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A Case Report on Cerebellar Ataxia – HIV Induced Chronic Cerebellitis

SANIYA SANOBER[1], SYED ABDUL MUBEEN SHAH QUADRI[2], SYED KUBRA MEHDI[3], JUVERIA SULTANA[4], SYEDA MAHEEN ABSAR[5], SAHEBA PARVEEN[6], FATIMA TABASSUM[7], SHAISTA MAHIN[8]

[1,2,3,4,5,6,7,8] Department of Pharmacy Practice MRM College of pharmacy,Chintapally guda, Hyderabad, Telagana.

ABSTRACT

HIV-induced cerebellar complications, which are often manifested as ataxia, typically occur from cerebellar lesions resulting from opportunistic infections, vasculitis, or neoplastic processes. A 59yrs old male patient known to have HIV infection from past 15 years, presented to our hospital with weakness, ataxic gait, lower back pain and slurred speech since last 2-3 days. He had been receiving anti-retroviral therapy for over 15 years, and there was no family history of such illnesses. The results of examination were notable for dysarthria, dysmetria and dysdiadokokinesia. MRI revealed ligamentum flavum hypertrophy at multiple cervical levels, prominence of bilateral cerebral sulci and sylvian fissure and chronic lacunar infarcts. The final diagnosis was CEREBELLAR ATAXIA-HIV INDUCED CHRONIC CEREBELLITIS.

KEYWORDS: Ataxia, Anti retro viral therapy, HIV

ARTICLE DETAILS

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INTRODUCTION

Cerebellar ataxia is a form of ataxia originating in the cerebellum. Chronic neurological effects of HIV infection include polyneuropathy and neurocognitive dysfunction [1]. Ataxia in patients with HIV is rare and should lead to further diagnostic testing.

Prior to the emergence of highly active antiretroviral therapy (HAART), opportunistic infections, progressive multifocal leukoencephalopathy, lymphomas, and varicella zoster virus vasculitis-related strokes were the most frequent clinical causes of ataxia. This spectrum has undergone significant alteration since HAART. (2)

PATHOGENESIS

Microglia present innate immune responses and release inflammatory cytokines (e.g., TNF-, IL-1, IL-6, etc.) in response to invaded pathogens (e.g., virus, bacteria, fungus, etc.) during the acute phase. These responses increase the intrinsic excitability of Purkinje cells in the cerebellar cortex and alter the presynaptic release and postsynaptic responsiveness of the excitatory synapses.

Purkinje cells exhibit a variety of physiological abnormalities during the acute or chronic phases of an infection. Possible dysfunctions include : an impairment of the parallel-fiber's long-term synaptic plasticity and reduced parallel-fiber innervations, excess climbing-fiber innervations, an increase in GABAergic synaptic transmission, loss of Ca2+ homeostasis of Purkinje cells, hypo excitability and aberrant oscillation, mitochondrial dysfunction, degeneration and loss of Purkinje cells, resultant reduction in the inhibitory input to deep cerebellar nuclei (DCN) neurons, and hyper excitability of DCN neurons[3,4].

Excess DCN activity is caused by abnormal Purkinje-cell activity and an impairment of error modification, which then sends the activity to efferent cerebellar pathways which subsequently produce tremor/ataxia.

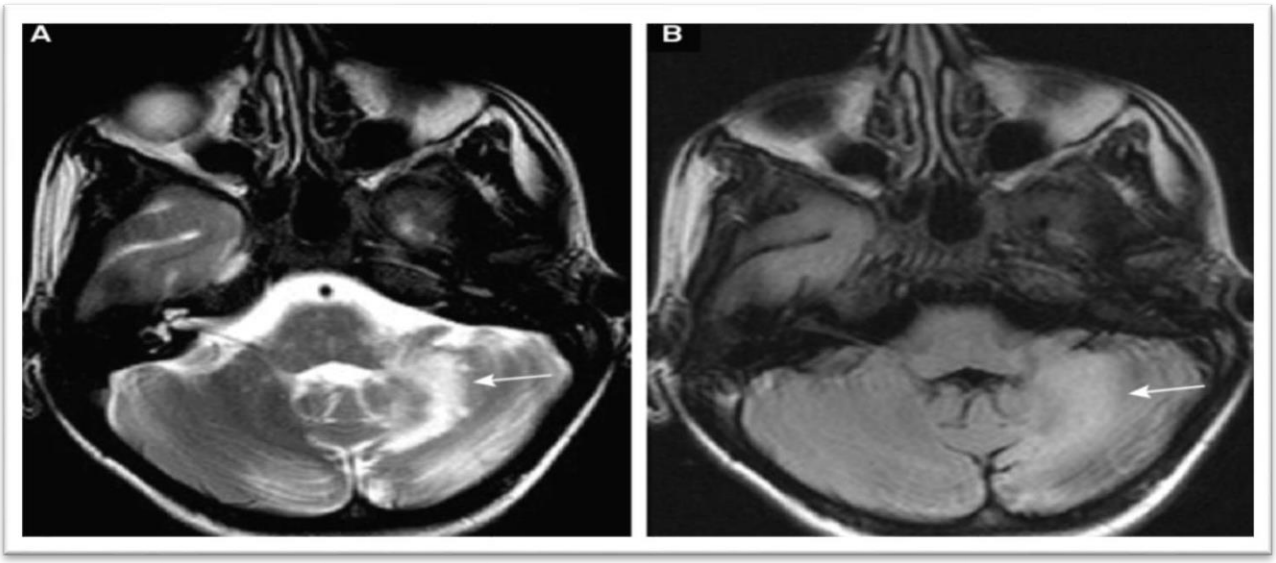

Figure 1(A) Axial T2-weighted and (B) axial FLAIR-weighted brain MRI shows asymmetric hyper intense signal in the cerebellum evolving to the left cerebellar hemisphere and middle cerebellar peduncle.

