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Calcareous Marine Algae, Sodium Bicarbonate and Magnesium Oxide Mixture for Curing Lactic Acidosis in Goats

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Abstract

This study was designed to evaluate the therapeutic efficacy of calcareous marine algae (AcidBuf), sodium bicarbonate and magnesium oxide mixture for the treatment of lactic acidosis in goats. In this study, a total of 57 goats were diagnosed with lactic acidosis and subjected to a therapeutic trial of 4g AcidBuf plus 10 g Na-bicarbonate plus 4g magnesium oxide/head once daily for three days. Before and 72 h after treatment, a clinical examination and blood and ruminal fluid samples were taken. Within 72 h of the therapeutic trial, the clinical signs of lactic acidosis gradually improved and resolved, and the rumen pH increased significantly from 5.16±0.460 to 6.54±0.246. Rumen contraction, rumen fluid physical properties (color, odor and consistency), protozoal motility and ruminal microflora differed significantly toward the normal of healthy goats. Regarding haemgasometry and hematobiochemical parameters, a highly significant decrease in Hb, HCT%, TLC, ALT, AST, creatinine and lactate values and a highly significant increase in blood pH, HCO_3^-, pCO_2, serum urea, total antioxidant capacity (TAC) and β-hydroxybutyric acid (βHBA) were recorded compared to the pre-treatment values. The current study concluded that calcareous marine algae, sodium bicarbonate and magnesium oxide mixture is very helpful in minimizing the severity of ruminal lactic acidosis in goats and could be used as the therapy of choice.

KEYWORDS
Calcareous marine algae, Goat, Ruminal acidosis, Therapeutic efficacy

INTRODUCTION

Ruminal lactic acidosis is a common managemental disease of goats (Koondhar and Khaskheli, 2021), may be fatal in severe cases in less than 24 h, and is clinically characterized by decreased rumen motility or stasis, a severe drop in rumen pH, dehydration, acid base imbalance (Nithin *et al*., 2020) and caused by the sudden ingestion of large amounts of highly and rapidly fermentable carbohydrates without prior adjustment (Ribeiro *et al*., 2020). The fermentation of these carbohydrates in the rumen by amylolytic bacteria mainly Streptococcus bovis and Lactobacillus spp. causes an increase in the production and accumulation of volatile fatty acids and lactic acid causing a critical drop of rumen pH below the optimal levels (Jaramillo-López *et al*., 2017).

When microbial production of lactate exceeds its utilization, lactate is absorbed into the blood stream and causes systemic changes that can be assessed by measuring certain hematobiochemical parameters, in particular those of liver, kidney and acid base balance which provide useful information for the diagnosis, prognosis and treatment of rumen acidosis (Sabes *et al*., 2017).

Treatment of lactic acidosis is difficult, and its recovery depends on the severity of the condition (Karapinar *et al*., 2008) focusing mainly on correcting rumen acidosis and acid base imbalance, inhibiting further lactic acid production, and restoring a normal rumen microenvironment (Anderson and Rings, 2008; Koondhar *et al*., 2020a,b).

Agents capable of neutralizing acids can be used to treat lactic acidosis. Numerous studies summarized the effectiveness of sodium bicarbonate in increasing rumen pH (Erdman, 1988; Hu and Murphy, 2005; Udainiya *et al*., 2020) but due to its rapid solubility, it is short lived in the rumen (Van Soest, 1994); Magnesium oxide (MgO) increased the rumen pH, but the effect developed slowly and was only relevant after 24 h of treatment (Calsamiglia *et al*., 2012) and the optimal way to apply MgO in the diet is one part MgO with two to three parts sodium bicarbonate (Hutjens, 2003). Calcareous marine algae is a natural product produced from calcified seaweed "*Lithothamnion calcareum*", contains high levels of calcium, magnesium and essential trace elements, has positive effect on rumen pH and has the potential to prevent the onset of lactic acidosis (Cruywagen *et al*., 2015). Therefore, we hypothesize that calcareous marine algae, sodium bicarbonate and MgO mixture might have curative activity against lactic acidosis. Therefore, current study was planned to assess the efficacy of this mixture for curing lactic acidosis.

