Anesthesiology
59:578–579, 1983

Enema-induced Hypocalcemia and Hyperphosphatemia Leading to Cardiac Arrest during Induction of Anesthesia in an Outpatient Surgery Center

JOHN C. REEDY, M.D.,* AND GERALD T. ZWIREN, M.D.†

Hypocalcemia and hyperphosphatemia have been reported secondary to the use of hypertonic phosphate enemas[1,2] and gavage[3] with resultant tetany and coma. Prolongation of the Q-T interval and complete heart block have occurred from hypocalcemia.[4] In this instance, in an outpatient surgical center, general anesthesia was induced in the presence of unsuspected hypocalcemia and hyperphosphatemia, and cardiac arrest followed.

REPORT OF A CASE

A 10-kg, 17-month-old boy with a history of chronic constipation was scheduled through the hospital's day surgery facility for rectal biopsy under anesthesia to rule out Hirschsprung's disease. He had no other significant medical history and had no previous exposure to anesthetic agents. Therapy for his chronic constipation consisted of Colace®, 3 ml po, bid, and a Fleet® pediatric enema every other day. On the day of surgery, a Fleet® pediatric enema was administered by the mother at 6:00 A.M. and another by the outpatient surgical nurse at 8:30 A.M. Preoperative evaluation by a staff anesthesiologist was unremarkable, and atropine, 0.15 mg, was given im. No unusual behavior was observed by either the parents or the operating room staff, and vital signs remained normal. The child was visibly upset and crying on arrival in the operating room. Efforts to calm the child while monitors were applied were unsuccessful. Induction commenced with inhalation of nitrous oxide 60% and halothane in increasing increments to 2% inspired concentration. Normal sinus rhythm became nodal but returned to sinus after stimulation by insertion of an iv catheter. Blood pressure was 76/40 mmHg. Following administration of succinylcholine, 10 mg, iv, intubation of the trachea easily was accomplished. Because of a pronounced leak, the endotracheal tube was removed and a larger tube selected. Following a repeat dose of succylcholine, 10 mg, iv, with atropine 0.1 mg, the trachea was reintubated. Sinus slowing of the heart rate from 140 to 100 bpm and widening of the QRS complex was observed. Arterial blood pressure remained 70/40 mmHg. Because of further slowing of the heart rate, nitrous oxide, and halothane were discontinued and ventilation was controlled with 100% oxygen. Color remained pink; however, the blood pressure and pulse became unobtainable. Electrical activity on the EKG revealed a profound bradycardia of approximately 40 bpm, with wide QRS complexes and tall, peaked T-waves. Epinephrine, 6 μg, was given iv, no response was evident, and external cardiac massage was initiated as electrical asystole developed. Epinephrine 100 μg was given, followed by sodium bicarbonate 8 mEq iv, but again there was no response. A second administration of epinephrine 100 μg, and sodium bicarbonate, 8 mEq iv, resulted in almost immediate return of electrical activity, however, with a protracted QRS complex and tall, peaked, T-waves. Calcium gluconate 200 mg was given and the QRS returned to normal. Arterial blood pressure was 100/70 mmHg and heart rate was 180 bpm. pH_a was 7.25, Pa_{CO_2} 36 mmHg, Pa_{O_2} 236 mmHg, and base excess -11 mEq·l^{-1}. Serum sodium and potassium concentrations were 130 mEq·l^{-1} and 9.4 mEq·l^{-1}, respectively, with a trace of hemolysis. When the report of the elevated serum potassium level was received, a repeat sample was obtained, which was reported as 5.8 mEq·l^{-1}. In addition, calcium 6.7 mg/dl (normal 10–12 mg/dl), and phosphorus 12.5 mg/dl (normal 4–6 mg/dl) serum concentrations were reported.

Within minutes of the resuscitation, the patient awoke and was resisting vigorously. He was taken to the recovery room, where extubation of the trachea was accomplished without problems; he was responsive and calling for his mother. After admission to the intensive care unit for observation, further increments of calcium and sodium bicarbonate were administered. During the next 8 h, the calcium and phosphorus levels returned to normal. Tetany never was observed in the operating room or in the intensive care unit. Serum levels of creatine phosphokinase, 135,000 IU/l, serum glutamic oxyltransferase, 2,300 IU/l, and lactic dehydrogenase, 5,400 IU/l, were documented. Fever to 38.5°C developed for approximately 8 h, which resolved without treatment other than hydration. Malignant hyperpyrexia was considered as a cause for these symptoms, however, cardiac arrest and resuscitation could be associated with such enzyme changes. Follow up enzyme studies 8 months later revealed creatine phosphokinase, 8,416 IU/l, serum glutamic oxyltransferance, 400 IU/l, and lactic dehydrogenase, 405 IU/l. At 1 yr after the incident, the patient was seen for evaluation by the department of genetics to see if any diagnosable varient of malignant hyperthermia existed. Creatine phosphokinase at that time was 3,150 IU/l and platelet exposure to halothane concentrations up to 2.4% produced results well within the limits of normal, indicating that the patient probably does not have malignant hyperthermia.

DISCUSSION

This patient demonstrated what has been reported before, that the Fleet® pediatric enema may cause hypo-

* Assistant Professor of Anesthesiology, Emory University School of Medicine, and Attending Anesthesiologist, Henrietta Egleston Hospital for Children, Inc.

