Metabolic diseases 1613

Production diseases

Are those type of diseases affected domestic animals and occur due to some defect in body metabolism ingestion, digestion or absorption. or are those diseases which are attributable to an imbalance between the rates of input of dietary nutrients and the output of production, Therefore change in the amount of the body's reserves of certain metabolites will occur.

In domestic animals, the metabolic diseases have their importance in dairy cows and pregnant ewes. However In the other species, these diseases occur only sporadically.

The incidence of metabolic diseases in cattle is increased mostly at the period of calving when milk production is started and increase and might extending to the peak of lactation because of turnover تقلب,demand احتياج and rapid rate of exchange of water, sodium, calcium, magnesium, chlorides and phosphates or a sudden variation in their excretion or secretion in the milk or a sudden variation in their intake because of changes in ingestion, digestion or absorption, were appeared during stages of lactation and might cause damaging changes in the internal environment of the animal.

Moreover the incidence of metabolic disease will also increase when nutritional demands is increased during pregnancy due to inadequate diet in the dry period, However The effect of pregnancy is particularly important in ewes, especially those carrying more than one lamb.

Compton metabolic profile test (CMP)

The Compton metabolic profile test is based on the concept المفهوم that the laboratory measurement of certain components of the blood will reflect the nutritional status of the animal, with or without the presence of clinical abnormalities.

Advantage of CMP…

1-Monitor metabolic health of the herd

2- Help diagnose metabolic problems and production diseases

3- Identify metabolically superior cows

4-The test included measurement of some hematological and biochemical concentrations in the blood of cows which may reflect nutritional intake and status at all times.

5- and quantitative adequacy tativeThe test able to detect the qualiof the diet of cows expected to produce a certain quantity of milk**.**

Measurement include evaluation of HB,PCV. Serum glucose, serum total proteins and albumin, Non-esterified fatty acids, Serum B-hydroxybutyric acid, Urea nitrogen, Serum inorganic phosphorus,Ca,Mg,Ph,sodium,K.

Timing of blood tests…

The CMP involved collecting 7-to-10 blood samples from 3 predefined groups of dairy animals,
a-Early lactation (EL) : between 10 and 20 days of lactation
b- Mid lactation (ML): between 50 and 120 days of lactation
c- Dry period (D): between 7 and 10 days of calving.
From the test results, averages for each metabolite were calculated for each respective group and compared to reference values**.**

The Mini profile test…
It measure level of blood glucose ,serum urea nitrogen and albumin in cows between 4-10 weeks after calving. this test were use to fast evaluation of adequacy of energy and protein intake.
At the time of sampling the following information must be add
Age, exact milk production, date of calving, weight, analysis of diet and quantity, total herd production

Parturient paresis (Milk fever)….
A disease of cattle, sheep, and goats occur around the time of parturition and caused by hypocalcemia and characterized by weakness, recumbency, and finally shock and death.
Functions of calcium …
1-Builds and maintains bones and teeth
2- Regulates heart rhythm
3-Helps to regulate the passage of nutrients in & out of the cell walls
4- Assists in normal blood clotting;
5- Helps maintain proper nerve and muscle functions as neurotransmitter
6- lowers blood pressure;
7- important to normal kidney function
8- Calcium is also essential for optimum milk production and growth rate.
Etiology….
1- A depression of the levels of ionized calcium in tissue fluids is the basic biochemical defect in milk fever
    - It was postulated that failure to secrete sufficient levels of parathyroid hormone(major Ca.regulator) or 1,25-dihydroxyvitamin D was the primary defect in cows which developed milk fever. While it is accepted that the calcium homeostatic mechanisms ,regulated by parathyroid hormone and 1,25-dihydroxyvitamin D, fail to maintain normal blood calcium concentrations resulting in severe hypocalcemia
    - It was also thought that calcitonin(minor Ca.regulator), a hormone which inhibits bone calcium resorption was a cause of milk fever but this has not been demonstrated in cows with milk fever

Some predisposing factors

a-Reticuloruminal hypomotility which affected the absorption of Ca.as in case of acidosis

b-Decrease absorption of intestinal Ca.due to Vit D level around the time of parturition

c- Starvation for 48 h also causes severe depression of serum calcium when cows fed on poor-quality roughage or affected with sever diarrhea of unknown etiology

d- The I.V administration of certain aminoglycosides, especially neomycin, elihydrostreptomycin and gentamicin, may cause a reduction in the degree of ionization of serum calcium and a syndrome similar to milk fever

e- Oral dosing with zinc oxide (40 or 120 mg Zn/kg BW) in ewes causes a serious fall in serum calcium levels 24 h later.

f- Coliform mastitis may also predispose to hypocalcemia in individual cows because E. coli endotoxin depresses serum calcium

g-High body weight cattle are predisposed to milk fever more than normal body weight animals .

Epidemiology….

Occurrence…

In Cattle….

The disease occurs most commonly in high-producing adult lactating dairy cattle.Lactating beef cows are affected but less commonly.

Age:

Mature dairy cows are most commonly affected 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 their3rd to 7th parturition; it is infrequent at the first parturition.

Note: Cattle at first time of parturition rarely develop milk fever because they are able to adapt rapidly to the high demands of calcium for lactation(increase intestinal absorption of Ca.) With increasing age, this adaptation process is decreased(due to decrease intestinal absorption) and results in moderate-to-severe hypocalcemia in most adult cows.

Time of occurrence…

In cattle, milk fever occurs at three main stages in the lactation cycle.

1-Prepartum period : Most prepartum cases occur in the last few days of pregnancy and during parturition but rare cases occur several weeks before calving. Some cases will occur a few hours before parturition

2-at the time of parturition: when second stage of parturition does not occur because of uterine inertia

3-Post partum: Most cases occur within the first 48 h after calving and the danger period extends up to about the 10th postpartum day.