MATERIALS AND METHODS

Ethical approval

All the procedures used in the present study were reviewed and approved by the Animal Welfare and Research Ethics Com-

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mittee, Faculty of Veterinary Medicine, Zagazig University, Egypt.

Location of the study

This study was conducted in the Veterinary Teaching Hospital, Faculty of Veterinary Medicine, Zagazig University, Egypt over a period of 6 months from November 2021 to April 2022.

Experimental procedures

A total of 57 goats (regardless of age, sex and breed) were admitted to the Veterinary Teaching Hospital, Faculty of Veterinary Medicine, Zagazig University, Egypt with a history of accidental ingestion of large amounts of highly fermentable carbohydrates, clinical manifestations of anorexia, abdominal distension and diarrhea were selected and screened for the presence of ruminal acidosis based on low rumen pH. Goats with lactic acidosis were subjected to the therapeutic trial using a mixture of 4g AcidBuf plus 10 g Na bicarbonate plus 4g Mg oxide/head once daily for three days. The response of therapeutic attempt was evaluated 72h after the treatment on the basis of improvement of their clinical state, the properties of ruminal fluid and certain hematobiochemical parameters.

Clinical examinations

All goats underwent a thorough clinical examination according to the methods described by Smith and Sherman (2009) before and 72 h after treatment. Data on body temperature, respiratory rate, heart rate and rumen contraction were recorded.

Sampling

Blood and ruminal fluid samples were collected before and 72 h after treatment.

Blood samples

Jugular vein puncture was used to collect four blood samples from each case; the first without anticoagulant for the separation of serum and the determination of AST (aspartate aminotransferase) and ALT (alanine aminotransferase) spectrophotometrically according to the method described by Reitman and Frankel (1957), creatinine according to the method described by Thomas (1992), serum urea according to the method described by Patton and Crouch (1977), βHBA, using commercial spectrophotometric kits (Pointe Scientific, Inc. USA) according to Koch and Fledbruegge (1987) and TAC, using a commercial test kit (Sigma-aldrich, USA) according to Miller and Evans (1997), the second on EDETA blood collection tubes for complete blood count (CBC), using an automated haematological cell counter, the third on vacuum-heparinized tubes with freeze-dried lithium heparin for blood gas analysis; Measurements of blood pH, carbon dioxide partial pressure ($pCO_{2)}$, bicarbonate concentration (HCO_3^-), were performed within half an hour after collection, using a blood gas analyzer (RAPIDlab 348EX SIEMENS blood gas system), and the fourth on tubes containing sodium fluoride for plasma separation and determination of plasma L-lactate according to Burtis (1999).

Ruminal fluid samples

10 ml of ruminal fluid was collected by stomach tube to evaluate ruminal fluid pH, physical properties (color, odor and consistency), protozoal motility and number and ruminal microflora. A digital pH meter (Adwa, AD11, ROMANI) calibrated with standard pH buffer was used for measuring of rumen pH immediately after collection according to Constable *et al.*, (2017). For the detection of rumen microflora, an air-dried smear of ruminal fluid was stained by the Gram method and observed under an oil immersion microscope to determine the ratio of Gram (+) to Gram (-) bacteria according to Brahma *et al.* (2020).

Statistical analysis

Data were analyzed using R 4.1.0 (R Foundation for Statistical Computing, Vienna, Austria). Descriptive statistics for the different clinical and biochemical parameters in before and after lactic acidosis treatment were presented. Paired t test was used to test the statistical difference between all parameters before and after treatment.

RESULTS

Clinical examination

The descriptive results of the clinical examination are summarized in Table 1. Values (mean ± SD) of respiratory and heart rate showed highly significant decrease after treatment. Respiratory rate was decreased from 37.5±2.74 to 24.0±2.41 and the heart rate was decreased from 100 ±3.87 to 76.0±2.91. Before treatment, rumen contraction/2minutes was 1 in 89.5% of cases and 2 in 10.5% but showed highly significant increase after treatment to be 2 in 5.3% and 3 in 94.7%. Body temperature revealed insignificant changes.

