Case Report

Hypokalemic periodic paralysis

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ABSTRACT

Hypokalemic periodic paralysis is a rare genetic disorder characterized by recurrent attacks of skeletal muscle weakness with associated hypokalemia which is precipitated by stress, cold, carbohydrate load, infection, glucose infusion, hypothermia, metabolic alkalosis, anesthesia, and steroids. We encountered one such incidence of prolonged recovery after general anesthesia, which on further evaluation revealed a case of hypokalemic paralysis. The key to successful management of such a patient was vigilant pre-operative evaluation, perioperative monitoring, and aggressive treatment of hypokalemia when it occurs.

Key words: General anesthesia, hypokalemia, paralysis

Introduction

Hypokalemic periodic paralysis (HKPP) is a rare genetic disorder with autosomal dominant inheritance and characterized by recurrent attacks of skeletal muscle weakness with associated hypokalemia which is precipitated by stress, cold, carbohydrate load, infection, glucose infusion, hypothermia, metabolic alkalosis, anesthesia, and steroids. The key to successful management of such a patient is vigilant preoperative evaluation, perioperative monitoring, and aggressive treatment of hypokalemia when it occurs.

Case Report

A 45-year-old man was scheduled for molar extraction under general anesthesia. The patient was assessed having an American Society of Anesthesiology (ASA) 1 status after thorough preoperative evaluation. The patient fasted for 8hours prior to surgery. In the pre-operative room, a 20-gauge i.v. cannula was placed after infiltration with 2% lidocaine and an infusion of 5% dextrose normal saline. In operating room, standard monitors were fixed. The patient was anaesthetized with 1 mg intravenous midazolam, 100 μg fentanyl followed by 120 mg propofol and laryngeal mask was inserted and patient maintained on oxygen, nitrous oxide, and isoflurane on spontaneous ventilation. The surgical procedure was completed within 30 min and the patient was transferred to the post-anesthesia care unit. Recovery nurse informed us about the flaccidity of the limbs though vital parameters remained absolutely normal.

On assessment, patient was not having any sensory loss and responded well to cold sensations and light touch but motor power in lower limbs was 2/5, upper limb was 3/5 even 5 h after surgery, but there was no difficulty in breathing and patient maintained oxygen saturation on room air. We decided to send high dependency unit (HDU) profile. HDU profile revealed patient having hypokalemia with potassium level of 2 mmol/l. Electro cardio graph (ECG) record also showed features of hypokalemia. Potassium infusion was started (10 meq/h KCl [2 meq/ml] administered at 5 ml/h through peripheral vein) and the patient started recovery and full motor power was regained after 6 h. After exhaustive counseling, patient's relative revealed history of early exhaustion and some muscular weakness felt by patient after sweating or hard work and this disappeared after fluid intake. Post-operative pain management was provided by intravenous paracetamol. He was discharged from the hospital on fourth post-operative day during which time he continued to show no evidence of muscle weakness.

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Discussion

Hypokalemic familial periodic paralysis is a rare channelopathy with muscle weakness and a matching fall in the potassium levels in the blood; it is a autosomal dominant genetic disorder[1] in the gene encoding for the dihydropyridine sensitive calcium channel.[2] The weakness may be limited to muscle groups or may present as severe muscle paralysis. The mechanism for a decrease in potassium is felt to be associated with abnormal uptake of potassium by the muscle cells[3,4] and usually presents either as a paralytic form or as a paramyotonic form.

In hypokalemic familial periodic paralysis, the factors that trigger weakness or paralysis are anesthesia, surgery, pregnancy, insulin, alcohol, strenuous exercise, and steroids.[5,6] Similar episode was observed in our case as pre-operative stress along with necessary fasting and administration of dextrose containing fluids precipitated the attack. The guidelines for care include control of plasma potassium, avoidance of large glucose and salt loads, maintenance of body temperature, acid–base balance, and careful use of neuromuscular blocking agents.[7,8] Good pre-medication to allay anxiety, avoidance of stress and adequate analgesia is vital in preventing an attack. Fluctuations in electrolytes, infection, and pain can lead to paralysis in the post-operative period. Hypokalemia manifests earlier than paralysis and so its correction can prevent paralysis.[9] Dextrose containing solutions administered during surgery should be avoided and normal saline (0.9%) should be preferred.

Aside from the classical periodic episodes of weakness in response to triggers, patients often report additional symptoms either before, during, or after attacks. These include paresthesias, sweating, myalgia, extreme fatigue, thirst, shortness of breath (either due to anxiety or to the episode itself), palpitations, clumsiness, irritability, and mental dullness.[10] The differential diagnoses stem from thyrotoxic HKPP, hyperkalemic periodic paralysis, paramyotonia congenita, potassium-aggravated myotonia, myotonia congenita (MC), both recessive and dominant MC, hyperaldosteronism and physiologically similar states, diuretic abuse, and myasthenia gravis. Provocative testing can be dangerous and is not a favored first-line method of diagnosis. Potassium challenge tests risk hyperkalemic arrhythmia, even in an acute care setting. Insulin challenge tests can be equally dangerous due to risk of hypoglycemia. Simple exercise challenge, which is relatively safe, is partly helpful when the serum potassium is high or low. Specifically, ECG, TSH, free T3 and free T4 are the minimum indicated laboratory investigations, with renal and adrenal function also recommended. With respect to genetic counseling, these disorders are autosomal dominant with male preponderance. To conclude, we emphasize that we can get cases of unanticipated delayed recovery after general anesthesia even after thorough pre-operative assessment and they are not always neuromuscular. But other causes like electrolyte imbalance should also be investigated and aggressive correction of hypokalemia can treat intraoperative HKPP if it occurs.

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