Note: occasional cases occur 6-8 weeks after parturition due to some predisposing factors such as
a-Excessive fatigue and excitement
b- Sever depression of appetite due to estrous(Increase estrogen always cause depress appetite ,anxiety and excitement) .
In Sheep and goats…..
a-The disease commonly occurs in outbreaks in groups of ewes exposed to forced exercise, long-distance transport, sudden deprivation of food and grazing on oxalate-containing plants or green cereal crops.
b- Mature ewes are the most susceptible, particularly in the period from 6 weeks before to 10 weeks after lambing
c- Hypocalcemia in sheep depresses endogenous glucose production and in late pregnancy in combination with hyperketonemia, facilitates the development of pregnancy toxemia.
Risk factors….
Serum calcium levels decline in all adult cows at calving due to the action or parturition and onset of lactation which increase the susceptibility to the disease
Calcium homeostasis.
Three factors affect calcium homeostasis and variations in one or more of them may be important in causing the disease in any individual:
1.Excessive loss of calcium in the colostrum beyond the capacity of absorption from the intestines and mobilization from the bones to replace.
2. Impairment of absorption of calcium from the intestine at parturition.
3.Mobilization of calcium from storage in the skeleton may not be sufficiently rapid to maintain normal serum levels.

Pathogenesis
1-Normal calcium concentration is 8.5 -10.4 mg/dL (2.1 and 2.6 mmol/L)
2-the hypocalcemia become more severe, when the concentration become (5 mg/dL) range 5-7mg/dL and Without treatment, levels may continue to decline (2 mg/dL) which is usually incompatible with life.
3-The pathogenic effect started with…
a-Neuromuscular dysfunction
b-Atony of skeletal muscle and plain muscle(recumbency)
c-A marked reduction in the stroke volume and cardiac output(brady cardia &arrhythmia)
d-Reduction in arterial blood pressure(hypotension)
e-A reduction in ruminal and abomasal tone and motility(constipation)
f-When hypomagnesemia coexists with hypocalcemia the clinical signs continue but with normal or higher than normal levels, relaxation, muscle weakness, depression, and coma.

g-Low serum phosphorus levels occur in milk fever and contribute to the clinical signs. Some cases of milk fever may not respond to calcium injections even though the serum calcium levels return to normal but may appear to recover when the udder is inflated and serum phosphorus levels rise.. moreover hypophosphatemia could prolong the duration of recumbency.

Clinical findings ….

Cattle…

Three stages of milk fever in cattle are commonly recognized and described.

Stage 1 or standing position….it characterized by

1- The cow is still standing. With brief stage of excitement and tetany with hypersensitivity and muscle tremor of the head and limbs.

2- The animal is disinclined to move and does not eat.

3- There may be a slight shaking of the head, protrusion of the tongue, and grinding of the teeth.

4- The rectal temperature is usually normal to slightly above normal

5- Stiffness of the hind legs is apparent, the animal is ataxic and falls easily and, on going down, the hind legs are stuck out stiffly.

6- in general there is anorexia, agalactia, rumen stasis, scant feces and a normal temperature, heart rate and respirations.

7-Inthis stage the animal respond quickly to Ca, therapy.

Stage 2 sternal recumbancy(pathognomic stage)it characterized by..

1- Prolonged sternal recumbency .depression and the animal have drowsy appearance with a lateral kink in the neck & the head turned to the flank and the animal unable to stand .(pathognomic sings).

2- Some of animals will open their mouths, extend their head and neck and protrude their tongues.

3- The muzzle is dry, the skin and extremities are cold, and the rectal temperature is subnormal (36-38°C).

4- There is a marked decrease in the absolute intensity of the heart sounds with Weak pulse.

5- Ruminal stasis and secondary bloat are common and constipation is characteristic. There is also relaxation of the anus and loss of the anal reflex.



Stage 3 lateral recumbency..characterized by…

1-There is lateral recumbency and the animal almost comatose

2- The depression of temperature and the cardiovascular system are more marked and the heart sounds are almost inaudible.

3-Finally the animal dye quietly from shock in a state of complete collapse.


Sheep and goats

The disease similar to that in cattle. The early signs include a abnormal gait and tremor of the shoulder muscles. Recumbency follows, sometimes with tetany of the limbs , posture is sternal recumbency, with the legs under the body or stretched out behind. The head is rested on the ground, there may be an accumulation of mucus exudates in the nostrils.

Clinical pathology….

1- Serum Ca.level is 5mg/dl or low

2-Serum inorganic phosphate is depressed to 1.5-3.0 mg/dL

3-Serum Mg.level is 4-5 mg/dL or lower
4-Increas level of CPK &AST

Differential diagnosis….

1-Downer cow syndrome
2-Physical injuries which might included
a- Injuries to the pelvis and pelvic limbs
b- Maternal obstetrical paralysis which include, traumatic injury to the pelvic nerves during passage of the calf,(sciatic or obturator nerve), dislocation of the hip, Ruptured gastrocnemius, slippery ground surface
3-Carbohydrate engorgement
4-Hypomagnesimic tetanines
5-sever toxemia due to acute diffuse peritonitis
6-Fat cow syndrome
7-Bovine spongyform encephalopathy
8- Degenerative myopathy due to prolong recumbency

Treatment..

Every effort should be made to treat affected cows as soon as possible after clinical signs are obvious to protect the animals from complication of prolong recumbency.using…
1- Calcium borogluconate at dose rate of 400-800 mL of a 25 % solution, However larger cows need 800-1000 mL and a small cow 400-500 mL. dose is better given half slow I.V and half S.C.
Note 1: The heart should be auscultated throughout the intravenous administration for evidence of gross arrhythmia, bradycardia, and or tachycardia
Note 2: over dose might cause increase heart rate, acute heart block& sudden death .
2- or Calcium borogluconate at dose rate of 600ml of 40% solution
3-Ancillary treatment may contain Glucose 500 mL of a 40% solution, sodium acid phosphate as 200 mL of a 15% solution, and magnesium sulfate as 200-400 mL of a 15% solution
4-In sheep and goats 100-150 mL of 25% solution half slow I.V and half S.C.

Sings of response to calcium therapy…

1-Belching
2- Muscle tremor, particularly of the flanks and often extending to the whole body
3- improvement in the amplitude and pressures of the pulse
4-Increase in the intensity of the heart sounds
5-Sweating of the muzzle
6- Defecation.

Failure to respond to treatment…may occur due to

1-Incorrect diagnosis, or inadequate treatment

2- Relapses after successful recovery, which usually occur within 48

3- Too old cow with poor response due to diminished skeletal reserves

Udder insufflation…

Insufflation of the udder with air was an alternative treatment for cows which continued to relapse following repeated calcium injections. With the availability and effectiveness of orally administered calcium gels, udder insufflation cannot be recommended.

Complications of milk fever

1-Ischemic muscle necrosis

2-dystochia

3-Retanted placental membranes and prolaps of uterus

4-Aspiration pneumonia

General management and clinical care procedures….include

1-The calf should be removed from the cow for the first 48 h and only sufficient milk should be drawn for the calf's maintenance.

