



# Revista Agrária Acadêmica

[Agrarian Academic Journal](#)

Volume 3 – Número 1 – Jan/Fev (2020)



doi: 10.32406/v3n12020/7-16/agrariacad

**Cardiac biomarkers in goats with experimental ruminal lactic acidosis and supplemented with monensin sodium.** Biomarcadores cardíacos em caprinos com acidose láctica ruminal experimental e suplementados com monensina sódica

[Ana Clara Sarzedas Ribeiro](#)<sup>ID<sup>1</sup>\*</sup>, [Gliére Silmara Leite Soares](#)<sup>ID<sup>2</sup></sup>, [Saulo de Tarso Gusmão da Silva](#)<sup>ID<sup>3</sup></sup>, [Eldiné Gomes de Miranda Neto](#)<sup>ID<sup>4</sup></sup>, [Rebeka Menezes Pontes](#)<sup>ID<sup>2</sup></sup>, [Pierre Castro Soares](#)<sup>ID<sup>5</sup></sup>, [Carla Lopes de Mendonça](#)<sup>ID<sup>6</sup></sup>, [José Augusto Bastos Afonso](#)<sup>ID<sup>6</sup></sup>

<sup>1</sup> Programa de Pós-Graduação em Sanidade e Reprodução de Ruminantes, Unidade Acadêmica de Garanhuns, Universidade Federal Rural de Pernambuco – UFRPE, Garanhuns/PE – Brasil. E-mail: [ac\\_sarzedas@id.uff.br](mailto:ac_sarzedas@id.uff.br)

<sup>2</sup> Programa de Pós-Graduação em Medicina Veterinária, Departamento de Medicina Veterinária, Universidade Federal Rural de Pernambuco – UFRPE, Recife/PE – Brasil. E-mail: [glieresoares87@gmail.com](mailto:glieresoares87@gmail.com), [rebekampontes@gmail.com](mailto:rebekampontes@gmail.com)

<sup>3</sup> Unidade Acadêmica de Garanhuns, Universidade Federal Rural de Pernambuco – UFRPE, Garanhuns/PE – Brasil. E-mail: [stetarso@hotmail.com](mailto:stetarso@hotmail.com)

<sup>4</sup> Unidade Acadêmica de Medicina Veterinária, Universidade Federal de Campina Grande – UFCG, Patos/PB – Brasil. E-mail: [eldinemneto@hotmail.com](mailto:eldinemneto@hotmail.com)

<sup>5</sup> Departamento de Medicina Veterinária, Universidade Federal Rural de Pernambuco – UFRPE, Recife/PE – Brasil. E-mail: [pcastro.pe@gmail.com](mailto:pcastro.pe@gmail.com)

<sup>6</sup> Clínica de Bovinos de Garanhuns, Universidade Federal Rural de Pernambuco – UFRPE, Garanhuns/PE – Brasil. E-mail: [carlалopes.mendonca@gmail.com](mailto:carlалopes.mendonca@gmail.com), [afonsojab@oi.com.br](mailto:afonsojab@oi.com.br)

\*Corresponding author

## Abstract

Aimed to evaluate creatine kinase MB (CK-MB) and cardiac troponin I (cTnI) in goats with experimental ruminal lactic acidosis (RLA) and supplemented with monensin sodium (MS). Twenty goats were divided into control group (CG) and treatment group (TG) supplemented with 33mg/animal/day of MS. The RLA was induced (10g/Kg body weight of sucrose) and before and post-induction clinical data, ruminal fluid and blood were collected. All animals presented mild RLA, with minimal ruminal pH at 8h post-induction. CK-MB and cTnI were higher in the CG and TG, respectively, without differ between moments. MS at the dose used did not prevent RLA whose severity was not sufficient to cause cardiac injury.

**Keywords:** Cardiac troponin I. Creatine kinase MB. Ionophores. Myocardial injury. Ruminal acidosis.

## Resumo

Objetivou-se avaliar a creatina quinase MB (CK-MB) e troponina I cardíaca (cTnI) em caprinos com acidose láctica (ALR) experimental e suplementados com monensina sódica (MS). Vinte caprinos foram divididos em grupo controle (GC) e grupo tratamento (GT) suplementado com 33mg/animal/dia de MS. A ALR foi induzida (10g/Kg de peso corporal de sacarose) e, antes e pós-indução, foram coletados dados clínicos, fluído ruminal e sangue. Todos os animais apresentaram ALR leve, com pH ruminal mínimo 8 horas pós-indução. CK-MB e cTnI foram maiores no GC e GT, respectivamente, sem diferença entre os momentos. A MS na dose utilizada não preveniu a ALR cuja gravidade não foi suficiente para causar lesão cardíaca.

