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Sudan University of Science and Technology

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Metabolic Profile of Dairy Cattle in the Khartoum State

معالم الاستقلاب في الأبقار الحلوب في ولاية الخرطوم

A theirs submitted in fulfillment for requirement of degree

for Master of Science in Preventive Medicine

By

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B.V.Sc. October 2009

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July 2014

Dedication

To my Parents, brothers and sisters, who surrounded me with love and care and whom I am always indebted , and also to my friends and all whom I love I dedicate this work.

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Acknowledgement

First and final , I thank Allah who bestowed upon me and giving me health and strength to complete this work .I would like to thank my supervisor Dr . Siham Elias Suliman Department of Medicine and Animal Surgery , College of Veterinary Medicine , Sudan University of Science and Technology (SUST) for her continuous attention, encouragement, support , guidance , reading draft manuscript , constructive criticism which gave me a lot of self confidence and advice throughout this study and I am deeply indebted 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 valnable Co- supervision , interest , guidance , providing me with all the necessary materials that helped me completing this study . I would like to thank my friend Dr. Maria Taha Hamid a sincere thanks for her close assistance and continuous saport in fullfilling this study . I would like to thank Dr. Wejdan Osman Department of Preventive Medicine (SUST) and Hassan Motwakel for his great help during this study .

Abstract

In this study a total of 40 whole blood and plasma samples were collected from 30 pregnant dairy cows in different stages (first , second and third stages) of pregnancy and 10 from empty cows as control to evaluate the profile of certain hematological parameters and biochemical parameters with main aim to protect pregnancy cows from production disease .

The survey was conducted at Khartoum State and the area selected were West Omdurman (Alrodwan), JabalAwlia (Aldikhinat) and East Nile (Sudan University farm and Soba) .Haematological findings revealed that , there was a significant changes in the packed cell volume (PCV) and slight changes in hemoglobin (Hb) within and between the group of pregnancy cows compared with control group (P< 0.05) . PCV was low in first stage (52.6±6.6ml/dl) , and in third stage (74.0±6.4mmol/l) respectively compared with control group (76.4±4.5mmol/l) , whereas in the second stage PCV was high (86.5±1.3mmol/l) . there was slight changes in haemoglobin concentration, during the first stage there was a decreases (7.3±1.3g/dl) , whereas in the second stage (12.8±0.7g/dl), and third stage (11.9±1.0g/dl) the level was more than control group was (11.4±0.9g/dl)

The total protein was (169.0±30.3 g/l), (88.2±8.8g/l) in first stage and in third stage respectively and both were higher as compared with controlled (67.8±3.6g/l) , the second stage (57.4±1.9g/l) it was low . The albumin value showed increases during first stage and third stage (69.9±12.9g/l) , (44.0±8.0g/l) respectively , whereas the second stage (26.6±1.8g/l) was lower than control group (28.5±3.9g/l) . The globulin in second stage of pregnancy was low (30.7±2.7g/l), while in first stage (99.8±17.9g/l)and third stage(42.4±7.0 g/l) it was high as compared with control group (39.3±4.1g/l) . Plasma glucose and calcium there were significant change within the group .The glucose level was decreased in the first and second stage of pregnancy (4.1±0.3mmol/l , 4.8±0.2mmol/l) respectively , whereas during the third stage (4.9±0.7mmol/l) these was increases as compared with control group (4.3±0.4mmol/l) . The value of calcium was (2.4±0.6mmol/l) in the first stage and (1.6±0.3mmol/l) in second stage , these values were both low as compared with (3.2±1.2mmol/l) in the control group , the third stage (3.8±0.9mmol/l)was increases as compared with control. The good management of pregnant cows before and after calving with treatment are important in controlling the production disease .

ملخص الاطروحه

فى هذه الدراسه تم فحص عدد 40عينه دم وبلازما , تم اخذ 30 عينه من بقر وحمل فى مراحل الحمل المختلفه (مرحله الحمل الاولى – الثانيه و الثالثه) و 10عينات من بقر غير وحمل لتقيم بعض معالم مخطط الدم و الكيمياء الاحيائيه لحمايه الابقار الحلوب اثناء مراحل الحمل من امراض الاستقلاب، وتم ذلك فى ولايه الخرطوم وشملت الاماكن الاتيه غرب امدرمان (الرضوان) ، جبل الاولياء (الدخينات) , شرق النيل (مزرعه جامعه السودان للعلوم والتكنلوجيا) و سوبا .فى معلومات مخطط الدم وجد تغير مفيد فى مكداس الدم ولكن الهيموقلوبين اوضح تغير بسيط فى مراحل الحمل المختلفه . مكداس الدم كان منخفضا فى مرحله الحمل الاولى (52.6±6.6 ملمول \التر) وكذلك فى المرحله الثالثه من الحمل (74.0±6.4 ملمول \التر) مقارنه مع البقر الغير حُمل (76.4±4.5 ملمول \التر) ولكن اظهر زيادة فى المرحله الثانيه من الحمل (86.5±1.3 ملمول \ لتر) . ووجد تغير بسيط فى الهيموقلوبين حيث اظهر انخفاض فى مراحل الحمل الاولى(7.3±1.3 جم \د لتر) وزيادة فى مرحله الحمل الثانيه والثالثه (12.8±0.7 جم\د لتر 11.9± 1.0 ,جم\د لتر) على التوالى مقارنه مع البقر الغير وحمل (11.4±0.9 جم\د لتر).

البروتين الكلى كان عالى فى كل من المرحله الاولى والثالثه من الحمل (, 169.0±30.3 88.2±8.8 جم\لتر) على التوالى مقارنه مع الابقار الغير حُمل (67.8±3.6 جم\لتر) وهنالك انخفاض فى مرحله الحمل الثانيه (56.0±1.9جم\لتر) , مستوى الالبيومين فى المرحله الاولى والثانيه عالى (44.0±8.0 , 69.9± 12.9 جم\لتر) اما المرحله الثانيه (26.6±1.8 جم\لتر) كانت منخفضه مقارنه مع الابقار الغير حُمل.

القلوبولين فى المرحله الثانيه من الحمل منخفض (30.7±2.7 جم\لتر) ولكن المرحله الاولى (94.8±17.9 جم\لتر) والثانيه (42.4±7.0 جم\لتر) كانت مرتفعه مقارنه مع الابقار الغير حُمل (39.3±4.1 جم\لتر).

مستوى الجلكوز منخفض فى المرحله الاولى والثانيه من الحمل (4.8±0.2 , 4.1±0.3 ملمول\لتر) على التوالى اما فى المرحله الثالثه كان مرتفع (4.9±0.7 ملمول\لتر) مقارنه مع الابقار الغير حُمل (4.3±0.4ملمول\لتر) .

مستوى الكالسيوم (2.4±0.6 ملمول\لتر) فى المرحله الاولى من الحمل و (1.6±0.3 ملمول\لتر) فى المرحله الثانيه من الحمل وفى هاتين المرحلتين كان منخفضا مقارنه مع الابقار الغير حُمل (3.2±1.2ملمول\لتر) اما فى المرحله الثالثه من الحمل كان مرتفعا (3.8±0.9 ملمول\ لتر) . الاداره الجيده للابقار الحُمل قبل وبعد الولاده مع العلاج مهم فى التحكم فى امراض الانتاج.

Introduction

Remarkable changes have occurred in dairy cattle farming in the last 35 years and new development continued to occur . Some of the obvious changes in dairying globally include the reduction in the size of the national dairy herd , increased annual production per cow , reduced number of dairy farms , increased size of dairy farms , and specialization , increased purchase of farm inputs, changed transportation pattern , and changes in the social and economic characteristics of dairy farmer (Crowley and Niedermerng , 1981). When the milk yield increase ; pathological phenomena becomemore manifested in form of metabolic profile and in organs such as the uterus the ovaries and the udder. Application of metabolic profile and nutritional deficiency disease are important (Payne *et al* , 1973) . Enumeration of the production disease such as parturient paresis , hypomagnesemia , acetonemia , all these diseases are attributed to imbalance between the rates of input of production calcium , phosphorus , magnesium and iron are the most important micro-elements that should

be included in the cow and calf diet (Radostits*et al* , 2000) .These elements are essential factors in formation of normal bone , teeth , muscle contraction , and energy metabolism (National Research Council , 1980 , Linn , *et al* , 1989) .

The control of health management to explore methods of predicting the occurrence of metabolic disease in advance monitoring of certain components of the blood on regular bases should be considered (Radostits*et al* ,2007).

Objective :-

1- To evaluate certain hematological parameters of pregnant cows.

2- To determine certain biochemical parameters of pregnant cows .

3- To prevent dairy cattle within the pregnancy from production disease .

Chapter One

Literature Review

1.1.Definition of Production Diseases In Cattle :-

These are diseases that characterized by imbalance between the rate of input of dietary nutrients and the output of production . The most important metabolic disease that were recorded in cattle are parturient paresis , acetonemia , downer cow syndrome , and acute hypokalemia ….act.

The Parturient paresis is defined as a disease around the time of parturition and is caused by decreasing of calcium , characterized by weakness , recumbency and ultimately shock and death(Radostits *et at*,2000).

David (2007) reported that milk fever is a disorder mainly of dairy cows close to calving. It is a metabolic disease caused by a low blood calcium level (hypocalcaemia). Between 3% and 10% of cows in dairying districts are affected each year, with much higher percentages occurring on some properties. Jersey cows that are mature and fat and graze lush, clover dominant pasture before calving are most susceptible.

Parturient paresis is an acute to peracute, afebrile, flaccid paralysis of mature dairy cows that occurs most commonly at or soon after parturition. It is manifestedby

changes in mentation, generalized paresis, and circulatory collapse (Barrington,2011)

Laurent and Alexander(2007) recorded that ,a downer cow is an animal that is unable to rise to a standing position . Downer cows are a common presentation in the periparturient period. They can be divided into 3 categories: (a) cows that are unresponsive to standard hypocalcaemic or milk fever therapy and do not exhibit other complications but remain alert , (b) alert recumbent animals that have traumatic musculoskeletal and nerve problems,(c) and recumbent animals that are affected with systemic diseases related to metabolic, toxic, alimentary, or neurologic conditions .

Strictly, the downer cow is defined as animal has been down for longer than 24 hours, but is not suffering from milk fever, in sternal recumbency, there is no obvious cause and is usually related to calving(Huxley ,2006).

Nicky (2007) reported that , very distinct problem for dairy cows is the disease of ketosis (acetonaemia). The occurrence of this disease in dairy cows is related to an increased demand for glucose by the animal. Ketosis also occurs in other animals and the problem is known by various names, e.g, pregnancy toxaemia in ewes. Most commonly, ketosis is seen either in high producing cows or cows on a poor diet. Signs of the disease can be seen before calving, but they occur most commonly in the first month after calving and occasionally in the second month. In

a herd, ketosis can either be sporadic with only individuals affected, or endemic with many cows affected over a period.

Anderson and Ewalt (1970) stated that, Ketosis is a metabolic disorder in which something goes wrong with the normal body processes and the cow becomes sick. There are no inflammatory organisms involved and the condition is not contagious. There seems to be a situation where the cow is temporarily producing more milk and thus requiring more feed nutrients than her feed intake provides. Nature may attempt to correct this situation by using body reserves of fat. When this occurs, some intermediary products of fat metabolism, called "ketone bodies," may build up in the system. These can be detected in the milk and urine and are indications of how serious the condition may be. Ketosis may even develop in average-producing animals when the energy needs exceed the energy intake.

1.2.Aetiological agents leading to production diseases in cattle:-

The main etiological agents that causes the milk fever is depression of the levels of ionized calcium in tissue fluids at a basic biochemical defect . A transient period of hypocalcaemia occurs at the onset of lactation caused by an imbalance between calcium out put in the colostrums and influx of calcium to the extracellular pool from intestine and bone (Radostits*et al* , 2000).

David (2007), recorded that when the amount of calcium in the diet is greater than is needed, the efficiency of absorbing calcium from the intestine and the efficiency of transferring calcium from the skeleton both become very sluggish and the chance of milk fever is greatly increased.Fat cows are at a greater risk than thin cows because calcium intake has been higher and partly because fat cows produce more milk at calving time.Some cows get milk fever several days or even weeks before or after calving. This is usually due to the feed, especially the dietary calcium, being insufficient to meet the heavy demand due to the rapidly growing foetus or milk production in early lactation and also .cows should receive as much calcium as possible, and clover-dominant pasture are therefore desirable. They will help to prevent grass tetany as well as milk fever .

