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## Ruminal lactic acidosis and its haemato-biochemical alterations in free ranging goats

**Saravanan S, Ramprabhu R, Mohanapriya T and Chitra R**

**Abstract**

About sixty two goats with the history of excess carbohydrate feeding presented signs suggestive of ruminal lactic acidosis. The physical properties of the ruminal fluid from the suspected cases like colour, consistency, odour and pH were assessed. In majority of the cases, the rumen fluid had the pH in the range of 5.0-6.0 (30.64%), greenish colour (38.7%), acrid odour (33.87%), watery consistency (59.67%) and absence of active protozoa (75.8%). Serum biochemical analysis revealed a significant decrease ( $P<0.05$ ) in the values (mean  $\pm$  SE) of globulin and ALP, and significant increase in ( $P<0.05$ ) in the values (mean  $\pm$  SE) of creatinine, ALT, AST, phosphorus and glucose. Whereas, a highly significant increase ( $P<0.01$ ) in the values (mean  $\pm$  SE) of BUN and chloride were observed. Haematological analysis revealed a significant increase ( $P<0.05$ ) in the values (mean  $\pm$  SE) of PCV, significant decrease ( $P<0.05$ ) in values (mean  $\pm$  SE) of monocytes and eosinophils count, and a highly significant increase ( $P<0.01$ ) in the values (mean  $\pm$  SE) of leucocytes and neutrophils.

**Keywords:** ruminal lactic acidosis (RLA), incidence, rumen fluid analysis, haemato-biochemical analysis, treatment

**1. Introduction**

Rumen acidosis can cause significant morbidity and mortality in small ruminants, making recognition of the disease important in the field <sup>[1]</sup>. Highly fermentable carbohydrate diet when fed in excessive quantities or gradual increase in concentrate feeding over a long period in ruminants results in reduction of ruminal pH favouring the multiplication of amylolytic bacteria with production of large quantity of lactic acid and long chained volatile fatty acids (VFA) <sup>[2]</sup>. Since lactate and VFAs increase the rumen osmolarity decreasing absorption of lactate and VFAs, leading to buildup of these compounds with a continued drop in pH <sup>[3]</sup>. VFAs and lactate within the rumen fluid are subsequently absorbed into the systemic circulation <sup>[4]</sup>. This results in ruminal acidosis, the degree of which may vary from mild to severe signs and leads to indigestion, rumen stasis, circulatory disturbance, decreased renal perfusion, dehydration, acidemia, hypovolumic shock, toxemia and frequent death <sup>[2], [5]</sup>.

In addition, endotoxins and histamines are released and may cause secondary complications like rumenitis, liver abscesses and laminitis <sup>[6], [7]</sup>. Severe dehydration and cardiovascular changes are common in addition to biochemical changes <sup>[8], [9]</sup>. Since the incidence of ruminal acidosis varies with feeding and grazing management, the present study reports incidence of ruminal lactic acidosis and associated haemato-biochemical changes in goats managed in Tirunelveli district of Tamil Nadu.

**2. Materials and Methods**

Out of 2050 goats, irrespective of their age (2 months to 6 years old), sex and breed (Salem Black, Kanni, Kodi and non-descriptive) brought to the Large Animal Medicine section of Veterinary Clinical Complex, Veterinary College and Research Institute, Tirunelveli for a period of 6 months, 62 goats had a history of feeding rice grain as part of diet by the farmers or accidental ingestion of cooked rice while grazing and signs like anorexia, depression, weakness, bloat, constipation, anuria and pastry or watery diarrhoea. The clinical signs observed in these cases were increased respiratory and heart rate, nasal discharge, low rectal temperature, reduced ruminal motility, fluid splashy rumen by ballottement, regurgitation of ruminal contents, mild to severe dehydration and sternal or lateral recumbency.

Rumen fluid was collected by stomach tube/ ruminocentesis for the identification of pH, colour, odour, consistency and protozoal activity (Table 1 and Fig 1).

The pH of ruminal fluid was measured immediately with the help of a pH indicator paper and protozoal activity was observed under low power microscope. The rumen fluid with pH  $\leq 6.0$  and no protozoa or very few with sluggish movement were considered positive for lactic acidosis<sup>[10]</sup>.

