

INFECTIOUS DISORDERS

EPSTEIN-BARR VIRUS AND OPSOCLONUS MYOCLONUS SYNDROME

A new case of opsoclonus-myoclonus syndrome (OMS) secondary to Epstein-Barr virus (EBV) is reported in an adult, and five previously documented cases (3 children, 2 adults) are reviewed from the University of Miami, FL. In the current case, acute onset oscillopsia, tremulousness, and ataxia were preceded by a 10 day history of fever, sore throat, and macular-morbilliform rash. Examination showed multidirectional eye saccades, persisting on eye closure and in sleep, truncal ataxia, and limb myoclonus. Brain MRI and EEG were negative. Head, chest, abdomen and pelvic CT scan and whole-body PET scan were negative. EBV capsid antigen test was positive at 1:640. PCR for EBV genome in the CSF and blood were positive. Methylprednisolone and immunoglobulin IV for 3 to 5 days were without immediate effect, and recovery occurred over 7 weeks. Examination at 1 year follow up was normal. In the 5 previously reported cases of OMS and EBV, none showed EBV in the CSF. (Verma A, Brozman B. Opsoclonus-myoclonus syndrome following Epstein-Barr virus infection. Neurology April (1 of 2) 2002;58:1131-1132). (Reprints: Dr Ashok Verma, 1150 NW 14th St, Ste 701, Miami, FL 33136).

COMMENT. In children OMS is usually a sign of neuroectodermal tumor and rarely is associated with infection. In adults, lung, breast, or pelvic cancer is usually uncovered, and infection occurs in about 20% of cases (authors' citation of literature). The identification of an infectious cause is rare, and EBS has been reported in only 5 previous cases. A postinfectious or parainfectious process is suggested in the present case with EBV. See Ped Neur Briefs Nov 2001;15:87, for an article on neurobehavioral outcome of OMS.

CYTOTOXIC T LYMPHOCYTES IN RASMUSSEN'S ENCEPHALITIS

The role of cytotoxic T lymphocytes and other mechanisms in the pathogenesis of Rasmussen's encephalitis (RE) is reviewed at the University of Vienna, Austria, and the University of Bonn, Germany. The densities of T cells, microglial nodules and glial fibrillary acidic protein positive astrocytes in surgically collected brain specimens are inversely correlated with disease duration. Inflammation and T cells are most pronounced at the earlier stages of RE. Brain specimens tested by immunochemistry and polymerase chain reaction for the presence of viruses such as enterovirus, Epstein Barr, cytomegalovirus and herpes simplex have failed to show a causal link between a specific virus and RE. Some patients with RE have high-titre anti-GLuR3 antibodies, and treatment with plasma exchange may result in a decrease in seizure frequency. However, anti-GLuR3 antibodies are not specific for RE, but may occur with noninflammatory focal epilepsy. The recent recognition of cytotoxicity by CD8/granzyme-B-positive T lymphocytes in brain biopsies or surgical specimens suggests that an autoimmune response mediated by cytotoxic T lymphocytes may be a key mechanism in the etiology of RE. (Bauer J, Bien CG, Lassmann H. Rasmussen's encephalitis: a role for autoimmune cytotoxic T lymphocytes. Current Opinion in Neurology 2002;15:197-200). (Respond: Dr Hans Lassmann, Brain Research Institute, Div of Neuroimmunology, University of Vienna, Spitalgasse 4, A-1090 Vienna, Austria).