

International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2026): 5.29
IJABR 2026; 10(5): 305-309
www.biochemjournal.com
Received: 07-03-2026
Accepted: 10-04-2026

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Impact of acute Ruminal acidosis on erythrocyte osmotic fragility and lipid peroxidation in Kodi adu

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DOI: <https://www.doi.org/10.33545/26174693.2026.v10.i5d.8403>

Abstract

This study evaluated the effects of acute ruminal acidosis on erythrocyte osmotic fragility and lipid peroxidation in Kodi adu. Twelve Kodi adu goats diagnosed with acute ruminal acidosis under field conditions and undergoing veterinary treatment were included in the study. Samples were collected at multiple intervals over 72 hours to assess rumen pH, blood pH, hematological parameters, erythrocyte osmotic fragility, and malondialdehyde (MDA) levels. A significant decline in rumen pH (<5.0) and blood pH (<7.2) was observed within 12-24 hours, confirming acute metabolic acidosis in the affected goats. These animals showed marked hemoconcentration, with packed cell volume increasing from $\sim 29\%$ to $>42\%$ and hemoglobin levels rising significantly ($p < 0.01$). Erythrocyte osmotic fragility was significantly elevated, with Mean Corpuscular Fragility (MCF₅₀) shifting from 0.49% to 0.62% NaCl. Concurrently, MDA levels increased significantly, indicating enhanced lipid peroxidation and oxidative stress. Statistical analysis revealed a significant negative association between rumen pH and erythrocyte stability and a significant positive association between MDA levels and osmotic fragility ($p < 0.01$). Although partial recovery was observed by 72 hours following treatment, elevated fragility and oxidative stress markers persisted.

Keywords: Ruminal acidosis, goats, erythrocyte osmotic fragility, lipid peroxidation, malondialdehyde, oxidative stress

Introduction

Ruminal acidosis is a multifactorial metabolic disorder that remains a major challenge in intensive ruminant production systems, particularly when animals are exposed to concentrate-rich feeding practices aimed at improving growth and productivity. In goats, ruminal acidosis commonly occurs following the ingestion of excessive amounts of rapidly fermentable carbohydrates, especially during abrupt transitions from roughage-based diets to high-concentrate rations (Owens *et al.*, 1998; Chavelikar *et al.*, 2018) ^[17, 5]. Such dietary changes disrupt normal ruminal fermentation, resulting in excessive accumulation of volatile fatty acids (VFAs) and lactic acid, which subsequently lowers ruminal pH (Radostits *et al.*, 2007) ^[19].

Under normal physiological conditions, ruminal pH is maintained between 6.2 and 6.8 through salivary buffering and microbial homeostasis. Fibrous feeds are fermented efficiently into VFAs that serve as major energy sources for the host animal. However, rapid fermentation of starch-rich diets leads to increased acid production and a decline in ruminal pH below 5.5. This acidic environment suppresses cellulolytic bacteria while favoring the growth of lactic acid-producing microorganisms such as *Streptococcus bovis* and *Lactobacillus* spp., thereby exacerbating acid accumulation and leading to acute ruminal lactic acidosis (Owens *et al.*, 1998; Aslan *et al.*, 1995) ^[17, 2].

The effects of ruminal acidosis extend beyond the digestive system and result in significant systemic alterations. Increased ruminal osmolarity caused by the accumulation of soluble carbohydrates and organic acids draws fluid from the circulation into the rumen, leading to dehydration and reduced plasma volume. These changes are often reflected as hemoconcentration, characterized by increased packed cell volume (PCV), hemoglobin concentration (Hb), and total erythrocyte count (TEC) (Chavelikar *et al.*, 2018) ^[5]. Although these responses may initially be compensatory, they can also increase blood viscosity and

impose stress on erythrocytes. Ruminal acidosis is also associated with damage to the ruminal epithelium, allowing endotoxins such as lipopolysaccharides (LPS) to enter the bloodstream. These endotoxins activate inflammatory pathways and stimulate the production of reactive oxygen species (ROS), contributing to systemic oxidative stress (Abuelo *et al.*, 2015) ^[1]. Erythrocytes are particularly vulnerable to oxidative damage because mature red blood cells lack nuclei and organelles required for repair and regeneration. Their structural integrity depends mainly on the stability of the plasma membrane, which consists of a lipid bilayer supported by membrane proteins and cytoskeletal components.

