Masseter Muscle Rigidity, Elevated Creatine Kinase, and Rhabdomyolysis Following Succinylcholine Administration: A Case Report

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This case report details the onset of masseter muscle rigidity, elevated creatine kinase levels, and rhabdomyolysis following a sevoflurane mask induction and succinylcholine administration in a 12-year-old boy. The patient had no family or personal history of neuromuscular disease or malignant hyperthermia. Hyperkalemia, metabolic acidosis, and rhabdomyolysis occurred within 75 minutes of masseter muscle rigidity. Subsequent to this event, it was recommended that the patient undergo a workup for neuromuscular disease and malignant hyperthermia with muscle biopsy. Until this workup is completed, the family should advise anesthesia providers that the patient is "malignant hyperthermia susceptible." Masseter muscle rigidity, elevated creatine kinase levels, and rhabdomyolysis will be thoroughly discussed in this article.

Keywords: Elevated creatine kinase, masseter muscle rigidity, rhabdomyolysis, succinylcholine.

Succinylcholine (SCh) is administered to facilitate airway management and is associated with many well-documented adverse effects. This case report outlines the development of masseter muscle rigidity (MMR), elevated creatine kinase (CK) levels, and rhabdomyolysis developing subsequent to the administration of SCh. On examination of the contemporary literature no case reports were found that illustrate the development of all 3 of these complications in a single patient following the administration of SCh. A case report by Shaaban et al[1] reports the occurrence of rhabdomyolysis and elevated CK levels subsequent to the administration of SCh in a 9-year-old child. Pedrozzi et al[2] discuss rhabdomyolysis after SCh was given to 2 otherwise healthy young boys. Flewellen and Nelson[3] report MMR induced by SCh in 6 boys. Comprehensive discussion of these occurring together is both intriguing and important in detailing appropriate perioperative management.

Case Report

A 12-year-old boy was scheduled for anterior spinal release of L1 to L4 vertebrae with posterior spinal fusion of L1 through L4 vertebrae with osteotomies, instrumentation, and autograft. His medical history included a diagnosis of Scheuermann disease (congenital failure of segmentation at L1 through L4 vertebrae), scoliosis, asthma, and encopresis. Following the preoperative evaluation he was assigned an ASA II physical status classification. His asthma was well controlled with the daily administration of salmeterol. His medication also included acetaminophen-oxycodone (325 mg/5 mg), 1 to 2 tablets every 6 hours as needed; hydroxyzine, 25 mg, 1 to 2 capsules every 4 hours as needed; docusate twice a day as needed; miralax twice a day; and vitamin C twice a day. This child had no previous anesthetics and no known family history of malignant hyperthermia (MH). His airway examination revealed a Mallampati score of III out of IV, a thyromental distance of 3 fingerbreadths, full range of neck motion, and positive mandibular subluxation. There was the potential for a difficult airway. The patient's weight was 55.8 kg; height, 151.2 cm; temperature, 36.5°C; blood pressure, 150/80 mm Hg; pulse, 90/min; and respiratory rate, 20/min. His preoperative laboratory work did not reveal dehydration from encopresis. The patient had nothing by mouth for more than 8 hours before surgery. He was an active boy and participated in basketball and football with some back pain. He did not have any weakness or paresthesia in his upper or lower extremities. A pulmonary function test was not done preoperatively, so restrictive lung disease could not be ruled out. All other systems were within normal limits. He had donated 3 units of autologous blood preoperatively. The table presents the preoperative laboratory results. The patient's parents consented to general endotracheal anesthesia, the insertion of peripheral intravenous (IV) lines, an arterial line, an intraoperative wake-up test, and the likelihood for intraoperative blood transfusion.

The patient was taken to the operating room, and following the placement of standard monitors, a mask induction with 30% oxygen, 70% nitrous oxide, and 6.6% inspired sevoflurane was accomplished. Following a loss of lid reflex, a 20-gauge, 1.25-in, left hand peripheral IV was inserted, followed by the administration of propofol. Mask ventilation was easily established and 60 mg of SCh was given (1 mg/kg). Heart rate increased to 110/min and

