

Etiology and Treatment of Intraoperative Hyperkalemia During Posterior Spinal Fusion in an Adolescent

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Abstract

Hyperkalemia, defined as a serum or plasma potassium greater than 5.5 mEq/L, while an uncommon occurrence in children, is a serious medical problem that warrants immediate attention as it can result in serious cardiac arrhythmias and death. Although hyperkalemia may occur in the critically ill patient or in the setting of renal failure and insufficiency, there are limited reports of its occurrence during intraoperative care. The authors report a previously healthy, 18-year-old patient, who developed hyperkalemia intraoperatively during posterior spinal fusion to treat adolescent idiopathic scoliosis. The potential etiologies of hyperkalemia are reviewed, a differential diagnosis for the possible etiologies presented, and treatment modalities discussed.

Keywords: Hyperkalemia; Potassium; Posterior spinal fusion

Introduction

Given the potentially lethal effects on the myocardium including dysrhythmias, hyperkalemia (serum potassium ≥ 5.5 mEq/L) is considered a medical emergency that warrants prompt evaluation and treatment [1, 2]. Regulated by various physiologic factors, the body maintains high intracellular levels of potassium required for cellular function such as growth and metabolism along with low extracellular levels to maintain a stable electrical gradient across cell membranes. This homeostasis can be disrupted by pathologic processes or events

resulting in hyperkalemia. Although rarely encountered during intraoperative care, the potential impact of hyperkalemia on outcome has been demonstrated as hyperkalemia from the rapid administration of transfused blood products, one of the most common identifiable cardiovascular causes of pediatric perioperative cardiac arrest [3]. We report a previously healthy, 18-year-old patient, who developed hyperkalemia intraoperatively during posterior spinal fusion to treat adolescent idiopathic scoliosis (AIS). The potential etiologies of hyperkalemia are reviewed, a differential diagnosis for the possible etiologies presented, and treatment modalities discussed.

Case Report

Review of this case and presentation in this format was in accordance with the guidelines of the Institutional Review Board of Nationwide Children's Hospital (Columbus, OH).

The patient was an 18-year-old, 63.8-kg previously healthy male who presented for posterior spinal fusion (T7 - 12) for correction of AIS. He was first evaluated for AIS 4 months prior to surgery at which point posterior spinal fusion was recommended due to the severity of the curve. He denied systemic signs or symptoms. Magnetic resonance imaging demonstrated severe scoliosis (62°) S-shaped rotary scoliosis with the dextro-scoliotic apex at T7 - 8 and the levo-scoliotic apex at L1 - 2. There was no evidence of intraspinal pathology and no canal or foraminal stenosis.

On the day of the procedure, the patient was held *nil per os* for 6 h. Preoperative vitals were normal with the patient being afebrile with a normal heart rate and blood pressure (BP) of 132/57 mm Hg. Preoperative laboratory analyses, obtained 2 weeks prior to surgery, were normal (sodium 141 mEq/L, potassium 4.5 mEq/L, blood urea nitrogen 15 mg/dL, and creatinine 0.96 mg/dL). The patient was transported to the operating room and standard American Society of Anesthesiologists' monitors were placed. After the inhalation of 50% nitrous oxide in oxygen, a peripheral intravenous cannula was placed. Anesthesia was induced with propofol (150 mg), sufentanil (30 μ g), lidocaine (60 mg), ketamine (30 mg), and methadone (7.1 mg). Neuromuscular blockade was achieved with rocuronium (40 mg) to facilitate direct laryngoscopy with a Macintosh 3 laryngoscope and endotracheal intubation with an 8.0-mm cuffed endotracheal tube. A radial arterial cannula and a second peripheral intravenous cannula were placed. Maintenance anesthesia included desflurane (titrated to main-

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Table 1. Intraoperative Laboratory Results and Clinical Care

Point-of-care testing results (normal values)	8:57 am	10:24 am	11:23 am	11:46 am	12:16 pm	1:09 pm
pH	7.37	7.39	7.39	7.46	7.47	7.43
PaCO ₂ (mm Hg)	42	40	38	36	34	36
Base deficit/excess	-1	-1	-2	+2	0	0
Hemoglobin (13.5 - 17.5 g/dL)	12.6	12.9	12.6	12.2	12.9	12.9
Ionized calcium (1.22 - 1.35 mEq/L)	1.14	1.18	1.18	1.10	1.17	1.19
Sodium (135 - 145 mEq/L)	140	136	134	138	138	139
Potassium (3.7 - 5.3 mEq/L)	3.9	5.3	5.9	5.7	5.3	4.7