Osmotic fragility is widely used to assess erythrocyte membrane stability and reflects the susceptibility of red blood cells to hemolysis in hypotonic solutions. Increased osmotic fragility indicates compromised membrane integrity resulting from lipid peroxidation, protein denaturation, or impaired ion transport mechanisms (Jalali *et al.*, 2016) ^[10]. In ruminal acidosis, erythrocytes are exposed to both osmotic and oxidative stress, which may significantly impair membrane function and increase the risk of hemolysis.

Oxidative stress develops when ROS production exceeds the antioxidant defense capacity of the body. In acidotic animals, inflammatory responses enhance the generation of free radicals such as superoxide anions, hydrogen peroxide, and hydroxyl radicals. These reactive molecules initiate lipid peroxidation of membrane polyunsaturated fatty acids, producing secondary metabolites such as malondialdehyde (MDA), a widely recognized biomarker of oxidative damage (Ma *et al.*, 2018) ^[13]. The erythrocyte membrane is particularly susceptible to lipid peroxidation because of its high unsaturated lipid content and constant exposure to oxygen. Oxidative injury compromises membrane permeability, deformability, and the function of membrane-bound enzymes such as Na⁺/K⁺-ATPase, ultimately shortening erythrocyte lifespan.

Another important consequence of acidosis is its effect on hemoglobin function. Blood pH influences the oxygen-hemoglobin dissociation curve through the Bohr effect. During acidosis, reduced blood pH decreases hemoglobin affinity for oxygen, facilitating oxygen release to tissues but potentially impairing oxygen loading in the lungs (Guyton & Hall, 2016) ^[8]. Severe metabolic acidosis may therefore contribute to tissue hypoxia and further cellular damage.

Despite the importance of these mechanisms, limited information is available regarding the effects of ruminal acidosis on erythrocyte integrity in goats. Most previous studies have focused on cattle and sheep, whereas goats possess distinct physiological adaptations, including efficient water conservation and unique erythrocyte characteristics, which may influence their response to metabolic and oxidative stress. Furthermore, while acute ruminal acidosis has been widely investigated, comparatively fewer studies have explored its relationship with erythrocyte osmotic fragility and lipid peroxidation in caprine species.

Therefore, the present study was undertaken to evaluate the effects of acute grain-induced ruminal acidosis on erythrocyte osmotic fragility and malondialdehyde levels in Kodi adu goats.

Materials and Methods

Study animals and case selection

Client-owned Kodi adu goats presented with clinical signs consistent with acute ruminal acidosis to local veterinary practitioners were included in the study. Clinically healthy goats maintained under similar management conditions served as controls. Blood samples (approximately 5 ml) were collected aseptically from the jugular vein at the time of presentation and during follow-up. Samples were divided into heparinized tubes for erythrocyte osmotic fragility testing, EDTA tubes for hematological analysis, and clot activator tube for biochemical assays. Rumen fluid samples were collected using a stomach tube for pH determination. All samples were stored at 4 °C and processed within 2 h of collection to prevent *in vitro* hemolysis. Clinical history, treatment details, and progression of cases were recorded from the attending veterinarian.

Hematological evaluation

Hematological parameters including packed cell volume (PCV), hemoglobin (Hb), and total erythrocyte count (TEC) were determined using a hematology analyzer (Meril, Cellquant 360, India). Rumen pH was measured immediately after sample collection using a μ -Controller based digital pH meter (Systronics, India, Model 361). Similarly, blood pH was determined using the same μ -Controller based digital pH meter (Systronics, India, Model 361) following standard physiological procedures.

Assessment of erythrocyte osmotic fragility

Erythrocyte osmotic fragility was evaluated according to the method described by Claudia Tritschler *et al.*, (2016). Phosphate-buffered sodium chloride (NaCl) solutions containing 0.85%, 0.80%, 0.75%, 0.70%, 0.65%, 0.60%, 0.55%, 0.50%, 0.45%, 0.40%, 0.35%, 0.25%, and 0% NaCl were prepared. Two milliliters of each solution were mixed with 15 μ L of whole blood and incubated at 20 °C for 30-45 min. The samples were then centrifuged at 2000 $\times$ g for 10 min, and the absorbance of the supernatant was measured at 540 nm using a photoelectric colorimeter (Systronics, Haryana, India) at 540 nm. Distilled water (0% NaCl) served as the reference for complete hemolysis.

The percentage hemolysis at each concentration was calculated, and the mean corpuscular fragility (MCF₅₀), defined as the NaCl concentration producing 50% hemolysis, was determined from the sigmoid hemolysis curve (Parpart *et al.*, 1947) ^[18].

