For Vet Professionals Only

Face2Vet
A Clinical Update

Volume 11 | Issue 7

SUB-ACUTE RUMINAL ACIDOSIS (SARA)

Gut Feeling - Healthy Rumen for a Healthy Ruminant

Dear Vets,

Rumen is an open, self-contained ecosystem in which feed consumed by the ruminant is fermented by rumen microbes to volatile fatty acids and microbial biomass those serve as source of energy and protein for the animals. More fermentation means more acid production and a lower rumen pH. Low rumen pH depresses fibre digestion and leads to metabolic disorders. SARA (Sub-acute Ruminal Acidosis) is a digestive disorder of ruminants occurring due to feeding of excess of rapidly fermentable carbohydrates and inadequate fiber diets leading to decrease in milk production. It is characterized by daily episodes of low ruminal pH and is most common in high yielding dairy cows. In SARA, the low ruminal pH is caused by excessive accumulation of volatile fatty acids (VFAs) without persistent lactic acid accumulation, later restored to normal pH by animal's own physiologic responses. SARA is an economically important condition observed even in well managed herds which results in subsequent deterioration of health and loss of production.

This issue of Face2Vet is oriented towards Sub-acute Ruminal Acidosis (SARA), the metabolic disorder of high yielding dairy cows. We hope that the issue will provide new insights and highlights both preventive and therapeutic approaches for managing SARA. We also welcome your views and tips to minimise SARA or even Ruminal Acidosis that have been adopted by you in clinical practice. We also request you to share your valuable feedback/thought on Face2Vet by scanning the below QR code or else alternatively, reach us *via* email at face2vet@intaspharma.com

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Introduction

India is significantly agro-based economy with 65% rural population. Dairy animal rearing for milk production is taken up as a small scale business occupation by many farmers, since it requires less investment and government is also providing and supporting variety of schemes that focus on milk production.

Cattle frequently suffer from digestive disorders leading to reduction in milk production thereby hampering producer's economics. Diseases which show noticeable clinical signs and severely affect production are quickly attended by animal owners but sub-acute condition does not show any detectable clinical signs and so they go unnoticed and untreated. Hence, economic losses from such disease are not noticed by animal owner. Sub-acute ruminal acidosis (SARA) is one of such disease in bovine.

SARA is a digestive disorder of ruminants occurring due to feeding of excessive rapidly fermentable carbohydrates and inadequate fiber diets leading to decrease in milk production. It is characterized by daily episodes of low ruminal pH and is most common in high yielding dairy cows. SARA is considered sub-acute when the low ruminal pH is caused by excessive accumulation of volatile fatty acids (VFAs) without persistent lactic acid accumulation, later restored to normal pH by animal's own physiologic responses. SARA is an economically important condition observed even in well managed herds which results in subsequent deterioration of health and production loss.

In SARA, cows do not exhibit any typical clinical sign of illness; however, feed intake and milking performance are often depressed. As observed in acute ruminal acidosis, ruminal pH also drops during SARA, but usually lies between 5.2 and 5.6. Differences between acute and sub-acute form is that during acute ruminal acidosis (ARA), pH depression is more severe, concentration of lactic acid in rumen digesta is higher and clinical signs are more prominent.

SARA is considered to be the third major cause for low milk fat syndrome in dairy cows. Apart from reduction in milk fat percentage, SARA also has long term devastating health and economic consequences, which include reduced feed intake and digestibility, diarrhoea, rumenitis, liver abscesses and lameness. Further complications of SARA such as polioencephalomalacia, abomasal illnesses, rumen tympany and epistaxis can lead to death of animal. In addition, it can also influence reproductive output of animals causing delay or inadequate maturation of first wave of post-partum ova and immune- suppression in them predisposing to other illnesses. Therefore, early and timely diagnosis of SARA in dairy herds has a tremendous significance in tackling life-threatening complications and also in stabilizing economy of dairy farms.

Acute Vs Sub-acute Ruminal Acidosis

SARA: More common, results from excessive volatile fatty acids (VFAs) production that exceeds ability of rumen to neutralize and exceeds absorption capacity of ruminal papillae. Rumen pH drops below 5.5 and remains below this threshold for three or more hours within a 24 hour period. Animals generally have mild diarrhoea, lowered dry matter intake and laminitis. Can develop into acute acidosis if pH is not corrected.

Acute Ruminal Acidosis (ARA): Less common, more severe, usually occurs when pH falls below 5. Usually associated to a drastic diet change. Animals have depressed productivity, go of feed, have elevated heart-rate, diarrhoea and may die. Severe lactic acidosis may cause irregular feeding behaviour and gorging, which creates more spikes of acid production.