Amy and Dave (2010) reported that the cause of Milk fever usually occurs within 72 hours of calving and is caused by a low blood calcium level. Increased risk occurs with cold, wet conditions; poor nutrition prior to calving age and breed .Milk fever has also been associated with uterine prolapse, 53 cows with uterine prolapse had significantly lower serum calcium concentrations than 53 matched cows (Risco*et al* ,1984). Multiparous cows with uterine prolapse were more likely to be hypocalcemic than primiparous cows with uterine prolapse. Delayed cervical and uterine involution during hypocalcemia may explain why hypocalcemia is associated with uterine prolapse (Odegaard , 1977; Risco*et al* ,1984).

Milk fever has also been qualitatively linked with increased incidence of cystic ovarian disease (Archbald*et al* ,1992). No direct mechanism for this link has been established. Cystic ovarian disease may be associated with any episode of peri parturient disease.

Barrington (2011) stated that the time near of parturition, and the onset of lactation results in sudden loss of calcium into colostrum and milk. Serum calcium levels decline from a normal of 8.5–10 mg/dL to 2–7 mg/dL. Commonly, serum phosphorus is decreased, and cows are hyperglycemic. The disease may be seen in cows of any age but is most common in high-producing dairy cows entering their third or greater lactation. Incidence is higher in the Jersey breed.

Most commonly, the downer cow is a complication of milk fever. Ischemic necrosis of the large muscles of the pelvic limbs and injuries to the tissues around the hip joint and of the obturator muscles are common in cows which do not fully recover and stand but remain recumbent following treatment for milk fever (Radostits*et al* , 2007). Injuries of the musculoskeletal system are also common as a result of cows' spreadeagling' their hindlimbs if they are unsteady during parturition or forced to stand or walk on a slippery floor immediately before or following parturition. Dystocia due to an oversized calf may result in extensive edema of the pelvic tissues and vulva, and failure of the cow to stand following

parturition. If these cows develop milk fever, it is unlikely they will be able stand following treatment with calcium(Radostits*et al* ,2007).

There is no universal cause of the downer cow syndrome, but it is most frequently a sequela to milk fever where complications, such as muscle necrosis and nerve paralysis, can arise because of delayed or insufficient calcium (Ca) replacement. Additionally, secondary metabolic disorders involving phosphorus (P), magnesium (Mg), and potassium (K) deficits have been suggested as risk factors, but without direct evidence of their involvement (Laurent and Alexander ,2007).

Joachim (2012) recorded that in most cases, downer cow syndrome is a complication of periparturienthypocalcemia (milk fever) ,in cows that do not fully respond to calcium therapy. Calving paralysis after dystocia may also result in recumbency due to traumatic injury to tissues and nerves inside the pelvic cavity. Regardless of the initial cause of recumbency, all cattle develop pressure-induced damage to muscles and nerves of the pelvic limbs, especially when lying on a hard surface. The hindlimb muscles of the leg the animal is lying on are compressed between the bones and the skin by the physical pressure from the weight of the recumbent cow.

The main cause of acetonemia(Bovine ketosis) is a multifactorial disorder of energy metabolism. Negative energy results in hypoglycemia and ketonemia the

accumulation in blood of acetoacetate, P-hydroxybutyrate and their decarboxylation products acetone and isopropanol (Radostits*et al* ,2007).

Disease occurs most frequently in early lactation and may be associated with other problems, such as fat cow syndrome, retained placenta, mastitis, metritis, and displaced abomasum. Ketosis-positive cows always should be examined for these other complicating factors (Smith,2007).

Nicky (2007) recorded that in late pregnancy, glucose is directed from normal bodily functions to the nutrition of the developing calf. As lactation starts, glucose is essential for the formation of lactose (milk sugar) and milk fat. The requirement for glucose is at such high levels that the blood becomes low in glucose (hypoglycaemia). Fifty grams of glucose is required for each litre of milk with a 4.8% lactose test and 30 grams for each litre of milk with a 4% fat test. Cows and other ruminants cannot be fed glucose in their diet; it has to be made in the rumen from suitable carbohydrates in the diet.If the amount of suitable carbohydrate in the diet is not enough to meet the glucose needs of the cow in full milk, the liver starts to manufacture glucose from other basic compounds in the body - usually fat reserves.Unfortunately the increased production by the liver also gives rise to undesirable by-products called ketones. These, together with the lack of blood sugar, cause the signs seen with the disease.

1.3.Epidemiologyof Production Diseases In Cattle :-

The epidemiology of the milk fever is the disease occurs most commonly in high-producing adult lactating dairy cattle, and commonly affected cattle are in the 5-10-year age group, although rare cases have been observed at the first and second calvings The hypocalcemia at calving is also age related and most marked in cows at their 3rd to 7th parturition, it is infrequent at the first parturition,Lactating beef cows are affected but less commonly(Radostits*et al* , 2007).

The average annual incidence of clinical milk fever for the period between 1998 to 2002 in British dairy herds has been reported as 5% (Whitaker *et al*,2004). The incidence is higher in dairy cows than beef cows and increases with yield. In some years, the incidence in September and October in the UK can be as high as 60% on some farms. During the winter months, when dry cows are housed, this figure can drop to 0-6%. Hypocalcaemia can potentially have widespread effects on the cow that predispose her to other metabolic and peri-parturient diseases (Goff, 2003). Cows with milk fever have a higher plasma cortisol concentration which may exacerbate the immunosuppression normal at calving. Additionally, the loss of muscle tone in the teat sphincter and the uterus may increase the risk of mastitis and uterine prolapse.

A reduction in appetite may exacerbate the negative energy balance after calving and increase the risk of ketosis and displaced abomasum, all of which can impact

on subsequent fertility in later lactation (Chebel*et al* 2004; Maizon*et al* 2004).

There are differences in susceptibility between the breeds but the differences are small. In cattle, milk fever occurs at three main stages in the lactation cycle. Most prepartum cases occur in the last few days of pregnancy and during parturition but rare cases occur several weeks before calving.

The morbidity and case fatality in the milk fever there are several epidemiological studies of milk fever have shown an incidence risk of5-10%, calculated either as the lactational incidence or incidence per cow year. Generally the disease is sporadic but on individual tarms the incidence may rarely reach 25-30% of high-risk cows (Radostits*et al* , 2007).

The epidemiology of the downer cow syndrome occurs most commonly within the first 2 or 3 days after calving in high-producing dairy cows immediately following milk fever. Cattle may also become persistently recumbent for many reasons other than complications of milk fever such as per acute coliform mastitis and carbohydrate engorgement. Downer cows can be divided generally into non-ambulatory cows with non progressive neurological findings and non-ambulatory with progressive neurological findings indicative of the presence of lesions in the nervous system as the cause of the recumbency (Radostits*et al* , 2007).

Animal risk factors is complication of milk fever. Prolonged recumbency after a long delay in the treatment of milk fever is a major risk factor. Prolonged

recumbency before treatment for milk fever (more than 4-6 h) results in ischemic necrosis due to obstruction of the blood supply, especially in a heavy cow if she lies on one leg for a long period (Radostits*et al* , 2007).

Environmental and management risk factors as slippery ground surface is a major risk factor. Cattle which must walk across slippery floors, especially at the time of calving, may slip and fall and injurey the large muscles of the pelvic limbs, resulting in an inability to stand. Prolonged recumbency results in ischemic necrosis and downer cow syndrom (Radostits*et al* , 2007).

Radostits*et al* (2007) reported that the epidemiology of Ketosis prevalent in most countries where intensive farming is practiced. It occurs mainly in animals housed during the winter and spring months and is rare in cows that calves on pasture. In housed or free-stalled cattle it occurs yearly around. The occurrence of the disease is very much dependent upon management and nutrition and varies between herds.

Oetzel (2007) recorded that since the late 1990's ketosis has emerged as the most important metabolic disease in dairy herds in the US, surpassing ruminal acidosis and milk fever in clinical significance. Fortunately, new testing methods have emerged during the same time period that now allow veterinarians to diagnose ketosis on a herd basis.

Herds with ketosis problems in early lactation cows also tend to have increased incidence of displaced abomasum (>8%) and increased herd removals in the first

60 days in milk (<8%). Affected herds may also have a higher proportion (>40%) of cows with milk fat to true protein percentages below 0.70 at first test after calving (Duffield and Bagg, 2002). However, none of these clinical findings are definitive evidence for a ketosis problem in a herd. A quantitative evaluation of the prevalence of ketosis is extremely useful in most dairy herds (Oetzel ,2007).

There are conflicting reports on the significance of risk factors for ketosis and subclinical ketosis which probably reflect that the disease can be a cause or effect of interacting factors. The disease occurs in the immediate postparturient period with 90% of cases occurring in the first 60 days of lactation. The age also affected in the risk of ketosis , cows of any age may be affected but the disease increases from a low prevalence at the first calving to a peak at the fourth. The herd differences in prevalence are very evident in clinical practice, and in the literature, with some herds having negligible occurrence. Although apparent difefrences in breed incidence are reported, evidence for an heritable predisposition within breeds is minimaI. Feeding frequency has an effect with the prevalence much lower in herds that feed TMR and libitum compared with herds that fed roughage and concentrate separately of that feed twice a day. There is no clear association with season. In some but not all summer grazing areas, a higher risk is generally observed in cattle during the winter housing period. There is a greater risk for the development of ketosis in cows that have an extended long dry period, that develop

milk fever, retained placenta, lameness or hypomagnesemia . There are Economic significance affected ketosis , the clinical and subclinical ketosis are major causes of loss to the dairy farmer. In rare instances the disease is irreversible and the affected animal dies but the main economic loss is due to the loss of production while the disease is present, the possible failure to return to full production after recovery and the increased occurrence of peri parturient disease. Both clinical and subclinical ketosis are accompanied by decreased milk yields and lower milk protein and milk lactose1 and increased risk for delayed estrus and lower first service conception rates, increased inter-calving intervals and increased risk of cystic ovarian disease, metritis and mastitis and increased involuntary culling (Radostits*et al* , 2007).

1.4.Diagnosis of production diseases in cattle :-

1.4.1 Clinical Manifestations :-

Smith (2007) reported the signs of milk fever include staggering, inability to rise, muscular weakness, recumbency (laying down) and a subnormal temperature. The following are other problems that may occur due to milk fever, difficult calving due to muscular weakness that prevents proper labor, Increased chance of uterine prolapse, tendencies to increase retained placentas, increased possibility of metritis (uterine infection),decreased reproductive performance, increased tendencies to bloat due to rumen muscle tone loss (atony), greater numbers of

abomasal displacements ,much greater risk of ketosis, considerably greater risk of developing mastitis ,greater risk of other infectious disease, decreased milk production ,reduced total productive life in the herd.

Amy and Dave(2010) reported that the symptoms include unsteady gait, cow lying down with head displaced to one side or into the flank , depression, staringes, pupils dilated, dry muzzle, cold ears, groaning, slight muscle spasms, inability to rise, coma and, if the animal is untreated can be death.

Barrington (2011) recorded that parturient paresis usually occurs within 72 hours of parturition. The disease can contribute to dystocia, uterine prolapse, retained fetal membranes,metritis,abomasal displacement, and mastitis.There are 3 discernible stages of parturient paresis. During stage 1, animals are ambulatory but show signs of hypersensitivity and excitability. Cows may be mildly ataxic, have fine tremors over the flanks and triceps, and display ear twitching and head bobbing. Cows may appear restless, shuffling their rear feet and bellowing. If calcium therapy is not instituted, cows will likely progress to the second, more severe stage.Cows in stage 2 are unable to stand but can maintain sternal recumbency. Cows are obtunded, anorectic, and have a dry muzzle, subnormal body temperature, and cold extremities. Auscultation reveals tachycardia and decreased intensity of heart sounds. Peripheral pulses are weak. Smooth muscle paralysis leads to GI stasis, which can manifest as bloat, failure to defecate, and

loss of anal sphincter tone. An inability to urinate may manifest as a distended bladder on rectal examination. Cows often tuck their heads into their flanks, or if the head is extended, an S-shaped curve to the neck may be noted .In stage 3, cows lose consciousness progressively to the point of coma. They are unable to maintain sternal recumbency, have complete muscle flaccidity, are unresponsive to stimuli, and can suffer severe bloat. As cardiac output worsens, heart rate can approach 120 bpm, and peripheral pulses may be undetectable. If the animal is untreated, cows in stage 3 may survive only a few hours.

David (2007) stated typical cases that show some initial excitement or agitation and a tremor in muscles of the head and limbs. Then they stagger and go down to a "sitting" position, often with a 'kink' in her neck, and finally lie flat on their side before circulatory collapse, coma and death. A dry muzzle, staring eyes, cold legs and ears, constipation and drowsiness are seen after going down. The heart beat becomes weaker and faster. The body temperature falls below normal, especially in cold, wet, windy weather.These signs are due mainly to lowered blood calcium levels. Sometimes there are additional signs due to complicating factors. Bloat is common in cows unable to "sit up" because the gas in the rumen is unable to escape. Pneumonia may affect cows left out in bad weather.

