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Case Report

Suspected Hyperkalemia-Induced Cardiac Arrest and Recovery Following Succinylcholine Use in a Trauma Patient

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INTRODUCTION

Succinylcholine is one of the most used drugs in anesthesiology and is the only depolarizing neuromuscular blocker in use today.[1] Succinylcholine functions by binding to nicotinic acetylcholine receptors (nAChR) located at the neuromuscular junction (NMJ). This interaction causes a conformational change in the nAChR resulting in cell depolarization, an efflux of potassium, and the generation of an action potential that results in a muscle contraction.[1] However, its clinical application is tempered with the occurrence of rare, yet devastating side-effects. When used in doses sufficient for intubation, succinylcholine has been observed to raise serum potassium concentrations by 0.3-1.0 mEq/L,[2,3] which can lead to life-threatening arrhythmias and cardiac arrest.[4] Moreover, nerve injury, as a sequela of crush injury, can lead to expression of nAChR receptors throughout the muscle cell membrane, and thus an exaggerated response to a standard dose of succinylcholine. In recognition of this possibility, the Food and Drug Administration (FDA) label for succinylcholine contraindicates its use in patients in the acute phase of an injury due to the potential for hyperkalemia. Our case describes its use and complications following administration to a patient in the subacute phase of recovery following a crush injury. Written, informed consent was obtained from the patient for publication.

CASE REPORT

Our patient was a 42-year-old male who presented as a Level II trauma activation after sustaining a crush injury with a steel beam to the abdomen. Per emergency medical services personnel, the patient was pinned under the 25-foot beam for approximately 10 minutes. He denied any loss of consciousness and remained hemodynamically stable with a Glasgow Coma Scale (GCS) of 15 throughout transport. A foley catheter was placed prior to arrival and bloody urine was noted upon arrival to our facility. Additional past medical history was significant for Stage III chronic kidney disease, but was otherwise non-contributory. The patient sustained a long hospital course in the intensive care unit (ICU) with multiple trips to the operating room (OR), the first occurring on hospital day two where an exploratory laparotomy was performed for decompression of a developing abdominal compartment syndrome. He subsequently underwent five additional abdominal operations under general anesthesia for debridement and wound vacuum changes without incident.

On hospital day 30, the patient was brought to the OR once again for final closure of his abdominal wound. He was attached to standard physiologic monitors and pre-oxygenated with 100% FiO2 via bag mask ventilation. Venous access consisted solely of a 24-gauge in the right hand, and the decision was made to proceed with this line through induction and place a larger gauge catheter once the patient was asleep. Anesthesia was induced with 5 mL lidocaine, 100 mcg fentanyl, and titrated doses of propofol to a total of 200 mg. The patient was a difficult bag-mask secondary to facial hair and jaw clenching. A 9 cm oral airway was placed with achievement of satisfactory ventilation, and 100 mg succinylcholine was then administered. An initial attempt at direct laryngoscopy proved difficult and was aborted secondary to desaturation to 63%; he was manually ventilated back to 100% and a second attempt at direct laryngoscopy was successful. Controlled ventilation was initiated, and the tube was taped in place while another anesthesia provider began looking for a second intravenous (IV) site. During this period of time the patient was noted to have peaked T-waves that were not found on pre-operative electrocardiogram (EKG) that quickly progressed to a wide-complex rhythm. Systolic blood pressure was noted to be about 70 mmHg and was unreadable on a recheck. Disorganized electrical activity was then noted on EKG with pulsatile wave form on plethysmography. A pulse check was performed at the carotid artery and no pulse was observable.

At this time chest compressions were initiated, and a code blue was called. Pharmacy was notified for treatment of presumed hyperkalemia. The patient's 24-gauge peripheral IV was no longer able to flush and was thought to have infiltrated sometime after induction. An IV attempt in the left external jugular vein was unsuccessful. Bilateral groins were prepped by surgery and a left central line was placed, along with a right femoral arterial line for pressure monitoring. One gram calcium gluconate was pushed, followed by 25 g of dextrose with 10 U of insulin, and 360 mcg of albuterol were delivered through the endotracheal tube. Return of spontaneous circulation was noted nine minutes after initiation of chest compressions and the code was called off. Systolic blood pressure following these interventions was 210 mmHg and returned to pre-induction values of about 140 mmHg over a course of a few minutes. EKG displayed normal sinus rhythm. Inhalational anesthetic was discontinued, and the patient was switched to spontaneous respirations on the ventilator. He demonstrated appropriate tidal volumes and respiratory rates and was extubated without incident. He was transported to the ICU and upon arrival the patient was GCS 15 and in no apparent distress. Labs were drawn on arrival to the ICU and showed a potassium of 3.9, compared to a pre-operative value of 4.1. The patient was taken back to the OR the following day for final closure of his abdominal wound. He was discharged to inpatient rehabilitation three days following the events described above.

DISCUSSION

The case above highlights the reversibility of hyperkalemia-induced cardiac arrest. Imperative to success in this example was the quick recognition of perioperative problems and multidisciplinary team work to initiate appropriate treatment modalities. As will be discussed below, this case also highlights numerous topics for further research, more

focused guidelines, and potential innovation.

