

Surgical Treatment of Experimentally Induced Lactic Acidosis in Nubian Goats

Mohamed Sayed Mohamed Nour

Department of Reproduction and Obstetrics, Faculty of Veterinary Medicine,
University of Khartoum, Khartoum North P. O Box 32, Sudan

Abstract: Experimental lactic acidosis was induced in five goats with sorghum flour at a dose rate of 50 g kg⁻¹ body weight. Animals were closely observed for the first appearance of the clinical signs the time at which animals were interfered with surgically (22 h post dosing). The surgical treatment was aimed at complete evacuation of the rumen with final replacement of the content with ruminal ingesta from healthy animal. Blood and ruminal parameters over a suitable time intervals were monitored to evaluate the treatment response. All treated animals showed a good response in their blood and intraruminal parameters. They resumed eating and drinking by 4 to 5 days post dosing and were bright and alert by the 6 to 7 days post-dosing.

Key words: Lactic acidosis, nubian goat, sorghum flour, surgical treatment

INTRODUCTION

Acute lactic acidosis in ruminants is an economically important disease. The goat that is considered as the poor man cow in Sudan suffers from this disorder. The disease is commonly encountered due to unintended ingestion of large quantities of sorghum flour kept for human consumption. The condition is becoming increasingly common among this species accompanied by high mortality rate^[1].

In our previous report^[1] the timing of clinical signs in correlation with the patho-physiological changes were investigated in acute experimentally acidotic goats. Considering the previous findings and our routine clinical trials, although fairly easier to treat moderate cases medicinally, acute acid indigestion presents a very difficult therapeutic challenge in this species as mentioned for other ruminants^[2,3], specially if the interference was delayed. Moreover, in acute cases, our preliminary trials towards correcting the rumen acidity and fluid loss, the major events accompanies the syndrome, resulted in signs of alkalosis and a considerable erythrocyte destruction respectively (Unpublished observations) a phenomenon need to be investigated in this species.

The present study was therefore planned to induce the disease experimentally and treat surgically, immediately at the onset of the clinical signs and observe the treatment outcomes by watching closely the clinical and the biochemical parameters.

MATERIALS AND METHODS

Animals, dosing and analytical techniques: Five apparently healthy Nubian goats aged 6-12 months and

weighed 15-25 kg were used in this study. Experimental lactic acidosis was produced as described previously^[1]. Briefly, all animals received sorghum flour intraruminally through a fixed ruminal cannula at a dose rate of 50 g kg⁻¹ body weight (50 g kg⁻¹ body weight). Samples of venous blood and rumen fluid were collected at 0 time and at a suitable intervals after dosing and treatment. All samples collected were prepared and tested for the following: venous blood for complete hemogram following the methods described by Shalm *et al.*^[4], lactic acid in rumen and plasma according to the method of Barker and Summerson^[5], Plasma bicarbonate according to the method of Segal^[6], Plasma and rumen glucose as described by Hyvarinen and Nikkila^[7]. At each time of sampling, animals were examined physically and their temperature, heart rate, pulse rate, respiratory rate and rumen motility were recorded as described by Kelly^[8].

Treatment: Immediately after the appearance of the clinical signs of the disease, animals were surgically interfered with by performing rumenotomy to evacuate the ruminal contents, followed by ruminal lavage with normal saline and finally replacement of fresh ruminal cud from healthy cannulated animal to restore the ruminal flora and fauna. A few handfuls of presoaked hay were also added for mechanical stimulation.

Statistical analysis: Data were analyzed by student's t-test using StatView software (Abacus Concepts, StatView, Abacus Concepts Inc., Berkeley, CA, 1992). Probabilities of p<0.05 were considered to be statistically significant.

RESULTS

All animals dosed with sorghum flour used in this study started to develop clinical signs of acute lactic acidosis by 18 to 22 h post-dosing.

General outcomes of the treatment: Animals were treated surgically 22 h post-dosing. All animals showed a good response in their blood and intraruminal parameters as shown in Fig. 1, 2 and 3. They resumed eating and drinking by 4 to 5 days post-dosing. They were bright and alert by the 6 to 7 days post dosing.