Table 1: Acute vs sub-acute ruminal acidosis

Parameters	Acute ruminal acidosis	Sub-acute ruminal acidosis
Clinical signs	Present	Absent
Mortality	Yes	No
Rumen pH	Less than 5	5-5.5
Lactic acid	50-120 mm	0-5 mm
Volatile fatty acids	<100 mm	150-225 mm
Lactic acid producing bacteria	Increase	Increase
Lactic acid utilizers	Decrease	Increase
Ciliate protozoa	Decrease	Decrease
Incidence	14 percent generally goes unnoticed	44 percent incidences Reduced dry matter intake and fiber digestion, milk fat depression, laminitis, liver abscesses, or death
Duration	<90 minutes in a day	111-180 minutes in a day

Epidemiology

Accurately determining the prevalence of SARA can be challenging due to the difficulty in diagnosing the condition.

- Incidence of SARA in cattle could be as high as 40 percent.
- SARA prevalence in intensive dairy farming had been estimated around 11-26 percent.
- Transition cows, animals with high dry matter intake (DMI) or on poorly formulated diets and cattle subjected to high degree of variability in their ration and meal pattern were likely to be at a higher risk of developing SARA.
- Holstein Friesian cows with higher milk fat percentage were more susceptible to negative energy balance (NEB) and development of SARA.
- SARA was found to be more prevalent among Jersey breed of cattle.
- High producing breeds of cattle are more vulnerable for the development of SARA.
- Primiparous cows are more likely than multiparous cows to have low ruminal pH, increased ruminal concentrations of volatile fatty acids and perhaps metabolic acidosis.

Cow at risk to develop SARA includes cows in early lactation, primiparous cows and cows grazing or fed with rapidly fermentable low fiber grass. Cows in early lactation are probably due to instability of bacterial population. Cows might be at greatest risk for SARA immediately postpartum due to diminished size and absorptive capacity of rumen papillae following feeding of lower energy density diets during dry period. Cows are apparently at higher risk for SARA in summer due to lack of ruminal buffering caused by heat stress, increased respiratory rate, respiratory alkalosis and low blood bicarbonate concentrations.

Etiology

The etiology encompasses several contributing factors, primarily rooted in nutrition. Key causes include (Fig. 1):

- **High Concentrate Diet and Low Crude Fiber:**

  Diets rich in concentrates and deficient in crude fiber can predispose cattle to SARA.
- **Inadequate Forage Particle Length:**

  When forage particles are too short, it can disrupt normal digestive processes.
- **Limited Buffering Mechanisms:**

  Insufficient buffering capacity in rumen can exacerbate acidosis. This nutritional imbalance can lead to disruptions in balance between lactic acid-producing and lactic acid utilizing bacteria.

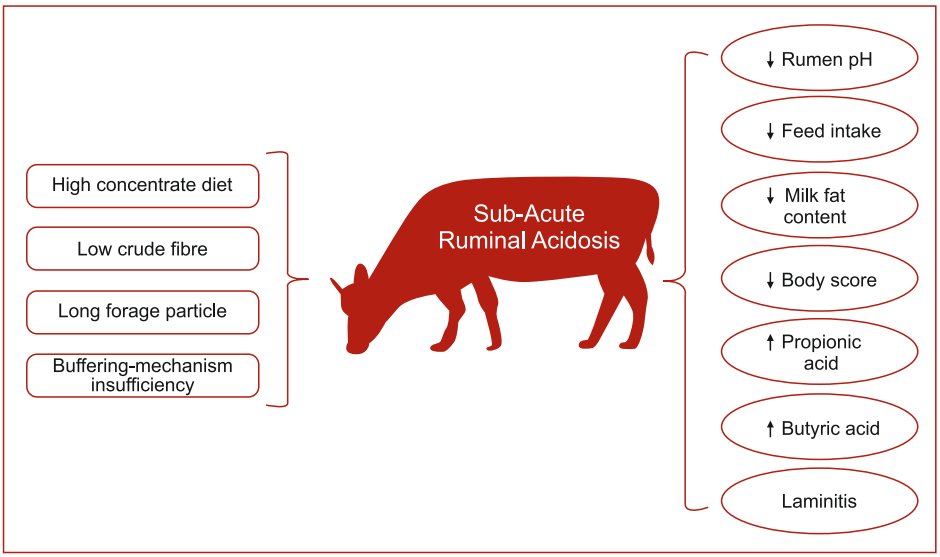

Fig. 1: Etiology and development of SARA

- **Feeding Rapidly Fermentable Carbohydrate**

Feeding quickly fermentable diets to cows recently adjusted to digest and utilize forage-based diet is the most agreed explanation of SARA in dairy herds. (Fig. 2)