Radostits*et al*(2007) stated that downer cow syndrome may occur independently,

or follow apparent recovery after treatment for milk fever, except for the prolonged recumbency. In the typical case, affected cows either make no effort or are unable to stand following treatment for parturient paresis. About 30% of cows treated for milk fever will not stand for up to 24 hours following treatment.

An alert downer cow does not show signs of systemic illness or depression, is able to eat and drink, and remains in sternal recumbency for no apparent reason. Anonalert downer cow appears systemically sick and depressed (Joachim ,2012).

The animals are usually bright and alert and, although the appetite is reduced, the cow eats and drinks moderately well. The temperature is normal and the heart rate may be normal or elevated to 80-100 bpm. Tachycardia and arrhythmia occur in some cows, especially immediately following the administration of calcium intra venous and sudden death has occurred, respirations are usually unaffected. Defecation and urination are normal but proteinuria is common and if marked may indicate extensive muscle damage .Some affected cows may make no effort to stand. Others will make frequent attempts to stand but unable to fully are extend their pelvic limbs and lift their hindquarters more than 20-30 cm from the ground. In some cases, the hind limbs are extended on each side of the cow and reach up to the elbows on each side. In this position, the cow is bearing considerable weight on the medial thigh musculature and causing ischemic necrosis. This abnormal position of the legs may also be due to dislocation of one or both hip joints or

associated with traumatic injuries surrounding the hip joints with or without rupture of the ligamentumteres. Regardless of the cause, the cow prefers this leg position and invariably will shift the legs back to the abnormal position if they are placed in their normal position .In some cows, the signs may be more marked and bizarre, including a tendency to lie in lateral recumbency with the head drawn back . When placed and propped up in sternal recumbency, these cows appear almost normal but, when they are left alone, within a short period of time they revert to the position of lateral recumbency . Still more severe cases are hyperesthetic and the limbs may be slightly stiff but only when the cow is lying in lateral recumbency. These severe cases do not usually eat or drink, have been described as 'non-alert downers', and are thought to have brain damage which has not been documented. Complications in the downer cow syndrome are common and often result in death or the need for euthanasia. Coliform mastitis, decubitus ulceration, especially over the prominences of the hock and elbow joint, and traumatic injuries around the tuber coxae caused by the hip slings are common. When these complications occur in the early stages of the disease, they commonly interfere with any progress being made and become the focus of clinical attention. Death may occur in 48-72 h following the onset and is usually associated with myocarditis (Radostits*et al*, 2007) .

Nicky (2007) recorded that common signs of ketosis in dairy cattle arecharacterized in the two major forms , the wasting and nervous forms. 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 objects, including dirt and stones. Consequently, milk yield falls quickly to a fraction of its initial level, but never ceases completely. By this stage the affected animal is obviously ill and is disinclined to move, may be staggery or unsteady on its legs, and the head is often carried low to the ground.Temperature, pulse, and respiration rates of the cow remain fairly normal as the animal loses weight. The coat is described as having a "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 cow's breath and less commonly in milk samples. Very few affected animals die but, without treatment, recovery is slow with milk yields gradually improving over one month but never fully returning to normal levels.This form can be very mild, the only clue to its presence being a small reduction in milk production.The nervous form of ketosis is less common. In typical cases the signs are quite striking. Affected cows can show a range of signs including apparent blindness, aimless wandering, and strange

movements of the 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.

Ketosis signs include “off-feed,” weight loss, decreased milk production, listlessness and other unusual signs (Smith, 2007). Symptoms include excessive loss of weight and condition, sweet smelling (acetone) breath, reduced intake, dullness, depression, staring expression, reduced milk production, constipation, mucous-covered faeces , lack of coordination, partial paralysis, highly excitable, licking, teeth grinding, shallow breathing(Amy and Dave,2010) ,in case of Subclinical ketosis many cows that are in negative energy balance in early pregnancy will have ketonuria without showing clinical signs, but will have diminished productivity inciuding depression of milk yield and a reduction in fertility. Clinical diagnosis is not effective and in one study, 22 diagnosis by routine urine testing at 5-12 days post partum was considerably more efficient (15.6% detected) than diagnosis by the herdsman (4.35% detected)(Radostits*et al* ,2007).

Anderson and Ewalt (1970) reported that,the first symptom of ketosis is a loss of appetite first for grain and then for silage, followed by a lack of interest in any

feed. There appear to be two types of reaction. 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. All this is followed by a drop in milk production. The breath may have a noticeable "sweet" odor due to the presence of acetone. Ketosis seems to occur more of ten in older animals, and some animals have a tendency to repeat. Ketosis is seldom a direct cause of death.

1.4.2. Clinical pathology:-

In milk fever the total serum calcium levels are reduced to below 8 mg per dl (2.0 mmol /l) , usually to below 5 mg (1.2 mmol/l) and some times to aslow as 2 mg (0.5 mmol/l). the reduction is usually , but not always proportional to the severity of the clinical blood levels of ionized calcium are abetter indicator of calcium statous but their estimation has been too difficult until recently .Normal levels of ionized calcium as (CaF) in venous whole blood of cows are 4.3_5.1 mg/dl and 1.06-1.26 mmol/l in serum . slighthypocalcemia the range between 4.2-3.2mg/dl in blood and 1.05-0.80mmol/l in serum . moderate 3.2-2.0 mg/dl in blood and 0.79-0.50 mmol/l in serum , and severe hypocalcimia less than 2mg/dl in blood , less than 0.30mmol/l in serum. Total serum calcium levels are reduced below normal in all cows at calving whether they have milk fever or not , Serum magnesium levels are usually moderately elevated to 4-3mg/dl (1.65-2.06 mmol/l) , but in some areas low levels may be encountered especially in cows at pasture .

Serum morgonic phosphorus levels are usually depressed to 1.5-3.0 mg/dl (0.48-0.97mmol/l).Blood glucose levels are ususlly normal although they may be depressed if ketosis occurs concurrently higher than normal blood glucose levels are likely to occur in cases of long duration and are therefore an indication of a poor than normal prognosis . Changes in the leukocyte count include an eosinopenia , aneutrophilia and lymphpenia suggestive of adrenal cortical hyperactivity but similar changes occur at calving in cow which don't develop parturient paresis high . plasma cortisol levels and packed cell volume occur in cows with milk fever , and are higher still in cows that do not respond to treatment they are expressions of stress and dehydration (Radostits*et al* ,2000).A normal plasma concentration of calcium is in the range 2.2-2.6 mmol/l (8.8-10.4 mg/100 ml). In many cows (between 30-40% of cows) this will fall below 2.0 mmol/l (6.0 mg/100 ml) at partuition (Roche, 2003).The measurement ofthe total serum calcium concentration which is comprised of the sum of ionized 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 (Radostits*et al* ,2007). Practically this is not required due to the correlation between the concentrations of ionized and total sera (Lincoln 1990 ; Hunt and Blackwelder 2002). In the last decade, therehave been many reports of diagnosis of milk fever

using total serum total calcium level in cows (Kajouri 2003 ; Sakna and Jamshidian 2003 ; Zadnik*etal* , 2006). Reducing the number of absorbable dietary cations and/or increasing the number of absorbable dietary anionsgreatly diminishthe incidence of hypocalcemia and milk fever in dairy cows.

In downer cow syndrome(Radostits*et al*,2007) recorded that the calcium, phosphorus, magnesium and glucose levels of the blood are within the normal range and the results of hematological examinations are usually consistent with those found in normal ,cows which have recently calved. The CPK and AST levels are usually markedly elevated by 18-24 hours after the onset of recumbency and continue to elevate within the next few days. Continued elevation of CPK levels indicates continued muscle damage. In experimentally induced recumbency in cows, the CPK levels remained within normal limits for the first 6 hours. However, by 12 hours there was a marked increase to mean values 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. The proteinuria may persist for several days or be absent within a few days . In severe cases, the urinemay be brown and turbid because of severe myoglobinuria. Low arterial blood pressures and abnormal electrocardiograms (ECGS) have been observed in some animals.Elevations of serum urea, muscle enzymes, and laboratory evidence of inflammation are considered the best prognostic indicators of an unfavorable

recovery. The recovery rate was lower in cows with a total protein fibrinogen ratio less than 10:1, and evidence of neutropenia and/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. The CPK levels need to be interpreted in relation to the days of recumbency when the sample was taken. Critical levels may be highest initially (up to50 times the upper normal reference range) and reduce to 10 times normal range at7 days of recumbency.

Low milk production and prolonged calving intervals also promote FCS. Feeding excessive quantities of concentrates after peak lactation or during the dry period, combined with free-choice feeding of corn silage or high-quality hay, predisposes a herd to development of FCS (Morrow 1976 ; Morrow *etal* ,1979). Likewise, either underfeeding of protein or an early decrease in milk production, combined with free-choice feeding of corn silage, contributes to excessive intake of energy and over conditioning of cows . In support of these findings, cows that were overconditioned during the dry period and fed a ration of 15% crude protein had a 69.4% incidence of metabolic diseases and a 31% incidence of the alert downer cows. In contrast, dry cows fed only an 8% crude protein ration had a 7% metabolic disease incidence and no alert downers.Cows affected with FCS may develop leukopenia and decreased hepatic function (Morrow *et al* , 1979).

Low cholesterol concentration in serum (less than 100 rag/100 ml) 8week before calving was associated with a 70% incidence of FCS in a German study (Sommer 1975).

The clinical pathology of ketosis ,hypoglycemia, ketonemia and ketonuria are characteristic by, blood glucose levels are reduced from the normal of approximately 50 mg/dL to20-40 mg/dL. Ketosis secondary to other diseases is usually accompanied by blood glucose levels above 40 mg/dL and often above 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. Plasma concentrations of BHBA significantly correlate with plasma concentrations of acetoacetate but acetoacetate is unstable in samples whereas BHBA is relatively stable. Normal cows have plasma BHBA concentrations less than 1000 μmol/L, cows with subclinical ketosis have concentrations greater than 1400μmol/L, and cows with clinical ketosis have concentrations often in excess of 2500 μmol/L. Plasma BHBA shows some diurnal variation in cows fed twice daily with peak concentrations occurring approximately 4 hours after feeding and higher concentrations in the morning than in the afternoon. This is not seen in cows fed a total mixed ration and libitum .Plasma BHBA is not a cost effective or convenient analysis for routine analysis and cow side monitoring and the content of acetoacetate or BHBA in urine and

milk are used for these purposes. Concentrations of BHBA and acetoacetate in urine and milk are less than those in blood and the correlation coefficients for blood and milk BHBA and blood and milk acetoacetate are 0.66 and 0.62, respectively. 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 on 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 negatives 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. Conventional wisdom is that milk powder tests are not sensitive for detection of subclinical ketosis (report too many false negatives) and urine tests are not sufficiently specific (report too many false positives. The sensitivity and specificity of the nitroprusside powder test with milk in various studies is reported as28-90% and 96-100%, respectively. More recently, a milk strip test detecting the presence of

BHBA in milk is available and is graded on the concentration of BHBA inμmol/L. In different studies it has a reported sensitivity and specificity of73-96% and 69-96%, respectively. These variations are, in part, due to different plasma BHBA reference values (1200 and 1400 μmollL) for designation of subclinical ketosis and different cut points used in urine BHBA. Somatic cell counts greater than 1 million cells/mL will cause an elevation in reading of both the BHBA strip test and the nitroprusside tests,nitroprusside tablet has a reported sensitivity and specificity of 100% and 59%, respectively, compared with serum BHBA concentrations above1400μmollL16 and a nitroprusside strip test a reported sensitivity and specificity of 78% and 96% with a urine cut point corresponding to 'small' on the color chart or 49% and 99% with a urine cut point corresponding to 'moderate' on the color chart.53 BHBA test strips when used with urine has a reported sensitivity and specificity of 73% and 96%, respectively at a urine cut point of 100 μmollL BHBA and27% and 99% at a urine cut point of200 μmollLBHBA.One author has suggested that the nitroprusside urine strip test or the BHBA milk strip test are best for screening individual cows for ketosis in herds with average prevalence but that the nitroprusside powder test would have limited application (Radostits*et al*,2007).

The dairy cattle affected ketosis when the glucose level below 1.94mmol/l (Kelly 1964).

One of the major changes in blood components is a decrease in glucose.Normal levels in ruminants are about 50 mg per 100 ml. values below 40 can be considered subnormal. Ketotic animals may have levels aslow as 25. The glucose decrease is 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. Although there is presumably increased gluconeogenesis from body protein this is not sufficient to maintain a normal blood level.A second major blood change is an increase in ketone bodies, already mentioned in relation to diagnosis. Following previous suggestions, normal levels would be considered something less than 10 mg %. Ketotic animals may have levels as high as 50. In nonketotic animals, themain source of these ketone bodies appears to be butyric acid from the rumen, with conversion to ketone bodies occurring in the rumen wall or liver .

