dialysis treatment. Clearly, further studies (perhaps with the addition of heparin to CLS) are indicated to determine whether catheter patency can be improved while still maintaining the high efficacy of CLS against CRB.

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References

Clinical and Therapeutic Management of Pulmonary Mycobacterium xenopi Infection in HIV-Positive Patients

Str—We read with interest the article by Kerbiriou et al. [1] in which they report treatment decisions for 20 HIV-positive patients with "significant" isolates of Mycobacterium xenopi and coexisting pathologic pulmonary conditions. HAART was initiated for all patients, but only 5 of 20 received antimycobacterial treatment. In consideration of satisfactory clinical improvement and the absence of relapses of M. xenopi infection, the authors suggest that an effective HAART-related immune reconstitution regimen is sufficient to manage M. xenopi infection.

M. xenopi is usually a nonpathogenic colonizer of airways. Most of the isolations are fortuitous, which raises concerns about their clinical significance, especially for HIV-infected patients in which concurrent pulmonary diseases are often present [2,3]. Therefore, only multiple M. xenopi isolates found in the absence of other pulmonary diseases could be considered to be "significant" isolates indicative of clinical disease [3]. The presence of coexisting pulmonary diseases should raise doubts about the role of M. xenopi as a pathogen.

From 1996 to 2001, we analyzed treatment decisions for 15 HIV-positive patients with at least 1 sputum culture positive for M. xenopi. All of the patients had pulmonary symptoms or pathologic radiological features and all had coexisting pulmonary diseases that could give reason for such symptoms (table 1). All of the patients were treated for the coexisting pulmonary diseases, which resulted in a complete resolution of symptoms and amelioration of the radiological alterations in most of the patients. None received antimycobacterial treatment.

Of the 15 patients, 4 patients were receiving stable HAART regimens; 6 patients initiated HAART during hospitalization, and 5 patients were treated only for the concomitant pulmonary diseases, delaying HAART introduction until after the resolution of clinical symptoms. No significant differences in pulmonary clinical outcomes were observed in the different groups of patients. During follow-up, sputum cultures for M. xenopi were persistently negative (table 1).

The presence of concomitant pulmonary infections associated with M. xenopi isolation represents quite an important bias for understanding the pathogenic role of M. xenopi and for the evaluation of the treatment effectiveness. Therefore, we decided not to start antimycobacterial therapy for any patient with M. xenopi isolations and concomitant pulmonary disease. On the contrary, we decided to treat only the underlying pulmonary infections and monitored the presence of M. xenopi in sputum samples. Concerning the role of HAART, we observed clinical and radiological improvements, and sputum cultures for M. xenopi promptly became negative, both for patients receiving HAART and for patients for whom HAART initiation was delayed.

In conclusion, in analyzing our treatment decisions, a different approach to the diagnosis and treatment of M. xenopi infection emerges, compared with the approach of Kerbiriou et al. [1]. Our data suggest that isolates of M. xenopi from patients with coexisting pulmonary diseases and also from patients with multiple isolates must be considered to be colonizers of the airways until different evidence emerges. We underline the importance of treating the coexisting pulmonary disease, which is probably the condition truly responsible for pulmonary symptoms. Moreover, our data suggest that timely initiation of HAART does not seem to play a determinant role in the management of M. xenopi infection in HIV-positive patients.

