Abstract: Subacute ruminal acidosis is considered as the most important nutritional disease in cattle. It is considered subacute when the low ruminal pH is caused by excessive accumulation of volatile fatty acids without persistent lactic acid accumulation, later restored to normal pH by animal’s own physiologic responses. It is considered as an economically important condition seen even in well managed herds which results in subsequent deterioration of health and loss of production. Subacute ruminal acidosis can induce various complications or sequelae, including milk fat depression, diarrhoea, laminitis, parakeratosis–rumenitis–liver abscess complex, poor body condition, metabolic acidosis, bloat, abomasal diseases, polioencephalomalacia and reproductive problems in ruminants especially in cattle farms.

Keywords: SARA, production performances, dairy.

INTRODUCTION

Sub acute ruminal acidosis (SARA) is the most important nutritional disease affecting dairy cattle (Enemark, 2008; Mohebbi Fani et al., 2010; Tajik, 2011). It can be defined as low ruminal pH, ranging from 5.0 to 5.5, caused by excessive accumulation of volatile fatty acids without persistent lactic acid accumulation which later restored to normal pH by animal’s own physiologic responses. Ruminal pH will fluctuate during the 24-hour period due to alternate concentrate and roughage feeding (Kleen et al., 2003).

A dairy herd is said to be SARA affected when thirty percentage of the herd has a rumen pH less than or equal to the cutoff value of 5.5 for more than 3 hours in a day (Garret, 1996). SARA is generally reported as a consequence of feeding high grain diet rather than forage diet to which ruminants are mainly adapted (Krause and Oetzel, 2006).
In cattle, a sudden change from dry cow ration to lactation ration can cause SARA. An optimal absorption capacity of ruminal mucosa is essential for effective pH regulating mechanisms within the body. Hence, an adaptive period of four weeks is very much essential for optimal proliferation of rumen mucosa and thereby proper absorption of nutrients (Dirksen et al., 1984).

Subacute ruminal acidosis can induce various complications or sequelae, including milk fat depression, diarrhoea, laminitis, bloat, poor body condition, polioencephalomalacia, abomasal displacement, reproductive problems, liver abscess complex, caudal vena cava syndrome, epistaxis, haemaptysis and immunosuppression in ruminants (Kleen et al., 2003; Enemark, 2008). Also it has been considered as the predisposing factor for some other diseases like hemorrhagic bowel syndrome (Tajik et al., 2010).

MILK-FAT DEPRESSION (MFD)
A depression in milk-fat percentage has been reported in cows affected by SARA (Nordlund et al., 1995; Chalupa et al., 2000; Oetzel, 2000; Kleen et al., 2003; Oetzel, 2003; Stone, 2004). Experimentally induced SARA, either by adding grain pellets to the diet or by replacing alfalfa hay with alfalfa pellets, reduced milk fat percentage but increased milk protein percentage (Fairfield et al., 2007; Khafipoor et al., 2007; PLAIZIER et al., 2008).

Low-milk fat syndrome and milk-fat depression (MFD) are frequently used terminologies to describe conditions where there is a considerable drop in milk-fat, mainly because of

Figure 1. Fermentation pattern and rumen environment characteristics in relation to rumen pH (Dirksen, 1984).
Effect of Sub-Acute ruminal Acidosis (SARA) on ....

Mistakes in feeding strategy (Baumann et al., 2001). The decrease of milk-fat remains undetected in the bulk tank testing because it usually occurs in individuals (Garrett, 1996; Nocek, 1997). Experimental induction of SARA affected the milk fatty acid profile (Enjalbert et al., 2008). Alterations in the ruminal fermentation pattern during SARA are considered to be responsible for the milk fat depression. SARA also causes transient reduction in overall milk production in cattle (Oetzel, 2000).

Milk fatty acid profile can be considered as an indicator tool for SARA in cattle. Higher proportions if iso fatty acids in solid associated bacteria suggest their enrichment with cellulolytic bacteria. In contrast, higher proportions of anteiso C15:0 in liquid associated bacteria indicate their enrichment in pectin and sugar fermenting bacteria (Fievez et al., 2012).