**Palavras-chave:** Troponina cardíaca I. Creatina quinase MB. Ionóforos. Lesão miocárdica. Acidose ruminal.

## Introduction

Ruminal lactic acidosis (RLA) is a nutritional and metabolic disorder of ruminants caused by excessive and unadapted ingestion of non-structural carbohydrates. After consumption in large quantities, these carbohydrates are fermented by amylolytic ruminal bacteria and lactic acid producing bacteria, especially *Streptococcus bovis* and *Lactobacillus* spp., which precipitate increased production and accumulation of volatile fatty acids (VFA) and lactic acid, causing a decrease in ruminal pH. The accumulation of lactic acid in the rumen and its excessive absorption into the bloodstream triggers, beyond the fermentative disorder, a metabolic acidosis, as well as several potentially fatal secondary processes. The severity of the clinical presentation varies according to the amount of carbohydrates ingested, the amount of lactate generated in the rumen, and the individual susceptibility (JARAMILLO-LÓPEZ et al., 2017; NAGARAJA; LECHTENBERG, 2007; OWENS et al., 1998; SNYDER; CREDILLE, 2017).

It is hypothesized that acid-base and electrolyte disturbances and secondary complications associated with RLA cause cardiovascular injury. Recently, cardiac injury biomarkers have been used in studies seeking to evaluate the association between myocardial injury and ruminal acidosis in small ruminants (FARTASHVAND; HAJI-SADEGHI, 2017; JOSHI et al., 2017; KIRBAS et al., 2014a) however, this relationship is still not fully understood.

Blood cardiac biomarkers, especially creatine kinase MB isoenzyme (CK-MB) and cardiac troponin I (cTnI), have been widely used in human medicine in the diagnosis of patients with heart disease and other critical diseases, allowing the establishment of therapy and prognosis, with greater accuracy in a shorter time (ALATASSI et al., 2018; ANTMAN, 2002; SINGH et al., 2010). In veterinary medicine, these markers have been used to determine the presence of myocardial injury in animals affected by primary cardiac diseases and non-cardiac disorders, being related to severity and prognosis (CUMMINS; AUCKLAND; CUMMINS, 1987; MELLANBY et al., 2007; RADCLIFFE et al., 2015; VARGA et al., 2013; YONEZAWA et al., 2010).

The preventive measures adopted for RLA include the gradual supply of carbohydrates in the diet and the use of food additives that regulate the ruminal environment, such as the ionophore antibiotics that selectively inhibit Gram-positive ruminal bacteria, among them the main producers of lactic acid, favoring the growth of Gram-negative bacteria (NAGARAJA; LECHTENBERG, 2007; RANGEL et al., 2008). Monensin sodium has been widely used in the diet of confined cattle to improve food efficiency and prevent the occurrence of acute ruminal lactic acidosis (NAGARAJA et al., 1982; SALLES; LUCCI, 2000a, 2000b) and its efficiency in the prevention of RLA has also been studied in goats, through clinical and laboratory observations, with inconsistent results (MIRANDA NETO et al., 2011).

There are very few studies on the use of blood cardiac biomarkers as a criterion for cardiac injury in goats with RLA, especially supplemented with ionophore, and if this preventive measure is effective. Thus, the objective of this study was to evaluate the serum concentrations of cTnI and CK-MB in goats with experimental RLA, which received monensin sodium in their diet.

## Materials and methods

The experimental design was submitted and approved by the Ethics Committee on the Use of Animals of the Federal Rural University of Pernambuco (CEUA/UFRPE) under license number

066/2018. The study was conducted in the Garanhuns Bovine Clinic (CBG), Federal Rural University of Pernambuco (UFRPE) *Campus*, Garanhuns, PE, Brazil. Twenty male, castrated, crossbred Anglo Nubian and Saanen goats were used, with an average age of two years, average body weight of 30Kg and clinically healthy. The animals were submitted to surgical intervention for the implantation of permanent ruminal cannulas (REICHERT NETO, 1996). A 40-day postoperative interval was established for complete recovery, as well as adaptation to the environment and feed management. During the adaptation period and the experimental phase, the goats received soybean meal (300g/animal/day), grasses *Pennisetum purpureum*, *Cynodon* spp., and *Brachiaria decumbens*, mineral salt and water *ad libitum*.

The goats were randomly divided into two groups of 10 animals, a control group (CG) and a treatment group (TG) which received 33mg/animal/day of monensin sodium (Rumesin 100, Elanco Química) through the ruminal cannula during the adaptation period and the experimental phase (BROWN; HOGUE, 1985). Ruminal acidosis was induced by intraruminal sucrose administration at a dose of 10g/Kg body weight (CAKALA; BORKOWSKI; ALBRYCHT, 1974; CAO et al., 1987). Before induction, characterizing the control moment (CM), and at the intervals of 4h, 8h, 12h, 24h, 32h, 48h, and 72h post-induction (PI), a clinical examination was performed and samples of ruminal fluid and blood were collected.

The clinical examination followed the recommendation by Smith; Sherman (2009) and characteristics such as attitude, behavior, appetite, heart and respiratory rate, reticulum-ruminal motility, rectal temperature and appearance of feces were evaluated.

Ruminal fluid samples were collected through the cannula and processed in the Clinical Laboratory of CBG/UFRPE, following the guidelines of Dirksen (1993). Immediately after collection, the physical characteristics such as color, odor and consistency were evaluated, as well the ruminal pH, using a digital pH meter (pH-100, PHTEK).

