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AN UNCOMMON CLINICAL SIGN IN A COMMON NEUROLOGICAL DISORDER: A CASE OF ISOLATED TRUNCAL ATAXIA

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ABSTRACT

Neurological diseases often present with varied clinical manifestations, and subtle clinical signs may provide important diagnostic clues. Cerebellar disorders commonly manifest with gait disturbances, limb incoordination, and nystagmus. However, isolated truncal ataxia as a predominant clinical sign is uncommon and may indicate involvement of the cerebellar vermis. Recognition of such atypical presentations is crucial for early diagnosis and appropriate management. We report a case of a 62-year-old male construction worker who presented with sudden onset loss of consciousness for four hours. The patient regained consciousness after initial emergency management but complained of slurring of speech and inability to sit without support. There was no history of seizures, trauma, headache, chest pain, dyspnea, or focal limb weakness. His past medical history was significant for systemic hypertension for three years and a previous ischemic cerebrovascular accident with right hemiparesis three years prior, for which he was not on regular treatment. On examination, the patient was drowsy but oriented to time, place, and person. Vital parameters were stable. Neurological examination revealed horizontal nystagmus and marked truncal ataxia, while limb coordination and gait were relatively preserved. There were no signs of meningeal irritation or autonomic dysfunction. Routine laboratory investigations, viral markers, cerebrospinal fluid analysis, ECG, and chest radiography were within normal limits. Fundus examination was normal. MRI brain was performed to further evaluate the underlying neurological pathology. The patient was managed with supportive care and close neurological monitoring, and further management was guided by neuroimaging findings. This case highlights the clinical importance of recognizing truncal ataxia as a rare but significant neurological sign, which may serve as an early indicator of cerebellar involvement. Careful neurological examination combined with appropriate neuroimaging plays a vital role in identifying the underlying etiology and guiding timely management.

KEYWORDS: Truncal ataxia, Cerebellar vermis, Cerebrovascular disease, Nystagmus, Neurological examination.

INTRODUCTION

Cerebellar disorders constitute an important group of neurological conditions characterized by abnormalities in coordination, balance, and ocular movements. The cerebellum plays a crucial role in maintaining posture, regulating muscle tone, and coordinating voluntary movements. Lesions affecting different parts of the cerebellum produce distinct clinical manifestations. Involvement of the cerebellar hemispheres typically results in limb ataxia and dysmetria, whereas lesions involving the cerebellar vermis predominantly lead to disturbances of axial posture and balance, commonly presenting as truncal ataxia.

Truncal ataxia refers to impaired control of the muscles responsible for maintaining upright posture and sitting balance. It is often associated with conditions affecting the cerebellar midline structures, including ischemic stroke, hemorrhage, tumors, infections, or degenerative disorders. Although cerebellar stroke is a relatively common neurological emergency, the presence of isolated truncal ataxia without significant limb incoordination or gait disturbance is an uncommon clinical presentation and may pose a diagnostic

challenge.

Early recognition of subtle cerebellar signs is essential because delayed diagnosis of posterior circulation stroke or cerebellar pathology may lead to significant morbidity and mortality. Careful neurological examination combined with appropriate neuroimaging plays a vital role in identifying the underlying cause.

Here, we report a case of a 62-year-old male with a history of hypertension and previous ischemic cerebrovascular accident who presented with sudden onset unconsciousness followed by slurring of speech and inability to sit without support, in whom neurological examination revealed horizontal nystagmus and prominent truncal ataxia, highlighting a rare clinical sign in a common neurological disease.

CASE REPORT

A 62-year-old male, a construction worker by occupation, presented to the emergency department with sudden onset loss of consciousness for four hours. He was brought to the triage unit where initial emergency care was provided, following which the patient regained consciousness. There was no history of chest pain, palpitations, dyspnea, seizures, trauma, urinary incontinence, or headache prior to the episode.

Further history obtained from the patient revealed slurring of speech and inability to sit without support. There was no history suggestive of limb weakness, difficulty walking in a straight line, clumsiness while using the hands, spillage of food while eating, jerky movements of the hands while reaching for objects, or difficulty in swallowing. There was also no history of fever, cough with expectoration, neck stiffness, bowel or bladder disturbances, or recent infections.

The patient had a past history of systemic hypertension for three years, for which he was not on regular treatment. He also had a history of ischemic cerebrovascular accident with right hemiparesis three years prior, following which he was not on regular follow-up or medications. There was no history of diabetes mellitus, bronchial asthma, epilepsy, thyroid disorders, or tuberculosis.

Personal history revealed that the patient consumed a mixed diet with normal bowel and bladder habits. He had a history of chronic alcohol consumption for nearly 20 years (approximately 270 ml, three to four times per week) and was a chronic smoker for 20 years (approximately 10 beedis per day). There was no significant family history of neurological or systemic illness.

On general examination, the patient was drowsy but partially obeying oral commands. He was well built and well nourished. There was no pallor, icterus, cyanosis, clubbing, generalized lymphadenopathy, or pedal edema. No neurocutaneous markers or peripheral nerve thickening were noted.

Vital signs were stable with a pulse rate of 89 beats per minute, regular in rhythm, and blood pressure of 150/90 mmHg in both upper limbs. The patient was afebrile with a temperature of 98.2°F, and the respiratory rate was 18 breaths per minute.

Higher mental function examination revealed that the patient was oriented to time, place, and person, with intact memory and preserved comprehension, repetition, reading, and naming abilities. There were no behavioral disturbances, delusions, or hallucinations.

