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Severe Hyperkalemia and Cardiac Arrest Following Rapid Sequence Induction and Intubation Using Succinylcholine: A Case report

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Abstract: A 48-year-old male patient who had undergone ventriculoperitoneal (VP) shunt surgery for hydrocephalus 18 days earlier was scheduled to undergo emergency VP shunt revision due to inflammation in the abdomen. Intubation was performed using succinylcholine because the patient was not in a fasting state. Following intubation, the patient experienced sudden cardiac arrest, and the potassium level showed an increase from the preoperative level of 3.4 mEq/L to 9.2 mEq/L. The patient underwent treatment for hyperkalemia while cardiopulmonary resuscitation (CPR) was performed. Following recovery of the vital signs, the patient underwent the shunt revision surgery. He was then transferred to the intensive care unit (ICU) for monitoring, and subsequently discharged from the hospital once his condition had stabilized.
Keywords: General anesthesia; hydrocephalus; hyperkalemia; succinylcholine.

INTRODUCTION

Succinylcholine is a depolarizing muscle relaxant that is often used for muscle relaxation in short surgeries or in cases in which rapid sequence induction and intubation (RSII) for anesthesia is necessary [1]. The adverse effect associated with the use of succinylcholine that has the greatest potential for fatality is hyperkalemia. In patients without neuromuscular disease, the serum potassium (K^+) concentration generally shows a transient increase of about 0.55 mEq/L [2], but severe hyperkalemia may occur in the presence of hypovolemia or metabolic acidosis [3].

The author herein report a case of a patient whose preoperative K^+ concentration was normal and in whom cardiac arrest following a potentially fatal arrhythmia occurred after the administration of succinylcholine, with subsequent full recovery.

CASE REPORT

A 162 cm, 60 kg, 48-year-old male patient was scheduled to undergo emergency revision of a ventriculoperitoneal (VP) shunt due to exacerbated hydrocephalus and infection of the VP shunt catheter.

Twenty-eight days earlier, the patient had been transported to the emergency room due to stuporous mentality that followed the sudden onset of headache. Brain computed tomography (CT) showed spontaneous subarachnoid hemorrhage, and the patient underwent emergency decompressive craniectomy. The postoperative brain CT showed that the brain swelling had increased in severity, and the patient's mental state had not improved. Therefore, extended craniectomy was performed that same day. The patient was transferred to the intensive care unit (ICU) for monitoring following the procedures.

On the first day after the surgeries, sudden hypotension (systolic blood pressure 70 mmHg) occurred, and continuous infusion of norepinephrine was initiated. Immediately after that, the electrocardiogram (EKG) showed atrial fibrillation (heart rate 176 beats/minute), and thus continuous infusion of amiodarone was started. Following this treatment, recovery of sinus rhythm was observed.

On the ninth day following the surgeries, follow-up brain CT was performed because the patient's mental state remained unchanged. The scan showed hydrocephalus. Therefore, VP shunt surgery was conducted, after which the patient's level of consciousness improved to a drowsy state.

On the sixth day following VP shunt surgery, the tympanic temperature was 38℃ or higher, and the white blood cell count was 24,670/μL. Continuous intravenous (IV) injection of antibiotics was performed, but the patient's condition did not improve. Flares and edema were observed around the site of implantation of the VP shunt catheter into the patient's abdomen. Emergency VP shunt revision was therefore planned.

Preoperative laboratory electrolyte tests performed the morning of the scheduled emergency VP

shunt revision showed sodium (Na^+) 136 mEq/L, potassium (K^+) 3.4 mEq/L, and chloride (Cl^-) 105 mEq/L, all of which were normal. The EKG also showed normal sinus rhythm (heart rate 79 beats/min).

The patient was administered fluid only through a nasogastric tube for two hours before the surgery, and thus the preoperative fasting period was insufficient. Thirty minutes before the start of the surgery, the patient received metoclopramide 10 mg, famotidine 20 mg, and glycopyrrolate 0.2 mg by intramuscular injection.