Blood samples were collected by jugular venipuncture in sterile vacuum tubes (BD Vacutainer, Becton Dickinson Ind. Cir. Ltda.), without anticoagulant and with sodium fluoride/potassium oxalate, to obtain serum and plasma, respectively. The samples were aliquoted and stored in a freezer at -80°C for further laboratory processing.

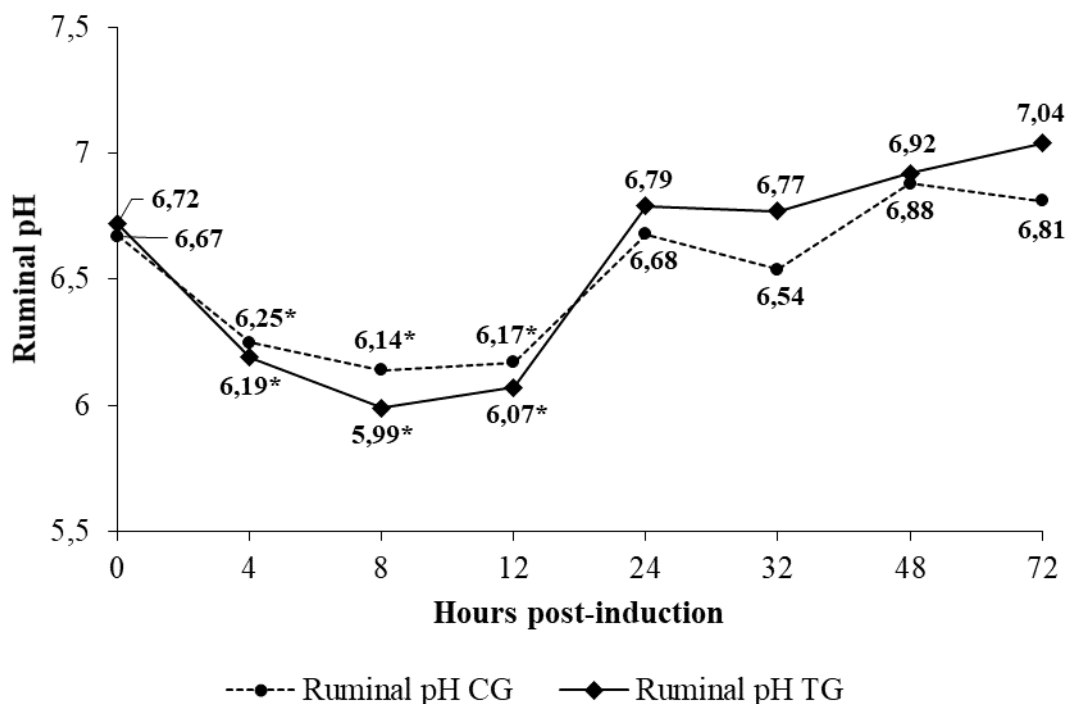
The analyzes of the biochemical blood variables were performed at the Clinical Laboratory of CBG/UFRPE; in the Research Support Center (CENAPESQ) and in the Laboratory of Nutritional and Metabolic Diseases of Ruminants, UFRPE, Recife, PE. The serum activity of the enzyme creatine kinase (CK-NAC Liquiform, Labtest Diagnóstica S.A.) and the plasma concentration of L-lactate were evaluated (Lactato Enzimático, Labtest Diagnóstica S.A.) in a semiautomatic analyzer (BIO 2000/Labquest, Bioplus Produtos Para Laboratórios Ltda.). The serum activity of the creatine kinase MB isoenzyme (CK-MB Liquiform, Labtest Diagnostica S.A.) was determined in an automatic biochemical analyzer (Labmax 240, Labtest Diagnóstica S.A.). Serum concentrations of cTnI (Access AccuTnI, Beckman Coulter) and cortisol (Access Cortisol, Beckman Coulter) were determined by chemiluminescence immunoassay (Access 2 Immunoassay System, Beckman Coulter). As recommended by the manufacturer and considering the linearity of the test of 0.01-100ng/mL, in the samples for which cTnI values were below the minimum detection limit of the assay, the results were recorded as <0.01ng/mL.

For statistical analysis, the data were initially tested for normality of distribution using the Kolmogorov-Smirnov test. The variables that did not meet the normality assumptions were submitted to log-transformation ( $\log_{10}$ ) or square root transformation ( $x+1$ ). Data were then submitted to analysis of variance (ANOVA two way) using the General Linear Model (GLM) procedure of the

MINITAB18 statistical program. If significance was observed in the ANOVA F Test, the contrast between the means was performed by the least significant difference (l.s.d.) of the Tukey test. The significance level of 5% was considered for all analyzes.

## Results

The experimental intake of 10g/Kg body weight of sucrose caused mild ruminal lactic acidosis in both groups. The clinical examination revealed alterations, mainly between 4h and 24h PI, such as sternal decubitus, apathy, moderate dehydration, anorexia or inappetence, abdominal distension, reduction in ruminal motility, loss of rumen stratification, and pasty or diarrheic feces, blackened and fetid. There was an increase ( $P<0.05$ ) in the heart rate between 4h and 8h PI and the rectal temperature at 8h PI, however these variables remained within the normal range for the species. The respiratory rate did not change ( $P>0.05$ ) throughout the experimental period (Table 1). Clinical reestablishment occurred from 48h PI in both groups and no animal died, in addition to which there was no need for therapeutic intervention.



