

HIV Associated Isolated Cerebellar Ataxia without Any Structural Abnormality in MRI

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Abstract

Objective: To report a rare presentation of HIV associated subacute onset cerebellar ataxia in the absence of any structural abnormality in MRI.

Background: Cerebellar ataxia in a patient with HIV is rare and requires a diagnostic evaluation. A 35 year old male presented to us with subacute onset cerebellar ataxia since 6 weeks. Neurological examination showed scanning speech, dysmetria, dysidiadochokinesia, and gait ataxia. Cognition was preserved. No sensory or motor deficit was observed. Brain MRI was normal. Liver and renal profiles were within normal limits. Chest X-ray and abdominal ultrasonography was normal. HIV test was positive and CD4 count was 175. CSF examination revealed elevated protein (53.5 mg/dL), normal sugar (69), lymphocytic pleocytosis (50 cells with 94% lymphocytes). CSF for AFB, bacterial culture, GenXpert, fungal culture, india ink was negative.

Conclusions: HIV testing should be considered in any young male presenting with acute or subacute onset cerebellar ataxia. In the absence of any abnormality in MRI and negative tests for opportunistic infections, a possibility of directly HIV associated cerebellar ataxia can be considered.

Keywords: HIV; Cerebellar ataxia; AIDS; Normal MRI; HIV associated ataxia without structural abnormality in MRI

Case Study

In February 2018, A 35 year old male, without any comorbidities, presented to us with six weeks history of ataxic dysarthria, followed by wide based ataxic gait, tremulousness of hands on writing and reaching out to objects within two weeks of onset of illness. The disease was sub acute in onset and progressive in course.

There was no history of fever, headache, cognitive impairment, visual disturbances, seizures, weakness of any limbs, sensory complaints, bladder bowel involvement.

Patient had no history of previous illness or drug abuse and his family history was unremarkable. Patient was occasional alcoholic and had been drinking 20-30 mL of whisky once in 2 to 3 months for last 15 years.

General physical examination was normal, higher mental functions and cranial nerves were intact. Speech was slow and scanning type.

Neurological examination revealed features of cerebellar ataxia. Dysmetria and dysidiadochokinesia was evident. His gait was broad based and ataxic. Finger nose finger test, Heel shin test was positive and patient couldn't perform tandem walking.

Cognition was preserved. There were no meningeal signs. Cranial Nerve examination was normal. Motor examination was normal. Joint position sense and vibration sense was preserved. Plantars were bilateral flexor. Deep tendon reflexes were normal. Fundus examination was normal.

Investigations

Patient's baseline investigations including complete blood count, liver function test, renal function test, lipid profile, blood glucose were unremarkable.

Patient was tested by ELISA for HIV that came out to be positive

for type 1 virus and was subsequently confirmed by western blot assay. Baseline CD4 count at the time of diagnosis was 175.

Venereal Disease Research Laboratory test was nonreactive. HBsAg and Anti HCV were negative. Chest skiagram was normal. Ultrasonography of abdomen was normal.

Thyroid function test was normal. Serum vitamin B12 levels were in the normal range. ANA was negative.

Brain MRI was normal. There was no evidence of cerebellar degeneration or focal lesion. Chest X-ray and abdominal ultrasound was normal.

Examination of CSF revealed protein concentration of 53.5 mg/dL, glucose concentration of 69 mg/dL, and WBC count of 50 cells/mm³ (94% lymphocytes). Bacterial culture of CSF samples showed no growth, and the results of Gram staining of CSF were negative. PCR analysis of CSF for herpesviruses (HSV 1 and HSV 2) were negative. CSF India ink and cryptococcal antigen was negative. CSF AFB and GenXpert was negative. Serum toxoplasma IgM and varicella IgM was negative.

Discussion

Subacute onset Ataxia in a middle aged male can be due to infective, neoplastic, paraneoplastic, toxic and metabolic causes.

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Vascular causes and compressive lesions were excluded with imaging techniques. Other toxic and metabolic causes were excluded with relevant investigations.

Our patient was found to be HIV positive and his MRI brain came out to be normal.

AIDS virus is known to be a neurotropic virus and CNS involvement is seen as presenting complaint in 10% of cases, with a spectrum of neurological manifestations.

As such, HIV is a rare cause for cerebellar ataxia. Cerebellar disorders associated with HIV infection are usually due to focal lesions resulting from opportunistic infections such as toxoplasmosis and progressive multifocal leukoencephalopathy or primary CNS lymphoma. Cerebellar involvement can also be seen with AIDS dementia or HIV-associated neurocognitive disorder. However cases have been reported where cerebellar dysfunction is seen in HIV in the absence of dementia [1].

Cases have been described previously where HIV was found to be a cause for the cerebellar ataxia either due to JC virus infection or in the form of unexplained cerebellar degeneration [2].

However, isolated cerebellar ataxia in a patient with HIV in the absence of any abnormality in MRI is very rare.

Another possibility could be post infectious acute cerebellitis. A few patients with cerebellitis have presented with a normal MRI. However, the absence of fever and prodromal symptoms makes the diagnosis of cerebellitis unlikely.

The exact pathogenesis of ataxia in this patient was not clear. A syndrome of unexplained cerebellar degeneration associated with HIV infection has been described previously [3] where MRI suggested cerebellar atrophy. However, MRI brain revealed no abnormality in our patient. Opportunistic infections were ruled out on CSF examination and MRI.

Since patient was occasionally alcoholic, possibility of alcoholic cerebellar degeneration can be considered but neuro imaging in alcoholic cerebellar ataxia demonstrates cerebellar atrophy and the presence of lymphocytic pleocytosis on CSF examination cannot

be explained with alcoholic cerebellar disease. Also patient was an occasional alcoholic.

Another possible etiology can be Paraneoplastic, for which patient was evaluated for any primary. However, on examination there was no palpable lymph node, testicular mass or thyroid nodule. Chest X-ray and Ultrasound of abdomen were also normal. Stool of occult blood was negative and PSA was within normal limits. Due to unavailability of auto-immune profile including anti-Yo, anti-Hu etc. in our set up, these investigations couldn't be sent.

Despite the limitation of not being able to evaluate for paraneoplastic auto-immune profile, this case study is remarkable because it reports cerebellar ataxia as a clinical presentation of HIV which itself is very rare. Another significant finding in the case report is the absence of any cerebellar degeneration or focal lesion in brain. The possible etiopathogenesis of ataxia in this case could either be paraneoplastic, or due to HIV invasion and both possibilities account to rare presentation and need detailed work up and evaluation.

Conclusion

HIV testing should be considered in any young male presenting with cerebellar ataxia. MRI brain should be done to look for any degeneration or focal lesion. CSF studies should be done to rule out any opportunistic infection.

Isolated Cerebellar ataxia in a patient with HIV in the absence of any abnormality in MRI is very rare.

In the absence of any abnormality in MRI, and opportunistic infections ruled out through CSF studies, a possibility of directly HIV related cerebellar ataxia can be considered.

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