

A Case Report: Malignant Hyperthermia with Successful Supportive Management

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Abstract: A Case report: malignant hyperthermia with successful supportive management

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Background: Malignant hyperthermia is a serious anesthetic-related disorder. Our case was successfully treated without dantrolene available. A 35-year-old Thai woman presented with thyroid nodule. She was scheduled for thyroid lobectomy. Anesthesia was induced by thiopental and morphine then tracheal intubation was facilitated by succinylcholine. Jaw stiffness was sensed but trachea was intubated successfully. Anesthesia

was maintained with sevoflurane and cis-atracurium. Tachycardia and hypercapnia followed by hyperthermia led to the diagnosis of malignant hyperthermia. The patient was treated promptly and further investigated to rule out other conditions.

Keywords: *Malignant hyperthermia, supportive management*

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Introduction

Malignant hyperthermia (MH) is a rare, but critical, autosomal-dominant inherited disorder that may lead to metabolic crisis of skeletal muscle in susceptible individuals following exposure to anesthetic triggering agents, such as volatile anesthetics or depolarizing muscle relaxants.¹

Ones susceptible to MH, the ryanodine receptor (RyR) in skeletal muscle is abnormal.² This interferes with regulation of calcium in the muscle causing a buildup of calcium in skeletal muscle. Massive metabolic reaction occurs. Early clinical signs include muscle rigidity, tachycardia, increase end-tidal carbon dioxide (EtCO₂), tachypnea, and hyperkalemia. The later signs include fever, myoglobinuria, and multiple organ failure.³

Case report

A 35-year-old woman presented with 2 centimeter-width of thyroid nodule for 3 years. She was unsuccessfully treated with eltroxin for 3 months in other hospitals. Her thyroid nodule size did not reduce after treatment. Her last thyroid function test, a month before the surgery, showed hyperthyroid state presenting normal FT3 (4.05 pg/mL, normal range 2.4-6.8 pg/mL), elevated FT4 (1.71 ng/dL, normal range 0.6-1.6 ng/dL) and suppressed TSH (<0.0025 uIU/mL normal range 0.3-5.0 uIU/mL) without clinical hyperthyroid symptoms. She was referred to our hospital to undergo subtotal thyroidectomy. She had no previous general anesthesia and had no known family history of anesthetic-related death or serious complication.

On admission, one day before surgery, she was 150 cm in height and weighed 50 kg. Her blood pressure was 100/60 mmHg, pulse rate was 90-110 beats per minute with a regular rhythm, and body temperature was 37.2°C. A 2 centimeters thyroid mass was palpable without tenderness or lymphadenopathy. She had no lid retraction or exophthalmos. Routine preoperative physical and laboratory examinations were normal but thyroid function test was not repeated.

The patient did not receive premedication. On the arrival, her heart rate and systolic/diastolic blood pressure were 86 beats per minute and 110/70 mmHg, respectively. Oxygen saturation was 98 percent. Anesthesia was induced with the intravenous administration of thiopental (5 mg/kg), morphine (0.12 mg/kg) and succinylcholine (1.5 mg/kg). We sensed jaw stiffness when attempting tracheal intubation under direct laryngoscopy but a 7.0 mm endotracheal tube was inserted successfully. Then, 0.08 mg/kg of cis-atracurium was given. Anesthesia was maintained with 1.5-2% sevoflurane in 50% nitrous oxide/50% oxygen at a total flow rate of 2 L/min. During the anesthesia procedure, an electrocardiogram, pulse oximeter, noninvasive blood pressure monitor including end-tidal CO₂ monitor were recorded but temperature was not monitored. After induction, 1% xylocaine with adrenaline (4 ml, 1:100,000) was injected subcutaneously by the surgeon to reduce bleeding and for early postoperative pain relief.

After intubation, vital signs remained stable but end tidal CO₂ started from 48 mmHg and was

rising continuously. We tried to increase minute ventilation by raising respiratory rate but end tidal CO₂ continued rising to 52 mmHg and her heart rate slightly increased from 86 to 120 beats per minute. The increasing heart rate was considered to cause by local anesthetic containing adrenaline. Thirty minutes later, the patient suddenly developed tachycardia of 155 beats per minute, end tidal CO₂ was 58 mmHg, even though minute ventilation increased from 5.4 L/min to 9 L/min (from tidal volume 450 mL and respiratory rate 12 breaths per minute to the same tidal volume with respiratory rate 20 breaths per minute). Oral temperature from thermometer showed 38°C at first and then the temperature was continuously rising to 38.2°C within 20 minutes. Head and neck sweating were noted. Soda lime absorber was noted to be hot, and its color was changed to purple. We suspected that malignant hyperthermia or thyroid storm had developed. After we informed the surgeon, sevoflurane and nitrous oxide were discontinued, and 100% oxygen was administered through self-inflating bag from the oxygen cylinder at a high flow rate (10 L/min). Midazolam and propofol were given for sedation. The operation was terminated as soon as possible. Active cooling was immediately initiated by tepid sponge. Blood sample was drawn for further investigation and evaluation including complete blood count, blood chemistry, coagulogram, thyroid function test, creatine phosphokinase (CPK) and arterial blood gas. Esmolol was titrated and dexamethasone was administered intravenously. Fifteen minutes after vital signs were stable and end tidal CO₂ returned to normal range, 1.2 mg of

atropine & 2.5 mg of neostigmine were given. Shortly after the patient was fully awake with good ventilation, she was extubated and transferred with nasal oxygen supplementation to recovery room. Trepid sponge with cold water was continued. Foley catheter was inserted to monitor urine output and its color. Two hundred milliliters of dark brown urine flew into the urine bag. Arterial blood gas result showed respiratory alkalosis (pH 7.44, PaCO₂ 27 mmHg, PaO₂ 269 mmHg, bicarbonate 18.4 mmol/L), CPK was elevated (5641 U/L, normal range 0-190 U/L). Intraoperative thyroid function test, blood chemistry and coagulogram were normal. A diagnosis of malignant hyperthermia was made based on hyperpyrexia, elevated EtCO₂, elevated CPK and normal thyroid function test. Dantrolene was not administered, as it was not available at that time. Two hours after the patient was observed in the recovery room, patient's oral temperature was still 38°C, blood pressure was 110/70 mmHg, and heart rate was 100-110 beats per minute and oxygen saturation was 100 percent. The patient was transferred to ward because no intensive care unit was available.