Hemogasometry and hematological parameters

Statistical analysis, as shown in Table 2, revealed a highly significant increase in values (mean ± SD) of blood pH (Fig. 1A), HCO_3^- and pCO_2 after treatment at P-value (<0.001). The pre-treatment means were 7.28±0.0477, 18.2±1.49 and 28.8±2.17 respectively, while post treatment they were 7.42±0.0116, 25.1±1.59 and 39.6±1.46 respectively. Means of Hb, HCT% and TLC revealed a highly significant decrease after treatment at P-value (<0.001). They were 13.7±0.61, 36.7±2.48 and 12.4±0.685 respectively before treatment, while after treatment they were 10.6±0.337, 24.2±0.963 and 10.0±0.359 respectively. RBCs count showed insignificant changes.

Biochemical parameters

Serum biochemical parameters, as clear in Table3, including lactate, ALT, AST, and creatinine revealed a highly significant decrease after treatment at P-value (<0.001). Their pre-treatment values (mean ± SD) were 32.2±5.46, 56.0±7.36, 85.8±5.32 and 1.05±0.0581 respectively, while they were 13.9±2.64, 31.3±3.86, 59.7±6.55, and 0.842±0.0607 respectively after treatment. Serum urea, βHBA and TAC showed a highly significant increase after treatment at P-value (<0.001). Their values (mean ± SD) before treatment were 31.4±3.98, 0.307±0.0851 and 0.920±0.161 respectively, while post treatment they were 54.9 ±3.11, 0.671±0.0860 and 2.34±0.490 respectively. Blood βHBA, TAC and lactate level before and after treatment are presented in Fig. 1B-D.

Ruminal fluid analysis

All acidotic goats before treatment have a rumen pH below

Table 1. Clinical parameters and rumen pH before and after lactic acidosis treatment

	Before TTT (n=57)	After TTT (n=57)	P-value
Temperature			
Mean (SD*)	39.5 (0.0571)	39.5 (0.0551)	0.055
Median [Min, Max]	39.5 [39.3, 39.6]	39.5 [39.3, 39.6]	
Heart rate/minute			
Mean (SD)	100 (3.87)	76.0 (2.91)	< 0.001
Median [Min, Max]	99.0 [94.0, 110]	76.0 [70.0, 82.0]	
Respiratory rate/minute			
Mean (SD)	37.5 (2.74)	24.0 (2.41)	< 0.001
Median [Min, Max]	37.0 [32.0, 43.0]	24.0 [20.0, 29.0]	
Rumen pH			
Mean (SD)	5.16 (0.460)	6.54 (0.246)	< 0.001
Median [Min, Max]	5.20 [4.30, 5.80]	6.60 [6.00, 6.90]	
Rumen contractions/2minutes			
1	51 (89.5%)	0 (0%)	
2	6 (10.5%)	3 (5.3%)	
3	0 (0%)	54 (94.7%)	

*SD : Standard Deviation

Table 2. Hematological and hemogasometric parameters before and after lactic acidosis treatment

	Before (n=57)	After (n=57)	P-value
RBCs (10^{12}/L)			
Mean (SD*)	13.5 (0.702)	13.6 (0.702)	0.2
Median [Min, Max]	13.3 [12.1, 14.8]	13.4 [12.3, 14.8]	
TLC (10^9/L)			
Mean (SD)	12.4 (0.685)	10.0 (0.359)	< 0.001
Median [Min, Max]	12.3 [11.2, 13.8]	10.1 [9.35, 10.8]	
Hb (g/dl)			
Mean (SD)	15.9 (16.5)	10.6 (0.337)	0.01
Median [Min, Max]	13.8 [12.2, 138]	10.7 [10.0, 11.1]	
HCT%			
Mean (SD)	36.7 (2.48)	24.2 (0.963)	< 0.001
Median [Min, Max]	37.2 [30.6, 39.5]	24.1 [22.0, 27.3]	
Blood pH			
Mean (SD)	7.28 (0.0477)	7.42 (0.0116)	< 0.001
Median [Min, Max]	7.30 [7.18, 7.34]	7.42 [7.40, 7.45]	
HCO_3^-(mmol/L)			
Mean (SD)	18.2 (1.49)	25.1 (1.59)	< 0.001
Median [Min, Max]	18.3 [15.2, 21.0]	25.3 [21.2, 27.3]	
pCO_2 (mmHg)			
Mean (SD)	28.8 (2.17)	39.6 (1.46)	< 0.001
Median [Min, Max]	29.0 [24.0, 33.0]	39.5 [36.0, 43.0]	