Physical parameters: The heart and respiratory rates increased progressively by the four hour after dosing. These rates decreased steadily after surgical treatment until normal parameters were recorded by the 7 to 8 days post dosing (Table 1).

Hematological findings: Hematological findings are summarized in Table 2. The total white blood cell count,

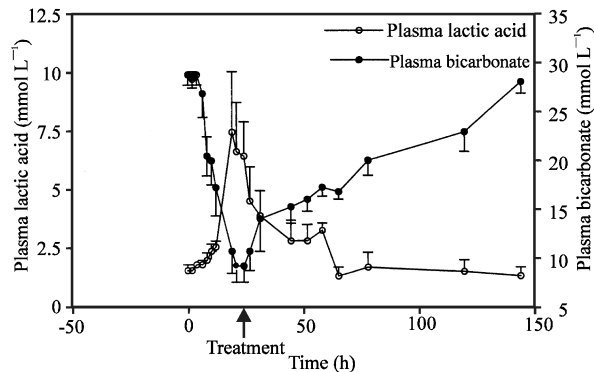


Fig. 1: Changes in the concentration and the relationship between plasma lactic acid and plasma bicarbonate in experiment goats

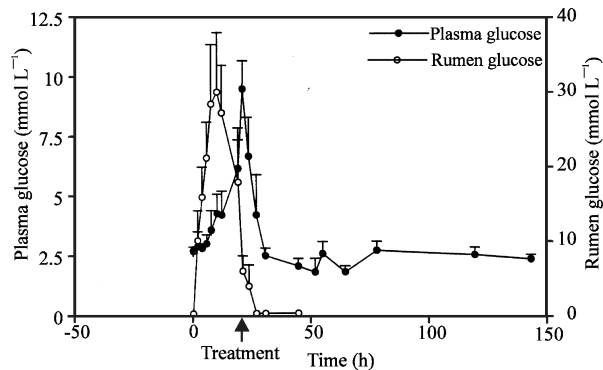


Fig. 2: Changes in the concentration and the relationship between plasma glucose and rumen glucose in experiment goats

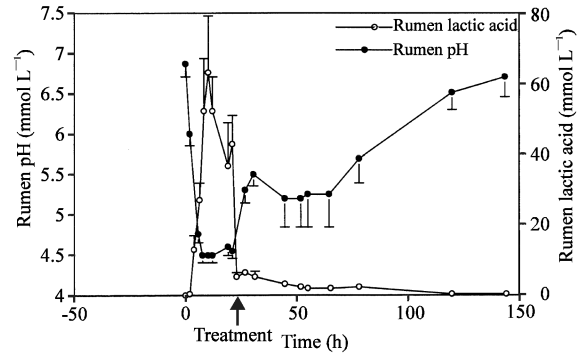


Fig. 3: Changes in the concentration of rumen lactic acid and its relationship to the changes in the rumen pH experiment goats

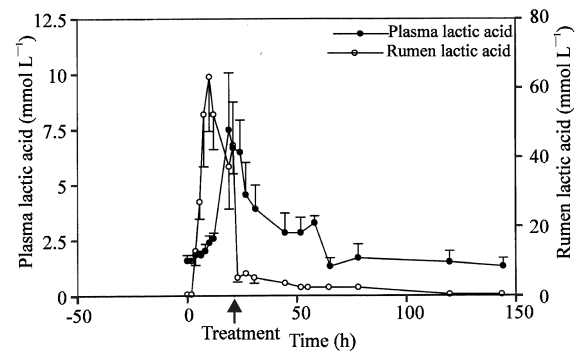


Fig. 4: The relationship between plasma and rumen lactic acid in experimental goats

