International
Research Journal of

ANIMAL AND VETERINARY SCIENCES

Volume 02 | Issue 01 | 2020

SciRange
PUBLICATIONS
www.scirange.com

SciRange

Open Access

International Research Journal of Animal and Veterinary Sciences

Review Article

pISSN: 2663-7154, eISSN: 2663-7162

The Relationship between Nutritional Strategies and Ruminants Disorders: A Review

Mohamed M. Abdou and Ahmed M. Abd El Tawab

Department of Dairy Sciences, National Research Centre, 33 Bohouth St. Dokki, Giza, Egypt

ARTICLE INFORMATION

Received: January 26, 2020

Accepted: February 29, 2020

Corresponding Author:
Dr. Ahmed M. Abd El Tawab,
Department of Dairy Sciences,
National Research Centre,
33 Bohouth St. Dokki, Giza, Egypt

ABSTRACT

Nutrition plays an important role in preventing ruminal disorders which takes place in transition period. Ruminal disorders (ketosis, acidosis, tympany and milk fever) have adverse effects on productive performance of dairy animals. Those types of problems take place in high yielding animals and using Nutritional Strategies can be overcome on it. The aim of this review was carried out to investigate the relationship between feeding practices and animal production, welfare and health. Transition period (21 days before calving to 21 days after calving) is disaster period for dairy animal because dairy animal need a lot of energy to compensate these efforts during parturition and lactation. Most important metabolic disorders happen at that time, acidosis, ketosis, milk fever and bloat (tympany). Metabolic disorder has significant health and economic problem that account for extraordinary financial loss in ruminants. Although the great efforts in management, metabolic disorder remains the main cause of animal mortality, weight gain delay and decreased milk production. It could be concluded that ruminal disorder is not a pathogenic problem but it is managerial problems in the transition period and it could be solve by managing transition cow to minimize metabolic disorder.

Key words: Ruminants, nutrition, ketosis, acidosis, bloat, milk fever

INTRODUCTION

In all lactating cattle, the imbalance between nutrient supply and nutrient demand can be quite evident in early lactation, with milk production sustained, in part, through mobilization of body tissues due to the cow's inability to consume sufficient dry matter to satisfy her nutritional needs. As a consequence, cows after calving may incur different metabolic problems (ketosis, milk fever and fatty liver). Also, it was important to recognize that rumen acidosis also occurs in intensive grazing systems. Research has indicated that cows consuming copious amounts of fresh pasture, containing high levels of soluble sugar, can have rumen pH values below 5.5 for significant periods during each day. Rumen acidosis has been associated with increased inflammatory response and laminitis. Laminitis, not only causes pain, but also compromises milk production[1,2]. Reduced rumen pH is associated with an accumulation of lipopolysaccharide in the rumen[3,4] due to the lysis of Gram-negative bacteria, which will elicit an inflammatory response. Moreover, Beef cattle reared under intensive conditions are equally exposed to rumen acidosis with similar consequences on laminitis as for

dairy cows. These animals are typically fed rations high in non-fiber carbohydrates to promote high daily weight gains. These problems are appearing when the fattening animal was fed on a large amount of the concentrates as a replacement of roughage in the diets[5]. Ketosis causing disturbance in metabolism of carbohydrate and fatty acids that lead to increase production of ketone bodies also, ketosis takes place as a result of negative energy balance which, cause fatty liver in dairy cattle[6]. Milk fever is the increase calcium absorption from the blood into the milk at calving. This causes inability of the cow to change to keep blood calcium levels up[7]. Bloat is a complex disease but simply expected under field conditions, as a result, field observations have led to varied and different theories about its causes and facts. It generally occurs up to 3 days after animals begin a new diet, moreover certain legumes such as alfalfa, ladino clover and grain concentrates, promote the formation of stable foam[8]. The aim of this review was carried out to investigate the relationship between feeding practices and animal production, welfare and health.

