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Equine Laminitis

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Laminitis is defined as the inflammation of the laminae of the foot. This disease, commonly known as founder, has been known for centuries, however the pathogenesis is still under discussion. Both cardiovascular as well as metabolic changes are known to be involved in the disease.[1] Capillary perfusion to the laminar corium is decreased in many ways. Histamine released from hypoxic mast cells causes capillary dilation. Bacterial endotoxin released during both alimentary and post-parturient laminitis causes circulatory changes of constriction of arterioles and venules resulting in capillary congestion and edema. Resultant arteriovenous shunts compounded by RBC's sludging and platelet emboli lead to diversion of blood flow from the laminar corium.[2] This deprived blood flow leads to the synthesis of structurally incompetent keratin and to a loss of the mechanical integrity of the disulfide bonds between the sensitive and insensitive laminae.[3]

Since the step-limiting substrates in keratin production are methionine, cysteine, and cystine,[1] a shortage of these amino acids has also been implicated. These detailed sequences of events within the hoof and especially within the laminae are still obscure. In view of the diverse conditions associated with laminitis, these changes within the lamina may not always even be the same.

Causes

1. Grain Founder.—Grain founder is caused by the consumption of quantities of grain greater than those to which the horse is accustomed. All types of grain and animal feeds can cause this type of founder although oats and bran are probably the least offensive. The consumption leads to a gastroenteritis which, according to different authors, initiates histamine release, leads to a bacterial enterotoxemia and/or even an immune response to the protein of the bacteria itself. This type of founder is believed by some to depend upon the presence of certain bacteria and can be transmitted to another horse with the transfer of ingesta.[4]

2. Water Founder.—Water founder is caused by the consumption of large amounts of cold water while the horse is still overheated. The exact mechanism of this phenomenon is still not fully understood.

3. Road Founder.—Road founder is common in unconditioned horses after strenuous work on a hard surface.

4. Grass Founder.—Grass founder is usually seen in overweight horses that consume more lush forage than they are accustomed to. Clover and alfalfa pastures are most commonly at fault. However, lush grass pastures have also been known to cause this type of founder. Wintering overweight horses on legume hays has occasionally also been incriminated.

5. Postparturient Founder.—This type of founder is a result of a retained placenta or even a uterine infection without retention of any of the fetal membranes. Other systemic infections such as pneumonia or viral respiratory diseases have also been thought to cause this severe type of founder.

6. Miscellaneous Causes.—Laminitis has been known to follow such things as worming, anaphylactoid reactions to some drugs, and large doses of phenylbutuzone

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The vaccine is produced from rabies virus grown in brain tissue taken from suckling mice. Virus particles are killed during processing, so that they cannot revert to an active, dangerous state. Thus, the vaccine is quite safe for animals and those who handle the vaccine or may be accidentally exposed to it. [1, 2]

By growing a large number of vaccine virus particles in a small number of brain cells, the manufacturer can produce a vaccine with high strength—or potency—and a low proportion of remaining animal tissue. Large numbers of animal cells could cause irritation and inflammation at the injection site. [1, 2]

Existing rabies vaccines are made either from killed virus or live virus that is modified, or altered to keep it from causing disease. Killed virus vaccines are generally safe, but have presented problems with limited potency and undesirable side effects. Modified live virus vaccines are quite potent, but present a small, but ever-present safety risk. [1]

Puppies and kittens should be vaccinated at three months of age—or revaccinated then if they were vaccinated earlier. Vaccination should be repeated yearly.

1. "Improved Rabies Vaccine Licensed for Dogs and Cats," *U.S. Dept. of Agr.*, News Release USDA 1098-73.
2. "Improved Rabies Vaccine Licensed for Dogs and Cats," News Section, *Jrnl. of the AVMA,* 162:12:1021.

