Malignant Hyperthermia

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Significance Of Pathophysiology
Long & Raas (2017) stated that if MH is not treated, the body’s innate ability to maintain homeostasis is overthrown and this can lead to cardiovascular collapse and eventually death. Additionally, it is strongly recommended that healthcare providers be prepared to quickly address and treat this frightening event.

Implications Continued
The patient must also be cooled via administration of cold IV fluids, being packed with ice packs, having cold irrigation fluids instilled into open body cavities as well as having a cold lavage of the stomach, rectum, and bladder as well.

Pathophysiology

Introduction
The topic of Malignant Hyperthermia (MH) will be presented in this poster. MH is the clinical expression that occurs when an individual exposed to a triggering agent reacts abnormally to a triggering agent. It is of the utmost importance to healthcare providers to be prepared to quickly address and treat this frightening event.

Signs and Symptoms
Rosenberg et al., (2015) stated that some individuals may have almost immediate reactions with exposure to triggers while others do not until well into the post-operative period.

The early phase presents as a respiratory or circulatory arrest (spontaneous ventilation), tachycardia as well as an irregular heart beat, and a rapid process that presents as muscle rigidity and isomcentration as well as a warm soma line container. The anesthesia provider would then observe an increase in minute ventilation, an increased end tidal CO2, and cardiac dysrhythmias, as well as pushed T waves. This is secondary to the patient having an increased AST, a metabolic and respiratory acidosis, and hyperkalemia due to the release of potassium into the extracellular space (Hines & Marschall, 2012).

Underlying Pathophysiology
In MH, the mutated RYR1 gene receptor threshold is decreased for Ca2+ release. The RYR2 receptors are also resistant to the negative feedback loop (increase Ca2+ and magnesium) that would normally decrease the Ca2+ entry.

This equates to an over release of Ca2+ required to respond to the increased intracellular Ca2+ interaction which causes the cross bridge between actin and myosin to activate. At the completion of the muscle contraction, Ca2+ is sequestered back into the sarcoplasm via the ryanodine receptors, but the Ca2+ in the sarcoplasm causes continued and spontaneous Ca2+ release which is attached to the troponin-tropomyosin complex.

Adenylate triphosphate (ATP) is consumed in every step of intracellular Ca2+ interactions. These steps include: - Desorption of Ca2+ from tropomyosin upper-pocket of Ca2+ - Binding to Ca2+ transporter (Ca2+) to the SR and mitochondria - Extraction into the extracellular milieu (Hines & Marschall, 2012).

The late phase presents with a generalized skeletal muscle rigidity secondary to an increased intracellular calcium and increased serum creatinine kinase level. There will be a prolonged bleeding time and col dal change in urine. The patient will be in primary medical emergency with metabolic acidosis and hyperkalemia associated with tachycardia. As the patient responds to dantrolene, the anesthesia provider will continue to notice cardiac dysrhythmias and pushed T waves but will begin to observe a decrease in the core temperature and as ventricular arrhythmia (Hines & Marschall, 2012).

Inhertances patterns include:
- Autosomal dominant
- Autosomal recessive
- X-linked dominant

Implications for Nursing Care
Schneiderbanger et al., (2014) stated that the severity of a MH crisis depends on how quickly the disease process is suspected and how rapidly an appropriate treatment is initiated. Therefore, MH should be considered in the differential diagnosis of a triggering agent. Anesthesia should immediately be converted to total intravenous anesthesia (TIVA) with the use of fentanyl, sedatives, and if the patient status necessitates, neuromuscular blocking agents to prevent administration of the volatile anesthetic should be removed and the patient hyperventilated with 100% oxygen at maximum fresh gas flow while an adulterated charcoal filter is placed. There must also be an increase in the minute volume by 3:2 times as a MH patient is in an attempt to flush the volatiles anesthetic from the patient system while also aiming to decrease the end-tidal CO2 to within normal limits. Hines & Raas (2017) stated that the main medications that should be used in a crisis is dantrolene sodium at 2.5 mg/kg IV every 5-10 minutes based on actual body weight. Long & Ross (2017) state that this medication interferes with the release of Ca2+ from the SR back into the sarcoplasmic reticulum in the Ca2+ in the myoplasm. This is because dantrolene is a specific ryanodine receptor antagonist. It is recommended that 36 vials of dantrolene sodium be on hand in any facility where the executive agents of MH are used to treat a patient in MH crisis as they will require a very large quantity of dantrolene. Once the dantrolene sodium is administered, and the administration of dantrolene has begun, other supportive measures must be started.

Hines & Raas (2017) states to administer sodium bicarbonate to correct the acid-base balance secondary to the mixed respiratory and metabolic acidosis.

Banek et al., (2013) stated that MH is a rare condition, historically, the MH disease process that presents as muscle hypermetabolic state usually results in exposure to certain anesthetic agents.

Rosenberg et al., (2015) stated that the main clinical features of MH are: - Unexplained elevation of inspiratory carbon dioxide despite an appropriate level of ventilation and oxygenation.
- Tachypnea.
- Tachycardia.
- Cardiac arrest in the presence of MH.
- Progressive metabolic acidosis.

According to Rosenberg et al., (2013), the use of dantrolene sodium, in combination with early recognition, accurate diagnosis, and appropriate treatment, has decreased rates of mortality from 80% in the 1970s to approximately 5% as of 2007.

Clinical Resources
In the clinical setting, the anesthesiologist provider and the anesthesiologist nurse must be able to quickly address and treat MH. One of the primary references is that the anesthesia provider and the support staff must be able to quickly address and treat MH. One of the primary references is that the anesthesia provider and the support staff must be able to quickly address and treat MH.

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