

Neurodevelopmental Consequences of HIV/AIDS Infection

The latest estimates regarding the rate of pediatric HIV/AIDS infection indicate that approximately 6,000 to 7,000 infants are born to HIV-infected women annually. Prior to 1994, approximately 25% of these infants became infected with HIV. More recently, transmission rates of 3% to 6% are now reported with the administration of zidovudine (ZDV), and 2% or less when ZDV is combined with elective caesarean delivery or when women are treated with highly active antiretroviral regimens that reduce maternal viral load to unquantifiable levels.

In contrast to HIV infection in adults, infection in perinatally infected children occurs in the context of an immature and developing immune system and, as a consequence, there are differences in the manifestations of HIV disease between children and adults. Central nervous system (CNS) disease is a significant complication of infection with HIV, particularly if the virus was acquired during the early stages of fetal or neonatal brain development. The degree of HIV-associated CNS involvement for infants and children is variable, and the severity and prevalence of HIV-related CNS manifestations vary by the patient's stage of systemic disease, the age at which they first became symptomatic, the rate of deterioration, the severity of deficits, the number of neurobehavioral domains affected, and the

patient's current age. Significantly fewer children older than 6 years of age develop CNS manifestations as compared with children under the age of 3.

Three profiles of neuropsychological dysfunction are currently recognized in infants, children and adolescents with HIV: those with encephalopathy, those with CNS compromise, and those with apparently normal functioning. In those with HIV-related encephalopathy, most of the CNS structures and functions appear to be equally compromised, resulting in global and severe deficits in cognitive, language, motor and social skills. These HIV-related encephalopathic issues can be either static or progressive. In those with CNS compromise, their development is characterized by overall functioning that is typically within normal limits but with evidence of a significant decline over time in neurobehavioral functioning that remains at least in the low average range or with significant impairments in selective neurodevelopmental functions. Children with HIV-related CNS compromise continue to have adequate functioning in school and activities of daily living, and rarely exhibit other significant evidence of CNS disease. They may have some mild to moderate brain scan abnormalities that do not seem to significantly affect their day-to-day functioning. Finally, some HIV-infected children exhibit apparently normal functioning in which their cognitive functioning is at least within the normal range and without

evidence of significant deficits, decline in functioning or therapy-related improvements, or other neurologic abnormalities that affect functioning and can be attributed to HIV.

Some children infected with HIV may display non-HIV related CNS impairments. Careful review of their medical, developmental and family history might suggest that factors other than HIV disease most likely explain the majority of their neurobehavioral deficits. Of these factors, prenatal exposure to alcohol and/or illegal drugs is a common confounding risk factor that may account for deficits in functioning.

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