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CASE REPORT

# Acute cerebellar ataxia in a patient with maple syrup urine disease: a case report

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## Abstract

**Introduction:** Maple syrup urine disease (MSUD) is an inborn error of amino acid metabolism, potentially resulting in metabolic intoxication crises accompanied by neurological manifestations. **Case report:** This article reports a case involving a six-year-old boy with MSUD who developed a post-infectious neurological disorder following COVID-19. Initially presenting with fever, cough, and ataxia, he was diagnosed with a metabolic crisis due to COVID-19. Despite an initial recovery, the patient experienced a relapse characterized by ataxia, tremor, and dysarthria, necessitating readmission. A subsequent investigation ruled out an MSUD crisis. A lumbar puncture and MRI failed to rule out a demyelinating acute disorder. Methylprednisolone treatment yielded no improvement, prompting the initiation of intravenous immunoglobulin, which led to improvement after 14 days. **Discussion:** The case posed challenges due to the overlapping symptoms between MSUD crises and post-infectious neurological conditions. This highlights the complexity of managing these patients, particularly due to the potential for more vigorous manifestations and slower recovery.

***Keywords:*** MSUD. PINS. COVID-19. Ataxia.

## *Ataxia cerebelar aguda num doente com leucinose: relato de caso*

## Resumo

**Introdução:** A leucinose é uma doença hereditária do metabolismo dos aminoácidos que pode levar a crises de intoxicação metabólica com manifestações neurológicas. **Relato de caso:** Este artigo relata um caso de distúrbio neurológico pós-infeccioso numa criança de 6 anos com leucinose após infeção por SARS-CoV-2. Inicialmente apresentou-se com febre, tosse e ataxia, testou positivo para SARS-CoV-2 e foi diagnosticado com uma crise metabólica. Após a recuperação, apresentou um quadro de ataxia, tremores e disartria, levando a um reinternamento. Após a exclusão de intoxicação metabólica, realizou punção lombar e ressonância magnética, não sendo possível excluir uma alteração desmielinizante. Sem melhoria sob

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metilprednisolona, foi tratado com imunoglobulina intravenosa, observando-se recuperação ao fim de 14 dias. **Discussão:** Este caso revelou-se desafiante devido à sobreposição de sintomas entre crises de leucinose e condições neurológicas pós-infecciosas. Destaca-se a complexidade da abordagem destes doentes particularmente por poderem apresentar manifestações mais exuberantes e uma recuperação mais lenta.

***Palavras-chave:*** MSUD. PINS. COVID-19. Ataxia.

## Keypoints

**What is known**
- Patients with MSUD are susceptible to metabolic decompensation crises featuring neurological symptoms during episodes of catabolism.
- Post-infectious neurological syndromes (PINS) have been linked to several viruses, including coronaviruses.
- Distinguishing between decompensation crises and PINS can be challenging in individuals with chronic neurological or metabolic disorders.

**What is added**
- The overlapping symptoms between MSUD crises and PINS pose a diagnostic, and thus therapeutic, challenge.
- Including prior imaging in these patients could provide valuable insights, aiding in the differentiation between acute neurological post-infectious inflammation and chronic manifestations of MSUD.

## Introduction

Maple syrup urine disease (MSUD) is an amino acid catabolic disorder that results in the inability to break down branched-chain ketoacids due to the absence of the appropriate enzyme (branched-chain ketoacid dehydrogenase). Branched-chain amino acids (valine, leucine, and isoleucine) and branched-chain ketoacids therefore accumulate. This accumulation interferes with the cerebral amino acid transport system and myelin formation. The accumulation of ketoacids may also lead to metabolic encephalopathy and cerebral edema[1]. During periods of catabolism, such as infection, surgery, exercise, or fasting, individuals with MSUD may present with metabolic intoxication crises, which are mainly managed with nutritional interventions. These crises may present clinically as refusal to feed and vomiting and are accompanied by neurological signs such as ataxia, dystonia, hyperactivity, somnolence, or stupor. In these patients, viral infections, such as SARS-CoV-2, may trigger metabolic crises.

Symptoms such as fever, cough, and anosmia are commonly reported in SARS-CoV-2 infection. However, the literature has also described rare neurological symptoms and post-infectious syndromes, which pose new challenges to our clinical reasoning. In patients with pre-existing neurological or metabolic disorders, differentiating between decompensation crises and acute or post-infectious neurological symptoms can be challenging.