Succinylcholine has been found to raise serum potassium levels by 0.3-1.0 mEq/L when administered in induction doses ranging from 0.5-1.0 mg/kg in patients with normal potassium levels prior to administration.[2,3] Hyperkalemia can lead to adverse cardiac effects and manifests as changes on EKG. These changes include peaked T-waves, diminished P-waves, widened QRS, and various arrhythmias including atrioventricular blocks, bradycardia, ventricular tachycardia, and ventricular fibrillation.[5-8] Cardiovascular instability usually occurs at potassium levels > 8 mEq/L, though values of more than 11 mEq/L have been recorded without any cardiovascular complications.[9,10] Though our patient appeared to show a step-wise progression from peaked T-waves to a wide-complex rhythm and ultimately pulseless electrical activity, EKG changes occur in a variable fashion and some patients with coexisting electrolyte abnormalities/pathologies, such as hypercalcemia, hypernatremia, alkalemia, myocardial ischemia, intraventricular conduction delay, and end-stage renal disease may exhibit no EKG changes secondary to the effect of disease-related fluctuations in calcium levels.[11]

Early and aggressive correction of potassium levels is an important first step when hyperkalemia-related cardiac arrest is suspected. Literature supports initial treatment with 1-2 g calcium chloride or gluconate to act as a cardiac membrane stabilizer prior to correction whenever EKG findings are first noticed.[12] Nebulized or inhaled β2-agonists and 1 ampule of D50 with 10 U of insulin IV promote potassium shift from the extracellular space into the intracellular compartment and represent the mainstay of treatment. The use of sodium bicarbonate to enhance intracellular shift is controversial.[12,13] These therapies have a quick onset of action and are temporizing measures that have been shown in studies to significantly lower potassium levels within 30 minutes.[12] We did not obtain lab values during the arrest in our case, yet labs drawn 71 minutes after the event revealed a normal potassium level of 3.9 mEq/L, compared to a pre-operative level of 4.1 mEq/L.

Notably, most of the literature discussing succinylcholine use after trauma describes patients with pre-existing elevated potassium levels (e.g., pathology involving an upregulation of post-junctional acetylcholine receptors). These patients are particularly susceptible to succinylcholine-induced hyperkalemia 24-72 hours following traumatic injury.[14] Our patient was never found to have an elevated potassium level throughout his entire hospital stay, nor did the day of the cardiac arrest fall within the aforementioned time frame. Also, according to the FDA-approved drug label for succinylcholine, patients in the acute phase of injury show a susceptibility for hyperkalemia, which peaks at 7-10 days after the event.[15] This is due to both receptor upregulation and the influence of rhabdomyolysis on intracellular potassium release.[9,16] Receptor upregulation is a manifestation of both increased numbers of acetylcholine receptors which spread to the surface membrane outside of the NMJ and a change in receptor subunit type from ε to γ, which represent an immature form through which potassium efflux is magnified.[16,17] Rhabdomyolysis is a breakdown in skeletal muscle membrane function leading to loss of cell contents including potassium and creatinine kinase (CK), and has been found in one study to result in a higher chance of unsuccessful resuscitation and thus conveys a greater risk than that related to receptor upregulation.[16]

Our patient was 30 days post-injury and still showed clinical evidence of hyperkalemia despite normal pre-operative potassium levels. His CK was only mildly elevated, reaching a peak of 1,194 U/L, early in his course and was resolved by the time of our encounter. Although the precise timeframe and duration of the risk period for hyperkalemia are 7-10 days, the upregulation of receptors can persist as long as the underlying condition that produced it continues to exist.[9,15] Furthermore, recovery of muscle dysfunction can be delayed as long as one to five years following critical illness requiring an ICU stay.[18,19] It is important to note, however, that our patient had received succinylcholine during induction of anesthesia four days prior to our event with no noted complications. In light of this, we felt reasonably confident that the patient had surpassed the window of susceptibility and thus could be administered succinylcholine safely.

Also unique to our case was the very rapid recovery experienced, with just nine minutes of advanced cardiovascular life support (ACLS) performed before return of spontaneous circulation. Succinylcholine-induced hyperkalemia often lasts 10-15 minutes,[20,21] however, some have a significantly longer (up to 90 minutes) timeframe before successful recovery.[22] The reasons behind the high inter-individual differences in recovery time remains unclear. Basic science studies have postulated this variability to the distribution of acetylcholine receptor and the degree of their mRNA expression,[23] with a higher number of receptors distributed throughout more muscle tissue resulting in more long-lasting and profound hyperkalemia. Other theories speculate about differences in AChR isomer activity, specifically the α7AChR, exhibiting a continuous potassium leak leading to the persistence of cell depolarization and a longer timeframe for recovery.[24]

A search of the National Anesthesia Clinical Outcomes Registry revealed the incidence of cardiac arrest associated with anesthesia to be 5.6 per 10,000 cases.[25] The incidence of hyperkalemia in hospitalized patients is estimated between 1 and 10 in 100 admitted patients.[26] While hyperkalemia-induced cardiac arrest remains infrequent, cardiac arrest in the perioperative period carries a significantly higher (30.5-80%) survivorship compared to inpatient arrest (10%) in other areas of the hospital.[27] This can likely be explained by the unique milieu in which anesthesiologists encounter such circumstances. In contrast to patients on the wards, those in the OR are constantly monitored, with real-time information available regarding changes in EKG rhythm, end-tidal CO_2, respiratory pattern, and circulatory status. All of this allows for near immediate feedback of treatment success or failure and thus significantly higher ACLS survivorship. Of note, this case highlights the importance of appropriate consideration of vascular access before induction of anesthesia. Our patient had a 24-gauge peripheral IV as his only access upon arrival to the OR. His resuscitation was complicated by the infiltration and loss of this route during ACLS, which could have greatly affected the outcome of our interventions.

In conclusion, EKG changes, in association with a patient at risk

for hyperkalemia, demands immediate recognition by the provider. Anesthesia-centered ACLS and prompt treatment utilizing additional care teams is paramount to successful intra-operative resuscitation. With the recent approval and increasing availability of sugammadex as a reversal agent for rocuronium, the use of succinylcholine is likely to decrease. However, anesthesiologists must remain vigilant in the perioperative period to achieve positive outcomes in even the most critical situations.

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