Laminitis: Compromised Suspension and Support!

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I don't know anyone that would disagree that this summer has had an odd weather pattern. Fluctuations of cool/wet weather with short spikes of heat and humidity have contributed to an increase in the number of laminitis cases then we would typically see during this time of year by driving up the sugars in the pasture.

Laminitis accounts for approximately 15% of lameness issues in horses…. But what is it? Simply, laminitis is an inflammation of the laminae within the foot of a horse (think of walking on a finger with a blood blister). Laminae can be found covering the entire surface between the hoof capsule and the coffin bone. It forms a tight bond that is able to withstand up to 4500lb on a single foot during athletic competition. The laminae are designed to be able to be an equal and opposite force to the pull of the deep digital flexor tendon (DDFT). The DDFT runs down the back of a horses' leg and attaches to the bottom (solar) surface of the coffin bone. Like any big elastic, it wants to "recoil". Without the opposing force of the laminae, the DDFT would be able to pull the tip of the coffin bone downwards, out of the hoof capsule. In laminitis cases where the laminae are so damaged that they can no longer oppose the DDFT, the coffin bone rotates and sometimes sinks, resulting in "Founder".

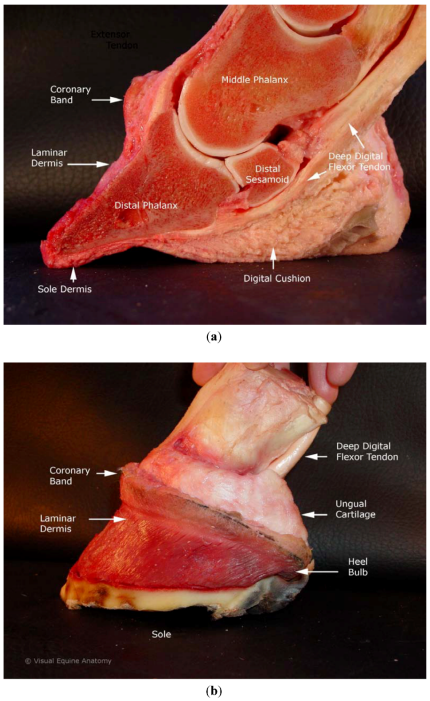


Fig. 1 A normal foot with anatomical labels (Lancaster, Animals 2012)

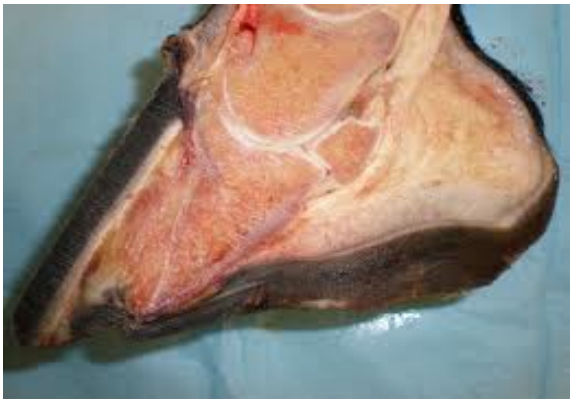


Fig. 2 A chronically laminitic foot with rotation of the coffin bone.

Fig. 2b The bony column of a chronic laminitis pony. The tip of the coffin bone has been remodelled and blunted over time. This gives it the "ski tip" appearance.

What causes laminitis?

The most common cause we see is obesity and insulin resistance. The characteristic image is of an insulin resistance pony that has a thick cresty neck and large fat pads behind the shoulders and on the rump. While this holds true, a more subtle body condition type has emerged within our sport horses where they develop fat pads over their rib cage. These horses often do not have the top line that their level of work suggests they should have and can be "flat backed" under the saddle.

How is it possible that insulin resistance and elevated blood glucose causes laminitis? There are a few things happening that make these horses more likely to have issues…. (1) When the fat storage cells reach maximum capacity they get stressed and release a substance that is inflammatory to the body. Anything that creates a chronic underlying inflammation within the body sets that organ up to be "at risk" for major disease.
(2) Under normal circumstances, insulin would tell the cells to suck up the glucose from the blood for energy and their own nutrition. Because the cells aren't listening to the insulin's signal to pull glucose inside the cell, they end up starving and unable to function properly.
(3) Insulin causes blood vessels in the limbs to contract (get smaller). This reduces the blood flow to the foot and further reduces the supply of fresh oxygen and nutrition.

Infection or sepsis is another major cause of acute laminitis. As I write this article, cases of Potomac Horse Fever are popping up around Ontario. This disease causes a significant colitis/diarrhea. As we discussed in last month's article - when the large colon is inflamed, it becomes leaky and allows bacteria and toxins to enter the body's blood stream. In addition to the systemic (whole body) inflammation, these toxins also attack the laminae and cause laminitis which can be severe and life threatening. Any case of significant diarrhea/colitis puts the horse at risk to develop laminitis. During foaling season you will notice that two words that get your veterinarian out ASAP is "Retained Placenta" in a broodmare. The reason for the panic is that the retained placenta begins to effectively decompose within the uterus and creates a

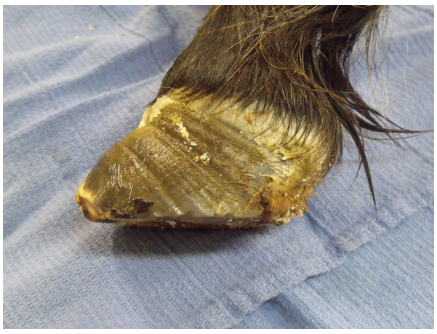
significant infection that easily reaches the blood stream. Similar to the colitis scenario, once the bacteria and toxins are in the blood they create systemic inflammation and gain access to the laminae in the feet.

