as evidenced by the very low levels of antibody found in our patients. In our study, no overlap in optical density values were seen between sarcoidosis and tuberculosis, but in a larger series overlap will probably be seen.

While it is not yet possible to discriminate between tuberculosis and other diseases with complete reliability, we would proceed to invasive modalities (transbronchial lung biopsy, mediastinoscopy or open lung biopsy with specimen culture) for diagnosing tuberculosis in any sputum-negative patient deemed—on clinical or pathologic grounds—to have sarcoidosis who has high levels of antibody to mycobacterial antigen.

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Nonspecific Pleural Effusion vs Malignant and Granulomatous Pleural Disease

To the Editor:

We have read with interest the retrospective study by Leslie et al (Chest 1988; 94:603-08) on the clinical features of patients with undiagnosed pleural effusions subjected to repeat pleural biopsies with the purpose of distinguishing those patients with nonspecific pleuritis (NPE) from malignant (MPE) or tuberculosis pleural effusions (TPE). According to their analysis, all patients having two or more of the following factors should undergo an aggressive diagnostic approach given the likelihood of underlying malignant or granulomatous disease: weight loss, fever >38°C, positive PPD, pleural fluid lymphocytosis greater than 95 percent, and a large effusion.

We have studied all patients with pleural effusions seen at our institution from October, 1987 to February, 1989. We applied the criteria proposed by Leslie et al and, in addition, we have considered another parameter—pleural adenosine-deaminase activity (ADA). Eighty-one patients (30 with TPE, 37 with MPE and 14 with NPE) were included. In general, our results are very similar to those of Leslie et al. When one criterion was present, sensitivity and specificity for granulomatous or malignant disease were 92 and 35 percent, respectively, and 62 and 71 percent when two or more criteria were present. PPD status did not have a good discriminatory power, since we found a higher percentage of positives in patients with MPE and NPE than that found by Leslie et al. This could be related to the higher prevalence of tuberculosis in our community.

Table—Correlation Between Clinical Criteria and Etiology in 81 Cases of Pleural Effusion

<table>
<thead>
<tr>
<th>Clinical Criteria</th>
<th>TPE (%)</th>
<th>MPE (%)</th>
<th>NPE (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss</td>
<td>4/29 (13.8)</td>
<td>21/56 (36.3)</td>
<td>5/14 (35.7)</td>
</tr>
<tr>
<td>Fever &gt;38°C</td>
<td>4/28 (14.3)</td>
<td>0/50</td>
<td>0/14</td>
</tr>
<tr>
<td>Positive PPD</td>
<td>27/28 (96.4)</td>
<td>3/10 (30)</td>
<td>3/10 (30)</td>
</tr>
<tr>
<td>&gt;95% lymphs in pleural fluid</td>
<td>6/26 (23)</td>
<td>3/26 (11.53)</td>
<td>1/14 (7)</td>
</tr>
<tr>
<td>Large effusion</td>
<td>12/50 (40)</td>
<td>22/57 (39)</td>
<td>3/16 (18.7)</td>
</tr>
<tr>
<td>Pathologic x-ray</td>
<td>7/30 (23.3)</td>
<td>24/37 (64.96)</td>
<td>2/14 (15.4)</td>
</tr>
<tr>
<td>ADA in pleural fluid</td>
<td>28/29 (96.5)</td>
<td>0/22</td>
<td>2/13 (14.3)</td>
</tr>
</tbody>
</table>

On the other hand, a high pleural ADA was found to be an excellent indicator for tuberculous pleurisy; with sensitivity of 96.5 percent and specificity of 94 percent, as recently reported by others. This is an easy and inexpensive enzyme determination that we think should be done in all undiagnosed pleural effusions; in our milieu, and by itself, it offers much more information in the diagnosis of TPE than the five criteria considered by Leslie et al.

Also, and differing from the work of Leslie et al, when we compared the clinical characteristics of NPE vs MPE we found that abnormal chest radiograph (without considering the calcified lesions) was significantly more frequent in MPE (64 percent) than in NPE (14 percent) (p<0.005, \(x^2=10.25\)). These differences could be the selection related to the population studied by Leslie et al. Within their NPE group, they included a significant number of patients with heart failure who had an initial pathologic chest x-ray film that subsequently normalized following diuretic treatment. It has been our experience and that of others that the presence of abnormalities of the palmonary parenchyma should lead us to suspect a malignant process, particularly if pleural ADA determination is negative; therefore, an aggressive diagnostic approach (including bronchoscopy) should be recommended.

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Accuracy of Asthma Mortality in France

To the Editor:

We read with great interest the study by Jackson et al on the trends in asthma mortality in 14 different countries. As stated by these authors, "it is clearly of considerable importance to ascertain in countries with increasing reported asthma mortality rates, whether these trends are real or due to changes in accuracy of certification or in diagnosis fashions". We wish to add some comments about the accuracy of methodology used in these epidemiologic studies reporting asthma mortality data, ie, the accuracy of death certificates. We wish to report a discrepancy between the French statistics concerning asthma deaths and our own experience, which was previously reported in this journal.

We compared the age distribution of asthmatic patients who died from asthma during the same period (1983 to 1984) as reported in the two following studies: 1) the report of asthma mortality in France given by Bousquet et al using the data of the Institut National de la Santé et de la Recherche Médicale (data also used by Jackson et al) and 2) the report of asthmatic deaths observed in a prehospital mobile emergency care unit in Paris, France.

Obviously, this comparison must be made with caution. As a matter of fact, our study was not an epidemiologic study and we failed to report all asthma deaths in Paris during this period; patients who used the other Mobile Emergency Care Unit available in Paris...