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Pseudocholinesterase Deficiency: Implications in Anesthesia
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Introduction

The use of neuromuscular blocking medications is a frequent and essential practice in establishing a secure airway in a patient preparing for surgery or in emergent situations such as respiratory distress. As an anesthesiologist, it is vital to know the mechanism of action, expected duration, and metabolism of the neuromuscular blocking agents, particularly succinylcholine. A potential adverse complication that may occur in patients that have been administered succinylcholine is prolonged neuromuscular blockade due to the deficiency of pseudocholinesterase. When succinylcholine is administered, the enzyme produced in the liver that metabolizes neuromuscular blocking agents, particularly succinylcholine and nirvacurium, as well as some local anesthetics. Pseudocholinesterase deficiency is caused by either a homozygous or heterozygous abnormal genetic variant of the enzyme itself. An individual that is homozygous for the enzyme deficiency can occur in one in every 3,500 people, the neuromuscular blocking agent succinylcholine will likely last four to eight hours and this genotype is more likely to experience apnea and the inability to be taken off of the ventilator post-operatively. An individual with a heterozygous gene which occurs in one in every 400 people, will experience the effects of succinylcholine two times longer than an individual with a normal genotype. There are multiple other causes that are less significant, but may cause a decrease in pseudocholinesterase activity including liver disease, old age, pregnancy, burns, malnutrition, neoplastic disease, the use specific medications such as reglan, oral contraceptives, monamine oxidase inhibitors, and anticholinesterase medications (Stoelting & Hiller, 2015).

Pathophysiology

- Pseudocholinesterase is an enzyme produced in the liver that metabolizes neuromuscular blocking agents, particularly succinylcholine and nirvacurium, as well as some local anesthetics.
- Pseudocholinesterase deficiency is caused by either a homozygous or heterozygous abnormal genetic variant of the enzyme itself.
- An individual that is homozygous for the enzyme deficiency can occur in one in every 3,500 people, the neuromuscular blocking agent succinylcholine will likely last four to eight hours and this genotype is more likely to experience apnea and the inability to be taken off of the ventilator post-operatively.
- An individual with a heterozygous gene which occurs in one in every 400 people, will experience the effects of succinylcholine two times longer than an individual with a normal genotype.
- There are multiple other causes that are less significant, but may cause a decrease in pseudocholinesterase activity including liver disease, old age, pregnancy, burns, malnutrition, neoplastic disease, the use specific medications such as reglan, oral contraceptives, monamine oxidase inhibitors, and anticholinesterase medications (Stoelting & Hiller, 2015).

Significance of Pathophysiology

- While pseudocholinesterase deficiency is not one of the most common complications seen with general anesthesia, it can be fatal if not recognized and treated properly.
- Since we know there is a genetic link, a thorough pre-operative assessment should be completed including family history of this condition. Sometimes asking if the patient or a family member was admitted to the ICU or had to be on the ventilator for longer than expected after surgery will help to reveal a pseudocholinesterase deficiency.
- The deficiency is most often found after succinylcholine has already been given and it is discovered that the patient’s muscle function has not returned within the expected timeframe.
- There is no treatment for this genetic condition, however supportive care, particularly ventilator support, must be given until the medication has been adequately metabolized and the patient has regained muscle strength (Stoelting & Hiller, 2015).

Implications for Nursing Care

- Nursing anesthesia providers to know the mechanism of action and potential adverse effects of all medications that are administered peri-operatively.
- Always assess muscle function using train of four monitoring with a peripheral nerve stimulator prior to an after a neuromuscular blockade is given. This will allow for comparison and the provider can be more confident in the degree of muscle strength recovery prior to making the decision to extubate the patient. (International Anesthesia Research Society, 2016).
- Do not extubate a patient that has not experienced muscle recovery, provide full ventilator support, and make arrangements for a ventilator set-up in the PACU (Stoelting & Hiller, 2015).
- Avoid the use of succinylcholine unless absolutely necessary and do not give until the benefits outweigh the potential costs (Quinn, 2012).
- PACU and ICU nurses should familiarize themselves with the significance of a pseudocholinesterase deficiency, the use of a peripheral nerve stimulator, and the necessary care for these patients postoperatively (Stoelting, 2003).

Signs and Symptoms

- A patient with pseudocholinesterase deficiency will not experience signs or symptoms unless he or she is administered a neuromuscular blocking agent.
- The medication sign for an individual with succinylcholine paralysis includes the diaphragm and intercostal muscles that are required for the patient to breath. Apnea and a lack of movement or muscle twitching when using a peripheral nerve stimulator will present until the medication is metabolized by the body which may not be until hours later (Whittington, et. al, 2012).
- The peripheral nerve stimulator is a method for monitoring neuromuscular blockade in which electrodes are placed either on the facial or vulvar nerve and stimulated using various modes to produce a muscle twitch. Train-of-four is a commonly used mode in which four simultaneous stimuli are deployed and the number and strength of the stimuli is observed by the anesthesia provider. The goal after using a neuromuscular blocking agent and prior to extubation is four twitches without fade which refers to the strength of the twitch. An individual with a pseudocholinesterase deficiency may not produce any twitches depending on the amount of time that has passed since administration and the type of genetic variant (Nagylko, & Plaus, 2013).

Additional Resources


References


