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SUCCINYLCHOLINE CAUSING CARDIAC ARREST

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ABSTRACT

A case history of a 45 year old male is presented, who suffered a cardiac arrest following the administration of a single dose of succinylcholine. The arrest was associated with hyperkalemia and massive elevation of serum creatine phosphokinase. Asystole was prolonged and refractory to treatment, although cardiac activity was eventually restored. The possible cause of the circulatory collapse is discussed and reports of similar cases reviewed.

Key words: general anaesthesia, succinylcholine, cardiac arrest, hyperkalemia

CASE REPORT

A 45yr old male, weight 50 kg and height 152 cm, posted for emergency laprotomy in view of intestinal perforation. His medical history included gastroesophageal reflux, for which he was prescribed omeprazole. He was apparently asymptomatic on the medications, until he had a sudden onset of pain in abdomen with distension since past three days. After a thorough pre-anaesthetic evaluvation patient was planned for Epidural Anaesthesia and General Anaesthesia with rapid sequence induction. Preoperatively heart rate was 104 beats/min regular, arterial blood pressure 100/74 mm Hg, and oxygen saturation between 99 to 100% on room air. Rest of the physical examinations were unremarkable and the laboratory values were as follows: Hb-13.1, TLC-27410, Plt-2.5, LFTs wnl, electrolytes wnl (Na-134, K-4.2). Electrocardiogram, pulse oximeter, and non-invasive blood pressure monitors were attached. The epidural catheter was placed at the L1–L2 inter-vertebral space in lateral position, catheter was easily inserted and no blood, cerebrospinal fluid, or paresthesia was noted. Patient was given supine position and pre-oxygenation was done with 100% oxygen and induced with injection.

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Thiopentone 5 mg/kg and inj. Succinylcholine 2 mg/kg with sustained cricoid pressure. The lungs were manually ventilated with oxygen (FiO_2 1.0). During laryngoscopy, and prior to intubation, sinus bradycardia developed, and oximetry indicated progressive desaturation (SpO_2 100% to 74%) and a slowing of the heart rate. The laryngoscope was removed and manual ventilation with oxygen was continued with anaesthesia circuit. The patient's rhythm deteriorated from a sinus bradycardia of 50 beats/minute to asystole. Atropine 0.6 mg iv was given, the trachea was intubated with ET tube no.8 under direct laryngoscopy without any difficulty, manual ventilation with oxygen continued, and CPCR commenced. Over the course of the resuscitation the patient received total inj. epinephrine 3mg, bicarbonate 100 mEq, and calcium chloride 1 g, all in divided doses. Approximately 12 min after induction there was a return of circulation with sinus tachycardia. Several episodes of ventricular tachycardia and fibrillation were successfully treated with defibrillation and boluses of inj. xylocard. At the initial return of circulation, laboratory values were as follows: arterial blood gases, pH 7.16, $PaCO_2$ 35 mmHg, PaO_2 371 mmHg, HCO_3^- 12 mmol/L; Na 132 mmol/L, K 7.7 mmol/L, Cl 100 mmol/L; CPK 7,713 UI (normal 0-200). An additional 50 mEq of sodium bicarbonate and a glucose-insulin infusion were given. Repeat data following stabilization approximately 40 minutes after induction, revealed: pH 7.35, $PaCO_2$ 32 mmHg, PaO_2 306 mmHg, HCO_3^- 16 mmol/L; Na 136 mmol/L, K 5.5 mmol/L, CI 101 mmol/L,HCO_3^-16 mmol/L; and CPK > 10,000 U/L. Surgery was allowed to continue after a

certain period of a vigilant monitoring. Throughout the surgery vitals remained stable, with a heart rate varied between 68 to 88 beats/min, blood pressure from 100/70 to 140/88, and oxygen saturation between 99 to 100% on oxygen and nitrous oxide. After giving test dose, epidural infusion was started, which was continued postoperatively as well.The entire surgical procedure lasted for 1 hour and 15 minutes without any other intraoperative complications.

The patient was then transferred to the ICU in stable condition, where he was maintained on ventilatory support with sedation and paralyzation for the next 8 hrs. Throughout the ICU stay patient's vitals remained stable, and he was successfully weaned off from the ventilator. A postoperative cardiology consultation was advised in view of a suspicion of any underline cardiac disorder, which has ruled out of any such cause.

DISCUSSION

Succinylcholine is a depolarizing skeletal muscle relaxant and has been used for rapid sequence tracheal intubation since its introduction into clinical practice in Europe in 1951 and in the USA in 1952.[1]As does acetylcholine, it combines with the cholinergic receptors of the motor end plate to produce depolarization. This depolarization may be observed as fasciculations. Subsequent neuromuscular transmission is inhibited so long as adequate concentration of succinylcholine remains at the receptor site. It gained popularity for its quick Onset of flaccid paralysis (less than 1 minute after IV administration), and ultrashort duration of action with single administration lasts approximately 4 to 6 minutes. Succinylcholine came to rule the practice of anaesthesia and continues to do so even today. It is the gold standard against which the other muscle relaxants are compared.[2]

However, a number of clinical case reports have shown clearly that the use of scoline has been associated with a number of serious adverse effects and its use has declined since 1992 [3,4].

Rosenberg and Gronert have recently described four cases of sudden cardiac arrest following the induction of anaesthesia with succinylcholine. They postulated that these arrhythmias were caused by succinylcholine induced rhabdomyolysis and hyperkalaemia in the presence of an undiagnosed myopathy [5].

Suxamethonium (succinylcholine) has been known to produce a bradycardia in infants and young children (Leigh *et al*., 1957 Telford and Keats, 1957). Martin (1958) and Bullough (1959) have reported that repeated doses of suxamethonium caused not only a slowing of heart rate but also a disturbance of rhythm. It seemed important to confirm these findings, and to find out in greater detail whether this relaxant drug could cause in clinical practice a disturbance of rhythm sufficient to produce a circulatory collapse [6,7].

Our report showed that how succinylcholine caused cardiac arrest. The cause of asystole in this patient could be attributed to hyperkalemia resulting from generalized muscle injury induced by succinylcholine. Previous reports have also invoked this mechanism, but confirmation has been lacking, since in most instances electrolytes were not obtained in the acute situation. In our patient the serum potassium raised to 7.7 mmol/L. An acute increase of this magnitude explains the cause of the arrest and why it proved so refractory to treatment. In addition to the resuscitative measures employed, specific therapy to reduce the serum potassium levels, such as a glucose-insulin infusion, might have been helpful. Succinylcholine has been shown to be more likely to cause muscle injury, as evidenced by raised CPK and serum myoglobin levels, in paediatric patients [8].

CONCLUSION

The important implication arising from this case is that succinylcholine should be used cautiously in patients. The most serious complication - intraoperative cardiac arrest appears to be precipitated by acute hyperkalemia. The arrest may be prolonged and difficult to reverse.

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