INTRODUCTION

Ruminants are suffering from many infectious and non-infectious diseases from their primitive stages. Ruminal acidosis is one of the most important non-infectious diseases in ruminants. Many of the species are suffered from ruminal acidosis due to improper practice of feeding resulting from the lack of knowledge about risk factors. Acidosis mainly occurs when a ruminant intake large amount of rapidly fermentable carbohydrates, primarily starch and sugars. This conditions increases the morbidity and mortality of stock, markedly reduces weight gain of the feedlot which is increasingly recognized in rustic and confined dairying.

Ruminal acidosis is a pathological condition associated with the accumulation of acid or depletion of alkaline reserves in body tissues and characterized by increased ion concentrations (Crichlow and Caplin, 1985). It is a series of conditions that reflect a decrease in P^H in the rumen. It develops when ingestion of large quantities of highly fermentable carbohydrates (starches, grains) and faster progression of acid producing bacteria such as *Lactobacillus lactis* or Streptococcal grow faster than other ruminal microflora (Russel and Hino, 1985).

Lactobacillus lactis and *Streptococcus bovis* can produce more lactic acid which exceeds the buffering capacity of rumen fluid. The increase of volatile fatty acid (VFA) succeeding decrease of rumen motility which leads to less rumination as well as less saliva production and comparatively reduce the bicarbonate concentration in the body of the ruminants (Ahrens 1967; Lee et al., 1982). Saliva also contains bicarbonate ion. So due to lack of bicarbonate and rise of lactic acid concentration the ruminal P^H is fallen down from 6.5 to 4.5. This incident makes the ruminal environment hostile to the auspicious protozoa, fungi and other infectious pathogens, chemical changes occur in the epithelium of rumen mucosa. There occur depletion of normal microflora of rumen. Pathogenic bacteria and mycotic organism invade the rumen wall and causing ruminitis. Rumen papillae are damaged and begin to slough off from the rumen wall (Mullenax and Keelen, 1966). Absorption pattern changes, endotoxins and histamine release are turns

to higher during the process of this mechanism (Boltoon and Pass, 1988). Fluid also drawn out from blood vessel to rumen.

Ruminal acidosis can affect the ruminant of all ages and breeds. As the name denotes, acidosis results in the rumen P^H becoming acidic, where accumulation of H^+ ions from lactic acid, characterized by blood lactate levels >5 mmole/L and arterial P^H <7.25 (Robert et al., 1999). In most of the cases, the animal are supplied with wheat bran, cooked rice and other concentrates more than normal level for a few days. The cases was responded in (UVH, Hathazari) clinical manifestation range from loss of appetite to death. The systematic impact of acidosis may have several physiological implications including diarrhea, loss of weight gain and laminitis. Management of feeding and husbandry practices can be executed to reduce the incidence of diseases.

Therefore, this study was chosen to assess the acidosis case with follow up treatment at Upazila, Veterinary Hospital, Hathazari with the following objectives:

- To know the mal-practice of feeding in animals
- To identify the predisposing risk factors of ruminal acidosis through manifested clinical signs and treat accordingly
- To observe animal for quick recovery and giving suggestion for proper feeding habit and rearing system

MATERIALS AND METHODS

Area and Study Population

This study was conducted on ruminants residing at Hathazari Upazila, Chattogram. The animal were examined mostly were local cattle (indigenous) with some exotic cross breed (e.g. Friesian) and local and black Bengal goat. A total of 510 cases were treated in Upazila Veterinary Hospital (UVH), Hathazari Upazila, Chattogram during November, 2019 to January, 2020.

