

CLINICO-THERAPEUTICAL TRIALS OF LACTIC ACIDOSIS IN SMALL RUMINANTS

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ABSTRACT

The present study was designed to investigate the clinical parameters of goats during lactic acidosis and then response to different therapeutic agents. For this purpose, two hundred goats with the history of diarrhoea were examined at outdoor hospital, University of Veterinary and Animal sciences Lahore. A total of 38 animals were found positive for lactic acidosis showing 19 % prevalence of disease. For therapeutic study the thirty diseased animals were divided into three groups A, B, C comprising of 10 animals each. Goats in group A were given Magnesium Hydroxide at 1.11g/kg b.wt, while those in group B were fed Sodium bicarbonate at 1g/Kg wt. Animal in group C were maintained as untreated diseased. Ten healthy animals were maintained as control (group D). The rectal temperature, pulse rate, respiration rate, rumen pH, serum pH and protozoa motility test were performed before and after treatment. It was found that treatment of group A has significant effect on rumen pH and serum pH. The temperature, pulse and respiration rate were turned to normal range after treatment. It was concluded that lactic acidosis is a common disease of goats and its severity can be effectively reduced by using magnesium hydroxide at the dose rate of 1.11g/Kg orally.

Key words: Lactic acidosis, Goat, Magnesium hydroxide, Sodium bicarbonate, Serum pH, Protozoa.

INTRODUCTION

Lactic acidosis among domestic ruminants has been well known as a managemental disease for many years. The disease is clinically characterized by anorexia, depression, abdominal distension, diarrhea, weakness and inactivity. The disease is commonly encountered due to unintended ingestion of large quantities of cereal grains or their flour kept for human consumption (Mohamed Nour *et al.*, 1998).

The condition is becoming increasingly common among this species accompanied by high mortality rates. Lactic acidosis in goats occurs by sudden ingestion of toxic doses of easily fermentable carbohydrate rich feed materials such as grains. The common causes include engorgement of apples, grapes, breads, baker's dough, cabbage, mangles, sugar beets and concentrated sucrose solutions. In clinically affected animals morbidity rate varies from 10-50 percent and case mortality in lactic acidosis may reach to 90 percent in untreated cases whereas it may be 30-40 percent in treated cases (Radostits *et al.*, 2007).

The lactic acid causes a chemical rumenitis and its absorption results in lactic acidosis. The pathophysiologic consequences are hemoconcentration, cardiovascular collapse, renal failure, muscular weakness, shock and death. Animals that survive may develop mycotic rumenitis in several days, hepatic necrobacillosis several weeks or months later, or chronic laminitis, as well as evidence of ruminal scars at slaughter (Allen *et al.*, 2005).

Lactic acidosis has been extensively studied by researcher but still correct diagnosis and proper therapy is in the way by which the valuable animals can be saved. Therefore in the present study emphasis was put on its diagnosis and treatment protocol.

MATERIALS AND METHODS

Study area: This study was performed at the Outdoor Hospital, University of Veterinary and Animal sciences Lahore and adjacent areas.

EXPERIMENTAL DESIGN

Clinical Examination: Clinical examination of 200 goats of mixed breed, age and sex with the history of diarrhea was performed at outdoor patient clinic of University of Veterinary and Animal Sciences Lahore. Out of all, 38 animals were found positive for lactic acidosis showing 19 % prevalence of disease. For therapeutic study the thirty diseased animals were divided into three groups A, B, C comprising of 10 animals each. Goats in group A were given Magnesium Hydroxide at 1.11g/kg b.wt, while those in group B were fed Sodium bicarbonate at 1g/Kg wt. Animal in group C were maintained as untreated diseased. Ten healthy animals were maintained as control (group D). The Clinical examination included clinical signs, physical examination like body temperature, pulse rate, respiration rate and ruminal movements.

The pulse rate was taken at femoral artery, on the medial aspect of the thigh. The fingers were placed on the femoral artery and gentle pressure was applied till the wave was detected. The waves were counted for one minute (Amulendu, 2006).

The respiration rate was calculated by counting the movements of the rib cage and abdomen (Amulendu, 2006)

The body temperature was measured with a clinical thermometer. Before inserting into rectum the thermometer was shaken to bring the mercury down in the column of the thermometer below the lowest point likely to be recorded. The bulb of the thermometer was lubricated with liquid paraffin before inserting into rectum. The thermometer was gently inserted into rectum in rotatory manner. The thermometer was kept in the rectum for two minutes with care that it is in close contact with the wall of the rectum. After two minutes the thermometer was taken out of the rectum and reading of the body temperature was recorded (Amulendu, 2006).