2-If the cow is recumbent for any length of time, she must be kept propped up in sternal recumbency and not left in lateral recumbency, which may result in regurgitation and aspiration pneumonia.

3- The cow should be rolled from side to side every few hours and provided with adequate bedding or moved to a suitable non -slip ground surface.

Control….steps for control include…

1-Dietary management during the period before and after calving

2-Administration of calcium gels orally at the time of parturition which will activate calcium homeostasis at calving. and administration of vitamin D before parturition to enhance the mobilization of calcium**.**

3-Diets high in calcium during the prepartum period can result in a high incidence of milk fever and diets low in calcium will reduce the incidence of milk fever in dairy cows**..**

4-Admistration of Ca. therapy before calving is contra indicated because it leads to depress secretion of parathyroid (PTH).as secretion of PTH

will increases renal reabsorption of calcium within minutes, stimulates calcium resorption from bone within hours to days, and stimulates renal vitamin D metabolism.

5-Avoid over fattening and stress at the time of parturition.

Downer cow syndrome

Its one of the complications or sequel of milk fever in which the cow still recumbent(sternal recumbency) and un response following 2 times treatment with milk fever and the sings of milk fever were disappear except that the animal unable to stand .

Etiology…

1-Complication of milk fever

2-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.

3-Injuries to the musculoskeletal system are also common as a result of cows 'spread eagling' their hind limbs if they are unsteady during parturition or forced to stand or walk on a slippery floor immediately before or following parturition.

4-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.

Pathogenesis …

1-In general A long delay in the treatment of milk fever can result in pressure damage and the subsequent inability to stand after treatment for the primary disease

2-Regardless of the cause, the prolonged recumbency results in varying degrees of ischemic necrosis of major muscles of the hind limbs, particularly the semitendinous muscle and muscles caudal to the stifle

3-Prolonged compression of the muscle leads to tissue anoxia, cell damage and inflammation which causes swelling; the swelling causes a further increase in pressure which limits tissue perfusion.

4-Sciatic nerve damage due to pressure also occurs and may contribute to downer cow syndrome
5-Proteinuria and in some severe cases myoglobinuria occur between 12 and 36 h after the onset of prolonged recumbency
6-Acute focal myocarditis may occur in about 10% of cases resulting in tachycardia, arrhythmia, and the unfavorable response to I.V calcium. the exact cause is unknown, although they thought that adrenocortical functions will be enhanced.
7-Prolonged recumbency can result in additional complications such as acute mastitis and decubitus ulcers.

Clinical findings…

1-Cows appear almost normal but, when they are left alone, within a short period of time they back to the position of lateral recumbency.
2- Affected cows either make some effort to stand or are unable to stand following treatment for parturient paresis, but they eat and drink normally and looked bright and alert at first time.
3- Frequent attempts with no standing result in ' crawling' or 'creeping' along the ground with both hind legs in a partially flexed position and displaced posteriorly - the frog leg position.
4- More severe cases are hyperesthetic and the limbs may be slightly stiff but only when the cow is lying in lateral recumbency. these cases are not eat or drink and call 'non-alert downers, it thought that they have brain damage which has not been documented
5- Coliform mastitis, decubitus ulceration(pressure ulcers), especially over the hock and elbow joint, and traumatic injuries around the tubercoxae caused by the hip slings are common.
6-Poor prognosis mostly occur with more complicated cases and death is usually associated with myocarditis .

Differential diagnosis…

All cases causing recumbency syndrome such as metabolic, nutritional, musculoskeletal, toxic, neurological, neoplastic, inflammatory, and infectious diseases.

Treatment…

The prognosis depends on the cause of the recumbency & time and whether or not treatment is indicated or if euthanasia should be recommended, therefore treatment include ..
1-Fluid and electrolyte therapy with vitamins
2- Provide the most comfortable bedding and to roll the cow from side to side several times.
3- Assisted lifting to aid standing

Transit recumbency of ruminants

Transit recumbency (tetany) occurs after prolonged transport, usually in cows and ewes in late pregnancy. It is also recorded in lambs transported to feedlots, and in cows, and sheep delivered to abattoirs. It is characterized by recumbency, alimentary tract stasis, coma, and is highly fatal. Large losses are encountered when cows and ewes in late pregnancy are moved long distances by rail, truck, or on foot.

The disease has also been recorded in cows recently calved, bullocks, steers, dry cows,and lambs. There is an increased incidence of the disease during hot weather

Etiology…

. The cause is unknown, although physical stress is an obvious factor

Predisposing factors…

1-Heavy feeding before shipment

2-Deprivation of feed and water for more than 24 h during transit

3-Unrestricted access to water

4- Exercise immediately after unloading.

Clinical signs may occur while the cattle are still on the transportation vehicle or up to 48 h after unloading. In the early stages, animals may exhibit excitement and restlessness, trismus, and grinding of the teeth. A staggering gait with paddling of the hind legs and recumbency occur, and are accompanied by stasis of the alimentary tract and complete anorexia. Animals that do not recover gradually become comatose and die in 3-4 days.

Biochemical abnormality are characterized by hypocacemia ,hypo Ph.and hypo mag.some times .

Treatment …

Some cases respond to treatment with combined calcium, magnesium, and glucose injections.

Hypomagnesemic tetanies

Tetany associated with depression of serum magnesium levels is a common occurrence in ruminants. The syndrome associated with hypomagnesemia is relatively constant, irrespective of the cause, but the group of diseases in which it occurs has been divided into hypomagnesemic tetany of calves, which appears to be due specifically to a deficiency of magnesium in the diet, and a group of hypomagnesemias in ruminants characterized by lactation tetany, in which there may be a partial dietary deficiency of magnesium but in which nutritional or metabolic factors reduce the availability,or increase the body's loss, of the element so that serum magnesium levels fall below a critical point..

In general, the occurrence of hypomagnesemic tetany is related to some of circumstances

1-Most common is the occurrence in lactating cows turned out on to lush, grass-dominant pasture in the spring after wintering in closed housing.

2-Wheat pasture poisoning may occur when any type of cattle or sheep is grazed on young, green cereal crops.

3-The third occurrence is in beef or dry dairy cattle running at pasture in the winter,usually when nutrition is inadequate and where no shelter is provided in changeable weather rather than in severe, prolonged cold.

4-Less common forms occur in housed animals (winter time)on poor feed.

