ACUTE CEREBELLITIS CAUSED BY HERPES SIMPLEX VIRUS

Lillian Gonçalves Campos¹, Roberto Rossato², Rodrigo Pires dos Santos³, Juliana Avila Duarte¹, Leonardo Vedolin¹,⁴

CASE PRESENTATION

A 29-year-old woman presented to the emergency department with gait imbalance and dysarthria. At admission, neurological examination revealed normal cognition, ataxia, dysarthria, dysmetria on both sides of the body, bilateral vertical nystagmus and loss of the lateral eye movement. Blood examination showed an increase in white blood cell (WBC) count and demonstrated erythrocyte sedimentation rate of 18 mm/h. Examination of cerebrospinal fluid (CSF) revealed protein concentration of 166 mg/dL, glucose concentration of 56 mg/dL, and pleocytosis. Serum glucose concentration was 126 mg/dL. The patient had no history of immunosuppression or any comorbidity and anti-HIV test was negative.

Neurological evaluation included a head computed tomography (CT) scan with normal findings. Magnetic resonance imaging (MRI) of the brain revealed increased bilateral signal intensity in the cerebellum on fluid-attenuated inversion recovery images (FLAIR)/T2, without contrast enhancement, suggesting an inflammatory process confined to the cerebellum (Figures 1 and 2). Furthermore, the cerebellar cortex appeared swollen, a finding consistent with diffuse cerebellitis. There were no changes in the brainstem. Initially, the possibility of bacterial rhombencephalitis caused by Listeria monocytogenes was considered, as it is the most common cause of rhombencephalitis.

After a few days of antibiotic therapy (ceftriaxone and ampicillin), polymerase chain reaction (PCR) test of the CSF was positive for herpes simplex virus 1/2 (HSV). Bacterial culture of CSF samples showed no growth, and the results of Gram staining of CSF were negative. Anti-Listeria antibody was also negative, and ampicillin was discontinued. CSF PCR analysis for other herpes viruses (varicella-zoster virus, Epstein-Barr virus, cytomegalovirus, and human herpesvirus types 6–8) and enteroviruses were also negative.

Treatment with acyclovir (50 mg/kg/day) for 21 days improved symptoms. One month after the first MRI, a significant reduction in imaging abnormalities was detected (Figure 3).

DISCUSSION

Acute cerebellitis remains an unclear clinical entity that has been associated with multiple etiologies including viruses (e.g., varicella zoster virus (VZV), measles, mumps, rubella, Epstein-Barr virus (EBV), cytomegalovirus, herpes simplex virus, parainfluenza virus, enterovirus and coxsackie virus) and bacteria (e.g., Salmonella typhi, Borrelia burgdorferi, Coxiella burnetii, Bordetella pertussis, and Mycoplasma pneumoniae)⁵. The pediatric population is more frequently involved, with fewer cases described in adults⁶. Acute cerebellitis is a neurological condition characterized by mild or high-grade fever, nystagmus, tremor, truncal ataxia, dysarthria, headache, and altered mental state⁷. Hydrocephalus and tonsillar herniation are most likely to be complications of the acute phase and may require neurosurgical intervention to prevent death⁸.

HSV encephalitis has a predilection for the temporal and frontal lobes, but occasionally affects the brainstem and less commonly only the cerebellum⁹.
When there is exclusive involvement of the cerebellum, bilateral diffuse hemispheric abnormalities represent the most common imaging presentation. Sometimes the changes can be restricted to one hemisphere, vermis or peduncles. MRI is more sensitive to assess the brainstem and cerebellum. There are few cases of HSV acute cerebellitis reported in the literature, in the absence of cerebral or brainstem lesions.

Differential diagnosis of acute cerebellitis includes infectious agents (e.g., viruses and bacteria), acute disseminated encephalomyelitis, Lhermitte-Duclos disease, diffusely infiltrating glioma or lymphoma, vasculitis, ischemic stroke, autoimmune diseases, paraneoplastic syndromes and drug-related inflammatory processes (e.g., drug intoxication). Imaging studies can be a very important tool in the distinction of these processes, as there are overlapping clinical and laboratory findings.

Conflicts of Interest
The authors declare no conflicts of interest.

REFERENCES