*SD : Standard Deviation; RBCs : Red blood cells count; TLC : Total leucocytic count; Hb: Hemoglobin; HCT: Hematocrite; HCO_3^-: Bicarbonate concentration; pCO_2: Carbon dioxide partial pressure.

the normal range. The pre-treatment rumen pH values (mean ± SD) were 5.16±0.460 with a minimum of 4.30 and a maximum of 5.80. After treatment, the rumen pH showed a highly significant increase of 6.54±0.246 with a minimum of 6.00 and a maximum of 6.90, as shown in Table 1 and Fig. 1E. As presented in Table 4, the physical properties of ruminal fluid before treatment were milky grey in color, with sour odor and watery consistency, which had improved after treatment towards the normal of healthy goats (greenish color, aromatic odor and viscous consistency). Pre-treatment rumen fluid smears showed a predominance of Gram (+) organisms, whereas after treatment Gram (-) organisms were predominant. Microscopic examination of the rumen fluid showed the absence of rumen protozoa before treatment, which become active and abundant after treatment.

DISCUSSION

The clear signs observed prior to treatment were anorexia, dullness, dehydration, teeth grinding, pasty feces, ruminal stasis, increased respiratory and heart rate as previously reported by Ribeiro *et al*. (2020) and Koondhar and Khaskheli (2021). These signs coincided with the low rumen pH, increased rumen osmolarity, mobilization of intra and extra vascular fluids in the rumen and haemoconcentration. Increased respiration may be due to stimulation of the respiratory center by the acidic blood pH as a compensatory mechanism for the correction of the metabolic acidosis that has occurred (Hajikolaei *et al*., 2006). Clinical signs gradually improved at the start of treatment and resolved within 72 h of treatment similar to what was previously reported by many authors (Arora *et al*., 2011; Valmik *et al*., 2017; Koondhar *et al*., 2020 a,b) in which they reported that the clinical signs resolved after treatment in all acidotic goats as relevant treatment was recommended.

Low rumen pH and motility and the changes of the ruminal fluid physical properties which recorded prior to treatment were similar to those reported by Chavelikar *et al*. (2018) and Saravanan *et al*. (2021). These findings may be attributed to the excessive accumulation of volatile fatty acids and lactic acid in the rumen, prolonged stasis of rumen, increased rumen osmolarity and passage of fluid from the circulation into the rumen (Constable *et al*., 2017).

Predominance of Gram positive flora and absence of rumen protozoa pre-treatment as previously reported by Gupta *et al*.

Table 3. Biochemical parameters before and after lactic acidosis treatment

	Before (n=57)	After (n=57)	P-value
Lactate (mg/dl)			
Mean (SD*)	32.2 (5.46)	13.9 (2.64)	< 0.001
Median [Min, Max]	30.5 [25.0, 46.7]	13.7 [10.3, 19.0]	
ALT (U/L)			
Mean (SD)	56.0 (7.36)	31.3 (3.86)	< 0.001
Median [Min, Max]	55.2 [43.0, 70.0]	31.3 [23.2, 38.3]	
AST (U/L)			
Mean (SD)	85.8 (5.32)	59.7 (6.55)	< 0.001
Median [Min, Max]	85.8 [76.3, 96.0]	59.0 [45.0, 73.0]	
Blood Urea (mg/dl)			
Mean (SD)	31.4 (3.98)	54.9 (3.11)	< 0.001
Median [Min, Max]	31.3 [22.0, 39.4]	55.0 [48.7, 63.0]	
Creatinine (mg/dl)			
Mean (SD)	1.05 (0.0581)	0.842 (0.0607)	< 0.001
Median [Min, Max]	1.03 [0.950, 1.18]	0.840 [0.750, 0.950]	
B-HBA (mmol/L)			
Mean (SD)	0.307 (0.0851)	0.671 (0.0860)	< 0.001
Median [Min, Max]	0.310 [0.100, 0.430]	0.660 [0.500, 0.830]	
TAC (ng/ml)			
Mean (SD)	0.920 (0.161)	2.34 (0.490)	< 0.001
Median [Min, Max]	0.900 [0.670, 1.65]	2.14 [1.45, 3.23]	

