BRIEF REPORTS

Discontinuation of Secondary Prophylaxis for Toxoplasmic Encephalitis in Human Immunodeficiency Virus Infection After Immune Restoration with Highly Active Antiretroviral Therapy

Toxoplasma gondii is one of the leading causes of neurological morbidity and mortality in patients with advanced HIV infection. Effective therapies for primary and secondary prevention of toxoplasmic encephalitis in HIV infection have been established [1, 2]. With the introduction of highly active antiretroviral therapy (HAART), discontinuation of primary and secondary prophylaxis for opportunistic infections in patients with HIV infection and good response to HAART have become a focus of interest. We describe an HIV-infected patient with toxoplasmic encephalitis and excellent response to HAART who discontinued secondary prophylaxis for toxoplasmosis.

A 26-year-old woman with HIV infection presented to our clinic with headache in November 1996. She had acquired HIV infection in 1986 by needle exchange, and, in the following years, did not seek regular medical assistance. She had not received any prophylaxis for opportunistic infections or treatment with antiretroviral drugs. Physical examination showed no neurological deficits. A CT scan revealed a 2.8-cm diameter solitary hypodense cerebral lesion with perifocal edema on the right parietotemporal hemisphere (figure 1*A*); this lesion was suspected to be toxoplasmic encephalitis. IgG antibodies to *T. gondii* were positive at 34 U/mL, her CD4 cell count was 29/mm^3, and the plasma viral load was 302,000 copies/mL. The patient was empirically treated with pyrimethamine, sulfadiazine, and leucovorin.

Four weeks later, a control CT scan showed regression of both the cerebral lesion and the perifocal edema. Because of a severe rash, sulfadiazine was changed to clindamycin, and after 6 weeks, the patient received maintenance therapy. Antiretroviral therapy with lamivudine, stavudine, and indinavir was then started. The response was excellent, and in the following 18 months, the CD4 cell count progressively increased to >1100/mm^3, and her plasma viral load was permanently suppressed (as measured by an ultrasensitive assay [Amplicor; Roche Diagnostic Systems, Rotkreuz, Switzerland]).

After 22 months, the patient insisted that her maintenance

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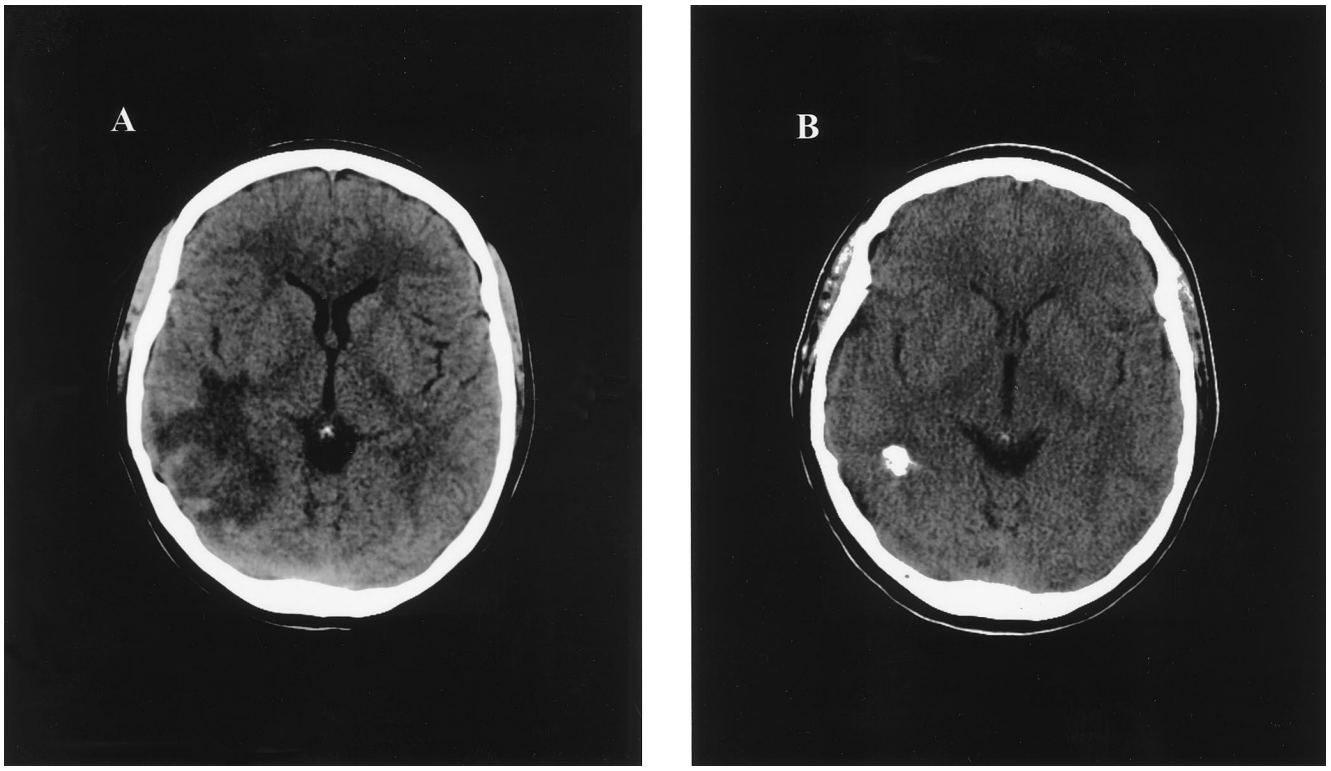

