Laminitis (Coriosis) - Major Risk Factors

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Sole ulcers and white line abscesses are two common causes of bovine lameness. Although lesions are seen on the external surface of the claw, the initial causes are largely associated with changes in the corium, i.e. coriosis. The term laminitis is often (and incorrectly) used to describe such changes, but laminae only occur under the wall of the hoof, whereas sole ulcers are associated with changes in the corium of the sole. This paper outlines the anatomical changes associated with sole ulcers and white line disease and examines aspects of the environment, management and nutrition which are involved.

Sole Ulceration

The axial aspect of the pedal bone is arched on its ventral surface, so that the weight of the cow is taken at its anterior and posterior points and along the abaxial wall. The posterior projection (the flexor tuberosity) is especially important. If excessive weight is taken at this point, then "pinching" of the corium occurs between the pedal bone above and the horn of the sole below. "Pinching" leads to release of blood, so that a mixture of blood and horn slowly moves through the thickness of the sole towards the surface during normal growth. This is eventually seen as blood at the sole ulcer site, referred to as bruising. As sole horn is 8-10mm thick, surface hemorrhage would be caused by trauma some 6-8 weeks previously. A sole ulcer occurs when damage to the corium has been so severe that the horn-forming tissue has been totally destroyed. This is likely to happen when there is a combination of excessive weight-bearing on a corium which is congested and fragile, e.g. in association with metabolic acidosis. Disruption of the laminar suspension of the pedal bone may lead to it "sinking" on to the underlying corium, thereby predisposing to both heel and toe ulcers and disrupting white line formation (Ossent & Lischer 1994). The pedal bone will now be compressing the corium of the sole and the corium is displaced both axially and abaxially, particularly abaxially. This leads to widening (and weakening) of the white line, a hoof which is much wider and poor sole horn production by the corium.

White Line Abscess

The white line is a cemented junction between the wall and the sole. It consists of interdigitating cells and horn leaflet cells (Logue *et al.* 1990). It lacks tubular horn and is therefore considerably weaker than the adjacent hoof of the wall or sole. The horn leaflet cells are produced by the laminar corium and facilitate distal movement of the wall over the underlying laminae. The interdigitating horn cells are produced by the corium of the sole. Any process leading to congestion and inflammation, or any other scenario compromising the function of the corium will weaken the white line. Abscessation may occur due to external penetration by dirt or other foreign bodies, or may be a sterile inflammatory reaction.

Causes of Laminitis and Coriosis

The incidence of sole ulcers and white line abscessation reaches a peak some 2-3 months after calving. This strongly suggests that parturition, or perhaps the initiation of lactation, renders the corium more susceptible to trauma. Similar changes are seen on the horn of a cow's head. A ring of arrested growth develops at each calving. If horn formation on the head is disrupted, then it seems likely that a similar disruption occurs on the horn of the hoof. Lameness increases when other stress factors are superimposed at the time of calving. The many causes of sole hemorrhage have been reviewed by Greenough (1990) and Blowey (1993). The many causes of sole hemorrhage have been reviewed by Greenough (1990) and Blowey (1993). The most common of these include:

1. **Excessive standing.** All animals show decreased lying and increased standing times around the period of parturition (Leonard *et al* 1992). This becomes pronounced in heifers, where there is a fear of aggression from older cows, or where there has been no free-stall training prior to parturition. Heifers may have been reared on pasture or dirt yards until the time of calving and immediately

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after parturition are moved on to concrete and expected to stand for quite long periods feeding and being milked. Lying times in some lactating heifers can be as low as 5 hours/day and these heifers develop quite severe sole hemorrhages 2-3 months post-partum.

2. **Poor free-stall comfort.** Length is the most important dimension determining free-stall comfort. Ideally, they should be 8 feet long (7 feet 6 inches for two facing rows) and 4 feet wide. The use of a triangular shaped block of concrete, 15 inches high and 5 feet 8 inches from the free-stall kerb, increases comfort at the same time as preventing excessive forward movement of the cow. Free-stalls should be well-bedded and comfortable. Lack of bedding can increase sole ulcers (Colam-Ainsworth *et al.* 1989). Mats and cow cushions are becoming increasingly popular in the UK, although both need to be covered with a thin layer of bedding material to prevent hock sores. Passageways should have ample crossing areas and should not be blind ended. There must be at least 10% more free-stalls than cows in the herd, to allow choice and thereby improving lying times. Lameness is much more common in free-stalls than in straw yards (35% vs. 8% in UK, Whittaker 1995). This should persuade farmers to look more carefully at free-stall comfort.

3. **Nutrition.** *Diets high in starch and low in fibre which lead to rumen acidosis are particularly dangerous.* Acidosis may be seen clinically as an increased incidence of digestive upset, irregular or loose fecal consistency and excessive regurgitation of the cud. Very short chop and highly digestible silage with lactic acid levels up to 20% are likely to lead to acidosis, although the addition of 1.0-1.5kg of straw to the diet can be highly advantageous in such circumstances. Levels of fat in excess of 4.0% and excess dietary protein have also been implicated. Ideally, dry cows should be fed a small quantity of the lactation ration for 1-2 weeks prior to calving and concentrate increases post-calving should be slow, reaching a peak no sooner than two weeks after calving. No more than 4kg of concentrate should be fed at any one feed.

   The precise mechanisms by which dietary acidosis affects coriosis are unknown. However, anything producing increased blood flow, edema, hypoxia or thrombosis in the corium, or any other factor leading to increased fragility of blood vessels, would increase the risk of sole hemorrhage (Ossent & Lischer 1994).

4. **General management.** Dry hoof is harder than wet hoof and therefore free-stall passages should be scraped at every milking. Cows should be allowed access to an adequate loafing area for exercise, but be handled quietly and walked slowly when on rough ground. If allowed to walk at their own pace over rough ground, cows will find a "safe" and soft footing. If rushed, this can lead to trauma to the corium and an increased incidence of lameness (Clackson & Ward 1991).

References

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