Malignant Hyperthermia

Devin Poncsak
Otterbein University, devin.poncsak@otterbein.edu

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A crisis of malignant hyperthermia is a medical emergency, and must be treated immediately with a coordinated, multidisciplinary team response in order to give the patient the highest chance for a successful recovery (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). Malignant hyperthermia is potentially lethal, and may be triggered by a vast array of anaesthetics and muscular relaxants used to support surgery during the anesthetic process. (Johns, Stoudt, Scholtis, & Gavel, 2012). More specific clinical signs of malignant hyperthermia include hyperpyrexia, tachycardia, tachypnea, and cardiac arrhythmias/arrests caused by MH, like disseminated intravascular coagulation, which can also cause disseminated intravascular coagulation (Cain, Riess, Gettrust, & Novalija, 2014). The CRNA must be attentive and track the patient’s temperature at least once every hour (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). This condition was termed in 1960, by Denborough and Lovell who described the disorder in a family with this autosomal dominant genetic inheritance (Bandschapp & Girard, 2012). For the rare event of malignant hyperthermia to occur, the patient experiencing these triggering agents, a pathologic stressor, and the patient has a rapid increase in metabolic demand (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). Carbon dioxide, hyperthermia, acidosis, muscle rigidity, and hyperkalemia are very rare, but can easily progress to death (Bandschapp & Girard, 2012). Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt (2013) state that they have a defect in the ryanodine receptor type 1 (RYR1) gene (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt (2013) state that they have a defect in the ryanodine receptor type 1 (RYR1) gene (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). By pairing this abnormal ryanodine receptor gene with the presence of a triggering agent like dantrolene (Bandschapp & Girard, 2012), an uncontrolled calcium release from the sarcoplasmic reticulum will result as an effect of the triggering agent causing the RYR1 to open unchecked (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). This calcium starts the actin-myosin interaction which makes the muscle contract by shortening the muscle fibers (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). Early on the muscle cells attempt to restore homeostasis through aerobic and anaerobic metabolism, however it is overpowered by the excessive amount of calcium in the myoplasm, and reaches the threshold levels for myoglobinuria (Revello, 2012).

Malignant hyperthermia is a genetic disorder caused by MH, like disseminated intravascular coagulation, which can also cause disseminated intravascular coagulation (Cain, Riess, Gettrust, & Novalija, 2014). When the muscle cells attempt to restore homeostasis through aerobic and anaerobic metabolism, however it is overpowered by the excessive amount of calcium in the myoplasm, and reaches the threshold levels for myoglobinuria (Revello, 2012). This process is significant in that it rapidly depletes adenosine triphosphate (ATP) and increases glucose metabolism, oxygen consumption, carbon dioxide production, and heat production (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). These symptoms must be recognized quickly and treated immediately, along with other complications that may develop, including electrolyte and acid-base abnormalities (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). These symptoms must be recognized quickly and treated immediately, along with other complications that may develop, including electrolyte and acid-base abnormalities (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). This can spread awareness about a situation that could allow me to affect my colleagues’ or my own career.

The condition was described in 1960, by Denborough and Lovell who described the disorder in a family with this autosomal dominant genetic inheritance (Bandschapp & Girard, 2012). Yes, it can happen in a normal patient. In fact, the mortality rates for the hyperthermia crisis were still over 80% and after 40 years of research the pathogenesis of the complication was discovered, along with the medical treatment in dantrolene (Bandschapp & Girard, 2012). Current mortality rates have decreased to less than 5%, because of the symptoms being immediately recognized and treated by anesthetists (Bandschapp & Girard, 2012). Malignant hyperthermia is still a very precarious state, yet very treatable under the proper clinician care.

A 34 y.o. man was admitted to the hospital for a routine orthopedic surgery. On preop assessment the CRNA found no relevant history or previous anesthetic history, a family history of anesthetic complications, but the patient could not tell what these were (Dirksen, Van Wicklin, Mashman, Neiderer, & Merritt, 2013). Nurse Anesthesia program, and providing the patient with awareness of how to handle the situation. That is why picking this case is important, both removal of the gallbladder, jaundice, hyperthermia, and muscle rigidity, and no apparent complications. After reviewing the chart, the patient had a temperature of 39.0ºC, HR, 75 bpm, BP, 127/82 mmHg, 1660 mLSat, 100%, temp, 36.9ºC. Upon further questioning the patient had a temperature of 39.5ºC, HR increases to 91 bpm, and RR 21/min, and stable hemodynamics. The patient was taking 4mg of Morphine IVP for pain and increasing to 5mg of Morphine IVP for 4. The nurse then continues to see if the CRNA and surgeon to report changes in the patient’s condition. After this, the patient shows muscle rigidity of the trunk, displays ventricular ectopy, and vital signs are now as follows: HR 127 bpm; BP, 167/101 mmHg; RR, 31/min; O2Sat, 82%; and body temp, 39.5°C. The CRNA, now at bedside, quickly makes the diagnosis of malignant hyperthermia and administers 2mg/kg of dantrolene IVP and 2mg/kg of bicarbonate and respiratory rate is 26. A blanket is then applied to the patient and an ABG and labs are taken that show: pH, 7.21; PO2, 57 mmHg; PCO2, 80 mmHg; base excess, 7.2 mmol/L. The patient is then given 10 units of regular insulin IV, followed by 50 mL of 50% dextrose in water IV, and CRNA is then transferred to the ICU unit (Anderson-Pompa, Foster, Parker, Wilks & Cheek, 2009).

The next few minutes are the critical signs and symptoms of the operative emergency, malignant hyperthermia (MH). This is a condition that develops rapidly and must be treated immediately. This genetic skeletal muscle disorder does not always happen immediately, some cases like this example, the signs and symptoms can occur after surgery during the recovery period (Anderson-Pompa, Foster, Parker, Wilks & Cheek, 2009). The condition results in the body developing into a hypermetabolic state with lactate acidosis, muscle rigidity, arrhythmias, muscle contractions, and changes in electrolyte and acid-base balances. The condition was termed in 1960, by Denborough and Lovell who described the disorder in a family with this autosomal dominant genetic inheritance (Bandschapp & Girard, 2012). Yes, it can happen in a normal patient. In fact, the mortality rates for the hyperthermia crisis were still over 80% and after 40 years of research the pathogenesis of the complication was discovered, along with the medical treatment in dantrolene (Bandschapp & Girard, 2012). Current mortality rates have decreased to less than 5%, because of the symptoms being immediately recognized and treated by anesthetists (Bandschapp & Girard, 2012). Malignant hyperthermia is still a very precarious state, yet very treatable under the proper clinician care.