Syphilis was one of the most common diseases at the beginning of the twentieth century in Europe and the United States, but in the 1950s, after the introduction of penicillin, syphilis became uncommon [3]. However, the incidence of syphilis increased dramatically after the emergence of the HIV epidemic [3]. Syphilitic aortitis is not usually recognized clinically until 10 to 30 years after the occurrence of primary infection. Given the short survival rate that characterized HIV disease until the introduction of the current therapeutic modalities, it is not surprising that aortic aneurysms have not been more frequent among patients with HIV. However, given the slow progression of HIV disease achieved with the use of protease inhibitors, it is possible that cardiovascular-related syphilis will become more common. Further to this point it should be noted that in southwestern European countries, a large percentage of HIV patients are drug addicts and prostitutes. These individuals are at high risk for contracting syphilis and frequently do not consult healthcare providers, and, if they do, are often not compliant with therapy. Therefore, treatment of primary disease as well as diagnosis and treatment of latent syphilis may be difficult.

In summary, with the rise in syphilis prevalence (in HIV-infected and non-HIV-infected patients) seen since the appearance of HIV, the low therapeutic compliance often observed in some of these patients, and the longer survival achieved for HIV patients with the new therapeutic regimens, some almost forgotten forms of tertiary syphilis may become more common. Among these syphilitic forms is the aortic aneurysm, which may present as a superior vena cava syndrome, as seen in the patient we described.

**Figure 1.** A chest radiograph showing a large noncalcified anterior mediastinal mass in an HIV-infected patient with superior vena cava syndrome secondary to a syphilitic aneurysm of the ascending aorta.

Recovery was uneventful. Treatment with intravenous sodium penicillin G (24 million units daily for 10 days), zidovudine (500 mg/d), and lamivudine (300 mg/d) was begun. After 18 months the patient continued to be asymptomatic.

SVCS was first described in 1757 by William Hunter in a patient with a syphilitic aortic aneurysm [1]. Until the beginning of this century the most common causes of SVCS were benign; however, the etiology of the syndrome has changed during the last 5 decades [1, 2]. A review of the medical literature from 1757 to 1949 [2] indicated that 30% of SVCS cases resulted from expanding thoracic aortic aneurysms. Currently, >90% of cases of SVCS occur because of malignant intrathoracic disorders [1].

**Leishmaniasis of the Tongue in a Renal Transplant Recipient**

Immunocompromised patients are at risk of increased morbidity and mortality from visceral leishmaniasis [1]. Several reports have described visceral leishmaniasis in kidney transplant recipients [2]. Mucosal leishmaniasis is rare in the Old World and has been reported most often from the Sudan [3]. Treatment with antimony compounds continues to be the main therapeutic option. However, because of side effects, several alternatives have been tried [4]. We report a kidney transplant recipient with tongue leishmaniasis that responded to therapy with liposomal amphotericin B.

A 55-year-old Sudanese man who had undergone kidney transplantation in 1984 had been maintained on azathioprine and prednisone; his creatinine level was 250 μmol/L. In 1995, he noticed a slowly progressing tongue lesion. There were no

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other symptoms, and there was no history of leishmaniasis. At examination, he was afebrile, and there was no lymphadenopathy or splenomegaly. A 1.5-cm × 3-cm, nontender ulcer with a clean base was noted at the edge of the tongue (figure 1). Laboratory evaluation revealed the following values: WBCs, 5,100/mm³; hemoglobin, 14.4 g/dL; platelets, 225,000/mm³; and creatinine, 248 μmol/L. Indirect hemagglutination (IHA) titer of antibody to Leishmania donovani was 1:4,096 (Cellognost Leishmaniasis; Behringwerke AG, Marburg, Germany). Histopathologic evaluation of a tongue biopsy specimen revealed numerous leishmanial bodies in the histiocytes and interstitium. A culture of the biopsy specimen was negative for Leishmania species. A serology for antibody to HIV was negative. Itraconazole (200 mg/d) therapy was given for 4 weeks.

After initial healing, the ulcer recurred a few months later. No symptoms, lymphadenopathy, splenomegaly, or blood abnormalities were noted. A culture of a repeated tongue ulcer biopsy specimen yielded Leishmania donovani zymodeme MON 83 as characterized by isoenzyme electrophoresis [5]. Itraconazole was resumed for 3 months. The ulcer healed in 5 weeks, and a repeated IHA titer of antibody to Leishmania was 1:64. However, a recurrence of the ulcer was noted 10 months later. Liposomal amphotericin B (4 mg/kg) was given daily for 5 days. The ulcer healed completely. After 7 months of follow-up, no relapse was noted.

Mucosal leishmaniasis was first described in patients from the Sudan by Christopherson in 1914 [6]. By 1997, ~80 patients with mucosal leishmaniasis had been reported. L. donovani, Leishmania major, and Leishmania infantum have been recovered from these lesions. In immunocompromised patients, mucosal leishmaniasis has been reported in patients with cancer and patients with HIV/AIDS. To our knowledge, based on a MEDLINE search (1966 to February 1998), we have described the first case of mucosal leishmaniasis in a transplant recipient. Because the patient had no symptoms of visceral leishmaniasis, and because of our institution’s experience with antimony-induced pancreatitis in kidney transplant recipients, we elected to treat with itraconazole. After the relapse, the patient refused to undergo further evaluation for systemic involvement and bone marrow evaluation.

Although antimony is the main treatment for leishmaniasis, the side effects associated with its use have prompted the search for other options [4]. Oral azole agents have been shown to have activity against several Leishmania species in vitro [7]. The results of itraconazole therapy for the treatment of cutaneous leishmaniasis are inconsistent [8]. Among immunocompromised patients, itraconazole therapy for visceral leishmaniasis has been associated with clinical improvement and relapse [9]. Amphotericin B has been used to treat visceral leishmaniasis in immunocompetent and immunocompromised patients [10]. Because of the agent’s nephrotoxicity, lipid formulations are more suitable for treatment in kidney transplant recipients. Given that there is a potential for visceral leishmaniasis relapse in kidney transplant recipients with antimony therapy, we recommend liposomal amphotericin B as an alternative for treatment of leishmaniasis in this patient population.

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