Post-infectious neurological syndromes (PINS) are rare heterogeneous entities with a post- or para-infectious onset. Typically, they manifest after viral or bacterial infections[2]. Numerous pathogens, including SARS-CoV-2, have been associated with these clinical entities. One example is acute post-infectious cerebellar ataxia (APCA), which usually manifests a few days to three weeks after a febrile illness[3]. It typically presents with rapid onset and progressive symptoms including gait disturbance, fine motor control problems, nystagmus, vomiting, and dysarthria. Several other neurological autoimmune disorders are believed to occur through post-infectious mechanisms. Acute disseminated encephalomyelitis (ADEM) is a rare demyelinating disease of the central nervous system (CNS) and typically appears four to 13 days after a febrile illness[4]. It presents with encephalopathy, multifocal neurological deficits, pyramidal signs, cerebellar ataxia, cranial neuropathies, myelopathy, and seizures[5,6].

With this article, the authors intend to report the first case described in the literature of PINS in a six-year-old boy with MSUD, after a SARS-CoV-2 infection.

## Case report

A six-year-old boy of Roma consanguineous descent with a neonatal diagnosis of a classic, severe phenotype of MSUD (homozygous c.117delC (p.R40fs*23) was seen at the Emergency Room (ER). The boy had been diagnosed in the neonatal period (leucine at the time of diagnosis (six days): 2400 μM/L), presenting with a poor general state, hypoglycemia, hypoactivity, failure to thrive, and seizures in the first week of life. He received renal replacement therapy during this first admission. He also presented with bradypnea, requiring non-invasive ventilation followed by a period

**Table 1.** Amino acid chromatography

| | MSUD acute decompensation | 2 weeks later | Reference values |
|---|---|---|---|
| Valine ( µM/L) | 426.2 | 423.8 | 80.2-245.7 |
| Isoleucine (µM/L) | 273.3 | 426.6 | 26.7-52.6 |
| Allo isoleucine (µM/L) | 108.8 | 456.0 | Not dosable |
| Leucine (µM/L) | 889.9 | 42.9 | 46.5-109.0 |

of mechanical ventilation. After the diagnosis and before the additional episode described, the patient presented with multiple decompensation crises, with leucine values ranging from 500-1000 µM/L, mainly concurrent with infections. In these crises, he usually presented with ataxia. The crises gradually resulted in cognitive impairment and a delay in the acquisition of psychomotor developmental milestones. However, from the age of four until the episode being reported he presented an otherwise normal baseline neurological examination, with no ataxia or other movement disorders.

At the ER, he presented with two days of fever, cough, excessive sleepiness, and a slight ataxic gait. A nasopharyngeal swab, performed by reverse transcription polymerase chain reaction, confirmed SARS-CoV-2 infection. A complete blood analysis evaluation was performed, including amino acid chromatography (AAC) (Tables 1 and 2).

He was diagnosed with a metabolic decompensation (maximum leucine 889.9 µM/L) related to SARS-CoV-2 infection, initially managed at home. However, six days later he was admitted to the hospital ward. He was already afebrile but presented axial and appendicular ataxia. The rest of the neurological examination was normal. Natural protein intake was interrupted, and synthetic protein as well as isoleucine and valine supplements were increased according to the treatment protocol. He had a successful recovery both clinically and in terms of the laboratory tests and was discharged after a week.

After two uneventful weeks, he developed sudden onset ataxia, tremor (videos 1 and 2), and excessive sleepiness, prompting readmission two days after the symptoms began. Vital signs were stable, with no fever or meningeal signs. The neurological examination revealed dysarthria, appendicular ataxia with dysmetria, intention tremor, axial ataxia, and brisk osteotendinous reflexes; cranial nerves, and motor and sensory functions were normal. The rest of the physical examination was unremarkable. No findings suggested that pediatric inflammatory multisystem syndrome temporally associated with COVID-19 (PIMS-TS) was observed.

Blood tests and AAC were performed (Tables 1 and 2), which showed normal values a few days later. Before the AAC results came in, and despite initially receiving treatment for an MSUD crisis, the patient did not improve. The clinical and laboratory results were also not compatible with metabolic decompensation. Thus, further investigation was needed.

