THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
February, 2023; 3(02), 253-260

Popular Article

Ketosis in Dairy Animals-An Overview

Dr. J. Shashank*, Dr. N. Rajanna, Dr. G. Ganesh, Dr.J. Saikiran, Dr. A. Raju
Krishi Vigyan Kendra, P.V. Narsimha Rao Telangana Veterinary University, Mamnoor, Warangal-506 166.
https://doi.org/10.5281/zenodo.7679019

Abstract

Ketosis is a metabolic disease in dairy cattle recorded in late pregnancy or early lactation stage. This mini-review depicted the prevalence rate, treatment, economic losses and dietary management. It is observed by increase in ketone bodes specially β-hydroxybutyrate in blood. It is divided as primary and secondary ketosis. Primary ketosis causes due to insufficient glucose supply to cattle which cause negative energy balance whereas secondary ketosis occurs as a result of concurrent disease which may cause anorexia. Prevalence rate of ketosis is closely related to diet and farm management and also it is identified to be closely related to animal's genetics. Concentration of serum β-hydroxybutyrate between 1,200-1,400 mmol/L is usually threshold for definition of ketosis and clinical (wasting and nervous) and sub-clinical ketosis are two differentially aspects of ketosis in dairy cattle, which are defined according to presence or absence of clinical signs of ketosis in cattle. Prevention of ketosis based on body score evaluation, efficient diet, periodic blood, milk and/or urine screening for ketone bodies' detection in dairy cattle farms are the best methods to early detection of ketosis in animals which can be easily applicable in dairy cattle farms and improve the production rate and economic rate. Treatment of ketosis is depending on the condition of animals.

Keywords: β –hydroxybutyrate, Late pregnancy, Ketosis, Ketone bodies.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com

Published 26.2.2023

Introduction

Ketosis was recorded for the first time in 1920 to 1930 by Stinson and was termed as acetonemia or ketosis. Ketosis is a transition period metabolic disorder with the risk zone lasting from one week before calving and up to 30 days post-calving and marked by elevated levels of ketone bodies measured in blood or milk indicating that the metabolic processes in liver are overwhelmed leading to cell stress and liver damage, thus reducing liver function. It is related to formation of ketone bodies (*i.e.* acetone, acetoacetate, β-hydroxybutyrate) and is a measure of liver and capacity to convert circulating non-esterified fatty acids (NEFA) into glucose by gluconeogenesis. Due to lack of supplementation of pasture, in many areas' ketosis can be common where intensive farming was practiced during winter-spring (McArt *et al.,* 2011). On the basis of clinical signs, clinical and sub-clinical ketosis can be diagnosed. Clinical ketosis has visible clinical symptoms such as anorexia, licking, blindness, hard dry feces, rapid loss of body condition, reduced milk production and etc. whereas sub-clinical ketosis is an excess level of ketone bodies in the circulation without clinical signs (MCA *et al.,* 2011). Clinical symptoms and several laboratory tests to measure ketone bodies in blood, milk and urine to diagnose clinical ketosis (Cook, 2001). The incidence rate of ketosis is estimated to range from 2-15% for clinical ketosis (Duffield *et al.,* 2009) and from 26-60% for sub-clinical (Duffield *et al.,* 1998; McArt *et al.,* 2012 and Wagner *et al.,* 2010). Cows compensate for rapid fetal growth in the final weeks of gestation and onset of lactogenesis by mobilizing fat stores. All cows at this stage of lactation must rely, to some degree on body reserves to meet the demands of lactation, but prolonged negative energy balance is often associated with ketosis (Iwersen *et al.,* 2009). Due to negative energy balance and rapid lipolysis results in hyperketonemia lead to over production of NEFA and inadequate hepatic metabolism result into accumulation of prominent ketone bodies: BHBA, acetoacetate and acetone (Andersson, 1988; Drackley, 1999 and Ingvartsen, 2006).

Etiology

Etiology of Ketosis is varying with body condition of cow and caused by negative energy balance and characterized by relatively high concentrations of ketone bodies acetoacetate, Beta-hydroxybutyrate (BHBA) and acetone and a concurrent low concentration of glucose in blood. A

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 26.2.2023

concentration of serum BHBA greater than 1,200-1,400 mmol/L is a common standard used for the diagnosis of ketosis (Youssef *et al.*, 2010). Starvation is one of the common cause of ketosis due to a decreased feed intake (anorexia) and also lack of feeding to cow during transition period (Melendez *et al.*, 2004). In ketosis, there is impaired metabolism of carbohydrate and volatile fatty acids (Seifi *et al.*, 2011). Due to hypoglycemia, which results in increase demands for glucose and insufficient propionate production during early post-partum period in dairy cows (Dann *et al.*, 2005). Ketosis can be caused by biochemical and hormonal factors. The predisposing factors also play role in occurrence of the disease (Duffield *et al.*, 2009). Silage containing high content of butyric acid is also cause of ketosis. When cows consume the silage, which contains high content butyrate the cow cannot metabolize as fast as intake of butyrate and disturb the metabolism. Because of this butyrate builds up in the rumen and eventually diffuses across the rumen wall and enters the bloodstream. In liver butyrate is converted to BHBA resulting in ketosis in dairy cattle (Duffield *et al.*, 2009).

