Pleural, Alveolar and Blood T-Lymphocyte Subsets in Pleuropulmonary Sarcoidosis

To the Editor:

In two patients with pleuropulmonary sarcoidosis, T-lymphocyte subsets have been evaluated in peripheral blood, bronchoalveolar lavage and pleural fluids.

A non-smoking man, 34 years of age, with no previous medical history, was referred to our hospital for right paratracheal and bilateral hilar lymph node enlargement and a right pleural effusion detected by routine chest roentgenogram. Usual biologic test results were normal. Serum angiotensin-converting enzyme activity was 135 µmol/min/ml (n<135). Tuberculin skin test was negative. Pulmonary function tests showed a mild restrictive syndrome (VC 63 percent predicted). A fiberoptic bronchoscopy with BAL and bronchial biopsy were performed. Pathologic examination of bronchial biopsy showed typical granulomas without necrosis consistent with sarcoidosis. Thoracocentesis recovered serosanguineous fluid with protein content of 48 mg/dl; the differential cell count was 1,800 leukocytes/ml with 73 percent lymphocytes. Sputum and pleural cultures for bacterial, fungal or viral infection were negative. No treatment was prescribed and six weeks later a chest x-ray film showed resolution of the pleural effusion.

A 45-year-old man, followed since 1982 for a stage 1 sarcoidosis with uveitis and treated with corticosteroid-therapy from July, 1982 to September, 1983, was referred to us in April, 1989 for recurrence of bilateral uveitis, diffuse pulmonary infiltrates of nodular pattern and a right pleural effusion. Usual biology was normal. Serum angiotensin-converting enzyme activity was 76 µmol/min/ml. A CT scan of the chest showed subpleural nodules next to pleural thickening and pleural effusion. The pulmonary function tests showed a mixed restrictive and obstructive syndrome (TLC 65 percent, VC 73 percent, FEV, 70 percent). Fiberoptic bronchoscopy was normal and a BAL was done. Pleural thoracocentesis yielded clear yellow fluid with protein concentration of 41 mg/dl and 330 leukocytes/ml (72 percent lymphocytes). On corticosteroid therapy, pleural effusion resolved after four weeks.

The lymphocyte subpopulations from both cases in pleural and BAL fluids and blood are shown in Table 1.

In these patients, simultaneous evaluation of lymphocyte populations in pleural effusion, BAL and peripheral blood showed a marked increase in the number of CD4 T-lymphocyte subsets in pleural and BAL fluid, contrasting with a low CD4 T-cell count in peripheral blood.

Table 1—T-Lymphocyte Subsets* in Peripheral Blood, BAL and Pleural Fluid

<table>
<thead>
<tr>
<th>Peripheral blood</th>
<th>BAL fluid</th>
<th>Pleural fluid</th>
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<tbody>
<tr>
<td></td>
<td>Case 1</td>
<td>Case 2</td>
</tr>
<tr>
<td>Total cells</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>770</td>
<td>1,067</td>
</tr>
<tr>
<td>OKT4</td>
<td>188</td>
<td>181</td>
</tr>
<tr>
<td>OKT8</td>
<td>208</td>
<td>427</td>
</tr>
<tr>
<td>CD4/CD8 ratio</td>
<td>0.9</td>
<td>0.4</td>
</tr>
</tbody>
</table>

*Surface phenotyping of T-lymphocytes was determined by indirect immunofluorescence microscopy using CD4 and CD8 monoclonal antibodies (OKT4 plus OKT4 A and OKT8; Ortho Diagnostics, Raritan, NJ).

†Expressed in absolute numbers in peripheral blood and in percentage of lymphocytes in BAL and pleural fluid.

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blood. Similar results in the pleural fluid have been reported by Groman et al. However, in this report BAL cell analysis was not available. In our patients, the lymphocyte profile found in pleural effusion was closer to that seen in BAL. Lymphocyte subpopulation findings in the pleural fluid seem to be representative of the pleural histologic involvement, as BAL is of alveolitis. By contrast, in another granulomatosis, an amiodarone-induced pleuropulmonary hypersensitivity pneumonitis, cell profile in BAL was quite different from that found in blood and pleural fluid. That would indicate a pathogenetic mechanism in sarcoidosis distinct from that involved in hypersensitivity granulomatosis.

