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REVIEW ARTICLE

Nutrition and Acidosis in Ruminants

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Abstract

Acidosis is a common digestive disorder in ruminants which are heavily fed on concentrate diets. Ruminal acidosis or increased accumulation of organic acids in the rumen reflects imbalance between microbial production, microbial utilization, and ruminal absorption of organic acids. Acidosis occurs when there is more lactic acid production than the capacity of ruminal microbes to utilize it. Amount and frequency of grain/concentrates feeding reflects the severity of acidosis. Two types of acidosis are observed in ruminants i.e. acute and chronic. Response of animals is inconsistent to readily fermentable carbohydrates. Acidosis is a major problem affecting production performance of animals. But the physiological basis of this has not been well understood. The rumen is complex consortium of various microbes. It is a highly reduced and anaerobic ecosystem in which microbial digestion of feedstuffs converts fermentable substrates into organic acids, which are then removed primarily by absorption and utilized as a source of energy. These microbes are depends on the availability of feed materials (substrates) from which they synthesize amino acid as per their requirements. Rate of fermentation in rumen is depends on the pH which is maintained around 6.2-6.8. Ruminal pH is the function of feed intake especially carbohydrate and absorption of various organic acids and thus vary throughout the day. Management tools such as proper ration formulation and feed management used to prevent acidosis. Control measures primarily include feed additives which inhibit lactate producing microbes, stimulate the activity of lactate utilizing bacteria and ruminal protozoa.

Keywords: Ruminants, Acidosis, Treatment, Nutrition.

1. Introduction

The major factor determining the severity of acidosis is pH, pH below 5.5 results in acidosis. In sub-acute or chronic acidosis rumen pH falls in the range of 5.0-5.5. Acute acidosis is the condition when pH of rumen fluid falls below 4.5 (Britton and Stock, 1989; Owens *et al.*, 1998; Krause and Oetzel, 2006). In acute acidosis, fall in ruminal pH is due to the excess accumulation of organic acids. Consequently, decreased concentration of total volatile fatty acids observed as normal rumen microflora diminished. Research suggests that non-dietary physiological factors, such as capacity for absorption of acid primarily regulate the pH in rumen (Aschenbach *et al.*, 2011).

Under modern production system of feeding, ruminants are generally fed on high grain diets to maximize energy intake and production. The ruminal papillae plays important role as it increases surface area and led to increase absorption rate of various nutrients (Mahesh *et al.*, 2014; Lakshmishree *et al.*, 2015, 2016). Further research is warranted in this direction. However, feeding highly fermentable diets often causes excess fermentation and results in accumulation of fermentation acids in the rumen, results in reduced feed intake, poor feed efficiency, liver abscesses, and lameness in animals. Apart from this acidosis corrodes arteries, veins and heart tissues, accelerates free-radical damage therefore results in subsequent heart related problems. The topic of acidosis in ruminants has been reviewed extensively by Counotte and Prins, 1981; Britton and Stock, 1989; Nocek, 1997; Owens *et al.*, 1998; Krause and Oetzel, 2006. In present article nutritional overview is summarized.

2. Etio-Pathogenesis

Common causes of acidosis are readily available source of soluble carbohydrates, low fibre in rations, finely grounded forage, shifting of animals from a high fiber to high concentrate diet that is rich in fermentable carbohydrates (starches and sugars). In ruminants, fermentation of soluble carbohydrates produces lactic acid which is efficiently utilized by lactic acid utilizing bacteria. When soluble carbohydrates are speedily consumed by ruminants then rate of lactic acid production exceeds the rate of utilization resulting in acidosis. At the same time hurriedly consumption causes less amount of saliva entering in the rumen which has buffering activity due to presence of carbonates and bicarbonates. Thus, saliva is not passed in rumen at proper rate resulting in increased acidity of rumen and pH of the rumen is not maintained around 6.2-6.8. Large amounts of starch and sugar stimulate the multiplication lactic acid producing bacteria. Consequently, bacteria that normally utilize lactic acid are unable to utilize as the rate of production of lactic acid is higher. This is adversely affects the population of cellulolytic microbes and protozoa. The osmolality of the ruminal fluid is increased which is attributed to increased concentration of lactic acid and stimulates the flow of water from blood stream to rumen resulting in dehydration. Rumen motility also decreases as rumen pH falls, sometimes motility even stop. This might be due to higher concentration of butyric acid but lactic acid probably exerts a reflex inhibitory action on entering duodenum. A significant decreased in water absorption from colon results in diarrhea (Lee, 1977). Consumption of grains causes proliferation of coliform and *Clostridium* species of microbes causing diarrhea (Slyster, 1976). In mild lactic acidosis, the plasma buffering system works well and animal may show recovery but in acute acidosis, all systems to neutralize acidity are failed. The reduced pH of ruminal fluid is favorable condition for growing and multiplication of fungi such as *Mucor*, *Rhizopus*, *Absidia sp.* which further invades blood vessels and produces thrombosis and infarction and spread to liver (liver abscess) apart from severe ruminitis produced. The ruminitis along with toxaemia is reported in acute acidosis which is fatal. In chronic acidosis, mild ruminitis along with hyperkeratosis may develop. Thus, it is recommended for dairy and fattening animals that concentrate mixture should be gradually increased over a period of 2 months and there should not be sudden shift to high concentrate diets.

