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Anesthetic Management of a Pediatric Patient with Hypokalemic Periodic Paralysis - A Case Report

Wei Chou^{1*}, Ping-Heng Tan² and Wen Chne I²¹Department of Anesthesiology, E-DA Hospital, Taiwan²Department of Anesthesiology, Chi-Mei Medical Hospital, Taiwan

*Corresponding author: Wei Chou, Department of Anesthesiology, E-DA Hospital No. 1, Yida Road, Jiao-Su Village, Yan-Chao District, Kaohsiung City 824, Taiwan, Tel: +886-7-6150011-Ext-252961; E-mail: mavisng@gmail.com

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Abstract

We report on the anesthetic management of an 11-year-old girl with hypokalemic periodic paralysis who underwent left radius plate removal surgery. This patient's course was complicated early in the postoperative period by an episode of bilateral lower limb paralysis. She presented with hypokalemia after general anesthesia; however, she recovered well after taking a potassium supplement. Patients with this disorder, who are apparently normal, can develop sudden paralysis when they are exposed to predisposing factors peri-operatively. Complications related to this rare genetic disorder, the factors that can precipitate these, and preventive measures are discussed.

Keywords: Postoperative weakness; Hypokalemic periodic paralysis; Paralysis

Introduction

Hypokalemic periodic paralysis is a rare genetic disorder characterized by recurrent attacks of skeletal muscle weakness due to associated hypokalemia [1]. It is an autosomal dominant disease [1]. Affected individuals may experience paralytic episodes with concomitant hypokalemia (<2.5 mmol/L) and occasionally may develop late-onset proximal myopathy [1].

The paralytic attacks are characterized by reversible flaccid paralysis, usually leading to paraparesis or tetraparesis but typically sparing the respiratory muscles and heart [1]. Penetrance is about 90% in males and may be as low as 50% in females, depending on the causative mutation [1]. The diagnostic criteria were based on clinical symptoms and genetics tests [1].

The age of onset of the first attack ranges from one to 20 years; the frequency of attacks is highest between ages 15 and 35 and decreases thereafter [1]. Symptom onset occurs at a younger age in females than in males [1].

An increased risk of paralysis symptoms after anesthesia has been reported. The most important considerations involve maintaining the potassium level and avoiding precipitating factors [1]. Generally, any condition leading to low plasma potassium concentrations has been implicated in noted paralysis attacks [1]. In most instances, there is an apparent shift of extracellular to intracellular potassium, a shift that alters resting membrane potential. Stress, large meals, glucose infusion, insulin, hypothermia, and metabolic or respiratory alkalosis all have been described as triggering factors. The symptoms may last hours or days [1].

Hypokalemia is treated with potassium replacement to normalize serum potassium and to shorten the duration of the attacks. We report a pediatric patient with hypokalemic periodic paralysis who experienced a sudden attack of bilateral lower limb paralysis after general anesthesia.

Case Presentation

An 11-year-old female patient (152 cm, 42 kg), known to have hypokalemic periodic paralysis, suffered a left radial bone fracture post-surgery treated with open reduction and internal fixation. The patient was scheduled to receive fixator removal surgery.

The patient had a history of familial hypokalemic periodic paralysis, which was diagnosed at the age of six. Her father had the disease, which resulted in weakness of his lower limb muscles during attack.

He experienced several severe attacks involving paraplegia without respiratory system effects. The symptoms usually resolved by taking potassium supplements. He is therefore on a long-term prescription of acetazolamine and potassium tablets. Her grandmother also has the disease. The patient complained of mild weakness of the proximal lower limb muscles, which increased after exercise.

Typically, she reported 2–3 episodes of severe weakness per year, which were precipitated by fatigue, stress, heavy meals, sweet snacks, and/or cold and salty food. She usually recovered several hours later by resting, eating a banana, or

taking potassium tablets. She did not receive medication for the disease.

Her preoperative physical examination showed normal muscle power in all extremities. Her breathing sound presented as clear bilaterally. Her laboratory data showed a normal blood hemoglobin level (12.3 g/dl), normal serum potassium (3.9 mEq), and normal liver function. Bleeding parameters were normal. The chest X-ray and ECG results were normal.

