Remained afebrile throughout his hospitalizations. Physical examination revealed a cushingoid appearance; fragile, parchment-like Flavobacterium odoratum

The patient's WBC count was 19,000/mm³ with 85% neutrophils, 8% band forms, 4% monocytes, and 3% lymphocytes; toxic granulation was present.

A 63-year-old man with severe chronic obstructive pulmonary disease who had been receiving maintenance therapy with prednisone (30 mg/d) for 3 years was admitted to the hospital with bilateral upper extremity cellulitis that had developed during the previous week. There was no history of trauma, use of antibiotics, swimming, or contact with animals within the previous year. Water for drinking and bathing at his home was obtained from a well.

After 10 days of intravenous piperacillin therapy, the patient was discharged and instructed to continue treatment with oral trimethoprim-sulfamethoxazole (160 mg/800 mg b.i.d.) for an additional 2 weeks.

References
home failed to yield *F. odoratum*, it remains tempting to speculate that his well water was the source of his infection.

Resistance to antibiotics is characteristic of *F. odoratum*. The bacterium is invariably resistant to aminoglycoside antibiotics and is usually susceptible to trimethoprim-sulfamethoxazole. Resistance to penicillins, extended-spectrum penicillins, cephalosporins, aztreonam, and imipenem is common but variable [2]. Of note, our patient’s cellulitis did not begin to respond to therapy until antibiotics demonstrating inhibitory activity in vitro were administered.

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### Polyarthritis Caused by *Leishmania* in a Patient with AIDS

Severely immunocompromised HIV-infected patients are prone to unusual clinical manifestations of visceral leishmaniasis and treatment-resistant visceral leishmaniasis [1–6]. We describe a patient with AIDS and polyarthritis due to *Leishmania*.

A 32-year-old HIV-positive man with a history of intravenous drug use and disseminated tuberculosis complained of tenderness and stiffness in both hands 3 years after his diagnosis of HIV infection. His wrists and some proximal and distal interphalangeal joints were hot and swollen. Laboratory studies disclosed the following: WBC count, 4,700/µL (CD4+ lymphocyte count, 28/µL); erythrocyte sedimentation rate, 80 mm/h; and C-reactive protein level, 81 µg/mL. Titers of antinuclear antibodies and rheumatoid factor were negative. A roentgenogram of the hands was unremarkable. An isotopic study showed foci with trace uptake. Naproxen therapy was started.

One month later, a 3-cm, elastic, nonadhesive, painless right axillary node developed. Examination of Giemsa-stained preparations of node material and bone marrow revealed *Leishmania* amastigotes. Scant synovial fluid was obtained from the right wrist, and examination of smears of this fluid demonstrated *Leishmania*. Therapy with meglumine antimoniate (20 mg/(kg·d)) was given for 3 weeks. The parasite level in the node decreased, and there was some clinical improvement in the swollen joints.

Four months later, he presented again because of fever (temperature, 38°C) and polyarthralgia. Cachexia, thrush, a right axillary node, and hepatosplenomegaly were noted. Joints of the hands, elbows, and ankles were swollen, tender, and painful. Laboratory studies disclosed results that were similar to those previously found. Bone roentgenograms were unremarkable, but scintigraphy showed trace uptake in multiple joints: carpus (figure 1), elbows, shoulders, knees, ankles, tarsus, and metatarsophalangeal joints. Recurrence of leishmaniasis was demonstrated in the bone marrow, right axillary node, and right ankle. Therapy with meglumine antimoniate was reinitiated, and allopurinol was added to the treatment regimen; however, the patient died suddenly on the 10th day of hospitalization. A necropsy was not allowed.

Leishmaniasis is endemic in the Mediterranean area. In an HIV-positive patient, it may be caused by reactivation or new infection due to *Leishmania donovani*. The clinical spectrum of visceral leishmaniasis can range from an asymptomatic form to a progressive form leading to death. In severely immunocompromised HIV-infected patients, the parasite may be found in many organs. Involvement of peripheral blood, the respiratory tract, and the digestive tract is frequently described [2–6]. The control of leishmanial infection relies on the ability of T lymphocytes to produce lymphokines that activate macrophages to kill protozoa. Deep cellular immunosuppression and lack of macrophage activation explain the characteristics of HIV-associated visceral leishmaniasis: dissemination of parasites throughout the body, frequent relapses (40%–50% of cases), and a progressive course [1, 6].

Parasitic arthritis or reactive arthritis due to parasites occurs infrequently [7]. To our knowledge, no cases of arthritis due to *Leishmania* species have been previously reported. Our patient complained of subacute symmetrical polyarthritis affecting his wrists and ankles. Although it seemed to be reactive arthritis, the parasite was identified in two smears of synovial fluid, and the characteristics of the fluid could not be analyzed. There was little clinical improvement after administration of naproxen and meglumine antimoniate.

Subacute polyarthritis may be a new clinical manifestation of leishmaniasis in patients with AIDS; the number of these uncommon manifestations is increasing. The treatment of and secondary prophylaxis for leishmanial infection need to be established; therefore, trials of new drugs are encouraged.

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