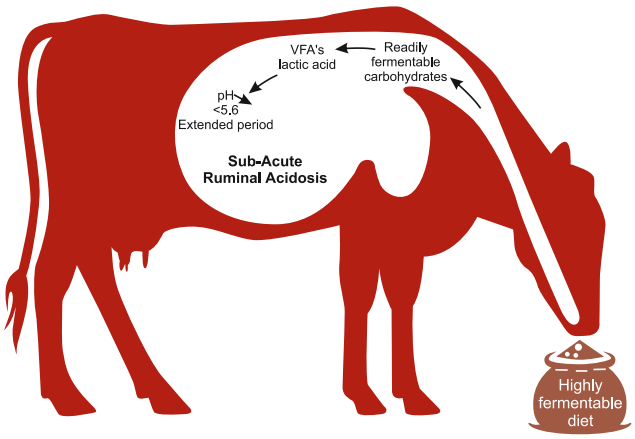

Fig. 2: Feeding rapidly fermentable carbohydrate

- **Inadequate Neutral Detergent Fiber (NDF) in diet**

In ruminant salivary buffer production relies upon length and force of cud chewing. Chewing time is influenced by the substance of coarse fiber in diet. The measure of genuine coarse fiber is evaluated by estimating NDF characterized as the capacity of a feed to invigorate chewing and buffering of salivation in rumen.

As a result lactic acid can accumulate in rumen, potentially triggering conditions like laminitis. There is an increase in volatile fatty acids (VFAs) production, further lowering rumen pH.

Pathogenesis

Low Ruminal pH

Ruminal pH is mainly affected by concentration of organic acids, such VFAs and lactic acids in rumen. Under normal fermentation condition, concentration of VFAs in rumen increases gradually with utilization of carbohydrates by microbiota and rumen pH gradually decreases (Fig. 3).

