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# "Multiple Sclerosis like Demyelination in Early HIV Infection-A Rare Presentation": Case Report and Literature Review

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### Abstract

A 35 year-old male presented with left sided weakness for past 3 months, along with progressive deterioration in higher mental faculties, vision and audition. Clinical and investigational information suggested the diagnosis of an acute demyelination involving various areas of the brain. He was seropisitive for HIV and had good cellular immunity at the time of our assessment. We present this case to highlight the association of Multiple Sclerosis (MS) with HIV, either during seroconversion or early during course of this infection.

Keywords: Multiple sclerosis; Demyelination; HIV infection

## Introduction

Infectious organisms, most likely viruses, have long been a suspect for triggering the autoimmune response in people genetically susceptible to MS. Although many infectious microorganisms have been investigated, no particular organism has emerged as a proven trigger The AIDS virus is a neurotropic virus and CNS involvement as the presenting complaint is seen in approximately ten percent of cases of HIV infection. The mechanisms of demyelination in HIV infection could be due to lesions directly related to infections of the nervous system by HIV itself, opportunistic infections and lymphomas, secondary to cell mediated immunodeficiency, and other general and systemic complications of HIV.

## **Case Report**

This 35 years old divorced alcoholic male was admitted to Medicine ward in a confusional state with a history of weakness of left side of body for the last three months. Weakness was acute in onset following a fall in the toilet which progressed gradually over next three months and was associated with gradual onset agitation, disorientation, difficulty in vision and hearing, pain in left lower limb with occasional painful spasms and loss of bowel and bladder control for the same duration. Moreover, there was a history of mild headache. There was no history of fever, abnormal body movements or vomiting. He was previously treated for sputum positive pulmonary tuberculosis two years back and was detected to have cataract of the right eye for last six years. General examination was unremarkable except for mild pallor. Vitals were within normal limits. Neurological examination revealed that the patient was conscious but disoriented. Ophthalmological examination revealed mature cataract in right eye, dilated sluggishly reacting pupil and optic atrophy in left eye. Other cranial nerves were within normal limits. Power, sensations, cerebellar functions and gait could not be tested because the patient was not obeying commands. All superficial and deep reflexes of right side of body were normal. On the left side, planter reflex was extensor, ankle jerk was absent while other deep reflexes were normal. A provisional diagnosis of chronic subdural hematoma was made. Baseline investigations including hemogram, sugar, urea, creatinine, electrolytes and LFT were normal except for mild increase of Aspartate transaminase (AST) at 72 IU/L (normal <45 IU/L). The CSF was colorless without any coagulum. CSF cell count was 10 cells/cumm with 95% lymphocytes and 5% neutrophils .CSF sugar was 57 mg/dl (normal 40-80 mg/dl) while CSF protein was 144 mg/dl (normal 20-40 mg/dl). CSF for fungal stain, cryptococcal antigen, VDRL, JC-virus PCR and Toxoplasma antigen were all negative. Oligoclonal band was however detected. HIV ELISA was positive, which was confirmed by Western Blot method. CD4 count was 444cells/L with CD4% of 27%. There was no significant difference between serum / csf HIV viral load. A thorough search for other opportunistic infections was unrewarding. EBV-PCR was done and came out to be negative. CT scan of brain was unremarkable except for mild diffuse brain atrophy. MRI of brain performed subsequently showed demyelinating lesions in periventricular region, posterior part of corpus callosum, caloseptal area and superior vermis. There were mild signal changes in both optic nerves and diffuse cerebral and cerebellar atrophic changes (Figures 1 and 2).

A stereotactic biopsy of the lesion showed multiple, welldemarcated, demyelinating foci, features suggestive of multiple sclerosis, though the patient did not receive HAART at that time. A diagnosis of Multiple Sclerosis (MS) was made in the light of above findings and tests as clinico-pathological findings did not support IRIS due to JC virus infection. Our colleagues from Neurology and Ophthalmology favoured initiation of steroids and we administered intravenous Methylprednisolone 1gm for three days followed by oral steroids for another three weeks. The patient was put on regular physiotherapy and supportive care. He gradually regained his bowel and bladder function. Weakness, orientation and hearing improved over next one month. Reexamination a month later revealed a mild confused state, left sided extensor plantar response, sluggish left ankle jerk and a residual hemiplegic gait. The patient was discharged after two months and has been on follow up for another six months at our HIV Clinic, without any clinical evidence of neurological relapse.

### Discussion

Demyelination or leukoencephalopathy with predominant

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Figure 1: T1-weighted MRI Scan of Brain showing periventricular demyelination.

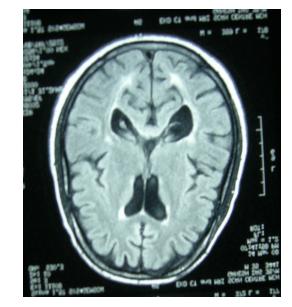


Figure 2: T2-weighted MRI Scan of Brain(Sagital section) showing demyelination in the posterior part of the corpus callosum.

involvement of myelin may occur in many neurological complications related to HIV. Acute multiple sclerosis-like leukoencephalopathy revealing HIV infection may also occur in rare cases. Only a few cases of HIV positive patients with MS or MS like lesions have been reported in literature. Berger et al first described 7 HIV patients with neurological diseases resembling multiple sclerosis. In four of them, the MS preceded the HIV infection by a long period. In the remaining three, HIV infection was documented concomitantly or within three months of the onset of the neurological symptoms [1]. Grey and coworkers described two cases of fulminant multiple sclerosis like leukoencephalopathy associated with HIV infection.

Both patients died two months after onset of illness and autopsy showed characteristic plaques of multiple sclerois in the brain [2]. In 1992 Berger JR et al again described a case of relapsing and remitting leukoencephalopathy which was steroid responsive. This patient had recently seroconverted for HIV and presented with weakness of limbs, bilateral optic neuritis, leg pain and spasm in early course of HIV infection [3]. The neurological presentations described above overlap with those observed in Progressive Multifocal Leukoencephalopathy (PML). However, PML is a classical association of advanced HIV illness and all the cases described above have ben in HIV patients with relatively intact imunocompetence. Our patient was diagnosed seropositive for HIV after admission and CD 4 count was 444cells/L. Greber el al reported a case of a 28 year-old HIV positive lady, who had classical temporal course of MS and had detectable HIV-1 RNA in her CSF, on the background of relatively preserved immunity with a CD4 count of 554cells/L [4]. Deshpande and Patnaik from Mumbai, India reported four HIV cases associated with CNS demyelination. Three of them presented with hemiplegia while another presented with cerebellar involvement. The mean CD4 count was 303cells/ L. One of them responded well with HAART. Of the two who were initiated on steroids, one responded while the other succumbed to the illness [5]. Although the precise timing of HIV seroconversion is often difficult to establish, the fact that all the cases of MS in HIV described in the literature had reasonably high CD4 counts raises the question of the primary manifestation of MS coinciding with HIV seroconversion. Conversely, a newly formed, still asymptomatic plaque could become symptomatic after the up-regulation of cytokines early in the course of HIV infection. In analogy to acute disseminated encephalomyelitis, which is observed after viral infections and vaccinations, HIV may be incriminated to trigger the demyelinating process.

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