

CASE REPORT

Succinylcholine induced masseter muscle rigidity: an isolated event

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ABSTRACT

Masseter muscle rigidity (MMR) during general anesthesia is considered to be an early warning sign of possible episode of malignant hyperthermia (MH). We report a case of 36 year old male patient, posted for surgical repair of epigastric hernia, which developed MMR following a standard dose of intravenous succinylcholine during induction of anesthesia. Suspecting MMR as an early indicator of malignant hyperthermia, all the precautions were taken and after securing the airway with endotracheal intubation anesthesia was maintained with propofol infusion, avoiding triggering factor like halogenated inhalational agents. Patient's temperature, end tidal CO₂, heart rate and blood pressure remained within normal limits during intraoperative and postoperative period. Patient was carefully monitored and investigated in postoperative period and a moderate rise in serum creatinine phosphokinase level (401 U/L) was recorded at 24 hours. He recovered well and was discharged uneventfully.

Key words: Succinylcholine; Masseter muscle rigidity; Malignant hyperthermia; MH; General anesthesia; Rhabdomyolysis

Citation: Sharma M, Sharma S, Kalra P, Kulshreshtha A. Succinylcholine induced masseter muscle rigidity: an isolated event. *Anaesth Pain & Intensive Care* 2013; 17(2):174-178

INTRODUCTION

Succinylcholine, a depolarizing neuromuscular blocking agent, is frequently administered to facilitate endotracheal intubation because of its rapid onset and relatively brief duration of action. One rare adverse effect of succinylcholine is masseter muscle rigidity (MMR) also known as masseter muscle spasm or 'jaw of steel'.^{1,2} MMR has been reported to be an early indicator of malignant hyperthermia (MH), though some believe that isolated MMR is not pathognomonic of MH.¹ The overall incidence of MH in literature is quoted to be 1:40,000 to 1:50,000 in adults and 1: 15,000 in children;³ however, incidence of MMR has been reported to be less than 1% in children⁴ and in adults it is unknown.⁵

The cause of MH is most often a mutation involving the sarcoplasmic reticulum (SR) calcium release channel (ryanodine receptor: RyR₁ gene). During MH episode the SR calcium channels open persistently and the resulting calcium influx causes sustained muscle contraction, hyperthermia, and increased metabolism leading to life threatening complications with high incidence of mortality.⁶ Hence it is important to consider possibility of MH in a patient who develops severe masseter spasm following administration

of induction agents. The decision whether to continue the procedure or not depends on the urgency of the surgery and severity of muscle rigidity.⁷

There are many case reports of MH in Western journals⁸ but sporadic cases of MMR and MH have been reported from Indian subcontinent.^{3,8} We report here a case of severe masseter spasm after succinylcholine administration; its pathogenesis and anesthetic implications are also discussed.

CASE REPORT

A 36 years old, 60 kg male with no known medical illness was posted for surgical repair of epigastric hernia. Patient's gastroscopy report showed esophagitis with hiatus hernia. He had no history of previous exposure to anesthetic agents; preoperative evaluation did not reveal any feature suggestive of difficult intubation. He had Mallampatti grade I, inter-incisor gap 4.5 cm, upper lip bite test class I and thyromental distance 7 cm. Patient's investigations and clinical examination were unremarkable and he was classified as ASA class I.

He was taken to operating room after 8 hours fasting and Ringer's lactate infusion was started via 20G IV cannula.

Monitoring included electrocardiography (ECG), non-invasive blood pressure (NIBP) and pulse oximeter (SpO₂). His baseline heart rate was 86/min, blood pressure was 120/80 mmHg, SpO₂ was 98% on air. Patient was pre-medicated with glycopyrrolate 0.2 mg, midazolam 1 mg and tramadol 100 mg IV.