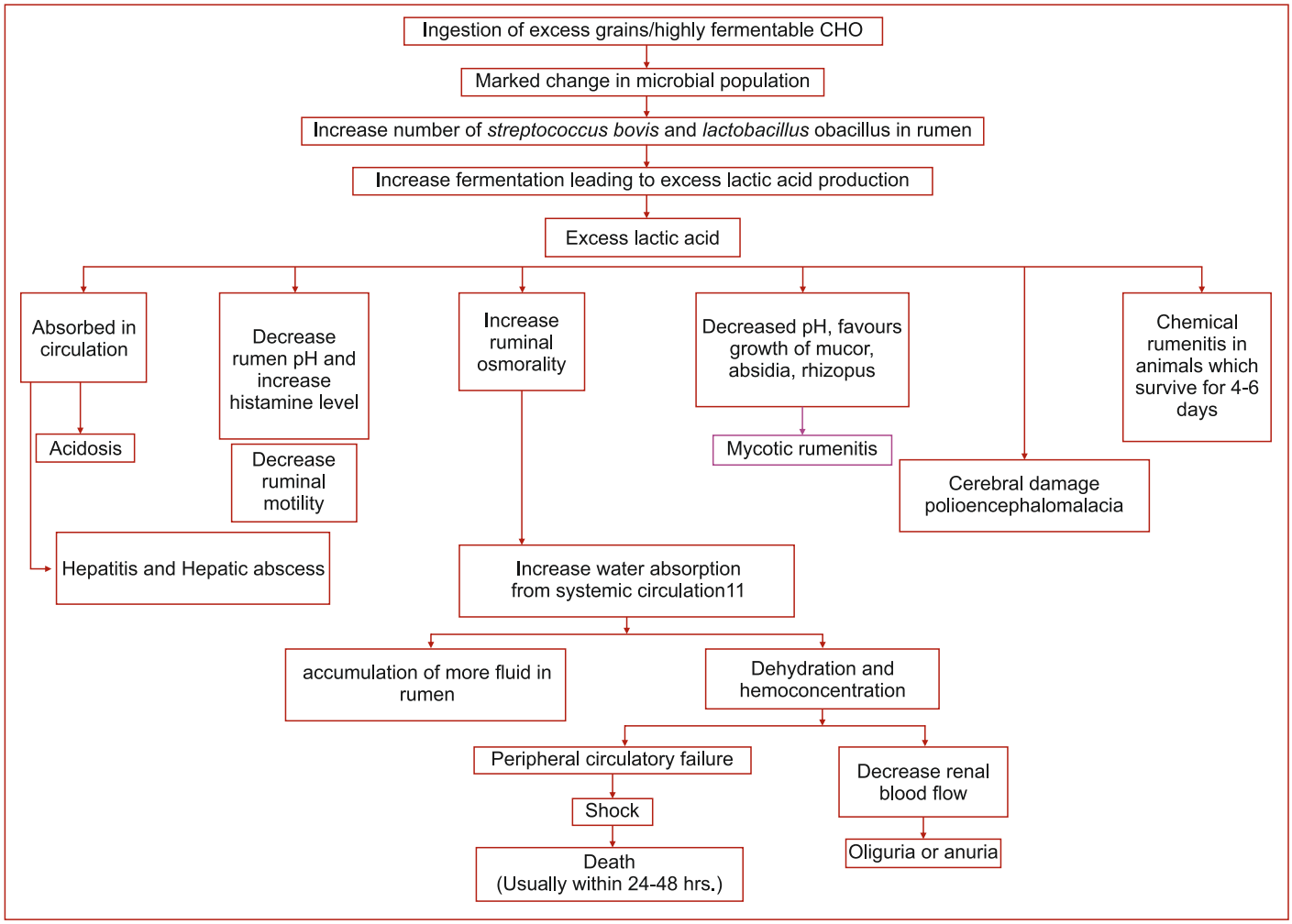

Fig. 3: Detailed pathogenesis

The acidic rumen environment promotes removal of VFAs absorption through the rumen epithelium. In addition, rumination will increase amount of saliva secretion, which contains many alkaline substances. Thus, the rumen pH is rapidly increased to normal physiological levels by absorption and neutralization of VFAs in the rumen and rumen pH is maintained at normal physiological levels before and after each feeding (Fig. 4).

After feeding, rumen microbes ferment carbohydrate intake of dairy cows to produce VFAs and lactate, which reduces rumen pH. Part of temporarily increased VFAs is absorbed by rumen wall and part is neutralized by secreted saliva to keep rumen pH within a certain range.

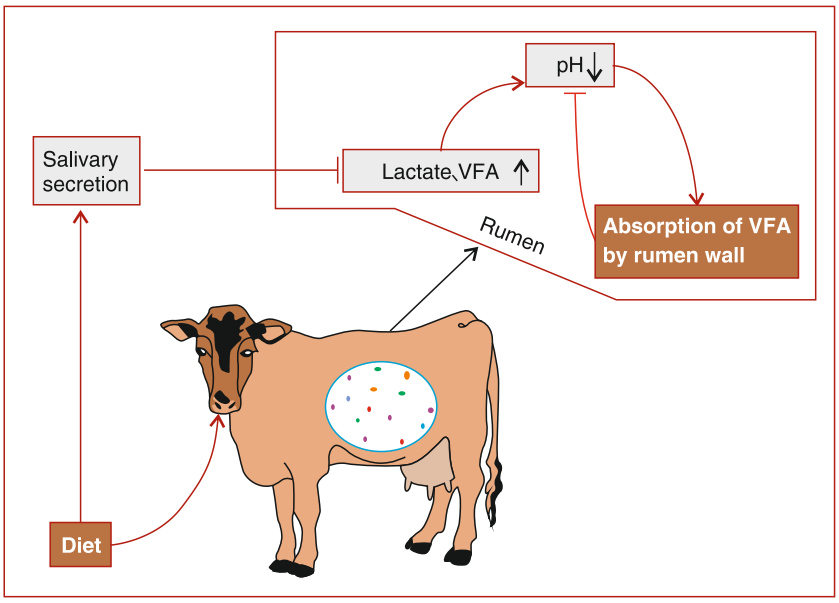

Fig. 4: Physiology of rumen fermentation

When dairy cows eat a large amount of concentrate feed for long time on one hand, excessive carbohydrates will produce excessive VFAs under fermentation of rumen microbiota. On other hand, a low proportion of neutral detergent fiber (NDF) can not stimulate ruminants and does not produce enough saliva to neutralize large accumulation of VFAs in rumen, resulting in rumen pH drops below physiological threshold (Fig. 5).

