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Nutritional Disorders in Dairy Cattle During Mid Lactation Period in Khartoum State

إلاضطرابات الغذائية في الابقار الحلوب في المرحلة الوسيطة من إلادرار في ولاية الخرطوم

By

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Dedication

To my dear father, mother, brothers and sisters

My engaged

Omyma mahmoud

My uncle

With love and respect

Ahmed

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First of all, unlimited thanks to the most gracious and merciful "Allah" who grant me everything to carry out this study. I would like to express my sincere gratitude and appreciation and recognition to my Supervisor Prof. Siham Elias Suliman Mohammed Department Of Medicine and Animal Surgery, College Of Veterinary Medicine, Sudan University Of Science and Technology (SUST) for her guidance, advice, valuable discussion, encouragement, support, reading draft manuscript and my deep gratitude's are also extended to Prof. Mohammed Abdel Salam Abdalla Department Of Preventive Medicine and Public Health, College Of Veterinary Medicine, Sudan University Of Science and Technology (SUST) for his great efforts and valuable advice of this study Co - Supervisor. I would like to express my sincere thanks to Dr. Rania Ibrahim Mustafa Ministry Of Agriculture Animal Wealth and Irrigation, Khartoum state for her continuous help, I would like to thanks Dr. Ahmed Sabeil, Khartoum University for his great help of this study.

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Abstract

The purpose of this study was to evaluate some biochemical parameters in dairy cows during mid-lactation period and to identify the types of nutrients and the factors that leading to nutritional disorders in the period from November to December 2017. The study was done in Jabal Awlia and Omdurman localities – Khartoum State. A total of 50 cows in mid lactation period were selected randomly from the two localities [25 cows from each locality]. Blood samples [50 samples] were taken from jugular vein for separation of serum to evaluate of total protein, albumin, glucose, calcium, inorganic phosphorus, sodium, and potassium. A questionnaire was conducted to obtain information about dairy men [50 men], diseases associated with dairy farms and feeding system. The results revealed that there was no significant changes biochemical parameters in these animals [p>0.05]. But the level of total protein was statistically significant [p<0.05]. Dairy men interviewed their age between 21 to over 40 years old. Most of them were uneducated [40%] and only 8% of them were working in dairy farm for over 19 years. The occurrence of nutritional disorders was common in two localities [48%] and most of these disorders occurred in dry season [48%]. Most of the diseases occurred in cross breed over 4 years old [26%] with morbidity rate 32.7% and mortality rate 40%. Some of risk factors in the farms leading to disturbance of nutrition that the animals were not grazing [36%] or with limited movement [56%] and using of semi – intensive system [48%]. In conclusion, supplementation of diet constitutes energy feed, minerals, vitamins, concentrate mixture improve the animal health and prevent occurring of the diseases in lactation period.

مستخلص البحث

الهدف من هذة الدراسة تقيم بعض إلاضطرابات الغذائية في الابقار الحلوب في المرحلة الوسيطة من إلادر ارفي ولاية الخرطوم و التعرف على انواع الاغذية والعوامل المؤثرة التي تؤدي الى إلاضطرابات الغذائية في الفترة من نوفمبر - ديسمبر 2017م. وتم ذلك في ولاية الخرطوم في كل من محليتي جبل اولياء وامدرمان. تم فحص عدد (50 عينة) دم من الوريد الوداجي بعد فصل السيرم منها من الابقار الحلوب في المرحلة الوسيطة من إلادرار عشوائيا في كل من المحليتين (25عينة من كل محلية) لتقيم البروتين الكلي، الالبيومين، الجلكوز، الكالسيوم، الفسفور، الصوديوم و البوتاسيوم. تم تصميم إستبيان لجمع المعلومات حوالي (50 شخص) من مزارع الالبان عن الامراض المرتبطة بها والنظام الغذائي. النتائج تشير لم يثبت اي فرق معنوي عندمستوى المعنو 0.05 p> (p> 0.05 (. ولكن البروتين الكلى قد اظهر فرق معنوي واضح (p < 0.05) . بعد المقابلة الشخصية في المزارع التي بها اشخاص اعمارهم ما بين 21 – اكثر من 40 سنة معظمهم غير متعلمين بنسبة (40%) وفقط الذين يعملون منهم في مزارع الالبان اعمارهم اكثر من 19 سنة. الاضطرابات الغذائية تحدث في كل من المحليتين بنسبة (48%) ومعظمها تحدث في الفصل الجاف ايضا بنسبة (48%). معظم الامراض تحدث في الابقار الهجين التي اعمارها اكثر من 4 سنين بنسبة (26%) بمعدل إصابة (32.7%) ومعدل النفوق (40%). بعض العوامل المؤثرة في توزيع الغذاء بالنسبة للنظام المغلق تمثل (36%) بينما (56%) للحيوانات محدودة الحركة و (48 %) للحيوانات زات النظام شبه المغلق. إلاضافات الغذائية تشمل اغذية الطاقة، المعادن، الفيتامينات، و الاعلاف المخلوطة تحسن من صحة الحيوان وتمنع حدوث الامراض في الفترة الوسيطة من إلادرار.

Introduction

Deficiency will occur in producing animals as results of dietary deficiency, abnormal absorption and utilization of feeding and presence of diseases downer cow, [Radostits et al., 2007]. A common nutritional deficiency occurred due to insufficiency of quantity and quality of feeding [Oetzel and Berger, 1986]. Also protein and energy deficiency are occurred concurrently in livestock with incomplete starvation. Decreasing of protein in the diet leading to hypoproteinemia of both young and mature animals. But ruminants sometimes not need dietary essential amino acids because rurminal flora synthesizes the necessary amino acids [Oetzel and Berger, 1985]. The prevention protein – energy malnutrition requirements in animals is depended on the age, stage of pregnancy and production, the environmental temperature and the cost of the feeding [Radostits et al., 2007]. Losses of sodium during lactation period result from milk, rapid growing young animal fed on lowsodium, cereal based diet, hot environment with heavy intensive physical work and low-sodium grazing pasture [Akitken, 1976]. At first weeks of lactation dairy cows are suffered from negative energy balance due to energy expansion associated with milk production and limited feed intake and the result high mobilization of limit from body fat reserves and hypoglycemia (Veenhuizen et al.,1991; Drackley, 1999; Oetzel, 2004). For decreasing of metabolic damage during production cause by negative balance, feeding glycogenic diet containing fat – enrich supplement (Gabor et al., 2016). Most important metabolic diseases associated with nutrition in lactation period in dairy cattle are parturient paresis, downer cow syndrome, acute hypokalemia in cattle, ketosis, fatty liver in cattle, and post parturient hemoglobinuria in cattle [Radostits et al., 2007]. Parturient paresis occurs at or near calving because of large calcium demand when the onset of milk produces. Cows are unable to meet the demand of calcium due to ration imbalance, vitamin D influence or parathyroid gland activity. All these factors influence regulation of the metabolism during the dry period [Smith et al., 2007]. Failure of regulation of these metabolites may lead to downer cow syndrome after 48hours of calving. But there are other causes of this disease including toxic mastitis, metritis, exhaustion from calving, calving paralysis, hip joint luxation and pelvic fracture. Also the presence of hypocalcemia concurrently with hypomagnesaemia, hypophosphatemia and hypokalemia in individual downer cow is expected (Clark et al., 1984). This disease can be treated medically with physiotherapy to correct the etiological factors (Kumari and Kaswan, 2015). For prevention of milk fever that by addition of sodium and potassium to reduce metabolic alkalosis and induced mild metabolic acidosis (Goff et al.,1997). Calcium concentration stimulates parathyroid hormones before calving and this hormones its function to activate bone osteoclasts for reabsorption of calcium and renal tubules to reabsorb urinary calcium and production of vitamin D prior to calving. Oral calcium at calving reduces the incidence of milk fever (Bacic et al., 2007). While ketosis or acetonemia occurs most frequently nearly lactation and may be associated with other problems, such as retained placenta, mastitis, metritis, fat cow syndrome and displacement of abomasum. This disease is associated with lack of energy during which liver converts fatty acids into ketone bodies [Burim, 1988]. But fat cow syndrome is due to excess energy [concentrate, corn silage, some hays] fed during the dry period may cause obese to the animals near calving time. These animals may become diseased by milk fever, ketosis, displaced abomasums, retained placenta or metritis [Smith et at., 2007]. Deficiency of potassium associated with dietary potassium intake and this can lead to

hypokalemia, especially in conjunction with other etiological agents of decreasing this element (Brobst, 1986; Carlson, 1989). Also the treatment of ketosis by glycocorticoids decrease plasma concentration of potassium and this effect may promote hypokalemia. The treatment of this condition by oral dosing of potassium chloride with addition of magnesium for in appetent lactating dairy cattle (Constable et al., 2013). But post parturient hemoglobinurea result from dietary phosphorus deficiency feeding cerciferous plants and also may be related to hypophosphatemia. This disease can be treated by phosphorus supplementation for a week with supportive therapy giving significant improvement in clinical and laboratory parameters (Wakayo et al., 2013). Fatty liver is known as accumulation of fat, mainly triacylgorol in the liver. Decreasing of feeding during the period of parturition resulting of more severe negative energy balance during early lactation and this leading to increase lipolysis of adipose tissue (Kirwvski and Sladojevic, 2017). Also fatty acids may enhance the metabolic process in transition cows and adaptation of calcium metabolism to lactation help by using nutritional programmes to decrease the cation – anion difference in the diet [Overton and Waldron, 2004]. Dairy cattle need a dietary source of minerals [calcium, phosphorus, magnesium, manganese, copper, cobalt, zinc and selenium]. In addition, vitamin such as vitamin A, D and in dairy animals concentrates are widely used, and also B -vitamins are added to milk replacers [Fraser and Mays, 1986].