Diagnosis

The diagnosis of clinical and sub-clinical ketosis can be done with the help of different qualitative and quantitative tests. Clinical ketosis diagnosis is based on history and clinical observation which includes decreased feed intake, weight loss and decreased milk production. So diagnosis of ketosis has been greatly enhanced by availability of several laboratory-based rapid cow-side diagnostic tests which include Fourier transform infrared (FTIR) spectrometry, Gas liquid chromatography, Nuclear magnetic resonance (NMR) spectroscopy and gas chromatography-mass spectrometry (GC-MS) tests. These tests have been developed to measure BHBA, acetoacetate and acetone in serum or plasma. Quantitative laboratory-based determination of BHBA, acetoacetate and acetone require special laboratory equipment, ultraviolet spectrophotometer, biochemistry analyzer, centrifugation, blood sampling, freezing of plasma or serum samples, transport of frozen materials to laboratory (Oetzel, 2004). These laboratory-based tests can be achieved by measuring ketone bodies concentration in blood, urine and milk (Duffield, 2000). These tests are expensive and sometime inconvenient. Therefore, to relieve inconveniences, to reduce laboratory costs and to provide results immediately after sampling cow-side diagnostic tests have been proven advantageous (McArt *et al.*, 2011). Several authors have used a cut-off point of 1.2 mmol/L (1200 mmol/L) concentration of BHBA to discriminate between healthy cows and cows affected by SCK (Oetzel,

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 26.2.2023

2004). The Precision Xtra meter is a useful cow side ketone test for diagnosis of sub-clinical ketosis in post-partum dairy cows. The Precision Xtra ketone monitoring system is a simple and a direct electrochemical test, which gives excellent results for measuring whole blood BHBA in cows. The ketone test strip contains the enzyme β - hydroxybutyrate dehydrogenase, which oxidizes BHBA to acetoacetate. This reduces nicotinamide adenine dinucleotide (NAD+) to NADH. The NADH is then re-oxidized to NAD+ by an electron transfer mediator molecule. The electrical current generated by this conversion is measured by meter and is directly proportional to BHBA concentration. Cows with blood BHBA levels above 14.4mg/dl are considered positive for ketosis. Rothera's test detects acetone and aceto-acetate but not beta- hydroxybutyric acid and show result by changes in color of urine and milk samples showed a color reaction varied from no color (-), slightly purple (+), moderately purple (++), black-purple (+++) and dark- purple (++++) in cases of ketosis (Seifi *et al.*, 2011). Ratio of percentage of milk fat to milk protein monitor the prevalence of sub-clinical ketosis in herd. The ratio of higher than 1.5 indicates sub-clinical ketosis and ratio of lower than 1.1 indicates suspected rumen acidosis. Moreover, sub-clinical ketotic cows showed elevated level of folic acid and long-chain fatty acids in milk fat during the first nine weeks of Ketosis in dairy animals, lactation (Oetzel, 2004). Therefore, these tests are used to diagnose the ketosis in cattle.

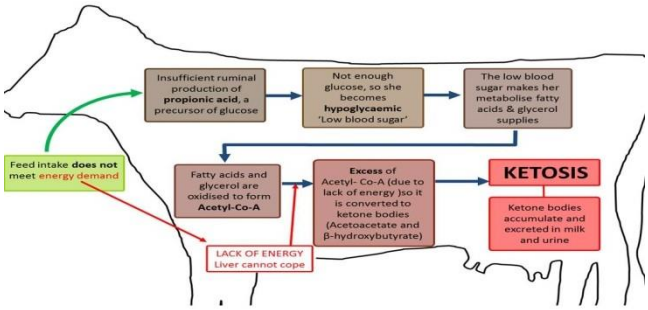

Fig.1 Pathogenesis of Ketosis

Differential Diagnosis

Sometime ketosis can be confused with listeriosis, lead poisoning and rabies. Those diseases associated with nervous signs including excessive licking, chewing and hyperesthesia, head pressing and apparent blindness (Bradford, 1990).