THE COMMON RUMINAL DISORDERS

Ruminal acidosis: Recently, high milk production in dairy cows is sustained by feeding high concentrates and less forage. This lead to rumen acidosis, causing decrease feed intake, low in in ruminal fermentation, diarrhea, laminitis, inflammation and liver abscesses[9]. Rumen acidosis was resulted by the rapid rumen fermentation of highly soluble carbohydrates that were consumed by large amounts cereal grains such as wheat, oats, barley, corn and sorghum[10]. Starch is high fermentable carbohydrate and produce lactic acid more than the absorption of rumen. The lactic acid production was affected by the type of feeding for such as wheat produce more than maize[11]. Increasing lactic acid in dairy cattle causes reduction in ruminal pH[10].

Ruminal acidosis can be classified into two types: The first type is acute ruminal acidosis which lead to decreased in blood pH and bicarbonate, loss of appetite, reduced rumination, dehydration, diarrhea and low in milk production and composition all those disorder result from ruminal acidosis[12-14]. The second type is subacute ruminal acidosis it is unnoticed and characterized by low productive performance of dairy animal and low ruminal pH as a result of low-fiber ration[15]. Ruminal pH was decline from 5.5 to 5.0 in subacute ruminal acidosis and lasts for 111 to 180 min in a 24 h period[16-19]. This period was not adequate to cause immediate clinical signs[20]. Prebiotics or probiotics can improve ruminal ecology.

HOW DOES ACIDOSIS HAPPEN IN DAIRY CATTLE?

- **Diagnosis of acidosis:** Included decline in blood pH below 7.2, reduction in rumen pH <4.5 and disturbance in nervous system with signs (Fig. 1)

