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The Ultimate Guide to SARA Diagnosis and Care: A Review

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ABSTRACT

Milk production per cow has increased due to better genetics and feeding practices, but high-energy diets can lead to digestive disorders like subacute ruminal acidosis (SARA) which can decrease milk yield and negatively impact cow health. The causes of SARA are excessive intake of rapidly fermentable carbohydrates and inadequate ruminal adaptation, with cows in early and mid-lactation being the most vulnerable. To manage SARA, proper transition from dry to lactation group is important along with monitoring palatability, maintaining a homogeneous TMR, managing forage length, keeping records and providing access to feed and water. A well-maintained resting area is also needed. Smooth transitions are important when changing formulas or forages. This review article covers the causes, diagnostic techniques, preventive and treatment methods of this disease in order to raise awareness about it. SARA is generally observed in high yielding cattle and buffalo so this review article aims to provide valuable information to dairy farmers who own large-scale farms and have high-yielding cattle and buffaloes.

Key words: High energy diet, Milk yield, SARA, TMR.

Over the past 20 years, milk production per cow has significantly increased due to genetic improvement and improved feeding management. Successful dairy farms now use total mixed rations with a high proportion of energetic dense feed components to provide optimal energy supply for high-producing cows (De Brabander *et al*., 2002). Diets high in starch and low in fiber are fed to increase intake of energy, but these diets increase the risk of subacute ruminal acidosis (SARA) (Bipin *et al*., 2016).

Sub-acute ruminal acidosis is a digestive disorder that affects high yielding dairy cows, leading to negative impacts on animal health and herd profitability. This disorder is particularly prevalent in well-managed dairy herds (Antanaitis *et al*., 2015). This digestive disorder is the consequence of feeding high grain diets to dairy cows, which are adapted to digesting predominantly forage diets. The current definition and ruminal pH threshold for SARA vary among studies. However, SARA generally occurs when ruminal pH stays in the range of 5.5-5.2 for a prolonged period (Li *et al*., 2013).

Etiology of SARA

Ruminal acidosis in dairy cattle can be caused by excessive intake of rapidly fermentable carbohydrates, inadequate ruminal buffering and inadequate ruminal adaptation to a highly fermentable diet. The major cause is the ingestion of large amounts of concentrates and inadequate amounts of fiber in early lactation (Constable *et al*., 2017). Poor quality fodder and finely ground grains also contribute to the problem. The transition period from 3 weeks before to 3 weeks after parturition is when SARA occurs, due to absence of the adaptation period for rumen microflora and papillae. Cows in early lactation and mid-lactation are particularly sensitive to sudden changes in feed or faults in feed composition and delivery (Nordlund *et al*., 1995).

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Occurrence of SARA

Subacute ruminal acidosis in the early post-partum period

During the calving period, dairy cattle face stress due to calving, lactation, low feed intake and changes in management. This leads to negative energy balance and higher risk of disease. A high lactation diet can increase the possibility of sub-acute ruminal acidosis. To mitigate these challenges, dairy farmers should monitor feed intake, provide appropriate nutrition and implement proper management practices. This is necessary for the success of their operations and prioritizing animal health and well-being (Nocek, 1997).

Subacute ruminal acidosis in mid-lactation

SARA in mid-lactation is caused by managerial factors such as feeding frequency, processing of feed and housing. Mistakes in automatic feeding or preparing total mixed rations can lead to this problem. In herds fed on component-based diets or TMR-fed, there can be issues with a too-high

concentrate component and fiber-deficient rations, as well as over-mixing of the ration leading to low-structure diets with high palatability that may not provide necessary buffering capacity within the reticulo-ruminal compartment (Nordlund *et al*., 1995; Garrett, 1996).

Pathogenesis

SARA begins when a low structured, high energy diet is consumed before the ruminal environment is prepared to efficiently ferment it while maintaining stable pH levels. The ruminal wall and papillae are integral to this process (Dirksen *et al*., 1985). The ruminal papillae are important for absorbing short chain fatty acids (SCFA) and their growth is promoted by SCFA fermentation. If the ruminal mucosa is not adapted to a sudden increase in SCFA, as seen when transitioning from a dry-period to high lactation diet, the papillae are too short, resulting in a smaller reabsorbing surface and insufficient bacterial population to metabolise lactic acid (Nordlund *et al*., 1995).

Fermentation of carbohydrates produces acids that can reduce ruminal pH, leading to rumenitis and other health issues. SARA is defined as a drop in ruminal pH below 5.5, caused by poor adaptation of ruminal microflora and mucosa. This results in chronic health problems, whereas acute ruminal acidosis poses a more immediate risk. The critical pH threshold is identified at pH 5.5, a few hours after concentrate feeding (Oetzal, 2000).

