Case Study: Delirium in an Adolescent Girl With Human Immunodeficiency Virus–Associated Dementia

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ABSTRACT

Delirium and human immunodeficiency virus (HIV)–associated dementia are well recognized neuropsychiatric consequences of HIV infection in adults. Almost nothing is known regarding the management of delirium in HIV–infected children and adolescents. HIV–related progressive encephalopathy is thought to represent the pediatric form of HIV–associated dementia; however, this condition occurs in HIV–infected infants and preschool children and is rapidly followed by death. This report describes the identification and treatment of apparent HIV–associated dementia complicated by delirium in an adolescent girl. *J. Am. Acad. Child Adolesc. Psychiatry,* 2006;45(1):104–108. **Key Words:** human immunodeficiency virus, dementia, delirium, adolescent.

One late-appearing neuropsychiatric consequence of human immunodeficiency virus (HIV) infection in adults is the development of HIV–associated dementia (HAD; McArthur et al., 1994; Navia et al., 1986). In children with HIV infection, two types of HIV–related encephalopathy have been observed: (1) progressive encephalopathy, characterized by corticospinal tract signs, acquired microcephaly, and loss of previously acquired skills, and (2) static encephalopathy, characterized by cognitive and motor delays but without frank loss of acquired skills or progressive neurological deficits (Belman, 1994). Progressive encephalopathy is the more serious of the two and is usually detected early in life. However, little, if anything, is known about dementia or encephalopathy that occurs in an adolescent in the context of acquired immunodeficiency syndrome (AIDS). This report describes a case of delirium superimposed on HAD in an adolescent girl.

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HISTORY OF PRESENT ILLNESS

The patient, a 15-year-old girl with a history of vertically acquired HIV infection, living in extreme poverty in the inner city, was admitted to hospital after a 4-day history of fatigue, lethargy, and poor oral intake. On admission, her CD4 was 1 cell/μL and her HIV RNA was >750,000 copies/mL. Medications were abacavir/lamivudine/zidovudine (combination), tenofovir, lopinavir/ritonavir (combination), trimethoprim/sulfamethoxazole (combination), azithromycin, filgrastim, and citalopram. These have been unchanged for some time and were unlikely to be the source of her symptoms. Because of resistance, however, her antiretroviral medications were discontinued.

Her hospital course was punctuated by multiple febrile episodes. Detailed investigations for viral, bacterial, and fungal infections, including studies of CSF, blood, sputum, stool, urine, and chest x-ray, were consistently negative. Transaminases were mildly elevated. The remaining blood chemistries were normal. The

patient had episodes of agitated and aggressive behaviors such as biting, grabbing, striking out, spitting, and sexual aggression. She also had periods of confusion, fear, and paranoia interspersed with periods of relative orientation. On hospital day (HD) 10, the Child and Adolescent Psychiatry Service was consulted to help evaluate and manage the patient's agitated and aggressive behaviors.

MENTAL STATUS EXAMINATION

The patient was in her room in bed and awake, hyperalert, and in four-point restraints. She was exceedingly disoriented. She appeared worried and suspicious. Her speech consisted of short phrases or single words. Her thought content was remarkable for an unfounded concern of being raped. She repeatedly asked people around her to check under the bed. Her thought process was disjointed. She was distractible, but occasionally became preoccupied and appeared to be hallucinating. She complained that the bed was moving.

NEUROLOGICAL EXAMINATION

The patient's motor deficits were reminiscent of Parkinson disease. She intermittently manifested a coarse resting tremor that mostly involved both upper extremities but occasionally involved both lower extremities. She appeared slow and rigid, but when asked, she denied any stiffness. Both forearms tended to be carried in a flexed position. When standing, her posture was slightly forward. Her gait was shuffling and narrow based, with no arm swing. At times, however, the patient could walk briskly. A detailed neuropsychological report done almost exactly 3 years before our examination was chosen as a baseline. Laboratories at that time were remarkable for CD4 = 20 cell/μL and HIV RNA = 269,490 copies/mL. Quoting from the report, ''gait, posture, and physical appearance were all in normal limits. No tremors, spasticity, dystonia, asymmetry, dysmorphia, or posturing were noted.''

NEUROPSYCHOLOGICAL TESTING

Neuropsychological testing done 4 months before our examination revealed a Verbal IQ of 62, a Performance IQ of 60, and a Full Scale IQ of 58. The Vineland Adaptive Behavior assessment (Sparrow et al., 2000) revealed the patient to be in the first percentile for adaptive functioning. Quoting from the report of 3 years earlier: Her ''functional independence was age appropriate to advanced compared to other children her age''; she scored at the 93rd percentile for adaptive functioning. ''Many of the patient's cognitive skills, including intellectual functioning, were in the impaired range. Her adaptive skills, her memory, and her executive skills, however, remained grossly intact.'' Verbal IQ was 58, Performance IQ was 57, and Full Scale IQ was 53.

PSYCHIATRIC HISTORY

Family psychiatric history was most remarkable for intravenous drug use in the patient's mother and alcohol use in the patient's father. The mother died of complications secondary to AIDS. The patient's psychiatric history was remarkable for depression first diagnosed at age 12. Four months before this admission, she was hospitalized on our inpatient child psychiatry unit for 16 days for disruptive behavior. At that time, she was diagnosed with mood disorder, adjustment disorder, and oppositional defiant disorder. Citalopram 10 mg/day was started.

MAGNETIC RESONANCE IMAGING (MRI)

Brain MRI on HD 4 revealed prominent ventricles and sulci consistent with atrophy that was remarkable for her age (Fig. 1) and not new in comparison with multiple prior examinations. There was also a focus of signal abnormality in the right caudate nucleus related to a resolving intraparenchymal hemorrhage that had been followed for several months. These findings were not considered to specifically relate to the current episode. Atrophy is common in individuals with longstanding HIV infection, and this patient's atrophy had not changed. Likewise, the onset of the caudate hemorrhage had not coincided with the psychiatric or neurological changes; the only symptom she exhibited that can be caused by caudate infarct was confusion and disorientation (Kumral et al., 1999), a nonspecific symptom whose onset should have coincided temporally if it were caused by the hemorrhage.

DIAGNOSIS

Based on the patient's history, clinical presentation, brain MRI findings, and dramatic loss of adaptive