

Succinylcholine-induced masseter muscle rigidity in an emergency department: a case report

琥珀膽鹼引起之咀嚼肌僵硬：一個在急症室內的個案報告

KW Suen 孫健榮, HY Lee 李凱揚, HF Ho 何曉輝

Succinylcholine is the most popular muscle relaxant employed in local accident and emergency departments because of its effectiveness and short-acting half life. Significant adverse reaction is rare and it increases the success rate of intubation. We describe a case of masseter muscle rigidity after administration of succinylcholine alone in the emergency room. The patient subsequently required nasotracheal intubation for ventilation. (*Hong Kong j.emerg.med.* 2010;17:281-284)

琥珀膽鹼是本地急症室內最常用的肌肉鬆弛劑，因它半衰期短及有效提升氣管內插管的成功率，而嚴重的副作用則罕見。本文描述一個在急症室內，只注射了琥珀膽鹼後咀嚼肌僵硬的個案，病人其後需經鼻氣管插管以換氣。

Keywords: Intratracheal intubation, malignant hyperthermia, spasm, trismus

關鍵詞：氣管內插管、惡性高熱、痙攣、牙關緊閉

Case

A 70-year-old lady was brought to the accident and emergency department by ambulance in December 2008, with the chief complaint of dyspnoea. She experienced low back pain two days before her attendance and consulted her private general practitioner. Paracetamol, nimesulide and famotidine were prescribed. She developed generalised oedema and difficulty in breathing afterward. She had a medical history of cervical cancer with radiotherapy 20 years ago. She had been ambulatory and enjoyed reasonably

good health in recent years. There was no history of head and neck malignancy or major operation.

On arrival, she was very tachypnoeic and showed signs of shock as evidenced by cool skin and impaired sensorium. The initial blood pressure was 122/92 mmHg with a pulse rate of 128/min, and SpO₂ was undetectable, probably due to poor peripheral circulation. She was semiconscious but able to stick out her tongue in response to instruction. There was bilateral pitting oedema over the lower limbs. Her condition continued to deteriorate and the blood pressure dropped to 69/33 mmHg. Point-of-care testing blood gas analysis showed metabolic acidosis and hyponatraemia (Na 118 mmol/L, K 4.5 mmol/L, pH 7.15, pCO₂ 2.8 kPa, pO₂ 44.2 kPa, HCO₃ 7 mmol/L, base excess -21 mmol/L, O₂ saturation 100%). The electrocardiogram revealed sinus tachycardia without apparent ischemic change. A central line was inserted and the first reading was 13 mmH₂O. She developed respiratory arrest after the central line insertion.

Correspondence to:

Suen Kin Wing, MRCS(Ed)

Queen Elizabeth Hospital, Accident and Emergency Department,
30 Gascoigne Road, Kowloon, Hong Kong

Email: kennethsuen@yahoo.com

Lee Hoi Yeung, FHKAM(Emergency Medicine)

Ho Hiu Fai, FHKAM(Emergency Medicine)

Ventilation was achieved by bag-valve-mask with satisfactory chest rise and oxygen saturation. A bolus dose of succinylcholine 100 mg was given intravenously. Oral endotracheal intubation was attempted without sedative agent as the patient was already unconscious and the situation was critical. Mouth opening was difficult due to severe jaw muscle spasm. The jaw was very stiff and barely allowed any movement even after the muscle fasciculation had subsided. Oral intubation was deemed impossible and no further attempt was made. Ventilation was successfully maintained by bag-valve-mask. The anaesthesiologist was consulted and nasotracheal intubation under flexible fiberoptic guidance was achieved. The position was confirmed by auscultation and end tidal CO₂ monitor. After intubation, the patient regained spontaneous respiration. Rocuronium 30 mg was given as part of the post-intubation care. The masseter muscle rigidity persisted despite the administration of non-depolarising muscle relaxant. The blood pressure remained on the low side and dopamine infusion was initiated to maintain adequate cardiac output. End tidal carbon dioxide level ranged from 14-20 mmHg. Repeat examination revealed persistent spasm of the masseter muscle one hour after the administration of succinylcholine. Her skin was cold with a body temperature of 35 degrees Celsius and the limbs were flaccid. She was transferred to the intensive care unit (ICU) for further care. Creatine kinase level was checked two hours after the onset of masseter muscle rigidity and the value was 5181 IU/L. Serial blood gas analysis detected worsening metabolic acidosis. Her condition ran a downhill course despite aggressive medical therapy in the ICU. She succumbed five hours after admission. The final diagnoses were acute renal failure and acute myocardial infarction.

