Rotavirus Cerebellitis?

Str—In a recent article, Lynch et al. [1] document the presence of rotavirus RNA in the CSF of 2 children with gastroenteritis and neurologic symptoms. We would like to present a related case of possible rotavirus cerebellitis.

Our patient was a previously healthy 3-year-old girl who presented with a 2-day history of vomiting and diarrhea and a temperature of 39.2°C. On the day of admission to the hospital, she abruptly stopped speaking. She began to have frequent screaming episodes that lasted 15 s, with back arching and stiffening legs and arms. The findings of an initial physical examination (which was performed between screaming episodes) were notable for diffuse hypotonia, inability to fix and follow, and failure to interact with her parents.

Laboratory evaluation included a complete blood count, the findings of which were normal. Analysis of electrolytes was notable for a mild metabolic acidosis, with a bicarbonate level of 16 mg/dL and an anion gap of 18. Results of a urine toxic screen were negative. Neurologic evaluation included a head CT, the findings of which appeared normal; lumbar puncture, which yielded a sample of CSF that normal values (WBC count, 2 cells/mm³; RBC count, 1170 cells/mm³; glucose, 53 mg/dL; and protein, 16 mg/dL); and an MRI and an electroencephalogram, both of which had normal findings. Results of cultures of urine/blood, urine, CSF, and stool were negative. Titters of antibodies to varicella zoster virus, Epstein-Barr virus, cytomegalovirus, and Mycoplasma species indicated no signs of acute infection. Tests of CSF for varicella zoster virus, Epstein-Barr virus, and herpes simplex virus had negative results, and PCR was negative for enterovirus. A stool sample was positive for rotavirus RNA.

One week after admission, the frequency of screaming episodes decreased, but the patient remained hypotonic and minimally interactive. She had no productive speech and had difficulty swallowing. Her diarrhea and vomiting completely resolved. Tests for Ala-δ-synthetase and porphobilinogen in urine had negative results, thus excluding the diagnosis of acute intermittent porphyria. A second sample of CSF was obtained and revealed the following values: WBC count, 17 cells/mm³ (72% lymphocytes; RBC count, 4 cells/mm³; glucose, 50 mg/dL; and protein, 27 mg/dL. A second MRI of the head was done, and T2-weighted images showed bitemporal hemispheric cerebellar enhancement, a finding consistent with diffuse cerebellitis. Empirical treatment with steroids (20 mg/kg for 5 days) was initiated but had no immediate effect. Over the subsequent 5 days, the patient developed intermittent esotropia and bradycardia with acute ventricular enlargement. She required placement of an external ventricular drain, which was removed when the infection subsided 2 weeks later.

Our patient has made a slow neurologic recovery. Five months after hospitalization, she is again interactive and has age-appropriate language skills. However, she still runs with a wide-base gait and has a moderate expressive aphasia. We believe that the neurologic findings can all be explained by the cerebellitis. The initial screaming episodes were consistent with “cerebellar fits” or seizures, which were first described by John Houghlings Jackson in 1871 [2]. Because of the depth of cerebellar structures, there are often no surface changes detectable on an electroencephalogram [3]. Diffuse cerebellar injury, regardless of its etiology, can cause hypotonia, irritability, and mutism.

But what caused the cerebellar inflammation? Results of all cultures and initial viral studies were negative, with the exception of the stool rotavirus test. CSF and stool samples subsequently tested positive for rotavirus by PCR. Reverse-transcription PCR genotyping revealed that the rotavirus isolated from the stool sample was the common P(8),G1 strain; the rotavirus isolated from the CSF sample was the common P(8),G1 strain; the rotavirus isolated from the stool sample was the common P(8),G1 strain; the rotavirus isolated from the stool sample was the common P(8),G1 strain.

We suggest that this patient’s cerebellar inflammation resulted from the rotavirus infection. Although rotavirus infection is common, it is rare for patients to have neurologic complications. The pathogenic mechanism that might explain this association remains speculative at this time and should be investigated further.

Acknowledgment

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References


Infectious Disease Pathology and the Autopsy

Str—I was surprised that Procop and Wilson [1], in their review of infectious disease pathology, made no mention of the autopsy. Their review purports to detail the role of the anatomic pathologist in the diagnosis of infectious diseases. However, for Procop and Wilson, the scope of anatomic pathology seems limited to surgical pathology and cytopath-
ology. I would like to point out that the autopsy has long been a cornerstone of anatomic pathology and has been frequently used for the diagnosis of infectious diseases. The autopsy has proven to be important in the recognition of emerging infectious diseases and in the description of the pathology of these conditions. In recent years, the autopsy has been critical in the recognition and description of AIDS, hantavirus pulmonary syndrome, and West Nile encephalitis [2–4].

An autopsy does not provide information that can be used directly for patient management in the same way as can information derived from a lung biopsy. However, information derived from an autopsy can guide management decisions for subsequent patients. Additionally, an autopsy can diagnose fatal communicable infections, such as tuberculosis [5], meningococemia [6], and plague [7], that may have a direct bearing on the clinical treatment of the deceased patient’s contacts. Certainly, the frequency of the hospital autopsy has declined substantially in recent years [8]. I worry that the neglect of the autopsy by pathologists might be contributing to its decline.

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References

Reply
Sir—The inference by Dr. Nolte [1], that our article on infectious disease pathology centered primarily on the use of these diagnostic techniques for the living, is correct. Perhaps a more exclusive title or a more inclusive text would have been a better choice on our part. Nevertheless, Dr. Nolte’s remarks afford me an opportunity to comment of the use of autopsy for the diagnosis of infectious diseases.

In the age of MRI and CT-guided biopsy of deep-seated lesions, some may feel the postmortem examination has little to offer. With this I contend. I vividly remember a patient during my residency training who developed an invasive pulmonary infection with the neurotropic fungus Ochroconis gallopava after lung transplantation. The fungus disseminated to the brain, and multiple nodules appeared in both lungs as well. Although treated aggressively with antifungal agents, the patient died. To the surprise of all, only a microscopic focus of fungal infection was apparent on dissection of the lungs; the remaining nodules consisted of an undiagnosed Epstein-Barr virus–related posttransplantation lymphoproliferative disorder. Although this example is extreme, it is not uncommon for the postmortem examination to reveal infections undiagnosed in the living patient. In addition, I wholeheartedly agree with Dr. Nolte regarding the usefulness of the autopsy for the diagnosis of communicable and emerging diseases.

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Kawasaki-like Syndrome: Abacavir Hypersensitivity?

Sir—Johnson et al. [1] described fever, rash, abdominal pain, conjunctivitis, and peripheral edema in 2 patients infected with HIV. The authors attributed these symptoms to a Kawasaki-like syndrome without considering the possibility that they were due to an abacavir hypersensitivity reaction, which can be life-threatening and is more severe when abacavir is reintroduced. There is much overlap in the signs and symptoms associated with these 2 clinical syndromes. Because the syndrome originally described by Kawasaki and colleagues appears to be extraordinarily rare in adults [2–4], these unusual clinical findings will be encountered more often in patients treated with abacavir who are experiencing a hypersensitivity reaction.

Reintroduction of abacavir, as described in the first case report [1], resulted in a syndrome of fever, rash, and abdominal pain. Abacavir was promptly and permanently discontinued. Although the patient in the second case report was not receiving antiretroviral therapy at the time of the event, inadvertent ingestion of abacavir after discontinuing it for reasons other than hypersensitivity has re-