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Western equine encephalitis: a pediatric case report

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ABSTRACT

Western equine encephalitis (WEE) is a zoonotic disease caused by an RNA virus of the genus *Alphavirus*, and humans are accidental hosts. Although most patients do not develop symptoms upon infection, children are at higher risk for neurological involvement.

Here we describe the case of a previously healthy 13-year-old male patient who lived in an urban area in the province of Buenos Aires, Argentina, who was hospitalized and diagnosed with meningoencephalitis. Due to the torpid course of his condition and because none of the most frequent microorganisms were isolated, a test for IgG antibodies for WEE virus in blood and cerebrospinal fluid was requested; both samples were positive.

WEE virus is often an underdiagnosed cause of encephalitis and should be taken into consideration in both rural and urban areas.

Keywords: *encephalitis; viral encephalitis; equine encephalitis.*

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INTRODUCTION

Western equine encephalitis (WEE) is a zoonotic disease caused by an RNA virus of the genus *Alphavirus*. The transmission cycle includes birds that act as viral reservoir and *Culex tarsalis* mosquitoes, as vectors. Mosquitoes transmit the virus to equines or humans, which, given that they develop low levels of viral load, act as final hosts.[1–3]

The virus is present in much of the Americas, with virological or serological evidence of circulation in western Canada, the United States, Mexico, and regions of South America, such as Ecuador, Brazil, Uruguay, and Argentina.[4]

In Argentina, the National Ministry of Health issued an epidemiological alert in November 2023 due to an increase in the number of cases of WEE in equines; the first case in humans since 1988 was reported in December 2023.[5]

Although most cases are asymptomatic or mild, children are at increased risk for central nervous system (CNS) infections.[6]

Here we describe a clinical case of meningoencephalitis caused by WEE virus in a patient living in the Metropolitan Area of Buenos Aires, Argentina.

CASE REPORT

A previously healthy 13-year-old male patient who lived in the city of Avellaneda, province of Buenos Aires, consulted in January 2024 due to disorientation in time and place and episodes of aggressiveness associated with fever, headache, and vomiting for the past 72 hours. On physical examination, he had fever and was wakeful and reactive, although he had confusion, disorientation, and bradypsychia. He had dysmetria; his pupils were equal and reactive; and his strength, tone, myotatic reflexes, and cranial nerves were preserved. He was hospitalized with a presumptive diagnosis of meningoencephalitis.

The following tests were ordered:

- Laboratory tests showed positive C-reactive protein with a value of 13 mg/dL.
- Lumbar puncture: cloudy aspect, xanthochromia, cerebrospinal fluid (CSF) glucose level of 67 mg/dL (blood glucose level of 113 mg/dL), total proteins of 1.7 g/dL, lactic acid of 34.4 mg/dL, 540 elements/mm^3 (predominantly polymorphonuclear).
- The CSF culture, which developed *Microbacterium paraoxydans* (interpreted as a possible contaminant), the BioFire® FilmArray® meningitis/encephalitis multiplex polymerase chain reaction (PCR), and the viral PCR for herpes 1 and 2 (HSV) and enterovirus were negative.

  Both blood cultures were negative.
- The viral PCR in nasopharyngeal secretions for influenza virus, respiratory syncytial virus, and SARS-CoV-2 was negative.
- The computed tomography (CT) of the brain without contrast showed a tendency to collapse of the lateral ventricles, decreased amplitude of the Sylvian and basal (superior) cisterns with effacement of cerebral sulci.

A bacterial-herpetic etiology was considered probable, so treatment was started with ceftriaxone 100 mg/kg/day, acyclovir 30 mg/kg/day, and dexamethasone 0.6 mg/kg/day.

Seventy-two hours after hospitalization, the patient continued with fever, bradypsychia, and dysmetria, but without episodes of aggression. Due to his torpid course, a new lumbar puncture was performed, which reported that the CSF had an opalescent aspect, CSF glucose level of 71 mg/dL, protein level of 1 g/dL, lactic acid of 18.6 mg/dL, 185 elements/mm^3 (predominantly polymorphonuclear). The sample analysis was extended to include arbovirus detection and a culture for mycobacteria, which were negative, and a new PCR for HSV was also negative. A new CT of the brain with contrast was performed, which reported a new finding of diffuse enhancement of leptomeninges with contrast; an electroencephalogram (EEG) did not find epileptogenic focus or paroxysmal spells.

Based on a negative PCR for HSV, a normal EEG, and a CT of the brain not compatible with herpetic etiology, treatment with acyclovir was discontinued. The patient completed 10 days of treatment with ceftriaxone with a diagnosis of meningoencephalitis without microorganism rescue.

During the course of his condition, the patient remained with fever and bradypsychia. On day 7 of hospitalization, he developed hypertonia and decreased strength, predominantly on the right side. On day 12, he had altered sensorium, ocular myoclonus, anisocoria, ocular retroversion episodes, and short-term absences.

Since the patient presented a case of meningoencephalitis with a torpid course in the context of an outbreak of WEE in the province of Buenos Aires, IgG antibody detection tests for WEE virus in blood and CSF were requested and sent to a referral facility, Instituto Nacional de Enfermedades Virales Humanas Dr. Julio I. Maiztegui.

CSF cytochemistry was normal, but a positive result of IgM antibody for WEE in CSF and blood was obtained by immunocapture ELISA (MAC-ELISA).

A magnetic resonance imaging (MRI) showed alterations compatible with WEE (*Figure 1a*).

On day 22 of hospitalization, the patient was wakeful and lethargic; his bradypsychia improved; and he had decreased strength in the right lower and upper limbs and increased tone in the limbs, predominantly on the right side. It was decided to discharge him from the hospital and continue with outpatient follow-up.

