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Ruminal acidosis associated with ruminal bloaty diarrhoea in a non-lactating cow in Bangladesh: a latest case study

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Abstract
This A mature dairy non-lactating cow that was suffered from acidosis after ingestion of large volume of cooked rice, rice gruel from a dustbin near a residential area at Nagorkandha, Faridpur. Primary diagnosis of this case was performed from owner's complain, clinical signs and physical examination of color, odor and consistency of rumen fluid and rumen microbe's movements. Confirmation diagnosis was performed by measuring the rumen fluid and blood pH. The predisposing factors of ruminal acidosis were from feeding of large amount of grain, green grasses. The body temperature, rumen microbe's motility, fluid pH and blood serum pH of the affected cattle were 101.5, 5.6, 5.5 and 7.2 °F respectively. The most effective treatment of acidosis in cattle was observed from the use of ruminal alkalizer (magnesium hydroxide, 5% sodium bicarbonate), some antibiotics such as potentiated Sulphonamides and Tetracycline along with fluid therapy. This study suggests the farmer, to avoid providing digestible carbohydrates to ruminant at large amount simultaneously and be cautious to formulate ration having desirable level of carbohydrate that will prevent ruminal acidosis.

Keywords: Acidosis, rumen pH, tentative diagnosis, alkalizer, cattle.

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1. Introduction

Livestock is an important sector which plays contribution to promote human health by supplying protein sources and help to achieve the sustainable development goals (SDGs) by solving poverty alleviation. But ruminant suffers from many infectious and non-infectious diseases. Ruminal acidosis is one of the most important non-infectious diseases throughout world. Many ruminants suffer from ruminal acidosis due to improper practice of feeding resulting from the lack of knowledge about risk factors. Ruminal acidosis causes when a ruminant animal consume large amount of rapidly fermentable carbohydrates, primarily starches and sugars (Beauchemin and Penner, 2009). A large number of farmers involved in cattle fattening program just

before 3 or 4 months of Eid-ul-Azha. It's such kind of clinical condition that provokes accumulation of huge amount of H^+ ions from lactic acid, characterized by blood lactate level > 5.0 mm/L and arterial pH < 7.25 (Robert et al., 1982). Ruminitis, metabolic acidosis, lameness, hepatic abnormalities, pneumonia and finally death may occur in such kind of acidosis (Lean et al., 2000). Additionally, the clinical conditions like lameness and laminitis impact significantly to make cow living comfortably and play a vital role in social wellbeing (Hall and Averhof, 2000; Oetzel, 2003). In most of the case of ruminal acidosis in sub-acute form goes unrecognized and undiagnosed till significant herd involvement. For this result, large economic losses associated with high prevalence of herd lameness, may be inescapable (Nocek, 1997).

2. Place of study

The study was conducted at Faridpur in Bangladesh of a road free grazing cow. In this study a eight year non-lactating dairy cow (Holstein Friesian) accidentally intake large amount of cooked rice and rice gruel from dustbin near a residential area that showing ruminal acidosis, which may severely compromise gastrointestinal disorder. The owner reported that the cow was off feed, and suffering from diarrhea.

3. Case presentation (Results)

An eight-year-old, 450 kg body weight named Shamoli non-lactating (Holstein Friesian) dairy cow, accidentally intake large amount of cooked rice and rice gruel that consequently suffered from ruminal acidosis. The owner reported that the cow showed off feed and distention of abdomen that indicates moderate bloat with frequently diarrhea. I was called by the owner for correction the clinical case. The cow was brought to near Upazilla Veterinary Hospital (UVH), Nagorkandha, Faridpur with me for correction the clinical condition. Presumptive diagnosis of the cases was performed on the basis of feeding history that was consuming of large amount of cooked rice and rice-gruel and grains. Additionally clinical history, the owner also reported that a diet consisting exclusively of hay and with supplemental vitamins and minerals was fed in one week ago where associated with clinical signs. Presumptive diagnosis was performed on the basis of feeding history that was easily digestible non-fiber carbohydrates (Cooked rice, rice gruel) associated with clinical signs of fluidly paste diarrhea with free gas. Additionally tentative diagnosis was performed by examination of rumen fluid color, consistency and odor of rumen fluid. Diarrhea was diagnosed by observing the frequent defecation of paste faces with fluid and mucus. Finally Confirmatory diagnosis was performed by exploring the low pH of rumen fluid by the help of pH indicator paper (Merck-universal indicator pH 1-10). Then I decided to collect rumen fluid by using stomach tube as aspiration method of rumen fluid collection. At first the stomach tube was cleaned and washed by antiseptic solution (Hexaclorphane ethaile alcohol 70%). Finally the stomach tube was dried and used lubricants for collection of rumen fluid from the rumen. To collect the ruminal fluid by using the stomach tube which length is 3½ m, width is 19 mm that was joined to a paddle pump. The opposite end should be fixed to a 10 cm to 15 cm length of copper pipe having multiple clefts drilled along all sides (Fig.1:A). The copper pipe minimizes the risk of blockages with ingesta at digestive tracts. The stomach tube was penetrated slowly and cautiously at through the esophagus. When the cow became coughing then makes an interval and tried again. Rumen fluid was collection by the help of suction pump which was attached on another side of stomach tube. Generally this sample pH was higher than the others method of collected fluid. There is no chance of salivary contamination in this method of rumen fluid collection. Physical characteristics (Color, consistency and odor) of rumen fluid were determined by using organoleptic test. In this cases the rumen fluid color was milky grey (Fig.1: C).The rumen fluid was found thick watery in consistency and the sour odor was found in rumen fluid. Then the rumen fluid is taken about 1 ml into a slide and a fragment of pH indicator paper (Merck-universal indicator pH 1-10, Merck

Limited, Worli, Mumbai-400 018) inserted into the slide containing fluid for 30 seconds. The color of pH indicator paper was changed rapidly. This color matched with the one of the different color of the color scale.