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or steroids. The exact mechanisms of these and other types of founder are not understood completely.[5]

Clinical Signs—Acute Stage

If all four feet are affected, the horse is reluctant to stand. When standing the horse will have his rear feet well under him anteriorly and his front feet carried posteriorly to have a very narrow based stance. Most commonly, only the two front feet are affected. In this case, the hind feet are carried well up under the animal and the front feet are placed forward with the horse carrying the weight upon the heels of the feet and showing a great reluctance to move. The sole, wall, and coronary band will be very warm and an increased digital pulse can be palpated on the digital vessels over the fetlock joint. Many horses show anxiety, trembling, injected mucous membranes, sweating, increased respiratory and heart rates, and may have an increased temperature depending upon the etiology. The walk of a foundered horse is said to be "peggy" as he will shuffle his feet and often stumble because of the difficulty to lift one leg as he throws more weight on the other affected foot. Hoof testers reveal a uniform tenderness over the entire sole. Blood eosinophil counts and histamine levels may raise but are often within the normal ranges. Death resulting from acute laminitis is very uncommon however the whole hoof may slough as a result of a separation of the sensitive and insenitive laminae.

Chronic Stage.—In chronic laminitis, the pull of the deep flexor tendon causes the third phalanx (P_3) to rotate in such a way that the anterior face of P_3 is pulled away from the anterior insensitive laminae of the hoof wall. This can be seen radiographically.[4] The rotation often is to such an extent as to cause the anterior point of P_3 to be pushed through the sole when the horse bears weight on the foot.

Horses affected with chronic laminitis have a tendency to walk on their heels. Because of this and because the inflammation causes the hoof to grow faster than normal, the toe of the hoof will become long and with time will curl up to form the "sleigh foot" appearance. Heavy ring formations will become evident on the hoof wall as a result of uneven horn growth because of the inflammation at the coronary band. These rings are usually present for the life of the horse.

Separation of the sensitive and insensitive laminae often predispose the animal to a condition known as 'Seedy Toe', an infection penetrating the white line and spreading up the sensitive laminae. Examination of the foot will reveal the sole to be almost flat rather than concaved as it is normally. The white line will become noticeably thicker than normal. The sole will be flaky but yet it will be easy to induce hemorrhage because of the increased vascularity. This tendency toward easy hemorrhage will remain for many months.

Diagnosis.—Diagnosis of laminitis is based upon the clinical signs mentioned above. The typical gate and attitude of the animal, the increased heat and tenderness of the hoof wall, and the almost bounding digital pulse should lead to a relatively easy diagnosis. However, a volar nerve block may have to be performed occasionally to rule out other source of the lameness. The radiographic evidence of the rotation of P_3 can also be used for the diagnosis of chronic laminitis.

Treatment

1. Acute Laminitis—Regardless of the specific cause of the acute laminitis, the therapy for each case should be devised using these basic principles: remove the underlying cause, relieve the pain, increase the capillary flow to the laminae, improve keratin synthesis, and prevent secondary infection.[1]

Since the exact mechanisms of drug-reaction laminitis and other miscellaneous causes are not understood, it has been proposed that all cases of acute laminitis (except postparturient laminitis) be treated as though their underlying cause is in the gastrointestinal tract. For this reason all horses with acute laminitis (except postparturient laminitis) should be purged

with mineral oil every 6 hours until all of the grain, etc. is removed from the gastrointestinal tract. If dehydration occurs, this should be treated with intravenous fluids. Horses affected with postparturient laminitis should be treated for metritis. Uterine irrigations, uterine infusion with antibiotics, parenteral antibiotics and perhaps estrogens have all been recommended.

Phenylbutazone is the drug of choice for the relief of the pain involved. Occasionally a posterior digital nerve block may have to be performed to ease the pain enough to enable the horse to move about. The ambulation of the patient is important to improve the circulation to the foot. However, the patient's movements should not be excessive and limited to soft surfaces so as to prevent further rotation of P_3. Antihistamines at recommended doses are used to increase blood flow to the laminae because histamines cause capillary pooling by constriction of arterioles and venuoles and dilation of capillaries. Corticosteroids may increase capillary flow by vasodilatation, however, their inhibition of local defense mechanisms and keratin synthesis make at least their repeated use contraindicated. However, certain authors have proposed the use of intra-arterials teroid injections into the digital artery over the abaxial surface of the sesamoid bone.[6] A tourniquet is applied immediately following the injection leading to supposedly better steroid action at the area involved. The feet should be soaked in warm water, possibly containing epsom salts and disinfectant, to improve circulation. The palmar nerve block, if performed, will also decrease peripheral resistance, via dilation of arterioles and venuoles.