Objectives:

- 1. To evaluate some biochemical parameters in dairy cows during midlactation period
- 2. To identify the type of feeding and risk factors associated with dairy farms

Chapter One

Literature Review

1.1. Etiological agents of diseases associated with nutritional disorders of cattle:

Milk fever is a metabolic diseases occurring shortly after parturition in dairy cattle and associated with hypocalcaemia, loss of consciousness and paralysis, acute to per acute, a febrile, flaccid paralysis of mature dairy cows and uterine prolapsed. The calcium is characterized by weakness, recumbency; depression of levels ionized in tissue fluid at a basic biochemical defect, however ultimately shock and death. A transient period of hypocalcaemia occurs at the onset of lactation caused by an imbalance between calcium output in the colostrums and influx of calcium to extracellular pool from intestine and bone. The diseases may be seen in cows of any age but is most common in high producing dairy cows over 5 years old. Incidence is higher in the jersey breed (Risco et al., 1984; Burim, 1988; Radostits et al., 2000; David, 2007; Barrington, 2011).

Ketosis is multifactorial disorders of energy metabolism that causes of acetonemia, characterized by partial anorexia and depression. Negative energy results in hypoglycemia and ketonemia, the accumulation in blood of acetoacetate, p-hydroxybutyrate and their decarboxylation products acetone and isopropanol. The diseases associated with lack of energy during which the liver converts fatty acids into ketone bodies, in which something goes wrong with the normal body processes and cow become sick. Reported that, very distinct problem for dairy cow is disease of ketosis, which occurrence related to an increased demand of glucose by the animals (Burim, 1988; Anderson et al., 1970; Radostits et al., 2000; Radostits et al., 2007).

Downer cow syndrome is a common sequel to milk fever in which the cow was sternal recumbency for several hours before being treated with calcium, all cattle which that develop pressure induced damage to muscle and nerves of the pelvic limbs specially when lying on a hard surface, the dystocia results of oversized calf which due to extensive edema of pelvic and valva, where complications milk fever, muscle necrosis, injuries and nerve paralysis that can arise because of delayed or insufficient calcium replacement, which additionally secondary metabolic disorder involving phosphorus, magnesium and potassium deficits have been suggested as risk factors, but without direct evidence of their involvement (Radostits et al., 2007; Laurent and Alexander, 2007; Roger and David, 2011; Joachim, 2012).

Fatty liver have greater adipose stores and mobilize more percentage of triacylglycerol (TAG), which lead to greater plasma nonesterified fatty acid (NEFA) concentration, because adipose tissue from cows with fatty liver is less and more responsive to lipogenic substances. The excess energy (concentrates, corn silage, some hays) feed during the dry period may cause obese cow near the calving time, which these "too fat" cows are more susceptible to number of other metabolic problems like milk fever and ketosis. The infection, trauma, and also parturition induce tumor necrosis factor (TNF) and secretion, which mediates inflammatory response that use great amount of glucose, aminoacid, and lipids. The disease is an exaggeration with a common occurrence in high producing dairy cows which are in a state of negative energy balance in early lactation (Holtenius, 1991; Bobe et al., 2004; Smith, 2007; Gross et al., 2013).

Acute hypokalemia in cattle may occurs commonly in lactating dairy cows, secondary to anorexia, diarrhea, mastitis, retained placenta, hepatic lipidosis, upper gastrointestinal obstruction, right side displacement, torsion and

impaction of the abomasum. Reduced potassium intake subsequent to metabolic alkalosis and hyperglycemia. The excessive use of corticosteroid with mineralocorticoid activity in cows with mastitis may lead to hypokalemia, naturally occurring dietary deficiency of potassium is thought to be rare (Robert et al., 1992 – 1997; Radostits et al., 2007; Constable et al., 2013).

Postparturient hemoglobinuria in north american, diet low in phosphorus or unsupplemented with phosphorus are usually associated with the diseases in adult dairy cattle during their third to sixth lactation, the disease also a sporadic characterized by intravascular hemolysis, hemoglobinuria and erythrocyte destruction lead to anemia. The hemolytic saponins from alfalfa interact with a low serum phosphorus concentration to produce post parturient hemoglobinuria or feeding of plant toxin (Brassica Spp, sugar beets or green forage) has been associated with the disease but many case occur unassociated with such diets and their roles as a cause is uncertain. The current hypothesis is that ingested hemolytic agents, some of them identified, for example in rape, some of them not, cause erythrocyte lysis in some circumstances. In New Zealand copper and selenium nutrition deficiency have been suggested as possible etiology of post parturient hemoglobinuria (Mac Willims et al., 1982; Wakayo et al., 2005; Radostits et al., 2007).

1.2. Diagnosis of diseases associated with nutritional disorders of cattle:

1.2.1. Clinical manifestations:

Milk fever is usually occurs within 72 hours of parturition. The disease contributes to dystocia, uterine prolapse, retained fetal membrane, mastitis, metritis and abomasum displacement. There are here stage of parturient paresis. Stage one, animals are ambulatory but show signs of hypersensitivity and excitability. Cows may be mild ataxic, have fine tremor over flanks and triceps, and display ear twitching and head bobbing. Cows may appear restless, shuffling their rear feet and bellowing. If the calcium therapy is not instituted, cows will likely progress to the second, more severe stage. Stage two the affected cows are unable to stand but can maintain sternal recumbence. Cows become dull anorectic and have a dry muzzle, subnormal body temperature, and cold extremities. Auscultation reveals tachycardia and decreased intensity of heat sounds. Peripheral pulses are weak. Smooth muscle paralysis leads to gastrointestinal stasis, which can manifest as bloat, failure to defecate, and loss of anal sphincter tone. An inability to urinate may manifest by a distended bladder on rectal examination. Cows often tuck their heads into their flanks, or if the head extended, an S - shaped curve to neck may be noted. Stage three cows lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbencey, then complete muscle flaccidity with unresponsive to stimuli, and can suffer severe boat. As cardiac output worsens, heat rate can approach 120 bpm, and peripheral pulses may be uncountable but cows in stage three may survive only a few hours by given calcium. The symptoms of include unsteady gait, cows lying down with head displaced to one side or into flank, depression, inappetence, pupils dilated, dry muzzle, cold ears, inability to rise, groaning, slight muscle spasms, coma and, if the animal is not treated it can be died (Amy and Dave, 2010; Barrington, 2011).

Nicky (2007) reported that common signs of ketosis in dairy cattle are characterized by two major form, the wasting form and nervous form. The wasting form is much more common, and the main sign is initially there may be a gradual decline in appetite over two to five days. Often the appetite is lost in an unusual manner and the cow may eat grass and hay but will not eat grain or silage. The appetite may appear depraved, with cows eating any object, including dirt and stones. The temperature, pulse and respiration rate of the cow remain fairly normal as the animals losses weight. The coat described as having "woody" appearance, presumably due to the loss of fat reserves under the skin. The ketones produced by the cow in this disease have a characteristic sweet "sickly" smell, which may be detected on the cows breath and less commonly in milk samples. The nervous form of ketosis is less common. Affected cows can show a range of signs including apparent blindness, aimless wandering and strange movements of tongue leading to incessant licking of the skin. Affected cows may also walk in circles and bellow loudly for no apparent reason. These kinds of behaviour can last for one or two hours, with the signs starting more suddenly than the wasting form of the disease. The first symptoms of ketosis is a loss of appetite first for grain and then silage, followed by a lack of feeding. The most common one is a dull and listless condition and unsteadiness in the rear legs. Also there are times when the affected animals may be quite nervous and easily excited, then followed by a drop in milk production. The breath may have a noticeable "sweet" odor due to presence of acetone. Ketosis seems to occurs in older animals, and some animal have a tendency to repeat the disease. Ketosis is seldom a direct cause of death. Clinical findings of ketosis of cattle in addition to wasting with decrease in appetite, fall in body condition, excessive loss of weight, constipation, mucous – covered feces and reduced milk production, some have short periods of bizarre neurological and behavioral abnormality and response to treatment is good. subclinical ketosis is detected by tests for ketones, usually in milk or urine. Subclinical ketosis in cows that are in negative energy balance in early pregnancy will have ketonuria without showing clinical signs, but will have diminished productivity including depression of milk yield and a reduction in fertility (Anderson and Ewalt, 1970; Little dike et al., 1981; Nicky, 2007; Smith, 2007; Amy and Dave, 2010).

