CHAPTER-5

**DISCUSSION**

**5.1 Prevalence of ruminal acidosis**

In present study the overall prevalence of ruminal acidosis was found 2.6%. A number of researcher showed nearly similar prevalence, **Bramley (2007)** found a herd prevalence of at least 3% in a survey of 100 Australian dairy cattle. **Krause and Oetzel (2006)** showed 4% prevalence of clinical acidosis in feedlot which is more or less similar with 4.04% found in cattle in present study. In goat, the prevalence found in present study was lower (1.9%), this is may be due to the present study was conducted on the cases that are taken into hospital. But **Blom (1993)** showed 0.2% prevalence; this is probably not a reliable indication **(Enemark and Jorgensen, 2001).**

A number of researcher showed higher prevalence of subacute ruminal acidosis, i.e **Bramley (2007)** found 10%, **Garret *et al*. (1997)** found 19% in early lactation and 26% in mid lactation, **Kleen *et al.* (2004)** found 11% in early and 18% in mid lactation and **Radostits *et al.* (2006)** found up to 20% prevalence in subacute ruminal acidosis. The prevalence of subacute ruminal acidosis is higher than that is found in present study, this is because in this study all are clinically affected animals that’s taken to hospital are considered. Some of the subacute cases may not be suspected by owner as sick or might not take into hospital for treatment.

There were found no statistically significance difference of prevalence of ruminal acidosis in between cattle and goats. These findings agree with **Radostits *et al.* (2006)** who reported that all types of ruminant are susceptible to ruminal acidosis.

**5.2 Risk factors**

It was revealed in the present study that sudden ingestion of large amount of easily digestible carbohydrates eg. feeding of cooked rice predispose the ruminal acidosis in most of the cases. Other carbohydrate source like- potato, jackfruit residue, bread, palm also predispose the condition in some cases. Several researchers also comment the predisposing factor of ruminal acidosis as easily digestible carbohydrates or grain **(Penner *et al.*, 2007; Beauchemin and Penner, 2009).**

**5.3 Clinical signs**

The clinical signs observed in animals with ruminal acidosis were very similar to that in other experimental models, where different kinds of substrates and animal species were used. Typical symptoms of ruminal acidosis as anorexia, decreased rumen motility, dehydration, oligouria and diarrhea. Such demonstrations coincided with decreasing pH of the rumen fluid, especially when values ​​were below 5. These observations were verified by **Aslan, *et al*. (1995), Owens *et al*. (1998) and Metkari *et al*. (2001)**, who reported this manifestations are, due to the increased concentration of lactic acid, and the elevation of ruminal osmolarity of the medium in relation to the bloodstream, which unleashed by these clinical changes.

In this study, there were found moderate dehydration in most of the cases and some also severe dehydration. This dehydration is due to high osmotic pressure of ruminal contents in acidotic condition that pulls up water from systemic circulation **(Owens *et al*., 1998)**. This high osmolarity of ruminal content reduce acid absorption, that further increase the osmotic pressure of rumen. Dehydration equals 4–6% body wt, but losses may reach 10–12% in severe cases **(Tabaru *et al*., 1990).** The anuria or oligouria is due to severe dehydration resulting reduced perfusion.

There were found anorexia and inappetance because of an elevation of osmotic pressure in the rumen is sensed by the wall of reticuloruminal to inhibit feed intake **(Carter and Grovum, 1990)**. **Krause and Oetzel (2006)** found anorexia in acute case of ruminal acidosis. **Underwood (1992)** also reported inappetance is a clinical sign of ruminal acidosis.

In present study, there were found abdominal distension in few cases as a clinical sign, it is due to high osmotic pressure (Above 350 mOsm) inhibit bacterial digestion of fiber and starch causing ruminal content to become stagnant **(Scott, 1975)** and also due to pulls up water from systemic circulation by high osmotic pressure of rumen **(Owens *et al*., 1998; Boton and Pass, 1988).** **Tremere *et al*. (1968)** reported that abdominal distension is a clinical sign of acute ruminal acidosis.

Diarrhoea found as a clinical sign of ruminal acidosis. As lactate passes from the abomasum into the intestinal tract, further absorption occurs, promoting an osmotic gradient. The resultant increase in fluid in the lumen is responsible for the profuse diarrhoea and subsequent dehydration seen in clinical acidosis. Increased proportions of undigested feed resulting from reduced microbial cellulolytic activity at a lower ruminal pH also contribute to diarrhoea **(Bolton and Pass, 1988).**  **Krause and Oetzel (2006)** also reported similar findings in a study on dairy cattle. However **Kleen *et al*. (2003)** and **Nordlund *et al*. (1995)** reported that the fecal alterations are usually transient and only a few animals have loose feces.

Lameness is found only one case in cattle but no lameness is found in goat affected with ruminal acidosis. Lameness is due to laminitis in ruminal acidosis **(Shaver, 2005)**. Laminitis is due to elevated histamine concentrations and blood vessel damage due to uncontrolled elevations in blood pressure inside the hoof **(Vermunt and Greenough, 1994). Tremere *et al*. (1968)** and **Owens *et al*. (1998)** also found lameness in ruminal acidosis. But **Stone (2004)** reported that the exact relationship between SARA and laminitis is not known. **Nordlund *et al*. (1995)** and **Enemark *et al*. (2002)** noticed subacute and chronic laminitis in SARA affected cow.

