


Aseptic Meningitis after Spinal Anesthesia in an Infant

R. Blaine Easley, M.D.,* Reggie George, M.D.,* Dean Connors, M.D., Ph.D.,† Joseph D. Tobias, M.D.‡

OVER the past 10 years, spinal anesthesia has gained increased popularity as an alternative to general anesthesia in children. Recent reports have demonstrated spinal anesthesia as a safe alternative to general anesthesia in high-risk neonates and infants with a decreased incidence of apnea, bradycardia, and postoperative respiratory complications.1 Although the emphasis has been placed on the adverse effects of general anesthesia, it must be recognized that spinal anesthesia also can be associated with adverse effects. Documented neurologic sequelae of spinal anesthesia in adults include meningitis, arachnoiditis, and lumbar puncture headache.2 We report an episode of aseptic meningitis that temporally followed spinal anesthesia in an infant.

Case Report

A 2.5-month-old (former 30-week preterm), 3.04-kg male infant was admitted to the pediatric intensive care unit of the University of Missouri Children's Hospital after undergoing intubation and resuscitation at an outside facility. The patient underwent evaluation and treatment for respiratory failure and presumed sepsis. Initial cerebrospinal fluid (CSF) cultures showed a cell count of 20, with a leukocyte count of 3 and an erythrocyte count of 17, protein 102 mg/dl, glucose 79 g/dl, and a negative Gram stain. The patient steadily improved and was weaned from mechanical ventilatory support over a 5-day period. The nasopharyngeal and tracheal aspirates were positive for parainfluenza virus type III. The blood, urine, and CSF cultures were negative. During his stay in the pediatric intensive care unit, a right inguinal hernia was identified. By hospital day 14, the patient's condition was considerably improved, and arrangements were made for herniorrhaphy and circumcision.

On the day of surgery, the infant was fasted for 4, and a peripheral intravenous infusion was started. The patient was brought to the operating room, and routine monitors were placed. Three milligrams of hyperbaric tetracaine (in 5% dextrose in water) with an epinephrine wash was drawn from an ampule via a filter needle into a disposable sterile syringe. The infant was placed in the sitting position and pre-
pared in sterile fashion with an iodine solution. Excess solution was wiped away, and the area was dried. A 25-gauge, 1.5 inch spinal needle with stylet in place was insertedatraumatically under sterile conditions into the L3-L4 interspace. After removal of the stylet and return of CSF, the hyperbaric tetracaine was injected without resistance. The stylet was then replaced, and the needle was withdrawn with no leakage of blood, CSF, or anesthetic from the puncture site. The surgical procedure was completed without incident. The infant was observed in the postanesthesia care unit until the motor block had resolved and then returned to the inpatient ward.

Acetaminophen (15 mg/kg per dose) was administered to the patient every 4 h as needed for postoperative pain. On hospital day 18 (postoperative day 2), the patient became febrile to 39.4°C but was otherwise tolerating feeds and seemed well. Physical examination was unremarkable except for a small, palpable hematoma over the L3-L4 interspace. Chest radiograph was normal. Laboratory tests and cultures of blood, urine, and CSF (both viral and bacterial) were obtained. The CSF cell count was 8,150, with a leukocyte count of 3,650 and an erythrocyte count of 4,500. Differential analysis of the CSF leukocytes included 91% neutrophils, 3% lymphocytes, 5% monocytes, and 1% eosinophils. The CSF glucose level was 42 mg/dl, and the protein level was 218 mg/dl. Microscopic examination was significant for xanthochromia, polymorphonuclear cells, and no organisms by Gram stain. A diagnosis of meningitis was made, and intravenous antibiotic therapy with cefotaxime and vancomycin was instituted. The infant’s fever quickly resolved and he underwent repeat lumber puncture on hospital day 21 (postoperative day 5). CSF studies demonstrated a total cell count of 15,100, with a leukocyte count of 83 and an erythrocyte count of 15,017. The leukocyte count differential was 51% neutrophils, 32% lymphocytes, and 17% monocytes. Protein and glucose levels were 89 mg/dl and 50 mg/dl, respectively. Microscopic examination was significant for rare polymorphonuclear cells and no organisms. Based on the second lumbar puncture and the negative CSF cultures, a diagnosis of aseptic meningitis was made, and the antibiotics were discontinued. All cultures were finalized as having no growth for bacteria and viruses, and the child experienced no sequelae. The child was doing well at the 8-week follow-up examination.

