

CLINICAL REPORTS

Ronald D. Miller, M.D., Editor

Anesthesiology
63:526-527, 1985

Anesthetic Management of a Patient with Hypokalemic Familial Periodic Paralysis for Coronary Artery Bypass Surgery

JAMES E. ROLLMAN, M.D.,* AND CHARLES M. DICKSON, M.D.†

The perioperative course and anesthetic management of a patient with undiagnosed hypokalemic familial periodic paralysis (HFPP) have been described.^{1,2} There are no reports of the anesthetic management of patients with HFPP undergoing cardiopulmonary bypass. We report such a patient in whom hypokalemia developed during the rewarming phase of coronary artery bypass surgery. He experienced truncal paralysis postoperatively but had none of the characteristic electrocardiographic or rhythm changes associated with hypokalemia develop, despite a serum potassium level as low as 1.2 mEq/l.

REPORT OF A CASE

A 39-year-old man with a history of myocardial infarction 6 months ago and increasingly frequent episodes of angina underwent treadmill exercise testing, during which he experienced substernal chest pain and was noted to have a 2-3 mm S-T-segment depression in leads V3-V5. Subsequent angiography showed three-vessel coronary artery disease, and the patient was scheduled for coronary artery bypass surgery.

Significant in his medical history was the diagnosis of hypokalemic familial periodic paralysis. Three episodes of paralysis during childhood required mechanical ventilation and tracheostomy. His last episode of paralysis was 8 years before his admission. He stated that he had been able to abort recent episodes of paralysis by taking oral potassium when the prodromal symptoms of tingling in his hands and feet occurred. Preoperative serum potassium was 3.5 mEq/l.

He was premedicated with diazepam 15 mg by mouth and morphine 12 mg im. Anesthesia was induced with diazepam 40 mg and fentanyl 500 µg iv. Pancuronium 10 mg iv was used to facilitate endotracheal intubation. This dose of pancuronium resulted in abolition of motor response to a tetanic stimulation (50 Hz). Anesthesia was maintained with additional iv doses of fentanyl and inhalation of nitrous oxide and halothane. Arterial blood gases and pH obtained after induction of anesthesia were normal. Intravenous fluids consisted of lactated Ringer's solution. Initial intraoperative serum potassium measured prebypass was 3.5 mEq/l. The pump prime was composed of 1,800 ml of lactated Ringer's solution, to which mannitol 70 g and sodium bicarbonate 50 mEq were added. During cardiopulmonary bypass, which lasted 73

min, hypothermia (30° C) and 250 ml of cardioplegic solution containing (5%) glucose, insulin (40 units/l) and a total of 10 mEq of potassium were used. As rewarming was initiated, serum potassium was 2.5 mEq/l; and 40 mEq of potassium was added to 1 l lactated Ringer's solution to be infused over 1 h. A mild metabolic acidosis (BE—4.5 mEq/l) was noted, and sodium bicarbonate 50 mEq was administered. Before weaning from cardiopulmonary bypass, calcium chloride 500 mg was given as the heart appeared to contract sluggishly. Two units of whole blood were infused iv during closure of the chest.

The trachea remained intubated, and no attempt was made to reverse any of the anesthetic drugs. Upon transfer to the recovery room, he could open his eyes but did not move his arms or legs. Serum potassium in the recovery room was 1.2 mEq/l, despite the iv potassium given intraoperatively. Arterial blood gases and pH were now normal. Sinus tachycardia of 110-120 beats/min was present with neither flattening of the T-waves nor the presence of U-waves. Propanolol 3 mg was given iv to control the tachycardia. Transcutaneous electrical stimulation of the ulnar and peroneal nerves produced no motor response, but stimulation of the facial nerve showed a normal train-of-four response indicative of no residual relaxant-induced paralysis. Four hours postoperatively the patient was alert and responded by affirmatively nodding his head when asked if he was having a paralytic episode. Despite receiving potassium chloride 100 mEq in divided doses via a central venous catheter and 40 mEq via a peripheral iv line, the serum potassium was only 2.1 mEq/l.

Six hours postoperatively he was beginning to move his fingers and arms but was still unable to move his legs. After receiving an additional 40 mEq of potassium over 2 h, serum potassium was 3.0 mEq/l. No further potassium was given, and 9 h later he was able to move his arms and legs. Serum potassium was then 4.4 mEq/l.

The trachea was extubated without difficulty the next morning, at which time his serum potassium was 3.9 mEq/l. The remainder of his course was uneventful, and he was discharged without experiencing further episodes of weakness.

DISCUSSION

Familial periodic paralysis is characterized as hypokalemic, hyperkalemic, or normokalemic.³ Hypokalemic familial periodic paralysis (HFPP) is a rare genetic disease thought to be transmitted as an autosomal dominant. Attacks of flaccid paralysis can be brought on by stress, cold, infections, and high-carbohydrate meals. Paralysis is generally incomplete, involving the arms, legs, or trunk but often sparing muscles supplied by the cranial nerves and diaphragm. This was borne out in our patient who had peripheral weakness at a time when electrical stimulation of the facial nerve revealed motor function to be present.

