heparin was prescribed. Four days later, prostaglandin E1 therapy was withdrawn and misoprostol (200 µg every 6 hours), nifedipine (10 mg every 8 hours), pentoxifylline (400 mg every 6 hours), and dexamethason (60 mg/d) were administered. The digital necrosis spread, and the pain became unbearable. Therefore, the distal phalanx of the left fifth finger was amputated 15 days after admission.

Pathological examination of specimens from the amputated phalanx revealed findings consistent with thrombotic microangiopathy affecting small-sized and capillary vessels. Leishmania species amastigotes were observed in the vessel lumen and walls (inside endothelial cells and fibroblasts) and around them (figure 1). Leishmania species amastigotes were also found in bone marrow aspirates. Meglumine antimoniate (850 mg of antimony per day for 28 days) was administered intravenously. There was good perfusion in all fingers except the third phalanx of the left fourth finger, which had an enlarging area of necrosis that required amputation. Three months later, the patient had no symptoms related to the previous episode of vasculitis. However, examination of a new bone marrow aspirate again demonstrated Leishmania amastigotes. Meglumine antimoniate therapy was administered again for 28 days.

There have been some small series and occasional case reports that described vasculitis in HIV-infected patients, although the real incidence of this infection is unknown. Polyarteritis nodosa–like syndromes, hypersensitivity vasculitides, lymphomatoid granulomatosis, primary vasculitis of the CNS, and other vasculitides of unspecified type are the most frequently described in HIV-infected patients [2–5]. Symptoms are usually related to skin, muscle, and peripheral nerve involvement, although the CNS and other organs may also be involved. The pathogenesis of these disorders is only partially understood but is related to immune disorders, drugs, and infectious diseases.

Infectious diseases may lead to vascular damage by means of two mechanisms. The first is by a direct lesion (a few cases have been reported that were due to Toxoplasma gondii, Pneumocystis carinii, Herpes simplex virus, and cytomegalovirus) [6–9], the second by immune-mediated damage (i.e., microbial antigen-induced immune complexes or cell-mediated immune response). Only one other case of digital necrosis in an HIV-1-infected patient has been reported. This case was due to microemboli of P. carinii, and there were no pathological findings of vasculitis in this case [10]. The patient we describe had a Leishmania species infection and developed vasculitis caused directly by the invasion of the parasite into the walls of small-sized and capillary vessels, provoking thrombosis, ischemia, and digital necrosis. Therefore, Leishmania species should be considered in the differential diagnosis of vasculitis and/or digital ischemia in HIV-infected patients.

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Cytomegalovirus Gastropathy in a Child: Resolution After Ganciclovir Therapy

Hypertrophic gastropathy, or Menetrier’s disease, is a rare disorder characterized by giant hypertrophy of gastric rugae and by protein loss. In adults, it is a chronic illness of unknown etiology that occasionally progresses to gastric lymphoma [1]. In contrast, in children there is usually spontaneous remission of the condition, and it has been associated with primary cytomegalovirus (CMV) infection [2]. We describe a child with a prolonged illness caused by CMV-associated gastropathy with protein loss and whose condition improved after ganciclovir therapy.

A 23-month-old boy was transferred to Children’s Hospital in Seattle after a 12-hour stay in a community hospital because of abdominal pain. He was previously healthy until 6 weeks before admission to the hospital, when he developed diarrhea with intermittent abdominal pain. Evaluation was notable for a WBC count of 38,000/mm³ with many atypical lymphocytes. On admission to Children’s Hospital, the patient was afibrile with periorbital and pedal edema. Shotty cervical and axillary lymphadenopathy and hepatomegaly were noted on physical
Examination of a specimen obtained by repeated gastric biopsy, performed 3 days after completion of ganciclovir therapy, showed only a prominent eosinophilic infiltrate. During 20 months of follow-up, the child has been well without peripheral edema or infections.

Protein-losing enteropathy is a rare complication of primary CMV infection. The association between CMV disease and gastric hypertrophy of childhood was initially noted by Lachman et al. [2]. Patients range in age from 22 months to 7.5 years old, and most are boys [3–6]. Periorbital and peripheral edema were found in all patients, reflecting low serum albumin levels. Despite frequent hypogammaglobulinemia, bacterial infections have been rare. Lymphocytosis with atypical lymphocytes has been reported, and eosinophilia occurs often. The evidence for CMV infection varies in the published reports, but many reports document typical inclusions in the epithelial cells of the gastric mucosa. The course of CMV gastropathy is generally benign, with spontaneous resolution of hypoalbuminemia after a 2–6 week course.

Successful Treatment of Disseminated Mucormycosis with Liposomal Amphotericin B and Surgery in a Child with Leukemia

Mucormycosis, which is caused by fungi of the order Mucorales, occurs almost exclusively in immunocompromised patients. Disseminated disease occurs more often in patients with hematologic malignancies [1]. Therapy is based on reversing the underlying predisposing condition in association with systemic antifungal therapy with amphotericin B and extensive surgical debridement [2]. We describe an immunocompromised leukemic child with disseminated mucormycosis who was successfully treated with liposomal amphotericin B.

In July 1995, acute lymphoblastic leukemia (ALL) was diagnosed in a 3-year-old boy in Portugal. On day 4 of induction chemotherapy, his neutrophil count was <500/μL. Fever of unknown origin occurred on day 16 of chemotherapy and persisted despite administration of broad-spectrum iv antibiotics and introduction of oral fluconazole therapy on day 22. Bone marrow aplasia was no longer noted on day 26, and examination of bone marrow aspirates showed complete remission of ALL. On day 33,