




Cerebelitis post infecciosa en niños caso clínico

Postinfectious cerebellitis in children case report

- ¹ Ipolita Cesibel Marin Zaruma
Catholic University of Cuenca
ipolita.marin.56@ucacue.edu.ec  <https://orcid.org/0009-0003-1183-2494>
- ² Rosa Veronica Sumba Portilla
Catholic University of Cuenca, Cuenca, Ecuador.
maria.merchan@ucacue.edu.ec  <https://orcid.org/0000-0002-1788-4389>
- ³ Rodrigo Jose Mendoza Rivas
Catholic University of Cuenca, Cuenca-Ecuador.
rodrigo.mendoza@ucacue.edu.ec  <https://orcid.org/0000-0001-6020-9226>



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Resumen

Introducción: la cerebelitis aguda posinfecciosa es una patología infrecuente y su diagnóstico es difícil, ya que la presentación clínica y su curso son muy variables. Sus causas están asociadas a infecciones virales previas, vacunación reciente o ingesta de alimentos contaminados. **Objetivo:** determinar el manejo y terapéutica de Cerebelitis Post infecciosa en un caso clínico con el fin de establecer elementos novedosos e instructivos de la enfermedad. **Metodología:** Análisis de caso clínico tipo descriptivo retrospectivo, para la recolección de información se realizó una revisión de historia clínica; para la descripción de la enfermedad se realizó una recopilación de información en bases de datos reconocidas como: *Scopus, Pubmed, web of Science, Lilacs*. **Resultados:** se presenta un caso clínico de un paciente masculino de 2 años 11 meses dismetrico, discronometrico, con nistagmo horizontal, tembloroso, atáxico, hipotónico, marcha en estrella de babinsky. inestabilidad postural. Con IGG para COVID19 de 265,8 (positivo) diagnosticado con cerebelitis que mediante el tratamiento antimicrobiano respectivo, presenta una clara mejoría clínica. **Conclusión.** El diagnóstico de la cerebelitis post infecciosa puede llegar a ser difícil al ser una patología infrecuente, un examen de imágenes confirma el diagnóstico clínico de cerebelitis aguda. sin embargo, en un contexto urgente, es posible que no se realice la resonancia magnética, especialmente en casos de inestabilidad clínica del paciente sin embargo el papel de la imagen craneoencefálica en el seguimiento de estos pacientes está relacionado con la detección de posibles complicaciones. **Área de estudio general:** medicina. **Área de estudio específica:** cardiología. **Tipo de estudio:** Casos clínicos.

Abstract

Introduction: Acute post-infectious cerebellitis is an infrequent pathology and its diagnosis is difficult since the clinical presentation and its course are very variable. Its causes are associated with previous viral infections, recent vaccination, or ingestion of contaminated food. Objective: to determine the management and therapeutics of post-infectious cerebellitis in a clinical case to establish novel and instructive

elements of the disease. **Methodology:** Analysis of a retrospective descriptive clinical case, for the collection of information a review of clinical history was performed; for the description of the disease a compilation of information in recognized databases such as: Scopus, Pubmed, web of Science, Lilacs was performed. **Results:** we present a clinical case of a male patient aged 2 years 11 months, dysmetric, dyschronometric, with horizontal nystagmus, tremor, ataxic, hypotonic, babinsky star gait and postural instability. With IGG for COVID19 of 265.8 (positive) diagnosed with cerebellitis that through the respective antimicrobial treatment, presents a clear clinical improvement. **Conclusion:** The diagnosis of post infectious cerebellitis can become difficult as it is an infrequent pathology, an imaging examination confirms the clinical diagnosis of acute cerebellitis. However, in an urgent context, MRI may not be performed, especially in cases of clinical instability of the patient however the role of cranioencephalic imaging in the follow-up of these patients is related to the detection of complications.

