Case Report

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Acute Disseminated Encephalomyelitis. Case Presentation

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ABSTRACT

Introduction: *Acute disseminated encephalomyelitis (ADEM) is a rare, immune-mediated demyelinating disorder of the central nervous system (CNS) with a predilection in early childhood. It is characterized by encephalopathy, multifocal neurological symptoms, and imaging evidence of central demyelination. It has an incidence of 0,4–0,6 per 100 000 children/year worldwide, with a predominance in males.*

Objective: *To describe the clinical case of an adolescent with acute disseminated encephalitis.*

Clinical Case: *A 12-year-old adolescent presented to the emergency department with an acute episode of severe headache, vomiting, unsteady gait, diplopia, and bradypsychia, preceded by a nonspecific fever. Physical examination revealed ataxic gait, dysmetria, and right cranial nerve paralysis of the third and sixth nerves. Fundoscopy revealed effacement of papillary margins with marked venous tortuosity and peripapillary hemorrhages. Brain MRI revealed edematous, hypointense lesions on T1-weighted images and hyperintense lesions on T2-weighted images located in the pons, cerebellar peduncle, part of the right cerebellar hemisphere, right occipital lobe, left parietal lobe, and the largest lesion in the right frontoparietal lobe. ADEM was diagnosed, and treatment with methylprednisolone at a dose of 30 mg/kg/day for 5 days was initiated, with symptom remission.*

Conclusion: *ADEM constitutes a diagnostic dilemma. It should always be considered in patients with CNS pathology and a history of immunizations and infections in the previous 2 weeks. Despite its dramatic clinical and radiological presentation, it shows good recovery with appropriate treatment.*

Keywords

Encephalomyelitis, Encephalopathy, Demyelination.

Introduction

Acute disseminated encephalomyelitis (ADEM) is a rare, immune-mediated demyelinating disorder of the central nervous system (CNS) with a predilection in early childhood. It is characterized by encephalopathy, multifocal neurological symptoms, and imaging evidence of central demyelination [1-3]. It has an incidence of 0,4 to 0,6 per 100 000 children/year worldwide, with a male predominance, a mean age between 5 and 8 years, and a higher frequency in winter and spring [4].

It is most often associated with infections, but not exclusively, especially viral infections [5]. Vaccines have also been implicated as a cause of ADEM, especially those for rabies, hepatitis B, polio, influenza, pertussis, measles, mumps, and rubella [6-8].

The disease is diagnosed based on the clinical presentation and neuroimaging findings. Treatment includes steroids, immunoglobulin, or mixed immunoglobulin plus steroid therapy in severe cases. Seizures may occur and are treated with antiepileptic drugs. Rehabilitation is important, and the prognosis is favorable, with good recovery. Some cases progress to multiple sclerosis [9,10].

The objective of this paper is to describe a clinical case of an adolescent with acute disseminated encephalomyelitis.

Clinical Case

A 12-year-old female adolescent with negative prenatal, natal, and postnatal history presented to the emergency department with an acute episode of severe headache, vomiting, unsteady gait, and diplopia, preceded by a nonspecific fever. Physical examination revealed ataxic gait, dysmetria, right cranial nerve paralysis of the third and sixth nerves, marked bradypsychia, and affective lability. Complementary tests (complete blood count, hemochemistry, abdominal ultrasound, and cytouria) were within normal limits. Cytochemical, Gram stain, and bacteriological studies of cerebrospinal fluid (CSF) were normal. The patient was admitted with intracranial hypertension syndrome, possibly a space-occupying lesion. A simple computed tomography (CT) scan and subsequent contrast-enhanced CT scan revealed no brain abnormalities. The patient was treated with cerebral edema control (mannitol 20%) at a dose of 0.5 g/kg/dose and showed significant clinical improvement, which was discontinued after 5 days of treatment.

At 72 hours, the patient presented with acute visual loss in the right eye (RE). Visual acuity examination revealed only light perception in the RE, and fundoscopy revealed blurring of the optic disc margins with notable venous tortuosity and peripapillary hemorrhages (RE). These findings, in conjunction with the sudden visual loss, were interpreted as right anterior optic neuritis. Magnetic resonance imaging (MRI) of the brain revealed edematous, hypointense lesions on T1 and hyperintense lesions on T2, located in the pons, right cerebellar peduncle, part of the cerebellar hemisphere adjacent to the peduncle, right occipital lobe, left parietal lobe, and the largest lesion in the right frontoparietal lobe (Figure 1).

The clinical picture and radiological findings were suggestive of ADEM. She was treated with methylprednisolone at a dose of 30 mg/kg/day for 5 days, followed by oral prednisone, and cerebral edema control was reintroduced for another 5 days. After the third dose of intravenous steroid, the patient regained right eye vision, and within a week, the neurological symptoms improved until they disappeared, leaving no sequelae. The patient is being followed up in the Neurology outpatient clinic.