At ward, nephrologist was consulted. The patient was hydrated and alkalized urine. Amount of urine output and urine pH were monitored every 6 hours. CPK increased from 5641 U/L (30-fold the normal level) after anesthesia to 37467 U/L (200 –fold the normal level) 24 hours after induction and decreased steadily. Renal function was preserved. Dark brown urine presented only 12 hours after induction and then

lightened up to normal color. The patient recovered smoothly without complications.

The postoperative period was uneventful and the patient was discharged from the hospital four days after the operation.

Discussion

Thyroid storm and malignant hyperthermia have many clinical similarities.⁴ Several case reports of thyroid storm during general anesthesia have mimicked malignant hyperthermia.⁵⁻⁹ Because of the normal preoperative thyroid function test, the incidence of perioperative thyroid storm was low.

Our patient was a healthy young woman with no family history of anesthesia related complication. Trismus developed suddenly after the induction of anesthesia was the first sign of malignant hyperthermia seen in this patient. An immediate and continuous increase of end-tidal CO₂ concentration was occurred as well as an immediate rise in body temperature (1.0°C within 30 minutes after the induction of general anesthesia). A differential diagnosis in this patient could have been a thyroid storm but nearly normal preoperative thyroid function test made this less possible. There are many other disorders that mimic malignant hyperthermia, such as neuroleptic malignant syndrome, sepsis and pheochromocytoma but none of these is explained why hypercapnia and hyperthermia are correlated with the use of succinylcholine and sevoflurane.

After malignant hyperthermia developed, we decided to discontinue using sevoflurane and used oxygen from the cylinder instead of anesthetic

machine. A dramatic reduction in end-tidal CO₂ concentration and no further increase in body temperature were noted. From this correlation, succinylcholine was the most likely trigger agent for malignant hyperthermia in this patient and sevoflurane enhanced severity. The major factors related to the good outcome of malignant hyperthermia are early diagnosis and rapid treatment.³ Dantrolene was not available in our hospital. In that manner she received only supportive and symptomatic treatment. Her condition steadily improved, therefore dantrolene was not given.

Caffeine-halothane contracture test (CHCT) is the gold standard to diagnose malignant hyperthermia susceptibility. This test was not widely available in our country, especially in our rural hospital, therefore we discussed with the patient and her mother about the complication but they refused to go for further testing in the university hospital. The diagnosis was made only by clinical presentation and ruled out less possible differential diagnosis. We informed the patient's family and took note on the patient's hospital card to prevent future catastrophe.

Conclusions

Even without a positive history of malignant hyperthermia, anyone using potent inhalation anesthetics or succinylcholine must prepare for treatment of malignant hyperthermia. We have presented a case with immediate muscle rigidity, hypercapnia and tachycardia after exposure to succinylcholine and sevoflurane. The good outcome of our patient is attributed to our early detection and

management. Close perioperative monitoring is the key to early diagnosis and better outcome.

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รายงานผู้ป่วย : การดูแลผู้ป่วย Malignant Hyperthermia แบบประคับประคอง

บทคัดย่อ

บทนำ: Malignant hyperthermia เป็นภาวะแทรกซ้อนทางวิสัญญีอย่างหนึ่งซึ่งโอกาสเกิดน้อย แต่หากเกิดขึ้นแล้วอาจมีผลกระทบที่รุนแรงถึงเสียชีวิตได้ กรณีตัวอย่างของเราสามารถรักษาได้โดยการรักษาตามอาการ เนื่องจากไม่มียา dantrolene ผู้ป่วยเป็นผู้หญิงอายุ 35 ปีมาโรงพยาบาลด้วยอาการมีก้อนที่ต่อมไทรอยด์และมาผ่าตัดก้อนออกตามนัด ผู้ป่วยได้รับการใส่ท่อช่วยหายใจด้วยยา morphinethiopental ตามด้วย succinylcholine หลังฉีดยา succinylcholine ผู้ป่วยมีอาการเกร็งบริเวณขากรรไกร ทำให้อ้าปากใส่ท่อช่วยหายใจลำบาก แต่ยังสามารถใส่ท่อช่วยหายใจได้สำเร็จ หลังจากนั้นดมยาสลบต่อด้วย sevoflurane ได้ยาหย่อนกล้ามเนื้อเป็น cis-atracurium หลังจากนั้นไม่นานผู้ป่วยมีอาการหัวใจเต้นเร็ว คาร์บอนไดออกไซด์ในลมหายใจออกสูงขึ้น ตามด้วยอุณหภูมิกายสูงขึ้น ทำให้นึกถึงภาวะ malignant hyperthermia ผู้ป่วยรับการรักษาอย่างทันที่และสังเกตพบเพิ่มเติมเพื่อวินิจฉัยแยกโรคอื่น

คำสำคัญ: Malignant hyperthermia, ประคับประคอง