Clinical findings

SARA is a significant nutritional disease in dairy cattle that makes them susceptible to other illnesses. There are no specific signs of illness in cows affected by SARA (Tajik *et al*., 2010). SARA, a condition found in dairy cows, is linked to inflammation in various organs and tissues, including rumenitis, rumen parakeratosis, liver abscesses and pulmonary bacterial emboli. These clinical signs are detectable during autopsy and indicate previous bouts of acidosis. Additional symptoms include fibrin casts in faeces, excessive body soiling, tail swishing, poor reproductive performance and environmental mastitis (Grove-White, 2004). The following are some of the most prevalent clinical indicators that will be discussed.

Rumenitis-liver abscess complex

Rumenitis is caused by increased production of volatile fatty acids, temporary rise in ruminal lactate concentration and fluctuations in rumen fluid pH. It leads to mucosal damage, allowing unfavourable bacteria to enter and spread to the liver, causing the Rumenitis-liver abscesses complex. Colonized papillae may also leak bacteria into circulation, leading to liver abscesses and peritonitis. These bacteria can also spread to other organs such as the lungs, heart valves, kidneys, or joints.

Laminitis

Laminitis is a complex disease with an unclear origin that leads to a breakdown of lamellae tissue. It is important to maintain a healthy weight as obesity increases the likelihood of developing laminitis, regardless of its cause (Akil *et al*., 2018). Laminitis in cattle is characterized by discoloured hooves, haemorrhages, ulcers and misshapen hooves. It can occur after a long period of SARA and is caused by vasoactive substances released during rumen pH reduction, causing injury to the microvasculature of corium. This results in reduced oxygen and nutrient supply to extremities, leading to degradation in joint tissues that affect movement.

Rumen tympany (bloat)

A diet low in fibre can cause a reduction in rumen motility and low ruminal pH, leading to the excess release of mucopolysaccharides and unknown macromolecules. This can create stable foam that prevents the release of gases, leading to ruminal tympany (Enemark, 2008).

Loss of body condition

Subacute ruminal acidosis (SARA) can cause a reduction in body condition in animals. During SARA, the accumulation of short-chain fatty acids in the rumen reduces the pH level and alters the rumen microflora, leading to reduced fiber and carbohydrate digestion and a loss of energy. This sometimes results in reduced body condition without a reduction in intake (Dijkstra *et al*., 2012).

Alterations in feces, diarrhoea

SARA affected cows often have inconsistent feces and loose stools due to lower fecal pH and potentially larger ingesta particles. However, these changes are usually temporary and often go unnoticed since only a few cows' exhibit symptoms at a time. SARA cases have distinct fecal characteristics - bright yellowish color, sweet-sour smell, foamy texture with gas bubbles and higher amounts of undigested fiber or grain. Due to an inadequate ruminal fiber mat, fiber is not retained in the rumen resulting in larger sized fiber particles in the feces (1-2 cm compared to the usual 0.5 cm) (Hall, 1999).

Polioencephalomalacia (cerebrocortical necrosis)

A high grain diet destroys normal bacteria in an animal's digestive system and allows bacteria that produce thiaminase enzymes to thrive. Two types of thiaminase enzymes are produced by different bacteria, which break down thiamine. This deficiency can cause Polioencephalomalacia (Hernandez *et al.,* 2014).

Sara diagnostic techniques

Li *et al*. (2013) suggest that feeding behavior can be used as a diagnostic tool in dairy cows. The article discusses the most common techniques and tips for diagnosing SARA (subacute ruminal acidosis) in cows.

Rumenocentesis technique

Rumenocentesis is a method for diagnosing SARA in dairy herds introduced by Nordlund *et al.* (1995). This involves obtaining rumen fluid from the caudoventral rumen using a percutaneous needle aspiration technique. To perform rumenocentesis, the puncture site should be clipped,

disinfected and locally anesthetized. The puncture is made 12-15 cm below the last rib's costochondral junction, on a horizontal line with the top of the patella, using a 16 gauge, 10 cm long needle. A 20 mL syringe can aspirate 10 mL of ruminal fluid (Nordlund, 2003).

Rumen cannula method

Ruminal cannulation is the best way to get samples of ruminal fluid, but it's only used for research purposes (Nocek, 1997). Repeatedly removing and replacing the cannula cover can cause discomfort for the animal and allow digesta to escape (Tajik and Nazifi, 2011).

Manure evaluation

Increasing grain content in diets may cause SARA and lead to more dietary nutrients bypassing the rumen and reaching the hindgut. This excessive hindgut fermentation changes the consistency and appearance of manure, which can be used as a diagnostic tool to evaluate rumen functionality. Normal rumen function should result in manure with few recognizable feed particles, no longer than half an inch. Watery and foamy manure indicates abnormal fermentation in the hind gut, while mucin casts suggest damage to gut epithelium (Li *et al*., 2013; Hall, 1999).

