Journal of Medical Case Reports and Reviews

Received 20 Aug 2019 | Revised 23 Aug 2019 | Accepted 26 Oct 2019 | Published Online 29 Oct 2019

JMCRR 02 (10) , 394-398 (2019) ISSN (O) 2589-8655 | (P) 2589-8647

JMCRR

Case Report

SUCCINYLCHOLINE INDUCED MASSETER MUSCLE RIGIDITY

Gopal Singh[1], Jai singh[2], Dheeraj singha[3], Manju*[4]

[1.]M.D. Anaesthesia Department of Anaesthesia, Zonal Hospital Dharamshala H.P.

[2.]Professor Department of Anesthesia Dr.RPGMC Tanda H.P.

[3.]Associate professor Department of Anaesthesia Dr.RPGMC Tanda H.P.

[4.]Senior Resident Department of Medicine Dr.RPGMC Tanda H.P.

Abstract: Masseter muscle rigidity (MMR) during general anesthesia is an early warning sign of possible episode of malignant hyperthermia (MH). We report a case of 56-year-old male patient, posted for open cholecystectomy which developed MMR following an intravenous dose of 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 agents like volatile anesthetic gases, muscle relaxants etc. Patient vitals viz, 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 (280 U/L) was recorded at 24 hours. He recovered well and was discharged uneventfully.

Key words: Succinylcholine; Masseter muscle rigidity; malignant hyperthermia; MH; Rhabdomyolysis. TMJ,Temporomandibular joint

Corresponding Author: Dr.Manju*, Senior Resident Medicine Department of Medicine Dr.RPGMC Tanda H.P.

Introduction:

Succinylcholine, a depolarizing neuromuscular blocking agent, is used for endotracheal intubation because of its rapid onset and short 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]. Muscle rigidity as seen in MMR has been said to be an early indicator of MH[4] Its incidence is less than 1% in children[5], while the incidence in adults is unknown, But some believe that isolated MMR is not pathognomonic of MH.1. The overall incidence of malignant hyperthermia in literature has been quoted as 1: 40,000 to 1:50,000 anesthetics in adults and 1: 15,000 anesthetics in children.[3] 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 leading to 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 regarding whether continuing 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 journals[8] but few 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 56 years old, 68kg male patient posted for open cholecystectomy under general anesthesia. There is no history DM, HTN, BRONCHIAL ASTHMA, SEIZURE AND ATT INTAKE. No history of previous exposure to anesthetic agents; preoperative evaluation did not reveal any feature suggestive of difficult intubation. Air way assessment Mallampatti grade 2, upper lip bite test class I and thyromental distance >6.5 cm, NM-adequate, METS>4, MO>3FB. Preoperative investigations CBC, RFT, LFT, Serum electrolyte-sodium and potassium and FBS were with in normal range and clinical examination cardiovascular system and respiratory system and CNS and CXR were normal and were classified as ASA class I. on shifting to Operation Theater. Ringer's lactate infusion was started via 18G IV cannula. Routine Monitor viz electrocardiography (ECG), noninvasive blood pressure (NIBP) and pulse oximeter (SpO2) were attached. His baseline heart rate was 76/min, blood pressure was 130/88 mmHg, SpO2 was 97% on room air. Premedication with injection midazolam 1 mg , glycopyrrolate 0.2 mg and fentanyl 100 mg IV given. After pre-oxygenation with 100% oxygen for three minutes, induction was performed using thiopentone 380 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 difficult to be opened. We could not introduce the laryngoscope blade. Keeping the possibility masseter muscle spasm, we continued mask ventilation and oxygen saturation was maintained up to 99-100%. After few minutes we again tried to open the mouth but failed due to severe MMR, the mask ventilation was continued. After few minutes, the patient resumed spontaneous respiratory effort, and inj. propofol 50 mg IV bolus was given. The jaw was felt slightly relaxed and laryngoscopy was again 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 (external laryngeal manipulation). Patient was intubated with cuffed endotracheal tube 7.5 no. And placement of ETT was confirmed by auscultation and EtCO2 monitor. Inj. Atracurium 30 mg 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 is used to watch for hyperthermia. Inspite of fact that the patient was hemodynamically stable and the expected duration of the surgery was one and half an hour, the decision was taken to proceed with the procedure. Avoided all halogenated anesthetic agents, and anesthesia was maintained with propofol infusion (100 µg/kg/min), atracurium and N2O and O2. Heart rate, blood pressure and SpO 2 were maintained within normal range during intra-operative period. EtCO2 remained between 30-33 mmHg. Temperature was normal and patient urine remained clear throughout the surgical procedure. Patient was reversed using neostigmine 3.5 mg and glycopyrrolate 0.6mg. And patient became fully conscious, regular respiration and adequate muscle power with good tidal volume, head lift >5 second was present and was extubated. Patient was kept in recovery room for observation. After one hour patient was assessed mouth opening and the jaw was completely relaxed and mouth opening had returned to preoperative status, confirming the diagnosis of succinylcholine induced MMR. His blood sample was sent for ABG analysis, electrolytes and creatinine phosphokinase (CPK) level. ABG's were normal with pH 7.42, serum K+ 6.02 meq/l and CPK 205 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 after 12 hours postoperatively. CPK value rise to 270 U/L, ABG's sodium,