Hpomagnesemic tetany

(lactation tetany, grass tetany, grass staggers, wheat pasture poisoning)

Magnesium is the major intracellular divalent cation, and is an essential element in a large number of enzymic activities in the body. For this reason it might be expected that hypomagnesemia would be rare. However, because of the problems of absorption of magnesium in the ruminant forestomachs, and the use of animal and pasture management systems that can lead to marginal magnesium uptake, ruminants are at risk of hypomagnesemia.

Etiology…

1-Magnesium homeostasis

magnesium concentrations in blood and extracellular fluid are essentially determined by the balance between dietary intake of magnesium, loss in feces and milk, and the modulating effect of magnesium homeostasis by the kidney. so any disturbances in this mechanism will cause the disease .

2- **Low Dietary intake of Mg.**

3- Renal excretion regulation ..

Regulation of magnesium excretion is partially under the control of parathyroid hormone and increased levels of parathyroid hormone will act to conserve magnesium and vis .

4- **Magnesium reserves**

There are large stores of magnesium in the body, especially in bone. These are available to the young calf but mobilization decreases with age and in the adult ruminant there is little mobilization in response to short-term deficits of magnesium.

5- **Lactation**

Increased requirement for magnesium is almost always associated with the loss of magnesium in the milk during lactation.

6-**Low pasture Ma.** The recommended minimal 'safe' concentration of magnesium in pastures is 2 g/kg DM.

Factors influencing absorption of magnesium…

1- magnesium concentration, is influenced by the Na:K ratio in the rumen, which is determined by the dietary and salivary concentrations of sodium and potassium. Young rapidly growing grass is low in sodium and high in potassium which lead to impairment of magnesium absorption

2-Saliva normally has a high Na:K ratio but where there is a deficit of sodium in the diet, a proportion of sodium in saliva may be replaced with potassium under the influence of aldosterone, which further negatively influences the uptake of magnesium.

3-Young grass fertilized with nitrogenous fertilizers has an increased crude protein which is readily fermentable and leads to increased ammonia concentrations. A sudden rise in ruminal concentrations of ammonia impairs magnesium absorption in the rumen.

4-magnesium absorption is depressed in ruminal acidosis.

5-The ratio of Ca.and Ph in rumen .2.1-1-1.

Note:

hypomagnesemia can have effect on calcium homeostasis as Hypomagnesemia reduces the production and secretion of parathyroid hormone and reduces hydroxylation of vitamin D in the liver, therefore hypomagnesemia can increase susceptibility to milk fever and downer cow syndrome .thus Mg, therapy must added to milk fever treatment.

Epidemiology…

1-Cattle in the first 2 months of lactation and 4-7 years of age are most susceptible, which probably reflects an increased risk due to a higher loss of magnesium in milk.

2- Dairy and beef cattle turned out to graze on lush, grass dominant pasture after winter housing as well as housed cattle are more susceptible and most cases occur during the first 2 weeks after the cattle are turned out to spring pasture..

3-High morbidity and mortalities.
4- Application of potash and nitrogenous fertilizers to pastures will decrease the concentration of calcium and magnesium in plants. and also interfere with absorption from intestine .
5-Incidence were always increase when animals exposed to diarrhea, cold or wet weather.

Pathogenesis…

1-Normal serum Mg. value is 1.7-3mg/dl and sings start when the level of Mg. become below1mg/dl
2-Magnesium has many influences on impulse transmission at the neuromuscular system, including effects on the release of acetylcholine, on the sensitivity of the motor end plate, on the threshold of the muscle membrane and on activation of the cholinesterase system, those were result in tetanic sings but the concurrent hypocalcemia may have a contributory effect.
3-Decrease magnesium concentrations in the cerebrospinal fluid are also important in creating alterations in CNS abnormal functions .

Clinical findings ….

The disease run into acute, subacute and chronic form

Acute form….

1-The animal may be grazing at the time and suddenly stop grazing.
2- the animal appear uncomfortable with twitching of the muscles and ears , severe hyperesthesia and slight disturbances precipitate attacks of continuous bellowing, frenzied galloping, and occasionally aggression.
3- The gait becomes staggering and the animal falls with obvious tetany of the limbs, which is rapidly followed by clonic convulsions lasting for about a minute. During the convulsive episodes there is:
Opisthotonos ,Nystagmus,Champing of the jaws, Frothing at the mouth, Pricking of the ears, and Retraction of the eyelids.
4- Between episodes, the animal become quiet and looked normal but a sudden noise or touch may precipitate another attack.
5- The temperature rises to 40-40.5°C, after severe muscle exertion, the pulse and respiratory rates are also high. The intensity of the heart sounds is increased so that they can be heard some distance away from the cow.
6- Death usually occurs within 1-5h due to asphyxia due to sever contraction of thoracic and intercostals muscles.

Subacute form…

1- In this form of the disease, the onset is more gradual. Over a period of 3-4 days,there is slight inappetence, wildness of the facial expression, and exaggerated limb movements.
2-The animal is refuse to move with Spasmodic urination and frequent defecation and decrease milk production.

3- Muscle tremor and mild tetany of the hind legs and tail with an unsteady, straddling gait may be accompanied by retraction of the head and trismus. Sudden movement, noise, the application of restraint or insertion of a needle may precipitate a violent convulsion.
4- Animals with this form of the disease may recover spontaneously within a few days or progress to a stage of recumbency .Treatment is usually effective but there is a marked tendency to relapse.

Chronic hypomagnesemia….

1- Many animals in affected herds have low serum magnesium levels but do not show clinical signs and There may be sudden death**.**
2- few animals may show sings of dullness, unthriftiness, decrease appetite paresis and a milk fever-like syndrome.

Clinical pathology…

1-estimation of serum Mg.level
2-Reduced serum Ca.level
3-Estimation of Magnesium concentrations in CSF can be used as a diagnostic procedure.
4- The occurrence of low urine magnesium levels is good evidence of hypomagnesemia.

Differential diagnosis…

Cattle
• Acute lead poisoning
• Rabies
• Nervous ketosis
• Bovine spongiform encephalopathy
Sheep
• Hypocalcemia
• Phalaris poisoning
• 'Stagger' syndromes.

Treatment….

