

Chapter 9

Laminitis Medical Therapy

Laminitis remedies

From the outset it must be understood that a therapeutic regime, using biological or chemotherapeutic agents able to arrest or block the triggering of laminitis, does not exist. On the other hand, there is a plethora of remedies, used empirically, that symptomatically help the horse after it has acquired laminitis. It is more the extent and severity of the lamellar pathology that influences the outcome for the horse, not the treatment regimen itself. An effective laminitis preventive may emerge when the mechanism behind the disintegration of the anatomy of the hoof wall lamellae is fully understood. Our discovery that a class of enzymes appears to be involved in the lamellar failure of laminitis has led us to commence trials of proteinase inhibitor therapy, specifically targeted at hoof wall matrix metalloproteinases.

Since laminitis usually develops as a sequela to a disease process in a body compartment other than the foot, it is of paramount importance that the primary disease is treated urgently and effectively. If the duration and severity of the primary disease can be reduced by intensive therapy, there is a strong chance that the severity of lamellar pathology may also be reduced, thus improving the prognosis for the horse. Nevertheless, severe laminitis is sometimes the outcome despite the best of current therapy.

Endotoxaemia therapy

Horses diagnosed with endotoxaemia during enteritis, colitis, strangulating colic, pleuropneumonia, septic metritis (retained placenta) and grain overload are at high risk of developing laminitis and ideally, medical therapy and mechanical support for the distal phalanx should be initiated before the clinical signs of foot pain appear. Addressing laminitis as soon as it appears in a sick horse should always be regarded as an emergency procedure. Even then it may be too late.

Anti-endotoxin hyperimmune serum (**Polymune, Veterinary Dynamics Australia,**) should be included in the intravenous fluid therapy for horses with, or at risk, of developing endotoxaemia.

Apart from vigorously treating the primary inciting disease, attention should be paid to reducing inflammation and foot pain by administering nonsteroidal anti-inflammatory drugs (NSAIDs). Flunixin meglumine (**Finadyne,**) administered intravenously at 0.25 mg/kg TID or 1.1 mg/kg BID has a proven ant-endotoxin effect by reducing prostaglandin production via cyclooxygenase inhibition and is valuable. Horses receiving flunixin meglumine and subsequently dosed with endotoxin had significantly lower blood prostaglandin and lactate concentrations and reduced clinical signs than control horses. However, the effectiveness of flunixin or any NSAID as an anti laminitis agent has never been tested. Phenylbutazone (4.4mg/kg IV or orally every 12 hours) appears to be a potent NSAID for the control of foot pain and is popular with most clinicians. Phenylbutazone and flunixin meglumine at the lower dose rate can be used concurrently; the former to control severe foot pain and the latter to control the effects of endotoxaemia. Intravenous ketoprofen (2.2mg/kg BID) can be used interchangeably with flunixin. Horses with acute laminitis usually require NSAID therapy for at least 2 weeks and, because of its low cost, phenylbutazone at the 2.2mg/kg dose rate is the best choice for maintenance therapy.

However NSAIDs have been administered to horses during experimental induction of laminitis without altering the outcome - laminitis still occurred. Alarmingly, laminitis *in vitro* studies indicate that MMP activation is slightly potentiated when NSAIDs are present in the culture system. This is borne out in practice. When the laminitis process is triggered, there is virtually nothing, by way of drug therapy, that will stop its relentless progress. The administration of phenylbutazone, during the developmental/acute stages, will abolish foot pain and create a more comfortable-looking horse, but the disease continues unabated. This creates an ethical dilemma; balancing the need to alleviate pain and suffering against the realisation that most of what is administered is only palliative. When NSAIDs are in use, the patient should be confined to a stall with deep bedding. Exercise, while under the influence of painkillers, such as phenylbutazone, is contraindicated.

Cryotherapy

The results of experiments at the AELRU, continuously evaluating foot temperature (and by implication foot circulation), as horses developed laminitis, showed that

vasoconstriction during the developmental stage of laminitis may have had a protective effect (**Figure 4.5**). The induction of digital vasoconstriction may be a useful preventive strategy in the developmental phase of laminitis. Limited anecdotal evidence from practicing veterinarians suggests that cryotherapy may halt the development of the disease. Cryotherapy requires packing the feet in a slurry of crushed ice or soaking the feet in circulating cold water continuously for 24 hours or even longer if the period of septic shock, pyrexia and digital vasodilation persists. Cold therapy decreases perfusion of the soft tissues of a horse's foot. It is safe, well tolerated and economical. Unlike humans, horses do not find cold therapy noxious. We have applied slurry of ice and water to the hoof, pastern, fetlock and lower cannon of normal horses continuously for 2 days, with no immediate or long-term ill effect. However further experimentation is required to prove the effectiveness of cryotherapy for halting laminitis onset.