Discussion

Aseptic and septic meningitis were once recognized as common complications of spinal anesthesia. As early as 1940, reports in the literature noted changes in cellular, protein, and glucose values in the CSF after lumbar puncture. These changes were the result of infectious complications or, more commonly, reactions to chemicals, detergents, and foreign materials (agents used to clean reusable spinal needles or impurities in spinal anesthetic agents) that had been introduced into the epidural or intrathecal space. Advances in technique, purity of chemical agents, and use of disposable equipment has resulted in a decrease in the incidence of aseptic meningitis and arachnoiditis after lumbar puncture or spinal anesthesia.

Septic (bacterial) and aseptic meningitis are rare but serious complications of spinal anesthesia. In adults, fever, headache, nuchal rigidity, and photophobia occurring 24–48 h after spinal anesthesia are suggestive of meningitis. Differentiating between septic and aseptic causes is based preliminarily on CSF characteristics and conclusively on the isolation of an organism by culture or detection on microscopic evaluation (i.e., Gram stain, acid fast stain). There are several reports in the literature that describe bacterial meningitis after spinal anesthesia in adults and, recently, in a 16-year-old boy.

The etiology of aseptic meningitis after spinal anesthesia in adults routinely has been attributed to chemical irritation from detergents and anesthetic agents, with only one reported case of a proven intercurrent viral infection. In 1960, Goldman and Sanford reported five cases of aseptic meningitis after spinal anesthesia in adults. The clinical course in these patients included fever (mean, 39.7°C) and meningitic symptoms (i.e., headache, photophobia, nuchal rigidity), which developed 5–48 h after spinal anesthesia. The opening pressure of the CSF was increased to 140–300 mmHg. CSF findings showed an increased leukocyte count of 2,600–27,000/mm³ with an average of 90% polymorphonuclear cells and normal glucose concentrations. Microscopic and culture results were negative. All patients showed rapid resolution of fever and meningitic symptoms in 48–72 h with no residual neurologic sequelae. The authors concluded that it was difficult to differentiate between bacterial and aseptic meningitis on the basis of lumbar puncture alone and suggested that this difficulty supported the practice of antimicrobial therapy in affected patients until cultures are negative. A sterilizing detergent was identified as the causative agent in their patients. Other anesthesiologists have made similar observations resulting in the increased use of disposable equipment, improved procedural techniques, and improved sterile processing of reusable equipment, which lead to the decline in incidence of post-spinal anesthesia aseptic meningitis during the late 1950s and early 1960s.

In the early 1970s, spinal anesthesia gained favor over general anesthesia in high-risk infants and children undergoing herniorrhaphy and orthopedic procedures. Complications of spinal anesthesia were noted, including fever, apnea, and bradycardia. However, review of the literature demonstrates no previous reports of aseptic meningitis in infants after spinal anesthesia. Although the need for a repeat lumbar puncture in a febrile infant after a surgical procedure is controversial, the pediatric literature and standard-of-care practices suggest that lumbar puncture along with other diagnostic tests (blood culture, complete blood count, chest radiograph, urinalysis, and urine culture) are indicated in high-risk infants aged less than 2 months who develop fever greater than 38.0°C (100.4°F).

