

Indexed, Abstracted and Cited: ISRA Journal Impact Factor, International Impact Factor Services (IIFS), Directory of Research Journals Indexing (DRJI), International Institute of Organized Research and Scientific Indexing Services, Cosmos Science Foundation (South-East Asia), Einstein Institute for Scientific Information {EISI}, Directory of Open Access Scholarly Resources,

[citefactor.org](http://citefactor.org) **journals indexing**

**Directory Indexing of International Research Journals**

## World Journal of Biology and Medical Sciences

Published by Society for Advancement of Science®

ISSN 2349-0063 (Online/Electronic)

Volume 2, Issue- 4, 115-125, October to December, 2015



WJBMS 2/04/51/2015

All rights reserved

A Double Blind Peer Reviewed Journal

[www.sasjournals.com](http://www.sasjournals.com)

[wjbmedsc@gmail.com](mailto:wjbmedsc@gmail.com)/[wjbms.lko@gmail.com](mailto:wjbms.lko@gmail.com)

REVIEW ARTICLE

Received: 19/08/2015

Revised: 07/09/2015

Accepted: 08/09/2015

## An Overview on Milk Fever in Dairy Cattle in and Around West Shoa

Erkihun Tadesse and \*Lingerih Belete

Addis Ababa University, College of Veterinary Medicine and Agriculture, P.O. BOX: 34,  
Debre Zeit Ethiopia

\*Hawassa University, Faculty of Veterinary Medicine and Agriculture, Hawassa, Ethiopia

### ABSTRACT

*Milk fever is a condition of older, third to sixth lactation, high producing dairy cows. It is associated with parturition, usually within 72 hours of giving birth. Because of the high volume of milk produced during this time and subsequent demand for calcium, these cows often develop hypocalcaemia, or abnormally low levels of calcium in the blood. Since calcium is required for the release of acetylcholine at the neuromuscular junction, affected animals will begin to experience muscle weakness. As this hypocalcaemia worsens, the cow will become too weak to stand and will eventually become comatose over a matter of hours. Parturient paresis is treated intravenously with calcium borogluconate. Affected cows have an excellent prognosis if treated early and properly. However, the worse the symptoms, the worse the prognosis tends to be. Any preventive measure must be aimed at eliminating the precipitous fall in blood calcium at parturition. For assessing economic losses caused by milk fever cost of medicines, cost of additional labour utilized, loss due to reduction in milk output, cost of animals dead and culled should be considered. Generally, the dairy profitability is directly related to the level of milk production.*

**Keywords:** Calcium Borogluconate, Dairy Cattle, Milk Fever and Parturient Paresis.

## INTRODUCTION

Among domestic farm animals, the metabolic diseases achieve their greatest importance in dairy cows. The high producing dairy cows always verge on abnormality because the breeding and feeding of dairy cattle for high milk yield is etiologically related to the diseases of metabolism so common in these animals. In dairy cows, the incidence of metabolic diseases is highest in the period commencing at calving and extending until the peak of lactation is reached, and this susceptibility appears to be related to the extremely high turnover of fluids, salts and soluble organic materials during the early part of lactation (Corohn, 1998).

Inadequate blood calcium concentrations can cause a cow to lose the ability to rise to her feet as calcium is necessary for nerve and muscle function. These results in the metabolic disease known as milk fever, although it is more properly termed hypocalcaemia or parturient paresis, as an elevated body temperature is not typically observed. It is a common metabolic disorder in dairy cattle that generally affects older, high producing cows. The lowered blood calcium in milk fever is due to the failure of the blood calcium regulatory mechanism to mobilize calcium from the tissue reserves rapidly enough to equal the withdrawal of calcium from the blood into the udder secretions. The parathyroid hormone is the primary regulator of blood calcium, its failure in milk fever is caused by either parathyroid inadequacy which resulting in the lack of sufficient hormone secretion, or by the presence of some metabolic condition in the tissues at parturition that renders the parathyroid hormone temporarily inactive (Radostits *et al.*, 2007).

