CARDIAC ARRHYTHMIA AFTER SUCCINYLCHOLINE ADMINISTRATION IN A PATIENT WITH GUILLAIN-BARRÉ SYNDROME

- A Case Report -

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Abstract

We report a case of cardiac arrhythmia occurring in a Guillain-Barré syndrome (GBS) patient after succinylcholine administration during third endotracheal intubation, on day 13 of illness. The probable cause of arrhythmia is succinylcholine-induced hyperkalemia. Of interest, this case demonstrated in the same patient that arrhythmia only occurred during third intubation, when duration of illness is prolonged, and not during previous two intubation episodes, despite succinylcholine was also being used. In GBS, muscle denervation resulted in up-regulation of acetylcholine receptors at neuromuscular junctions, causing the muscle cell membrane to become supersensitive to succinylcholine, leading to severe hyperkalemia and arrhythmia when succinylcholine was administered.

Key words: Guillain-Barré syndrome, Succinylcholine, Cardiac arrhythmia, Endotracheal intubation, Acetylcholine receptor up-regulation, Denervation.

Introduction

Succinylcholine-induced hyperkalemia causing life-threatening cardiac arrhythmia has been reported in Guillain-Barré syndrome (GBS)\(^1\)\(^2\) and in chronic polyneuropathy\(^3\). Here, we present a case of cardiac arrhythmia in a patient with GBS after succinylcholine administration during the third endotracheal intubation, on day 13 of illness. Of interest, this case demonstrated in the same patient that arrhythmia only occurred during third intubation, when the duration of illness is prolonged, and not during previous two intubation episodes, despite succinylcholine was also being used.
Case Report

A 30-year-old woman presented with 4-day history of hands and feet numbness, followed by weakness of upper and lower limbs that started distally. One day prior to admission, she became unable to walk, breathless, and having dysphagia. On presentation, she was conscious, afebrile but tachypneic. Power of both upper and lower limbs was 2/5, and with generalized areflexia.

A clinical diagnosis of GBS was made. She deteriorated fast and was intubated for respiratory failure. Midazolam 5mg intravenous (i.v.) and succinylcholine 100mg i.v. were given prior to intubation. She was admitted to intensive care unit (ICU) for mechanical ventilation, and was given i.v. immunoglobulin 0.4g/kg/day for 5 days.

On day 6 of ICU stay (day 10 of illness), her muscle power improved to 4/5 for upper limbs and 3/5 for lower limbs. She was extubated and put on oxygen supplementation. However, 1 hour after extubation, she became breathless and was unable to speak. She was re-intubated, using i.v. midazolam and succinylcholine as before. The potassium level prior to re-intubation was 4.32 mmol/L.

On day 9 of ICU stay (day 13 of illness), her condition has again improved clinically. She was extubated and given oxygen supplementation. However, 1.5 hour after extubation, she again became tachypneic. Nevertheless, she remained conscious and oxygen saturation by pulse oxymetry was maintained at 99%. Re-intubation was decided. To facilitate intubation, i.v. midazolam 5mg and i.v. succinylcholine 100mg were given. Just before attempt for intubation, ventricular tachycardia (VT) was noted on cardiac monitor. She was promptly intubated, and cardiopulmonary resuscitation (CPR) was immediately initiated, and was continued for about 15 minutes. During CPR, she was defibrillated 3 times for pulseless VT, before being reverted to sinus tachycardia. Her potassium level on that morning was 4.41mmol/L. The potassium level repeated after CPR was 4.63 mmol/L.

On day 13 of ICU stay, there was a leak at the endotracheal tube. She was extubated and given non-invasive ventilation. She tolerated well and was weaned off ventilator the next day. Her muscle power eventually returned to normal. She could walk, talk and swallow well, and was discharged after 20 days of hospitalisation.

Discussion

The probable cause of cardiac arrhythmia in this patient is succinylcholine-induced hyperkalemia. Noxious stimuli such as laryngoscopy and endotracheal intubation may also trigger cardiac arrhythmia, but in this case arrhythmia has occurred prior to such manipulation.

Normally, serum potassium increases by 0.5 to 1 mmol/L after succinylcholine administration. In GBS, muscle denervation results in increased production of acetylcholine receptors. With this up-regulation, the muscle cell membrane becomes supersensitive to succinylcholine, resulting in a great efflux of potassium upon succinylcholine administration, that may lead to severe hyperkalemia and cardiac arrhythmia. This supersensitivity increases dramatically by day 5, and can continue for weeks or even longer. This explains why this patient did not develop arrhythmia on admission day and on day 6 when succinylcholine was used, but subsequently developed it on day 9 of ICU stay (day 13 of illness). It is important to note that prior to second intubation on day 6 and third intubation on day 9, the potassium levels were 4.32mmol/L and 4.41mmol/L, respectively. Despite the very similar potassium level, arrhythmia only happened on a later occasion.

Succinylcholine-induced hyperkalemia is a transient phenomenon, where potassium level peaked at 3 - 5 minutes to around 8 or 9 mmol/L in denervated muscle, then started decreasing over time, and eventually back to normal range by 20 - 30 minutes. This may explain why severe hyperkalemia was not seen in her post-CPR blood results, as blood was only withdrawn about 30 minutes after onset of VT.

From the literature, succinylcholine-induced hyperkalemia has not been reported in patients with acquired pathologic states of < 4 days’ duration. This case re-affirms the observation, where cardiac arrhythmia (most probably due to hyperkalemia) only occurred at day 13 of illness, and not earlier even though succinylcholine was used twice before
that. Of particular interest, this case demonstrated in the same patient that arrhythmia did not occur with succinylcholine administration on days 1 and 6 of admission, but happened on day 9 of admission during third intubation, despite the very similar pre-intubation potassium level of around 4.4mmol/L during each occasion.

In conclusion, succinylcholine should be avoided in GBS, as has been recommended by others\(^1\), especially when the duration of illness was > 4 days\(^5\). Instead, non-depolarizing muscle relaxant with relatively more rapid onset such as rocuronium should be considered.

References


