Suxamethonium and Cardiac Arrest

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ABSTRACT

We report a case of cardiac arrest due to hyperkalaemia following administration of suxamethonium during a procedure to facilitate a change of endotracheal tube in a septic patient. The cause of this rare but fatal complication is briefly described and discussed. In view of this, suxamethonium should be used with great caution in patients with burns and other forms of physical injury, in a number of nervous system disorders, and in critically ill patients requiring prolonged ITU care.

Keywords: suxamethonium, hyperkalaemia, cardiac arrest

INTRODUCTION

Cardiac arrest due to administration of suxamethonium was first reported in a burns patient in 1958⁽¹⁾ and hyperkalaemia was reported to be responsible for this complication in 1967(2). It is now recognised that an increase in plasma potassium level following administration of suxamethonium may be greatly exaggerated in patients with burns (2,3) and with other forms of physical injury and in a number of nervous system disorders(5). We report a case of cardiac arrest due to hyperkalaemia following administration of suxamethonium in a septic patient.

CASE REPORT

A 57-year-old gentleman with known cerebral palsy underwent elective repair of hiatus hernia following failure of 10 years of conservative treatment to control upper gastrointestinal symptoms and recurrent bleeding. Surgical intervention consisting of a gastroplasty and Nissen fundoplication through a left thoracotomy was performed. The post-operative recovery was initially complicated with right chylothorax and later dehiscence of the suture-line of the left hemidiaphragm which resulted in herniation of the abdominal viscera and aspiration of the stomach content. The chylothorax was dealt with by ligation of the thoracic duct with video-assisted thoracoscopic surgery while the diaphragmatic dehiscence was re-repaired. As a result of respiratory complication, the patient required prolonged mechanical assisted-ventilation. On the twentieth day after the initial surgery, the endotracheal tube slipped out accidentally and in the process of reinserting the tube, suxamethonium 100 mg and propofol 160 mg were given. The patient suffered a cardiac arrest due to ventricular fibrillation moments after administration of the drugs and required advanced cardiopulmonary resuscitation (CPR). Serum potassium measured at the time of CPR was found to be profoundly elevated at 9.2 mmol/L. This was treated with a combination of intravenous glucose, insulin and bicarbonate. Unfortunately, the patient deteriorated rapidly following this and died two days later.

DISCUSSION

Klupp et al⁽⁶⁾ were attributed to being the first to demonstrate the increase in serum potassium following administration of suxamethonium in 1954. It usually occurs three to five minutes after intravenous injection of suxamethonium, and is raised by 0.5-1.0 mmol/L although an increase of up to 2 mmol/L have been reported. The increase of serum potassium level is not related to the dose of suxamethonium administered, ie, increasing the dose of suxamethonium has been found to increase serum potassium slightly or not at all⁽³⁾. The potassium originates from skeletal muscles and is thought to be liberated by depolarisation at the neuromuscular junction, causing movement of potassium across the post-synaptic membrane(7). It may also be released from muscle fibres damaged by the incoordinate contractions induced by suxamethonium, since raised blood levels of creatine kinase and myoglobin may occur following use of the drug⁽⁸⁾.

Cardiac arrest following administration of suxamethonium was first reported almost thirty years ago(1) and hyperkalaemia was reported to be responsible some 20 years ago(2). As discussed above, a small increase in serum potassium following administration of suxamethonium is often seen in most normal patients. However, this phenomenon may be greatly exaggerated in patients with burns⁽²⁾ and with other forms of physical injury(4), in a number of nervous system disorders(5), and in critically ill patients requiring prolonged ITU care⁽⁹⁾. For reasons not well understood, there is a proliferation of postsynaptic acetylcholine receptors beyond the area of the neuromuscular junction, beginning a few days after injury(10). Therefore, potassium movement across the muscle membrane is then no longer confined to the

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neuromuscular junction during depolarisation upon the administration of suxamethonium. This results in sudden and excessive increase of serum potassium, more than it does in normal subjects.

CONCLUSION

The lesson is that suxamethonium should be used with great caution in patients with burns, with other forms of physical injury, in a number of nervous system disorders, and in critically ill patients requiring prolonged ITU care.

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