About 5 ml of whole blood was collected in EDTA containing vials for the estimation of haemoglobin (Hb), packed cell volume (PCV), white blood cells (WBC), differential leucocyte count (DLC) and total erythrocyte count (TEC) and platelet count in cases of ruminal lactic acidosis (n=57) and apparently healthy goats as controls (n=10). Serum variables like blood urea nitrogen (BUN), total protein, albumin, alanine transaminase (ALT), aspartate transaminase (AST), alkaline phosphatase (ALP), potassium, sodium, chloride and glucose were estimated by automated analyser in cases of ruminal lactic acidosis (n= 50) apparently healthy goats (controls, n=10). Haemato-biochemical parameters (Table 2) were analysed statistically (Student t test) using SPSS software.

### 3. Results and Discussion

The clinical signs observed varied from mild to severe or per acute, acute and subacute. The clinical findings like fluid-splasy rumen due to accumulation of large quantity of fluid in the rumen, low rectal temperature due to dehydration or depression of cardiovascular system, increased heart rate due to endotoxaemia, increased respiratory rate due to increased carbon-dioxide tension of blood and dehydration due to movement of excessive quantities of fluid into the rumen are in accordance with that of Constable *et al.*<sup>[10], [2]</sup>.

Out of 2050 goats brought for the treatment of various ailments, ruminal lactic acidosis was recorded in 3.0 per cent. The incidence was higher than that of Mahmood *et al.*,<sup>[11]</sup> who recorded an incidence of 1.2 per cent for lactic acidosis in goats. Where as, Gonzalez *et al.*<sup>[12]</sup> reported a much higher incidence of 18.0 per cent in goat herds. The low incidence in this study might be attributed to extensive grazing and feeding

management. Analysis of rumen liquor, complete blood count (CBC) and serum biochemical analysis could be useful not only in clinical diagnosis, assessment of severity and physiological imbalance, but also to determine the prognosis and therapeutic interventions<sup>[1]</sup>.