Upon transfer to the operating room, the patient's level of consciousness was drowsy, and he was receiving continuous infusion of amiodarone and norepinephrine. Following arrival in the operating room, EKG, noninvasive pulse oximetry (SPO_2), and noninvasive blood pressure (NIBP) were monitored. Initially, the patient's NIBP was 111/91 mmHg, the SPO_2 was 99%, and the EKG showed sinus tachycardia (heart rate 114 beats/min).

The Sellick maneuver was performed during the induction of anesthesia with the IV injection of etomidate 12 mg. After verifying the loss of consciousness, succinylcholine 100 mg was IV injected for RSII, and, about one minute later, intubation was performed using direct laryngoscopy.

One minute after the tracheal intubation, the measurement of SPO_2 suddenly failed, and the EKG showed ventricular tachycardia (VT). Direct current (DC) cardioversion at 200 J was performed immediately, with cardiac massage. Epinephrine 1 mg was IV injected two times with an interval of three minutes, but normal rhythm was not restored. Under continuous cardiac massage, an electrolyte test was performed, and the results were Na^+ 136 mEq/L, K^+ 9.2 mEq/L, and Cl^- 105 mEq/L. IV injection of calcium gluconate 2 g and furosemide 20 mg was immediately performed. A solution prepared by mixing regular insulin 10 IU in 50% dextrose 50 mL was administered IV. Temporary recovery of normal rhythm was observed ten minutes after the DC cardioversion.

However, the EKG again showed VT ten minutes later. DC cardioversion at 200 J was once again performed, followed by cardiac massage. Epinephrine 1 mg was injected IV. Electrolyte testing showed Na^+ 131 mEq/L, K^+ 6.8 mEq/L, and Cl^- 105 mEq/L. Under hyperventilation, the solution prepared by mixing regular insulin 10 IU in 50% dextrose 50 mL was again administered IV. Five minutes after the second DC cardioversion, recovery of normal heart rhythm was observed. Left radial artery cannulation was performed to measure the arterial blood pressure (ABP).

Subsequently, the heat rate was maintained at about 100 beats/min, and the ABP was about 120/65 mmHg. The EKG continued to show sinus rhythm. Therefore, in consultation with the department of neurosurgery, it was decided to resume the surgery.

Electrolyte testing performed immediately prior to the end of the surgery showed Na^+ 133 mEq/L, K^+ 3.3 mEq/L, and Cl^- 103 mEq/L. However, the blood glucose level was 469 mg/dL, and thus regular insulin 10 IU was injected IV.

Following the surgery, the patient was immediately transferred to the ICU, without awakening, and was placed on a ventilator. Electrolyte tests performed three hours after the surgery showed Na^+ 140 mEq/L, K^+ 3.2 mEq/L, and Cl^- 104 mEq/L. The EKG showed no remarkable findings except sinus tachycardia (heart rate 129 beats/min).

The patient remained in the ICU due to his drowsy mental status, with no significant abnormalities in the vital signs. Chest X-ray performed on the fourth postoperative day showed pneumonic consolidation, and thus the patient underwent antibiotic therapy for one month. After the chest X-ray findings showed improvement, the patient was transferred to a local clinic.

DISCUSSION

Succinylcholine, a depolarizing muscle relaxant with a rapid onset and ultrashort duration of action, demonstrates its full muscle-relaxing effect within one minute after administration at 1 mg/kg [4], and 90% of the muscle strength may be recovered within 9 to 13 minutes [5].

Due to these characteristics, succinylcholine is frequently used to avoid RSII in situations of bowel obstruction, nasogastric tube placement, pregnancy, morbid obesity, and diabetic gastroparesis, in which regurgitation of gastric contents is highly probable [6].