Table 1: Physical parameters (Mean±SE) of experimental goats treated surgically

| Parameters | | | |
|------------|----------------|----------------|---------------|
| Time (h) | Temperature °F | Heart rate/min | Resp.rate/min |
| 0 | 101.6±0.5 | 58±2.6 | 15±0.8 |
| 4 | 102.5±0.1 | 70±8.9 | 17±1.9 |
| 8 | 102.4±0.2 | 92±12 | 18±3.5 |
| 12 | 102.1±0.6 | 115±4.9 | 19±3.6 |
| 21 | 100.7±0.6 | 132±8.9 | 27±3.1 |
| Treatment | | | |
| 24 | 100.5±0.6 | 118±11 | 21±2.3 |
| 28 | 101.7±0.2 | 121±10 | 20±3.3 |
| 44 | 101.6±0.3 | 125±4 | 17±3.3 |
| 67 | 101.0±0.5 | 126±13.3 | 16±3.7 |
| 79 | 101.5±0.5 | 125±16.3 | 17±3.5 |
| 5th day | 101.8±0.4 | 103±16.8 | 16±3.7 |
| 6th day | 102.0±0.5 | 84±19.5 | 15±1.3 |
| 7th day | 101.7±0.4 | 68±7.3 | 15±1.3 |
| 8th day | 101.3±0.5 | 63±4.4 | 14±1.3 |

neutrophil and lymphocyte percentages were back to normal level 22 h after treatment. The PCV dropped to its normal level 2 h after treatment.

Blood chemistry: The plasma lactic acid showed slight elevation until the 35 h post treatment and finally decreased to a normal level 45 h post treatment (Fig. 1).

Table 2: Hematological findings (Mean±SE) of experimental goats treated surgically

| Parameters | Time (h) | | | | | | | | | |
|---|-----------|------------|------------|------------|----|------------|------------|------------|-----------|------------|
| | 0 | 4 | 8 | 19 | TR | 24 | 28 | 44 | 96 | 120 |
| RBC (10 ¹² L ⁻¹) | 14.15±0.8 | 16.52±1.3 | 16.93±0.6 | 17.67±2.7 | | 13.33±2.2 | 11.82±1.5 | 15.31±2.0 | 12.03±2.1 | 11.00±2.3 |
| PCV (mL dL ⁻¹) | 24.10±0.7 | 24.00±1.0 | 27.80±0.8* | 28.33±1.9* | | 24.00±1.1 | 24.50±1.1 | 24.00±0.6 | 21.00±0.8 | 19.00±0.6* |
| Hb (g dL ⁻¹) | 10.30±0.9 | 12.40±0.7 | 13.20±0.8 | 12.60±0.6 | | 10.20±0.3 | 10.20±0.3 | 10.70±1.0 | 10.30±0.8 | 9.90±1.0 |
| WBC (10 ⁹ L ⁻¹) | 11.30±0.4 | 16.50±0.9* | 16.01±1.2* | 15.60±1.6* | | 15.21±1.2* | 14.92±1.2* | 9.60±0.8 | 13.90±0.2 | 12.70±0.3 |
| Neut (%) | 22.50±1.4 | 40.00±4.5* | 40.20±1.6* | 62.00±2.1* | | 40.67±2.3* | 43.00±3.1* | 36.00±3.9* | 21.30±0.8 | 21.60±2.6 |
| Lympho (%) | 73.50±1.4 | 56.67±4.7* | 56.86±1.6* | 36.00±2.1* | | 54.33±2.3* | 55.00±3.1* | 61.00±3.9* | 75.70±0.8 | 75.40±2.6 |
| Eosino (%) | 1.0±0.0 | 0.00 | 1.0±0.1 | 1.0±0.1 | | 2.00±0.3 | 1.00±0.2 | 1.00±0.1 | 1.00±0.1 | 2.00±0.2 |
| Baso (%) | 0.0 | 0.0 | 0.0 | 1.0 | | 0.0 | 1.0±0.1 | 0.0 | 0.0 | 0.0 |
| Mono (%) | 3.00±0.4 | 3.00±0.3 | 2.00±0.4 | 0.0 | | 3.00±0.2 | 0.0 | 2.00±0.2 | 2.00±0.3 | 1.00±0.3 |

*Mean values differ significantly (at least p<0.05) from 0 h values
TR = Treatment

The plasma bicarbonate level (Fig. 1) recorded a minimum level of 9.2 mmol L⁻¹ one hour before treatment. The level began to increase steadily 6 h after treatment until normal levels were reached by the 6 day post dosing. The plasma glucose began to decrease immediately after treatment reaching normal values, 8 h after treatment (Fig. 2).

