We report the first case, to our knowledge, of a patient with a history of recurrent zosteriform (dermatomal vesicular) skin eruptions and concurrent aseptic meningitis. Polymerase chain reaction (PCR) amplification of herpes simplex virus 2 (HSV-2) DNA in the cerebrospinal fluid (CSF) proved that aseptic meningitis was caused by HSV-2.

**REPORT OF A CASE**

A 37-year-old woman developed fever, chills, acute occipital and nuchal headache described as the “worst of my life,” nuchal rigidity, and photophobia in association with blisterlike lesions on her lower back and buttock in the region of the right L4 and L5 dermatomes. She reported recurrent episodes every 3 to 4 months of crops of identical vesicular lesions on an erythematous base for the past 4 years, unassociated with systemic or neurological symptoms. There was no history of genital herpes. On examination, she was afebrile. The skin over the right L4 and L5 dermatomes exhibited resolving vesicular lesions covering an area of 4 to 5 cm. There were no perineal or vaginal lesions. Findings from neurological examination revealed nuchal rigidity, a Brudzinski sign, and mild short-term memory impairment but otherwise normal results. The findings of a brain computed tomographic scan were normal. The CSF contained 345 white blood cells, 96% mononuclear; the protein level was 150 mg/dL, and the glucose level was 56 mg/dL (3.1 mmol/L). Because the zosteriform lesions were resolving, no skin scrapings were obtained or examined for HSV DNA. However, because of the possibility that the zosteriform lesions and aseptic meningitis were caused by HSV, the patient was treated with 10 mg/kg of intravenous acyclovir for 1 to 2 days followed by 800 mg of oral acyclovir 5 times daily for 1 week. One week later, the CSF contained 307 white blood cells, all mononuclear; the protein level was 78 mg/dL, and the glucose level was 51 mg/dL (2.8 mmol/L). The PCR analysis of the initial CSF specimen, using primers specific for a homologous region within the DNA polymerase gene of HSV-1 and HSV-2, revealed HSV DNA; restriction enzyme digestion patterns of the amplified DNA identified it as HSV-2.

**COMMENT**

Herpes simplex virus is a ubiquitous human pathogen that, after primary infection, becomes latent in the cranial nerve ganglia (HSV-1) or lumbosacral ganglia (HSV-2). Genital herpes is caused by HSV-2 reactivation. Less often, reactivation produces aseptic meningitis, although HSV-2 is the most common cause of recurrent aseptic meningitis in adults. Our patient had aseptic meningitis caused by HSV-2, as confirmed by PCR, simultaneous with an episode of recurrent L4 and L5 zosteriform skin lesions. To our knowledge, this is the first demonstration of HSV-2 DNA in the CSF by PCR in a patient with recurrent skin lesions and concomitant viral meningitis. We suspected HSV-2 because it causes recurrent skin lesions with neuropathy as well as recurrent genital lesions with neuropathy, urinary retention, and CSF pleocytosis. Previously, virological verification has been
obtained using serological methods or isolation of the virus from skin lesions.

Zosteriform lesions are also caused by varicella-zoster virus. However, recurrent zoster (shingles) is rare except in severely immunocompromised individuals. In contrast, HSV can often recur, frequently involving the same dermatome and often in immunocompetent individuals such as our patient who had experienced more than 10 recurrences of zosteriform lesions.

Identification of the specific virus causing aseptic meningitis is not always straightforward; some viruses that cause the disease are more prevalent than others at certain times of the year. For example, enterovirus infections occur in the summer and account for most cases of aseptic meningitis. togavirus infections occur in late summer and autumn (the mosquito and tick season), whereas lymphocytic choriomeningitis virus infections occur in early winter (when mice come indoors). Mumps and other parainfluenza virus and influenza virus infections are most frequent in the winter, and chickenpox (varicella) occurs predominantly in the spring. In contrast, HSV meningitis occurs year-round. Diagnosis is aided by a confirmed history of recurrent genital lesions or recurrent neuropathy, which suggests HSV-2 (Table). The profile of our patient further implicates HSV-2 as the causative agent of aseptic meningitis when zosteriform lesions are concomitant or there is a history of recurrent zosteriform eruptions. Diagnosis can be proved by the demonstration of PCR-amplifiable HSV-2 DNA in the CSF.

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