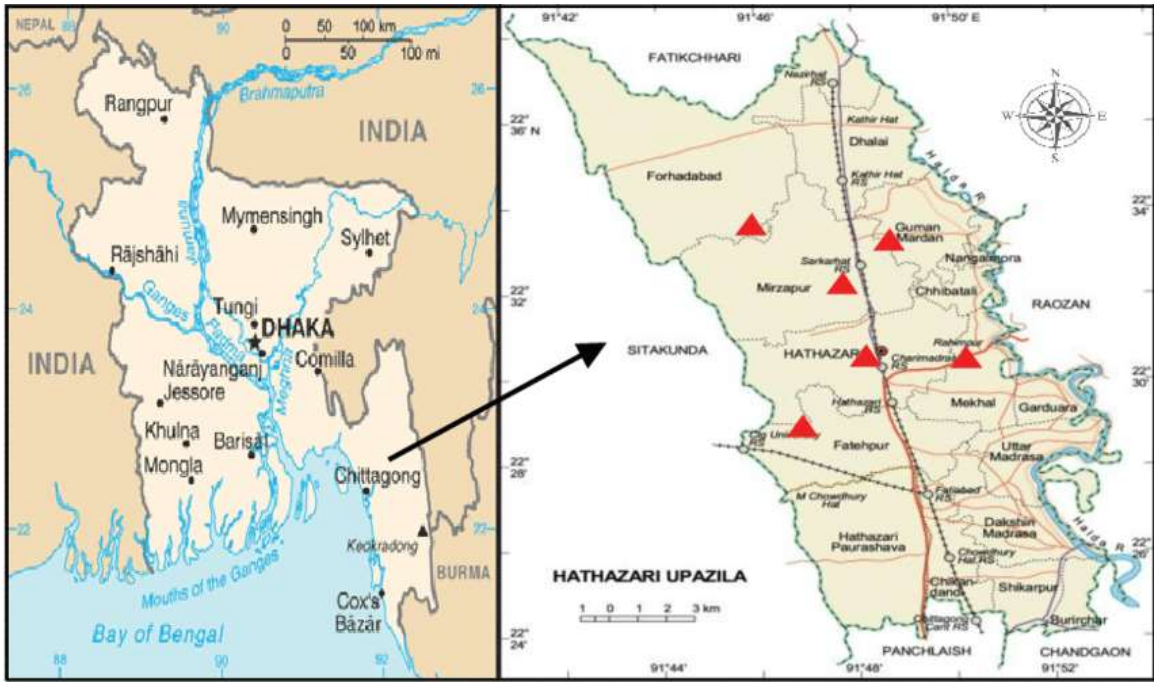
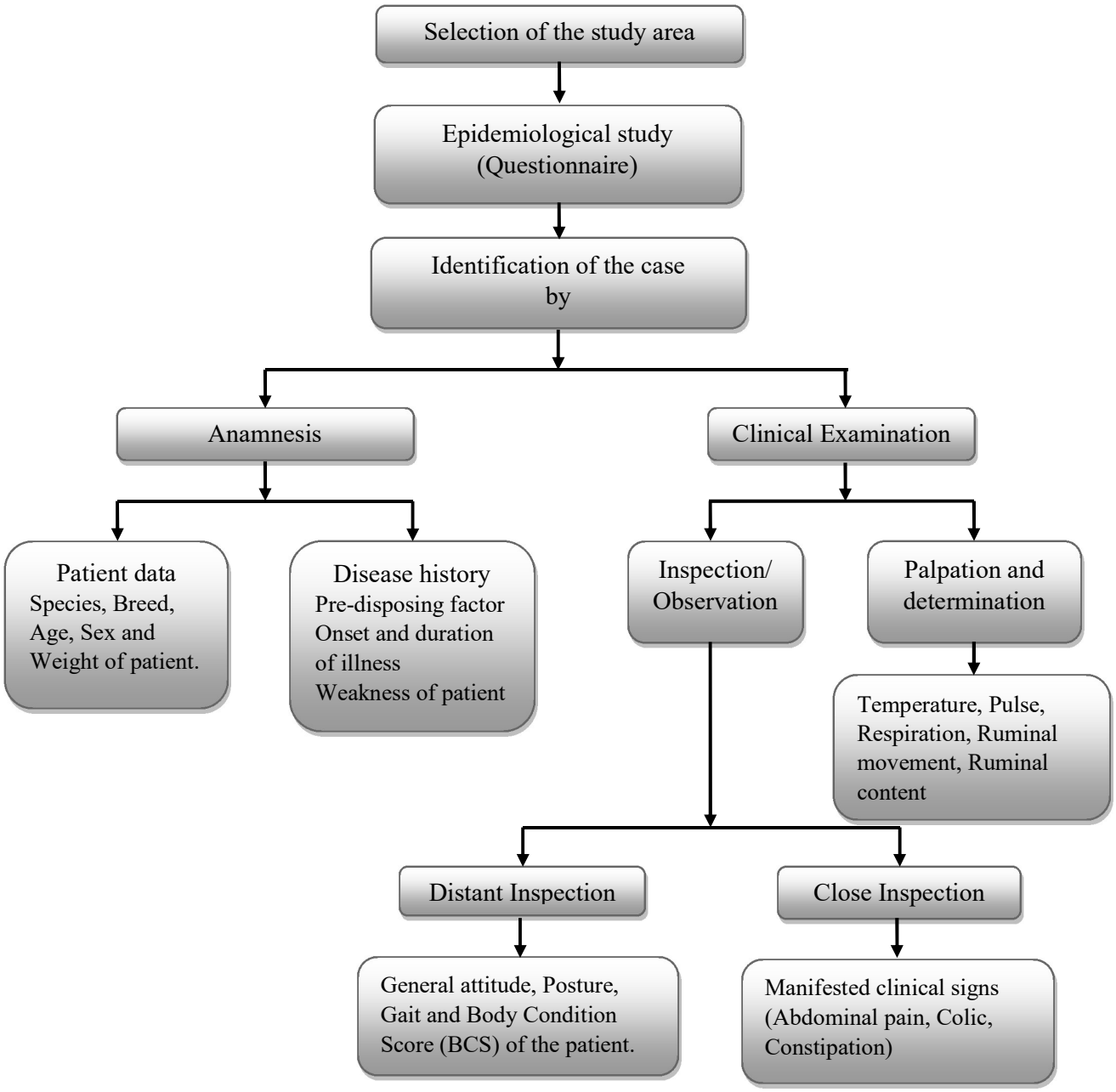