There are risk factors for the precipitation of milk fever in the dairy cows to occur including age, breed, body condition and dietary factors. Milk fever is a disease of

considerable importance for dairy cow welfare and economy. Although treatment with intravenous infusion of calcium salt solutions cure most clinical cases of hypocalcaemia (Curtis *et al.*, 1994). It has been proposed that a specific control program is relevant when the incidence of milk fever increases to above 10% among high risk cows that is cows entering third or later lactations. So prevention of milk fever is economically important to the dairy farmer because of reduced production loss, death loss, and veterinary costs associated with clinical cases of milk fever (Bradford, 1996).

Therefore, the objectives of this seminar paper are:

- To give an overview on milk fever in dairy cattle and
- To indicate appropriate prevention and control methods of milk fever in dairy cattle.

## GENERAL ACCOUNTS ON MILK FEVER IN DAIRY COWS

Milk fever is a metabolic disturbance usually of dairy cows, characterized by reduced blood calcium levels. It is most common in the first few days of lactation, usually within 72 hours, when the demand of calcium for milk production exceeds the body's ability to mobilize calcium reserves. Fever is a misnomer, as body temperature during the disease is usually below normal. Low blood calcium level interferes with muscle function throughout the body, causing general weakness, depression and death. It is more common in older dairy cows which have reduced ability to mobilize calcium from bone and in high milk producing breeds (Huntjens, 1995).

### Etiology

Deficiency of calcium ion in the tissue fluids and fall in serum calcium level at calving due to the onset of lactation is the major biochemical defect in parturient paresis. Serum calcium level falls in all

adult cows at calving (Radostits *et al.*, 2007). The delay in the operation of calcium homeostatic mechanisms is vital in causing milk fever. Calcium homeostasis is affected by three factors and variations in one or more of them are instrumental in causing the disease in any individual. These are excessive loss of calcium in the colostrums beyond the capacity of absorption from the intestines and mobilization from the bones to replace, impairment of absorption of calcium from the intestine at parturition and insufficiency of mobilization of calcium from storage in the skeleton, which could arise because of parathyroid insufficiency since the gland is relatively quiescent due to the decreased calcium and phosphorus metabolism of the dry period (Mulligan *et al.*, 2006).

#### **Epidemiology**

Parturient paresis is a disease of high producing dairy cattle. It is estimated that 3% to 8% of cows are affected by this disease with some herds having prevalence as high as 25% to 30%.

Parturient paresis occurs before calving, but most cases occur within the first 48 hours following calving (Goff, 2004).

The risk factors for hypocalcaemia are grouped into intrinsic risk factors, which are associated within the animal itself, and extrinsic risk factors, which are outside of the animal's body (Peterson and Beede, 2002).

**Age:** The hypocalcaemia at calving is age related and most marked in cows from third to seventh parturition; it is infrequent at the first parturition. This is because while some degree of hypocalcaemia occurs during the first few days of lactation, they are able to adapt rapidly to the high demands of calcium for lactation. With increasing age, this adaptation process is decreased and results in moderate to severe hypocalcaemia in most adult cows. The

adaptation mechanism is directly related to the efficiency of intestinal absorption of calcium, which decreases with increasing age. Most cases occur in animals older than five years of age. This is as a result of increasing milk production with age and decreasing efficiency of dietary calcium absorption and bone resorption (Bradford, 1996).

**Breed:** There are differences in susceptibility of milk fever among breeds. There is a genetic predisposition of cows to milk fever and this is well recognized in certain breeds of high producing Jersey and other breeds. This is associated with higher milk production per unit of body weight, reduction of intestinal vitamin D<sub>3</sub> receptors as Jersey's age, and a higher than normal production of parathyroid hormone related proteins by the mammary gland, which increases calcium transport from bone to milk (Colls, 1990).