On arrival to the OR, we set up monitors, including ECG, pulse oximetry, and non-invasive blood pressure. Anesthesia was administered—fentanyl 50 µg, thiamylal 325 mg, lidocaine 40 mg, and cisatracurium 2 mg. Laryngeal mask airway (LMA) size 3 was smoothly inserted. Inhalation anesthetic was maintained with MAC 1.3–1.5 of sevoflurane. Hypocapnia was avoided by monitoring end tidal carbon dioxide.

An oropharyngeal temperature probe was used to monitor intraoperative temperature. Measures were taken to prevent hypothermia, which included drapes to cover the patient, an air warming device, and warm fluid. Maintenance fluid was normal—saline 0.9%. The surgery lasted 1.5 hours. The hemodynamics were stable throughout the procedure, with a heart rate 80–90 beats/min and a mean blood pressure of 75–80 mmHg. Blood loss was minimal. LMA was removed when the patient was fully awake and could respond to the order. The operation was successful without complications. She was then transferred to a post-anesthesia care unit (PACU).

In the PACU, she found that she could not move her legs bilaterally. We did a physical examination and found that her lower limb muscle power was grade 1. She could only move her toes slightly. Her lower limb reflexes were absent. Her vital signs were stable, and she breathed smoothly. She had no other discomfort.

Her family ensured us that she would recover within a few hours and that she did not need aggressive examination or evaluation. We monitored her for weakness progression or effects on the respiratory muscle. After 1.5 hours, she could barely elevate her legs about 1–2 cm off the bed. No respiratory distress was found. After discussion with her, her family, and her orthopedics doctor, we decided to check her electrolytes. Her serum potassium was 2.6 mEq/L, and sodium was normal. She received an intravenous KCL drip (10 mEq/500 ml). Her muscle power recovered fully after 20 hours. She was discharged the day after surgery.

Discussion

This report is the first to describe the anesthetic management of a pediatric patient with familial hypokalemia periodic paralyses (FHPP). FHPP is a group of disorders involving muscle weakness and skeletal muscle ion channel mutations [1].

For children with neuromuscular disease, we have some concerns during pre-operation evaluation. Pulmonary function testing should be performed if the patient is capable of

participating [2]. If they cannot take the test, SpO₂ must be examined by pulse oximetry [2].

Pulmonary dysfunctions should be prepared for. Electrocardiogram can be done to rule out cardiac arrhythmia [2]. Malnutrition puts all patients at risk for perioperative loss of skin integrity due to pressure ulcerations, respiratory complications, and poor postoperative wound healing [2].

The great emphasis on anesthetic management of affected patients involves the prevention of episodes of muscle weakness. Carbohydrates induce paralysis attack [3]; therefore, glucose and insulin should be avoided [3].

Blood potassium levels should be checked and corrected preoperatively. Because there is a correlation between periodic hypokalemia paralysis and malignant hyperthermia, we should use an MH-trigger-free anesthetic method [4]. During mechanical ventilation, we can keep controlled hypercapnia (pCO₂ about 40 mmHg) to avoid alkalosis. Prevention of hypothermia is important for a pediatric patient.

The use of warm blankets and the avoidance of low operating room temperature would be helpful. Use a temperature monitor to prevent chilling, which seems to precipitate the attack. The use of a short-term, non-depolarizing muscle relaxant is suggested [5]. Atracurium has been reported as useful without complication [5]. Neuromuscular monitoring is mandatory.

The patient's potassium level was within normal range preoperatively. Her temperature was kept over 36°C by a warm blanket and warm fluid infusion. Choice of IV fluid was normal saline with no glucose added. We maintained her normothermia and normocapnia during the operation. The muscle relaxant administered was short-acting cisatracurium.

Hypokalemia and limbs paralysis still occurred postoperatively. She recovered well after potassium supplementation and had no long-term complication. Anesthesia is a triggering factor, yet no definite mechanism was discovered. However, treating the electrolyte imbalance aggressively and avoiding or mitigating aggravating factors are the most important considerations during anesthesia.

Conclusion

In conclusion, adequate preoperative preparation, vigilant intraoperative monitoring and management of potassium levels, avoiding factors that can trigger hypokalemia and good postoperative pain relief play important roles in the successful management of this group of patients. A carefully-planned, multidisciplinary approach to the perioperative care of pediatric patients with FHPP helps avoid anticipated and unanticipated complications.

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