Case Report

Malignant hyperthermia in a 4-year-old girl: A rare but lethal anesthetic emergency: A case report

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ABSTRACT

Malignant hyperthermia (MH) is a rare but serious life-threatening complication of drugs involved in general anesthesia. It involves the release of calcium when exposed to triggers such as depolarizing muscle relaxants (succinylcholine) or volatile anesthetic agents (such as halothane, sevoflurane, desflurane). It occurs when a patient with a mutation in the ryanodine or dihydropyridine receptor genes is exposed to neuromuscular blocking agents. We present a case of suspected MH in a child being operated on for developmental dysplasia of the hip. The development of MH in response to depolarizing neuromuscular blockers is a very rare phenomenon, but should remain on the list of differential diagnoses in the setting of a rapid rise in temperature intraoperatively.

Key words: General anesthesia, Isoflurane, Life-threatening, Malignant hyperthermia, Succinylcholine

Malignant hyperthermia (MH) is an autosomal dominant disorder that occurs due to mutations in either the ryanodine receptor subtype 1 (RYR1) or dihydropyridine receptor genes of skeletal muscles. It is a fatal condition that presents as a hypercatabolic myopathy when the patient is administered succinylcholine or volatile inhaled anesthetics. It has an incidence of 1:40,000 administered anesthetics [1]. MH crisis may develop at the very first exposure to anesthetic agents known to trigger an MH episode, or may occur after multiple exposures. Females are more prone to crisis than males (2:1), and all ethnic groups of the world are affected. The highest incidence is in young people, and it has been found that children under 15 years of age comprised 52.1% of all reactions [2]. The pathophysiologic changes of MH are due to an uncontrolled increase in myoplasmic calcium, which causes biochemical processes related to muscle activation. This process causes an increase in metabolic rates, leading to tachycardia and hypercarbia, which are the earliest clinical manifestations of MH. Due to ATP depletion, the muscle membrane integrity is compromised, which leads to hyperkalemia and rhabdomyolysis. Over 90 mutations have been identified in the *RYR-1* gene located on chromosome 19q13.1, and at least 25 are causal for MH [3]. MH is primarily diagnosed on clinical grounds, with definitive confirmation obtained through the caffeine–halothane contracture test or targeted genetic analysis when available. Management of a suspected episode requires immediate institution of supportive therapy, including discontinuation of all triggering anesthetic agents, administration of 100% oxygen with controlled hyperventilation, replacement of the anesthesia breathing circuit and carbon dioxide absorbent, and initiation of definitive pharmacological treatment as indicated [4]. Dantrolene is the only known reversal agent for MH, which exerts its effect by binding to the RYR1 in order to stop unregulated release of calcium [5].

Here, we report a rare but life-threatening case of MH in a 4-year-old child because of its unique presentation and the diagnostic and therapeutic challenges it posed, highlighting the importance of early recognition and timely management.

CASE PRESENTATION

A 4-year-old female child presented with a case of developmental dysplasia of the hip. She was scheduled for surgical correction under general anesthesia after preoperative evaluation. She was found to be of 15 kg in weight, with no previous history of any surgery. Laboratory investigations were within normal range, and American Society of Anesthesiologists

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grading I. Anesthetic induction was performed with intravenous midazolam (1 mg) and propofol (2 mg/kg). Neuromuscular relaxation was done with succinylcholine (2 mg/kg). She was intubated with a 5.5 mm uncuffed endotracheal tube and taken on mechanical ventilation on a closed circuit. General anesthesia was maintained with isoflurane, and paralysis was maintained with atracurium with an initial dose of 0.5 mg/kg and a maintenance dose of 0.05 mg/kg. In the 1st h of surgery, the patient's vitals were normal with mean arterial pressure (MAP) (69), heart rate 90/min, and end-tidal carbon dioxide ($ETCO_2$) 36–38 mmHg. Ventilator settings of volume control with TV of 120 mL, Respiratory rate of 25/min with fraction of inspired oxygen (Fio_2) of 50% at 1.5 L/min, and nitrous oxide 50% at 1.5 L/min was set.

