Human Immunodeficiency Virus-Associated Neurocognitive Disorder Masquerading as Psychiatric Illness

Jitender Aneja, Navratan Suthar, Gopal K Bohra, Pawan Garg

Departments of Psychiatry, *Internal Medicine and ‡Interventional Radiology, All India Institute of Medical Sciences, Jodhpur, Rajasthan, India

Abstract

Human immunodeficiency virus-1 (HIV-1)/acquired immunodeficiency syndrome (AIDS) has been shown to be associated with a constellation of neuropsychiatric disorders. The presence of depressive symptoms may obscure the evaluation of cognitive manifestation of a neurodegenerative disorder like dementia associated with AIDS. Further, it is uncommon to see catatonia in association with HIV/AIDS. In this report, we present a case who had an array of psychiatric syndromes as the initial manifestation of AIDS. The subsequent management issues are also dealt with.

Keywords: Catatonia, depression, human immunodeficiency virus dementia, human immunodeficiency virus/acquired immune-deficiency syndrome

INTRODUCTION

Human immunodeficiency virus-1 (HIV-1) has been estimated to infect 34 million persons worldwide. Many persons infected with HIV remain unaware of their serostatus and thus go untreated and are at a high risk of morbidity and mortality, especially involving the central nervous system (CNS).[1] The involvement of CNS may lead to presentation of such patients with a range of neuropsychiatric manifestations such as depression, anxiety, psychosis, suicide, neurocognitive disorders, and rarely, catatonia and mania.[2] We encountered a patient who presented with depression, catatonia, and possibly dementia as the initial presentation of acquired immunodeficiency syndrome (AIDS).

CASE REPORT

A 57-year-old married male with well-adjusted premorbid personality and no past or family history of mental illness was brought to us with complaints of depressed mood and gradual loss of interest in work for 9 months, decline in memory and performance of activities of daily living (ADL) for 6 months, and refusal to eat for 1 month. Stressors in the form of financial problems and inability to bear a child could be elicited. The initial symptoms of illness were sadness of mood, reduced socialization, anhedonia, disturbed sleep, and reduced appetite, but no ideas of worthlessness, hopelessness, sin, or guilt. In addition, there was a significant decline in attention and concentration, impairment of new learning, and recent as well as remote memory. A month prior to admission to our inpatient unit, he had stopped performing his routine ADL and required the assistance of family. At occasions, he remained motionless for hours with wide-open eyes that stared at one point. He lost control of bowel and bladder too. In keeping with possible diagnosis of depression and dementia made by a private psychiatrist, he was treated with escitalopram 10 mg/day, clonazepam 0.5 mg hs, amisulpride 200 mg/day, and piracetam 1200 mg twice a day for 2 weeks prior to admission, but without any relief. His mental state examination revealed stupor, mutism, and rigidity in all the limbs, and negativism, posturing, and grimacing. He was emaciated and had coarse tremors in the upper extremities and whitish lesions in the oral cavity. No focal neurological deficits were observed. Our working diagnosis was severe depression with psychotic symptoms (in view of catatonia, rating on Bush–Francis Catatonia Rating Scale [BFCRS] was 16), with a possibility of dementia. He was thoroughly investigated for possible human immunodeficiency virus-associated neurocognitive disorder masquerading as psychiatric illness.
etiologicals of a dementing illness including an magnetic resonance imaging (MRI) brain [Table 1 and Figure 1]. The patient tested positive on HIV enzyme-linked immunosorbent assay and typing revealed HIV-1 infection with very low CD4 counts (57 cells/cm²), so he was diagnosed with AIDS too. In view of the absence of history of head trauma, seizures, fever, hypertension, diabetes mellitus, substance abuse, and exposure to psychotropic medications prior to the onset of catatonic symptoms and physical investigations, the possible etiologies for catatonia could have been depression, dementia, or HIV infection.

