lary lymph nodes bilaterally. His WBC count was 300/µL, and his hematocrit was 21.3.

After specimens were obtained for cultures, he was treated with IV vancomycin and acyclovir. A Tzanck test and culture of a skin plaque were negative for viruses. Examination of a skin biopsy specimen revealed dermal abscesses and many gram-positive cocci in clusters. Cultures of a skin biopsy specimen and of a fine-needle aspirate from a lymph node yielded S. aureus. The results of blood cultures were negative. Culture of the same skin biopsy specimen also yielded Mycobacterium avium-Mycobacterium intracellulare, although an acid-fast smear was negative. On the third day, therapy with IV acyclovir and vancomycin was stopped, and the patient was treated with ceftriaxone (500 mg orally four times daily for 11 days) and acyclovir (400 mg orally three times daily for 5 days). The pustules healed, lymphadenopathy was no longer noted, and the skin lesions completely healed.

Lymphadenopathy occurs in more than one-half of patients with primary HIV-1 infection and usually regresses in these patients. As HIV infection progresses to AIDS, focal or generalized lymphadenopathy becomes more common but is usually associated with opportunistic infections or malignancies. Bacterial infections are common in persons with HIV infection and AIDS. Focal staphylococcal infections—especially folliculitis, impetigo, furunculosis, ecchyma, and plaques—and staphylococcal bacteremia are common in these persons [1, 2]. However, to our knowledge, we report the first case of staphylococcal lymphadenitis in a patient with AIDS. The patient’s WBC count was unusually low on admission, which may have contributed to the pathogenesis of his S. aureus infection, but S. aureus lymphadenitis is rare in neutropenic patients as well.

A mycobacterial culture of the skin biopsy specimen also yielded M. avium-M. intracellulare, but acid-fast bacilli were not detected. We consider it unlikely that M. avium-M. intracellulare was involved in the pathogenesis of his skin plaques or lymphadenitis. The skin biopsy showed abscesses, and the gram-stained material from both skin and lymph node specimens revealed neutrophils and gram-positive cocci compatible with S. aureus. The skin and lymph node lesions responded rapidly to treatment for S. aureus infection.

Staphylococcal lymphadenitis occurs occasionally in persons without AIDS or any other predisposing underlying disease. S. aureus has recently been associated with lymphocutaneous syndrome in a person with diabetes mellitus [3]. Given the frequency of other staphylococcal infections in persons infected with HIV, it is surprising that staphylococcal lymphadenitis has not been reported more frequently in this population.

Fine-needle aspiration was useful for making the diagnosis in our patient’s case. When used in cases of lymphadenopathy, it may help identify many other conditions common in patients with AIDS. Our patient, who did not have documented staphylococcal bacteremia, responded well to antistaphylococcal therapy of relatively brief duration.

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References

Disseminated Infection Due to Multidrug-Resistant Mycobacterium bovis in a Patient Who Was Seropositive for Human Immunodeficiency Virus

Most cases of tuberculosis in HIV-infected patients are caused by Mycobacterium tuberculosis, but cases of infection with Mycobacterium bovis and infection with the BCG vaccine strain of M. bovis have also been described [1, 2]. While infections due to multidrug-resistant M. tuberculosis are well known, infections due to multidrug-resistant M. bovis are extremely rare.

A 47-year-old homosexual man of Dutch origin was known to be seropositive for HIV type 1 since 1987. In April 1995, his CD4 cell count was 0.05 × 10⁹/L. In May 1995, a diagnosis of pulmonary tuberculosis was made based on a positive examination of a Ziehl-Neelsen–stained smear of sputum and a positive rRNA amplification test (MTD, Gen-Probe, San Diego) of sputum for M. tuberculosis complex. Examination of Ziehl-Neelsen–stained smears of a bone marrow aspirate and blood were negative, as were cultures of these specimens. Treatment with isoniazid (300 mg/d), rifampin (600 mg/d), pyrazinamide (2,000 mg/d), and ethambutol (1,200 mg/d) was started.

After 6 weeks of therapy, he still complained of fever and malaise. A chest roentgenogram was unchanged in comparison with the roentgenogram taken 6 weeks earlier. While examination of Ziehl-Neelsen–stained smears of sputum and feces still showed acid-fast rods, examination of a stained smear of a bone marrow aspirate now revealed acid-fast rods as well. Culture of a sputum specimen collected in May 1995 yielded positive growth on Löwenstein-Jensen medium, but the results of drug susceptibility testing were not yet known. Antituberculous therapy was continued.

Fourteen days later, a chest roentgenogram showed progression of preexisting abnormalities. Intravenous amikacin (1,000 mg/d) was added to the therapeutic regimen. However, during the next day, the patient died suddenly. At the time of autopsy, large necrotic lymph nodes were found in the mediastinum and at multiple sites in the thorax and abdomen but not in the intestines. Culture of specimens from these sites, including CSF, yielded mycobacteria that were identified as M. tuberculosis complex by means of...