**Figure 1.** Mean ruminal pH values of goats submitted to experimental induction of ruminal lactic acidosis and supplemented with monensin sodium. CG, control group; TG, treatment group. \*Significant difference ( $P<0.05$ ) between moments.

**Table 1.** Heart rate, respiratory rate, and rectal temperature (mean ± standard error) of goats submitted to experimental induction of ruminal lactic acidosis and supplemented with monensin sodium.

	Group	CM	4h	8h	12h	24h	32h	48h	72h	OM
HR (bpm)	CG	109.60±10.63	129.20±8.74	117.20±7.35	110.20±9.16	107.00±5.51	106.00±6.00	102.40±6.69	95.20±5.52	109.60 <sup>A</sup>
	TG	98.80±6.93	119.00±7.11	116.00±5.66	101.60±6.68	108.60±6.41	103.80±4.35	111.20±6.97	91.40±6.10	106.30 <sup>A</sup>
	OM	104.20 <sup>ab</sup>	124.10 <sup>a</sup>	116.60 <sup>a</sup>	105.90 <sup>ab</sup>	107.80 <sup>ab</sup>	104.90 <sup>ab</sup>	106.80 <sup>ab</sup>	93.30 <sup>b</sup>	
RR (rmpm)	CG	32.10±5.83	27.60±2.56	29.60±3.64	22.80±1.47	22.00±1.23	24.40±2.10	23.60±2.02	24.90±1.77	25.87 <sup>A</sup>
	TG	30.00±6.65	30.00±4.59	47.00±12.13	23.40±2.63	28.80±2.44	39.20±10.22	26.80±2.92	26.00±2.88	31.40 <sup>A</sup>
	OM	31.05 <sup>a</sup>	28.80 <sup>a</sup>	38.30 <sup>a</sup>	23.10 <sup>a</sup>	25.40 <sup>a</sup>	31.80 <sup>a</sup>	25.20 <sup>a</sup>	25.45 <sup>a</sup>	
Temperature (°C)	CG	38.82±0.15	38.79±0.15	39.32±0.12	39.29±0.16	38.76±0.11	39.38±0.21	38.98±0.19	38.66±0.15	39.00 <sup>A</sup>
	TG	38.86±0.15	38.73±0.14	39.45±0.15	38.89±0.15	38.67±0.17	39.16±0.14	38.39±0.15	38.28±0.18	38.80 <sup>B</sup>
	OM	38.84 <sup>bcd</sup>	38.76 <sup>cd</sup>	39.38 <sup>a</sup>	39.09 <sup>abc</sup>	38.71 <sup>cd</sup>	39.27 <sup>ab</sup>	38.68 <sup>cd</sup>	38.47 <sup>d</sup>	

HR, heart rate; bpm, beats per minute; RR, respiratory rate; rmpm, respiratory movements per minute; CG, control group; TG, treatment group; OM, over mean; CM, control moment. <sup>A,B</sup> Different capital letters in the same column indicate significant differences ( $P<0.05$ ) between groups. <sup>a,b,c,d</sup> Different lowercase letters on the same line indicate significant differences ( $P<0.05$ ) between moments.

**Table 2.** Serum biochemical variables (mean ± standard error) of goats submitted to experimental induction of ruminal lactic acidosis and supplemented with monensin sodium.