1-Slow I.V administration of preparations containing magnesium or magnesium and calcium are used.
combined calcium-magnesium preparation 500 mL of a solution containing 25% calcium borogluconate and 5% magnesium hypophosphite for cattle,50 mL for sheep lV. followed by a SC injection of a concentrated solution of a magnesium salt.
or magnesium sulfate 200-300 mL of a 20% solution injected half IV and half SC.
Note: The rapid IV injection of magnesium may induce cardiac dysrhythmia, or medullary depression which may be severe enough to cause respiratory failure.
2- lV chloral hydrate may be administered to reduce the severity of convulsions during treatment with magnesium.

Hypomagnesaemia tetany of calves
(Whole milk tetany)

The disease occur when the dietary intake of magnesium is inadequate for the requirements of the calf. Affected animals may have concurrent hypocalcemia.

Etiology…

Normally Milk has low concentrations of magnesium therefore the disease occur when calves have only a milk as a sole diet when the animal in grow up state .الغذاء الوحيد.

Predisposing factors …..

1-A significant loss of magnesium occurs in calves allowed to chew fibrous material such as bedding; the chewing stimulates profuse salivation and creates greater loss of endogenous magnesium.

2- calves fed milk-replacer diets.

3- Hypomagnesemic tetany in calves may complicated in field cases by the coexistence of other diseases, especially enzootic muscular dystrophy.

4-Calves with diarrhea are more susceptible.

Epidemiology…

1- Cases may occur sporadically or a number of deaths may occur on the one farm within a short period of time.

2- Most commonly, hypomagnesemic tetany occurs in calves 2-4 months of age or older.

Clinical findings…

1-Affected calf show constant movement of ears , Hyperesthesia to touch, exaggerated tendon reflexes, Shaking of the head, opisthotonos, ataxia without circling followed by a droopy, backward carriage of the ears , difficulty in drinking,

2- Later, fine muscle tremors appear, followed by kicking at the belly, frothing at the mouth and spasticity of the limbs. convulsions follow, beginning with stamping of the feet, head retraction, champing of the jaws and falling, decrease respiratory movements , tonic and clonic movements of the limbs, involuntary passage of urine and feces protrusion and retraction of the eyeballs

Treatment…

Response to magnesium injections (100 mL of a 10% solution of magnesium sulfate).

Lactation tetany of mares (eclampsia, transit tetany)

It caused by hypocalcemia and characterized by abnormal behavior such as incoordination and tetany. The exact cause of the hypocalcemia has not been determined**.** High-producing mares engaged with physical hard work and grazing on lush pasture are more susceptible. however the disease can also occur in transporting and race horses.

The effect of feeding diets high in calcium, during late pregnancy, and a sudden changes in diet after parturition may predispose to the disease.

Most cases occur in lactating mares, with in10th day after foaling or 1-2 days after weaning with high mortality rate.

Clinical sings ….

1-affected animals sweat profusely and have difficulty in moving because of tetany of the limbs and incoordination.The gait is stiff and the tail is slightly raised. Rapid, labored respirations and wide dilatation of the nostrils are often accompanied by diaphragmatic flutter ارتجاف('thumps')or thumping sound

2- Muscular fibrillation, particularly of the masseter and shoulder region ,and trismus are evident but there is no prolapse of the 3rd eye lid.

3- Within about 24 h the untreated mare becomes recumbent, tetanic convulsions develop and die with io about 48 h after the onset of illness..

D.D…

1-Tetanus

2-Laminitis

Treatment….

300-500 mL of a 25% solution of calcium borogluconate or gluconate administered slowly IV(over 15-30 min) is recommended .

Ketosis

Its an important metabolic disease of cattle and sheep, caused by impairment metabolism of carbohydrate and volatile fatty acids, biochemically it characterized by hypoglycemia, ketonemia, ketonuria, and ketolacia and low levels of hepatic glycogen. clinically the disease call acetonemia in cattle and pregnancy toxemia in sheep.

Glucose metabolism in ruminants...

The main function of glucose in the body is regulation of energy metabolism.

Normally Ingested carbohydrate were fermented in rumen to short chains fatty acids such as

1-Acetate(acetic acid) (70%)which, converted to activated acetate transported to peripheral tissues and to the mammary gland and metabolized for storage as lipids or secretion as milk fat. Moreover it will utilized for energy via tricaboxylic acid cycle after conversion to

- Aceto acetic acid
- B-hydroxy butyric acid
- Aceton or ketone

In the presence of oxaloacetate

Note: oxaloacetate is play an important role to bring ketone bodies to tricaboxylic acid cycle for energy production and in absence of oxaloacetate ketone bodies will accumulated and the disease will occur .

2-Propionate (propionic acid)(20%), is the most important glucose precursor(glycogenic), produced in the rumen from starch, fiber, and proteins which converted to glucose and lactose .

3- Butyrate(butyric acid) (10 %). Is the ketogenic precursor produce ketone bodies after ruminal digestion

Etiology….

1-Negative Energy balance….In high-producing dairy cows there is a negative energy balance in the first few weeks of lactation because of low concentration of serum glucose and insulin due to high demands for milk productions therefore cows will mobilize adipose tissue with consequent increases in serum concentrations of non-esterified fatty acids to maintains gluconeogenesis and the end results is ketogenesis.

Cows will reduce milk production in response to a reduction of energy intake, but this does not happen in early lactation because hormonal stimuli for milk production overcome the effects of reduced food intake and glucose metabolism . Under these circumstances, lowered blood glucose levels result in a lowered blood insulin thus Long chain fatty acids are released from fat stores under the influence of both a low insulin and this leads to increased ketogenesis.

Ketone formation …

Ketone bodies are arise from two major sources…

1-Butyric acid in the rumen… A large proportion of Butyric acid produced by rumen fermentation of the diet is converted to B-hydroxybutyrate in the rumen epithelium and is absorbed to blood.

2- Mobilization of fat….

a- Free fatty acids produced from the mobilization of fat are transported to the liver and oxidized to produce acetyl-CoA.

b- Acetyl-CoA oxidized via tricaboxylic acid cycle and metabolized to acetoacetylCoA.its oxidation depend on oxaloacetate therefore in the absence of oxaloacetae ,acetoacetylCoA will converted to acetoacetic acid and B-hydroxybutyric acid.

Role of insulin and glucagons…

The regulation of energy metabolism in ruminants is primarily controlled by insulin and glucagons

1- Insulin acts as a glucoregulatory hormone stimulating glucose use by tissues and decreasing hepatic gluconeogenesis and is also act to prevent lipolysis.

