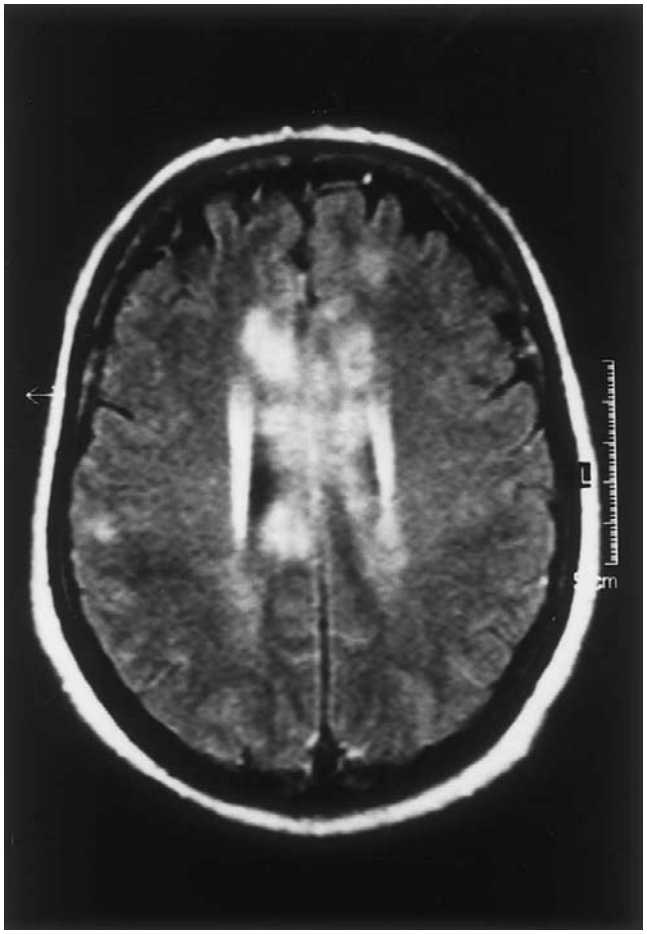

Figure 1. MRI of the brain, showing lesions caused by Epstein-Barr virus encephalitis

the virus) a few days after transplantation [2–4], but, in our patient, reactivation occurred almost 8 years after transplantation. However, as described in previous reports [1, 4, 5], PCR detection of viral DNA in CSF samples allowed for diagnosis of the disease, which had already been entertained on the basis of the clinical features. The PCR results also excluded the diagnosis of encephalitis due to other viruses, such as herpes simplex virus, CMV, or human herpesvirus 6.

Successful treatment with intravenous ganciclovir has been reported in other cases of encephalitis caused by EBV [3]. Our observations support those findings and emphasize the importance of starting treatment as soon as the disease is suspected on the basis of clinical features, because encephalitis caused by EBV can have considerable neurological sequelae and can be fatal.

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References

1. Pednault L, Katz BZ, Miller G. Detection of Epstein-Barr virus in the brain by the polymerase chain reaction. Ann Neurol **1992**; 32: 184–92.
2. Kim S-C, Jang H-J, Han D-J. Acute disseminated encephalomyelitis after renal transplantation in patients with positive Epstein-Barr virus antibody. Transplant Proc **1998**; 30: 3139.
3. Dellemijn PLI, Branderburg A, Niesters HGM, et al. Successful treatment with ganciclovir of presumed Epstein-Barr meningo-encephalitis following bone marrow transplant. Bone Marrow Transplant **1995**; 16:311–2.
4. Freymuth F, Rossignol P, Hurault de Ligny B, Gallet E. Meningoencephalitis, expression of Epstein-Barr virus primarily in a patient with renal transplantation: value of the search of genome by PCR. Presse Med **1994**; 23:1314–6.
5. Landgren M, Kyllerman M, Bergström T, et al. Diagnosis of Epstein-Barr virus–induced central nervous system infections by DNA amplification from cerebrospinal fluid. Ann Neurol **1994**; 35:631–5.

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HIV Lipoatrophy and Mosquito Bites

Sir—In the summer of 1999, several patients infected with HIV type 1 described to us an increase in the frequency of mosquito bites received since being treated with potent antiretroviral therapy (ART). To analyze this possibility, we performed a prospective cross-sectional study aimed at identifying an eventual association between ART and mosquito bites.

During 4 consecutive weeks (August–September), 122 HIV-infected patients who were seen in our outpatient HIV clinic and received treatment were included in the study. Data on prevalence of mosquito bites, demographics, CD4 count, HIV load, type of ART received, and ART-associated adverse events were collected using a standardized questionnaire. Multivariate logistic regression was performed using STATA software, version 6.0 (Stata Corporation).

Seventy-four patients (61%) described having received 1 or more mosquito bites during the 3 months before the study. A total of 28 patients reported that, compared with people in their immediate environment, they had a greater susceptibility to being bitten by mosquitoes. Ten patients believed that they were more susceptible to being bitten since having tested positive for HIV infection, and 12 believed that such susceptibility had increased since they began receiving ART.