A rapid sequence induction was planned due to risk of aspiration of gastric contents. After pre-oxygenation with 100% oxygen for four minutes, induction was performed using thiopentone 350 mg and succinylcholine 100 mg IV. No fasciculation's were observed. After a minute, laryngoscopy was attempted, but the teeth were tightly clenched and the mouth was unable to be opened. We could not introduce even the tip of the laryngoscope blade. Considering the masseter muscle spasm, we continued mask ventilation and oxygen saturation was maintained at 100%. A few moments later we again tried to open the mouth but failed due to severe MMR; the mask ventilation was continued. After 5 minutes, the patient resumed spontaneous respiratory effort, and inj. propofol 40 mg IV bolus was given. The jaw was felt relatively relaxed now and laryngoscopy was attempted. This time we could successfully introduce the laryngoscope blade (MacIntosh curved) with difficulty and we could visualize the posterior 1/3rd of moving vocal cords with OELM (optimum external laryngeal manipulation). Patient was intubated with cuffed endotracheal tube No. 8 and tube placement was confirmed by EtCO₂ monitor. Inj. atracurium was given and IPPV started. Keeping in view the possibility of developing overt MH and subsequent myoglobinuria, the patient was catheterized to monitor the color of urine. Temperature monitoring was started with nasopharyngeal thermister to watch for hyperthermeia. Considering the fact that the patient was hemodynamically stable and the expected duration of the surgery was less than an hour, the decision was taken to proceed with the procedure. We

avoided all halogenated anesthetic agents, and anesthesia was maintained with propofol infusion (100 µg/kg/min), atracurium and N₂O and O₂ in 3:2 ratio. Patient's heart rate, blood pressure and SpO₂ were maintained within normal range during intra-operative period. EtCO₂ remained between 28-30 mmHg. Temperature was normal and urine remained clear throughout the surgical duration (35 minutes). Patient was reversed using neostigmine 2.5 mg and glycopyrrolate 0.5mg. He became fully conscious, his muscle power was adequate with good tidal volume, head lift >5 second was present and was extubated. Patient was kept in operating room for 1 more hour for observation. At 1 hour mouth opening was assessed. Now the jaw was completely relaxed and mouth opening had returned to preoperative status, confirming the diagnosis of succinylcholine induced MMR. Patient's blood sample was sent for arterial blood gas (ABG's) analysis, electrolytes and creatinine phosphokinase (CPK) level. ABG's were normal with pH 7.42, serum K⁺ 6.02 meq/l and CPK 198 U/L (reference value 55.0-170.0 U/L). Patient was shifted to ICU and was monitored for any sign of MH. Repeat blood sample was sent at 12 hours postoperatively. CPK showed a rise to 305 U/L, serum K⁺ and ABG's were normal. At 24 hours CPK was 401 U/L, and the ABG's and serum K⁺ were normal (Table 1).

Throughout the postoperative period patient remained afebrile and his urine remained clear. Patient's family history regarding MMR and MH and myopathies was taken and found to be negative. Patient was diagnosed as a case of isolated MMR following succinylcholine as evidenced by masseter spasm, elevated serum K⁺ levels and slight increase in serum CPK level. As urine remained clear throughout, urine sample for myoglobin was not sent. Postoperative recovery was uneventful and he was discharged on third postop day.

Patient was advised muscle biopsy for halothane

Table 1: Postoperative laboratory investigations of the patient

Parameter	Reference range	1 hour postop	12 hour postop	24 hour postop
pH	7.35-7.45	7.43	7.39	7.35
pCO ₂	35-45 mmHg	32.6mmHg	34 mmHg	40 mmHg
PO ₂	80-100 mmHg	78.4 mmHg	92 mmHg	87 mmHg
BE*	- 2-2meq/l	- 2.5 meq/l	- 3.9 meq/l	- 3.3 meq/l
HCO ₃ ⁻	22-26 meq/l	22 meq/l	20.6 meq/l	22.1 meq/l
Na ⁺	135-145 mmol/l	136.2 mmol/l	138 mmol/l	135 mmol/l
K ⁺	3.5-5.5 mmol/l	6.02 mmol/l	4.5 mmol/l	4.6 mmol/l
Cl ⁻	98-107 mmol/l	83.9 mmol/l	99 mmol/l	98 mmol/l
CPK**	55-170U/L	198U/L	305 U/L	401U/L