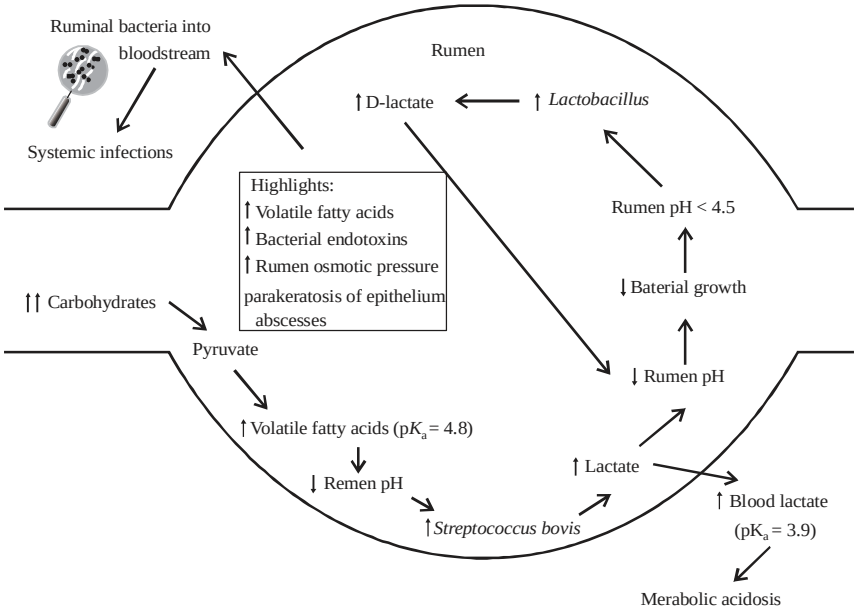

Fig. 1: Diagnosis of acidosis

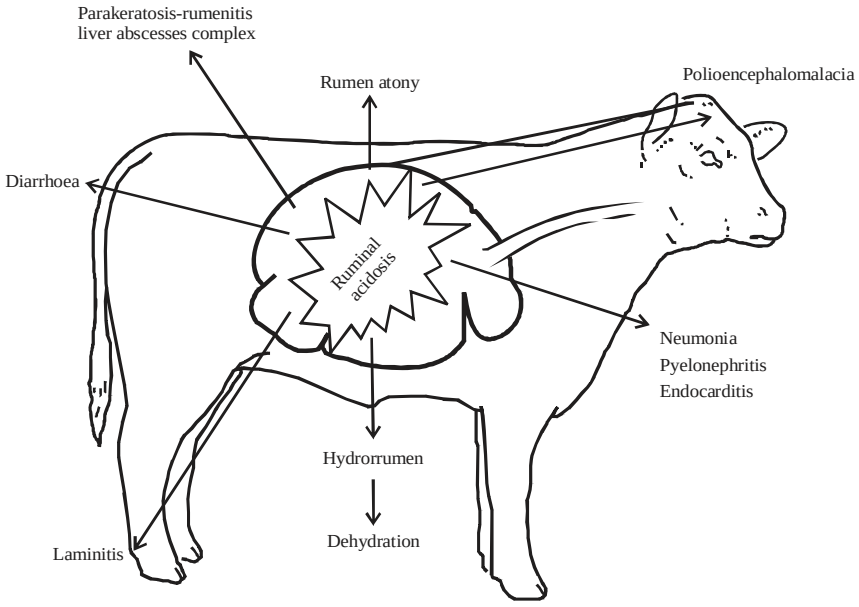

Fig. 2: Clinical signs of ruminal acidosis

- **Clinical signs:** Included the symptoms depend on the kind and a lot of feeding, moreover the time of feeding. Acidosis can be classified into subacute, acute and pre-acute forms[21] (Fig. 2)

In subacute cases, affected animals remain bright, alert, anorexia, dehydration, rumen motility is reduced, diarrhea and abdominal pain. In dairy animals, low milk production. In some cases, there are abortions and premature births[22].
In acute cases, affected animals obtunded, ataxic, tympany and weak ruminal motility, anorexia, diarrhea, grey feces and blooded feces. Also, the animal appeared in high rectal temperatures and dehydration in early stages[23].

STRATEGIES TO REDUCE OR PREVENT RUMINAL ACIDOSIS

Buffer additives: To overcome ruminal acidosis can add buffer additives to eliminate the negative effects of acidosis and the rate of addition in rations was by about 0.5 to 2.5%. Sodium bicarbonate, desmodium carbonate, magnesium oxide, potassium carbonate and limestone were used in dairy cows' rations[24].

Phytogenic additives: Plant extracts (oil) was used to improve rumen ecology in dairy animal when the ruminal pH was decreased (pH 5.5). *Allium sativa*, *Capsicum anuum*, *Yucca schidigera* and *Cinnamonum cassia* extracts change ruminal fermentation to produce more propionate[25]. Essential oils modify microbial activity and increase the total of Volatile Fatty Acids (VFA)[26]. Moreover, these additives were used in beef heifers which fed on high concentrate ration through modification of rumen fermentation[27]. Addition of a garlic oil, propyline modify ruminal fermentation where produce more propionate[28].

Prevention of ruminal acidosis: In animals fed high-concentrate rations, high digestible ration should be avoid Changes in feed stuffs should be gradually (in transition period) because adaptation of the rumen microbes to new feeds takes long time. It is necessary to increase roughage (to increase saliva), good quality hay should be added. Feed additives can be added were ionophores antimicrobials, bicarbonate and limestone which can reduce rumen acidosis[29].

Ruminal ketosis: Ketosis takes place in transition period between 21 days before calving and 21 days after calving. Decreasing in energy sources were causing disturbance in metabolism of carbohydrate and fatty acids that lead to increase production of ketone bodies. There are two types of ketosis: Primary ketosis as a result of low glucose which necessary for milk production. Secondary ketosis Anorexia takes place as a result secondary for ketosis. Ketosis is two types, clinical and subclinical ketosis. Subclinical ketosis is defined as increasing of ketone bodies without obvious symptoms. Whereas, clinical ketosis is due to increase of ketone bodies in addition to obvious clinical signs including anorexia, weight loss and blindness[30]. Ketosis takes place as a result of negative energy balance which, cause fatty liver in

dairy cattle[6] and hypoglycemia, because of increasing requirement for glucose and low propionate production during 21 days after calving. Starvation is the main cause of ketosis during transition period[31].

Clinical signs ketosis: There is a high concentrations of ketone bodies were found in blood, urineor milk during early lactation,with anorexia, loss of body weight, blindness, hard dry feces, hypoglycemia, low level of hepatic glycogen, low appetite to concentrate and low milk production[32]. Nervous symptoms in primary ketosis[33] mell of ketone bodies in breath or milk in dairy animal affected with ketosis.

Subclinical ketosis: Without symptoms, only high ketone bodies are showed[34].

