Table 2—BAL Data of Six Patients and Seven Control Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Cells/ml, ×10⁶</th>
<th>Alveolar Macrophages</th>
<th>Lymphocytes</th>
<th>Neutrophils</th>
<th>Eosinophils</th>
<th>Albumin, mg/100 ml</th>
<th>IgG-Albumin Ratio</th>
<th>IgA-Albumin Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>232</td>
<td>82.0 (190.2)</td>
<td>17.5 (40.6)</td>
<td>0.5 (1.1)</td>
<td>0 (0)</td>
<td>7.45</td>
<td>0.27</td>
<td>0.10</td>
</tr>
<tr>
<td>2</td>
<td>208</td>
<td>72.5 (190.8)</td>
<td>19.5 (40.5)</td>
<td>6.5 (13.5)</td>
<td>1.5 (3.1)</td>
<td>6.24</td>
<td>0.34</td>
<td>0.10</td>
</tr>
<tr>
<td>3</td>
<td>192</td>
<td>95.5 (183.3)</td>
<td>4.0 (7.6)</td>
<td>0.5 (1.0)</td>
<td>0 (0)</td>
<td>3.15</td>
<td>0.42</td>
<td>0.10</td>
</tr>
<tr>
<td>4</td>
<td>146</td>
<td>50.0 (72.8)</td>
<td>34.0 (49.5)</td>
<td>15.0 (21.9)</td>
<td>1.0 (1.4)</td>
<td>6.89</td>
<td>0.25</td>
<td>0.12</td>
</tr>
<tr>
<td>5</td>
<td>255</td>
<td>93.0 (237.1)</td>
<td>6.5 (16.5)</td>
<td>0.5 (1.3)</td>
<td>0 (0)</td>
<td>6.89</td>
<td>0.39</td>
<td>0.11</td>
</tr>
<tr>
<td>6</td>
<td>185</td>
<td>77.5 (140.3)</td>
<td>10.0 (35.1)</td>
<td>3.5 (6.4)</td>
<td>0 (0)</td>
<td>8.90</td>
<td>0.31</td>
<td>0.10</td>
</tr>
<tr>
<td>Mean</td>
<td>203.0</td>
<td>78.4 (162.9)</td>
<td>16.7 (31.6)</td>
<td>4.4 (7.5)</td>
<td>0.4 (0.7)</td>
<td>6.58</td>
<td>0.33</td>
<td>0.10</td>
</tr>
<tr>
<td>SD</td>
<td>34.7</td>
<td>15.0 (50.5)</td>
<td>9.8 (14.7)</td>
<td>5.2 (7.8)</td>
<td>0.6 (1.1)</td>
<td>1.74</td>
<td>0.05</td>
<td>0.01</td>
</tr>
<tr>
<td>Control subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>175.7†</td>
<td>92.6±(162.4)†</td>
<td>6.8±(12.0)</td>
<td>0.5±(1.0)†</td>
<td>0†(0)</td>
<td>3.36†</td>
<td>0.26†</td>
<td>0.13†</td>
</tr>
<tr>
<td>SD</td>
<td>34.5</td>
<td>1.5 (31.4)</td>
<td>1.2 (3.0)</td>
<td>0.4 (0.9)</td>
<td>0 (0)</td>
<td>3.30</td>
<td>0.14</td>
<td>0.07</td>
</tr>
</tbody>
</table>

*Values are expressed as percentages (×10⁶).
†Difference between this value and that for six patients with EMC is not significant.
§Difference between this value and that for six patients with EMC is significant at level of p = 0.05.
‡Difference between this value and that for six patients with EMC is significant at level of p = 0.01.

Analysis of the BAL fluid (Table 2) showed an insignificant increase in the number of cells recovered. An abnormal differential count of BAL cells was noted in four of six patients. Lymphocyte alveolitis (lymphocytes ≥15 percent with or without increased percentage of neutrophils (neutrophils ≥3 percent) occurred in four patients (patients 1, 2, 4, and 6). An increased percentage of eosinophils was detected in two patients (patients 2 and 4). The alveolar macrophages were decreased in terms of percentage (p<0.05) but not absolute number.

The albumin and immunoglobulin ratios in EMC patients were not statistically different from those in the control group. No correlations were found between the BAL data and the duration of the disease.

In conclusion, these preliminary results demonstrate the presence of subclinical inflammatory interstitial involvement in EMC patients without clinical, functional, and radiologic evidence of lung disease. This interstitial cellular traffic does not seem to be related to plasma leakage.

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REFERENCES


Pleural Fluid Adenosine Deaminase and Lysozyme Levels in the Diagnosis of Tuberculosis

To the Editor:

The usefulness of adenosine deaminase (ADA) levels in the pleural fluid (PF) and the ratio between the PF and serum values (PF/S) for lysozyme (LZ) has been widely accepted in the differentiation of tuberculous from nontuberculous pleural effusion. We present the case of a 66-year-old man with malignant neoplastic pleural effusion secondary to adenocarcinoma in which the ADA level and the LZ PF/S ratio highly suggested tuberculosis pleural effusion.