blood pressure decreased to 100/45 mm Hg. If we were unable to intubate, our plan was to perform an asleep fiberoptic intubation with spontaneous respirations following the recovery from SCh. It was previously demonstrated that we could easily mask ventilate this patient. We chose SCh because muscle function and strength returns within 10 to 15 minutes in patients with normal plasma cholinesterase.[4] A transexamic acid bolus of 100 mg/kg was initiated and infused over 20 minutes. Following mild fasciculations, masseter muscle rigidity (MMR) was noted with manual manipulation of the jaw. The patient's mouth opening was restricted to approximately 2.5 cm. Direct laryngoscopy was done revealing a grade 2 view with cricoid pressure. Endotracheal intubation was successful with the first attempt, and placement was verified with positive end-tidal carbon dioxide and equal bilateral breath sounds. A 20-gauge, 1.25-in, right radial arterial line; a 16-gauge, 1.25-in, right wrist peripheral IV; and a 22-gauge, 1.25-in, right wrist peripheral IV were started. The patient was positioned in the right lateral decubitus position. Remifentanil and transexamic acid infusions were initiated at 0.25 µg/kg per minute and 560 mg/h, respectively, along with isoflurane, nitrous oxide, and oxygen, providing a final inspired concentration of 0.8%. Mechanical ventilation was established using a fractional concentration of oxygen in inspired gas of 29%, volume control with a tidal volume of 538 mL, respiratory rate of 10/min, and peak inspiratory pressure of 29 mm Hg. End-tidal carbon dioxide level was 42 mm Hg. Motor evoked potential and somatosensory evoked potential monitoring was initiated and continued throughout the surgical procedure.

Baseline laboratory values were obtained at 9:12 AM, within 1 hour of anesthetic induction (see Table). Seventy-five minutes following the onset of MMR, orange urine was visible in the urinary catheter tubing. All lactated Ringer's solution IVs were replaced with 0.9% normal saline (NS). We believed that rhabdomyolysis had developed following SCh administration and the noted appearance of MMR. A fluid bolus of 1,000 mL of NS was given and warmed at 41°C. Mechanical ventilation was increased from 10/min to 14/min because of metabolic acidosis. Blood and urine samples were obtained for an analysis of CK, myoglobin, and arterial blood gas (see Table).

The anesthetic team discussed discontinuing isoflurane and initiating a propofol infusion eliminating known triggers of malignant hyperthermia. At this point, the end-tidal carbon dioxide was 35 and the patient's core temperature was 36.9°C. The initial anesthetic and uninterrupted monitoring for early signs of MH were continued. Three hours later, the CK had more than doubled and was 30,569 U/L. The alkalinization of the urine with a sodium bicarbonate infusion and the administration of mannitol and/or lasix were considered to enhance urinary output in light of myoglobinuria. The literature is unclear as to which therapy (alkalinization of the urine vs administration of diuretics) helps prevent renal failure.[5-7] The gold standard is to give large amounts of fluid.[5] After the orange urine appeared, 2,400 mL of 0.9% NS and 750 mL of 5% albumin were given over the next 3 hours. Urine output increased to 300 mL/h or more.

During the posterior fusion, the surgeon requested a mean arterial pressure of 55 mm Hg to 60 mm Hg. The remifentanil infusion was increased to 0.75 µg/kg per minute. The mean arterial pressure was greater than 60 mm Hg so a nitroprusside infusion was initiated at 0.15 µg/kg per minute and titrated to maintain a mean arterial pressure of 55 mm Hg to 60 mm Hg. Potassium, hemoglobin, hematocrit, lactic acid, and arterial blood gases were drawn repeatedly throughout the perioperative period (see Table). Intravenous fluids were aggressively administered to prevent renal failure due to the myoglobinuria. The last set of laboratory work in the operating room revealed a potassium level of 4.2 mEq/L, hemoglobin level of 8.8 g/dL, and hematocrit level of 27% with worsening metabolic acidosis (see Table). The hematocrit/hemoglobin levels had dropped because of aggressive fluid administration (dilutional effect) and blood loss. One unit of autologous blood was infused via gravity to increase oxygen carrying capacity. The higher the hemoglobin level, the more oxygen is bound to it. With metabolic acidosis, the hemoglobin-oxygen dissociation curve shifts to the right displacing oxygen from the hemoglobin and increasing the amount available to the tissues or organs.[8] Total fluids administered throughout the case were 5,500 mL of crystalloid, 1,000 mL of 5% albumin, and 1 unit of packed red blood cells for an estimated blood loss of 250 mL to 300 mL. The urine output was 1,425 mL. Estimated blood loss tends to be underestimated for these cases because of continuous oozing from the surgical site, length of case, and the amount of laparotomy sponges used. Laparotomy sponges are not weighed to get an accurate estimated blood loss at our institution. At the conclusion of the surgical procedure the patient was transferred to the pediatric intensive care unit, intubated, and sedated with a propofol infusion.