Figure 1. *A,* CT scan before empirical therapy for toxoplasmic encephalitis in an HIV-infected patient. *B,* CT scan 2.5 years later, after immune restoration with highly active antiretroviral therapy and 8 months without maintenance therapy for toxoplasmic encephalitis.

therapy for toxoplasmic encephalitis be discontinued to reduce her pill load of 20 tablets a day. At this time, a CT scan showed a 1.7-cm diameter cerebral lesion with calcification that was identical to that on a previous CT scan at a 12-month follow-up. Twelve months after discontinuation of secondary prophylaxis for *T. gondii* infection, the patient had no clinical signs of relapse, and her CT scan remained unchanged (figure 1*B*).

Data from observational studies on the discontinuation of primary prophylaxis for *Pneumocystis carinii* pneumonia in patients with persistent CD4 lymphocyte counts >200/mm^3 and antibodies to *T. gondii* have shown that no relapses of *P. carinii* pneumonia and *T. gondii* infection occurred [3, 4]. However, the number of patients with antibodies to *T. gondii* was too small to allow for firm conclusions as to whether discontinuation of primary prophylaxis for toxoplasmic encephalitis is safe.

Discontinuation of maintenance therapy for patients with established toxoplasmic encephalitis is not recommended [5]. Relapse rates after discontinuation of maintenance therapy, as reported from the pre-HAART era, may be ⩾50% [2]. However, secondary prophylaxis for toxoplasmic encephalitis is all but satisfactory. Rates of breakthrough relapses associated with sulfadiazine and pyrimethamine, the most effective drug combination, are 10%–40%, and rates of drug-limiting toxicity associated with this combination are 30%–43% [2].

Because of side effects, high pill load, and drug interactions, discontinuation of maintenance therapy for toxoplasmosis after immune restoration with HAART is likely to improve the quality of life in patients with AIDS and to increase adherence to antiretroviral therapy. Therefore, studies evaluating whether secondary prophylaxis for toxoplasmic encephalitis may be discontinued for selected patients with excellent response to HAART seem timely.

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Intestinal Involvement by Nontuberculous Mycobacteria After Heart Transplantation

The prevalence of disease due to nontuberculous mycobacteria (NTM) in the United States is 1.8 cases per 100,000 population [1]. In the general population, 5%–10% of mycobacteria causing infections are NTM, whereas in transplant recipients NTM cause 25%–40% of all mycobacterial infections [2]. We describe a case of infection with *Mycobacterium avium*/*Mycobacterium intracellulare* in a heart transplant recipient and review the literature on disease due to NTM [3–12].

A 56-year-old man underwent orthotopic heart transplantation in June 1990 for ischemic cardiomyopathy. In November 1992, he was admitted to the hospital because of fever (temperature, 39°C) during treatment with cyclosporine, azathioprine, and deflazacort. Physical examination disclosed a small epitrochlear node and a tender periumbilical mass. His erythrocyte sedimentation rate was 92 mm/h. Blood cultures and serological tests for *Brucella, Salmonella, Yersinia,* cytomegalovirus, and HIV were negative. Ziehl-Neelsen staining of urine and sputum samples were negative, as were cultures of these samples on Löwenstein-Jensen medium. Purified protein derivative skin testing was also negative. A CT scan of the chest and abdomen revealed enlarged retroperitoneal and mesenteric lymph nodes.

Biopsy of the epitrochlear node revealed granulomas with scarce acid-fast bacilli, and culture of the node on Lowënstein-Jensen medium was negative. Laparotomy showed mesenteric and retroperitoneal lymphadenopathies. Histopathologic examination of the nodes demonstrated noncaseating granulomatous adenitis, whereas Ziehl-Neelsen staining of the nodes and culture of the nodes on Löwenstein-Jensen medium were negative. Azathioprine therapy was discontinued, and the cyclosporine dose was reduced. Subsequently, the fever subsided, the erythrocyte sedimentation rate normalized, and lymphadenopathies regressed.

The patient remained asymptomatic until December 1993, when he presented because of abdominal pain and a 5-kg weight loss. Gastrointestinal examination revealed a distorted duodenum with duodenal and jejunal mucosal thickening. An ab-

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