Sub acute ruminal acidosis and its effects on production

Manju K Mathew\textsuperscript{1}, S Ajithkumar\textsuperscript{2}

\textsuperscript{1,2}(Dept. of Clinical Veterinary Medicine, College of Veterinary and Animal Sciences, Mannuthy, Kerala Veterinary and Animal Sciences University, Kerala, India, 680651)

Abstract: Sub acute ruminal acidosis (SARA) or subclinical rumen acidosis is an economically important and common condition seen in well managed dairy cattle herd as a consequence of feeding high grain and low fibre diet resulting in subsequent ruminal fermentation disorders and other changes which adversely affects production performance and finally economy of cattle rearing. Diagnosis of the condition is a tedious task as the clinical changes will be expressed only at an irreversible stage. Proper management of the postpartum cattle along with adequate feeding practices will help in prevention of the condition and improving economy of the farm.

Keywords: economic loss, postpartum, ruminal acidosis, transition cow management

I. Introduction

Subacute ruminal acidosis is an important production problem all over the world since it leads to drastic reduction in the production capacity of the cattle all over the world. The condition is often easily overlooked and so all the consequences are a surety since the clinical signs will be expressed only at a late serious stage. This review article throws light on the etiological factors and major consequences of SARA of which veterinarians and farmers should be aware of to improve the economic losses of the farm.

II. Etiology

Ingestion of large amounts of concentrates and inadequate amounts of fiber in early lactation is the major proposed cause of SARA (Radostits et al, 2009). Intake of poor quality fodder is also a major contributing factor. Grains are more toxic when ground finely or crushed or cracked which exposes the starch component of grain to ruminal microflora.

The transition period starting from 3 weeks before to 3 weeks after parturition is the period during which SARA occurs in dairy cattle. Absence of the adaptation period for rumen microflora and papillae leads to disease development. Cows in early lactation are exposed to energy-rich rations too rapidly resulting in low rumen pH and cows in mid-lactation, due to their high feed intake, are particularly sensitive to sudden changes of feed or faults in feed composition and delivery (Nordlund et al, 1995).

III. Pathogenesis

Pathogenesis of SARA in lactating dairy cows is not clearly understood. Diet deficient in physically active fiber and rich in highly fermentable carbohydrates causes SARA in early lactating dairy cows. Fermentation of nonstructural carbohydrates leads to production of large quantity of volatile fatty acids and lactate which on accumulation in rumen subsequently reduces its pH. No detailed reports on biochemical changes associated with SARA are available. Lactic acid levels in animals affected with SARA showed only mild elevation suggesting that excessive volatile fatty acid production may be the more important factor causing SARA and change in pH. SARA affects productivity of dairy cattle by reducing the fibre digestion since low pH adversely affects cellulolytic bacteria.

IV. Clinical Findings

SARA is considered as the most important nutritional disease affecting dairy cattle which predisposes animals to various other disease conditions (Tajik et al, 2010). No typical clinical signs are seen in animals affected with SARA and at times manifestations may take weeks to express. Cyclic decrease in dry matter intake characterized by high dry matter intake on one day followed by a low intake on the following day is a consistent feature and sensitive indicator of SARA. An increased production of volatile fatty acids, a temporary rise in ruminal lactate concentration and fluctuations in the osmolality of the rumen fluid are involved in the development of rumenitis. Mucosal damage favour entry of unfavorable bacterial organisms which may result in embolic spread to liver resulting rumenitis – liver abscesses complex. Rumen tympany because of reduced rumen motility caused by low fibre ration and low rumen pH is noticed. The condition is characterized by daily episodes of low ruminal pH between 5 and 5.5 (Krause and Oetzel, 2006). Excess production of
mucopolysaccharides and release of unknown macromolecules from rumen bacterial disintegration forms a stable froth which hinders eructation of gas.

Laminitis characterized by hoof discolouration, sole hemorrhages, sole ulceration and misshapen hooves is seen in affected cattle. There can be a long time gap between SARA occurrence and clinical signs of laminitis. Vasactive substances like histamine and endotoxins released during reduction of rumen pH, bacteriolysis and tissue degradation causes injury to microvasculature of corium resulting in reduction in oxygen and nutrient supply to extremities and physical degradation of junctures between tissues that are critical for locomotion. The insidious rotation of distal phalanx results in permanent anatomical change.

