that the efficacy of the artemisinin drugs relies more on intact splenic function than does the efficacy of other antimalarial agents; this reliance is to the point where continued parasite development during initial treatment can mimic high-grade drug resistance. There may be a case for using quinine as first-line treatment in such cases, especially if there are complications, although combination therapy (such as quinine and an artemisinin drug) may be a better alternative. In cases where such a combination regimen has to be prolonged, there appears to be little risk of artemisinin-associated toxicity.

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Primary Septic Arthritis and Osteomyelitis Due to Mycobacterium avium Complex in a Patient with AIDS

Bone and joint infections due to Mycobacterium avium complex (MAC) have been well described as a secondary manifestation of disseminated disease in patients with AIDS [1–3]. We report the case of an AIDS patient who developed primary MAC septic arthritis and osteomyelitis in the absence of any history of antecedent trauma.

A 46-year-old homosexual man whose HIV infection had been known since 1989 had a history of syphilis, hepatitis B, and recurrent perianal herpes. He presented to the hospital in September 1996 with a 3-week history of progressive left knee swelling, erythema, and pain with walking. He did not have a history of trauma to the joint or overlying skin, and he denied constitutional symptoms of fever, night sweats, or weight loss. His medications at the time of presentation included antiretroviral therapy with zidovudine and lamivudine (11-month course) and indinavir (3-month course). Physical examination revealed a swollen, warm, and mildly erythematous left knee with decreased range of motion and a detectable effusion. The rest of the physical examination was unrewarding.

A complete blood cell count at this time demonstrated a total WBC count of 4.1 × 10^9/L, a hemoglobin level of 12.7 g/dL, and a platelet count of 115 × 10^9/L. Electrolyte levels and liver function tests were within normal limits. In July 1996, the patient’s CD4 cell count was 8 × 10^9/L, and an HIV PCR assay demonstrated 292,000 copies/mL. A tuberculin skin test was nonreactive. A radiograph of the joint showed prepatellar soft tissue swelling and a lytic lesion of the lateral tibial plateau (figure 1). An MRI scan confirmed a destructive abnormality of the tibial plateau extending to the soft tissue that was consistent with an abscess.

After an unsuccessful attempt at needle aspiration of the joint, an incisional biopsy and drainage of the lesion were performed; these procedures revealed that bone was replaced with a yellow fatty material and a fluid collection. Microscopic examination of the bone specimen demonstrated granulomatous osteomyelitis. Kinyoun staining of the joint fluid and tissue revealed many acid-fast organisms. Subsequent cultures yielded MAC. No other fungi or bacteria were recovered, and blood and sputum cultures obtained at this time were negative for mycobacteria.

Treatment with isoniazid, rifampin, ethambutol, and pyrazinamide was begun; this treatment was changed to rifabutin, clarithromycin, and ethambutol when MAC was identified from the culture of the joint fluid. Although the swelling and pain initially subsided, a fistulous tract with chronic drainage developed at the operative site. In November 1996 a second operative debridement was performed that resulted in closure of the wound and resolution of drainage. A repeated CD4 cell count was 98 × 10^9/L, and HIV-6 DNA assay revealed 800 copies/mL. The knee pain and swelling slowly subsided over several months with chemotherapy.

Septic arthritis due to MAC has been reported in four patients with AIDS. Disseminated MAC disease was documented for three of the four patients whose cases were reported [1–3]. The single patient with a presumed primary infection had sustained a remote penetrating injury to the joint, and thus the subsequent septic arthritis may have represented a flare of chronic infection in the context of waning immunity [4]. Development of MAC arthritis years after

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such a penetrating trauma has been well described in immunocompetent hosts, most notably in a 43-year-old man who developed septic arthritis due to MAC 35 years after a similar injury [5]. Our patient did not have disseminated disease or a history of trauma to the site of infection, thus clearly defining this infection as primary septic arthritis.

The presence of a vigorous local immune response with the formulation of granulomas despite the marked immunodeficiency has been noted previously [2]. It has been speculated that this response may represent recruitment of CD3\(^+\), CD4\(^+\), and Thy1\(^+\) cells, which are relatively preserved in patients with advanced HIV infection [2]. An alternate explanation in our patient’s case is that the recently introduced antiretroviral regime had induced enough viral suppression to allow a degree of immune reconstitution. This hypothesis is supported by the increase in CD4 cell count and the decrease in the viral load seen in November 1996.

This case illustrates that in the growing spectrum of MAC disease in the AIDS patient, septic arthritis can present in the context of disseminated disease, as a de novo infection or after local injury with inoculation. Since a response to chemotherapy was documented in all reported cases of arthritis due to MAC [1–5], MAC should be considered early in the differential diagnosis of subacute arthritides in patients with AIDS even in the absence of systemic mycobacterial disease.

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