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References
Table 1. Characteristics of 15 HIV-infected patients from whom *Mycobacterium xenopi* was isolated and who did not receive antitubercular treatment.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Year of isolation</th>
<th>Symptoms</th>
<th>Radiological features</th>
<th>Concomitant pulmonary diseases</th>
<th>No. of isolates</th>
<th>CD4* cell count, cells/μL</th>
<th>HAART received</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1996</td>
<td>Fever, cough, breathlessness</td>
<td>Right hilar infiltrate</td>
<td><em>S. aureus</em> pneumonia</td>
<td>1</td>
<td>74</td>
<td>No</td>
<td>Relapse of bacterial pneumonia</td>
</tr>
<tr>
<td>2</td>
<td>1997</td>
<td>Fever, productive cough</td>
<td>Left basal infiltrate and pleural effusion</td>
<td>Bacterial pneumonia*</td>
<td>1</td>
<td>130</td>
<td>Yes</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>3</td>
<td>1997</td>
<td>Fever, cough, breathlessness</td>
<td>Multiple alveolar infiltrates</td>
<td><em>P. aeruginosa</em> and <em>H. influenzae</em> pneumonia</td>
<td>1</td>
<td>21</td>
<td>Yes</td>
<td>Relapse of bacterial pneumonia</td>
</tr>
<tr>
<td>4</td>
<td>1997</td>
<td>Fever, cough</td>
<td>Bilateral infiltrates and interstitial pneumonia</td>
<td>Pulmonary aspergillosis</td>
<td>1</td>
<td>85</td>
<td>No</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>5</td>
<td>1997</td>
<td>Fever, cough, chest pain</td>
<td>Left basal infiltrate</td>
<td><em>P. aeruginosa</em> pneumonia</td>
<td>1</td>
<td>17</td>
<td>Yes</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>6</td>
<td>1997</td>
<td>Fever, cough and weakness</td>
<td>Multiple alveolar infiltrates and left basal pulmonary cyst</td>
<td>Pulmonary aspergillosis</td>
<td>4</td>
<td>170</td>
<td>Yes</td>
<td>Lost to follow-up</td>
</tr>
<tr>
<td>7</td>
<td>1997</td>
<td>Fever, cough, abdominal pain</td>
<td>Interstitial pneumonia</td>
<td>*E. coli, S. aureus pneumonia and sepsis</td>
<td>1</td>
<td>68</td>
<td>Yes</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>8</td>
<td>1998</td>
<td>Fever, breathlessness</td>
<td>Left pulmonary cyst</td>
<td><em>H. influenzae</em> pneumonia</td>
<td>3</td>
<td>66</td>
<td>No</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>9</td>
<td>1999</td>
<td>Fever, dry cough, breathlessness</td>
<td>Interstitial pneumonia</td>
<td>*P. carinii pneumonia, P. aeruginosa pneumonia</td>
<td>1</td>
<td>36</td>
<td>No</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>10</td>
<td>1999</td>
<td>Fever, cough, breathlessness</td>
<td>Multiple alveolar infiltrates</td>
<td><em>P. aeruginosa</em> pneumonia</td>
<td>1</td>
<td>15</td>
<td>Yes</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>11</td>
<td>1999</td>
<td>Fever, chest pain</td>
<td>Left basal infiltrate</td>
<td>Suspected pulmonary aspergillosis</td>
<td>1</td>
<td>52</td>
<td>No</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>12</td>
<td>2001</td>
<td>Fever, cough</td>
<td>Alveolar infiltrate</td>
<td><em>H. influenzae</em> pneumonia</td>
<td>1</td>
<td>4</td>
<td>Yes</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>13</td>
<td>2001</td>
<td>Fever, weakness, breathlessness</td>
<td>Bilateral infiltrates and interstitial pneumonia</td>
<td>*P. carinii pneumonia, S. aureus sepsis, ARDS</td>
<td>1</td>
<td>13</td>
<td>Yes</td>
<td>Death</td>
</tr>
<tr>
<td>14</td>
<td>2001</td>
<td>Fever, productive cough</td>
<td>Right basal infiltrate</td>
<td>COPD, bacterial pneumonia*</td>
<td>1</td>
<td>137</td>
<td>Yes</td>
<td>Clinical improvement</td>
</tr>
<tr>
<td>15</td>
<td>2001</td>
<td>Fever, chest pain</td>
<td>Multiple alveolar infiltrates</td>
<td><em>S. aureus</em> pneumonia</td>
<td>1</td>
<td>4</td>
<td>Yes</td>
<td>Clinical improvement</td>
</tr>
</tbody>
</table>

**NOTE.** ARDS, acute respiratory distress syndrome; COPD, chronic obstructive pulmonary disease; *E. coli, Escherichia coli; H. influenzae, Haemophilis influenzae; P. carinii, Pneumocystis carinii; P. aeruginosa, Pseudomonas aeruginosa; S. aureus, Staphylococcus aureus.