	Group	CM	4h	8h	12h	24h	32h	48h	72h	OM
L-lactate (mmol/L)	CG	0.86±0.11	0.85±0.10	1.10±0.09	1.11±0.18	0.93±0.17	0.89±0.16	0.70±0.06	0.63±0.08	0.88 <sup>B</sup>
	TG	0.90±0.17	1.40±0.20	1.46±0.20	1.07±0.09	1.10±0.15	1.26±0.23	0.89±0.16	1.40±0.20	1.10 <sup>A</sup>
	OM	0.88 <sup>abc</sup>	1.12 <sup>ab</sup>	1.28 <sup>a</sup>	1.09 <sup>ab</sup>	1.02 <sup>abc</sup>	1.08 <sup>abc</sup>	0.80 <sup>bc</sup>	0.65 <sup>c</sup>	
Cortisol (nmol/L)	CG	13.21±1.94	17.11±4.72	25.04±4.83	24.53±3.97	16.15±2.31	16.45±2.34	8.96±1.59	10.32±2.74	16.47 <sup>A</sup>
	TG	12.90±3.55	11.12±2.29	24.53±8.07	21.46±3.92	14.65±3.50	11.95±3.10	13.48±3.46	8.65±2.13	14.82 <sup>A</sup>
	OM	13.05 <sup>ab</sup>	14.12 <sup>ab</sup>	24.79 <sup>a</sup>	23.00 <sup>a</sup>	15.40 <sup>ab</sup>	14.28 <sup>ab</sup>	11.06 <sup>b</sup>	9.49 <sup>b</sup>	
CK (U/L)	CG	14.09±1.07	12.28±0.96	13.43±0.94	12.63±1.01	13.36±1.16	14.78±1.37	12.93±1.64	13.48±1.41	13.37 <sup>B</sup>
	TG	16.75±1.55	19.06±1.71	18.94±1.57	17.24±1.32	17.24±1.16	16.51±1.44	14.81±1.22	14.08±1.48	16.83 <sup>A</sup>
	OM	15.42 <sup>a</sup>	15.67 <sup>a</sup>	16.19 <sup>a</sup>	14.93 <sup>a</sup>	15.30 <sup>a</sup>	15.65 <sup>a</sup>	13.87 <sup>a</sup>	13.78 <sup>a</sup>	
CK-MB (U/L)	CG	212.56±24.72	277.08±25.90	258.98±23.21	252.24±23.04	256.17±23.18	244.66±23.06	222.83±19.56	187.46±19.96	239.00 <sup>A</sup>
	TG	151.44±31.00	174.72±29.56	165.29±30.18	162.83±29.70	155.49±20.70	147.06±22.00	139.26±19.77	139.04±11.93	154.39 <sup>B</sup>
	OM	182.00 <sup>a</sup>	225.90 <sup>a</sup>	212.14 <sup>a</sup>	207.54 <sup>a</sup>	205.83 <sup>a</sup>	195.86 <sup>a</sup>	181.04 <sup>a</sup>	163.25 <sup>a</sup>	
cTnI (ng/mL)	CG	0.013±0.004	0.013±0.002	0.018±0.005	0.021±0.005	0.013±0.003	0.014±0.004	0.010±0.001	0.009±0.000	0.014 <sup>B</sup>
	TG	0.030±0.007	0.030±0.006	0.040±0.009	0.030±0.010	0.020±0.007	0.030±0.009	0.030±0.015	0.040±0.014	0.030 <sup>A</sup>
	OM	0.024 <sup>a</sup>	0.020 <sup>a</sup>	0.028 <sup>a</sup>	0.025 <sup>a</sup>	0.018 <sup>a</sup>	0.021 <sup>a</sup>	0.019 <sup>a</sup>	0.023 <sup>a</sup>	

CG, control group; TG, treatment group; OM, over mean; CM, control moment. <sup>A,B</sup> Different capital letters in the same column indicate significant differences ( $P<0.05$ ) between groups. <sup>a,b,c</sup> Different lowercase letters on the same line indicate significant differences ( $P<0.05$ ) between moments.

The ruminal fluid examination revealed alterations in color, odor, and consistency from 4h PI, with the exception of the color, which in the TG was altered at 8h PI. In both groups, the ruminal fluid coloration of the goats became milky green, the odor changed from aromatic to acidic, and the consistency became aqueous. After 48 hours PI, normality in these characteristics of the ruminal fluid had been restored in almost all the animals. The ruminal pH reduced significantly ( $P<0.05$ ) from 4h PI, reaching the minimum threshold at 8h PI with a mean of 6.07 and returning to basal values at 24h PI. There was no significant difference ( $P>0.05$ ) in this variable between the groups studied (Figure 1).

The results of the blood metabolites are described in Table 2. Plasma concentrations of L-lactate presented maximum values at 8h PI and when the two groups were compared, values were higher in the TG ( $P<0.05$ ). The highest serum concentrations of cortisol were recorded between 8h and 12h PI, returning to baseline values after this time, with no significant differences ( $P>0.05$ ) between the groups studied. Serum creatine kinase (CK) activity was higher in the TG ( $P<0.05$ ) with no significant differences ( $P>0.05$ ) between the moments. Regarding cardiac biomarkers, concentrations of CK-MB and cTnI were higher ( $P<0.05$ ) in the CG and TG, respectively, but there were no significant differences ( $P>0.05$ ) between the studied moments.

## Discussion

The clinical observations in the goats of the two groups in this study was characteristic of RLA and the intensity of the clinical manifestations coincided with the decrease in ruminal pH, corroborating with other authors who evaluated the disease in this species under experimental conditions (CAO et al., 1987; MIRANDA NETO et al., 2005) and cases of natural occurrence (JOSHI et al., 2017). The majority of the animals in the two groups presented clinical reestablishment 48h after induction, differing from other authors who observed in sheep experimentally submitted to ruminal acidosis, early recovery in animals receiving monensin sodium in the diet, when compared to the control group, allowing the authors to infer that supplementation with ionophore contributed to faster recovery of the ruminal environment, reducing the severity of the clinical presentation (AFONSO et al., 2005; REIS et al., 2018a).

The sudden supply of carbohydrates, followed by fermentation of this substrate by the amylolytic and lactic acid producing ruminal bacteria, causes accumulation of VFA and lactic acid in the rumen, reducing ruminal pH and substantially increasing the osmolarity of ruminal fluid, which becomes hypertonic in relation to the plasma, causing infusion of water from the intra and extracellular compartments into the organ. These factors contribute to the establishment of metabolic acidosis and secondary processes responsible for the alterations observed in the clinical examination of the animals in this study (NAGARAJA; LECHTENBERG, 2007; OWENS et al., 1998; SNYDER; CREDILLE, 2017).