At 6:00 PM, 45 mEq of sodium bicarbonate was given over 20 minutes in the pediatric intensive care unit for continued metabolic acidosis. Serial CKs were drawn and peaked at 49,247 U/L, 20.5 hours following MMR. The CKs finally trended downward 22 hours after the initial CK results. The ionized calcium level peaked at 5.1 mg/dL and the serum urea nitrogen and creatine levels remained stable at 7 mg/dL and 0.5 mg/dL, respectively.

Because of the continued rise in CK, MMR, and continued rhabdomyolysis, we contacted the MH hotline the following morning. Because of the abnormal presentation of MMR, rhabdomyolysis, and increasing CK levels the following recommendations were made:

- Administer a single dantrolene dose of 2 mg/kg.

Laboratory results	Time	Sodium mEq/L	Potassium mEq/L	Chloride mEq/L	CO_2 mEq/L	BUN/ creatine mg/L
Preoperative results		140	3.6	103	23	12/0.7
Baseline results	9:12 AM	135	5.9			
Following 1,000 U NS bolus	9:56 AM	137	5.6	107		
Intraoperative results	1:00 PM		4.0			
Last set of intraoperative results	2:34 PM		4.2			
First set of labwork in PICU	3:50 PM	141	4.8	114		12/0.8

Laboratory results	Hgb/Hct	Ionized calcium mg/dL	Urinalysis	Lactic acid mEq/L	Creatine kinase U/L	Myo-globin ng/mL
Preoperative results	14.7/41%					
Baseline results	12.6/39%			3.8		
Following 1,000 U NS bolus	10.7/33%	4.1	+3 blood +2 protein	3.4	12,429	48,638
Intraoperative results	9.9/31%			4.1	30,569	
Last set of intraoperative results	8.8/27%			4.3		
First set of results in PICU	9.5/29%	5.1	+3 blood +1 protein	5.0	45,947	

Arterial blood gas readings	pH	Pco_2 mm Hg	Pao_2 Mm Hg	Base excess mEq/L	Bicarbonate mEq/L
Baseline results	7.33	44	141	–3	23
Following 1,000 U NS bolus	7.36	37	137	–4	20
Intraoperative results	7.35	35	146	–5	19
Last set of intraoperative results	7.33	35	141	–7	18
First set of results in PICU	7.25	40	124	–9	17

Table. Laboratory Results
BUN indicates serum urea nitrogen; PICU, pediatric intensive care unit; NS, normal saline; Hgb, hemoglobin; Hct, hematocrit.

- Obtain a full neuromuscular workup.
- Consider this patient to be "MH susceptible" (MHS) until a definitive MH diagnosis is made with a positive muscle biopsy.

Between 12 PM and 1:00 PM that day, 112 mg of dantrolene was given (2 mg/kg) as recommended by the MH hotline. The patient continued to have mild weakness in all 4 extremities, which did not change following the administration of dantrolene. The CK levels began to drop before the dantrolene was given and continued decreasing throughout the day. At 4:45 AM the following day, the CK level was 49,247 U/L; at 12:05 PM the CK level was 35,091 U/L; and by 9:10 PM the CK level was 29,601 U/L. Each dantrolene vial contained 20 mg of dantrolene and 3,000 mg of mannitol;[9] therefore, this patient received 112 mg of dantrolene and 16.8 g of mannitol. Although administration of mannitol remains controversial, the drug may have helped to protect the kidneys. One study postulated that NS preserved renal function more effectively than mannitol.[7]

The patient's parents were extensively questioned preoperatively and again postoperatively regarding preexisting muscular weakness and family history of neuromuscular disorders. Neither was present. The parents were strongly advised to have their son undergo an extensive neuromuscular workup on an outpatient basis. A positive diagnosis of neuromuscular disease would trigger an additional evaluation of immediate family members. They were also encouraged to have their son undergo a muscle biopsy for MH susceptibility. The parents were advised that SCh should be avoided in subsequent anesthetics in light of the occurrence of MMR and rhabdomyolysis after

receiving SCh. The patient remained in the care of the pediatric intensive care unit for 3 days and was transferred to the pediatric surgical floor for the remainder of his postoperative stay. The patient was discharged to home 7 days after his surgery without renal dysfunction.

