

ENCEPHALOPATHIES

GLUTAMIC ACID DECARBOXYLASE AUTOANTIBODIES-RELATED ENCEPHALITIS

Researchers at University Hospital of Geneva, Switzerland, report a case of glutamic acid decarboxylase autoantibodies (GADA)-related encephalitis in a 6-year-old girl and review the literature. She developed refractory seizures at age 25 months followed by mental regression and type 1 diabetes mellitus, in association with elevated plasma and CSF GADAs. A dramatic decrease in serum GADA levels was observed 2 weeks after plasmapheresis and was maintained by oral prednisone and 2 plasmapheresis sessions per week. At 8 years, her gait was normal and she could understand and speak short sentences, but seizures were only partially controlled. Only two previous reports were published, in 2002 and 2009. One child recovered completely within 3 months of disease onset, despite persistently high values of plasma GADA. (Korff CM, Parvex P, Cimasoni L, et al. Encephalitis associated with glutamic acid decarboxylase autoantibodies in a child. *Arch Neurol* Aug 2011;68(8):1065-1068). (Respond: Christian M Korff MD, Pediatric Neurology, Child and Adolescent Department, University Hospital of Geneva, 6 Rue Willy-Donze, CH-1211 Geneva 14, Switzerland. E-mail: christian.korff@hcuge.ch).

COMMENT. The concept of epilepsy caused by autoantibodies to specific neuronal membrane proteins is a growing area of interest in epilepsy research. (Vincent A et al. *Curr Opin Neurol* 2010;23(2):144-150). Voltage-gated potassium channels, N-methyl-D-aspartate receptors, and glutamic acid decarboxylase are involved in some cases of limbic encephalitis. Patients whose seizures do not respond to conventional anticonvulsants should be tested for autoantibodies and considered for a trial of immunotherapy.

INFECTIOUS DISEASES

***MYCOPLASMA PNEUMONIAE* POST-ENCEPHALITIC EPILEPSY**

Researchers at Chang Gung Children's and Memorial Hospitals, Taoyuan, Taiwan, investigated the clinical manifestations, laboratory, EEG, and neuroimaging features of *M pneumoniae*-related encephalitis and its prognosis. Ninety-nine patients seen between Jan 2001 and June 2010 were all positive by serology, and 47 (47.5%) developed postencephalitic epilepsy. Onset ranged from 9 months to 14 years (mean age 6.8 years). Seizures occurred in the acute phase in 53 (53.5%), most commonly focal with secondary generalization (40%). Status epilepticus occurred in 25%, refractory in 15%. Initial symptoms were fever, altered consciousness, and personality or behavior change. Elevated CSF protein was a risk factor for postencephalitic epilepsy. Initial EEG was most commonly focal/diffuse cortical dysfunction (37%) and focal epileptiform discharge (26%). Focal epileptiform EEG in the acute phase increased the risk of developing postencephalitic epilepsy 5-fold. Follow-up ranged from 6-131 months. Significant risk

factors for postencephalitic epilepsy include seizures in the acute illness and focal epileptiform discharges in the initial EEG. All children with status epilepticus during hospitalization developed postencephalitic epilepsy. Of the 47 children with postencephalitic epilepsy, 19 (40%) had intractable seizures, and 40% had favorable outcomes. (Lin J-J, Hsia S-H, Wu C-T, Wang H-S, Lin K-L. *Mycoplasma pneumoniae*-related postencephalitic epilepsy in children. **Epilepsia** Aug 2011;E-pub ahead of print). (Respond: Kuang-Lin Lin, Division of Pediatric Neurology, Chang Gung Children's Hospital, 5 Fu-Shin Street, Kwei-Shan, Taoyuan 333, Taiwan. E-mail: linchg@adm.cgmh.org.tw).

COMMENT. Previous reports of postencephalitic epilepsy from centers in Taiwan have examined clinical and prognostic factors (Chen YJ et al. **J Child Neurol** 2006;21(12):1047-1051). Of 44 patients, 20 had a favorable outcome and 24 a poor outcome. Factors indicating a poor prognosis during the acute encephalitis phase were 1) status epilepticus as the first seizure ($p<0.005$), 2) slow background activity ($p<0.001$) and multifocal spike discharges on EEGs ($p<0.01$), and 3) herpes simplex viral encephalitis ($p<0.01$). Herpes simplex virus type 1 is a major pathogen causing postencephalitic epilepsy. Early identification of the HSV-1 and treatment with antivirals may improve the outcome and prevent the epilepsy complication.

Hippocampal dynorphin and epilepsy. Animal studies at UC-Irvine have investigated the relation of the dynorphin promoter system in the hippocampus to HSV-1 precipitated seizures. Reduced dynorphin expression in the dentate gyrus due to HSV-1 infection leads to an increased propensity to seizures. The loss of dynorphin immunoreactivity in the hippocampus is independent of a direct viral induced cell loss, suggesting a neurochemical basis for viral-induced seizures. Viruses are environmental triggers for seizures. (Solbrig MV et al. **Neurobiol Dis** 2006;23(3):612-620).

Influenza vaccination (and/or *Mycoplasma pneumoniae*) as cause of transverse myelitis. (Ambrose CS et al. **Arch Neurol** Aug 2011;68(8):1085-6). In response to a report of transverse myelitis (TM) in a 27-year-old woman 4 days after vaccination with monovalent A (H1N1) live, nasal attenuated influenza vaccine, Ambrose et al comment that the patient may have had mycoplasma pneumonia 20 days before symptom onset. M pneumoniae is a well-established cause of TM, with an interval of 4 to 30 days between respiratory illness and neurologic symptoms. Possible mycoplasma infection and influenza vaccination should be considered as potential etiologies of TM in this patient.

VASCULAR DISORDERS

ACUTE ISCHEMIC STROKE IN CHILDREN VS YOUNG ADULTS

Clinical characteristics, stroke etiology, and outcome (modified Rankin scale [0-6] score at 3-6 months) in children (1 month-16 years) and young adults (16-45 years) with arterial ischemic stroke (AIS) were compared in a multicenter study at University of Bern and other centers in Switzerland. Using the Swiss NeuroPediatric Stroke Registry and Bernese stroke database, data collected prospectively from Jan 2000 to Dec 2008