Roentgenographic Manifestation of Pulmonary Tuberculosis

To the Editor:

We read with interest the article by Farman and Speir in which they stated that atypical roentgenographic presentation of lung tuberculosis is still uncommon in adults. This is in contrast with other studies in which an increased frequency of unusual presentations similar to those in childhood are described (especially in older patients) and are attributed to the fact that these patients have outlived their initial infecting mycobacteria and become more susceptible to exogenous reinfection.

We reviewed the chest x-ray films from 114 adult patients with bacteriologically-proven lung tuberculosis diagnosed between 1981 and 1985. We made a subdivision between “usual postprimary tuberculosis” consisting of infiltrations (with or without cavitation) in the apicoposterior segments of the upper and/or lower lobes whether or not these were associated with other lesions, and “unusual” localizations without even minor inactive sequelae in the apicoposterior zones on tomographs (Table). We found no difference in presentation between patients younger vs older than 60 years of age (Table). Our findings were in agreement with those of Farman and Speir and Hadlock et al, but in contrast with others.

We conclude that, in our country, endogenous reactivations remain the main pathogenic pathway in the elderly. The difference between the published studies may at least partly be attributed to sometimes arbitrary differences in classification as usual or unusual form. We therefore agree with Kovnat that the importance of a uniform classification should be stressed.

P Van Den Brande, M.D.; E. De Keyser, M.D.; U. Van Walleghem, M.D.; A Cyselen, M.D., and M. Demedts, M.D., F.C.C.P., Department of Internal Medicine, Division of Pulmonary Diseases, University of Leuven, Belgium

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Lysozyme Level of Pleural Fluid

To the Editor:

Verea Hernando et al have reported that a ratio of the lysozyme level in the pleural fluid to that in the serum (PL/SL ratio) higher than 1.2 strongly suggests a diagnosis of tuberculosis when empyema has been excluded. They indicated that, at the present time, no conclusion could be drawn concerning an effusion due to a collagen disease.

Since this article appeared, we found a false-positive result in a 56-year-old woman suffering from rheumatoid arthritis for 20 years; she presented with dyspnea due to a large, right-sided pleural effusion. On thoracentesis, this proved to be a neutrophilic exudate (protein 3.64 g/dl [6.1 in the serum], LDH 2,688 U/L [230], WBC 600x10^6/L with 68 percent polynuclear neutrophils [PMN], many of them undergoing lytic changes). Typically, glycopleura was very low (3 mg/dl) and didn’t increase up to 180 min after a 25 g intravenous charge of glucose; rheumatoid factor was weakly positive; lactic acid was 10.6 mmol/L (1.1) and pH 7.03. Lysozyme determined by a turbidimetric spectrophotometric method was 29.5 mg/dl in the pleural fluid, against 9.0 in the serum (PL/SL ratio 3.3). Bacteriologic study was negative, as were three specimens taken for Ziehl stain and Lowenstein culture. Cytologic examination was negative for malignant cells, but the CEA level was high (14.4 ng/ml vs 0.4 in the serum); this prompted us to carry out thoracoscopy. Parietal pleura appeared diffusely inflamed, with a finely granulous aspect in its lower part. Several biopsy specimens showed a mild mononuclear infiltration covered by strongly hyperplastic mesothelium.

This patient thus presented with features of rheumatoid pleurisy without any sign of tuberculosis, her PL/SL ratio was 3.3, well above the cutoff point and highly suggestive of a mycobacterial origin.

In a series by Klockars and Pettersson, three of 13 patients with rheumatoid effusion also had a PL/SL ratio higher than 1.2; including our case, this represents 29 percent false positive results in this setting.

The origin of lysozyme in the pleural fluid of rheumatoid patients remains obscure; it might be explained by an extracellular release during the lysis of PMN, the mechanism involved in bacterial

Table—Radiographic Manifestations in the Age Groups Above and Below 60 Yrs

<table>
<thead>
<tr>
<th>Age Group</th>
<th>&lt;60 yrs</th>
<th>≥60 yrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 59)</td>
<td>(n = 55)</td>
<td></td>
</tr>
<tr>
<td><strong>Usual apicoposterior localisations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solitary</td>
<td>30</td>
<td>28</td>
</tr>
<tr>
<td>With pleural effusion</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>With miliary pattern</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>With lower lung field tuberculosis and/or effusion</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>Solitary unusual localisations</td>
<td>15 (25%)</td>
<td>12 (22%)</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Miliary pattern</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Lower lung field tuberculosis (± effusion)</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Tuberculoma (± effusion)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Adenopathies</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>
empyema. However, the pleural lysozyme in rheumatoid disease has been found not to be correlated with the number of PMN. There was an inverse correlation with glucose level in the pleural fluid, which rather points to the pleural membrane as the origin of the high lysozyme level; serosal pathologic changes in rheumatoid disease may indeed mimic those of tuberculosis. Whatever the mechanism, we suggest that rheumatoid disease should definitely be considered as a possible cause of high PL/SL ratio.