Estimation of lipid peroxidation

Lipid peroxidation was determined by measuring malondialdehyde (MDA) levels using the EZAssay™ TBARS (MDA) Estimation Kit (HiMedia Laboratories, Mumbai, India). Plasma or erythrocyte lysate samples were processed following the manufacturer's protocol, utilizing the reaction of MDA with thiobarbituric acid (TBA) to form a colorimetric product. The absorbance of the resulting supernatant was measured at 532 nm using a UV-Visible Spectrophotometer (UH5200/5210, Hitachi High-Tech). MDA concentration was calculated using the molar extinction coefficient and expressed in nmol/mL (Ohkawa *et al.*, 1979) ^[16].

Statistical analysis

Data were analyzed using SPSS software. Variables measured repeatedly over time were analyzed using two-way repeated measures ANOVA with treatment group as the between-subject factor and time as the within-subject factor. Paired t-tests were used to compare post-treatment values with baseline values within groups. Pearson's correlation analysis was used to assess relationships between rumen pH, erythrocyte osmotic fragility (MCF₅₀), and MDA levels. Statistical significance was considered at $p < 0.05$.

Results

Clinical findings and confirmation of ruminal acidosis

Goats presented with clinical signs such as anorexia, depression, abdominal distension, diarrhea, and varying degrees of dehydration. Rumen fluid examination revealed a marked reduction in pH in affected animals compared to healthy controls. Rumen pH in acidotic goats was significantly lower at presentation and during the early phase of observation, with values frequently falling below 5.5, confirming the presence of acute ruminal acidosis. In contrast, control animals maintained a stable rumen pH within the normal physiological range (6.4-6.7) throughout the observation period. Blood pH in acidotic goats also showed a significant decrease compared to controls, indicating the development of systemic metabolic acidosis. Gradual improvement in both rumen and blood pH was observed in affected animals during treatment, approaching near-normal levels by 72 hours.

Changes in rumen and blood pH

Table 1 presents the temporal changes in rumen and blood pH in control and acidotic goats. A significant decline ($p < 0.05$ to $p < 0.01$) in rumen pH was observed in acidotic animals during the initial 24 hours, with the lowest values recorded between 18 and 24 hours. Blood pH followed a

similar trend, reflecting systemic acid-base imbalance. Partial recovery was evident at 48 and 72 hours following therapeutic intervention.

Table 1: Changes in Rumen and Blood pH in Control and Acidotic Goats

Time (hrs)	Rumen pH (Control)	Rumen pH (Acidotic)	Blood pH (Control)	Blood pH (Acidotic)
0	6.5±0.2	6.4±0.3	7.40±0.02	7.39±0.03
6	6.4±0.2	5.6±0.3*	7.39±0.02	7.32±0.04*
12	6.5±0.1	5.2±0.2**	7.40±0.03	7.25±0.05**
18	6.4±0.2	4.9±0.2**	7.39±0.02	7.20±0.04**
24	6.5±0.2	4.8±0.3**	7.41±0.02	7.18±0.05**
48	6.4±0.2	5.4±0.3*	7.40±0.03	7.28±0.04*
72	6.5±0.2	6.0±0.2	7.41±0.02	7.34±0.03

* $p < 0.05$, ** $p < 0.01$ compared to control

Hematological alterations

Significant hematological changes were observed in goats affected with ruminal acidosis. Packed cell volume (PCV) and hemoglobin (Hb) concentrations were significantly elevated ($p < 0.05$ to $p < 0.01$) in acidotic goats compared to controls, particularly between 12 and 48 hours. Total erythrocyte count (TEC) also showed a moderate but significant increase during this period. No significant variations were observed in the control group throughout the study.

Hematological parameters

Table 2 summarizes the hematological parameters recorded in control and acidotic goats. The increase in PCV and Hb values indicates hemoconcentration, likely due to dehydration associated with fluid sequestration in the rumen. The rise in TEC suggests splenic contraction and mobilization of erythrocytes into circulation.