potassium were normal. After 24 hours CPK was 294 U/L, and the ABG's and serum K+ were normal during postoperative period patient remained afebrile and his urine remained clear and had no relatives who had problems previously under general anesthesia during surgery. 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. Postoperative recovery was uneventful and he was discharged on fourth postop day. Patient was advised muscle biopsy for halothane caffeine contracture test as per the protocol 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 written on discharge slip. Throughout the postoperative period patient remained afebrile and his urine remained clear. 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. Postoperative recovery was uneventful.

Discussion:

Succinylcholine has been frequently used for crash induction and rarely may it 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., fibreoptic nasotracheal intubation[11] and/or surgical cricothyrotomy have been used following MMR in emergency cases. LMA have also been used successfully in the event of succinylcholine induced MMR[12]. 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 ELM.[,13] 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].Apart from interference with oral intubation MMR has been said to be an early indication of MH.[1]

Muscle spasm following succinylcholine should raise suspicion of MH as it presages clinical MH in up to 30% of cases. However, MMR did not lead to MH in our 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[15]. Triggering agents of MH should be avoided and anesthesia can be continued if the EtCO2, ABG's, BP, HR, temperature, serum CPK, and 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[16]. 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. 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. However, maximum level of CPK, 1339 U/L on day 2 following major surgery [17] has also been reported. Serum CPK generally peak 6-12 hours after MMR. Increase in succinylcholine induced masseter muscle rigidity .In our case CPK showed a moderate rise up to 280U/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.[18] 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 4.6 meq/l. Muscle biopsy for microscopic examination and halothane caffeine contracture test was advised as per the protocol.Cases have been reported with negative caffeine halothane contracture test who had a known clinical episode of MH under anesthesia.[19] 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.[20]. All these causes were ruled out in our case. MMR and MH have been reported in patients with no muscular disease as in our case. Sometime, unsuspected temporomandibular joint (TMJ) dysfunction had been mistaken for MMR, But this possibility was ruled out in this case by the absence of inability to open the mouth to >1 cm and the absence of firmly approximated teeth in the post-operative period. Based on clinical and laboratory findings the case was diagnosed as succinylcholine induced isolated MMR.

Conclusion:

succinylcholine may produce isolated MMR leading to difficult laryngoscopy and intubation. Airway must be secured immediately with 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. Patient advised for muscle biopsy done for halothane caffeine contracture test to rule out MH and also explained about possible risk on subsequent exposure to general anesthesia.