Figure 1: Study area

Study Lay out:



Data collection

A structured questionnaire was carefully prepared and this was filled up by repeated questioning to the animal owner, personal observation of patient. Important animal level data recorded including affected animals, species, breed, age, sex, body condition of the animal, weakness of the animal, onset and duration of illness, frequencies of infection and pre-disposing factors of the case (Thrusfield, 2005). Other information sought included history of de-worming and vaccination, pregnancy status, parity, housing pattern, type of the floor in the animal house (Katcha/ dirty floor/ muddy/ brick/ concrete/ rubber bedded), rearing system (intensive/ semi-intensive/ free-range) as well as whether or not a system of grazing or zero-grazing was practiced. Clinical examinations were performed according to questionnaire designed mentioning about temperature, respiration, general attitude of animal, posture, gait, wound, depthness of wound etc.

Case Identification and Diagnosis

i. Owner's complaint

ii. Anamnesis: History was taken about weakness of patient, onset and duration of illness from owner/ farmer and feeding history (feeding of easily digestible carbohydrates such as cooked rice, jackfruit residue, potato etc.)

iii. Clinical examination of patient; It includes:

Inspection

Distant Inspection: Firstly the general attitude of the patient (alertness/ dullness/ depression) was carefully inspected. Following this, the body condition of the animal (Cachectic/ poor/ fair/ good/ fat/ over fat) was observed as described by Radostitis et al., 2000. In addition, posture and gait (normal or defective) were examined according to the condition of the animal.

Close Inspection: Following distant inspection, the patient was closely examined where clinical signs includes complete anorexia, abdominal pain, rapid heart rate, abnormally fast breathing, diarrhea, constipation, lethargic star gaze and low ruminal P^H, low blood P^H and stop or reduced motility of microflora (Owens et al., 2009).

Collection of ruminal fluid

Ruminal fluid was collected by inserting the needle (14G, 5inch long needle) with single thrust in the rumen through the lower region of triangular flank area. Sometimes ruminal fluid was collected by using stomach tube. Therefore ruminal fluid color, consistency, odor, P^H and microflora per focus was examined cautiously.

Table 1: Interpretation among samples (Studied clinical parameters)

