The mean RBC count (15,495±11,438/mm³) and lactate dehydrogenase (846±210) on the 9th postoperative day were similar to those of our patient. Both the mean percentage and absolute number of lymphocytes on the 9th postoperative day in these patients were less than in

**Figure 2.** Chest radiograph performed 13 days after right lung transplantation shows a moderate-sized right pleural effusion.

**Figure 3.** Chest radiograph performed 85 days after transplantation shows residual right pleural thickening.
the present patient. Two-week surveillance bronchoscopy on these nine patients revealed normal lung (grade A0) in four and minimal lung rejection (Grade A1) in five.

The presumptive mechanism for a pleural effusion from acute lung rejection is the movement of pulmonary edema fluid along a pressure gradient from the lung interstitium to the pleural space. If fluid movement into the pleural space exceeds pleural lymphatic clearance, then a pleural effusion will be clinically apparent. Impaired lung lymphatic drainage,7,8 therefore, may play a role in the development of pleural fluid. Such effusions are probably most common when acute lung rejection occurs within 1 month of transplantation because two animal studies7,8 have demonstrated that allograft lymphatics are reconstituted and become functional between 2 and 4 weeks after transplantation.

Although a pleural effusion from acute lung rejection is a diagnosis of exclusion, we found no other cause of this exudate with greater than 90% lymphocytes. The differential diagnosis of pleural fluid exudates with greater than 80% lymphocytes includes tuberculosis, chyllothorax, lymphoma, rheumatic pleurisy, trapped lung, sarcoidosis, and yellow nail syndrome.9 Lymphoma, yellow nail syndrome, and rheumatic pleurisy can be excluded on clinical grounds. Tuberculosis was not likely given the history of a negative tuberculin skin test with positive controls, negative mycobacterial cultures, and a normal thoracicercopy examination. A normal thorascopy makes the diagnosis of malignancy and sarcoidosis unlikely. A pleural fluid triglyceride concentration of 16 mg/dL is inconsistent with a chyllothorax.10 Finally, the resolution of the pleural effusion immediately after high-dose corticosteroid treatment for acute lung rejection supports that the effusion was the result of acute lung rejection. Acute lung rejection should be added to the differential diagnosis of a lymphocyte-predominant exudative pleural effusion.

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Exercise-Induced Ventricular Arrhythmias and Sudden Cardiac Death in a Family*

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Members of a family have been investigated because of three sudden deaths among them. Two young sisters, aged 12 and 16, died suddenly while swimming and running, while their 19-year-old brother died suddenly during emotional stress. In no case did autopsies reveal any structural abnormalities. Their 39-year-old mother and her 19-year-old daughter gave a history of syncopes, while having a normal physical examination and normal ECGs. During a treadmill test, multiple ventricular extrasystoles and bursts of polymorphic ventricular tachycardia were provoked. Patient-members of this family have undergone echocardiography, catheterization of the left and right sides of the heart, endomyocardial biopsy, and electrophysiologic studies. A differential diagnosis of an inherited long QT interval syndrome, catecholamine-induced arrhythmias, and arrhythmogenic right ventricular dysplasia have been suggested. Patients were given atenolol and were followed up for 18 months. This therapy has greatly reduced the exertional arrhythmias as assessed by serial treadmill tests.

(CHEST 1997; 111:1130-33)

Key words: beta blockers; exertional ventricular arrhythmias; polymorphic ventricular tachycardia; sudden cardiac death; treadmill test

Ventricular arrhythmias can occur in a structurally normal heart. Such syndromes presenting different clinical and electrocardiographic features already have been described. One syndrome refers to patients with a prolonged QT interval with or without deafness.1,2 These individuals present with ventricular arrhythmias and torsades de pointes ventricular tachycardia related to adennergic stimulation and may sustain attacks of syncope and

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