† Clinical Assistant Professor of Surgery, Emory University School of Medicine, and Attending Surgeon, Henrietta Egleston Hospital for Children, Inc.

Received from the Departments of Anesthesiology and Surgery, Henrietta Egleston Hospital for Children, Inc., and Emory University School of Medicine, 1405 Clifton Road, N.E., Atlanta, Georgia 30322. Accepted for publication May 16, 1983.

Address reprint requests: Dr. Reedy: Department of Anesthesia, Henrietta Engleston Hospital for Children, Inc., 1405 Clifton Road, N.E., Atlanta, Georgia 30322.

Key words: Anesthesia: pediatric; outpatient. Complications: arrest, cardiac. Neuromuscular relaxants: succinylcholine. Hypertonic phosphate enemas. Ions: calcium, phosphate.

calcemia and hyperphosphatemia.[1,2] Persistent elevations of creatine phosphokinase indicate that he may have some form of muscular dystrophy. The fact that he sustained cardiac arrest on induction of anesthesia could be related to either of these two states as well as the use of succinylcholine, which has been documented to produce sinus arrest after repeat administration in children and adults.[5,6]

Whatever the cause or causes of cardiac arrest in this patient, it is of concern that he was being brought to surgery as an outpatient, a category reserved for patients only in the best of health. His electrolyte inbalance was unsuspected, as was his possible muscular dystrophy. In retrospect, this patient does not fit the ASA Class I patient group. The chronic use of any medication, even enemas, should have made this patient inelegible for outpatient surgery. If admitted the night before surgery, he would not necessarily have had the benefit of electrolyte screening. It should stand as encouragement to anesthesiologists to have a high index of suspicion regarding the chronic use of any drug or substance, even enemas, and to check for abnormalities accordingly.

References

1. Sotos JF, Cutler EA, Finkel MA, Doody D: Hypocalcemic coma following two pediatric phosphate enemas. Pediatrics 60:305–307, 1977
2. Davis RF, Eichner JB, Bleyer WA, Okamoto G: Hypocalcemia, hyperphosphatemia, and dehydration following a single hypertonic phosphate enema. J Pediatrics 90:484–485, 1977
3. Bachrach L, Correa A, Levin R, Grossman M: Iron poisoning: Complications of hypertonic phosphate lavage therapy. J Pediatrics 94:147–149, 1979
4. Griffin HJ: Neonatal hypocalcemia and complete heart block. Am J Dis Child 110:672–675, 1966
5. Craythorne NWB, Turndorf H, Dripps RD: Changes in pulse rate and rhythm associated with the use of succinylcholine in anesthetized children. ANESTHESIOLOGY 21:465–470, 1960
6. Williams CH, Deutch S, Linde HW, Bullough JW, Dripps RD: Effects of intravenously administered succinylcholine on cardiac rate, rhythm, and arterial blood pressure in anesthetized man. ANESTHESIOLOGY 22:947–954, 1961

Anesthesiology
59:579–583, 1983

The Antiemetic Effect of Droperidol Following Outpatient Strabismus Surgery in Children

MICHAEL D. ABRAMOWITZ, M.B., B.CH.,* TAE H. OH, M.D.,* BURTON S. EPSTEIN, M.D.,†
URS E. RUTTIMANN, PH.D.,‡ DAVID S. FRIENDLY, M.D.†

Although strabismus surgery is performed readily in an ambulatory setting, persistent vomiting following anesthesia may delay the patient's discharge from the hospital or may even lead to hospitalization.[1] In our institution, as well as others,[2,3] patients undergoing general anesthesia for strabismus surgery have a higher incidence of postoperative vomiting than those receiving the same anesthesia for other types of ambulatory surgical procedures. In addition to discomfort to patients, vomiting often presents many risks, such as dehydration, electrolyte imbalance, tracheal aspiration, and wound contamination particularly for those with no eye dressing placed after surgery.[4]

In a preliminary investigation we studied the antiemetic effects of droperidol in children undergoing strabismus surgery.[5] We found that approximately 80% of the children not receiving any antiemetic vomited after strabismus surgery and that droperidol 50 mcg/kg iv administered during the surgery was not totally satisfactory in reducing the frequency and severity of vomiting. We speculated that better results might have been produced if a higher dose had been used. In this study, we, therefore, extended our clinical experiments to investigate the effect of higher doses of droperidol.

Methods

Fifty-two ASA physical status I patients (ranging from 2 to 13 yr of age) scheduled for correction of strabismus were studied. On admission to our ambulatory surgical center, parents were questioned for a history of motion sickness and vomiting after previous ophthalmic surgery in their children. Patients who had received any drug or who had viral or bacterial infections within the 2 weeks

* Associate Professor.
† Professor and Chairman.
‡ Staff Scientist, NIDR, National Institutes of Health.

Received from the Departments of Anesthesiology, Ophthalmology and Child Health and Development, Children's Hospital National Medical Center, George Washington University School of Medicine, Washington, D. C. Accepted for publication May 16, 1983. Presented in part at the Annual Meeting of the American Society of Anesthesiologists, 1981.

Address reprint requests to Dr. Abramowitz: Department of Anesthesiology, Children's Hospital, National Medical Center, 111 Michigan Avenue, N.W., Washington, D. C. 20010.

Key words: Anesthesia: pediatric, ophthalmologic, outpatient; Vomiting: incidence, droperidol.

Supported in part by a grant from Janssen Pharmaceutica, Inc.