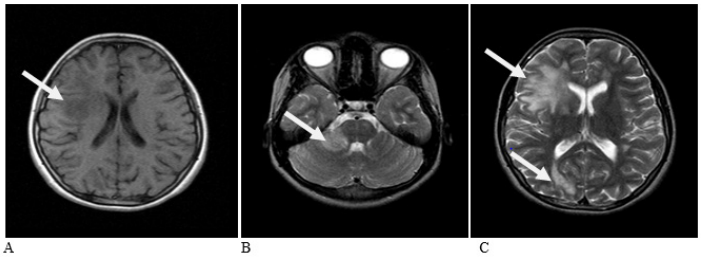

Figure 1: MRI. A. Hypointense lesions on T1-weighted images. B and C. Hyperintense lesions on T2-weighted images.

Discussion

Acute disseminated encephalomyelitis is a condition that can mimic other neurological diseases, such as multiple sclerosis, neurometabolic disorders, and other inflammatory processes of the CNS. It is triggered by an exaggerated and dysfunctional immune response in genetically susceptible individuals, typically presenting with encephalopathy and multifocal brain lesions [1,5,9].

The main etiology is a previous intestinal infection or respiratory illness (63% to 93%), of cryptogenic origin (30%) and post-vaccine origin (2,5% to 15%), associated with influenza, smallpox, rabies, mumps, and measles vaccines [7]. In the case presented, there is a history of a nonspecific fever one week before the acute illness, with no history of prior vaccination.

The pathogenesis consists of perivenular sleeves, which generate demyelination, associated with inflammatory infiltrates laden with myelin, macrophages, T and B lymphocytes, occasionally plasma cells, and granulocytes. Mimicry is likely involved in triggering this inflammatory cascade; Thus, it is believed that autoantigens are the target of this molecular adaptation, due to the similarities with viral sequences, which include the myelin basic protein (MBP), proteolytic protein (PPL) and myelin oligodendrocyte glycoprotein (MOG) [1,5,6].

The clinical presentation of ADEM is based on neurological symptoms, which develop 1 to 2 weeks after an infection or vaccination. In most cases, pyramidal signs are present, followed by hemiparesis, ataxia, cranial nerve abnormalities, seizures, language disorders, hemiparesthesia, and altered consciousness. Clinical diagnosis can be challenging due to the multiple diseases that can present with most of the aforementioned symptoms [4,9,11]. In our patient, altered consciousness, cranial nerve abnormalities, and cerebellar signs were present. These, combined with headache and vomiting, initially led us to consider a possible brain tumor, which is one of the differential diagnoses to consider. Cerebrospinal fluid (CSF) study is performed to rule out pathology of infectious origin, meningitis being the most frequent; in the EMAD CSF study, it presents what will be pleocytosis with definitions that vary between > 5 and > 20 Cells/ml, the predominant cell type is lymphocytes [6]. In the described case, the CSF was clear and without cells.

Brain computed tomography is of little use, with MRI being the study of choice for diagnosis. T2-weighted fluid-attenuated inversion recovery (T2-FLAIR) sequences show demyelinating lesions in the same stage, which are large, multiple, and asymmetric. They generally appear in the bilateral gray-white matter junction, cerebellum, brainstem, thalamus, in symmetric basal ganglia, and bilateral periventricular white matter. The appearance of multiple brain lesions requires extending the study to the spinal cord level [4,10]. Spinal MRI with or without contrast should be considered if there are sensory changes, limb weakness, and/or bowel and bladder dysfunction [9,12]. The patient underwent plain and contrast-enhanced CT, both of which were normal, with MRI

being the study that supported the diagnosis.

First-line treatment consists of intravenous methylprednisolone at a dose of 30 mg/kg/day (maximum 1000 mg/day) for 3 to 5 days, followed by a gradual taper of oral prednisone over 4 to 6 weeks. Early discontinuation of steroids (<3 weeks) may increase the risk of relapse. Steroid treatment requires close monitoring of blood pressure, electrolytes, and glucose, and the administration of gastric protection [5,8].

Intravenous immunoglobulin (IV IgG) is prescribed as second-line treatment for steroid-unresponsive ADEM at a total dose of 2 g/kg for 2 to 5 days [5,8]. In this patient, methylprednisolone was administered followed by oral prednisone, resulting in complete recovery; the use of IgG was not necessary.

Conclusion

Acute disseminated encephalomyelitis poses a diagnostic dilemma. It should always be considered in patients with central nervous system pathologies and a history of immunizations and infections in the last 2 weeks. Despite its dramatic clinical and radiological presentation, it shows good recovery with appropriate treatment.

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