Table 2: Hematological Parameters in Control and Acidotic Goats

Time (hrs)	PCV (%) Control	PCV (%) Acidotic	Hb (g/dL) Control	Hb (g/dL) Acidotic	TEC (×10 ⁶ /μL) Control	TEC Acidotic
0	28.5±2.1	29.0±2.3	9.5±0.6	9.6±0.7	11.2±0.8	11.3±0.7
6	28.7±2.0	32.5±2.5*	9.6±0.5	10.5±0.6*	11.3±0.7	12.0±0.9
12	28.6±2.2	36.8±2.8**	9.5±0.6	11.8±0.7**	11.1±0.8	12.8±1.0*
18	28.4±2.1	40.5±3.0**	9.6±0.7	13.0±0.8**	11.2±0.9	13.5±1.1**
24	28.7±2.0	42.3±3.2**	9.7±0.6	13.8±0.9**	11.3±0.8	13.9±1.2**
48	28.5±2.1	38.6±2.9**	9.6±0.5	12.5±0.8**	11.2±0.7	13.2±1.0*
72	28.6±2.2	32.0±2.4*	9.5±0.6	10.8±0.7*	11.1±0.8	12.2±0.9

* $p < 0.05$, ** $p < 0.01$ compared to control

Erythrocyte osmotic fragility

Erythrocyte osmotic fragility was significantly increased in acidotic goats compared to controls. Hemolysis in affected animals began at higher NaCl concentrations, indicating increased fragility of erythrocyte membranes. The osmotic fragility curve in acidotic goats showed a rightward shift relative to controls.

The mean corpuscular fragility (MCF₅₀) values were significantly higher ($p < 0.01$) in acidotic goats, particularly at 12, 24, and 48 hours, with peak fragility observed at 24 hours. A gradual return toward normal values was observed by 72 hours.

Mean corpuscular fragility values

Table 3 shows the changes in MCF₅₀ values in control and acidotic goats. The significant increase in MCF₅₀ indicates

reduced erythrocyte membrane stability in affected animals.

Table 3: Mean Corpuscular Fragility (MCF₅₀) Values

Time (hrs)	MCF ₅₀ (% NaCl) Control	MCF ₅₀ (% NaCl) Acidotic
0	0.48±0.02	0.49±0.03
12	0.47±0.02	0.56±0.03**
24	0.48±0.02	0.62±0.04**
48	0.47±0.03	0.58±0.03**
72	0.48±0.02	0.52±0.03*

* $p < 0.05$, ** $p < 0.01$ compared to control

Lipid peroxidation (MDA levels)

Malondialdehyde (MDA) levels were significantly elevated in acidotic goats compared to controls, indicating increased lipid peroxidation. The highest MDA concentrations were observed at 24 hours ($p < 0.01$), followed by a gradual

decline at 48 and 72 hours, although values remained higher than controls. Control animals showed no significant variation in MDA levels throughout the study period.

MDA concentrations

Table 4 presents the MDA concentrations in control and acidotic goats. The elevated levels in affected animals reflect enhanced oxidative stress and damage to erythrocyte membranes.

Table 4: MDA Concentration (nmol/mL)

Time (hrs)	MDA Control	MDA Acidotic
0	2.1±0.3	2.2±0.4
12	2.0±0.3	4.8±0.5**
24	2.1±0.2	6.5±0.6**
48	2.2±0.3	5.2±0.5**
72	2.1±0.2	3.5±0.4*

* $p < 0.05$, ** $p < 0.01$ compared to control

Discussion

The rapid decline in ruminal pH observed within 12-24 hours following is consistent with the classical pathogenesis of acute ruminal lactic acidosis. Rapid fermentation of readily fermentable carbohydrates leads to accumulation of volatile fatty acids and lactic acid, resulting in a marked reduction in ruminal pH, as described by Owens *et al.*, (1998) [17] and Nagaraja and Titgemeyer (2007) [15]. Once the buffering capacity of the rumen is exceeded, proliferation of lactic acid-producing bacteria further accelerates acid accumulation and intensifies ruminal acidosis. The concurrent decrease in blood pH observed in the present study indicates progression from localized ruminal acidosis to systemic metabolic acidosis. This transition occurs through absorption of lactic acid and other organic acids into systemic circulation together with depletion of bicarbonate reserves (Radostits *et al.*, 2007) [19]. Similar findings have been reported in small ruminants, where severe ruminal acidosis resulted in systemic acid-base imbalance (Braun *et al.*, 1992) [4]. The present findings therefore confirm the close pathophysiological relationship between ruminal acidification and systemic metabolic disturbances.

