IJAS Iranian Journal of Applied Animal Science

Rumen Acidosis in Small Ruminants and Its Therapeutic Management

Research Article

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Received on: 10 Nov 2011
Revised on: 29 Jan 2012
Accepted on: 8 Feb 2012
Online Published on: Mar 2013

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Online version is available on: www.ijas.ir

ABSTRACT

Forty two small ruminants, 26 (61.90%) sheep and 16 (38.10%) goats were treated for rumen acidosis. 19 (45.24%), 12 (28.57%), 6 (14.29%) and 5 (11.90%) animals had eaten apple, cooked rice (wazwan left over) turnip and chapatti respectively and manifested clinical form of ruminal acidosis with, 2.86%; (n=18) as mild (Rumen fluid pH=6.32 ±0.09316), 38.09%; (n=16) moderate (Rumen fluid pH=5.71 ±0.074) and 19.05%; (n=8) (Rumen fluid pH=4.54 ±0.159) as severe ruminal acidosis and accordingly they were classified as group I, II and III, respectively. In group I rumen motility was reduced (1.67±0.162) and subsequently it was almost absent in group III (0.13±0.125). Rectal temperature recorded to be 102.58±0.166, 101.26±0.188 and 100.83±1.061 in group I, II and III respectively. There was a significant increase heart and respiratory tares before treatment in all the groups. A significant increase in blood glucose and Hb, PCV and TEC was also observed in mild, moderate and severe acidotic animals. Therapeutic measures consisted of neutralization of acidity by oral and / or parenteral use of isotonic (1.3%) and hypertonic (5%) sodium bicarbonate with adequate fluid therapy. Oral and parenteral use of isotonic (1.3%) sodium bicarbonate was given to moderate rumen acidosis. Administration of oral sodium bicarbonate, bolus Rumentas was given to groups I and II and a course of antihistaminic drug was also given to all the groups of animals. Moreover, animals of group II and III were also offered intravenous injection of high dose vitamin B_1 along with fluids. Gastric lavage and cud transplantation following rumenotomy was done in animals of group III. All animals except two (one each from group II and III) were recovered uneventfully.

KEY WORDS grain overload, rumen acidosis, small ruminants, treatment.

INTRODUCTION

Small ruminants significantly contribute to agrarian economy and play a vital role in lively hood security of marginal and landless farmers of Kashmir valley. They provide household nutritional security and family income through meat, wool / fiber, skin, milk and manure with little or no feed supplementation. Mostly these animals are reared under free range system in this locality. In summer they are being grazed in high land pasture and during winter fed with hay, tree leaves and agriculture by products under household condition.

The Kashmir valley is known for fruit industry especially for apple. The animals while grazing in apple orchards consume rotten apple which causes sub acute to acute form of rumen acidosis. In marriage seasons the left over of the special feast wazwan (a mixture of rice, meat, multiple spices and vegetables) is left over in the lanes of Kashmir. The small ruminants easily consume it and develop acute rumen acidosis resulting in heavy economic losses due to

high morbidity and mortality. Now, it becomes a serious problem for the free range small ruminants.

Acidosis also known as lactic acidosis, rumen acidosis or grain overload is a carbohydrate fermentation disorder of the rumen that can affect sheep and goats of all breeds. As the name implies, acidosis results in acidic pH of rumen (normal being 6.2-6.8). Acidosis is caused by the feeding of highly fermentable carbohydrates, feeding of low fiber diet, poor management practices or a combination of these. Degree of acidosis varies from seriousness, a slight drop in feed intake (mild) to death (severe). Acute form of the disease in ruminants is characterized by indigestion, rumen stasis, dehydration, acidosis, toxemia, incoordination, collapse and frequently death.

It is one of the most important clinical emergencies in small ruminants (sheep and goats) and results in high mortality (Radostits *et al*. 2000). Lactic acid is known to be increased in the rumen from 1 to 1500 mg/100 mL (Uhart and Carrol, 1967; Walker, 1968) and in the blood from 4.5 to 90 mg/100 mL (Heuter *et al*. 1956; Dunlop and Hammond, 1965) following consumption of large quantity of grain. In addition, a large proportion of the lactic acid may not be metabolized systemically (Dunlop and Hammond, 1965). Systemic changes occurring during lactic acidosis require greater understanding for proper treatment and preventive measures to reduce the risk of grain engorgement in ruminants.