Laboratory results from arterial blood. After the results at 10:24 am, minute ventilation was increased by 20%. After the results at 11:23 am, the following steps were taken: esmolol infusion discontinued, Normosol® changed to 0.9% normal saline, sodium bicarbonate administered (50 mEq), furosemide (10 mg) administered, and the inhalational agent (desflurane) was discontinued and propofol started. It was verified that none of the infusions had inadvertently been mixed in high potassium containing fluids.

tain the bispectral index at 50 - 60), sufentanil infusion (0.1 - 0.5 µg/kg/h), lidocaine 30 (µg/kg/min), ketamine (0.25 mg/kg/h), and esmolol 5 - 35 (µg/kg/h). A tranexamic acid infusion (50 mg/kg bolus followed by 5 mg/kg/h) and intraoperative cell saver were used to limit the need for allogeneic blood products. Intravenous fluids were provided using Normosol®. Arterial blood gases, electrolytes, and hemoglobin were analyzed at regular intervals during the course of the procedure using point-of-care testing (POCT). The initial POCT at 8:56 am showed a normal pH 7.37, sodium 140 mEq/L, hemoglobin 12.6 g/dL and potassium of 3.9 mEq/L. The surgical procedure proceeded uneventfully without excessive blood loss (total estimated blood loss for the case was 200 mL) or hemodynamic instability. Report POCT testing performed at 10:24 am was remarkable for an increase of the potassium level to 5.3 mEq/L (Table 1). No change in the electrocardiogram (ECG) was noted. Given the unexplained increase in the serum potassium, the minute ventilation was increased by approximately 20% and a repeat serum potassium was obtained in 1 h. At 11:22 am, the serum potassium was 5.9 mEq/L. At this point, the esmolol infusion was discontinued, the Normosol® was discontinued and 0.9% normal saline was used for intraoperative fluid replacement. Sodium bicarbonate (50 mEq) was administered to treat a base deficit of -3 and furosemide (10 mg) administered intravenously. The furosemide resulted in an increase in urine output to 100 - 250 mL/h over the next 2 - 3 h. Due to the concern for an occult myopathy and rhabdomyolysis, the inhalational anesthetic agent was discontinued and total intravenous anesthesia with a propofol infusion (75 - 100 µg/kg/min) titrated to maintain the bispectral index at 50 - 60 [4]. Throughout this time, no change in the ECG was noted. Pharmacy was contacted to verify there had been no inadvertent use of concentrated potassium solutions to mix the intraoperative infusions. No high potassium concentration solutions are available in the operating room pharmacy. Dissection of the paraspinal muscles and exposure of the spine was completed at 10:45 am. POCT at 11:46 am showed that the potassium had decreased to 5.7 mEq/L, and then to 4.7 mEq/L at 1:09 pm. At the conclusion of the surgical procedure, the patient's trachea was extubated, and he was transferred to the post-anesthesia care unit. On postoperative day 1, the serum potassium was

3.6 mEq/L. Creatinine phosphokinase was mildly elevated at 848 U/L (range of 36 - 289 U/L), which was attributed to the surgical dissection. The remainder of his postoperative course was uneventful.

Discussion

Given the potential for morbidity related to its effects on cardiac condition, acute intraoperative hyperkalemia requires immediate investigation and treatment. Hyperkalemia may result from various mechanisms or pathologic processes including excessive potassium intake or administration, increased endogenous production (rhabdomyolysis), decreased renal excretion, and intracellular to extracellular shifts (Table 2). Our 18-year-old male patient undergoing posterior spinal fusion developed moderate hyperkalemia of unknown etiology, which progressed during the early stages of the surgical procedure. Our initial approach focused on attempts to identify potential etiologies of the hyperkalemia as well as interventions aimed at decreasing the serum potassium concentrations.

In general practice, there are a myriad of potential causes for hyperkalemia. The majority of these are generally apparent based on the history and a review of the patient's comorbid conditions. However, in our patient, a more thorough investigation was necessary as there was no apparent etiology. Spurious hyperkalemia is a false elevation of the serum potassium due to a variety of possible causes. In clinical practice, hemolysis of the sample during drawing the blood, placing it into the specimen tube or its subsequent handling can result in hemolysis and a falsely elevated potassium level [5, 6]. If such a scenario is considered possible, it may be appropriate to repeat the value prior to investigation and treatment. However, delays in treatment should not occur while waiting for the repeat value to be processed. In our patient, we considered that there were no comorbid conditions which would explain the hyperkalemia, no ECG changes and therefore a repeat value was obtained.

