Toxoplasmosis-Associated Immune Reconstitution Inflammatory Syndrome in an Allogenic Hematopoietic Stem Cell Transplant Recipient

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A 34-year-old man with chronic myelogenous leukemia status post allogeneic hematopoietic stem cell transplant (HSCT) on tacrolimus for graft-vs-host disease prophylaxis, presented with a 1-week history of fevers and severe headaches. Brain magnetic resonance imaging (MRI) demonstrated numerous small focal lesions in the left basal ganglia and left temporal lobe with a ring pattern, as

**FIGURE 1.** Brain magnetic resonance imaging (MRI) obtained before initiation of antitoxoplasma therapy. Axial T2 FLAIR (A, B), diffusion-weighted (C), and post-gadolinium T1-weighted images. There are multiple diffusion restricted lesions in the brain, some of which demonstrate a ring pattern. Some of the lesions, including a large left occipital lesion, demonstrate prominent T2 signal hyperintensity and enhancement. In addition, there is prominent leptomeningeal FLAIR signal hyperintensity and enhancement involving the left occipital lobe.
well as a large lesion in the left occipital lobe with mild leptomeningeal enhancement (Figure 1). Cerebrospinal fluid (CSF) analysis revealed lymphocytic pleocytosis, and Toxoplasma gondii was detected by polymerase chain reaction (PCR). The patient was started on intravenous trimethoprim-sulfamethoxazole, with complete resolution of symptoms. Tacrolimus dose was decreased concomitantly.

A week later, the patient developed right homonymous hemianopsia. A repeat MRI of the brain revealed new bilateral supratentorial parenchymal lesions, with progressive leptomeningeal enhancement (Figure 2). CSF analysis was repeated, demonstrating improvement of previous parameters and negative T. gondii PCR. In the setting of clinical worsening on appropriate therapy, a diagnosis of immune reconstitution inflammatory syndrome (IRIS) was established. The patient was started on methylprednisolone.

Cerebral toxoplasmosis associated with IRIS is an uncommon complication, with most cases described in human immunodeficiency virus-infected patients.1 The diagnosis should be suspected following a paradoxical clinical worsening after initiating proper therapy. Glucocorticoids may be needed for patients who develop severe symptoms, and the treatment for the underlying infection should be continued. Nonetheless, treatment failure and superimposed infection should be ruled out before initiating steroid treatment.

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