Case Report
Volume 7:1, 2023

Clinical and Medical Case Reports

ISSN: 2684-4915 Open Access

Post-infectious Acute Ataxia in Children: A Case Report

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Abstract

Acute cerebellar ataxia (ACA) is a clinical syndrome presenting with gait abnormality and instability while the mental status remains intact. Although it is the commonest cause of ataxia within pediatric population; patients presenting with a rapid onset gait disturbance must undergo a comprehensive diagnostic approach in order to exclude more serious etiologies that might have a similar presentation. In this article, we report a case of a previously well 2-year-old boy with a recent enterovirus infection, presenting with acute cerebellar ataxia. This case report aims to highlight the appropriate approach to diagnosing post-infectious ACA.

Keywords: Cerebellar syndrome • Pediatric • Cerebellar ataxia • Post-infectious • Acute ataxia

Introduction

Acute ataxia is a non-localizing complaint that includes disorders in which there is a lack of coordinated movement with an onset of fewer than 72 hours [1]. Of all childhood ataxia cases, acute cerebellar ataxia (ACA) accounts for 30-50% [2]; it is characterized by a lack of coordination of movement that is not due to motor loss, sensory loss, or the presence of involuntary abnormal movements. Although it is the most common cause of childhood ataxia; ACA shares certain characteristics with other serious illnesses and potentially life-threatening conditions such as toxic ingestion, opsoclonus-myoclonus, acute cerebellitis and raised intracranial pressure [3]. Therefore a stepwise approach is recommended to exclude other differential diagnoses. In this article, we report a case of acute cerebellar ataxia and the diagnostic approach followed in pediatric inpatient settings.

Case Presentation

A 24-months old boy with no significant medical history presented with difficulty walking and truncal instability for two days. His gait was broad-based and associated with an intentional tremor of feet when taking a step and abnormal eye movements. The parents denied any history of head trauma, loss of consciousness, or altered mental status. He had recently had a viral upper respiratory tract infection (URTI) 2 weeks ago, which was diagnosed and managed by his primary care provider at a local health center and had resolved completely before his admission. He had not been taking any medications in the last few days and had no access to any other substances at home. There was no history of fever, seizures, headache, dizziness, or projectile vomiting. On general physical examination, he was alert, conscious and afebrile. His vital signs were within the normal range for his age and his growth parameters were all in the 50th centile. There were no visible rashes, nor any hypo-pigmented or hyper-pigmented areas on the skin. His pupils were equal bilaterally and reactive to light.

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Received: 16 January, 2023, Manuscript No. cmcr-23-91983; **Editor assigned:** 18 February, 2023, Pre QC No. P-91983; **Reviewed:** 10 February , 2023, QC No. Q-91983; **Revised:** 15 February, 2023, Manuscript No. R-91983; **Published:** 22 February, 2023, DOI: 10.37421/2684-4915.2023.7.243

On further assessment, it was noted that the patient had difficulty standing without support and had a tendency to fall forward when standing independently. His gait was broad-based, associated with a tremor of both feet when taking a step. He exhibited bilateral horizontal nystagmus on extreme medial and lateral gazes. Other cerebellar signs such as dysmetria, heel-shin incoordination and dysdiadokinesia couldn't be assessed due to the patient's young age. He had normal tone, power and reflexes in all four limbs and had good head and neck control. There were no deficits of the cranial nerve; he had no facial asymmetry, weakness, or loss of sensation and he had intact extraocular movements. There was no deviation of uluva or tongue. There was also no neck stiffness or other signs of meningeal irritation. Apart from the abnormal gait; his gross motor, fine motor, language and social developmental milestones were average for his age. The remainder of the physical examination and review of systems was unremarkable.

A complete blood count revealed normal hemoglobin, platelets and white blood cell count and a comprehensive metabolic panel showed normal levels of serum urea, creatine kinase, C-reactive protein and electrolytes (Table 1). An initial computed tomography (CT) was reported normal with no significant intracranial abnormalities and a subsequent magnetic resonance imaging (MRI) following the recommendation of the radiologist was also reported normal with no visible pathological changes in the study. A respiratory viral panel was performed and the RNA of Enterovirus was detected (Table 2).

Table 1. Laboratory results of the patient.