Mosquito bites were observed on 18 patients (14.8%), 7 of whom had ⩾5 bite marks. Using a multivariate logistic regression model adjusted for age, sex, CD4 count, HIV load, and HIV-transmission group, we identified an independent association between lipoatrophy and (1) a history of having been bitten by mosquitoes in the 3 months before the study (OR, 2.44; 95% CI, 1.06–5.59) and (2) the presence of mosquito bites at the time of physical examination (OR, 10.13; 95% CI, 2.65–38.73). No specific type of ART was found to be associated with mosquito bites.

Lipoatrophy has been increasingly described among individuals who receive nucleoside reverse-transcriptase inhibitors [1]. Lipoatrophic subcutaneous tissue may present a more accessible capillary network and an increased release of volatile substances from the skin surface. A similar mechanism, triggered by increased blood flow, has been suggested as an explanation for the attraction of mosquitoes to the skin of pregnant women [2, 3]. Whether the observed increase in mosquito bites may result in greater risk for insectborne infections, such as those caused by *Leishmania* and *Plasmodium* species, can only be speculated. The association of lipoatrophy with an increase in mosquito bites has no bearing on HIV transmission, because insect vectors play no role in the spread of HIV [4].

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References

1. Saint-Marc T, Partisani M, Poizot-Martin I, et al. A syndrome of peripheral fat wasting (lipodystrophy) in patients receiving long-term nucleoside analogue therapy. AIDS **1999**; 13:1659–67.
2. Lindsay S, Ansell J, Selman C, et al. Effect of pregnancy on exposure to malaria mosquitoes. Lancet **2000**; 355:1972.
3. Martinez EF, Alecrim WD, Daniel-Ribeiro CT. Attraction of mosquitoes to pregnant women. Lancet **2000**; 356(9230):685.
4. Booth W. AIDS and insects. Science **1987**; 237: 355–6.

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Recurrent Nonmenstrual Toxic Shock

Sir—Andrews et al. [1] recently published a review entitled "Recurrent Nonmenstrual Toxic Shock Syndrome: Clinical Manifestations, Diagnosis and Treatment." As their title suggests, although recurrent menstrual toxic shock is not uncommon, few reports of this syndrome have been published. The authors review 9 previous cases and report 3 of their own, including 1 in a patient with AIDS.

In 1992 we described 5 patients with a recalcitrant, erythematous desquamative disorder associated with toxic shock toxin-1 (3 patients), staphylococcal enterotoxin A (1 patient) and staphylococcal enterotoxin B (1 patient) [2]. Three of the 5 patients died; autopsies of 2 patients confirmed residual staphylococcal infection, and, interestingly, both survivors developed recurrent disease. All patients were homosexual men. One of the patients significantly improved after receiving commercial iv gammaglobulin, which contains staphylococcal toxin antibody [3]. Indeed, the HIV-1–infected patient treated by Andrews et al. [1] also responded favorably to iv gammaglobulin.

We suggested that the recalcitrant, erythematous desquamative disorder was related to defective chemotaxis [4], but observed that a lack of staphylococcal toxin antibody formation could also play a role. Therefore, the disorder—the characteristics of which have subsequently been confirmed [5, 6]—may have an immunologic relationship to recurrent nonmenstrual toxic shock syndrome in patients infected with HIV-1; patients with these disorders likely lack staphylococcal toxin antibody production. The absence of toxin antibody may be due to the "superantigen" molecular behavior of staphylococcal toxins.

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References

1. Andrews M-M, Parent EM, Parsonnet J. Recurrent nonmenstrual toxic shock syndrome: clinical manifestations, diagnosis, and treatment. Clin Infect Dis **2001**; 32:1470–9.
2. Cone LA, Woodard DR, Byrd RG, Schulz K, Schlievert PM. A recalcitrant, erythematous, desquamating disorder associated with toxin producing staphylococci in patients with AIDS. J Infect Dis **1992**; 165:638–43.
3. Chesney PJ. Clinical aspects and spectrum of illness of toxic shock syndrome: overview. Rev Infect Dis **1989**; 11(Suppl 1):S1–7.
4. Cone LA, Thind D, Fiala M, Woodard DR, Casareale D. Normal neutrophil phagocytosis but impaired chemotaxis in homosexual male patients with AIDS, ARC and neither disorder. In: Proceedings of the 3d International Conference on AIDS (Washington, DC). Washington, DC: Bio-Data, **1987**.
5. Dondorp AM, Veenstra J, van der Poll T, Mulder JW, Reiss P. Activation of the cytokine network in a patient with AIDS and the recalcitrant erythematous desquamating disorder. Clin Infect Dis **1994**; 18:942–5.
6. Verbon A, Fisher CJ Jr. Severe recalcitrant erythematous desquamating disorder associated with fatal recurrent toxic shock syndrome in a patient without AIDS. Clin Infect Dis **1997**; 24:1274–5.

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Antifungal Prophylaxis and the Rate of Bacteremia among Neutropenic Patients

Sir—We recently published an article in *Clinical Infectious Diseases* that explored the possible association between antifungal prophylaxis and the rate of documented bacteremia among febrile neutropenic patients with cancer [1]. This