HIV Presenting as Cerebellar Ataxia

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Abstract
Presenting features of HIV has always been a Pandora’s box largely due to multisystem affection by the virus and a large array of opportunistic infections. We hereby report an extremely rare case of a 40 yr old patient presenting as symmetric, progressive cerebellar ataxia later found to be HIV positive.

Thorough knowledge of rare presentations of HIV and a high index of suspicion are necessary for early diagnosis and efficient treatment of HIV.

Introduction
This virus affects almost every organ system of the body and presents with opportunistic infections which are uncommon in healthy individuals. Since HIV alters human defence system, even common infections have atypical presentations covering almost the whole spectrum of symptomatology known to mankind.

Patients with HIV infection may develop hyperkinetic or hypokinetic movement disorders such as hemichorea-ballismus, myoclonus, dystonia, tremor, and ataxia. Movement disorders may be the presenting manifestation of HIV infection itself may result from an opportunistic infections in the brain. Cerebellar involvement in HIV presenting as ataxia is extremely rare and only two such cases have been reported. It presents as insidious onset gait impairment, visual blurring and hand incoordination.

Ataxia describes a lack of muscle coordination during voluntary movement. Common causes are alcohol, drugs and toxins; stroke; multiple sclerosis; inherited degenerative disorders; viral infections. Among these causes, HIV is an extremely rare cause of ataxia.

These changes are proposed due to AIDS-related multifocal leukoencephalopathy or opportunistic infections. We hereby report a rare case of a 40 yr old HIV positive patient presenting as cerebellar ataxia.

Case Report
A 40 year old female presented with 8 months history of gradual onset, symmetric, progressive difficulty in walking, impairment of vision and inability to perform fine activities due to increased tremors for which she was admitted to our ward. On further enquiry, she gave history of intermittent low grade fever, diarrhoea and weight loss since 1 year. History of increased forgetfulness and occasional outburst of temper was given by the relatives. There was no history of seizures, sensory symptoms or bowel bladder dysfunction. Patient was non-alcoholic; chronic tobacco abuser. No prior history of blood transfusions or surgical interventions.

On general examination she was malnourished, having pallor and oral candidiasis. Her vitals were stable. Although she was conscious, detailed mental function evaluation was not possible due to poor attention span. Cranial nerve examination was normal except nystagmus with fast beating towards right. Pupils and fundus were normal. On motor system examination, tone was reduced in all four limbs, wasting present, power 4+ in all limbs. Superficial and deep reflexes were intact with pendular knee jerk. Sensory and autonomic signs and signs of meningeal irritation were absent. Cerebellar signs were present in both upper and lower limb. Gait could not be assessed as patient was unable to stand with backward swaying.

In laboratory evaluation, hemogram showed pancytopenic picture (Hb- 5.8gm, TLC-1,300, plt-77,000). Her serum electrolytes, RFT, LFT, chest X-ray, USG abdomen were normal. HIV Elisa was positive, CD4 count was 41/μl. HBsAg was positive, VDRL negative. Serum B12 and TSH levels were normal. CSF examination showed 120 cells/cumm with 80% lymphocytes and 20% neutrophils; protein 34 mg/dl; sugar 46 mg/dl and ADA was 8. MRI brain revealed gliosis in bilateral middle cerebellar peduncles, antero-superior aspect of bilateral cerebellar hemisphere and in vermis with dilation of 4th ventricle (Figures 1 and 2); suggestive of spinocerebellar ataxia.

Discussion
Clinically relevant movement disorders are identified in 3% of patients with HIV infection in hospital-based studies.¹ Various movement disorders have been described in HIV patients

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like dystonia, chorea, hemiballismus, tics, paroxysmal dyskinesias and myoclonic ataxia.² A rare case of myoclonic ataxia as a presenting feature of HIV has been reported.³ Arvind et al reported a study of 19 patients of HIV presenting as stroke and proposed inclusion of HIV screening in patients of stroke with unknown cause.⁴

The pathogenesis of movement disorder in HIV infection remains unclear. Opportunistic infections involving the subcortical structures are the common cause of movement disorders. HIV infection-related pathology involving basal ganglia, brainstem and cerebellum may also result in movement disorders.⁵ Few hypothesis explaining these disorders have been proposed; a) development of calcific vasculopathy, b) evidence of central synthesis of HIV specific IgG and c) ischaemic infaction due to HIV-induced hypercoaguable state.

In HIV-1 infection, activated brain macrophages and microglia release quinolinic acid, a neurotoxin and N-methyl-D aspartate (NMDA) receptor agonist. Elevated cerebrospinal fluid quinolinic acid levels are associated with region specific cerebral volume loss in HIV infection and has been implicated in the development of cognitive deficits. Fluorodeoxyglucose positron emission tomography (FDG-PET) studies have shown relative hypermetabolism in the basal ganglia and the thalmi in patients with AIDS dementia complex with motor disorders. FDG-PET studies have also shown global cerebral hypometabolism in advanced HIV infection.

Ataxia in HIV infection can be caused by HIV encephalopathy, vasculitis and opportunistic infections like toxoplasmosis and JC virus.⁶

**References**