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CURRENT DIAGNOSIS and NUTRITIONAL MANAGEMENT

of Tying-Up

Researchers estimate that three of every 100 performance horses will experience signs consistent with a diagnosis of tying-up. For many horsemen the signs of tying-up are unmistakable—stiff, stilted gaits and loss of impulsion to the point of stopping and stretching as if to urinate or paw the ground. When pressed to continue exercise, horses sweat excessively, breathe more quickly, and become so painful they might stop and remain standing in one place.

Two important research milestones transformed the way scientists now approach tying-up. The first involved the recognition of multiple causes of acute muscle pain and cramping. Speaking at the 2010 Kentucky Equine Research (KER) Nutrition Conference, held in Lexington, Kentucky, Stephanie Valberg, D.V.M., Ph.D., a professor at the University of Minnesota, said, “For decades, progress in understanding tying-up was hampered by searching for one specific cause of muscle pain and by the false assumption that tying-up pain was due to lactic acidosis. In fact, there is no scientific validation that lactic acid accumulates in the muscle of horses with tying-up, and many valid studies show that lactic acid is not present in high levels in horses with tying-up.”

A second research landmark was the adaptation of a muscle biopsy technique for horses. According to Valberg, over the last 20 years, the use of muscle biopsies has been integral in diagnosing problems in individual horses and advancing the knowledge of muscle disease in general. “The repository of over 3,000 muscle and DNA samples from across North America at the Neuromuscular Diagnostic Laboratory, University of Minnesota, has proved to be invaluable in further defining the histological, clinical, epidemiological, pathological, and genetic basis for specific muscle disorders in horses.”

Valberg and other researchers are unraveling the genetic basis for tying up. As equine genome mapping techniques progress, DNA-based tests are increasingly becoming part of the diagnostic approach to muscle disease in horses. Four DNA-based tests for muscle disorders in horses are available. Individual tests identify hyperkalemic periodic paralysis (HYPP), glycogen branching enzyme deficiency (GBED), malignant hyperthermia (MH), and type 1 polysaccharide storage myopathy (PSSM1). DNA diagnosis uses hair roots or blood samples, and provides a less invasive and more accurate diagnosis than histological interpretation of muscle biopsy.

Left: Regardless of the discipline in which they are used, Thoroughbreds are often diagnosed with tying-up. For this steeplechaser, such a diagnosis could keep him from being a useful racehorse unless proper management strategies are followed.

Right: Tying-up occurs in about 3% of exercised horses. Because of its often chronic nature, it can be a frustrating condition to manage.

Valberg mentioned that advances in establishing accurate diagnosis for the cause of tying-up are invaluable because precise diagnosis “(1) defines the likelihood of recurrence of the condition; (2) establishes reasonable expectations for the horse; (3) provides for the appropriate selection of targeted dietary therapy and exercise regimes; and (4) determines the likelihood that the horse will pass on the disorder to potential offspring.”

Case Studies

As part of her presentation at the KER Nutrition Conference, Valberg described three case studies of horses with muscle disease and the recommended therapies for each.

The first case presented was a three-year-old Quarter Horse gelding with typical signs of tying-up. For this gelding, exercise intolerance and toe dragging occurred at 15-20 minutes after the onset of exercise. Physical examination revealed symmetrical muscle development and normal neurological assessment. Lameness examination uncovered no unsoundness. Elevated serum creatine kinase (CK) levels indicate muscle damage has occurred, but this gelding's resting levels were normal. The exercise challenge involved a two-minute walk and a two-minute trot, which was repeated for 15 minutes with careful observation. CK levels following exercise were abnormally high. PSSM and MH were considered possible diagnoses. Diagnostic approach included genetic testing and biopsy. Genetic testing, which normally requires a 7- to 14-day turnaround time, revealed the horse had PSSM1 and MH.

Nutritional management was the key to keeping this horse pain-free. In general, avoiding high-starch feeds is one way to treat PSSM. A low-starch, high-fat, high-fiber feed (Re-Leve[®] Original) is indicated for horses with high energy needs. PSSM horses are often easy keepers, so rations should not exceed daily caloric requirement. For this gelding, Re-Leve[®] Concentrate was indicated, as this formula reduced calorie consumption but contained essential protein, vitamins, and minerals. Hay with a low nonstructural carbohydrate (NSC) content should be selected. The outcome for this horse was positive with less stiffness, though there was occasional recurrence of disease in spring and fall.

The second case involved an eight-year-old Dutch Warmblood gelding. He performed well for his owner for six months but then became lame in a foreleg. Once the lameness was addressed, he became difficult to ride with trouble holding the canter and periodic bucking. Lameness evaluation revealed a torn hind suspensory ligament. After a three-month rest, he was put back into work. Follow-up physical examination revealed weight loss and tight musculature, especially over the back. When worked on the longe line, he was explosive initially and then lost all enthusiasm. Muscle biopsy revealed excessive glycogen concentrations. The genetic test for PSSM1 was normal. The diagnosis, therefore, was PSSM2. Reworking this gelding's nutrition helped significantly. Alfalfa hay was replaced with a low-NSC hay, and circulating fat was increased by feeding a moderate amount of Re-Leve[®]. Exercise included light work, with collection and can-

ter work beginning after one month. The horse returned to normal with this protocol.

