IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 22, Issue 1 Ser.6 (January. 2023), PP 34-36
www.iosrjournals.org

Malignant Hyperthermia- An Unusal Case Report

Dr. Vipul Bansal[1], Dr. Melchisedec[2], r. Narjeet Osahan[3], Dr. Satinderjit Kaur[4]

ABSTRACT
Malignant Hyperthermia is a potentially fatal condition which occurs after an exposure to certain triggering agents in susceptible individuals. There is a huge variation in the order and timing of the symptoms presentation which makes the diagnosis difficult. Here is a case report of a 51-year-old gentleman posted for incision and drainage of abscess presenting with MH.

KEY WORDS
MH, sevoflurane, succinylcholine, general anesthesia

Date of Submission: 05-01-2023 Date of Acceptance: 19-01-2023

I. INTRODUCTION

Malignant Hyperthermia is a hypermetabolic syndrome that develops in susceptible individuals when exposed to triggering agents like volatile anesthetic gases (Sevoflurane, isoflurane, halothane), Succinylcholine, vigorous exercise and heat [1,2]. It presents with tachycardia, hypercapnia, tachypnea, acidosis along with high grade fever. In this case report we present a case of MH after exposure to sevoflurane and succinylcholine.

II. CASE REPORT

51-year-old male was posted in emergency for incision and drainage of right Parotid abscess and Para pharyngeal and Retro pharyngeal abscess. On Pre-anesthetic evaluation, patient was a diabetic on regular medications and had no previous anesthetic exposure. On examination, patient was average built with weight of 82kg, short neck, double chin, mouth opening was adequate with MPS III and restricted neck extension, suggesting anticipated difficult airway.

Before induction, his blood pressure(NIBP) was 124/82 mm Hg, Heart rate(HR) 88 beats per minute, Temperature 98.4°F, Oxygen saturation(SpO_2) 98% on room air and respiratory rate 15 breaths per minute. Blood sugars were 356 mg/dL which were corrected with insulin.

After pre oxygenation for 3 minutes, patient was induced with injection fentanyl (75µg), lidocaine (60mg), propofol (160mg) and succinylcholine (100mg) intravenously. Patient was intubated with cuffed endotracheal tube no. 6.5 using BURP technique and a bougie. After tube confirmation, injection atracurium(25mg) intravenously was given. Anesthesia was maintained with oxygen, sevoflurane and nitrous oxide. Surgery continued for 50 minutes. All vital parameters including NIBP, HR, SpO_2, Electrocardiography(ECG), $EtCO_2$ and temperature were normal throughout the surgery. At the end of the surgery, Sevoflurane and nitrous oxide was stopped, patient reversed with intravenous injection neostigmine (3mg) and glycopyrrolate (0.6mg). Patient was extubated once he started breathing spontaneously and was able to follow commands.

After 5 minutes, patient became apprehensive, tachypneic and started desaturating. On auscultation, there was decreased air entry in all lung fields. Bag and mask ventillation started. There was increased resistance in manual ventilation. Diagnosis of Laryngospasm was made. He was then ventilated with 100% oxygen, injection propofol (40mg) was given intravenously. Saturation could not be picked up. Injection Succinylcholine (25mg) was given intravenously. Resistance during ventilation reduced and the $SpO_2$of 99% was attained.

After 30 minutes of manual ventilation, patient didn't attain spontaneous ventilation. His vitals were NIBP- 146/100 mm Hg, HR- 164/min, SpO_2- 96%, $EtCO_2$-65mm Hg, Temperature- 104°F and RBS- 251mg/dL. Blood gases were sent which reported pH= 6.818, PaO_2=85.5mm Hg, $PaCO_2$= 141mm Hg, HCO_3= 21.6mmol/L, ABE= -19.5mmol/L, SBE= -11.7mmol/L, Saturation=94.9%, lactates= 7.7mmol/L. MH was suspected. Circuits and Sodalime was changed, patient was reintubated and shifted to Intensive Care Unit.

In Intensive Care Unit, Patient was electively hyperventilated. Injection fentanyl (30µg/hour) was started for sedation. Active cooling in view of hyperthermia was done using cold sponging, fanning and cold iv fluid. Blood investigations were done which showed increased LDH levels (400 IU/L), elevated Creatinine kinase (81 IU/L), deranged renal parameters with serum urea 59mg/dL, serum creatinine 1.87mg/dL, normal serum electrolytes and thyroid function tests.

