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Malignant Hyperthermia

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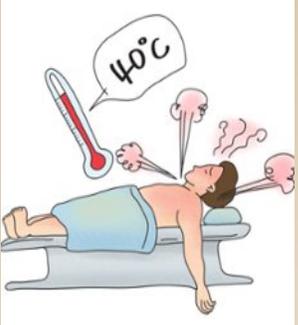
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Malignant Hyperthermia

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Introduction: What is the topic? Malignant Hyperthermia



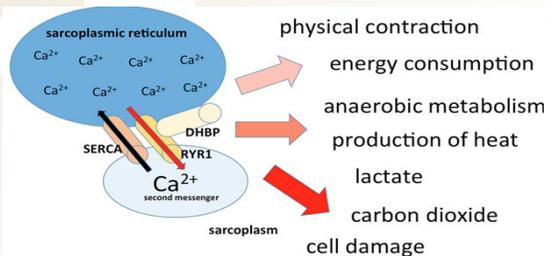
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Why choose Malignant Hyperthermia?

- Anesthesia providers and perioperative nurses play a pivotal 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)
- With proper education and understanding of the pathophysiology of the disorder, negative patient outcomes can be prevented
- The understanding of MH is essential as an anesthesia care provider

Presentation of Case/Process

- A 20-year-old male patient presented following a motor vehicle crash with altered mental status and a laceration to the parietal region of his head.
- The prehospital care providers reported that the driver of a vehicle that struck a concrete guardrail at a high speed.
- Initial assessment revealed a GCS of 10. The decision was made to intubate the patient based on continued deterioration of his mental status
- Vitals signs during intubation were stable at HR of 82 blood pressure 152/70, SpO2 100%, and his temperature was not recorded prior to induction
- Patient was preoxygenated with a non-rebreather mask and high-flow nasal cannula and given 1.5 mg/kg of both ketamine and succinylcholine
- After administration of the succinylcholine, the patient experienced prolonged muscle fasciculations of his pectoral and abdominal muscles, and then desaturated to 60% despite ongoing oxygenation with a nonrebreather mask and nasal cannula
- The patient's glottic opening was easily visualized with a video laryngoscope, and his vocal cords continued to abduct/adduct.
- The emergency medicine resident was able to pass the tube successfully on the first attempt.
- The patient was then placed on continuous waveform capnography, and the end-tidal CO2 (EtCO2) was 71 mmHg.
- The patient also became tachycardic within 2 min of induction, with a heart rate noted in the 150-160 range.
- The treatment team initially felt the patient was inadequately sedated and administered midazolam and fentanyl
- Despite providing adequate ventilation and sedation, the patient remained hypercapnic and tachycardic, with his EtCO2 ranging from 50 to 70 mmHg and heart rate 140-160
- The team suspected MH at this stage and activated the MH protocol. The protocol, prompts a pharmacist to respond to the patient location with dantrolene (Ryanodex) and cooling agents in a designated cart.
- The patient's CAT scan showed spinous process fractures and rib fractures; there were no other injuries and no intracranial abnormality noted.
- Bedside point-of-care chemistry revealed a potassium of 3.7, creatinine of 1.9, venous pH of 7.19, PCO2 of 65 and bicarbonate of 25
- The patient was administered 2.5 mg/kg of dantrolene. His temperature at this time was 37.1°C (98.8°F)
- Patient was admitted to the trauma intensive care unit and progressed to extubation within 36 hr. postinjury
- The patient continued to receive additional doses of dantrolene (Ryanodex) 250 mg every 6 hr. for 24 hr.
- He was discharged on Day 7 postinjury. Patient was neurologically intact and scheduled to follow up for his orthopedic injuries (Luckey-Smith & High, 2018)



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Pathophysiological Processes

Normal Physiology

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

Underlying Pathophysiology

- MH is a rare yet life threatening autosomal dominant disorder (affected individual has one copy of a mutant gene and one normal gene on a pair of autosomal chromosomes) (Nagelhout & Elishia, 2018, p. 773).
- MH occurs because of a mutation on the ryanodine receptor, which is the major calcium release channel of the sarcoplasmic reticulum (Nagelhout & Elishia, 2018, p. 774).
- The anesthetic agents trigger these mutations, and in turn, 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, heat production, and lactic acid build up. Results can then lead to systemic acidosis, hyperkalemia, and hyperthermia (Smith et al., 2018).

Signs and Symptoms

- MH may present anytime during general anesthesia and the early postoperative phase (Mullins, 2018)
- Early and specific sign of MH is an unexplained rise in end-tidal CO2 levels with increased ventilation. If succinylcholine is used, the rise in CO2 may be more rapid (Smith, et al, 2018)
- Other symptoms include, tachycardia and dysrhythmia, abrupt rise in core body temperature, generalized skeletal muscle rigidity and rhabdomyolysis (Smith et al, 2018)
- Progression of the crisis leads to respiratory and metabolic acidosis, increased serum potassium, elevated creatine kinase levels, and myoglobinuria may occur. The patient may also suffer from acute renal failure and/or disseminated intravascular coagulation (Smith et al., 2018)

Implications for nursing care cont.

- Once stabilized, patient should be prepared to go to the ICU (Borth, 2019) →
 - decreased heart rate
 - decreased end-tidal carbon dioxide (EtCO2)
 - resolution of hyperkalemia
- Regular MH drills should be performed to demonstrate that staff can mobilize MH supplies (Borth, 2019)
- It is required that anesthesia staff along with the registered nurses working in the PACU must know the response plan for an MH event (Borth, 2019)



Conclusion

- The prevalence of MH is estimated at 1:100,000 administered anesthetics, but this is likely underreported. (Borth, 2019)
- Half of the patients diagnosed with MH have received a prior anesthetic without manifesting MH. (Borth, 2019)
- Without treatment, the mortality rate for an acute MH episode is as high as 80% (Borth, 2019)
- Complications increase 3X per every 2°C increase in temperature and 1.6 X per 30-minute delay in dantrolene administration (Borth, 2019)
- All staff members involved in the care of patients receiving MH-triggering agents should receive annual education on MH treatment (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

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MALIGNANT HYPERTHERMIA	
By T. Kyle Harrison, MD • Larry F. Chu, MD • Sara Goldhaber-Fiebert, MD	
SIGNS	EARLY: 1. Increased EtCO ₂ 2. Tachycardia 3. Tachypnea 4. Acidosis 5. Masseter spasm/trismus
	LATE: 1. Hyperthermia 2. Trunk/limb rigidity 3. Myoglobinuria
INFORM SURGEON CALL FOR HELP CALL FOR MH CART • START PREPARING DANTROLENE!	
RULE OUT	- Light anesthesia - Hypoventilation - Over-heating (external) - Thyroid storm - Pheochromocytoma - Hypoxemia
TREATMENT	1. Discontinue anesthetic triggers (volatiles and succinylcholine) and increase fresh gas flow to 10 L/min. Do NOT change machine or circuit. 2. Convert to TIVA for maintenance. 3. Hyperventilate . FiO ₂ 100%, high flow O ₂ . 4. Prepare 2.5 mg/kg IV Dantrolene bolus . Dilute each 20 mg Dantrolene vial in 60 mL sterile water. 5. Rapidly administer dantrolene . Continue giving until patient stable (may give up to 10 mg/kg). 6. Administer sodium bicarbonate 1-2 mEq/kg for metabolic acidosis/hyperkalemia.

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