Neurological examination revealed horizontal nystagmus and marked truncal ataxia.

Limb coordination and gait were relatively preserved, and the patient was able to perform tandem walking. Romberg's test was negative. There were no signs of meningeal irritation, cranial nerve deficits, or autonomic dysfunction.

Examination of other systems was unremarkable. Cardiovascular examination revealed normal S1 and S2 heart sounds without murmurs. Respiratory examination showed normal vesicular breath sounds without added sounds. Abdominal examination revealed a soft, non-tender abdomen with no organomegaly.

Laboratory investigations including renal function tests, liver function tests, thyroid profile, and urine analysis were within normal limits. Viral markers (HIV, HBsAg, and HCV) were non-reactive. Cerebrospinal fluid analysis showed no significant abnormalities. Fundus examination was normal.

Electrocardiography demonstrated sinus rhythm with normal PR interval and QRS duration, without significant ST-T changes or chamber enlargement. Chest radiography was within normal limits.

Complete Blood Count	At time of Admission	At time of Discharge
TC	8900	13900
DC[N/L/E]	[67/28]	[71/26]
RBC	5.11	4.95
HB	15.1	14.6
PCV	45.5	46.5
MCV	87	92
PLATELET	2.55L	3.32L

ESR	15	5
CRP	22	7

RENAL FUNCTION TEST

UREA	31.6	36.5
CREATININE	0.9	1.0
SODIUM	141	142
POTASSIUM	4.3	4.2
RBS	95	98

LIVER FUNCTION TEST

TOTAL BILIRUBIN	0.98	0.56
INDIRECT BILIRUBIN	0.8	0.3
DIRECT BILIRUBI	0.19	026
SGOT	51	51
SGPT	21	21
ALP	64	57
PROTIEN	6.08	5.99
ALBUMIN	3.5	3.35
GLOBULIN	2.6	2.6

DISCUSSION

Cerebellar disorders represent an important category of neurological diseases that manifest with disturbances in coordination, balance, and ocular movements. The cerebellum is anatomically divided into the hemispheres and the midline vermis, each responsible for distinct motor functions. Lesions affecting the cerebellar hemispheres commonly produce limb ataxia, dysmetria, and intention tremors, whereas involvement of the cerebellar vermis primarily leads to impairment of axial posture and balance, resulting in truncal ataxia.

Truncal ataxia is characterized by difficulty in maintaining an upright posture and inability to sit or stand without support. It is most commonly associated with lesions affecting the cerebellar vermis or midline cerebellar structures. Several etiologies have been described, including ischemic stroke, cerebellar hemorrhage, infections, tumors, degenerative diseases, and toxic or metabolic causes. Among these, posterior circulation ischemic stroke remains one of the most important causes in elderly patients with vascular risk factors.

In the present case, the patient had significant vascular risk factors including systemic hypertension, chronic alcohol consumption, and smoking, along with a previous history of ischemic cerebrovascular accident. These factors substantially increase the risk of recurrent cerebrovascular events. The patient presented with sudden onset loss of consciousness followed by slurring of speech and inability to sit without support, which raised suspicion for a posterior circulation neurological event.

One of the most notable clinical findings in this patient was the presence of horizontal nystagmus and prominent truncal ataxia in the absence of significant limb incoordination. Such a presentation suggests predominant involvement of the cerebellar vermis, which plays a crucial role in maintaining truncal stability and equilibrium. The preservation of limb coordination and relatively normal gait in the early stages may obscure the diagnosis and delay recognition of cerebellar pathology.

Posterior circulation strokes account for approximately 20–25% of all ischemic strokes and often present with nonspecific symptoms such as dizziness, vertigo, imbalance, or altered consciousness. Compared with anterior circulation strokes, cerebellar strokes are frequently underdiagnosed due to their subtle clinical manifestations. Early recognition is essential because delayed diagnosis may lead to complications such as brainstem compression, obstructive hydrocephalus, and neurological deterioration.

Neuroimaging plays a critical role in confirming the diagnosis. Magnetic Resonance Imaging (MRI) of the brain is considered the most sensitive modality for detecting posterior circulation infarcts and cerebellar lesions. In patients presenting with unexplained truncal ataxia or cerebellar signs, early neuroimaging is essential to identify the underlying etiology and guide appropriate management.

The present case highlights the importance of meticulous neurological examination in identifying subtle cerebellar signs. Recognition of truncal ataxia as a key clinical clue can facilitate early diagnosis of cerebellar involvement, particularly in patients with pre- existing vascular risk factors.

CONCLUSION

This case highlights the clinical significance of truncal ataxia as an important neurological sign that may indicate cerebellar vermis involvement. Although cerebrovascular disease is a common neurological condition, atypical presentations with subtle cerebellar signs can pose diagnostic challenges. In patients with vascular risk factors such as systemic hypertension, chronic alcohol consumption, and smoking, the presence of sudden onset neurological symptoms should prompt careful evaluation for posterior circulation stroke.

A thorough neurological examination combined with early neuroimaging plays a crucial role in identifying cerebellar pathology and preventing diagnostic delay. Recognition of rare clinical signs in common neurological diseases is essential for timely diagnosis and appropriate management.

This case emphasizes the importance of maintaining a high index of suspicion for cerebellar involvement when patients present with isolated truncal ataxia, as early detection and prompt intervention can significantly

improve clinical outcomes.

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