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## Effects of Experimentally Induced Ruminal Lactic Acidosis on Blood pH, Bicarbonate and pCO<sub>2</sub> in the Sheep

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**Abstract:** Rumen lactic acidosis in 5 clinically healthy four years old female sheep with 43.2±1.15 kg weight was successfully produced experimentally using sugar at a dose rate of 18 g kg<sup>-1</sup> BW given intraruminally through a fixed rumen fistula. Prior to induction of the disease (0 h), rumen samples in order to determine baseline of rumen pH were obtained and blood collected via jugular vein to determine baseline of blood pH, pCO<sub>2</sub> and bicarbonate. Samples of rumen fluid and venous blood were collected at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 h after induction of acidosis. Results indicated that blood pCO<sub>2</sub> decreased at 24 h, blood pH decreased at 18, 21, 24, 30, 36 and 48 h, blood bicarbonate decreased at 15, 18, 21, 24, 30, 36 and 48 h, rumen pH decreased at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 h, heart rate increased at 9, 12, 15, 18, 21, 24, 30 and 48 h, rectal temperature decreased at 24, 30 and 36 h significantly and respiratory rate remained without detectable significant changes through the experiment.

**Key words:** Acidosis, experimentally, sheep, acid-base, sugar

### INTRODUCTION

Rumen lactic acidosis is a metabolic disease that most common cause of the disease is ingestion of excessive quantities of highly fermentable carbohydrate feed. These feeds are rapidly fermented in the rumen and yield lactic acid at such a rapid rate that the normal buffering capacity of the rumen is overwhelmed. This leads initially to a rumen acidosis followed by rumen stasis, metabolic acidosis, dehydration, recumbency, hypovolemic shock and frequently death (Anderson, 1992; Wendy, 1992). Less common causes include engorgement of apples, grapes, bread, baker's dough, sugar beet, mangels, sour wet brewer's grain that was incompletely fermented in the brewery and concentrated sucrose solutions used in apiculture. All type of ruminant, cattle and sheep, are susceptible, but the disease occurs most commonly in feedlot and dairy ruminants feed on high-level grain diets. Outbreaks of the disease occur in ruminants kept on grain farms. Depending on the species of grain, the total amount eaten and the previous experience of the animal, the morbidity will vary from 10-50%. The case fatality rate may be up to 90% in untreated cases while, in treated cases it still vary be up to 30-40% (Radostist *et al.*, 2000). Selection a useful treatment manner in the special time for the successful treatment of rumen lactic acidosis necessitates to known acid-base status changes during rumen lactic

acidosis. The aims in the present study were to determine the changes in blood pH, bicarbonate and pCO<sub>2</sub> following the administration of sugar at the rate of 18 g kg<sup>-1</sup> body weight.

### MATERIALS AND METHODS

The study was carried out in the Department of Large Animal Internal Medicine and Surgery, Veterinary Faculty of Shahid Chamran University of Ahwaz, Iran in January 2006.

Five healthy female sheep four years old, with average weight of 43.2±1.15 kg were used for this study. The animals were dewormed by Albendazole 15 mg kg<sup>-1</sup> body weight orally and Ivermectin 0.2 mg kg<sup>-1</sup> body weight subcutaneously and were kept under uniform managerial condition for adaptation in new environment during two weeks. One week prior to induction of rumen lactic acidosis, rumen of these animals were fistulated by ruminostomy technique. For maintenance of anaerobic environment in rumen, wooden door placed in the fistula and during one week they were ensured to be healthy and free from any clinically detectable abnormality. Prior to induction of the disease (0 h), rumen samples to determine baseline of rumen pH (pH measured with digital pH meter model C G822 Schott Gerate that made in Germany) were obtained by using a 60 mL syringe. To indicate blood pCO<sub>2</sub>, pH and bicarbonate (measured by Radiometer

ABL 5 made in Copenhagen) blood samples collected via jugular vein by plastic syringe with 0.1-0.2 mL heparine (5 U mL<sup>-1</sup>), the syringe was covered with a tight fitting cap and beside ice delivered to the laboratory immediately. Rumen lactic acidosis was induced by intraluminal administration of sugar 18 g/body weight. Samples of rumen fluid and venous blood were collected at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 h after induction of rumen lactic acidosis for above-mentioned purposes. Data were analyzed by one way ANOVA to determine significant difference. Probabilities of  $p < 0.05$  were considered to be statically significant.

