Succinylcholine-induced Hyperkalemia in a Patient with Metastatic Rhabdomyosarcoma

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Severe hyperkalemia following administration of succinylcholine, resulting in cardiac arrhythmias and cardiac arrest, occurs in many conditions including burns, severe trauma, neurologic disease, and certain types of neuromuscular disorders. A feature common to many of these conditions is skeletal muscular involvement either due to direct trauma or muscle denervation. We report a case in which hyperkalemia and ventricular arrhythmias developed after administration of succinylcholine to a patient with extensive metastatic rhabdomyosarcoma.

REPORT OF A CASE

A 4-yr-old, 18-kg boy was admitted because of severe general malaise of 3 wk duration. On the day before admission he had developed pyrexia (38.9°C). Apart from an uneventful operation for strabismus at the age of 5 yr, there was no other relevant medical history. No details of the anesthetic given for that operation were available. Physical examination on admission showed an ill child. The only relevant finding was a temperature of 39.6°C. No focus of infection could be identified, nor was there any evidence of neurologic or muscular disorder. A radiologic skeletal survey showed widespread osteolytic areas in multiple areas. Nineteen days after admission, a tibial bone biopsy was scheduled under general anesthesia. Between admission and surgery, his temperature had remained elevated around 39°C. Prior to surgery the hemoglobin was 5.3 mmol/l (8.2 mg/dl), the hematocrit 0.27, the leukocyte count was 10.2 × 10⁹/l with 23% metamyelocytes. Serum electrolytes and creatinine were within normal limits. The potassium was 4.2 mmol/l and had varied between 3.4 and 4.2 mmol/l between admission and surgery. The lactate dehydrogenase (LDH), which had been 4,110 U/l on admission, had decreased to 1,960 U/l 3 days prior to surgery, with a distribution of LDH isoenzyme fractions 1–5 of 18%, 54%, 24%, 5%, and 0%, respectively. The normal range for LDH isoenzymes fractions in our laboratory is 1, 15–26%; 2, 34–44%; 3, 23–33%; 4, 5–12%; and 5, 1–7%, respectively. The distribution pattern in our patient is not characteristic for a particular type of tissue damage.

Premedication consisted of 40 mg of oral trimipramine. Anesthesia was uneventfully induced with halothane in O₂/N₂O. Monitoring in the induction room consisted of ECG registration and arterial blood pressure measurement by oscillometry. After induction of anesthesia, a 20-g iv cannula was inserted, and 0.05 mg of atropine and 20 mg of succinylcholine were given iv. The trachea was then intubated and normal breath sounds heard over both lungs. During induction the ECG showed a sinus tachycardia of 160 beats/min. Just before transport to the adjacent operating room, approximately 5 min after administration of the succinylcholine, the QRS complexes were noted to have widened, and shortly thereafter the ECG showed a ventricular tachycardia with a rate of 170 beats/min (fig. 1). The systolic arterial blood pressure was 70 mmHg. Nitrous oxide and halothane (at that moment the inspired halothane concentration was 1.5%) were discontinued, and ventilation was controlled with an FIO₂ of 1.0. Soon after, a ventricular flutter-like pattern developed, arterial pulsations became undetectable, and external cardiac massage was started. Lidocaine (20 mg iv) was administered. The ventricular flutter changed to ventricular fibrillation. At that moment the pupils were widely dilated. After an additional 20 mg of lidocaine iv and defibrillation with an energy of 100 J, the ventricular rhythm followed with a rate of 65 beats/min and detectable peripheral arterial pulsations. Atropine (0.05 mg iv) was given, the heart rate increased, and ventricular flutter again resulted with a rate of 260 beats/min. This reverted to a sinus tachycardia of 150 beats/min after synchronized cardioversion with an energy of 100 J. The pupils narrowed and shortly thereafter the patient regained consciousness.

An arterial blood sample obtained approximately 9 min after the administration of succinylcholine showed serum potassium 7.3 mm, pH 7.35, PaCO₂ 4.2 kPa (31.5 mmHg), PaO₂ 50.5 kPa (379 mmHg), bicarbonate 20 mm, base excess −5 mmol/l, and O₂ saturation 100%. An infusion of 100 ml glucose 10% containing 2 units of insulin was started. Because the vital signs remained stable and no new arrhythmias occurred, it was decided to proceed with the biopsy. Anesthesia was recommenced with O₂/N₂O/enflurane and spontaneous respiration via a Jackson Rees circuit. In addition to a bone biopsy, a muscle biopsy...
was also taken from the left tibialis anterior muscle because of the abnormal and unexpected response to succinylcholine. At the end of the operation the serum potassium was 3.5 mEq. Recovery from anesthesia was uneventful. In the recovery room the patient was fully alert and showed no clinical signs of cerebral damage.