Adverse effects of succinylcholine include hyperkalemia, dysrhythmias, increased intraocular pressure, increased intragastric pressure, increased intracranial pressure, and masseter spasm [7]. Following the administration of succinylcholine, the K+ concentration is generally increased by about 0.55 mEq/L in patients having no neuromuscular disease, but by about 1.8 mEq/L in patients with a neuromuscular disease [2].

According to the main mechanism of induction of hyperkalemia by succinylcholine, the upregulation of the skeletal muscle acetylcholine receptors increases the number of acetylcholine receptors in the end plate and converts the subunit type of the receptors from the gamma (ε) type to the epsilon (γ) type, which prolongs the average channel opening time and enhances the efflux of K^+, resulting in hyperkalemia [8].

Situations in which hyperkalemia may easily be caused by the administration of succinylcholine include the presence of functional or anatomical denervation due to lower or upper motor neuron injury; immobilization of a patient during ICU treatment or for other reasons; infections, burns, or direct muscle trauma; and catatonic schizophrenia, meningitis, necrotizing pancreatitis, purpura fulminans, or gastrointestinal mucositis [8,9].

Hyperkalemia, an electrolyte imbalance that may lead to fatal outcomes, is caused when K^+ is not excreted by the kidneys or when the body's capability to transport K^+ from the circulatory system to the cells is lowered by medication or adrenal insufficiency. The diagnosis of hyperkalemia begins with review of a patient's clinical history and medications. The EKG shows tall, peaked T waves, loss of P waves, and widening of the QRS complex [10].

Treatment of hyperkalemia is generally performed when EKG changes are observed or when the measured serum K^+ level is 6.0 mEq/L or higher [10]. The first approach to treatment of hyperkalemia is the IV injection of 10% calcium gluconate 10 to 20 mL over two to three minutes, which does not affect the serum K^+ level but is performed for protection of the myocardium. The second treatment approach is the IV injection of a solution prepared by mixing regular insulin 10 IU and 50% glucose 50 mL or the nebulization of albuterol 10 to 20 mg to shift K^+ into the cells. Finally, furosemide may be injected IV to enhance the excretion of K^+ through the kidneys, or 50 g of sodium polystyrene sulfonate may be administered orally or rectally to induce the excretion of K^+ through the gut [10].

In the present case report, the patient was administered a fluid diet via nasogastric tube for two hours before the surgery, and thus the fasting period was insufficient prior to the administration of succinylcholine. The patient experienced a seemingly fatal complication, as the serum K^+ level increased by 5.8 mEq/L, from 3.4 mEq/L to 9.2 mEq/L, after the induction of anesthesia. Causes of this increase may include hypovolemia induced by the preoperative IV injection of mannitol, which was performed as a result of the brain edema and hydrocephalus; the patient's state of immobilization during the preceding 28 days in the ICU; and muscle injury that might have occurred during the two craniectomy procedures previously performed. Certain clinical findings also suggested inflammation and infection.

Although recovery was achieved through immediate countermeasures in the present case, the mortality of cardiac arrest caused by hyperkalemia following the administration of succinylcholine is 19% or higher, indicating that hyperkalemia caused by the administration of succinylcholine may result in a very dangerous situation [8]. Therefore, in cases in which a patient has significant risk factors for the development of hyperkalemia following administration of succinylcholine, the use of rocuronium, a nondepolarizing neuromuscular blocking agent with a rapid onset of action, may be considered. Rocuronium at a dose of 1.2 mg/kg is known to have effectiveness equivalent to that of succinylcholine [11]. Sugammadex may be administered to accomplish predictable, complete, and rapid reversal of rocuronium-induced neuromuscular blockade [12].

CONCLUSION

As shown in the present case report, in a patient with various risk factors for the development of hyperkalemia following administration of succinylcholine, the potentially fatal adverse effects should be anticipated in advance, and providers should be prepared to perform immediate diagnosis through EKG monitoring or rapid electrolyte testing, as well as immediate treatment. Administration of rocuronium at 1.2 mg/kg may be considered in lieu of succinylcholine.

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