Therapeutic Efficacy of Monensin on Lactic Acidosis in Buffaloes

NANAGOUDA A PATIL[1*], AJAY S SATBIGE[2], SANDEEP C HALMANDAGE[3], RAVINDRA G BHOYAR[4]

[1]Directorate of Extension, Karnataka Veterinary, Animal and Fisheries University, Nandinagar PB No. 6, Bidar- 585401, Karnataka, INDIA
[2]Department of Veterinary Medicine, Veterinary College, Nandinagar PB No. 6, Bidar-585401, Karnataka, INDIA
[3]Department of Veterinary Medicine, Veterinary College, Nandinagar PB No. 6, Bidar-585401, Karnataka, INDIA
[4]Department of Teaching Veterinary Clinical Complex, Veterinary College, Nandinagar PB No. 6, Bidar-585401, Karnataka, INDIA

Corresponding Author's E-mail: napvety@gmail.com

Abstract: - Three non-descript buffaloes were presented to Veterinary College Hospital, Bidar with the history of ingestion of large quantity of wheat based bakery products. Clinical examination revealed mild distension of left abdomen, passing semisolid pasty dung. Rumen pH, Rumen lactate and total protozoal count, Blood pH and HCO_3^- were analysed before and after treatment. Acidotic buffaloes were treated orally with monensin at the dose rate 3 mg/kg body weight along with fluid therapy and repeated after 24 hours. Healthy ruminal cud was given orally after 48 hours. Monensin was found effective in the treatment of lactic acidosis in buffaloes.

Key-words: – Lactic acidosis, Buffaloes, Monensin, Rumen lactate, Blood HCO_3^-, Rumen protozoa

1. Introduction

Lactic acidosis is a common alimentary tract disease occurring in ruminants causing severe economic loss to the farmers due to significant reduction in milk yield and high mortality rate. Most of the clinical cases of lactic acidosis in ruminants are observed due to accidental ingestion of carbohydrate rich food stuffs which promotes growth and multiplication of lactic acid producing bacteria which dominate the ecosystem of the rumen and eventually production of lactic acid lowers down the ruminal pH leading to lactic acidosis [1].

Enormous work has been done on physio-pathological aspects and therapeutic management of lactic acidosis in ruminants. In therapeutic aspects of lactic acidosis, most of the existing treatments have not withstood critical evaluation. Commonly indicated oral antacids in lactic acidosis may be beneficial if administered within four to six hours after overeating and before lactic acid production has commenced. In acute cases of lactic acidosis, antacids administered directly into the rumen may be contraindicated because of risk of alkalosis which may occur during the recovery phase. A summation of alkaline material absorbed from the recovering rumen together with the alkaline products of lactate metabolism, may prove a metabolic embarrassment [2]. Evidence from other species certainly indicates a substantial risk from overuse of biocarbonate in the correction of metabolic acidosis particularly lactic acidosis [3]. Clinical aids to make acid base assessment in the field are not readily available. Excessive bicarbonate therapy without establishing the severity of metabolic acidosis in rumen acidosis creates the risk of an "overshoot metabolic alkalosis" [4].

It has been well documented that growth of lactate producing bacteria *streptococcus bovis* and *lactobacillus spp* is inhibited by ionophore antibiotic monensin [5][6] [7]. Monensin exerts favorable effects of rumen fermentation by enhancing propionate formation and inhibiting methane production [8]. The primary ways in which monensin modify rumen function is by decreasing ruminal population of gram positive bacteria relative to that of gram negative bacteria [9]. Literature on use of monensin in clinical cases of lactic acidosis in buffaloes is scanty. Hence the therapeutic efficacy of monensin was evaluated in clinical cases of lactic acidosis in buffaloes.

2. Material and Methods

Three buffaloes suffering from lactic acidosis due to ingestion of large quantity of wheat based bakery products were utilized for the present study. General examinations of affected buffaloes were carried out. The ruminal fluid was collected using rumen fluid collection apparatus. The pH of ruminal fluid was estimated immediately with pH meter. Lactic acid concentration of rumen fluid was estimated as per the method of (Barker and Summerson, 1941) [10]. Blood samples were drawn into heparinized syringes for analysis of pH and $HCO3^{-}$ by blood gas analyzer.