SL No.	Feeding history	Ruminal fluid color and P^H	Micro-flora count per focus	Feces color and consistency
1	Rice	Grayish and 4.5	7	Yellowish and Liquid
2	Rice	Grayish and 4.5	6	Yellowish and Liquid
3	Rice	Grayish and 4.6	6	Greenish and Semisolid
4	Rice	Yellowish and 4.7	7	Greenish and Semisolid
5	Wheat bran	Yellowish and 4.8	8	Greenish and Semisolid
6	Wheat bran	Grayish and 4.6	8	Yellowish and Semisolid
7	Wheat bran	Grayish and 4.5	7	Yellowish and Semisolid
8	Wheat bran	Grayish and 4.5	8	Yellowish and Semisolid
9	Cabbage	Yellowish and 4.6	7	Yellowish and Semisolid
10	Cabbage	Yellowish and 4.7	8	Greenish and Semisolid
11	Cabbage	Grayish and 4.8	7	Greenish and Semisolid
12	Cabbage	Grayish and 4.6	7	Greenish and Semisolid
13	Rice	Grayish and 4.5	7	Yellowish and Semisolid
14	Rice	Yellowish and 4.5	7	Yellowish and Semisolid
15	Jackfruit leaf	Grayish and 5.1	7	Blackish and Manure like
16	Jackfruit leaf	Grayish and 5.1	7	Blackish and Manure like
17	Jackfruit leaf	Grayish and 5.2	7	Blackish and Manure like
18	Jackfruit leaf	Grayish and 5.2	7	Blackish and Manure like
19	Jackfruit leaf	Grayish and 5.1	7	Blackish and Manure like
20	Jackfruit leaf	Grayish and 5.2	7	Blackish and Manure like

***Here 1-14 are cattle and 15-20 are goat sample**

Table 2: Treatment and associated dosage for four groups of animal

Groups	Animals	Treatment	Route
A	4 cattle and 2 goat	Ruminal alkalizer (Sodium bicarbonate @1g/kg BW)	Oral
B	4 cattle and 2 goat	Ruminal alkalizer (Sodium bicarbonate @1g/kg BW) and purgatives (Magnesium hydroxide @1g/kg BW)	Oral
C	3 cattle and 1 goat	Systemic alkalizer (5% sodium bicarbonate @11ml/kg BW)	I/V
D	3 cattle and 1 goat	Ruminal alkalizer (Sodium bicarbonate @1g/kg BW) and Systemic alkalizer (5% sodium bicarbonate @11ml/kg BW)	Oral and I/V
All four groups		Vitamin B complex (Vplex vet®) was given @ 5ml per animal per day for 5 days	I/V

BW= Body weight; I/V= Intra venous

Data Analysis

All the data that were collected (categorical variables like breed and continuous variables like age, BCS etc.) were entered into MS excel (Microsoft office excel-2007, USA). Descriptive analysis was done and to identify the association between a categorical explanatory variable with the outcome, chi-square (χ^2 test) test was performed. An association was regarded as significant if the p value was <0.05.

RESULT

The assessment of acidosis was evaluated on cattle and goat at upazila veterinary hospital (UVH), Hathazari under Chattogram district. A total of 510 were recorded during the study period and 20 animals (3.92%) were exclusively identified with ruminal acidosis.

The prevalence of ruminal acidosis in cattle and goat, breed, age were presented in Table 3 where there were no significant variation ($p > 0.05$). On the prevalence of species category the cattle 8.05% and goat 1.79%. In case of breed of cattle, prevalence of non-descriptive local breed was 5.17% and the Holstein Friesian was 2.87%. On other side, in case of goat, Black Bengal Goat was 1.19% Jamnapari breed was 0.6%. According to age the effect of ruminal acidosis was in cattle (<2years 2.87%, >2years 5.17%) whereas in goat (<1.5years 0.6% and >1.5years 1.19%).

Table 3: Prevalence of ruminal acidosis in different species, breeds and age groups

Categories	Variables	Parameters	Positive cases (%)	Negative cases (%)	p-value
Species (N = 510)	Cattle (n = 174)		14 (8.05)	160 (91.96)	0.09
	Goat (n = 336)		6 (1.79)	330 (98.21)	
Breed	Cattle (n = 174)	ND	9 (5.17)	125 (71.84)	0.31
		HF cross	5 (2.87)	35 (20.11)	
	Goat (n = 336)	BBG	4 (1.19)	178 (52.98)	0.33
		Jamnapari	2 (0.6)	147 (43.75)	
Age	Cattle (n = 174)	< 2 years	5 (2.87)	68 (39.08)	0.37
		>2 years	9 (5.17)	92 (52.87)	
	Goat (n = 336)	< 1½ years	2 (0.6)	206 (61.31)	0.39
		>1½ years	4 (1.19)	124 (36.90)	

Breed and age wise prevalence:

The breed and age specific prevalence of acidosis is shown in Figure 2 and Figure 3. The prevalence of acidosis was higher in Non-descriptive local (5.17%) than the HF cross

breed (2.87%) in case of cattle. Prevalence of acidosis is higher in Black Bengal goat than Jamnapari in case of goat.

