Disseminated Mycobacterium chelonae Infection Resulting in Endocarditis

*Mycobacterium chelonae* is being recognized as a cause of an increasing spectrum of illnesses, including soft-tissue infections, pulmonary disease, and postoperative infections. However, none of the mycobacteria have a propensity to cause infections on native cardiac valves. We report a case of disseminated infection with *M. chelonae* that involved the tricuspid valve.

A 66-year-old man with insulin-dependent diabetes, hypertension, and sick sinus syndrome (for which a pacemaker with a ventricular lead was implanted in 1988) was admitted to our institution in June 1995 for evaluation of fever and fatigue. Other than requiring several urologic procedures, he had been in stable condition until he developed migratory polyarthritides in October 1994. Two months later, he developed fevers that were typically low grade, but he had intermittent elevations in his temperature to 102°–103°F with shaking chills. An extensive evaluation ensued that included five sets of blood cultures, chest and abdominal CT, and transthoracic echocardiography. The only abnormalities were a mildly elevated serology for *Borrelia burgdorferi* (titer, 1:5, as determined by ELISA; normal, <0.08) and a positive tuberculin test with PPD (induration not recorded).

Because of concern over Lyme disease, an empirical trial of doxycycline was initiated; the patient’s temperature declined, but his fever persisted at a low grade. Findings on a transesophageal echocardiogram were normal in March 1995, but chest CT disclosed a rounded infiltrate in the right upper lobe. A biopsy specimen contained two ill-defined noncaseating granulomas; fungal and acid-fast stains and cultures were negative. The patient was treated with isoniazid, rifampin, and ethambutol for presumed pulmonary tuberculosis, without improvement in his condition. Therapy was discontinued after 1 month because of marked elevations in the results of liver function tests.

At the time of admission to the hospital in June 1995, the patient had been having intermittent fevers and profound fatigue and had lost 22 pounds. On physical examination a 2/6 systolic murmur was audible at the left sternal border. His WBC count was 6.3 × 10⁹/L with 70% polymorphonuclear leukocytes and 2% band forms; the hematocrit was 35%. A transesophageal echocardiogram showed a mobile mass on the tip of the tricuspid valve as well as a separate mass at the base of the valve leaflet in proximity to the pacemaker lead. A chest CT scan showed a slight decrease in the size of the right-upper-lobe abnormality. Wedge resection of this lesion yielded a specimen containing suppurative granulomatous inflammation with numerous acid-fast organisms. In <4 days, acid-fast organisms were seen in all three sets of ISOSTAT (Wampole Laboratories, Cranbury, NJ) blood cultures.

The patient was discharged and continued to receive empirical therapy for atypical mycobacterial infection (clarithromycin and ciprofloxacin); he was subsequently readmitted for removal of the pacemaker and its lead and for debridement of the tricuspid valve. Isolates from blood, pulmonary tissue, and the tricuspid valve were identified as *Mycobacterium chelonae* subspecies *chelonae* by the Massachusetts Department of Health laboratory; this identification was confirmed by National Jewish Hospital in Denver. The isolate proved susceptible to cefoxitin, erythromycin, and clarithromycin and tentatively susceptible to ciprofloxacin. The patient developed interstitial nephritis while receiving treatment with cefoxitin but had an excellent response to a 6-month course of clarithromycin and ciprofloxacin. He remains well 7 months later, and his blood cultures are sterile.

*M. chelonae* (formerly *M. chelonae* subspecies *chelonae*), *Mycobacterium abscessus* (formerly *M. chelonae* subspecies *abscessus*) [1] and *Mycobacterium fortuitum* are the most important human pathogens of the rapidly growing group formerly known as Runyon group IV. Although widespread in the environment, they only rarely cause a wide array of infections in immunocompetent and immunocompromised hosts; these infections typically include skin and soft-tissue infections following puncture wounds or inoculations as well as pulmonary infections, infections of foreign material (porcine and prosthetic cardiac grafts, tympanotomy tubes, intravenous and dialysis catheters); these organisms may also cause postoperative complications including sternal wound infections, infections after augmentation mammoplasty, and those following outpatient procedures [2, 3]. Although numerous reports of porcine and prosthetic valve endocarditis due to the rapidly growing mycobacteria exist, endocarditis of native valves is exceedingly uncommon. To our knowledge, only two cases of native valve endocarditis due to rapidly growing mycobacteria have been reported in the English-language literature; both occurred on abnormal valves. In one case the pathogen was identified as belonging to Runyon group IV, but it was not further identified to the species level [4]; the other case involved *M. fortuitum* endocarditis in a patient receiving chronic dialysis [5].