The initial management included maintenance of hydration and vitals with intravenous fluids and nasogastric feeding and a trial of intravenous lorazepam (6–8 mg/day) for catatonia, but with minimal improvement (BFCRS declined from 16 to 13 in 1 week). Antiretroviral therapy in consultation with the district integrated treatment and counseling center was started. In addition, prophylactic co-trimoxazole and fluconazole were given. The dose of escitalopram was hiked to 20 mg/day and amisulpride was substituted with olanzapine 5 mg/day. In view of minimal improvement in catatonia, electroconvulsive therapy (ECT) was started after obtaining informed consent from the family. He was administered five sessions of modified bilateral ECTs. Even though catatonia melted away with ECT (BFCRS score reduced from 13 to 2), the patient did not reach his premorbid level of functioning. Although he started walking and regained bowel and bladder control, he required assistance in performing most of the ADLs. Also, at times, he could not comprehend the instructions but responded by just smiling. Due to this, we could not perform lobar function tests or neuropsychological battery, so planned to do it at follow-up. However, the patient was not brought for follow-up despite repeated reminder calls. Instead, the family sought faith healing and stopped all medications. The patient succumbed to illness after 4 months of discharge due to deterioration of health. No autopsy was performed as per the family’s wish.

Figure 1: T2-weighted axial and coronal fluid-attenuated inversion recovery magnetic resonance sequence showing prominent cortical sulci (dashed arrows), cerebrospinal fluid spaces, and dilated bilateral ventricular system (solid arrow) consistent with cerebral atrophy

Table 1: Physical investigation of the patient

| Name of Investigation                  | Result                                                                 |
|----------------------------------------|------------------------------------------------------------------------|
| Complete hemogram with ESR             | Hb=13.4g/dl, TLC=6230/microL, DLC=N72L17M9E1; ESR=5 mm in the 1st h  |
| Serum electrolytes                      | Na+=139, K+=4.06, Cl-=105 mmol/L                                       |
| Blood sugar                            | 103 mg/dl                                                              |
| Liver function test                    | Total bilirubin=0.85 mg/dl, direct/indirect bilirubin=0.12/0.73 mg/dl, AST/ALT=19/22 U/L, ALP=158 U/L |
| Kidney function test                   | Urea=34 mg/dL, Creatinine=0.62 mg/dL                                   |
| Serum calcium                          | 9.07 mg/dL                                                            |
| Creatinine phosphokinase NAC           | 203 U/L                                                               |
| VDRL (RPR test)                        | Nonreactive                                                           |
| HCV ELISA                              | Nonreactive                                                           |
| HBsAg                                  | Negative                                                              |
| HIV1/2 ELISA                           | Reactive                                                              |
| HIV1/2 Comb AIDS and Qual Pro          | Reactive for HIV-1                                                    |
| CSF examination                         | CSF biochemistry: Sugar=68 mg/dL, proteins=53 mg/dL, chloride=128 mg/dL |
|                                         | CSF for Cryptococcus negative                                         |
|                                         | CSF culture - No growth at 48 h                                       |
|                                         | CSF-ADA (8 U/L); cellularity - normal                                 |
| Chest X-ray PA view                    | No lesion observed                                                    |
| ZN staining of sputum                   | No AFB                                                                |
| Vitamin B12 levels                     | 287 pg/ml                                                             |
| Thyroid panel                          | FT3=3.26 pg/ml, FT4=0.97 ng/dl, TSH=2.31 mIU/l                         |
| MRI brain (image 1)                    | Dilatation of ventricular systems and subarachnoid spaces             |

Hb=Hemoglobin, ESR=Erythrocyte sedimentation rate, TLC=Total leukocyte count, DLC=Differential leukocyte count, AST=Aspartate aminotransferase, ALT=Alanine aminotransferase, ALP=Alkaline phosphatase, HIV-1=Human immunodeficiency virus-1, AIDS=Acquired immune-deficiency syndrome, CSF=Cerebrospinal fluid, TSH=Thyroid-stimulating hormone, AFB=Acid-fast bacilli, MRI=Magnetic resonance imaging, HCV=Hepatitis C virus, ZN=Ziehl–Neelsen, VDRL=Venereal Disease Research Laboratory, ELISA=Enzyme-linked immunosorbent assay, HBsAg=Hepatitis B surface antigen, RPR=Rapid Plasma Reagin, PA=Posteroanterior, ADA=Adenosine Deaminase, FT3=Free tri-iodothyronine, FT4=Free tetraiodothyronin
**Discussion**