*BE-Base-excess, **CPK-Creatinine Phospho-Kinase

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caffeine contracture test (after 3 months) as per the protocol suggested by Larach et al⁹ to rule out malignant hyperthermia. The facility for this test is not available at our center and he was advised to get it done at a higher centre. He was also counseled regarding the life threatening risk of MH in future anesthesia; a special note regarding it and not to use succinylcholine was mentioned on discharge ticket. Throughout the postoperative period patient remained afebrile and his urine remained clear. Patient's family history regarding MMR and MH and myopathies was taken and found to be negative. Patient was diagnosed as a case of isolated MMR following succinylcholine as evidenced by masseter spasm, elevated serum K⁺ levels and slight increase in serum CPK level. As urine remained clear throughout, urine sample for myoglobin was not sent. Postoperative recovery was uneventful and he was discharged on third postop day.

Patient was advised muscle biopsy for halothane caffeine contracture test (after 3 months) as per the protocol suggested by Larach et al⁹ to rule out malignant hyperthermia. The facility for this test is not available at our center and he was advised to get it done at a higher centre. He was also counseled regarding the life threatening risk of MH in future anesthesia; a special note regarding it and not to use succinylcholine was mentioned on discharge ticket.

DISCUSSION

Succinylcholine has been frequently used for crash induction and rarely it may be associated with some serious adverse effects, e.g. MMR, rhabdomyolysis, elevated CPK and MH.

The MMR, the so called 'jaw of steel'¹⁰ is defined as marked stiffness of the jaw which barely allows any mouth opening instead of mere increase in the muscle tone.¹¹ The MMR causes difficult or impossible laryngoscopy leading to difficult or failed intubation. The later is an important cause of morbidity and mortality during anesthesia. Alternative techniques to secure the airway, e.g. retrograde endotracheal intubation¹², fiberoptic nasotracheal intubation¹¹ and/or surgical cricothyrotomy have been used following MMR in emergency cases. The trachlight¹³ and LMA have also been used successfully² in the event of succinylcholine induced MMR. In our case after the recovery from the effects of succinylcholine, an additional bolus of propofol was given and the lower jaw relaxed; we could intubate the patient with difficulty with the help of OELM, as was experienced by Saxena et al.⁸ It has been suggested that when used as an induction agent, propofol reduces the masseter muscle tension more effectively than thiopentone.¹⁴ High doses of fentanyl have also been commonly known to produce rigidity at induction in the nonparalysed patients.¹⁵ We used tramadol

which may cause seizures and serotonin syndrome¹⁶ but tramadol induced rigidity is not yet reported. Apart from interference with oral intubation MMR has been said to be an early indication of MH.¹

The pathophysiology of MH involves abnormal skeletal muscle calcium homeostasis in response to trigger agents (succinylcholine, halogenated inhalation agents). Sustained high levels of Ca⁺ in sarcoplasmic reticulum leads to increased aerobic and glycolytic metabolism leading to acidosis, rigidity along with rhabdomyolysis, myoglobinuria, hyperkalemia, hyperthermia and hemodynamic instability. A rise in EtCO₂ out of proportion to the clinical setting is the first sign of MH under general anesthesia.⁸ Muscle spasm following succinylcholine should raise suspicion of MH as it presages clinical MH in upto 30% of cases.⁶ However, MMR did not lead to MH in present case, as the patient remained afebrile, EtCO₂ remained normal, vital signs were stable and urine was clear throughout. Serum CPK and K⁺ showed a moderate rise as evidence of rhabdomyolysis. MMR as isolated event has been reported sporadically.² Generally, there is 20-30 min interval between MMR and the development of signs of MH.¹⁷ Accordingly, some authors advocate abandoning the surgical procedures following occurrence of MMR and to monitor the patient for signs of MH.¹⁸

