Sub-acute ruminal acidosis: Understanding the pathophysiology and management with exogenous buffers

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Abstract
Sub-acute ruminal acidosis (SARA) is an economically important clinical condition and contributes to loss of farm returns, second only to mastitis. It is more prominent in cows in their early and mid-lactation, having peak milk yield and considerably high dry matter intake. SARA detection in a dairy farm is difficult as it does not present any pathognomonic symptoms, and the manifestation of clinical signs is delayed. SARA’s characteristic feature is the occurrence of daily fluctuations of pH when the pH drops to the range of 5.2 to 6 for a considerable period due to the accumulation of volatile fatty acids in the rumen. Grain-based diets, which have higher proportions of non-structural carbohydrates, high-quality fermentable forages like legumes, and lack of physically adequate dietary fibre (peNDF), are the significant causes of SARA. SARA consequences include the inflammation of rumen mucosa and several other organs and long-term health and economic losses like reduced feed intake reduced fibre degradability, drop in milk yield and milk fat, damage to the gastrointestinal tract, laminitis, liver dysfunctions, and lameness. SARA can be prevented and treated by the right combination and judicious use of exogenous dietary buffers like sodium bicarbonate, magnesium oxide, and direct-fed microbial like yeast. This review aims to provide a gist of the recent literature available on the pathophysiological aspects, indicators, detection techniques, prevalence, and preventive measures for SARA, including the mechanism of action and utility of the commonly used dietary buffers and direct-fed microbials.

Keywords: sub-acute ruminal acidosis, diagnosis, dairy cattle, rumen pH, buffers

Introduction
India ranks first in the world in terms of milk production. The annual milk production was 176.3 million tonnes for the year 2017-18 [1]. Nonetheless, the per capita production of milk is still far below the world average. Having the world’s largest herd, the country has enormous prospects of transforming the dairy sector into a humongous enterprise, provided the livestock is fed with nutritionally adequate diets. Current practices at intensive dairy systems advocate concentrate feeding in order to elevate the plane of nutrition. Thus, cattle are fed high starch and low fibre [2] to increase milk production [3, 4]. Ruminants are adapted to digest mainly forage diets [5]. Any alteration in the physical form or effectiveness of the diet, e.g. smaller forage particle size or fine grinding of grain, decreases ruminal pH, giving rise to sub-acute ruminal acidosis (SARA) [6]. Thus, concentrate feeding affects rumen health. The more serious concern about the decline in ruminal pH is its sub-clinical nature, making it more difficult to detect and cure, thus causing considerable losses to the animal and the farm productivity. SARA has been a significant menace to Indian dairy farms over the years, second only to mastitis regarding the monetary losses caused [7]. It alters fermentation patterns, reduces dry matter intake, milk yield, fat content, and consequently, farm profitability [8]. Garret et al. [9] reported that approximately 19% of cows in their early-lactation period and 26% of cows in their mid-lactation period are affected with metabolic acidosis in the United States. The figures remain similar even after 23 years [10], as SARA’s diagnosis is quite challenging due to its sub-clinical nature. Early lactation cattle are prone to acidosis due to their energy-dense diet and unstable microflora [11], and mid-lactation cows suffer as they have a higher dry matter intake [12].
Acute and sub-acute ruminal acidosis

The significant difference between acute and sub-acute ruminal acidosis lies in their duration of onset of symptoms. While acute acidosis is a grave condition with a poor prognosis, the number of incidences in dairy cattle is relatively minor. It is not a significant concern in feedlot cattle.\(^{[13]}\)

Table 1: Differences between acute and sub-acute ruminal acidosis (Plaizier et al., 2008\(^{[14]}\); Calsamiglia et al., 2008\(^{[7]}\))