All biochemical parameters were normal. Skin test with tuberculin (purified protein derivative, 5 TU) showed an induration of 22 mm. Three cytologic examinations of sputum for malignant cells and acid-fast bacilli were negative. Examination of the pleural effusion showed a clear yellow fluid with the following values: pH, 7.23; glucose, 0 mg/dL; lactate dehydrogenase, 3,300 IU/L; protein, 4.3 g/dL; amylase, 450 IU/L; cholesterol, 153 mg/dL; ADA, 48 IU/L; LZ PF/S ratio, 1.29; leukocyte count, 360/cu mm with lymphocytic predominance. Cultures in Löwenstein’s medium were negative.

Cytologic examination of PF revealed mesothelial cell plaques with hyperplastic reactive changes and small atypical cell groups, suggesting adenocarcinoma. One of three pleural biopsy specimens showed histologic changes suggestive of mesothelioma or adenocarcinoma; biopsy cultures in Löwenstein’s medium were negative.

Pleuroscopy showed whitish irregular exsiccations in both visceral and parietal pleura; histologic examination of them revealed reactive mesothelial hyperplasia together with malignant neoplastic proliferation of acinar architecture and marked nuclear atypia. The tumor was classified as an adenocarcinoma after use of immunohistochemistry techniques (periodic acid-Schiff stain, hyaluronic acid,
Alcian blue) and ultrastructural study. The primary tumor remained unknown after fiberoptic bronchoscopy, thoracic and abdominal computed tomography, and barium radiographic studies of the digestive tract. An enzyme involved in purine catabolism, ADA has been frequently reported to be an important marker in the differential diagnosis of pleural effusions of tuberculous origin. An ADA value higher than 45 IU/L in PF is highly suggestive of tuberculosis, with a sensitivity of 100 percent and a specificity of 97 percent. Nevertheless, increased values of ADA in PF have been reported in some cases of rheumatoid arthritis, systemic lupus erythematosus (SLE), malignant neoplasias, and empyema.

Lysozyme is a bacteriolytic enzyme of extensive bodily distribution, which reflects recent granuloma activity. It has been identified in granuloma epithelioid cells, activated macrophages of lymphoid nodules adjacent to tuberculous lesions, and empyema granulocytes. The LZ PF/S ratio offers a higher diagnostic yield than serum or PF LZ levels considered independently; a sensitivity of 100 percent and a specificity of 97 percent have been reported for an LZ PF/S ratio over 1.2 in pleural tuberculosis. However, pleural effusions secondary to neoplasia, SLE, and sarcoidosis can meet this criterion in isolated cases.

Fontan Bueso et al have used both biochemical parameters simultaneously (ADA in PF and LZ PF/S ratio) with sensitivity, specificity, positive predictive value, negative predictive value, and accuracy for diagnosing tuberculosis of 100 percent. Our patient's pleural effusion met both criteria, with a PF ADA value of 48 IU/L and an LZ PF/S ratio of 1.28, but this was due to adenocarcinoma of unknown origin. It must be noted, however, that values were just above the cutoff level for PF ADA and LZ PF/S ratio, near the transition zone between tuberculous and nontuberculous effusions. Nevertheless, tuberculosis had been definitively ruled out in view of the histopathologic data and Liwenstein results. All pleural biopsies were specifically reassessed in order to look for any granulomatous reaction against atypical cells at the tumor site or in the draining lymphatic ganglia. No granulomatous reaction was detected that could account for the high LZ values in the PF.

We believe that although the combined determination of the PF ADA level and the LZ PF/S ratio is easily performed in daily routine practice and is helpful in the diagnosis of tuberculosis, it is not absolutely specific, as proved in the present case. We must be very cautious about establishing a diagnosis of tuberculous pleural effusion in the absence of microbiologic or histopathologic evidence.

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REFERENCES


Peak Expiratory Flow Rates in Possible Occupational Asthma

To the Editor:

We would like to bring to the attention of readers a point concerning an article of ours, which appeared in the July 1991 issue of Chest. This point involves the calculation of the specificity of the peak expiratory flow rate interpretations. We reported this in the article with the exclusion of patients with nonoccupational asthma as being 77 percent. However, since the primary interest was in the peak flow interpretation of occupational asthma (OA), the specificity of the test should be more appropriately calculated among those without OA, that is, among those with objective diagnoses of asthma or normal combined. Thus, the specificity after excluding the indeterminates should be 90 percent (19/21), which is quite comparable to that reported by Burge et al and Cote et al, which were referred to in our article.

Gary M. Liss, M.D., Health Studies Service, Ontario Ministry of Labour, and Susan M. Tarlo, M.B., B.S., F.C.C.P., Toronto Western Hospital, University of Toronto, Toronto

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1 Liss GM, Tarlo SM. Peak expiratory flow rates in possible occupational asthma. Chest 1991; 100:63-9
2 Burge PS, O’Brien IM, Harries MG. Peak flow rate records in the diagnosis of occupational asthma due to colophony. Thorax 1979; 34:308-16

Errata

We thank Dennis M. Williams, Pharm. D., of Chapel Hill, NC, for calling to our attention two errors in the article "Acute Aortic Dissection," which appeared in the March 1991 issue (Chest 1991; 99:724-29). First, in Table 2, on page 727, under the heading "Continuous IV," the dosage for labetalol should be 0.5-2.0 mg/min, not 0.2-2.0 mg/kg/min. Second, the text on page 726 (right-hand column, lines 34-36) states that "intravenous labetalol can safely be used in doses exceeding 60 mg per 24 h with only minor adverse hemodynamic and biochemical effects." The correct dose, as reported in the study being cited, is 623 mg (range, 325 to 1,290 mg).