Case Report ISSN: 3065-5641

Global Journal of Critical Care and Emergency Medicine

Suspected Malignant Hyperthermia in Pediatric Male During an Elective Procedure

Shanta Ramsaran, DO[1]*, Austin Han, MD[2], William Nguyen[3], Casey Chai Gyi, MD[4] and Marguerite Canter, MD[5]

[1]*Department of Pediatrics, NYU Langone Hospital – Long Island, Mineola, USA.*

[2]*Department of Pediatrics, Division of Neonatal-Perinatal Medicine, Cohen Children's Medical Center, Northwell Health, New Hyde Park, NY, USA.*

[3]*NYU Grossman Long Island School of Medicine, Mineola, USA.*

[4]*Department of Anesthesiology, NYU Langone Hospital – Long Island, Mineola, USA.*

[5]*Division of Pediatric Critical Care, Department of Pediatrics, NYU Langone Hospital – Long Island, Mineola, USA.*

***Correspondence:**
Shanta Ramsaran, Department of Pediatrics, NYU Langone Hospital Long Island, 259 First Street, Mineola, New York, USA, Phone: 631-873-6057.

Received: 03 Feb 2026; **Accepted:** 09 Mar 2026; **Published:** 20 Mar 2026

Citation: Shanta Ramsaran, Austin Han, William Nguyen, et al. Suspected Malignant Hyperthermia in Pediatric Male During an Elective Procedure. Glob J Emerg Crit Care Med. 2026; 3(1); 1-3.

ABSTRACT

Malignant hyperthermia is a rare pharmacogenetic disorder that can be life threatening. It is triggered by all inhaled anesthetics and succinylcholine, with the pediatric population being the most common age group. Presentations vary, but most enter a hypermetabolic state with tachycardia, elevated end-expired carbon dioxide levels, and muscle rigidity. An 18-month-old male undergoing elective penoplasty and chordee repair with circumcision with no prior surgery developed malignant hyperthermia after exposure to sevoflurane. The patient was treated with Ryanodex (Dantrolene Sodium) and continued to be monitored. This case highlights a rare and potentially life-threatening disorder that should be considered prior to all surgical operations.

Keywords

Pediatric, Malignant Hyperthermia, Case Report.

Introduction

Malignant hyperthermia (MH) is a rare, but potentially life-threatening pharmacogenetic disorder. Triggered by medication exposure, patients enter a hypermetabolic state, characterized by muscle rigidity, hyperthermia, and organ dysfunction. The reported incidence of malignant hyperthermia ranges from 1 in 10,000 to 1 in 220,000, reflecting the variability and limitations of available clinical data [1]. This condition has been associated with a higher incidence in pediatric patients; the mean age of affected individuals is 18.3 years, with those under 15 accounting for 52.1% of all reported reactions [2].

Patients who develop MH usually have a genetic mutation which predisposes them to the condition. The most commonly identified mutation involves the ryanodine receptor (RYR1) gene, and it is inherited in an autosomal dominant pattern with incomplete penetrance [3]. The ryanodine receptor is an integral component of the muscle contraction cascade, activated by dihydropyridine-sensitive L-type voltage dependent calcium channels (DHPR) during muscle depolarization via excitation-contraction coupling. Subsequently, calcium is released from the sarcoplasmic reticulum (SR) and binds to troponin C, inducing a conformational change that shifts tropomyosin away from the myosin-binding sites, thereby initiating muscle contraction. Afterwards, calcium is reabsorbed into the SR. An additional mutation implicated in MH is the CACNA1S gene, which accounts for 14–21% of affected families and encodes the primary subunit of the voltage-sensitive calcium channel in T-tubules [4].

MH is triggered by all inhaled anesthetics and succinylcholine, with pediatric patients being most commonly affected [2]. Although the exact mechanism remains unknown, the leading hypothesis involves an excessive amount of calcium release from the SR, prolonging muscle contraction, and creating a hypermetabolic state [5]. Consequently, presenting symptoms include tachycardia, elevated end-expired carbon dioxide levels despite increased minute ventilation, muscle rigidity, often involving the jaw and limbs, and a later onset of hyperthermia [2]. Pediatric patients may have varied presentations: younger children typically present with elevated temperature and generalized muscle rigidity, whereas older pediatric patients more commonly exhibit tachycardia and hypercarbia [6]. Although the presentations vary, the condition progresses rapidly and can be fatal if untreated, necessitating prompt recognition and intervention.

