A cardiac arrest following the administration of succinylcholine

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Abstract: A twenty-three year old woman, admitted to the intensive care unit (ICU) with a diagnosis of meningitis and associated lower limb ischemia suffered a cardiac arrest, due to extreme hyperkalemia, following the administration of succinylcholine in order to replace an endo-tracheal tube. After prolonged cardiopulmonary resuscitation (CPR) lasting 45 minutes, during which 8 mg of epinephrine was administered, cardiac output was restored. Four weeks later the patient left intensive care, having made a full recovery with no neurological deficit. The following case highlights the risks of succinylcholine in the septic patient and that prolonged resuscitation can have a successful outcome.

Key words: Succinylcholine; hyperkalemia; cardiac arrest.

A twenty-three year old female patient presented to the acute medical admission unit with a history of headache, neck stiffness and rash. A differential diagnosis of meningitis pre-empted a lumbar puncture which confirmed pneumococcal meningitis. This was treated with intravenous benzyl penicillin and cefotaxime. The development of septic shock and acute renal failure (ARF) lead to a rapid deterioration and the patient was admitted to ICU. The patient was intubated, ventilated and invasive monitoring was instituted. Severe hypotension was treated with infusions of epinephrine and norepinephrine. Renal replacement therapy was established by means of continuous veno-venous hemodiafiltration (CVVHDF).

During her admission, the patient developed discolouration and duskiness of the toes, feet and pretibial areas bilaterally (Fig. 1). A vascular surgical opinion was sought determining that there was superficial lower limb ischemia due to high doses of vasopressor that would require delayed surgical debridement.

Despite the continued need for renal support (CVVHDF) and invasive ventilation, inotropic and vasopressor requirements reduced over the following days. Ten days into her ICU stay the patient developed pseudomonas pneumonia, which led to the productive of thick tenacious sputum causing partial occlusion of the endotracheal tube (ETT). This necessitated a change of ETT, carried out using a rapid sequence induction (RSI). With CVVHDF continuing, the serum potassium was 3.6 mmol/l. With regards to the RSI, pre-oxygenation was done for three minutes, cricoid pressure was applied and propofol 150 mg and succinylcholine 100 mg were administered intravenously. At the time of this case no Glide scope was available however it is now used as standard in the ICU. The ETT was changed without difficulty. Shortly after intubation, the patient’s heart rate slowed to 20 beats per minute, with a normal blood pressure. Atropine was given in three 1mg boluses without effect and the patient then developed complete asystole.

Cardiopulmonary resuscitation (CPR) was rapidly commenced. During the prolonged resuscitation attempt, lasting 45 minutes in total, the patient was defibrillated three times. The first 1 mg dose of epinephrine was given after the third shock, with further 1 mg doses given every 5 minutes without interrupting CPR. A total of 8 mg of epinephrine, 2 g of calcium chloride and 50 mls of 8.4% sodium bicarbonate were given during the resuscitation. There was no return of spontaneous circulation during this time. Seeking potential reversible causes, an arterial blood sample was then taken which demonstrated the patient was hyperkalemic with a potassium level of 9.3 mmol/l. A further 2 g of calcium chloride was administered followed by 30 units of Actrapid insulin with 50% Dextrose. Spontaneous cardiac output was subsequently restored with a heart rate of 30 beats per minute. Atropine 1mg was given and the heart rate increased to 90 beats per minute in sinus rhythm.

The cardiac arrest was followed by an acute pulmonary oedema, which was confirmed using a chest x-ray. An arterial blood sample demonstrated
no clinical signs of anaphylaxis such as tachycardia, bronchospasm or rash. Although rocuronium could have been used, succinylcholine was administered as a result of the patient being a grade three intubation when she was intubated on admission to intensive care. Furthermore, the patient had a full stomach as she was enterally fed. Nowadays, sugammadex is readily available and would have made the usage of rocuronium more safe in case of a difficult intubation (6). Routine usage of tube exchanger or the glidescope was introduced in our unit following this incident to avoid the usage of succinylcholine and its side effects.

