LAMENESS IN DAIRY CATTLE: NUTRITIONAL APPROACHES FOR PREVENTION AND MANAGEMENT

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Received: 18-06-2014 Accepted: 12-01-2015

ABSTRACT

Role of nutritional factors on incidence and management of lameness in dairy cattle has been discussed. Excessive grain feeding and high ratio of non-fibre carbohydrate to neutral detergent fibre can affect the ruminal function resulting in lameness. Feedstuffs which contain more soluble carbohydrate will increase the production of more lactic acid in rumen resulting in release of vaso-active substances like histamines and endotoxins, which may alter the microvasculature of hoof and results in laminitis. Fat and body condition score have a positive role in improving hoof quality and thickness of the digital cushion, thus prevent sole ulcers and white line disease. Feeding management during transition period is very critical for management of lameness. Total mixed ration containing sufficient amount of fibre can be effectively used to reduce the incidence of lameness. Incidence of lameness can be lowered by adequate incorporation of biotin and organic trace minerals that helps in the production and maintenance of healthy keratinised tissues.

INTRODUCTION

Lameness is considered as one of the most significant welfare and productivity issue causing considerable economic loss for the producer and the dairy industry in general. Reduced milk production, lowered fertility and involuntary culling of lactating dairy cattle can result from lameness. In India, the prevalence of clinical lameness in lactating cows and buffaloes is about 9 and 2 per cent respectively and 40 to 50 per cent cases have subclinical lesions (Randhawa, 2006). Lameness can have multiple causes including nutritional, managemental, environmental, genetic and infective factors. Early detection and proper treatment of the condition can minimize losses, improves the outcome, and reduces animal suffering.

Laminitis is a leading reason for lameness. Laminitis in dairy cattle has been closely associated with rumen function, with production of excessive organic acids in the rumen. Incidence of laminitis also reflects improper ration formulation or feeding management. Feeding of high concentrate diet to lactating cows in order to optimize milk production can contribute to the incidence of lameness. Moreover, rapid urbanization has limited the availability of cultivable land, which in turn leads to a rapid reduction in production and availability of fibre resources. Due to these reasons, dairy farmers in Kerala uses concentrate as the major feed resource and inclusion of fibre in the diet is limited, which can predispose the animal to lameness. Evidence exists on the effects of carbohydrates, protein, non-forage fiber sources and length of fiber particles, as well as other nutritional components such as

macro-minerals, trace minerals and vitamins on hoof epidermis and hoof horn quality. The present article focuses on the role of nutritional factors on the incidence and management of lameness in dairy cattle.

NUTRITIONAL FACTORS CONTRIBUTING TO LAMENESS

- High carbohydrate diet
- Low fibre
- High dietary protein
- Dietary anti-nutritional factors
- Vitamin disorders
- Mineral deficiencies

Effect of dietary carbohydrates

Inclusion of high levels of fermentable carbohydrates in the diet can cause an increase in the level of volatile fatty acids (VFA), a reduction in rumen pH and finally accumulation of lactic acid in rumen (Nordlund *et al*., 2004). During acidosis, there will be production of vaso-active substance like histamine through ruminal conversion of amino acid histidine from the dietary protein by the bacteria *Allisonella histaminiformans*. The low ruminal pH also enhance the endotoxin release by gram negative bacteria (such as *E. coli*). These substances can cause vascular changes within the dermal capillary beds of corium. The pooling of blood in corium leads to ischemia, inflammation and necrosis of the corium-epidermal junction. Due to these events the functioning of keratin produced cells gets impaired resulting hoof lesions (Westwood *et al*.,2003). In milder and prolonged cases of acidosis, the surviving animals suffer from laminitis. The pH stabilization in rumen can be achieved by use of dietary buffers, particularly when the diet contains a high proportion of cereal grains. Agents used as buffers include sodium bicarbonate, sodium sesquicarbonate, potassium bicarbonate, magnesium carbonate, calcium carbonate and bentonite. Microbial feed additives (e.g. *Megasphaera elsdenii* and *Selenomonas ruminantium*) can also be used to control excessive accumulation of acids in the rumen.