The optimal rumen pH should be 6.2-6.8. Lactic acid is about 8-10 times stronger acid than the other rumen acids and causes the rumen pH to decrease (acidic). As the rumen pH drops below 5.8, bacteria that digest fiber begin to die and thus, fiber digestion is depressed. Because the end products of fiber digestion are used for milk fat synthesis, a drop in milk fat is a sign of acidosis. If the rumen pH continues to decline and falls below 5.5, many other beneficial rumen bacteria also begin to die. As lactic acid accumulates, it is absorbed and lowers the pH of the blood also. High levels of acid can also causes ulcers in the rumen resulting in infiltration of bacteria into blood that causes liver abscesses. Endo-toxins resulting from high acid production in the rumen also affect blood capillaries in the hoof, causing them to constrict resulting in laminitis. Common symptoms of acidosis are dullness, falling on ground, bulging of rumen with doughy sound on palpation, reduced rumination, diarrhoea, acidic pH of rumen contents, liver abscess, laminitis, and reduction in milk fat percent. Symptoms also includes loss of appetite, even affected animals refused to drink water, soft and watery faeces with sweet-sour smell, undigested feed particles may also seen in faeces. Subnormal temperature, increased heart and respiration rate are reported (Nocek, 1997). Recent research findings indicate that rumen acidosis negatively affects nutrient digestibility and animal health (Gressley *et al.*, 2011) and endo-toxins released can also cause systemic inflammatory responses (Andersen, 2010) and laminitis. Some researchers reported that acidosis and laminitis is commonly seen the areas which have prevalence of mycotoxins. This probably might be due to alteration in lactic acid metabolism which causes accumulation of lactic acid leading to acidosis. This needs to validate through further research in this direction. Ruminal fluid is withdrawn with the help of needle and syringe from the flank region (rumenocentesis) and the acidity of rumen fluid can be measured either by pH strip or pH meter which indicates lower pH of rumen fluid, absence of rumen protozoa is seen microscopically. Some biochemical test are used for diagnosis are acidic urine, increased lactate, inorganic phosphorus and reduced bicarbonate in blood and marginal hypocalcaemia. Diagnosis should be based on clinical signs and history and confirm by above mentioned tests. Acidosis can be differentiated from milk fever in which firm and dry faeces are seen than former which showed soft with fluid pockets in faeces.

3. Acidosis and Microbial Changes in Rumen

In rumen, rate of bacterial growth and fermentation is increased in response to availability of readily fermentable substrates leading to increased volatile fatty acids (VFA) production. When readily fermentable sources of carbohydrates are available

abundantly, then bacterial changes occur. These are primarily starch and soluble sugar fermenting bacteria and lactic acid producing bacteria (Nagaraja and Titgemeyer, 2006). Lactate is an intermediate product of ruminal fermentation. This lactate is again metabolized to VFA. The adaptation of the rumen to the high grain diet (concentrates) results in increased populations of bacteria capable of utilizing lactic acid (Huber *et al.,* 1976; Mackie and Gilchrist, 1979; Counotte and Prins, 1981). Major lactate utilizing bacteria are *Megasphaera elsdenii* and *Selenomonas ruminantium* in concentrates fed animals (Huber *et al.,* 1976; Mackie *et al.,* 1978). Decline in pH of ruminal contents is mainly due to lactic acid produced by *Streptococcus bovis*, which in turn inhibits growth of other beneficial bacteria in rumen leading to acute acidosis. Thus, acid tolerant *Lactobacilli* become more predominant. Therefore, *Streptococcus bovis* is considered to be a major etiologic agent of acute acidosis. Intervention to prevent acidosis includes strategies such as antibiotics and vaccines are often targeted at controlling the growth of *Streptococcus bovis* in the rumen (Nagaraja and Miller, 1989; Gill *et al.,* 2000). *Lactobacilli* are more resistant to low pH than *Streptococcus bovis* that is why they are more dominant in the acidic rumen (pH<5.6) (Slyter *et al.,* 1976). A considerable increased in the population of ruminal *lactobacilli* is a common feature of both acute and sub-acute acidosis (Slyter, 1976; Nagaraja and Miller, 1989; Goad *et al.,* 1998).