Relationship between SARA and milk fat depression depends on lactation state, breed of animal and lactation diet. Increased intraruminal propionate production and subsequent increase in blood glucose leads to increased lipogenesis in fat tissue results in lowered milk fat content (<1 in SARA). Increased blood glucose also increases milk lactose content. Milk urea nitrogen will be <3.0 mmol/L as a result of reduced ruminal ammonia formation.

Variable faecal consistency especially loose faeces with size of ingesta particles larger than normal are seen and are low in pH. Faeces are bright, yellowish, with a sweet–sour smell and appear foamy with gas bubbles. Presence of fibrin casts (1-2cm sized) in faeces, excessive body faecal soiling, continous tail swishing, poor reproductive performance, environmental mastitis is also reported.

SARA may indirectly affect fertility in addition to calving and health of the newborn calf.

V. Diagnosis

Diagnosis of SARA is difficult because of variable and subtle changes and also because some changes occur weeks or months later. Lack of pathognomonic signs is also a reason for delay in diagnosis.

Rumen fluid examination

The major parameter which is analyzed on rumen liquor is its pH. SARA is defined as repeated bouts of decreased ruminal pH below 5.6 for 3-5h /day. Presence of an indwelling catheter continuously monitors changes in ruminal pH and provides better information about pH changes. Various methods are adopted for collection of rumen liquor. A pH of 5.5 has been considered as the cut off between normal and abnormal animals and cows with a rumen pH of 5.5 or less at the time of estimation has SARA. A decrease in rumen pH causes the decrement of cellulolytic bacteria and predomination of gram positive cocci and rods. Microbial population of rumen is different in SARA affected and unaffected animals (Khafipour et al, 2009).

Rumen content of free lipopolysaccharide (LPS) is found to be increased in induced SARA due to increase in lysis of gram negative bacteria. Ruminal pH has a negative relationship with ruminal temperature (Al-Zahal et al, 2008). Temperature range from 39-41°C corresponds to ruminal pH range of 5-5.6 which is critical for detection of SARA.

Urine pH

Positive correlation has been established between rumen pH and urine pH in some studies and is believed that urine acidity is an efficient parameter in SARA diagnosis (Enemark et al, 2002).

Faecal sieving

On sieving fecal sample under running water using a standard sieve, presence of large particles of fibre (>2.5cm), undigested grains and fibrin casts are suggestive of presence of ruminal acidosis. No scoring method based on this is available so far.

Necropsy findings

Changes which are noticed on autopsy include rumenitis, rumen parakeratosis, liver abscesses and pulmonary bacterial emboli.

VI. Treatment And Prevention

In severe cases of SARA, therapeutic measures applicable to acute lactic rumen acidosis may be applied and in less severe cases, a modification of the diet of the animal along with additional supplementation of ruminal buffers will reduce the consequences of SARA. Increasing focus is now paid on prevention rather than treatment.

Feeding and management

Correction of feed rations and feeding management is essential to solve the problem of SARA. Proper adaptation of the ruminal mucosa and microflora in the periparturient keeps ruminal pH in physiological ranges and helps in disease prevention.

Dietary buffers

Addition of buffers when fiber content of feed is low is found to be effective. Addition of 150gm of sodium bicarbonate to the lactation feed per day has a positive effect on milk yield and milk fat percentage.
Direct fed microbials (DFM)

Addition of yeast cultures to the feed is found to have a beneficial effect. Incorporation of culturable and cellulolytic bacteria recovered from rumen has also a positive effect.

Stimulation of lactolytic flora

Genetic manipulation of lactolytic bacteria increases the lactate conversion capacity and acid resistance of the bacteria but no commercial products are available. Supplementation of dicarboxylic acids, such as fumarate and maleate can stimulate lactolytic flora.

Immunization

Immunization with a live Streptococcus bovis vaccine is shown to reduce the risk of lactic acidosis. Antibodies produced will be released through saliva. Research to improve this hypothesis is not yet available.

Antibiotics

Use of antibiotics to control the lactate production by S bovis and Lactobacillus sp has been studied but is still doubtful. Use of ionophores like monensin which increase total tract nutrient digestion with less effect on DM intake, rumen pH regulation and change in milk yield has also been documented.

VII. Conclusion

SARA is capable of causing irreparable economic loss both directly and indirectly. Losses are mainly due to reduced milk production, decreased efficiency of milk production, premature culling and increased death loss. Diagnosing SARA is difficult because clinical signs are subtle and delayed after the time of the acidic insult. As a result, routine monitoring and recording of related disease incidences, clinical signs and dynamics of the affected, para-clinical parameters may be the only ways to recognise SARA at an early stage to allow for corrective measures in management or feeding procedures.

References