Downer cows does not show signs of systemic illness or depression is able to eat and drink, and remains in sternal recumbency for no apparent reasons a non-alert downer cow appears systemically sick and depressed. The skin on the carpal joints were bruised and the joint were swollen which developed superficial wounds. The essential findings in the syndrome were that all cow had an initial clinical episode suggestive of milk fever but showed an unsatisfactory clinical response to calcium borogluconate therapy. After a day or two all cows become laterally recumbency, some exhibited expiratory moaning and all developed mucoid faeces which in many cases contained spots of blood, the cows that suffering either from hypocalcaemia or complication of other diseases such as mastitis and metritis. The animals are usually respirations unaffected and the defecation, sternal recumbency for 6, 9 or 12 hours with the right pelvic limb position under the body and urination are normal but proteinuria is common and if marked many indicate extensive muscle damage and some affected cows may make no effort to stand, and tachycardia is occur in some cow specially immediately following the administration of calcium intravenous and sudden death has occurred, others animals will make frequent attempts to stand but unable to fully are extend their pelvic limbs and lift their hind quarters from the ground $20-30\,\mathrm{cm}$, although the appetite is reduced, the animal are usually bright and alert and which the abnormal position of the legs may be also due to dislocation of one or both hip joints or association with traumatic injuries surrounding the hip joints with or without rupture of the ligaments. The death may occur in $48-72\,\mathrm{hours}$ and usually associated with myocarditis (Asma and Fetaih , 1994 ; Radostits et al., 2007; Mwaura and Kiarie , 2009; Joachim 2012; Peter , 2017).

In dairy cattle, fat cow syndrome occurs usually within the first few days following parturition and commonly precipitated by any condition which interferes with the animals appetite temporarily such as parturient hypocalcaemia, left sided displacement of the abomasum, indigestion, retained fetal membranes, and dystocia, affected cows are usually excessively fat with body condition scores of 5/5 or higher. Excessive quantities of subcutaneous fat are palpable over the flanks, the shoulder areas and around the tail head. The affected cow usually does not respond to treatment for some of these diseases and becomes anorexic. The temperature, heart rate, and respiration within normal ranges. Metabolic consequences of triglyceride accumulation in the liver reduced gluconeogenesis, ureagenesis, hormone clearance, and hormone responsiveness. Fatty liver is likely to develop concurrently with other disease after calving. Rumen contractions are weak or absent and the feces are usually scant. Periods of prolonged recumbency are common and affected cows may have difficulty in standing when they are coaxed to stand. Affected cows will not eat and gradually become weaker, totally recumbent and die in 7 - 10 days, some cattle exhibit nervous signs consisting of a staring gaze, holding the head and neck. Terminally there is coma and tachycardia, clinical finding are much less severe and most will recover within several days if they continue to eat even small amounts of hay. In dairy cattle there is a relationship between the occurrence of subclinical fatty liver within the first few weeks after parturition and inferior reproductive performance due to a delay in the onset of normal estrus cycles and a reduction in the conception rate which results in an increase in average days between calving and conception. The fat cow syndrome may be also associated with an increased incidence of parturient paresis and responsive treatment for ketosis in early lactation period (Katoh, 2002; Radostits et al., 2007; Walter, 2011).

Affected cows of hypokalemia are recumbent profoundly weak, appeared flaccid and lay in sternal or lateral recumbency, the are unable to support the weight of their heads off the ground and commonly hold them in their flanks. profound weakness of the lateral cervical muscles may occur and anorexia is common. Unappetizing also observed, altered renal function or iatrogenic alteration of normal electrolyte homeostasis (Robert et al., 1992 – 1997; Radostits et al., 2007).

of affected **Post** parturient hemoglobinuria cows observed were hemoglobinuria, inappetence, decreased milk production, and weakness develop suddenly and there is a severe depression, although in some less acute case the cow continues to eat and milk normally for 24 hours after discoloration of the urine is evident. Dehydration develops quickly and anemia, mucous membranes are pallid and become icteric, as the heart rate increases (80 - 130 beats/min) are much augmented, breathing becomes rapid and shallow, moderate temperature rise 40 C° often occurs, the feces are usually dry and firm. Dyspnea may be obvious and tachycardia is common, jaundice may be apparent in the late stages, pica also may be observed in the

other animals in the group. The course of the acute disease extends from 3-5 days; the cow becomes weak and staggery which finally recumbent and death occur within a few days (Mac Willims et al., 1982; Radostits et al., 2007).

1.2.2. Clinical pathology:

There have been many reports of diagnosis of milk fever using total serum calcium level in cows, reducing the number of absorbable dietary cations or increasing the number of absorbable dietary anions greatly diminish the incidence of hypocalcaemia and milk fever in dairy cow. The total serum calcium levels are reduced to below 2.0 mmol/l and sometimes to a slow as 0.5 mmol/l, the reduction is usually but no always proportional to the severity of the clinical blood levels of ionizing calcium are a better indicator of calcium status but their estimation has been too difficult until recently. The normal levels of ionized calcium in serum are, Slight which hypocalcaemia the range between 1.05 - 0.80 mmol/l, moderate 0.79 - 0.50 mmol/l and severe hypocalcaemia less than 0.30 mmol/l. Total serum calcium levels are reduced below normal in all cows at calving whether they have milk fever or not. The measurement of the total serum calcium concentration which is comprised of the sum of ionizing calcium and the calcium bound protein. Ionized calcium is important for immediate metabolic functions however, the analysis of ionized calcium in the field is not practical due to the unavailability of equipment and the high expense involved. Diagnostic confirmation of milk fever by response to treatment with the calcium borogluconate, Serum calcium level vary only within narrow limits and are not sensitive indicators of input – output balance. However, abnormally low levels in late pregnancy indicate a dangerous situation (Radostits

2000 ; Houe et al., 2001 ; Kojouri , 2003 ; Oetzel , 2004 ; Radostits et al., 2007) .

The ketosis is characteristic by blood glucose levels are reduced from the normal 50 mg/dl to 20 - 40 mg/dl approximately. The dairy cattle affected of ketosis when the glucose level below 1.94. One of major changes in blood components is a decrease in glucose which presumably due to the large amount of glucose removed by the mammary gland to make lactose, coupled with insufficient feed intake to replenish the glucose supply; there is evidence that the mammary gland produces acetoacetate under ketotic conditions but the origin is not clear. Ketosis secondary to other diseases is usually accompanied by blood glucose levels above 40 mg/dl and often above the normal. Most commonly plasma or serum B-hydroxybutyrate (BHBA) measured in (SI) units is used for analysis of ketonemia, BHBA is the predominant circulating ketone body. The clinical diagnosis of ketosis is not effective and in one study of 22 urine cows diagnosed by routine urine testing at 5-12 days postpartum was considerable more efficient than diagnosis by the herdsman. Cowside tests have the advantage of being inexpensive, giving immediate results, and they can be used as frequently as necessary. A minor source of error is that the concentration of ketone bodies in these fluids will depend not only by the ketone level of the blood but also on the amount of urine excreted or on the milk yield. Milk is less variable, easier to collect and may give fewer false negative with subclinical ketosis. Milk and urine ketone levels have been traditionally detected by the reaction of acetone and acetoacetate with sodium nitroprusside and can be interpreted in a semi quantitative manner based on the intensity of the reaction. Several products are available commercially as test powders or strips are commonly accompanied by a color chart that allows a classification in grades such as

negative, trace, small, moderate, large, based on the intensity of the color of the reaction. The nitroprusside urine strip test or BHBA milk strip test are best test for screening individual cows for ketosis in herds with average prevalence but that the nitroprusside powder test would have limited application (Kelly, 1964; Oetzel, 2004; Radostits et al., 2007).

Diagnosis of downer cow syndrome very important to obtain the case history of animal including age of animals, duration of recumbency, any previous clinical the abnormalities before the recumbent stage and after all other known causes of recumbency have been eliminated is a cow which had milk fever and failed to stand with 24 hours following two successive course of treatment. Clinical examination of downer cow can be very difficult and challenging depending on the environmental circumstances. In downer cow syndrome recorded the calcium, magnesium and glucose levels of the blood are with the normal range and the results of hematological examinations are usually consistent with those found in normal, cows which have recently calved, the normal range for bovine serum calcium 2 - 2.6 mmol/l, phosphorus $1.3 - 2.5 \, \text{mmol/l}$, potassium $3.9 - 5.8 \, \text{mmol/l}$ and magnesium $0.63-1.15\ \text{mmol/l}$. The Creatine phosphokinase (CPK) and Aspartate transferase (AST) levels are usually markedly elevated by 18 – 24 hours after the onset of recumbency and continue to elevate within next the few days. In experimental induced recumbency in cows, the CPK levels remained within normal limits for the first 6 hours, whoever by 12 hours there was a marked increase to mean value of 12000 U/L rising to 40000 U/L by 24 hours there may be moderate ketonuria. A marked proteinuria is usually evident by 18 -24 hours after the onset of recumbency may persist for several days or be absent within a few days. In severe cases, the urine may be brown and turbid because of severe myoglobinuria. Low arterial blood pressures and abnormal

electrocardiograms (ECGS) have been observed in some animals. Elevation of serum urea, muscle enzymes, and laboratory evidence of inflammation are considered the best prognostic indicators of an unfavorable recovery rate was lower in cows with a total protein fibrinogen ratio less than 10:1, and evidence of neutropenia or left shift. Cows with a serum urea level above 25 mmol/l and serum creatinine levels above 130 mmol/l had a poor prognosis, Blood test can be very useful in assessing the prognosis (Melody, 1984; Poulton and Steinfort, 2001; Radostits et al., 2007; Peter, 2017).

