POST-INFECTIOUS CNS DISORDERS

PSEUDOTUMOR CEREBRI COMPLICATING MEASLES

A novel case of pseudotumor cerebri (PTC) that presented in an 8-year-old girl 3 weeks after measles is reported from Ondokuz Mayis University, Samsun, Turkey. She was admitted with severe headache, vomiting, retroorbital pain and photophobia. Examination revealed normal temperature and mental orientation, and no meningeal irritation. Visual acuity was 20/20, visual fields were full, and funduscopic examination showed bilateral papilledema with tortuous vessels. EEG, MRI and cranial CT performed 7 days after hospitalization were normal, and MR venography performed 27 days later showed no signs of sinus vein thrombosis. CSF opening pressure on lumbar puncture was 30 cm H2O. Laboratory studies were negative except for elevated serum varicella immunoglobulin G (243 AU) and measles immunoglobulin M (183 AU) titers. Following treatment with prednisolone, mannitol, acetazolamide and furosemid, headache improved in 5 days, and in 14 days, the patient was symptom free, with normal CSF pressure (6-12 cm H2O). Withdrawal of prednisolone and reduction in acetazolamide were followed by relapse and temporary renewal of therapy. A repeat lumbar puncture 2 months later revealed a CSF pressure of 12 cm H2O, and funduscopic examination was normal. A post measles autoimmune-mediated vasculitis is postulated as the cause of PTC in this child. (Tasdemir HA, Dilber C, Totan M, Onder A. Pseudotumor cerebri complicating measles: A case report and literature review. **Brain Dev** July 2006;28:395-397). (Respond: C Dilber, e-mail: cengizd@omu.edu.tr).

COMMENT. Reports of infectious or post-infectious disorders associated with pseudotumor cerebri (PTC) have included sinusitis, otitis media, mastoiditis, HIV, Lyme disease, SSPE (Tan H et al. **J Child Neurol** 2004;19:627-629), and varicella (Konrad D et al. **Eur J Pediatr** 1998;157:904-906). PTC was initially considered in a case of zoster-associated intracranial hypertension in a 14-year-old female who presented with headache, vomiting, rash and papilledema (Millichap JJ, Freeman JL. **Pediatr Neurol** 2005;32:211-212). Despite the absence of fever or meningism and the child's high body mass index in keeping with PTC, the finding of CSF pleocytosis and elevated protein was inconsistent with the diagnosis, and varicella-zoster virus reactivation was invoked as the cause. Given the elevated serum varicella immunoglobulin G, a measles-induced reactivation of varicella virus in the present case might have been considered by repeat serology during convalescence (For reference to viral reactivation by concurrent infection, see Suga S et al. **J Med Virol** 1992;38:278-282; and Hall CB, Epstein LG, et al. **N Engl J Med** 1994;331:432-438).

VARICELLA AND STROKE

Four cases of cerebrovascular disease following varicella infection are reported from the Giannina Gaslini Children's Hospital and Research Institute, Genoa, Italy. All were male, and ages ranged from 6 months to 6 years. Primary varicella zoster viral (VZV) infection was confirmed by detection of specific IgM antibodies. None was immunocompromised. Three

children presented with hemiparesis and one with facial paresis, hypotonia and gait impairment. Neuroimaging with MRI, MRA, and/or CT showed occlusion or stenosis of the middle cerebral artery in 3 cases or nucleo capsular signal alteration without vessel occlusion in one. The incubation time from rash to onset of stroke was 2 to 30 days. Congenital prothrombotic abnormalities present in 2 cases, and a concurrent streptococcal bacteremia in one other may have contributed to the cerebrovascular disease. Treatment included acyclovir and acetylsalicylic acid (ASA). One patient developed cerebral hemorrhage and ASA was discontinued. The 3 patients with hemiparesis had persistent deficits on follow-up. The facial palsy resolved. (Losurdo G, Giacchino R, Castagnola E, et al. Cerebrovascular disease and varicella in children. **Brain Dev** July 2006;28:366-370). (Respond: Dr Raffaella Giacchino, Infectious Diseases Unit, G Gaslini Children's Hospital, Largo G Gaslini 5, 16147 Genoa, Italy).

COMMENT. Pathogenic mechanisms for varicella-associated stroke include vasculitis, acquired protein S deficiency, antiphospholipid and anticardiolipin antibodies, and lupus anticoagulant activity. No common mechanism is described. Antiphospholipid antibody was positive in 2 of the above 4 cases; it became negative after 1 month follow-up. VZV infection is a rare but significant cause of stroke in children.

Varicella and delayed stroke. Acute hemiplegia developed 7 weeks to 4 months after varicella infection in 4 children in Japan (Ichiyama T et al. **Pediatr Neurol** 1990;6:279-281). Carotid angiography showed segmental narrowing and occlusion of the middle cerebral artery, and cerebral angiitis was cited as the cause. The frequency of delayed stroke was estimated at 1 in 6500 cases of varicella in Japan.

VESTIBULAR NEURITIS AND ADENOVIRUS

A 4-year-old boy with vestibular neuritis and a serological diagnosis of adenovirus infection is reported from the University of Siena, Italy. He was admitted with paroxysmal vertigo, vomiting and unsteadiness, leaning left on attempted walking or during the Romberg test. A similar episode had occurred one year before; symptoms lasted 1 week and the patient was not hospitalized. On present examination, spontaneous horizontal nystagmus to the right decreased with visual fixation. EEG and ENT examinations were normal, and showed no evidence of seizure disorder or middle ear disease. Family history was negative for migraine. On partial recovery within a few days, a water caloric test showed no response in the left ear. Serum antibody complement fixation test for a battery of viral infections was positive only for adenovirus, the titer on admission being 1/16, 1/8 at 2 weeks, and undetectable at 4 weeks. PCR for adenovirus on lymphocytes and saliva was negative. Brain MRI was normal. Minor unsteadiness, nystagmus, and absent caloric response in the left ear persisted at 1 month. (Zannolli R, Zazzi M, Muraca MC, et al. A child with vestibular neuritis. Is adenovirus implicated? **Brain Dev** July 2006;28:410-412). (Respond: Dr Raffaella Zannolli: e-mail: zannolli@unisi.it).

COMMENT. This case report is suggestive of an etiologic role for adenovirus infection in vestibular neuritis. Detection of the virus by pharyngeal culture or antigen is the preferred diagnostic method for adenovirus (AAP Red Book, 25th ed, 2000;162-163), and the negative PCR on saliva may militate against a recent infection. A reactivation of a previous