Human immunodeficiency virus encephalitis

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Abstract

Here, we present a case of pyrexia with altered sensorium in a young healthy male individual. On evaluation, he was detected to have human immunodeficiency virus infection with low CD4. He had no opportunistic infection or any other acquired immunodeficiency syndrome-defining illnesses although his magnetic resonance imaging brain showed features of encephalitis. He recovered tremendously within 3 months of antiretroviral therapy.

Key words: Human immunodeficiency virus, human immunodeficiency virus encephalitis, low CD4, and encephalitis

INTRODUCTION

Human immunodeficiency virus (HIV) has been shown to have primary neurotropic properties.[1] Neurological lesions with HIV can occur in patients with acquired immunodeficiency syndrome (AIDS) or in asymptomatic individuals.[1]

The widespread use of efficacious antiretroviral therapy has tremendously influenced the natural history of the HIV infection. The frequency of the central nervous system (CNS) opportunistic infections, which were accompanied with high mortality, has decreased dramatically in countries where antiretroviral agents are available.[2] Two clinical neuropsychiatric disorders remain prevalent following the introduction of antiretrovirals, the first is a major depressive disorder,[3] and the second is HIV-associated neurocognitive disorder (HAND).[4] Bell *et al.* noted that HIV encephalitis (HIVE) occurred independently of opportunistic infections and lymphomas and showed different associations with risk groups, immunosuppression, and antiviral treatment.[5] Compared to the pretreatment era, in the post highly active antiretroviral therapy (HAART) era, there has been shift in the HIVE from a subacute to a chronic protracted form probably due to the increased longevity of HIV-infected patients and more exposure to HIV proteins and emergence of resistant HIV strains.[6]

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CASE REPORT

Our patient, a 35-year-old right-handed male, a soldier by occupation and a resident of Greater Noida, Uttar Pradesh, India, presented with the complaints of fever for the past 2 weeks and altered sensorium for the past 4 days following an episode of generalized tonic–clonic seizure. There was no associated upper or lower respiratory tract symptoms, no burning micturation, ear discharge, rashes, or other skin lesions or head trauma. His investigations showed that he was HIV-1 reactive. He had a history of prolonged fever for 1 month a year back and had been diagnosed with tubercular lymphadenitis by the fine-needle aspiration study. He was prescribed rifampicin, isoniazid, pyrazinamide, and ethambutol for the same, and he was continuing to take all of them till date. There was no history of addictions. He was married for 7 years and had two children. Examination revealed normal vitals in a conscious but disoriented patient with a staring look and a Glasgow Coma Scale score of 11/15. General examination revealed pallor and oral thrush. Neurological examination revealed a Folstein's Mini-Mental State Examination (MMSE) score of 12/30 with no signs of meningismus. Due to the condition of the patient, whatever restricted examination of motor system, sensory system, and cranial nerves was possible was normal. Examination of

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cardiovascular, respiratory, abdominal, and musculoskeletal system was within normal limits.

Investigations revealed an erythrocyte sedimentation rate of 33 mm (1st h) and a mild normocytic normochromic anemia (hemoglobin – 10.4 g%). Other biochemical parameters (liver function, kidney function, and blood sugar) were normal. Urine examination was also normal. A repeat testing of HIV by the ELISA confirmed the presence of antibodies against HIV-1. The CD4 count was 156/mm^3, with a CD4 percentage of 14%. The differential diagnosis includes HIV with the likelihood of CNS opportunistic infections or HIV encephalopathy. Cerebrospinal fluid (CSF) examination reveals 10 cells, all mononuclear, with a sugar of 56 mg%, and a protein of 46 mg%. The adenosine deaminase levels were found to be 10 IU/L (within normal limits), and the CSF did not show any organism on Gram stain or Ziehl–Neelsen stain. The India ink staining for *Cryptococcus* was also negative. The polymerase chain reaction (PCR) in CSF was negative for *Mycobacterium tuberculosis*, human herpesvirus-6/herpes simpex virus, *Toxoplasma gondii*, and *Cytomegalovirus.* The PCR for JC virus could not be carried out due to financial constraints. Serum Widal, venereal disease research laboratory, hepatitis B surface antigen, Hepatitis C RNA, and IgG antibodies against toxoplasma were all negative.

Imaging of the brain revealed bilaterally symmetrical, T2/FLAIR hyperintensities noted in the white matter, involving the bilateral centrum semiovale, periventricular, posterior limb of the internal capsule, and midbrain (cerebral peduncle) suggestive of encephalitis on magnetic resonance imaging [Figure 1].

The patient's attendant did not give consent for a brain biopsy. The final diagnosis was made to be HIVE in a treatment-naïve patients with a CD4 count of 156/mm^3 with oral candidiasis.

He was started on antiretroviral therapy with triple-drug regimens of tenofovir, lamivudine, and efavirenz along with antieplieptics and fluconazole. After 3 months of follow-up, he was seizure free, and his MMSE revealed marked improvement (from 12/30 to 20/30). The CD4 count also showed improvement from 156/cumm to 325/cumm.

DISCUSSION

Infection of the CNS by HIV is associated with neurologic conditions, from HIV-associated dementia (HAD) to milder HANDs.[7] HAART helps to prolong the life of infected patients, reducing the incidence of HAD.[2] Although cases with severe HIV-related cognitive impairment have decreased, the frequency of HIVE, characterized by the presence of infected macrophages in the CNS, microgliosis, astrogliosis, and myelin loss, has remained the same or slightly increased.[5]

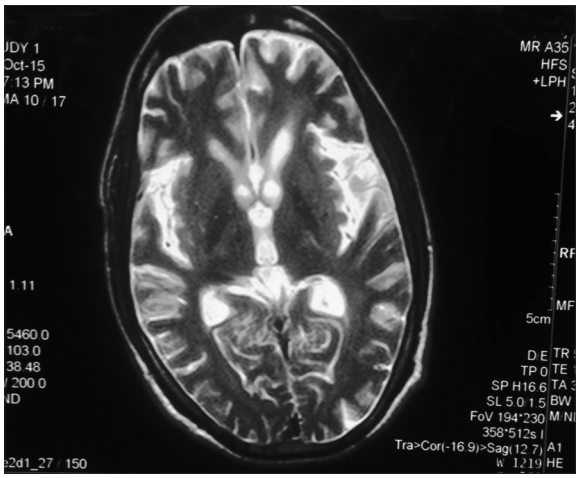

Figure 1: T2-weighted image of magnetic resonance imaging brain showing encephalitis

Brain, in particular, represents a sanctuary for HIV-1 latency, where the provirus can persist due to the variable and poor penetration of antiretroviral drugs.[7]

Neuroinflammatory responses play an important role in neurodegeneration in HIV1 latent and HIVE groups.[8-10]

There is no single test that can confirm the diagnosis of HIVE. Diagnosis is made largely by exclusion, ruling out other possible causes for the impairment such as herpes simplex virus encephalitis, toxoplasmosis, progressive multifocal leukoencephalopathy, *Cytomegalovirus* encephalitis, and CNS lymphoma.

Although the exclusion of other possibilities in our case (by imaging and serology testing) coupled with characteristic imaging properties of HIVE makes the diagnosis of the same almost certain.

The aim of presenting this case was to highlight a rare presentation of HIV infection with only acute impaired mentation without evidence of any severe AIDS-defining illness or opportunistic infection and to demonstrate the tremendous response to the ART.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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