The serum biochemical changes associated with fatty liver syndrome in cows have been described based on blood and liver samples taken from cows during abdominal surgery, transcutaneous liver biopsy or at the abattoir immediately after slaughter. The serum biochemical abnormalities will depend on the severity of the fatty liver, serum biochemical evidence of liver diseases as determined by the presence of test value of two fold or greater than the upper limit of the reference range for at least two of the four serum tests: Gamma glutamyl transferase (GGT), Aspartate aminotransferase (AST), and Sorbitol dehydrgenase (SDH) activities and Bile concentrations. Although cattle with severe fatty liver had significantly higher serum bilirubin concentration and AST and SDH than cattle with less severe fatty liver, the specificity of abnormally high SDH or bilirubin concentration and for severe fatty liver was only 8 %, which abnormally high serum AST was 83 % sensitive and 62 % specific for severe fatty liver. Serum glucose and total carbondioxide concentrations were significantly lower in cattle with severe fatty liver than in those with mild or moderate fatty liver, low serum glucose or total carbondioxide concentrations were rare in cattle without severe fatty liver. Liver biopsy can be used to determine the severity of the fatty liver and the concentration of triglyceride and is the most reliable method of accurately

estimating the degree of fatty infiltration of the liver. The triglyceride concentration of liver in normal cows range from 10 - 15 % on a wet weight (WW) basis, estimation of the lipid content of bovine liver sample obtained by biopsy may be made biochemical or histological methods, both methods provide reasonable estimates of liver fat content over wide range of values, Because liver biopsy is an invasive technique. Measurement of glucagon in liver addition to TAG increase the usefulness of the information, as concentration of TAG and glucagon vary inversely during the early postpartum period, there for it is recommended using rather ratio of liver TAG to liver glucagon, rather than liver TAG alone, as diagnostic indicator for fatty liver. Ecography may be considered as noninvasive on farm method for diagnosis of fatty liver in dairy cow, serum biomarkers that distinguish cow with hepatic lipidosis from those affected by other peripartal disorders. Serum levels of total protein, albumin and urea are indicators of hepatic functionality and decreases in their concentration may reflect fat infiltration in animals with high lipomobilization (Gaal et al., 1983; Herd, 1988; Radostits et al ., 2007; Walter, 2011).

In cattle and other mammals serum potassium levels were below 2.3 mEq/L , and the normal levels ranging from 1.4-2.3 mEq/L (3.5-5.8 mmol/L), with no apparent causes for recumbency however, cardiac arrhythmias are detectable on auscultation and atrial fibrillation is present on electrocardiography (WARD , 1966; Robert et al., 1992 – 1997; Radostits et al., 2007).

In marginal phosphorus deficiency areas, normal non lactating animal in an affected herd may have serum inorganic phosphorus levels within the normal range. Lactating cows in affected herd may have moderately low levels of 2-3 mg/dl (0.65-0.92 mmol/l) and affected animals extremely low levels of 0.4 may

-1.5 mg/dl (0.13-0.48 mmol/l). The inorganic phosphate level in the blood provided an indication of the dietary phosphate intake, and the hemoglobin was measured to assess the presence of anemia. The urine is dark red-brown to black in color and usually moderately turbid, but no red cells present in the urine. Hematologically which PPH has the features of an acute intravascular hemolytic anemia however, the packed cell volume (PCV) falls rapidly to it are lowest level from 4-9 days after the onset of hemoglobinuria the animal becomes icteric as destruction of erythrocytes progresses. Erythrocyte counts and hemoglobin levels are also greatly reduced however; Heinz bodies may be present in erythrocyte in the New Zealand disease. A low copper status of the blood, and liver of affected cows are swollen (MacWillims et al., 1982; Radostits et al., 2007).

1.3. Treatment, control and prevention of diseases associated with nutritional disorders of cattle:

The standard treatment of milk fever is calcium borogluconate at $10-200~\rm g$ promptly with intravenous or subcutaneous is the treatment of choice. The solutions available vary from 18 to 40 % calcium borogluconate. Most cows with milk fever can be treated successfully with $8-10~\rm g$ of calcium (calcium borogluconate is $8.3~\rm \%$ calcium) for cattle, $400-800~\rm mL$ of a 25 % solution is the usual dose. The dose rate of calcium is frequently under discussion. There is a general tendency for veterinarians to under dose with calcium salts, largely because of the toxic effects which tend to occur when all of the calcium is given IV. As an initial dose a large cow of $540-590~\rm kg$ requires $800-100~\rm mL$ of a 25 % solution and a small cow of $320-360~\rm kg$ given $400-500~\rm mL$. Under dosing increases the chances of incomplete response, with inability of the cow to rise, or of relapse. In general, $12~\rm g$ of calcium is superior to $8~\rm g$ which in turn is superior to $6~\rm g$. Treatment is directed toward

restoring normal serum calcium levels as soon possible to avoid muscle and nerve damage and recumbency. Recommended treatment is IV injection of a calcium gluconate salts, although SC and IP routes are also used. A general rule for dosing is 1 g calcium / 45 kg (100 lb) body weight. Most solutions are available in single dose, 500 mL bottles that contain 8 - 11 g calcium. In large, heavily lactating cows, a second bottle given SC may be helpful because it is thought to provide a prolonged release of calcium in to the circulation. SC calcium treatment alone may not be adequately absorbed because of poor peripheral perfusion and should not be the sole route of therapy. Solutions containing formaldehyde or > 25 g dextrose / 500 mL are irritating if given SC. Although administration of phosphorus and magnesium is not usually necessary in uncomplicated parturient paresis, deterimenal effects of their use have not been reported. Magnesium may protect against myocardial irritation caused by the administration of calcium. Calcium propionate in propylene glycol gel or powdered calcium propionate (0.5 kg dissolved in 8-16 L water administered as a drench) is effective, less injurious to tissue, avoids the potential for metabolic acidosis caused by calcium chloride, and supplies the gluconeogenic precursor propionate. Oral administration of 50 g of soluble calcium results in 4 g calcium being absorbed into the circulation. In milk fever there are various methods for control of disease in ruminants, especially dairy cows. these methods include dietary management during the transition period before and after calving administration of calcium gels orally at the time of parturition and administration of vitamin D and its metabolites and analogs immediately before parturition to enhance the mobilization of calcium. When the incidence of milk fever increases to above 10 % of high - risk cows (third or later lactation), a specific control program is necessary. When the incidence is low, a specific control program may not be economical and the alternative is to monitor cows carefully at the time of parturition and for 48 % hours after parturition and treat the affect animals during the first stage of the disease if possible. The two control strategies predicted to be most relevant were calcium gel orally in peripartum used alone or in combination with a low – calcium diet. In general, the most comprehensive control strategies were economically inferior to similar but less comprehensive control strategies. Prevention from milk fever by stimulating cows calcium mobilization before calving, feed a transition diet three weeks to calving, include anionic salts such as calcium chloride, magnesium chloride, magnesium sulfate, calcium sulfate, ammonium sulfate and ammonium chloride, avoid feeds high potassium sodium and calcium in diet, increase dietary calcium immediately after calving, administration vitamin D injections 2 – 8 days before calving for cows with a history of milk fever. Prevention of parturient paresis has been approached by feeding low – calcium diet during the dry period, large doses of vitamin D (20 - 30 million IU) given in the feed for 5 - 7 days before parturition reduces the incidence. However, if administration is stopped more than 4 days before calving, the cows are more susceptible. Dosing for periods longer than those recommended should be avoided because of potential toxicity. Administering large doses of calcium gel (150 g) are given one day before, the day of, and one day after calving (Horst, 1997; Radostits et al ., 2007; Amy and Dave, 2010; Barrington, 2011).

