Pseudocholinesterase Deficiency

Anesthetic Pearls: Anesthetic Implications and Management of Pseudocholinesterase Deficiency

Pseudocholinesterase Deficiency: Plasma cholinesterase is an enzyme found in plasma and most tissues (except RBC), which degrades acetylcholine released from the neuromuscular junction. The plasma cholinesterase enzymes are manufactured in the liver. This enzyme is of interest to anesthesiologists because the action of commonly used drugs such as **succinylcholine**, **mivacurium** and rarely **ester-linked local anesthetics** can be altered with abnormalities in these enzymes.

Factors which may <u>decrease</u> cholinesterase activity:

The body has a large reserve of this enzyme; <u>a decrease in enzymatic activity **greater than 75%** is clinically significant</u>.

1. **Inherited Cholinesterase Variants**
2. **Disease**: liver disease, malnutrition, some collagen disease, uremia, malnutrition, myxedema, burns
3. **Treatments**: plasmapheresis, extracorporeal circulation, radiation therapy
4. **Drugs**: Ecothiphate, Neostigmine, Pyridostigmine, Cyclophosphamide, MAO-Inhibitors, Pancuronium, Contraceptives, Cyclophosphamide.
5. **Physiologic**: decreased in 3rd trimester of pregnancy, decreased activity in newborns

Inherited cholinesterase variants & the Dibucaine Number:

It is felt that a single gene locus is responsible for the abnormal cholinesterase with many genetic variants. Of these abnormal genes, the Dibucaine variant is the most common. Dibucaine is a local anesthetic (amide), which inhibits pseudocholinesterase to varying degrees depending on the type of enzyme. The **Dibucaine Number (DN)** represents the percentage of enzyme inhibition by the local anesthetic.

80% inhibition = normal individual. Normal response to succinylcholine
60% inhibition = heterozygous. Most will still have normal response to succinylcholine.
20% inhibition = homozygous. Marked prolongation of succinylcholine.

Clinical implications:

A. The duration of action of **Succinylcholine** is determined by its metabolism. It is estimated that less than 5% of an IV dose actually reaches the site of action due to rapid metabolism. As anesthesiologists, we need be acutely aware of acquired conditions, drugs, and diseases which may alter the patients response to succinylcholine.
B. Succinylcholine induced neuromuscular blockade in homozygous patients can last up to 300 minutes. They have a similar onset of blockade and display onset fasiculations. There are often no early warning signs at the beginning of the anesthetic as to who may have an abnormal variant. The safest treatment in a patient who fails to breath with 15 minutes after succinylcholine administration is continued mechanical ventilation until the patient regains adequate muscle tone.
C. Two controversial additions to this conservative treatment are administration of a reversal agent and administration of blood. Patients may see a temporary improvement in symptoms after neostigmine, but may re-paralyze when it wears off or get intensification of the block if it is given before there is evidence of fade. A blood transfustion of 2 units of pRBC's may contain enough RBC and plasma pseudocholinesterase to reverse the block however it puts the patient at risk for transfusion reaction or viral infections.
D. Patients with a suspected abnormal pseudocholinesterse reaction should be hemotologically tested and wear a medical alert bracelet to avoid further incidence.