Once the presence of hyperkalemia was documented by repeating the value using another point-of-care device, further treatment was initiated based on the potential etiologies for the hyperkalemia. This included an investigation into the potential

Table 2. Potential Etiologies of Hyperkalemia

Spurious
Hemolysis
Exogenous administration
Medications (antibiotics: penicillin)
Parenteral nutrition
Intravenous fluids (lactated ringers, Normosol [®] , Plasmalyte [®])
Blood and blood products
Cardioplegia solution
Medication error (diluent)
Intracellular-extracellular shift
Acidosis
Beta adrenergic blockade
Increased production
Malignant hyperthermia
Rhabdomyolysis
Tumor lysis
Hemolysis (cell saver, cardiopulmonary bypass)
Succinylcholine
Decreased excretion
Decreased cardiac output
Medications (aldosterone antagonist, ACE inhibitor)
Renal insufficiency
Adrenal failure or insufficiency
Hydrocortisone
Aldosterone

ACE: angiotensin-converting enzyme.

exogenous administration of potassium (Table 1). In the intensive care unit (ICU) setting, high potassium concentrations (parenteral nutrition) may be in place to replace ongoing losses. In this setting, alterations in renal function and decreased potassium elimination may result in hyperkalemia. When hyperkalemia is noted, all exogenous sources of administration or the use of medications which limit potassium excretion should be stopped. Particular attention should be directed toward investigating potential sources of potassium including blood and blood products, medications, intravenous fluids, parenteral nutrition, cardioplegia administration, medication diluents and infusion solutions, and cell saver fluids [7]. We communicated with our pharmacist and ensured that none of the infusions for intraoperative care were prepared incorrectly and contained potassium. The isotonic fluid, Normosol[®], which we routinely use for fluid administration and resuscitation during such procedures, was changed to 0.9% normal saline. Although the amount of potassium in Normosol[®] is minimal (5 mEq/L), the intraoperative fluids were switched to 0.9% normal saline.

Other potential etiologies that were considered included increased production from occult rhabdomyolysis related to an undiagnosed myopathy or drug-induced hyperkalemia (Table

2). Malignant hyperthermia was rapidly ruled out by the lack of associated clinical findings including elevated body temperature, tachycardia, and hypercarbia [8]. Although the primary diagnosis in our patient was idiopathic scoliosis, skeletal deformities including scoliosis are common in patients with neuromyopathic conditions [9]. In patients with neuromyopathic conditions, various factors may result in hyperkalemia. Most commonly, hyperkalemia has been reported following the administration of the neuromuscular blocking agent, succinylcholine [10, 11]. However, with increased knowledge of the risks related to succinylcholine in this patient population and the general surgical population in general, its use is now known to be contraindicated in muscular dystrophy patients and its use restricted to emergent or urgent airway management scenarios. Aside from succinylcholine-induced rhabdomyolysis and hyperkalemia, prolonged exposure to the volatile anesthetic agents may destabilize the sarcolemma in patients with myopathic conditions. Although this association has been questioned and some authorities still opine that volatile anesthetic agents can be safely used in patients with muscular dystrophies, this potential etiology was considered in our patient and the anesthetic care was switched from desflurane to propofol [10, 12].

Table 3. Treatment of Hyperkalemia

Resuscitation as needed according to guidelines
Consider extracorporeal support for refractory hemodynamic instability
Administration of calcium gluconate or chloride if ECG changes are noted
Enhanced extracellular-to-intracellular shift
Increase pH (hyperventilation or administration of sodium bicarbonate)
Glucose and insulin
Beta adrenergic agonists
Inhaled albuterol
Intravenous epinephrine or terbutaline
Enhanced elimination
Hydrocortisone in setting of adrenal insufficiency
Loop diuretics
Kayexalate
Hemodialysis or renal replacement therapies

ECG: electrocardiogram.