Alterations in the physical characteristics of the ruminal fluid observed in the two groups, such as milky color, acid odor, and aqueous consistency, are related to the accumulation of organic acids and consequent increase in ruminal osmolarity (CAO et al., 1987) and were similar to those described in natural cases of the disease by Joshi et al. (2017) and under experimental conditions by Miranda Neto et al. (2005), who also observed the beginning of these alterations from 4h PI and reestablishment of these characteristics concomitantly with the elevation in the ruminal pH and the clinical recovery of the animals.

The significant reduction in ruminal pH observed in both groups from 4h after induction of the fermentative disorder was similar to that observed in other models when different doses of sucrose and animal species were used (CAO et al., 1987; HAJI HAJIKOLAEI et al., 2006; MIRANDA NETO et al., 2005). These authors reported ruminal pH values lower than 5.0 between 12h and 36h after induction of RLA and reestablishment of this variable from 48h. This difference in relation to the present study, in which the minimum ruminal pH values were close to 6.0, can be attributed to the lower dose of the substrate used to induce the disease, which also allowed the early return to basal ruminal pH values from 24h PI. The re-establishment of ruminal pH in a shorter time after RLA induction was reported in sheep supplemented with monensin sodium when compared to the control group (AFONSO et al., 2002, 2005), but this difference was not observed between groups in the present study. For the authors, this fact was related to the lower concentration of lactic acid in the ruminal fluid of the animals that received the ionophore antibiotic, since it has an inhibitory effect on Gram-positive rumen microbiota, the main producer of lactic acid and responsible for the decline in ruminal pH during the course of the disease.

The elevation of plasma L-lactate coincided with the lowest ruminal pH values recorded during the experiment, corroborating with the findings of Patra; Lal; Swarup (1996). In RLA the digestion of non-structural carbohydrates produces a significant amount of lactic acid that dissociates into lactate and H<sup>+</sup> ions, which are absorbed into the bloodstream through the ruminal epithelium, causing consumption of bicarbonate reserves and lowering of blood pH (MØLLER et al., 1997; SNYDER; CREDILLE, 2017). Elevations in ruminal lactic acid and systemic metabolic acidosis, characterized by hyperlactatemia and concomitant reduction in blood pH, have been reported in other studies with experimental ruminal acidosis in small ruminants (AFONSO et al., 2002, 2005; CAO et al., 1987; REIS et al., 2018b). The higher values of L-lactate in the TG disagree with other studies that did not observe a significant difference in plasma lactate between animals supplemented or not with monensin sodium (AFONSO et al., 2002, 2005; REIS et al., 2018b).

The elevation in cortisol between 8h and 12h PI, periods of lower ruminal pH, may stem from the stress related to the painful and inflammatory condition associated with the disease. This hormone has been used as an indicator of stress and pain in livestock animals and an increase in serum cortisol has been reported in different species affected by acute and subacute ruminal acidosis (AFONSO et al., 2002; BUSTAMANTE et al., 2015; JIA et al., 2014; SEESUPA; WACHIRAPAKORN; AIUMLAMAI, 2017), suggesting that these animals experienced a state of stress during the illness.

The serum activity of CK, although higher in the TG since the control moment, did not present alterations between the moments studied, corroborating with the results of Almeida et al. (2008). On the other hand, Lal et al. (1991) and Patra; Lal; Swarup (1996) observed increased activity of this enzyme after induction of ruminal acidosis in sheep and goats, respectively, suggesting the occurrence of musculoskeletal damage associated with the disease, probably related to the severity of the clinical signs presented by the animals in these studies.

Although the serum CK-MB activity and serum cTnI concentration were higher in the CG and TG, respectively, these cardiac injury biomarkers remained within the normal range, not varying after induction of the disease, disagreeing with the reports of Fartashvand; Haji-Sadeghi (2017), Kirbas et al. (2014b), and Joshi et al. (2017) who showed elevation in cTnI and CK-MB in sheep and goats clinically affected by RLA, suggesting some degree of myocardial injury in these animals which presented severe clinical manifestations and a high mortality rate. Possible reasons

for the occurrence of cardiac damage in ruminal acidosis include: metabolic acidosis; severe dehydration; oxidative stress, with excessive production of reactive oxygen species; endotoxemia; increased synthesis and release of inflammatory cytokines (CHALMEH et al., 2014; FARTASHVAND; HAJI-SADEGHI, 2017; JOSHI et al., 2017; KATRUKHA, 2013; KIRBAS et al., 2014a, 2014b). Fartashvand; Haji-Sadeghi (2017) indicated a relation between cTnI and the prognosis in ruminal acidosis, finding that animals with more critical ruminal pH values presented the highest concentrations of this biomarker and died acutely. The fact that CK-MB and cTnI concentrations were not elevated in the present study may be related to the milder clinical manifestations and lower decline in ruminal pH presented by the goats during the experimental course of the disease.

## Conclusion

The experimental model used induced mild clinical manifestations and alterations in ruminal fluid and blood metabolites characteristic of ruminal lactic acidosis. Monensin sodium, at a dose of 33mg/animal/day, did not prevent the onset of the fermentative disturbance in goats. There were no alterations in blood cardiac biomarker concentrations, suggesting that the severity of the disease was not sufficient to cause myocardial injury. However, CK-MB and cTnI may represent important prognostic indicators in animals with ruminal acidosis, although additional studies are needed to support this hypothesis.

