vant history of disease or exposure, except for “idiopathic eczema” that had been topically treated with steroids. He was not taking any systemic drug. The findings of a general physical examination were normal. Laboratory evaluation revealed the following findings: WBC count, 3.7 × 10^6 cells/L (0.85 × 10^6 absolute lymphocytes/L); hemoglobin level, 13 g/dL; and platelet count, 159 × 10^9 platelets/L. Chest radiographs were normal. Thoracic and abdominal CT demonstrated a moderately enlarged liver and spleen, but significant lymphadenopathies were not found. Diverse microbiologic tests all had negative results. Serological testing was negative for HIV. A bone marrow biopsy demonstrated numerous free and intramacrophage *Leishmania* species. Intravenous liposomal amphotericin B was administered at a dosage of 5 mg/kg on days 0, 1, 2, 3, 4, 10, and 21. Fever disappeared 24 h after the first dose was administered. The general performance of the patient improved in the next week, and he was discharged from the hospital. Eight months later, he was admitted to the hospital again with a 4-week history of fever and chills. He related that his current symptoms were similar to those he had experienced at the time of the first hospital admission. The results of serological testing and viral load measurements for HIV were negative. *Leishmania* species were found in bone marrow. Intravenous liposomal amphotericin B was administered again. A more complete series of immunological tests demonstrated normal levels of immunoglobulins (IgG, IgA, and IgM) and complement factors (C3 and C4). The patient’s CD4+ T lymphocyte count was 134 cells/mm^3 (9% of total lymphocytes). Six months later, the patient was asymptomatic for leishmaniasis, and a new CD4+ T lymphocyte count revealed 127 cells/mm^3 (8% of total lymphocytes). Serological test results were once again negative for HIV. A diagnosis of idiopathic CD4+ T lymphocytopenia was made. One year later, the patient’s CD4+ T lymphocyte count was 61 cells/mm^3 (6% of total lymphocytes), and serological test results and viral load measurements were negative for HIV once again.

Idiopathic CD4+ T lymphocytopenia was described in 1992. The following criteria were proposed for diagnosis: a persistent CD4+ T lymphocyte count <300 cells/mm^3 or <20% of total T cells (measured on at least 2 occasions) in the absence of HIV infection and any other known immunodeficiency or therapy associated with lymphocytopenia [4]. The present patient fulfilled these criteria. Idiopathic CD4+ T lymphocytopenia has been associated with various opportunistic infections, such as histoplasmosis, cryptococcosis, aspergillosis, mycobacteriosis, *Pneumocystis jiroveci* infection, polyoma virus infection, cytomegalovirus infection, and human papillomavirus infection, as well as hematologic malignancies [5, 6].

To our knowledge, this is the first reported case of visceral leishmaniasis in a patient with idiopathic CD4+ T lymphocytopenia. This type of immunodeficiency should always be considered in patients with visceral leishmaniasis and no alternative immune system disease.

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**References**


Wound Infection and Septic Shock Due to *Aeromonas trota* in a Patient with Liver Cirrhosis

To the Editor—*Aeromonas trota* has never been reported as a cause of wound infection and septic shock. A 78-year-old woman with hepatitis C virus–related liver cirrhosis (Child classification B) fell into a pond while she was riding a motorcycle and experienced multiple laceration and abrasion wounds on her face and hands. When the patient arrived at the emergency department 1 h after the accident, her blood pressure was 126/70 mm Hg. One day later, the patient developed fever (temperature, 38.6°C), hypotension (blood pressure, 88/38 mm Hg), and erythematosus change and pus discharge from a wound on her left hand. Laboratory examinations revealed a WBC count of 10,200 cells/mm^3, with 82% neutrophils. Because of the impression of wound infection associated with septic shock, empirical treatment with intravenous floxef (1 g every 8 h) was administered. Complete septic analyses, including cultures of urine, ascites, blood, and pus discharge from the infected wound on her left hand were performed prior to initiation of antibiotic therapy. The urinalysis result was negative for pyuria, and analysis of ascites revealed a neutrophil count of 50 neutrophils/mm^3. Two days later, the pus culture yielded confluent growth of