Reference:

[1.] Bauer S J, Orio K, Adams B D. Succinylcholine induced masseter muscle spasm during rapid sequence intubation may require surgical airway. Emerg Med J 2005; 22: 456-58. [PubMed] [Free Full text]

[2.] Onyeka C V. Masseter muscle rigidity atypical malignant hyperthermia presentation or isolated event? Saudi J Anaesth 2010; 4: 20502. [PubMed] [Free Full Text]

[3.] Gopalakrishnan CV, Suparna B, Arun V. A rare case of malignant hyperthermia in the Indian subcontinent. Anaesthesia. 2010;65:1141–2. [PubMed] [Google Scholar]

[4.] Fitzpatrick LR. Masseter muscle rigidity, elevated creatine kinase, and rhabdomyolysis following succinylcholine administration: a case report. AANA J. 2008;76:349–54. [PubMed]

[5.] Bauer SJ, Orio 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.

[6.] Looi I, Bakar AA, Lim C H, Khoo TH, Samuel PE. Anaesthetists' Nightmare: Masseter Spasm After Induction in an Undiagnosed Case of Myotonia Congenita. Med J Malaysia 2008;63(5):425. [PubMed]

[7.] Gill M, Graeme K, Guenterberg K. Masseter spasm after succinylcholine administration. J Emerg Med 2005;29 (2):167-71.

[8.] Saxena K, Dua C K. Malignant Hyperthermia-A case report. Indian J Anaesth. 2007;51:53435. [PubMed]

[9.] Larach M G. Standardization of the caffeine halothane muscle contracture test. North American Malignant Hyperthermia Group. Anesth Analg 1989;69:511–5. [PubMed] [Free Full Text]

[10.] Woodward KT. Patient 37. In: Duke JC, editor. Anesthetic pearls. Philadelphia: Hanley and Belfus Inc;2003. p.151-54.

[11.] Hopkins P M. Malignant hyperthermia: advances in clinical management and diagnosis. Br. J Anaesth. 2000;85:118-28. [PubMed] [Free Full Text]

[12.] Gill M, Graeme K, Guenterberg K. Masseter spasm after succinylcholine administration. J Emerg Med 2005;29:167-71. [PubMed]

[13.] Des Ordons AR, Townsend DR. Trachlight management of succinylcholine-induced subluxation of the temporo-mandibular joint: a case report and review of the

literature. Can J Anaesth 2008;55:616-21. [PubMed]

[14.] Ummenhofer WC, Kindler C, Tschaler G, Hamphl KF, Drewe J, Urwyler A. Propofol reduces succinylcholine induced increase in masseter muscle tone. Can J Anaesth. 1998;45(5 Pt 1):417-23. [PubMed]

[15.] Roy S, Fortier LP. Fentanyl-induced rigidity during emergence from general anesthesia potentiated by venlafexine. Can J Anaesth. 2003;50:32-5. [PubMed]

[16.] Sansone RA, Sansone LA. Tramadol Seizures, Serotonin Syndrome, and Coadministered Antidepressants. Psychiatry (Edgmont) 2009;6(4):17–21. [PubMed] [Free Full Text]

[17.] Muldoon S M, Karen S M. Hyperthermia and hypothermia. In Roger M C, Tinker J H, Conino B G, Lonnecker D E Eds. Principles and Practice of Anaesthesiology. St Louis; MO Mosby-Year Book 1993: 2503.

[18.] Roesnberg H. Trismus is not trivial. Anesthesiology 1998;67:453-56. [PubMed] [Free Full Text]

[19.] Berry F A, Lyrach C 3rd. Succinylcholine and trismus. Anesthesiology 1989;70(1):161-63. [PubMed] [Free Full Text]

[20.] Fitzpatrick LR. Masseter muscle rigidity,elevated creatine kinase,and rhabdomyolysis following succinylcholine administration:a case report. AANA J. 2008;76:349-54. [PubMed.