Introduction

The presence of viruses usually causes mild illnesses in children, affecting the respiratory tract, gastrointestinal system and eyes, and usually resolve on their own, however, it is a rare cause of Postinfectious Cerebellitis, acute cerebellitis is a rare condition that is often considered within the group of acute postinfectious cerebellar ataxia despite its distinctive clinical and imaging features, When evaluating patients with possible tumor syndromes, it is important to also consider rarer inflammatory syndromes that may masquerade as neoplasia; Postinfectious cerebellitis is a syndrome, so clearly knowing its clinical manifestations and approach is important to limit complications that occur over time (1).

Acute cerebellitis following infection is a fairly common emergency encountered by the pediatric neurologist in daily life. The variety of possible causes of acute ataxia in children is extensive, however, a detailed and accurate clinical history is essential to distinguish them and a careful clinical examination can narrow it down and help guide investigations.(2). For the assessment, it is important to evaluate the diagnostic aids that allow the certainty of the pathology and to provide the patient with an effective and timely

treatment. The conditions that can simulate brain tumors or vascular malformations are rare. The adequate identification of these is crucial to avoid unnecessary surgical interventions, especially when they can respond to medical treatment. In the back of the skull, acute cerebellitis is an autoimmune inflammatory syndrome that is generally differentiated from a tumor by affecting the cerebellum symmetrically and bilaterally (3).

This syndrome predominantly affects children and may occur at the same time as infection, after infection or after vaccination. The typical presentation is the experience of head pain, nausea and a reduced level of consciousness in a child; ataxia may also be present. Radiographically, T2-weighted magnetic resonance imaging (MRI) reveals bilaterally symmetrical cerebellar hyperintensities, cerebellar edema and possible obstructive hydrocephalus. High amounts of steroids are used in treatment and the vast majority of patients achieve full recovery.(3). Although usually bilateral, acute cerebellitis has also been reported to occur unilaterally and is termed hemicerebellitis; cases of pseudotumoral hemicerebellitis, with symptoms and radiographic features mimicking space-occupying lesions, have also been reported; cerebellar hemorrhage may also cause a mass effect and obstructive hydrocephalus and may be due to vascular malformations, fistulas, or, rarely, complications of occult neoplasia.(4).

Acute post-infectious cerebellar syndromes show a wide spectrum of acute severity and may occur with acute febrile illness or receipt of vaccines, historically, chickenpox has been the most frequent reason, related to a quarter of the cases in large clusters, post-infectious cerebellitis is frequent during chickenpox in childhood and is associated with prolonged hospitalization, the neurological presentation is mainly characterized by ataxia, difficulty speaking, vomiting, headache and dysmetria; this study aims to describe a clinical case of post-infectious cerebellitis in children(4).

It is the main cause of acute cerebellar dysfunction in children. Previously, this pathology is rare to identify this condition, since its clinical manifestation and evolution are extremely variable and complicated to diagnose, it is considered an underdiagnosed disease in the pediatric field, therefore, the true frequency of the situation remains in the dark.(5)Acute cerebellitis is usually infectious, post-infectious or post-vaccination, the rarest causes include toxic ones, the clinical presentation is usually benign, however, severe cases with brain stem compression may present as coma. Based on three clinical situations in children, the authors briefly review this phenomenon, with special emphasis on its causes and imaging findings.(6).

Acute cerebellitis is an acute neurological condition characterized by cerebellar ataxia or dysfunction attributable to a recent or concurrent infectious disease, recent vaccination, or medication ingestion, and in which there is evidence of predominantly cerebellar inflammation on magnetic resonance imaging.(7). It may have a fulminant course, with cerebellar edema resulting in permanent damage or even sudden death.(8)This is an

urgent medical situation that requires great attention, being considered the most serious form of infectious-inflammatory disorders of the cerebellum, with a high risk of complications and death.(9).

Very little is known about the pathophysiology of this disease, and it is still a point to be clarified. A significant edematous process has been identified with the presence of infiltration of lymphocytes and eosinophils without showing signs of demyelination; an autoimmune mechanism has also been suggested for acute cerebellitis in view of its post-infectious origin and the detection of autoantibodies in some patients against Purkinje cells, centrosomes, glutamate receptors, gangliosides, cardiolipin and glutamic acid decarboxylase.(10). Infectious agents associated with acute cerebellitis include several types of viruses. An autoimmune mechanism for acute cerebellitis has also been suggested in view of its post-infectious origin and the detection of autoantibodies in some patients against Purkinje cells, centrosomes, glutamate receptors, gangliosides, cardiolipin and glutamic acid decarboxylase.(11).