Rumen chemistry: The mean rumen pH values (Fig. 3) began to rise reaching normal levels at pH 6.5-6.7 four days after treatment. The rumen lactic acid level (Fig. 3) decreased sharply 2 h after treatment reaching normal levels by the 4-day post treatment.

The glucose content of the ruminal fluid decreased steadily 2 h after treatment and the zero level was reached 6 h after treatment (Fig. 2).

DISCUSSION

Very few studies have been published on the outcomes of therapy of experimentally induced grain engorgement lactic acidosis, specifically in goats^[9,10], specifically in acute cases.

In this study, the significant finding of this therapeutic trail is that early surgical intervention (at time of clinical signs detection) is a valuable therapeutic tool for engorgement toxemia in experimental goats. The complete evacuation of the reticulorumen from the toxins and toxic contents and thorough lavage adopted, markedly decreased the ruminal lactic acid level and consequently the rumen pH increased steadily. As a result, there was no more absorption of lactic acid from the rumen (Fig. 4). Although plasma pH under this experimental condition was not measured, lactacidaemia was steadily corrected as indicated by the increased levels of bicarbonate. The pronounced restoration of the ruminal physiological activity could be mainly due to the incorporation of the normal ruminal cud with the soaked hay. Present clinical response is in general agreement to the reports of Dunlop^[11,12] and Blood and Radostits^[2] in sheep and cattle, respectively.

The delayed return of heart rate towards normal and more speed recovery in this study needs further investigation, however, recovery of normal cardiovascular parameters could have been accelerated by supportive therapy.

Generally early surgical intervention gave a fairly good response and led to complete recovery, however the mid and late intervention at the so called irreversible state needs thorough investigations. Suitable and powerful mechanical or therapeutic preventive measures would be of great value in reducing the disease occurrence at risk areas.

REFERENCES

1. 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 Res.*, 31: 7-17.
2. Blood, D.C. and O.M. Radostits, 1989. Acute Carbohydrate Engorgement of Ruminants (Rumen Overload), In: Blood, D.C. and O.M. Radostits, (Eds.) *Veterinary Medicine*. Baillière Tindal, London, pp: 246-253.
3. Dirksen, G., 1970. Acidosis., In: Phillipson, A.T. (Ed.) *Proceedings of the 3rd International Symposium on the Physiology of Digestion and Metabolism in the Ruminant*. Oriel Press., New Castle, England., pp: 612-625.
4. Schalm, O.W., N.C. Jain and E.J. Carrol, 1975. *Veterinary Haematology*, 3rd Edn., Lea and Fbiger., Philadelphia, pp: 55-60.
5. Barker, S.B. and W.H. Summerson, 1941. Preparation and colorimetric determination of lactic acid. *J. Bioyl. Chem.*, 138: 535.
6. Segal, M.A., 1955. A rapid electrotitrimetric method for determining Co₂ combining power in plasma or serum. *Am. J. Clin. Pathol.*, 25: 1212-1216.

7. Hyvarinen, A. and E.A. Nikkila, 1962. Specific determination of blood glucose with o-toluidine. *Clin. Chem. Acta*, 7: 140-143.
8. Kelly, W.R., 1984. *Veterinary Clinical Diagnosis*, 3rd Edn., Bailliere Tindal, London, pp: 26-39.
9. Cao, G.R., P.B. English, L.J. Fillippich and S. Inglis, 1987. Experimentally induced lactic acidosis in the goats. *Aus. Vet. J.*, 64: 367-370.
10. Gnanaprakasam, V., 1970. Rumen acidosis in goats. *Ind. Vet. J.*, 47: 904-910.
11. Dunlop, R.H., 1970. Acidosis. Discussion of Paper by G. Dirksen, In: Phillipson, A.T. (Ed.) *Proceedings of the 3rd International Symposium on the Physiology of Digestion and Metabolism in the Ruminant*. Oriel press, Newcastle, England, pp: 626-629.
12. Dunlop, R.H., 1972. Pathogenesis of Ruminant Lactic acidosis. In: *Advances In Veterinary Science And Comparative Medicine*. Academic press, New York, NY, pp: 259-302.