Haemophilus influenzae infections of cerebrospinal fluid shunts

Report of two cases

STEPHEN J. LERMAN, M.D.

Departments of Pediatrics and Medical Microbiology, College of Medicine, University of Nebraska, Omaha, Nebraska

Two (1%) of 165 episodes of Haemophilus influenzae infection of the central nervous system occurred in patients with cerebrospinal fluid shunts. Both cases were caused by strains that could not be typed. The clinical presentation was similar to that of other forms of shunt infection, yet the pathogenesis may be similar to that of H. influenzae meningitis in children without shunts. Systemic antibiotic therapy, without shunt replacement or intraventricular antibiotic administration, may be more successful in shunt infections caused by H. influenzae than in those caused by other organisms.

KEY WORDS: cerebrospinal fluid shunt • Haemophilus influenzae • antibiotic therapy

Case Reports

Case 1

This 5-month-old girl with hydranencephaly was admitted to the University of Nebraska Medical Center with increasing head circumference and irritability. A ventriculoperitoneal (VP) shunt had been inserted at 1 month of age. During the month prior to admission, her head circumference increased 3 cm and, during the 2 weeks before admission, she was treated with penicillin for fever and cough.

When admitted, her temperature was 37.9°C, tympanic membranes were normal, and there was no neck stiffness. The white blood cell count was 31,900 cu mm, with 63% segmented and 25% band neutrophils. Serum sodium concentration was 129 mEq/liter, and a chest film showed no pneumonia. Blood culture grew ampicillin-sensitive H. influenzae that could not be typed. Shunt fluid was not aspirated for culture. No surgery was performed, nor was antibiotic therapy administered in view of the patient’s underlying condition. She became increasingly irritable, weak, and hypothermic. A blood culture obtained 3 days later again yielded H. influenzae, and she died on the following day. On postmortem examination, the fluid in the cranial cavity was turbid and yellow, and grew H. influenzae on culture.

Case 2

This 3-year-old boy with hydrocephalus due to congenital aqueductal stenosis was admitted to Children’s Memorial Hospital with fever. His current ventriculoatrial (VA) shunt had been inserted only 20 days before admission. He had had a slight fever for 3 days, and on the day of admission was vomiting and had a temperature of 40.5°C. However, he appeared alert, playful, and minimally toxic. The left tympanic mem-
brane had decreased mobility, and the superior posterior portion was red and bulging. There was no redness or tenderness over the mastoid area. His neck was supple. White blood cell count was 11,600/cu mm, with 67% segmented and 21% band neutrophils. Serum sodium concentration was 132 mEq/liter. Mastoid films showed cloudiness on the left.

Shunt aspiration yielded cloudy fluid with a protein concentration of 100 mg%, a glucose level of less than 5 mg%, and 410 white blood cells/cu mm, 90% of which were neutrophils. Gram-negative pleomorphic coccobacilli were present on smear. Shunt fluid and blood cultures grew ampicillin-sensitive non-typable H. influenzae. The ventricular end of the shunt was removed, a ventriculostomy placed, and systemic therapy with ampicillin initiated.

The patient's temperature was normal on the following day. Despite negative cultures, ventriculostomy fluid glucose levels remained low for the next 4 weeks. White blood cell counts initially decreased, and then increased markedly before gradually declining. On the 11th hospital day, he underwent bilateral myringotomy, and sterile serous fluid was found on both sides. A new VA shunt was inserted on the 13th hospital day. Ampicillin therapy was continued for a total of 1 month. He has had subsequent CSF shunt infections, but none due to H. influenzae.

Discussion

These two cases of CSF shunt infection represent 1% of 165 episodes of H. influenzae infection of the central nervous system (CNS) found during the 6-year period of surveillance between 1974 and 1979.

Children with CSF shunts appear to be at greater risk of H. influenzae infection than children in the general population. Schoenbaum, et al., reported on a series of 289 CSF shunts placed in Boston; of these, one shunt became infected with H. influenzae. In a study in Seattle, Shurtleff, et al., reported that, among 102 children with shunts, one developed H. influenzae infection. In a second study from Seattle, two instances of H. influenzae infection were reported in a group of 217 children.