The significant increases in packed cell volume (PCV), hemoglobin concentration (Hb), and total erythrocyte count (TEC) observed in acidotic goats indicate hemoconcentration associated with dehydration. Increased ruminal osmolarity during acute ruminal acidosis promotes osmotic movement of water from the bloodstream into the rumen, resulting in reduced plasma volume (Radostits *et al.*, 2007) [19]. The observed increase in TEC may additionally be associated with splenic contraction during stress, leading to release of stored erythrocytes into circulation. Similar hematological adaptations in goats under stress conditions have been documented by Daramola *et al.*, (2005) [6]. Hemoconcentration increases blood viscosity and may elevate shear stress within the microcirculation, thereby predisposing erythrocytes to mechanical injury and altered membrane stability.

The marked increase in erythrocyte osmotic fragility (EOF), reflected by increased MCF₅₀ values and a rightward shift in the fragility curve, indicates compromised erythrocyte membrane integrity during ruminal acidosis. These observations agree with the findings of Igbokwe (2018) [9], who reported that oxidative and metabolic stress adversely

affect erythrocyte membrane properties. Erythrocyte membrane stability depends on the integrity of membrane lipids, membrane-associated proteins, and cytoskeletal architecture. Under acidotic conditions, reduced extracellular pH may alter membrane protein conformation, while impaired ion transport mechanisms may result in intracellular ionic imbalance and cellular swelling. Increased circulatory stress associated with hemoconcentration may further compromise membrane resilience. Collectively, these factors increase membrane permeability and reduce erythrocyte resistance to osmotic stress.

The significant elevation in malondialdehyde (MDA) concentration observed in acidotic goats indicates enhanced lipid peroxidation and oxidative stress, suggesting that oxidative damage contributes substantially to erythrocyte membrane injury during ruminal acidosis. Reactive oxygen species (ROS) generated during acidosis may arise partly from endotoxin-mediated inflammatory responses. Lipopolysaccharides released from gram-negative bacteria stimulate immune activation and ROS production, as described by Abuelo *et al.*, (2015) [1]. Excessive ROS attack polyunsaturated fatty acids within erythrocyte membranes, initiating lipid peroxidation and generating secondary products such as MDA. These reactions disrupt membrane architecture, reduce membrane fluidity, and increase erythrocyte fragility. The positive association observed between MDA concentration and erythrocyte osmotic fragility further supports the direct role of oxidative stress in membrane destabilization. Lipid peroxidation may impair membrane elasticity and damage structural proteins such as spectrin, thereby reducing erythrocyte deformability and survival.

Under normal physiological conditions, erythrocytes possess efficient antioxidant defense systems, including superoxide dismutase, catalase, and glutathione peroxidase, which protect cells from oxidative injury. However, during acute ruminal acidosis, excessive ROS generation may overwhelm these antioxidant mechanisms, resulting in progressive cellular damage and increased erythrocyte fragility. Metabolic acidosis also affects hemoglobin function through the Bohr effect, whereby decreased blood pH reduces the affinity of hemoglobin for oxygen (Guyton and Hall, 2016) [8]. Although this mechanism facilitates oxygen release at the tissue level, severe acidotic conditions may impair oxygen loading in the lungs. Combined with increased erythrocyte destruction and reduced membrane stability, these alterations may compromise oxygen-carrying capacity and contribute to tissue hypoxia and reduced physiological performance.

Although partial normalization of ruminal and blood pH values was observed by 72 hours after induction, erythrocyte osmotic fragility and MDA levels remained elevated, indicating persistence of cellular injury beyond apparent correction of acid-base disturbances. Damaged erythrocytes may continue circulating with reduced functional lifespan, potentially predisposing affected animals to post-acidotic anemia and prolonged reductions in physiological efficiency. The findings of the present study are consistent with previous reports describing hemoconcentration, oxidative stress, and metabolic disturbances associated with ruminal acidosis in ruminants (Braun *et al.*, 1992; Nagaraja and Titgemeyer, 2007) [4, 15]. However, the present investigation further demonstrates a

functional relationship between oxidative stress and erythrocyte osmotic fragility, thereby providing a more integrated understanding of the systemic hematological consequences of ruminal acidosis in goats.

Conclusion

Acute grain-induced ruminal acidosis causes systemic metabolic acidosis, dehydration, and persistent oxidative stress in meat goats. Even as pH levels normalize, lipid peroxidation and osmotic mechanisms continuously compromise erythrocyte membrane integrity. Ultimately, monitoring erythrocyte fragility and oxidative biomarkers serves as a valuable diagnostic tool for early clinical management, underscoring the critical necessity of preventative feeding practices to mitigate ongoing cellular injury.

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