Abstract

Skeletal infections with atypical mycobacteria are usually a manifestation of advanced HIV disease with most patients having CD4 counts of less than 100 cells/mm$^3$. We report a case of *Mycobacterium kansasii* vertebral osteomyelitis on highly active antiretroviral therapy with a CD4 count of 320 cells/mm$^3$ and viral load below the level of detection at the onset.


Key words: *Mycobacterium kansasii*, osteomyelitis, HAART

Case Report

A 33-year-old HIV-infected male, complaining of sudden onset of back pain radiating to the right chest wall, was admitted to our hospital in August 2003. He was known to have AIDS, of which the first manifestation was systemic cryptococcosis, diagnosed in October 2000 in our hospital. His initial CD4 cell count at the time of diagnosis of AIDS was 4 cells/mm$^3$, and viral load was 69,000 copies/ml. He had been successfully treated with antifungal drugs including amphotericin B and flucytosine, followed by HAART started in January 2001. His recent medications included zidovudine 600 mg, lamivudine 300 mg, and efavirenz 600 mg, and prophylactic itraconazole 100 mg daily. His CD4 cell count was maintained >200/mm$^3$ and viral load less than 50 copies/ml for last 12 months prior to admission. He was doing relatively well except for 5 kg body weight loss over 3 months before his backache appeared.

His temperature was 38.2°C. Physical examination revealed no abnormalities. Lungs were clear to auscultation. A chest X-ray film did not show any abnormal findings. Complete blood count and biochemical data were normal except for a leukocyte count of 8.9×10$^9$/l and C-reactive protein 15.8 mg/ml. A computed tomography (CT) of the chest revealed a soft tissue density mass around the lower thoracic vertebrae, and a small amount of pleural effusion on the right side. A magnetic resonance image (MRI) of his spine showed altered signals within the 7th through the 9th thoracic vertebrae and a small amount of pleural effusion on the right side. A magnetic resonance image (MRI) of his spine showed altered signals within the 7th through the 9th thoracic vertebrae and encapsulated effusion around the affected bodies of these vertebrae, suggestive of osteomyelitis and a secondary perivertebral abscess, respectively (Fig. 1). His CD4 cell count, measured at day 2 of admission, was 320 cells/mm$^3$. Skin test with purified protein derivatives was
positive. A CT-guided puncture from the perivertebral abscess obtained purulent materials that revealed acid-fast bacilli on Ziel-Neelsen stain.

A diagnosis of mycobacterial infection, possibly tuberculosis, was rendered, and therapy was initiated with isoniazid 400 mg, rifampicin 750 mg, and pyrazinamide 1,500 mg daily. Therapy resulted in resolution of chest pain and fever. An MRI of the spine, taken 2 weeks after the initiation of therapy, showed improvement of osteomyelitis, but also revealed increments of pleural effusion, which then decreased and vanished within 1 month.

A photochromatogen was isolated from needle-aspirated material approximately 4 weeks after incubation. The isolate was confirmed as \( M. \) \textit{kansasii} by DNA prove. According to the result of its antimicrobial susceptibility, pyrazinamide was discontinued, and ethambutol at 450 mg daily was added. The patient was discharged on day 39, and is alive and well on continuation of the therapy.

**Discussion**

Skeletal infection caused by mycobacteria is a rare manifestation of HIV disease. McNaghten et al (6) reported a cumulative prevalence of 0.6% (330 out of 51,531) with osteomyelitis, mostly associated with gram-positive or -negative bacteria, in HIV-infected patients. Only one case was attributed to MAC in this study. To our knowledge, this is the first case of \( M. \) \textit{kansasii} osteomyelitis in a patient on successful HAART.

\( M. \) \textit{kansasii} is usually reported as a cause of pulmonary infection in patients with predisposing lung diseases, such as obstructive lung disease, bronchiectasis, or pneumoconiosis (7). Hematogenous dissemination has often been associated with pancytopenia and immunosuppressive therapy.

With the occurrence of the HIV epidemic, there has been an increase in the number of both pulmonary and disseminated diseases caused by \( M. \) \textit{kansasii}, which is now the second most common nontuberculous mycobacterium following MAC, in HIV-infected patients (8). Large-scale retrospective studies clearly indicate that advanced immunosuppression is associated with extrapulmonary \( M. \) \textit{kansasii} infection (2, 3, 9, 10). Mean CD4 counts at diagnosis of disseminated disease caused by \( M. \) \textit{kansasii} in HIV-infected patients are 10–28 cells/mm\(^3\) in these studies (2, 9, 10). Meanwhile, CD4 cells <40/mm\(^3\) at the time of the first AIDS defining illness is also reported to be a risk factor for later nontuberculous mycobacterial infection (11), suggesting the importance of considering infections associated with the nadir of CD4 cells, not merely the count at the onset. This finding may support the hypothesis that he was infected by the mycobacteria before the start of HAART, and gradually it proliferated over a long time.

Another explanation of the course of this case might be an inflammatory reaction against \( M. \) \textit{kansasii} after the reconstitution of his cell immunity as a result of HAART. Such phenomena were first reported in regard to MAC infections (12), and now are termed immune reconstitution syndrome. But this inference is relatively unlikely in regard to this case, because of the long period of time between the onset of his osteomyelitis and the start of HAART, and the lack of deterioration of pulmonary disease, which is usually seen in immune reconstitution syndrome with mycobacterial infections (13). This particular case clearly indicates the possibility of \( M. \) \textit{kansasii} as a causative of disseminated infection, even when a patient has undergone successful HAART for more than 2 years.

**References**