The etiology of acute cerebellitis is unknown in almost two-thirds of patients, and the remaining third is infectious. These symptoms have been found to be related to different infectious agents such as measles, rubella, mumps, chickenpox, Epstein-Barr virus, cold sores, rotavirus, cytomegalovirus, polio, influenza, respiratory syncytial virus, Coxsackie virus, Salmonella, Borrelia, Bordetella, Coxiella, Streptococcus pneumoniae and Mycoplasma pneumoniae.(12). MRI is the most accurate test to evaluate the cerebellum and brain stem and should be considered the gold standard diagnostic tool. It is the best way to detect inflammation in the brain and spinal cord. Increased brightness in the cerebellum may be seen on MRI images, descending herniation may be indicative of acute infectious cerebellitis, MRI findings of acute cerebellitis have previously been classified into 3 main groups: bihemispheric cerebellitis, hemispheric cerebellitis, and cerebellitis with encephalitic findings(13,14).

Mild or moderate acute cerebellitis was associated with MRI changes at the level of the cerebellar cortex and, in more severe cases, the cerebellar cortex and cerebellar white matter, composed of the deep gray matter that houses the dentate nuclei, are preserved, a finding that helps differentiate them from metabolic disorders.(15). Not all children with acute cerebellitis undergo routine imaging tests; therefore, its true incidence is unknown, serum markers of infection or inflammation, such as serum white blood cell count, C-reactive protein, or erythrocyte sedimentation rate, may be elevated. However, these markers are often nonspecific and not useful.(16).

When a lumbar puncture is possible, cerebrospinal fluid microscopy may be normal; however, pleocytosis with a predominance of monocytes is often seen. Cerebrospinal fluid biochemistry is also rarely useful. The poor yield of lumbar puncture must be weighed against the risk of increased intracranial pressure and cerebellar tonsillar

herniation.(17)Electroencephalography (EEG) is often not useful, mostly non-specific findings, since it depends on the neuronal activity and diseases of the patient, and in childhood it has a low sensitivity and specificity, being very rarely chosen for diagnosis as such.(18).

At the critical juncture, a CT scan is advantageous in ruling out other possible reasons for the symptoms and in detecting the sudden onset of hydrocephalus or severe compression of the brain stem. Findings may include mild dilation of the third and lateral ventricles, or obvious hydrocephalus, as well as a reduced fourth ventricle and crowding of structures in the posterior fossa. It is also particularly useful in the early diagnosis of surgically treated sequelae.(19)There is a long list of pathologies that can simulate the clinical presentation of acute cerebellitis, such as tumors, abscesses, intoxication, metabolic disease, hereditary degenerative disorders, meningitis, encephalitis and acute disseminated encephalomyelitis.(20).

Neurological sequelae have been reported in 27% of cases suffering from this disease, among which tremor, ataxia, cognitive deficit, deterioration of motor and verbal functions have been described in the follow-up.(21). It is essential to act quickly to improve the results, therefore, if a clinical picture is observed that suggests a disease in the back of the skull, the possibility that this is the condition should be considered.(22). It shows a wide range of clinical evolution: from a benign self-limiting course to a sudden and severe presentation with life-threatening consequences due to compression of the posterior fossa, the appearance of acute hydrocephalus and elevated pressure within the skull.(23)In 92% of children, this disease manifests itself in a prodromal manner. The average duration from the onset of initial symptoms to the development of acute cerebellitis was one week. The most common prodromal illnesses were respiratory tract infections, diarrheal illnesses or gastroenteritis, headaches, and unspecified febrile illnesses.(24).

Symptoms of elevated intracranial pressure due to severe swelling include headache, upset stomach, nausea, impaired mental alertness, and/or drowsiness. It is usually due to obstructive hydrocephalus caused by the inflammatory process compressing the Sylvian aqueduct and the fourth ventricle.(25).

