Mycobacterium szulgai Causing Knee Abscess and Osteomyelitis in a Patient With Acquired Immunodeficiency Syndrome and Subsequent Immune Reconstitution Syndrome

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**Abstract:** Mycobacterium szulgai is a rarely reported human pathogen that causes a variety of clinical syndromes. We report a case of an M. szulgai septic arthritis and osteomyelitis in a patient with human immunodeficiency virus infection manifesting as an immune reconstitution syndrome.

*(Infect Dis Clin Pract 2006;14:392–393)*

*Mycobacterium szulgai* is a nontuberculous mycobacterium that is a relatively rare human pathogen and causes a variety of clinical syndromes. It is often associated with advanced human immunodeficiency virus (HIV) disease. Here, we report a case of *M. szulgai* causing septic arthritis and osteomyelitis of the knee in a patient with hepatitis C virus infection (HCV) and acquired immunodeficiency syndrome (AIDS) with subsequent immune reconstitution syndrome after the start of antiretroviral therapy (ART).

**CASE REPORT**

A 48-year-old man with chronic HCV and HIV infections presented to the infectious disease clinic for evaluation of pain, swelling, and stiffness in his right knee, which he reported as having persisted for more than 6 months. He was evaluated for his knee pain 4 months earlier, and a magnetic resonance imaging (MRI) at that time suggested a Brodie abscess or an osseous neoplasm. A biopsy was planned, but the patient refused, and several months later, he presented to the infectious disease clinic for management of his HIV and HCV. At that visit, the patient was afebrile but complaining of increased knee pain and difficulty in ambulating. He had not yet initiated ART, and his CD4 count was 28 cells/µL with an HIV viral load of 250,000 copies/mL (Amplicor; Roche Diagnostics, Basel, Switzerland) and an HCV viral load of more than 700,000 copies/mL (Amplicor HCV monitor 2.0; Roche Diagnostics). Physical examination revealed no erythema, warmth, or effusion of the knee, but there was a limitation in his range of motion. He continued to refuse patella biopsy. ART was initiated with lopinavir/ritonavir, tenofovir, and emtricitabine, and the patient had a good response. Six weeks later, his viral load was 89 copies/mL, and his CD4 count had climbed to 136 cells/µL. Two months later, the patient presented with right knee effusion, erythema, tenderness to palpation, and an overlying eschar that was draining yellow purulent material. His CD4 count was 134 cells/µL, and viral load was not obtained. An MRI done at that time revealed tibial edema and a large 2.2 × 1.1-cm destructive abscess within the patella extending anteriorly and posteriorly (Fig. 1). The patient underwent operative debridement and was treated with vancomycin and aztreonam. Operative specimens revealed acute and chronic inflammation on pathology, and acid-fast bacilli on culture. A sample was sent for tuberculosis polymerase chain reaction testing (Amplified *Mycobacterium tuberculosis* Direct Test; Gen-Probe, San Diego, Calif); the results of which were negative. The patient was empirically treated with clarithromycin, rifampicin, isoniazid, pyrazinamide, and ethambutol while pending pathogen confirmation, and his ART was temporarily suspended secondary to drug interactions with the empirical antibiotics. Four weeks later, mycobacterial cultures grew an acid-fast bacillus that was characterized as a chromogen, and the culture was probed for *Mycobacterium gordonae* and *Mycobacterium kansasii* (Accuprobe; Gen-Probe); both of which were negative. The patient’s antibiotic regimen was tapered to clarithromycin and ethambutol, and the previous ART regimen was restarted. The acid-fast bacillus was eventually speciated as *M. szulgai* (West Haven Veterans Administration Medical Center Reference Laboratory, West Haven, Conn), which was sensitive to clarithromycin, ethambutol, rifampicin, streptomycin, ciprofloxacin, and clarithromycin. The antibiotic regimen was tailored to these sensitivities, and the patient currently remains on clarithromycin and ethambutol with resolution of the patient's knee abscess.

**FIGURE 1.** An MRI of the patient’s knee. The arrow shows a prepatellar bursal infection extending into an intrapatellar abscess.
his symptoms after 4 months of therapy. Again, he had a good response to ART with undetectable HIV viral loads and a steadily increasing CD4 count, which is 152 cells/μL.

**DISCUSSION**

*M. szulgai* was first described in 1972 by Marks et al.\(^2\) It is a scotochromic mycobacterium (Runyon group II) that is a photochromogen at 25°C and produces pigment at 37°C. There is no known natural habitat for this mycobacterium, but there have been reports of an association with swimming pools,\(^3\) tropical fish, and aquariums.\(^4\) *M. szulgai* only accounts for less than 1% of the nontuberculous mycobacterium (NTM) isolated from humans, but when recovered, it is often associated with disease.\(^5\) It most commonly causes pulmonary disease in patients with chronic obstructive pulmonary disease or alcoholism and can be indistinguishable from pulmonary tuberculosis.\(^3,6–8\) However, it has also been reported to cause skin infections,\(^1,7,9–11\) olecranon bursitis, osteomyelitis,\(^1,7,12,13\) and disseminated disease.\(^6,14\) Most of the *M. szulgai* infections occur in the setting of immunodeficiency, and our patient had significant immunosuppression caused by his untreated HIV infection. This is the third case in the literature to our knowledge of an *M. szulgai* causing osteomyelitis and septic arthritis in a man with AIDS\(^1,15\) and the first case of *M. szulgai*-associated immune reconstitution syndrome.

Appropriate therapy is imperative in the treatment of NTM in immunodeficient patients, and proper identification is a critical step in this process. Unfortunately, the identification of *M. szulgai* can be difficult,\(^1,13\) and high-performance liquid chromatography of the mycobacterial lipids may be required for definitive diagnosis. Sequencing of the 16S ribosomal DNA may also be useful for speciation of the mycobacterium.\(^1,6\) In this case, *M. szulgai* was identified by high-performance liquid chromatography. Limited reports suggest that *M. szulgai* is susceptible to most conventional antimycobacterials, excluding pyrazamide and streptomycin. Second-line agents ciprofloxacin and rifampicin have also been reported to be effective.\(^1,12,14,15,17\) Our patient was treated with clarithromycin and ethambutol and had a good clinical response. Similar to other NTM infections, we recommend a minimum of 12 to 24 months of therapy, depending on the clinical response.\(^1,14\)

The clinical scenario was complicated by a temporal relation of the flare of knee symptoms and the introduction of ART. The patient’s symptoms worsened as his CD4 count improved in response to ART. We suspect that this patient had an *M. szulgai* knee infection when he initially presented with knee pain and an abnormal MRI but was only mildly symptomatic because of his inability to mount an effective immune response, given that his CD4 count was only 28 cells/μL. We believe that his more severe clinical manifestations, occurring approximately 3 months after initiation of ART, were caused by an immune reconstitution syndrome. It is thought that a rapid increase in the number of lymphocytes with specificity, in this case, toward mycobacterial antigens results in significant inflammation and clinical symptoms. Immune reconstitution syndrome has been commonly seen with tuberculosis and *Mycobacterium avium* complex infections but rarely with other mycobacterial infections. There are cases reported with *Mycobacterium leprae* in the skin and eyes,\(^18,19\) and *Mycobacterium xenopi* in the lungs.\(^20,21\) But to our knowledge, this is the first reported case of an immune reconstitution syndrome with *M. szulgai*. This report highlights the need for continued clinical suspicion for smoldering NTM infections in AIDS patients that can manifest clinically after the start of highly active ART.

**ACKNOWLEDGMENTS**

The authors thank Edward Cachay for his involvement with the care of this patient.

**REFERENCES**