MH is clinically diagnosed upon trigger exposure and recognition of the hypermetabolic state. Sustained muscle contraction and subsequent adenosine triphosphate depletion results in cell membrane disruption and release of muscle cell contents, including electrolytes, myoglobin, and creatinine kinase [3]. Therefore, frequent electrolyte monitoring is critical. Definitive diagnostic testing requires muscle biopsy and conducting in vitro contracture response testing with exposure to substances including halothane, caffeine, and other agents [7]. Ultimately, confirmatory diagnosis involves RYR1 gene mutation testing. However, multiple mutation variants in the RYR1 gene have been identified in MH.

The first-line treatment for MH is intravenous dantrolene, which inhibits calcium release through the ryanodine receptor. Ryanodex, a concentrated formulation of dantrolene, is available. When MH was first described, the mortality rate was 70–80%. However, with earlier detection and rapid administration of dantrolene, mortality has decreased to 2.9% [8]. However, these MH treatments are associated with muscle relaxation, potentially compromising patients' airways. As such, patients may be intubated for airway protection for the duration of treatment. The half-life of dantrolene is 4-8 hours, while Ryanodex is 8.5-11.4 hours, which affects the length of post treatment monitoring prior to extubation [9,10]. Twenty percent of patients with MH experience recrudescence with symptoms returning after an average of 13 hours post treatment [11]. Individuals with greater muscle mass or who are exposed to triggering anesthetic agents for longer than 150 minutes have an increased risk of MH recurrence [12]. Fluid and electrolyte resuscitation measures are often necessary to mitigate the consequences of muscle cell breakdown. As such, patients are monitored in an intensive care unit (ICU) setting with frequent vital sign checks, end-tidal capnography, and electrolyte monitoring.

Long-term prevention for future MH cases involves avoiding anesthetic triggers in future operations. For high-risk surgeries, anesthesiologists may consider dantrolene prophylaxis. Genetic counseling and MH screenings are recommended for family members.

Case Presentation

An 18-month-old full-term male, small for his age (<3rd percentile for height and weight), with a history of congenital chordee and hypospadias, presents for an elective penoplasty and chordee repair with circumcision. A laryngeal mask airway was placed, and general anesthesia was induced with sevoflurane. Afterwards, a caudal block was administered. He remained stable throughout the procedure. Preoperative and intraoperative medications administered include dexamethasone, ondansetron, cefazolin, glycopyrrolate, fentanyl, and propofol. Upon procedural completion, the patient was noted to be tachycardiac to 190 beats per minute, hyperthermic with an elevated axillary temperature of 38.7°C (101.6°F), and hypercarbic, evidenced by end-tidal carbon dioxide monitor of 57 mmHg. Sevoflurane was discontinued and charcoal filters were placed. Arterial blood gas (ABG) was obtained, ice packs were applied, and the MH hotline was contacted. ABG demonstrated combined respiratory and metabolic acidosis (pH 7.17, pCO_2 52 mmHg, HCO_3 19 mmol/L). Upon discussion with MH hotline consultations, the team proceeded to treat the patient as an MH case. Intubation, arterial line placement, and administration of Ryanodex 2.5mg/kg were performed. Urine myoglobin level was obtained. The patient was transferred to the pediatric ICU for further management.

Upon PICU admission, the patient had a normal heart rate but was hypothermic with a rectal temperature of 89.6°F (32.0°C). The medical team contacted the MH hotline, and based on recommendations, continued to administer Ryanodex every 4 hours for a total of 24 hours. During this period, the patients returned to normothermia with stable vital signs and end-tidal capnography. Serial basic metabolic panels, creatine phosphokinase (CPK) levels, and ABGs were obtained to monitor for recurrence of acidosis, electrolyte derangement, and muscle breakdown. These values remained stable during this 24-hour period. The patient remained intubated and sedated with dexmedetomidine and fentanyl for airway protection during this timeframe.

The following morning, the patient was evaluated for extubation. A good air leak was noted audibly on both physical examination and ventilator assessment. Gradual dose reduction of sedative medications was performed. The patient tolerated continuous positive airway pressure support trial with stable oxygenation and end-tidal CO_2 with adequate respiratory effort and drive. The patient was extubated 3 hours after the last Ryanodex dose, however experienced significant stridor and hypoxemia with oxygen saturation dropping to the low 70s. The patient immediately received racemic epinephrine and required supplemental oxygen support and escalated from nasal cannula to Bilevel positive airway pressure (BiPAP). On examination, the patients appeared to have truncal weakness and difficulty tolerating secretions.