Milk fat depression has been associated with a reduction in the acetate to propionate ratio, increased insulin (Bauman and Griinari, 2003; Plaizier et al., 2008), and increased production of trans-octadecenoic acids in the rumen by biohydrogenation pathway (Griinari et al., 1998; Bauman and Griinari, 2003; Plaizier et al., 2008; Fievez et al., 2012). Propionate concentration will be increased during SARA causing decrease in acetate to propionate ratio (Gozho et al., 2006; Fairfield et al., 2007; Khorasani et al., 2007; Plaizier et al., 2008). An increase in insulin level will decreases lipolysis and this explain why stage of lactation and energy balance could affect the milk fat depression (Bauman and Griinari, 2003; Plaizier et al., 2008). The conversion of body fat into milk fat is much greater in cows those are in negative energy balance (Plaizier et al., 2008).

Reasons for low-milk fat syndrome includes, feeding strategy with a ration high in carbohydrate but deficient in roughage, feeding of processed roughage (e.g. by pelleting) and introduction of unsaturated fatty acids in the feeds (Kleen et al., 2003). MFD was associated with alteration in the ruminal fermentation pattern. Acetate concentration in the rumen liquor was found to be reduced while propionate and butyrate levels were increased (Murphy et al., 2000; Khorasani and Kennelly, 2001). Some studies stated that butyrate level was decreased together with the acetate in the rumen liquor (Kennelly et al., 1999). Introduction of buffering substances to the high-concentrate diet re-established a higher ruminal pH and prevented the drop in milk-fat content (Khorasani and Kennelly, 2001). It was concluded that formation of trans-C18:1 fatty acids, which would inhibit the synthesis of milk-fat in the mammary gland thereby resulting in MFD (Kennelly et al., 1999). It seems
that MFD also may occur in situations in which the adaptation of the ruminal flora prevents the development of SARA with its clinically detectable consequences (Enjalbert et al., 2008).

**LAMINITIS**

Laminitis is the most common clinical sign which is repeatedly mentioned with subacute ruminal acidosis (SARA) in cattle (Garrett, 1996; Nocek, 1997; Enemark et al., 1998; Oetzel, 2000; Ivany et al., 2002; Manju and Ajithkumar, 2014). It imparts an important role in dairy farming by considerable economic loss, impaired animal welfare and pre-disposition to other diseases (Nelson and Cattell, 2000).

Cattle fed with a diet high in concentrates are more prone to laminitis (Livesey and Fleming, 1984; Manson and Leaver, 1988; Kelly and Leaver, 1990). The feeding regime in which frequency as well as quantity of concentrate feed has a certain influence and more concentrate in a shorter period of time will cause locomotive pathology (Bergsten, 1994).

SARA can cause release of certain vasoactive substances like histamine, lipopolysaccheride endotoxins in to the circulation by the disintegration of Gram negative bacteria. It will cause injury to microvasculature of corium followed by hypoxia to extremities leading to laminitis (Plaizier et al., 2009).

Body condition of dairy cows is having a certain relation in the development of non-infectious laminitis (Gearhart et al., 1990). Over conditioned cows at drying off period are at greater risk for foot problems while cows experiencing moderate net energy balance are at slightly lower risk of having non-infectious lameness (Heuer et al., 2000). Hoof discoloration, ulcers, abscesses, sole haemorrhages and misshapen hooves have been observed as a complication of SARA in cattle (Nordlund et al., 1995; Oetzel, 2000). The existence of SARA should be examined in herds having reports of clinical lameness (Garrett, 1996).