Symptoms of meningitis and other serious illness are often difficult to assess in a neonate. Fever after surgery...
and anesthesia in a neonate, especially those aged less than 2 months, may indicate an infectious process either related or unrelated to the operative and anesthetic techniques. We report the first documented occurrence of aseptic meningitis in an infant after spinal anesthesia. Because of the infant's underlying status and recent life-threatening illness, it was not possible to definitely prove a causal relationship between the aseptic meningitis and the spinal anesthesia. Although no definitive treatment is necessary, the recognition of aseptic meningitis and the spinal anesthetic. Although no definitive treatment is necessary, the recognition of aseptic meningitis and the spinal anesthetic. Although no definitive treatment is necessary, the recognition of aseptic meningitis may avoid the prolonged and indiscriminate use of antibiotics if the fever is attributed to other sources.

References


Anesthesiology
1999; 91:307-10
© 1999 American Society of Anesthesiologists, Inc.
Uptington Williams & Wilkins, Inc.

INHALED nitric oxide (NO) is a selective pulmonary vasodilator with therapeutic importance in pulmonary hypertension and respiratory failure. NO increases intracellular cyclic guanosine monophosphate (cGMP), which stimulates smooth muscle relaxation. Phosphodiesterase type 5 (PDE5) is found in high concentrations in the lung and contributes substantially to cGMP degradation within vascular smooth muscle. Abrupt discontinuation of NO may be complicated by life-threatening events, and phosphodiesterase activity may play a role in this phenomenon. Sildenafil (Viagra; Pfizer Laboratories, New York, NY) is a potent and selective inhibitor of cGMP-specific PDE5, the predominant isoenzyme that hydrolyzes cGMP in the corpus cavernosum. We hypothesized that sildenafil may potentiate pulmonary vasodilation with NO or ameliorate the deleterious effects of abrupt discontinuation of NO by increasing intracellular and circulating cGMP, preventing rapid depletion of cGMP when the gas is withdrawn.

Sildenafil Ameliorates Effects of Inhaled Nitric Oxide Withdrawal

Andrew M. Atz, M.D.,* David L. Wessel, M.D.†

INHALED nitric oxide (NO) is a selective pulmonary vasodilator with therapeutic importance in pulmonary hypertension and respiratory failure. NO increases intracellular cyclic guanosine monophosphate (cGMP), which stimulates smooth muscle relaxation. Phosphodiesterase type 5 (PDE5) is found in high concentrations in the lung and contributes substantially to cGMP degradation within vascular smooth muscle. Abrupt discontinuation of NO may be complicated by life-threatening events, and phosphodiesterase activity may play a role in this phenomenon. Sildenafil (Viagra; Pfizer Laboratories, New York, NY) is a potent and selective inhibitor of cGMP-specific PDE5, the predominant isoenzyme that hydrolyzes cGMP in the corpus cavernosum. We hypothesized that sildenafil may potentiate pulmonary vasodilation with NO or ameliorate the deleterious effects of abrupt discontinuation of NO by increasing intracellular and circulating cGMP, preventing rapid depletion of cGMP when the gas is withdrawn.

Case 1

A 6-week-old 3.1-kg female infant with complex bilateral pulmonary vein obstruction underwent surgical resection of an obstructive mem-

* Assistant in Cardiology, Children’s Hospital; Instructor in Pediatrics, Harvard Medical School.
† Director, Cardiac Intensive Care Unit, Children’s Hospital; Associate Professor of Pediatrics (Anesthesia), Harvard Medical School.

From the Department of Cardiology, Children’s Hospital, and the Departments of Pediatrics and Anesthesia, Harvard Medical School, Boston, Massachusetts. Submitted for publication September 16, 1998. Accepted for publication February 18, 1999. Dr. Atz is supported by an award from the National Institutes of Child Health and Human Development (P40HD27805) and a grant from the United States Food and Drug Administration. Dr. Wessel is supported by grants from the United States Food and Drug Administration (FDR-001316-01) and the Research Endowment of Children’s Hospital (89430-02).

Address reprint requests to Dr. Wessel: Cardiac ICU Office, Children’s Hospital, 300 Longwood Avenue, Farley 653, Boston, Massachusetts 02115. Address electronic mail to: wessel@al.tch.harvard.edu

Key words: Congenital heart disease; cyclic guanosine monophosphate; phosphodiesterase inhibition; pulmonary hypertension; rebound pulmonary hypertension.