Discussion

Succinylcholine is associated with MMR, elevated CK levels, rhabdomyolysis, and MH. Masseter muscle rigidity occurs when there is an increase in masseter muscle tone making it difficult to fully open the mouth. This can make oral endotracheal intubation very challenging. The incidence of MMR in the pediatric population following SCh administration with halothane is 1% and increases to 2.8% in children having strabismus surgery, but it decreases to 0.05% in children given SCh and a barbiturate.[10-15]

Fierobe et al[16] report the development of rhabdomyolysis in a 26-year-old man given SCh during nitrous oxide, oxygen, and isoflurane anesthesia. He was eventually diagnosed as MHS following a positive muscle biopsy. Gronert[17] states, "a positive biopsy is not enough evidence by itself; there must be undisputed evidence for clinical episodes of MH."

It is extremely important to know if this patient has an underlying neuromuscular disease and if he truly is MHS. Ideally, results from a full neuromuscular workup and muscle biopsy should be obtained before any future anesthetic. Subsequent anesthesia providers need to be apprised of the perioperative events of this case and the results of all follow-up evaluations. This patient should be strongly advised to obtain a medical alert bracelet stating "no SCh." Practitioners should avoid SCh in any patient with a history of MMR after receiving the drug.[18]

Manifestations of MH include hypercarbia, tachycardia, tachypnea, temperature elevation, hypertension, cardiac dysrhythmias, acidosis, hypoxemia, hyperkalemia, skeletal muscle rigidity, and myoglobinuria.[10] This patient exhibited MMR, followed by acidosis, hyperkalemia, and myoglobinuria.

There is generally a 20 to 30 minute interval between MMR and signs of MH.[13] A 1994 study reported an incidence of 59% of MH associated with MMR confirmed by a positive muscle biopsy. Of the MH patients, 12% developed clinical MH in less than 10 minutes following MMR.[19] Accordingly, some advocate abandoning the surgical procedure following the occurrence of MMR and treating the patient as MHS. They also recommend a muscle biopsy.[11] Others may abort the procedure and monitor the patient for signs of MH, send blood for serial CKs over 24 hours, and test the urine for myoglobinuria.[13] Berry and Lynch[12] suggest that the surgical procedure be continued, discontinuing known triggers of MH (inhalation agents) and proceeding with an intravenous technique, carefully monitoring for early signs of MH (tachycardia, increased end-tidal carbon dioxide). Gronert[14] suggests that the surgical procedure may continue if the end-tidal carbon dioxide, arterial blood gases, blood pressure, pulse rate, temperature, serum CK, urine color, and muscle tone are normal. Early signs of MH were not visible in this patient; therefore, isoflurane was continued.

A 1991 retrospective study examined patients who developed MMR following the administration of IV SCh. In all 68 pediatric patients (2.3 to 12 years old), inhaled anesthetics used for induction were continued following MMR. Most of the patients had either hypercarbia and/or metabolic acidosis along with elevated CK levels. The authors suggest that the anesthetic may be continued as long as careful monitoring accompanies diagnostic evaluation.[20] This patient exhibited metabolic acidosis and elevated CK levels; however, temperature, blood pressure, pulse rate, and the end-tidal carbon dioxide were in the normal physiological range. Accordingly, isoflurane administration was continued.

Anesthesia providers have been strongly urged to avoid SCh in the pediatric population unless indicated for the treatment of laryngospasm or to facilitate emergent airway management. In 1993, the US Food and Drug Administration (FDA) and Burroughs Wellcome & Company changed the SCh package inserts to read "Except when used for emergency tracheal intubation or in instances where immediate securing of the airway is necessary succinylcholine is contraindicated in children and adolescent patients…." After many letters and a public forum, the FDA changed this "contraindication" to a warning. The package insert was revised: "In infants and children, especially in boys under 8 years of age, the rare possibility of inducing life-threatening hyperkalemia in undiagnosed myopathies by the use of succinylcholine must be balanced against the risk of alternative means of securing the airway."[21]

An inadequate dose of SCh (less than the recommended dose of 1 mg/kg, IV), inadequate time for the onset of SCh, temporomandibular joint dysfunction, and myotonic syndrome may produce MMR.[10]