Treatment of ketosis by parenteral dextrose and propylene glycol are necessary for treatment of the ketosis and to avoid fatty liver as complication. Post-surgical convalescence of cows with LAD is clearly related to disturbance in energy metabolism and fatty liver. During convalescence in cows with no fatty liver or moderate fatty liver, the feed intake and daily milk

production increases steadily. In cows with severe fatty liver feed intake remains low. This emphasized the need for effective treatment of excessive lipomobilization, ketosis and fatty liver along with surgical correction of the LDA. All cases of LDA should be corrected as soon as possible to minimize the incidence of peritoneal adhesions and abomasal ulcers, which may perforate and cause sudden death. Treatment of ketosis is to restore the lack of glucose in the body, a quick - acting glucose supplement is required immediately. IV administration of a dextrose solution by a veterinarian is effective in short term, but follow – up treatment is essential if relapses are to be avoided. Drenching with propylene glycol or glycerine has longer term effects, the benefit of ease of administration. Treatment should be continued for two to four days. Many of the long – action corticosteroids have beneficial effects in ketosis. They are administered by the veterinarian as a single injection. Ketosis is treated by using IV injection of 500 mL of a 50% solution of glucose results in transient hyperglycemia, increased insulin and decreased glucagon secretion, and reduced plasma concentration of nonesterified fatty acids and may use other sugars especially fructose, either alone or as a mixture of glucose and fructose. Propylene glycol and glycerine or glycerol to overcome the necessity for repeated injections, propylene glycol can be administered as a drench with the traditional dose is 225 g twice daily for two days, followed by 110 g daily for two days to cattle, but higher volumes are also used. Treatment by hormonal therapy like glucocorticoids, insulin, anabolic steroids are aid in treatment of the disease. Most widely used treatments of the disease is injection of glucose into neck vein or under the skin, injection of cortisone or ACTH, or oral administration of propylene glycol or sodium propionate. The hormone injections are sometimes effective, but they are expensive and repeat treatments may be needed. All of the treatments are an attempt to increase the blood sugar and thus reduce ketones and fat mobilization. The treatment or combination of treatments which should be used is a matter for the veterinarian and the dairyman to decide, based on the individual situation. Ketosis the prevention form depends on adequate feeding and management practices. In times of feed deficiency because of drought or other reasons, the provision of supplementary feed with adequate amounts of carbohydrates is essential. Cows should be on a rising plane of nutrition up to calving with the aim to calving in good condition. After calving, the cow has the potential to reach maximum efficiency in milk production, but feed requirements for high production are often greater than the voluntary intake of pasture can provide. Therefore an energy supplement is required and there is evidence that this will improve production and reproductive performance, and decrease the risk of ketosis. The best supplements are good quality hay, silage, or cereal grains. Supplements should be feed at least until the peak of lactation is reached or longer depending on the quality and quantity of available pasture. Prevention of ketosis would be more satisfactory than treatment if the methods involved were practical. Do not have cows excessively fat at calving time. It appears desirable to have them in good condition, but excess fat means that more fat is burned at the time of stress, putting an extra load on the liver. Keeping the cows full of good quality roughage. This is just good dairy management, and it helps of keeping the balance between income and outgo. Picking out the best hay for borderline ketosis cows as well as feeding them more often may be helpful. Feeding a moderate amount of grain prior to calving also may be beneficial provide facilities for adequate comfort, exercise, and ventilation. All general management practices which tend to increase the appetite and aid the comfort of the cow are desirable. The control of clinical ketosis is

generally related to the adequate nutrition of the cow in the dry lactating period. This encompasses details such as dry matter intake, fiber digestibility, particle size distribution, energy density, fat incorporation in early lactation rations protein content, feeding systems, rumen size, other factors better covered in texts on nutrition. It is difficult to make general recommendations for the control of the disease because of the many conditions under which it occurs, its probable multiple etiology, and feeding systems that vary from those that feed components separately to those that feed total mixed rations. Cows should neither have been starved not be overfat at calving. Careful estimation of diet by reference to feed value tables is recommended and detailed recommendations on diet and management available with the caveat that planned rations can deviate from feed bunk rations and feed bunk dry matter and actual dry matter intake may not be the same. Too low a feeding frequency and the feeding of concentrates separate from roughage rather than as a total mixed ration can lead to an increase in rates of ketosis. Prevention of ketosis is via nutritional management should be aimed at minimizing this reduction. Some feed additive including niacin, calcium propionate, sodium propionate, propylene glycol, and rumen - protected choline, may be beneficial in preventing and managing ketosis. Reducing body condition in the dry period may even be counterproductive, resulting in excessive adipose mobilization prepartum (Anderson and Elwalt, 1970; Baird, 1982; Nicky, 2007; Radostits et al., 2007).

Joachim (2012) reported that the treatment is, assisting the cow to rise on every day of the recumbent, an attempt should be made to bring the cow to it is feed. Several simple but effective techniques can be tried, in one method the clinician stands with feed pressed under the cow at point below the scapulohumeral joint. A sharp blow is delivered by driving the kness into the

muscle mass below and caudal to scapula, this method must not be used on the thoracic wall unprotected by the muscle mass to avoid fracturing the ribs. If the animal struggles to rise, an assistant should grasp the root of the tail with both hands and lift. Lifting on any other part of the tail may causes damage. Some workers use electric goads and various traditional methods of inflicting pain to stimulate a cow to rise, these measures have a low success rate inexperienced hands. The value of hip champs is controversial, their proper use requires experience skill and a delicate touch, continual causes of trauma and pain that is the counterproductive, the fore limbs support 60 % of cows weight and therefore the use of a canvas sling under the sternum is almost mandatory for consistent success. The other method of treatment is moving the cow to allocation with an earthen floor, in warm relatively dry weather the best location for recumbent cow is grassy pasture, although this means that a method for lifting the cow must be readily available, otherwise the location selected should have a roof and some protection from the elements. Recumbent cattle should be examined daily to determine any change in ability to rise or bear weight. Every effort should made to treat affected cow as soon as possible after clinical signs are obvious. Treatment during the first stage of the disease, before the cow is recumbent is the ideal situation. Complication of milk fever occurs when cows have been in sternal recumbency for more than 4 hours. The standard treatment calcium borogluconate at 10 - 200 g is the treatment of choice. The solutions available vary from 18 - 40 % calcium borogluconate. Most cows with milk fever can be treated successfully with 8 – 10 g of calcium for cattle 400 - 800 ml of a 25 % solution is the usual dose, the dose rate of calcium is frequently under discussion. There is a general tendency for veterinarians to under dose with calcium salts, largely because of toxic effects which lead to occur when all of the calcium is given IV. As an initial dose a large cow (540 – 590 kg) requires 800 – 1000 ml of 25 % solution and small cow (320 - 360 kg) 400 - 500 ml. Under dosing increases the chances of incomplete response with inability of the cow to rise or of relapse. The standard rate of administration is rapid IV administration of the calculated dose of calcium borogluconate (often supplemented with phosphorus, magnesium and glucose) over a period of 15 minutes, in the cows treated rapidly the serum calcium and magnesium levels increased rapidly compared with the infused cows. Treatment was done by medicine a long with the physiotherapy to correct the etiology factor. Injection oxytetracycline at the dose rate 10 mg/kg body weight were administration intramuscularly for five and three days respectively. Injection calcium and magnesium borogluconate (Mifex) 450 ml IV slow and 150 ml SC and injection 5 % DNS 2000 ml IV once were administered, injection vitamin B complex 10 ml IM for 3 days were administered. Powder potassium chloride 30 mg orally for 5 days was given and physiotherapy comprised of soft bedding and supportive slings. Sand was used as bedding material because cow as easily rolled from one side to other and for the regular removed excreta. Cow was able to stand up without help of slings after complete recovery effective strategies to prevent or control of milk fever are important to decrease downer cow syndrome and prophylactic administration of calcium of all cows with second or later lactation is beneficial in herd with high incidence of milk fever (Radostits et al., 2007; Joachim, 2012; Nirmala and Kaswan, 2013).

The prognosis for severe fatty liver is unfavorable and there is no specific therapy. In general cows with the severe fat cow syndrome which are totally anorexic for 3 days or more usually die in spite of intensive therapy. Fluid therapy and electrolyte therapy directed at correcting the effects of the ketosis

and fatty liver is required. The recommended treatment including continuous IV infusion of 5% glucose and multiple electrolyte solution, and the intraruminal administration of rumen juice 5 – 10 L from normal cows in an attempt to stimulation the appetite of affected cows. Glucagon injection SC of 15 mg/dl for 14 days beginning at day 8 postpartum decreases liver triglyceride concentration in cows older than 3.5 years, daily administration of the same amount 15 mg/dl for several days prepartally in limited number of cows were effective in preventing fatty liver during the early postparturient period. Glucocorticoids at 200 mg given IM daily for days decreased liver triglyceride concentration. Propylene glycol given orally at 1 L/day promotes gluconeogenesis and is used for the treatment of ketosis. Insulin as zinc protamine at 200 – 300 SC twice daily promotes the peripheral utilization of glucose. injection of prednisolone at 200 mg per day for five days have been shown to decrease the amount of TAG in the liver. In addition of glucocorticoids and adrenocorticotrop hormone (ACTH) have been used to treat fatty liver as well as to increase concentration of glucose in plasma. Several strategies have been applied to prevent fatty liver including general management practices as well as hormonal and chemical treatment. Feeding cows a balanced diet according to the recommended dietary requirement for the transition period, treat sick cows immediately and aggressively in the periparturient period, also is highly recommended and addition insulin used at 0.14 IU/ Kg body weight was able to prevent accumulation of TAG in the liver. The control and prevention of fatty liver in cattle depend on decreasing or eliminating most of the potential risk factors for these disease. The early recognition and treatment of disease which affect the voluntary dietary intake in late pregnancy and immediately after parturition is necessary to minimize the mobilization of body fat stores to meet the overall energetic requirements

of the cow during the period of negative energy balance, and to maintain or increase hepatic glucogenesis (Herd, 1998; Bobe et al., 2004; Osman et al., 2008).

