

Case Report

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

Partha Sardar<sup>1</sup>, Pradipta Guha<sup>2\*</sup>, Deeptarka Roy<sup>3</sup>, Dipanjan Bandyopadhyay<sup>3</sup> and Sanjoy K Chatterjee<sup>3</sup>

<sup>1</sup>Lal Bahadur Shastri Hospital, Khichripur, New Delhi-110091, India

<sup>2</sup>Calcutta National Medical College, Kolkata, 24, Gorachand Road, Kolkata – 700014, West Bengal, India

<sup>3</sup>Medical College, Kolkata, 88, College Street, Kolkata-700073, West Bengal, India

## 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 seropositive 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, well-demarcated, 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

**\*Corresponding author:** Pradipta Guha, RMO Cum Clinical Tutor, Department of General Medicine, Calcutta National Medical College, Kolkata Address:335, Nandan Nagar, Belgharia, Kolkata 700083, West Bengal, INDIA, Tel: 091-33-25412415; Fax No: 091-33-22198727; E-mail: [dr.pradipta@yahoo.co.in](mailto:dr.pradipta@yahoo.co.in)

Received January 03, 2011; Accepted May 10, 2011; Published May 15, 2011

**Citation:** Sardar P, Guha P, Roy D, Bandyopadhyay D, Chatterjee SK (2011) “Multiple Sclerosis like Demyelination in Early HIV Infection-A Rare Presentation”: Case Report and Literature Review. J AIDS Clinic Res 2:124. doi:10.4172/2155-6113.1000124

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