Creatine kinase is an enzyme that exists predominantly within skeletal muscle. The reference range is 22 U/L to 198 U/L. Elevated CK levels indicate muscle damage due to either a chronic disease state or an acute muscle injury. In rhabdomyolysis, or acute muscle breakdown, CK levels can reach 50,000 U/L to 200,000 U/L.[22] Increases in CK have been found to occur following both major and minor surgery, following MMR as in the present case, and following muscle fasiculations that accompany SCh administration. A recent study examined increases in CK after minor and major surgery in children. Succinylcholine was not used because of its known influence upon CK values. The results showed a median CK elevation (range) of 43 U/L in patients having major surgery.[23] This patient's CK level peaked at 49,247 U/L. A 2000 study showed CK reached a

maximum level of 1,339 U/L on day 2 following major surgery.[24] Levels have been shown to increase after the administration of SCh, but they increase even more with MMR. Serum CK levels generally peak 6 to 12 hours after MMR.[13] "If CK increases to several thousand or more, the response is decidedly abnormal and should be treated to prevent renal complications."[13] Studies have reported an increase in CK levels in pediatric patients who received IV SCh and halothane or enflurane,[25] sevoflurane or isoflurane.[26] A very recent case report describes a child receiving only sevoflurane who developed severe muscle rigidity, increased end-tidal carbon dioxide, and increased CK levels.[27] A rise in CK levels can be caused by MMR, rhabdomyolysis, SCh, or sevoflurane.

Rhabdomyolysis occurs following the breakdown of skeletal muscle fibers causing increased levels of potassium, phosphate, myoglobin, CK, and urate to be released into the circulation. High plasma levels of myoglobin can ultimately lead to renal damage due to renal tubular obstruction. An early sign of rhabdomyolysis is tea-colored urine. Confirmation of rhabdomyolysis is made with a positive serum or urine myoglobin.[5] Rhabdomyolysis can occur with the use of volatile agents and/or SCh in patients without a history of Duchenne muscular dystrophy.[28] Sauret and Wang[5] suggest giving 1.5 L of 0.9% NS per hour to maintain a urine output of 300 mL/h until myoglobinuria subsides. Some advocate alkalinizing the urine with sodium bicarbonate, while others think it may make hypocalcemia worse or precipitate calcium deposits within tissues.[6] Administration of mannitol remains controversial. One study postulated that NS preserved renal function more effectively than mannitol.[7]

Myoglobinuria occurs in 0.4% of pediatric patients following SCh administration.[29] Pedrossi et al[2] conducted a literature review in 1996 and found 66 pediatric patients had anesthesia-related rhabdomyolysis. Forty-three of these patients had received SCh. They also reported rhabdomyolysis in 2 children following SCh during a general anesthetic. Myoglobinuria was also reported in a healthy 9-year-old who received SCh, nitrous oxide, and isoflurane.[1]

In a recently published study, Flick et al[30] examined 274 medical records of children under the age of 21 who received volatile anesthetic agents, and only 3 received SCh during a muscle biopsy. They concluded that patients with a suspected neuromuscular disorder have a 1.09% or less risk of developing MH or rhabdomyolysis following exposure to a volatile anesthetic agent.

Patient positioning has also been associated with the development of rhabdomyolysis. There is an interesting case report of rhabdomyolysis occurring in an obese man positioned in the lateral decubitus position. The authors postulated that muscle necrosis and the ensuing rhabdomyolysis occurred from the direct and prolonged pressure upon the gluteal and flank muscles.[31]

Conclusion

In conclusion, this case report reveals MMR, elevated CK levels, and rhabdomyolysis developing following SCh administration in a 12-year-old boy. This case report is noteworthy because MMR, rhabdomyolysis, and elevated CK levels all appeared in this patient. Unfortunately, he has not had a full neuromuscular workup or a muscle biopsy done. He did have blood drawn for ryanodine receptor 1 mutation screening. This screening looks at certain parts of the ryanodine receptor 1 gene and detects only about 30% at risk for MH.[32] This patient's ryanodine receptor 1 mutation screening was in the reference range; therefore, MH still cannot be ruled out. This patient does have a medical alert bracelet indicating MHS. As anesthesia providers we can recommend and strongly encourage patients' parents to do the right tests but ultimately they may not do what is recommended. The knowledge of what occurred during this anesthetic is extremely important for the next anesthesia provider caring for this very intriguing patient.

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