A 2-year-old Boy with Seizures

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A previously healthy 2-year-old boy with no history of seizures presented to our hospital in status epilepticus. Patient was apparently well 4 days before admission when he fell from his bicycle. He hit the left side of his head on the pavement; however, there was no external bleeding or loss of consciousness. Two days before admission, he had three episodes of non-bilious, non-bloody, projectile vomiting. Caregivers also reported unusual drowsiness and tactile fever. He had trouble finding the right words and had been using incorrect words in sentences.

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Editor’s note: Each month, this department features a discussion of an unusual diagnosis in genetics, radiology, or dermatology. A description and images are presented, followed by the diagnosis and an explanation of how the diagnosis was determined. As always, your comments are welcome. Please e-mail pedann@slackinc.com.
On the day of admission, caregivers reported that he started staring into space and did not respond to his mother’s calls or touch. This was followed by shaking movements of the right upper and lower extremity and loss of bladder control. No injury was sustained. The child was brought to the hospital where the seizure was controlled with an age-appropriate dose of lorazepam; the child was intubated and started on phenytoin drip. On arrival to the unit, the patient had another episode of seizure, which started from the right upper and lower extremity but progressed to a more generalized form. Seizure was controlled with another dose of lorazepam. Phenytoin levels were done subsequently, which were within range.

On admission, the patient was afebrile, intubated, sedated, and muscle relaxed. But later the same day, he developed a fever of 104.1°F. CBC showed a total white count of 8.6, with a normal differential. The patient was started on intravenous levetiracetam. The computerized tomography (CT) scan of head showed multiple, acute, left temporal intraparenchymal bleeds, with mild to moderate perilesional edema. There was no shift of midline structures or hydrocephalus. A magnetic resonance image (MRI) of the brain was done on the same day, which showed hemorrhagic contusions involving left anterior and posterior temporal cerebral lobes, including hippocampi gyrus, posterior insular, claustrum, and external capsules, with diffuse contusion involving left parietoccipital and temporal cerebral lobes.

A lumbar puncture was performed, which showed CSF red blood cells of 5,578 mm$^3$, total white cell count of 547 mm$^3$, with 86% lymphocytes, 6% neutrophils, and 8% monocytes, with a protein of 76 mg/dL, and glucose of 60 mg/dL. An EEG showed slowing on left, with multifocal epileptiform discharges seen on both sides, but which were more prominent on the left.

Child abuse was considered as a possible cause for the bleeding. PPD was negative. A urine toxicology screen was negative. Polymerase chain reaction (PCR) of the cerebrospinal fluid (CSF) revealed the diagnosis.

Figure 2. MRI of brain showing left temporal lobe hemorrhagic contusion.
HSV is notorious for causing serious, life-threatening encephalitis in children and adolescents, who account for 2% to 5% of all cases of encephalitis. Most of them are caused by HSV 1. With relative decline in the encephalitis, caused by measles, mumps, rubella, and varicella, the relative incidence of HSV encephalitis has increased, although the absolute numbers remain stable. Unlike some common viral encephalitis, HSV encephalitis is not seasonal. Typically, children with HSV encephalitis present with non-specific symptoms, such as fever, malaise, and irritability, which may gradually progress to significant neurological deficits. Altered mental status is the most commonly reported initial clinical presentation (97%), followed by fever (91%) and persistent seizures (71%).

The diagnosis of HSV encephalitis is based upon a constellation of clinical and laboratory findings. No single clinical or laboratory evaluation is confirmatory for HSV infection. The initial laboratory investigations should include a CSF analysis and, if required, brain imaging. Depending upon the presentation, it may also include an electroencephalogram. CSF analysis generally reveals pleocytosis with lymphocytic predominance in more than 90% of the cases. In 75% to 80% of the cases, CSF may be positive for red blood cells, which usually indicate hemorrhagic necrosis. Imaging may include CT scan and a MRI, which usually show changes, such as contrast enhancement, edema, mass effect, and hemorrhagic necrosis, mainly in the temporal area. Although necrotizing encephalitis as a complication of HSV infection has been reported in the past, radiographically apparent intracranial hemorrhage associated with HSV is comparatively rare. MRI is more sensitive than CT scan for the detection of HSV encephalitis.

In most cases, diagnosis can be established by PCR analysis of CSF, which is 98% sensitive and 94% specific, as compared with brain biopsy. However, false-negative PCR may be present early in course of the disease, which may delay treatment. Thus, antiviral therapy should begin if HSV encephalitis is suspected. Despite the widespread use of anti-viral therapy for HSV, nearly two-thirds of the survivors have residual neurological deficits, which could be irreversible brain necrotic change caused by HSV.

The dilemma that remained unsolved in our patient was about severity of disease, which was the reason for progression to necrotizing encephalitis. Immunoglobulin levels were performed to rule out Herpes Simplex Virus (HSV) Encephalitis.

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any immunoglobulin deficiency, which were normal. Interestingly, some animal studies have postulated that human neuronal cells possess intracellular Toll-like, receptor-mediated, innate immune protection against HSV infection. A deficiency of these receptors could theoretically make an individual more susceptible to HSV encephalitis. However, this is still an area of ongoing research.

CONCLUSION

Essentially, as in this patient, the correct and timely consideration of HSV remains important to provide the right treatment, as the differentials of HSV encephalitis include many treatable conditions. It is important for a clinician to keep a high index of suspicion while dealing with any patient with intracerebral bleed because HSV is a treatable cause of encephalitis, and a delay in treatment can lead to morbid outcomes.

REFERENCES