As the condition progresses, the cow is forced to mobilize body fat, resulting in an increase in plasma free fatty acids (FFA). Normal levels are something less than 10 mg %. Ketoticanimals may have levels as high as 50. They are carried in the plasma as an albumin complex. Under fed conditions, correlations between glucose and FFA or ketones and FFA are low (Adler *etal* , 1963 ; Rodloffand Hoekstra 1960) . However, under fasting conditions,or in cows positive to the milk test for ketosis, there is a significant negative correlation between glucose and FFA as well as between glucose and ketones, with a significant positive correlation between

FFA and ketones . It seems clear that under fasting or ketotic conditions, FFA become an important source of ketones. This conversion takes place mainly inthe liver. The negative correlation between blood glucose and both FFA and ketones in early ketosis suggests that the low availability of glucose is an important factor in the development of the condition. There is evidence in goats that a glucose-drain condition imposed by phlorizin causes increased conversion of butyric acid to ketone bodies in the rumen wall, as well as increased mobilization of FFA, with their subsequent conversion to ketone bodies in the liver (Menahan*et al* , 1966). There is also evidence that the mammary gland produces acetoacetate under ketotic conditions (Kronfeld 1966), but the origin is not clear. The significance of a fourth change in the blood of cows in early ketosis, a small but significant decrease in triglyeerides (TG), is not apparent. Levels in cows positive to the milk test were about 15 mg % compared to 20 mg % when negative (Rodloff and Scholtz 1967). High FFA levels are generally considered to result in an increase rather than a decrease in TG (Steirberg 1963).

Illustrates the results of the effects of season and stage of pregnancy on the concentrations of plasma glucose and serum organic constituents. Winter data indicate that there was a non-significant decrease in plasma glucose during late pregnancy(Ahmed and Abdalla 2012).

1.5. Treatment and Prevention of Production diseases in cattle :-

David and Hamilton (2007) stated that the treatment of milk fever should be given as soon as possible, using of 300 ml, or more, of a 40% solution of calcium borogluconate or, preferably, a combined mineral solution such as "three-in-one" or "four-in-one", often 600ml may be required.The combined solutions contain additional ingredients such as magnesium, phosphorus and dextrose (for energy), which may also be at low levels in the blood while cows have milk fever.Packets of solution together with an injection kit are best kept on hand for emergencies. All equipment should be kept sterile to avoid abscess formation at the site of injection.Injection of the solution by farmers should be in several places under the skin on the neck or behind the shoulder, unless the cow is in a coma or there are other reasons for desiring a quick response.Injection into a vein should be left to a veterinarian as it can cause sudden death if not carried out properly. Veterinary assistance is also advisable if there is not a quick response to treatment, because other problems may also be present.Cows that are "flat out" should be propped up into a normal resting position to relieve bloat. If weather conditions are bad, or the response to treatment is slow, transfer the cows to shelter to prevent exposure and other complications and Provided feed and water. Some cows that have been comatosed may have regurgitated and inhaled rumen content into the lungs. If there is ruminal material around the nose one should be suspicious that this may have happened and intensive antibiotic treatment should be commenced as soon as

possible as inhalation pneumonia is often fatal.Recovered cows should not be milked for 24 hours; then the amount of milk taken should be gradually increased over the next 2-3 days.

Barrington(2011) said that the treatment is directed toward restoring normal serum calcium levels as soon as possible to avoid muscle and nerve damage and recumbency. Recommended treatment is intravenous (IV) injection of a calcium gluconate salt, 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 into the circulation.Subcutenious calcium treatment alone may not be adequately absorbed because of poor peripheral perfusion and should not be the sole route of therapy. No matter what route is used, strict asepsis should be used to lessen the chance of infection at the injection site. Solutions containing formaldehyde or >25 g dextrose/500 mL are irritating if given (SC). Manysolutions contain phosphorus and magnesium in addition to calcium. Although administration of phosphorus and magnesium is not usually necessary in uncomplicated parturient paresis, detrimental effects of their use have not been reported. Magnesium may protect against myocardial irritation caused by the administration of calcium. Magnesium is also necessary for appropriate

parathyroid hormone (PTH) secretion and activity in response to hypocalcemia. Most products available to veterinarians contain phosphate salts as the source of phosphorus. However, phosphorus found in blood and tissues of cattle is primarily in the form of the phosphate anion. Because no pathway exists for the conversion of phosphate to the usefull phosphate form, it is unlikely that these solutions are of any benefit in addressing hypophosphatemia. Administration of oral calcium avoids the risks of cardiotoxic side effects and may be useful in mild cases of parturient paresis; however, it is not recommended as the sole approach for clinical milk fever cases. Products containing calcium chloride are effective but can be caustic to oral and pharyngeal tissues, especially if used repeatedly. 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 tissues, 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.The treatment of milk fever include , promptly with slow intravenous or subcutaneous calcium borogluconate injection(Amy and Dave , 2010)Most cows with milk fever can be treated successfully with 8-10g of calcium (calcium borogluconate is8.3% 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 underdose with calcium salts, largely because of toxic effects which tend 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 a 25% solution and a small cow320-360 kg) 400-500 mL. Underdosing (increases the chances of incomplete response, with inability of the cow to rise, or of relapse. In general, 12 g of calcium is superior to 8 g, which in tum is superior to 6g (Radostits*et al*, 2007).

And prevent milk fever by stimulating cow's calcium mobilisation before calving, feed a transition diet three weeks to calving, include anionic salts, avoid feeds high in potassium sodium and calcium in the springer diet, increase dietary calcium immediately after calving, avoid fat cows at calving, administer vitamin D injections 2–8 days before calving for cows with a history of milk fever (Amy and Dave , 2010) .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.

The control of milk fever in the dairy cows is mainly by using Calcium metabolism of close-up dry cows and fresh cows is critical for reducing incidence of metabolic disorders. Anionic salts fed to the close-up group can reduce the incidence of milk fever, displaced abomasum, and subclinical hypocalcemia in early postpartum dairy cattle. Dairies experiencing a high incidence of milk fever

or displaced abomasum would likely benefit from use of anionic salts. In addition, dairies that feed high-potassium forages, such as alfalfa hay, to dry cows may benefit from supplementing with anionic salts. Anionic salts must be used with caution, however, because they are unpalatable and can reduce dry matter intake. They should only be fed to close-up dry cows. Controlled feeding, precise ration formulation using the DCAD concept, and monitoring of urine pH are necessary to achieve success using anionic salts (John , 1998).

Barrington (2011) recordedhistorically, prevention of parturient paresis has been approached by feeding low-calcium diets during the dry period. The negative calcium balance results in a minor decline in blood calcium concentrations. This stimulates PTH secretion, which in turn stimulates bone resorption and renal production of 1,25dihydroxy-vitamin D. Dihydroxyvitamin D increases bone calcium release and increases the efficiency of intestinal calcium absorption. Although mobilization of calcium is enhanced, it is now known that feeding low-calcium diets is not as effective as initially believed. Furthermore, on most dairy farms today, it is difficult to formulate diets that are low enough in calcium (<20 g absorbed calcium/cow/day), although the use of dietary straw and calcium-binding agents such as zeolite or vegetable oil may make this approach more useful.

Administration of vitamin D_3 and its metabolites is effective in preventing parturient paresis. 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 cow is more susceptible. Dosing for periods longer than those recommended should be avoided because of potential toxicity. A single injection (IV or SC) of 10 million IU of crystalline vitamin D given 8 days before calving is an effective . The dose is repeated if the cow does not calve on the due date. Newer compounds used (where available and approved) in lieu of vitamin D and less likely to cause hypervitaminosis include 25-hydroxycholecalciferol, 1,25-dihydroxycholecalciferol, and 1α-hydroxycholecalciferol. After calving, a diet high in calcium is required. Administering large doses of calcium in gel form (PO) is commonly practiced. Doses of 150 g of calcium gel are given 1 day before, the day of, and 1 day after calving. Use of synthetic bovine PTH may prove to be superior to administration of vitamin D metabolites. Vitamin D metabolites enhance GI calcium absorption, whereas PTH enhances GI calcium absorption and stimulates bone resorption. PTH is administered either IV 60 hours before parturition, or IM 6 days before parturition. Drawbacks to the use of PTH include increased labor requirements for administration, as well as the availability of such compounds.

Radostits*et al* (2007) reported that the treatment of downer cow syndrom depends on the prognosis of a downer cow and this depends on the cause of the recumbency and whether or not treatment is indicated or if euthanasia should be recommended

because of the presence of abnormalities which are unlikely to respond favorably to treatment and also be economical. If the prognosis is poor, euthanasia on the farm should be recommended. If the prognosis is favorable, the clinician should inform the owner about the nature of the treatment which will be necessary and its duration which may be several days of supportive care and therapy, and should outline the costs which will be incurred.

Huxley (2006) said that even if treatment of the initial cause of the downer cow is successful, if that cow does not rise straight away, then good nursing and management by the stockperson becomes essential for that cow's chances of ever getting up. The treatment acodeine to Huxley (2006) includes : Treat the initial cause promptly call in the veterinarian to identify the initial cause. Your vetrenarian should identify this on the initial visit supported if required by necessary laboratory tests (i.e. Ca, Mg, Ph, liver profiles, muscle profiles) and treat immediately. Identification of initial cause should also allow for prevention strategies to be put into place , milk fever → DCAB , toxic mastitis → dry cow therapy ,dystocia → appropriate bull selection , slips/falls → appropriate flooring . Nursing and management there is essential pointers of nursing the down cow until she rises. All these pointers aim to alleviate the symptoms of downer cow syndrome, this point is ,moved the cow to clean dry comfortable lying area ,Provision of adlib good quality feed and fresh water,turn at least every 3 hours,

milking ,hobbling hindlimbs ,Physiotherapy to help venous return and muscle perfusion each time the animal is turned, the limb that has been laid on should be vigorously massaged and manipulated (i.e. flexed and extended repeatedly) ,Anti-inflammatory drugs (NSAIDS) there action reduces the effects of downer cow syndrome, analgesic action reduces pain i.e. carprofen,flunixin, ketoprofen, meloxicam ,Encouragement to rise If she does not attempt to stand on her own, from time to time should be encouraged to rise by slapping and gently kneeing in the ribs . Assist of the animal back onto feet (if necessary) ,the animal may be lifted to aid diagnosis and prognosis and also to assist in nursing the down cow (i.e. massage of limbs, rebedding, milking). Methods of lifting include: Tail lift , Nets/slings/cradles/harnesses ,Bagshaw hoists ,Inflatable bags,Flotation tanks. Initial lifting should be done under supervision of the vet and the vet must ensure those involved in lifting the animal are fully competent in using the equipment available and considerate to the welfare of the cow. If used incorrectly all the above can do more harm than good to the cow. The animal should be lifted allowing her to find her feet then gently lowered to the point she either starts to bear weight or begins to collapse.

Joachim (2012)reported thatthe treatment is,assisting the cow to riseon every day of the recumbency, an attempt should be made to bring the cow to its feet. Several simple but effective techniques can be tried. In one method, the clinician stands

with feet pressed under the cow at a point below the scapulohumeral joint. A sharp blow is delivered by driving the knees into the muscle mass below and caudal to the 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 cause damage. Recently calved cows can be motivated to rise if they hear their own calf bawling with hunger. The calf is best restrained close to the cow but out of her sight. Some workers use electric goads and various anecdotal or traditional methods of inflicting pain to stimulate a cow to rise. These measures have a low success rate in inexperienced hands.

The value of hip clamps is controversial. Their proper use requires experience, skill, and a delicate touch. Continual causes of trauma and pain that is counterproductive. The forelimbs support 60% of a cow's weight and, therefore, the use of a canvas sling under the sternum is almost mandatory for consistent success. A chest band is required to prevent the sling from slipping backwards. If the sling is suspended from the tine at one end of a fork lift, and the hip clamps from a tine at the other end, minimal trauma results. If a fork lift is not available, a T-bar suspended by a pulley from an overhead beam (or a tripod for animals at pasture) will serve. The jaws of the clamps must be well protected with synthetic foam or rubber secured in place with a wrap of duct tape. Hip clamps should not be

applied too tightly and should lift the cow slowly to allow time for the circulation of the limbs to become reestablished. The device is lifted until the hindfeet just touch the ground. Often, the cow will hang with the limbs slightly flexed. This should not be confused with unilateral flexion, which indicates peroneal paralysis. Next, one assistant on each side of the cow presses a shoulder into the paralumbar fossa while facing the hindlimb. The device is slowly lowered as the assistants attempt to force each hindlimb into a weightbearing posture and to reduce the flexion by manipulating the stifle and hock. As soon as any weight is supported by the 2 limbs, the device should be lowered 1–2 inches . This process may have to be repeated several times .the other method of treatment is moving a recumbent cow ,the chances of resolution are considerably enhanced by moving the cow to a location with an earthen floor. In warm, relatively dry weather, the very best location for a 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. These conditions often exist in a hay barn or implement shed, which may have the added benefit of allowing installation of a pulley system to lift the cow.