Major depressive disorders as well as subsyndromal depressive symptoms are more prevalent in HIV-infected persons, with rates up to 2%–65% across studies.[1,2] The various predisposing factors for depression in persons with HIV include previous history of psychiatric illness, substance abuse, psychosocial stressors, reaction to the diagnosis of HIV and associated stigma, co-morbid medical illnesses, and the direct effect of the virus on CNS. In the index case, there was no past history of psychiatric illness or substance abuse or any medical illness, but stressors were present. However, we could not elicit how and to what extent it could have impacted him due to lack of clinical interview with the patient. Although the index case fulfilled the Diagnostic and Statistical Manual of Mental Disorders-5 diagnostic criteria for major depressive disorder with catatonia, the later part of the illness was dominated by cognitive symptoms along with motor retardation. Furthermore, though catatonia was successfully treated, the patient did not reach his premorbid level of functioning.

Catatonia in persons with AIDS has been scarcely reported.[4‑9] In most of the cases, catatonia was successfully treated with lorazepam except one where ECT was used.[7] The earliest case report[4] of a 19-year-old boy without any previous psychiatric illness and comorbid CNS infection had almost a similar picture as the index case. Similarly, Hisamoto et al.[10] recently reported new-onset catatonia in a 19-year-old boy who had a 10-day history of paranoid and bizarre behavior and tested positive for HIV-1. He responded to treatment with triple antiretroviral therapy, antipsychotics, and lorazepam. At 6 months of follow-up, only some memory issues were unresolved. Furthermore, as suggested by Alisky,[11] catatonia may be a sequel of neuronal injury due to a range of disparate processes (including HIV infection) with a dementing process in the background and has proposed using lorazepam or ECT in such patients. Therefore, in the last part, we contend the possibility of dementia in our patient.

HIV-associated neurocognitive disorder or the previously known HIV-associated dementia (HAD) or HIV encephalopathy has now been classified depending on the severity of cognitive symptoms.[12] The prominence of cognitive symptoms historically, a rapid progression, very low CD4 counts, absence of any other CNS pathology, and lack of return to premorbid functioning level in the present patient favor a diagnosis of dementing illness associated with AIDS. Besides, the radiological findings [Figure 1] did not show any white-matter change, and the atrophy was not to the extent seen in patients with dementia. However, of late, it has been suggested that even patients with HIV/AIDS dementia may present similar to Alzheimer’s dementia, and functional neuroimaging (functional MRI or single-photon emission computed tomography or positron emission tomography scan) to delineate the deficits is recommended.[13] Clinically, the index case fulfilled the Stage 3 criteria of AIDS dementia complex according to earlier definitions as well as the new definition of HAD.[14] However, the lack of follow-up and evidence of cognitive decline on neuropsychological test limited the ascertainment of diagnosis of dementia.

**Conclusion**

Such a presentation of AIDS is uncommon to be seen in a routine clinic. Furthermore, the presence of a range of neuropsychiatric manifestations led to diagnostic dilemmas too. This report is limited by the lack of follow-up after discharge which could have helped in clarification of the diagnostic issues. The utilization of ECT as a safe and effective measure in elderly depression is well described. Moreover, the evidence for its safe and effective use in elderly patients with dementia and superimposed depression is positive, but it is not that strong. Hence, we initially hesitated to administer it and were also unsure of the outcome in case the symptoms were due to HAD. This case report highlights the major neuropsychiatric manifestations of HIV/AIDS and suggests that among other possible causes, one should also look for HIV infection in elderly patients presenting with new-onset catatonia.

**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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