CASE REPORT

Mycobacterium celatum pulmonary infection mimicking pulmonary tuberculosis in a patient with ankylosing spondylitis

Che-Kim Tan, Chih-Cheng Lai, Chien-Hong Chou, Po-Ren Hsueh*

a Department of Intensive Care Medicine, Chi-Mei Medical Center, Yungkang, Tainan, Taiwan
b Departments of Internal Medicine, Yi-Min Hospital, Taipei, Taiwan
c Department of Internal Medicine, National Taiwan University Hospital Yun-Lin Branch, Yun-Lin, Taiwan
d Departments of Laboratory Medicine and Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan

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Introduction

Mycobacterium celatum, a slow-growing acid-fast bacillus, was proposed as a new species in 1993. It is a slow-growing acid-fast bacillus that mimics pulmonary tuberculosis in a patient with ankylosing spondylitis. The literature was reviewed and clinical features of eight HIV-negative patients with M. celatum infection are discussed. The clinical presentation of M. celatum is indistinguishable from tuberculosis, especially in patients with a previous history of pulmonary tuberculosis. Proper treatment depends on a definitive identification of this pathogen, which requires 16S rDNA sequencing or mycolic acid high performance liquid chromatography analysis.

Summary

Mycobacterium celatum, a slow-growing acid-fast bacillus, is an uncommon cause of human infection, mainly occurring in patients with AIDS. Rarely, infections restricted to the lung and lymph nodes have been reported in immunocompetent hosts. We report herein a case of M. celatum pulmonary infection that mimicked pulmonary tuberculosis in a patient with ankylosing spondylitis. The literature was reviewed and clinical features of eight HIV-negative patients with M. celatum infection are discussed. The clinical presentation of M. celatum is indistinguishable from tuberculosis, especially in patients with a previous history of pulmonary tuberculosis. Proper treatment depends on a definitive identification of this pathogen, which requires 16S rDNA sequencing or mycolic acid high performance liquid chromatography analysis.

Keywords

Mycobacterium celatum; Tuberculosis; Ankylosing spondylitis

* Corresponding author. Tel.: +886 2 23123456x65355. E-mail address: hsporen@ntu.edu.tw (P.-R. Hsueh).

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Case report

A 50-year-old man developed a cough with blood-tinged sputum, with onset 3–4 days before presentation. His medical history was unremarkable except for previous pulmonary TB...
<table>
<thead>
<tr>
<th>Case No. [Ref.]</th>
<th>Year</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Country</th>
<th>Underlying disease</th>
<th>Presentation</th>
<th>Chest radiograph</th>
<th>Clinical diagnosis</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 [6]</td>
<td>1994</td>
<td>15 months</td>
<td>M</td>
<td>Germany</td>
<td>None</td>
<td>Painless submandibular lymphadenopathy</td>
<td>Normal</td>
<td>Lymphadenitis</td>
<td>Complete excision</td>
<td>No recurrence</td>
</tr>
<tr>
<td>2 [7]</td>
<td>1998</td>
<td>73</td>
<td>F</td>
<td>Germany</td>
<td>Diabetes mellitus</td>
<td>Nonproductive cough, fever, respiratory failure</td>
<td>LUL cavity</td>
<td>Pulmonary infection</td>
<td>Cip, Cla, Pyr, Eth</td>
<td>Died after treatment for 6 weeks</td>
</tr>
<tr>
<td>3 [8]</td>
<td>2001</td>
<td>61</td>
<td>M</td>
<td>Netherlands</td>
<td>None</td>
<td>General malaise, productive cough, weight loss</td>
<td>RUL infiltrate with extensive cavities</td>
<td>Pulmonary infection</td>
<td>Eth, Cla, Rib</td>
<td>Improvement</td>
</tr>
<tr>
<td>4 [9]</td>
<td>2003</td>
<td>63</td>
<td>F</td>
<td>Italy</td>
<td>Pulmonary tuberculosis, hypertensive cardiovascular disease</td>
<td>Fever, night sweats, RUL cavity and nodular infiltration</td>
<td>Pulmonary infection</td>
<td>Inh, Eth, Cla</td>
<td>Cure</td>
<td></td>
</tr>
<tr>
<td>5 [10]</td>
<td>2003</td>
<td>79</td>
<td>M</td>
<td>Ireland</td>
<td>TB lymphadenitis, COPD</td>
<td>Dyspnea, night sweats, general malaise, weight loss, fever</td>
<td>RUL consolidation</td>
<td>Pulmonary infection</td>
<td>Rif, Eth, Cla</td>
<td>Improvement</td>
</tr>
<tr>
<td>6 [2]</td>
<td>2004</td>
<td>22 months</td>
<td>F</td>
<td>USA</td>
<td>None</td>
<td>Painless submandibular lymphadenopathy</td>
<td>Normal</td>
<td>Lymphadenitis</td>
<td>Incomplete excision + Azi Cla, Cip</td>
<td>Regression of lymphadenopathy</td>
</tr>
<tr>
<td>7 [2]</td>
<td>2004</td>
<td>37</td>
<td>F</td>
<td>USA</td>
<td>None</td>
<td>Productive cough, dyspnea</td>
<td>LLL nodular, right lung diffuse infiltrates</td>
<td>Pulmonary infection</td>
<td>Cla, Cip</td>
<td>Progress</td>
</tr>
<tr>
<td>Present case</td>
<td>2008</td>
<td>50</td>
<td>M</td>
<td>Taiwan</td>
<td>Pulmonary tuberculosis</td>
<td>Cough, hemoptysis</td>
<td>RUL infiltrates</td>
<td>Pulmonary infection</td>
<td>Cip, Cla</td>
<td>Improvement</td>
</tr>
</tbody>
</table>

