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garrett.erickson@otterbein.edu

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Malignant Hyperthermia
Garrett Erickson, BSN, RN, CCRN
Otterbein University, Westerville, Ohio

Introduction

Malignant hyperthermia (MH) is a potentially life-threatening emergency that any diligent certified registered nurse anesthetist (CRNA) needs to be aware of. MH can occur during the induction of anesthesia, and can occur any time during surgery. MH can occur in as many as one of every 100,000 hospital admissions. Along with a myriad of conditions and potential complications that can occur during anesthesia, MH stands out as one that requires prevention, prompt recognition, and treatment to avoid further complications and death of the patient.

MH is a genetic disorder known as a pharmacogenetic disorder which manifests itself in the skeletal muscle (Heyton, Forger, Scholts, & Veyssens, 2015). When a susceptible patient, who carries the autosomal dominant MH allele variant along with calcium release channel ( RyR1 ) variants: proceedings of the 2013 MHAUS symposium (MacKay, Wilkerson, Hazrati, & Johnson, 2012). Malignant hyperthermia requires immediate recognition and treatment to increase the patient’s chance of survival. Providers need to know its triggers (calcium and/or catecholamines, anesthetics and succinylcholine), and a thorough pre-operative assessment must be completed to identify any history or risk factors for MH. It is important for all OR staff regularly review the standards and procedure for managing an MH crisis, as it is a rare occurrence. The whole operating room must work as a team and be familiar with signs and symptoms of MH and understand the protocol in order to properly react and treat the patient for the best possible outcome.

Pathophysiology

Malignant hyperthermia occurs in patients who have a genetic defect in their ryanodine receptor subtype 1 ( RYR1 ), which is a large ion channel that facilitates the release of calcium from the sarcoplasmic reticulum (SR) in skeletal muscle (Schneiderbanger et al., 2014). Calcium is released from the SR during muscle contraction and release of calcium underpins after muscle contraction has been completed. During an MH crisis, when susceptible patients are exposed to anesthetic agents (volatile anesthetics and succinylcholine), there is a prolonged opening of the RYR1 receptor channels, which results in an uncontrolled release of calcium and unrelenting muscle activation which presents as rigidity (Schneiderbanger et al., 2014). This uncontrolled muscle contraction increased sympathetic activity which stimulates the hypothalamus to release adrenocorticotropic hormone (ACTH) (Stratman, Flynn, & Hatton, 2009). ACTH activates the adrenal gland to produce epinephrine and cortisol, which is a large ion channel that facilitates the release of calcium from the sarcoplasmic reticulum (SR) in skeletal muscle (Schneiderbanger et al., 2014). Calcium is released from the SR during muscle contraction and release of calcium underpins after muscle contraction has been completed. Muscle rigidity, especially in the jaw, chest, and extremities OHM (Hatton, Flynn, & Hyman, 2009).

Early Signs and Symptoms

• Early clinical signs of MH include increased heart rate and end tidal carbon dioxide (Stratman, Flynn, & Hatton, 2009)
• Hyperthermia, with temperature rising as quickly as 1 degree Celsius every 5 minutes (Stratman, Flynn, & Hatton, 2009)
• Muscle rigidity, especially in the jaw, chest, and extremities ODH (Hatton, Flynn, & Hyman, 2009)
• Lactic acidosis and hyperkalemia, myoglobinemia, myoglobinuria, rhabdomyolysis, cardiac arrhythmias, pyrexia, and electrocardiographic abnormalities

Significance of Pathophysiology

Malignant hyperthermia causes significant and potentially lethal distress to the body if not properly recognized and treated. It’s important to realize that since this condition is genetically linked, it can be avoided. Because of its significance, malignant hyperthermia should be a priority during an anesthesia provider’s preoperative assessment. Patient’s should be asked if they or their family members have ever experienced any complications related to anesthesia, specifically relating to a malignant hyperthermia risk. This simple assessment can avoid the risks associated with volatile anesthetics and depolarizing neuromuscular blockers, and prevent the patient from experiencing deadly symptoms such as ventilation fibillation, rhabdomyolysis and renal failure, pulmonary edema, DIC, etc. This condition shares many of the same signs and symptoms of other pathological disorders. It becomes even more important to be diligent with assessment skills and understand the pathology to comprehend why this might happen and cause a lethal condition. Because it is such a pathologically serious disorder, increased education for patients, their families, and providers is necessary and important to provide safe anesthetic care.

Implications for Nursing Care

As mentioned previously, the goal should be to avoid triggering agents in the presence of someone who has a history of MH or has a family member with a history of MH. There are often special OR’s with specific equipment that can be used in these circumstances. However, the RYR1 defect may not be known and a MH crisis may occur in the middle of surgery. MH should be promptly recognized and many things need to happen at the same time.

• All triggering agents should be discontinued, an increase in oxygenation to 100% FiO2 should be completed, and the patient should be given non-triggering reagents and anesthetics such as propofol and/ or midazolam (NHMA, 2010).
• Dantrolene should be administered. Dantrolene is a specific ryanodine receptor antagonist that prevents calcium from being released and prevent the muscle cells from contracting (Seifert, Wahr, Pace, Cochran, & Bagnola, 2014). The dose of dantrolene is 2.5 mg/kg every 5 minutes repeated at a maximum dose of 30 mg/kg. Intravenous infusion should be rapidly infused through a large bore IV (Seifert et al., 2014).

References


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Table 1

<table>
<thead>
<tr>
<th>Early Signs and Symptoms</th>
<th>Increased ICTO2</th>
<th>Muscle Rigidity</th>
<th>Cardiac abnormalities</th>
<th>Generalized erythematous flush</th>
<th>Electrolyte imbalance</th>
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<tbody>
<tr>
<td>(Stratman, Flynn, &amp; Hatton, 2009)</td>
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