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 26.2.2023

Treatment

Cows may recover from ketosis without any treatment. But it takes a long time and cows may suffer from any other diseases which reduce milk production and weight loss. Therefore immediate treatment is necessary (Seifi *et al.*, 2011). Main cause of ketosis is insufficient feed intake so treatment should be started with increased feed intake and intravenous dextrose solutions, glucocorticoids and oral propylene glycol which establish the normal carbohydrate and fat metabolism. Propylene glycol is a routine treatment for ketosis. Propylene glycol is absorbed from the rumen as propylene glycol, some propylene glycol is metabolized to propionate in rumen, but most is absorbed intact and metabolized to glucose in liver. Propylene glycol increases serum glucose, decreases serum β-OH butyrate and NEFA concentrations but only if a functional liver as propylene glycol must be metabolized. Propylene glycol is beneficial only if rumen motility is present to aid mixing and absorption. Plasma concentrations of both glucose and insulin increase significantly about 48 hours after injection with dexamethasone (Jorritsma *et al.*, 2004). The intravenous injections of 500 ml of 50% glucose solution results in transient hyperglycemia (2 to 2.5 hours), increased insulin and decreased glucagon secretion and reduced plasma concentration of non-esterified fatty acids. Its effect has a marked improvement in most cows but relapses occur commonly unless repeated treatment is used (Walsh *et al.*, 2007). Glycerol (same dose rate as propylene glycol) and sodium propionate (uncertain dose rate) also reported to be of use but are both considered inferior to propylene glycol. Sodium propionate may have palatability problems. Calcium propionate has been examined, but the evidence is not convincing that it is superior to propylene glycol, even though it also has calcium. Not very soluble and large volumes need to be administered.

Prevention and Control

To control the ketosis, feed intake should be increased and minimize negative energy balance. Maintain carbohydrate and fat metabolism, the aims of the transition period are to allow cows to develop a strong immune system, maintain normal concentrations of calcium in blood, prevent calving related diseases and develop a rumen adapted to the post-partum diet (Melendez *et al.*, 2004). During early lactation, cows should not be too fat at calving because this depresses their feed intakes. A body condition score of 2.5-3.0 on a 1-5 scale is optimal and anything higher is considered too fat

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 26.2.2023

and at greater risk of ketosis (Melendez and Risco, 2005). Ketosis prevention and control is the most important method in dairy industry for increasing milk yield products and dairy cows free from any disease as this affects the immunity of cows. One of the guiding principles of sustainable livestock production is to feed high levels of roughage in the diet to promote good rumen digestion. For dairy cattle feed at least 60% fresh or conserved roughage, should have high quality during early lactation to meet the energy and protein requirements. This is especially important in the winter diet based on home-gown conserved forages and it may be difficult to supply sufficient energy if cows are high yielding (Bystrom *et al.,* 2002). Concentrates feed during lactation should be introduced in small amounts, approximately two weeks before calving, to allow adjustments of the rumen micro flora and dietary changes during early lactation should be given gradually to reduce disease. Efforts should be made to ease the transition from gestation to lactation by offering highly palatable forage at calving, providing suitable accommodation and assistance where necessary (Moorby *et al.,* 2002).

1. As much as possible implement good management practice (adequate bedding space, limit overcrowding and ensure proper feed delivery and adequate water access) in dairy cattle.
2. 2. Cows should neither have been starved nor be over fat at calving.
3. The cow should be preventing from concurrent diseases.
4. Should give great attention for immediate diagnosis and proper treatment of affected cow to restore the milk yield and feed intake.

References

Andersson, L (1988). Sub-clinical ketosis in dairy cows *Vet. Clin. North Am. Food Anim. Pract*. **4**. 233-251.

Bradford, PS. (1990). Ketosis in ruminants. *Textbook of Large Animal Internal Medicine* 4" Edn., Louis, Toronto USA. 1364-1365.

Bystrom, S., Jonsson, S. and Martinsson, K. (2002). Organic versus conventional dairy farming studies from the Ojebyn Project. In: *Proc of the UK Organic Research 2002 Conference.* Organic Centre Wales, Institute of Rural Studies, University of Wales Aberystwyth. 179-184.

Carrier, J., Stewart, S., Godden, S., Fetrow, J. and Rapnicki, P. (2004). Evaluation and use of three cowside tests for detection of sub-clinical ketosis in early post-partum cows. *J Dairy Sci.* **87:** 3725-35.

Cook, N., Ward, W. and Dobson, H. (2001). Concentrations of ketones in milk in early lactation and reproductive performance of dairy cows. *Vet Rec.* **148**: 769-72.