Azi, azithromycin; Cip, ciprofloxacin; Cla, clarithromycin; COPD, chronic obstructive pulmonary disease; Eth, ethambutol; Inh, isoniazid; LAM, lymphangioleiomyomatosis; LLL, left lower lobe; LUL, left upper lobe; Pyr, pyrazinamide; Rib, rifabutin; Rif, rifampin; RUL, right upper lobe; TB, tuberculosis.
status after a complete course of anti-TB treatment and ankylosing spondylitis without treatment. He had not experienced fever, fatigue, anorexia, regional adenopathy, gastrointestinal symptoms, or weight changes during the course of this illness. Physical examination was unremarkable except for coarse crackles in the right lung. Laboratory examination revealed a white blood cell count of 8.14 × 10^9/L, with 62% neutrophils and 26.2% lymphocytes. Chest X-ray showed a pulmonary infiltration in the right middle lobe and a calcification plaque at the right costophrenic angle. Three consecutive sputum samples were positive for acid-fast bacilli. A reactivation of tuberculosis was assumed, and anti-TB therapy with isoniazid, rifampin, and ethambutol was started. Twenty-eight days later, culture of sputum grew Mycobacterium spp. The isolate was a slowly growing scotochromogen (producing a weak yellow pigment in the dark at 45°C) and was negative for niacin accumulation, nitrate reduction, hydrolysis of Tween 80, and arylsulfatase activity (3 days). The isolate was further identified as M. celatum by partial 16S rRNA gene (98%) analysis (accession number AV215233). These findings met the American Thoracic Society criteria for the diagnosis of nontuberculous mycobacterial disease, and indicated that M. celatum caused the pulmonary infection. Chemotherapy was changed to clarithromycin (500 mg every 12 hours) and ciprofloxacin (500 mg every 12 hours), and the patient’s symptoms and radiographic findings improved thereafter.

Discussion

M. celatum has been established as a pathogen causing infection mainly in AIDS patients, and its role in disease among other populations is rarely described. Here we have described the first reported case of isolation of this organism in Taiwan. Our review of the literature also revealed seven previously reported cases with clinical details of M. celatum infection in HIV-negative patients. The clinical features of these seven cases together with the present case are summarized in Table 1.

Patient age ranged from 13 months to 79 years and four were male. Two patients had a history of pulmonary TB and another patient had TB lymphadenitis. One patient was a lung transplant recipient. No obvious immune disorder was detected in three patients. Two children presented with painless lymphadenopathy and the other six adults had pulmonary infection. Among the six patients with pulmonary infection, cough was the most common symptom, followed by fever, night sweating, and weight loss. Upper lobes were the most commonly involved lung fields.

In summary, the presentation of M. celatum pulmonary infection, including clinical manifestations and radiographic findings, mimics pulmonary TB. However, the identification of M. celatum is difficult in routine practice, and definitive identification of the clinical isolates requires 16S rDNA sequencing or mycolic acid high performance liquid chromatography (HPLC) analysis. Therefore, the clinician should remain alert to M. celatum as a possible cause of pulmonary infection and lymphadenitis in non-HIV infected patients and the need for advanced technology for diagnostic accuracy.

Susceptibility testing of M. celatum is not standardized but the organism is generally susceptible to azithromycin, clarithromycin, and ciprofloxacin. In an animal model, clarithromycin, azithromycin, and ethambutol were shown to be the most active agents. The antimicrobial management of M. celatum infection varied among these eight reported cases. In the cases of the two children with cervical lymphadenitis, conservative neck dissection was performed in one and the other received incomplete excision and 6 months of azithromycin. Both of these children had a favorable outcome. All six patients with M. celatum pulmonary infection received combination therapy including clarithromycin and ethambutol or ciprofloxacin. Four of the six patients had clinical improvement, one patient had radiographic worsening after 3 months of treatment, and one made a temporary improvement but died 6 weeks after starting treatment. Although the case data are very limited, clarithromycin-based chemotherapy resulted in clinical improvement in most reported cases of M. celatum infection.

In conclusion, M. celatum can cause cervical adenitis in children and pulmonary infection in non-HIV infected patients. Because the clinical presentation is indistinguishable from Mycobacterium tuberculosis infection, precise identification of the causative species is imperative to avoid missing this infrequent pathogen. Macrolide-based therapy may lead to a better outcome.

Conflict of interest: No conflict of interest to declare.

References
