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Malignant Hyperthermia
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Introduction

A crisis of malignant hyperthermia is a medical emergency, and must be treated immediately with a coordinated, multidisciplinary team response in order to give the patient the highest chance for a successful recovery (Dirksen, Van Wicklin, Neiderer, & Merritt, 2013). Malignant hyperthermia is predictable, preventable, and treatable by Bandschapp & Girard, (2012), as "a disturbance of the skeletal muscle, occurring homogeneously triggered by volatile anesthetics and depolarizing muscle relaxants."

Although the prevalence rate is low there is good chance an anesthesiologist will experience malignant hyperthermia crisis in his/her career. A study by Cain et al. (2012) found the mortality rate for the healthcare providers is 50,000 to 150,000 times more likely to occur compared to the chance of dying in a traffic accident (Cain, Riess, Gettrust, & Novalija, 2014). Many of these deaths are preventable. Therefore, every person involved in the OR team should be clued to the symptoms, and dantrolene's role in the therapy. The underlying pathophysiology behind malignant hyperthermia begins with the excitation-contraction coupling mechanism where the muscle is suddenly overpowered by the excessive amount of calcium in the myoplasm, and reaches over 39.5 C. The CRNA, now at bedside, must be able to rapidly make the diagnosis and implement treatment. This requires a thorough surgical and anesthesiology history, and every person involved in the OR team must be prepared in advance to handle any crisis that may occur during surgery in the recovery period (Cain, Riess, Gettrust, & Novalija, 2013).

Case Presentation

A 34 y.o. man was admitted to the hospital for an emergency surgery. On preop assessment the CRNA found no signs or symptoms of malignant hyperthermia, a family history of anesthetic complications, but the patient could not tell whether his father had the condition or not. The patient had Nurse Anesthesia program, and providing information about this crisis spread awareness about a situation that could affect my colleagues' or my own career. The condition was termed in 1960, by Douglas Hood. In 1954, the disorder was described in a family with this autosomal dominant inheritance (Bandschapp & Girard, 2012). Yearly, more than 500 cases are reported, and more than 50,000 people are susceptible to malignant hyperthermia (Bandschapp & Girard, 2012). The event of malignant hyperthermia is very rare, but can w Ally progress to a life-threatening situation (Cain, Riess, Gartrutt, & Novela, 2014). Dirksen et al. (2012, p.1) states that malignant hyperthermia occurs in approximately 1,300,000 to 5,000,000 surgical procedures each year in the United States are used.

Presentation of Case

The patient reports muscle rigidity of the patient during the surgical procedure, and the patient could not tell what Dr. was doing. The CRNA does not have any problems with the removal of the ventilator, surgical, or anesthetic agents. On the other hand, there are no apparent complications. After an hour has passed, the patient's vital signs start to deteriorate. HR 75 bpm, BP, 127/82, RR, 16/min, C02, 30 mmHg, SatO2, 96% and body temp 39.5 C. The CRNA, now at bedside, quickly makes the diagnosis of malignant hyperthermia and administers 2mg/kg of dantrolene and 2mg/kg of bicarbonate andministered. A blanket is then applied to the patient and an ABG and labs are done that show: pH, 7.21; Po2, 57mmHg; Po2, 80, PaCO27, 56mmHg. The patient is then history 10 units of regular insulin IV, followed by 50 of 50% dextrose in water IV 4%. The patient is then transferred to the ICU unit (Anderson-Pompa, Foster, Parker, Wilkins & Chess, 2009). This is the most common signs and symptoms of theoperative emergency, malignant hyperthermia (MH). This is a condition that develops rapidly and must be treated immediately. This genetic skeletal muscle disease does not always happen immediately, some cases like this example, the signs and symptoms can occur after surgery during the recovery period (Dirksen, Van Wicklin, Neiderer, & Merritt, 2014). The condition results in the body developing into a hypermetabolic state with lactate, pyruvate, K+ and Ca++ overload, metabolic acidosis, and changes in electrolyte and acid-base balances. The patient displays, signs and symptoms of malignant hyperthermia such as fever (greater than 39º C), myoglobinuria and eventually renal failure, hyperkalemia, metabolic acidosis, altered mental status, rhabdomyolysis associated with hyperthermia. Once MH is suspected, all triggering agents should be discontinued including any anesthetic agents or muscle relaxants (Dirksen, Van Wicklin, Neiderer, & Merritt, 2012). The condition results in the body developing into a hypermetabolic state with lactate, pyruvate, K+ and Ca++ overload, metabolic acidosis, and changes in electrolyte and acid-base balances. The patient displays, signs and symptoms of malignant hyperthermia such as fever (greater than 39º C), myoglobinuria and eventually renal failure, hyperkalemia, metabolic acidosis, altered mental status, rhabdomyolysis associated with hyperthermia. Once MH is suspected, all triggering agents should be discontinued including any anesthetic agents or muscle relaxants (Dirksen, Van Wicklin, Neiderer, & Merritt, 2012). The condition results in the body developing into a hypermetabolic state with lactate, pyruvate, K+ and Ca++ overload, metabolic acidosis, and changes in electrolyte and acid-base balances. The patient displays, signs and symptoms of malignant hyperthermia such as fever (greater than 39º C), myoglobinuria and eventually renal failure, hyperkalemia, metabolic acidosis, altered mental status, rhabdomyolysis associated with hyperthermia. Once MH is suspected, all triggering agents should be discontinued including any anesthetic agents or muscle relaxants (Dirksen, Van Wicklin, Neiderer, & Merritt, 2012). The condition results in the body developing into a hypermetabolic state with lactate, pyruvate, K+ and Ca++ overload, metabolic acidosis, and changes in electrolyte and acid-base balances. The patient displays, signs and symptoms of malignant hyperthermia such as fever (greater than 39º C), myoglobinuria and eventually renal failure, hyperkalemia, metabolic acidosis, altered mental status, rhabdomyolysis associated with hyperthermia. Once MH is suspected, all triggering agents should be discontinued including any anesthetic agents or muscle relaxants (Dirksen, Van Wicklin, Neiderer, & Merritt, 2012).