## RESULTS AND DISCUSSION

Mean value ( $\pm$ SE) for constituents measured in rumen fluid, venous blood and for clinical parameters in the 5 sheep are shown in Table 1. Mean ruminal pH fell rapidly over the first 3 h and decreased significantly at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 h of rumen lactic acidosis. Blood pH initially increased at 3 h and significantly began to decrease at 18, 21, 24, 30, 36 and 48 h of rumen lactic acidosis. Blood bicarbonate slightly increased at 3 h and was significantly decreased at 15, 18, 21, 24, 30, 36 and 48 h of acidosis. Blood pCO<sub>2</sub> partially increased at 3 h then followed to decrease significantly by 24 h after dosing a metabolic acidosis. Heart rate increased significantly at 9, 12, 15, 18, 21, 24, 30 and 48 h. Respiratory rate remained without detectable significant changes through the experiment and Rectal temperature gradually rose at 3 and 6 h than zero time and decreased significantly at 24, 30 and 36 h.

The present finding will be compared largely with those experiment observed previously in sheep. The reduction of ruminal pH to a minimum of 4.04 at 36 h after sugar administration, caused most of systemic and clinical

changes that were observed in the present study. The ingestion of excessive quantities of highly fermentable feeds by a ruminant is followed within 2-6 h by a marked change in the microbial population in the rumen. There is an increase in the number of *Streptococcus bovis* which utilize the carbohydrate to produce large quantities of lactic acid. In the presence of a sufficient amount of carbohydrate the organism will continue to produce lactic acid which decreases the rumen pH (Smith, 2002; Radosits *et al.*, 2000). Andersen *et al.* (1994) recorded in cattle given of barley 70 g kg<sup>-1</sup> body weight, a similar decreased in ruminal fluid to minimum of 4.09 at 16 h. Further, when lactic acidosis was induced in sheep with sucrose at a dose rate of 18 g kg<sup>-1</sup> body weigh (Cao *et al.*, 1987), minimum fluid values of 4.7 to 4.54 were found between 12 and 24 h after dosing. Low rumen pH during the course of illness was similarly reported in sheep (Patra *et al.*, 1993). The decrease of rumen pH in our study has much similarity to earlier workers (Basak *et al.*, 1993; Mohamed Nour *et al.*, 1999) in goats. Three hours after the administration of sugar, a metabolic alkalosis developed. A feature of the acid-base disturbance following the administration of sugar in the present experiment was that lactic acidosis was preceded by a metabolic alkalosis. Thus probably occurred because over the first few hours the lactic acid, absorbed from the rumen and metabolized to bicarbonate by the liver, increased the blood bicarbonate concentration. However, the amount of lactic acid absorbed from the rumen by 18 h after dosing had overwhelmed the capacity of the liver to metabolize it and lactic acidosis developed. Furthermore, in ruminal lactic acidosis the fermentation not only produces acid but an increased osmotic concentration of breakdown solutes and this leads to accumulation of fluid within the rumen. In addition, gas production causes ruminal

Table 1: Measurement of rumen pH, blood parameters and clinical signs (mean $\pm$ SE) in experimentally induced ruminal lactic acidosis in the sheep