Recumbent cattle should be examined daily to determine any change in ability to rise or bear weight. The chance of improvement is very low if the cow does not show any improvement within 5 days of moving to a location with good footing

and correction of any serum electrolyte abnormalities.Supportive Care of Recumbent Cows it is vital that recumbent cows be provided with clean water at all times. A shallow rubber feed bowl prevents spillage. If the cow does not drink, she must be given fluid therapy either by drench or parenterally. Every effort must be made to roll the cow from one side onto the other at least 3 times a day, with more frequent movement being desired. If this is not done, the weight of the cow results in continued ischemia of the muscles of the hindlimb and exacerbation of the compartmental syndrome.Protection from the elements is essential. Rain and wind can reduce body temperature considerably and worsen shock if present. A windbreak of straw bales is vital. Straw bedding should be provided to help insulate the cow from the ground. A recumbent cow does not require a warm environment; however, in a cold environment, an inactive animal can gradually succumb to hypothermia.The downer cows most difficult to treat are those that do not try to eat. A cow that salivates on its feed will not eat it later. Rather than being offered large amounts of feed, the cow should be tempted with sweet hay. This should be cleared away every 30 min if not accepted. Placing bitter-tasting weeds such as ivy or dandelion in the mouth may provoke salivation and an interest in eating. Lettuce and cabbage leaves are accepted by some cows. In extreme cases, the cow can be drenched with rumen contents (Joachim ,2012) .

Prevention by the early detection and treatment of milk fever will reduce the

incidence and severity of downer cow syndrome. Under ideal conditions, cows should be treated during the first stage of milk fever before they become recumbent. Once recumbent, cows should be treated as soon as possible and not delayed for more than 1 hours. Cows with milk fever should be well bedded with liberal quantities of straw, or moved to a soft-ground surface. Recumbent cows should be coaxed and assisted tostand if possible after treatment for milk fever. If they are unable to stand, they should be rolled from one side to the other every few hours if possible. It is usually difficult to get owners to comply with this recommendation but frequent rolling from side to side is necessary to minimize the ischemic necrosis. Dairy cows should be placed in a comfortable well-bedded box stall prior to calving and should be left in that box stall until at least 48 h after partition in the event that milk fever develops(Radostits*et al* ,2007).

The prevention by effective strategies to prevent milk fever are important to decrease downer cow syndrome. All dairy cows should be monitored closely around calving for early signs of parturient paresis . Prophylactic administration of calcium to all cows, beginning with cows entering their second or greater lactation, is beneficial in herds with a high incidence of milk fever, especially in smaller farms that cannot implement feeding acidogenic salt diets.The elapse of several hours from the commencement of clinical signs of milk fever until treatment seems to be the critical issue. Every cow that has been successfully treated for

hypocalcemia should, if necessary, be moved to a location with a good footing and remain there for 48 hr. Straw over sand provides good insulation and good footing (Joachim ,2012).

Ketosis is treated by useing the intra venous (I/V) 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 non -esterified fatty acids. and may use other sugars, especially fructose, either alone or as a mixture of glucose and fructose (invert sugar), and xylitol, have been used in an effort to prolong the response but idiosyncrasies to some preparations, in the form of polypnea, muscle tremor, weakness and collapse, can occur while the injection is being given. Propylene glycol and glycerine /glycerol to overcome the necessity for repeated injections, propylene glycol can be administered as a drench. The traditional does is225 g twice daily for 2 days, followed by110 g daily for 2 days to cattle, but higher volumes are also used. and also treated by hormonal therapy like Glucocorticoids, Insulin, Anabolic steroids all of this aid in treatment (Radostits*et al*, 2007) .

Anderson and Ewalt (1970) recorded that over the years at least 25 different treatments for ketosis have been suggested. The fact that some cows may recover spontaneously without treatment makes evaluation of the treatments difficult. The

present most widely used treatments include the injection of sugar (glucose) into the neck vein or under the skin, injection of hormones (cortisone or ACTH) , or oral administration of propylene glycol or sodium propionate. More recently, methionine deficiency has been considered, and injections of L-methionine have given good results in a limited number of cases. It is difficult to say that any one of these treatments is the best since each has its advantages and disadvantages. Often they are used together to supplement each other. The use of sugar in the neck vein gives temporary benefit, but the injected sugar is used up in a few hours and there may be frequent relapses which require retreatment. The hormone injections are sometimes, effective, but they are expensive and repeat treatments may be needed. The oral materials usually are given at the rate of 4 to 8 ounces night and morning for about 10 days. There is always some danger of getting the material in the lungwhen drenching also, this is not a one-shot treatment but must be given for several days. All of the treatments are an attempt to increase the blood sugar and thus reduce ketones and fat mobilization. The oral materials are absorbed from the rumen and intestines and converted to blood sugar in the liver. 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. The dairyman can add to the treatment by giving a little special care such as feeding the best quality hay, feeding more often, and making the cow as comfortable as possible.

Nicky (2007) stated that the initial aim of treatment is to restore the lack of glucose in the body,a quick-acting glucose supplement is required immediately. Follow-up treatment is aimed at providing a long term supply of glucose.Intravenous administration of a dextrose solution by a veterinarian is effective in the 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. Several commercial compounds contain propylene glycol and glycerine .Many of the long-acting corticosteroids have beneficial effects in ketosis. They are administered by the veterinarian as a single injection.Corticosteroids have the ability to break down protein in muscles to produce glucose, which immediately replenishes the depressed blood glucose levels.When using corticosteroids, it is important to supply an adequate amount of glucose either as a high carbohydrate diet and/or propylene glycol drenches to prevent excessive breakdown of muscle protein.

Amy and Dave (2010) said that the prevention with balanced ration, particularly energy intake; maximise dry matter intake before, at and after calving; adapt dry cows to milking-cow diet by lead feeding, avoid over fat cows at calving (BCS>6 out of 8), lower potassium and calcium levels in the dry cow and springer diet; provide niacin as it may be effective if fed at 6 grams per day, feed monens in the

last month before calving. Ketosis is best prevented by keeping cows in good condition, but not fat, during the dry period. Begin to “lead feed” grain 10-15 days prior to calving. Increase the grain ration about 1 pound per day up to a maximum level of 15 pounds. Feed changes during the first six weeks of lactation also should be gradual. During lactation, good quality, high energy, palatable feeds should be fed .

It is important to prevent ketosis from occurring, rather than treating cases as they appear.Prevention 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 carbohydrate is essential. The body condition of the dairy cow is important at calving. Cows should be on a rising plane of nutrition up to calving with the aim to calve in good condition (at least score 5 on theellinbank Condition Scoring System).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 fed at least until the peak of lactation is reached or longer depending on the quality and quantity of available pasture. Occasionally, very high

producing cows will be susceptible to ketosis every year. In these cases a preventive drenching program of propylene glycol immediately after calving may avert ketosis in individual problem cows(Nicky , 2007)

Anderson and Ewalt (1970) recorded that , obviously, prevention of ketosis would be more satisfactory than treatment if the methods involved were practical. At the present time no methods will insure 100 percent freedom from ketosis. Suggestions for reducing the incidence of ketosis can be made, however. Although there is disagreement regarding the basic cause of ketosis and the extent to which hormones are involved, most re- search workers agree that factors which would tend to balance feed intake with milk production without throwing the cow off feed would be beneficial. In line with this reasoning, the following suggestions and comments regarding ketosis prevention are made. 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. Excess internal fat also may reduce feed intake. Keep the cows full of good quality roughage. This is just good dairy management, and it helps keep 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. Bring the cows up to full feed on concentrates as rapidly after calving as good judgment indicates. This will depend upon the individual cow, but most cows can be on full feed in 10 days.

This also is an attempt to balance intake and outgo. Feeding a moderate amount of grain prior to calving also may be beneficial. Do not make abrupt changes in the ration. This refers to marked reductions in grain feeding at calving time as well as abrupt changes in the type of roughage, or in the proportions of roughage to concentrate.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. There is some evidence to indicate that susceptibility to ketosis is inherited, but the fact that it appears to be closely related to high production makes selection away from it difficult and probably impractical.

Chapter tow

Materials and methods

2.1. Area of the study :-

Khartoum State lies 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 States of Kassala , Gedaref and Gezira . Most of Khartoum State lies in the climatic of semi – desert region , while northern areas lies in desert zone The climatic of state is ranging from hot to very hot . The weather is rainy in summers , cold and dry in winters . (Khartoum www.krt.gov.sd 2012).

The population of animal in Khartoum state is 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 , 20360in JabalAwlia , 5626 in Khartoum localities (Khartoum census 2008) .

2.2. Systems of feeding :-

The general feeding in the farm during the area of study containing from concentrated diet and green grass . The concentrated diet either preparing in the

farm and contain from daresh , ompaz , radaah , molass and some use manufacturing concentrate .

The pregnant cows in some farm separated from other cows and feeding by especial diet until delivered and other don't separate and feed with all cows . but some owner use over feeding like green grass , concentrate and biscuits with molass as additive feed .

2.3. sampling method :-

Random sampling method was followed to select the study animals ,in Khartoum state was divided to three governorates (Khartoum , Baharri , Omdurman) . Localities were randomly chosen (JabalAwlia , East Nile and Omdurman) . The area selected were West Omdurman (Alroduan), JabalAwlia (Aldkhinat) and East Nile (Sudan University farm and Soba) .

The number of 40 blood sample were collected from dairy cows in the different stages of pregnancy , 10 sample in lactation period (control) , 10 sample at first stage(1-3 month) ,10 sample at second stage (4-6 month) , and 10 sample at third stage (7-9 month) of pregnancy .

The blood sample were taken from the jugular vein or milk vein after cleaning and disinfecting the area , for hematology whole blood was taken and for biochemical test plasma was prepare or preserved at -20c$^{o.}$

2.4. Haematology:

2.4.1. Packed Cell Volume (PCV):-

Whole blood was mixed very well , capillary tube were filled with blood by capillary traction until ¾ of its length , then sealed at one end by plastic seal . The capillary tube was placed in micro hematocrit centrifuge and centrifuged at12.000 cycle /min an automatic time switch of 5 minutes . The reading was recorded as percentage of PCV cells to the total blood volume (Kelly 1984).

2.3.2. Hemoglobin:-

Hemoglobin reagent is based on cyanmethemoglobin method that has been adopted as a standard method . Erythrocytes were lysed by a stromalitic agent in the presence of a surfactant to release their hemoglobin into solution . Haemoglobin was oxidized to methemoglobin by ferricyanide and the methemoglobin was converted to cyanmethemoglobin by addition of KCN (Walters 1968)

The tube was containing 2.5ml of working solution and was added 0.01ml of blood sample with mixture . The developed color was measured in Jenway spectrophotometer (Jenway 6305-U.V/VIS. Spectrophotometer UK) in wavelength 540nm .

Calculation :-

Hemoglobin concentration (mmol/l) = Absorption of sample x 22.82

2.5. Biochemical Tests :-

2.5.1. Glucose:-

Glucose in the sample originates, by means of the coupled reactions described by (Fridman and Young ,2001).

The first tube contained the blank (1.0 ml reagent A), the second tube contained the standard (1.0 ml reagent A plus 10 µl glucose) and the third tube contained plasma (1.0 ml reagent A plus 10 µl plasma). All tubes were mixed thoroughly and incubated for 10 minutes at room temperature and then reading was done at 500 nm against the Blank in Jenway spectrophotometer (Jenway 6305-U.V/VIS. UK).

Calculations :-

Absorbance of sample \ absorbance of standard x concentration standard = concentration of glucose in sample

2.5.2. Total Protein:-

Total protein in the sample reacts with copper(II) ion in alkaline medium forming a colouredcomplex which is measured by spectrophotometer this done according to the procedure of Burtis*et al* ,(2005).

One ml (1.0 ml reagent A with 20µl distilled water) was considered as blank tube, the (20 µl protein standard plus 1.0 ml reagent A) was standard tube and

(1.o ml reagent A with 20 µl plasma) was put in the sample tube . These tube were prepared ,and mixed thoroughly and incubated for 10 minutes at room temperature and were read at 545 nm against the Blank in Jenway spectrophotometer (Jenway 6305-U.V/VIS. UK).