EFFECT OF DIETARY FIBRE

An adequate intake of fibre is necessary for maintaining ruminal pH within the normal range. Roughage stimulates chewing and saliva secretions, which will neutralize the acids produced during rumen fermentation and maintain optimum pH. The concept of total mixed ration (balanced mixture of concentrate, roughage and micronutrients / feed additives) can be effectively used to incorporate fibre sources such as crop residues to animals in a completely balanced form. The total dietary dry matter (DM) of total mixed ration should contain a minimum of 25 per cent neutral detergent fibre (NDF), with 19 per cent NDF from coarse forage (NRC, 2001). Moreover, the cows should fed diets with adequate amount of physically effective NDF. i.e., feed particle containing NDF with size greater than 1.2 cm. This particle size stimulates rumination and salivation in cattle.

DIETARY PROTEIN

An inadequate supply of the sulphur containing amino acids (methionine and cysteine) may increase incidence of lameness as a result of the formation of soft horn. Adequate proportion of cysteine and methionine in claw horn of cows are necessary for the formation of disulphide bond during keratinization. High concentrations of rapidly degradable protein may produce high levels of rumen ammonia that may 'buffer' changes in rumen pH. A rapid association of ammonia with hydrogen ions removes hydrogen from solution and may neutralize upto10–15 per cent of VFA produced. Further, microbial growth provides a quantitatively important sink for hydrogen. The toxic effects of high concentrations of

blood ammonia and (or) urea may compromise the sensitive germinal cells of the lamellae and corium. The release of toxic amine – histamine from the amino acid histidine can lead to coriosis and lameness. Use of bypass protein technology for animals producing more 10 litres of milk will be desirable for optimizing production and to reduce the incidence of lameness.

TRANSITION PERIOD AND LAMENESS

The transition period for a dairy cow is from 3 week pre-partum to 3 week post-partum. The transition period is a turning point in the productive cycle of the cow. The manner in which these changes occur and how they are managed are of great importance as they are closely linked to lactation performance, clinical and subclinical post-partum diseases, and reproductive performance that can significantly affect profitability. The transition from low energy – high fibre (dry cow ration) to high energy – low fibre (lactation ration) requires ruminal adaptation of the animals. Such rations also help in maintaining the normal body condition score (Bilcalho *et al.,* 2013). High yielding cows requires more nutrients and energy for milk production and are usually prone to lameness than low yielding cows (Green *et al.,* 2002). The decreased body condition score of the cow during calving to early lactation may predispose to fat mobilization and lameness. Cows mobilize fat from the digital cushion of hoof reaching its lowest point four months into lactation and the incidence of lameness was highest during first lactation (Hirst *et al.,* 2000; Livesey *et al.,* 2000).

ANTI-NUTRITIONAL FACTORS

Nitrate

Nitrate toxicity is seen in dairy cows grazing rapidly grown crops and pastures following dry or drought conditions. Forage crops such as maize, oat hay, alfalfa hay and Johnson grass have tendency to accumulate nitrate. Under normal rumen pH there is rapid reduction of nitrate to nitrite. This nitrite in rumen reduced to ammonia and is either utilized by the rumen microbes or converted to urea and excreted through urine. Toxicity happens if the reduction of nitrate to nitrite exceeds the reduction of nitrite to ammonia. Excess nitrite in circulation converts haemoglobin to methaemoglobin, reducing the oxygen carrying ability of the blood. Nitrite is a potent vasodilator. Stagnation and pooling of blood in the peripheral circulation, including the vascular beds of the corium, may induce anoxia and the accumulation of tissue toxins, causing laminitic lesions.

Mycotoxins

Ergotism: Ergot poisoning results from ingestion of alkaloids produced by the fungus Claviceps purpurea, which infects the mature seed head of rye grass, wheat and barley. This can result in lameness, swelling and gangrene of foot and lower hindlimbs, and loss of extremities as a result of the vaso-constricting alkaloids, ergotamine, ergonovine (ergometrin) and ergocriptine.

