Suxamethonium-induced hyperkalaemia in a patient with a normal potassium level before rapid-sequence intubation

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ABSTRACT

The use of suxamethonium (succinylcholine) for rapid-sequence intubation may be limited by hyperkalaemia. Modest pre-induction hyperkalaemia is usually disregarded. We present a patient who underwent emergency surgery for a perforated peptic ulcer after being bedbound for 26 days because of a head injury. Serum potassium level was 4.0 mmol/L. The patient was intubated after injection of sedative and suxamethonium and, about 3 minutes later, developed ventricular arrhythmia. Blood tests during resuscitation showed a serum potassium level of 8.8 mmol/L. Immobilisation, denervation and intra-abdominal infection were risk factors for hyperkalaemia in this patient. This report reinforces the need to identify risk factors for hyperkalaemia before administration of suxamethonium, even when serum potassium levels are normal.

Clinical record

An 80-year-old man with diabetes mellitus and hypertension (weight, 62 kg) was admitted to the emergency department with a perforated peptic ulcer. He had suffered traumatic intracranial haemorrhage 26 days before, with residual hemiplegia. Pre-operative serum potassium level was 4.0 mmol/L. Mild muscle weakness was noted.

In the operating room, rapid-sequence intubation was performed following intravenous fentanyl (100 μg), etomidate (20 mg) and suxamethonium (60 mg) injection. Intubation was completed within 60 seconds of induction. Arterial blood pressure and electrocardiogram (ECG) were monitored continuously. Two to three minutes post-induction, the ECG showed narrow complex tachycardia with tented T waves. The patient became hypotensive. Volatile anaesthetic (inhaled desflurane) was discontinued immediately, and the patient was ventilated with pure oxygen. Abrupt onset of cardiac arrest occurred after another minute. Cardiopulmonary resuscitation was commenced promptly. Sinus rhythm with stable blood pressure was regained within 3–5 minutes. Blood tests taken during resuscitation showed dramatic elevation of serum potassium level (up to 8.8 mmol/L). Calcium gluconate and insulin in dextrose solution were administered to correct hyperkalaemia. Twenty minutes later, the serum potassium level fell back to 3.2 mmol/L. Transoesophageal echocardiography excluded pericardial effusion or regional wall motion abnormalities. The operation was completed uneventfully 3 hours later, and the patient was discharged from the ward 4 days later with no additional neurological deficits.

Discussion

Previous reports suggest that suxamethonium provides better and more rapid intubating conditions than other non-depolarising muscle relaxants, such as rocuronium. Therefore, suxamethonium is still commonly used for rapid-sequence intubation, especially in patients with a potentially full stomach. However, life-threatening hyperkalaemia is a known risk of suxamethonium. The common risk factors are shown in the Table. Many anaesthetists avoid using suxamethonium in any patient with a serum potassium level over 5.5 mmol/L. Schow et al systemically reviewed the Duke Anesthesiology electronic database of 41117 patients who received suxamethonium over a 70-month period, and found 38 patients with serum potassium levels > 5.5 mmol/L before intubation. All underwent anaesthesia with no documented cases of dysrhythmia or unexpected intensive care admission. The authors concluded that use of suxamethonium is safe in
patients with modest hyperkalaemia. In view of this finding, we wish to reinforce the need for careful evaluation of patients at high risk of suxamethonium-induced hyperkalaemia, even those with normokalaemia.

In our patient, the serum potassium level was elevated by 4.8 mmol/L after administration of suxamethonium. This degree of hyperkalaemia induced ventricular arrhythmia and cardiac arrest. As the patient was fully monitored, the cardiac event was diagnosed and treated promptly.

The most likely factor contributing to hyperkalaemia in this patient was denervation secondary to traumatic intracranial haemorrhage and/or immobilisation. Suxamethonium-induced hyperkalaemia may occur from 7 days up to 6 months after immobilisation.3,4 Patients with cerebrovascular events are also known to have decreased myogenic activity, also suggesting denervation of affected muscles.2 Denervation causes upregulation of acetylcholine receptors in skeletal muscle, therefore sensitising muscle response to suxamethonium.2 Administration of suxamethonium rapidly depolarises these upregulated acetylcholine receptors, leading to massive efflux of intracellular potassium.

Another potential risk factor in this patient was intra-abdominal infection. Sepsis may increase serum potassium levels by 2.3 mmol/L in patients who are confined to bed for days.5

In summary, suxamethonium-induced hyperkalaemia should always be considered when suxamethonium is used in patients at high risk, even in those with normokalaemia before induction of anaesthesia.

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**References**