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Acute acidosis</th>
<th>Subacute acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Mortality</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Rumen pH</td>
<td>&lt;5</td>
<td>5-5.5</td>
</tr>
<tr>
<td>Lactic acid</td>
<td>50-120 mM</td>
<td>0-5 mm</td>
</tr>
<tr>
<td>Volatile fatty acids</td>
<td>&lt;100 mm</td>
<td>150-225 mm</td>
</tr>
<tr>
<td>Lactic acid producing bacteria</td>
<td>Increase</td>
<td>Increase</td>
</tr>
<tr>
<td>Lactic acid utilizers</td>
<td>Decrease</td>
<td>Increase</td>
</tr>
<tr>
<td>Ciliate protozoa</td>
<td>Decrease</td>
<td>Decrease</td>
</tr>
<tr>
<td>Incidence</td>
<td>14% incidences</td>
<td>Reduced dry matter intake and fiber digestion milk fat depression, laminitis, liver abscesses, or death</td>
</tr>
<tr>
<td>Duration</td>
<td>&lt;90 minutes in a day</td>
<td>111-180 minutes in a day</td>
</tr>
</tbody>
</table>

pH and buffering of rumen

The ruminal pH is about 6.2-6.8, which fluctuates by ±2.5 points depending on the type and frequency of feeding.\(^{[15]}\) The major contributors to the buffering action of rumen liquor are phosphate-bicarbonate buffer with urea and mucous secreted in the saliva. Cattle produce about 200-300 L of saliva daily, which has about 100-140 mEq of bicarbonates. It constitutes about 30-40% of the buffering capacity of the rumen.

The rumen papillae, mainly adopted to absorb and transport VFA from the rumen to the bloodstream, may erode. Thus, gram-negative bacteria may leak into the systemic circulation, causing septicemia and giving rise to various disorders like ruminitis, rumen parakeratosis, metabolic acidosis, lameness, hepatic abscession, pneumonia, and death.\(^{[18]}\)

\[ \text{HPO}_4^{2-} + \text{H}_3\text{O}^+ \leftrightarrow \text{H}_2\text{PO}_4^- + \text{H}_2\text{O} \quad \text{(1)} \]

\[ \text{HCO}_3^- + \text{H}_3\text{O}^+ \leftrightarrow \text{H}_2\text{CO}_3 + \text{H}_2\text{O} \leftrightarrow \text{CO}_2 + 2\text{H}_2\text{O} \quad \text{(2)} \]

Equations (1) and (2)\(^{[16]}\) summarize the buffering mechanism of saliva and rumen.

By increasing the amount of fermentable carbohydrates by grain feeding, pH drops below the expected levels by accumulating lactate. The ruminal pH pattern displays a biphasic curve with a decline in pH immediately after feeding.
The pH achieves a minimum value 2 to 3 h after feeding and increases continuously until the next feeding [19]. However, the pattern of pH is much more stable and higher (=6.5) when the animal is maintained on ad-libitum hay, as opposed to concentrating feeding [15].

Rumen microflora: The rumen milieu is a complex ecosystem. Protozoa engulf bacteria to salilate their nitrogenous needs [20]. This bacterial uptake drops to nil if the pH falls to 5. The protozoal population is completely demolished at this pH [21]. A decline in ruminal pH also drastically reduces the population of cellulolytic bacteria, as they are adapted to grow in near-neutral pH [22, 23]. The accumulation of lactic acid favors lactobacilli’s growth, which carries out fermentation and further worsens the rumen milieu [24].

Clinical signs of SARA: Although there are no confirmatory signs for SARA, and it is called ‘silent sickness’ of the herd [25], some symptoms can be considered indicative of the condition.

- Decreased voluntary DMI
- Losing of body condition and emaciation
- Reduction in milk yield and fat
- Rumenitis–caudal vena cava syndrome complex,
- Liver abscesses
- Lameness [36].

Diagnosis of SARA: The easiest reliable technique to detect sub-acute ruminal acidosis is to monitor reticulo-ruminal pH [27] continuously. The ruminal pH is lowest till 5-8 h after feeding TMR. 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 the presence of bubbles and lipopolysaccharides, measurement of ruminal thickness, and blood acid-base analysis [10].

Clinical Sequelae of SARA
1. Metabolic acidosis: It is unclear that lactate accumulates in the rumen and has a metabolic acidosis role [28]. However, it induces inappetence in early lactation periods due to high dry matter intake [29]. Due to ruminal acidosis, cellular functions are impaired, and VFA concentration rises in the peripheral circulation, which affects insulin secretion [30], reduced phagocytic activity [31], reduced neutrophil migration [32], increased cortisol secretion [33]. Long-term acidosis may lead to immunosuppression and a decrease in milk production [34].