Blood Investigations		
Test Item	Value	Reference Range
Hemoglobin	11.6 g/dL	11.0 - 14.5 g/dL
MCV	79.0 fL	78.0 - 95.0 fL
Platelet Count	247 × 10^9/L	150 - 450 × 10^9/L
White Cell Count	4.1 × 10^9/L	2.4 - 9.5 × 10^9/L
Bicarbonate	23 mmol/l	22 - 29 mmol/L
Chloride	100 mmol/L	98 - 107 mmol/L
Creatinine	60 umol/L	45 - 84 umol/L
Potassium	4.2 mmol/L	3.5 - 5.1 mmol/L
Sodium	136 mmol/L	135 - 145 mmol/L
Urea	3 mmol/L	2.8 - 8.1 mmo/L
C-Reactive Protein	4 mg/L	< 10 mg/L

Table 2. Respiratory viral panel showing that enterovirus RNA was detected in this patient.

Respiratory Viral Panel	
Human Bocavirus DNA	Not detected
Rhinovirus RNA	Not detected
Adenovirus DNA	Not detected
Enterovirus RNA	Detected

The patient was admitted to the ward with the diagnosis of acute ataxia and was hospitalized for 3 days. He received supportive care and was assessed by the physiotherapy team in the hospital on a daily basis. He showed clinical improvement in his gait and by the time he was discharged, the patient was able to ambulate without support. The diagnosis of post-infectious ACA was made on the basis of the history, clinical examination and negative laboratory and imaging studies.

Discussion

ACA is a clinical syndrome that commonly affects children aged 2-6 years old [3]. It is characterized by the rapid deterioration in coordinated movement and truncal instability; while the patient's mental status and function remain intact. Despite the clinical course of pure ACA being benign and self-limiting [1,3]; it is important to exclude other differential diagnoses through a detailed history, careful physical examination and thorough investigations before making a final diagnosis.

On presentation, a comprehensive history should be obtained from the patient as well as the caregiver; details about the presenting symptom aid in having a better perspective about the severity and the urgency of the possible underlying diagnosis. It is essential for the treating physician to inquire about the red flags that can point toward life-threatening conditions; such as altered mental status, any recent history of head or neck trauma, possible toxin ingestion at home, symptoms of raised intracranial pressure like severe, intractable headache and projectile vomiting. It is also equally important to ask questions about the patient's condition prior to the onset of ataxia; as most cases of pure ACA tend to be associated with a recent viral infection. Examples of the possible causative agents include and not limited to the following: echovirus, enterovirus, hepatitis A, human herpesvirus 6, Ebstein-Barr virus measles, mumps and parvovirus B19 [4].

When approaching a patient presenting with acute ataxia; it is important to take into account the general physical examination; and then subsequently focus on a detailed neurological assessment. The child's overall condition on presentation and vital signs give clues about the potential diagnosis; an agitated or ill-appearing patient with abnormal vital signs should alert the physician that a diagnosis of ACA is unlikely. The neurological examination must focus on higher mental functions, speech disturbances, focal deficits, cerebellar signs and four limbs' tone, power and reflexes.

As the diagnosis of ACA is that of exclusion, diagnostic laboratory tests and neuroimaging, MRI in particular [5], must be ordered promptly as the key differential of acute cerebellitis cannot be ruled out without a contrast MRI [6]. The results of these investigations are usually normal as in this case and thus provide solid evidence against other differentials such as intracranial tumors, vascular events, opsoclonus-myoclonus syndrome (OMS) and acute disseminated encephalitis (ADEM). In our case, a diagnosis of post-infectious ACA was made on the basis of the patient's presentation and negative investigations.

The pathogenic mechanism of post-infectious ACA had not been definitively established; however, it is believed to have an underlying autoimmune process as there are reported cases where antiviral antibodies, as well as autoantibodies against centrosomes, glutamate receptor delta 2 and myelin-associated glycoprotein, were isolated from patients' serum and/or cerebrospinal fluid (CSF) [4]. ACA carries a good prognosis, especially in younger children; it typically resolves without any long-term sequelae within 2-3 weeks of presentation. A worse prognosis had been associated with Epstein-Barr virus infection and in patients presenting at an older age [4].

Conclusion

Post-infectious ACA is the commonest cause of ataxia in children. It is a self-limiting benign syndrome that resolves without any sequelae and only requires supportive care. Despite that, it is essential that the treating physician is aware of other differential diagnoses and approaches patients presenting with acute ataxia appropriately. The focus should always be on excluding life-threatening and serious causes of acute ataxia through comprehensive history taking, physical examination and investigations. Throughout the course of admission, patients diagnosed with ACA must be observed for any deterioration and the diagnosis should be reconsidered.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Abdulaal, Hawra. "Post-infectious Acute Ataxia in Children: A Case Report." *Clin Med Case Rep* 7 (2023): 243.