Haematological changes in ruminal acidosis are important to assess the severity of the disease. Severe dehydration and cardiovascular involvement are common (Shihabudhin *et al*. 2003) in addition to biochemical changes (Sarma and Nath, 2005). The present study reports alterations in clinical parameters and haemato-biochemical changes in small ruminants suffering from ruminal acidosis due to consumption of rotten apple, turnip, left over of wazwan and chapattis with its therapeutic management.

MATERIALS AND METHODS

Sporadically, within a period of one year, a total of forty two small ruminants including 26 (61.90%) sheep and 16 (38.10%) goats irrespective of their sex and breed were brought to the Clinical Complex of the Faculty with history of accidental feeding of apple (19), turnip (6), left over of wazwan (12) and chapattis (5). During clinical examination, rectal temperature, rumen motility, heart rate and respiratory rate were taken. After rumenocentesis, approximately 2-5 mL of ruminal fluid was aspirated from left paralumbar fossa for the estimation of pH and protozoal activity. Ruminal fluid pH was measured immediately with the help of wide range pH indicator paper and protozoal activity was observed under low power microscope.

Approximately, 5 mL of whole blood was collected out of which 2 ml of blood was taken in EDTA (1.5 mg/mL) containing vials for the estimation of Hb, PCV and TEC as per method described by Jain (1986) and rest 3 mL of blood was kept for serum analysis. Blood glucose from serum was estimated within few hours by spectrophotometer. On the basis of severity of the disease the animals were randomly divided into three groups (I, II and III).

Group I (N=18, sheep=11 and goats=7) with mild acidosis were treated with sodium bicarbonate ($NaHCO_3$) @ 20 g stat orally followed by 5 g bid orally daily for 3 days and rumenotoric bolus Rumentas (Intas Pharmaceuticals) @ 1bolus bid orally daily for 3 days.

Group II (N=16, sheep=10 and goat=6) with moderate ruminal acidosis were treated with sodium bicarbonate ($NaHCO_3$) @ 20 g stat orally followed by 5 g bid orally daily for 3 days, bolus Rumentas (Intas Pharmaceuticals) @ 1bolus bid orally daily for 3 days, isotonic sodium bicarbonate (1.3%) @ 50 mL/kg body weight intravenously daily for 3 days and injection of Vitamin B_1, B_6 and B_{12} (Tribivet, Intas) @ 5-10 mL intravenously along with drip daily for 3 days.

Group III (N=8, sheep=5 and goat=3) with severely affected animals were treated with hypertonic sodium bicarbonate (5%) @ 10 mL/kg body weight intravenously stat followed isotonic sodium bicarbonate (1.3%) @ 50 ml/kg body weight intravenously daily for 2 days and injection Vitamin B_1, B_6 and B_{12} (Tribivet, Intas) @ 5-10 mL intravenously along with normal saline @ 100 mL/kg body weight daily for 3 days. In this group of animals emergency rumenotomy was carried out using standard operative procedure. The contents of rumen were evacuated and the rumen was lavaged with 5% sodium bicarbonate solution. Fresh ruminal cud from slaughter house was collected and transplanted into rumen. Post operatively the animals were given intramuscular injection of broad spectrum antibiotic (Ceftriaxone @ 5 mg/kg body weight) and pain killer (Meloxicam @ 0.2 mg/kg body weight) daily for 5 days. A course of antihistaminic drug (Pheniramine maliate) was also given @ 2-3 mL intramuscularly daily for 3 days to all the groups of animals. The data obtained were analyzed statically (analysis of variance and t-test) per Snedecor and Cochran (1994).

RESULTS AND DISCUSSION

On the basis of history, clinical and laboratory examination all the forty two animals were diagnosed as rumen acidosis. Out of which 26 (61.90%) sheep and 16 (38.10%) goats were involved ruminal acidosis and were treated according to the severity of the disease. On the basis of history it was observed that 19 (45.24%) animals had eaten apple, 12

