Several hours after hospitalization, the patient experienced a rapid onset of confusion with decreased alertness, which prompted her transfer to intensive care. Clinical examination revealed bilateral Babinski’s sign. A CT of the cranium did not show evidence of intracranial hemorrhage. Given that these neurological symptoms arose in the context of fever associated with mechanical hemolytic anemia and thrombocytopenia, they were suggestive of TTP.

A treatment regimen consisting of high-dose corticosteroids together with blood plasma transfusions led to rapid improvement in the patient’s condition in a few days (based on clinical and laboratory evaluations). An ELISA revealed IgM antibodies to CMV and the progressive appearance of IgG antibodies (initial level, 2 IU/L, increasing to 700 IU/L 8 months later). There was also evidence of CMV in the patient’s urine. Testing for antigen pp65 was not performed. In view of these results, treatment with foscamet was administered secondarily for 3 weeks despite the improvement in the patient’s condition, as already demonstrated on clinical and hematologic examination.

Eight months later the patient was symptom free, and only evidence of slight hepatic cytolysis persisted (AST level, 56 IU/L; ALT level, 118 IU/L). Serological tests for hepatitis A, B, and C and the immunologic work-up remained negative. Examination of a liver biopsy specimen showed a few inflammatory lesions of the portal spaces and fragmented and agglutinated erythrocytes in the sinusoids; results of histochemical evaluation for CMV were negative.

TTP is a disorder that can be classified among thrombotic microangiopathies along with hemolytic uremic syndrome (HUS). In TTP, microangiopathic hemolytic anemia is associated with schistocytosis, thrombocytopenia, and neurological dysfunction (often with fever and renal involvement) [3]. TTP has been described as occurring in the setting of viral infections, principally HIV and, more rarely, human T-lymphotropic virus type 1 [4]. From a pathophysiologic standpoint, TTP results from damage to vascular endothelial cells and platelet aggregation, leading to the formation of arteriolar microthrombi in different organs, predominately the kidney and brain [5]. CMV is known to have an affinity for the vascular endothelium [6], and endothelial-cell lesions linked to viral replication might thus give rise to TTP.

The prognosis of TTP in the absence of treatment is poor, with death often occurring in days or weeks [5]. Treatment generally consists of corticosteroids and plasma transfusions [7]. Such management led to our patient’s rapid recovery.

The present case of TTP and another case involving HUS [2] probably justify systematic searching for an underlying primary CMV infection in patients with thrombotic microangiopathies.


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Central Nervous System Infections with Nontuberculous Mycobacteria

SIR—We read with interest the article by Flor et al. [1] describing two cases of Mycobacterium avium complex (MAC) meningitis and the article by Smith et al. [2] reporting a fatal case of Mycobacterium fortuitum meningitis in a patient with AIDS. We recently published a comprehensive review of CNS infections due to all species of nontuberculous mycobacteria that included meningitis, encephalitis, and brain or spinal cord abscesses [3]. We believe that each of us was responding to an apparent increase in these diseases in recent years.

We identified 56 cases of MAC disease of the CNS (48 of the patients had AIDS). Most of these patients had meningitis or meningoencephalitis. Two of the patients without AIDS had brain abscesses, and on the basis of postmortem or brain biopsy examinations, nine were described as having encephalitis without evident meningeval disease. Six of nine patients with Mycobacterium kansasi disease of the CNS had AIDS. Two of these patients had abscesses without evident meningitis. Seven cases of M. fortuitum CNS disease were identified, including two cases (one case of ventriculitis and one case of brain abscess) not included in the review by Smith et al. [2]. Five of these patients had meningitis, three had abscesses of the CNS, and one each had subdural empyema and ventriculitis. Four of six patients with adequate histories had infections associated with trauma or surgery, and one each had otitis media and mastoiditis. Four additional case reports of CNS infection due to Mycobacterium gordoneae, Mycobacterium genavense, and Mycobacterium terrae were identified [3].

Thus, with the reports of CNS disease due to MAC [1] and M. fortuitum [2], there have been a total of at least 79 reported cases of CNS disease, including meningitis, encephalitis, and brain abscess, due to nontuberculous mycobacteria. As the epidemic of HIV infection and the application of immunosuppressive treatments expand, we may anticipate further occurrences of these uncommon infections, with involvement of the brain itself, as well as the meninges.
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