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INFECTIOUS DISEASES

ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS)

Central nervous system involvement is reported in 61 of 68 infants and children with asymptomatic AIDS (13 with ARC-AIDS-related complex) followed for 1 to 48 months (average, 18 mos) in the Depts of Neurology and Pediatrics at SUNY, Stony Brook, NY. Acquired microcephaly in 34 patients (55%), cognitive deficits in 38 patients (62%), and spastic paresis in 52 patients (85%) were the most frequent CNS complications. Seizures in only 6 patients, ataxia in 6, extrapyramidal rigidity or dystonia in 5, and tremor in 3 were uncommon manifestations of CNS dysfunction. Lymphoma of the CNS, cerebrovascular accidents, and CNS infection occurred in 10 children (15%) who showed a rapidly progressive encephalopathy. Neurological deterioration was subacute but progressive in 11, and began with a plateau in 31 patients. A static course with cognitive deficits was noted in 17 children. CT scans performed on children with a subacute or plateau course frequently showed cerebral atrophy, white matter attenuation and bilateral symmetric calcification of the basal ganglia. Neuropathological findings in 16 of 34 children who died during the study period included an inflammatory response, reactive astrocytosis, foamy macrophages, multinucleated cells, and pyramidal tract degeneration, Calcification of the basal ganglia was present in all cases. (Belman AL et al. Pediatric acquired immunodeficiency syndrome. Neurologic syndromes. AJDC 1988; 142: 29-35).

COMMENT. This longitudinal study of neurological complications of ATDS in children is the largest yet reported and is a valuable informative article for the pediatric neurologist called as a consultant on these cases. The neurological complications of ATDS

PEDIATRIC NEUROLOGY BRIEFS ⁹1988 covers selected articles from the world literature and is published monthly. Subscription requests (\$25 US or £15 UK) may be sent to: Pediatric Neurology Briefs - J. Gordon Millichap, MD, FRCP - Editor, P.O. Box 11391, Chicago, IL 60611, USA, or P.O. Box 1605, London W8 7JD, UK. The Editor is Professor of Neurology and Pediatrics at Northwestern University Medical School, Chicago, Illinois, USA. in children differ from those in adults (Snider WD et al. Ann Neurol 1983; 14: 403). The incidence of CNS involvement is higher in children but unlike adult patients, CNS opportunistic infections appear to be uncommon, occurring in only 8% of the children in this study. A progressive dementing encephalitis (AIDS dementia complex) accompanied by regional cerebral metabolic alterations is the chief neurological complication of AIDS in adult cases (Rottenberg DA et al. <u>Ann Neurol</u> 1987; <u>22</u>: 700). Peripheral neuropathy with painful dysesthesias and retinopathy with cotton-wool spots reported in 16% and 20%, respectively, of adult cases were not recognized in the pediatric group. The static encephalopathy diagnosed in 17 (28%) children was cause for optimism but this was tempered by a later progressive course in 5 similar cases. The incidence of potential AIDS cases in children may be higher than expected: a recent survey showed that 1 in 61 babies born in NY City carried antibodies to AIDS virus, indicating that their mothers were infected. About 40% of infants showing antibodies are estimated to be infected and may develop ATDS.

ACUTE VIRAL ENCEPHALITIS

In a retrospective study of children with viral encephalitis admitted to the University Paediatric Unit, Queen Mary Hospital, Hong Kong, during the past 10 years, 57 satisfied the diagnostic criteria. The presumed viral etiology was determined in 15 (26%) patients of whom 9 had post-infectious encephalitis (mumps-4, measles-1, rubella-1, influenza A-2, influenza B-1). The viruses isolated were: influenza (3 cases), Coxsackie (2), adenovirus (2), mixed adenovirus and cytomegalovirus (1), and herpes simplex (1). Presenting neurologic features included focal signs (33%), convulsion (30%), headache (25%), drowsiness (18%), nuchal rigidity (10%), and coma (9%). Seven (12%) who developed status epilepticus within 24 hrs of admission died subsequently. A total of 16 (28%) died, and 5 were less than 1 year old. Indicators of a poor outcome were an onset in infancy and rapid deterioration in the level of consciousness.

Recovery was complete in 76% of 41 survivors; focal neurological deficits remained in 29% and epilepsy in 4% of 31 with sequelae. Eight children with suspected herpes simplex encephalitis on admission were treated with Acyclovir and none died; one in whom the diagnosis was confirmed developed spastic quadriplegia, mental retardation, and infantile spasms. (Wong V, Yeung CY. Acute viral encephalitis in children. Aust Paediatr J 1987; 23: 339-342).

COMMENT. Unlike previous reports from the USA and Scandinavia which have emphasized herpes simplx virus (HSE) as a major cause of acute sporadic viral encephalitis, only 1 case was identified in this study and none of the 16 patients who died had histological evidence of herpes simplex infection. Brain biopsy, a controversial diagnostic test, was not performed. A consensus panel of the Jrnl of Pediatric Infectious Disease has recommended