* Clinical Assistant Staff Physician.

† Associate Staff Physician.

Received from the Department of Anesthesia, Hartford Hospital, Hartford, Connecticut 06106. Accepted for publication June 4, 1985.

Address reprint requests to Dr. Rollman.

Key words: Anesthesia: cardiac. Neuromuscular junction: familial periodic paralysis.

The exact mechanism for the decrease is not known but is felt to be associated with an abnormal uptake of potassium by muscle cells, which alters membrane potential and renders skeletal muscles inexcitable.³ Potassium is not lost in the urine or stool.⁴ Metabolic changes and medications that cause a reduction in serum potassium appear to precipitate attacks, while those that produce a rise in serum potassium may abort an attack. Acetazolamide, which induces metabolic acidosis and causes an elevation of serum potassium, has been chronically used in patients with HFPP. Administration of halothane, nitrous oxide, and succinylcholine for appendectomy in a patient receiving acetazolamide was, nevertheless, followed by hypokalemic paralysis.⁵

Guidelines for the anesthetic management of these patients have been suggested.^{4,6} Electrolyte values must be measured preoperatively and supplemental potassium given if needed. Since many patients report an increase in weakness following a large carbohydrate meal, patients should have a light supper the evening before surgery. The amount of glucose infused should be kept at a minimum, as glucose may promote the intracellular movement of potassium. Temperature should be monitored and normothermia maintained during surgery.

The role of muscle relaxants is controversial. In 21 anesthetics administered to members of one diseased family, three hypokalemic paralytic episodes occurred after the only three operations during which muscle relaxants were used.¹ Since none were life threatening, this author recommended that muscle relaxants be used sparingly, while others recommend complete avoidance.¹ Electrocardiographic signs of hypokalemia are reported to be even more dramatic than would be expected from similar levels in people without HFPP.¹ Both oscilloscopic monitoring and paper recording of the electrocardiogram are essential.

Our case demonstrates the unique problems encountered when a patient with HPFF undergoes a major surgical procedure in which hypothermia, nondepolarizing muscle relaxants, and cardioplegic solutions are considered necessary adjuncts to the surgical procedure.

In our specific case we were reluctant to change the composition of our cardioplegia, *i.e.*, delete glucose or insulin, as we felt that postoperative paralysis was treated

easily with mechanical ventilation, but myocardial damage was an obviously more severe complication. We omitted glucose from all intraoperative and postoperative iv fluids as has been recommended. In addition, we measured serum potassium levels at approximately hourly intervals during surgery. Based upon our experience with a precipitous fall in serum potassium after bypass, we would begin supplementation before bypass, provided urine output is adequate.

Our decision to use muscle relaxants, contrary to previous recommendations, was based upon the belief that facilitation of the surgical procedure was most important and the knowledge that most postcoronary-bypass patients require some period of mechanical ventilation postoperatively. Our use of bicarbonate in the postbypass period probably was unnecessary. Indeed, a mild acidosis may have helped treat the symptoms of HFPP in this patient.

Finally, contrary to what has been reported, our patient had none of the electrocardiographic changes associated with hypokalemia, develop despite a serum potassium as low as 1.2 mEq/l.

In summary, we describe a patient with HFPP who underwent successful coronary artery bypass surgery with cardiopulmonary bypass. Although he required mechanical ventilation postoperatively because of prolonged hypokalemic paralysis, he suffered no serious cardiac dysrhythmias related to his disease and no permanent sequelae.

REFERENCES

1. Melnick B, Chang J, Larson C, Bedger R: Hypokalemic familial periodic paralysis. *ANESTHESIOLOGY* 58:263-265, 1983
2. Bashford AC: Case report: Anesthesia in familial hypokalemic periodic paralysis. *Anesth Intensive Care* 5:74-75, 1977
3. Gibbs PS, Kim KC: Skin and musculoskeletal disease, *Anesthesia and Co-existing Disease*. Edited by Stoelting R, Dierdorf S. Philadelphia, WB Saunders, 1983, pp 589-591
4. Miller J, Lee C: Muscle diseases, *Anesthesia and Uncommon Diseases; Pathophysiologic and Clinical Correlations*. Edited by Katz J, Benumof J, Kadis L. Philadelphia, WB Saunders, 1981, pp 554-557
5. Silar JN, Discavage WJ: Anesthetic management of hypokalemic periodic paralysis. *ANESTHESIOLOGY* 43:489-490, 1975
6. Horton B: Anesthetic experience in a family with hypokalemic familial periodic paralysis. *ANESTHESIOLOGY* 47:309-310, 1977