#### 3.1 Rumen Fluid Analysis

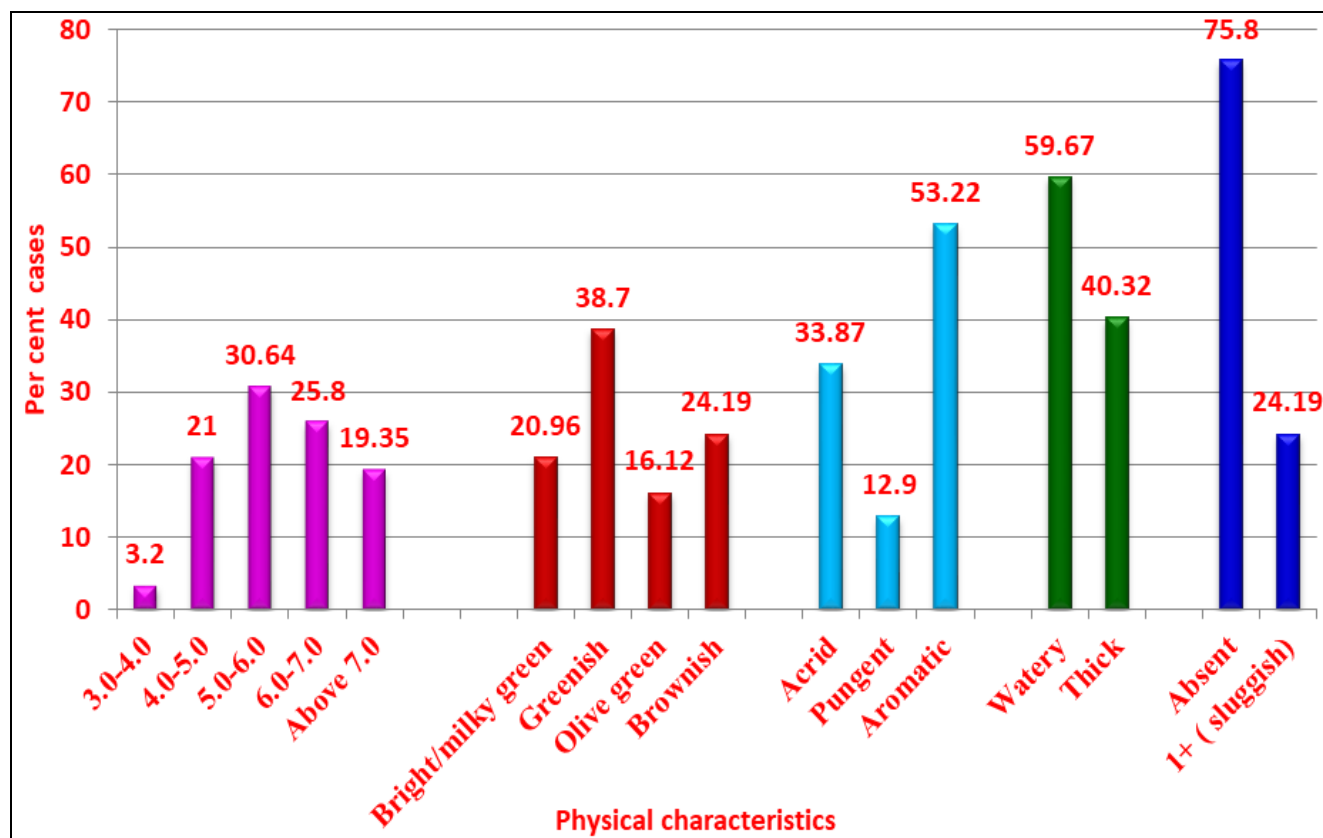
Majority of the cases (30.64%) had the rumen pH with the range of 5.0-6.0 followed by 25.8% with a pH range of 6.0 - 7.0, 21.0% with a pH range 4.0-5.0, 19.35% with pH above 7.0 and 3.2% with a pH range of 3.0-4.0 (Table 1 and Fig 1). However, Mahmood *et al.*<sup>[11]</sup>, reported majority of the cases (11.9%) to have a healthy range of rumen pH (above 6.0). The low pH and motility in rumen in acidotic cases might be due to excess accumulation of lactic acid in rumen associated with the rapid fermentation of carbohydrates by proliferation of acid resistant bacteria (*Lactobacillus* and *Streptococcus bovis*) and an increase in the production of volatile fatty acids and lactate<sup>[12]</sup>. The inconclusive evidence of or increased pH in some of the acidotic cases might be due to the practice of oral administration by the farmers of herbal products or baking soda or beetal leaves as a first aid in the farm premises.

The colour of the rumen fluid was greenish in majority of the cases (38.7%) followed by brownish in 24.9% cases, milky grey in 20.96% cases and olive green in 16.12% cases. The odour of the rumen fluid was acrid in 33.87% cases and pungent in 12.9% cases, whereas aromatic in 53.22 % cases. The consistency of the rumen fluid was watery in 59.67% cases and thick in 40.32% cases. These findings are concurrent to that of Tufani *et al.*<sup>[5]</sup>.

The rumen protozoa was absent in majority of the cases (75.8%) followed by the presence of 1+ concentration in 24.19% cases. The defaunation or reduction in rumen protozoa in terms of number and motility in acidotic cases associated with low pH is in agreement with that of previous reports<sup>[13] [5]</sup>.

**Table 1:** Physical properties of the rumen fluid collected from the Ruminal lactic acidosis

S. No.	Properties of rumen fluid	Changes observed	No. of cases (n=62)	Morbidity (%)
1.	pH	3.0-4.0	02	3.2
2.		4.0-5.0	13	21.0
3.		5.0-6.0	19	30.64
4.		6.0-7.0	16	25.8
5.		Above 7.0	12	19.35
1.	Colour	Milky grey	13	20.96
2.		Greenish	24	38.7
3.		Olive green	10	16.12
4.		Brownish	15	24.19
1.	Odour	Acrid	21	33.87
2.		Pungent	8	12.9
3.		Aromatic	33	53.22
1.	Consistency	Watery	37	59.67
2.		Thick	25	40.32
1.	Protozoa	Absent	47	75.80
2.		1+	15	24.19