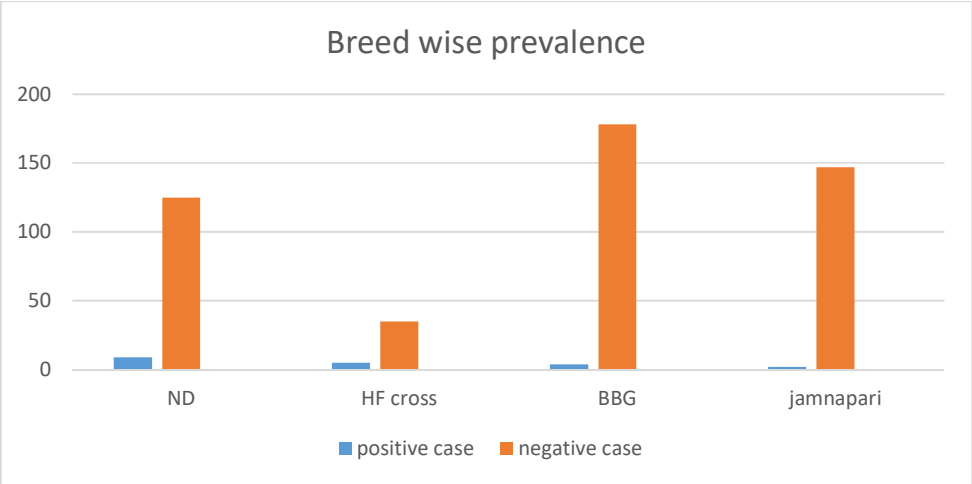


Figure 2: Prevalence of acidosis based on breed

The prevalence of acidosis was higher in more than 2 years aged animal (5.17%) than the younger aged cattle (2.87%) in case of cattle. Same as cattle, older aged goat are predominantly attacked with acidosis than the younger goat.

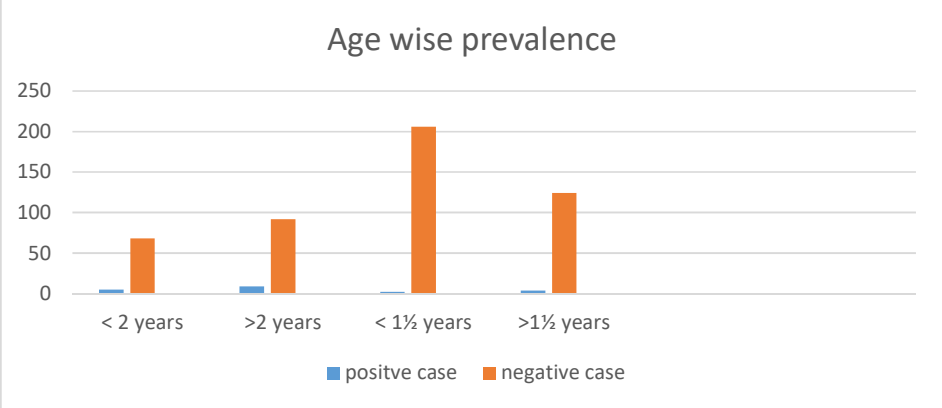


Figure 3: Age wise prevalence

Table 4: Response of different groups of animals in different treatments

Group	Cases	Treatment response (days)	p- value	Level of significance
A	6	7.00 ± 0.41	1.00	NS

B	6	7.00 ±1.00		
A	6	7.00 ±0.41	0.001	**
C	4	4.50 ±0.50		
A	6	7.00 ±0.41	0.00	**
D	4	1.81 ±0.09		

Drug response & total recovery day

In response of treatment of different groups of animal there was no significant difference in treatment between group A and B ($p > 0.05$). This study also exposed that in comparison between the other groups (except A and B) has significant differences ($p < 0.05$) in response to treatment. Any comparison with group D the difference in response to treatment was highly significant ($p = 0.00$). So treatment with both ruminal alkalizer and systemic alkalizer is the most effective treatment and animal response more quickly than other type of treatment (Figure 4).