Patient had a BP recording of 60/40mm Hg and HR 140/min. Injection Noradrenaline was started as infusion. Cardiac evaluation was done in ICU, ECG showed sinus tachycardia & prolonged QT_c and

Echocardiography(ECHO) showed Normal sized cardiac chambers, Global Hypokinesia, mild MR, TR with left ventricular ejection fraction(LVEF) 30%.

After overnight corrective measures, ABG showed pH= 7.22, PaO_2= 138mm Hg, $PaCO_2$= 39.2mm Hg, HCO_3= 15.6mm Hg, BE= -6.3mmol/L, saturation=98.4%, lactate= 4.5mmol/L. A temperature of 98.8°F and HR of 110/min was attained. Injection Noradrenaline was tapered and stopped on postop day 2 following which patient was extubatedand shifted to ward on subsequent day.

Repeat blood investigations showed elevated serum creatinine and urea levels which gradually decreased over days with fluid therapy and diuretics. Creatinine kinase also followed a decreasing trend.

Later, ECG showed normal sinus rhythm and ECHO findings as Normal sized cardiac chambers, moderate TR, mild MR, trivial AR with mildly reduced cardiac contractility and LVEF 50%.

Since MH is rare finding and there were no definitive tests available to confirm the diagnosis in our institution, we made our diagnosis based on clinical findings and grading scale suggested by Larach et al, the RAW score which was 53 in our patient and MH rank was 6.

DISCUSSION

MH is a hypermetabolic syndrome that develops in susceptible individuals when exposed to triggering agents like volatile anesthetic gases (Sevoflurane, isoflurane, halothane), Succinylcholine, vigorous exercise and heat [2,3]. The incidence of MH during general anesthesia is estimated to range from 1:5000 to 1:50000 in individuals[4]. However, genetic prevalence to develop MH is up to 1:2000 because of its autosomal dominant trait. Reaction develops more commonly in males then females (2:1).

Very few cases have been reported so far in India and there is widely held belief that this is a disease of the western world. Saxena et al[5] reported first case of MH in India.

During an episode of MH, clinical manifestations are due to cellular hypermetabolism, leading to sustained muscular contraction and breakdown, anaerobic metabolism, acidosis, and their sequelae. Early manifestations are metabolic (elevated $EtCO_2$, increased oxygen consumption, hyperkalemia, respiratory acidosis), cardiovascular like cardiac arrhythmias and unstable blood pressure. Late manifestations are hyperkalemia, a rapid increase in core body temperature, grossly elevated serum creatinine phosphokinase and myoglobin, dark colored urine, arrhythmias, cardiac arrest and disseminated intravascular coagulation.

In the above case, patient was exposed to two of the most common triggers of MH namely sevoflurane and succinylcholine. Other drugs which were used, namely fentanyl, propofol, nitrous oxide and atracurium are not known triggers for MH. Here, MH developed 85 minutes after the induction of anesthesia. Many cases have been reported with delayed onset of MH after exposure of sevoflurane. Kinouchi et al[6] reported development of MH about 40 minutes after induction of anesthesia with sevoflurane in a 9 month old boy undergoing accessory ear resection. Kim et al[7] described MH developing 95 minutes after initiation of general anesthesia with sevoflurane in a 77 year old male posted for enucleation of prostate for benign prostatic hyperplasia. Chen et al[8] reported similar case of delayed onset after 90 minutes of induction with sevoflurane in a 5 year old boy for repeat orthopedic surgery. It has been postulated that the onset of MH after exposure to sevoflurane may be associated with release of calcium ion from sarcoplasmic reticulum. Delayed effects is attributed to latent effect of sevoflurane on muscles.

There is possibility that the triggering event in this could also be the second dose of succinylcholine which was given to treat laryngospasm. PM Hopkins[9] suggests succinylcholine has been implicated in causing MH alone as well as when used with volatile anesthetic agents. Chen et al[8] reported MH happened in subsequent episode of exposure to drug when the patient has no such event in the prior anesthetic exposure.

In a study conducted in North America, it was found that earliest symptoms of MH were hypercapnia (92%), sinus tachycardia (73%) and masseter muscle rigidity (27%) [10]. In 63.5% cases, temperature abnormality was one of the first 3 signs [10]. In the above study first suspicion came with the finding of fever and tachycardia, based on which blood gases were sent. Hypercapnia with mixed respiratory and metabolic acidosis strengthen the suspicion.