**Body condition score:** A high BCS increases the risk of milk fever. Dairy cows that are over conditioned at calving are up to four times more likely to develop milk fever (Ostergaard *et al.*, 2003). This is due to dairy cows with higher BCS at calving have a higher calcium output in milk, making them more prone to milk fever and over conditioned cows take small amount of calcium this is caused by reduced appetite in the critical period around calving which predisposes them to the development of hypocalcaemia (Harris *et al.*, 1999).

**Dietary factors:** Diets providing dry cows a high daily intake of calcium are associated with an increased incidence of parturient paresis. At this level the maintenance requirement of calcium can be met predominantly by passive absorption since active absorption of dietary calcium and bone resorption are then suppressed. Cows in this condition are not able to quickly replace plasma

calcium lost in milk and become severely hypocalcaemic (Boda and Cole, 2003).

Diets high in phosphorus at the onset of lactation also increase the incidence of milk fever and the severity of hypocalcaemia. High dietary level of phosphorus decreases the production of 1, 25 dihydroxy vitamin D<sub>3</sub> and thus reduces the intestinal calcium absorption mechanisms (Horst, 1996). Pre-partum diets high in cations like sodium and potassium are associated with an increased incidence of milk fever while diets of high in anion, especially chlorides and sulfides are associated with decreased incidence of the disease. The addition of anions to the diet of dairy cows prior to parturition effectively reduced the incidence of milk fever by inducing a metabolic acidosis, which facilitates bone resorption of calcium (Bradford, 1996).

#### **Pathophysiology**

During the dry period, calcium demand is relatively low. Hence, intestinal absorption and bone resorption of calcium is relatively inactive during this time. The onset of lactation at the time of parturition results in sudden loss of calcium through milk. When the calcium homeostatic mechanism is unable to meet the demand of calcium for milk production, hypocalcaemia or low blood calcium level occurs (Ramberg *et al.*, 1994).

This fall in blood calcium level stimulates the calcium homeostatic mechanism to improve intestinal calcium absorption and increase bone resorption. Approximately 24 hours of elevated 1, 25 dihydroxy vitamin D<sub>3</sub> is required to improve intestinal transport of calcium and an increased rate of bone resorption requires 48 hours of PTH stimulation. When these compensatory mechanisms are prolonged, clinical hypocalcaemia or milk fever develops. Consequently, most cows

with clinical hypocalcaemia have higher levels of PTH and 1, 25 dihydroxy vitamin D<sub>3</sub> (Harris, 2002). Hypocalcaemia affects muscular contraction mainly in three ways. Firstly, calcium has a membrane stabilizing effect on the peripheral nerves. Hyperesthesia and mild tetany seen in early stages of milk fever are due to lack of nerve cell membrane stabilization. Secondly, calcium is required for the release of acetylcholine at the neuromuscular junction. The inability to release acetylcholine, due to hypocalcaemia causes paralysis by blocking the transmission of nerve impulse to the muscle fibers. Thirdly, calcium is directly required by muscle cells for contraction. Paralysis of various muscle types results in the clinical signs of parturient paresis (Iggo, 1994). There is decreased contractility of cardiac muscle and lowered stroke volume cause reduction in arterial blood. Then this reduced peripheral perfusion resulted in hypothermia and depression of consciousness. In addition, there is GIT stasis and bloat resulted from smooth muscle atony. Hypophosphatemia is seen in cows suffering from milk fever. This is as the result of phosphorus loss in milk, inadequate feed intake around the time of parturition, and increase renal and salivary excretion of phosphate caused by a high circulating level of PTH. Hyperglycemia associated with milk fever is caused by increased glucocorticoid release and gluconeogenesis caused by stress. There is an occurrence of hypermagnesemia in cows suffering from parturient paresis due to the effect of PTH by increasing tubular resorption by the kidney (Bartlet *et al.*, 1990).

#### **Clinical sign**

Based on the degree of hypocalcaemia and time of occurrence the clinical sign of milk fever are grouped in to three stages (Aiello *et al.*, 1998).