Vasodilator therapy

The use of vasodilatory therapy and hot water footbaths during the developmental phase of laminitis appears to be contraindicated. Drugs with vasodilator action such as isoxuprine hydrochloride, ace promazine and glyceryl trinitrate (applied as a patch to the pastern) may be beneficial after lamellar damage has occurred, when healing is required, but should be administered with caution during the developmental phase. Exercise of an intensity which raises core temperature and local anesthetic blockade of the palmar or plantar nerves both result in hoof wall heating (and by implication vasodilation) and are contraindicated during the developmental stage. In addition, horses given local anaesthetic to block foot pain, and then encouraged to walk, will almost certainly sustain greater lamellar damage than a rested, confined horse. Forced exercise to any horse with acute laminitis is strongly contraindicated.

Free radical scavengers

Dimethylsulfoxide (DMSO) may be given intravenously for its free radical scavenging and anti-inflammatory effects. DMSO (90% solution) mixed with polyionic solutions and 5% dextrose is best administered slowly at about 8 litres per hour. The concentration of DMSO must remain below 20% to avoid the risk of intravascular haemolysis. However, despite the potential of DMSO, its promise as an effective laminitis therapy has not been fulfilled. There is no evidence that ischaemia, reperfusion injury and the generation of free radicals are involved in the pathogenesis of most cases of laminitis.

A recommended treatment strategy

The list of pharmaceuticals that have been administered to horses with laminitis is long and, apart from the NSAIDs, none have achieved particular prominence. The author's recommended treatment strategy is to aggressively treat the primary disease entity, systematically addressing the problems the horse may have as proactively as possible. Fluid and electrolytes, antibiotics and NSAIDs are administered as required. Horses with septic metritis/retained placenta also require uterine lavage.

The administration of 4 litres of mineral oil four times /day may be beneficial in the case of laminitis developing from grain overload. It has a laxative effect and its presence in the large intestine is said to block the absorption of toxins. Similarly, activated charcoal is an effective adsorbent of a range of toxins and may be useful in cases of grain overload if administered promptly. In Australia, doses of 1-5 g/kg/day have been used to treat plant toxicoses in large animals. The higher dose is indicated if a large quantity of grain has been consumed. However activated charcoal has not been tested against alimentary laminitis, so its true effectiveness is unknown. The application of cold therapy to the front feet, strict confinement to a stall with a deep bedding of sand or shavings and mechanical support for the distal phalanx are also recommended (**Figure 9.1**).

Key Points

- Vigorous treatment of the primary inciting disease is of paramount importance.
- The diagnosis of endotoxaemia is associated with a high risk of developing laminitis and requires the initiation of medical therapy and mechanical support for the distal phalanx before the appearance of clinical signs of hoof pain.
- Inflammation and foot pain can be reduced with NSAIDs, however their effect is only palliative and will not stop the development of laminitis.
- Cryotherapy may prove to have a protective effect in horses developing laminitis, while vasodilator therapy and forced exercise are contraindicated. The administration of mineral oil or activated charcoal may be beneficial in cases of laminitis developing after ingestion of excess grain.



▲FIGURE 9.1 Cryotherapy for a horse with acute, severe colitis and bloody diarrhoea. The boots are made of synthetic rubber (Bigfoot Iceboots, see appendix 2). They contain a slurry of water and ice cubes - as the ice melts more is added so that there is always ice in the water. The horse was treated with intravenous fluids, equine gamma globulin, flunixin meglumine (Finadyne), paraffin oil and activated charcoal. Antibacterial therapy was added to the regime because of persistent fever. An expected outcome of severe colitis in horses is laminitis but this did NOT occur in this case; the horse made a complete recovery. Photo courtesy Eagle Farm Equine Veterinary Hospital, Hendra, Brisbane, Queensland, Australia