The final case concerned a 10-year-old Quarter Horse that was used for dressage. He was being fed a high-fat diet and grass hay. He had chronic lameness issues. After a long, hard lesson, the horse seemed to be suffering from tying-up with stiffness and mild elevation of CK. The owner felt the gelding's gaits were not as fluid as they had been, particularly the canter. He became incredibly short-strided and stiff. Genetic testing revealed no PSSM or MH. Muscle

biopsy revealed huge lipid droplets in certain fibers, and the notion of a lipid storage myopathy was advanced by Valberg. To appropriately manage this horse, he was placed on oats and a ration balancer as well as a good-quality grass hay. He was turned out as often as possible, and he recovered fully.

Over the last few decades, research has played a pivotal role in the diagnosis of tying-up. Likewise, nutritional management has become an essential part of any treatment plan for horses with the disease.

REFERENCE GUIDE TO TYING-UP

Separating one form of tying-up from another can be difficult without a working knowledge of the various diseases. Here is a quick guide to tying-up syndromes.*

POLYSACCHARIDE STORAGE MYOPATHY (PSSM). Two forms exist, type 1 and type 2.

TYPE 1 PSSM (PSSM1):

Breeds affected: At least 20 breeds. Quarter Horse-related bloodlines, Belgians, Percherons, Morgans, Mustangs, Tennessee Walkers, and some Warmblood breeds.

Bloodlines: Widespread in many breeds; in the Quarter Horse it is most common in halter horses.

Prevalence: 36-50% of Belgians and Percherons, 8% of the Quarter Horse-related breeds. Rare in Clydesdales and Shires.

Age affected: Signs usually begin by 2 to 3 years of age but may occur in weanlings. May be subclinical.

Clinical signs: Firm painful muscles, stiffness, skin twitching, sweating, weakness, and reluctance to move with light exercise. Sometimes gait abnormalities, mild colic, and muscle wasting. Serum CK and AST activity elevated except in drafts.

Testing: Muscle biopsy samples evaluated for presence of amylase-resistant crystalline polysaccharide.

Genetic testing on mane or tail hair roots, or unclotted blood samples.

TYPE 2 PSSM (PSSM2):

Breeds affected: Quarter Horse-related breeds, a few Arabians and possibly other light breeds.

Paints, Quarter Horses, and Appaloosas are just as susceptible to tying-up as members of other breeds.



Age affected: Signs usually begin by 2 to 3 years of age but may occur in weanlings.

Clinical signs: Rhabdomyolysis with or without exercise.

Testing: Muscle biopsy samples evaluated for presence of abnormal polysaccharide.

MALIGNANT HYPERTHERMIA (MH)

Breeds affected: Quarter Horse-related bloodlines.

Bloodlines: Present at a very high frequency in two Quarter Horse bloodlines. Often coexists with PSSM.

Prevalence: <1% of the Quarter Horse breed is affected.

Age affected: Adults.

Clinical signs: High temperature, metabolic failure, and death under anesthesia. Exertional rhabdomyolysis especially if present with a certain gene mutation.

Testing: Genetic testing.

RECURRENT EXERTIONAL RHABDOMYOLYSIS (RER)

Breeds affected: Thoroughbreds and possibly Standardbreds and Arabians.

Bloodlines: Unknown, possibly more common in those selected for nervousness and speed combined.

Prevalence: 5-10% of Thoroughbreds.

Age affected: Signs usually present when horses are fit, fed more than five pounds of high-starch concentrate, and when excited. More common in young fillies than geldings.

Clinical signs: Firm painful muscles, lameness,

stiffness, sweating, short stride, and reluctance to move after moderate exercise.

Testing: Based on breed, clinical signs, lack of histopathological evidence of PSSM in muscle biopsy samples.

GLYCOGEN BRANCHING ENZYME DEFICIENCY (GBED)

Breeds affected: Quarter Horse-related bloodlines.

Bloodlines: Horses descended from Zantanon and King.


Prevalence: 8% of the Quarter Horse breed are carriers.

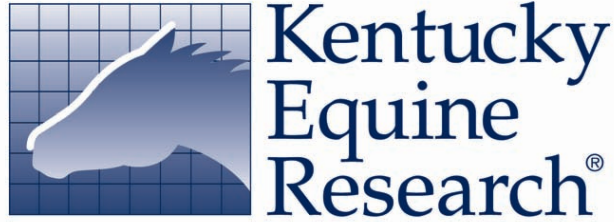
Age affected: Signs usually present in utero or at birth.

Clinical signs: Abortion or stillbirth, may be born alive or weak at birth. With supportive care may live up to 18 weeks of age. Death may be sudden when exercised on pasture, associated with weak respiratory muscles or the result of euthanasia due to persistent recumbency. Treatable flexural deformities of all limbs and recurrent hypoglycemia (low blood sugar) and seizures occur in some affected foals.

Testing: Histopathological tissue samples or genetic testing.

*Valberg, S.J. 2010. "Pursuing the Genetic Basis for Tying-Up Syndromes in Equine Breeds." In: Proc. Kentucky Equine Research Nutrition Conference, Feeding and Veterinary Management of the Sport Horse. p. 34-41.

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