(28.57%) animals had eaten left over of wazwan, 6 (14.29%) animals had consumed turnip and 5 (11.90%) had eaten chapatti, which were showing the clinical syndrome of ruminal acidosis. Following ingestion of large quantity of highly fermentable carbohydrate rich diet, the lactate producing rumen bacteria (*Streptococcus spp*) proliferate and ferment the readily available carbohydrate resulting in excess accumulation of lactic acid in the rumen and its subsequent absorption into blood circulation causes systemic acidosis. Clinically it was observed that 18 (42.86%) animals affected only with mild ruminal acidosis could be due to less consumption of fermentable carbohydrate rich diet and/or early presentation for treatment. However, 16 (38.09%) animals were showing moderate clinical form of ruminal acidosis and only a few animals, 8 (19.05%), were clinically affected with severe form of lactic acidosis due to excess ingestion of highly fermentable carbohydrates and/or delayed presentation for treatment. Animals of group III were put to emergency rumenotomy followed by therapeutic management. Even though, 1 goat of this group III had died due to cardiovascular arrest as a result of severe dehydration. Animals with severe acidosis showed 87.5% (7/8) survival in the present study. There was significant difference in various clinical, haemato-biochemical parameters between and within groups before and after treatment, aimed at returning the measured parameters close to normal levels in accordance to Radostits *et al*. (2000), in acidotic animals (Table 1). After exhaustive therapeutic measures all the recovered animals, except two (one each from group II and III) died even after treatment, were found active and alert and also found physiologically and clinically normal like healthy animals within a week of treatment. In the mild form of rumen acidosis (group I), the rumen motility was reduced (1.67±0.162) but not entirely absent and the animals were anorectic but bright and alert and diarrhea was common. In severe acidosis (group III), 4 animals were recumbent with severe dehydration, 3 were staggering and 1 was standing quietly with complete anorexia but rumen motility was discernable and exhibited distension of abdomen of left flank. In addition, the primary contractions of the rumen was completely absent, although the gurgling sounds of gas rising through the large quantity of fluid was usually audible on auscultation. The contents of the rumen, as palpated through the left paralumbar fossa, may feel firm and doughy in initial stage particularly in group I. Fluid-splashing sounds in the rumen could be due to accumulation of large quantity of fluid (Radostits *et al*. 2000) particularly in severe cases at the terminal stage. However, the clinical signs of group II animals were mixed type as in group I and II. Clinically the rectal temperature (˚F) was measured below normal with mean values of 102.58±0.166 in group I, 101.26±0.188 in group II and 100.83±1.061 in group III before treatment. In severe case of acidosis, some animals exposed to the sun in hot weather, temperature may be increased to 106 ˚F could be due to failure of thermoregulatory centre (hypothalamus) of brain (Radostits *et al*. 2000). The decreased level of rectal temperature, which was in accordance to Nour *et al*. (1998), may be due to lactic acidosis, leading to dehydration; fall in total plasma volume and severe depression of cardiovascular system (Dunlop and Hammond, 1965). The heart rate significantly increased, might be due to toxic effects of lactic acid, reduced plasma volume and circulatory failure (Radostits *et al*. 2000), in accordance with severity of the acidosis (Table 1) and may goes up to 155/min in severe cases. The heart rate became almost normal in all the treated groups within 3-7 days depending upon the severity of the case. There was significant increase in respirations which was shallow and rapid and increased up to 46/min in moderate (35.06±1.521) and severe (38.13±2.207) cases. Similar, increase in respiratory rates has been reported by Tanwar and Mathur (1983) and Ram *et al*. (2007) in acidotic animals. This increase in respiration might be due to stimulation of respiratory centre by increased carbon-dioxide (CO_2) tension of blood and decreased blood pH (Huber, 1976). The ruminal fluid of affected animals was milky, had a sour odou.

There was significant decrease in mean pH of rumen fluid in all the groups and fall to 4.54±0.159 in severe acidosis and at this low pH almost entire rumen protozoal activity ceases and no live protozoal population was observed under low power microscope. This great change in the microbial population in the rumen following ingestion of toxic amounts of highly fermentable carbohydrates occurs within 2-6 hours. The number of gram positive bacteria (*Streptococcus spp*) increased markedly, which resulted in the production of large quantities of lactic acid. The rumen pH falling to or below 5, destroying protozoa, cellulolytic organisms, and lactate-utilizing organisms, and severely impaired rumen motility. This low pH further allows the lactobacilli to utilize the carbohydrate and to produce excessive quantities of lactic acid. The superimposition of lactic acid and its salt, lactate, on the existing solutes in the rumen liquid causes osmotic pressure to rise substantially, which results in the movement of excessive quantities of fluid into the rumen and ultimately moderate to severe dehydration followed (Radostits *et al*. 2000). Evacuation of rumen and transplantation of fresh rumen cud following surgical procedure in group III animals helped restitution of normal function of rumen. There was significant increase in blood glucose level (mg/dL) in mild (67.89±1.078), moderate (83.25±0.951) and severe (92.88±1.217) acidotic sheep and goats and differs significantly among the groups in accordance to Kasaralikar *et al*. (2007).

