tion of a lumbar drain, or implantation of a VP shunt is recommended if neither focal neurologic signs nor obtunded consciousness with space-occupying lesions visible by radiographic imaging were observed [2, 9]. However, frequent lumbar punctures and large-volume drainage in the presence of elevated ICP have raised concerns among clinicians about the risk of brain herniation [6]. Antinori and colleagues [6] reported 2 cases of cryptococcal meningitis in patients who experienced loss of consciousness and died soon after lumbar puncture. At autopsy, brain herniation was considered by Antinori et al. [6] to be the cause of death; however, inadequate CSF drainage and the resultant brain herniation—not the lumbar puncture—was suspected by Graybill and Sobel [10] to be the cause of death.

In our patients with cryptococcal meningitis who had extremely high ICP and poor prognostic predictors of death during initial therapy (i.e., abnormal mental status, a CSF antigen titer of >1:1024, and a CSF WBC count of <20/μL [11]), we found that, with frequent, multiple lumbar punctures combined with antifungal therapy consisting of amphotericin B and fluconazole, 7 patients survived the disease. The condition of the patient who developed visual loss because of extremely high ICP improved with our aggressive external CSF drainage. The 2 deaths were most likely caused by delayed presentation for appropriate HIV care. In 1 of the 2 patients, signs of increased ICP resolved gradually, and altered consciousness returned to normal after multiple procedures. However, extremely high ICP recurred and resulted in death. This case should alert clinicians to the possibility of recurrent high ICP despite antifungal therapy, and careful monitoring of the ICP is indicated.

In conclusion, the findings of our study support the recommendation of aggressive use of lumbar puncture for the management of elevated ICP to reduce the risk of early mortality and late morbidity, even for patients with extremely high ICP. Multilevel lumbar punctures and large-volume CSF drainage in HIV-infected patients with elevated ICP due to cryptococcal meningitis is safe after the presence of space-occupying lesions is excluded.

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Hemophagocytic Syndrome in a Patient with Acute Human Immunodeficiency Virus Infection

Sir—In their recent case report, Sproat et al. [1] described a patient with advanced HIV infection and hemophagocytic syndrome (HPS) who did not respond to HAART, and they underlined the fact that HPS caused by HIV infection alone has not been widely reported.

Recently, we observed an acute HIV infection complicated by severe HPS that occurred in a 27-year-old Italian man. The patient was admitted to our institution (Ospedale Belcolle, Viterbo, Italy) with a 7-day history of fever (temperature, 40°C), headache, sore throat, and malaise. At admission, he had a diffuse rash, generalized lymphadenopathy, and hepatosplenomegaly. Laboratory results showed mild anemia (Hb 9.8 g/dL), leukopenia (WBC count, 3400 cells/mm³), and raised enzyme levels (alanine aminotransferase, 468 U/L; γ-glutamyl transferase, 1,974 U/L; lactate dehydrogenase, 9,404 mU/mL). The patient denied having any risk behaviors for HIV infection. His serum sample was initially negative for HIV antibody by ELISA and Western blotting. Antibodies to Epstein-Barr virus, cytomegalovirus, herpes simplex virus, parvovirus B19, hepatitis C virus, and Toxoplasma gondii were absent, as was antibody to hepatitis B surface antigen. The patient’s CD4+ lymphocyte count was 138 cells/mm³ (CD4+ percentage, 22%), with a CD4+:CD8+ ratio of 0.66. A total body CT scan showed he-
patosplenomegaly without focal lesions, and there were no abnormal findings of examination of the brain and the thorax. Results from examination of CSF were normal. A specimen from a laterocervical node biopsy showed small residual follicular areas and an intense histiocytic proliferation, with marked hemophagocytosis. The patient refused a bone marrow biopsy; however, the clinical and histopathological features of his illness were consistent with HPS [2].

Four days after admission, oral candidiasis developed. Results from a second ELISA test for HIV were negative, but the presence of HIV p24 antigen (level, 93 pg/mL) and a high HIV RNA value (27 × 10^6 copies/mL) revealed that the patient had an ongoing primary HIV infection. HAART with zidovudine, lamivudine, and lopinavir/ritonavir was promptly started, and, 5 days later, the patient’s clinical condition quickly improved, and all initial symptoms disappeared completely. ELISA and Western blot result became clearly positive only 15 days after admission, and, at that time, all blood laboratory test results were normal. After 4 weeks, his lymphocyte CD4+ count was 809 cells/mm³, and it remained stable for months thereafter. HIV load showed a progressive decrease, and it was undetectable 6 months after the start of HAART, as well as later.

The patient’s partner, a 30-year-old Haitian woman who had been living in Italy for 12 years, was tested for the first time and was found to be an asymptomatic HIV carrier. She was considered to be the source of the infection in the case patient because of the presence of the same I93L polymorphism of the protease gene on strains of the virus isolated from PBMC and plasma samples of both donor and recipient. Both isolates were apparently indistinguishable with respect to replication efficiency, coreceptor utilization (CCR5), and predicted phenotype (M-R5). Molecular analysis showed the presence of a virtually homogeneous V3 quasi species in the recipient, although highly divergent variants were found in the donor.

In our patient, HAART was very effective in controlling both the acute HIV infection and HPS. This case further confirms the direct role of HIV in causing HPS. Finally, although the biologic and molecular characteristics of virus isolates from the patient and his partner were apparently indistinguishable, clinical manifestations of HIV infection had a very different presentation.

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