Cerebellar signs have been grouped into 4 categories(26):

- Neocerebellar syndromes that manifest mainly with dyssynergia, dysmetria, dysdiadochokinesia and dysarthria.
- Paleocerebellar syndromes that manifest mainly with gait disturbances and ataxias.
- Vestibulocerebellar or flocculonodular syndrome presented as imbalance with ataxias.

Neuropsychological features such as mutism and pathological laughter were recorded as separate cerebellar syndromes. The most common symptoms at presentation were headache, nausea, and fatigue; whereas the most frequent cerebellar signs included lack of coordination, difficulty speaking, and errors in precision of movements.(25) Fulminant cerebellitis with cerebellar inflammation usually leads to the creation of obstructive hydrocephalus due to obstruction of normal CSF flow by compression of the fourth ventricle, and the inflamed cerebellum eventually compresses brain stem structures. Children may present with symptoms of elevated intracranial pressure, including coma, without any signs of cerebellar dysfunction, the rate of tonsillar herniation and obstructive hydrocephalus were 73.3% and 26.6%, respectively.(27).

The condition is usually self-limiting, but can sometimes lead to permanent disability and may even be fatal due to complications related to compression of the formations in the back, acute hydrocephalus and/or intracranial hypertension. The presence of acutely increased intracranial pressure with inflammation is a life-threatening situation that requires emergent neurosurgical intervention by decompressive craniotomies or cerebrospinal fluid shunts to prevent brain stem compression and herniation.(28).

There is still no general agreement on how to treat acute cerebellitis, as its evolution is unpredictable, so each case must be treated individually. In mild cases where symptoms do not progress or there are no worrying signs on X-rays, the most appropriate approach is to opt for a conservative approach and maintain close observation. Corticosteroids are the first therapeutic option to reduce the pressure caused by inflammation, and in some cases it may be necessary to resort to an external ventricular drainage (EVD) procedure to treat hydrocephalus.(29).

High suspicion and early aggressive intervention are the best tools to achieve optimal results in fulminant cerebellitis in children. In medical and surgical emergency situations, high-dose corticosteroids are presented as the mainstay of treatment, as they improve long-term outlook and reduce the duration of the disease, empirical antibiotic treatment is mandatory, as is the use of antivirals.(30) The use of complementary treatments with immunomodulators such as immunoglobulin infusion or plasmapheresis generates mixed opinions.(31) Thus, the purpose of this study is to carry out an analysis of post-infectious cerebellitis in the pediatric population.

Methodology

This article presents a case report with a descriptive, retrospective bibliographic review describing the disease sinoatrial nodal block. To collect information about the case, the clinical history was verified and analyzed. Regarding the writing, the Vancouver style was used to reference; for the description of the pathology, the structure was applied: definition of the pathology, pathophysiology, risk factors, diagnosis, prognosis, signs and

symptoms, consequences, nursing care plan and medical treatment. For the description of the pathology, we worked through the collection of articles extracted from recognized databases such as: Scopus, Porquest, Pubmed, Web of Science, lilacs. In English and Spanish published in the last 5 years.

A systematization of the clinical case information was carried out. The data was obtained according to the patient's clinical history (secondary database) describing: reason for consultation, current illness of the patient upon admission, diagnostic impression (IDX), personal history, family history, medications commonly used by the patient, physical examination, initial laboratory tests taken from the patient, therapeutic management plan, complementary tests, outcome (improvement, lack of response, or death).

In the last part, a discussion and synthesis of knowledge was carried out on the particularities that holistically affect the patient's outcome. Finally, the data obtained were compared with those from other investigations in order to structure the clinical case article.

Results

Case presentation

Patient aged 2 years and 11 months, son of elderly parents and a 40-year-old mother. Unplanned pregnancy, which proceeded without complications, born by cesarean section in a health home; crying at birth without signs of asphyxia, was hospitalized in the neonatology service for neonatal jaundice, after one day he was discharged without complications. In his neurological development, adequate head support is described, he did not have the crawling period, he sat up at 5 months of age, and began to walk at one year and two months of age, in addition, he speaks and understands at 2 years and 2 months.