*SD: Standard Deviation; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; ß-HBA: Beta hydroxybutyric acid; TAC : total antioxidant capacity

Table 4. Rumen juice analysis before and after lactic acidosis treatment

Parameters	Before treatment	After treatment
Color	Milky gray	Greenish
Odor	Sour	Aromatic
Consistency	Watery	Viscous
Protozoal motility	+/- Inactive	+++ Highly active
Microflora	Predominant gram (+)	Predominant gram (-)

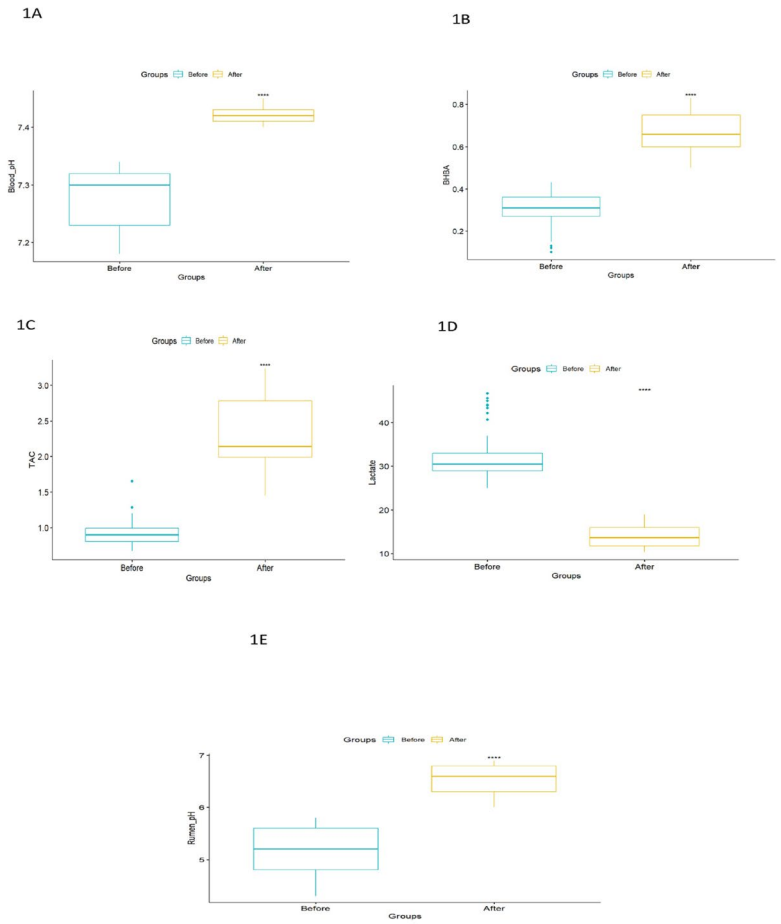

Figure 1. (A-D). Blood and ruminal pH, lactate, β-hydroxybutyric acid (βHBA) and total antioxidant capacity (TAC) before and after lactic acidosis treatment.

(2012) and Brahma *et al*. (2020) that may be attributed to the acidic rumen pH which not only adversely affects rumen motility but also rumen protozoa number and motility and microbial population, death of many Gram (−) bacteria and proliferation of other Gram (+), especially Streptococcus bovis and Lactobacilli spp., lactate producer bacteria (Hernández *et al*., 2014). After the therapeutic trial, ruminal pH elevated significantly, and the other fermentation characteristics returned to the normal of healthy goats as in Table 4.