2- Glucagon is the primary counter-regulatory hormone to insulin. play a role in the homeostatic control of glucose.

Ingeneral: A low insulin: glucagon ratio stimulates lipolysis in adipose tissue and ketogenesis in the liver.

3- Cows in early lactation have low insulin: glucagon ratios because of low blood insulin and are in a catabolic state.

4-Elevated ketones may stimulate insulin production and may act as a negative feedback

Predisposing factors

1-Factors that decrease the energy supply .

2-Factors that increase the demand for glucose

3-Factors increase the utilization of body fat as an energy source are likely to increase ketone production and ketonemia.

4-It have been postulated that dysfunction of adrenal gland might play role which cause additional stress of malnutrition.

5-Hypothyrodism

Types of bovine ketosis….

1-Primary ketosis (production ketosis)

This is the ketosis of most herds, which called acetonemia. It occurs in cows in good to excessive body condition that have high lactation potential and are being fed good-quality rations but that are in a negative energy balance.

2-Secondary ketosis

This occurs when other disease cause a decreased food intake such as, abomasal displacement,traumatic reticulitis, metritis, mastitis.

3-Alimentary ketosis

This form is due to excessive amounts of butyrate in silage(Silage made from succulent material may be more ketogenic than other types of Silage because of its higher content of preformed butyric acid) and possibly also due to decreased food intake resulting from poor palatability of high butyrate Silage. This type of ketosis is commonly subclinical but it may predispose to the development of production or primary ketosis.

4-Starvation ketosis

This occurs in cattle that are in poor body condition and that are fed poor-quality feedstuffs. There is a deficiency of propionate and protein from the diet and a limited capacity of gluconeogenesis from body reserves. Affected cattle recover with correct feeding.

5-Ketosis due to specific nutritional deficiency

Specific dietary deficiencies of cobalt and possibly phosphorus may also lead to a high incidence of ketosis because the deficiency cause a failure to metabolize propionic acid into the tricarboxylic acid.

Epidemiology..

Occurrence..

1-It occurs mainly in animals housed during the winter and spring months and is rare in cows that calve on pasture.

2-The disease occurs in the immediate postparturient period with 90% of cases occurring in the first 60 days of lactation.

3- 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.

4-Fatt body condition increase the incidence of ketosis.

5-High incidence were also recorded in animals affected with milk fever, retained placenta, lameness hypomagnesemia & animals with twins .

6-The disease cause high economic losses.

Pathogenesis…..

1-The principal metabolic disturbances observed, are hypoglycemia and ketonemia.

2-Accumulation of ketone bodies (isopropyl alcohol) affected all body tissues especially the brain and cause reversible encephalopathy .

3-Immunosuppression has been demonstrated with energy deficiency and ketosis.

Cinical findings...

Two major clinical forms of bovine ketosis are described - wasting and nervous form

Wasting form.. is the commonest form characterized by

1-gradual but moderate decrease in appetite and milk yield over 2-4 days. In.

2-Body weight is lost rapidly, at a greater rate. Farmers usually

describe affected cows as having a 'woody' appearance due to the apparent wasting and loss of cutaneous elasticity due to loss of subcutaneous fat.
3-The feces are firm and dry but serious constipation does not occur.
4-The cow is moderately depressed with hangdog ذليل appearance and refuse to move and to eat with mild abdominal pain.
5-A characteristic odor of ketones is detectable on the breath and often in the milk some times .
6- Normal heart and respiratory rate with low ruminal contractions.

Nervous form….

Signs are usually bizarre and begin quite suddenly characterized by
1-Walking in circles with Hyperesthesia some times
2-" Straddling or crossing of the legs
3- Head pushing or leaning into the stanchion
3- Apparent blindness
4-Aimless movements and wandering
5-" Vigorous licking of the skin and objects
6-" Depraved appetite
7-Chewing movements with salivation.

Subclinical ketosis…

Many cows that are in negative energy balance in early pregnancy will have ketonuria without showing clinical signs but will have decreased productivity of milk yield and a reduction in fertility.

Clinical pathology…

1- Blood glucose will reduced from normal level 45-75mg/dL to 20-40mg/dl.
2-Mesurment of Blood or plasma ketone bodies (ketonemia)
Normal keton bodies level is 1000 milimol/L,in subclinical ketosis level become 1400 milimol/L and in clinical ketosis level become 2500 milimol/L.
2- Mesurment of milk and urine ketone bodies…which done by cowside test(***Ross test or*** **Rotheras test** ***or nitroprusside test***) test as Milk and urine ketone levels have been detected by the reaction of acetone and acetoacetate with sodium nitroprusside.
DD…..

Wasting form

• Abomasal displacement
• Traumatic reticulitis
• Primary indigestion
• Cystitis and pyelonephritis
• Diabetes mellitus.

Nervous form

• Rabies

• Hypomagnesemia
• Bovine spongiform encephalopathy.

Treatment…

1-Glucose (dextrose) The IV injection of 500 mL of a 50% solution of glucose results in transient hyperglycemia, increased insulin and decreased glucagon secretion and reduced plasma concentration of non – esterified fatty acids

2- propylene glycol or glycerol 225 g twice daily for 2 days, followed by 110 g daily for 2 days to cattle. Propylene glycol is absorbed directly from the rumen and acts to reduce ketogenesis them metabolized to glucose .

3-Adminstaration of sodium propionate 110-225 g doses daily as glucogenic precursor.

4- Glucocorticoids therapy:

It cause re-partitioning of glucose in the body which lead to Hyperglycemia occurs within 24 h of administration such as dexamethasone 10mg/kg Bw Iv or Im, flumethasone (5 mg),

5-Insuline :

Which will facilitates cellular uptake of glucose, suppresses fatty acid metabolism and stimulates hepatic gluconeogenesis and give as protamine zinc insulin 200-300 IU per animal administered SC every 24-48 h as required.

6- ancillary treatment such as Vitamin B12 and cobalt

Pregnancy toxemia in sheep

Its a metabolic disease affected sheep& goats and characterized by Hypoglycemia and hyperketonemia.

Etiology…

1-a decline in the plane of nutrition during the last 4 to 6 weeks of pregnancy. This is the period when fetal growth is rapid and the demands for energy markedly increased, particularly in ewes that are carrying twins or triplets.

Classification of pregnancy toxemia…according to cause it classified to

1-Primary pregnancy toxemia

This is the most common type results from a combination of a fall in the plane of nutrition during the latter half of pregnancy with a short period of food deprivation due to shearing, change of environment, drenching, poor quality hay.