After an hour, the patient developed tachycardia with a pulse rate (PR) of 170/min. The patient was given a maintenance dose of atracurium and propofol to deepen the sedation. There was an increase in peak pressure of the ventilator even after giving a proper dose of muscle relaxant. The patient's endotracheal tube position was confirmed to check for any kink or obstruction. On examination, there was jaw stiffness, which causes tube obstruction by clenching of teeth. The patient was febrile on touch. Intravenous paracetamol was given at a dose of 10 mg/kg, and ice packing was placed in the axilla. Monitoring shows MAP of 78, PR of 174/min, and an increase in $ETCO_2$ of 126 mmHg. Ventilator settings were changed to TV 140 mL, respiratory rate of 35/min, and fiO_2 of 80%. The patient's arm was found to be in a flexed state, and the patient was still febrile. After a few minutes, even after an increase in minute ventilation, monitoring showed $ETCO_2$ of 137 mmHg, PR: 174/min and body temperature of 101°F.

A provisional diagnosis of MH was made, and isoflurane and nitrous oxide were immediately discontinued. The patient was taken off the closed circuit and ventilated with a Jackson Rees circuit with fiO_2 100% at 6L/min. Total intravenous anesthesia (TIVA) was maintained with propofol at 250 mcg/kg/min. Arterial blood gas (ABG) analysis was done, which showed pH 7.02; partial pressure of arterial carbon dioxide ($paCO_2$) 83.9; partial pressure of oxygen (PaO_2) 308; base excess −10; bicarbonate 21.2.

A decision was made in accordance with the physician to administer dantrolene sodium, but it was not available in our institute. Hence, it was decided to continue the surgery on TIVA. Patient was ventilated with Jackson Rees circuit with fiO_2 100% at 6l/min and respiratory rate of 35/min. After 1 h, the patient's body temperature dropped to 100°F and ice packing was continued along with intravenous cold normal saline. Patient's vitals were MAP: 70 mmHg; PR: 177/min, and $ETCO_2$ reduced to 63 mmHg.

At the completion of surgery, the patient was shifted to intensive care. The patient was taken on mechanical ventilation in volume control mode, and regular monitoring of $ETCO_2$ was done. Twelve hours postoperatively, the patient showed generalized improvement with normalization of body temperature. Post-operative ABG was done after 8 h, which showed pH 7.29; $PaCO_2$ 35; PaO_2 155; base excess −9.1; bicarbonate 16.6.

Patient regained consciousness and was successfully extubated after a weaning off trial and ABG. The patient was kept in the intensive care unit (ICU) under observation for the next 2 post-operative days, which were uneventful. She was discharged on the 5th day with a diagnosis of MH being explained to the family for any future need of surgery. In routine follow-up after a week of discharge, the patient did not show any kind of sequalae and was living a healthy life.

DISCUSSION

MH is defined as a hypermetabolic response that occurs after exposure to volatile inhaled anesthetics such as isoflurane, halothane, sevoflurane, and depolarizing neuromuscular agents such as succinylcholine [6]. MH has an autosomal dominant pattern of inheritance with mutations in any of three genes: RYR1, CACNS1S (dihydropyridine receptor), and STAC3 [7]. The maximum number of cases is estimated to be caused by mutations in the *RYR1* gene. Since patients susceptible to this disorder do not present with any external signs preoperatively, identification of such patients at risk is more difficult. The earliest symptoms of MH crisis include the masseter spasm with hypercapnia and tachycardia. Hypercapnia can be considered the most specific symptom, which occurs in more than 90% of cases [8].