Due to the presence of hyperthermia, hypercapnia and sinus tachycardia, differential diagnosis of Phaeochromocytoma, thyroid storm and neuroleptic syndrome were considered. Phaeochromocytoma was ruled out since the blood pressure recordings remained within normal limits during preoperative and intraoperative period. Also ultrasonography of abdomen didn't show any relevant findings. Neuroleptic syndrome was less likely based on the absence of history of any medications implicated for this condition. Due to normal values of thyroid function tests and absence of features of enlarged thyroid gland or thyrotoxicosis, thyroid storm was also rules out. Thus diagnosis of MH was made. This was confirmed based on the MH clinical scale suggested by Larach et al[11]. In this case, we attained a raw score of 53[table 1], which denotes a MH rank of 6[Table 2]. It indicates the diagnosis of MH was almost certain.

The gold standard for diagnosing MH is Caffeine halothane contracture test (CHCT) [12]. It involves exposing a sample of live muscle fiber to halothane and caffeine to determine muscle response to halogenated anesthetics. Since the test is not available widely, the burden of diagnosis of MH lies on clinical scale by Larach et al[11].

There are also genetic tests for detection of mutations of RYR1 or associated genetic variants associated with MH. MH is inherited as an autosomal dominant trait. Majority of case reported are caused by mutation of RYR1 and CACNA 1s gene[13]. Both these genes are related to calcium ion regulation inside sarcoplasmic reticulum in skeletal muscles. The abnormal ryanodine receptors produced by mutation of these genes causes excessive release of calcium ions once triggered by specific triggering agents.

The treatment of choice for MH is Dantrolene Sodium which is a calcium channel blocker. The drug was not available in the pharmacy. So it could not be used for treatment as well as confirmation of episode of MH.

III. CONCLUSION

We have encountered a full blown episode of MH with signs and symptoms. It was confirmed based on clinical scoring but could not be supported by definitive tests due to unavailability. The episode could have a delayed onset after exposure to sevoflurane. Also, the possibility of the incidence happening after subsequent dose of succinylcholine could not be ruled out.

REFERENCES

[1]. Mathur PR, Rundla M, Jain N, et al. MH in a 6-month-old infant. Saudi. J. Anaesth. 2016;10:353-5
[2]. Arcangeli M, Feola A, Marsella LT. MH: A case report. Acta Med. Mediterr. 2013;33:807-09
[3]. Ferhi F, Dardour L, Tej A, et al. MH in a 4-year-old girl during anesthesia induction with sevoflurane and succinylcholine for congenital ptosis surgery. Saudi J. Ophthalmol. 2019;33:183-87
[4]. Kim DC. MH. Korean J Anaesthesiol. 2012 Nov;63(5)391-401
[5]. Saxena, Kirti N., Dua, et al. MH- a case report. Indian J. Anaesth.2007 Nov-Dec;51(6):534-35
[6]. Kinouchi K, Okawa M, Fukumitsu K, et al. Two pediatric cases of MH caused by sevoflure. Masui. 2001 Nov;50(11)48-56
[7]. Kim SJ, Hong SH, Chung MY, et al. Delayed onset MH in postanesthetic care unit: A case report. J. Int. Med. Res. 2021 Sep;49(9):03000605211044201
[8]. Chen PL, Day YJ, Suu BC, et al. Delayed onset of sevoflurane-induced juvenile MH after second exposure. ActaAnaesthesiolTaiwa. 2007 Sep;45(3):183-93
[9]. Hopkins PM. MH: pharmacology of triggering. Br J Anaesth. 2011 Jul;107(1):48-56
[10]. Larach MG, Localio AR, Allen GC, et al. Clinical presentation, treatment and complications of MH in North America from 1987 to 2006. AnaesthAnalg. 2010;110:498-507
[11]. Larach MG, Localio AR, Allen GC, et al. A clinical grading scale to predict MHsusceptibililty. Anesthesiology. 1994 Apr;80(4):771-9
[12]. Rosenberg H, Davis M, James D, et al. MH. Orphanet J Rare Dis. 2007 Apr;24(2):21
[13]. Rosenberg H, Pollock N, Scheimann A, et al. MH: a review. Orphanet J Rare Dis. Aug 2015;93:10

Table 1: MH score in our patient

Clinical Indicator	Points
$PaCO_2$>60 with controlled ventilation	15
Rapid increase in temperature	15
Inappropriate tachycardia	3
Arterial base excess more negative than -8mEq/L	10
Arterial Ph	10
Total Points	53

Table 2: Raw score, MH rank and their prediction

Raw Score	MH Rank	Prediction
0	1	Almost Never
3-9	2	Unlikely
10-19	3	Somewhat less than likely
20-34	4	Somewhat greater than likely
35-49	5	Very likely
>50	6	Almost certain

XXXXX, et. al. "Malignant Hyperthermia- An Unusal Case Report." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 22(1), 2023, pp. 34-36.