Number of ciliated protozoa is reported to increase on moderate feeding of concentrates (Dennis *et al.,* 1983). However, increased amount of concentrates results in complete elimination of protozoa (Nagaraja and Towne, 1990; Towne *et al.,* 1990 ab; Franzolin and Dehority, 1996; Goad *et al.,* 1998). It is known that reduced pH in rumen causes destruction of protozoa (defaunation) as they are more sensitive to pH changes (Whitelaw *et al.,* 1984). Some ciliated protozoa viz. *Entodinium, Polyplastron, Isotricha,* and *Dasytricha* (Towne *et al.,* 1990a) are resistant to low pH in rumen. The genus *Entodinium* is the most resistant to low ruminal pH. That is why genus *Entodinum* is the most dominant (about 90 to 99%) of the total protozoal population, in cattle fed on concentrate diets (Towne *et al.,* 1990 ab; Franzolin and Dehority, 1996; Hristov *et al.,* 2001).

4. Laminitis

Decarboxylation of amino acids results in production of histamines, tyramines, tryptamine etc. which are believed to cause the laminitis. Reduction in pH of rumen causes the accumulation of histamine (Van Der Horst, 1961; Irwin *et al.,* 1979). The major factor responsible for laminitis is the destruction of normal haemo-dynamic process by histamines (Nocek, 1997). Brent (1976) reported that the histamine is a potent vasodilator which increases the capillary permeability thus results in laminitis. Rodwell, (1953) assumed that ruminal *lactobacilli* is the main producers of histidine decarboxylase. But recently, Garner *et al.* (2002) reported the *Allisonella histaminiformans* a gram-negative and ovoid species, as an important histamine producer in the rumen. This bacterium is acid tolerant and grows even at a pH of 4.5. The increase in absorption of histamine may be because of increased epithelial permeability or decreased catabolism in the epithelial cells.

5. Nutritional Control Measures

In case ruminant animals are noticed soon after consuming large amounts of grain and before they drink water, problems may be avoided by keeping them away from water for up to 24 hours. Some other approaches include oral administration of mineral oil or sodium bicarbonate along with activated charcoal, anti-endotoxin therapy and surgical emptying of the rumen in severe cases. Management tools such as proper ration formulation and feed management used to prevent acidosis. Control measures primarily include feed additives that can inhibit lactate producing microbes, stimulates the activity of lactate utilizing bacteria and ruminal protozoa. Inoculation of rumen with microbes which prevents the accumulation of glucose and lactate and they can also metabolize lactate at low ruminal pH. Maintenance of pH is very important to restore fiber digestion. This can be done by increasing buffering capacity of rumen, use of slowly fermentable polysaccharides and microbial feed additives. Incidences of acidosis can also be reduced by proper grain processing, salivary flow stimulants, feeding management, feed additives. Feeding more roughages and limiting the quantity of concentrates reduces the incidences of acidosis. Avoiding sudden switch to more concentrate ration will be a good start for the prevention of acidosis.

6. Conclusion

Animals fed heavily on concentrate diets produces acidosis. Acidosis occurs when there is more lactic acid production than the capacity of ruminal microbes to utilize it. These acids reduce ruminal pH and caused a shift in microbial populations even damages gut epithelium. A damage to gut epithelium in response to exposure to fermentation acids produced during acidosis can allow for systemic entry of bacteria, amines, or toxins and result in laminitis and other health disorders. Acidosis is a major problem

affecting production performance of animals. Management such as proper ration formulation and feeding used to prevent acidosis. Other control measures primarily include feed additives which inhibit lactate producing microbes, stimulate the activity of lactate utilizing bacteria and ruminal protozoa.

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