Laminitis in Grazing Equines

What is Laminitis?
Laminitis is the separation of the hoof from the coffin bone via the laminar layer. All equine breeds can be affected by this potentially crippling disease. The laminae are an important support structure in the hoof because they hold the coffin bone (also known as the third or distal phalanx and the pedal bone) within the hoof wall. Separation of the laminae, along with weight applied to the limb can cause severe lameness by displacing the coffin bone. An average horse weighs around 1100 pounds and most of the weight is distributed to the front legs. A horse undergoing an episode of laminitis could easily rotate the coffin bone because of the separated laminae. There are three classifications of laminitis: acute, subacute and chronic. Acute laminitis is described as being less than three days duration and no displacement of the coffin bone. It is very crippling and has a sudden onset. Subacute laminitis has mild symptoms and a duration time greater than three days but still no displacement of the coffin bone. Chronic laminitis is severe because symptoms are not initially noticed and the coffin bone becomes displaced or rotated during any time duration. Once the coffin bone is displaced or rotated and inflammation subsides, the disease is referred to as founder.

Symptoms and diagnoses
Laminitis becomes a concern for owners when a horse is unwilling to apply weight to a limb or limbs. Typically, horses shift their weight to the opposite end of the body to avoid applying pressure to affected hooves. Horses bear most of their weight on their forelimbs. Accordingly, laminitis is more commonly observed in the forelimbs. One symptom of laminitis is lack of movement. Equines with affected forelegs will stand with their front legs outstretched and hind legs brought underneath their body to support more weight and take pressure off of the affected legs. Typical stance for an equine with all four legs affected is for all four legs to be positioned away from the body. Figure 2: A-C compares a normal equine stance against two horses with laminitis. Other symptoms include a warm hoof wall, palpable bounding pulse in the distal legs, tenderness in the feet, muscular tremors, increased pulse and respiration and, in more severe cases, blood surrounding the coronary band. Formal diagnosis of laminitis includes checking for these symptoms, using short term nerve blocks and radiographing the hoof.

Figure 1: The contrast between a normal, laminitic and foundered hoof. As the coffin bone is separated from the hoof wall by the stretching of sensitive and insensitive laminae there is lack of support and the bone can rotate depending on the severity of the laminitis. A rotated coffin bone due to laminitis is called founder.

Founder is a serious and often fatal diagnosis in horses depending on the severity. A rotated or displaced coffin bone results in: bone remodeling; additional stress on joints, tendons and ligaments; abnormal hoof growth; and stretched white line (insensitive laminae). The downward rotation of the coffin bone can result in the tip of the coffin bone puncturing the sole of the hoof if the sensitive and insensitive laminae become separated. This occurrence is often called sinking because the laminar separation causes the coffin bone to drop. Sinking causes severe pain and high risk of infection. Due to intense, unmanageable pain and infection, horses with founder that has progressed to sinking of the coffin bone are often euthanized to prevent suffering.
Causes
High levels of non-structural carbohydrates (fructan, sugars and starches) in the diet are a cause of several illnesses in equines, including laminitis\(^1\). Grain-based diets have previously been considered the primary cause for laminitis, but laminitis is also seen in equines on forage-based diets. Approximately half of the laminitis cases in the US occur in horses maintained exclusively on pasture\(^4\). Sugars produced during photosynthesis aid

Other Causes:
- Off-shifting weight to another leg (common with injuries)
- Exercise on pavement (also known as Road Founder)
- Retained Placenta
- Infections
- Circulatory Issues

Pathophysiology of Laminitis
Excess dietary non-structural carbohydrates (sugars, starches, and fructose) can overwhelm the equine digestive system. Non-structural carbohydrates that are not absorbed in the small intestine are rapidly fermented in the hindgut (cecum and large intestine). The rapid fermentation causes an elevation of lactic acid that reduces the pH, causing hindgut bacteria (Streptococcus bovis) to release toxins (endotoxins or exotoxins) into the bloodstream and also dilate the blood vessels\(^5\). The toxins deposit in the hoof and ultimately alter the blood flow to the laminae in the hoof\(^6\). Although the complete mechanism is still unknown, there appears to be an increase of matrix metalloproteinase enzymes (MMPs; the enzymes that usually balance laminae as they often need repair)\(^6\). The increased MMPs in the hoof result in metabolic imbalance and they begin destroying laminae and capillaries\(^6\). The reduction of laminae and capillaries in the hoof along with increased blood flow due to the dilated blood vessel result in limited pathways for blood to reach the laminae. Therefore, more inflammation occurs and laminae continue to deteriorate.

Figure 2A: A normal stance for horses is for the front legs to be directly underneath the shoulders and the hind legs to be underneath the hips. K. Strawderman Photo Credit

Figure 2B: Stance of a horse with laminitis in both front legs. She is standing with her hind legs tucked under and her front legs outstretched, trying to relieve some pressure. T. Cubitt Photo Credit

Figure 2C: Horse with laminitis in all four legs. Notice the completely outstretched, almost “buckled” appearance while attempting to take as much pressure off of each leg as possible. C. Pawsey Photo Credit
in plant growth, but the unused portion of sugars are stored as carbohydrates\(^3\). High levels of non-structural carbohydrates, from grain or forage, are not easily digested by horses. Non-structural carbohydrates that bypass digestion in the small intestine are fermented by microbes in the hindgut, leading to laminitis symptoms.

