

Acute cerebellar ataxia in a child: Rotavirus coinfection with SARS-CoV-2

¹Sevgi YİMENİCİOĞLU

²Ayşen AKSOY GENÇ

³Ayşe TEKİN YILMAZ

⁴Murat ŞAHİN

¹Division of Pediatric Neurology,
Department of Pediatrics, Eskişehir City
Hospital, Eskişehir, Turkey

²Department of Pediatrics, Eskişehir
City Hospital, Eskişehir, Turkey

³Division of Pediatric Infection,
Department of Pediatrics, Eskişehir City
Hospital, Eskişehir, Turkey

⁴Department of Radiology, Eskişehir
City Hospital, Eskişehir, Turkey

ORCID ID

SY : 0000-0002-1598-4423

AAG : 0000-0003-0265-9629

ATY : 0000-0002-5116-0181

MŞ : 0000-0001-8141-507X



ABSTRACT

Acute cerebellar ataxia (ACA) is more common in childhood. Stroke, infectious, toxic, immune-mediated, paraneoplastic, structural, and metabolic diseases, and vitamin deficiency are the main diseases causing ACA. There may be neurological symptoms in rotavirus and SARS-CoV-2 infection. The objective of this case report is to highlight ACA which was seen as a coinfection in the course of rotavirus infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) without fulminant course.

Keywords: Acute cerebellar ataxia, rotavirus, SARS-CoV-2.

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Correspondence: Sevgi YİMENİCİOĞLU, MD. Eskişehir Şehir Hastanesi, Çocuk Sağlığı ve Hastalıkları Anabilim Dalı, Çocuk Nöroloji Kliniği, Eskişehir, Turkey.

Tel: +90 222 611 40 00 **e-mail:** sevgifahri@yahoo.com

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INTRODUCTION

Ataxia is impaired coordination of motor activity with an inability to ambulate. Ataxia manifests as an ataxic gait often described as a “wide-based gait” with “truncal instability.” Young children may refuse to ambulate.^[1,2] Acute cerebellar ataxia (ACA) is a syndrome that occurs in less than 72 h, in previously healthy subjects. The main groups of diseases that may cause acute ataxia are stroke, infectious, toxic, immune-mediated, paraneoplastic, vitamin deficiency, structural lesions, and metabolic diseases.^[1] The most frequent cause of ACA is infections in childhood.^[1,2] It results from cerebellar immune-mediated inflammation during or after infectious processes, or after vaccination.^[1] Epstein–Barr virus, influenza A and B, mumps, varicella-zoster, Coxsackie virus, rotavirus, echovirus, and mycoplasma are common agents causing ACA.^[1] Both rotavirus and coronavirus have neurotrophic features. They may cause central nervous system (CNS) symptoms.^[3,4]

Previously, ACA with SARS-CoV-2 was reported. Also, some cases of coinfections of respiratory pathogens with SARS-CoV-2 have been reported.^[5] Copathogens of SARS-CoV-2 constitute viruses such as influenza, rhinovirus/enterovirus, parainfluenza, metapneumovirus, influenza B virus, and human immunodeficiency virus.^[5] The main purpose of this case presentation is to emphasize the rotavirus coinfection with SARS-CoV-2 without a fulminant course, and coinfections of SARS-CoV-2 with rotavirus have not been reported earlier.

CASE REPORT

A 2-year-old girl was admitted to the hospital with vomiting and diarrhea. The symptoms had begun 2 days before admission. Based on the characteristic symptoms and a positive stool rotavirus antigen test, rotavirus gastroenteritis has been diagnosed. The patient was the fourth child of consanguineous parents with an uneventful birth. She had a febrile convulsion 1 year ago. On the third day of admission, diarrhea had passed. She had a tendency to sleep, and an unsteady gait while standing still. She had a temperature of 37.3°C, a blood pressure of 80/60 mmHg, a pulse rate of 90 beats/min, and a respiratory rate of 20 min⁻¹. A physical examination revealed no lateralization or neck stiffness. She had nystagmus. She had head titubation, trunk sway, and a staggering gait with impaired tandem. She had intention tremors while she wanted to take something or hold something. She had dysmetria. She could not obey the explanations for dysdiadochokinesia and ataxia during walking. Deep tendon reflexes were normal. The mother reported the contact of the patient with a SARS-CoV-2 PCR positive relative. A nasopharyngeal swab revealed a positive SARS-CoV-2 reverse transcriptase PCR. The patient’s kidney function tests and electrolyte values were within the normal range. C-reactive protein was determined to be 0.1 mg/L. Metabolic screening tests including urinary organic acid, ammonia, lactate, pyruvate, and tandem mass spectrometry (including amino acid metabolism, fatty acid oxidation, organic acid metabolism, and carnitine metabolism disorders) were normal. Parental consent did not permit lumbar puncture. Brain magnetic resonance imaging (MRI), magnetic resonance angiography, and diffusion-weighted images were normal. We diagnosed ACA due to rotavirus coinfection with SARS-CoV-2 based on the clinical findings, a positive rotavirus

