Suspected malignant hyperthermia in a 13-month-old: Today's "typical" episode—A case report

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A case of suspected malignant hyperthermia in a 13-month-old female, to whom succinylcholine was not administered, is presented. The patient presented for a repair of the right radial nerve under general anesthesia. Induction was accomplished with halothane, nitrous oxide, and oxygen. Tracheal intubation was facilitated with intravenous vecuronium. Controlled ventilation was initiated, and anesthesia was maintained with isoflurane, nitrous oxide, oxygen, morphine sulfate, and vecuronium.

At the conclusion of the surgical procedure, an abrupt increase in ETCO₂, an elevation in body temperature, and a mixed acidosis was observed. Resolution of symptoms followed the administration of dantrolene sodium. The patient underwent an uneventful postoperative recovery and was discharged home. It was felt that the patient was too young to undergo a muscle biopsy for a caffeine-halothane stimulation test.

Key words: Malignant hyperpyrexia, malignant hyperthermia.

Introduction
Malignant hyperthermia (MH) has been widely reported in the anesthesiology literature as a potentially fatal complication of anesthesia. In 1970, Barlow and Isaacs presented three fatal cases of malignant hyperpyrexia where they described symptoms of tachycardia, tachypnea, hypervolemia, acidosis, mottling of the skin, and cyanosis with a reported mortality rate of approximately 70%.¹ The trait is thought to be a genetically linked defect in the sarcoplasmic reticulum's calcium pump, most commonly triggered by the administration of halothane and succinylcholine, resulting in increased muscular metabolism. The increased metabolism is manifested by increased CO₂ and lactate production yielding a mixed acidosis. Myoglobin levels increase with the breakdown of muscle cells and may lead to rhabdomyolysis.²

In 1960, Denborough and Lovell first described malignant hyperthermia in a family with a history of anesthetic deaths.³ The incidence of MH, reported by Britt in 1972, was approximately one in 50,000 anesthetics in adults and one in 15,000 anesthetics in children.⁴ Ording, in 1985, reported the incidence of fulminant malignant hyperthermia as one in 250,000 anesthetics.⁵ Other data note suspicion of MH occurred in one in 16,000 anesthetics and one in 4,200 anesthetics when succinylcholine was administered with a potent inhaled anesthetic. A current review of published cases of malignant hyperthermia was presented by Strazis and Fox.⁶ They reported the mean age of all reviewed cases to be 18.3 years and that patients under the age of 15 years comprised 52.1% of all MH cases.

Malignant hyperthermia occurs most frequently in patients 3 to 30 years of age.⁷ It is rare to encounter MH in patients under 2 years of age. Withoît presented a case report of possible MH in a 7-week-old infant, the youngest reported case to date.⁸ We report the developments of a case that may represent today's "typical" suspected malignant hyperthermic episode.

Case summary
The patient was a 13-month-old, 10-kg, cauca-
sian female scheduled for a right radial nerve exploration and repair subsequent to an accidental knife injury, which resulted in wrist drop. The patient was a full-term infant from an uncomplicated birth and offered no history of systemic disease and no known neuromuscular defect. She had a negative family history for malignant hyperthermia. Preoperative laboratory results and vital signs were within normal limits.

The patient was premedicated in the holding area with 5 mg midazolam and 200 μg atropine. Thirty minutes later, the patient was carried to the operating room where standard monitors were placed (noninvasive blood pressure, electrocardiograph, pulse oximeter, precordial stethoscope, capnometer, and rectal temperature probe) and an inhalation induction with nitrous oxide, halothane, and oxygen was performed. Intravenous (IV) access was established in the right foot with a #22 gauge catheter and a dextrose in 5% lactated Ringer’s solution infusion was started. Endotracheal intubation was facilitated by the intravenous administration of 1 mg vecuronium and a #3.5 unuffed endotracheal tube was placed under direct laryngoscopy. Breath sounds were auscultated over all lung fields, and an ETCO2 waveform was noted with values in the 40s. A leak around the endotracheal tube was auscultated at 15 cm of H2O pressure.

The carina was identified at 14 cm and the endotracheal tube was secured at the teeth (12 cm) with benzoin and tape. The patient was placed in a left lateral decubitus position, and the endotracheal tube position and breath sounds verified.

Anesthesia was maintained with isoflurane, nitrous oxide, and oxygen. The perioperative course was unremarkable. The surgical procedure was completed 2 hours after induction. The anesthetic was discontinued as the surgical team applied an upper thorax cast to splint the right arm.

At induction plus 2 hours and 10 minutes, an increase in the patient’s heart rate was noted from a rate in the 120s to 160 beats per minute (bpm), oxygen saturation of 100%, and the patient began spontaneously ventilating and demonstrated purposeful movement with an ETCO2 in the 30s. Within 3 minutes (induction plus 2 hours and 13 minutes), the ETCO2 was noted to increase to greater than 80. Vigorous ventilation with 100% oxygen was instituted immediately. The ETCO2 filter was changed and recalibrated (rezeroed). The ETCO2 was then noted at 78 with a heart rate of 189 bpm (induction plus 2 hours and 15 minutes).

