

Malignant Hyperthermia during Desflurane - Succinylcholine Anesthesia for Neurosurgery: A case report

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Citation

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Abstract

Desflurane has been known as a weak triggering anesthetic of malignant hyperthermia (MH). It may produce a delayed onset of symptoms. In addition the use of succinylcholine may aggravate occurrence of MH. The prolonged interval after exposure may occur more than 3 h after the induction of anesthesia. Although, the treatment of MH with Dantrolene is gold standard, it is not available in most countries. Because, MH has been rarely reported all over the world. So we present the first MH suspected case in our hospital.

INTRODUCTION

Malignant hyperthermia (MH), an anesthetic related disorder of skeletal muscle calcium regulation, is triggered by succinylcholine and volatile anesthetics. It is characterized by hypercarbia, hyperthermia, tachycardia, acidosis, and muscle rigidity (1, 2). The common use of desflurane anesthesia all over the world has changed the clinical presentation of malignant hyperthermia.

We describe a case of suspected MH in adult patient in whom succinylcholine and desflurane were the only MH triggering drugs administered.

CASE DESCRIPTION

A 49-yr-old female patient, 80 kg, ASA physical status II, was scheduled for total cervical laminectomy. There was no known family history of malignant hyperthermia or muscle disease. So far, the woman was healthy and had already been anesthetized for major surgery twice (lumbar laminectomy, hepatic hydatid cyst) without any problem.

One hour after premeditation with 0.07 mg midazolam intramuscularly (IM). After breathing 100% oxygen, anesthesia was induced with fentanyl (2 µg/kg), propofol (2 mg/kg), and succinylcholine (1 mg/kg) intravenously. The trachea was intubated (spirally endotracheal tube), and controlled ventilation was initiated using a circle anesthesia system with a soda lime CO₂ absorber. Anesthesia was

maintained with O₂, air, desflurane (4.5%–6%), and intermittent boluses of fentanyl and rocuronium. In addition to standard monitoring, an arterial line (radial artery) and a central venous catheter (internal jugular vein) were inserted. A Foley catheter was introduced to measure urine output. Baseline heart rate was 68 bpm, peripheral oxygen saturation (SaO₂) 98%, end-tidal CO₂ (etCO₂) 29 mm Hg, and skin temperature 36.4°C. The patient was then turned into prone position to perform the cervical release.

Three hours after the induction, severe hypercarbia developed, with an increase in end-tidal CO₂ to 79 mm Hg. Firstly we checked the tracheal tube and auscultated the lungs. The patient began profusely sweating, his axillary temperature reached 39.6°C, and (we could not notice the muscle rigidity because of prone position). Twenty minutes after the first symptoms, a severe respiratory acidosis was diagnosed, with arterial pH 7.11, PaCO₂ 74 mm Hg, PaO₂ 100 mm Hg, and base excess 6.7 mEq/L. Fresh gas flow was increased to 8 L/min. We suspected the patient had malignant hyperthermia (MH).

Desflurane was turned off, and hypnosis was maintained by propofol. We warned to neurosurgeon to finish (completed) the operation as soon as possible. Fresh gas flow was adjusted to 10 L/min. Soda lime and tubes of the ventilation system were replaced. Hypercarbia could not be sufficiently corrected by hyperventilation. The patient became more and

more hemodynamically unstable. Tachycardia up to 150 bpm, ST segment alterations. At this time, body temperature was 40,2°C.

Dantrolene is not generally available in Turkey, because MH occurs very rarely heard of in Turkish patients. Although we could not give dantrolene, we instituted the following measures: a) discontinued the triggering anesthetic (desflurane) and operation immediately; b) initiated a non-triggering anesthetic (propofol infusion at 20-30 mg/h); c) provided 100% oxygen; d) exchanged the entire anesthetic circuit; e) increased the fresh gas flow rate to 10 L/min to prevent re-breathing; f) measured the esophageal temperature; g) began aggressive cooling (ice packs and cooling); i) administered sodium bicarbonate as indicated by blood gas analysis; j) Forced diuresis was induced with mannitol, furosemide and dexametasone to preserve renal function and brain edema.

Forty five minutes after the life-saving interventions, the patient's esophageal temperature decreased to 37.2°C. One hour later, the patient was transferred to the intensive care unit and continuously observed by an anesthesiologist for further management. In the ICU sedation was provided with fentanyl and propofol infusion. Temperature and ETco2 were closely monitored. The endotracheal tube was removed in the intensive care unit 4 h after admission. Vital signs remained stable for the next 24 h. The creatine phosphokinase peak was 36,761 U/L 26 h postoperative second day. However at that time potassium was 2,6 mEq/L (Table 1). The patient was discharged from ICU at seventh day of surgery with no hemodynamic complications, likelihood improvement of laboratory outcomes to neurosurgical ward.

Postoperative blood CPK, myoglobune, LDH Levels

Figure 1

Date	CPK	myoglobin	LDH
Day of surgery	2597	>800	400
Day of surgery	3313	>800	600
POD 1	36761	>800	1118
POD2	20252	790	1300
POD3	9383	477	1638
POD4	5003	360	1068
POD 5	1569	183	408
POD 6	713	120	396
POD 7	504	114	368

POD: Postoperative day

CPK: Creatine Phosphokinase

LDH: Lactat-Dehydrogenase

DISCUSSION

Malign hyperthermia is a rare (1/15000 in pediatric patients and 1/40000 adult patients) myopathy, characterized by an acute hyper-metabolic state within muscle tissue following induction of general anesthesia (3). In our hospital from 1998 to 2007 years, this is the first MH case in 16.099 patients exposure to general anesthesia.

In experimental and clinical studies have shown that role of desflurane as a trigger agent of MH, and it is contraindicated in susceptible patients, sharing that characteristic with other volatile anesthetics (4,5,6). Like desflurane, succinylcholine is well known trigger agent of MH too.

In our case, during the first two hours of operation temperature, oxygenation and hemodynamic values were normal, after that elevated etCO₂ was the first evidence of difficulties. Because the patient position was prone, we did not observe even if masseter spasm was occurred. When hemodynamic instability began and temperature increased, we assumed the patient had MH. Another important issue was the past history of the patient. She had already been two operations via general anesthesia without any sign.

Under these conditions Dantrolene, which is the only rescue drug for MH, must be readily available. As a conclusion; we thought that although the treatment of MH specifically depends on Dantrolene, it should be avoided early increased intracranial pressure related to hyperthermia which is primer result of morbidity and mortality, with out use of

Dantrolene.

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