**Treatment**

The severity of laminitis dictates the need for quick veterinary care. The displacement of the coffin bone can occur quickly and prognosis of recovery is often unknown\(^1\). Laminitis is a difficult disease for veterinarians to treat because of the unknown progression, which complicates prevention and treatment\(^2\). There are many ways veterinarians attempt to treat laminitis once it is diagnosed. However, treatments are inconsistent because they are based on limited knowledge of the disease process\(^2\). The use of ice water on affected feet has proven beneficial in recent studies\(^1\). A first line of defense if other options are limited is to apply cold water on the affected lower extremities using a water hose. Nonsteroidal anti-inflammatory drugs, shoe removal, applying padding to the hooves and surgery are several additional methods used to treat different stages of laminitis\(^1\). Standard diet protocol for a laminitic horse is the removal of grains and fresh forage to reduce sugar consumption and lessen the severity of laminitis. Soaking hay in water prior to feeding is the typical diet for horses during a laminitic episode.

**Prevention and Management**

There is no absolute method to prevent laminitis, but prevention may be accomplished by managing equine weight, grain supplementation and the pastures horses are allowed to graze. An overweight horse may have or develop health issues that can easily contribute to laminitis. Rapidly fermented grain should be avoided or minimized when possible. Diet should be adjusted gradually to slowly introduce grains. Grass generally has higher sugar content in the seeds and lower stems\(^3\). Pastures should be kept mowed so they cannot seed out\(^4\). Once a pasture has been grazed to its maximum potential (close to the base of the stem), horses should be rotated to another pasture to give the forage time to grow again\(^1\). However, since laminitis can also be triggered by other health conditions and grain consumption, pasture management is not a sure method to prevent laminitis.

After a horse is diagnosed with laminitis, management of dietary sugar intake is the only way to manage the disease. Eliminating grains and increasing fibrous feed is a necessary diet change. Equines who are more susceptible to laminitis (ponies and overweight horses) or those that have been diagnosed with laminitis should have increased pasture management. A grazing muzzle can be used to limit the amount of forage they are able to graze, especially since more sugars accumulate at the base of the grass stem\(^4\). Figure 5 depicts the rise of non-structural carbohydrates throughout the day, with the highest levels occurring around sunset. If the laminitis is not chronic, these horses can be turned out to pasture in the morning when sugar accumulation is lowest\(^3,4\).
Pasture turnout should be avoided any time there is a situation that will decrease growth, as sugars increase when growth doesn’t occur⁸. During and just after a drought are two scenarios in which pasture grazing should be avoided for susceptible horses⁶. The dry conditions limit forage growth and horses ingest an excess of non-structural carbohydrates stored near the stem. High light intensity or low temperatures can also increase the sugar content in forages⁴. Shaded pastures are less likely to have heavy sugar build up throughout the day³,⁴. Preventing forage from seeding by mowing and rotating pastures often should be practiced by owners caring for susceptible or laminitic horses.

Warm-season grasses such as bermudagrass and bahiagrass (Figure 6A; in the Southeast) should be the preferred forage planted for horses with laminitis, as they are not as high in non-structural carbohydrates as cool season grasses (Figure 6B; fescue, orchardgrass, oats, annual ryegrass;⁸). Cool season grasses are more durable in cooler weather, but they produce large amount of carbohydrates that are easily converted to simple sugars⁹. High sugar intake should be minimized for at-risk or laminitic equines.

Equines with chronic laminitis should be contained to an area with no fresh forage (dry lot) and fed hay that has been tested for low NSC content⁸. In some cases, hay can be soaked prior to feeding to rinse sugars⁹. The hay should be drained of the “sugary water” and then fed. Feed with more slowly fermentable carbohydrates (beet pulp or rice bran) may also be fed to horses with chronic laminitis.

**Conclusion**

Laminitis is a painful disease in equines that can have fatal effects if not treated and managed carefully. Inflammation causes the coffin bone to separate from the hoof wall via the laminar layers. This can occur if a horse ingests excess non-structural carbohydrates resulting in increased fermentation in the hindgut, reducing the pH and thus altering the microbial populations. Although there is no single method to prevent laminitis, appropriate planting of forage for the region and season and practicing good pasture management are good places to start. As soon as symptoms of laminitis are present, a veterinarian should be contacted and treatment should begin immediately. As limited information about the disease process is known, different veterinarians may have varying treatment protocols. Ice water soaking of...
affected legs and NSAIDS are usually used. Although good pasture management such as rotating pastures that have been fully grazed and mowing before seed heads emerge should be common practice with every equine owner, any equine that is diagnosed with laminitis should have increased pasture management. Some may be able to stay on pasture with the use of a grazing muzzle. Others should have selective turn-out to pasture based on time of day, depending on weather, environmental changes and forage type. In severe cases, equines should be confined to a dry lot and fed a high fiber, low soluble carbohydrate hay. If necessary, hay may be soaked and drained prior to feeding to minimize sugar content. Grain utilization should be minimized in susceptible horses, and only those of lower NSC content fed to horses suffering from this metabolic disease.

References