Histologic examination of the muscle biopsy revealed only small and nonspecific myopathic changes. There was some variation in muscle fiber diameter with distributed atrophic fibers but no signs of extensive denervation.

Histologic examination of the bone biopsies from the left tibia revealed a malignant sarcomatous tumor, which, on its histologic morphology and by immunohistochemical investigation, was classified as an embryonal rhabdomyosarcoma. The tumor mass completely filled the bone marrow spaces and was partly necrotic. The primary source of the tumor was not determined.

DISCUSSION

Several possible factors could have contributed to the cardiac dysrhythmias and cardiac arrest in the patient. Halothane, used for induction of anesthesia, predisposes the myocardium to ventricular arrhythmias. The threshold for ventricular ectopic activity during halothane anesthesia is further lowered by atropine. We gave iv atropine to the patient immediately before administration of succinylcholine. Although the most common dysrhythmia observed with succinylcholine is bradycardia, ventricular dysrhythmias can also occur.¹

The initial ECG changes observed in the patient were characteristic for hyperkalemia, i.e., widening of the QRS complex with a decreased amplitude of the R wave, appearance of a prominent S wave, and depression of the ST-segment.² These changes were in keeping with a measured potassium concentration of 7.3 mEq immediately after resuscitation, an increase of 3 mEq above the preoperative level. It is unlikely that such an increase could have been caused by the resuscitation process. Indeed, there are clinical reports of hypokalemia after resuscitation from cardiac arrest.³ Martin et al.⁴ studied hyperkalemia during cardiac arrest and resuscitation in an experimental dog model. They found that the potassium concentration increased from a baseline value of 3.5 mEq to a maximum of 4.9 mEq 10 min after the start of resuscitation. However, in their model resuscitation was not started until 5 min after the initiation of cardiac arrest. It is our view that the primary factor responsible for the events observed in our patient was hyperkalemia induced

![Fig. 1. ECG recordings before induction of anesthesia (A) and 3 min (B) and 4 min (C) after administration of 20 mg of succinylcholine. Paper speed 25 mm/s. A. Sinus rhythm, heart rate 84 beats/min. B. Wide QRS complexes, absent P waves, heart rate 145 beats/min. C. Sudden transition to ventricular tachycardia.](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931366/)
by succinylcholine, and the risk of cardiac dysrhythmias would have been further increased by the simultaneous use of halothane.

In normal patients administration of succinylcholine causes a small, but clinically insignificant, increase in serum potassium seldom exceeding 0.5 mEq/L. By contrast, massive increases in serum potassium levels, often resulting in cardiac arrhythmias or even cardiac arrest, have been reported when succinylcholine was given to patients with burns, trauma, or neurologic disease. There was no evidence of any neurologic disorder in our patient. The mechanism of this succinylcholine-induced hyperkalemia is, in some cases, thought to be due to increased chemosensitivity of the muscle membrane resulting in a marked increase in potassium efflux induced by succinylcholine.

The reason for the severe hyperkalemia, resulting in cardiac arrhythmias, following administration of succinylcholine in our patient is not immediately obvious. He had been pyrexic for 3 wk prior to surgery, although no focus of infection could be found, and he had a normal leukocyte count at the time of surgery. Infection has been implicated as contributing to succinylcholine-induced hyperkalemia. However, although the mean increase in serum potassium after administration of succinylcholine in infected patients is usually not more than 20% greater than in control patients, an increase from 4.8 to 7.55 mEq/L has been reported. In our patient it is likely that the prolonged pyrexia was a response to extensive necrosis of widespread metastases from an embryonal rhabdomyosarcoma. We can only hypothesize that the combination of pyrexia, malaise, and as a consequence relative immobility, might have altered the sensitivity of his muscles to succinylcholine.

Another possibility is that the potassium efflux after administration of succinylcholine originated directly from the large metastatic tumor mass. We have been unable to find any reference in the literature to succinylcholine-induced hyperkalemia directly associated with muscle tumors. However, Cairoli et al. reported cardiac arrest after 100 mg of succinylcholine in an otherwise healthy patient who had received radiotherapy for sarcoma in one leg. They subsequently investigated the effect of radiation on succinylcholine-induced potassium efflux in rats and concluded that radiation could have been responsible for the cardiac arrest in their patient. At the time of surgery our patient had not received radiation therapy.

In conclusion, we have reported cardiac arrhythmias associated with hyperkalemia following administration of succinylcholine in a young patient with prolonged pyrexia and extensive metastatic embryonal rhabdomyosarcoma, anesthetized with halothane. Based on our experience, we would recommend caution in the use of succinylcholine in patients with either prolonged fever or with extensive muscle tumors.

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REFERENCES