Treatmentof ketosis: Cows may win over ketosis through decrease milk production that's lead to a loss of cost whereas, treatment is very important[33]. The treatment as following: increasing feed intake, intravenous dextrose solutions, glucocorticoids and oral propylene glycol[35] in case of anorexic, IV of glucose or possibly fructose. Administration of glucose is allowing the reversal of ketogenesis[36]. The intravenous injections were of 500 mL of 50% glucose.

Prevention of ketosis: Nutritional management must save enough energy in ration of dairy cattle during transition period and high fiber, avoiding decrease particle size, protein content, feeding systems and rumen size to prevent of ketosis. Non-nutritional treatment of the animals with oral sodium propionate, oral propylene glycol is effective to prevent ketosis and high milk production[37].

Milk fever (hypocalcemia): The main cause of milk fever was due to disturbance in calcium balance. There are three factors which effect on this balance: 1. high colostrum content of calcium more than the ability of absorption from intestine, 2. difficulty of calcium absorbed from intestine at calving, 3. low mobilization of calcium storage in skeleton causes problems to sustain normal serum levels[38] (Table 1).

Table 1: Factors effect on milk fever

No.	Causes	Results
1	Nutritional state before calving	High K, over fat cow and very thin cows
2	Decrease feed intake at parturition time	Decrease absorbed calcium and intake
3	High calcium intake before calving	Parathyroid hormone decrease
4	High P intake before calving	Vit-D metabolism Was inhibited
5	Low Mg before calving in ration	Inhibition of PTH
6	DCAD balance	Increase DCAD balance

Mechanism of milk fever in dairy cow: The cause of milk fever is the increase calcium absorption from the blood into the milk at calving. This causes inability of the cow to change to keep blood calcium levels up. Two ways a cow can get more calcium, calcium bone or ca from the small intestine. Parathyroid hormone (PTH) and active vitamin D are affected by this process. PTH release is stimulated by low blood calcium[7,39].

Prevention of milk fever: There are many methods to prevent milk fever as follows: Feeding ration deficient in calcium in the late dry period, feeding ration rich in calcium before calving and vitamin D in ration of dairy cattle, low DCAD and Mg additives[40], magnesium level before calving must be adjusted, should be aware body condition, must adjusted carbohydrate intake to avoid milk fever, shortening of the dry period and reduced milking in early lactation[38,39].

Treatment of milk fever

- Oral Administration of calcium borogluconate is the best approach to reduce hypocalcemia (cows in stage one)
- Intravenous of calcium borogluconate should be treated immediately especially in stage two and three[39-41]

Bloat (tympany) in ruminants: Cattle produced gas from 30 to 50 L in hour because of microbial fermentation and as found at the top of the rumen. These gases are gotten ride through esophagus (160 to 225 cm per second). Bloat takes place as a result of prevention gases through eructation which cause pressure on the diaphragm and lungs, which cause difficult for breathing and animals will die. Bloating are two different types. Primary bloat (frothy): Frothy bloat is due to fixed foam in the rumen which inhibits eructation of gas[42]. Secondary bloat (free gas): Free-gas bloat takes place as a result of foreign body in esophagus. Moreover, there are other factor including physical problems or pathological problem, all of those prevent release of gases[43].

Several causes of bloat, genetic reasons, kind of feeding, high concentrate and low-quality hay, kind of ruminal fermentation and foreign objects in esophagus[44].

Diagnosis and clinical signs: There are several marked appear on aimals such as, open mouth breathing and distended left-sided abdomen unable to walk, the behind region with red colour and appear massing it, the obvious symptoms was

high temperature in that case[45] and discomfort, frequent urination, kicking of belly and collapse[44].