The patient's relative (father) reports that approximately 24 hours ago the patient vomited on one occasion, it is unknown if it was preceded by nausea. After the condition, he presented ataxia when walking and postural instability. He was also irritable, which is why he went to the health center. Physical examination showed that the patient was asymmetrical, dyschronometric, had horizontal nystagmus, was trembling, was ataxic, hypotonic, and had a Babinsky star gait. Postural instability.

Laboratory tests: IGG for COVID 19 of 265.8 (positive) 19: 3.30. Leukocytes: 7,810, Neutrophils 45,200, Lymphocytes 45,700, Hemoglobin 12.5 g / dl. Hematocrit 35.10%. Platelets 398,000, Potassium 4.7. Chlorine 115. SGOT: 20, SGPT: 9.6. LDH: 214. Calcium 10.27, Creatinine: 0.25. Phosphorus: 6.16. PT: 13.4 PTT: 29.6. INR: 1.06. He reports a history of a cold 15 days ago. It is classified as an unspecified ataxic condition,

after carrying out complementary tests and due to a history of previous infections, the patient is diagnosed with acute post-infectious cerebellitis.

He received treatment with methylprednisolone 250 mg in 100 ml of saline solution over 2 hours for 2 days, metoclopramide 5 mg and paracetamol 150 mg orally after administration of methylprednisolone. 0.9% saline solution 1000 plus 10 ml of BA complex 10 ml/hour.

Treatment sent to medical discharge: prednisone in descent, 20 mg orally for 5 days, 10 mg for 5 days, 5 milligrams for 5 days, 2.5 mg for 5 days. Complex B 3 ml orally once a day for 1 month. undergoes physical rehabilitation for 3 months, in the first month he maintains a slight tremor, improves stability and after 3 months the patient is asymptomatic.

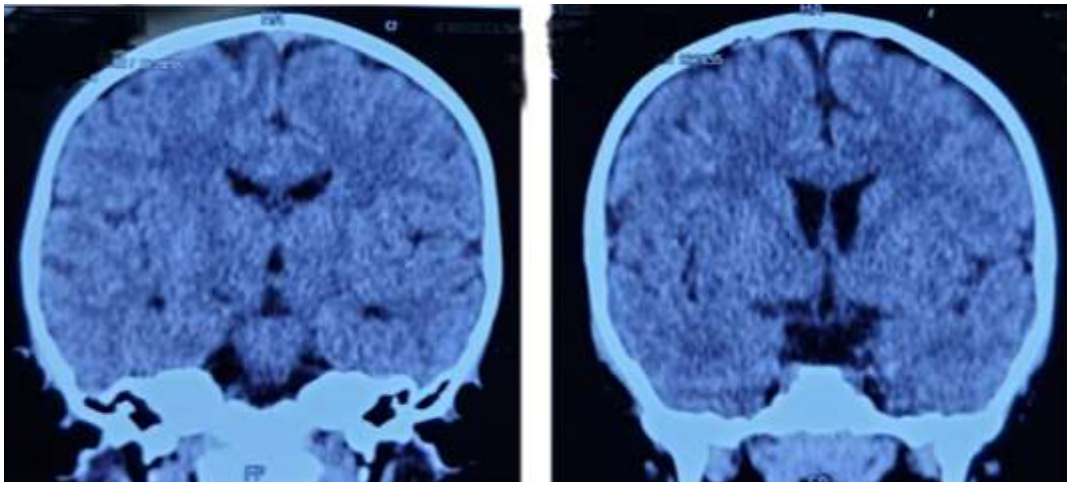


Figure 1. Simple computed axial tomography of the skull in coronal section

Report: In the posterior fossa, the IV ventricle, brain stem, peduncles, as well as the cerebellar hemispheres with normal characteristics are observed.