Item							
Time (h)	Rumen pH	Blood pH	Bicarbonate	pCO <sub>2</sub>	Heart rate	Respiratory rate	Temperature
0	6.91 $\pm$ 0.11	7.41 $\pm$ 0.03	25.85 $\pm$ 2.08	40.2 $\pm$ 2.17	66.6 $\pm$ 9.8	22.6 $\pm$ 3.45	39.46 $\pm$ 0.12
3	6.21 $\pm$ 0.26*	7.42 $\pm$ 0.04	26.2 $\pm$ 2.72	43.0 $\pm$ 1.89	71.4 $\pm$ 6.25	22.2 $\pm$ 3.8	39.52 $\pm$ 0.05
6	5.35 $\pm$ 0.3*	7.39 $\pm$ 0.03	25.2 $\pm$ 2.26	42.2 $\pm$ 1.88	75.6 $\pm$ 6.81	23.0 $\pm$ 4.15	39.52 $\pm$ 0.11
9	4.87 $\pm$ 0.31*	7.37 $\pm$ 0.03	23.6 $\pm$ 2.5	41.2 $\pm$ 2.26	91.8 $\pm$ 9.7*	23.8 $\pm$ 4.05	39.88 $\pm$ 0.14
12	4.71 $\pm$ 0.32*	7.34 $\pm$ 0.03	20.8 $\pm$ 1.68	38.8 $\pm$ 1.28	96.4 $\pm$ 10.4*3	26.6 $\pm$ 3.61	39.66 $\pm$ 0.22
15	4.53 $\pm$ 0.26*	7.27 $\pm$ 0.05	16.6 $\pm$ 2.18*	36.8 $\pm$ 1.2	100.2 $\pm$ 9.96*	25.4 $\pm$ 3.54	39.34 $\pm$ 0.23
18	4.34 $\pm$ 0.18*	7.21 $\pm$ 0.07*	15.6 $\pm$ 2.65*	38.0 $\pm$ 1.44	98.6 $\pm$ 8.48*	22.4 $\pm$ 2.22	39.18 $\pm$ 0.2
21	4.2 $\pm$ 0.17*	7.15 $\pm$ 0.05*	13.2 $\pm$ 1.88*	36.4 $\pm$ 0.92	99.0 $\pm$ 7.79*	23.0 $\pm$ 1.76	38.76 $\pm$ 0.22
24	4.1 $\pm$ 0.14*	7.19 $\pm$ 0.06*	13.34 $\pm$ 2.11*	35.04 $\pm$ 0.81*	95.8 $\pm$ 5.96*	19.0 $\pm$ 2.3	38.62 $\pm$ 0.39*
30	4.05 $\pm$ 0.08*	7.15 $\pm$ 0.09*	13.0 $\pm$ 1.76*	37.6 $\pm$ 2.37	93.6 $\pm$ 6.14*	18.8 $\pm$ 1.68	38.46 $\pm$ 0.46*
36	4.04 $\pm$ 0.14*	7.15 $\pm$ 0.08*	12.1 $\pm$ 1.61*	36.2 $\pm$ 1.28	86.0 $\pm$ 6.41	19.0 $\pm$ 1.89	38.62 $\pm$ 0.51*
48	4.19 $\pm$ 0.14*	7.23 $\pm$ 0.06*	14.25 $\pm$ 1.43*	35.25 $\pm$ 1.7	98.0 $\pm$ 6.05*	21.0 $\pm$ 2.28	39.07 $\pm$ 0.24

Values are significant at \* $p < 0.05$

distension and impedes venous return. These factors may impair hepatic perfusion and thus superimpose poorer lactate utilization and systemic acidosis occurs. Cao *et al.* (1987) recorded in goat given 18 g kg<sup>-1</sup> body weight sucrose, a similar founding in acid-base disturbance. The initial metabolic alkalosis in the experimented indicates that the early administration of bicarbonate intravenously for correction of systemic acidosis might be contraindicated. Initially a slight compensatory respiratory acidosis was also evident as a result of conversion the lactate to bicarbonate and a compensatory respiratory alkalosis were occurred at 24 h as result of elimination of CO<sub>2</sub> to response the systemic acidosis. The fell in blood pH will act as a stimulus to the respiratory control system, resulting in an increase in alveolar ventilation and a fall in pCO<sub>2</sub>. This respiratory adjustment of plasma pCO<sub>2</sub> will begin with a few minutes but will be maximally developed for up to 24 h. During metabolic acidosis the concentration of blood CO<sub>2</sub> increase due to the reduction of blood bicarbonate ion. Aslan *et al.* (1995) recorded that pCO<sub>2</sub> fell markely, probably due to hyperventilation. But Owen *et al.* (1998) and Brown *et al.* (2000) recorded opposite finding about pCO<sub>2</sub>. They recorded that during metabolic acidosis the concentration of blood CO<sub>2</sub> increase due to the reduction of blood bicarbonate ion and lower concentration of CO<sub>2</sub> is indicator of reduced risk of metabolic acidosis. Clinical signs changes in this study were similar to previous researchers (Cao *et al.*, 1987; Basak, 1993; Lal *et al.*, 1993).

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