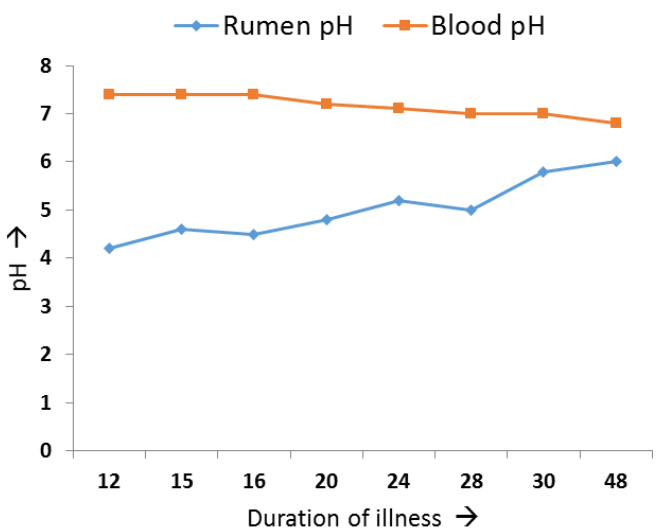

Fig. 1: The rumen and blood pH was changed correlated with time of illness

The value of matched color was indicating the pH of the rumen fluid. The result of rumen fluid pH was 5.5 and serum pH was 6.1, the temperature was 101.5 °F. To identify the motility of the rumen microflora, two drops of rumen fluid were taken into two clean glass slides, gently make slide smear and then observed under microscope at low power objective (10X). The ruminal microflora movement was dramatically reduced in one slide and another slide was absent but some protozoas were found due to acidic environment in the rumen. Diarrhea was diagnosed by the foul odor of faces with fluid and mucus. After diagnosis of ruminal acidosis and consequently of diarrhea case then decision was taken for treatment. Firstly the cow was restrained by keeping into a trabeach. Treatment comprised antacids such as Mg(OH)2, NaHCO3 which dose was 1 gm/kg body weight after diluted with water and administered orally. It was helped to neutralize the rumen pH. Oral electrolyte solutions were also given to minimize the dehydration, preferably those containing additional NaHCO3 to solve metabolic acidosis (Solorzano, 1989). Then use systemic ruminal alkalizer: Sodium bicarbonate (Inj. Sodib®, M. R. Chemicals, India) which was 5% sodium bicarbonate and administered at the rate of 750 ml for 450 kg body weight initially over a period of 30 minutes followed by isotonic sodium bicarbonate (1.3%) at 15 ml/kg body weight, I/V over next 6-12 hours. Intravenous fluid e.g. hypertonic saline (0.9% sodium chloride solution) was given at the rate of 50-100 ml/kg body weight (depending upon severity of dehydration), I/V, once daily for 3 days and combined antibiotics including sulphonamides (333mg/kg) and tetracycline (20mg/kg) was given to mitigate the risk of liver abscessation (Divers and Peek (2008).

4. Conclusion

The cause of ruminal acidosis is not a pathogen, but self-created complication by owner and the major predisposing factors are in feeding practices. This study indicates feeding of large amount of cooked rice, rice gruel and grains, which predispose the ruminal acidosis. In present study, the rumen fluid color, consistency, odor, absent of ruminal flora movement, lower rumen fluid pH gives clues 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 demonstrates that correct feeding practice can reduce the ruminal acidosis. Free Feeding habit from dustbin and rotten carbohydrates should be avoided.

5. Discussion

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 pH. 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 predispose 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. These findings were in agreement with those reported by some authors that relate changes with decreasing pH in the rumen caused by excessive rise 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 (Kolver, E. S. and De Veth, M.J. 2002). In present study, there was 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 that 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 pH drops to values 5.0 to 5.5, disintegrating or suffering rumen mucosa layer lysis occurs when an increase in acidity of the medium and pH 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 have similarity with Khafipour et al. (2009), they use ruminal alkalizer (Sodium bicarbonate) and intravenous hypertonic sodium bicarbonate (5%) in severe cases in an induced acidosis and observed the entire animal recovered. (Redostits et al, 2006) suggested using 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.

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The following symptomatic change was observed such as anorexia, decreased rumen motility, dehydration and diarrhea. Such demonstrations coincided with decreasing pH of the rumen fluid, especially when values were below 5. In this study, there was found moderate dehydration due to fluid loss by the paste diarrhea. This dehydration is also due to high osmotic pressure of ruminal contents in aciditic condition that pulls up water from systemic circulation. There were found anorexia and inappetance because of an elevation of osmotic pressure in the rumen wall that inhibit feed intake.

In my study, there was 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 that abdominal distension is a clinical sign of acute ruminal acidosis. Frequently defecation of paste faces with irregular gas was observed as a clinical sign of ruminal bloaty diarrhoea which was foul odor. These findings have similarity with Khafipour *et al*. (2009), they use ruminal alkalizer (Sodium bicarbonate) and intravenous hypertonic sodium bicarbonate (5%) in severe cases in an induced acidosis and observed the entire animal recovered. (Redostits *et al*., 2006) suggested using 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 animal is due to full utilization of the acids and the gradual modification of the microbial population of the rumen fluid, where there was a reduction or disappearance of agents considered harmful, acid producer lactic flora and the restoration of Gram-negative bacteria considered as the main fermentative lactate.

Conflicts of interest

The authors declare no conflicts of interest.

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