PEDIATRIC NEUROLOGY BRIEFS A MONTHLY JOURNAL REVIEW

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INFECTIOUS DISORDERS

FOCAL ENCEPHALITIS FOLLOWING VARICELLA-ZOSTER VIRUS REACTIVATION WITHOUT RASH

Staff members at the Mayo Clinic, Rochester, MN, and University of Iowa Children's Hospital, Iowa City, report a healthy 22-year-old man with a focal encephalitis following varicella-zoster virus (VZV) reactivation without rash, triggered by varicella vaccination required for employment in a hospital. Between 2 to 3 weeks following vaccination he developed intermittent left temporal headaches and 2 days later, he had a seizure. MRI demonstrated a T2/FLAIR hyperintense and T1 hypointense lesion involving the left inferior temporal sulcus. A routine EEG recorded 2 subclinical left temporal lobe seizures. The differential diagnosis favored a low-grade astrocytoma or oligodendroglioma. The pathology report on the resected lesion excluded a tumor, and perivascular inflammation of leptomeningeal vessels indicated a viral etiology for the gliosis. VZV immunostaining of brain tissue and VZV serology were positive for VZV antigens, and wild-type VZV sequences were detected. He was treated with valacyclovir. At follow-up examination 1 year later he was free of CNS symptoms. Further examination of childhood records revealed that at 6 months of age he was exposed to varicella in his older sister, but he had no exanthem at that time. At 27 months of age he had developed a left-sided T6/T7 dermatomal rash and a diagnosis of herpes zoster. It was concluded that this case represents VZV reactivation, most likely in the trigeminal ganglion, in the absence of clinical herpes zoster. (Halling G, Giannini C, Britton JW, et al. Focal encephalitis following varicella-zoster virus reactivation without rash in a healthy immunized young adult. Jrnl Infect Dis 2014 Sep 1;210(5):713-6).

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COMMENTARY. The close temporal proximity of the VZV reactivation with 2 varicella vaccinations during the previous 2 to 3 weeks is strong suggestive evidence for a causal association. However, the authors argue in favor of a coincidental association since virology sequencing showed that the VZV infection in the brain was caused by wild-type VZV. Wild-type VZV has been isolated from vesicles in patients with herpes zoster after immunization, indicating that herpes zoster in immunized people also may result from natural varicella infection that occurred before or after immunization [1]. They suggest that the varicella vaccination, rather than having a detrimental effect, may have prevented further inflammation by stimulating a rapid anti-VZV immune response. Furthermore, the surgical excision of the area of inflammation in the temporal lobe may have prevented further extension of viral infection.

Link between VZV reactivation and CNS disease. Neurological complications of VZV reactivation include zoster induced postherpetic neuralgia, myelitis, meningoencephalitis, VZV vasculopathy, and stroke [2]. Stroke as a complication of VZV reactivation is reported in the elderly but is rare in childhood [3]. When these complications occur without rash (zoster sine herpete), VZV-induced disease is diagnosed by detection of VZV DNA or anti-VZV antibody in CSF. Awareness of the expanding spectrum of neurological complications of VZV reactivation leads to earlier diagnosis and antiviral treatment. A case of congenital varicella syndrome (CVS) in a male infant presented with generalized clonic cerebral seizures at age 4 months [4]. An intracerebral viral reactivation following intrauterine VZV infection was suspected and confirmed. Antiviral treatment was aimed at preventing progression of the disease. CVS presents with skin lesions, neurological defects, eye diseases, and limb hypoplasia.

References.

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INTRACRANIAL HYPERTENSION

SYMPTOMS AND ETIOLOGIES OF PSEUDOTUMOR CEREBRI

Investigators from Ankara Pediatrics, Turkey, evaluate the clinical symptoms and etiology in records of 53 patients (32 female) diagnosed with pseudotumor cerebri (PTC) in a child neurology department between 2005 and 2012. Mean age at presentation was 10.9 years (range 3-17) and one half were age 11 years or younger. Prepubertal patients (under 12 years old) were male in >50%, while 74% patients at puberty were girls. Etiology was undetermined or idiopathic in 30 and symptomatic in 23. Obesity rate was 41% for pubertal patients and 31% for prepubertal patients. Obesity was not related to etiology or puberty. In idiopathic cases, headache was the most common symptom (in 88%), nausea and/or vomiting in 30%, diplopia in 28%, and dizziness in 9%. Papilledema was found in 100%, and VI or VII nerve palsy in 11.3%. An etiologic factor for symptomatic PTC was identified in 43% of patients and included cerebral venous sinus