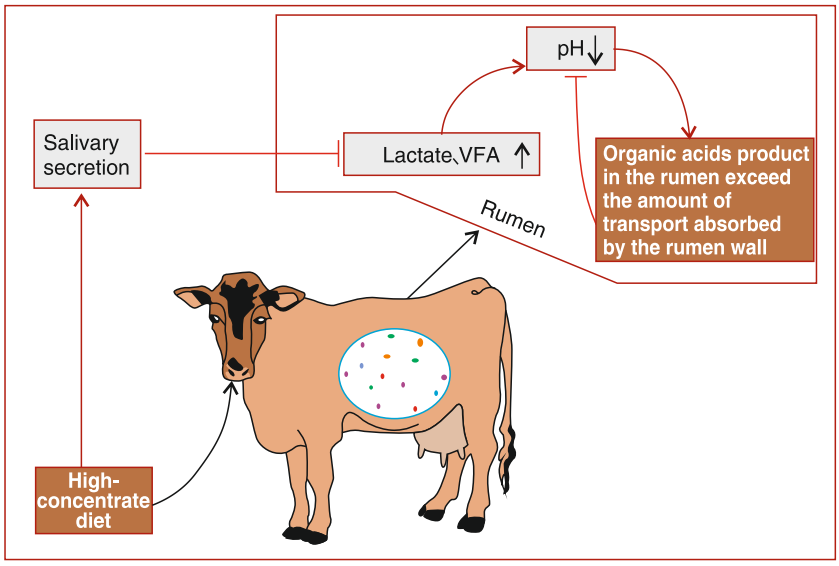

Fig. 5: Pathogenesis of SARA

Once dairy cows ingest too many carbohydrates, the VFA generated in rumen and lactic acid energy substances can not be timely absorbed by rumen wall and amount of saliva secretion is reduced resulting in a pH lower than 5.6-5.8 in rumen and SARA occurs.

Increased Ruminal Lactate

Depression of ruminal pH during SARA is apparently due to accumulation of VFAs and is not caused primarily by lactate accumulation. Nonetheless, frequent measurement of ruminal lactate during the day under conditions associated with SARA will reveal transient spikes of ruminal lactate between about 10 and 40 mm. The exact role of these transient lactate spikes is not known, but they probably contribute to lowered ruminal pH.

Increased Ruminal Valerate

Valerate is produced in rumen by lactolytic bacteria in presence of lactate. High concentrations of ruminal valerate may indicate a prior occurrence of SARA.

Reduced Dry Matter Intake

SARA is self-limiting largely because it depresses DMI. Depressed intake reduces acid production in rumen and allows rapid restoration of normal ruminal pH. Potential causes of intake depression include decreased frequency and amplitude of ruminal contractions, increased ruminal lactic acid concentrations, increased ruminal osmotic pressure, and inflammation of ruminal epithelium.

Cow Behavior

Bouts of ruminal acidosis have little overall effect on measures of cow behavior, such as standing time, lying time or feeding time. Rumination activity decreases slightly during bouts of ruminal acidosis.

Reduced Feed Efficiency

SARA decreases feed efficiency because ruminal cellulolytic bacteria are likely sensitive to low ruminal pH. The extent of reduction in fiber digestibility during SARA is substantial, between about 20% and 25%. Reduced fiber digestibility directly reduces herd profitability and increases the environmental footprint of milk production.

Clinical Sequelae of SARA

Milk Fat Depression

SARA cause milk fat depression; however, this effect is inconsistent and complex. Low ruminal pH apparently causes milk fat depression by inhibiting bacteria responsible for fatty acid bio-hydrogenation in rumen. Incomplete bio-hydrogenation of these fatty acids increases the amount of absorbed trans-fatty acids. Longer term SARA may be necessary before milk fat depression is evident. Other important causes of milk fat depression include overfeeding of unsaturated fats and Monensin supplementation. All of these causes of milk fat depression are interrelated because they all affect the same pathway: incomplete ruminal bio-hydrogenation of fatty acids.

Rumenitis

Ruminal epithelial cells are not protected by mucus (as abomasal cells are), so they may be vulnerable to chemical damage by excessive acidity. Low ruminal pH has been associated with inflammation of ruminal epithelium (rumenitis), which may advance to erosion and ulceration. Maintaining integrity of ruminal epithelium is crucial because it serves as only protective layer between rumen environment and portal circulation. Unfortunately, dramatic structural changes to ruminal epithelium occur within a few days of an acidotic insult. Ruminal acidosis weakens cellular adhesion between cells in the ruminal epithelium, which allows transmural migration of rumen microbes into portal circulation. Weakened adhesion between cells may also increase translocation of endotoxins into bloodstream. Parakeratosis (thickening of stratum corneum of ruminal epithelium) is result of chronic rumenitis. Parakeratosis is clinically important because it decreases VFAs absorption and may predispose affected animals to future SARA episodes.