Calculations :-

Absorbance of sample \ Absorbance of standard x concentration of standard = concentration of total protein in the sample

2.5.3. Albumin

Albumin in the sample react with bromocresol green in acid medium forming a coloured complex (Burtis*et al* ,2005). The procedure considered as the tube was containing the amounts of (1.0 ml reagent A) in the blank tube (10 µl albumin standard plus 1.0 ml reagent A) in the standard tube and(1.o ml reagent A with 10 µl plasma) in the tested sample tube .These were mix thoroughly and incubated for 10 minutes at room temperature and was measured at 630 nm against the Blank in Jenway spectrophotometer (Jenway 6305-U.V/VIS. UK).

Calculations :-

Absorbance of sample \ Absorbance of standard x concentration of standard = concentration of albumin in the sample

2.5.4. Urea

The reactions of urea is described by the change of colour in the samples . The procedure considered of one tube containing the amounts of 1.0 ml reagent A as blank tube ,10 µl urea standard plus 1.0 ml reagent A as the standard tube , 1.o ml reagent A with 10 µl plasma as the tested sample tube . These were mixed thoroughly and incubated for 10 minutes at room temperature , then the 1.0 ml of reagent B were added to all tube and mixed thoroughly and incubated for 10 minutes at room temperature. The developed colour was measured in Jenway spectrophotometer (Jenway 6305-U.V/VIS. UK). Reading the absorbance at 600nm against the blank (Burtis*et al* ,2005).

Calculations :-

Absorbance of sample \ absorbance of standard x concentration of standard x Sample dilution factors = concentration of urea in the sample

2.5.5.phosphorus

Organic phosphorus in the sample reacts with molybdate in acid medium forming a phosphomolybdate complex that can be measured by spectrophotometry (Burtis*et al* ,2005).

One tube was contained the amounts of (1.0 ml working reagent with10 µl distilled water) as a reagent blank tube ,(10 µl plasma plus 1.0 ml reagent A) as the sample blank tube and (1.o ml working reagent with 10 µl plasma)as the

tested sample tube and (10 µl phosphorus standard with1.0 ml working reagent) were prepared and mixed thoroughly and the tube were let stand for 5 minutes at room temperature The developed colour was measured in Jenway spectrophotometer (Jenway 6305-U.V/VIS. UK). The absorbance of the sample blank was read at 340nm against distilled water , and absorbance of the sample and of the standard was read against the reagent blank.

Calculation :-

Absorbance sample – absorbance sample blank \ absorbance standard x concentration standard x Sample dilution factors = concentration of phosphorus in the sample

2.5.6. calcium

The O-C resolphthalein complex one (O-CPC) , reacts in alkaline medium with calcium to yield a colour complex .the intensity of the colour is directly proportional to the amount of calcium present in the sample .

Blank tube contained (1000µl reagent (R2+R3)plus 10µl distilled water) andstandard tube contained (10 µl calcium standard plus 1000µl reagent (R2+R3)) andsample tube contained (1000µl reagent (R2+R3) with 10 µl plasma) .Tube were mixed thoroughly and incubated for 5 minutes at room temperature and were measured at 578 nm against the Blank in Jenway spectrophotometer the (Jenway 6305-U.V/VIS. UK). (Moorehead and Briggs 1977)

Calculation :-

Calcium = absorbance of specimen / absorbance of standard x10

2.5.7. Sodium

The present method is based on reaction of sodium with a selective chromogenproducting a chromophore whose absorbance varies directly at the concentration of sodium in test (Maruna 1958) .

1.0 ml reagent R2 added to distilled water for the blank tube and 10 µl sodium standard plus 1.0 ml reagent R2 for standard tube and 1.o ml reagent R2 with 10 µl plasma for sample tube. All tube were prepared and mixed thoroughly and incubated for 5 minutes at room temperature and the test tube read at 623 nm against the blank in Jenway spectrophotometer (Jenway 6305-U.V/VIS. Spectrophotometer UK).

Calculation :-

Sodium = absorbance of sample / standard x150

2.5.8. Potassium

The a mount of potassium is determined by using sodium tetraphenylboron in a sepecifically prepared mixture to produce a colloidal suspension , the turbidity of which is proporational to concenteation of K+ in the range of 2-7mEq/L (Maruna 1958).

Then tube was containing the amounts of either, (1.0 ml reagent R2 with 20µl distil water) as the blank tube and(20 µl potassium standard plus 1.0 ml reagent R2) as the standard tube and (1.o ml reagent R2 with 20 µl plasma) as the tested sample tube .These were prepared ,and mixed thoroughly and incubated the tube for 5 minutes at room temperature and was measured the absorbance (A) of the standard and the sample at 623 nm against the blank in Jenway spectrophotometer (Jenway 6305-U.V/VIS. Spectrophotometer UK).

Calculation:-

Potassium = absorbance of sample / standard x5.0

2.6. Data analysis :-

The data were analyzed using the software statistical package for the social sciences version 16.0(SSPS Inc . and Chicago ,IL, USA) . All blood sample were converted to log 10 cfu cm^2 for analysis and Analysis of Variance (ANOVA) was performed to evaluate the significant differences in TVC between the different operational points/ critical control points. Statistical significance was sct at a *p-value of* < 0.05.

Chapter three

Results

Haematological findings indicated that , there was a significant changes in the packed cell volume (PCV) but there was no significant change in hemoglobin within and between the group of pregnant cows and the control group . The PCV was low in first stage (52.6±6.6mmol/l) , and in third stage (74.0±6.4mmol/l) respectively compared with control group (76.4±4.5mmol/l) , whereas in the second stage PCV was high (86.5±1.3mmol/l) . There was slight changes in hemoglobin concentration, in the first stage there was decrease (7.3±1.3g/dl) , whereas the second stage (12.8±0.7g/dl), and third stage (11.9±1.0g/dl) there was an increase as compared with the control group (11.4±0.9g/dl) (Table 4).

There was significant changes at the biochemical findings at different stage of pregnancy and control group. The total protein was (169.0±30.3 g/l) in first stage and in third stage was (88.2±8.8g/l) and both reading were high as compared with control (67.8±3.6g/l) , the second stage of pregnancy (57.4±1.9g/l) was low as compared with control group . The albumin value in first stage of pregnancy was increase (69.9±12.9g/l) and in third stage (44.0±8.0g/l) of pregnancy , whereas during the second stage albumin

(26.6±1.8g/l) was lower than control group (28.5±3.9g/l) (Table 5) . The globulin in second stage of pregnancy was low (30.7±2.7g/l), whereas in first stage (99.8±17.9g/l)and third stage(42.4±7.0 g/l) was high as compared with control group (39.3±4.1g/l) (Table 5) . Glucose level there was a decrease in the first and second stage of pregnancy (4.1±0.3mmol/l , 4.8±0.2mmol/l) respectively , whereas during the third stage (4.9±0.7mmol/l) there was a increase as compared with control group (4.3±0.4mmol/l) (Fig 1). The value of calcium was (2.4±0.6mmol/l) in the first stage and (1.6±0.3mmol/l) in second stage , there values were both lower than the control group (3.2±1.2mmol/l) , third stage showed increased value (3.8±0.9mmol/l) as compared with control (Fig 2) . The concentration of the urea showed low value in the first stage (32.3±2.8mg/dl) and the second stage of pregnancy (33.7±3.2mg/dl) , but was high than control (47.5±9.4mg/dl) in third stage (55.3±9.8mg/dl) of pregnancy (Table 5).

The Phosphorus level showed decrease in first stage (3.2±0.8mmol/l) , whereas second stage (6.4±2.3mmol/l) it showed an increase as compared with control . The third stage of pregnancy was similar in level of phosphorous in control (5.0±1.2mmol/l) (Table 5) .The sodium in the first stage (125.4±1.5mmol/l) of pregnancy was lower ,whereas in the second stage (131.3±1.6mmol/l) and third stage (130.2±1.3mmol/l) was high as compared with

control (128.9±1.6mmol/l)(Table 5) . There was no change in Potassium between the first stage of pregnancy and control group (8.8±0.6mmol/l) , and in the second stage the level increased (10.0±0.3mmol/l) , but in the third stage there decreased as compared with control (7.8±0.6mmol/l) (Table 5) .

There was different in the haematological and biochemical parameters between the different location of area of study , Khartoum , Omdurman and Este Nile area that more clear in the table (1 , 2 , and 3) and figures (5.6.7.and 8)

Table 1 : The hematological and biochemical parameters in pregnant cows in Khartoum

Location / **Parameters**	**Khartoum**
PCVmmol/l	69.8±5.6
Hemoglobin g/dl	9.6±0.9
Total protein g/l	111.9±18.6
Albumin g/l	46.9±7.9
Globulin g/l	64.3±11.3
Glucosemmol/l	4.7±0.2
Calciummmol/l	1.9±0.3
Phosphorousmmol/l	4.0±1.2
Urea mg/dl	33.5±2.4
Sodiummmol/l	128.7±1.1
Potassiummmol/l	9.6±0.3

Table 2 : The hematological and biochemical parameters in pregnant cows in Omdurman

Location / Parameters	Omdurman
PCVmmol/l	68.7±5.9
Hemoglobin g/dl	11.5±0.9
Total protein g/l	86.7±8.6
Albumin g/l	45.0±6.9
Globulin g/l	41.9±6.9
Glucosemmol/l	4.3±0.6
Calciummmol/l	5.3±1.0
Phosphorousmmol/l	4.5±1.1
Urea mg/dl	63.9±10.1
Sodiummmol/l	129.9±1.8
Potassiummmol/l	7.7±0.6

Table 3 :The hematological and biochemical parameters in pregnant cows in Este Nile

Location / Parameters	Este Nile
PCV mmol/l	84.1±2.2
Hemoglobin g/dl	13.2±0.5
Total protein g/l	65.1±2.9
Albumin g/l	26.3±0.9
Globulin g/l	38.8±2.9
Glucose mmol/l	4.3±0.3
Calcium mmol/l	1.4±0.3
Phosphorous mmol/l	7.5±1.0
Urea mg/dl	34.0±3.5
Sodium mmol/l	128/4±1.7
Potassium mmol/l	8.6±0.3

Table 4: The values of PCV and hemoglobin in pregnant cows at different stage of pregnancy

	Stage of pregnancy			
Parameters	**Control group**	**First stage**	**Second stage**	**Third stage**
PCV mmol/l	76.4±4.5*	52.6±6.6*	86.5±1.3*	74.0±6.4*
Hemoglobin g/dl	11.4±0.9	7.3±1.3	12.8±0.7	11.9±1.0

*Significant at level (p> 0.05)

Table 5: The concentration of various parameters in pregnant cows at different stage of pregnancy

	Stage of pregnancy			
Parameter	**Control group**	**First stage**	**Second stage**	**Third stage**
Total protein g/l	67.8±3.6*	169.0±30.3*	57.4±1.9*	88.2±8.8*
Albumin g/l	28.5±3.9*	69.9±12.9*	26.6±1.8*	44.0±8.0*
Globulin g/l	39.3±4.1*	99.8±17.9*	30.7±2.7*	42.4±7.0*
Phosphorous mmol/l	5.0±1.2	3.1±0.8	6.4±2.3	5.0±1.2
Urea mg/dl	47.5±9.4*	32.3±2.8*	33.7±3.2*	55.3±9.8*
Sodium mmol/l	128.9±1.6	125.4±1.5	131.3±1.6	130.2±1.3
Potassium mmol/l	8.8±0.6	8.8±0.5	10.0±0.3	7.8±0.6

*Significant at level ($p > 0.05$)