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An Unusual Cause of Pneumothorax During Percutaneous Pleural Biopsy

To the Editor:

Percutaneous pleural biopsy has been used for over 30 years as a diagnostic technique in the evaluation of pleural effusions. Pneumothorax is a well-described complication of this procedure, occurring in 3 to 15 percent of cases. The usual mechanism is that of air entry into the pleural space through the lumen of the biopsy needle or from laceration of the lung. Recently, we observed the passage of air around a closed Abrams pleural biopsy needle during manipulation of the instrument, resulting in pneumothorax.

A 72-year-old white man was admitted to our facility with increasing dyspnea, a 30-pound weight loss, and a right-sided pleural effusion. The effusion had been noted six months previously and serial thoracenteses revealed an exudative effusion; however, other studies (including cytology and tuberculous cultures and smears) were nondiagnostic. He had a prior history of pulmonary tuberculosis which was diagnosed 13 years previously and treated with isoniazid and ethambutol for 18 months. He was also eight years post-radiation therapy (4,500 rads) to the right chest for presumed curative treatment of a squamous cell carcinoma of the right upper lobe bronchus. On examination the patient was a cachetic man who weighed 114 pounds and was 68 inches tall. Diminished breath sounds and dullness to percussion was noted in the lower half of the right hemithorax. Posteroanterior, lateral, and right lateral decubitus chest radiographs demonstrated a large mobile right-sided pleural effusion. No pneumothorax was present.

We performed percutaneous pleural biopsies using an Abrams biopsy instrument. Using sterile technique, the skin and underlying parietal pleura were anesthetized with a 1 percent lidocaine solution and a skin laceration was made. The biopsy needle was inserted without difficulty and pleural fluid was withdrawn. One operator performed four biopsies, and the instrument was removed for retrieval of the specimen. The instrument was reinserted through the original puncture site without difficulty and fluid was withdrawn to confirm proper placement. With the apparatus in the closed position, the operator demonstrated a technique alleged to improve biopsy yield. This included applying lateral pressure on the instrument in the direction of the hook, as well as sharply angling it to a position approximately 30 to 45° from the plane of the back, thus placing the hook orifice of the needle in more direct contact with the pleura. During this maneuver, the patient inhaled and passage of air around the closed instrument into the pleural space produced a sucking sound which was clearly audible. The patient experienced no change in his symptoms and three additional biopsies were taken by a second operator using standard technique (lateral pressure but no angulation) without further passage of air. A chest radiograph obtained shortly after completion of the procedure revealed a 20 percent pneumothorax. The patient remained symptomatically unchanged and the pneumothorax resolved without intervention. Pleural biopsy specimens demonstrated "nonspecific chronic pleuritis." No lung tissue was found in the biopsy specimen.

This case demonstrates an unusual and previously unrecognized cause of pneumothorax: passage of air around a closed instrument during percutaneous pleural biopsy. We speculate that the combination of an enlarged hole in the pleura from previous pleural biopsies plus the virtual absence of subcutaneous fat in this cachectic patient contributed to the poor seal around the instrument at the level of the pleura and subcutaneous tissue. The combination of lateral pressure and sharp angulation of the instrument probably reduced the seal at the skin, allowing passage of air into the pleural space as the patient inhaled. We recommend, therefore, that marked angulation of the instrument during pleural biopsy be undertaken carefully, particularly when taking multiple biopsies in cachectic patients.

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Complication with a Transbronchial Histology Needle

To the Editor:

I am writing to describe a complication which occurred during use of the Wang 18-gauge transbronchial histology needle (Dual Histology Needle 18121 GA #MW-418-1, Lot 10089-5535, Mill Rose Laboratories Inc, Mentor, Ohio). I learned of its use through publication of an article in Chest by Schenk et al in August, 1989.

I found the technique relatively easy to use, and shortly thereafter made a diagnosis of adenocarcinoma of the lung metastatic to the mediastinum. I then started to do routine mediastinal core aspirates on all suspected lung carcinomas.

Recently, I began finding it difficult to pass the needle through the bronchoscope. Then I saw small, thread-like plastic material coming out of the tip of the bronchoscope as I passed the needle through it. I always passed the needle with the tip retracted, as per