Treatment of bloat (tympany) in ruminant

The approach to treatment depends on the condition of bloat occurrence whether:

- **Free gas bloat (early case):** Animal can be treated with antifoaming agents like paraffin oil, oral and animal can be treated with stomach tube only
- **Frothy bloat (mild case):** This case could be treated with stomach tube contain antifoaming to release gases which inside the frothy thus, the gases can release from frothy because reducing frothy bloat with the tube only, so,difficult[43]. Recently, simethicone in cattle, sheep and goats were administered once at a time[42]
- **In severe cases of bloat:** Using a sharp knife, a quick incision of 10-20 cm through the skin was directly into the rumen, to release the rumen content. The trocar and cannula have been used for these cases. When the trocar is effective for reducing sever bloat, the antifoaming agent can be added through fistula to release gases from it[43]

Prevention and control of bloat (tympany): The best approaches for prevention bloat including: gradual access to pastures, Chemical agents were used to reduce bloat, Poloxalene is an anti-foaming agent which prevents pasture bloat for 12 h because breaks down the surface tension of bubbles in froth, thus releasing gas and enabling it to escape from the rumen and reticulum. Because Poloxalene prevents bloat for only a short period, it must be consumed (1-2 g/50 kg of body weight) daily. Poloxalene can be fed as a dressing on feed, in a grain mixture fed free-choice, in liquid supplements, or in molasses blocks[44,45].

CONCLUSION

It could be concluded that ruminal disorder is not a pathogenic problem but it is managerial problems in the transition period and it could be solve by managing transition cow to minimize metabolic disorder. Actually, that's because from 30 to 50% get sick in this period. Manage transition cows by plan including the following issues: identify behavior that related to metabolic disorder, determine which management practice is effective to prevent metabolic disorder, be careful with early diagnosis so we can save the animal's life. It is important to notice subclinical cases acidosis or ketosis through production because these cases without obvious symptoms, suddenly animal die within few hours and free feeding must be avoided. Also, future direction traditionally, herbal medicine has been used to treat and prevent various ruminant disorders. Recently, there has been an increase in the utilization of herbal medicine, reflecting trust in such therapeutic approaches. Moreover, herbal plants and essential oils have more compatibility with the animal body to reduce these sides effect.

REFERENCES

1. Green, L.E., V.J. Hedges, Y.H. Schukken, R.W. Blowey and A.J. Packington, 2002. The impact of clinical lameness on the milk yield of dairy cows. J. Dairy Sci., 85: 2250-2256.
2. Bach, A., I. Iglesias, M. Devant and N. Ràfols, 2006. Performance and feeding behavior of primiparous cows loose housed alone or together with multiparous cows. J. Dairy Sci., 89: 337-342.
3. Andersen, P.H., B. Bergelin and K.A. Christensen, 1994. Effect of feeding regimen on concentration of free endotoxin in ruminal fluid of cattle. J. Anim. Sci., 72: 487-491.
4. Gozho, G.N., J.C. Plaizier, D.O. Krause, A.D. Kennedy and K.M. Wittenberg, 2005. Subacute ruminal acidosis induces ruminal lipopolysaccharide endotoxin release and triggers an inflammatory response. J. Dairy Sci., 88: 1399-1403.
5. FAO, 2012. Impact of animal nutrition on animal welfare-Expert Consultation 26-30 September 2011-FAO Headquarters, Rome, Italy. Anim. Prod. Health Rep., pp: 10-12.
6. Wang, X., 2012. Correlation between compositions of the bacterial community concentration of volatile fatty acids in the rumen during the transition period ketosis in dairy cows. Appl. Environ. Micriol., 78: 2386-2392.
7. Hesam, A. Seifi and K. Samuel, 2018. Subclinical Hypocalcemia in Dairy Cows: Pathophysiology, Consequences and Monitoring. Iran. J. Vet. Sci. Tech., pp: 1-15.
8. Radostitis, O.M., C.C. Gay, D.C. Blood and K.W. Hinchliff, 2007. Veterinary Medicine. A Text Book of the Diseases of Cattle, Sheep, Goats and Horses. 10th Edn., WB Saunders Co.
9. Saha, S., L. Gallo, G. Bittante and S. Schiavon, 2019. A study on the effects of rumen acidity on rumination time and yield, composition and technological properties of milk from early lactating Holstein cows. Animals, 9: 66-75.
10. Aschenbach, J.R., G.B. Penner, F. Stumpff and G. Gabel, 2011. Ruminant nutrition symposium: Role of fermentation acid absorption in there gulation of ruminal pH. J. Anim. Sci., 89: 1091-1107.
11. Lettat, A., P. Mozieré, M. Silbeerberg, D. Morgavi and C. Berger, 2010. Experimental feed induction of ruminal lactic, propionic, or butyric acidosis in sheep. J. Anim. Sci., 88: 3041-3046.