Treatment including IV and oral administration of potassium chloride is drug of choice for treating dairy cattle with hypokalemia and fluid therapy but the response is commonly infective. Addition of potassium chloride to a 0.9 % saline solution given as a continous IV infusion at rates of up to 300 mmol of potassium per hour has been used in 3 years old cow. oral supplementation with potassium chloride salt at 230 g two to three times daily for 3 days was associated with recovery. palatable hay and propylene glycol orally are recommended, hypokalemia in severe weakness and recumbency treated with isoflupredone acetate but for ketosis repeated doses well decreases plasma concentration of potassium by 70–80 % which suggests a strong mineralocorticoid activity (Robert et al., 1992 – 1997; Radostits et al., 2007).

A transfusion of whole blood is indicated in severe cases of postparturient hemoglobinuria, a minimum which given of 5 L of blood to a 450 kg of cow is recommended, this will usually suffice for up to 48 hours by which time an additional transfusion may be necessary if the cow is weak and mucous membranes pale, administration 2000 ml of 5 % serum dextrose for 2 days IV, dexamethasone 4.4 mg/ml given IM once a day for 2 days, thiamin hydrochloride 30 mg given IM once a day for 5 days, and tocopherol 50 mg and selenium 1.5 mg/ml which given IM once a day for 3 alternating days. Fluid therapy is recommended as both supportive therapy and to minimize the danger of hemoglobinuric nephorosis. The administration of phosphorus to acutely ill animals should include the IV administration of 60 g of sodium acid phosphate in 300 ml of distilled water and a similar does SC, followed by

further SC injections at 12 hours intervals on three occasions and similar daily does by mouth. Oral dosing with bone meal 120 g twice daily or dicalcium phosphate or a suitable source of calcium and phosphorus daily for 5 days is recommended followed by inclusion in the ration. Ketosis is a common complication of the disease and additional treatment for it may be required. Control an adequate intake of phosphorus according to the requirements for maintenance and milk production should be insured particularly in early lactation. The incidence of the disease is reported after copper supplemented of cattle in a copper – deficient area (Mac Willims et al., 1982; Radostits et al., 2007).

Chapter Two

Material and methods

2.1. Study Area:

The study was carried out from November to December 2017 in Jabal Awlia and Omdurman localities in Khartoum state. The khartoum state line between longitudes 31.5-34 east and latitudes 15-16 north in an area about 28-165 square kilometers. It is bordered to the north and east side by the River Nile state, north western by the Northern state and to the east and south eastern by state of Kassala, Gazira and Gadaref. The Khartoum in climatic of semi-desert region. The weather is rainy in summers, cold and dry in winters. (Khartoum www krt.gov.sd 2012). The populations of animals in Khartoum state at 240003 for cattle, 6300000 for birds, 513000 for sheep, 19000 for goat, 6585 for camel. The number of cattle distributed in Khartoum state is 138067 in east Nile, 28016 in Baharri, 13578 in Ombeda,13901 in Karari,20455 in Omdurman, 20360 in Jabalawlia, 5626 in Khartoum localities (Khartoum census 2008). The study was carried out at Jabalawlia locality which is located in south of Khartoum state namely: Akalakla, Mayo, Alazhary, Tuba, Aldekhenat, Eseli, Araksaleh, Neaam and Darelsalam Jabal Awlia also the study include Omdurman locality which is located in west of Khartoum state namely: Ombeda, Fatiha, Salha, Elgamoea, Laybia, Eleskan and Elradwan.

2.2. Collection of Samples:

The total of 50 blood samples were collected randomly from 2-4 years old dairy cows [25 samples from each locality]. Whole blood (20ml) was taken from each animal for separation of serum [5ml] for biochemical analysis.

2.3. Biochemical Tests:

Biochemical parameters associated with dairy cattle in lactation period were measured spectrophotometrically. Total protein, albumin, glucose, inorganic phosphorus, calcium, sodium and potassium were analysed by test kits (Bio system, S.A.Spain).

2.3.1. Total proteins determination:

Protein in the samples reacted with copper ion in alkaline medium forming a color and measured by Jenway spectrophotometry at 623 nm (Gornall et al., 1949).

2.3.2. Determination of Phosphorus:

Inorganic phosphorus measurement based on reduction of phosphomolybdate to molybdenum. The intensity of the color measured photometrically at 600 nm (Taussky et al., 1953).

2.3.3. Determination of Calcium:

The test based on formation of color from calcium reacted with Cresolphtalein in alkaline medium. The measurement of the test at wavelength 570 nm (Farell et al., 1984).

2.3.4. Determination of Sodium:

The reaction based on sodium in combined to selective chromogen and produced chromophore. the test was read by spectrophotometer at 623 nm (Maruna, 1958).

2.3.5. Determination of Potassium:

By using sodium tetraphenylboron to produce a colloidal suspension forming turbidity. The test was read at 623 nm (Maruna, 1958).

2.3.6. Glucose determination:

The test was based upon the oxidation of sugar in the blood according to Trinder et al., (1969). Phenol and 4. Amino – antipyrine with enzyme

peroxidase were produced red chinonimin. The test was read at 546 nm using spectrophotometer.

2.3.7. Albumin determination:

Measurement of albumin was based on its binding to indicator dye bromocresol green in pH 4.1 to form a blue-green color. The reading the test at 623 nm (Doumas et al., 1971).

2.4. Questionnaire design and data collection:

Questionnaire was designed to obtain information on demographic characteristic of the dairy men, nutritional disorders in dairy cows in lactation period, history of diseases associated with nutrition and production, risk factors associated with dairy farms and feeding system. The questionnaire included 50 dairy people in the Jabal Awlia [25 person], and Omdurman [25 person] localities using face to face questions.

2.5. Statistical Analysis:

The data of biochemical analysis was analyzed using statistical package for social science (SPSS), especially independent T- test for the proportion of the some different groups. Analysis of the questionnaire data by using Chi-square Test for examination difference between group and to test the association the independent variable and infection status [p<0.05].

Chapter Three

Results

There were no significant changes in biochemical parameters as shown in table 1. While serum protein level showed statistical significant difference [p<0.05] in Jabal Awlia and Omdurman localities. In table 2 the ages of the respondents were between 21 to over 40 years, while 40% of them were uneducated, but only 12% were graduated. Most of them their experience of working in the farms were low (36%), but only 8% having a knowledge about the work (Work over 19 years). Questionnaire results showed that [48%] of Nutritional disorders in dairy cows in mid lactation period commonly occurred [Table3]. All nutritional diseases were higher in Omdurman locality than Jabal Awlia locality [Table 3]. The management system in the two localities represented to the source of nutritional disorder to the dairy animals [Table 4]. The animals in common grazing [32%] with lower nutritional disorders than animals were no grazing [36%]. Also the animals moving in and out the farm showed lower affection by the nutritional diseases [34%], whereas, the animals with limited movement were susceptible by these diseases [56%]. Pastoral farming system recorded low diseased animals [14%] than semiintensive system [48%] in the two localities [Table 4]. Feeding system in the two areas composed of concentrates and roughages. The constitutes of the concentrates were corn, wheat, oats. The roughages used in two areas were pasture, hay, silage green chap and sawdust. Other supplements were calcium, potassium, sodium chloride, antibiotics, antioxidant, copper and molasses.

Table (1): The mean \pm standard error of nutritional elements in lactating cows (n= 50) in Khartoum state

| Elements | Are | Sig | |
|---------------|-------------|-------------|---------|
| | Jabal Awlia | Omdurman | |
| Total protein | 7.74±0.08 | 7.94±0.04 | 0.039 * |
| Phosphorus | 3.57±0.05 | 3.60±0.05 | 0.674 |
| Calcium | 8.31±0.06 | 8.36±0.07 | 0.565 |
| Sodium | 131.38±1.20 | 134.28±0.82 | 0.053 |
| Potassium | 3.60±0.06 | 3.62±0.06 | 0.872 |
| Glucose | 78.22±3.69 | 74.47±2.38 | 0.398 |
| Albumin | 3.76±0.09 | 3.55±0.08 | 0.109 |

^{* =} Significant difference (P<0.05)

Table (2): Demographic characteristics of dairy men [n=50] in Khartoum state

| Respondents | Areas | | Total | Chi | Sig |
|----------------|----------------|--------------|-------------|--------|-------|
| | Jabal Awlia | Omdurman | | square | |
| Age (years) | | | | | |
| 21—25 | 4(16%) | 3(12%) | 7(14%) | | |
| 26—30 | 4(16%) | 5(20%) | 9(18%) | | |
| 31—35 | 4(16%) | 5(20%) | 9(18%) | 0.442 | 0.979 |
| 36—40 | 6(24%) | 6(24%) | 12(24%) | | |
| Over 40 | 7(28%) | 6(24%) | 13(26%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |
| Level of educa | ation | | | | |
| Uneducated | 12(48%) | 8(32%) | 20(40%) | | |
| Primary | 7(28%) | 7(28%) | 14(28%) | | |
| High School | 2(8%) | 4(16%) | 6(12%) | 4.610 | 0.330 |
| Secondary | 4(16%) | 3(12%) | 7(14%) | | |
| Graduate | 0(0%) | 3(12%) | 3(12%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |
| Number of ye | ears of experi | ence in anim | al business | | |
| 1 - 4 | 9(36%) | 9(36%) | 18(36%) | | |
| 5 – 9 | 5(20%) | 6(24%) | 11(22%) | | |
| 10 - 14 | 7(28%) | 6(24%) | 13(26%) | 0.168 | 0.997 |
| 15 – 19 | 2(8%) | 2(8%) | 4(8%) | | |
| Over 19 | 2(8%) | 2(8%) | 4(8%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |

