Pseudocholinesterase Deficiency: Implications in Anesthesia

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The major presenting sign is prolonged skeletal muscle paralysis including the muscle strength will be restored, however its duration of action will be prolonged in a deficient patient depending on the type of genetic abnormality present.

Pathophysiology

Succinylcholine is an enzyme produced in the liver that metabolizes neuromuscular blocking agents, particularly succinylcholine and neostigmine, as well as some local anesthetics.

Succinylcholine hydrolyzes succinylcholine in the plasma so quickly that only 10% of the medication actually reaches and acts upon the neuromuscular junction (NMJ).

Once at the NMJ, succinylcholine binds to acetylcholine receptor sites causing sustained depolarization for a relatively short period of time in which the patient experiences skeletal muscle fasciculations followed by flaccid paralysis.

Succinylcholine diffuses away from the NMJ down its concentration gradient at which point muscle function will return.

Typically, an intravenous dose of succinylcholine is rapidly hydrolyzed by succinylcholine within 9 to 13 minutes in which 90% of the muscle strength will be restored, however its duration of action will be prolonged in a deficient patient depending on the type of genetic abnormality present.

Significance of Pathophysiology

Pseudocholinesterase deficiency is caused by either a heterozygous or homozygous abnormal genetic variant of the enzyme itself. An individual that is homozygous for the genetic abnormality, the enzyme is inactivated and the patient is unable to metabolize succinylcholine.

If the patient has a normal genotype, 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.

Individuals with a heterozygous gene which occurs in one in every 480 people, will experience the effects of succinylcholine two times longer than an individual with a normal genotype. There are many other causes that are less significant, but may cause a decrease in pseudocholinesterase activity including liver disease, old age, pregnancy, burn, malnutrition, neoplastic disease, the use of specific medications such as statins, oral contraceptives, monoclonal antibodies, and anticholinesterase medications (Stoelting & Hiller, 2015).

Implications for Nursing Care

Nursing anesthesia providers to know the mechanism of action and potential adverse effects of all medications especially neuromuscular blockers in order properly recognize and treat a pseudocholinesterase deficiency.

Always assess muscle function using train of four 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 Anesthesiology 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 saline unless the benefits outweigh the potential costs (Quyen, 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.

Additional Resources


Conclusion

This condition will not affect the patient’s everyday life, however it may become a life threatening condition if the patient must undergo general anesthesia for surgery.

For these reasons anesthests providers should always consider using less invasive airway options or nerves blocks, both of which would not require the administration of neuromuscular blocking agents, like succinylcholine.

If the use of a NMB cannot be avoided and there are no absolute contraindications, then providers must be prepared to recognize, assess, and provide support to a patient that presents with a pseudocholinesterase deficiency.

References