The time between the exposure to triggers and the development of symptoms varies depending on the anesthetic drugs used. This time is shorter when the volatile inhalation anesthetic is administered along with succinylcholine. MH event may also occur after succinylcholine as the only trigger used. In our case, the use of succinylcholine along with the use of isoflurane triggers the initial symptoms of masseter spasm.

MH crisis can be differentiated from neuroleptic malignant syndrome (NMS) as non-depolarizing muscle relaxants will result in a partial or complete resolution of muscle rigidity in a patient suffering from NMS. The diagnosis of MH can be assessed by using a clinical grading scale by Larach, with a score above 50 signifying an almost certain MH event (Table 1) [9].

In our case, we have a score of 58 due to the unavailability of an advanced laboratory setup and dantrolene sodium. If there is suspicion of MH, any volatile agents should be discontinued first, and the patient should be hyperventilated with 100% oxygen. The breathing circuit and carbon dioxide absorber of the anesthesia workstation should be changed if there is no sign of improvement in hemodynamics. If it is not possible to abort the surgery, anesthesia should be maintained with total intravenous anesthesia, and surgery

Table 1: Criteria used in the clinical grading scale for malignant hyperthermia

Clinical finding	Manifestation
Respiratory acidosis (15)	$ETCO_2$ >55 mmHg, $PaCO_2$ >60 mmHg
Cardiac involvement (3)	Unexplained sinus tachycardia, ventricular tachycardia, or ventricular fibrillation
Metabolic acidosis (10)	Base deficit >8 mEq/L, pH <7.25
Muscle rigidity (15)	Generalized rigidity, severe masseter muscle rigidity
Muscle breakdown (15)	Serum creatine kinase concentration >20,000/L units, cola-colored urine, excess myoglobin in urine or serum, plasma (K+) >6 mEq/L
Temperature increase (15)	Rapidly increasing temperature, T >38.8°C
Others	Rapid reversal of malignant hyperthermia signs with dantrolene (score=5), elevated resting serum creatine kinase concentration (score=10)
Family history (15)	Consistent with autosomal dominant inheritance

$ETCO_2$: end-tidal carbon dioxide, $PaCO_2$: Partial pressure of arterial carbon dioxide

should be completed as soon as possible [10]. When a crisis of MH is suspected, 2.5 mg/kg of dantrolene sodium should be administered intravenously immediately. After the surgery, the patient should be transferred to the ICU for observation for at least 24 h. To prevent any relapse, 1 mg/kg of dantrolene sodium should be given intravenously every 6 h for the next 24–48 h from the appearance of first symptoms. Measures should be taken to correct hyperkalemia, hyperthermia, acidosis, hypoxemia, arrhythmias, and preserve renal function. Complete recovery from the MH crisis can occur if the signs and symptoms of MH are recognized early and proper treatment is started. The mortality rate is <5%.

There are only a few reported cases of MH in India that survived this ordeal with early diagnosis and supportive management. In 2007, Kirthi and Saxena published a case of MH where the patient survived, and the triggering agents used were halothane and succinylcholine [11]. In 2010, Gopalakrishnan *et al*. reported a similar case of MH, and the agent used was sevoflurane [12]. In 2017 and 2019, Iqbal *et al*. and Ramanujam *et al*. published cases with almost certain diagnosis of MH, respectively [13,14]. All these reported cases that survived were symptomatically managed without having received the drug of choice, that is, dantrolene sodium.

The successful management of MH without dantrolene focuses on the need for strict monitoring, early detection and immediate and effective treatment. Essential monitoring of temperature and $ETCO_2$ should be used to detect the early warning signs in such cases where general anesthesia is used.

CONCLUSION

This case demonstrates that strict monitoring of patients should be done where volatile anesthetics and other triggering agents are used. Although dantrolene sodium may be unavailable in some small cities and setups, early detection and proper supportive care can save the life of a patient. Still, strong advocacy should be done for the availability of Dantrolene and advanced lab facilities to confirm diagnosis and management in the future.

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