Discussion

Succinylcholine is the most popular muscle relaxant employed in local accident and emergency departments because of its effectiveness and short-acting half life.¹ Masseter muscle rigidity is a known complication of succinylcholine. It is especially common in children

anaesthetised with succinylcholine and volatile inhalational anaesthetics, such as halothane. The incidence of masseter muscle spasm of this group of patients is estimated to be around 1%.²⁻⁵ Most of the previous studies on masseter muscle spasm described occurrence of this rare complication during the combined use of agents and in paediatric patients undergoing procedures in the operating theatre. Recently there have been some case reports on masseter muscle spasm after receiving succinylcholine in the emergency setting⁶⁻⁹ (Table 1). The diagnosis of masseter muscle rigidity should only be made after temporomandibular joint pathology and inadequate succinylcholine dosage are excluded.

Succinylcholine normally increases masseter muscle tone. van der Spek et al demonstrated reduced mouth opening and increased jaw stiffness in children anaesthetised with halothane or isoflurane and paralyzed by succinylcholine.^{10,11} Plumley et al also came out with a similar finding.¹² Succinylcholine increased resting muscle tension with a more pronounced effect on the masseter. It was described as a normal pharmacological response. However, true masseter muscle rigidity is defined as marked stiffness of the jaw which barely allows any mouth opening instead of the mere increase in muscle tone.

Apart from interference with oral intubation, masseter muscle rigidity is associated with malignant hyperthermia. It can be the herald of malignant hyperthermia. Malignant hyperthermia is triggered by succinylcholine or volatile inhalational anaesthetics as a result of hypermetabolism of skeletal muscle. Clinical features consist of hyperthermia, increased end tidal CO₂, rigidity, tachycardia, tachypnoea, labile blood pressure, hyperkalaemia and acidosis. The likelihood of patients suffering from masseter muscle rigidity subsequently developing malignant hyperthermia is an interesting clinical question. Previous experiences, mainly in children, showed that masseter muscle rigidity was often an isolated phenomenon. Malignant hyperthermia would not follow if the offending agent was stopped.²⁻⁴ Furthermore, the incidence of masseter muscle rigidity is much higher than that of malignant hyperthermia. In one study by O'Flynn et al, 59%

Table 1. Case reports on masseter muscle spasm after succinylcholine in the emergency setting

Author	Case history	Clinical course	Outcome
Gill et al ⁹	20/F sustained cervical injury. Intubated for agitation and cervical spine protection.	Masseter spasm after lorazepam, lidocaine, etomidate and succinylcholine. Vecuronium and additional two doses of succinylcholine given but masseter muscle spasm persisted. Failed nasotracheal intubation with fiberoptic visualisation.	Successfully performed retrograde endotracheal intubation. No evidence of MH. Discharged uneventfully.
Bauer et al ⁶	56/M presented with GIB and hypotension. Intubated for unconsciousness and airway protection. History of head and neck tumour, but mandibular opening was checked before intubation.	MMR after etomidate and succinylcholine. Further dose of succinylcholine given without effect. Attempted nasotracheal intubation with fiberoptic visualisation but failed.	Circothyroidotomy performed. No evidence of MH. Died 14 days in ICU.
Roman et al ⁸	36/M ingested an overdose of clonidine. Intubated for unconsciousness.	MMR after etomidate and succinylcholine. Intubation unsuccessful.	MMR resolved with vecuronium. No evidence of MH.
Ramirez et al ⁷	25/M presented with chest wall injury and left upper limb fracture in a traffic accident. Intubated for agitation and hypoxaemia. Then transferred for angiography and operation.	Masseter muscle spasm developed after etomidate and succinylcholine. Intubation still possible and remained stable afterward. Given isoflurane in operating theatre. Noticed hyperthermia, tachycardia and rise in end tidal CO ₂ level.	Diagnosed MH, supported by biochemical change. Dantrolene given. Uneventful recovery. Confirmed MH susceptible by muscle biopsy.

GIB=gastrointestinal bleeding, ICU=intensive care unit, MH=malignant hyperthermia, MMR=masseter muscle rigidity

paediatric patients with masseter muscle rigidity were shown to be malignant hyperthermia susceptible on muscle biopsy result.¹³ However, the data should be interpreted with caution as patients referred for muscle biopsy may represent a selected group of patients. Furthermore, positive malignant hyperthermia susceptibility does not imply development of malignant hyperthermia upon exposure to trigger agents.

Succinylcholine-induced masseter muscle rigidity is usually transient and resolved spontaneously,¹⁴ but may be prolonged as in this case. This signifies the importance of pre-oxygenation in rapid sequence intubation and a contingency plan in failed airway. Elevation of creatine kinase level is common in succinylcholine-induced masseter muscle spasm.³

Myoglobinuria is occasionally detected. Arterial blood gas and potassium level should be normal. Significant rise in creatine kinase level (over 20,000 U/L) and generalised muscle rigidity should arouse the suspicion of malignant hyperthermia.¹⁵

