

CASE REPORT

Masseter Muscle Rigidity Following Succinylcholine Administration

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Abstract

Masseter muscle rigidity (MMR) during general anaesthesia is considered to be an early warning sign of possible episode of malignant hyperthermia (MH). We report a case of a male child, who developed MMR following a standard dose of intravenous Succinylcholine during induction of anaesthesia. We suspected MMR to be an early indicator of MH and took all the precautions and secured the airway with endotracheal intubation. Later on anaesthesia was maintained with Propofol infusion, and triggering factors like halogenated inhalational agents were avoided. Patient's vitals like 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 there was a moderate rise in serum creatinine phosphokinase level recorded after 24 hours. Later on the patient recovered well and was discharged uneventfully.

Key words: Anterior longitudinal ligament, Malignancy, Metabolic disorders, Ossification skeleton.

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Case Report

A 9 year old male child weighing 26 kgs with no known medical illness was posted for emergency appendicectomy. His Ultrasonography of abdomen showed acute appendicitis. There was no previous history of exposure to any anaesthetic agents and no family history of malignant hyperthermia or neuromuscular disease. Preanaesthetic checkup was done and did not reveal any feature suggestive of difficult intubation. He was an average built boy, with good mouth opening with a Mallampatti grade- I, upper lip bite test class I and Thyromental distance 3 finger breadths, full range of neck motion, no weakness or paresthesia in upper or lower extremities. The clinical investigations were all normal except for the USG Abdomen suggestive of acute appendicitis. He was given an ASA class I.

The patient had nothing by mouth for more than 4 hours before surgery. He was shifted to the operating room with a 20 G IV cannula. Ringers lactate was started through it. Monitoring included electrocardiography, non invasive

blood pressure (NIBP), and pulse oximetry (sPO₂) with ETCO₂ monitoring. His baseline heart rate was 98 / min, blood pressure was 110/70 mm Hg, sPO₂ was 98 % at room air.

Patient was premedicated with Injection Glycopyrolate 0.1 mg, Injection Midazolam 0.5 mg and Injection Zofer 2 mg. After pre-oxygenation with 100% oxygen for three minutes, induction was performed using Thiopentone 100 mg and Succinylcholine 30 mg. No fasciculations were observed. After one minute, laryngoscopy was attempted, but the teeth were tightly clenched and the mouth could not be opened. Even the tip of laryngoscope blade could not be introduced. Considering the masseter muscle spasm, mask ventilation was continued and sPO₂ was maintained at 100%.

One minute later again we tried to open the mouth, but failed to do so. So a MMR was diagnosed, then mask ventilation was continued. Then it was decided to give non depolarizing muscle relaxant, and gave Injection Vecuronium 1.6 mg and ventilated for 3 minutes. Laryngoscopy was attempted after 3 Minutes and now there was relative relaxation of the jaw. This time we could successfully introduce the laryngoscope blade (Macintosh curved) without

difficulty and the posterior 1/3rd of the vocal cords with OELM (optimum external laryngeal manipulation) was visualized. Patient was intubated with size 5 cuffed endotracheal tube and tube placement was confirmed by EtCO₂ monitor. IPPV started with N₂O and Oxygen in a 3:2 ratio. Halothane was avoided for fear of triggering MH. Anaesthesia was maintained with Injection Propofol infusion and Injection Vecuronium.

The patient was hemodynamically stable and the expected duration of the surgery was less than an hour and since it was an emergency, the decision was taken to proceed with the

procedure. Ringer's lactate solution was replaced with Dextrose normal saline (NS). Keeping in view the possibility of developing overt MH and subsequent myoglobinuria, the patient was catheterized to monitor the colour of urine. Temperature monitoring was started to watch for hyperthermia. Patient's heart rate, blood pressure and SPO₂ were maintained within normal range during intra-operative period. EtCO₂ remained between 25-35 mmHg. Temperature was normal and urine remained clear throughout the surgical duration (40 minutes).

Figure-1: Inability to open mouth, difficult laryngoscopy, mouth opening after depolarizing agent and successful intubation



1,2,3,4- Inability to open the mouth, Difficult laryngoscopy, mouth opening after nondepolarising agent & successful intubation

Patient was reversed using Neostigmine 50 mcg/kg and Glycopyrrolate 10 mcg/kg. He became fully conscious, his muscle power was adequate with good tidal volume, head lift was >5 seconds. He was extubated successfully. Patient was kept in operating room for 1 more hour for observation. At 1 hour his mouth opening was assessed. Now the jaw was completely relaxed and mouth opening had returned to preoperative status, which confirmed our diagnosis of Succinylcholine induced MMR.

Patient's blood sample was sent for arterial blood gas (ABG) analysis, electrolytes and creatinine phosphokinase (CPK) level. Patient was shifted to Post Operative ICU and was monitored for any sign of MH. Repeat blood sample was sent after 8 hours postoperatively. CPK showed a rise to 2816 U/L, serum K⁺ and ABG's were normal. At 24 hours the CPK was 1018 U/L, and the ABG's and serum K⁺ were normal (Table 1).