Diarrhoea

Diarrhoea, frothy feces and presence of mucin casts in feces have been clinically associated with SARA. Mucin or fibrin casts found in feces may be a secondary response to hindgut sloughing of epithelial cells followed by secretion of mucous and fibrin to protect the injured tissue. Changes in fecal consistency are not typically noted in SARA induction studies, even when fecal pH is very low. One possibility is that hindgut protein is more likely culprit when herd wide diarrhoea is observed without an apparent infectious cause. Soluble proteins in hindgut (whether of microbial or dietary origin) can not be absorbed from hindgut. As a result, they may remain osmotically active there and cause diarrhoea. In contrast, organic acids produced by microbial fermentation of carbohydrates in hindgut can be absorbed.

Liver Abscess Formation

Liver abscesses are a common complication of SARA. They are caused by bacterial leakage across damaged ruminal epithelium. Portal blood flow then carries the bacteria to liver, where they establish a new infection that is walled off into an abscess. SARA is associated with a chain of events that liberate Fusiformis necrophorus, Fusobacterium necrophorum, Gram negative, obligate anaerobic bacterium which is the primary etiologic agent of liver abscess. Low ruminal pH damages the surface of rumen wall and causes ulceration of epithelium. Once the ruminal epithelium is damaged, bacteria transported to liver through portal circulation, causing liver abscesses. Infection in liver originates from a necrobacillary rumenitis. Leukotoxin facilitate resistance to phagocytosis. Bacterial emboli from the lesions invade hepatic portal venous system and transported to liver, where they establish infectious foci of necrobacillosis which eventually develop liver abscesses.

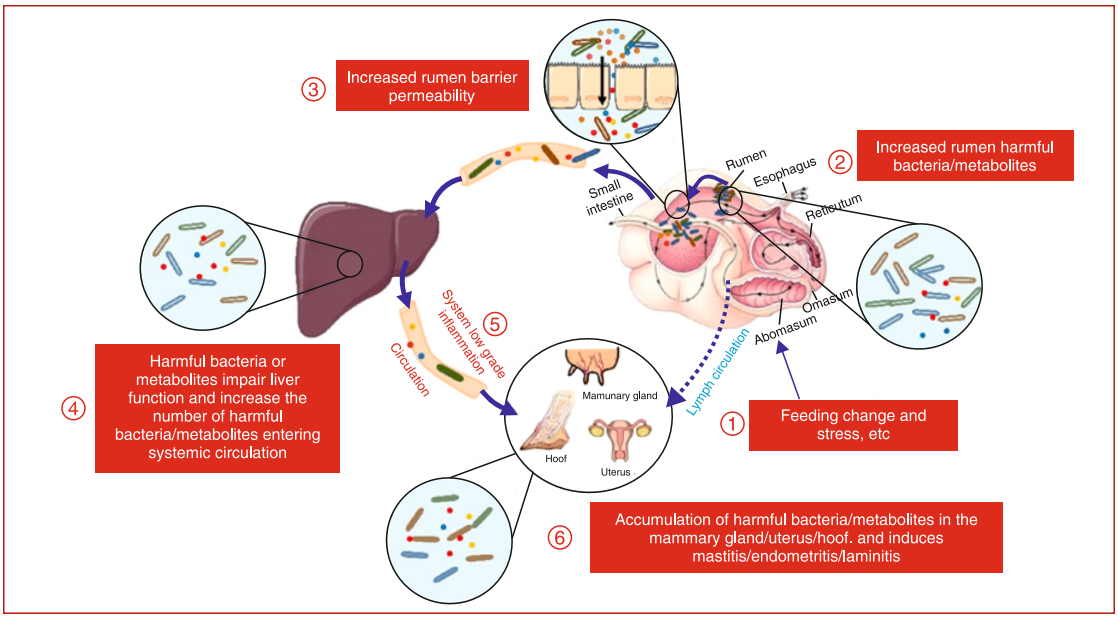

Fig. 6: Mechanism for SARA to increases infectious and metabolic diseases in dairy cows

Lameness

Endotoxins produced by Gram-negative bacteria in rumen migrate to various organs of body by embolism. If they reach hoof, induce a vascular reaction leading to vasoconstriction. Inflammation and pododermatitis follow the course. Laminitis, an aseptic inflammation of hoof dermal layers, is the major source of lameness which is a major health and welfare concern for dairy industry. Especially acute and sub-acute ruminal acidosis is associated with laminitis. Sub-acute or chronic laminitis has been described in SARA affected cows and its clinical signs are discoloration of hoof, sole hemorrhages, sole ulceration and misshapen hooves.