Most isolates of *M. chelonae* are resistant to numerous antibiotics, but with sufficient variability to require susceptibility testing in each case. Overall, clarithromycin is the drug most active against *M. chelonae*; 95% of isolates are susceptible at an MIC of ≤0.25 μg/mL and 100% at ≤1 μg/mL. Clarithromycin is 10–50 times more active than erythromycin and significantly superior to azithromycin in vitro [6]. Most isolates are resistant to cephalosporins and show variable susceptibility to aminoglycosides, doxycycline, imipenem, and ciprofloxacin. Trimethoprim-sulfamethoxazole and the newer β-lactam antibiotics have little or no activity against *M. chelonae* [7].

Combination therapy for infections due to the rapidly growing mycobacteria seems advisable, as monotherapy has been shown to select for the emergence of resistance [8]. There have been no controlled trials of therapy, the results of which could help direct

References

Reprints or correspondence: Dr. Karin Galil, Special Pathogens Section, CRDB, DBMD, NCID, CDC, Mailstop C23, 1600 Clifton Road, Atlanta, Georgia 30333.
the length of treatment, although prolonged courses of antibiotic therapy have been advocated for most infections. Minimal localized disease may respond to a 2- to 4-month course of therapy, whereas more severe infection may require >6 months of antimicrobial therapy or 3 months of antimicrobial therapy following aggressive debridement. Consideration should be given to excising the focus of infection in patients who are candidates for surgery and to removing infected foreign material [9], as there is an increasing number of reports in the literature that detail treatment failures when foreign material is involved [10].

Infections with *M. chelonae* and other rapidly growing mycobacteria can present difficulties in diagnosis. It is worth considering atypical mycobacterial infection in the evaluation of cases of indolent cutaneous and pulmonary disease, postoperative infections, and a broad spectrum of clinical syndromes— including endocarditis—in both immunocompetent and immunocompromised hosts.

**Muscle Abscess Due to Aspergillus fumigatus in a Patient with AIDS**

The incidence of invasive aspergillosis among HIV-infected patients is between 0.9% and 8.6%; this condition affects almost exclusively patients with advanced immunosuppression and CD4 cell counts of <50/mm³ [1, 2]. We report an unusual case of a large multiseptate abscess in the erector spinae and psoas muscle in a patient with AIDS.

A 41-year-old woman, who was a former iv drug user, was admitted to the hospital for evaluation of fever and pain in the right flank. HIV infection was first diagnosed 20 months before admission. At that time, she underwent evaluation for oral thrush and weight loss, and her CD4 cell count was 40/mm³. Her medical history was also significant for cholelithiasis, recurrent thrush, disseminated *Mycobacterium avium/Mycobacterium intracellulare* complex infection, and chronic active hepatitis B.

Over the previous 18 months she had been admitted to the hospital four times for treatment of recurrent pyoderma gangrenosum in the extremities. Seven months before admission, biopsy specimens from the skin lesions were negative for acid-fast bacilli, bacteria, and fungi. Her absolute neutrophil count during the last year was between 700/mm³ and 1,200/mm³. Her CD4 cell count 6 months before admission was <10/mm³.

Medications on admission included zalcitabine, clarithromycin, ethambutol, ciprofloxacin, and dapsone. The patient also had been receiving treatment with iraconazole (200 mg/d) for the past 6 months for thrush that was refractory to fluconazole therapy. She was compliant with treatment, and her thrush responded to iraconazole therapy.

The patient presented to the emergency department with progressively increasing generalized malaise and pain in the right flank; she had had these symptoms for the past 3 weeks. On admission, her oral temperature was 39°C. Tenderness at the right paralumbar area was noted; the overlying skin appeared normal. The WBC count was 3,200/mm³ with 76% segmented neutrophils. The results of urinalysis and chest roentgenography were normal. Cultures of blood and urine did not yield any growth. CT of the abdomen revealed a multiloculated, low-density collection (5 × 10 cm) located within the right erector spinae muscle and the posterior aspect of the right psoas muscle (figure 1).

Treatment with piperacillin and vancomycin was started. The patient underwent CT-guided percutaneous drainage of the abscess, and 130 mL of purulent fluid was obtained. The next day, the patient became progressively lethargic. Repeated aspiration of a persisting loculation of fluid was performed, and an additional 60 mL of purulent fluid was obtained. The aspirated fluid obtained during the