**Stage I:** Milk fever often goes unobserved because of its short duration less than one hour. This are seen when animals are in standing position. Signs observed during this stage include excitability, nervousness, hypersensitivity, weakness, and hyperesthesia. There is smooth muscle paralysis that results in an inability to swallow, consequently in appetance and low thirst, ruminal stasis with the passing of small amounts of dry faeces, and the suspension of urination (Bradford, 1996).

**Stage II (Sternal recumbency):** The clinical signs of stage II milk fever can last from 1 to 12 hours. This is frequently seen with lateral kink or S-shape neck curvature in which the cow tends to lie with her head tucked into her flank. Her temperature is subnormal, her muzzle dry, coldness of skin and extremities. The heart rate will be rapid exceeding 100 beats per minute, gastrointestinal atony predisposes to constipation and mild bloating. She exhibits in coordination when walking. (Aiello *et al.*, 1998).



**Figure 1. Cow in sternal recumbency.**  
Source: Kimura *et al.*, 2006

**Stage III (Lateral recumbency):** It is characterized by inability to stand and a progressive loss of consciousness leading to coma. There is a marked fall in temperature and heart sounds become

nearly inaudible and the heart rate increases to 120 beats per minute or more. Cows will not survive for more than a few hours without treatment in this stage (Radostits *et al.*, 2007).



**Figure 2. Cow in lateral recumbency.**  
Source: Kimura *et al.*, 2006

## DIAGNOSIS AND TREATMENT

### Diagnosis

Diagnosis is based on the cow's history, clinical signs and response to intravenous calcium borogluconate solution. The occurrence of paresis and depression of consciousness in cows that have recently given birth to young are diagnostic signs for the disease. The diagnosis is confirmed by laboratory examination of the blood and rapid characteristic response to treatment with calcium borogluconate. The most notable changes occurring in the blood are a decrease in blood calcium and blood phosphorus levels and an increase in blood magnesium levels (Radostits *et al.*, 2007).

Cows with serum calcium lower than 7.5 mg/dl are as considered as hypocalcaemic. Animals with serum calcium level of 5.5 to 7.5 mg/dl show sign of stage I hypocalcaemia. Stage II hypocalcaemia seen with calcium levels of 3.5 to 6.5 mg/dl and stage III seen when calcium concentration falls to as low as 2 mg/dl. Prolonged recumbency results in ischemic muscle necrosis and increases in the serum muscle enzymes CPK and AST. The value CPK normally ranges between 105 to 409 IU/L, a value greater than 1000 IU/L indicates severe muscle damage from being down, and AST levels over 500 IU/L indicates severe muscle damage (Bradford, 1996).

For an accurate diagnosis of milk fever, differential diagnosis is important to determine milk fever from other disorders with similar clinical sign like hypoglycemia. Typical signs and indications that differentiate milk fever from hypoglycemia includes in hypocalcaemia there is rapid progression of the disease with death after 6 to 12 hours, but in hypoglycemia there is slow progression of the disease with death 5 to 7 days. Elevation of the chin (star gazing posture) with slow progression to recumbency over

2 to 3 days after onset of initial signs is seen in hypoglycemia but during hypocalcaemia rapid recumbency over 3 to 4 hours with sternal recumbency. In response to treatment, in hypoglycemia there is no response to hypocalcaemia treatment dose rates and usually poor and slow doses of glucose or energy.

During hypocalcaemia response is rapid and good recovery is seen after injection of treatment doses of commercial calcium doses

(<http://www2.dpi.qld.gov.au/sheep/10182-html>).

### Treatment

Treatment of the disease usually consists of intravenous or subcutaneous injection of calcium borogluconate at dose rate of 400 to 800 ml of a 20% solution. Early intravenous calcium borogluconate is the treatment of choice for severely affected patients. The solution must be given slowly because rapid calcium infusion may result in cardiac arrest. Concurrent use of subcutaneous calcium borogluconate may prevent recurrence, by slow release of biologically available calcium from the tissues into the bloodstream (Anderson *et al.*, 1998).

