

Ruminal lactic acidosis in cow: clinical assessment

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ABSTRACT

Ruminal lactic acidosis is a condition that arises in ruminants due to the ingestion of excess quantities of easily digestible grains leading to diarrhea, anorexia and physiological derangements which can be fatal in severe cases. A clinical case of cow with history of rice flour ingestion showing signs of acute ruminal lactic acidosis was managed. The pH of ruminal fluid was 5.1. The number of live protozoa in the ruminal fluid was almost nil. Blood analysis confirmed marked alterations due to dehydration. Electrolyte imbalance was evident in biochemical tests. Cow was treated with intraruminal administration of Magnesium hydroxide and Sodium bicarbonate. Full recovery was achieved without any complications.

Key Words: Cow; Rumen; Lactic acidosis; Rumen pH; Rumen Microbes

Introduction

Acute ruminal lactic acidosis (commonly known as grain engorgement or acid indigestion) is a disorder of microbial fermentation process in rumen. This condition arises due to the sudden and excess ingestion of carbohydrate-rich feed such as grains or their flour (Owens, 1998; Rodostitis *et al.*, 2000). The amount and type of carbohydrate rich feed determine the severity and clinical outcome (Gentile *et al.*, 2004). Group feeding aggravates the condition because of group competition which induces the animals to ingest more feed (Rodostitis *et al.*, 2007). The etio-pathology begins with abrupt ingestion of easily fermentable carbohydrates which alter the ruminal microbial population leading to excessive ruminal fermentation to acids. The disease is clinically characterized by anorexia, depression, ataxia, abdominal distension, diarrhea and weakness. In some severe cases, it can be fatal in less than 24 hrs. Clinical definitions of ruminal acidosis are largely based on rumen pH cut points (Nagarajam, *et al.*, 2007; Plaizier, *et al.*, 2008). In carbohydrate engorgement, there is abnormal distension of rumen, lactic acidosis and atony of rumen leading to the retention of fermented gases and even death of the animal, if left untreated. We report feed ingested, clinical presentation, and rumen fluid and blood-biochemical profiles of a cow exposed to feed high in readily fermentable carbohydrates.

Case History and Diagnosis

A cross-bred Jersey cow of 3 years age was presented by the owner with the complaint that his cow became off-feed, diarrheic and reluctant to move after consuming rice flour accidentally a few hours back. In Kashmir villages, people use rice flour for making traditional bread. Clinical observations were dullness, depression, diarrhea, complete rumen atony, ataxia, anorexia, rapid respiration and pulse, and signs of dehydration.

History of rice flour consumption was highly suggestive of acute ruminal lactic acidosis. The clinical signs of diarrhea, inappetence, and reluctance to move observed within hours of consumption of readily fermentable carbohydrate are consistent with the diagnosis of acute ruminal acidosis (Plaizier *et al.*, 2008; Afshin *et al.*, 2011).

Blood sample was collected from jugular vein and transferred to two vials, one containing anti-coagulant (EDTA) for flood profile (RBC, PCV, WBC, TLC and DLC) and another without anti-coagulant for biochemical picture (glucose, BUN, calcium, sodium, potassium, chloride) (Pagana and Pagana, 2010). These tests were performed by automatid blood analyser. Physiological alterations are presented in Table 1.

Table 1: Alterations of Physiological Values in Cow with Lactic Acidosis

Parameters	Value
Rumen Protozoal Activity	(---)
Rumen fluid pH	5.10
Glucose (mg/dL)	83.12
Na ⁺ (mmol/L)	126.54
K ⁺ (mmol/L)	6.50
Cl ⁻ (mmol/L)	91.16
Ca (mg/dL)	8.71
RBC (10 ⁹ /dL)	8.30
Hb (g/dL)	13.80
PCV (%)	44.00
Leucocytes (10 ⁶ /dL)	11.00

Ruminal fluid was collected using a stomach tube connected to a plastic syringe for suction purpose. The sample was sieved and immediately examined for ruminal pH by digital pH meter and protozoal activity which was visualised microscopically (Abd El- Roaf, *et al.*, 2007). Rumen fluid pH was 5.1. This low pH value clearly indicates acidic conditions in the rumen. The pH value less than 5.5 is non-physiological and detrimental for normal rumen microbes (Leek, 1983). Microscopic examination of rumen fluid showed the presence of only a few numbers of live protozoa. Previous studies by Martin *et al.*, 2006 report a sharp decline in protozoan count in cows with ruminal acidosis. Death of microflora occurs due to acidic conditions as microflora best thrive in neutral media of 6.4-7.0 (Steen, 2001).

Treatment

For treatment, neutralization of acid present in rumen marks the first and foremost logical step. Magnesium hydroxide, 400 gm dissolved in 10 L water was pumped into rumen by stomach tube to neutralize the acidity. Magnesium hydroxide is a potent alkalinizing agent to be used in ruminants as an antacid (Smith and Correa, 2004). This was followed by fluid therapy in the form of NaCl solution 1000 ml as IV drip to counter dehydration and electrolyte imbalance. An injection of thiamine was also given. Sodium bicarbonate, 400 gm was advised to be orally drenched twice daily for two days (Suzuki *et al.*, 1999). After two days of treatment, the cow began normal feed intake and was clinically sound.

Ruminal acidosis leads to dehydration with the resulting hemoconcentration which alters blood constituents. Thiamine is normally synthesized by the rumen microbes (bacteria, protozoa etc). In lactic acidosis, the acidic rumen pH results in decrease in the population of rumen microbes; this leads to decline in biosynthesis of thiamine. The oral administration of thiamine not only restores the function of the cells and tissues by replenishing thiamine deficiency but also promotes metabolism of excess lactic acid thereby reducing acidosis (Wilson, *et al.*, 1991). Lactic acidosis was associated with hematological changes such as significant elevation of erythrocytes, leucocytes, PCV (Garry, 2002). Hyperglycemia is attributed to increased lactic acid level of blood and its utilization in gluconeogenesis (Garry, 2002). Biochemical changes such as hyperglycemia, decreased total protein, hypocalcemia, hyponatremia, hyperkalemia, increased urea nitrogen and creatinine levels have been recorded earlier (Brown *et al.*, 2000; Jorg and Enemark, 2008). Electrolyte changes are both due to diarrhea and their loss into rumen via damaged ruminal wall.

Dairy cow owners are inclined to feed excess of concentrated feeds in order to increase milk production and meet the animal's energy requirements; but this is judged as a sword-edge between maximal productivity and development of rumen disorders. Consumption of rice flour develops lactic acidosis quickly. Mechanisms of rumen pH regulation are based on feed type, percentage of moisture and its physical structure also (Hutjens *et al.*, 1996). This clinical case draws attention to the potential dangers of crushed/powdered grains in the diet that they can readily lead to the onset of ruminal acidosis.

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