**ALTERATIONS IN FAECES & DIARRHOEA**

Sub-acute ruminal acidosis can cause alteration in the faeces as well as nonspecific diarrhoea in affected cattle (Nordlund et al., 1995; Garry, 2002; Oetzel, 2000). These alterations in faeces are generally transient in nature (Garry, 2002). Rumination, activity of the ruminal flora and ruminal passage determines the structure and consistency of the faeces (Garry, 2002). The colour appears to be brighter and yellowish; pH is lower than normal. It will have a sweet–sour odour (Oetzel, 2000). Size of ingesta particles will be large, around 1–2 cm in which whole undigested cereal grains may be present in faeces (Garry, 2002).
Massive outflow of fermentable carbohydrates from the rumen causes post-ruminal fermentation in intestine which subsequently alters the consistency of faeces (Oetzel, 2000). Increased osmolarity in gastrointestinal tract can lead to binding of fluid in to the intestinal lumen resulting in diarrhoea (Garry, 2002).

**PARAKERATOSIS–RUMENITIS–LIVER ABSCESS COMPLEX**

SARA is associated with liver abscesses (Dirksen, 1985; Nordlund et al., 1995; Garry, 2002; Oetzel, 2000), abscesses or inflammation in kidneys (Oetzel, 2000), lungs (Nordlund et al., 1995), and heart (Oetzel, 2000). Sub acute ruminal acidosis causes inflammations of different organs and tissues and subcutaneous abscesses can be easily identified by physical examination (Nordlund et al., 1995). Moreover, haemoptysis and epistaxis are also reported in SARA affected herds. These are related to either caudal vena caval syndrome or bacterial pneumonia (Nordlund et al., 1995; Oetzel, 2000).

Growth of ruminal epithelium has a direct link to the short chain fatty acids (SCFAs) present in the tissue. Propionic and butyric acid mainly promote the growth of ruminal papillae and provide a higher absorption from the rumen by the mucosa (Kleen et al., 2003). The clearance of volatile fatty acids from rumen is affected by the size and density of rumen papillae and that determine how fast it is absorbed (Plaizier et al., 2009). Freshly calved cows are at a higher risk of SARA compared to cows in mid and late lactation since ruminal absorption capacity for volatile acids can decreased by 50% during the dry period due to a reduction in the length and density of rumen papillae (Dirksen et al., 1985).

Whenever, SCFA accumulate in rumen during the phases of acidosis in larger amounts, which will lead to parakeratosis and rumenitis (Gabler, 1990). Rumen papillae will clump together and reduces effective surface area followed by its erosion (Dirksen et al., 1984). The ruminal mucosa which normally acts as a barrier between ruminal environment and bloodstream, undergo impairment. Translocation of rumen bacteria (*Fusobacterium necrophorum* and *Arcanobacterium pyogenes*) to liver via portal blood flow results in liver abscesses. It can be metastatic to pulmonary circulation via posterior vena cava causing rupture of pulmonary capillary vessels leading to epistaxis, haemoptysis and sudden death (caudal vena caval syndrome). It can also spread to other tissues in the body-like lungs and kidneys (Nordlund et al., 1995; Nocek, 1997).

Economic consequences for parakeratosis–rumenitis–liver abscess complex include low milk yield, higher rate of culling and reduced annual turnover within the dairy herd. The loss may be exaggerated due to condemnation of the carcasses at meat inspection. Once the
parakeratosis is severe enough to alter the resorption capacity, it will negatively influence the body condition of the animal (Kleen et al., 2003).

**LOSS OF BODY CONDITION**
Number of thin dairy cows with low body condition score is a common finding in SARA affected herds (Kleen et al., 2003; Nourdlund et al., 1995). However, body condition score can’t be used as a tool to differentiate SARA affected and unaffected dairy herds (Kleen, 2004; Tajik et al., 2009).

**RUMEN TYMPANY (BLOAT)**
Low fibre ration in the diet causes reduced rumen motility and low ruminal pH. Excess release of mucopolysaccharides and unknown macromolecules synthesized from rumen bacterial disintegration can create stable foam which hinders eructation of gases leading to ruminal tympany (Enemark, 2008). Ruminal stasis may also allow accumulation of free gases (Rebhun, 1995).

**REPRODUCTIVE PROBLEMS**
SARA can indirectly affect fertility, calving as well as health of the newborn calf. Negative energy balance produced by cyclic feeding and reduced dry matter intake result in inadequate maturation of the first wave of post partum ova (Britt, 1995; Enemark, 2008). Steroid profiles of the body will changes in association with SARA prior to calving causes impairment in uterine contractility, dystocia and retention of placenta (Matthias et al., 2002).

