HIV Infection Presenting with Dementia

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Abstract
We present a case of dementia in a young healthy individual. On evaluation he was detected to have HIV infection with low CD4 count and a high viral load. He had no opportunistic infections or any other AIDS defining illnesses. He recovered fully within 3 months of antiretroviral therapy.

Introduction
Neurologic manifestations in human immunodeficiency virus (HIV) infected individuals can be varied. These may be due to primary infection, opportunistic infections, neoplasms or drug toxicity. HIV-associated neurocognitive disorders (HAND) are a spectrum of disorders ranging from asymptomatic neurocognitive impairment (ANI) to severe dementia known as HIV-associated dementia (HAD). It is also known as AIDS dementia complex or HIV encephalopathy. HAD is characterized by global deterioration of cognitive function, psychomotor retardation, changes in affect, ataxia, faecal and urinary incontinence. Aphasia, apraxia and agnosia are uncommon. This mainly affects the subcortical areas of brain though all parts of neuraxis can be affected. It is generally a late complication of HIV infection and occurs due to direct effect of the virus on central nervous system (CNS) macrophages or glial cells leading to release of neurotoxins and cytokines like IL-1β, TNF-α and IL-6.¹ HAD is the initial AIDS-defining illness in only 3% of patients with HIV infection and is rarely seen preceding the clinical evidence of immunodeficiency. Prevalence of HAD ranges from 10-24% in Western countries² whereas in India it is reported to be only 1-2% patients with HIV infection.³,⁴ The risk and severity of HIV-associated dementia increases with deterioration in immunologic function.

We report a case of severe HAD in an otherwise healthy young individual who improved with antiretroviral therapy (ART)

Case Report
24 yr old, unmarried male, was brought to hospital by his family members with history of abnormal behavior in the form of remaining aloof, talking less and passing urine and stools in clothes for last 15 days. There was no history of fever, headache, seizures, weight loss, loss of appetite, chronic diarrhea or cough. There was no history to suggest substance or alcohol abuse, high risk sexual behaviour or head injury.

On examination, patient was afebrile with no pallor, icterus, or lymphadenopathy. His BMI was 21.30 kg/m². He had staring look, affect was dull and depressed, clothes were smelling of urine, speech was low volume, monotonous, slow, but comprehensible, patient was oriented but had impaired recent memory. Mini mental state examination (MMSE) score was 19/30. He had no hallucinations, delusions or psychotic features. There were no signs of meningeval irritation or any focal neurological deficit. Other systems were normal. Hemogram, biochemical parameters and chest X-ray were normal. USG abdomen showed mild hepatosteatosis. Antibodies to HIV-I and II were detected by ELISA. CD4 count was 119/µL with a high HIV viral load of 2,828,638 IU/ml. CECT head revealed calcified granuloma in left paracentral gyrus. MRI brain (Figure 1) revealed multiple patchy areas of altered signal intensity (hyperintense on FLAIR / T2WI and isointense on T1WI) in the white matter involving the centrum semiovale, corona radiata and parieto-occipital regions that were symmetrical in distribution, an arachnoid cyst in posterior fossa along with calcified granuloma in left paracentral gyrus. CSF was acellular with raised proteins (158 mg/dl) and raised globulins. CSF staining with Gram, Ziehl-Neelsen and India ink and cryptococcal antigen were negative. CSF ADA was 6 U/L. Serum TORCH titres were normal. HBsAg, HCV and VDRL were negative. Patient was diagnosed as a case of HIV related dementia and started on combination anti-retroviral therapy (Zidovudine, Lamivudine and Nevirapine) as well as cotrimoxazole prophylaxis. Patient showed remarkable improvement within a couple of weeks, urine and stool incontinence improved and could carry out his activities of daily living independently. At 3 months follow-up, the patient was asymptomatic. CD4 count was 482/µL. MMSE score improved to 30/30.

Discussion
HIV enters the central nervous system (CNS) early in the course of the infection and almost all patients have neurologic involvement as evidenced by CSF abnormalities in 90%. HIV encephalopathy usually occurs in advanced disease when the patient’s CD4 count is below 200 cells/µL and is generally accompanied by a high viral load but it can also occur in patients with CD4 count >350 cells/µL. Currently there are two theories, direct model and indirect model that account for neurodegeneration in HIV infection.³ Direct model proposes that neuronal death occurs following release of viral proteins from infected cells and indirect model proposes that neuronal death is mediated by inflammatory response mounted by infected cells against the virus and the viral proteins. Neurotoxic processes and impairment of neurogenesis both contribute to the development of HIV-

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associated neurocognitive disorders. There is no single test that can confirm the diagnosis of HAD. Diagnosis is made largely by exclusion, ruling out other possible causes for the impairment like progressive multifocal leukoencephalopathy (PML) and cytomegalovirus (CMV) encephalitis. PML is characterized by focal neurologic deficits though imaging shows multifocal involvement. CMV encephalitis occurs with CD4 count less than 50/µL and typically presents as a confusional state evolving over weeks. Novel therapeutic approaches are under trial targeting key pathways causing HIV induced neuronal injury. ART along with adjunctive neuroprotective agents (minocycline, memantine and antioxidants like selegiline) may become future treatment of HAD. There has been a decline in the incidence and severity of HAD with the advent of ART but minor and asymptomatic neurocognitive impairment are reported more due to improved survival among HIV patients.

The aim of presenting this case was to highlight a rare presentation of HIV infection with only dementia without any evidence of AIDS defining illnesses or opportunistic infection and to show the tremendous response to ART.

References


