CONCISE COMMUNICATIONS

Mycobacterium kansasii septic arthritis in a patient with acquired immune deficiency syndrome

Mycobacterium kansasii septic arthritis is rare, occurring mainly in joints with preexisting abnormalities or in an immunocompromised host (1,2). Infection may arise from hematogenous spread (i.e., from the lungs) or from local trauma, but some cases have no identifiable source. While M kansasii causes several infectious syndromes in patients with acquired immune deficiency syndrome (AIDS), M kansasii septic arthritis in an AIDS patient has not previously been reported. We recently treated a patient infected with the human immunodeficiency virus (HIV) who developed a septic monarthrits of the elbow due to M kansasii.

The patient was a 28-year-old HIV-positive man who was admitted to University of Michigan Hospital with pain and swelling of the right elbow (pain of 1 month's duration, swelling of 4 days' duration). He denied recent fever, cough, dyspnea, dysuria, penile discharge, ocular inflammation, skin eruption, or trauma to the right arm. Over the last year he had lost 40 pounds and had experienced chronic diarrhea, nausea, and night sweats. His HIV infection had been confirmed by enzyme-linked immunosorbent assay and by Western blot (HIV and p24 antigen). He had been treated for Entamoeba histolytica diarrhea, tinea cruris, tinea corporis, chronic oral candidiasis, and recurrent cutaneous herpes simplex. At admission, he was being treated with acyclovir, cimetidine, clotrimazole troches, cephalexin (taken for 48 hours prior to admission without improvement), and pentamidine (monthly, by aerosol).

His physical examination was notable for an asthenic appearance, but fever, skin lesions, and abnormal breath sounds were absent. His right elbow was warm, swollen, markedly tender, and held at 45° flexion, permitting only 5° of further motion. Findings of peripheral blood studies included leukopenia (3,500 white blood cells [WBC]/mm³ with 64% polymorphonuclear cells, 19% bands, 10% lymphocytes, 6% monocytes, and 1% eosinophils), absolute CD4+ lymphopenia (12 cells/mm³, with a CD4:CD8 ratio of 0.03), and a rapid erythrocyte sedimentation rate (75 mm/hour; Westergren method). Radiographic studies revealed normal findings in the chest and no abnormalities, other than soft tissue swelling, in the right elbow. Arthrocentesis yielded 12 cc of synovial fluid classified as type II (6,450 WBC/mm³: 91% polymorphonuclear cells, 2% lymphocytes, 7% histiocytes) that contained no crystals seen on polarized light microscopy and no organisms seen by Gram’s, acid-fast, or KOH stains.

Initial treatment with intravenous cefazidime and vancomycin for presumed bacterial septic arthritis was changed on the sixteenth hospital day to isoniazid, rifampin, and ethambutol when acid-fast bacilli (nontuberculous, photochromogenic)—later identified as M kansasii—were identified in cultures of the initial synovial fluid (and later from the subsequent arthrocenteses). Findings from urine culture were negative. His symptoms improved significantly after 2 weeks of antitubercular treatment, and repeat radiography of the right elbow showed reduced soft tissue swelling. The patient moved to another state where he continued antitubercular therapy and noted resolution of the elbow symptoms; however, he contracted several opportunistic infections and died 9 months after the presentation with septic arthritis.

M kansasii is a photochromogenic atypical mycobacterium (Runyon group 1) that can occur as a disseminated process (3), but often appears as a local infection, affecting the lungs (4), genitourinary tract (5), skin (6), and bones, joints, or tendon sheaths (7). Predisposing factors for M kansasii infection are not well established, although many reported cases have involved an immunocompromised host. Hence, it is surprising that M kansasii septic arthritis in an AIDS patient had thus far not been reported. A recent history of local trauma (including arthrocentesis or joint injection) has frequently been associated with M kansasii septic arthritis and/or tenosynovitis. Preexisting joint disease can serve as a predisposing factor in some cases, and may lead to delayed diagnosis when indolent progression of symptoms in a chronically abnormal joint is erroneously ascribed to progression of underlying joint disease. However, in a number of reported cases, there was no apparent predisposing condition.

