FOX VALLEY EQUINE PRACTICE

1N303 LaFox Rd, La Fox, IL 60147
FVEP@foxvalleyequine.com
(630) 365-5600

Hyperkalemic Periodic Paralysis (HYPP)

Definition

Hyperkalemic periodic paralysis (HYPP) is a genetic condition affecting Quarter horses and Quarter horse crosses. Kalemia refers to the presence of potassium in the blood. Hyperkalemia is when there are excess levels of blood potassium. Horses with HYPP experience episodes of abnormal muscle contraction when blood potassium levels rise. This condition is heritable in an autosomal dominant manor, meaning offspring can be affected even if only one parent carries the genetic abnormality. Interestingly, the genetic mutation can be traced all the way back to a Quarter horse stallion named "Impressive" who was born in 1969. Because of this stallion's popularity as a halter horse, 4% of today's Quarter horses are affected by HYPP as a result of extensive breeding.

Pathophysiology

Horses with HYPP have a genetic mutation resulting in abnormal skeletal muscle sodium channels, which are important in muscle contraction. These sodium channels reside on the surface of skeletal muscle cells. When sodium is allowed to enter the cell, it initiates cellular changes ultimately leading to muscle contraction. In horses with HYPP, their abnormal sodium channels become "leaky" and allow sodium to enter the cell in an irregular fashion. This results in muscle excitability and uncontrolled contraction. The sodium channels become leaky when blood potassium levels rise (hyperkalemia).

Clinical Signs

Some horses with the genetic mutation appear clinically unaffected while others show signs of the condition. When signs do develop, they tend to appear around two years of age. Repeated bouts of muscle fasciculations, trembling, sweating, and third eyelid prolapse can occur when blood potassium levels rise. "Dog-sitting," collapse, laryngeal dysfunction, and respiratory muscle failure have also been reported but tend to occur much less frequently. These attacks can last anywhere from minutes to hours. Diets high in potassium such as alfalfa hay and molasses can trigger an attack as well as sedation, concurrent illness, or a stressful situation such as trailer rides or horse shows.

Diagnosis

Diagnosis is made upon signalment, history, clinical signs, and genetic testing. Genetic testing is common in susceptible breeds and can be performed inexpensively with a sample of the horse's mane/tail hair or blood. DNA testing of all breeding horses of susceptible breeds is strongly encouraged as breeding affected horses should be avoided. The AQHA registry requires HYPP genetic testing of all "Impressive" descendants born in 2007 or after.

Treatment

Many horses with mild cases spontaneously recover from an episode without any treatment, however, an owner can offer the horse corn syrup or grain to induce a hyperglycemic state as an aid to lower potassium levels. The main goal of treatment, particularly in more severe cases, is aimed at decreasing blood potassium levels. This can be achieved by administering dextrose, sodium bicarb, or calcium gluconate. Intravenous administration of dextrose results in a hyperglycemic state and insulin release which in turn drives potassium out of the bloodstream.

Prevention

Diet is key to aiding in prevention of an attack. Grass hay, pasture grass, oats, corn, and beet pulp are good alternatives to alfalfa hay, molasses, and corn oil, which are high in potassium. Feeding smaller meals more frequently throughout the day will also aid the body in maintaining steady electrolyte levels as opposed to big meals less frequently. Regular exercise and reducing stress are also recommended. Acetazolamide may be recommended in some cases to increase renal potassium excretion.

Prognosis

With a low-potassium diet and consistent exercise, many horses do quite well with infrequent to no attacks. Regular acetazolamide administration can be used in some cases to help horses maintain proper potassium levels longer term. Horses homozygous for HYPP (inherited the genetic mutation from both the dam and sire) are more clinically affected than heterozygotes (inherited the genetic mutation from only one parent). The best course of action is to talk with your vet on how to best manage an affected horse and to create a plan going forward.