**Fig 1:** Changes in the physical characteristics of rumen fluid collected from acidotic cases

### 3.2 Haematological Analysis

Haematological analysis revealed an increase in haemoglobin (Hb) in 60.0 per cent of acidotic cases, which is in accordance with that of Sarma and Nath<sup>[9]</sup> and this could be due to haemoconcentration associated with mild dehydration. Neutrophilia and leukocytosis were observed in 72.0 and 52.0 per cent of acidotic cases, respectively. Statistical analysis also revealed a highly significant increase ( $P < 0.01$ ) in the values (mean  $\pm$  SE) of leucocytes and neutrophils (Table 2). This could be attributed to an increase in the cortisol level during stress which causes an increase in the TLC<sup>[11]</sup>. A reduction in Hb, PCV, platelets and lymphocyte count was recorded in 10.0, 20.0, 16.0 and 66.0 per cent of the acidotic cases, respectively. However, statistical analysis revealed a significant increase ( $P < 0.05$ ) in the values (mean  $\pm$  SE) of PCV and decrease in values (mean  $\pm$  SE) of monocytes and eosinophils count (Table 2). The observed changes might be due to dehydration, malnutrition and associated immunosuppression.

### 3.3 Serum Biochemical Analysis

Serum biochemical analysis revealed an increase in BUN, creatinine, total protein, globulin, ALT, ALP, glucose, sodium, potassium, chloride and magnesium in 94.0, 10.0, 42.0, 32.0, 82.0, 4.0, 64.0, 32.0, 6.0, 44.0 and 22.0 per cent of the acidotic cases respectively. However, statistical analysis revealed a significant decrease ( $P < 0.05$ ) in the values (mean  $\pm$  SE) of globulin and ALP, and significant increase in ( $P < 0.05$ ) in the values (mean  $\pm$  SE) of creatinine, ALT, AST, phosphorus and glucose (Table 2). Whereas statistically, a

highly significant increase ( $P < 0.01$ ) in the values of BUN and chloride could be observed (Table 2).

Increase in total protein and globulin may be attributed to dehydration associated with movement of water from the vascular system into the rumen<sup>[14]</sup>. The hyperglycaemia could be attributed to low utilization of glucose by the peripheral tissues and hepatic glycogenolysis as a result of hyperactive adrenal medulla and low levels of immune reactive insulin<sup>[15]</sup>.

The hypernatremia, hyperkalemia, hypermagnesemia might be attributed to haemoconcentration, where as, elevated ALT and ALP levels could be associated with hepatocellular damage due to toxic products like alcohol, histamine, thiaminase and endotoxins entering the portal circulation from the rumen<sup>[10]</sup>. Elevated levels of BUN and creatinine could be due to reduced glomerular filtration rate associated with impaired renal perfusion and arterial blood pressure<sup>[16]</sup>.

However, a reduction in total protein, albumin, globulin, calcium, phosphorus, glucose, potassium, sodium, chloride and magnesium could be observed in 28.0, 30.0, 26.0, 16.0, 34.0, 20.0, 32.0, 10.0, 8.0 and 18.0 per cent of acidotic cases, respectively. Hypoproteinemia, hypoalbuminemia, hypoglobulinemia, hypophosphatemia, hypoglycaemia and hypomagnesemia and hypocalcaemia might be due to malnutrition or diarrhoea<sup>[10]</sup>, and hyponatremia and hypochloraemia may be due to the shift of these electrolytes by osmolarity from the blood to rumen or diarrhoeic losses<sup>[17]</sup>. Hypokalemia might be due to retention of sodium and excess excretion of potassium by the kidney<sup>[2]</sup>.