Magnetic resonance imaging (MRI) and a lumbar puncture (LP) were performed, yet they did not provide a clear etiology. The cerebrospinal fluid (CSF), in addition to cytochemical and culture analyses, was also tested for auto-immune encephalitis (Table 2), and an electroencephalogram was performed, revealing slow, abrupt waves, with no paroxysmal activity.

The results were consistent with both APCA with previous MSUD-related findings and ADEM. Since no previous MRI had been performed, it was difficult to completely rule out the latter hypothesis.

The patient was started on methylprednisolone 30 mg/kg/day for five days, yet no clinical improvement was observed. A second MRI was then performed, and the findings remained unchanged. He was then started on intravenous immunoglobulin (IVIG) for two days (1 g/kg/day).

After the second treatment, the patient gradually recovered and was discharged after 14 days, only experiencing headaches, a slight intention tremor, and dysmetria (video 3). Steroids were tapered off over four weeks.

## Discussion and conclusions

Coronaviruses were already recognized as important human pathogens. Still, the significant number of infections on a global scale has led to an increased understanding of the manifestations of this pathogen and the associated clinical conditions[7].

SARS-CoV-2 infection has been linked to multiple neurological symptoms and syndromes. The most

**Table 2.** Blood and CSF analysis

| | MSUD acute decompensation | 2 weeks later | Recovery | Reference values |
|---|---|---|---|---|
| **Blood test evaluation** | | | | |
| Hemoglobin (g/dL) | 10.9 | 9.9 | 9.6 | 11.5-15.5 |
| WBC (× $10^9$/L) Neutrophils, n (%); Lymphocytes, n (%) | 6.930 (N, 76.7; L, 18.8) | 8.630 (N, 71.7; L, 22.7) | 17.60 (N, 38.4; L, 55.2) | 5.0-13.0 (N, 2.0-8.0; L, 1.0-5-0) |
| Platelets (× $10^9$/L) | 448 | 951 | 922 | 180-400 |
| Glucose (mg/dL) | 76 | 88 | 76 | 70-110 |
| Urea (mg/dL) | 6 | 20 | 14 | 16-49 |
| Creatinine (mg/dL) | 0.37 | 0.31 | 0.36 | 0.29-0.47 |
| AST/ALT (U/L) | 280/130 | 121/ 164 | 43/126 | 0-31/0-41 |
| GGT (U/L) | 248 | 271 | 127 | 0-60 |
| C-reactive protein (mg/dL) | - | < 0.1 | - | < 0.5 |
| Anti-nuclear antibodies | - | - | Negative | - |
| Anti-LKM antibodies | - | - | Negative | - |
| Anti-DS-DNA antibodies | - | - | Negative | < 27 |
| Anti-neutrophil cytoplasmic antibodies (c-ANCA/PR3) | - | - | Negative | < 20 |
| Anti-neutrophil cytoplasmic antibodies (p-ANCA/MPO) | - | - | Negative | < 20 |
| SARS-CoV-2 antibodies | - | 44.70 U/mL | - | ≥ 0.8 = Positive |
| **CSF analysis** | | | | |
| Color | - | Transparent | - | - |
| Proteins (mg/dL) | - | 14.3 | - | 15-45 |
| Glucose (mg/dL) | - | 64 | - | 40-70 |
| Cells (/mm3) | - | 1.6 (no predominance) | - | < 10 |
| Culture | - | *Staphylococcus epidermidis* (likely contaminated) | - | - |
| Anti-Aquaporin-4 | - | < 1/10 (negative) | - | < 1/10 |
| Anti-MOG | - | < 1/20 (positive)* | - | < 1/10 |

commonly reported symptoms in various case series include headaches, myalgias, encephalopathy, and dizziness[8]. Movement disorders such as ataxia, motor and sensory deficits, and seizures are less commonly reported[7]. Several cases of autoimmune para-infectious and post-infectious neurological manifestations, such as ADEM[9,10] and APCA have also been documented[11,12].

In our patient, an initial MSUD acute decompensation was suspected due to the clinical features that were consistent with previous crises. However, this diagnosis was later ruled out based on both the AAC results and the treatment's lack of effectiveness.

Subsequently, both APCA and ADEM were considered possible diagnoses. Our patient exhibited features consistent with APCA, including ataxia, tremor, irritability, and dysarthria. ADEM presents with encephalopathy, which in our patient manifested as somnolence and attention impairment, and patients may also present irritability and ataxia. The time sequence was also consistent with both diagnoses.