Table 1 Changes in clinical and haemato-biochemical parameters observed before and after treatment (Mean±SE)

Parameters	Group I (n=18)	Group II (n=16)†	Group III (n=8)†	Overall (N=42)†
Clinical parameters‡				
Mortality	0	1 (6.25%)	1 (12.5%)	2 (4.76%)
Temperature (˚F)				
Before treatment	102.58^{a}±0.166	101.26^{b}±0.188	100.83^{b}±1.061	101.74±0.244
After treatment	102.42±0.074^{ns}	102.13±0.112**	102.17±0.263^{NS}	102.27±0.071**
Heart rate / min				
Before treatment	95.17^{a}±2.576	127.75^{b} ±2.032	133.75^{b} ±5.028	114.93±3.136
After treatment	80.72^{a} ±2.507**	88.60^{b} ±1.641**	90.57^{b} ±3.258**	85.40±1.534**
Respiratory rate / min				
Before treatment	27.61^{a}±0.964	35.06^{b}±1.521	38.13^{b}±2.207	32.45±1.051
After treatment	21.78^{a}±0.952**	25.60^{b}±1.206**	27.86^{b}±0.937**	24.28±0.737**
Rumen motility / 2 min				
Before treatment	1.67^{a}±0.162	0.75^{b}±0.144	0.13^{c}±0.125	1.02±0.130
After treatment	3.61^{a}±0.200**	3.00^{ab}±0.169**	2.57^{b}±0.297**	3.20±0.135**
Rumen fluid pH				
Before treatment	6.32^{a}±0.093	5.71^{b}±0.074	4.54^{c}±0.159	5.75±0.116
After treatment	6.93^{a}±0.079**	6.65^{ab}±0.094**	6.34^{b}±0.162**	6.72±0.065**
Protozoal motility‡				
Before treatment				
Absent	0	5 (31.25%)	7 (87.50%)	12 (28.27%)
Mild	10 (55.56%)	10 (62.50%)	1 (12.50%)	21 (50.00%)
Moderate	7 (38.89%)	1 (6.25%)	0	8 (19.05%)
High	1 (5.55%)	0	0	1 (2.38%)
After treatment				
Absent	0	0	0	0
Mild	1 (5.55%)	5 (33.33%)	0	6 (15.00%)
Moderate	7 (38.89%)	7 (46.67%)	6 (85.71%)	20 (50.00%)
High	10 (55.56%)	3 (20.00%)	1 (14.29%)	14 (35.00%)
Haematochemical parameters‡				
Blood glucose (mg/dL)				
Before treatment	67.89^{a}±1.078	83.25^{b}±0.951	92.88^{c}±1.217	78.50±1.652
After treatment	57.00±0.950**	57.53±0.844**	57.43±0.649**	57.28±0.535**
Haemoglobin (g/dL)				
Before treatment	14.09^{a}±0.167	15.06^{b}±0.217	15.90^{c}±0.204	14.81±0.156
After treatment	13.31±0.239**	13.99±0.201**	13.74±0.295**	13.64±0.147**
PCV (%)				
Before treatment	41.28^{a}±0.651	44.88^{b}±0.598	49.63^{c}±0.730	44.24±0.610
After treatment	37.33±0.813**	38.53±0.593**	37.57±0.997**	37.83±0.460**
TEC (million/cmm)				
Before treatment	9.60^{a}±0.127	10.79^{b}±0.173	12.50^{c}±0.370	10.61±0.197
After treatment	8.50^{a}±0.125**	9.49^{b}±0.129**	9.26^{b}±0.429**	9.01±0.125**

† Two animals, one each from group-II and III were died even after treatment.

‡ The means within the same row with at least one common letter, do not have significant difference (P>0.05).

** The means within the same column with at least one common letter, do not have significant difference (P>0.05).

NS: none significant.