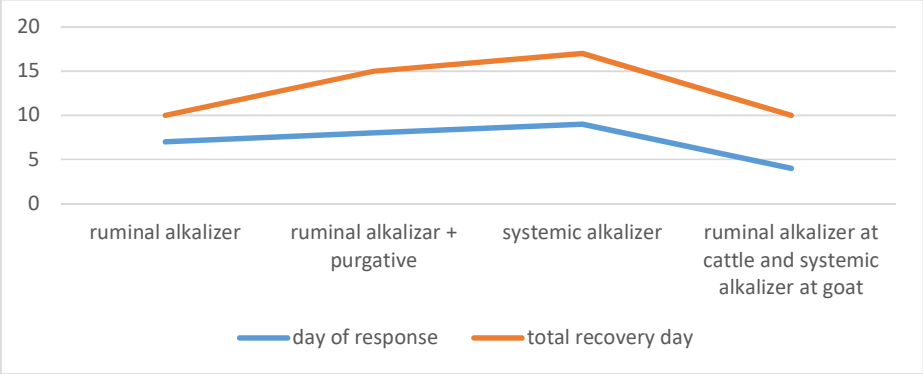


Figure 4: Treatment wise recovery

DISCUSSION

The overall prevalence of ruminal acidosis found 3.92% in the current study which is not similar to the findings of Kleen et al., 2013 who found a herd prevalence of 20% in cases sub-acute acidosis in cattle in Germany.

The objective of this case report was to describe how the structure and function of the rumen adapts during the initial stage of ruminal acidosis just after correction of rumen P^H. It was revealed in the present study that sudden intake of large amount of easily digestible non-fiber carbohydrates e.g. feeding of cooked rice, rice gruel predisposes the ruminal acidosis in most of the cases. In the case a remarkable changes the physical characteristics of ruminal fluid observed during the period of rumen acidosis, such as becoming milky color, watery consistency and souring odor with Jasmin et al., 2011 who reported changes were similar to the observation in sheep where rumen P^H was 4.2 to 6. These findings were in agreement with those reported by some authors that relate changes with decreasing P^H in the rumen caused by excessive rice in the concentration of VFA and lactic acid, which increases the osmolarity of the medium, making it hypertonic in relation to plasma, causing a greater flow of water from the intracellular and extracellular compartments into the digestive tract, especially the rumen according to Kolver and De Veth, 2002. Carbohydrate engorgements increased amount of lactic acid with VFA that decreased rumen flora (Dunlop, 1972) which is agreed in present study with Dunlop, 1972 where found abdominal distension in the case as a clinical sign, it is due to high osmotic pressure inhibit bacterial digestion of fiber and starch causing ruminal content to become stagnant and also due to pulls up water from systemic circulation by high osmotic pressure of rumen reported as abdominal distension is a clinical sign of acute ruminal acidosis. Diarrhea found as a clinical sign of ruminal acidosis. Changes in microbial fauna of the rumen fluid of animal studied with respect to decreased motility or absence of motility. Protozoa lose their activity when the P^H drops to values between 5.0, disintegrating or suffering rumen mucosa layer lysis occurs when an increase in acidity of the medium and P^H reaches values below 5.0. In present study, it was revealed that use of ruminal and systemic alkalizer is more effective treatment in ruminal acidosis. These findings is the agreement with Hart and polan, 1984 they use ruminal alkalizer (Sodium bicarbonate) and intravenous hypertonic sodium bicarbonate (5%) in severe

cases in an induced acidosis and observed all the animal recovered. Stone, 2004 suggested to use ruminal antacids orally to neutralize the ruminal acids and intravenous hypertonic sodium bicarbonate to neutralize systemic acidosis and correction of dehydration. The recovery of the animals is due to full utilization of the acids and the gradual modification of the microbial population of the rumen fluid.

A number of research found higher prevalence of sub-acute ruminal acidosis (Nogues, 2013; Kleen et al., 2013; Golder et al., 2012; Jasmin et al., 2011; Radostits et al., 2000) which is similar with this study where there were no significant variation was found among species, age and breed. These findings are also agreement with the report of all types of ruminant are susceptible to ruminal acidosis which was revealed as high amount of carbohydrate ingestion is the main cause of acidosis (Kleen et al., 2013; Kolver and De Veth, 2002).