Less common causes of laminitis include mechanical or “Support limb” laminitis. Remember that the blood supply to the foot is dependent upon the horse walking around. They were never designed to stand still in stalls. In horses with a prolonged severe lameness on one foot are naturally going to put more weight on the opposite foot. The constant pressure on the limb reduces blood flow to the laminae and starves the tissues of oxygen. The result is a build up of “cell garbage” (like lactate) that creates inflammation and therefore, laminitis. We all remember the magnificent race horse “Barbaro”. In the end it was his “good foot” that became compromised from bearing the load while his fracture was healing. The resulting support limb laminitis created a situation where euthanasia was the best option for him as he literally didn’t have a good leg to stand on anymore.

Acute versus Chronic Laminits

Acute laminitis often results from some form of systemic insult (infection, grain overload, pasture associated) that damage the laminae. Horses’ are often found in the typical “saw horse” stance where they have rocked backwards onto their hind limbs to relieve pressure on the toes of their front feet. They are often reluctant to walk and very painful when asked to turn in a tight circle. The feet may feel hot to the touch and the digital pulses are often easily palpated or “bounding”. Control of inflammation and supporting the hoof are the keys to reducing the exposure of the laminae to the damaging substances and controlling the severity of the laminitis attack.

In contrast, chronic laminitis horses have a mechanical issue which must be managed in order to prevent an acute attack to occurring and to maintain soundness. Chemical treatment has a secondary role in chronic laminitis cases. Support of the foot and ensuring a balanced trim to reduce mechanical stress on an already compromised foot.



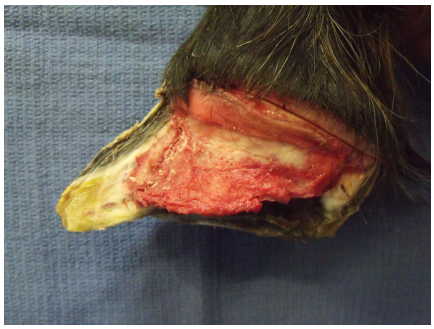


Fig. 3a + 3b: A chronically laminitic pony’s foot. Notice how distorted the hoof capsule is in 3a and the moth eaten (demineralized) edge of the coffin bone in 3b.

Treatment of acute laminitis.

As mentioned earlier, in acute laminitis cases, the immediate goal is to control and reduce inflammation systemically and in the feet. This can be accomplished by Non-steroidal inflammatory drugs (NSAIDs) like phenylbutazone or flunixin (Banamine). Personally, because so many of these cases are due to some sort of systemic inflammation/bacterial toxin exposure, I prefer using Banamine in my cases because at a 1/2 dose it has an “anti endotoxin” effect. In addition to anti-inflammatory drugs, the judicious use of ice therapy during the first 72 hours is of great benefit. Using 5L IV fluid bags filled with ice and water (secured using Elastoplast) is a more straight forward way of icing feet safely. The key to is maintain submersion in ice water continuously for 48-72 hours.
Ensuring that the foot is balanced and removing any excess toe length helps to reduce additional mechanical forces on the inflamed structures. Once the foot is balanced, solar support through the use of therapeutic boots with impression material packed into the back 1/2 of the hoof (sulca and frog to the heel bulbs) is the easiest way to take the pressure off the toe area and improve comfort in the patient. Alternative options include using heart bar shoes or wooden clogs with impression material packed in the back half of the foot. The most important point to make is that each case is individual and must be managed as its own entity. What works for one case, may not work for the next.
In cases where pasture associated laminitis or insulin resistance is suspected as the cause, a drastic reduction of calories is also involved in the initial management.

Management of chronic laminitis.

Maintaining the hoof balance through frequent trims (every 4-5 weeks) +/- mechanical support through boots or shoes is the foundation of managing these cases. Nutritional management through the use of low non-structural carbohydrate diets (NSCs/sugar) and supplementation with omega fatty acids is beneficial. I think we underestimate the impact of our hay on laminitis cases. Testing your hay to determine exactly what you are feeding is key. Many cases that have been well managed and suddenly find themselves in an active laminitis attack when “nothing has changed” can be traced back to starting a new hay source which had sugar levels far greater than the previous source. Never underestimate the nutritional content of your hay.

Controlling and managing any underlying diseases will also be critical. For example, insulin resistant horses often do best in dirt paddocks or turned out in grazing muzzles to reduce their intake. Exercise also plays a key role in improving insulin sensitivity as much as possible. Recent studies from the U.K. established that 30-40 minutes of moderate exercise (sweaty and out of breath) 3-5 times per week was the most beneficial.

Managing acute and chronic laminitis cases is most successful when the owner, farrier and veterinarian work as a team. Radiographs, blood work and nutrition consults/modification are the most common aspects of my role as a veterinarian, but without a knowledgeable farrier to balance and monitor the foot while it stabilizes, our chances of success diminish. Equally, the owner must be on board with the management required. The temptation to feed treats and the feeling of being ‘cruel’ by using dry paddocks and grazing muzzles is something all owners struggle with in the beginning. If we can focus on the ‘long game’ and the result of stabilizing the hoof and maintaining soundness in our patients then the end often justifies the means!