Fescue foot and Perennial ryegrass endophyte: Lameness in cattle grazing standard (or wild type) high endophyte tall fescue (Festuca arundinacea) is associated with alkaloids produced by the endophyte Neotyphodium coenophialum and Neotyph odium lolii particularly the ergot peptine alkaloid, ergovaline, effects as a dopamine agonist and vaso-constrictor. It will compromise blood flow to the extremities of cattle leading to sloughing of the hoof.

Vitamins

Biotin

Biotin is required for the synthesis of long

chain fatty acids and helps in the production of complex lipid molecules in the intercellular cementing substance. Adult rumen may synthesize adequate amounts of biotin for various biological needs. Typical dairy diets containing more than 50% of DM as grain or concentrate may decrease ruminal synthesis of biotin. Supplementation of biotin (non-rumen protected) in feed at 20 mg/day can reduce the incidence of lameness (Hedges *et al.*, 2001). Ration with high forage can stimulate ruminal biotin synthesis.

Other vitamins

Vitamins A, D and E have integral role in the structure and quality of keratinized horn tissue. Vitamin A is required in the differentiation of keratinizing cells. Vitamin D is a regulator of calcium metabolism and has a positive effect on keratinization. Vitamin E is a lipid-soluble anti-oxidant and maintains lipid-rich, cellular membranes in the intercellular cementing substance of horn tissue.

Minerals

Copper:. It activates thiol oxidase enzyme, responsible for formation of disulfide bonds between Cystein residues of keratin filaments. Cattle suffering from a subclinical Cu deficiency showed heel cracks, foot rot, and sole abscesses due to insufficient cytochrome C oxidase activity, resulting in deficient energy supplies for differentiating keratinocytes (Ballantine *et al.*, 2002).

Zinc: Zinc helps in the formation of the structural proteins during the keratinization process. It regulates calmodulin, protein kinase C, thyroid hormone binding, and inositol phosphate synthesis. Zinc is required for activation of the cytosolic enzyme Cu/Zn superoxide dismutase (SOD), which prevent peroxidation of lipid.

Manganese: Manganese helps in the activation of galacto transferase and glycosyl transferase enzymes needed for the synthesis of chondroitin-sulfate side chains of proteoglycan molecules that are essential in the formation of cartilage and bone. Manganese activates pyruvate carboxylase and is responsible for gluconeogenesis.

Selenium: It is a constituent of the enzyme glutathione peroxidase, and protects both the intra- and extra-cellular lipid membranes against oxidative damage. Excessive supple mentation of Se may be damaging to developing keratinocytes.

Calcium: Helps in the keratinization and cornification process. Calcium is needed for activation of epidermal transglutaminase (TG), which is active in cross-linkage of the cell envelope keratin fibers and involved in the terminal differentiation of the epidermal cells (Tomlinson *et al.*, 2004). Inadequate vascular supply or hypocalcemia may lead to depressed TG activity and formation of dyskeratotic horn.

NUTRITIONAL MANAGEMENT

Ensure optimum fibre in the diet and at least 15 per cent of fibre particles should exceed 1.5 inches in length. Forage NDF should exceed 19 to 21 per cent depending on forage digestibility and inclusion level of non-forage fibre.

Avoid excessive grain feeding and enhance use of slowly fermentable polysaccharides. Add buffers such as sodium bicarbonate (100 to 150 gm per day) to lactation diets. Use total mixed ration. Feed forages before grain or concentrates if total mixed ration is not used.

Follow transition ration. Use minimum of two rations (close-up dry cow and fresh cow rations) for rumen adaptation during transition period. Do not change the energy level between the transition rations to more than 10 per cent.

CONCLUSION

Attention to proper hoof health is an important part of day-to-day dairy management. By improving hoof quality and reducing the effects of hoof injuries and diseases, significant improvements can be made to dairy profitability. Proper nutrition is a key factor in this regard since it acts as a first line of defense in maintaining the hoof integrity. Hoof health management requires a team approach and involves the dairyman, veterinarian and nutritionist. Research should be focused to develop farmer friendly technologies for management of lameness and hoof health in cattle.

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