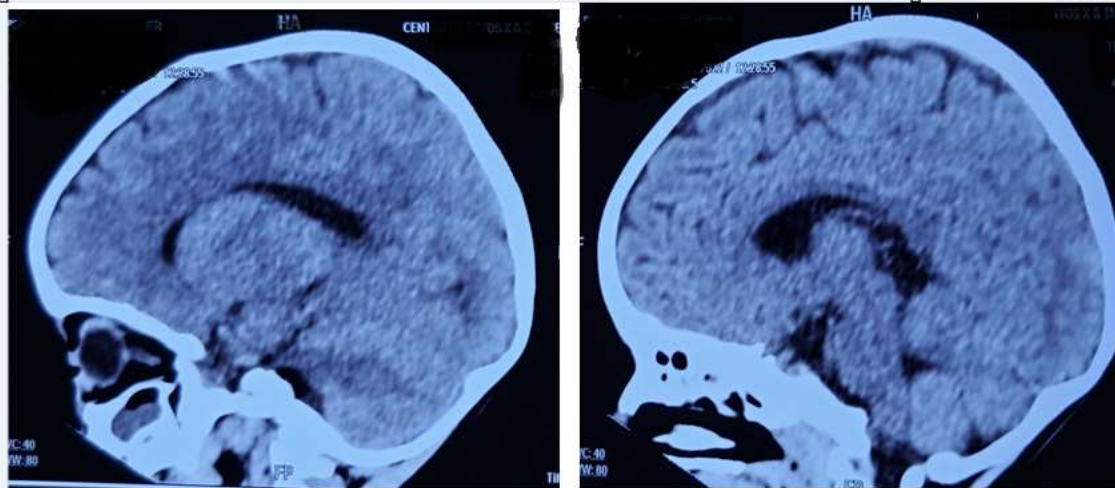


Figure 2. Simple computed axial tomography of the skull in sagittal section

Report: The basal cisterns do not show alterations. In the supratentorial region, the cerebral hemispheres have homogeneous density, there is no evidence of focal lesions of solid or cystic type or images of pathological calcifications.

The infra and supratentorial ventricular system is within normal limits. The cortical regions, as well as the midline structures, are normal. The study concludes that it is within normal limits (figure 1 and 2).

Discussion

Acute post-infectious cerebellitis is an important neurological condition in pediatric patients. According to Iñiguez et al, the most common initial symptoms include headache, vomiting and drowsiness, while ataxia, dysarthria and dysmetria were the most prevalent cerebellar signs. They also indicate that computed tomography images are usually normal (33). This is related to the clinical picture with which the patient was admitted, in addition to a simple tomography in which no relevant changes were seen in the acute phase.

Magnetic resonance imaging findings indicating acute inflammation have been required for the diagnosis of acute cerebellitis. However, the observation of inflammatory changes on magnetic resonance imaging is related to the time and imaging technique; our results do not show alterations in the imaging study performed. Therefore, clinical characteristics should also participate in the distinction between acute post-infectious cerebellar ataxia and acute cerebellitis (34). The latter, unlike acute post-infectious cerebellar ataxia, does not present with isolated ataxia but with multiple symptoms that Symptoms such as headache, nausea, and loss of lucidity are present. The microorganisms responsible for acute cerebellitis include varicella zoster virus, Epstein Barr virus, herpes simplex virus-

1, influenza, and respiratory syncytial virus, rotavirus. This fact may be related to the history of the common cold that with laboratory results gave positive COVID 19 that the patient had, 15 days before the cerebellitis.

A diagnosis of acute cerebellitis should be considered rather than acute post-infectious cerebellar ataxia when a child presents with ataxia along with headache and vomiting. Acute cerebellitis heals with sequelae in approximately one in three situations. The absence of deaths in our series suggests an early diagnosis and treatment with steroids may increase the chances of recovery (35). In this case, the patient had a favorable recovery and was asymptomatic after 3 months.

Regarding its etiology, the disease usually manifests during the development of an infection, as in the case of our patient, who experienced symptoms of a common cold 15 days ago. The clinical course of the disease is highly variable, ranging from a relatively benign self-limiting evolution to a fulminant presentation with life-threatening associated complications, such as compression of the posterior fossa, acute hydrocephalus and intracranial hypertension. It refers to a medical-surgical emergency that can be treated with high doses of corticosteroids and, in more critical situations, may require the placement of an external ventricular shunt and even decompressive surgery (36, 37). This was not the case for the patient.

