NEUROLOGIC AND MRI ABNORMALITIES IN ACUTE DISSEMINATED ENCEPHALOMYELITIS AND RESPONSE TO PLASMAPHERESIS

The relation between the clinical course and MRI findings and response to plasmapheresis were determined by a retrospective record review of 13 children with acute disseminated encephalomyelitis (ADEM) admitted to St Christopher’s Hospital for Children, Philadelphia, PA, during 1998-2003. Six of 12 children responded to initial treatment with high-dose methylprednisolone and IV immunoglobulin; one improved spontaneously. The 6 who were unresponsive had a progressive neurologic course, and 5 had delayed onset of MRI abnormalities involving deep gray matter and brainstem. These 5 recovered slowly following plasmapheresis, with residual neurologic deficits. (Khurana DS, Melvin JJ, Kothare SV et al. Acute disseminated encephalomyelitis in children: discordant neurologic and neuroimaging abnormalities and response to plasmapheresis. Pediatrics August 2005;116:431-436). (Reprints: Divya S Khurana MD, Section of Neurology, St Christopher’s Hospital for Children, Erie Avenue at Front Street, Philadelphia, PA 19134).

COMMENT. MRI abnormalities may appear late in the course of ADEM, leading to a delay in diagnosis and treatment. In almost half the patients in the above series, a significant lag occurred, up to a few weeks, between the presentation of symptoms with fever, ataxia, paresis and mental changes, and the appearance of MRI lesions, usually involving the basal ganglia, thalami, and brainstem, as well as subcortical white matter. Late development of MRI lesions involving deep gray matter and brainstem may be predictive of a prolonged course and lack of response to corticosteroids. The absence of white-matter changes in the MRI up to 3 weeks after onset of symptoms does not exclude a diagnosis of ADEM. Plasmapheresis is recommended in patients with a severe progressive course, unresponsive to corticosteroids and IV immunoglobulin.

BRAIN TRAUMA

LATE SEQUELAE OF INFLECTED BRAIN INJURY IN INFANTS

Twenty five children with inflicted traumatic brain injury (TBI) seen in Scotland between 1980 and 1999 were followed prospectively with neurologic and cognitive examinations performed at the Department of Child Life and Health, University of Edinburgh, Scotland. Sixty eight per cent of survivors showed abnormalities at mean follow-up of 59 months; impairments were severe with complete dependency in 36%, moderate in 16%, and mild in 16%. Outcome correlated with the Pediatric Trauma and Glasgow Coma Scores but not with age at injury or mechanism of injury. Inflicted TBI has a poor prognosis and correlates with severity of injury. Behavior problems in 52% began to manifest at age 2 to 3 years, and learning difficulties and attention and memory deficits were recognized only when the child attended school. (Barlow KM, Thomson E, Johnson D, Minns RA. Late neurologic and cognitive sequelae of inflicted traumatic brain injury in infancy. Pediatrics August 2005;116:e174-e185). (Reprints: Dr Karen M Barlow, University of Calgary, Alberta Children’s Hospital, 1820 Richmond Rd, Calgary, Alberta, Canada T2T 5C7).