Determination of blood pH: The 5 ml whole blood was collected into a test tube from the jugular vein using 16 gauge needle and was allowed to clot at room temperature for one hour to obtain serum (Hudson and Hay, 1989). The serum from clotted blood was used for determination of blood pH. Serum pH was measured with the use of wide range pH indicator paper and pH meter. The pH indicator paper was dipped into serum and the color of the strip was matched with the standard colors. The reading of the strip was taken immediately after dipping to avoid the change in color by exposure to air. The serum was put into beaker and pH meter was dipped into it. The reading of the pH meter was recorded. The mean value of both the readings was calculated (Radostits *et al.*, 2007).

Ruminal fluid pH: The ruminal fluid was collected through rumenocentesis using 16 gauge needle with a disposable syringe. The puncture site was the ventral rumen at left side on the horizontal line level with the top of the patella about 5-10 cm posterior to the last rib. The site was disinfected before inserting the needle. After the collection of rumen fluid the sample site was again disinfected to avoid the chance of infection. The ruminal fluid collected was used for determination of ruminal pH. The ruminal fluid pH was measured using wide range pH indicator paper and pH meter. The ruminal fluid was put on the paper and pH was recorded by change in the color of indicator paper and matching it with the standard colors of the indicator paper. The pH of ruminal fluid was determined immediately after collection before exposure to air as it can cause increase in pH if exposed to air. The ruminal fluid was put in beaker and pH meter was inserted into it. The pH value was recorded as the mean value.

Protozoa motility test: Few drops of collected ruminal fluid were placed on a glass slide with a tooth pick or platinum wire. Coverslip was put on it. The slide was examined under low power of microscope for the presence or absence of ruminal protozoa

Statistical Analysis: The data was statistically analyzed using one-way ANOVA technique. Significant differences were present in the Rumen and blood pH of lactic acidotic and healthy control group before treatment. After treatment significant differences were present in the rumen and blood pH values of group A, B, C and D, which are given in the Table 2.

RESULTS

Pulse Rate: It was found that all the lactic acidotic animals were having high pulse rate as compared to healthy control group D. The mean pulse rate values before treatment for group A, B, C and D were 100.6/min \pm 3.40008, 97.2/min \pm 4.24745, 103.6/min \pm 2.52636, and 86.3/min \pm 1.85 respectively. After treatment the pulse rate of the group A, B, C and D was again determined. Comparatively low values of the pulse rate of the treated groups i.e. A and B were obtained. The pulse rate of group C decreased slightly and the pulse rate of group D remained unchanged. The mean pulse values calculated after treatment were 88.55/min \pm 1.77768, 92/min \pm 1.75123, and 94.3/min \pm 1.90938 and 86/min \pm 1.06460 for group A, B, C and D respectively. Significant differences were found in the pre and post treatment values of temperature, pulse and respiration rate of the groups.

Respiration Rate: The respiration rate of group A, B, C and D was calculated before and after treatment. It was observed that all the lactic acidotic animals were having high respiration rate before treatment as compared to the healthy animals. The mean respiration rate values counted before treatment were 24.1/min \pm 0.86218, 28.2/min \pm 2.52020, 24/min \pm 0.84329 for group A, B, and C respectively. While the mean respiration rate of group D (healthy animals) was 19.8/min \pm 0.59256.

After treatment mean respiration rate values of group A, B, C and D were obtained. The respiration rate of group A and B decreased considerably and that of group C and D decreased slightly. The mean respiration rate values of group A, B, C and D after treatment were 20.55/min \pm 0.78953, 22.8/min \pm 0.80001, 24.5/min \pm 0.79234 and 20.7/min \pm 0.66751 respectively.

Temperature: The rectal temperature of all the groups i.e. A, B, C and D was determined using a clinical thermometer. The rectal temperature was determined before and after treatment. It was found that the rectal temperature before treatment was subnormal in all the lactic acidotic goats as compared to control group. The

mean rectal temperature values recorded before treatment were $99.74 F^0 \pm 0.67153$, $100.59 F^0 \pm 0.75580$, $101.25 F^0 \pm 0.49448$ and $102.86 F^0 \pm 0.30155$ for group A, B, C and D respectively.

After treatment rectal temperature values of all the groups were obtained. The mean temperature values of group A after treatment increased from $100.19 F^0$ to $102.47 F^0 \pm 0.21294$ and of group B increased from $100.59 F^0$ to $101.9 F^0 \pm 0.27849$. A very slight decrease in rectal temperature of group C occurred from $101.25 F^0$ to $101.19 F^0 \pm 0.41965$ and a very slight increase in the rectal temperature of group D was recorded from $102.86 F^0$ to $102.95 F^0 \pm 0.16142$. The increase in body temperature was a positive indication towards the recovery.