During control visits, the patient persisted with bradypsychia and decreased strength in the right side of the body, although these symptoms improvement gradually. Mild chorea-like movements of the right upper limb were added a month after hospital discharge.

Two months after discharge, a control MRI showed slight improvement (*Figure 1b*).

The patient returned to school and continues currently, 3 months after discharge, receiving follow-up by the areas of motor kinesiology and educational psychology and has mild motor and cognitive sequelae.

Figure 1.a shows diffuse signal alteration, abnormally enhanced in T2 in the striata, internal and external capsules, medial aspect of thalamus, pes pedunculi, and dorsum of the pons, with signs of edema (red arrows). Non-specific findings compatible with WEE.

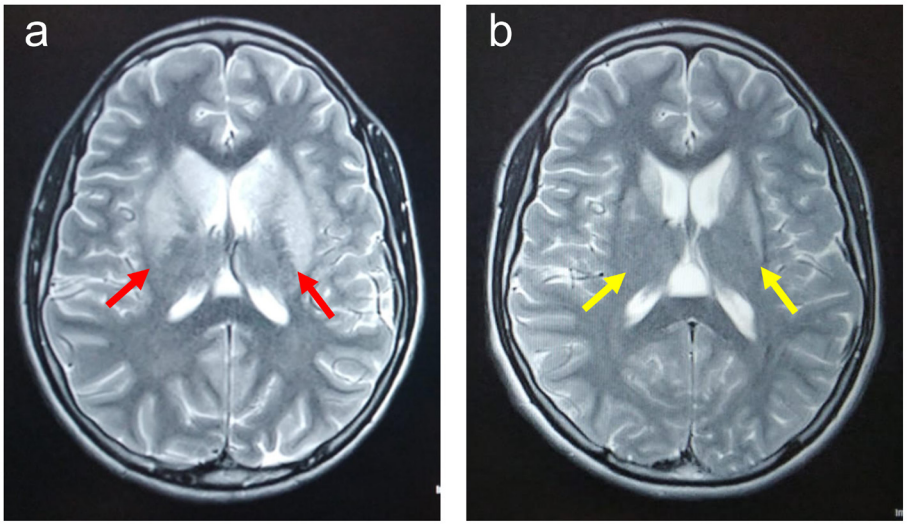
Figure 1.b shows an increased T2 signal intensity in the striata, with slightly reduced volume (yellow arrows), frankly lower compared to the previous examination shown in a.

DISCUSSION

Since the epidemiological alert issued on 11-26-2023 through 5-29-2024, 107 human cases of WEE have been reported to the National Health Surveillance System across 8 jurisdictions identified: the City of Buenos Aires and the provinces of Buenos Aires, Córdoba, Santa Fe, Entre Ríos, Santiago del Estero, La Pampa, and Río Negro. Buenos Aires, the jurisdiction where our patient lives, is the province with the highest number of confirmed cases since the beginning of the outbreak.[7]

The annual incidence of reported infections tends to be highly variable, as there are periods of inactivity and periods of outbreaks. This is related to the warm weather and rains that result in vector proliferation. Although the *Culex tarsalis* mosquito is primarily an ornithophilic vector during the spring-summer months, in summer it increases

Figure 1. Magnetic resonance imaging of the central nervous system; a) at 2 weeks; b) at 2 months



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its feeding on mammals. It was precisely in the summer months when the highest number of cases in humans and horses was reported,[7] coinciding the peak of encephalitis cases with the peak of feeding on mammals.[8]

Due to the large number of cases reported, the definition of suspected case was modified for those months and the need to have an epidemiological link was excluded.[9]

During the outbreak, confirmed cases were identified across all age groups, with a median age of 58 years (maximum: 81 years, minimum: 4 months). Sixty-one percent of the confirmed cases were in patients between 50 and 69 years of age; 83% were male. Most cases usually progress without symptoms, but the proportion of symptomatic and asymptomatic infections increases with age: 1:1 in children < 1 year, 58:1 in children aged 1 to 4 years, and > 1000:1 in children > 14 years.[6]

Symptomatic cases usually have an incubation period of 2 to 10 days, followed by headache and fever, sometimes accompanied by myalgia, abdominal pain, or irritability. As in the case presented here, it has been described that encephalitis usually has an abrupt onset and a rapidly progressive course.[10] The most frequent symptoms are generalized weakness and tremors.[11]

In the CSF cytochemistry of WEE, elevated proteins and pleocytosis are generally observed, initially with a predominance of neutrophils, but evolving to lymphocytes. The initial CSF sample of our patient presented these characteristics, but the shift to lymphocytic predominance was not evidenced in the subsequent lumbar punctures.

In relation to imaging tests, findings suggestive of this pathology are common in MRIs, such as inflammatory changes, vasculitis, or focal hemorrhages in the thalamus or basal ganglia.[12]

Although the case fatality rate is 3–4%, patients who recover from acute neurological disease may persist for years with fatigue, headache, or irritability. It is estimated that 15–30% of patients experience severe neurological sequelae. Neurological sequelae include decreased motor skills and gait disturbance, intellectual or learning disabilities, facial paralysis, speech problems, and parkinsonism.[10]

Our patient continues with gait disturbance, learning difficulties, and speech disturbances even 3 months after experiencing the acute clinical episode.

There is no specific treatment, only supportive therapy. It is not necessary to isolate the patient to prevent mosquito bites because, unlike other vector-borne diseases, the period of viremia is short.[13]

Here we described the case of an adolescent with a torpid course of WEE in the context of an outbreak of vector-borne transmission of the disease. It is necessary to consider WEE among the differential diagnoses of encephalitis in times of vector increase. ■

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