Berry and Lurch¹⁹ suggested continuing the surgical procedure with careful monitoring for signs of MH. Triggering agents of MH (inhalation agents) should be avoided and anesthesia can be continued if the EtCO₂, ABG's, BP, HR, temperature, serum CPK, urine color and muscle tone are normal. Early signs of MH were not visible in our case. We decided to continue the surgery with careful monitoring for signs of MH. We avoided halogenated inhalational agents in view of the risk of developing MH and anesthesia was maintained with propofol infusion. This may be the reason that the MMR did not progress to MH. Some authors continued halothane⁸ or isofurane²⁰ after MMR, because at that time EtCO₂ was normal and patients were afebrile, but after one hour frank MH developed requiring intensive care treatment. Both of these cases showed a grater rise in CPK and serum K⁺ levels as compared to the present case.

CPK is an enzyme that exists predominantly in skeletal muscles. The reference range is 55-170 U/L. Elevated CPK levels indicate muscle damage either due to chronic disease or an acute muscle breakdown. It may increase after major or minor surgery, following MMR or muscle fasciculation accompanying succinylcholine administration. A study performed on children undergoing major surgery²¹ showed a median elevation of 43 U/L in CPK levels. However, maximum level of CPK, 1339 U/L on day 2 following major surgery²² has also been reported. Serum CPK generally peak 6-12 hours after MMR. Increase in

CPK levels have been reported in pediatrics patients who received succinylcholine and halogenated agents with no associated MMR.^{23,24} In our case CPK showed a moderate rise upto 401 U/L at 24 hours, which could be due to MMR or due to surgical trauma.

In normal muscles succinylcholine induced depolarization releases enough K⁺ to raise the serum level by 0.5 meq/l.²⁵ In our case the rise was more as evident by serum K⁺ levels of 6.02 meq/l at 1 hour postoperatively compared to preoperative level of 4.5 meq/l. Muscle biopsy for microscopic examination and halothane caffeine contracture test was advised as per the protocol of European Malignant Hyperthermia Group.²⁶ Cases have been reported with negative caffeine halothane contracture test who had a known clinical episode of MH under anesthesia.²⁷ The MH Raw score given by Larach et al⁹, a clinical grading scale considered a valuable tool for detection of MH.⁸ Among seven criteria of this score only two criteria i.e. masseter spasm following succinylcholine (15 points) and serum K⁺ >6meq/l (3 point) were present, leading to a total score of 18 corresponding to MH rank 3, classified as 'MH somewhat less likely'.⁹

An inadequate dose of succinylcholine (less than the recommended dose of 1 mg/kg), inadequate time for the onset of succinylcholine action, Duchenne muscular dystrophy, myotonia congenita and other muscle disorders

may produce MMR.^{6,28,29} All these causes were ruled out in the present case. MMR and MH have been reported in patients with no muscular disease⁸ as in our case. Cases have been reported where a patient of severe anterior dislocation of temporomandibular disc was mistakenly diagnosed as MMR.³⁰ Temporomandibular joint dysfunction was ruled out in our case by careful postoperative examination. Based on clinical and laboratory findings the case was diagnosed as succinylcholine induced isolated MMR

CONCLUSION

This case report highlights that succinylcholine may produce isolated MMR leading to difficult laryngoscopy and intubation. In such event airway must be secured promptly through any convenient means to avoid morbidity and mortality associated with difficult airway. In a patient with MMR, possibility of MH should always be kept in mind and trigger factors of MH should be avoided during maintenance of anesthesia. With due precautions surgery can be conducted uneventfully but the patient must always be monitored in postoperative period.

In addition such patients should get a muscle biopsy done for halothane caffeine contracture test to rule out MH and they should be explained and counseled about possible risk on further exposure to general anesthesia.

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