PEDIATRIC NEUROLOGY BRIEFS
A MONTHLY JOURNAL REVIEW

J. GORDON MILLICHAP, M.D., F.R.C.P., EDITOR

Vol. 13, No. 2 February 1999

INFECTIOUS DISORDERS

ENTEROVIRUS 71 MENINGOENCEPHALITIS

Seven cases of encephalitis and 5 of aseptic meningitis caused by an outbreak of enterovirus 71 (EV71) during the summer of 1997 are reported from the Otsu Municipal Hospital, Shiga, Japan. Skin and mucosal involvement included hand-foot-and-mouth syndrome in 7 and herpangina in 2. The interval between fever and initial CNS symptoms, including headache, nausea, vomiting, and neck stiffness, was 0 to 5 days. Major neurological manifestations, localized to cerebral hemispheres, brainstem, cerebellum, and diencephalon, included seizures in 5, coma or somnolence in 6, truncal ataxia in 5, and diabetes insipidus in 1. MRIs performed in 7 patients were normal in 6 and showed high-intensity lesions in the pons in 1. EEGs showed diffuse high voltage slow wave activity in 6, focal changes in 2, and normal recordings in 4. Evidence of recent EV71 infection relied on unusually high reciprocal EV71 neutralizing antibody titers in 10 patients and a fourfold change in acute and convalescent-phase serum antibody titer in 2. CSF pleocytosis ranged from 8 to 693 cells/mcL. Attempts to detect the viral genome in CSF, using reverse transcriptase-polymerase chain reaction and Southern blot hybridization procedures, were negative. Clinical recovery was complete in 11, and one, a 2-week-old neonate, developed a motor disorder at several months follow-up. (Komatsu H, Shimizu Y, Takeuchi Y, Ishiko H, Takada H. Outbreak of severe neurologic involvement associated with enterovirus 71 infection. Pediatr Neurol Jan 1999;20:17-23). (Dr Hiroshi Komatsu, Department of Pediatrics, Kyoto First Red Cross Hospital, 15-749 Honmachi, Higashiyama-ku, Kyoto 605-0981, Japan).

COMMENT. The nonpolio, RNA enteroviruses include 23 group A coxsackieviruses, 6 group B coxsackieviruses, 31 echoviruses, and 4 enteroviruses (types 68-71). Enteroviruis 71 is associated with hand-foot-and-mouth syndrome, encephalitis, aseptic meningitis, and poliolike paralysis. (AAP 1997 Red Book). The virus is spread from mother to infant at birth, and by fecal-oral and respiratory routes in older children. The incubation period is 3 to 6 days.

EEG in diagnosis and outcome of acute encephalitis was

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investigated at Helsinki University Hospital, Finland. An analysis of 204 EEG recordings from 98 consecutive acyclovir-treated patients with acute encephalitis found that, in the acute phase, clinical epileptic seizures, and focal abnormalities and periodic complexes in the EEG, but not diffuse background slowing, were predictive of a poor outcome. In follow-up EEG recordings, diffuse slowing was significantly associated with poor outcome. The EEG is valuable in assessment of progression in the level of consciousness and epileptic activity in the unconscious patient with encephalitis. (Siren J, Seppalainen A-M, Launes J. Is EEG useful in assessing patients with acute encephalitis treated with acyclovir? Electroenceph clin Neurophysiol Oct 1998;107:296-301).

Of interest, the neonate in the above Japanese study, whose neurologic exam had returned to normal one week after the onset of convulsions and the acute phase of enterovirus 71 meningoencephalitis, had repeated EEGs revealing no abnormalities. The developing motor disorder at several months follow-up could be explained as a lower motor neuron poliolike muscle weakness.

HIV INFECTION PRESENTING WITH STROKE AND SEIZURES

Two children, ages 2 years and 2 months, presenting with acute hemiparesis and focal seizures as the initial manifestations of human immunodeficiency virus (HIV) infection are reported from Ramathibodi Hospital, Mahidol University, Bangkok, Thailand. CT in the 2-year-old with hemiparesis showed acute infarction of the right thalamus and internal capsule, and old infarction with bilateral basal ganglia calcification and cerebral atrophy. MRI revealed narrowing of the right middle cerebral artery. The 2-month-old infant with a one week history of multiple focal seizures and impaired consciousness was jaundiced, and the CT revealed hemorrhagic infarction of the right cerebral hemisphere, with acute intraparenchymal bleeding. MRI showed narrowing and irregularity of the right middle cerebral and internal carotid arteries. HIV infection was documented only after the onset of acute neurologic manifestations. Neither coagulopathy nor other cause of stroke was identified. Tests for opportunistic infections were negative. (Visudtibhan A, Visudhiphan P, Chiemchanya S. Stroke and seizures as the presenting signs of pediatric HIV infection. Pediatr Neurol Jan 1999;20:53-56). (Respond: Dr Anannit Visudtibhan, Department of Pediatrics, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok 10400, Thailand).

COMMENT. Stroke may be the first clinical manifestation of HIV infection in infants and young children. A test for HIV should be included in the work-up of cases of stroke in children at risk of HIV infection.

My colleague, Dr Leon Epstein, Head of the Division of Neurology, Children's Memorial Hospital, Chicago, has written extensively on the neurologic manifestations of HIV infection in children (In Pediatrics 1986;78:678-687, and other subsequent publications). A progressive encephalopathy is reported most commonly. In his experience, seizures are not a prominent symptom of HIV infection, and opportunistic infections, including measles, must be excluded as possible alternative causes of neurologic manifestations.

In a study reviewed in Ped Neur Briefs (June 1998;12:44), Cooper ER et al. report encephalopathy in 21% of 128 HIV-perinatally infected children, with a mortality of 41%. Failure to gain weight predated the onset of encephalopathy in infected infants. A high viral load during infancy, failure to thrive, and early signs of hepatomegaly and lymphadenopathy are risk factors for HIV