

Early Recognition and Management of Suspected Malignant Hyperthermia During General Anesthesia: A Case Report

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ABSTRACT

Malignant hyperthermia is a rare but life threatening hypermetabolic crisis triggered by volatile anesthetic agents and succinylcholine in genetically susceptible patients. Early recognition and prompt treatment with dantrolene are critical to reducing morbidity and mortality. We report the case of a 28 years old male with no known personal or family history of anesthetic complications who developed suspected malignant hyperthermia during an emergency laparoscopic appendectomy under general anesthesia. After induction with propofol, fentanyl, rocuronium, and maintenance with sevoflurane, the patient developed rapidly rising end tidal carbon dioxide, tachycardia, generalized muscle rigidity, metabolic acidosis, and hyperthermia. Sevoflurane was discontinued, the anesthesia machine was flushed, dantrolene was administered, and active cooling measures were initiated. The patient stabilized intraoperatively and was transferred to the intensive care unit for monitoring. This case highlights the importance of maintaining a high index of suspicion for malignant hyperthermia when unexplained hypercarbia, tachycardia, rigidity, acidosis, and hyperthermia occur during anesthesia. Immediate discontinuation of triggering agents, administration of dantrolene, supportive care, and postoperative monitoring are essential.

INTRODUCTION

Malignant hyperthermia is an inherited pharmacogenetic disorder of skeletal muscle calcium regulation. It is most commonly associated with mutations affecting the ryanodine receptor, leading to uncontrolled calcium release from the sarcoplasmic reticulum after exposure to triggering anesthetic agents.^[1,3] Common triggers include volatile inhalational anesthetics and succinylcholine.^[2]

Although malignant hyperthermia is rare, it can progress rapidly and become fatal if not treated immediately. Early signs may include an unexplained rise in end-tidal carbon dioxide, tachycardia, muscle rigidity, metabolic acidosis, hyperkalemia, and increasing core temperature.^[1,2] Hyperthermia may occur later, so waiting for fever before treating can delay life-saving management.^[2]

This case report describes the intraoperative recognition and successful management of suspected malignant hyperthermia in a young adult undergoing emergency surgery.

CASE REPORT

A 28 years old male with no significant past medical history presented to the emergency department with acute right lower quadrant abdominal pain, nausea, and leukocytosis. Imaging was consistent with acute appendicitis, and the patient was scheduled for emergency laparoscopic appendectomy.

The patient had no known drug allergies. He denied previous anesthesia exposure and had no known family history of anesthetic complications, unexplained perioperative death, or neuromuscular disease. Preoperative vital signs were stable. Airway examination was reassuring, with Mallampati class II, normal mouth opening, and full neck range of motion.

In the operating room, standard American Society of Anesthesiologists monitors were applied. General anesthesia was induced with intravenous propofol, fentanyl, and rocuronium. Endotracheal intubation was successful on the first attempt. Anesthesia was maintained with sevoflurane in oxygen and air.

Approximately 25 minutes after induction, the patient developed a progressive increase in end tidal carbon dioxide from 38 mm Hg to 68 mm Hg despite increased minute ventilation. This was followed by sinus tachycardia, with heart rate rising from 92 beats/min to 145 beats/min. The anesthesia circuit, endotracheal tube, and ventilator settings were checked, and no mechanical obstruction or equipment malfunction was identified.

Over the next several minutes, the patient developed generalized muscle rigidity and a rising core temperature from 37.1°C to 39.2°C. Arterial blood gas analysis demonstrated mixed respiratory and metabolic acidosis with elevated carbon dioxide. Serum potassium was elevated, and creatine kinase later increased significantly. Based on the rapid development of unexplained hypercarbia, tachycardia, muscle rigidity, acidosis, and hyperthermia, malignant hyperthermia was suspected.

Sevoflurane was immediately discontinued. The anesthesia team switched to 100% oxygen at high fresh gas flows. The volatile anesthetic vaporizer was removed, and the breathing circuit and carbon dioxide absorbent were changed. Intravenous dantrolene was administered at an initial dose of 2.5 mg/kg. Active cooling was started using ice packs, cooled intravenous fluids, and surface cooling. The surgical team was notified and expedited completion of the procedure.

After dantrolene administration and supportive treatment, the patient's end-tidal carbon dioxide gradually decreased, heart rate improved, and core temperature began to normalize. Additional dantrolene was available but was not required after clinical stabilization. The patient was transferred intubated to the intensive care unit for close monitoring.

In the intensive care unit, serial laboratory testing was performed, including arterial blood gases, electrolytes, creatine kinase, renal function, and coagulation studies. The patient was monitored for recurrent malignant hyperthermia, rhabdomyolysis, hyperkalemia, and renal injury. He remained stable, was extubated the following day, and was discharged after continued clinical improvement.

The patient and family were counseled regarding suspected malignant hyperthermia susceptibility. He was advised to avoid triggering anesthetic agents in the future and to obtain formal malignant hyperthermia evaluation, including referral for genetic counseling and possible confirmatory testing.

DISCUSSION

This case demonstrates several important principles in the recognition and management of malignant hyperthermia. The earliest and most important sign in this patient was a rapid, unexplained rise in end-tidal carbon dioxide despite increased ventilation. Hypercarbia is often one of the earliest indicators of malignant hyperthermia because skeletal muscle hypermetabolism leads to excessive carbon dioxide production.^[1,2]

The diagnosis of malignant hyperthermia is clinical during an acute crisis. Treatment should not be delayed while waiting for confirmatory laboratory testing. In this case, the combination of hypercarbia, tachycardia, rigidity, acidosis, hyperkalemia, and rising core temperature was highly concerning. Early action was appropriate because malignant hyperthermia can progress quickly to arrhythmias, disseminated intravascular coagulation, rhabdomyolysis, renal failure, and death.^[1,2]

Immediate management requires discontinuation of triggering agents, administration of 100% oxygen, prompt dantrolene therapy, active cooling, correction of acidosis and electrolyte abnormalities, and close postoperative monitoring.^[2-4] Dantrolene is the specific treatment because it reduces abnormal calcium release from skeletal muscle. The recommended initial dose is commonly 2 to 2.5 mg/kg intravenously, with repeat dosing as needed until clinical signs improve.^[2-4]

This case also highlights that malignant hyperthermia may occur even when there is no known family history. A negative history does not exclude susceptibility because patients may never have been exposed to triggering agents before.³ For future anesthetics, the patient should receive a non-triggering anesthetic technique, including avoidance of volatile anesthetics and succinylcholine.^[5] Anesthesia machines should be prepared appropriately, and the care team should clearly document the suspected malignant hyperthermia episode in the medical record.^[5]

Postoperative care is also essential. Patients require monitoring for recurrence, hyperkalemia, acidosis, rhabdomyolysis, renal injury, and compartment-related complications. Serial creatine kinase, electrolytes, renal function, urine output, and acid-base status should be followed until the patient is clinically stable.^[1,2]

CONCLUSION

Malignant hyperthermia is a rare but critical anesthesia emergency. This case emphasizes that unexplained rising end-tidal carbon dioxide during general anesthesia should prompt immediate evaluation for malignant hyperthermia, especially when accompanied by tachycardia, rigidity, acidosis, hyperkalemia, or hyperthermia. Early discontinuation of triggering agents, rapid dantrolene administration, active cooling, supportive management, and intensive postoperative monitoring can lead to favorable outcomes.

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