Antimicrobial therapy should always be considered in these patients, as acute cerebellitis may be associated with various infectious agents. Ataxia may manifest as a symptom of both viral encephalitis and bacterial meningitis. Therefore, if lumbar puncture cannot be performed due to the risk of brain herniation, empirical antibiotic therapy is essential (38). Antibiotic therapy was not used in this case.

Post-infectious cerebellitis is often complicated by cerebellar ataxia, which is a clinical syndrome that is usually benign and transient, although life-threatening conditions may occur that may require immediate interventions. This clinical picture presented by our patient can impact individuals of all ages, however, it usually manifests mainly in children under six years of age. Although situations have been reported in older children and adolescents, it commonly manifests as a disorder that appears after an infection. Classic manifestations are gait disturbance and nystagmus, while associated symptoms may include dysarthria, vomiting, irritability or headache, seizures and even alterations in consciousness may occur (39).

The most common causes of acute onset ataxia are known to be drug ingestion, vaccination, viral or bacterial infections, malignant tumors, and lead, mercury, alcohol, or ethylene glycol poisoning, in our case it was post-infectious cerebellitis. Several causal events can be pointed out to explain the onset of acute cerebellar ataxia in our patient, including a possible trigger due to the HPV vaccination performed a couple of weeks

before the visible symptoms appeared. However, from the symptoms observed, laboratory analyses and serological tests detected the presence of IgG and IgM antibodies of the virus in the capsid, together with a positive result in the monospot test (35).

Several studies suggest an autoimmune process, where ataxia is usually an autobenign manifestation driven by an immune-mediated mechanism triggered by the viral infection, therapies targeting the immune system could be useful in the treatment of post-EBV cerebellar ataxia. Clinical improvement in some patients occurs only after the administration of IVIG treatment applied in patients who did not respond to high-dose steroid therapy (33). The patient gave a favorable response to corticosteroids without the need for IVIG administration results. Therefore, the resumption of the patient's daily activities and the relief of his parents' distress underline the importance of focusing on the aspect of improving medical care to raise well-being and quality of life, as has also been highlighted in other diseases. Immunological treatments are the most promising tools to achieve clinical remission and improve well-being in various diseases to raise quality of life.

In most of the situations described in the texts, the affected individuals are usually minors or adults, young, and the clinical presentation is generally solitary in the form of acute cerebellar syndrome, which may be associated with complaints of intense headache, stomach discomfort, and vomiting episodes. Fever and meningismus may or may not exist. In general, the clinical progression is usually mild and limited, with the presence of focal cerebellar problems in most situations, however, in cases where cerebellar involvement is diffuse, brain stem compression may occur, and acute cerebellitis may possibly occur, as well as an altered state of consciousness (42).

The clinical diagnosis of acute cerebellitis should be confirmed with an imaging test (CT or MRI) that also allows for the consideration of possible complications, such as hydrocephalic obstruction caused by compression of the fourth ventricle or protrusion of the cerebellar tonsils through the magnum occipital foramen (43).

From the histopathological point of view, perivascular inflammation, interstitial edema and demyelination are described, which translates into hyperintensity in long TR weightings of magnetic resonance, as observed in the T2 images of the patient presented.

a) Axial T2: symmetrical and diffuse cerebellar hyperintensity predominantly affecting the white matter. b) Sagittal T1 - increased volume of the cerebellum, with supratentorial obstructive hydrocephalus and loss of peribulbar liquor cisterns due to the low position of the cerebellar tonsils. a) Sagittal T1: increased volume of the cerebellum, with obliteration of the fluid spaces of the posterior fossa, compression of the IV ventricle and descending herniation of the cerebral tonsil. b) Coronal T2: diffuse cerebellar cortical hyperintensity and bilateral focal occipito-parietal cortical hyperintensities. c) Axial-hippocampal hypersignal acute infectious cerebellitis, a possible etiopathogenic role is

also attributed to the direct infection of the cerebellar parenchyma by the infectious agent, since it was not confirmed in our patient, as it was not a lumbar puncture (45).

