induces an alveolar reaction. The agents are capable of causing cellular activation and there may be a risk that even low exposure is not entirely harmless.

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REFERENCES

Smear Negative, Culture Positive AFB Bronchial Washings

Infection Control Implications

To the Editor:

Discontinuing isolation for a patient for whom tuberculosis (TB) is being ruled out at our university-affiliated, 600-bed teaching hospital requires three acid-fast bacilli (AFB) negative smears of either sputum, bronchial washings, or a combination thereof. Moreover, sputum specimens must be obtained on separate days. Once these criteria are met, isolation is discontinued.

Recently, our infection control department was asked to discontinue isolation on the basis of a single AFB negative bronchial washing. The rationale given was that bronchial washings are the most sensitive type of specimen and further sputum specimens would not be required. It is certainly correct that bronchial washings provide very accurate results if the whole specimen is submitted for AFB smear. In practice, however, the bronchial washings specimen is divided among several laboratories for a variety of tests, eg, cytology, microbiology, etc. Only a small amount, sometimes as little as 5 mL, is available specifically for acid-fast staining. This diluted, small-volume specimen is spun down and the pellet stained by truant auramine-rhodamine for AFB. If the patient’s specimen contains a relatively small number of mycobacteria, these may not be seen on an AFB smear prepared from such a diluted specimen.

In the past 36 months, we have had eight cases that illustrate this point very well. All eight patients had negative AFB smears from their bronchial washings, but cultures later grew mycobacteria (Table 1). Also, the time to positive growth for the mycobacteria does not differentiate nontuberculous mycobacteria from Mycobacterium tuberculosis. Most bronchial washings specimens sent to our microbiology laboratory are smear positive, culture positive, but some are smear negative, culture positive. One may presume that such patients have small inocula and are not very infectious; nevertheless, it demonstrates that bronchial washings not obtained and submitted in toto solely for AFB testing, do not always rule out mycobacterial disease. The problem is further complicated by nontuberculous mycobacteria that may cause pulmonary disease even in normal hosts.1-4 Contacts of nonisolated patients growing M tuberculosis from a negative bronchial specimen require follow-up investigation for potential exposure.5

To determine what policies other centers have in this regard, we contacted two hospitals with recognized expertise in the field, ie, the National Jewish Center for Immunology and Respiratory Diseases in Denver and the North Shore Hospital and Medical Center in Manhasset, Long Island, New York. Both institutions require three negative acid fast smears, ie, any combination of bronchial washings and sputa. If patients are unable to expectorate sputum before or after the bronchial washings, the Jewish Center induces sputum from the patient. North Shore Hospital will discontinue TB isolation after a single negative bronchial washing only if the patient’s PPD (Siebert purified protein derivative of tuberculin) is nonreactive (negative) and there are no radiologic findings consistent with a diagnosis of tuberculosis. Both institutions do not consider a single diluted negative AFB bronchial washings specimen sufficient to discontinue isolation.

Therefore, we have decided to continue our present policy of requiring three negative AFB specimens of any kind before TB isolation can be discontinued for an immune competent patient. We present this information to share our experience with others facing

<table>
<thead>
<tr>
<th>Time (d) Between Negative Smear and Growth on Culture Media</th>
<th>AFB Smears</th>
<th>AFB Cultures</th>
<th>Treatment Initiated on the Basis of Bronchoscopy Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
<td>Underlying Disease</td>
<td>Prebronchscopy Chest x-ray</td>
<td>AFB</td>
</tr>
<tr>
<td>1</td>
<td>Arthritis, CAD</td>
<td>RUL infiltrate</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>Emphysema, diabetes mellitus</td>
<td>RLL infiltrate</td>
<td>49</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>RUL infiltrate</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>End-stage renal disease</td>
<td>RUL cavitary lesion</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>Lymphoma</td>
<td>Bilateral interstitial infiltrates</td>
<td>45</td>
</tr>
<tr>
<td>6</td>
<td>NA</td>
<td>NA</td>
<td>49</td>
</tr>
<tr>
<td>7</td>
<td>Hepatitis B/IVDU</td>
<td>Bilateral alveolar infiltrates</td>
<td>17</td>
</tr>
<tr>
<td>8</td>
<td>Hypertension, CAD</td>
<td>NA</td>
<td>34</td>
</tr>
</tbody>
</table>

*CAD=coronary artery disease; RUL=right upper lobe; RLL=right lower lobe; NA=not available; IVDU=intravenous drug use.
the common and continuing problem of trying to develop a rational basis to discontinue isolation.

