TRAUMATIC BRAIN DISORDERS

CAUSE OF SUBDURAL HEMORRHAGE IN INFANTS

The frequency of child abuse and unintentional injury as a cause of infant and toddler subdural hedmorrhage (SDH) was determined by a prospective study of 66 cases admitted to Harborview Medical Center, a level I pediatric trauma center, and the Children's Hospital and Regional Medical Tertiary Care Center, Seattle, WA, from March 1995 through December 1998. The cause of SDH was confirmed abuse in 39 (59%), unintentional injury (motor vehicle accident or major trauma) in 15 (23%), and indeterminate in 12 (18%). The mean age at injury for abuse and accidental cases was 8.7 and 19.1 months, respectively. The presenting history for abuse injury was a minor fall or unknown reason in 33 (84%) of 39 patients, while that of unintentional injury was a motor vehicle accident or other major trauma in all cases. Chronic SDH or mixed acute and chronic SDH occurred only in abused children (17 [44%]), and in those with indeterminate injuries (8 [67%]). Long bone and/or rib fractures were present in 20 (51%) of abuse cases, and in only 1 of unintentional, accidental head injury cases. Retinal hemorrhages were diagnosed in 28 (72%) of abuse cases, none of 7 indeterminate cases, and in 1 of 3 accidental cases examined. The authors conclude that any infant or toddler with SDH or subdural effusion, in the absence of motor vehicle or other major trauma, should be checked for child abuse. (Feldman KW, Bethel R, Shugerman RP et al. The cause of infant and toddler subdural hemorrhage: a prospective study. Pediatrics September 2001;108:636-646). (Reprints: Kenneth W Feldman MD, 2101 East Yesler Way, Seattle, WA 98122).

COMMENT. Unintentional, accidental trauma accounts for almost one quarter of cases of subdural hemorrhage (SDH) in infants and toddlers, and 60% are related to a motor vehicle accident. Falls are an infrequent cause, occurring in only 4%, and always from heights greater than 10 feet on to hard surfaces. When obvious accidental cause is ruled out, abuse is most likely in 76% of cases. Associated reasons to suspect abuse include a younger age group, absence of a significant history of trauma, evidence of chronic SDH, retinal hemorrhages, long bone or rib fractures, and multiple injuries. An infant or young child presenting with a history of head injury following a minor fall and signs of bruising or other evidence of trauma should receive a head CT scan and be evaluated for child abuse.

INFECTIOUS DISORDERS

TREATMENT OF NEUROCYSTICERCOSIS WITH SUBARACHNOID CYSTS

The medical treatment of 33 patients, 8 to 81 years (mean, 48y), with neurocysticercosis (NC) complicated by giant subarachnoid cysts (50mm or larger) and intracranial hypertension was evaluated at the Hospital de Especialidades, Mexico City. All received albendazole, 15mg/kg/d for 4 weeks, and dexamethasone. The treatment was repeated with a second course in one half the cases. Ten also received praziquantal 100mg/kg/day/4 weeks. Only 5 had undergone surgery for the cysts before receiving medical treatment. All patients showed improvement after a median follow-up of 59 months (range, 7 to 102 months), and the cysts had resolved or became calcified. Four required a further course of treatment. Anticonvulsant medications were continued in only 11 of 22 with a history of seizures. Ventriculoperitoneal shunt was performed in 15

patients with hydrocephalus. Sequelae from the cysts included optic atrophy, stroke, or diplopia in 4 patients. Medical treatment alone can be effective in the treatment of NC with giant subarachnoid cysts. Surgical removal of cysts is reserved for patients with life-threatening intracranial hypertension despite treatment with corticosteroids. (Proano JV, Madrazo I, Avelar F et al. Medical treatment for neurocysticercosis characterized by giant subarachnoid cysts. N Engl J Med September 20, 2001;345:879-885). (Reprints: Dr Proano, Santiago Valverde No 68, Col Presidentes Ejidales, CP 04470, Mexico DF, Mexico).

COMMENT. Patients with neurocysticercosis with giant cysts who respond to corticosteroids or shunting should receive cesticidal drug treatment. The majority will respond to one or more courses of albendazole or praziquantal, and surgical removal of cysts may be necessary in a minority with persistent intracranial hypertension.

Human cysticercosis caused by the larvae of *T solium* is a leading cause of epilepsy in underdeveloped countries and is uncommon in the United States. Diagnosis of taeniasis is by demonstration of ova in feces or perianal swab. Diagnosis of neurocysticercosis is by head CT or MRI. An enzyme immunotransfer blot assay is available to detect antibody to *T solium* in serum or CSF. Treatment is individualized based on the viability of cysts. For patients with calcified nonviable cysts, symptomatic therapy with anticonvulsants and shunting for hydrocephalus are advised. For those with viable parenchymal cysts and inflammation, the role of antiparasitic drugs has not been firmly established. The use of antiparasitic drugs is favored by many clinicians, and the coadministration of corticosteroids for the first few days is recommended to decrease edema and adverse events. (American Academy of Pediatrics Red Book, 25th ed, 2000;pp560-562).

NEUROCUTANEOUS SYNDROMES

AUTISM AND EPILEPSY IN TUBEROUS SCLEROSIS COMPLEX

The relationship between autism and epilepsy and functional brain abnormalities in children with tuberous sclerosis complex (TSC) were examined at the Children's Hospital of Michigan, Wayne State University, Detroit. Based on tests for autism, behavior and intelligence, patients were divided into three groups: autistic (9 cases); mentally-retarded nonautistic (9); and normal intelligence (8). PET studies in the autistic compared to the retarded nonautistic groups showed decreased glucose metabolism in the lateral temporal gyri bilaterally, increased glucose metabolism in the deep cerebellar nuclei bilaterally, and increased a-methyl-tryptophan (AMT) uptake in caudate nuclei. Disturbed communication skills were associated with a history of infantile spasms and glucose hypometabolism in the lateral temporal gyri. Stereotypical behavior and impaired social and communication interaction were correlated with glucose hypermetabolism in deep cerebellar nuclei and increased AMT uptake in caudate nuclei. (Asano E, Chugani DC, Muzik O et al. Autism in tuberous sclerosis complex is related to both cortical and subcortical dysfunction. Neurology October (1 of 2);57:1269-1277). (Reprints: Dr Diane C Chugani, PET Center, Children's Hospital of Michigan, 3901 Beaubien Blvd, Detroit, MI 48201).

COMMENT. In children with TSC, infantile spasms and functional deficits in the temporal cortex are associated with delayed communication skills, and functional imbalance in subcortical circuits are correlated with stereotypical behavior and impaired social interaction.