Following dexamethasone and 5 racemic epinephrine treatments over 24 hours, resolution of stridor was achieved with improved muscle tone. Respiratory support was reduced from BiPAP to room air by hospital day 3. The team obtained post-extubation venous

blood gas, electrolyte panel, and CPK levels. While most values remained stable, a notable increase in CPK was observed. The patient was discharged with follow-up appointments for genetic evaluation and urologic post-procedural visits.

Conclusion

A unique aspect of this case was the challenging extubation post-Ryanodex. Although the patient demonstrated adequate respiratory effort and gas exchange, he developed post-extubation stridor and hypoxemia requiring escalated respiratory support, racemic epinephrine, and corticosteroids. The transient truncal weakness and difficulty managing secretions may reflect rare but known side effects of dantrolene. Generalized muscle weakness and respiratory failure have been reported in 21.7% and 3.8% of patients, respectively [11,13].

This case report highlights several key clinical considerations. First, early diagnosis and aggressive MH treatment are critical and were effectively implemented in this case. Second, while dantrolene and Ryanodex are lifesaving, the muscle-relaxant properties may complicate post-extubation recovery. Lastly, a multidisciplinary approach involving anesthesiology, critical care, and respiratory therapy is essential in successful patient management and improved outcomes.

Although difficult extubation following dantrolene administration is uncommonly reported, this case adds to the current evidence suggesting the need for heightened caution during extubation and an observation period post-dantrolene administration, particularly in younger and smaller patients. Airway complications related to Ryanodex and dantrolene underscore the complex clinical considerations in managing MH beyond the initial critical phase. Further research into predictors of post-treatment respiratory compromise in MH is warranted.

References

1. In J, Ahn EJ, Lee DK, et al. Incidence of malignant hyperthermia in patients undergoing general anesthesia protocol for a systematic review and meta-analysis. Medicine (Baltimore). 2017; 96(49): e9115. http://ovidsp.ovid.com/ovidweb.cgi?T=JS&NEWS=n&CSC=Y&PAGE=fulltext&D=ovft&AN=00005792-201712080-00134.
2. Rosenberg H, Davis M, James D, et al. Malignant hyperthermia. Orphanet J Rare Dis. 2007; 2: 21.
3. Kim D. Malignant hyperthermia. Korean journal of anesthesiology. 2012; 63: 391-401. https://www.ncbi.nlm.nih.gov/pubmed/23198031.
4. Miller DM, Daly C, Aboelsaod EM, et al. Genetic epidemiology of malignant hyperthermia in the UK. Br J Anaesth. 2018; 121: 944-952.
5. Yang T, Allen PD, Pessah IN, et al. Enhanced excitation-coupled calcium entry in myotubes is associated with expression of RyR1 malignant hyperthermia mutations. The Journal of biological chemistry. 2007; 282(52): 37471-37478.
6. Otsuki S, Miyoshi H, Mukaida K, et al. Age-specific clinical features of pediatric malignant hyperthermia: a review of 187 cases over 60 years in Japan. Anesth Analg. 2022; 135: 128-135.
7. Larach MG. Standardization of the caffeine halothane muscle contracture test. Anesth Analg. 1989; 69: 511-515.
8. Salazar JH, Yang J, Shen L, et al. Pediatric malignant hyperthermia: risk factors, morbidity, and mortality identified from the Nationwide Inpatient Sample and Kids' Inpatient Database. Paediatr Anaesth. 2014; 24: 1212-1216.
9. Dantrolene sodium (Ryanodex) [package insert]. Eagle Pharmaceuticals, Inc; 2020.
10. Ratto D, Joyner RW. Dantrolene. StatPearls. 2023. https://www.ncbi.nlm.nih.gov/books/NBK535398/.
11. Burkman JM, Posner KL, Domino KB. Analysis of the clinical variables associated with recrudescence after malignant hyperthermia reactions. Anesthesiology. 2007; 106: 901-1078.
12. Watt S, McAllister RK. Malignant hyperthermia. StatPearls. 2023. https://www.ncbi.nlm.nih.gov/books/NBK430828/.
13. Brandom BW, Larach MG, Chen MSA, et al. Complications associated with the administration of dantrolene 1987 to 2006: a report from the North American Malignant Hyperthermia Registry of the Malignant Hyperthermia Association of the United States. Anesth Analg. 2011; 112: 1115-1123.