Figure- 2: Postoperative mouth opening and jaw relaxation



Postoperative mouth opening & jaw relaxation

Table 1: Postoperative laboratory investigations of the patient

Parameter	Reference range	30 Min Postoperatively	8 Hour Postoperatively	24 Hour Postoperatively
pH	7.35-7.45	7.42	7.38	7.34
pCO ₂	35-45 mm Hg	31.9	34.6	39.0
pO ₂	80-100 mm Hg	80.4	86.2	94.0
HCO ₃	20-24 meq/l	22	21.6	22.3
Na ⁺	135-145 mmol/l	135	138	135
K ⁺	3.5-5.5 mmol/l	5.5	4.9	4.6
Cl ⁻	98-107 mmol/l	92.1	98.1	96.0
CPK	55-195 U/L	1400	2816	1018

CPK-Creatinine Phospho-Kinase

Throughout the postoperative period patient remained afebrile, and his urine remained clear. Patient's family was enquired again for any history of MMR, MH or myopathies in their family, and it was 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 postoperative day 8. The child's parents were strongly advised to have their son undergo an extensive neuromuscular workup on an outpatient basis. A positive diagnosis of neuromuscular disease would trigger an additional evaluation of immediate family members. They were also encouraged to have their son undergo a muscle biopsy for MH susceptibility. The parents were advised that Succinylcholine should be avoided in subsequent anaesthetics in light of the occurrence of the MMR. We advised a muscle biopsy for halothane caffeine contracture test (after 3 months) as per the protocol suggested by Larach et al to rule out malignant hyperthermia.

Discussion

The MMR, the so called 'jaw of steel' is defined as marked stiffness of the jaw which barely allows any mouth opening instead of mere increase in the muscle tone. The MMR causes difficult or impossible laryngoscopy leading to difficult or failed intubation. The latter is an important cause of morbidity and mortality

during anaesthesia. Succinylcholine is well known to cause MMR and thereby increase in the CK levels, Rhabdomyolysis, and Malignant Hyperthermia. Masseter muscle rigidity occurs when there is an increase in masseter muscle tone making it difficult to fully open the mouth. This can make oral endotracheal intubation very challenging. Succinylcholine has been frequently used for crash inductions also.

Alternative techniques to secure the airway, e.g. retrograde endotracheal intubation, fiberoptic nasotracheal intubation and/or surgical cricothyrotomy have been used following MMR in emergency cases. The trachlight and LMA have also been used successfully in the event of Succinylcholine induced MMR.

In our case we have given a nondepolarising muscle relaxant and attempted for intubation with OELM after there was a relaxation of the jaw. Propofol infusion was given intraoperatively avoiding the triggers for MH like Halogenated inhalational agents. It is extremely important to know if this patient has an underlying neuromuscular disease and if he truly is suffering from MHS. Before planning any future anaesthetics for this patient ideally he should get a muscle biopsy done and rule out MH. These patients should be given a medical alert stating "no Succinylcholine." Practitioners should avoid Succinylcholine in any patient with a history of MMR after receiving the drug. Accordingly, some advocate abandoning the surgical procedure following the occurrence of MMR and treating the patient as MHS. They also recommend a muscle biopsy. Others may abort the procedure and monitor the patient for signs of MH, send blood for serial CKs and over 24 hours, and test the urine for myoglobinuria. Berry and Lynch suggest that the surgical procedure be continued, discontinuing known

triggers of MH (inhalation agents) and proceeding with an intravenous technique, carefully monitoring for early signs of MH (tachycardia, increased end-tidal carbon dioxide). Gronert suggests that the surgical procedure may continue if the end-tidal carbon dioxide, arterial blood gases, blood pressure, pulse rate, temperature, serum CK, urine color, and muscle tone are normal.

An inadequate dose of Succinylcholine (less than the recommended dose of 1 mg/kg, IV), inadequate time for the onset of Succinylcholine, Temporomandibular joint dysfunction, myotonic syndrome, Duchenne muscular dystrophy, myotonia congenita and other muscle disorders may mimic MMR. All these causes were ruled out in the present case. MMR and MH have been reported in patients with no muscular disease as in our case.

Creatine Kinase is an enzyme that exists predominantly within skeletal muscle. The reference range is 22 U/L to 198 U/L. Elevated CK levels indicate muscle damage due to either a chronic disease state or an acute muscle injury. In rhabdomyolysis, or acute muscle breakdown, CK levels can reach 50,000 U/L to 200,000 U/L. Increases in CK have been found to occur following both major and minor surgery, following MMR as in the present case, and following muscle fasciculations that accompany Succinylcholine administration. Levels have been shown to increase after the administration of Succinylcholine, but they increase even more with MMR. Serum CK levels generally peak 6 to 12 hours after MMR. A rise in CK levels can be caused by MMR, rhabdomyolysis, Succinylcholine, or Sevoflurane inhalation.

The pathophysiology of MH involves abnormal skeletal muscle calcium homeostasis in response to trigger agents (Succinylcholine, halogenated inhalation agents). Sustained high levels of Ca^{+2} 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 anaesthesia. Muscle spasm following Succinylcholine should raise suspicion of MH as it presages clinical MH in

upto 30% of cases. However, MMR did not lead to MH in present case.

Triggering agents of MH (inhalation agents) should be avoided and anaesthesia 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 anaesthesia was maintained with Propofol infusion. This may be the reason that the MMR did not progress to MH.

In normal muscles Succinylcholine induced depolarization releases enough K⁺ to raise the serum level by 0.5 meq/l. In our case the rise was more as evident by serum K⁺ levels of 5.5 meq/l after 30 Minutes postoperatively, compared to preoperative level of 4.0 meq/l. Muscle biopsy for microscopic examination and halothane caffeine contracture test was advised as per the protocol of European Malignant Hyperthermia Group. The MH Raw score given by Larach et al, a clinical grading scale considered a valuable tool for detection of MH.

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'.

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 anaesthesia. 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 anaesthesia.

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