Rumen microbiota disorder causes rumen bacteria and their harmful metabolites to be released into the blood, damages liver function and induces systemic chronic inflammatory response. Then, these harmful substances enter body through circulation of blood and lymph circulation, enter various tissues and organs, increase risk of metabolic diseases, reduce defensive capabilities of tissues and organs and increase risk of infectious diseases.

Rumen bacteria and/or their metabolites can also migrate to mammary gland, uterus and other organs, affect immune function and increase susceptibility to infectious diseases (Fig. 6).

Diagnosis of SARA

Reticulo-Ruminal pH

The easiest reliable technique to detect sub-acute ruminal acidosis is to monitor reticulo-ruminal pH continuously. Ruminal pH is lowest till 5-8 hours after feeding. More accurate estimation of pH can be done by collecting ruminal samples by various methods viz. oral intubation using a probe and ruminal pump, rumenocentesis, intraluminal sensors, evaluation of dung for presence of bubbles and lipopolysa-ccharides, measurement of ruminal thickness and blood acid-base analysis.

Manure Evaluation

Increase in grain content in diets, which may induce SARA, can also result in more dietary nutrients by passing the rumen and reaching hindgut. Excessive hindgut fermentation then changes consistency and appearance of manure. Hence, manure observation may be used as a diagnostic tool to evaluate rumen functionality. Watery and foamy manure indicates the abnormal fermentation in hindgut and mucin casts in manure suggests damage of gut epithelium.

Fecal Lipopolysaccharide (LPS)

Feeding high-grain diets to induce SARA in dairy cows has been associated with increase in concentration of lipopolysaccharide endotoxin originating from Gram-negative bacteria in feces. Induction of SARA increases LPS concentration in feces. Dairy farms with low dietary NDF had higher fecal LPS about 2 times greater than farms with a high dietary NDF.

Prevention and Treatment

SARA being a silent condition, displays delayed symptoms and hence makes prevention difficult. Nevertheless, adequate nutrition and adaptation of microflora to feed are crucial to preventing SARA incidences in herd. Physically effective fibre (NDF>1.18) in diet stimulates saliva production, and hence ruminal buffering, assisting in maintaining rumen pH. Exogenous preventive measures like buffers and direct-fed microbials (DFMs) also provide an effective tool for monitoring and preventing SARA. Mode of action and effects of specific dietary buffers and DFMs are described below:

Saliva- A Natural Buffer

In ruminants, as in other animals, saliva not only aids in the swallowing of feed but also plays a crucial role in feed intake and utilization. Saliva acts as a buffering agent and a nitrogen source for the microbial population in rumen. It also serves as a preventive agent against bloat. This versatility underscores the importance of salivation in ruminants. It is estimated that ruminants produce 117 to 183 kg of saliva per day.

Effects of exogenous buffers on rumen health

- Decline in ruminal urea concentration
- Increased flow of undegraded starch from rumen
- Greater microbial utilization of ammonia with an increased level of energy supplied
- Increase water intake, stabilize rumen pH
- Buffers enhance cellulose digestion and increase rumen turnover
- Buffers improve protein solubility; hence microbial protein synthesis is better
- Buffers increase the completeness of bio-hydrogenation and decrease formation of intermediates. They also increase acetate and decrease propionate.
- Buffers increase milk protein content due to better utilization by microbes. Cationic salts improve lactation performance by improving ruminal buffering ability, blood pH, rumen microbial synthesis and bio- hydrogenation in rumen.

Mode of action and effects of specific dietary buffers is described below.

1. Bicarbonates

The dissociation constant of sodium bicarbonate is 6.25 and they have a short half-life. Bicarbonates have a significant buffering action, which compensates for saliva and increases the DMI. Buffers increase HCO_3- concentration in ruminal fluid and shift equilibrium towards CO_2, decreasing free H+ ion concentration and increasing pH. By adding dietary buffers there is an increase in the proportion of acetate, while the molar proportions of propionate and butyrate remain the same. For every mole of VFAs that leaves rumen, one proton is added. Bicarbonates neutralize protons and increase the dilution rate of rumen. Bicarbonates increase voluntary water intake by animals, decreasing rumen osmolality. Hence the flow of starch increases, preventing its accumulation. This assists in hindering growth of lactobacilli in the rumen. Increased bacterial nitrogen flow increases bacterial protein synthesis, while the rise in pH improves protein utilization by increasing its solubility.