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an oxidase-positive, gram-negative bacillus. However, culture results of the other specimens were all negative. The biochemical profile of the isolate generated by the Vitek GNI Plus card (bioMérieux) and negative reactions for esculin hydrolysis and Voges-Proskauer test were in agreement with the identification of *A. trota*. The isolate was resistant to cefazolin, cefmetazole, and flomoxef but was susceptible to ampicillin, cefazidime, cephalase, and imipenem, using the standard disk diffusion method. The patient responded unsatisfactorily to flomoxef treatment, with persistent fever and hypotension necessitating inotropic agent use. Antibiotic treatment was switched to intravenous cefazidime (1 g every 8 h) after the notification of the susceptibility test results; fever subsided 1 day later, and the wound improved gradually. The patient experienced an uneventful recovery during 14 days of cefazidime treatment.

Human infections with *Aeromonas* species are most often associated with trauma involving exposure to contaminated fresh or brackish water or soil [1]. The predominant *Aeromonas* species associated with human infections are *Aeromonas hydrophila*, *Aeromonas caviae*, and *Aeromonas sobria* [1–3]. *A. trota* is commonly isolated from human feces, and isolation of this organism from a human appendix has also been reported [4]. However, *A. trota* has rarely been documented as a causative agent of human infections [2, 4, 5].

Voss et al. [6] reported that 43% of *Aeromonas* species–associated wound infections were water-related and that the striking of a submerged object (e.g., roots, tree branches, or rocks) while walking barefoot along the bank of a stream, river, or lake was the common precipitating event. Our patient was initially hospitalized because of injuries sustained from a fall into a pond while she was riding a motorcycle. This history suggests that *A. trota* infection developed as a result of exposure of the abrasion injury to an environmental source (water or soil) containing aeromonads. Most infections caused by *Aeromonas* species are found in immunocompromised hosts, especially in patients with liver cirrhosis (as in our patient) and malignancies [7, 8].

In previous reports, isolates of *A. trota* were shown to be susceptible to many antimicrobial agents, including ampicillin and piperacillin, but some were resistant to cefazolin (to which 20% of isolates were resistant) and cephalase (i.e., cefoxitin, to which 13% of isolates were resistant) [4, 9]. The antibiogram of our isolate supported the identification of *A. trota* [9]. The poor in vivo response to flomoxef experienced by our patient also paralleled the in vitro resistance of our isolate to this agent.

In summary, exposure to a fresh water environment and underlying liver cirrhosis were important precipitating factors for the development of an aeromonal wound infection in our patient. This case demonstrates that *A. trota* infection can present as wound infection and septic shock.

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Invasive Aspergillosis in Patients with Acute Leukemia: Update on Morbidity and Mortality—SEIFEM-C Report

To the Editor—Aspergillus species represent the main cause of fungal infections in patients with acute leukemia [1, 2]. During the past few years, we have conducted 2 consecutive multicenter studies to evaluate the incidence of and mortality rate associated with aspergillosis among these patients [2, 3]. In the first study (conducted from 1987 through 1998), among 4448 cases of acute leukemia (both lymphoid and myeloid), we identified 209 cases of proven or probable invasive aspergillosis, with an incidence of 4.7% and an attributable mortality rate (AMR) of 48% [2]. More recently (from 1999 through 2003), among a population of 4185 patients with acute leukemia, 257 proven or probable cases of aspergillosis were diagnosed, with an incidence of 6.1% [3]; the AMR was 38.5% (99 of 257 cases ended in death). Six institutions participated in both studies; an analysis of all patients with acute leukemia from 1987 through 2003 has been possible. An absolute increase in cases of aspergillosis was