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That’s No Lady

To the Editor:

To reprise Henny Youngman’s famous joke, I would take exception with the designation of the woman with Mycobacterium avium complex (MAC) disease involving her lingula and right middle lobe as suffering from “Lady Windermere syndrome” (CHEST 1995; 108:1156-57). Certainly, the portrait of a demure damsels who refuses to cough because it is not genteel is highly poetic imagery.1 But, it is extremely improbable to me that volitional suppression of the powerful cough reflex could explain this pathologic complex.

Certainly, as indicated, the lingula and right middle lobe suffer from some degree of anatomical vulnerability to poor drainage, and this may well play a role in the frequent involvement of these regions with pulmonary MAC disease. Indeed, we have also observed predilection for lingular/right middle lobe MAC disease in women. However, we find that it occurs as well in those who cough with vigorous abandon.

I therefore submit an alternative hypothesis for this condition. MAC is distributed widely in the environment including potable water. Mycobacteria therefore are presumably deposited regularly in our airways, for instance, as we shower. Failure to clear the microbes selectively from the lingula/right middle lobe due to suppressed cough does not seem logical. Rather, I believe it can be more plausibly related to diminished tensile strength of the airways/air spaces in these women, probably associated with a subtle deficit of the connective tissue matrix. Evidence in support of this are associated thoracic anomalies seen among our series of women with MAC including pectus excavatum, straight-back syndrome, scoliosis, and mitral valve prolapse.2

But if there truly is an underlying disorder of the connective tis-

sue, why should there be excessive disease localized to the lingula/right middle lobe? Rather than voluntary cough suppression, I believe the answer lies with the relationship of these lung regions to the pulsating heart. Among our patients, CT scans clearly showed the earliest and most severe abnormalities to lie in the medial segment of the right middle lobe and the inferior segment of the lingula, areas juxtaposed to the ventricles (also seen with Byrd’s patient). Often before inflammatory changes are seen in these areas, high resolution CT imaging suggests rarefaction and distortion of the parenchyma in these regions. Potential mechanisms for diminished defenses might include distorted tissue organization or disrupted mucus orancy clearance secondary to the repetitive mechanical trauma.

To paraphrase Mr. Youngman, “That’s no Lady (Windermere), that’s bad fibrillin.”

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Magnesium Saga

To the Editor:

We read with interest the article by Bloch and colleagues (CHEST; 107:1576-81) about adjunct use of IV magnesium sulfate in the treatment of acute asthma. At first glimpse, the results look promising and represent a relative risk reduction of 58%, and even more reassuring, an absolute risk reduction of 0.45. The number needed to treat (NNT) to prevent one hospitalization is 2.2.1 However, we believe that at least three areas relative to methodology of the study are worthy of discussion.

First, the hypothesis of this study was to test whether IV magnesium sulfate, compared with placebo, would improve pulmonary function and decrease hospital admissions in all asthmatic patients, and not solely in severe asthmatic patients. Subgroup analysis is always problematic in such a small study. We reanalyzed their data by moving one patient from the placebo group to the magnesium group and using a two tailed t test, wherein the p value changed dramatically from an impressive 0.009 to a meager 0.055.

Second, the total number of patients in the severe group was only 35. For an event rate as high as 0.78 (hospitalizations in placebo group) at least a total of 87 patients would have been required to detect a clinically significant difference of 25% risk reduction.1

Third, our concerns are strengthened by recent examples of the LIMIT-2 trial2 that revealed significant benefit of IV magnesium in patients suspected of acute myocardial infarction. When the same therapy was tested in a considerably larger trial (ISIS-4),3 however, the beneficial effects of the LIMIT-2 were not observed. In the ISIS-4 trial, IV magnesium therapy resulted in significant untoward effects in patients suspected of acute myocardial infarction.

Considering these facts, we want to express a note of caution about use of IV magnesium sulfate as adjunct therapy in any form of asthma. Evidence of benefit at present is inconclusive. Until more