2-Fat ewe pregnancy toxemia

This occurs in ewes that are very well fed and are in an over fat condition with lack of exercise in late pregnancy. Fat ewes will exposed to fall in food intake in late pregnancy, due to the reduction of the rumen volume by the pressure of intra-abdominal fat and the developing fetus.

3-Starvation pregnancy toxemia

This occurs in ewes that are thin, where there is prolonged drought time and no alternative feed supply with mismanagement

4-Secondary pregnancy toxemia

This occurs as a sporadic disease as the result of the effect of intercurrent disease such as foot rot or foot abscess, Heavy worm infestation,which affects food intake.

5-**Stress-induced pregnancy toxemia**

Its less common type affected ewes occur due to stress such as transport of late pregnant sheep .

Epidemiology…

1-Its primarily a disease of sheep in late pregnancy time(last 6 weeks of pregnancy, last month, with the peak incidence in the last 2 weeks of pregnancy) especially those carry twins or triples. ewes carry a single, large lamb may also be affected.

2-high mortality rate will occur even with treatment some times .

Pathogenesis….

1- 60% of fetal growth takes place in the last 6 weeks of pregnancy. therefore high demands for glucose by the growing fetuses resulting in hypoglycemia,lipid mobilization and increase cortisole level and the accumulation of ketone bodies.

2-Excessive lipid mobilization will elevated concentrations of B-hydroxybutyric acid which suppress the endogenous glucose production.

3-Accummulated ketone bodies can cause irreversible encephalopathy.

4- The increase of plasma concentrations of non-esterified fatty acids results in a depression of cellular and humoral immune responses.

Clinical findings…

1-Separation from the group, refuse moving ,appearance blindness and failure to come up for feeding.

2- The ewe will stand still when approached by attendants or dogs and will turn and face them but make no attempt to escape.

3- If it is forced to move, it blunders into objects.

4- Many affected ewes stand in water troughs all day and lap the water. Constipation is usual and the feces are dry and scanty and there is grinding of the teeth.

5-In later stages, marked drowsiness develops and episodes of more severe nervous signs such as tremors of the muscles of the head ,twitching of the lips, champing of the jaws and salivation, accompanied by clonic contraction of the cervical muscles causing dorsiflexion or lateral deviation of the head, followed by circling.

6- A smell of ketones may be detectable on the breath of the ewe.

Clinical pathology…

Hypoglycemia, ketonemia, and ketonuria are characteristic of the disease.

Treatment…

1- 5-7 g of glucose I.V 6-8 times a day in conjunction with 20-40 units of zinc protamine insulin given I.M every other day for 3 days.

2- IV injection of isotonic sodium bicarbonate or lactated Ringer's solution

3- Standard doses of corticosteroids is notrecomended

4- propylene glycol or glycerine (110 g/d) given by mouth is used to support parenteral glucose.

Fatty liver in cattle (Fat cow syndrome, hepatic lipidosis,pregnancy toxemia in cattle)

Its a major metabolic disease of dairy cows in early lactation and is associated with decreased health status and reproduction.

Etiology…

Fatty liver is caused by the mobilization of excessive quantities of fat from body depots to the liver. It develops when the hepatic uptake of lipids exceeds the oxidation and secretion of lipids by the liver. Excess lipids are stored as triacylglycerol in the liver and are associated with decreased metabolic functions of the liver. It occurs because of a sudden demand of energy in the immediate postpartum period in well-conditioned lactating dairy cows. It also occurs because of a sudden deprivation of feed in fat pregnant beef cattle, and is especially severe in those bearing twins.

Epidemiolog…

1-is common in high producing dairy cattle from a few weeks before and after parturition and is associated with several periparturient diseases,such as ketosis, left-side displacement of the abomasum, mastitis, retained fetal membranes, milk fever, and the downer cow syndrome

2-In dairy cows, fatty liver occurs primarily in the first 4 weeks after calving when up to 50% of all cows have some accumulation of triacylglycerol in the liver.

3-Dairy cows with abnormally long, dry periods also have a tendency to become obese and develop the fatty liver syndrome ot parturition.

4-The disease can occur in non-lactating dairy cows by the imposition of a partial starvation diet in late pregnancy in an attempt to reduce the body weight of cows which are considered to be too fat.

Pathogenesis….

1-Under normal physiological conditions, the total amount of fat increases in the liver beginning a few weeks before calving, rises to an average of about 20% 1 week after calving and declines slowly to the normal level of less than 5 % by 26 weeks after calving.

2-The low glucogenic capacity(gluconeogenesis because of highly demand for glucose)after parturition leads to low blood glucose concentrations, low insulin levels and high rates of mobilization of fatty acid, causing severe hepatic lipidosis.

3-Fatty liver develops when the uptake of lipids exceeds the oxidation and secretion of lipids by the liver. Excess lipids are stored as triacylglycerol in the liver and are associated with decreased metabolic functions of the liver.

4-The heavy demands for energy in the high-producing dairy cow immediately after parturition, or in the pregnant beef cow which may be

bearing twins, result in an increased rate of mobilization of fat from body reserves, usually SC fat, to the blood which transports it to body tissues, particularly liver but also muscle and kidney.

Clinical findings….

1-In dairy cattle, fat cow syndrome occurs usually within the first few days following parturition and is commonly precipitated by some conditions such as Parturient hypocalcemia, Left-sided displacement of the abomasums, Indigestion, Retained fetal membranes and Dystocia. The affected cow usually does not respond to treatment for some of these diseases and becomes anorexic

2-Affected cows are usually excessively fat Excessive quantities of SC fat are palpable over the flanks, the shoulder areas and around the tailhead

3-The temperature, heart rate, and respiration are within normal ranges, however Rumen contractions are weak or absent and the feces are usually scant.

4-Periods of prolonged recumbency are common and affected cows may have difficulty in standing when.

5-A severe ketosis which does not respond to the usual treatment may occur with marked ketonuria

6-Some cattle exhibit nervous signs consisting of a staring gaze, holding the head high and muscular tremors of the head and neck. Terminally there is coma .

7-Affected cows will not eat and gradually become weaker, totally recumbent and die in 7-10 days.

Clinical pathology…

1-High level of AST.GGT.SDH and bilirubin

2-cowsite test or **Rotheras test** for detection of ketone bodies

3-Liver biobsy

4- Ultrasonography of liver.