* Two patients presenting with pulmonary disease were empirically treated with amoxicillin and clavulanate and had clinical and radiological improvement. Etiological pathogens were not isolated; however, the prompt response to the treatment suggests a bacterial etiology.


Financial support: Grant of the National Institute of Health (ISI), Minister of Health, projects 0AL/F and RF/101, Rome, Italy.

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Clinical Infectious Diseases 2004; 38:1642-4
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Reply

Sir—We thank Gazzola et al. [1] for their comments on our study and have looked with interest at the additional data provided. We agree that the presenting respiratory symptoms are most likely to be attributable to the coexisting pulmonary disease and that treatment of that condition takes therapeutic priority. However, there are differences between the populations described. We excluded patients who had only a single sputum culture positive for Mycobacterium xenopi, but, in their series, only 2 of the 15 cases identified had multiple isolates. It has previously been suggested that a single isolate may represent contamination or colonization [2]. What is less clear from the literature, particularly in relation to HIV infection, is the significance of isolates found repeatedly in sputum samples or of isolates from sterile sites, such as bronchoalveolar lavage fluid, as we found in our study. The current guidelines state that lifelong antimycobacterial treatment should be administered to an immunocompromised individual [2]. Our study looked specifically at whether this is necessary for patients receiving HAART. Although, at initial presentation, many of the symptoms can be attributed to the coexisting pathogens, there are many documented cases of disease developing with continuing immunodeficiency in the longer term. Some studies have suggested that coexisting pulmonary conditions increase the risk of disseminated M. xenopi disease [3]. Both our series and the series described by Gazzola et al. [1] do not demonstrate this to be the case, despite median CD4 cell counts of <50 cells/μL. However, given the chronic nature of mycobacterial disease, it is the long-term data that are essential in evaluating the need for treatment. In our series, we described a median follow-up period of 44 months, and, although outcomes are described in the series of Gazzola et al. [1], it is not documented at which time-point this was recorded. HAART started at admission to a hospital or shortly afterwards should result in immune reconstitution that may protect patients from the long-term consequences of M. xenopi infection. Gazzola et al. [1] have also not indicated at which point HAART was commenced, if not at the original admission.

In conclusion, although initial symptoms are likely to be, at least in part, due to the coexisting disease, we believe that, in the long term, HAART may reduce the progression to disease among patients from whom either isolates from a sterile site or multiple isolates of M. xenopi are recovered, without the need for the antimycobacterial treatment previously recommended.

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Clinical Infectious Diseases 2004; 38:1644
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Duration of Nontyphoidal Salmonella Carriage in Asymptomatic Adults

Financial support: Bangkok Metropolitan Administration; Division of Medical Science, Ministry of Public Health; and Sayomporn Educational Grant, Ramathibodi Foundation.

Sir—While recommendations for treatment of asymptomatic food workers who are nontyphoidal Salmonella (NTS) carriers need additional convincing evidence, there is inadequate information about duration of excretion of NTS in healthy asymptomatic adults. In a previous report, we found that 254 (98.8%) of 257 asymptomatic adult NTS excreters had eradicated their initial NTS infection at the first bacteriologic assessment (day 12 after a positive screening culture result) [1], which was a shorter time than an earlier study had reported [2]. An examination of 32 studies including a total of 2814 patients who were observed after being diagnosed with Salmonella infection showed that the median duration of NTS excretion was ~5 weeks and that the duration of convalescent-phase Salmonella carriage varied according to host factors, Salmonella serotype, the status of infection at the time of study entry, and the criteria used for determining whether Salmonella had been eradicated [2].

We had an opportunity to confirm our findings while preparing for the Asia Pacific Economic Cooperation Forum in Bangkok, Thailand, in 2003. Screening of asymptomatic adult hotel workers for NTS carriage was performed by rectal swab culture. Hygienic education about enteric infection and prevention of spreading was given to all participants at the beginning of the study. A second rectal swab