12. Dijkstra, J., J. Ellis, E. Kebrab, A. Strathe and S. Lopez, 2012. Ruminal pH regulation and nutritional consequences of low pH. Anim. Feed Sci. Tech.,172: 22-23.
13. Hernandez, P.M., A.Z. Salem, M.M. Elghandour, M. Cipriano-Salazar, B. Cruz Lagunas and L.M. Camacho, 2014. Anthelmintic effects of *Salix babylonica* L. and *Leucaena leucocephala* Lam. extracts in growing lambs. Trop. Anim. Health Prod., 46: 173-178.
14. Laskoski, M., S. Muraro, S. Santana-Junior, B. Carvalho and H. Freitas, 2014. Sodium bicarbonate as prevention of metabolic acidosis in sheep submitted to experimental ruminal acidosis. Pesqui. Vet. Brasil, 34: 822-826.
15. Zhao, X., T. Zhang, M. Xu and J. Yao, 2011. Effects of physically effective fiber on chewing activity, ruminal fermentation and digestibilityin goats. J. Dairy Sci., 89: 501-509.
16. Krause, K. and G. Oetzel, 2006. Understanding and preventing subacute ruminal acidosis in dairy herds: A review. Anim. Feed. Sci. Tech., 126: 215-236.
17. Rustom, B., O. Aizahal, J. Cant, M. Fan and T. Duffield, 2006. Effects of rumen acid-load from feeds on ruminal pH, dry matter intake, fiber degradability and milk production in the lactating dairy cow. J. Anim. Sci., 84: 216.
18. Penner, G., M. Oba, G. Gäbel and J. Aschenbach, 2010. A single mild episode of subacute ruminal acidosis does not affect ruminal barrier function in short term. J. Dairy Sci., 93: 4838-4845.
19. Castillo-López, E., B. Wiese, S. Hendrick, J. Mckinnon and T. Mcallister, 2014. Incidence, prevalence, severity and risk factors for ruminal acidosis in feedlot steers during back grounding diet transition and finishing. J. Anim. Sci., 92: 3053-3063.
20. Maulfair, D., K. Mcintyre and A. Heinrichs, 2013. Subacute rumial acidosis and total mixed ration preference in lactating dairy cows. J. Dairy Sci., 96: 6610-6620.
21. Rochfort, S., A.J. Parker and F.R. Dunshea, 2008. Plant bioactives for ruminant health and productivity. Phytochemistry, 69: 299-322.
22. Castillo, C., J. Hernandez, V. Pereira and J.L. Benedito, 2012. Update about Nutritional Strategies in Feedlot for Preventing Ruminal acidosis. Jenkins, O.P. (Ed.), Nova Science Publishers, New York, NY, USA., Adv. Zool. Res., 4: 1-84.
23. Hashemi, S.R. and H. Davoodi, 2011. Herbal plants and their derivatives as growth and health promoters in animal nutrition. Vet. Res. Commun., 35: 169-180.
24. Gastaldello, A., A. Pires, I. Susin, C. Mendes, M. Quiroz, 2013. Limestone with different particle size and sodium bicarbonate tofeedlot lambs fed high grain diets with or without monensin. Small Rum. Res., 114: 80-85.
25. Cardozo, P., S. Calsamiglia, A. Ferret and C. Kamel, 2005. Screening forthe effects of natural plant extracts at different pH *in vitro* rumenmicrobial fermentation of a high concentrate diet for beef cattle. J. Anim. Sci., 83: 2572-2579.
26. Castillejos, L., S. Calsamiglia, J. Martin-Tereso and H. Ter-Wijlen, 2008. *In vitro* evaluation of effects of ten essential oils at three doses on ruminal fermentation of high concentrate feedlot-type diets. Anim. Feed Sci. Tech., 155: 259-270.
27. Cardozo, P., S. Calsamiglia, A. Ferret and C. Kamel, 2006. Effects of alfalfa extract, anise, capsicum and a mixture of cinnamaldehyde and eugenol on ruminal fermentation and protein degradation in beef heifers fed high-concentrate diet. J. Anim. Sci., 88: 2801-2808.
