definitive diagnosis in suspected cases. With the advent of aggressive chemotherapeutic regimens for the treatment of LGLM, it is likely that additional cases of atypical PCP may be seen. PCP must be considered in the differential diagnosis of pneumonia syndromes in chemotherapy-treated patients with LGLM, even when bronchoscopy is nondiagnostic. Open lung biopsy remains an important diagnostic procedure for these patients.

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Recurrent Cellulitis and Bacteremia Caused by Flavobacterium odoratum

Flavobacterium odoratum is an aerobic gram-negative bacterium that is common in environmental freshwater sources but is rarely isolated from clinical specimens [1, 2]. We describe a case of recurrent cellulitis and bacteremia due to F. odoratum.

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.

On admission, the patient’s temperature was 37.4°C, and he remained afebrile throughout his hospitalizations. Physical examination revealed a cushingoid appearance; fragile, parchment-like skin on the extremities, with multiple superficial ecchymoses and excoriations; erythema, tenderness, warmth, and edema of most of the right arm from wrist to mid-biceps; and warmth and edema of the entire left forearm. Axillary adenopathy was not detected.

On the day of admission, the patient’s WBC count was 19,000/mm³ with 85% neutrophils, 8% band forms, 4% monocytes, and 3% lymphocytes; toxic granulation was present.

Intravenous cefazolin therapy was begun. On the second hospital day, the cellulitis was unchanged; blood cultures (Bectec; Becton Dickinson Instrument Systems, Sparks, MD) were positive and yielded an organism that was eventually identified as F. odoratum [1]. In both disk diffusion and broth microdilution assays [3, 4], the isolate proved to be resistant to vancomycin, gentamicin, amikacin, cefazolin, and ceftazidime and was immediately susceptible to ceftriaxone. It was susceptible to trimethoprim-sulfamethoxazole, piperacillin, and imipenem. When these results became available, the patient’s therapy was switched to intravenous imipenem/cilastatin (500 mg q6h). The cellulitis abated within 24 hours of this change in therapy, and it continued to diminish. Following 10 days of intravenous 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.

Four days after completing antimicrobial therapy, the patient returned to the hospital with recurrent cellulitis of his left upper extremity. Once again, he was afebrile and did not appear toxic. However, blood cultures were again positive for F. odoratum, with an identical antibiotic susceptibility pattern. Following 10 days of intravenous piperacillin therapy (5 g q6h), the patient was discharged with instructions to take trimethoprim-sulfamethoxazole (160 mg/800 mg b.i.d.) for an additional month. When the patient was seen for a follow-up examination a few months later, his cellulitis had resolved. A sample of the patient’s home tap water, which was obtained during the second hospitalization, failed to yield F. odoratum.

As of 1979, only 24 cases involving isolation of F. odoratum from clinical specimens had been reported; infection was suspected in five of these cases [2]. Urinary tract infection was present in three cases, and cutaneous infection was present in two. Both skin infections affected immunologically compromised sites—an amputation stump of a patient with peripheral vascular disease and a gangrenous frostbitten foot of the other patient. Only one other report mentions isolation of F. odoratum from cutaneous sites—a single case in which the organism was described as a colonizer of a gangrenous amputation stump [5].

In our patient’s case, F. odoratum infection also involved a cutaneous site with impaired defenses. The portal of entry was likely the area of cutaneous breakdown on the upper extremities, and chronic corticosteroid therapy was the predisposing factor. The findings in our case further illustrate the invasive potential of this opportunistic pathogen, which spread from the cutaneous site of origin to the bloodstream on two occasions. Although tap water from the patient’s
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 tuberculous 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 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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