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General Anesthesia and Hyperkalemic Periodic Paralysis

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The periodic paralyses are a group of disorders characterized by recurrent attacks of weakness, associated with changes in serum potassium concentration. Three distinct groups of periodic paralysis have been described: hyperkalemic, hypokalemic, and normokalemic. Only scanty anesthetic experience has been reported with the hyperkalemic group. In 1959, Egan and Klein described three patients with hyperkalemic periodic paralysis who underwent anesthesia.[1] In each case, upon awakening from general anesthesia, the patient was unable to move for several hours. The anesthetic given in two cases for dental procedures was thiopental. The anesthetic administered in the third case for childbirth was unspecified. One patient delivered a child later under spinal anesthesia without developing paralysis. In 1980, Flewellen and Bodensteiner described a general anesthetic in a 10-yr-old girl for correction of esotropia and gastrocnemius muscle biopsy.§ General anesthesia was induced with inhalation of halothane with 50% nitrous oxide in oxygen. The patient's trachea was intubated and controlled hyperventilation was instituted without the use of muscle relaxants. During the course of the anesthetic, sinus rhythm was interrupted by occasional unifocal extrasystolic beats. Halothane was discontinued and enflurane was begun with return of sinus rhythm. The patient awoke with return of spontaneous respiration and her trachea was extubated without difficulty. We present a description of the first successful management of an adult male patient with hyperkalemic periodic paralysis who received general anesthesia along with muscle relaxants and did not develop muscle weakness on awakening or during recovery.

CASE REPORT

The patient was a 40-yr-old, 85-kg man who began having muscle weakness in his legs at age 9 yr. The weakness occurred shortly after awakening and was more profound following rest after exercise. No myotonia was present and the neurological exam was normal. Hyperkalemic periodic paralysis was diagnosed at age 20 yr by oral administration of KCl which provoked his usual attack. He was receiving hydrochlorothiazide 25 mg/day for 2 yr, and had been free of symptoms. In 1988, he developed numbness and weakness in his left arm and left thumb. After studies, a herniated nucleus pulposus was revealed at C5–6 with spurring of C6–7 and surgery was scheduled for anterior cervical discectomy and fusion.

Admission laboratory values were within normal limits, including a serum potassium of 3.6 meq/l and an EKG that showed normal sinus rhythm, without T-wave abnormalities. The patient received hydrochlorothiazide 25 mg orally 1 h prior to arrival to the operating room. In the operating room, an iv infusion was started and a radial artery was cannulated. EKG, blood pressure cuff, and pulse oximeter were applied. Peripheral nerve stimulator electrodes were applied over the ulnar nerve. Anesthesia was induced with thiopental 250 mg iv. Six mg of vecuronium iv resulted in rapid onset of paralysis and tracheal intubation was accomplished without difficulty. The maintenance anesthetic was nitrous oxide (3 l/min), oxygen (2 l/min), isoflurane (.6–.8%), and fentanyl 750 μg in divided doses over 4 h. Evidence of return of neuromuscular function by train-of-four monitoring was apparent within the first hour of surgery and no additional muscle relaxant was given after the initial dose. Fluids not containing potassium were used throughout the perioperative period (5% dextrose with 0.9% normal saline 500 ml, normal saline 500 ml, 5% dextrose with 0.45% normal saline 1000 ml). Normothermia was maintained using a heated humidifier and fluid warmer. Serial blood gases, serum sodium, potassium, and glucose concentrations were monitored throughout the course. The intraoperative serum potassium concentrations, measured hourly, were 3.6, 4.1, 3.8, and 3.5 meq/l. The surgical procedure was completed in 4 h. The patient was awakened after demonstration of adequate return of neuromuscular function including full train-of-four

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§ Flewellen EH, Bodensteiner JB: Anesthetic experience in a patient with hyperkalemic periodic paralysis. Anesthesia Review 7:44–46, 1980

and tetany without fade at 50 Hz. He could follow commands appropriately. His trachea was extubated and he was taken to the recovery room. He showed no evidence of muscle weakness during the recovery period of 2 h or at postoperative visits at 24 h and 48 h.

Discussion

Although hypokalemic periodic paralysis was originally described in 1882, the hyperkalemic form was not reported in the literature until 1957.[2] Both forms of the disease are genetically transmitted with autosomal dominant inheritance and muscle weakness may be precipitated with rest after exercise or cold weather. A carbohydrate meal may provoke muscle weakness in the hypokalemic type whereas it may alleviate the symptoms of the hyperkalemic type. Generally, attacks of the hypokalemic form are nocturnal and prolonged, whereas those of the hyperkalemic form are brief and occur during the daytime. In the hyperkalemic form, the attacks usually begin as stiffness in the legs and this may spread to the back and arms. The cranial nerves may be affected, especially with myotonic symptoms such as stiff eye movements, blurred vision, and dysphagia.[3] Familial periodic paralysis may also be associated with cardiac arrhythmias which are refractory to conventional therapy.[4,5]

Hyperkalemic periodic paralysis is actually a misnomer, since the concentration of the serum potassium may be within normal limits during an attack. The diagnosis is made by the finding of the appropriate clinical symptoms and the confirmation by provocation of paralysis with oral KCl 0.05 to 0.15 g/kg.[6] Exclusion of the hypokalemic form is made by an intravenous test with glucose and insulin to provoke the shift of potassium into cells. The pathogenesis of this disease is unknown, but may involve altered resting membrane potentials secondary to abnormalities in the membrane permeability to sodium or potassium.[3,6]

The anesthetic considerations for the disease have been reviewed.[7] They include avoidance of fasting and intravenous infusion on the night before surgery of a dextrose-containing fluid that contains no potassium. These patients often exhibit myotonia and succinylcholine may induce muscle spasm in myotonic patients.[8,9] Succinylcholine should be avoided in view of the danger of inducing severe muscle spasm. If muscle relaxation is necessary, an intermediate duration nondepolarizing neuromuscular blocker, atracurium or vecuronium, does not seem to be contraindicated. Intraoperatively, normothermia should be maintained, as cold may provoke an attack. Potassium concentration may rise by only 20% during an attack and still be within the normal laboratory limits.[7] For longer operations, serial glucose and potassium concentrations should be monitored. EKG should be monitored for evidence of hyperkalemia.

Patients with this disease are usually receiving either acetazolamide or thiazide diuretics in order to lower the serum concentration of potassium. Once an attack is underway, aborting it may be attempted by driving potassium back into cells. This may be accomplished with glucose and insulin, epinephrine, calcium gluconate, or glucagon.[6] Recently, metaproterenol has been shown to prevent attacks and to hasten recovery.[10]

In conclusion, we describe the first successful management of a patient suffering from hyperkalemic periodic paralysis who required general anesthesia for cervical laminectomy. We used an intermediate duration neuromuscular blocker for intubation and monitored the return of neuromuscular function. We monitored serum potassium concentration throughout the intraoperative course. The patient awoke with good muscle strength and showed no exacerbation of the disease.

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