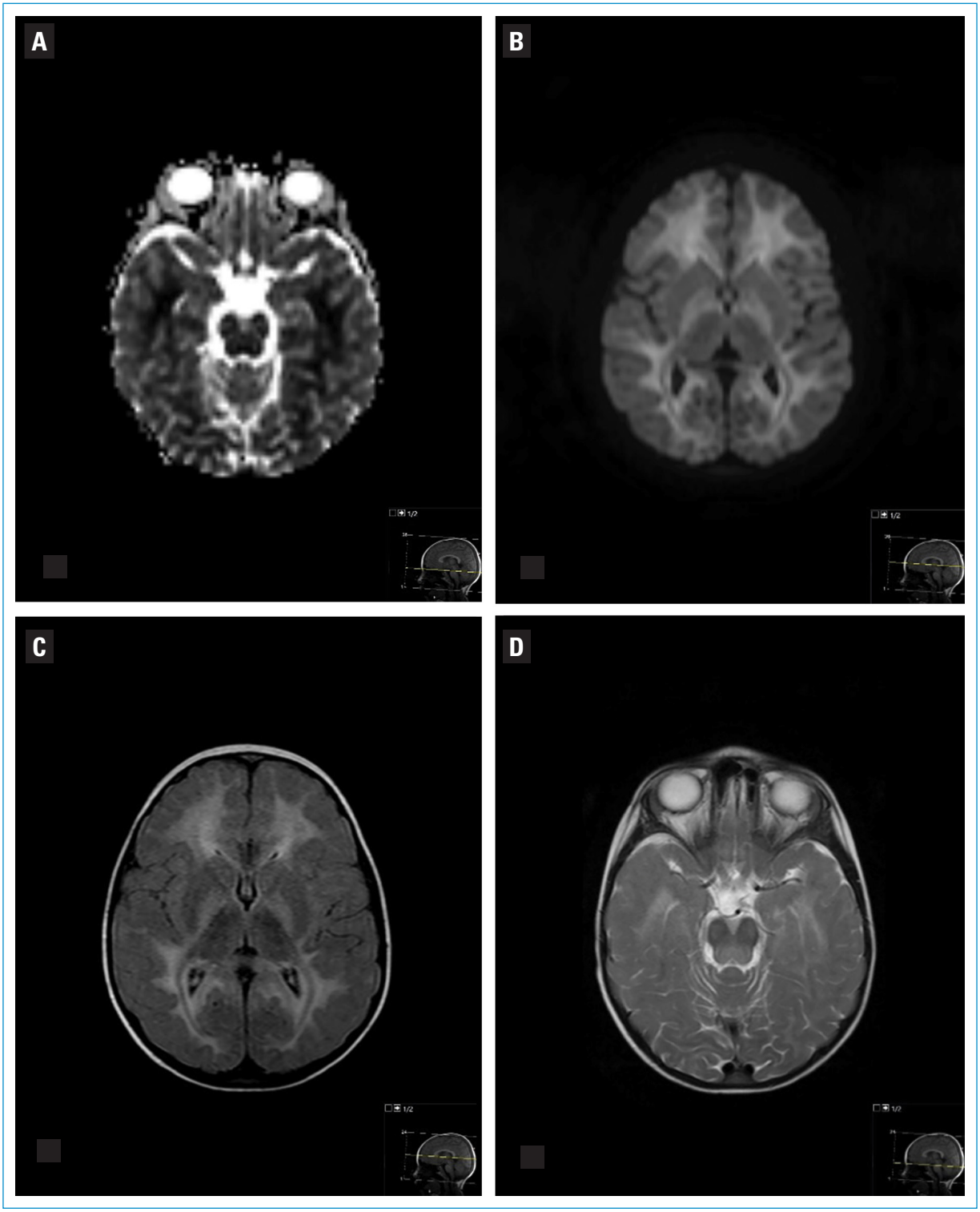


**Figure 1. A-D:** both MRIs showed diffuse restricted diffusion (high diffusion-weighted imaging (DWI) signal with a low apparent diffusion coefficient (ADC)) involving the bilateral cerebellar white matter, the dorsal part of the pons and brainstem, the dorsal limb of the internal capsule and the frontotemporoparietal white matter. There is also T2/FLAIR (fluid-attenuated inversion recovery) hyperintensity in the above-mentioned regions. The areas of restricted diffusion represent cytotoxic oedema. **A:** ADC, **B:** DWI, **C:** FLAIR, and **D:** T2.