2. Potassium Carbonate

It has a similar action mechanism as Sodium bicarbonate. Nonetheless it is a more potent neutralizing agent. It is generally preferred to alleviate incidences of fat milk depression, as it favors the predominant pathway of milk fat dehydrogenation. Milk fat increases by 24 percent on a matter basis in cows fed with Potassium carbonate, while the milk yield declines. Cows with their Potassium carbonate as top dressing have more forage intake than cows fed with Sodium bicarbonate.

3. Magnesium Oxide

Magnesium oxide is yet another effective and commonly used buffer in ruminants. It is generally preferred top dressing over feed in combination with Sodium bicarbonate. It increases uptake of blood metabolites like plasma acetate and triglycerides by mammary gland hence raising the fat content. Its efficacy depends on its particle size.

4. Sodium Sesquicarbonate

It is a double salt of Sodium bicarbonate and Sodium carbonate, having a pH of 9.9, as opposed to bicarbonates, which have a pH of 8.4. Hence the acid-neutralizing capability is higher than bicarbonate, with the added advantage of being cost-effective. Dietary supplementation of sesquicarboante decreases the molar proportions of butyrate and valerate. It improves milk fat and 4% FCM yield.

5. Maximizing Ruminal Buffering

Ruminant animals have a highly developed system for buffering organic acids produced by ruminal fermentation of carbohydrates. Although the total effect of buffering on ruminal pH is relatively small, it can still account for margin between health and disease in dairy cows fed large amounts of fermentable carbohydrates. Endogenous buffers are produced by cow and secreted into rumen with saliva flow during eating and ruminating. Endogenous buffering is maximized when cows are provided adequate long particles in diet, as described above. Dietary buffering represents the inherent buffering capacity of diet and is largely explained by dietary cation-anion difference (DCAD). Diets high in Na and K relative to Cl and S have a higher DCAD and promote higher ruminal pH. Buffers such as $NaHCO_3$ and $K2CO_3$ are added to diets to increase DCAD beyond the inherent DCAD of diet ingredients. **Bufzone** is an combination of metabolic booster, yeast and ruminal buffers for management of ruminal acidosis including sub-acute ruminal acidosis.

6. Direct Fed Microbials and Yeast

DFMs and yeast prevent lactate accumulation and allow better fiber digestion by improving the reducing conditions of rumen and fibrinolytic bacteria's stimulation. Conversion of lactate to propionate is enhanced, and ruminal pH is stabilized showed that three different organisms (*Enterococcus faecium, Lactobacillus plantarum, Saccharomyces cerevisiae*) administered at 105 cfu/ml stabilized rumen acidity and improved digestion. Yeast supplementation may reduce the risk for SARA, particularly during abrupt changes from high-forage to high-grain diets. Mechanisms proposed for this benefit are mainly focused on optimizing fiber digestion.

Treatment

- Correction of ruminal pH by giving alkalizer e.g. Sodium bicarbonate, Magnesium hydroxide and Magnesium oxide orally.
- Ruminal buffering with **Bufzone** @200 g daily for 3-4 days.
- Pre-probiotics such as **Ecotas bolus** @2 boli daily for 4 days.
- Correction of fluid and electrolyte imbalance by giving isotonic Sodium bicarbonate (1.3%) and balanced electrolyte solutions (Ringer's solution). In severe acidosis – Sodium bicarbonate @ 5 liter IV in 30 min. followed by isotonic balanced fluids @ 150 ml/kg body weight for 6-12 hours. Hydration of animal with Inj. **Intalyte** @ 500-2000 ml IV based on degree of dehydration.
- Oral electrolytes **(Intalyte oral)** @ 30 g in 1 litre of water twice daily.
- Use of oral antibiotics to control growth of bacteria e.g. Sulphadimidine **(Bol. Pabadine)** @1 boli / 50 kg b. wt., Levofloxacin + Ornidazole **(Lornitas)** @ 5 ml / 20 kg b. wt.

- Oral ruminotorics to restore motility of rumen. e.g. **Rumentas** 2 bolus orally daily for 2-3 days twice daily.
- Antihistaminics and corticosteroids to prevent shock. e.g. **Anistamin** @ 5-10 ml IM, Dexamethasone @ 0.04 mg/kg IM.
- Cud transplantation @ 10-20 liter to replenish ruminal microflora.

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