## Acknowledgements

The authors would like to thank the Foundation for Support to Science and Technology of the State of Pernambuco (FACEPE) for financial support (APQ-0282-05.05/15) and the Research Support Center of the Federal Rural University of Pernambuco (CENAPESQ/UFRPE) for performing the chemiluminescence immunoassay.

## References

- AFONSO, J. A. B. et al. Metabolismo oxidativo dos neutrófilos de ovinos tratados com monensina sódica e experimentalmente submetidos à acidose ruminal. **Pesquisa Veterinária Brasileira**, v. 22, n. 4, p. 129–134, 2002.
- AFONSO, J. A. B. et al. Avaliação do uso da monensina sódica na prevenção da acidose láctica ruminal experimental em ovinos. **Veterinária Notícias**, v. 11, n. 1, p. 35–43, 2005.
- ALATASSI, A. et al. Association between troponin-I levels and outcome in critically ill patients admitted to non-cardiac intensive care unit with high prevalence of cardiovascular risk factors. **BMC Anesthesiology**, v. 18, n. 1, p. 1–11, 2018.
- ALMEIDA, M. Z. P. R. B. et al. Estudo clínico, hematológico e bioquímico em caprinos submetidos à acidose láctica ruminal induzida experimentalmente. **Veterinária e Zootecnia**, v. 15, n. 1, p. 100–113, 2008.
- ANTMAN, E. M. Decision Making with Cardiac Troponin Tests. **New England Journal of Medicine**, v. 346, n. 26, p. 2079–2082, 2002.



- BROWN, D. L.; HOGUE, D. E. Effects of Feeding Monensin Sodium to Lactating Goats: Milk Composition and Ruminant Volatile Fatty Acids. **Journal of Dairy Science**, v. 68, n. 5, p. 1141–1147, 1985.
- BUSTAMANTE, H. A. et al. Stress and pain response after oligofructose induced-lameness in dairy heifers. **Journal of Veterinary Science**, v. 16, n. 4, p. 405–411, 2015.
- CAKALA, S.; BORKOWSKI, T.; ALBRYCHT, A. Rumen acidosis in Sheep induced with different doses of saccharose. **Polskie Archiwum Weterinaryjne**, v. 17, n. 1, p. 117–130, 1974.
- CAO, G. R. et al. Experimentally induced lactic acidosis in the goat. **Australian Veterinary Journal**, v. 64, n. 12, p. 367–370, 1987.
- CHALMEH, A. et al. Alterations in electrocardiographic parameters and serum cardiac biomarkers in an ovine experimental endotoxemia model. **Istanbul Universitesi Veteriner Fakultesi Dergisi**, v. 40, n. 2, p. 211–219, 2014.
- CUMMINS, B.; AUCKLAND, M. L.; CUMMINS, P. Cardiac-specific troponin-I radioimmunoassay in the diagnosis of acute myocardial infarction. **American Heart Journal**, v. 113, n. 6, p. 1333–1344, 1987.
- DIRKSEN, G. Sistema Digestivo. In: DIRKSEN, G.; GÜNDER, H. D.; STÖBER, M. (Eds.). **Rosenberger Exame Clínico dos Bovinos**. 3. ed. Rio de Janeiro, RJ, Brasil: Guanabara Koogan, 1993. p. 163–224.
- FARTASHVAND, M.; HAJI-SADEGHI, Y. Evaluation of serum cardiac biomarkers in sheep with acute lactic acidosis. **Journal of the Hellenic Veterinary Medical Society**, v. 68, n. 2, p. 219–224, 2017.
- HAJI HAJIKOLAEI, M. R. et al. Effects of experimentally induced ruminal lactic acidosis on blood pH, bicarbonate and pCO<sub>2</sub> in the sheep. **Pakistan Journal of Biological Sciences**, v. 9, n. 10, p. 2003–2005, 2006.
- JARAMILLO-LÓPEZ, E. et al. Ruminal acidosis: Strategies for its control. **Austral Journal of Veterinary Sciences**, v. 49, n. 3, p. 139–148, 2017.
- JIA, Y. Y. et al. High concentrate-induced subacute ruminal acidosis (SARA) increases plasma acute phase proteins (APPs) and cortisol in goats. **Animal**, v. 8, n. 9, p. 1433–1438, 2014.
- JOSHI, V. et al. Evaluation of oxidant-antioxidant status, serum cytokine levels and some cardiac injury biomarkers in acute ruminal lactic acidosis in goats. **Small Ruminant Research**, v. 149, p. 6–10, 2017.
- KATRUKHA, I. A. Human cardiac troponin complex. structure and functions. **Biochemistry (Moscow)**, v. 78, n. 13, p. 1447–1465, 2013.
- KIRBAS, A. et al. Evaluation of serum cardiac troponin I concentration in sheep with acute ruminal lactic acidosis. **Veterinarski Arhiv**, v. 84, n. 4, p. 355–364, 2014a.
- KIRBAS, A. et al. Status of lipid peroxidation and some antioxidants in sheep with acute ruminal lactic acidosis. **Medycyna Weterinaryjna**, v. 70, n. 6, p. 357–361, 2014b.
- LAL, S. B. et al. Biochemical alterations in serum and cerebrospinal fluid in experimental acidosis in goats. **Research in Veterinary Science**, v. 50, n. 2, p. 208–210, 1991.
- MELLANBY, R. J. et al. Serum cardiac troponin I concentrations in cattle with pericarditis. **Veterinary Record**, v. 161, n. 13, p. 454–455, 2007.
- MIRANDA NETO, E. G. et al. Aspectos clínicos e a bioquímica ruminal de caprinos submetidos à acidose láctica experimental e suplementados ou não com monensina sódica. **Pesquisa Veterinária Brasileira**, v. 31, n. 5, p. 416–424, 2011.
- MIRANDA NETO, E. G. et al. Estudo clínico e características do suco ruminal de caprinos com acidose láctica induzida experimentalmente. **Pesquisa Veterinária Brasileira**, v. 25, n. 2, p. 73–78, 2005.
- MØLLER, P. D. et al. Absorption and fate of L- and D-lactic acid in ruminants. **Comparative Biochemistry**

