

Case Report

Malignant Hyperthermia : A Case Report in Thai Anesthesia Incidents Study (THAI Study)

Aksorn Pulnitiporn MD, FRCAT*, Somrat Charuluxananan MD, MSc, FRCAT**,
Panjai Inphum MD, FRCAT*, Wassana Kitsampanwong MD, FRCAT*

*Department of Anesthesiology, Khon Kaen Regional Hospital, Khon Kaen , Thailand

**Department of Anesthesiology, Faculty of Medicine, Chulalongkorn University,
Rama IV Rd., Pathumwan, Bangkok 10330, Thailand

A 3-year-old Thai boy underwent open reduction and internal fixation with K-wire of condylar fracture of humerus under general anesthesia. The patients developed generalized muscle rigidity, masseter muscle spasm, elevated creatinine kinase, high temperature (39.3 C), inappropriate tachycardia, and arterial base excess was more than-8 mEq/L. The clinical grading scale of diagnosis of malignant hyperthermia was 58 (grade D6; almost certain malignant hyperthermia). Succinylcholine has been identified as the trigger agent, as other possible trigger agents were not involved. The treatment included hyperventilation, external cooling and cold IV fluids without administration of dantrolene. The patient fully recovered and discharged on day 12. This case report showed an incidence of malignant hyperthermia of approximated 1:150,000 in Thai Anesthesia Incidents Study (THAI Study).

Keywords : Malignant hyperthermia, Complication, Adverse event, Succinylcholine

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Malignant hyperthermia (MH) is a life-threatening pharmacogenetic disorder of calcium regulation within skeletal muscle associated predominantly with anesthesia. It is triggered by an administration of commonly used volatile anesthetics (halothane, enflurane, isoflurane, desflurane and sevoflurane) and depolarizing muscle relaxant succinylcholine^(1,2). It is characterized by hyperthermia, tachycardia, acidosis and muscle rigidity^(1,2).

In Thailand there were few cases reported of suspected MH but we still do not know the approximated incidence of MH. The Thai Anesthesia Incidents Study (THAI Study) of adverse outcomes, hosted by the Royal College of Anesthesiologists of Thailand, studied the incidence of 20 adverse outcomes^(3,4) including suspected MH since February 2003, has extended to phase 2 of the study for surveillance of rare events. We report a case of suspected MH after receiving succinylcholine.

Correspondence to : Aksorn Pulnitiporn, Department of Anesthesiology, Khon Kaen Regional Hospital, Khon Kaen 40000, Thailand. E-mail : aksornp@hotmail.com

Case Report

A 3-year-old Thai boy, diagnosed as condylar fracture of the left humerus, was scheduled for an open reduction and internal fixation with K-wire as an emergency case at Khon Kaen Regional Hospital. This hospital is a 900-bed tertiary hospital with sixteen operating theaters and approximated 50 cases of anesthesia per day. Anesthesia personnel comprised of 6 MD anesthesiologists and 24 nurse anesthetists.

Medical and surgical histories were unremarkable. There was no history of MH, neuromuscular disorders or other anesthesia related complications in the patients and other family members. He had no known allergies and was taking no medications.

Physical examination revealed an awake and alert Thai boy who was well nourished and well developed (15 kg body weight). Preoperative vital signs included the following : noninvasive blood pressure 115/60 mm Hg; heart rate 124 bpm. Initial examination revealed edema and tenderness around the lateral condyle of the left humerus. Radiography confirmed a fracture of the condyle of the left humerus; however

his chest x-ray was not performed. His hematocrit was 36.4%. Results of leukocyte count and differential, serum electrolytes, glucose were all within normal limits.

The initial monitoring were noninvasive blood pressure and pulse oximetry. Preoperative intravenous fluid was 5% dextrose in one-third strength of normal saline. Rapid sequence induction and intubation technique with cricoid pressure was performed by intravenous injection of thiopentone 70 mg and succinylcholine 15 mg. After preoxygenation, his heart rate increased to 160 bpm and massive fasciculation occurred with difficulty to open his mouth. The attending MD anesthesiologist could intubate the patient uneventfully. The heart rate was reduced to 128 bpm. Anesthesia was maintained, without using volatile anesthetic agent, with N₂O/O₂ (50/50) plus morphine 1 mg and intravenous infusion of propofol throughout the operation. The surgical operation was performed successfully within 55 min; then nitrous oxide and propofol drip were turned off. The patient had spontaneous ventilation with very warm face, head and neck area. Capnometry and thermister were installed and revealed end-tidal carbon dioxide of 50 mm Hg and temperature of 39.3 degree Celcius. Heart rate increased to 170 bpm.

Therapy for MH was initiated, which included hyperventilation with 100% oxygen, external cooling, cold IV fluids. The heart rate decreased to 157-160 bpm, oxygen saturation was 100%, end-tidal carbon dioxide decreased to 40 mm Hg, temperature decreased to 37.8 degree Celcius and the patients became conscious. An arterial blood gas analysis showed pH 7.25, PaCO₂ 41 mmHg, PaO₂ 375 mmHg, bicarbonate 17 mmol/L and base excess -9. Other laboratory investigation revealed Cr 0.7 mEq/L, Na 139 mEq/L and K 5.5 mEq/L. After 40 minutes of treatment subsequent arterial blood gas revealed pH 7.3, PaO₂ 511 mm Hg, PaCO₂ 41 mmHg, bicarbonate 18 mmol/L, base excess -7; potassium decreased to 4.3 mEq/L, creatinine phosphokinase (CPK) 3446 unit/L. After treatment of MH for 1.30 hr, the respiration rate decreased to 20 per min, heart rate 125 bpm, body temperature 37.2 degree Celcius, the patient was fully conscious, extubated and transferred to the intensive care unit.