28. Foskolos, A., A. Siurana, M. Rodriguez-Prado, A. Ferret and D. Bravo, 2015. The effect of a garlic oil chemical compound, propyl-propanethiosulfate, on ruminal fermentation and fatty acid outflow in a dual-flow continuous culture system. J. Dairy Sci., 98: 5482-5491.
29. Emily, S. and C. Brent, 2017. Diagnosis and treatment of clinical rumen acidosis. Vet. Clin. Food Anim., 33: 1-12.
30. Getachew, T., 2019. Ketosis and it is Economic Importance in Dairy Cattle: A Review. J. Dairy Vet. Sci., 10: 555800.
31. McArt, J., D. Nydam, P.A. Ospina and G.R. Oetzel, 2011. A field trial on the effect of propylene glycol on milk yield and resolution of ketosis in fresh cows diagnosed with subclinical ketosis. J. Dairy Sci., 94: 6011-6020.
32. Zhang, Z., G. Liu, H. Wang and Z. Wang, 2012. Detection of subclinical ketosis in dairy cows. Pak. J. Vet., 32: 156-160.
33. Gordon, J., S. LeBlanc and T. Duffield, 2013. Ketosis treatment in lactating dairy cattle. Vet. Clin. N Am. Food. Anim. Pract., 29: 28-30.
34. Carrier, J., S. Stewart, J. Godden and P.F. Rapnicki, 2004. Evaluation and use of three cow side tests for detection of subclinical ketosis in early postpartum cows. J. Dairy Sci., 87: 3725-3735.
35. Gordon, J., S. LeBlanc, L. Neuder, T. Herdt and D. Kelton, 2012. Efficacy of combination but aphosphan cyanocobalamin product insulin for ketosis treatment. J. Dairy. Sci., 95: 177.
36. Wagner, S. and V. Schimek, 2010. Evaluation of the effect of bolus administration of 50% dextrose solution on measures of electrolyte energy balance in postpartum dairy cows. Anim. J. Vet. Res., 71: 1074-1080.
37. Madreseh-Ghahfarokhi, S., A. Dehghani-Samani and A. Dehghani-Samani, 2018. Ketosis (acetonaemia) in dairy cattle farms: Practical guide based on importance, diagnosis, prevention and treatments. J. Dairy Vet. Anim. Res., 7: 299-302.
38. Nese, K., 2018. Prevention of milk fever: A herd health approach to dairy cow nutrition. Arch. Anim. Husbandry Dairy Sci., pp: 1-3.
39. Wubishet, F., T. Dechassa, A. Nejash and M.A. Wahid, 2016. Milk fever and its economic consequences in dairy cows: A review. Global Vet., 16: 441-452.
40. Bernard, C., K. Erick, M. Borden, S. Shepherd and H. Gervais, 2017. A study of the incidence of milk fever in Jersey and Holstein cows at a dairy farm in Beatrice, Zimbabwe. J. South Afr. Vet. Assoc., pp: 1-6.

41. Erkihun, T. and B. Lingerih, 2015. An overview on milk fever in dairy cattle in and around West Shoa. World J. Biol. Medic. Sci., 2: 115-125.
42. Rahman, M.M., M.M.U. Bhuiyan, Md. TaohidulIslam and M. Shamsuddin, 2018. Efficacy of simethicone for treatment of bloat in ruminants. Asian J. Med. Biol. Res., 2: 635-638.
43. Tagesu, A., 2018. Study on the prevalence of bovine frothy bloat in and round Kebele Encha, TokkeKutaye District, Oromia Region. Approaches Poult. Dairy Vet. Sci., 2: 144-153.
44. Karin, L., 2020. How to treat and prevent bloat in cattle. BSc in Agriculture, Animal Science, University of Alberta, Canada.
45. Faez, F.J.A., A. Lawan, Z.S. Mohd, Y.O. Abdinasir, W.H. Abdul, N.A. Dayang and R. Noorashimah, 2013. Concurrent bloat and rectal prolapse in a cow. Int. J. Livestock Res., 4: 155-160.