Malignant Hyperthermia

Courtney Stevenson
stevenson2@otterbein.edu

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Introduction: Why choose Malignant Hyperthermia?

- Anesthesia providers and perioperative nurses play a crucial role in the identification and successful management of the condition.
- For a patient to survive a malignant hyperthermia crisis, prompt recognition and treatment is vital (Mullins, 2018).

Pathophysiological Processes

Normal Physiology

- In a normal muscle contraction, an action potential causes membrane depolarization and release of calcium from the sarcoplasmic reticulum (SR).
- The calcium moves into the sarcoplasm via the ryanodine receptor. The receptor voltage-gated ion channels.
- The calcium interacts with the tropomyosin-troponin complex. It creates the cross-bridge between actin and myosin and a muscle contraction occurs.
- At the end of muscle contraction, calcium migrates back into the sarcoplasmic reticulum, and another stimulus is needed to release calcium for a muscle contraction (Schneiderbarger et al., 2014).

Underlying Pathophysiology

- MH is a rare yet life-threatening autosomal dominant disorder (affected individual has one copy of a genetic mutation and normal gene on a pair of autosomal chromosomes) (Nagelhof & Elstma, 2010, p. 773).
- MH occurs because of a mutation on the ryanodine receptor, which is the major calcium release channel of the sarcoplasmic reticulum (Nagelhof & Elstma, 2010, p. 774).
- The anesthetic agents trigger these mutations, calcium migrates back, cause an atypical increase in release of calcium from the sarcoplasmic reticulum of skeletal muscle cells (Mullins, 2018).
- This results in sustained muscle contraction and abnormal muscle metabolism.
- The response leads to hypermetabolic activity of increased oxygen consumption, carbon dioxide production, and lactic acid build-up. It can then lead to systemic acidosis, hyperkalemia, and hyperthermia (Smith et al., 2016).

Signs and Symptoms

- MH may present anytime during general anesthesia and the early postoperative phase (Luckey et al., 2019).
- Early and specific sign of MH is an increase in end-tidal CO2 levels with increased ventilations. If succinylcholine is used, the increase can be more rapid (Smith et al., 2018).
- Other signs include tachycardia, hyperkalemia, alveolar hypoventilation, temperature, generalized muscle rigidity, and rhabdomyolysis (Smith et al., 2018).
- Progression of the crisis leads to respiratory and metabolic acidosis, increased serum lactate levels, and myoglobinuria may occur. The patient may also suffer from acute renal failure and disseminated intravascular coagulation (Smith et al., 2018).

Implications for nursing care.

- The severity of MH crisis depends on how quickly the disease process is suspected and treated. It can progress rapidly and an appropriate treatment is initiated (Schneiderbarger et al., 2014).
- The patient should be trained in the PACU and the registered nurses should be prepared and educated for the MH crisis (Borth, 2019).
- The prevalence of MH is estimated at 1:10,000 administered anesthetics, but this is likely underreported (Borth, 2019).
- Half of the patients diagnosed with MH have received a prior unmonitored anesthetic agent (Borth, 2019).
- Although MH is a rare medical emergency, it is vital that clinicians and patients are prepared and educated for the surgical procedure and the risk for complications with a detailed plan in place.