the Denes et al. report was given it early in her hospital stay. The available evidence supports methylprednisolone as an essential drug in the management of ADEM.

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References

In Reply: The disease that we reported (1) was encephalomyelitis induced by a human herpesvirus 6 (HHV-6) reactivation. Our aim was to emphasize that HHV-6 can cause such a disease, even when the patient is immunocompetent, and to urge physicians to search for it.

Implicating HHV-6 in the pathogenesis of neurologic manifestations in the reported case can be challenged, as suggested by Dr. Soto-Hernandez (2). Polymerase chain reaction (PCR) results must be interpreted cautiously, especially in cases that lack corroborating clinical evidence of infection. In our case, the diagnosis was made initially when HHV-6 was found in the patient’s cerebral spinal fluid (CSF) by using PCR, by the absence of other cause, and by our experience; adult CSF is usually negative for HHV-6 by using PCR. Moreover, in our case, clinical symptoms and HHV-6 in the patient’s CSF evolved in the same way. The neurologic tropism of HHV-6 is well known, and the main manifestation in adults is encephalitis, especially in an immunosuppressed context. Diagnosis is usually based on finding HHV-6 genetic material in the CSF (3,4), which has now replaced brain biopsy. Positive tissue results do not distinguish latent from productive infections when PCR-Positive CSF indicates viral particle production in the central nervous system (CNS). In our case, the absence of brain tissue did not allow immunohistochemical staining or in situ hybridization. In the study by Caserta et al. (5), cited by Dr. Soto-Hernandez, HHV-6 PCR was positive in CSF and negative in peripheral blood mononuclear cells in 28.9% of children ≤3 years old with prior HHV-6 infection. These results provide evidence of HHV-6 persistence in the CNS; this phenomenon is now well recognized. Nevertheless, HHV-6 persistence after primary infection is quite different from reactivation in an immunocompetent adult. High-avidity anti-HHV-6 immunoglobulin G detected in the patient’s serum when the symptoms started proved that our patient had been infected with HHV-6 previously.

We appreciate Dr. Soto-Hernandez’s suggestion concerning acute demyelinating encephalomyelitis (ADEM) in our case. ADEM is an inflammatory demyelinating disease of the CNS, occurring mostly in children and rarely in young adults, soon after an infection or a vaccination. The disease is often associated with exanthema. A virus is often thought to be the cause, but viral symptoms are often not documented and rarely treated. ADEM may evolve into multiple sclerosis, and HHV-6 has been proposed as one of the causes of that condition (6). For example, multiple sclerosis developed in 14% of children and 35% of adults with ADEM in the study by Schwarz et al. (7).

Spontaneous improvement of ADEM is regularly reported; however, when treatment is needed, especially during the acute phase, steroid therapy is frequently used. In our case, corticosteroids did not affect the evolution of the patient’s neurologic symptoms. Conversely, introducing the antiherpes drugs (cidofovir and ganciclovir) was followed by improved clinical signs and negative results for HHV-6 in the CSF by PCR. Corticosteroids likely influence inflammation associated with ADEM, but if ADEM is the result of a viral infection with persistent viral replication, steroids might be deleterious, allowing an increase in viral replication (8,9).

In conclusion, we think that viruses, particularly HHV-6, should be considered in ADEM, even in immunocompetent patients. In case of a positive result, antiviral treatment must be given, eventually in association with corticosteroids. We cannot recommend using corticosteroids alone because of the risk of spreading the infection.
Vancomycin Heteroresistance in Methicillin-resistant Staphylococcus aureus, Taiwan

To the Editor: In 1997, Hiramatsu and colleagues reported the first clinical isolate of methicillin-resistant Staphylococcus aureus (MRSA) showing reduced susceptibility to vancomycin (1). Soon thereafter, vancomycin-intermediate S. aureus (VISA) or heteroresistant VISA was reported to have disseminated in Japanese hospitals (2). In Taiwan, a survey of >5,000 clinical isolates of S. aureus at one tertiary medical center from 1999 to 2001 showed negative results for VISA or vancomycin-resistant S. aureus (VRSA) (3,4). We report the first two isolations of heteroresistant VISA in Taiwan.

In June, 2000, an 89-year-old man (patient A) with a history of cerebrovascular accident underwent ileal resection for ischemic bowel disease, and primary MRSA bacteremia developed during the hospitalization. Vancomycin was given for 14 days, and his fever rapidly abated. In October 2000, a Port-A-Cath (Smiths Industries Medical Systems, Deltec, Inc., St. Paul, MN) was inserted in the left subclavian vein. On June 14, 2001, he had another blood isolate of S. aureus resistant (VISA) or vancomycin-resistant S. aureus (VRSA) (3,4).

A 72-year-old man (patient B) with coronary artery disease and chronic renal insufficiency underwent coronary artery bypass grafting and aortic grafting for abdominal aortic aneurysm in December 1999. The postoperative course was complicated with second-degree atrioventricular block and progressive renal failure. He was implanted with a permanent pacemaker and started long-term hemodialysis in March 2000. In April 2000, catheter (double lumen for hemodialysis)-related MRSA bacteremia recurred from May to July 2000, despite an adequate serum level of vancomycin (trough level = 13–21 µg/mL and peak level = 24–38 µg/mL). Transesophageal echocardio-