Because bovine tuberculosis has been eradicated in most parts of the Western world, infections with M. bovis are thought to be due to reactivation of a latent infection. However, primary infections may still be acquired abroad or may result from contact with individuals with open tuberculosis due to reactivation of M. bovis infection [7–10]. Only 1%–3% of cases of tuberculosis in both HIV-infected and HIV-uninfected individuals in the Western world are caused by M. bovis [1, 2, 7, 9].

M. bovis is intrinsically resistant to pyrazinamide, but primary resistance to first-line antituberculous drugs is rare (2.8%–10% of the strains are resistant to isoniazid; 0–1.4%, to rifampin; and none, to both) [9, 10]. To our knowledge, only one case of infection with a multidrug-resistant strain of M. bovis has been described in an HIV-infected patient [8]. This strain was still susceptible to the new fluoroquinolones. The strain we isolated was also resistant to fluoroquinolones. It is not clear where our patient contracted his infection. This strain has never been isolated in the Netherlands before. The patient may have acquired his infection during a short stay in a hospital in Spain in January 1995. Further investigations will determine whether this is indeed the case.

References

4. van Soolingen D, Qian L, de Haas PE, et al. Predominance of a single M. bovis- specific DNA-RNA hybridization (Accuprobe, Gen-Probe). One week later, biochemical analysis identified the isolates from the sputum specimens collected before the start of treatment as M. bovis, not the BCG vaccine strain.

The species identification was confirmed by the absence of the M. tuberculosis–specific MTP-40 DNA sequence in the isolate [3]. Analysis of DNA polymorphisms in the direct repeat cluster region of strains of M. tuberculosis complex revealed M. bovis– specific deletions [4]. IS6110-based DNA fingerprinting [5] showed a two-band pattern, which was not present in the central database of fingerprints that includes 3,000 patterns and did not resemble the two known IS6110-based DNA fingerprints of the BCG vaccine strains (figure 1). In addition, the IS1081-based DNA fingerprint was not specific for the BCG vaccine strains (figure 1) [6]. The strain was resistant to isoniazid, rifampin, rifabutin, streptomycin, amikacin, pyrazinamide, ethionamide, clarithromycin, and ciprofloxacin; intermediately susceptible to ethambutol; and susceptible to cycloserine and clofazimine.

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**Safety and Efficacy of Cefprozil as Part of a Parenteral-Oral Antibiotic Regimen for the Treatment of Suppurative Skeletal Infections in Children**

For more than two decades, combined parenteral-oral antimicrobial regimens have proved safe and effective for the treatment of osteomyelitis and suppurative arthritis in children [1–6]. We conducted this study to evaluate the safety and efficacy of oral cefprozil as follow-up therapy after initial treatment with parenteral antibiotics for suppurative osteoarticular infections in children. Twenty-five children younger than 13 years of age with a clinical diagnosis of osteomyelitis or suppurative arthritis who were admitted to Children’s Medical Center or Parkland Memorial Hospital in Dallas were enrolled in the study between May 1993 and September 1994. Written informed consent for participation in the study was obtained in all cases. Laboratory studies performed at the time of admission included the following: WBC count with differential, erythrocyte sedimentation rate, C-reactive protein level, and blood culture. Orthopedic specialists were consulted for needle aspiration or surgical drainage of the affected site and for follow-up procedure(s), as clinically indicated.

Intravenous antibiotic therapy was started at the time of diagnosis and continued until there was evidence of clinical improvement and a concomitant decrease over time in the indices of inflammation, at which time oral therapy with cefprozil (90 mg/kg given daily in three divided doses) was initiated. Serum inhibitory and serum bactericidal titers of antibodies to the patient’s pathogen were determined by a standard microtiter method after the third dose of cefprozil was administered [7, 8]. The dosage of cefprozil was adjusted to achieve a serum bactericidal titer of \( \geq 1:8 \).

A patient was isolated from 12 patients. *Staphylococcus aureus* was the most common organism isolated, and no gram-negative pathogens were isolated. All isolates were susceptible to cefprozil (MIC, \( \leq 1.0 \mu g/mL \)). Five patients (42%) each had osteomyelitis and suppurative arthritis and two (17%) had both. With the exception of one patient, serum bactericidal titers were consistently \( \geq 1:8 \). That one patient required an increased dosage of cefprozil (125 mg/kg given daily in three divided doses) to attain a serum bactericidal titer of \( \geq 1:8 \), which was measured at steady state. Patients with osteomyelitis received therapy with cefprozil for a median of 24 days (range, 19–74 days), and patients with suppurative arthritis received this drug for a median of 20 days (range, 9–37 days). Cefprozil was well tolerated, and no serious side effects occurred. Two children were considered to have chronic osteomyelitis because of undrained foci of infection when treatment with cefprozil was initiated. In these two cases, cefprozil therapy was given for 10 weeks (the limit was defined by the study protocol); the clinical and inflammatory indices improved while the patients were receiving therapy. The 10 patients with acute disease were observed for 6 months or longer and, as of this writing, are doing well.

In conclusion, cefprozil is a suitable alternative for the treatment of musculoskeletal infections in children who have received an initial course of iv antibiotic therapy. Excellent tolerance and thrice-daily dosing help to assure compliance with cefprozil therapy.

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