2. Rumenitis: Accumulation of VFA's like butyrate, propionate and lactate may be involved in the pathogenesis of rumenitis. Parakeratosis results from acute acidic conditions, which also affect the long-term absorption capacity of the ruminal mucosa, making it susceptible to the entry of gram-negative bacteria like Fusobacterium necrophorum. The bacteria might also migrate to the liver as emboli, leading to rumenitis liver abscess complex [35].

3. Abomasal displacement: Increased flux of ruminal gases and VFA between abomasum and rumen may lead to abomasal displacement, which is complemented by the fact that low functional fiber in the ration also causes the same [35].

4. Laminitis: Endotoxins produced by gram-negative bacteria in the rumen migrate to various organs of the body by embolism. If they reach the hoof, induce a vascular reaction leading to vasoconstriction. Inflammation and pododermatitis follow the course [36].

5. Bloat: The release of macromolecules like mucopolysaccharides and endotoxins unknown macromolecules from gram-negative bacteria leads to the formation of a static foam, leading to a drop in pH and accumulation of gas [37].

6. The decrease in milk fat: An increase in the concentration of protons and a decline in the proportion of acetate in the rumen leads to the incomplete biohydrogenation of unsaturated fats to various intermediates. Hence the final fat yield is decreased [38].

Prevention and treatment: SARA being a silent condition, displays delayed symptoms and hence makes the prevention difficult. Nevertheless, adequate nutrition and adaptation of microflora to the feed are crucial to preventing SARA incidences in the herd [39]. Physically effective fibre (pNDF>1.18) in the diet stimulates saliva production, and hence ruminal buffering, assisting in maintaining rumen pH [40]. Exogenous preventive measures like buffers and direct-fed microbials also provide an effective tool for monitoring and preventing SARA. The mode of action and effects of specific dietary buffers and DFMs are described below:

Effects of exogenous buffers on rumen health
- The decline in ruminal urea concentration
- The increased flow of undegraded starch from the rumen
- Greater microbial utilization of ammonia N 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 [41]
- Buffers increase the completeness of biohydrogenation & decrease the formation of intermediates. They also increase acetate and decrease propionate [42]
- 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 biohydrogenation in the rumen [43].

The mode of action and effects of specific dietary buffers is described below.

Bicarbonates: The dissociation constant of sodium bicarbonate is 6.25, and they have a short half-life [44]. Bicarbonates have a significant buffering action, which compensates for saliva and increases the DMI [45]. Buffers increase the HCO₃⁻ concentration in ruminal fluid and shift the equilibrium towards CO₂, decreasing the free H⁺ ion concentration and increasing the 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 VFA that leaves the rumen, one proton is added (Fig. 2). Bicarbonates neutralize protons & increase the dilution rate of
Bicarbonates increase the voluntary water intake by the animals, decreasing the rumen osmolality. Hence the flow of starch increases, preventing its accumulation. This assists in hindering the growth of lactobacilli in the rumen.[47] Increased bacterial nitrogen flow increases bacterial protein synthesis,[48] while the rise in pH improves protein utilization by increasing its solubility.[49] Buffers also improve nitrogen retention by increased nitrogen retention.[50]

**Potassium carbonate:** It has a similar action mechanism as sodium bicarbonate. Nonetheless it is a more potent neutralizing agent.[51] 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% on a matter basis in cows fed with potassium carbonate, while the milk yield declines.[52] Cows with their potassium carbonate as top dressing have more forage intake than cows fed with sodium bicarbonate. In a study by Zali et al.,[53] two new buffers called HBNa and HBK containing sodium and potassium carbonates respectively, were developed and evaluated for milk yield in Holstein Friesian cows. It was observed that there was no difference in milk yield and 3.5% FCM between the groups fed with two different buffers. Milk fat and protein%, calcium levels in cows fed with potassium carbonate buffer were higher than the sodium-based buffer. The new commercial buffer HBK proved to give the best results in milk composition at the level of 6% of DMI.