Serum pH: 5ml blood was collected from jugular vein of all the experimental animals using 16 gauge needle. Serum was obtained for blood pH determination. The blood pH of group A, B, C and D was determined before treatment as well as after treatment. The blood pH of all lactic acidotic goats before treatment was found to be lower than the blood pH of healthy control group. It was also a diagnostic point for diseased condition. The mean blood pH values recorded before treatment were 7.18 ± 0.04667 , 7.1 ± 0.05375 , 7.21 ± 0.05044 and 7.33 ± 0.02808 for group A, B, C and D respectively.

The blood pH values of group A and B increased after treatment. The blood pH values of group C decreased and that of group D increased. The blood pH values obtained after treatment were 7.35 ± 0.03 , 7.20 ± 0.06 , 7.09 ± 0.09 , 7.28 ± 0.12 for group A, B, C and D respectively. The results showed significant increases in the blood pH values of group A and B after administration of drugs.

Ruminal fluid pH: Ruminal fluid was collected through ruminocentesis from group A, B, C and D before and after 24 hrs of treatment. The ruminal fluid was used for pH determination. It was observed that the ruminal fluid pH before treatment in all the lactic acidotic cases was significantly lower than the normal range. The normal range of rumen pH for goats is between 6 and 7 while the ruminal pH of lactic acidotic goats was between 4 and 5. The mean values of rumen pH obtained before treatment were 4.43 ± 0.14533 , 4.49 ± 0.13287 , 4.76 ± 0.17075 and 6.39 ± 0.13699 for group A, B, C, and D respectively.

After treatment considerable increase occurred in the rumen pH of group A and B. The rumen pH of group C increased slightly and the rumen pH of group D also slightly increased. The mean values of rumen pH obtained after treatment were 6.13 ± 0.12 , 5.29 ± 0.12 , 4.94 ± 0.12 , and 6.42 ± 0.12 for group A, B, C and D respectively.

Protozoa motility test: Ruminal protozoa need an optimum pH for survival. After pH determination the

ruminal fluid collected was used for protozoa motility test. Few drops of the ruminal fluid were placed on a glass slide with a tooth pick or platinum wire to observe the ruminal protozoa. When observed under low power of microscope the ruminal protozoa were absent in the ruminal fluids collected from lactic acidotic goats. While a large number of protozoa were examined in the ruminal fluid obtained from healthy animals of group D.

After 24hrs of treatment again the protozoa motility test was repeated for all the groups. A slight to moderate amount of protozoa were observed in the ruminal fluids collected from group A and B. Protozoa were not observed in the ruminal fluids of group C. Abundant number of protozoa were observed in the ruminal fluids obtained from group D.

Treatment Trials: On the basis of change in ruminal and blood pH, protozoa motility test and recovery rate of animals it was found that magnesium hydroxide was more affective than sodium bicarbonate as it caused greater increase in ruminal pH and improved general body condition and rapid recovery. The change in pH was significant after the administration of drugs.

DISCUSSION

The decreased rectal temperature, noted in the present study was in accordance with Nour *et al.* (1998) which may be due to lactic acidosis, leading to dehydration. Increased rate of respiration (shallow and rapid) observed in the present case was in accordance to Allen *et al.*, 2005 and Radostits *et al.*, 2007. This increase in respiration might be due to stimulation of respiratory centre by increased carbon-dioxide (CO₂) tension of blood and decreased blood pH. Increased pulse rate was observed in lactic acidotic group similar finding has been reported by (Braun *et al.*, 1992; Hajikolaei *et al.*, 2006).

The decrease in pH of the ruminal fluid noted in the present study corresponds to increase production of volatile fatty acids like acetic, propionic and butyric acid. The decrease in pH of the rumen favors the growth of streptococci with decline in the number of normal Gram negative bacteria and protozoa, which further aggravates the process of lactic acid production. Similar findings have also been reported by several other researchers (Kezar and Church 1, 1979; Cao *et al.*, 1987; Crichlow, 1989; Aslan *et al.*, 1995; Mohamed Nour *et al.*, 1998).

The blood pH of lactic acidotic group was found to be lower than normal value which is in agreement with (Cao *et al.*, 1987; Braun *et al.*, 1992; Patra *et al.*, 1993; Angelov *et al.*, 1996). The decrease in the pH may be due to over-distention of rumen which impedes venous return to heart. This factor impairs hepatic perfusion and poorer lactic acid utilization which in turns leads to systemic lactic acidosis, manifesting decrease blood pH.

In the present study two different alkalinizing agents i.e. magnesium hydroxide and sodium bicarbonate were used for the treatment of lactic acidotic animals. Both the drugs were able to correct the acidotic condition which was evident from the increase in ruminal and blood pH and recovery rate of the animals after their administration. Magnesium hydroxide was found to cause greater increase in rumen pH than sodium bicarbonate. Which indicated that it is more affective than sodium

bicarbonate which is in close agreement with (Van 1983; Cao *et al.*, 1987; Aslan *et al.*, 1995; Smith and Correa 2004; Galip 2006).