Although APCA was considered more likely, ADEM could not be ruled out, so an MRI and lumbar puncture were performed. Generally, patients with APCA may have normal imaging or show bilateral diffuse abnormalities of the cerebellar hemispheres[11,13,14]. There are no pathognomonic alterations. However, the imaging of patients with ADEM usually reveals bilateral and asymmetrical lesions in the white matter, typical of demyelination and diffusion restriction, which was present in this patient[15].

Again, the pre-existing metabolic disorder posed a confounding factor. Initially, the clinical picture was more compatible with APCA. However, since there was no prior CNS imaging results from this patient, the MRI findings could either be indicative of an acute demyelinating syndrome or reflect previous alterations from the metabolic disease. Since ADEM could not be ruled out, the patient was started on methylprednisolone and subsequently received IVIG due to the lack of clinical improvement.

As for the treatment and clinical course, APCA usually requires only supportive treatment, although some case reports of refractory illness require glucocorticoids or immunoglobulin[16,17]. Conversely, patients with ADEM should be started on immediate immune-modulating therapy, with glucocorticoids considered the first-line treatment and immunoglobulin or plasma exchange a second-line option. Regarding the clinical course, patients with APCA typically experience symptoms for 10 to 12 days and completely recover within two to three weeks[13,14]. Usually, patients with ADEM make a complete recovery over four to six weeks[18], although some may experience persistent sequelae[19].

APCA after SARS-CoV-2 infection has been reported in children[11,20,21] and also in adults[22,23] worldwide. Tomar et al.[11] reported a case of a 13-year-old who was treated with methylprednisolone and recovered over 20 days. O'Neill et al.[21] reported another case of a five-year-old who received only supportive and physical therapy and made a complete recovery after two months. These cases developed 10 and eight days after the initial COVID-19 diagnosis, respectively. Sanchéz-Morales et al.[20] reported several neurological manifestations of COVID-19, including APCA. No mention of treatment was made, and the infant made a full recovery.

ADEM after SARS-CoV-2 infection has also been reported[23,24], although it remains a rare post-infectious autoimmune complication. Most of the published cases involve adults. According to a review[24], the median time between the documentation of the SARS-CoV-2 infection and the neurological symptoms was 15.5 days. Treatment varied among the reported cases, with some patients receiving steroids, others receiving IVIG, and some reports stating that no treatment was administered.

Due to the similarity between a MSUD acute decompensation and the new onset neurological condition, our patient presented a challenge. Furthermore, the MRI findings were equivocal as it was unclear whether they were caused by the metabolic disorder or derived from an acute neurological condition. In patients with classic, severe phenotype MSUD, atypical symptoms or unusual recovery patterns may be observed. Thus, a prior MRI would have been valuable in this patient to differentiate between acute neurological post-infectious inflammation and chronic MSUD neuro-image manifestations in more severe cases.

To our knowledge, this was the first case report of acute cerebellar ataxia after SARS-CoV-2 infection in a patient with MSUD.

To conclude, SARS-CoV-2 infection and its post-infectious consequences pose new challenges, particularly in patients with severe inborn errors of metabolism that manifest with neurological symptoms. In such cases, decompensation crises may share similar symptoms. These patients may also present with more vigorous manifestations with slower recovery.

## Data availability

The video materials referenced in this article are available from the corresponding author upon reasonable request.

## Author contributions

C. Castro drafted the paper; P. Lipari contributed to the idea behind it, drafted part of the paper and substantively revised it; I. Carneiro drafted part of the work and contributed to the interpretation and incorporationg of the images; P. Janeiro, J. Coelho, S. Quintas and A. Gaspar made substantial contributions to the idea behind the paper and substantively revised it. F. Furtado substantively revised the work. All authors read and approved the final manuscript.

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## Conflicts of interest

None.

## Ethical considerations

**Protection of humans and animals.** The authors declare that no experiments involving humans or animals were conducted for this research.

**Confidentiality, informed consent, and ethical approval.** The authors have followed their institution's confidentiality protocols, obtained informed consent from patients, and received approval from the Ethics Committee. The SAGER guidelines were followed according to the nature of the study.

**Declaration on the use of artificial intelligence.** The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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