**Magnesium oxide:** MgO is yet another effective and commonly used buffer in ruminants. It is generally the preferred top dressing overfeed, in combination with sodium bicarbonate. It increases the uptake of blood metabolites like plasma acetate and triglycerides by the mammary gland, hence raising the fat content. Its efficacy depends on its particle size. The dose rate is 45-90 g/d. The preferred ratio is 2:3:1 with NaHCO₃.[54] In a comparative evaluation of MgO and soda bicarb by Bach et al. (2018)[55], it was found that 0.4% MgO can sustain pH fluctuations in rumen more than 0.8% soda bicarb when the animal is subjected to a high concentrate challenge.

**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 sesquicarbonate decreases the molar proportions of butyrate and valerate. It improves milk fat and 4% FCM yield. Although in an in-vitro study by Sharma et al.,[56] it was found that there was no change in in vitro DM digestibility, ammonia nitrogen, and molar proportions of VFAs. In-vivo studies suggest differently.[57]

**Zeolite:** It has a high attraction for water & cations like K⁺, NH₄⁺, Ca²⁺, and Mg²⁺, which are reversibly bound. When these ions are released, fermentation is facilitated. Osmotic activity regulates pH by buffering against hydrogen ions of organic acids. It also improves nitrogen utilization. A comprehensive summary of the effect of different buffers by various researchers over the years is presented in table 2.

### Table 2: Effect of different buffers on different diet patterns by various researchers

<table>
<thead>
<tr>
<th>Diet</th>
<th>Buffer and dose rate</th>
<th>Effect on milk fat%</th>
<th>Effect on milk yield</th>
<th>Effect on milk protein%</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn silage</td>
<td>180g NaHCO₃</td>
<td>0.25% Increase</td>
<td>0.12% increased</td>
<td>No change</td>
<td>(Fishert and Mackay, 1983)[58]</td>
</tr>
<tr>
<td>68% concentrate</td>
<td>1.5% NaHCO₃</td>
<td>0.45% increase</td>
<td>3.5% FCM was higher</td>
<td>No effect</td>
<td>(Xu et al., 1994) [59]</td>
</tr>
<tr>
<td>Rotational grazing</td>
<td>1.25% NaHCO₃</td>
<td>No change</td>
<td>No change</td>
<td>No change</td>
<td>(Rearte et al., 1984) [59]</td>
</tr>
<tr>
<td>Hay crop silage 70% roughage + 30% concentrate</td>
<td>0.7% NaHCO₃</td>
<td>0.09% increase</td>
<td>FCM decreased by 0.3 kg</td>
<td>0.04% rise</td>
<td>(Stokes et al., 1985) [60]</td>
</tr>
<tr>
<td>Corn silage 40% +60% concentrate</td>
<td>1% Bicarbonate 1% sesquicarbonate</td>
<td>0.15% increase</td>
<td>4% FCM higher for sesquicarbonate</td>
<td>No effect</td>
<td>(Ghorbani et al., 1989) [61]</td>
</tr>
<tr>
<td>23.1% starch</td>
<td>1% NaHCO₃</td>
<td>No change in Milk fat or milk yield</td>
<td>--</td>
<td>from 5.9-6.2</td>
<td>(Bougouin et al., 2018) [62]</td>
</tr>
<tr>
<td>Concentrate challenge</td>
<td>90 g/d Acid buff + 180 g /d NaHCO₃</td>
<td>5.42 vs control (5.19) vs (5.26)</td>
<td>5.42 vs control (5.19) vs (5.26)</td>
<td>--</td>
<td>(Beya et al., 2007) [65]</td>
</tr>
</tbody>
</table>

**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 [63].

Nocek, and Kautz (2006) [64] showed in a study that three different organisms (Enterococcus faecium, Lactobacillus plantarum, Saccharomyces cerevisiae) administered at 10^6 cfu/mL stabilized rumen acidity and improved digestion.

Conclusions

SARA's economic losses are relatively high (approximately Rs.20,000 per cow per lactation). So, it poses a significant threat to the dairy industry if not appropriately treated. Exogenous dietary buffers have been proven to help overcome acidosis, though the results are not consistent. The type of buffer, its dose, and the type of diet are the major factors affecting the buffers’ efficiency. Yeast may be an effective alternative for bicarbonate buffers. The success of an effort in preventing SARA depends on the coordinated efforts between nutritionists and clinicians.

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