Strains of H. influenzae involved in two previously reported CSF shunt infections were type b. The H. influenzae strains infecting our two patients were, however, not typable. Meningitis patients without CSF shunts had type b strains infecting 98% of cases, attesting to the unusual vulnerability of shunt patients to CNS infection with low-virulence organisms. In patients without shunts, non-typable H. influenzae infection occurred most commonly in the form of bacteremia alone, without CNS invasion, in patients with systemic underlying conditions.

One should consider the proportion of H. influenzae infections of CSF shunts that are caused by non-typable strains when recommending for such patients the vaccine that is being developed specifically against H. influenzae type b; such a vaccine will be ineffective against non-typable strains.

The pathogenesis of H. influenzae shunt infections has not been delineated, and the relative importance of the shunt, underlying hydrocephalus, and other host factors in increasing the risk of infection is not known. Bacteremia probably precedes CNS invasion in shunt patients, as it does in children without a foreign body in place. Although common in patients with infected VA shunts, bacteremia is distinctly unusual with infected VP shunts. In patients with H. influenzae-infected shunts, for whom the data were given, all three with VP shunts (Case 1 of this report, and two patients of Patriarca and Lauer*), as well as two with VA shunts (Case 2 of this report, and one patient of Shurtleff, et al.,) had bacteremia. The occurrence of H. influenzae infection with VP shunts, as noted in Case 1 and by others, and also in the instance of a lumboureteral shunt, indicates that retrograde infection from the blood stream through the shunt tubing is probably not important, as would be suggested if infections were found only with VA shunts. Furthermore, six of seven patients with adequate data (Case 1 of this report and five others)*, had onset of infection more than 30 days after the shunt operation, suggesting that the infecting organism was not introduced during the intraoperative period.

The clinical presentation of these two children with H. influenzae shunt infection more closely resembled that of shunt infections with other organisms than H. influenzae meningitis in normal children. They had nonspecific complaints such as low-grade fever, vomiting, and malaise, and they exhibited signs of increased intracranial pressure, suggesting that their shunts were malfunctioning. Neither patient had rapid deterioration, extreme toxicity, or a stiff neck. On the other hand, the clinical presentation of the two previously reported patients with type b infection was more consistent with H. influenzae meningitis. In addition, three of the four patients had otitis media, and all four had low CSF glucose levels, uncommon findings with the usual shunt infection.

Shunt replacement and intraventricular antibiotics may have lesser roles in the therapy of H. influenzae shunt infections than in other forms of shunt infection. Formulation of firm treatment recommendations, however, will require more extensive experience. The patient in Case 2 of this report, who was treated with systemic antibiotics and complete shunt replacement, was cured, but the response was slow. One patient of Patriarca and Lauer* was a treatment failure, despite systemic antibiotics, intraventricular antibiotics, and complete shunt replacement.

In a series of 20 patients with Gram-negative shunt infections, the only two patients cured with systemic antibiotic therapy alone or systemic antibiotic therapy plus incomplete shunt revision were the two patients with H. influenzae.* H. influenzae shunt infection was apparently eradicated in four patients by systemic and
Influenza shunt infections

intraventricular antibiotics without shunt removal. The contribution of the intraventricular antibiotics in two of the patients, however, may have been minimal, since they were being treated with systemic antibiotics that have greater activity against H. influenzae than the antibiotics being administered intraventricularly. One patient received intraventricular methicillin and systemic chloramphenicol, and the other received intraventricular cephalothin and systemic ampicillin.

References

1. Fraser DW, Geil CC, Feldman RA: Bacterial meningitis in Bernalillo County, New Mexico: a comparison with three other American populations. Am J Epidemiol 100:29-34, 1974


Address reprint requests to: Stephen J. Lerman, M.D., Pediatric Infectious Disease Unit, University of Nebraska Medical Center, 42nd and Dewey Avenue, Omaha, Nebraska 68105.