Patients with mild signs are appropriately treated with subcutaneous or oral calcium products. The response to properly administered calcium therapy is quite characteristic. Cows generally rise within one hour. The cow's symptoms will appear to reverse themselves as they had previously progressed. The laterally recumbent cow will sit up to sternal position, and then it will often begin to have tremors over its body. As all bodily functions affected by hypocalcaemia begin to reverse, the affected animal may urinate, and then begin the wobbly effort to rise. Repeated treatment may be necessary within 12 hours if the cow is still unable to rise. Approximately 75% of cows

with milk fever respond to only one treatment and recover (Blowey, 1995).

The prognosis is excellent if cows are treated early and properly. As the symptoms worsen so does the prognosis. Cows down for more than 48 hours may develop muscle inflammation and never be able to stand. Relapse is relatively common unless longer acting supplements are added to the treatment plan. Older cows and cows displaying signs prior to calving have the highest risk for relapse (Oetzel, 1988).

### **ECONOMIC SIGNIFICANCE**

Milk fever in dairy production reduces the efficiency with which inputs are converted into outputs (milk yield and fertility) and hence result in decreased overall productivity. There are costs directly associated with the disease including the cost of veterinary treatments and the herdsman's time spent dealing with the affected animals. The indirect cost of the milk fever is due to increased risk to associated health problems, increased risk of calving problems, and the possible risk of fatality (Kossaibati and Esslemont, 1997). Milk fever predisposes cows to many potential health problems (Brosberry and Dabson, 1989).

**Milk fever and fertility:** Milk fever results in reduced fertility in dairy cows due to its effect on uterine muscle function, slower uterine involution and reduced blood flow to the ovaries. There are also indirect effects of milk fever on fertility, which is mediated through dystocia, endometritis and retained placenta (Jonsson and Daniel, 1997).

**Milk fever and mastitis:** Cows that have suffered from clinical milk fever are 8 times more likely to develop mastitis than normal cows. This phenomenon is mainly due to a reduction in smooth muscle function at the teat sphincter and hence an easy routine for infection after milking and an exacerbated suppression of

immunity in milk fever cows when compared with normal cows. Cortisol is an important component of the suppressed immunity experienced by peri parturient dairy cattle since milk fever cause an increase in the normal cortisol response at parturition (Goff, 2004).

**Milk fever and GIT function:** There is a reduction in the motility of rumen and abomasum in clinically hypocalcaemic cows. This reduction in ruminal and abomasal motility will likely cause a reduction in feed intake (Jorgensen *et al.*, 2001).

**Milk fever, dystocia and uterine prolapse:** Loss of uterine muscle tone due to hypocalcemia in cows suffering from milk fever is a major cause of uterine prolapse (Buxton *et al.*, 1997). Cows with milk fever are developing dystocia 6 times more than that of normal cows. This is because of a reduced ability of smooth and skeletal muscle contraction causes for cow's long period in labour, which predisposes to dystocia (Correa *et al.*, 1993).

### **PREVENTION AND CONTROL**

A specific control program is relevant when the incidence of milk fever increases to above 10% among high-risk cows that is cows entering third or later lactations. Several milk fever control principles and factors are applied in commercial dairy farms today. These includes oral drenching around calving with a supplement of easily absorbed calcium, the feeding of acidifying rations by anionic salt supplementation during the last weeks of pregnancy, feeding low calcium rations during the last weeks of pregnancy and management practices such as body condition control and shortening the dry period (Radostits *et al.*, 2007).

#### **Oral calcium drenching around calving**

Preparations containing easily absorbed calcium salt such as calcium chloride, providing 40 to 50 g of calcium per dose can be used. Most documented

preventive programs involve administration of 3 to 4 doses distributed evenly during the period from 12 to 24 hours before calving to 24 hours after calving (Agger *et al.*, 1997).

Oral calcium treatment presumably increases luminal calcium concentration favouring passive transport of calcium into the extra cellular fluids. The capacity of the passive transport of calcium is in principle unlimited and independent of stimulation by 1, 25 dihydroxy vitamin D<sub>3</sub>. Increasing the amount and the number of drenching with calcium compounds providing free calcium ions therefore quickly increases the amount of calcium ions absorbed into the bloodstream (Goff and Horst, 1993).