**Table 2:** Haemato-biochemical values (mean  $\pm$  SD) with statistical analysis associated with ruminal lactic acidosis in goats

Values	Acidotic cases	Controls	P Value
<b>Haematological values (Mean <math>\pm</math> SE)</b>			
Hb (g/dl)	12.940 $\pm$ 1.441	14.536 $\pm$ 0.427	0.311
PCV (%)	25.540 $\pm$ 0.946	28.830 $\pm$ 1.259	0.042
RBC ( $\times 10^6$ /ul)	12.150 $\pm$ 0.623	11.143 $\pm$ 0.348	0.179
WBC ( $\times 10^3$ /ul)	18510.000 $\pm$ 1742.760	38438.596 $\pm$ 4540.027	< 0.001
Platelet ( $\times 10^5$ /ul)	401800.000 $\pm$ 16980.250	368810.526 $\pm$ 10151.294	0.115
Neutrophils (%)	28.800 $\pm$ 0.712	57.719 $\pm$ 2.564	< 0.001
Lymphocytes (%)	65.100 $\pm$ 0.348	38.947 $\pm$ 2.360	< 0.001
Monocytes (%)	2.300 $\pm$ 0.396	1.175 $\pm$ 0.159	0.022
Eosinophils (%)	3.800 $\pm$ 0.389	2.526 $\pm$ 0.230	0.012
Basophils (%)	-	-	-
<b>Biochemical values (Mean <math>\pm</math> SE)</b>			
BUN (mg/dl)	27.118 $\pm$ 2.709	58.527 $\pm$ 6.196	< 0.001
Creatinine (mg/dl)	0.900 $\pm$ 0.042	1.330 $\pm$ 0.124	0.002
Total protein (g/dl)	7.230 $\pm$ 0.242	6.686 $\pm$ 0.150	0.073
Albumin (g/dl)	2.800 $\pm$ 0.304	3.064 $\pm$ 0.118	0.434
ALT (u/l)	4.430 $\pm$ 0.287	3.620 $\pm$ 0.202	0.033
AST (u/l)	22.600 $\pm$ 2.252	41.080 $\pm$ 6.955	0.014
ALP (u/l)	68.100 $\pm$ 13.902	119.140 $\pm$ 7.629	0.006
Calcium (mg/dl)	376.700 $\pm$ 62.365	186.360 $\pm$ 15.395	0.014
Phosphorous (mg/dl)	10.540 $\pm$ 0.363	11.294 $\pm$ 1.423	0.610
Glucose (mg/dl)	3.930 $\pm$ 0.480	6.286 $\pm$ 0.565	0.003
Sodium (meq/l)	70.970 $\pm$ 12.298	137.130 $\pm$ 16.119	0.002
Potassium (meq/l)	145.790 $\pm$ 3.429	151.420 $\pm$ 2.077	0.179
Potassium	3.698 $\pm$ 0.461	4.183 $\pm$ 0.243	0.367
Chloride	92.192 $\pm$ 1.872	107.110 $\pm$ 2.489	< 0.001
Magnesium	7.030 $\pm$ 3.330	3.330 $\pm$ 0.149	0.295

### 3.4 Treatment

The cases were treated with slow intravenous administration of isotonic sodium bicarbonate solution in dextrose normal saline [6] to prevent metabolic acidosis followed by daily administration of Pheneramine maleate @ 0.5 mg/kg body weight, IM, as an antihistaminic, flunixin meglumine to prevent toxemic effects, B-complex as a source of thiamine by IV, oral oxytetracycline hydrochloride @ 250 -1000 mg to control lactic acid producing bacteria until recovery. Thirty one cases were orally administered with a herbal preparation, Lithosacc (SynPro®) -37 grams containing a rumen buffer and live yeast, *Saccharomyces cereveciae* @ 20 billion cfu powder once on first day in addition to the above treatment and the rumen pH on the next day remained normal than in cases without treatment using herbal preparation which took three days.

### 4. Conclusion

Prevention of rumen acidosis aims at management practices like restricted access to high concentrate ration, avoiding sudden changes in the diet since the adaptation of the rumen microbes to new feed might take several weeks. Acute ruminal lactic acidosis resulted in significant alterations in the haematological and biochemical values reflecting significant deviation in the health status. The prognosis of acute acidotic cases presented for treatment in the late stage becomes generally grave due to severe acedemic status. Hence, an early treatment of cases of ruminal acidosis is necessary to prevent a fatal outcome.

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