Our patient experienced masseter muscle rigidity and oral intubation was impossible. It was fortunate that ventilation could be maintained by bag-valve-mask and definite airway was subsequently achieved with the nasotracheal route. The masseter muscle rigidity was a protracted one and not relieved by non-depolarising muscle relaxant. Clinical examination did not show any clinical evidence of malignant hyperthermia. Elevated creatine kinase level could be found in patients with masseter muscle spasm alone. Worsening

metabolic acidosis reflected tissue hypoperfusion as a result of inadequate cardiac output. End-tidal carbon dioxide was not elevated and there was neither generalised muscle rigidity nor hyperthermia. We believe her clinical condition and death were attributable to underlying medical conditions. However, there was a remote possibility that manifestations of malignant hyperthermia, including elevation of end-tidal carbon dioxide level, could be blunted by her severe cardiovascular instability. Malignant hyperthermia may develop several hours after exposure to trigger agents. A clinical grading scale is available to predict the likelihood of malignant hyperthermia during a suspected event.¹⁶ The Malignant Hyperthermia Association of the United States has provided guidance on the management of malignant hyperthermia.¹⁷ All events must be documented carefully so that appropriate anaesthetic plans and family counselling can be offered.

Conclusion

Emergency physicians must be aware of the side effects of succinylcholine including masseter muscle rigidity. When challenged with such a condition, a difficult airway algorithm could be followed. Adequate ventilation should best be achieved by bag-valve-mask. If oral tracheal intubation is impossible, nasotracheal or surgical airway is an alternative. After the airway has been secured, the patient should be closely monitored for clinical evidence of malignant hyperthermia.

References

- Choi YF, Wong TW, Lau CC, Siu AYC, Lo CB, Yuen MC, et al. A study of orotracheal intubation in emergency departments of five district hospitals in Hong Kong. *Hong Kong J Emerg Med* 2003;10(3):138-45.
- Carroll JB. Increased incidence of masseter spasm in children with strabismus anesthetized with halothane and succinylcholine. *Anesthesiology* 1987;67(4):559-61.
- Schwartz L, Rockoff MA, Koka BV. Masseter spasm with anesthesia: incidence and implications. *Anesthesiology* 1984;61(6):772-5.
- Kosko JR, Brandon BW, Chan KH. Masseter spasm and malignant hyperthermia: a retrospective review of a hospital-based pediatric otolaryngology practice. *Int J Pediatr Otorhinolaryngol* 1992;23(1):45-50.
- Lazzell VA, Carr AS, Lerman J, Burrows FA, Creighton RE. The incidence of masseter muscle rigidity after succinylcholine in infants and children. *Can J Anaesth* 1994;41(6):475-9.
- Bauer SJ, Orto K, Adams BD. Succinylcholine induced masseter spasm during rapid sequence intubation may require a surgical airway: case report. *Emerg Med J* 2005;22(6):456-8.
- Ramirez JA, Cheatham ED, Laurence AS, Hopkins PM. Suxamethonium, masseter spasm and later malignant hyperthermia. *Anaesthesia* 1998;53(11):1111-6.
- Roman CS, Rosin A. Succinylcholine-induced masseter muscle rigidity associated with rapid sequence intubation. *Am J Emerg Med* 2007;25(1):102-4.
- Gill M, Graeme K, Guenterberg K. Masseter spasm after succinylcholine administration. *J Emerg Med* 2005;29(2):167-71.
- van der Spek AFL, Reynolds PI, Fang WB, Ashton-Miller JA, Stohler CS, Schork MA. Changes in resistance to mouth opening induced by depolarizing and non-depolarizing neuromuscular relaxants. *Br J Anaesth* 1990;64(1):21-7.
- van der Spek AFL, Fang WB, Ashton-Miller JA, Stohler CS, Carlson DS, Schork MA. The effects of succinylcholine on mouth opening. *Anesthesiology* 1987;67(4):459-65.
- Plumley MH, Bevan JC, Saddler JM, Donati F, Bevan DR. Dose-related effects of succinylcholine on the adductor pollicis and masseter muscles in children. *Can J Anaesth* 1990;37(1):15-20.
- O'Flynn RP, Shutack JG, Rosenberg H, Fletcher JE. Masseter muscle rigidity and malignant hyperthermia susceptibility in pediatric patients. An update on management and diagnosis. *Anesthesiology* 1994;80(6):1228-33.
- Rosenberg H. Clinical presentation of malignant hyperthermia. *Br J Anaesth* 1988;60(3):268-73.
- Larach MG, Rosenberg H, Larach DR, Broennle AM. Prediction of malignant hyperthermia susceptibility by clinical signs. *Anesthesiology* 1987;66(4):547-50.
- Larach MG, Localio AR, Allen GC, Denborough MA, Ellis FR, Gronert GA, et al. A clinical grading scale to predict malignant hyperthermia susceptibility. *Anesthesiology* 1994;80(4):771-9.
- Malignant Hyperthermia Association of the United States. Emergency therapy for malignant hyperthermia. [2008 May]. [cited 2010 Feb 5]. Available from: <http://www.mhaus.org>