CASE PRESENTATION

A 59-year-old male patient presented to hospital with chief complaints of weakness, ataxic gait, lower back pain and slurred speech since 2-3days.The patient had a history of HIV in the past 15 years. He had been on regular antiretroviral therapy [HAART] from past 15years. There was no history of fever, visual disturbances. The patient was hospitalized for 5days. At the time of presentation, the patient had elevated blood pressure [150/90mmHg] and motor weakness.

INVESTIGATIONS

Examination	**Observed value**	**Normal limits**
Hemoglobin	13.9	14-18g/dl
RBC count	4.9	3.8-5.8 x 10^{12}/L
T.WBC	6640	4000-11000cells/L
Platelet count	226 x 10^9	150-450×10^9 cells/L
ESR	60	0-20mm/hr.
Sodium	144	135-145 mmol/L
Potassium	3.86	3.5-5.0 mmol/L
Chloride	102	95-107 mmol/L
CRP	2.6	0-1.6mg/dl
S.Cr	1.1	0.-1.2mg/dl
SGPT	28	0-35Units
SGOT	33	0-35Units
ALP	66	30-120Units/L
Bilirubin-T	0.46	0.1-1mg/dl
Bilirubin-D	0.09	0-0.2mg/dl
Albumin	4.2	3.3-4.8g/dl
Globulin	3.8	2.3-3.5g/dl
MRI of spine: At C4-C5 and C5-C6: posterior disc osteophyte complex with posterior disc buldge causing indentation. Ligamentum flavum hypertrophy noted at multiple cervical levels contributing to spinal cord stenosis.		
MRI of Brain: Multiple confluent T2/FLAIR hyper intensity. Chronic lacunar infarcts involving left capsuloganglionic region. Mild prominence of Bilateral cerebral sulci, sylvian fissure.		

The patient was diagnosed with CEREBELLAR ATAXIA-HIV INDUCED CHRONUC CEREBELLITIS as per the MRI reports of spine and brain.

VITALS DURING THE HOSPITAL STAY

On examination the patient was conscious, coherent, oriented. Throughout the stay [Day1-Day5] temperature was consistent 98.6°F; pulse rate and blood pressure were normal and there was slight improvement in walking ability and relief from muscle stiffness.

TREATMENT

During the hospital stay the patient was treated with Inj.Ceftriaxone 1gm (Antibiotic) BD, Inj.Thiamine 1amp OD, Tab. Gabapentin+nortriptyline [neuropathic pain] 100/10mg BD, Inj.pantoprazole [PPI] 40mg OD, Tab.cotrimoxazole [antibiotic] OD , vitamin supplements and Frenkel Exercises are designed to improve coordination in the patient.

DISCHARGE SUMMARY

The patient was discharged based on his improvements in his health.Discharge medications include:
TAB.REMETOR CV-10mgOD
TAB.DALSTEP-10mgBD
TAB PANTOCID-40mgOD
TAB.BACTRIM DS-BD
TAB.TOLIFAST-150mgTID
TAB.REJUNEX CD3-OD
TAB.SPINFREE-P/O-BD
TAB.GABATIN-NT 100/10mg
CAP.AMANTREL-100mg BD

DISCUSSION

The neurological system appears to be affected by HIV at all disease levels and stages. Ataxia is the main manifestation of the cerebellar consequences of HIV infection, which typically originate from lesions in the cerebellum brought on by vasculitis, neoplastic processes, or opportunistic infections. Ataxia is a condition that can be caused due to certain retroviral (such as raltegravir) and the way those medications interact with one another. Common spinocerebellar ataxias can develop in HIV-positive patients even in the absence of a family history.(6).

According to the prevalent hypothesis on the neuropathology of HIV infection, neurological impairment and dysfunction are frequently caused by microglial or macrophage infection, which is followed by the release of cytokines and chemokines.

First treatment choice in this patient is Frenkel exercises. Patients with ataxia can help restore their coordination by performing a sequence of increasingly demanding moves called Frenkel Exercises. To regain the rhythmic, smooth, and coordinated motions, Frenkel exercises are carried out [7-8].

GABAergic medications have been shown to be effective in adult patients with ataxia in several case studies and small case series. Gabapentin increases GABA transmission by activating alpha2delta P/Q calcium channels [9,10]. Clinical outcome includes Improvements in gait and body sway.

CONCLUSION

Cerebellar ataxia is the result of wide range of pathophysiologic mechanisms. HIV infection should be considered as an etiology in clinical setting of sub-acute ataxia, particularly in a young or immunocompromised patient.

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