Changes in microbial fauna of the rumen fluid of animal studied with respect to decreased motility or absence of motility. According to **Krogh (1959)**, protozoa lose their activity when the pH drops to values ​​between 5.5 and 5.0, disintegrating or suffering rumen lysis occurs when an increase in acidity of the medium, and pH reaches values ​​below 5.0; **Ahuja *et al*. (1990)** also reported that increasing the osmotic pressure in the environment causes changes in rumen protozoa population. In present study, it was noticed that in some of the animals (1 cattle and 2 goats) in delayed cases there were appear microflora movement, which is consistent with the information **Basak *et al*. (1993)** who reported this event, synchronized with the improvement of the condition in the rumen environment.

**5.4 Characteristics of rumen fluid**

In most of the cases 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 **(Dunlop, 1972; Dougherty *et al*., 1975)**. These changes were similar to events observed in goats and sheep with rumen acidosis studied by **Huber (1971)** and **Cao *et al*. (1987).**

**5.5 Rumen fluid and blood pH**

In the present study, the rumen fluid pH were found between minimum value of 4.2 and maximum of 6 in cattle and 4.2 and 5.8 respectively in goat. **Nocek (1997)** and **Owens *et al*. (1998)** showed the diagnostic ruminal fluid pH for acute acidosis is < 5 – 5.2 and for subacute acidosis is between 5 – 5.2 **(Nagaraja and Titgemeyer, 2007)**. In present study, the cases that showed clinical sign among them 6 cattle and 7 goat fall in this pH range, remaining 2 cattle and 1 goat (pH is higher but not above 6) also in acidotic condition because **Plaizier *et al.* (2008)** reported that ruminal pH below 6.16 for more than 5.2 hours per 24 hours also consider as ruminal acidosis. **Owners *et al.* (1998)** and **Brown *et al*. (2000)** showed clinical diagnosis of ruminal acidosis require blood pH to fall below 7.3 but in present study there were found blood pH fall in 5 cases of cattle and 6 cases in goat. There were only slide decrease of blood pH and the remaining 5 cases blood pH do not fall at all, it’s because pH of the body fluid is buffered by bicarbonate. For this reason body fluid pH may or may not be depressed during acidosis, depending on degree to which bicarbonate compensation is possible **(Owens *et al.,* 1998)**. Although **Lachmann and Siebert (1980)** found that the blood gas parameters were not affected in case of chronic ruminal acidosis.

**5.6 Serum calcium**

In this study, the average serum calcium level was found 9.09 $\pm $ 1.26mg/dl, but there were found hypocalcaemia in four cases. These findings have similarity with **Divers and Peek (2008)** who reported that hypocalcaemia is constant finding in ruminal acidosis. **Bolton and Pass (1998)** found hypocalcaemia resulting from calcium malabsorption.

**5.7 Correlation between the duration of illness of different cases and ruminal fluid and blood pH**

The findings of positive correlation between the duration of illness of different cases and ruminal fluid pH i.e. there was a decline in values ​​for ruminal pH early in the process, and these findings were similar to those found by **Dunlop** **(1972)** and **Nocek (1997)** who attribute this decrease in pH alteration is due to changes rumen microflora, where Gram-negative bacteria (acid-sensitive) are replaced by Gram-positive bacteria, particularly *S.* *bovis* and *Lactobacillus* *sp*, which are the main producers of lactic acid, forms D (-) and L (+) which is regarded as a strong acid, by having a p*K*a very low.

**Sato *et al.* (2012)** in a study of induced acidosis on 10 cross breed goat were found similar correlation between rumen pH and duration of induction i.e. they showed up to certain duration the rumen pH decrease and then increase. In their findings they showed lower values of pH observed in 12 hours post induction than (PI) 4 hours PI. Ruminal pH decrease up to 20 hours of onset of acidosis then increase with increase duration.

Starting restoration to normal values of pH occurred from 48h PI. The initial low pH is due to feeding of large amount of starch, the growth of *Streptococcus bovis* is no longer restricted by lack of energy source and this population grows faster than other species of rumen bacteria **(Russell and Hino, 1985).** *Streptococcus bovis* is a major culprit in lactic acidosis. In latter stage the ruminal pH increases due to absorption of acid in circulation **(Tabaru *et al*., 1990).**

There were also found negative correlation between duration of illness of different cases and blood pH. Blood pH decrease due to absorption of acids into blood **(Tabaru *et al*., 1990)**, but changes is very limited because blood has buffering capacity to neutralize the acid and liver have greater capacity to metabolize lactate **(Naylor *et al*., 1994)**. So only if catabolism of lactate is overloaded to liver (In ruminal acidosis) it creates systemic acidosis **(Owens *et al*., 1998)**.

**5.8 Response to treatment**

The recovery time was variable in clinical animals. **Kezar and Church (1979)** and **Afonso *et al*. (2002)** reported, to be clinical recovery of animals, it is necessary to pH above six levels into the rumen. 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 all the animal recovered. **Redostits *et al*. (2006)** 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, where there was a reduction or disappearance of agents considered harmful, acid producers lactic flora and the restoration of Gram-negative considered as the main fermentative lactate. Creating an improvement in rumen environment, especially for pH, thus favoring the return of appetite, with this improved buffering and facilitated the restoration of the microbial population that facilitated this way clinical recovery **(Goad *et al*., 1998)**.