This might be due to decreased utilization of glucose by the peripheral tissues and hyperglycaemic response in acidotic animals due to hepatic glycogenolysis owing to hyperactive adrenal medulla under acidotic stress and decreased immune reactive insulin (Basak *et al*. 1994 and Kaneko *et al*. 1999).

After exhaustive therapeutic measures the blood glucose levels become normal in all the groups of animals (Table 1).

The significant increase in Hb, PCV and TEC before treatment in mild, moderate and severe acidotic animals (Table 1), as in accordance to Sarma and Nath (2005) and Shihabudhin *et al*. (2003), could be due to haemoconcentration as a result of mild (4-6%) to severe (up to 10-12%) dehydration following drawing of systemic fluid in the rumen and profuse diarrhoea (Radostits *et al*. 2000). The levels of Hb, PCV and TEC become normal in all the groups of animals after therapeutic measures in all the groups.

In the present study, therapeutic measures consisted of neutralization of acidity by oral or both by oral and parenteral use of isotonic (1.3%) and hypertonic (5%) sodium bicarbonate and by rigorous fluid therapy to correct the acidosis and dehydration and to restore renal function. Initially, over a period of 30 min, 5% sodium bicarbonate solution was given intravenously (10 mL/kg) followed by balanced electrolyte solution (100 mL/kg), and 1.3% solution of sodium bicarbonate given intravenously (50 mL/kg) in severe case (group I) of acidosis till urination resume and to combat systemic acidosis. In moderate cases (group II), isotonic sodium bicarbonate (1.3%) @ 50 mL/kg intravenously was given to combat systemic acidosis. Oral sodium bicarbonate 20 g stat followed by 5 g bid for 3 days in all the groups were administered for neutralizing the lactic acids produced locally inside the rumen, to prevent chemical rumenitis and to restore normal rumen pH. Rumenotoric drug (bolus Rumentas) was given to restore rumen motility and appetite as well, in all the groups of animals. A course of antihistaminic drug was also given to all the groups of animals to counteract histamine released due to chemical rumenitis. Moreover, animals of group II and III were also offered intravenous injection of high dose thiamin to prevent its deficiency by abnormal growth of thiaminase enzyme producing bacteria inside the rumen. Thiamin causes metabolism of lactic acid and thus helps in preventing further systemic lactic acidosis. Almost all animals except two (one each from group II and III) were recovered completely and become normal within 3-7 days. These therapeutic measures can be employed for mild, moderate and severe cases of ruminal acidosis in sheep and goats, pertinently the animals with sever form of acidosis need to be put to ruminal lavage with the cud transplant to restore early recovery. Moreover, review your feeding program and management practices to prevent future problems. The key to prevention is a properly balanced diet and proper feeding management practices. First and foremost, feed animals a forage-based diet. These forages can be in the forms of natural browse and pasture or good-quality hay. Always provide forages on a free choice basis. Healthy animals should spend a significant portion of their time chewing their cud. If they are not, increase forage amounts. Avoid too much grain or commercial feed. Grains and some commercial feeds contain high levels of rapidly fermentable carbohydrates and / or sugars (molasses) that can upset the delicate balance of rumen microflora and cause acidosis as explained above due feeding of apple, wazwan, turnip and chapattis.

When introducing a new ration, gradually change the diet over a period of several weeks. Never make rapid changes to the diet of a ruminant. It will upset the microflora balance and could lead to acidosis. Also, when feeding grains or commercial feeds, it is best to split up the daily amount into 2 or 3 feedings per day rather than one “slug feeding” per day. This is especially important for animals that are receiving relatively large amounts of concentrates (over 25% of their daily diet).

CONCLUSION

In conclusion, acidosis can be a problem in all types of sheep and goats. Acidosis is caused by improper feeding practices. As a rule, always provide good to excellent quality forages to goats on a free choice basis and always offer less than half the total diet in concentrates to help avoid acidosis. In this clinical trial, it can be concluded that free range animals are usually and easily consumed rotten apple in orchard and left over wazwan that causes mild to severe lactic acidosis and death may occurs in severe condition leading to heavy economic losses to the poor farmers in the form of death and production loss. It is therefore, suggested that animals should rear under intensive farming for proper care and management and to avoid unnecessary or accidental feeding of apple, turnip, wazwan and chapattis. Moreover, the respective treatment employed for mild, moderate and severe ruminal acidosis is very much effective and should be applied in field condition.

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