Fecal lipopolysaccharide (LPS)

Dairy cows fed high-grain diets that cause sub-acute ruminal acidosis are at risk of having higher levels of lipopolysaccharide (LPS) endotoxin in their feces, which originates from gram-negative bacteria (Li *et al*., 2013).

Blood gas analysis

Using gas analysis is a non-invasive method to diagnose acidosis in dairy cows, according to Gianesella *et al*. (2010). This technique provides an accurate evaluation of acidosis and is less invasive than rumen pH analysis. Considering that SARA is an acid overload in the rumen, it can be concluded that it may cause an acid-base imbalance in cows.

Management and prevention of SARA

Therapeutic measures

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.

Initially, over a period of ~30 min, 5% sodium bicarbonate solution should be given IV (5 L/450 kg). During the next 6-12 hr, a balanced electrolyte solution, or a 1.3% solution of sodium bicarbonate in saline, may be given IV, up to as much as 60 L/450 kg body wt. Urination should resume during this period.

Procaine penicillin G (22,000 U/kg/day) should be administered IM to all affected animals for at least 5 days to minimize development of bacterial rumenitis and liver abscesses.

Thiamine should also be administered IM to facilitate metabolism of l-lactate *via* pyruvate and oxidative phosphorylation; animals with grain overload also have low concentrations of thiamine in rumen fluid because of increased production of thiaminase by ruminal bacteria. Increasing focus is now paid on prevention rather than treatment.

Feeding and management

Steps to address SARA

1. Ensure proper rumen adaptation especially at calving when shifting cows from the dry group to the lactation group.
2. Control ingredients' palatability.
3. Ensure homogeneity of TMR and proper forage length cut. Keep records of maintenance of mixer (balances, knives).
4. Ensure proper access to the feed bunks and an adequate supply of water.
5. Avoid stressful situations such as moving animals too much between production groups.
6. Keep first calving heifers separated from older cows when possible.
7. Resting area. Ensure good layout, maintenance and bedding. Insufficient lying time will cause cows to change feeding pattern.
8. When formulas or forages are changed a smooth transition is highly advisable.

Ration particle size

To ensure healthy cows and high milk fat production, adequate particle size in their diet is necessary to avoid digestive problems. Cow diets should include fiber and forage to stimulate chewing and maintain rumen health. Particle size separators like the Penn State Particle Size Separator can be used to measure particle size distribution in feeds and monitor changes in forage harvesting and feed mixing. The separator categorizes particles into three groups based on size and identifies which particles will stimulate cud chewing and saliva production, are moderately digestible, or are readily digestible or rapidly removed from the rumen.

Dietary buffers

Buffers are substances that resist changes in pH by neutralizing acids. True buffers prevent acidity from increasing but don't raise pH beyond a certain level. Bicarbonate, Sesicarbonate, Limestone and Bentonite are true buffers. Among these, Magnesium Oxide, Bicarbonate and Sesicarbonate are the best rumen buffers. These feed additives increase the Acetate:Propionate ratio and aid fiber digestion by preventing low rumen pH caused by fermentation and metabolism. Antacids that may be given orally, include Magnesium Hydroxide (cattle: 100-300 g; sheep: 10-30 g) and Magnesium Carbonate (cattle: 10-80 g; sheep: 1-8 g). Antacids should be mixed in ~10 Liters of warm water to ensure adequate dispersion through the ruminoreticular contents. Administration of Activated Charcoal (2 g/kg) orally is believed to protect the ruminoreticular mucosa from further injury by inactivating toxins.

Antibiotics

The effectiveness of antibiotics in controlling lactate production by *S. bovis* and *Lactobacillus* sp. is uncertain. However, the use of Ionophores like monensin has been studied as a method to increase nutrient digestion without affecting DM intake, rumen pH regulation and milk yield.

Stimulation of lactolytic flora

Genetically modified lactolytic bacteria show improved lactate conversion and acid resistance but are not yet available commercially. Dicarboxylic acid supplements like fumarate and maleate can stimulate lactolytic flora.

CONCLUSION

SARA can cause significant economic losses in dairy herds, with reduced milk production, premature culling and increased mortality being the main factors. Diagnosis is challenging due to subtle clinical signs and delayed effects. Early monitoring and recording of disease incidences, clinical signs and Para-clinical parameters is crucial for recognizing SARA and implementing corrective measures. Early diagnosis is important for preventing life-threatening complications and stabilizing dairy farm production.

Conflict of interest

The authors declared that there is no conflict of interest.

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