**POLIOENCEPHALOMALACIA (CEREBROCORTICAL NECROSIS)**
Thiamine or B1 vitamin is normally produced by rumen bacteria and cattle do not require it normally through the feed (Abeysekara et al., 2007). Whenever there is high grain diet in the feed, normal bacterial flora gets destroyed while those producing the thiaminase enzyme will predominate in the rumen. Thiaminase 1 is produced by Bacillus thiaminolyticus and Clostridium sporogenes while thiaminase II by Bacillus aneurinolyticus, that catalyses cleavage of thiamine. It will cause thiamine deficiency leading to Polioencephalomalacia (Hernandez et al., 2014).

**ABOMASAL DISPLACEMENT AND ABOMASAL ULCERS**
SARA is considered to be a major risk factor for abomasal displacement. Increased backward and forward flow of ruminal derived gases (SCFA, CO₂ and CH₄) between the abomasums and the forestomach result in abomasal atony followed by dilatation and subsequent displacement. Low fibre content in the ration is the most important single factor for the occurrence of abomasal displacement (Enemark, 2008). Occurrence of abomasal ulcers has
been linked to intensive management and feeding of highly acidic diets consisting of concentrates and silage (Rebhun, 1995; Enemark, 2008).

**HIGH CULLING RATE**

Unexplained death, loss of body condition, lameness and nonresponsive pathological conditions are the most important causes for the increased culling rate (Oetzel, 2003; Kleen *et al*., 2003; Tajik *et al*., 2011).

**METABOLIC ACIDOSIS**

Sub acute ruminal acidosis causes increased accumulation of total volatile fatty acids, especially propionic acid and butyric acid, rather than lactic acid in the rumen (Da-cheng *et al*., 2013). Among the short-chained volatile fatty acids (VFAs), only the acetic acid will reach the peripheral circulation while butyric acid is transformed largely in the rumen wall into β-hydroxy-butyric acid which can lead to alimentary form of ketosis. Propionic acid is converted into glucose in liver (Owens *et al*., 1998; Enemark, 2008). Serious cases of intracellular acidosis can occur even under mild grade of sub acute or chronic acidosis (Enemark, 2008). It will compromise cellular functions in rumen wall as well as in liver, resulting in high concentrations of VFAs in the peripheral circulation and it will lead to metabolic acidosis (Owens *et al*., 1998).

**IMMUNOSUPPRESSION**

Metabolic acidosis produced as a complication of SARA can lead to increased cortisol secretion in the body, decreased migration of neutrophil and decreased phagocytosis which can result in immunosuppression. In addition, increased blood levels of aspartate aminotransferase (ASAT) and Glutamate dehydrogenase (GLDH) along with impaired liver function can also create immunosupression leading to increased susceptibility to infectious diseases like mastitis, endometritis and pneumonia (Matthias *et al*., 2002).

**Conclusion**

Sub acute ruminal acidosis results in so many complications including laminitis, bloat, polioencephalomalacia, abomasal diseases in ruminants. It also affects the milk quality by lowering the milk fat content. It triggers an oxidation process of protein, carbohydrate and lipids in liver, thereby consumes more precursors needed for milk fat synthesis resulting fat and protein reduction in cow milk.

Diagnosis of SARA affected cattle herd has its own importance since it will affect the economy of the farmers as well as the health status of the animals. Certain complications of SARA like polioencephalomalacia, liver absesses, abomasal diseases, rumen tympany and
Epistaxis can result in death of animals. SARA can affect reproductive performance of the animals and cause a delay or inadequate maturation of first wave of post partum ova there by resulting reproductive abnormalities. It can also cause immunosuppression to the animals which will predispose so many other diseases.

Hence, early and prompt diagnosis of SARA in dairy herds has an immense importance to tackle such life threatening complications and also to stabilize the economy of the dairy farms in terms of production.

References