Hyperkalemia leading to cardiac arrest following the administration of succinylcholine has previously been reported (7). This extreme hyperkalemia has been reported in patients with burns, renal failure, muscle paralysis, sepsis, myositis and severe muscle trauma (8). The hyperkalemia results from an increase in the population of extra-junctional acetylcholine receptors throughout the muscle in these pathological conditions. At the molecular level, the extra-junctional acetylcholine receptors differ from normal junctional acetylcholine receptors in that the Є subunit of the junctional receptor is replaced by the γ subunit (9). Functionally they differ from junctional receptors in that they stay open for a longer time period upon activation by agonists such as acetylcholine or succinylcholine. This results in large quantities of potassium shifting from the myocyte into the plasma causing potentially dangerous and malignant hyperkalemia (10). Cooperman et al. went on to suggest that the degree of hyperkalemia is related to the degree and extent of muscle affected in cases of neuromuscular disease or nerve damage (4). Supporting Cooperman’s theory, the patient’s lower limb ischemia was extensive (Fig. 1), and correlated with the substantial rise in potassium levels.

Fig. 1. — Bilateral impending necrosis in the lower limbs due to septic emboli from a source of meningitis.

Patients surviving prolonged CPR have been reported before (11). In this patient a total of 45 minutes of CPR was performed during which a PaO₂ of 7.7 kPa (FiO₂ 1.0). A pulmonary artery catheter was inserted demonstrating a cardiac index of 2.1, systemic vascular resistance of 1800 dynes/cm², and a pulmonary capillary wedge pressure of 22 mmHg. A dobutamine infusion was commenced in order to reduce pulmonary vascular resistance and improve cardiac output. The patient went on to make a rapid recovery and improved dramatically over the next 24 hours. Oxygen requirements fell (FiO₂ 0.5) with spontaneous respiration and the dobutamine infusion was quickly weaned. After 39 days in ICU the patient made a good recovery, including normal renal function, however did require a left sided below knee amputation and amputation of the great and middle toes on the right foot at a later date. She made an otherwise full recovery and was later discharged home.

**DISCUSSION**

Succinylcholine is commonly used for intubating patients in emergency situations (1). It provides rapid and optimal muscle paralysis for intubation of the larynx and has a short duration of action (1). In support of Miller et al., the use of succinylcholine was desired due to its rapid onset of action; only matched by rocuronium at higher doses (2). However, succinylcholine does have well documented side effects including hyperkalemia, myalgia, increased intraocular and intracranial pressures and arrhythmias (3-5). Anaphylaxis is another side effect of succinylcholine, which however was not believed to be the cause of the cardiac arrest in this patient since succinylcholine had been used before in the same patient in ICU and secondly there were
large quantity of epinephrine was administered. It is our belief that the persistent hyperkalemia was the reason such large amounts of epinephrine were needed despite the early administration of calcium chloride and sodium bicarbonate. In retrospect an earlier arterial blood sample may have highlighted the hyperkalemia with earlier administration of insulin and dextrose.

The case also highlights that despite prolonged resuscitation, rapid institution of CPR with the maintenance of a cardiac output can lead to favourable outcomes and that resuscitation should continue until all reversible causes have been excluded.

CONCLUSION

The above case highlights the potentially lethal side-effect of hyperkalemia in patients receiving succinylcholine. In healthy individuals the average rise is thought to be approximately 0.5 mmol/l. The need to avoid its use in patients with neuromuscular disorders, paralysis and burns has been well described. Underlying myositis due to septic emboli from meningitis is likely to have been the origin of the massive potassium release in this patient. The use of succinylcholine should be questioned in those patients with limb ischemia and impending muscle necrosis, due to a potential source of massive potassium release and subsequent cardiac arrest.

References

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