#### **Feeding of acidifying rations (dietary cation-anion difference)**

It is now widely accepted that the homeostatic mechanisms that result in milk fever prevention work more efficiently when DCAB is negative. The most common strategy employed to achieve this negative DCAB is the addition of anionic salts to the diet of pre calving cattle (Goff, 2004). This is most efficiently achieved by the ingestion of rations having a surplus of acidifying anions. The effect is mediated via an enhancement of the stimulatory effects of PTH. Bone resorption may be involved and an increase in the apparent calcium absorption from the digestive tract (Jorgenson *et al.*, 2001).

#### **Low calcium intake and low calcium/phosphorus in late pregnancy**

This principle is based on preventing calcium homeostatic mechanisms from becoming quiescent during the dry period (Oetzel, 1996). The calcium demand of the dry cow is very limited in the last two months of pregnancy. When calcium intake exceeds the requirements, the calcium demand can be met almost entirely by passive diffusion from the

intestinal tract, rendering the calcium homeostatic mechanisms relatively inactive (Boxton *et al.*, 1997).

By feeding low calcium diets in the dry period, the calcium homeostatic mechanisms are activated before calving, and the cow is thus capable of absorbing calcium more efficiently from the intestinal tract as well as drawing calcium from the bone around the time of calving (Leclerc and Block, 1999).

A high in dietary phosphorus suppressed vitamin D hydroxylation in the kidney, thereby indirectly reducing calcium absorption. The reduced intestinal calcium absorption elicited an increase in the PTH level, which in turn aided in the prevention of milk fever by increasing the amount of calcium in the blood by tubular absorption of calcium from the intestine and resorption from the bone. In addition, low dietary phosphorus is helpful in preventing milk fever, when the level of dietary calcium is high. Since the low dietary phosphorus results in an increase formation of 1, 25 dihydroxy vitamin D<sub>3</sub>, which in turn increases the efficiency of intestinal absorption of calcium and phosphorus (Allen and Davies, 1995).

#### **Management practices**

##### ***Body condition score management***

Over conditioned cows at calving have a higher incidence of milk fever and other diseases post partum. Achieving the correct BCS at calving and drying off is critical for the prevention of milk fever (Ostergaard *et al.*, 2003).

##### ***Shortening of the dry period***

Cows with long non-lactating periods are predisposed to becoming excessively fat, since cows do not regulate intake according to their physiological requirements. Besides obesity, a long dry period may result in a more quiescent calcium homeostasis (Davicco *et al.*, 1992).

### **Magnesium supplementation**

Ensuring adequate magnesium supplementation is vital for the prevention of milk fever. Magnesium plays a very important role in calcium metabolism; it is a key intermediate in the resorption of calcium from bone by parathyroid hormone. In a recent review, increasing magnesium supplementation has the greatest influence among dietary strategies for the prevention of milk fever. Therefore, dietary magnesium concentration for pregnant dairy cattle should be in the region of 0.4% of dry matter (Lean *et al.*, 2006).

### **CONCLUSION AND RECOMMENDATIONS**

Milk fever or parturient paresis is a common metabolic disturbance in dairy cows resulting from hypocalcaemia that occurs in older, third to sixth lactation, high producing dairy cows that are near calving or have recently calved. It is mainly characterized by progressive muscle weakness and depression that progresses into coma if not treated promptly. Calving causes a high volume of milk production and with it, a high demand for calcium from the cow's body. If the body is unable to respond quickly to this demand, the cow develops hypocalcaemia. Hypocalcaemic cows will begin trembling and will no longer be able to stand. Subsequently the cow becomes recumbent, first in the sternal position, and then laterally. Parturient paresis is favorable to early treatment with intravenous calcium supplementation. Economically, it reduces milk yield and fertility. There are also losses due to cost of treatment, herdsman's time spent for dealing the diseased animal and increase the risk of associated health problems. As with most illnesses, prevention is the key. Dietary calcium levels must be low in the weeks leading up to calving. Management practices like body condition score

management and shortening the dry period are also critical for the prevention of the disease.