At the intensive care unit, the patients received further monitoring which included body temperature. A pediatrician was consulted. The patient developed rhabdomyolysis (phosphokinase increased to maximum of 51460 U/L) and myoglobinuria (BUN and creatinine still within normal limit) and was treated with alkalization of urine and adequate hydration.

There was no evidence of myocardial ischemia. Within three days in the intensive care unit, phosphokinase decreased to 6233 U/L, creatinine 0.4 mEq/L, the patient was moved to surgical ward. Blood phosphokinase decreased to 3446 U/L on day 10. He fully recovered from the operation and the MH incident. There was no family history of muscle disease and MH. The patient was discharged on day 12 and the 3-month follow up phosphokinase decreased to normal limit.

Discussion

The first description of the syndrome that would eventually be named malignant hyperthermia (MH) was made in the early 1960s.⁽⁵⁾ By 1970, it became clear that alterations in skeletal muscle constituted the primary defect in MH. It was the defect in the ability of muscle to adequately regulate its myoplasmic Ca²⁺ concentration⁽⁶⁾.

We present a case of suspected MH triggered by succinylcholine. Tachycardia is a common response to increased metabolism in MH. In our patient, his heart rate increased from 124 bpm to 170 bpm. We found this alarming together with generalized muscle rigidity and masseter spasm shortly following succinylcholine administration. However, we could ultimately intubate the patient, changed to a new anesthesia circuit and maintained anesthesia using nitrous oxide, oxygen and propofol avoiding triggering agents. The patient had a clinical grading scale of 58 (grade D6). Grades D4 (somewhat greater than likely), D5 (very likely) and D6 (almost certain MH) are defined by scores of 20-34, 35-49 and ≥ 50 , respectively.⁽⁷⁾ The clinical grading scale uses six process indicators and a seventh other category indicator: rigidity, muscle breakdown, respiratory acidosis, temperature increase, cardiac involvement, family history, and indicators not part of a single process with 3-15 points awarded to subcategories of each indicator⁽⁷⁾. Our patients was scored as follows: (a) masseter spasm (15 points); (b) elevated creatinine kinase >20000 unit after succinylcholine (15 points); (c) inappropriately rapid increase in temperature (15 points); (d) inappropriate sinus tachycardia (3 points); (e) arterial base excess more negative than -8 mEq/L (10 points).

Our patient received no other trigger agent but succinylcholine. This patient was not treated with dantrolene because dantrolene was already expired and the patients improved after a supportive treatment with hyperventilation, external cooling and cold IV fluids. During anesthesia end-tidal carbon dioxide increased to 50 mmHg, subsequent arterial blood gas also re-

vealed metabolic acidosis and phosphokinase increased to more than 50000 U/L in the intensive care unit.

Relapsed of the syndrome post-operatively is not uncommon⁽⁸⁾. Thus, patients should be closely monitored. Our patient had been admitted in the hospital for twelve days for monitoring.

In Thailand, cases of suspected MH had been reported since 1979⁽⁹⁻¹³⁾. In phase 2 of the THAI Study of anesthetic outcomes, this is the first case occurred with approximated incidence of 1 : 150,000. The Royal College of Anesthesiologists of Thailand supports stocking of dantrolene at regional medical schools in all parts of country. There is a limitation about the supply of dantrolene in Thailand because of short expiration time which is about two years. Other limitation is there is no diagnostic laboratory test in Thailand. Genetic study of MH susceptible is possible but required high budget.

In summary, this is a case report of suspected MH triggered by succinylcholine with approximated incidence of 1 : 150,000 in the THAI Study.

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ภาวะ Malignant Hyperthermia : รายงานผู้ป่วยจากการศึกษาอุบัติการณ์ภาวะแทรกซ้อนทางวิสัญญีในประเทศไทย

อักษร พูลนิติพร, สมรัตน์ จารุลักษณะนันท์, ปานใจ อินพุ่ม, วาสนา กิจสัมพันธ์วงศ์

เด็กชายไทยอายุ 3 ปี ได้รับการให้ยาระงับความรู้สึกแบบทั้งตัว เพื่อผ่าตัดยึดกระดูกโดยใช้ลวดบริเวณ condyle ของกระดูก humerus ยาหย่อนกล้ามเนื้อ succinylcholine เป็นสารกระตุ้นให้เกิดภาวะ Malignant hyperthermia โดยไม่มีการใช้สารกระตุ้นชนิดอื่น ผู้ป่วยเกิดอาการเกร็งของกล้ามเนื้อโดยทั่วไป, กล้ามเนื้อแข็งกระด้างปากยาก, ระดับ creatinine kinase สูงขึ้นมาก, อุณหภูมิสูงถึง 39.3 องศาเซลเซียส หัวใจเต้นเร็วมาก ความเป็นด่างมากกว่า -8 mEq/L การวินิจฉัยโดยใช้ clinical grading scale ได้ 58 แต้ม (ระดับ D6 almost certain malignant hyperthermia) ได้รับการรักษาโดยการช่วยหายใจเพิ่มขึ้น, เซ็ดตัวด้วยน้ำเย็น, ให้น้ำเกลืออุณหภูมิต่ำ โดยมีได้รับยา dantrolene ผลการรักษาผู้ป่วยอาการกลับเป็นปกติ และเฝ้าดูในโรงพยาบาล 12 วัน รายงานผู้ป่วยนี้แสดงอุบัติการณ์ของการเกิดภาวะ Malignant hyperthermia ประมาณ 1 : 150,000 จากโครงการศึกษาภาวะแทรกซ้อนทางวิสัญญีในประเทศไทย