Brain lesions resulting from acute CO poisoning are explained by a hypoxic-ischemic mechanism. The affinity of CO for the hemoglobin molecule is approximately 250 times greater than that of oxygen. Acute and intense exposure to CO causes a decrease in the ability of hemoglobin to transport oxygen, resulting in a lack of oxygen in the tissues. Within the central nervous system (CNS), neurons are the cells that require the most oxygen and glucose to function properly and are therefore more susceptible to a hypoxic-ischemic insult^{5,6}. In the second clinical case, this mechanism explains the predominantly cerebellar affectation. cortical and focal signal changes, occipito-parietal, cortical and in both hippocampi. On this occasion, a diffusion magnetic resonance study was not carried out, because the images were captured on a 0.5 Tesla equipment, which does not allow this type of analysis (46).

In this case report, the patient's age was 2 years 11 months. The median time between signs/symptoms at onset (fever, rash, viral infections) and onset of cerebellar symptoms was 6 days (range 0 to 30 days), similar to most reports describing a median time of 7 days between onset of VZV rash and hospital admission at 7 days. On admission, ataxia was the most frequent sign, with a wide gait. The neurological presentation was also often characterized by dysmetria and slurred speech (45).

These findings may have a clinical consequence as these patients could be the ones selected to receive steroid treatment, although there are currently no data in the literature to suggest this strategy. However, the possible usefulness of brain CT in the acute phase, to detect acute hydrocephalus, cerebellar edema or brain stem compression, the idea has already been raised, since some cases of CA, with hydrocephalus as the initial sign, have been mentioned in the literature. It is essential to highlight that in most places it is difficult to access a brain MRI on an urgent basis, however, in most cases there is a usual "waiting time" of approximately 7 days, while it is easy to perform a brain CT scan at the time of presentation of the patient in the emergency room. (48)

International guidelines do not clearly state whether immunocompetent children with cerebellitis should receive acyclovir and/or intravenous steroids. Similarly, most authors use steroids, mainly for more complicated cases, but it is not clear what is meant by more severe cases and what is the best steroid, dose, mode of administration and duration of therapy (48). Our patient was treated only with corticosteroids achieving a satisfactory result.

Since we showed that children with pathological brain CT or MRI on admission were more likely to have long-term neurological sequelae, this subset of patients might be the one to be selected for early and more aggressive treatment. AC/ACA is the most common

neurological complication of chickenpox, occurring in approximately 1/4000 cases of chickenpox in children. In the literature we found other reports describing VZV infection and neurological complications (encephalitis, meningitis, cerebellitis, polyradiculopathy, transverse myelitis) in adults and children. Other reports describe acute ataxia in children, but they analyzed a different pathogenesis, not just infectious and postinfectious acute cerebellitis (40).

Of note, this study included mostly children with no changes in mental status and normal imaging studies. In contrast, Hennes and colleagues detailed a group of 11 children (age range: 3 years to 14 years and 10 months), six of them with severe disease manifestation including mental status changes, MRI abnormalities, who were followed for a mean period of 4 years and 4 months. Neurological sequelae ranging from ataxia to mild tremor were reported in five children, and cognitive deficits (spatial visualization ability, language skills, and concentration) were found in six patients (40).

Conclusions

- An imaging examination confirms the clinical diagnosis of acute cerebellitis. MRI is more sensitive for detecting parenchymal changes than CT; however, in an urgent setting, MRI may not be performed, especially in cases of clinical instability of the patient. The role of cranial imaging in the follow-up of these patients is related to the detection of possible complications. In the most severe cases, with diffuse cerebellar involvement and enlarged cerebellum, there may be brain stem compression and active hydrocephalus, with altered state of consciousness and even death, if timely surgical intervention is not performed, i.e., ventricular bypass placement or surgical decompression of the posterior fossa.

Conflict of interest

The authors declare that there is no conflict of interest in relation to the submitted article.

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