^{* =} Significant difference (P<0.05)

 $\label{eq:constraint} \begin{picture}(2000)(20$

| Occurrence on the | Areas | | Total | Chi - | Sig |
|---------------------|----------------------|---------|----------|--------|-------|
| farm is | Jabal Awlia Omdurman | | - | square | |
| First time | 5(20%) | 3(12%) | 8(16%) | | |
| Second time | 5(20%) | 4(16%) | 9(18%) | | |
| Third time | 5(20%) | 4(16%) | 9(18%) | 1.389 | 0.708 |
| Common | 10(40%) | 14(56%) | 24(48%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |
| Season of the occur | rence | I | | | |
| Dry | 11(44%) | 13(52%) | 24(48%) | | |
| Rainy | 5(20%) | 5(20%) | 10(20%) | 0.417 | 0.812 |
| Any season | 9(36%) | 7(28%) | 16(32%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |

^{* =} Significant difference (P<0.05)

Table (4): Risk factors associated with dairy farms in Khartoum state

| | Aı | reas | Total | Chi – | Sig |
|--------------------------------------|----------------|-----------|----------|--------|-------|
| Affect of disease on sheep and goats | Jabal Awlia | Omdurman | | square | |
| Present | 25(100%) | 25(100%) | 50(100%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |
| Source of livestock feed | on the farm | | | | |
| Communal grazing | 8(32%) | 8(32%) | 16(32%) | | |
| Enclosed | 8(32%) | 8(32%) | 16(32%) | 0.000 | 1.000 |
| Zero grazing | 9(36%) | 9(36%) | 18(36%) | | |
| Total | 25(50%) | 25(505) | 50(100%) | | |
| Source of water for ani | mals | | | | |
| Communal water | 10(40%) | 11(44%) | 21(42%) | | |
| Individual water | 15(60%) | 14(56%) | 29(58%) | 0.082 | 0.774 |
| Total | 25(50%) | 25(50%) | 50(100%) | | |
| Movement of animals in | nto and out to | o of farm | | | |
| Any direction | 12(48%) | 5(20%) | 17(34%) | | |
| Limited | 10(40%) | 18(72%) | 28(56%) | 5.368 | 0.068 |
| Pre-testing | 3(12%) | 2(8%) | 5(10%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |
| Farming system | | | | | |
| Pastoral | 1(4%) | 6(24%) | 7(14%) | | |
| Mixed | 12(48%) | 7(28%) | 19(38%) | 4.887 | 0.087 |
| Semi-intensive | 12(48%) | 12(48%) | 24(48%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |
| Source of diseases | | | | | |
| Infected animals | 1(4%) | 1(4%) | 2(4%) | | |
| Contacts | 3(12%) | 3(12%) | 6(12%) | | |
| Wild animals | 0(0%) | 1(4%) | 1(2%) | | |
| Movement | 5(20%) | 3(12%) | 8(16%) | 1.967 | 0.854 |
| No | 14(56%) | 16(64%) | 30(60%) | | |
| Total | 25(50%) | 25(50%) | 50(100%) | | |

^{* =} Significant difference (P<0.05)

Chapter Four

Discussion

A variety of biochemical parameters can be measured in dairy cows in lactation period associated with nutrition to evaluate the abnormalities in this stage. In this study all biochemical parameters evaluated [Table 1] provide no changing in their value [p>0.05], but total protein was significantly decreased and this is in accordance with the results of Rania (2014) who reported that the measured parameters are normal in lactated cows and these increase in late lactation and decreased in early lactation. Also adequate amounts of protein are required not only for the animal its self, but also the rumen microflora in low quantity [Faster and mays, 1986]. The major factor leading to decrease of protein with shortage of energy is poor pasture [Non legume, forages, specially roughages]. Generally, the seasonal effect can be influenced glucose, calcium, phosphorus, sodium and potassium levels (Cerutti et al., 2018). Blood glucose level is normal in lactation stage in in these results. These findings are in agreement with Rodastits et al.(2000); Flore et al.(2015); Dioavic et al.(2016) who recorded that detection of hypoglycemia in early lactation period in the cows is indicative values. From the results the disorder in nutrition occurred in dry season [Table 3] and in old cows with high morbidity and mortality rates [Table 4]. This may be attributed to dairy men who have no knowledge of practicing in dairy farms [Table 2]. However, special clinical and laboratory examinations of animals are of valuable to diagnosis of diseases and measurement of blood or tissue concentration of minerals and vitamins or biochemical markers in apparently healthy animals that to avoid the occurrence of nutritional deficiencies specially in lactation stage [Rodastits et al., 2007].

Conclusion

In conclusion, the results of this study revealed that all biochemical parameters measured were normal in the lactation period in these cows. But morbidity and mortality rates were high in the two areas with low knowledge of practicing in animal farms. Also supplementation of diet constitutes energy feed, minerals, vitamins, concentrate mixture improve the animal health and prevent occurring of the diseases in lactation period.

Recommendation

- Must be to correct the diet and feed additive during the lactation period especially high energy to controlling the hypocalcaemia.
- Treatment of diseases animal symptomatically to avoid the prolong recumbency that lead to some of diseases.
- Regular monitoring of animal behaviour to detect any signs like recumbency, lacrimation, bloat and diarrhea to treatment.
- Routine examination and differentiation of metabolic disorders and other systematic toxic condition.
- To reduce the cost of roughages through increasing the production of the integrating green fodder agriculture and rotation system, adoption of artificial insemination technique.
- To understand feed standard for the lactation cows.
- To train dairy farmers to improve their managerial skills and knowledge, and use of high production breeds to avoid competition from abroad regarding milk production.
- To encourage improved research in dairy production in Sudan.

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Appendices:

Appendix (1)

Table (1): Value of some elements in nutritional disorders in lactating cows (n=50) in Khartoum state

| Elements | Resul | normal levels | |
|----------------|----------------|---------------|-----------------|
| | Jabal Omdurman | | |
| | Awlia | | |
| Total proteins | 7.74 g/dl | 7.94 g/dl | 6-9 g/dl |
| Phosphorus | 3.57 mg/dl | 6.60 mg/dl | 2- 6 mg/dl |
| Calcium | 8.31 mg/dl | 8.36 mg/dl | 6-11 mg/dl |
| Sodium | 131.38 | 134.28 | 90-210 m.mol |
| | m.mol | m.mol | |
| Potassium | 3.60 m.mol | 3.62 m.mol | 3– 7 m. mol |
| Glucose | 78.22 mg/dl | 74.47 mg/dl | 50-150 mg/dl |
| Albumin | 3.76 g/dl | 3.55 g/dl | 2.5 - 5.5 g/dl |

Questionnaire Data

Appendix (2)

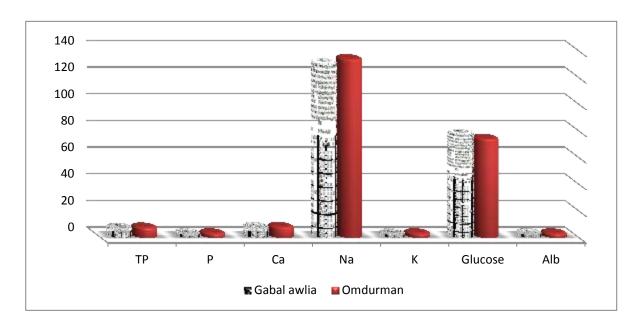


Figure (1) : Some nutritional elements from lactated cows (n=50) in Khartoum state

Appendix (3)

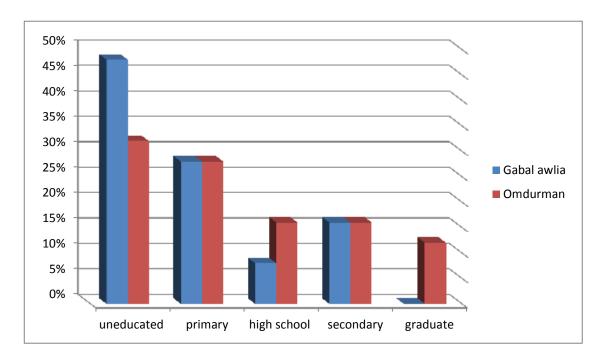


Figure (2) : Level of education of the respondents (n=50) in Khartoum state

Appendix (4)