The results of the hematological analysis were in line with Brahma *et al*. (2020) and Saravanan *et al*. (2021).The increase in Hb and HCT% values before treatment can be attributed to the haemocencentration that occurred following the influx of water from the circulation to the hyperosmotic rumen and hence elevation the Hb and HCT% (Ceroni *et al*., 2012), while the increase in total leucocytic count (TLC) may be due to elevated cortisol levels during the stress of eating high concentrate diet and may be due to rumenitis and damage to the rumen epithelium (Joshi *et al*., 2017).

The results concerning blood gas analysis agreed with those of Hajikolaei *et al*. (2006) and Ribeiro *et al*. (2020). There is a close relationship between the rumen pH and blood pH. Lactic acid, produced by the fermentation of rapidly digestible carbohydrates, breaks down into lactate and H^+, which are absorbed into the blood stream by the rumen wall. Absorbed lactate is metabolized by the liver until the amount of lactate increases and the liver capacity is overwhelmed. This acid accumulates, causing reduction of blood pH and development of metabolic acidosis. The bicarbonate reserves were depleted by buffering of the accumulated acid to counteract the incoming of metabolic acidosis as a result of rumen acidosis, causing a decrease of blood pH. This reduction acts as a stimulus to the respiratory center, increases alveolar ventilation and removes excess CO_2 from the lungs leading to a decrease in pCO_2 (Hajikolaei *et al*., 2006).

Results of ALT and AST activities before treatment similar to those reported by Gupta *et al*. (2012) and Saravanan *et al*. (2021) which decreased significantly after treatment. These findings could be attributed to muscle and hepatocellular damage due to the toxic products as histamine, thiaminase and other endotoxins produced in rumen and entering the portal circulation (Constable *et al*., 2017).

Plasma L-lactate concentration before treatment showed a marked increase over the normal range of goats as documented in numerous studies (Vieira *et al*., 2012; Gupta *et al*., 2012; Brahma *et al*., 2020; Ribeiro *et al*., 2020) which may be due to the large production of lactate in the rumen and its absorption into the blood stream (Ribeiro *et al*., 2020).

Regarding TAC and βHBA results prior to treatment, the high level of glucose associated with eating rapidly fermentable carbohydrates, reduced the βHBA concentration (Van Knegse *et al*., 2005). Ruminal acidosis has also been associated with production of excessive reactive oxygen species (ROS) and oxidative stress, leading to depletion of enzymatic and non-enzymatic antioxidants in their neutralization, as evident from the reduction in TAC in acidotic goats (Kirbas *et al*., 2014). These results coincided with (Joshi *et al*., 2017) in which they recorded a significant decrease in TAC level during acute ruminal lactic acidosis in goats and coincided with (Marchesini *et al*., 2013) for βHBA results.

The marked decrease in serum urea concentration in acidotic goats pre- treatment was consistent with Vieira *et al*. (2012) which recorded a significant decrease in serum urea concentration during induction of ruminal lactic acidosis in sheep, which can be attributed to the change in the intra rumen fermentation pattern and the reduction of NH_3^- producing microbial population associated with ruminal acidosis, reflecting a reduction in serum urea (Braun *et al*., 2010). The elevated creatinine level be-

fore treatment agrees with Brahma *et al*. (2020) and Saravanan *et al*. (2021) that can be attributed to reduced renal perfusion and glomerular filtration rate associated with rumen acidosis (Chavelikar *et al*., 2018).

Within 72 h of the therapeutic trials the general health of goats, ruminal pH and motility, the hematobiochemical parameters and blood gas analysis improved to be within the normal range of healthy goats.

CONCLUSION

The current study concludes that calcareous marine algae, sodium bicarbonate and magnesium oxide mixture is significantly effective for curing lactic acidosis in goats. Helps minimize the severity of lactic acidosis in goats and improves their general health. Therefore, it can be used as the therapy of choice for curing lactic acidosis in goats. Further studies with larger sample size are recommended to confirm the results on a large scale.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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