Drug-induced hyperkalemia should also be ruled out when evaluating a patient with hyperkalemia. Various medications may lead to hyperkalemia through various physiologic processes through inducing an intracellular-to-extracellular shift, by inhibiting renal excretion of potassium through inhibition of the renin-angiotensin-aldosterone system, or by decreasing glomerular filtration rate [13]. We would recommend a thorough review and consideration of each medication as anecdotal reports have implicated relatively unlikely medications such as ketorolac [14]. Anecdotal reports exist regarding various beta-adrenergic antagonists causing an elevation of the serum potassium by causing an intracellular-to-extracellular shift or through effects on the renin-angiotensin-aldosterone pathway, especially in patients with altered renal function [15, 16]. This effect has been reported with both cardio-selective and non-selective agents. Given these concerns, the esmolol infusion, which was being used as part of our intraoperative anesthetic regimen, was discontinued [17].

Hyperkalemia has been reported anecdotally related to muscle injury following dissection of the paraspinal muscles during spinal fusion [18]. Das et al reported hyperkalemia (serum potassium of 5.9 mEq/L) with ECG changes in a 10-year-old girl during laminoplasty from T4 to S1 [18]. The authors postulated that damage to the paraspinal muscles during surgical retraction led to muscle damage and release of potassium. In our patient, the dissection was similar to other cases and extensive muscle damage was not thought to be the etiologic event. Additionally, a creatinine phosphokinase value obtained on postoperative day 1, although elevated, was not severely elevated as is usually seen in muscle injury severe enough to cause rhabdomyolysis and hyperkalemia [19].

Etiologies such as adrenal insufficiency with aldosterone or hydrocortisone deficiency as well as decreased cardiac output based on a lack of past history and maintenance of adequate hemodynamic stability during intraoperative care. Although there was not an extensive workup to evaluate renal

function, blood urea nitrogen and creatinine were normal, and the intraoperative urine output was adequate.

The treatment pathway of hyperkalemia and its urgency are dictated by the patient's clinical status and changes in the ECG. ECG manifestations of hyperkalemia include peaked T waves, widening of QRS complex, heart block, followed by the terminal rhythms of a sine wave and ventricular fibrillation or asystole. Given the potential for rapid and significant deterioration related to hyperkalemia, a treatment algorithm has been provided by the Society for Pediatric Anesthesia (PediCrisis, Critical Events Cards) which is available on their website or app for hand-held electronic devices.

Patients with ECG changes require the administration of calcium (calcium chloride or calcium gluconate) to stabilize the electrical conduction system of the myocardium. Other treatment options that are effective in decreasing the serum potassium through an extracellular-to-intracellular shift or increased elimination are outlined in Table 3. One unique option available from pharmacy in our hospital combines sodium acetate to raise the pH, calcium gluconate to prevent dysrhythmias, and glucose with insulin to enhance intracellular movement of potassium into one solution. In our patient, the first intervention was to raise the pH with modest hyperventilation and administer sodium bicarbonate. In patients receiving mechanical ventilation, hyperventilation is a reliable, rapid, and easy maneuver to increase pH and lower serum potassium. Additionally, factors which may have caused the hyperkalemia were addressed by changing from desflurane to propofol, stopping Normosol[®], and discontinuing the esmolol. This was followed by the administration of furosemide to enhance renal excretion of potassium. These interventions resulted in a slow decline of the serum potassium concentrations over the ensuing 3 - 4 h. Therefore, more aggressive therapies as illustrated in Table 3 to lower serum potassium were not necessary.

In summary, we present an 18-year-old, previously healthy male who developed intraoperative hyperkalemia. Given its ef-

ffects on the cardiac conduction system, hyperkalemia is recognized as a medical emergency that frequently requires immediate intervention. Treatment starts with ensuring that all exogenous sources of potassium administration are discontinued followed by treatment of comorbid conditions, which may be accelerating potassium production. Potential drug-induced etiologies should be identified. This is followed by therapy to maintain hemodynamic stability, prevent the effects of hyperkalemia on cardiac conduction, and lower extracellular serum potassium concentrations. In our patient, no specific etiology was identified although the two primary causes were thought to be either the effects of esmolol on the intracellular-to-extracellular shift of potassium or potassium release related to surgical dissection of the paraspinal muscles. Even without a definitive diagnosis, effective therapy resulted in a prompt control of serum potassium values.

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Financial Disclosure

None to declare.

Conflict of Interest

None to declare.

Informed Consent

In accordance with IRB of Nationwide Children's Hospital, need for documentation of informed consent was waived.

Author Contributions

RL reviewed the case, prepared the initial and subsequent drafts. AC performed the patient care and reviewed the final draft. JT generated the concept, performed the patient care and reviewed all the drafts. WS and AB performed the patient care and reviewed the final draft.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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