Acidotic buffaloes were treated orally with monensin at a dose of 3mg/kg body weight along with fluid therapy and repeated after 24 hours. Fresh ruminal fluid collected from healthy buffaloes was drenched orally after 48 hours. Therapeutic efficacy of monensin was evaluated based on clinical examination, rumen profile and blood profile changes before treatment and 24 hr, 48 hr. and 72 hr. after treatment. The data were subjected to statistical analysis. [11]

3. Results

All three affected buffaloes were anorectic, dull, depressed and passing semisolid pasty dung. There was mild abdominal distension. Rumen pH observed before treatment was 5.5 ±0.15 (Table). Marked improvement in Rumen pH was observed after 72 hours of treatment (6.7± 0.06). Rumen lactate observed before treatment was 130.60 + 7.83 mg/d1 (Table). Significant reduction in rumen lactate concentration was observed at 24 hour, 48 hour and 72 hours after treatment.

Total protozoal count observed before treatment was $0.55 \pm 0.09 \times 10^5$/ml (Table). There was no significant improvement in total protozoal count even after 48 hours of treatment. Slight improvement in total protozoal count was observed after 72 hours of treatment. The blood pH observed before treatment was 7.20 ± 0.01 (Table). Significant improvement in blood pH was observed after 48 hour and 72 hours of treatment. The blood HCO_3^- observed before treatment was 18.53±0.39 mμ/L (Table). Significant increase in blood HCO_3^- was observed after 72 hours of treatment.

4. Discussion

Decrease in rumen pH and increase in rumen lactate in lactic acidosis were also reported [7] [12]. This may be attributed to faster and complete fermentation of starch by amylolytic bacteria in the rumino-recticulum compartment of engorged animals which led to the production of large amount of lactic acid in the rumen [1]. Significant improvement in rumen pH and reduction in rumen lactate was observed after 48 hours of treatment with monensin. Similar observations were made by earlier workers [6] [7]. Reduction in rumen lactate observed in the present study may be attributed to selective inhibition of growth of lactate producing bacteria by monensin [5] [6] [7].

Decrease in total protozoal count observed in the present study concurred with earlier reports [7] which could be attributed to high concentration of rumen lactate and low rumen pH [13]. There was no significant improvement in total protozoal count even after 48 hours of monensin treatment. This could be attributed to adverse effect of monension on protozoa population [5] [7]. Slight improvement in total protozoal count observed after 72 hours in the present study may be attributed to inoculation of ruminal cud from healthy buffaloes after 48 hours.

Reduction in blood PH and blood HCO_3^- in the present study concurred earlier reports [7][14]. The decrease in blood pH and blood HCO_3^- could be attributed to uncompensated metabolic acidosis due to increased absorption of lactic acid from the rumen. Significant improvement in blood pH and blood HCO_3^- was observed after 48 hour and 72 hours of treatment with monensin.

5. Conclusion/Recommendation

Clinically all the three affected buffaloes restored their normal appetite, ruminal movements and normal faeces. It would be, thus concluded from the results of the present study that oral administration of monensin along with inoculation of fresh ruminal cud was effective in clinical cases of lactic acidosis in buffaloes.

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Table -1

Effect of monensin on rumen and blood profile in acidotic buffaloes (Mean ± SE)

Parameters	Time	Rumen pH	Total protozoal count (n x10^5/ml)	Rumen lactate (mg/dl)	Blood pH	Blood HCO_3^- (Mm/L)
Acidotic buffaloes	0 hour	5.5±0.15	0.55±0.09	130.60±7.83	7.20±0.01	18.53±0.39
	24 hour	5.9±0.08	0.64±0.08	62.66±4.91	7.23±0.01	19.41±0.53
	48 hour	6.3±0.38*	0.77±0.08	30.53±2.02	7.34±0.02	21.32±0.54*
	72 hour	6.7±0.06*	1.23±0.06*	18.15±1.76*	7.35±0.04*	23.39±0.47*
Healthy Control		6.91±0.02	3.21±0.06	18.24±1.68	7.39±0.02	24.82±0.40

*Significant (P≤0.05)