Dann, H. Drackley, J., Douglas,G.N., Janovick, J. and Lither. N. (2005) Physiological and pathological adaptation in dairy cows that may increase susceptibility to peri- parturient

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com

Published 26.2.2023

diseases and disorders. *Italy J. Anim. Sci* **4**. 323-344

Drackley, J.K. (1999). Biology of dairy cows during the transition period - The final frontier. *J. Dairy Sci*. 82 2259-73.

Duffield, T., Lissemore, K., McBride, B. and Leslie, K. (2009). Impact of hyperketonemia in early lactation dairy cows on health production. *J. Dairy Sci* **92**: 571-580.

Duffield, T. F (2000). Sub-clinical ketosis in lactating dairy cattle. *Vet. Clin, North Am. Food Anim. Pract.* **16**: 231-53

Duffield, T.F., Sandals, D., Leslie, K.E., Lissemore, K.. McBride, B.W. Lumsden, J.H., Dick, P. and Bagg. R. (1998). Efficacy of monensin for prevention of subclinical ketosis in lactating dairy cows. *J. Dairy Sci.* **81**: 2866-73

Duffield, T.F., Lissemore, K.D., McBride, BW. and Leslie, K.E. (2009). Impact of hyperketonemia in early lactation dairy cows on health and production. *J. Dairy Sci.* **92**: 571-80.

Gordon, J., LeBlanc, S., Neuder, L., Herdt, T. and Kelton, D. (2012). Efficacy of combination butaphosphan cyanocobalamin product insulin for ketosis treatment. *J. Dairy Sci*. **95**: 177.

Grummer, R.R. (1993). Etiology of lipid-related metabolic disorders in peri-parturient dairy cows. *J. Dairy Sci.* **76**:3882-3896

Ingvartsen, K.L. (2006). Feeding and management related diseases in the transition cow - Physiological adaptations around calving and strategies to reduce feeding-related diseases. *Anim. Feed Sci. Technol*. **126**: 175-183

Iwersen, M., Falkenberg. U., Voigtsberger, R. Forderung. D. and Heuwieser, W. (2009). Evaluation of an electronic cowside test to detect sub-clinical ketosis in dairy cows. *J. Dairy Sci.* **92**: 2618-2624.

Jorritsma, R., Thanasak, J., Houweling, M., Noordhuizen, J. and Muller, K. (2004). Effects of a single dose of dexamethasone-21-isonicotinate on the metabolism of heifers in early lactation. *Vet. Rec*. **155**: 521-523.

McArt, J., Nydam, D., Ospina, P.A. and Oetzel, G.R. (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.

McArt, J.A.A., Nydam, D.V. and Oetzel, G.R. (2012). Epidemiology of sub-clinical ketosis in early lactation dairy cattle. *J. Dairy Sci.* **95**: 5056-66.

Melendez, P., Donovan, G., Risco, C. and Goff, J. (2004). Plasma mineral energy metabolite concentrations in dairy cows fed an anionic prepartum diet that did or did not have retained fetal membranes after parturition. *Am. J. Vet. Res.* **65**: 1071-1076.

Moorby, J., Dewhurst, R., Evans, R. and Fishert, W. (2002). Effects of level of concentrate feeding during the second gestation of Holstein-Friesian dairy cows. 2. Nitrogen balance plasma metabolites. *J. Dairy Sci* **85**: 178-89.

Oetzel, G. (2004). Monitoring testing dairy herds for metabolic disease. *Vet. Clin. North Am. Food Anim. Pract.* **20**: 651-674.

Ospina, P.A., Nydam, D.V., Stokol, T. and Overton, T.R. (2010). Evaluation of nonesterified fatty acids and Beta- hydroxybutyrate in transition dairy cattle in the northeastern United States-Critical thresholds for prediction of clinical disease. *J. Dairy Sci.* **93**: 546-554.

Seifi, H., LeBlanc, S., Leslie, K. and Duffield, T. (2011). Metabolic predictors of post-partum disease culling risk in dairy cattle. *Vet J.* **188**: 216-220.

Wagner, S. and Schimek, D. (2010). Evaluation of the effect of bolusi administration of 50% dextrose solution on measures of electrolyte energy balance in post-partum dairy cows. *Am. J. Vet. Res*. **71**: 1074-1080.

Walsh, R., Walton, J., Kelton, D., LeBlanc, S. and Leslie, K. (2007). The effect of sub-clinical ketosis in early lactation on reproductive performance of post-partum dairy cows. J. Dairy Sci. **90**: 2788-2796.

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.

Youssef, M., Khodery, E., Deeb, E. and Amaiem. (2010). Ketosis-Clinical findings the associated oxidative stress level. *Trop. Anim. Health. Prod*. 42: 1771-1777.