Based on the above conclusion the following recommendations are forwarded:

- Avoid over fattening by either reducing the energy concentration of the ration or restricting the intake during the pre partum period.
- Implementation of a well monitored feeding strategy for dairy cattle especially during the pre partum period.
- Education of dairy farmers to make frequent observations of cows prone to milk fever from 48 hours before to 72 hours after parturition for evidence of milk fever.
- At calving, the cow should receive an oral dose of a calcium salt in a gel, as set out later, followed by a diet with high calcium content.
- Further study should be conducted on the epidemiology of the disease and its economic impact in dairy farms.

### **ACKNOWLEDGMENTS**

I would like to thank my advisor, Dr. Dereje Bantigegn, in assisting me to prepare this paper.

My special thanks also go to my best friends especially Aysheshim M. (editor) and Kassahun B., who are helping me while I am preparing this paper.

### **REFERENCES**

**Agger, N., Katholm, J. and Lomborg, K. (1997).** A pharmacokinetic study of plasma calcium concentration in dairy cows following four oral administrations at 12 hour intervals around calving of a calcium chloride paste formulation. *The Bovine Proceedings*, **30**: 72-75.

- Aiello, S., Mays, A., Amstutz, H. and Anderson, D. (1998).** Parturient paresis. The Merck Veterinary Manual, 9<sup>th</sup> edition, Merck Company Incorporated press, USA, Pp. 806-808.
- Allen, W. M. and Davies, D. C. (1995).** Milk fever, hypomagnesaemia and the downer cow syndrome. *British Veterinary Journal*, **137**: 435-441.
- Blowey, R. W. (1995).** Metabolic disorders. Veterinary Book of Dairy Farms, 2<sup>nd</sup> edition, Farming press, UK, Pp. 137-152.
- Boda, J. M. and Cole, H. H. (2003).** Calcium metabolism with special references to parturient paresis in dairy cattle. *Journal of Dairy Science*, **39**: 1027.
- Bradford, P. (1996).** Disorders of calcium metabolism. Large Animal Internal Medicine, 2<sup>nd</sup> edition, Donlading press, USA, Pp. 1464-1470.
- Buxton, D., Horst, R., Goff, J. and Reinhardt, T. (1997).** Strategies for preventing milk fever in dairy cattle. *Journal of Dairy Science*, **80**: 1269-1280.
- Colls, G. E. (1990).** Parturient paresis. Veterinary Clinical Pathology, 3<sup>rd</sup> edition, Saunder Company press, USA, Pp. 305.
- Correa, M., Erb, H. and Scarlett, J. (1993).** Path analysis for seven postpartum disorders in Holstein cows. *Journal of Dairy Science*, **76**: 1305-1312.
- Curtis, C. R., Sniffen, C. J. and Smith, R. D. (1994).** Epidemiology of parturient paresis. Predisposing factors with emphasis on dry cow feeding and management. *Journal of Dairy Science*, **67**: 817-825.
- Davico, M., Remond, B., Jabet, W. and Bartel, J. (1992).** Plasma calcium concentrations in cows around parturition. The influence of a regular versus a very short dry period. *Reproductive Nutrition Development*, **32**: 313-319.
- Corohn, Y. T. (1998).** Epidemiology of metabolic disorder in the parturient dairy cows. *Journal of Dairy Science*, **9**: 2557.
- Goff, J. P. (2004):** Macro mineral disorders of the transition cow. *Veterinary Clinics of North America*, **20**: 471-494.
- Goff, J. P. and Horst, R. L. (1993).** Oral administration of calcium salts for treatment of hypocalcaemia in cattle. *Journal of Dairy Science*, **76**: 101-108.
- Harris, D. J. (2002).** Factors predisposing to parturient paresis. *Australian Veterinary Journal*, **57**: 357-361.
- Harris, D., Rukkwamsuk, T. and Wensing, T. (1999).** Relationship between over feeding and over conditioning in the dry period and the problems of high producing dairy cows during the post parturient period. *Veterinary Quarterly*, **21**: 71-77.
- Horst, R. L. (1996).** Regulation of calcium and Phosphorus homeostasis in dairy cows. *Journal of Dairy Science*, **69**: 604-605.
- Huntjens, M. F. (1995).** Metabolic problems related to nutrition. *Dairy Reference Manual*, **6**: 494.
- Iggo, A. (1994).** Activity of peripheral nerves and junctional regions. Physiology of Domestic Animals, 10<sup>th</sup> edition, California University press, USA, Pp. 612-615.
- Jonsson, N. N. and Daniel, R. C. (1997).** Effect of hypocalcaemia in blood flow to the ovaries of cow. *Journal of the American Veterinary Association*, **44**: 281-287.
- Jorgensen, R., Houe, H., Larsen, T., Sorensen, J., Agger, J. and Blom, J. (2001).** Milk fever and subclinical hypocalcaemia an evaluation of parameters on incidence, diagnosis, risk factors and biological effects as input for a decision support system for disease control. *Scandivian Veterinary Journal*, **42**: 1-29.