antigen test in stool, and a positive SARS-CoV-2 PCR test. The patient was hydrated. Trunk sway disappeared within 3 days, and then dysmetria resolved. She did not have a fulminant course. There was no need for corticosteroids or intravenous immunoglobulin. She recovered within 1 week. She was released with no further treatment.

DISCUSSION

ACA is a syndrome that occurs in previously well children, often presenting as a postinfectious disorder.^[1] In our case, the patient had an existing rotavirus infection. Clinical findings related to ACA have emerged during rotavirus infection without any respiratory symptoms. Formerly, Tomar et al.^[6] reported a 13-year-old male child initially having classical symptoms of COVID-19 and developing cerebellar ataxia with normal brain MRI findings. Sharma et al.^[7] reported a 12-year-old boy and a 10-year-old boy with moderate to severe acute cerebellitis as a rare presentation of COVID-19 with cerebellar symptoms. Rotavirus coinfection with SARS-CoV-2 has not been reported earlier.

Concurrent infection that occurs with an existing infection is referred to as coinfection. In the review of the literature, it was found that rotavirus and SARS-CoV-2 reside and multiply in the mucosa of the upper respiratory and digestive tracts, and both cause upper respiratory tract infections and gastrointestinal tract infections.^[8]

Coronavirus rarely causes acute gastroenteritis in infants.^[8] Lung epithelial cells and enterocytes in the gastrointestinal tract express the receptor protein ACE2, which SARS-CoV-2 uses to enter cells.^[9] Rotavirus causes acute gastroenteritis in young children.^[9] Acute rotavirus gastroenteritis results in various frequencies of CNS involvement, including meningitis, encephalitis, convulsions, encephalopathy, central pontine myelinolysis, and Guillain–Barre syndrome.^[3,10] Rotavirus is a neurotropic virus, and its direct invasion cannot be proven.^[10] Rotavirus may be a possible cause of ACA. Most patients with CNS involvement have a complete recovery; a small number of patients may sustain sequels or die.^[10]

SARS-CoV-2 has neurotropic features and can lead to neurologic consequences. The mechanism of neuroinvasion is still incompletely known.^[4] Headache, tiredness, walking disturbance, cerebral hemorrhage, and cerebral infarction are neurologic manifestations.^[11] ACA due to infection is more common in children and young adults. Nystagmus and dysmetria are the most commonly associated neurologic findings. Gait recovery takes less than 14 days on average. It is a self-limiting disease. If there is an atypical presentation or if there is no improvement after 1–2 weeks, clinicians should consider imaging studies.^[12] This pure cerebellar syndrome may have normal or abnormal brain MRI findings at the onset. Some have T1 sequence abnormalities.^[1,10] Povlow and Auerbach^[13] diagnosed a 30-year-old patient with isolated cerebellar symptoms during SARS-CoV-2 infection without MRI findings. Sharma et al.^[7] described two cases presenting as acute fulminant cerebellitis showing confluent cerebellar hyperintensity with compression of the fourth ventricle and mild ventricular prominence (after external ventricular drain placement) with T2 images in the first patient, confluent cerebellar involvement and compression of the fourth ventricle, and patchy folial enhancement with contrast-enhanced T1 image in the second.^[7]

Our patient was an infant, with gastroenteritis without any respiratory signs or symptoms. The course was not fulminant. This case is important to state the clinical features and progress of the coinfections with rotavirus and SARS-CoV-2. Coinfections may be benign, of course, instead of being fulminant.

Statement

Informed Consent: Written informed consent was obtained from the patient's family for the publication of the case report.

Peer-review: Externally peer-reviewed.

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