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ACUTE DISSEMINATED ENCEPHALOMYELITIS AFTER LEPTOSPIROSIS INFECTION

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INTRODUCTION

Acute disseminated encephalomyelitis (ADEM) is a monophasic demyelinating disease of the central nervous system (CNS) and patients present with encephalopathy, focal neurological deficits and epileptic seizures (1,2). On magnetic resonance imaging (MRI) of the skull, the lesions are typically hyperintense on T2 and FLAIR sequences, in multiple numbers, bilateral and asymmetrical, with involvement of the central and subcortical white matter, in addition to being able to affect the thalamus, basal ganglia, cerebellum and brainstem (1).

ADEM probably stems from an autoimmune process triggered by infection or immunization and often no causal factor is identified (1,2,3). The organisms most related to this pathology are the viruses and there are few cases of post-infection ADEM described in the world literature (3,4,5,6).

In this case report, we will describe a patient with ADEM after leptospirosis infection.

CASE REPORT

A 42-year-old man, homeless, alcoholic, crack user, is brought to the hospital emergency room for a seizure episode. After the event, the patient presented with encephalopathy, myalgia, oliguria and fever. He was submitted to a Computed Tomography (CT) of the skull, which showed no abnormalities and laboratory tests with the presence of renal dysfunction, elevation of liver and muscle enzymes, and signs of systemic inflammation (Creatinine: 1.34 mg/dL, Total Creatine Kinase (CPK) : 51200 U/L, Urea: 29 mg/dL, Potassium: 3.2 mEq/L, Aspartate Aminotransferase: 880 U/L, Alanine Aminotransferase: 198 U/L, C-Reactive Protein (CRP): 240 mg/L, Leukocytes: 7300/mm^3 with 12% of rods, Total Hemoglobin (Hb): 10.7 g/dL, Platelets: 73,000/mm^3). The patient was maintained with supportive clinical treatment, but evolved with the need for renal replacement therapy.

After a week of hospitalization, he presented a new generalized tonic-clonic crisis, and then, it was decided to administer 20mg/kg of intravenous phenytoin followed by maintenance of the medication. He performed a new CT scan of the skull with the presence of hypodense areas in the subcortical white matter of the left parietal, bilateral occipital and right thalamocapsular lobes. He underwent CSF examination which showed no abnormalities. This was followed by extensive investigation and identification of positive IgM serology for Leptospirosis in serum enzyme immunoassay. He performed a cranial MRI that demonstrated diffuse T2/Flair hyperintense lesions in the supratentorial and infratentorial white matter, deep gray matter, and brainstem. Thus, with the hypothesis of Acute Disseminated Encephalomyelitis, it was decided to administer Methylprednisolone 1000mg a day intravenously for 5 consecutive days, in addition to antimicrobial treatment aimed at leptospirosis.

Four weeks after the end of the treatment, a new cranial MRI was performed, which showed significant resolution of the previous lesions. The patient was discharged from the hospital and referred to a municipal shelter, using phenytoin and without recurrence of epileptic seizures. He also recovered from encephalopathy, renal dysfunction, and normalization of inflammatory markers and muscle damage.

DISCUSSION

Leptospirosis is a zoonosis with a predominant geographical distribution in developing countries, in Brazil there are about 4,000 confirmed cases of the disease per year (7). The clinical presentation is variable with symptoms of fever, myalgia, arthralgia, vomiting and jaundice, however the involvement of the nervous system is

uncommon (8,9). In a study carried out by Panicker et al. (9) with forty patients diagnosed with leptospirosis and who initially presented with neurological symptoms, none had ADEM (9). The described manifestations were aseptic meningitis, myeloradiculopathy, myelopathy, Guillain-Barré syndrome, meningoencephalitis, intracerebral bleeding, cerebellar dysfunction, iridocyclitis and tremor with rigidity (9).

In this case report, we demonstrate the occurrence of ADEM after leptospirosis infection. The patient in question was exposed to the vector of the disease because he lived on the streets and did not have basic sanitation conditions. On hospital admission, the patient showed systemic signs and symptoms compatible with leptospirosis infection and presented signs of nervous system involvement, with epileptic seizures and encephalopathy, even though there was no alteration in the initial CT examination of the skull. During hospitalization, with diagnosis and targeted treatment, the patient presented complete clinical resolution.

There are few cases described in the literature on the occurrence of ADEM after leptospirosis. This report presents the relevance of alerting about the neurological involvement as an atypical manifestation of the disease. Low diagnostic suspicion can lead to therapeutic delay or even mistaken therapies, resulting in loss of care.

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