- Kaneko, J. J. (1989).** Calcium regulatory hormone and abnormal mineral metabolism. *Clinical Biochemistry of Domestic Animals*, 4<sup>th</sup> edition, Academic press, USA, Pp. 699-752.
- Kimura, K., Reinhardt, T. and Goff, J. (2006).** Parturition and hypocalcaemia blunts calcium signals in immune cells of dairy cattle. *Journal of Dairy Science*, **89**: 2588–2595.
- Kossaibati, M. A. and Esslemont, R. J. (1997).** The cost of production diseases in dairy herds in England. *The British Veterinary Journal*, **154**: 41-50.
- Lean, I., Degaris, P., Mcneil, D. and Block, E. (2006).** Hypocalcaemia in dairy cows. *Journal of Dairy Science*, **89**: 669-684.
- Leclerc, H. and Block, E. (1999).** Effects of reducing dietary cation-anion balance for pre partum dairy cows with specific reference to hypocalcaemic parturient paresis. *Canadian Journal of Animal Science*, **69**: 411-423.
- Mulligan, F., Grady, L., Rice, D. and Doherty, M. (2006).** A herd health approach to dairy cow nutrition and production diseases of the transition cow. *Animal Reproduction Science*, **96**: 331-353.
- Oetzel, G. R. (1988).** Parturient paresis and hypocalcaemia in ruminant livestock. *Veterinary Clinics of North America*, **4**: 331.
- Oetzel, G. R. (1996).** Effect of calcium chloride gel treatment in dairy cows on incidence of peri-parturient diseases. *Journal of American Veterinary Medical Association*, **209**: 958–961.
- Ostergaard, S., Sorensen, J. and Houe, H. (2003).** A stochastic model simulating milk fever in a dairy herd. *Preventive Veterinary Medicine*, **58**: 125-143.
- Peterson, A. B. and Beede, D. K. (2002).** Peri-parturient responses of holster in cows to varying pre-partum dietary phosphorus. *Journal of Dairy Science*, **85**: 187
- Radostits, M., Gay, C., Blood, C. and Kenneth, W. (2007).** Metabolic disturbance. *Veterinary Medicine*, 10<sup>th</sup> edition, Baillire Tindal publisher, London, Pp. 1627-1642.
- Ramberg, C. F., Johnson, E. D. and Fargo, R. D. (1994).** Calcium homeostasis in cows with special reference to parturient hypocalcaemia. *American Journal of Physiology*, **246**: 698.
- Web site consulted**  
<http://www2.dpi.qld.gov.au/sheep/10182.html>. Accessed in April, 2013.

---

Corresponding author: Erkihun Tadesse, Addis Ababa University, College of Veterinary Medicine and Agriculture, P.O. BOX: 34, Debre Zeit Ethiopia.  
 Email: [erkihunt@yahoo.com](mailto:erkihunt@yahoo.com)