Fig 1: The level of calcium in pregnant cows at different stages of pregnancy

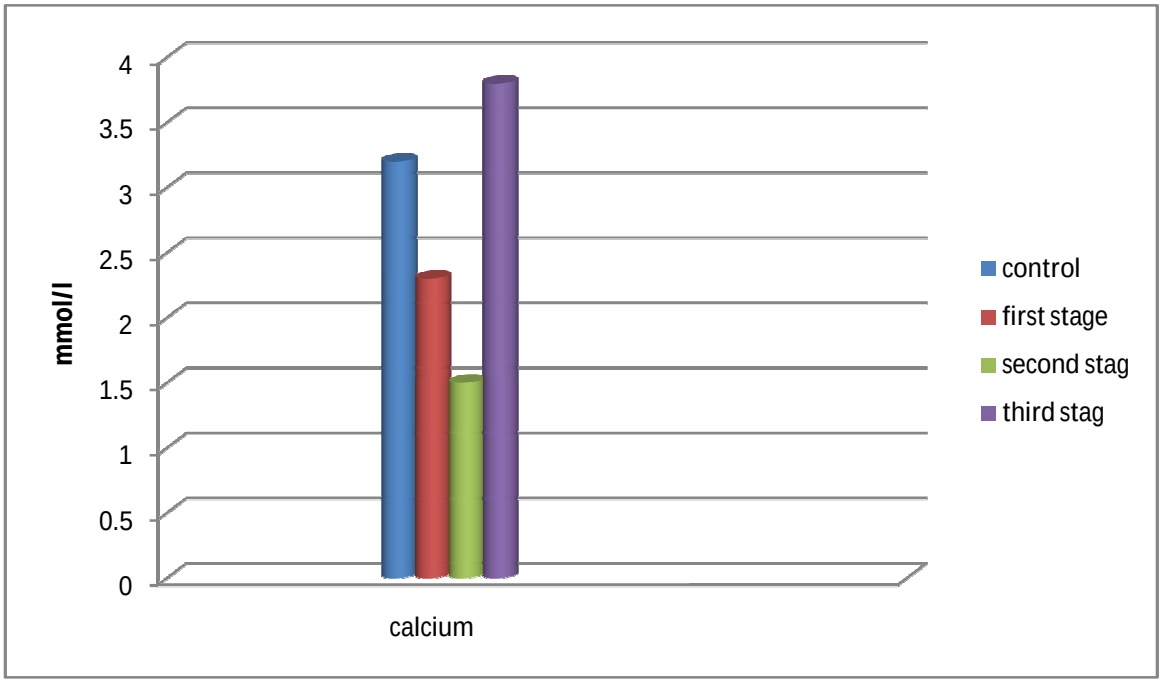


Fig 2: The level of glucose in pregnant cows at different stage of pregnancy

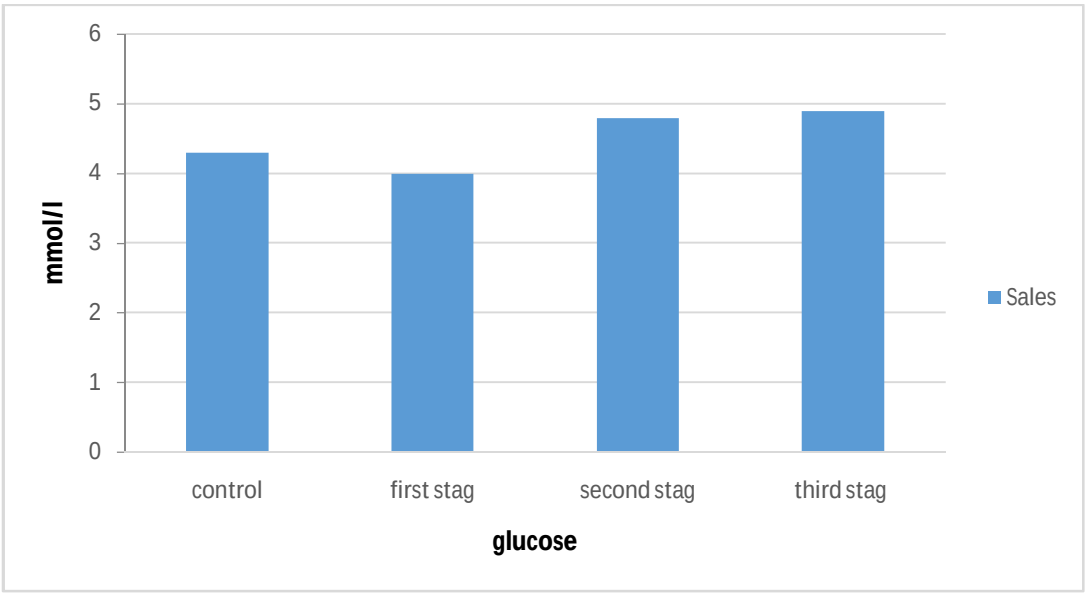


Fig 3: The level of phosphorus in pregnant cows at different stages of pregnancy

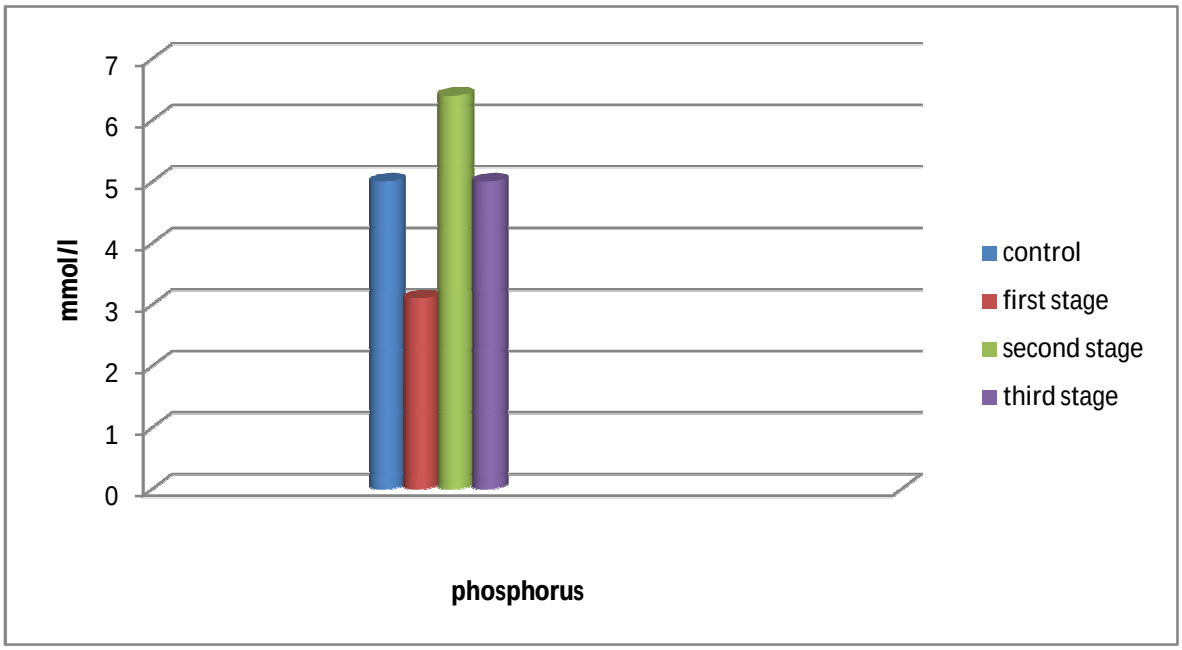


Fig 4: The level of potassium in pregnant cows at different stages of pregnancy

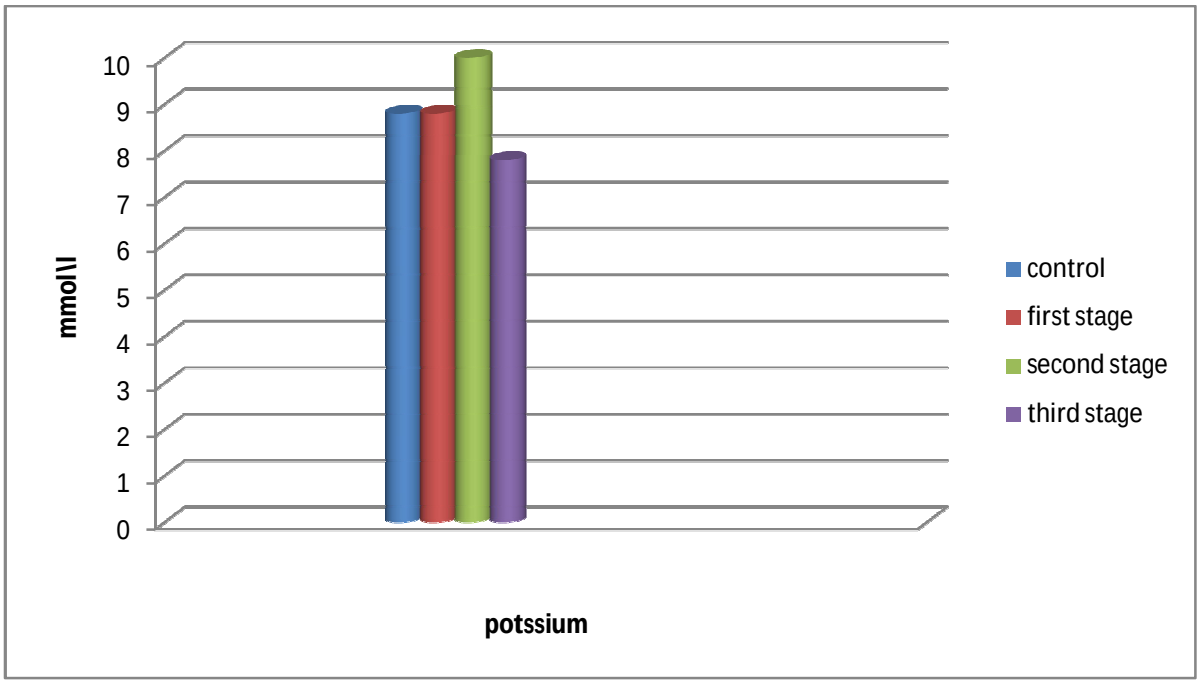


Fig 5: the different in the PCV and hemoglobin level in different location

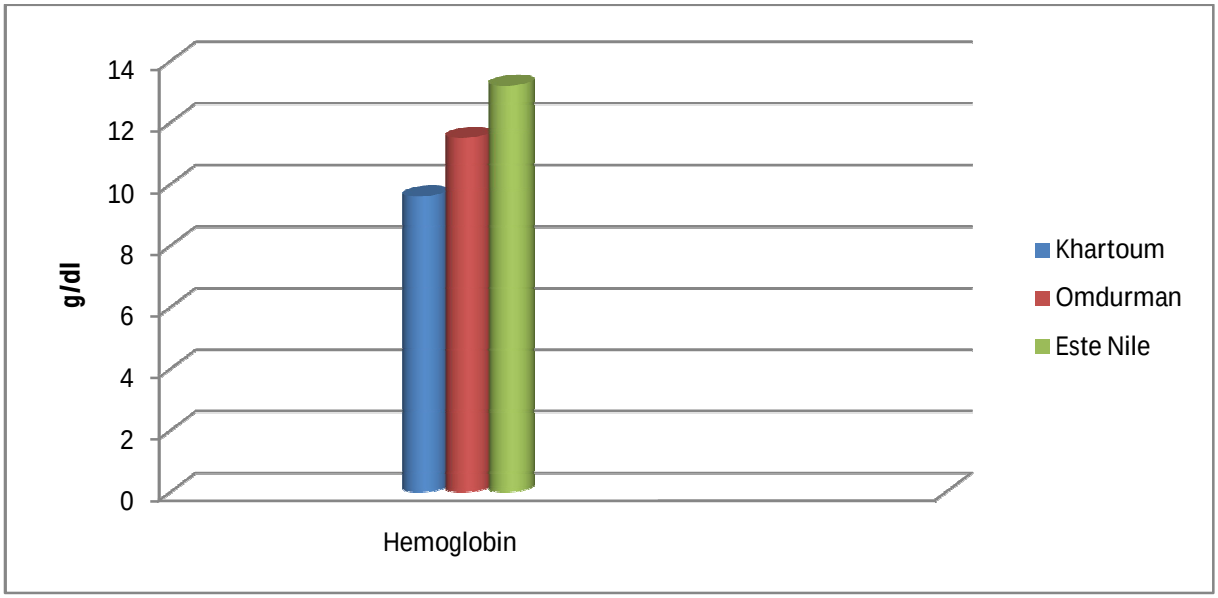


Fig6: The different in the total protein , albumin and globulin in different location

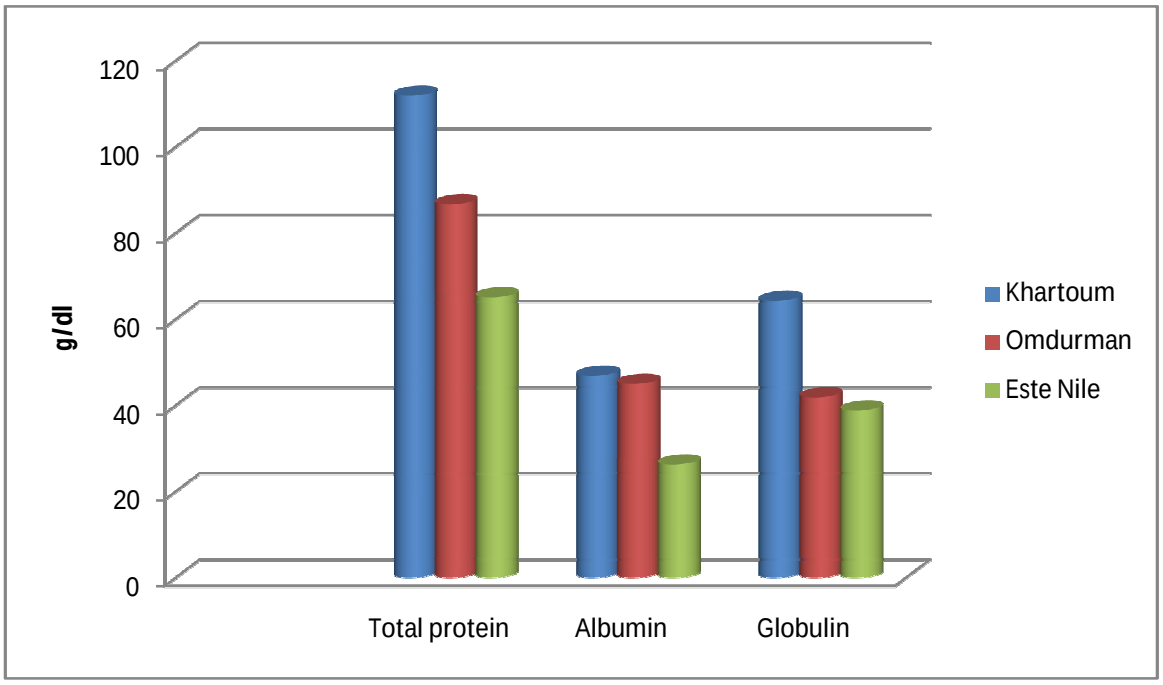


Fig 7: The different in the glucose , calcium and phosphorus in different location