CONCLUSION

Ruminal acidosis is an important nutritional problem in ruminants in terms of economic point of view and as a substantial health problem. The cause of ruminal acidosis is not a pathogen, but self-created complication by owner or farmer and the major predisposing factors are in feeding practices. This study indicates feeding of large amount of grain, cooked rice, rice gruel that predisposes the ruminal acidosis. In present study, the rumen fluid color, consistency, odor, absent of ruminal flora movement, lower rumen fluid P^H gives clue for diagnosis. This study shows the use of ruminal and systemic alkalizer along with fluid in treatment of ruminal acidosis is more effective and has a quicker resolution. This study also indicates correct feeding practice as well as gradually change of feeding habit can reduce ruminal acidosis.

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REFERENCES

1. Cricklow EC and Chaplin, 1985. Ruminal lactic acidosis: Relationship of fore stomach motility to volatile fatty acid. Am. J. Vet. Res. 46 (9): 1908-1911.
2. Russel and Hino, 1985. Regulative of lactate production in *Streptococcus bovis*. J Dairy Sci, 68:1712- 1721.
3. Ahrens FA, 1967. Lactic acid as factors in developing ruminitis. Am. J. Vet. Res. 28 (126): 1335-1342.
4. Mullenax CH, Keelen, 1966. Physiological response to toxic factor excreted from rumen bacteria. Am. J. Vet. Res. 27: 857-868.
5. Kolver, ES and De Veth MJ, 2002. Prediction of ruminal P^H from pasture-based diets. J Dairy Sci, 85: 1255-1266.
6. Dunlop RH, 1972. Pathogenesis of ruminant lactic acidosis. Adv Vet Sci Com Med, 16: 259-302.
7. Hart SP and Polan CE, 1984. Effect of sodium bicarbonate and disodium phosphate on animal performance, ruminal metabolism, digestion and rate of passage in ruminating calves. J Dairy Sci, 67:2356-2368.
8. Stone WC, 2004. Nutritional approaches to minimize sub-acute ruminal acidosis and laminitis in dairy cattle. J Dairy Sci, 87: 13-26.
9. Kleen JL, Upgang L and Rehage J, 2013. Prevalence and consequences of sub-acute ruminal acidosis in Germany dairy herds. Acta veterinaria scandinavica, 55:48.
10. Nogues A, 2013. Sub-acute ruminal acidosis (SARA).
11. Jasmin BHRC, Modesto RB and Schaer TP, 2011. Perioperative ruminal P^H changes in domestic sheep. Lab. anim. sci. 50 (1): 27-32.
12. Golder HM, Celi P, Rabiee AR, et al, 2012. Effects of grain, fructose and histidine on ruminal P^H and fermentation products during an induced sub-acute acidosis protocol. J Dairy Sci. 95:1971-1982.
13. Ahrens FA, 1967. Histamine, lactic acid and hypertonicity as factors in the development of rumenitis in cattle. Am J Vet Res., 28:1335-1342.
14. Aschenbach JR, Gabel G, 2000. Effect and absorption of histamine in sheep rumen: Significance of acidotic epithelial damage. J Anim Sci. 78:464-470.

15. Gabai G, Testoni S, Piccinini R, Marinelli L, Stradaioli G, 2004. Oxidative stress in primiparous cows in relation to dietary starch and the progress of lactation. Anim Sci., 79:99-108.
16. Celi P, 2011. Oxidative stress in ruminants. In: Mandelker L, Vajdovich P, editors. Oxidative Stress in Applied Basic Research and Clinical Practice. New York, New York: Humana Press; pp. 191-231.
17. Penner G, Oba M, Gäbel G, Aschenbach J, 2010. A single mild episode of sub-acute ruminal acidosis does not affect ruminal barrier function in short term. J Dairy Sci 93: 4838-4845.
18. Radostits OM, Gay CC, Blood DC and Hinchcliff KW, 2000. In: Veterinary Medicine, A textbook of disease of cattle, sheep, pigs, goats and horses, 9th edition. W B Saunders, pp: 1387-1416.
19. Thrusfield MV, 2005. Criteria for Success of Questionnaire. In: Veterinary Epidemiology. 3rd edition. Oxford, UK: Blackwell Science. pp: 189-213.
20. Owens JA, 2009. A clinical overview of sleep and attention-deficit/hyperactivity disorder in children and adolescents. J Can Acad Child Adolesc Psychiatry, 18 (2): 92-102.

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