Necropsy findings…

1- the liver is grossly enlarged, pale yellow, friable, and greasy

Treatment…

1- The prognosis for severe fatty liver is unfavorable and there is no specific therapy. but we can use

2- Fluid and electrolyte therapy

3- The subcutaneous injection of 15 mg/d of glucagon for 14 days beginning at day 8 post partum decreases liver triglyceride concentrations

4- Prednisolone at 200 mg 1M daily decreased liver triglyceride concentrations.

Postparturient hemoglobinuria in cattle

Is a disease of highly producing dairy cattle occur soon after calving and characterized by intravascular hemolysis ,hemoglobinuria and anemia .

Etiology…

1-Diets low in phosphorus or un supplemented with phosphorus are usually associated with the disease in dairy cattle.

2-Feeding of cruciferous plants and ingested of hemolytic agents such as rape and turnips which cause erythrocyte lysis some times associated with the disease

Epidemiology…

1-Only adult cows develop the typical hemolytic syndrome, usually in the period 2-4 weeks after calving and High-producing dairy cows in their third to sixth lactations are most commonly affected.

2-The disease does not occur commonly in beef cattle

3-Phosphorus-deficient soils and drought conditions are considered as predisposing causes

Pathogenesis…

Hypophosphatemia results in a decrease in red blood cell glycolysis and adenosine triphosphate (ATP) synthesis which predispose red blood cells to altered function and structure, with loss of normal deformability, and an increase in fragility and hemolysis resulting hemoglobinemia and hemoglobinuria.

Clinical findings….

1-Hemoglobinuria, inappetence, and weakness develop suddenly and there is a severe depression of the milk yield.

2-Dehydration develops quickly and Dyspnea are obvious, the mucous membranes are pale, with tachycardia , Jaundice may be apparent In the late stages.

3-The feces are usually dry and firm

4- The course of the acute disease extends from 3 to 5 days, the cow becomes weak and staggery and finally recumbent.

5- Gangrene and sloughing of the tip of the tail or the digits has been observed occasionally.

6- Death may occur within a few days and In non-fatal cases, convalescence requires about 3 weeks and recovering animals often show pica.

Clinical pathology…

1-low phosphorus level 3-5 mg/dl

2-Decrease Hb,RBC,PCV

3-Dark brown urine

D.D…

Diseases caused Hb uria and hematuria

Treatment…

1-IV administration of 60 g of sodium acid phosphate in 300 mL of distilled water and a similar dose Sc, followed by further SC injections at 12-hourly intervals on three occasions and similar daily doses by mouth

2-Oral dosing with bone meal (120 g twice daily)

3-Admeniastration of hematonics such as Iron therapy and B12

4-Blood transfusion if necessary .

Azoturia in horses (Acute exertional rhabdomyolysis, Tying-up syndrome,Paralytic myoglobiburia,Monday disease)

Is a disease of horses occur during exercise after a period of inactivity on full ration and characterized by myoglobinuria and muscular degenerations .

Etiology…

1-The exact cause is still un known

2-The commonly accepted theory is that large stories of glycogen are laid down in muscles during a period of rest and when exercise will start the glycogen is rapidly metabolized to lactic acid ,when accumulation occur rapidly it will cause coagulative necrosis of muscle fibers and liberation of myoglobin which escape in urine.

3-Deficincy of Vit E and selenium have some role in pathogenesis also .

4-More other suspected causes include

Hypothyroidism, sodium or potassium deficiency, viral infection, high carbohydrate diets, *Streptococcus equi* infection (strangles) and abnormal exhaustion and hyperthermia although the disease can occur with as little exercise as slow draft work or turn out to pasture after stabling .

5-An important contributing factor is a prolonged period (days to weeks) of rest in a horse have an normal regular exercise.

Epidemiology…

1-The disease is mostly occur as sporadic cases

2-the disease can affected any type of horses at any time but horses with exercise are more susceptible .

3- The disease is of important economic impact especially in athletic horses.

Pathogenesis…

1-The main pathogenic effect is suggested due to dysfunction and death of myocytes with subsequent release of cellular constituents, including the enzymes creatine kinase, aspartate aminotransferase and carbonic anhydrase,and myoglobin.

2- The role of myocytes death is uncertain which may be due to the effect of increase oxidant formation during exercise or inadequate antioxidant activity.

3- Necrosis of myocytes caused pain and inflammation in the muscle, with infiltration of inflammatory cells.

4- Myoglobin, and other cell constituents, are nephrotoxic which might cause myoglobinuric nephrosis and acute renal failure may develop.

Clinical findings…

1- Clinical sings start with stiff or short stepping gait , reluctant to move, pawing, and frequently shift its weight.

2- More severely affected horses can be unable to continue to exercise, have hard and board like painful muscles (usually gluteal muscles), sweat excessively,tremble or have widespread muscle fasciculations.
3-Anorexia ,abdominal pain with increase heart and respiratory rate .bodytempreture may rise to 40c
4- Deep red brown urine (myoglobinuria) occurs but is not a consistent finding.
5- Severely affected horses may be recumbent,first assuming dog-sitting position followed by lateral recumbency with repeated attempt to rise.\

Clinical pathology…

1-Increase serum creatine kinase (CK) aspartate aminotransferase (AST), lactate dehydrogenase and carbonic anhydrase
2- Serum myoglobin concentrations increase markedly during exercise in affected horses, and decline within 24-48 h
3- Severely affected horses are hyponatremic , hyperkalemic, hypochloremic and azotemic (increased serum urea nitrogen and creatinine concentrations).
4- Hemoconcentration increased serum total protein concentration.
5-Muscle biopsy during the acute or convalescent stages reveals myonecrosis.

Treatment…

1-Rest, correction of dehydration and electrolyte abnormalities & to minimize the nephrotoxicity of myoglobin
2-phenylbutazone (2.2 mg/kg, orally or IV every 12 h for 2-4 d) or flunixin (1 mglkg IV every 8 h) or ketoprofen (2.2 mglkg IV every 12 h) should be given to provide analgesia.
3- Acetylpromazine 0.02-0.04 mglkg IM, or xylazine, 0.1 mglkg IM to provide mild sedation and decrease muscles pain .
4- Vitamin E 1 UN/kg and 2.5 Microgram /kg selenium daily for 3-5dyas.
5-The urine should kept alkaline tp prevent precipitation of myoglobin in renal tubules by given sodium bicarbonate orally or IV