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Case Study *Medical research*

Acute cerebellar ataxia due to drug toxicity

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ABSTRACT

To report the case of a pediatric patient with Acute cerebellar Ataxia A 7 year old boy presented with Sudden onset of weakness in both the Lower limbs with abnormal movement of the eyes and ataxic Gait. He had no history of fever, trauma or any vaccination, No history of loss of consciousness, past history of seizure which was preceded by fever at 3 years of age, On examination the child is consious alert and oriented , no abnormal tone, with equal power in all four limbs and Extensor plantar Response. Examination of cranial nerves, motor system and sensory system did not reveal any abnormality. Patient had difficulty in performing finger-nose, heel-shin test and tandem gait. He had Nystagmus with fast component towards the side of gaze. His gait was wide based **Results:** MRI was normal and Serum Phenytoin level was 40 mcg/ml, which was far above the therapeutic level (10-20mcg/ml

Keywords: Phenytoin , Cerebellar ataxia , Drug toxicity

INTRODUCTION

Ataxia is a disorder that affects the coordination of movement, resulting in poor fine movement and postural control. The cerebellum is responsible for movement coordination and also capable of assessing motions as they are performed, allowing us to fine-tune as well as synchronize our actions. When the cerebellum is dysfunctional, it may disrupt movement strength and coordination, as well as eye movements and feeding as well as play behavior, as well as certain elements of cognitive function (1,2). This structure is made up of the cerebellar hemispheres and also the cerebellar vermis, which is located between the hemispheres. The hemispheres of the cerebellum are in charge of the extremity, whilst the vermis is in charge of the body's central nervous system. A lesion of the hemispheres may result in dysmetria, dysdiadochokinesis, intention tremor, as well as speech abnormalities, among other symptoms. A unilateral cerebellar hemisphere lesion will manifest as symptoms in the ipsilateral limb in the majority of cases. Gait ataxia (requiring patients to walk with a wide space gait), titubation of the head and trunk, dysmetria (typically affecting the lower extremities more than the upper extremity), aberrant eye movements, as well as vertigo are all symptoms of vermis lesions. The fact that coordination of movement also involves motor input to the muscles required for the movement, as well as proprioceptive sensory input returning from the body, must be kept in mind all the time. The synchronization of movement will be hindered if one of these factors is compromised. As a result, before assuming that a lesion in the cerebellum is the source of someone's unsteadiness, it is necessary to rule out any motor or sensory problems.

Drug or toxin use is the most prevalent cause of acute ataxia. Numerous treatments, including phenytoin, phenobarbital, carbamazole, benzodiazepines, antihistamines, as well as other psychoactive prescriptions or street narcotics, are used to treat psychiatric disorders. It is also possible to develop ataxia after exposure to toxic substances such as ethanol and ethylene glycol. Young children (ages 1-4) and teenagers who are experimenting with drugs are the most at risk of being intoxicated, according to the CDC. Patients suffering from ataxia induced by medications or toxins may experience changes in their mental state as well as other side effects, which will vary depending on the substance they are taking. Certain drugs, such as phenytoin, may cause nystagmus as well as ataxia without having any effect on sensorium[1].

Acute cerebellar ataxia (ACA) is an inflammatory central nervous system disorder that manifests itself in previously healthy individuals in less than 72 hours. Symptoms are usually associated with a very mild viral disease, although

they have also been observed after vaccinations. Immunological investigations imply that both conditions are caused by autoimmune destruction of axon tracts, with pathological as well as radiographic data suggesting a relationship with demyelinatng disorders. The emergency response should be focused on ruling out more serious infections, including meningitis or an intracranial mass lesion [2].

Cerebellar ataxia is a frequent condition in neurological practice, and it can be caused by a variety of conditions ranging from chronic as well as slowly-progressing cerebellar degenerations to severe cerebellar lesions caused by infarction, edema, as well as hemorrhage, which can result in a true neurological emergency. Acute ataxia is frequently associated with hospitalization with intensive laboratory testing. Clinicians are often confronted with judgments about the scope as well as the timing of first screening tests, which are especially important in detecting treatable causes [3].

