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LAMINITIS

by Don Lucas*

DEFINITION

Laminitis is an inflammation of the foot. It may be caused by either infectious or noninfectious agents and is characterized by passive congestion of the laminae with blood. Severe pain results from the inflammation caused by pressure on the sensitive laminae.

ETIOLOGY AND PATHOGENESIS

Laminitis is caused by numerous etiological factors, not all of which are fully understood. Causes commonly recognized include:

1. Grain Founder.—Grain founder is caused by ingestion of greater quantities of grain than can be tolerated by the horse. The amount varies, since a certain degree of tolerance develops in those horses accustomed to eating large quantities of grain. This type of founder is associated with gastroenteritis.

2. Water Founder.—Ingestion of large amounts of cold water by an overheated horse is considered to be a cause of laminitis. The phenomenon may be due to gastroenteritis or possibly to histamine formation.

3. Road Founder.—This is the result of concussion to the feet from hard work or fast work on a hard surface. Unconditioned animals are especially subject to this type of laminitis, as are those horses having thin walls and soles.


17. Nakamura, Keizo. Allergy and Anaphylaxis. Dept. of Bacteriology, Nippon Medical School, Tokyo, Japan. 1954.


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4. Postparturient Laminitis.—A mare may develop this type of laminitis shortly after foaling as a result of retaining a portion of the fetal membranes or an uterine infection without retention of fetal membranes.

5. Superpurgation.—Superpurgation, described as a cause of laminitis, follows administration of purgative drugs; the exact cause is unknown.

6. Grass Founder.—Grass founder is common among horses which are grazed on summer pastures. Clover and alfalfa pastures commonly cause difficulties because of ingestion of large amounts of succulent material. Grass founder usually is thought to refer to animals developing laminitis on grass, not legumes.

7. Miscellaneous Causes.—Laminitis has been recorded in mares that did not come into heat or were in heat for an indefinite period. Hormonal influences may be an etiological factor.

Although laminitis is probably one of the most characteristic equine diseases, it has long evaded a rational explanation. Since antiquity its association with the consumption of large amounts of grain has been recognized; indeed, it was known to the Romans as "hordeatio," from "hordeum," barley, and to the Greeks as "krithiasis," from "krithe," a barley corn. The disease is solely confined to the Ungulates and almost solely to the Equidae, although cattle are affected.

Eric Akerblom, a Swedish veterinarian, has investigated fodder founder due to eating rye and has taken no cognisance of puerperal founder following influenza, overexertion and others of acquired nature. It would not be surprising if the same fundamental cause was responsible in all cases.

Akerblom started from the assumption that the three most likely explanations were (a) the presence of bacteria or molds in the fodder, (b) a preformed toxic substance in the fodder, and (c) toxic products of digestion or metabolism derived from fodder after its consumption. The first was easily and quickly disposed of. Various molds found on rye were grown in cooked rye and fed in large amounts to horses without effect. Next, large amounts of uncooked rye were extracted with various solvents. In no case did the extractives produce ill effects, whereas the residual rye was still capable of producing founder in susceptible horses. It was therefore concluded that the toxic principle was not of alkaloidal or glucosidal nature but was probably a substance produced during digestion.

Akerblom has shown this substance to be histamine or β-imidazolyethylamine. Injected intravenously in small repeated doses it produced many of the local systemic symptoms of founder. It can be demonstrated in the intestinal contents of horses suffering from founder and is absent from normal horses.

Histamine is derived by certain bacteria from the essential amino-acid histidine by a process called decarboxylation, during which carbon dioxide is split off. Organisms of the E. coli type, capable of effecting this change, have been found in the intestinal contents of about 50% of normal horses examined. Normally, histidine, like other amino-acids, is released in the intestine from proteins by peptic and pancreatic digestion and is thereupon absorbed in the upper segments of the bowel and used in metabolism. It is probably only when digestion is delayed, as when large quantities of grain are consumed, that large amounts of histidine reach the lower segments of the bowel. If various conditions not yet clearly understood are suitable, for example, if the hydrogen-ion concentration is favorable, if there is excess carbohydrate and if bacteria capable of decarboxylating histidine are present, then the harmless and essential food stuff histidine is converted into the highly poisonous derivative, histamine, which is absorbed in continuous small amounts via the portal circulation and produces the systemic and peripheral symptoms known as founder.