It is concluded that lactic acidosis is a common managemental disease of goats and it can be rapidly diagnosed by determining the rumen and blood pH. Sodium bicarbonate is the affective and cheap drug which can be satisfactorily used as a treatment.

Table.1. drugs and their doses used in the treatment of lactic acidotic goats.

Group No.	No. of animals	Drug	Dose (g/kg) orally
A	10	Magnesium hydroxide	1.11
B	10	Sodium bi carbonate	1
C	10	Diseased	Untreated
D	10	Healthy	Untreated

Table. 2. Rumen pH, blood pH, Temperature (F), Pulse rate (per min) and respiration (per min) of goats after treatment (Mean ± S.E)

Group	Rumen pH	Blood pH	Temperature	Pulse rate	Respiration rate
A	6.13 ± 0.12 ^a	7.35±0.03 ^a	102.74±0.34 ^{ab}	88.60 ± 1.64 ^{bc}	21.31±0.95 ^{cd}
B	5.29 ± 0.12 ^b	7.20±0.06 ^c	102.77±0.65 ^{ab}	92.00 ± 1.64 ^b	25.65±1.85 ^{ab}
C	4.94 ± 0.12 ^b	7.09±0.09 ^{cd}	102.51±1.01 ^b	94.30 ± 1.64 ^{ab}	29.19±2.90 ^a
D	6.42 ± 0.12 ^a	7.28±0.12 ^{abc}	104.93±1.38 ^a	85.80 ± 1.64 ^c	27.24±3.97 ^{abc}

Values in each column with different superscript are significantly different at $p \leq 0.05$.

REFERENCES

- Allen, D. G., D. P. Anderson, L. B. Jeffcott, K. E. Quesenberry, O. M. Radostits, P. T. Reeves and A. M. Wolf (2005). The Merck Veterinary Manual, 9th Ed. pp. 178-179.
- Angelov, G., Y. Nikolov and A. Angelov (1996). Changes in acid-base parameters, blood sugar and blood lactate in experimental acute rumen acidosis in sheep. *Ind. Vet. J.* 73(No.3) S85.
- Aslan, V., S. M. Thamsborg, R. J. Jørgensen and A. Basse (1995). Induced acute ruminal acidosis in goats treated with yeast (*Saccharomyces cerevisiae*) and bicarbonate. *Acta Vet Scand.* 1995; 36(1):65-77.
- Braun, U., T. Rihs and U. Schefer (1992). Ruminal lactic acidosis in sheep and goats. *Vet. Rec.* 130(16):343-9.
- Cao, G. R., P. B. English, L. J. Filippich and S. Inglis (1987). Experimentally induced lactic acidosis in the goat. *Aust. Vet. J.* 64(12):367-70
- Crichlow, E. C. and R. K. Chaplin (1985). Ruminal lactic acidosis: relationship of forestomach motility to nondissociated volatile fatty acids levels. *Am. J. Vet. Res.* 46(9):1908-11.
- Galip, N. (2006). Effect of supplemental yeast culture and sodium bicarbonate on ruminal fermentation and blood variables in rams. *J. Anim. Physiol. Anim. Nutr. (Berl.)* 90(11-12):446-52.
- Hajikolaei, H. M. R., M. Nouri, F. S. Afshar and A. J. Dehkordi (2006). Effects of Experimentally Induced Ruminal Lactic Acidosis on Blood pH, Bicarbonate and pCO₂ in the Sheep. *Pak. J. Bio. Sci.* 9(10): 2003-2005, 2006, ISSN 1028-8880.
- Kezar, W. W. and D. C. Church 1 (1979). Ruminal Changes during the Onset and Recovery of Induced Lactic Acidosis in Sheep. *J. Ani. Sci.* 49:1161-1167.
- Mohamed Nour, M. S., M. T. Abusamra and B. E. D. Hago (1998). Experimentally induced lactic acidosis in Nubian goats: Clinical, biochemical and pathological investigations. *Small Ruminant Research*, 31, Issue 1, pp.7-17.
- Radostits, M. O., C. C. Gay, K. W. Hinchcliff and P. D. Constable (2007). Text book of Diseases of Cattle, horses, Sheep and Goats, 10th Ed., pp. 318-321.
- Patra, R. C., S. B. Lal and D. Swarup (1993). Physicochemical alterations in blood, cerebrospinal fluid and urine in experimental lactic acidosis in sheep. *Res. Vet. Sci.* 54(2):217-20.
- Smith, G. W. and M. T. Correa (2004). The effects of oral magnesium hydroxide administration on rumen fluid in cattle. *J. Vet. Intern. Med.* 18(1):109-12.
- Van, A. S. R. (1983). Oral antacid treatment in clinical rumen acidosis. *J. S. Afr. Vet. Assoc.* 54(4):265-6.