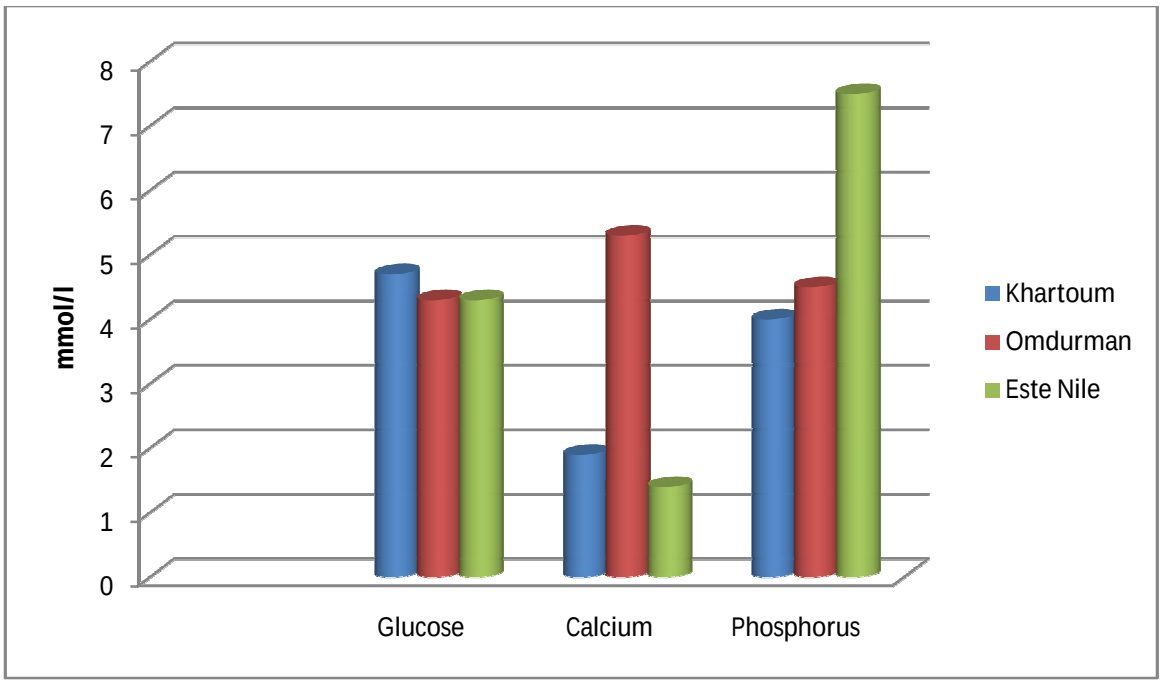
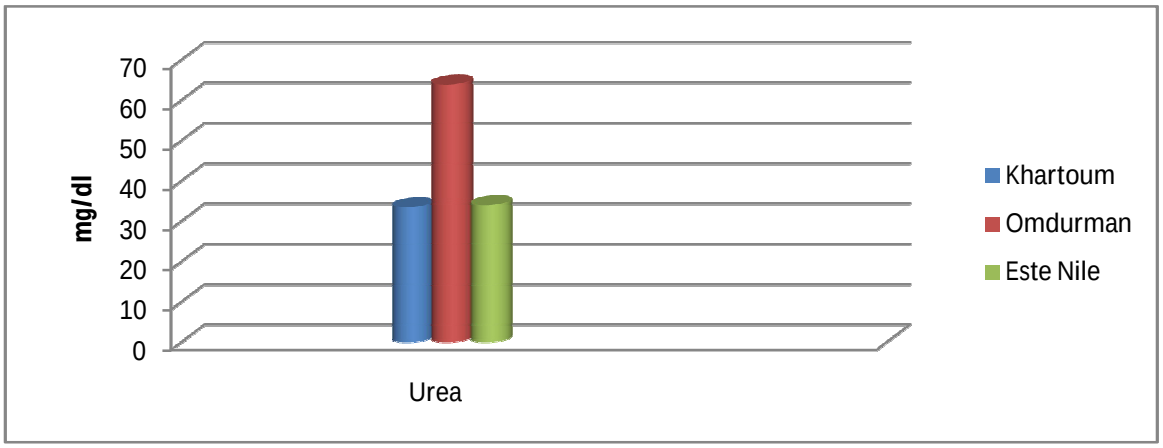
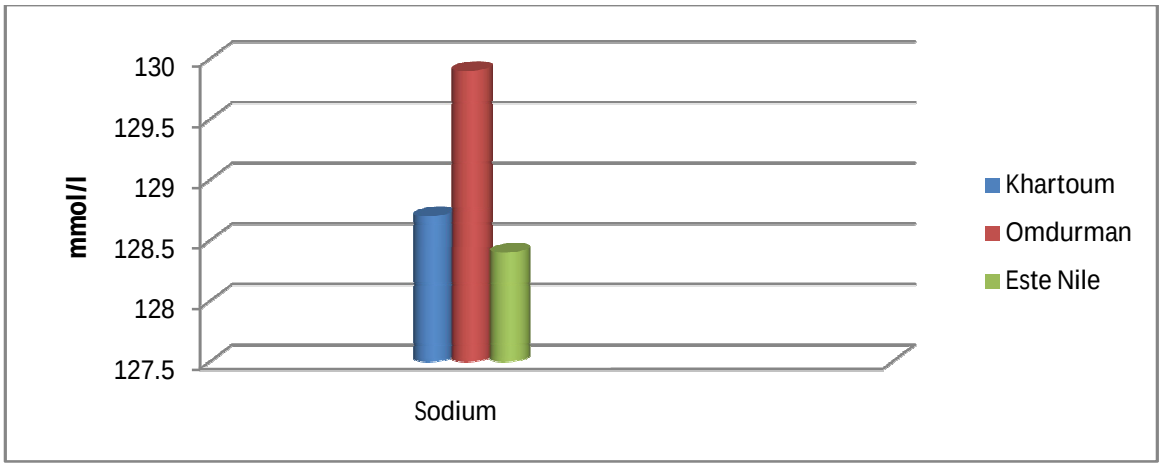
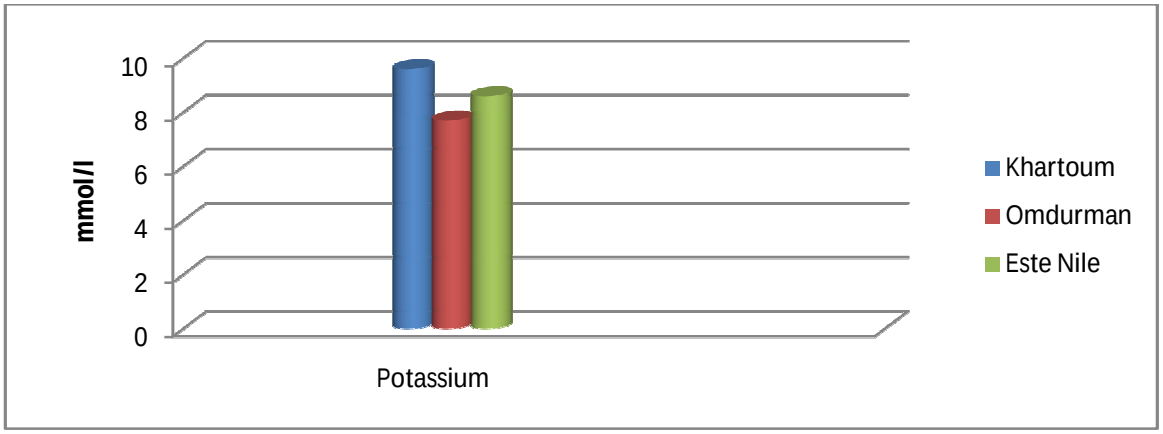


Fig8: The different in the urea , sodium and potassium in different location







Chapter four

Discussion

The metabolic profile test is valuable for measuring the preliminary assessment of the hygienic status as far as feeding is concerned .

In the present study , there was significant changes in the packed cell volume (PCV) and there was slightl changes in hemoglobin within and between the group of pregnancy cows . The increase of PCV in the second stage and the decrease in first and third stage together with no significant changes in (Hb) these results indicated that there was no anemia or dehydration in late stages of pregnancy , and this finding is contrary to the finding of Radostits*et al* , (2007) who said that PCV and Hb just higher in dry cows or in non - lactating cows and low in early stages of lactations.

Total Protein there increase in the third stage of pregnancy , may be due to increase of the crud protein in the ration in feeding at tested cows , this result is similar to that of Morrow *et al* (1979) ,more ever the changes in the total protein which identified according to its characteristic solubility as albumin and globulin is usually affected by nutritional status of animals (Coles , 1986) . Plasma protein serves as a source of nutrition for tissues and plays a dynamic equilibrium which exists between the proteins of plasma and those of tissue .

There was significant changes in the glucose and calcium within and between the all group of the pregnant cows . Glucose level showed a decrease in the two early stages but in the third stage there was an increase , this finding is in contrary to the result of Radostits*et al* (2000) who said that glucose decreased in late stage because it moved to mammary gland to make lactose whereas , Ahmed and Abdalla (2012) reported that there was a non-significant decrease in plasma glucose during late pregnancy . In results calcium was high in the third stage of pregnancy (Fig 2) this result is in dis agreement with result of Roche (2003) and Radostits*et al* (2000) who recorded that calcium levels are reduced below normal in all cows at calving whether they have milk fever or don't milk fever , but in present the an increase in glucose and calcium in the late stage may be due to correction of the dietary program in this period .

Phosphorousdecrease in the early stages and increased in late stage this disagreement with record ofRadostits*et al* (2000) . Sodium was high in third stage and potassium was low in present study this may be attributed to electrolytes imbalance during pregnancy . Radostits*et al* (2000) recorded that low levels of serum sodium occur in early lactation in cows and serum potassium levels have been difficult to interpret because the levels of the electrolyte in serum are not necessarily indicative of potassium deficiency. The decreased in the potassium in the present is result in agreement with Ender *et al* (1971) and Block (1984)

who said that the decrease in the potassium lead to production disease . The high levels in urea in third stage may be related to enhancement of metabolic activities and the use of amino acid as source of energy , that in agreement with (Bell , 1995; Harris , 1995 and Westwood *etal* , 2000).

There was different in the result in present study between the different location of area that collected the samples that may be due to the systems of feeding and management of pregnant cows in the farms .

Conclusion

1. The PCV was low in the first and second stage of pregnancy but there was high in third stage as compare with control group .
2. Hemoglobin was decrease in the first stage whereas an increase in second and third stage .
3. Total protein in the both first and third stage was high but low in the second stage. The albumin in the first and third stage was an increase whereas in second stage was low .Globulin was lower in second stage as compare with first and third stage was high .
4. Glucose level there was decrease in the first and second stage respectively whereas during the third stage there was an increase .
5. The value of calcium was lower in the both first and second stage but in the third stage was an increase
6. The concentration of urea showed lower value in the first and second stage but was an increase in the third stage of pregnancy .
7. Phosphorous level showed decrease in the first stage whereas second stage it showed an increase but the third stage was similar to control group .
8. Sodium in the first stage was lower whereas second and third stage was high .

9. Potassium in the first stage was similar to control group and the second stage was an increase and third stage there was decrease .
10. There was different between the different location of present study .

Recommendation

- Routine examination of cows to detect the pregnant cows early .
- Routine differentiation of metabolic profile of cattle
- Stop the milking and separation the pregnant cows from the seven month in other cage , and correct the diet to avoid the milk fever before or after calving .
- Treatment of the diseased animal rapidly to avoid the prolong recumbency that may lead to cow syndrome .
- Correct the diet by adding more energy supply especially in high lactation period after calving to controlling the hypoglycemia .
- In the first days after calving the milking of cows didn't complete to decrease the risk of production disease .

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