Akerblom concludes that founder is essentially a "histaminosis" and that in horses the capillary bed of the stratum vasculosum in the hoof is a predilection site for its effect. It is well known that histamine has a damaging effect on capillaries and thereby produces edema. Anatomical considerations show that edema between the horny lamellae and the corium, if severe
enough, will produce a separation of the wall that will be aggravated by the muscular pull of the flexor tendons and muscles on the third phalanx. Therefore, Akerblom, along with many others, considers typical founder to be due to the effect of histamine. (1)

Obel of Sweden found degenerative changes in the cornifying intermediate zone—the “keratinogenic layers”—of epithelium and believed them to be of primary importance of laminitis. (It will be recalled that the inner, sensitive laminae consist of the connective-tissue corium plus a thin covering formed by the germin al and uncornified layer of epithelium; the hoof and its lining of insensitive laminae constitute the cornified layer of epithelium, greatly over-developed as compared to ordinary skin.) The changes described by Obel included loss of the eleidin granules and of many acidophilic “onychogenic” fibrils. In severe cases they were complicated by early necrosis. He believed these degenerative processes to be responsible for the separation of the laminae. (8) It is difficult to believe that the initial pathogenesis of a disease which strikes with such fulminating fury lies primarily in degenerative processes.

Jean Chavance of Saint-Florentin, France states that the diversity of etiological factors in view of the constancy of symptomatology and lesions suggests the existence of a single intermediary factor. Histamine may be this single factor. In laminitis there is a disturbance of equilibrium of the autonomic nervous system beginning with a vagotonic crisis and due to heterogeneous albumins. Histamine activates the secretion of adrenalin, the hypertensive hormone; by vasoconstriction of the arteries, arterioles, veins and venu les. Histamine has vaso-dilatory action which affects little besides the capillaries. Pain is a very important hypertensive factor, its principle effect being a vasoconstriction of small arteries and arterioles caused especially by medullary and upper reflexes. The keratogenous tissue, congested, and on that account compressed between the bone and the rigid horny casing, is certainly the seat of an intense pain.

The hormonal or reflex vasoconstriction is a consequence both unfavorable if generalized and hypertensive, and favorable locally by constriction of the arterioles, which saves the paralyzed capillaries from the rush of blood. The position of the feet aggravate the congestion through gravity. (4)

A review of the literature indicates the histamine theory now stands as the classical single factor in the description of laminitis.

SYMPTOMS

The general symptoms usually start with an interference of locomotion. As the laminitis develops the pulse becomes full, hard and strong; the temperature soon rises, reaching 105° to 106°F. The respiration becomes rapid, the nostrils are widely dilated and the mucous membranes are highly congested. The facial expression is indicative of acute suffering. The body is more or less bathed in sweat. Diarrhea may or may not be present. The urine is scanty and highly colored with an increase in the specific gravity, owing to the water being eliminated by the skin instead of the kidneys. The appetite is impaired and sometimes complete anorexia is present. The affected feet are hot and dry and are relieved as much as possible from weight bearing. Tapping with a hammer or causing the animal to stand upon one affected member causes intense pain. Any one or more of the feet may become the subject of the disease although it is more common in the fore feet, except in cases due to parturition, where the hind feet are more commonly involved. If only one foot is affected the symptoms are mild. When made to move the affected foot is held in the air or placed on the ground and quickly removed as the other foot is advanced. Progression in a straight line is easier than turning. When both feet are affected the lameness is well marked, the animal being nearly immovable. When standing the head is down or rests upon the manger to afford support to relieve the painful feet. In attempting to turn the animal, his head will be drawn around before he moves his feet, and it is nearly impossible to back him. In the majority of cases the animal stands till the disease
is over, but if recumbency is voluntarily assumed it will usually be persisted as it affords relief. When in the recumbent position the animal usually prefers to lie broad side. In this position the general symptoms subside greatly and it is with difficulty that the animal is made to rise. If the hind feet are affected they are advanced forward so that the heels receive most of the weight. The fore feet are placed beneath the body so as to bear as much weight as possible. Progression is even more difficult than with the fore feet affected.

Horses suffering from chronic laminitis exhibit a tendency to land on the heel in an exaggerated motion. The sole is dropped and flat, showing excessive quantities of flaky material. The hoof grows more rapidly than normal because of chronic inflammation, and the feet may develop a long toe that curls up at the end. Chronic laminitis causes heavy ring formation on the wall; these rings, usually present throughout the life of the horse, are caused by inflammation in the coronary band. In chronic laminitis, rotation of the third phalanx will occur. This rotation may cause the toe of the third phalanx to push out through the sole of the foot. Rotation of the third phalanx may be caused partially by the inflammation which causes some separation of the sensitive and insensitive laminae. The pull of the deep flexor tendon at its attachment on the semilunar crest of the third phalanx may also aid in displacement of the bone. (2,3)