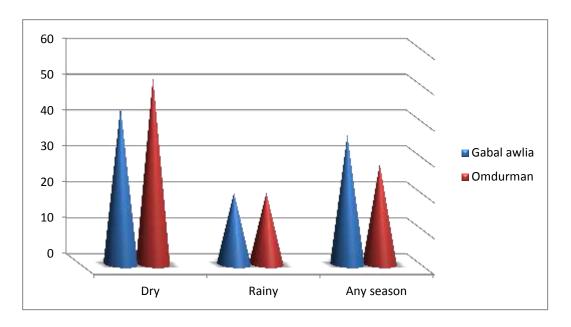


Figure (3): Season of the occurrence of nutritional disorders in lactated cows (n=50) in Khartoum state

Appendix (5)

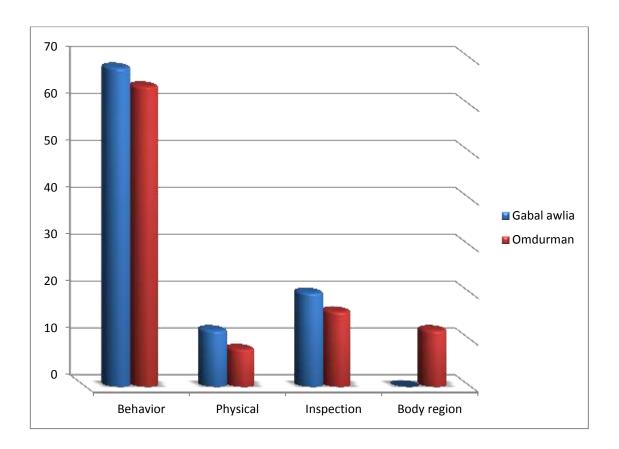


Figure (4): Methods of detection nutritional disorders in lactated cows (n = 50) in Khartoum state

Appendix (6)

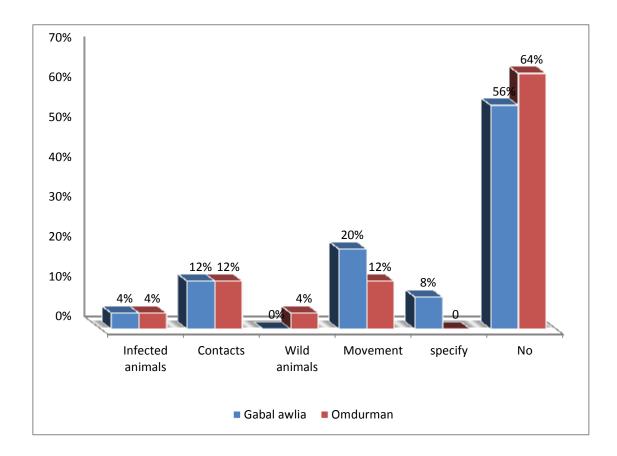


Figure (5): Source of diseases of nutritional disorders in lactated cows (n = 50) in Khartoum state

Appendix (7)

Questionnaire about knowledge of nutritional disorder in dairy cows in lactation period in Khartoum state:

| No | |
|---------------|---|
| State | Locality Date |
| 1. Demograp | phic characteristics of dairy men in Khartoum state |
| a. Name: | |
| b. Address: . | |
| c. Age: | |
| 21-25 | years |
| 26-30 | years |
| 31-35 | years |
| 36-40 | years |
| >40 | years |
| d. Level of e | ducation:- |
| Un educated | |
| Primary | |
| High school | |
| Secondary | |

| Graduat | e | | | | |
|-----------|-----------|---------|---------------|-------|---------------------------------|
| Professi | onal | | | | |
| e. Numb | er of ye | ears of | experience i | n ani | mal business: |
| 1-4 | years | | | | |
| 5-9 | years | | | | |
| 10-14 | years | | | | |
| 15-19 | years | | | | |
| >19 | years | | | | |
| 2. Nutri | tional d | lisorde | ers in dairy | cows | in lactation period In Khartoum |
| state | | | | | |
| 2.1. Occ | currence | of dise | eases in farn | n is: | |
| First nar | me: | yes | | no | |
| Second | time: | yes | | no | |
| Third tin | me: | yes | | no | |
| Commo | on: | yes | | no | |
| 2.2. Sea | son of tl | ne occi | ırrence: | | |
| Dry seas | son: | yes | | no | |
| Rainy se | eason: | yes | | no | |
| Any sea | son: | yes | | no | |

| 2.3. Nutrition | al disorders in f | arms: | | |
|-----------------|-------------------|---------------------|----|--|
| 200 - 2005 : | yes | no 🔲 | | |
| 2006 - 2010 : | yes | no 🗌 | | |
| 2011 : | yes | no | | |
| None: | yes | no 🔲 | | |
| 3. History of | diseases in Kh | artoum state: | | |
| 3.1. First outb | reak and date re | eported occurrence: | | |
| 2000-2005: | yes | no 🗌 | | |
| 2006-2010: | yes | no 🗀 | | |
| 2010-2015: | yes | no 🔲 | | |
| None: | yes | по | | |
| 3.2. Livestock | population in t | he farm: | | |
| 3.2.1. Total po | opulation at risk | : : | | |
| Exotic breed | 2-4 years: | yes | no | |
| | >4 years: | yes | no | |
| Cross breed | 2-4 years: | yes | no | |
| | >4 years: | yes | no | |
| Local breed | 2-4 years: | yes 🗆 | no | |
| | >4 years: | ves 🖂 | no | |

| 3.2.2. Total number of animal affected / sick (Morbidity): | | | | | | |
|--|---------------|-----------------|-------------------------------------|--|--|--|
| Exotic breed | 2-4 years: | yes 🗆 | no 🗀 | | | |
| | >4 years: | yes | no 🔲 | | | |
| Cross breed | 2-4 years: | yes | no 🗌 | | | |
| | >4 years: | yes | no 🗌 | | | |
| Local breed | 2-4 years: | yes | no 🗌 | | | |
| | >4 years: | yes 🗆 | no \square | | | |
| 3.2.3. Total n | umber of anim | al dead (Morta | ality): | | | |
| Exotic breed | 2-4 years: | yes 🔲 | no | | | |
| | >4 years: | yes 🗀 | no | | | |
| Cross breed | 2-4 years: | yes 🗀 | no 🗀 | | | |
| | >4 years: | yes | no 🗀 | | | |
| Local breed | 2-4 years: | yes | no | | | |
| | >4 years: | yes | no | | | |
| 3.2.4. Major | symptoms obs | erved in differ | ent group of nutritional disorders: | | | |
| Exotic breed | 2-4 years: | yes 🗌 | no 🔲 | | | |
| | >4 years: | yes 🗆 | no 🗌 | | | |
| Cross breed | 2-4 years: | yes 🗌 | no 🗌 | | | |
| | >4 years: | yes 🗌 | no 🗌 | | | |

| Local breed 2- | -4 years: | yes | | no 🗌 | |
|--------------------|----------------|----------|-------------|---------------|----------------|
| ` | >4 years: | yes | | no 🗌 | |
| 3.2.5. Methods of | f deduction n | utrition | al disorder | in dairy cov | v in lactation |
| period: | | | | | |
| - Behavio | our and gener | al appe | arance: | yes | no 🗌 |
| - Physica | ıl examination | n metho | ods: | yes | no 🗌 |
| - Inspect | ion of body re | egions : | | yes \square | no 🗆 |
| - Clinical | l examination | of bod | y regions : | yes 🔲 | no 🗌 |
| 4. Risk factors a | ssociated wit | th dair | y farms in | Khartoum | state: |
| 4.1. Effect of the | disease on sh | eep and | d goats: | | |
| Prese | ent: yes | s 🗀 | n | 0 🗌 | |
| Abse | nt: yes | | no |) [| |
| Not k | nown: yes | | no | о 🗌 | |
| 4.2. Source of liv | estock feed o | n the fa | arm: | | |
| Common grazing | g: yes | | no | о | |
| Enclosed / fenced | d grazing: yes | | no | | |
| Zero grazing: | yes | | no | | |
| 4.3. Source of wa | ater for anima | ls: | | | |
| Common water: | yes | s 🗀 | n | 0 🔲 | |
| Individual source | e: yes | s 🔲 | no | o 🔲 | |

| 4.4. Movement of animal | into and out to | of the farm: | | | | | | |
|---|-----------------|--------------|------|--|--|--|--|--|
| Possible in any direction: | yes | no 🗌 | | | | | | |
| Limited movement: | yes 🗌 | no 🗌 | | | | | | |
| Requires pre-testing for livestock disease: | | | | | | | | |
| 4.5. Farming system: | | | | | | | | |
| Pastoral: | yes | no 🔲 | | | | | | |
| Mixed: | yes | no | | | | | | |
| Semi-intensive / intensive | : | | | | | | | |
| 4.6. Source of diseases: | | | | | | | | |
| Introduction of infected ar | nimal (s): | yes 🗌 | no 🗌 | | | | | |
| Contact at communal point | at (specify): | yes 🗌 | no 🗌 | | | | | |
| Contact with wild animals | : | yes 🗌 | no 🗌 | | | | | |
| Movement of in fected and | imal (s): | yes 🗌 | no 🗌 | | | | | |
| No: | | yes 🗌 | no 🗌 | | | | | |