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Malignant Hyperthermia

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Underlying Pathophysiology

The pathophysiological process initiated by these pharmacological insults is characterized by a disruption in the regulation of calcium within muscle cells. This buildup of intracellular calcium results in the initiation of an immense hypermetabolic reaction stimulating widespread muscle contraction (McCance & Heuther, 2014, p. 501). This buildup of intracellular calcium is a result of “uncontrolled release of calcium caused by mutations in the ryanodine receptor subtype 1 (RYR1)” (Schuster, Johannsen, Schneiderbanger, & Roewer, 2013). This gene encodes the channel in the skeletal muscle cell through which calcium flows. “In the presence of an abnormal RYR1 gene in MH-susceptible individuals, a triggering agent such as halothane, isoflurane, sevoflurane, desflurane, or enflurane, either alone or in combination with the depolarizing muscle relaxant succinylcholine, initiates uncontrolled calcium release. This sets off the classic actin-myosin tropion interaction, shortening of muscle fibers, and consequent muscle contraction. The uncontrolled rise in intracellular calcium causes a sustained state of muscle contraction, leading to the hyperthermic MH response” (Diksen, Van Wicklin, Mashman, Neiderer, & Merrill, 2013, p. 331).

Signs & Symptoms

“Almost all patients who are MH susceptible have no phenotypic changes without anesthesia, it is impossible to diagnose susceptibility without either exposure to the “trigger” anesthetics or by specific diagnostic testing” (Rosenberg et al., 2015, p. 2). This reinforces the importance of educating anesthesia providers, and nurses alike on the signs and symptoms of this pharmacological insult. “Characteristic clinical signs of MH during general anesthesia include hypothermia, hyperpyrexia, tachycardia, hyperkalemia, rhabdomyolysis, acute muscle weakness, acidosis, hyperuricemia, and hyperkalemia and hyperthermia” (Schuster et al., 2013, p. 1). The “malignant hyperthermia clinical grading scale” bases the level of severity of MH crises on a combination of rigidity, muscle breakdown, respiratory acidosis, temperature increase, cardiac involvement, and other indicators of metabolic derangement not part of a single process (Heytens, van Scheltema, Scholtes, & Veyckemans, 2010, p. 506). “An increase in end-tidal carbon dioxide despite increased minute ventilation provides an early diagnostic clue” (Rosenberg et al., 2015, p. 1).

Implications for Nursing Care

“Many clinicians are unprepared to manage an MH crisis in the perioperative setting because it requires the use of low-frequency, high-risk skills and procedures” (Cain, Riess, Gettrust, & Novaljia, 2014, p. 301). Nurses should familiarize themselves with the signs and symptoms of MH, and be ready to implement their institutions treatment plan in the event of this complication. The widely accepted treatment for MH is to first remove the triggering anesthetic agent when an impeding MH crisis is expected (Cain et al., 2014, p. 302). Dantrolene is a commonly used medication in the treatment of MH, which directly interferes with skeletal muscle contractions by decreasing the level of calcium in the muscle cells. Dantrolene does not block neuromuscular transmission, but rather potentiates nondepoloizing neuromuscular blockade (Brandom, & Riazi, 2015, p. 17). Active cooling in patients suffering from a MH crisis is paramount, with ice being the most widely used surface coolant. The patient’s core temperature should be lower than 39 degrees Celsius (Cain et al., 2014, p. 302).

Conclusion

MH is a rare, but life-threatening complication. Many clinicians may find themselves unprepared to manage a patient in a MH crisis. Hospitals should encourage their anesthesia providers to take part in routine clinical simulations to be better prepared to react promptly and efficiently. The research surrounding MH is ongoing, and complicated due to the rarity of these events. The cessation of previously known triggering agents, such as halothane, has substantially decreased the incidence of MH in the last 20 years (Heytens, van Scheltema, & Veyckemans, 2015, p. 508). However, the need for research and development of treatment plans for this complication is vital to the continued decline in MH crisis events.

References