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INFECTIOUS DISORDERS

CNS INVOLVEMENT IN HIV INFECTION

The neurological findings in 41 HIV-seropositive children are described from the Departments of Paediatric Neurology and Hematology and Oncology, Zentrum der Kinderheilkunde, Goethe Universitat, Frankfurt, Germany. 23 children were symptomatic. HIV encephalopathy was manifested by acquired microcephaly, developmental regression and progressive motor deterioration. Progressive pyramidal signs, ankle clonus and generalized muscle weakness were documented for 6 children, 4 had extrapyramidal and cerebellar symptoms, and 5 infants of drug-addicted HIV infected mothers had seizures during the neonatal period. Behavioral changes consisting of aggression, anxiety and depression were noted in 5 children. Autistic behavior combined with deterioration in play and loss of language skills developed in one child. 4 patients had recurrent headaches which disappeared in 3 cases after treatment with azidothymidine (AZT). 8 children treated with immunoglobulin therapy (IVIG) and 7 treated with IVIG and AZT have not deteriorated neurologically since therapy began (Schmidt B et al. Central nervous system involvement of children with HIV infection. Dev Med Child Neurol June *1991; 33:535-540*).

> **COMMENT.** The differentiation of static and progressive encephalopathy is sometimes difficult. The majority of HIV infected children have developmental delay and deficits in cognitive functions, language and motor skills. Seizures are an uncommon feature and usually an isolated event associated with fever or opportunistic CNS infection. Opportunistic CNS infections are uncommon with childhood HIV infection. Isolated cases of cerebrovascular involvement including

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intracranial hemorrhage and ischemic infarctions have occurred among children with immune thrombocytopenia or arteritis. Acute hemiplegia secondary to a large infarct is described in a 16-month-old infant with congenially acquired HIV infection (Kugler SL et al. Ped. Neurol May/June 1991; 7:207-10). School achievement and tasks requiring motor speed, attention and concentration were impaired at 4-8 years of age in 15 HIV-1 seropositive children infected through neonatal blood transfusion (Cohen SE et al. Pediatrics July 1991; 88:58-68).

CT scans may demonstrate enlarged CSF spaces and bilateral symmetrical calcification of the basal ganglia and frontal white matter 17-61% (Belman A L et al. A J D C 1988; 142:29-35). Wiley C A et al. report neocortical damage during HIV infection in an autopsy study of adults (Ann Neurol June 1991; 29:651-657). Wizdicks E F M et al. report a fatal disseminated hemorrhagic toxoplasmotic encephalopathy as the initial manifestation of AIDS in a 28 year-old woman (Ann Neurol June 1991; 29:683-686).

MYOPATHY IN ZIDOVUDINE TREATED HIV INFECTION

Elevations in serum CK levels in 80% of 87 children enrolled in a multi-centered trial of oral zidovudine treatment for symptomatic HIV infection are reported from the Department of Pediatrics, University Medical Center, Durham, NC. A myopathy developed in a 3 year old girl with acquired HIV infection who received long-term zidovudine therapy. Muscle biopsy was consistent with a non-inflammatory myopathy but a beneficial response to prednisone suggested an inflammatory component. Severe neurologic impairment related to HIV encephalopathy masked the early signs of proximal muscle weakness. Electron microscopy revealed no mitochondrial changes. She had received zidovudine (180 mg/m^2 every 6 hours) for two years. Therapy was discontinued because of fever and neutropenia. The CK level fell from a peak of 25,945 IU/L to 185 IU/L at the time of discharge. (Walter E B et al. Myopathy in human immunodeficiency virus-infected children receiving long-term zidovudine therapy. J Pediatr July 1991; 119: 152-155).

COMMENT. Myopathy associated with HIV infection is usually an inflammatory polymyositis with proximal muscle weakness and an elevated CK level. It may also be non-inflammatory or linked to treatment with antiretroviral agents such as zidovudine. Myopathy associated with zidovudine is characterized by reversible changes in muscle mitochondria, whereas mitochondrial features are normal in specimens from HIV infected patients not receiving zidovudine.

Low-dose zidovudine in 65 children with HIV-1 infection acquired in the perinatal period was well tolerated in a study reported from the Pediatric Neurology Division, Bicetre Hospital, Institut National de la Sante et de la Recherce Medicale, France (Blanche S et al. Pediatrics Aug 1991; 88:364-370). The dosage (400 mg/m^2 per day)