Other potential causes of false positive results with respect to tuberculosis might be pleural effusions associated with sarcoidosis or lupus erythematosus. As indicated by Verea Hernando et al, although a valuable indicator one may not completely rely on the PL/SL ratio for diagnosing a tuberculous effusion; the case of rheumatoid effusion is of particular concern.

Philippe Collard, M.D.;
Laurence Galanti, M.D., and
Luc Delannois, M.D., Ph.D., F.C.C.P.,
Catholic University of Louvain,
Yvoir, Belgium

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Erratum
To the Editor:

In the paper, "Early Methylprednisolone Treatment for Septic Syndrome in the Adult Respiratory Distress Syndrome," it is stated in the discussion that methylprednisolone did not impede the reversal of ARDS. This should be: methylprednisolone did impede the reversal of ARDS. This is stated correctly in the abstract, results, and correctly illustrated in the figure. However, to avoid confusion, this correction should be noted. As noted in the methods, 17 of 19 centers participating in the septic shock study participated in the retrospective collection of data on ARDS. Not mentioned in the methods but pertinent to the analysis of these data, patients with ARDS on admission were eliminated from end point analysis in the 17 centers that participated in retrospective analysis.

My errors as noted above does not affect the validity of the results or the conclusions in the study but should be corrected to avoid confusion.

Dr. James A. Kruse was a participating investigator in the Methylprednisolone Severe Sepsis Study Group. Unfortunately, his name was spelled incorrectly ("Jose" instead of "James").

Roger C. Bone, M.D., F.C.C.P.,
Rush-Presbyterian-St. Luke's Medical Center,
Chicago

REFERENCE

The Cost of Lavage Sample Interpretation
To the Editor:

In his editorial on clinical use of broncoalveolar lavage (Chest 1987; 92:71-72), Dr. Springmeyer states: "Bronchoscopic examination without lavage is an incomplete evaluation in the diagnosis of diffuse lung disease." He enumerates a number of specific tests of lavage fluid that may be diagnostically useful, including cell count and differential, lymphocyte T4:T8 ratios and cell surface markers, special stains (GMS, iron), culture and viral cultures, electron microscopy and cytology.

Lavage is a simple procedure to perform but an expensive one to interpret. Taking a hypothetical patient with undiagnosed diffuse pulmonary infiltrates, we examined the cost of performing the above analyses individually at our institution. The total laboratory charge would be $465. This is apart from the cost of biopsy interpretation or any other costs associated with the performance of bronchoscopy. We will leave aside the issue of where this cost will fall in the current era of prospective payment. We will also assume that Dr. Springmeyer does not mean that every test should be performed in every patient. Clearly, discretion should be used in ordering lavage fluid analyses, just as in any other use of the laboratory. The real question is the utility of the tests. It is not enough to state, as Dr. Springmeyer does, that lavage is useful in the diagnosis of several diffuse lung diseases. Rather, we need to decide if it is cost effective to supplement or replace transbronchial lung biopsy with broncoalveolar lavage.

In our opinion, sufficient data are not currently available to draw this conclusion. Lavage is clearly indicated in the circumstance where biopsy cannot be performed (for example, in the patient with a bleeding diathesis). Perhaps in some patients with risks for opportunistic infection lavage may supplement biopsy. This seems to be particularly true in bone marrow transplant recipients, where transbronchial lung biopsy has been shown to often be unrevealing. However, in a prospective study of the utility of lavage in diffuse lung disease, Stoller et al showed that major diagnostic changes suggested by lavage results were always confirmed by transbronchial biopsy. In the important idiopathic interstitial diseases and in many infections, it is not clear that lavage results add important diagnostic information to biopsy.

Until prospective studies are available to support their utility, we should be judicious in ordering additional expensive tests on bronchoscopic specimens.

Mark J. Schiff, M.D., and
Lucy B. Palmer, M.D.,
North Shore University Hospital,
Manhasset, New York

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