A 32-year-old man developed a low grade fever, nonproductive cough, and a left upper lobe infiltrate with a small central cavity and mediastinal adenopathy (Fig 1) 3 months after the diagnosis of AIDS. He was found to have Mycobacterium kansasii pulmonary infection and was placed on isoniazid, rifampin, and ethambutol. His symptoms and radiographic infiltrate improved, but 1 month later he was admitted with acute hepatitis. The hepatitis resolved after the antituberculosis drugs were withheld. One year later, he presented with fever, cough, and progression of his left upper lobe infiltrate. Sputum and blood were both positive for M kansasii and his symptoms improved with rifampin and ethambutol. Six weeks later he was admitted with headache, fever, and a cough productive of purulent sputum.

**Physical Examination**


**Laboratory Findings**

White blood cell count, 4,800/µL; CD4 count, 5/µL. Chemistries: AST, 50 U/L; alkaline phosphatase, 121 U/L. Bone marrow and sputum smears: positive for acid-fast bacilli. Lumbar puncture: glucose, 43 mg/dL; protein, 50 mg/dL; WBC count, 10 cells/mm³ (all lymphocytes); India ink, negative; cryptococcal antigen titer, 1:32. Chest radiograph: bilateral infiltrates (Fig 2).

What is the etiologic agent causing the recurrent respiratory symptoms? What antimicrobial therapy would be appropriate at this time?

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Diagnosis: Multiple drug-resistant M kansasii infection in a patient with AIDS

Although M kansasii is the third most common mycobacterial species after Mycobacterium tuberculosis and Mycobacterium avium-intracellulare that causes disease in patients with HIV infection, it comprises only 0.44 percent of HIV-related mycobacterial infections in endemic areas and 0.08 percent in nonendemic areas. The rarity of M kansasii as a cause of disease in patients with AIDS has been attributed to the rarity of its isolation in nature.

The clinical presentation of M kansasii pulmonary infection resembles that of other mycobacterial species with common manifestations of fever, cough, and weight loss. Less common symptoms include chills, hemoptysis, and chest pain. The chest radiograph usually shows nodular interstitial infiltrates, although a variety of patterns have been described that include consolidation, mass-like lesions, cavities, and pleural effusions. Mediastinal lymph node enlargement, as observed on the initial chest radiograph in the present patient, is a characteristic feature of mycobacterial infections in patients with AIDS. The more common form of M kansasii infection with AIDS is disseminated infection that most frequently causes fever and weight loss. The chest radiograph in patients with disseminated disease may show the typical features associated with pulmonary mycobacterial infections or may be normal.

The diagnosis of pulmonary M kansasii infection depends on the presence of typical lung infiltrates not explained by another disease, isolation of the organism from two or more sputa, or its demonstration in tissue specimens, and response to appropriate chemotherapy. The diagnosis of disseminated disease is established by isolation of the organism from blood, bone marrow, or other body tissues or fluids; M kansasii may or may not be present in the sputum in patients with disseminated disease.

Initial antibiotic therapy of M kansasii infections should include isoniazid, rifampin, and ethambutol. The most important drug is rifampin, to which all strains have been reported to be initially sensitive. The organism is usually sensitive to isoniazid and ethambutol but is rarely sensitive to pyrazinamide. There is a paucity of reported experience in the long-term management of patients with HIV infection who develop M kansasii disease. Duration of treatment has not been defined; however, since relapse is common in AIDS patients with mycobacterial infections, it is reasonable to continue multiple drug therapy indefinitely.

Treatment failures and relapses after treatment in HIV negative patients are uniformly related to the development of resistance to rifampin. Drug sensitivities should be obtained in all patients who do not respond to therapy as expected. A recommended therapeutic regimen in patients with rifampin-resistant infections includes high doses of isoniazid (900 mg) and ethambutol (25 mg/kg) daily with the addition of sulfamethoxazole and amikacin. Prognosis with disseminated M kansasii infections in HIV positive patients is poor, related in part to the coexistence of other infections.

The present patient’s second chest radiograph (Fig 2) showed bilateral upper lobe fibronodular infiltrates with cavitation. His sputum and bone marrow cultures were positive for M kansasii, which was resistant to isoniazid and ethambutol, but sensitive to amikacin, rifampin, streptomycin, and sulfamethoxazole. He was placed on rifampin, ethambutol, intravenous amikacin, and cotrimoxazole, as well as amphotericin B for cryptococcal meningitis. His fever, headache, and cough resolved, and he was eventually sent home on fluconazole 200 mg three times daily, rifampin 600 mg daily, cotrimoxazole 160/800 mg three times daily, and intramuscular amikacin 400 mg twice weekly. Soon after leaving the hospital, the patient developed low grade fever, anorexia, and progressive fatigue. He refused further evaluation and died 6 weeks after discharge.

Clinical Pearls

1. Mycobacterium kansasii is the third most common mycobacterium species causing disease in HIV positive patients.
2. Chest radiographs show a variety of abnormalities, including nodular interstitial and alveolar infiltrates, lung masses and cavities, and hilar and mediastinal adenopathy.
3. Initial mycobacterial isolates are uniformly sensitive to rifampin, and most are sensitive to isoniazid, ethambutol, and streptomycin; the organism is rarely sensitive to pyrazinamide.
4. Organisms that develop resistance to the first-line drugs are commonly susceptible to sulfamethoxazole and amikacin.
5. Disseminated M kansasii infections have a poor prognosis due in part to the coexistence of other pathogens.

Suggested Readings


American Thoracic Society. Diagnosis and treatment of disease...
Pastores SM, Naidich DP, Aranda CP, McGuinnes G, Rom WN.

Intrathoracic adenopathy associated with pulmonary tuberculosis in patients with human immunodeficiency virus infection. Chest 1993; 103:1433-37