TREATMENT

Acute laminitis. In a review of the literature, I found that in the 1800's and early 1900's the treatment was "bleeding" the animal. When bleeding from the point of the frog, the sole was thinned as much as possible, the toes shortened, and with a sharp knife, a free incision was made at the point of the frog. Blood flows freely from the incision and the foot is immediately put in a warm bran poultice, to which a reliable antiseptic has been added. At the same time a dose of physic is given. In about six hours after bleeding the foot is put into a hot antiseptic bath for about half an hour. This treatment is repeated several times daily. In about 24 hours there is a marked improvement. (6)

Another method of bloodletting is jugular venesection. Six to ten liters of blood are withdrawn. This lowers the blood-pressure for a definite time and has a direct effect upon the condition. It may cure without delay. (6)

Bloodletting is contraindicated in pregnant mares approaching parturition on account of its harmful influence on lactation and on the fetus itself. Febrile cases which may at any time take on an adynamic character are likewise not favorable for this treatment. In founder caused from a chronic digestive disorder, bloodletting may augment the indigestion or cause serious constipation, because, when a large amount of blood is drawn, the volume is restored by absorption of liquids from the system. (6)

Exercise is very beneficial in treating founder. The patient emerges from the stable with great difficulty and apprehension, but after a few minutes walks easily. The pain noticeably diminishes and the blood pressure of the foot becomes lower. The pulsation of the metacarpals becomes more feeble. The walking should be continued for not less than a quarter of an hour.

Another reference suggested withdrawing 50 to 200 cc. of blood from the jugular vein and then inject intramuscularly, divided into several doses. This apparently has a vaso-constrictor effect upon the vessels. (11)

In grain founder treatment is directed at neutralizing the effects of the ingested grain. Purgation should be used, either with mineral oil or magnesium sulfate. This should be repeated at approximately four to six hour intervals until all grain has been removed from the intestinal tract. To aid the mineral oil, peristaltic stimulant drugs may be used. Intravenous dextrose and electrolytes should be used as replacement fluids for the diarrhea which results both from ingestion of grain and purgation. An effort to cool the feet by standing the horse in a stream, in a pit filled with cool water, or by using ice bags, may be helpful. (10)
In postparturient laminitis the mare must be treated for metritis as well as the laminitis. If retained membranes are still present in the uterus, they should be removed and the uterus packed with antibiotics.

Iodinated casein and thyroid extract have been recommended for grass founder upon the theory that they increase metabolism and aid in reducing the weigh of the horse. Iodinated casein does not give spectacular results, but seems to be worthwhile in reducing weight.

The classic treatments of acute laminitis are interesting. Nearly all reveal an agreement with the histaminic theory, if not to combat the cause itself, at least its immediate manifestations. Antihistamines are the most useful in counteracting the effect of histamine. (4,5)

In summary, the treatment of acute laminitis may be set up according to the mode of action:

1. Lowering of general hypertension.
2. Stimulation of return circulation of the limbs (exercise).
3. Vasconstriction of arteries and arterioles whose local effect protects the capillaries.
4. Elimination, destruction or neutralization of the toxins and the histamine.

In chronic laminitis the foot should be trimmed as nearly normal as possible. Shoes with a wide web may be used as an aid in preventing dropping of the sole. Neurectomies or alcohol block of the median or volar nerves are sometimes used to alleviate pain. Testosterone injections may be valuable in fat geldings. When infection is present in sensitive tissues as a result of a defect in the white line, the defect should be allowed to drain. The foot should be treated locally with tincture of iodine and the foot bandaged. Treatment with corticosterone acetate (cortal 2.5%) gave favorable response in one experiment. The dosage was 500 milligrams of corticosterone acetate intramuscularly per day for three days. (9).

PROGNOSIS

The prognosis is always guarded in any case of laminitis. If the symptoms continue for more than ten days, the prognosis is unfavorable in most cases.

PATHOLOGY

If laminitis persists, permanent deformity is likely to be the result. There may be a separated and downward pointing os pedis and the convex “dropped sole”. The normally straight anterior contour of the hoof becomes concave from the coronet to the toe, which is unduly prolonged, and the unevenly growing hoof forms a series of bulging rings parallel to its coronary origin, each representing a different phase of growth.

Laminitis is not usually fatal, but if a necropsy examination is carried out on an acute case, the stomach contents usually contain excessive amounts of grain, have a pasty consistency and an odor suggestive of putrefaction of protein. Retained placenta and metritis may be present in postparturient laminitis in mares. No other gross findings are visible although there may be perceptible engorgement of the vessels of the sensitive laminae. Histological examination reveals disappearance of some of the keratogenic structures of the inner zone of cornification in the epidermal laminae.

REFERENCES