**and Physiology - A Physiology**, v. 118, n. 2, p. 387–388, 1997.

NAGARAJA, T. G. et al. Effect of Lasalocid, Monensin or Thiopeptin on Lactic Acidosis in Cattle. **Journal of Animal Science**, v. 54, n. 3, p. 649–658, 1982.

NAGARAJA, T. G.; LECHTENBERG, K. F. Acidosis in Feedlot Cattle. **Veterinary Clinics of North America - Food Animal Practice**, v. 23, n. 2, p. 333–350, 2007.

OWENS, F. N. et al. Acidosis in Cattle: A Review. **Journal of Animal Science**, v. 76, n. 1, p. 275–286, 1998.

PATRA, R. C.; LAL, S. B.; SWARUP, D. Biochemical profile of rumen liquor, blood and urine in experimental acidosis in sheep. **Small Ruminant Research**, v. 19, n. 2, p. 177–180, 1996.

RADCLIFFE, R. M. et al. The clinical value of whole blood point-of-care biomarkers in large animal emergency and critical care medicine. **Journal of Veterinary Emergency and Critical Care**, v. 25, n. 1, p. 138–151, 2015.

RANGEL, A. H. N. et al. Utilização de ionóforos na produção de ruminantes. **Revista de Biologia e Ciências da Terra**, v. 8, n. 1, p. 264–273, 2008.

REICHERT NETO, N. C. Fistulação ruminal em ovinos. In: ANAIS DO 15º CONGRESSO PANAMERICANO DE CIÊNCIAS VETERINÁRIAS 1996, Campo Grande, MS. **Anais...** Campo Grande, MS: CBC - Cia Brasileira de Comunicações, 1996.

REIS, L. F. et al. Prevention of acute ruminal lactic acidosis in sheep by probiotic or monensin supplementation: Clinical aspects. **Semina: Ciências Agrárias**, v. 39, n. 4, p. 1575–1583, 2018a.

REIS, L. F. et al. Comparative assessment of probiotics and monensin in the prophylaxis of acute ruminal lactic acidosis in sheep. **BMC Veterinary Research**, v. 14, n. 1, p. 1–8, 2018b.

SALLES, M. S. V.; LUCCI, C. S. Monensina para Bezerros Ruminantes em Crescimento Acelerado. 1. Desempenho. **Revista Brasileira de Zootecnia**, v. 29, n. 2, p. 573–581, 2000a.

SALLES, M. S. V.; LUCCI, C. S. Monensina para Bezerros Ruminantes em Crescimento Acelerado. 2. Digestibilidade e Parâmetros Ruminais. **Revista Brasileira de Zootecnia**, v. 29, n. 2, p. 582–588, 2000b.

SEESUPA, S.; WACHIRAPAKORN, C.; AIUMLAMAI, S. Effects of induced subacute ruminal acidosis and laminitis on lipopolysaccharide binding protein, cortisol and progesterone levels in dairy heifers. **Thai Journal of Veterinary Medicine**, v. 47, n. 4, p. 501–511, 2017.

SINGH, V. et al. Cardiac biomarkers - The old and the new: A review. **Coronary Artery Disease**, v. 21, n. 4, p. 244–256, 2010.

SMITH, M. C.; SHERMAN, D. M. **Goat Medicine**. 2. ed. Oxford, UK: Wiley-Blackwell, 2009.

SNYDER, E.; CREDILLE, B. Diagnosis and Treatment of Clinical Rumen Acidosis. **Veterinary Clinics of North America - Food Animal Practice**, v. 33, n. 3, p. 451–461, 2017.

VARGA, A. et al. Preliminary investigation of cardiac troponin i concentration in cows with common production diseases. **Journal of Veterinary Internal Medicine**, v. 27, n. 6, p. 1613–1621, 2013.

YONEZAWA, L. A. et al. Marcadores cardíacos na medicina veterinária. **Ciência Rural**, v. 40, n. 1, p. 1–9, 2010.

Recebido em 31 de outubro de 2019  
Aceito em 3 de janeiro de 2020