CASE PRESENTATION

A case study of a 7-year-old boy who developed acute onset abnormal jerky movement of the eyes and staggering gait with sideways swaying while walking without preceding history of febrile illnessi n last 2 weeksor trauma .On examination, his higher mental functions were normal. Cranial nerves, motor system, sensory system examination did not reveal any significant abnormalities .All of the above features were the actual findings of this case. Following further assessment of a patient, it was discovered that the patient had trouble executing the finger-nose test, the heel-shin test, as well as the tandem gait test with end gaze Nystagmus which he couldn't control. Past history of single episode of febrile seizure at age of 3 yrs for which he received phenytoin for 3 next years.

DIFFERENTIAL DIAGNOSIS

1. Intracranial space occupying lesion
2. Acute infectious cerebellitis
3. Post-infectious cerebellar ataxia
4. Drug ingestion

INVESTIGATIONS

For the above complain child was investigated ,MRI Brain was performed to rule out any intracranial space occupying lesion, CSF was next performed to rule out intracranial infection which was normal ,autoimmune pane l was planned but parents could not afford, lastly serum phenytoin level was sent keeping a possibility of drug induced ataxia in view of past history of febrile seizures and prolonged phenytoin treatment surprisingly his levels were significantly elevated (40 micrograms per milliliter of blood) than the recommended therapeutic range of 10-20 micrograms per millilitre, on further asking child reveals that he is taking remaining phenytion tablets for the last 10 days from the shelf where her mother kept during cleaning and forgot to place it at a safer place.

Acute cerebellar ataxia	Infectious/immune mediated	Acute postinfectious cerebellar ataxia
		Acute cerebellitis
		Acute disseminated encephalomyelitis
		Multiple sclerosis
	Intoxications	
	Paraneoplastic	Opsoclonus-myoclonus syndrome
	Traumatic	Postconcussion ataxia
		Traumatic vertebral dissection
	Vascular	Ischemic stroke
		Hemorrhagic stroke
	First event intermittent ataxia[a]	Maple syrup urine disease
		Pyruvate dehydrogenase deficiency
		Urea cycle disorders
		Glucose transporter type 1 deficiency
	First episode episodic ataxia	

TREATMENT

Child was managed symptomatically and kept under observation for 48 hours ,ECG was taken to rule out any cardiac arrhythmias due to phenytoin which was normal , symptoms significantly improves after 48 hrs hence discharged and follow up was planned 2 week later for any residual symptoms.

OUTCOME AND FOLLOW-UP

Child did not came for follow up but on having telephonic conversation with his mother she told us that his ataxia has been resolved completely with no more jerky eye movements or residual symptoms ,child is active ,playful without any motor deficits

DISCUSSION

The time-course/progression of symptoms aids in narrowing the differential diagnosis. When a hazardous intake occurs, acute ataxia is presumed unless proved differently. Always inquire about medications that the kid may be taking at home. Concussions & moderate head trauma, along with migraine variations, are all potential causes of ataxia in children. A novel focal neurological deficiency or the occurrence of encephalopathy always seems to be concerning and should require additional assessment. According to our research, this is the first documented instance of phenytoin-induced ataxia. Routine follow-up is required to evaluate patient compliance with prescribed drug dosage regimens and respond accordingly. There is a need to educate patients and their caregivers about the symptoms of phenytoin poisoning so that it can be diagnosed and treated early and effectively.

LEARNING POINTS

1. Urgent neuroimaging to be done in signs of raised intracranial pressure
2. Always ask for drug intake/intoxication
3. Be guarded in a case of 1st episode of ataxia without known cause, it could be Recurrent/Intermittent ataxia

CONCLUSION

To our knowledge, this is a first case report shreds an evidence about the phenytoin-induced ataxia. There is a need for routinely follow up, to assess patient compliance and riposte to prescribed drug dosage regimen. There is need for educating patients and their caregivers about the clinical manifestations of phenytoin toxicity, so that is can be recognized early and treated appropriately.

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