To enhance keratinization, methionine, cysteine, and cystine, the step-limiting substrates of keratin, may have to be added to the ration. Ten gm. daily for three days followed by five gm. daily for ten days has been proposed as an adequate amount of methionine for a 1,000 lb. horse. With normal liver function methionine is converted to cysteine and cystine.

To prevent secondary infections of the corium, body temperature should be monitored daily until the patient is recovering well. This is important since secondary sepsis of the laminae is probably the most common cause of failure of the therapy. Immediate treatment of an infection is indicated. Infection is usually treated with systemic antibiotics, opening of the wound to enhance drainage, tetanus antitoxin, and dressing the foot with tar and oakum.

Autogenous blood therapy has long been used although its mode of action is not known. This is done by withdrawing 50–200 cc of blood from the jugular vein and administering this intramuscularly in several divided doses. This mode of treatment was derived from the treatment of shingles in man.

The treatment of acute equine laminitis is largely empierical and requires more study and improvement. However, one fact is for certain: acute laminitis should always be handled as an emergency. This principle is based on the many experimentally induced models that have been studied.[7]

2. Chronic Laminitis.—Chronic laminitis is very common in overweight horses and especially ponies. These obese horses often show high cholesterol and serum glutamic oxaloacetic transaminase (SGOT) levels. Horses with SGOT levels above 400 RF units are often given dextrose, protein hydrolysate, and vitamin B complex intravenously, ACTH intramuscularly and placed on a reducing diet when necessary. The recommended dosages of these are: 50 ml. of 50% dextrose per 100 lbs., 500 ml. of 5% protein hydrolysate, 10 ml. B complex, 400 units ACTH on day one, and 200 units on days 2, 3, 4 & 5. A high protein diet with approximately 50% of the previous caloric intake has been recommended.[8,9]

All horses affected with chronic laminitis should also have the affected feet correctively trimmed so as to bring P_3 back to its normal angle. When this is accomplished the base of P_3 should be as close to parallel to the sole of the foot as possible. This can be accomplished in many ways. The severity of the rotation as determined radiographically should be the deciding factor as to which procedure

is chosen.

If the rotation is not very severe, simply lowering the heel and stubbing off the excess toe with hoof nippers and a rasp may be sufficient. The hoof should then be shod with perhaps a bolt-on steel plated shoe to protect the sole. The area between the sole and plate is packed with pine tar and oakum to help prevent infection. This dressing is changed weekly and careful attention should be paid to developing absceses.

When the distal end of P_3 has rotated more than one cm. (as suggested by one author)[8] radical acrylic repair is recommended. This is accomplished by again trimming the heel as much as possible and undercutting as much of the abnormally shaped toe wall as is needed. This may be a small gap or the entire toe wall as far as up to one cm. from the coronet. The toe wall is then replaced with an acrylic, plastic or fiberglass mixture and reshaped to form a more normal looking hoof wall.[10] The exact procedure has been described in detail numerous times in literature and by manufacturers. This new hoof wall material protects the laminae, provides a more normal angle of resistance for the hoof wall to grow against and can allow more radical improvement to the angle of P_3. Sometimes some of this plastic, mixed to a softer texture, is placed under the sole to protect it. The foot is then shod using a shoe with high toe and quarter clips to give good support without using many nails. All of these different types of shoes should be reset every four to six weeks. Radiographs should be taken periodically to determine the progress being made.

In cases not subjected to correction of pedal rotation, vertical grooves may be cut into the hoof wall to allow more expansion of the wall which eases congestion and pain.[11] Two grooves are cut ¾ inch on either side of the median line of the toe wall almost to the depth of the laminae. These grooves can be cut with a hoof knife or bistoury although this is very time consuming and arduous. Several authors have suggested the use of small, high-speed hand drills which can be fitted with a variety of heads and make the job much easier and safer. After this is accomplished the heels are lowered and the toe is rolled to complete the job.

The principles of the treatment of chronic laminitis are to return P_3 to its normal position, prevent secondary infection and to prevent the recurrence of the disease. This can be accomplished in many ways, but the most logical and simple approach should be used. The owner should also be advised that horses recovered from laminitis are thought to be more susceptible to its recurrence.

Discussion

Often the exact etiology of laminitis is unknown and corrective hoof trimming is all that can be done. With time many of these horses can be returned to reasonable soundness but few horses regain complete soundness.

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