Septic monarthrits and tenosynovitis are observed most often, though polyarthritis and osteomyelitis have also been reported (1,2). The outcome of M kansasii septic arthritis is variable, possibly related in part to the presence or absence of underlying rheumatic disease or alterations in immune function. The majority of previously healthy patients recover fully; bone and joint destruction are more commonly observed in patients with underlying disease. Medical therapy—consisting of combination chemotherapy with isoniazid, rifampin, and at least one additional antitubercular agent—is usually effective, although surgical debridement of infected tissue is required in cases in which there is lack of response to a trial of pharmacologic therapy. Response to chemotherapy is often good despite in vitro resistance of the organism to one or more of the agents used (1,2).

Mycobacterial disease is an important cause of morbidity and mortality in patients with HIV infection (7). Disease due to Mycobacterium tuberculosis may occur at any stage of AIDS (7). Mycobacterium avium complex infection is the most common nontuberculous mycobacterial pathogen in AIDS patients, and is well recognized as a late-stage, poorly responsive manifestation. M kansasii is the second most common nontuberculous pathogen in AIDS and usually occurs as a pulmonary process, which generally responds well to antitubercular chemotherapy (8). Reported M kansasii bone and joint involvement in AIDS patients has been limited to 2 case reports of osteomyelitis (9,10) and 1 case of osteomyelitis mentioned in a report of a record review of a series of AIDS patients with M kansasii infection in AIDS (8). In fact, only 2 cases of septic arthritis due to mycobacteria in HIV-infected patients have been reported, both involving M avium complex (11,12).

Septic arthritis is less common than many of the other musculoskeletal syndromes reported to occur in HIV-infected patients (13). Although an occasional report has suggested that septic arthritis in an AIDS patient may present in an occult manner and follow an indolent course.
Sterile oily abscess from depot gold therapy

Two men of asthenic habitus with seropositive rheumatoid disease requested that their gold injection (aurthiogluconate [ATG] in sesame oil; Solganal; Schering, Kenilworth, NJ) be given in the arm. After complete response, each patient opted for no maintenance injections, and subsequently, they experienced a relapse of symptoms.

During the third course of ATG treatment, at a cumulative dose of 10,125 mg, the ATG was stopped in patient 1 because of a lack of response. Five months after the injections were stopped, the patient experienced pain, warmth, and swelling in the deltoid region. Similar symptoms developed in patient 2, who had an incomplete response during the second course of treatment, at a cumulative dose of 10,345 mg of ATG.

Surgical exploration of the deltoid region in each patient demonstrated a sterile abscess, with oily yellow-brown material. Histologic features of the skeletal muscle were similar: walls of a thick layer of histiocytes, including giant cells, interrupted by various-sized vacuoles; golden-brown pigment. Polarizing light microscopy demonstrated numerous doubly refractile bodies.

To determine whether there were adverse effects of ATG at the recommended intragluteal injection sites, 7 consecutive patients on long-term regimens of ATG injections were screened using ultrasound. None of these patients had symptoms or palpable changes. Ultrasound defects were identified in the 2 patients who had received virtually all ATG injections on one side. One patient (cumulative dose 9,035 mg of ATG) had 2 hypoechoic areas, measuring 1.3 cm and 2.4 cm; the other patient (10,300 mg of ATG) had several hypoechoic areas, measuring <1 cm each. No defined hypoechoic areas were noted in the 5 patients who had received their injections on alternate sides (cumulative doses 5,410, 9,230, 10,730, 11,340, and 17,055 mg of ATG, respectively).

Sesame oil–based ATG has long been the only formulation available. Its benefits are a rarity of vasomotor nitritoid reactions, even with high doses, and longer intervals between injections once response is achieved (1). A computer literature search to 1966 yielded no reports of sterile abscesses from injections of ATG in oil. Comroe had stated that "a short period of massage . . . will usually prevent the formation of hard nodules . . . " (2) at the injection site. Sterile abscesses were rarely seen (Hollander JL: personal communication).

In a relevant study of 109 psychiatric patients who were receiving depot neuroleptics (3), 4 patients had sympotomatic oily abscesses which required surgical drainage. These patients were receiving perphenazine enanthate in sesame oil, in a large volume (8-11 ml once weekly). No lesions were palpable in patients who were receiving smaller volumes less frequently.

Initial trials of oil adjuvant influenza vaccine demonstrated a high rate of sterile abscesses (4). Subsequent studies in monkeys showed that oil globules, granulomas, and giant cells were present at the injection site of any preparation containing oil. The breast muscle of a 21-day-old chicken was shown to contain fat globules, but no tissue reaction was seen.

In studies of pigs given injections of an antibiotic in