INFECTIOUS DISORDERS

AGE-RELATED DIFFERENCES IN CLINICAL FEATURES OF NEUROCYSTICERCOSIS

Clinical, radiologic, and inflammatory features of neurocysticercosis (NC) in 92 pediatric (<15 years) and 114 adult Mexican patients were compared in a study at three hospitals in Mexico City. Diagnosis was based on CT and/or MRI, before treatment. Ages of pediatric patients ranged from 11 months to 14 years. Symptoms in order of frequency in both age groups were seizure, headache, intracranial hypertension (ICH), and focal deficits. Seizures were more frequent in children (80.4% vs 56.1%; p<0.0001), and headache and ICH were more frequent in adults (35.1% vs 21.7%; p=0.04). The cause of ICH differed in the two groups: in children, an increased inflammatory response to subarachnoid parasites, and in adults, obstruction of CSF circulation by cysticerci in basal cisterns or ventricles. Colloidal parenchymal cysts were single in children with seizures, and multiple in adults. The number, location, and stage of parasites differed between the 2 age populations: a single colloidal or calcified parenchymal parasite was most frequent in children, and multiple parasites in basal cisterns or ventricles were most frequent in adults. CSF inflammatory response was significantly greater in adults than in children (p=0.02). (Saenz B, Ruiz-Garcia M, Jimenez E et al. Neurocysticercosis. Clinical, radiologic, and inflammatory differences between children and adults. Pediatr Infect Dis J Sept 2006;25:801-803). (Respond: Agnes Fleury M D, Instituto Nacional de Neurologia y Neurocirugia, Av Insurgentes Sur 3877, Col La Fama, Del Tlalpan, CP 14269, Mexico, DF, Mexico).

COMMENT. Neurocysticercosis in Mexican children presents with a single degenerating parasite located in the parenchyma, while multiple viable parasites located in basal cisterns or ventricles are more common in adult patients.

Cysts of Taenia solium in the CNS (neurocysticercosis) are a leading cause of epilepsy in some countries. The host reaction to degenerating cysts in the brain may cause, in addition to seizures, meningitis, obstructive hydrocephalus, and behavioral disorders. Cysts in the spinal column can cause gait disturbance, pain, or transverse myelitis. Subcutaneous cysts appear as nodules, and ocular cysts cause visual impairment. Ocular and spinal cysts generally are not treated with anthelmintic drugs, which will exacerbate inflammation. Ophthalmic examination to rule out ocular cysts is recommended before beginning treatment with albendazole. (AAP. Red Book: 2006 Report of the Committee on Infectious Diseases. 27th ed;644-646).

VACCINE-INDUCED ACUTE METABOLIC CRISIS

Acute metabolic crisis occurring in 7 children (6 at ages 3 - 9 months, and 1 at 5 years), between 3 and 12 hours after administration of Japanese encephalitis, diphtheria, and tetanus toxoids, and acellular pertussis, hepatitis B, and measles vaccines, is reported from Peking University First Hospital, Beijing, China. Preexisting primary diseases, not known before vaccination, included Leigh disease in 3 infants, glutaric aciduria type 1,
methylmalonic aciduria, and 21-hydroxylase deficiency in one 3 month old and one previously diagnosed 5 year old. Four patients were previously healthy, and suffered fever, seizures, coma, acidosis, and hypoglycemia soon after vaccination, all succumbing to respiratory failure before 2 years of age. Symmetric foci, cystic cavitations with neuronal loss, and vascular proliferation were found at autopsy. (Yang Y, Sujan S, Sun F et al. Acute metabolic crisis induced by vaccination in seven Chinese patients. Pediatr Neurol 2006;35:114-118). (Respond: Dr Yang, Department of Pediatrics, Peking University First Hospital, Beijing, 100034, China).

COMMENT. One patient had a family history of infantile deaths and was diagnosed with congenital adrenal hyperplasia and 21-hydroxylase deficiency before vaccination. A second patient subsequently diagnosed with 21-OH deficiency was malnourished in infancy, and a 3 month-old with methylmalonic aciduria was hypotonic. The remaining 4 infants appeared normal before vaccination. Vaccination in infants with unrecognized metabolic disorders carries a risk of metabolic crisis. Infants who are malnourished, slow in development, hypotonic or have symptoms suggesting a metabolic disorder should receive appropriate laboratory tests before vaccination.

NEURAL CORRELATES OF AUTISM

GAIT DISORDERS IN AUTISM

Gait in 11 children with autism (age range 4 – 7 years) and 11 controls was analyzed, using the GAITRite electronic walkway connected to a computer in a study at Department of Psychological Medicine, Monash University, Clayton, Victoria, Australia. Children with autism had difficulty walking tandem, reduced stride regularity, and variable velocity, compatible with cerebellar dysfunction. They were also less coordinated and erratic in their movements. Postural abnormalities in head and trunk suggested basal ganglia involvement. Abnormal gait is proposed as a useful clinical screening test for autism. (Rinehart NJ, Tonge BJ, Iansek R et al. Gait function in newly diagnosed children with autism: cerebellar and basal ganglia related motor disorder. Dev Med Child Neurol Oct 2006;48:819-824). (Respond: Dr Rinehart, Department of Psychological Medicine, Monash University, Level 3, Block P, Monash Medical Centre, Clayton, Victoria 3168, Australia).

COMMENT. Neuropathological findings in autism (Palmen SJMC et al. Brain 2004;127:2572-2583; Ped Neur Briefs Dec 2004;18:89-90) document an increase in head circumference, brain weight and brain volume, decrease in Purkinje cells in the cerebellum, and dysgenesis in the cerebral cortex. Autism is a neurodevelopmental, genetically determined disorder characterized by impairments in social interaction and communication skills, cognitive rigidity, abnormal language development, and repetitive, stereotypical behaviors.

A significant increase in prevalence of autism and pervasive developmental disorders (PDD), from 14.7 to 30.8/10,000 between 1980 and 1993, is reported in France. Morphogenetic anomalies (chromosomal, CNS and other anomalies), and hospitalization rates in the neonatal period are also increased in children with PDD (Guillem P et al. Dev Med Child Neurol November 2006;48:896-900).