The patient’s rectal temperature slowly increased during the second hour of the case, approximately 0.1°C every 10 to 15 minutes. Active and passive temperature conservation techniques were utilized to include increasing the ambient room temperature, wrapping exposed skin in plastic, placing a thermal blanket under the patient and placing a heat and moisture exchanger in the anesthesia circle system. When the rectal temperature reached 37.5°C (induction plus 1 hour and 35 minutes) all of the above-listed temperature conservation techniques were discontinued. At induction plus 2 hours and 10 minutes, the patient’s rectal temperature was 37.3°C and achieved a maximum temperature of 38.9°C at induction plus 2 hours and 35 minutes. The patient’s color was flush and in a cape distribution. An arterial blood gas was obtained at induction plus 2 hours and 17 minutes (pH 7.08, PacO2 65, PacO2 469, base excess (BE) -6.8) and additional anesthesia support was summoned. The malignant hyperthermia cart was obtained and dantrolene sodium, 2 mg/kg (20 mg) and 14 mEq of sodium bicarbonate was administered (induction plus 2 hours and 25 minutes).

A second IV line, an arterial line, and an indwelling catheter were placed. The operating room was cooled and the thermal blanket turned to the cooling mode. The patient was mottled. A second arterial blood gas obtained at induction plus 2 hours and 30 minutes (pH 7.20, PacO2 54, PaO2 438, BE -2.1). A full oxygen tank and Jackson-Reese circuit were obtained and the anesthesia circle system was abandoned, although ETCO2 monitoring was maintained. A third arterial blood gas was obtained at induction plus 2 hours and 35 minutes (pH 7.23, PacO2 58, PaO2 556, BE -2.6). A second dose of dantrolene sodium 2 mg/kg (20 mg) was administered approximately 10 minutes after the initial dose. A fourth arterial blood gas was obtained at induction plus 2 hours and 45 minutes (pH 7.48, PacO2 93, PaO2 524, BE 2.2).

At induction plus 2 hours and 50 minutes, breath sounds decreased and peek inspiratory pressures increased to ≥ 46 cmH2O. The upper torso cast was removed. A suction catheter was passed down the endotracheal tube where it met resistance. Another #3.5 uncuffed endotracheal tube was placed under direct visualization, and a large mucous plug was noted at the end of the old endotracheal tube. A portable chest x-ray was obtained, which showed normal structures and was negative for pneumothorax and for consolidation. A fifth arterial blood gas was obtained at induction plus 2 hours and 55 minutes (pH 7.45, PacO2 33, PaO2 373, BE 1.0).

The patient received 1 mg vecuronium and 0.5 mg morphine and was transported to the pediatric intensive care unit (PICU), manually ventilated, Sao2 98-100%. Upon arrival in the PICU, the
A patient was placed on mechanical ventilation. Vital signs were: temperature 96.1, heart rate 113, respiratory rate 20 (V), blood pressure 100/49.

The patient was extubated within 6 hours of admission to the PICU and experienced an uneventful overnight stay. All urine myoglobin tests were negative. Serum creatine phosphokinase (CPK) results at 6, 12, and 18 hours postevent were as follows: 149 U/L, 180 U/L, and 156 U/L.

The patient was transferred to the pediatric ward and was discharged to home after counselling with a staff anesthesiologist, who provided counselling to the family regarding malignant hyperthermia, the malignant hyperthermia hotline (1-800-MH HYPER or 1-800-644-9737), and medical alert bracelets.

**Discussion**

This case may very well demonstrate a "typical" presentation of a possible malignant hyperthermic episode in an atypical patient. The only triggering agents used in this case were potent inhaled anesthetics. When succinylcholine is administered during induction of anesthesia, the manifestations of MH are accelerated and a fulminating course can present over 5 to 10 minutes. The routine use of succinylcholine in children has diminished over the past few years subsequent to U.S. Food and Drug Administration (FDA) changes which stated that except for emergent securing of the airway, succinylcholine is contraindicated in children and adolescents. (The Anesthetic and Life Support Drugs Advisory Committee of the FDA has recommended that this contraindication be downgraded to a warning.)

Therefore, when succinylcholine is not administered, the presentation of MH will most often follow a dose-dependent response. The concentration of potent inhaled anesthetic must rise within the skeletal muscle to the individual patient's triggering threshold before manifestations of MH are observed. These manifestations may not be observed until late in the case, as seen in this case, until the patient arrives in the postanesthesia care unit or on the nursing unit. With the trend toward same-day surgery, practitioners may see an increased mortality associated with MH as a consequence of earlier discharges.

Since 1985, reported fatality rates have decreased to 10%. This decline is due to several factors: greater awareness and education of anesthesia providers, the advent of \( R_{\text{CO}_2} \) monitoring and its judicial use; however, the most substantial contribution to the decrease in mortality rates must be attributed to the advent and early administration of dantrolene sodium. Malignant hyperthermia is an anesthetic complication that occurs infrequently. We encourage others to actively evaluate trends and not dismiss the possibility that a patient in your care may be experiencing an onset of MH. There need not be a family history of anesthesia complications, because MH can occur at any time and with any patient. Having a well-established plan of action when encountering suspicious signs and symptoms will enable the anesthesia practitioner to act quickly, confidently, and successfully.

**REFERENCES**


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