Pathophysiology and Significance

The underlying pathophysiology behind malignant hyperthermia begins with the excitation-contraction coupling mechanism where the muscle is suddenly overpowered by the excessive amount of calcium in the myoplasm, and reaches over 39.5 C. The CRNA, now at bedside, quickly makes the diagnosis of malignant hyperthermia and administers 2mg/kg of dantrolene and 2mg/kg of bicarbonate andministered. A blanket is then applied to the patient and an ABG and labs are done that show: pH, 7.21; Po2, 57mmHg; Po2, 80, PaCO27, 56mmHg. The patient is then history 10 units of regular insulin IV, followed by 50 of 50% dextrose in water IV 4%. The patient is then transferred to the ICU unit (Anderson-Pompa, Foster, Parker, Wilkins & Chess, 2009). This is the most common signs and symptoms of theoperative emergency, malignant hyperthermia (MH). This is a condition that develops rapidly and must be treated immediately. This genetic skeletal muscle disease does not always happen immediately, some cases like this example, the signs and symptoms can occur after surgery during the recovery period (Dirksen, Van Wicklin, Neiderer, & Merritt, 2014). The condition results in the body developing into a hypermetabolic state with lactate, pyruvate, K+ and Ca++ overload, metabolic acidosis, and changes in electrolyte and acid-base balances. The patient displays, signs and symptoms of malignant hyperthermia such as fever (greater than 39º C), myoglobinuria and eventually renal failure, hyperkalemia, metabolic acidosis, altered mental status, rhabdomyolysis associated with hyperthermia. Once MH is suspected, all triggering agents should be discontinued including any anesthetic agents or muscle relaxants (Dirksen, Van Wicklin, Neiderer, & Merritt, 2012). The condition results in the body developing into a hypermetabolic state with lactate, pyruvate, K+ and Ca++ overload, metabolic acidosis, and changes in electrolyte and acid-base balances. The patient displays, signs and symptoms of malignant hyperthermia such as fever (greater than 39º C), myoglobinuria and eventually renal failure, hyperkalemia, metabolic acidosis, altered mental status, rhabdomyolysis associated with hyperthermia. Once MH is suspected, all triggering agents should be discontinued including any anesthetic agents or muscle relaxants (Dirksen, Van Wicklin, Neiderer, & Merritt, 2012).

This process is significant in that it rapidly depletes adenosine triphosphate (ATP) and increases glucose metabolism, carbon dioxide production, heat production, and causes the generation of lactic acid (Revello, 2012). As ATP stores become exhausted, the 

References


